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The role of Polycyclic Aromatic Hydrocarbons (PAHs) in urban air as neuroendocrine disruptors in the development of menstrual mood disorders: A narrative review

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Abstract

The increasing concentration of chemical pollutants in metropolitan air, particularly polycyclic aromatic hydrocarbons (PAHs), has generated emerging concerns regarding women's mental health and reproductive function. Although prior research has primarily focused on respiratory outcomes, the complex effects of these compounds on regulatory body systems have received comparatively limited attention. This narrative review examines the dual mechanisms of PAHs as concurrent disruptors of the nervous and endocrine systems in the pathophysiology of menstrual mood disorders, including premenstrual syndrome (PMS) and premenstrual dysphoric disorder (PMDD). Relevant literature was retrieved from Scopus, PubMed, Web of Science, and Google Scholar, with emphasis on recent publications and region-specific evidence from Iran. Analysis of the literature indicates that PAHs, through binding to aryl hydrocarbon receptors (AhR) and induction of oxidative stress in ovarian tissue, promote apoptosis of corpus luteum cells and contribute to luteal phase insufficiency. This process is associated with abrupt progesterone decline and menstrual cycle irregularities. Additionally, fine particulate matter carrying these compounds may cross the blood–brain barrier, leading to neuroinflammation and reduced expression of developmental genes in the brain, thereby lowering psychological resilience to hormonal fluctuations. Furthermore, epidemiological evidence derived from large-scale datasets and studies conducted in Tehran confirms a significant association between air pollution exposure and adverse reproductive outcomes, including stillbirth. The findings highlight the necessity of recognizing air pollution as a key environmental risk factor in the etiology of psychoneuroendocrine disorders. Implementation of emission reduction policies in urban areas represents a strategic approach to decreasing the burden of menstruation-related mood disorders.

Keywords: Polycyclic aromatic hydrocarbons, Air pollution, Menstrual mood disorders, Neuroinflammation, Premenstrual dysphoric disorder.

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Introduction

Ambient air pollution represents one of the most significant public health challenges of the present century, contributing annually to millions of premature deaths and a substantial global burden of disease (1-3). Among the wide spectrum of toxic pollutants in urban atmospheres, polycyclic aromatic hydrocarbons (PAHs), primarily generated from incomplete combustion of fossil fuels, heavy traffic, and industrial activities, have raised particular concern due to their environmental persistence and high toxicity (4, 5). This concern is intensified in densely populated industrial metropolitan areas such as Tehran, where residents are chronically exposed to elevated concentrations of fine particulate matter (PM2.5), nitrogen dioxide (NO2), and volatile organic compounds (6-8).

Local evidence indicates that pollutant levels in Tehran have reached thresholds capable of influencing sensitive pregnancy outcomes, including stillbirth (9-11). Robust scientific evidence demonstrates that PAHs function as endocrine-disrupting chemicals (EDCs) and may cross biological barriers, thereby affecting the hypothalamic–pituitary–ovarian (HPO) axis (12, 13). Recent large-scale epidemiological investigations using Mobile Health Data have confirmed that long-term exposure to particulate matter carrying these pollutants is significantly associated with menstrual cycle irregularities and alterations in cycle length among women of reproductive age (14-16). Specifically, exposure to pollutants derived from fossil fuel combustion has been linked to shortening of the luteal phase (17), potentially through induction of oxidative stress, apoptosis of granulosa cells, and impairment of corpus luteum function (18). In addition, benzo(a)pyrene (BaP), a representative compound within this group, may bind to aryl hydrocarbon receptors (AhR) and interfere with Gs protein signaling pathways, thereby disrupting steroidogenesis and altering estradiol and progesterone levels (19, 20). These compounds have also been shown to suppress expression of the steroidogenic acute regulatory protein (StAR), which plays a critical role in sex hormone synthesis (21).

Beyond reproductive effects, the systemic impact of air pollution extends to the central nervous system (CNS), which constitutes a major target of toxicity. Fine particulate matter and PAHs may penetrate the blood–brain barrier or enter via olfactory pathways, resulting in neuroinflammation and oxidative stress within neural tissue (1, 22). Animal studies have demonstrated that developmental exposure to PAHs can reduce expression of genes involved in neural function, such as the Met receptor, thereby predisposing individuals to behavioral abnormalities (23, 24). Furthermore, direct associations have been reported between air pollution exposure and increased risk of psychiatric disorders, including depression, anxiety, and suicide (1, 25).

