

Aluminum Foil Shield Diminishes the Electromagnetic Radiation of Mobile Phones in the Cerebellum of Adult Male Rats

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Abstract: Aluminum foil (AF), which is a good conductor and non-magnetic, is proposed to reflect almost all of the electromagnetic wave's exposure. The effect of aluminium foil to the malondialdehyde (MDA) levels of adult male rats' cerebellum exposed to the electromagnetic radiation (EMR) from the mobile phones was analyzed. Thirty-two adult male rats were placed equally into four groups (n=8). Group I is the control group without AF or EMR; group II is given the AF only; group III is treated to the EMR; group IV is treated to the EMR with AF shield. A mobile phone was taped at the inner floor of a plastic box container where the animal was placed. The AF shield has thickness of 2mm and the size of 20 x 16cm (Klin Pak, Indonesia) and covered the mobile phones wholly inside the plastic bag (in group IV). The mobile phone used has a specificity absorption rate Europe (SAR EU) of 0.84 - 1.86W/ kg with a frequency band of 2100MHz (GSM 2100 MHz). The exposure to the mobile phone's EMR was four hours daily for 30 days. The MDA levels were measured with the spectrophotometer (TBARS method) and analyzed statistically using *KruskalWallis Test* and *Mann Whitney U* ($p < 0.05$). The MDA levels in P2 were significantly lower compared to those of P1 ($p < 0.05$). Meanwhile, the MDA levels in P1 were significantly higher compared to the control group ($p < 0.05$). In conclusion, aluminum foil could provide protective effect against the exposure of EMR oxidative stress in rat cerebellum.

1 INTRODUCTION

The use of mobile telecommunication devices (mobile phones) in everyday life is increasing, along with the development of modern society (Miyakoshi, 2013). The public has abandoned static home phones and switched to battery-based phones which can be taken anywhere (Tarigan *et al.*, 2013; Kogoya, 2015). The population of mobile phone users is over 80% in some countries (Tang *et al.*, 2015).

Mobile phone is a two-way communication tool that uses electromagnetic waves which is the wave radiofrequency (RF) (Swamardika, 2009). Mobile phones produce electromagnetic wave radiation (EMR) due to the presence of electric fields and magnetic fields (Talib *et al.*, 2010). Mobile phone EMR can increase the production of free radicals,

which will increase lipid peroxidation resulting in oxidative stress and lead to the activation of apoptotic pathways (Ozben, 2007).

The brain is one of the most sensitive organs targeted by EMR, where mitochondrial injury occurs earlier and worse than other organs (Hao *et al.*, 2015). Electromagnetic wave radiation emitted by wireless devices can interfere with learning and memory and cause degenerative effects on the human brain by increasing oxidative stress and decreasing enzymatic antioxidant brain tissue, which may affect the physiological function of the nerves (Nazziroglu and Akman, 2014).

Aluminum foil is a good conductor and non-magnetic; therefore, it can reflect almost any exposure to electric waves, thus providing protection against EMR (Ott, 1976; Pratap *et al.*, 2014). Aluminum foil can reflect about 90% of electromagnetic waves at

wavelengths of 200nm (nanometers) up to 1 μ m (micrometer), increasing to about 99% at wavelengths above 1 μ m, and may weaken more than 80dB (decibels) of EMR at frequencies over 100MHz. The magnetic field will lose about 63% of its energy with a single sheet of aluminum foil (Pratap *et al.*, 2014).

