does not necessarily detract from their importance as confounders. If adjustment for height provides a more accurate effect estimate, it should be done, even if height is only a marker for truly causal factors. Of course, if height is only an intermediate between the study exposure and outcome, then adjustment for it is inadvisable (unless one wishes to eliminate effects mediated through body size). Rarely will we know that height is only a marker or only an intermediate, however, and hence a height-adjusted analysis will be needed to complete a sensitivity analysis of the results.

Moreno et al. (2) seem to make a point similar to Dr. Hense’s comment on biologic maturity. They argue that there are significant differences in body mass index before and after the onset of puberty and, thus, pubertal status might be a confounder. While it might be reasonable to additionally adjust for pubertal status, as outlined above, such data are rarely available in epidemiologic studies, and height might serve as a proxy for developmental status. It appears that Moreno et al. have made a computational error; as with the 13.0- to 13.9-year-old girls, the difference in body mass index before and after menarche among 14.0- to 14.9-year-old girls also has \( p < 0.001 \).

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RE: “ALCOHOL CONSUMPTION AND CORONARY HEART DISEASE MORBIDITY AND MORTALITY”

In their recently published paper, Rehm et al. (1) reported evidence of a relation between alcohol consumption and coronary heart disease, that is, incidence and mortality rates among their study subjects. They found a U-shaped association between consumption and both incidence and mortality in females and mortality only in males. There was, in fact, no upturn in risk at higher intake for the incidence of coronary heart disease in males. These are important findings in terms of prevention of heart disease. There are, however, some conceptual and methodological limitations in their study that may affect the conclusions.

The first two issues are related to the assessment of alcohol consumption. 1) The authors categorized alcohol consumption into lifetime abstainers, current abstainers, and current drinkers (from <2 drinks/week to >42 drinks/week). The National Health and Nutrition Examination Survey (NHANES) questionnaire (2) does not allow for the examination of past alcohol consumption patterns among current abstainers. Those exdrinkers could be mainly composed of ex-heavy drinkers. The group “current abstainers” should be separated into “used to be moderate to heavy drinkers” or any other classification assessing former drinking patterns. 2) The NHANES questionnaire permits only the assessment of drinking patterns in the past year, and it might take longer than 1 year to have symptomatic coronary heart disease (much more for death). Hence, questions assessing the former drinking behavior, in addition to current behavior, would provide a better measure of drinking patterns that relate to heart disease.

The last three comments pertain to methodological and conceptualization issues. 1) Preexisting diseases are important determinants (3, 4) that should be considered as a covariate in this study. Other medical conditions, such as hypertension, are associated with alcohol consumption and coronary heart disease. Relative rate estimates that are not adjusted for those medical backgrounds may be biased. 2) The authors reported that relative rates of different categories were adjusted first for smoking and age and second (data reported to be shown) for all other covariates (age, smoking, body mass index, physical activity, and aspirin use). Nevertheless, in table 3 they are adjusting for age only. In table 4, relative rates are adjusted only for age and smoking. Were these omissions purposeful or oversights on the part of the authors? For the sake of clarity, relative rates, adjusted for all covariates, should be presented in the tables. 3) Finally, are the distributions of covariate and drinking patterns by sex in tables 1 and 2 weighted or unweighted?

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Sarr (1) has pointed out potential limitations in our recently published paper (2). We would like to take the opportunity to respond to his comments and to discuss some more general points about the methodology of alcohol epidemiology.

The main results of Rehm et al. (2) confirm the beneficial effect of moderate alcohol consumption on coronary heart disease morbidity and mortality, but we also found for females an increased risk of coronary heart disease associated with drinking more than four drinks a day relative to abstainers. To avoid misinterpretation of the results due to the "sick quitter" hypotheses (3, 4), all drinking categories were compared with lifetime abstainers. Current abstainers were treated separately but not further subdivided as suggested by Sarr (1). Such a subdivision could not have affected the main results in any way, however. The main thrust of the article was on the effects of moderate consumption, and lifetime abstention is the best baseline for these effects.

A second point referred to the assessment of lifetime drinking behavior in addition to drinking status of the past year. The recent literature in alcohol epidemiology has been discussing the need to develop lifetime measures extensively (5, 6), but new measures to be included in epidemiologic studies have only been developed recently (6). Thus, we have to wait to see the results from cohort studies using such measures. In the meantime, methodological research indicates that for the age cohorts used in Rehm et al. (2), the correlations between total lifetime consumption and consumption reported at different age points are reasonable indicators of lifetime consumption (average correlation above 0.7; see Lemmens et al. (5)).

Rehm et al. (2) showed results adjusted for age, smoking status, physical activity, body mass index, and aspirin use (see table 3). We did not adjust for hypertension as this variable is considered a causal pathway between alcohol and coronary heart disease (7). Methodologically, adjusting for variables in the causal pathway leads to biased estimates of relative risk for the determining factor, in this case alcohol consumption (8). In studies where the demonstration of such pathways is the explicit aim (7), models with and without the inclusion of hypertension should be compared. However, this was not the main goal of our study. In terms of preexisting diseases, we excluded all persons who reported a history of heart disease at baseline.

Finally, the issue of weighting was raised by Sarr (1). All our analyses are unweighted, following the tradition of Groves (9) who distinguishes between studies testing causal hypotheses and describing populations. As our study clearly belongs to the former category, analyses were conducted without weighting.

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