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DNA and Chromosomal Damage in Residents Near a Mobile Phone Base Station

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ABSTRACT Mobile phone base stations, continuously emit low-frequency radiofrequency (RF) radiations and thus are a cause of public health concern. In the present study, genetic damage in peripheral blood leukocytes (single cell gel electrophoresis/ comet assay) and buccal mucosal cells (buccal micronucleus cytome assay) of individuals residing in the vicinity of a mobile phone tower (n=50, power density 11.18±0.13 W/m^2), and in healthy controls from areas with no nearby towers (n=25, power density, 0.04±0.00 W/m^2), was assessed. Damage frequency, damage index, mean DNA migration length, frequencies of micronucleated, basal and pyknotic cells were significantly elevated (p=0.000) in the sample group. Age, diet, location of residences, distance from mobile phone base station and phone-set Specific Absorbance Rate values were significant predictors of genetic damage. Hence the observations indicate that 24x7 continuous exposure from base stations may pose genetic-damage threat to the populace residing nearby.

INTRODUCTION

The upsurge in the Indian mobile phone subscriber base reaching ~891 million (Dot 2013) has been concurrent with installation of mobile phone base station, there being 540,000 (Dot 2012). In the city of Amritsar there has been gross violations of installation norms and a rise in associated non-specific health symptoms in those residing near the base stations (Gandhi et al. 2013; Gandhi et al. 2014). Rather the increasing utility of radiofrequency radiation (RFR)-emitting consumer devices has for quite sometime drawn the attention of the public to possible health effects from RFR (10 KHz -300 GHz)- exposure (Maes et al. 2006). Epidemiological studies have suggested associations with symptoms such as headache, fatigue, and difficulty in concentration among people living in vicinity of mobile towers with higher potential exposures to radiation (Santini et al. 2002; Santini et al. 2003; Abdel-Rassoul et al. 2007; Gandhi et al. 2014; Suleiman et al. 2014). Documented biological effects have included EEG changes and calcium-ion efflux from extremely low frequency (ELF) electromagnetic fields (3Hz – 3KHz) and from radiofrequency (10 KHz -300 GHz) radiations (Lai 2001). Reports on genetic effects of ELF – EMF and RFR also exist (Phillips et al. 2009; Lai 2012;Singh and Kapoor 2014). Besides increase in DNA strand breaks (Diem et al. 2005; Franzellitti et al. 2010; Mihai et al. 2014), DNA – protein and DNA – DNA cross links (Blank and Goodman 2009) and chromosomal damage (Maes et al. 2006; Winker et al. 2005), neurological effects have also been observed associated with cases of dysaesthesia (Hocking and Westerman 2003). Genetic defects that predispose to the development of cancer particularly lymphomas and leukemias as well as birth defects such as Down's syndrome have also been reported (Carpenter 2010),though some studies have not demonstrated any link between increased cancer risk and RF exposure (INTERPHONE study group 2010; Frei et al. 2011). In fact rather the ability of radio frequency radiations in the microwave range to induce mutagenesis, chromosomal aberrations and carcinogenesis in different *in vivo* and *in vitro* systems has been equivocally reported (Hansteen et al. 2009; Ruediger 2009).

The growth in installation of mobile phone base stations has been very rapid in order to cater to the demands of increasing use of mobile telecommunication technology. The presence of base stations in densely-populated areas emitting RFR 24x7 may impact and compromise the

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health of those in the vicinity. A number of studies on adverse health effects as non-specific health symptoms in those staying near mobile phone base stations exists (Santini et al. 2002; Gadzicka et al. 2006; Abdel-Rassoul et al. 2007; Gandhi et al. 2014; Suleiman et al. 2014), yet studies on genetic damage assessment have not yet come to attention.

Objectives

In the present study DNA damage in the peripheral blood leukocytes (PBL) and chromosomal damage in buccal mucosal cells of some persons residing in the vicinity of mobile phone base stations was assessed.

MATERIAL AND METHODS

Mobile telecommunication services in Amritsar started in 1997 and by 2007 there were 105 mobile towers. One of the earliest base station was by the Airtel service provider, operating at a frequency of 900 MHz with nine antennas erected in 1998 on the roof-top of a hospital. RF measurements (in decibels) were taken at 50m intervals from base station during the course of the study using a hand-held monitor. The study was cleared by the base station where the study participants resided during the course of the study using a hand-held monitor. The study was cleared by the Institutional Ethics Committee. Study participation was requested from those residing/working near the base station. Written informed consent was taken from those voluntarily willing.

