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REVIEW ARTICLE



The effects of radiofrequency radiation on male reproductive health and potential mechanisms

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ABSTRACT

Recent studies have demonstrated that radiofrequency (RF) radiation emanating from devices such as mobile phones and Wi-Fi may have adverse effects on male reproductive health. This radiation can elevate testicular temperature, potentially compromising sperm quality and DNA integrity, and influence the specific absorption rate (SAR) across different body regions, leading to detrimental reproductive outcomes. Furthermore, exposure to RF radiation has been linked to conditions that could affect male reproductive function, such as oxidative stress, alterations in ion transitions across cell membranes, and inflammation. The article reviews research conducted on both humans and animal models regarding the effects of electromagnetic radiation on sperm quality, DNA damage, oxidative stress, hormone levels, and testicular function, suggesting that exposure to electromagnetic radiation could have harmful implications for male reproductive health. However, further research is necessary to fully understand the mechanisms and implications of non-ionizing electromagnetic radiation on male infertility.

PLAIN-LANGUAGE SUMMARY

In this review, the effects of RF on male reproduction and the mechanisms related to them were examined and the results were discussed. “The effects of RF on sperm, reproductive hormones and testis, occupational exposure and male reproductive health, mechanisms of RF effects on reproductive system” were discussed here. In conclusion, further research is necessary to fully understand the mechanisms and implications of non-ionizing electromagnetic radiation on male infertility.

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KEYWORDS

Radiofrequency radiation exposure; male reproductive health; biological effects; mechanism

Introduction

Infertility is defined as the inability to conceive despite regular unprotected intercourse and can result from dysfunctions in either the male or female reproductive system. Male infertility is associated with abnormalities in sperm production, motility, or function and accounts for an estimated 20–40% of infertility cases worldwide. It is estimated that approximately 48 million couples and 186 million individuals globally are affected by infertility, underscoring its significance as a critical public health issue (Sharma and Shrivastava 2022). In an analysis conducted in 2023, it was reported that “between 1973 and 2018, a significant decline in sperm count has been observed among men from North America, Europe, and Australia, with a similar trend noted among unselected men from South/Central America, Africa, and Asia, and this decline has accelerated since 2000, now recognized as a significant public health issue” (Levine et al. 2023). As a result, many of

these couples turn to costly assisted reproduction treatments (Sharma and Shrivastava 2022; Thoma et al. 2013). Simultaneously, global communication technology, particularly mobile phones, has seen significant advancements, becoming an integral part of our daily lives and expected to continue growing. This surge in mobile phone use has led to an increase in the presence of radiofrequency (RF) electromagnetic radiation, raising concerns about potential health effects.

RF radiation, spanning between 100 kHz and 300 GHz, form the backbone of modern technologies such as Wi-Fi and mobile phones. While RF-EMF is non-ionizing and lacks the energy for ionization, recent research has highlighted its potential adverse health effects (Choi et al. 2020; Ishai et al. 2024; Kaur et al. 2023; Lin 2023; Schüz et al. 2022; Weller et al. 2023). In response to growing concerns, the World Health Organization (WHO) conducted an international survey among RF-EMF experts in 2018, identifying six key

health topics for prioritization: cancer, adverse reproductive outcomes, cognitive impairment, symptoms, oxidative stress, and heat-related effects (Verbeek et al. 2021). Among these, reproductive health, particularly infertility, emerged as a critical area of concern, emphasizing the need for continued investigation into the effects of RF-EMF exposure on the reproductive system.

Based on recent studies, male-related infertility is a global concern, with male factors contributing to 30–50% of infertility cases. While significant progress has been made in identifying specific causes, such as testicular dysfunction, endocrinopathies, and gonadotoxic exposures, a considerable percentage remains classified as unexplained infertility. These findings underscore the importance of further research to understand and address the complexities of male infertility (Eisenberg et al. 2023). Emerging evidence suggests that prolonged RF exposure might influence male fertility, including reduced sperm count, motility, and viability, by mechanisms such as oxidative stress and DNA damage (Cordelli et al. 2024; Kenny et al. 2024). Specifically, a systematic review and meta-analysis highlighted associations between RF exposure from mobile devices and declining sperm parameters, although findings remain inconsistent due to varying exposure conditions and methodological limitations (Kenny et al. 2024). Moreover, experimental studies in animal models have indicated that RF exposure may mitigate heat-induced testicular damage, suggesting its potential protective role in specific scenarios rather than exacerbating reproductive toxicity (Reza Mahmoudi et al. 2024). Collectively, these findings warrant further investigation into RF's impact on male reproductive health to refine safety guidelines and exposure thresholds. As the evidence suggests that RF radiation can have detrimental effects on reproductive health, understanding the way in which this radiation is absorbed by different tissues, particularly those associated with reproduction, is crucial.

The testicles are highly susceptible to RF radiation (Chu et al. 2023; Xu et al. 2008). Around 60 years ago, the first study on microwave radiation's effects on testicles was conducted (Prausnitz and Sutsskind 1962). Subsequent studies have consistently reported the harmful effects of RF radiation on testicles and spermatogonia (Agarwal et al. 2009, 2011; Avendano et al. 2012; Kaur et al. 2023; Kesari et al. 2018; Kirimlioglu et al. 2024; Sciorio et al. 2022; Shokri et al. 2015; Zahmatkesh et al. 2024). Cell phone usage has been associated with decreased sperm quality, and a relationship has been established between daily cell phone use duration and declining sperm parameters (Agarwal et al. 2008; Chu et al. 2023; Rahban et al. 2023).

The detrimental effects of RF radiation from wireless communication technologies on the reproductive system can have serious implications for human existence. Numerous studies consistently highlight the negative impacts of RF radiation on both male and female reproductive systems, leading to hormonal imbalances, DNA damage, and reduced fertility. However, the exact biological mechanisms by which RF radiation induces these effects remain partially understood, with oxidative stress, thermal effects, and direct DNA damage being the primary hypothesized pathways. The observed inconsistencies across studies underscore the need for more standardized research protocols and long-term studies to establish clearer causal relationships. Despite extensive research on various animal and human groups regarding RF radiation's effects on fertility, there remains a lack of comprehensive understanding regarding the precise mechanisms through which RF radiation influences the reproductive system.

Methods

In this study, we aimed to investigate the effects of RF radiation on male reproductive health by reviewing the available literature. We adopted the following methodology for the selection and evaluation of relevant studies to ensure a comprehensive and unbiased analysis:

Literature Search: We conducted a systematic search using leading academic databases such as Web of Science, Scopus, and PubMed. The search was conducted using keywords such as “radio frequency radiation,” “RF exposure,” “male reproductive health,” “sperm quality,” “DNA damage,” “oxidative stress,” and “testicular function.”

Inclusion Criteria: Studies were selected based on their relevance to the topic, with a focus on studies investigating the biological effects of RF radiation on male reproductive health. Both human and animal studies and *in vitro* studies were included to provide a comprehensive overview. Studies in peer-reviewed journals published predominantly in the last two decades were selected.

Exclusion Criteria: Articles that did not directly address the effects of RF radiation on male reproductive health or lacked sufficient methodological rigor were excluded. In addition, non-English language studies were also excluded to maintain consistency of analysis.

Assessment: A total of 90 articles were evaluated in this review. The results were carefully summarized and discussed, highlighting the effects of RF radiation on male reproductive health and areas for future research.

To further contextualize the findings, we also considered Specific Absorption Rate (SAR), a critical metric for evaluating RF radiation exposure and its biological effects.

Specific absorption rate (SAR)

SAR (Specific Absorption Rate) is a metric used to quantify the amount of electromagnetic energy absorbed by tissue exposed to an electromagnetic field. It is typically measured in watts per kilogram (W/kg) and represents the energy absorption normalized to body mass for a specific tissue volume. SAR values reflect the intensity of energy absorption within the body. SAR is crucial for assessing devices emitting RF radiation, such as mobile phones and wireless communication devices. International organizations establish SAR limits to regulate human exposure to radiofrequency radiation, ensuring device safety and minimizing potential health risks.

SAR values can vary based on the electromagnetic field's frequency, power level, and exposure duration. Higher SAR values indicate more intense energy absorption, potentially leading to biological effects. Therefore, SAR values must be considered in device design and usage to remain within established limits (ICNRP 2010; IEEE 1992; Teixeira and Hasan 2016). The amount of RF energy absorbed by living organisms depends on factors like body size, exposure duration, and device placement (Davis et al. 2023). Consequently, exposure standards differ for the head, torso, and extremities. Aligning a human's longitudinal axis parallel to the RF field vector results in peak SAR values. Larger adults tend to have lower resonant absorption frequencies, while shorter adults, children, infants, and seated individuals tend to have higher frequencies (Ahmad and Shaharun 2023). Keeping a phone close to the head during calls or storing it near the testicles or in the pants pocket results in higher energy absorption rates in those areas (Agarwal et al. 2011; Gombarska et al. 2023; Moazzen et al. 2024). Additionally, different tissues in the human body have varying electrical conductivity, leading to different RF energy absorption rates (Ahmad and Shaharun 2023; ICNRP 2010; Razek 2023).

Results

The effects of RF radiation on sperm

Research on the impact of non-ionizing microwave energy on testicles began half a century ago with a study on testicular degeneration in mice (Prausnitz and Sutsskind 1962). With the widespread use of RF emitting devices, extensive research has focused on RF exposure and its potential effects on male infertility, especially concerning testicles (Kesari et al. 2013, 2018; La Vignera et al. 2012; Yadav et al. 2021; Zha et al. 2019). Both human and animal studies consistently show that

testicular organs are highly susceptible to RF, with scenarios like keeping a mobile phone near the reproductive area during conversations affecting fertility parameters such as sperm motility, count, morphology, and apoptosis (Agarwal et al. 2009; Kesari et al. 2018).

Meta-analyses and systematic reviews of epidemiological studies on mobile phone radiation's impact on sperm quality in adult males have found significant reductions in sperm movement, viability, and quantity, correlating with the extent of mobile phone use (Adams et al. 2014; Dama and Bhat 2013; Kim et al. 2021; Yu et al. 2021). Several factors related to exposure circumstances can influence study outcomes, including energy penetration depth, exposure duration, transmission technology, device proximity to body tissues or testes, and SAR-detected energy density. For example, a study by Zilberlicht et al. (2015) noted increased instances of abnormal sperm concentrations when mobile phones were kept within 50 cm of the groin (Zilberlicht et al. 2015).

