iJRASET
International Journal For Research in Applied Science and Engineering Technology

INTERNATIONAL JOURNAL FOR RESEARCH

IN APPLIED SCIENCE & ENGINEERING TECHNOLOGY

Volume: 14 **Issue:** V **Month of publication:** May 2026

DOI: https://doi.org/10.22214/ijraset.2026.82937

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International Journal for Research in Applied Science & Engineering Technology (IJRASET)
ISSN: 2321-9653; IC Value: 45.98; SJ Impact Factor: 7.538
Volume 14 Issue V May 2026- Available at www.ijraset.com

Impact of Lifestyle Factors on Human Fertility: A Review of Diet, Stress, Sleep and Environmental Influences

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Abstract: ***Infertility has become an important global health concern, affecting nearly 12% of couples worldwide. Earlier, infertility was mostly considered a medical or genetic issue, but recent scientific studies show that everyday lifestyle habits also have a strong influence on reproductive health. This review paper examines findings from 20 previously published research studies to understand how factors such as diet, stress, sleep quality, body weight, physical activity, and environmental exposure affect fertility in both men and women.***

The reviewed evidence suggests that unhealthy lifestyle habits increase oxidative stress and hormonal imbalance, which can damage sperm cells and egg quality. Poor nutrition, lack of sleep, smoking, alcohol use, and continuous psychological stress negatively affect reproductive function, while healthier routines such as balanced diets, moderate exercise, proper sleep, and stress management may improve fertility outcomes. The paper concludes that infertility management should not rely only on medical treatment; instead, lifestyle improvement should also be considered an important part of reproductive care for couples.

Keywords: ***Infertility, Lifestyle Factors, Diet and Nutrition, Psychological Stress, Sleep Disturbances, Environmental Toxins, Oxidative Stress, Reactive Oxygen Species (ROS), Male Semen Quality, Female Ovarian Reserve, Modifiable Risk Factors, Assisted Reproductive Technology (ART).***

I. INTRODUCTION

When a couple cannot get pregnant after trying for one full year without using birth control, doctors call it infertility [9]. Today, this problem affects millions of people around the world, making it a major reproductive health crisis [19]. In the past, when a couple faced trouble conceiving, doctors usually focused almost entirely on the woman's body. They looked at ovarian function, fallopian tubes, and uterine health. However, modern clinical data reveals a major shift: in about half of all infertility cases, the underlying issue is related to the male partner's sperm quality and reproductive health [2]. Therefore, modern medicine now views infertility as a shared, dyadic issue that affects both partners together as a team.

Over the last few decades, standard semen quality in young men has dropped significantly, and women are experiencing lower egg reserves and poor egg quality at much younger ages [1, 7]. Because human genetics cannot change this quickly in just a few generations, the real culprit must be our rapidly changing environment, modern routines, and poor daily habits. Scientists call this entire combination of factors the "exposome"—which simply means the complete mix of mental stress, nutritional intake, sleep patterns, and environmental pollution we experience every day [14].

The good news is that unlike our chronological age or core genetics, our daily behavioral habits are "modifiable factors," meaning we have the absolute power to change and fix them [10]. Medical treatments like IVF (In Vitro Fertilization) or ICSI (Intracytoplasmic Sperm Injection) are highly advanced, but they are incredibly expensive, physically painful, emotionally draining, and do not always guarantee a baby [12]. Investigating how simple, non-invasive changes in lifestyle can improve natural fertility is a much cheaper, safer, and healthier way to help couples become parents. This comprehensive review paper summarizes findings from 20 high-quality reference papers to explain the exact biological pathways through which everyday habits change human fertility and what couples can do about it.

II. LITERATURE REVIEW

To establish a foundation for how lifestyle modifications alter human reproductive capacity, previous clinical benchmarks must be evaluated. Across historical data, clinical settings heavily isolated non-modifiable causes such as mechanical obstructions or severe genetic mutations. However, contemporary literature highlights a massive shift toward analyzing the systemic environment of both partners.

Studies focus on tracking key biomarkers of reproductive longevity, such as circulating Anti-Müllerian Hormone (AMH) levels to gauge ovarian reserve [1], and observing variations in traditional semen parameters including sperm morphology, volume, and forward motility against established World Health Organization (WHO) baselines [7]. A primary consensus across recent research highlights the phenomenon of lifestyle-induced cellular damage. The literature demonstrates that behavioral risk parameters act as catalysts for systemic issues, leading to poor egg development, broken sperm structures, and microbial imbalances within the reproductive tract [13, 18].

