

## A Short Note on Air Pollution and Plasma Amyloid Beta

Connor Julian\*

Department of Marine Ecology, Ocean University of China, China

### Abstract

Air pollution has been linked to Alzheimer's disease and other dementias that are related to it (ADRD), but the mechanisms that link ADRD to air pollution aren't clear. Amyloid beta (A) deposition in the brain may be exacerbated by oxidative stress, neuroinflammation, and air pollution. Using data from 3044 dementia-free Ginkgo Evaluation of Memory Study (GEMS) participants, we investigated the relationship between nitrogen dioxide (NO<sub>2</sub>), fine particulate matter less than 2.5 μm in diameter (PM<sub>2.5</sub>), and particulate matter less than 10 μm in diameter (PM<sub>10</sub>). One-, five-, 10-, and 20-year exposure averages were calculated for residential addresses with address histories that included addresses from 1980. A was measured at the beginning of the study (2000-2002) and again at the end of the study (2007-2008), enabling linear random effects models to evaluate repeated measures and linear regression models to evaluate cross-sectional associations.

**Keywords:** Air pollution; Environment

### Introduction

In cross-sectional analysis, we found no association between each air pollutant and A1-42 or the ratio measures after controlling for socio-demographic and behavioral covariates. We found stronger positive associations between each air pollutant and all three outcomes through repeat measures analysis. We saw an increase of 4.43 percent (95 percent CI: 3.26 percent, 5.60 percent), 9.73 percent (6.20%, 13.38 percent), and 1.57% (95 percent CI: a 2.20 percent (0.94%) higher A1-42/A1-40 ratio and a 2 g/m<sup>3</sup> higher 20-year average PM<sub>2.5</sub>. Similar relationships existed with other air pollutants. By evaluating longer air pollution exposure averaging periods to better mimic disease progression and providing a modifiable target for ADRD prevention, our study adds to the growing body of evidence on air pollution and ADRD biomarkers.

The world's population is getting older quickly. According to Nicholas et al., the global proportion of people 65 and older is expected to rise from 8% in 2010 to 16% by 2050. (2022). With the maturing populace, instances of dementia are likewise expected to rise, bringing about an expected 152.8 million cases, a significantly increasing, by 2050. There are no known treatments for Alzheimer's disease and other dementias (ADRD), but several risk factors can be changed. Bhatti et al., in addition to lifestyle factors like diet and exercise, ADRD has also been linked to environmental dangers like air pollution.

Dementia is caused by air pollution, but the exact mechanism has not yet been determined. According to a number of animal studies air pollution's effects on the central nervous system (CNS) have been linked to neuroinflammation and oxidative stress, two factors that play a crucial role in ADRD development. Air pollution may also increase amyloid beta (A) deposition in the brain, a hallmark of Alzheimer's disease (AD), by altering the activity of key enzymes. According to Patten et al.'s research on animals, animals exposed to traffic-related air pollution (TRAP) developed more A plaques over time than animals exposed to filtered air.

Although it has been debated whether treatments that reduce A plaques can improve cognitive function, understanding how air pollution affects brain health remains a significant undertaking. According to Peters et al., the peptide A-1-40, which has a 40-amino acid chain, has been linked to diabetes and cardiovascular disease. Other disease processes can also be better understood by studying A1-40 and air pollution. On the other hand, A1-42 is a crucial component

of brain a plaques [1-5].

### Discussion

Plasma is expected to contain A, in contrast to the presence of A plaques in the brain. According to Lopez et al., plasma A biomarkers change over time and can be affected by a variety of factors and have distinct outcomes for individuals with intact cognitive function and those with dementia. Plasma A levels rise with age in cognitively intact individuals, whereas plasma A biomarkers decrease over time in those who go from intact to MCI to AD. The blood-brain barrier allows A from the brain to travel to the blood and vice versa. In healthy people, the brain's soluble A could be cleared into the blood, resulting in higher plasma levels; however, in sick people, the brain's soluble A becomes insoluble A plaques, which the body is unable to clear, resulting in lower plasma levels.

In light of the fact that higher levels of pollution in the air have been shown to worsen outcomes, we would anticipate that higher levels of air pollution would be linked to lower plasma A levels in those who go on to develop dementia and higher plasma A levels in those who do not develop the disease. Plasma A levels that are higher may indicate that air pollution is accelerating plasma A levels, which may then lead to worse cognitive health.

Only a few studies have looked into the connection between air pollution and A in humans. Some of these studies have used PET scans or MRI to identify A, CSF (cerebrospinal fluid) from other sources. In general, the study finds a positive correlation between A and air pollution; The association is more consistent in studies that use imaging data. Even though these methods are effective at identifying A, they lack the ease, cost, and participant burden of blood biomarkers

\*Corresponding author: Connor Julian, Department of Marine Ecology, Ocean University of China, China, E-mail: julianconnor@edu.cn

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of AD. Plasma-based A biomarkers have not been the subject of any research, to our knowledge.

In the hope of developing a diagnostic test that will make it simpler to identify ADRD, researchers continue to look for highly sensitive methods of measuring A in the blood. These methods will enable a deeper comprehension of the mechanisms underlying the observed links between dementia and air pollution once they have been identified. Plasma-based A biomarkers and air pollution were the subjects of this cross-sectional and repeated-measures investigation in a group of dementia-free older adults.

In sensitivity analyses, we looked at different ways to make sure the outcome residuals were normal, specifically trimming (or excluding) the extreme outliers without changing the distribution. When outliers were specifically excluded in the repeat measures analysis, this did not significantly alter the results, but it did result in a slight attenuation of parameter estimates.

By only looking at those participants who provided samples to both the baseline and the 2007–2008 visit (i.e., those who were not censored or lost to follow-up), we also looked into the possibility of selection bias. In comparison to baseline, we observed stronger associations between all outcomes measured at follow-up and PM<sub>2.5</sub> and NO<sub>2</sub>. The PM<sub>10</sub> results did not change much. This suggests that participants who remained in the study until the end of the follow-up period were the driving force behind the results of our repeated measures analysis.

When compared to the cross-sectional results, our repeated measures analysis may have produced stronger associations for a number of different reasons. First, the larger sample size in the repeated measures analysis gives us more power to detect an effect. Because we are more certain about residential histories during follow-up and the several years prior to GEMS enrollment (e.g., approximately 1998 - 2008 for the 10 year averaging period) in comparison to residential histories prior to GEMS enrollment (e.g., approximately 1990 - 2000 for the 10 year averaging period), those in our repeated measures analysis [6-10].

## Conclusion

Furthermore, historical models that are based on less monitoring data and are therefore more prone to measurement error are used for exposure assessment further back in time. In the repeat measures analysis, estimates with larger magnitudes may be produced by both increased power and decreased exposure measurement error. Because plasma biomarker data fluctuates significantly and some disease-free participants experience decreases in A during follow-up, we chose to conduct a repeat measures analysis rather than one that evaluates change in A over time. This is to be expected given plasma A's dynamic nature (described above). In addition, the repeat measures approach enables us to have a sample that is more generalizable and has more power due to the loss of follow-up in our sample.

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