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Electromagnetic Biology and Medicine

ISSN: 1536-8378 (Print) 1536-8386 (Online) Journal homepage: www.tandfonline.com/journals/iebm20

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To cite this article: Ravindra Tiwari, N. K. Lakshmi, S. C. Bhargava & Y. R. Ahuja (2015) Epinephrine, DNA integrity and oxidative stress in workers exposed to extremely low-frequency electromagnetic fields (ELF-EMFs) at 132 kV substations, Electromagnetic Biology and Medicine, 34:1, 56-62, DOI: 10.3109/15368378.2013.869755

To link to this article: https://doi.org/10.3109/15368378.2013.869755

Published online: 24 Jan 2014.

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ELECTROMAGNETIC BIOLOGY AND MEDICINE

http://informahealthcare.com/ebm
ISSN: 1536-8378 (print), 1536-8386 (electronic)

Electromagn Biol Med, 2015; 34(1): 56–62
© 2015 Informa Healthcare USA, Inc. DOI: 10.3109/15368378.2013.869755

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ORIGINAL ARTICLE

Epinephrine, DNA integrity and oxidative stress in workers exposed to extremely low-frequency electromagnetic fields (ELF-EMFs) at 132 kV substations

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Abstract

There is apprehension about widespread use of electrical and electromagnetic gadgets which are supposed to emit electromagnetic radiations. Reports are controversy. These electromagnetic fields (EMFs) have considerable effect on endocrine system of exposed subjects. This study was focused to assess the possible bioeffects of extremely low-frequency (ELF)-EMFs on epinephrine level, DNA damage and oxidative stress in subjects occupationally exposed to 132 kV high-voltage substations. The blood sample of 142 exposed subjects and 151 non-exposed individuals was analyzed. Plasma epinephrine was measured by enzyme-linked immunosorbent assay, DNA damage was studied by alkaline comet assay along with oxidative stress. Epinephrine levels of sub-groups showed mean concentration of 75.22 ± 1.46, 64.43 ± 8.26 and 48.47 ± 4.97 for high, medium and low exposed groups, respectively. DNA damage ranged between 1.69 μm and 9.91 μm. The oxidative stress levels showed significant increase. The individuals employed in the live-line procedures were found to be vulnerable for EM stress with altered epinephrine concentrations, DNA damage and increased oxidative stress.

Keywords

132 kV substation, comet assay, ELF-EMFs, epinephrine, oxidative stress

History

Received 27 July 2013
Revised 29 August 2013
Accepted 15 September 2013
Published online 21 January 2014

Introduction

The extensive use of technology that radiates electromagnetic fields (EMFs), electromagnetic radiation (EMR), and extremely low-frequency (ELFs) is a subject of controversy one. Some of thought is for over these decades EMFs can increase temperature of live tissue and have thermal effect on bio-organisms. These effects can change membrane electric potential and ion distribution and finally can disrupt biochemical reactions in cells (Lee et al., 2004). EMFs have considerable effect on endocrine system, particularly stress hormones (Hong et al., 2011; Koyu et al., 2005).

Studies have been carried out on pituitary hormones and hormones released from thyroid, adrenal and reproductive organs. The studies on EMF effects indicated both positive (Braune et al., 2002; Craviso et al., 2003; Toler et al., 1988) and negative (Buchner and Eger, 2011; Vangelova and Israel, 2005; Vangelova et al., 2002, 2007) impact on epinephrine secretion.

There have been several epidemiological studies of the possible adverse health effects associated with environmental exposure to ELF-EMFs, such as those emitted by power cables and electric substations, linking such exposure to childhood leukemia, brain tumors, genotoxic effects, neurological effects, neurodegenerative diseases, immune system deregulation, allergic and inflammatory responses, breast cancer, miscarriage and some cardiovascular effects (Hardell and Sage, 2008; Repacholi, 2012). The specific causes for the development of different types of human cancers are still poorly understood. However, certain investigations have provided sufficient mechanistic information relevant to the assessment that melatonin suppression may be responsible for various biological effects (Blask et al., 2011). There has also been speculation that ELF-EMFs can act as promoters or co-promoters of cancer (Wertheimer and Leeper, 1979; Zhu et al., 2003).

