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The developmental psychopathology of alcohol use and alcohol disorders: Research achievements and future directions

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Abstract

The last 25 years have seen significant advances in our conceptualization of alcohol use and alcohol use disorders within a developmental framework, along with advances in our empirical understanding that have been potentiated by advances in quantitative methods. These include advances in understanding the heterogeneity of trajectories of alcohol outcomes; new insights about early childhood antecedents, and adolescence and emerging adulthood as important developmental periods for alcohol outcomes; a more nuanced understanding of the influences of developmental transitions, and their timing and contexts; a greater appreciation for the importance of considering multiple levels of analysis (including an increasing number of genetically informative studies); a continuing focus on studying multiple pathways underlying alcohol outcomes; and an increasing focus on studying the effects of alcohol exposure on future development. The current paper reviews these advances and suggests directions for future study.

It seems particularly fitting that this 25th Anniversary Issue of *Development and Psychopathology* should include reflections on the past 25 years of research achievements in the study of alcohol use and alcohol use disorders (AUDs) because a fundamental achievement during this time period was the reconceptualization of alcohol use and AUDs within a developmental framework, including the explicit labeling of AUDs as "developmental disorders of young adulthood" (Sher & Gotham, 1999). Early roots of this reconceptualization can be seen in Cloninger, Bohman, and Sigvardsson's (1981) distinction between alcoholism subtypes based on their differing ages of onset and Zucker's (1986) description of the "four alcoholisms" based on differences both in age of onset and developmental course as well as earlier typologies (e.g., Knight, 1937). Moreover, in addition to identifying age-related patterns of alcohol use and AUDs, researchers during this period discovered the value of bringing a developmental psychopathology approach to the study of etiological factors. In an editorial accompanying a 1999 Special Issue of *Development and Psychopathology*, Cicchetti and Luthar argued that a developmental psychopathology approach was able to integrate theories and findings that had previously emerged from different disciplines working in isolation. They noted the importance of studying transitions from substance use to substance abuse, understanding why some people avoided this transition and why some substance use problems were developmentally limited, whereas others persisted into adulthood. They also argued for the importance of studying multiple etiological pathways underlying the development of substance use disorders. These themes, as applied specifically to alcohol use and AUDs, were reiterated and elaborated in the recent (2008) special issue of *Pediatrics*, which was devoted to studies of underage drinking within a developmental framework (Masten et al., 2008). These themes were also illustrated in the strategic plan (2007–2011) of the National Institute on Alcohol Abuse and Alcoholism, which described existing and future alcohol research opportunities using a life span developmental framework as the organizing principle (National Institute on Alcohol Abuse and Alcoholism, 2006). Finally, the growing importance of a developmental perspective over the last 25 years is reflected in research attention to the developmental appropriateness of diagnostic criteria as applied to AUDs in adolescence (Chung & Martin, 2005; Winters, Martin, & Chung, 2011).

In this paper, we attempt to summarize some of the central research achievements in the past 25 years, many of which have resulted from applying a developmental psychopathology perspective to the study of alcohol use and AUDs. These include advances in understanding the heterogeneity of trajectories of alcohol outcomes, new insights into early antecedents and into adolescence and emerging adulthood as important developmental periods for alcohol outcomes, the influences of developmental tasks and the timing of developmental transitions, the importance of considering multiple levels of analysis (including an increasing number of genetically informative studies), the importance of studying multiple pathways underlying alcohol outcomes, and the importance of considering the effects of alcohol exposure on

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future development. We do not attempt a comprehensive review, but rather we illustrate some of the major accomplishments and point to directions for future research. Moreover, we restrict our focus to issues of developmental course and etiology, and do not attempt to cover advances in intervention research (see Spoth, Greenberg, & Turrisi, 2008, for a review of alcohol prevention research and Deas, 2008, for a review of adolescent alcohol treatment research).

It is interesting that alcohol research during the last 25 years has been conducted against the backdrop of general declines in drinking among adolescents (Johnston, O'Malley, Bachman & Schulenberg, 2012) and adults (in the largely white Framingham Atudy sample; Zhang et al., 2008). It is more difficult to assess parallel changes in AUDs because of changes in diagnostic systems over time. Over the past 30 years, there have been several large-scale, population-based epidemiological surveys using structured diagnostic interviews in the United States that have provided estimates of AUDs. These include the Epidemiologic Catchment Area study (Helzer, Burnam, & McEnvoy, 1991; Robins & Price, 1991); the National Comorbidity Survey (NCS; Kessler, Crum, Warner, & Nelson, 1997; Kessler, McGonagle, Zhao, Nelson, Hughes, Eshleman, et al., 1994); the National Comorbidity Survey—Replication (NCS-R; Kessler, Berglund, Demler, Jin, & Walters, 2005; Kessler, Chiu, Demler, & Walters, 2005), the National Longitudinal Alcohol Epidemiologic Survey (NLAES; Grant, 1997; Grant et al., 1994; Grant & Pickering, 1996), and the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC; Grant et al., 2004). All of these studies report very high past year and lifetime prevalence rates of AUDs (13.8% lifetime and 6.8% past year DSM-III in Epidemiologic Catchment Area; 23.5% lifetime and 7.7% past year DSM-III-R in NCS; 18.2% lifetime and 7.41% past year DSM-IV in NLAES; 30.3% lifetime and 8.46% past year DSM-IV in NESARC; and 18.6% lifetime and 4.4% past year DSM-IV in NCS-R). Although it is hard to compare across different diagnostic systems and different measurement approaches, NLAES and NESARC were highly similar in design (albeit with some very subtle changes in instrumentation) and separated by a 10-year period. This allows some ability to compare rates from the early 1990s to the early 2000s. Although the overall rates of past-year AUDs were roughly similar, Grant et al. (2004) note that this overall trend reflects a decrease in dependence that was more than offset by an increase in abuse (despite the general declines in consumption over this period). However, it is difficult to determine if these changes reflect true changes in prevalence or are an artifact of very subtle variations in instrumentation (Vergés, Littlefield, & Sher, 2011).

Advances in Quantitative Methods

As we will describe in this paper, significant advances have been made in the past 25 years in the conceptualization of alcohol use and AUDs within a developmental framework, including complex and dynamic hypotheses about individual variability in continuous developmental trajectories; the joint influence of time and timing; the contribution of multiple environmental and biological contexts; the impact of transition periods and role acquisition; and the articulation of dynamic and bidirectional pathways of onset, escalation, maintenance, and desistence of alcohol use. However, these increasingly complex questions can only be tested using designs and statistical models that directly correspond to the research hypotheses under study (e.g., Curran & Willoughby, 2003; Wohlwill, 1991). Here we briefly review the developments in quantitative methods that have occurred over the past 25 years that have allowed for corresponding advances in our understanding of the developmental psychopathology of alcohol use and disorders.

As with many areas of scientific inquiry, early empirical studies of child and adolescent alcohol use and abuse were primarily based on cross-sectional designs. However, the developmental psychopathology perspective requires an understanding of development over time, better revealed by longitudinal data. Twenty-five years ago, perhaps the most common statistical modeling framework applied to longitudinal data was the autoregressive cross-lagged (ARCL) panel model (e.g., Hertzog & Nesselroade, 1987; Mayer & Carroll, 1987). However, although the ARCL approach offered many advantages, a particularly salient disadvantage was that this statistical model did not correspond well to the increasingly complex developmental theories of child and adolescent alcohol use. Whereas the ARCL model was focused on a series of time-adjacent relations among measured variables, contemporary developmental theories hypothesized the existence of individual differences in dynamic developmental trajectories of alcohol use and abuse. It was almost exactly 25 years ago when methodological and computational advances first allowed for the estimation of these hypothesized individual trajectories of development and growth. This class of analytical techniques is generally referred to as growth curve modeling.

