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Behavioral genetics and population health interventions for alcohol problems: at odds or oddly in agreement?

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This commentary argues that findings from behavioral genetics research can be reconciled with population health approaches to dealing with alcohol problems. Such a contention may seem counterintuitive, as these approaches to the causes of, and responses to, alcohol problems appear to be at odds with one another. Studies on behavioral genetics found that ~50–70% of the population variability in the risk for alcohol dependence can be attributed to genetic influences.[1] Biomedical advocacy groups align these findings with definitions of alcohol dependence as a brain disease and state that medical approaches are the ideal way to deal with this issue.[2] Population health, on the other hand, has little concern for individualized approaches and instead concentrates on interventions that are directed at the entire population, e.g., increasing the price of alcohol through taxation. Such interventions lead to reductions in both alcohol consumption and drinking problems, including among those who have severe problems.[3] Aggregate relationships between consumption and a wide range of problems are very well established.

However, there are also some commonalities in these approaches when one scratches beneath the surface. Results from behavioral and molecular genetics studies clearly imply that alcohol dependence is not caused by one gene, or even one discrete set of genes. Rather, as is the case with most complex behaviors and disorders, genetic contributions to the risk for alcohol dependence likely reflect the aggregate impact of a multitude of genetic variants and gene networks. These variations collectively contribute to the expression of alcohol problems of a range of different severities, up to and including alcohol dependence. This perspective, which is consistent with findings that alcohol problems and related behaviors reflect continuous rather than categorical phenotypes, suggests an underlying unimodal distribution of genetic vulnerability to alcohol problems across the general population rather than a bimodal distribution in which a minority of people have a genetic predisposition to alcohol dependence and the rest do not.

In this unimodal distribution behavioral genetics may align with a population health orientation to dealing with alcohol problems. It was once unclear whether patterns of alcohol consumption and related problems in the general population were unimodally or bimodally distributed. This is no longer the case, and it is now widely accepted that a unimodal distribution of drinkers, albeit with skew toward the heavier consumption end, most accurately captures the way in which alcohol problems are distributed. The precise shape of the unimodal distribution varies in different societies, according to history and culture.[3]

Population health interventions are also well suited for dealing with the problems associated with alcohol use that result, at least in part, from genetic variation. The major implication of the unimodal distribution is that effective means of addressing alcohol problems need to be applied to the entire population to "shift" the distribution to the left (including those at the severe end).[3] This makes alcohol problems just like other problems targeted in preventive medicine. The alternate approach would be to target only those with severe problems, but there is little evidence for the efficacy of this at the population level. We stress that this does not mean that clinical approaches are not important to help individuals with alcohol concerns. There remains a need for clinical interventions, both psychosocial and pharmacological, to aid individuals who decide to seek help for alcohol problems. However, because only a small minority of those with alcohol dependence will ever access treatment, it should be expected that population interventions will have a broader impact. Moreover, the complex polygenic nature of alcohol dependence, coupled with evidence for strong contributions of environmental factors, means there is little evidence or likelihood that molecular genetic approaches to identifying those at greatest risk would be feasible or effective on a broad level. If the goal is to have an impact on the incidence of alcohol problems in the general population, then the unimodal distribution needs to be shifted to the left (i.e., fewer drinks on average) for the incidence of those with severe alcohol problems to be reduced. The only interventions with a strong evidence base for reducing the prevalence of alcohol consumption and the resultant consequences at the general population level are increasing price (taxation), reducing availability, drinking and driving legislation, and making heavy drinking less culturally acceptable through marketing restrictions.[3] One limitation of these population health interventions is that there is often strong opposition to their implementation, and different countries have at different times adopted, or repealed, these policy approaches. However, these policy changes have been accompanied by concomitant shifts

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in the distribution of alcohol consumption and consequences, thereby reinforcing the evidence base in support of their use.[3]

The rationale for population-level interventions can also be viewed through the lens of behavioral genetic research documenting gene-by-environment interactions on the risk for alcohol use. For example, the magnitude of genetic influences on alcohol consumption is often reduced in the context of environmental contingencies that limit access to, or acceptability of, alcohol use.[4] Therefore, increasing environmental controls could arguably decrease the variability in risk attributable to genetic influences, perhaps yielding net benefits, particularly for those at the moderate or high end of the distribution of genetic risk. It is also noteworthy that although genetic factors contribute substantially to population variability in the risk for alcohol dependence, genetically informative designs reveal that recovery from alcohol dependence is attributable entirely to environmental influences.[5]

The environment thus has an important direct role in the development of, and a predominant role in recovery from, alcohol dependence. Alongside evidence that environmental factors may attenuate genetic risk for alcohol dependence, these observations imply that population health interventions will have a more influential role than biomedical treatments in reducing the overall burden of alcohol consumption and alcohol dependence. Strange bedfellows indeed.

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DISCLOSURE

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