Based on the epidemiological association between residential exposure to ELF-EMFs and childhood leukemia, the International Agency for Research on Cancer (IARC) classified ELF-EMFs as a ''possible human carcinogen'' (IARC, 2002).

Hence this study was carried out and to investigate the effect of ELF-EMFs on epinephrine levels in plasma of the exposed subjects. DNA damage in peripheral blood leukocytes, the plasma levels of malondialdehyde (MDA), nitric oxide (NO) were also investigated as the markers of oxidative stress. The association between selective markers of

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oxidative stress, DNA damage and epinephrine level induced by ELF-EMFs was examined.

Methodology

Participants

A total of 293 volunteers participated in the study. The randomized controlled sampling of participants included subjects ($n = 142$) exposed to EMFs emitted from high-voltage substation for more than 2 years of occupational exposure. The control population ($n = 151$) included individuals who are not exposed to such EMFs. The screened control population was age matched with similar socioeconomic status shown in Table 1. The subjects did not have any family history of genetic anomaly or major illness. Informed consent was taken from all the participants.

Performa/questionnaire

Age, gender, diet, exercise and recent infection were used as criteria for the selection of both exposed and control populations. Detailed questionnaire on work characteristics, psychological perceived stress and non-specific symptoms experienced by exposed populations were responded by 94% of participants. Questionnaire on subjective symptoms related to EMFs exposure included.

Sampling

After receiving informed consent, 5 ml of peripheral blood was collected in the morning between 8 AM and 10 AM from each of the 142 exposed subjects and 151 controls by venepuncture into a sterile disposable syringe. A total of 2 ml of anticoagulated whole blood components were processed into plasma for Epinephrine estimation and MDA levels. In total, 1 ml of whole blood was placed in ice to prevent exogenous damage and processed in the laboratory within an hour of collection for DNA damage. A total of 2 ml of whole blood was allowed to clot and serum was separated by centrifugation for NO estimation.

Epinephrine assay (enzyme linked immunosorbent assay)

Chemicals

Epinephrine standard (256 ng/ml), acylation buffer, acylation reagent, assay buffer, extraction buffer, catechol-*O*-methyltranferase, *S*-adenosyl-L-methionine, enzyme buffer, epinephrine antiserum, anti-rabbit IgG conjugated with peroxidase, TMB substrate – LDN GmBH & Co KG, Germany, Hydrochloric acid-SD Fine Chemicals, Mumbai, India.

Table 1. Demographic characteristics of the study groups and control.

S. No	Variables	Total	ELF-EMF exposed	Control
1	Number (n)	293	142	151
2	Age (range in years)	293	20–58	25–55
3	Sex – Males	142	142 (100%)	151 (100%)
4	Dietary pattern			
	Vegetarian	105	14 (09.86%)	15 (09.93%)
	Non-vegetarian	188	128 (90.14%)	136 (90.07%)
5	Duration in years			–
	Mean ± SD	142	9.27 ± 7.93	
	Range		2–30	

Assay

Plasma epinephrine was quantitatively measured by enzyme linked immunosorbent assay (ELISA). Epinephrine was extracted using a Cis-diolspecific affinity gel, then acylated to *N*-acylepinephrine and after this converted enzymatically during the detection procedure into *N*-acylmethanephrine. The competitive ELISA kit uses the microtiter plate format. Epinephrine is bound to the solid phase of the microtiter plate. Acylated epinephrine from the sample and solid phase bound epinephrine compete for a fixed number of antiserum binding sites. When the system is in equilibrium, free antigen and free antigen-antiserum complexes are removed by washing. The antibody bound to the solid phase catecholamine was detected by an anti-rabbit IgG-peroxidase conjugate using TMB as a substrate. The reaction was monitored at 450 nm with the amount of antibody bound to the solid phase catecholamine being inversely proportional to the catecholamine concentration of the sample.

