International Journal of Community Medicine and Public Health
Markanday BK et al. Int J Community Med Public Health. 2026 Jan;13(1):464-470
http://www.ijcmph.com
pISSN 2394-6032 | eISSN 2394-6040

DOI: https://dx.doi.org/10.18203/2394-6040.ijcmph20254462

Review Article

An overview on some important factors affecting male fertility

Bhagwati Kumar Markanday[1], Arunima Sur[1], Shobha Gupta Gawri[2]*

[1]Amity Institute of Biotechnology, Amity University Chhattisgarh, Raipur, Chhattisgarh, India
[2]Seth Phoolchand Agrawal Smriti Mahavidhyalaya, Nawapara-Rajim Chhattisgarh, India

Received: 02 August 2025
Revised: 06 December 2025
Accepted: 08 December 2025

***Correspondence:**
Dr. Shobha Gupta Gawri,
E-mail: shobhagawri@gmail.com

ABSTRACT

Infertility is a disease of the reproductive system. It makes a person unable to have children. It can affect both man and women. Any dysfunction in the reproductive system may lead to infertility. Infertility in males is observed as one of the major problem faced by several advanced countries. Over the past several years, a decline in male reproductive health has been reported. Globally, it has been noticed that the incidences of testicular cancer (TC) and poor sperm quality is increasing with time. Certain drugs, lifestyle, tobacco, smoking and alcohol all have a short and long term health effects, including effects detrimental to the production and quality of sperms. In his review we have tried to through some light on the important factors such as lifestyle factors and environmental factors, that can greatly influence over all health wellbeing including fertility.

Keywords: Male infertility, Lifestyle factors, Alcohol, Tobacco, Smoking occupational hazards, Environmental factors

INTRODUCTION

Infertility is a serious health issue, affecting approximately 8%–12% of couples worldwide. As per the World Health Organization reports 60–80 million couples are suffering from infertility every year. Probably between 15 and 20 million (25%) are in India alone. According to a report by the World Health Organization, one in every four couples in developing countries is affected by infertility.[1] Infertility is a disease of the male or female reproductive system defined by the failure to achieve a pregnancy after 12 months or more of regular unprotected sexual intercourse.[2] Facing infertility can be very difficult for both men and women it is emotionally stressful for most couples.[3] Globally, it has been noticed that the incidences of testicular cancer (TC) and poor sperm quality is increasing with time. In about 40% infertile couples male infertility plays a key role in successful.[3] Certain drugs, lifestyle, tobacco, smoking and alcohol all have a range of short and long term health effects. These include harmful effects for the production and quality of sperms. Reports reveal that lifestyle factors, like alcohol intake and cigarette smoking affect male fertility. This review focuses on key lifestyle and environmental factors that are associated with male infertility such as smoking cigarettes, alcohol intake, psychological stress, advanced paternal age, obesity, and dietary practices, environmental factors such as occupational hazards, testicular heat stress, exposure to endocrine disruptive agrochemical, electromagnetic radiation from mobile phone use.

LIFESTYLE FACTORS

Some lifestyle factors include alcohol intake and cigarette smoking, which have been reported to show negative impact on male fertility. Cigarette smoking and alcohol intake, both are largely avoidable. They both have a negative impact on male fertility.[4] In the areas of in-vitro fertilization (IVF) and pregnancy, they influence oocyte production, fertilization rates, pregnancy and pregnancy

loss, while chronic, low-grade oxidative stress may result in poor outcomes for some IVF cases.[5]

