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Phenotypic and genotypic characterization of antioxidant enzyme system in human population exposed to radiation from mobile towers

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Abstract In the present era, cellular phones have changed the life style of human beings completely and have become an essential part of their lives. The number of cell phones and cell towers are increasing in spite of their disadvantages. These cell towers transmit radiation continuously without any interruption, so people living within 100s of meters from the tower receive 10,000 to 10,000,000 times stronger signal than required for mobile communication. In the present study, we have examined superoxide dismutase (SOD) enzyme activity, catalase (CAT) enzyme activity, lipid peroxidation assay, and effect of functional polymorphism of SOD and CAT antioxidant genes against mobile tower-induced oxidative stress in human population. From our results, we have found a significantly lower mean value of manganese superoxide dismutase (MnSOD) enzyme activity, catalase (CAT) enzyme activity, and a high value of lipid peroxidation assay in exposed as compared to control subjects. Polymorphisms in antioxidant MnSOD and CAT genes significantly contributed to its phenotype. In the current study, a significant association of genetic polymorphism of antioxidant genes with genetic damage has been observed in human population exposed to radiations emitted from mobile towers.

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Keywords Antioxidant gene polymorphism · Electromagnetic fields (EMFs) · Mobile tower base stations · Oxidative stress · Reactive oxygen species

Introduction

During the last more than two decades, non ionizing electromagnetic radiation has increased its approach in several areas like telecommunication, medical, industrial and domestic heating, lighting, etc. Thus, the subject of the impact of non-ionizing radiation on living cells, tissues, and live animals including human beings has also evoked much interest in recent times. In the present era, India has about 800 million users of mobiles and mobile handsets that are connected to around 375,000 towers. The majority of these towers are mounted near the residential and office buildings to provide good mobile phone coverage to the users. The numbers of cell phones and cell towers are increasing without heeding to their disadvantages. There is a big question on the associated health impacts on human beings due to radiation from mobile phones and mobile towers all over the world. In India, millions of people reside within these high-radiation zones.

Biological mechanisms that can explain the link between exposure to radiofrequency energy (RF) and possible harmful effects are still lacking. One of the proposed mechanisms is the stimulation of oxidative stress (excess formation of free radicals generating oxidative DNA damage) [1, 2]. Reactive oxygen species (ROS) are free radicals that are derived from oxygen metabolism. They are an important part of the defense mechanism against infections, but excessive generation of free oxygen radicals may damage tissues [3, 4]. ROS plays an important role in the aging process as well as in a number of human

diseases, including cancer, ischemia, and failures in immunity and endocrine functions. Recent evidence suggests that ROS also plays an important role in cell death and signal transduction through non-ionizing radiations [5–7]. Different ROS products can cause peroxidation of membrane lipids and attack proteins or DNA [8, 9]. When oxidative stress arises as a consequence of a pathologic event, there are various defense mechanisms existing in the human body to safeguard against the accumulation of ROS by regulation and expression of several non-enzymatic and enzymatic antioxidant activities. In oxidative stress condition, lipid peroxidation level increases and antioxidant protection decreases which generate epoxides. These epoxides may spontaneously react with nucleophilic centers in the cell and thereby covalently bind to DNA, RNA, and protein and lead to cytotoxicity, allergy, mutagenicity, and/or carcinogenicity, depending on the properties of the epoxide in question [10]. Lipid peroxidation can be evaluated by the thiobarbituric acid-reactive substances (TBARS) method for malondialdehyde, the last product of lipid breakdown caused by oxidative stress [11–13]. The enzymatic and non-enzymatic antioxidant defenses include superoxide dismutase (SOD), glutathione peroxidase (GPX), catalase (CAT), ascorbic acid (vitamin C), α-tocopherol (vitamin E), glutathione (GSH), β-carotene, and vitamin A, which can be evaluated using easy photometric assays [14, 15]. There is a balance between both the activities and the intracellular levels of these antioxidants that is essential for the survival of organisms and their health [16, 17]. Superoxide dismutase (Enzyme nomenclature EC 1.15.1.1) is the antioxidant enzyme that catalyzes dismutation of the highly reactive superoxide anion to O_2 and to the less reactive H_2O_2. Peroxide can be destroyed by CAT- or GPX-catalyzed reactions [18, 19]. Catalase (Enzyme nomenclature EC 1.11.1.6), a heme enzyme, is present in the peroxisomes found in most eukaryotic organisms as a dumbbell-shaped tetramer of four identical subunits. CAT plays a key role in antioxidant defense to maintain the redox balance in human body preventing oxidative stress. In the present study, we have examined the activities of superoxide dismutase (SOD) and catalase and lipid peroxidation assay as biomarkers of the effect resulting from non-ionizing radiation-induced oxidative stress in human population.

