

# Research Journal of Pharmaceutical, Biological and Chemical Sciences

## Impact of Mobile Phone Radiations on Human Health with Special Reference to SAR (Specific Absorption Rate)

Sharanabasappa M Awanti<sup>\*1</sup>, Gurulingappa A Patil<sup>2</sup>, Revansidappa B Patil<sup>2</sup>, Santoshkumar R Jeevangi<sup>3</sup>, Jagadish B Ingin<sup>4</sup>, Rashmi Sinha, Raghunandana R.

<sup>1</sup>Dept of Biochemistry, M.R Medical College, Gulbarga,

<sup>2</sup>Dept of Anaesthesiology, M.R Medical College, Gulbarga

<sup>3</sup>Dept of Physiology, M.R Medical College, Gulbarga

<sup>4</sup>Dept of Pharmacology, M.R Medical College, Gulbarga

### ABSTRACT

In recent times there has been considerable public scientific and media interest in the possible adverse effects associated with radiofrequency radiations emitted from the mobile phones on human health. Radiofrequency radiations can cause physiological change in brain cells and cause other biological damage through heating, this can lead to headache, blurring of vision, short term memory loss, bad sleep etc. These effects may be associated with SAR (Specific Absorption Rate) which is defined as rate of energy absorbed at a point of absorbing body [(expressed in (W/Kg)]. The present study was designed to know whether greater SAR of mobile devices has more severe effects on human health. The study was conducted on 60 volunteers who were using different model handsets of different manufacturers for a period of 2-5 years. The volunteers were grouped into 2 groups. Group-I volunteers used mobile phones with SAR values of their handset ranging from (0.5-0.8w/kg) and group II volunteers used mobile phones with SAR ranging from (0.8-1.2w/kg). Plasma obtained from the venous blood sample was used for estimation of lipid hydroperoxide, total thiols & Ferric reducing ability of plasma (FRAP) using spectrophotometric methods. There was significant increase in lipid hydroperoxide ( $p < 0.05$ ) and significant decrease in FRAP ( $p < 0.05$ ) and total thiols ( $p < 0.05$ ) in group-II volunteers compared to group-I. SAR (Specific Absorption Rate) of mobile device correlated negatively with thiols ( $p < 0.01$ ) and positively with lipid hydroperoxide ( $p < 0.01$ ). Decrease in antioxidants like FRAP and thiols and increase in oxidants like lipid hydroperoxide in group-II volunteers compared to group-I volunteers proves that greater SAR of a mobile handset may cause more ill effect on human body.

**Keywords:** FRAP (Ferric reducing ability of plasma), Total thiols, Lipid hydroperoxide, Oxidative stress, Antioxidants.

*\*Corresponding author*



## INTRODUCTION

Mobile phones are low power radio devices that transmit and receive radio frequency radiation in the microwave range of 900 - 1,800 MHz through an antenna used close to the user's head. Various types of analogue and digital cellular phones are in use across the globe. Analogue telephones transmit modulations of the amplitude or frequency of electromagnetic waves which are transmitted continuously. On the other hand, the digital telephones transmit data in series of pulses or fast bursts. Electromagnetic radiation (EMR) produced by mobile phones and base stations produce is absorbed in the body and produces heat, but the body's normal thermo regularity processes carry this heat away.

Several previous studies have reported that electromagnetic radiations emitted from mobile phones can lead to physiological changes in brain, brain cells and cause other biological damage through heating effects, leading to headaches, blurring of vision, short-term memory loss, burning sensation and bad sleep [1,2,3].

Because of the public concern on biological effect of mobile phone on human health including children's, various public organization such as World Health Organization (WHO) and International Commission on Non-Ionization Radiation Protection (ICNIRP) have established safety guidelines. The International Commission on Non-Ionization Radiation Protection (ICNIRP) has stated a maximum SAR limit of 1.6 W/kg on currently-marketed cell phones. Specific Absorption Rate (SAR) which is defined as rate of energy absorption at a point of absorbing body (expressed in W/kg).

