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Lifestyle Factors and Fertility in Couples Trying to Conceive: A Review of Evidence-Based Interventions

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ABSTRACT

Introduction

Infertility is a growing global public health concern, affecting an estimated 8–12% of couples of reproductive age. The causes are multifactorial, including non-modifiable biological and genetic factors as well as modifiable lifestyle and environmental influences. In recent years, increasing attention has been paid to how daily habits, diet, physical activity, substance use, mental health, sleep quality, and environmental exposures impact fertility outcomes. Additionally, the biological aging of the reproductive system - particularly in the context of delayed parenthood - has become increasingly relevant. A comprehensive understanding of these factors and their mechanisms is essential for optimizing fertility care and improving reproductive outcomes.

Aim of the study

This study aims to systematically review and summarize current scientific evidence on the influence of modifiable lifestyle, behavioral, and environmental factors on fertility in both women and men. It also explores age-related reproductive decline, highlights immune system interactions, and evaluates the efficacy of evidence-based interventions designed to enhance reproductive health. The ultimate goal is to provide practical guidance for couples trying to conceive and to support holistic clinical approaches in fertility counseling and treatment.

Materials and methods

A systematic literature review was conducted using databases including **PubMed, Web of Science, Scopus,** and **Google Scholar.** The search covered peer-reviewed articles published between **2015 and 2024**. Priority was given to **systematic reviews, meta-analyses, randomized controlled trials,** and **observational studies** relevant to both male and female fertility.

Conclusion

Fertility is a complex interplay of biological, environmental, and behavioral factors. This review demonstrates that lifestyle modifications - including improved nutrition, regular physical activity, stress management, substance cessation, and adequate sleep - can significantly enhance reproductive potential. Age remains a critical determinant, particularly for female fertility, due to the natural decline in oocyte quality and ovarian reserve. Similarly, advanced paternal age is linked to genetic and epigenetic changes in sperm, with implications for offspring health. Environmental pollutants and immunological dysregulation also contribute to reproductive impairment in both sexes. The integration of evidence-based lifestyle and psychological interventions into fertility care may improve outcomes and support healthier pregnancies. Further research should focus on personalized, multi-dimensional approaches to reproductive health that include prevention, education, and preconception care.

Keywords

Fertility, lifestyle factors, diet, physical activity, stress, mental health, smoking, alcohol, sleep, circadian rhythm, hormonal balance, immunological factors, environmental toxins, biological age, reproductive aging, medications, supplements.

Epidemiology

Infertility is recognized as a reproductive system disease affecting both women and men. It is defined as the inability to achieve pregnancy after 12 months or more of regular, unprotected sexual intercourse. This condition affects a significant proportion of the reproductive-age population worldwide, with estimates suggesting that between 8% and 12% of couples experience difficulties conceiving(1). The causes of infertility can be categorized into modifiable factors, which are influenced by lifestyle changes, and non-modifiable factors, stemming from biological or genetic origins(2).

Contemporary demographic and social trends, such as the increasing tendency to delay starting a family, substantially raise the risk of fertility problems. In light of these challenges, lifestyle modification has become an essential strategy for improving reproductive health and enhancing the chances of natural conception(2).

The aim of this review is to assess and summarize the current knowledge on the impact of modifiable lifestyle factors on fertility in both women and men. Special emphasis is placed on evidence-based interventions that can be recommended to couples trying to conceive. Furthermore, the review highlights the importance of health education, preconception counseling, and preventive measures in improving reproductive health at both individual and population levels.

Nutrition

An increasing number of studies indicate that diet plays a significant role in couples' fertility. It is recommended to increase the intake of monounsaturated fatty acids, as well as fruits and vegetables, which are good sources of protein and non-heme iron. Additionally, the consumption of full-fat dairy products has been found beneficial for female fertility. At the same time, it is advised to limit the intake of trans fatty acids and carbohydrate-rich foods with a high glycemic index(3).

An important component of the diet is also multivitamin supplementation, especially preparations containing folic acid, vitamin B12(4), and long-chain omega-3 fatty acids, which have a beneficial effect on female fertility(5). Supplementation helps prevent deficiencies of these nutrients. In men, free radicals play an important role in the development of infertility. Adequate intake or supplementation with antioxidants can help prevent this process and support treatment. Nutrients such as zinc, selenium, and folic acid positively affect semen quality(4).

