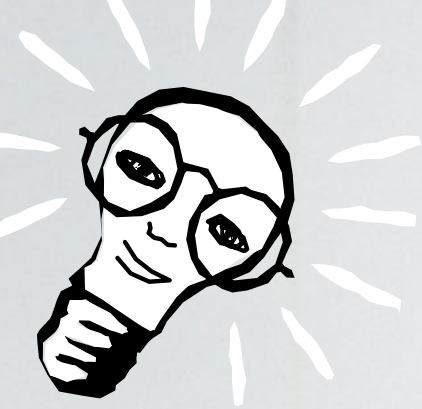




OVERVIEW

for a clearer picture

No. 10

**Does mobile phone radiation
affect the development of
embryos and babies?**

Overview No. 10

Does mobile phone radiation affect the development of embryos and babies?

As of: 29 April 2026

Summary

The diagnose:funk publication series *"OVERVIEW for a clearer picture"* reports on the state of research into the health effects of non-ionising radiation from mobile communications. The scientific literature search draws on the EMFdata.org and EMF-Portal.de databases.

Using smartphones, tablets and Wi-Fi can harm fertility in both men and women. In this Overview, we document over 90 studies and eight reviews on prenatal effects on egg-cell formation and embryonic development, and the consequences for babies. This evaluation of studies is a risk dossier, rather than a review. Prenatal exposure to wireless radiation can cause developmental disorders, oxidative cellular stress, metabolic disorders and structural tissue changes with postnatal effects. These possible effects range from neurobiological consequences, behavioural problems and delayed growth to cancer. This Overview shows that existing studies are not adequately incorporated into official risk communication. Health authorities should therefore consistently apply the precautionary principle and adopt regulatory measures to avert hazards. By informing themselves about the studies, medical professionals and midwives can advise parents before and after their child's birth about using wireless devices (e.g. smartphones, smartwatches, tablets, Wi-Fi and Bluetooth). This is how we protect children from the effects of wireless radiation.

You are welcome to share this Overview online (websites, social media) if you inform diagnose:funk beforehand and acknowledge the source.

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1 Particular vulnerability during pregnancy

Pregnancy involves highly sensitive developmental processes in mother and child that are especially susceptible to disruption. At the same time, using smartphones, smartwatches and tablets is part of everyday life for many pregnant women. Wi-Fi is always on and available everywhere; Bluetooth headphones are convenient and widespread. It is therefore all the more important to assess reliably whether, and to what extent, this radiation - more precisely, radiofrequency electromagnetic fields - may pose a risk.

Many studies have appeared in recent years, and a substantial number show very clearly that biological effects exist which must not be ignored. For this Overview No. 10, we have therefore specifically compiled studies demonstrating such effects, to make visible the risks described by research.



Photo: Pexels / Amina Filkins

Health-conscious women avoid alcohol, smoking, caffeine and foods contaminated with pesticides or mercury during pregnancy. Yet many pregnant women use smartphones without concern in everyday life. They are thus exposed to mobile phone radiation: non-ionising radiation currently in the 0.7 to 6 GHz range. The electromagnetic radiation emitted by mobile phones - more precisely, radiofrequency electromagnetic fields (RF-EMF) - is associated with many biological effects.

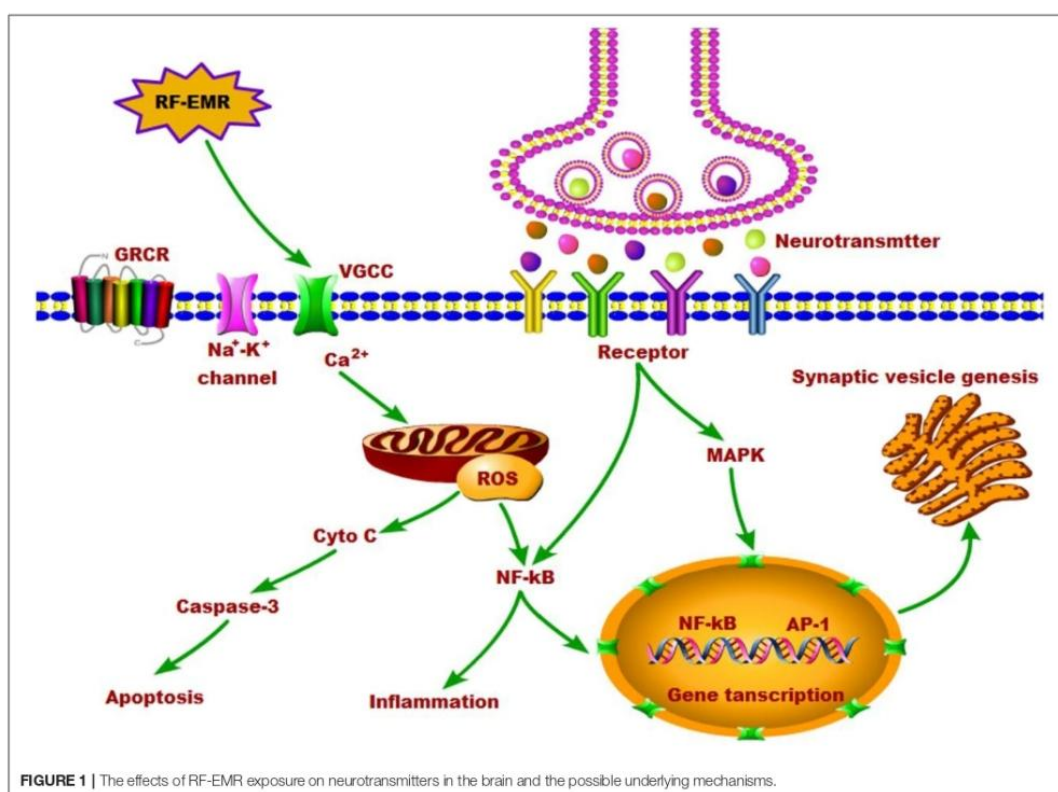
Numerous animal and cell studies show that prenatal exposure causes developmental disorders, oxidative stress, metabolic disorders and structural tissue changes with postnatal effects such as behavioural problems or delayed growth. Medical professionals, midwives and expectant parents should keep up to date with the research to make prudent decisions about recommendations for mobile-phone use. Pregnant women need information.

Vienna professors Kundi and Hutter (environmental medicine, Medical University of Vienna) published *"Assessing the Hazards of Children's Exposure to Electric, Magnetic and Electromagnetic Fields"* on RF-EMF effects and smartphone use by pregnant women. They write that *"adverse health effects not only cannot be ruled out, but the evidence for such effects is increasing"* (umwelt-medizin-gesellschaft, 3/2019).

1.1 Early radiation exposure leads to behavioural problems

This issue was already widely discussed in science in 2008 after the studies by Divan et al. appeared. With US health authorities' involvement (NIEHS/NIH, National Institute of Environmental Health Sciences / National Institutes of Health), two large epidemiological investigations were conducted in Denmark in 2008 and 2010. The first, entitled *"Prenatal and Postnatal Exposure to Cell Phone Use and Behavioral Problems in Children"*, included 13,159 children aged seven. It showed that prenatal and postnatal mobile-phone exposure was associated with general behavioural problems, including ADHD symptoms, emotional problems and problems with peers (Divan et al. 2008). The findings were confirmed in a new group of 28,745 children; this replication study was completed in 2010 and published in 2012 (Divan et al. 2012). The highest risk of behavioural problems was observed in children exposed both before and after birth. These epidemiological studies demonstrated correlations whose causality is confirmed by subsequent biological and medical studies.

1.2 Biological causes have been investigated

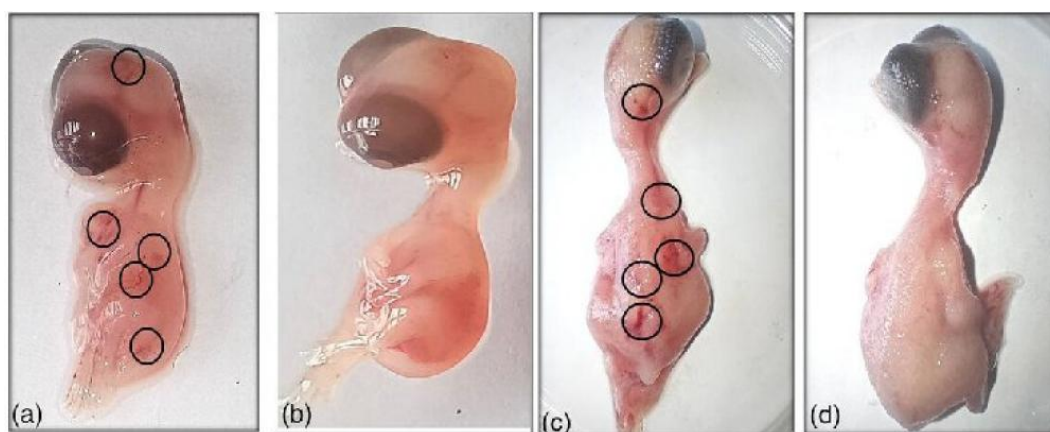


Non-thermal mechanisms of non-ionising radiation and their cascades of harm.
Graphic: Hu et al. (2021).