Alcohol

A recent study demonstrated significantly inferior semen quality (sperm analysis parameters) and hormonal characteristics in daily drinkers as compared to occasional drinkers.[6] Other research suggests that alcohol consumption also impacts on sperm morphology and sperm count.[7] Higher cases of teratozoospermia and oligozoospermia were reported in heavy alcohol consumers against men who do not consume alcohol.[3] In addition, an analysis which included 57 studies found that alcohol consumption led to a significantly reduced semen volume.[8] Heavy drinking may be one factor causing frequent changes in testicular function. Moderate consumption of alcohol may affect semen quality more often than previously thought, whereas high alcohol consumption may even be associated with serious disorders of spermatogenesis. Spermatogenesis appears to gradually decline with increasing levels of alcohol intake. Partial or complete spermatogenic arrest and Sertoli cell-only syndrome were more commonly present amongst heavy drinkers compared to non-drinkers. Chronic alcohol intake was found to have a detrimental effect on both semen quality and the levels of male reproductive hormones.[9] A positive correlation with daily alcohol intake was observed amongst alcoholics, with percentage of cases increasing from mild to heavy alcoholic sub-groups.[3]

Tobacco and smoking

Now a day's cigarette smoking is considered as a major public health problem.10 A high prevalence of smoking is observed in young adult males during their reproductive period.[11] It is known to be a potential risk factor for decreased male fertility.[12] One study showed that smokers had significantly lower semen volume, sperm concentration, sperm motility, total sperm count, sperm morphology, free testosterone and follicle stimulating hormone ($p < 0.05$ respectively), compared with non-smokers. Smokers were at a higher risk of developing oligospermia, Asthenozoospermia and Teratozoospermia (OR=3.1, 4.2 and, 4.7; $p<0.05$) than non-smokers.[10,13] Cigarette smoking leads an increased exposure to hazardous substances such as tar, nicotine (which are highly addictive), carbon monoxide, and heavy metals (e.g. cadmium and lead).[14] A large meta-analysis involving males from 26 countries/regions concluded that smoking causes a decline in sperm quality in both fertile and infertile men.[8] A 13–17% lower sperm concentration was reported in male smokers as compared to that of non-smokers. Cigarette smoking has been found negatively associated with sperm count, motility, and morphology. Besides its association with impaired male fertility, tobacco smoking is also reported responsible for increased DNA damage, aneuploidies, and mutations in sperm.[15]

Recreational drugs

Marijuana, cocaine, anabolic androgenic steroids (AAS), opiates (narcotics), and methamphetamines are examples of prohibited drugs that exert a negative impact on male fertility.[16] Marijuana is one of the most commonly used drugs around the world, and it acts both centrally and peripherally to cause abnormal reproductive function. Marijuana contains cannabinoids which bind to receptors located on reproductive organs such as the uterus or the ductus deferens. In males, cannabinoids have been reported to reduce testosterone released from Leydig cells, modulate apoptosis of Sertoli cells, decrease spermatogenesis, decrease sperm motility, decrease sperm capacitation and decrease acrosome reaction.[17] Marijuana smoking has been found to lower sperm concentration and total sperm count amongst youngsters, and this effect was further amplified when marijuana was used in combination with other recreational drugs.[18] Cocaine is a highly addictive, strong stimulant drug. Male rats given high doses of cocaine chronically before mating had lower pregnancy rates and offspring birth weights. Both acute and chronic exposure to cocaine disrupted spermatogenesis and damaged the testicular ultrastructure. Long-term cocaine users were associated with lower sperm concentration and motility, and a higher fraction of sperm with abnormal morphology.[19] Studies have reported permanent ill-effect of steroid use on male fertility.[20]

Obesity

Obesity or excess weight is not only linked to increased risk of chronic diseases but also increases the risk of reproductive problems.[21] The patho-physiology of obesity has been reported to have negative impact on male fertility. A study over 1558 younger men having paramilitary physical activity found that overweight men had reduced sperm concentration as compared to men with normal weight.[22] Overweight and obesity are associated with excessive fat accumulation, which can be evaluated using the body mass index (BMI). Overweight (BMI 25–<30 kg/m^2) and obese (BMI <30 kg/m^2) males are associated with a decrease in sperm quality and a higher risk of infertility. Recent reports suggest that weight plays an important role in fertility, and controlling and maintaining an ideal weight may provide a way for couples to increase their fertility. Men and women who are underweight or overweight are at risk of changes in hormone levels which heavily influence their fertility.[12] A systematic review of 30 studies comprising 115158 males found that paternal obesity was associated with lowered male reproductive potential. Men who were obese had a higher percentage of sperm with DNA fragmentation, abnormal morphology, and low mitochondrial membrane potential (MMP), and were more likely to be infertile. Obese men who opt for ART have reduced rates of live birth per ART cycle.[23] Obese men are three times more likely to exhibit a reduction in semen quality than men of a normal. There are several studies that demonstrate that