Activity levels of these enzymes are likely affected by functional polymorphisms in the genes encoding them. The alanine allele of *MnSOD* Val9Ala polymorphism has been associated with overexpression of MnSOD, resulting in an increased production of H_2O_2, which has been proposed to generate increased levels of ROS, if not subsequently neutralized [20]. CAT participates in defense mechanisms against oxidative stress by controlling the intracellular concentration of H_2O_2 by conversion into H_2O and O_2 [21]. The variant T allele of *CAT* C262T polymorphism has been associated with lower enzyme activity compared to the C allele and thus increased levels of ROS [21]. The aim of the present study is to determine phenotypic and genotypic characterization of this antioxidant enzyme system in human population exposed to radiations from mobile towers.

Materials and methods

Subjects and sampling

The study population consisted of 116 individuals residing near mobile towers and 106 healthy control volunteers who were residing far away (more than 800 m) from the mobile towers. As a part of a routine health examination, each subject from both groups was interviewed by a health physician. A structured questionnaire was filled to gather information about previous exposure to diagnostic or therapeutic radiations and medical use of therapeutic drugs. The demographic data for the housewives also included the use of house appliances like microwave, gauzier, and induction vessels. The control group had no exposure history from any source of radiations and the subjects were selected where there were no mobile towers or any other sources of radiation up to 800 m radius. None of the individuals from the control group underwent diagnostic X-ray examinations or received any therapeutic irradiation or chemotherapeutic drugs and used any type of house appliances like microwave in the past 12 months before the start of the study.

Informed consent was obtained from all subjects. The research protocol was approved by the Institutional Human Ethics Committee (IHEC) of Kurukshetra University, Kurukshetra (Haryana). Demographic data of all subjects including age, gender, diet, mobile usage, exposure duration, and consumption habits (smoking, tobacco consumption, drinking habits, etc.) were collected through a standard questionnaire.

Exclusion criteria for participation in the study included recent treatment with mutagenic agents (such as X-ray, radiography), chronic conditions (such as autoimmune diseases), and recent acute infections up to 3 months before sampling that required medication such as antibiotics or any type of vaccination.

Blood sampling

Blood samples (about 2 mL) were collected by venipuncture from each subject exposed to radiations from mobile towers and control subjects and stored in disposable presterilized potassium EDTA-coated vacutainer tubes with

the help of a trained technician. The blood samples were brought to the laboratory in a well-insulated ice box with care to avoid freezing of samples. Total serum was isolated from whole blood and assays were performed immediately.

Estimation of superoxide dismutase activity (Enzyme nomenclature E.C.1.15.1.1)

Superoxide dismutase was estimated by the method of Marklund et al. [22]. Briefly, the assay mixture contained 750 µL of blood serum, 2 mL of 0.1 M Tris HCl buffer (pH 8.2), and 750 µL of distilled water. The air-equilibrated assay mixture was then mixed with 0.5 mL of 2 mM pyrogallol. The rate of inhibition of pyrogallol autoxidation after the addition of the enzyme into plasma was measured using a Systronics spectrophotometer. The amount of enzyme required to achieve 50% inhibition of pyrogallol autoxidation was considered as one unit of enzyme activity. The enzyme activity was expressed as Units/mg protein.

