

SUPPLEMENT ARTICLE

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

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ABSTRACT

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Developmental pathways to underage drinking emerge before the second decade of life. Many scientists, however, as well as the general public, continue to focus on proximal influences surrounding the initiation of drinking in adolescence, such as social, behavioral, and genetic variables related to availability and ease of acquisition of the drug, social reinforcement for its use, and individual differences in drug responses. In the past 20 years, a considerable body of evidence has accumulated on the early (often much earlier than the time of the first drink) predictors and pathways of youthful alcohol use and abuse. These early developmental influences involve numerous risk, vulnerability, promotive, and protective processes. Some of these factors are not related directly to alcohol use, whereas 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 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 and the 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.

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Key Words

aggregation of risk, susceptibility, alcoholism, early childhood risk

Abbreviations

SUD—substance use disorder
AUD—alcohol use disorder
ADHD—attention-deficit/hyperactivity disorder
COA—child of alcoholic

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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. First, 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 Family history of antisocial behavior, child maltreatment, and other negative life experiences are well-established precursors of later alcohol problems and alcohol use disorders (AUDs). 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.

Second, at the same time that children develop behavior problems not specific to alcohol, they acquire knowledge about the existence of alcohol as an object in the social environment. Learning about alcohol includes developing beliefs about alcohol on the basis of an awareness of its special characteristics as a drug (how it produces changes in cognition, feeling, and behavior) and its place in social relationships (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.

Third, in tandem with the development of behaviors and beliefs related to alcohol, other developmental changes that influence behavior occur within the individual and in the social context. As the brain is developing, consumption of

beverage alcohol interacts with changing brain structures and functions related to appetite, reward, planning, and affective and behavioral control. These neurobehavioral processes proceed from the interplay of genes and experience, in many cases operating through intermediate endophenotypes.⁴ The latter are traits or biological indicators that are genetically simpler than the diagnostic phenotype and are more proximal to the genetic influence but are part of the vulnerability pathway for the disorder.

Fourth, 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 the situations in which it is unacceptable, and these social norms have been incorporated into legal norms that specify the appropriate sanctions for violations of alcohol use regulations.

Fifth, this multilevel dynamic interplay of biological, psychological, and social processes shapes not only risk but also normal development.⁵ Normal development has the potential to alter risk parameters and pathways of behavior profoundly and even to move at-risk children 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 before the second decade of life). We recognize that puberty may be well underway for some young people <10 years of age; however, our focus is on early and middle childhood and processes that generally precede pubertal development and the social changes that characterize adolescence.

BRIEF DEVELOPMENTAL PORTRAIT OF THE TIME BEFORE 10 YEARS OF AGE

The years before 10 years of age encompass all of 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, day care, 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 (Table 1).

The pace of development during the first 10 years of life is astonishing, from conception to fetuses 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, in structure, organization, and function, over this period. 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, includ-

TABLE 1 Developmental Periods, Transitions, Contexts, and Tasks of the Time Before 10 Years of Age

Developmental periods (and key transitions)
Prenatal (conception)
Infancy (birth)
Toddler and preschool years (upright locomotion and preschool entry)
Middle childhood (transition to elementary school)
Key contexts of development
Prenatal environment
Caregiver relationships
Family
Day care
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

ing 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. By 10 years of age, however, 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 and human and social capital they have accumulated in childhood, as well as their record of achievements and failures in meeting the various developmental tasks of childhood. Therefore, it is not surprising that many of the influential factors associated with early drinking emerge and are shaped during the first decade of life.

This review has 6 sections. In the first section, we describe how core developmental processes, such as behavioral and emotional dysregulation, function as predisposing risk factors for youthful alcohol use. In the second section, we review other non-alcohol-specific risk factors that enhance drinking risk. In the third section, we describe alcohol-specific risk factors in childhood that are associated with subsequent alcohol use. In the fourth section, we summarize what is known about risk and resilience developmental pathways, either to-

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ward or away from problematic alcohol use. In the fifth section, we describe briefly the next-step tasks needed for the formulation of policy in this area. In the sixth section, 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 AUDs.

NONSPECIFIC CHILDHOOD RISK FACTORS

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

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Emergence of Behavioral and Emotional Dysregulation and Predisposition to Alcohol Involvement

Newborns emerge into the world with cries of greater or lesser intensity, lasting for shorter or longer periods of time, and with quicker or slower responses to the caretaking agents who attempt to soothe and to comfort them. If the comforting (feeding, handling, and being engaged by the caretaker) is sufficient, then the infant begins to display signs of satisfaction and relaxation. If it is not, then the affective expression continues. The display of emotion, 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 one hand and pleasure, comfort, and happiness on the other. These are basic temperamental characteristics that serve signaling (communicative) functions, facilitate social engagement, and serve as organismic motivators either to sustain current activity or to 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 there are fetal differences in activity levels even before birth, 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, sound, and touch. Some respond more quickly 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 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 achievement and academic achievement are more difficult, and risk for substance use disorders (SUDs) is substantially elevated. In fact, one of the most prominent theories for the development of AUDs and SUDs posits the importance of a central dysregulatory trait, involving delayed or deficient development of behavioral, emotional, and cognitive regulation, in the early emergence of SUDs.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}

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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 understand fully the mechanisms of impact of behavioral regulation and attentional control, these domains seem 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. The choice to use alcohol for the first time (ie, drinking onset) is a cognitive choice (regarding whether this is a wise act and what the consequences are of doing so at any particular time). 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.

