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Early Developmental Processes and the Continuity of Risk for Underage Drinking and Problem Drinking

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Abstract

Developmental pathways to underage drinking emerge before the second decade of life. Nonetheless, many scientists, as well as the general public, continue to focus on proximal influences surrounding the initiation of drinking in adolescence, such as the social, behavioral, and genetic variables relating to availability and ease of acquisition of the drug, social reinforcement for its use, and individual differences in drug response. Over the past 20 years, a considerable body of evidence has accumulated on the early predictors and pathways of youthful alcohol use and abuse, often much earlier than the time of first drink. These early developmental influences involve numerous risk, vulnerability, promotive and protective processes. Some of these factors are not directly related to alcohol use per se, while others involve learning and expectancies about later drug use that are shaped by social experience. The salience of these factors-- identifiable in early childhood-- for understanding the course and development of adult alcohol and other drug use disorders is evident from the large and growing body of findings on their ability to predict these adult clinical outcomes.

This review summarizes the evidence on early pathways toward and away from underage drinking, with a particular focus on the risk and protective factors, mediators and moderators of risk for underage drinking that become evident during the preschool and early school years. It is guided by a developmental perspective on the aggregation of risk and protection, and examines the contributions of biological, psychological, and social processes within the context of normal development. Implications of this evidence for policy, intervention, and future research are discussed.

Keywords

aggregation of risk; susceptibility to alcoholism; early childhood risk

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INTRODUCTION

Several basic themes provide guidance for developing a perspective on the timing, processes, and experiences in earlier life relevant to the acquisition, use, and problem use of alcohol.

(1) Much of the causal structure underlying youthful alcohol use and abuse is not specific to alcohol, and in particular is either directly or indirectly the result of the development of externalizing and internalizing behaviors.⁽¹⁻³⁾ Thus, family history of antisocial behavior, child maltreatment, and other negative life experiences are well established precursors of later alcohol problems and alcohol use disorders. These predictors are nonspecific risks for alcohol involvement because they also predict a broad array of other problematic outcomes, including problems of undercontrolled or dysregulated behavior, such as conduct problems, impulsivity, attention problems, aggressiveness, antisocial personality disorder, and depressive spectrum disorders.

(2) At the same time that children develop behavior problems not specific to alcohol, they also acquire knowledge about the existence of alcohol as an object in the social environment. Learning about alcohol includes developing beliefs about alcohol based on an awareness of its special characteristics as a drug (how it produces changes in cognition, feeling, and behavior) and its place in social relationships, discovering who uses it and why, and ultimately, developing expectancies about its use. To a large degree, these cognitive variables regulate when and how much consumption takes place, and shape recognition of the appropriate circumstances for desistance from use.

(3) In tandem with the development of behaviors and beliefs related to alcohol, other developmental changes are occurring within the individual and in the social context that also influence behavior. As the brain is developing, consumption of beverage alcohol is interacting with changing brain structures and functions related to appetite, reward, planning, affective and behavioral control. These neurobehavioral processes unfold from the interplay of genes and experience, in many cases, operating through intermediate endophenotypes.⁴ The latter are traits or biologic indicators, genetically simpler than the diagnostic phenotype and more proximal to genetic influence, yet being part of the vulnerability pathway for the disorder.

(4) Social environmental influences in the family, peer group, school, community and larger macrosystems of society also play a significant role in modeling alcohol intake and the contexts of acceptable use. At the cultural level, social norms specify the age grades and social roles within which alcohol use/heavy use is acceptable and when it is not acceptable, and these social norms have been incorporated into legal norms that specify the appropriate sanctions for violations of these alcohol use regulations.

(5) This multilevel, dynamic interplay of biological, psychological, and social processes not only shapes risk, but also normal development⁵. Normal development has the potential to profoundly alter risk parameters and pathways of behavior, and even move the at-risk child into a different, nonproblem pathway. The present review focuses on processes of risk for underage and adult drinking that emerge before adolescence, generally defined as prior to the second decade of life. We recognize that puberty may be well underway for some young people under the age of 10; however, our focus is on early and middle childhood and processes that generally precede pubertal development and the social changes that characterize adolescence.

A BRIEF DEVELOPMENTAL PORTRAIT OF THE UNDER 10 YEARS

The “under 10” years encompass all the growth and development from conception to the beginnings of adolescence. These years are often divided into prenatal development, infancy, early childhood or the toddler and preschool years, and middle childhood. Key contexts after

birth include attachment relationships, the family and home, the family neighborhood, daycare and preschool settings, kindergarten and the early primary grades of school, playgrounds, peer play groups, school classrooms, and increasingly, the media worlds afforded by television, music, electronic toys and games, computers, and movies (see Table 1).

The pace of development during the first 10 years of life is astonishing, from conception to fetus to children who can manipulate their parents, play card games, build elaborate castles from sand or blocks, cruise the internet, hit a baseball, gossip, read and write stories, understand other people and feel guilty about breaking the rules. The human brain undergoes remarkable growth and change over this interval, in structure, organization, and function. During these years, fundamental self-regulation and social regulation systems develop, including the regulation of sleep, stress, and behavior.

During these years, many of the most basic human systems for adapting to the world are developing, including ways we perceive and learn, solve problems, communicate, regulate emotion and behavior, respond to stress, and get along with other people. What we call “personality” is taking shape as a result of individual differences in genetically influenced temperament, experience, and their complex interactions over time. All of these adaptive systems continue to change with development and experience throughout the life course. However, by age 10, many fundamental adaptive systems of the human organism, both those embedded in the person and those embedded in relationships and connections to the social world, have assembled and exhibit some stability. Children arrive at the transitions and challenges of adolescence with the personality, human and social capital they have accumulated in childhood, as well as with their record of achievements and failures meeting the various developmental tasks of childhood. Thus it is not surprising that many of the influential factors associated with early drinking emerge and are shaped during the first decade of life

Organization of the Review

This review has six sections. In Section I, we describe how core developmental processes, such as behavioral and emotional dysregulation, function as predisposing risk factors for youthful alcohol use. In Section II, we review other non-specific-to-alcohol risk factors which enhance drinking risk. In Section III, we describe alcohol-specific risk factors in childhood that are associated with subsequent alcohol use. In Section IV, we summarize what is known about risk and resilience developmental pathways, either toward or away from problematic alcohol use. In Section V, we briefly describe the next-step-tasks needed for the formulation of policy in this area, and in Section VI, we outline the implications of existing knowledge for the development of focused interventions. In addition, we identify critical gaps, problems and questions that need to be addressed as part of a new developmental research agenda for understanding and addressing the problems of underage drinking, both as problems in their own right, and as precursors in the pathway to later alcohol problems and disorder.

NON-SPECIFIC CHILDHOOD RISK FACTORS

This section presents on non-specific factors that predict likelihood for subsequent alcohol involvement, such as behavioral dysregulation/undercontrol (including such factors as conduct disorder, attentional deficits, aggressiveness); other childhood psychopathology; environmental influences such as family, peer and school relationships; and precocious puberty.

The Emergence of Behavioral and Emotional Dysregulation and Predisposition to Alcohol Involvement

Newborns emerge into this world with cries of greater or lesser intensity, lasting for shorter or longer periods of time, and with a quicker or slower response to the caretaking agents who attempt to soothe and comfort them. If the comforting (feeding, handling, being engaged by the caretaker) is sufficient, the infant begins to display signs of satisfaction and relaxation. If it is not, the affective expression continues. Thus the display of emotion as well as its intensity, and the degree to which it is capable of being modulated, are basic characteristics of the human organism for display of displeasure, discomfort, and pain on the one hand, and for the display of pleasure, comfort, and happiness on the other. These are basic temperamental characteristics that serve signaling (communicative) functions, that facilitate social engagement, and that serve as organismic motivators to either sustain current activity, or drive us to seek a change of state.^{6,7} Such differences are observable even at birth, and form the substructure for later more differentiated feelings of happiness, self satisfaction, sadness, and anxiety.

Parallel to the emergence of emotionality and the existence of individual differences in affective expression, a developmental sequence is present for the emergence of motoric behavior and for attention. We know that even before birth, there are fetal differences in activity level, and such variations are quickly evident after birth. Infants vary in how much they move, as well as how quickly they respond to stimuli of light and sound and touch. Some respond quicker than others. Similarly, very early differences are evident in the degree to which children sustain focus or attention on an object and shift focus when a new set of stimuli are presented, and in the amount of information they can retain. Such differences reflect the rudiments of a behavioral regulation and control system on the one hand, and an attentional regulation and control system on the other, which ultimately determine the ability to plan, to inhibit responses while reflecting on alternative plans, and to access a broad array of information used in deciding whether it is wiser to carry out or to inhibit a particular action. These regulation functions are essential to such basic processes as learning, planning and forethought. When they function poorly, or when the social environment makes it difficult for them to develop (such as in homes where there is abuse and violence), social and academic achievement are more difficult, and risk for substance use disorders is substantially elevated. In fact, one of the most prominent theories for the development of alcohol and other drug use disorders posits the importance of a central dysregulatory trait, involving delayed or deficient development of behavioral, emotional, and cognitive regulation in the early emergence of substance use disorders.⁸ The dysregulation is identifiable as “difficult” temperament in infancy and early childhood, and as an array of behavioral and neuropsychological deficits in adolescence. A substantial body of evidence supports the validity of this dysregulatory hypothesis.^{9,10}

The processes we have described here are basic to the development of all children, and are relevant to the many tasks of adolescence and adult life. Although we do not yet fully understand the mechanisms of impact of behavioral regulation and attentional control, these domains appear to be highly relevant to the acquisition and maintenance of alcohol use, as well as the progression into problem use. Given the centrality of these processes to relationships, to purposive behavior, to making choices, and to desistance, it is not surprising that they also are tied to the emergence of alcohol problems. Thus, the choice to use alcohol for the first time (i.e., drinking onset) is a cognitive choice, about whether or not this is a wise act, what the consequences are of doing so at any particular time, etc. It also is a behavioral act, and is more likely to take place among young people who act impulsively, and who are interested in new sensations and new experiences. Finally, it is an emotional act, driven to some degree by one's sense of satisfaction or discontent with the world as one knows it before drinking. The possibility that a drink can create a change is more attractive if one is unhappy with one's-self and one's social relationships.

