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ENVIRONMENTAL POLLUTION AND MENSTRUAL CYCLE: A REVIEW OF BIOLOGICAL MECHANISMS AND HEALTH IMPLICATIONS

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ABSTRACT

Research increasingly points to environmental pollution as a significant factor in disrupting women's reproductive health, particularly menstrual cycles. This review synthesizes current evidence on how air pollutants, water contaminants, soil-based toxicants, and endocrine-disrupting chemicals (EDCs) interfere with menstrual function. The mechanisms at play include endocrine disruption, oxidative stress, inflammation, and genetic damage, all of which can destabilize the hypothalamic-pituitary-ovarian axis, alter hormone synthesis, and impair follicle development. Many studies associate these exposures with irregular cycles, dysmenorrhea, anovulation, and amenorrhea. However, the evidence remains limited by inconsistent exposure assessments, residual confounding, and varying definitions of menstrual disorders. To strengthen causal inferences, better biomarkers, both for exposure and biological response, are needed. While current data suggest environmental pollutants pose a real risk to menstrual health, more rigorous, longitudinal studies with standardized measures and improved exposure tracking are essential. Only then can we clarify dose-response relationships, identify critical windows of vulnerability, and understand how mixtures of pollutants interact in the body.

Keywords: Environmental Pollution, Menstrual Cycle, Endocrine Disrupting Substances, Contaminants, Biomarkers

1 INTRODUCTION

Environmental pollution comprises a wide range of physical, chemical, and biological contaminants released into the air, water, and soil through industrialization, urbanization, agriculture, transportation, and domestic activities (Britannica, 2023). Major pollutants include particulate matter (PM), gaseous pollutants such as sulphur dioxide and nitrogen oxides (World Health Organization, 2021), heavy metals, pesticides, and persistent organic pollutants (POPs) generated by industrial processes and agricultural runoff (Alengebawiy et al., 2021). More recently, endocrine-

disrupting chemicals (EDCs) and emerging contaminants, including per- and polyfluoroalkyl substances (PFAS) and microplastics, have attracted growing attention because of their environmental persistence and potential health risks (Osuolale et al., 2024).

Human exposure to these contaminants has become a major public health concern owing to their links with cardiovascular, respiratory, neurological, metabolic, and reproductive disorders (Landrigan et al., 2018; Heindel et al., 2017).

There is increasing evidence suggesting that these pollutants disrupt women's reproductive health through multiple, overlapping pathways. Endocrine disruption, oxidative stress, chronic inflammation, mitochondrial dysfunction, epigenetic modifications, and direct cellular damage all contribute to systemic effects (Gore et al., 2024; La Merrill et al., 2020). Together, these mechanisms interfere with the hypothalamic-pituitary-ovarian (HPO) axis, impair ovarian hormone production, disrupt folliculogenesis, and alter endometrial function. The consequences often manifest as menstrual irregularities (Heindel et al., 2022; Mínguez-Alarcón et al., 2022).

The menstrual cycle serves as a sensitive indicator of reproductive health, reflecting the integrated function of the hypothalamus, pituitary, ovaries, and uterus. When disrupted, it can present as irregular cycles, dysmenorrhea, amenorrhea, abnormal uterine bleeding, or anovulation, and these are potential indicators of reproductive toxicity or endocrine dysfunction (Critchley et al., 2020; Gore et al., 2024). Beyond fertility, menstrual health is closely tied to overall well-being, making the investigation of the effects of environmental pollutants on menstrual function both critical and time-sensitive.

