Executive summary of the systematic review paper by Isabel Wilke: Biological and pathological effects of 2.45 GHz radiation on cells, fertility, brain, and behaviour (January 2018)

Subject Matter

The research draws attention to the need to reintroduce to policy-makers and the public debate the crucial matter of the effects of non-ionising radiation at the microwave frequency of 2.45 GHz and the health risks it might pose. The number of studies conducted and the significance of their findings still remains reversibly analogous to the measures taken to prevent its EMF/RF's detrimental effects. Insofar, numerous experiments have been carried out in cell cultures, humans, animals, bacteria, and plants.

Methods of Analysis

More than 100 studies published in peer-reviewed journals were selected for analysis through the databases LIVIVO (ZBMED) and PubMed. The chosen studies were conducted by different research teams, aimed at different study objectives and focused on different exposure levels, addressing a broad spectrum of issues. No date limitation was applied.

Key Findings

The review is sectioned area-wise, focusing on different areas, such as organs, functions, cells, and organisms. Table 1 includes the parameters affected and also references of the studies used in the review. The following summaries refer to studies performed in a murine model, unless stated otherwise.

Testes and sperm

The results of the associated studies are consistent with the hypothesis that 2.45 GHz radiation compromises fertility in regards to sperm production and quality.

Specifically, the testicular tissue showed increased sensitivity to DNA damage in relation to other organs (Akdag et al., 2016). The developmental stage plays a role as an added sensitivity factor, as shown from a study by Atsoy et al. (2013), who chronically exposed young male rats, whole-bodily, reporting significantly more pronounced effects of oxidation on the developing testes, in relation to other samples. Sperm motility was significantly affected in human subjects, accounted for by a higher percentage of immotile sperm and reduced progressive sperm motility (Avendaño et al., 2012). Sperm morphology was also affected, as was shown by increased percentages of defective sperm heads (Dasdag et al., 2015). Dasdag et al. (2015) also reported significantly reduced weight of the epididymis and seminal vesicle, reduced diameter of the seminiferous tubules, and reduced thickness of the tunica albuginea. In accordance with those findings, Shokri et al. (2015) recorded reduced weight of the seminal vesicles, reduced diameter and increased apoptosis rate (increased caspace-3 activity) in the seminiferous tubules, as well as significant damage to the latter's germinal epithelia (fewer cell layers). Meena et al. (2014) and several papers by Prof. M. Naziroğlu group concluded that the adverse effects on testes are caused by oxidative stress, and can be reduced by melatonin. In agreement with the above, Kumar et al. (2011) reported increased oxidative stress (increased caspase-3 activity) in the testes, and increased apoptosis, while melatonin and testosterone levels were significantly lower in the serum. The latter state that oxidative stress and thus its impacts can be alleviated by a pulsed electromagnetic field of 100 Hz. Shahin et al. (2014) documented testicular tissue degeneration, and increased oxidative and nitrosative stress, following their findings, the degeneration of the seminiferous tubules and reduction of their diameter, and damage on the Leydig cells, decreased sperm count and motility, highly significant alteration of ROS concentration in the testes, as well as significant changes to nitrate, nitrite concentrations, lipid peroxidation and antioxidant enzymes, reduced 3β-HSD activity and testosterone concentration, and elevated iNOS levels.

The following review papers also collectively conclude that 2.45 GHz RF radiation impairs male fertility: Agarwal (2009), Adams et al. (2014), Agarwal et al. (2011), Behari/Rajamani (2012), Bellieni/ Pinto (2012), British Columbia Centre for Disease Control (BCCDC) (2013), Dama/Bhat (2011), Gye/Park (2011), and La Vignera et al. (2012).