Quantitative determination

The concentration of plasma epinephrine in the samples was calculated from calibration curve obtained by plotting the absorbance readings of the standards (linear, *y*-axis) against the corresponding standard concentrations (logarithmic, *x*-axis) using a non-linear regression for curve fitting technique.

Alkaline comet assay

Chemicals

The sources of chemicals were as follows: Agarose low melting (20 °C) and normal melting point (35 °C)], sodium lauryl sarcosinate, titron X-100, silver nitrate, catalase from Sigma (St. Louis, MO); tungstostilic acid from Koch-light Laboratories (Colnbrook-Bucks, England); sodium chloride, sodium hydroxide, potassium chloride, TRIS, EDTA, potassium dihydrogen phosphate and sodium phosphate dibasic from Glaxo (Mumbai, India); zinc sulphate and ammonium nitrate from Fischer (Madras, India); thiobarbituaric acid, butylated hydroxyl toluene, sulphosalicyclic acid and *N*-1-napthyl ethylene diamine dihydrochloride from SD Fine (Mumbai, India).

Assay

Alkaline Comet assay or Single Cell Gel Electrophoresis (SCGE) was used to study DNA damage (Singh and Lai, 1998).

Preparation of slides

On a clean, dry plain slide 100 µl of 0.75% Normal melting agarose (NMA) prepared in phosphate buffered saline (PBS) was layered. These precoated slides were dried at 37 °C. On top of this layer, 30 µl of PBL suspended in PBS, mixed with 110 µl of 0.5% low melting agarose (LMA) prepared in PBS was layered. The third layer consisted of 100 µl of LMA.

The slides were incubated in cold lysis buffer (2.5 M NaCl, 100 mM Na_2EDTA, 10 mM Tris;1% sodium lauryl sarcosinate;1% Triton X-100 and DMSO added fresh) at 4 °C overnight.

Alkali treatment and electrophoresis

The slides were removed from the lysing solution and placed side by side in a horizontal electrophoresis unit. The slides were completely immersed in freshly prepared alkaline electrophoretic buffer (1 mM Na_2EDTA and 300 mM NaOH, pH 13) for 30 min to facilitate the DNA unwinding and expression of alkali labile sites.

After alkali treatment, the electrophoresis was carried out for 30 min at 300 mA and 0.67 V/cm. The slides were carefully lifted from the electrophoresis unit and gently washed with neutralizing buffer (0.4 M Tris buffer, pH 7.5). The slides were then washed with distilled water and air dried.

Silver staining

The air dried slides were immersed in the fixing solution (15% w/v trochloroacetic acid, 5% w/v zinc sulphate and 5% w/v glycerol) for 10 min and washed gently with double distilled water several times. For staining, 32 ml of staining solution A (5% w/v Na_2CO_3) was mixed with 68 ml of staining solution B (0.02% w/v NH_4NO_3, 0.02% w/v $AgNO_3$, 0.1% w/v tungtosilic acid and 0.05% v/v formaldehyde) and poured over the slides so as to cover the slides uniformly and left for about 2 min. The staining solution was drained and the step was repeated with a fresh mixture of staining solution, until a grayish color developed on the colorless slides. The staining process was stopped by draining the staining solution and immersing the slides in stopping solution (1% acetic acid) for 5 min. The slides were then washed with double distilled water and air dried for screening (Ahuja et al., 1999).

Evaluation of DNA damage

For visualization of DNA damage, a bright field, transmission light microscope (Leitz) was used at 400× magnification. Comet tail length was measured, using an ocular micrometer fitted in the eyepiece, in 200 cells per treatment. Mean comet tail length, which is an estimate of DNA damage, was calculated for each sample.

Lipid peroxidation – thiobarbituric acid reactive substances (TBARS) assay

MDA is the stable end product of lipid peroxidation (**LPO**) and an efficient parameter for lipid oxidative damage (Halliwell and Gutteridge, 1999). Formation of MDA is assessed by quantification of thiobarbituric acid reactive substances (TBARS).