This article is a structured narrative review that synthesizes current evidence on the relationship between environmental pollution and menstrual health. The review draws on peer-reviewed literature identified from major biomedical databases, including PubMed, Scopus, and Web of Science, as well as reference lists of relevant articles. Priority was given to recent epidemiological studies, experimental investigations, systematic reviews, and meta-analyses examining the effects of environmental pollutants on menstrual function and the underlying biological mechanisms. The evidence was synthesized narratively, with emphasis on biological plausibility, consistency of findings across studies, methodological limitations, and implications for clinical practice and future research. The primary objectives were to identify the major classes of environmental pollutants associated with menstrual dysfunction; to examine the biological mechanisms underlying these disruptions; to evaluate the existing evidence, from both epidemiological studies and experimental models, linking environmental exposures to specific menstrual disorders; and to consider additional factors, such as age, metabolic health, reproductive stage, and socioeconomic status, that may influence individual susceptibility to pollutant-induced menstrual disturbances.

2 ENVIRONMENTAL MEDIA AND EXPOSURE PATHWAYS

2.1 Waterborne Contaminants

Water contamination encompasses a range of substances, including PFAS, plasticizers, pesticides, industrial effluents, pharmaceutical residues, and heavy metals like lead and mercury. Exposure occurs primarily through drinking water but also via cooking, bathing, swimming, and other direct contact with contaminated sources. Many of these chemicals act as endocrine disruptors or reproductive toxicants. For instance, elevated mercury levels in blood or urine have been correlated with hormonal imbalances and an increased likelihood of anovulation (Henriques, M. C., et al; Pollack et al., 2021).

Despite clear laboratory evidence of hormonal disruption, real-world studies linking specific waterborne contaminants to menstrual disorders remain scarce. The challenge lies in the complexity of water as an exposure medium; it rarely contains a single pollutant but rather a dynamic mixture. Isolating individual effects is difficult. Future prospective studies incorporating high-resolution water quality monitoring and improved exposure assessment are therefore needed to clarify how waterborne pollutants influence ovarian function and menstrual physiology.

2.2 Soil-borne Contaminants and Trophic Transfer

Soil pollution accumulates over time, with persistent organic pollutants (POPs), petroleum derivatives, pesticides, and heavy metals like cadmium and arsenic entering the environment through industrial activity, mining, and intensive agriculture. Human exposure occurs through incidental ingestion of contaminated soil or dust, dermal contact, inhalation of particulate matter, and, most significantly, consumption of crops that have absorbed these chemicals from the soil. Cadmium, for example, readily enters the food chain, and chronic exposure has been associated with hormonal imbalances, impaired follicle development, and menstrual irregularities (Gollenberg et al., 2010; Wang et al., 2022).

The difficulty in assessing soil contaminants lies in tracing their contribution to menstrual disorders. Most biomonitoring studies measure total exposure through blood or urine but cannot pinpoint the source. As a result, research often relies on indirect measures, such as proximity to industrial sites, mines, or e-waste processing facilities. These proxies lack precision and risk misclassifying exposure. A more robust approach would integrate geospatial modelling, remote sensing, environmental monitoring, and biomonitoring to better isolate the role of soil-derived contaminants in menstrual dysfunction.

2.3 Airborne Contaminants and Inhalation Pathways

Air pollution consists of fine particulate matter (PM_{2.5} and PM₁₀), nitrogen oxides, sulfur dioxide, carbon monoxide, ozone, and volatile organic compounds, primarily emitted by vehicles, industrial processes, fossil fuel combustion, and wildfires. When inhaled, the smallest particles and gases can enter the bloodstream, triggering systemic inflammation, oxidative stress, and vascular damage, effects that extend far beyond the respiratory system (Landrigan et al., 2018).

Growing epidemiological evidence suggests that air pollution may also adversely affect menstrual health. Women who breathe in more nitrogen dioxide, sulfur dioxide, or fine particulate matter tend to have cycles that deviate from the usual pattern such as menstrual irregularity, shorter cycle length, and altered bleeding patterns. Certain periods of development appear to be particularly vulnerable. Exposure during adolescence, especially around menarche, has been linked to persistent menstrual irregularities later in life, highlighting this stage as a potential window of increased susceptibility (Mahalingaiah et al., 2018). Population-based cohort studies have likewise reported a higher prevalence of dysmenorrhea among women living in areas with elevated concentrations of NO, NO₂, NO_x, CO, and PM_{2.5} (Lin et al., 2021).

