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Alcohol Misuse Across the Lifespan: Insights from Developmental Studies in Behavior Genetics

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Abstract

Alcohol misuse, one of today's greatest public health challenges, is a developmentally dynamic, complex behavior at the intersection of genetic and environmental influences. This review examines such influences from a behavior genetics perspective and discusses implications for public policy. Alcohol misuse is moderately heritable with genetic influences accounting for around 50% of its variance, but to date few specific genes have been identified. However, numerous environmental and social factors moderate genetic risk, including parents, peers, romantic partners, family dynamics, employment, laws, and cultural influences. These moderating factors change in salience across development, and, accordingly, no one-size-fits-all approach is suitable for reducing alcohol misuse at a large scale. We provide examples of some effective prevention and intervention programs and discuss a framework for using the behavior genetics evidence to inform future public policy efforts.

Keywords

alcohol misuse; alcohol use disorder; public policy; behavior genetics; development

Overview

Alcohol misuse (AM) is one of today's greatest public health challenges (Office of the Surgeon General, 2016). In the United States alone, alcohol-related diseases are the third leading cause of preventable death (Mokdad, Marks, Stroup, & Gerberding, 2005), and AM costs the U.S. nearly $250 billion annually (Rehm et al., 2009; Sacks, Gonzales, Bouchery, Tomedi, & Brewer, 2015). As a public health concern, the consequences of an individual's AM are not self-contained but impact multiple aspects of society: from family and peer networks (e.g. damaged relationships, physical and sexual assault) to regional and national communities (e.g. drunk driving, burdens on the healthcare system). Social influences, in turn, reciprocally shape individuals' alcohol use (Galea, Nandi, & Vlahov, 2004).

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The intersection of individual predispositions with environmental and social facilitation (or deterrence) has made AM a key outcome for investigation in the behavior genetics (BG) field. This science seeks to unravel the contributions of genetic and environmental influences to complex behaviors. The BG perspective has conceptualized AM as a dynamic phenomenon in which one's innate predispositions are shaped towards or away from alcohol problems by the risk and protective factors encountered across development (Tarter & Vanyukov, 1994). In recent decades, BG research has begun to identify the genetic influences on those predispositions and discover what shapes their expression.

AM encompasses multiple behaviors involving the excessive, inappropriate, or harmful consumption of alcohol that causes negative consequences in health, work, legal, or social functioning. It is often defined based on alcohol use disorder (AUD) criteria set forth by the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), or by measures of acutely harmful excessive consumption such as binge drinking, defined by the National Institute on Alcohol Abuse and Alcoholism as 4 or more drinks in one occasion for women and 5 or more for men (Wechsler & Nelson, 2008). Although binge drinking is episodic, AUDs reflect a progressive pattern of excessive use despite repeated negative consequences.

In the U.S. population, the lifetime prevalence of AUDs is estimated at 14% (Grant, Goldstein, Saha, & et al., 2015) and past-month binge-drinking prevalence at 17–25% (Center for Behavioral Health Statistics and Quality, 2016). However, the prevalence of alcohol use and misuse differs across demographic factors. Age is one: AUD prevalence increases sharply between adolescence and adulthood (Substance Abuse and Mental Health Services Administration, 2014). This increase is more pronounced for males than females (Chen, Yi, & Faden, 2015). After mid-adolescence, males consistently drink and experience problems at higher rates than females, although gender differences have attenuated recently due to secular trends of increasing AM among females (Grucza, Bucholz, Rice, & Bierut, 2008). AM also varies significantly across racial/ethnic groups (Chartier & Caetano, 2010), with higher rates in European Americans than minority populations in the U.S. (Grant et al., 2015). Education, religious affiliation, and geographic region are also linked to rates of AM (Meyers, Brown, Grant, & Hasin, 2016; Naimi et al., 2003).

These many contributing factors demonstrate the malleability of AM and highlight why policy interventions can shape outcomes. Although consumption and metabolism of alcohol is a basic biological process, individuals' alcohol use is complex and modified by higher-order factors (Kendler, 2012). Alcohol use is also developmentally dynamic, with biological and psychosocial influences both changing constantly throughout the lifespan, as we review in the following section.