SAR measurement is used to determine compliance with theoretical and estimated non-ionizing radiation hazard standards of the mobile handsets, and its use has never been proven valid or safe. Several previous reports have indicated the role of electromagnetic waves on biological system causing enhanced production of free radicals which intern causes increased lipid peroxidation leading to oxidative stress [4]. The present study was designed to evaluate the effect of exposure to electromagnetic radiations emitted from commercially available mobile phones on the plasma parameters of oxidative stress and antioxidants and to know whether greater SAR of mobile devices has more severe effects on human health.

## MATERIALS AND METHODS

The study was conducted on plasma samples of 60 volunteers. The volunteers were grouped into two. Group-I volunteers (n=30) used mobile phones with SAR values of their handset ranging from (0.5-0.8w/kg) and group II volunteers (n=30) used mobile phones with SAR ranging from (0.8-1.2w/kg). Informed consent was taken from all the subjects and was approved by institutional review board. The investigation confirms to the ethical guidelines for biomedical research on human subjects.

Under aseptic conditions blood samples (5ml) were drawn in heparinized vacutainers. Blood was centrifuged at 2000g for 15 minutes at 4°C for clear separation of plasma and all assays were performed immediately. All chemicals were purchased from sigma chemicals Co (St.Louis, MO, USA). Plasma total thiols are measured by spectrophotometric method using 5' 5' dithio-bis (2- nitrobenzoic acid) DTNB [5]; 900µl of 0.2 M Na<sub>2</sub>HPO<sub>4</sub> containing 2Mm Na<sub>2</sub>EDTA, 100 µl Serum and 20 µl of 10Mm DTNB in 0.2 M Na<sub>2</sub>HPO<sub>4</sub> were taken in an eppendorf tube and warmed to 37°C. the solution was mixed in a vortex mixer and transferred to cuvette and the absorbance was measured at the end of 5minutes at 412nm in Genesys 10 UV spectrophotometer. Appropriate sample and reagent blanks were prepared and corrected absorbance values [absorbance of T-(absorbance of standard blank + absorbance of reagent blank)] were used to calculate the concentrations of thiols using calibration curve. Values were expressed in µmoles/L for plasma thiols.

The lipid hydroperoxide content of plasma was determined with Fox version II assay for lipid hydroperoxides (Fox<sub>2</sub>) [6] The Fox<sub>2</sub> reagent was prepared by dissolving ammonium ferrous sulphate (9.8mg) in 10 ml of 250 mmoles/L H<sub>2</sub>SO<sub>4</sub> to give a final concentration of 250µmoles/L ferrous ion in acid. This solution was then added to 90ml of HPLC grade methanol containing 79.2mg butylated hydroxyl toluene (BHT). Finally, 7.6 mg of xylenol orange was added with stirring to make the final working reagent (250 µmol/L ammonium ferrous sulphate, 100 µmol/L xylenol orange, 25mmol/L H<sub>2</sub>SO<sub>4</sub> and 4mmol/L BHT in 90% vol/vol methanol in a final volume of 100ml), the working reagent was routinely calibrated against solutions of H<sub>2</sub>O<sub>2</sub> of known concentration.

Aliquots (90µl) of serum were transferred to two test tubes. Triphenylphosphine (TPP) in methanol (10 µl of 10mmol/L) was added in one to remove hydroperoxides. Methanol (10 µl) was added to the other test tube. This generated the blank and test samples respectively. Aliquots were then vortex mixed and incubated at room temp for 30 minutes before the addition of 900 µl FOX<sub>2</sub> reagent with mixing. After incubation at room temperature for further 30 min, the vials were centrifuged at 2000g for 10 minutes. The absorbance of the supernatant was then determined at 560nm. Hydroperoxide content in the serum sample was determined as a function of the mean absorbance difference of samples with and without elimination of hydroperoxides by TPP. Concentration of hydroperoxides was calculated using the extinction coefficient  $4.5 \times 10^{-4} \text{M}^{-1} \text{CM}^{-1}$ , and results were reported as micromoles per liter (µmol/L).

