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. 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 al 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. It is unlikely that diet and O₃ are directly linked although geographic differences in both diet and air pollutants might drive some underlying correlations. To this end, we explored whether geographic location modified our associations but found no evidence of such modification.

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.

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.

References

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