

This **special issue** of the BERENIS newsletter contains an up-to-date assessment of a possible correlation between oxidative stress and exposure to magnetic and electromagnetic fields and their putative effects on health. For this purpose, relevant animal and cell studies published between 2010 and 2020 were identified and summarized. An extended report presenting these recent studies in more detail will be published soon by the FOEN¹. This special issue contains a short version of the report.

Is there evidence for oxidative stress caused by electromagnetic fields?

A summary of relevant observations in experimental animal and cell experiments related to health effects in the last ten years

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Introduction and aims of this report

Reactive oxygen species (ROS) are involved in many processes of the organism, including cellular signalling pathways; therefore, physiological concentrations of ROS in cells need to be maintained by engaging protective mechanisms (antioxidative enzymes and antioxidants). On the other hand, external and internal factors influence the amount of ROS by altering the activity of the ROS-forming and -degrading enzymes. For instance, the increased energy requirement during physical activity leads to a temporary oxidative stress and many environmental stress factors such as UV light or radioactive irradiation act via ROS formation. An oxidative imbalance has an effect on many important physiological processes and functions, such as inflammation, cell proliferation and differentiation, wound healing, neuronal activity, reproduction and behaviour by altering biochemical processes or even leading to DNA damage or peroxidation of fats. In particular, changes in cell proliferation and differentiation are closely related to carcinogenesis and the growth and development of organisms.

Concerning putative adverse health impact, the influence of electromagnetic fields (EMF), as an environmental factor, on the formation of ROS and triggers of oxidative stress was considered. Respective hypotheses and experimental findings were summarised and discussed in scientific reviews as well as in reports of public organisations [1-12]. Despite some evidence of ROS formation and oxidative stress by EMF, a scientific consensus is not yet achieved, especially when it comes to possible negative and long-term effects on our health.

Aiming at an up-to-date assessment of a possible correlation between oxidative stress and exposure to magnetic and electromagnetic fields and their putative effects on health, this topic is revisited here by identifying and summarizing recent and relevant animal and cell studies. This primarily involves experimental data of animals and cultured cells from peer-reviewed publications (about 150 studies in total) in the period 2010-2020. An extended report presenting these recent studies in more detail will

¹ The report will be available on the FOEN website at:
<https://www.bafu.admin.ch/bafu/en/home/topics/electrosmog/publications-studies.html>

be published soon on the FOEN website. Herein, the focus was set on publications dealing with EMF in the frequency ranges relevant to the environment and technology: extremely-low-frequency magnetic fields (ELF-MF), typical for 50/60 Hz alternating current power lines, as well as radio-frequency electromagnetic fields (RF-EMF) from 800 MHz to 2.5 GHz as used for mobile communication systems. The experimental studies investigated the influence of EMF exposure on the formation of ROS, markers of oxidative stress, and changes in the protective mechanisms that counteract oxidative stress. These publications may be of a purely descriptive nature, while others may contain mechanistic aspects that specifically track and investigate interrelationships and influenced processes. It should be noted that not only primary but also established and cancerous cells have been used to study the formation and occurrence of ROS and related changes in cellular signalling pathways and protective mechanisms. In animal studies, the balance of ROS and the antioxidative mechanisms can be studied in the whole organism. In contrast to cell culture studies, functional changes caused by a permanent oxidative imbalance can be investigated in animals, providing more relevant data for the evaluation of health impacts. Therefore, animal studies in which functional changes are considered and assessed are particularly important for the assessment of the impact of EMF on human health.

General information on oxidative stress

The chemical oxidation and reduction reactions are the basis for all biochemical processes that make biological activities and life possible. The relatively reactive molecular oxygen in our atmosphere plays a key role in generating energy from sunlight and in the conversion of this energy through cellular respiration in the mitochondria to make it available for other biological processes. For the functionality of cells and the organism, it is therefore essential that the reducing and oxidising molecules are in balance. This is known as redox equilibrium, which is controlled and maintained by the cell's own sensors, signalling pathways and defence mechanisms. If imbalanced in direction of an increase in oxidative processes, we speak of oxidative stress [13, 14]. If this state persists over a longer period of time or occurs repeatedly, it can lead to changes in the biological material and thereby to health-related malfunctions of cells and organs. Thus, increases in biomarkers for oxidative stress, either as cause or consequences, are observed in many diseases, such as cancer, diabetes, congenital malformations or neurodegenerative diseases [14, 15].

