Original Research Article
Cellular Effects Following Exposure to Wireless DECT Base Radiation and Presentation of a Device for Their Compensation

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ABSTRACT

Background: Wireless telecommunication sources working with frequencies ranging from 0.9 to 2.5 GHz are still increasing rapidly. Among these are digitally enhanced cordless telecommunication (DECT) phones which have been considered to emit only a weak radiation when an active DECT base and handset are separated from each other.
Aim of the study: Prompted by this background this study investigated the cellular effects of DECT base radiation and its possible compensation by a specially designed device, named memonizerCOMBI Standard A.
Materials and methods: Connective tissue fibroblasts (L-929) were exposed to the radiation of an active commercially available DECT base with a frequency of 1.885 GHz for 24 hours ± memonizerCOMBI beneath the incubator. Unexposed cells in another incubator placed with a distance of about 10 m in the same laboratory rooms served as corresponding controls. Cell vitality was checked by enzymatic measurement of the activity of mitochondrial dehydrogenases by XTT.
Results: The results clearly demonstrate that exposure to DECT base radiation caused a significantly reduced cell vitality by 47.6 ± 7.4 % (mean value ± standard deviation; P = .01; Wilcoxon-Mann-Whitney test). Reduction in cell vitality was accompanied by marked morphological changes in the cells such as intracellular vacuolization, rounding and detachment which are similar to alterations observed during oxidative stress by the presence of reactive oxygen species. Reduction in cell vitality after DECT base radiation exposure was compensated by use of memonizerCOMBI by two-thirds yielding a reduction in cell vitality by only 17.5 ± 8.1 % (mean value ± standard deviation; P = .01 vs. exposed cells without memonizer; Wilcoxon-Mann-Whitney test).
Conclusions: The results indicate that exposure of cultured connective tissue cells to DECT base radiation at a frequency of 1.885 GHz causes a significantly reduced cell vitality which can be extensively compensated by using a memonizerCOMBI device.

Keywords: Electromagnetic radiation; health effects; DECT base; memonizerCombi; cell death; cell culture.

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1. INTRODUCTION

The continuous increase of wireless telecommunication sources, such as mobile phones, digitally enhanced cordless telecommunication (DECT) phones, routers and many others have caused a dramatic increase in environmental levels of electromagnetic radiation [1-4]. All these sources emit radiation in a wide spectrum of frequencies with different characteristics ranging from 0.9 to 2.5 GHz. Although the energy of this type of radiation is weak compared to ionizing radiation, recent research provides strong evidence that electromagnetic radiation is able to affect biological and biochemical processes and might lead to oxidative stress, cell death, cellular dysfunction and even carcinogenesis [see, for instance refs. 5-11].

Due to its world-wide importance with more than 5 billion users [12], mobile phone technology has been extensively investigated for its health effects at the cellular, experimental animal, and epidemiological level [see, for instance refs. 6, 7, 13-16]. Epidemiological and experimental research on DECT base and handset radiation exposure which might be also potentially harmful to millions of people is very limited [17].

Given the limited available data, the objective of the present study was to investigate the effects of radiation emitted from an active DECT base on cultured connective tissue cells with their wide-spread distribution within the body and the use of a newly created device for the compensation of DECT base radiation.

2. MATERIAL AND METHODS

2.1 DECT phone

The active base of a commercially available DECT phone (Gigaset 4010 Classic; Siemens, Germany) was used for the experiments described here. Analysis of the frequency characteristics gave a sharp peak at 1.885 GHz with - 46.47 dBm (Fig. 1).

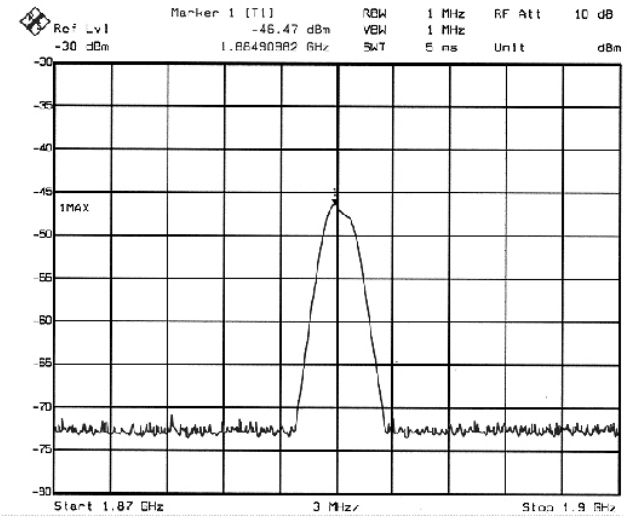

Fig. 1: Analysis of frequency for the active DECT base (Siemens Gigaset 4010 Classic) ranging from 1.87 GHz to 1.90 GHz.