Despite these findings, accurately characterizing airborne exposure remains challenging. Pollutant concentrations vary across geographic regions, and studies often employ different exposure assessment methods, including land-use regression models, satellite-derived estimates, and stationary monitoring networks. In addition, differences in the definition and assessment of menstrual outcomes contribute to variability between studies. Future prospective research combining personal exposure monitoring with standardized menstrual tracking tools may provide more reliable estimates of the independent effects of airborne pollutants on menstrual health.

3 MOLECULAR AND BIOLOGICAL MECHANISMS OF ACTION

Environmental contaminants from air, water, and soil affect female reproductive physiology largely through their endocrine-disrupting properties. Many of these compounds interfere with hormone synthesis, secretion, transport, receptor binding, metabolism, and intracellular signalling. Major classes of endocrine-disrupting chemicals (EDCs) associated with menstrual health include bisphenols, phthalates, parabens, per- and polyfluoroalkyl substances (PFAS), pesticides, dioxins, and polychlorinated biphenyls (PCBs) (Gore et al., 2024; La Merrill et al., 2020). Disruption of these pathways alters the normal function of the hypothalamic–pituitary–ovarian (HPO) axis, affects gonadotropin secretion, and impairs follicular development (Heindel et al., 2022). The reproductive effects of environmental pollutants are primarily mediated through three interconnected mechanisms: endocrine disruption and hormonal imbalance, oxidative stress and mitochondrial dysfunction, and genotoxicity and cellular damage.

3.1 Endocrine Disruption and Hormonal Imbalance

Endocrine disruption is one of the principal mechanisms through which environmental pollutants negatively affects menstrual function. Many contaminants interfere with the HPO axis by altering hormone synthesis, secretion, transport, receptor binding, metabolism, and feedback regulation. Chemicals such as bisphenols, phthalates, pesticides, and PFAS can disrupt oestrogen, progesterone, androgen, thyroid hormone, and glucocorticoid signalling. These disturbances affect the pulsatile release of luteinizing hormone (LH) and follicle-stimulating hormone (FSH), leading to impaired follicular development, ovulatory dysfunction, and inadequate luteal function (Gore et al., 2024; La Merrill et al., 2020).

In addition to altering circulating hormone levels, these pollutants interfere directly with ovarian steroidogenesis. They modify the expression and activity of enzymes involved in steroid hormone synthesis, including aromatase (CYP19A1), steroidogenic acute regulatory protein (StAR), and 3 β -hydroxysteroid dehydrogenase (3 β -HSD). Disruption of these pathways impairs follicular maturation and corpus luteum function, contributing to menstrual irregularities, abnormal cycle length, and anovulation (Heindel et al., 2022; Mínguez-Alarcón et al., 2022).

3.2 Oxidative Stress and Mitochondrial Dysfunction

Oxidative stress is another major player in how pollution damages reproductive health. When women are exposed to particulate matter, heavy metals, pesticides, or plastic additives, their bodies start producing more reactive oxygen species (ROS) while their natural antioxidant defences are adversely affected. Over time, this imbalance leads to mitochondrial damage, lipid peroxidation, protein oxidation, DNA breaks, and inflammation in reproductive tissues.

In the ovaries, excess ROS disrupts follicle development, lowers egg quality, alters hormone production, and speeds up follicle death. Similar effects occur in the endometrium, where oxidative damage impairs tissue receptivity and disrupts the vascular changes required for normal menstruation. These alterations have been associated with luteal phase deficiency, menstrual irregularities, dysmenorrhea, and reduced fertility (Gore et al., 2024; Mínguez-Alarcón et al., 2022). Because oxidative stress interacts closely with endocrine and inflammatory pathways, it is considered a key mechanism through which multiple environmental pollutants exert reproductive toxicity.

