Available online at www.pelagiaresearchlibrary.com

Pelagia Research Library

Advances in Applied Science Research, 2011, 2 (2): 246-251

ISSN: 0976-8610
CODEN (USA): AASRFC

Microwave radiation exposures affect cardiovascular system and antioxidants modify the effects

[#1]M. A.Aweda, [1]R. O. K. Meindinyo, [2] S. O. Gbenebitse and [1] A. Z. Ibitoye

[1]Department of Radiation Biology and Radiotherapy, College of Medicine/Lagos University Teaching Hospital, Idi-Araba, P.M.B. 12003, Lagos, Nigeria.
[2]Department of Physiology, College of Medicine/Lagos University Teaching Hospital, Idi-Araba, P.M.B. 12003, Lagos, Nigeria.

ABSTRACT

Effects of 2.45 GHz Microwaves (MW) exposures on the blood pressure and heart pulse rate (PR) in Wistar rats have been studied using a non-invasive technique with a polygraph blood pressure (BP) recorder and MW radiation generator model ER6660E from Toshiba UK Ltd. BP and PR in rats were monitored for a period of 8 weeks post-irradiation. The MW exposures caused an increases in the values of parameters from the normal mean of 123.0 ± 1.2 mmHg and 430 ± 2.0 beats per minute (BPM) to a maximum of 145.0 ± 5.0 mmHg and 480.0 ± 6.8 BPM within the first 2 h, and then gradually reduced to normal values after about a week. 4 mgkg^{-1} body weight of ascorbic acid administered 4 days pre-irradiation caused a decrease in the values of these parameters to a minimum of 128.0 ± 0.4 mmHg and 440.0 ± 1.8 BPM within the first day. The results showed that MW exposures cause significant increase in the BP and PR. They also demonstrate the protective effect of the administered anti-oxidants, vitamins C and E. The changes in the BP and PR are attributed to N_2O_3 inhibition by free radicals produced by the MW interactions while the antioxidant may have suppressed these harmful effects by scavenging some of the free radicals.

Key words: Microwave radiation, Blood Pressure, Pulse Rate, Vitamin C, Vitamin E.

INTRODUCTION

Microwaves (MW) are non-ionizing electromagnetic radiations whose interactions with matter usually cause atomic excitation, molecular vibration and rotation as well as heat production [1]. The interactions of MW in living systems have been reported to cause among others, enzyme inactivation, cell damage, lipid peroxidation, DNA single strand breaks and oxidative stress [2-

11]. In particular MW exposures have been associated with changes in the electrocardiogram (ECG) and electromyogram (EMG), increases in peripheral vascular resistance and hypertension [12-13]. The effects of MW exposures have been categorized into three, viz, athermal, thermal and non-heating effects. The thermal and non-heating are effects identified with temperature changes and heat balance not attributable to the direct conversion of MW energy to heat in tissue. At power levels between 5 $mWcm^{-2}$ and 10 $mWcm^{-2}$, the energy transformed into heat in tissue is nearly equal to the heat loss per square centimeter of the body surface in humans and warm-blooded animals under normal environmental conditions. Hence at such power levels, MW interactions are classified as non-heating although thermal mechanisms may be involved. These interactions may actually depend on the characteristics of the radiation, the environment and the biological receptor. Also recent studies have indicated that interactions of MW with biological systems lead to increased free radicals production that may be responsible for most of the athermal harmful effects such as oxidative stress, lipid peroxidation and cell membrane damage [3,6].

Ascorbic acid is a water-soluble vitamin and likely the most efficient antioxidant in the cytosol of living cells. With vitamins A and E it forms the primary vitamin antioxidant in the body. The function of anti-oxidants is to scavenge free radicals that are produced in excess under certain conditions like hyperthermia, stress and vigorous exercise, in this study, as a result of MW radiation interactions. The consequences of excess free radical production have been shown to include increased membrane fluidity, compromised integrity and inactivation of membrane bound receptors and enzymes. Ascorbic acid has the ability to donate electrons, which makes it a potent water-soluble antioxidant [14]. It readily scavenges free radicals such as molecular oxygen, superoxide, hydroxyl radical and hypochlorous acid [15]. High doses of ascorbic acid induce vasodilation in the brachial and coronary arteries [16,17]. Salomen [18] had earlier reported that ascorbic acid supplementation reduced blood pressure in subjects by 7.5 mmHg. Vitamin C could improve vasodilation and vascular reactivity perhaps by decreasing the interaction of N_2O_3 with oxidants. N_2O_3 causes rapid relaxation of vascular smooth muscle [19].
This paper, which is one of our series of studies conducted on MW radiation safety and protection, reports the effects of MW radiation exposures on blood pressure (BP) and heart pulse rate (PR) using Wistar rats as animal model. The results hopefully will indicate the potential health risk in exposed individuals through cardiopulmonary complications from MW exposures.

