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TOPICAL REVIEW

Recent Reports of Wi-Fi and Mobile Phone-Induced Radiation on Oxidative Stress and Reproductive Signaling Pathways in Females and Males

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Abstract Environmental exposure to electromagnetic radiation (EMR) has been increasing with the increasing demand for communication devices. The aim of the study was to discuss the mechanisms and risk factors of EMR changes on reproductive functions and membrane oxidative biology in females and males. It was reported that even chronic exposure to EMR did not increase the risk of reproductive functions such as increased levels of neoantigens abort. However, the results of some studies indicate that EMR induced endometriosis and inflammation and decreased the number of follicles in the ovarium or uterus of rats. In studies with male rats, exposure caused degeneration in the seminiferous tubules, reduction in the number of Leydig cells and testosterone production as well as increases in luteinizing hormone levels and apoptotic cells. In some cases of male and female infertility, increased levels of oxidative stress and lipid peroxidation and decreased values of antioxidants such as melatonin, vitamin E and glutathione peroxidase were reported in animals exposed to EMR. In conclusion, the results of current studies indicate that oxidative stress from exposure to Wi-Fi and mobile phone-induced EMR is a significant mechanism affecting female and male reproductive systems. However, there is no evidence to this date to support an increased risk of female and male infertility related to EMR exposure.

Keywords Infertility · Female · Male · Testosterone · Electromagnetic radiation · Antioxidant · Oxidative stress

Abbreviation

EMR	Electromagnetic radiation
GSH	Glutathione
GSH-Px	Glutathione peroxidase
PUFAs	Polyunsaturated fatty acids
RF	Radiofrequency
ROS	Reactive oxygen species
SAR	Specific absorption rate
SOD	Superoxide dismutase
Wi-Fi	Wireless fidelity
WLAN	Wireless local area networks

Introduction

Wireless local area networks (WLANs) are an increasing alternative to wired data networks in workplaces, homes and public places. Nevertheless, as previously reported for other electromagnetic radiation (EMR) sources, the rapid increase in WLANs in our daily environment, especially in private, academic and clinical surroundings, has caused great public concern about the possible effects on human health. This concerning situation requires further investigation of the possible biological effects of exposure to WLAN signals, requiring new experiments both in vivo and in vitro (Mailankot et al. 2009).

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Reproduction is a critical function of the organism and involves two systems: the male and female genital organs. Recent evidences indicate that both ionizing and non-ionizing radiation induce oxidative stress in reproductive tissues (Gul et al. 2009; Esmekaya et al. 2011). However, there is limited knowledge on the involvement of EMR-induced free radicals in female infertility. In addition, near the time of ovulation, an increase in various substances in the follicle can physiologically induce oxidative stress and reactive oxygen species (ROS) production. Follicles may be vulnerable to oxidative stress induced by oocytes and become exposed to ROS continuously generated via the autooxidation of polyunsaturated fatty acids (PUFAs) of the follicles (Özkaya and Nazıroğlu, 2010; Tola et al. 2013). It is believed that oxidative stress may be a cause of poor oocyte quality. In addition, it has been suggested that oxidative stress might play a role in endometriosis development and endometriosis-associated infertility (Guney et al. 2007). The role of EMR-induced ROS in relation to female reproductive function has been a subject of recent interest (La Vignera et al. 2012).

One of the most relevant aspects of dosimeter studies is the measurement of biological parameters, i.e., growth of mice. The induced dose, i.e., specific absorption rate (SAR), and its maintenance throughout the exposure period have to be accurately investigated, due to the fast changes in body mass and size the animals undergo during their first weeks of life (Pinto et al. 2010).

EMR exposure by mobile phone and wireless fidelity (Wi-Fi) technology has increased in real life, and it has some degenerative effects on the reproductive cellular system. For example, the use of mobile phones has been connected to several genetic defects (Tice et al. 2002). More recently, exposure to radiofrequency and EMR has also increased concerns regarding the influence of wireless communication in general on health. Questions on the possible adverse influence of EMR on children and embryos/fetuses during pregnancy have been recurrent (Otto and von Mühlendahl 2007).

