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Review

A review of effects of electromagnetic fields on ageing and ageing dependent bioeffects of electromagnetic fields

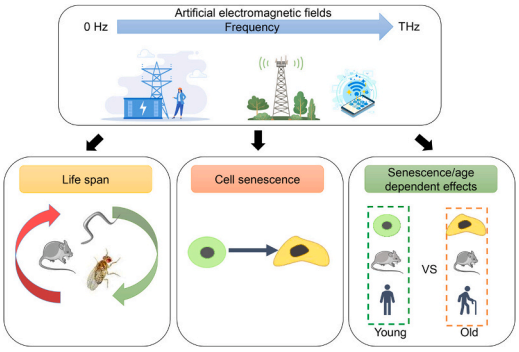
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HIGHLIGHTS

- Electromagnetic fields (EMF) exposure has an effect on lifespan, but the effect may vary with type of EMF and animal model.
- Extremely-low-frequency EMFs exposure can affect cellular senescence in a wave shape- and/or magnetic density-dependent manner.
- Radio-frequency EMFs promotes cellular senescence in a frequency- and intensity-dependent manner.
- The influence of EMFs on cellular, animal, and human health exhibits a degree of age-dependency.

GRAPHICAL ABSTRACT



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ABSTRACT

Thanks to the progress of science and technology, human life expectancy has dramatically increased in the past few decades, but accompanied by rapid ageing of population, resulting in increased burden on society. At the same time, the living environment, especially the electromagnetic environment, has also greatly changed due to science and technology advances. The effect of artificial electromagnetic fields (EMFs) emitted from power lines, mobile phones, wireless equipment, and other devices on ageing and ageing-related diseases are receiving increasing attention. However, the information on the relationship between EMFs and ageing and ageing related susceptibility to EMFs is fragmentary, a review is needed. Only few studies directly investigate the effect of EMFs on ageing, and we reviewed the impact of EMFs on lifespan and cellular senescence to pry whether EMFs have an effect on ageing, and reviewed the age-dependent bioeffects and health impacts of EMFs to see whether ageing would affect biological susceptibility to EMFs. The results indicated that EMFs may have an effect on longevity and cellular senescence, but the results were inconsistent which may depend on EMF types (frequency, intensity, wave shape, etc.), species, and cell lines. Ageing has an impact on the biological or health effects of EMFs; however, the results differ depending on the EMF type and the endpoint or health outcome. Age-dependent changes in free radical metabolism, ion homeostasis, gene expression, enzyme activity, and tissue biophysical properties may be the reason; however, the underlying mechanisms are not fully elucidated.

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1. Introduction

Electromagnetic fields (EMFs), such as geomagnetic fields (GMFs), which are usually relatively stable (Sarimov et al., 2023) and adapted to by living organisms, are common in natural environments, but their balance may be disturbed by human activities. With the rapid development of electric, magnetic and electromagnetic technology, such as mobile communication, and increased consumption of electricity, artificial EMFs emitted from power lines and various electronic devices, such as mobile phones, have been diffusing to almost all human living and working environments, and the potential health hazards have caused widespread concern. Hazards of EMFs, such as the induction of DNA damage (Lai, 2021; Sun et al., 2023) and apoptosis (Rain et al., 2023), have been occasionally reported in experimental studies, and a possible correlation between EMF exposure and increased risk of diseases, such as cancer (Zhang et al., 2024), neurodegenerative disease (Sorahan and Nichols, 2022), or cardiovascular disease (Chai et al., 2023; Parizek et al., 2023), has also been revealed in epidemiological studies. Therefore, we need pay attention to the potential health effects of EMFs.

Owing to advances in technology and medicine, the average life expectancy of humans has significantly increased, but with this, the problem of ageing has arisen. The reason ageing is considered a problem is that the "health span" has not kept up with the increasing lifespan (Cai et al., 2022). As a predominant risk factor, ageing leads to an increased risk of most chronic diseases, such as cardiovascular diseases, cancer, and neurodegenerative diseases, and has become a formidable global socioeconomic burden (Scott et al., 2021). Therefore, it is of paramount importance to promote "healthy ageing" (Lopez-Otin and Kroemer, 2021; Partridge et al., 2018). Although people are paying increasing attention to healthy and active ageing and related research is being carried out, the influence of the electromagnetic environment, which has changed dramatically over the past century due to the rapid development of technology, seems to be ignored.

Ageing leads to a loss of ability to adapt to stress owing to issues such as frailty (Weiss, 2011), which can increase the sensitivity of older people to various stresses, including social vulnerability (Orlandini et al., 2024), air pollution (Park et al., 2024), pathogen infection (Mbalinda et al., 2024), and others (Romano-Lieber et al., 2019). However, whether ageing causes the body to be sensitive to EMFs remains to be elucidated. Based on previous research and reports, the biological effects of EMFs are usually impacted by a variety of factors, including research conditions, the experimental environment, EMF exposure parameters and biological endpoints. For example, in 2013, Xu et al. reported that even under the same experimental conditions, the effect of 1800 MHz radiofrequency EMFs (RF-EMFs) on DNA is different among different types of cells, indicating that the state of the organism itself also determines the biological effects of EMFs (Xu et al., 2013). Therefore, under certain conditions, the biological effects of EMFs may be increased and even lead to improved health. Recently, Xi et al. investigated the prevalence and correlation of multiple chemical sensitivities and electromagnetic hypersensitivity (EHS) with age, sex, and depression in a Japanese population and reported that EHS was positively correlated with age and sex and that older people were more likely to exhibit EHS (Lu et al., 2023). Therefore, ageing might be a risk factor for the health effects of EMFs, which has long been a concern (Redmayne and Johansson, 2015).

In this review, we summarize the current literature on the biological effects of exposure to EMFs, including static magnetic fields (SMFs), rotating magnetic fields (RMFs), extremely low-frequency EMFs (ELF-EMFs), and RF-EMFs, on lifespan and cellular senescence to determine the potential influences of EMFs on ageing and the age-dependent biological and health effects of EMFs to determine the ageing related potential health risk under EMF exposure.

2. Methods

2.1. Search strategy

This study summarized the epidemiological and experimental research by conducting literature searches on EMF-Portal (https://www.emf-portal.org/en) and PubMed, with a cutoff date of December 20, 2024 (Fig. 1). In EMF-Portal, the key search terms included "ageing", "age", "ageing", "senescence", "old", "elder", "lifespan", "life span", "longevity", "survival". In PubMed, the key search terms included MeSH term "electromagnetic fields" combined with the above key words.

2.2. Inclusion criteria

2.2.1 The type of literature is research article.

2.2.2 Articles focusing on ageing, senescence, lifespan, survival, or age/senescence dependent effects.

2.3. Exclusion criteria

2.3.1 Not research articles, such as review and commentaries.

2.3.2 Studies that focus solely on age-related diseases, like Alzheimer's disease or cardiovascular diseases, rather than the biological processes of ageing or senescence.

2.3.3 Studies that inadequately compare results across different age groups regarding the age-dependent effects EMFs.

2.3.4 Experimental studies lacking precise experimental conditions, including electromagnetic field flux density, temperature monitoring or control, sham exposure, or background field data for the control group.

2.3.5 Be able to obtain the full text.

3. The effect of electromagnetic fields (EMFs) on lifespan

Longevity is the comprehensive result of overall health status and is naturally closely linked to ageing. Thus, we summarized the findings of published studies on the effects of EMFs on lifespan (Fig. 2 and Table 1). However, it should be noted that lifespan is influenced by multiple factors. Many other bioeffects induced by EMFs, such as oxidative stress, genotoxic effects and carcinogenesis, may also have an impact on the lifespan of organisms. Therefore, the purpose of this section is to tried provide more information on the influence of EMFs on ageing, without making scientific assertions. For further reference on the biological effects of EMFs, this book can be consulted (Panagopoulos, 2022).

3.1. Static magnetic fields (SMFs)

The effects of SMFs on lifespan have been investigated in *Caenorhabditis elegans* (*C. elegans*) and mice. Exposure to SMFs at 200 mT significantly reduced the lifespan of *C. elegans* (Hung et al., 2010) and also its offspring (Yang et al., 2022). But, in contrast, exposure to SMFs at 10 mT could promotes *C. elegans* longevity (Song et al., 2022), indicated that low flux intensity SMFs are beneficial, while high flux intensity is harmful. However, long-term (1.7 years) exposure to SMFs at 70–220 mT not only prolonged the lifespan but also increased the health span in healthy male C57BL/6 mice, the exploratory and locomotive activities of the aged mice were also significantly increased (Fan et al., 2023). The beneficial effect observed in mice may be due to the ability of SMFs to mitigate oxidative damage during ageing (Fan et al., 2023). It should be noted that in these studies, the geomagnetic field was not shielded, which might have interfered with the research results. Meanwhile, some studies did not provide specific experimental conditions, such as the direction of the magnetic field (Hung et al., 2010), the exact exposure time of the day (Yang et al., 2022) or the background field data (Table 1), making it difficult to replicate and verify the results subsequently. Therefore, more strictly designed studies are still needed in the future.