Exclusion Criteria

Vulnerable group (children, adolescents, elderly) and those adults with any past/present history of any incidental/accidental/occupational exposure(s) were excluded.

Inclusion Criteria

Healthy adults (18-45y) with no present or past (at least one year) history of any disease residing and working near a base station for 5-10y with a maximum stay of 15-24h/day.

A case-control approach was followed and age-, sex- and socio-economic status- matched healthy individuals who were not living in the vicinity of mobile phone base stations and had preferably never used mobile phone formed the control group. The power density was measured using a hand-held monitor (Reliance KP100FL-01-, India) both in area with and without a mobile phone base station. On a designed questionnaire, detailed information from the participants about time since residing/working in the vicinity of the tower, location of residence (in front of, besides) with respect to the tower, use of mobile phone, exposure history (chemicals/dyes/other radiations), diet, smoking, alcohol consumption, physical exercise, any major illness, medication, treatment in the past one year, etc. was recorded. Information for cytogenetic biomonitoring as per Carrano and Natarajan (1988) was also obtained.

Finger-prick blood samples (50 µl) were collected from the participants (n=75) in heparinized microcentrifuge tubes and were transported to laboratory in an ice-box and processed within 3-4 hours of collection for the alkaline Single Cell Gel Electrophoresis (SCGE/Comet) assay (Singh et al. 1988). The SCGE assay detects DNA damage viz. single strand breaks, alkali-labile sites and cross links. The buccal epithelial cells were taken to assess chromosomal damage using the Micronucleus (MN) Test (Nair et al. 1991). The MN Test provides a measure of both, chromosome breakage and loss and is an indicator of chromosomal damage as sensitive as classical metaphase chromosome analysis (Fenech and Morley 1985). All chemicals were procured locally.

Some modifications of the SCGE assay included use of 1% normal melting point agarose-coated slides in lieu of frosted slides and silver staining of nucleoids instead of using ethidium bromide (Ahuja and Saran 1989).Briefly, on the agarose-coated slide, a middle layer containing 30 µl of whole blood sample mixed in 0.5% low melting point agarose (LMPA) was poured and allowed to set followed by a third layer of 0.5%LMPA.Two slides were prepared per sample. In the next step, cell-lysis was carried out by treating the slide preparations with a freshly-prepared lysis solution (2.5M NaCl, 100mM Na_2EDTA,10Mm Tris-HCl, pH=10 to which 1%Triton X-100 and 10% dimethyl sulfoxide were added prior to use) followed by treatment with electrophoresis buffer (300Mm NaOH, 1Mm Na_2EDTA, pH 13) and an electrophoretic run (25min,25V,300mA, 1V/cm).The preparations were then neutralized and stained with silver nitrate (Delincee 1995). DNA damage was assessed from coded, blind slides at 40X using a standard

transmission binocular microscope (Magnus-MLX-DX 4B523830, India). For each sample, a total of 100 nucleoids (50 per slide) were scored and the DNA migration length was determined using a calibrate using a hand-held monitor oculo-micrometer. The nucleoids were visually categorized into different classes and assigned a value of 0 to 4 based on extent of DNA migration length as per Collins et al. (1995). Depending on the number of cells with tails, the damage frequency (DF) and damage index (DI) was determined for each sample (Franke et al. 2005).

The standard protocol of Nair et al. (1991) was followed for the buccal MN test. For this, separate buccal smear samples from right and left cheeks were fixed in methanol: acetic acid (3:1 ratio), hydrolysed in 1N HCl at 60ºC, stained in Aceto-orcein (2%) and counterstained in 0.1% Fast Green. Preparations were coded and scored blind. The criteria for scoring micronuclei and micronucleated cells of Tolbert et al. (1991) were followed and their presence was independently under oil immersion (100X) and by another observer. A total of 2000 cells (1000 cells per slide) were scored under a binocular microscope for chromosomal damage, cell proliferation and cell death markers as recommended for Buccal Cytome assay (Thomas et al. 2009).Chromosomal damage markers included micronucleated cells and nuclear buds, basal cells and binucleated cells were cell proliferation markers and cell death markers were observed as number of apoptotic (condensed chromatin, karyorrhectic, pyknotic) and necrotic (karyolytic) cells. The scoring method of Thomas et al. (2009) was followed viz. initially 1000 cells were scored per participant for the number of Basal, Differentiated, Binucleated (cell proliferation markers) and also a record of Condensed chromatin, Karyorrhectic, Pyknotic and Karyolytic cells (cell death markers)was made. Subsequently 2000 cells were scored for the presence of micronuclei and for nuclear buds to assess chromosomal (MNd cells) and DNA (nuclear buds) damage markers. Repair index was calculated as RI = (karyolytic + karyorrhectic) / (micronuclei + nuclear bud) (Ramirez and Saldanha 2002).