The increase in exposure to Wi-Fi communication signals has also raised public health concerns, especially about infertility and pregnancy. Despite ongoing public discussions on the consequences of the increased use of wireless communication devices, there is still considerable need of studies on this subject (Leszczynski and Xu 2010). Animal studies investigating the effects of early life and prenatal exposure to this source of EMR, in the radiofrequency range, with special emphasis on development and behavior, have been considered as high-priority research needs by the World Health Organization (Poulletier de Gannes et al. 2012).

ROS occur during physiological cellular functions such as mitochondrial respiration and phagocyte immune defense pathways (Nazıroğlu 2007). It has been proposed that the increasing use of electric power may be responsible for the increased incidence of male and female infertility in industrialized countries, and an oxidative stress hypothesis has been postulated that EMR-dependent suppression of antioxidant synthesis may be responsible for the stronger increase in infertility (La Vignera et al. 2012). However, some conflicting reports have indicated that overproduction of ROS though Wi-Fi- and mobile phone-induced EMR exposure have no effects on fertility in human and experimental animal studies (Poulletier de Gannes et al. 2012; Guney et al. 2007).

In this review, we discuss the results from recent reports on chronic EMR exposure-induced infertility in females and males and correlate them with findings on EMR-induced oxidative stress.

EMR and Female Fertility

Female reproductive organs have critical functions in the organism. As a result of reproductive tissue damage, abnormal embryo development may occur. More recently, there has been increasing concern about EMR-induced abnormal development of the fetus (Table 1). Exposure to non-ionizing EMR through Wi-Fi and mobile phones has been suggested as a potential risk factor for infertility (Poulletier de Gannes et al. 2012). However, some reports indicate that there are no degenerative effects of EMR on gestational development in animals. For example, it was recently reported that 2.14-GHz exposure for 20 h/day during gestation and lactation periods did not have any adverse influences on pregnancy or development of rats (Takahashi et al. 2010). Effects of long-term exposure to EMR on the reproductive system were also investigated, and surprisingly, no developmental or reproductive effects of 1,966 MHz fields were observed in four different generations of mice (Sommer et al. 2009).

Code division multiple access is a channel access method used by various radio communication technologies. When applying the code division multiple access signal to mice within the gestational period with an exposure time of 45 min twice per day, 15-min intervals in between and final examination 18 days later, no significant adverse influence on mouse fetuses was observed (Lee et al. 2009).

Neoantigens are presented by antibody molecules on the surface of tumor cells. Effects of Wi-Fi frequency on neoantigens were investigated by Aït-Aïssa et al. (2012). For this aim, the authors exposed rats to radiation of 2.45 GHz (2 h/day, 5 days weekly for a period of 30 days; whole body of rats in uterus and postnatal). The 2.45-GHz signal at SAR levels up to 4 W/kg for the dams and transiently up to 9 W/kg for the pups had no influence on the

Table 1 Effects of mobile phone and Wi-Fi frequencies on female reproductive system and molecular pathways in animals

Frequency (MHz) and animal	Effects	Exposure time (daily/weekly/total)	Molecular pathways	Reference
900 and rat	Negative	30 min/7 days/30 days	Endometrial inflammation and oxidative stress	Poulletier de Gannes et al. (2012)
900 and rat	Negative	11 h/21 days	Ovarian follicles	Guney et al. (2007)
2,140 and rat	No	20 h/7 days/10 weeks	Gestation and lactation periods	Gul et al. (2009)
900 and rat	No	45 min/7 days/18 days	Gestation period	Takahashi et al. (2010)
900 and mouse	No	45 min/7 days/18 days	Teratogenicity	Lee et al. (2009)
2,450 and rat	No	2 h/5 days/30 days	Selected neoantigens	Aït-Aïssa et al. (2012)
2,450 and mouse	No	6 h/7 days/5 and 9 days	Visceral and skeletal changes, brain cholinesterase activity and histology	Nawrot et al. (1985)
2,450 and mouse	Increased	6 h/5 days of pregnancy	Embryo lethality	Nawrot et al. (1985)

production of selected neoantigens. The authors concluded that the neoantigens are not connected to pathological processes or gestational outcome (Aït-Aïssa et al. 2012).