One of the significant and modifiable factors influencing fertility is excessive calorie intake, which leads to overweight and obesity. Obesity constitutes a serious health issue, exerting a negative impact on reproductive function in both women and men. Excess body weight disrupts the hormonal balance of the body. Among other things, it leads to a decrease in the level of sex hormone-binding globulin and testosterone, which interferes with the proper functioning of the hormonal system that regulates reproductive functions. Moreover, obesity is often accompanied by insulin resistance and a chronic low-grade inflammatory state, both of which further impair reproductive system function.

These disturbances contribute to reduced ovarian function, lower endometrial receptivity, and decreased semen quality. As a result, the time required to achieve pregnancy is prolonged, success rates of assisted reproductive technologies decline, and the risk of miscarriage increases(6).

A well-balanced diet, therefore, appears to play a key role in preventing infertility in both women and men.

Physical activity

In the field of reproductive health, the latest recommendations from the World Health Organization indicate that women planning pregnancy should engage in at least 150 minutes per week of vigorous aerobic exercise. This not only enhances the chances of conception but also contributes to overall health improvement(7). Regular, long-term physical activity in women experiencing infertility has been shown to lower insulin and free androgen levels, which in turn supports the restoration of reproductive function through mechanisms involving the hypothalamic - pituitary - gonadal axis(8).

Studies show that engaging in physical activity may help reduce lead accumulation in the body and lower gonadotropin levels in women with infertility(9,10). Beyond these mechanisms directly related to fertility, physical activity is also known to activate antioxidant pathways and enhance immune function. It has been shown that regular exercise significantly reduces inflammatory biomarkers such as interleukin-6 and tumor necrosis factor-alpha, decreases oxidative stress - measured by reactive oxygen species and malondialdehyde levels - and enhances enzymatic antioxidant defenses, including superoxide dismutase, catalase, and total antioxidant capacity(11).

These changes have been associated with significant improvements in semen parameters, sperm DNA integrity, and overall reproductive function in men affected by infertility, suggesting that physical activity may serve as an effective adjunctive strategy in fertility management for this population(12).

Research findings further indicate that moderate to vigorous physical activity significantly reduces the risk of infertility(11).

Psychological Stress and Mental Health

In addition to its detrimental effects on psychological well-being, stress also impacts physiological processes, such as the menstrual cycle. Psychological stress activates the hypothalamic – pituitary - adrenal (HPA) axis, leading to increased secretion of cortisol and corticotropin-releasing hormone (CRH), which suppress the release of gonadotropin-releasing hormone (GnRH). This results in decreased levels of luteinizing hormone (LH) and follicle-stimulating hormone (FSH), ultimately impairing follicular development, reducing oocyte quality, and disrupting ovulation.

Furthermore, stress can alter the sensitivity of the hypothalamic–pituitary–gonadal (HPG) axis to hormonal signals, collectively contributing to reproductive dysfunction and decreased fertility. Chronic stress, in particular, elevates glucocorticoid levels, which adversely affect oocyte maturation and fertilization capacity. These effects are mediated by hormonal imbalances, alterations in the follicular microenvironment, and increased apoptosis of ovarian cells.

Additionally, stress-related hormonal disturbances are associated with limited endometrial proliferation, disrupted expression of growth factors, and reduced uterine receptivity, all of which significantly diminish fertility(13).

Chronic stress is a psychological condition that can contribute to various sexual difficulties, including reduced libido as well as erectile and ejaculatory dysfunction(14).

Stress can adversely affect testicular function, resulting in decreased circulating testosterone levels, impaired spermatogenesis, and reduced semen quality. Numerous experimental studies have demonstrated a positive correlation between chronic stress and erectile dysfunction. Prolonged stress disrupts the normal structure of the penile corpora cavernosa, which in turn impairs the ability to achieve and maintain an erection(15).

Therefore, effective stress management may play a crucial role in preserving reproductive health and fertility.

Tobacco Smoking

Available biological, experimental, and epidemiological data suggest that up to 13% of infertility cases may be attributed to cigarette smoking. Smoking appears to accelerate the decline of reproductive function and may lead to an earlier onset of menopause by one to four years. One of the potential mechanisms through which smoking negatively affects fertility is gamete mutagenesis, resulting in damage to the genetic material of reproductive cells. In men, smoking is associated with poorer semen parameters and reduced performance in functional sperm tests, which may further decrease the likelihood of conception(16).

Substances found in tobacco have a detrimental effect on ovarian follicle maturation. Smoking also significantly disrupts endometrial receptivity, making proper embryo implantation more difficult. In men, this habit leads to reduced sperm production, increased oxidative stress, and DNA damage in sperm cells, resulting in diminished fertilization capacity. Embryos originating from smokers tend to have lower implantation potential. Moreover, fetal exposure to tobacco components in utero may lead to a reduced sperm count in adulthood(17).