III. PROBLEM IDENTIFICATION

Despite advancements in surgical interventions and pharmaceutical protocols, global infertility rates continue to climb steadily. A primary limitation of traditional reproductive medicine is its tendency to treat infertility strictly as an unchangeable medical disorder, over-relying on invasive, expensive technologies while isolating the female partner. This approach frequently overlooks the collective, insidious impact of modifiable behavioral routines, including disrupted sleep patterns, chronic psychological distress, nutritional deficiencies, physical inactivity, and subconscious toxic exposures.

Furthermore, poor modern routines heavily accelerate underlying metabolic disorders like Polycystic Ovarian Disease (PCOD) and Polycystic Ovary Syndrome (PCOS). When nutritional, physical, or circadian disruptions interact with these pre-existing endocrine imbalances, chronic ovulatory failure rates intensify. A critical gap persists in simple, consolidated academic literature that maps out the precise cellular pathways through which these daily behaviors damage human gametes. Without identifying these modifiable behavioral variables, couples face diminished natural conception rates and avoidable failures within clinical ART frameworks.

IV. METHODOLOGY

To write this comprehensive review paper, a systematic search was conducted to collect relevant, high-quality scientific data. We gathered a total of 20 reference papers published in trusted, peer-reviewed medical and reproductive health journals.

A. Search Strategy and Selection

The papers were found using major online scientific databases like PubMed, EMBASE, and Google Scholar. The search words used to locate these papers included specific terms like *"infertility," "lifestyle factors," "diet and fertility," "male sperm quality," "stress and pregnancy," "sleep disturbances," "oxidative stress in reproduction,"* and *"endocrine disruptors."* To keep the collected data highly accurate and reliable, we used strict guidelines to pick the final papers:

- Inclusion Criteria: We selected papers that focused directly on human fertility, analyzed modifiable habits (like diet, weight, sleep, smoking, and anxiety), and clearly explained the biological reasons behind fertility issues.
- Exclusion Criteria: We completely left out papers that only talked about non-modifiable issues (like severe genetic mutations, permanent structural blockages, or advanced age) or did not provide clear evidence.

B. Data Analysis and Synthesis

After collecting the papers, they were studied carefully to extract their core data. A synthesis matrix was created to list the authors, the factors studied, and their main conclusions. This data is organized chronologically in Table 1 below.

Table 1: Matrix of the 20 Reviewed Reference Papers

Paper ID	Authors & Year	Core Focus	Key Contributions & Findings
[1]	Werner et al. (2024)	BMI, smoking, exercise, and AMH levels	Tracks how lifestyle choices impact female ovarian reserve by monitoring AMH levels, notably in metabolic conditions like PCOS.
[2]	Ilacqua et al. (2018)	Stress, nutrition, heat, mobile phone use	Proves male fertility and semen quality are heavily lowered by mental and environmental stress.
[3]	Rooney & Domar (2018)	Anxiety, depression, therapy interventions	Confirms a deep link between mental distress and female infertility; therapy helps boost pregnancy rates.
[4]	Dağ & Dilbaz	Female obesity and	Details how excess weight causes menstrual issues and

