

ORIGINAL ARTICLE

A cross-sectional case control study on genetic damage in individuals residing in the vicinity of a mobile phone base station

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*Department of Human Genetics, Guru Nanak Dev University, Amritsar, Punjab, India***Abstract**

Mobile phone base stations facilitate good communication, but the continuously emitting radiations from these stations have raised health concerns. Hence in this study, genetic damage using the single cell gel electrophoresis (comet) assay was assessed in peripheral blood leukocytes of individuals residing in the vicinity of a mobile phone base station and comparing it to that in healthy controls. The power density in the area within 300 m from the base station exceeded the permissive limits and was significantly ($p = 0.000$) higher compared to the area from where control samples were collected. The study participants comprised 63 persons with residences near a mobile phone tower, and 28 healthy controls matched for gender, age, alcohol drinking and occupational sub-groups. Genetic damage parameters of DNA migration length, damage frequency (DF) and damage index were significantly ($p = 0.000$) elevated in the sample group compared to respective values in healthy controls. The female residents ($n = 25$) of the sample group had significantly ($p = 0.004$) elevated DF than the male residents ($n = 38$). The linear regression analysis further revealed daily mobile phone usage, location of residence and power density as significant predictors of genetic damage. The genetic damage evident in the participants of this study needs to be addressed against future disease-risk, which in addition to neurodegenerative disorders, may lead to cancer.

Keywords

DNA damage, radiofrequency radiations, peripheral blood leukocytes

History

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Introduction

The wireless technology has seen unprecedented expansion the world over and has become all pervasive. Though on one hand, there has been indispensable improvement in the quality of communication, yet it has also emerged as an unceasing source of radiofrequency radiations (RFRs) being emitted, both from mobile (cell) phone base stations and the cell phone itself, which acts as a two-way radio, i.e. transceiver (Kwan-Hoong, 2005), generally operating in the frequency range of 900 MHz–1.9 GHz (Levitt and Lai, 2010). According to the Telecom Industry of India (Telecom sector in India, 2012), the Indian Telecommunications network is the third largest in the world and the second largest among the emerging economies of Asia; the industry continues to grow having 540 000 communication towers with more and more towers being erected (DoT, 2012). There is also correspondingly high mobile phone subscribers' base, being second after China (Das, 2012). The need for an expansive network to maintain the escalating mobile phone subscribers' base has resulted in the proliferation of antennas atop masts, both in urban as well

as rural areas, adding to the quagmire of environmental pollutants as the RFRs.

The continuous emission of RFR has prompted concerns about its effect and the potential risks to those living near mobile phone base stations despite the fact that the micro-waves in the RFR spectrum are of low frequency (ARPANSA, 2011). Besides affecting the well-being and performance of the population, headaches, sleep disturbances, discomfort, irritability, depression, memory loss and concentration problems have been documented in France (Santini et al., 2002), Spain (Navarro et al., 2003), Poland (Bortkiewicz et al., 2004) and Egypt (Abdel-Rassoul et al., 2006). In Austria, where the exposure limits (0.001 W/m^2) are among the lowest in the world, health symptoms included buzzing in the head, heart palpitations, unwellness, lightheadedness, anxiety, breathlessness, respiratory problems, nervousness, agitation, headaches, tinnitus, heat sensation and depression (Oberfeld et al., 2004). Of more concern are studies on the occurrence of cancers among those residing near mobile phone base stations. A four-fold increase in the incidence of cancers of all kinds among residents living within 300-m radius of a mobile phone mast from three to seven years has been reported (Wolf and Wolf, 2004). In another study, a three-fold increase in the incidence of malignant tumors of blood, breast, ovary, pancreas, stomach, lung, kidney, bowel, prostate and skin melanoma was found after five years' exposure in people

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living within 400-m radius of a mobile phone mast (Eger et al., 2004). An earlier study had indicated an association between an increase in cancer and living in proximity to a mobile base station (Cherry, 2000). According to a study near the transmitter station of Radio Vatican, there were 2.2 times more leukemia cases in children within a radius of 6 km, as well as an increase in adult mortality from leukemia (Michelozzi et al., 2002).

The carcinogenic influence of RFRs (Hardell et al., 2007; Stein et al., 2011), however, continues to be debated though the International Agency for Research on Cancer (IARC, 2011) classified RF as possibly carcinogenic to humans (Group 2B carcinogen), based on an increased risk for glioma (malignant type of brain cancer) associated with wireless phone use (Hardell et al., 2013).

Besides effects on human health and association of cancer in persons staying near mobile phone base stations, a vast array of other biological effects have been documented to be associated with exposure to RFR in both, *in vitro* and *in vivo* studies. Some of the recent literature reinforces the earlier studies on causation of oxidative stress (Kesari et al., 2010; Garaj-Vrhovac et al., 2011; Khalil et al., 2012) and DNA strand breaks (Kim et al., 2012; Schwarz et al., 2008) as well as affecting reproduction, particularly with effects on sperm physiology and DNA (Atasoy et al., 2012; Avendaño et al., 2012; Chavdoula et al., 2010). Electromagnetic fields (EMFs), from extremely low frequency (ELF, 30–300 Hz), radio frequency and microwave frequency (100 kHz–300 GHz) ranges, have also been reported to activate the cellular stress response as a protective mechanism inducing the expression of stress response genes (heat shock protein 70) causing the low energy EMF to interact with DNA, causing DNA strand breaks (Panagopoulos et al., 2007).