Etiology of alcohol misuse

Genetic influences

The heritability of adult alcohol use and AUD is approximately 50% (Dick, Meyers, Rose, Kaprio, & Kendler, 2011; Verhulst, Neale, & Kendler, 2015), indicating that genetic factors account for roughly half of the inter-individual differences in both alcohol consumption and AUD in the population. Shared genetic influences are found between heavy alcohol

consumption and AUD symptoms (Dick et al., 2011), and between these alcohol use behaviors and disorders such as other drug dependence, conduct disorder, and antisocial personality disorder (Dick, Prescott, & McGue, 2009). This suggests a broad genetic predisposition toward "externalizing" behaviors, which may reflect a common intermediate mechanism such as impulsivity or reward-seeking (Dick et al., 2010).

Although well-replicated research demonstrates genetic influences on alcohol use/misuse, identifying the specific genes that underlie this heritability has been challenging. AM is polygenic: many genetic variants of small effects across the genome contribute to its heritability (Sullivan, Daly, & O'Donovan, 2012). The large sample sizes needed to detect such small effects have made it difficult to identify the genes involved. Notable exceptions include variants in genes that code for alcohol dehydrogenase and aldehyde dehydrogenase, which help metabolize alcohol (Hart & Kranzler, 2015). Some variants of these genes result in unpleasant side effects when drinking (nausea, facial flushing), leading to decreased alcohol consumption. Genetic variants in neurotransmitter systems show mixed evidence (Buhler et al., 2015), but meta-analyses implicate some receptors for dopamine (Wang, Simen, Arias, Lu, & Zhang, 2013), GABA (Zintzaras, 2012), and opioids (Schwantes-An et al., 2016). However, few genes have been implicated by the stringent, genome-wide association tests considered standard today.

The lack of gene identification despite considerable efforts suggests that AM is not a uniform outcome with a single set of genetic risk factors. Indeed, research supports numerous potential genetic predisposition pathways to AM, such as sensitivity to its rewarding or withdrawal-induced effects (Koob & Le Moal, 2008), impaired executive functioning and impulse control (Everitt & Robbins, 2005), and intoxication susceptibility (Schuckit et al., 2009).

Environmental and social influences

The environmental influences on AM expand outward from the individual, who is nested in the immediate social microsystem, which is nested in the community exosystem, all of which is encompassed by the broader geographical, cultural, and temporal macrosystem (e.g. Bronfenbrenner, 1979). At the microsystem level, family characteristics (such as cohesion, support, acceptance, and affection) are associated with lower rates of AM among adolescents (Reeb et al., 2015), whereas family conflict, aggression, and neglect are associated with higher risk (Repetti, Taylor, & Seeman, 2002). Parenting behaviors, such as monitoring and involvement in children's lives, influence adolescent AM (Barnes, Reifman, Farrell, & Dintcheff, 2000; Profe & Wild, 2015), while peers shape AM in adolescents and young adults (Samek, Keyes, Iacono, & McGue, 2013), and romantic partners begin to impact AM in young adulthood (Fischer & Wiersma, 2012).

Outside of the immediate social circles, local communities also play important roles. School-level norms and attitudes about alcohol use predict students' individual alcohol use (Su & Supple, 2016). Perceived social norms regarding drinking predict AM in college campuses (Perkins, 2002). Adverse neighborhood conditions, including economic and social disadvantage, are associated with increased AM (Cerdá, Diez-Roux, Tchetgen, Gordon-Larsen, & Kiefe, 2010; Mulia & Karriker-Jaffe, 2012).

Broader social conditions such as national or local legal restrictions can directly affect one's ability to obtain alcohol, and therefore one's likelihood to develop problems. Increases in the minimum legal drinking age reduce alcohol consumption (Wagenaar & Toomey, 2002), as do policies like higher taxes on alcohol sales (Elder et al., 2010; Wagenaar, Salois, & Komro, 2009). Economic conditions affect drinking rates: alcohol consumption is greater in wealthier countries (Rehm et al., 2009), but economic instability and income inequality also increase alcohol problems (de Goeij et al., 2015; Elgar, Roberts, Parry-Langdon, & Boyce, 2005). Cross-cultural differences in alcohol use/misuse patterns show higher rates for nations (and ethnic groups) with more individualistic than collectivist cultural orientations (Johnson, 2007). For racial/ethnic minority populations, culturally relevant experiences such as discrimination (Martin, Tuch, & Roman, 2003) and racial socialization (Hughes et al., 2006) predict risk for alcohol problems. In the U.S., immigrants and ethnic minorities have lower rates of AM than European Americans (Hasin, Stinson, Ogburn, & Grant, 2007), but rates increase as a function of integration into the national culture (Savage & Mezuk, 2014).

