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Lifestyle Factors and Female Infertility: A Review of the Evidence on Diet, Exercise and Environmental Toxins

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ABSTRACT

Female infertility, which affects an estimated 10-15% of women of reproductive age worldwide, is not solely a consequence of immutable biological factors; it is profoundly modulated by lifestyle determinants. Emerging epidemiological and mechanistic research highlights the critical impact of nutrition, physical exercise, and environmental exposures in determining reproductive capacity. This review compiles current research on these changeable domains, including large-scale cohort studies, randomized trials, and experimental studies. Dietary patterns high in unrefined plant-based foods, omega-3 fatty acids, and antioxidant micronutrients, such as the Mediterranean diet, are consistently linked to increased fecundity and assisted reproductive technology outcomes, whereas energy-dense, ultra-processed diets exacerbate metabolic dysregulation, ovulatory dysfunction, and impaired endometrial receptivity. Similarly, moderate-intensity physical activity promotes hormonal homeostasis, insulin sensitivity, and ovulatory regularity, but excessive or energy-deficient exercise can cause hypothalamic amenorrhea and monthly irregularities. Parallel to these behavioural influences, ubiquitous environmental contaminants, particularly endocrine-disrupting chemicals such as bisphenols, phthalates, and persistent organic pollutants, along with ambient air pollution, have been implicated in diminished ovarian reserve, impaired folliculogenesis, altered steroidogenesis, and reduced implantation success, which are mediated through hormonal interference, oxidative injury, and epigenetic perturbations. By integrating insights across nutritional, behavioral, and environmental domains, this review elucidates the multifactorial, interdependent nature of lifestyle–reproduction interactions. We further highlight translational implications for preconception counselling, public health policy, and future research priorities aimed at embedding lifestyle modifications into comprehensive infertility care.

INTRODUCTION

Infertility represents a deeply personal yet increasingly common challenge: approximately one in six adults worldwide face difficulty conceiving, a burden that cuts across borders, cultures, and income levels. The World Health Organization estimates a lifetime prevalence of infertility at roughly 17.5%, reinforcing that this issue is neither rare nor exclusive to any particular demographic [1]. According to the Global Burden of Disease study, over 110 million women were affected by infertility in 2021, nearly doubling the figure reported in 1990, with disparities emerging across regions and socioeconomic brackets [2].

Beyond the statistical weight, infertility carries profound emotional, social, and economic costs. Women often bear the brunt of societal pressures, stigma, and psychological distress, even though root causes frequently span far beyond any individual's control [3]. These personal narratives underscore the urgency of addressing infertility not just medically, but holistically.

Lifestyle factors such as what we eat, how we move, and what we breathe or absorb, have emerged as powerful influences on reproductive capacity. Decades of research have begun to unearth how dietary quality, fitness habits, and exposure to environmental toxins, including endocrine-disrupting chemicals and air pollutants, interplay with the complex biology of ovarian function, hormonal regulation, and early embryo development [4]. While the evidence varies in strength, the cumulative picture suggests that these modifiable elements are far from incidental, they can tip the scales toward or away from fertility [5].

This review aims to critically synthesize current scientific evidence on the interplay between diet, physical activity, and environmental toxins in shaping female reproductive health, with a particular focus on infertility outcomes. Specifically, it seeks to (i) evaluate how dietary patterns, nutrient composition, and body weight regulation influence hormonal balance, ovulatory function, and conception rates; (ii) assess the benefits and risks of varying intensities and modalities of physical activity on reproductive physiology; and (iii) examine the mechanisms and reproductive consequences of exposure to endocrine-disrupting chemicals, persistent organic pollutants, and air pollutants. By integrating mechanistic insights with epidemiological data, the objective is to provide a comprehensive, evidence-based resource for clinicians, fertility specialists, and public health practitioners, offering actionable recommendations to incorporate lifestyle modification into infertility prevention and management strategies.

