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Single- and DNA breaks in human hair root cells exposed to mobile phone radiation

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Abstract

Purpose: To analyze the short-term effects of radiofrequency radiation (RFR) exposure on genomic deoxyribonucleic acid (DNA) of human hair root cells.

Subjects and methods: Hair samples were collected from eight healthy human subjects immediately before and after using a 900-MHz GSM (Global System for Mobile Communications) mobile phone for 15 and 30 min. Single-strand DNA breaks of hair root cells from the samples were determined using the 'comet assay'.

Results: The data showed that talking on a mobile phone for 15 or 30 min significantly increased ($p < 0.05$) single-strand DNA breaks in cells of hair roots close to the phone. Comparing the 15-min and 30-min data using the paired t -test also showed that significantly more damages resulted after 30 min than after 15 min of phone use.

Conclusions: A short-term exposure (15 and 30 min) to RFR (900-MHz) from a mobile phone caused a significant increase in DNA single-strand breaks in human hair root cells located around the ear which is used for the phone calls.

Keywords: Radiofrequency radiation, 900 MHz mobile phone, hair root cells, DNA single strand break, comet assay

Introduction

The widespread use of mobile phones has given rise to a growing concern on the possible adverse health effects of the radiation they emit, particularly genetic effects. Although the consensus is that radiofrequency radiation (RFR) does not contain sufficient energy to break chemical bonds directly, that is, cannot directly induce deoxyribonucleic acid (DNA) damage, a substantial number of published studies have demonstrated genotoxic effects of RFR in vivo and in vitro (Lai and Singh 1995, 1996, Diem et al. 2005, Ruediger 2009, Campisi et al. 2010). Thus, the effects have to be caused by indirect mechanisms. Consequently, if RFR is an agent which has genotoxic attributes, it could be carcinogenic. Therefore, the importance of its indirect effects on DNA damage has

been emphasized (Verschaeve et al. 1994, Lai et al. 1997a, Lai and Singh 1997, Phillips et al. 1998, Wu et al. 2008).

The 'comet assay', also known as the 'single-cell gel electrophoresis assay' is the most frequently used technique to study RFR-induced DNA strand breaks. The alkaline comet assay used measures single-strand breaks and alkali-labile sites in the DNA molecule. In the assay, DNA is first unwound under alkaline conditions and then subjected to electrophoresis. DNA fragments migrate towards the anode, thereby forming a comet-like appearance (Ostling and Johanson 1984, Singh et al. 1988). The amount of DNA damage is quantified by the length and density of the comet tail.

The hair root undergoes rapid cell proliferation with short turnover time (Potten et al. 1996). As a consequence, the matrix cells in an anagen hair root would be expected to be sensitive to genotoxic agents and the damage would be rapidly manifested.

Since hair roots are close to the irradiation antenna of a mobile phone, they provide a convenient situation for studying genetic damages in human cells exposed to mobile phone radiation. For the first time in the present investigation, the short-term effects of RFR exposure on genomic DNA of human hair root cells were analyzed. About 85–90% of the hairs on one's head are in the anagen phase at a given time. Thus, almost all of the hair pulled out would be in the anagen phase. Forcibly plucked hairs in normal individuals have a normal anagen appearance, with an inner and outer root sheath (Pride and Tunnessen 1996). DNA strand breaks can be measured in these cells using the comet assay.

Subjects and methods

Eight human subjects (six women, two men, aged 30–47 years) participated in the present study. This study was approved by the ethics committee of the Gazi University (Report no. 104). More detailed information of the subjects is given in Table I. Each subject participated two times in the experiment. In the first run, hair samples were taken immediately before and after 15 min of mobile phone (iphone, Apple Inc., Cupertino, CA, USA) use, whereas in the second run, at two weeks

Table I. General information and confounding factors among volunteers.

	Age	Gender	Occupation	Smoking	Alcohol	Chemical	Medication	Radiation
1	45	Male	Medical Doctor					
2	37	Female	Chemical Eng.					
3	37	Male	Veterinarian					
4	45	Female	Biologist					
5	30	Female	Physicist					
6	30	Female	Biologist					
7 [*]	30	Female	Cleaner					
8 ^{**}	47	Female	Biologist					

^{*} 6. Drugs for asthma by chronic allergy; ^{**} 7. Drug for cardiac problems; ^{***} 8. Drug for hypertension.

later, hairs were taken immediately before (control) and after 30 min of phone use.