Estimation of catalase activity (Enzyme nomenclature E.C.1.11.1.6)

Catalase was assayed by the method of Aebi [23]. Briefly, the assay mixture contained 2.25 mL phosphate buffer, 0.65 mL hydrogen peroxide, and 0.1 mL enzyme source. The decrease in absorbance was measured immediately at 240 nm, against a blank containing all the components except the enzyme at 10-s interval for 3 min using a spectrophotometer (Systronics).

Lipid peroxidation

Lipid peroxidation was measured by the method of Beauge and Aust [24]. Malondialdehyde (MDA), the byproduct of lipid peroxidation, forms adducts (a product of a direct addition of two or more distinct molecules, resulting in a single reaction product containing all atoms of all components; the resultant is considered a distinct molecular species) with TBA on boiling and produces a pink-colored complex, which absorbs maximally at 532 nm.

Genotyping of antioxidant genes

MnSOD genotyping

The standard PCR technique was modified by Rahman et al. [25] to include 10 pmol of each of the MnSOD primers, forward primer 5′-AGCACCAGCAGGCAGCTGG CTCCAG-3′ and reverse primer 5′-GAAGCGAGTTCTC-CACGGAGA-3′, with an annealing temperature of 67 °C. The PCR product (308 bp) was digested overnight with *Alu*I at 37 °C and separated on a 3% agarose gel to reveal the generated restriction fragments for the wild-type V allele (185 and 123 bp) and mutant A allele (185, 98, and 25 bp).

Catalase genotyping

A modified protocol of Rahman et al. [25] was incorporated with primers as forward primer 5′-AATCA-GAAGGCAGTCCTCCC-3′ and reverse primer 5′-TCGGGGAGCACAGAGTGTAC-3′, with an annealing temperature of 70 °C. The 185-bp PCR product was digested with *Sma*I for 4 h and analyzed on a 2% agarose gel. The T (−262) variant yields an undigested product of 185 bp, relative to the digested C (−262) variant, which was visualized as a 155-bp fragment.

Statistical analysis

The Student's *t* test was used for comparison of different variables between the studied groups. Distribution of MnSOD and catalase genotyping among the study population was determined by Chi-square test. The influence of genetic polymorphisms of antioxidant genes and confounding factors on the studied biomarkers among multiple subgroups was determined by post hoc analysis and Tukey's HSD test using Multivariate Analysis of Variance (MANOVA). All tests were performed using statistical software system SPSS 16.0. The level of significance was set at 0.05.

Results

Demographic characteristics

The demographic characteristics of the studied subjects are summarized in Table 1. No significant difference was found in the demographic characteristics of 116 exposed individuals and 106 unexposed control subjects.

Mobile tower exposure level and antioxidant enzyme activity

Radiofrequency electromagnetic radiation (RF-EMR) in terms of power density was measured from different mobile towers at different locations of Kurukshetra region in North India. Most of the measured values were in the range of 0.037–12.20 mW/m^2 [26]. In our study, we found significantly lower mean values of MnSOD activity (3.22 ± 1.28) and catalase activity (10.11 ± 11.01) and a higher mean value of lipid peroxidation assay (4.96 ± 2.43) in the exposed subjects when compared to the mean values of MnSOD activity (6.11 ± 1.91),

Table 1 Demographic characteristics of the control and exposed groups

Variables	Control	Exposed	*P* value
All *N* (%)	106 (100.0)	116 (100.0)	
Age in years (Mean ± SD)	33.79 ± 14.46	32.87 ± 12.95	0.446
Age			
<25	48 (45.3)	43 (37.1)	0.147
25–45	32 (30.2)	55 (47.4)	
>45	26 (24.5)	18 (15.5)	
Gender			
Male	63 (59.4)	68 (58.6)	0.086
Female	43 (40.6)	48 (41.4)	
Diet			
Veg	61 (57.5)	78 (67.2)	0.208
Nonveg	45 (42.5)	38 (32.8)	
Smoking			
Non-smokers	90 (84.9)	86 (74.1)	0.409
Smokers	16 (15.1)	30 (25.9)	
Alcohol users			
Non-alcoholic	77 (72.6)	85 (73.3)	0.530
Alcoholic	29 (27.4)	31 (26.7)	
Exposure duration			
≤8 years	–	93 (80.17)	–
≥9 years	–	23 (19.83)	
Mobile usage (in hours)			
(Mean ± SD)	1.43 ± 0.87	1.79 ± 1.14	0.977