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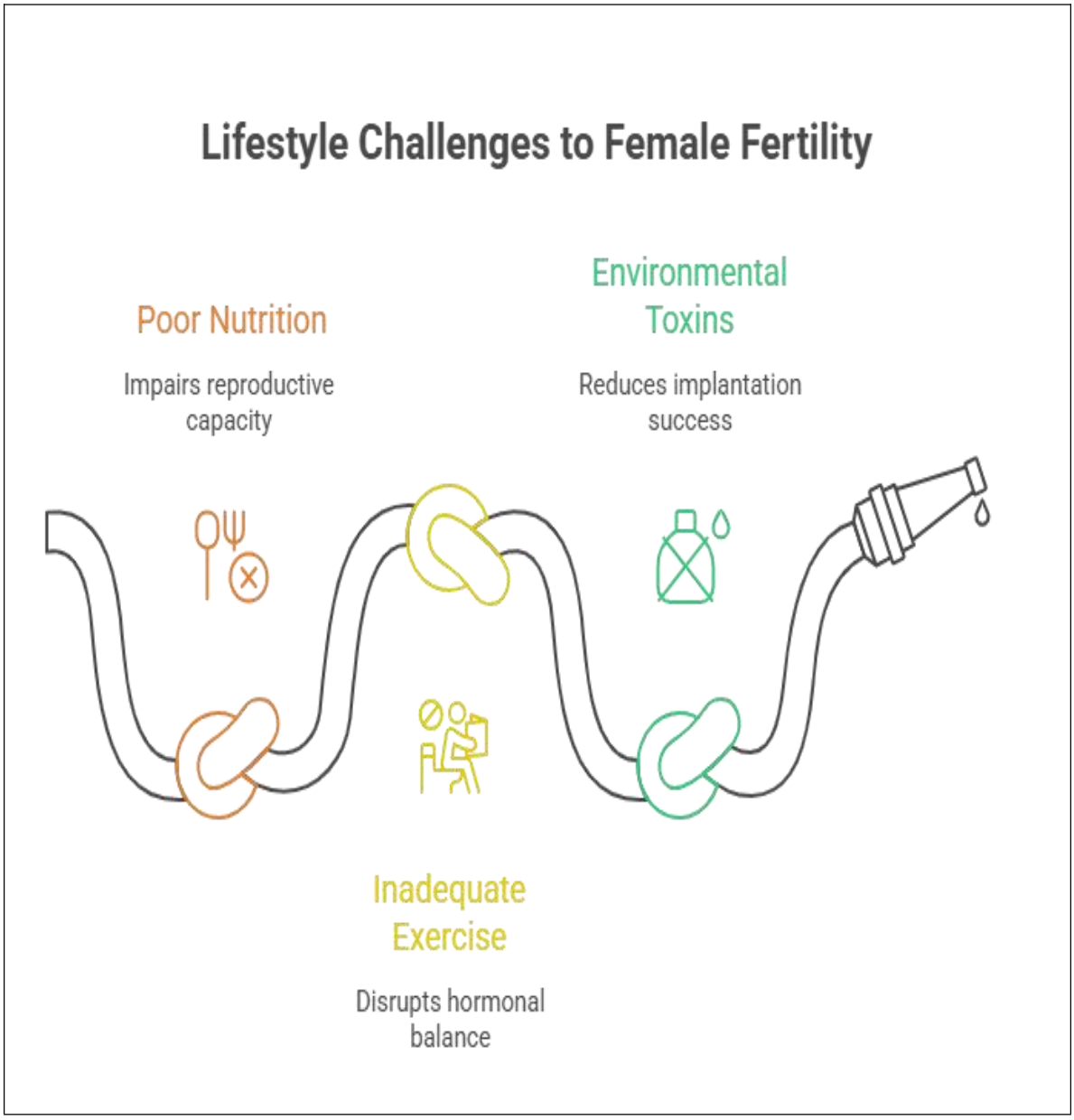