Considering that RFR can induce cell death (Blank and Goodman, 2009), genetic damage (Lixia et al., 2006) and contribute toward neoplastic cell transformation (Halliwell, 2000; Marnett, 2001) and since no studies on the genomic damage assessment in individuals residing near base stations have come to attention, this study (a first of its kind) investigated DNA damage using the alkaline single cell gel electrophoresis (SCGE/comet) assay in the peripheral blood leukocytes (PBL) of a group of individuals residing near a mobile phone base station.

Materials and methods

Study-design

A case–control cross-sectional study was carried out in the city of Amritsar (31.6167°N, 74.8500°E), Punjab, India, on a total of 91 individuals with 70% ($n=63$) residing in a populated area with a mobile phone base station (the sample group) and 30% ($n=28$) in a sparsely-populated zone without any nearby base stations (the control group). At the time of the study (2007–2009), it was fortunate that such areas existed; random sampling (one person per household with every fifth house being sampled) was carried out both, near the mobile phone base station and from an area without any nearby mobile phone base station, and hence the importance of this study can in no way be undermined.

The network providers avail the 800–2200 MHz bandwidth part of the frequency range for use in mobile phone communication technology. This wavelength is generated *via* an EMF generator located at the base of a mast/tower, which is in the operation mode to provide round-the-clock communication facility (WHO, 2011). The microwaves from the point of generation are fed through cables leading to the top of the tower-structure and beamed 360° through antennae (disc- or sector-shaped) placed on the lattice-structure of the tower/mast. The microwave forms a floral pattern as it is beamed from the antennae with central petal forms being positioned at the horizontal plane up to 300 m or so and shorter ones falling closer to the tower (Kimura and Ebine, 2005). Areas from 50 to 250 m have the most radiation; concrete buildings facing the towers have higher RFR, followed by those below and as compared to adjacent ones (Nayyeri et al., 2013). The continuous 24 × 7 RF (microwave) emissions within a 300-m radius of a mobile base station therefore must be having an impact on those residing there. In this study, the focus was to assess genetic insult in the PBL using the well-validated SCGE assay for human bio-monitoring studies (Dusinska and Collins, 2008).

Power of the study

A sample size of 70 subjects was calculated to be sufficient for the primary endpoint of the study as derived from genetic damage data obtained in a pilot study with the significance level of $p < 0.05$ and a power of 80%. Retrospectively also, the power of study is 92% implying that this study sample size of 91 individuals is more than sufficient to discern statistical increase in genetic damage at 5% level of significance.

Study participants

Inclusion criteria

The sample group comprised individuals who fulfilled the inclusion criteria of being above 18 years of age and residing within a distance of 50–300 m from a mobile phone tower, absence of any other exposure(s), disease or recent illnesses and not being on any prescribed/other medication. The control group comprised healthy individuals matched for age and sex, neither residing nor working in areas with mobile phone base station(s) nearby and without any other exposures.

Ethical clearance and informed consent

Approval of the study (consideration of the ethical aspects) was obtained from the Institutional Ethics Committee, and only those individuals who gave their voluntary written consent (after the detailed information about the study had been explained to them) formed the study group.

The mobile phone base station

Among the base stations/towers ($n=90$) in Amritsar at that period of time, the base station as a lattice structure in Kabir Park opposite Guru Nanak Dev university had been erected in 1998 by the Airtel Network provider with as many as eight dish- and 11 sectored-antennas, arranged equilaterally so as to provide 360° network coverage. The tower was installed on the roof-top of a residence (5 m) with the mast height of 15 m.

The roof-top had been rented out to the Airtel Network Company for a period of 12 years, while the owners resided on the ground floor. The residence on which the tower is erected faces a road on one side, while there are residences on all the other three sides. As per guidelines of siting of mobile base stations, the installation height should be 20–30 m, and the tower should not be within the premises of schools, hospitals and in narrow lanes (≤ 5 m) having no nearby buildings especially right in front of the antenna (DoT, 2012).

RF measurements

Radiofrequency field measurements were taken at varied locations from households from where sampling was done. A hand-held monitor (Reliance KP100FL-01-, Mumbai, India) was used to record the radiofrequency as per the instruction manual. Power density was recorded in decibels (dbm). RF measurements were taken for a total of 91 sites; at each site, the best reading was recorded and converted to power density (W/m^2) by using a conversion formula ($dbm = 10 \times \log_{10} mW/m^2$) as per Elliott et al. (2010). In India, the safety limits for public exposures from base stations at the time of study were 36.6 dbm ($4.5 W/m^2$) for 900 MHz, 74.82 dbm ($9.2 W/m^2$) for 1800 MHz frequency, which have been decreased to one-tenth level as since September 2012 as per Department of Telecommunications guidelines (DoT, 2012).

Methodology

A questionnaire, after consultation of literature to record relevant and appropriate information for the objectives of the study, was prepared in collaboration with other members of the laboratory doing a parallel study (in preparation). A face-to-face interview method was used, and information on general demography, genetic, family and exposure histories, life-style (smoking, alcohol consumption and dietary pattern), occupation, duration of stay near and distance of household from mobile phone base station, mobile phone usage (average daily use and duration of using) and on medical issues (exposure to X-rays, vaccinations and medication) was recorded on the questionnaire.

Sample collection

Finger-prick blood samples (300 μ l) were collected from the study participants in heparin-containing vials, which were transported on ice to the laboratory and processed within 2–3 h for genetic damage assessment.

Principle of the SCGE assay

The SCGE assay is a sensitive yet simple and versatile technique, for the assessment of genetic damage. The agarose-embedded leukocytes are treated with lysing solution, which aids in removal of cell membranes, cytoplasm, nucleoplasm and in the dissolution of the nucleosomes (Nandhakumar et al., 2011). The treatment of the nucleoid with alkaline solution causes supercoiling of DNA leading to unwinding thereby exposing the alkali-labile sites, which migrate toward the anode (DNA being negatively charged) on electrophoresis. DNA migration from the nucleoid produces a comet-like appearance, and this is an important measure of genetic damage assessment (Collins, 2004).