RF radiation has been shown to adversely affect sperm movement, viability, and morphology, leading to male infertility. However, study conclusions have been inconsistent due to variations in experimental setups, involving humans, animals, and in vitro studies (Yadav et al. 2021). Rats exposed to mobile phone frequencies for two hours daily for 35 days (SAR = 0.9 W/kg) exhibited reduced sperm counts and increased ROS levels (Kesari et al. 2010). Similarly, rats exposed to 2575–2635 MHz 4 G mobile phone radiation for six hours daily for 150 days experienced a decline in sperm quality and led to testicular morphologic injury (Yu et al. 2020).

In early investigation conducted by Cairnie and Harding (1981) involved exposing mice to elevated levels of microwave radiation for extended periods, though the applicability of their findings to humans is limited due to inter-species variations (Cairnie and Harding 1981). Agarwal et al. (2008) unearthed a possible detrimental effect of continuous mobile phone usage on sperm quality in human subjects. They scrutinized data from 361 males undergoing infertility assessments and discerned that individuals using mobile phones for more than four hours daily exhibited inferior sperm parameters compared to those with less than two hours of usage (Agarwal et al. 2008). Eroglu et al. (2006) delved into the impact of RF radiation on human sperm motility in an in vitro setting. Their investigation entailed exposing sperm samples from 27 healthy individuals to varying durations of RF radiation, which yielded a noteworthy reduction in sperm mobility (Eroglu et al. 2006). On a parallel note, Falzone et al. (2008) observed that "RF irradiation led to diminished sperm motility while leaving mitochondrial

membrane potential unaffected” (Falzone et al. 2008). Fejes et al. (2005) undertook a comprehensive examination of the correlation between mobile phone usage and human sperm characteristics in a cohort of 371 males. Their analysis divulged that extended phone ownership and daily usage correlated inversely with rapidly progressing sperm and positively with slowly moving sperm (Fejes et al. 2005). Gutschi et al. (2011) ventured into a study encompassing 2,110 male participants to scrutinize the effects of mobile phone usage on sperm parameters. The research illuminated that “mobile phone users exhibited a heightened proportion of sperm with anomalous shapes, elevated testosterone levels, and diminished luteinizing hormone levels” (Gutschi et al. 2011). Meanwhile, Mortazavi et al. (2013) embarked on an investigation pertaining to the influence of jammer RF radiation on sperm motility. The study unearthed a conspicuous decline in sperm motility when exposed to RF radiation from a jammer device for a duration of 2 to 4 hours, underscoring potential repercussions on fertility. Consequently, they recommended the formulation of regulations to curtail the usage of wireless jammers (Mortazavi et al. 2013).

In another investigation, Sajeda and Al-Watter (2011) meticulously examined 300 patients classified into groups based on mobile phone usage patterns and durations. The seminal parameters displayed significant variations across all user groups, with protracted daily usage manifesting as the harbinger of more pronounced reductions in semen quality, encompassing sperm count, motility, and morphology (Sajeda and Al-Watter 2011). Furthermore, Wdowiak et al. (2007) conducted an inquiry into the ramifications of mobile phone use on male reproductive health, particularly during infertility therapy. The findings unveiled a correlation between RF waves emanating from mobile phones and the attenuation of sperm forward mobility, coupled with anomalies in sperm shape (Wdowiak et al. 2007). Kamali et al. (2017) ventured into the exposure of sperm specimens to radiation emitted by 3 G and WiFi modems, culminating in a substantial reduction in sperm motility, notably among non-progressively motile spermatozoa (Kamali et al. 2017). A study by Falzone et al. (2011) scrutinized the influence of 900-MHz radiation from mobile phones on sperm characteristics, revealing significant decrements in sperm head region and acrosome proportion in exposed sperm, potentially impinging on the fertilization process (Falzone et al. 2011). Another study looked at how environmental factors, such as mobile phone towers, affect man reproduction capabilities. The study group consisted of 100 couples who were infertile, while the control group consisted of 100 healthy couples.

A questionnaire was used to measure both environmental exposure and employment status. The analysis of sperm was performed on subfertile men, and greater rates of aberrant semen analysis were discovered. Significant variations in the subfertile group were connected with occupational and environmental risks. According to the findings, avoiding such risk factors may enhance sperm quality in infertile men (Al-Quzwini et al. 2016). Ahmad and Baig (2011) delved into the protective potential of vitamin E against the deleterious effects of RF radiation on sperm quality. RF exposure induced reductions in sperm motility and heightened oxidative stress markers, though vitamin E mitigated these effects (Ahmad and Baig 2011). Meanwhile, Oni et al. (2011) scrutinized the ramifications of 2.4 GHz RF radiation on human sperm in an *in vitro* context, ultimately unveiling substantial reductions in sperm concentration, motility, and morphology (Oni et al. 2011).

Multiple research endeavors have explored the influence of RF radiation on the quality of sperm in animals. In one study, Wistar rats were exposed to RF radiation at 50 GHz for 2 hours daily over 45 days, resulting in a SAR of 8.0×10^{-4} W/kg. This exposure led to significantly lower sperm glutathione peroxidase (GPx) and superoxide dismutase (SOD) activity, higher catalase activity, increased apoptotic cells, reduced cell cycle stages, and lower histone kinase activity (Kesari and Behari 2010b). Another study examined the effects of RF radiation from mobile phones on sperm and testosterone levels in male rats. Twenty-five rats were exposed to various RF frequencies for 30 days. All groups experienced a notable loss in sperm viability, except the group exposed to missed calls (Asl et al. 2021). In an 18-week study, male Sprague-Dawley rats were exposed to mobile phone emissions twice daily for 3 hours each. It was reported that “using mobile phones near reproductive organs may negatively affect male reproduction by increasing sperm cell death and abnormal aggregation” (Yan et al. 2007). Furthermore, male Wistar rats exposed to RF radiation (2.45 GHz, 2 h/day for 35 days) exhibited adverse effects on testicular tissues, including apoptotic cell presence and altered antioxidant enzyme levels. There was a substantial decrease in GPx and SOD activities and total sperm count, with an elevation in CAT activity in the exposed group (Kesari and Behari 2010a). Another study investigated the impact of RF radiation (900 MHz, 2 hours/day for 50 days) on rat sperm cells, revealing increased dead sperm cells, ROS levels, and total antioxidant capacity. The radiation also altered gene expression related to cell death, inducing sperm death via signaling pathways like bcl-2, bax, cytochrome c, and caspase-3 (Liu et al.

2015). Long-term RF irradiation effects on spermatogenesis were explored in male rats across varied intensities and durations of electromagnetic fields exposure. Prolonged exposure led to reduced spermatogenesis, indicated by lower sperm, germ cell, and Leydig cell counts (Oh et al. 2018). On the other hand, Yu et al. (2023) demonstrated that “short-term RF exposure did not decrease sperm quality and spermatogenesis, but increased the levels of testosterone and zinc transporter 9 (ZIP9) in Sertoli cells, while with the increase in continuous exposure time, testicular ZIP9 gradually decreases and testicular MDA increases” (Yu et al. 2023). Another investigation revealed that “long-term RF exposure originating from 4 G cell phones to the scrotum resulted in testicular damage, disruption of the blood-testis barrier, and decreased semen quality” (Cheng et al. 2021).

A study examined the impact of RF radiation on free radicals and sperm characteristics in male albino Wistar rats. Rats were exposed to active GSM RF radiation (1 h/day for 28 days), while a control group received no radiation. The results showed a significant reduction in motile sperm, increased lipid peroxidation, and decreased glutathione (GSH) concentrations in the testes and epididymis of the exposed group. However, overall sperm count did not differ significantly between the two groups (Mailankot et al. 2009). In another study, the influence of mobile phone usage on reproduction in male Wistar rats was examined. Animals were exposed to a mobile phone frequency (900 MHz, GSM, 0.9 W/kg) for 2 hours per day for 35 days. The researchers observed a decrease in antioxidant enzymes and an increase in free radical generation, leading to disruptions in the sperm cell cycle and micronuclei formation (Kesari et al. 2011). A study assessed the long-term effects of RF radiation from mobile phones on testicular structure and function in male rabbits. Rabbits were exposed to RF radiation emitted by a mobile phone in standby mode for eight hours daily for 12 weeks. The results showed a significant reduction in sperm count and mobile sperm density, along with a decrease in the size of seminiferous tubules, crucial for sperm generation and transport (Salama et al. 2010). A recent similar study has demonstrated that “prolonged exposure to RF radiation leads to a decrease in sperm count and morphological changes and melatonin administration also reduces the negative outcomes” (Özgen et al. 2023).

Furthermore, an investigation examined “the impact of low power density 2.4 GHz Wi-Fi irradiation on the reproductive systems of infertile and normal mice”. Thirty mature Balb/c male mice were exposed to Wi-Fi radiation, resulting in boosted sperm levels in healthy mice and improved spermatid cells in infertile animals.

These findings suggest that low-energy Wi-Fi irradiation may positively affect sperm concentration and histomorphometric characteristics in mice (Delavarifar et al. 2020). A study conducted at Zhejiang University’s Department of Reproductive Endocrinology examined the impact of extensive daily mobile phone use on sperm motility in 1634 men. The findings revealed significant changes in various sperm movement parameters, indicating potential harm to male reproductive health. However, call duration did not show a clear impact on sperm movement, suggesting a complex relationship between mobile phone usage and fertility (Zhang et al. 2022). Cohort studies spanning from 2012 to 2020 in Denmark and North America, involving 3100 men, investigated the effects of mobile phone usage on fertility. Surveys collected data on phone usage and conception time, revealing little overall effect of storing phones in front pants pockets on fertility. However, a notable negative effect was observed in men with a BMI under 25, indicating that body weight might influence the impact of phone exposure on fertility. The study also found limited links between phone exposure and semen quality, recognizing potential exposure misclassification and confounding factors (Hatch et al. 2021). Experiments conducted on sperm samples obtained from healthy and fertile men aged 25–35 have observed a decrease in sperm motility and viability when smartphones utilize WiFi connection. On the other hand, it has been determined that “exposure to 4 G and 5 G RF does not have a significant effect on these outcomes” (Chu et al. 2023). Another study involving 1454 men aged 22–45 explored the correlation between cell phone use, RF radiation, and sperm quality. It found that longer use of electronic devices was associated with reduced sperm motility, and extensive cell phone and computer use correlated with lower sperm concentration and motility. Increased call duration also negatively impacted sperm concentration and count, particularly when using headphones during calls. However, no clear link was established between device usage and overall sperm quality (Chen et al. 2022).