How oxidative stress occurs

Oxidative stress develops primarily when the amount of reactive oxygen species (ROS) exceeds the neutralisation capacity. Besides the short-lived superoxide (O_2^-) and hydroxyl (OH) radicals, ROS include hydrogen peroxide (H_2O_2) and singular oxygen ($^1\text{O}_2$) as well as some organic compounds [13, 14].

A major source of ROS is the mitochondria, which are cell organelles present in every cell. They play a crucial role in the energy supply and are also called the cells' power stations. ROS are formed as by-product during metabolic processes of the mitochondrial electron transport chain ("respiratory chain") and cytochrome P450 oxidases, in particular the superoxide anion radical O_2^- , hydrogen peroxide (H_2O_2) and the hydroxyl radical OH . It is estimated that in the mitochondrial respiratory chain about 2% of the oxygen consumed is not converted to water but to the superoxide radical. ROS formation and oxidative stress leads to the breakdown of mitochondria, microfilaments and proteins, which lose their functionality through oxidative modifications, leading, for instance, to an impairment of metabolic processes.

Another important ROS source are the NADPH oxidases (NOX). These enzyme complexes consist of multiple subunits and exist in distinct compositions in different cell types [16]. They produce

superoxide radicals from molecular oxygen, which is used, depending on the cell type or organ, to defend against pathogens but also serves as a signal molecule. Accordingly, the NADPH oxidases are either located on the membrane of cells or specific organelles (phagosomes) of macrophages, neutrophil granulocytes, dendritic cells of the immune system, in which trapped microorganisms are killed [17].

In addition to ROS, a reactive nitrogen-containing molecule, the gaseous free radical nitric oxide ($\cdot\text{NO}$), plays a role in immune and other types of cells, and is produced by three types of nitric oxide synthases (NOS) [13, 15]. NO itself is an important messenger substance with a short life span, and is involved in the regulation of the blood circulation by vasodilation, in neuronal functions and in immune defence. While a physiological concentration of NO does not have a cytotoxic effect per se, it can react spontaneously with superoxide to form highly reactive peroxynitrites, which can result in damage to DNA and proteins, but is also engaged in the defence against infections in macrophages.

Protective mechanisms to prevent oxidative stress

Although these reactive molecules can potentially cause damage to biological material and thereby impair its functionality, their presence and production should not be considered harmful per se. As exemplified in the previous chapter, they are even indispensable for some functions and mechanisms [13, 15, 18]. For instance, nitric oxide ($\cdot\text{NO}$) and hydrogen peroxide (H_2O_2) are important for many physiological processes, metabolic processes and immune response. Hydrogen peroxide is needed for wound healing or the correct formation of certain protein structures. It is therefore important that ROS concentrations are kept within certain barriers, which is achieved through the interaction of antioxidants and enzymatic mechanisms of protection against oxidative stress.

Provitamin A, vitamins B and C or glutathione (GSH) act as antioxidants and superoxide radicals ($\cdot\text{O}_2^-$) are immediately converted to hydrogen peroxide by superoxide dismutases (SOD). This family of enzymes is therefore the first antioxidative line of defence to eliminate this by-product of oxygen metabolism or product of activated immune cells [19]. Using metal ions as cofactors, they convert superoxide radicals to the less reactive hydrogen peroxide (H_2O_2). Superoxide dismutases exist in different variants in most living organisms and cell types, acting in the cytoplasm, in the mitochondria but also in extracellular spaces. Furthermore, there are a number of enzymes, such as catalase (CAT), glutathione peroxidases (GPx) and the GSH system, which have an important role in the control of ROS levels [14, 15, 20].

Summary of assessed studies related to EMF and oxidative stress

The majority of animal studies investigating oxidative stress and EMF (ELF-MF and RF-EMF) have been published dealing with possible effects on the nervous system and reproduction, with a predominance for studies on increased ROS production and/or antioxidant protection mechanisms in the brain or specific brain regions. Concurrently, neural cells or neuron-like cells were also most frequently used in cell studies. Animal studies on oxidative stress related to a possible impairment of reproduction and development follow in second place, examining the impact of EMF exposure on various aspects and stages (sperm maturation, early stages of pregnancy such as implantation, effects in foetuses directly after birth and after a few weeks upon exposure of the mother). These animal studies were complemented by some cell studies, mainly executed in mouse cell lines of the male reproductive system and with spermatozoa. Not unexpectedly, more cell studies were published overall. In addition to the above mentioned cell types of the nervous and reproductive system, immune cells and isolated cells from the skin and epithelia were employed. For this summary report, a subset of animal and cell studies that were considered of relevance in terms of quality and research question addressed were

assessed and included, in order to provide an overview of the current research. Hence, it is not a systematic review.