TBARS, MDA, an end product of **LPO**, reacts with TBA to form a 1:2 adduct with thiobarbituric acid which can be measured by spectrophotometry.

In the process, 1 ml of protein free plasma obtained after centrifugation was treated with thiobarbituric acid (0.33%) in boiling water bath for 30 min to form an 1:2 adduct which is measured at 535 nm with spectrophotometer (Schimadzu UV-160 A, Cat No: 204-04550-01, Made in Japan). Mean and ± SD or SE was calculated for the samples. The concentration of MDA equivalents in nmol/ml in samples were interpolated from the standard curve.

Estimation of serum nitric oxide

As NO is an unstable compound, it is rapidly converted to nitrates and nitrites in the body; hence their concentration is parallel to NO levels. Total production of nitric oxide (NO) was determined by assaying for nitrite and nitrate (Green et al., 1982). Nitrate and nitrite concentrations were then estimated by the Griess method (Stuehr et al., 1989). In this method nitrate is first reduced to nitrite which is treated with sulfanilamide and N1-napthyl-ethylene diamine. The coloured compound is formed after which its characteristic absorption spectrum was determined on spectrophotometry.

A total of 500 µl of sulfosalicylic acid was added to 500 µl of serum in a centrifuge tube. The reaction was allowed for 5 min with mild stirring carried out for every 30 s followed by centrifugation at 3000 rpm for 20 min. After centrifugation, to 200 µl of supernatant, 30 µl of 10% NaOH and 300 µl of Tris HCl buffer (pH 9.0) were added, 500 µl of freshly prepared Griess reagent was added to the above mentioned reaction mixture. The reaction was allowed in dark for 10 min and later read at 540 nm against double distilled water as blank. The lower limit detection of the assay was 5 µmol/l. The optical densities of the samples were then obtained on a spectrophotometer (Schimadzu UV-160 A, Cat No: 204-04550-01, Made in Japan) at 540 nm.

Statistical analysis

The comet assay slides were coded during processing and decoded at the time of statistical analysis. Descriptive statistics were applied for calculating the distribution of various characteristics. For statistical evaluation, observations on each parameter for each group were pooled and mean ± SD (SE) was calculated. Student's *t* test (paired and unpaired comparisons), one-way analysis of variance (ANOVA) were performed to evaluate various differences.

An unpaired student's *t* test and one-way ANOVA were applied to determine the significance of biochemical changes of MDA and NO levels. Multiple regression analysis was done to study the effects of confounding factors. Statistical software SPSS 16 (South Asia Pvt. Ltd., Bangalore, India) was used to carry out statistical analysis.

Results

The demographic details of the exposed and control groups are shown in Table 1. The age and socioeconomic status and lifestyle factors were matched between exposed groups and control. Most of the subjects in the exposed group and control belonged to low and middle socioeconomic status. When dietary pattern of the subjects were analyzed, most of the subjects were non-vegetarians.

Analysis by exposure characteristics

The sources of exposure include high-voltage substations (132 kV). The electric utility workers were exposed for a mean duration of 9 years, to the field intensities as shown in Table 1.

Results of epinephrine levels

Using independent *t* test the plasma epinephrine concentration did not differ significantly between the sub-station workers and controls as shown in Table 2. The sub-groups based on exposure levels also showed no significant difference from ANOVA.

The distance from indoor office areas to 132 kV sub-stations were measured. Two average distances were calculated from the different sampling stations as less than 2 m and 10 m. The effects of distances on stress hormone concentrations were evaluated using multinational logistic regression. Distance less than 2 m had higher relevance to the altered epinephrine concentration as shown in Table 3.

Results of DNA damage

The results demonstrated DNA migration or comet tail length as an index of DNA damage ranging between 0.965 μm and 9.939 μm. The maximum tail length (9.939 ± 0.67 μm) was observed in an ELF-exposed individuals working as live-line personnel. Mean tail length was observed to be (9.113 ± 0.42 μm). The results of basal DNA damage assessed by alkaline comet assay in terms of comet tail length, standard deviation (SD), range and variance are summarized in Table 2.