3.2. Rotating magnetic fields (RMFs)

Compared to SMFs, the only difference of RMFs is the addition of a rotational change. Exposure to 4 Hz RMFs (200 mT) promoted health status, reduced age-related changes and prolonged the lifespan of *C. elegans*, and oxidative stress regulation were suggested to be the possible mechanism (Xu et al., 2019). If the oscillation of magnetic field (MF) plays a decisive role in determining its biological effect, we need to consider that the relative change in phase caused by animal movement during SMF exposure could also cause a relative change in MF distribution *in vivo*, which may be the reason for the inconsistent effect of SMFs on the lifespan of nematodes and mice mentioned above (Fan et al., 2023; Hung et al., 2010). However, the back ground field data were not provided in this study (Xu et al., 2019) which is a potential interfering factor.

3.3. Extremely low frequency electromagnetic fields (ELF-EMFs)

ELF-EMFs usually refer to EMFs with frequencies between >0 and 300 Hz, which are usually generated by changing current induction. Exposure to 50 Hz MFs at up to 1.0 mT for a lifetime (from prenatal life to natural death) had no significant effect on the lifespan of *Sprague–Dawley* rats (Bua et al., 2018; Soffritti et al., 2016), but exposure to 50 Hz electric field at 10 kV/m decreased the life expectancy of *Balb/C* mice (de Jager and de Bruyn, 1994). Exposure to 75 Hz pulsed MFs at 0.55 mT reduced the lifespan of *Drosophila melanogaster* (*D. melanogaster*) (Agrawal et al., 2021), but exposure to 50 Hz electric field at 35 kV/m elongated the lifespan of *D. melanogaster* under low nutrient conditions (Kawasaki et al., 2021). These research findings suggest that ELF-EMFs can influence the lifespan of animals, with the effects of electric fields and MFs potentially differing. The inconsistent results may be attributed to several factors. For instance, a study found that exposure to 50 Hz MFs extended the lifespan of houseflies (*Musca domestica* L.) at 24 °C but not at 19 °C (Stanojević et al., 2005), indicating that experimental temperature is a critical factor. Additionally, other variables such as environmental stressors (*e.g.*, low nutrition, restraint) and background field interference may also play a role.

3.4. Radio frequency electromagnetic fields (RF-EMFs)

RF-EMFs have a wide range of frequency bands (300 kHz–300 GHz), only a small proportion of which has been studied, and the frequency bands used in wireless technology are of greatest concern. Exposure to RF-EMFs from global system for mobile communication (GSM) mobile phones, wireless-fidelity (Wi-Fi) routers and wireless digital enhanced cordless telecommunications (DECT) phones showed no significant effect on the lifespan of *C. elegans* (Fasseas et al., 2015) or honeybees (Treder et al., 2023; Westerdahl and Gary, 1981). But chronic exposure to GSM-like signal reduced the survival of female *Sprague-Dawley* rats (Bartsch et al., 2010), and exposure to 2450 MHz microwave at 6.8 W/kg reduced the lifespan of female CD-1 mice (Liddle et al., 1994). This might be due to the different doses of RF-EMFs applied in these studies. Because, under the same conditions, 2.0 W/kg 2450 MHz microwave had no effect on the lifespan of female CD-1 mice (Liddle et al., 1994). For other frequency bands, exposure to pulsed high-peak power microwaves (9.4 GHz) did not affect basic life incidence of *C. elegans* (Sun et al., 2022). But exposure to 10 GHz microwave radiation (9.8 mW/kg) for 5 h continuously or 3 + 3 h (with a half-hour break) extended the lifespan of Basc stock female *D. melanogaster* (Memmi and Ünlü, 2007), and the fronds of *Spirodela polyrhiza* (L.) schleiden cultures under radiation from the radio location station (156–162 MHz) had a shorter lifespan (Magone, 1996). Therefore, based on existing research, the impact of RF-EMFs on lifespan appears to vary depending on the organism species and the specific exposure conditions.

3.5. Terahertz radiation

Terahertz radiation refers to EMFs with frequencies ranging from 0.1 to 3.0 THz, and terahertz technology has developed rapidly in recent years (Cherkasova et al., 2021); at the same time, the biological effects of terahertz radiation have also received increasing attention. Terahertz radiation (0.1–2.2 THz) had an indirect effect on gene expression and signalling pathways that control survival and lifespan, with some positive influence on male *D. melanogaster* survival during the stage of a monotonic decrease in fly number and negative effect during the stage of a relatively stable number of flies (Fedorov et al., 2014), but no significant effect on the average lifespan of F1 progeny in either sex was observed (Fedorov and Weisman, 2015). Therefore, terahertz radiation can also affect the lifespan of organisms, however, these studies did not measure the precise dose of terahertz radiation exposure on *D. melanogaster*, which may lead to difficult for subsequent verification study.

3.6. Multiple electromagnetic fields (multi-EMFs) exposure

In real-life environments, unlike single EMF exposure in the laboratory, we are constantly exposed to complex environmental EMFs, and whether this multi-EMFs exposure affects ageing is noteworthy. It has

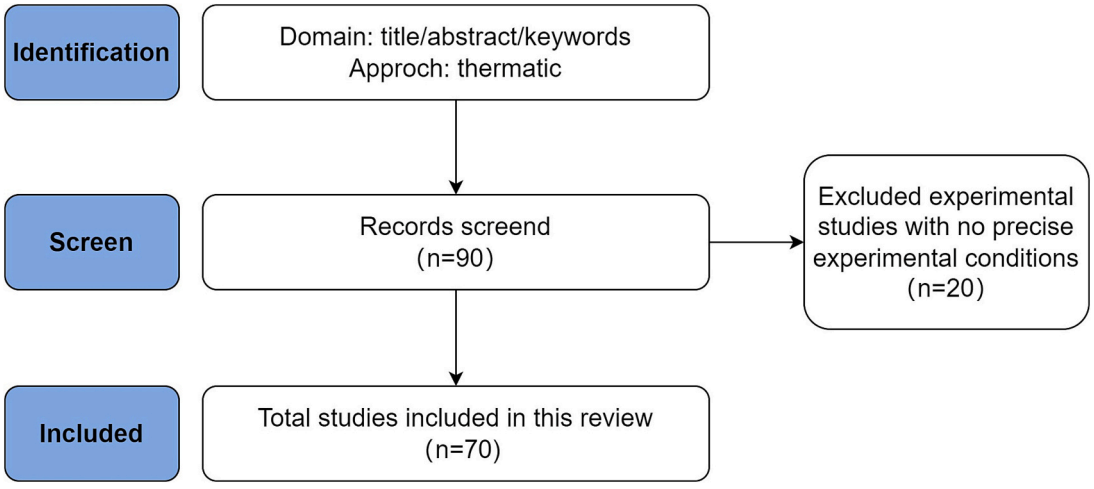


Fig. 1. Literature search and inclusion.

been reported that three-dimensional (3D) oscillating low-frequency electric and magnetic field (3D OLF-EMFs) exposure has a significant effect on *D. melanogaster* lifespan, and both decreasing and increasing effects are dependent on the specific parameters of the 3D OLF-EMFs (Makarov and Khmelinskii, 2014), suggesting that multi-EMFs exposure has an effect on lifespan and that the effects differ depending on the parameters of the various EMFs to which an individual is exposed. Nevertheless, a randomized controlled trial showed that the use of a bed with an insulating system of EMFs could improve immune function and reduce redox and inflammatory states and decrease the rate of ageing in people who sleep on these beds (Diaz-Del Cerro et al., 2020), suggesting that environmental multi-EMF exposure has an effect on ageing, however this await more study to verify.

4. The effect of electromagnetic fields (EMFs) on cellular senescence

Cellular senescence has been identified as the origin and basis of body senescence and functional decline in tissue and organ systems (Lopez-Otin et al., 2013; Tuttle et al., 2021). Many studies have investigated the effect and underlying mechanisms of EMFs on cellular senescence (Fig. 3 and Table 2). Ageing is a highly complex biological process intricately linked with various biological phenomena, including DNA damage and oxidative stress (Panagopoulos, 2022). Given the extensive existing reviews on the bioeffects EMFs, this review specifically focused on research that directly investigated the impact of EMFs on the senescence.

4.1. Static magnetic fields (SMFs)

SMFs have an effect in delaying cellular senescence in both natural and drug-induced senescent cells (Fan et al., 2023; Maredziak et al., 2017). Exposure to 500 mT SMFs for 7 days could increase cell viability, faster proliferation in naturally primary human adipose-derived mesenchymal stem cells (MSCs), which could be blocked by a PI3K/Akt inhibitor (Maredziak et al., 2017). In D-galactose-induced senescent PC12 rat pheochromocytoma cells, exposure to 100–500 mT SMFs significantly reduced senescence-associated β-galactosidase (SA-β-gal) activity (Fan et al., 2023), and the oxidative stress elicited by D-galactose can be alleviated by SMFs by increasing superoxide dismutase and catalase and reducing malondialdehyde levels (Fan et al., 2023). Considering PI3K/Akt signalling pathway is also involved in oxidative stress regulation (Bäumer et al., 2008); regulating oxidative stress may be one of the main mechanisms by which SMFs delay cellular senescence.

