

Long term exposure to air pollution linked to heightened autoimmune disease risk

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Long term exposure to air pollution is linked to a heightened risk of autoimmune disease, particularly rheumatoid arthritis, connective tissue and inflammatory bowel diseases, finds research published online in the

open access journal *RMD Open*.

Environmental [air pollution](#) from vehicle exhaust and industrial output can trigger adaptive immunity—whereby the body reacts to a specific disease-causing entity. But sometimes this adaptive response misfires, prompting systemic inflammation, [tissue damage](#), and ultimately autoimmune disease.

Examples of autoimmune disease include rheumatoid arthritis; [systemic lupus erythematosus](#); inflammatory bowel diseases, such as ulcerative colitis; [connective tissue](#) disease, such as osteoarthritis; and multiple sclerosis.

Both the incidence and prevalence of these conditions have steadily increased over the past decade, the reasons for which aren't entirely clear. And whether air pollution is linked to a heightened risk of autoimmune disease remains a matter of debate, say the researchers.

To try and shed some light on the issues, the researchers mined the national Italian fracture risk database (DeFRA) and retrieved comprehensive medical information on 81,363 men and women submitted by more than 3500 doctors between June 2016 and November 2020.

Most were women (92%) with an average age of 65, and 17866 (22%) had at least one co-existing health condition.

Each participant was linked to the nearest air quality monitoring station run by the Italian Institute of Environment Protection and Research via their residential postcode.

The researchers were particularly interested in the potential impact of particulate matter (PM10 and PM2.5). Levels of $30\mu\text{g}/\text{m}^3$ for PM10 and

20 $\mu\text{g}/\text{m}^3$ for PM2.5 are the thresholds generally considered harmful to human health.

Some 9723 people (12%) were diagnosed with an autoimmune disease between 2016 and 2020.

Information on air quality was obtained from 617 monitoring stations in 110 Italian provinces. Average long term exposure between 2013 and 2019 was 16 $\mu\text{g}/\text{m}^3$ for PM2.5 and 25 $\mu\text{g}/\text{m}^3$ for PM10.

Exposure to PM2.5 wasn't associated with a heightened risk of an autoimmune disease diagnosis. But PM10 was associated with a 7% heightened risk for every 10 $\mu\text{g}/\text{m}^3$ increase in levels, after accounting for potentially influential factors.

Long term exposure to PM10 above 30 $\mu\text{g}/\text{m}^3$ and to PM2.5 above 20 $\mu\text{g}/\text{m}^3$ were associated with, respectively, a 12% and 13% higher risk of autoimmune disease.

And long term exposure to PM10 was specifically associated with a heightened risk of rheumatoid arthritis, while long term exposure to PM2.5 was associated with a heightened risk of rheumatoid arthritis, connective tissue diseases, and inflammatory bowel diseases.

Overall, long term exposure to traffic and industrial air pollutants was associated with an approximately 40% higher risk of rheumatoid arthritis, a 20% higher risk of inflammatory bowel disease, and a 15% higher risk of connective tissue diseases.

This is an observational study, and as such, can't establish cause. And the researchers acknowledge several limitations which might have affected their findings.

These include: the lack of information on the dates of diagnosis and start of autoimmune disease symptoms; that air quality monitoring might not reflect personal exposure to pollutants; and that the findings might not be more widely applicable because study participants largely comprised older women at risk of fracture.

But air pollution has already been linked to immune system abnormalities, and smoking, which shares some toxins with fossil fuel emissions, is a predisposing factor for [rheumatoid arthritis](#), they explain.

More information: Association between long-term exposure to air pollution and immune mediated diseases: a population-based cohort study, *RMD Open*. [DOI: 10.1136/rmdopen-2021-002055](https://doi.org/10.1136/rmdopen-2021-002055)

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