The main peak is found at 1.885 GHz with -46.47 dBm.

2 Device for compensation of DECT base radiation

The device which was tested for its potential to compensate DECT base radiation was a memonizerCOMBI Standard A (COT-STD.A). This device is commercially available from memon® bionic instruments GmbH, D-83026 Rosenheim, Germany.

2.3 Operation principle of the device

Certain mineral groups are able to compensate negative health effects caused by non-ionizing radiation. These are used as so-called metamaterials in the technological application. It is known that such phyllosilicates have ionic clathrate hydrates which can interact with natural and artificial electrons and have properties of typical monochromatic infrared and terahertz frequencies as well as cyclotron resonances [18]. They show octahedral quantum resonances [19] at terahertz

frequencies, UV, IR and other lower and higher frequencies. Phyllosilicates are able to protect DNA molecules from ionizing radiation and to act as catalysts in RNA synthesis [20]. Protective mechanisms against biotic and abiotic influences have also been discovered [21].
Typical resonances for living organisms are within certain FIR frequencies, supported by octave resonances of the phyllosilicate metamaterial in the IR, NIR and UV spectra. Artificially generated electromagnetic waves block access to natural octave resonances for living organisms and water clathrate systems. Restoration of this natural field is achieved by using the phyllosilicate metamaterial [22]. These are, together with water molecules, able to transfer their own or induced terahertz resonances [23] and thus to compensate biological effects generated by artificial, non-thermal, non-ionizing radiation.
Selected electromagnetic resonance spectra have led to a marked increase in the activity of ornithine decarboxylase (ODC) in L-929 cells in similar experiments [24]. The technology used in this experiment utilizes specially induced natural resonance spectra in phyllosilicate metamaterials, which are believed to lie within the terahertz gap and can be transferred to living systems. The hypothesis of the EM wave transmutation [22] also stated that the quantum states of such phyllosilicates and their particular resonances in biological evolution could have been decisive in the creation of the first living cells [25].

2.4 Cell culture and test procedure

The main problem of studies of whole multi-cellular organisms such as rats, mice, drosophila and others is the complexity of the test systems. There are numerous unknown variables which are difficult to be established. In contrast to these models, cultivation of eukaryotic cells can be standardized and provides the opportunity to vary different factors depending on the experimental needs.
In this present study, cultured connective tissue fibroblasts (cell line L-929; Leibniz-Institut, Deutsche Sammlung für Mikroorganismen und Zellkulturen, Braunschweig, Germany) as a standard cell line for toxicological studies were taken at passages 22 to 50 over a total experimental period of 4 months. Cells were routinely cultivated in the moist atmosphere of an incubator at 37 °C and gassed with 5 % CO_2 and 95 % air to yield a constant pH value. Culture medium was RPMI 1640 with 10 % fetal calf serum and standard amounts of penicillin and streptomycin. All cell culture reagents were from GE Healthcare Life Sciences; Freiburg, Germany.
For the tests, cells were seeded from 80 to 90 % confluent mass cultures at a density of 20,000 cells/well into 16 wells in the middle part of a 96 well-plate (200 µL culture medium/well). After 24 hours to ensure cell attachment and metabolization, culture medium was exchanged to 250 µL/well of Leibowitz L-15 medium (Biochrom; Berlin, Germany) containing 10 % fetal calf serum and standard amounts of penicillin and streptomycin. This culture medium guarantees a pH value at 7.4 even at normal atmospheric conditions. Plates were transferred to a Cultura M mini incubator and cultivated at 37 ± 1 °C without CO_2-gassing.
The active DECT base was directly placed on the lid of the culture plate and cells were exposed to the DECT base radiation at continuous operation for the next 24 hours. Approximately 10 meters distance in the same laboratory rooms, a second Cultura M mini incubator was taken for the untreated control cells in a 96-well plate at the same cultivation conditions.
After 24 hours of exposure, cell vitality was checked by morphological observation of the cell cultures and by enzymatic activity. For this purpose, cell culture medium was removed and replaced by 120 µL fresh culture medium and 12 µL XTT (Xenometrix AG, Allschwil, Switzerland) and incubated for 120 minutes in the incubator at 37 °C.
XTT is the sodium salt of 2,3-bis[2-methoxy-4-nitro-5-sulfo-pheny]-2H-tetrazolium-5-carboxyanilide and has a yellowish color. Mitochondrial dehydrogenases of viable cells cleave the tetrazolium ring of XTT to yield orange formazan crystals which are soluble in aqueous solutions. The intensity of the resulting orange solution correlates directly with cell vitality and metabolic activity [26, 27].
After 120 minutes, optical density was measured as a differential measurement ΔOD = 450 minus 690 nm after a 4 second shaking interval using an ELISA reader (BioTek Slx808). The experiments were performed in 4 independent test series on different days with 14 wells per test. Statistical analysis was done using Wilcoxon-Mann-Whitney test.
In a second series of experiments, the same experimental design as described above was used with the modification that the device for compensation was placed directly beneath the Cultura M mini incubator containing the 96-well plate and the active DECT base. The experiments were performed in 6 independent test series on different days with 14 wells per test. Statistical analysis was done using Wilcoxon-Mann-Whitney test.