	(2015)	overweight	ovulation failure, severely impacting PCOD/PCOS outcomes.
[5]	Li et al. (2024)	Sleep quality, duration, sleep apnea	Connects bad sleep to lower egg quality, worse embryo grades, and lower fertilization rates.
[6]	Matuszczak et al. (2019)	Bisphenol A (BPA) environmental exposure	Shows how everyday plastic chemicals block or mimic natural human hormones, damaging fertility.
[7]	Balawender & Orkisz (2020)	Unhealthy habits in young men	Documents a steady drop in young men's sperm parameters compared to standard WHO guidelines.
[8]	Harlev et al. (2015)	Tobacco smoke and nicotine	Proves that smoking creates harmful oxidative stress that breaks down male sperm shape and viability.
[9]	Van Heertum & Rossi (2017)	Alcohol consumption	Links regular drinking to lower chances of getting pregnant and higher risks of early pregnancy loss.
[10]	Palomba et al. (2018)	Stress, lifestyle, and quality of life	Shows a "vicious circle" where female infertility causes major stress, which then hurts reproductive health.
[11]	Mussawar et al. (2023)	Physical activity intensity	Shows moderate exercise helps fertility and restores ovulation in PCOS, but excessive training stops ovulation.
[12]	Rossi et al. (2015)	Behavioral risks and chemical toxins	Connects standard behavioral habits with industrial pollution and high costs of IVF treatments.
[13]	Moustakli et al. (2025)	Oxidative Stress (OS) and lifestyle	Explains how bad habits create cellular stress in both eggs and sperm, and how to fix it.
[14]	Zlatnik (2016)	Phthalates, phenols, pesticides	Details how industrial chemical exposure causes long-term, transgenerational damage to human fertility.
[15]	Shao et al. (2021)	Circadian rhythms and the HPG axis	Explains how late-night light and shift work disrupt the brain signals needed to release eggs.
[16]	Torres-Arce et al. (2021)	Dietary antioxidants (Vitamins C, E, Zinc)	Evaluates how antioxidant supplements protect sperm from damage and improve movement.
[17]	Silvestris et al. (2019)	High-calorie diets and metabolism	Explains how bad nutrition leads to sugar processing issues, hurting female egg development.
[18]	Barraza-Ortega et al. (2025)	Gut and vaginal microbiomes	Connects modern unhealthy lifestyles to body bacteria imbalances, causing reproductive inflammation.
[19]	Collins & Rossi (2015)	Natural adjustments and vitamins	Reviews how natural changes like weight control, yoga, and vitamins improve conception success.
[20]	Aitken et al. (2014)	Sperm mitochondria and DNA damage	Shows that damaged sperm rely entirely on the female egg to repair their DNA after fertilization.

V. CORE FACTOR ANALYSIS & DISCUSSION

A. Diet, Body Weight, and the Exercise Balance

Nutritional habits and body mass index (BMI) directly regulate endocrine balance and reproductive function. In females, excess adipose tissue drives hyperinsulinemia and systemic insulin resistance [17]. This metabolic dysfunction is highly deleterious for patients managing underlying PCOD/PCOS [4]. Elevated insulin concentrations stimulate ovarian theca cells to overproduce androgens, halting follicular development and causing chronic anovulation [4]. Consequently, natural conception rates fall, while clinical ART interventions face poor oocyte retrieval, elevated miscarriage risks, and lower live birth outcomes [4, 17]. In males, hyperadiposity accelerates the conversion of testosterone into estrogen within peripheral fat tissues [7]. This hormonal shift suppresses neuroendocrine signals from the brain, induces testicular hyperthermia, and significantly degrades sperm count, concentration, and progressive motility [2, 7].

Conversely, targeted dietary adjustments can mitigate this damage. Regimens rich in exogenous antioxidants—including Vitamins C and E, Zinc, Selenium, and Coenzyme Q10—shield developing gametes from oxidative degradation [16]. These micronutrients neutralize free radicals, protecting sperm structural integrity and optimizing forward motility [16]. Physical activity acts as a critical balancer of this metabolic equation. For PCOD/PCOS management, consistent, moderate-intensity exercise (e.g., brisk walking or swimming) improves insulin sensitivity, reduces systemic inflammation, and restores natural ovulatory cycles [11, 19]. However, extreme, uncalibrated high-intensity over-exertion induces an energetic crisis. The hypothalamus interprets excessive physical strain as a survival threat, down-regulating the neuroendocrine signals required for follicular maturation and resulting in anovulation [11].

B. Mental Stress and the Mind-Body Connection

The relationship between emotional trauma and reproductive pathology operates as a complex, bidirectional feedback loop [10]. Infertility challenges trigger acute elevations in patient anxiety, clinical depression, and psychological guilt [3]. This emotional burden activates the Hypothalamic-Pituitary-Adrenal (HPA) axis, driving sustained systemic elevations of cortisol and adrenaline [10]. In females, hypercortisolemia directly suppresses hypothalamic functioning, disrupting the pulsatile secretion of gonadotropins required for regular ovulation and altering endometrial receptivity, which compromises embryo implantation [3, 19]. In males, elevated psychological stress down-regulates Leydig cell functionality, directly reducing testosterone production and precipitating oligospermia, asthenozoospermia, abnormal morphology, and psychogenic erectile dysfunction [2].

Conversely, structured psychological interventions demonstrate highly positive clinical outcomes [3]. Incorporating cognitive-behavioral therapy (CBT), peer support networks, or mindfulness yoga significantly suppresses systemic cortisol levels [3]. Alleviating this psychological burden restores neuroendocrine equilibrium, stabilizes natural menstrual cycles, and correlates with elevated conception success rates across both natural and assisted reproductive models [3, 19].