an increase in BMI is correlated with a decrease in sperm concentration and motility. 22,2425 Increased DNA damage in sperm have been reported in over weight men.[26] A relationship also exists between obesity and erectile dysfunction (ED).[27]

Psychological Stress

Stress whether it is physical, social, or psychological has now become an important issue in the society. Infertility itself is stressful, due to the societal pressures, testing, diagnosis, treatments, failures, unfulfilled desires, and even financial costs.[28] Stress and depression are thought to reduce testosterone and luteinizing hormone (LH), disrupt gonadal function and ultimately reduce spermatogenesis and sperm parameters.[29] Reports are there which show that positive moods have been correlated with increased chances of delivering a live baby while higher levels of anxiety increased chances of stillbirth. Increased stress decreases the fertilization of oocytes. A study was conducted to evaluate the effects of psychological stress on reproductive hormones and sperm quality in the male partner of infertile couples. The level of psychological stress was assessed using the Hospital Anxiety and Depression Score (HADS) questionnaire. 27% of the studied subjects were found to have significant psychological stress. A meta-analysis of 57 cross-sectional studies involving 29914 participants reported that psychological stress could lower sperm concentration and progressive motility, and increase the fraction of sperm with abnormal morphology.[8] A study revealed the association between psychological stress in the form of occupational, life stress, family functioning, and semen quality. It was found that occupational stress was negatively associated with semen quality, with a positive association between stress and percentage of sperm with DNA damage (Durairajanayagam, 2018).[12] These findings suggest that relationship was found between psychological stress and sperm concentration, motility, and morphometry in a study of 157 volunteers who were enrolled in a prepaid health plan. Investigator measured psychological job stress and life-event stress by telephone interview.

Advanced paternal age

Among other factors, fertility is also affected by the age of an individual. Attempting pregnancy before the age of 30 for women and before 35 for men may provide the highest chances of successful fertilization. Due to pursuit of education and other factors, many couples are choosing to delay child- bearing. Reports are there regarding fertility concerns for the aging male.[30] Fertility peaks and then decreases over time in both men and women, thus the reproductive timeline may be one aspect to consider when determining the ideal time to start a family. In men, testosterone levels begin to decrease and hypogonadism results with increasing age. Semen parameters begin to decline as early as age 35 (Dunson et al., 2004); semen volume and motility both decrease and morphology may become increasingly abnormal.[32] After the age of 40, men can have significantly more DNA damage in their sperm, as well as decline in both motility (40%) and viability (below 50%) (n=504, $p<0.001$). The reproductive timeline for women is complex. Increasing age increases a woman's time to pregnancy. When under the age of 30, a woman's chances of conceiving may be as high 71%; when over 36, it may only be 41%.[32] In addition, chromosomal abnormalities and aneuploidy may increase the risk of spontaneous abortion and implantation loss with increasing age.[31]

Advanced maternal age is defined as the age of 35 years, beyond which there is significantly increased risks of adverse reproductive outcome for women. A meta-analysis of 90 studies involving 93839 participants reported an age-associated decline in semen volume, sperm total, and progressive motility, normal sperm morphology along with an increase in DNA fragmentation.[12] These age-dependent changes in semen quality could probably be attributed to normal physiological changes in the reproductive tract that occur with ageing, decreased capacity for cellular and tissue repair of damage induced by exposure to toxicants or diseases, and increased chances with age of having reproductive damage resulting from exogenous exposures such as smoking or infection.[33] The fact that both normal physiological processes and environmental factors could be held responsible for the effects of ageing on the male reproductive system adds to its complexity.[34] In couples undergoing IVF, implantation and pregnancy rates decreased with increasing paternal age when the maternal age was between 30–34 years.[35]