Two broad lines of statistical methods led to the development of growth curve models. First, the multilevel modeling (MLM) framework approached this problem from the perspective of hierarchical structure such that repeated assessments were naturally nested within individuals (e.g., Bryk & Raudenbush, 1987). This in turn allowed for the incorporation of time as a continuous explanatory variable in studies of stability and change. Second, the structural equation modeling (SEM) framework approached this problem from the perspective of repeated measures as multiple indicators that defined one or more underlying latent factors (e.g., Meredith & Tisak, 1990). These latent "growth" factors were hypothesized to represent unobserved continuous trajectories of change over time. The ability to empirically estimate individual variability in smoothed developmental trajectories using either the MLM or the SEM approach has allowed for a much greater correspondence between the theoretical models and corresponding statistical models.

Growth modeling methods have been widely used in alcohol research in the last 25 years. For example, in our own

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work, Chassin, Curran, Hussong, and Colder (1996) used a latent growth curve model to test the relation between parental alcoholism and trajectories of adolescent substance use as mediated by parenting, temperament, and stress and negative affect. Curran, Stice, and Chassin (1997) extended this model to examine the simultaneous relations between trajectories of adolescent substance use and trajectories of deviant peer affiliations. Similarly, Jackson, Sher, and Schulenberg (2005) applied multivariate growth models to study the conjoint development of problem behaviors and young adult alcohol use.

The design and dissemination of both the MLM and the SEM growth models has greatly enhanced our ability to estimate individual trajectories of alcohol use and to test an entire class of research hypotheses in ways not previously possible. However, these growth models imposed assumptions that may not always be met in practice. One key assumption is that the sample under study is a random one draw from a homogeneous population in which all individuals are governed by the same parametric form of the growth trajectory; any differences among individuals is reflected in the magnitude of these trajectory parameters.

Substantial problems can arise if there are subsets of individuals within the sample who are characterized by fundamentally different trajectories. For example, a subset of children may follow an increasing and then stable trajectory of alcohol use while another subset follows an increasing but then decreasing trajectory of use. To fit a single parametric function to the pooled subset of individuals would not accurately capture the relevant trajectories for either of the groups. This concern prompted the development of a broad collection of models commonly termed latent class analysis or growth mixture modeling (e.g., Muthen, 2001; Muthen & Shedden, 1999; Nagin, 1999; Nagin & Tremblay, 2001).

These latent class models do not assume that the sample represents a random one drawn from a homogeneous population. They instead allow for the potential existence of two or more discrete groups (or classes) of individuals where class membership is not directly observed (and are thus latent). The typical goal of the analysis is to first identify these latent classes and then assign individuals to the most likely class based on information that was observed in the sample. These latent class models have been applied to test a variety of questions in the development of alcohol use, most notably concerning multiple age-related trajectories of alcohol outcomes (described in more detail below).

Growth mixture models offer an important alternative to the sometimes restrictive assumption of homogeneity of functional form imposed by standard MLM and SEM growth models (e.g., Muthen, 2003; Nagin, 2004). Although highly intriguing from a theoretical perspective, a variety of concerns have been voiced about the utility of these methods in practice (e.g., Bauer, 2007; Bauer & Curran, 2003, 2004; Eggleston, Laub & Sampson, 2004; Sher, Jackson & Steinley, 2011). A detailed discussion of these concerns is well beyond the scope of the current review, but researchers should be aware of these potential threats to validity when using these approaches.

In addition to the introduction of growth modeling and growth mixture modeling methods, there are a plethora of additional advances over the past 25 years in design, measurement, and analysis that have enhanced our ability to test developmental theories of alcohol use and AUDs. Examples include the ability to model multiple levels of context in development (e.g., repeated measures nested within a child, and a child nested within a family; e.g., Raudenbush & Bryk, 2002); advances in estimation methods that allow for the modeling of dependent variables that are continuously but nonnormally distributed or discrete scales such as binary, ordinal, or counts (e.g., Hedeker & Mermelstein, 2000); methods for studying separate "trait" and age-related "state" components of alcohol use and alcohol problems (Chassin et al., 2012; Park, Sher, Todorov, & Heath, 2011); methods that provide for the incorporation of partially missing data structures both within and across time (e.g., Enders, 2010); and the development of intensive repeated measures designs that permit the gathering of multiple data points on a daily or even hourly basis (e.g., Bolger, Davis, & Rafaeli, 2003). These advances allow us to test our ever increasingly complex theories of the developmental course, causes, and consequences of alcohol use with appropriate analytic methods.

Age-Related Trajectories of Alcohol Use and Alcohol Problems

There is little debate that, at least in most Western cultures, average trajectories of both heavy use and alcohol-related difficulties are characterized by escalation in adolescence, peak levels of use and prevalence of diagnosis in the early 20s, and a decline from this peak into later adulthood (albeit with some variation as a joint function of sex and ethnicity; e.g., Grant et al., 2004). However, these average trajectories are a mixture of different patterns, some of which can deviate dramatically from the average pattern. In order to characterize this heterogeneity, many investigators, employing a range of approaches (e.g., cluster analysis or growth mixture modeling) empirically attempted to "pull apart" the mean trajectory into its component parts.

As summarized in Sher et al. (2011), several broad classes of trajectories have consistently emerged: a low or nonusing trajectory ("low"), a chronic/persistently high use trajectory ("high"), a trajectory marked by high use that gradually declines over the timespan ("decrease"), and a trajectory marked by low use that gradually increases over the time span ("increase"). In addition, small group of studies also have identified a "fling" or "time-limited" trajectory (e.g., Schulenberg, O'Malley, Bachman, Wadsworth, & Johnson, 1996) or a trajectory with moderate levels of alcohol involvement (e.g., Colder, Campbell, Ruel, Richardson, & Flay, 2002). It is perhaps surprising that, with the exception of those studies whose designs likely censored the ability to resolve either "decreasing trajectories" or "increasing trajectories," the number and forms of trajectories obtained were highly similar despite study variation with respect to baseline age of the

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sample, the observation period covered, the frequency of measurement occasions, and the specific drinking-related measure employed. That is, the observed trajectories tended to follow, but not invariably so, the "cat's cradle" pattern (i.e., low, high, increase, decrease), although their relative prevalences vary as a function of age in expected ways. This cat's cradle phenomenon is observed in other areas of substance use research (e.g., tobacco; Hu, Muthen, Schaffran, Griesler, & Kandel, 2008) and in the study of conduct problems (Odgers et al., 2007). The seeming ubiquity of these patterns of trajectories, independent of the stage of development being studied, suggests that we should be cautious when interpreting the meaning of these trajectories and always be mindful that these techniques are not "carving nature at her joints" and therefore should avoid reifying them (Nagin & Tremblay, 2005).

Moreover, when thinking about trajectories of alcohol involvement, it may often be more fruitful to shift the emphasis of the "intercept" or start point of the trajectory from baseline age to a baseline drinking milestone (e.g., first time used or first time drunk) as is often done in studies of telescoping (Hussong, Bauer, & Chassin, 2008; Jackson, 2010) and shift our level of analysis to stage of use and use age as a moderator because the correlates of use at a given age may be different than the correlates of stage of use (Sher, Gotham, & Watson, 2004). More traditional trajectory modeling may obscure this important difference.

The Importance of Adolescence as a Developmental Period

Although adolescence has long been recognized as an important developmental period for the onset and escalation of drinking behavior, the past 25 years have seen significant new insights into the neurobiology of adolescent development as well as the impact of alcohol use on the adolescent brain and cognitive functions, both of which have influenced our understanding of the developmental psychopathology of AUDs. Recent studies have suggested that adolescent development is characterized by a gap between changes in dopaminergic reward systems (producing increases in sensation seeking and reward seeking beginning at puberty) and the slower and more gradual development of top-down cognitive control, which is correlated with increased myelination both within the prefrontal cortex and between the cortical and subcortical areas (Paus, 2005; for a review of adolescent neurobiological development and its implications for risk-taking behavior, see Steinberg, 2008). This gap between increased neurobiologically based increases in reward seeking and slower developing cognitive control systems predisposes adolescents toward risk-taking behavior. Moreover, although it has long been recognized that adolescent alcohol use is heavily influenced by peer contexts (including peer modeling and reinforcement of alcohol use as well as peer provision of drinking opportunities), recent neuroscience approaches have revealed that, for adolescents, the presence of peers itself activates the same reward centers that lead to risky behavior (Chein, Albert, O'Brien, Uckert, & Steinberg, 2011). Thus, peers may serve to accentuate reward seeking and to make alcohol use particularly rewarding for adolescents. Note that this neurobiological underpinning of adolescent risk taking is consistent with an evolutionary perspective, which points to increases in risk taking and exploration during adolescence across animal species and the adaptive value of adolescent exploration and risk taking for promoting independence and mate selection (Ellis et al., 2012). Nevertheless, although exploration and risk taking may have adaptive value, risk taking that manifests in the form of adolescent heavy episodic drinking also results in significant elevation in risk for short-term negative consequences (including physical assault, rape, and traffic accidents; Hingson, Zha, & Weitzman, 2009) as well as long-term risk for AUDs (e.g., Grant et al., 2006).

