GEM PATIENT EDUCATION CENTER

Industrialization, Environmental Pollution & Endometriosis

What the evidence suggests – and what is still unproven

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WHAT CURRENT EVIDENCE SUPPORTS

Endometriosis is a complex disease shaped by genetic, hormonal, immune and inflammatory factors. Environmental exposures – particularly some endocrine-disrupting chemicals such as dioxins, PCBs, bisphenols and phthalates – are being investigated because they may influence hormone signaling, inflammation, oxidative stress and gene regulation. Several studies report associations with endometriosis, but the evidence is **not sufficient to conclude that industrialization or pollution causes the disease, determines its severity, or explains geographic clusters**.

A Cleaner Environment for a Healthier Tomorrow

Research framework: environmental exposures and endometriosis

Exposure patterns differ by pollutant, region and period. This framework does not demonstrate a uniform worldwide rise in pollution or an endometriosis cause in any city.

EXPOSURES UNDER STUDY

- Air pollution / PM2.5
- Endocrine-disrupting chemicals
- Persistent organic pollutants

- Occupational and consumer exposures

POSSIBLE PATHWAYS

- Hormone signaling
- Inflammation and oxidative stress
- Epigenetic regulation
- Immune-system effects

QUESTIONS TO TEST

- Who may be at higher risk?
- Does age of onset differ?
- Does phenotype or severity differ?
- Could diagnostic access explain a geographic difference?

What Population Studies Should Test

1 MEASURE EXPOSURE
Measure pollutants or biomarkers and timing; a city name is not an exposure measurement.

2 CONFIRM DIAGNOSIS
Use comparable clinical, imaging or surgical definitions across populations.

3 DEFINE PHENOTYPE
Study superficial disease, endometrioma and deep disease, including onset and severity.

4 ACCOUNT FOR BIAS
Consider referral patterns, access to care, imaging, surgery, socioeconomic factors and case ascertainment.

What Can Be Done?

GEM supports environmental protection, safer products and workplaces, international research, and better awareness and access to diagnosis. These are public-health and research aims; none is established here as an endometriosis-specific prevention treatment.

KEY POINT: Reported associations justify further research. They do not prove that industrialization, pollution or a named location caused an individual's endometriosis.

Evidence & interpretation

HOW TO READ THE RESEARCH MODEL

The infographic proposes questions for research: measure exposures, assess biological pathways, define disease accurately and account for bias. It does not establish that pollution causes an individual's endometriosis or that any named city has a greater disease burden because of pollution.

Associations do not establish causation

Reviews report associations for selected endocrine-disrupting chemicals, but study designs, timing of exposure measurement, selection and confounding limit inference. Reviews reach differing assessments of the overall strength of evidence.

Mechanisms are research hypotheses

Hormone signaling, oxidative stress, inflammation, immune responses and epigenetic regulation are potential pathways. The boxes in the graphic organize research questions; they do not establish a verified causal sequence or identify the cause of one person's disease.

Geography and policy

Exposure trends differ by pollutant, region and period. Differences between populations may also reflect access to diagnosis and other sources of bias. Environmental protection and research are reasonable policy aims; the diagram does not establish a specific intervention as prevention or treatment for endometriosis.

Reference identity

Vallée et al. is a narrative review in the 2024 issue (online 2023). Sirohi et al. is a systematic literature review in the 2021 issue (online 2020). Becker et al. is the ESHRE clinical guideline, article hoac009; it is not a global-trends study.

Full references

- **[1]** Coiplet E, Courbiere B, Agostini A et al. Endometriosis and environmental factors: A critical review. J Gynecol Obstet Hum Reprod. 2022;51(7):102418. doi:10.1016/j.jogoh.2022.102418. PMID 35667590. Source
- **[2]** Wieczorek K, Szczęsna D, Jurewicz J. Environmental Exposure to Non-Persistent Endocrine Disrupting Chemicals and Endometriosis: A Systematic Review. Int J Environ Res Public Health. 2022;19(9):5608. doi:10.3390/ijerph19095608. PMID 35565013. Source
- **[3]** Upson K. Environmental risk factors for endometriosis: A critical evaluation of studies and recommendations from the epidemiologic perspective. Curr Epidemiol Rep. 2020;7(3):149-170. doi:10.1007/s40471-020-00236-3. PMID 33042729. Source
- **[4]** Vallée A, Ceccaldi PF, Carbonnel M et al. Pollution and endometriosis: A deep dive into the environmental impacts on women's health. BJOG. 2024;131(4):401-414. doi: 10.1111/1471-0528.17687. PMID 37814514. Source
- **[5]** Sirohi D, Al Ramadhani R, Knibbs LD. Environmental exposures to endocrine disrupting chemicals (EDCs) and their role in endometriosis: a systematic literature review. Rev Environ Health. 2021;36(1):101-115. doi:10.1515/reveh-2020-0046. PMID 32903210. Source
- **[6]** Becker CM, Bokor A, Heikinheimo O, et al. ESHRE guideline: endometriosis. Human Reproduction Open. 2022;2022(2):hoac009. doi:10.1093/hropen/hoac009. Source

References link to the source article, bibliographic record or issuing organization. No listed institution endorses GEM.

Medical information

Do not delay care; seek emergency help for a suspected emergency.

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