GEM PATIENT EDUCATION CENTER

Genes, Environment and Endometriosis

A simple explanation of why endometriosis may behave differently from one woman to another

Version 1.2 · September 2026



WHAT MAY DRIVE ENDOMETRIOSIS?

Endometriosis is a complex disease. Research suggests that it develops through interaction between **inherited genetic susceptibility** and biological changes that occur during life. Hormones, inflammation, the immune system, acquired cellular changes and **epigenetic changes** may all contribute.

WHERE MAY THE ENVIRONMENT FIT?

Inherited genes are largely fixed, but gene activity is dynamic. Environmental exposures – including air pollution and some endocrine-disrupting chemicals – may influence hormones, inflammation, oxidative stress and epigenetic regulation. How much these exposures affect an individual woman's disease remains under investigation.



FOUNDER'S CLINICAL PERSPECTIVE

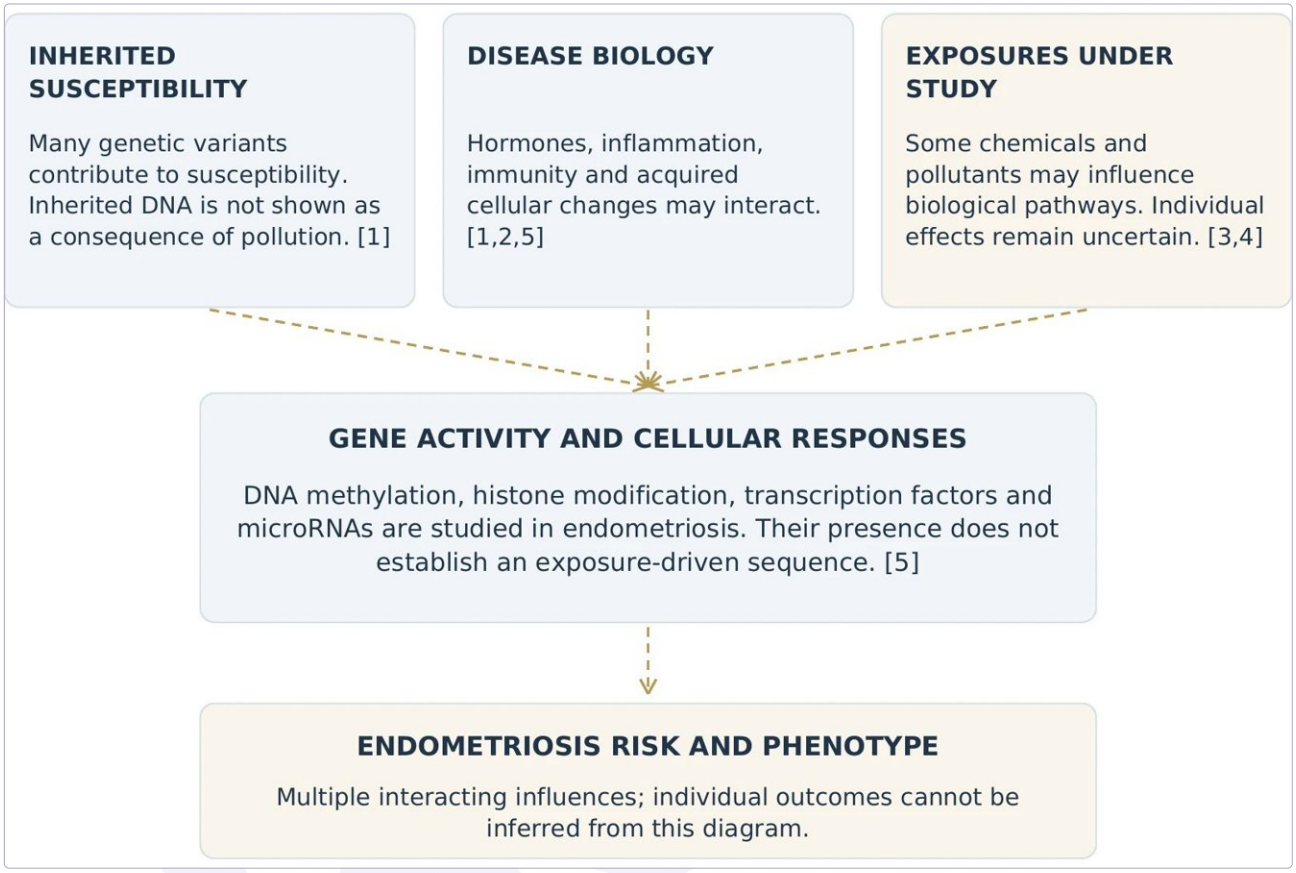
During more than four decades of training, teaching and operating on women with endometriosis in different parts of the world, I have repeatedly observed differences in the presentation and behavior of the disease among patients from different geographic environments. My clinical, teaching and humanitarian work has included the Middle East – particularly Iran – as well as Africa and China.

