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# Effect of peat biomass smoke exposure on oxidative stress in *Wistar* rats

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## ABSTRACT

**Background:** Indonesia ranks third in the world regarding air pollution due to forest and land fires; most of the land burned is a peatland. Particulate matter (PM) 2.5 is the largest component of the total smoke particles. Short-term and long-term exposure to PM2.5 remains a hazard to human health.
**Objective:** This study aims to examine the effect of exposure to peat biomass smoke on serum malondialdehyde (MDA) levels and body weight of Wistar rats.
**Methods:** Experimental animals were randomly divided into three groups: Control group (C) is not given treatment, and treatment groups (X1 and X2) are exposed to smoke from peat biomass of 100 g and 150 g of biomass for 60 seconds per day for 14 days. The body weight was examined before and after treatment, while Serum MDA levels were examined after treatment.
**Results:** The results showed significant differences ($p < 0.05$) in serum MDA levels between groups. The highest serum MDA levels were found in group X2 (3.03 ± 0.185 nmol/ml), followed by group X1 (2.67 ± 0.212 nmol/ml) compared to the control group (2.24 ± 0.476 nmol/ml). In contrast, increasing body weight between groups did not show a significant difference.
**Conclusion:** Exposure to PM 2.5 from peat biomass smoke increases oxidative stress in experimental animals.

***Latar Belakang:*** *Indonesia menempati peringkat ketiga di dunia terkait polusi udara akibat kebakaran hutan dan lahan; sebagian besar lahan yang terbakar adalah gambut. Partikel materi partikulat (*particulate matter, *PM) 2.5 merupakan komponen terbesar dari total partikel asap. Paparan jangka pendek dan jangka panjang terhadap PM2.5 dapat menjadi ancaman bagi kesehatan manusia.*
***Tujuan:*** *Penelitian ini bertujuan untuk melihat efek paparan asap biomassa gambut terhadap kadar malondialdehida (MDA) serum dan berat badan tikus Wistar.*
***Metode:*** *Hewan percobaan dibagi secara acak menjadi tiga kelompok: kelompok kontrol (C) tidak diberi perlakuan, dan kelompok perlakuan (X1 dan X2) terpapar asap biomassa gambut sebanyak 100 g dan 150 g biomassa selama 60 detik per hari selama 14 hari. Penimbangan berat badan dilakukan sebelum dan setelah perlakuan. Sedangkan kadar MDA serum diperiksa pada akhir perlakuan.*
***Hasil:*** *Hasil penelitian menunjukkan perbedaan signifikan ($p < 0.05$) kadar MDA serum antara kelompok. Kadar MDA serum tertinggi ditemukan pada kelompok X2 (3.03 ± 0.185 nmol/ml), diikuti oleh kelompok X1 (2.67 ± 0.212 nmol/ml) dibandingkan dengan kelompok kontrol (2.24 ± 0.476 nmol/ml). Sedangkan peningkatan berat badan hewan coba antar kelompok tidak menunjukkan perbedaan yang bermakna.*
***Kesimpulan:*** *Paparan PM2.5 dari asap biomassa gambut meningkatkan stres oksidatif pada hewan percobaan.*

## INTRODUCTION

The worst forest and land fire incident in Indonesian history occurred in 1997 due to the El-Nino phenomenon.[1] Riau Province is one of the most affected areas, with approximately 40% of its total land area, or roughly 3.9 million hectares, composed of peatland, a portion of which is influenced by tidal inundation. Every dry season fires occur in Riau Province, but not as severe as in 1997-1998.[1,2] Forest and peatland fires are surface fires where the fire burns material above the surface, then the fire spreads slowly below the surface produced smoke generally consists of carbon monoxide, carbon dioxide, ozone, sulfur oxides, particulate matter (PM), and several other materials such as aldehydes (acrolein, formaldehyde), polycyclic aromatic hydrocarbons (PAHs, for example, benzo-a-pyrene), benzene, toluene, styrene, metals, and dioxins although found in small amounts. Particulate matter is a concern in forest fire smoke due to its small size, allowing it to be inhaled deeply into the lungs.[3,4,5]

The influential and sensitive particulate parameters that affect the air quality index (AQI) criteria are particles with a diameter of 2.5 micrometres or PM2.5. During the analysis of aerosol samples collected from peatland fires in the Riau Province in 2012, PM2.5 was found in high concentrations, measuring 7120 ± 3620 $\mu g/m^3$. These results are significantly higher than the PM2.5 threshold level set by Government Regulation No. 41 of 1999 concerning air pollution control (65 $g/m^3$).[6]

