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Ambient Air Pollution and Depressive Symptoms in Older Adults: Wellenius et al. Respond

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Ambient Air Pollution and Depressive Symptoms in Older Adults

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Wang et al. (2014) investigated the relationship between ambient air pollution and depressive symptoms in a prospective study of elderly people over age 65 years. We are very surprised that the authors found no association between ambient air pollution and depressive symptoms, which is inconsistent with most previous studies (e.g., Banerjee et al. 2012; Calderón-Garcidueñas et al. 2014). A study of a population of elderly men indicated one potential biological mechanism may be methylation, which was decreased after acute exposure to fine particulate matter (Madrigano et al. 2012).

We identified three issues with the study by Wang et al. (2014). First, these authors used outdoor air pollution as the exposure of interest. Long-term exposure was estimated based on residential distance from the nearest major road. Considering only residential distance to the nearest major roadway, without taking into account other sources of exposure, may be insufficient to accurately estimate long-term exposure to traffic pollution. Short-term exposure to ambient air pollution was estimated based on pollutant levels measured at only one monitoring site, the Harvard–U.S. Environmental Protection Agency Supersite stationary ambient monitoring site. Although the monitoring site was located < 20 km from the home of any study participant, the number of monitoring sites was insufficient. Thus, participants' exposures to ambient pollutant might be misclassified. Moreover, considering that the participants were elderly, they were less likely than other groups to spend time outdoors (Kerr et al. 2012). Hence, outdoor air pollution might not reflect their true exposures. Lacking validation of these estimated exposures, the negative conclusion is unconvincing.

Second, the Revised Center for Epidemiologic Studies Depression Scale (Eaton et al. 2004) is not the first choice for measuring depressive symptoms in the elderly; the Geriatric Depression Scale (Yesavage et al. 1982) is more suitable. The evidence from clinical diagnosis of depression shows a positive association between air pollution and depression. For example, Cho et al. (2014) found that ambient air pollution was positively associated with emergency department visits for depressive episodes among 4,985 Koreans using clinical diagnosis of depression episode as outcome. Of

note, clinical diagnosis of depression is a more objective measure than depressive symptoms.

Third, Wang et al. did not address health status in their reported association. There is convincing evidence supporting the association between cardiovascular disease (CVD) and both ambient air pollution (e.g., Grahame and Schlesinger 2010) and depression (e.g., Sun et al. 2013). Hence, previous history of CVD could confound or modify the association between ambient air pollution and depressive symptoms. Stratified analysis according to health or CVD status is warranted. Without such analysis, the negative conclusion is not well supported.

The authors declare they have no actual or potential competing financial interests.

Yongqing Gao,[1] Tan Xu,[2,3] and Wenjie Sun[1,3]

[1]School of Food Science, Guangdong Pharmaceutical University, Zhongshan, Guangdong, China; [2]School of Public Health and Jiangsu Key Laboratory of Preventive and Translational Medicine for Geriatric Diseases, Medical College of Soochow University, Suzhou, Jiangsu, China; [3]School of Public Health and Tropical Medicine, Tulane University, New Orleans, Louisiana, USA
E-mail: sunwenjie2002@hotmail.com

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We thank Gao et al. (2015) for their interest in our recent publication examining the association between ambient air pollution and depressive symptoms in a cohort of Boston-area elderly participants (Wang et al. 2014).

Gao et al. (2015) suggest that the null results we found are due to excessive exposure measurement error stemming from the use of pollutant concentrations measured at a single stationary monitoring site. In this investigation we examined associations with short-term exposure to ambient fine particulate matter mass, sulfate, black carbon, and ultra-fine particles measured at the Harvard–U.S. Environmental Protection Agency Supersite, which is located < 20 km from the participants' homes. Particle measurements from this monitoring site have been shown to be strong proxies for personal exposure to particles of ambient origin (Brown et al. 2009) and have been used in hundreds of prior studies. Nonetheless, as we acknowledged in our article, exposure misclassification likely resulted in wider confidence intervals for our effect estimates, but is not expected to have biased our results (Zeger et al. 2000). We also failed to find evidence of an association with long-term exposure to traffic pollution based on both residential proximity to major roadways and residential black carbon levels predicted by a spatiotemporal model (Gryparis et al. 2009). Both of these exposure metrics have been used in a large number of air pollution health effects studies in the Greater Boston area, most of which have found associations with a large spectrum of health outcomes (Hart et al. 2014; Lue et al. 2013; Suglia et al. 2008; Wellenius et al. 2012; Wilker et al. 2013).

Gao et al. (2015) further suggest that our null results might be due to excessive misclassification of the outcome because we assessed the presence of depressive symptoms using the Revised Center for Epidemiological Studies Depression Scale (CESD-R) (Eaton et al. 2004) rather than using a scale specifically designed for the elderly or relying on clinical diagnoses of depression. The CESD-R has been validated in the general population (Van Dam and Earleywine 2011), and the Center for

Epidemiological Studies—Depression Scale, on which the CESD-R is based, has been validated (Radloff 1977) and used extensively to study depressive symptoms in the elderly. Nonetheless, psychometric properties clearly differ across instruments, and the use of different instruments certainly could contribute to the heterogeneity observed across studies. Moreover, as we discuss in our paper, the CESD-R assesses the presence of depressive symptoms within the preceding 2 weeks rather than depression episodes requiring clinical attention or the presence of clinically diagnosed depression.

In their letter, Gao et al. (2015) criticize our decision not to adjust for prevalent cardiovascular disease (CVD) in our primary analyses, suggesting that we should have adjusted for it because *1)* air pollution is believed to cause CVD, and *2)* there is a well-documented association between depression and CVD. However, CVD is a downstream consequence of exposure to air pollution, and therefore, adjusting for it in analyses of air pollution health effects requires caution and appropriate caveats (Hernan et al. 2002). Nonetheless, in our paper we presented sensitivity analyses that were additionally adjusted for body mass index, physical activity, alcohol consumption, smoking, diabetes mellitus, hypertension, and hyperlipidermia, and showed that the results were not materially different. Thus, it does not seem likely that our null results are due to lack of adjustment for CVD or its risk factors.

Finally, Gao et al. (2015) suggest that our results stand in contrast to those of "most previous studies." This may be true, but it is worth noting that there are very few other studies available for direct comparison, and thus this remains very much an open research question. Additional studies in diverse populations are clearly needed to confirm or refute the presence of an association between air pollution and depressive symptoms.

The authors declare they have no actual or potential competing financial interests.

Gregory A. Wellenius,[1] Petros Koutrakis,[2] and Yi Wang[3]

[1]Department of Epidemiology, Brown University School of Public Health, Providence, Rhode Island, USA; [2]Department of Environmental Health, Harvard T.H. Chan School of Public Health, Boston, Massachusetts, USA; [3]Department of Environmental Health, Indiana University Richard M. Fairbanks School of Public Health, Indianapolis, Indiana, USA
E-mail: gregory_wellenius@brown.edu

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