

# The Effect of Aluminium Foil Shielding in Hampering Electromagnetic Radiation Emitted from A Mobile Phone as an Oxidative Stressor in The Cerebra of Adult Male Rats

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**Abstract:** Mobile phones are among the main sources of electromagnetic radiation (EMR). Increased use of mobile phones with internet access in daily life might increase the adverse effects of EMR as one of the oxidative stressors to the human body. Thus, protection against EMR exposure from mobile phones is required; aluminium foil (AF) is one of the proposed materials due to its EMR absorption loss and reflective loss potency. This study aimed to investigate the effect of AF shielding against EMR emitted from mobile phones shown by the malondialdehyde (MDA) levels in the cerebra of adult male Wistar rats. Thirty-two adult male rats were divided into 4 groups (n=8). Group I was the control group without AF or EMR, group II was the control group given the AF only, group III was treated with EMR, and group IV was treated with EMR with an AF shield. Each animal was placed in a plastic box container sized 20 x 16 x 9 cm with a fencing wire cover. A mobile phone (GSM 2100 MHz; SAR 0.84-1.86 W/kg) was taped to the inner floor of this plastic box container. The AF shield had a thickness of 2 mm and covered the mobile phone. The exposure was 4 h/day for 30 days. The MDA levels of the right hemisphere of the cerebrum were measured with a spectrophotometer. Data were analyzed with significance level of  $p < 0.05$ . The MDA levels in group III were significantly higher when compared to the others. The MDA levels in group IV were significantly lower when compared to group III. The AF here might have acted as a shield against EMR from the mobile phone and was likely to have reduced the oxidative stress effect of the EMR on the exposed rats' cerebra; this could be shown by the lower levels of the MDA in the shielded subjects compared to the unshielded controls.

## 1 INTRODUCTION

Communication technology in the 21<sup>st</sup> century is progressing very rapidly, and one of the developments of communication technology is the mobile phone. The use of mobile phones is increasing day by day, and it is estimated that approximately 4.8 billion people worldwide are using mobile phones currently. Due to the wide and growing use of mobile communication, there is increasing concern about the interactions of electromagnetic radiation (EMR) with the human organs in particular with the brain, because the brain

absorbs 80% of the EMR emitted by a mobile phone (Schorborn *et al.*, 1998).

This EMR may have biological effects. Various nonthermal effects of EMR from mobile phones on the central nervous system, including permeability of the blood-brain barrier, neuronal electrical activity, nerve cell damage, changes neurotransmitter balance, and cognitive function, have been reported (Narayanan *et al.*, 2010). Formation of reactive oxygen species (ROS) and increased oxidative stress may be involved in the action of EMR on the biological system (Kim *et al.*, 2008). ROS cause injury by reacting with biomolecules, such as lipids, proteins, and nucleic acids, as well as by depleting enzymatic and/or

nonenzymatic antioxidants in the brain (Bodera *et al.*, 2015). There is a need for a material that can reduce the effect of the magnetic field and the electric field of electromagnetic wave formers.

The use of aluminium foil (AF) in reducing the EMR exposure produced by microwave ovens to cook food has been widely recognized by the public. AF can also reduce the EMR exposure generated by cellular phones. It is evidenced that AF can reflect  $\pm 90\%$  electromagnetic wavelength 200 nm (nanometer) to 1  $\mu\text{m}$  (micrometer); then this increases up to  $\pm 99\%$  in the wavelength above 1  $\mu\text{m}$  compared to other metals such as gold and silver, and also AF can absorb 63% of the magnetic field (Sabitha, 2013).

The purpose of the present study is to evaluate the intensity of oxidative stress in the brain of animals exposed to mobile phones and potential protective effects of an AF shield in reducing oxidative stress brain injury.

## 2 METHODS

### 2.1 Animals

Healthy male Wistar rats (3 months old, with initial weight of 200-300 g) were used in this experiment. They were obtained from Farmakologi Laboratory, Faculty of Medicine, Universitas Airlangga. The rats were housed at standard temperature and humidity with free access to food and water *ad libitum*, with a 12 h light and 12 h dark cycle. The experimental protocol was approved by the Ethical Committee on Health Research, Faculty of Medicine, Universitas Airlangga, Surabaya (No. 179/EC/KEPK/FKUA/2017).