3. RESULTS

The results of all experiments presented here were consistent and reproducible, considering they were conducted within a time period of several months with breaks between the investigation intervals. This demonstrates that this experimental setup has been established successfully and provides data which can be directly compared with each other.
As depicted in Fig. 2, the exposure of connective tissue fibroblasts to the active DECT base for 24 hours caused a reduced cell vitality in all experiments when compared to untreated control cells. When the results of the single experiments are taken together, this reduction in cell vitality was 47.6 ± 7.4 % (mean value ± standard deviation) and was

statistically different from untreated control cells as checked by Wilcoxon-Mann-Whitney test (*P* = .01; Fig. 5). The reduced cell vitality after DECT base radiation exposure also resulted in a largely altered morphology of connective tissue fibroblasts (Fig. 3) with intracellular vacuolization and rounding of cells with long cytoplasmic protrusions or even detachment. These changes were irreversible, because cells did not achieve normal cell morphology after another 24 hours of incubation in fresh culture medium and without any further DECT base radiation exposure (not depicted).

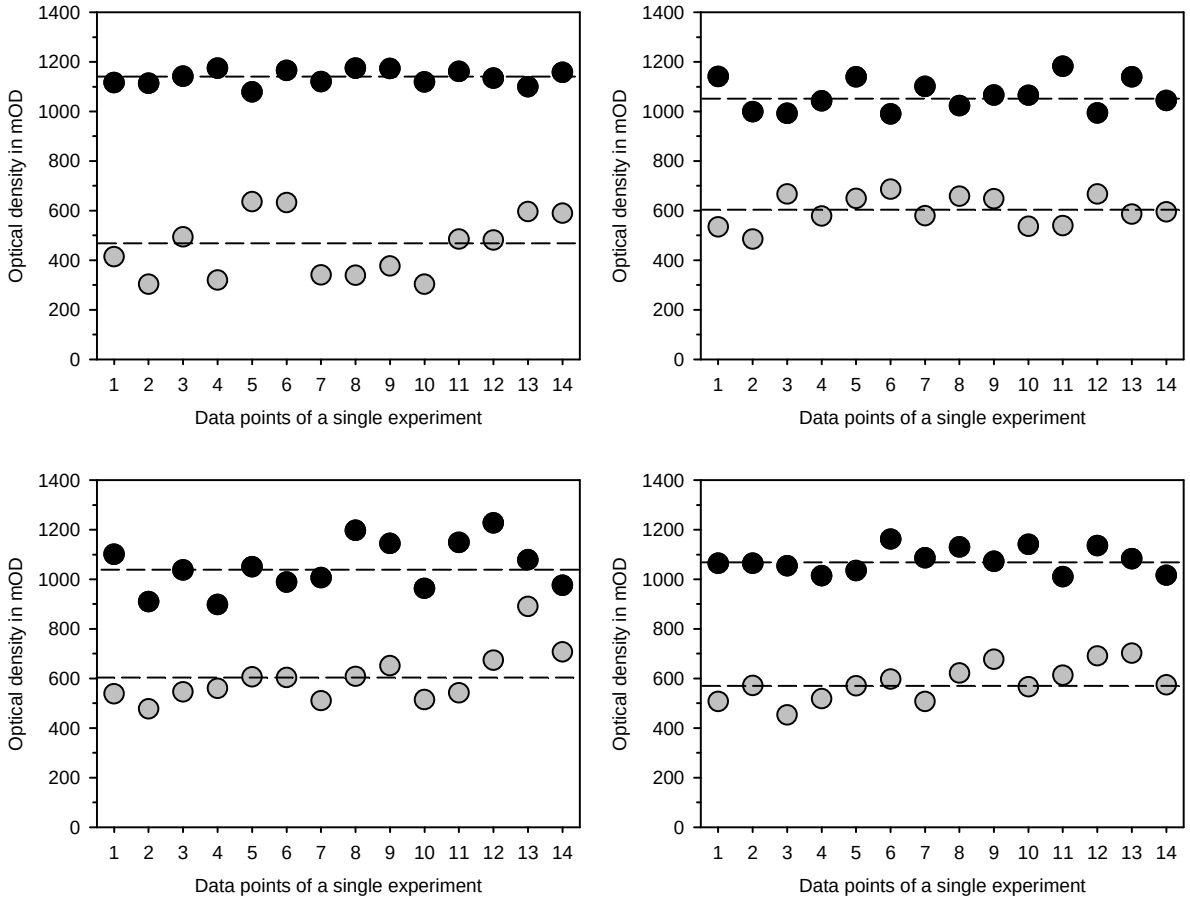

Fig. 2: Original measurements of 4 independent experiments with exposure of connective tissue fibroblasts to the active DECT base for 24 hours (gray circles) in comparison to untreated control cells (black circles).
Each data point given in the diagrams represents the cell vitality in one single well of the appropriate 96-well plate.

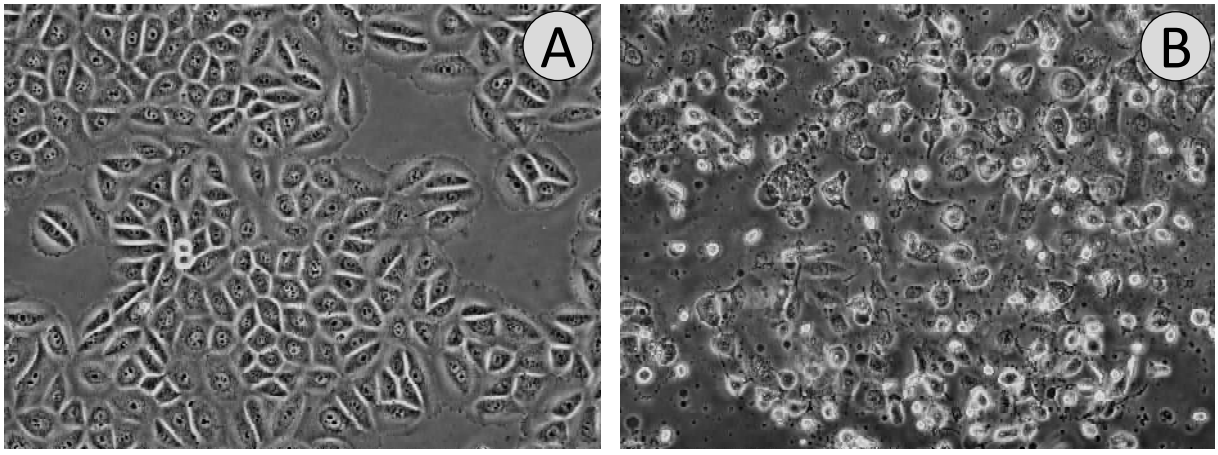

Fig. 3: Micrographs illustrating the alterations in cell morphology of connective tissue fibroblasts which were exposed to DECT base radiation for 24 hours (B) in comparison to untreated control cells (A).
Note the marked cell rounding, detachment and intracellular vacuolization in (B) which might be related to oxidative stress. Phase contrast microscopy at an Olympus IX50 inverted microscope equipped with an Olympus 20x Planachromate and an Olympus E-10 digital camera at 4 megapixels.