Statistical Analysis

The statistical analysis was performed using the SPSS (16 version) for windows. The Data on DNA and chromosomal damage variables are presented as mean±S.E.M. On finding normal distribution of data by Kolmogorov and Smirnov tests, statistical analysis of demographic variables and observed values of parameters for DNA and chromosomal damage within and between the sample and control groups was carried out using the Students't-test .Chi square analysis was performed on socio-demographic data to find whether the sample and the control groups matched. In order to find if there was any association between the confounding variables and indices of genetic damage, the analysis of variance (ANOVA), Pearson correlation analysis and univariate linear regression analysis were performed. The results on univariate regression analysis were further followed on multivariate linear regression analysis to confirm whether these are predictors of the observed genetic damage.

RESULTS

The general and the demographic characteristics of the study participants are presented in Tables 1 and 2. The study participants (n=75) included those residing/working in the vicinity of a base station (n=50; 28.70±1.19y) and a control group (n=25; 28.88±1.70y), all belonging to the middle socio-economic class. Those residing and working near the base station had residences/offices either facing (n =25) or besides (n =25) with respect to the antenna's position on the tower between 50-200m and staying there for 5-10y (9.82±0.10y).The power density in the area with mobile phone base station (8.82-13.22 W/m^2; 11.18±0.13 W/m^2) was significantly (p=0.000) elevated from that in the control areas without any mobile phone base station (0.01-0.10 W/m^2; 0.04±0.00 W/m^2). Mobile phone usage was from 0.5-10.0y (3.45±0.33y) with daily usage of 0.5 to 7.0h (1.74±0.21h). Among males (n=25), alcohol intake was commoner (22%) than smoking (4%), 50% were non-vegetarians and were students or grocery owners. The females (n=25) were house wives, students or teachers. Males had significantly higher average duration (4.20±0.53y; p=0.023) and daily (2.27±0.33h; p= 0.013) usage of mobile phones than the females (2.70±0.34y; 1.22±0.22h). The control group (n=25) was gender, age and socio-economic status-matched to the sample group as well as for alcohol drinking and smoking habits but not

Table 1: General characteristics of the participants residing in the vicinity of a mobile phone base station and the controls

Characteristics		*Sample group n=50(%)*	*Control group n=25 (%)*	*÷² value*	*P value*
Age(y)	18-31	33(66.00)	15(60.00)	0.065	0.7986
	32-45	17(34.00)	10(40.00)		
Gender	Females	25(50.00)	13(52.00)	0.007	0.9349
	Males	25(50.00)	12(48.00)		
Diet	Veg	25(50.00)	16(64.00)	0.814	0.3670
	Non- veg	25(50.00)	09(36.00)		
†Smoking habit	Smokers	02(04.00)	01(04.00)	0.391	0.5320
	Non-smokers	48(96.00)	24(96.00)		
††Alcohol consumption	Yes	11(22.00)	02(08.00)	1.407	0.2355
	No	39(78.00)	23(92.00)		
Mobile phone usage	Users	50(100.00)	16(64.00)	17.187	**0.0001**
	Non- users	-	9(36.00)		
Occupation	Student	18(36.00)	10(40.00)	6.871	0.550
	House wives	11(22.00)	05(20.00)		
	Clerical job	06(12.00)	01(4.00)		
	Teachers	05(10.00)	07(28.00)		
	Grocers	05(10.00)	01(04.00)		
	Cloth merchants	02(04.00)	01(04.00)		
	Advocate	01(02.00)	-		
	Staff nurse	01(02.00)	-		
	Policeman	01(02.00)	-		

†3-5 cigarettes/week, †† 100-200ml/week
p value in bold is significant (p=0.0001)

Table 2: Demographic characteristics of the participants residing in the vicinity of a mobile phone base station and the controls