Diet

Diet and nutrition plays an important role in semen quality. Eating a healthy and varied diet may be a key part of maintaining good overall health. However, there are certain vitamins and food groups that could have a greater impact on reproductive health than others. Aspects of a male's diet may have an impact on his fertility. Consuming a diet rich in carbohydrates, fiber, folate, and lycopene as well as consuming fruit and vegetables correlates with improved semen quality.[36] A recent exhaustive systematic review of observational studies concluded that intake of a healthy, balanced diet could improve semen quality and fecundity rates amongst males.[37] Vegetables and fruits, fish and poultry, cereals and low-fat dairy products were amongst the foods positively associated with sperm quality. However, diets consisting of processed meat, full-fat dairy products, alcohol, coffee, and sugar-sweetened beverages were associated with poor semen quality and lower fecundity rates.[37]

ENVIRONMENTAL FACTORS

Male reproductive system is highly sensitive to environmental factors that may lead to infertility. Certain

chemicals including pesticides, herbicides, cosmetics, preservatives, cleaning materials, municipal and private wastes, pharmaceuticals and industrial by-products enter our bodies in a variety of forms. Exposure to these chemicals contaminants, which are estrogen like substances and endocrine disruptors, has been found to be one of the possible factors contributing to the increasing male infertility.[38]

Occupational exposure to pesticides

The term "occupational exposure" refers to a potentially harmful exposure to hazardous chemicals at workplace. These may be toxic substances just like pesticides and herbicides. Several studies have demonstrated that human semen quality has declined over the past decades and some of them have associated it with occupational exposure to pesticides. Agricultural chemicals were found to reduce semen quality in fertile men. Pesticides form a large group of heterogeneous chemicals, which are used to kill insects, weed, and fungi.[39] Pesticide exposure mainly occurs through ingestion of contaminated food, but can also occur through dermal contact and inhalation. Exposure to pesticides can be assessed using blood, urine or semen samples as. Timing, combinations of agrochemicals, duration of exposure, and dose may play critical roles in pregnancy outcomes. Nevertheless, several currently used pesticides, especially those having endocrine disruptive properties, are known to adversely impair reproductive competence of males working in laboratories, fields, clinics, or factories.[40] Azoospermia was diagnosed in six factory workers and the suspected cause was chronically exposed to the chemical 1,2-dibromo-3-chloropropane (D.B.C.P.).

Exposure to pesticides affects many body organs including reproductive system. Spermatogenesis, testis weight and sperm parameters such as, sperm density and motility, sperm counts, viability, inducing sperm DNA damage, abnormal sperm morphology affected by Organophosphoruses.[41] Pesticides may directly damage spermatozoa, alter Sertoli cell or Leydig cell function, or disrupt the endocrine function in any stage of hormonal regulation (hormone synthesis, release, storage, transport, and clearance; receptor recognition and binding).[42] Additionally, pesticides have shown decreased normal sperm morphology, count, volume and motility.[40] Organophosphate exposure also reduced semen volume and increased pH. Exposure to phthalates decreased sperm concentration, normal morphology, and motility.[40]