Student's *t* test was applied for comparing the mean value of age and mobile usage between the control and exposed groups. Chi-square test was applied to test for differences in age, gender, diet, and consumption habits among the study population

Table 2 SOD, catalase activity, and lipid peroxidation assay in the exposed and control groups

	Mean ± SD	*P* value
SOD (Units/mg protein)	6.11 ± 1.91	<0.05
Control	3.22 ± 1.28	
Exposed		
Catalase (mUnits/mg Protein)	25.52 ± 3.63	<0.05
Control	10.11 ± 11.02	
Exposed		
Lipid peroxidation assay (nmoles/mg protein)	1.53 ± 0.25	<0.05
Control	4.96 ± 2.43	
Exposed		

Student's *t* test was applied for comparing mean values between the control and exposed groups

catalase activity (25.52 ± 3.63), and lipid peroxidation assay (1.53 ± 0.25) in control subjects (Table 2; Fig. 1).

Antioxidant enzyme activity at different distances from mobile towers is shown in Table 3. Minimum antioxidant enzyme activity and maximum lipid peroxidation assay were found near 150 m distances (2.70 ± 1.90 for MnSOD activity, 9.36 ± 3.90 for catalase activity, and 6.13 ± 2.20 for lipid peroxidation assay). Maximum damage was found near 150 m distances as compared to shorter distances because of the angular coverage of 120° in the horizontal direction.

Effect of age, gender, consumption habits, and exposure duration on antioxidant enzymes

Age, diet, and consumption habits such as smoking and alcohol intake did not significantly affect MnSOD activity, catalase activity, and lipid peroxidation assay in both

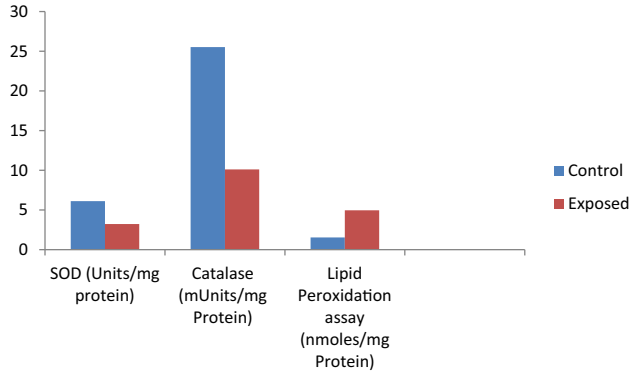

Fig. 1 SOD, catalase activity, and lipid peroxidation assay in the exposed and control groups

exposed and control population, though the values were found to be low for MnSOD activity and catalase activity and high for lipid peroxidation assay in smokers and alcoholics in the exposed group. However, we found a significant decrease in MnSOD activity and catalase activity and a significant increase in lipid peroxidation assay in females of the exposed group. Exposure duration has not much significant effects (Table 4). To evaluate the damage resulting from exposure to radiations from mobile towers, maximum samples were collected at 100 and 150 m distance range from towers. Because of angular coverage of 120° in the horizontal direction, maximum radiations were found at these distances.

Association between antioxidant genotype and antioxidant enzyme activity

In our study, MnSOD activity was found to be significantly higher in the mutant group as compared to the wild-type group of MnSOD gene, but in case of catalase its activity was found to be significantly lower in the mutant group as compared to the wild-type group. Significantly higher activity of lipid peroxidation assay was found in the mutant group as compared to the wild-type group (Table 5). Results of linear regression analysis adjusted for demographic characteristics such as age, gender, consumption habit, exposure level, and duration are shown in Table 6.