These studies collectively suggest that the effects of mobile phone radiation on male fertility are multifaceted and may depend on various factors, including the amount and duration of exposure, as well as individual health conditions. While some investigations found little difference in sperm quality between exposed and non-exposed groups, others reported lower sperm parameters in men with prolonged mobile phone usage. Additionally, carrying phones in pant pockets and exposure to jammer RF radiation were associated with reduced sperm quality. Factors such as toxic

compounds, ionizing radiation, stress, and electromagnetic radiation have been implicated in male infertility, with mobile phone use identified as a potential contributing factor.

In conclusion, despite an incomplete understanding of the precise mechanisms underlying the impact of non-thermal RF radiation on sperm and testicular function, research across animal and human studies consistently suggests a connection between increased levels of ROS and/or DNA damage and negative impacts on fertility factors. This indicates that the negative effects of RF radiation on sperm parameters may primarily be linked to the induction of ROS. Furthermore, the variability in study outcomes underscores the complexity of RF radiation's effects, which may be influenced by factors such as exposure duration, frequency, and individual susceptibility. Therefore, limiting mobile phone use may be advisable to mitigate the incidence of male infertility. However, further research is warranted to explore the long-term effects of mobile phone radiation on male fertility and the generalizability of these findings (Table 1). Future studies should aim to clarify the specific biological pathways affected by RF radiation and standardize experimental protocols to resolve inconsistencies in the literature.

The effects of RF radiation on DNA

Low-intensity RF radiation possesses enough energy to induce DNA ionization, albeit indirectly. Numerous studies have consistently demonstrated that RF exposure elevates the production of ROS within living cells. Consequently, one indirect consequence of such energy is the provocation of oxidative damage to DNA. Among ROS, the hydroxyl radical, which can be generated by the superoxide radical and hydrogen peroxide, stands out as the most potent agent capable of inducing oxidative DNA damage. These specific ROS can be generated within cells following exposure to low-intensity RF radiation (ICBE-EMF, 2022).

Several studies have explored the impact of RF radiation on DNA damage and sperm characteristics. For instance, Akdag et al. (2016) found that “in their investigation on the long-term effects of 2.4 GHz frequency RF radiation from Wi-Fi devices on DNA damage in various tissues of rats, except for the testes, prolonged exposure to RF did not induce DNA damage in tissues such as the brain, kidney, liver, and skin, indicating a greater vulnerability of testicular tissue to RF exposure” (Akdag et al. 2016). Shahin et al. (2019) stated that “RF exposure disrupts sperm structure and hormone profiles in rats, leading to impaired steroidogenesis and spermatogenesis, and

additionally, application of spermine partially protects against RF-induced testicular and reproductive abnormalities through antioxidant, anti-inflammatory, and anti-apoptotic pathways” (Shahin et al. 2019). Similarly, examining the effects of RF exposure from mobile phones on sperm parameters, Zalata et al. (2015) found that “exposure to mobile phone radiation significantly reduced sperm movement, linear velocity, and acrosin activity, while also leading to increased sperm DNA disintegration and levels of seminal clusterin gene expression and protein, particularly in cases of oligoasthenoteratozoospermia” (Zalata et al. 2015). Atasoy et al. (2013) investigated the effects of RF radiation from residential Wi-Fi internet connection equipment on rat testes. They found that rats exposed to RF experienced DNA damage in their testes, along with decreased activity of catalase and glutathione peroxidase enzymes, indicating potential impacts on DNA structure and enzyme activity (Atasoy et al. 2013). A study carried out on 23 healthy donors from a reproductive clinic discovered that RF exposure reduced the movement of sperm and survivability and raised the amount of ROS, but had no effect on total antioxidant capacity or DNA harm (Agarwal et al. 2009). A further study on 29 healthy sperm donors discovered that Wi-Fi radiation exposure reduces the movement of sperm and raises sperm DNA breakage, both of which can lead to infertility. Placing a laptop on one's lap can also reduce sperm motility by raising the warmth of the scrotum (Avendano et al. 2012). Exposure to 905 MHz RF (SAR = 2.2 W/kg) for 12 hours a day for five weeks resulted in reduced sperm motility, increased ROS production, and DNA damage in mice (Houston et al. 2019). It was found that “RFR (900 MHz, 4–8 h/d, 35 days) had a significant negative impact on male germ cells and sperm morphology in Swiss albino mice”. These effects were attributed to RFR-induced oxidative stress, causing DNA damage, cell cycle disruptions, and a decrease in sperm count in mice (Pandey et al. 2017).

However, not all studies have shown significant effects of mobile phone radiation on sperm quality. For instance, Falzone et al. (2010) found no significant impact of cellular phone radiation on apoptosis-related properties in highly mobile spermatozoa (Falzone et al. 2010). Similarly, Hassanzadeh-Taheri et al. (2022) observed decreased sperm viability and motility, as well as increased dead sperm and fragmented DNA, but these effects were not consistently significant across all parameters (Hassanzadeh-Taheri et al. 2022).

Nevertheless, Ding et al. (2018) demonstrated that “prolonged exposure to electromagnetic radiation from

Table 1. The effects of RF radiation on sperm.

Reference	Species	Specific absorption rate (W/kg)	Frequency (MHz)	Duration of exposure	Main outcomes
Zilberlicht et al. (2015)	Human semen analysis (questionnaires on demographics and cell phone usage)	NA	NA	≥1 h/d	Abnormal semen concentration
Özgen et al. (2023)	Wistar albino rat	NA	2100 MHz	30 m/d for 90 consecutive days	Increased abnormal sperm morphology and decreased sperm count. Melatonin administration reversed these effects.
Erogul et al. (2006)	Human Semen sample	NA	900 MHz (GSM)	5 min	Decreased sperm motility. No effect on sperm concentration.
Agarwal et al. (2008)	Human Semen sample	NA	NA	<2 h/d 2–4 h/d >4 h/d	Decreased sperm motility, viability, and normal morphology as the duration of daily exposure to mobile phones increased.
Falzone et al. (2008)	Human Semen sample	2.0 and 5.7 W/kg	900 MHz (GSM)	1 h	No effect on mitochondrial membrane potential and sperm motility
Falzone et al. (2011)	Human Semen sample	2.0 W/kg	900 MHz (GSM)	1 h	No effect on sperm's propensity for the acrosome reaction. Decreased sperm head area and acrosome percentage of the head area and sperm binding to the hemizona.
Fejes et al. (2005)	Human Semen sample	NA	NA	<15 min/d >60 min/d	Decreased sperm motility.
Gutschi et al. (2011)	Human Semen sample	NA	NA	NA	Increased levels of pathological morphology in spermatozoa and serum testosterone, and decreased serum luteinizing hormone levels. No effects on follicle stimulating hormone and prolactin.
Kesari et al. (2010)	Wistar rat	NA	NA	2 h/d for 35 days	Decreased in protein kinase activity and total sperm count. Increased in apoptosis.
Kesari and Behari (2010a)	Wistar rat	0.11W/kg	2.45 GHz	2 h/d for 35 days	Increased apoptotic cells in testis. Decreased total sperm count, GPx, and SOD. Increased CAT.
Kesari and Behari (2010b)	Wistar rats	8.0×10^{-4} W/kg	50 GHz	2 h/d for 45 days.	Decreased sperm GPx, SOD, and histone kinase activity; increased catalase activity and the percentage of apoptosis; decreased percentage of G(2)/M transition phase of the cell cycle; and no significant differences in the G(0)/G(1) phase.
Mailankot et al. (2009)	Wistar rat	NA	0.9/1.8 GHz (GSM)	1 h/d for 28 days	No effect on sperm count. Decreased sperm motility. Increased lipid peroxidation and decreased GSH in the testis and epididymis.
Sajeda and Al-Watter (2011)	Human Semen sample	NA	NA	2 h/d, 3 h/d, 4 h/d	No effect on serum testosterone. Decreased sperm count, motility, and normal morphology.
Wdowiak et al. (2007)	Human Semen sample	NA	NA	Using mobile phones sporadically for 1–2 years. using mobile phones regularly for more than 2 years.	Decreased sperm motility. Increased abnormal morphology.
Asl et al. (2021)	Wistar rat	0.1 W/kg	900 MHz (GSM)	1 h/d, 2 h/d, 50 missed calls/d for 30 days.	No effect on serum testosterone. Decreased sperm count and morphology.
Kamali et al. (2017)	Human Semen sample	1.3 W/kg	2400–2446.5 MHz	NA	No effect on sperm count and morphology. Decreased sperm motility and velocity.
Yan et al. (2007)	Sprague Dawley rat	1.80 W/kg	1.9 GHz (CDMA)	3 h +3 h a day for 18 weeks	No effect on structural sperm mutations, sperm counts. Decreased sperm motility and morphology.
Ahmad and Baig (2011)	Human Semen sample	NA	900 MHz (GSM)	1 h	Increased spermatozoal MDA, non-motile sperms percentages.
Oni et al. (2011)	Human Semen sample	NA	2.4 GHz	1 h	No effect on sperm concentration and morphology grading. Decreased sperm motility
Delavarifar et al. (2020)	Balb/c mice	30 mW/kg and 92 mW/kg	2.4 GHz	2 h/d for 4 days	No effect on cellular diameter, number of tubules, numerical density and cellular area. Increased sperm concentration in epididymis and spermatids proliferation.
Liu et al. (2015)	Sprague Dawley rat	0.66 ± 0.01 W/kg	900 MHz	2 h/d for 50 days	Increased ROS and decreased TAC in the rat sperm. Decreased the protein and mRNA expression of bcl-2 and increased that of bax, cytochrome c, and caspase-3.

(Continued)

Table 1. (Continued).