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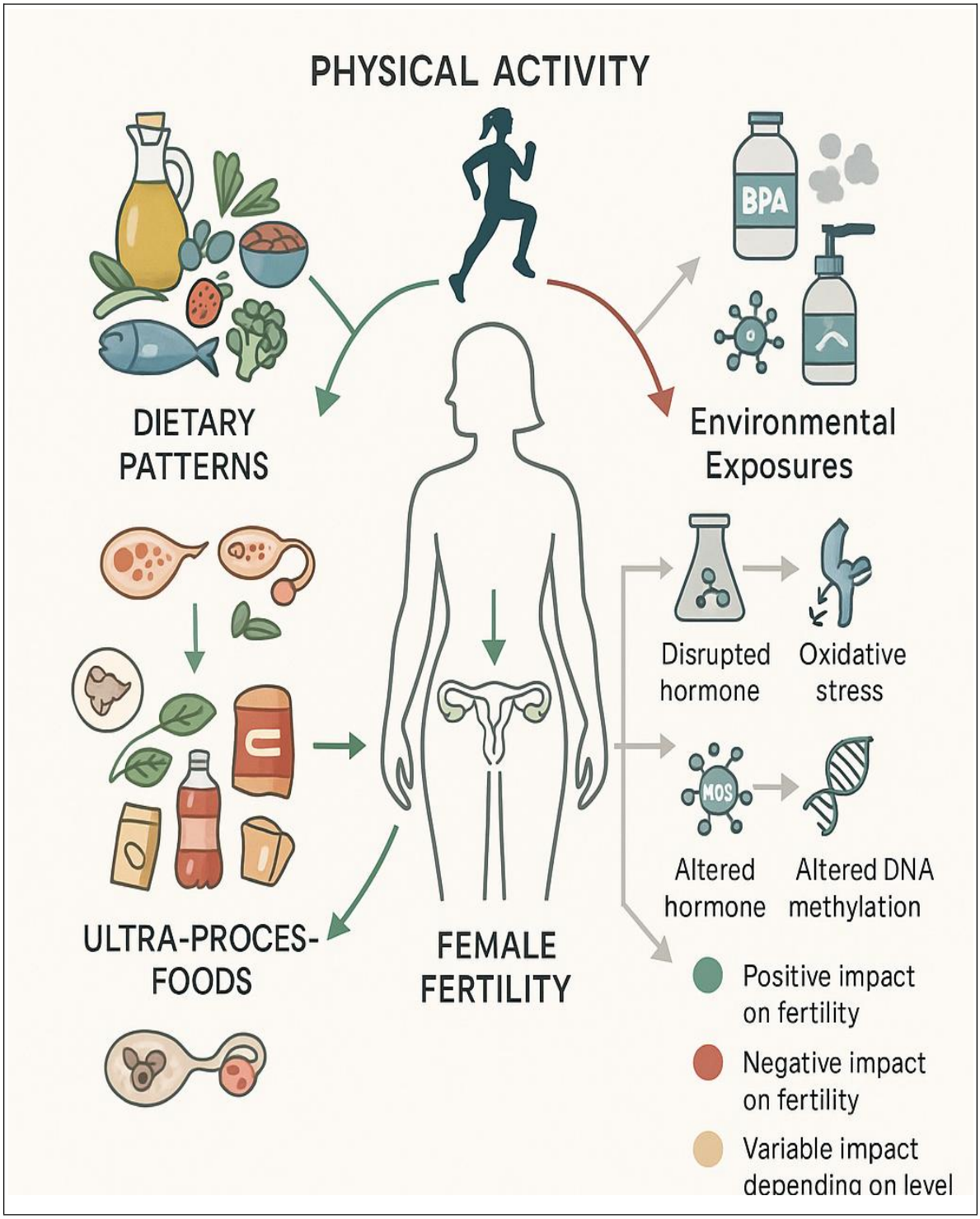

GRAPHICAL ABSTRACT, LIFESTYLE FACTORS AND FEMALE INFERTILITY: A REVIEW OF THE EVIDENCE ON DIET, EXERCISE AND ENVIRONMENTAL TOXINS

DIETARY PATTERNS AND FECUNDITY

(a) MEDITERRANEAN DIET AND FERTILITY OUTCOMES

Imagine a diet rooted in sun-drenched Mediterranean life, olive oil, vibrant vegetables, legumes, whole grains, fish, nuts, and seasonal fruit, rich in mono- and polyunsaturated fats, antioxidants, and micronutrients. This isn't just a feast for the senses; it's emerging as a beacon for fertility. Several observational studies and meta-analyses have linked higher adherence to Mediterranean-style dietary patterns with improved pregnancy rates and live births, particularly in women undergoing assisted reproductive technologies (ART). For instance, a systematic review and meta-analysis found that among ART patients, those with higher adherence to the Mediterranean diet had about 1.9 times higher odds of pregnancy or live birth compared to those with lower adherence (OR 1.91; 95% CI 1.14-3.19) [6]. Similarly, in cohort research, women in the highest tertile of Mediterranean diet scores achieved significantly higher clinical pregnancy and live birth rates than those in the lowest tertile, especially under age 35 [7]. These signals suggest that prioritizing wholesome, minimally processed foods abundant in healthy fats and vegetal diversity may support reproductive resilience.

(b) WESTERN DIET AND ADVERSE REPRODUCTIVE OUTCOMES

By contrast, the "Western diet", laden with ultra-processed foods, red and processed meats, refined grains, sugary drinks, and saturated fats, casts a shadow on reproductive health. Its metabolic consequences, including insulin resistance, chronic inflammation, and hormonal disruption, are well-established. One review highlighted how excessive saturated fat can perturb ovarian steroidogenic pathways and ovulatory function, while simple carbohydrates fuel hyperinsulinemia that fosters anovulation [8, 9].

Prospective cohort data echo these mechanisms: dietary patterns high in junk foods, solid oils, sweets, and refined grains were associated with significantly diminished odds of achieving clinical pregnancy during ART (adjusted OR as low as 0.09 in certain studies) [10]. Even short-term exposure to Western-style diets in animal models has shown deleterious effects on follicular health and oocyte quality. Collectively, the data point toward a paradigm where dietary quality, in both composition and processing, can actively tip the scales toward or away from fertility.

(c) ALCOHOL, CAFFEINE AND OTHER DIETARY FACTORS

Alcohol and caffeine; two ubiquitous dietary components, have attracted considerable attention in

fertility research for their potential to influence reproductive outcomes. Evidence suggests that heavy or chronic alcohol consumption (>5-7 drinks/week) can prolong time-to-pregnancy, impair ovulatory function, and modestly reduce live birth rates in women undergoing assisted reproductive technologies (ART), whereas light-to-moderate intake appears to exert minimal or inconsistent effects, especially when consumption is reduced during periconceptional periods [11]. Caffeine, in moderate amounts (<200 mg/day), is generally not associated with significant reductions in fecundability or ART success, although high intake and caffeine from sugar-sweetened soft drinks have been linked to increased risks of ovulatory disorders and metabolic dysregulation [12].

