population. Similarly, in Utah, this subgroup accounted for 5.0% of the hospitalizations in contrast with 0.9% representation of the state population.

Discussion | This analysis identified considerable disparities in the prevalence of COVID-19 across racial/ethnic subgroups of the population in 12 US states. These findings are consistent with an earlier Centers for Disease Control and Prevention analysis of 580 hospitalizations between March 1 and March 30, 2020, that found disproportionately high COVID-19 hospitalizations for the Black population.6 Similarly, a study of 1052 confirmed COVID-19 cases between January 1 and April 8, 2020, at a California health system reported higher odds of hospitalization in non-Hispanic Black individuals compared with non-Hispanic White individuals.4 In addition, we observed high hospitalization rates for Hispanic individuals in most of the states analyzed and high hospitalization rates for American Indian and Alaskan Native populations in select states.

These findings highlight the need for increased data reporting and consistency within and across all states. Only 12 of 50 US states have consistently reported hospitalizations by race/ethnicity during our study period. New Jersey and Florida recently started reporting data on COVID-19 hospitalizations by race/ethnicity. The present study is limited in that there was no adjustment for age, sex, comorbidities, and socioeconomic factors within each racial/ethnic group that are likely to be associated with COVID-19 hospitalizations.

A large body of research has identified racial/ethnic health disparities in the risk of infection associated with a higher prevalence of comorbidities, less access to health care, adverse economic conditions, and service-related occupations.2 The unique clinical, financial, and social implications of COVID-19 for racial/ethnic populations that are often systematically marginalized in society must be well understood to design and establish effective and equitable infrastructure solutions.

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Disparities in Secondhand Smoke Exposure in the United States: National Health and Nutrition Examination Survey 2011-2018

Secondhand smoke exposure (SHSe) is one of the causes of sudden infant death syndrome, respiratory tract infections, ear infections, and asthma attacks in infants and children; coronary heart disease, stroke, and lung cancer in adult non-smokers; and low birth weight, premature deliveries, and congenital defects in pregnancies.1 It results in nearly 42,000 deaths (more than 41,000 adults and 900 infants) among non-smokers every year in the US, with Black individuals accounting for 24% to 36% of the infant deaths.2 The US Surgeon General determined that there is no risk-free level of SHSe.3 With the outbreak of coronavirus disease 2019, which affects lung function, improving smoke-free policies to enhance air quality should be a growing priority.
This study was conducted using data from 4 cycles of the National Health and Nutrition Examination Survey (NHANES) from 2011 to 2018. The most recent data (2017-2018) were released in March 2020. The NHANES is designed to monitor nationwide health and uses a complex, multi-stage probability design to sample the non-institutionalized civilian population residing in all 50 states. Sensitive and personal questions are administered through computer-assisted personal interview and the audio computer-assisted self-interview. The biological specimens for laboratory testing were collected at a mobile examination center.

Among nonsmokers, individuals having serum cotinine levels of 0.05 to 10 ng/mL were considered to have SHSe. The primary outcome of this study is the prevalence of SHSe in nonsmokers in the US population 3 years and older. Because the NHANES data are deidentified and publicly available, this secondary data analysis was exempt from institutional review board approval and informed consent in accordance with the Common Rule and University of Texas MD Anderson Cancer Center policy.

The survey-adjusted weights were used to estimate the prevalence of SHSe in nonsmokers. A survey logistic regression was used to test for trends in SHSe over the 4 two-year intervals (2011-2012, 2013-2014, 2015-2016, and 2017-2018) of NHANES. Also, a multivariable survey logistic regression was performed to identify factors associated with SHSe. For all statistical analyses, P values were calculated using Wald test statistic, and significance was defined as 2-sided P value ≤ .05.

The analysis was performed using the survey package in R, version 4.0.0 (R Foundation for Statistical Computing).

**Results**

This study found that disparities in SHSe among non-Hispanic Black individuals compared with people of other races and among those below the poverty levels have persisted throughout 2011 to 2018 (Table 1). For example, in the 2017-2018 cycle, the SHSe prevalence continued to be twice as high among nonsmoker non-Hispanic Black individuals (48.02%) compared with non-Hispanic White individuals (22.03%) and among those living below the poverty level (44.68%) compared with those living above the poverty level (21.33%). Similarly, children aged 3 to 11 years continued to experience SHSe at high rates (38.23%). Multivariable logistic regression identified younger age (odds ratio [OR], 1.88, for 12-19 years, and OR, 2.29, for 3-11 years), non-Hispanic Black race/ethnicity (OR, 2.75), less than high school education (OR, 1.59), and living below the poverty level (OR, 2.61) as risk factors for SHSe in the 2017 to 2018 cycle, with little change across all data cycles (Table 2).

