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Full Length Research Paper

Microwave radiation exposures affect the ldl, hdl, tcl and trg status in rats

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Effects of Microwave (MW) radiation exposures on LDL, HDL, TCL and TGR have been studied as a means for assessment of health impacts. Exposures caused increase in LDL (354.2%), reduction in HDL (18.6%), increase in TCL (68.4%) and decrease in TGR (31.4%) within the first 24 h. The modifying roles of administered anti-oxidants were evaluated. Vitamins C and E reduced the effects of MW exposures on LDL by 35.4 and 37.7% respectively, on HDL by 50.0 and 40.5% respectively, on TCL by 36.8 and 26.4% respectively and on TGR by 35.7 and 48.5%, respectively. Results showed MW exposure health risks include atherosclerosis and acute coronary syndrome while diets fortified with vitamins C and E are recommended for persons working within MW fields in order to minimize the associated MW exposure health hazards.

Key words: Microwave, radiation, rats, health hazards.

INTRODUCTION

The use of microwave (MW) radiation is fundamental not only in modern communications systems such as mobile telephones, but also in Nuclear Magnetic Resonance Diagnostic Imaging and Hyperthermia and Thermal Ablation Therapeutic techniques. The proliferation of MW applications has generated concerns about the safe use (Valberg et al., 2007) due to the suspected health hazards associated with exposures. These hazards led some relevant National and International organizations to establish guidelines and legislations and to determine exposure limits for safe practice (ICNIRP, 1998; ICNIRP, 2004; ARPANSA, 2002; IEC, 2002; NRPB, 2004; IEEE, 2005; Kanal, 2007; EMF, 2007; HPA, 2008). These guidelines were derived from the results of various studies on internal electric fields, currents and MW energy deposition within the body tissues during exposures. The molecular phenomena involved in the conversion electromagnetic (e-m) energy to heat and the biological implications have been extensively reviewed in the literature (Schwan and Foster, 1980; Stuckly, 1979; McRee and Davies, 1987). MW interactions occur through the stimulation of the excitable membranes of nerve and muscle cells. Those associated with heating lead to perturbation in biochemical reactions, reaction rates, current flow and destruction of cell membranes (Forster, 1996; Pichard and Rusanbaum, (1978); Barsoun and Pickard, 1982), thereby producing some observable and measurable physiological parameters. Recent studies have shown that exposure to MW radiation produced effects which include activation of peritoneal macrophages to a viricidal state (Barsoun and Pickard, 1982), increased immune response (Rao et al., 1983), modification of the lipid peroxidation conditions (Veyret, 1991) and several others (Aweda et al., 2002). Many of these effects are caused by free oxygen, free radicals, peroxides and superoxides. Free radicals promote oxidation of amino acid residue side chains, formation of protein-protein cross-linkages (e.g. sulfhydryl mediated) and oxidation of the protein backbone resulting in fragmentation and aggregation (Fesenko et al., 1999). Oxidative modification enhances degradation of critical enzymes by the multi - catalytic proteasome complex

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Abbreviations: MW, Microwave; **LDL,** low density lipoprotein; **HDL,** high density lipoprotein; **ROS,** reactive oxygen species; **TGR,** triglyceride; **TCL,** total cholesterol.

(Azinge et al., 2001), thereby raising havoc throughout the cell.

The rapidly increasing and numerous industrial, medical and research applications of MW radiations cause significant increase in human and environmental exposures. Exposures could result from the practice of profession, type of occupation or domestic and industrial uses of devices and equipment using or generating MW radiations. This paper reports the study of the influence of MW radiation exposures on the Low Density Lipoprotein (LDL), High Density Lipoprotein (HDL), Total Cholesterol Lipoprotein (TCL) and Triglyceride (TRG) levels.

Ascorbic acid, a water-soluble vitamin C plays essential metabolic roles *in vivo* (Retsky et al., 1993), it is a good scavenger of reactive oxygen species (Igweh et al., 2003) and it also helps to recycle α-tocopherol *in vivo*. Vitamin E is a major membrane bound antioxidant present in the lipid core of cell membrane and lipoprotein where it protects polyunsaturated fatty free radical-mediated peroxidation. It is also essential along with cholesterol, for the structural stability of the membrane. Because the physiological parameters in this study are due to oxidative processes, the effects of administering these anti-oxidants to MW exposed organisms were studied to determine their eventual modifying role. The results of this study will be essential in the assessment of the health impacts of environmental, professional and occupational exposures and in developing strategies of minimizing the hazards.

MATERIALS AND METHODS

The source of MW used was generator the model ER6660E, Serial No. 2 XC 21744 from Toshiba UK Ltd. The detector used was the non-interactive thermistor RS 141, which has a resistance of 4.7 kΩ at 25°C. The thermistor was calibrated in a 0.12 × 0.06 × 0.04 m^3 size water phantom with the aid of standard mercury-in-glass laboratory thermometer as reference. Details of the system calibration and the determination of the Specific Absorption Rates (SAR) of the experimental animals have been described elsewhere (Aweda et al., 2002). The MW generator was operated at room temperature of 25 ± 2°C and 56 ± 4% relative humidity.

