Chapter 6

Ambient Particulate Matter Air Pollution Exposure and Hypertension

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Additional information is available at the end of the chapter

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Abstract

The relationship between exposure to ambient particulate matter (PM) and hypertension in humans is controversial. The aim of the present meta-analysis was to quantify the predictability of ambient PM on the risk of incident hypertension in humans. The selection criteria included the studies that could provide quantitative estimates of the change in hypertension prevalence linked with exposure to either indicator of PM. The health outcome of "hypertension" was defined as systolic blood pressure (SBP) of 140 mmHg and greater and/or diastolic blood pressure (DBP) of 90 mmHg or greater and/or taking antihypertensive drugs or diagnosis by a physician or self-reported hypertension. A total of 647 studies were initially identified through online database searches, and finally five studies met the inclusion criteria. The combined results of reported relative risk from the five included studies revealed that ambient PM was positively associated with hypertension (OR = 1.03; 95% CI: 0.99–1.06), but this was not statistically significant. When stratified by the PM size, the results showed that the odds ratio for hypertension increased by 1.03 (95% CI: 0.93–1.14) per 10 μg/m^3 increase in $PM_{2.5}$, 1.04 (95% CI: 0.98–1.10) in PM_{10}, and 0.99 (95% CI: 0.92–1.07) in $PM_{2.5–10}$. In conclusion, to get more information about the associations between PM and hypertension, many studies need to be undertaken further to clarify these relationships.

Keywords: ambient particulate matter, hypertension, $PM_{2.5}$, PM_{10}, human

1. Introduction

Hypertension is the major risk factor for cardiovascular disease and other vascular diseases [1, 2] and renal disease [3, 4]. It has been considered as the most important cause of disability, and high blood pressure (BP; SBP > 110–115 mmHg) increased from the fourth-ranked

risk factor for burden of disease in 1990 to the leading risk factor in 2010 [5]. Previous studies have concluded that high BP and hypertension are associated with genetic factor, diet and lifestyle [6], and environmental factors which include ambient air pollution as a potential risk factor as well [7].

Ambient air pollution is a complex mixture containing particulate matter (PM). In academia, PM can be divided into four fractions on the basis of aerodynamic diameter of PM, including thoracic particles (<10μm, PM_{10}), coarse particles (2.5–10μm, $PM_{2.5–10}$), fine particles (<2.5μm, $PM_{2.5}$), and ultrafine particles (<0.1μm) [8]. There are mounting evidences, especially relating PM_{10} and $PM_{2.5}$, which suggest that those kinds of particles of different aerodynamic diameter have been linked with elevated BP [9–11]. And several studies have reported ambient PM and risk of hypertension [12–19], but until now, there are no comprehensive evidences about outdoor PM exposure and risk of hypertension.

The aim of this study was to provide synthesized evidence of the hypertension risk associated with exposure to PM in ambient air, specifically $PM_{2.5}$ and PM_{10} and DPM, through conducted meta-analysis.

2. Methods

2.1. Literature search

We developed search terms to identify associated papers by using the medical headings (MeSH) in PubMed. Meanwhile, studies were also identified through Web of Science (WOS) and Scopus using relevant search terms, which include terms related to the exposure (air pollution, PM, inhalable particle, fine particle, $PM_{2.5}$, PM_{10}) and the outcome (hypertension/high BP). The reference lists of the papers were searched for possible relevant articles. We conducted literature search at PubMed, Web of Science (WOS), and Scopus on 6 November 2015, 1 January 2016, and 11 January 2016, respectively. The limitation of language is English. Through this process, a total of 662 potentially relevant papers were searched.

2.2. Study selection

Studies were included in the current meta-analysis to know if they provided quantitative estimates of residential exposure to $PM_{2.5}$ and/or PM_{10} and other PM. Further, studies were required to provide quantitative estimates of the change in hypertension prevalence linked with exposure to either indicator of PM; this could be reported as the change in risk per microgram per cubic meter or per quantile of exposure. Studies that reported results for the association of hypertension with other air pollutants or exposure to traffic but did not provide quantitative estimates for PM were not included in the meta-analysis. The health outcome of "Hypertension" was defined as systolic blood pressure (SBP) of 140 mmHg and greater and/or diastolic blood pressure (DBP) of 90 mmHg or greater and/or taking anti-hypertensive drugs or diagnosis by a physician or self-reported hypertension.