Collection of human hair roots

About 6 or 7 hair roots each were obtained from each subject before and after exposure. Hairs were plucked from behind the right ear (on which the mobile phone was used) within a 60 × 60 mm area. Hairs were cut gently close to their roots and then placed into a 1.5-ml Eppendorf microfuge tube (Greiner bio-one, Monroe, NC, USA) with the roots towards the bottom of the container.

Exposure source

A scenario for the exposure was planned to imitate the daily life use of mobile phone. The subjects used a commercial GSM (Global System for Mobile Communication) mobile phone operating in the frequency band of 900-MHz at 2 W maximal power output. The peak specific absorption rate (SAR) was declared to be 0.974 W/kg in the human head by the manufacturer. It could not represent the actual exposure. The distance between mobile phone and base station determine the actual radiofrequency (RF) energy exposure to the volunteer. The distances were the same for all volunteers. The handset was fully charged before each exposure. Exposures of mobile phone were performed at the same location in the laboratory. The mobile phone was held over the right ear in the same manner as when calling in daily life, and the volunteers spoke on the mobile phone's microphone for 15 or 30 min. Although the subjects could not keep holding the handset exactly at the same position during exposures, we considered it optimal since we wanted to know how daily mobile phone use affected hair roots in a realistic way.

Comet assay

DNA strand break assay was carried out immediately after hair sample collection. In hair root cell preparation, 6 or 7 hair roots were immersed in a 1.5-ml Eppendorf microfuge tube containing 30 µl of a solution that contained 1 × Hanks' Balanced Salt Solution (HBSS Ca and Mg free, Life Technologies/GIBCO, Carlsbad, CA, USA), 20 mM Ethylenediaminetetraacetic acid (EDTA, Sigma Chemicals, St. Louis, MO, USA), 10% Dimethyl sulfoxide (DMSO, Merck&Co., Whitehouse Station, NJ, USA). A plastic stick was used to loosen the cells from the hair roots. This cell suspension consisted of different types of hair root cells. The comet assay was conducted under alkali conditions basically as described by Singh (1996) with some modifications. Fifteen microliters of the cell suspension were mixed in 80 µl low melting

agarose (0.5%, Sigma Chemicals) and pipetted onto a slide pre-coated (Sail Brand, Shanghai, China) with 2% normal melting agarose (Sigma Chemicals). Cells were spread with a cover glass, and maintained on ice for 15 min to solidify. After removing the cover glass a layer of 0.5% low melting agarose was added and solidified on ice as before. The slides were immersed for 1 h in a lysing solution (2.5 M NaCl, 100 mM EDTA, 10 mM Tris (Sigma Chemicals), 1% Triton X-100 (Merck&Co., Whitehouse Station, NJ, USA, 10% DMSO) at 4°C. The slides were then placed in an electrophoresis unit (MSCHOICE, MP-250N omniPAC, Cleaver Scientific Ltd, Warwickshire, UK) containing a buffer (300 mM NaOH, 1 mM EDTA, pH 13) for 40 min at room temperature to allow for DNA unwinding. Electrophoresis was then carried out for 30 min at 25 V (~ 300 mA). The slides were then neutralized with Tris-HCl buffer (0.4 M, pH 7.5) for 5 min and air-dried. This was repeated three times. A total of 7.5 µl of the SYBR Green (Lonza Group Ltd, Basel, Switzerland) solution was diluted with 600 µl of distilled water and 65 µl of this diluted solution was applied to each slide. Slides were coded before analysis and each slide was analyzed by using a fluorescence microscope (Olympus BX 51 fluorescence model with 4',6-diamidino-2-phenylindole (DAPI)/f t c/Texas Red filter under blue excitation: wavelengths of excitation with a beam with half-width (400/15 -495/15 -570/25 nm) emission with a beam with half-width (460/20 -530/30 -625/650 nm) and dichroic (410-510-590 nm) filters and dichroic attachments at a resolution of 600 × 800 by digital color video camera (Pixera Corp., Los Gatos, CA, USA), Olympus Corp., Tokyo, Japan). All experimental set up and chemicals were supplied from company's distributors in Ankara, Turkey. From each slide, 100 cells were analyzed by comet assay software project <http://www.casp.of.pl>

(Poland) (Konca et al. 2003). To quantify the DNA damage, tail length (TL) and tail moment (TM) were evaluated. Tail length is measured from right border of head area to end of tail in micrometer (µm). Tail moment was calculated as the product of the tail length and the percentage of DNA in the tail in arbitrary units (Tail moment = tail length × % of DNA in the tail).

