mortality in the United States (1) and share the goal of improving our understanding of the burden of HP and increasing our awareness of this disease to improve diagnosis and treatment.

HP is assuredly a complex, multifaceted lung disease, involving both genetic and environmental risk factors. We agree that the racial differences observed in our study are not solely due to genetic factors. As noted in our Discussion, the contribution of specific exposures is an important but unmeasured variable in our study, and region-specific variations in HP mortality are almost certainly affected by differences in occupational exposure across states. However, regardless of ethnicity/race confounding bias by workplace exposure, genetic variation (which can be related to and differ by race) is likely one of the many risk factors that act together to cause disease and should be considered when interpreting epidemiological data. For example, significant ethnic/racial differences have been described in idiopathic pulmonary fibrosis that may be related to genetic differences (2), and differences in specific gene polymorphisms associated with susceptibility to HP have been observed both between groups with different ethnic backgrounds and within ethnic/racial groups (3–5). Further work is needed to fully understand the role of genetic variation in the molecular and cellular processes, clinical characteristics, and outcomes of HP.

We certainly agree that occupational exposures are an important contributor to the overall burden of HP in the United States and that they should always be considered in both individual and population studies. Since the 1980s, various agricultural activities, including crop and animal farming, have been the most frequently recorded industries and occupations on death certificates for individuals with HP. However, when studying the role of occupation, an important limitation of the National Occupational Mortality Surveillance database is that less than half of the states provide data on the usual industry and occupation of decedents (limiting generalizability). Importantly, in contrast to work-related HP, nonoccupational HP cases are not reportable and therefore are likely undercounted and underrepresented in assessments of the relative contribution of occupational and nonoccupational exposures responsible for HP cases nationwide (6).

We hope that the insight gained from our study and the complementary data of Hall and colleagues will serve as a call to state health department officials and federal agencies to revise key policies regarding disease surveillance programs, risk management, and enforcement of exposure limits, particularly in the agricultural sectors, in an effort to reduce the impact of HP in the country. ■

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Are Rural Residence and Poverty Independent Risk Factors for Chronic Obstructive Pulmonary Disease in the United States?

To the Editor:

With great interest, I read the article by Raju and colleagues (1) in which they concluded that living in a rural community and poverty were significantly associated with the incidence of chronic

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obstructive pulmonary disease among nonsmokers and ever-smokers.

In their study, Raju and colleagues used multivariate models adjusted for age, sex, race/ethnicity, region of residence, smoking duration, community poverty, urban/rural status, census-level data on the use of solid fuels in homes and the percentage of inhabitants in occupations associated with lung disease, and individual socioeconomic factors. However, some important factors that may affect the incidence of chronic obstructive pulmonary disease were not described in detail and seemed to be ignored in the study, such as FEV_1 (2) and the body mass index (3). It is quite possible that if the authors had adjusted for these factors in their multivariate models, they would have drawn a different conclusion. This issue should be resolved in future studies. ■

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Reply to Wei

From the Authors:

We thank Dr. Wei for his letter and interest in our recent publication (1). Dr. Wei brings attention to the lack of lung function data in the National Health Interview Survey data used for our study. We noted in our paper that the lack of lung function data, including FEV_1, was a limitation of our study. This limitation was partially countered by the large-scale nature of the National Health Interview Survey, which allowed us to uniquely examine the community and individual level factors that contribute to chronic obstructive pulmonary disease (COPD) development across the United States. We were additionally able to perform multiple sensitivity analyses within this data set, using differing definitions of COPD, demonstrating that rural residence remained independently associated with COPD. Ultimately, although confident in our study, we do agree that there should be efforts to validate these results in a nationally representative sample that includes lung function data. The second point Dr. Wei raises is in regard to whether body mass index and obesity may be confounders. Although not included in the final paper, an earlier version of our analysis accounted for obesity, which ultimately did not change the association between rural residence and COPD prevalence. Current efforts are underway to better understand the contribution of obesity and diet to COPD morbidity in rural regions of the United States. Last, the articles that Dr. Wei references describe the importance of better understanding factors that may contribute to lung development and early lung function impairment. Although this was beyond the scope of our cross-sectional analysis, we have great interest in future efforts to study factors in rural, poor regions, including nutrition and environmental exposures, which may influence lung development and contribute to the observed urban–rural disparities in COPD prevalence (2–4). ■

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