The significant reproductive risks associated with smoking, along with evidence that much of the smoking-related decline in fertility can be reversed within a year of quitting, serve as a strong incentive to stop smoking(16).

Alcohol

Alcohol consumption has a detrimental effect on male reproductive function. Ethanol and its metabolites act as genotoxic agents, altering the expression of genes involved in the hormonal regulation of spermatogenesis and increasing sperm DNA fragmentation, which may contribute to transgenerational effects impacting offspring health.

Alcohol disrupts the feedback mechanisms of the hypothalamic–pituitary–gonadal (HPG) axis, impairing the production and secretion of luteinizing hormone (LH) and follicle-stimulating hormone (FSH), ultimately leading to a reduction in Sertoli cell numbers. Decreased levels of testosterone, LH, and FSH result in abnormal sperm development and maturation, reduced sperm production, gonadal atrophy, erectile dysfunction, infertility, and diminished secondary male sexual characteristics(18).

Chronic alcohol use and alcohol dependence are associated with impaired reproductive function in both men and women. The proper function of the HPG axis and its hormones is essential for normal reproductive physiology. In individuals with excessive alcohol intake, HPG axis dysfunction has been linked to reduced libido, infertility, and gonadal atrophy. Numerous studies have documented alcohol's negative effects on all three components of the HPG axis: the hypothalamus, the pituitary gland, and the gonads.
In women, alcohol consumption during the premenopausal period is associated with menstrual irregularities, diminished ovarian reserve, anovulatory cycles, an increased risk of spontaneous miscarriage, and a higher likelihood of early menopause or hyperprolactinemia(19).

Sleep Quality and Circadian Rhythm

Sleep disturbances and circadian rhythm disruptions are increasingly recognized as factors that negatively impact fertility in both men and women. In men, population studies and meta-analyses have shown that abnormal sleep duration - whether too short or too long - is associated with decreased total sperm count, concentration, progressive motility, and normal morphology. The underlying biological mechanisms include reduced testosterone levels, circadian rhythm disruption, increased oxidative stress, and elevated inflammatory markers. Poor sleep habits also impair the expression of clock genes such as **BMAL1, CLOCK, PER,** and **CRY**, which play a critical role in regulating spermatogenesis, Leydig cell function, and sperm acrosomal enzyme activity. Studies in both animals and humans confirm that chronic sleep deprivation impairs sexual function, reduces testosterone levels, and worsens semen quality(20).

Similar mechanisms apply to female fertility. The suprachiasmatic nucleus (SCN), as the central pacemaker of the circadian rhythm, regulates the hypothalamic–pituitary–gonadal (HPG) axis and the secretion of reproductive hormones, influencing ovulation, implantation, and embryo development. Circadian genes are rhythmically expressed in the ovaries, uterus, and fallopian tubes, and their dysfunction may contribute to premature ovarian insufficiency and infertility. Melatonin, secreted at night, not only helps maintain circadian stability but also acts as a potent antioxidant. Its high concentration in follicular fluid supports oocyte maturation and embryo quality. Melatonin supplementation has shown beneficial effects in women with polycystic ovary syndrome (PCOS).Moreover, sleep disorders can activate the hypothalamic–pituitary–adrenal (HPA) axis, increasing cortisol levels and disrupting hormonal balance, thus impairing reproductive function. In women with PCOS, obstructive sleep apnea (OSA) may exacerbate insulin resistance and hormonal disturbances. The accompanying oxidative stress and chronic inflammation further deteriorate oocyte quality and reduce the chances of conception(21).

Environmental Factors

Endocrine-disrupting chemicals (EDCs), such as bisphenol A (a synthetic estrogen), phthalates (found in cosmetics, plastics, and packaging), organophosphate pesticides, heavy metals (e.g., lead), perfluorinated compounds (used in non-stick cookware and food packaging), and flame retardants, can significantly interfere with the function of sex hormones by binding to their receptors or altering gene expression.

Exposure to these substances, especially during prenatal and developmental periods, leads to long-term reproductive disorders - such as premature ovarian insufficiency, polycystic ovary syndrome, and ovulatory dysfunction in women, and reduced semen quality, demasculinization of the reproductive system, erectile dysfunction, and decreased libido in men. Evidence from both epidemiological and experimental studies also suggests potential transgenerational effects, meaning these chemicals can impact the health and fertility of future generations. Additionally, exposure to these environmental toxins has been linked to lower pregnancy rates, increased miscarriage risk, neurodevelopmental disorders in offspring, and disruptions in thyroid hormone function, which is essential for proper fetal brain development(22).