For some time, substance abuse researchers have been aware of these nonspecific-to-alcohol processes, and this in turn has led to the search for the traits that underlie them. Over the last 20 years, an increasing amount of evidence from longitudinal studies has identified two such traits that are detectable very early in life, that predict alcohol (and to some degree other drug) involvement, and that appear to be markers of an underlying genetic diathesis for early use, heavy use, problem use, and alcohol use disorder. This work, coming from six long term prospective studies,¹¹⁻¹⁵ provides a remarkable convergence with the genetic literature in demonstrating that externalizing (aggressive, impulsive, undercontrolled) and to a lesser degree internalizing (anxious, sad, depressive) symptomatology appearing in early childhood is predictive of SUD (Substance Use Disorder) outcomes some 15 to 20 years after the first appearance of the drug-nonspecific behavioral risk (see Zucker³ for a review of this work). Moreover, these traits are known to be relatively stable over the course of childhood and adolescence,^{16,17} with the individuals showing the greatest continuity of problems also the most likely to develop the more chronic and severe forms of SUD in adulthood.^{18,19}

Neurobiological and Cortical Development of the Regulatory Systems

At the neurocognitive level as well, a number of constructs have been identified as being important to risk. Executive functioning entails the ability to regulate behavior to context and to maintain a goal set; it relies on multiple constituent functions.²⁰ This is a multi-component construct, including such elements as response suppression/inhibition (the ability to strategically suppress a prepotent or prepared motor response), working memory (itself multi-componential), set shifting (shifting from one task set, or “set of rules” to another), and interference control (inhibition of a relatively dominant response system in order to allow another one to operate).²¹ These capacities are represented to a large degree in parallel frontal-subcortical-thalamic neural loops. Important structures include right inferior frontal cortex-to-basal-ganglia (response inhibition²²) dorsolateral prefrontal cortex and associated structures (working memory²³) and anterior cingulate cortex. These networks are heavily subserved by catecholamine innervation. To the extent that they translate directly into behavioral differences, they have relevance to a spectrum of activities that elevate or dampen risk. They relate to wisdom in choice of peers, understanding the importance of context for appropriate drinking behavior, and the ability to resist peer pressure to drink when negative drinking consequences are likely (such as increasing intoxication and the inability to get to school or work the next day and function adequately).

Extensive theory as long as a generation ago attempted to link aspects of executive control to alcoholism risk, but findings supporting this linkage have been mixed.^{24-26,7} More recent work²⁷ suggests that the risk element here is primarily related to response inhibition. In addition, Finn and colleagues²⁸ have theorized that auditory working memory moderates temperamental risk for alcoholism. Other neuropsychological theories of individual vulnerability to alcoholism are numerous, but most are at a low level of specificity.⁸ It is essential to develop (and test) models having a higher level of specificity.

Closely intersecting these processes is the domain of motivation, and reward responsivity in particular. Reward response involves dopaminergic pathways in the mesocortical and mesolimbic pathways that are closely related to those involved in executive control. The literature clearly indicates that executive and reward response mutually influence one another⁶ both in development and dynamically. Extensive research suggests that at both the behavioral and neural level, substance use problems are associated with a dysregulation of reward responsivity, such that the subcortical, involuntary elements (subserved by limbic and striatal circuitry) are over-responding to salient drug-associated stimuli, and the normal cortical control (via frontal circuitry) over this response is impaired or inhibited, leading to excessive risk-taking behavior.²⁹⁻³¹ Furthermore, there is preliminary evidence for a dysregulation of

reward-related circuitry in at-risk populations even before alcohol and illicit drug use occurs.
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During the same developmental period in which alcohol use and alcohol problems escalate, neural alterations are occurring in the frontal executive and reward systems involved in impulse and emotion regulation. The dorsolateral prefrontal cortex (important to executive functioning as well as motivation) is one of the last brain regions to mature, with myelogenesis continuing at least until early adolescence and potentially all the way into early adulthood.³³ Progressive increases in the white matter of this region have been shown during childhood and adolescence.³⁴ These developmental changes directly impact impulse and emotion regulation. It is known that throughout childhood there are developmental gains in the ability to suppress or inhibit prepotent responses and in the ability to suppress irrelevant information.³⁵ Social and emotional skills, such as the ability to discriminate emotional facial expressions, also develop throughout childhood and early adolescence, with associated changes in amygdala responsivity.³⁶ Furthermore, during the interval from childhood through adolescence, the prefrontal cortex gains greater efficiency in its inhibitory control over the amygdala and other limbic structures involved in emotion and reward response.^{37,38} In addition to these structural brain changes, both human and animal studies indicate that there is an alteration in mesocorticolimbic dopamine systems in the brains of adolescents.³⁹ Dopamine input to the prefrontal cortex peaks during adolescence in nonhuman primates,⁴⁰ and dopamine binding, primarily in the striatum but also in the nucleus accumbens (important for reward responsivity), peaks during adolescence.⁴¹

Understanding, at the neural activation level, how these mechanisms operate is crucial to a full explanation of individual risk using neurocognitive and neurobehavioral models. The developmental significance of these changes is substantial when superimposed on a social structure that is supportive of alcohol use. Extensive evidence from neuroimaging studies indicates that alcohol and other substances of abuse have acute and lasting effects on these fronto-limbic and fronto-striatal systems that are implicated in impulse control and reward responsivity.⁴²⁻⁴⁴ Such effects are thus superimposed on this developing circuitry. A major issue not yet addressed concerns the relative importance of amount and timing of alcohol (and other drug exposure) in bringing about such changes, the degree to which other environmental exposures (e.g. stress) also play a role, and the degree to which early neurocognitive vulnerabilities interact with the drug exposure in producing change. An understanding of these processes will require a multilevel-multisystem explanatory structure.

The Genetics of Dysregulation

The strong evidence reviewed above for temperamental individual differences in behavioral regulation and control are paralleled at the genetic level by evidence from a number of heritability studies indicating that one of the core pathways of genetic risk for substance use disorders is carried through a major common externalizing/disinhibitory factor.^{1,45,2} A number of molecular genetic studies also support this relationship, with genetic variants in the serotonergic system having received the largest amount of work thus far. Serotonin (5-HT) is believed to operate as a regulator, with increased 5-HT associated with inhibition of behavior,⁴⁶ and genetic variants of tryptophan hydroxylase (TPH), the rate-limiting enzyme of the biosynthesis of 5-HT being associated with anger-related traits.⁴⁷ Genetic variants in monoamine oxidase A, specifically involving the MAO A promoter, have been associated with impulsive aggression,⁴⁸ antisocial alcoholism,⁴⁹ and impulsive antisocial behavior in the context of childhood maltreatment.¹¹ The 5-HT_{1B} receptor has been linked with antisocial alcoholism in humans⁵⁰ and with increased impulsive aggression in mice.⁵¹ Other potential candidate genes with apparent relationships to the externalizing/undercontrol domain include GABRA2, associated both with childhood and adult conduct disorder and drug use disorder

as well as alcohol dependence in adulthood^{52,53} and DRD4, associated with attention deficit/hyperactivity disorder(ADHD).⁵⁴

In addition to this major common genetic pathway, a number of more specific factors have been identified whose level of influence and role in the etiology of SUD varies across the different drugs of abuse. For alcohol use disorder (AUD), by far the majority of these have involved genes linked to the metabolism of alcohol but given the heterogeneity of the phenotype, it would not be surprising if other pathways of genetic control are also uncovered.

Environmental Influences on Development of Regulatory and Attentional Risk, and Protective Factors

Environmental experiences such as stress, arousal, nurturance, and other aspects of social interaction (e.g., physical abuse, observed family conflict) impact the brain either directly through changes in the development of neural networks or through the production of hormones which alter their development. The brain is thus the playing field within which gene-behavior-environment interactions ultimately take place. A critical question is: what sites appear to have a predispositional vulnerability, both to impairment and to alcohol-seeking behavior?

A substantial basic science literature demonstrates, in animal models, strong effects of maternal rearing characteristics on the development of the biological stress response systems and the drug reinforcement pathways of the brain.^{55,56} Adverse environmental exposures can strongly influence the ontogenic development of the limbic-hypothalamic-pituitary axis (LHPA), and the mesolimbic dopamine “reward” pathways of the brain. The evidence increasingly suggests that adverse socioenvironmental influences, acting in concert with genetic factors, alter the physiological reactions to stressors and to later exposure to alcohol and other drugs of abuse, as well as predicting the cognitive and behavioral responses to later prevention interventions.

An impressive body of preclinical research has demonstrated, at least in the rat, that the ontogeny of the stress response system is regulated in part by maternal factors during early life. Groundbreaking work by Levine and colleagues has demonstrated that at least three aspects of maternal behavior in the rat play a role in the regulation of the LHPA during development. These are tactile stimulation, feeding behavior, and passive contact. The maternal factors have important analogues in human maternal care and attachment. Also in the rat model, Meaney and colleagues have investigated how variations in maternal care impact upon offspring responses to stress across the lifespan,⁵⁷ and they have elucidated the epigenetic mechanisms through which variations in maternal stress response behavior are transmitted from one generation to the next, independent of genetic influences. This group has also demonstrated that early environment stress, and maternal rearing behavior not only predict the ontogeny of the stress response circuitry but also the ontogeny of the mesolimbic dopamine reward pathway that underlies drug reinforcement.⁵⁶ Studies in non-human primates and humans have confirmed that exposure to early-life stressors will alter the response to stress and its underlying circuitry in adults. This observation was confirmed in women who experienced childhood abuse. A history of childhood abuse was found to predict neuroendocrine stress reactivity, which was further enhanced by exposure to additional stressors in adulthood. This work has some parallels with the longitudinal behavioral literature on the long term effects of child abuse, but its correspondence is not perfect. In a long term study by Widom et al.,⁵⁸ on children abused and/or neglected at age 11 or younger and followed up 20 years later, childhood neglect but not abuse related to later alcohol abuse for women; but neither neglect nor abuse related for men. Later analyses showed that graduation from high school served as a protective factor for the women's later alcohol symptoms.⁵⁹ Work needs to be done to resolve these inconsistencies.