Female reproduction

Nakamura et al. (2000) reported increased secretion of progesterone and prostaglandin $F_2\alpha$, both observed only in pregnant animals, disturbances in the uterus/placenta cycle that they attribute to the elevation of the latter, and increased corticosterone and decreased estradiol, regardless of whether or not pregnant. Yüksel et al. (2016) examined the effects on chronically exposed pregnant rats and their offspring, concluding that both the adults and the developing rats exhibited elevated intra-uterine temperature, hormonal changes (decreased prolactin, estrogen and progesterone) and in maternal rats increased uterine oxidative stress during gestation. Othmann et al. (2017) demonstrated significant effects on fecundity (50% reduction) of exposed pregnant rats, and multiple effects on young offspring who had been exposed prenatally (in-utero). As juveniles (of 28, 30, 31 days old), the offspring were afflicted with delayed brain development (delayed reactions, impaired sense of balance, impaired maturation of the motor system), and altered antioxidant enzyme activities in the brain, due to global oxidative stress in the brain. During adulthood the offspring presented behavioral (anxiety, motor deficit, exploratory behavior impairments) and biochemical (altered levels of phosphorus, magnesium, glucose, triglycerides and calcium in the serum) impairments. In the Drosophila model, significant increase in apoptosis rate was reported during oogenesis, and fecundity was reduced by 10% and 30% after exposure to a) Wi-Fi and Bluetooth, and b) cell phone or cordless phones, respectively (Margaritis et al., 2014). The authors attribute the effects on the pulses rather than the ELF components, which explains the similar level of impact between low exposure pulsed and 43 times higher exposure of non-pulsed signals. Shahin et al. (2013) documented increased embryonic mortality, suppressed implantation and poor/delayed embryonic development as a result of irradiation-induced physiological stress responses. Particularly, due to oxidative stress (increased ROS levels, decrease in NO and ROS-scavenging enzymes activity), the elevation of progesterone and estradiol, DNA strand breaks, and the induction of an inflammatory process (increased leukocytes count, hemoglobin and erythrocytes count).

Moreover, Özorak et al. (2013) demonstrated the increased sensitivity of younger rats (higher at 4 and reduced towards 5 and 6 weeks old) in relation to older individuals to the effects of the radiation in regards to oxidative stress damage in testes and kidneys as reflected by increased levels of lipid peroxidation, oxidizable iron, and lower concentrations of trace elements, TAS and glutathione (GSH). Accordingly, said developmental stage in rats is comparable to puberty in humans.

Electroencephalogram (EEG), brain function and brain development

Early studies on human subjects revealed though EEG recording alterations in the alpha activity (alpha waves) (Lebretcht von Klitzing, 1995), that were confirmed by multiple recent studies, one of which is that of Maganioti et al. (2010), who found significant changes in the alpha and beta bands in combination with insignificantly increased energy in men and significantly decreased energy in women, due to sex-dependent physiological effects on the excitability of the cerebral cortex. Papageorgiou et al. (2011) used EEG to record P300 waves, components that are activated during thought and memory processes, and found significant amplitude in P300 between exposed vs non-exposed individuals and between the genders (increased in men and decreased in women).

The following studies were conducted on the murine model. Aggarwal et al. (2013) observed significant differences across all frequency bands (alpha, beta, delta, theta), varying in intensity based on the exposure time, while theta and beta band changes were more prominent. The alterations were attributed to changes in the synchronization or desynchronization of firing neurons, and accordingly may affect the blood-brain barrier and the concentration levels of neurotransmitters in synapses, causing psychopathophysiological impairments. In a series of follow-up studies, Lai/Singh et al. (1987a, 1987b, 1988, 1989a, 1989b, 1991, 1994, 1996b) uncovered the mechanisms by which Wi-Fi radiation affects cholinergic activity, thus impairing spatial learning and memory. The researchers reported the involvement of endogenous opioid-neurotransmitter systems, the reduced intake of choline by the hippocampus, and the increased concentration of muscarinic cholinergic receptors in the hippocampus. Naziroğlu and Gümral (2009)