Results of oxidative stress

The mean MDA concentration (nmoles/ml) of the ELFs exposed showed significant increase in MDA levels when compared with controls as shown in Table 2.

In both control and the exposed group, the levels of serum **NO** are determined as nitrites or nitrates. The overall data revealed an increased level of serum **NO** in the exposed groups $p = 0.054$ as shown in Table 2.

The effect of duration of work in electric utility workers were significantly high in MDA and NO levels (>14 years) among the sub-groups as shown in Table 4. Frequency of daily exposure had significant effect in oxidative stress parameters among the sub-groups shown in Table 5. The oxidative stress markers showed no relationship with exposure variables as assessed from regression analysis.

The high exposure group of live-liners showed significantly different oxidative stress parameter levels (>7 h) compared to other sub-groups. To a lesser extent, the maintenance workers had significant MDA levels compared to the low exposed sub-group as shown in Tables 5 and 6.

Discussion

The research into the potential health effects of exposure to EMF has been underway for several decades. A number of independent scientific committees and working groups have reviewed the literature and concluded that, exposures to EMF may impose adverse health effects in humans (AGNIR, 2006; Ahlbom et al., 2008; NIEHS, 1998; Vijayalaxmi, 2005). Nonetheless, considering the scientific ''weight of evidence'', mostly from human epidemiological data, the IARC concluded that EMF exposure acts as ''possible human carcinogen'' in the IARC category of Class 2B (IARC, 2010).

Bioeffects on occupational exposure to ELF-EMFs

The hypothesis that chronic exposure to residential and or occupational ELF-EMFs may cause adverse health effects has led to a great deal of research. Epidemiological reports particularly on increased risk of childhood leukemia, breast cancer and neurological disorders have been a subject of

Table 3. Effect of distance on stress hormonal configurations.

Distance	B	SE	Significance	OR	95% CI
Less than 10 m epinephrine	0.017	0.011	0.129	1.017	0.99–1.04

Table 2. Results of DNA damage, MDA, NO and EPINEPHRINE levels in the exposed compared with control group.

DNA damage					
		Comet tail length (arbitrary units)			
Subjects	*N*	Mean ± SD	Range	Variance	
ELF-EMF	142	3.98 ± 1.31	1.69–9.91	1.713	
Control	151	3.48 ± 1.13	1.44–7.38	1.287	
MDA levels (nmoles/ml)					
Subjects	*N*	MDA levels (nmoles/ml)		T value	*p* Value
		Mean ± SD	Range		
ELF exposed	142	2.67 ± 0.82	0.90–9.40	2.045	0.042
Control	151	2.37 ± 1.46	0.94–5.20	–	–
NO (μmoles/ml)					
ELF exposed	142	2.25 ± 0.09	0.42–4.58	2.148	0.033
Control	151	2.00 ± 0.07	0.66–4.30	–	–
Plasma epinephrine (pg/ml)					
ELF exposed	142	49.66 ± 5.17	10.45–33.76	2.002	0.08
Control	151	58.42 ± 6.48	11.25–33.05		

MDA, malondialdehyde; NO, nitric oxide.

Table 4. Oxidative stress in relation to duration (years) of occupationally exposed electric utility workers.

Observation		Group based on duration of exposure (years) A (2–5)	B (6–13)	C (14–30)	*F* value	*p* Value	Group comparison A–B	A–C	B-C
MDA	*N*	42	45	55	3.428	0.035	1.22[a]	0.16	−1.05[a]
	Mean ± SE	2.77 ± 0.23	2.33 ± 0.18	2.87 ± 0.23					
NO	*N*	42	45	55	4.593	0.012	0.07	0.57[a]	0.50[a]
	Mean ± SE	2.21 ± 0.13	2.07 ± 0.17	2.43 ± 0.15					

MDA, malondialdehyde; NO, nitric oxide.
[a]The mean difference is significant at 0.05 level.

Table 5. Oxidative stress in relation to frequency of exposure (hours/day) to occupational ELF-EMFs of electric utility workers.