The alkaline single cell gel electrophoresis assay

The alkaline SCGE assay was performed according to Singh et al. (1988) with minor modifications and use of chemicals of analytical grade procured locally (SRL, Mumbai, India). Blood (30 μ l) was mixed with 100 μ l of 0.5% low-melting point agarose and spread on the slides previously coated with 1% normal melting point agarose. For each, two slides per sample were prepared. The slides were immersed for 2–4 h in fresh lysing solution (2.5 M NaCl, 100 mM EDTA, 10 mM Tris-HCl, pH 10) in which at the time of use, addition of 1% Triton X-100 and 10% dimethyl sulfoxide was done. The slide preparations were submitted to electrophoresis for 25 min at 300 mA and 25 V (1.0 V/cm run rate) after an earlier incubation of 25 min in alkaline buffer solution (300 mM NaOH, 1 mM EDTA, pH > 13.0). The slides were then neutralized by washing with the neutralization buffer (400 mM Tris-HCl, pH 7.5). Following the fixation of the cells in fixing solution (75 g of TCA, 25 g of ZnSO₄ and 25 ml glycerol), the slide preparations were stained with silver nitrate (0.2% AgNO₃) as per Nadin et al. (2001).

Slide scoring

One-hundred cells per individual (two slides scored for 50 cells each) were analyzed at 40 \times under a binocular microscope (Magnüs MLX-DX 4B 523830, Olympus, India); a calibrated ocular micrometer was used for the manual measurement of DNA migration length as well as for visual scoring of nucleoids, which is considered a well-validated evaluation method (Collins, 2004). Comet assay parameters were recorded, according to da Silva et al. (2008). The mean DNA migration length (μ m) was obtained as the difference between the comet tail (from head to the trailing end of comet) and the radius of the head. Based on the number of cells with tails and their categorization into classes 0–4 (Collins, 2004), the number of cells with tails comprised the damage frequency (DF). The damage index (DI) was calculated (da Silva et al., 2008) for each sample depending on nucleoids with/without damage, ranging from 0 (no damage) to 400 (maximum damage) by multiplying the cell category with cells in each category from 0 to 4. The DI is based on the length of migration and on the amount of DNA in the tail; it is considered a sensitive measurement of detectable DNA damage (Grisolia et al., 2009).

Statistical analysis

The values are expressed as mean \pm standard error of mean. The Kolmogorov and Smirnov tests revealed normal distribution of the data, and hence parametric tests were performed. Chi-square analysis was carried out for comparison of categorical variables of the exposed and control groups. The Students *t*-test was performed for comparison of the mean values within and of the various study variables with the genetic damage parameters. To check for association (if any) between the confounding factors and genetic damage, the analysis of variance and the Pearson's correlation analysis were performed. To assess the independent statistical relationships of the confounding factors simultaneously, a multivariate linear regression analysis was also carried out.

The tests were interpreted using a 5% degree ($p \leq 0.05$) of significance. Statistical analyses were conducted using the Statistical Package for Social Sciences, version 16.0 software (SPSS, Chicago, IL) and the MedCalc statistical software package for Windows (Ostend, Belgium).

Results

The general characteristics of the study participants are listed in Table 1. The 38 male (25.93 ± 0.85 years) and 25 female (26.35 ± 1.15 years) residents in the sample group had been staying within 300 m of the base station for an average of 7.42 ± 0.25 years (range 4–10 years). All (but one resident) were cell phone users (average duration 4.03 ± 0.23 years), while there were eight users (28.57%) in the control group (0.33 ± 0.14 years) showing significant difference ($p = 0.000$) for mobile phone usage between the groups. The respective average daily cell phone usage was 2.03 ± 0.16 h and 0.50 ± 0.00 h, also being highly significant ($p = 0.000$) between the groups. Health complaints were recorded in 26.98% of residents near mobile phone base stations mainly related to headache and tinnitus. The groups matched for age (25.93 ± 0.85 years of sample group vs. 26.35 ± 1.15 years in controls) as well as for gender, alcohol drinking and occupation-type but not for mobile phone usage, dietary pattern, smoking habits or health complaints on Chi-square analysis.

In respect of RF, the power density measurements at the sampling sites ($n = 63$) near the base station ranged from 7.6 W/m^2 – 14.59 W/m^2 (average $11.49 \pm 0.17 \text{ W/m}^2$), whereas in the areas from where the control group was sampled ($n = 28$ sites), the value was significantly ($p = 0.000$) lower (0.001 – 0.1 W/m^2 ; average $0.045 \pm 0.00 \text{ W/m}^2$). The highest power density values (Table 2) were at a distance of 50–100 m ($12.21 \pm 0.32 \text{ W/m}^2$) followed by those at 151–200 m ($11.01 \pm 0.42 \text{ W/m}^2$), 201–250 m ($10.60 \pm 0.39 \text{ W/m}^2$) and with least at 251–300 m ($10.41 \pm 0.22 \text{ W/m}^2$) indicating that as the distance from mobile phone base station increased, the power density decreased. This was also evident on correlation analysis as a significant negative association between distance from base station and the power density values ($r = -0.495$, $p = 0.000$) were obtained. Significantly increased power density was observed at a distance of 50–100 m compared to those at 151–200 m ($p = 0.035$), 201–250 m ($p = 0.005$) and 251–300 m ($p = 0.001$). At distance 101–150 m, the power density was also significantly increased from that at 151–200 m ($p = 0.010$), 201–250 m ($p = 0.002$) and 251–300 m ($p = 0.000$). On comparing the values of power density for location of residence (opposite/adjacent) from the base station, no statistically significant differences were observed.