More generally, the attentional regulation and control system appears to be subject to the effects of early environmental experience, and an increasing body of evidence suggests that the interactional experiences affecting this system's development play a role as well in the development of drinking behavior. Early stress has lasting effects on brain areas and neurochemical systems involved in impulse control and reward circuitry, systems which increase risk for alcoholism by facilitating onset of drinking, maintenance of drinking behavior, and relapse. Recent work by Nigg and colleagues⁶⁰ also shows that poor response inhibition contributes uniquely to early drinking onset and problem use over and above the usual family risk variables, and plays a predictive role separate from that of behavioral undercontrol.

Emotional display and its obverse, emotional regulation, reflect a process of social transaction between the infant and his/her caretakers.⁶¹ Changes in emotional display and ability to regulate have been shown to be influenced by the degree of attentiveness and responsiveness of the mother, and as the infant becomes older, by the mother's broader social environment (including her relationships with the father and with other adults in her support network⁶², ⁶³ and her own prior social experience, including her own history of abuse or other trauma. Eiden and colleagues have also shown the contribution that fathers make to this process, even early in the life of the child. Thus, alcoholic fathers are lower in sensitivity and higher in negative affect toward their children than are non-alcoholic fathers, and this parenting behavior in turn predicts the reciprocal effect; i.e., lower infant responsivity to the parents. Paternal depression, antisocial behavior, and aggression also were associated with lower sensitivity.

Rearing environments characterized by greater warmth, moderate discipline, and less stress are the most effective in instituting lower levels of externalizing behavior in children and adolescents,¹⁹ and ultimately, in producing lower drug involvement in adolescence.⁶⁴ The circumstances of "mismatch" between parent(s) and child are of greatest interest here, because they offer the greatest opportunity for the dampening of risky child temperament, on the one hand, and the greatest potential for altering developmental course in a destructive way, on the other. Parents who are responsive to their child's needs gradually increase the self regulatory capacity of the child.⁶⁵ Conversely, parents who are aggressive toward their children, and who create a conflict-laden family climate, diminish the child's capacity to regulate and control their own behavior.^{19,16}

From the perspective of prevention, perhaps the most promising preclinical finding is that the effects of an adverse rearing environment are reversible. Enrichment of the rearing environment enhances the functioning of the frontal cortex of the brain, including the medial prefrontal cortex that provides inhibitory regulation over LHPA responses to stress. Furthermore, environmental enrichment reverses the effects of maternal separation on stress reactivity in the rat model.⁵⁶ Consistent with this preclinical finding is the observation that childhood interventions can offset the cognitive and emotional developmental risks associated with family stress, and those children who demonstrate the most profound deficits show the greatest improvements with intervention.

Nonspecific Childhood Risk Factors for Alcohol Involvement

A number of antecedent risk factors have been identified in childhood that predict the early onset of drinking and the development in adolescence or adulthood of alcohol problems and alcohol use disorders. Many of these involve higher-order constructs such as behavioral undercontrol, dysregulation, and negative affectivity. They are assessed variously through personality measures, symptom counts, and even by way of formal DSM-IV child psychiatric diagnoses. In addition to these individual factors, two socialization domains have also consistently been identified as risk factors: one involving neglectful or poor parenting; the other involving earlier exposure to alcohol and other drug use by parents and by peers. As noted in the following review, predictors in these domains have been replicated many times over.

Other antecedent risk factors not falling so readily into these domains have also been identified, including early childhood sleep problems, attention problems, and deficits in reading achievement. In the neurophysiological domain, investigators have also suggested that the P300 waveform of event-related brain potential (ERP) is a marker of a risk endophenotype for substance use disorders⁶⁶ (Porges review). P300 appears about 300 milliseconds following presentation of a discrete auditory or visual stimulus. The measure has a variable latency depending on the complexity of the eliciting task and the processing speed of the individual. The measure is conceptualized as reflecting a memory updating process in response to stimulus-driven changes in memory representations. It is believed to index the allocation or updating of working memory as well as a cortical orienting reflex⁶⁷. (Fredrick and Iacono reference). A reduction in the amplitude of the P300 potential has been hypothesized to be an endophenotype for SUDs, possibly reflecting CNS disinhibition⁶⁶[Porjesz review]. Because much of this work has not been replicated yet, and because the predictors do not fall so easily into the above domains, they have received less attention. Nevertheless, these findings are robust and need to be considered in any comprehensive explanation of the early development of risk for drinking and for progression into drinking problems and alcohol use disorder.

The multiplicity of factors identified here, and as well as their substantial overlap, suggest that: (a) a clearer understanding concerning the core individual vulnerabilities and which are secondary needs to be established; (b) the manner in which individual and environmental factors interact needs to be specified more clearly, and (c) a better understanding of sequencing is also required. The following sections provide a detailed account of the pertinent studies.

Antecedent Predictors of Childhood Onset Drinking (initiation before age 13)

Previous longitudinal research on children has tended to focus on adolescent, young adult, or adult, rather than childhood, alcohol use outcomes. Where childhood initiation has been studied, the focus has been on substance use more generally (alcohol, tobacco or marijuana use) rather than focusing solely on alcohol use due to the generally low rates of use in children. 68-70 Significant antecedent predictors of children's substance use initiation in these studies include lower prosocial family processes (monitoring, rules, parent-child attachment), deviant peer affiliation, peer drug use, parental tolerance of substance use, parental drug abuse, child over-activity, child social skills deficits, and single-parent families.

Among the few studies examining antecedent predictors of child alcohol use are those by Baumrind⁷¹ and Bush and Iannotti.⁷² In her study of preschool children tested at ages 4-5, 9-10, and 14, Baumrind⁷¹ reported that earlier ages of onset of alcohol use were associated with less social assertiveness for both sexes. For girls, earlier onset also correlated with less parental responsiveness and less encouragement of the child's individuality at age 4, and with less parental monitoring and lower socioeconomic status at age 9. For boys, earlier onset of alcohol use correlated with less parental encouragement of independence and individuality at age 4, and with less individuation and self-confidence at age 9. When alcohol use occurred during the early elementary school years, the child was generally introduced to the substance by an adult, usually a parent or close family member. Later ages of initiation were generally due to peer instigations. Bush and Iannotti,⁷² in their study of a largely African- American sample of 4th graders, found that child socialization as rated by other students did not predict the onset of alcohol use without parental permission.

Childhood Predictors of Early-Onset Drinking After Childhood

When early-onset is defined as initiation by age-14 or 15 rather than as childhood onset, a number of other studies have found early predictors. These include studies predicting early-onset drinking (vs. later onset), as well as those using survival analyses to predict the *age of onset* of first use. Studies involve both high-risk and population samples. In the high-risk Seattle

Social Development Study⁷³ for example, earlier age of alcohol initiation was predicted by the following age 10-11 predictors: white ethnicity, greater parental drinking, less bonding to school, and having more friends who drink. In another high-risk study of boys from Pittsburgh, age of onset of alcohol use (use of at least one standard drink per episode) up through age 15 was predicted by antisocial disorder (conduct disorder and oppositional defiant disorder), but not attention deficit hyperactivity disorder or negative affect disorder (anxiety or mood disorder). An earlier analysis in this sample, using a lower threshold of any alcohol use, found that the number of conduct disorder symptoms from mother's report at child age 10-12 as well as child executive cognitive functioning were unrelated to alcohol use at age 12-14.⁷⁵ In another, community-based high-risk sample of families,⁷⁶ parental alcoholism and mothers' ratings of child sleep problems, trouble sleeping, and being overtired at ages 3-5 predicted onset of alcohol use by ages 12-14. Parental alcoholism also predicted onset of drunkenness by ages 12-14. The authors interpreted the sleep problems measure as an indicator of instability of biological rhythm as well as of social dysregulation. Finally, Dobkin et al.⁷⁷ found in a lower socio-economic sample of boys from Montreal that ratings of fighting and hyperactivity at age 6 and of their aggressiveness and friends' aggressiveness at age 10 predicted drunkenness at age 13. Age of onset of drunkenness (by age 15) was predicted in these same boys by teacher ratings of higher novelty-seeking and lower harm avoidance at ages 6 and 10.¹⁴

Turning to population samples, studies suggest factors very similar to those found in high risk samples also predict early initiation of use. Among 10-12 year old abstainers selected from the Minnesota Twin Family Study, antecedent predictors of alcohol initiation at age 14 were conduct disorder, oppositional defiant disorder and any externalizing disorder, but not major depressive disorder or ADHD.⁷⁸ In another study on the same sample, King et al.⁷⁹ found these same externalizing factors to also predict regular use, ever drunk, and heavy drinking at age-14. Several other studies provide significant overlap with the Minnesota findings, but have also extended the network of predictors. In the Ontario Child Health Study, children rated by teachers as having conduct disorder at age 8-12 were more likely to be regular drinkers four years later.⁸⁰ In still another study of a birth cohort of New Zealand children, Lynskey & Fergusson⁸¹ found that conduct problems at age 8 predicted usual intake of alcohol, maximum intake of alcohol, and alcohol-related problems experienced before age-15 (even after controlling for gender, family SES, parental illicit drug use, and parental conflict, which also relate to later alcohol use). In this sample, attention deficit behaviors in childhood were not related to alcohol behaviors and problems at age 15 (similar to results found by McGue et al.⁷⁸).