Poulletier de Gannes et al. (2012) found that non-ionizing EMR exposure at 2.45 GHz and whole-body specific absorption rates of 0.008 and 4 W/kg had no effect on lethality, abnormalities and clinical signs of rats (1 h/day for 5–6 weeks, short exposure).

Wi-Fi and Mobile Phone EMR-Induced Oxidative Stress in Reproductive Tissues

Oxidative stress and ROS formation occur during physiological processes including phagocytic and mitochondrial activities. If ROS formation is not controlled by antioxidants, it will cause oxidative degenerations on lipids, nucleic acids and proteins of reproductive cells (Lavranos et al. 2012; Nazıroğlu et al. 2013). During EMR exposure, concentrations of ROS are kept under strict control by the activity of a complex defense system including enzymatic and nonenzymatic antioxidants such as vitamin C and vitamin E (Chen et al. 2012; Nazıroğlu et al. 2012a). Enzymatic antioxidants include superoxide dismutase (SOD), catalase and glutathione peroxidase (GSH-Px). SOD enzyme induces dismutation of superoxide radicals to hydrogen peroxide, whereas catalase and GSH-Px detoxify hydrogen peroxide to water (Kovacic and Somanathan 2008). Vitamin E, α-tocopherol, is the most important antioxidant in the lipid phase of cells. Vitamin E acts to protect cells against the effects of EMR-induced free radicals, which are potentially damaging by-products of the body's metabolism (Kovacic and Somanathan 2008). Vitamin C, as well as being a free radical scavenger, recycles oxidized vitamin E to its active reduced form. Antioxidants are also essential for inhibition of EMR-related ROS production (Fig. 1) (Chen et al. 2012).

It is well known that EMR-induced injury leads to increased production of ROS. When water, which is a main constituent of cells, is exposed to EMR, ROS occurs through a variety of mechanisms (La Vignera et al. 2012). Although all respiring cells are equipped with protective enzymatic and nonenzymatic antioxidants, increased oxidative stress in cells stemming from EMR may overwhelm the protective systems and cause oxidative depletion of these antioxidants, leading to cell injury and apoptosis (Merhi 2012; Kesari et al. 2013). The resulting ROS formation is usually scavenged by the enzymatic and nonenzymatic antioxidants (Kovacic and Somanathan 2008; Merhi 2012; Kesari et al. 2013).

There are few reports on oxidative stress, EMR and female fertility, although there are numerous studies on the subject of oxidative stress related to radiofrequency. The scarce data on the subject are also conflicting (Kesari et al. 2010).

It was reported that vitamins C and E in combination modulated GSM-900-induced oxidative stress and inflammation in the endometrium of rats (Guney et al. 2007). In the study, the vitamin C and E combination increased SOD, catalase and GSH-Px values and decreased lipid peroxidation and nitric oxide values in the endometrium of GSM-900-exposed rats. It was also reported that exposure of female rats to microwaves of mobile phones decreased the number of follicles (Gul et al. 2009).

Recently, Oksay et al. (2012) investigated the protective effects of melatonin on Wi-Fi (60 min/day for a period of 30 days)-induced oxidative stress and antioxidant systems in testis of rats. They observed that Wi-Fi-induced EMR caused oxidative damage in testis by increasing the levels of lipid peroxidation and decreasing the concentrations of vitamins A and E. However, melatonin supplementation for 30 days prevented oxidative damage induced by EMR and supported the antioxidant redox system in the testis (Oksay et al. 2012).

