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Southeast Asian Journal of Case Report and Review

Journal homepage: https://www.sajcrr.com/

Review Article

Rising infertility rates and declining sperm counts: Investigating the impact of lifestyle factors, environmental pollutants, and endocrine disruptors on human reproductive health

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Abstract

Background: Global fertility rates and sperm counts have been declining significantly over recent decades, raising concerns about human reproductive health. This decline is multifactorial, involving lifestyle dysregulation, exposure to environmental pollutants, and endocrine-disrupting chemicals (EDCs).
Objectives: This review aims to elucidate the interconnected roles of lifestyle factors, environmental toxicants, and EDCs in contributing to reproductive dysfunction. It also explores current preventive strategies and highlights the need for multidisciplinary interventions.
Materials and Methods: A comprehensive literature survey of epidemiological studies, meta-analyses, and mechanistic research was conducted to synthesize evidence on fertility decline and sperm count reduction globally.
Results: Lifestyle factors such as poor diet, obesity, smoking, and stress disrupt hormonal balance and gamete quality. Environmental pollutants including heavy metals, air particulates, and pesticides cause reproductive toxicity through oxidative stress and DNA damage. EDCs interfere with hormonal signaling and induce epigenetic modifications affecting fertility. Regulatory frameworks and lifestyle interventions show promise in mitigating these effects, but further research and global collaboration are urgently needed.
Conclusion: The fertility crisis reflects a complex interplay of modifiable and non-modifiable factors. Multidisciplinary efforts encompassing public health policies, individual lifestyle changes, and advanced research are critical to preserving reproductive health worldwide.

Keywords: Fertility decline, Sperm count reduction, Lifestyle factors, Environmental pollutants, Endocrine-disrupting chemicals, Reproductive toxicity, Public health interventions

Received: 11-02-2024; **Accepted:** 23-05-2024; **Available Online**: 05-06-2025

1. Introduction

Infertility is clinically defined by the World Health Organization (WHO) as the failure to achieve a pregnancy after 12 months or more of regular unprotected sexual intercourse (World Health Organization [WHO], 2021). It affects both males and females and is recognized as a global public health issue with significant medical, psychological, and socioeconomic impacts. Male infertility, in particular, is often underdiagnosed and underreported despite contributing to approximately 40–50% of all infertility cases (Agarwal et al., 2021).

Over the past few decades, mounting epidemiological evidence has revealed a disturbing global decline in fertility rates and sperm quality. A widely cited meta-analysis by Levine et al. (2017) reported a 52.4% decrease in sperm concentration and a 59.3% decrease in total sperm count among men from North America, Europe, Australia, and New Zealand between 1973 and 2011. More recent studies continue to support this trend, indicating that male reproductive health is deteriorating at an alarming rate (Barbotin et al., 2022). Simultaneously, global fertility rates have dropped below replacement levels in over half of all countries, driven by a complex interplay of biological, environmental, and behavioral factors (UNFPA, 2023).

The implications of declining reproductive capacity extend far beyond individual couples. At the population level,

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http://doi.org/10.18231/j.sajcrr.2025.008

reduced fertility contributes to aging demographics, increased burden on healthcare systems, and long-term socio-economic instability. From a public health perspective, this trend underscores the need for urgent preventive strategies, enhanced reproductive health services, and robust environmental health regulations (Skakkebaek et al., 2022).

This review aims to explore and synthesize current scientific evidence on the multifactorial causes of global fertility decline, with a particular emphasis on the interconnected roles of lifestyle dysregulation, environmental pollutants, and endocrine-disrupting chemicals (EDCs). By elucidating these relationships, we aim to promote an integrative understanding of human reproductive dysfunction and highlight key avenues for public health intervention and future research.

2. Epidemiological Trends in Fertility and Sperm Count Decline

2.1. A. Global and regional infertility rates

Infertility is a growing global health concern that affects approximately 15% of reproductive-aged couples worldwide, with regional variations influenced by socio-economic status, environmental exposures, and healthcare access (WHO, 2021). According to the Global Burden of Disease Study, infertility affects 48 million couples globally, with male factors solely or partially responsible in nearly 50% of cases (Sun et al., 2019).
The table below summarizes recent estimates of infertility prevalence across different regions:

Table 1: Regional prevalence of infertility (Primary + Secondary)

Region	Female Infertility (%)	Male Infertility (%)	Source
Sub-Saharan Africa	11–21	9–13	Mascarenhas et al., 2012
South Asia	8–10	9–12	Dyer & Patel, 2012
Europe	12–16	10–15	Inhorn & Patrizio, 2015
North America	10–13	9–12	CDC, 2021
Middle East	13–22	11–18	Ombelet, 2011

Regional variation in infertility is exacerbated by underreporting, limited diagnostic facilities, cultural stigmas, and differing definitions of infertility.