In evaluating animal studies, a distinction between studies that are purely descriptive and those that also report about functional effects such as learning behaviour and memory performance has to be made. The latter provide more profound information on a potential health hazard due to increased oxidative stress caused by EMF exposure. For the assessment of health relevance it is also important to note that, as mentioned at the beginning, ROS formation and temporary oxidative stress is not harmful by nature and necessarily affects health. These reactive molecules are also part of physiological processes and fulfil functions, for example in the immune response or the correct formation of protein structures. Damage with possible health implications only occurs if the oxidative balance (redox equilibrium), which is controlled and maintained by sensors, cellular signalling pathways and protective antioxidative mechanisms, is disturbed over a long period of time, either lastingly and/or repeatedly. If this is the case, various physiological processes such as cell proliferation, neuronal differentiation and activity, immune responses and developmental processes may be affected. Altered production of ROS and reduced antioxidant counter-regulation also occur in processes of ageing. Therefore, models investigating an influence of oxidative balance under EMF exposure are of interest and importance for a possible impact on elderly individuals or those with pre-existing conditions (neurodegeneration, diabetes, etc.).

In the sections below, important aspects of oxidative stress observed in animal and cell studies are discussed with respect to different cells/organs and their implications for the assessment of health effects. General considerations that are independent of cell type and/or organ/tissue but relevant for such an assessment are discussed as well.

Influences of EMF-induced ROS formation and oxidative stress on the nervous system and cognitive abilities

Because of their longevity and limited renewal, neurons are considered to be particularly sensitive to oxidative stress, which, for example caused by chronic inflammation, can lead to cell damage. Furthermore, ROS formation is associated with the aging process as well as with many neurodegenerative diseases [15, 16, 18]. An involvement or contribution of EMF-induced oxidative stress is indeed possible, besides many other factors and environmental influences. However, it should also be noted that the formation of ROS plays a fundamental role in many processes of neuronal development, plasticity and signal transduction to ensure normal functionality [18]. Thus, an increase in ROS formation does not inevitably result in health-related and negative effects.

Mostly small rodents (rats and mice) were used to investigate ROS production, but also the relevant protective mechanisms (antioxidative enzymes), after short or long-term EMF exposure. Unless several animal groups with different exposure durations were employed, notably, it is often not easy to determine whether EMF induces a transient or permanent ROS production. Well-founded conclusions on health implications are only achievable if additional functional investigations, such as learning behaviour or the occurrence of DNA damage, are included in such studies. Group sizes of 5 animals or more are quite meaningful in such investigations, in contrary to the so-called "cancer bioassays" (studies in animal models on the induction and/or promotion of carcinogenesis by environmental influences or chemicals).

Associated with longer exposure periods over weeks or months, even for just a few hours per day, an increased occurrence of ROS, an overload and exhaustion of antioxidative protective mechanisms and damage to the DNA were reported for RF-EMF at various frequencies and doses (specific absorption rate; SAR), even at SAR values below the regulatory limits. However, one study also found that a recovery and return to the baseline after the end of exposure occurred. In this context, research on

underlying mechanisms, such as those relating to voltage-dependent calcium channels, is particularly informative. Such ion channels, for instance the TRPV1 ("transient receptor potential") channel are involved in pain transmission and can be activated not only by stimuli such as heat, capsaicin (active ingredient in chilli peppers), but also by oxidative stress. RF-EMF induced oxidative stress could therefore lead to increased calcium influx and trigger physiological as well as pathological processes.

Environmental cofactors may influence the occurrence of, and response to, oxidative stress. Altogether, these studies suggest that various parameters are important for the outcome of EMF exposure. It is conceivable that a constant or reoccurring stimulation of ROS formation elevates incrementally the antioxidative defence system. In addition to the duration and dose of exposure, adaptive processes and age-related capacities to respond to oxidative stress are of great significance.