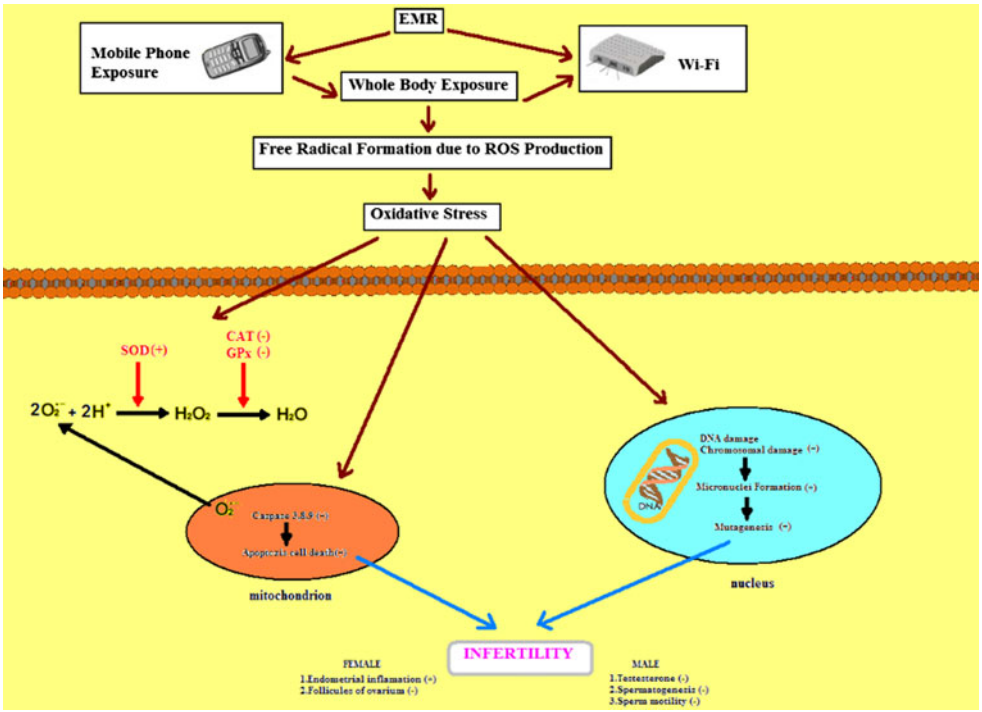

Fig. 1 A molecular pathway describing the mechanism of Wi-Fi and mobile phone frequency-induced electromagnetic radiation (*EMR*) on oxidative stress in infertility of females and males. (−) decrease, (+) increase

Avendaño et al. (2012) investigated the effects of Wi-Fi on human spermatozoa. They observed that Wi-Fi induced a decrease in progressive sperm motility and an increase in DNA fragmentation in human sperm. In another study, it was reported that sperm caspase and creatine kinase values increased by exposure to 2.45-GHz EMR for 2 h/day for a period of 60 days and that at the same time serum testosterone and melatonin values were decreased by the exposure (Avendaño et al. 2012).

In two recent studies, rats were exposed to commercial cell phones kept in standby mode for 2 h/day for 35 days (0.9 W/kg), and the results suggest that an increase in protein kinase activity may be related to overproduction of ROS under microwave field exposure; it was concluded that radiofrequency EMR from commercially available cell phones might negatively affect the fertilizing potential of spermatozoa (Kesari et al. 2010, 2011). The role of mobile phone frequency on glutathione levels was investigated in male rats, and the radiofrequency emitted from EMR exposure from GSM (0.9/18 GHz) mobile phones (1 h/day for 28 days) in male rats induced a high lipid peroxidation level and low glutathione concentrations (Mailankot et al. 2009). Related to this subject, Atasoy et al. (2013) investigated the effects of 2.45-GHz EMR on antioxidant and oxidant values in the sperm of rats. They observed that 2.45-GHz EMR induced an increase in lipid peroxidation and pathological degeneration as well as a decrease in catalase and GSH-Px antioxidant enzymes in the testis of rats. Similarly, Esmekaya et al. (2011) reported that 900-MHz EMR (1.20 W/kg and 20 min/day for 3 weeks) induced oxidative injury in testis by increasing nitric oxide levels and suppressing antioxidant defense mechanisms.