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 ^{o}C. The thermistor and the entire MW generator system were calibrated in a 12 cm x 6 cm x 4 cm size water phantom with the aid of standard mercury-in-glass laboratory thermometer as reference. Details of the calibration of the system and the determination of the Specific Absorption Ratio (SAR) of the experimental animals have been described elsewhere [3]. The generator was operated at room temperature of 25 ± 2 ^{o}C and 56 ± 4 % relative humidity.

Studies were conducted with the aid of the Polygraph model Tail Cuff w + w BP Recorder model 8005 Ugo Basil from Biomedical Research Apparatus Comerio Va Italy available at the

Physiology Department of the College. This machine utilizes a non-invasive technique to automatically record the BP (mean arterial pressure) on a special electronically sensitive paper and the PR on a digital readout. The rats were fixed with a special device (restrainer), connected to the machine with leads. Each measurement was repeated thrice for determination of the mean and the SEM of arterial blood pressure and PR.

RESULTS AND DISCUSSION

Figure 1 presents the variation of BP measured over a period of 8 weeks after MW irradiation, compared with unexposed rats. The BP increased from 125.0 ± 1.3 to 145.0 ± 5.0 mmHg (117.2 % of control mean value) immediately after irradiation. The value decreased to 140.2 ± 4.2 mmHg (113.3 % of control mean) one week after and then decreased gradually to about the control value after two weeks. On the figure is also shown the variation in the exposed groups without and with vitamins C and E administration. The value of the BP is always less than those without the antioxidant throughout the monitoring period. Increase with vitamin C was by 113.2 % instead of 117.2 %, and was 114.0 % with vitamin E. Vitamins C and E caused the value to reduce from 140.0 ± 1.2 mmHg and 141.0 ± 0.5 mmHg immediately after exposure to 132.1 ± 0.8 mmHg and 128.0 ± 0.4 mmHg respectively a week after. The value further decreased to 124.0 ± 0. 8 mmHg and 120.0 ± 1.1 mmHg 2 weeks and 120.1 ± 0.8 mmHg and 121.0 ± 0.8 mmHg 4 weeks after irradiation respectively.

Figure 2 presents the variation of the PR measured over the same period after MW irradiation compared with the control. The MW exposure caused the PR to increase immediately after irradiation from 430.0 ± 2.6 for the control to 480.1 ± 2.4 BPM, (111.6 % of mean value). One day after irradiation, the value decreased to 460.22 ± 2.2 BPM (106.9 % of mean) and then further gradually decreased to about the control value 4 weeks after. The mean value within this period was 446.2 ± 1.6 BPM, which is 103.7 % the control mean value.

The figure also shows the variation in PR in the exposed groups of rats without and with vitamins C and E administration. Exposure causes a 106.2 % increase instead of 116.7 % with vitamin C and 105.1 % with vitamin E. Administration of vitamins C and E caused the value of the PR to decrease from 457.1 ± 1.8 to 439.1 ± 1.2 and 452.1 ±2.2 to 436.1 ±1.6 BPM respectively one week after. This was followed by a steady decrease to 430.2 ± 0.8 and 428.2 ± 0.4 BPM respectively at the 8^{th} week after exposure.

The interaction mechanisms of MW leading to the observed cardiovascular changes are yet not very clear. It may probably be as a result of nitric oxide inhibition, a well known vascular smooth muscle relaxant by products of lipid peroxidation and oxidative stress due to increased free radicals production from the MW exposure [3,20]. These results agree with the findings of Kalns et al. [21]. In their work, oxidative stress precedes circulation failure (CF) induced by 35 GHz MW heating. They hypothesized that oxidative stress might have played a role in the pathophysiology of MW induced CF. This oxidative stress has been identified to result from depletion of antioxidants which includes vitamin C, and excessive production of free radicals and other reactive oxygen species like OH°, O° and $H_2O_2^{\circ}$ resulting from MW-tissue interactions.