4.2. Rotating magnetic fields (RMFs)

Like SMFs, RMFs also delay cellular senescence. Exposure to 4 Hz RMFs at 200 mT significantly delayed cellular senescence, indicated by reduced SA-β-gal activity, in human umbilical vein endothelial cells (HUVECs) (Xu et al., 2019). The anti-ageing effect of RMFs may occur through interfering intracellular calcium signalling that RMFs increased intracellular Ca^{2+} concentrations and decreased mitochondrial membrane potential (MMP) and adenosine triphosphate (ATP) production in HUVECs (Xu et al., 2019). Calcium signalling and mitochondrial

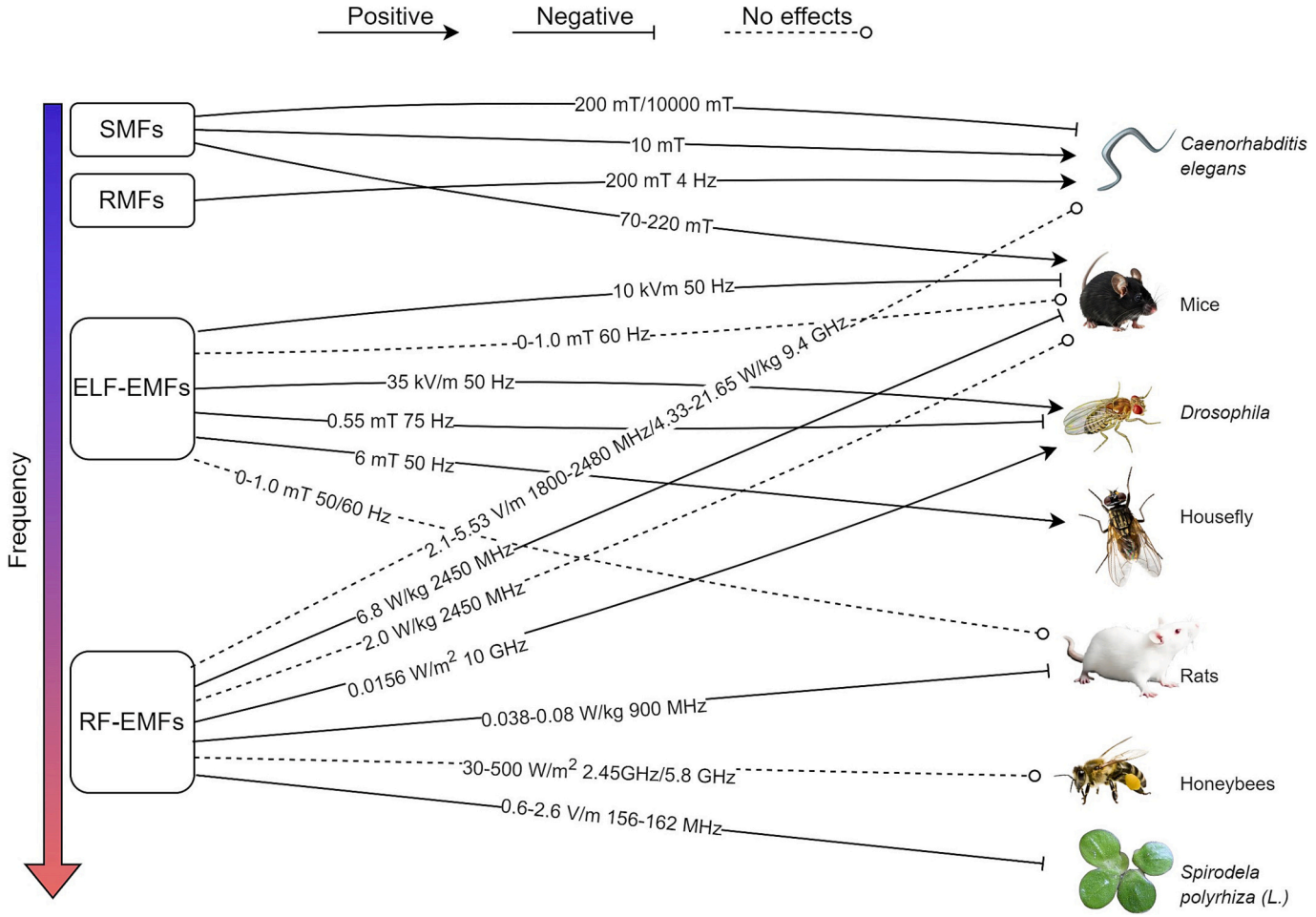


Fig. 2. The effect of EMFs on lifespan. The effect of EMFs on lifespan in *Caenorhabditis elegans*, *Drosophila*, housefly, honeybees, mice, rats and *Spirodela polyrhiza* (L.).

metabolism play important roles in cellular senescence (Gao et al., 2024; Martin et al., 2023), and molecules involved in both are suggested to be critical messengers that mediate the biological effects of EMFs (Ma et al., 2023; Santini et al., 2018). Therefore, calcium signalling may be one of the main mechanisms mediating the effect of EMFs on cellular senescence; however, further studies are needed.

4.3. Extremely low frequency electromagnetic fields (ELF-EMFs)

The results on the effects of ELF-EMFs on cellular senescence were inconsistent which may depend on the signal type and/or magnetic flux density of EMFs.

4.3.1. Signal type-dependent effect of ELF-EMFs on pro-senescence

One of the main differences in the effect of ELF-EMFs on cellular senescence was observed between sinusoidal and pulsed signals, and it seems that the effect of pulsed EMFs was more obvious. Exposure to 1.5 mT 25 Hz pulsed MFs significantly promoted the ageing process of *Saccharomyces cerevisiae* (*S. cerevisiae*), but exposure to 2.45 mT 50 Hz sinusoidal MFs had no significant effect (Mercado-Saenz et al., 2019).

Table 1
The effect of EMFs on lifespan.

EMFs	Main parameters	Exposure time (model)	Organism	Results	References
SMFs	70–220 mT, vertical, without shielding	1.7 years (continuous)	Male C57BL/6 mice	Prolong	(Fan et al., 2023)
	10 mT, vertical, without shielding	Lifetime (continuous)	*Caenorhabditis elegans*		(Song et al., 2022)[a]
	200 mT, direction unknown, without shielding	Lifetime (continuous)	*Caenorhabditis elegans*	Reduce	(Hung et al., 2010)[a]
	10, 000 mT, vertical, without shielding	3, 5, and 12 h (parental exposure)	Offspring of male *Caenorhabditis elegans*		(Yang et al., 2022)[a]
RMFs	200 mT, 4 Hz	Lifetime (intermittent exposure, 1, 2 or 4 h/d)	*Caenorhabditis elegans*	Prolong	(Xu et al., 2019)[a]
ELF-EMFs	35 kV/m, 50 Hz	12 h in day time	*Drosophila melanogaster* under low nutrient conditions	Prolong	(Kawasaki et al., 2021)[a]
	6 mT, 50 Hz sine	Lifetime (continuous)	Housefly (*Musca domestica* L.) at 24 °C		(Stanojević et al., 2005)[a]
	0–1.0 mT, 50 Hz sine	Lifespan whole-body exposure, 19 h/d (continuous or 30 min on/30 min off)	*Sprague–Dawley* rats	No effects	(Bua et al., 2018; Soffritti et al., 2016)
	6 mT, 50 Hz sine	Lifetime (continuous)	Housefly (*Musca domestica* L.) at 19 °C		(Stanojević et al., 2005)[a]
	0.002, 0.2, 1.0 mT, 60 Hz	8 weeks, 18.5 h/d, 7 d/week (continuous and intermittent, 1 h on/1 h off)	F344/N rats and B6C3F1 mice		(Boorman et al., 1997)
	10 kV/m, 50 Hz	From conception until death (20 h/d)	Generation 1 and 2 of Balb/C mice	Reduce	(de Jager and de Bruyn, 1994)[a]
	0.55 mT, 75 Hz pulse	Acute exposure (6 h), chronic exposure (6 h/d for 14 d)	*Drosophila melanogaster*		(Agrawal et al., 2021)[a]
RF-EMFs	5.53 V/m, 1800 MHz GSM (217 Hz modulation)	Lifetime (intermittent exposure, 3 h/2 d)	*Caenorhabditis elegans*	No effects	(Fasseas et al., 2015)
	3.75 V/m, 1800–1900 MHz DECT (100 Hz repetition rate pulses)	Lifetime (30 min on/30 min off)			
	2.1 V/m, 2400–2480 MHz Wi-Fi (10 Hz repetition rate pulses)	Lifetime (30 min on/30 min off)			
	4.33, 8.66, and 21.65 W/kg, 9.4 GHz (10, 20, and 50 Hz pulse)	30 min	*Caenorhabditis elegans*		(Sun et al., 2022)
	2450 MHz (177.83 mW) and 5.8 GHz (10 mW), at a distance of 2 m	Lifespan (12–14 weeks) whole-body exposure	Honeybees (adult worker)		(Treder et al., 2023)
	30–500 W/m^2, 2450 MHz	0.5, 6, and 24 h continuous	Adult honeybees		(Westerdahl and Gary, 1981)
	2.0 W/kg, 2450 MHz	From the 55th day of age to death (1 h/d, 5 d/week)	Female CD-1 mice		(Liddle et al., 1994)
	0.0156 W/m^2, 10 GHz	3 + 3 h discontinuous (30-min resting period)	*Drosophila melanogaster*	Prolong	(Memmi and Ünlü, 2007)
	156–162 MHz, 1250 Hz pulse, 0.6–2.6 V/m	120 h	Fronds of *Spirodela polyrhiza* (L.) Schleiden cultures	Reduce	(Magone, 1996)
	0.038–0.080 W/kg, 900 MHz (217 Hz pulse)	Up to 37 months (exposure was practically permanent with necessary interruption)	Female *Sprague–Dawley* rats		(Bartsch et al., 2010)
	6.8 W/kg, 2450 MHz	From the 55th day of age to death (1 h/d, 5 d/week)	Female CD-1 mice		(Liddle et al., 1994)
Terahertz radiation	0.1–2.2 THz, 10 kHz modulated, 76 MHz pulse frequency, pulse power 8.5 mW	30 min	*Drosophila melanogaster*	Positive influence	(Fedorov et al., 2014)[b]
	0.1–2.2 THz, 10 kHz modulated, 76 MHz pulse frequency, pulse power 8.5 mW	30 min	F1 progeny of *Drosophila*	No effects	(Fedorov and Weisman, 2015)[b]
3D OLF-EMFs	650–1500 V/m, 12,550–27,000 Hz	24 h (continuous exposure)	*Drosophila melanogaster*	Prolong/reduce (Effect were dependent on the exposure parameters)	(Makarov and Khmelinskii, 2014)