Genetic damage was significantly ($p = 0.000$) increased in the sample group compared to that in the controls (Tables 3–5). The Student's *t*-test revealed a highly ($p = 0.000$) significant (2.5-fold) increase in DF, 3.5 times of DI and 4.5-fold elevated mean DNA migration length in comparison to values in the control group. The females compared to males among mobile phone base station residents had significantly elevated DF ($p = 0.004$), whereas the DNA migration lengths showed no statistical differences between the genders.

Genetic damage as a function of independent variables of age, location of residence (opposite/adjacent), distance

(50–300 m), duration of stay (4–10 years) as well as mobile phone usage/non-usage and duration of usage (≤ 5 / > 5 years; ≤ 3 / > 3 years), was also assessed.

As a function of distance of residence from the mobile phone base station (50–300 m), genetic damage at all sub-intervals of 50 m (Table 4) had significantly elevated genetic damage in both, those using ($p \leq 0.01$) or not using cell phones ($p \leq 0.001$). Both, males and females residing between 50 and 100 m had statistically elevated DI and DNA migration lengths compared to those residing further away. DI ($p = 0.001$) and mean DNA migration length ($p = 0.002$) were significantly higher in males compared to values in females residing between 50 and 100 m of the base station though this was vice-versa in those at 151–200 m (data not shown). Significantly elevated ($p = 0.000$) genetic damage for all parameters was observed in groups residing from 4 to 7 years and from 8 to 11 years in the vicinity of mobile phone base station (Table 2) compared to controls. Females staying near mobile phone base station also had significantly increased ($p = 0.012$) DF compared to values in males of this group (data not shown).

Genetic damage as a function of features of mobile phone usage and of SAR values of phone-sets is listed in Table 5. There were 33 (52.38%) individuals in the sample group using mobile phones ≤ 5 y and 50 (79.36%) with mobile phone usage of ≤ 3 h. Among controls, 28.57% had been using mobile phones for ≤ 5 years with a daily usage of ≤ 3 h. As a function of mobile phone usage as well as SAR values of mobile phone models used by the study participants, significantly elevated ($p = 0.000$) genetic damage was observed in sample group compared to the values in controls. The residents in all these categories had higher genetic damage and no gender differences.

The analysis of variance and Pearson's correlation analysis (Table 6) revealed significant association of DF with daily mobile phone usage ($p = 0.002$), of DI with location of residence ($p = 0.015$) and of mean DNA migration length with both, location ($p = 0.018$) and age ($p = 0.035$), while all the three parameters of genetic damage showed significant association with power density ($p = 0.032$ for DF; $p = 0.017$ for DI; and $p = 0.015$ for mean DNA migration length). Among controls, mobile phone users ($p = 0.000$), duration ($p = 0.000$), daily mobile phone usage ($p = 0.000$) and SAR values ($p = 0.000$) contributed to the elevated DF and DI, while duration of mobile phone usage ($p = 0.000$) and SAR ($p = 0.000$) value also showed association with mean DNA migration length. The location of residence, power density and daily mobile phone usage emerged as significant predictors of genetic damage on multivariate linear regression analysis.

Discussion

The consistent human exposure from electromagnetic radiations as a result of network expansion has raised public concerns in those using cell phones and in particular more so in those with residential proximity to mobile phone base stations. The emissions are ultra-high frequency waves (microwaves) with amplitude modulation and ELF pulsation (Santini, 1999). A number of studies (Elliott et al., 2010;

Table 1. Demographic characteristics of individuals residing in the vicinity of a mobile phone base station and of controls.

Characteristics	Range	Sample group		Control group		χ^2/t -Value	<i>p</i> Value (χ^2/t -test)
		<i>N</i> (%)	Mean \pm SEM*	<i>N</i> (%)	Mean \pm SEM*		
Age (years)	18–31 32–45	48 (76.19) 15 (23.80)	25.93 \pm 0.85	23 (82.14) 05 (17.85)	26.35 \pm 1.15	0.129/0.294	0.719/0.779
Gender							
Males		38 (60.31)	–	15 (53.57)	–	0.138	0.709
Females		25 (39.68)		13 (46.42)			
Lifestyle							
Dietary preference							
Veg		29 (46.03)	–	20 (71.42)	–	4.061	0.043
Non-Veg		34 (53.96)		08 (28.57)			
Alcohol drinking							
Yes		01 (1.58)	–	02 (7.14)	–	0.539	0.463
No		62 (98.41)		26 (92.85)			
Smoking habit							
Yes		16 (25.39)	–	01 (3.57)	–	4.727	0.029
No		47 (74.60)		27 (96.42)			
Occupation							
Student		36 (57.14)	–	12 (42.85)	–	7.419	0.283
Clerks		07 (11.11)		03 (10.71)			
Housewives		07 (11.11)		09 (32.14)			
Teachers		07 (11.11)		03 (10.71)			
Salesperson		03 (4.76)		–			
Shopkeepers		02 (3.17)		01 (3.57)			
Land owners		01 (1.58)		–			
Time since residing in the vicinity of the base station(y)							
4–7		31 (49.20)	7.42 \pm 0.25	–	–	–	–
8–11		32 (50.79)					
Distance from mobile phone base station (m)							
50–100		13 (20.63)	180.16 \pm 9.63	–	–	–	–
101–150		18 (28.57)					
151–200		14 (22.22)					
201–250		09 (14.28)					
251–300		09 (14.28)					
Location of residence							
Opposite		29 (46.03)	–	–	–	–	–
Adjacent		34 (53.96)					
Mobile Phone usage							
User		62 (98.41)	–	08 (28.57)	–	49.404	0.0001
Non user		01 (1.58)		20 (71.42)			
Mobile Phone using since (years)							
\leq 5		33 (52.38)	4.03 \pm 0.23	08 (28.57)	0.33 \pm 0.14	4.606/13.32	0.031/0.000
$>$ 5		29 (46.03)		–			
Daily mobile phone use (h)							
\leq 3		50 (79.36)	2.03 \pm 0.16	08 (28.57)	0.50 \pm 0.00	0.755/11.11	0.385/ 0.000
$>$ 3		12 (19.04)		–			
SAR (W/kg) (range)		0.43–0.92	0.68 \pm 0.02	0.50–0.88	0.17 \pm 0.10	7.911	0.000
Power density (W/m ²) at residential sites (range)		7.6–14.59	11.49 \pm 0.17	0.01–0.1	0.045 \pm 0.00	43.92	0.000
Health complaints							
Tinnitus		12 (19.04)	–	–	–	–	–
Headache		06 (9.52)					
Irritability		04 (6.34)					
Discomfort		03 (4.76)					
Nausea		02 (3.17)					
Combinational effects							
Tinnitus + Irritability		02 (3.17)	–	–	–	–	–
Tinnitus + Discomfort		02 (3.17)					
Tinnitus + Irritability + Discomfort		01 (1.58)					
Nausea + Tinnitus + Irritability		01 (1.58)					
Headache + Tinnitus		01 (1.58)					