Occupational exposures to pesticides in parents working in areas of high pesticide application area may increase parental risk of adverse reproductive and pregnancy outcomes poor fertilization, fetal death, stillbirth, spontaneous abortion, preterm delivery, and congenital anomalies.[41-43] There are reports people experienced a decreased birth rate due to well-known occupational testicular toxin of Dibromochloropropane. A study observed Oligozoospermia and Azoospermia in workers of a DBCP-producing factory. A similar report was published a year later in Israel about workers in a pesticide plant who came in contact with the same material. A research group studied the relation between occupational fenvalerate exposure and spermatozoa DNA damage of pesticide factory workers.[44] Results indicated that occupational exposure to fenvalerate exposure induced a significant increase in sperm DNA damage. Fenvalerate induced a significant decrease in testis weight, epididymal sperm counts, and sperm motility and marker testicular enzymes for testosterone biosynthesis.[44] A similar study reported epidemiological data showing increase in prevalence of diseases associated with endocrine-disrupting chemicals, such as breast, prostate, and testis cancer, diabetes, obesity, and decreased fertility over the last 50 years. A study found that men exposed to dioxins had a greater number of morphologically abnormal sperm and low linear motility. A research group reported that increased exposure to polychlorinated biphenyls (PCBs) was associated with decreased sperm count, motility, and normal morphology.[39] Phthalates, BPA, dioxins, and PCB all were found associated with decreased semen quality; however, their mechanisms of action affect different parts of the endocrine system in relation to semen quality and male reproduction.[45]

A combination of the Comet and TUNEL assays would offer more comprehensive information for a better understanding of sperm DNA damage, and the biological significance of sperm DNA damage in sperm function and male infertility. DNA damage may result in cell death or induction of mutations. In sperm, DNA damage may carry mutations into the next generation or result in male infertility. DNA integrity may become more important in evaluating sperm health for male infertility.[44]

All these studies suggest that pesticides may impair semen quality in humans which indirectly affects fertility in both males amd females.

Occupational heat exposure

It is well known that in most mammals, spermatogenesis is temperature dependent. This temperature dependence has been clearly demonstrated by several experimental studies showing that artificial increases in scrotum or testicle temperature in fertile men reduce both sperm output and quality (Thonneau et al). Several studies have reported a gradual decline in sperm production in men. Endocrine disrupters as well as lifestyle have been suggested as risk factors. One lifestyle factor that may affect human fertility is driving a vehicle for a prolonged period. Several authors have suggested that driving position may increase the scrotal temperature. Thus, after 2h of driving, the mean scrotal temperature was increased by 1.7°C on the left side and 2.2°C on the right side when compared to the corresponding mean scrotal temperatures during the walking period (Bujan et al). This study concluded that occupational heat exposure is a significant

risk factor for male infertility, affecting sperm morphology and resulting in delayed conception.[46]

Other lifestyle risk factors

Genital heat stress resulting from scrotal hyperthermia is a substantial risk factor for male infertility. Prolonged hours of sitting or exposure to radiant heat, varicocele, and cryptorchidism can all lead to testicular heat stress.[47] Elevated scrotal temperatures lead to spermatogenic arrest, germ cell apoptosis, oxidative stress, and sperm DNA damage. Sleep disturbances may possibly have adverse effects on male fertility, as semen volume was lower in patients with difficulty in initiating sleep, including those who smoked or were overweight.[48]

Exposure of radiation

Exposure to various kinds of radiation can have lasting effects in humans. It may also depend upon the amount and duration of radiation. Radiation in the form of x-rays and gamma rays can be devastating to the sensitive cells of the human body, including germ and Leydig cells. Leydig cells, seminiferous tubules, and spermatozoa are the main targets of the damage caused by mobile phones on the male reproductive tract. In particular, cellular phone exposure reduces testosterone biosynthesis, impairs spermatogenesis, and damages sperm DNA.[49] The damage, depending on the age of the patient and dose, can result in permanent sterility.[50] A study conducted showed a positive correlation between mobile phone radiation exposure, DNA-fragmentation level and decreased sperm motility. There have been a number of studies demonstrating negative effects of the radiofrequency electromagnetic waves (RFEMW) utilized by cell phones on fertility. Cell phone usage has been linked with decreases in progressive motility of sperm ,decreases in sperm viability, increases in ROS, increases in abnormal sperm morphology, and decreases in sperm counts.[51,52] One meta-analysis reported that mobile phone exposure is associated with reduced sperm motility and viability.[53] One study evaluating 52 men demonstrated that men who carried a cell phone around the belt line or hip region were more likely to have decreased sperm motility compared to men who carried their cell phones elsewhere or who did not carry one at all.[54]