[a] Studies without background field data.
[b] Studies without accurate data of EMFs exposure.

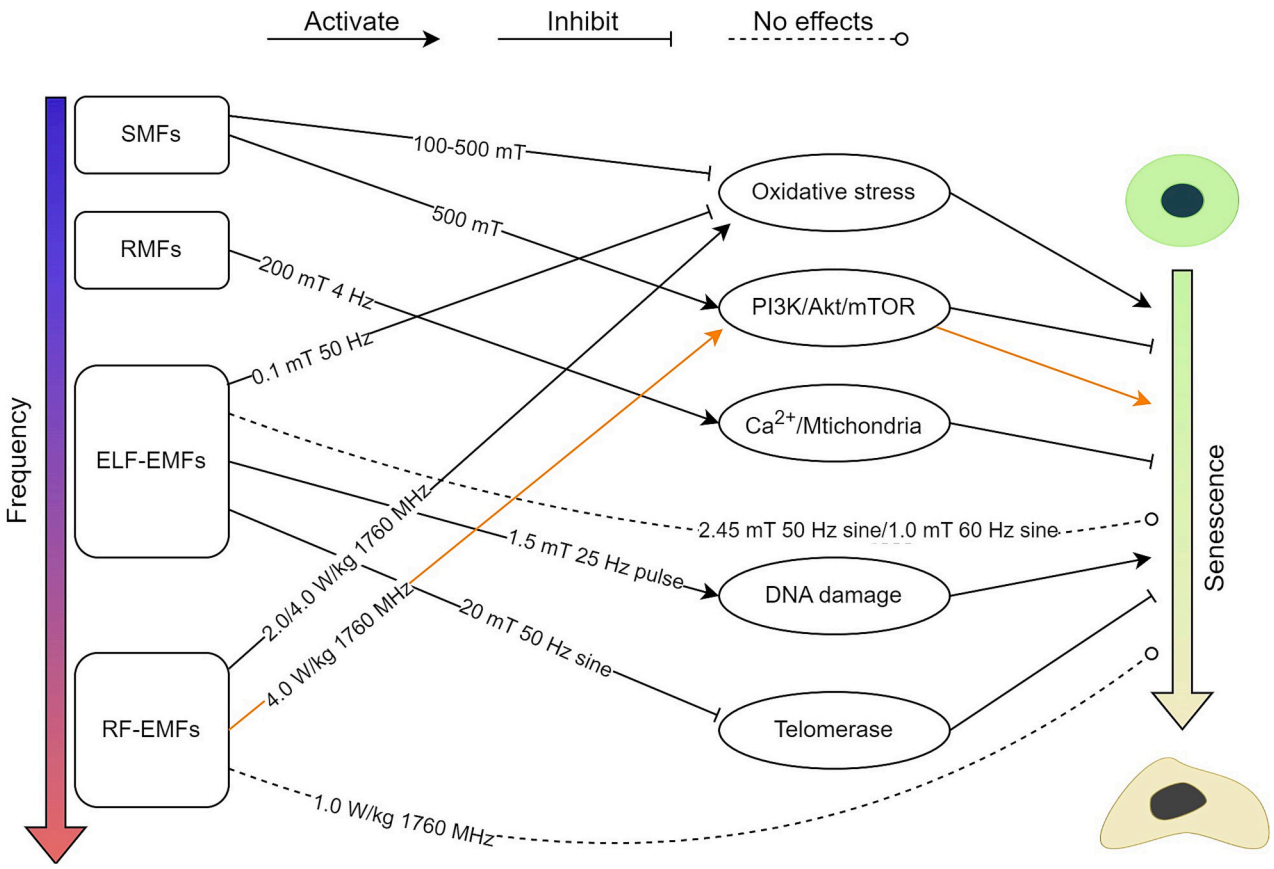


Fig. 3. The effect of EMFs on cellular senescence and the possible underlying mechanisms. EMFs have an effect on cellular senescence, but the effects are complex and different and may depend on specific experimental conditions, including EMF parameters, cell types, and other factors. Oxidative stress, the PI3K/Akt/mTOR signalling pathway, Ca^{2+}/mitochondria metabolism, DNA damage, and telomerase were reported to possibly be involved in this process.

DNA damage is one of the most critical factors in triggering cellular senescence, and 25 Hz pulsed MF exposure increased DNA damage and mutation frequency in *S. cerevisiae* (Mercado-Saenz et al., 2022), thus pulsed MFs may promote cellular senescence through inducing DNA damage. Sun et al. reported that exposure to 1.0 mT 10 Hz pulsed MFs for 2 weeks increased DNA damage but did not induce significant cellular senescence in human foetal fibroblasts (Sun et al., 2021), suggesting that DNA damage may occur earlier than cellular senescence under pulsed MF exposure.

4.3.2. *Magnetic flux density-dependent effect of ELF-EMFs on pro-senescence*

Unlike low magnetic flux density, sinusoidal ELF-EMFs with high magnetic flux density exhibited significant pro-senescence effect. Exposure to 50 Hz sinusoidal MFs at 20 mT for 21 days significantly increased the percentage of senescent cells rat adipose-derived MSCs (Fathi et al., 2017), while neither exposure to 50 Hz sinusoidal MFs at 2.45 mT affected the *S. cerevisiae* senescence (Mercado-Saenz et al., 2019) nor exposure to 60 Hz MFs at 1.0 mT induced cellular senescence in a human breast epithelial cell line (MCF10A) (Hong et al., 2012). Perhaps there is a threshold of magnetic flux density for sinusoidal ELF-EMFs to exert the effect on cellular senescence, however, the cell lines applied in these studies were different which may also contribute to the inconsistent results. The mechanism of how sinusoidal ELF-EMFs promote cellular senescence is unknown. Fathi et al. reported that 50 Hz sinusoidal MFs inhibited telomerase reverse transcriptase (TERT) expression (Fathi et al., 2017). TERT is the rate-limiting component that controls telomerase activity, and both TERT and telomerase activity are necessary to maintain the telomere length required for sustained cell proliferation, which is closely related to cellular senescence (Yuan and Xu, 2019). Thus, the telomere maintaining related biological process might be involved, more studies are required.

4.3.3. *Anti-senescence effects*

Despite pro-senescence, ELF-EMFs also have anti-senescence effect. Exposure to 8 Hz pulsed MFs at 3.8 mT attenuated the senescence of $LepR^{+}$ MSCs in bone marrow in glucocorticoid-treated mice, and this process was modulated by the polycomb histone methyltransferase enhancer of zeste homologue 2 (EZH2) and its trimethylation of histone H3 on lysine 27 (H3K27me3) (Wang et al., 2022). However, the *in vivo* process is much more complicated than the *in vitro*, and further investigation is needed to determine whether the *in vivo* anti-ageing effect of pulsed MFs occurs through eliminating senescent cells or delaying cellular senescence. Notwithstanding this, exposure to a 50 Hz MFs at 0.1 mT was found to delay senescence and activate antioxidant enzymes in etiolated seedlings of *Cucumis sativus* L., as reported by Piacentini et al. (Piacentini et al., 2001).

4.4. *Radio frequency electromagnetic fields (RF-EMFs)*

RF-EMFs in the wireless communication-related frequency bands have an effect in promoting cellular senescence, which had even been observed in plants. However, the effects of RF-EMFs in other frequency bands were different. The different effects might also depend on the frequency and/or intensity of RF-EMFs.