Approximately 90 peer-reviewed studies and eight reviews address different biological and medical endpoints. Kashani et al. (2023) examined what the overall evidence says and with what strength of evidence in *"Electromagnetic fields exposure on fetal and childhood abnormalities: Systematic review and meta-analysis"*.

This review addresses the effects of increasing electromagnetic-field exposure on unborn and young children. It focused on malformations and chromosomal abnormalities originating before birth. The 14 studies recorded and analysed 85.4154 individuals. When mothers were exposed to higher field strengths, particularly in the first trimester, compared with mothers without field exposure, increased oxidative stress, altered gene expression, DNA damage and more embryonic abnormalities were detected in umbilical-cord blood. Because stem cells are more sensitive to ionising and non-ionising radiation, more cancer cells and developmental disorders, such as language problems, may also occur in childhood. The analysis also found that defects / abnormalities were more common in fetuses and children whose parents were exposed to electromagnetic fields of greater intensity.

Islam et al. (2023) studied the effects of 4G mobile communications (2100 MHz) on embryonic development in chicks, a model organism. 4G exposure caused growth disorders, abnormalities of central organs and changes in gene expression in the immune and vascular systems (diagnose-funk.org/2024).



The figure shows chicken embryos from the 4G-exposed group (a and c) with subcutaneous haemorrhages on days 7 and 10, respectively, marked by circles. The control group (b and d) showed no signs of haemorrhage. (Graphic: Islam 2023; source: <https://onlinelibrary.wiley.com/doi/10.1002/vms3.1273>)

The research, comprising animal experiments, cell research, epidemiological investigations and reviews, shows that prenatal RF-EMF exposure affects biological processes central to embryonic development. The findings in chicken embryos are therefore not isolated cases.

1.3 Seven documented major forms of harm

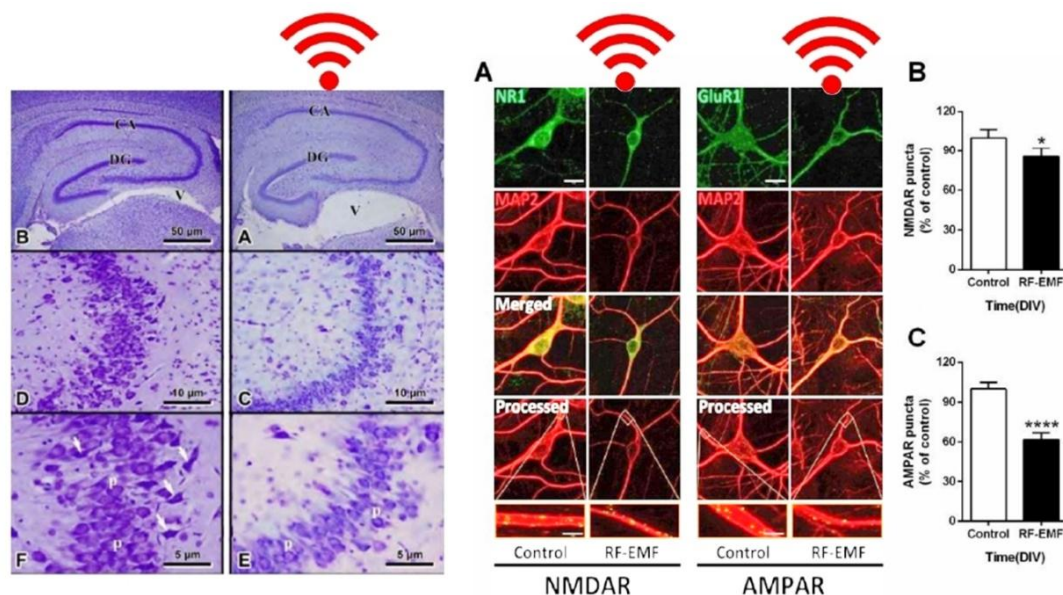
These worrying effects can be grouped into seven outcomes repeatedly reproduced in the literature. Many studies demonstrate several forms of harm and their interactions. The text refers to studies reviewed in ElektrosmogReport (emfdata.org/de/elektrosmogreport) and on EMF:data (emfdata.org); further studies appear in the appendix table. Without diagnose:funk's research work, these 90 studies would lie dormant in databases and no institution would report on them. Our documentation shows that a review by independent scientists is overdue.

1.3.1 Central mechanism: oxidative cellular stress

Oxidative cellular stress causes inflammatory diseases. Numerous experimental studies describe RF-EMF-induced overproduction of reactive oxygen species (ROS, free radicals) alongside weakened antioxidant systems (Desai 2009; Kesari 2012; Burlaka 2013; Özorak 2013; Shahin 2013). This mechanism occurs in embryos, testes and ovaries, and is regarded as a starting point for further defects such as inflammation, mitochondrial stress and cellular dysfunction. Recent studies confirm oxidative stress in ovaries and the brain after prenatal exposure (Salameh 2022; Jha 2025), forming a consistent biological model. Reviews by Yakymenko (2016) and Schuermann / Mevissen (2021) emphasise its importance. Our 2020 foundational article explains the possible consequences of oxidative cellular stress as an established mechanism (diagnose-funk.org/1550).

1.3.2 Direct genetic damage and disrupted cellular regulation

DNA strand breaks, a precursor to genetic alterations and cancer, have repeatedly been demonstrated in embryos and germ cells (Panagopoulos 2007, 2012; De Iuliis 2009; Chavdoula 2010; Hanci 2013; Gorpichenko 2014). Newer studies also show changes in gene expression affecting protein formation and disturbances in neuronal stem-cell differentiation (Islam 2023; Bodin 2025). These findings are especially critical because genetic damage in early development can have long-term structural and functional consequences.



Left figure: control (left column), exposure (right column): fewer granule cells and morphological abnormalities in hippocampal pyramidal cells (Odaci et al. 2008).

Right figure: hippocampal neurons showing NMDAR (NR1, green) and AMPAR (GluR1, green), with MAP2 (marker, red). RF-EMF exposure reduced NMDAR and AMPAR expression (A, B, C) (Kim et al. 2021).

1.3.3 Disruption of key developmental processes: cell division (cell proliferation) and cell death (apoptosis)

Studies report both increased apoptotic processes (Hanci 2013; Hou 2015; Umur 2013) and reduced cell division and neuronal development (Kim 2024; Bodin 2025). This combination - increased cell death alongside reduced formation of new cells - indicates fundamental dysregulation of embryonic development, especially in the nervous system.

1.3.4 Ovaries are damaged

Girls are born with all their egg cells already present. These remain dormant in the ovaries from birth to puberty. The egg cells may therefore be continuously exposed to radiation from the time they form. Puberty begins with menstruation. The egg that is released may thus already have a history of radiation exposure. Greek research groups in particular - Magras & Xenos (1997), Chavdoula (2010), Panagopoulos (2004, 2007, 2009, 2012) and Margaritis (2014) - investigated the highly sensitive stages of egg maturation (oogenesis).

The Greek group led by Panagopoulos et al. provides important clues about the mechanism of harm during oogenesis in *"Cell Death Induced by GSM 900 MHz and DCS 1800 MHz Mobile Telephony Radiation"*. The experiments used flies. The main finding: *"Cell death was detected during all developmental stages of early and middle oogenesis, from the germarium to stage 10, and in all types of egg-chamber cells (nurse cells, follicle cells, oocytes). The germarium and stages 7-8 were found to be the developmental stages most sensitive to electromagnetic stress induced by GSM and DCS fields, with the germarium even more sensitive than stages 7-8"* (Panagopoulos et al. 2007, EMF-Portal).

The fruit fly *Drosophila*'s genes are remarkably similar to human genes. It therefore serves as a model for studying human embryonic development and many diseases. Combining Panagopoulos's findings on cell death during egg maturation in fruit flies with Magras's findings on embryonic exposure demonstrates microwave radiation's enormous potential for harm (mobile phone radiation belongs to the microwave range). Professor I. Magras's group (Aristotle University of Thessaloniki, Greece) reported as early as 1997 (Magras 2008):

"Over the past twenty(!) years, comparative experiments have been conducted on different laboratory animals (mice, rats, quail and chickens) to document their embryos' sensitivity to radiofrequency radiation. ... Twelve pairs of mice were placed in this field experiment at locations with different radiation levels (168-1053 nW/cm²), allowing five successive matings to be studied. ... The number of newborns per mother declined continuously, and ultimately the loss of fertility was irreversible. These results clearly indicated that radiofrequency radiation sources can have embryotoxic effects. ... The high sensitivity of mouse, rat, chicken and quail embryos to weak microwave radiation clearly indicates similarly high sensitivity in embryos of higher mammals, including humans, because their early prenatal developmental stages are alike."