Animal preparation

200 Wistar rats fed with normal rat feeds (rat chou) and given water *ad libitum* under the normal laboratory conditions specified above and kept in plastic cages of 0.05 × 0.10 × 0.25 m^3 size. The rats were grouped in fives, 10 groups of five served as control at various times. Another 10 groups of five were exposed to MW without administration of anti-oxidants. 10 groups were treated with ascorbic acid (vitamin C) at 1 $mgkg^{-1}$ body weight, while another 10 other groups were treated with α-tocopherol (vitamin E), also at 1 $mgkg^{-1}$ body weight continuously for 4 days before exposures in both cases. All, except the control groups were subjected to MW irradiation using a central maximum power density of 60 Wm^{-2}.

Determination of SAR

The animals were irradiated from the open side of the rectangular horn–antenna with a 0.12 × 0.05 m^2 apartment from the distance of 0.12 m to the animal. The field intensity at the animal location was measured with the aid of a pre-calibrated thermistor probe. SAR values were determined by anal insertion of the thermistor probe connected to a read out meter. The animals were allowed free movement during exposures. The local variation of incident power density was measured in the presence and absence of the animal. This variation was within ± 3%. The variations in the studied physiologic parameters were monitored for 8 weeks in both exposed animals and the control.

Biochemical assay

The reagents used were obtained from BIOLABO, France. The HDL and TLC concentrations in the blood serum were determined using the method of Allain and Poon (1974). The LDL and TRG concentrations were determined following the methods described by Grove (Grove, 1998) and Esders and Michrina (1979).

RESULTS

LDL

Figure 1 presents the variations in the LDL status measured over a period of 8 weeks post MW exposures compared with control. The variations in the control group throughout the monitoring period were small, with a mean value of 0.0251 ± 0.0019 × 10^{-6} kg l^{-1}. Immediately after irradiation, the value of the LDL increased from 0.0220 ± 0.0024 × 10^{-6} kg l^{-1} to 0.0735 ± 0.0016 × 10^{-6} kg l^{-1} (234.1%). The value further increased to 0.1140 ± 0.0023 × 10^{-6} kg l^{-1} (354.2%) within the first 24 h and then decreased gradually to 0.0213 ± 0.0008 × 10^{-6} kg l^{-1} after two weeks. Thereafter, they varied between 0.0333 ± 0.0017 and 0.0298 ± 0.0019 × 10^{-6} kg l^{-1}. In the groups administered with vitamins C and E, the LDL values increased from 0.0220 ± 0.0024 × 10^{-6} kg l^{-1} to attain 0.0678 ± 0.0046 (208.2%) and 0.0560 ± 0.0007 × 10^{-6} kg l^{-1} (154.5%) respectively. Within the first 24 h, these values rose to 0.0825 ± 0.0004 (228.7.0%) and 0.0805 ± 0.0010 × 10^{-6} kg l^{-1} (220.7%) respectively, then decreased to normal values after two to three weeks post irradiation. Vitamins C and E caused decreases in the value as much as 273.8 and 253.0% within the first week of exposures respectively.

HDL

Figure 2 shows the variations in the HDL status over the same period, comparing the variations in the exposed groups without and with vitamins C and E. The variations in the HDL values in the control group were again small, having a mean value of 0.0299 ± 0.0013 × 10^{-6} kg l^{-1}. Immediately after MW radiation exposures, the value decreased from 0.0280 ± 0.0016 × 10^{-6} kg l^{-1} to 0.0228 ± 0.0016 × 10^{-6} kg l^{-1} (18.6%). It then increased to 0.0290 ± 0.0009 × 10^{-6} kg l^{-1} (0.7%) within the first 24 h and then further decreased and varied between 0.0180 ± 0.0003

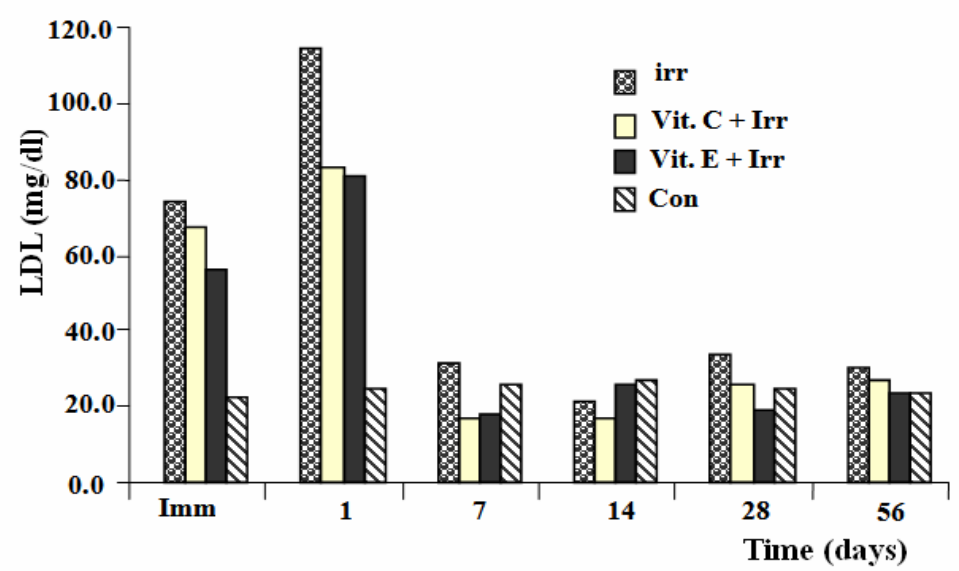

Figure 1. Variation of LDL with time with and without vitamins C and E.

