



POSTER PRESENTATION

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Estimation of the environmental attributable fraction of asthma among Canadian children: a systematic review

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Background

We systematically summarized studies that evaluated the associations between environmental exposures and asthma development by calculating the population attributable fraction (PAF) of Canadian childhood asthma due to modifiable environmental exposures.

Methods

Asthma incidence among Canadian children was estimated from population-based surveys and administrative datasets. The prevalence of Canadian exposure to airborne pollutants, environmental tobacco smoke (ETS), indoor allergens, and home mould and moisture were determined from peer-reviewed publications and government reports. International estimates of the relative risk of physician-diagnosed asthma were determined from peer-reviewed studies and used to determine attributable risk (AR) for PAF calculation.

$$\text{PAF} = \frac{\text{AR} * \text{Exposure prevalence}}{\text{Asthma incidence}} * 100\%$$

Results

The Canadian childhood asthma incidence was between 2.8% and 5.3%. Canadian exposure prevalences were: PM₁₀ 16%, outdoor PM_{2.5} 7.1%, indoor PM_{2.5} 1.7%, outdoor NO₂ 25%, indoor NO₂ 3.3%, O₃ 22%, SO₂ 0.1%, CO 0.1%, environmental tobacco smoke (ETS) 9.0%, cat 22%, dog 12%, mouse 17%, cockroach 1.7%, dust mite 30%, moisture 14%, and mould 33%. Median odds ratios

of physician-diagnosed asthma used to determine the AR were above 1.00 for PM₁₀, PM_{2.5}, NO₂, CO, ETS, mouse, cockroach, moisture, and mould. The PAF estimates were: PM₁₀ 11%, outdoor PM_{2.5} 1.2%, indoor PM_{2.5} 0.30%, outdoor NO₂ 1.4%, indoor NO₂ 0.19%, ETS 4.0%, mouse 3.8%, cockroach 0.22%, moisture 4.5%, mould 10%, and 0 for O₃, SO₂, CO, cat, dog, and dust mites.

Conclusions

This systematic review suggests contributions to Canadian childhood asthma development from exposure to particulates, NO₂, ETS, mouse, cockroach, mould, and moisture, although the results are not consistent enough to imply causation. The associations with cat, dog and dust mite allergen exposure appear to be more complex. These findings highlight the need for longitudinal methods to more accurately estimate the contributions of modifiable environmental exposures to childhood asthma development.

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