C. Sleep Disturbances and Circadian Rhythm Disruption

Human reproductive endocrinology relies on a preserved 24-hour master clock known as the circadian rhythm [15]. This infrastructure regulates the Hypothalamic-Pituitary-Gonadal (HPG) axis, controlling the precise timing of essential reproductive signals, including the nocturnal Luteinizing Hormone (LH) surge required to trigger ovulation [15]. Chronic sleep restriction (under 7 hours nightly) or sleep fragmentation—driven by shift work or late-night blue-light device exposure—destabilizes this central neuroendocrine oscillator [15].

For patients managing PCOD/PCOS, circadian misalignment is exceptionally damaging; it accelerates ovarian insulin resistance and disrupts downstream gonadotropin signaling [15]. Sleep-deprived females exhibit a compromised ovarian reserve, characterized by reduced anti-Müllerian hormone (AMH) output [1, 5]. At the cellular level, inadequate rest accelerates the intracellular accumulation of Reactive Oxygen Species (ROS) [5]. This oxidative surge triggers microenvironmental inflammation within the reproductive tract, directly causing oocyte DNA fragmentation, poor embryo grading, and elevated failure rates within clinical fertility programs [5, 15].

D. Environmental Toxins, Smoking, and Alcohol Consumption

Modern industrial environments continuously introduce a dense mix of chemical compounds into the human exposome [14]. Bisphenol A (BPA), found in plastic consumer products and food containers, serves as a primary reproductive hazard [6]. Functioning as a potent endocrine-disrupting chemical (EDC), BPA mimics or competitively blocks endogenous estrogen and testosterone receptors [14]. It bioconcentrates within adipose tissue and follicular fluid, driving oocyte attrition and disrupting early embryonic cleavage [6]. Similarly, widespread exposures to synthetic plasticizers like phthalates and household pesticides induce transgenerational epigenetic modifications that alter the fertility profiles of future generations [14].

Recreational behaviors multiply this total chemical burden:

1) Tobacco Smoking: Inhaling tobacco smoke introduces heavy metals (cadmium, lead) and nicotine directly into systemic circulation, inducing profound oxidative stress [8]. These toxins compromise the spermatozoa's mitochondria, disabling the primary metabolic engine that powers progressive motility [20]. This oxidative insult causes structural lesions in the paternal genome, leading to high sperm DNA fragmentation [8]. Because mature spermatozoa lack the intracellular machinery required for DNA self-repair, they rely entirely on the maternal oocyte to stitch together broken paternal strands post-fertilization [20]. If this paternal genetic damage exceeds the oocyte’s repair capacity, it triggers early blastocyst arrest or spontaneous miscarriage [8, 20].

2) Alcohol Consumption: Regular or heavy ethanol intake acts as a direct cellular toxin to sensitive reproductive tissues [9]. It disrupts female sex-steroid homeostasis, downgrades oocyte viability, and induces microbial dysbiosis within the gut and vaginal microbiomes [18]. This resulting microbial shift triggers systemic inflammatory cascades that prevent proper embryo-endometrial cross-talk, significantly reducing live birth outcomes [9, 12, 18].

VI. RECOMMENDATIONS & THERAPEUTIC INTERVENTIONS

Based on the cellular analysis, couples facing fertility difficulties can utilize structured, non-invasive behavioral adjustments alongside regular medical advice to maximize conception success rates:

1) Nutritional Restoration: Patients—particularly those navigating PCOD/PCOS—should transition to low-glycemic, antioxidant-rich diets to mitigate insulin resistance, reduce free radicals, and improve sperm motility metrics.

2) Circadian Stabilization: Restricting late-night blue-light device exposure and preserving an uninterrupted 7-to-8-hour nightly rest cycle helps repair the HPG axis and protect the ovarian reserve.

3) Mind-Body Interventions: Integrating structured counseling, meditation, or stress-relief yoga helps suppress high cortisol levels, effectively unlocking natural ovulation paths.

4) Toxic Exposure Mitigation: Eliminating tobacco use, minimizing alcohol intake, and deliberately lowering food contact with plastic materials minimizes the presence of endocrine-disrupting chemicals.

VII. CONCLUSION

In conclusion, human reproductive capacity is profoundly governed by modifiable lifestyle behaviors rather than unchangeable genetic wiring. Infertility operates as a shared, dyadic condition where the nutritional status, stress profiles, sleep hygiene, and toxic exposures of both partners interactively dictate outcomes. Pre-existing endocrine pathologies, including PCOD/PCOS, act as metabolic accelerators that aggressively worsen under adverse habits.