Though multiple publications included overlapping study populations, we included the publication that considered the largest number of cases and/or that evaluated results based on the longest follow-up. In addition, we did not place any restrictions based on whether or not a study adjusted for specific confounders. All risk estimates were abstracted by one of the authors and double checked for accuracy by a second author.

2.3. Data synthesis and analysis

All study estimates were converted to represent the change in hypertension risk per 10 μg/m^3 unit increase in exposure to $PM_{2.5}$, PM_{10}, or other PM. If we could not reliably convert the values in a particular study to the aforementioned units, we contacted the authors of the original study for further information. If information necessary to convert estimates could not be obtained, the study was excluded from consideration.

Estimates from the studies were combined using a random-effects model, which allowed between-study heterogeneity to contribute to the variance [20]. I^2 values are reported, representing the estimated percent of the total variance that is explained by between-study heterogeneity [21]. We also conducted chi-square tests of homogeneity to compare meta-estimates divided into subgroups by pollutants ($PM_{2.5}$, $PM_{2.5–10}$, PM_{10}, DPM). Finally, forest and funnel plots were created to provide a visual summary of the distribution of study-specific effect estimates. Instead of statistical tests of funnel plot asymmetry, we conducted trim and fill analyses. All analyses were conducted using STATA (v12.1; StataCorp, College Station, TX, USA). Noted $P < 0.05$ (two-tailed) was considered statistically significant except for the heterogeneity test, for which $P < 0.10$ (one-tailed) was used.

3. Results

3.1. Literature search and study characteristics

The detailed steps of our literature search are shown in **Figure 1**. A total of 647 citations were screened during the primary search. According to the title and abstract of every citation and included and excluded criteria, we identified 11 papers. After a detailed evaluation, two papers [18, 22] were excluded due to lack of data and two papers [17, 23] were excluded since the target population of the two studies comprises black women in the USA. Finally, the remaining five studies [12, 13, 15, 16, 19] published between 2009 and 2015 were included in the present meta-analysis. Of these, five cross-sectional studies were included. Two studies were conducted in the USA [15, 19], two in China [13, 16], and one in Germany [12].

3.2. Combined estimates of ambient particulate matter and the risk of hypertension

The effect estimates of individual studies and the combined effect estimates for hypertensive associated with PM, including $PM_{2.5}$, $PM_{2.5–10}$, PM_{10} and DPM, are presented in **Figure 2**. Hypertension among study population was not statistically significantly associated with a 10 μg/m^3 increment in PM (OR = 1.03; 95% CI: 0.99–1.06).

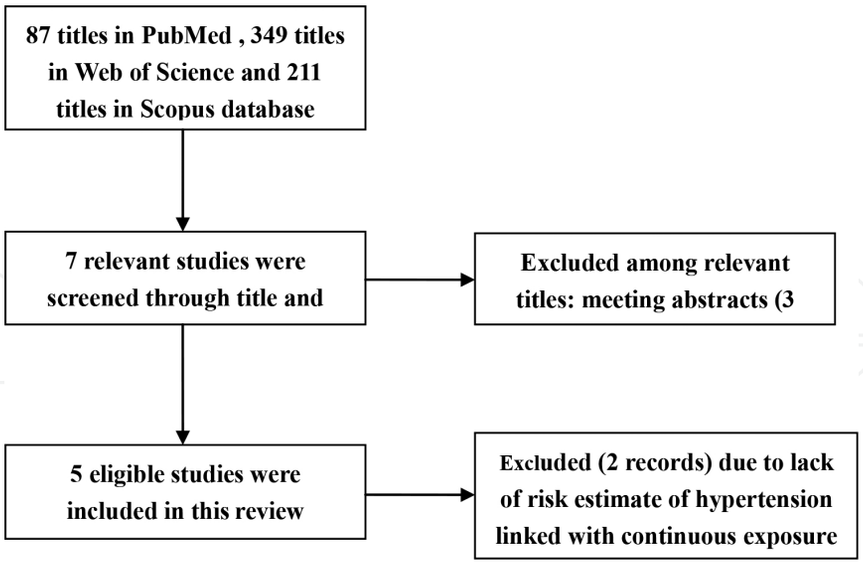

Figure 1. Flow diagram of screened, included and excluded publications.

