

Respiratory Health Effects of Ambient Air Pollution: An Update

Francesco Sava, MD, MSc, FRCPC^{a,b},
Chris Carlsten, MD, MPH^{a,b,*}

KEYWORDS

• Air pollution • Gene • Environment • Respiratory health • Asthma • COPD • Lung cancer

KEY POINTS

- The last decade of research on ambient air pollution (AAP) has confirmed its adverse impact on respiratory morbidity and mortality, quality of life, and economic burden on health care systems.
- Efforts to reduce AAP have been fruitful, in terms of reduced mortality, but the concentration-effect relationship seems steepest at the lower end of the concentration range, suggesting that further reductions in AAP may confer marked additional benefit.
- Evidence for incident asthma caused by AAP has proliferated, strengthening the claim of a cause-effect relationship, particularly in children.
- This finding is of critical importance because such insight may support additional efforts to prevent new lung disease, and prevention of new disease generally has a larger impact than does preventing exacerbations of existing disease.
- Research has demonstrated that co-exposures have the potential to dramatically augment the effects of AAP and, thus, lower the threshold of effect of a given pollutant.

INTRODUCTION

A lot has happened since the topic of the health effects of ambient air pollution (AAP) was last reviewed in the *Clinics in Chest Medicine* 10 years ago,¹ with major contributions from epidemiology, toxicology, and particularly genetics and epigenetics. With this review, the authors do not aim to be comprehensive regarding the effect of AAP on human respiratory health because the topic has been covered from several perspectives in recent years.^{2–5} Instead, the purpose of the current review is to reflect on specific areas where progress has been most significant in the last decade. For each such topic, the authors include the most up-to-date evidence and explain how it has advanced

our understanding of long-standing concerns. The authors' approach is to integrate findings from epidemiology, controlled human experimental exposures, and toxicology, highlighting key areas of novel insight and describing prominent gaps in the evidence to motivate future research. AAP generally refers to particulate matter, as well as ozone and other gasses, largely generated by the combustion of fossil fuels by vehicles or power plants, outdoor biomass burning, and a variety of industrial processes and then dispersed within the commonly inhabited airspace. Therefore, the authors are not covering indoor air, including indoor biomass burning, cardiovascular disease (the subject of much attention, appropriately, elsewhere⁶), or disorders outside the lung.

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^a Air Pollution Exposure Laboratory (APEL), Vancouver Coastal Health Research Institute, University of British Columbia, Vancouver General Hospital (VGH)-Research Pavilion, 828 West 10th Avenue, Vancouver, British Columbia V5Z 1L8, Canada; ^b The Lung Centre, Department of Medicine, Vancouver General Hospital, 7th Floor, 2775 Laurel Street, Vancouver, British Columbia V5Z 1M9, Canada

* Corresponding author.

E-mail address: Carlsten@mail.ubc.ca

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The authors divide the material into (1) epidemiologic findings, (2) experimental results (especially as they are oriented to mechanistic support for observational population-based findings), and (3) genetic and epigenetic insights, given the particularly prominent role of these disciplines in recent literature and their potential to identify at-risk populations and novel mechanisms that might guide upcoming policy decisions and further research.

EPIDEMIOLOGIC STUDIES

Particulate Matter: Acute Effects

The most powerful epidemiologic evidence of the effects of short-term exposure to air pollution has typically come from large cooperative efforts that allow the standardization of analytic methods and reporting across a broad range of international jurisdictions. One of the biggest such collaborative studies, Air Pollution and Health: A Combined European and North American Approach (APHENA), includes studies from the Air Pollution and Health: A European Approach (APHEA) study in Europe; the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) in the United States; and others from Canada.^{7,8} The effect of particulate matter with a diameter less than 10 μm (PM10) on increased daily death rates ranged from 0.2% to 0.6% for each 10 $\mu\text{g}/\text{m}^3$ increase in ambient PM10 concentration. The main objective of APHENA was to assess the coherence of the findings of the multicity studies carried out on different continents; APHENA found similar effects when comparing Europe and the United States, showing the robustness of the data and the different modeling approaches. Across regions, effects were stronger for people older than 75 years. Most of the effect was driven by cardiovascular mortality, but a significant increase in respiratory mortality was also noted.

The daily variation in disease burden caused by urban air pollution was further assessed by measuring the number of emergency visits and hospital admissions caused by respiratory diseases. In the APHEA cohorts, a 10 $\mu\text{g}/\text{m}^3$ increase in PM10 was associated with increases of 1.2% in children asthma admissions, 1.1% in adult asthma admissions, and 0.9% in respiratory diseases as a combined end point (chronic obstructive pulmonary disease [COPD], asthma, and other respiratory diseases). Effects were stronger in children with asthma without antiinflammatory medication; notably, the association between PM10 and respiratory hospital admissions was significant on the same day of the PM10 assessment (zero day lag) and also over lags of several subsequent days. The principle novelty in APHENA is its assessment of the acute effects of AAP across such diverse

geographic regions with such different modeling approaches, and the coherence of its findings adds substantial confidence in the collective evidence of prior decades connecting AAP to the exacerbation of respiratory disease.

Progress has also been made regarding the acute impact of PM on patients with established respiratory conditions, such as asthma and COPD. Such individuals are considered vulnerable because of their compromised respiratory reserve. In a national database comprising daily measurements of PM2.5, Dominici and colleagues⁹ showed an association between acute changes in the PM2.5 concentration and hospitalizations for heart failure and COPD. Halonen and colleagues¹⁰ showed, similarly, that acute changes in levels of PM2.5 were associated with hospital admissions for asthma and COPD with a lag of 4 days in children but additionally demonstrated an immediate effect in the elderly. Children may be another vulnerable group and one whose vulnerability to traffic-related air pollution (TRAP) may not always be apparent. Rabinovitch and colleagues¹¹ found that environmental tobacco smoke (ETS) obscured the relationship between morning maximum ambient PM2.5 levels and urinary leukotriene E4 (LTE4) in a group of asthmatic children; notably, only in the absence of elevated ETS exposure was PM2.5 significantly associated with significant increases in LTE4.

Although fine PM has become a dominant focus of concern over the past decades, there remains interest in toxicity attributable to the coarse fraction of PM (between 2.5 and 10 μm in diameter). A Danish study by Iskandar and colleagues¹² studied the association between pediatric hospital admissions for asthma and daily PM measurements, stratifying by particle size within a case-crossover design. There was a significant association of admissions with PM10 and PM2.5 but not ultrafine particles, suggesting a prominent role for coarse PM. Another study in patients with asthma noted a significant positive correlation between daily coarse particle exposure and peripheral blood eosinophils but not pulmonary function.¹³

Particulate Matter: Chronic Effects

Teasing out the contribution of each component of TRAP, inherently a mixture of PM and gases, is difficult because of the substantial correlation between TRAP components within epidemiologic studies. With that caveat, the association of particulate matter with mortality has been most extensively documented. Studies from 4 landmark cohorts, the Harvard Six Cities Study,¹⁴ NMMAPS,^{15–17} the American Cancer Society's Cancer Prevention Study II (CPS-II),¹⁸ and APHEA,¹⁹ have

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