Early-onset alcohol use (by age 14) was predicted in the Finnish Twin Study by a number of social contextual factors assessed at ages 11-12, including lower parental monitoring and worse home environment. In addition, individual differences measures, including greater behavior problems and fewer emotional problems, as well as gender, also predicted this outcome.⁸² Genetic analyses showed that shared environmental influences predominate as influences on drinking initiation in early adolescence. Finally, the Great Smoky Mountain Epidemiologic Study of Youth tested children at ages 9, 11, and 13; antecedent predictors of having initiated alcohol use four years post-baseline were greater depression, less separation anxiety, and greater generalized anxiety.⁸³

Precocious Physical Development

A number of investigators have found a relationship between early pubertal maturation in girls and early onset alcohol use.⁸⁴⁻⁸⁶ This relationship is usually explained by precocious affiliation with older, drinking peers, but the possible interplay between the social facilitation that drinking peer involvement creates, and the biological changes that take place here which may make alcohol use more pleasurable/reinforcing, have not been evaluated. While

adrenarche typically occurs before age 10 and menarche typically after (although before age 10 among precocious maturing girls), clearly the consequences of these pubertal processes alter development in lasting ways that are highly salient during adolescence. These relationships are discussed in more detail in another paper in this issue.⁸⁷

Childhood Predictors of Middle-Adolescent Drinking

Several studies have linked childhood functioning to later adolescent alcohol consumption levels of the children. For example, in the Woodlawn Study,⁸⁸ teacher ratings of aggressiveness in first grade predicted more frequent use of alcohol at age 16-17 in African American boys (but not girls). There was also a trend for shyness to relate to less alcohol use for boys but not for girls. In a follow-up study of children diagnosed with ADHD and controls, childhood symptoms of inattention measured at age 5-12 were predictive of frequency of drunkenness and alcohol problems in adolescence⁸⁹ (Molina & Pelham, 2003). In contrast to the findings of Kellam et al. for a large general population sample⁸⁸, Hill et al. (2000)⁹⁰ studied families at high risk for alcoholism due to their dense family history for alcoholism. They found that age of onset of regular drinking with negative consequences was predicted by greater extraversion, deficits in reading achievement, reduced P300 (visual and auditory), and greater postural sway.

Childhood Predictors of Adolescent Problem Drinking

Only two groups have so far examined early childhood predictors of problem drinking assessed within adolescence. Both involved high risk samples. In the Seattle Social Development project,⁷³ the strongest predictors of age-16 problem drinking were an earlier age of initiation of drinking and being male. The effects of other age 10-11 predictors (parental drinking, friends drinking, school bonding, perceived harm of drinking) were mediated by age of initiation. In another paper involving the Michigan Longitudinal Study high-risk sample, Wong et al.⁹¹ observed that although the normal pattern of increase in behavioral control over the course of childhood was present in the sample, a *slower rate of increase* in behavioral control from preschool through middle childhood predicted more drunkenness and more problem alcohol use in adolescence.

Earlier Childhood Predictors of Young Adult Problem Drinking/Alcohol Dependence

A number of studies followed up children as young adults and assessed their experience of alcohol problems. Pulkkinen & Pitkanen,⁹² for example, found in a sample of Finnish children that aggressiveness at age 8 was predictive of problem drinking at age 26 for boys but not for girls; whereas social anxiety at age 8 was predictive for girls but not for boys. Similarly, in a community sample in New York, childhood aggression at ages 5-10, as assessed by anger, sibling aggression, noncompliance, temper, and nonconforming behavior, related to DSM-III-R alcohol abuse at ages 16-21.⁹³ Other evidence for the predictive power of childhood undercontrol comes from a birth cohort study of children from Dunedin, New Zealand,¹⁰ which found that boys (but not girls) who were under-controlled (impulsive, restless, distractible) at age 3 were over twice as likely as control children to exhibit a diagnosis of alcohol dependence at age 21. The one study that did not replicate the undercontrol findings was also a birth cohort study from New Zealand, the Christchurch Health and Development Study.⁹⁴ Here, conduct problems at ages 7-9 did not relate to DSM-IV alcohol dependence at ages 21-25. While it is impossible to know what the sample differences might be that led to these divergent findings, another review by Zucker³ of six other longitudinal studies, some population based, some high risk, indicates that the relationship of undercontrol to adult alcohol problem use is extraordinarily robust (all six studies replicated the finding), which in turn suggests that the Ferguson et al findings are anomalous.

However, it still remains to be determined which facets of undercontrol are responsible for this predictive relationship. Although the undercontrol relationship is a robust one, other facets of cognitive control also appear to predict the early drinking outcome. Thus, a study of males (n=122) recruited in antenatal clinics in a small community outside of Stockholm⁹⁵ found that lower ability to concentrate at age 10 and lower levels of school achievement at age 10 related to hazardous use of alcohol prior to age 21 and at age 36. (Hazardous use was defined on the basis of police register data on public drunkenness and drunken driving, and high levels of reported alcohol intake.) Similarly, as noted earlier, Nigg et al.,⁶⁰ found that poor response inhibition also predicted early initiation of drunkenness and problem use, even after conduct problems (as an index of behavioral undercontrol) was controlled.

In addition to individual difference factors, early contextual influences also predict later problem alcohol use. Data from the New York Longitudinal Study⁹⁶ show that parental conflict over childrearing and maternal rejection of the child, both assessed at child age 3, were significant predictors of greater (more severe) alcohol involvement at child age 19.

The work of Guo and colleagues⁹⁷ extends the conceptual framework of predictors in a more integrated fashion. They used a “Social Development Model” that included both individual difference, familial, and neighborhood factors to predict alcohol use disorder outcome in adulthood. They assessed internalizing disorders, externalizing disorders, male gender, delinquency, unclear family rules, poor family monitoring, less bonding to school, living in a neighborhood with more troublemakers, having antisocial friends, having friends who drink frequently, bonding to antisocial friends, higher intentions to use alcohol, and more favorable attitudes toward alcohol at age-10. They found that a model integrating all of these factors was predictive of DSM-IV alcohol abuse and dependence at age-21.

Childhood Predictors of Adult Alcohol Use and Disorders

Studies linking childhood data to follow-up data collected later than young adulthood are rare. In the Terman Life-Cycle Study, low conscientiousness and high sociability ratings at age 12 related modestly to alcohol involvement at ages 40-50.⁹⁸ Among Hawaiian elementary schoolchildren followed up at average age 45, higher teacher ratings of Extraversion and lower ratings of Emotional Stability were associated with greater adult alcohol intake.⁹⁹ Cloninger et al.¹² reported that Swedish children who were rated as higher on novelty-seeking and lower on harm avoidance and reward dependence at age 11 were more likely to be involved in alcohol abuse at age 27 (as defined by registration with the Swedish Temperance Board, arrests for drunkenness or driving while intoxicated, or treatment for alcoholism). In the Danish Longitudinal Study of Alcoholism,¹⁰⁰ measures of motor development in the first year of life (muscle tone at day-5, inability to sit without support at age 7 months, and inability to walk at 1 year) related to age-30 diagnosis of alcohol dependence. In the age-42 follow-up of African-American children first studied in first grade as part of the Woodlawn Study,¹⁰¹ a diagnosis of adult alcohol abuse or dependence was associated with lower math achievement scores in first grade and lower ratings of shyness for boys only, and with mother's regular alcohol use for both genders. Lastly, in the Stockholm antenatal study referred to above, Wennberg & Bohman⁹⁵ also found that psychologist ratings at age 4 predicted outcomes not just at the end of adolescence, but also well into adulthood. Ratings of extrovert/aggressive at age four correlated .27 ($p < .05$) with frequency of intoxication at age 25, while ratings of extrovert/outgoing correlated .22 ($p < .05$) with lifetime alcohol problems to age 36.

ALCOHOL-SPECIFIC RISK FACTORS IN CHILDHOOD

In contrast to the prior section, the focus here is on those *alcohol-related* factors that predict risk for later alcohol use and abuse as well as those that predict *actual drinking* and drinking outcomes. Although these two sets of variables often overlap, they are not always the same.

This section presents data on the rates of alcohol use among children, on the development of alcohol-related beliefs and expectancies in childhood, on the social contexts encouraging children to use alcohol, and on the several mechanisms through which children in alcoholic families are at risk of early onset and later problems.

Societal Levels of Alcohol Use

Alcohol is the most used and also the most abused drug in American society. According to the most recent data available on the NIAAA web site,¹⁰² the great majority of American adults (75.3%) have tried alcohol at some point in their lives, and a clear majority (61.1%) have had a drink in the past year (42.1% are light drinkers, 14.2% are moderate drinkers, and 4.8% are heavier drinkers). Men are more likely than women to be current drinkers (67.6% versus 55.1%), and substantially more likely to be moderate drinkers (21.6% vs. 7.3%, respectively) or heavier drinkers (5.6% vs. 4.0%, respectively). College graduates are more likely to be current drinkers than are adults with less education. Non-Hispanic Whites and Hispanics are more likely to be current drinkers than are non-Hispanic Blacks and Other non-Hispanics. Adults living in the South are more likely to be abstainers than adults from other regions of the country.