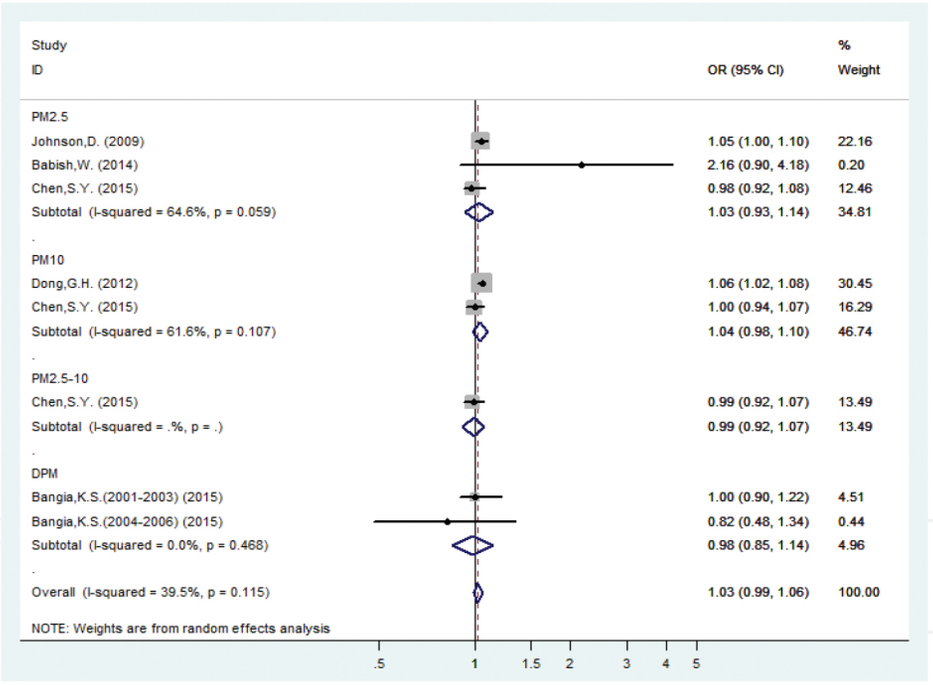

Figure 2. Estimates of hypertension risk associated with a 10 μg/m^3 change in exposure to PM overall and by pollutant, including $PM_{2.5}$, $PM_{2.5–10}$, PM_{10}, DPM.

3.3. Subgroup analyses and publication bias

The results of our subgroup analyses showed that our pooled OR for the prevalence was 1.03 (95% CI, 0.93–1.14) with I^2 of 64.6% in $PM_{2.5}$ and 1.04 (95% CI, 0.98–1.10) with I^2 of 61.6% heterogeneity in PM_{10} and 0.98 (95% CI, 0.85–1.14) with no heterogeneity.

Funnel plots for each pollutant exhibited a notable asymmetry, reflecting no publication bias from vision (**Figure 3**). Introducing "trim and fill method", the results suggested no existed publication bias exist. For all exposures, the *P* values of Egger tests were statistically non-significant ($P = 0.496$). To sum up, the possibility of publication bias is small.