In some instances, the changes in redox balance have been accompanied by morphological changes that are implicated in neurodegenerative diseases. The majority of animal studies that investigated, in addition to ROS and anti-oxidative markers, effects of EMF on behaviour and cognitive functions showed a reduction in memory performance. There is thus evidence that, at least in animal models, increased ROS production by EMF is associated with an impairment of cognitive abilities. Pre-damage of the brain by neurodegeneration (i.e. Alzheimer's disease) affected the memory performance of RF-EMF-exposed animals more significantly than controls, suggesting that learning performance impaired by RF-EMF is enhanced by predispositions. In addition to such pre-existing conditions, other environmental or risk factors may also contribute to whether and to what extent oxidative stress by EMF exposure occurs. There is evidence that age is such a risk factor, as elderly individuals are less able to compensate for increased ROS formation due to their reduced antioxidative capacity in the brain and adaptive processes are exhausted more quickly than in young individuals. On the other hand, animals and individuals immediately after birth, are also unable able to fully compensate for oxidative stress, since their antioxidative protection mechanisms are not yet fully developed in the first days or weeks of life, depending on the species.

Methodological issues must also be taken into account when assessing the studies. Often RF-EMF exposure was carried out in so-called carousel exposure systems, in which the animals are placed in narrow tubes, allowing for a homogeneous and defined exposure. This approach provides a source of error if the animals are not trained in advance, as constriction stress can also lead to oxidative stress. Therefore, it is important to include sham controls and to prepare the animals for the conditions during the exposure. In addition to increased ROS production, altered anxiety behaviour, but not memory performance, was seen in rats kept in such tubes, which was enhanced by WiFi-like exposure.

Also in the cell culture studies of the last 10 years, EMF-induced oxidative stress was most frequently investigated in neural cells. Cell models applied were mainly tumour cells of neuronal origin, but also cell lines and freshly isolated (primary) neurons of the brain as well as astrocytes from humans and rodents. When investigating the influence of EMF in tumour cell lines, it is important to note that tumour cells often have a disturbed oxidative balance and could therefore react differently to EMF or other treatments than a normal and healthy cell.

Nevertheless, there is relatively consistent evidence in cultured cells of neuronal origin that exposure to a 50 Hz ELF-MF leads to increased formation of ROS, usually in a transient manner. This can activate a variety of regulatory mechanisms and trigger respective cell responses, but also lead to persistent oxidative stress situations. After short-term ELF-MF exposure, little or no signs of changed markers for oxidative stress were found, while longer exposures led to later increases of these markers, as well as changes in cell response to additional stress. It is also worth mentioning that such observations were also made for weak magnetic fields below the threshold values ($\leq 100 \mu\text{T}$) in combination with other triggers of oxidative stress, whereby the cellular adaptations and consequences were still detectable later on. Comparable observations were also made in RF-EMF-exposed neuronal cells, although here

the findings are less conclusive and partly contradictory. However, this could also be due to the technically and dosimetrically more demanding application in this frequency range and the diversity and variability of the EMFs investigated (different carrier frequencies, with or without modulation etc.).

In the long run, the understanding of the mechanisms leading to the observations in animal models can be developed in cell studies. There is good evidence for an influence on cellular signalling pathways regulated by ROS. Here, the magnitude of activation as well as the possibility of compensation must be taken into account, when evaluating health impact in case of exhaustion of counter-regulation. Again, the state of differentiation was critical; differentiated cells reacted less sensitively than undifferentiated or early stage differentiated cells. Higher dose exposures displayed more pronounced effects, however, an effect caused by increase in temperature cannot always be ruled out. Nevertheless, there were also observations of increased oxidative stress in exposures conditions with field strengths/SAR values below the regulatory limits. Other methodological factors, such as keeping sham controls in another incubator, also pose a risk of false-positive results. Here, for example, vibrations, EMF of the incubator or their inadequate shielding come into play and it cannot be excluded that these factors have influenced the outcome and conclusion of some studies. The duration of exposure also seems to be relevant; shorter exposure durations of a few hours tended to increase ROS production and reduce antioxidative processes more often than when long exposures were applied.

ROS and oxidative stress in the blood and immune system

The influence of manmade EMF on cells of the immune system has also been a frequent subject of studies in recent years [2, 5, 6]. The functioning of the immune system is intrinsically linked to the formation of ROS and NO. On one hand, these play an important role in the elimination of foreign or damaged cells (feeding activity, phagocytosis). On the other hand, they are involved in the inflammatory reaction and the activation of the immune response [13, 17]. In this sense, it is conceivable that a suppression, as well as a constant activation, of these processes by EMF could lead to negative health implications in the long run. For this reason, the influence of EMF on various aspects of the immune response and developmental stages of haematopoietic cells and brain immune cells (microglia) has been studied.