Characteristics	*Sample group (Mean ± S.E.M.)*			*Control group (Mean ± S.E.M.)*		
	Females	*Males*	*Total*	*Females*	*Males*	*Total*
Age (y)	30.64±1.73	26.76±1.59	28.70±1.19	28.69±2.10	29.08±2.82	28.88±1.70
Distance from mobile phone base station (m)	137.40±9.12	144.48±7.55	140.90±5.88	-	-	-
Time since residing near base station(y)	10.00±0.00	9.64±0.19	9.82±0.10	-	-	-
Power Density (W/m^2)	11.03^{***}±0.21	11.34^{***}±0.18	11.18^{***}±0.13	0.04±0.12	0.06±0.01	0.04±0.00
Daily mobile phone usage (h)	1.22^{*}±0.22[a]	2.27^{**}±0.33[a]	1.74^{***}±0.21	0.35±0.10	0.22±0.03	0.29±0.05
Duration of mobile phone usage (y)	2.70±0.34[b]	4.38^{*}±0.53[b]	3.45^{*}±0.33	2.18±0.58	1.97±0.66	2.07±0.42
†SAR (W/kg)	0.76±0.03	0.77±0.04	0.77^{**}±0.02	0.85±0.06	0.71±0.07	0.78±0.05

Values with similar letter are significantly different (females vs males); [a] highly significant, p=0.013; [b]significant, p=0.023; *** Very highly significant in comparison to parallel control group (p=0.000); ** Highly significant in comparison to parallel control group (p=0.005); † available from: www.sarvalues.com

staying/working near base stations and with 64% using mobile phones as well as for alcohol drinking and smoking habits. Their daily usage was very significantly (p=0.001) higher in the sample group participants (1.74±0.21h) compared to that in controls (0.29±0.05h).

Table 3: DNA damage in individuals residing in the vicinity of a mobile phone base station and the controls

Study group		*Number of individuals*	*Damage frequency (Mean ± S.E.M.)*	*Damage index (Mean ± S.E.M.)*	*Mean DNA migration length (Mean± S.E.M.)*
Sample Group	Males	25	94.64*** ± 1.4	173.76***±9.06[a]	30.01*** ± 1.82
	Females	25	97.72 ***± 0.95	111.24***±4.57[a]	34.76*** ± 2.48
	Total	50	96.18*** ± 0.87	142.50***±6.72	32.38 ***± 1.56
Control Group					
Males	Mobile phone user	08	39.25 ± 3.67	40.75±4.28	16.52 ± 1.46
	Non Mobile phone user	04	35.00 ± 5.08	36.25±5.20	15.36 ± 0.81
	Total	12	37.83 ± 2.9	39.25±3.26	16.13 ± 0.99
Females	Mobile phone user	08	38.37 ± 6.27	38.37±6.27	17.71 ± 1.55
	Non Mobile phone user	05	42.80 ± 6.22	44.00±5.22	18.63 ± 1.37
	Total	13	40.08 ± 4.41	40.07±4.41	18.06 ± 1.06
Total	Mobile phone user	16	38.81 ± 3.51	39.56±3.68	17.11 ± 1.04
	Non Mobile phone user	09	39.33 ± 3.51	39.88±4.07	17.18 ± 0.98
	Total	25	39.00 ± 2.64	39.68±2.72	17.14 ± 0.74

Values with similar letters are significantly different (females *vs.* males); [a] highly significant, p=0.000; *** Very highly significant in comparison to parallel control group (p=0.000)

The results of the SCGE assay for DF, DI and the mean DNA migration length in sample group were highly significant (p=0.000) from the control values (Table 3). DF was 2.46 fold higher, DI was 3.59 fold higher and DNA migration length was 1.88 fold elevated than in controls. On stratification by gender DI was significantly (p=0.000) elevated in males compared to the females. Damage frequency and DNA migration length levels were higher in females but did not reach significant levels. Similarly on comparing the genetic damage indices between those residing and those going away to work 49 hours on week days but staying near the base station no statistical differences were observed.

The results of the buccal micronucleus cytome assay (Table 4) revealed significantly elevated frequencies of micronucleated cells (3.76 fold, p=0.000), nuclear buds (5.33 fold; p=0.000), basal cells (1.18fold; p = 0.01) and pyknotic cells (2.41fold; p=0.000) in the sample group individuals compared to the control group. The repair index on the other hand was significantly (p=0.01) increased in the controls (38.92±5.82) compared to the sample group (36.72±0.87). Among the sample group participants gender differences were observed for nuclear buds and repair index. Frequency of nuclear buds was significantly (p=0.002) elevated in males in comparison to females though the repair index was significantly (p=0.006) higher in females than in the males implying that there was more inhibition of cell-proliferation in males. In the control group similarly nuclear buds (p=0.034) and basal cells (p=0.025) were higher in males. In controls, mobile phone usage did not affect genetic damage as assessed in the SCGE (Table 3) and in the buccal MN cytome (Table 4) assays.