Substance abuse researchers have been aware of these non-alcohol-specific processes for some time, which has led to the search for the traits that underlie them. In the past 20 years, an increasing amount of evidence from longitudinal studies has identified 2 such traits that are detectable very early in life, that predict alcohol (and to some degree other drug) involvement, and that seem to be markers of an underlying genetic diathesis for early use, heavy use, problem use, and AUD. This work, coming from 6 long-term prospective studies,¹¹⁻¹⁵ provides a remarkable convergence with the genetic literature in demonstrating that externalizing (aggressive, impulsive, and undercontrolled) and to a lesser degree internalizing (anxious, sad, and depressive) symptoms appearing in early childhood are predictive of SUD outcomes 15 to 20 years after the first appearance of the non-drug-specific behavioral risk (see ref 3 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 being the most likely to develop the more-chronic and more-severe forms of SUDs in adulthood.^{18,19}

Neurobiological and Cortical Development of 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 multicomponent construct, including such elements as response suppression/inhibition (the ability to suppress strategically a prepotent or prepared motor response), working memory (itself multicomponent), set shifting (shifting from one task set or "set of rules" to another), and interference control (inhibition of a relatively dominant response system to allow another system to operate).²¹ These capacities are represented to a large degree in parallel frontal-subcortical-thalamic neural loops. Important structures include the 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 increase or decrease risk. They relate to wisdom in choice of peers, understanding of 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 to function adequately).

Extensive theories as long as a generation ago attempted to link aspects of executive control to alcoholism risk, but findings supporting this linkage have been mixed.^{7,24–26} More-recent work²⁷ suggests that the risk element is related primarily to response inhibition. In addition, Finn et al²⁸ 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 to test) models with a higher level of specificity.

Closely intersecting these processes is the domain of motivation, particularly reward responsivity. 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 responses influence one another,⁶ both in development and dynamically. Extensive research suggests that at both the behavioral and neural levels, substance use problems are associated with dysregulation of reward responsivity, such that the subcortical involuntary elements (subserved by limbic and striatal circuitry) over-respond 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 dysregulation of reward-related circuitry in at-risk populations even before alcohol and illicit drug use occurs.³²

During the developmental period in which alcohol use and alcohol problems escalate, neural alterations occur 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 into early adulthood.³³ Progressive increases in the white matter of this region during childhood and adolescence have been demonstrated.³⁴ These developmental changes directly affect impulse and emotion regulation. It is known that, throughout childhood, there are developmental gains in the ability to suppress or to 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 period 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 responses.^{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 frontolimbic and frontostriatal systems that are implicated in impulse control and reward responsivity.^{42–44} Such effects are thus superimposed on this developing circuitry. Major issues not yet addressed concern the relative importance of the amount and timing of alcohol (and other drug) exposure in bringing about such changes, the degree to which other environmental exposures (eg, 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 requires a multilevel/multisystem explanatory structure.

Genetics of Dysregulation

The strong evidence reviewed above for temperamental individual differences in behavioral regulation and control

is paralleled at the genetic level by evidence from a number of heritability studies indicating that one of the core pathways of genetic risk for SUDs involves a major common externalizing/disinhibitory factor.^{1,2,45} A number of molecular genetic studies also support this relationship, with genetic variants in the serotonergic system having received the largest amount of work to date. Serotonin is thought to operate as a regulator, with increased levels being associated with inhibition of behavior⁴⁶ and genetic variants of tryptophan hydroxylase, the rate-limiting enzyme in the biosynthesis of serotonin, being associated with anger-related traits.⁴⁷ Genetic variants of monoamine oxidase A, specifically involving the *MAOA* promoter, have been associated with impulsive aggression,⁴⁸ antisocial alcoholism,⁴⁹ and impulsive antisocial behavior in the context of childhood maltreatment.¹¹ The serotonin 5-HT_{1B} receptor has been linked to antisocial alcoholism in humans⁵⁰ and to increased impulsive aggression in mice.⁵¹ Other potential candidate genes with apparent relationships to the externalizing/undercontrol domain include *GABRA2*, associated with conduct disorder and SUD in childhood and adulthood and alcohol dependence in adulthood,^{52,53} and *DRD4*, associated with attention-deficit/hyperactivity disorder (ADHD).⁵⁴

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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 development of SUD vary across the different drugs of abuse. For AUD, by far the majority of these have involved genes linked to the metabolism of alcohol; however, given the heterogeneity of the phenotype, it would not be surprising if other pathways of genetic control are also uncovered.

Environmental Influences on Regulational and Attentional Risk Development and Protective Factors

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

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 influence strongly the ontogenic development of the limbic-hypothalamic-pituitary axis and the mesolimbic dopamine reward pathways of the brain. The evidence suggests increasingly that adverse socioenvironmental influences, acting in concert with genetic factors, alter the physiologic 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 rats, that the ontogeny of the stress

response system is regulated in part by maternal factors during early life. Groundbreaking work by Levine⁵⁵ demonstrated that at least 3 aspects of maternal behavior in rats play a role in the regulation of the limbic-hypothalamic-pituitary axis during development, that is, tactile stimulation, feeding behavior, and passive contact. The maternal factors have important analogues in human maternal care and attachment. Also in the rat model, Liu et al⁵⁷ investigated how variations in maternal care affect offspring responses to stress across the lifespan, and they 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 also demonstrated that early environmental stress and maternal rearing behavior predict not only the ontogeny of the stress response circuitry but also the ontogeny of the mesolimbic dopamine reward pathway that underlies drug reinforcement.⁵⁶ Studies in nonhuman primates and humans have confirmed that exposure to early-life stressors alters the response to stress and its underlying circuitry in adults. This observation was confirmed in women who had experienced childhood abuse. A history of childhood abuse was found to predict neuroendocrine stress reactivity, which was 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 al⁵⁸ of children who were abused and/or neglected at ≤ 11 years of age and evaluated 20 years later, childhood neglect but not abuse was related to later alcohol abuse for women but neither neglect nor abuse was 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 performed to resolve these inconsistencies.