If PM2.5 is inhaled through the upper respiratory system, it cannot be filtered and will penetrate the deepest part of the lungs. Short- and long-term exposure to PM2.5 remains adverse effects on human health.[7] The mechanism of PM in causing an impact on health is the formation of reactive oxygen and reactive nitrogen species (ROS/RNS). Free radicals will cause cell damage, lipid peroxidation, and protein oxidation. The end product of lipid peroxidation is malondialdehyde (MDA). Inflammation that occurs in the short term increases the risk of recurrence of several diseases, such as chronic obstructive pulmonary disease (COPD), asthma, and autoimmune diseases, and in the long term, causes various metabolic and cardiorespiratory diseases.[8,9]

Studies from various countries that have experienced the haze disaster from biomass fires report that the type of biomass physicochemical properties of the PM contained could cause different effects on the body.[10,11,12] Smoke exposure in recent in vivo studies affected biochemical, histopathological, cellular, to molecular changes.[13] There are three mechanisms of health disturbance due to biomass burning haze, specifically increasing oxidative stress, triggering inflammatory processes, and genotoxic effects.[12] However, no similar studies have been conducted on the effects of smoke exposure from forest-sourced biomass in Indonesia, especially in Riau. Several published reports only contain short-term effects on human health.

Previous studies have demonstrated that exposure of *Wistar* rats to PM2.5 from motor vehicle fumes for 100 seconds per day over 14 days resulted in increased serum MDA levels compared to a control group.[8] Additionally, rats showed decreased endogenous antioxidants, specifically superoxide dismutase (SOD) and glutathione peroxidase.[14] Herein, investigating the impact of PM2.5 from peat biomass smoke on serum MDA levels in *Wistar* rats was conducted.

A study conducted by Fitry et al. demonstrated increasing serum MDA levels in *Wistar* rats exposed to PM2.5 from motorbike exhaust for 100 seconds per day for 14 days compared to controls.[8] Another investigation by Lu et al. similarly highlighted heightened MDA levels and a concomitant reduction in the endogenous antioxidant enzymes, superoxide dismutase (SOD) and glutathione peroxidase, within rats subjected to PM2.5.[14] In accordance with an earlier study, exposure to PM2.5 from ambient air and motor vehicle emissions led to an escalation in serum MDA levels.[14] Based on a previous study, serum MDA levels increased by exposure to PM2.5 from air ambient and motor vehicle fumes. Therefore, this study was conducted to determine the effect of exposure to PM2.5 from peat biomass smoke on oxidative stress and increasing body weight in *Wistar* rats.

## METHODS

This experimental study employed a post-test-only control group design. The Medical Research Ethics Commission / Health Faculty of Medicine Sultan Agung Islamic University Semarang approved the study protocol and procedure with certificate No. 287/VIII/2022/

Commission on Bioethics.

**Subject of study**

This study uses experimental animals as subjects. They were male white *Wistar* rats aged 2-3 months, had 150-200 g body weight, were active, and had no physical disability. To ensure a controlled environment, the animals underwent a seven-day acclimatisation period in cages with a room temperature ranging from 25-28°C and a room humidity of 70-75%. The lighting schedule followed a 12-hour light and 12-hour darkness pattern. The rats had *ad libitum* access to food and water throughout the experiment. Each group of rats was individually housed in separate cages. Using a random number table, the rats were randomly divided into three groups (six rats each). The control group (C) was not given treatment; treatment groups (X1 and X2) were exposed to peat biomass smoke from 100 g and 150 g of peat. The procedure of smoking exposure is in accordance with a previous study.[9] The exposure to smoke in the treatment groups was given 60 seconds daily for 14 days. The outputs of this study are the concentration of PM2.5, the body weight of animals before and after exposure, and serum MDA levels after exposure.

**Time and place of study**

This study was carried out from November 2021 to October 2022. Peat biomass smoke was taken from the Pelalawan area, Riau Province. Maintenance, feeding, and exposure to smoke were conducted in the animal house of Abdurrab University. Examining PM2.5 levels was conducted in the Laboratory of Air Quality of Brawijaya University, and examination of serum MDA levels in the Biochemistry Laboratory of Andalas University.

**The procedure of peat biomass smoke exposure**

The biomass was accurately weighed and burned within a specialised combustion chamber to generate peat biomass smoke, modified from a previous study.[9] The resulting smoke was collected in a reservoir and connected to the smoking chamber (Figure 1). To ensure that only particles with a diameter of ≤ 2.5 μm were released, the smoke from the reservoir was effectively filtered using Whatman paper type GF/B. Kanomex equipment was employed to measure the levels of PM2.5 in the smoking chamber.