Reference	Species	Specific absorption rate (W/kg)	Frequency (MHz)	Duration of exposure	Main outcomes
Salama et al. (2010)	Rabbit	0.43 W/kg	800 MHz	8 h/d for 12 weeks.	Decreased sperm concentration and motile sperm population. Decreased diameter of seminiferous tubules.
Mortazavi et al. (2013)	Human Semen sample	NA	800 MHz to 2.4 GHz	2 h and 4 h	Decreased sperm motility.
Oh et al. (2018)	Sprague Dawley rat	3.0 W/kg	2.104 GHz continuously	6 h/d and 18 h/d	Decreased germ cell counts and Leydig cell count.
Cairnie and Harding (1981)	Mice	20 W/kg	2450 MHz	16 h/d for 30 days	No effect on sperm count and morphology.
Al-Quzwini et al. (2016)	Standard questionnaire and Human Semen sample	NA	NA	NA	Increased oligozoospermia and teratozoospermia.
Hassanzadeh-Taheri et al. (2022)	Human Semen sample	NA	NA	1 h	Increased apoptotic sperms and DNA fragmentation. Decreased sperm viability and motility. No effect on sperm morphology, chromatin abnormality, and maturity.
Chen et al. (2022)	Human semen sample	NA	4 G network	6.7 h/d, 4.0 h/d, 2.1 h/d, 18.1 h/d, and 20.5 min/d for the past three months	Reduction effect on semen quality, lower sperm concentration and total count.
Chu et al. (2023)	Human	NA	4 G/5 G, Wi-Fi	6 h	4 G/5 G RF emitted by a contemporary cellphone did not have negative effects on sperm motility and viability. WiFi exposure did have negative effects.
Yu et al. (2020)	Sprague-Dawley rat	1.05 W/kg	2575–2635 MHz (TD-LTE)	6 h, for 50, 100 and 150 days	Decreased sperm concentration, motility, viability, and normal morphology. Increased disorder in spermatogenesis, germ cell loss. Lower epithelium height, lower Johnsen score, and higher Cosentino score. Suppressed formation of MMP14–Spock3 complex, the formation of MMP14–P-MMP2 complex, and inhibited MMP2 activity.
Yu et al. (2023)	Sprague-Dawley rat	1.05 W/Kg	2605 MHz		No differences on sperm quality and spermatogenesis. Increased testicular testosterone and zinc transporter 9 (ZIP9), apoptosis rate and MDA in Sertoli cells.
Cheng et al. (2021)	Sprague-Dawley rat	NA	NA	6 h/d for 150 days	Disorder of the blood-testis barrier. Decreased sperm concentration, viability, motility, the percentage of morphologically normal sperm. Increased Coentino's score and MDA level, reduced levels of SOD, GSH and CAT, and down-regulated expressions of occludin, ZO-1, CAR and N-Cadherin.
Zhang et al. (2022)	Human semen analysis	NA	NA	NA	Decreased in sperm motility.
Hatch et al. (2021)	Human semen analysis	NA	NA	NA	No differences on semen volume, sperm concentration, totalsperm count, motility, and total motile count.

NA, not mentioned or conducted in study.

4 G and Wi-Fi networks increased ROS and decreased antioxidants, leading to DNA damage in sperm and reduced sperm quality” (Ding et al. 2018). This underscores the potential deleterious impact of electromagnetic radiation on male fertility through oxidative stress. Overall, these findings highlight the importance of further research to better understand the long-term effects of RF radiation on male fertility and the potential mitigation strategies to minimize adverse impacts. Jamaludin et al. (2017) found that “even low levels of radiation from mobile phones can reduce sperm concentration and viability while increasing DNA damage,

likely through the NOX5 pathway” (Jamaludin et al. 2017). De Iuliis et al. (2009) exposed human spermatozoa to RF radiation and discovered reductions in mobility and viability, along with increased DNA fragmentation, correlating strongly with SAR levels (De Iuliis et al. 2009).

Gorpinchenko et al. (2014) found a decrease in sperm motility and an increase in DNA damage in healthy men exposed to mobile phone radiation for five hours (Gorpinchenko et al. 2014). Aitken et al. (2005) investigated 900 MHz RF radiation's impact on DNA damage in mouse sperm, observing significant deterioration in

mitochondrial and nuclear genome integrity (Robert John Aitken et al. 2005). Mahmoudi et al. (2015) explored “the effects of short-term exposure to 2.4 GHz RF radiation on male rat sperm, finding significant differences in sperm motility and number but no impact on testicular weight or DNA fragmentation” (Mahmoudi et al. 2015). Moreover, it was determined that “oxidative stress intensity and DNA fragmentation decrease in rat testes exposed to RF due to the potent antioxidant effects of melatonin” (Sokolovic et al. 2015).

In conclusion, these studies highlight the harmful effects of RF radiation on male reproductive health, including DNA damage, decreased sperm mobility and vitality, increased DNA fragmentation, and oxidative stress. The variability in the findings, driven by differences in study design, exposure parameters, and biological models, suggests that the biological impact of RF radiation may be multifactorial, involving complex interactions between ROS production, DNA repair mechanisms, and cellular apoptosis pathways. It is advisable to limit RF exposure by keeping mobile phones away from the pelvic region and minimizing the use of RF-emitting electronic devices. However, given the inconsistent outcomes across various studies, future research should focus on standardizing experimental conditions and exploring the dose-response relationship to better understand the threshold levels at which RF radiation becomes detrimental to reproductive health. Further research is necessary to fully understand the long-term implications of RF radiation on human reproductive health (Table 2).

The effects of RF radiation on reproductive hormones and testis

Several studies have explored the impact of RF radiation on male hormone levels and testicular morphology. Andrašková et al. (2022) found impaired testicular development in male pups exposed to RF radiation in utero, suggesting potential adverse effects on offspring (Andrašková et al. 2022). Another recent study has demonstrated that there is a significant decline in sperm quality following RF exposure, accompanied by changes in the secretion levels of Sertoli cells (SCs) and Leydig cells (LCs), as well as alterations in testis morphology. Furthermore, an increase in apoptosis within testicular cells was observed, suggesting a potential adverse effect of RF exposure on sperm quality in rats, possibly due to disruption in LCs secretion function and induction of apoptosis within testicular cells (Guo et al. 2019). Additionally, it was found that “blood testosterone levels decrease due to oxidative stress induced by RF and its impact on Leydig cells” (Lin et al. 2017). Dasdag et al. (2015) observed changes in reproductive

characteristics in male rats exposed to Wi-Fi RF radiation, including alterations in testicular weight and morphology, recommending caution regarding prolonged RF exposure (Suleyman Dasdag et al. 2015). Moreover, Hanci et al. (2018) demonstrated that “exposure to 900 MHz RF during adolescence leads to changes in rat testis morphology and oxidative stress biomarkers” (Hanci et al. 2018). Ozguner et al. (2005) investigated RF radiation’s effects on rat reproductive organs, noting hormonal changes but no impact on sperm production (Ozguner et al. 2005). Almasıová et al. (2014) observed negative effects on the seminiferous epithelium in rats exposed to RF (Almasıová et al. 2014). Almasıová et al. (2018) found “increased testicular temperature and histological changes in rats exposed to microwave radiation, indicating tissue oxidative injury” (Almasıová et al. 2018). Djeridane et al. (2008) studied RF’s impact on hormone levels in men, finding no changes in most hormones but decreases in growth hormone and cortisol during exposure periods (Djeridane et al. 2008). Jonwal et al. (2018) exposed mice to RF, resulting in oxidative stress in the testes and histological alterations (Jonwal et al. 2018). Shahin et al. (2014) found decreased sperm count and viability, as well as hormonal changes, in mice exposed to low-energy RF radiation (Shahin et al. 2014). In their study, Qin et al. (2021) examined “the extent of RF-induced toxicity in the testes and its impact on the expression of lncRNAs, noting disparities between data obtained from morning and evening exposure, and highlighting the crucial roles of differentially expressed lncRNAs associated with RF exposure in the damage inflicted on developing pubertal testes” (Qin et al. 2021). These findings highlight the potential risks of RF radiation on male reproductive health, including effects on hormone levels, testicular morphology, and sperm parameters. Further research is needed to fully understand the mechanisms and long-term implications of RF exposure on male fertility.

In their 2016 study, Saygin et al. aimed to counteract RF radiation effects on rat testes using gallic acid (GA). The RF-exposed group showed increased levels of Malondialdehyde and total oxidant status (TOS), along with decreased testosterone and vascular endothelial growth factor (VEGF) levels. Conversely, the GA-treated group exhibited reduced TOS and oxidative stress index levels, increased total antioxidant status activities, and elevated testosterone and VEGF levels. Additionally, inflammation markers decreased, sperm count increased, and mitotic activity improved in the GA-treated group compared to the RF-exposed group (Saygin et al. 2016). Saygin et al. (2011) investigated the impact of 2.45 GHz RF radiation on rat testicular tissue in a separate study. The RF-exposed group showed

Table 2. The effects of RF radiation on DNA.

Reference	Species	Specific absorption rate (W/kg)	Frequency (MHz)	Duration of exposure	Main outcomes
Akdag et al. (2016)	Wistar Albino rat	141.4 μ W/kg and 7127 μ W/kg	2.4 GHz	24 h/d for 12 months	Increased DNA damage in testes
Zalata et al. (2015)	Human Semen sample	1.46 W/kg	850 MHz	1 h	Increased sperm DNA fragmentation percent, seminal clusterin (CLU) gene expression and CLU protein.
Atasoy et al. (2013)	Wistar albino rat	0.091 W/kg	2.437 GHz	24 h/d for 20 weeks	Decreased catalase and glutathione peroxidase. Increased DNA damage in testes.
Agarwal et al. (2009)	Human Semen sample	1.46 W/kg	850 MHz	1 h	Decreased sperm motility and viability, increased ROS, and decreased in ROS- total antioxidant capacity (TAC) score. No effect on TAC and DNA damage.
Avendano et al. (2012)	Human Semen sample	NA	2.4 GHz	4 h	Increased DNA fragmentation. Decreased sperm motility.
Falzone et al. (2010)	Human Semen sample	2.0 and 5.7 W/kg	900 MHz (GSM)	1 h	No effect on caspase 3 activity, externalization of phosphatidylserine, DNA strand breaks and generation of reactive oxygen species.
Hassanzadeh-Taheri et al. (2022)	Human Semen sample	NA	NA	1 h	Increased apoptotic sperms and DNA fragmentation. Decreased sperm viability and motility. No effect on sperm morphology, chromatin abnormality, and maturity.
Ding et al. (2018)	Human Semen sample	$3.19 \pm 0.07 \times 10^{-7}$ W/kg	1800-MHz and 2.45 GHz	<30 min 31–120 min >121 min.	Increased reactive oxygen, 8-OHdG and DNA fragments. Decreased sperm quality, count, vitality, motility and antioxidant.
Jamaludin et al. (2017)	Sprague Dawley rat	NA	4200 MHz and 9700 MHz	2, 4 and 6 h/d for two weeks.	Increased DNA damage and NOX5. Decreased sperm concentration, viability.
De Iuliis et al. (2009)	Human Semen sample	0.4 W/kg to 27.5 W/kg	1.8 GHz	16 h	Increased DNA fragmentation, 8-OH-dG and ROS. Decreased sperm vitality and motility.
Gorpinchenko et al. (2014)	Human Semen sample	NA	900/1800 MHz (GSM)	Every 10 m for 5 h	No effect on sperm vitality, concentration, and morphology. Decreased progressive movement and increased non – progressive movement. Increased DNA fragmentation.
Aitken et al. (2005)	CD1 Swiss mice	90 mW/kg	900 MHz	12 h/d for 7 days	No effect on sperm number, morphology, vitality and DNA strand breakage. Increased mitochondrial genome and the nuclear β -globin locus damage.
Mahmoudi et al. (2015)	Wistar rat	0.091 W/kg	2.4 GHz	2 h/d for 7 days	No effect on testes weight, sperm DNA fragmentation, and frequency of normal morphology. Decreased sperm count and motility.
Sokolovic et al. (2015)	Wistar rat	0.043–0.135 W/kg	900 MHz (GSM)	4 h/d for 20, 40 and 60 days	Increased MDA, protein carbonyl, xanthine oxidase, alkaline and acid DNase, decreased catalase.
Shahin et al. (2019)	Wistar rat	1.075 W/Kg	900 MHz	2 h/d for 8 weeks	DNA fragmentation, increased lipid peroxidation, FSH and LH, triggered nuclear factor-kappa B p65, inducible nitric oxide synthase, decreased catalase, glutathione peroxidase, and testosterone. Changes in testicular histology and histomorphometry.
Houston et al. (2019)	C57BL/6 mice	2 W/kg	905 MHz	12 h/d for 1, 3 or 5 weeks	No histological change, adversely impacted the vitality and motility profiles of mature epididymal spermatozoa. Increased ROS levels, elevated DNA oxidation and fragmentation.
Pandey et al. (2017)	Swiss albino mice	0.0516 and 0.0054 W/kg	900 MHz	4 h/d and 8 h/d for 35 days	Increased damage index in germ cells and sperm head defects. Decreased epididymal sperm count, tissue index for the testis and epididymis, percentage of epithelium area, hypospermatogenesis, maturation arrest, appearance of multinucleated giant cells, and disorganization of seminiferous tubules. Increased population of cells with depolarized mitochondrial membranes and DNA damage in germ cells.

