



IMPACT OF ENVIRONMENTAL FACTORS ON FEMALE FERTILITY

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Abstract The female reproductive system is a delicate mechanism that is influenced by numerous factors. Changes occurring at any stage of gametogenesis may negatively affect the ability to conceive. Modern lifestyle, environmental problems, social and behavioral trends, and psychological factors all leave a significant imprint on reproductive function in general and on ovarian reserve in particular. Although female fertility possesses a certain capacity to adapt to changes in lifestyle, environmental pollution, social behavior, and other factors, special attention should be paid to age-related risk factors, surgical interventions on the adnexa, intensive physical activity, normalization of work-rest schedules, and exposure to toxins.

Keywords: reproduction, reproductive age, fertility, infertility, exogenous factors, women.

ВЛИЯНИЕ ВНЕШНЕЙ СРЕДЫ НА ФЕРТИЛЬНОСТЬ ЖЕНЩИНЫ

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Аннотация Репродуктивная система женщины – тонкий механизм, который подвержен влиянию множества факторов. Изменения на любом этапе



гаметогенеза могут негативно сказываться на способности к зачатию. Жизнь в современном мире, экологические проблемы, социальные поведенческие тренды, психологические факторы накладывают определённый отпечаток на детородную функцию в целом и овариальный резерв в частности. Хотя женская фертильность обладает определённой способностью адаптироваться к новому образу жизни, загрязнению окружающей среды, социальному поведению и другим факторам, следует серьёзно относиться к возрастным факторам риска, хирургическим вмешательствам на придатках, интенсивным физическим нагрузкам, нормализации режима труда и отдыха, а также к употреблению токсинов.

Ключевые слова: репродуктивность, фертильный возраст, бесплодие, экзогенные факторы, женщина.

Relevance Environmental factors undoubtedly affect human physiology, and this influence may be either beneficial or harmful. Environmental factors include climate, temperature, seasonality, radiation, pollutants in water, air, and soil, occupational conditions, dietary factors, endocrine-disrupting chemicals (EDCs), as well as alcohol, tobacco, and narcotic substances. Environmental factors not only influence biological functions but also shape the social and behavioral environment of individuals [1].

A considerable number of chemicals present in the environment possess endocrine-disrupting properties. According to the definition of the World Health Organization, an endocrine-disrupting chemical (EDC) is an exogenous substance or mixture that alters the function of the endocrine system and consequently causes adverse health effects in an intact organism, its offspring, or (sub)populations [2]. Moreover, many substances not officially recognized as EDCs may exert similar effects. EDCs demonstrate disruptive activity both individually and as components of mixtures, affecting hormonal signaling pathways. Their activity is mediated



through binding to hormone receptors, where they may act as agonists or antagonists, stimulating or inhibiting receptor activity, respectively.

In addition, EDCs interfere with enzymatic functions, induce oxidative stress, and cause epigenetic and even transgenerational changes. In both men and women, a negative correlation has been identified between environmental EDC exposure and reproductive function. Most current studies have focused on major categories of EDCs, including heavy metals (cadmium, lead, mercury, arsenic), plasticizers (bisphenols and phthalates), pesticides, parabens, industrial chemicals and their by-products (e.g., brominated flame retardants, polychlorinated biphenyls, dioxins), antibacterial agents (triclosan), perfluorochemicals, and pharmaceuticals such as diethylstilbestrol.

Excessive physical activity has also been associated with an increased risk of infertility due to menstrual cycle disturbances, regardless of obesity status. However, restoration of ovulation has been reported following moderate physical exercise and/or weight reduction in obese women. Ovulatory dysfunction has been observed in women consuming lower amounts of mono- and polyunsaturated fatty acids (PUFAs) compared with trans fats. This phenomenon has been attributed to increased inflammatory activity and insulin resistance [3].

A high intake of animal protein has been associated with ovulatory infertility. Conversely, consumption of plant-based proteins appears to protect against ovulatory dysfunction, likely due to improved insulin sensitivity. For example, among 2,145 women in Spain, the highest adherence to the Mediterranean diet was associated with a 44% reduction in the risk of impaired conception compared with women showing the lowest adherence to this dietary pattern. Furthermore, exposure to advanced glycation end-products (AGEs), commonly generated through the consumption of highly processed foods (fast food), has been shown to negatively affect ovarian function. Intraovarian accumulation of AGEs promotes inflammation, oxidative stress, insulin resistance, and subsequent ovulatory dysfunction [4].



Regarding occupational factors, working more than 52 hours per week was associated with infertility among Korean women under 40 years of age. Infertile women were found to have significantly higher serum concentrations of bisphenol A (BPA) compared with women exhibiting normal fertility. Elevated serum lead (Pb) concentrations among women in Bangladesh were positively correlated with an increased risk of idiopathic infertility. Cadmium (Cd) concentrations measured in endometrial biopsies collected between days 20 and 24 of the menstrual cycle (the expected implantation period) were significantly higher in women with idiopathic infertility compared with fertile women. Cadmium-induced reproductive toxicity has been attributed to mechanisms including ovulatory disruption, inhibition of steroidogenesis, endometrial dysfunction, and implantation failure [5].

Urinary concentrations of parabens (ethyl- and methylparaben) in women before conception were associated with reduced conception rates during one year of follow-up, resulting in a 34% decrease in couple fecundity within the general population. Among industrial chemicals and by-products, researchers identified a linear relationship between serum concentrations of low-chlorinated polychlorinated biphenyls (PCBs) and the risk of anovulation. In the Seveso Women's Health Study, a tenfold increase in serum 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) concentration was associated with approximately a twofold increase in infertility risk [6].