4.4.1. *Frequency-dependent effect of RF-EMFs on cellular senescence*

RF-EMFs in wireless communication-related frequency bands, 1760 MHz (Choi et al., 2020; Kim et al., 2021b), 2998.5 MHz (Alessio et al., 2019), and 866–868 MHz (Czerwiński et al., 2023), have been reported

Table 2
The effect of EMFs on senescence.

EMFs	Main parameters	Exposure time (model)	Organism	Results	References
SMFs	500 mT, without shielding	7 d (consecutive)	Human adipose-derived MSCs	Anti-senescence	(Maredziak et al., 2017)[a]
	100–500 mT, without shielding	48 h (consecutive)	D-gal treated rat pheochromocytoma PC12 cells		(Fan et al., 2023)
RMFs	200 mT, 4 Hz rotating	3 d (2 or 4 h/d)	HUVECs	Anti-senescence	(Xu et al., 2019)[a]
ELF-EMFs	1.5 mT, 25 Hz pulse	40 d (8 h/d)	*Saccharomyces cerevisiae*	Pro-senescence	(Mercado-Saenz et al., 2019)
	20 mT, 50 Hz sine	21 d (30 min/d)	Rat adipose-derived MSCs		(Fathi et al., 2017)[a]
	1.0 mT, 60 Hz sine	4 h (consecutive)	Human breast epithelial cells (MCF10A)	No significant effect	(Hong et al., 2012)
	2.45 mT, 50 Hz sine	40 d (consecutive)	*Saccharomyces cerevisiae*		(Mercado-Saenz et al., 2019)
	1.0 mT, 10 Hz pulse	2 weeks (24 h/2 d)	Human foetal lung fibroblasts		(Sun et al., 2021)
	3.8 mT, 8 Hz pulse	4 weeks (1 h/d)	Senescent $LepR^+$ bone marrow mesenchymal stromal cells induce by glucocorticoids (*in vivo*)	Anti-senescence	(Wang et al., 2022)[a]
	0.1 mT, 50 Hz sine	2 weeks (consecutive) in the dark	*Cucumis sativus* L. etiolated seedlings		(Piacentini et al., 2001)
RF-EMFs	4.0 W/kg, 1760 MHz	4 d (2 h/d)	Human keratinocytes (HaCaT)	Pro-senescence	(Kim et al., 2021b)
	4.0 W/kg, 1760 MHz	4 d (4 h/d)	Neuroblastoma cells (SH-SY5Y)		(Kim et al., 2021a)
	2.0 W/kg, 1760 MHz	72 h (consecutive)	Human ACSs, human hepatocellular carcinoma stem cells (Huh7)		(Choi et al., 2020)
	1.0 W/kg, 1760 MHz	72 h (consecutive)	Human ACSs, human hepatocellular carcinoma stem cells (Huh7)	No significant effect	(Choi et al., 2020)
	0.4–8.0 W/kg, 1760 MHz	72 h (consecutive)	Human ACSs, human hepatocellular carcinoma stem cells (Huh7), Human adenocarcinoma HeLa cells, B103 rat neuroblastoma cells	No significant effect	(Goh et al., 2024)
	0.0124–0.0167 W/m^2, 866–868 MHz	Lifespan	*Trifolium arvense*	Pro-senescence	(Czerwiński et al., 2023)
	3.75 W/m^2, 154–162 MHz, 1250 Hz pulse	An observational study	*Pine trees*		(Selga and Selga, 1996)
TTfields	1.33 V/m, 200 kHz	72 h	Human derived MSCs	Pro-senescence	(Strack et al., 2024)
Electric currents	100 $\mu A/mm^2$, 448 kHz	Up to 48 h (5 min on/ 4 h off)	Human dermal fibroblasts	Anti-senescence	(Toledano-Macías et al., 2024)

[a] Studies without background field data.

to have effect in promoting cellular senescence. Exposure to 1760 MHz RF-EMFs at 4.0 W/kg for 4 days induced significant senescence in a human keratinocyte cell line (HaCaT) (Kim et al., 2021b) and neuroblastoma cell line (SH-SY5Y) (Kim et al., 2021a). Exposure to 1760 MHz RF-EMFs at 2.0 W/kg for 3 days also induced senescence in human adipose-derived stem cells (ACSs) and human hepatocellular carcinoma stem cells (Huh7), as indicated by increased SA-β-gal activity and p21 expression (Choi et al., 2020), however, the pro-senescence effect of 1760 MHz RF-EMFs were disappeared after they had the exposure system updated (Goh et al., 2024). Reactive oxygen species (ROS) generation (Choi et al., 2020; Kim et al., 2021b) and Akt/mTOR signalling pathway activation (Kim et al., 2021a) may be the main mechanisms involved. RF-EMFs had been reported to have an effect in promoting plants senescence. Exposure to 866–868 MHz exhibited pro-senescence effect on *Trifolium arvense* plants that promote the plant entry into the senescence phase but had no effect on the other 9 wild plants (Czerwiński et al., 2023). An observational study also reported that RF-EMFs (154–162 MHz) emitted from radio location station would promote senescence of nearby pine trees (Selga and Selga, 1996).

4.4.2. Intensity-dependent effect of RF-EMFs on cellular senescence

The effect of RF-EMFs on cellular senescence may also depend on the intensity of RF-EMFs, as high-intensity RF-EMFs are more likely to induce cellular senescence. Exposure to 1760 MHz RF-EMFs at 2.0 W/kg for 72 h increased ROS levels and induced cellular senescence in human ACSs and human Huh7 cell lines, but this effect was not observed when the intensity was reduced to 1.0 W/kg (Choi et al., 2020).

4.5. Other EMFs

Exposure to tumour treating fields (TTfields, 1.33 V/cm, 200 kHz) for 72 h could induce apoptosis and senescence in mesenchymal stromal cells (MSCs) (Strack et al., 2024). Unlike the electric fields, electric currents (448 kHz) reduced cellular senescence markers in three types of human skin fibroblasts (Toledano-Macías et al., 2024).

5. Cellular senescence and age-dependent bioeffects and health impacts of EMFs

Ageing can make the body or cells becoming fragile and more sensitive to extremal stimuli. Therefore, it is important to know whether ageing can also cause the human body to become sensitive to EMFs, and the potential health impact. To review the age-dependent difference on the effects of EMFs, only studies on both young and senescent cells as well as individuals at different age groups were included (Fig. 4 and Table 3).

5.1. Statistical magnetic fields (SMFs)

5.1.1. Cellular senescence-dependent effect of SMFs on cell apoptosis and calcium signalling

In isolated human lymphocytes, exposure to 6 mT SMFs could affect the apoptotic rate (spontaneously and drug-induced) and calcium signalling, and aged (5 days old) cells were more susceptible than freshly isolated (1 day old) cells (Tenuzzo et al., 2009). This result indicated that cellular senescence is an important factor in determining the bioeffects of SMFs on cell apoptosis and calcium signalling.

5.1.2. Age-dependent effect of SMFs on tissue hydration

Cell hydration is one of the fundamental metabolic factors that determines not only the functional activity of a cell (Häussinger, 1996a; Häussinger, 1996b) but also its sensitivity to various environmental factors (Ayrapetyan, 2012), and cell hydration was suggested to be a primary responder that may mediate the biological effect of EMFs (Ayrapetyan, 2006; Ayrapetyan et al., 1994; Danielyan and Ayrapetyan, 1999; Danielyan et al., 1999). Animal studies have shown that SMFs