Observation		Group based on frequency of exposure (hours/day) A (1–3)	B (4–6)	C (7–9)	F value	*p* Value	Group comparison A–B	A–C	B–C
MDA	*N*	59	45	38	0.625	0.537	0.217	−159	−0.376
	Mean ± SE	2.70 ± 0.19	2.48 ± 0.22	2.86 ± 0.27					
NO	*N*	59	45	38	1.422	0.245	0.192	−0.206	−0.398
	Mean ± SE	2.26 ± 0.15	2.07 ± 0.13	2.56 ± 0.18					

MDA, malondialdehyde; NO, nitric oxide.

Table 6. Oxidative stress in relation to exposure metric of occupationally exposed electric utility workers.

S.no	Exposure metric	Subjects (*n*)	Observation	Mean ± SE	95% CI for mean
1	Low	87	MDA	1.61 ± 0.22	2.47–3.18
		87	NO	2.45 ± 0.12	2.21–2.69
2	Medium	44	MDA	2.67 ± 0.20[a]	2.26–3.07
		44	NO	1.88 ± 0.13	1.61–2.15
3	High	11	MDA	2.82 ± 0.18[a]	1.12–2.10
		11	NO	2.38 ± 1.13[a]	1.62–3.14

[a]Significant at the 0.05 level.

National and International reviews (Hardell and Sage, 2008). There has been a steady increase in investigations examining the genetic effects of EMF exposure in *in vitro* experimental designs and *in vivo* animal models. However, very few epidemiological studies have directly evaluated the cause of such bioeffects. As studies on genotoxicity are highly relevant in evaluation of carcinogenicity, the present study is focused on such an issue in occupational settings of ELF exposure. Moreover, in India such studies are not conducted for evaluating the role of epinephrine and oxidative stress effects of magnetic fields on high-voltage substation workers.

It has been suggested that EMFs may induce cell specific activation processes, which in turn activate signal transduction pathways causing excessive reactive oxygen species (ROS) formation/cell cycle disturbances. These disturbances have the potential to disrupt DNA integrity (Lai and Singh, 2004).

ELF effect on oxidant–antioxidant balance

The MDA levels in erythrocytes of ELF-exposed subjects suggest a statistical significance in **LPO** activity. Owing to the great number of biological actions of free radicals, the LPO activity could have an important implication in the comprehension of the biological stress response to ELF-EMFs. Oxidative DNA damage and LPO activity in exposure to ELF-EMFs was reported in certain *in vivo* experimentations and animal models. A time-dependent significant higher TBARS levels ($p < 0.001$) and induced DNA damage ($p < 0.05$) in the exposed group was observed (Yokus et al., 2005).

The total nitrite concentration was statistically higher in the sub-station workers. A similar observation indicating the oxidative stress in the samples of men exposed to 1.5 T static magnetic field was reported (Sirmatel et al., 2007). Altered **NO** production in prolonged ELF-EMFs exposure at intensities of exposure limits was determined by International Commission on Non-Ionizing Radiation Protection (ICNIRP) for public and occupational exposure (Akdag et al., 2007). Few studies have reported the role and involvement of **NO** in magnetic field effects (Jelenkovic et al., 2006; Reale et al., 2006; Whissell and Persinger, 2007). Negative results on **NO** levels are shown in other studies (Kirichuk et al., 2007).

It is also shown that **NO** synthase is supposed to be stimulated by the EMF exposure (Jeong et al., 2006; Kim et al., 2002; Seaman et al., 2002). This compound may be involved in the stimulation of lipid peroxidation indirectly. This hypothesis could thus explain the increased TBARS production in the exposed group.

Furthermore, few studies have focused on the involvement of oxidative stress in the action mechanism of EMF exposure (Hook et al., 2004). Exposition to electric fields (50 Hz) of

Sprague-Dawley rats did not change the antioxidant activity and lipid peroxide level of unstressed animals but decreased the plasma peroxide level in stressed rats (Harakwa et al., 2005; Simko et al., 2001). A diminution in the activity of MDA after 24 and 48 h of EMF exposition (180–195 Hz) was observed in preadipocytes (Torres-Duran et al., 2007; Zwirska-Korczala et al., 2005). The early increase in the TBARS concentration observed in our study could be the result of the reduction in the activity of antioxidant enzymes or and the increase of free radical production.