Data from the recent (2001-2002) National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) show substantial variation in the rates of current alcohol abuse and alcohol dependence across subsets of the American population.¹⁰³ Overall, 8.5% of Americans exhibited either alcohol abuse or dependence. Alcohol dependence in the past twelve months was seen among 5.4% of men and 2.3% of women. Whites, Native Americans, and Hispanics had significantly higher rates of alcohol dependence (3.8%, 6.4%, and 4.0%, respectively) than did Asian Americans (2.4%). Although religious background is not covered in federally-sponsored surveys, alcohol dependence has historically been higher among Catholics and liberal Protestants than among fundamentalist Protestants and Jews. As a result of this variation in adult alcohol use and abuse, children's exposure to alcohol use in the home also varies as a function of region of the country, parental education, religious denomination of the parents, and ethnic/racial background.

Rates of Alcohol Use and Abuse among Children Lifetime Alcohol Use

There is currently little good information on how many children have ever had experience with alcohol, either from retrospective recall by adolescents or from surveys of children themselves. Retrospective reports of the age at first drink are not very reliable for this life stage. Age of onset generally increases the older the adolescents being questioned.^{104,105} For example, in the most recent national data from the 2005 Youth Risk Behavior Survey, the percentage saying they drank alcohol before age 13 decreased from 33.9% for 9th grade students to 19.3% for 12th grade students.¹⁰⁶ That these are not in fact cohort effects, but rather evidence of "forward telescoping," is shown by the fact that similar percentages of students in these grades reported drinking before age 13 in each of the five previous surveys (1995, 1997, 1999, 2001, 2003) that asked this question. Similar findings are also obtained in the national Monitoring the Future surveys comparing 8th graders and 12th graders across multiple annual surveys on their reported incidence rates of alcohol use by sixth grade¹⁰⁷ (see Chapter 6). Thus, average or modal age of initiation of alcohol use based on retrospective recall is problematic. It varies depending on the age of the population sampled, the number of years since initiation, and the age categories presented as responses, and cannot be used with any confidence to characterize the level of current alcohol use in the child population. Surveys of children asking about current or recent drinking are more likely to capture normative data on ages of onset than are retrospective recall reports provided by adolescents or adults.

Large-scale epidemiologic surveys of alcohol use that include children aged 10 and under however, are extremely rare. According to the most recent Partnership Attitude Tracking Study (PATS; sponsored by the Partnership for a Drug-Free America), which surveyed a national probability sample of nearly 2,400 U.S. elementary school students in 1999, 9.8% of 4th graders, 16.1% of 5th graders, and 29.4% of 6th graders had had more than just a sip of alcohol in their life.¹⁰⁸ Recent data on the use of alcohol in the past year (rather than lifetime) has been reported annually by PRIDE Surveys (Dr. Thomas Gleaton, Director; see www.pridesurveys.com).¹⁰⁹ According to the 2003-2004 summary¹¹⁰ of school district surveys performed across the U.S., 4.2% of 4th graders, 5.6% of 5th graders, and 8.7% of 6th graders had had a beer in the past year. Slightly more had had wine coolers (4.4%, 6.7%, and 10.3%, respectively), and approximately half as many reported drinking liquor in the past year (1.9%, 2.8%, and 5.2%, respectively). These data, while based on a large sample of children from many school districts across the country, reflect a convenience sample rather than a representative national sample, and hence contain an unknown level of bias. Clearly, national surveillance efforts need to be directed toward monitoring the alcohol involvement of children starting in grade four. Initiating surveillance at grade 8 (as in the Monitoring the Future study) or grade 9 (as in the Youth Risk Behavior Survey) or at age 12 (as in the National Survey of Drug Use and Health) is simply too late.

Comparison of American and European children's experience with alcohol is possible to a very limited extent. Information on the number of U.S. 11-year-old children who have at least tasted alcohol comes from a World Health Organization survey of health behavior in 11-, 13-, and 15-year-old schoolchildren carried out in 1997-98 on 120,000 students in 28 countries in all parts of Europe as well as Canada and the United States.¹¹¹ Rates of having at least tasted alcohol vary widely across countries. The percent having at least tasted alcohol (averaged across genders) ranged from 91% of 11-year-old children in Slovakia to 85% in Scotland, 78% in England, 73% in Ireland, 71% in Sweden, 69% in Canada and Greece, 63% in Germany, 59% in Austria, 57% in Poland, 52% in France, 44% in Israel, 40% in Switzerland, and about 35% in Norway. Ever experience with alcohol was reported by 62% of 11-year-old boys and 58% of 11-year-old girls from the U.S.. These U. S. prevalence rates ranked 16th among the 28 countries studied. In most countries, more male than female 11-year-olds had at least tasted alcohol.

Alcohol Problems in Children

Although there are anecdotal reports and clinical reports of child alcoholics,^{112,113} the little available evidence suggests that few children exhibit problematic levels of involvement with alcohol such as alcohol abuse or dependence. In the few studies that have examined this, the incidence of diagnosed alcohol use disorders at age 12 and under is close to zero in the general population.¹¹⁴ Subclinical levels of alcohol problems are somewhat more prevalent in childhood. For example, Chen, Anthony and Crum¹¹⁵ found that 4.8% of fifth graders in Baltimore had already experienced one or more alcohol problems.

Early Alcohol Use Onset as a Risk Factor for Later Problems

It is critical to develop better information on the extent of alcohol experience among American children because younger ages of onset of alcohol use are associated with a greater likelihood of developing both problem drinking in adolescence^{116,117,118,73} and alcohol abuse or dependence in adulthood.^{119,120,121} While studies do not agree on whether it is alcohol use in childhood (age 12 and under) or in early adolescence (at age 13 or 14) that carries greater risk, they do agree that early alcohol use predicts later problematic drinking. Given this linkage between the early onset of drinking and later alcohol problems, it is also crucial to develop a better understanding of the factors that influence the initiation of alcohol use within childhood.

In addition to the increased risk for later alcohol problems, early onset drinking has been shown to relate to a variety of other problematic outcomes. Onset of drinking by age 10-12 is also associated with absences from school, drinking and driving, and marijuana and other illicit drug use in grade 12.¹¹⁸ Onset by grade-7 (ages 12-13) has been found to relate to more school problems, more delinquent behavior, more smoking, and more illicit drug use in grade 12 than later onset, and to smoking, illicit drug use, drug selling, and criminal behavior at age 23.¹¹⁶ In a follow-up study in grade 10, those who onset drinking by fall of 7th grade reported more recent drinking, drunkenness, and alcohol or drug problems, and were more likely to have initiated sexual intercourse, to have had more than 2 partners, and to have gotten pregnant (or gotten someone pregnant).¹²²

Methodological problems in this area have to do with the use of retrospective reports, variability in the definition of “early onset” across studies, the apparent use of age of onset as a substitute or proxy for examination of a larger array of alcohol “landmark” behaviors that may be of relevance for later alcohol problems (e.g., regular use, first drunkenness), and the absence of questions on context of first use (e.g., use as part of religious services or ceremonies, with family members, or with friends).

Development of Children's Beliefs and Expectancies about Alcohol

The developmental process through which children's attitudes toward alcohol are transformed from “tastes yucky” to “tastes great, less filling” has been largely unexplored. Relatively little is known about the milestones along this transition in orientation toward alcohol.¹²³

Preschool children's ability to identify alcoholic beverages by smell increases with age, and is associated with the level of alcohol use by their parents.^{124,125} This ability increases throughout childhood, with greater accuracy of identification with age from age 6 through age 10.^{126,127}

By age 6½-7½, the majority of children can demonstrate the concept of “alcohol” by correctly labeling photographs of bottles of alcoholic beverages and by being able to explain the difference between clusters of bottles of alcohol and other bottles. Younger children (aged 4½-6½), while they could label individual bottles correctly, could not explain how the bottles were grouped. Fossey¹²⁷ replicated the original bottle grouping task used by Jahoda & Cramond¹²⁶ and found that older children did better than younger children at grouping actual bottles.

Learning about alcohol in childhood involves more than identifying it by smell or grouping bottles. Children also learn that alcohol use is an activity in which adults typically engage. An early form of alcohol expectancies consists of “alcohol schemas,” as measured at child ages 3 to 5 by a task in which children were presented with drawings of child and adult figures in common social situations (e.g., two adults on a sofa in front of a fireplace, a family eating dinner, a man watching TV), and were asked what kind of beverage the figures were drinking (alcohol vs. non-alcohol).¹²⁸ Alcoholic beverages were attributed to adults more often than to children pictured and to men more than to women. In other words, even while in preschool, children already know the norms about drinking in the adult culture, namely that adults drink alcoholic beverages and children don't, and that men drink more than women! Furthermore, as might be anticipated, these drinking attributions were more evident for children of alcoholics than for children of nonalcoholics.¹²⁸

A more recent study demonstrates similar alcohol schemas using a “shopping” paradigm.¹²⁹ Children aged 2 to 6 were observed role playing as adults shopping for a social evening with friends in a miniature grocery store stocked with 73 different products, including beer,

wine, and cigarettes. Sixty-two percent of the children bought alcohol for this adult situation, and those with parents who drank at least monthly were more likely to do so.

Affective components of children's alcohol schema also vary as a function of age. Studies of normal samples over a twenty-year period show that children's ratings of adults depicted drinking alcohol are basically neutral at age 6 and become more negative up through age 10.^{126,127} Further research using this same paradigm shows these attitudes become more positive between ages 10 and 14.¹³⁰ Between third and seventh grade, significantly more children say it is "okay" for people to drink alcohol.¹³¹

Children have also been shown to have definite beliefs about the characteristics of drinkers and the behavioral effects of drinking by age 10.^{127,133,134} Females, particularly in the younger grades (kindergarten and third grade), were found to provide more coordinated, psychological, and causal responses than males when asked to explain why men and women described in the vignettes were drinking.¹³³

Children's expectancies about the effects of alcohol on drinkers are also generally negative¹²⁴ but become more positive as the children get older¹³⁴ and as they move into adolescence.¹³² Early expectancies emphasize the affective dimension (positive versus negative) with effects such as wild, dangerous, rude, and goofy being chosen, while later developing expectancies incorporate a pharmacological dimension (sedation versus arousal), exemplified by wild, dangerous, talkative, and cool.¹³² In the age range from age 8 to age 12, both positive and negative expectancies increase concurrently, presenting evidence of increasing ambivalence regarding the effects of alcohol.¹³¹ Positive expectancies have been shown to predict onset of drinking in adolescence,¹³⁵ although studies linking child expectancies to adolescent drinking are currently lacking.