Developmental influences

On average, alcohol use and AM increase sharply from age 12–17, peak around age 18–25, and level off into adulthood (Substance Abuse and Mental Health Services Administration, 2014). However, such averages mask individual differences in alcohol use trajectories. Although most people exhibit a stable pattern of low or moderate use after initiation, more problematic trajectories also occur: a) early initiation and persistent heavy use throughout life (heavy chronic users), b) initiation in mid/late high school and a rapid, steep increase in use shortly after (late-onset users), and c) time-limited brief periods of heavy use, typically in adolescence ("fling" drinkers) (Brown et al., 2008; Schulenberg, O'Malley, Bachman, Wadsworth, & Johnston, 1996).

Interaction among genetic, environmental, and developmental factors

Different aspects of the environment vary in salience across development (Masten, Burt, & Coatsworth, 2006), so genetic and environmental factors influencing AM change across the lifespan. Gene by development (GxD) and environment by development (ExD) effects shaping AM can occur as certain factors "come online" (or offline) at each life stage. However, genetic and environmental factors can also moderate each other (gene-by-environment interaction; GxE), and these interaction effects may also change across development (GxExD). This makes for an extraordinarily complex picture.

GxD—Genetic influences on alcohol use change across life, particularly during the transition from childhood to adulthood. Estimates of heritability for alcohol use start at zero for adolescents aged 13–15 and increase throughout late adolescence and early adulthood, with a plateau near 50% around age 24 (Dick et al., 2007; Dick et al., 2009). Environmental factors therefore largely influence initiation of use, while genetic influences play an increasing role on developing alcohol use patterns later in life. Genetic influences on AM also change qualitatively. In adolescence, genetic predispositions impacting alcohol use are mostly shared with antisocial behavior, while in young adulthood genetic influences become more specific to AM (Meyers et al., 2014). Some studies have shown that the same genes influence AM across development (van Beek et al., (2012) while others show evidence of

new genes coming online later in life (Edwards & Kendler, 2013; Long, Lonn, Sundquist, Sundquist, & Kendler, 2017).

ExD—As reviewed above, family, peers, and schools have their greatest influences on alcohol use/misuse during childhood and adolescence when home and school environments dominate an individual's life. Adolescence represents a period linking childhood and adulthood, with tremendous transitions in social/occupational roles, family, and self-identity (Windle et al., 2008). The increased autonomy, opportunities, and responsibilities experienced during the transition mean that experiences in these new domains (e.g., leaving home, changing occupational roles, romantic relationships, parenthood/family) become more salient predictors of adult alcohol use/misuse (for a review, see Stone, Becker, Huber, & Catalano, 2012).

GxE—The most consistent GxE effects are for environments that differ in the opportunity to express genetic predispositions toward alcohol problems. Generally, genetic influences on alcohol outcomes are greater in more permissive environments (Shanahan & Hofer, 2005). In adolescence, greater genetic influences emerge in the context of lower parental monitoring/knowledge (Miles, Silberg, Pickens, & Eaves, 2005) and higher peer deviance (Dick et al., 2007; Harden, Hill, Turkheimer, & Emery, 2008). Urban residency, a higher percentage of young adults in one's neighborhood, and higher neighborhood transiency (frequent migration in and out of the community) are associated with greater genetic influences on adult alcohol outcomes (Dick, Rose, Viken, Kaprio, & Koskenvuo, 2001; Rose, Dick, Viken, & Kaprio, 2001).

Also related is the process of gene-environment correlation (rGE), whereby individuals create or self-select into particular environments that are associated with their genotype (Scarr & McCartney, 1983). For example, genetic factors that contribute to AUD are also associated with divorce (Salvatore et al., 2017). Thus, individuals who have a genetic predisposition for AUD may also be more likely to find themselves in environments that increase their risk.