1.3.5 Structural and functional organ damage and clinically relevant developmental disorders

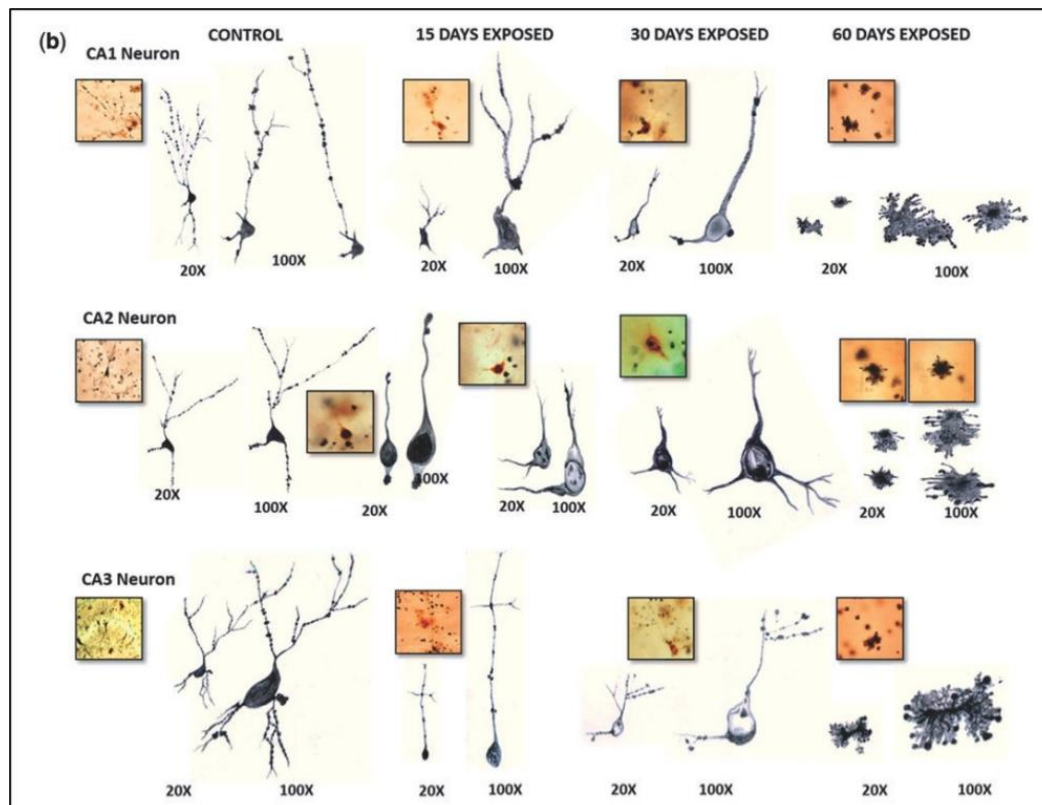
These include changes in the brain (Tan 2022; Kim 2024), heart muscle (Bozok 2023), liver and testes (Holovská 2021; Andrašková 2022), as well as growth disorders and lower birth weight (Boileau 2020; Bodin 2024). Developmental delays in infants' communication, motor skills and problem-solving have also been observed, depending on household radiation exposure levels (Setia 2025). These findings link molecular observations with observable clinical effects.

1.3.6 Brain regions for learning and memory are especially sensitive to mobile phone radiation

The brain relies on finely coordinated electrochemical and electromagnetic processes, especially in the hippocampus, which is central to memory formation and learning. Studies show that electromagnetic fields can disrupt these processes by impairing neuronal rhythms, synapse formation and memory (long-term potentiation) (Hoffmann 2001; Hu 2021; Tan 2022). Experimental studies demonstrate reduced hippocampal neurogenesis and changes in key mechanisms of the Hebbian learning synapse. Important factors in brain development, learning and memory (long-term potentiation, LPT) are inhibited, including NMDA glutamate receptors and calcium signalling pathways. BDNF brain-growth-factor expression, synaptic plasticity and dendritic connections are reduced (Kim 2021, 2024; Bodin 2025). These changes cause measurable cognitive deficits, particularly in learning and memory.

Animal studies demonstrate impaired learning and memory and increased neuronal degeneration under Wi-Fi exposure (Shahin 2015, 2018). Human epidemiological investigations show associations between radiation exposure, memory performance and behavioural problems (Divan 2008, 2012; Foerster 2018; Setia 2025).

At the neurochemical level, reviews indicate that RF-EMF alters neurotransmitter systems such as dopamine, serotonin and glutamate, directly affecting regulation of learning, memory and behaviour (Hu 2021; Chatha 2026). Particularly concerning is that these effects may be amplified in the developing brain because neuronal networks and plasticity systems are not yet stable. Our Overview No. 4, *"Does Mobile Communication Affect the Brain?"*, addresses these connections (diagnose-funk.org/2268).



Degeneration of brain cells through Wi-Fi exposure: structure and shape of hippocampal neurons in irradiated mice after EMF exposure. "The number of dendrites decreased significantly over time in all exposed groups of mice." "The number of dying or degenerating neurons increased in mice exposed to 2.45 GHz for 30 and 60 days." "Note the neuronal clumping or congestion in mice exposed to 2.45 GHz for 60 days." (Text and graphic: Shahin et al. 2015; German translation: diagnose:funk.)

1.3.7 Long-term and intergenerational effects

Prenatal exposure can cause damage persisting into adulthood, such as impaired spermatogenesis and structural changes in the testes (Gelenli Dolanbay 2025). Animal models also show transgenerational effects on offspring's development and behaviour (Mehta 2025).

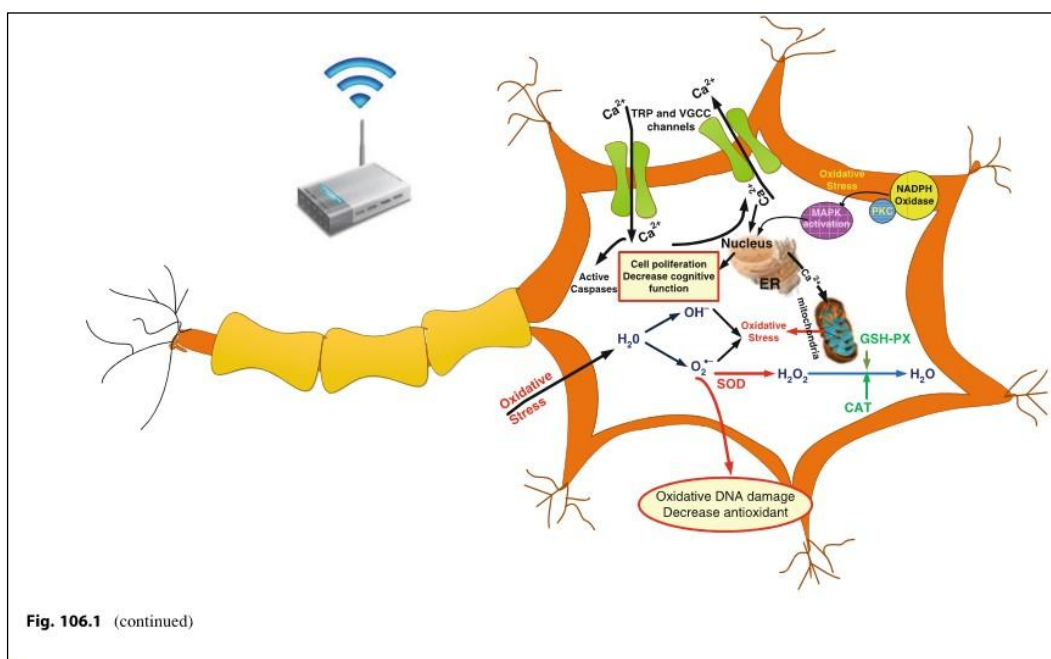
2 Biological and pathological effects of 2.45 GHz radiation (Wi-Fi) on cells, fertility, the brain and behaviour

From the study by Isabel Wilke (2018)

Wi-Fi is one of the most widely used frequencies: routers stand in homes, and hotspots are found in every hotel and restaurant, even in hospitals. In 2014, Nazıroğlu / Akman published a review in a Springer reference book demonstrating Wi-Fi's damaging effects on cells. Isabel Wilke's comprehensive analysis, "Biological and Pathological Effects of 2.45 GHz Radiation

on Cells, Fertility, the Brain and Behaviour”, appeared in umwelt-medizin-gesellschaft (1/2018). She wrote about effects on pregnant women:

“The internationally renowned Karolinska Institute in Stockholm issued a press release on 3 February 2011 warning against wireless applications (mobile and cordless phones, Wi-Fi) because they pose hazards to children and pregnant women. Exposure limits must be lowered: ‘Current US and ICNIRP standards for radiofrequency and microwave radiation from wireless technologies are entirely inadequate. They never were intended to address the kind of exposures from wireless devices that now affect over 4 billion people.’”