When a memonizerCOMBI Standard A was placed beneath the incubator in which cells were exposed to DECT base radiation for 24 hours, the reduction in cell vitality was markedly different from previous experiments without the device. Vitality of connective tissue fibroblasts was reduced now only by 17.5 ± 8.1 % (mean value ± standard deviation) which is equivalent to a compensation of unwanted cellular effects of approximately two-thirds (Fig. 4 and Fig. 5). The statistical significance of the summarized values between exposed cells ± memonizerCOMBI Standard A was $P = .01$ confirming that the device was able to reduce cellular effects in a quite effective way. However, there was still a statistical difference of $P = .05$ between untreated control cells and exposed cells with memonizerCOMBI Standard A (Fig. 5).
There is another point which should be mentioned here. It took at least 7 days after installation of the device below the Cultura M mini incubator until its information transfer field had developed and was efficient enough to compensate the unwanted cellular effects of DECT base radiation as presented above.

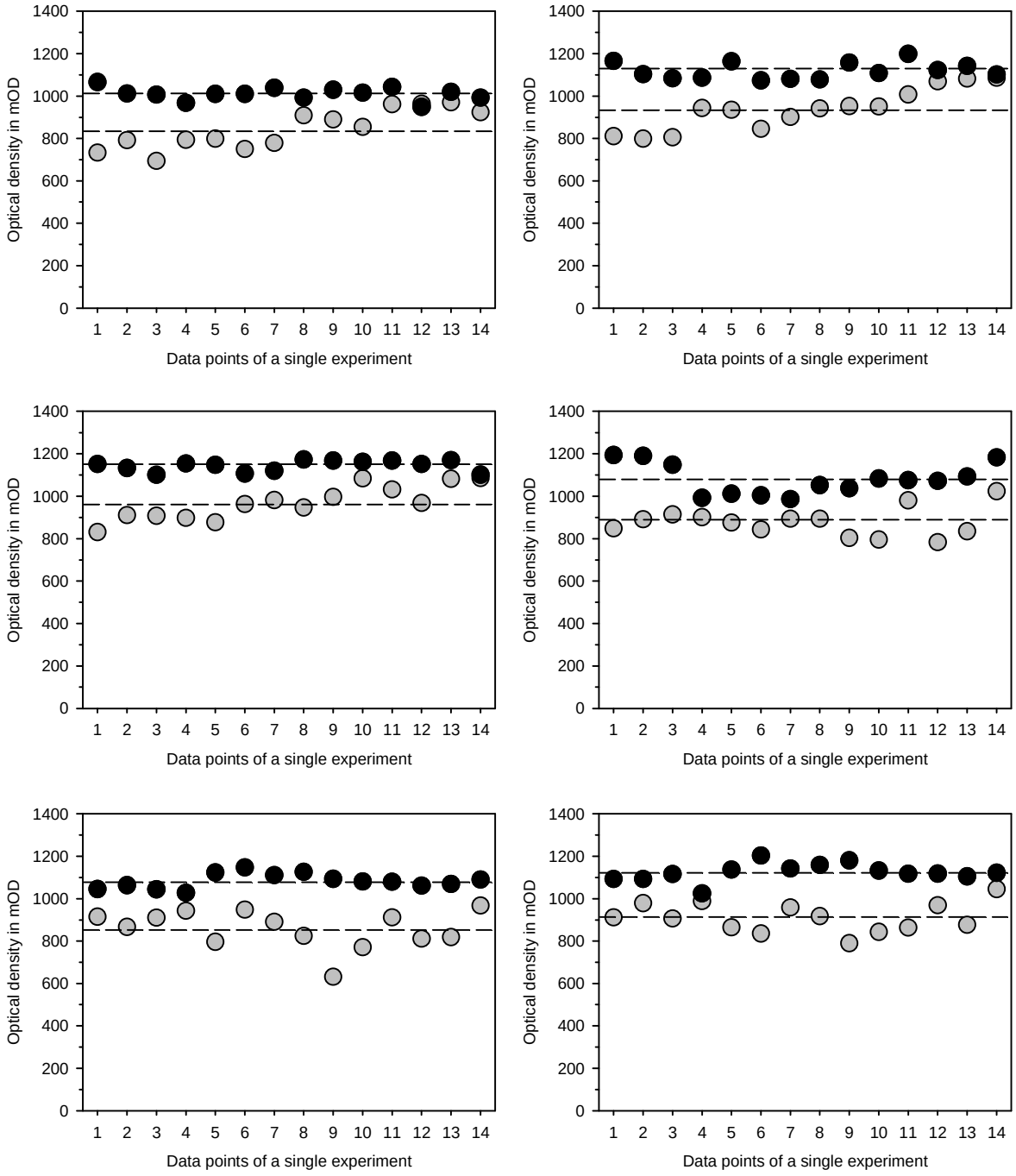

Fig. 4: Original measurements of 6 independent experiments with exposure of connective tissue fibroblasts to the active DECT base with memonizerCOMBI Standard A for 24 hours (gray circles) in comparison to untreated control cells (black circles).

Each data point given in the diagrams represents the cell vitality in one single well of the appropriate 96-well plate.

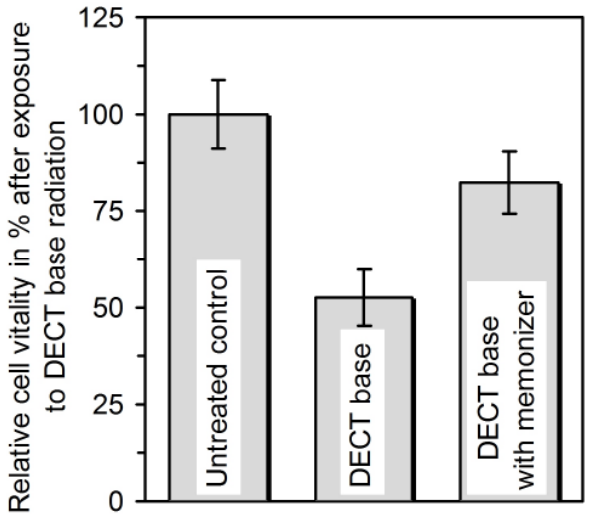

Fig. 5: Summarized presentation of the reduced cell vitality of connective tissue fibroblasts after exposure to DECT base radiation for 24 hours and its compensation by use of a memonizerCombi Standard A device.
Data represent mean value ± standard deviation of 4 experiments (untreated control vs. DECT base radiation; P = .01; Wilcoxon-Mann-Whitney test) and 6 experiments (untreated control vs. DECT base radiation with memonizer compensation; P = .05; Wilcoxon-Mann-Whitney test).

4. DISCUSSION

The fact that wireless telecommunication sources might cause unwanted health effects is still under controversial discussion. However, one should also take the different relevant frequencies under consideration which are ranging from 0.9 GHz to 2.5 GHz and might vary from country to country. Although mobile phones have become the main wireless telecommunication source worldwide, wireless DECT phones are still