SAR, specific absorption rate.

*Student's *t*-test.*p* Values in bold are significant (*p* < 0.05).

Table 2. Power density as a function of distance and location of residence from mobile phone base station.

Study variable	Power Density(W/m ²) ± SEM		
Distance from mobile phone base station (m)	50–100	12.21 ± 0.32 ^{a-c}	
	101–150	12.32 ± 0.17 ^d	
	151–200	11.01 ± 0.42 ^e	
	201–250	10.60 ± 0.39 ^f	
	251–300	10.41 ± 0.22 ^g	
Location with respect to mobile phone base station	Opposite	11.32 ± 0.23	
	Adjacent	11.69 ± 0.24	
Pearson correlation analysis			
Study variable	Pearson's correlation	Distance	Location of residence w.r.t base station
Power Density (W/m ²)	<i>r</i>	−0.495	−0.140
	<i>p</i>	0.000	0.275

Values with different letters are significantly different;

^aSignificant when compared to ^e($p=0.035$); ^bhighly significant when compared to ^f($p=0.005$); ^cvery highly significant when compared to ^g($p=0.000$); ^dhighly significant when compared to ^f($p=0.010$); ^every highly significant when compared to ^f($p=0.002$).

Value in bold is significant ($p<0.001$).

Heinrich et al., 2010) have revealed that RF exposures in publicly accessible areas are below the set-standards recommended by International Commission on Non-Ionizing Radiation Protection (ICNIRP, 1998). In India, the ICNIRP limit-levels before September 2012 were 4.5–9.2 W/m², while one-tenth of these since then (0.45 W/m²–0.92 W/m²). The SAR values of handsets were also reduced to 1.6 W/kg averaged over one gram of human tissue instead of 2.0 W/kg averaged over 10 gram of human tissue (DoT, 2012). During this study, the power density levels were much higher with average of 11.49 ± 0.17 W/m² and the range of 7.60–14.59 W/m², implying excessive radiation exposure.

Apart from the non-specific but nonetheless adverse health effects self-reported by the participants of this study from such continuous RF-EMF exposure and as also reported worldwide (Abdel-Rassoul et al., 2006; Eltiti et al., 2007; Oberfeld et al., 2004), the 2.5–4.5-fold increase in DNA damage in peripheral blood lymphocytes of persons staying near a mobile phone base station is of acute concern given that all neoplasia initiate via unrepaired DNA damage (Bernstein et al., 2013; Nambiar and Raghavan, 2011). This in no way can be ignored in the light of documented cases of malignancy in those staying in the vicinity of mobile phone base stations (Dode et al., 2011; Elliott et al., 2010). Location of residence with respect to mobile phone base station also turned out to be a significant predictor of the genetic damage in the residents. Comparative data from other studies are lacking as genetic damage has not been assessed in other studies making this study to be the first of its kind to the best of our knowledge. Results from other studies reveal a significant increase in neurological complaints in those having residences beneath compared to those staying opposite to mobile base stations. (Abdel-Rassoul et al., 2006). The facing-position of residents toward phone base stations and <100-m distance caused increased prevalence of sleep disturbances, fatigue and feelings of discomfort (Alazawi, 2011). Besides, the self-reported ill-health symptoms of tinnitus, headaches, irritability, discomfort and nausea the results of this study have also shown increased genetic damage (2.5–4.5-fold) in those residing at 50-m

intervals (50–100 m to 251–300 m) from a mobile phone base station.

Mobile phone usage was prevalent among 98.41% of the study participants and genetic damage as function of mobile phone usage revealed 2.00-fold in those having 4.03 ± 0.23 years of usage and 1.30-fold increase in those with 2.03 ± 0.16 h of daily usage. It is also of interest that in the control group there is almost 3-fold difference in DNA migration length between mobile phone users and non-users. The difference could probably have accrued from the usage of mobile phone, and as communication in mobile telephony is through RFR, therefore this sub-group of the controls could be manifesting increased DNA damage from mobile phone usage (from RFR) compared to non-mobile phone users given that this is the main difference in the control sub-groups, which can influence genetic damage. This can be borne-out as on carrying out ANOVA, correlation and multiple linear analyses, SAR of hand-sets, duration and average daily phone usage showed association with DNA migration length. These results substantiate earlier studies from our laboratory in which mobile phone usage in all probability (in the absence of other exposures/disease) caused significantly increased DNA migration and micronucleated cells in PBLs (Gandhi and Anita, 2005; Gandhi and Singh, 2011) as well as clastogenic/aneugenic events in both, buccal mucosa and cultured peripheral blood lymphocytes (Gandhi and Singh, 2005). This increase in genetic damage has implications as it is the first step in carcinogenesis. The risk of ipsilateral cerebral cancers and benign tumors of the acoustic nerve were reported in mobile phone users after a latency period of ≥10 years have also been reported (Hardell et al., 2006; Hardell and Carlberg, 2009).