### 2.2 Experimental Design

Thirty-two adult male rats were placed equally into 4 groups (n=8). Group I was the control group without AF or EMR, group II was the control group given the AF only, group III was treated with EMR, and group IV was treated with EMR with an AF shield. Each animal was placed in a plastic box container sized 20 x 16 x 9 cm with a fencing wire cover. The animals were free to move in the cage. A mobile phone was taped to the inner floor of this plastic box container and covered with a plastic bag. The AF sheet (Klin Pak, Indonesia) had a thickness of 0.2 mm, and for this study 10 layers (thickness of 2 mm) were used; after measuring the effectiveness of shielding using an *electromagnetic radiation*

*tester*, it showed a reduction by half of the initial electric and magnetic field. The AF size was 20 x 16 cm and it covered the mobile phones wholly inside the plastic bag (Fig. 1).

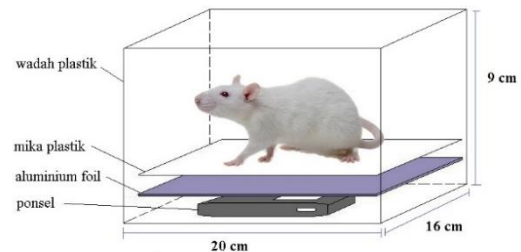


Figure 1: The Wistar rats were placed in a plastic box, with a mobile phone in active mode placed on the bottom of the box. The AF was directly above it and covered with plastic mica (group IV).

### 2.3 Electromagnetic Exposure

The animals were exposed to EMR for 30 days (4 h/day during light period). The EMR was produced by a mobile phone (BlackBerry Bold 9790, Taiwan) connected to Wi-fi, portable Andromax M2Y (Smartfren, Indonesia). EMR exposure was performed in the same room where all the animals were housed. The mobile phone had GSM (Global System for Mobile communication) at 2100 MHz with specific absorption rate Europe (SAR EU) of 0.84-1.86 W/kg. The animals were sacrificed to study MDA levels in the cerebrum.

### 2.4 Tissue Processing

The Wistar rats (n=8/groups) were anesthetized using ethylchloride on 31<sup>st</sup> day, prior to surgery. Subsequently, the cerebra were removed and each cut into 2 hemispheres (midsagittal). The right hemispheres were placed in an empty container to be sent to Universitas Brawijaya, Malang, using a cool box. The delivery process takes  $\pm 2$  hours.

### 2.5 Measurement of Oxidative Stress

In order to examine the effects of EMR on the oxidative stress in the cerebra of Wistar rats, MDA formation was used, which can be measured using a spectrophotometer with the thiobarbituric acid reactive substances (TBARS) method (Ilhan *et al.*, 2004). First, 150 mg of cerebrum samples was weighed out and then homogenized with buffer fosfat 2 cc. Next, 200  $\mu\text{l}$  of the samples was taken and aquadest 0.5 cc added to the tube. Then 200  $\mu\text{l}$

HCL vortex was added to homogenize it, and then 250  $\mu$ l TCA 40% vortex was added to homogenize it. Then 250  $\mu$ l TBA (Na-thiobarbituric acid) vortex was added to homogenize it. The tube was heated in the waterbath at 100°C for 25 min. Homogenates were centrifuged at 3,000 rpm for 10 minutes, supernatants were collected and aquadest added to 3 cc for MDA quantification. Absorbance at 532 nm was measured using spectrophotometer UV- VIS (Hitachi U-2810 Model: 122-000 No: 1819-011a, Japan).

## 2.6 Statistical Analysis

The data are presented as the mean  $\pm$  standard deviation (SD). Statistical differences were analyzed using a nonparametric test with post hoc, using SPSS 17 software with  $p < 0.05$  considered to indicate a statistically significant difference.

## 3 RESULTS

### 3.1 Quantification and analysis of MDA levels in the rat cerebrum samples

In order to investigate the effects of EMR-induced oxidative stress, MDA levels were measured in the cerebrum samples of all rats. The MDA levels varied between groups. The lowest levels of 0.138 ng/mL were obtained in group I (without AF or EMR), and the highest MDA level of 0.896 ng/mL was obtained in group III (treated with EMR). The mean of MDA levels for each group are shown in Table 1.

Table 1: The mean of MDA levels in rat cerebrum samples.

