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The Effects Of Coffee Cherry Extract (Arabica) On Liver And Kidney Function In Streptozotocin-Induced And High-Fat Diet-Fed White Rats (Rattus norvegicus)

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Abstract

*Streptozotocin (STZ) is cytotoxic to β-cells, which normally respond to glucose stimulation by releasing insulin. STZ can induce diabetes mellitus (DM), modeling both type 1 and type 2 diabetes. Diabetes mellitus can lead to renal complications, specifically diabetic nephropathy. This study aimed to determine the effect of coffee cherry (Arabica) extract administration on liver and kidney function in white rats (*Rattus norvegicus*) induced with streptozotocin and a high-fat diet. This was a true experimental study utilizing a randomized posttest-only control group design. The study sample consisted of 20 rats divided into five groups. The research was conducted from October 2024 to November 2024. Data were analyzed using One-Way ANOVA and the Kruskal-Wallis test. One-Way ANOVA results indicated no significant effect of coffee cherry extract administration on liver and kidney function. Administration of the extract did not significantly affect SGOT levels ($p=0.092$; $p>0.05$) or SGPT levels ($p=0.401$; $p>0.05$). Regarding urea levels, the significance value was $p=0.422$ ($p>0.05$). Kruskal-Wallis analysis also showed no significant effect of the extract on kidney function; specifically, there was no significant effect on creatinine levels ($p=0.067$; $p>0.05$). In conclusion, the administration of coffee cherry extract to white rats (*Rattus norvegicus*) induced with streptozotocin and a high-fat diet did not affect SGOT, SGPT, urea, or creatinine levels.*

Keywords: ***Streptozotocin (STZ), Coffee Cherry Extract, Liver Function, Kidney Function.***

INTRODUCTION

Streptozotocin (STZ) is a diabetogenic agent that can induce pancreatic β-cell damage. STZ acts by generating free radicals that cause oxidative stress, resulting in damage to cell membranes, proteins, and deoxyribonucleic acid (DNA). STZ enters pancreatic β-cells through glucose transporter 2 (GLUT-2), thereby disrupting insulin production by pancreatic β-cells and subsequently causing metabolic disturbances in the liver and kidneys.[1] Administration of STZ to white rats (*Rattus norvegicus*) can induce experimental diabetic hyperglycemia within three days.[2]

Streptozotocin (STZ) can induce both type 1 and type 2 diabetes mellitus (DM). Diabetes mellitus can cause complications in the kidneys, particularly diabetic nephropathy. This condition is characterized by persistent albuminuria and a progressive decline in the glomerular filtration rate, while diabetic nephropathy is also associated with lesions in the renal proximal tubules.[3] Previous studies have found that abnormal mitochondrial function plays an important role in the development of diabetic nephropathy. In addition, other studies have shown that STZ can cause alpha-2u globulin nephropathy in rats, characterized by mitochondrial degeneration and disruption of the renal filtration apparatus.[4] STZ-induced rats may also experience impaired renal potassium handling, resulting in abnormal potassium homeostasis. This condition is caused by dysregulation of transport proteins in the distal nephron, which affects potassium excretion.[5]

Other studies have also reported that STZ has adverse effects on liver tissue by inducing hepatotoxicity, characterized by increased levels of liver enzymes such as serum glutamic-oxaloacetic transaminase (SGOT) and serum glutamic-pyruvic transaminase (SGPT) in diabetic rats, indicating liver damage and dysfunction.[6,7] STZ interferes with glucose oxidation and is cytotoxic to β-cells and hepatocytes. This compound binds to glucose receptors because its structure resembles that of glucose. Other studies have also demonstrated that STZ increases inflammation and oxidative stress in the liver.[8]

A high-fat diet is a diet containing a high proportion and concentration of fat. Excessive fat intake can lead to fat accumulation in the body, and when fat accumulates in the kidneys, it may

increase the risk of chronic kidney disease and hypertension.[9] Other studies have shown that administration of a high-fat diet can increase creatinine levels by more than twofold and trigger a decline in renal function. The increase in creatinine levels is associated with a high-fat diet, which can lead to hypercholesterolemia.[10]

A high-fat diet can also cause triglyceride accumulation in the liver, thereby inducing inflammation and oxidative stress in hepatic tissue. The greater the accumulation of triglycerides, the greater the oxidative stress in the liver. This condition can lead to hepatic steatosis, which is characterized by the presence of fatty accumulation in the liver.[11]