Data analysis

The mean tail length and tail moment from the 100 cells measured for single-strand breaks in each volunteer's control and exposed samples were used in data analysis. Data were analyzed using a SPSS Software Package for Statistical Analysis (SPSS for Windows, Version 16.0.0, SPSS Inc., Chicago, IL, USA). Both comet parameters measured in the

Table II. Hair root cell tail lengths (in μm) of individual volunteers exposed to a 900-MHz mobile phone radiation for 15 and 30 min. Each value is the average of 100 cells.

Volunteers	Control mean	SE	15 minutes mean	SE	Control mean	SE	30 minutes mean	SE	
1	12.9	1.0	27.2	1.7	15.2	1.0	32.6	1.4	
2	22.0	1.1	24.8	1.4	23.6	1.6	26.2	1.2	
3	16.1	1.2	23.5	1.5	13.1	1.0	32.2	1.6	
4	12.7	0.8	32.4	1.7	11.9	0.9	39.3	1.8	
5	12.6	0.9	28.0	1.6	12.6	0.9	32.2	1.4	
6	25.4	2.2	39.8	1.9	24.7	1.7	45.1	1.7	
7	21.3	1.3	39.9	1.5	25.8	1.6	46.5	2.2	
8	24.9	1.1	40.2	1.8	26.4	1.2	43.3	1.6	
mean	SE	18.5	1.9	32.0	2.5	19.1	2.3	37.2	2.6

exposed and control groups were evaluated using the paired t -test. The level of difference at $p = 0.05$ was considered statistically significant.

Results

Tail length data from individual subjects are presented in Table II. Average tail length and tail moment data are plotted in Figures 1 and 2, respectively. The data showed that talking on a mobile phone for 15 or 30 min significantly increased ($p = 0.05$) single-strand DNA breaks in cells of hair roots close to the phone. Comparing the 15-min and 30-min data using the paired t -test also showed that significantly more damages resulted after 30 min than after 15 min of phone use.

Discussion

The results of the present study indicate that a short-term exposure (15 and 30 min) to RFR (900-MHz) from a mobile phone caused a significant increase in DNA single-strand breaks in human hair root cells located around the ear which is used for the phone calls. It was reported that the maximal peak SAR occurs in the skin behind the ear near the antenna where the RF intensity is maximal (915 MHz, antenna distance 1.5 cm from the head). In our experiment, the hair roots were plucked from the same area behind the ear. We observed significant increase in DNA single-strand breaks

in hair root cells from the region where maximum SAR was measured as indicated by the studies of Okoniewski and Stuchly (1996) and Christ et al. (2010). The exposure was carried out by mobile phone operating in the frequency band of 900-MHz at 2 W maximal power output. However, the actual power output of the mobile phone could not be measured throughout the experiment across all volunteers. In addition, we had analyzed hair samples from subjects without cell phone exposure (the same individuals but unexposed) taken 5 and 15 min apart and found no significant difference. Therefore, the changes after cell phone exposure were not caused by the process of hair removal, such as skin irritation.

The present study is in agreement with many recent studies showing a significant increase in DNA single-strand breaks in cells after exposure to RFR. Increases in DNA strand breaks and micronucleation in lymphocytes obtained from cell phone user and increase in DNA damage in human lens epithelial cells at 0 and 30 min after 2 h of exposure to an 1800-MHz field at 3 W/kg have been reported (Gandhi 2005, Lixia et al. 2006). An increase in single-strand DNA breaks as assayed by the alkaline microgel electrophoresis method in lymphocytes from human blood samples exposed for 1–2 h in vitro to a 954-MHz RFR with SAR of 1.5 W/kg was also shown (Verschaeve et al. 1994). DNA damage in Molt-4 human lymphoblastoid cells exposed to low intensities mobile phone RFR (2.4–26 $\mu\text{W/g}$) for 2–21 h was reported by Phillips et al. (1998). Reactive oxygen species (ROS) production