Other dietary factors, such as high consumption of refined sugars, trans fats, and ultra-processed foods, may exacerbate insulin resistance, systemic inflammation, and hormonal imbalance, while nutrient-rich diets and targeted supplementation, particularly with folic acid, can support reproductive health when integrated into an overall balanced nutritional framework (Figure 1). Collectively, these findings reinforce that moderation, nutrient density, and reduced intake of metabolic disruptors form the cornerstone of dietary strategies to optimize female fertility [13].

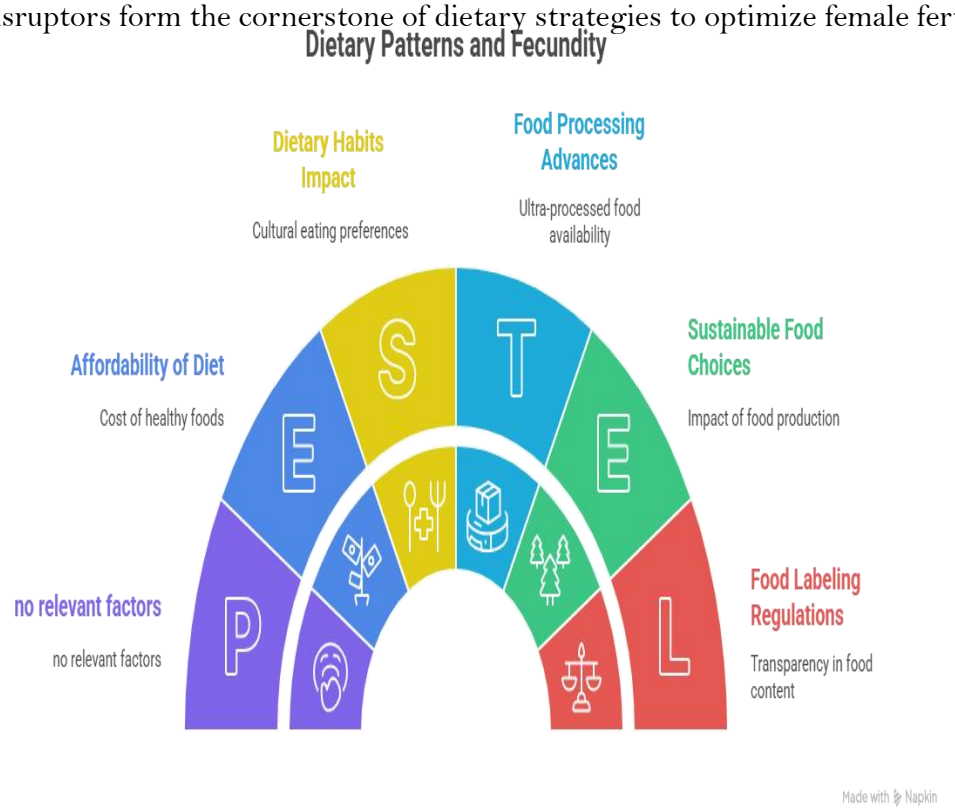

FIGURE 1: DIETARY PATTERNS AND FECUNDITY, EFFECTS OF MEDITERRANEAN DIET AND FERTILITY OUTCOMES

PHYSICAL ACTIVITY AND FEMALE INFERTILITY

(a) BENEFITS OF MODERATE EXERCISE

Moderate, regular physical activity, characterized by approximately 150 minutes per week of brisk walking, cycling, swimming, or similar exertion, has been consistently associated with favorable reproductive outcomes through multiple physiological pathways. By enhancing insulin sensitivity, improving lipid metabolism, and reducing low-grade systemic inflammation, moderate exercise helps maintain hormonal equilibrium within the hypothalamic-pituitary-ovarian (HPO) axis [14]. It supports optimal gonadotropin secretion, stabilizes luteal phase progesterone production, and promotes regular ovulatory cycles. Additionally, its role in weight regulation mitigates the adverse effects of central adiposity on ovarian function, particularly in women with metabolic risk factors [15]. In both natural conception and assisted reproductive technology (ART) contexts, these benefits can translate into improved fecundability, higher clinical pregnancy rates, and better live birth outcomes when paired with adequate nutrition and rest [16].