2 METHODS

This research uses laboratory experimental research with Posttest Only Control Group Design method. The study was approved by the Medical Research Ethics Committee, Faculty of Medicine, Universitas Airlangga (No. 176/EC/KEPK/FKUA/2017) to use experimental animals as research objects. The research was conducted in July and August 2017 at Pharmacology Laboratory, Faculty of Medicine, Universitas Airlangga, and MDA level examination was conducted in the Pharmacology Laboratory, Faculty of Medicine, Universitas Brawijaya. The number of samples was calculated by Frederer formula of 32 male white rats Wistar strain (*Rattus norvegicus*) then divided into four treatment groups, each group consisted of eight rats. The K1 group (Control 1) was a group of untreated rats. Group K2 (Control 2) was a group of rats protected by aluminum foil shield. The P1 group (Treatment 1) was a group of rats given exposure to mobile phone EMR with the specificity absorption rate Europe (SAR EU) 0.84-1.86W/kg and frequency 2100MHz (GSM 2100MHz). Group P2 (Treatment 2) was a group of rats given exposure to mobile phone and aluminum foil shield. Each rat was inserted into a 30x25x7cm plastic container. The plastic container for the K1 group was an empty container. The plastic container for the K2 group was given an aluminum foil shield on the bottom of the baking sheet. The plastic container for the P1 group had a mobile phone placed on the bottom of the baking sheet. The plastic container for the P2 group had a mobile phone and aluminum foil shield placed on the bottom of the baking sheet. Treatment was given four hours/day for 30 days.

After euthanasia with ketamine, cerebellar preparations were taken for examination of MDA level. The examination of cerebellar MDA level was performed by using the method of measuring the concentration of Thiobarbituric Acid Reactive Substances (TBARS) using a spectrophotometer with a wavelength of 532nm. Quantitative data were the levels of cerebellar MDA in mmol/L in analysis with Kruskal Wallis and Mann Whitney U test

using statistical product and service solutions (SPSS) software version 23 ($p < 0.05$).

3 RESULTS

Table 1 lists the mean and standard deviations of rat cerebellar MDA levels at the end of treatment. The lowest levels of rat cerebellar MDA levels were obtained in the K1 group while the highest was in the P1 group (Figure 1).

Table 1: Average of MDA cerebellar level.

Groups	Cerebellar MDA level ($\bar{X} \pm SD$)
K1 (n=8)	0.08 \pm 0.02
K2 (n=8)	0.12 \pm 0.07
P1 (n=8)	0.38 \pm 0.13
P2 (n=8)	0.09 \pm 0.05

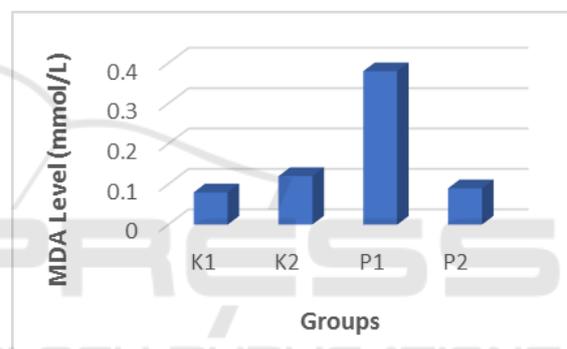


Figure 1: Graph of average of cerebellar MDA levels.

The normality test of Saphiro Wilk and homogeneity test of Levene Test showed variable of MDA levels content not normally distributed ($p < 0.05$) and not homogeneous ($p < 0.05$), but Kruskal Wallis test showed there was difference of MDA levels ($p < 0.05$).

Table 2 lists the Mann Whitney U test of MDA levels. Mann Whitney U test results showed that MDA levels of P1 group differed significantly with K1 group, K2 group and P2 group.

Table 2: Mann Whitney U test result of cerebellar MDA level.

Group (I)	Groups (J)	p
K1	K2	0.43
	P1	0.001*
	P2	0.207
K2	P1	0.002*
	P2	0.401
P1	P2	0.001*

* $p < 0.05$

4 DISCUSSION

In this study it was found that cerebellar MDA levels in P2 group were significantly lower than P1 group, and there was no significant difference in comparison with K2 group and K1 group. In addition, cerebellar MDA levels in the P1 group were significantly higher than in the control group. It can be argued that mobile phone EMR causes elevated levels of cerebellar MDA in P1 groups compared to the control group, but there was an attempt to improve the level of cerebellar MDA by administration of aluminum foil shield in group P2 compared to P1 group. This is possible because aluminum foil shield can reflect almost any exposure to electric waves, thus providing protection against EMR (Pratap *et al.*, 2014). The aluminum foil shield has non-magnetic properties, so it can reflect almost all incidents of electrical waves, can weaken electric fields over 80dB at frequencies over 100MHz and approximately 63% of magnetic field energy (Pratap *et al.*, 2014).