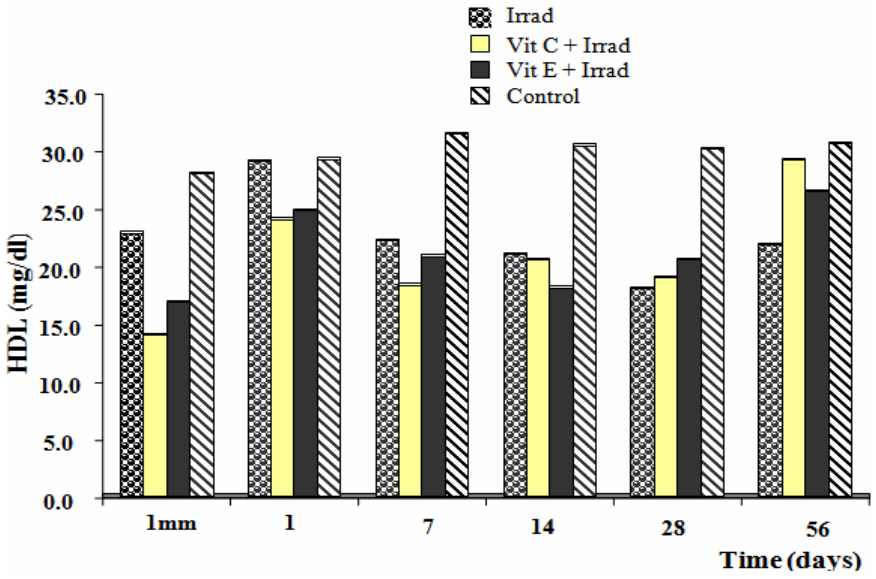

Figure 2. Variation of high density lipoprotein cholesterol (HDL) after MW exposure and administration of vitamins C and E.

and 0.0222 ± 0.0019 × 10^{-6} kg l^{-1} throughout the rest study period. The HDL values in the groups administered with vitamins C and E decreased from 0.0280 ± 0.0016 to 0.0140 ± 0.0006 (50.0%) and 0.0168 ± 0.0010 × 10^{-6} kg l^{-1} (40.0%) respectively. These values later rose to 0.0248 ± 0.0004 (15.1%) and 0.0248 ± 0.0014 × 10^{-6} kg l^{-1} (15.1%) each after the first 24 h. At the end of the 8th week the values attained were 0.0291 ± 0.0006 and 0.0264 ± 0.0010 × 10^{-6} kg l^{-1} respectively. Vitamins C and E caused decreases in the value as much as 50.0% immediately after exposure and 40.5% during the second week respectively.

TCL

In Figure 3 are shown the variations of the TCL status measured before and after MW exposures. The variations in the control group over the monitoring period were small, having a mean value of 0.0852 ± 0.0027 ×

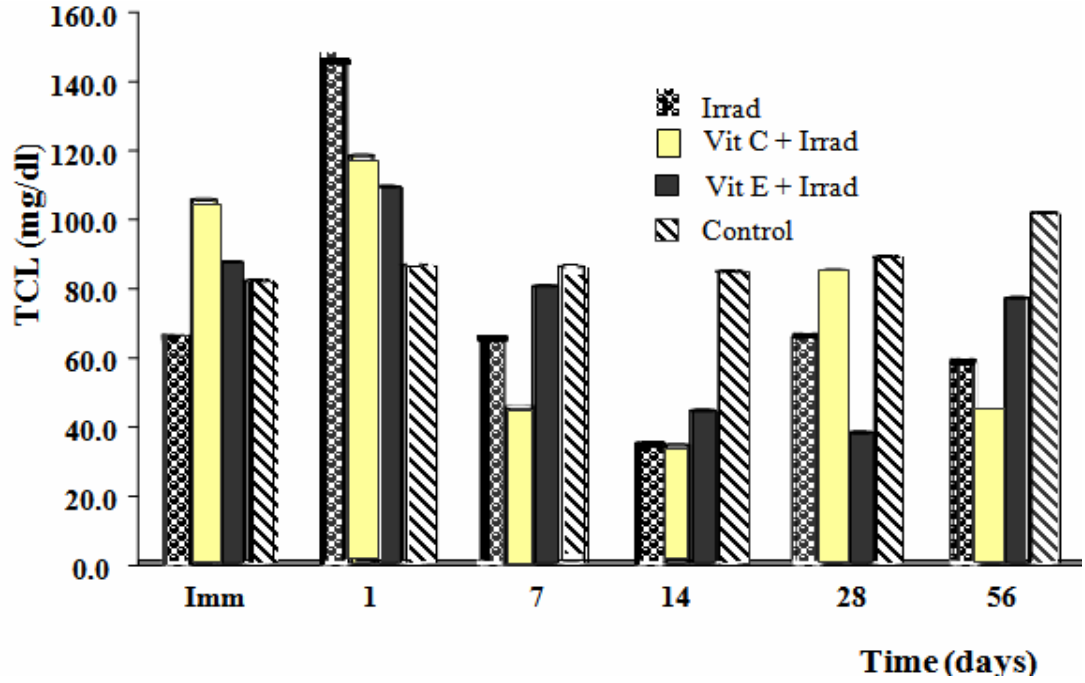

Figure 3. Variation of total cholesterol lipoprotein (TCL) after exposure and administration of vitamins C and E.