(b) RISKS OF EXCESSIVE EXERCISE

While movement is essential for reproductive health, pushing the body beyond its energetic limits can have the opposite effect. High-intensity or prolonged endurance training, particularly when combined with inadequate caloric intake, can disrupt the delicate hormonal signaling of the HPO axis, suppressing gonadotropin-releasing hormone (GnRH) pulsatility and subsequently luteinizing hormone (LH) and follicle-stimulating hormone (FSH) secretion (Figure 2). This cascade often manifests clinically as functional hypothalamic amenorrhea, characterized by menstrual irregularities or complete cycle cessation, diminished estrogen production, and impaired folliculogenesis [17].

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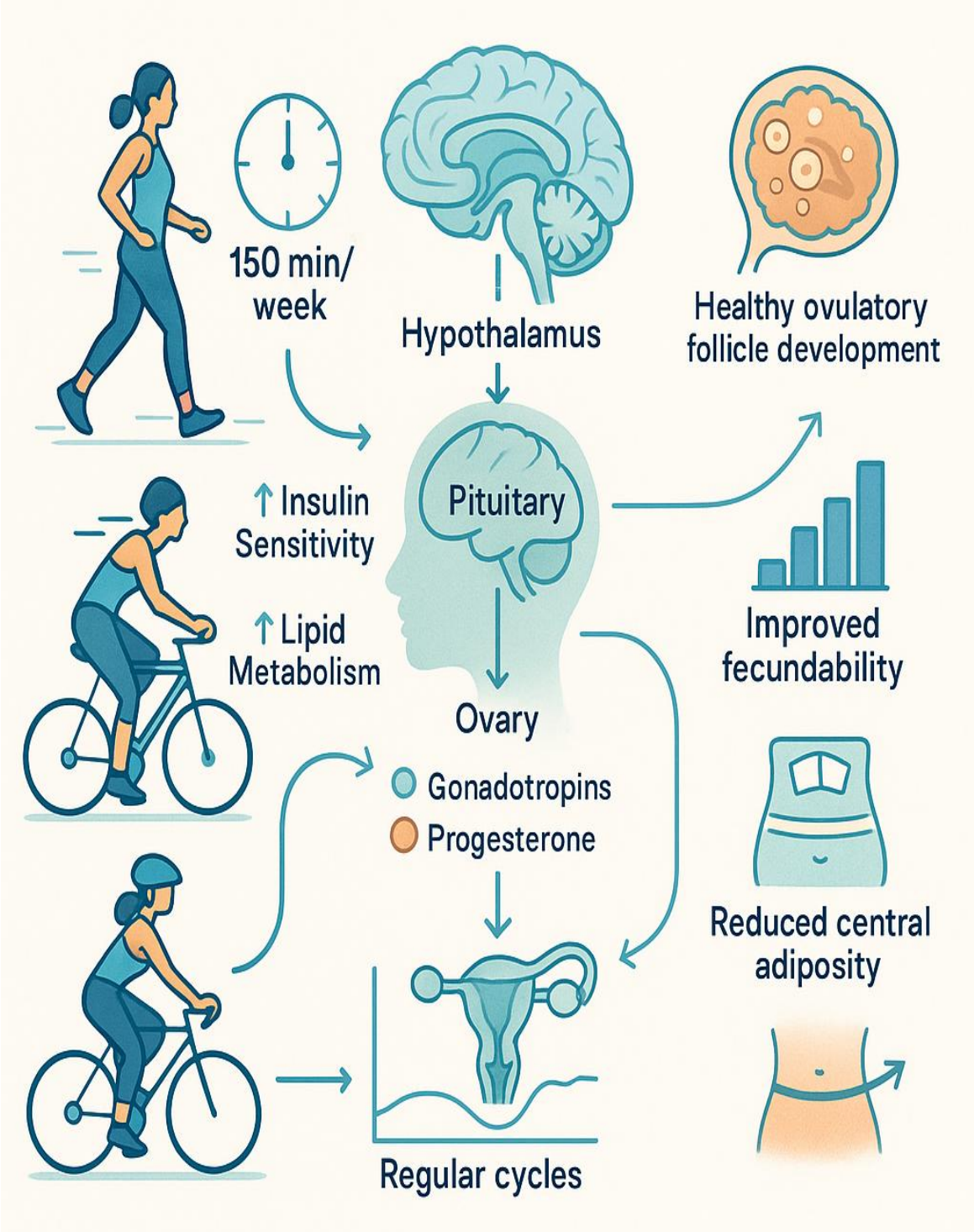

FIGURE 2: LIFESTYLE FACTORS, PHYSICAL ACTIVITY AND FEMALE INFERTILITY BENEFITS AND DRAWBACKS

TABLE 1: SUMMARY OF EVIDENCE ON DIETARY PATTERNS AND FEMALE FERTILITY

Dietary Pattern	Key Components	Reported Effects on Fertility
Mediterranean Diet	High intake of fruits, vegetables, whole grains, legumes, fish, olive oil; moderate dairy; low red meat	Improved ovulatory function, increased IVF success rates, reduced oxidative stress
Western Diet	High processed meats, refined grains, saturated fats, sugar-sweetened beverages	Increased risk of ovulatory infertility, metabolic dysfunction, poorer ART outcomes
Plant-Based Diet (balanced)	Predominantly plant proteins, unsaturated fats, fiber-rich carbs	Improved insulin sensitivity, lower risk of ovulatory disorders
High Glycemic Load Diet	Refined carbs, sugary foods, low fiber	Increased insulin resistance, higher ovulatory infertility risk