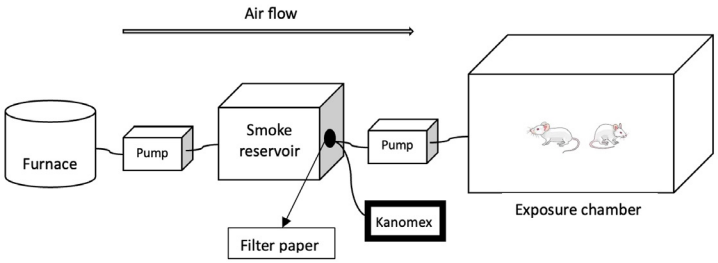


Figure 1. Exposure procedures

**Body weight and MDA serum levels examination**

Before and after exposure, the body weight of the rat was examined using digital scales Metler Toledo and blood samples (3 ml) were collected after exposure from the heart of rats using syringes. Before the procedure, the rats were anesthetised with an intramuscular injection of 60 mg/kg BW of Ketamine. The blood samples were put into a microcentrifuge tube, left for 2 hours at room temperature, and then centrifuged at 4000 rpm for 10 minutes to obtain blood serum. The blood serum is transferred into a new empty microcentrifuge tube with a label. The serum is stored in a freezer until the serum MDA levels are checked.

The blood serum (50 μL) was transferred to a test tube and diluted with water to a final volume of 500μL. Trichloroacetic acid (TCA) (250 μL of 20%) was added to the serum solution. The blood serum and TCA mixture were thoroughly mixed and allowed to stand at room temperature for 5 minutes. Subsequently, the solution was centrifuged at a medium speed (2000g) for 10 minutes, and the supernatant was carefully collected. 500μL of distilled water was added to the collected supernatant and mixed to prepare the final solution. This solution was left at room

temperature for 5 minutes. Next, 500µL of 0.67% TBA (thiobarbituric acid) was thoroughly mixed into the solution. The solution was then heated in a closed tube for 10 minutes at 100°C. After heating, the tube was removed and cooled in an ice-water bath before being centrifuged at a medium speed (2000 × g) for 10 minutes. Finally, the levels of serum MDA were measured using a spectrophotometer at a wavelength of 530 nm.[15.]

**Data analysis**

The data presented are in the form of increased body weight and serum MDA levels. The parametric one-way ANOVA test was used to analyse the MDA level with peat biomass smoke, and the Kruskal-Wallis test was performed to analyse the relationship between biomass smoke and body weight. The p-value is considered significant if p <0.05 with a 95% confidence interval.

## RESULTS

**The serum MDA and peat biomass smoke levels**

The results for PM2.5 levels of peat biomass smoke samples used in this study showed that from 100 g and 150 g of peat biomass, 7.87 g and 16.96 g of biomass burned with concentrations of PM2.5 were 12901 ± 1881µg/$m^3$, and 25672 ± 1004 µg/m3 respectively. The MDA serum levels in control and treatment groups after 14$^{th}$ days of peat smoke exposure are shown in Table 1. The highest serum MDA level was in group X2 at 3.03 ± 0.185 nmol/mL.

The LSD Post Hoc Test was carried out to find out which group had a significantly higher difference, with the results of group X2 (p = 0.001) having a significantly higher difference, followed by group X1 (p = 0.035) compared to the control.

**Characteristics of animal samples**

The animal characteristics in this study were determined by calculating the change in body weight, which was obtained by subtracting the body weight before treatment from the body weight after treatment (Table 2). Among the groups, the control group exhibited the greatest rise in body weight, measuring 17.33 ± 24.985 g. Notably, no statistically significant differences were observed in the delta of animal body weight across the various groups.

Table 1. The serum MDA level after exposure to peat biomass smoke

| Group | MDA level (nmol/mL) | p |
|---|---|---|
| C | 2.24 ± 0.476 | 0.003* |
| X1 | 2.67±0.212 | |
| X2 | 3.03 ± 0.185 | |

*One way ANOVA p<0,05, Note; C: control group, X1: exposure group 100 g of peat biomass, and X2: exposure group of 150 g peat biomass

Table 2. Body weight of animals before and after treatment of peat biomass smoke exposure

| Group | Before treatment | After treatment | Delta | p |
|---|---|---|---|---|
| C | 194.17±19.631 | 211.50±25.642 | 17.33±24.985 | |
| X1 | 171.67±26.778 | 185.83±23.575 | 14.17±34.661 | 0.927 |
| X2 | 191.00±45.166 | 200.17±50.527 | 9.17±82.684 | |

*Kruskal wallis p>0.05; C: control group, X1: exposure group 100 g of peat biomass, and X2: exposure group of 150 g peat biomass

## DISCUSSION

This study discovered that leukocytosis, aneIn this study, a significant difference exists in increasing the MDA level of peat biomass smoke exposure. The result proves that PM2.5 exposure increases the production of free radicals, which cause oxidative stress, as indicated by high levels of MDA as a marker of lipid peroxidation. Induction of oxidative stress due to exposure to PM2.5 was also found in previous *in-vitro* studies on human bronchial epithelial BEAS-2B cells, A549 cells (neoplastic transformation of human lung type II epithelial alveolar cells), and U937 cells (human monocytic leukaemia cells) which exposed to