NA, not mentioned or conducted in study.

lower Johnsen biopsy scores and Leydig cell counts, along with alterations in apoptosis-related genes. This suggests that RF exposure leads to spermatogenesis difficulties and apoptosis in testis tissue (Saygin et al. 2011). Shokri et al. (2015) exposed Wistar rat testes to 2.45 GHz radiation for varying durations, resulting in decreased sperm parameters, increased dead cell count, and elevated caspase-3 activity. Seminal vesicle weight was also significantly reduced, raising concerns about

prolonged Wi-Fi exposure (Shokri et al. 2015). Similarly, Šimaiová et al. (2018) subjected juvenile Wistar albino rats to 2.45 GHz radiation, leading to morphological changes in seminiferous tubules and increased apoptosis (Šimaiová et al. 2018). Additionally, mice exposed to 1800 MHz GSM RF radiation showed elevated serum testosterone levels without significant alterations in reproductive organs (Forgacs et al. 2006).

Furthermore, adult male rats exposed to 200 MHz RF radiation exhibited reduced fertility due to disruptions in spermatogenesis, without significant changes in blood testosterone concentrations (Khillare and Behari 1998). Bilgici et al. (2018) found that “RF radiation increased inflammation and testicular damage, evidenced by differences in blood levels of IL-6 and CRP, and abnormalities in testicular tissue histology, although no significant differences in IL-6, IL-10, and IL-32 concentrations were observed between control and experimental groups” (Bilgici et al. 2018). Dasdag et al. (1999) conducted a study exposing male rats to microwave radiation from mobile phones for two hours daily over a month. Despite no significant changes in epididymal sperm count or abnormal forms, rats exposed during speech mode exhibited histological testes changes and elevated rectal temperatures compared to the standby group (Dasdag et al. 1999). Dasdag et al. (2003) investigated “the adverse effects of RF radiation from mobile phones on various parameters of rat testes, revealing no significant changes in lipid composition, malondialdehyde level, p53 immunological response, sperm quantity, shape, testicular histology, or rectal temperature” (Suleyman Dasdag et al. 2003). Similarly, Ribeiro et al. (2007) found no noticeable differences in testicular function between rats exposed to RF radiation for one hour daily over 11 weeks and the control group (Ribeiro et al. 2007). Gautam et al. (2019) investigated “the effects of 3 G mobile phone radiation on male Wistar rat fertility. They observed a decline in spermatogenic cells, changes in sperm membrane composition, increased ROS and lipid peroxidation, and alterations in sperm count and tail shape” (Gautam et al. 2019). Jonwal et al. (2018) exposed healthy male Swiss albino mice to RF radiation for thirty days, leading to heightened oxidative stress markers and histopathological changes in seminiferous tubules, indicating negative impacts on male fertility (Jonwal et al. 2018). Oksay et al. (2014) reported that “Melatonin has a protective effect against testicular injury caused by 2.45 GHz RF in rats, preventing lipid peroxidation and strengthening antioxidant defenses” (Oksay et al. 2014). Shahin, Singh, and Chaturvedi (2018b) found that “mobile phone radiation disrupts testicular redox balance, raising ROS, NO, and MDA levels while lowering antioxidant enzymes. This imbalance leads to increased testicular cell apoptosis by affecting apoptotic proteins”. Similarly, it was indicated that “exposure to 2.45 GHz RF radiation disrupts testicular redox balance, leading to increased apoptosis via the p53-dependent Bax-caspase-3 pathway, with the severity of apoptosis also influenced by exposure duration” (Shahin, Singh, and Chaturvedi 2018a).

Al-Damegh (2012) and Kumar et al. (2011a) studied the impact of 10-GHz microwave radiation on male rat fertility, finding significant changes in ROS levels, histone kinase activity, cell death, and cell cycle stage, leading to male sterility (Al-Damegh 2012; Kumar et al. 2011a). However, in another study, high-frequency RF radiation did not result in testicular structure and function of rats (Xue et al. 2022). Similarly, in the study by Imai et al. (2011), no signs of testicular toxicity were observed (Imai et al. 2011). Kumar et al. (2011b) examined the effects of 2.4 GHz RF exposure on rat reproductive systems. They observed increased levels of caspase and creatine kinase, decreased testosterone and melatonin levels, suggesting negative impacts on male fertility, although RF treatment could counteract oxidative stress and potentially restore reproductive function (Kumar et al. 2011b). In another recent similar study, it was observed that “RF induces oxidative stress leading to testicular damage, and melatonin administration reduces these adverse effects” (Shokri et al. 2020). The study by Zeleke et al. (2022) sheds light on the correlation between mobile phone use, RF exposure, and male risk perception. It reveals that carrying devices close to the body during phone use increases men’s perceived risk of RF exposure, especially when phones are kept near reproductive organs. This underscores the potentially heightened impact of such practices on male reproductive health (Zeleke et al. 2022).

In conclusion, the research discussed in this section indicates that RF radiation exposure could negatively affect male reproductive health, leading to changes in hormone levels, testicular morphology, and sperm count and viability (Table 3). Prolonged exposure to wireless internet and mobile phones has been associated with alterations such as increased abnormalities in sperm heads and reduced weight of reproductive organs. Additionally, RF radiation can impact testicular parenchyma in rats, resulting in irregularly shaped seminiferous tubules with epithelial cell abnormalities. The complexity of these findings suggests that RF radiation may induce a cascade of biological responses, including oxidative stress, hormonal imbalances, and apoptotic processes, which collectively contribute to testicular damage and compromised reproductive health. The observed effects appear to be dose-dependent, with longer durations and higher intensities of RF exposure correlating with more severe reproductive outcomes. These findings emphasize the importance of avoiding long-term exposure to RF radiation emissions, particularly from wireless technologies, to prevent potential harm to male reproductive health. Future research should focus on elucidating the precise mechanisms by which RF radiation affects the reproductive system and identifying potential protective strategies to mitigate these effects.

Table 3. The effects of RF radiation on reproductive hormones and testis.

