Development and Psychopathology (2024), 1–15
doi:10.1017/S0954579424000671

CAMBRIDGE UNIVERSITY PRESS

Special Issue Article

Understanding alcohol use and alcohol use disorders from a developmental psychopathology perspective: Research advances, challenges, and future directions

Laurie Chassin[1] and Kenneth J. Sher[2]

[1]Arizona State University, Tempe, USA and [2]University of Missouri, Columbia, MO, USA

Abstract

As part of the special issue of *Development and Psychopathology* honoring the remarkable contributions of Dr Dante Cicchetti, the current paper attempts to describe the recent contributions that a developmental psychopathology perspective has made in understanding the development of alcohol use and alcohol-related problems over the lifespan. The paper also identifies some of the future challenges and research directions. Because the scope of this task far exceeds the confines of a journal length article this paper does not attempt a comprehensive review. Rather, it builds on an earlier review and commentary that was published in *Development and Psychopathology* in 2013, with a similar goal.) Building on that work and updating its conclusions and suggestions for future directions, the current paper emphasizes findings from the research areas that were identified for further study in 2013 and the findings that have been published since that time.

Keywords: alcohol use; alcohol use disorders

(Received 12 January 2024; accepted 19 January 2024)

We are delighted to contribute to this special issue of *Development and Psychopathology* to honor the remarkable career contributions of Dr Dante Cicchetti. In his 1984 editorial in *Child Development*, Dr Cicchetti wrote about the historical isolation of academic psychology from clinical psychology and psychiatry and emphasized the importance of integrating multiple disciplines and multiple levels of analysis within a developmental psychopathology framework. In the 40 years since that influential special issue, the field of developmental psychopathology has grown dramatically and made major contributions to the understanding of trajectories of adaptation and maladaptation over the lifespan, as exemplified by Dr Cicchetti's own ground-breaking work.

In this paper, we attempt to describe recent contributions of a developmental psychopathology perspective to understanding the development of alcohol use and alcohol-related problems over the lifespan and to identify some of the future challenges and research directions. We recognize that the scope of this task far exceeds the confines of a journal length article so we do not attempt a comprehensive review. Rather, we build on an earlier review and commentary that we published with colleagues in *Development and Psychopathology* in 2013, that had a similar goal (Chassin et al., 2013).

Our earlier paper largely focused on studies of three major multilevel developmental pathways underlying the development of alcohol use and alcohol-related problems, noting that these pathways were probabilistic, and not mutually exclusive, demonstrating equifinality and multifinality. These pathways, which still dominate the research on alcohol use disorders (AUDs), are the externalizing pathway, the internalizing pathway and the alcohol effects pathway. The internalizing and externalizing pathways emphasize gene-environment interplay between child temperament and parenting behaviors that results in children who are dysregulated, resulting in internalizing and/or externalizing symptomatology. In a developmental cascade, those children experience early school difficulties. Externalizing children are likely to either 'select' or be 'selected' into affiliations with peers who use alcohol or facilitate alcohol use whereas internalizing children may use alcohol to regulate negative affect. The alcohol effects pathway emphasizes the reinforcing effects of alcohol, suggesting that individuals for whom alcohol has the largest positive benefits and the least negative effects are most at risk (see e.g., Chassin et al., 2013).

Our 2013 paper identified multiple directions for future research. We called for research on the role of early trauma and expansion of studies of gene-environment interplay with larger samples. We called for studies of alcohol problems in midlife and late life as well as studies of the consequences of adolescent drinking and the effect of minimum legal drinking ages. Building on our earlier work, the current paper emphasizes findings from the research areas that we identified for further study. We focus on research findings published since our 2013 paper and we attempt to highlight contributions of work that was conducted from a developmental psychopathology perspective.

Corresponding author: L. Chassin; Email: laurie.chassin@asu.edu

Cite this article: Chassin, L., & Sher, K. J. (2024). Understanding alcohol use and alcohol use disorders from a developmental psychopathology perspective: Research advances, challenges, and future directions. *Development and Psychopathology*, 1–15, https://doi.org/10.1017/S0954579424000671

Characterizing the problem: definitions and the epidemiology of alcohol involvement from a life course perspective

Definitions and distinctions

When conceptualizing alcohol involvement over the life course, we must first distinguish among various aspects of alcohol use and its consequences. At the most basic level, we need to distinguish alcohol consumption per se from its social, personal, and physical sequelae, including but not limited to AUDs. It is also important to delineate important facets of both consumption and consequences since more refined distinctions have important implications for understanding different etiological correlates and pathways and consequences.

At its most fundamental level, we can distinguish whether someone is *a drinker or not*; if someone does not drink, they are not at risk for an AUD and related conditions. However, this crude distinction obscures whether one is drinking infrequently and/or at low levels or engaged in a heaver pattern of use.[1] Various governmental agencies (Dietary Guidelines Committee, 2020; National Institute on Alcohol Abuse and Alcoholism (NIAAA), 2023) have attempted to provide guidance on what constitutes low level or moderate use and have proposed criteria for drinking levels that might be considered to be minimally harmful to overall health. Recent guidelines have reduced the consumption levels that are considered minimally harmful with respect to diverse health outcomes and vulnerability to drinking problems. In the United States, drinkers are encouraged to stay within both daily limits (e.g., 1 drink/day for women, 2 drinks/day for men) and weekly limits (e.g., 7 drinks/week for women, 14 drinks/week for men) (Dietary Guidelines Committee, 2020; National Institute on Alcohol Abuse and Alcoholism (NIAAA), 2023); Canada has recently proposed low-risk drinking guidelines not to exceed 2 standard drinks per week (Paradis et al., 2023). The issue of where to draw the threshold (or indeed, if there should be a threshold versus a continuum of risk) and how to communicate this information are areas of contemporary debate (Shield et al., 2024). Especially controversial is the issue of whether or not such guidelines should be developed for specific age groups (Global Burden of Disease 2020, Bryazka et al., 2022).

"Binge drinking" and "high-intensity drinking" (HID)

Drinking at high levels is often the focus of developmental psychopathology studies given the likelihood of acute harms and association with AUDs. In particular, there is interest in drinking that results in acute intoxication, often operationalized as obtaining a blood alcohol concentration of .08% or more and the associated concept of drinking four or more drinks (women) or five or more drinks (men) over two hours (National Institute on Alcohol Abuse and Alcoholism (NIAAA), 2023). Given that many individuals drink at levels that far exceed the 4+/5+ threshold, some recent research has focused on "high-intensity" drinking (HID) or drinking at levels corresponding to eight or more (women) or ten or more (men) on a drinking occasion (Patrick & Azar, 2018). Of particular relevance to developmental studies, the 4/5 binge drinking recommendation is based on assumptions about "typical" body mass and ethanol metabolism of adults and may not be optimal for comparing individuals at different ages since both of these variables can change over the course of development. Some researchers argue for different developmental thresholds based on body mass considerations alone (Donovan, 2009).

Overall, level of consumption appears to be largely monotonically related to a wide range of health outcomes, often as an exponential function when one gets past low levels of consumption, with the classic example being likelihood of fatal motor vehicle crashes (e.g., Taylor & Rehm, 2012). In addition to some consequences being associated with acute intoxication (e.g., injuries, STIs) other medical consequences (e.g., various cancers and gastrointestinal diseases) appear to reflect the cumulative effect of drinking over time (e.g., Bergmann et al., 2013).[2] While the association between alcohol and STIs is most likely mediated by risky sexual behavior, it is also possible that some risk is conveyed by alcohol's effect on biological susceptibility to infection (Llamosas-Falcón et al., 2023). Thus, multiple pathways, including both acute and chronic effects of alcohol operating via different mechanisms could contribute to a given alcohol-outcome relation.

The association between consumption and health must be viewed developmentally since different ages are associated with different health conditions and different vulnerabilities. For example, alcohol-related adverse health effects in adolescents and young adults are more likely to be attributable to intentional and nonintentional injuries and infectious diseases where in older adults, effects are more associated with cancer and cardiovascular disease (Bryazka et al., 2022). Moreover, these age effects are conditioned by geographic regions since different regions of the world tend to be characterized by different disease burdens which are differentially affected by alcohol (Bryazka et al., 2022).

Although heavy consumption is associated with a host of behavioral, medical, and public safety concerns and those who drink more heavily are more likely to be diagnosed with an AUD, the association is far from unity and some individuals diagnosed with mild or moderate AUD report comparable heaviness of drinking compared to those with severe AUD (e.g., Lane & Sher, 2015). Even among those reporting binge drinking on a daily basis, a substantial minority (28% of young adults 21–37 and 34% of older adults 38+) failed to meet criteria for an AUD (Vergés et al., 2018) in one national study. On the other hand, many individuals who meet criteria for AUD do so with relatively light drinking patterns. Although alcohol consumption is a necessary condition for diagnosing with an AUD, amount or frequency of consumption is not part of the criteria set or diagnostic algorithms for AUDs.

[1]Historically, interest in alcohol consumption has been concerned with drinking at levels associated with "typical" adult consumption, that is, one or more full drinks on an occasion. However, recently, there has been interest in "sipping" alcohol among children and early adolescents (Donovan & Molina, 2008; Jackson et al., 2015; Watts et al., 2021), whether the correlates of early sipping are similar to those of "full drinks" and whether such early experimentation presages later heavy use or problems. Available data suggest that even among 9 and 10-year old's, these seemingly minor forays into alcohol sampling by sipping are associated with externalizing related personality traits, mood disorder symptomatology, and psychotic-like symptoms (Watts et al., 2021), all risk factors for substance use problems.

[2]Until recently, low level use, in comparison to abstention or heavier use, was considered "healthy" (at least for middle-aged men) (e.g., Klatsky et al., 1992). However, these findings have been challenged by newer data and by meta-analyses that attempt to control for the "sick quitter" effect (i.e., some individuals quit drinking alcohol because of health-related issues) (e.g., Stockwell et al., 2016; Zhao et al., 2023). While some meta-analyses suggest that for some medical conditions, lower levels of alcohol use may pose little risk and may even have salutary effects on conditions like ischemic heart disease and Type 2 diabetes (e.g., Rehm et al., 2021), there appears to be consensus that consumption of approximately 2 standard drinks/day or more is generally associated with poorer health outcomes at any age (e.g., Rehm et al., 2021).

https://doi.org/10.1017/S0954579424000671 Published online by Cambridge University Press

AUD criteria set and diagnostic algorithm

In the Diagnostic and Statistical Manual, version 5 (DSM-5; American Psychiatric Association, 2013) AUD diagnosis is based on meeting two or more of 11 diagnostic criteria: hazardous use (e.g., drinking and driving), drinking despite social interpersonal problems related to use, failure to fulfill role responsibilities, withdrawal, tolerance, repeated attempts to quit or cut down, much time spent using and getting over the effects of drinking, continuing to use despite physical/psychological problems related to use, giving up important activities due to use, and craving alcohol. Within DSM-5, the severity of an AUD is based on criterion counts with 2–3 criteria met considered mild, 4–5 criteria met considered moderate, and 6+ criteria met considered severe irrespective of specific criteria met. Many individuals meeting minimum criteria for an AUD do not fit clinical and lay stereotypes of or addiction (Bickel et al., 2019; Lane & Sher, 2015) as someone can diagnose with an AUD on the basis of having, say, some tolerance to alcohol and a heedless pattern of drinking (e.g., hazardous use) with no signs of compulsive use, craving, withdrawal, or major interpersonal, psychological, or physical problems (Bickel et al., 2019).

AUD heterogeneity

There are 2036 possible AUD symptom configurations (out of 2048 possible combinations of the 11 AUD criteria) and, especially within mild and moderate levels of severity, there can be no or little overlap in symptoms profiles between two individuals with AUD. Severe AUD, which requires 6 or more criteria, guarantees at least minimal overlap but the symptom profiles of these individuals still display considerable heterogeneity (Lane & Sher, 2015). The exact nature of the heterogeneity is important since recent research indicates that only some criteria presage progression from mild- or moderate-severity AUD to severe AUD (Miller et al., 2023). This high degree of heterogeneity and lack of a conceptual "core" to the construct of AUD has led some investigators to consider alternatives to the overall syndromal approach embodied in the DSM. At least three of these alternative approaches warrant mention. The first is the abandonment of the categorical approach that is embodied in the DSM-5 in favor of a hierarchical, dimensional approach that views alcohol-related pathology as part of higher-order dimension of externalizing pathology as in the Hierarchical Taxonomy of Psychopathology (HiToP; Kotov et al., 2017). Such a dimensional approach eschews what some may argue is the arbitrary categorical distinction between the presence/absence of disorder and makes the additional prediction that AUD severity is not just related to comorbidity but also the severity of comorbid conditions (e.g., Helle et al., 2020).