Though no similar studies on humans from RF exposures have come to attention, RF exposures in the mobile phone range (900 MHz–1800 MHz) have been observed to induce genomic damage *in vivo* and *in vitro*. Contradictory results with no effects have also been documented in *in vitro* studies where 900 MHz irradiation was not mutagenic to human–hamster hybrid cells (Hintzsche and Stopper, 2010) and failed to induce DNA strand breaks in rat cells (Usikal and

Table 3. Genetic damage in individuals residing in the vicinity of mobile phone base station as a function of gender and age.

Characteristics	Study group	Sample group				Control group			
		n	Percent cells with tails (DF) ± SEM	Damage index (DI) ± SEM	Mean DNA migration length (µm) ± SEM†	n	Percent cells with tails (DF) ± SEM	Damage index (DI) ± SEM	Mean DNA migration length (µm) ± SEM†
Age (years)	≤30	M	29	95.75 ± 1.24*** ^a	135.31 ± 4.75***	27.60 ± 1.06***	45.91 ± 7.11	46.00 ± 7.14	6.66 ± 1.21
		F	19	99.52 ± 0.37***	132.42 ± 6.58***	26.45 ± 1.43***	26.63 ± 5.82	28.63 ± 4.08	5.00 ± 1.24
		T	48	97.25 ± 0.80	134.17 ± 3.84	27.15 ± 0.85	36.69 ± 4.97	37.69 ± 4.51	5.87 ± 0.86
	>30	M	09	95.55 ± 1.95***	145.11 ± 12.50**	30.75 ± 2.45***	48.00 ± 20.03	48.00 ± 20.03	7.31 ± 3.53
		F	06	100.00 ± 0.00***	140.08 ± 14.04**	28.76 ± 2.65**	40.50 ± 30.50	29.50 ± 19.50	6.21 ± 4.60
		T	15	97.33 ± 1.28	143.40 ± 9.01	29.95 ± 1.76	43.00 ± 11.44	40.60 ± 13.37	6.87 ± 2.43
Gender	M	38	95.71 ± 1.04***	137.63 ± 4.62***	28.35 ± 1.00***	46.33 ± 6.58	46.40 ± 6.60 ^c	6.79 ± 1.13	
	F	25	99.64 ± 0.28***	134.44 ± 5.92***	27.01 ± 1.24	28.76 ± 6.16	28.76 ± 4.07 ^c	5.19 ± 1.17	
	Total	63	97.26 ± 0.68***	136.37 ± 3.62***	27.81 ± 0.78***	38.17 ± 4.76	38.21 ± 4.28	6.05 ± 0.81	

†Taken as average of means.

***Very highly significant ($p \leq 0.0001$).**Highly significant ($p \leq 0.01$) when compared to controls, values with similar letters are significantly different.^aSignificant when compared within sample group ($p = 0.021$).^bHighly significant when compared within sample group ($p = 0.004$).^cSignificant when compared within control group ($p = 0.038$).

Table 4. Genetic damage in individuals residing in the vicinity of Mobile Phone Base Station as a function of staying near the base station.

Characteristics	Study group	n	Percent cells with tails (DF) ± SEM	Damage index (DI) ± SEM	Mean DNA migration length (µm) ± SEM†
Distance (m)	50–100	13	99.03 ± 0.51***	138.16 ± 7.66***	27.90 ± 1.72***
	101–150	18	96.83 ± 1.32***	137.88 ± 7.36***	27.98 ± 1.55***
	151–200	14	95.50 ± 2.17***	95.50 ± 2.17***	124.57 ± 5.78***
	201–250	09	97.22 ± 1.60***	98.22 ± 1.18***	27.03 ± 1.25***
	251–300	09	98.22 ± 1.18***	132.88 ± 6.22***	27.03 ± 1.25***
Time since residing near tower (years)	4–7	31	97.42 ± 0.76***	132.82 ± 4.12***	27.11 ± 0.92***
	8–11	32	96.88 ± 7.15***	145.22 ± 7.36***	29.58 ± 1.41***
Location of residence	Besides Facing	19	96.31 ± 1.57***	143.10 ± 6.96***	29.14 ± 1.46***
	Facing	44	97.68 ± 0.70***	133.45 ± 4.21***	27.24 ± 0.92***
Power density (W/m ²)	≤11.5	32	95.81 ± 1.11***	127.50 ± 4.27***	25.97 ± 0.98***
	>11.5	31	98.77 ± 0.69***	145.52 ± 5.50***	29.71 ± 1.13***
Control group	Mobile phone user	08	74.12 ± 3.28 ^a	67.62 ± 5.95 ^b	11.52 ± 0.68 ^c
	Mobile phone non user	20	23.80 ± 2.34 ^a	26.45 ± 2.42 ^b	3.86 ± 0.61 ^c
	Total	28	38.17 ± 4.76	38.21 ± 4.28	6.05 ± 0.81

†Taken as average of means.