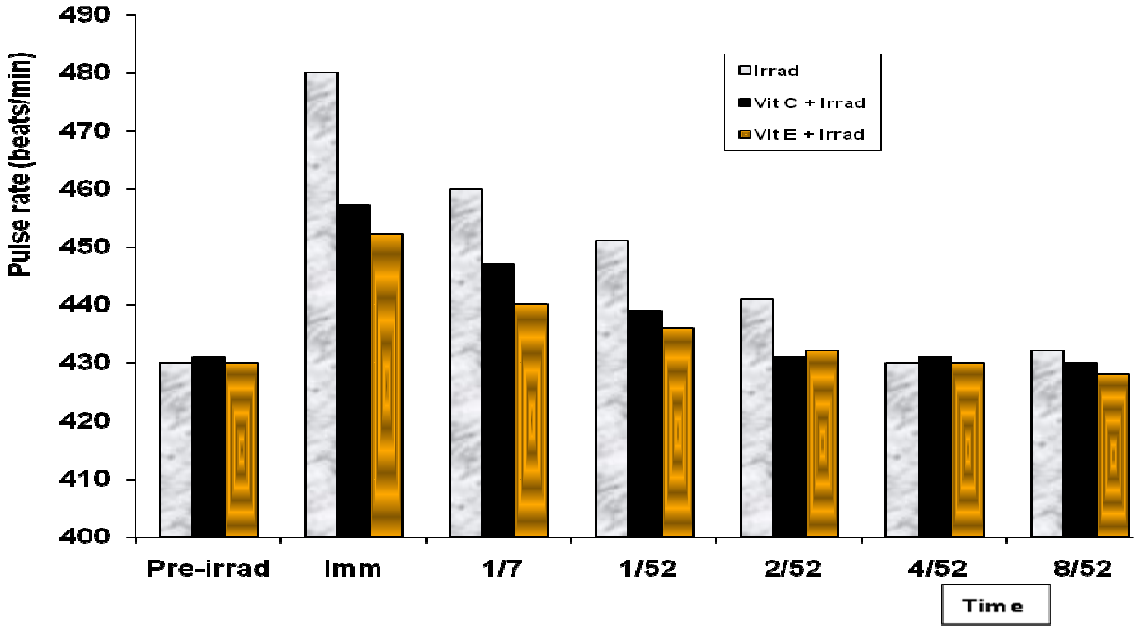

Fig. 1: Variation of pulse rates over a period of 8 weeks after MW exposure and administration of vitamins C and E

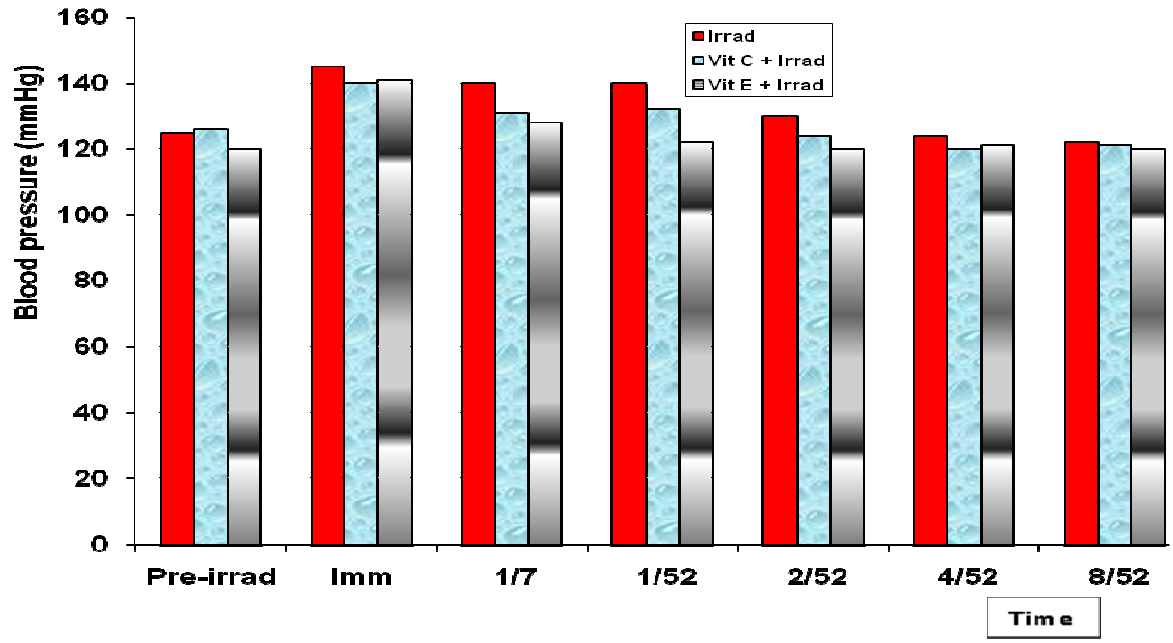

Fig. 2: Variation of blood pressures over a period of 8 weeks after MW exposure and administration of vitamin C and E