A second development is the movement away from diagnostic entities towards studying symptoms as important phenomena in their own right, a tactic employed in symptom network modeling approaches (e.g., Anker et al., 2017; Conlin et al., 2022; Huth et al., 2022). Such approaches are well suited for studying the functional relationships among symptoms and the unfolding of more severe symptomatology and holds the potential for more clearly delineating cascades and symptom progression associated with different etiologies.

A third approach is an endophenotypic one that focuses on underlying mechanisms/processes such as the Alcohol Addiction Research Domain Criteria/Addictions Neuroclinical Assessment (AARDoC/ANA, Kwako et al., 2016), the Etiologic, Theory Based, Ontogenetic Hierarchical Framework (ETOH; Boness et al., 2021), and NIDA's Phenotyping Assessment Battery (PhAB; Watts et al., 2023). Such endophenotypic approaches arguably facilitate translation between basic research on different pathways to addiction prominent in developmental psychopathology approaches and potentially identify promising targets for intervention. For studying AUDs developmentally, these endophenotypic approaches are arguably less "hobbled" than symptom-based approaches, which suffer from the dual problems of relevance of particular diagnostic criteria for different life stages and situational contexts as well as developmental variation in assessing these criteria described in the next section. However as noted by Conradt et al. (2021), despite holding great promise for a more refined approach to studying developmental psychopathology and consistency with ideas outlined by Cicchetti (1984), these approaches are not as developmentally informed as they could be.

Developmental considerations

Developmental issues affect the diagnosis of AUDs (and dimensional alternatives such as HiToP) in, at least, two major ways: (1) developmental relevance of various criteria, and (2) age-related understanding of interview questions used to diagnose AUDs (Marmet et al., 2019). In the case of developmental relevance, compulsive use is assessed, in part, on the basis of role interference (e.g., as a parent, worker, student). However, various important life roles change as a function of development. For example, the responsibilities of adolescents or emerging adults are often more limited than those in mid-adulthood. However, in later adulthood, the situation may reverse with reduced parenting and work responsibilities as children become independent and with retirement. With respect to age-related understanding of interview questions designed to assess AUD criteria, querying about change in amount of alcohol needed to achieve a certain effect (i.e., tolerance) requires detailed consideration of someone's drinking history (Chung & Martin, 2005; O'Neill & Sher, 2000). Consequently, it is not surprising to find that the self-reported prevalence of tolerance decreases rapidly in early adulthood even though one would expect it to be increasing as a function of neuroadaptation (Marmet et al., 2019; O'Neill & Sher, 2000). Notably, "drinking more than intended" is often misinterpreted in ways that could lead to false positive reports (e.g., drinking in response to peer pressure rather than impaired control) or possibly false negative reports (e.g., the question presupposes limits which may not be present, especially. in younger individuals). Given variation in the developmental relevance and age-related understanding of survey questions, researchers have called for more nuanced and developmentally sensitive assessment of AUD criteria (e.g., Karriker-Jaffe et al., 2015). Nevertheless, available data need to be viewed through the lens that assessments of AUD might not be equally reliable and valid over the life course. For example, in a study of DSM-IV dependence criteria, Vergés et al. (2021), found that some criteria (persistent desire or unsuccessful efforts to cut down or control drinking and drinking despite physical or psychological problems) were less predictive of re-endorsement (i.e., persistence) three years later in younger as compared to older adults and speculated that this was "spurious desistance (particularly from milder AUDs) due to false positive symptom endorsement" (p. 454). The dramatic increase in AUD prevalence in emerging adulthood may be due, in part, to age-related problematic criteria (although desistance of AUD in the third decade of life appears to be largely a valid finding; Vergés et al., 2012). These caveats must be considered when examining the age-prevalence curves described below.

https://doi.org/10.1017/S0954579424000671 Published online by Cambridge University Press

Race/ethnicity and cultural variables in AUD diagnosis

When trying to understand group differences in the development and course of drinking, a number of considerations come into play ranging from population stratification of alcohol metabolizing enzymes (Wall et al., 2016) to macro-environmental, cultural variables. However, overall, these pharmacokinetic differences may be less important than other risk factors (Jones, 2019).

Of particular importance from a developmental psychopathology perspective is whether there is intrinsic bias in diagnostic criteria that will tend to either over-pathologize certain groups or fail to resolve important symptomatology. Boness et al. (2023) have recently argued that traditional diagnostic approaches overly focus on presumed deficits within an individual and fail to consider who is defining what is normal and pathological and, equally if not more importantly "contributions of systemic factors and contexts that may influence alcohol consumption" (p. 308). They go on to argue that "considering contextual factors in parallel to mechanisms may also encourage more explicit treatment of AUD as a 'culturally bound syndrome' influenced by sociopolitical factors" (p.310).

Recent data from electronic health records (EHRs) in a large cohort of more than 700,000 veterans (Vickers-Smith et al., 2023) suggest considerable bias in that there is substantial variation in rates of clinical (ICD-defined) AUD across race/ethnicity, with Black adult males having substantially higher prevalence of AUD in comparison to Hispanic and White adult men at a given level of consumption for all but the highest and lower levels of consumption. A similar, but not identical pattern, was observed in women. Note that these racial disparities were not evident when comparing rates of medical conditions associated with heavy alcohol consumption. In combination, these findings suggest there might be significant racial bias in the diagnosis of AUD, at least in clinical practice. This bias might reflect under-diagnosis in White patients, over-diagnosis in Black patients, or both.

Vickers-Smith et al. (2023) point out that population-based epidemiological studies using standardized, structured interviews fail to reveal the type of disparities noted in their study. Indeed, in large, population-based studies such as NESARC-III (Grant et al., 2015), lifetime AUD rates were higher in White (32.6%) than in Black (22.0%) participants suggesting clinical bias more than definitional bias was primarily responsible for the observed differences in the Vickers-Smith et al. (2023) study but the question remains open.[3] Item Response Theory (IRT) studies have demonstrated differential item functioning (DIF) across race/ethnicity groups. For example, Harford et al. (2009) found, "compared to their White counterparts with the same AUD severity, Black males were more likely to report tolerance, withdrawal, and legal problems and less likely to report hazardous use." Indeed, the high DIF surrounding legal problems led to this criterion being dropped in the DSM-5 (Hasin et al., 2013). However, AUD-IRT meta-analyses have demonstrated considerable heterogeneity of both thresholds and discrimination estimates across AUD assessment instruments (Lane et al., 2016; Vize & Lane, 2022) but owing to a lack of diversity in many existing AUD-IRT studies, the reliability of DIF associated with race/ethnicity has not been systematically explored across studies. Based on the existing literature, it would seem that there is some degree of systematic bias in diagnostic criteria, their operationalizations, and their implementation. Characterizing the nature and extent of such biases is important for both basic research and clinical practice and should be a high priority for research on the validity of diagnosis across different demographic groups. Since there is evidence that not just overall prevalence of AUD appears to vary by race/ethnicity but that the shape of the age-prevalence curves does too (e.g.,Vasilenko et al., 2017), it is important to consider whether such findings reflect true differences or higher-order (e.g., ageXrace) DIF.

[3]Careful examination of NESARC-III past-12-month data (Grant et al., 2015) indicates that White and Black participants have comparable AUD prevalences and focusing on Severe AUD, rates appear somewhat higher in Blacks (3.9%) vs. Whites (3.2%). Because the VA Study used lifetime rates from EHRs, it makes some sense to use lifetime rates from NESARC-III. However, lifetime rates derived from a single assessment occasion are known to grossly underestimate rates derived from multiple assessments in survey studies (Haeny et al., 2014). Consequently, attributing the bias to clinicians rather than something intrinsic to the criteria and their operationalization is likely premature.

Age-related prevalence curves of consumption and AUD

If there is one salient "fact" about the epidemiology of both alcohol consumption and AUD it is that there are strongly age-graded systematic increases in the prevalence of both variables in late adolescence and early adulthood, peak prevalences typically observed in the third decade of life, and decreases in the prevalence thereafter (e.g. Vergés et al., 2012). Figure 1 provides illustrative recent data from two national studies in the United States that sought to characterize alcohol use and AUDs from adolescence into late adulthood.

Figure 1 (left panel) illustrates some prototypic patterns of alcohol consumption prevalence from late adolescence through later adulthood. Note these US-based data show large increases in the prevalence of any past-month consumption as late adolescents approach and then traverse the minimal legal drinking age (MLDA) with relatively stable high rates throughout adulthood. A similar pattern is noted in the prevalence of "binge drinking" (defined as drinking five or more drinks "in a row") with the notable exception that we see more desistance in this behavior in mid to later adulthood. (Not shown in Figure 1 is the prevalence of daily drinking which shows a considerably lower prevalence but a different developmental pattern with individuals showing a steady pattern of increase from age 18 (~1%) to age 60 (~14%).)

While, at least in the US, the prototypic pattern of increasing rates of binge drinking during the teen years, peak bingeing during in the early 20s, and decreasing rates of binge drinking thereafter has held firm, there has been a notable shift in both the timing of peak prevalence and the rate of decrease following this peak over the past 30 years, with later-born cohorts exhibiting later peaks and slower declines, especially among women (Patrick et al., 2019). While changes in the MLDA and changes in a variety of social roles (e.g., college attendance, marriage, parenthood) could explain some of the observed changes in the age-prevalence curves of binge drinking through early adulthood, adjusting for these covariates did not eliminate the cohort differences in these age-prevalence curves (Patrick et al., 2019). Shifts in the age-prevalence curves of drinking in recent years are not limited to the US but despite superficial similarities, are not necessarily attributable to the same factors. For example, decreases in "overall consumption" have been observed in Russia in more recently born cohorts, ostensibly due to economic factors and reduction of disposable income resulting in not only a decrease in overall consumption but more notable decreases in the consumption of spirits (vodka) (Radaev & Roshchina, 2019). Overall, decreases in youthful drinking appear to have global dimensions with similar observations in the UK, Europe, and Australia. In the United States, however, this decrease represents an increased age of onset and a more protracted period

https://doi.org/10.1017/S0954579424000671 Published online by Cambridge University Press

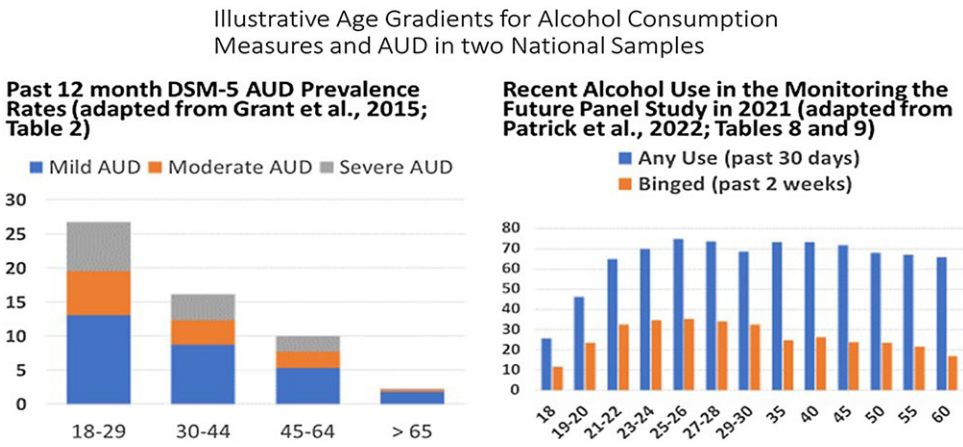

Figure 1. Age-prevalence curves for AUD and alcohol consumption.

of excessive drinking (Patrick et al., 2019) and its attendant harms, which are important to understand from a basic developmental perspective and for public health efforts.

As shown in Figure 1 (right panel), the overall pattern of past-12-month AUD prevalence largely mirrors that observed for binge drinking despite the fact that AUDs are often assumed to be more severe conditions and the stereotype of someone diagnosing with an AUD is not someone in their early 20s[4]. With the introduction of DSM-5, AUDs are now scaled on a severity gradient and so it is useful to consider whether there are differential age patterns associated with the severity of the AUD diagnosis. One might speculate that developmentally limited forms (those evident only in adolescence and/or early adulthood) would be the most likely to be "mild" in severity. However, the proportion of cases that are "mild" remains relatively constant (about 50% of all cases from ages 18 to 64). Perhaps equally important, the proportion of both "moderate" and "severe" cases also remains fairly constant from late adolescence throughout most of adulthood.