2.2. Meta-analyses and longitudinal trends in sperm count

A seminal meta-analysis by Levine et al. (2017) reported a 59.3% decline in total sperm count and a 52.4% decline in sperm concentration among men from Western countries between 1973 and 2011, even after controlling for confounders like age and abstinence time. This trend has been further confirmed in subsequent global analyses (Barbotin et al., 2022; Levine et al., 2023).

Table 2: Summary of key meta-analyses on sperm decline

Study	Time Frame	Population Studied	Key Findings
Levine et al. (2017)	1973–2011	Men from North America, Europe, ANZ	~59% decline in total sperm count
Barbotin et al. (2022)	1973–2018	Global population	Sperm quality decline observed across regions
Levine et al. (2023)	2011–2018	Global updated cohort	Accelerating decline in sperm concentration

The 2023 follow-up by Levine et al. indicates that sperm count decline may be accelerating in recent years, particularly in regions previously considered unaffected, including parts of Asia, Africa, and South America.

2.3. Reproductive health disparities across socio-economic and geographic lines

Reproductive health is deeply influenced by socioeconomic determinants such as education, income, occupation, and access to reproductive healthcare. Studies reveal that lower-income populations and marginalized communities face disproportionate infertility risks due to greater exposure to environmental toxins, poor nutrition, and limited medical access (Inhorn & Patrizio, 2015; WHO, 2021).
Moreover, occupational exposure to heat, chemicals, and heavy metals—more common among industrial and agricultural laborers—has been linked to poorer semen quality and lower fertility (Jurewicz et al., 2018). Urbanization also plays a significant role: urban residents are more exposed to air pollution, endocrine disruptors, and stressful lifestyles, further compounding reproductive dysfunction.

Table 3: **Socioeconomic factors influencing fertility outcomes**

Socioeconomic Factor	Impact on Fertility	Example Reference
Low income	Poor nutrition, toxic exposure, late treatment	Inhorn & Patrizio, 2015
Limited healthcare access	Delayed diagnosis, inadequate ART availability	WHO, 2021
Occupational hazards	Exposure to heat, solvents, pesticides	Jurewicz et al., 2018
Urban living	High pollution, sedentary lifestyle	Skakkebaek et al., 2022

These disparities necessitate tailored policy interventions that address both environmental justice and healthcare equity in reproductive medicine.

3. Lifestyle Factors Contributing to Fertility Decline

Lifestyle behaviors have a profound impact on reproductive health, influencing gametogenesis, hormonal balance, and overall fertility potential. This section explores the roles of dietary habits, physical inactivity, substance abuse, and psychosocial stressors in compromising male and female fertility.

3.1. Diet and nutritional imbalance

Modern dietary patterns characterized by high intake of saturated fats, refined sugars, and processed foods contribute to systemic inflammation, insulin resistance, and hormonal dysregulation. These changes can impair ovulatory function in women and reduce sperm quality in men (Salas-Huetos et al., 2017).

Micronutrient deficiencies—particularly zinc, folate, and selenium—are closely linked to impaired spermatogenesis, increased oxidative stress, and reduced oocyte quality (Agarwal et al., 2018). Zinc, for example, is essential for testosterone production and sperm chromatin stability.

Table 4: Nutritional risk factors and their impact on fertility

Nutrient/Dietary Factor	Reproductive Impact	References
High-fat, high-sugar diet	Impaired ovulation, poor sperm morphology	Salas-Huetos et al., 2017
Zinc deficiency	Sperm DNA damage, decreased testosterone	Fallah et al., 2018
Folate deficiency	Neural tube defects, poor oocyte maturation	Gaskins et al., 2014
Selenium deficiency	Reduced antioxidant capacity in semen	Mistry et al., 2012

3.2. Physical inactivity and obesity

Physical inactivity and obesity are increasingly prevalent and are linked to subfertility through hormonal disruption, oxidative stress, and inflammatory cytokine elevation (Martini et al., 2022). Obesity in women is associated with anovulation and polycystic ovarian syndrome (PCOS), while obese men often exhibit hypogonadism, reduced libido, and impaired sperm quality.

