**Effect of Mangosteen Peel Extract on SGOT and SGPT in Rats Fed Reused Cooking Oil**

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**ABSTRACT**

**Background:** Free radicals that enter the body due to consumption of reused cooking oil can cause liver cell damage. Mangosteen peel extract (Garcinia mangostana L) is known to contain mangostin as an antioxidant. However, it is not known whether it can repair liver damage. **Objective:** To determine the effect of mangosteen peel extract on the levels of SGOT and SGPT of Wistar rats fed with reused cooking oil.

**Methods:** This study was a true experimental study with post-test only controlled group design. Twenty four male Wistar rats were randomly divided into 4 groups randomly. The CN-G group was given the standard diet, the MJ-G group was given a standard diet and cooking oil, the MJM-400 group was given standard diet, reused cooking oil, and mangosteen peel extract at a dose of 400 mg/KgBW, and the MJM-800 group was fed with a standard, reused cooking oil, and mangosteen peel extract at 800mg/KgBW. The treatment was carried out for 28 days, and then continued with examination of SGOT and SGPT levels using the International Federation of Clinical Chemistry (IFCC) method without Pyridoxal Phosphate 37°C.

**Results:** Kruskal Wallis test showed that SGOT and SGPT levels showed no significant differences between groups (p = 0.197 and 0.063, respectively).

**Conclusion:** Administration of mangosteen (Garcinia mangostana L) peel extract did not affect SGOT levels, even tended to increase SGPT levels in rats induced by cooking oil.

**Keywords:** reused cooking oil, mangosteen peel extract, SGOT levels, SGPT levels

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**INTRODUCTION**

Frequent consumption of fried food prepared using reused cooking oil has been associated with organ damage. Reused cooking oil is cooking oil used or heated repeatedly and continuously. According to Susenas data, consumption of cooking oil 2011 was 8.24 liters per capita annually and increased to 9.33 liters in 2012 (Susenas, 2012). The study conducted by Vanessa and Bouta stated that 77% of 163 respondents were willing to consume the used cooking oil (Vanessa and Bouta, 2010). Repeated heating in cooking oil causes the process of hydrolysis, oxidation, and polymerization which produces Reactive Oxygen Species (ROS) such as anionic superoxide (O$_2^•$), hydrogen peroxide (H$_2$O$_2$), and hydroxyl radical (HO•). Excessive ROS production can cause oxidative stress which can then cause liver cell

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The increasing use of traditional medicine as an alternative treatment resulted in the increased research on medicinal plants including mangosteen (Garcinia mangostana L), fruit especially its peel. Several studies have shown that mangosteen peel extract containing xanthones has an antioxidant effect (Jung et al. 2006). Transaminase (SGPT) because liver cell membrane damage causes the liver cell to release SGOT and SGPT enzymes to the bloodstream resulting in increased in SGOT and SGPT levels in the blood (Van Beek et al. 2013).

The purpose of this study was to determine the effect of mangosteen peel extract on the levels of SGOT and SGPT in Wistar rats fed used cooking oil.

METHODS
This research was a true experimental study with post-test only controlled group design conducted at the Laboratory of Diponegoro University Faculty of Medicine between April and June 2017. Twenty four male Wistar rats were randomly divided into 4 groups. The CN-G group was given the standard diet. The MJ-G group was given standard diet and reused cooking oil. The MJM-400 group, given standard diet, reused cooking oil, and mangosteen peel extract at a the dose of 400mg/KgBW, and the MJM-800 group, were fed standard diet, reused cooking oil and mangosteen peel extract at 800mg/KgBW. Provision of reused cooking oil and mangosteen was carried out for 28 days, then continued with blood collection through retroorbital plexus for examination of SGOT and SGPT levels. This research was carried out after obtaining rats fed a high trans-fatty acid diet had a cytoplasmic vacuolization, hepatocyte hypertrophy, hepatocyte ballooning, and necro-inflammation (Dhibi et al. 2011). Liver cell damage can be detected by biochemical parameters, such as serum glutamic-oxaloacetic transaminase (SGOT) and serum glutamic-pyruvic Transaminase (SGPT) (Apreliantino 2013) because liver cell membrane damage causes the liver cell to release SGOT and SGPT enzymes to the bloodstream resulting in increased in SGOT and SGPT levels in the blood (Van Beek et al. 2013).

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<table>
<thead>
<tr>
<th>Variables</th>
<th>CN-G (N=6)</th>
<th>MJ-G (N=6)</th>
<th>MJM-400 (N=6)</th>
<th>MJM-800 (N=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight (grams)</td>
<td>198.83 ± 13.58</td>
<td>192.06 ± 8.02</td>
<td>206.27 ± 8.09</td>
<td>216.25 ± 14.35</td>
</tr>
<tr>
<td>SGOT (U/L)</td>
<td>113.10 (±13.00)</td>
<td>106.08 ± 22.43</td>
<td>85.40 ± 23.89</td>
<td>176.62 ± 109.55</td>
</tr>
<tr>
<td>SGPT (U/L)</td>
<td>52.40 (±10.67)</td>
<td>46.66 ± 7.04</td>
<td>41.76 ± 7.03</td>
<td>99.10 ± 67.03</td>
</tr>
</tbody>
</table>

Figure 1. Mean SGOT and SGPT Concentration; Kruskal Wallis: SGOT and SGPT p > 0.05.
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Statistical Analysis

Statistical analysis was carried out using the Kruskal Wallis test considering the data obtained were not normally distributed and not homogeneous. The results of the analysis were considered significant if the value of p <0.05.