In a recent study, four groups of rabbits (pregnant and nonpregnant rabbits and their corresponding EMR-exposed littermates) were exposed to 1,800-MHz EMR for 15 min/day for a period of 7 days (Kismali et al. 2012). They observed no changes in levels of lipid peroxidation, cholesterol, total protein, albumin, uric acid, creatinine, creatine kinase and creatine kinase-myocardial band isoenzyme in the blood samples. Hence, they concluded that there were no effects of 1,800 MHz on oxidative stress toxicity in pregnant rabbit blood cells (Kismali et al. 2012).

EMR and Male Fertility

Recent results have indicated that male reproductive tissues are also affected by EMR, although some results have been conflicting (Table 2). For example, the weight of reproductive organs (testes, epididymides, seminal vesicles and prostate) of male rats differed as a result of exposure to 1,950-MHz EMR for 5 h/7 days and a period of 5 weeks (Imai et al. 2011). Short (2 h/5 days for 2 weeks) exposure of male rats to 1,800-MHz EMR did not induce differences in the morphology of testes, epididymides and prostate (Forgács et al. 2006). Related to this subject, Dasdag et al. (2003, 2005) observed no significant effects of short (20 min/day for 1 month) and long (2 h/7 days for 10 months) periods of mobile phone exposure on the apoptotic cell number in the testes. Aitken et al. (2005)

Table 2 Effects of mobile phone and Wi-Fi frequencies (MHz) on male reproductive system and molecular pathways in rats and rabbits

Frequency (MHz)	Effects	Time (daily/weekly/total)	Pathways	Reference
2,450	Negative	2 h/60 days	Serum testosterone and melatonin, sperm caspase and creatine kinase	Kumar et al. (2011)
2,450	Negative	4 h	Sperm motility and DNA fragmentation	Avendaño et al. (2012)
1,950	Negative	5 h/7 days/5 weeks	Testis weight	Imai et al. (2011)
1,800	No	2 h/5 days/2 weeks	Male organ morphology	Forgács et al. (2006)
900	No	20 min/7 days/1 months	Testis apoptosis	Dasdag et al. (2008)
900	No	2 h/7 days/10 months	Testis apoptosis	Dasdag et al. (2003)
900	No	12 h/7 days	Sperm number and morphology	Aitken et al. (2005)
800	No	8 h/12 weeks	(Rabbit) Male organ morphology	Salama et al. (2010)
900	No	2 h/7 days/1 months	Male organ morphology	Dasdag et al. (1999)
900	Negative	2 h/7 days/35 days	Fertilization of spermatozoa	Kesari et al. (2011)
900–1,800	Negative	1 h/7 days/28 days	Sperm motility	Mailankot et al. (2009)
2,450	No	16 h/7 days/30 days	Sperm count and morphology	Ono et al. (2004)
2,450	No	60 min/7 days/28 days	Diameter of seminiferous tubules and pyknotic, karyolectic and karyotic cell diameters	Saygin et al. (2011)
2,450	Negative	60 min/7 days/28 days	Leydig cells	Saygin et al. (2011)
2,450	Positive	60 min/7 days/28 days	Testicular apoptosis	Kismali et al. (2012)

reported that sperm number, morphology and vitality from mice exposed to 900-MHz EMR in a wave guide at 90 mW/kg 12 h/day for 7 days did not show significant differences. EMR-induced changes were also investigated in seminiferous tubular diameters of rat testes, and EMR-mobile phone exposure in rats for 2 h/day for 1 month (0.14 W/kg whole body) caused decreases in seminiferous tubular diameters when compared to the sham group. No difference in the study was found in sperm morphology (Dasdag et al. 1999). Similarly, Saygın et al. (2011) did not observe any significant effect of Wi-Fi (2.45 GHz and 3.21 W/kg) on the diameter of seminiferous tubules, pyknotic and karyolectic cells and caspase values of testis in rats. However, they observed a significant decrease in the number of Leydig cells and a significant increase in testicular apoptotic cell count in EMR-exposed rats. In a study conducted by Ono et al. (2004), mouse testis was exposed to 2.45-GHz EMR for 16 h/day for 30 days. They observed no significant differences in sperm count and sperm morphology.