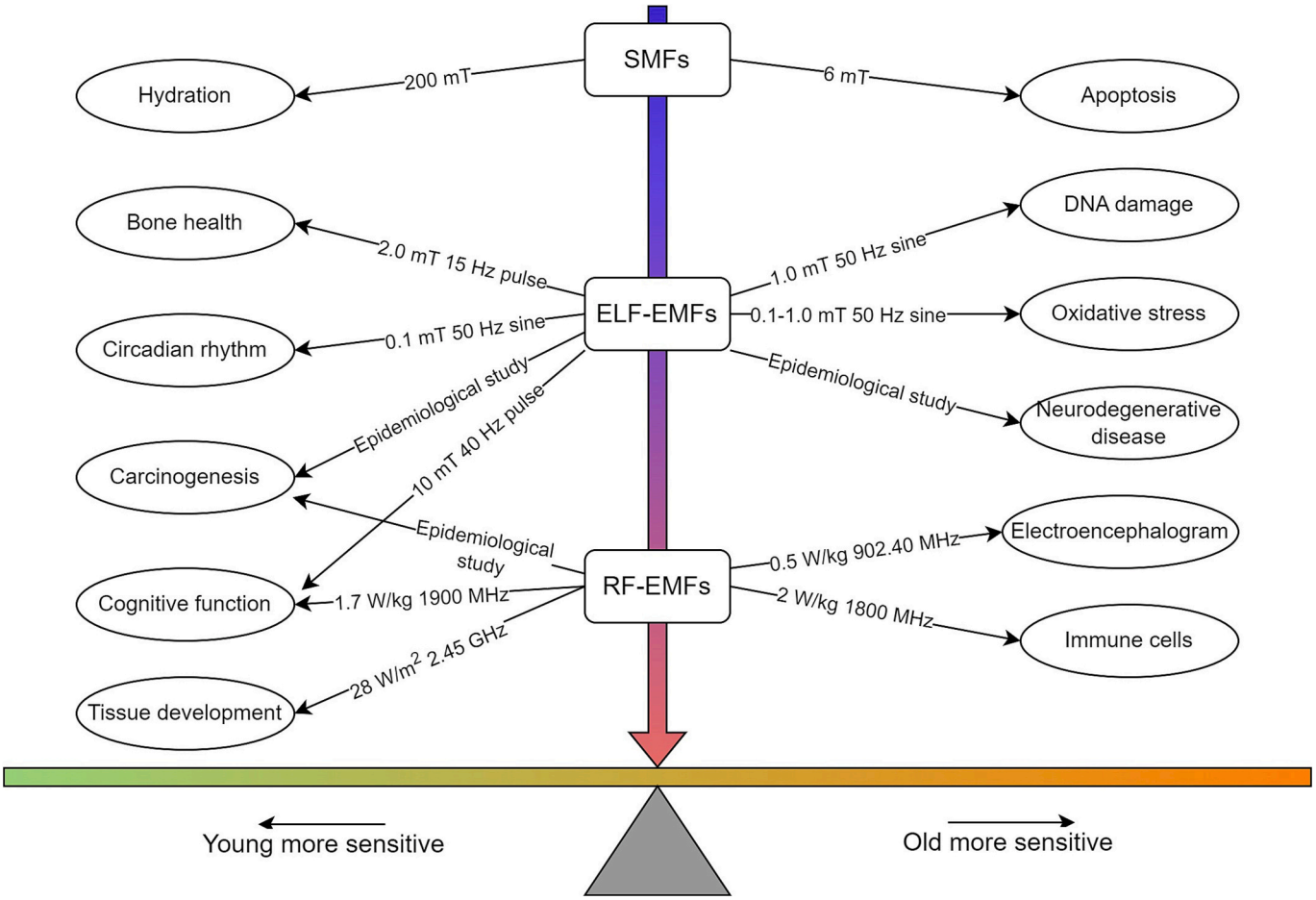


Fig. 4. Age-dependent bioeffects of EMFs. Age-dependent bioeffects of SMFs, ELF-EMFs, and RF-EMFs have been observed in *in vitro*, *in vivo*, and epidemiological studies; however, the effects are different, and the underlying mechanisms are unclear.

have an age-dependent effect on brain and heart muscle hydration. Exposure to 6 mT SMFs for 15 min led to a decrease in the water content in the brain tissues of young (6-week-old) male albino rats but an increase in adult (18-week-old) and aged (18-month-old) rats (Deghoyan et al., 2014). Heart muscle hydration was more sensitive to SMF exposure in young (6-week-old) rats than in older (12-month-old) rats (Narinyan et al., 2012; Narinyan et al., 2013). The initial state of tissue hydration may play a crucial role in animal age-dependent MF sensitivity, possibly due to age-dependent Na^+/K^+ pump dysfunction (Deghoyan et al., 2014) and decreased expression of high-affinity ouabain receptors (Narinyan et al., 2013). Thus, the different effects of SMFs on tissue hydration may lead to age-dependent health impacts. However, the background field data and the specific time of conducting the experiments (the time period of the day) were not mentioned in these studies, which might have an impact on the results.

5.2. Extremely low frequency magnetic fields (ELF-EMFs)

5.2.1. Age-dependent effect of ELF-EMFs on human-derived primary cells

Cells derived from human body of different ages respond differently to ELF-EMFs. The effect of 1.0 mT 50 Hz MF exposure on DNA damage in cultured fibroblasts was clearly more pronounced in older healthy donors compared to young donors, and age-related decrease in DNA repair efficiency may be the reason (Ivancsits et al., 2003). An increase in the impact of ELF-EMFs on cells due to senescence-related repair deficiency was also reported in yeast, that exposure to 25 Hz pulsed MFs at 1.5 mT induced significant damage in senescent (40 days) yeast cells but had no significant effect on young (0 or 20 days) cells (Mercado-Sáenz et al., 2023). However, senescence-related dysfunction cannot explain everything. Nam et al. reported that exposure to 25 Hz pulsed MFs at 70 % of the maximum power (3.0 T) for 5 min could increase fibroblastic colony formation and cell proliferation in bone marrow-derived MSCs from older patients compared with those from young patients *via* transient nitric oxide production and extracellular signal-regulated kinase 1/2 activation, but this effect diminished when the exposure time was extended to 10 or 15 min (Nam et al., 2020).

5.2.2. Age-dependent effect of ELF-EMFs on oxidative stress

ELF-EMFs are more likely to induce oxidative stress in the brain tissue of older animals. Exposure to 50 Hz MFs at 0.1 mT for 10 days significantly inhibited antioxidant defence systems and increased ROS in aged rat brains, which were reversed in young rats (Falone et al., 2008). Exposure to 50 Hz MFs at 0.1–0.5 mT for 7 days induced more oxidative stress in all tested brain regions in middle-aged (10-month-old) Mongolian gerbils than adult (3-month-old) ones, and the effect still existed 3 days after exposure in middle-aged but not in adult gerbils (Selakovic et al., 2013). There are no explanations for why older animals are more sensitive to ELF-EMFs induced oxidative stress. Sun et al. reported age-dependent dysfunction of mitochondria play a role in sensitizing cells to 50 Hz MFs (Sun et al., 2025); however, this need to be verified by animal studies.

5.2.3. Age-dependent correlation of ELF-EMFs with neurodegenerative diseases

Oxidative stress is a risk factor for neurodegenerative diseases (Zhang and Butterfield, 2017), and evidence from animal studies has

Table 3
Age-dependent bioeffects of EMFs.

EMFs	Research type	Main parameters	Cells	Endpoints	More sensitive	References
SMFs	*In vitro*	6 mT, 24 h, without shielding	Aged (5 days old) and fresh (1 day old) human lymphocytes	Cell apoptosis	Old	(Tenuzzo et al., 2009)
	In vivo (animal studies)	200 mT, 15 min, without shielding	Young (6 weeks), adult (18 week), aged (18 months) male albino rats	Effect on brain tissue hydration	Reversed effect in young and aged animals, no effect in adult animals	(Deghoyan et al., 2014)[a]
		200 mT, 15 min, without shielding	Young (6 weeks), adult (4 months), and older (12 months) albino rats	Effect on heart muscle hydration	Young	(Narinyan et al., 2012)[a]
ELF-EMFs	*In vitro*	70 % of the maximum power (3000 mT), 25 Hz pulse, 5 min	Bone marrow derived MSCs from older and young patients	Induction of colony formation and cell proliferation	Old	(Nam et al., 2020)[a]
		1.0 mT, 50 Hz sine, 15–19 h	Fibroblasts from different age donors	Induction of DNA strand breaks		(Ivancsits et al., 2003)
		1.5 mT (peak), 25 Hz pulse, 8 h/day 40 d	*S. cerevisiae*	Cell survival		(Mercado-Sáenz et al., 2023)
		1.0 mT, 50 Hz sine, 24 h	*Human foetal lung fibroblasts*	Induction of DNA damage and oxidative stress		(Sun et al., 2025)
		2.45 mT, 50 Hz sine, continuously for 40 d	*S. cerevisiae*	Cell survival	No significant effect	(Mercado-Sáenz et al., 2023)
	In vivo (animal study)	2.0 mT, 15 Hz pulse, 2 h/d for 8 consecutive weeks (without daily exposure time)	5- and 20-month-old gonad-intact male Sprague–Dawley rats	Increased bone mass, strength, and anabolism	Young	(Cai et al., 2020)
		0.1 mT, 50 Hz sine, 18 h/d (14:00 to 08:00) for one week	9-week and 23-month-old male Wistar rats	Decreased serum melatonin levels		(Selmaoui and Touitou, 1999)
		10 mT (peak value), 40 Hz (5 Hz pulse), 1 h/d for 8 consecutive weeks	10-week and 18-week-old female 5xFAD mice	Improved learning and memory abilities		(Wang et al., 2025)
		0.65 mT, 50 Hz sine, 21 d (continuous)	Newborn CD-1 mice (Pregnancy to 3 days after birth) and adult CD-1 mice	Clastogenicity and aneuploidy		(Udroiu et al., 2006)[a]
		0.1 mT, 50 Hz sine, continuously for 10 d	3- and 19-month-old female Sprague–Dawley rats	Increased brain ROS	Old	(Falone et al., 2008)
		0.1, 0.25, 0.5 mT, 50 Hz sine, continuously for 7 d	3- and 10-month-old Mongolian gerbils	Increased oxidative stress in brain		(Selakovic et al., 2013)
	In vivo (human trial)	Transcranial magnetic stimulation (TMS), 90 pairs of TMS pulses were delivered continuously at a rate of 0.1 Hz for 15 min	Young adults (24 ± 3 years), elderly adults (72 ± 6 years)	Increased action performance and corticomotor excitability	Young	(Turrini et al., 2023)
		Intermittent theta burst stimulation (iTBS) 50 Hz, each burst was repeated at 5 Hz for 2 s, and repeated every 8 s for 20 cycles, totalling 600 pulses	Young adults (24.7 ± 5.0 years), elderly adults (67.0 ± 5.3 years)	primary motor cortex plasticity	No difference	(Liao et al., 2024)
	Epidemiological studies	50–60 Hz power frequency	<75 years and >75 years	Increased deleterious effect of MF exposure	Old	(Davanipour et al., 2014)
			<75 years and >75 years	Association with neurodegenerative disease		(Frei et al., 2013)
			<75 years and >75 years	Association with neurodegenerative disease	Young	(Andel et al., 2010)
			<40 years and >40 years	Association with testicular cancer		(Baumgardt-Elms et al., 2005)
			<15 years and >15 years	Association with MPDs		(Lowenthal et al., 2007)
			<15 years and >15 years	Association with melanoma		(Khan et al., 2022)
RF-EMFs	*In vitro*	1800 MHz, 2 W/kg, 44 h (10 min on/20 min off)	Stimulated CD4+ T lymphocytes from young (26 ± 5 years) and elderly (88 ± 2 years) donors	Downregulation of CD95	Old	(Capri et al., 2006)
	In vivo (animal study)	900 MHz, 6.0 W/kg, 15 min (at 9:00 to 12:00)	Middle-aged (12-month-old) and young (6-week-old) rats	Increased IL-1β in the olfactory bulb and improved contextual memory	Old	(Bouji et al., 2012)
		2450 MHz, 28 W/m^2 short-term exposure (2 d, 4 h/d) or long-term exposure (3 d, 8 h/d)	Newborn (P7) and senescent (24 months) rats	Proliferation of rostral migratory stream	Young	(Orendacova et al., 2009)[b]
		1950 MHz, 5 W/kg, 8 months (2 h/day, 5 d/week)	14-month-old C57BL/6 mice	Influence of age on induction of oxidative stress or neuroinflammation	No effects	(Jeong et al., 2018)
		35 GHz, 13 W/kg, continued until death	Young (3–4 month), middle-aged (15–16 month), and old (24–25 month) Male Sprague–Dawley rats	Thermal distribution and cardiorespiratory responses	No difference	(Ryan et al., 1997)