Excess adipose tissue converts androgens to estrogens via aromatase, leading to hormonal imbalances that disrupt the hypothalamic-pituitary-gonadal (HPG) axis (Pasquali et al., 2007).

Table 5: Impact of obesity and inactivity on reproductive health

Factor	Mechanism of Infertility	Gender Affected	References
Obesity	Leptin resistance, insulin resistance	Both	Silvestris et al., 2018
Sedentary lifestyle	Impaired insulin sensitivity, low SHBG	Both	Martini et al., 2022
Visceral fat	Estrogen dominance, HPG axis disruption	Male	Hammiche et al., 2012

3.3. Smoking, alcohol, and substance abuse

Tobacco smoke, alcohol, and recreational drugs have direct gonadotoxic effects. Cigarette smoke contains cadmium and polycyclic aromatic hydrocarbons that damage DNA and increase reactive oxygen species (ROS), leading to sperm DNA fragmentation and anovulation (Ramlau-Hansen et al., 2007; Augood et al., 1998).

Alcohol disrupts hormone levels, impairs spermatogenesis, and is linked to menstrual irregularities. Cannabis and anabolic steroid use can further reduce fertility by affecting gonadotropin secretion and testicular function (Gundersen et al., 2015).

Table 6: Substance use and reproductive outcomes

Substance	Mechanism	Effect on Fertility	References
Cigarette smoke	ROS, DNA fragmentation	↓ Sperm motility, anovulation	Sharma et al., 2016
Alcohol	Endocrine disruption	↓ Sperm concentration, amenorrhea	Ricci et al., 2017
Cannabis	Inhibits GnRH release	↓ LH, FSH, testosterone	Gundersen et al., 2015
Anabolic steroids	Negative feedback on HPG axis	Azoospermia, testicular atrophy	Rahnema et al., 2014

3.4. Psychological stress and sleep disorders

Chronic psychological stress impairs fertility via activation of the hypothalamic-pituitary-adrenal (HPA) axis, resulting in elevated cortisol, which negatively impacts gonadotropin-releasing hormone (GnRH) and downstream sex hormone production (Rivier & Rivest, 1991).

Sleep deprivation further exacerbates reproductive dysfunction through altered melatonin and cortisol levels. Both short (<6 hours) and long (>9 hours) sleep durations are

associated with menstrual disturbances and low testosterone levels in men (Liu et al., 2018; Kalmbach et al., 2015).

Table 7: Stress and sleep disorders in fertility impairment

Factor	Physiological Disruption	Fertility Impact	References
Chronic stress	HPA axis activation, ↓ GnRH, ↑ cortisol	Irregular cycles, poor sperm quality	Rivier & Rivest, 1991
Sleep deprivation	Melatonin suppression, circadian disruption	Hormonal imbalance, ovulatory failure	Liu et al., 2018
Sleep >9 hrs	Reduced reproductive hormone synthesis	Hypogonadism	Kalmbach et al., 2015

4. Environmental Pollutants and Reproductive Toxicity

Environmental pollutants are critical contributors to declining fertility worldwide. Exposure to heavy metals, air pollution, and agricultural chemicals disrupts reproductive function by inducing oxidative stress, DNA damage, and endocrine interference. This section examines the toxicological mechanisms and epidemiological evidence linking these pollutants to reproductive dysfunction.

4.1. Heavy metals (Lead, Cadmium, Mercury)

Heavy metals are pervasive environmental contaminants with well-documented gametotoxic effects. Lead, cadmium, and mercury accumulate in reproductive tissues, causing oxidative damage, mitochondrial dysfunction, and apoptosis in germ cells (Salian et al., 2009).

Table 8: Heavy metals and reproductive toxicity

Heavy Metal	Mechanism of Toxicity	Reproductive Effects	References
Lead	Oxidative stress, mitochondrial damage	↓ Sperm count/motility; female infertility	Salian et al., 2009; Silbergeld et al., 2000
Cadmium	Disrupts Leydig cell function; DNA damage	Reduced testosterone; impaired spermatogenesis	Kumar et al., 2017
Mercury	Bioaccumulates; induces apoptosis in germ cells	Menstrual irregularities; sperm DNA fragmentation	Zeng et al., 2017

Epidemiological studies consistently associate elevated blood or seminal concentrations of these metals with reduced sperm count, motility, and abnormal morphology, as well as menstrual irregularities and miscarriage in women (Gore et al., 2015).