Moreover, recent and accumulating evidence suggests that adolescents may be particularly sensitive to the neurotoxic effects of alcohol, with associated neurocognitive damage that may be relatively durable. Heavy exposure to alcohol in adolescence has been associated with structural and functional brain deficit, as well as deficits in cognitive functioning (Clark, Thatcher, & Tapert, 2008; De Bellis et al., 2000; Hanson, Medina, Padula, Tapert, & Brown, 2011; Hargreaves, Quinn, Kashem, Matsumoto, & McGregor, 2009; Tapert, Brown, Baratta, & Brown, 2004; Zeigler et al., 2005). The apparent heightened sensitivity of the adolescent brain to alcohol-related insult is thought to be associated with neurodevelopmental vulnerability to disruption of the extensive remodeling of the brain that takes place in adolescence (e.g., synaptic pruning; Clark et al., 2008). The associated neurocognitive deficits, especially those associated with deficits in prefontal and limbic systems (Monti et al., 2005), could pose added risk for heavier alcohol involvement and engagement in a range of externalizing behaviors (e.g., Bava & Tapert, 2010). Although a definite causal relation in humans has yet to be established due to the paucity of truly prospective designs that assess youths prior to their first alcohol exposures, rodent models of adolescent ethanol exposure (Crews, Braun, Hoplight, Switzer, & Knapp, 2000; Spear, 2000; Swartzwelder, Wilson, & Tayyeb, 1995) suggest that adolescence is a time of heightened sensitivity to persistent neurologic damage (i.e., greater deficits associated with adolescent exposures than with preadolescent or adult exposures). In addition, these rodent models allow molecular analyses of mechanisms underlying neurotoxic effects that appear heightened in adolescence and could presage enhanced susceptibility to addiction (Crews & Vetreno, 2011). Multiple research programs are currently tracking cognitive functioning, neurophysiological correlates of cognition, and structural brain changes along with alcohol (and other drug) exposures. These studies will more definitively establish temporal ordering of these exposures and aberrant neurodevelopment and cognitive deficits, characterize dose–response relationships, and identify specific risk factors that increase neurologic vul-

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nerability to alcohol-related (and other-substance related) neurological insult.

Importance of Emerging Adulthood as a Developmental Period

That alcohol use and AUDs often decline when individuals reach their early 20s and take on adult work and marital roles has been recognized in the scientific literature for more than 25 years. For example, in the mid-1980s, Zucker (1986) described "developmentally limited" AUDs and Yamaguchi and Kandel (1985) linked age-related declines in marijuana use to role socialization pressures that are associated with the demands of adult roles. However, in the last 25 years there have been important advances in our understanding of the importance of emerging adulthood as a developmental stage. Arnett introduced the term "emerging adulthood" in 2000 to describe the period of exploration in between adolescence and the full assumption of adult roles that occurs in some cultures. The last 25 years have also seen prospective longitudinal studies that have documented age-related patterns of alcohol use (e.g., Chen & Kandel, 1995). We now know that "maturing out" of alcohol use and AUDs is more complex than originally proposed. For example, Lee and Chassin (in press) found that declines in drinking were not uniform, but rather they were more common among heavier, problem drinkers than among other types of drinkers, and declines among heavy drinkers reflected moderation of drinking rather than cessation of drinking. Vergés et al. (2012) using NESARC data found that, although persistence in alcohol dependence was somewhat lower in early adulthood than later in life, age-related declines in alcohol dependence were largely produced by reductions in new onset (i.e., decreasing hazard rates). Their findings question the notion that "developmentally limited" alcohol dependence should be considered a distinct subtype of AUD, suggesting instead that alcohol dependence might be thought of as either "short duration" or "chronic and episodic" (while acknowledging that short duration AUDs may be more common at earlier ages). They also note that role transitions influence alcohol dependence at all ages, not just during emerging adulthood, albeit perhaps in different ways for men and women, for different roles and at different ages.

Moreover, although research has confirmed the relation of role transitions to changes in alcohol use and AUDs, studies have now revealed other changes that occur during emerging adulthood that also contribute to age-related changes in alcohol outcomes. As described earlier, neurobiological research has documented the gradual maturation of cognitive control systems that continues into the early to mid-20s (Paus, 2005; Steinberg, 2008) and these increases in cognitive control would be expected to reduce risk-taking behavior in general and alcohol use more specifically.

Accompanying neurodevelopment and a shifting landscape of developmental roles and responsibilities are changes in personality. A major change that has occurred in our understanding of human development that has been increasing gaining recognition is that personality is not a fixed characteristic that is immutable over the life course but, rather, shows fairly dramatic normative changes throughout adolescence and later adulthood (e.g., Roberts, Walton, & Viechtbauer, 2006) with increasing psychosocial maturity evidenced by decreases in negative affectivity and increases in conscientiousness and related self-control. Recent studies have demonstrated that individual differences in age-related changes in personality, including declines in behavioral disinhibition/impulsivity and negative emotionality/neuroticism and increases in conscientiousness, are correlated with declines in alcohol use in emerging adulthood and early adulthood (Littlefield, Sher, & Wood, 2009). It is important that the association between personality change and change in drinking in emerging adulthood persists even when adult role occupancies are considered (Littlefield et al., 2009). Moreover, even in late adolescence when (on average) rates of alcohol use and levels of use are increasing at the same time that impulsivity and sensation seeking are decreasing, we see that smaller decreases in these traits are associated with bigger increases in alcohol (and other substance) use (Quinn & Harden, 2013).

In addition, the association between individual differences in personality change and changes in drinking is further associated with concomitant changes in drinking motivation (Littlefield, Sher, & Wood, 2010) placing the well-documented association between personality and drinking motivation (e.g., Cooper, Frone, Russell, & Mudar, 1995) firmly within a developmental context. That is, change in personality is associated with changes in the reasons that drinkers report for their drinking.

Of course, the association between changes in personality and changes in alcohol use may reflect either the influence of personality on drinking or the influence of drinking on personality. It has been known for many years that some personality traits in alcoholics (especially those related to negative affectivity) become more normalized over a period of abstinence. Such findings are consistent with basic neurobiological findings showing that neuroadaptation to chronic alcohol (or other drug) use creates a persistent negative affective state motivating continued use (e.g., Baker, Piper, McCarthy, Majeskie, & Fiore, 2004; Koob & Le Moal, 2008). What is less clear is the degree to which less severe drinking patterns alter personality traits and, if they do, how persistent such changes are. There is some recent evidence that alcohol use might influence age-related personality change, but the results are not totally consistent. Hicks, Durbin, Blonigen, Iacono, and McGue (2012) found that individuals whose AUD began in adolescence and was persistent into young adulthood did not show normative declines in negative emotionality, and they suggested that alcohol use interfered with age-related declines in negative emotionality. However, Littlefield, Vergés, Wood, and Sher (2012) found that the effects of alcohol involvement on personality appeared to operate on shorter timeframes and to be dependent upon the developmental period under investigation (see also Quinn, Stappenbeck, &

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Fromme, 2011). At this point, the relation between personality change and drinking patterns has yet to be fully characterized, but existing data suggest that there is a fairly strong association and that the relation between personality and drinking is a dynamic one.