The results of earlier study showed that MW exposures lead to increased free radical production and lipid peroxidation. The products of lipid peroxidation, especially the oxidized low density

lipoprotein cholesterol are responsible for the nitric oxide inhibition, and hence the elevation of the BP and PR. The lower values in these parameters with the vitamins C and E treated groups, showed the strong scavenging action by the antioxidant on the MW induced excess free radicals.

CONCLUSION

The results of this study show that MW exposure has influence on the cardiovascular system; both the blood pressure and pulse rate were initially raised from 125 mmHg to 145 mmHg and from 430 BPM to 480 BPM respectively, immediately after exposure. They indicate 15 % and 12 % rise over the control values respectively. The results affirm that MW exposures affect the cardiovascular system in Wistar rats. This could be attributed to the heating effect, but more importantly increased free radical production. Administration of vitamins C and E cushioned the harmful effects of MW exposures on the cardiovascular system, showing protective effect on the harmful action of the MW radiation exposures. Implication of this observation is that persons habitually found or working in MW fields may fortify their diet with these antioxidants to reduce the potential health detriments.

REFERENCES

[1] M. A. Aweda. *J. Nig. Med. Rehab Therapists*, **2000,** 5, 15.
[2] B. Halliwell, J. M. C. Gutteridge. *Free Radicals in Medicine and Science*. Clarendon Press, Oxford, UK, **1989**.
[3] M. A. Aweda, R. O. K. Meindinyo, S. O. Gbenebitse. *The Nig. Postgrad Med. Journ.* **2002,** 10, 243.
[4] T. L. Broderick, R. W. Currie, D. J. Paulson. *Can J Physiol Pharmacol* **1997,** 75, 1273.
[5] H. W. Chen, C. Hsu. *Cell Stress Chaper-ones* **2000,** 5, 188.
[6] M. A. Aweda, S. O. Gbenebitse, M. O. Kehinde. *Nig. J. Health and Biomed Sces*. **2004**. 3, 1, 56.
[7] M. A. Aweda, M. R. Usikalu, O. O. Adeyemi, O. K. Yemitan. *Archiv Appl Science Res*, **2010**, 2, 37.
[8] M. A. Aweda, M. R. Usikalu, J. H. Wan, N. Ding, J. Y. Zhu. *Int'l J. Gen. Mol. Biol*. **2010**, 2,189
[9] M. A. Aweda, S. Gbenebitse, R. O. Meindinyo. *Int'l J.Phys. Sci.* **2010**, 5, 1015.
[10] M. A. Aweda, M. R. Usikalu, F. O. Awobajo. *Int'l J. Current Research* **2011**, 2, 120.
[11] M. R. Usikalu, M. A. Aweda, E. B.. Babatunde, F. O. Awobajo. *Am. J. Sci. Ind. Res*., **2010**, 1, 410.
[12] S. I. Jaja, S. I. Aisuodionwe, M. O. Kehinde, S. O. Ghenebitse. *The Nig. Postgrad Med. Journ.*, **2002,** 9, 92.
[13] R. O. K. Meindinyo, PhD thesis, University of Lagos, (Lagos, Nigeria **2005**)
[14] H. E. Sauberlich. In: Present Knowledge in Nutrition. Brown ML, (ed) Washington, DC. (International Life Sciences Institute, **1990**) 132.
[15] S. J. Padayatty, M. Levine. *Am. J. Clin. Nutr*. **2002**, 71, 1027.
[16] E. Bassenge, N. Fink, M. Skatchkov, B. Fink. *J. Clin. Invest.* **1998,** 102, 67.
[17] A. C. Carr, B. Frei. *Am. J. Clin. Nutr*. **1999,** 69, 1086.
[18] R. Salomen, H. Korpela, K. E. Nyyssonen, E. Porkkala, J. T. Salonen. *Life Chem. Rep.* **1994**, 12, 65.

[19] A. Alver, J. Collier P. Vallance. *Experimental Physiol.* **1993,** 78, 303.
[20] B. Halliwell. *Nutrition Review* **1994,** 52, 253.
[21] J. Kalns, K. R. Mason, J. G. R. Bruno, J. L. Kiel. *Shock* **2000**. 13, 52.