Recent evidence also suggests that exposure to PAHs and related substitutes is associated with a spectrum of adverse reproductive outcomes, including miscarriage, premature ovarian failure (POF), and polycystic ovary syndrome (PCOS), and may negatively affect placental cell survival and differentiation (5, 25, 26). Although the independent effects of air pollution on reproductive health and mental health are well documented, the convergent role of these pollutants in the development of menstruation-related mood disorders, such as premenstrual syndrome (PMS) and premenstrual dysphoric disorder (PMDD), has received limited attention. These conditions, characterized by a psycho-neuro-endocrine nature, are likely the result of a complex interaction between abnormal hormonal fluctuations secondary to ovarian dysfunction and underlying neuroinflammatory processes within the CNS.

Accordingly, the present narrative review examines the role of PAHs in urban air as concurrent disruptors of the nervous and endocrine systems in the pathophysiology and development of menstrual mood disorders.

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Method

This study was designed and conducted as a Narrative Review. To identify relevant scientific literature, major international databases including Scopus, PubMed, Web of Science, ScienceDirect, and Google Scholar were systematically searched up to February 20, 2026 (search end date). The search was performed without time restriction and included publications in Persian and English.

The search strategy incorporated the following Persian and English keywords and their logical combinations using the Boolean operators AND and OR: polycyclic aromatic hydrocarbons (PAHs), air pollution, menstrual mood disorders, premenstrual syndrome (PMS), premenstrual dysphoric disorder (PMDD), particulate matter (PM2.5), neuroinflammation, and endocrine disruptors.

Inclusion criteria comprised original research articles (epidemiological and experimental studies) and review papers that directly examined the association between exposure to PAHs or air pollution indicators and mechanisms involved in menstrual cycle dysregulation, hormonal alterations (such as progesterone decline or corpus luteum dysfunction), and neuro–cellular and molecular pathways including oxidative stress and neuroinflammation.

Articles were excluded if they focused solely on other environmental pollutants without reference to PAHs or air pollution; addressed infertility or cancer without discussion of menstrual physiology or mood-related outcomes; consisted exclusively of psychiatric investigations without consideration of environmental risk factors; or lacked access to full text.

Eligible sources were systematically reviewed, and key findings were extracted, thematically categorized, and analytically synthesized across the relevant sections of the manuscript.

Results

Findings from the reviewed studies indicate that polycyclic aromatic hydrocarbons (PAHs) and particulate matter containing these compounds (PM2.5) influence menstrual physiology and mood through complex and concurrent peripheral (ovarian) and central (neural) mechanisms. The findings were categorized into four principal domains:

1. Disruption of Menstrual Cycle Regulation and Shortening of the Luteal Phase

Epidemiological evidence strongly suggests that exposure to air pollution disrupts the biological rhythm of the menstrual cycle. In the largest cohort study conducted using Mobile Health Data derived from the Clue application, encompassing more than 2.2 million menstrual cycles, long-term exposure to PM2.5 was significantly associated with an increased prevalence of irregular and abnormal menstrual cycles (16). More specifically, a study among urban women demonstrated that exposure to pollutants generated from fossil fuel combustion, the primary source of PAHs, was significantly associated with shortening of the luteal phase (17).

This finding has substantial clinical relevance, as a shortened luteal phase generally reflects corpus luteum insufficiency and reduced progesterone levels, which are key contributors to premenstrual syndrome (PMS). Furthermore, monitoring of urinary PAH metabolites has revealed correlations between these compounds and alterations in follicular phase length as well as luteinizing hormone (LH) and estradiol levels (20). As presented in Table 1, multiple epidemiological investigations across diverse geographic regions have demonstrated significant associations between exposure to urban air pollutants, including PAHs and PM2.5, and disturbances in female reproductive system function.

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Table 1. Summary of key epidemiological studies on the relationship between air pollution and reproductive health

Study Type / Exposure Sample	Exposure/pollutants	Key findings	References
Prospective cohort (app data), 2,220,281 menstrual cycles	Fine particulate matter (PM2.5), long-term exposure	Significant increase in the prevalence of irregular and prolonged menstrual cycles in areas with high pollution.	(16)
Case-Crossover, 5,311 stillbirths	NO_2, SO_2, PM2.5, CO – urban air of Tehran	Direct association of gaseous pollutants and particulate matter with increased risk of stillbirth (indicating reproductive toxicity).	(10)
Cross-sectional, 791 women	NO_2, CO, PM2.5 – urban air of Tehran	Increased nitrogen dioxide (NO2) was associated with increased breast tissue density (cancer risk factor).	(6)
Cross-sectional, 133 women, (daily monitoring)	PM10, SO_2 (from fossil fuel combustion)	Exposure to pollutants was specifically associated with shortened luteal phase (corpus luteum insufficiency).	(17)
Prospective cohort, 51 women	Urinary PAH metabolites (1-hydroxypyrene (1-OHP) and others)	Association of high levels of PAHs with altered follicular phase length and abnormal fluctuations in the hormones LH and estradiol.	(20)