Reference	Species	Specific absorption rate (W/kg)	Frequency (MHz)	Duration of exposure	Main outcomes
Mahmoudi et al. (2024)	Wistar albino rats	0.091 W/kg	2450 MHz	2 h/d for 52 days	RF mitigated heat-induced sperm damage, improved testicular structure, sperm parameters, and Leydig cell count.
Yardim et al. (2024)	Wistar rats	0.297 W/kg	2600 MHz	30 min/d, 5 d/week for 4 weeks	Increased oxidative stress (elevated MDA and NOx levels), decreased antioxidant levels (GSH, GSH-Px), testicular edema, and reduced connective tissue. Melatonin mitigated these effects by restoring antioxidant levels and reducing structural damage.
Ok et al. (2024)	Wistar albino rat	0.14 W/kg	900 MHz	4 h/d for 8 weeks	Increased testicular damage, seminiferous tubule degeneration, interstitial edema, and tunica albuginea thinning. Elevated IL-4 and IFN- γ expression levels, potentially linked to germ cell tumor initiation.
Gupta and Srivastava (2022)	Immature male chickens (Gallus gallus domesticus)	0.9978 W/kg	2450 MHz	2 hours/day for 30 days	Significant decrease in testicular weight, volume, and gonadosomatic index (GSI); histological changes in seminiferous tubules (elongation, distortion, decreased diameter); increased oxidative stress markers (H ₂ O ₂ and malondialdehyde); no significant change in antioxidant enzyme activities (SOD, catalase, GSH); downregulation of estrogen receptor alpha (ER- α); increased pro-inflammatory cytokine IL-1 β and decreased anti-inflammatory cytokine IL-10.
Guo et al. (2019)	Sprague – Dawley rats	0.030 W/kg and 0.014 W/kg	220 MHz (Puls modulated)	1 h/d for one-month	Decreased sperm quality, changes in secretion levels of factors released from Sertoli and Leydig cells, abnormal testis morphology, and increased apoptosis in testicular cells
Shokri et al. (2020)	BALB/c mice	1.3 watts/kg	900 MHz	4 h/d for 30 days	Decreased testosterone, increased germinal epithelial thickness and seminiferous tubule diameter, increased testicular damage.
Xue et al. (2022)	Sprague – Dawley rats	3.36 W/kg	5.8 GHz	1 h/d for 30 days	No significant differences in sperm count, sperm abnormality, and levels of T, FSH, LH, GDNF, SCF, and caspase-3.
Shahin et al. (2018)	Swiss albino mice	0.05 W/Kg	1800 MHz	3 h/d for 120 days	Decreased seminiferous tubule diameter, sperm count, and viability. Increased germ cells apoptosis and decreased serum testosterone
Shahin et al. (2018)	Swiss albino mice	0.0146 W/kg	2.45 GHz (CW)	2 h/d for 15, 30 and 60 days	Increased ROS, NO, MDA, p53, Bax, decreased SOD, Bcl-xL, Bcl-2, inactive executioner caspase-3, and DNA repairing enzyme (uncleaved PARP-1), testosterone, and seminiferous tubule diameter.
Lin et al. (2017)	Mouse Leydig (TM3) cells	3 W/kg	1950 MHz	24 h	Decreased cell proliferation, cell cycle distribution, Testosterone, and P450scc mRNA level. There is no change in cell apoptosis, ROS, and STAR mRNA level.
Hanci et al. (2018)	Sprague Dawley rat	0.0067 W/kg	900 MHz	1 h/d, for 21 days	No difference in GSH. Increased MDA, SOD, and CAT, decreased testis weight, seminiferous tubule degeneration, loss of integrity of the basal lamina, endoplasmic reticulum dilation, vacuolated mitochondria.
Ozguner et al. (2005)	Sprague-Dawley rat	NA	900 MHz (CW)	30 min/d, 5 d/week for 4 weeks	Decreased serum testosterone, plasma luteinizing hormone and follicle stimulating hormone, diameter of the seminiferous tubules and the mean height of the germinal epithelium. No effect on testis weight, testicular biopsy score count and the percentage of interstitial tissue to the entire testicular tissue.
Almášiová et al. (2014)	Wistar rat	NA	2.45 GHz	3 h/d for 3 weeks	No effect on interstitial Leydig cells. Increased in degenerative changes in seminiferous epithelium. Increased irregular shaped seminiferous tubules. Increased in the number of necrotizing and shriveled spermatogonia.
Almášiová et al. (2018)	Wistar rat	NA	2.45 GHz	3 h/d for 3 weeks	Increased dilated and congested blood vessels and degenerative changes on seminiferous epithelium. No effect on Leydig cells. Increased ultrastructural changes on developing sex cells, Sertoli cells, and endothelial cells. Increased immunoreactivity to superoxide dismutase 1 in spermatogonia and Leydig cells.
Djeridane et al. (2008)	Human blood samples	0.3 W/kg	900 MHz GSM	2 h/d, 5 days/week, for 4 weeks	No effect on prolactin, thyroid-stimulating hormone, adrenocorticotropin, and testosterone circadian pattern. Decreased growth hormone and cortisol
Jonwal et al. (2018)	Swiss albino mice	0.09 W/kg	2.45 GHz	2 h/d for 30 days.	Increased catalase, malondialdehyde, and ROS. Decreased glutathione peroxidase, testosterone, and superoxide dismutase. Increased histopathological changes in seminiferous tubules. Increased micronuclei in blood.
Shahin et al. (2014)	Swiss strain mice	0.018 W/Kg	2.45 GHz	2 h/d for 30 days	Increased plasma testosterone and testicular i-NOS expression. Decreased sperm count and viability, seminiferous tubule diameter, and testicular 3 β HSD activity.

(Continued)

Table 3. (Continued).

Reference	Species	Specific absorption rate (W/kg)	Frequency (MHz)	Duration of exposure	Main outcomes
Imai et al. (2011)	Sprague-Dawley rat	0.4 and 0.08 W/kg	1.95 GHz (W-CDMA)	5 h/d, for 5 weeks	No differences in body weight gain or weights of the testis, epididymis, seminal vesicles, prostate. Increased testicular sperm count. No differences in sperm motility or morphology and the histological appearance of seminiferous tubules, including the stage of the spermatogenic cycle.
Qin et al. (2021)	Mause (C57BL/6)	0.50 W/kg	1800 MHz	2 h/d, for 21 days	Induced expressed lncRNAs.
Saygin et al. (2016)	Sprague-Dawley rat	3.21 W/kg	2.45 GHz	3 h/day for 30 day	Increased MDA, TOS, PGE2, CGRP and mitotic activity. Decreased Sperm count, testosterone and VEGF.
Saygin et al. (2011)	Wistar Albino rat	3.21 W/kg	2.45 GHz	60 min/d for 28 days	No effect on TNF- α , Caspase-3, and Bcl-2. Increased Bax gene and Caspase-8 enzyme.
Shokri et al. (2015)	Wistar rat	NA	2.45 GHz	1 h, 7 h/day for 2 months	Increased apoptosis-positive cell and caspase-3 activity. Decreased sperm parameters and seminal vesicle weight.
Šimaiová et al. (2018)	Wistar albino rat	NA	2.45 GHz	2 h/day for 3 weeks	Increased damage on seminiferous tubules shape, sertoli cells and spermatocytes. Increased seminiferous tubules total area, apoptotic nuclei and Cu-Zn-SOD and Mn-SOD positive cells.
Forgacs et al. (2006)	Mice	0.018–0.023 W/kg	1800 MHz (GSM)	2 h/day for 10 days	Increased serum testosterone, red blood cell counts. No effect on Leydig cells, reproductive organs, serum chemical parameters and body weight.
Khillare and Behari (1998)	Wistar rat	2.0–1.65 W/kg	200 MHz	2 h/day for 35 days	Increased on leydig cells, spermatids, and acrosomes abnormalities. No effect on serum testosterone.
Bilgici et al. (2018)	Wistar rat	0.0233 W/kg	2.45 GHz	1 hour/day for 30 days	Increased serum IL-6, CRP and testicular damage. No effect on IL-6, IL-10, IL-32 in testis.
Dasdag et al. (1999)	Wistar Albino rat	0.141 W/kg	890 $\pm$ 915 MHz (GSM)	1 hour/day for 30 days	No effect on epididymal sperm counts and sperm form. Decreased seminiferous tubular diameter. Increased histological changes in testes and rectal temperature.
Dasdag et al. (2003)	Sprague-Dawley rat	0.52 W/kg (whole body) 3.13 W/kg (1 g averaged peak)	890 $\pm$ 915 MHz (GSM)	20 minutes/d for 30 days	No effect on lipid composition, malondialdehyde concentration, p53 immune reactivity, sperm count, morphology, histological structure of testes, and rectal temperature.
Ribeiro et al. (2007)	Wistar rat	NA	1,835 to 1,850 MHz	1 h/d for 11 weeks	No effect on testicular and epididymal weight, lipid peroxidation, serum testosterone, the epididymal sperm count, testicular histology, seminiferous tubular diameter, pachytene spermatocytes, sertoli's cells and round spermatid.
Gautam et al. (2019)	Wistar rat	0.26 W/kg	1915 MHz (UMTS)	2 h/d for 45 days	Decreased on testis weight, sperm viability, sperm count percentage of tail-coiled spermatozoa and number of spermatogenic cells. Increased ROS, MDA. No effect on sperm head morphology. Seminiferous tubules size alteration, sperm tail morphology and germinal layer disruption.
Oksay et al. (2014)	Wistar Albino rat	0.143 W/kg	2.45 GHz	1 h/d for 30 days	No effect on testicular weight, histology, GSH and GSH-Px. increase in lipid peroxidation
Al-Damegh (2012)	Wister albino rat	0.9 W/kg	900, 1800, 1900 MHz (GSM)	15, 30, and 60 min daily for two weeks.	Decreased glutathione and glutathione peroxidase in serum and testis. Increased conjugated diene, lipid hydroperoxide, and catalase and seminiferous tubules diameter.
Andrašková et al. (2022)	albino rat	1.73 W/kg	2.45 GHz	2 hours per day throughout the pregnancy.	Adverse effects on the postnatal testicular development. Structural changes in the testes, such as irregular shape of seminiferous tubules, significant decrease in the diameter of seminiferous tubules and in the height of the germinal epithelium, disorganisation of germ cells, desquamations of immature germ cells, formation of giant multinucleated cells, and significant expansion of the interstitium. Basement membrane irregularities in seminiferous tubules, vacuolation of the cytoplasm and adversely affected organelles in sertoli cells, germ cells, leydig cells, peritubular and endothelial cells. Enhanced necrotizations of germ cells and declined germ cells proliferation.
Dasdag et al. (2015)	Wistar Albino rat	2420 mW/kg	2.4 GHz	24 h/d for 12 months	No effect on sperm concentration and motility. Increased head defect. Decreased weight of epididymis, seminal vesicles, seminiferous tubules diameter, and tunica albuginea thickness.
Kumar et al. (2011a)	Wistar rat	0.014 W/kg	10 GHz	2 h/d, 45 days	Increased reactive oxygen species. Decreased G ₂ /M phase and histone kinase activity. No effect on G ₀ /G ₁ phase.
Kumar et al. (2011b)	Wistar rat	0.014 W/kg	2.45 GHz and 100 hz	2 h/d for 60 days	Increased caspase and creatine kinase. Decreased testosterone and melatonin.

NA, not mentioned or conducted in study.

Occupational exposure and male reproductive health

A retrospective cohort study conducted on 22,197 police officers working with traffic radar revealed an increase in the incidence of testicular cancer (Finkelstein 1998). Conversely, a population-based case-control study conducted in Germany by Baumgardt-Elms et al. (2002) examined the relationship between testicular cancer and electromagnetic fields in the workplace. No association was found between working near radar units and RF transmitters and testicular cancer (Baumgardt-Elms et al. 2002). A cross-sectional study conducted on 1487 male navy personnel and civilian staff working with RF equipment in Norway using a questionnaire showed a potential association between RF exposure and decreased reproductive ability (Møllerløkken and Moen 2008). Another cross-sectional study among 10,497 male soldiers working in the Royal Norwegian Navy found an increase in infertility with increasing RF exposure (Baste et al. 2008). In a cross-sectional study of young men aged 18–22, an increase in cell phone usage was found to be associated with low sperm concentration and low sperm count, although keeping the phone in the pants pocket was not effective (Rahban et al. 2023). On the other hand, conducted on 33 male soldiers in the Texas Signal Corps exposed to radar found no significant effect on sperm quality, follicle-stimulating hormone, luteinizing hormone, and testosterone levels (Schrader et al. 1998). Similarly, a study conducted on radar equipment operators in the US military, based on interactive questionnaire-based assessment of potential exposure, observed a decrease in sperm counts in the RF exposure group but no difference in exogenous sexual gland and sperm cell function assessment results (Weyandt et al. 1996). Another study involving 12 male heater operators living in the US showed that “RF exposure led to a decrease in sperm quality and hormonal differences” (Grajewski et al. 2000).