reported a slight hyperexcitability in EEG, while their subsequent findings include signs of oxidative damage in the brain tissue, all of which could be prevented in different degrees by the administration of selenium or L-carnitine (lower GSH peroxidase activity, increased lipid peroxidation and decreased levels of vitamins A, C and E). Eser et al. (2013) were presented with tissue changes, increased oxidative status, increased apoptosis, inflammation (interleukin 1 beta), and neural degeneration in areas of the brain (frontal cortex, cerebellum, brain stem) that according to the researchers may cause functional impairments and trigger carcinogenesis. In a comparative study between newborn and adult rats, Orendáčová et al. (2009) were led to the conclusion that the effects of Wi-Fi radiation on neurogenesis (development and maturation of neurons) are dose- and age-dependent (hence more harmful to newborns). Age-dependent immunohistochemical effects were also found in the rostral migratory stream (RMS), concerning the expression of immediate early genes (product Fos proteins), and the earlier appearance and higher prevalence of NO-producing cells in exposed newborns and young adults (Orendáčová et al., 2011). In the brain cells of exposed rats, the activity of Calcium-dependent protein kinase C (PKC) (an enzyme that regulates the release of neurotransmitters and the formation of long-term memory) was decreased, glial cell numbers were increased (Paulraj and Behari, 2006a), and the mean concentration of acetylcholine (ACh) dropped reaching its lowest level 5 hours after exposure – implying the null contribution of thermal effects (Testylier et al., 2002). Sinha (2008) was presented with a 2.1% increase of the water content in the brain of rats, causing it to swell by 6.97%. In mice, various changes in the gene expression occurred, involving among others the tumor necrosis factor (TNF-α), and inducible nitric oxide synthase (iNOS). Furthermore, microglia activation was initiated, causing the activation of a pro-inflammatory response and the JAK-STAT signal pathway (Yang et al., 2010). Following that study, Yang et al. (2012) identified stress-related genes that were altered due to exposure, counting 41 in the hippocampus. Those concerned heat shock proteins (HSP27 and HSP70), metabolism, signal transduction, cytoskeleton, apoptosis, cell attachment, DNA repair, and other. In the hippocampus of male rats, Chauhan et al. (2017) observed degenerative manifestations due to oxidative stress, after exposing the animals to a microwave oven at 0.2 mW/ cm^2 for 2 hours per day for 35 days. Furthermore, structural tissue damage (increased lipid peroxidation) was also detected in the testes, spleen, liver and kidneys.

Behavior

Studies regarding behavior were conducted with regards to tracing changes in learning, memory, brain performance, typical behavioural patterns, and other responses, including anxiety and hyperactivity.

During 5-minute-long exposure to a device (notebook) with activated Wi-Fi function caused ants to become disorientated and behave erratically, looking sick, while, following exposure to Wi-Fi routers for 30 minutes, their normal foraging pattern was disturbed, escalating with time, requiring 6 to 8 hours post-exposure to be re-established (Cammaerts und Johansson, 2014).

In male mice, spatial memory was impaired as it was determined through running wheel and water labyrinth activity assessment (Chaturvedi et al., 2011), while further exposure-dependent impairments in learning and memory were observed, including neuronic degeneration, decreased levels of creatine kinase and antioxidant enzymes (SOD, CAT, GSH-Px), and increased levels of apoptosis, ROS, RNS, lipid peroxidation, DNA damage, and protein damage, were detected by Shahin et al. (2015).

In male rats the ability to differentiate between known and unknown objects (Hassanshahi et al., 2017), spatial orientation, spatial learning, other forms of learning, memory performance (Deshmukh et al., 2015, Li et al., 2008), and general brain performance (Deshmukh/Banerjee et al., 2016) were impaired, and alongside these findings, HSP70 levels and motor activity were elevated, and hyperactivity was also observed (Sinha, 2008). Physiological changes also occurred in rodent brains, involving the neurotransmitter system (serotonin, catecholamine, and acetylcholine), the hippocampus (increased apoptosis, the function of glucocorticoid receptors, regions CA1/ CA3/ dentate gyrus, decreased cholinergic activity) (Li et al., 2008), the frontal cortex (decreased cholinergic activity) (Wang/Lai, 2000), and increased single and double-strand DNA breaks in brain cells.

DNA

Higher prevalence of single and double DNA strand breaks in samples from brain tissue, blood, lymphocytes, testes and sperm, and less pronounced in the liver, skin and the kidneys (Akdag et al., 2006) was reported in several studies in the murine and human model systems. Such occurrence is evidence of increased oxidative stress that leads to genotoxic and mutagenic effects, that when frequent may lead to neurodegenerative diseases, disorders, impairments of neurological functions, vascular damage in the brain tissue, neurasthenia, tumor promotion/cancer, mutations, apoptosis, and free radicals associated with various other diseases (Lai and Singh, 1997).