10^{-6} kg l^{-1}. Immediately after irradiation, the value of the TLC decreased from 0.0815 ± 0.0024 × 10^{-6} kg l^{-1} to 0.0650 ± 0.0020 × 10^{-6} kg l^{-1} (20.2%). It then increased rapidly to 0.1450 ± 0.0020 × 10^{-6} kg l^{-1} (68.4%) within the first 24 h and again fell gradually to attain a minimum of 0.0340 ± 0.0024 × 10^{-6} kg l^{-1} after two week. The values then increased gradually to 0.0581 ± 0.0023 ± × 10^{-6} kg l^{-1} at the end of the 8th week. There were increases in the values with the groups administered with anti-oxidants. The values increased to 0.1050 ± 0.0026 (28.8%) and 0.0870 ± 0.0015 × 10^{-6} kg l^{-1} (6.7%) for vitamins C and E respectively immediately after irradiation. These values increased to 0.1178 ± 0.0020 (36.8%) and 0.1088 ± 0.0028 × 10^{-6} kg l^{-1} (26.4%) respectively within the first 24 h and then decreased rapidly to 0.0450 ± 0.0015 and 0.0800 ± 0.0015 × 10^{-6} kg l^{-1} respectively after a week. Thereafter, they varied between 0.0343 ± 0.0024 and 0.0845 ± 0.0021 × 10^{-6} kg l^{-1} with vitamin C and between 0.0376 ± 0.0026 and 0.0766 ± 0.0022 × 10^{-6} kg l^{-1} with vitamin E for the rest study period. Vitamins C and E caused decreases in the value as much as 59.6 and 80.1% within the second and fourth weeks respectively.

TRG

Figure 4 shows the TRG variations with a mean value in the control group as 0.0577 ± 0.0012 × 10^{-6} kg l^{-1}. In the group without anti-oxidant, the value decreased from 0.0591 ± 0.0008 × 10^{-6} kg l^{-1} to 0.0460 ± 0.0023 × 10^{-6} kg l^{-1} (22.2%) immediately after exposure. The value further decreased to 0.0400 ± 0.0004 × 10^{-6} kg l^{-1} (31.4%) within the first 24 h. The value again decreased to attain a minimum of 0.0326 ± 0.0021 × 10^{-6} kg l^{-1} at the end of two weeks. This was then followed by a gentle rise through 0.0445 ± 0.0017 × 10^{-6} kg l^{-1} at the 4th week to 0.0485 ± 0.0016 × 10^{-6} kg l^{-1} at the 8th week. In the groups administered with vitamins C and E, the TRG values immediately decreased to 0.0380 ± 0.0005 × 10^{-6} kg l^{-1} (35.7%) in each case. Within the first 24 h, the values became 0.0500 ± 0.0004 (14.2%) and 0.0300 ± 0.0004 × 10^{-6} kg l^{-1} (48.5%) with vitamins C and E respectively. The values then decreased to a minimum of 0.0323 ± 0.00008 × 10^{-6} kg l^{-1} after a week with vitamin C and to 0.0300 ± 0.0016 × 10^{-6} kg l^{-1} after two weeks with vitamin E. At the end of the 8th week, the values became 0.0528 ± 0.0010 and 0.0500 ± 0.0020 × 10^{-6} g l^{-1}, respectively. Vitamins C and E caused decreases as low as 46.0 and 50.2% respectively during the second week.

DISCUSSION

Significant differences in the physiologic parameters were observed in the MW exposed animals throughout the 8 week study period. The LDL value increase in irradiated rats was as high as 354% due to effects of MW exposures after a day compared with control. Extreme LDL elevations are commonly associated with primary or genetic hyperlipidemias (McCrindle, 2000). In children and

