

Air pollution and atopic dermatitis: critical windows of risk in early life

Linked Article: Lin *et al.* *Br J Dermatol* 2025; **192**:1038–1046.

<https://doi.org/10.1093/bjd/ljaf109>

In this issue of the *BJD*, Lin *et al.* report on how fine particulate matter (PM_{2.5} – particulate matter less than or equal to 2.5 µm in diameter) may influence the risk of atopic dermatitis (AD) in children born between 2004 and 2013 in Taiwan. The role of air pollution in the development of AD remains a topic of ongoing debate, as designing a study capable of demonstrating a causal relationship is particularly challenging.

In their study, Lin *et al.* take advantage of Taiwan's comprehensive registry system to establish a birth cohort of 564 869 live-born children, with follow-up data available until the age of 5 years. Among them, 79 944 children were diagnosed with AD. To assess PM_{2.5} exposure, the authors integrated multiple data sources, including satellite imagery, meteorological observations and ground-based air quality measurements, allowing for precise estimation of PM_{2.5} concentrations over time.¹

Their findings are particularly compelling. The study shows that cumulative exposure to PM_{2.5} is associated with an increased risk of developing AD, with two critical exposure windows: from gestational week 34 until birth and from 33 weeks of age after birth. These results suggest that air pollution – specifically fine particulate matter – plays a significant role in increasing the incidence of AD. The results also highlight key periods during fetal development and early infancy when exposure may be particularly harmful.

There is evidence that airborne pollution may have a detrimental effect on the skin, and increase both incidence and symptoms of skin disease including AD.^{2,3} The exact mechanism remains elusive, but oxidative stress disturbance of the epidermal barrier and pH, and alterations in skin microbiota, may all be factors that contribute to the increased risk of AD in later life. Furthermore, maternal exposure to PM_{2.5} may lead to exposure of the fetus as the particulate matter can cross the placental barrier and cause immune dysregulation and inflammation.^{2,4}

This study leverages big data by integrating information from diverse sources, yielding intriguing findings that enhance our understanding of AD development. Moreover, these results have the potential to influence policymakers in shaping environmental policies.

Christian Vestergaard

Department of Dermatology, Aarhus University Hospital, Aarhus, Denmark

Email: chrivest@rm.dk

Funding sources: This commentary received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

Conflicts of interest: C.V. has received honoraria from service on advisory boards for and/or received research grants from Almirall, AbbVie, CellDex, Novartis, LEO Pharma, Pfizer, Sanofi, Galderma, Eli Lilly and MEDA.

Data availability: Not applicable.

Ethics statement: Not applicable.

Patient consent: Not applicable.

References

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