Discussion

With the introduction of new systems like portable cellular phones, towers, and various implants, issues related to direct and indirect radiation hazards are being critically studied with interest and inquiry. It is now recognized that studies on the effect of non-ionizing electromagnetic fields on human body help in explaining some of the important unresolved biological issues. In the lower frequency side, impacts of magnetic fields on the human blood lymphocytes have been established.

In our study, we have one case of cancer out of 115 individuals exposed to radiations for more than 9 years. We are not able to provide any conclusion due to statistically insignificant data, although Wolf et al. [27] indicated an association between increased incidence of cancer and living in proximity to a cellphone station in the studies carried out in Israel. To our knowledge, only a few studies have been reported, concerning the damage induced by mobile tower radiation on humans. Therefore, our results can serve as a reference for future studies.

Kumar et al. [28] recorded the signs of tumor promotion at various frequencies by indicating alterations in antioxidant enzymes, micronuclei, histone kinase, and cell cycle

Table 3 SOD, catalase activity, and lipid peroxidation assay by distance from towers and exposure duration

Distance in meters	*N*	SOD activity (Mean ± SD)	Catalase activity (Mean ± SD)	Lipid peroxidation assay (Mean ± SD)
50	26	4.13 ± 2.14	10.83 ± 4.21	4.08 ± 2.54
100	41	3.38 ± 1.40	9.93 ± 3.69	5.41 ± 2.33
150	31	2.70 ± 1.90[a]*	9.36 ± 3.9[b]*	6.13 ± 2.20[c]*
250	10	4.10 ± 1.88	10.70 ± 4.78	3.64 ± 1.26
400	8	4.75 ± 1.62	10.48 ± 4.12	2.82 ± 1.34
Exposure in years				
<8 years	93	3.23 ± 1.99	10.61 ± 10.25	4.89 ± 2.45
>9 years	23	2.92 ± 1.47	10.20 ± 13.95	5.25 ± 2.38

* Significant at $P < 0.05$, multivariate ANOVA test was used with post hoc analysis for the comparison of SOD activity, catalase activity, and lipid peroxidation assay in multiple subgroups between the exposed and control groups separately

[a] $P = 0.035$, Tukey's HSD test with 50-m subgroup as the reference

[b] $P = 0.027$, Tukey's HSD test with 50-m subgroup as the reference

[c] $P = 0.001$, Tukey's HSD test with 50-m subgroup as the reference

Table 4 SOD, catalase activity, and lipid peroxidation assay by age, gender, diet, smoking, and alcohol intake

Variables	Control group				Exposed group			
	N	SOD activity (Mean ± SD)	Catalase activity (Mean ± SD)	Lipid peroxidation assay (Mean ± SD)	*N*	SOD activity (Mean ± SD)	Catalase activity (Mean ± SD)	Lipid peroxidation assay (Mean ± SD)
Age								
<25	48	6.14 ± 1.43	25.32 ± 11.20	1.52 ± 0.24	43	3.21 ± 1.08	10.05 ± 3.65	5.00 ± 2.40
25–45	32	6.48 ± 2.01	26.96 ± 11.47	1.52 ± 0.26	55	3.35 ± 1.47	10.31 ± 3.47	4.83 ± 2.42
>45	26	5.81 ± 1.94	24.47 ± 10.67	1.57 ± 0.27	18	3.04 ± 1.14	9.98 ± 3.89	5.11 ± 2.51
Gender								
Male	63	5.90 ± 1.85	28.48 ± 10.84	1.51 ± 0.25	68	3.98 ± 1.16	10.96 ± 3.79	4.51 ± 2.29
Female	43	5.76 ± 1.89	28.44 ± 10.73	1.54 ± 0.26	48	3.19 ± 1.37*	10.08 ± 3.54*	5.59 ± 2.51*
Diet								
Veg	61	5.92 ± 2.04	25.11 ± 11.14	1.54 ± 0.25	78	3.06 ± 1.25	9.52 ± 3.72	5.07 ± 2.48
Nonveg	45	6.20 ± 1.85	25.37 ± 10.86	1.51 ± 0.26	38	3.34 ± 1.30	10.55 ± 3.53	4.73 ± 2.34
Smoking								
Non-smoking	90	5.16 ± 1.79	24.96 ± 11.56	1.53 ± 0.25	86	2.76 ± 1.14	10.04 ± 3.61	5.07 ± 2.53
Smoking	16	5.35 ± 1.90	25.15 ± 10.88	1.52 ± 0.30	30	3.30 ± 1.30	10.50 ± 3.81	4.67 ± 2.17
Alcohol								
Non-alcoholic	77	6.37 ± 1.71	25.45 ± 11.44	1.54 ± 0.31	85	2.93 ± 1.08	9.82 ± 3.49	5.06 ± 2.53
Alcoholic	29	6.38 ± 1.91	25.55 ± 10.93	1.53 ± 0.23	31	3.33 ± 1.34	10.86 ± 3.92	4.69 ± 2.18