CONCLUSION

The major lifestyle factors discussed in the present review are amongst the multiple potential risk factors that could impair male fertility. This review focuses on key lifestyle factors that are associated with male infertility such as alcohol intake, tobacco consumption, cigarettes smoking, psychological stress, advanced paternal age, obesity, and dietary practices. Other factors environmental factors such as occupational hazards, testicular heat stress, exposure to endocrine disruptive agrochemical, electromagnetic radiation from mobile phone use are briefly discussed. This study aims at evaluating the impact of major lifestyle factors and environmental exposures to Agrochemical, (pesticides, herbicides) on semen quality, by reviewing published literature. We suggest that lifestyle factors and exposure to different chemicals can greatly influence over all health including fertility.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Mascarenhas MN, Flaxman SR, Boerma T, Vanderpoel S, Stevens GA. National, regional, and global trends in infertility prevalence since 1990: a systematic analysis of 277 health surveys. PLoS Med. 2012;9(12):e1001356.
2. World Health Organization. ICD-11: International Classification of Diseases 11th Revision. Geneva: WHO; 2018.
3. Gaur DS, Talekar MS, Pathak VP. Alcohol intake and cigarette smoking: impact of two major lifestyle factors on male fertility. Indian J Pathol Microbiol. 2010;53(1):35-40.
4. Brugo-Olmedo S, Chillik C, Kopelman S. Definition and causes of infertility. Reprod Biomed Online. 2001;2(1):41-53.
5. Firns S, Cruzat VF, Keane KN, Joesbury KA, Lee AH, Newsholme P, et al. The effect of cigarette smoking, alcohol consumption and fruit and vegetable consumption on IVF outcomes: a review and presentation of original data. Reprod Biol Endocrinol. 2015;13(1):134.
6. Condorelli RA, Calogero AE, Vicari E, La Vignera S. Chronic consumption of alcohol and sperm parameters: our experience and the main evidences. Andrologia. 2015;47(4):368–79.
7. La Vignera S, Condorelli RA, Balercia G, Vicari E, Calogero AE. Does alcohol have any effect on male reproductive function? A review of literature. Asian J Androl. 2013;15(2):221-5.
8. Li Y, Lin H, Li Y, Cao J. Association between socio-psycho-behavioral factors and male semen quality: systematic review and meta-analyses. Fertil Steril. 2011;95(1):116–23.
9. Muthusami KR, Chinnaswamy P. Effect of chronic alcoholism on male fertility hormones and semen quality. Fertil Steril. 2005;84(4):919–24.
10. Olayaki LA, Edeoja EO, Jimoh OR, Ghazal OK, Olawepo A, Jimoh AG, et al. Effects of cigarette smoking on urinary testosterone excretion in men. Biokemistri. 2008;20(1):29-32.
11. Colagar AH, Jorsaraee GA, Marzony ET. Cigarette smoking and the risk of male infertility. Pak J Biol Sci. 2007;10(21):3870–4.
12. Durairajanayagam D. Lifestyle causes of male infertility. Arab J Urol. 2018;16(1):10–20.
13. Asare-Anane H, Bannison SB, Ofori EK, Ateko RO, Bawah AT, Amanquah SD, et al. Tobacco smoking