Since free radicals are able to damage many biomolecules, including DNA, enzymes, lipids and proteins, we can hypothesize that modulation in antioxidative parameters observed in our study could be due to overproduction of NO and MDA under ELF-EMFs exposure. The altered oxidant-antioxidant imbalance was highly pronounced in live-line workers. Oxidative damage was evident in these sub-group workers with increased MDA and NO levels.

ELF-EMFs effect on neuroendocrine hormone and stress parameters

The circulating level of specific hormone of neuroendocrine system, the epinephrine was evaluated to elucidate the effect of ELF-EMFs on altered release of the catecholamine. The mean concentration of epinephrine was not significantly altered from those of healthy individuals. Relevant to our findings, the *in vitro* magnetic field experimentations had shown no altered response on catecholamine levels (Craviso et al., 2003; Zwirska-Korczala et al., 2005).

After adjusting the potential cofounders, increase in epinephrine levels were shown in subjects reporting electromagnetic hypersensitive symptoms. Significant differences in sub-groups with job titles and distances were noted. However, such response of epinephrine activity to EMFs exposure was assessed in very few studies with contradictory results (Braune et al., 2002; Craviso et al., 2003; Jeong et al., 2006).

Men employed as live-line workers, or as line-men, could be at risk, with indications of increased incidences of cytogenetic damage and stress. The live-line workers in high-voltage substation were identified as vulnerable groups. The live-line workers in our sampled substation use bare-hand methods. The line-men is positioned in direct contact with the live components and is livened or raised to the electrical potential of the conductor being worked on. In practice live-line work is potentially very hazardous and involves high risk. The average distance measured from working area to transformer locations in different 132 kV substations was related to stress. Though the safeties of workers are ensured, the impact of their routine procedures in maintaining live-lines might result in adverse stress effects.

Stress as a predicting factor was also evaluated based on self-reported subjective symptoms and questionnaires. About 8% experienced induction and shocks in their occupation. The subjective symptoms reported by the workers include predominantly headache (8%), followed by sleeplessness (2%).

Relevance of electromagnetic field exposure indices

The exposure indices at 132 kV substations such as magnetic field strength, duration, intensity, job titles and distance of working area from transformer in 132 kV substations were considered. The inclusion of duration and intensity of exposure in evaluating personal exposure level can be of prime importance. For the duration estimates, we systematically used the number of years of exposure, which are expected to provide better estimates of the duration of long-term exposure. Furthermore, intensity is estimated separately from daily exposure or working hours per day.

Another important strength of examining job titles over other indices was the simplicity and ease of predicting the exposure intensity. The results from our earlier report on cytogenetic assays suggested the induction of DNA damage and increase in frequency of micronuclei in substation workers, probably due to genotoxic action of ELF-EMFs (Selmaoui et al., 1996). This genotoxic effect may also increase on co-exposure of occupational magnetic field densities with other environmental mutagen.

Conclusion

The present study explains the possible bioeffects of electromagnetic exposure to occupational environments.

The long-term effects of ELF exposures are also denoted in the genetic material. This finding on genomic instability could be important for understanding the causal effect of EMF exposure and health effects. The possibility that EMFs might induce genomic instability has important implications, since human populations are likely to be exposed concomitantly with this type of physical agent. Among the sub-station workers, the individuals employed in the live-line procedures were found to be vulnerable for EMF stress with altered epinephrine concentrations.

Hence it is assumed that, there is an urgent need to implement a master plan for EMFs and public health. More research is needed on studies carried out for stress markers like epinephrine and oxidative stress biomarkers. The live-line workers should be educated about the risk associated with EMFs. Prevention plans like changing work shifts weekly, maintaining adequate distance from the live-lines, minimizing the duration of exposure and wearing protective suits should be mandatory.

Declaration of interest

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

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