One explanation for the high prevalence of AUDs exhibited by younger adults is that many of these diagnoses are false positives based on either limitations of current assessment procedures or the developmental relevance of different diagnostic criteria. However, if this was the case we would expect overall desistance rates to vary dramatically over age, with younger individuals showing higher rates of desistance/remission owing to the presumed higher instability of false positive endorsements. (We might also expect to see larger decreases in younger adults due to higher rates of role transitions.) However, Vergés et al. (2012), found that while desistance/remission rates are somewhat higher among those younger than 30, most of the dramatic age-related decrease in prevalence rates comes from decreases in "new onsets" and "recurrences." That is, "maturing out"/desistance doesn't fully (or even largely) explain the large decreases in the age-prevalence curves of AUDs. Moreover, much of the higher levels of desistance noted in the third decade of life appears to come from individuals "maturing out" of more severe AUDs rather than mild or moderate AUDs (Lee et al., 2018). This suggests that despite the fact many cases of AUDs in the 20s are, by definition, "developmentally limited" in that they are likely to remit, they can often be fairly severe, at least symptomatically.

As noted earlier, population-based samples reveal racial differences in the age-prevalence curves for AUD. For example, in emerging adulthood, Whites have significantly higher rates of AUDs than Blacks and Latinos However, there is a steeper decrease among Whites and by the mid-30s rates of AUD and are similar among Whites and Blacks, both of whom diagnose more frequently than Latinos. These differences disappear as the overall prevalence continues to decline so that by late adulthood, prevalence in all groups tend to be low and similar (see Vasilenko et al., 2017).

The importance of a global perspective

Although data from the US and other Western nations help to define the phenomena of interest, a global perspective is also useful because important determinants of drinking vary dramatically across culture and can be developmentally graded. Perhaps the most tangible example is the dramatic variability across countries in the minimum legal drinking age (MLDA; World Health Organization, 2018). For example, some countries (e.g., Afghanistan, Iran) have total bans on alcohol and others have no MLDA (e.g., Bangladesh, Benin, Guinea). Most countries have limits on sales ranging from ages 16–21 (World Health Organization, 2018) with the United States having one of the highest MLDAs, at age 21. The MLDA appears to be meaningfully related to drinking patterns developing in late adolescence and carrying over into later adulthood (Luukkonen et al., 2023). Legal access to alcohol is associated with the frequency and intensity of drinking, and these effects appear to be stronger in boys and those from lower social classes (Ahammer et al., 2022). Although many believe that the older MLDAs such as in the US may have contributed to drinking excess in adolescence . . . a "forbidden fruit" or psychological reactance explanation, data suggest that adolescents in countries with lower MLDAs typically drink more than their North American age peers (Grube, 2005).

A global perspective on alcohol consumption challenges parochial views of the nature of drinking, its course, and its correlates and cautions us not to overgeneralize our theories. As described earlier, in the US and other Western cultures, there is a strong age-graded, relation between various aspects of consumption (and correlated problems), most typically, systematic increases in consumption during adolescence and young

[4]In one multi-site study across six European countries, Rehm et al. (2015) found that the prevalence of structured interview-based diagnoses of alcohol dependence versus primary care physicians clinical diagnoses (which were based on a standardized set of measures) were roughly comparable. Notably, there was relatively little overlap in the "cases" identified by both methods. Perhaps more critical from a developmental perspective, the age-prevalence trend of diagnoses derived from the structured clinical interviews roughly mirrored those of general population, epidemiological surveys, that is, peak prevalence in the 20s and monotonically declining thereafter. However, the clinicians' diagnoses showed the opposite trend, with the lowest prevalences in the 20s and the highest in late adulthood!! Consequently, epidemiologic data do not necessarily comport with clinicians' perceptions.

https://doi.org/10.1017/S0954579424000671 Published online by Cambridge University Press

adulthood with decreases in later adulthood. However, cross-national studies indicate that these "developmental aspects" of drinking are not universal and age-related declines in consumption tend to occur less outside of North America and Europe (Wilsnack, 2012). Even if there are strong common developmental phenomena (e.g., age-related changes in sensitivity to the motivational properties of alcohol such as reinforcement and punishment) observable across human and non-human animals, these universal influences are moderated by effects of cultural context, which cannot be ignored.

The importance of cohort and period effects

Cohort effects

As noted above, we must consider how historical periods shape not only overall prevalence rates but also the factors behind these shifts so that we can understand them from a developmental psychopathology perspective. For example, in a meta-analysis of six large, nationally representative U.S. studies conducted between 2000 and 2015 (Grucza et al., 2018), past-year prevalence of any use was found to increase overall, reflecting a cohort effect (an effect based on the individual's year of birth) with those born in more recent years showing higher consumption rates. However, this overall increase was largely attributable to women who showed significant increases in past-year drinking in comparison to men whose prevalence was largely unchanged over this period. Of particular interest to developmental theory, the largest increases were observed among those who were older in age. The past-year prevalence of binge drinking also increased over this fifteen-year period. While younger individuals tended to binge more frequently than those who were older (> 50 years of age), here too the oldest participants showed the biggest increases. As noted by Grucza and colleagues, although the increases in the older cohorts are generally modest, the health-related vulnerabilities of older individuals magnify the expected effects of increased consumption: "... it is clear that elevated binge drinking rates among older Americans correspond to a significant increase in alcohol-related morbidity. These observations shed some light on the apparent paradox between relatively small increases in per capita alcohol consumption and large increases in alcohol-related health problems" (Grucza et al., 2018, p. 1948)[5]. However, we should not necessarily assume that just because key aspects of drinking have been changing over time that established correlates of drinking are changing too. For example, in a multi-cohort study of Australian adolescents, Taylor et al. (2024), found significant increases in alcohol abstinence in more recently born cohorts but found that the personality and sociodemographic correlates were similar across cohorts.

Period effects

While cohort effects are those associated with the historical time in which someone was born, period effects refer to effects that influence individuals at the same point in time. It is worth noting that levels of alcohol consumption and associated problems have varied greatly over history (e.g., Hanson, 2013). With currently increasing concerns about the effects of alcohol on health, new drinking guidelines, and increased stigmatization of even lower levels of consumption, could tend to suppress overall levels of drinking. Consequently, we must further consider the historical time period under consideration and how risk factors can be amplified or muted based on the larger sociohistorical context.

With the COVID-19 pandemic, there has been intense interest in how this major event influenced alcohol involvement. In a systematic review Sohi et al. (2022), found that the effects on alcohol consumption appeared to vary considerably across the (largely, high-income) countries included in their review and tentatively concluded that "alcohol use may have decreased in some countries, while heavy episodic drinking and the proportion of people with problematic alcohol use may have increased "(p. 498). The authors speculated that some of these effects may be attributable to increases in off-premise consumption (e.g., "homes" broadly defined) in response to decreases in on-premise (e.g., bars, restaurants) consumption. A recent set of analyses based on the Monitoring the Future Study (Patrick et al., 2022), found that expected (taking into account ongoing historical changes) past-month drinking prevalence during COVID ostensibly decreased while drinking frequency ostensibly increased and, for young adults age 19–-30 binge drinking increased among drinkers. For middle adults (age 35–55), there was in increase in daily drinking. In addition, there was an increase in drinking to cope and more in-home drinking. Moreover, these findings tended to be moderated by age.

Taken together, these findings highlight the embeddedness of lifetime courses of alcohol involvement within larger social and historical backgrounds. From a methodological perspective, they point to the value of cohort-sequential designs in providing an enriched empirical database for understanding developmental psychopathological phenomena so that age, period, and cohort effects can be separately estimated.

Identifying developmental pathways and mechanisms

Gene-environment interplay and the developmental psychopathology of AUDs

One assumption of the developmental psychopathology approach to AUDs is that there are multiple etiological pathways. As described earlier, some of these pathways are alcohol-specific, focusing on the development of individual differences in the acute reinforcing and punishing effects of alcohol and neuroadaptive changes associated with chronic use. Others are more general, including the development of externalizing and/or internalizing problems, which, in turn lead to affiliation with alcohol-using peers who facilitate alcohol involvement (externalizing pathway) or motives to drink to cope with negative affect (internalizing pathway). Developmental psychopathology studies of non-alcohol-specific pathways have focused on temperament/personality and the socializing influences of parents and peers in childhood and adolescence, because adolescence is the typical period for drinking initiation. Broadly speaking, these studies have hypothesized that children with particular high -risk temperaments who receive poor parenting are likely to develop internalizing and/or externalizing problems and that these problems also make more likely for them to affiliate with peers who model and encourage alcohol use (see Chassin et al., 2013).

One important contribution of the developmental psychopathology perspective has been to promote the study of gene-environment interplay in these etiological pathways. A long history of twin and adoption studies have reported that AUDs show significant heritability, but developmental psychopathology theory has produced complex, multilevel studies to directly test gene-environment interaction and gene-environment correlation

[5]Lee et al. (2023) noted that health concerns tended to lead to longitudinal reductions in drinking, especially among older adults but that this phenomenon appeared limited to those with limited AUD symptoms.

https://doi.org/10.1017/S0954579424000671 Published online by Cambridge University Press

processes underlying parent and peer influences over development (Elam et al., 2023; Wilson & Rhee, 2022). Recently, these have included measured genomic risk (often polygenic risk scores, PRS).

Although non-genetically informed studies have provided consistent evidence of parent monitoring, support, and discipline predicting adolescent drinking, (Chassin et al., 2013), some recent genetically informed studies have produced more mixed findings. For example, Zheng et al. (2023) found that correlations between parent supervision and peer drunkenness and alcohol initiation were explained by genetic rather than environmental risk. Moreover, parent supervision moderated genetic risk in opposite directions for alcohol use initiation at ages 12 and 15 (increasing risk at age 12 and decreasing risk at age 15). Stephenson et al. (2022) found little prediction from adolescent social relationships to alcohol resistance among individuals at genetic risk, with the exception of father-child relationship quality. Similarly, Gresko et al. (2022) studied adopted and non-adopted adolescents and found that only parent-child relationship quality (but not other parenting measures) predicted drinking outcomes. Thus, there was little evidence of environmental effects of parenting other than parent-child relationship quality (which predicted drinking among both adoptive and non-adoptive families). Finally, Pasman et al. (2023) did not find parenting effects on drinking outcomes at age 22. The mixed findings from these examples suggest that the socializing influences of peers and parents are unlikely to operate as simple environmental main effects on the development of AUDs but rather need to be considered in the context of gene-environment interplay.

One recent trend in genetically informed studies of AUD etiology is the use of polygenic risk scores to study what Reiss and Leve (2007) termed social mediation of genetic effects, or sometimes called genetic nurturance (Thomas et al., 2023). That is, gene-environment correlations can reflect processes by which genetic risk causes particular environmental exposures, which then cause alcohol use outcomes (Elam et al., 2023, Thomas et al., 2023). Moreover, different forms of gene-environment correlations can underlie social mediation. These include passive gene-environment correlation (for example parents providing both genetic risk for alcohol-related outcomes and genetically influenced parenting practices to their children), evocative gene-environment correlation (for example a child's genetically influenced temperamental characteristics evoking particular social reactions) and active gene-environment correlations (for example, adolescents with particular genetically influenced personality characteristics, choosing particular peer environments).

There is some empirical support for these genetic nurturance effects. Thomas et al. (2023) found that parent relationship discord and divorce mediated the effects of parent genetic risk on offspring alcohol outcomes. However, effects were found for a European American subsample with no evidence of genetic nurturance among a (smaller) African American subsample. Thus, etiological mechanisms may vary with culture. Moreover, social mediation effects may vary for different risk factors. Hicks et al. (2013), using a twin design, found that socialization at age 11 (i.e., a willingness to follow rules and socially conventional values, which was moderately heritable) predicted exposure to risk environments, which predicted substance use at age 17. However, boldness at age 11 (which was highly heritable), directly predicted later substance use, without environmental mediation. It is worth noting that these two studies also illustrate different forms of gene-environment correlation. In the Thomas et al., study, the offspring exposure to parent divorce and discord more likely reflects passive gene-environment correlation. In contrast, in the Hicks et al. (2013) study, the environment included peer contexts, which might be actively sought out differentially by children with different personality risk (an example of active gene-environment correlation). It is also possible that evocative gene-environment correlations create social mediation of genetic risk. For example, Elam et al. (2016) found that childrens' polygenic risk predicted their impulsivity, which predicted later family conflict, which then predicted later substance use. They suggested that impulsive children evoke higher family conflict. These social mediation pathways represent an important contribution of the developmental psychopathology perspective to understanding alcohol use outcomes and they have potentially useful intervention implications. For example, framing parenting interventions to include ways of coping with evocative effects and matching appropriate parenting to particular child styles can reduce parent stigma and self-blame without 'blaming' the child (Reiss & Leve, 2007). In addition, these pathways suggest the usefulness of research studies testing intervention effects on reducing gene-environment covariation (Elam et al., 2023).