In more recent studies Wi-Fi- and mobile phone-induced hormonal changes were investigated in experimental animals. One of these studies exposed rabbits to 800-MHz EMR from mobile phones in standby position for 8 h/day for 12 weeks, and hormonal assays indicated no significant differences between the study groups (Salama et al. 2010). Pinto et al. (2010) reported that the whole-body averaged Wi-Fi (2.45 GHz) SAR drastically changed during the exposure period according to the size and weight of the newborn mice. Thus, to expose newborn animals to defined and constant SAR levels, dosimetric issues need to be carefully investigated with daily assessments.

Conclusions

It should be noted that EMR exposure from Wi-Fi and mobile phones is related to oxidative stress and overproduction of free oxygen radicals in female and male infertility. Use of mobile phones and wireless devices has been increasing day by day. There are very scarce data on Wi-Fi-induced reproductive dysfunction in female and male individuals. However, carcinogenic and proliferative effects of mobile phones (Kim et al. 2010) and Wi-Fi (Kumar et al. 2011; Kesari et al. 2011; Nazıroğlu et al. 2012b) have been reported in animals and cell culture systems, although there is no report on Wi-Fi- or mobile phone-induced cancer in reproductive tissues of female and male individuals. In the future, the role of EMR from mobile phones and wireless devices in female and male fertility should be investigated.

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References

Aït-Aïssa S, Billaudel B, Poulletier de Gannes F, Ruffié G, Duleu S, Hurtier A, Haro E, Taxile M, Athané A, Geffard M, Wu T, Wiart

J, Bodet D, Veyret B, Lagroye I (2012) In utero and early-life exposure of rats to a Wi-Fi signal: screening of immune markers in sera and gestational outcome. Bioelectromagnetics 33:410–420

Aitken RJ, Bennetts LE, Sawyer D, Wiklendt AM, King BV (2005) Impact of radio frequency electromagnetic radiation on DNA integrity in the male germline. Int J Androl 28:171–179

Atasoy HI, Gunal MY, Atasoy P, Elgun S, Bugdayci G (2013) Immunohistopathologic demonstration of deleterious effects on growing rat testes of radiofrequency waves emitted from conventional Wi-Fi devices. J Pediatr Urol 9:223–229

Avendaño C, Mata A, Sanchez Sarmiento CA, Doncel GF (2012) Use of laptop computers connected to internet through Wi-Fi decreases human sperm motility and increases sperm DNA fragmentation. Fertil Steril 97:39–45

Chen L, Hu JY, Wang SQ (2012) The role of antioxidants in photoprotection: a critical review. J Am Acad Dermatol 67:1013–1024

Dasdag S, Ketani MA, Akdag Z, Ersay AR, Sari I, Demirtas OC, Celik MS (1999) Whole-body microwave exposure emitted by cellular phones and testicular function of rats. Urol Res 27:219–223

Dasdag S, Zulkuf Akdag M, Aksen F, Yilmaz F, Bashan M, Mutlu Dasdag M, Salih Celik M (2003) Whole body exposure of rats to microwaves emitted from a cell phone does not affect the testes. Bioelectromagnetics 24:182–188

Dasdag RJ, Bennetts LE, Sawyer D, Wiklendt AM, King BV (2005) Impact of radio frequency electromagnetic radiation on DNA integrity in the male germline. Int J Androl 28:171–179

Dasdag S, Akdag MZ, Ulukaya E, Uzunlar AK, Yegin D (2008) Mobile phone exposure does not induce apoptosis on spermatogenesis in rats. Arch Med Res 39:40–44

Esmekaya MA, Ozer C, Seyhan N (2011) 900 MHz pulse-modulated radiofrequency radiation induces oxidative stress on heart, lung, testis and liver tissues. Gen Physiol Biophys 30:84–89

Forgács Z, Somosy Z, Kubinyi G, Bakos J, Hudák A, Surján A, Thuróczy G (2006) Effect of whole-body 1800 MHz GSM-like microwave exposure on testicular steroidogenesis and histology in mice. Reprod Toxicol 22:111–117