While there have been some publications on oxidative stress upon EMF exposure in isolated and cultured blood and immune cells, the number of animal studies is limited, and some of them have only provided information on ROS markers in blood. The available data do not currently allow a conclusive assessment of possible health effects. However, there are indications in immune cells that RF-EMF influences the response to other stress factors. This adaptive responses play an important role in real life, since, in contrast to experimental studies, humans and animals are exposed concomitantly to different as well as changing stress factors. In animals and cells, oxidative stress caused by chemical substances were modulated by exposure to RF-EMF. So, it was shown that immune responses and phagocytosis were altered by exposure.

Similar to findings for the central nervous system, there is evidence in the lymphoid system that the effects of EMF (RF-EMF including WiFi) are age-dependent. Very young animals could not compensate for oxidative stress, even after a recovery phase, whereas this was possible in older animals after complete development of the anti-oxidative protection system. The time of analysis of oxidative stress seems to play also a role in cell systems and short-term exposure was more likely to lead to an increase in oxidative stress in immune as well as in leukaemia cells. This increase was mostly temporary and the processes triggered by EMF were in part similar to a normal immune response.

Consequences of EMF on reproduction

The influence of EMF on fertility and on the development of fetuses is also an important issue, as developing organisms and cells are particularly sensitive to external stress factors. Effects of EMF on reproduction have been studied in male reproductive organs and in sperm and their precursors. In addition, dams were exposed to EMF to investigate possible impairment in the early and late stages of pregnancy and in the offspring.

The majority of the findings from the animal studies indicate a functional and morphological impairment of spermatozoa by RF-EMF exposure, which is associated with an increase of ROS, reduction of antioxidant capacity and lipid peroxidation. Here as well, a preceding insult or pre-existing condition (i.e. diabetes) was a risk factor that led to increased oxidative stress by exposure that could not be compensated. After exposure of the dams, an age-dependent effect on oxidative stress markers was seen in the offspring, but this was different depending on the organ system and in some cases did not show any evidence for induced oxidative stress. A study on impairments in early stages of pregnancy provided indications for reduced blastocyst implantation.

With regard to their role in fertility, cells of the reproductive system were also examined for effects of EMF. The majority of cell studies published in the last 10 years focused on investigations of RF-EMF effects, so that hardly any data are available on the influence of ELF-MF on oxidative balance. Due to their temperature sensitivity, developmental characteristics and accessibility, mainly male germ cells and cells from the reproductive organ were used in this context. As they are very temperature sensitive, temperature fluctuations must be excluded during exposure, otherwise false-positive findings will influence the evaluation. This was not the case in many cell studies, meaning that such false-positive findings cannot be excluded. All in all, the few cell studies do not provide any reliable evidence for an impairment of sperm cells and their precursors by EMF-induced oxidative stress.

Further observations on oxidative stress by EMF in other cell types and organs

In addition to the considerable literature on the influence of EMF on the nervous, immune and reproductive systems, there are also a number of studies on oxidative stress in other organs and cell types. Adaptive processes with recovery after a period of time were found to act, when there was increased ROS production and anti-oxidative counter-regulation by EMF exposure. Again, a pre-existing disease led to more pronounced effects in various organs, while in exposed cell cultures or primary cells, induction of oxidative stress by EMF were dependent on the cell type. Cancer cells, representing the majority of the cell lines studied, were more variable in their response, which might be due to their aberrant metabolism and identity.

Because of their function as a barrier and first line of defence against the environment, skin and epithelial cells were also examined for the response to EMF. In the last 10 years, there have only been experimental studies with cultured cells, mainly in the ELF-MF range, and none with animals. Due to the use of a wide range of cell types and the limited number of directly comparable studies, the current picture regarding the effects of EMF exposure on skin and epithelial cells is rather patchy. However, there is evidence that EMF can lead, at least temporarily, to an increase in ROS production and oxidative cell stress.

Conclusions

In summary, the majority of the animal and more than half of the cell studies provided evidence of increased oxidative stress caused by RF-EMF or ELF-MF. This notion is based on observations in a large number of cell types, applying different exposure times and dosages (SAR or field strengths), also in

the range of the regulatory limits. Certainly, some studies are burdened with methodological uncertainties and weaknesses or are not very comprehensive in terms of exposure time, dose, number and quantitative analysis of the biomarkers used, to name a few. Taking these methodological weaknesses into account, nonetheless, a tendency becomes apparent, namely that EMF exposure, even in the low dose range, can lead to changes in oxidative balance. Organisms and cells are generally able to react to oxidative stress, and many studies showed adaptation to EMF exposure after a recovery phase. Pre-existing conditions, such as immune deficiencies or diseases (diabetes, neurodegenerative diseases), compromise the body's defence mechanisms, including antioxidative protection, and it is therefore possible that individuals with these conditions experience more severe health effects. In addition, the studies show that very young and elderly individuals can react less efficiently to oxidative stress induced by EMF, which of course also applies to other stressors that cause oxidative stress. More extensive studies under standardised conditions are necessary, to better understand and confirm these phenomena and observations.