Elam et al. (2023) propose a developmental cascade model based on the social mediation of these different forms of gene-environment correlation, in which genetic risk causes children to be exposed to high-risk environments whose effects accrue over time (see Fig. 2). They build on prior work on social mediation of genetic effects by expanding the environments under consideration from typically-studied parent and peer effects to broader neighborhood and cultural effects (emphasizing the importance of cultural genomics, c.f., Causadias & Cicchetti, 2018). They also incorporate the importance of individual differences in reactions to these environments, as a potential point of intervention.

Although the developmental psychopathology perspective has made important contributions by emphasizing these multilevel genetically informed studies, these studies also face challenges. The complex models require careful attention to sample size and ascertainment (Wilson & Rhee, 2022). Replication is challenging not only because of sample size but because there is little standardized measurement. For example, parenting has been measured by a wide variety of scales (which vary across multiple reporters) as well as different types of behavioral observation and their effects may vary by the age, gender, or other characteristics of the child. Similarly, alcohol outcomes that have been studied vary from initiation and initial sips to heavy drinking, regular drinking, and AUD symptoms or diagnosis and the significance and determinants of these different alcohol outcomes vary with development. In addition, because the statistical models that have been tested are themselves complex and often contain multiple covariates, they are difficult to compare across studies. Most challenging for comparing findings, polygenic risk scores are derived for different alcohol-related phenotypes and samples of differing ancestry, ages, and demographics across studies. Finally, it is important to note that these models of gene-environment correlation also need to consider gene-environment interactions and thus that they are not meant to provide a complete model of etiology. For example, the social mediation models reported above emphasize the way that genetic effects influence environmental exposures "outside of the skin." These models are complemented by others that emphasize environmental effects on gene expression (i.e., epigenetic models) or ways that environmental stressors can get 'under the skin' (e.g., Nusslock & Miller, 2016).

https://doi.org/10.1017/S0954579424000671 Published online by Cambridge University Press

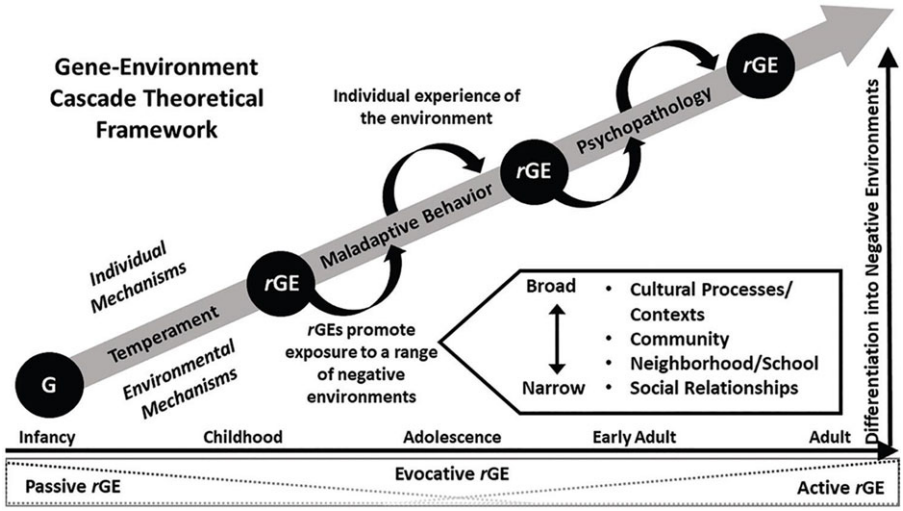

Figure 2. A gene-environment cascade framework (from Elam et al., 2023). Reproduced with permission from the American Psychological Association.

Studying the developmental psychopathology of AUDs in diverse populations

The last decade has seen the continued expansion and interest in incorporating the study of diverse populations within a developmental psychopathology framework (Causadias & Cicchetti, 2018; Ferguson et al., 2023). These studies have included diversity in race/ethnicity, sexual orientation and gender identity, and intersectionality of identities as well as a call for research on diversity of family structures (Pearce et al., 2018). There have also been calls for incorporating diverse populations and cultural considerations into genomic studies (Causadias & Cicchetti, 2018; Elam et al., 2023). A recent review of studies of genetic and environmental influences in diverse populations (Chartier et al., 2017) found more studies of African American and Asian samples compared to Native American or Hispanics and more studies of family and peer influences than broader community or cultural factors. For the developmental psychopathology of AUDs, important contributions of research on diverse populations include identifying diversity in trajectories of alcohol use and the finding that minority individuals (particularly African-Americans) may drink less but experience more alcohol-related problems (see Zapolski et al., 2014 for a review). Zapolski et. al. (2014) suggest that reduced use may be related to cultural and historical protective factors including less integration of alcohol into social functions and greater disapproval of use. However, the increased rates of alcohol-related problems among African-Americans may reflect greater increased surveillance, racial discrimination, use of beverages that are high in alcohol content, and vulnerability to liver disease (Zapolski et al., 2014). In general, research on racial, ethnic, and sexual minority group members has tested whether alcohol use is related to culturally patterned norms and acceptance of alcohol use, acculturation (with patterns of alcohol use increasingly resembling the majority culture over generations and time since immigration, e.g., Chartier et al., 2023; Ruiz et al., 2022) and protective factors such as ethnic identity and cultural values like familism. Studies have also identified alcohol use as a way of coping with discrimination and minority stress. (e.g., Pittman et al., 2019). Finally, studies have incorporated these findings into tests of culturally-tailored intervention programs. For example, a recent meta-analysis of such programs for adolescents (Bo et al., 2023) found significant effects with slightly smaller effects on alcohol use compared to use of other substances and slightly smaller effects for Native Americans (perhaps because none of the programs targeted at Native Americans involved parents). A recent review of interventions for sexual minority adults (Dimova et al., 2022) found reductions in alcohol use although most studies focused on men who have sex with men. Few interventions focused on sexual minority women or transgender individuals. Studies of diverse populations have enhanced our understanding of general and culturally specific risk and protective factors. Useful directions for future research include an increased emphasis on a developmental perspective (Causadias & Cicchetti, 2018) and on intersectionality of identities, acknowledging that studies of intersectionality require large sample sizes.

The significance of childhood as a developmental period

Compared to other developmental periods, there has been somewhat less research focused on childhood risk as a foundation for later alcohol use and alcohol use disorders. It is worth noting that there is a large literature on prenatal alcohol exposure, although this literature has been largely independent of research on early risk for alcohol disorder.

An important finding that boys' behavior at age 3 predicted adult alcohol problems pointed to the significance of early childhood risk. Caspi et al. (1996) found that boys who were either undercontrolled or inhibited at age three were more likely to have alcohol problems compared to other children, even controlling for social class. More recent work from a developmental psychopathology perspective has confirmed the importance of early self-regulation (Robson et al., 2020) and the interplay between early childhood temperament and parenting variables in shaping risk for alcohol outcomes (Eiden et al., 2020; Puttler et al., 2017). Moreover, research suggests that the shared variance between low effortful control and anger reactivity in early childhood is a significant predictor of adolescent negative urgency, which is a facet of impulsivity that consistently predicts negative alcohol outcomes (Waddell et al., 2021). The interplay between parenting and child temperament may create a developmental cascade from childhood to adolescent and adult substance use outcomes. For example, Eiden et al. (2016) found that parent warmth and sensitivity in preschool predicted greater parent monitoring in childhood and early adolescence, which predicted less adolescent affiliation with substance using peers and less alcohol use. Poor self-regulation in preschool led to childhood

https://doi.org/10.1017/S0954579424000671 Published online by Cambridge University Press

externalizing behavior and affiliation with substance using peers, which predicted adolescent drinking.

One important contribution of the developmental psychopathology perspective to understanding early childhood risk has been the study of early adversity. Gee and Cohodes (2023) suggest that corticolimbic circuity and emotion regulation are important mediators of resilience to early adversity and that infancy and early toddlerhood are sensitive periods for caregiver input into those mediators. In particular, secure attachment to caregivers and an environment of safety and predictability support resilience to adversity.

Recent theory (McLaughlin et al., 2014; McLaughlin, 2016) also called for greater clarity about the construct of early adversity and proposed an important distinction between the effects of early deprivation and early threat. Deprivation is a lack of environmental input that reduces learning opportunities, including a lack of consistent routines and structure and (in severe forms), child neglect. Deprivation is thought to alter functioning in frontoparietal regions and impair executive functioning. In turn, impaired executive functioning is associated with poor self-regulation, conduct problems, and substance use. In support of this hypothesis, Johnson et al. (2021) meta-analysis found that early deprivation (compared to threat) was more strongly associated with inhibitory control and working memory. Moreover, Silveira et al. (2020) studied adolescents from the National Consortium on Alcohol and Neurodevelopment (NCANDA) and found that a retrospective measure of child trauma was related to self-reported executive functioning. This relation was mediated by baseline resting state connectivity and executive functioning and functional connectivity prospectively predicted adolescent heavy drinking.

In contrast to deprivation, McLaughlin et al. (2014) conceptualized threat as actual harm or threat of harm to the physical safety of a child, including experiencing or witnessing violence and child abuse. Threat is proposed to disrupt emotion processing and emotion regulation resulting in biased attention to threat, altered neural response to negative emotional cues, and disrupted autonomic nervous system reactivity. These disruptions make it more likely that a child will develop externalizing or internalizing problems, raising risk for later substance use. McLaughlin (2016) also notes that threat and deprivation can co-occur and that vulnerability to these environmental adversities may be moderated by genetic influences and child temperament.

Research advances into the study of early adversity include important work on child maltreatment, which is one of the most severe forms of early adversity and has been established as a risk factor for later substance use disorders. A recent review of this literature from a developmental psychopathology perspective (Cicchetti & Handley, 2019) found robust support for an externalizing pathway to substance use disorders. Although the internalizing pathway had less consistent support, Cicchetti and Handley (2019) suggest that maltreated children may develop disturbed stress responses; that dysregulated HPA axis responses may mediate maltreatment effects on substance use; and that maltreatment increases allostatic load, producing the dysregulation of multiple systems. Cicchetti and Handley also suggest that maltreatment may alter neural brain networks, making maltreated children more vulnerable to contextual risk factors such as neighborhood disadvantage or violence. As with the distinction between effects of deprivation and threat, Cicchetti and Handley note that different types of child maltreatment as well as their timing, chronicity, and severity will likely produce different outcomes. Future research on early childhood risk should address these differential effects as well as the interplay between environmental threat and deprivation and the study of moderators of environmental adversity including greater integration of genetic risk.

The significance of adolescence as a developmental period

An important contribution of the developmental psychopathology perspective is a focus on possible developmental differences in the causes and effects of alcohol use and AUDs. Adolescence is a period of particular significance because alcohol use typically onsets in adolescence and both alcohol use and AUDs typically peak in late adolescence/emerging adulthood (Chassin et al., 2013). Adolescence is a period of increasing importance of peer social interactions, increasing importance of independence from parents, and increased opportunities for peer social interaction in the absence of adult supervision, all of which may contribute to alcohol use initiation and escalation (Chassin et al., 2013). Dual systems theories of adolescence suggest that a combination of puberty-related increases in reward seeking/sensation seeking along with a slower rate of increase in cognitive control create a gap between high levels of sensation seeking and less developed cognitive control making adolescents more likely to engage in rewarding but potentially risky activities such as alcohol use (c.f. Casey, 2015). Although these models remain highly influential, there have also been suggestions for a broadening of focus. For example, a recent review of the role of dopamine in these processes (Franca & Pompeia, 2023) argues that rather than a simple competition between "hot" subcortical reward systems and "cool" prefrontal cognitive control systems, dopamine actually contributes to both reward seeking and cognitive control systems and promotes goal-directed effort (rather than just promoting reward seeking). Franca and Pompeia (2023) note that decisions about "risky" behaviors involve more than reward and control systems but also include processes of evaluating options, appraising risk, anticipating outcomes, and executing actions, and that to varying extents, dopamine is involved in all of these components. This model places provides a somewhat broader view of adolescent decision making with greater emphasis on adolescents as tolerating ambiguity, exploring, and adaptively learning from their experiences.