Graphic from Nazıroğlu / Akman's review (2014) on oxidative cellular stress as a mechanism, published in a Springer reference book.

Margaritis et al. (2014) investigated egg development in the ovaries of two *Drosophila* strains (fruit flies) exposed to electromagnetic fields at different frequencies, and whether *Drosophila* is suitable as a test system (biomarker). They examined reproductive capacity (fecundity) and apoptosis using two tests during oogenesis, when apoptosis must normally occur at particular stages. They also studied whether pulsed and continuous radiation produce different biological effects. Results are presented for microwave ovens, Bluetooth (lowest field strength 0.3 V/m, equivalent to 238.7 $\mu\text{W}/\text{m}^2$ during the first seven days) and Wi-Fi (2.44 GHz, 2.1 V/m, equivalent to approximately 11,698 $\mu\text{W}/\text{m}^2$, pulsed at 10 Hz with a 1 ms pulse duration). Controls were untreated and sham-exposed animals. All types of radiation, including Bluetooth's weak fields and mobile phones' fields up to 22 V/m (approximately 1.3 W/m^2), showed statistically significant increases in apoptosis. At almost every frequency, fecundity fell by between 10% (Wi-Fi and Bluetooth) and 30% (mobile and DECT phones). Radiation appears to affect still-unknown mechanisms at the transitions between egg-development stages.

The number of pupae among the offspring was significantly reduced. The authors suggest that pulses, rather than high field intensities, are responsible, since some devices do not have high intensities. Bluetooth, for example, despite its very low field strength, caused the same damage to apoptosis and reproduction as the unpulsed signals of an FM generator with 43 times greater intensity, 13 V/m (approximately 448 mW/m²).

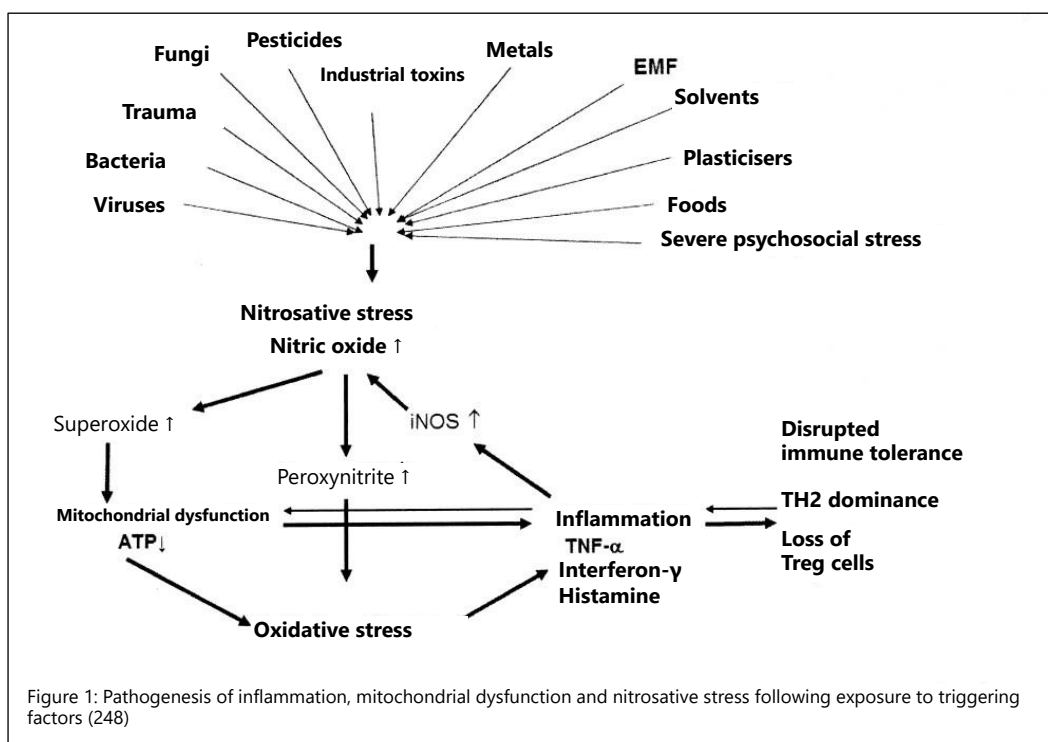
Özorak / Nazıroğlu (2013) investigated male offspring of female rats irradiated during pregnancy at 2450, 900 and 1800 MHz (one hour/day, five days/week, and for six weeks after birth at 120 mW/m²: 10 V/m; whole-body SAR 0.01-1.2 W/kg, mean 0.18 ± 0.07 W/kg). The double-blind study examined kidneys and testes for trace elements (chromium, copper, iron, manganese, selenium and zinc, which act as cofactors in antioxidant enzymes). It then measured lipid-peroxidation indicators of oxidative stress, antioxidant glutathione and glutathione peroxidase, and vitamins A and E and β -carotene in kidney and testicular tissue of offspring aged four, five and six weeks. Four-week-old animals were more sensitive to radiation than those aged five or six weeks. In six-week-old animals, oxidative stress produced significant differences from controls in the kidneys only for TAS, lipid peroxidation, copper and iron. The authors conclude that 900, 1800 and 2450 MHz can cause oxidative stress in young rats' kidneys and testes, evident in increased lipid peroxidation and oxidisable iron, and reduced trace elements, TAS and glutathione in developing animals, comparable to human puberty.

Shahin et al. (2013) studied female mice exposed to 2.45 GHz (non-thermal power density 335.49 mW/m², SAR 23.023 mW/kg, two hours/day for 45 days). After 45 days, blood and liver, kidney and ovarian tissue homogenates were collected to determine blood-cell parameters (erythrocytes, leukocytes, neutrophil-to-lymphocyte ratio, haemoglobin), DNA breaks, NO, NO₂/NO₃ (nitrite/nitrate), progesterone (P4), estradiol (E2), ROS, and the antioxidant enzymes superoxide dismutase (SOD), catalase and glutathione peroxidase (GPx). Irradiated animals showed significantly increased ROS, haemoglobin, total erythrocytes and leukocytes, DNA strand breaks and hormones (plasma P4 and E2 rose relative to controls, but only E2 significantly), and significantly reduced NO and antioxidant-enzyme activity in all three organs. Low-field-strength microwave radiation caused physiological stress in pregnant mice and embryo death. Enlarged follicles were seen in the ovaries, significantly fewer embryos were present and development lagged. The authors attribute the harm to oxidative stress (ROS impair antioxidant defences and can cause apoptosis), altered progesterone and estradiol concentrations, and DNA strand breaks. Altered leukocyte counts indicate inflammation. At this high intensity, 2.45 GHz radiation is a strong trigger of oxidative stress.

Sangün et al. (2015), in the first longitudinal study of this radiation, examined the effects of long-term Wi-Fi exposure (2.45 GHz) on young female rats' growth and development. During embryogenesis, the most sensitive period, serious harm can arise from external exposure to chemicals or

radiation. Young females underwent sham, prenatal or postnatal exposure (2.45 MHz radiation, one hour daily until puberty at 45.5 V/m, approximately 5.5 W/m² in the near field; whole-body SAR 143 mW/kg). At puberty, serum, ovarian and brain tissue were collected and oxidative / antioxidant status determined. Chronic oxidative stress was observed in both organs. Serum concentrations of follicle-stimulating hormone (FSH), luteinising hormone (LH), 17 β -estradiol (E2) and insulin-like growth factor 1 (IGF-1) were measured. Only serum LH rose significantly after prenatal and postnatal exposure compared with sham controls. Histological analyses of the hypothalamus and ovaries identified cellular and tissue changes; no significant differences were found. Prenatal 2.45 GHz exposure restricted growth and delayed puberty in female rats. Radiation intensities were within international limits. Chronic Wi-Fi exposure may harm growth and puberty, particularly during the intrauterine phase and early childhood. The authors recommend precautionary measures, especially near such radiation sources and during long-term exposure.

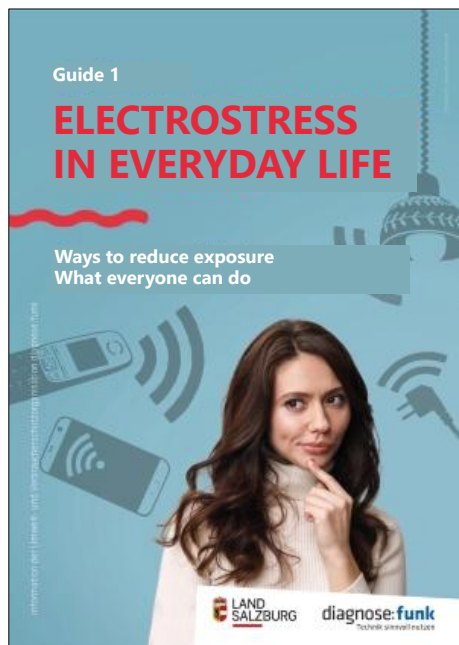
Yüksel / Nazıroğlu et al. (2016) irradiated female rats for one year at 900, 1800 and 2450 MHz, then examined mothers and offspring for hormone status and oxidative stress (described in section 6.1 on oxidative stress). *"The offspring aged four, five and six weeks generally showed stronger effects than the mothers, with particularly marked differences at 2.45 GHz."*



Schematic showing how toxic substances trigger cellular cascades leading to oxidative cellular stress and subsequently inflammatory diseases (from umwelt-medizin-gesellschaft, 2012; von Baehr).