2. Cellular Mechanisms: Oxidative Stress and Disruption of Steroidogenesis

Molecular evidence indicates that PAHs, particularly Benzo(a)Pyrene (BaP), impair the function of granulosa and luteal cells. Xu et al. demonstrated that active metabolites of PAHs induce oxidative stress and activate the p38 mitogen-activated protein kinase (p38MAPK) pathway (18), leading to cellular apoptosis and a marked reduction in progesterone levels. These mechanistic findings are fully consistent with recent reports that emphasize the central role of oxidative stress in ovarian dysfunction (15).

Degeneration of the corpus luteum directly results in decreased progesterone production, a hormone whose abrupt fluctuation or decline constitutes a primary trigger of mood symptoms during the luteal phase. In addition, PAHs interfere with protein kinase A (PKA) signaling and Gs protein–mediated pathways, thereby suppressing expression of essential steroidogenic genes such as steroidogenic acute regulatory protein (StAR) (19, 21). Reduced StAR expression limits cholesterol transport into mitochondria and consequently impairs the initiation of sex hormone synthesis.

Table 2. Summary of experimental studies (In vivo/In vitro) on the mechanisms of effect of PAHs on the nervous and reproductive systems

Study model	Biological Target/Tissue	Compound studied	Proposed mechanism and observed effects	Proposed mechanism and observed effects
In vivo (mouse) / In vitro	Corpus Luteum Granulosa cells	Benzoalphapyrene (BaP)	Induction of oxidative stress (ROS) and	(18)

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		metabolite of BPDE	activation of the p38MAPK pathway, leading to cell apoptosis and a severe decrease in progesterone.	
In vivo (rat) / In vitro	Leydig cells (steroid-producing model) Steroidogenesis genes	Benzoalphapyrene (BaP)	Suppression of StAR protein expression (vital for hormone production) via increased ROS; resveratrol had a protective effect.	(21)
In vitro (human/mouse cell lines)	LH/hCG receptor Gs protein	Benzoalphapyrene (BaP) (Environmental concentrations)	Impaired Gs protein activation and reduced cAMP production in response to gonadotropins (cell type-dependent effect).	(19)
In vivo (mouse - fetal exposure)	Brain (neocortex) Met receptor tyrosine kinase	Benzoalphapyrene (BaP)	Reduced Met gene expression in the brain, leading to impaired development of neural circuits and behavioral/cognitive deficits.	(24)
In vitro	Placental cells Throphoblasts	PAHs and BPA alternatives	Induction of cell necrosis and reduced cell viability (indicating direct tissue toxicity).	(26)

3. Neuroinflammation and Neuropsychiatric Outcomes

The findings indicate that the effects of PAHs are not confined to ovarian tissue. Particulate matter carrying PAHs may penetrate the blood–brain barrier or access the central nervous system via the olfactory nerve, leading to microglial activation and chronic neuroinflammation (1). This systemic and central inflammatory state contributes to an increased risk of psychiatric disorders, including depression, anxiety, and suicide in highly polluted regions (1, 28, 29).

Beyond established evidence, recent investigations indicate that prenatal exposure to air pollutants may disrupt expression of genes involved in neurodevelopment through alterations in exosomal signaling, thereby predisposing individuals to behavioral and mood disturbances later in life (23).

4. Local Evidence from Metropolitan Tehran

Studies conducted in Iran indicate that women residing in Tehran are exposed to levels of PAHs and other air pollutants high enough to exert measurable systemic effects on reproductive health. A recent investigation by Mohammadi demonstrated that ambient concentrations of SO_2, NO_2, and PM2.5 in Tehran were significantly associated with an increased risk of stillbirth (10), suggesting deep penetration of environmental toxicants into the reproductive system (10, 30).