(C) EXERCISE IN SPECIFIC CONDITIONS

In reproductive disorders such as polycystic ovary syndrome (PCOS), endometriosis, and certain ovulatory dysfunctions, tailored exercise prescriptions can serve as an adjunctive therapeutic strategy. For women with PCOS, moderate aerobic and resistance training improves insulin sensitivity, reduces androgen excess, and can restore more predictable ovulatory cycles. In endometriosis, low-impact activities such as yoga, Pilates, and swimming may help alleviate chronic pelvic pain and modulate inflammatory pathways without exacerbating symptom. For women with anovulatory disorders unrelated to metabolic syndrome, gentle, consistent exercise can improve cardiovascular fitness and mood while avoiding the HPO-axis suppression seen in high-intensity regimens [18].

ENVIRONMENTAL TOXINS AND FEMALE INFERTILITY

(a) ENDOCRINE-DISRUPTING CHEMICALS (EDCS)

Endocrine-disrupting chemicals such as phthalates, bisphenol A (BPA), and parabens are pervasive in plastics, personal care products, and food packaging, posing insidious threats to female reproductive health. These compounds can mimic or antagonize endogenous hormones, binding to estrogen or androgen receptors and perturbing the hypothalamic–pituitary-ovarian (HPO) axis [19].

Chronic exposure has been linked to anovulation, altered menstrual cyclicity, diminished ovarian reserve, and poorer in vitro fertilization (IVF) outcomes. Phthalates may accelerate follicular atresia, while BPA can interfere with oocyte meiotic spindle formation, leading to compromised embryo quality. Parabens, though less studied, have been associated with subtle yet significant disruptions in ovarian steroidogenesis [20]. Given their ubiquity and bioaccumulative nature, minimizing exposure to these chemicals remains a pressing public health and reproductive medicine priority.

(b) PERSISTENT ORGANIC POLLUTANTS (POPS) AND HEAVY METALS

Persistent organic pollutants, including pesticides, dioxins, and polychlorinated biphenyls (PCBs), and toxic metals such as mercury and lead represent a formidable class of reproductive toxicants due to their environmental persistence, lipophilicity, and ability to bioaccumulate in human tissues. These agents can cross the placental barrier, disrupt steroid hormone synthesis, and impair folliculogenesis by inducing oxidative stress and mitochondrial dysfunction in ovarian cells. Epidemiological studies have linked pesticide exposure to longer time-to-pregnancy and higher miscarriage rates, while elevated mercury and lead levels have been associated with luteal phase defects, altered gonadotropin secretion, and reduced fecundability [21, 22]. Dioxins and PCBs may exert transgenerational effects, compromising not only maternal fertility but also offspring reproductive capacity. Mitigating exposure through policy regulation, dietary interventions, and occupational safety measures is critical in safeguarding female reproductive potential.

TABLE 2: COMMON ENVIRONMENTAL TOXINS IMPLICATED IN FEMALE INFERTILITY

Toxin Type	Examples	Primary Sources	Mechanism of Reproductive Impact	References
Endocrine-Disrupting Chemicals (EDCs)	Phthalates, Bisphenol A (BPA), Parabens	Plastics, cosmetics, food packaging	Mimic or block hormone action, disrupt HPO axis, impair folliculogenesis	Modica et al., 2023
Persistent Organic Pollutants (POPs)	Pesticides, Dioxins	Agricultural runoff, contaminated food	Bioaccumulate in adipose tissue, oxidative damage to oocytes	Lefebvre et al., 2023

Heavy Metals	Mercury, Lead, Cadmium	Industrial emissions, seafood, contaminated water	Mitochondrial dysfunction, oxidative stress, DNA damage in oocytes	Xiao and Lai, 2025
Air Pollutants	PM2.5, NO_2, O_3	Vehicle emissions, industrial pollution	Systemic inflammation, hormonal dysregulation	Bain et al., 2023