Predictors of DF were diet (p=0.011), mobile phone usage (p=0.026), phone set SAR value (p=0.044); of DI were age (p=0.051), diet (p=0.000) and alcohol drinking (p=0.020) whereas mean DNA migration length was influenced only by the location (p=0.012) of residences near base station (Table 5). The frequency of nuclear buds was significantly affected by alcohol drinking (p=0.005) and phone set SAR values (p=0.007); age influenced frequency of basal cells (p=0.056); location of residences (p=0.001) was associated with karyorrhectic cell frequency and the frequency of condensed chromatin cells was associated with distance (p=0.011) of residences from base station. Repair index was associated with diet(p=0.000),alcohol drinking (p=0.013),duration of mobile phone usage(p=0.036) and the phone set SAR value (p=0.026).The multivariate linear regression analysis further revealed that location of residences from base station emerged as significant predictor of damage frequency (p=0.024); age (p=0.044) and diet (p=0.011)of damage index and location of residences (p=0.004) with of mean DNA migration length. Phone set SAR value (p=0.012), location (p=0.002), distance (p=0.039) and diet (p=0.030)

Table 4: The buccal MN cytome assay in individuals residing in the vicinity of a mobile phone base station and the controls Group

Study group	*Gender*	*n*	*DNA damage markers*		*Cell proliferation markers*		*Cell death markers*		*Repair*		*Index*
			Micronucleated cells (‰)	*Nuclear buds (‰)*	*Basal cells (‰)*	*Binucleated cells (‰)*	*Karyorrhectic cells (‰)*	*Condensed chromatin cells (‰)*	*Karyolytic cells (‰)*	*Pyknotic cells (‰)*	
Sample Group	Males	25	$0.50^{***}\pm0.11$	$0.60^{***}\pm0.04^{a}$	3.21 ± 0.16	0.86 ± 0.05	2.15 ± 0.15	1.56 ± 0.14	7.48 ± 0.35	0.24 ± 0.03	12.64 ± 1.64^{b}
	Females	25	$0.48^{***}\pm0.07$	$0.37^{***}\pm0.05^{a}$	2.93 ± 0.18	0.71 ± 0.05	2.49 ± 0.16	1.66 ± 0.19	7.46 ± 0.36	0.34 ± 0.04	$60.79^{**}\pm16.08^{b}$
	Total	50	$0.49^{***}\pm0.06$	$0.48^{***}\pm0.03$	$3.07^{**}\pm0.12$	0.78 ± 0.03	2.32 ± 0.11	1.61 ± 0.11	7.47 ± 0.25	$0.29^{***}\pm0.03$	36.72 ± 0.87
Control Group											
Mobile Phone Users	Males	08	0.17 ± 0.05	0.11 ± 0.02	2.80 ± 0.17	0.70 ± 0.07	2.41 ± 0.23	1.75 ± 0.38	6.53 ± 1.07	0.12 ± 0.02	35.89 ± 3.82
	Females	08	0.15 ± 0.04	0.06 ± 0.01	2.38 ± 0.19	0.63 ± 0.04	2.18 ± 0.24	2.16 ± 0.04	7.43 ± 0.69	0.15 ± 0.03	30.69 ± 11.06
	Total	16	0.16 ± 0.034	0.08 ± 0.01	2.59 ± 0.13	0.66 ± 0.04	2.30 ± 0.16	1.95 ± 0.28	6.98 ± 0.62	0.13 ± 0.02	33.29 ± 5.69
Non Mobile Phone Users	Males	04	0.10 ± 0.04	0.15 ± 0.02	2.92 ± 0.21	0.75 ± 0.13	2.90 ± 0.26	0.25 ± 0.25^{e}	5.27 ± 1.05	0.10 ± 0.04	35.37 ± 8.43
	Females	05	0.08 ± 0.02	0.06 ± 0.04	2.26 ± 0.29	0.72 ± 0.10	2.22 ± 0.23	$1.80^{*}\pm0.55^{e}$	7.34 ± 0.93	0.10 ± 0.05	59.76 ± 21.17
	Total	09	0.08 ± 0.02	0.10 ± 0.02	2.55 ± 0.21	0.73 ± 0.07	2.52 ± 0.20	1.11 ± 0.41	6.47 ± 0.74	0.10 ± 0.03	48.92 ± 12.44
Total	Males	12	0.15 ± 0.03	$0.12^{*}\pm0.02^{c}$	$2.84^{*}\pm0.13^{d}$	0.71 ± 0.06	2.57 ± 0.18	1.25 ± 0.33	6.11 ± 0.78	0.11 ± 0.02	35.71 ± 3.55
	Females	13	0.12 ± 0.03	0.06 ± 0.01^{c}	2.33 ± 0.15^{d}	0.66 ± 0.04	2.20 ± 0.16	2.02 ± 0.33	7.40 ± 0.53	0.13 ± 0.02	41.87 ± 10.86
	Total	25	0.13 ± 0.02	0.09 ± 0.01	2.58 ± 0.11	0.69 ± 0.03	2.38 ± 0.12	1.65 ± 0.24	6.78 ± 0.47	0.12 ± 0.01	$38.92^{**}\pm5.82$