Studies have also suggested alcohol-specific pathways in which age differences in alcohol effects make adolescence a vulnerable age period for the onset of alcohol use, heavy drinking, and AUDs. Animal data suggest that adolescents are more sensitive to the stimulant and social-facilitation effects of alcohol than are adults but less sensitive to the sedative, aversive, and motor-impairing effects and more likely to show acute tolerance over the course of a drinking session (Spear, 2014). Human data are more sparse and ethical constraints constrain U.S. laboratory studies of alcohol administration for underage drinkers. However, a small number of studies have examined subjective effects among heavy drinking adolescents after a real-world drinking episode. Studying a predominantly college-student sample with an age range from 17 to 32, Treloar et al. (2017) found that younger age was related to greater self-reported stimulation at lower BACs and particularly among heavier drinkers and those with higher AUDIT scores and Miranda et al. (2014) found that adolescent heavy drinkers had greater self-reported sensitivity to stimulant effects than did adult heavy drinkers. Taken together, these studies suggest that alcohol may be particularly rewarding for adolescents and that lowered sensitivity to negative effects, along with greater acute tolerance, may produce not only onset but escalation of heavy drinking,

https://doi.org/10.1017/S0954579424000671 Published online by Cambridge University Press

raising risk for later AUDs. Although differences between adolescent and adult heavy drinkers may be confounded by adults' longer duration of alcohol exposure compared to adolescents, there are some similar findings from rat models in which duration of alcohol exposure is controlled. For example, Spear and Varlinskaya's (2010) review found that adolescent rats were more sensitive to the positive rewarding effects of alcohol and less sensitive to the aversive effects compared to adults.

There has also been considerable progress in understanding the impact of adolescent drinking on brain development and cognitive function (Salmanzadeh et al., 2020). Lees et al (2020) reviewed the longitudinal human literature as well as animal findings in which selection effects, effects of co-occurring substance use, and dosage effects can be controlled. Effects of binge drinking that were identified in both human and animal studies were alterations in gray and white matter and poorer memory, learning, and executive functioning. In longitudinal human data, there have been reported effects of heavy drinking on greater declines in frontal gray matter volume and deterioration in white matter integrity (Tapert & Eberson-Shumante, 2022). Lees et al. (2020) and Tapert and Eberson-Shumante (2022) note that less is known about the effects of recovery from drinking and the small human literature has shown mixed findings. They also note that ongoing large, longitudinal studies such as ABCD (c.f., Luciana et al., 2023) and NCANDA (Brown et al., 2015) will be able to address dosage effects, effects of co-use of other substances, and changes over time in human data.

Finally, animal studies that attempt to model adolescent binge drinking suggest that adolescent binge drinking may produce brain changes associated with persistent, long-lasting effects that encourage drinking escalation and the development of AUDs including effects on increased social anxiety, increased impulsivity and disrupted sleep (Crews et al., 2019). Thus, adolescent drinking exposure itself may set off cascading effects into future developmental periods including "locking in" developmental phenotypes such as impulsivity (Crews et al., 2019). However, although adolescence may be a period of heightened sensitivity to some alcohol effects, it is also important to note that adolescence has been seen as time of increased sensitivity to other environmental effects, including the care-giving environment. This allows not only vulnerability to negative effects such as alcohol exposure but also recovery from early adversity (Colich et al., 2021). The study of these developmental cascades is an important contribution of the developmental psychopathology approach to AUDs.

The significance of middle adulthood as a developmental period

Epidemiological evidence indicates that midlife is a period marked by continued declines in drinking to intoxication, binge drinking and the prevalence of AUD with relatively low levels of new onsets and relatively high levels of chronicity of AUD (Lee et al., 2018; Lee & Sher, 2018). This is likely due, in part, to the effects of greater role constraints on heavy drinking (resulting in fewer new onsets) combined with decreased new role transitions and more gradual changes in trait-like dispositions (e, g,, impulsive personality traits) associated with "maturing out" and desistance. The impact of role transitions can also be seen in cohort effects (for women) such that increased heavy drinking and AUD symptoms in recent female cohorts are related to delayed parenthood (Adams et al., 2023). One factor that does seem to become more important in midlife, as compared to earlier in development is the role of health concerns as a motivating factor in drinking reduction. This ostensible health-related desistance is reduced by the presence of AUD symptoms, presumably because of higher levels of compulsivity underlying drinking. Stress reduction after drinking may also stronger in midlife than in younger adults, suggested in a small ecological momentary assessment study (Peterson-Sockwell et al., 2024). Finally, unemployment may be more strongly predictive of substance use disorder (including AUD) as individuals age at midlife (although substance use disorder may more weakly predict later unemployment (Kendler et al., 2023). Overall, although many of the processes observed during this midlife reflect similar, but muted, processes observed earlier in development e.g., role transitions and personality development), there may also be increased importance of factors such as health, stress reduction, and unemployment.

The significance of older adulthood as a developmental period

Recently, there has been renewed interest in older adulthood (older than 65) as an important developmental stage for alcohol use and AUDs. In part this is because the current "baby boomer" cohort of older adults who reached adolescence in a period of prevalent substance use have carried their high levels of substance use into old age. Moreover, as noted earlier, age-related changes in alcohol metabolism, body fat, and increased prescription medication use make older adults particularly vulnerable to alcohol-related harms, including liver disease and falls (Barry & Blow, 2016). Compared to younger individuals, older adults reach higher BACs and are more sensitive to the sedative and motor-impairing effects of alcohol (Perkins et al., 2019). Recently, there has been increasing interest in the role of alcohol in vascular and Alzheimer's dementias (e.g., Wiegmann et al., 2020).

There may also be age-specific risk factors for the development of AUDs in older adulthood. For example, just as the assumption of adult roles in early adulthood predicts the 'maturing out" of alcohol problems, role loss associated with retirement may predict increased drinking and AUDs. Available evidence, however, suggests a more complicated pattern. Kuerbis and Sacco (2012) reviewed 13 studies and found that pre-retirement conditions and characteristics of retirement (e.g., high job satisfaction, high job stress, and involuntary retirement) were associated with increased drinking after retirement. Emiliussen et al. (2017) reviewed nine studies of heavy drinking or AUD onset after age 50 and concluded that stress, role loss, and peer approval of drinking all increased risk for negative alcohol outcomes. However, retirement or death of a spouse did not. Both reviews note a general lack of research on age-specific risk factors for late-life AUD onset as well as a lack of consistent, clear, operational definitions of constructs. Emiliussen et al. (2017) further call for qualitative studies to identify novel risk factors and intervention targets for this age group. For example, the COVID pandemic served to heighten awareness of social isolation as a potential risk factor, which might be particularly salient in late life. Older adults often have underlying medical and psychological conditions that can motivate drinking including chronic pain and loneliness (Kuerbis & Sacco, 2012) In short, an understanding of age-specific influences on late-life onset, consequences, and recovery from AUDs remains an important future direction for the developmental psychopathology of AUDs Moreover, given the unique circumstance of the "baby boomer' cohort and the relative lack of studies of late-life specific risk factors, it is important for

https://doi.org/10.1017/S0954579424000671 Published online by Cambridge University Press

future research to consider both cohort and age effects on late-life drinking.

Future directions

Effective prevention and treatment of potentially harmful alcohol use and AUDS will require more integrated and developmentally informed, multilevel models given the myriad individual, familial, and broader social factors that determine the course of alcohol involvement. While advances in developmental psychopathology research will undoubtedly continue along multiple fronts, several areas can be targeted as high priority. Based on the considerations discussed above, we can suggest several directions for future research.

1. Encourage multilevel models that allow integrated analyses of developmental cascades that facilitating testing of causal models of key mechanisms.

2. Refine key alcohol concepts and their measurement in a developmentally sensitive manner that accounts for age-related differences in context and that minimize differential item functioning. This will improve the validity of assessments over the life course to better model age-related consequences and the course of alcohol misuse and AUDs. Use of "recommended" assessments across studies and investigators will facilitate harmonization and cross-study integration. Focusing on specific signs/symptoms or symptom clusters/factors rather than global diagnosis will better demonstrate the connections between specific etiological pathways and specific outcomes (e.g., externalizing pathway and symptoms reflecting disinhibition). This is important because both genetically informative studies (e.g., Kendler et al., 2012) and purely phenotypic studies (e.g., Watts et al., 2023) suggest that undifferentiated syndromal approaches can obscure specificity of relations. Conducting longitudinal panel studies and intensive longitudinal studies using symptom network modeling (SNM) approaches will better characterize symptom progression and course (Conlin et al., 2022). Embedding these SNM models in a larger developmental psychopathology framework holds promise for better understanding the nature of AUDs since symptom network structures may differ across key demographic variables, especially age (Huth et al, 2022).

3. Continue use of genetic strategies in order to both identify the role of gene-environment correlations and interactions as well as exploit these strategies as statistical instruments to bolster confidence in identifying various developmental mechanisms and cascades.

4. Recognize the importance of co-occurring and concurrent substance use (e.g., alcohol and cannabis use) and determine the degree to which these are manifestations of similar or different pathways, the transactional effects of substance on the course of the other, and additive and synergistic effects on important behavior and health outcomes (e.g., motor vehicle crashes).

5. Recognize the importance of premorbid and co-occurring psychopathology. Failure to do so precludes the ability to identify alcohol-specific mechanisms and consequences. Although alcohol-focused endophenotypic approaches such as AARDoC/ANA and ETOH frameworks share much with more general endophenotypic approaches such as RDoC, some key processes (e.g., incentive sensitization, habit, opponent-processes) are not well represented in more general approaches and should be considered (Boness et al., 2021).

6. Develop more specific theories that attempt to specify the time-bound functional relationships among study constructs. Most longitudinal panel studies are not designed with the likely temporal dynamics of various cascades in mind with the result that the intervals between measurement occasions may be too long or too short to resolve the mechanisms of interest. In the absence of explicit theory, more frequent assessments (including intensive longitudinal designs with EMA bursts in panel studies), could be useful for identifying key processes.

7. Focus more on childhood precursors, related both to environmental exposures (e.g., adverse childhood experiences), gene-environment interplay, critical periods, and the persistence of related outcomes. Identify early protective traits (e.g., self-regulation) that can be used to monitor the effectiveness of early interventions and identify individuals at enhanced risk.

8. Focus more on late life, recognizing the changing demographic profiles in many countries (e.g., US and China), the legacy of having high levels of drug exposures in youth, and the prevalent risks associated with alcohol and polypharmacy and major health problems and risks associated with older adulthood (e.g., pain, mild cognitive impairment, and dementia).

9. Increase the diversity of samples both within a given culture (e.g., with respect to social class, race/ethnicity, sexuality minority representation, and intersectionality of identities) as well in multiple countries that differ with respect to their degree of industrialization, economic health, secularization/religious beliefs in order both to broaden knowledge and help distinguish universal mechanisms and problems from those that are more culture-bound; both are important.

10. Increase use of passive assessments e.g., software that monitors the type and duration of different smartphone and tablet applications, smart watches that can be used to measure physical activity, sleep (onset, duration, quality). Similarly, archival and administrative data could be used both to measure social contexts (e.g., racial and socioeconomic profiles of neighborhood using census data) and outcomes (e.g., motor vehicle records, electronic health records).

11. Better exploit existing data with emerging statistical approaches that allow stronger causal inferences (e.g., modern causal inference approaches including use of instrumental variables) or models that are potentially more sensitive to certain developmental processes where the influence of one variable on another is cumulative, and not instantaneous (e.g., diffusion models).

Importantly, addressing these future directions requires multiple approaches including animal models and human data. Extremely valuable existing resources are the large ongoing longitudinal studies whose data are in the public domain (e.g., ABCD, NCANDA, COGA). These rich, multilevel studies have large sample sizes and ranges of assessments beyond the resources of individual investigators and will likely lay the groundwork for the next generation of studies, which should also include smaller, targeted projects and integrative data analyses of multiple studies.

Funding statement. None.

Competing interests. None.

References

Adams, R. S., McKetta, S. C., Jager, J., Stewart, M. T., & Keyes, K. M. (2023). Cohort effects of women's mid-life binge drinking and alcohol use disorder symptoms in the United States: Impacts of changes in timing of parenthood. *Addiction*, *118*(10), 1932–1941.