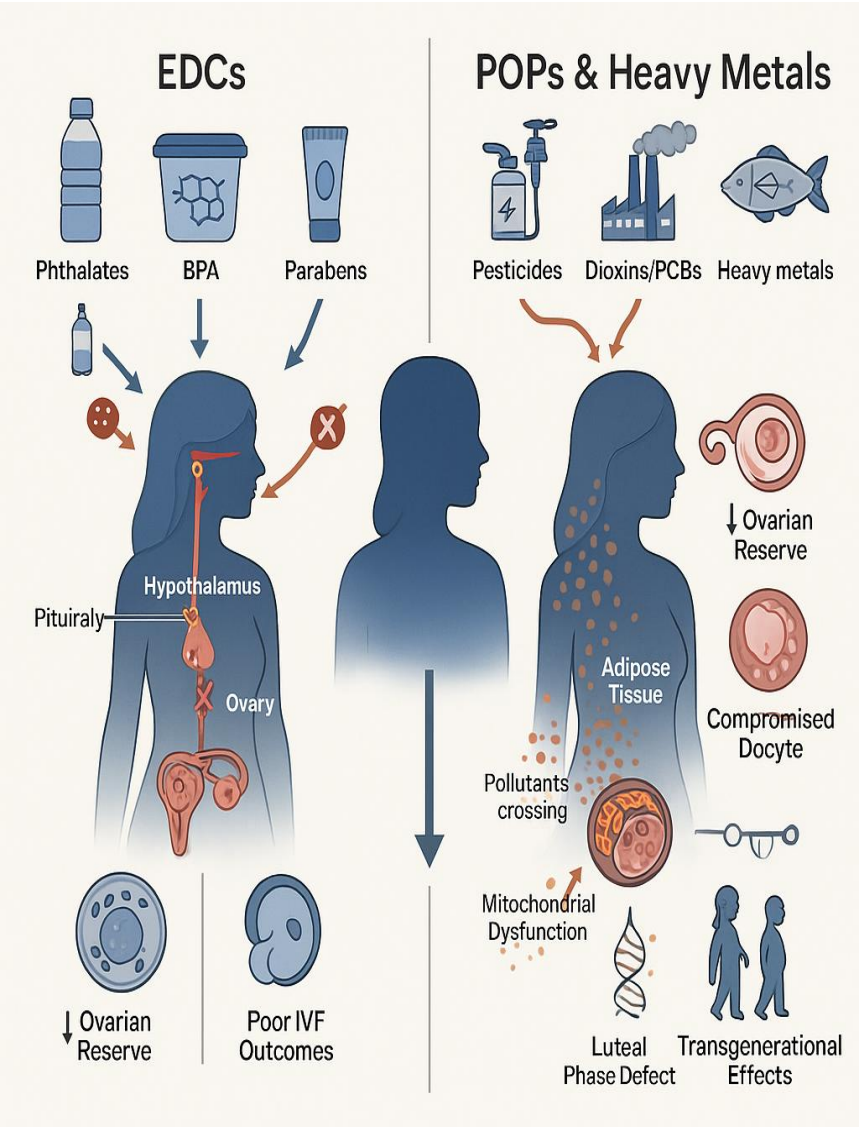

FIGURE 3: IMPACTS OF COMMON ENVIRONMENTAL TOXINS I IN FEMALE INFERTILITY

MECHANISTIC PATHWAYS LINKING LIFESTYLE TO REPRODUCTIVE FUNCTION

Lifestyle factors influence female reproductive capacity through complex, interconnected biological pathways that regulate ovarian physiology and endometrial receptivity. Nutritional patterns, physical activity, and environmental exposures modulate the hypothalamic-pituitary-ovarian (HPO) axis, altering the secretion of gonadotropin-releasing hormone (GnRH), luteinizing hormone (LH), and follicle-stimulating hormone (FSH), which directly impact follicular maturation and ovulation. Unhealthy diets, sedentary behavior, and exposure to endocrine-disrupting chemicals exacerbate oxidative stress, leading to mitochondrial dysfunction in oocytes and granulosa cells, thereby compromising energy production, meiotic spindle integrity, and embryo developmental competence [23].

lifestyle-driven metabolic imbalances can induce epigenetic modifications, such as altered DNA methylation and histone acetylation, within oocytes, which may disrupt gene expression patterns essential for early embryogenesis and could have transgenerational effects. Chronic low-grade inflammation, often arising from obesity, poor diet, or toxin exposure, further disrupts ovarian steroidogenesis, impairs endometrial receptivity, and dysregulates immune tolerance mechanisms critical for implantation and pregnancy maintenance. Together, these mechanistic pathways illustrate that lifestyle is not merely a peripheral modifier of reproductive potential, but a central determinant of the cellular, molecular, and epigenetic landscape underpinning female fertility [24, 25].

CLINICAL IMPLICATIONS AND RECOMMENDATIONS

Translating the growing body of evidence into clinical practice requires a proactive, multidisciplinary approach in fertility care. Lifestyle counseling should be integrated into routine consultations at fertility clinics, enabling clinicians to identify modifiable risk factors, such as suboptimal diet, excessive or insufficient physical activity, and environmental toxin exposure, early in the patient journey. Evidence-based dietary advice, emphasizing a Mediterranean-style pattern rich in whole grains, plant-based proteins, omega-3 fatty acids, and antioxidants, can enhance ovulatory function and improve outcomes in both natural and assisted reproduction settings [26].