GxExD—The importance of different environmental moderators of genetic risk matter varies across developmental stages. For example, parental monitoring shows a greater moderating role on alcohol use starting earlier in adolescence, while the moderating role of peer substance use does not emerge until later in adolescence (Dick et al., 2007). This suggests that genetic influences on alcohol use require understanding the environment, while also keeping mind that the relevance of many environmental factors shifts across development.

Implications for policy

BG methodologies are informative for evidence-based policy because they can tease apart which outcomes are genetically or environmentally influenced and refute the idea that behaviors are biologically determined, or static across environments. They also can achieve a greater traction on causality than epidemiological studies, and thus aid in finding the most efficacious targets for prevention and intervention efforts (McGue, Osler, & Christensen,

2010). However, policy efforts have largely not yet integrated this kind of information. In the interim, we provide some examples of policy interventions that have reduced AM, and we discuss the implications of the existing scientific evidence for developing future policy approaches.

Policy examples

The level-of-response-to-alcohol paradigm (Schuckit, Kalmijn, Smith, Saunders, & Fromme, 2012) tailors prevention toward college students with a low level of response to alcohol (LR), which is a genetically influenced risk factor (Schuckit et al., 2009). Students decreased their heavy drinking when enrolled in a prevention program matched to their known risk factor of low LR. A subsequent replication found that the low LR participants were the most likely to respond to a prevention program, whether it was tailored or not (Savage et al., 2015), indicating that those at highest risk for AM are also most likely to benefit from programming efforts.

Preventure (Conrod, Stewart, Comeau, & Maclean, 2006) is a school-based brief intervention that draws on evidence showing specific personality profiles to be more at risk for developing alcohol problems (i.e., sensation seeking, impulsivity, anxiety sensitivity, and hopelessness/negative thinking). The focus is to teach adolescents more effective coping strategies for their given personality. Preventure reduced drinking quantity, binge drinking rates, and alcohol problems in a pilot study in Canada (Conrod et al., 2006) and demonstrated similar efficacy in the UK (O'Leary-Barrett et al., 2013), Australia (Teesson et al., 2017), and the Netherlands (Lammers et al., 2017).

These examples of personalized prevention/intervention suggest that individually tailored efforts can be effective, illustrating that selective rather than universal prevention might be most beneficial. However, as discussed above, individual-level risk factors are only one piece of the puzzle. In a larger scale program in Iceland, for example, a broad range of individual, family, community, and societal level factors were targeted through collaborations between scientists, practitioners, policy-makers, and the community (Sigfúsdóttir, Thorlindsson, Kristjánsson, Roe, & Allegrante, 2009). Some initiatives supported parents in enforcing curfews, discouraging unsupervised parties, and monitoring youth in the community. Adolescents' recreational and extracurricular activities opportunities were increased, and support networks between parents, youth, and the community were encouraged. After implementation, the percentage of 10^{th} graders who reported drinking to intoxication in the last 30 days was cut in half over a decade (Sigfúsdóttir et al., 2009). The Icelandic findings suggest that prevention efforts are most effective when they simultaneously strengthen protective factors and reduce risk factors at all levels - individual, family, community, and societal.

The U.S. has relied primarily on tax policy interventions and minimum drinking age laws to reduce AM, with some demonstrated effectiveness. Two systematic reviews showed that higher alcohol costs or taxes consistently decreased excessive drinking, alcohol-related health outcomes, and alcohol-related harms (Elder et al., 2010; Wagenaar et al., 2009). Likewise, a large critical review examining the effect of minimum drinking age law showed an inverse relationship between the minimum drinking age and alcohol consumption as well

as traffic accidents (Wagenaar & Toomey, 2002). Thus, large-scale policy efforts that reduce access to alcohol are one effective means of reducing alcohol consumption and related consequences.

Inferences from research

The epidemiological, neurobiological, and behavior genetics research indicate possible directions to improve existing policy. First, although the difficulties in identifying the specific genes or genetic variants that confer risk for AM has made the move towards personalized medicine challenging (Collins & Varmus, 2015), we have gained some knowledge about how genes (in aggregate) influence AM. Research has delineated pathways of genetic risk for alcohol use disorder, and how environmental factors may shape the ultimate expression of that genetic predisposition. For example, knowing that environmental influences matter more than genetic influences on adolescent alcohol use suggests reducing adolescents' alcohol misuse will be dependent on parental, peer, and school interventions. Likewise, knowing that genetic predispositions for alcohol use disorder manifest earlier in development as higher levels of sensation seeking and impulsivity can help identify children who may benefit most from preventive strategies aimed at channeling those tendencies into more pro-social outlets (e.g., rock climbing, rather than binge drinking) later in life. The gene-environment interplay for alcohol outcomes highlights the roles for parents, peers, romantic partners, family dynamics, employment opportunities, legal restrictions, and cultural norms to shape whether and how a genetic predisposition for alcohol misuse may manifest.