Ahammer, A., Bauernschuster, S., Halla, M., & Lachenmaier, H. (2022). Minimum legal drinking age and the social gradient in binge drinking. *Journal of Health Economics*, *81*, 102571. https://doi.org/10.1016/j.jhealeco.2021.102571

https://doi.org/10.1017/S0954579424000671 Published online by Cambridge University Press

American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Author.

Anker, J. J., Forbes, M. K., Almquist, Z. W., Menk, J. S., Thuras, P., Unruh, A. S., & Kushner, M. G. (2017). A network approach to modeling comorbid internalizing and alcohol use disorders. *Journal of Abnormal Psychology*, *126*(3), 325–339. https://doi.org/10.1037/abn0000257

Barry, K. L., & Blow, F. C. (2016). Drinking over the lifespan: Focus on older adults. *Alcohol Research: Current Reviews*, *38*(1), 115–120.

Bergmann, M. M., Rehm, J., Klipstein-Grobusch, K., Boeing, H., Schütze, M., Drogan, D., & Ferrari, P. (2013). The association of pattern of lifetime alcohol use and cause of death in the european prospective investigation into cancer and nutrition (EPIC) study. *International Journal of Epidemiology*, *42*(6), 1772–1790.

Bickel, W. K., Crabbe, J. C., & Sher, K. J. (2019). What is addiction? How can animal and human research be used to advance research, diagnosis, and treatment of alcohol and other substance use disorders? *Alcoholism: Clinical and Experimental Research*, *43*(1), 6–21.

Bo, A. I., Goings, C., Evans, C., Sharma, A., Jennings, Z., Durand, B., Bardeen, A., & Murray-Lichtman, A. (2023). Culturally sensitive prevention programs for substance use among adolescents of color: A systematic review and meta-analysis of randomized controlled trials. *Clinical Psychology Review*, *99*, 102233. https://doi.org/10.1016/j.cpr.2022.102233

Boness, C. L., Votaw, V. R., Francis, M. W., Watts, A. L., Sperry, S. H., Kleva, C. S., Nellis, L., McDowell, Y., Douaihy, A. B., Sher, K. J., & Witkiewitz, K. (2023). Alcohol use disorder conceptualizations and diagnoses reflect their sociopolitical context. *Addiction Research & Theory*, *31*(5), 307–312.

Boness, C. L., Watts, A. L., Moeller, K. N., & Sher, K. J. (2021). The etiologic, theory-based, ontogenetic hierarchical framework of alcohol use disorder: A translational systematic review of reviews. *Psychological Bulletin*, *147*(10), 1075–1123. https://doi.org/10.1037/bul0000333s

Brown, S. A., Brumback, T., Tomlinson, K., Cummins, K., Thompson, W. K., Nagel, B. J., De Bellis, M. D., Hooper, S. R., Clark, D. B., Chung, T., Hasler, B. P., Colrain, I. M., Baker, F. C., Prouty, D., Pfefferbaum, A., Sullivan, E. V., Pohl, K. M., Rohlfing, T., Nichols, B. N. . . . Tapert, S. F. (2015). The national consortium on alcohol and neuroDevelopment in adolescence (NCANDA): A multisite study of adolescent development and substance use. *Journal of Studies On Alcohol and Drugs*, *76*(6), 895–908. https://doi.org/10.15288/jsad.2015.76.895.PMCID:

Bryazka, D., Reitsma, M. B., Griswold, M. G., Abate, K. H., Abbafati, C., Abbasi-Kangevari, M., Abbasi-Kangevari, Z., Abdoli, A., Abdollahi, M., Abdullah, A. Y. M., Abhilash, E. S., Abu-Gharbieh, E., Acuna, J. M., Addolorato, G., Adebayo, O. M., Adekanmbi, V., Adhikari, K., Adhikari, S., Adnani, Q. E. S. . . . Gakidou, E. (2022). Population-level risks of alcohol consumption by amount, geography, age, sex, and year: A systematic analysis for the global burden of disease study 2020. *The Lancet*, *400*(10347), 185–235. https://doi.org/10.1016/S0140-6736(22)00847-9

Casey, B. J. (2015). Beyond simple models of self-control to circuit-based accounts of adolescent behavior. *Annual Review of Psychology*, *66*(1), 295–319. https://doi.org/10.1146/annurev-psych-010814-015156

Caspi, A., Moffitt, T. E., Newman, D. L., & Silva, P. A. (1996). Behavioral observations at age 3 years predict adult psychiatric disorders. Longitudinal evidence from a birth cohort. *Archives of General Psychiatry*, *53*(11), 1033–1039. https://doi.org/10.1001/archpsyc.1996.01830110071009

Causadias, J. M., & Cicchetti, D. (2018). Cultural development and psychopathology. *Development and Psychopathology*, *30*(5), 1549–1555. https://doi.org/10.1017/S095457941800220

Chartier, K. G., Karriker-Jaffe, K. J., Cummings, C. R., & Kendler, K. S. (2017). Review: Environmental influences on alcohol use: Informing research on the joint effects of genes and the environment in diverse U.S. populations. *American Journal of the Addictions*, *26*(5), 446–460. https://doi.org/10.1111/ajad.12478

Chartier, K. G., Kendler, K. S., Ohlsson, H., Sundquist, K., & Sundquist, J. (2023). Triangulation of evidence on immigration and rates of alcohol use disorder in Sweden: Evidence of acculturation effects. *Alcoholism: Clinical and Experimental Research*, *47*(1), 104–115, https://doi.org/10.1111/acer.14969

Chassin, L., Sher, K. J., Hussong, A., & Curran, P. (2013). The developmental psychopathology of alcohol use and alcohol disorders: Research achievements and future directions. *Development and Psychopathology*, *25*(4 Pt 2), 1567–1584. https://doi.org/10.1017/S0954579413000771

Chung, T., & Martin, C. S. (2005). What were they thinking?: Adolescents' interpretations of DSM-IV alcohol dependence symptom queries and implications for diagnostic validity. *Drug and Alcohol Dependence*, *80*(2), 191–200.

Cicchetti, D. (1984). The emergence of developmental psychopathology. *Child Development*, *55*(1), 1–7. https://www.jstor.org/stable/1129830

Cichetti, D., & Handley, E. D. 2019). *Childhood maltreatment and the development of substance use and disorder*. Neurobiology of Stress.

Colich, N. L., Sheridan, M. A., Humphreys, K. L., Wade, M., Tibu, F., Nelson, C. A., Zeanah, C. H., Fox, N. A., & McLaughlin, K. A. (2021). Heightened sensitivity to the caregiving environment during adolescence: Implications for recovery following early-life adversity. *Journal of Child Psychology and Psychiatry*, *62*(8), 937–948. https://doi.org/10.1111/jcpp.13347

Conlin, W. E., Hoffman, M., Steinley, D., & Sher, K. J. (2022). Cross-sectional and longitudinal AUD symptom networks: They tell different stories. *Addictive Behaviors*, *131*, 107333.

Conradt, E., Crowell, S. E., & Cicchetti, D. (2021). Using development and psychopathology principles to inform the Research Domain Criteria (RDoC) framework. *Development and Psychopathology*, *33*(5), 1521–1525.

Crews, F. T., Robinson, D. L., Chandler, L. J., Ehlers, C. L., Mulholland, P. J., Pandey, S. C., Rodd, Z. A., Spear, L. P., Swartzwelder, H. S., & Vetreno, R. P. (2019). Mechanisms of persistent neurobiological changes following adolescent alcohol exposure: NADIA consortium findings. *Alcoholism: Clinical and Experimental Research*, *43*(9), 1806–1922. https://doi.org/10.1111/acer.14154

Dietary Guidelines Advisory Committee (2020). Scientific report of the 2020-2025 Dietary guidelines advisory committee: Advisory report to the secretary of agriculture and the secretary of health and human services. *Agricultural Research Service*, *10*, 49.

Dimova, E. D., Elliott, L., Frankis, J., Drabble, L., Wiencierz, S., & Emslie, C. (2022). Alcohol interventions for LGBTQ+ adults: A systematic review. *Drug and Alcohol Review*, *41*(1), 43–53. https://doi.org/10.1111/dar.13358

Donovan, J. E. (2009). Estimated blood alcohol concentrations for child and adolescent drinking and their implications for screening instruments. *Pediatrics*, *123*(6), e975–e981.

Donovan, J. E., & Molina, B. S. (2008). Children's introduction to alcohol use: Sips and tastes. *Alcoholism: Clinical and Experimental Research*, *32*(1), 108–119.

Eiden, R. D., Godleski, S. A., Colder, C. R., Livingston, J. A., Leising, M. C., & Leonard, K. E. (2020). Early childhood risk and protective factors predicting resilience against adolescent substance use. *Adversity and Resilience Science*, *1*(2), 107–119. https://doi.org/10.1007/s42844-020-00007-5

Eiden, R. D., Lessard, J., Colder, C. R., Livingston, J., Casey, M., & Leonard, K. E. (2016). Developmental cascade model for adolescent substance use from infancy to late adolescence. *Developmental Psychology*, *52*(10), 1619–1633. https://doi.org/10.1037/dev0000199

Elam, K. K., Lemery-Chalfant, K., & Chassin, L. (2023). A gene-environment cascade theoretical framework of developmental psychopathology. *Journal of Psychopathology and Clinical Science*, *132*(3), 287–296. https://doi.org/10.1037/abn0000546

Elam, K. K., Wang, F. L., Bountress, K., Chassin, L., Pandika, D., & Lemery-Chalfant, L. (2016). Predicting substance use in emerging adulthood: A genetically informed study of developmental transactions between impulsivity and family conflict. *Development and Psychopathology*, *28*(3), 673–688. https://doi.org/10.1017/S0954579416000249

Emiliussen, J., Nielsen, A., & Andersen, K. (2017). Identifying risk factors for late-onset (50+) alcohol use disorder and heavy drinking: A systematic review. *Substance Use and Misuse*, *52*(12), 1575–1588. https://doi.org/10.1080/10826084.2017.1293102

Ferguson, G. M./, Causadias, J. M., & Simenec, T. S. (2023). Acculturation and psychopathology. *Annual Review of Clinical Psychology*, *19*(1), 381–411. https://doi.org/10.1146/annurev-clinpsy-080921-080622

Franca, T. A., & Pompeia, S. (2023). Reappraising the role of dopamine in adolescent risk-taking behavior. *Neuroscience and Biobehavioral Reviews*, *147*, 105085. https://doi.org/10.1016/j.neubiorev.2023.105085

Gee, D. G., & Cohodes, E. M. (2023). Leveraging the developmental neuroscience of caregiving to promote resilience among youth exposed to adversity. *Development and Psychopathology*, *35*(5), 1–18. https://doi.org/10.1017/S0954579423001128

Grant, B. F., Goldstein, R. B.; B., Saha, T. D., Chou, S. P., Jung, J., Zhang, H., Pickering, R. P., Ruan, W. J., Smith, S. M., Huang, B., & Hasin, D. S. (2015). Epidemiology of DSM-5 alcohol use disorder: Results from the national epidemiologic survey on alcohol and related conditions III. *JAMA Psychiatry*, *72*(8), 757–766.

Gresko, S. A., Rieselbach, M., Corley, R. P., Reynolds, C. A., & Rhee, S. H. (2022). Associations between parenting characteristics and adolescence substance use: A genetically informed, longitudinal adoption study. *Development and Psychopathology*, *34*(5), 1702–1715. https://doi.org/10.1017/S0954579422000748

Grube, J. (2005). *Youth drinking rates and problems: A comparison of European countries and the United States*. Pacific Institute for Research and Evaluation.

Grucza, R. A., Sher, K. J., Kerr, W. C., Krauss, M. J., Lui, C. K., McDowell, Y. E., Hartz, S., Virdi, G., & Bierut, L. J. (2018). Trends in adult alcohol use and binge drinking in the early 21st-century United States: A meta-analysis of 6 national survey series. *Alcoholism: Clinical and Experimental Research*, *42*(10), 1939–1950.

Haeny, A. M., Littlefield, A. K., & Sher, K. J. (2014). Repeated diagnoses of lifetime alcohol use disorders in a prospective study: Insights into the extent and nature of the reliability and validity problem. *Alcoholism: Clinical and Experimental Research*, *38*(2), 489–500.