**Discussion**

Although the prevalence of SHSe among nonsmokers in the US declined substantially (87.5% to 25.3%) from 1988 to 2012, progress has stagnated since then, with persisting racial and economic disparities. Populations with a lower socioeconomic status have higher smoking rates, lower knowledge about health risks of tobacco, higher risk of workplace exposure.
exposure, and higher likelihood of living in low-income multi-unit housing and have their communities targeted more by tobacco companies, which would possibly explain the high SHSe observed in our study. Furthermore, in households with smokers, non-Hispanic Black individuals are less likely to have a complete smoking ban in homes, while parents of any race or ethnicity with a child on Medicaid or uninsured (a proxy for lower income) are less likely to have a complete smoking ban in family vehicles. We conclude that more needs to be done to implement enhanced and equitable comprehensive smoke-free laws throughout the US (currently implemented in only 27 states). The serum cotinine levels in nonsmokers provide a measure of overall SHSe, regardless of the sources or locations of exposure; therefore, these laws should be expanded to include other forms of vaping and should include private properties (eg, cars) for meaningful reduction in SHSe among vulnerable populations (eg, young children). The primary limitation of the study is the potential nonresponse bias in the NHANES data collection, although our study is survey weighted to account for the nonresponse.

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Table 2. Multivariable Logistic Regression Results Showing Adjusted Odds Ratios (ORs) of Nonsmokers Having Serum Cotinine Levels 0.05 to 10 ng/mL—National Health and Nutrition Examination Survey, 2011-2018

| Variable                | 2011-2012 OR (95% CI) | P value | 2013-2014 OR (95% CI) | P value | 2015-2016 OR (95% CI) | P value | 2017-2018 OR (95% CI) | P value |
|-------------------------|-----------------------|---------|-----------------------|---------|-----------------------|---------|-----------------------|---------|
| Sex                     |                       |         |                       |         |                       |         |                       |         |
| Male                    | 1.37 (1.22-1.54)      | .001    | 1.30 (1.11-1.51)      | .02     | 0.94 (0.79-1.12)      | .52     | 1.22 (0.96-1.55)      | .15     |
| Female                  | 1 [Reference]         |         | 1 [Reference]         |         | 1 [Reference]         |         | 1 [Reference]         |         |
| Age                     |                       |         |                       |         |                       |         |                       |         |
| 3-11 y                  | 2.32 (1.76-3.05)      | .001    | 1.84 (1.52-2.23)      | .002    | 2.22 (1.84-2.69)      | <.001   | 2.29 (1.81-2.90)      | <.001   |
| 12-19 y                 | 1.77 (1.37-2.30)      | .004    | 1.68 (1.33-2.13)      | .007    | 1.88 (1.48-2.38)      | .004    | 1.88 (1.43-2.48)      | .004    |
| ≥20 y                   | 1                     | [Reference] | 1                     | [Reference] | 1                     | [Reference] | 1                     | [Reference] |
| Race/ethnicity          |                       |         |                       |         |                       |         |                       |         |
| Mexican American        | 0.6 (0.40-0.91)       | .046    | 0.49 (0.31-0.76)      | .03     | 0.52 (0.38-0.71)      | .01     | 0.48 (0.33-0.68)      | .006    |
| Other Hispanic          | 0.57 (0.40-0.81)      | .02     | 0.71 (0.42-1.19)      | .25     | 0.57 (0.40-0.82)      | .03     | 0.73 (0.48-1.10)      | .18     |
| Non-Hispanic            | 1 [Reference]         |         | 1 [Reference]         |         | 1 [Reference]         |         | 1 [Reference]         |         |
| White                   | 2.23 (1.64-3.01)      | .001    | 2.62 (1.87-3.66)      | .002    | 2.08 (1.51-2.86)      | .007    | 2.75 (2.12-3.58)      | <.001   |
| Black                   | 1.57 (1.16-2.13)      | .02     | 1.69 (1.28-2.24)      | .01     | 1.36 (0.93-2.00)      | .18     | 1.59 (1.19-2.12)      | .02     |
| Education               |                       |         |                       |         |                       |         |                       |         |
| Less than high school   | 2.06 (1.74-2.43)      | <.001   | 2.50 (2.04-3.07)      | <.001   | 1.97 (1.57-2.47)      | .002    | 2.61 (2.04-3.35)      | <.001   |
| High school or above    | 1 [Reference]         |         | 1 [Reference]         |         | 1 [Reference]         |         | 1 [Reference]         |         |
| Poverty level           |                       |         |                       |         |                       |         |                       |         |
| Above                   | 2.03 (1.62-2.55)      | <.001   | 1.85 (1.48-2.30)      | .003    | 1.98 (1.51-2.59)      | .004    | Data not released     |         |
| Below                   |                       |         |                       |         |                       |         |                       |         |
| Housing                 |                       |         |                       |         |                       |         |                       |         |
| Owned or being bought   | 1 [Reference]         |         | 1 [Reference]         |         | 1 [Reference]         |         | 1 [Reference]         |         |
| Rented                  | 2.01 (1.62-2.55)      | <.001   | 1.85 (1.48-2.30)      | .003    | 1.98 (1.51-2.59)      | .004    | Data not released     |         |

a P values presented are based on the 2-sided Wald test.
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**COMMENT & RESPONSE**

**The Assessment of Different Diets and Mortality Fails to Address Unmeasured Confounding**

To the Editor Shan et al1 investigated the relationship between low-carbohydrate and low-fat diets and mortality, finding that the association of such diets with mortality may depend on the quality and source of these macronutrients.1 The authors are careful to point out that their results do not indicate causality, instead suggesting that the quality of macronutrients in terms of fiber, vitamins and minerals, and phytochemicals may be more important than the quantity of macronutrients in affecting mortality.