(*continued on next page*)

Table 3 (*continued*)

EMFs	Research type	Main parameters	Cells	Endpoints	More sensitive	References
	In vivo (human trial)	902.40 MHz, 0.5 W/kg 45 min	Elderly (47–84 years) and young (20–37 years) healthy people	Increase in the interhemispheric coherence of frontal and temporal alpha rhythms	Old	(Vecchio et al., 2010)
		894.6 MHz, 0.7 W/kg (peak)	Adolescents (13–15 years), young adults (19–40 years), older adults (55–70 years)	Effect on resting alpha activity	Young	(Croft et al., 2010)
		1900 MHz, 1.7 W/kg (peak)	Adolescents (13–15 years), young adults (19–40 years), older adults (55–70 years)	Effect on sensory and cognitive processing		(Leung et al., 2011)
		894.6 MHz, 0.7 W/kg (peak)	Adolescents (13–15 years), young adults (19–40 years), older adults (55–70 years)	Effect on sensory and cognitive processing	No difference	(Leung et al., 2011)
		1900 MHz, 1.7 W/kg (peak)	Adolescents (13–15 years), young adults (19–40 years), older adults (55–70 years)	Effect on resting alpha activity	No effects	(Croft et al., 2010)
		900–915 MHz (217 Hz pulse), 2 W/kg, 385 MHz (17.6 Hz pulse), 6.0 W/kg	Young (18–30 years) and older (>60 years) adults	Effect on sleep		(Eggert et al., 2020)
	Epidemiological studies	Use of wireless phones	18- to 65-year-old people	Associations of wireless phone use and the serum concentration of β-trace protein	Young	(Soderqvist et al., 2012)
			20- to 80-year-old people	Association with brain tumours		(Hardell et al., 2004)

[a] Studies without background field data.
[b] Studies without temperature data and animal culture condition.

indicated that ELF-EMFs may be more likely to cause neurodegenerative diseases in older individuals; coincidentally, this finding is supported by epidemiological studies. A population-based case–control study investigated severe cognitive dysfunction and occupational ELF-EMF (50–60 Hz) exposure among elderly (>65 years) Mexican Americans and found that when the analyses were limited to individuals aged ≥75 years, the interactions between medium or high occupational ELF-EMF exposure and severe cognitive functional impairment were statistically significant, indicating that older age may increase the deleterious effect of ELF-EMF exposure (Davanipour et al., 2014). A case–control study conducted in Denmark to investigate the possible association between residence distance from high-voltage power lines and neurodegenerative diseases, especially Alzheimer's disease, revealed no dose–response relationship according to the number of years living within 50 m of a power line, but there were weak indications of increased risk for persons diagnosed by the age of 75 years (Frei et al., 2013). Thus, epidemiological evidence suggests that old age is a risk factor for ELF-EMF-associated neurodegenerative diseases. But there were also studies with different results. A population-based study of dementia in Swedish Twins showed that high levels ELF-EMF exposure were associated with increased dementia risk in cases with onset by age 75 years (Andel et al., 2010). Furthermore, these age-dependent differences are also reflected in the therapeutic effects of EMFs, that exposure to 40 Hz pulsed MFs improved cognitive performance in 10-week-old but not in 18-week-old Alzheimer's disease model mice (Wang et al., 2025).

5.2.4. Age-dependent correlation of ELF-EMFs with carcinogenesis

In contrast to neurodegenerative diseases, epidemiological studies indicated that younger age is a risk factor for ELF-EMF-associated carcinogenesis. A population-based case-control study revealed no substantial association between residence distance from high-voltage lines and the risk of testicular cancer; however, the risk for young men younger than 40 years was slightly increased (Baumgardt-Elms et al., 2005). A case–control study indicated that prolonged residence close to high-voltage power lines, especially early in life (0–15 years old), may increase the risk of developing myeloproliferative disorders (MPDs) and lymphoproliferative disorders (LPDs) (Lowenthal et al., 2007). A cohort study indicated that for residential exposure to ELF-EMFs, a non-significantly increased risk for melanoma and a non-significantly decreased risk for squamous cell carcinoma, but analysis of the age at the start of residence showed a statistically significant increased risk (HR 2.55; CI 1.15–5.69) for melanoma among those who lived in apartments when they were <15 years old (Khan et al., 2022). In addition to epidemiological investigations, an animal experiment study showed that exposure to 50 Hz MFs has obvious genotoxicity on neonatal mice, but has no significant effect on adult mice (Udroiu et al., 2006). Therefore, it appears that young age is a risk factor for carcinogenesis possibly associated with ELF-EMFs exposure, and this age-dependent effect should be a focus of further research.

5.2.5. Age-dependent effect of ELF-EMFs on bone health and the circadian rhythm in animal studies

There are several noteworthy bioeffects of ELF-EMFs that are also age dependent. Animal studies showed that exposure to 15 Hz pulsed EMFs at 2.0 mT improved bone health, as indicated by changes in bone mass, strength and anabolism, in 5-month-old rats but not in 20-month-old rats (Cai et al., 2020), indicating that the effect of pulsed EMFs on promoting bone health is greater at a young age. Exposure to 50 Hz MFs at 0.1 mT altered the production of serum melatonin through inhibiting pineal *N*-acetyltransferase (NAT) activity in young rats, whereas no effect was observed in older rats (Selmaoui and Touitou, 1999). Melatonin is closely related to the circadian rhythm; thus, a young biological clock may be more easily disturbed by ELF-EMF exposure. However, further studies are needed.

5.2.6. Age-dependent effect of ELF-EMF treatment

Age-dependent bioeffects of ELF-EMFs have also been observed in humans. Sonia et al. reported that transcranial cortico-cortical paired associative stimulation (ccPAS) over the ventral premotor-motor pathway (0.1 Hz for 15 min) improves action performance and corticomotor excitability in young adults (24 ± 3 years) more than in elderly adults (72 ± 6 years) (Turrini et al., 2023). Differences in the effects of electromagnetic stimulation at different ages may have an impact on the effectiveness of treatment. However, Liao et al. reported that intermittent theta burst stimulation (0.1 Hz) do not have different effects on primary motor cortex plasticity in young and older adults (Liao et al., 2024). As the application of EMFs in the treatment of human diseases is a very complex matter and these studies cannot provide precise

electromagnetic dosages, further research is still needed in this area.

5.3. Radio frequency electromagnetic fields (RF-EMFs)

5.3.1. Age-dependent effect of RF-EMFs on immune cells

There are few studies on the age-dependent effect of EMFs on immunity, but it was found that exposure to 1800 MHz RF-EMFs at 2 W/kg induced slight but significant downregulation of CD95 expression in stimulated $CD4^+$ T lymphocytes from elderly but not young donors (Capri et al., 2006), indicating that the immune system of older people may be more sensitive to RF-EMFs.