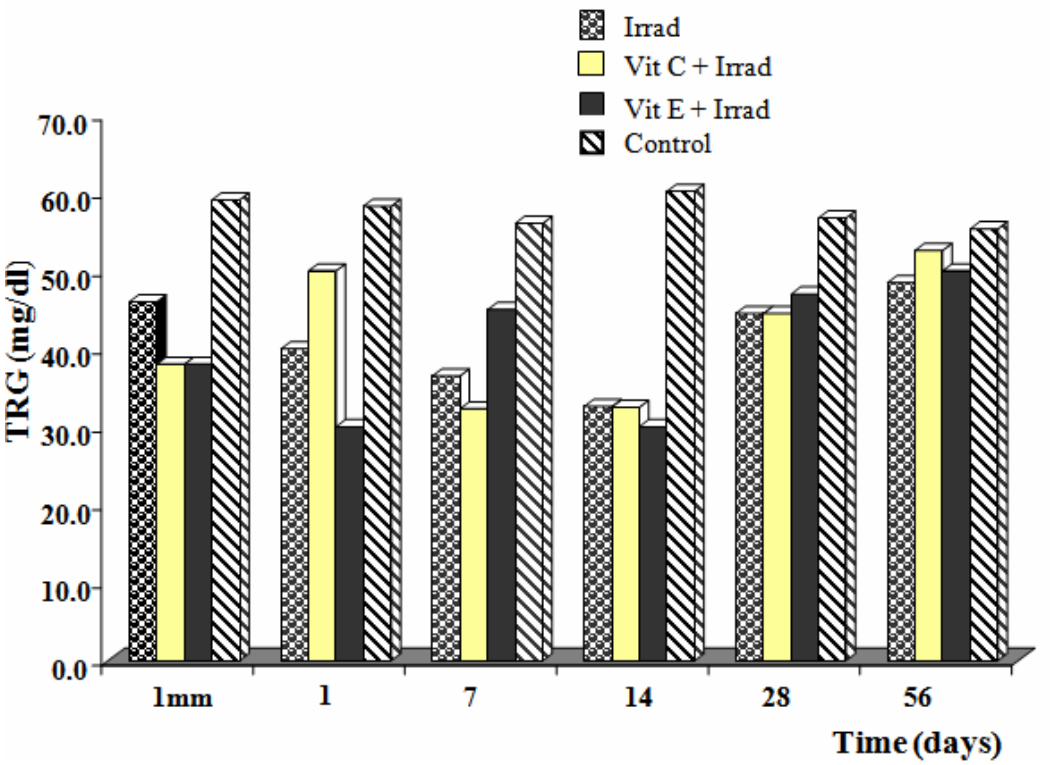

Figure 4. Variation of triglyceride status after mw exposure and administration of vitamins C and E.

adolescents. Hyperlipidemia may generally be as a result of either obesity or effect of medications. Lipid abnormalities have been reported to be generally associated with vascular pathology (Weijenberg et al., 2009). Effective lipid-lowering therapy has been shown to improve these abnormalities by several authors (DeJongh et al., 2002; Wiegman et al., 2004) including the reports of the National Cholesterol Education Programme (Chan and Watts, 2006; Ballantyne et al., 2005; NCEP, 2002). Of utmost clinical importance are the modified forms of LDL, mainly oxidized LDL, LDL sub-fractions, primarily LDL-III or small dense LDL which are products of intravascular remodelling of triglyceride-rich lipoproteins (Rizzo and Berneis, 2006). Effective management of hyperlipidemia aimed toward lowering LDL cholesterol levels, can lead to significant reduction in cardiovascular-related morbidity and mortality. LDL is the primary plasma lipid carrier with a single apolipoprotein molecule B-100 per particle. Apolipoprotein B-100 is a 500 kD peptide chain, one of the largest known monomeric proteins, highly insoluble in aqueous environment, which in contrary to other apolipoprotein is not substitutable by other lipoprotein particles. It is of hepatic origin, reaching the circulatory system via the very-low-density lipoproteins, which constitute LDL through the process of gradual intravascular remodelling. LDL constitutes highly atherogenic particles that, dominantly over other lipoproteins, favour cholesterol accumulation in foam cells produced by macrophages. Numerous epidemiological and clinical studies have consistently demonstrated the causal relationship between LDL cholesterol and atherosclerosis. The elevated status of LDL contributes to accelerated atherosclerosis, with demonstration of cardiovascular diseases within the first 2 decades of life for homozygotes (Al-Shaikh et al., 2002), and beginning in early to mid-adulthood for heterozygotes (Umans-Eckenhausen et al., 2001). The significant increases in LDL status due to MW radiation exposures is an indication of potential health risks that may be associated with exposures. Administration of vitamins C and E, vitamins well known powerful antioxidants caused decreases in the values of LDL by as much as 273.8 and 253.0% within the first week of exposures respectively. The biophysical processes of the MW radiation interaction processes are yet unclear. The results obtained however demonstrated that oxidation reactions may have been involved, leading to production of radicals, peroxides, superoxides etc. This speculation was confirmed by the effects of the introduced vitamins which are scavengers of these radicals. The results agree with earlier reports that MW exposures affect the peroxidation status in mammals (Aweda et al., 2002).

The MW radiation exposure produced to 18.6% reduction in the level of plasma HDL immediately after exposure as compared with control (Figure 2). The level was further lowered to about 51 and 46%, respectively by

the administration of vitamins C and E respectively. The corresponding p-value shows a significant effect of the MW exposure on the HDL status ($p < 0.001$). Administration of antioxidants, vitamins C and E further enhances this effect, thus suggesting a stimulatory effect on the enzymatic action on the plasma HDL. HDL particles are composed of an outer amphipathic layer of free cholesterol, phospholipid and several apolipoproteins and of a triglyceride and cholesterol ester hydrophobic core. The particles also carry enzymes, such as paraoxonase, platelet activating factor acetylhydrolase, lecithin, cholesterol acyltransferase, and cholesteryl ester transfer protein (Von, 2004; Chapman, 2006). Cholesterol concentration in HDL is an inverse predictor of future atherosclerotic cardiovascular disease. Experimental evidences have shown that increasing the status and/or function of HDL can have strong vascular protective effects, ranging from prevention to stabilization and regression, independent of total or non-high-density lipoprotein cholesterol (Chan and Watts, 2006; Ballantyne et al., 2005; NCEP, 2002; Chapman 2006; Ohashi et al., 2005). HDL accomplishes its basic anti-atherogenic function by reducing cholesterol accumulation in the artery walls, up-taking it and transporting it back to the liver for elimination from the body.