The level of cerebellar MDA in the P1 group was higher than in the control group because of the occurrence of oxidative stress in the cerebellum. MDA levels are generally used as a marker of oxidative stress due to free radicals (Gawel *et al.*, 2004; Asni *et al.*, 2009). Increased free radicals lead to increased MDA production. Oxidative stress that causes elevated cerebellar MDA levels in the P1 group may be due to mobile phone EMR. Radiofrequency radiation can affect individuals by increasing the production of free radicals, which increases lipid peroxidation (LPO), thus causing oxidative damage (Ozben, 2007). Free radicals can cause damage to enzymes, proteins, cell membranes, subcellular organelles, lipofuchsin pigments and DNA. Radiofrequency radiation increases ROS production or interferes with ROS production by reducing enzymatic antioxidant activity (Consales *et al.*, 2012). Radiofrequency radiation produces ROS by stimulating the oxidation of NADH (nicotinamide adenine dinucleotide) cell membranes, leading to extracellular superoxide production and oxidative stress, resulting in cellular damage (Consales *et al.*, 2012). Exposure to RF radiation in rat cerebellum can lead to increased activity of the enzyme NADH oxidase, which increases the production of ROS (Friedman *et al.*, 2007). Changes in enzyme activity and GSH decrease can be considered as an indicator of increased ROS production that occurs during the exposure period and may reflect the pathophysiological process of exposure (Dogan *et al.*, 2012). Excessive ROS

production can reduce serum levels of melatonin, which are efficient free radical binders, and gene expression stimulation of some important endogenous antioxidant enzymes, leading to decreased activity and total suppression of antioxidant capacity (Kesari *et al.*, 2013). Decreased melatonin levels due to inhibition of melatonin production by EMR can cause vertigo (Enny, 2014).

The brain, being one of the most sensitive target organs for mobile phone EMR, is where mitochondrial injury occurs earlier and worse than other organs. It caused increasing levels of cerebellar MDA of the P1 group due to mobile phone EMR (Hao *et al.*, 2015). The brain absorbs about 50% of electromagnetic waves energy when using mobile phones near the head (Dimbylow and Mann, 1994). The brain is susceptible to ROS because of the high brain oxygen consumption, the mitochondrial neurons produce O_2 , the simple antioxidant defense mechanism and the polyunsaturated fatty acid (PUFA) of neuron membrane (Friedman, 2011). Fat is often the target of oxidation because it has a molecular structure with reactive double bonds (Ho *et al.*, 2013; Gawel *et al.*, 2004). Malondialdehyde is formed when ROS reacts with fatty acids from the cell membrane resulting in fatty peroxidation, which causes the breakdown of fatty acid chains into various toxic compounds.

The results of this study are in accordance with the research of Hussein (2016), who analyzed mobile phone radiation with frequency 1800MHz, SAR 0.6W/kg for 120 min/day for three months. It caused a significant elevation of MDA level and significant inhibition in the activities of SOD and GPX in the brain regions (hippocampus and cerebellum). Exposure to RF-radiation emitted from mobile phones is associated with overproduction of ROS and altered antioxidant activities.

5 CONCLUSIONS

Based on the results of the research, it was found that giving mobile phone EMR with SAR 0.84-1.86W / kg, frequency 2100MHz, four hours/day for 30 days caused increased levels of cerebellar MDA and aluminum foil shield can provide protection by reducing the effects of radiation.