Influences of the Timing and Context of Developmental Transitions

Another clearly emerging trend in the last 25 years is the increased consideration of the timing and context surrounding developmental transitions. Accumulating findings generally reflect a core tenet of developmental theory; namely, the salience not only of developmental milestones but also of the timing and social context surrounding those milestones. Two exemplars of these trends are studies of pubertal timing and role transitions involving leaving home, parenthood, and marriage.

In terms of pubertal timing, recent studies replicate early findings that girls with an earlier age of pubertal onset, even within the same family (Dick, Rose, Viken & Kaprio, 2000), show an increased risk for alcohol use (Aro & Taipale, 1987; Stattin & Magnusson, 1990). For boys, findings are less consistent. Although studies of European boys find a greater likelihood of alcohol use among early maturers (Bratberg, Nilsen, Holmen, & Vatten, 2007; Dick & Mustanski, 2006), studies of American boys have reported a greater risk of alcohol use for both early maturers (Costello, Sung, Worthman, & Angold, 2007) and late maturers (e.g., Ge et al., 2006; Graber, Seeley, Brooks-Gunn, & Lewinsohn, 2004). Finally, there is evidence of "catch-up" effects in which the effects of early maturing are reduced by late adolescence and early adulthood (Dick et al., 2000; Graber et al., 2004; Taga, Markey, & Friedman, 2006). However, longer term effects can still be detected. For example, early-maturing boys, though not girls, have reported continued elevations in alcohol use in late adolescence (Kaltiala-Heino, Koivisto, Marttunen, & Frojd, 2011) and a greater onset of AUDs in the transition to adulthood (Graber et al., 2004).

Studies of mechanisms indicate that morphological rather than hormonal changes accompanying puberty are more strongly predictive of alcohol involvement (Costello et al., 2007). Such morphological changes may increase risk for drinking, particularly in girls, because they are a social signal of maturity that increases the likelihood of associating with older (and thus more alcohol involved) peers. Support for this hypothesis remains mixed, however. For example, Westling, Andrews, Hampson, and Peterson (2008) showed that a lack of parental monitoring strengthened the relation between early pubertal timing and alcohol use for boys and girls, but deviant peer affiliations only mediated the pubertal timing effect in girls. Moreover, other studies fail to find that peer affiliations account for this association between pubertal timing and alcohol use (Dick et al., 2000). Finally, moderators of early pubertal maturation have been identified such that the risk for substance use associated with early pubertal maturation is increased by lax parental supervision (for girls) and a family history of substance use, psychiatric problems, or crime (for boys; Costello et al., 2007) as well as urban residence (Dick et al., 2000).

Studies have also examined the association between alcohol use and the timing of role transitions, including the transition to independent living (i.e., leaving the parental home), marriage, and parenthood. Youths who leave the family home earlier, with greater conflict, disagreement, and negative feelings, and motivated by more risk-promoting reasons, are more likely to come from families with well-established risk factors, such as parental alcoholism (Hussong & Chassin, 2002). Although not directly studied in relation to drinking outcomes, early transitions out of the family home for reasons such as seeking freedom and unhappiness were, in turn, associated with greater adjustment problems (i.e., internalizing and externalizing symptomatology) in young adulthood.

In terms of the timing of marriage, Leonard and Eiden (2007) note that alcohol use may lead both to early marriage as part of a pattern of risky decision making and to delayed marriage owing to alcohol-related difficulties in interpersonal relationship functioning. The data provide little clarity on which of these processes may be more common, with studies showing that drinking predicts both early and late marriage as well as that drinking is unassociated with the timing of marriage (Bachman, Wadsworth, O'Malley, Johnston, & Schulenberg, 1997; Fu & Goldman, 1996; Newcomb & Bentler, 1986). In terms of parenthood, pregnancy reduces substance use in women, though not in men, but the transition to parenthood does produce a reduction in men's drinking (O'Malley, 2004). However, these effects differ as a function of the timing of parenting. For example, Little, Handley, Leuthe, and Chassin (2009) showed that early parenthood was associated with increases in substance use among men and did not produce the typical reductions in substance use for women, whereas an older and more normative age of parenthood was associated with declines in use.

Although we have focused on developmental transitions (and the timing of transitions) occurring in the adolescent and early adult years, difficulties in negotiating earlier developmental transitions (e.g., school entry) may also be critically important (Zucker, Donovan, Masten, Mattson, & Moss, 2008). Difficulties in negotiating early developmental transitions may initiate cascading processes of risk (see the 2010 Special Issue of *Development and Psychopathology* for studies of developmental cascades). Similarly, later life transitions such as retirement also influence drinking, although, as we have discussed, the specific context of the transition (in this case involuntary job loss versus voluntary retirement) in interplay with characteristics of the individual determine the effect (for a review, see Kuerbis & Sacco, 2012).

Advances in Etiology: Modeling Multiple Pathways

As shown in our discussion to this point, the past 25 years have seen important research advances in identifying hetero-

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geneity in trajectories of alcohol use and AUDs in relation to developmental milestones, particularly during adolescence and emerging adulthood. Similar advances have been seen in our understanding of etiological mechanisms. Consistent with a developmental psychopathology perspective, there has been an emphasis on equifinality; that is, multiple pathways leading to the development of AUDs (Cicchetti & Rogosch, 1996; Cicchetti & Toth, 2009). Each of these pathways reflects the interplay between variables at multiple levels, from genetic risk to broad societal and historical context (Burnette & Cicchetti, 2012; Cicchetti & Dawson, 2002). Moreover, these models propose cascading effects over development (Masten & Cicchetti, 2010), thus tracing risk and resilience pathways from prenatal exposure, early adversity, child maltreatment, and early childhood characteristics to adult alcohol outcomes (for a review of advances in understanding of these early developmental antecedents, see Zucker et al., 2008). Here we describe some of the advances in three major biopsychosocial etiological models of AUD: deviance proneness (externalizing) models, stress and negative affect (internalizing) models, and alcohol effects models. Note that these models are not mutually exclusive and are systematically interrelated (Sher, 1991).

Deviance Proneness Pathways

An "externalizing" or "deviance proneness" pathway to alcohol use and AUDs has long been recognized and has been associated with an early onset of AUD (Cloninger et al., 1981; Iacono, Malone, & McGue, 2008; Zucker, 1986) These models view adolescent alcohol use and AUDs within a broader externalizing spectrum (Iacono et al., 2008) and provide one explanatory mechanism both for the development of adolescent alcohol use in general and for the intergenerational transmission of AUDs. In these models, children of parents with AUDs are at risk for a heritable predisposition to "behavioral undercontrol" (Sher, 1991) or "behavioral disinhibition" (Iacono et al., 2008). The effects of behavioral undercontrol are thought to be exacerbated by poor parenting, which includes low levels of parental support, lack of monitoring, and lack of moderate, consistent discipline. Such poor parenting is likely to be provided by parents who themselves have AUDs and is also likely to be evoked by undercontrolled children (Barnow, Schuckit, Lucht, John, & Freyberger, 2002; Chassin, Pillow, Curran, Molina, & Barrera, 1993; Eiden, Edwards, & Leonard, 2007; Mezzich et al., 2007). In addition to experiencing poor parenting, children who are behaviorally "disinhibited" or "undercontrolled" are at risk for school failure and for ejection from mainstream peer groups (Veroneau, Vitaro, Brendgen, Dishion, & Tremblay, 2010), which leaves them exposed to similarly undercontrolled peers (Sijtsema, Lindenberg, & Veenstra (2010) who provide opportunities and norms that encourage substance use behavior (Haller, Handley, Chassin, & Bountress, 2010).

Although deviance proneness pathways have long been recognized, the last 25 years have seen important progress toward understanding these etiological mechanisms. Consistent with a developmental psychopathology perspective, these models have been formally articulated as probabilistic risk pathways involving the interplay of factors at multiple levels (Sher, 1993). That is, a heritable propensity to behavioral undercontrol exerts its effects in interplay with family, peer, school, neighborhood, and larger societal influences.