Coffee cherries are among the world's most important agricultural commodities and are produced in more than 50 tropical and subtropical countries, with Brazil being the largest producer. Historically, coffee fruit has long been recognized as a source of nutrients, stimulants, and health-promoting properties. Coffee cherries are a good source of various phytonutrients.[12] Coffee cherries have a drupe-like fruit morphology and contain two seeds enclosed within a fleshy outer layer and skin. Coffee cherries account for approximately 29% of dry matter.[13]

The United States Food and Drug Administration (FDA) considers caffeine to be generally recognized as safe when consumed in cola-type beverages at concentrations of up to 0.02%. The average caffeine intake from all sources is approximately 70 mg/person/day. Approximately 90% of adults report regular caffeine consumption, with an average daily intake of 227 mg. The primary source of caffeine among adults is coffee (70%). The European Food Safety Authority (EFSA) has established safe levels of caffeine consumption for the general population. Adults are advised to limit single caffeine intake to 200 mg (approximately two regular cups of coffee, each containing 125 mL) and total daily intake to 400 mg. Pregnant women should not consume more than 200 mg of caffeine per day, while for children and adolescents, the safe limit is 3 mg/kg/day.[14,15]

Epidemiological studies worldwide indicate that coffee consumption may protect against several diseases, including liver fibrosis, cirrhosis, chronic liver disease, and liver cancer.[16] Coffee may contribute to the prevention of diseases associated with inflammation and oxidative stress, such as obesity, metabolic syndrome, and type 2 diabetes. In addition, coffee consumption has been associated with a lower incidence of several types of cancer and a reduced risk of all-cause mortality.[17] Furthermore, caffeine has been reported to provide several health benefits, including stimulation of the central nervous system, relaxation of smooth muscles, and stimulation of cardiac muscle.[18]

The benefits of coffee cherries can be observed from their composition, particularly from compounds isolated from coffee cherries. Coffee cherries contain several therapeutic components with high antioxidant activity. These components can be isolated and used individually or in various combinations to treat diabetes and diabetes-related conditions, including diabetic retinopathy.[19]

Based on the foregoing, the researchers are interested in investigating the effects of coffee cherry extract (*Arabica*) on liver and kidney function in white rats (*Rattus norvegicus*) induced with streptozotocin and a high-fat diet.

RESEARCH METHODS

This study employs a true experimental design—specifically, a randomized posttest design with a control group. The study utilized a sample of 20 white rats (*Rattus norvegicus*). Data analysis was conducted using One-Way ANOVA and the Kruskal-Wallis test to determine the effect of coffee cherry extract administration on liver and kidney function.

RESULTS AND DISCUSSION

Liver Function Test

Based on the data collected from 20 samples divided into five treatment groups, the mean results of the examinations, namely SGOT and SGPT levels, are presented in the table below.

Table 1. Mean SGOT and SGPT Levels

Parameter	Mean ± SD	K1	K2	K3	K4	K5
SGOT		175.8 ± 21.5	148.8 ± 17.1	199.25 ± 43.7	146.5 ± 24.0	167.8 ± 23.9
SGPT		74.5 ± 22.3	55 ± 13.2	60.7 ± 17.2	68.5 ± 37.0	45.3 ± 8.8

Table 1 shows that the highest mean SGOT level was found in group K3, while the lowest was found in group K4. Group K1 had the highest mean SGPT level, whereas group K5 had the lowest mean SGPT level.

Kidney Function Test

Based on the data collected from 20 samples divided into five treatment groups, the mean results of the examinations, namely urea and creatinine levels, are presented in the table below.

Table 2. Mean Urea and Creatinine Levels

Parameter	Mean ± SD	K1	K2	K3	K4	K5
Urea		36.5 ± 6.5	38.8 ± 6.8	38.7 ± 12.3	41.0 ± 7.7	49.0 ± 12.3
Creatinine		0.78 ± 0.03	0.91 ± 0.13	0.86 ± 0.08	0.83 ± 0.08	1.07 ± 0.15

Table 2 shows that the highest mean urea level was found in group K5, while the lowest was found in group K1. Group K5 had the highest mean creatinine level, whereas group K1 had the lowest mean creatinine level.

Normality and Homogeneity Tests

The data collected were subjected to the Shapiro-Wilk normality test to determine the appropriate analytical test for each examination variable. The results of the normality test are presented in the table below.