Safe exercise guidelines should promote moderate-intensity aerobic and resistance training while cautioning against the reproductive risks of chronic high-intensity regimens, particularly in underweight women or those with irregular cycles. Equally important is patient education on minimizing toxin exposure, including practical measures such as reducing contact with plastic food

containers containing BPA or phthalates, choosing pesticide-free produce when possible, and avoiding high-mercury fish [27]. By embedding these recommendations into personalized treatment plans, clinicians can not only optimize fertility outcomes but also support long-term reproductive and metabolic health.

Beyond individual patient management, integrating lifestyle optimization into fertility care has broader implications for public health and preventive medicine. Community-based reproductive health programs can bridge the gap between evidence and practice by promoting preconception wellness through educational campaigns, workplace wellness initiatives, and digital health platforms that deliver personalized nutrition, exercise, and toxin-reduction guidance [28].

Collaboration between reproductive endocrinologists, dietitians, exercise physiologists, and environmental health specialists can ensure that interventions are both comprehensive and culturally tailored, thereby increasing adherence and sustainability. Furthermore, routine screening for lifestyle-related reproductive risk factors, such as elevated BMI, poor dietary quality, sedentary habits, and high levels of endocrine-disrupting chemicals, can be incorporated into annual health check-ups for women of reproductive age, enabling early intervention before infertility develops [29]. This integrated approach not only enhances fertility outcomes but also contributes to the prevention of chronic conditions such as type 2 diabetes, cardiovascular disease, and hormone-related cancers, underscoring the interconnectedness of reproductive and general health.

RESEARCH GAPS AND FUTURE DIRECTIONS

Despite growing evidence linking diet, exercise, and environmental toxin exposure to female fertility, significant research gaps persist. Most available studies are cross-sectional or short-term, limiting the ability to establish causal relationships between lifestyle factors and reproductive outcomes. Large-scale, longitudinal studies following diverse populations across reproductive life stages are urgently needed to validate current findings and uncover population-specific risk modifiers [30, 26].

Another major challenge is the lack of standardized methods for assessing exposure, particularly for endocrine-disrupting chemicals and dietary patterns, which hinders cross-study comparability and the formulation of precise clinical recommendations. Moreover, while lifestyle optimization is often encouraged alongside assisted reproductive technologies (ART), its integration into ART protocols remains inconsistent, with few randomized trials rigorously testing its impact on implantation and live birth rates [31, 32]. Addressing these gaps will require collaborative, multidisciplinary research

frameworks that combine epidemiology, molecular biology, and clinical trials to generate actionable, high-quality evidence capable of informing both clinical guidelines and public health policies.

Beyond simply identifying associations, there is a real need to understand how lifestyle factors shape fertility at the cellular and molecular level. Early evidence points to a role for oxidative stress, mitochondrial health, and even subtle epigenetic "reprogramming" in oocytes, which can influence everything from embryo quality to endometrial receptivity [33]. Encouragingly, certain lifestyle modifications, such as antioxidant-rich diets, regular but moderate exercise, and limiting exposure to environmental toxins, may help protect these reproductive pathways, but most of this work comes from small studies or animal models. To translate these findings into real-world fertility care, researchers need to combine advanced tools like metabolomics, proteomics, and epigenomic profiling with large, diverse patient cohorts [34]. It is also crucial to address global disparities, as women in low- and middle-income regions often face a double burden of environmental exposures and nutritional inadequacies that magnify reproductive risk. By linking cutting-edge science with population-focused studies, the field can move toward truly personalized lifestyle strategies for improving fertility outcomes.

CONCLUSION

Lifestyle factors, particularly diet, physical activity, and environmental exposures, play a pivotal yet often underestimated role in shaping female reproductive potential. The evidence consistently suggests that nutrient-dense dietary patterns, such as the Mediterranean diet, moderate physical activity, and minimizing contact with endocrine-disrupting chemicals can improve hormonal balance, enhance oocyte quality, and optimize endometrial receptivity. Conversely, poor dietary habits, sedentary lifestyles, and chronic exposure to environmental toxins are linked to ovulatory dysfunction, diminished ovarian reserve, and adverse fertility outcomes. Despite these insights, gaps remain in translating this knowledge into routine fertility care. Many studies are small, observational, or limited to specific populations, making it difficult to establish universal guidelines. Moving forward, a precision lifestyle medicine approach, one that tailors dietary, exercise, and environmental recommendations to a woman's genetic, metabolic, and socio-environmental profile, offers a promising pathway to improve natural conception rates and support assisted reproductive technologies. Ultimately, empowering women with evidence-based, individualized lifestyle interventions has the potential not only to enhance fertility but also to improve overall reproductive health and long-term

well-being.

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