is associated with decreased semen quality. Reprod Health. 2016;13(1):90.
14. Dai JB, Wang ZX, Qiao ZD. The hazardous effects of tobacco smoking on male fertility. Asian J Androl. 2015;17(6):954-60.
15. Beal MA, Yauk CL, Marchetti F. From sperm to offspring: assessing the heritable genetic consequences of paternal smoking and potential public health impacts. Mutat Res Rev Mutat Res. 2017;773:26–50.
16. Fronczak CM, Kim ED, Barqawi AB. The insults of illicit drug use on male fertility. J Androl. 2012;33(4):515–28.
17. Battista N, Pasquariello N, Di Tommaso M, Maccarrone M. Interplay between endocannabinoids, steroids and cytokines in the control of human reproduction. J Neuroendocrinol. 2008;20:82–9.
18. Gundersen TD, Jørgensen N, Andersson AM, Bang AK, Nordkap L, Skakkebæk NE, et al. Association between use of marijuana and male reproductive hormones and semen quality. Am J Epidemiol. 2015;182(6):473–81.
19. Bracken MB, Eskenazi B, Sachse K, McSharry JE, Hellenbrand K, Leo-Summers L. Association of cocaine use with sperm concentration, motility, and morphology. Fertil Steril. 1990;53(2):315–22.
20. De Souza GL, Hallak J. Anabolic steroids and male infertility: a comprehensive review. BJU Int. 2011;108(11):1860–5.
21. Catalano PM. Management of obesity in pregnancy. Obstet Gynecol. 2007;109(2):419–33.
22. Jensen TK, Andersson AM, Jørgensen N, Andersen AG, Carlsen E, Petersen JH, et al. Body mass index in relation to semen quality and reproductive hormones among Danish men. Fertil Steril. 2004;82(4):863–70.
23. Campbell JM, Lane M, Owens JA, Bakos HW. Paternal obesity negatively affects male fertility and assisted reproduction outcomes: a systematic review and meta-analysis. Reprod Biomed Online. 2015;31(5):593–604.
24. Hammoud AO, Wilde N, Gibson M, Parks A, Carrell DT, Meikle AW. Male obesity and alteration in sperm parameters. Fertil Steril. 2008;90(6):2222–5.
25. Martini AC, Tissera A, Estofán D, Molina RI, Mangeaud A, de Cuneo MF et al. Overweight and seminal quality: a study of 794 patients. Fertil Steril. 2010;94(5):1739–43.
26. Chavarro JE, Toth TL, Wright DL, Meeker JD, Hauser R. Body mass index in relation to semen quality, sperm DNA integrity, and serum reproductive hormone levels. Fertil Steril. 2010;93(7):2222–31.
27. Corona G, Mannucci E, Schulman C, Petrone L, Mansani R, Cilotti A, et al. Psychobiologic correlates of the metabolic syndrome and associated sexual dysfunction. Eur Urol. 2006;50(3):595–604.
28. Anderson K, Nisenblat V, Norman R. Lifestyle factors in people seeking infertility treatment – a review. Aust N Z J Obstet Gynaecol. 2010;50(1):8–20.
29. Gollenberg AL, Liu F, Brazil C, Drobnis EZ, Guzick D, Overstreet JW, et al. Semen quality in fertile men in relation to psychosocial stress. Fertil Steril. 2010;93(4):1104–11.
30. Stewart AF, Kim ED. Fertility concerns for the aging male. Urology. 2011;78(3):496–9.
31. Kimberly L, Case A. Advanced reproductive age and fertility. Int J Gynaecol Obstet. 2012;117(1):95–102.
32. Mutsaerts MAQ, Groen H, Huiting HG, Kuchenbecker WKH, Sauer PJJ, Land JA, et al. Influence of maternal and paternal factors on time to pregnancy. Hum Reprod. 2012;27(2):583–93.
33. Eskenazi B, Wyrobek AJ, Sloter E, Kidd SA, Moore L, Young S, et al. The association of age and semen quality in healthy men. Hum Reprod. 2003;18(2):447–54.
34. Gunes S, Hekim GNT, Arslan MA, Asci R. Effects of aging on the male reproductive system. J Assist Reprod Genet. 2016;33(4):441–54.
35. Wu Y, Kang X, Zheng H, Liu H, Liu J. Effect of paternal age on reproductive outcomes of IVF. PLoS One. 2015;10(9):e0135734.
36. Mendiola J, Torres-Cantero AM, Vioque J, Moreno-Grau JM, Ten J, Roca M, et al. A low intake of antioxidant nutrients is associated with poor semen quality. Fertil Steril. 2010;93(4):1128–33.
37. Salas-Huetos A, Bulló M, Salas-Salvadó J. Dietary patterns, foods and nutrients in male fertility parameters. Hum Reprod Update. 2017;23(4):371–89.
38. Lacasaña M, Vazquez-Grameix H, Borja-Aburto VH, Blanco-Muñoz J, Romieu I, Aguilar-Garduño C, et al. Maternal and paternal occupational exposure to agricultural work and anencephaly risk. Occup Environ Med. 2006;63(10):649–56.
39. Jurewicz J, Hanke W, Radwan M, Bonde J. Environmental factors and semen quality. Int J Occup Med Environ Health. 2009;22(4):305–29.
40. Figà-Talamanca I, Traina ME, Urbani E. Occupational exposures to metals, solvents and pesticides: evidence on male reproductive effects. Occup Med (Lond). 2001;51(3):174–88.
41. Tielemans E, Van Kooij R, Te Velde ER, Burdorf A, Heederik D. Pesticide exposure and decreased fertilisation rates in vitro. Lancet. 1999;354(9177):484–5.
42. Sidorkiewicz I, Zaręba K, Wołczyński S, Czerniecki J. Endocrine-disrupting chemicals: mechanisms of action on male reproductive system. Toxicol Ind Health. 2017;33(7):601–9.
43. Arbuckle TE, Sever LE. Pesticide exposures and fetal death: review of epidemiologic literature. Crit Rev Toxicol. 1998;28(3):229–70.
44. Bian Q, Xu LC, Wang SL, Xia YK, Tan LF, Chen JF, et al. Occupational fenvalerate exposure and