In recognizing the multilevel nature of these models, an important advance in the past 25 years has been the increasing number of tests of gene–environment interaction from a developmental perspective. Twin studies suggest that the heritability of alcohol use is low in adolescence and increases with age, and heritability also increases in environments with greater alcohol availability and exposure (Kendler, Gardner, & Dick, 2011; Kendler, Schmitt, Aggen, & Prescott, 2008; Young-Wolff, Enoch, & Prescott, 2011). Moreover, findings from twin studies suggest that gene–environment interactions likely operate at two broad levels (Sher et al., 2010): environments that interact with genes to affect underlying vulnerability (e.g., affectivity or self-regulation) and environments that interact with genes to facilitate the expression of vulnerability (e.g., permissive environments). The task of selecting environments to study in the context of gene–environment interplay is daunting because of the extensive range of potential environmental influences, including life-stage specific factors (e.g., prenatal exposures and various social roles that vary over the life course). Given the number of possible single gene by single environment interactions, the task of identifying valid gene–environment interactions becomes quite challenging. Strategies must be developed that enhance the likelihood that valid, meaningful interactions are detected, and spurious ones minimized, including a need for careful replication.

With this caveat in mind, the last 25 years have seen a noteworthy increase in the number of studies of various environmental factors that have incorporated measured genes into their assessments (Dick, Latendresse & Riley, 2011; Young-Wolff et al., 2011). These studies suggest that genetic risk for adolescent alcohol use is magnified in the presence of poor parenting and reduced in the presence of good parenting. For example, studies have found that parental supervision and involvement reduced the risk for adolescent substance use that was associated with the methionine allele of the catechol-*O*-methyltransferase Val158Met genotype (Laucht et al., 2012) and the serotonin transporter linked polymorphic region gene (Brody et al., 2009) and that parental rule setting reduced the risk for adolescent alcohol use that is associated with the TAQA1 genotype (Van der Zwaluw et al., 2010) although this genotype did not show differential effects as a function of parent rejection, overprotection, or warmth (Creemers et al., 2011). Less work has tested the evocative effects of adolescents' genotypes on parenting, and this is an important direction for future study (Leve, Harold, Ge, Neiderhiser, & Patterson, 2010). Moreover, the challenge for future research is to employ sufficiently large samples (either by studying large cohorts or by pooling samples through data sharing) to have adequate power to conduct meaningful tests

of gene–environment interaction, while allowing for multiple testing with adequate control of Type 1 errors.

Another important advance in studies of deviance proneness models has been the recognition that behavioral undercontrol is a complex construct that is actually composed of multiple different propensities for impulsive behavior or "rash action" and that these propensities are differentially related to alcohol outcomes. Multiple taxonomies have been proposed to capture dimensions of behavioral undercontrol including a differentiation between top-down and bottom-up processes, and between the inability to control behavior in "hot" motivational contexts involving response to rewards and punishments versus "cold" contexts in which rewards and punishments are not salient (Castellanos-Ryan, Rubia, & Conrod, 2011; Handley et al., 2011; Nigg, 2000; Whiteside & Lynam, 2001). Moreover, multiple measures have been developed to assess these propensities, including trait questionnaire measures (e.g., the UPPS; Whiteside & Lynam, 2001) and behavioral tasks of response inhibition, attentional control, and working memory (see Dick et al., 2010; Nigg, 2000). In addition, behavioral tasks have been developed that draw on multiple dimensions of self-regulation that affect behaviors such as decision making (e.g., the Iowa Gambling Task; Bechara, Damasio, Damasio, & Anderson, 1994), risk taking (e.g., the Balloon Analogue Risk Task; Lejuez et al., 2002), and the delay discounting of rewards (e.g., the Monetary Choice Questionnaire; Kirby, Petry, & Bickel, 1999). Although measures that tap different aspects of behavioral undercontrol are only weakly related and self-report measures are generally weakly related to behavioral tasks (Birkley & Smith, 2011; Dick et al., 2010; White et al., 1994), many of these measures predict alcohol use outcomes (Dick et al., 2010; Lejuez et al., 2010), with weaker and less consistent relations being found for lack of perseverance, poor response inhibition, and working memory (Birkley & Smith, 2011; Castellanos-Ryan et al., 2011; Handley et al., 2011). As described below, traits reflecting controlled, effortful processes such as working memory and response inhibition may be better thought of as moderators that either limit or enhance the influence of more automatic, motivational, approach tendencies toward alcohol (such as traits like sensation seeking, positive and negative urgency, as well as automatically activated positive associations to alcohol use). The idea that controlled, reflective processes moderate the effects of automatically activated alcohol-related associations is the central proposition of dual process models of alcohol use (see, e.g., Wiers, Ames, Hofmann, Krank, & Stacy, 2010).

Deviance proneness models view affiliation with alcohol-use-promoting peers as the proximal mediator that leads to alcohol use among "behaviorally undercontrolled" individuals. Although peer influences on alcohol use have long been recognized, the last 25 years have seen advances both in methods of investigation and in our understanding of peer influences. Genetically informative studies have helped to disentangle the effects of peer selection from the effects of peer influence. In support of a peer selection effect, genetic factors have been shown to influence peer affiliation (Chassin et al., 2012; Cleveland, Wiebe, & Rowe, 2005; Fowler, Settle, & Christakia, 2011; Hill, Emery, Harden, Mendle, & Turkheimer, 2008). However, peers further influence substance use outcomes over and above genetically mediated peer selection effects (Chassin et al., 2012; Harden, Hill, Turkheimer, & Emery, 2008). Thus, peer influences may serve to mediate the effects of genetic risk on alcohol outcomes. There is also support for interactions between genetic risk and peer influences. For example, Harden et al. (2008) found that adolescents who were genetically at risk for alcohol and tobacco use were also the most vulnerable to influences from their closest friends. Moreover, Park et al. (2011) found that carriers of the dopamine receptor D4 long allele were more affected by sorority/fraternity involvement in influencing alcohol dependence in young adulthood.

Another recent methodological advance has been to move beyond individuals' reports of their peers' drinking and map the individual's social network. Social network analysis not only identifies the characteristics of networks that are associated with alcohol use but also models the spread of drinking through a social network. For example, Ennett et al. (2006) found that adolescent substance users were less embedded in a social network, had greater status in the network, and had great social proximity to other peers who used substances. Rosenquist, Murabito, Fowler, and Christakis (2010) found that changes in the drinking behavior of an individual's social network predicted later changes in the individual's drinking (with female network members having stronger influence than male network members). Drinkers could be influenced either toward abstinence or toward increased drinking, suggesting that the social network can have either positive or negative influence. However, an individual's drinking was not influenced by the drinking behavior of neighbors or coworkers. In addition, alcohol use itself may function to facilitate the formation of social groups. Sayette et al. (2012) administered alcohol to small groups of social drinkers who were initially unacquainted with each other. Those who were given alcohol (compared to placebo) showed more nonverbal social bonding behavior and also self-reported more social bonding.

Stress and Negative Affect or Internalizing Pathways

Over the years, research regarding the roles of stress and negative affect in the development of alcohol use and AUD has increasingly focused on identifying mechanisms of risk, moderating factors that indicate contexts and intrapersonal resources that exacerbate or mitigate this risk, and the developmental unfolding of these processes over time and drinking history. In part driving these foci was the need to address inconsistent support in the literature for the association between indicators of stress/negative affect and alcohol involvement, particularly for adolescents.

Although much of the focus on stress-drinking relations concern adolescent and adult samples, exposure to stress and trauma early in development have also been shown to have long-term impact on alcohol involvement. For example, child-

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hood maltreatment and exposure to other traumatic events are associated with alcohol dependence in adolescence and adulthood (Clark et al., 1997; Fenton et al., 2012). Current developmental psychobiological models view stress influences as dynamic and likely to change with development and with different patterns of stress exposure, caregiving support, and genetic vulnerability (Gunnar & Quevado, 2007). Early trauma and chronic stress, in particular, are posited to inhibit neurogenesis, and frequent activation of the stress response will tax finite resources, increasing overall allostatic load and resulting in disruptions of neuronal plasticity and neurotoxicity. In these ways, early severe or chronic stress exposure impacts not only the immediate stress response but also future neurobiological stress responding, such that those with early stress exposure, including various forms of trauma and maltreatment, are more vulnerable to the detrimental effects of later stress exposure, forming a negative feedback loop over development. Andersen and Teicher (2009) also propose that early stress exposure produces anhedonia, a state that increases motivation for substance use. De Bellis (2001) extended these links among environmental stress, physiological stress responding, and problem behavior in development. He notes that dysregulation in the major biological stress response systems associated with childhood trauma and maltreatment have adverse influences on brain development that enhance vulnerability to psychopathology, including posttraumatic stress disorder and depression that precede AUDs.