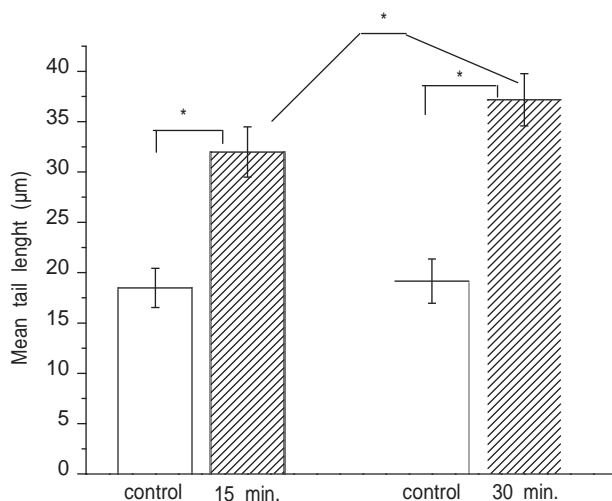


Figure 1. Influence of exposure time on the mean values of tail lengths in human hair root cells as determined by the alkaline comet assay (*statistically significant at $p = 0.05$). Error bars indicate the standard error of the mean (SEM) for $n = 8$ independent experiments.

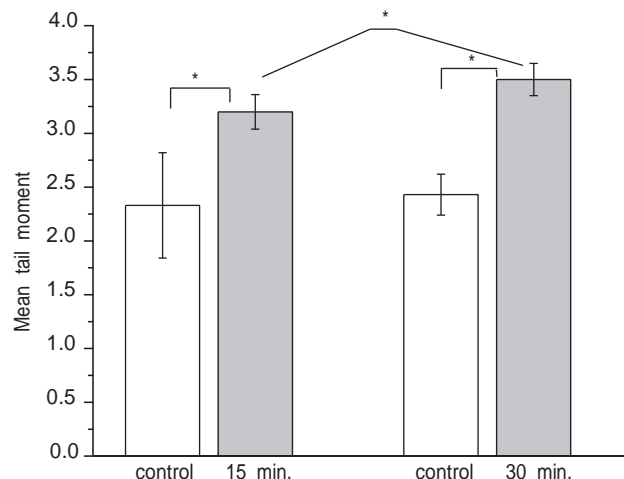


Figure 2. Influence of exposure time on the mean values of tail moments in human hair root cells as determined by the alkaline comet assay (*statistically significant at $p = 0.05$). Error bars indicate the standard error of the mean (SEM) for $n = 8$ independent experiments.

and DNA damage were observed in human spermatozoa exposed in vitro to RFR at 1.8 GHz and covering a range of SAR from 0.4 W/kg to 27.5 W/kg (De Iuliis et al. 2009). Increases in both single- and double-DNA strand breaks, as measured by the comet assay, in brain cells of rats exposed for 2 h to a 2450-MHz RFR at whole body SAR of 0.11, 0.6 and 1.2 W/kg were reported (Lai and Singh 1995, 1996, Kesari et al. 2010). A significant increase in ROS levels and DNA fragmentation was found after exposure of the astrocytes to modulated 900-MHz electromagnetic fields for 20 min (Campisi et al. 2010). It was reported also that under conditions of different mobile-phone modulations, RFR (1800 MHz) induces DNA single- and double-strand breaks in human diploid fibroblasts and in rat granulosa cells in culture (Diem et al. 2005). On the other hand, there are more studies with no significant cellular DNA damage from exposure to RFR. It was reported (Speit et al. 2007) that a genotoxic effect of RF electromagnetic field on human fibroblasts (ES1 cells) was not detected under the same experimental conditions as used by Diem et al. (2005). In a series of studies with cultured human lymphocytes, it was reported that chromosome aberrations, micronuclei or sister chromatid exchange were not measured after exposure to a 900 MHz GSM signal at 0.2–10 W/kg for various periods of time and also, in a study with human leukocytes, it was reported that a 2-h exposure to a 900 MHz GSM signal at 0.3 and 1 W/kg did not significantly affect levels of DNA strand breaks (Zeni et al. 2003, 2005, Scarf et al. 2006). It was also shown that 24 h of exposure to a 935-MHz GSM basic signal at 1 or 2 W/kg did not cause DNA strand breaks in human blood cells (Stronati et al. 2006). Several hypotheses have been proposed to explain these contradictory results from different laboratories including use of different versions of the comet assay, different detection sensitivities of laboratories, the experimenters' experiences and the exposure conditions (Hardell and Sage 2008, Phillips et al. 2009).