sperm DNA damage in pesticide workers. Occup Environ Med. 2004;61(12):999–1005.
45. Rehman S, Usman Z, Rehman S, Aldraihem M, Rehman N, Rehman I, et al. Endocrine disrupting chemicals and impact on male reproductive health. Transl Androl Urol. 2018;7(3):490-503.
46. Thonneau P, Bujan L, Multigner L, Mieusset R. Occupational heat exposure and male fertility. Hum Reprod. 1998;13(8):2122–5.
47. Durairajanayagam D, Sharma RK, du Plessis SS, Agarwal A. Testicular heat stress and sperm quality. In: Male Infertility. 2014:105–25.
48. Viganò P, Chiaffarino F, Bonzi V, Salonia A, Ricci E, Papaleo E, et al. Sleep disturbances and semen quality: an Italian study. Basic Clin Androl. 2017;27(1):16.
49. Sharma R, Biedenharn KR, Fedor JM, Agarwal A. Lifestyle factors and reproductive health. Reprod Biol Endocrinol. 2013;11(1):66.
50. Meirow D, Biederman H, Anderson RA, Wallace WHB. Toxicity of chemotherapy and radiation on female reproduction. Clin Obstet Gynecol. 2010;53(4):727–39.
51. Agarwal A, Deepinder F, Sharma RK, Ranga G, Li J. Effect of cell phone usage on semen analysis. Fertil Steril. 2008;89(1):124–8.
52. Agarwal A, Desai NR, Makker K, Varghese A, Mouradi R, Sabanegh E, et al. Effects of RF-EMW from cellular phones on ejaculated semen: in vitro pilot study. Fertil Steril. 2009;92(4):1318–25.
53. Adams JA, Galloway TS, Mondal D, Esteves SC, Mathews F. Effect of mobile phones on sperm quality: systematic review. Environ Int. 2014;70:106–12.
54. Kilgallon SJ, Simmons LW. Image content influences men's semen quality. Biol Lett. 2005;1(3):253–5.

Cite this article as: Markanday BK, Sur A, Gawri S. An overview on some important factors affecting male fertility. Int J Community Med Public Health 2026;13:464-70.