***Very highly significant ($p \leq 0.0001$) when compared to controls, values with similar letters are significantly different^{a–c}Highly significant when compared within control group ($p = 0.000$).

Table 5. Genetic damage as a function of mobile phone usage in individuals residing in the vicinity of a mobile phone base station and in controls.

Characteristics	Study group	Sample group				Control group			
		Number of individuals (n)	Percent cells with tails(DF) \pm S.E.M.	Damage index (DI) \pm S.E.M.	Mean DNA migration length (μ m) \pm S.E.M.†	Number of individuals (n)	Percent cells with tails(DF) \pm S.E.M.	Damage index(DI) \pm S.E.M.	Mean DNA migration length (μ m) \pm S.E.M.†
Mobile usage	Users	63	97.26 \pm 0.68***	136.36 \pm 3.62***	27.81 \pm 0.78***	08	74.12 \pm 3.28 ^a	67.62 \pm 5.95 ^b	11.52 \pm 0.68 ^c
	Non-users	01	100.00 \pm 0.00	151.00 \pm 0.00	32.75 \pm 0.00	20	23.80 \pm 2.34 ^a	26.45 \pm 2.42 ^b	3.86 \pm 0.61 ^c
Duration of mobile phone usage (years)	\leq 5	52	97.15 \pm 0.77***	135.69 \pm 3.80***	27.54 \pm 0.81***	08	74.12 \pm 3.28	67.62 \pm 5.95	11.52 \pm 0.68
	$>$ 5	10	97.60 \pm 1.62***	138.40 \pm 11.84***	28.74 \pm 2.52***	–	–	–	–
Daily mobile phone usage (h)	\leq 3	50	96.80 \pm 0.83***	134.64 \pm 4.42***	27.35 \pm 0.94***	08	74.12 \pm 3.28	67.62 \pm 5.95	11.52 \pm 0.68
	$>$ 3	12	99.00 \pm 0.74***	142.33 \pm 4.46***	29.35 \pm 1.06***	–	–	–	–
SAR (W/kg)	\leq 0.6	20	96.30 \pm 1.16***	132.25 \pm 6.05***	26.55 \pm 1.27***	04	70.50 \pm 4.29	62.75 \pm 8.49	10.26 \pm 0.69
	$>$ 0.6	09	97.66 \pm 0.85***	137.98 \pm 4.62***	28.30 \pm 0.99***	04	77.75 \pm 4.80	72.50 \pm 8.79	12.79 \pm 0.78

†Taken as average of means.

***Very highly significant ($p \leq 0.0001$) when compared to controls; values with similar letters are significantly different.a–c Highly significant when compared within control groups ($p = 0.000$).

Akinyemi, 2012) and micronuclei in cultured human peripheral blood lymphocytes after 2450 MHz radiofrequency exposure (Vijayalaxmi et al., 2013). In studies reporting non-significant cytogenetic and genetic damage, the emphasis has been on the lack of thermal effects from microwaves (Verschaeve, 2005, 2009; Vijayalaxmi and Obe, 2004).

However, any biological response from RF exposure may be a result of thermal and non-thermal effects (Foster and Glaser 2007; Gaestel, 2010). Although the molecular mechanism is not apparent, changes in cell cycle (Friedman et al., 2007), induction of cell death/apoptosis (Caraglia et al., 2005) and modification of protein expression (Li et al., 2007) from oxidative stress (Oktem et al., 2005) resulting from microwave exposure have been reported. In spite of the fact that thermal effects cannot directly cause structural alteration of DNA (Foster, 2000), yet there can be the dysfunction of proteins involved in regulation of chromosome segregation and DNA replication (Mashevich et al., 2003). The phosphorylation of heat-shock proteins involved in cellular signal transduction pathways from exposure to electromagnetic low frequency radiations can lower the defense mechanism (Leszczynski et al., 2004) and cause oxidative stress. This has potential to oxidize proteins and lipids and inactivate enzymes resulting in structural and functional abnormalities and in causing oxidative damage to DNA and RNA, thereby increasing the mutation frequency and triggering carcinogenesis (Behari, 2010). Also as EMFs can induce apoptosis affecting cell division and cell proliferation, the cell cycle is altered (Blank and Goodman, 2009) besides promoting the formation of reactive oxygen species, which can cause cell damage (Zmysłony et al., 2004). Several experimental studies have reported both, single- and double-strand breaks in DNA and other chromosomal damage after exposure to EMFs (Çam and Seyhan, 2012; Deshmukh et al., 2013; Tsybulin et al., 2013). Following the WHO/International Agency for Research on Cancer (IARC, 2011) classifying radiofrequency EMFs as possibly carcinogenic to humans (Group 2B carcinogen), based on an increased risk for glioma, associated with wireless phone use, the Department of Telecom, India (DoT, 2012) had reduced the limits of RF radiated power from base station antennas to one-tenths of the existing level, i.e. 0.45 and 0.92 W/m² at 800 MHz and 1800 MHz, respectively. However, the RF measurements taken in the course of this study are higher than these lower limits, and the significantly elevated genetic damage in those staying near microwave emitters (mobile phone base stations) in the absence of other exposures further reinforce the cataclysmic nature of the radiations.

In the light of the above observations and the statistically significant genetic damage observed in those residing within 300 m of a mobile phone base station in this study, it implies that the effects of radiations from mobile phone base stations (in the absence of any other incidental/accidental exposures) cannot be overlooked, as unrepaired DNA damage can lead to cancer, precocious ageing and age-related effects (Kennedy et al., 2012; Bernstein et al., 2013). Such human biomonitoring studies should in no way be ignored. Rather, this study is a useful contribution to public health risk assessment, while more in-depth studies in this direction can help to elucidate the mechanism(s) underlying these observations.