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More generally, the attentional regulation and control system seems 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 also play a role 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 that increase the risk for alcoholism by facilitating the onset of drinking, maintenance of drinking behavior, and relapse. Recent work by Nigg et al⁶⁰ showed 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 infants and their 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 grows older, by the mother's broader social environment, including her relationships with the father and with other adults in her support network,^{62,63} and her own previous social experience, including her own

AQ: J history of abuse or other trauma. Eiden et al⁶³ also showed the contribution that fathers make to this process, even early in the life of the child. Alcoholic fathers are lower in sensitivity and higher in negative affect toward their children than are nonalcoholic fathers, and this parenting behavior predicts the reciprocal effect (ie, lower infant responsiveness 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 adolescents¹⁹ and, ultimately, in producing lower drug involvement in adolescence.⁶⁴ The circumstances of “mismatch” between parents and children are of greatest interest here, because they offer the greatest opportunity for the dampening of risky child temperament on one hand and the greatest potential for altering the developmental course in a destructive way on the other. Parents who are responsive to their children’s needs gradually increase the self-regulatory capacities of the children.⁶⁵ Conversely, parents who are aggressive toward their children and who create a conflict-laden family climate diminish the children’s capacity to regulate and to control their own behavior.^{16,19}

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, which provides inhibitory regulation of limbic-hypothalamic-pituitary axis 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 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 in childhood that predict the early onset of drinking and the development of alcohol problems and AUDs in adolescence or adulthood have been identified. 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 formal child psychiatric diagnoses. In addition to these individual factors, 2 socialization domains have been identified consistently as risk factors, one involving neglectful or poor parenting and 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 that do not fall so readily into these domains have also been identified, including early childhood sleep problems, attention problems, and deficits in reading achievement. In the neurophysiologic domain, investigators have also suggested that the P300 waveform of event-related brain potential is a marker of a

risk endophenotype for SUDs.⁶⁶ P300 appears ~300 milliseconds after 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 thought to index the allocation or updating of working memory, as well as a cortical orienting reflex.⁶⁷ Reduction in the amplitude of the P300 potential has been hypothesized to be an endophenotype for SUDs, possibly reflecting central nervous system disinhibition.⁶⁶ Because much of this work has not yet been replicated and because the predictors do not fall so easily into the aforementioned 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 risks for drinking and for progression into drinking problems and AUDs.

The multiplicity of factors identified here and their substantial overlap suggest that (1) a clearer understanding regarding the core individual vulnerabilities and which are secondary needs to be established, (2) the manner in which individual and environmental factors interact needs to be specified more clearly, and (3) a better understanding of sequencing is required. The following sections provide a detailed account of the pertinent studies.

Antecedent Predictors of Onset of Drinking in Childhood (Initiation Before 13 Years of Age)

Previous longitudinal research on children tended to focus on adolescent, young adult, or adult, rather than child, 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 solely on alcohol use, because of the generally low rates of use by children.^{68–70} Significant antecedent predictors of children’s substance use initiation in those studies included lower prosocial family processes (monitoring, rules, and parent-child attachment), deviant peer affiliation, peer drug use, parental tolerance of substance use, parental drug abuse, child overactivity, 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 children tested at 4 to 5, 9 to 10, and 14 years of age, Baumrind⁷¹ reported that earlier ages of onset of alcohol use were associated with less social assertiveness for both genders. 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 generally involved peer

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instigations. Bush and Iannotti,⁷² in their study of a largely black sample of fourth-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 was defined as initiation by 14 or 15 years of age, rather than onset in childhood, a number of studies found early predictors. These included studies predicting early onset of drinking (compared with later onset), as well as those using survival analyses to predict the age of first use. Studies involved both high-risk and population samples. In the high-risk Seattle Social Development Study,⁷³ for example, earlier age of alcohol initiation was predicted by the following predictors at 10 to 11 years of age: white ethnicity, greater parental drinking, less bonding to school, and having more friends who drink. In a high-risk study of boys from Pittsburgh,⁷⁴ age of onset of alcohol use (use of ≥ 1 standard drink per episode) through 15 years of age was predicted by antisocial disorder (conduct disorder or oppositional-defiant disorder) but not ADHD or negative-affect disorder (anxiety or mood disorder). An earlier analysis of that sample, using a lower threshold of any alcohol use, found that the number of conduct disorder symptoms from mothers' reports for children 10 to 12 years of age and children's executive cognitive functioning were not related to alcohol use at 12 to 14 years of age.⁷⁵ In a community-based, high-risk sample of families,⁷⁶ parental alcoholism and mothers' ratings of children's sleep problems, trouble sleeping, and being overtired at 3 to 5 years of age predicted onset of alcohol use by 12 to 14 years of age. Parental alcoholism also predicted onset of drunkenness by 12 to 14 years of age. The authors interpreted the sleep problems measure as an indicator of instability of biological rhythms, as well as of social dysregulation. Finally, Dobkin et al⁷⁷ found, in a lower-socioeconomic status sample of boys from Montreal, Canada, that ratings of fighting and hyperactivity at 6 years of age and ratings of their aggressiveness and friends' aggressiveness at 10 years of age predicted drunkenness at 13 years of age. Age of onset of drunkenness (by 15 years of age) was predicted for these boys by teachers' ratings of higher novelty-seeking and lower harm avoidance at 6 and 10 years of age.¹⁴

For population samples, studies suggested that factors very similar to those found in high-risk samples also predicted early initiation of use. Among 10- to 12-year-old abstainers selected from the Minnesota Twin Family Study, antecedent predictors of alcohol use initiation at 14 years of age were conduct disorder, oppositional-defiant disorder, and any externalizing disorder but not major depressive disorder or ADHD.⁷⁸ In another study with the same sample, King et al⁷⁹ found that the same externalizing factors predicted regular use, ever being drunk, and heavy drinking at 14 years of age. Several other studies provided significant overlap with the Minnesota findings but also extended the network of predictors. In the Ontario Child Health Study, children rated by teachers as having conduct disorder at 8 to 12 years of age were more likely to be regular drinkers 4 years

later.⁸⁰ In a study of a birth cohort of children in New Zealand, Lynskey and Fergusson⁸¹ found that conduct problems at 8 years of age predicted usual intake of alcohol, maximal intake of alcohol, and alcohol-related problems experienced before 15 years of age (even controlling for gender, family socioeconomic status, 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 15 years of age (similar to results found by McGue et al⁷⁸).