3.3 Genotoxicity and Cellular Damage

Several environmental pollutants also impair reproductive health through direct genotoxic effects. Studies show that women with higher levels of heavy metals like cadmium, lead, and mercury in their bodies often have altered hormone levels, fewer healthy eggs, and trouble ovulating (Pollack et al., 2021; Wang et al., 2022). At a cellular level, these metals, along with persistent organic pollutants and other industrial chemicals, can cause DNA breaks, chromosomal abnormalities, and cell death in granulosa and endometrial cells. Over time, this kind of damage may accelerate reproductive aging.

Evidence from both in vitro and in vivo studies supports these observations. Experimental models demonstrate that environmental pollutants induce DNA damage, mitochondrial dysfunction, and epigenetic alterations that compromise germ cell integrity and ovarian function (La Merrill et al., 2020). Although findings from animal studies should be interpreted cautiously because of species differences in reproductive physiology and toxicokinetics, they provide important mechanistic evidence supporting the biological plausibility of pollutant-induced menstrual dysfunction.

4 EPIDEMIOLOGICAL AND EXPERIMENTAL EVIDENCE

4.1 Human Studies

Human epidemiological studies provide the strongest evidence linking environmental pollution with menstrual disorders, although methodological differences continue to limit causal inference. Recent studies have combined geographic exposure modelling, biomonitoring, self-reported menstrual histories, mobile health applications, and clinical reproductive assessments to investigate these associations. While exposure misclassification and reliance on self-reported menstrual outcomes remain important limitations, the overall body of evidence suggests that exposure to environmental pollutants is associated with a range of menstrual disturbances (Yesildemir and Celik, 2024; Mínguez-Alarcón et al., 2022).

Among airborne pollutants, large cohort and population-based studies have consistently associated exposure to fine particulate matter (PM_{2.5}), nitrogen dioxide (NO₂), sulphur dioxide (SO₂), and traffic-related air pollution with menstrual irregularity, dysmenorrhea, and altered cycle length (de Souza et al., 2025; Kim et al., 2025; Lin et al., 2021). Similarly, biomonitoring studies have linked elevated urinary and serum concentrations of bisphenol A (BPA), phthalate metabolites, and parabens with changes in follicular and luteal phase duration, reduced ovarian reserve, and decreased fecundability (Mínguez-Alarcón et al., 2016, 2022).

Heavy metals, including lead, cadmium, and mercury, have also been associated with altered reproductive hormone profiles, irregular menstrual bleeding, and an increased risk of anovulation (Pollack et al., 2021; Wang et al., 2022). Although findings across different populations generally point in the same direction, variations in exposure assessment, pollutant mixtures, participant characteristics, and definitions of menstrual outcomes contribute to considerable heterogeneity between studies. These limitations underscore the need for standardized exposure assessment, consistent outcome definitions, and well-designed longitudinal studies to strengthen causal inference.

4.2 Animal Studies

Animal studies have played an important role in elucidating the biological mechanisms through which environmental pollutants impair female reproductive function. Experimental studies in rodents and non-human primates have shown that exposure to endocrine-disrupting chemicals (EDCs), heavy metals, combustion by-products, and persistent organic pollutants (POPs) disrupts normal estrous cyclicity, impairs follicular development, inhibits steroid hormone synthesis, and accelerates reproductive ageing (Gore et al., 2024; Heindel et al., 2022).

Unlike observational studies in humans, experimental models allow investigators to control exposure dose, timing, and duration while examining tissue-specific effects. These studies have demonstrated pollutant-induced ovarian histopathological changes, mitochondrial dysfunction, inflammatory responses, and alterations in gene expression. They also show that exposure during prenatal development, early life, and puberty can produce long-lasting reproductive effects, supporting the concept of critical windows of susceptibility (La Merrill et al., 2020).

Despite their value, findings from animal studies cannot be directly extrapolated to humans because of differences in reproductive physiology, toxicokinetics, exposure patterns, and the distinction between the rodent estrous cycle and the human menstrual cycle. Nevertheless, these models provide important mechanistic evidence that complements epidemiological findings and helps identify biological pathways, susceptible developmental periods, and pollutants that warrant further investigation in human populations.