Table 6. Analysis of variance (ANOVA), Pearson's Correlation and Multiple linear regression analyses in individuals residing in the vicinity of mobile phone base station and in controls.

Genetic damage parameters	Source of variation	Sample group							Control group <i>p</i> Value
		ANOVA		Multiple linear regression			Correlation <i>r</i>	<i>p</i> Value	
		F (variance ratio)	Mean square	B value	<i>t</i> Value	<i>p</i> Value			
DF	Age	0.532	15.783 29.650	0.093 (−0.130–0.278)	0.730	0.093	0.468	0.512	
	Dietary pattern	0.037	1.114 29.890	−0.025 (−3.030–2.497)	−0.193	−0.025	0.848	0.256	
	Alcohol drinking	0.102	3.042 29.859	−0.041 (−12.772–9.256)	−0.319	−0.041	0.751	0.603	
	Smoking habits	2.164	62.518 28.884	0.185 (−0.882–5.399)	1.471	0.185	0.146	0.943	
	Mobile phone use	0.254	7.574 29.784	0.064 (−8.226–13.775)	0.504	0.064	0.616	0.000	
	Distance from base station	0.288	8.567 29.768	−0.069 (−0.023–0.013)	−0.536	−0.069	0.594	–	
	Time since residing in the vicinity of mobile phone base station	0.225	6.707 29.798	0.061 (−0.517–0.838)	0.474	0.061	0.637	–	
	Location with respect to base station	0.082	2.436 29.868	−0.037 (−3.157–2.368)	−0.037	−0.286	0.776	–	
	Duration of mobile phone usage	1.350	39.227 29.265	0.147 (−0.309–1.157)	1.158	0.147	0.251	0.000	
	Daily mobile phone usage	10.194	261.227	0.378 (0.588–2.560)	3.193	0.378	0.002	0.000	
	SAR	1.359	38.600 29.276	0.145 (−3.460–12.792)	1.148	0.145	0.225	0.000	
	Power density	4.801	133.123 27.726	0.270 (0.095–2.077)	2.191	0.270	0.032	0.702	
	DI	Age	2.802	2255.836 805.029	0.210 (−0.173–1.953)	1.674	0.210	0.099	0.603
		Dietary pattern	1.143	944.583 826.525	0.136 (−6.763–22.300)	1.069	0.136	0.289	0.201
		Alcohol drinking	0.090	75.764 840.768	−0.038 (−67.221–49.673)	−0.300	0.038	0.765	0.561
Smoking habits		0.646	538.300 833.185	−0.102 (−23.422–9.991)	−0.804	−0.102	0.425	0.938	
Mobile phone use		0.260	217.635 838.442	0.065 (−43.49573.237)	0.509	0.065	0.612	0.000	
Distance from base station		0.332	278.123 837.450	−0.074 (−0.124–0.068)	−0.576	−0.074	0.567	–	
Time since residing in the vicinity of mobile phone base station		1.284	1058.965 824.650	0.144 (−1.545–5.585)	1.133	0.144	0.262	–	
Location with respect to base station		6.204	4741.528 764.280	−0.304 (−31.379–3.432)	−2.491	−0.304	0.015	–	
Duration of mobile phone usage		0.983	814.312 828.661	0.126 (−1.966–5.832)	0.991	0.126	0.325	0.000	
Daily mobile phone usage		1.669	1367.886 819.586	0.163 (−1.973–9.177)	1.292	0.163	0.201	0.000	
SAR		0.154	129.439	0.050 (−34.978–52.067)	0.393	0.050	0.696	0.000	
Power density		6.063	4643.473 765.887	0.301 (1.205–11.621)	2.462	0.301	0.017	0.472	
Mean DNA migration length		Age	4.667	169.158 39.244	0.267 (0.018–0.469)	2.160	0.267	0.035	0.250
		Dietary pattern	1.454	55.393 38.109	0.153 (−1.239–5.002)	1.206	0.153	0.233	0.491
		Mobile phone use	0.640	24.713 38.611	0.102 (−7.514–17.536)	0.800	0.102	0.427	0.000
	Alcohol drinking	0.156	6.057 38.917	−0.050 (−15.056–10.094)	−0.395	−0.050	0.695	0.461	
	Smoking habits	2.217	83.473 37.648	−0.187 (−6.196–0.907)	−1.489	0.187	0.142	0.776	
	Distance from base station	0.235	9.127 38.867	−0.062 (−0.026–0.016)	−0.485	0.062	0.630	–	
		1.114	42.686 38.317	0.134 (−0.363–1.174)	1.055	0.134	0.295	–	

(continued)

Table 6. Continued

Genetic damage parameters	Source of variation	Sample group						Control group p Value
		ANOVA		Multiple linear regression		Correlation	p Value	
		F (variance ratio)	Mean square	B value	t Value	r	p Value	
	Time since residing in the vicinity of mobile phone base station							
	Location with respect to base station	5.871	208.951 35.591	−0.296 (−6.669–0.638)	−2.423	−0.296	0.018	–
	Duration of mobile phone usage	1.469	55.964 38.099	0.153 (−0.329–1.343)	1.212	0.153	0.230	0.000
	Daily mobile phone usage	1.506	57.338 38.077	0.155 (−0.464–1.939)	1.227	0.155	0.224	0.461
	SAR	0.242	9.401 38.863	0.063 (−7.059–11.665)	0.492	0.063	0.625	0.000
	Power Density	6.238	220.821 35.397	0.305 (0.279–2.518)	2.498	0.305	0.015	0.329

DF, damage frequency and DI, damage index.

Values in bold are significant ($p < 0.05$).

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this article.

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