TCL

As seen in Figure 3, MW exposures modify the metabolism of total cholesterol in the blood plasma quite significantly as shown by the p-value ($p < 0.005$). This is to be expected, since both the LDL and HDL levels were significantly affected by exposure and as well by the administration of the antioxidants.

TRG

MW exposures affected the triglyceride status significantly. Also the vitamins C and E supplementation produced further enhancement of this effect. The corresponding p-values lends credence to this fact which again is consistent with the report by Lee et al. (Lee et al., 2002), that antioxidants provide a protective effect on oxidative stress as may be induced by exercise and as in the case in this study, MW exposures.

Ionizing radiations are known to produce free oxygen, free radicals, peroxides and superoxides when biological tissues are exposed to them and these contribute immensely to cell damage, a phenomenon applied in radiotherapeutic management of malignancies based on the indirect effects of ionizing radiation (IAEAVA, 2003). Oxidative stress is produced by the peroxidation and oxidation of many cell lipids, proteins, carbohydrates, and nucleic acids. The detailed chemistry of oxygen radical generation and the countervailing effect of oxygen radical scavengers have been reviewed (Pryor et al., 2006; Szabo et al., 2007).

Reactive oxygen species (ROS) are very small molecules that include oxygen ions, free radicals and peroxides, both inorganic and organic that are highly reactive due to their unpaired valence electrons. Due to environmental stress resulting from MW, ionizing radiations and heat exposures, ROS levels can increase dramatically, and this may result in significant damage to cell structures. ROS can damage DNA, RNA and proteins which theoretically contributes to the physiology of aging, oxidations of poly-desaturated fatty acids in lipids, oxidation of amino acids in proteins and inactivate specific enzymes by oxidation of co-factors. While ROS are products of normal cellular functioning, excessive amounts can cause deleterious effects (Marian et al., 2007).

Antioxidants vitamins C and E act by donating an electron to a free radical and converting it to a non-radical form. Such process can terminate radical chain reactions and reduce hydroperoxides and epoxides to less reactive derivatives. Vitamin E has been proposed and later rejected as a potential method of protection against pulmonary oxygen toxicity (Terry and Lysiak, 2008; Aweda et al., 2004; Mazor et al., 2008). There is however some experimental evidence that it aides in preventing *in vivo* lipid peroxidation and free radical damage and therefore prevent retinal changes (Joubert et al., 2008). It is possible that more than one type of interactions of electron transfer agents in biological tissues take place *in vivo*. For example, the dyskinesia associated with Wilson's disease, which occurs at the stage in which there is no visible brain damage, probably caused by the affinity of copper for brain melanin. Later damage to the basal ganglia and to liver may be the result of copper-catalyzed peroxidation of membrane lipids. Numerous other indirect processes are possible such as disruption of lysosomal function by altering ascorbate metabolism, degradation of DNA by cysteine or by diphenols (Schatte (1977), or melanin-related alterations in pineal function. Cooperative interactions, such as a transition-series metal operating in conjunction with a purine to produce a psychoactive protein-free radical, are also possible (Peter (1972).

Conclusion

MW interactions in biological tissues lead to production of ROS which cause oxidative reactions that eventually affect the status of the physiological parameters LDL, HDL, TCL and TGR. Consequences of the changes in the LDL, HDL TCL and TGR status on health include atherosclerosis by LDL, induced cytotoxicity and its inhibition by HDL in human vascular smooth muscle and endothelial cells in culture etc (Hessler and Robertson, 1997; Zhang et al., 1989). The effects of the radicals on

DNA and RNA may possibly produce delayed effects such as malignancies. The possible health impacts of MW exposures are no more news as a lot of works using experimental animals, human experiences and epidemiological studies have been published. With the ever increasing applications of MW radiations, public exposures will continue to increase proportionately, hence the need to be vigilant and make concerted efforts to minimize the possible health impacts. Workers in MW industries, Telecommunications and allied industries, Physiotherapeutic, Radiotherapeutic users and other Medical equipment operators and researchers need to be cautious and possibly, should be monitored for MW exposures to ensure they do not exceed the recommended annual SAR limits, a practice similar to personal dosimetry of workers and users of ionizing radiations.

The modifying role of ascorbic acid and α-tocopherol on the effects of LDL, HDL, TCL and TGR as obtained from this study suggests that their administration could cushion the health detriments of MW exposures. Dietary habits rich in these anti-oxidants will be of much assistance to regular and professional MW workers and users in addition to the adoption of exposure optimization principles of distance, time and SAR limitation, similar to what applies to ionizing radiation workers and users (IAEA, 2002).