DNA damage could result in significant health issues. Over accumulation of genetic damage in cells in time can be the triggering factor of cancer (Ames 1989), aging (Targovnik et al. 1985, Mullaart et al. 1990, Ames et al. 1993), and cell death (Evan and Littlewood 1998). RFR-induced DNA damage in hair root cells may force the hair to transit from anagen into catagen phase prematurely like that of ultraviolet radiation exposure (Lu et al. 2009). Cumulative DNA damage and/or defect in DNA damage repair in hair roots also lead to graying of hair (Nishimura et al. 2005, Sharpless and DePinho 2007). The role of DNA damage in pathogenesis of androgenetic alopecia was shown in a study by El-Domyati et al. (2009).

The mechanism by which RFR produces DNA strand breaks is still being debated. Since the energy of RFR cannot directly break chemical bonds in DNA, indirect mechanisms probably lead to the genotoxic effects. One of the proposed mechanisms is that RFR enhances free radicals activity in cells, which in turn lead to DNA damage (Brocklehurst and McLauchlan 1996). It is also based on the research by Lai and Singh. They found that effects of RFR on DNA were blocked by antioxidants (Lai and Singh 1997). Another suggested mechanism is that RFR radiation may disturb DNA repair processes (Phillips et al. 1998, Sykes et al. 2001). The

thermal effects are also considered as a mechanism of RFR genotoxicity. However, the majority of genotoxic studies were performed at SAR too low to cause a significant increase in temperature to damage DNA. After 15 and 30 min of use of a 900-MHz mobile phone, the increases in temperature at the ear and surrounding area of the head were found to be about 2°C (Straume et al. 2005). It was reported that a temperature higher than 40°C is required to damage DNA (Takahashi et al. 2004, Purschke et al. 2010). Also, the work of Mashevich et al. (2003) showed that while an 830-MHz signal after 72 h of exposure at SAR ranged from 1.6–8.8 W/kg led to an increase in chromosome 17 aneuploidy in human peripheral blood lymphocytes, temperature elevation alone in the range of 34.5–38.5°C did not produce this genotoxic effect, although a significant effect was observed at higher temperatures of 40–41°C.

We presented here the effect of short-term exposure to mobile phone RFR on DNA of hair root cells. Continuous long-term exposure to RFR is rapidly increasing in the environment. The long-term effect of repeated exposure to RFR in hair root cells should also be studied by taking into consideration the DNA repair processes in order to identify the biological mechanisms involved. Further study would investigate double-strand DNA breaks in both short-term and long-term exposure conditions in the region of the head where maximum SAR occurs.

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Declaration of interest

The authors report no conflicts of interest.

References

- Ames BN. 1989. Endogenous oxidative DNA damage, aging, and cancer. *Free Radical Research Communications* 7:121–128.
- Ames BN, Shigenaga MK, Hagen TM. 1993. Oxidants, antioxidants, and the degenerative diseases of aging. *Proceedings of the National Academy of Sciences of the USA* 90:7915–7922.
- Brocklehurst R, McLauchlan KA. 1996. Free radical mechanism for the effects of environmental electromagnetic fields on biological systems. *International Journal of Radiation Biology* 69:3–34.
- Campisi A, Gulino M, Acquaviva R, Bellia P, Raciti G, Grasso R, Musumeci F, Vanella A, Triglia A. 2010. Reactive oxygen species levels and DNA fragmentation on astrocytes in primary culture after acute exposure to low intensity microwave electromagnetic field. *Neuroscience Letters* 473:52–55.
- Christ A, Gosselin MC, Christopoulou M, Kühn S, Kuster N. 2010. Age-dependent tissue-specific exposure of cell phone users. *Physics in Medicine and Biology* 55:1767–1783.
- De Iuliis GN, Newey RJ, King BV, Aitken RJ. 2009. Mobile phone radiation induces reactive oxygen species production and DNA damage in human spermatozoa in vitro. *Plos One* 4:e6446.
- Diem E, Schwarz C, Adlkofer F, Jahn O, Ruediger HW. 2005. Non-thermal DNA breakage by mobile-phone radiation (1800 MHz) in human fibroblasts and in transformed GFSH-R17 rat granulosa cells in vitro. *Mutation Research* 583:178–183.
- El-Domyati M, Attia S, Saleh F, Bassyouni MI, El-Fakahany H, Wahab HA. 2009. Proliferation, DNA repair and apoptosis in androgenetic alopecia. *Journal European Academy of Dermatology and Venereology* 23:7–12.