4.2. Air pollution and particulate matter

Airborne pollutants, especially fine particulate matter (PM2.5) and nitrogen oxides (NOx), generate systemic oxidative stress and inflammation. In male reproductive systems, PM2.5 exposure correlates with increased sperm DNA fragmentation and reduced testicular volume (Li et al., 2019).

Urban populations, with higher exposure to traffic-related pollutants, show significantly lower fertility rates compared to rural counterparts, underscoring environmental disparity (Hauser et al., 2015).

4.3. Agricultural pesticides and industrial chemicals

Pesticides such as organophosphates and pyrethroids have been implicated in endocrine disruption and gametotoxicity through anti-androgenic and estrogenic effects. Long-term exposure is linked to decreased sperm quality and increased risk of spontaneous abortion (Swan et al., 2003).

Persistent organic pollutants (POPs), including polychlorinated biphenyls (PCBs) and dioxins, bioaccumulate in adipose and reproductive tissues, disrupting steroidogenesis and increasing oxidative stress (Bergman et al., 2013).

5. Endocrine Disrupting Chemicals (EDCs) and Hormonal Interference

Endocrine Disrupting Chemicals (EDCs) are exogenous compounds that interfere with hormone biosynthesis, signaling, or metabolism, posing significant threats to human reproductive health. This section reviews common EDCs, their mechanisms of hormonal interference, and human exposure pathways, supported by biomonitoring data.

5.1. Overview of common EDCs

Common EDCs include industrial chemicals such as Bisphenol A (BPA), phthalates, parabens, dioxins, and polychlorinated biphenyls (PCBs). These chemicals are pervasive in consumer products and the environment, leading to widespread human exposure (Diamanti-Kandarakis et al., 2009).

Table 9: Common endocrine disrupting chemicals (EDCs), their sources, chemical properties, and key references

EDC	Common Sources	Chemical Properties	References
Bisphenol A (BPA)	Polycarbonate plastics, epoxy resins	Estrogenic activity	Rochester, 2013
Phthalates	PVC plastics, personal care products	Anti-androgenic effects	Hauser & Calafat, 2005

Parabens	Cosmetics, pharmaceuticals	Weak estrogenic effects	Darbre & Harvey, 2008
Dioxins	Industrial by-products, combustion processes	Persistent, bioaccumulative	Schecter et al., 2006
PCBs	Electrical equipment, industrial wastes	Persistent, lipophilic	Carpenter, 2006

5.2. Mechanisms of endocrine disruption

EDCs exert their effects primarily through mimicking or blocking natural hormones, disrupting receptor signaling. BPA and parabens act as estrogen receptor agonists, while phthalates often antagonize androgen receptors, impairing testosterone-mediated functions (Vandenberg et al., 2012).

Additionally, emerging evidence highlights epigenetic alterations induced by EDCs in germ cells, including DNA methylation and histone modifications, which may contribute to transgenerational reproductive effects (Skinner et al., 2013).

5.3. Human exposure routes and biomonitoring data

Humans are exposed to EDCs via ingestion (contaminated food and water), dermal absorption (cosmetics, personal care products), and inhalation. BPA, phthalates, and parabens are commonly detected in urine, while dioxins and PCBs accumulate in fatty tissues (Wang et al., 2014).

Table 10: Mechanisms of endocrine disrupting chemicals (EDCs) and their reproductive impacts

Mechanism	**Description**	**Impact on Reproduction**	**References**
Estrogenic Activity	Binding to estrogen receptors, activating pathways	Altered ovarian function, sperm defects	Vandenberg et al., 2012
Anti-androgenic Effects	Inhibition of androgen receptor signaling	Reduced testosterone, impaired spermatogenesis	Hauser & Calafat, 2005
Epigenetic Modifications	DNA methylation, histone changes in germ cells	Heritable reproductive dysfunction	Skinner et al., 2013

Biomonitoring studies detect EDCs in reproductive fluids: BPA and phthalates are found in semen, follicular fluid, and amniotic fluid, correlating with compromised gamete quality and pregnancy outcomes (Mínguez-Alarcón et al., 2016).