The FRAP assay was done according to Benzie and Strain [7] with some modifications. The stock solution included 300Mm acetate buffer (3.1g C<sub>2</sub>H<sub>3</sub>NaO<sub>2</sub>.3H<sub>2</sub>O and 16 ml C<sub>2</sub>H<sub>4</sub>O<sub>2</sub>), pH 3.6, 10mM TPTZ (2,4,6 tripyridyl-3-triazine) solution in 40mM Hcl, and 20Mm FeCl<sub>2</sub>.6H<sub>2</sub>O solution. Fresh working FRAP solution was prepared by mixing acetate buffer, TPTZ, FeCl<sub>2</sub> in 10:1:1 ratio. The 50 µl of plasma was made to react with working FRAP solution. The solution was mixed in a vortex mixer and transferred to cuvette and the absorbance was measured at the end of 6 minutes at 593nm in Genesys 10 UV spectrophotometer. The standard curve was linear between 25-80 µM.

### Statistical analysis

All the values are expressed as mean  $\pm$  SEM. A p value less than 0.05 is considered as significant. Statistical analysis was done using SPSS (statistical package for social sciences, SPSS-17, Chicago, USA). Independent sample t test was used to compare mean values. Pearson's correlation was used to correlate between the parameters.

### RESULTS

There was significant increase in lipid hydroperoxide ( $p < 0.05$ ) and significant decrease in FRAP ( $p < 0.05$ ) and total thiols ( $p < 0.05$ ) in group-II volunteers compared to group-I (Fig-1). SAR (Specific Absorption Rate) of mobile device correlated negatively with thiols ( $p < 0.01$ ) (Fig-2) and positively with lipid hydroperoxide ( $p < 0.01$ ) (Fig-3).

Figure 1. Total thiols, FRAP, Lipid hydroperoxide levels in mobile phone users with different SAR values