- Evan G, Littlewood T. 1998. A matter of life and cell death. *Science* 281(5381):1317–1322.
- Gandhi AG. 2005. Genetic damage in mobile phone users: Some preliminary findings. *Indian Journal of Human Genetics* 11:99–104.
- Hardell L, Sage C. 2008. Biological effects from electromagnetic field exposure and public exposure standards. *Biomedicine & Pharmacotherapy* 62:104–109.
- Kesari KK, Behari J, Kumar S. 2010. Mutagenic response of 2.45 GHz radiation exposure on rat brain. *International Journal of Radiation Biology* 86:334–343.
- Konca K, Lankof A, Banasik A, Lisowska H, Kuszewski T, Gozdz S, Koza Z, Wojcik A. 2003. A cross-platform public domain PC image-analysis program for the comet assay. *Mutation Research* 534:15–20.
- Lai H, Singh NP. 1995. Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells. *Bioelectromagnetics* 16:207–210.
- Lai H, Singh NP. 1996. Single- and double-strand DNA breaks in rat brain cells after acute exposure to radiofrequency electromagnetic radiation. *International Journal of Radiation Biology* 69:513–521.
- Lai H, Carino MA, Singh NP. 1997a. Naltrexone blocks RFR-induced DNA double strand breaks in rat brain cells. *Wireless Networks* 3:471–476.
- Lai H, Singh NP. 1997. Melatonin and a spin-trap compound block radiofrequency electromagnetic radiation-induced DNA strand breaks in rat brain cells. *Bioelectromagnetics* 18:446–454.
- Lixia S, Yao K, Kaijun W, Deqiang L, Huajun H, Xiangwei G, Baohong W, Wei Z, Jianling L, Wei W. 2006. Effects of 1.8-GHz radiofrequency field on DNA damage and expression of heat shock protein 70 in human lens epithelial cells. *Mutation Research* 602:135–142.
- Lu Z, Fischer TW, Hasse S, Sugawara K, Kamenisch Y, Kregel S, Funk W, Berneburg M, Paus R. 2009. Profiling the response of human hair follicles to ultraviolet radiation. *Journal of Investigative Dermatology* 129:1790–1804.
- Mashevich M, Folkman D, Kesar A, Barbul A, Korenstein R, Jerby E, Avivi L. 2003. Exposure of human peripheral blood lymphocytes to electromagnetic fields associated with cellular phones leads to chromosomal instability. *Bioelectromagnetics* 24:82–90.
- Mullaart E, Boerrigter ME, Boer GJ, Vijg J. 1990. Spontaneous DNA breaks in the rat brain during development and aging. *Mutation Research* 237:9–15.
- Nishimura EK, Granter SR, Fisher DE. 2005. Mechanisms of hair graying: Incomplete melanocyte stem cell maintenance in the niche. *Science* 307:720–724.
- Okoniewski M, Stuchly MA. 1996. A study of the handset antenna and human body interaction. *IEEE transactions on microwave theory and techniques* 44:1855–1864.
- Ostling O, Johanson KJ. 1984. Microelectrophoretic study of radiation-induced DNA damages in individual mammalian cells. *Biochemical and Biophysical Research Communications* 123:291–298.
- Phillips JL, Ivaschuk O, Ishida-Jones T, Jones RA, Campbell-Beachler M, Haggren W. 1998. DNA damage in Molt-4 T-lymphoblastoid cells exposed to cellular telephone radiofrequency fields in vitro. *Bioelectrochemistry and Bioenergetics* 45:103–110.
- Phillips JL, Singh NP, Lai H. 2009. Electromagnetic fields and DNA damage. *Pathophysiology* 16:79–88.
- Pride HB, Tunnessen WW. 1996. Clinical picture. *Archives of family medicine*. www.archfam.med.com
- Potten CS, Burt PA, Roberts SA, Deshpande NA, Williams PC, Ramsden J. 1996. Changes in the cellularity of the cortex of human hairs as an indicator of radiation exposure. *Radiation and Environmental Biophysics* 35:121–125.