Table 3. Normality Test Results

Parameter	*p*-value K1	K2	K3	K4	K5
SGOT	0.340	0.833	0.769	0.484	0.062
SGPT	0.456	0.900	0.214	0.568	0.276

Based on Table 3, all *p*-values for the SGOT examination were >0.05. Therefore, the SGOT and SGPT data were considered normally distributed. Accordingly, a homogeneity test was performed, followed by a One-Way ANOVA test. For normally distributed data, a homogeneity test was conducted to determine whether the variances among the examination data were homogeneous or heterogeneous. Data were considered homogeneous if the significance value was >0.05 and non-homogeneous if the significance value was <0.05.

Table 4. Normality Test Results

Parameter	*p*-value K1	K2	K3	K4	K5
Urea	0.206	0.362	0.829	0.414	0.145
Creatinine	0.034	0.146	0.899	0.048	0.114

Based on Table 4, all *p*-values for the urea examination were >0.05. Therefore, the urea data were considered normally distributed, and the analysis consisted of a homogeneity test followed by One-Way ANOVA. However, the creatinine normality test showed that the data were not normally distributed (*p*-value <0.05). Therefore, One-Way ANOVA could not be performed and was replaced by the non-parametric Kruskal-Wallis test.

For normally distributed data, a homogeneity test was subsequently performed to determine whether the variances among the examination data were homogeneous or heterogeneous. Data were considered homogeneous if the significance value was >0.05, whereas data were considered non-homogeneous if the significance value was <0.05. The results of the homogeneity test are presented below.

Table 5. Homogeneity Test Results for SGOT and SGPT

Parameter	Levene Statistic	df1	df2	Sig.
SGOT	0.473	4	15	0.755
SGPT	1.234	4	15	0.338

Table 5 shows that the significance value of the homogeneity test was 0.755 for SGOT (*p*-value >0.05) and 0.338 for SGPT (*p*-value >0.05). Therefore, the variances of the SGOT and SGPT data were homogeneous, indicating that the data could be analyzed using One-Way ANOVA.

Table 6. Homogeneity Test Results for Urea

Parameter	Levene Statistic	df1	df2	Sig.
Urea	0.948	4	15	0.464

Table 6 shows that the significance value of the homogeneity test for urea was 0.464 (*p*-value >0.05). Therefore, the variances of the urea data were homogeneous, indicating that the data could be analyzed using One-Way ANOVA.

Bivariate Analysis

The SGOT, SGPT, and urea examinations showed normally distributed data with homogeneous variances; therefore, One-Way ANOVA was used for the analysis. The results are presented in the table below.

Table 7. ANOVA Test Results for SGOT and SGPT Levels

Parameter Assessed	ANOVA Sig.
SGOT	0.092
SGPT	0.401

Table 7 shows that the significance value of the ANOVA test for SGOT was 0.092 (*p*-value >0.05). Meanwhile, the ANOVA test for SGPT yielded a significance value of 0.401 (*p*-value >0.05).

Table 8. ANOVA Test Results for Urea Levels

Parameter Assessed	ANOVA Sig.
Urea	0.422

Table 8 shows that the significance value of the ANOVA test for urea was 0.422 (*p*-value >0.05). For creatinine, the data were not normally distributed; therefore, the Kruskal-Wallis test was used. The results of the creatinine analysis are presented in the table below.

Table 9. Kruskal-Wallis Test Results for Creatinine Levels

	Creatinine
Kruskal-Wallis H	8.777
df	4
Asymp. Sig.	0.067

The Kruskal-Wallis test in Table 9 shows that the significance value for creatinine was 0.067 (*p*-value >0.05).

Discussion

Streptozotocin (STZ) is a prominent diabetogenic chemical that is widely used in experimental animals to establish animal models of both type 1 and type 2 diabetes mellitus.[20] Streptozotocin induction at a dose of 30 mg/kg body weight combined with a high-fat diet in experimental rats can cause hyperglycemia and increased body weight. STZ reacts with pancreatic β-cells, causing alterations in blood insulin levels. This results in reduced insulin production, thereby impairing glucose uptake into cells and increasing blood glucose levels.[21]

Streptozotocin induces pancreatic β-cell damage due to its toxic properties. STZ enters and circulates within β-cells through Glucose Transporter 2 (GLUT-2) on the plasma membrane. GLUT-2 is associated with the liver and kidneys, which may also be damaged by streptozotocin. The effects of streptozotocin, such as pancreatic swelling and degeneration of the β-cells of the islets of Langerhans, inhibit and reduce insulin production, thereby leading to diabetes mellitus.[26]

In this study, the mean SGOT and SGPT levels in all study groups were higher than the reported normal values. Several previous studies have also reported that SGOT and SGPT levels in normal control rats were 126.5 U/L[27] and 53.25 U/L,[28] respectively, which were above the normal values.