In adolescence, there are consistent findings that those who experience high levels of life stress are more likely to use alcohol and to escalate the quantity and frequency of their use over time (Chassin et al., 1996; Hussong & Chassin, 2004; Wills, Vaccaro, McNamara, & Hirky, 1996). However, empirical support for negative affect as the mediator of the association between stress and alcohol use is more consistent in studies of adults than in studies of adolescents. Studies of adolescents support a more nuanced association, with negative affect predicting drinking behavior only for some youths and in some contexts (see Colder, Chassin, Lee, & Villalta, 2010).

Although there are several mechanisms that may underlie the association between negative affect and alcohol use, an important emerging distinction is between mechanisms that identify between-person patterns of risk versus within-person patterns of risk. The increased use of temporally informative designs (e.g., diary studies, experience sampling, events sampling, and ecological momentary assessment) have provided a novel approach to distinguishing affect as an indicator of when a given individual is at risk (i.e., on days when they have more negative affect than usual, a within-person indicator) as well as which individuals are at risk (i.e., those with greater negative affect or stress, a between-person indicator). This distinction has important implications not only for prevention and intervention program development but also for refining our etiological theories of the developmental mechanisms that underlie these drinking behaviors.

Key within-person mechanisms implicated in the relation between negative affect and drinking include variants of the classic negative reinforcement model (i.e., drinking to alleviate distress; Jellinek, 1960; Wikler, 1948). Much of the intensive daily assessment research that evaluates this mechanism focuses on early adulthood, when drinking is more frequent and more easily observed on a daily basis. In general, these studies show that on days when adults report higher levels of negative mood than is typical for themselves, they also tend to report higher rates of consumption, urges to drink, and alcohol-related problems (Armeli, Tennen, Affleck, & Kranzler, 2000; Litt, Cooney, & Morse, 2000; Simons, Gaher, Oliver, Bush, & Palmer, 2005). Although this association is generally posited to be stronger in those with coping motives for drinking, evidence has both supported the cross-level interaction between coping motives and daily negative affect predicting drinking (Arbeau, Kuiken, & Wild, 2011) and failed to do so (Armeli, Conner, Cullum, & Tennen, 2010; Hussong, Hicks, Levy, & Curran, 2001; Todd, Armeli, Tennen, Carney, & Affleck, 2003). Identifying additional potential moderators of this risk, other studies show that individuals with daily intense negative emotions are less likely to consume alcohol on a given day if they are better at identifying and differentiating discrete forms of negative emotion (Kashdan, Ferssizidis, Collins, & Muraven, 2010) and if they have positive social support in their close friendships (Hussong et al., 2001). A challenge with these models is that the interval over which to test the association between negative affect and drinking (i.e., hours, days, or weeks) is unclear. Moreover, survival analyses of daily assessment data suggest that the size of the interval may also be psychologically meaningful, because factors such as coping motives and greater alcohol-related consequences predict a shorter interval between peak levels of negative emotion and subsequent drinking (Hussong, 2007).

Many fewer studies use these methods to study drinking in adolescence (although, for a review of this work predicting adolescent smoking, see Mermelstein, Hedeker, & Weinstein, 2010). Following a small sample of rising ninth graders using a 21-day experience sampling paradigm, we have found higher rates of drinking on days following elevated negative mood only in youths who report more depressive symptoms, fewer conduct problems, and poorer parent emotion socialization (Gould, Hersh, & Hussong, in press; Hersh & Hussong, 2009; Hussong, Feagans-Gould, & Hersh, 2008; Reimuller, Shadur, & Hussong, 2011). Moreover, some of these effects may strengthen over time. For example, we found that after (but not before) the transition to high school, adolescents who reported more sadness than usual were also more likely to report same-day drinking, but only if they had lower levels of parental involvement in their lives (an indicator of social support; Gottfredson & Hussong, 2011). Thus, associations between negative affect and drinking could strengthen over times of transition, stress, or developmental gain, particularly for youths lacking alternative coping skills or resources.

Augmenting the intensive daily assessment designs and physiological studies of negative affect-drinking associations are studies of the potential genetic underpinnings of this risk mechanism. There are fewer genetically informative studies of stress-negative affect pathways than of the deviance prone-

ness pathways that were described earlier (though see Nurnberger, Foroud, Flury, Meyer, & Wiegand, 2002). Although a review of the literature by Saraceno, Munafó, Heron, Craddock, and Van Den Bree (2009) found an emerging set of potential markers for co-occurring alcohol problem use and internalizing symptoms (e.g., the serotonin transporter short allele, the monoamine oxidase A low-activity alleles, and the dopamine D2 receptor *Taq* A1 allele), this literature is increasingly focused on identifying gene–gene and gene–environment interactions underlying this association.

Recognition is also emerging in the literature that more integrative models are needed to understand the association between negative affect and drinking associations within a larger developmental context. For example, Hussong, Jones, Stein, Baucom, and Boeding (2011) define the potentially unique risk processes underlying the internalizing pathway as emphasizing problems with emotion regulation across the life span. This pathway recognizes negative reinforcement as a central process translating deficits in emotion regulation into alcohol-related behaviors and risk for addiction, particularly pertinent for predicting a negative affect form of AUD as a salient outcome. Drawing from a larger developmental literature, this pathway posits that risk for later AUD may first emerge as inhibited temperament and emotion dysregulation in early childhood. Studies showing that these early temperament markers predict later alcohol use further support the salience of early behavioral inhibition for the internalizing pathway to AUD. For example, Caspi, Moffitt, Newman, and Silva (1996) found that inhibited (fearful, shy, and easily upset) 3-year-olds, compared to their peers, had higher rates of depression and, for boys, alcohol-related problems at age 21. Other studies also suggest that indices of internalizing behavior between ages 3 and 10 are predictive of more alcohol-related problems and disorder in midadolescence to early adulthood (for a review, see Zucker, 2006). For those following this pathway, accumulated risks associated with continued emotion dysregulation over development further increase risk for later AUD, particularly for youths in at-risk homes marked by parental alcoholism and comorbid affective disorders with well-documented associations with poor child outcomes (Hussong, Flora, Curran, Chassin, & Zucker, 2008; Hussong et al., 2007).

In turn, early emotion dysregulation and related internalizing symptoms are associated with interpersonal skill deficits and difficulties in peer contexts (Graber & Sontag, 2009; Nelson, Rubin, & Fox, 2005; Rubin & Mills, 1991). Some forms of negative affect, particularly those that may relate to cautious behavior and withdrawal from peers, may actually reduce risk for drinking during adolescence. For example, Kaplow, Curran, Angold, and Costello (2001) showed that young teens with a separation anxiety disorder (often linked to reduced peer interaction) delayed the onset of alcohol use compared to their peers, whereas teens with a generalized anxiety disorder (which may not pull youths out of peer contexts) had an earlier onset of alcohol use. Whether or not adolescents engage in drinking associated with negative affect may also be moderated by a host of factors (Colder et al., 2010; Sher, 1991). For example, theorized moderators associated with the internalizing pathway predict that adolescents with more positive expectations for the effects of alcohol use, interpersonal skill and coping deficits that lead to associations with deviant peers or to social withdrawal and the desire to self-medicate, and coping motives for alcohol use may be more likely to drink in response to cues for negative affect and internalizing symptoms. Supporting this assertion is evidence suggesting that a stronger endorsement of coping motives increases the risk for drinking on days characterized by greater fear and shyness (Hussong, Galloway, & Feagans-Gould, 2005) and that more disengaged coping increases the association between stress and substance use in adolescents (Wills, Sandy, Yaeger, Cleary, & Shinar, 2001). However, by young to midadulthood, access to alcohol is easier and may not be as strongly mediated by the peer and social context. To the extent that drinking behavior is initiated, positive expectancies for alcohol use and related coping motives for drinking may be reinforced by experience, creating the potential for cyclical patterns of negative affect and drinking implicated in negative reinforcement models of addiction (Baker et al., 2004; McCarthy, Curtin, Piper, & Baker, 2010).