Gul A, Celebi H, Uğraş S (2009) The effects of microwave emitted by cellular phones on ovarian follicles in rats. Arch Gynecol Obstet 280:729–733

Guney M, Ozguner F, Oral B, Karahan N, Mungan T (2007) 900 MHz radiofrequency-induced histopathologic changes and oxidative stress in rat endometrium: protection by vitamins E and C. Toxicol Ind Health 23:411–420

Imai N, Kawabe M, Hikage T, Nojima T, Takahashi S, Shirai T (2011) Effects on rat testis of 1.95-GHz W-CDMA for IMT-2000 cellular phones. Syst Biol Reprod Med 57:204–209

Kesari KK, Kumar S, Behari J (2010) Mobile phone usage and male infertility in Wistar rats. Indian J Exp Biol 48:987–992

Kesari KK, Kumar S, Behari J (2011) Effects of radiofrequency electromagnetic wave exposure from cellular phones on the reproductive pattern in male Wistar rats. Appl Biochem Biotechnol 164:546–559

Kesari KK, Kumar S, Nirala J, Siddiqui MH, Behari J (2013) Biophysical evaluation of radiofrequency electromagnetic field effects on male reproductive pattern. Cell Biochem Biophys 65:85–96

Kim KB, Byun HO, Han NK, Ko YG, Choi HD, Kim N, Pack JK, Lee JS (2010) Two-dimensional electrophoretic analysis of radiofrequency radiation-exposed MCF7 breast cancer cells. J Radiat Res 51:205–213

Kismali G, Ozgur E, Guler G, Akcay A, Sel T, Seyhan N (2012) The influence of 1800 MHz GSM-like signals on blood chemistry and oxidative stress in non-pregnant and pregnant rabbits. Int J Radiat Biol 88:414–419

Kovacic P, Somanathan R (2008) Unifying mechanism for eye toxicity: electron transfer, reactive oxygen species, antioxidant benefits, cell signaling and cell membranes. Cell Membr Free Radic Res 1:56–69

Kumar S, Kesari KK, Behari J (2011) The therapeutic effect of a pulsed electromagnetic field on the reproductive patterns of male Wistar rats exposed to a 2.45-GHz microwave field. Clinics (Sao Paulo) 66:1237–1245

La Vignera S, Condorelli RA, Vicari E, D'Agata R, Calogero AE (2012) Effects of the exposure to mobile phones on male reproduction: a review of the literature. J Androl 33:350–356

Lavranos G, Balla M, Tzortzopoulou A, Syriou V, Angelopoulou R (2012) Investigating ROS sources in male infertility: a common end for numerous pathways. Reprod Toxicol 34:298–307

Lee HJ, Lee JS, Pack JK, Choi HD, Kim N, Kim SH, Lee YS (2009) Lack of teratogenicity after combined exposure of pregnant mice to CDMA and WCDMA radiofrequency electromagnetic fields. Radiat Res 172:648–652

Leszczynski D, Xu Z (2010) Mobile phone radiation health risk controversy: the reliability and sufficiency of science behind the safety standards. Health Res Policy Syst 8:1478–1505

Mailankot M, Kunnath AP, Jayalekshmi H, Koduru B, Valsalan R (2009) Radio frequency electromagnetic radiation (RF-EMR from GSM 0.9/1.8 GHz) mobile phones induces oxidative stress and reduces sperm motility in rats. Clinics (Sao Paulo) 64:561–565

Merhi ZO (2012) Challenging cell phone impact on reproduction: a review. J Assist Reprod Genet 29:293–297

Nawrot PS, McRee DI, Galvin MJ (1985) Teratogenic, biochemical, and histological studies with mice prenatally exposed to 2.45-GHz microwave radiation. Radiat Res 102:35–45

Nazıroğlu M (2007) New molecular mechanisms on the activation of TRPM2 channels by oxidative stress and ADP-ribose. Neurochem Res 32:1990–2001

