Response to Letter Regarding Article, “Childhood Air Pollutant Exposure and Carotid Artery Intima–Media Thickness in Young Adults”

We thank Wang et al for their commentary regarding our recently published article relating childhood O3 exposure to carotid intima–media thickness (CIMT) in adulthood.1 The authors suggest that adjustment for additional clinical information may strengthen our conclusion regarding O3 as a novel predictor of CIMT. Specifically, they suggest we include data on childhood dietary patterns, adolescent blood pressure (BP), and glucose tolerance testing. Although we agree that these data are relevant clinical parameters when assessing cardiovascular disease risk and atherosclerosis, it is unlikely that they will act as confounders of our observed association with O3 if they are not related to exposure.

First, the evaluation of elementary and lifetime diet, as suggested by the authors, is a daunting task, and one highly susceptible to recall bias. Most food frequency questionnaires are designed to address recall only within the past 12 months, not over a period of 20 years spanning periods of major life changes. It also is important to note that effect estimates between subclinical cardiovascular disease markers and dietary patterns in the cited study by Nettleton et al2 were notably attenuated when models were adjusted for mechanistic variables such as C-reactive protein, lipids, blood pressure, insulin, and glucose. Because we adjusted for most of these same variables in our models of O3 and CIMT, we will have at least partially adjusted for some dietary influences. Although we recognize that diet is an important consideration for measurement of CIMT, in our study it will only affect our conclusions about O3 as a novel predictor of CIMT if diet is also associated with O3.

Second, evaluation of adolescent BP was not possible in our study, because we recruited participants when they were already in college. In our study population, cumulative windows of O3 exposure were not associated with current BP. In addition, inclusion or exclusion of current BP in our models did not alter our results, suggesting that it was not a mediator of negative health effects of O3. BP measured 8 to 10 years earlier during adolescence is not likely to alter our observed associations substantially because adolescent and adult BP are correlated.1 4

Third, the administration of an oral glucose tolerance test to 861 college students during their study visit was not feasible. We did, however, measure fasting glucose levels in serum collected during the study visit. In additional sensitivity analyses, we included adjustment for glucose level in the models evaluating O3 and CIMT and observed no change in effect estimates. Moreover, childhood and lifetime O3 exposures were not associated with glucose level, suggesting that glucose is neither a confounder nor an intermediate in the association.

In conclusion, the atherogenic process has many important determinants, and we provide new evidence that childhood O3 exposure may also be important.

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Disclosures
None.

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