Early-onset alcohol use (by 14 years of age) was predicted in the Finnish Twin Study by a number of social contextual factors assessed at 11 to 12 years of age, including lower parental monitoring and worse home environment. Individual difference 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 predominated as influences on drinking initiation in early adolescence. Finally, the Great Smoky Mountain Epidemiologic Study of Youth tested children at 9, 11, and 13 years of age; antecedent predictors of having initiated alcohol use 4 years after baseline assessment 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 may make alcohol use more pleasurable or reinforcing has not been evaluated. Although adrenarche typically occurs before 10 years of age and menarche typically occurs after 10 years of age (but before 10 years of age among precociously maturing girls), 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 article in this issue.⁸⁷

Childhood Predictors of Drinking in Middle Adolescence

Several studies have linked childhood functioning to later adolescent alcohol consumption levels. For example, in the Woodlawn Study,⁸⁸ teacher ratings of aggressiveness in first grade predicted more-frequent use of alcohol at 16 to 17 years of age for black boys (but not girls). There was also a trend for shyness to be related to less alcohol use for boys but not girls. In a follow-up study of children diagnosed as having ADHD and control subjects, childhood symptoms of inattention measured at 5 to 12 years of age were predictive of frequency of drunkenness and alcohol problems in adolescence.⁸⁹ In contrast to the findings of Kellam et al⁸⁸ for a large general population sample, Hill et al⁹⁰ studied families at high risk for alcoholism because of their dense family history of alcoholism. They found that age of onset of regular drinking with negative consequences was pre-

dicted by greater extraversion, deficits in reading achievement, reduced P300 (visual and auditory), and greater postural sway.

Childhood Predictors of Adolescent Problem Drinking

To date, only 2 groups have examined early childhood predictors of problem drinking assessed within adolescence. Both studies involved high-risk samples. In the Seattle Social Development project,⁷³ the strongest predictors of problem drinking at 16 years of age were younger age of initiation of drinking and being male. The effects of other predictors at 10 to 11 years of age (parents' drinking, friends' drinking, school bonding, and perceived harm of drinking) were mediated by age of initiation. In another report on the Michigan Longitudinal Study high-risk sample, Wong et al⁹¹ observed that, although the normal pattern of increases in behavioral control over the course of childhood was present in the sample, a slower rate of increase in behavioral control from preschool age 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 evaluated children as young adults and assessed their experience of alcohol problems. Pulkkinen and Pitkanen,⁹² for example, found in a sample of Finnish children that aggressiveness at 8 years of age was predictive of problem drinking at 26 years of age for boys but not for girls, whereas social anxiety at 8 years of age was predictive for girls but not for boys. Similarly, in a community sample in New York, childhood aggression at 5 to 10 years of age, assessed as anger, sibling aggression, noncompliance, temper, and nonconforming behavior, was related to *Diagnostic and Statistical Manual of Mental Disorders, Revised Third Edition*, alcohol abuse at 16 to 21 years of age.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 undercontrolled (impulsive, restless, or distractible) at 3 years of age were more than twice as likely as control children to exhibit a diagnosis of alcohol dependence at 21 years of age. The 1 study that did not replicate the undercontrol findings was also a birth cohort study from New Zealand, the Christchurch Health and Development Study.⁹⁴ There, conduct problems at 7 to 9 years of age did not relate to *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*, alcohol dependence at 21 to 25 years of age. Although it is impossible to know what the sample differences might be that led to these divergent findings, a review by Zucker³ of 6 other longitudinal studies, some population-based and some high-risk, indicated that the relationship of undercontrol to adult alcohol problem use is extraordinarily robust (all 6 studies replicated the finding), which in turn suggests that the findings of Fergusson et al⁹⁴ are anomalous.

It 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 seem to predict the early drinking outcome. A study of boys ($n = 122$) recruited in prenatal clinics in a small community outside Stockholm, Sweden,⁹⁵ found that lower ability to concentrate at 10 years of age and lower levels of school achievement at 10 years of age were related to hazardous use of alcohol before 21 years of age and at 36 years of age. (Hazardous use was defined on the basis of police register data on public drunkenness and drunk driving and high levels of reported alcohol intake.) Similarly, as noted earlier, Nigg et al⁶⁰ found that poor response inhibition also predicted early initiation of drunkenness and problem use, even controlling for conduct problems (as an index of behavioral undercontrol).

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

The work of Guo et al⁹⁷ extends the conceptual framework of predictors in a more-integrated fashion. Those authors used a social development model that included individual difference, family, and neighborhood factors to predict AUD outcomes 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, greater intentions to use alcohol, and more-favorable attitudes toward alcohol at 10 years of age. They found that a model integrating all of these factors was predictive of *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*, alcohol abuse and dependence at 21 years of age.

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 12 years of age were related modestly to alcohol involvement at 40 to 50 years of age.⁹⁸ Among Hawaiian elementary schoolchildren evaluated at an average age of 45 years, higher teacher ratings of extraversion and lower ratings of emotional stability were associated with greater adult alcohol intake.⁹⁹ Cloninger et al¹² reported that Swedish children who were rated higher in novelty-seeking and lower in harm avoidance and reward dependence at 11 years of age were more likely to be involved in alcohol abuse (defined as registration with the Swedish Temperance Board, arrests for drunkenness or driving while intoxicated, or treatment for alcoholism) at 27 years of age. 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 7 months, and inability to walk at 1 year) were related to the diagnosis of alcohol dependence at 30 years of age.

In the age 42 follow-up evaluation of black children who were 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 prenatal study referred to above, Wennberg and Bohman⁹⁵ found that psychologist ratings at 4 years of age predicted outcomes not just at the end of adolescence but also well into adulthood. Extrovert/aggressive ratings at age 4 were correlated with frequency of intoxication at age 25 ($r = 0.27$; $P < .05$), whereas extrovert/outgoing ratings were correlated with lifetime alcohol problems to age 36 ($r = 0.22$; $P < .05$).

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ALCOHOL-SPECIFIC RISK FACTORS IN CHILDHOOD

Focus

In contrast to the previous section, the focus here is on 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 2 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 US society. According to data from the National Institute on Alcohol Abuse and Alcoholism,¹⁰² a large majority of US 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% vs 55.1%) and are substantially more likely to be moderate drinkers (21.6% vs 7.3%) or heavier drinkers (5.6% vs 4.0%). College graduates are more likely to be current drinkers than are adults with less education. Non-Hispanic white and Hispanic individuals are more likely to be current drinkers than are non-Hispanic black and other non-Hispanic individuals. Adults living in the South are more likely to be abstainers than are adults from other regions of the country.