While the authors acknowledge these and other limitations, such as 24-hour recall to assess diet, thus subjecting the analysis to recall bias and inability to assess any changes in diet, there are additional limitations that should be considered, particularly as this is an observational study rather than a clinical trial. Alcohol and tobacco use were assessed in binary terms (≥12 drinks in one year and ≥100 cigarettes over a lifetime), but such stratification significantly limits and artificially constrains variability for these factors—eg, an individual who has 1 drink per month would be classified in the same category as someone who drinks daily, yet a heavier drinker may have worse outcomes; similarly with tobacco use.

The association of diet with health is an area of significant research and public health interest, as indicated by daily news reports and journal publications on this topic. Yet, nearly all such analyses fail to account for unmeasured confounders adequately. One way to address such unmeasured confounding is through an E-value analysis, which indicates the “minimum strength of association, on the risk ratio scale, that an unmeasured confounder would need to have with both the treatment and outcome ... to fully explain away a specific treatment-outcome association.”2 This analysis would add significant insight to qualify the findings of this study. We calculated E-values for the main effects reported in Tables 2 and 3 of the study (Tables 1 and 2), finding that unmeasured confounders could easily account for the diet/mortality results. Most E-value lower confidence bounds were close to 1 (suggesting no dietary benefit after unobserved confounder adjustment). No E-value confidence bound exceeded 1.74, indicating that unmeasured confounding could easily explain the findings. Such confounders could include mental health, stress, health-promoting behaviors, and social interactions. As we...

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**Table 1. E-values for Associations Between Low-Carbohydrate-Diet Scores and Total Mortality**

| Characteristic | Quintiles of low-carbohydrate-diet scores | P for trend | Per 20-percentile increase |
|---------------|------------------------------------------|------------|---------------------------|
|               | Quintile 1 | Quintile 2 | Quintile 3 | Quintile 4 | Quintile 5 |          |                    |
| Overall low-carbohydrate-diet score |  |  |  |  |  |  |  |
| E-value (CI bound), age- and sex-adjusted | 1 [Reference] | 1 (1) | 1.29 (1) | 1 (1) | 1.16 (1) | NA | 1.11 (1) |
| E-value (CI bound), multivariable-adjusted | 1 [Reference] | 1.29 (1) | 1.53 (1) | 1.46 (1) | 1.53 (1) | NA | 1.21 (1) |
| Unhealthy low-carbohydrate-diet score |  |  |  |  |  |  |  |
| E-value (CI bound), age- and sex-adjusted | 1 [Reference] | 1.54 (1.11) | 1.64 (1.21) | 2.04 (1.59) | 2.06 (1.62) | NA | 1.51 (1.37) |
| E-value (CI bound), multivariable-adjusted | 1 [Reference] | 1.34 (1) | 1.34 (1) | 1.64 (1.21) | 1.59 (1) | NA | 1.34 (1.16) |
| Healthy low-carbohydrate-diet score |  |  |  |  |  |  |  |
| E-value (CI bound), age- and sex-adjusted | 1 [Reference] | 1.85 (1.39) | 1.7 (1.16) | 1.96 (1.5) | 2.04 (1.63) | NA | 1.39 (1.25) |
| E-value (CI bound), multivariable-adjusted | 1 [Reference] | 1.7 (1.25) | 1.6 (1) | 2.08 (1.67) | 2.08 (1.63) | NA | 1.43 (1.29) |

Abbreviation: NA, not applicable.

E-values for each model (age-adjusted, sex-adjusted, and multivariable-adjusted) are shown for each quintile and 20-percentile increase in diet score. Only scores in quintile 5 indicate slightly less unmeasured confounding; however, the effects are weak, as no E-value CI bound crosses 1.63.

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1 Adjusted for age (20-34, 35-49, 50-64, and ≥65 years), sex (male or female), race/ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, and other), educational level (less than high school, high school graduate or General Educational Development, and some college or above), ratio of family income to poverty (<1.30, 1.30-3.49, or ≥3.5), family history of diabetes (yes or no), family history of heart disease (yes or no), history of diabetes (yes or no), history of heart disease (yes or no), history of cancer (yes or no), physical activity (0, 0.1-0.9, 1.0-3.4, 3.5-5.9, or ≥6 hours per week), alcohol consumption (0, 0.1-4.9, 5-14.9, 15-29.9, or ≥30 g/d), smoking status (never smoker, former smoker, or current smoker [1-14, 15-24, or ≥25 cigarettes per day]), cholesterol (quintiles), and body mass index (calculated as weight in kilograms divided by height in meters squared) (<21, 21-24.9, 25-29.9, 30-35, and ≥35).

2 Low-high-quality carbohydrate and high animal protein and unsaturated fat.

3 Low-high-quality carbohydrate and high plant protein and unsaturated fat.