Second, a BG perspective recognizes that individuals, and their inherent biology, exist within multi-leveled systems. Influences at each level shape AM, so the range of targets for policy implementation is broad. An integrative policy approach might include programs that strengthen parenting behaviors and reduce misperceptions of alcohol use norms among peers (at the micro-level), improve neighborhood cohesion and provide school/community activities as an alternative to drinking (at the exo-level), and affect legal availability and reshape cultural practices that normalize binge drinking and other harmful alcohol use (at the macro-level). Such programs could take shape in countless ways.

Third, developmental research provides an overarching structure to interpret and actualize other findings. Because AM is developmentally dynamic and its genetic and environmental determinants change (quantitatively and qualitatively) across time, effective policies for reducing AM will differ across the lifespan. What is considered problematic alcohol use changes from childhood to adulthood; thus the target behavior to be modified and effective policies with which to do so also change rapidly as different genetic, environmental, and social risk/resilience factors become more salient. Even at a within-person level of development, different programming (prevention vs. intervention vs. treatment) will be most applicable at different stages of an individual's alcohol use trajectory. No one-size-fits all program can effectively reduce AM on a large scale.

Conclusions

Although much remains to be learned, a few conclusions follow from existing research. We can reliably say that any factor that facilitates easier alcohol access increases AM, while socially or legally restrictive factors reduce AM. This well-replicated pattern points to a social/environmental suppression of genetic predispositions. Such mechanisms work indirectly because they reduce overall alcohol use, not specifically problematic use. However, the costs to society are not driven solely by binge drinking and AUDs, as even normative levels of alcohol use can contribute to health consequences, productivity loss, accidents, and crime (Office of the Surgeon General, 2016). On the other hand, individual-level risk factors can be effectively incorporated into targeted programs to prevent or reduce AM among a high-risk subset of the population, who are also the most likely to benefit from programming. The goal of a policy intervention determines which approach is most desirable, although the complexity of alcohol misuse calls for an integrative, multifaceted approach for any effective policy.

A BG perspective provides two takeaways for planning such approaches. First, the heritability of AM means that some people are more at risk of developing problems than others. Knowing the specific genes involved and how genetic risk is shared with other traits and disorders identifies the etiological pathways essential to understanding the nature of AM. Second, the fact that genetic influences on AM change across environmental contexts and across development tells us that policies are worthwhile, because AM is not static, unchangeable behavior. This approach also informs when certain policies would be most effective, and which environmental moderators could be used to enact them. Although we are still far from predicting individual risk of alcohol problems from DNA, gene identification for other complex behavioral traits are rapidly gaining success (CONVERGE Consortium, 2015; Otowa et al., 2016; Schizophrenia Working Group of the Psychiatric Genomics Consortium, 2014). As BG research evolves, identification of specific genes involved in AM may yield richer insight into the biological mechanisms behind heritability and gene-environment interaction. Changing social environments, such as norms about marriage and other social relationships, may provide new avenues for environmental and developmental moderation. Despite these changes, alcohol misuse will always be a complex behavior at the intersection of individual predispositions and nuanced social influences, unfolding across development. Any efforts to fully understand this behavior, and especially to change it, require recognition of its complexities.

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Highlights

- Alcohol misuse prevalence and characteristics differ across development and numerous demographic factors.
- Alcohol misuse is heritable, but many genes have small individual effects, most genes have not yet been identified, and their importance changes at different ages.
- Environmental and social factors can deter access to alcohol, reduce misuse, and diminish the expression of genetic predispositions toward developing problems.
- Both individually tailored programs and universal policy changes can reduce alcohol misuse, depending on context and goals.
- The complexity of alcohol misuse requires an integrative, multifaceted policy approach that recognizes individuals as nested within sociocultural influences that change across development.