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REFERENCES

- Asni E., Harahap IP., Prijanti AR., Wanandi SI., Jusman SWA., Sadikin M. 2009. Pengaruh Hipoksia Berkelanjutan Terhadap Kadar Malondialdehid, Glutathion Tereduksi, dan Aktivitas Katalase Ginjal Tikus, *Maj Kedokt Indon*, 59(12): 595-600.
- Consales C, Merla C, Marino C, Benassi B. 2012. Electromagnetic Fields, Oxidative Stress, and Neurodegeneration. *International Journal of Cell Biology*. doi:10.1155/2012/683897
- Dimbylow PJ, Mann SM, 1994, SAR calculations in an anatomically realistic model of the head for mobile communication transceivers at 900 MHz dan 1,8 GHz. *Phys Med Biol*. 39, 1537–1553.
- Dogan M, Turtay MG, Oguzturk H, Samdanci E, Turkoz Y, Tasmemir S, Alkan A, Bakir S. 2012. Effects of Electromagnetic Radiation Produced by 3 G Mobile Phones on Rat Brains: Magnetic Resonance Spectroscopy, Biochemical, and Histopathological Evaluation. *Hum. Exp. Toxicol*. 31(6): 557–564.
- Enny. 2014. Efek Samping Penggunaan Ponsel. *Gema Teknologi*. 17(4): 178-183.
- Friedman J, Kraus S, Hauptman Y, Schiff Y, Seger R. 2007. Mechanism of Short-Term ERK Activation by Electromagnetic Fields at Mobile Phone Frequencies. *Biochem. J*. 405(3): 559–568.
- Gawel S, Wardas M, Niedworok E, Wardas P. 2004. Malondialdehyde (MDA) as a lipid peroxidation marker. *Wiad Lek*. 57(9-10):453-5.
- Hao Y, Zhao L, Peng R, 2015. Effects of microwave radiation on brain energy metabolism dan related mechanisms. *Mil. Med. Res*. 2: 4.
- Ho E., Galougahi KK., Liu CC., Bhindi R., Figtree GA. 2013. Biological markers of oxidative stress: Applications to cardiovascular research and practice. *Redox Biology*. (1) 483–491.
- Ha BY. 2001. Stabilization and destabilization of cell membranes by multivalent ions. *Phys Rev*.
- Kesari KK, Siddiqui MH, Meena R, Verma HN, Kumar S. 2013. Cell Phone Radiation Exposure on Brain and Associated Biological Systems. *Indian J. Exp. Biol*. 51(3): 187–200.
- Kogoya D. 2015. Dampak Penggunaan Handphone Pada Masyarakat: Studi Pada Masyarakat Desa Piungun Kecamatan Gamelia Kabupaten Lanny Jaya Papua. *Acta Diurna J.*, 4(4): 1-14.
- Miyakoshi J, 2013. Cellular dan Molecular Responses to Radio-Frequency Electromagnetic Fields The human exposure to nonionized EMF in connection with WPT applications is examined in this paper, highlighting various biological dan biomedical aspects in connection with DNA, mutation, cell, dan gene. *Proceedings of the IEEE*. 101: 6, 1494.
- Nazzziroglu M, Akman H, 2014. Effects of Cellular Phone- dan Wi-Fi-Induced Electromagnetic Radiation on Oxidative Stress dan Molecular Pathways in Brain. *Systems Biology of Free Radicals dan Antioxidants*. 2431-49.
- Pratap S, Khatri J, Jain P, dan Banga D, 2014. Electromagnetic Stress – A Danger to Human Health. *International Journal of Emerging Technologies in Computational dan Applied Sciences*. 14(832): 305-309.
- Ott H, 1976. *Noise Reduction Techniques in Electronic Systems*. United States: Wiley Interscience.
- Ozben T, 2007. Oxidative stress dan apoptosis: impact on cancer therapy. *J. Pharm. Sci*. 96, 2181–2196.
- Swarmadika IBA. 2009. Pengaruh Radiasi Gelombang Elektromagnetik terhadap Kesehatan Manusia. *Jurnal Teknologi Elektro*. 8 (1): 106-9.
- Talib SH, Patil P, Nikam P. 2010. Mobile phone and health hazard. *Journal Indian Academy of Clinical Medicine*, 11 (3): 212-9.
- Tang J, Zhang Y, Yang L, Chen Q, Tan L, Zuo S, Feng H, Chen Z, Zhu G, 2015. Exposure to 900 MHz electromagnetic fields activates the mcp-1/ERK pathway dan causes blood-brain barrier damage dan cognitive impairment in rats. *Brain Research*. 1601: 92-101.
- Tarigan TRP, Gani UA, Rajagukguk M. 2013. *Studi Tingkat Radiasi Medan Elektromagnetik Yang Ditimbulkan Oleh Telepon Selular*. Pontianak: Fakultas Teknik Universitas.