- Purschke M, Laubach HJ, Anderson RR, Manstein D. 2010. Thermal injury causes DNA damage and lethality in unheated surrounding cells: Active thermal bystander effect. *Journal of Investigative Dermatology* 130:86–92.
- Ruediger HW. 2009. Genotoxic effects of radiofrequency electromagnetic fields. *Pathophysiology* 16:89–102.
- Scarf MR, Fresegna AM, Villani P, Pinto R, Marino C, Sarti M, Altavista P, Sannino A, Lovisolo GA. 2006. Exposure to radiofrequency radiation (900 MHz, GSM signal) does not affect micronucleus frequency and cell proliferation in human peripheral blood lymphocytes: An interlaboratory study. *Radiation Research* 165:655–663.
- Sharpless NE, DePinho RA. 2007. How stem cells age and why this makes us grow old. *Nature Reviews Molecular Cell Biology* 8:703–713.
- Singh NP, McCoy MT, Tice RR, Schneider EL. 1988. A simple technique for quantization of low levels of DNA damage in individual cells. *Experimental Cell Research* 175:184–191.
- Singh NP. 1996. Microgel electrophoresis of DNA from individual cells: Principles and methodology. In: Pfeifer J, editor. *Technology for detection of DNA damage and mutation*. New York: Plenum Press. pp 3–24.
- Speit G, Scütz P, Hofman H. 2007. Genotoxic effects of exposure to radiofrequency electromagnetic field (RF-EMF) in cultured mammalian cells are not independently reproducible. *Mutation Research* 626:42–47.
- Straume A, Oftedal G, Johnsson A. 2005. Skin temperature increase caused by a mobile phone: A methodological infrared camera study. *Bioelectromagnetics* 26:510–519.
- Stronati L, Testa A, Moquet J, Edwards A, Cordelli E, Villani P, Marino C, Fresegna AM, Appolloni M, Lloyd D. 2006. 935 MHz cellular phone radiation: An in vitro study of genotoxicity in human lymphocytes. *International Journal of Radiation Biology* 82:339–346.
- Sykes PJ, McCallum BD, Bangay MJ, Hooker AM, Morley AA. 2001. Effect of exposure to 900 MHz radiofrequency radiation on intrachromosomal recombination in pKZ1 mice. *Radiation Research* 156:495–502.
- Takahashi A, Matsumoto H, Nagayama K, Kitano M, Hirose S, Tanaka H, Mori E, Yamakawa N, Yasumoto J, Yuki K, Ohnishi K, Ohnishi T. 2004. Evidence for the involvement of double-strand breaks in heat-induced cell killing. *Cancer Research* 64:8839–8845.
- Targovnik HS, Locher SE, Hariharan PV. 1985. Age associated alterations in DNA damage and repair capacity in *Turbatrix acetaboli* exposed to ionizing radiation. *International Journal of Radiation Biology* 47:255–260.
- Wu W, Yao K, Wang KJ, Lu DQ, He JL, Xu LH, Sun WJ. 2008. Blocking 1800 MHz mobile phone radiation-induced reactive oxygen species production and DNA damage in lens epithelial cells by noise magnetic fields. *Zhejiang Da Xue Xue Bao Yi Xue Ban* 37:34–38.
- Verschaeve L, Slaetes D, Van Gorp U, Maes A, Vankerom J. 1994. In vitro and in vivo genetic effects of microwaves from mobile telephone frequencies in human and rat peripheral blood lymphocytes. In: Simunic D, editor. *Proceedings from the COST 244 meetings on mobile phone communication and extremely-low-frequency field: Instrumentation and measurements in bioelectromagnetics research*. Bled, Slovenia. pp 74–83.
- Zeni O, Chiavoni AS, Sannino A, Antolini A, Forigo D, Bersani F, Scarf MR. 2003. Lack of genotoxic effects (micronucleus induction) in human lymphocytes exposed in vitro to 900 MHz electromagnetic fields. *Radiation Research* 160:152–158.
- Zeni O, Romano M, Perrotta A, Lioi MB, Barbieri R, d'Ambrosio G, Massa R, Scarf MR. 2005. Evaluation of genotoxic effects in human peripheral blood leukocytes following an acute in vitro exposure to 900 MHz radiofrequency fields. *Bioelectromagnetics* 26:258–265.