5 SPECIFIC MENSTRUAL CYCLE DISTURBANCES

Environmental pollution has been associated with a range of menstrual disturbances that reflect disruption of normal reproductive endocrine function. These include menstrual irregularity, dysmenorrhea, ovulatory dysfunction, amenorrhea, and abnormal uterine bleeding (AUB), all of which involve alterations in the normal regulation of the hypothalamic–pituitary–ovarian (HPO) axis (Critchley et al., 2020; Gore et al., 2024).

5.1 Menstrual Irregularity and Variable Cycle Length

Menstrual irregularity is the most frequently reported reproductive outcome in studies investigating the effects of environmental pollution. It is characterized by marked variation in cycle length, unpredictable menstrual timing, or abnormal bleeding intervals. Epidemiological studies have consistently associated exposure to ambient air pollutants, including fine particulate matter (PM_{2.5}) and gaseous pollutants, with an increased risk of irregular menstrual cycles and shortened cycle length (Kim et al., 2025). These findings are supported by multinational prospective cohort studies using mobile health applications, which have reported similar associations between long-term air pollution exposure and menstrual cycle instability (de Souza et al., 2025).

5.2 Dysmenorrhea and Uterine Hypercontractility

Dysmenorrhea is another menstrual disorder that has been linked to environmental pollution. Population-based studies have reported a higher prevalence and greater severity of painful menstruation among women living in areas with elevated concentrations of carbon monoxide (CO), nitrogen oxides (NO, NO₂, NO_x), and PM_{2.5} (Lin et al., 2021). Experimental evidence suggests that these pollutants promote inflammatory cytokine release and increase endometrial prostaglandin production, leading to enhanced uterine contractility and pain during menstruation (Gore et al., 2024; Mínguez-Alarcón et al., 2022).

5.3 Ovulatory Dysfunction and Phase Defects

Normal ovulation depends on tightly regulated hormonal signalling, making ovulatory function particularly sensitive to environmental disruption. Biomonitoring studies have associated exposure to heavy metals such as cadmium, lead, and mercury with altered reproductive hormone concentrations and an increased risk of anovulation (Pollack et al., 2021; Wang et al., 2022). Similarly, exposure to endocrine-disrupting chemicals (EDCs), including bisphenol A (BPA) and phthalates, has been linked to shortened follicular phases and impaired luteal function, suggesting disruption of normal ovulatory processes (Yesildemir and Celik, 2024).

5.4 Amenorrhea and Follicular Depletion

Amenorrhea, defined as the absence of menstruation, has received less attention in environmental epidemiology because it may arise from a variety of physiological and pathological conditions, including pregnancy, hormonal contraceptive use, and endocrine disorders. Nevertheless, mechanistic evidence suggests that severe disruption of the HPO axis, impaired steroidogenesis, ovarian toxicity, and accelerated follicular depletion caused by environmental pollutants may contribute to amenorrhea in highly exposed individuals (Gore et al., 2024; La Merrill et al., 2020).

5.5 Abnormal Uterine Bleeding (AUB)

Abnormal uterine bleeding (AUB), which includes changes in menstrual frequency, duration, regularity, and volume, has recently gained attention as a potential consequence of environmental exposure (Critchley et al., 2020). Although direct epidemiological evidence remains limited, pollutant-induced endometrial inflammation and altered progesterone-to-estrogen balance provide biologically plausible mechanisms through which environmental contaminants may contribute to abnormal bleeding patterns (Heindel et al., 2022). Future studies should adopt standardized clinical definitions of AUB to improve comparability across populations.

6 INTERACTION WITH OTHER FACTORS

The relationship between environmental pollution and menstrual dysfunction is complex and influenced by multiple biological, behavioural, and socioeconomic factors. Environmental exposures rarely occur in isolation, and individual susceptibility is shaped by both intrinsic characteristics and external conditions.