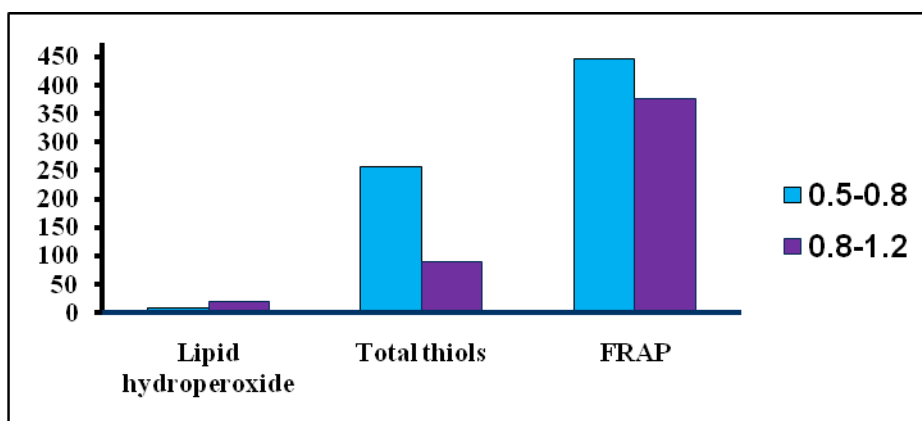


Figure 2. Correlation between total thiols and SAR value of mobile phones

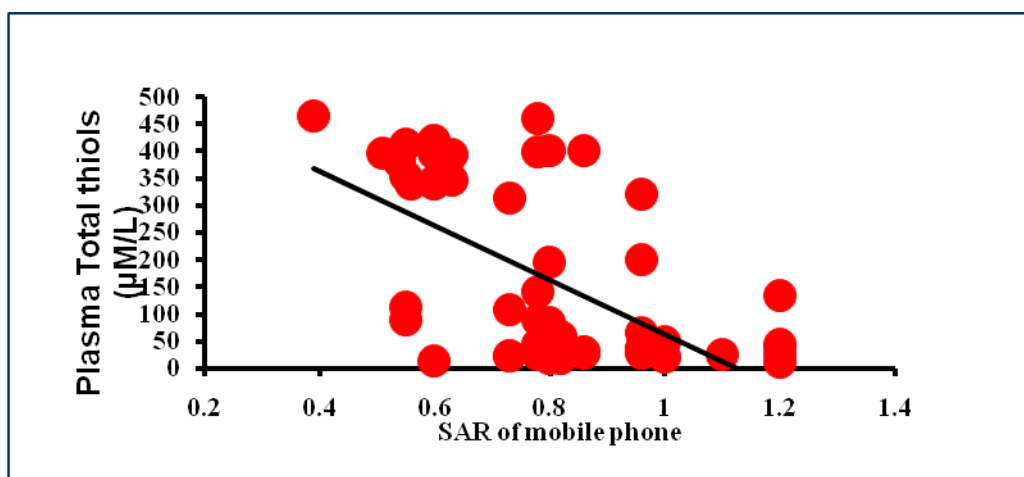
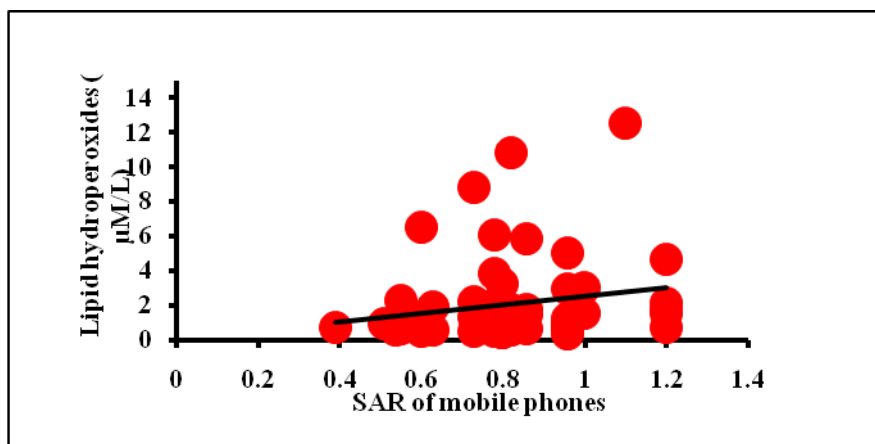


Figure 3. Correlation between lipid hydroperoxide and SAR value of mobile phones



### DISCUSSION

Oxidative stress refers to an imbalance between the intracellular production of reactive oxygen species (ROS) and cellular defense mechanisms. Proteins, lipids and DNA are sensitive targets of ROS. An excess availability of free radicals accompanied with a reduction of the capacity of the natural antioxidants systems leads to cellular dysfunction and death [8]. Hydroxy radical ( $\cdot\text{OH}$ ),  $\text{O}_2^{\cdot-}$  are the predominant cellular free radicals, while hydrogen peroxide ( $\text{H}_2\text{O}_2$ ) and peroxynitrate ( $\text{ONOO}^-$ ), although not themselves free radicals, but aid substantially to the cellular redox state [9]. The cytotoxicity of free radicals is related to the ability of these molecules to oxidize cell constituents, particularly lipid and nucleic acids.