Prenatal exposure to diethylstilbestrol (DES) among 4,653 women was associated with an increased cumulative risk of infertility throughout reproductive life. Studies have demonstrated that many of the most extensively investigated EDCs, including lead, DEHP, BPA, PCBs, parabens, and triclosan, alter both the composition and functionality of the gut microbiota. These alterations may impair female fertility through mechanisms affecting estrogen metabolism, genital tract microbiota, and immune system regulation, thereby contributing to disorders such



as polycystic ovary syndrome, endometriosis, and premature ovarian insufficiency [7].

The reproductive lifespan of women begins at puberty and ends with menopause following depletion of the ovarian follicle pool that constitutes the ovarian reserve. Ovarian reserve is the cornerstone of female fertility. Clinical assessment commonly includes a woman's age, antral follicle count (AFC), anti-Müllerian hormone (AMH) levels, and basal reproductive hormone levels, including follicle-stimulating hormone (FSH), luteinizing hormone (LH), and estradiol.

Numerous studies have identified various determinants that negatively affect ovarian reserve and female fertility. However, these factors should be evaluated comprehensively due to their interactions and the complexity of interpretation.

Environmental toxins may trigger the formation of reactive oxygen species (ROS), including free radicals, oxygen ions, and hydrogen peroxide within cells. Oxidative stress develops when cellular antioxidant defenses become overwhelmed by excessive ROS production. Strong evidence indicates that ROS participate in the initiation of apoptosis in antral follicles. The role of oxidative stress in the pathogenesis of premature ovarian insufficiency has also been confirmed [7,8].

Epigenetic modifications may result from environmental pollutant-induced alterations in DNA methylation, thereby affecting ovarian function. For instance, bisphenol A, widely used in plastic manufacturing, can remove methyl groups and suppress enzymes responsible for DNA methylation.

According to numerous scientific studies, tobacco smoking has multiple adverse effects on female reproductive function, including detrimental impacts on ovarian function, induction of germ-cell mutations, increased risk of miscarriage, and poorer outcomes of assisted reproductive technology cycles [9].

Tobacco toxins negatively affect the follicular pool, accelerating follicular atrophy and atresia, reducing follicle numbers, altering reproductive hormone levels,



and ultimately decreasing fertility. Smoking has been associated with reduced serum estradiol and estriol concentrations, elevated testosterone levels, and a tendency toward increased FSH levels. Additional adverse effects of tobacco toxins include increased apoptosis and necrosis in various tissues, as well as enhanced apoptosis in primordial germ cells differentiated from human stem cells in vitro [30]. These effects may manifest as increased follicular apoptosis and ovarian dysfunction. Indeed, polycyclic aromatic hydrocarbons contained in cigarette smoke are toxic to ovarian follicles, as demonstrated in both animal models and human studies [10].

Alcohol consumption is also considered a risk factor for reduced female fertility. The biological mechanisms through which alcohol impairs reproductive function remain insufficiently understood. One hypothesis suggests that alcohol reduces fertility through alterations in endogenous hormone concentrations. Another theory proposes direct detrimental effects on oocyte maturation, early blastocyst development, and implantation [3, 6].

Obesity, a disease closely associated with lifestyle, has become a global health problem. Currently, more than two billion people worldwide suffer from overweight and obesity. Obesity negatively affects female reproductive function through disturbances in hypothalamic-pituitary-ovarian axis regulation, reduced oocyte quality, and impaired endometrial physiology. These associations have been demonstrated in a cohort of more than 47,000 Danish women and confirmed by Russian researchers. Obesity contributes to menstrual irregularities and increases the frequency of anovulatory cycles. Evidence suggests that reduced fertility in obese women may, in part, be associated with diminished ovarian reserve [10].

Chronic stress is another factor adversely affecting endocrine function. Individual responses to increased physical and psychological stress may alter regulatory mechanisms through elevated corticosteroid levels, which subsequently influence the hypothalamic-pituitary-ovarian axis [4, 8].



Surgical interventions involving pelvic organs have a pronounced negative effect on ovarian reserve because a portion of ovarian tissue may be removed during surgery, and blood supply to reproductive organs may be compromised. A Chinese cohort study involving 8,323 women demonstrated through both univariate and multivariate analyses that adnexal surgeries significantly reduced ovarian reserve indicators such as AFC and AMH among women aged 20–30 and 31–36 years [2].

Adnexal surgery may impair ovarian blood supply and antral follicle maturation, while ovarian tissue removal during enucleation of benign or malignant ovarian tumors reduces ovarian cortical tissue volume, ultimately leading to a decline in ovarian reserve [5].

In conclusion, female fertility is significantly influenced by a complex interaction of environmental, lifestyle, occupational, and medical factors. Exposure to endocrine-disrupting chemicals, heavy metals, tobacco smoke, alcohol, obesity, chronic stress, excessive physical activity, and pelvic surgical interventions can adversely affect ovarian reserve, hormonal regulation, ovulation, implantation, and overall reproductive potential. The mechanisms underlying these effects include oxidative stress, endocrine disruption, epigenetic modifications, impaired folliculogenesis, and altered hypothalamic-pituitary-ovarian axis function. Although the female reproductive system possesses a certain adaptive capacity, prolonged or intense exposure to harmful environmental factors may lead to a substantial decline in fertility and an increased risk of infertility. Therefore, prevention of toxic exposures, promotion of healthy lifestyle habits, early identification of reproductive risk factors, and preservation of ovarian reserve should be considered essential components of reproductive healthcare for women of reproductive age.

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