Lai and Singh (1995) detected significantly higher levels of DNA strand breaks 4 hours after exposure, thus excluding any thermal effects. The experiment was repeated with the addition of free radical scavengers (melatonin and N-tert-butyl-alpha-phenylnitrone(PBN)), that proved protective as they reduced the effect (Lai and Singh, 1997). Reduced levels of DNA damage were also reported when a magnetic field of 45 mG was used in combination with the 2.45 GHz radiation (Lai and Singh, 2005). Akdag et al. (2016) and Avendaño et al. (2012) demonstrated significant DNA fragmentation on the testicular tissue of rats and human sperm, respectively. Single strand DNA breaks that could be significantly prevented by garlic extract intake were observed in rats in their brain tissue (indicated by elevated 8-hydroxy-deoxyguanosine (8-OHdG)) and the blood (as indicated by the levels of protein oxidation – Advanced Oxidation Protein Products)(Gürler et al., 2014). Increased double-strand breaks were pinpointed via comet assays in rat brains, in addition to other findings, including reduced activity levels of the enzymes SOD, glutathione and histone kinase, and increased catalase levels (Kesari/Behari et al., 2010a, 2010b, 2012). Human lymphocytes also exhibited signs of EMF radiation-mediated DNA damage, more specifically, a threefold increase in chromosomal aberrations and increased frequency of micronuclei (indication of chromosomal damage in mitogen-stimulated human lymphocytes) (Maes et al., 1993; Zotti-Martelli et al., 2000). The elevation of micronuclei was deemed by the authors to be exposure, duration and field strength dependent (Zotti-Martelli et al., 2000).

Cancer cells

Frequencies of EMF radiation promote cancer development via indirect pathways, either through changes in enzyme activities, cell membrane properties, or singe- and double-strand DNA breaks, that lead to metabolic changes. In a review paper of 89 studies, Naziroğlu et al. (2012c) concluded that the radiation-induced disturbance of the calcium homeostasis is related to breast cancer. Melatonin, on the other hand, acts as a radical oxidative stress scavenger, proving protective against breast tumorigenesis. It was found that at night EMF/RF exposure downregulates the production of melatonin, therefore, reducing its protective role as a calcium channel regulator. In continuation of the previous, Cig & Naziroğlu (2015), demonstrated that 2.45 GHz radiation, as well as 900 & 1800 MHz radiation cause excessive oxidative responses (DNA damage and cell cycle arrest), and apoptosis (via activation of some transcription and apoptotic factors) in breast cancer cells when emitted at 10 cm distance, via increasing the permeability of TRPV1 cation channels, thus increasing cytosolic calcium accumulation. In distance greater than 10 cm, the adverse effects on cancer cells were decreased. In human lymphocytes significant cell damage was observed under exposure to 2.45 GHz radiation, peaking under combined stressor conditions of EMF/RF and increased temperature, presented with increased prevalence of degeneration (lymphoblastic transformation) (Czerska et al., 1992).

Cardiac activity

In vivo studies in rabbits (Saili et al., 2015) and rats (Zhu et al., 2016, Kim & Rhee, 2004) demonstrated the dysregulation of the cardiovascular system through interference with receptor and ligand bonds, resulting in higher heart rate and increased blood pressure (Saili et al., 2015), dose-dependent changes in various biochemical parameters (including ATP activity, mitochondrial respiratory chain, creatin kinase, lactate dehydrogenase, aspartate transaminase, troponin I, the oxidative parameters SOD and GSH, lipid peroxidation), morphological changes (irregular striations on heart muscle cells, discoloration of nuclei), malfunctioning mitochondria (fewer in number, with destroyed membranes and other degenerative manifestations), and significantly increased levels of apoptosis of heart muscle cells (apoptosis proteins and rates) (Zhu et al., 2016), and indications of oxidative damage (formation of superoxide radical, increased lipid peroxide, oxidized protein, and lipofuscin, and weakened antioxidative defense system of heart tissue) (Kim & Rhee, 2004). Additionally, according to Von Klitzing (2014), the 10 Hz pulse included in Wi-Fi radiation plays an additional detrimental role on heart rate variability.

