suggest that we adjust observed estimates using standardized high-risk RS rates. This would indeed produce relative rates that reflect the true population-level difference. Although the cancer registries from which women were sampled are population based, and past investigations have demonstrated that SEER cancer-specific mortality rates for White and Black women with breast cancer were similar to national rates, it is possible that our estimates differ from the underlying populations of interest. \(^3\)

We agree that a population-standardized estimate, if available, would be ideal. However, to the extent that we demonstrated an independent association with high RS by race, we believe that our findings provide valid evidence that Black women disproportionately develop biologically aggressive, estrogen receptor-positive tumors.

Long et al suggest using propensity score matching to account for the imbalance of prognostic factors between Black and White women rather than the multivariable regression approaches we used. This method, which is often used in observational studies that compare 2 treatment groups, may be preferred over conventional multivariable regression methods for reducing bias when many measured covariates and confounders exist and there are few exposed outcomes. \(^4\) Our study included a limited number of measured confounders for adjustment. In this case, matching patients from different racial/ethnic groups on a scalar propensity score value would result in substantial attrition of patients and loss of data in a research context that is already hampered by small numbers of events among the racial/ethnic minority patients in the data set. It is speculative to suggest that propensity score matching would materially change the findings of our study. Regardless, the principal limitation of all adjustment methods is that they do not account for unmeasured factors, including the wide-ranging manifestations of structural racism underlying health disparities.

We thank Obeng-Gyasi and Carlos for highlighting the central role of social determinants of health in cancer disparities and for pointing out that race is a social construct. It is important to add that socioeconomic forces can also influence biology; racial differences in prognostic gene expression assays should not be interpreted as implying that ancestry-related genetic variants are necessarily the mechanism driving disproportionately aggressive biology in tumors in Black women. We wholeheartedly agree that more work is needed to unravel the complex interactions between social determinants of health, tumor biology, and racial disparities in breast cancer mortality. Space constraints prevented us from addressing that question in our article,\(^1\) but we hope to publish findings from a mediation analysis with socioeconomic measures available in the SEER Oncotype DX test data set in the near future. Taken in the context of its limitations, we hope that our study generates new hypotheses and advances research to uncover the multilevel mechanisms driving racial disparities in breast cancer mortality.

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CORRECTION

Error in Author Name: The Original Investigation titled “Efficacy of Reduced-Intensity Chemotherapy With Oxaliplatin and Capecitabine on Quality of Life and Cancer Control Among Older and Frail Patients With Advanced Gastroesophageal Cancer: The GO2 Phase 3 Randomized Clinical Trial” published online May 13, 2021, included an error in the name of one author. The author’s name is Konstantinos Velios Kamposioras. This article has been corrected online.


Error in a Supplement: In the article titled “Association of Convalescent Plasma Therapy With Survival in Patients With Hematologic Cancers and COVID-19,” published online June 17, 2021, and also in the August 2021 issue of JAMA Oncology, \(^1\) there was a formatting error in Supplement 2 listing the COVID-19 and Cancer Consortium members. The supplement was corrected online.