Data from the 2001–2002 National Epidemiologic Survey on Alcohol and Related Conditions showed substantial variation in the rates of current alcohol abuse and alcohol dependence across subsets of the US population.¹⁰³ Overall, 8.5% of US adults exhibited either alcohol abuse or dependence. Alcohol dependence in the past 12 months was seen for 5.4% of men and 2.3% of women. White, Native American, and Hispanic individuals had significantly higher rates of alcohol dependence (3.8%, 6.4%, and 4.0%, respectively) than did Asian American individuals (2.4%). Although religious background is not covered in federally sponsored sur-

veys, alcohol dependence has historically been higher among Catholics and liberal Protestants than among fundamentalist Protestants and Jews. As a result of these variations in adult alcohol use and abuse, children's exposure to alcohol use in the home 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 age at the first drink are not very reliable for this life stage. Age of onset generally increases as older adolescents are questioned.^{104,105} For example, in national data from the 2005 Youth Risk Behavior Survey, the proportions of respondents who stated that they drank alcohol before age 13 decreased from 33.9% for 9th-grade students to 19.3% for 12th-grade students.¹⁰⁶ These are not cohort effects but rather are evidence of "forward telescoping," as shown by the fact that similar proportions of students in these grades reported drinking before age 13 in each of the 5 previous surveys (1995, 1997, 1999, 2001, and 2003) that asked this question. Similar findings were also obtained in the national Monitoring the Future surveys, comparing eighth-graders and 12th-graders across multiple annual surveys regarding their reported incidence rates of alcohol use by sixth grade.¹⁰⁷ Therefore, determination of average or modal age of initiation of alcohol use on the basis of retrospective recall is problematic. Results vary 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 ≤ 10 years of age are extremely rare, however. According to the most-recent Partnership Attitude Tracking Study (sponsored by the Partnership for a Drug-Free America), which surveyed a national probability sample of nearly 2400 US elementary school students in 1999, 9.8% of fourth-graders, 16.1% of fifth-graders, and 29.4% of sixth-graders had had more than just a sip of alcohol in their lives.¹⁰⁸ Data on the use of alcohol in the past year (rather than lifetime) has been reported annually by PRIDE Surveys.¹⁰⁹ According to the 2003–2004 summary of school district surveys performed across the United States, 4.2% of fourth-graders, 5.6% of fifth-graders, and 8.7% of sixth-graders had had a beer in the past year.¹¹⁰ Slightly more had had wine coolers (4.4%, 6.7%, and 10.3%, respectively), and approximately one half as many reported drinking liquor in the past year (1.9%, 2.8%, and 5.2%, respectively).

These data, although 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 therefore contain an unknown level of bias. Clearly, national surveillance efforts need to be directed toward monitoring the alcohol involvement of children starting in grade 4. Initiating surveillance in 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 US and European children's experience with alcohol is possible to a very limited extent. Information on the number of 11-year-old US 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 that was conducted in 1997–1998 with 120 000 students in 28 countries, including countries in all parts of Europe, Canada, and the United States.¹¹¹ Rates of having at least tasted alcohol varied widely across countries. The proportions of 11-year-old children who had at least tasted alcohol (averaged across genders) ranged from 91% 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 ~35% in Norway. Ever experience with alcohol was reported by 62% of 11-year-old boys and 58% of 11-year-old girls in the United States. These US prevalence rates ranked 16th among the 28 countries studied. In most countries, more male than female 11-year-old children had at least tasted alcohol.

Alcohol Problems in Children

Although there are anecdotal reports and clinical reports of alcoholic children,^{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 AUDs at ≤ 12 years of age is close to 0 cases in the general population.¹¹⁴ Subclinical levels of alcohol problems in childhood are somewhat more prevalent. For example, Chen et al¹¹⁵ found that 4.8% of fifth-graders in Baltimore, Maryland, had already experienced ≥ 1 alcohol problem.

Early Alcohol Use Onset as Risk Factor for Later Problems

It is critical to develop better information regarding the extent of alcohol experience among US children, because younger ages of onset of alcohol use are associated with a greater likelihood of developing both problem drinking in adolescence^{73,116–118} and alcohol abuse or dependence in adulthood.^{119–121} Although studies do not agree on whether alcohol use in childhood (≤ 12 years of age) or in early adolescence (13–14 years of age) 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 in childhood.

In addition to the increased risk for later alcohol problems, early-onset drinking has been shown to be related to a variety of other problematic outcomes. Onset of drinking by 10 to 12 years of age is associated with absences from school, drinking and driving, and marijuana and other illicit drug use in grade 12.¹¹⁸ Onset by grade 7 (12–13 years of age) was found to be related to more school problems, more delinquent behavior, more smoking, and more illicit drug use in grade 12, compared with 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 began drinking by the autumn of grade 7 reported more recent drinking, drunkenness, and alcohol or drug problems and were more likely to have initiated sexual intercourse, to have had >2 partners, and to have gotten pregnant (or gotten someone pregnant).¹²² Methodologic problems in this area involve the use of retrospective reports, variability in the definition of "early onset" across studies, apparent use of age of onset as a substitute for examination of a larger array of alcohol "landmark" behaviors that may be of relevance for later alcohol problems (eg, regular use and first drunkenness), and the absence of questions regarding context of first use (eg, 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-aged 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 6 years through 10 years of age.^{126,127}