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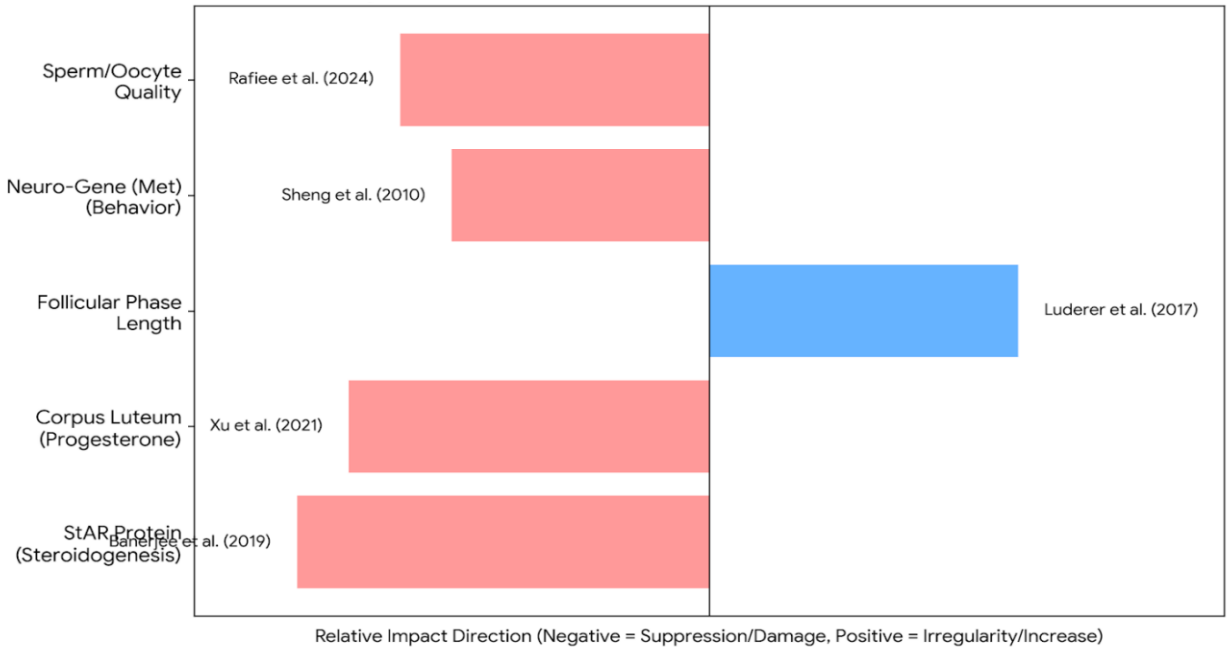

Figure 1. Overview of the Adverse Effects of Polycyclic Aromatic Hydrocarbons (PAHs) on Key Neurobiological and Reproductive Biomarkers (5, 18, 20, 21, 24).

Furthermore, significant associations have been reported between Tehran's air pollution and alterations in breast tissue characteristics, including increased mammographic density and elevated breast cancer risk, reinforcing the endocrine-disrupting properties of these pollutants in Iranian women (5, 6). Although Nakhjirgan et al. did not observe a statistically significant relationship between pollutant exposure and birth weight in Tehran, they reported elevated concentrations of aromatic compounds in maternal blood samples, indicating substantial internal exposure (31).

5. Correlation with Other Reproductive Disorders

The reviewed evidence also suggests that PAHs are associated with a broader spectrum of reproductive disorders, including polycystic ovary syndrome (PCOS) and premature ovarian failure (POF) (5). Given that women with PCOS experience higher rates of depression and anxiety, these findings further support the hypothesis that PAHs contribute to the intersection of metabolic–hormonal and psychiatric disturbances. Also, direct cytotoxic effects of PAHs and their derivatives on placental and trophoblastic cells have been documented, providing a plausible mechanistic explanation for miscarriage and implantation failure (25, 26, 32).

As illustrated in Figure 2, the central hypothesis of this review is based on a dual-pathway model in which urban pollutants converge through both peripheral (ovarian) and central (neural) mechanisms, ultimately contributing to the development of menstrual mood disorders.