Exposure to heavy metals such as arsenic (As), cadmium (Cd), and lead (Pb) can significantly disrupt reproductive function and lead to infertility. Arsenic induces oxidative stress, damages oocytes, reduces levels of sex hormones (estradiol, LH, FSH), and causes follicular atresia and thinning of the uterine myometrium, which impairs embryo implantation. Lead negatively affects oocyte maturation, decreases levels of serotonin and norepinephrine, and is associated with a reduced number of oocytes, lower implantation rates, and decreased fertilization success. Cadmium disrupts sperm morphology and can damage the structure of female gametes, affecting the quality of reproductive cells and the fertilization process(23).

Heavy metals such as lead (Pb), cadmium (Cd), arsenic (As), mercury (Hg), chromium (Cr), copper (Cu), and nickel (Ni) have a highly toxic impact on male fertility. Their effects include hormonal disruptions, damage to the testes, Sertoli and Leydig cells, and impaired spermatogenesis. These metals can interfere with the hypothalamic–pituitary–testicular (HPT) axis, reduce testosterone production, and deteriorate semen quality, including sperm morphology, motility, and count. Additionally, they induce oxidative stress, leading to sperm DNA damage, apoptosis, and epigenetic changes that may affect future generations. Exposure can occur through environmental sources (polluted air, food, water), occupational hazards (e.g., welders, smelters), or lifestyle factors such as smoking(24).

Air pollution is increasingly recognized as a significant factor negatively affecting human reproductive function. Among the main pollutants linked to fertility problems are particulate matter (PM2.5 and PM10), which have been shown to reduce fertility rates and embryo implantation success while increasing the risk of miscarriage. Nitrogen dioxide (NO_2) is correlated with higher rates of pregnancy loss and poorer outcomes in in vitro fertilization (IVF) procedures. Sulfur dioxide (SO_2), on the other hand, may cause chromosomal damage, further increasing the likelihood of pregnancy loss. Carbon monoxide (CO) impairs the oxygen-carrying capacity of hemoglobin, leading to fetal hypoxia and a higher risk of miscarriage(25).

Air pollutants can disrupt the endocrine system by activating aryl hydrocarbon (AhR), estrogen, or androgen receptors. They may also act as oxidants or generate free radicals, triggering oxidative stress and inflammation. Some pollutants can alter DNA or induce epigenetic modifications that may impact future generations(26).

Exposure to electromagnetic fields (EMF) can disrupt the functioning of the reproductive system at multiple stages. In the pineal gland, a reduction in melatonin production is observed - a hormone that regulates the circadian rhythm and protects cells against oxidative stress. In men, EMF negatively affects sperm by impairing morphology, reducing fertility, increasing apoptosis, and lowering intracellular calcium levels, which compromises sperm viability and fertilization capacity. In women, EMF disrupts the estrous cycle and inhibits the growth of ovarian follicles, limiting oocyte maturation and reducing the likelihood of ovulation. At the embryonic level, EMF increases the risk of congenital abnormalities, interferes with cell division, and impairs blastocyst formation - a crucial step for successful implantation in the uterus. In offspring, the effects of EMF exposure may include testicular development disorders as well as alterations in body weight and growth. Overall, the evidence suggests that electromagnetic fields can have harmful effects not only on fertility but also on the health of future generations(27).

Oxidative stress (OS), a condition defined by an imbalance between pro-oxidants (reactive oxygen and nitrogen species) and antioxidant defense mechanisms, plays a key role in the pathogenesis of subfertility in both women and men. An excess of reactive oxygen species (ROS) can damage reproductive cells and impair their function, significantly affecting fertility. The main sources of oxidative stress are lifestyle-related factors, such as obesity, malnutrition, drug use, smoking, alcohol consumption, and exposure to environmental pollutants. This unfavorable state promotes the development of reproductive disorders, including polycystic ovary syndrome (PCOS), endometriosis, premature ovarian insufficiency, and unexplained infertility, all of which greatly reduce the chances of conception. In women, ROS are involved in regulating the ovulatory cycle. While they are necessary for ovulation, excessive ROS levels can lead to apoptosis of ovarian cells and follicular damage. In the corpus luteum, ROS also play a regulatory role - at optimal levels, they support progesterone production, but their increase during the regression phase accelerates luteal breakdown(28).