in use in millions of domestic homes and at workplaces. It has been considered that DECT phones emit only a weak and uncritical radiation when an active DECT base and handset are separated from each other. Indeed, this seems to be not the case under the test conditions as presented here. In the tests, the radiation of an active DECT base reduced cell vitality by approximately 50 %.
One might argue that an active DECT base for a continuous period of 24 hours and a distance of only some centimeters between cells and DECT base might be not a realistic situation. This may be right, although there are numerous people who have a DECT base nearby and the handset placed on the table nearly every day. Under these circumstances, the cellular effects of an active DECT base become more prominent. However, the cellular effects in terms of morphology and vitality as observed here are in accordance with previous studies on other cell types [28, 29].
Oxidative stress is a biochemical condition, which is defined by an imbalance between reactive oxygen species and the body's antioxidant protection. The presented morphological results with an active DECT base at a frequency of 1.885 GHz point to previous findings which described that the generation of reactive oxygen species and the resulting oxidative stress seems to be one of the main mechanisms causing cell death by apoptosis (see, for instance [28-31]. Dasdag and Akdag [32] evaluated in their review available in vitro and in vivo studies carried out on the relation between radiofrequency radiation and oxidative stress. The results of their studies indicated that radiofrequency radiation might be a factor which causes oxidative stress.
Quite surprising and unexpected were the results when a memonizerCOMBI Standard A was placed beneath the incubator in which the cells were exposed to DECT base radiation. As shown here in a number of independent experiments, this device is able to compensate the unwanted cellular effects of the radiation by approximately two-thirds. How the memonizerCOMBI Standard A really acts on the cells and reduces the unwanted effects of DECT base radiation is currently unknown and only a subject of speculation.
Further studies are currently undertaken to come to a closer understanding of the mechanisms how this device compensates DECT base radiation. However, many electro sensitive persons report an improvement of their situation when a memonizerCOMBI Standard A is used over a longer period of time.