These data suggest that even though relatively few children have initiated drinking at these ages, there is development of attitudes, beliefs, and expectancies that place them at increasing risk for movement into alcohol use.

Childhood Social Contexts that Facilitate Drinking

Within childhood, children are exposed to alcohol use by a number of social mechanisms. These include drinking by their parents and other adults in the family context, as well as alcohol use by adolescents and adults that is portrayed in the mass media (television, movies, print media, advertising). In the absence of their own experience with alcohol, this vicarious learning is the major influence on their attitudes toward alcohol and their expectancies about the effects of drinking.

Home

Parents constitute the major source of children's exposure to alcohol use. Research over the past 40 years is consistent in indicating that children are more likely to eventually become drinkers if their parents are drinkers. Among children, self-reports of alcohol use correlate significantly with child perceptions of parent drinking.¹³⁶

In addition to modeling alcohol use through their own drinking, parents increase the likelihood of their child's drinking through having alcohol available and accessible in the home as well as through active encouragement of child experimentation with alcohol. Research has shown that when children are asked where they got their first drink of alcohol, they overwhelmingly cite their parents or home as the source. For example, among third- through sixth-grade children participating in 1993-94 in the Bogalusa Heart Study who had ever tried alcohol, the majority first tried it with someone in the family (78%), 8% tried it alone, 8% tried it with someone their own age, and 6% tried it with someone older than themselves.¹³⁷ Fifty-six percent reported

that they got the alcohol from someone in their family, 32% drank out of someone else's drink, 6% took it from home, and 6% got the alcohol from another kid (see also 135,131). In a recent community survey of children in Oregon, 138 few of the children, especially in the younger grades (1-4), who had ever tried alcohol had done so without their parents' knowledge. There is currently little research on subcultural, religious, or regional variation in parents' beliefs about the appropriateness of introducing their children to alcohol in the home.

Mass Media

In addition to their observation of parental drinking, children learn about alcohol use and its effects through their exposure to movies and television content as well as through their exposure to advertisements. The alcohol industry spends over \$1.6 billion a year on advertising in radio, television, magazines, newspapers, billboards, etc.¹³⁹ The alcohol industry routinely exposes adolescents aged 12-20 to high levels of alcohol advertisements through their placement of ads at times when adolescents are most likely to be watching or listening, in magazines they are likely to read, on radio stations to which they are more likely to listen, and during television programs in which they are more likely to be interested. For example, in 2003, teens saw twice as many beer ads, more than three times as many ads for alcopops (sweet flavored alcoholic drinks), and 50% more spirits ads in magazines on a per capita basis than did adults aged 35 and over.¹⁴⁰

There is less evidence, however, that children are exposed to alcohol advertisements to the same degree as adolescents. First, magazine and radio audience data do not include children under 12, so their exposure to alcohol ads in these media cannot be measured. Second, children under 12 may be less exposed to magazine ads due to their reading levels and reading choices (books rather than magazines, or magazines having advertising restrictions). Third, another recent Center on Alcohol Marketing and Youth report from July 2005 suggests that children aged 2 to 11 are underexposed to alcohol advertisements on television relative to their prevalence in the overall population. They are exposed to less than half as many television alcohol advertisements as are 12-20 year olds. This does not mean, however, that they are not exposed: on average, children aged 2-11 saw 99.4 alcohol advertisements on television between January and October of 2004 (81% for beer and ale, 11% for spirits, 5% for alcopops, 3% for wine, calculated from their table 3). At this rate, the average child could have seen almost 1,200 alcohol advertisements on television before age 12 (assuming a similar rate across years).

Alcohol advertisements are not the only source of alcohol portrayals on television. Portrayals of alcohol use and its (lack of) consequences are pervasive on television programs aired in prime time (8-11pm) when children may be watching. Estimates from the 1998-99 season were that 71 percent of sampled episodes included alcohol use by characters on the shows.¹⁴¹ Most disturbing is that 38 percent of shows with a TV-G rating (appropriate for most children) depicted alcohol use. More episodes characterized drinking as a positive experience than a negative experience. Negative consequences were portrayed or mentioned in only 23 percent of episodes.

Children's animated films have also been analyzed for alcohol content. All G-rated, animated films released by five major studios between 1937 and 1997 and that were available on videotape were reviewed for episodes of tobacco and alcohol use.¹⁴² Of the 50 films reviewed, 50 percent included alcohol use that was portrayed by 63 characters for a total of 27 minutes. Seven of the 50 films depicted effects of alcohol use (e.g., drunkenness, passing out, losing balance, falling), but none addressed any of the negative health consequences of alcohol use.

In a study of fifth and sixth grade students,¹⁴³ greater awareness of beer advertisements (ability to correctly identify brand names of still photographs from current television commercials) significantly related to greater intentions to drink as an adult through its relation to more

positive beliefs about alcohol (a mediated path). A recent study of 10-14 year old non-drinkers found that level of exposure to alcohol use in motion pictures predicted whether they were drinkers 1-2 years later.¹⁴⁴ Considerably more research is necessary, however, to determine the linkage of media exposure to drinking on children's initiation of alcohol use. Of major importance is determination of the impact of media exposure as a function of parental modeling of alcohol use in the home.

Children in Alcoholic Families: A Special Early Risk Population

According to National Longitudinal Alcohol Epidemiologic Survey (NLAES) data,¹⁴⁵ approximately 9.7 million children age 17 or younger, or 15 percent of the child population in that age range, were living in households with one or more adults classified as having a current (past year) diagnosis of alcohol abuse or dependence. Approximately 70 percent of these children were biological, foster, adopted, or step-children. That is, 6.8 million children meet the formal definition of children of alcoholics (COA), although not all are exposed to the same level of risk for use, problem use, and alcohol use disorder (AUD). So far as socialization risk is concerned, these figures reflect only acute (past-year) exposure to at least one alcoholic adult. According to other data from the NLAES survey, 43 percent of the under-18 population, or slightly less than half of all children, were exposed to either a currently or previously alcoholic adult in the household. The figure for just COAs is 30 percent of the under-18 population, but even this represents an enormous population at risk. The sheer size of this group indicates that any approach to risk identification will be extremely complex politically, and will need to considerably differentiate the risk variability among these families. At the same time, it is essential that this be done, given the magnitude of the problem. Children of alcoholics are between 4 and 10 times as likely to become alcoholics themselves.¹⁴⁶ Prior to that, they are also at elevated risk for earlier drinking onset¹⁴⁷ and earlier progression into drinking problems.¹¹⁹

Genetic Risk

Although the observation that alcoholism runs in families has been known for centuries, it has only been within the past generation that definitive studies have been carried out. These have involved children with an alcoholic biological parent who were raised by non-alcoholic adoptive parents,¹⁴⁸ thus enabling a test of the separate influence of genetics and environment on the development of alcoholism. Despite the lack of modeling of alcohol abuse in the home, these adopted children were still significantly more likely to develop alcoholism later in life than were control children with no genetic risk for alcoholism.

While studies such as this establish the baseline relationship of family risk to later disorder, ongoing research is working to identify the specific aspects of genetic risk that produce this outcome and to identify environmental factors that moderate or mediate the influence of genetic risk for alcoholism. It is essential to keep in mind that some of the elevated risk is attributable to exposure and socialization effects found in alcoholic households, some to genetically transmitted differences in response to alcohol that make the drinking more pleasurable and/or less aversive, and some is attributable to elevated transmission of risky temperamental and behavioral traits that lead the COA into greater contact with earlier and heavier drinking peers.

Factors Involved in Familial Transmission

Familial alcoholism status (being "family history positive" or FH+) is heavily used as a proxy for "alcoholism risk" on the one hand, and "socialization risk" on the other, but the familial designation is more precisely a proxy for multiple but more specific risk factors, not all of which may be present in all cases. Thus, being FH+ implies elevated genetic risk, on average, although the alcoholic genetic diatheses may not have been passed on to a particular child. One

may be a child of an alcoholic (COA) without being undercontrolled, having an attention deficit hyperactivity disorder (ADHD) diagnosis, etc.

Socialization risk involves familial exposure, but given the high divorce rates in this population, evaluating the level of socialization risk is complex. This is because it involves quantification not only of how long the exposure to the actively alcoholic parent has been, but also the developmental period during which the exposure took place. Some developmental periods have the potential to produce more vulnerability than others.¹⁶ In addition, a substantial amount of assortative mating occurs in alcoholic families,¹⁴⁹ that is, alcoholic men often marry women with alcoholism. When assortment is present, risk exposure is multiplied, and COA effects become a function of genetic risk(s), individual parent risk, as well as the synergistic risk created by impaired marital interactions.¹⁶

The potential for indirect socialization effects is also higher in alcoholic families. Parental psychopathology has been documented as a risk factor for poorer parental monitoring¹⁵⁰ which in turn leads to a higher probability of involvement with a deviant peer group, including earlier exposure to alcohol- and other drug-using peers.