* Significant at $P < 0.05$, multivariate ANOVA test was used with post hoc analysis for the comparison of SOD activity, catalase activity, and lipid peroxidation assay in multiple subgroups between the exposed and control groups separately

Table 5 SOD, catalase activity, and lipid peroxidation assay by SOD and catalase genotypes

Genotypes	Control group-106				Exposed group-116			
	N (%)	SOD assay (Mean ± SD)	Catalase assay (Mean ± SD)	Lipid peroxidation assay (Mean ± SD)	*N* (%)	SOD assay (Mean ± SD)	Catalase assay (Mean ± SD)	Lipid peroxidation assay (Mean ± SD)
mnSOD								
wt/wt	23	4.01 ± 0.85	26.39 ± 11.14	1.32 ± 0.22	29	1.97 ± 0.61	11.05 ± 4.13	3.99 ± 1.77
wt/mt	63	6.13 ± 1.32	28.92 ± 9.66	1.53 ± 0.22	62	3.17 ± 0.95	8.56 ± 2.42	5.01 ± 2.46
mt/mt	20	8.49 ± 0.97*	20.75 ± 10.60	1.61 ± 0.37	25	4.80 ± 1.08*	10.38 ± 3.70	5.95 ± 2.67*
CAT								
wt/wt	51	6.23 ± 1.78	45.28 ± 4.05	1.44 ± 0.25	48	3.20 ± 1.06	15.81 ± 1.82	3.93 ± 2.02
wt/mt	49	6.67 ± 1.86	31.83 ± 6.78	1.61 ± 0.23	63	3.48 ± 1.40	12.63 ± 1.95	5.63 ± 2.38
mt/mt	06	5.36 ± 1.75	15.19 ± 5.58*	1.67 ± 0.25	05	2.97 ± 1.15	7.02 ± 1.93*	6.38 ± 3.54*

* Significant at $P < 0.05$, multivariate analysis of covariance test was used to test for differences in SOD activity, catalase activity, and lipid peroxidation assay adjusted for age, gender, diet, consumption habits, and exposure duration among the exposed and control groups separately

attributed to an increased level of ROS, although the mechanism of their interaction with EMF is not fully understood.

Different studies have demonstrated that the microwaves at several frequencies are able to induce several changes in the levels of DNA (single and double strand) and micronuclei (chromosomal abbreviation) [29, 30]. Some researchers found different fertility problems in males due to the mobile phone radiations [31], while others have not observed any change in sperm motility [32]. At 2.45 and 50 GHz radiations, significant changes have been identified in brain antioxidant enzymes [33, 34]. Oktem

Table 6 Association of MnSOD and CAT genotypes with SOD, catalase, and lipid peroxidation assay as found by linear regression analysis

Genotype	SOD activity (Mean ± SD)			Catalase activity (Mean ± SD)			Lipid peroxidation assay (Mean ± SD)		
	β[a]	R^2	P^b value	β[a]	R^2	P^b value	β[a]	R^2	P^b value
Control group (106)									
MnSOD	1.223	0.287	0.000*	1.800	0.285	0.083	0.279	0.318	0.000*
CAT	0.164	0.093	0.683	5.420	0.304	0.000*	−1.277	0.127	0.008*
Exposed group (116)									
MnSOD	0.931	0.165	0.004*	3.243	0.114	0.139	−1.781	0.204	0.045*
CAT	−0.366	0.567	0.477	17.179	0.378	0.000*	−3.432	0.175	0.001*