Group (n=8)	Concentration (ng/mL)
Control	0,263 $\pm$ 0,070
Control + AF	0,374 $\pm$ 0,080
EMR	0,662 $\pm$ 0,140
EMR + AF	0,265 $\pm$ 0,047
	(Mean $\pm$ SD)

Data analysis was carried out using the *Kruskal-Wallis test* followed by the *Mann-Whitney test* (Table 2). In the treated EMR group, 30 days of exposure to a mobile phone produced a significant increase in MDA levels in the cerebrum tissue ( $p < 0.001$ ), an index of lipid peroxidation, when compared with control. In the EMR + AF group, the AF shielding significantly prevented the increase in

the MDA levels in the cerebrum tissue ( $p < 0,001$ ) after exposure to a mobile phone when compared with the group treated with EMR without shielding (Table 2 and Fig. 2).

Table 2: *Mann-Whitney test* result between groups for MDA levels in the cerebrum.

Group	Control	Control + AF	EMR	EMR + AF
Control	-	0,012 ***	0,001 ***	1,000
Control + AF	0,012 ***	-	0,001 ***	0,009***
EMR	0,001 ***	0,001 ***	-	0,001***
EMR + AF	1,000	0,009 ***	0,001 ***	-

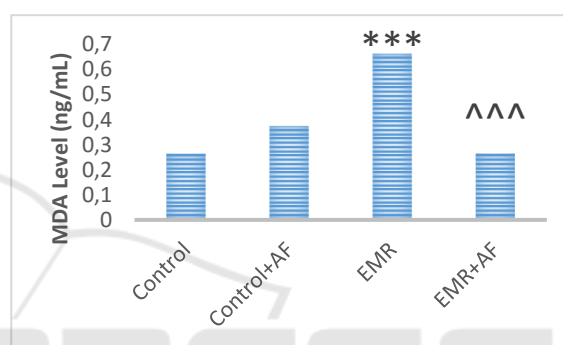


Figure 2: The effect of AF on lipid peroxidation (MDA level-ng/mL) in the cerebra of rats exposed to EMR. \*\*\* $p < 0.001$  (vs. control); ^^ $p < 0.001$  (vs. EMR).

## 4 DISCUSSION

Oxidative stress resulting from excessive ROS formation or the deterioration of antioxidant defense capacity has been closely linked to the pathogenesis of neuronal dysfunction or death (Ning *et al.*, 2007). The cerebrum is particularly vulnerable to ROS formation due to its high metabolic rate, its deficient oxidant defense mechanisms, nerve membranes rich in PUFA (*Polyunsaturated Fatty Acids*), and its diminished cellular turnover (Floyd, 1999; Friedman, 2011). One study reported an increase in MDA levels and decreased levels of GSH and CAT in guinea pigs' cerebrum tissue after exposure to a GSM mobile phone (SAR 0.043-0.135 W/kg for 20, 40, and 60 days); this proves EMR can lower at the level of antioxidants, increase lipid peroxidase, and form free radicals in brain tissue (Sokolovic *et al.*, 2008). The cerebrum may absorb emitted EMR more than other internal organs because mobile phones are generally used near the head.

#### 4.1 Lipid Peroxidation Increase in the Cerebra of Rats after Exposure to a Mobile Phone

Increased production of lipid peroxidation is initiated by reactive free radicals. This highly reactive lipid peroxidation causes damage to neurons through direct or indirect effects. The direct effects include the loss of fluidity, a decrease in fluidity, a depression in the protein mobility in the membrane, and the increased phospholipid exchange between the bilayers of the membrane (Sokolovic *et al.*, 2008). The breakdown of cell membranes in the worst case causes inactivation of membrane-bound enzymes and loss of decompartmentalization; this will cause the neuron membrane to leak until enzymes can pass through the damaged membrane, resulting in the destruction of membrane function as a barrier. Lipid peroxidation also results in loss of ionic homeostasis leading to impaired  $\text{Ca}^{2+}$  ion compartments. The loss of  $\text{Ca}^{2+}$  homeostasis causes the loss of metabolic control of neuronal cells. The indirect effects through the activation of other mediators by the product lipid peroxidation (Consales *et al.*, 2012). The MDA that is produced as a consequence of lipid peroxidation is biologically active.

