By innovative use of administrative data sources, Han et al1 established a cohort study of 1537 836 adult Asian individuals aged 20 to 39 years and examined the association between self-reported alcohol consumption and risk of atrial fibrillation (AF). During a median follow-up period of 5.6 years, AF occurred in 3066 participants. Persistent and moderate to heavy drinking and higher cumulative alcohol consumption was associated with higher risk of AF. These findings are in line with a recent dose-response meta-analysis on alcohol and risk of AF based on published studies up to and including 2020, which included 645 826 participants of mainly European ancestry and 23 079 cases of AF, which documented increasing risk of AF by increasing alcohol consumption.2 The results of the current study1 extend our knowledge on the unfavorable association between alcohol intake and AF toward younger adults, an age group usually not at high risk of AF.

In 2021, 2 very large cohort studies confirmed an association of alcohol with long-term incidence of AF. A pooled community-based cohort study followed 107 845 individuals over a median time of 13.9 years, and 5854 individuals developed AF. It was documented that increasing alcohol consumption was associated with a nonlinearly increasing risk of AF.3 The UK Biobank Study4 included 403 281 middle-aged individuals and over a median follow-up time of 11.4 years, 21 312 participants developed AF. A J-shaped association of total alcohol consumption was observed with the lowest risk of AF with fewer than 7 UK standard drinks equivalent to 4 US standard drinks per week, and the risk of AF increased by increasing alcohol consumption.

A well-conducted open-label, randomized, controlled trial5 documented the role of modification of factors associated with risk. Alcohol abstinence in adults with paroxysmal or persistent AF who consumed 10 or more standard drinks of 12 g of pure alcohol per week reduced AF burden significantly. In patients with paroxysmal AF, an n-of-1 trial6 of self-selected AF triggers in 446 participants demonstrated acute exposure to alcohol as an exogenous trigger of AF episodes. Contrasting these findings, the Framingham Heart study7 surprisingly reported that alcohol consumption was not significantly associated with population-attributable risk of incident AF.

How can we understand the paradox that exposure to alcohol is associated with risk of AF and that alcohol abstinence is associated with a lower AF burden, but alcohol is not associated with population-attributable risk of AF? We find it likely that acute outcomes of alcohol on atrial electrophysiology and the autonomic nervous system may explain the excess risk of AF in young people over a short follow-up period,1 but that the excess risk of AF by alcohol consumption over many years is mediated via the association between alcohol and other factors associated with risk for AF such as overweight, obesity, hypertension, and diabetes. These factors associated with risk are highly concurrent with alcohol intake, in particular in individuals who drink higher doses. Such factors associated with risk were updated over time and statistically adjusted for in the Framingham Heart Study,7 but not in other cohort studies. These mediated outcomes of alcohol via other factors associated with risk for AF may explain why alcohol was not associated with risk of AF in the Framingham Heart Study.7

We do not think that the current evidence base in relation to the association between alcohol consumption and risk of AF in the general population can be improved substantially, because randomization to long-term exposure to alcohol cannot be ethically justified. We have to live with the inherent risk of bias in observational cohort studies. There are particular concerns in relation to biases in self-reported alcohol consumption, which most of the studies had to rely on. Those with the highest consumption may report a lower consumption than the true consumption, which will bias the
true risk associated with alcohol downward. There may also be detection bias of AF among heavy drinkers because of low self-care. This will also bias the true risk downwards.

The public health implications in relation to our current knowledge of alcohol and AF are clear and based on firm evidence. To prevent AF, drink less than 2 standard drinks of alcohol per day. To reduce AF burden, abstain from alcohol. A recommendation to start alcohol consumption to prevent AF can certainly not be given. Salute to the investigators for the elegant study on alcohol and AF in young adults presented in this issue of JAMA Network Open.¹

ARTICLE INFORMATION
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