Several mechanisms are in place to neutralize these effects; which include a system of nutritional and indigenous antioxidants defense that generally hold the production of free radicals and prevent oxidative stress and subsequent tissue damage [10]. The balance between the oxidants and antioxidants may be disturbed by an increase in free radical production or by reduction in antioxidants [11]. This imbalance between the oxidants and antioxidants can lead to oxidative stress in a series of peculiar and potentially damaging reactions [11] particularly susceptible to oxidative damage by free radicals are the polyunsaturated fatty acids, acyl chain of phospholipids, which leads to lipid peroxidation. Uncontrolled lipid peroxidation is a toxic process resulting in the deterioration of biological membrane [12]. Lipid peroxidation products eg; malondialdehyde has been taken as a biomarker to oxidative stress in biological system, which is relatively non-specific in biological samples like plasma [13]. Therefore, we have estimated lipid hydroperoxide along with antioxidants like thiols, & FRAP. Epidemiological studies still remain inconclusive with regard to the health effects of prolonged exposure to electromagnetic field [14-19]. Wireless communications especially mobile phones are rapidly gaining popularity knowing little about the harmful effects of these phones on human health.

The present showed decrease in FRAP and thiols and increase in oxidants like lipid hydroperoxide indicating increased oxidative stress in this population. Negative correlation of

FRAP and total thiols with lipid hydroperoxide depicts increased consumption of antioxidants in such oxidative environment. Negative correlation of SAR of mobile phones with thiols and positive correlation with lipid hydroperoxide indicates its positive association with oxidative stress. In conclusion our study showed decrease in antioxidants like FRAP and thiols and increase in oxidants like lipid hydroperoxide in group-II volunteers compared to group-I volunteers proves that greater SAR of a mobile handset may cause more ill effect on human body. However, our data is just an indication of such possibilities, to prove this conclusively one need to undertake well designed studies to understand the molecular details of these findings.

### REFERENCES

- [1] Lai H, Singh NP. Bioelectromagnetics 1995; 16: 207-210.
- [2] Lai H, Singh NP. Bioelectromagnetics 1997; 18: 156-165.
- [3] Hamblin DL, Wood AW. Int J Radiat Biol 2002; 78: 659-669.
- [4] Faruk Oktema, Fehmi Ozgunerb, Hakan Mollaoglu, Ahmet Koyub et al. Biophysical 2005; 113(3): 245-253.
- [5] Ellman GL. Arch Biochem Biophys 1959; 82:70-7.
- [6] Nourooz-zadeh J. Methods in enzymology 300. California: Academic Press; 1999:58-62.
- [7] Benzie IF, Strain JJ. Analytical Biochemistry 1996; 239:70-76.
- [8] Atilla Ilhan S, Durmus Ali A Ferah, Ahmet G and OmerA. J Neurol Sci Turki 2004; 21:255-262.
- [9] Lowrey OH, Rosebraugh NJ, Farr AL and RandallRJ. J Biol Chem 1951;183:265-275.
- [10] Hallwell B. Lancet 1994; 344:721-724.
- [11] Hallwell B, Gutteridge JMC. In free radicals in biology and medicine, 2nd edition 1989; Oxford University, UK.
- [12] Jaite J, Grzegorzcyk J, Zmyslony M, Rajkowska E. Bioelectrochemistry 2002;57(2):107-111.
- [13] Gutteridge JMN, Hallwell B. TIBS 1990:129-35.
- [14] Hamblin DL, Wood AW. Int J Radiat Biol 2002; 78:659-669.
- [15] Grigor'evlug. Radiat Biol Radioecol 2003; 43:541-543.
- [16] Heynick LN, Johnston SA and Mason PA. Bioelectromagnetics 2003; 6:574-100.
- [17] Jirillo E, Altamura M, Casale D, Pepe T, Venezia FP, Soleo L, L'Abbate N, Raino A and Stefanelli R. G Ital Med Lav Ergon 2003;25:369-370.
- [18] La Regina M, Moros EG, Pickard WF, Stranbe WL, Baty J and Roti Roti JLO. Radiat Res 2003; 160:143-151.
- [19] Krause CM, Harrala C, Sillanmaki L, Koivisto M, Alnko K, Reyonsuo A, Laine M and Hamalianen H. Bioelectromagnetics 2004; 25:33-40.