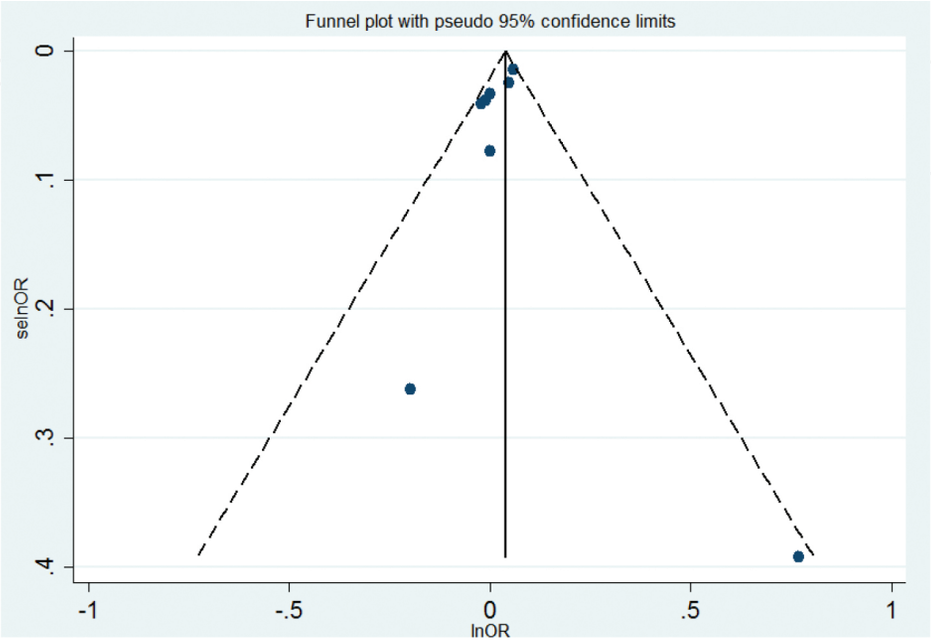

Figure 3. Funnel plot with pseudo 95% confidence limits.

4. Discussion

To our knowledge, this is the first systematic review and meta-analysis to pooled estimates linked with ambient PM and hypertension among general population. We selected and reviewed five large population-based cross-sectional studies, including a total of >200,000 general population. The pooled estimator of our meta-analysis showed that ambient PM was marginally associated, but not statistically significant, with hypertension among general population.

There are an increasing number of studies which have reported the association between PM and BP. Some studies showed that BP increased with short-term exposure to $PM_{2.5}$ of 10 μg/m^3 [SBP 0.81 (95% CI: 0.19, 1.43) mmHg and DBP 0.63 (95% CI: 0.17, 1.10) mmHg] [24] and for each 10 μg/m^3 increase of PM_{10}, pulse pressure and SBP increased by 0.583 (95% CI: 0.296–0.870) mmHg and 0.490 (95% CI: 0.056–0.925) mmHg, respectively in Geneva, and 0.183 (95% CI: 0.017–0.348) mmHg and 0.036 (95% CI: 0.042–0.561) mmHg, respectively in Lausanne [25]. Meanwhile, several studies also assessed the association between long-term exposure to particulate air pollution and BP. Fuks et al. reported that an interquartile increase in $PM_{2.5}$ (2.4 μg/m^3) was associated with an estimated increase in mean SBP and DBP of 1.4 (95% CI: 0.5–

2.3) mmHg and 0.9 (95% CI: 0.4, 1.4) mmHg, respectively [18]. Also, Dong et al. found that the estimated increases in mean SBP and DBP were 0.87 (95% CI: 0.48–1.27) mmHg and 0.32 (95% CI: 0.08–0.56) mmHg per 19 μg/m^3 interquartile increase in PM_{10}, respectively [13].

Till now, only three epidemiology studies [14, 17, 26] have explored the effect of air pollution on the incidence of hypertension, and the results have been varied. A cohort study of 35,303 non-hypertensive adults from Ontario, Canada, with follow-up from 1996 to 2005, reported that the adjusted hazard ratio of incident (IRR) hypertension was 1.13 (95% CI: 1.05–1.22) for incident hypertension among individuals who were exposed to per 10 μg/m^3 increase of $PM_{2.5}$ [14]. Coogan et al. reported that in a cohort of black women living in Los Angeles, with exposure to $PM_{2.5}$ and nitrogen oxides, over follow-up from 1995 to 2005, the incidence rate ratio (IRR) for hypertension for a 10 μg/m^3 increase in $PM_{2.5}$ was 1.48 (95% CI: 0.95–2.31), and the IRR for the interquartile range (12.4 parts per billion) of nitrogen oxides was 1.14 (95% CI: 1.03–1.25) [17]. The third cohort of 33,275 participants was enrolled in this population-based cohort study. The IRR of self-reported incident hypertension among individuals who were exposed to nitrogen oxides (NOx) in the 95th quantile (>26.6 parts per billion), contrasted with those in the 5th quantile (<16.1 parts per billion), is 1.06 (95% CI: 0.92–1.23) after a mean follow-up of 5.3 years [26].

