The Effect of Cd (II) Metal Ion Induction to Organ Experiment Rats

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Abstract. Cd (II) is a hazardous compound with high toxicity. Cadmium toxicity contributes to a large number of health problems, including diseases that are the primary killers of heart disease, cancer and diabetes. The ability of Cd (II) to produce free radicals can cause a damaging effect on plasma lipids and lipoproteins through lipid oxidation and contribute to the ability of Cd (II) to produce effects on the cardiovascular system. This study aims to investigate distribution of Cd (II) in various organs through in vivo model using rats as a sample. The method used in this study is the Batch system. Using 6 rats as samples, which were divided into 2 groups. The first group as a control and remain group act as Cd (II) group (Injected by 1000 mg/L). The highest until the lowest level of Cd (II) are the liver ($45.66 \times 10^{-3}$ mg/g), lungs ($34.16 \times 10^{-3}$ mg/g), kidney ($11.75 \times 10^{-3}$ mg/g) and spleen ($6.11 \times 10^{-3}$ mg/g), respectively. From this study it can be concluded that the highest accumulation Cd (II) in the liver and lowest level was in spleen.

Keywords: Cd (II), experiments rats, organ

1. Introduction
Compound Cd(II) is a hazardous compound with high toxicity. Cadmium toxicity contributes to a large number of health problems, including diseases that are the primary killers of heart disease, cancer and diabetes. Cadmium concentrated in the kidneys, liver and various types of organs is estimated to have a higher toxicity than those of the common cold. Sensitive Cadmium at a level of tenths of lead, mercury, aluminum or nickel Compound Cd (II) is a hazardous compound with high toxicity. Cadmium toxicity contributes to a large number of health problems, including diseases that are the primary killers of heart disease, cancer and diabetes. Sensitive Cadmium at a level of tenths of lead, mercury, aluminum or nickel [1]
The sources of contamination of Cd (II) environment are electroplating, processing metals, pigments, plastics, batteries, mining and refining processes [2]. The ability of Cd (II) to produce free radicals can cause a damaging effect on plasma lipids and lipoproteins through lipid oxidation and contribute to the ability of Cd (II) to produce effects on the cardiovascular system. For some recent research carried out by Olisekodiaka [3]. Exposure to cadmium can cause damage to organs especially in humans which can cause "disease". Cadmium compounds are classified as carcinogenic elements to humans [4]. According to the Central Pollution Control Board (CPCB), the permissible limits for Cd (II) in wastewater are 2 mg/L and the permissible limits for Cd (II) in drinking water 0.001mg/L.

Recently, some studies are aim to look for resolve about toxicity of Cd (II). One of these study is Nasution et al. (2015) [5] that reported seed and fleshes of Mahkota Dewa (Phaleria macrocarpa) as an alternative low cost of biosorbents for removal of Cd (II) ion. Using Scanning Electron Microscope (SEM) and Fourier Transform Infra Red Spectroscopy (FT-IR), the maximum biosorption capacitiees (Q) estimated from Langmuir isotherm model for Cd (II) ion were 21.4592 mg/g and 24.7629 mg/g for seed and flesh of mahkota dea, respectively.

Regarding information that have been mentioned above. The resercher interest for investigation the distribution of Cd (II) in various organs through in vivo model using rats as a sample. The benefit is knowing toxicity Cd (II) in animal organs.

2. Methods

![Figure 1. The Model of Intervention in The Group of Samples](image)

The method used in this study is the Batch system. The solution of Cd (II) 1000 mg/L was made by dissolving 2.3699 grams (CH$_3$COO)$_2$Cd.2H$_2$O into 1 liter soluble using aquadest as the solvent. There
were 6 rats as samples. Those rats are divided into 2 (two) groups with 3 (three) rats in each group. The first group (control) were given a diet with normal diet without injection of Cd (II) and the remain group (Metal) were injected by Cd (II) 1000 mg/L at a dose of 1 mL x weight of mice/200 g of rat weight by means of inject in the part of intraperitoneal. After 30 daoy from the injection, the rats’ organ would be measured the level of the Cd (II). The organs which would be measured level of cadmium were liver, lungs, kidney, and spleen.

Measuring level of Cadmium in the organ sampel was using Percelain Scale. For the beginning, heated porcelain saucer in an oven with a temperature of 100-105°C for one hour then put into the desiccator ± 15 minutes, weighed the mass, put it in the oven again for 1 hour and weighed again until the mass was constant. The porcelain cup was then filled with rat organs which had been crushed with mortar first. Then put it again in the oven until the water content is gone and the mass of porcelain + organ is constant. Then put into the furnace for 6 hours with a temperature of 600°C. Samples that have become ash are then destroyed. Samples in porcelain were inserted into the fume hood then added 3mL of 86% HNO₃ and heated until the remaining HNO₃ was slightly in the porcelain and added 1 mL of distilled water. Steering is dissolved to 10 mL in volumetric. The level of Cd (II) in Porcelain was measured by atomic Absorption Spectrocopy (AAS).

3. Results and Discussion

| No | Organs | Control (x 10⁻³ mg/g) | Metal Cd (x 10⁻³ mg/g) |
|----|--------|-----------------------|------------------------|
|    |        | ( Group 1 )           | Group II               |
| 1  | Liver  | Unknown               | 45.66                  |
| 2  | Lungs  | Unknown               | 34.16                  |
| 3  | Kidney | Unknown               | 11.75                  |
| 4  | Spleen | Unknown               | 6.11                   |

The accumulation of Cd (II) metals in each organ of the rat experimental exception in the brain. Based on the table 1 above, it can be seen that the differences in the distribution of cumulative metal Cd (II) in the rat organ of the experiment.

The highest until the lowest accumulation of Cd (II) are the liver (45.66 x 10⁻³ mg/g), lungs (34.16 x 10⁻³ mg/g), kidney (11.75 x 10⁻³ mg/g) and spleen (6.11 x 10⁻³ mg/g), respectively. Cd (II) affects various organs such as bones, liver, kidneys and even nervous systems. Cd (II) is an important environmental agent in the ground, water, air and food. Anthropogenic sources contribute Cd (II) to 3-10 times more than in natural sources.
Based on the figure 2 above, the level of cadmium was only found in metal Cd (II) group. In the control group, cadmium wasn’t found. The cadmium was distributed in liver, lungs, kidney, and spleen.

More toxic oxygen from Cd (II) than can cause damage to the eye and exposed animals. Josthna et al. (2012) in his research report that was photographed with the metal Cd (II) will experience the accumulation of Cd (II) in several organs, where the most accumulated organs of Cd (II) are liver, kidney and tissue. After absorption by the body, Cd (II) will be transported throughout the body, usually Cd (II) will bond with the sulphydryl group, a group containing metallothienin protein. About 30% Cd (II) will accumulate in the liver and spread throughout the body [6]. The same results were reported by Annabi et al. (2013) where the fish of exposure to Cd (II) term effect of cadmium high dose would accumulate significantly in the tissues and liver organs followed by kidney organs.

Long-term exposure of cadmium showed hepatic and renal injury, when it was injected in rat, cadmium level in liver and kidney increases linearly for the first few weeks after that, the cadmium level in kidney remain steady and decrease gradually in hepatic [7].

4. Conclusion

From this study it can be concluded that the highest accumulation Cd (II) in the liver (45.66 x 10^-3 mg/g) and lowest level was in spleen (6.11 x 10^-3 mg/g).

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