An active area of research concerns the intersection of negative affect and behavioral undercontrol indicators of risk. Studies that control for externalizing symptoms when predicting alcohol use from internalizing symptoms often fail to find unique effects of negative affect (Capaldi, 1991; Capaldi & Stoolmiller, 1999; Miller-Johnson, Lochman, Coie, Terry, & Hyman, 1998). However, studies indicate that the two forms of symptomatology may interact. Some studies suggest that the two together exacerbate risk (Simons et al., 2005; Wardell, O'Connor, Read, & Colder, 2012). In addition, borderline personality disorder, a condition associated with high levels of negative affectivity, affective instability, and poor impulse control, is associated with high levels of substance use and dependence (Trull, Sher, Minks-Brown, Durbin, & Burr, 2000). However, other studies, including those using experience sampling data, indicate that negative affect may be a stronger predictor of drinking behavior in the absence of externalizing symptomatology (Dierker, Vesel, Sledjeski, Costello, & Perrine, 2007; Hussong, Feagans-Gould, et al., 2008). Physiological data provide another perspective for considering this interaction. Some laboratory data suggest that individuals who are temperamentally "underregulated" may derive the strongest psychophysiological stress-response-dampening benefits from consuming alcohol (Levenson, Oyama, & Meek, 1987), consistent with a hypothesized stronger link between stress or negative affect and substance use for individuals who are low in self-regulation. In general, although deviance proneness and "stress and negative affect models" have often been studied in isolation, the interrelations between these models are an important future research direction.

In addition, it is important to understand how negative affect influences drinking not only over development but also

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over the course of drinking history. In a reformulation of the classic negative reinforcement model, Baker et al. (2004; McCarthy et al., 2010) suggest that after problematic drinking patterns become entrenched, negative affect and internalizing symptoms that motivate alcohol use may occur outside of awareness, triggered by interceptive cues that precede affective symptoms of withdrawal. As a result, the phenomenological experience of negative affect as a cue for drinking may change over the course of drinking history, necessitating changes not only in our conceptualization of this association but also in our methods for assessing it.

Finally, research is needed concerning specific types of negative affect that signal risk for drinking. For example, Shoal, Castaneda, and Giancola (2005) suggest that worry may reduce risk for substance use in adolescents who are high in negative affect. Moreover, the role of positive affect remains an area of increasing study. Although distinct models for drinking related to negative affect (i.e., negative reinforcement models driven by coping motives for drinking) and positive affect (i.e., positive reinforcement models driven by enhancement motives for drinking; Cooper, 1994) have received support over the years, alternative revised models of reinforcement sensitivity (Gray & McNaughton, 2000) may suggest ways in which these models may also be integrated in future research.

Alcohol Effects Pathways

Alcohol affects virtually all major neurotransmitter systems, especially at levels associated with intoxication, and these neurotransmitter systems play a key role in regulating cognition, affect, and behavior (e.g., Vengeliene, Bilbao, Molander, & Spanagel, 2008). For purposes of discussion, the subjective and hedonic effects of alcohol can be separated into three broad classes: (a) positive reinforcing effects (e.g., euphoric or arousing) that are thought to be mediated, like other drugs of abuse, by dopamine reward pathways; (b) negatively reinforcing effects (e.g., anxiolytic or antidepressant) that are thought to be largely mediated via GABA-ergic pathways; and (c) punishing effects such as acute sedation and discomfort that could arise from a number of factors (e.g., peripheral effects of alcohol or its metabolite, acetaldehyde, on the gastrointestinal and vascular system or direct effects on brain systems related to sedation). From the perspective of pharmacological vulnerability, one could be at increased risk for the development of alcohol problems because of individual differences in any of these effects (e.g., heightened reward or decreased sensitivity to punishment). Early evidence suggested that individuals at risk for alcohol problems experienced a low level of response to alcohol effects (Schuckit, 1984; Schuckit & Smith, 1996) with later modifications suggesting a low level of response to aversive effects but increased levels of response to reinforcing effects (Newlin & Thompson, 1990; see Morean & Corbin, 2010; Quinn & Fromme, 2011, for recent reviews).

In the past 25 years, there has been considerable debate as to how much various effects of alcohol are attributable to direct effects of alcohol on brain systems associated with reward or punishment versus how much is mediated via effects on cognition in interaction with environment. Less than 25 years ago, Steele and Joseph (1990) proposed the "alcohol myopia" theory of alcohol effects, which posits that the effect of alcohol is contingent upon information processing of more or less salient features of the drinking context, and such an interaction between cognition and the environment could lead to reinforcement, punishment, or disinhibition depending upon the nature of the situational context. After a decade of research, Lang, Patrick, and Stritzke (1999) reviewed the available human literature on the effects of alcohol and emotion and concluded that "evidence of intrinsic reward or selective stress reduction seems neither powerful enough nor reliable enough to account for the widespread appeal of alcohol and the prevalence of alcoholism" (p. 360). They also argued that most of alcohol's effects on affect and emotional responding were secondary to effects on cognition (and subsequent processing of relevant contextual cues and their relevance to the self). However, it has long been known that intermediate doses of alcohol have unpredictable effects on negative emotions, though as the dose of alcohol approaches those associated with "binge" levels of intoxication, negatively and positively reinforcing effects tend to be observed reliably, independent of context (see Sher, 1987). More recent studies (e.g., Donohue, Curtin, Patrick, & Lang, 2007; Sher, Bartholow, Peuser, Erickson, & Wood, 2007) have clearly demonstrated unconditional effects on negative emotions across diverse measures and experimental paradigms (see Sher & Grekin, 2007). From an etiological perspective, the evolving research evidence suggests that different processes may be involved in understanding the reinforcing properties of alcohol at lower and higher doses, and suggests that there are different individual difference risk factors for negative consequences at lower versus higher doses.

The reinforcing effects of alcohol typically are experienced on the ascending limb of the blood alcohol concentration (BAC) curve (measured from the time that drinking is initiated until individuals reach their peak BAC), and the punishing effects typically occur on the descending limb of the BAC curve (measured at the time subsequent to that when individuals have reached their peak BAC), rendering the net effect of alcohol to be one that is biphasic (Sher, Wood, Richardson, & Jackson, 2005). These biphasic effects are observable in the laboratory, and individuals also report expecting to experience such effects prior to drinking (Earleywine, 1994; Earleywine & Martin, 1993). Heavier drinkers have been found more likely to experience stronger stimulant effects relative to sedative effects, whereas lighter drinkers have been found to experience the opposite. The biphasic effects of alcohol are subject to individual differences (see Sher & Wood, 2005; Sher et al., 2005).

From the point of view of developmental psychopathology, an important advance in studies of pharmacological vulnerability models is evidence that adolescents may show unique patterns of reactivity to ethanol that make them par-

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ticularly susceptible to drinking at high levels. Spear and Varlinskaya (2005) review studies with rodents indicating that adolescents are less sensitive than are their adult counterparts to a variety of punishing effects that could serve to limit consumption both acutely (e.g., sedation or motor impairment) and following intoxication (e.g., hangover-like symptoms). In addition, adolescents appear to be especially sensitive to alcohol-related social reward, which can goad further drinking. This configuration of factors is similar to the pattern of effects noted for children of alcoholics (e.g., Newlin & Thomson, 1990) and could represent an added level of risk associated with adolescence. In addition to these different levels of sensitivity, chronic alcohol administration effects on tolerance development appears to differ between adolescents and adult rats in complex ways (Morales, Varlinskaya, & Spear, 2011). For ethical reasons, there are few highly controlled studies of alcohol consumption in adolescents, but tolerance is one of the most commons symptoms of dependence in high school drinkers (among those who meet criteria for an AUD and among those who do not; Lewinsohn, Rohde, & Seeley, 1996). Moreover, during emerging adulthood, rates of tolerance *decrease* even among those who maintain heavy drinking patterns over an extended period of time (O'Neill & Sher, 2000), findings consistent with adolescence being a period of time of altered alcohol sensitivity.