The strength of this study is mainly related to the large number of residents. In addition, none of the previous meta-analyses focused on investigating the relationship between ambient PM and hypertension. Compared with the cohort study of Canada [14], the existing problem of this meta-analysis is the study design. The evidence strength of the cohort study is higher than the cross-sectional study. Because of this, we need think about seriously the combined effects of this meta-analysis. And researchers should pay more attention to PM and hypertension.

4.1. Biological mechanisms associated with PM and hypertension

A potential mechanism relating exposure to $PM_{2.5}$ to hypertension may be its indirect effects mediated through systemic proinflammatory and oxidative responses, which may lead to increased sympathetic tone and potentially cause arterial remodeling. Oxidative stress may also increase the circulation of activated immune cells and inflammatory cytokines, which may subsequently induce endothelial dysfunction, leading to an imbalance in vascular homeostatic responses. If this happens repeatedly, it could result in an increased total peripheral resistance and a fixation of elevated BP [18]. Other mechanisms by which $PM_{2.5}$ may elevate BP include autonomic nervous system imbalance and direct vasoconstriction [17, 27]. Finally, PM exposure can reduce daytime sodium excretion and blunt the normal nocturnal reduction in BP. Over time, impaired renal handling of excess sodium may be partly responsible for elevated BP [27].

5. Limitations

There are some limitations of this meta-analysis which should be noted. First, only five screened papers were selected in this study due to the low number of studies associated with

ambient PM and hypertension among general population till now. And these five studies examined the relationship between PM and hypertension among general population using cross-sectional epidemiological study design, which precludes a causal interpretation of the complex relationship between outdoor PM and hypertension. Second, the exposure assessment of ambient PM is not consistent, so the risk of misclassification of different-level concentration might be amplified. Third, the degree of control for potential confounders is different. Covariates in common are included for all studies, including gender, age, body mass index, education, smoking status in most, but not all studies. And some reports indicated that diabetes mellitus and COPD (chronic obstructive pulmonary disease) are often associated with increased risk for hypertension [28, 29], but no studies had information on COPD and diabetes mellitus. Finally, not only do ambient PM concentrations and toxicity of major components vary largely over space [30] but also possibly other parameter such as season may be a potential confounder because seasonal variation in air pollution is well known [31].

6. Conclusion

The present meta-analysis of cross-sectional studies suggests that ambient PM may not contribute to the prevalence of hypertension from statistical perspective, but the evidence for a relationship between ambient particulate matter exposure and hypertension is still inconclusive because of limitations in study populations, study design, exposure characterization, and adjustment for important confounders and need researchers to pay more attention to PM and hypertension.

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References

[1] Poulter, N.R., D. Prabhakaran, and M. Caulfield, *Hypertension.* Lancet, 2015. 386(9995): pp. 801–12.

[2] Wong, N.D., et al., *Global cardiovascular risk associated with hypertension and extent of treatment and control according to risk group.* Am J Hypertens, 2012. 25(5): pp. 561–7.

[3] Klag, M.J., et al., *Blood pressure and end-stage renal disease in men.* N Engl J Med, 1996. 334(1): pp. 13–8.

[4] Hsu, C.Y., et al., *Elevated blood pressure and risk of end-stage renal disease in subjects without baseline kidney disease.* Arch Intern Med, 2005. 165(8): pp. 923–8.

[5] Lim, S.S., et al., *A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: A systematic analysis for the Global Burden of Disease Study 2010.* Lancet, 2012. 380(9859): pp. 2224–2260.

[6] Rahimi, K., C.A. Emdin, and S. MacMahon, *The epidemiology of blood pressure and its worldwide management*. Circ Res, 2015. 116(6): pp. 925–36.

[7] Hansson, G.K., *Inflammation, atherosclerosis, and coronary artery disease*. N Engl J Med, 2005. 352(16): pp. 1685–95.

[8] Brook, R.D., *Why physicians who treat hypertension should know more about air pollution.* J Clin Hypertens (Greenwich), 2007. 9(8): pp. 629–35.

[9] Hicken, M.T., et al., *Fine particulate matter air pollution and blood pressure: the modifying role of psychosocial stress.* Environ Res, 2014. 133: pp. 195–203.