Further Wi-Fi studies on embryotoxicity have since appeared and been reviewed on EMF:data and in ElektromogReport, including Holovská et al. (2021), Andrásková et al. (2022), Tahir et al. (2024), Tan et al. (2022), Popovičová et al. (2024) and Cakir et al. (2025). They are described in the tabular study overview.

2.1 Distance is your friend!



Health authorities should widely communicate these findings on embryotoxicity, especially for widely used Wi-Fi in nurseries, schools, hospitals and maternity wards. Unfortunately, this does not happen. Even Telekom warns in a router manual:

"Radio signals: Your Speedport's integrated antennas transmit and receive radio signals, for example to provide Wi-Fi. Avoid placing your Speedport in the immediate vicinity of bedrooms, children's rooms and living areas, to keep exposure to electromagnetic fields as low as possible" (2017).

Many users "love" their smartphones so much that they refuse to acknowledge these risks. Protection is possible.

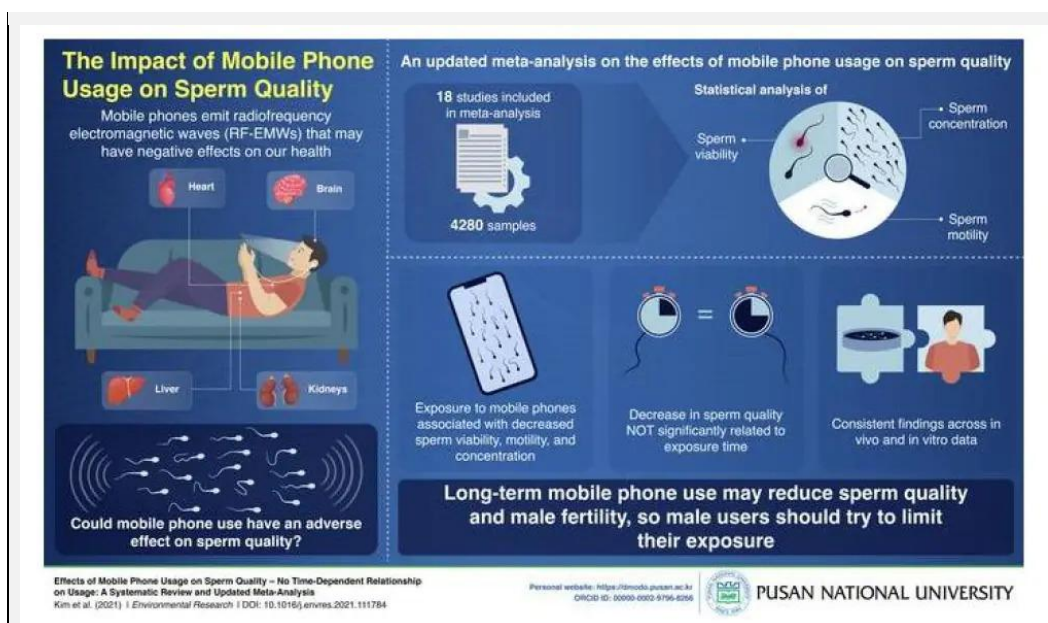
Pregnant women can protect themselves by strictly avoiding use. Doctors and midwives must inform them about this. Otherwise, the rule is: learn about all measures to minimise radiation, following the principle *"Distance is your friend!"* diagnose:funk's Guide 1, *"Electrostress in Everyday Life"*, offers many tips (diagnose-funk.org/ratgeber1). It was written jointly by Salzburg's Health Directorate and diagnose:funk.



These leaflets provide brief, clear information on the health risks of wireless radiation. They are well suited for display in medical practices or pharmacies, for example.

Midwives and doctors can request these leaflets free of charge through our online shop:
shop.diagnose-funk.org/Funkpause-Flyer
shop.diagnose-funk.org/Tote-Hose-Flyer

2.2 Effects on male fertility



Main findings of Kim et al.'s 2021 meta-analysis: a clear association between mobile phones and sperm damage. Mobile-phone use reduced sperm count and quality. Sperm were less motile and viable. Graphic: Pusan National University, Kim et al. 2021.

Levine et al.'s meta-analysis (2022) found that between 1973 and 2018, average sperm concentration worldwide fell by 51.6% and total sperm count per ejaculate by 62.3%. In more recent decades, the decline was even steeper, with multiple causes cited. One cause is radiation exposure, especially from carrying smartphones in trouser pockets. In 2016, diagnose:funk published a research assessment in its Focus report "130 Study Findings: Smartphones & Tablets Harm Testes, Sperm and Embryos" (<https://kurzlinks.de/0bsn>). In 2019, Mutter / Hensinger published "Declining Sperm Quality: Environmental-Medical Causes" in Zeitschrift für Komplementärmedizin (Thieme). Two extensive reviews and a large epidemiological study on sperm effects are now available. We limit the following discussion to three key works.

- Pusan National University's 2021 meta-analysis (review), Korea, on mobile phones' effects on sperm quality concludes: "The results indicate that mobile-phone use leads to reduced sperm motility, viability and concentration." Because findings were consistent both in vivo and in vitro (cultured sperm), Dr Kim warns: "Male mobile-phone users should try to reduce use to protect their sperm quality" (Kim et al. 2021).
- "Health Impact of 5G" (2022) was published by the European Parliament's technology-assessment panel, STOA. Led by the renowned cancer-research Ramazzini Institute, a team of scientists documents, across 200

pages, all scientific studies published since 1945 on mobile phone radiation and cancer or reduced fertility. The conclusion: mobile phone radiation is probably carcinogenic, clearly harmful to male fertility and possibly harmful to embryos in women.

- The largest epidemiological study to date in soldiers, conducted in Switzerland, confirms laboratory and animal findings. Soldiers using mobile phones more than 20 times daily had significantly lower average sperm concentrations than soldiers using them no more than once weekly (Rahban 2023; see diagnose-funk.org/2020).

2.3 Is wireless radiation dangerous?

What the Federal Office for Radiation Protection says:

Issue 8/26 of the women's magazine *Freundin* asked experts about various health topics, including the Federal Office for Radiation Protection (BfS) about carrying a mobile phone in a trouser pocket:

"Does a phone in your trouser pocket affect fertility? NO. Even at maximum transmission power, phones must comply with limits protecting against all health-relevant effects," the BfS explains. *"This also applies when it remains close to the body, such as in a trouser pocket."* The device's CE mark is the manufacturer's declaration that these limits are met.

"According to current knowledge, mobile-phone radiation is harmless, contrary to what many think. Phones transmit calls and data using electromagnetic fields, as does Wi-Fi. This radiation can only warm our bodies, and the limits protect against that. Sleep is not negatively affected by phones either. From the BfS's perspective, there is therefore no need to keep a phone outside the bedroom. Psychologically, however, a device on the bedside table can certainly disturb sleep: constant notifications and the feeling of being reachable create subconscious stress and prevent restful nights."

What science says:

The review *"Effects of Mobile Phone Use During Pregnancy on Fetal Development and Birth Defects: A Review"* (Chatha et al. 2026) summarises the evidence as follows:

"The literature reports that electromagnetic radiation (EMR) exposure is associated with genetic changes through altered DNA methylation and histone modification. These changes can disrupt gene-expression patterns important for fetal development. They may have long-term consequences, including predisposition to developmental disorders or chronic disease. At the cellular level, EMF exposure has been associated with altered calcium signalling, mitochondrial dysfunction and DNA strand breaks. These effects are especially concerning during embryogenesis, a phase characterised by rapid cell division, cells' migration to their destinations

in the body and their subsequent differentiation. Epidemiological evidence suggests EMF exposure may affect pregnancy outcomes. Studies of mobile-phone use during pregnancy and adverse outcomes have found interesting patterns: • Low birth weight (LBW) ... • Premature birth: studies such as Megha et al. (2022) ... • Congenital anomalies ... • Maternal mobile-phone use has been associated with behavioural abnormalities and cognitive impairment in offspring ..."

3 The dispute over interpretation

Why are girls, boys and parents not informed on the basis of this research? The Federal Office for Radiation Protection (BfS) objects that this is unnecessary: causality criteria have not been met, many studies report no findings, and overall too much uncertainty remains. Communicating risks now would be alarmism. The BfS claims no risks have been demonstrated below exposure limits, and excludes studies demonstrating non-thermal effects from its risk assessment. Our Overview No. 3, *"Does Mobile Communication Also Have Non-Thermal Effects?"*, addresses this position (diagnose-funk.org/2034).