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REFERENCES

National Radiological Protection Board (NRPB) (2004). Advice on limiting exposure to electromagnetic fields (0–300 GHz); Doc. NRPB 15: 1-35.

Allain CC, Poon LS (1974). Cholesterol estimation. Clin. Chem., 20: 470-470.

Al-Shaikh AM, Abdullah MH, Barclay A, Cullen-Dean G, McCrindle BW (2002). Impact of the characteristics of patients and their clinical management on outcomes in children with homozygous familial hypercholesterolemia. Cardiol. Young., 12: 105-112.

Aweda MA, Gbenebitse S, Meindinyo RO (2002). Effects of 2.45 GHz MW exposures on the peroxidation status in Wistar rats. Nat'l. Postgrad. Med. J., 10(4):243-246.

Aweda MA, Gbenebitse S, Meindinyo RO (2004). Effects of 2.45 GHz Radiofrequency Exposures on Normal and Sickle Cell Erythrocytes. Nig. J. Health Biomed. Sci., 3(1): 56-59.

Azinge EC, Mabayoje M, Sofola OA (2001). LDL peroxidation and total antioxidant status in Nigerian patients with sickle-cell disease. Nig. J Hosp. Med., 11(1-4): 46-50.

Ballantyne C, Arroll B, Shepherd J (2005). Lipids and CVD management: towards a global consensus. Eur. Heart J., 26(21): 2224-2231.

Barsoun YH, Pickard WF (1982). Effects of em radiation in the range 40-440 MHz on the vascular potential of Characean cells. Bioelectromagnetics, 3: 193-201.

Barsoun YH, Pickard WF (1982). The vascular potential of Characean cells subjected to em radiation in the range 200-8200 MHz. Bioelectromagnetics, 3: 393-400.

Chan DC, Watts GF (2006). Apolipoproteins as markers and managers of coronary risk. QJM. 99(5): 277-878.

Chapman MJ (2006). Therapeutic elevation of HDL-cholesterol to prevent atherosclerosis and coronary heart disease. J. pharmthera., 02:003.

DeJongh S, Lilien MR, op't Roodt J, Stroes ES, Bakker HD, Kastelein JJ (2002). Early statin therapy restores endothelial function in children with familial hypercholesterolemia. J. Am. Coll. Cardiol., 40: 2117-2121.

Esders TW, Michrina CA (1979). Triglyceride estimation. J. Biol. Chem., 254: 2710-2710.

Fesenko EE, Maker VP, Novoselova EG, Sadovnikov VB (1999). Microwaves and cellular immunity I: Effects of wholebody MW irradiation on tumour necrosis factor production in mouse cells. Bioelectrochem. Bioenergetics, 49: 29-35.

Forster KR (1996). Electromagnetic Field Effects and Mechanisms. IEEE Eng. Med. Biol., 26(6): 50-54.

Grove TH (1998). HDL Assay, Clin. Chem., 25: 560-650.

Guidelines for limiting exposure to time-varying electric, magnetic, and electromagnetic fields (up to 300 GHz). International Commission on Non-Ionizing Radiation Protection (ICNIRP); Health Phys., **7**4: 494-522 (1998).

Hessler JR, Robertson AL (Jr.), Chisolm GM (1997). LDL-induced cytotoxicity and its inhibition by HDL in human vascular smooth muscle and endothelial cells in culture. Atherosclerosis, 32: 213-229.

IEEE Standard for Safety Levels with respect to Human Exposure to Radio Frequency Electromagnetic Fields, 3 kHz to 300 GHz. Institute of Electrical and Electronic Engineers. New York: IEEE Topical Review R282 (2005).

Igweh JC, Nwafia WC, Ejezie FE (2003). Human low density lipoprotein as a vehicle of atherosclerosis. Nig. J. Health Biomed. Sci. 2(1): 7-11.

International Atomic Energy Agency (IAEA) (2002). Occupational radiation protection: protecting workers against exposure to ionizing radiation. Proc. Intern'l Conf., Geneva, August, pp. 26-30.

Joubert V, Bourthoumieu S, Leveque P, Yardin C (2008). Apoptosis is Induced by Radiofrequency Fields through the Caspase-Independent Mitochondrial Pathway in Cortical Neurons. Radiat. Res., 169: 38-45.

Kanal E (2007). American College of Radiologists guidance document for safe MR practices. Am. J. Roentgenol., 188: 1–27.

Lee K, Park JS, Kim YJ, Soo-Lee YS, Sook-Hwang TS, Kim DJ, Park EM, Park YM (2002). Differential expression of Prx I and II in mouse testis and their up-regulation by radiation. Biochem Biophys. Res. Commun., 296: 337-342.

Marian VDL, Jan M, Mark TD, Cronin MM, Joshua T (2007). Free radicals and antioxidants in normal physiological functions and human disease. Int. J. Biochem. Cell Biol., 39(1): 44-84.

Mazor R, Korenstein-Ilan A, Barbul A, Eshet Y, Shahadi A, Jerby E, Korenstein R (2008). Increased Levels of Numerical Chromosome Aberrations after *In Vitro* Exposure of Human Peripheral Blood Lymphocytes to Radiofrequency Electromagnetic Fields for 72 Hours. Radiat. Res., 169: 28-37.