In this study, increased levels of lipid peroxidation were found in the cerebra of rats exposed to the EMR of a mobile phone (2100 MHz and SAR of 0.84-1.86 W/kg) during observed periods of 30 days (Fig. 2). the treated EMR group without shielding had a significant increase in MDA levels in the cerebrum tissue ( $p < 0.001$ ) compared with all groups. This result suggests that the EMR can induce brain damage in exposed rats by increasing oxidative stress and lipid peroxidation. Similar findings were presented by Sokolovic *et al.* (2008). They found that MDA levels in rat brain tissues were increased significantly after exposure to a mobile phone.

#### 4.2 AF Shielding Can Reduce the Oxidative Stress Effect of Mobile Phones

The present study was also designed to explore the protective effects of AF shielding against free radicals in EMR-induced brain oxidative stress. In the treatment group with AF shielding, the high MDA level in the cerebrum after the EMR-induced oxidative stress was prevented. In the EMR + AF group, there was significant prevention in increase in the MDA content of the cerebrum tissue, after 30

days of exposure to mobile phones ( $p < 0.001$ ) when compared with EMR group (Table 2 and Fig. 2).

There are 2 basic mechanisms of AF used as shielding; there is reflection and absorption losses, both of which play a major role in shielding. AF can reflect  $\pm 90\%$  of the electromagnetic wavelength and absorb 63% of the magnetic field; with this capability AF can reduce the EMR of a radiation source (Sabitha, 2013).

## 5 CONCLUSIONS

The AF here might have shielded against the EMR from the mobile phones and was likely to have reduced the oxidative stress effect of the EMR on the exposed rats' cerebra. This could be shown by the lower levels of the MDA in the shielded subjects compared to the unshielded controls.

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## REFERENCES

- Bodera, P. *et al.*, 2015. 'Influence of Electromagnetic Field ( 1800 Mhz ) on Lipid Peroxidation in Brain , Blood , Liver And Kidney In Rats.', *International Journal of Occupational Medicine and Environmental Health*, 28(4), pp. 751–759.
- Consales, C. *et al.*, 2012. 'Electromagnetic fields, oxidative stress, and neurodegeneration', *International Journal of Cell Biology*, 2012.
- Floyd, R. A., 1999. 'Antioxidants, Oxidative Stress, and Degenerative Neurological Disorders', *Proceedings of the Society for Experimental Biology and Medicine*, 222(3), pp. 236–245.
- Friedman, J., 2011. 'Oxidative Stress and Free Radical Damage in Neurology', pp. 19–28.
- Ilhan, *et al.*, 2004. 'Ginkgo biloba prevents mobile phone-induced oxidative stress in rat brain.' *Clinica Chimica Acta*, vol. 340, pp. 153-162.

- Kim, T.-H. T.-H. *et al.*, 2008. 'Local exposure of 849 MHz and 1763 MHz radiofrequency radiation to mouse heads does not induce cell death or cell proliferation in brain.', *Experimental & molecular medicine*, 40(3), pp. 294–303.
- Narayanan, S. N. *et al.*, 2010. 'Effect of radio-frequency electromagnetic radiations (RF-EMR) on passive avoidance behaviour and hippocampal morphology in Wistar rats.', *Upsala journal of medical sciences*, 115(December 2009), pp. 91–96.
- Ning, W. *et al.*, 2007. 'Effects of GSM 1800 MHz on dendritic development of cultured hippocampal neurons', *Acta Pharmacologica Sinica*, 28(12), pp. 1873–1880.
- Sabitha, S., 2013. 'International Journal of Emerging Technologies in Computational and Applied Sciences (IJETCAS)', pp. 513–519.
- Schornborn, F., Burkhardt, M., Kuster, N., 1998. 'Differences in energy absorption between heads of adults and children in the near field of sources', *Health Phys*, 74, pp. 160–168.
- Sokolovic, D., Djindjic, B., Nikolic, J., Bjelakovic, G., Pavlovic, D., Kocic, G., Krstic, D., Cvetkovic, T. and Pavlovic, V., et al., 2008. 'Melatonin Reduces Oxidative Stress Induced by Chronic Exposure of Microwave Radiation from Mobile Phones in Rat Brain', *Journal of Radiation Research*, 49(6), pp. 579–586.