3.1 Assessing the overall evidence

Embryotoxicity studies show remarkable consistency across different models: oxidative stress, DNA damage, disrupted cellular processes and neuronal dysfunction repeatedly occur together, forming a plausible biological mechanism. Existing research reveals a systemic pattern rather than an isolated phenomenon: mobile phone radiation interferes with key regulatory processes in embryonic and brain development. Whether this constitutes definitive *"proof"* depends on the scientific standard. Under the precautionary principle, however, the data are consistent enough to describe a biologically plausible, repeatedly demonstrated risk potential requiring protective measures.

The argument that these are mainly animal studies whose findings cannot be transferred to humans is absurd. Dr Ulrich Warnke comments in an interview: *"Animal studies are the backbone of medical risk assessment. Without them, there would be no drug approval and no environmental or consumer protection. Basic biological mechanisms are highly comparable in humans and animals. Dismissing animal studies here abandons scientific logic"* (diagnose-funk.org/2327).

3.2 Demanding balance to create uncertainty and paralysis

Studies finding no effects do not refute studies demonstrating effects. The decisive question is how evidence is weighted, rather than whether it is complete. Authorities currently favour criteria emphasising uncertainty while downplaying indications of risk, particularly by questioning findings or citing null results. This asymmetric interpretation

has a major influence on conclusions.

Authorities' treatment of embryotoxicity findings illustrates agnogenesis: constructing non-knowledge, analysed by Pinto (2019) in *"Knowing or Not: Agnotology and the Social Construction of Ignorance in Commercially Oriented Research"*. Ignorance is produced and feigned by excluding important studies from risk assessment through neglect, questioning or distorted interpretations. Alleged non-knowledge is then presented as knowledge. Pinto calls these downplaying tactics agnogenesis. Another method, Pinto says, is demanding that critics also cite studies finding no risk. This is roughly equivalent to requiring tobacco critics to give equal weight to favourable Big Tobacco studies, opponents of nuclear power to cite studies promising safety, and environmentalists addressing glyphosate to acknowledge Monsanto publications. Pinto writes, using tobacco as an example:

"As expected, influencing media reporting also became an important tool for the tobacco industry in fighting regulation. Invoking journalistic balance under the 'fairness doctrine', which required broadcasters to include opposing viewpoints when covering controversial issues of public importance, industry lawyers demanded equal airtime for both sides. Whenever scientific findings were cited to argue that smoking causes lung cancer, industry lawyers contacted the editors and supplied a list of 'independent' scientific experts who questioned the findings. Such appeals to 'balance' and 'objectivity' led the public to assume that experts had not yet reached scientific consensus on the connection between smoking (and later passive smoking) and lung cancer. In Oreskes and Conway's words (2010): 'Balance apparently came to mean giving both sides equal weight rather than giving both sides due weight.' Much of the tobacco industry's success in buying mass-media airtime and advertising space rested on journalism's commitment to presenting both sides of the story."

This creates paralysis by relativising risks and claiming balance is a scientific criterion. We address these tactics in *"David Michaels: When Scientific Findings Threaten Powerful Interests. Industry Tactics: Sowing Doubt to Prevent Protective Rules"* (diagnose-funk.org/1882).

At the same time, evidence requirements are formulated in ways difficult to meet under real research conditions. Insisting on established causality means risks are recognised only after they have materialised, undermining the precautionary principle. Our Focus report *"The Causality Deception"* addresses this mechanistic understanding of causality (diagnose-funk.org/1539).

3.3 The silent dimension of possible harm

Discussion of embryotoxicity is often reduced to whether clearly demonstrable malformations exist that can be directly transferred from animal experiments to humans.

This perspective is too narrow. The relevant dimension of possible effects lies in subtle changes to developmental processes. When electromagnetic fields affect processes such as cell division, neuronal networking or gene regulation during sensitive phases of embryonic development, the consequences need not be immediately visible. They may emerge only years later, as cognitive impairments, behavioural problems or greater vulnerability to neurological disorders. This delay makes scientific demonstration difficult and favours systematic underestimation of possible risks. Absence of immediate, clearly attributable effects, especially when ethical constraints prevent many human biological studies, is then wrongly interpreted as evidence of safety.

3.4 The precautionary principle and regulatory practice

The precautionary principle calls for preventive action in the face of plausible risks, even when scientific uncertainty remains. It recognises that waiting can cause irreversible harm. Uncertainty is often used against precaution rather than as a reason for preventive action. Evidence assessment has now become a political debate. In *“Health and Safety Practices and Policies Concerning Human Exposure to RF/Microwave Radiation”*, Professor James C. Lin (2025) analyses why findings of harm are disputed. He describes an industry-regulatory complex comprising the WHO EMF Project, ICNIRP and the BfS, whose reassurances protect the mobile-communications industry's billion-euro business. In an interview on [infosperber.ch](https://www.infosperber.ch), scientist Professor Meike Mevissen said:

“What bothers me is that institutions such as Germany's Federal Office for Radiation Protection constantly argue everything away. Even if the effects found are small, that can be communicated. The Office wants science to say there are no effects. That means it need not make a decision, which apparently is not wanted, even for small effects. Research is very political, and we repeatedly encounter the attitude that health risks must not exist. That sometimes obstructs our work” (16 January 2026). <https://www.infosperber.ch/politik/schweiz/unsere-forschung-wurde-behindert>

To give reassurance, the BfS interprets studies one-sidedly and incorrectly, and has staged reassuring press campaigns (diagnose-funk.org/2143 and diagnose-funk.org/1657). Several of our Focus reports address this risk-downplaying policy. Below, we document criticism of the BfS's reassuring discussions of two studies investigating prenatal and postnatal effects. The criticism of its interpretation of the MOBI-Kids study on childhood cancer is from Peter Hensinger's *“The Struggle over Authority to Interpret the Risks of Mobile Phone Radiation”*, *umwelt-medizin-gesellschaft* 2/2023. Criticism of its interpretation of Bozok et al.'s study (2023) on embryonic heart-muscle tissue appeared in *ElektrosmogReport* 3/2025, written by Dr Klaus Scheler (see p. 23 of this Overview).

4 The struggle over authority to interpret the risks of mobile phone radiation (excerpt)

By Peter Hensinger, M.A.

The MOBI-Kids study was conducted in 14 countries from 2010 to 2015, when mobile communication use was rising rapidly. It is the largest study of its kind to date. The main study examined tumour histories in 671 brain-tumour patients aged 10 to 24.

The Federal Office for Radiation Protection views MOBI-Kids as proof that mobile-phone radiation cannot cause brain tumours in children. Study authors disagree with this interpretation. A fierce controversy over interpretation has arisen, documented in EMF-Portal. See source (1) at the end of Chapter 4.

4.1 The Federal Office for Radiation Protection's interpretation

When commissioning MOBI-Kids, the EU research service CORDIS was aware of its potential impact: *"The results of the study are expected to attract very high public attention and could have considerable societal implications"* (2). This attention was achieved worldwide. In a powerful press message, the BfS announced the alleged study finding: *"Using mobile and DECT phones does not increase the risk of brain tumours in children and adolescents. This is suggested by the recently published international MOBI-Kids study"* (7 February 2022) (3). Print media, radio, television and scientific and medical portals repeated the BfS statement without scrutiny, conveying that the study had definitively proved children and adolescents could use smartphones and tablets without risk.

4.2 Study authors consider the Federal Office for Radiation Protection's interpretation a misinterpretation

MOBI-Kids does not prove, as the BfS claims, that children face no brain-tumour risk. Study authors reject this interpretation. Four Medical University of Vienna department heads, lecturers and professors Hutter, Kundi, Moshhammer and Wallner, including two MOBI-Kids authors (Hutter, Kundi), responded in the Austrian medical journal *medi.um*. The journal exposes the BfS interpretation as a misinterpretation, describing it editorially as *"amateurism"* or *"lobbying"*. The article clarifies: *"There is ... no basis whatsoever for associating the MOBI-Kids results with an assumption of no risk from mobile-phone exposure, or indeed from wireless radiation generally"* (4). The authors explain:

- MOBI-Kids has not demonstrated that children cannot develop brain tumours from mobile-phone use. In the overwhelming majority (at least 80%) of children with tumours studied, the tumour had already developed before phone use began, prenatally or shortly after birth.

• MOBI-Kids indicates that later mobile-phone use may have accelerated the development of existing tumours that were not initiated by phone use. The results plausibly demonstrate a cancer-promoting effect of non-ionising radiation: *"It is believed that many childhood and adolescent tumours may be initiated prenatally or shortly after birth. Tumours eligible for MOBI-Kids therefore peak mainly in the third year of life and decline thereafter. In the 10-24 age range, many tumour patients may thus already have had a growing mass of neoplastic cells that led to their brain-tumour diagnosis after they began using mobile phones. Under these circumstances, mobile-phone use could have increased these emerging tumours' growth rate and led to earlier diagnosis"* (MOBI-Kids study, p. 15).