McCrindle BW (2000). Screening and management of hyperlipidemia in children. Pediatr. Ann., 29: 500-508.

McRee DT, Davies HG (1987). Effects of energy absorption, orientation and size of animals exposed to microwaves. Health Phys., 56(5): 39-43.

Medical Electrical Equipment: Part 2. Particular requirements for the Safety of Magnetic Resonance Equipment for Medical Diagnosis. International Electrotechnical Commission (IEC); Geneva: IEC Report (2002).

Medical magnetic resonance (MR) procedures: Protection of patients. International Commission on Non-Ionizing Radiation Protection (ICNIRP); Health Phys., 87:197-216 (2004).

National Cholesterol Education Program (NCEP) (2002). Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Circulation, 106: 3143-3421

Ohashi R, Mu H, Wang X, Yao Q, Chen C (2005). Reverse cholesterol transport and cholesterol efflux in atherosclerosis. QJM, 98(12): 845-856.

Peter P (1972). Electron-transfer factors in psychosis and dyskinesia.

Physiol Chem. Phys., 4: 349-360. http://www.drproctor.com/crcpap2.htm (downloaded 20th Nov. 2008).
Pichard WF, Rusanbaum FJ (1978) . Biological effects of MW radiation at membrane levels: Two possible athermal electrophysiological mechanisms and a proposed experimental test. Math. Biosci., 39: 235-253.
Possible effects of Electromagnetic Fields (EMF) on Human Health, Brussels: Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR): European Commission Health and Consumer Protection DG Report (2007).
Protection of Patients and Volunteers Undergoing MRI Procedures. Chilton: Health Protection Agency (HPA) Report (2008).
Pryor WA, Houk KN, Foote CS, Fukuto JM, Ignarro LJ, Squadrito GL, Davies KJA (2006). Free radical biology and medicine: it's a gas, man! Am J Physiol Regul Integr Comp Physiol., 291: R491-R511.
Radiation Oncology Physics (2003). (Edit.) Podgorsak E. B. International Atomic Energy Agency Vienna, Austria.
Radiation Protection Standard - Maximum Exposure Levels to Radiofrequency Fields – 3 kHz to 300 GHz. Australian Radiation Protection and Nuclear Safety Agency (ARPANSA). Victoria: ARPANSA Report (2002).
Rao RGV, Cain CA, Lockwood V, Tompkins WAF (1983). Effects of microwave exposure on hamsters immune system II: Pentoneal macrophage function. Bioelectromagnetics. 4: 141-155.
Retsky KL, Freeman MW, Frei B (1993). Ascorbic acid oxidation product(s) protect human low density lipoprotein pro-oxidant activity of vitamin C in the presence of transition metal ions. J. Biol. Chem., 268(2): 1304-1309.
Rizzo M, Berneis K (2006). Low-density lipoprotein size and cardiovascular risk assessment. QJM, 99(1): 1-14.
Schatte CL (1977). Dietary selenium and vitamin E as a possible prophylactic to pulmonary oxygen poisoning. Proc. Sixth Intern'l Congr. Hyperbaric Med, Scotland 84-91. Aberdeen University Press.
Schwan HP, Foster KR (1980). RF-fold interaction with biological systems. Proc. Inst. Elec. Electron. Engr., 68: 104-113.
Stuckly MA (1979). Interaction of radiofrequency and MW radiations with living systems: A review of mechanisms. Radiat. Environ. Biophys., 16: 1-14.
Szabo C, Ischiropoulus H, Radi R (2007). Peroxynitrite: biochemistry, pathophysiology and development of therapeutics. Nature Rev., 6: 662-679.
Terry TT, Jeffrey JL (2008). Oxidative stress: a common factor in testicular dysfunction. J. Androl., 1-17.
Umans-Eckenhausen MA, Defesche JC, Sijbrands EJ, Scheerder RLJM, Kastelein JJ (2001). Review of first 5 years of screening for familial hypercholesterolaemia in the Netherlands. Lancet, 357: 165-168.
Valberg PA, van Deventer E, Repacholi MH (2007). Workgroup report: base stations and wireless networks -radiofrequency (RF) exposures and health consequences. Environ. Health Perspect., 115: 416-424.
Veyret B (1991). Antibody response of mice to low-power MW under combined, pulsed and amplitude modulation. Bioelectromagnetics, 87: 375-380.
Von EA (2004). Therapeutic approaches for the modification of high-density lipoproteins. Drug Discov Today. Therap Strat., 1(2): 177-87.
Weijenberg MP, Feskens EJ, Kromhout D (2009). Total and high density lipoprotein cholesterol as risk factors for coronary heart disease in elderly men during 5 years of follow-up. Circulation, 119: 1108-1115.
Wiegman A, Hutten BA, deGroot E, Rodenburg J, Bakker HD, Buller HR, Sijbrands EJ, Kastelein JJ (2004). Efficacy and safety of statin therapy in children with familial hypercholesterolemia: a randomized controlled trial. JAMA, 292: 331-337.
Zhang H, Davis WG, Chen X, Whisler RL, Cornwell DG (1989). Studies on low density lipoprotein: Controlled oxidation and prostaglandin artifact. J. Lipid Res., 30: 141-148.