Source-specific air pollution and risk of chronic obstructive pulmonary disease: A pooled cohort study

Manuella Lech Cantuaria,[1,2*] Aslak Harbo Poulsen,[1] Ole Raaschou-Nielsen,[1,3] Étienne Audureau,[4,5] Ralph Epaud,[4,6,7] Sophie Lanone,[4] Jørgen Brandt,[3,8] Lise Marie Frohn,[3] Matthias Ketzel,[3] Anja Olsen,[9,10] Lau Caspar Thygesen,[11] Mette Sørensen[1,12]

[1] Work, Environment and Cancer, Danish Cancer Institute, Copenhagen, Denmark

[2] Department of Clinical Research, University of Southern Denmark, Odense, Denmark

[3] Department of Environmental Science, Aarhus University, Roskilde, Denmark

[4] University Paris-Est Créteil, INSERM, IMRB, Creteil, France

[5] Public Health Department, Clinical Research Unit (URC), Hôpital Henri-Mondor, Assistance Publique Hôpitaux de Paris (APHP), Créteil, France

[6] Department of General Pediatrics, Centre Hospitalier Intercommunal de Créteil, Créteil, France

[7] Center for Rare Lung Diseases (RESPIRARE), Créteil, France

[8] iClimate - Interdisciplinary Centre for Climate Change, Aarhus University, Roskilde, Denmark

[9] Diet, Cancer and Health, Danish Cancer Institute, Copenhagen, Denmark

[10] Department of Public Health, University of Aarhus, Aarhus, Denmark

[11] National Institute of Public Health, University of Southern Denmark, Copenhagen, Denmark

[12] Department of Natural Science and Environment, Roskilde University, Roskilde, Denmark

* Corresponding author. Email: mlc@cancer.dk ; Phone number: +45 2721-1181; Work address: Strandboulevarden 49, 2100 København

Conflict of interest:
The authors declare they have no conflicts of interest related to this work to disclose.

Abstract

Background:
The evidence linking long-term exposure to air pollution and development of chronic obstructive pulmonary disease (COPD) is still controversial. Furthermore, most studies have investigated associations with particulate matter (PM) and nitrogen dioxide (NO_2), disregarding their emission source and other relevant air pollutants, such as ultrafine particles (UFP) and elemental carbon (EC).

Objectives:
This study aimed to assess associations between long-term residential exposure to $PM_{2.5}$, NO_2, UFP, and EC and risk of COPD, distinguishing the effects of air pollution from local traffic and other sources.

Methods:
We pooled data from two large Danish cohorts - the Diet, Cancer, and Health cohort and the Danish National Health Survey. For all participants (N = 159,769), we estimated long-term air pollution exposure to total, local traffic, and other contributions, based on complete address histories. We used Cox proportional hazards models to estimate associations between 10-year time-weighted averaged air pollution and incident COPD, adjusting for demographic, socioeconomic, and lifestyle factors, including smoking. We evaluated possible modification of these associations by sex, smoking status, and previous asthma diagnosis.

Results:
Long-term exposures to $PM_{2.5}$, NO_2, UFP, and EC were associated with higher risk of COPD. The highest hazard ratio (HR) per interquartile range of total contributions was observed for $PM_{2.5}$ (HR: 1.11 [95% confidence interval: 1.05, 1.17]), followed by NO_2 (1.08 [1.04, 1.13]), UFP (1.05 [0.99, 1.11]), and EC (1.02 [1.00, 1.05]), after full adjustment. $PM_{2.5}$ from other sources than local traffic was more strongly associated with COPD than $PM_{2.5}$ from local traffic, while for UFP and EC, the contributions from local traffic seemed most harmful. Effect modification analyses showed stronger associations among women, never smokers, and those with an asthma diagnosis.

Discussion:
Our findings suggest that air pollution from local traffic and other sources contribute to COPD risk, with variations depending on the pollutant type. Further research is needed to validate these findings across different populations and geographical settings.