References

1. Yakymenko, I., et al., *Oxidative mechanisms of biological activity of low-intensity radiofrequency radiation*. *Electromagnetic Biology and Medicine*, 2016. **35**(2): p. 186-202.
2. Wang, H. and X. Zhang, *Magnetic Fields and Reactive Oxygen Species*. *International Journal of Molecular Sciences*, 2017. **18**(10).
3. Tamrin, S.H., et al., *Electromagnetic Fields and Stem Cell Fate: When Physics Meets Biology*. *Reviews of Physiology, Biochemistry and Pharmacology*, 2016. **171**: p. 63-97.
4. Santini, S.J., et al., *Role of Mitochondria in the Oxidative Stress Induced by Electromagnetic Fields: Focus on Reproductive Systems*. *Oxidative Medicine and Cellular Longevity*, 2018. **2018**: p. 5076271.
5. Rosado, M.M., et al., *Immune-Modulating Perspectives for Low Frequency Electromagnetic Fields in Innate Immunity*. *Frontiers in Public Health*, 2018. **6**: p. 85.
6. Manna, D. and R. Ghosh, *Effect of radiofrequency radiation in cultured mammalian cells: A review*. *Electromagnetic Biology and Medicine*, 2016. **35**(3): p. 265-301.
7. Lai, H., *Exposure to Static and Extremely-Low Frequency Electromagnetic Fields and Cellular Free Radicals*. *Electromagnetic Biology and Medicine*, 2019. **38**(4): p. 231-248.
8. Falone, S., et al., *Extremely Low-Frequency Magnetic Fields and Redox-Responsive Pathways Linked to Cancer Drug Resistance: Insights from Co-Exposure-Based In Vitro Studies*. *Frontiers in Public Health*, 2018. **6**: p. 33.
9. Dasdag, S. and M.Z. Akdag, *The link between radiofrequencies emitted from wireless technologies and oxidative stress*. *Journal of Chemical Neuroanatomy*, 2016. **75**(Pt B): p. 85-93.
10. Health Council of the Netherlands, *Background document to the advisory report 5G and health. Background document to 5G and health*. The Hague: Health Council of the Netherlands, 2020; publication no. 2020/16Ae.
11. IARC monographs on the evaluation of carcinogenic risks to humans: *Non-ionizing radiation, Part 1: static and extremely low-frequency (ELF) electric and magnetic fields*. *IARC Monogr Eval Carcinog Risks Hum* 2002;80:1-395.
12. IARC monographs on the evaluation of carcinogenic risks to humans: *Non-ionizing radiation, Part 2: Radiofrequency electromagnetic fields*. *IARC Monogr Eval Carcinog Risks Hum* 2013;102:1-460.
13. Droge, W., *Free radicals in the physiological control of cell function*. *Physiological Reviews*, 2002. **82**(1): p. 47-95.

14. Sies, H., C. Berndt, and D.P. Jones, *Oxidative Stress*. Annual Review of Biochemistry, 2017. **86**: p. 715-748.
15. Brieger, K., et al., *Reactive oxygen species: from health to disease*. Swiss Medical Weekly, 2012. **142**: p. w13659.
16. Bedard, K. and K.H. Krause, *The NOX family of ROS-generating NADPH oxidases: physiology and pathophysiology*. Physiological Reviews, 2007. **87**(1): p. 245-313.
17. Yang, Y., et al., *Reactive oxygen species in the immune system*. International Reviews of Immunology, 2013. **32**(3): p. 249-70.
18. Oswald, M.C.W., et al., *Regulation of neuronal development and function by ROS*. FEBS Letters, 2018. **592**(5): p. 679-691.
19. Wang, Y., et al., *Superoxide dismutases: Dual roles in controlling ROS damage and regulating ROS signaling*. The Journal of Cell Biology, 2018. **217**(6): p. 1915-1928.
20. Brigelius-Flohe, R. and M. Maiorino, *Glutathione peroxidases*. Biochimica et Biophysica Acta, 2013. **1830**(5): p. 3289-303.

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Additional information:

[BERENIS - Swiss expert group on electromagnetic fields and non-ionising radiation](#)

[List of abbreviations \(pdf\)](#)