PM2.5 from ambient air.[12,16] Oxidative stress occurs due to an imbalance in ROS with antioxidants. ROS is a term for oxygen-derived species in the form of free radicals (superoxide, hydroxyl radicals, alkoxyl, and peroxyl) and non-radicals (hydrogen peroxide). Free radicals can be generated from normal cellular metabolisms and environmental factors such as air pollution or cigarette smoke, which will react with biomolecules such as proteins, lipids, and DNA.[17] ROS can have both beneficial and toxic effects on the body. In small amounts, ROS act as a defence against pathogenic microorganisms, cellular signalling, mitogenic responses, and redox regulation, and in large amounts can cause oxidative stress. Oxidative stress can cause cancer, neurological disorders, atherosclerosis, hypertension, diabetes, COPD, and asthma.[18]

The results of testing PM2.5 levels in this study were higher than PM2.5 levels in Bengkalis Regency, Riau Province, when forest fires occurred, 7120 ± 3620 g/m$^3$ and PM2.5 levels at forest fire locations in Siak were 2163.49 g/m$^3$.[19,20] Meanwhile, the PM2.5 threshold in ambient air is set by the United State Environmental Protection Agency (USEPA) at 35 g/m$^3$, and in Indonesia, the PM2.5 threshold is by Government Regulation No. 41 of 1999 concerning Air Pollution Control of 65 g/m$^3$. The results of the characterisation that have been identified in PM2.5 there are 15 elements, namely Al, Ca, Ti, Cl, Mn, As, Br, Na, K, Se, Fe, Zn, Cr, Co, Sb, and the highest concentration were Al, Ca, and Ti.[21] This result might be higher than the level of PM2.5 when fires occurred in Bengkalis and Siak because this study uses an exposure model in a laboratory; the animals inhaled smoke directly.

The World Health Organisation (WHO) has identified health impacts associated with emissions from burning biomass, specifically PM. Fine particles, known as PM2.5, can penetrate the alveolar areas of the lungs, while ultrafine particles can penetrate the epithelial layer and adhere to the alveolar walls, potentially interacting with respiratory epithelial cells.[22] Airway epithelial cells are the first line of defence against airborne pollutants. In the tracheobronchial airways, the mucus layer captures the particles; then, phagocytic cells remove these particles. Particles in the airways could activate inflammatory cells to produce ROS/RNS in high concentrations, as well as play a role in the formation of other reactive compounds directly, such as superoxide compounds, hydroxyl radicals, hydrogen peroxide, nitric oxide, and peroxynitrite. ROS/RNS produced by neutrophils, airway epithelial cells, and phagocytic cells is thought to play a direct or indirect role in the biological response of the airway epithelium to exposure to airborne particles. ROS can directly damage tissues through cellular lipid peroxidation, double-strand DNA damage, and protein structure and function oxidation.[23] Many studies reported that environmental factors comprising motor vehicle fumes, cigarette smoke, and herbicides commonly used in agriculture have increased MDA levels.[8,24,25]

The level of exposure to inhaled PM by humans depends on the composition of the PM, which varies in terms of particulate size. The presence of fast particles, equivalent to PM2.5, can scatter light and impair visibility.[2] To assess air quality and provide the public with information about the level of air pollution in their environment, the air pollutant standard index (*indeks standar pencemar udara*, ISPU) is utilised. The ISPU, as defined by the Regulation of the Minister of Environment and Forestry of the Republic of Indonesia, consists of seven indicators, including particulate matter (PM10 and PM2.5), carbon monoxide, nitrogen dioxide, sulfur dioxide, ozone, and hydrocarbons that pose a hazardous.[6]

The increase in animal body weight in the exposure group was smaller than in the control group. The same result was also found in a previous study, that exposure to biomass smoke showed the smallest increase in body weight compared to the group exposed to cigarette smoke and the control group.[26] Other studies investigating the effects of PM2.5 exposure, especially from motorised vehicle exhaust, also showed a similar trend. The treatment group exposed to PM2.5 showed a lower average body weight than the control group.[27] Various stress-triggering factors can affect weight gain in experimental animals. In the study above, the group of rats given a stressor did not show a significant increase in body weight compared to the control group.[26-28]

## CONCLUSION

The concentration of PM2.5 was associated with MDA serum level but not with body weight. This study revealed that peat biomass smoke can cause an increase in oxidative stress which is likely

related to metabolic disorders and organ damage. Therefore, it is highly recommended to conduct further study.

## CONFLICT OF INTEREST

There is no conflict of interests.

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