Socioeconomic status (SES) influences exposure to environmental pollutants as well as access to healthcare, nutrition, and sanitation. Women living in socioeconomically disadvantaged communities are often exposed to higher levels of environmental contaminants while also experiencing chronic psychosocial stress, poorer housing conditions, and limited access to healthcare. Together, these factors may increase susceptibility to endocrine disruption and menstrual disorders (Yesildemir and Celik, 2024; Vrijheid et al., 2021).

Lifestyle factors can further modify the reproductive effects of environmental pollutants. Cigarette smoking, alcohol consumption, diet, physical inactivity, and the use of certain medications or recreational drugs all influence reproductive health. Smoking, in particular, alters oestrogen metabolism and ovarian function and may act synergistically with endocrine-disrupting chemicals (EDCs) to increase the risk of menstrual irregularities (Mínguez-Alarcón et al., 2022). Obesity and metabolic disorders may also enhance pollutant toxicity because adipose tissue both stores lipophilic contaminants and functions as an active endocrine organ that regulates oestrogen metabolism and inflammatory cytokine production (Heindel et al., 2022; La Merrill et al., 2020).

Psychological stress and inadequate sleep are additional factors that may influence menstrual health. Chronic activation of the hypothalamic–pituitary–adrenal (HPA) axis increases cortisol secretion, suppresses gonadotropin-releasing hormone (GnRH) pulsatility, and may disrupt ovulation and menstrual cyclicity. Environmental pollutants may further aggravate these effects by promoting oxidative stress and systemic inflammation (Mínguez-Alarcón et al., 2022).

Age and reproductive stage also affect vulnerability to environmental pollutants. Adolescence is considered a particularly sensitive period because the HPO axis is still developing, whereas hormonal changes during the menopausal transition may increase susceptibility to environmentally induced menstrual disturbances (Gore et al., 2024; La Merrill et al., 2020). Women with pre-existing reproductive or endocrine disorders, including polycystic ovary syndrome (PCOS), endometriosis, thyroid disease, and infertility, may likewise be more susceptible because underlying hormonal and metabolic abnormalities can modify responses to environmental exposures (Gore et al., 2024; Mínguez-Alarcón et al., 2022).

Another important consideration is that environmental exposure usually involves complex mixtures rather than individual chemicals. People are simultaneously exposed to combinations of air pollutants, heavy metals, pesticides, PFAS, phthalates, bisphenols, and other endocrine-disrupting chemicals, which may interact through additive, synergistic, or antagonistic mechanisms. This complexity makes it difficult to attribute specific menstrual outcomes to individual pollutants and highlights the need for exposome-based approaches and advanced mixture analyses in future epidemiological research (Heindel et al., 2022; Vrijheid et al., 2021).

Recognizing these interacting factors is essential for interpreting epidemiological findings, identifying vulnerable populations, and developing effective strategies to reduce the reproductive health effects of environmental pollution.

7 BIOMARKERS OF EXPOSURE AND MENSTRUAL OUTCOMES

Accurate assessment of both environmental exposure and menstrual outcomes is essential for understanding the relationship between environmental pollution and menstrual health. Exposure biomarkers provide an estimate of the internal dose of contaminants and include parent compounds or metabolites measured in urine, blood, serum, hair, nails, breast milk, and adipose tissue. Urinary biomarkers are commonly used to assess exposure to phthalates, bisphenols, and certain pesticides because these compounds are rapidly excreted, whereas persistent pollutants such as heavy metals, organochlorine pesticides, and per- and polyfluoroalkyl substances (PFAS) are more reliably measured in blood or serum (Gore et al., 2024; Vrijheid et al., 2021). Although biomonitoring generally provides a more accurate estimate of internal exposure than ambient environmental measurements, its interpretation can be influenced by toxicokinetics, metabolism, biological half-life, and the timing of sample collection within the menstrual cycle (La Merrill et al., 2020).