Values with similar letters are significantly different (females *vs* males); [a] highly significant, p=0.002; [b] highly significant, p=0.006; [c] significant (p=0.034); [d] significant (p=0.025); e significant (p=0.047) [***] Very highly significant in comparison to parallel control group (p=0.000);[**] significant in comparison to parallel group (p=0.004)

Table 5: Predictors of genetic damage in sample and the control individuals

Study Group	*Predictors*	*Source of variation*	*ANOVA*			*Correlation*		*Univariate linear regression*			*Multivariate linear regression*		
			F (variance-ratio)	*Mean sum of squares*	*P value*	*r*	*P value*	*B value (95%CI)*	*t value*	*P value*	*B value (95%CI)*	*t value*	*P value*
Sample Group	*Damage Frequency*	Diet	7.058	233.280 33.050	0.011	-0.358	0.011	-0.358 (-7.589- -1.051)	-2.657	0.011	-0.301 (-7.400- 0.130)	-1.953	0.058
		Daily mobile phone usage	5.273	180.106 34.158	0.026	-0.315	0.026 -0.157)	-0.315 (-2.366-	-2.296	0.026 -0.162)	-0.274 (-2.364	-1.763	0.086
		SAR	4.293	149.386 34.798	0.044	-0.287	0.044	-0.287 (-17.020-	-2.072	0.044 -0.256)	-0.142 (-12.870	-1.012 4.286)	0.318
		Location	2.414 36.095	87.120	0.127	0.219	0.127 (-0.777- 6.057)	0.219	1.554	0.127 (0.523 -7.159)	0.318	2.342	0.024
	Damage Index	Age	4.006	8606.058 2148.084	0.051	-0.278	0.051	-0.278 (-3.136- 0.007)	-2.002	0.051	-0.284 (-3.153- -0.045)	-2.082	0.044
		Diet	14.188	25900.880 1787.775	0.000	0.482	0.000	0.482 (21.474- 69.566)	3.806	0.000	0.398 (9.271 -65.913)	2.685	0.011
		Alcohol	5.858	12090.388 2075.494	0.020	-0.329	0.020	-0.329 (-68.810- -6.267)	-2.414	0.020	-0.234 (-60.894 -7.485)	-1.580	0.122
	Mean DNA Migration Length	Location	6.836	634.392 92.802	0.012	0.353	0.012	0.353 (1.646- 12.602)	2.615	0.012	0.402 (2.112- 14.118)	2.734	0.004
		Nuclear Bud Alcohol	8.490	0.5140.061	0.005	-0.388	0.005	-0.388 (-0.414- -0.076)	-2.914	0.005	-0.314) (-0.403 -0.006)	-1.963	0.057
		SAR	7.849	0.481 0.061	0.007	0.375	0.007	0.375 (0.138- 0.84)	2.802	0.007	0.386 (0.119 -0.890)	2.644	0.012
	Basal Cells	Age	3.835	2.461 0.642	0.056	0.272	0.056	0.272 (0.000- 0.054)	1.958	0.056	0.222 (-0.011- 0.054)	1.335	0.190
	Karyorrhectic Cells	Location	11.707	5.917 0.505	0.001	-0.443	0.001	-0.443 -(-1.092- -0.284)	-3.422	0.001	-0.468 (-1.169- -0.284)	-3.321	0.002
	Condensed Chromatin Cells	Distance	7.021	4.407 0.628	0.011	-0.357	0.011	-0.357 (-0.013- -0.002)	-2.650	0.011	-0.324 (-0.013- -0.000)	-2.139	0.039
	Repair Index	Diet	16.681	6186.723 370.882	0.000	-0.508	0.000	-0.508 (-33.199- -11.295)	-4.084	0.000	-0.340 (-28.234- -1.550)	-2.258	0.030

Table 5: Contd...