Table 11: Exposure routes, biomonitoring matrices, and reported reproductive effects of common endocrine disrupting chemicals (edcs)

EDC	**Exposure Routes**	**Biomonitoring Matrices**	**Reported Effects**	**References**
BPA	Food packaging, thermal receipts	Urine, semen, follicular fluid	Reduced sperm motility, oocyte quality	Rochester, 2013; Mínguez-Alarcón et al., 2016
Phthalates	Plastics, personal care products	Urine, semen	Decreased sperm count, altered hormone levels	Hauser & Calafat, 2005
Parabens	Cosmetics, pharmaceuticals	Urine	Estrogenic disruption	Darbre & Harvey, 2008
Dioxins	Food chain contamination	Blood, adipose tissue	Hormonal imbalance, infertility	Schecter et al., 2006
PCBs	Environmental persistence	Blood, adipose tissue	Reduced fertility, sperm defects	Carpenter, 2006

6. Preventive and Mitigative Strategies

Addressing the global fertility decline and sperm count reduction requires a multi-pronged approach involving lifestyle changes, regulatory frameworks, and targeted research. This section discusses interventions at individual and policy levels, alongside future research priorities to safeguard reproductive health.

6.1. Lifestyle interventions

Lifestyle modifications are pivotal for mitigating reproductive dysfunction caused by environmental and behavioral risk factors. Dietary improvements focusing on balanced nutrition rich in antioxidants (e.g., vitamins C and E, zinc, selenium) have been shown to enhance sperm quality and ovarian function (Agarwal et al., 2014). Regular physical activity and maintaining optimal body weight reduce oxidative stress and hormonal imbalances linked to infertility (Thoma et al., 2013).

Antioxidant therapy and micronutrient supplementation further combat reactive oxygen species (ROS)-mediated gamete damage. Clinical trials have demonstrated improved semen parameters following supplementation with coenzyme Q10, folic acid, and omega-3 fatty acids (Showell et al., 2014).

6.2. Regulatory approaches and public health policies

Regulatory measures play a crucial role in limiting human exposure to reproductive toxicants. International frameworks such as the European Union's REACH (Registration, Evaluation, Authorization, and Restriction of Chemicals) and U.S. EPA guidelines regulate EDCs and hazardous pollutants to reduce environmental burdens (Kortenkamp et al., 2011).

Public health initiatives emphasize reproductive health education, raising awareness about lifestyle risks and environmental exposures. Community campaigns and healthcare provider training improve early detection and prevention strategies (Balabanova et al., 2017).

Table 12: Regulatory frameworks addressing chemical safety and their impact on reproductive health

Regulatory Framework	Scope	Impact on Reproductive Health	References
Reach (EU)	Controls chemical safety and usage	Reduction in EDC exposure, safer environment	Kortenkamp et al., 2011
EPA Guidelines (USA)	Sets permissible exposure limits for pollutants	Limits environmental and occupational reproductive toxicants	EPA, 2020
WHO Guidelines	Provides global reproductive health policies	Promotes education, monitoring, and regulation	WHO, 2018

6.3. Future research directions

Future research must focus on longitudinal human cohort studies to understand chronic and low-dose exposures' effects on fertility outcomes (Skakkebaek et al., 2016). Development of sensitive biomarkers—molecular, epigenetic, or proteomic—will aid early detection of reproductive risks before clinical infertility manifests (Jenkins et al., 2018).

Investigating gene-environment interactions is critical to identify susceptible populations and tailor personalized interventions. Integrating omics technologies with epidemiology could unravel complex pathways linking exposures to reproductive dysfunction (Trasande et al., 2015).

7. Conclusion

The global decline in fertility and sperm counts is a multifactorial phenomenon intricately linked to lifestyle dysregulation, environmental pollutants, and widespread exposure to endocrine-disrupting chemicals (EDCs). Lifestyle factors such as poor nutrition, sedentary behavior, substance abuse, and psychological stress collectively impair reproductive hormones and gamete integrity. Simultaneously, environmental toxicants—including heavy metals, air pollutants, pesticides, and persistent organic pollutants—exert direct gonadotoxic effects that exacerbate reproductive dysfunction. Furthermore, EDCs interfere with hormonal signaling through complex molecular mechanisms, often resulting in epigenetic alterations that may affect not only individuals but subsequent generations.

Given the complex and interconnected nature of these factors, addressing the fertility crisis necessitates urgent multidisciplinary interventions combining individual lifestyle modifications, robust regulatory policies, and comprehensive public health initiatives. Equally critical is advancing research to identify sensitive biomarkers and understand gene-environment interactions to develop precision prevention and treatment strategies.