By 6.5 to 7.5 years of age, 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 clusters of other bottles. Younger children (4.5–6.5 years of age), although they could label individual bottles correctly, could not explain how the bottles were grouped. Fossey¹²⁷ replicated the original bottle-grouping task used by Jahoda and Cramond¹²⁶ and found that older children did better than younger children in 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," which were measured at 3 to 5 years of age with a task in which children were presented with drawings of child and adult figures in

common social situations (eg, 2 adults on a sofa in front of a fireplace, a family eating dinner, and a man watching television) and were asked what kind of beverages the figures were drinking (alcoholic versus nonalcoholic).¹²⁸ Alcoholic beverages were attributed to adults more often than to children pictured and to men more often than to women. In other words, even in preschool, children know the norms about drinking in the adult culture, namely, adults drink alcoholic beverages and children do not and men drink more than women. As might be anticipated, these drinking attributions were more evident for children of alcoholics (COAs) than for children of nonalcoholic parents.¹²⁸

A more-recent study demonstrated similar alcohol schemas by using a shopping paradigm.¹²⁹ Children 2 to 6 years of age 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 schemas also vary as a function of age. Studies of normal samples over a 20-year period showed that children's ratings of adults depicted drinking alcohol are basically neutral at age 6 and become more negative up through 10 years of age.^{126,127} Additional research with this same paradigm showed that these attitudes become more positive between 10 and 14 years of age.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 10 years of age.^{127,132-134} Girls, particularly in the younger grades (kindergarten and third grade), were found to provide more coordinated, psychological, and causal responses than boys 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 become 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, whereas later expectancies incorporate a pharmacologic dimension (sedation versus arousal), exemplified by wild, dangerous, talkative, and cool.¹³² In the age range from 8 years to 12 years, 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, although relatively few children have initiated drinking at these ages, there is the development of attitudes, beliefs, and expectancies that place them at increasing risk for movement into alcohol use.

Childhood Social Contexts That Facilitate Drinking

Importance

In childhood, children are exposed to alcohol use through a number of social mechanisms, including 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, and 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 the children's perceptions of their parents' 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 and 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 the Bogalusa Heart Study in 1993-1994 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 from someone else's drink, 6% took it from home, and 6% got the alcohol from another child (see also refs 131 and 135). In a community survey of children in Oregon,¹³⁸ few of the children, especially in the younger grades (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 variations 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 movie and television content and advertisements. The alcohol industry spends more than \$1.6 billion per year on advertising in radio, television, magazines, newspapers, and billboards.¹³⁹ The alcohol industry routinely exposes adolescents 12 to 20 years of age to high levels of alcohol advertising, through the placement of advertisements 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 likely to listen, and during television programs in which they are likely to be interested. For example, in 2003, teens saw twice as many advertisements for beer,

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>3 times as many advertisements for alcopops (sweet-flavored alcoholic drinks), and 50% more advertisements for spirits in magazines, on a per-capita basis, than did adults ≥ 35 years of age.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 <12 years of age; therefore, their exposure to alcohol advertisements in these media cannot be measured. Second, children <12 years of age may be less exposed to magazine advertisements because of their reading levels and reading choices (books rather than magazines, or magazines with advertising restrictions). Third, a Center on Alcohol Marketing and Youth report from 2005 suggested that children 2 to 11 years of age are underexposed to alcohol advertisements on television, relative to their prevalence in the overall population.¹⁴⁰ They are exposed to less than one half as many television alcohol advertisements as are 12- to 20-year-old youths. This does not mean that they are not exposed, however. On average, children 2 to 11 years of age saw 99.4 alcohol advertisements on television between January 2004 and October 2004 (81% for beer and ale, 11% for spirits, 5% for alcopops, and 3% for wine, calculated from data in that article). At this rate, the average child could have seen almost 1200 alcohol advertisements on television before age 12 (assuming similar rates 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 (8–11 PM), when children may be watching. Estimates from the 1998–1999 season indicated that 71% of sampled episodes included alcohol use by characters on the shows.¹⁴¹ Most disturbing was the finding that 38% of shows with a TV-G rating (appropriate for most children) depicted alcohol use. More episodes characterized drinking as a positive experience than as a negative experience. Negative consequences were portrayed or mentioned in only 23% of episodes.

Children's animated films have also been analyzed for alcohol content. All G-rated, animated films that were released by 5 major studios between 1937 and 1997 and were available on videotape were reviewed for episodes of tobacco and alcohol use.¹⁴² Of the 50 films reviewed, 50% included alcohol use, which was portrayed by 63 characters for a total of 27 minutes. Seven of the 50 films depicted effects of alcohol use (eg, drunkenness, passing out, losing balance, or 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 identify correctly the brand names for still photographs from current television commercials) was related significantly to greater intentions to drink as an adult through its relationship to more-positive beliefs about alcohol (a mediated path). A recent study of 10- to 14-year-old non-drinkers found that the level of exposure to alcohol use in motion pictures predicted whether the subjects were drinkers 1 to 2 years later.¹⁴⁴ Considerably more research

is necessary, however, to determine the linkage of media exposure to drinking and 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

Prevalence

According to National Longitudinal Alcohol Epidemiologic Survey data,¹⁴⁵ ~9.7 million children ≤ 17 years of age, or 15% of the children in that age range, were living in households with ≥ 1 adult classified as having a current (past-year) diagnosis of alcohol abuse or dependence. Approximately 70% of those children were biological children, foster children, adopted children, or stepchildren. That is, 6.8 million children meet the formal definition of COAs, although not all are exposed to the same level of risk for use, problem use, and AUD. As far as socialization risk is concerned, these figures reflect only acute (past-year) exposure to ≥ 1 alcoholic adult. According to other data from the National Longitudinal Alcohol Epidemiologic Survey,¹⁴⁵ 43% of the <18-year-old population, or slightly less than one half of all children, were exposed to a currently or previously alcoholic adult in the household. The figure for just COAs was 30% of the <18-year-old 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 differentiate considerably the risk variability among these families. It is essential that this be done, given the magnitude of the problem. COAs are 4 to 10 times more likely to become alcoholics themselves.¹⁴⁶ They are also at elevated risk for earlier drinking onset¹⁴⁷ and earlier progression to 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 conducted. Studies have involved children with an alcoholic biological parent who were raised by nonalcoholic adoptive parents,¹⁴⁸ thus enabling a test of the separate influences 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.

Although 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 responses to alcohol that make drinking more pleasurable and/or less aversive, and some to elevated

transmission of risky temperamental and behavioral traits that lead COAs into greater contact with earlier- and heavier-drinking peers.