Study Group	*Predictors*	*Source of variation*	*ANOVA*			*Correlation*		*Univariate linear regression*			*Multivariate linear regression*		
			F(variance-ratio)	*Mean sum of squares*	*P value*	*r*	*P value*	*B value (95%CI)*	*t value*	*P value*	*B value (95%CI)*	*t value*	*P value*
		Alcohol	6.713	2943.363 438.452	0.013	0.350	0.013	0.350 (4.148- 32.895)	2.591	0.013	0.181 (-6.544 -25.669)	1.201	0.237
		Duration of mobile phone	4.634	2112.249 455.767	0.036	-0.297	0.036	-0.297 (-5.383- -0.184)	-2.157	0.036	0.023 (-4.135- 4.817)	0.154	0.878
		SAR	5.309	2388.939 450.003	0.026	- 0.316	0.026	-0.316 (-64.686- -4.399)	-2.304	0.026	-0.206 (-52.895 -7.899)	-1.497	0.142
Control Group	*Basal Cells*	Duration of mobile phone usage	7.134	0.978 0.137	0.023	- 0.645	0.023	-0.645 (-0.033- -0.003)	-2.671	0.023	-0.794 (-0.057- -0.012	-1.784	0.149
	Condensed Chromatin Cells	SAR	5.239	1.701 0.325	0.045	-0.586	0.045	-0.586 (-2.008 - -0.027)	-2.289	0.045	-1.203 (-5.626- -1.450)	-1.638	0.177
	Repair Index	Duration of mobile phone usage	24.388	8233.627 337.60	0.001	0.842	0.001	0.842 (0.908- -2.400)	4.938	0.001	0.944 (0.931 -2.776)	5.577	0.005

p values in bold are significant ($p<0.05$)

were significant predictors of frequencies of nuclear buds, karyorrhectic, condensed chromatin cells and repair index, respectively.

DISCUSSION

The results of the single cell gel electrophoresis assay and the buccal MN Cytome assay have revealed increased levels of DNA and chromosomal in those in the vicinity of mobile phone base station in comparison to the control group. The significantly elevated frequency of Micronucleated cells and greater percentage of damaged cells along with the greater DNA migration lengths undoubtedly imply genomic damage in two different tissues- buccal epithelium and peripheral blood leukocytes. The observed micronucleated cell frequency in buccal epithelial cells is the result of genetic damage in these cells which has persisted despite repair during the preceeding cell division in the buccal epithelial cells (Fenech 2002). The comet assay detects alkali-labile sites, single- or double-strand DNA breaks that can be repaired (Collins 2004). The significantly higher genomic damage in healthy individuals with no exposure history (past or present) except for their place of stay/office near mobile phone base station with power density levels (11.18±0.13 W/m^2) significantly higher ($p=0.000$) than from where control group was sampled (0.04±0.00 W/m^2) imply that differences in RFR are in all probability responsible for the individual genetic damage.

A perusual of literature has also revealed that microwave radiation interfered with the recombining of DNA leading to more double-strand breaks (Ruiz-Gómez and Martínez-Morillo 2009), micronucleus induction (Gustavino et al. 2014) disruption of blood brain barrier (Nittby et al. 2009), interfering with normal sleep patterns and increase heat-shock protein levels on repeated exposures and cause inflammatory response (Kesari et al. 2013). RF exposure induced genotoxicity consistently, and specifically caused chromosomal instability (Maschevich et al. 2003), altered gene expression (Zhang et al. 2008), gene mutations (Koyama et al. 2007), DNA fragmentation and DNA structural breaks (Lai and Singh 2004). DNA integrity in male CD1 Swiss mice germline (Aitken et al. 2005) was affected by the radio frequency electromagnetic radiation. Oxidative stress in terms of increased malondialdehyde and oxidative DNA damage as 8-hydroxy-2'-deoxyguanosine levels in in the brain tissue of pregnant and non-pregnant New Zealand White rabbits (Guler et al. 2010) and of 8-hydroxyguanine in the mitochondria of primary cultured neurons (Xu et al. 2010) have also been reported to be affected by the radiofrequency radiation.

