on my fellow leaders, including those in predominantly Muslim countries, to consider the findings of this study and work together—with ambitious resolve, across borders and sectors—to fully implement the 2030 agenda. Investing in women’s, children’s, and adolescents’ health is a smart place to start.

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I declare no competing interests.

Thresholds for safer alcohol use might need lowering

Guidelines for levels of alcohol use that pose a low risk to drinkers’ health are provided by many countries, usually based on meta-analyses of epidemiological studies.1–3 However, to devise such guidelines is challenging because alcohol is linked to poor health in various and complex ways. Injury, suicide, and assault, for example, are associated with drinking to intoxication, whereas regular alcohol consumption increases the risks of liver cirrhosis, gastrointestinal diseases, cardiovascular disease, dementia, and some types of cancer.4,5 Debate continues about whether or not moderate alcohol consumption reduces mortality from myocardial infarction.6,8

The study by Angela Wood and colleagues9 in The Lancet substantially improves on previous meta-analyses to define low-risk drinking thresholds.

The investigators analysed individual-participant data for alcohol use in 599,912 current drinkers in 83 prospective studies in 19 countries, each of which collected extensive self-report and biological measures of cardiovascular disease risk. Primary analyses were restricted to current drinkers who had no previous history of cardiovascular disease. Non-drinkers were excluded to minimise the possibilities of reverse causality (eg, if ex-drinkers had abstained because of poor health) or unmeasured effect modification (ie, if lifetime abstainers fundamentally differ from drinkers). Wood and colleagues used data from three large consortia of prospective studies.10–12 These studies all used similar methods to quantify alcohol use, cardiovascular risk factors, and cardiovascular disease outcomes and cause-specific deaths; provided data for individual participants for detailed statistical analyses; were done in high-income or developed countries with similar drinking patterns; and measured alcohol consumption in ways that could be harmonised using the same units (grams of pure ethanol). All participants were followed up for at least 1 year (for 5·4 million person-years of follow-up) and analyses were adjusted for stability of alcohol consumption using serial alcohol consumption assessments where available (n=71,011 individuals, with assessment done a median of 5·6 years apart).

Around half of the total study sample reported consuming more than 100 g of alcohol per week and 8·4% drank more than 350 g per week. There were 40,317 deaths from all causes and 39,018 first incident cardiovascular disease events; these large numbers


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mean that the study had excellent statistical power for the major statistical analyses.

Wood and colleagues noted a positive curvilinear association between alcohol use and premature mortality. The lowest risk of premature death was in those consuming alcohol at levels of 100 g per week or less. As consumption increased, the risk of death increased from stroke, coronary disease (excluding myocardial infarction), heart failure, fatal hypertensive disease, and fatal aortic aneurysm. As has been reported in other studies, moderate levels of alcohol consumption were associated with a reduced risk of mortality from myocardial infarction.

With some exceptions, the findings persisted after adjustment for known cardiovascular disease risk factors. The notable exceptions were adjustment for HDL cholesterol weakened the inverse association between alcohol use and myocardial infarction while strengthening the association between alcohol consumption and increased risks for coronary heart disease and heart failure. Meanwhile, adjustment for systolic blood pressure strengthened the inverse association between alcohol use and myocardial infarction while weakening the positive associations with all other cardiovascular outcomes.

In secondary analyses, former drinkers (n=29,726) and—to a lesser extent—never drinkers (n=53,851) had a higher risk of cardiovascular disease (and all-cause mortality) than even the heaviest drinkers in the sample. However, the ex-drinkers and never-drinkers probably represent quite different groups of people with distinct measured and unmeasured health characteristics. For example, compared with current drinkers, in never drinkers there was a higher proportion of women, non-white participants, people with poorer educational outcomes, and a higher prevalence of diabetes.

Wood and colleagues estimate that, at the population level, reductions in alcohol consumption could increase life expectancy by up to 2 years in a 40-year-old drinker. However, these gains only become evident at alcohol consumption below 100 g per week, and are not offset by the reductions in rates of myocardial infarction. The 100 g per week threshold is substantially lower than current guidelines in many high-income countries (eg, 196 g/week in the USA). These associations have been less robust in studies in lower income countries, so further research is needed to investigate the stability of these findings in such populations over longer periods of time.

The drinking levels recommended in this study will no doubt be described as implausible and impracticable by the alcohol industry and other opponents of public health warnings on alcohol. Nonetheless, the findings ought to be widely disseminated and they should provoke informed public and professional debate.

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