Although we have made great strides in reducing the burden of cardiovascular disease over the past 50 years, blacks in the United States still face increased cardiovascular mortality, along with a disproportionate burden of risk factors. However, if race-based comparisons become the basis for action, it is essential that they be measured and interpreted accurately. Blacks experience higher mortality rates of noncardiovascular disease (non-CVD). Some have argued that without accounting for non-CVD related deaths among blacks, epidemiologists may be overestimating the true comparative rate of incident cardiovascular events among blacks. In other words, by failing to mind the competition, we may be overestimating the race-based gap in CVD.

### Outcomes Being Compared

A competing risks model may be best suited in a comparison of outcomes that have similar implications for practice and policy. Non-CVD deaths may not have similar implications as a composite of fatal and nonfatal cardiovascular events. Furthermore, CVD death was parcelled among multiple different categories of outcomes, fatal and nonfatal cardiovascular event subtypes, and other CVD death, precluding direct cause-of-death comparisons. We might surmise that, despite some overlap, for the most part, preventive interventions may differ for cardiovascular and noncardiovascular causes of death.

### Differences Among the Cohorts

Although the cohort studies the authors used offer several strengths, large numbers of well-characterized populations...
without CVD at baseline, there are important differences, limiting comparisons across them.

The event rates for non-CVD death and cardiovascular events differ across the cohorts with lower rates being present in the younger MESA group (1.3% versus 4.7%, respectively) and much higher rates in the older MESA group (4.8% and 12.1%, respectively) and CHS cohort (15.7% versus 43.2%, respectively). Additionally, length of follow-up was much shorter in MESA than in ARIC and CHS. Thus, perhaps the results represent 3 different analyses in 3 different populations.

Comparisons between the old and the young may not be valid because they come from different cohorts (with the exception of MESA in which the group is divided by age presumably for ease of comparison). When one examines the birth dates among the cohorts, the oldest and youngest participants in CHS were born in 1888 and 1945, respectively, in 1923 and 1942 for ARIC, and in 1916 and 1955 for MESA. There may be important, unaccounted for birth cohort effects that impacted the nature of the competing risks from non-CVD death. The older cohort in CHS represents a population that has survived to 65 years of age, raising concerns about a survival bias that may affect blacks and whites differently. Blacks who survive to 65 years of age may have social, economic, and health characteristics that support their longevity in comparison with blacks who die earlier in life.

There are other possible cohort-based effects, including differences in secular trends in risk factor management. Racial groups within a cohort could experience secular trends differently because of variations in access to care, location of care, achievement of treatment goals (eg, hypertension control), and differential impact of emerging risk factors (such as obesity). Other explanatory factors beyond survival bias could account for the findings, particularly in CHS in which there were no statistically significant differences by race in first events for this older cohort. The CHS observations may be confounded by the fact that the cohort was older at inception and likely experienced a different approach to risk factor management for the years preceding their enrollment in the cohort. If both blacks and whites experienced comparable less aggressive risk factor management than what is delivered today, racial differences may not have been apparent. On the other hand, higher risk factor burden among blacks with known less aggressive risk factor management could contribute to race-based differences seen in ARIC.

Generalizability

The relatively small number of black participants in CHS and MESA limited the ability of the authors to produce precise and reliable estimates of the association between race and cardiovascular events. Furthermore, these black participants may not properly represent the greater black population of the United States. The cohort studies collected data from a small number of specific sites in the United States where competing non-CVD deaths could be either much higher (because of violence or accidents, for example) or much lower than in communities where most black Americans live.

Conclusions

Despite these limitations and the complexity of the report by Feinstein et al, I hope that one key message does not get lost in interpretation. Whereas race, per se, was not a strong independent predictor of cardiovascular risk after accounting for competing risks, socioeconomic factors, and classical risk factors, the authors described important disparities that have important policy implications. Blacks experienced higher burdens of baseline risk factors (in particular, hypertension and diabetes) and greater socioeconomic disadvantage. Blacks, in particular, black women, experienced greater comparative risks for non-CVD death and cardiovascular events. Classic risk factors, risk factors for which we have evidence-based preventive interventions, were stronger correlates for racial differences than socioeconomic characteristics. Risk factor burdens appear particularly important for hypertension and diabetes mellitus, which occur at a younger age with longer duration among blacks. The authors’ analyses suggest that racial differences are a reflection of the differing predisposition to risk factors and, to a lesser extent, greater socioeconomic challenges. In these analyses, race may serve as a construct representing the confluence of these issues.

What is most compelling about a competing risks analysis is the idea that we may be able to identify events that occur earliest as specific targets for prevention. Thus, if we know that differences in cardiovascular outcomes exist by race, then understanding whether this difference accounts for the competing risk of non-CVD death could be less important if it does not offer an opportunity to alter an outcome. The strong relationship between risk factor burden and cardiovascular events creates actionable targets, because aggressive action to control risk factors has the potential to alter mortality. If we want to close the gap in racial differences in cardiovascular events, then we need to focus our research, practice, and policy efforts on risk factor reduction and management.

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