Nazıroğlu M, Tokat S, Demirci S (2012a) Role of melatonin on electromagnetic radiation-induced oxidative stress and Ca^{2+} signaling molecular pathways in breast cancer. J Recept Signal Transduct Res 32:290–297

Nazıroğlu M, Ciğ B, Doğan S, Uğuz AC, Dilek S, Faouzi D (2012b) 2.45-GHz wireless devices induce oxidative stress and proliferation through cytosolic Ca^{2+} influx in human leukemia cancer cells. Int J Radiat Biol 88:449–456

Nazıroğlu M, Yoldaş N, Uzgur EN, Kayan M (2013) Role of contrast media on oxidative stress, Ca^{2+} signaling and apoptosis in kidney. J Membr Biol 246:91–100

Oksay T, Naziroğlu M, Doğan S, Güzel A, Gümral N, Koşar PA (2012) Protective effects of melatonin against oxidative injury in rat testis induced by wireless (2.45 GHz) devices. Andrologia 10:1111–12044

Ono T, Saito Y, Komura J, Ikehata H, Tarusawa Y, Nojima T, Goukon K, Ohba Y, Wang J, Fujiwara O, Sato R (2004) Absence of mutagenic effects of 2.45 GHz radiofrequency exposure in spleen, liver, brain, and testis of lacZ-transgenic mouse exposed in utero. Tohoku J Exp Med 202:93–103

Otto M, von Mühlendahl KE (2007) Electromagnetic fields (EMF): do they play a role in children's environmental health (CEH)? Int J Hyg Environ Health 210:635–644

Özkaya MO, Nazıroğlu M (2010) Multivitamin and mineral supplementation modulates oxidative stress and antioxidant vitamin levels in serum and follicular fluid of women undergoing IVF. Fertil Steril 94:2465–2466

Pinto R, Lopresto V, Galloni P, Marino C, Mancini S, Lodato R, Pioli C, Lovisolo GA (2010) Dosimetry of a set-up for the exposure of

newborn mice to 2.45-GHZ Wi-Fi frequencies. Radiat Prot Dosim 140:326–332

Poulletier de Gannes F, Billaudel B, Haro E, Taxile M, Le Montagner L, Hurtier A, Ait Aissa S, Masuda H, Percherancier Y, Ruffié G, Dufour P, Veyret B, Lagroye I (2012) Rat fertility and embryo fetal development: influence of exposure to the Wi-Fi signal. Reprod Toxicol 36C:1–5

Salama N, Kishimoto T, Kanayama HO, Kagawa S (2010) Effects of exposure to a mobile phone on sexual behavior in adult male rabbit: an observational study. Int J Impot Res 22:127–133

Saygın M, Caliskan S, Karahan N, Koyu A, Gumral N, Uguz A (2011) Testicular apoptosis and histopathological changes induced by a 2.45 GHz electromagnetic field. Toxicol Ind Health 27:455–463

Sommer AM, Grote K, Reinhardt T, Streckert J, Hansen V, Lerchl A (2009) Effects of radiofrequency electromagnetic fields (UMTS) on reproduction and development of mice: a multi-generation study. Radiat Res 171:89–95

Takahashi S, Imai N, Nabae K, Wake K, Kawai H, Wang J, Watanabe S, Kawabe M, Fujiwara O, Ogawa K, Tamano S, Shirai T (2010) Lack of adverse effects of whole-body exposure to a mobile telecommunication electromagnetic field on the rat fetus. Radiat Res 173:362–372

Tice RR, Hook GG, Donner M, McRee DI, Guy AW (2002) Genotoxicity of radiofrequency signals. I. Investigation of DNA damage and micronuclei induction in cultured human blood cells. Bioelectromagnetics 23:113–126

Tola EN, Mungan MT, Uğuz AC, Nazıroğlu M (2013) Intracellular Ca^{2+} and antioxidant values induced positive effect on fertilization ratio and oocyte quality of granulosa cells in patients undergoing in vitro fertilisation. Reprod Fertil Dev 25:746–752