Editorial

Across borders, across time: early-life environmental exposures and juvenile dermatomyositis

G. Adami

Rheumatology Unit,
Department of Medicine,
University of Verona, Italy.

Giovanni Adami, MD, PhD

Please address correspondence to:
Giovanni Adami
Rheumatology Unit,
University of Verona,
Piazzale L.A. Scuro 10,
37134 Verona, Italy.
E-mail: giovanni.adami@univr.it

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Juvenile dermatomyositis (JDM) sits at a crossroads of genetics, immune disruption, and environment. We have long accepted that "the exposome" matters in systemic autoimmune rheumatic diseases (SARDs), yet in paediatric myositis the field still struggles to move from plausible triggers to actionable prevention or mechanistic stratification. In this context, Arabi *et al*. provide a timely and thought-provoking comparison of prenatal and early postnatal environmental exposures in JDM patients from Brazil versus the United States, highlighting how place, socioeconomic context, and daily-life exposures may shape disease onset and possibly phenotype (1).

The authors report two observations: first, Brazilian children developed JDM at a younger age than U.S. children. Second, the "exposure landscape" differed substantially: Brazilian mothers were more often exposed to dust and passive smoke, more likely to live/work closer to factories/quarries, and more often relied on bus transportation, while U.S. mothers more frequently worked outside the home in office settings and used subway commuting.

These patterns matter because they are not merely lifestyle descriptors, they are plausible proxies for mixtures of inhalable particulate matter, diesel exhaust constituents, silica-like dusts, and second-hand smoke exposure, all of which can prime innate and adaptive immunity through oxidative stress pathways, epithelial/vascular activation, and epigenetic remodelling.

Importantly, the paper also underscores a message rheumatology is increasingly forced to confront: environmental risk is not evenly distributed. The "where you live, work and commute" signature is a social and structural determinant as much as it is a biologic one. That observation aligns with broader rheumatology literature linking climate-related exposures (especially air pollution) to autoimmune disease risk and activity; while simultaneously exposing how unprepared our specialty remains to translate this into care pathways or advocacy (2).

The study is cross-sectional and questionnaire-based, and it compares two national cohorts without contemporaneous local controls. That design, despite not proving causal relationship and with its limits, is appropriate for hypothesis generation. Many measured variables (*e.g*., "distance to factory/quarry," "bus *vs*. subway commute," "dust exposure") function as proxies for unmeasured pollutant mixtures and social context. This is not a weakness per se, it is the reality of paediatric exposome research, but it does mean we should avoid over-reading any single factor as "the trigger."

Still, it would be a mistake to dismiss these findings as merely descriptive. The paper highlights clusters of exposures that plausibly shift the timing of immune priming and the threshold for clinical disease onset. The broader rheumatology literature supports the concept that early-life windows are special. For example, a recent SARD-focused analysis reported that in utero and early-life exposure to smoking is associated with SARD development even after accounting for genetic risk, reinforcing the idea that prenatal/childhood exposures can leave durable immune "imprints" (3).

Likewise, in adult SARD research, large prospective cohorts have linked long-term air pollutants with incident systemic lupus erythematosus and gene-environment interactions, providing a proof-of-principle that pollutant

exposures can operate alongside inherited susceptibility (4).

JDM pathogenesis is tightly tied to microvascular injury and interferon-driven immune activation. From a mechanistic standpoint, inhaled pollutants and tobacco smoke can plausibly influence endothelial activation, oxidative stress, and innate immune programming pathways that map onto JDM biology. The more interesting (and clinically relevant) hypothesis raised by Arabi et al. is not simply "pollution causes JDM," but rather: different exposure profiles may shift the age of onset and potentially contribute to phenotype heterogeneity.

Other recent evidence, from the same group, in JDM supports and refine the hypothesis that environmental pollution contributes to disease risk. A 2024 case–control study from São Paulo identified maternal occupational exposure to inhalable agents and higher ozone exposure during early childhood as factors associated with JDM onset, while most prenatal air-pollution measures were not independently associated (5). These findings suggest that the timing and type of exposure may be more relevant than generic pollutant burden. However, available studies remain small, largely questionnaire-based, and geographically limited, underscoring the need for objective exposure modelling and replication across diverse settings.

Three next steps feel realistic:

- Harmonised case-control designs with geocoded exposure modelling. Questionnaires should be paired with satellite/land-use regression estimates for $PM_{2.5}$, NO_2, O_3, and traffic-related pollutants, and, crucially, time-windowed around pregnancy trimesters and early childhood. This is already feasible in many countries and would reduce reliance on proxy variables.
- Mechanism-informed stratification. JDM is heterogeneous (clinical course, myositis-specific autoantibodies, IFN signatures). The key question is whether exposure clusters are associated with serologic or molecular subgroups, not only with disease presence. This could reveal distinct "environment-responsive" endotypes.
- Translation to prevention and counselling without blaming families. Even now, rheumatologists can responsibly emphasize avoidance of tobacco smoke exposure in pregnancy and early life, an intervention supported by converging evidence across SARDs. For air pollution, the emphasis should be on advocacy (clean air policies) and practical mitigation for high-exposure settings, while recognising that individual-level control is limited and inequitable.

In aggregate, emerging data suggests that early-life environmental exposures may modulate susceptibility and timing of JDM onset, but definitive causal pathways remain to be established through larger, mechanistically informed studies.

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