The BfS omits this statement about a cancer-promoting effect.

4.3 Wishful thinking leads to misinterpretation

How does the BfS arrive at its misinterpretation? The abstract states, seemingly unambiguously: *"Overall, our study provides no evidence of a causal association between mobile-phone use and brain tumours in young people."* Its meaning becomes unambiguous only when the whole study is read and a clause derived from its overall content is added:

"Overall, our study provides no evidence of a causal association between mobile-phone use and brain tumours in young people because most brain tumours recorded in the study had developed before(!) mobile-phone use began" (bold addition by diagnose:funk).

In their detailed analysis in *umwelt-medizin-gesellschaft* 3/2022, Scheler / Hensinger criticise the BfS's out-of-context interpretation as wishful thinking: *"In our view, the largely uncritical, one-sided adoption of statements in MOBI-Kids, with its devastating impact, is irresponsible. Conclusion: the BfS is failing its obligation to take precautionary action and its protective mandate. It should withdraw its MOBI-Kids press release and publish a correction"* (5).

In a letter dated 6 February 2023, diagnose:funk called on the BfS president to correct the interpretation. The BfS replied in a single sentence: *"The BfS sees no reason to change its position on Mobi-Kids"* (8 March 2023).

Sources

(1) Castaño-Vinyals et al. (2021): Wireless phone use in childhood and adolescence and neuroepithelial brain tumors: Results from the international MOBI-Kids study, *Environ Int* 2021; 160: 107069, <https://www.emf-portal.org/46426>

(2) CORDIS (2009): Risk of brain cancer from exposure to radiofrequency fields in childhood and adolescence, <https://cordis.europa.eu/article/id/86082-mobile-phone-use-and-childhood-brain-cancer/de>

- (3) Federal Office for Radiation Protection (7 February 2022): Mobile communications: no increased tumour risk for children. The MOBI-Kids study supports radiation-protection risk assessment. <https://www.bfs.de/SharedDocs/Pressemitteilungen/BfS/DE/2022/003.html>
- (4) Kundi M, Hutter H-P, Wallner P, Moshhammer H (2022): Statement on the results of the Mobi-Kids study. medi.um 01/2022; download: <https://www.aegu.net>
- (5) Scheler K, Hensinger P (2022): Results of MOBI-Kids and their misinterpretation disseminated by the Federal Office for Radiation Protection, umwelt-medizin-gesellschaft 3/2022.



These two Focus reports examine how the WHO EMF Project, ICNIRP and the Federal Office for Radiation Protection interpret the research.

5 The Federal Office for Radiation Protection's assessment of the study by Bozok et al.

By Dr Klaus Scheler

Introduction

Bozok et al. (2023) (source [1] at the end of Chapter 5), *"The Effects of Long-Term Prenatal Exposure to 900, 1800 and 2100 MHz Electromagnetic Field Radiation on Myocardial Tissue of Rats"*, investigated for the first time the long-term effects of exposing pregnant Sprague-Dawley rats (white rats) to common mobile-phone frequencies on their newborn male offspring's heart-muscle tissue. Six groups of pregnant rats (n=3 each) were studied: group 1, unexposed controls; group 2, 900 MHz, 24 h/day; group 3, 1800 MHz, continuous 6 h/day; group 4, 1800 MHz, continuous 12 h/day; group 5, 1800 MHz, 24 h/day; group 6, 2100 MHz, 24 h/day. Animals were exposed for a

period of 20 days. After 60 days, offspring were killed and heart-muscle tissue examined histopathologically and biochemically.

Offspring in exposed groups showed atypical myocardial morphology: pyknotic nuclei (degenerated cell nuclei), cytoplasmic vacuolisation [2], eosinophilic cytoplasm and enlarged myocardial muscle fibres. Heart-muscle damage increased with frequency (900, 1800, 2100 MHz) at the same exposure duration (24 h/day), and with exposure duration (6, 12, 24 h/day) at 1800 MHz. The latter finding therefore indicates a dose-response relationship. Controls did not show this damage: their heart tissue had typical heart-muscle-cell structure, regular cell boundaries and intact endomysium.



ElektromogReport 3/2023 [3] reviewed the study and assessed its findings as a constructive contribution to understanding RF-EMF's effects on heart muscle. Almost simultaneously with its appearance in EMF-Portal [4], the BfS's Competence Centre for Electromagnetic Fields (KEMF) published an assessment [5] dismissing the study entirely: *"For this reason, the study provides no reliable contribution to current knowledge concerning RF-EMF and effects on the heart."*

The reasons given fail to do justice to the study's aims and findings. The criticism must therefore be rejected, as the following detailed response shows.

5.1 Misrepresentation of published facts

1. The BfS claims it is unclear whether pregnant rats were assigned randomly to exposure groups. This is incorrect: the study was a *"randomized study"*, clearly stated in its *"Methods"* section. Assignment was therefore random, as is customary because of the generally accepted risk of bias otherwise.
2. The BfS also claims pregnant rats were distributed among cages. This is incorrect: each cage's females were assigned a male for mating. Pregnancy therefore did not begin simultaneously. Exposure began only after pregnancy occurred. The following BfS criticism is thus also unfounded: *"It is not mentioned ... whether exposure of the animals took place simultaneously ..."* [5]. The procedure is described indirectly in the publication.
3. The BfS further claims it is unclear whether *"the [unexposed] animals were subjected to the same environmental conditions as exposed animals"*.

This too is largely apparent from the publication's *"Animal Care and Welfare"* section, especially its reference to observing and checking compliance with the Health Guidelines for the Care and Use of Laboratory Animals.

4. The small number of animals criticised is well justified in the *"Discussion"*: *"However, animal welfare should not be ignored in these animal studies. Thus, we used a minimal number of pregnant rats (n:3) per exposure group and we examined a minimal number of male pups (n:6) from these litters in our study."*

5.2 Disregard for the overall context

The BfS further criticises the substances measured, MDA, a lipid-peroxidation biomarker (cell-membrane damage), and glutathione (GSH), a cellular antioxidant, as invalid ROS biomarkers (reactive oxygen species, oxygen radicals). It concludes: *"The authors' claim that their results support a role of RF-EMF in generating oxidative stress therefore cannot, in the BfS's view, be derived from the methods used."*

This criticism shows disregard for the role MDA and GSH measurements play in the study's overall investigative context, outlined briefly below.

As noted above, the authors' statement rests partly on finding a dose-response relationship between RF-EMF exposure and different forms of heart-muscle damage absent without exposure. They also demonstrated consistency with similar findings in other studies. Important Bradford Hill criteria [6] are thus met, providing substantial support for regarding RF-EMF exposure as a cause of heart-muscle damage and assuming a causal relationship. Since other studies repeatedly found increased ROS after RF-EMF exposure in organs, the authors investigated related biochemical markers, MDA and GSH, to further support causality. MDA and GSH values, with one exception, showed a statistically significant dose-response relationship analogous to heart-muscle damage: MDA, indicating lipid peroxidation (cell-membrane damage), rose significantly with radiation duration (dose), while cellular antioxidant glutathione concentration fell correspondingly.

The BfS criticism states that MDA primarily marks lipid peroxidation and not necessarily ROS, since MDA can arise through other pathways [7]. MDA therefore does not automatically imply ROS; ROS, however, always implies lipid peroxidation and corresponding MDA levels. GSH is similar, being considered a valid ROS biomarker only in vitro.

Formally, the authors' claim that RF-EMF promotes ROS formation may therefore not necessarily follow from MDA / GSH data alone(!), detached from those findings. That was never their intention. Yet given the absence of heart-muscle-cell damage and thus cellular lipid peroxidation in controls, and the fact that exposed and control groups were otherwise subjected to identical investigative

conditions, RF-EMF remains the only relevant factor explaining the measured MDA and GSH levels and dose-response relationship. The findings are therefore, as the authors write, consistent with the assumption of increased ROS through RF-EMF exposure.

Because the BfS rejects this conclusion without considering the argument's overall context, its assessment is rejected as incomprehensible and unjustified.

5.3 Failure to weigh the relevance of influencing factors

The BfS further criticises the publication for not mentioning certain influencing factors (blinding? age of pregnant rats? cage locations and arrangement, e.g. all groups in one room? controls equipped with an inactive antenna?). The BfS critics could have asked the authors about these. Concluding that a *"considerable risk of bias"* [5] therefore(!) cannot be excluded is suggestive and agenda-driven. Risk of bias simply cannot be assessed without knowing whether these factors were considered. One could just as well conclude that, despite undocumented factors, considerable relevance cannot be ruled out.

Lack of knowledge permits a neutral conclusion only in this form: *"Cannot be assessed conclusively without further details."* It does not justify concluding that factors were ignored and downgrading is therefore warranted. All questions about missing factors might be answered positively. If all criticisms were resolved or proved marginal, one could or should conclude that the study has considerable relevance.