Oxidative stress plays a significant role in the reduction of male fertility. It can be triggered by various factors such as infections, environmental pollution, unhealthy lifestyle, or occupational exposure. Additionally, the presence of abnormal sperm with excess residual cytoplasm and leukocytes in the semen promotes the overproduction of reactive oxygen species (ROS) and weakens the antioxidant defense system. An imbalance between ROS and antioxidants leads to oxidative stress, which damages reproductive cells - causing DNA, protein, and lipid damage in sperm, as well as inducing apoptosis of germ cells. As a result, there is a decrease in sperm count and impairment of their functions, including motility, viability, capacitation, and the ability to undergo the acrosome reaction(29).

Medications and Supplements

The use of anabolic-androgenic steroids (AAS) negatively affects male fertility. AAS users exhibit reduced levels of FSH and LH, which disrupt spermatogenesis, with these effects persisting up to six months after discontinuation. Although hormone levels generally return to normal after one year, total testosterone often remains lower. AAS use decreases sperm motility and testicular volume. After stopping AAS, issues with libido and erectile function are common. In many cases, additional medical treatment is necessary to restore fertility(30).

Preconception use of antibiotics, particularly sulfonamides and macrolides, has been associated with reduced reproductive capacity compared to those who did not use them(31). The chart illustrates the impact of various antibiotics detected in women's urine on the risk of infertility. Certain substances, particularly sulfonamides (e.g., sulfamethoxazole, sulfaclozine, sulfamonometoxine) and chlortetracycline, were significantly associated with an increased risk of infertility. In contrast, antibiotics such as amoxicillin, cefaclor, azithromycin, and pefloxacin showed a protective effect, reducing the risk. The influence of specific antibiotics varied depending on the participant's age and the exclusion of statistical outliers. Some compounds were detected in a majority of subjects, indicating widespread environmental exposure. These findings confirm that the effect of antibiotics on female fertility depends on the active compound, dosage, and exposure source(32).

Both human and animal models suggest that the use of antidepressants may negatively impact fertility by affecting the levels of the neurosteroid allopregnanolone. Studies in rodents indicate that antidepressant-induced increases in allopregnanolone can disrupt the hypothalamic–pituitary–ovarian (HPO) axis. Incubation of hypothalamic tissue with allopregnanolone inhibits GnRH secretion, which may interfere with ovulation. A prospective study involving 957 women showed that the use of antidepressants – mainly SSRIs – during attempts to conceive may be associated with reduced natural fertility, particularly in women with a history of depression or anxiety. These findings suggest caution when using antidepressants during the preconception period, although they do not serve as a definitive contraindication to continued use(33).

The impact of specific supplements and nutrients on male fertility appears to be significant, especially in the context of supporting the treatment of idiopathic infertility. Selenium supplementation at doses of 100–300 μg per day has shown beneficial effects on semen quality by improving sperm motility, concentration, and morphology. When combined with N-acetylcysteine, selenium also positively affects hormonal balance by increasing testosterone, LH, and inhibin B levels, while reducing FSH levels. Zinc plays an important role as well—its supplementation improves sperm concentration and enhances total motility. Omega-3 fatty acids (DHA and EPA), particularly in higher doses (up to 2 g/day), have a positive effect on sperm count, concentration, motility, and morphology. Coenzyme Q10, taken at 200–300 mg per day for a period of 3 to 6 months, significantly improves semen parameters and also regulates reproductive hormone levels, with reported increases in inhibin B and reductions in LH and FSH. Lastly, carnitines - both L-carnitine and L-acetylcarnitine - have a favorable impact on sperm motility and morphology. Supplementation with 2–3 g of LC and 1 g of LAC daily provides particular benefits for men with oligozoospermia and asthenozoospermia (34).

Immunological Factors

Immunological disorders and chronic inflammation have a significant impact on male reproductive health. It is estimated that 5–10% of male infertility cases are associated with inflammation or autoimmune processes, including the presence of anti-sperm antibodies, orchitis, and prostatitis. When the immune system becomes dysregulated, it may damage reproductive cells through the overproduction of pro-inflammatory cytokines, activation of NK and Th cells, or impaired function of regulatory T cells (Tregs).