Cell cycle

Out of the two reviewed studies regarding the cell cycle, both had their methods adjusted to rule out any changes owing to thermal effects. In the first study by Cleary et al. (1996), exposure of synchronized hamster ovarian cells demonstrated changes during all stages of the cell cycle under controlled temperature (37°C). In the study conducted by Ballardin et al. (2011), V79 Chinese hamster cells exhibited reversible changes in the mitotic spindle apparatus during cell division, resulting in increased apoptosis (reduced viability), after short-term radiation exposure of only 15 minutes.

The liver

2.45 GHz radiation reportedly triggers oxidative stress to the liver, causing cellular and tissue damage, thus compromising the optimal functionality of the organ. Holovska et al. (2015) reported alterations in the structure and ultrastructure of the liver, such as increased blood in the vessels, the sporadic presence of necrotic cells, dilation of liver sinusoids, foci of inflamed tissue in hepatic lobules, and occurrence of hepatocyte abnormalities, including binucleated cells, irregular nuclei, condensed chromatin contained in the nuclei, presence of more vesicles and lipid droplets, and abnormal membrane structures. Additionally, Kumari et al. (2012) examined the liver of exposed rats, observing indications of overproduction of free radicals responsible for oxidative cell damage, i.e. increased MDA, TBIL, ALP, ALT, and AST concentrations, and significant suppression of antioxidant enzymes (shown by increased levels of catalase, and reduced levels of GSH-Px and liver SOD activity).

The thyroid

Significant changes were determined on the levels of heat shock proteins and thyroid hormones, the morphology of the thymus, and the responses of the animals. Particularly, signs of stress were detected, as heat shock protein concentrations (HSP70 and HSP90) were significantly reduced (Misa-Agustiño et al., 2012, 2015). The authors added that those findings indicate impairment of maturity and development of thymocytes, and improper regulation of the apoptotic and immune response mechanisms. The morphological changes reported by Misa-Agustiño et al. (2015) include the increased permeability of the endothelium in the thymus, the formation of new vessels, and the increased presence of reticular epithelial cells, red blood cells, and glucocorticoid receptors (GR) in the thymus cortex. Impaired thyroid hormone levels, i.e. decreased T3, increased T4 (Sinha, 2008; Sinha/Aggarwal et al., 2008) were also recorded.

Gene expression

In a study by Lee et al. (2005), the gene expression in human cell cultures of the HL60 cell line was found to be altered, even after only 2 hours of exposure (221 genes affected). After 6 hours of exposure, 896 genes were affected, 154 of which were unknown. Genes involved in apoptosis, metabolism, polysaccharide biosynthesis, and RNA functions and translation were upregulated, while transport, metabolism, RNA functions and cell cycle genes were downlegulated. The authors note that the non-significant increase of the gene expression of heat shock proteins demonstrates the non-thermal nature of 2.45 GHz radiation impact.

Cell membranes

Cell membranes were shown to also be affected by 2.45 GHz radiation exposure, through polarization that dysregulates calcium (Ca^{2+}) ion channels (i.e. VGCC and TRPM2), even in extremely low exposure levels, due to its independency to power density levels (Panagopoulos et al., 2015). Particularly, the dysregulation of calcium homeostasis causes oxidative damage and affects functions such as cell growth, signal transmission, and apoptosis. Through the same mechanism, the increased Ca^{2+} influx into the cytoplasm was reported to have increased cell growth in human leukemia cells (Naziroğlu et al., 2012b). By counteracting oxidative stress, melatonin can reduce the adverse effects on brain tissue (Naziroğlu et al., 2012a).

Bacteria

Taheri et al. (2015, 2017) reported the development of antibiotic resistance in Klebsiella pneumonia, Listeria monocytogenes and Escherichia coli, to 5,1 and 6 antibiotics, respectively. Additionally, growth rates (E. coli & L. monocytogenes), and cell density (L. monocytogenes) significantly increased. An increase of sensitivity to radiation - possibly through the radiation-mediated increase of cell wall permeability, followed by a decrease of sensitivity, and

then the development of resistance to antibiotics was observed. The authors attribute this sequence of events to the activation of the bacterial defense mechanisms after reaching a certain damage threshold. The responses were frequency-dependent.