In conclusion, the diverse findings from these studies shed light on the complex relationship between occupational exposure, particularly to radiofrequency radiation, and male reproductive health outcomes (Table 4). The variability in study outcomes highlights the challenges in establishing a clear causal link, suggesting that individual susceptibility, exposure duration, and the specific characteristics of RF equipment used in different occupational settings may play significant roles. Further research is warranted to elucidate the underlying mechanisms and to inform occupational health guidelines and practices. Future studies should focus on standardizing exposure assessment methods and

consider long-term monitoring of workers in high-risk occupations to better understand the cumulative effects of RF radiation. Additionally, exploring potential protective strategies, such as shielding technologies and exposure limits, could be critical in mitigating the reproductive risks associated with occupational RF exposure.

Discussion

Mechanisms of RF radiation effects on reproductive system

Thermal effects

While the precise mechanism by which RF radiation affects human health remains unclear, its negative effects are often attributed to tissue heating caused by RF radiation exposure (Deepinder et al. 2007; Kesari et al. 2018). RF radiation, penetrating the body and interacting with tissues, is extensively absorbed, leading to a rise in temperature. Numerous studies have shown that when RF radiation interacts with tissues, it induces oscillating current and energy transfer, resulting in rapid heating (Avendano et al. 2012; Challis 2005; Kesari et al. 2018).

The thermal effects of RF radiation bear conceptual similarities to the microwave heating mechanism known as dipole polarization. Dipole polarization involves the alignment and re-alignment of polar molecules, such as water, with an oscillating electromagnetic field, resulting in energy transfer and heat generation through intermolecular friction. Similarly, RF radiation transfers energy within tissues through a comparable mechanism. However, operating at lower frequency ranges than microwaves, the dynamics of energy absorption and heating effects differ. As described by Foster (2000), the heating effects of RF radiation within tissues depend on factors such as water content, specific absorption rate (SAR), and the frequency of the electromagnetic field (Foster 2000). Thus, while the thermal effects of RF radiation share similar principles with microwave heating, the degree and mechanisms of interaction in biological systems vary depending on specific conditions.

Temperature increases have been studied particularly in relation to germ cells, which exhibit active mitotic processes (Shiraishi et al. 2012). Human sperm cells and initial spermatids have been found to be highly sensitive to temperature fluctuations (Carlsen 2003). Animal studies have shown that RF radiation elevates testicular temperature and affects the seminiferous tubule epithelium. These studies have revealed histological changes in the seminiferous epithelium, reduced sperm count, and abnormalities in sperm structure (R John Aitken

Table 4. Occupational exposure and male reproductive health.

Reference	Species	Specific absorption rate (W/kg)	Frequency (MHz)	Duration of exposure	Main outcomes
Finkelstein (1998)	Human	NA	NA	NA	Increased in incidence of testicular cancer
Baumgardt-Elms et al. (2002)	Human	NA	NA	NA	No relationship between testicular cancer and RF.
Schrader et al. (1998)	Human	NA	NA	NA	No differences on follicle-stimulating hormone, luteinizing hormone, testosterone and sperm parameters (sperm concentration, sperm/ejaculate, semen volume, percent normal morphology, percent motile, percent viable)
Weyandt et al. (1996)	Human	NA	NA	NA	Decreased in sperm counts/ml and sperm/ejaculate. No differences on sex gland, and sperm cell function.
Grajewski et al. (2000)	Human	NA	from 12 to 57 mHz	NA	Decreased in semen quality and increased in follicle-stimulating hormone.
Møllerløkken and Moen (2008)	Human	NA	NA	NA	Reduced fertility
Baste et al. (2008)	Human	NA	NA	NA	Reduced fertility
Rahban et al. (2023)	Human	NA	NA	NA	Decreased in sperm concentration and total sperm count.

NA, not mentioned or conducted in study.

et al. 2009; Kowalczyk et al. 1983; Varma and Traboulay 1975). Furthermore, studies have demonstrated that heat stress induces apoptosis in germ cells, mediated by both intrinsic and extrinsic pathways, disrupting spermatogenesis and leading to impaired sperm quality (Shahat et al. 2020). This highlights the direct role of heat-induced damage on male reproductive health.

When exposed to high levels of RF radiation, the resulting heating effect, surpassing the body's ability to regulate its temperature, is termed thermal effect. Thermal effects of RF radiation can potentially disrupt various biological activities (Deepinder et al. 2007). However, the International Commission on Non-Ionizing Radiation Protection (ICNIRP), responsible for global non-ionizing radiation safety, has established standard limits for RF radiation exposure, considering thermal effects. These guidelines aim to set maximum levels of radiation exposure globally to limit adverse health effects associated with RF radiation.

Consequently, the energy carried by RF radiation can be transferred to tissues, causing an increase in temperature. If excess heat is not absorbed by the body, it can lead to injury. Thermal effects of RF radiation can affect the male reproductive system, which is highly sensitive to temperature fluctuations. Even slight temperature increases can negatively impact sperm production and quality (Almášiová et al. 2018; Avendano et al. 2012). Due to this sensitivity, testicles have a thermoregulatory system known as countercurrent heat exchange in the pampiniform plexus, which minimizes heat stress (Sciorio et al. 2022). Radiation-induced temperature increases have been shown to cause reversible damage to spermatogenesis. Additionally, temperature elevations can lead to structural changes in

heat-sensitive proteins and alterations in heat shock protein production (IARC, 2013).

Most identified negative effects are primarily attributed to scrotal heating (Deepinder et al. 2007). Due to its external location, the human testis absorbs more RF radiation energy than internal organs. However, testicular temperature is critical for sperm formation and remains several degrees below body temperature (Durairajanayagam et al. 2015; Wallach et al. 1988). Consequently, RF radiation may negatively impact reproductive function by altering testicular function through thermal effects (Deepinder et al. 2007). Carrying a mobile phone in pants pockets or using a laptop near the testicles for prolonged periods can lead to heat-related complications (Deepinder et al. 2007; Bilgici et al. 2018). A 2005 study found that males who carried mobile phones near their waistline experienced a decrease in sperm count compared to those who kept their phones elsewhere or did not use them (Kilgallon and Simmons 2005). However, to discuss the thermal effects of RF radiation, a tissue temperature increase of at least 1°C is required (IARC, 2013). Mobile phone manufacturers claim that this situation is unlikely under normal usage conditions. Nonetheless, research has shown that high scrotal temperature leads to loss of testicular germ tissue and decreased spermatogenesis (Durairajanayagam et al. 2015; Munkelwitz and Gilbert 1998). Furthermore, a Danish study found a scrotal temperature-related decrease in inhibin B levels, resulting in a reduction in sperm count (Hjollund et al. 2002).

Mechanism of Action:

RF radiation can cause localized heating in tissues, including the testes (Gautam et al. 2022; Yadav et al.

2021). The testes require a temperature slightly lower than the body's core temperature for optimal spermatogenesis. Elevated temperatures can disrupt this process, leading to decreased sperm production, reduced motility, and increased rates of sperm abnormalities. This thermal effect is particularly relevant in scenarios where electronic devices are kept close to the reproductive organs for prolonged periods (Kaur et al. 2023).

The thermal effects of RF radiation on the reproductive system primarily arise from the absorption of RF energy by the tissues, which is then converted into heat. This localized heating can disrupt the normal function of the testes, which are highly sensitive to temperature changes. The disruption of the thermoregulatory mechanism in the testes can lead to an increase in testicular temperature, which adversely affects the process of spermatogenesis. Spermatogenic cells are particularly vulnerable to heat, and elevated temperatures can cause cell apoptosis, reduce sperm count, and impair sperm motility (Yardim et al. 2024). Recent studies indicate that thermal stress induced by RF radiation can destabilize DNA repair mechanisms in germ cells, leading to increased DNA fragmentation and further impairing spermatogenesis (Gao et al. 2022; Gautam et al. 2022; Yadav et al. 2021).

Moreover, the thermal effect can also impact the blood-testis barrier, increasing its permeability and allowing harmful substances to reach the spermatogenic cells, further impairing their function. Prolonged exposure to elevated temperatures can lead to chronic changes in the testicular environment, including oxidative stress and inflammation, which can exacerbate the negative effects on sperm quality and fertility (Kaur et al. 2023).

In occupational settings, individuals who are exposed to high levels of RF radiation for extended periods are at a higher risk of experiencing these thermal effects (Yadav et al. 2021). The cumulative thermal load on the testes can lead to significant reproductive health issues, highlighting the need for protective measures and monitoring in such environments.

Non-thermal mechanisms

The adverse effects of RF radiation on male reproductive health are still under investigation, and the specific mechanisms through which it may affect male fertility remain unclear. However, several studies have suggested that RF irradiation can alter ion transitions in cell membranes and affect ion channel activity, leading to an imbalance in the body's production of ROS and oxidative stress. Additionally, RF radiation may penetrate tissues and deposit energy, resulting in increased

temperature, which can negatively impact sperm production and motility. While much remains to be understood about the effects of RF radiation on male fertility, acknowledging its potential impact on the male reproductive system is essential for developing risk mitigation strategies.

Oxidative stress

Exposure to RF radiation has been shown in studies to induce oxidative stress in the human body (Dasdag and Akdag 2016; Lai 1996; Yakymenko et al. 2016). Oxidative stress occurs when there is an imbalance between the body's ability to neutralize ROS and its production of ROS. ROS are chemicals that can damage cells and DNA if not neutralized by antioxidants, posing a threat to cell integrity and DNA stability. Sperm cells, in particular, are highly susceptible to ROS damage due to their high content of unsaturated fatty acids in the cell membrane. Oxidative stress is one of the primary non-thermal effects of RF radiation. Increased ROS levels have been shown to damage sperm DNA, impair motility, and disrupt the sperm membrane integrity. This is supported by findings from Butković et al. (2024), who demonstrated that RF exposure induces significant ROS production, leading to oxidative damage in sperm DNA and membranes, ultimately compromising motility and viability (Butković et al. 2024). Upon exposure to RF radiation, ROS production is triggered, leading to oxidative stress and damage to sperm cells. This damage can result in reduced sperm motility, abnormal sperm morphology, and decreased sperm count, all of which can impact male fertility (Eşmekaya et al. 2011; Gautam et al. 2022; Liu et al. 2015). Oxidative stress has been linked to reproductive issues, including male infertility and impaired sperm function (Martinez et al. 2014). ROS-induced oxidative stress, in particular, has been associated to male infertility and reduced the function of sperm (Lavranos et al. 2012; Tremellen 2008). ROS-induced oxidative stress can attack the unsaturated fatty acids in the sperm membrane, leading to lipid peroxidation, membrane degradation, axoneme destruction, and compromised sperm function and viability (Tremellen 2008; Wang et al. 2003).