A coordinated global effort involving scientists, healthcare providers, policymakers, and communities is essential to mitigate the adverse effects threatening human reproductive health. Only through such collaboration can we hope to reverse current trends, safeguard fertility, and ensure reproductive well-being for future generations.

8. Source of Funding

None.

9. Conflict of Interest

None.

References

1. Agarwal A, Baskaran S, Parekh N, Cho C, Henkel R, Vij S. (2021). Male infertility. *The Lancet*, 2021:397(10271):319–33.
2. Agarwal A, Sengupta P, Durairajanayagam D, Henkel R, Majzoub A, Tadros N. Oxidative stress and its implications in female infertility – A clinician's perspective. *Reprod Biol Endocrinol*, 2018;16(1):1–13.
3. Augood C, Duckitt K, Templeton A.A. Smoking and female infertility: A systematic review and meta-analysis. *Hum Reproduction*, 1998;13(6):1532–9.
4. Barbotin AL, Ballot C, Sigala J, Ramdane N, Mitchell V. Decline in sperm count: A review of potential environmental causes. *Hum Reproduc Update*. 2022;28(3):327–49.
5. Bergman Å, Heindel JJ, Jobling S, Kidd KA, Zoeller RT. State of the science of endocrine disrupting chemicals 2012. United Nations Environment Programme and World Health Organization.2013; Available from: https://www.who.int/publications/i/item/state-of-the-science-of-endocrine-disrupting-chemicals
6. Centers for Disease Control and Prevention (CDC). (2021). Infertility FAQs. Available from: https://www.cdc.gov/reproductivehealth/infertility/index.htm