At the same time, COA risk is not simply risk for the development of an alcohol use disorder (AUD).¹⁵¹ Given what is known about the elevated psychiatric comorbidities among offspring of alcoholics, being a COA is also a marker of elevated risk for a variety of behavioral and cognitive deficits. These include attention deficit disorder, behavioral undercontrol/conduct disorder, delinquency, lower IQ, poor school performance, low self esteem, and others.¹⁵² Furthermore, the evidence strongly implicates some of these non-alcohol specific characteristics as being causal to both problem alcohol use and elevated risk for AUD.^{11, 153} Thus, in a community study of high-risk families, Wong et al.⁷⁶ found that parental alcoholism was a significant predictor of early-onset alcohol use and drunkenness (both by age 14), but that early sleep problems, possibly an indicator of a central dysregulatory deficit, was an independent predictor of this outcome. Similarly, in another longitudinal study following boys with and without parents with a substance use disorder (SUD), Tarter et al.⁸ found that the effect of the father's and mother's SUD on a the son's diagnosis of SUD at age 19 was mediated by neurobehavioral disinhibition (operationalized as the sum of disruptive behavior disorder symptoms on the SCID), social maladjustment, and drug use frequency at age 16. Other studies investigating the mediators of these effects include those of Hill and colleagues,⁹⁰ showing not only that children in the high-risk families had an earlier age of initiating regular drinking with negative consequences than did children from low-risk families, but also that this relation was mediated by the temperament variable of extraversion.

These factors implicate the COA population as a large and important component of the underage drinking population. It is essential to determine which components of that family risk envelope are the strongest mediators of the underage drinking outcome. Given the overlap of socialization and genetic risk in all of these studies, it is essential to determine which components of the risk designation are the strongest mediators of underage drinking, and which may be subsumed as proxies for other mechanisms. As specific genes are identified which carry alcoholism risk, investigators will be better able to model the interactions between social environment and genetic vulnerability that may very well be taking place here.¹¹ Such studies are essential.

Fetal Alcohol Exposure

An additional potential risk for early onset drinking as well as for the development of risk factors for later alcohol problems is the exposure of the child to alcohol in utero. Given the assortative mating that occurs, in which alcoholic men marry women with the same problem, some children will be affected not only by genetic risk and by socialization risk, but also by

risk arising from the teratogenic effects of alcohol exposure during fetal development. These teratogenic risks, however, can occur even at levels of alcohol intake during pregnancy that are not symptomatic of maternal alcoholism. While it is still not clear what level of alcohol intake is safe during pregnancy, research reveals that even relatively modest levels of alcohol intake can have negative effects on the developing fetus. Depending on the level of alcohol exposure and the timing (trimester) of exposure, these effects can be morphological, growth-related, neurological, and behavioral, and reflect a spectrum of alcohol-related neurodevelopmental disorders (FASD, or fetal alcohol spectrum disorders). Prenatal alcohol exposure effects on development have been extensively studied in both humans and animals. 153-160 Findings relevant to this report are the effects of prenatal exposure to alcohol on response inhibition, attention, executive functioning, delinquent behavior, and school achievement in childhood, all of which are themselves risk factors for later alcohol problems.

In 1974-75, as part of the Seattle Longitudinal Study on Alcohol and Pregnancy, 1,529 pregnant women were interviewed in their fifth month regarding their demographic characteristics, their nutrition, use of tobacco, alcohol, and caffeine, and their use of medications. In 1989-90, 464 families reflecting a continuum of mother's drinking during pregnancy were followed up when the children were age 14. Mother's alcohol intake during pregnancy, and hence the child's prenatal exposure to alcohol, significantly predicted adolescent experience of the negative consequences of drinking (i.e., personal and social difficulties resulting from alcohol use, such as getting into a fight, neglecting responsibilities, having a bad time), even when controlling for family history of alcoholism, for parental current drinking, and for several parenting variables. Family history of alcoholism was not a significant predictor when prenatal alcohol exposure was controlled for statistically. A later follow-up of this sample found that prenatal exposure to alcohol as well as family history of alcoholism predicted young adult (age 21-24) scores on the Alcohol Dependence Scale.¹⁶¹

There are a number of ongoing longitudinal studies of cohorts of children exposed prenatally to alcohol that will shortly have data on the adolescent alcohol involvement of these children (e.g., Nancy Day's Maternal Health Practices and Child Development Project) and that will soon be able to test the generality of these results. In the meantime, there is ample evidence that prenatal exposure to alcohol has effects on a number of risk factors for later alcohol abuse and dependence.

DEVELOPMENTAL UNFOLDING OF RISK AND RESILIENCE

Risk Aggregation

There is considerable evidence, both from the child and adult literatures that risks are correlated at the individual and familial levels as well as at the neighborhood level. At the individual level, the literature has increasingly acknowledged the clustering of comorbid symptomatology, social dysfunction, and alcoholism severity among adults¹⁶² and in fact such assortment has been one of the driving forces for the notion that subtypes of disorder need to be demarcated. In the same vein, the association of severe alcoholism with poverty has a long and visible history^{163,164} and analyses at the micro-environmental level have documented an association between neighborhood disadvantage and alcoholism rates.¹⁶⁵ The most common explanation of this has been that poverty, and the neighborhood structure in which it is embedded, drive the alcoholism (i.e., a top-down explanation). What has been less clear is the degree to which individual processes are also at work here. Some evidence suggests that there is, at least for children from antisocial alcoholic families. Thus, antisocial alcoholic men are more likely to marry/partner with antisocial and heavy drinking/alcoholic women.¹⁶⁶ The families they create are more likely to be disadvantaged in their capacity to socialize offspring. Antisocial alcoholism is also associated with downward social mobility,¹⁶⁷ and offspring in these families, even early in life, appear to be developmentally more disadvantaged; that is,

they have more learning disabilities and intellectual deficits than do offspring from alcoholic but not antisocial families.²⁷

A risk cumulation theory would suggest that as these factors continue to cumulate, they produce a risk structure that moves the child into peer networks high in aggression, negative mood, and substance use, thus providing a familial, a neighborhood, and a peer structure, all of which act in concert to encourage the development of (1) an expectancy structure that is positive toward use and abuse of alcohol and other drugs, (2) very early onset for such use, and (3) a stable repertoire of behaviors that are prototypic for the eventual emergence of abuse/dependence.

Research is needed to determine the degree to which such a risk aggregation structure is synergistic for the development of risk. For example, normative studies of adolescence have shown the enhanced effects upon drug use and the timing of onset when family conflict, association with deviant peers, and poor academic performance are clustered.¹⁶⁸

Resilience and Risk: Key Developmental Pathways

As indicated earlier in this review, considerable evidence indicates that later use can be predicted from developmental patterns evident well before 10 years of age, suggesting that children have already started down developmental paths leading toward early use and abuse of alcohol.^{108,150} In most cases, these paths also lead to other problems associated with alcohol use, such as smoking, drug use, delinquency, school drop out, or depression. In some cases, high risk pathways are so well established that these pathways are clear targets for preventive intervention,¹⁸ although it should always be remembered that these are probabilistic pathways and not certain roads to underage drinking. In fact, there are children who appear to be on the same pathways who do *not* begin to drink early or who take a turn for better development; such children serve as a powerful reminder that this is a risk pathway and not a “certainty pathway.” It is important to understand the processes leading away from this pathway as well as those processes leading children to continue down this road.

Two major pathways of risk for underage drinking (and other related problems of adolescence) are (1) the antisocial behavior (externalizing) pathway and (2) the emotional distress (internalizing) pathway.

The Externalizing Pathway

Evidence has mounted for some time that there are children who show early difficulties with self-control of impulses and attention, manifest unusually high levels of aggression during the preschool years, and develop early academic problems related to their behavior once they begin school.^{18,19,169} These children often live in disadvantaged families with poor discipline and few resources. Their parents often have mental health or behavior problems, including alcohol abuse or antisocial personality. They show multiple problems in multiple domains related to self-control and compliance. These children are often described as stress reactive, with high negative emotionality or difficult temperaments. During late childhood and early adolescence, a portion of these children disengage from school, begin to associate with deviant peers, engage in increasingly risky behaviors, and escalate in delinquent behavior. Sometime during the transition to adolescence, these youth are at high risk for early alcohol use as well as other behaviors in the problem-behavior spectrum of substance abuse, early and risky sexual activities, truancy, etc.

The Internalizing Pathway

A second pathway implicated by the longitudinal data on risk for underage drinking that may have its beginnings in childhood involves depressive-spectrum-disorder symptomatology and related antecedents, including anxiety and shy/inhibited personality.^{88,10} Evidence is weaker

for this internalizing pathway in relation to earlier alcohol use, though there appears to be a link between depression in adolescence and risk for alcohol initiation.⁸³ However, the evidence is considerably stronger for an internalizing pathway to alcohol use disorder.¹⁵⁰

Low-Risk Pathways

Implicitly, patterns of risk also implicate patterns of lower risk for underage drinking, though these pathways have not been as well-defined. Children with a track record of success in age-salient developmental tasks throughout childhood, with the benefits afforded by good self-regulation skills and effective parents, who handle stress well, engage and succeed in school, and associate with prosocial peers who engage in little risky or antisocial behavior presumably are on a low-risk path with respect to early alcohol use. One community high-risk study has documented this pathway for a group of children who start out with low levels of the risky externalizing and internalizing traits, who are born into lower family adversity environments. As shown in Figure 1, the pattern of adaptation for these “nonchallenged” children remains stably better from age 3 into their early teens.¹⁷⁰ Another group of children, who similarly began with low levels of the externalizing and internalizing traits but who were born into higher adversity, alcoholic and sometimes antisocial alcoholic homes, showed a similar pattern of relative stability of lower levels of impulsivity and aggressiveness through out early and middle childhood and early adolescence. These children were called “resilient” by Zucker et al.¹⁷⁰ However, they also showed some evidence of “weathering” over time, at least with regard to internalizing traits. Anxiety, sadness, and depression remained low during the preschool and early school years, then began to increase, approaching the levels found among more vulnerable children by early adolescence. The authors suggest that the exposure to high family adversity over long periods of time eventually “wore away” the earlier, more sunny disposition these children had when they were younger.