Such autoimmune responses reduce sperm motility, viability, and morphology. Chronic inflammation, whether localized (e.g., in the reproductive tract) or systemic (e.g., due to obesity or inflammatory bowel disease), disrupts spermatogenesis. It leads to oxidative stress, DNA damage in sperm, hormonal imbalances (e.g., reduced testosterone levels), and apoptosis of germ cells. Pro-inflammatory cytokines such as TNF-α, IL-6, and IL-1β impair testicular function and the hypothalamic - pituitary - gonadal (HPG) axis, affecting the synthesis and secretion of sex hormones and sperm production. Bacterial infections (e.g., *E. coli*, *Chlamydia trachomatis*) further impair semen quality through direct sperm damage and induction of local inflammatory responses. Additionally, obesity increases leptin and aromatase levels, which lowers testosterone concentrations and disrupts the hormonal balance necessary for proper spermatogenesis(35).

Immunological causes of female infertility primarily involve the presence of autoantibodies, dysregulation of mucosal immunity, and overactivation of immune cells. Antibodies against the zona pellucida (ZP) can prevent sperm penetration and embryo implantation. Antiphospholipid antibodies (APA) are associated with recurrent miscarriage and placental complications. Conditions such as PCOS and endometriosis show increased immune activity and the presence of autoantibodies, disrupting fertility. Cervical mucus and vaginal secretions contain immunoglobulins and cytokines that fluctuate throughout the menstrual cycle, influencing the reproductive environment. The presence of anti-sperm antibodies (ASA) can impair fertilization. Disruptions in both local and systemic immune responses may affect every stage of reproduction - from fertilization to embryo implantation and pregnancy maintenance(36).

Biological Age and Reproductive Aging

In Western countries, an increasing number of women are choosing to pursue motherhood at a later age, which raises the risk of age-related infertility(37). Female reproductive aging is primarily associated with a decline in oocyte (egg cell) quality, which significantly impacts fertility, especially after the age of 35. Studies have shown that older women achieve higher pregnancy rates when using oocytes from younger donors. The main causes of declining oocyte quality include aneuploidies (chromosomal abnormalities), which increase with age, and the loss of structural proteins such as cohesin and shugoshin that are essential for chromosomal stability. With age, the number of available oocytes also decreases, ovarian reserve markers (AMH, FSH) decline, and the ovaries undergo structural changes, including fibrosis. The reduction in estrogen and progesterone levels leads to hormonal imbalances and a general deterioration in health. Advanced maternal age is also linked to a higher risk of genetic disorders in offspring and reduced effectiveness of assisted reproductive technologies (ART). Additionally, aging oocytes exhibit epigenetic changes that may impair their function, although the exact mechanisms behind these changes remain unclear(38).

Although men retain the ability to produce sperm throughout their lives, paternal age affects both semen quality and offspring health. As men age, genetic and epigenetic changes accumulate in sperm, driven by factors such as aging, environmental exposures, lifestyle, and assisted reproductive technologies (ART). These changes may increase the risk of neurodevelopmental and psychiatric disorders in children. Research shows that aging is associated with alterations in DNA methylation, histone modifications, and small RNA profiles in sperm. These changes are not random; rather, they target specific genes involved in embryonic development and metabolic functions. Lifestyle factors and exposure to environmental chemicals can further influence these epigenetic processes. Further research is needed to understand the mechanisms of male epigenomic aging, as well as to raise public awareness about the impact of paternal age on fertility and offspring health. In the future, targeted therapies may be developed to improve sperm health in older men(39).

Summary

Infertility affects 8–12% of couples globally and is influenced by both non-modifiable (e.g., age, genetics) and modifiable factors such as lifestyle and environment. This review highlights how nutrition, physical activity, stress, sleep, substance use, environmental toxins, and medications impact fertility in both women and men. A healthy diet rich in antioxidants, omega-3, vitamins, and minerals improves reproductive function, while obesity and poor nutrition impair hormonal balance and gamete quality. Regular physical activity enhances hormone regulation and reduces oxidative stress, benefiting both male and female fertility. Chronic stress, smoking, and alcohol use disrupt hormonal pathways and damage reproductive cells, lowering fertility potential. Sleep disturbances and circadian rhythm disruption also negatively affect hormone levels and gamete health. Environmental exposures, including endocrine disruptors, heavy metals, and air pollution, harm reproductive health through hormonal and epigenetic disruptions - with possible long-term effects on offspring. Reproductive aging, especially in women after 35, reduces oocyte quality and ovarian reserve. In men, aging causes genetic and epigenetic sperm changes, increasing risks for the child. In summary, modifying lifestyle and environmental exposures offers significant potential to improve fertility outcomes and reproductive health in both sexes.

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Patient Consent:

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Conflicts of Interest

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