Plants

Chen et al. (2009) examined the response of wheat seeds to 2.45 GHz radiation, reporting the development of tolerance to salt stress through mechanisms that involve the stimulation of the antioxidant defence system (NO production, enzymatic and non-enzymatic antioxidants), that could prove beneficial for agriculture.

Soran et al. (2014) comparing the effects of 900 GHz with those of 2.45 GHz radiation, concluded that signs of stress responses were demonstrated under both frequencies (significantly reduced water transport, upregulation of the release of green leaf volatile compounds – especially monoterpenes). Anatomical changes (thickness of cell walls, length of chloroplasts, size of mitochondria significantly reduced) occurred under both stressors, however with more pronounces changes under 2.45 GHz. Additionally, the essential oils secretion was increased under 900 GHz irradiation, but inhibited under 2.45 GHz irradiation. Overall, the effects of 2.45 GHz radiation were more severe.

Mechanism of Action

The core mechanism of action of exposure to non-ionizing radiation (including 2.45 GHz frequency) has been established to be oxidative cell stress by the vast majority of the corresponding studies. In their review paper of 100 scientific papers, Yakumenko et al. (2016) found that in 93% of them, oxidative stress in the forms of intracellular ROS, free radicals, and oxidative DNA damage in biological systems (in-vitro, in plants, animals, humans) was the main mechanism of damage. Even though oxidative processes such as the formation of free radicals and ROS normally develop in all cells as a result of metabolism, cells possess the ability to counteract and neutralise its effects by secreting antioxidants. When external factors disturb the balance between oxidative processes and the release of antioxidant molecules, they cause the initiation of apoptosis, inflammatory cellular damage (mitochondria, ion channels, cell membranes, electron transport chain, proteins, etc), DNA damage, damage on Ca^{2+} dependent signal pathways, disturbance of the protein conformation, the initiation and promotion of tumorigenesis, and tissue and organ fibrosis (in the long term). Also, the effect of the interaction of free radicals with cellular components can cause the formation of secondary radicals, thus initiating and maintaining a chain reaction.

On top of the overproduction of free radicals and ROS, microwave radiation was also found to suppress antioxidant enzyme activity, further shifting the balance towards oxidation (Kumari et al., 2012). Oral intake, or in the case of experimental studies otherwise administered antioxidant substances such as green tea catechins, selenium, vitamins A, C, E, beta carotene, beta-glucan, L-carnitine, melatonin, and olive leaf extract, has proven to be protective in diverse tissue samples (mucus membranes, skin, cardiac tissue, sperm cells, testicular tissue, blood, liver, kidney, eye lens) in numerous studies, due to their free-radical scavenging activity (Aweda et al., 2003; Aynali et al., 2013; Ceyhan et al., 2012; Gümral/Naziroğlu, 2009; Kim/ Rhee, 2004; Türker et al., 2011; Meena et al., 2014; Oksay et al., 2014; Salah et al., 2013; Tök/Naziroğlu, 2014).

Table 1: Summary table of the studies included in the Review, featuring the targeted organ or function and the number and brief information of the studies that correspond to each. Note that some of studies apply to more than one organ or function.

Targeted organ/ function	№	Studies (First author & Year of Publication)
Apoptosis	10	Ballardin 2011, Cig 2015, Deshmuk 2013, Kumar 2011, Margaritis 2014, Meena 2014, Misa-Agustiño 2015, Shahin 2015, Shokri 2015, Zhu 2016
Calcium ion channels (Ca^{2+})	8	Cig 2015, Hassanshahi 2017, Kesari 2012, Nazıroğlu 2012c & 2012a, Panagopoulos 2015, Taheri 2015 & 2017
Cancer	5	Czerska 1992, Eser 2013, Sarkar 1994, Szmigielski 1982, Yang 2010
Cell growth and cycle	4	Ballardin 2011, Cleary 1996,Nazıroğlu 2012b, Orendáčová 2009