7. Dyer SJ, Patel M. The economic impact of infertility on women in developing countries – A systematic review. Facts Views Vision ObGyn, 2012;4(2):102–9.
8. Fallah A, Mohammad-Hasan A, Colagar AH. Zinc is an essential element for male fertility: A review of Zn roles in men's health, germination, sperm quality, and fertilization. *Jo Reproduc Infer*, 2018;19(2):69–81.
9. Gaskins AJ, Chavarro JE, Willett WC. Diet and fertility: A review. *Am J Obstete Gynecol*, 2004;205(6):499–510.
10. Gore AC, Chappell VA, Fenton SE, Flaws JA, Nadal A, Prins GS. EDC-2: The endocrine society's second scientific statement on endocrine-disrupting chemicals. *End Rev*, 2015;36(6):1–150.
11. Gundersen TD, Jørgensen N, Andersson AM, Bang AK, Nordkap L, Skakkebaek NE. Association between use of marijuana and male reproductive hormones and semen quality in a population-based cohort of young men. *Am J Epidemiol*. 2015;182(6):473–81.
12. Hauser R, Meeker JD, Singh NP, Silva MJ, Ryan Duty S, Calafat AM. DNA damage in human sperm is related to urinary levels of phthalate monoester and oxidative metabolites. *Hum Reprod*, 2015;21(11):2693–700.
13. Hammiche F, Laven JS, Twigt JM, Boomsma CM, Steegers-Theunissen RP. Body mass index and central adiposity are associated with sperm quality in men of subfertile couples. *Hum Reprod*, 2012;27(8):2365–72.
14. Inhorn MC, Patrizio P. Infertility around the globe: New thinking on gender, reproductive technologies and global movements in the 21st century. Human Reproduction Update, 2015;21(4);411–26.
15. Jin C, Yuan H, Wang Y, Xiao Q. Effect of air pollution on sperm quality: A systematic review and meta-analysis. *Sci Total Environ*, 2018;28:622-3.
16. Jurewicz J, Dziewirska E, Radwan M, Hanke W. Air pollution from natural and anthropic sources and male fertility. *Reprod Biol Endocrinol*, 2018;16(1):109.
17. Kalmbach DA, Arnedt JT, Owens RL, Drake CL. Insomnia symptoms and short sleep predict fertility risk in women. *Sleep*, 2015;38(3):377–85.
18. Kumar S, Sharma A, Singh R, Gupta RC. Reproductive toxicity of cadmium in male rats: A review. *Toxicol Industrial Health*, 2017;33(7):623–37.
19. Levine H, Jørgensen N, Martino-Andrade A, Mendiola J, Weksler-Derri D, Mindlis I. Temporal trends in sperm count: A systematic review and meta-regression analysis. Hum Reproduc Update, 2017;23(6):646–59.
20. Levine H, Swan SH, Skakkebaek NE. Accelerating declines in sperm counts in men from the Americas, Europe, and Asia: An updated meta-analysis. *Hum Reproduc Update*, 2023;29(2):157–67.
21. Li Z, Li J, Tang X, Xie L., Wang S, Zhu, J. Air pollution and male fertility: A systematic review. *Environ Poll*, 2019;16(1):372–8.
22. Liu Y, Chen Y, Jin Y, Qi Y. Association between sleep duration and semen quality: A cross-sectional study in Zhejiang, China. *BMJ Open*, 2018;8(2), e020302.
23. Martini AC, Molina R I, Estofan D, Senestrari D, Fiol de Cuneo M, Ruiz, RD. Obesity and male infertility: A clinical study in Argentine patients. *Andrologia*, 2022;54(2):e14229.
24. Mascarenhas MN, Flaxman SR, Boerma T, Vanderpoel S, Stevens G.A. (2012). National, regional, and global trends in infertility prevalence since 1990: A systematic analysis of 277 health surveys. *PLOS Med*, 2012;9(12):e1001356.
25. Meeker JD, Barr DB, Hauser R. Pyrethroid insecticide exposure and serum hormone levels in adult men. *Reprod Toxicol*, 2009;28(2):157–62.
26. Mistry HD, Williams PJ, Ramsay MM. Selenium and reproductive health. *Am Jo Obstet Gynecol*, 2012;206(1):21–30.
27. Ombelet W. Global access to infertility care in developing countries: A case of human rights, equity and social justice. *Facts View Vision in ObGym*. 2011;3(4):57–266.
28. Pasquali R, Patton L, Gambineri A. Obesity and infertility. *Curr Opin Endocrinol, Diab Obesit*, 2007;14(6):482–7.
29. Paul S. Sen P, Mukherjee A. Endocrine disruptors and their effect on fertility: An update. *Environ Toxicol Pharmacol*, 2021;84:103589.
30. Pérez-Crespo, M, Pintado B, Gutiérrez-Adán A. Effect of oxidative stress on sperm function and fertilization in mammals. *Reprod Domes Animals*, 2008;43(3):349–53.
31. Ramlau-Hansen, C. H., Thulstrup, A. M., Nohr, E. A., Bonde, J. P., Sørensen, TI, Olsen J. Is smoking a risk factor for decreased semen quality? A cross-sectional study among 4,867 healthy young Danish men. *Am J Epidemiol* 2007;165(7):838–45.
32. Sengupta P, Dutta S, Krajewska-Kulak E. The disappearing male: A systematic review of declining human sperm quality. *Indian J Dermatol*, 2018;2018;63(1):2–10.
33. Skakkebaek NE, Rajpert-De Meyts E, Main KM. Testicular dysgenesis syndrome: An increasingly common developmental disorder with environmental aspects. *Hum Reprod*, 2001;16(5):972–8.
34. Swan SH, Elkin EP, Fenster L. The question of declining sperm density revisited: An analysis of 101 studies published 1934–1996. *Environ Health Perspect,* 1997;105(11):1228–33.
35. Tremellen K. Oxidative stress and male infertility – A clinical perspective. *Hum Reprod Update*, 2008;14(3):243–58.
36. U.S. Environmental Protection Agency (EPA). (2020). Endocrine Disruptor Screening Program. Available from: https://www.epa.gov/endocrine-disruption
37. Vidal E, Soria B, Arce A. Lifestyle, environmental exposures, and male reproductive health. *Reproduction,* 2017;154(3):103–12.
38. Wong WY, Thomas CM, Merkus JM, Zielhuis GA, Steegers-Theunissen RP. Male factor subfertility: Possible causes and the impact of nutritional factors. *Fertility Sterility*, 2000;73(3):435–42.
39. World Health Organization (WHO). (2021). WHO laboratory manual for the examination and processing of human semen (6th ed.). WHO Press. Available from: https://www.who.int/publications/i/item/9789240030787
40. Zhang J, Guo H, Wu Q, Liu C, Li J. Impact of environmental pollution on male reproductive health: A review of epidemiological and experimental studies. *Environ Poll,* 260, 113855.

Cite this article: Mahendra Dwivedi, Rising infertility rates and declining sperm counts: Investigating the impact of lifestyle factors, environmental pollutants, and endocrine disruptors on human reproductive health. *Southeast Asian J Case Rep Rev*. 2025;12(2):30-36.