Hanson, D. (2013). Historical evolution of alcohol consumption in society. In P. Boyle, P. Boffetta, A. B. Lowenfels, H. Burns, O. Brawley, W. Zatonski, & J. Rehm (Ed.), *Alcohol: Science, Policy and Public Health* (pp. 3–12). Oxford University Press. https://doi.org/10.1093/acprof:oso/9780199655786.001.0001

Harford, T. C., Yi, H. Y., Faden, V. B., & Chen, C. M. (2009). The dimensionality of DSM-IV alcohol use disorders among adolescent and adult drinkers and symptom patterns by age, gender, and race/ethnicity. *Alcoholism: Clinical and Experimental Research*, *33*(5), 868–878.

Hasin, D. S., O'Brien, C. P., Auriacombe, M., Borges, G., Bucholz, K., Budney, A., Compton, W. M., Crowley, T., Ling, W., Petry, N. M., Schuckit, M., & Grant, B. F. (2013). DSM-5 criteria for substance use disorders: Recommendations and rationale. *American Journal of Psychiatry*, *170*(8), 834–851.

Helle, A. C., Trull, T. J., Watts, A. L., McDowell, Y., & Sher, K. J. (2020). Psychiatric comorbidity as a function of severity: DSM-5 alcohol use disorder and hiTOP classification of mental disorders. *Alcoholism: Clinical and Experimental Research*, *44*(3), 632–644.

Hicks., B. M., Johnson, W., Durban., C. E., Blonigen., D. M., Iacono., W. G., & McGue, M. (2013). Gene-environment correlation in the development of adolescent substance abuse: Selection effects of child personality and mediation via contextual risk factors. *Development and Psychopathology*, *25*(1), 119–132. https://doi.org/10.1017/S0954579412000946

Huth, K. B. S., Luigjes, J., Marsman, M., Goudriaan, A. E., & van Holst, R. J. (2022). Modeling alcohol use disorder as a set of interconnected symptoms-assessing differences between clinical and population samples and across external factors. *Addictive Behaviors*, *125*, 107128.

Jackson, K. M., Colby, S. M., Barnett, N. P., & Abar, C. C. (2015). Prevalence and correlates of sipping alcohol in a prospective middle school sample. *Psychology of Addictive Behaviors*, *29*(3), 766–778. https://doi.org/10.1037/adb0000072

Johnson, D., Policelli, J., Li, M., Dharamsi, A., Hu, Q., Sheridan, M. A., McLaughlin, K. A., & Wade, M. (2021). Associations of early-life threat and deprivation with executive functioning in childhood and adolescence: A systematic review and meta-analysis. *Journal of the American Medical Association: Pediatrics*, *175*(11), e212511. https://doi.org/10.1001/jamapediatrics.2021.2511 PMID: 34309651 PMCID: PMC8314173.

Jones, A. W. (2019). Alcohol, its absorption, distribution, metabolism, and excretion in the body and pharmacokinetic calculations. *Wiley Interdisciplinary Reviews: Forensic Science*, *1*(5), e1340.

Karriker-Jaffe, K. J., Witbrodt, J., & Greenfield, T. K. (2015). Refining measures of alcohol problems for general population surveys. *Alcoholism: Clinical and Experimental Research*, *39*(2), 363–370.

Kendler, K. S., Aggen, S. H., Prescott, C. A., Crabbe, J., & Neale, M. C. (2012). Evidence for multiple genetic factors underlying the DSM-IV criteria for alcohol dependence. *Molecular Psychiatry*, *17*(12), 1306–1315.

Kendler, K. S., Ohlsson, H., Sundquist, J., & Sundquist, K. (2023). Cross-lagged analyses across middle-adulthood of the bidirectional associations between substance use disorders and psychosocial dysfunction. *Journal of Studies On Alcohol and Drugs*, *84*(2), 185–197.

Kotov, R., Krueger, R. F., Watson, D., Achenbach, T. M., Althoff, R. R., Bagby, R. M., Brown, T. A., Carpenter, W. T., Caspi, A., Clark, L. A., Eaton, N. R., Forbes, M. K., Forbush, K. T., Goldberg, D., Hasin, D., Hyman, S. E., Ivanova, M. Y., Lynam, D. R., Markon, K. … Zimmerman, M. (2017). The hierarchical taxonomy of psychopathology (HiTOP): A dimensional alternative to traditional nosologies. *Journal of Abnormal Psychology*, *126*(4), 454–477. https://doi.org/10.1037/abn0000258

Kuerbis, A., & Sacco, P. (2012). The impact of retirement on the drinking patterns of older adults: A review. *Addictive Behaviors, May*, *37*(5), 587–595. https://doi.org/10.1016/j.addbeh.2012.01.022

Kwako, L. E., Momenan, R., Litten, R. Z., Koob, G. F., & Goldman, D. (2016). Addictions neuroclinical assessment: A neuroscience-based framework for addictive disorders. *Biological Psychiatry*, *80*(3), 179–189.

Lane, S. P., & Sher, K. J. (2015). Limits of current approaches to diagnosis severity based on criterion counts: An example with DSM-5 alcohol use disorder. *Clinical Psychological Science*, *3*(6), 819–835. https://doi.org/10.1177/2167702614553026

Lane, S. P., Steinley, D., & Sher, K. J. (2016). Meta-analysis of DSM alcohol use disorder criteria severities: Structural consistency is only 'skin deep'. *Psychological Medicine*, *46*(8), 1769–1784.

Lee, M. R., Boness, C. L., McDowell, Y. E., Vergés, A., Steinley, D. L., & Sher, K. J. (2018). Desistance and severity of alcohol use disorder: A lifespan-developmental investigation. *Clinical Psychological Science*, *6*(1), 90–105. https://doi.org/10.1177/2167702617736

Lee, M. R., Boness, C. L., McDowell, Y. E., Vergés, A., Steinley, D. L., & Sher, K. J. (2018). Desistance and severity of alcohol use disorder: A lifespan-developmental investigation. *Clinical Psychological Science*, *6*(1), 90–105.

Lee, M. R., MacLean, M. G., Stephenson, A., Kady, A., Kwan, T., Bowlby, D., … & Sher, K. J. (2023). Age specificity of effects of health problems on drinking reduction: A lifespan developmental analysis. *Prevention Science*, *24*(5), 887–900.

Lee, M. R., & Sher, K. J. (2018). Maturing out, of binge and problem drinking. *Alcohol Research: Current Reviews*, *39*(1), 31–42.

Lees, B., Meredith, L. R., Kirkland, A. E., Bryant, B. E., & Squeglia, L. M. (2020). Effect of alcohol use on the adolescent brain and behavior. *Pharmacology, Biochemistry and Behavior*, *192*, 172906. https://doi.org/10.1016/j.pbb.2020.172906

Llamosas-Falcón, L., Hasan, O. S., Shuper, P. A., & Rehm, J. (2023). Alcohol use as a risk factor for sexually transmitted infections: A systematic review and conclusions for prevention. *International Journal of Alcohol and Drug Research*, *11*(1), 3–12.

Luciana, M., Barch, D., & Hering, M. M. (2023). Adolescent brain cognitive development study: Longitudinal methods, developmental findings, and associations with environmental risk factors. *Developmental Cognitive Neuroscience*, *64*, 101311. https://doi.org/10.1016/j.dcn.2023.101311

Luukkonen, J., Tarkiainen, L., Martikainen, P., & Remes, H. (2023). Minimum legal drinking age and alcohol-attributable morbidity and mortality by age 63 years: A register-based cohort study based on alcohol reform. *The Lancet Public Health*, *8*(5), e339–e346.

Marmet, S., Studer, J., Bertholet, N., Grazioli, V. S., Daeppen, J. B., & Gmel, G. (2019). Interpretation of DSM-5 alcohol use disorder criteria in self-report surveys may change with age. A longitudinal analysis of young swiss men. *Addiction Research & Theory*, *27*(6), 489–497.

McLaughlin, K. A. (2016). Future directions in childhood adversity and youth psychopathology. *Journal of Clinical Child and Adolescent Psychology*, *45*(3), 361–381. https://doi.org/10.1080/15374416.2015.1110823

McLaughlin, K. A., Sheridan, M. A., & Lambert, H. K. (2014). Childhood adversity and neural development: Deprivation and threat as distinct

dimensions of early experience. *Neuroscience and Biobehavioral Reviews*, *47*, 578–591. https://doi.org/10.1016/j.neubiorev.2014.10.012

Miller, A. P., Kuo, S. I..-C., Johnson, E. C., Tillman, R., Brislin, S. J., Dick, D. M., Kamarajan, C., Kinreich, S., Kramer, J., McCutcheon, V. V., Plawecki, M. H., Porjesz, B., Schuckit, M. A., Salvatore, J. E., Edenberg, H. J., Bucholz, K. K., Meyers, J. L., Agrawal, A., Foroud, T. . . . Parsian, A. (2023). & collaborative study on the genetics of alcoholism, 2023, diagnostic criteria for identifying individuals at high risk of progression from mild or moderate to severe alcohol use disorder. *JAMA Network Open*, *6*(10), e2337192–e2337192.

Miranda, R., Monti, P. M., Ray, L., Treloar, H. R., Reynolds, E. K., Ramirez, J., Chun, T., Gwaltney, C., Justus, A., Tidey, J., Blanchard, A., & Magill, M. (2014). Characterizing subjective responses to alcohol among adolescent problem drinkers. *Journal of Abnormal Psychology, Feb*, *123*(1), 117–129. https://doi.org/10.101037/a0035328

National Institute on Alcohol Abuse and Alcoholism (NIAAA), 2016. Drinking Levels Defined. https://www.niaaa.nih.gov/alcohol-health/overview-alcohol-consumption/moderate-binge-drinking. Accessed 28 November 2016.

Nusslock, R., & Miller, G. E. (2016). Early-life adversity and physical and emotional health across the lifespan: A neuroimmune network hypothesis. *Biological Psychiatry*, *80*(1), 23–32. https://doi.org/10.1016/j.biopsych.2015.05.017

O'Neill, S. E., & Sher, K. J. (2000). Physiological alcohol dependence symptoms in early adulthood: A longitudinal perspective. *Experimental and Clinical Psychopharmacology*, *8*(4), 493–508.

Paradis, C., Butt, P., Shield, K., Poole, N., Wells, S., Naimi, T., & Sherk, A. (2023). *The low-risk alcohol drinking guidelines scientific expert panels, 2023, Canada's guidance on alcohol and health: Final report*. Canadian Centre on Substance Use and Addiction.

Pasman, J. A., Smit, K., Vollebergh, W. A. M., Nolte, I. M., Hartman, C. A., Abdellaoul, A., Verweij, K. J. H., Maciejewski, D., & Vink, J. M. (2023). Interplay between genetic risk and the parent environment in adolescence and substance use in young adulthood: A TRAILS study. *Development and Psychopathology*, *35*(1), 396–409. https://doi.org/10.1017/s0954579421000811x

Patrick, M. E., & Azar, B. (2018). High-intensity drinking. *Alcohol Research*, *39*(1), 49–55.

Patrick, M. E., Schulenberg, J. E., Miech, R. A., Johnston, L. D., O'Malley, P. M., & Bachman, J. G. (2022). *Monitoring the future panel study annual report: National data on substance use among adults Ages 19 to 60, 1976-2021*. University of Michigan Institute for Social Research.

Patrick, M. E., Schulenberg, J. E., Miech, R. A., Johnston, L. D., O'Malley, P. M., & Bachman, J. G. (2022). *Monitoring the future panel study annual report: National data on substance use among adults ages 19 to 60, 1976-2021. Monitoring the future monograph series*. University of Michigan Institute for Social Research.

Patrick, M. E., Terry-McElrath, Y. M., Lanza, S. T., Jager, J., Schulenberg, J. E., & O'Malley, P. M. (2019). Shifting age of peak binge drinking prevalence: Historical changes in normative trajectories among young adults aged 18 to 30. *Alcoholism: Clinical and Experimental Research*, *43*(2), 287–298.

Pearce, L. D., Hayward, G. M., Chassin, L., & Curran, P. J. (2018). The increasing diversity and complexity of family structures for adolescents. *Journal of Research On Adolescence*, *28*(3), 591–608. https://doi.org/10.1111/jora.12391

Perkins, A. E., Varlinskaya, E. I., & Deak, T. (2019). From adolescence to late aging: A comprehensive review of social behavior, alcohol, and neuroinflammation across the lifespan. *International Review of Neurobiology*, *148*, 231–303. https://doi.org/10.1016/bs.irn.2019.08.001

Peterson-Sockwell, H., Rejeski, W. J., Fanning, J., Porges, S. W., Heilman, K. J., Laurienti, P. J., & Gauvin, L. (2024). Differential momentary reports of stress and affect associated with alcohol consumption in middle-aged versus younger adults. *Substance Use and Misuse*, *58*(5), 666–675.