From a theoretical perspective, individual differences in alcohol effects should influence beliefs and expectancies about alcohol, which further influence drinking behavior. The last 25 years have seen the recognition that consciously held beliefs and expectancies about alcohol are important, but so are positive and negative associations to alcohol that are more automatic and less likely to be in conscious awareness (such as implicit attitudes and automatic approach or avoidance tendencies). There have been important developments in methods for measuring these implicit associations as well as evidence that these automatic associations predict alcohol outcomes (Roefs et al., 2011; Stacy and Wiers, 2010). In addition to global scores on these measures, researchers have just begun to apply process models of these tasks to alcohol outcomes. For example, O'Connor, Lopez-Vergara, and Colder (2012) found that (for children ages 10–12), those who had begun to drink had weaker automatic activation of negative alcohol-related associations than did those who were abstainers. Moreover, the relation between automatic associations and drinking outcomes varies for individuals at differing genetic risk, in terms of μ-opioid receptor M1, dopamine receptor D4 (Pieters et al., 2011), aldehyde dehydrogenase 2, and catechol-*O*-methyltransferase (Hendershot, Lindren, Liang, & Hutchison, 2012). The last 25 years have also seen the development of dual process models of drinking behavior, in which the relation between automatically activated alcohol associations and drinking behavior is moderated by reflective, conscious, controlled processes (for a review, see Wiers et al., 2010). In support of these models, the relation between drinking behavior and implicit associations or automatic approach tendencies toward alcohol has been shown to be weaker for individuals with higher levels of working memory and for individuals with higher levels of response inhibition (Houben & Wiers, 2009; Peeters et al., 2012; Thush et al., 2008).

Conclusions and Future Directions

The last 25 years have seen impressive advances in our understanding of the developmental psychopathology of alcohol use and AUDs. Much has been learned about the early antecedents and cascading processes of risk that set the stage for later alcohol problems, and one future direction that emerges from this work is the need to understand the ways in which early adversity and maltreatment influence the later development of risk for AUD. Moreover, although the last 25 years have seen an expansion of a developmental psychopathology approach as applied to alcohol outcomes in adolescence and emerging adulthood, there has been less application of a developmental psychopathology perspective in terms of understanding alcohol outcomes in midlife and later life. Age-specific etiological factors in midlife and late-life (including the effects of role transitions) are in need of future study. Future research on aging samples is warranted, particularly because higher alcohol use among the baby boomer cohort forecasts increases in alcohol problems among older individuals (National Institute on Alcohol Abuse and Alcoholism, 2000). These alcohol outcomes are not only important in their own right but also are of potential significance in influencing alcohol use in offspring and grandchildren. Thus, future research should continue to expand the study of risk and resilience processes across the life span and across generations.

Much has been learned about the importance of adolescence and emerging adulthood as developmental stages for the initiation and decline in alcohol use and AUDs, and these recent advances illuminate areas of needed future research. Prospective studies are needed to identify potential neurotoxic effects of adolescent alcohol exposure and the effects of adolescent alcohol use on cognitive functioning. These studies require multiple levels of measurement (including neuropsychological assessment and imaging studies of underlying neurocircuitry). These studies should identify the dose–response relation between adolescent drinking and potential cognitive sequelae, and should specify the role of adolescent drinking in the context of correlated risk factors such as other forms of drug use. Such research is currently underway through a National Institute on Alcohol Abuse and Alcoholism initiative. Moreover, research is needed to illuminate the influence of adolescent exposure to alcohol on the normative development of cognitive control from adolescence to adulthood. Although there appear to be complex, bidirectional relations between alcohol use and personality development, little is known about the dosage and duration of alcohol intake that might influence the development of cognitive control and psychosocial maturity at these ages. In addition, further research is needed to clarify the nature of sensitivity to alcohol's rewarding and aversive effects during adolescence and the

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ways in which such developmentally specific alterations in sensitivity may influence trajectories of alcohol consumption.

The recent concerns with potential neurotoxic effects of adolescent exposure to alcohol as well as adolescents' altered sensitivity to the rewarding and aversive effects of alcohol have potential implications for policy. In particular, they raise concerns with recent proposals (such as the Amethyst initiative) to lower the minimum legal drinking age. Many arguments against the lowering of the minimum legal drinking age cite public health protections against the short-term adverse consequences of drinking, such as reductions in fatal and nonfatal accidents and crime (e.g., Carpenter & Dobkin, 2011), and these are clearly important arguments. However, if adolescent alcohol exposure does prove to have neurotoxic effects and creates cognitive impairment, the potential long-term consequences provide additional arguments against lowering the drinking age. A better quantification of the ages of vulnerability and dose effects that produce negative effects will better inform such policy debates. Moreover, if adolescent alcohol exposure is found to impair the normative age-related development of cognitive control and psychosocial maturity, this may also have policy issues in terms of adolescents' legal culpability. Steinberg and Scott (2003) argued that adolescents have reduced criminal culpability because of their immature decision-making capacity. If adolescent alcohol exposure reduces the development of cognitive control and psychosocial maturity, it might result in a longer duration of immaturity or a greater lack of maturity, both of which can be factors in determining criminal culpability.

For methodological issues, the increasing sophistication of quantitative methods requires continuing improvement in measurement and in understanding measurement equivalence across developmental periods. Such a developmental approach to measurement will be important in assessing the performance of the new DSM-5. Moreover, although assessments of AUDs are currently based on self-report, in the future it may be possible to incorporate the use of behavioral and psychophysiological methods that tap into underlying processes of addiction. Finally, given the growing interest in gene–environment interaction, it is important for future studies to adopt strategies that provide sufficient statistical power to detect interactions, use appropriate methods to probe the form of interactions, and minimize spurious findings through replication and controls for multiple testing. Studies of gene–environment interaction also need to test hypotheses within a developmental context and within the context of gene–environment correlation (i.e., passive, active, and evocative effects).

In terms of etiological pathways, both the deviance proneness pathway and the stress and negative affect pathway are in need of studies that provide greater specificity in terms of the facets of behavioral undercontrol and "negative affect" that predict alcohol outcomes. Understanding these facets on multiple levels (e.g., behavioral tasks and psychophysiological measures) is a particularly important and challenging task. Applying process models that illuminate the multiple processes underlying behavioral task performance may be particularly helpful in achieving this goal. Moreover, future studies should identify potential contextual influences (such as parenting and family influences) that affect the development of behavioral undercontrol and affect regulation. Identifying these potentially modifiable contextual factors is particularly important, because they are potential targets for preventive and treatment interventions. In this regard, it will be important to study alcohol problems in terms of their boundaries with other forms of internalizing and externalizing psychopathology as well as their boundaries with other forms of substance use disorders. Moreover, although we have made substantial progress in understanding the deviance proneness, stress and negative affect, and alcohol effects models, these models are not meant to be considered in isolation of each other, but rather they are systematically interrelated. For example, individuals with high levels of behavioral undercontrol may experience greater stress response dampening benefits of alcohol and thus be more likely to use alcohol to cope with stress. Moreover, early trauma exposure may also be a common antecedent with important implications for both emotional and behavioral dysregulation. These mechanisms integrate all three hypothesized pathways, and future studies should examine such integrative effects.

Finally, future research should also continue to test these questions on multiple levels. We have already described the increasing number of genetically informative studies with specific suggestions for future methods, but other multiple-level studies are needed that include identifying underlying neural mechanisms, social network influences, and neighborhood and social policy influences. It is particularly important to continue to develop theory and empirical evidence to identify the ways in which developmental pathways of risk and resilience to AUDs might be influenced by varying cultural values across demographic groups (race, ethnicity, socioeconomic status, and gender). Given the gains of the past 25 years and the current amount of research interest and activity, we anticipate that the next 25 years of advances in the developmental psychopathology of alcohol use and AUDs will be equally productive.

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