[a] Unstandardized coefficient

[b] Model *P* value. Regression analysis was used to test for differences in SOD activity, catalase activity, and lipid peroxidation assay adjusted for age, gender, diet, exposure duration, and consumption habits

* Values are significant

et al. [35] investigated the effects of 900 MHz mobile-phone-induced oxidative stress in the rat kidney tissues and observed that MDA level increased and SOD, CAT, and GSH-Px activities decreased in the kidney tissues. The cause of increased MDA is the increase in lipid damage by the $O^{\bullet 2}$ radicals arising from the decrease in SOD activity.

Although antioxidant activity was not significantly associated with gender, the values were found to be low in females. We have not found any association with age groups. In some studies, in contrast to our results, elder people and children were more prone to damage from the mobile tower radiations [36]. Liubimova and Vorobtsova [37] found a linear increase in the number of aberrant cells with age among healthy population exposed to low dose of radiation.

Our findings showed that smoking and alcohol intake did not have a significant effect on antioxidant activity in both groups [38]. Maes et al. [39] also observed the absence of any relation between smoking habit and induction of genotoxicity, professionally exposed to RF-EMR. Bishop et al. [40] have not found any relation between alcoholic beverages and RFR toxicity although they contain mutagenic substances [41]. According to Dittberner et al. [42], alcohol use can increase the number of micronuclei. In the present work, no significant association of antioxidant activity was detected either in the exposed group or in the control group in individuals with drinking habits.

Behrendt et al. [43] observed the involvement of glutathione (GSH) and enzymes such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPX) in quenching excessive levels of ROS and other free radicals by non-ionizing radiation. Activity levels of these enzymes are likely affected by functional polymorphisms in the genes encoding them. In the present study, it was found that MnSOD and CAT polymorphisms were significantly associated with antioxidant activity. Single-nucleotide polymorphisms (SNPs) of antioxidant enzymes may contribute to diseases associated with oxidative stress. These SNPs and changes in the activities of the respective enzymes have been associated with altered progression and/or risk of diseases, including breast cancer, lung cancer, diabetic neuropathy, and acute kidney injury [35, 44–46].

This is the first study that shows the effect of exposure of mobile tower radiations on human health in Kurukshetra region of Haryana state in North India. Member Scientist, ICMR has indicated that the hot tropical climate of the country, low body mass index (BMI), and low fat content of an average Indian as compared to European countries and high environmental concentration of radio frequency radiation may place Indians under high risk of adverse effects of RF radiation. So there is a need to explore the possibility of the impact of geographical location on adverse health effects from EMF radiation from mobile towers. The literatures available remain sufficiently controversial to exclude any potential RF genetic hazard. In India, the prescribed reference level at 1800 MHz is revised to 0.92 W/m^2. The safe limit adopted in India is still very high as found in our study where also significant genetic damage was found in exposed subjects even at a power density of 12.2 mW/m^2, much below the current safety level adopted in India. Many countries in the world have adopted much stricter maximum radiation density values of 0.001–0.24 W/m^2 (1/100th to 1/1000th of ICNIRP guidelines) after studying extensively the health hazards of cell tower radiations. The evaluation of early biological effects such as genotoxic, cytotoxic, and oxidative damage could contribute to clarify the mechanisms of action of RF from mobile towers and to identify biomarkers of early cell disturbances and DNA damage useful in terms of health risk assessment.

However, the issue of mobile tower radiation is still controversial. Although the difference is statistically insignificant in females and children, a close look at Tables 1 and 2 discloses the higher values in the study

group compared to controls, which might indicate that the statistical meaning can change when larger populations are recruited. That is why we should be more hesitant to accept base stations launched close to residential areas. Science and technology should help each other to protect human health.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval The research protocol was approved by the institutional human ethics committee of Kurukshetra University, Kurukshetra (Haryana).

Informed consent Informed consent was obtained from all subjects.

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