[10] Brook, R.D., et al., *Hemodynamic, autonomic, and vascular effects of exposure to coarse particulate matter air pollution from a rural location.* Environ Health Perspect, 2014. 122(6): pp. 624–30.

[11] Bellavia, A., et al., *DNA hypomethylation, ambient particulate matter, and increased blood pressure: findings from controlled human exposure experiments.* J Am Heart Assoc, 2013. 2(3): p. e000212.

[12] Babisch, W., et al., *Associations between Traffic Noise, Particulate Air Pollution, Hypertension, and Isolated Systolic Hypertension in Adults: The KORA Study.* Environ Health Perspect, 2014. 122(5): pp. 492–8.

[13] Dong, G.H., et al., *Association between long-term air pollution and increased blood pressure and hypertension in China.* Hypertension, 2013. 61(3): pp. 578–84.

[14] Chen, H., et al., *Spatial association between ambient fine particulate matter and incident hypertension.* Circulation, 2014. 129(5): pp. 562–9.

[15] Johnson, D. and J.D. Parker, *Air pollution exposure and self-reported cardiovascular disease.* Environ Res, 2009. 109(5): p. 582–9.

[16] Chen, S.Y., et al., *Associations between long-term air pollutant exposures and blood pressure in elderly residents of Taipei city: A cross-sectional study.* Environ Health Perspect, 2015. 123(8): p. 779–784.

[17] Coogan, P.F., et al., *Air pollution and incidence of hypertension and diabetes mellitus in black women living in Los Angeles.* Circulation, 2012. 125(6): p. 767–72.

[18] Fuks, K., et al., *Long-term urban particulate air pollution, traffic noise, and arterial blood pressure.* Environ Health Perspect, 2011. 119(12): pp. 1706–11.

[19] Bangia, K.S., et al., *A cross-sectional analysis of polycyclic aromatic hydrocarbons and diesel particulate matter exposures and hypertension among individuals of Mexican origin.* Environ Health, 2015. 14:51.

[20] DerSimonian, R. and N. Laird, *Meta-analysis in clinical trials.* Control Clin Trials, 1986. 7(3): pp. 177–88.

[21] Higgins, J.P., et al., *Measuring inconsistency in meta-analyses.* Br Med J, 2003. 327(7414): pp. 557–60.

[22] Orru, H., et al., *Chronic traffic-induced PM exposure and self-reported respiratory and cardiovascular health in the RHINE Tartu Cohort.* Int J Environ Res Public Health, 2009. 6(11): pp. 2740–51.

[23] Basile, J.N. and M.J. Bloch, *Exposure to air pollution increases the incidence of hypertension and diabetes in black women living in Los Angeles.* J Clin Hypertens, 2012. 14(11): pp. 819–20.

[24] Chang, L.T., et al., *Short-term exposure to noise, fine particulate matter and nitrogen oxides on ambulatory blood pressure: A repeated-measure study.* Environmental Research, 2015. 140: pp. 634–40.

[25] Tsai, D.H., et al., *Short-term effects of particulate matters on pulse pressure in two general population studies.* J Hypertens, 2015. 33(6): pp. 1144–52.

[26] Sorensen, M., et al., *Long-term exposure to traffic-related air pollution associated with blood pressure and self-reported hypertension in a Danish Cohort.* Environ Health Perspect, 2012. 120(3): pp. 418–24.

[27] Tsai, D.H., et al., *Short-term increase in particulate matter blunts nocturnal blood pressure dipping and daytime urinary sodium excretion.* Hypertension, 2012. 60(4): pp. 1061–9.

[28] Mannino, D.M., et al., *Prevalence and outcomes of diabetes, hypertension and cardiovascular disease in COPD.* Eur Respir J, 2008. 32(4): pp. 962–9.

[29] Stone, I.S., N.C. Barnes, and S.E. Petersen, *Chronic obstructive pulmonary disease: a modifiable risk factor for cardiovascular disease?* Heart, 2012. 98(14): pp. 1055–62.

[30] Lelieveld, J., et al., *The contribution of outdoor air pollution sources to premature mortality on a global scale*. Nature, 2015. 525(7569): pp. 367–71.

[31] Liang, C.S., et al., *Review on recent progress in observations, source identifications and countermeasures of PM2.5.* Environ Int, 2016. 86: pp. 150–70.

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