Cellular analysis demonstrates that systemic inflammation, hyperinsulinemia, and accelerated oxidative stress (ROS) are the primary pathways through which poor dietary choices, high BMI, chronic anxiety, sleep fragmentation, smoking, and environmental toxins degrade gamete quality. Because these behavioral factors are entirely modifiable, prioritizing non-invasive adjustments presents a highly accessible, cost-effective therapeutic strategy. Systematically optimizing daily habits alongside standard medical protocols protects natural gamete reserves, mitigates underlying endocrine imbalances, and significantly maximizes the clinical efficacy of advanced reproductive treatments.

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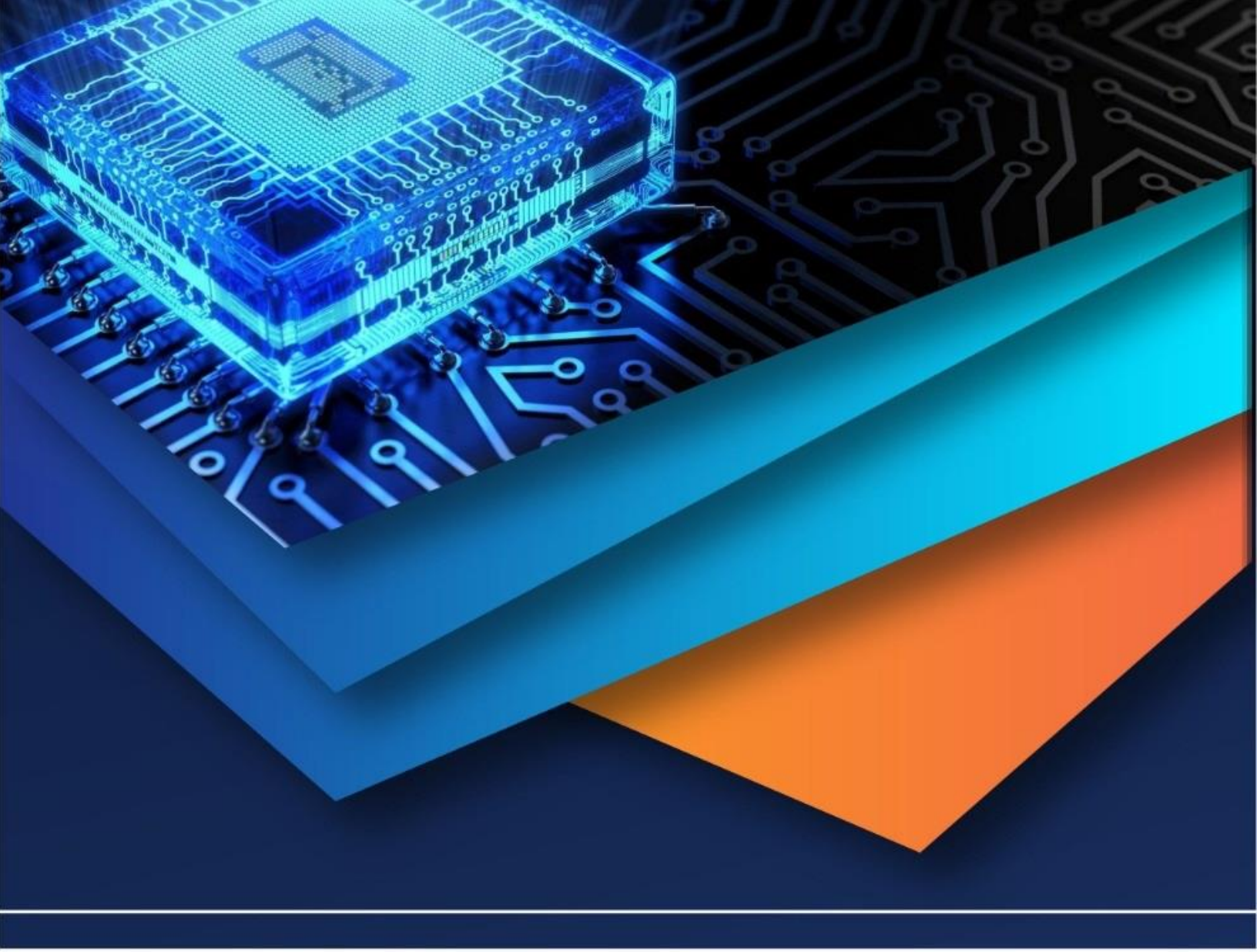

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