Environmental measurements offer another way to estimate exposure. Researchers might track air pollutants like PM_{2.5} or NO₂, use geographic data to assess proximity to industrial sites or busy roads, or even rely on satellite imagery. These methods are useful for large-scale studies, but they have drawbacks. They do not account for individual behaviours or variations in personal exposure, which can lead to inaccuracies.

Reliable assessment of menstrual outcomes is equally important. Menstrual characteristics are commonly evaluated using validated questionnaires, menstrual tracking applications, daily menstrual diaries, and clinical assessment. One major problem is that definitions of irregular cycles, heavy bleeding, or painful periods can vary widely between studies. Standardized definitions and validated tools would improve consistency and reduce recall bias, where participants misremember details about their cycles.

Hormone levels can also provide valuable insights. Measuring oestrogen, progesterone, luteinizing hormone (LH), follicle-stimulating hormone (FSH), anti-Müllerian hormone (AMH), or thyroid hormones can reveal how the reproductive system is functioning. Markers of oxidative stress and inflammation, such as C-reactive protein (CRP) or specific enzymes, may help explain how pollutants disrupt the menstrual cycle. Since both pollutant levels and hormone concentrations fluctuate throughout the cycle, timing these measurements correctly is crucial. Combining biomarkers, hormone data, and standardized menstrual health assessments could strengthen the evidence linking pollution to reproductive health outcomes.

8 PUBLIC HEALTH IMPLICATIONS

The growing evidence linking environmental pollution with menstrual disturbances has important implications for clinical practice, public health, and environmental policy. Menstrual disorders, including irregular cycles, dysmenorrhea, abnormal uterine bleeding, and ovulatory dysfunction, can adversely affect quality of life, fertility, reproductive outcomes, and psychological well-being (Critchley et al., 2020; WHO, 2022). Although menstrual dysfunction is influenced by multiple factors, environmental pollution represents a potentially modifiable risk factor. Incorporating environmental and occupational exposure histories into routine clinical assessment may help identify women at increased risk, particularly those living or working in highly polluted environments (Carugno et al., 2022; Gore et al., 2024).

At the population level, reducing environmental pollution has the potential to improve reproductive health while also reducing the burden of cardiovascular, respiratory, and metabolic diseases (Landrigan et al., 2018; WHO, 2022). Environmental justice should also be considered, as socioeconomically disadvantaged communities often experience disproportionately high exposure to air pollution, industrial emissions, contaminated water, pesticides, and other endocrine-disrupting chemicals, contributing to persistent reproductive health inequalities (Vrijheid et al., 2021).

Addressing these challenges requires coordinated environmental and public health policies. Strengthening air quality standards, reducing industrial emissions, improving waste management practices, limiting exposure to hazardous endocrine-disrupting chemicals, and promoting sustainable agricultural practices could reduce environmental risks to reproductive health. Occupational health measures and public education aimed at minimizing environmental exposures should also form part of these strategies (UNEP, 2023; Gore et al., 2024).

Although additional longitudinal studies are needed to strengthen causal inference, the current evidence supports precautionary measures to reduce unnecessary exposure to environmental pollutants. Menstrual disturbances should also be recognized as potential early indicators of broader reproductive and endocrine dysfunction. Continued investment in biomonitoring, exposome research, and studies evaluating the effects of chemical mixtures will provide a stronger evidence base for environmental and reproductive health policies (Heindel et al., 2022; Vrijheid et al., 2021).

9 RESEARCH GAPS AND FUTURE DIRECTIONS

There are still major gaps in establishing the link between pollution and menstrual problems. Most existing research relies on cross-sectional or retrospective designs, which makes it difficult to establish whether pollution directly causes these issues. Longitudinal studies that follow individuals over time, measuring their chemical exposures while monitoring their menstrual cycles are needed. This approach could reveal patterns, identify vulnerable periods, and clarify how exposure translates into health problems (Mínguez-Alarcón et al., 2022; Vrijheid et al., 2021).