This is my clinical observation and should not be interpreted as proof that pollution caused the disease. But I believe the consistency of this observation is important and deserves serious scientific investigation.

If environment can influence gene regulation and disease biology, we should investigate whether geography and environmental exposure help explain why endometriosis may appear or behave differently in different populations.

The concept at a glance

A research model of interacting influences, not a proven causal chain



Dashed arrows mark possible interactions. They do not prove that pollution changes inherited DNA, causes endometriosis, or transmits the disease across human generations.

GEM KEY MESSAGE

The teaching slide is a hypothesis model, not a proven causal sequence. Inherited susceptibility and environmental influences may interact through molecular pathways, but the arrows do not establish why any individual developed endometriosis.

Terms: DNMT = DNA methyltransferase; TF = transcription factor. Epigenetic regulation changes gene activity without necessarily changing the DNA sequence. microRNAs help regulate gene expression.

Evidence & interpretation

Genetic susceptibility

Rahmioglu et al. analyzed 60,674 cases and 701,926 controls and identified 42 loci with 49 distinct association signals. These associations support polygenic susceptibility, not a deterministic test for an individual.

Epigenetics and environment

The diagram combines possible mechanisms; it is not a proven chronological causal chain. Observational exposure associations and epigenetic findings do not show why a particular person developed disease. The epigenetic review is a 2025 publication, online in 2024.

Founder observation and treatment

The Founder's geographic impressions are retained as attributed personal observations, not independently measured epidemiology. Hormones can suppress activity and symptoms; they do not surgically excise lesions. Genetic or environmental findings alone do not establish the diagnosis.

What the microRNA sources actually show

Wei et al. (2015) addresses airborne pollution, microRNAs and lung inflammation/cancer. It supplies cross-disease background, not proof of endometriosis causation. Roush and Slack (2008) and Bartel (2009) are general microRNA reviews. They do not establish an endometriosis cause or a current ranking of the least-studied pathways.

Exposure across generations

During pregnancy, a mother (F0), her fetus (F1) and the fetus's developing germ cells (potential F2) can all be directly exposed. This is not by itself evidence of transgenerational inheritance. These sources do not establish a human exposure-induced, multigenerational endometriosis pathway.

Full references

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- **[2]** Becker CM, Bokor A, Heikinheimo O, et al. ESHRE guideline: endometriosis. Human Reproduction Open. 2022;2022(2):hoac009. doi:10.1093/hropen/hoac009. Source
- **[3]** 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
- **[4]** 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
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- **[7]** Roush S, Slack FJ. The let-7 family of microRNAs. Trends Cell Biol. 2008;18(10):505-516. doi: 10.1016/j.tcb.2008.07.007. PMID 18774294. [Background source] Source
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- **[9]** Heard E, Martienssen RA. Transgenerational epigenetic inheritance: myths and mechanisms. Cell. 2014;157(1):95-109. doi:10.1016/j.cell.2014.02.045. PMID 24679529. [Background source] 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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