Factors Involved in Familial Transmission

Familial alcoholism status (being “family history positive”) is heavily used as a proxy for “alcoholism risk” on one hand and “socialization risk” on the other hand, 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. A positive family history implies elevated genetic risk, on average, although the alcoholic genetic diatheses might not have been passed on to a particular child. One may be a COA without being undercontrolled or having an ADHD diagnosis.

Socialization risk involves familial exposure but, given the high divorce rates in this population, evaluating the level of socialization risk is complex, because it involves not only quantification of the duration of exposure to the actively alcoholic parent but also determination of 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 risks, individual parent risks, and the synergistic risks created by impaired marital interactions.¹⁶

The potential for indirect socialization effects is also higher in alcoholic families. Parental psychopathological conditions have been documented as a risk factor for poorer parental monitoring,¹⁵⁰ which leads to a higher probability of involvement with a deviant peer group, including earlier exposure to alcohol- and other drug-using peers.

COA risk is not simply risk for the development of an AUD.¹⁵¹ Given what is known about the elevated psychiatric comorbidities among COAs, being a COA is also a marker of elevated risk for a variety of behavioral and cognitive deficits, including ADHD, 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} In a community study of high-risk families, Wong et al⁷⁶ 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 regulatory deficit, represented an independent predictor of the former outcome. Similarly, in a longitudinal study monitoring boys with and without parents with a SUD, Tarter et al⁸ found that the effect of the father’s and mother’s SUD on a son’s diagnosis of SUD at age 19 was mediated by neurobehavioral disinhibition (operationalized as the sum of disruptive behavior disorder symptoms on the Structured Clinical Interview for Axis II Disorders), social maladjustment, and drug use frequency at age 16. Other studies investigating

the mediators of these effects included that by Hill et al,⁹⁰ which showed not only that children in high-risk families had an earlier age of initiation of regular drinking with negative consequences than did children in low-risk families but also that this relationship 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 risks 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 considered as proxies for other mechanisms. As specific genes that carry alcoholism risk are identified, investigators will be better able to model the interactions between social environment and genetic vulnerability that very well may be taking place.¹¹ Such studies are essential.

Fetal Alcohol Exposure

An additional potential risk for early-onset drinking and 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 and socialization risks but also by risks arising from the teratogenic effects of alcohol exposure during fetal development. These teratogenic risks can occur even at levels of alcohol intake during pregnancy that are not symptomatic of maternal alcoholism. Although 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 morphologic, growth-related, neurologic, and behavioral and reflect a spectrum of alcohol-related neurodevelopmental disorders (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 1974–1975, as part of the Seattle Longitudinal Study on Alcohol and Pregnancy, 1529 pregnant women were interviewed in their fifth month regarding their demographic characteristics, nutrition, use of tobacco, alcohol, and caffeine, and use of medications. In 1989–1990, 464 families, reflecting a spectrum of maternal drinking during pregnancy, were evaluated when the children were 14 of age.¹⁶⁰ The mother’s alcohol intake during pregnancy, and hence the child’s prenatal exposure to alcohol, significantly predicted adolescent experiences of the negative consequences of drinking (ie, personal and social difficulties resulting from alcohol use, such as getting into a fight, neglecting responsibili-

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ties, or having a bad time), even controlling for family history of alcoholism, current parental drinking, and several parenting variables. Family history of alcoholism was not a significant predictor when prenatal alcohol exposure was controlled for statistically. A later follow-up study of this sample found that prenatal exposure to alcohol and family history of alcoholism predicted young adult (age: 21–24 years) scores on the Alcohol Dependence Scale.¹⁶¹

There are a number of ongoing longitudinal studies of cohorts of children exposed prenatally to alcohol that should soon have data on the adolescent alcohol involvement of the children (eg, the Maternal Health Practices and Child Development Project) and should 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, from both the child and adult literature, that risks are correlated at the individual and familial levels and at the neighborhood level. At the individual level, the literature has increasingly acknowledged the clustering of comorbid symptoms, social dysfunction, and alcoholism severity among adults.¹⁶² In fact, such assortment has been one of the driving forces for the notion that subtypes of disorders 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 microenvironmental 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 (ie, a top-down explanation). What has been less clear is the degree to which individual processes are also at work; some evidence suggests that there are, at least for children from antisocial alcoholic families. 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, seem 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 suggests 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 familial, neighborhood, and peer structures that 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 on 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, which suggests 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 dropout, and depression. In some cases, high-risk pathways are so well established that these pathways are clear targets for preventive interventions,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 seem 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 the 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.

Externalizing Pathway

There is mounting evidence 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, such as 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 proportion of these children disengage from school, begin to associate with deviant peers, engage in increasingly risky behaviors, and escalate in delinquent behavior. At some time during the transition to adolescence, these youths are at high risk for early alcohol use, as well as other behaviors in the problem behavior spectrum such as substance abuse, early and risky sexual activities, and truancy.

Internalizing Pathway

A second pathway implicated by the longitudinal data on risk for underage drinking that may have its begin-

nings in childhood involves depressive spectrum disorder symptoms and related antecedents, including anxiety and shy/inhibited personality.^{10,88} Evidence is weaker for this internalizing pathway in relation to earlier alcohol use, although there seems 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 AUD.¹⁵⁰

Low-Risk Pathways

Implicitly, patterns of risk also implicate patterns of lower risk for underage drinking, although these pathways have not been as well defined. Children who have a record of success in age-salient developmental tasks throughout childhood, with the benefits afforded by good self-regulation skills and effective parents, and 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 documented this pathway for a group of children who start out with low levels of the risky externalizing and internalizing traits and are born into environments with less family adversity. As shown in Fig 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 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 throughout 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 levels remained low during the preschool and early school years and then began to increase, approaching the levels found among more-vulnerable children by early adolescence. The authors suggested that the exposure to more family adversity over long periods eventually “wore away” the sunnier 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, that is, promotive factors, which are generally associated with better outcomes across levels of risk or adversity (main effects, in statistical terms), and protective factors, which 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 as both promotive and protective factors. Considerable literature evidence implicates good-quality parenting as a promotive factor with respect to many positive developmental outcomes; at the