5. CONCLUSION

The results indicate that exposure of cultured connective tissue cells to DECT base radiation at a frequency of 1.885 GHz causes a significantly reduced cell vitality which can be greatly compensated by using a memonizerCOMBI device.

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CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

AUTHORS' CONTRIBUTIONS

Author PCD designed the study, performed the experiments and wrote the first draft of the manuscript. Author TD wrote the chapter describing the operation principle of the compensation device. All authors read and approved the final manuscript.

REFERENCES

1. Ahlbom A, Feychting M. Electromagnetic radiation: Environmental pollution and health. Br Med Bull. 2003; 68: 157-165.
2. Havas M. Electromagnetic hypersensitivity: biological effects of dirty electricity with emphasis on diabetes and multiple sclerosis. Electromagnetic Biol Med. 2006; 25:259-268.
3. Levitt BB, Lai H. Biological effects from exposure to electromagnetic radiation emitted by cell tower base stations and other antenna arrays. Environ Rev. 2013; 18:369-395.
4. Margaritis LH, Manta AK, Kokkaliaris KD, Schiza D, Alimisis K, Barkas G, Georgiou E, Giannakopoulou O, Kollia I, Kontogianni G, Kourouzidou A, Myari A, Roumelioti F, Skouroliakou A, Sykioti V, Varda G, Xenos K, Ziomas K. *Drosophila oogenesis* as a bio-marker responding to EMF sources. Electromagn Biol Med. 2014; 33:165-189.
5. Blank M and Goodman R. Electromagnetic fields stress living cells. Pathophysiology. 2009;16:71-78.
6. Fragopoulou AF, Miltiadous P, Stamatakis A, Stylianopoulou F, Koussoulakos SL, Margaritis LH. Whole body exposure with GSM 900 MHz affects spatial memory in mice. Pathophysiology. 2010; 17:179-187.
7. Fragopoulou AF, Samara A, Antonelou MH, Xanthopoulou A, Papadopoulou A, Vougas K, Koutsogiannopoulou E, Anastasiadou E, Stravopodis DJ, Tsangaris GT, Margaritis LH. Brain proteome response following whole body exposure of mice to mobile phone or wireless DECT base radiation. Electromagn Biol Med. 2012; 31:250-274.
8. Sonmez OF, Odaci E, Bas O, Kaplan S. Purkinje cell number decreases in the adult female rat cerebellum following exposure to 900 MHz electromagnetic field. Brain Res. 2010; 1356:95-101.
9. Kesari KK, Siddiqui MH, Meena R, Verma HN, Kumar S. Cell phone radiation exposure on brain and associated biological systems. Indian J Exp Biol. 2013; 51:187-200.
10. Manta AK, Stravopodis DJ, Papassideri IS, Margaritis LH. Reactive oxygen species elevation and recovery in Drosophila bodies and ovaries following short-term and long-term exposure to DECT base EMF. Electromagn Biol Med. 2014; 33:118-131.
11. Vijayalaxmi, Scarfi MR. Biological health effects of radiofrequency fields. Int J Environ Res Public Health. 2014; 11:9376-9408.
12. Davis DL, Kesari S, Soskolne CL, Miller AB, Stein Y. Swedish review strengthens grounds for concluding that radiation from cellular and cordless phones is a probable human carcinogen. Pathophysiology. 2013; 20:123-129.
13. Hardell L, Carlberg M. Mobile phones, cordless phones and the risk for brain tumours. Int J Oncol. 2009; 35:5-17.
14. Chavdoula ED, Panagopoulos DJ, Margaritis LH. Comparison of biological effects between continuous and intermittent exposure to GSM-900-MHz mobile phone radiation: Detection of apoptotic cell-death features. Mutat Res. 2010; 700:51-61.
15. Fragopoulou AF, Koussoulakos SL, Margaritis LH. Cranial and postcranial skeletal variations induced in mouse embryos by mobile phone radiation. Pathophysiology. 2010; 17:169-177