Protective Factors

In contrast to antecedent risk factors, there has been little attention paid to positive antecedent factors. Two kinds of positive factors have been delineated in the literature on risk, competence and resilience: *promotive factors* that are generally associated with better outcomes across levels of risk or adversity (main effects in statistical terms); and *protective factors* that are associated with better outcomes particularly in the context of higher risk or adversity (moderator effects in statistical terms)⁶¹. Some factors, such as parenting, have been widely implicated both as promotive and protective factors: considerable literature implicates good quality parenting as a promotive factor with respect to many positive developmental outcomes; at the same time, parenting quality appears to play a special protective role under very risky or hazardous conditions

Many of the most widely studied promotive and protective factors in human development are bipolar in nature, reflecting dimensions of variation along a continuum, with a desirable to undesirable range. Parenting is a classic example, because good parenting can be viewed as promotive or protective, and bad parenting can be viewed as a risk or vulnerability factor for underage drinking and many other outcomes among children. With continuously distributed predictors, it is often difficult to determine “where the action is” along a continuum. Distinguishing a risk from a promotive factor or a vulnerability from a protective effect is a challenging problem given that these may be arbitrary labels for one or the other end of a dimension that has influences on development across the range of observable differences. In studies where only a high-risk sample is examined, one cannot distinguish a promotive factor from a protective factor, or a risk factor from a vulnerability factor. Without a low-risk group, one cannot establish if the factor of interest has comparable effects across all levels of risk rather than a special role among high-risk people.

For alcohol use, factors that predict fewer problems would be viewed as promotive factors and factors that moderated the effects of risk or adversity on problem outcomes would be viewed as protective factors. For example, a protective factor may be associated with attenuated (lower than expected) alcohol-related outcomes for the general level of risk for alcohol use or AUDs present. Among children living in poverty in bad neighborhoods, surrounded by deviant peers who encourage underage drinking (where risk for underage drinking appears to be high), effective parenting may be particularly important and may have protective effects beyond the generally positive effects of good parenting on child outcomes. Relatively few studies in the alcohol literature have focused on establishing moderators of risk, particularly in longitudinal analyses for children under the age of 10. The data exist but the field has not yet aggressively addressed this issue.

DATA NEEDS RELEVANT TO POLICY IN THIS AREA

It is clear from the present review that there is a lack of national surveillance data on child and early adolescent alcohol use, covering children and preadolescents in grades 4 through 7. Extant data suggest that there are non-trivial numbers of children who have had some experience with alcohol in these grades. Instituting an ongoing series of nationwide surveys of children's alcohol experience is critical for a number of reasons. First, it is necessary to determine the prevalence of alcohol use in this population in order to monitor both the need for, and the success of, prevention efforts in the elementary schools. Second, alcohol use onset is one of the initial stages in the progression into illicit drug use.¹⁷¹ Knowing how many children have experience with alcohol thus serves as an indicator of the number potentially at risk for illicit drug use. Third, as noted above, childhood onset of alcohol use predicts alcohol problems in adolescence as well as alcohol abuse and dependence in adulthood.^{119,73}

While it is clear that early onset drinking is problematic, it is also clear that some parents believe children should be introduced to responsible alcohol use in a family context. The little research on this suggests that early onset is problematic whether it occurs in a family context or whether it occurs in a peer context.¹⁷² More research on whether and how onset context matters (e.g., family versus peer context) is very important. Moreover, this issue highlights how little is known about adult Americans' beliefs about the anticipatory socialization of alcohol use in childhood.

Additionally, while there once was a literature on cultural contexts of drinking and their influencing role (e.g., Irish, Italian, or Jewish traditions),^{173 174} there is little current research to indicate whether and how adult norms for child and adolescent drinking vary across ethnic, racial, and religious groups in the United States. Where there is sub-cultural support in the home for such drinking, it is unlikely that school-based prevention programs that ignore such influences will be effective. Similarly, little is known about protective effects of cultural traditions or contexts on the development of underage drinking.

IMPLICATIONS FOR INTERVENTION

There are a number of points prior to the initiation of alcohol use in childhood and early adolescence that are implicated in this review as candidates for different types of interventions. Findings indicate it is essential to consider these developmental pathways of risk. Thus, prevention efforts can target parents before conception, prenatally or at many points in child development, long before initiation of alcohol use by children. Based on this review, we recommend five target areas for intervention, as follows.

1. Treat alcohol problems in potential parents

Given the importance of genetic risk for alcoholism and socialization risks associated with alcohol problems in parents, adults with alcohol problems likely to become parents are an important target for intervention. Examples include: a) an emphasis on treatment for the alcoholic parent(s) to reduce the parent's problem drinking and thereby to reduce the children's exposure to such drinking in the home; b) provision of parental training to instill more effective parenting practices and to reduce instances of child neglect and maltreatment; and c) provision of marital/couples' counseling to ensure there is less conflict in the home. Such parental training and counseling should be offered as part of the parents' alcoholism treatment. The goal here is to make the intergenerational transmission of alcoholism less likely.

2. Boost efforts to reduce prenatal drinking in mothers

Prenatal exposure to alcohol is a risk factor for developmental anomalies such as FAS, and appears to be a risk factor for problem drinking in adolescence and young adulthood,¹⁶¹ although further research is necessary to confirm this. Given the growing evidence of multiple negative consequences of prenatal exposure to alcohol, prevention efforts need to focus on better education and dissemination of negative consequences of drinking during pregnancy, greater emphasis and dissemination of the need for antenatal care during pregnancy, better screening for women's alcohol use as part of antenatal visits, and greater referral of drinking pregnant women to effective alcohol interventions.

3. Include screening for alcohol use and alcohol risk behaviors in pediatric well-child visits

The previous review also implicates prenatal exposure to alcohol as a factor influencing the development of a variety of other risk factors for alcohol problems, including executive functioning deficits, inattention, poor academic performance, lowered response inhibition, and delinquent behavior (although genetic risk could also figure here). Pediatric well-child care should include screening for prenatal alcohol/drug exposure if antenatal care records are not passed along from the mother's obstetrician. Among those children identified as having prenatal exposure to alcohol, early childhood interventions should be instituted prior to school entry targeted on instilling child and parent behaviors that enhance child functioning in academic tasks, that enhance response inhibition, and that reduce inattention. Screening for ADHD should be part of such interventions, given the proven benefits of pharmacological agents for reducing problems associated with this disorder.

4. Address high-risk externalizing pathways early

Substantial evidence was cited earlier in our review for the role of externalizing disorders as a risk factor for earlier onset of drinking and the development of alcohol problems in adolescence. Relevant recommendations in this arena are the following: a) to develop better surveillance systems in the schools, pediatric medicine, social services, and public safety (police) to identify children already displaying evidence of such problems; and b) to develop a program to enhance or enable collaboration between alcohol researchers and other developmental researchers in allied fields who may already have successful prevention or intervention programs to reduce conduct problems in children and preadolescents.¹⁷ Important considerations would include determining what ages are likely to provide the most preventive “bang for the buck,” what are the most engaging and least stigmatizing venues for such interventions, and how best to reduce barriers to parental involvement in the programs.

5. Intervene early in pathways to deviant peers and promote pathways to prosocial peers

Although childhood onset of alcohol use is less likely affected by affiliation with deviant peers, this is a major risk factor for early adolescent onset of drinking and for movement into problematic drinking in adolescence. Although not reviewed here, affiliation with deviant peers

is associated with a variety of family risk factors, including harsh and inconsistent discipline, low levels of parental warmth, low parental support, less parental monitoring, and low parental attachment and identification. The seeds for later affiliation with deviant peers are thus sown early in the school years. Research suggests that the most effective interventions involve parent education in school family resource centers rather than child interventions that group and segregate children at risk.

A CONCLUDING COMMENT

This review has documented a host of factors and pathways evident during the “under-10 period” that influence risk for underage drinking and progression into problem use. Some of this research has been in the literature for more than a generation and much of it has been known for at least a decade. Despite the preponderance of evidence, it is still rare for researchers or clinicians to recognize that drinking problems of youth have their beginnings well before alcohol use is initiated. Why would this be the case? Two possibilities occur to us. One is the failure to understand that nonspecific risk factors are at least as important as the alcohol-specific risk factors in the early stages of a drinking career, especially when the focus is on understanding what creates risk for onset. The second possibility is that most researchers and clinicians are more comfortable with proximal causes, with the result that more distal developmental connections between early/middle childhood and adolescence are to a large extent ignored or dismissed. Whatever the explanation, the evidence presented in this review provides a compelling rationale for expanding the causal model for the development of drinking risk into the earlier childhood years, and into the parental context that surrounds them.

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Child Psychopathology in Preschool

		In Normal Range	High
Family adversity	Low	Non-challenged	Troubled
	High	Resilient	Vulnerable

A 2X2 matrix of child individual differences in psychopathology and family adversity during the preschool years

Figures 1A-1C.

Typology of Family/Risk and Adversity and changes in externalizing and internalizing symptoms in the different risk/adversity groups over time

Table 1
Developmental Periods, Transitions, Contexts, and Tasks of the Under-10 Period

Developmental Periods (and Key Transitions)
 Prenatal (Conception)
 Infancy (Birth)
 Toddler and preschool years (Upright locomotion, preschool entry)
 Middle childhood (Transition to elementary school)
 Key Contexts of Development
 Prenatal environment
 Caregiver relationships
 Family
 Daycare
 Preschool
 Kindergarten
 Primary grades of elementary school
 Peer play and activity groups
 Friendships
 Media
 Neighborhood
 Developmental Tasks and Issues
 Attachment
 Understanding and speaking the language of the family
 Understanding, speaking, reading and writing the language of the culture/school
 Sitting, walking, skipping and other developmental motor milestones
 Compliance with rules for conduct and impulse control
 Toilet training
 Playing with peers
 Acceptance among peers in key community or school contexts
 School adjustment
 Learning to read