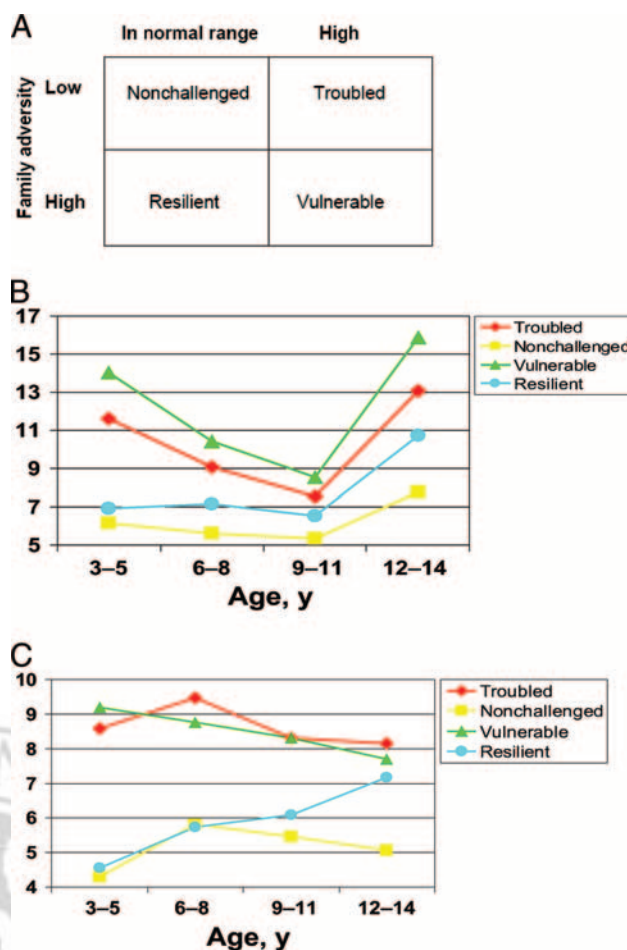


FIGURE 1 Typologic features of family/risk and adversity groups and changes in externalizing and internalizing symptoms in the different risk/adversity groups over time. A, Child psychopathological conditions in preschool. Shown is a 2 × 2 matrix of children’s individual differences in psychopathological conditions and family adversity during the preschool years. B, Externalizing symptom scores for different risk/adversity groups. C, Internalizing symptom scores for different risk/adversity groups.

same time, parenting quality seems 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 factor from a promotive factor or a vulnerability factor 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 in which 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 whether 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 predicted 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 seems 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 <10 years of age. The data exist, but the field has not yet addressed this issue aggressively.

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 nontrivial 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, to monitor both the need for and the success of prevention efforts in elementary schools. Second, alcohol use onset is one of the initial stages in the progression to 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, onset of alcohol use in childhood predicts alcohol problems in adolescence, as well as alcohol abuse and dependence in adulthood.^{73,119}

Although it is clear that early-onset drinking is problematic, it is also clear that some parents think that 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 occurs in a peer context.¹⁷² More research on whether and how onset context (eg, family versus peer context) matters is very important. Moreover, this issue highlights how little is known about US adults' beliefs about anticipatory socialization regarding alcohol use in childhood.

In addition, although there once was a literature on cultural contexts of drinking and their influencing roles (eg, 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 subcultural support in the home for such drinking, it is unlikely that school-based prevention pro-

grams 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 INTERVENTIONS

There are a number of points before 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 that it is essential to consider these developmental pathways of risk. Therefore, prevention efforts can target parents before conception, prenatally or at many points in child development, long before initiation of alcohol use by children. On the basis of this review, we recommend 5 target areas for intervention, as follows.

First, treat alcohol problems in potential parents. Given the importance of genetic risks for alcoholism and socialization risks associated with alcohol problems in parents, adults with alcohol problems who are likely to become parents are an important target for intervention. Examples include (1) an emphasis on treatment for alcoholic parents, to reduce the parents' problem drinking and thereby to reduce children's exposure to such drinking in the home; (2) provision of parental training to instill more-effective parenting practices and to reduce instances of child neglect and maltreatment; and (3) provision of marriage/couples counseling, to ensure that there is less conflict in the home. Such parental training and counseling should be offered as part of the parents' alcoholism treatment. The goal is to make the intergenerational transmission of alcoholism less likely.

Second, boost efforts to reduce prenatal drinking in mothers. Prenatal exposure to alcohol is a risk factor for developmental anomalies such as fetal alcohol syndrome and seems to be a risk factor for problem drinking in adolescence and young adulthood,¹⁶¹ although additional 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 regarding negative consequences of drinking during pregnancy, greater emphasis and dissemination regarding the need for prenatal care during pregnancy, better screening for women's alcohol use as part of prenatal visits, and greater referral of drinking pregnant women to effective alcohol interventions.

Third, include screening for alcohol use and alcohol risk behaviors in pediatric well-child visits. The review cited above¹⁶¹ also implicated 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, decreased response inhibition, and delinquent behavior (although genetic risks could also figure here). Pediatric well-child care should include screening for prenatal alcohol/drug exposure if prenatal care records are not forwarded from the mother's obstetrician. Among children identified as having prenatal exposure to alcohol, early childhood interventions should be instituted before school entry, targeted toward instill-

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ing 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 pharmacologic agents in reducing problems associated with this disorder.

Fourth, 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 (1) 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 (2) to develop programs to enhance or to 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 venues are the most engaging and least stigmatizing for such interventions, and how barriers to parental involvement in the programs can best be reduced.

Fifth, intervene early in pathways to deviant peers and promote pathways to prosocial peers. Although onset of alcohol use in childhood 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, less parental support, less parental monitoring, and less 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.

CONCLUSIONS

This review has documented a host of factors and pathways evident before 10 years of age 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 ≥ 1 decade. Despite the preponderance of evidence, it is still rare for researchers or clinicians to recognize that drinking problems of youths 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 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 largely 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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