Accurately measuring exposure is another challenge. Pollution in the air does not always translate to the same levels in the body. Some chemicals, like phthalates and bisphenols, leave the system quickly, so tests only capture recent exposure rather than long-term accumulation. Better tools such as personal monitors, geographic information systems (GIS), wearable sensors, and real-time pollution mapping might provide a clearer picture of who is actually exposed and to what extent (Guo et al., 2022; Vrijheid et al., 2021).

Residual confounding and co-exposure to multiple pollutants further complicate interpretation of epidemiological findings. Factors such as socioeconomic status, diet, tobacco use, obesity, psychological stress, and pre-existing endocrine disorders independently influence menstrual function and may modify the effects of environmental contaminants. Future studies should therefore adopt exposome-based approaches together with advanced statistical methods, including weighted quantile sum (WQS) regression, Bayesian kernel machine regression (BKMR), and machine learning techniques, to evaluate the cumulative and interactive effects of complex chemical mixtures (La Merrill et al., 2020; Vrijheid et al., 2021).

Greater standardization of menstrual outcome assessment is also needed. The use of internationally accepted definitions for menstrual irregularity, dysmenorrhea, abnormal uterine bleeding, amenorrhea, and ovulatory

dysfunction would improve comparability across studies and strengthen future meta-analyses (Critchley et al., 2020). Future research should also account for differences in susceptibility across the life course, as adolescents, reproductive-aged women, and perimenopausal women may respond differently to environmental exposures (Gore et al., 2024).

Finally, greater integration of mechanistic and epidemiological research is needed to strengthen the evidence base. Combining biomarkers of endocrine disruption, oxidative stress, inflammation, and DNA damage with clinical reproductive outcomes could improve understanding of the biological pathways linking environmental pollutants to menstrual dysfunction. Intervention studies remain particularly limited. Evaluating whether reductions in environmental exposure achieved through cleaner air policies, workplace interventions, indoor air filtration, or chemical regulations lead to measurable improvements in menstrual health would provide valuable evidence to guide future public health and environmental policies (UNEP, 2023; WHO, 2025).

10 CONCLUSION

Environmental pollution is increasingly recognized as an important and potentially modifiable determinant of female reproductive health. Current evidence indicates that exposure to endocrine-disrupting chemicals, air pollutants, heavy metals, pesticides, and per- and polyfluoroalkyl substances (PFAS) can adversely affect menstrual function through multiple biological mechanisms. These include disruption of the hypothalamic–pituitary–ovarian axis, altered steroid hormone synthesis, oxidative stress, mitochondrial dysfunction, chronic inflammation, epigenetic modifications, and cellular damage, all of which may contribute to impaired ovarian function and menstrual disturbances.

Epidemiological studies consistently associate environmental exposures with menstrual irregularity, dysmenorrhea, ovulatory dysfunction, and other reproductive disorders. However, differences in study design, exposure assessment, outcome definitions, and residual confounding continue to limit causal interpretation. Although the available evidence supports a biologically plausible relationship between environmental pollution and menstrual dysfunction, further high-quality prospective studies are needed to clarify the magnitude of these associations and identify the populations at greatest risk.

Protecting menstrual health will require collaboration across environmental science, reproductive medicine, epidemiology, toxicology, and public health. Future research should prioritize longitudinal cohort studies, improved biomonitoring strategies, and exposome-based approaches to better characterize the health effects of single pollutants and complex chemical mixtures. At the same time, policies that reduce environmental pollution, strengthen occupational protections, and limit exposure to hazardous chemicals may contribute to improved reproductive health. As recognition of the menstrual cycle as an important indicator of overall health continues to grow, reducing preventable environmental exposures should become an integral component of strategies to promote women's reproductive health.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this work, the author(s) used ChatGPT for polishing the write-up and identifying reference articles. The author(s) reviewed and edited the output as needed and take full responsibility for the content of the published article.

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