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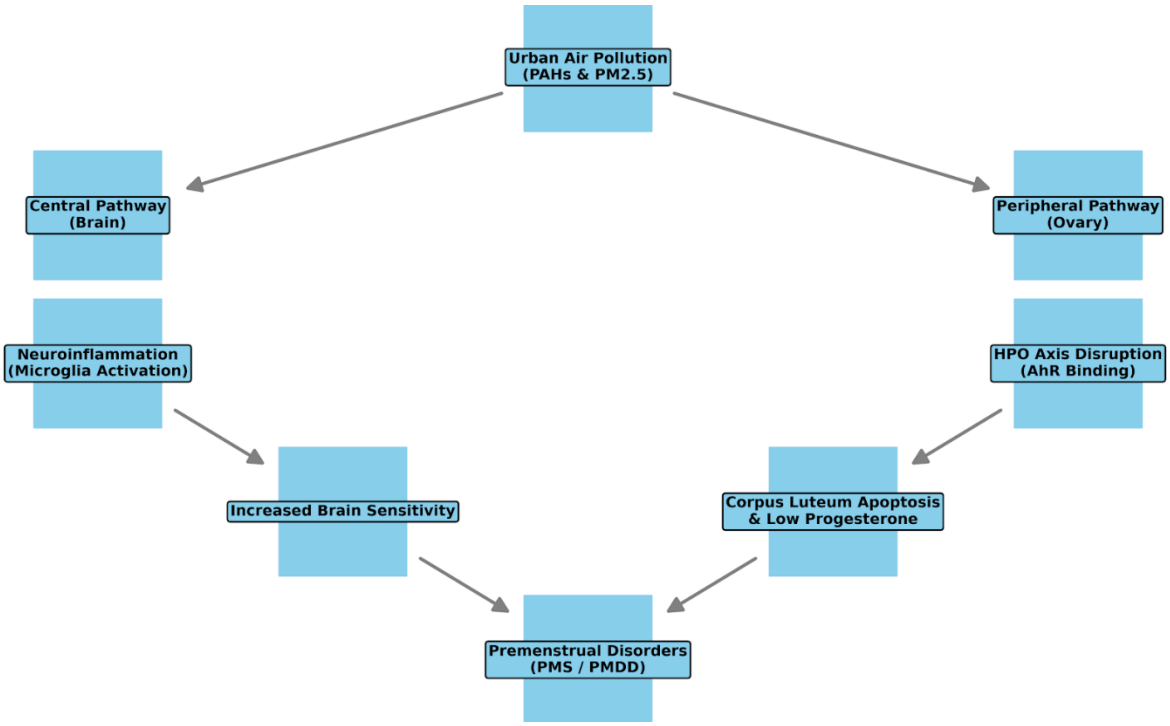

Figure 2. Proposed Conceptual Model for the Dual Mechanism of Urban Air Pollution (PAHs) in the Development of Menstrual Mood Disorders (1, 18).

Discussion

This narrative review was conducted to examine the role of polycyclic aromatic hydrocarbons (PAHs) present in urban air in the pathophysiology of menstrual mood disorders. Synthesis of the selected evidence indicates that these pollutants are not solely respiratory toxins; rather, they act as dual neuro-endocrine disruptors. The central hypothesis emerging from this review is that PAHs operate via a "two-hit" mechanism: 1) luteal phase insufficiency and abnormal progesterone fluctuations, and 2) neuroinflammation and altered neural sensitivity, together creating a milieu that promotes the onset or exacerbation of premenstrual syndrome (PMS) and premenstrual dysphoric disorder (PMDD).

1. Peripheral Mechanism: Ovarian Axis Disruption and Luteal Phase Deficiency

A key finding of this review is the detrimental effect of PAHs on corpus luteum function. The corpus luteum, a temporary gland, secretes progesterone during the second half of the menstrual cycle, and maintaining stable levels of this hormone is critical for mood regulation. Xu et al. demonstrated that Benzo(a)Pyrene (BaP) induces severe oxidative stress and activates apoptotic pathways, leading to premature luteal cell death and abrupt progesterone withdrawal (18). This mechanistic evidence supports the epidemiological findings of Merklinger-Gruchala et al. (17), who reported a significant association between air pollution exposure and shortened luteal phase in urban women. Clinically, rapid progesterone withdrawal is a primary trigger of anxiety and irritability in the premenstrual period.

Beyond structural damage, PAHs target hormone biosynthesis machinery. Banerjee et al. showed that BaP suppresses the expression of the steroidogenic acute regulatory protein (StAR), which mediates cholesterol transport into mitochondria for steroid synthesis (21). While Lazzaretti et al. suggested that acute effects of

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BaP on human granulosa cells may be limited (19), large-cohort evidence from Priyanka et al. (16), encompassing over 2.2 million menstrual cycles, confirms that chronic urban exposure produces clear cycle irregularities and hypothalamic–pituitary–ovarian (HPO) axis disruption (16).