Any study may involve factors that were not considered or documented, for whatever reason. While this may warrant criticism, an essential aspect of assessing a study's evidential strength is ignored: the numerous factors do not all have equal relevance. Bias caused by failing to consider particular factors need not automatically be substantial; it may be marginal.

A balanced assessment must therefore weigh the relevance of the various considered and unconsidered factors. Until their relevance is explained and weighed, an assessment cannot be called balanced.

Downgrading findings solely because factors are undocumented, without weighing their relevance within the study and acknowledging its constructive elements, is like disparaging a flower that has not yet bloomed.

Summary

1. Published facts were ignored. Much of the criticism is therefore unjustified and can only be rejected.
2. The BfS criticism also fails to consider the argument's overall context. Individual observations are taken out of context, making the authors' conclusions seem less compelling. Only this enables the BfS to formulate its spurious criticism.
3. The study's dismissal also rests on a few undocumented influencing factors (see above). The BfS does not know whether or how these were considered, but assumes their relevance to the study's evidential strength is substantial. It acknowledges that missing information is necessary *"to assess the study's quality appropriately"* [5]. Despite this lack of knowledge, it assesses the evidence negatively. Presenting non-knowledge as knowledge and thereby downgrading findings deceives readers through seemingly logical reasoning.
4. The relevance of all influencing factors, especially those documented and investigated, has not been weighed or assessed. Missing a few details of study design does not justify criticism of its findings, still less disparagement.

The final wholesale dismissal therefore rests on spurious arguments. A BfS assessment scheme also emerges that makes maximal demands and follows an all-or-nothing approach. The BfS decides (a) which factors must be adequately considered, (b) which relationships must be demonstrated by which methods, and (c) that a study must be downgraded if any factor is unconsidered or undocumented, or relationships are not established with absolute certainty. This disregards accepted principles of assessing and weighing all(!) considered and possibly unconsidered aspects, inevitably producing a one-sided, distorted, questionable and contestable assessment. An all-or-nothing scheme also opens the door to abusing assessment for partisan purposes.

Sources

- [1] **Bozok S, Karaagac E, Sener D, Akakin D, Tumkaya L (2023):** The effects of long-term prenatal exposure to 900, 1800, and 2100 MHz electromagnetic field radiation on myocardial tissue of rats. *Toxicol Ind Health* 2023 Jan; 39(1): 1-9; <https://pubmed.ncbi.nlm.nih.gov/36383165/>
- [2] Vivien Hornawsky (2025): Vacuole: Definition, Formation and Functions. See <https://www.medi-karriere.de/wiki/vakuole/>
- [3] ElektrosmogReport 3/2023: <https://www.emfdata.org/de/studien/detail&id=798>
- [4] EMF-Portal entry for Bozok et al.: <https://emf-portal.org/de/article/49027>
- [5] Competence Centre for Electromagnetic Fields (KEMF) (2023): Spotlight on *"The effects of long-term prenatal exposure to 900, 1800, and 2100 MHz electromagnetic field radiation on myocardial tissue of rats"* by Bozok et al. in *Toxicology and Industrial Health* (2022). <https://kurzlinks.de/t07g>
- [6] Wikipedia (German): Bradford Hill criteria: [https://de.wikipedia.org/wiki/%C3%84tiologie_\(Medizin\)#Bradford-Hill-Kriterien](https://de.wikipedia.org/wiki/%C3%84tiologie_(Medizin)#Bradford-Hill-Kriterien)
- [7] Wikipedia (English): Thiobarbituric acid reactive substances (TBARS): <https://en.wikipedia.org/wiki/TBARS>

Glossary

Apoptosis: Programmed cell death.

AMPA (AMPA receptors): Ionotropic glutamate receptors responsible for rapid excitatory signal transmission in the brain; important for synaptic plasticity and learning and memory processes.

BDNF: Brain growth factor important for learning and synaptic plasticity.

Corticosterone: Stress hormone regulating metabolism and immune responses.

DECT: Digital standard for cordless phones using pulsed microwave radiation.

Dendritic connections: Branches of nerve cells important for processing information.

DNA strand break: Disruption of the DNA backbone's structure. Single-strand breaks affect one strand; double-strand breaks sever both strands.

Embryogenesis: Early developmental phase of an embryo.

Embryotoxicity: An influence's ability to damage an embryo.

Estradiol (E2): Principal female sex hormone, controlling the menstrual cycle and pregnancy.

Fecundity: Reproductive capacity or the number of offspring produced.

Follicles: Ovarian sacs in which egg cells mature.

FSH: Hormone controlling maturation of egg cells in the ovary.

Gene expression: Activation of genes to produce RNA or proteins.

Germanium: Earliest stage of oogenesis in the ovaries of insects and other arthropods.

Glutathione (GSH): The cell's principal antioxidant molecule.

Glutathione peroxidase (GPx): Antioxidant enzyme neutralising free radicals.

Haemoglobin: Red blood pigment transporting oxygen.

RF-EMF: Radiofrequency electromagnetic fields. The literature often defines radiofrequency as 100 kHz-300 GHz.

Hippocampus: Brain region for memory formation, learning and spatial orientation.

IGF-1: Growth factor important for cell growth and development.

Ionising radiation: Radiation energetic enough to cause ionisation (e.g. alpha particles, X-rays and gamma rays).

LH: Hormone controlling ovulation and ovarian hormone production.

Lipid peroxidation: Oxidative damage to cell membranes by free radicals.

MAP2 (microtubule-associated protein 2): Key structural protein in neurons' cytoskeleton, playing a crucial role in nervous-system development and function.

Mitochondrial stress: Mitochondrial dysfunction caused by oxidative stress.

Neutrophils / lymphocytes: Subgroups of immune cells.

Neurogenesis: Formation of new nerve cells.

Non-ionising radiation: Electromagnetic fields without ionisation energy, e.g. radio waves, microwaves, infrared radiation and visible light.

Non-thermal effects: Biological effects of electromagnetic fields not caused by heating.

NMDA receptor: Ionotropic glutamate receptor central to synaptic plasticity, learning and memory.

NO / nitrite / nitrate: Nitrogen compounds important for vascular regulation.

Oogenesis: Formation and maturation of egg cells.

Oocyte: Maturing egg cell.

Oxidative stress: Imbalance between free radicals and antioxidant systems.

Progesterone: Pregnancy hormone stabilising the uterine lining.

Prostaglandin F2 α : Tissue hormone regulating uterine contractions and blood flow.

ROS: Reactive oxygen species, or oxygen radicals: aggressive molecules that can damage cells.

SAR: A measure of radiation power absorbed in the body.

Synaptic plasticity: Adaptability of neuronal connections; the basis of learning.

TAS (total antioxidant status): A measure of a tissue's overall antioxidant capacity.

Cell proliferation: Cell division and multiplication.

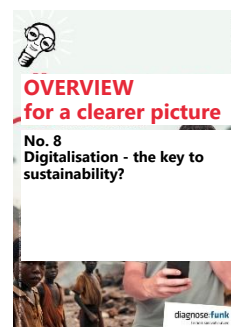
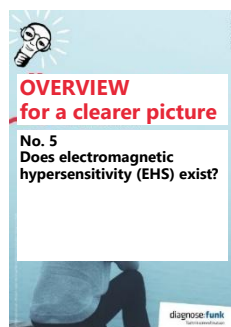
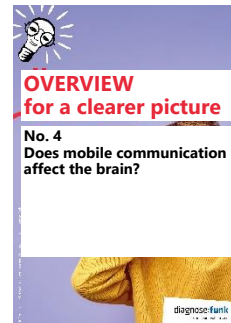
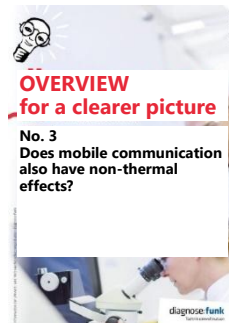
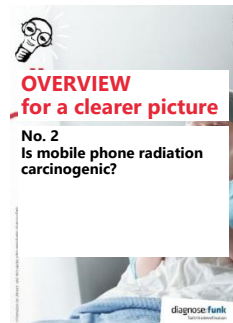
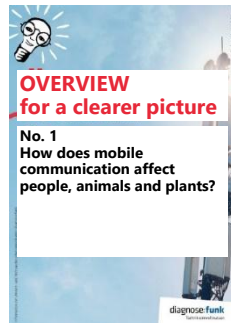
Cell death: Loss of cells through apoptosis or damage.

Cellular dysfunction: Impairment of key cellular processes.

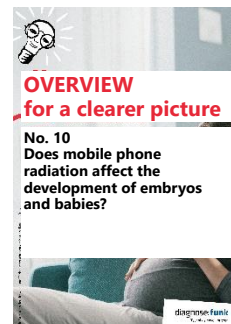
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