Pittman, D. M., Brooks, J. J., Kaura, P., & Obasic, E. M. (2019). The cost of minority stress: Risky alcohol use and coping motivated drinking behavior in African American college students. *Journal of Ethnicity and Substance Abuse*, *18*(2), 257–278. https://doi.org/10.1080/15332640.2017.1336958

Puttler, L. I., Fitzgerald, H. E., Heitzeg, M. M., & Zucker, R. A. (2017). Boys, early risk factors for alcohol problems and the development of the self: An interconnected matrix. *Infant Mental Health Journal*, *38*(1), 83–96. https://doi.org/10.1002/imhj.21618

Radaev, V., & Roshchina, Y. (2019). Young cohorts of Russians drink less: Age-period–cohort modelling of alcohol use prevalence 1994-2016. *Addiction*, *114*(5), 823–835.

Rehm, J., Rovira, P., Llamosas-Falcón, L., & Shield, K. D. (2021). Dose–response relationships between Levels of alcohol use and risks of mortality or disease for all People, and for different ages, by sex, and for groups defined by risk factors. *Nutrients*, *13*, 2652. https://doi.org/10.3390/nu13082652

Rehm, J. C.;rgen, Allamani, A., Elekes, Z., Jakubczyk, A., Manthey, J., Probst, C., Struzzo, P., Della Vedova, R., Gual, A., & Wojnar, M. (2015). Alcohol dependence and treatment utilization in Europe – a representative cross-sectional study in primary care. *BMC Family Pract*, *16*(1), 90. https://doi.org/10.1186/s12875-015-0308-8

Reiss, D., & Leve, L. D. (2007). Genetic expression outside the skin: Clues to mechanisms of genotype by environment interaction. *Development and Psychopathology*, *19*(4), 1005–1027. https://doi.org/10.1017/S0954579407000508

Robson, D. A., Allen, M. S., & Howard, S. J. (2020). Self-regulation in childhood as a predictor of future outcomes: A meta-analytic review. *Psychological Bulletin*, *146*(4), 324–354. https://doi.org/10.1037/bul0000227

Ruiz, M., Johnson, A., & Campbell, L. (2022). Acculturation and drinking behavior among Latinxs: A narrative review. *Journal of Ethnicity and Substance Use*, *30;1-37*(1), 21–57. https://doi.org/10.1080/15332640.2022.2080144

Salmanzadeh, H., Ahmadi-Soleimani, S. M., Pachenari, N., Azadi, M., Halliwell, R. F., Rubino, T., & Azizi, H. (2020). Adolescent drug exposure: A review of evidence for the development of persistent changes in brain function. *Brain Research Bulletin*, *156*, 105–117.

Shield, K., Paradis, C., Butt, P., Naimi, T., Sherk, A., Asbridge, M., Myran, D., Stockwell, T., Wells, S., Poole, N., Heatley, J., Hobin, E., Thompson, K., & Young, M. (2024). New perspectives on how to formulate alcohol drinking guidelines. *Addiction*, *119*(1), 9–19. https://doi.org/10.1111/add.16316

Silveira, S., Shah, R., Nooner, K. B., Nagel, B., Tapert, S., de Bellis, M., & Mishra, J. (2020). Impact of childhood trauma on executive function in adolescence-mediating functional brain networks and prediction of high-risk drinking. *Biological Psychiatry, Cognitive Neuroscience, and Neuroimaging*, *5*(5), 499–509. https://doi.org/10.1016/j.bpsc.2020.01.011.PMID: 32299789 PMCID: PMC8366521.

Sohi, I., Chrystoja, B. R., Rehm, J., Wells, S., Monteiro, M., Ali, S., & Shield, K. D. (2022). Changes in alcohol use during the COVID-19 pandemic and previous pandemics: A systematic review. *Alcoholism: Clinical and Experimental Research*, *46*(4), 498–513.

Spear, L. P. (2014). Adolescents and alcohol: Acute sensitivities, enhanced intake, and later consequences. *Neurotoxicology and Teratology*, *0*, 51–59. https://doi.org/10.1016/j.ntt.2013.11.006

Spear, L. P., & Varlinskaya, E. I. (2010). Sensitivity to ethanol and other hedonic stimuli in an animal model of adolescence: Implications for prevention science? *Developmental Psychobiology*, *52*(3), 236–243. https://doi.org/10.1002/dev.20457

Stephenson, M., Aliev, F., Kuo, S. I..-C., Edwards, A. C., Pandey, G., Su, J., Kamarajan, C., Dick, D., & Salvatore, J. E. (2022). The role of adolescent social relationships in promotion alcohol resistance: Interrupting the intergenerational transmission of alcohol use. *Development and Psychopathology*, *34*(5), 1841–1855. https://doi.org/10.1017/S0954579422000785

Stockwell, T., Zhao, J., Panwar, S., Roemer, A., Naimi, T., & Chikritzhs, T. (2016). Do "moderate" drinkers have reduced mortality risk? A systematic review and meta-analysis of alcohol consumption and all-cause mortality. *Journal of Studies on Alcohol and Drugs*, *77*(2), 185–198

Tapert, S. F., & Eberson-Shumante, S. (2022). Alcohol and the adolescent brain: What we've learned and where the data are taking us. *Alcohol Research Current Reviews*, *42*(1), 1–9. https://doi.org/10.35946/arcr.v42.1.07

Taylor, B., & Rehm, J. (2012). The relationship between alcohol consumption and fatal motor vehicle injury: High risk at low alcohol levels. *Alcoholism: Clinical and Experimental Research*, *36*(10), 1827–1834.

Taylor, N., Callinan, S., Pennay, A., & Livingston, M. (2024). Have the personality and socio-demographic profiles of Australian adolescent drinkers changed? *Drug and Alcohol Review*, *43*(3), 604–615. https://doi.org/10.1111/dar.13793

Thomas, N. S., Salvatore, J. E., Kuo, S. I., McCutcheon, A. F., Mayers, V. V., Bucholz, J. M., Brislin, K. K., Chan, S. J., Edenberg, G., Kamarajan, H., Kramer J. R., C., Kuperman., S., Pandey., G., Plawecki, M. H., Schuckit, M. A., Dick, D. M., & **COGA Collaborators** (2023). Genetic nurture effects for alcohol use disorder. *Journal of Molecular Psychiatry*, *28*, 759–766. https://doi.org/10.1038/s41380-022-01816-z

Treloar, H., Celio, M. A., Lisman, S. A., Miranda, R., & Spear, L. P. (2017). Subjective alcohol responses in a cross-sectional, field-based study of adolescents and young adults: Effects of age, drinking level, and dependence/consequences. *Drug and Alcohol Dependence*, *170*, 156–163. https://doi.org/10.1016/j.drugalcdep.2016.11.009

Vasilenko, S. A., Evans-Polce, R. J., & Lanza, S. T. (2017). Age trends in rates of substance use disorders across ages 18-90: Differences by gender and race/ethnicity. *Drug and Alcohol Dependence*, *180*, 260–264.

Vergés, A., Ellingson, J. M., Schroder, S. A., Slutske, W. S., & Sher, K. J. (2018). Intensity of daily drinking and its relation to alcohol use disorders. *Alcoholism: Clinical and Experimental Research*, *42*(9), 1674–1683.

Vergés, A., Jackson, K. M., Bucholz, K. K., Grant, J. D., Trull, T. J., Wood, P. K., & Sher, K. J. (2012). Deconstructing the age-prevalence curve of alcohol dependence: Why "maturing out" is only a small piece of the puzzle. *Journal of Abnormal Psychology*, *121*(2), 511–523. https://doi.org/10.1037/a0026027

Vergés, A., Lee, M. R., Martin, C. S., Trull, T. J., Martens, M. P., Wood, P. K., & Sher, K. J. (2021). Not all symptoms of alcohol dependence are developmentally equivalent: Implications for the false-positives problem. *Psychology of Addictive Behaviors*, *35*(4), 444–457. https://doi.org/10.1037/adb0000723

Vickers-Smith, R., Justice, A. C., Becker, W. C., Rentsch, C. T., Curtis, B., Fernander, A., Hartwell, E. E., Ighodaro, E. T., Kember, R. L., Tate, J., & Kranzler, H. R. (2023). Racial and ethnic bias in the diagnosis of alcohol use disorder in veterans. *American Journal of Psychiatry*, *180*(6), 426–436.

Vize, C. E., & Lane, S. P. (2022). Reliability of differential item functioning in alcohol use disorder: Bayesian meta-analysis of criteria discrimination estimates. *Assessment*, *29*(5), 925–939.

Waddell, J. T., Sternberg, A., Bui, L., Ruof, A., Blake, A., Grimm, K., Elam, K. K., Eisenberg, N., & Chassin, L. (2021). Relations between child temperament and adolescent negative urgency in a high-risk sample. *Journal of Research On Personality*, *90*, 104056. https://doi.org/10.1016/j.jrp.2020.104056

Wall, T. L., Luczak, S. E., & Hiller-Sturmhöfel, S. (2016). Biology, genetics, and environment: Underlying factors influencing alcohol metabolism. *Alcohol Research: Current Reviews*, *38*(1), 59–68.

Watts, A. L., Boness, C. L., Loeffelman, J. E., Steinley, D., & Sher, K. J. (2021). Does crude measurement contribute to observed unidimensionality of psychological constructs? A demonstration with DSM-5 alcohol use disorder. *Journal of Abnormal Psychology*, *130*(5), 512–524.

Watts, A. L., Latzman, R. D., Boness, C. L., Kotov, R., Keyser-Marcus, L., DeYoung, C. G., Krueger, R. F., Zald, D. H., Moeller, F. G., & Ramey, T. (2023). New approaches to deep phenotyping in addictions. *Psychology of Addictive Behaviors*, *37*(3), 361–375. https://doi.org/10.1037/adb0000878

Watts, A. L., Watson, D., Heath, A. C., & Sher, K. J. (2023). Alcohol use disorder criteria exhibit different comorbidity patterns. *Addiction*, *118*(8), 1457–1468.

Watts, A. L., Wood, P. K., Jackson, K. M., Lisdahl, K. M., Heitzeg, M. M., Gonzalez, R., Tapert, S. F., Barch, D. M., & Sher, K. J. (2021). Incipient alcohol use in childhood: Early alcohol sipping and its relations with psychopathology and personality. *Development and Psychopathology*, *33*(4), 1338–1350.

Wiegmann, C., Mick, I., Brandl, E. J., Heinz, A., & Gutwinski, S. (2020). Alcohol and dementia–what is the link? A systematic review. *Neuropsychiatric Disease and Treatment*, *16*, 87–99. https://doi.org.10.2147/NDT.S198772

Wilsnack, S. C. (2012). The GENACIS project: A review of findings and some implications for global needs in women-focused substance abuse prevention and intervention. *Substance Abuse and Rehabilitation*, *3*(sup1), 5–15. https://doi.org.10.2147/SAR.S21343

Wilson., S., & Rhee, S. H. (2022). Special issue editorial: Leveraging genetically informative study designs to understand the development and familial transmission of psychopathology. *Development and Psychopathology*, *34*, 1645–1652. https://doi.org/10.1017/S0954579422000955

World Health Organization (2018). *Global health observatory data repository*. World Health Organization, Geneva. https://apps.who.int/gho/data/view.main.54500.

Zapolski, T., Pederson, S., McCarthy, D., & Miller, G. T. (2014). Less drinking, yet more problems: Understanding African American drinking and related problems. *Psychological Bulletin, Jan*, *140*(1), 188–223. https://doi.org/10.1037/a0032113

Zhao, J., Stockwell, T., Naimi, T., Churchill, S., Clay, J., & Sherk, A. (2023). Association between daily alcohol intake and risk of all-cause mortality: A systematic review and meta-analyses. *JAMA Network Open*, *6*(3), e236185–e23618.

Zheng, Y., Meyer, Z., Unger, J. B., & Rijsdijk, F. (2023). Gene-environment interplay linking perceived parental supervision and peer drunkenness with Chinese adolescent alcohol initiation. *Child Development*, *94*(4), 853–864. https://doi.org/10.1111/cdev.13902

https://doi.org/10.1017/S0954579424000671 Published online by Cambridge University Press