Contrary to these reports are documentation on non-thermal nature of these RFR (Gaestel2010) and that the energy of non-ionizing EMF is not sufficient to break chemical bonds directly (Phillips et al. 2009).However the RFR can act by indirect mechanisms resulting consequently in the production of free radicals (Phillips et al. 2009) which are very potent and cytotoxic molecules (Lai and Singh 2004). Pulse-modulated RF(900 MHz, specific absorption rate (SAR) level of 1.20 W/kg 20 min/day for three weeks) radiation caused oxidative injury in liver, lung, testis and heart tissues mediated by lipid peroxidation, increased level of nitric oxide and suppression of antioxidant (glutathione) defense in male Wistar albino rats (Esmekaya et al. 2011). A reduced percentage of motile sperm in rats from RF-EMR(0.9/1.8 GHz GSM mobile phone for 1 hour continuously per day for 28 days) exposure and a significant increase in lipid peroxidation and low GSH content in the testis and epididymis (Mailankot et al. 2009) as well as significant increase in brain lipid and protein oxidation from 900-MHz EMF at a whole body average specific absorption rate (SAR) of 1.08 W/kg for 1 h/day for 3 weeks (Bilgici et al. 2013) have also been observed. Free radicals have pleiotropic effects varying from inducing mutagenic responses depending upon concentration and duration of exposure and cellular tissue type (Wolf et al. 2005).

DNA damage observed in the present study assessed by the SCGE assay includes single- and double-DNA strand breaks and alkali-labile sites (Singh et al. 1988; Olive and Banath 1990). The DNA double-strand breaks are very critical and are usually lethal (Pfeiffer et al. 2000). A statistically significant increase in single-strand and/or double-strand DNA breaks (DNA damage) was reported in lymphocytes of cell phone users (Gandhi and Anita 2005; Rekhadevi et al. 2009; El-Abd and Eltoweissy 2012); in human lens epithelial cells (Yao et al. 2008), in renal and liver cells of rat exposed to 915 MHz (GSM) with power density of 2.4 W/m2, SAR of 0.6 W/kg for one hour/day, seven days/week during two

weeks period (Trošic et al. 2011) and in brain cells of Fischer rats after 30 days of exposure with three different frequencies of microwaves, that is, 900 MHz, 1800 MHz and 2450 MHz and at a whole body SAR value of 5.953×10^{-4} W/kg, 5.835×10^{-4} W/kg, and 6.672×10^{-4} W/kg (Deshmukh et al. 2013). Unrepaired DNA breaks can induce permanent cell-cycle arrest, apoptosis or mitotic cell death from loss of genomic material (Rothkamm et al. 2003; Gustafsson et al. 2014) while incorrect repair can trigger carcinogenesis through translocations, inversions or deletions of genomic material (Hoeijmakers 2001; van Gent 2001). On basis of these facts, there has been equivocal evidence on the non-genotoxic nature of RFR *in vitro* even at 18.0 GHz , 16.5 GHz and GSM-900 (Hansteen et al. 2009; Bourthoumieu et al. 2011; Lai 2014) and *in vivo* (Maes et al. 2006; Lai 2014) and a lack of association with genotoxicity as well as carcinogensis (Vijayalaxmi and Obe 2004; Frei et al. 2011). DNA strand breaks have been correlated with cell death (Schindowski et al. 2000), aging (Katyal and McKinnon 2008) and cancer (Khanna and Jackson 2001), therefore by implication, the participants of the present study may also be susceptible to these effects.

The significantly increased MN frequency and cytotoxicity observed in the buccal epithelial cells also reflect genetic damage in the study participants. The buccal Cytome assay provides a measure of DNA and chromosomal damage as well as of cytotoxicity and cell damage. The basal layer of the oral epithelium contains stem cells that may express genetic damage (chromosome breakage or loss) as micronuclei during nuclear division with some cells differentiating and exfoliating into cells with condensed chromatin, fragmented nuclei (karyorrhectic cells), shrunken nuclei (pyknotic cells), with no nuclear material (karyolitic/ ghost cells) or as nuclear buds which biomarkers of gene amplification; and in this manner are important measures of cytotoxicity and cytostatic effects (Kashyap and Reddy 2012). Exposures may inhibit or enhance basal cell proliferation and also affect micronuclei expression (Holland et al. 2008).The importance of such a cytogenetic assay is its important role in toxicological hazard evaluation as a first step towards quantification of cancers (Ghosh et al. 2008).Besides damage via oxidative stress which in turn can cause cancer stimulation (Yakymenko et al. 2011), ROS in cells act as a secondary messenger for certain intra-cellular signaling cascades which can induce oncogenic transformation (Storz 2005).

CONCLUSION

The long-term exposure to low intensity electromagnetic microwaves as emitted continuously by mobile phone base station may provoke ill-health effects which may further lead to cancer development.

RECOMMENDATIONS

The recommendations and safety limits set by many regulatory bodies for technical devices emitting microwave radiations need to be re-assessed and additional studies for unprejudiced risk assessment be carried out.

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