

# Why we want to drink, what that has to do with genes, and why it matters for our alcohol risk

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Motives for drinking—to party, to conform, to cope, or to feel good—are consistent through young adulthood, and genes play a role in

how those motives influence alcohol use, a new study of college students suggests. Understanding the mechanisms linking genetic variants to differences in drinking behaviors could present opportunities for predicting individuals' vulnerability to Alcohol Use Disorder (AUD) and intervening to prevent it.

Genetic factors are responsible for about 50% of individual risk of AUD. Much of how that heritability functions is unexplained, however. The relationship between genes and drinking behavior is complex, involving thousands of genetic variants that each have small effects. Critical factors known as endophenotypes, or intermediary phenotypes, affect how an individual's genetic predisposition manifests as a behavioral trait.

For their new study in *Alcoholism: Clinical & Environmental Research*, investigators sought to determine whether drinking [motives](#) are one such factor, and the roles of associated genetic and [environmental influences](#). Drinking motives can be negative or positive: a response to unpleasant emotions (coping), an urge to fit in (conformity), a desire for pleasurable effects (enhancement), or a part of enjoying others' company (social). These motives contribute to [alcohol use](#) and [alcohol-related problems](#), but there has been relatively little study of the causal pathways involved in their development, especially [genetic factors](#).

Researchers worked with data from 10,000 first-year students enrolled at a U.S. public university in 2011-2015. Half of the participants were white, and 60% were women. The students completed an initial online questionnaire and annual follow-up surveys throughout their college years. The surveys covered alcohol use, AUD symptoms, drinking motives, and relevant environmental exposures (how much autonomy they'd been allowed around drinking, peer behaviors such as getting drunk and cutting school, and exposure to trauma, e.g., assault or natural disaster). The DNA of 4,900 participants was analyzed. The researchers

used statistical analysis to explore associations between the students' drinking motives, demographic and environmental characteristics, and genetically influenced pathways that contribute to alcohol use.

The study demonstrated that drinking motives were stable throughout the college years, similarly to [personality traits](#). Some environmental predictors of alcohol misuse were associated with all types of drinking motives; parental involvement was linked to lower levels and peer deviance to higher levels. Trauma, however, was more specifically linked—to lower social motives and higher coping motives. The study also found correlations between drinking motives and alcohol use outcomes. Drinking to cope was linked with AUDs and enhancement and social motives with both consumption and AUDs.

The study also provided promising but inconclusive evidence on the biology underlying drinking motives and the influence of genetic variants on alcohol misuse. Genetic factors seemed to link coping motives with AUD. Some genetic variants appeared to be associated with drinking for conformity, and others with drinking for enhancement. The process by which genetic variants influence positive drinking motives (enhancement, social) may differ from that of negative motives (coping, conformity).

The stability of drinking motives across early adulthood and support for motives as endophenotypes influencing alcohol misuse both facilitate further research. Given the complexity of genetic influences on behavioral and psychiatric traits, larger studies are needed to investigate which genes are involved with which [drinking](#) motives.

**More information:** Jeanne E. Savage et al, Genetic and environmental etiology of drinking motives in college students, *Alcoholism: Clinical and Experimental Research* (2022). [DOI: 10.1111/acer.14930](https://doi.org/10.1111/acer.14930)

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