2. Central Mechanism: Neuroinflammation and Neural Sensitization

The second dimension of this pathology involves direct effects on the central nervous system (CNS). Fine particulate matter (PM2.5) carrying PAHs can cross the blood–brain barrier or enter the brain via the olfactory nerve, triggering microglial activation and chronic neuroinflammation (1). In women with PMDD, the brain responds abnormally to natural hormonal fluctuations. Sheng et al. reported that PAH exposure reduces the expression of genes critical for neurodevelopment, such as Met. Consequently, chronic pollution-induced neuroinflammation lowers the brain's tolerance threshold, rendering it more susceptible to luteal phase hormonal fluctuations that are themselves exacerbated by pollution (24). Positive correlations between air pollution and rates of depression and suicide, as observed by Hahad et al. (1), further support this neurotoxic effect.

3. Local Evidence and Cumulative Risk in Megacities

Studies conducted in Tehran, a megacity with chronic pollution, add a concerning local dimension. Mohammadi Dashtaki et al. (10) reported that pollutant exposure was associated with increased stillbirth risk, suggesting that maternal blood concentrations of toxicants in Tehran have reached levels sufficient for cellular toxicity. When pollutants can cross the placenta and harm the fetus, they are likely capable of disrupting the delicate balance of neurotransmitters and sex hormones in non-pregnant women as well.

Additionally, Eslami et al. (6) reported effects of NO_2 on mammographic breast tissue density, and Minaee et al. (4) noted links to breast cancer, confirming the estrogenic and endocrine-disrupting (EDC) nature of Tehran's air. These local data suggest that women living in industrial Iranian cities face heightened risk and severity of PMS/PMDD symptoms due to the synergistic effects of systemic oxidative stress and hormonal disruption.

4. Association with Other Metabolic–Reproductive Disorders

Another noteworthy aspect is the overlap of these mechanisms with other female disorders. Rafiee et al. (5) highlighted the role of PAHs in the development of polycystic ovary syndrome (PCOS) and premature ovarian failure (POF). Since mood disorders (depression/anxiety) frequently co-occur with PCOS, air pollution may act as a common environmental factor in the pathogenesis of both metabolic and neuropsychiatric conditions.

Furthermore, the necrotic effects of PAHs and BPA analogs on placental cells, as reported by Jo et al. (26), indicate the tissue-destructive potential of these compounds. This cytotoxicity may also extend to the endometrial tissue, potentially exacerbating menstrual pain (dysmenorrhea), although this aspect requires further investigation (26).

Conclusion

The findings from this narrative review indicate that polycyclic aromatic hydrocarbons (PAHs) present in urban air, beyond their well-known respiratory and carcinogenic effects, act as dual neuro-endocrine disruptors, playing a significant role in the pathophysiology of menstrual mood disorders. Extensive epidemiological evidence confirms that long-term exposure to particulate matter carrying PAHs disrupts the hypothalamic-pituitary-ovarian (HPO) axis, leading to menstrual cycle irregularities and alterations in phase length.

Specifically, this review highlights that PAHs exert their effects through two parallel and synergistic mechanisms:

1. Peripheral (Ovarian) Level: PAHs and their active metabolites (e.g., BPDE) induce oxidative

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stress and apoptosis in granulosa and luteal cells, causing luteal phase deficiency and sudden drops in progesterone. These hormonal fluctuations are the primary triggers for anxiety and irritability symptoms during the luteal phase.

2. Central (Brain) Level: PAHs cross the blood–brain barrier, inducing neuroinflammation and altering the expression of developmental genes in the brain. This inflammatory state sensitizes the central nervous system to normal hormonal fluctuations, lowering women's psychological tolerance during the menstrual cycle.

Moreover, local evidence from Tehran indicates that pollutant concentrations such as PM2.5 and NO2 have reached levels that not only threaten general health but also reproductive and fetal well-being. Observed correlations between PAHs and other reproductive disorders, such as polycystic ovary syndrome (PCOS) and premature ovarian failure (POF), further support the role of air pollution as a pervasive environmental risk factor for women's health.

Based on these findings, air pollution should be considered a key environmental risk factor in the etiology and clinical management of PMS and PMDD. For public health policymakers, reducing PAH emissions in urban areas is a preventive priority to enhance women's mental health and reproductive outcomes. Future research should include longitudinal cohort studies in polluted Iranian cities to precisely assess the correlation between serum PAH levels and the severity of premenstrual dysphoric symptoms, providing more direct clinical evidence.

Conflict of Interest: None.

Declaration

During the preparation of this work the authors used intelligence (AI) tool in order to improve the readability and language of the manuscript.

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