Results

After 28 days of treatment, the blood samples were carried out through retroorbital plexus for the evaluation of SGOT and SGPT levels. The results is shown in Table 1.

The results shown in table 1 shows the highest SGOT and SGPT levels were found in the MJM-800 group followed by CN-G, MJ-G, and MJM-400 group. To find out whether there were differences between groups, statistical analysis were needed. Since the data were not normally distributed and homogeneous, the statistical analysis used was Kruskal Wallis. The results of the analysis showed no significant differences in SGOT and SGPT levels between groups, p> 0.5 (figure 1).

Discussion

The results of this study indicated that the administration of reused cooking oil did not increase SGOT but increases SGPT, that may be strongly associated with the production of free radicals in the body. The reference value of SGOT levels for male Wistar rats ranged from 63-175 U/L, while the SGPT reference values ranged from 19-48 U/L (Giknis & Clifford 2008). In the control group (CN-G), the mean SGOT level (113.10 ± 13.00 U/L) was in the normal range, while the mean SGPT level (52.40 ± 10.67 U/L) was slightly higher than normal. The findings of this study was different from a study conducted by Totani and Ojiri in 2007, stating that the administration of reused cooking oil can cause an increase in SGOT and SGPT levels in wistar rats (Totani & Ojiri, 2007). The fundamental difference between this present study and the previous study was the duration of the administration of reused cooking oil in rats. In Totani and Ojiri's study, rats were given ad libitum reused cooking oil for 12 weeks, while in this study the administration of the oil was for 28 days (4 weeks). The administration for 4 weeks could not induce rat liver abnormalities, characterized by an increase in SGOT and SGPT levels. In addition, this present study also used a different composition of repeatedly heated cooking oil. Totani and Ojiri's research used a combination of soybean oil and canola reused cooking oil from food processing industry (high temperature >180ºC and heated for > 20 hours), while this study used unbranded palm cooking oil repeatedly used to fry cassava (temperature <180 C and frying time <20 hours). Reused cooking oil from the food processing industry containing various toxic substances such as polar compounds, carbonyl compounds, monoeoxy fatty acids, and other substances such as acrylamide which have the potential to cause stress due to free radicals (Totani et al, 2006; Totani & Ojiri, 2007). The administration of reused cooking oil increases free radicals in the body so the body needs exogenous antioxidants (Choe & Min 2007).
the those of research conducted by Maulina et al (2013) showing that the macroscopic feature of MSG induced liver damage in Mus musculus mice could be improved by the administration of mangosteen peel ethanol extract (Maulina et al. 2013).

The hepatoprotective effect of mangosteen peel extract, due to a group of xanthone compounds, has been shown to have antioxidant activity through binding of free radicals. The most abundant xanthones found in mangosteen rind, (-)-mangostin and (–)-mangostin, will specifically scavenge hydroxyl radicals and superoxide ions. In addition, these compounds can also inhibit lipid peroxidation. Due to free radicals scavenging, the oxidation reaction chain will be interrupted, and liver cell damage can be prevented (Kosem et al. 2007). (-)-mangostin has also been shown to reduce secretions from inflammatory mediators in various body tissues and activate monocyte derived human macrophages (Gutierrez-Orozco et al, 2013).

Based on the results of the study, a group of rats fed with reused cooking oil and mangosteen peel extract at a dose of 800 mg/KgBB (MJM-800) had the highest mean of SGOT and SGPT levels among the three other groups in this study. The MJM-800 group had a higher SGOT level compared to the MJ-G (66.50%) and MJM-400 groups (106.81%). Similarly, mean level of SGPT in the MJM-800 group was higher than that of the MJ-G (143.73%) and MJM-400 groups (137.31%). The difference between SGOT and SGPT levels between MJ-G and MJM-800 groups, and between MJM-400 and MJM-800P3 groups were not statistically significant (p > 0.05).

Based on the mean comparison of SGOT levels between the MJM-400 and MJM-800 groups, it can be concluded that instead of increasing the antioxidant effect on rats fed used cooking oil, the administration of mangosteen peel extract to 800 mg/KgBW increase the risk of liver cell damage. These findings were different from a study by Saraswati et al (2014) showing that toxic effects of mangosteen peel extract might affect the findings. Chivapat et al (2011) study of chronic toxicity tests of mangosteen peel extract on Wistar rats conducted for 6 months showed that at the oral dose of 500 and 1000 mg/KgBW increased the levels of SGOT, SGPT, BUN, creatinine, and the presence of hepatocellular degeneration (Chivapat et al., 2011).

CONCLUSION

The administration of mangosteen peel extract at the dose of 400 mg/kgBW for 28 days reduced SGOT and SGPT levels of wistar rats fed given reused cooking oil. However, this decline was not statistically significant. The administration of high doses of mangosteen peel (800 mg/kg BW) for 28 days increased the levels of SGOT and SGPT of Wistar rats fed reused cooking oil. This increase in SGOT and SGPT was associated with liver damage. Instead of increasing, the administration of high doses of mangosteen peel extract lower SGOT and SGPT levels.

Further studies are needed on the effect of mangosteen peel extract on SGOT and SGPT levels of Wistar rats fed reused cooking oil with a longer exposure period (more than 4 weeks), test research methods measuring liver enzyme levels before and after treatment.

CONFLICT OF INTEREST

There is no conflict of interest.

REFERENCES


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