DNA damage	24	Akdag 2016, Avendaño 2012, Chartuvedi 2011, Czerska 1992, Deshmuk 2013 & 2015, Gürler 2014, Kesari 2010a, 2010b, 2012, Lai 1996 &1997, Lai/ Singh 1995 &1996 & 1997 & 2005, Meena 2014, Megha 2015, Paulraj/Behari 2006, Sakar 1994, Shahin 2013, Taheri 2015 & 2017, Zotti-Martelli 2000
EEG / brain development	12	Chauhan 2017, Maganioti 2010, Nazıroğlu /Gümral 2009, Othmann 2017, Papageorgiou 2011, Paulraj/Behari 2006, Sinha 2008, Testylier 2002, von Klitzing 1995 & 2016, Yang 2010, Yang 2012
Embryo / pregnanc y / reproductive capacity	8	Cleary 1996, Margaritis 2014, Nakamura 2000, Özorak 2013, Othmann 2017, Sangün 2015, Shahin 2013, Yüksel 2016
Gene / gene expression	5	Kesari 2010b, Lee 2005, Orendáčová 2011, Yang 2010, Yang 2012
Glucose metabolism	1	Salah 2013
Heart	5	Kim/Rhee 2004, Saili 2015, von Klitzing 2014 & 2016, Zhu 2016
Heat shock proteins (HSP)	4	Deshmuk 2015, Misa-Agustiño 2012 & 2015, Yang 2012
Hormones	2	Shahin 2013, Yüksel 2016
Kidneys	7	Akdag 2016, 2016, Chauhan 2017, Özorak 2013, Özorak/ Nazıroğlu 2013, Shahin 2013, Shahin 2014
Liver	4	Chauhan 2017, Holovska 2015, Kumari 2012, Salah 2013
Memory / learning / behavior	22	Cammaerts/Johannson 2014, Chartuvedi 2011, Deshmuk 2015, Hassanshahi 2017, Lai 2004 & 1987a & 1987b & 1988 & 1989a & 1989b & 1991 & 1994 & 1996b, Li 2008, Othmann 2017, Orendáčová 2011, Paulraj 2006a, Sangün 2015, Shahin 2015, Sinha 2008, Thomas 1980, Wang/Lai 2000
Neurotransmitters	2	Aggarwal 2013, Lai 1996
Oxidative stress in cells	41	Atasoy 2013, Aweda 2003, Aynali 2013, Ceyhan 2012, Chauhan 2017, Chen 2009, Cig 2015, Deshmuk 2013 & 2015, Eser 2013, Gümral 2009, Gürler 2014, Kesari 2010a, 2010b, 2012, Kim/Rhee 2004, Kumar 2011, Kumari 2012, Lai/Singh 1994, Meena 2014, Megha 2015, Misa-Agustiño 2015, Nazıroğlu /Gümral 2009, Nazıroğlu 2012a & 2012b & 2012c, Oksay 2014, Othmann 2017, Özorak 2013, Saili 2015, Salah 2013, Sangün 2015, Shahin 2013 & 2014 & 2015, Soran 2014, Tök/ Nazıroğlu 2014, Türker 2011, Yakymenko 2016, Yüksel 2016, Zhu 2016
Sperm / testes	10	Akdag 2016, Atasoy 2013, Avendaño 2012, Dasdag 2015, Meena 2014, Oksay 2014, Özorak 2013, Sarkar 1994, Shahin 2014, Shokri 2015
Spleen	1	Chauhan 2017
Testosterone	1	Meena 2014
Thyroid glands	3	Misa-Agustiño 2013 & 2015, Sinha 2008

Conclusions

The report evaluates this range and concludes that based on the extensive body of research and the positive demonstration of adverse health and behavioral effects by the majority of the studies, it is recommended that ALARA measures are applied, especially for the protection of particularly vulnerable groups such as newborns, children and adolescents. Radiation of 2.45 GHz is a potent trigger of oxidative stress, that is the main source of implications, as was demonstrated, and can affect multiple organs and processes through large chemical chain reactions, either directly or indirectly. Various enforceable measures and compounds displayed protective efficacy against the effects of 2.45 MHz EMF/RF, yet the current official exposure limits do not correspond with research recommended limits.

Limitations of the Review Study

The author highlights that while the examined studies confirm potential health impacts, the very limited amount of long-term and epidemiological studies, as well as studies on human subjects currently available disallows the disclosure of reliable figures.

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