5.3.2. Age-dependent effect of RF-EMFs on the nervous system

Exposure to 2450 MHz GHz RF-EMFs at 28 W/m^2 could induce dose-dependent changes on the rostral migratory stream proliferation in newborn rats, but for older rats, the changes in proliferation activity and dynamics were negligible (Orendacova et al., 2009), suggesting young individuals at the neurodevelopmental stage possibly being more affected by RF-EMFs exposure. Exposure to 1950 MHz RF-EMFs at 5 W/kg for 8 months (2 h/day, 5 days/week) did not influence oxidative stress or neuroinflammation occurred during ageing in mice (Jeong et al., 2018), suggesting that older animals are also not sensitive to RF-EMFs exposure on oxidative stress in the nervous system. However, the results would be different under different experimental conditions. Exposure to 900 MHz RF-EMFs at 6.0 W/kg significantly increased IL-1β in the olfactory bulb and improved contextual memory in middle-aged (12-month-old) rats but increased plasma levels of corticosterone in young (6-week-old) rats (Bouji et al., 2012). Exposure to 900 MHz RF-EMFs at 0.7–10 W/kg had no significant on the development and differentiation processes of the central nervous system in Wistar rats at the juvenile, adult, or presenile stage (aged 14 days to 19 months) (Klose and Lerchl, 2013). Therefore, the age-dependent effect of RF-EMFs on the nervous system are inconsistent.

5.3.3. Age-dependent effect of RF-EMFs on cell proliferation

RF-EMFs may have an age-dependent effect on cell proliferation. Exposure to 2450 MHz RF-EMFs at 28 W/m^2 significantly affected the rostral migratory stream proliferation in newborn rats but not aged rats (Orendacova et al., 2009), and exposure to 4 MHz RF-EMFs at a high energy to increase the skin temperature up to 3 and 4 degrees activated fibroblast proliferation in the tail of young (2 months) but not older (12 months) rats (Beltran-Frutos et al., 2021). These studies indicated that RF-EMFs exposure has an effect on cell proliferation, and young individuals at the developmental stage are more sensitive.

5.3.4. Age-dependent effect of RF-EMFs on human brain electrical activity

The effects of cell phone radiation on brain electrical activity have been extensively studied, but the results have been inconsistent. Some suggested that humans at young age are more sensitive. Exposure to second-generation (2G; 894.6 MHz modulated at 217 Hz, <0.7 W/kg) mobile phone signals significantly increased alpha activity in young adults (19–40 years) but not in adolescents (13–15 years) or elderly adults (55–70 years) (Croft et al., 2010). This effect was not observed in the third-generation (3G; 1900 MHz, <1.7 W/kg) mobile phone signal group (Croft et al., 2010), but the impaired performance induced by 2G and 3G signals was found to be stronger in adolescents (Leung et al., 2011). Some studies showed that elderly people are more sensitive. Vecchio et al. reported that, compared to young individuals (20–37 years), elderly individuals (47–84 years) were more sensitive to RF-EMFs that exposure to 902.40 MHz RF-EMFs at a maximum specific absorption rate (SAR) of 0.5 W/kg significantly increased the inter-hemispheric coherence of frontal and temporal alpha rhythms (Vecchio et al., 2010). Brain electrical activity is closely related to sleep, although no age-dependent effects of RF-EMF exposure signals (GSM900 at 2 W/kg, TETRA at 6 W/kg) on sleep were observed (Eggert et al., 2020), long-term wireless phone use was negatively associated with the serum concentration of β-trace protein (lipocalin-type prostaglandin D synthase), a key enzyme in the synthesis of prostaglandin D(2), an endogenous sleep-promoting neurohormone, in the youngest age group (18–30 years) but not in all age groups (18–65 years) (Soderqvist et al., 2012). Therefore, RF-EMFs have an age-dependent effect on brain electrical activity, but the health consequences and the underlying mechanisms are unknown.

5.3.5. Age-dependent correlation of RF-EMFs with carcinogenesis

The link between RF-EMFs exposure and risk of carcinogenesis has been widely studied, but this remains inconclusive. An investigation on the association of cellular and cordless telephone use and brain tumours in different age groups (20–80 years) revealed the greatest risk in the 20–29-year age group (Hardell et al., 2004), indicating that young age may be a risk factor on brain tumours under RF-EMFs exposure, and age-dependent effects cannot be ignored to elucidate the relationship between RF-EMFs and cancer.

5.3.6. Age-dependent variations on the biophysical properties of the RF-EMFs

Thermal effects are one of the important mechanisms of RF-EMF-induced biological effects which are mainly dependent on the biophysical properties of the tissue. Therefore, the age-dependent effect of RF-EMFs may be caused by changes in the biophysical properties during ageing. The dielectric properties of tissues at various cell phone frequencies vary with age due to changes in the water content and tissue composition of tissues (Peyman et al., 2001). These differences could result in an age-dependent increase in electromagnetic power and heat deposition in near-surface tissues exposed to 26- and 60-GHz RF-EMFs, in which the transmitted power density (electromagnetic power deposition and resulting heating in the near-surface tissues) increases with age, while the peak SAR decreases, and the peak steady-state heating increases from 5 to 70 years of age by approximately 11 % at 26 GHz and 13 % at 60 GHz (Sacco et al., 2021). Therefore, age-related changes in tissue biophysical properties may be an important cause of the age-dependent biological effects of RF-EMFs. However, Ryan et al. reported that age did not alter thermal or cardiorespiratory responses to 35 GHz RF-EMFs exposure in food-restricted rats (Ryan et al., 1997). Moreover, at cell phone signal bands, no age-dependent changes in the peak spatial SAR were detected when averaged over the entire head of cell phone users (Christ et al., 2010). Therefore, the age-dependent biological effects of RF-EMFs cannot simply be attributed to changes in biophysical properties.

6. Conclusion

With the intensification of ageing and the variety of EMFs in the environment continues to increase, the effects of EMFs on ageing and ageing-dependent effects of EMFs will get constant attention. Although current research is insufficient to answer this question, some important information can be gleaned. The ageing state of the cell or body plays a role in determining the biological or health effects of EMF exposure, but the exact effect depends on the specific conditions, and further investigation of the underlying mechanism is needed.

6.1. The effect of EMFs on ageing

Ageing is a complex and expanding concept (López-Otín et al., 2023); therefore, understanding the effects of EMFs on ageing is not to be done in a short time, but we can gain insights into this mystery from the study of lifespan and cellular senescence. SMFs have an effect on delaying cellular senescence (Fig. 3) and extending the lifespan of *C. elegans* and mice (Fig. 2); however, SMFs at high flux intensity had negative effects on *C. elegans* (Fig. 2). RMFs have both beneficial effects on anti-cellular senescence and prolong the lifespan of *C. elegans* (Figs. 2 and 3). The impact of ELF-EMFs on lifespan and cellular senescence had

been found to be inconsistent, potentially dependent on wave shape and/or magnetic flux density (Figs. 2 and 3). Exposure to RF-EMFs in the wireless communication bands (1800 MHz–2480 MHz) appears to have no significant impact on lifespan (Fig. 2). However, these RF-EMFs may influence cellular senescence in a frequency- and intensity-dependent manner (Fig. 3).

6.2. Ageing-dependent effect of EMFs

With the global ageing of the population, it is very meaningful to understand the age-dependent health effects of EMFs. Although the current research is still in the preliminary stage, the biological effects of SMFs, ELF-EMFs, and RF-EMFs have been reported to be age dependent (Fig. 4). SMFs have an age-dependent effect on cell apoptosis, calcium signalling and tissue hydration. ELF-EMFs have an age-dependent effect on human-derived primary cells and brain oxidative stress in animals, and epidemiological studies have shown that the relationship between ELF-EMFs and the risk of neurodegenerative disease is greater in older individuals and that the risk of carcinogenesis is greater in young individuals. RF-EMFs have an age-dependent effect on immune cells, tissue development, and brain activity but do not interfere with sleep. Importantly, RF-EMFs are more strongly related to the risk of carcinogenesis in younger age groups.

6.3. Possible mechanisms for the age-dependent effect of EMFs

Cellular senescence is the basis of ageing, so the age-dependent effect may be fundamentally due to cellular senescence (van Deursen, 2014). A deeper understanding of the molecular mechanism underlying the interactions between EMFs and cells may lead to new findings. Free radical metabolism and calcium signalling have mostly been studied for their ability to mediate the bioeffects of EMFs; thus, age-related functional changes in these two aspects might be the cause of age-dependent effects (Falone et al., 2008; Selakovic et al., 2013). Existing studies have shown that other possible mechanisms, such as age-dependent Na^+/K^+ pump dysfunction (Deghoyan et al., 2014) and decreased expression levels of high-affinity ouabain receptors (Narinyan et al., 2013), play important roles in the age-dependent effect of SMFs on tissue hydration. The effect of EMFs is also dependent on the biophysical properties of tissues or cells, and ageing-related differences in the biophysical properties that result in different responses to EMFs may also be the cause of age-dependent effects.

CRediT authorship contribution statement

Xiaoxia Wei: Writing – review & editing, Writing – original draft, Conceptualization. **Yun Huang:** Funding acquisition, Investigation, Validation, Visualization. **Chuan Sun:** Writing – review & editing, Writing – original draft, Validation, Funding acquisition.

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Declaration of competing interest

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Data availability

No data was used for the research described in the article.

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