16. Panagopoulos DJ, Johansson O, Carlo GL. Real versus simulated mobile phone exposures in experimental studies. BioMed Res. 2016; Int Article ID 607053.
17. Redmayne M, Inyang I, Dimitriadis C, Benkeab G, Abramson MJ. Cordless telephone use: implications for mobile phone research. J Environ Monit. 2010; 12:809-812.
18. Liboff AR. Ion cyclotron resonance interactions in living systems. Società italiana biofisica elettrodinamica. Atti iv convegno nazionale pavia. October 19, 2013.
19. Pitkänen M. More precise TGD based view about quantum biology and prebiotic evolution. 2015. http://tgdtheory.com.
20. Ferris JP. Mineral catalysis and prebiotic synthesis: Montmorillonite-catalyzed formation of RNA. Elements. 2005; 1:145-149.
21. Sposito GS, Skippe NT, Sutton R, Park S, Soper AK, Greathouse JA. Surface geochemistry of the clay minerals. Proc Natl Acad Sci USA. 1999; 96:3358–3364.
22. Geesink JH, Meijer KF. Quantum wave information of life revealed: An algorithm for electromagnetic frequencies that create stability of biological order, with implications for brain function and consciousness. NeuroQuantology. 2016; 14:106-125.
23. Avakyan SV, Voronin NA, Serova AE. The role of Rydberg atoms and molecules in the upper atmosphere. Geomag Aéronom 1997: 37.
24. Litovitz TA, Farrell JM, Barber M and Krause D. The superposition of a temporally incoherent magnetic field inhibits 60 Hz-induced changes in the ODC activity of developing chick embryos. Bioelectromagnetics. 1998: 19:53-56.
25. Hashizume H. Role of Clay Minerals in Chemical Evolution and the Origins of Life. 2012; DOI: 10.5772/50172.
26. Roehm NW, Rodgers GH, Hatfield SM, and Glasebrook AL. An improved colorimetric assay for cell proliferation and viability utilizing the tetrazolium salt XTT. J lmmunol Meth. 1991; 142:257-265.
27. Brosin A, Wolf V, Mattheus A, Heise H. Use of XTT-assay to assess the cytotoxicity of different surfactants and metal salts in human keratinocytes (HaCaT). A feasible method for in vitro testing of skin irritants. Acta Dermato-venereologica. 1997; 77:26-28.
28. Lixia S, Yao K, Kaijun W, Deqiang L, Huajun H, Xiangwei G, Baohong W, Wei Z, Jianling L, Wei W. Effects of 1.8 GHz radiofrequency field on DNA damage and expression of heat shock protein 70 in human lens epithelial cells. Mutat Res. 2006; 602:135–142.
29. Hou Q, Wang M, Wu S, Ma X, An G, Liu H, Xie F. Oxidative changes and apoptosis induced by 1800-MHz electromagnetic radiation in NIH/3T3 cells. Electromagn Biol Med. 2015; 34:85-92.
30. Lantow M, Lupke M, Frahm J, Mattsson MO, Kuster N, Simko M. ROS release and Hsp70 expression after exposure to 1800 MHz radiofrequency electromagnetic fields in primary human monocytes and lymphocytes. Radiat Environ Biophys. 2006; 45:55–62.
31. Lantow M, Schuderer J, Hartwig C, Simko M. Free radical release and HSP70 expression in two human immune-relevant cell lines after exposure to 1800 MHz radiofrequency radiation. Radiat Res. 2006; 165:88–94.
32. Dasdag S, Akdag MZ. The link between radiofrequencies emitted from wireless technologies and oxidative stress. J Chem Neuroanat. 2016; 75:85-93.