Enzyme antioxidants such as SOD, catalase, and GPX play a crucial role in protecting sperm against oxidative damage (Agarwal and Said 2005). However, previous research has shown that RF radiation exposure in rats can decrease levels of GPX and SOD while increasing catalase activity (Kesari et al. 2011). This alteration in the metabolic antioxidant capacity can elevate ROS levels, triggering the cell death cascade and potentially leading to male infertility due to excessive apoptosis.

Mechanism of action

RF radiation has been shown to increase the production of ROS within cells, leading to oxidative stress (De Iuliis et al. 2009; Kesari and Behari 2012). This imbalance between ROS production and the body's antioxidant defenses can cause significant cellular damage (Kesari and Behari 2012). Specifically, in the reproductive system, oxidative stress can damage sperm DNA, impairing its integrity and function (De Iuliis et al. 2009). The increased ROS can also attack the cellular membranes and proteins, leading to compromised sperm motility and viability (Houston et al. 2016). Antioxidant enzymes such as SOD, catalase, and glutathione peroxidase play crucial roles in neutralizing ROS, but their function can be disrupted by RF radiation, exacerbating oxidative damage (Kesari and Behari 2012).

Ionic imbalance

The effects of RF radiation on male reproductive health are actively under investigation, and the specific mechanisms through which RF radiation may influence male fertility are not fully understood. However, several studies have indicated that RF irradiation can alter ionic transitions in cell membranes and affect ion channels, potentially impacting male reproductive function (Hernández-Morales et al. 2020; Marchionni et al. 2006; Oh et al. 2021; Rao et al. 2008; Ruigrok et al. 2018). RF energy has the ability to disrupt ionic transitions in cell membranes by modifying the membrane's electrical properties.

The cell membrane, which is permeable, regulates the movement of ions in and out of the cell. This ion migration is essential for various biological processes, including nerve signal transmission and muscle contraction. By generating electric fields that disrupt normal ionic transitions across the membrane, RF radiation can alter the electrical characteristics of the cell membrane, potentially leading to changes in membrane potential and the function of ion channels and transporters.

There is evidence suggesting that RF radiation may affect the conductivity of specific ion channels, such as voltage-gated calcium and potassium channels, which play crucial roles in regulating cellular excitability (Kim et al. 2019; Pall 2013). Ion channels, proteins located in the cell membrane, act as gatekeepers for the passage of ions like calcium, potassium, and sodium into and out of the cell. These ions are involved in numerous cellular functions, including signal transmission, cell communication, and cell development and differentiation.

Certain studies indicate that RF irradiation can alter ion channel function and ion flow across these channels (Hu et al. 2021; Kovacic and Somanathan 2010). For

example, RF radiation exposure may impair the function of calcium channels, critical for regulating spermatogenesis. Additionally, RF radiation has been linked to changes in ion transitions within cellular membranes, affecting overall cell performance. Some studies suggest that RF radiation exposure may elevate intracellular calcium levels, potentially triggering various cellular responses and impairing sperm function (Gautam et al. 2022).

Evidence suggests that RF radiation-induced ionic imbalance disrupts the delicate equilibrium of calcium (Ca^{2+}), potassium (K^+), and sodium (Na^+) ions within sperm cells and testicular tissues, potentially impairing their normal functionality and male fertility (Chao et al. 2023; Panagopoulos et al. 2022). Calcium ions play a pivotal role in sperm motility, capacitation, and the acrosome reaction, which are essential processes for successful fertilization. Disruption of calcium homeostasis by RF radiation has been associated with abnormal intracellular Ca^{2+} levels, which may impair these critical processes and reduce sperm functionality (Panagopoulos et al. 2022; Srivasatav et al. 2022; Yadav et al. 2021). Similarly, potassium imbalances caused by RF radiation may affect the osmotic balance and structural integrity of sperm cells, compromising their motility. This dysregulation is believed to interfere with flagellar activity, an energy-dependent process reliant on tightly controlled ion fluxes (Chao et al. 2023; Gautam et al. 2023; Panagopoulos et al. 2022). Sodium ions, essential for maintaining the resting membrane potential and regulating cellular excitability, may also be affected by RF radiation, potentially undermining metabolic processes critical for sperm survival and functionality (Chao et al. 2023; Gautam et al. 2023; Panagopoulos et al. 2022).

Moreover, RF radiation-induced dysfunction of voltage-gated ion channels (VGICs) may exacerbate these ionic imbalances. This dysfunction has been proposed to initiate biochemical pathways that overproduce ROS, contributing to oxidative stress, DNA damage, and impaired sperm quality. Processes such as increased calcium signaling, NADPH oxidase activation, and dysfunction of the Na^+/K^+ pump are among the mechanisms thought to link ionic dysregulation with cellular damage and infertility (Panagopoulos et al. 2022; Wust et al. 2020). While the exact mechanisms remain incompletely understood, these findings emphasize the need for further research into how RF radiation-induced ionic imbalances influence male reproductive health.

Overall, while the precise mechanisms by which RF radiation may affect male reproductive function are not fully understood, research suggests that alterations in ion transitions across cell membranes and changes in

ion channel function may play a role. However, further investigation is needed to comprehensively understand the detrimental effects of RF radiation on male reproductive health and develop risk mitigation strategies.

Mechanism of action

RF radiation can disrupt the ionic balance within cells, particularly affecting ions such as Ca^{2+} , K^+ , and Na^+ (Chao et al. 2023; Gautam et al. 2023; Panagopoulos et al. 2022). These ions play crucial roles in maintaining cellular homeostasis, membrane potential, and signal transduction. Disruption of ionic balance by RF radiation can lead to altered cell function and increased cellular stress. For instance, elevated intracellular Ca^{2+} levels can activate various cellular pathways that lead to apoptosis (programmed cell death), further impairing reproductive health. Additionally, ionic imbalance can affect the motility and viability of sperm cells by altering the function of ion channels and transporters critical for sperm function (Panagopoulos et al. 2022; Srivasatav et al. 2022; Yadav et al. 2021).

Inflammation:

RF radiation exposure can induce inflammation in cells, potentially interfering with sperm production and function (Bilgici et al. 2018; Saygin et al. 2016). Absorption of RF radiation can lead to inflammation in the male reproductive system, which may result from an immune response to injury or infection. Inflammation, if chronic or persistent, can have detrimental effects on the body.

Studies have shown that RF irradiation can increase the production of inflammatory cytokines, signaling molecules that promote inflammation (Gupta and Srivastava 2022; Jaffar et al. 2019; Szilágyi et al. 2020). Elevated cytokine levels may lead to chronic inflammation in tissues, adversely affecting sperm production and quality (Saygin et al. 2016). Chronic inflammation in the male reproductive system can induce oxidative stress and damage sperm cells, impair spermatogenesis, and promote fibrosis, or scarring, in the testes, further reducing sperm quantity and quality (Agarwal et al. 2020). The specific mechanism by which RF radiation induces inflammation is not fully understood, and additional research is needed to fully grasp its potential impact on the male reproductive system.

In summary, the effects of RF radiation on male reproductive health are still being investigated, and the mechanisms by which it may affect male fertility are not completely understood. Research suggests that RF radiation can disrupt ion transitions in cell membranes and alter ion channel function, potentially leading to oxidative stress and inflammation, which can severely impact sperm production and mobility. Further

research is necessary to fully comprehend the effects of RF radiation on male fertility and to develop measures to mitigate potential risks. Understanding the potential influence of RF radiation on the male reproductive system is crucial for safeguarding human health. Additionally, the current exposure guidelines set by regulatory bodies do not adequately address the potential risks associated with long-term RF radiation exposure, highlighting the need for further investigation into its effects over prolonged periods.

Mechanism of action

RF radiation can induce inflammatory responses in tissues, including the reproductive organs. Inflammation is a biological response to harmful stimuli, such as pathogens or damaged cells, and can be triggered by oxidative stress. The increased production of ROS due to RF radiation can activate inflammatory pathways, leading to the release of pro-inflammatory cytokines and chemokines. RF radiation has been shown to induce inflammation in male reproductive tissues by triggering oxidative stress-mediated pathways (Shahin et al. 2019; Sisakht et al. 2020). This process is driven by the overproduction of ROS, which can activate key inflammatory signaling cascades (Gupta and Srivastava 2022; Ok et al. 2024). For example, RF radiation exposure may stimulate the nuclear factor-kappa B (NF- κ B) pathway, promoting the release of pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF- α) (Ok et al. 2024). These cytokines exacerbate cellular damage, impair spermatogenesis, and disrupt the hormonal balance necessary for normal sperm function (Gupta and Srivastava 2022). Chronic inflammation in reproductive tissues can result in fibrosis, leading to scarring and permanent damage to testicular structure and function (Shahin et al. 2019). Furthermore, studies suggest that inflammation induced by RF radiation can alter the permeability of the blood-testis barrier (Shahin et al. 2019; Sisakht et al. 2020), exposing germ cells to harmful immune responses and oxidative damage. Despite these findings, the precise molecular mechanisms linking RF radiation to chronic inflammation and its downstream effects on male fertility remain incompletely understood, highlighting the need for further investigation.

The insights from the mechanisms of RF-induced inflammation, alongside thermal and non-thermal effects, collectively highlight the complex interplay between RF exposure and male reproductive health. These findings underscore the necessity for comprehensive regulatory frameworks and further research to elucidate long-term implications, as discussed in the Conclusion.

Conclusion

This study comprehensively examined the mechanisms by which RF radiation may impact male reproductive health, focusing on both thermal and non-thermal pathways. The findings indicate that RF exposure, particularly through increased oxidative stress, ionic imbalances, and inflammation, can disrupt spermatogenesis and impair sperm quality. While thermal effects highlight the role of testicular temperature elevation, non-thermal mechanisms such as ROS generation and ionic dysregulation further emphasize the multifaceted nature of RF-induced reproductive toxicity. Despite the robust body of evidence, inconsistencies across studies warrant standardized research protocols to resolve methodological discrepancies. Future investigations should prioritize long-term exposure assessments and explore protective strategies to mitigate the risks associated with RF radiation. These efforts are critical for informing public health guidelines and ensuring reproductive health safety in the era of pervasive wireless communication technologies. Findings underline the importance of revisiting current regulatory standards to better protect reproductive health.

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