The mean SGOT and SGPT levels in group K2, consisting of rats induced with STZ, were 148.8 U/L and 55 U/L, respectively, which were higher than those in the normal group. Previous studies have shown that STZ administration can cause hepatic damage, as indicated by increased SGOT and SGPT activity in the blood.[26] This may occur due to other uncontrolled factors, such as environmental conditions and physical activity of the rats, viral or bacterial infections that cannot be completely prevented, and stress experienced by the rats themselves.[22]

Treatment group 1 (P1) served as the control group and received no treatment other than a normal rat diet. The mean SGOT and SGPT levels in group 1 (normal) were higher than the reported normal SGOT and SGPT levels in rats, namely 175.8 U/L and 74.5 U/L, respectively. Other studies have also reported that increased SGOT and SGPT levels may be caused by extrahepatic conditions, such as persistent tachycardia, excessive physical exercise, prolonged fasting, hypothermia, obesity, and excessive protein intake. The amount of food provided to the rats, cage temperature and humidity, as well as several uncontrolled factors, may affect SGOT and SGPT levels in rats.[28] However, the mean SGOT and SGPT levels decreased in two groups. In the group administered coffee cherry extract, namely group K4, the mean SGOT level decreased compared with group K2, which was induced with STZ. Meanwhile, the mean SGPT level in group K5 decreased compared with group K2.

Based on the data analysis, the effect of treatment on liver function showed significance values of p=0.092 for SGOT (p>0.05) and p=0.401 for SGPT (p>0.05), as determined by One-Way ANOVA. This indicates that coffee cherry extract had no significant effect on liver function. This finding is consistent with a previous study examining hepatic necrosis in mice induced with monosodium glutamate (MSG) and methanolic extract of Robusta coffee beans. That study found no effect of MSG and ethanol extract of Robusta coffee beans compared with the control group, either in terms of worsening or improvement.[23]

This study also analyzed the effect of treatment on kidney function. The mean urea levels in all study groups were higher than the reported normal values.[29] Several previous studies have also reported that the urea level in normal control rats was 28.63 mg/dL,[30] which was below the values observed in this study. The mean urea level in group K2, consisting of STZ-induced rats, was 38.8 U/L, which was higher than that in the normal group. Based on the One-Way ANOVA analysis, the significance value for urea was 0.422 (p-value >0.05), indicating that coffee cherry extract had no significant effect on urea levels in the experimental animals.

For creatinine, the Kruskal-Wallis test yielded a significance value of 0.067 (p-value >0.05), indicating that coffee cherry extract had no significant effect on creatinine levels in the experimental animals. A previous study found that individuals who consumed coffee had a lower risk of chronic kidney failure than those who did not consume coffee.[25]

Caffeine in coffee may act as an antioxidant in the body, thereby helping to reduce the harmful effects of free radicals. However, coffee is a xenobiotic substance that must be metabolized in the liver and excreted through the kidneys. In excessive amounts, coffee may increase the workload of the liver and kidneys and potentially cause damage to these organs.[24]

This study has several limitations. The presence of uncontrolled factors may have contributed to impaired liver and kidney function in the control group. This may have resulted from human error during the administration of treatments to the experimental animals and during liver and kidney function examinations using reagents intended for human samples. These errors may have introduced bias into the study results because it was not possible to determine whether the impaired liver and kidney function observed in the other treatment groups was caused by the administered treatment or by other factors. Future studies should involve a larger sample size, control potential sources of bias, and use animal-specific reagents. Researchers should also prepare additional backup samples in case of animal mortality. Future researchers may also consider extending the treatment period and implementing stricter treatment protocols to determine the toxic dose of coffee for the liver and kidneys and to identify more clearly its potential adverse effects.

CONCLUSION

One-Way ANOVA analysis showed no effect of coffee cherry extract administration on liver and kidney function. The administration of coffee cherry extract did not significantly affect liver function, as the significance values for SGOT and SGPT were 0.092 and 0.401, respectively ($p > 0.05$). Furthermore, the administration of coffee cherry extract did not significantly affect kidney function, as the significance value for urea levels was $P = 0.422$ ($p > 0.05$). Kruskal-Wallis analysis also indicated no effect of coffee cherry extract administration on kidney function; the extract did not significantly affect creatinine levels, with a significance value of $P = 0.067$ ($p > 0.05$).

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