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Effects of Red Coconut Tembuluk (*Cocos nucifera* var. *rubescens*) Extract on Uric Acid Level Reduction in Mice (*Mus musculus*)

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Abstract

Increased levels of uric acid in the blood can lead to metabolic disorders such as gouty arthritis, which require effective management. This study aimed to determine the effect of red coconut tembuluk extract (*Cocos nucifera* var. *rubescens*) on reducing uric acid levels in mice (*Mus musculus*). The study employed an experimental research design using a Completely Randomized Design (CRD). A total of 28 mice were used, divided into seven groups with four replications each. The groups consisted of a normal control group (KN), which received food and water ad libitum; a negative control group (K+); a positive control group (K−); and four treatment groups (PI, PII, PIII, and PIV). The negative control group (K+), positive control group (K−), and all treatment groups were induced with lard oil for 14 days to increase uric acid levels. The positive control group (K−) was treated with allopurinol at a dose of 0.026 mg per 20 g body weight. The normal control (KN) and negative control (K+) groups received food and water ad libitum without extract administration. Treatment group PI received 10% red coconut tembuluk extract, PII received 20%, PIII received 40%, and PIV received 80%. The data obtained were analyzed using Analysis of Variance (ANOVA). The results showed that administration of red coconut tembuluk extract significantly reduced uric acid levels in mice induced with lard oil. The most effective concentration in reducing uric acid levels was 80% (PIV), with an average uric acid level of 5.4 mg/dL.

Keywords: *Cocos nucifera* var. *rubescens*; Hyperuricemia; *Mus musculus*; Tembuluk; Uric acid levels.

INTRODUCTION

Gouty arthritis, or gout, is a degenerative disease caused by abnormal purine metabolism and characterized by elevated blood uric acid levels (hyperuricemia). This condition is accompanied by the formation and deposition of urate salt crystals in the joints, leading to joint inflammation, particularly in the knees and fingers (Jufri et al., 2023). Uric acid is a metabolic end product of purine metabolism in the human body. Under normal conditions, uric acid is soluble in blood; however, when its concentration exceeds normal levels, blood plasma becomes supersaturated, resulting in a condition known as hyperuricemia or gout (Amrullah et al., 2023). Gout predominantly affects men aged 30–50 years and is often associated with hereditary factors, whereas women are generally affected after menopause. Uric acid levels are considered elevated when they exceed normal reference values. Normal uric acid levels range from 3.4–7.0 mg/dL in men and 2.4–5.7 mg/dL in women (Amatucci et al., 2023; Jufri et al., 2023).

The primary cause of gout is elevated blood uric acid levels, which may be triggered by genetic factors and increased dietary intake of purine-rich foods (Fadhla et al., 2023). Uric acid levels can be controlled through pharmacological treatment or herbal remedies available in the surrounding environment, such as red coconut (kelapa wulung). Red coconut (*Cocos nucifera* var. *rubescens*) is characterized by a pink to reddish coloration of its mesocarp (husk). The outer skin (epicarp) is not always green and may also appear yellowish. Red coconut has traditionally been used to treat various diseases due to its content of bioactive compounds (Mulyanto et al., 2019). From a health perspective, red coconut contains bioactive compounds such as flavonoids and tannins. This variety is known to have a higher tannin content, which enhances its medicinal properties. Due to its rich nutritional composition, red coconut offers numerous health benefits, and its nutrient content is considered higher and more beneficial compared to other coconut varieties (Sirisangsawang & Phetyim, 2023; Rozani et al., 2024).

Flavonoids are a group of natural compounds with a basic phenolic structure and belong to the class of secondary metabolites commonly found in various plant parts, including fruits, vegetables, seeds, bark, roots, stems, and flowers. Flavonoids provide a wide range of health benefits and are widely applied in nutraceutical,

pharmaceutical, medicinal, and cosmetic fields. These benefits are associated with their antioxidant, anti-inflammatory, and antimutagenic properties (Fitri & Putra, 2021). Tannins are natural compounds containing free phenolic hydroxyl groups and are capable of forming stable complexes with proteins. Generally, tannins are used as astringent agents. They are beneficial for treating digestive tract disorders, skin abrasions, and as antidotes for glycoside and alkaloid poisoning, as well as reagents in the distillation of gelatin, proteins, and alkaloids. Tannins are commonly found in plant parts such as leaves, fruits, bark, and branches. In addition to their use in the leather tanning industry, tannins are widely utilized in medicine to treat diarrhea, stop bleeding, manage hemorrhoids, and treat various other diseases (Listiana et al., 2022; Tanaka, 2025). This study aimed to determine the effect of red coconut tembuluk extract (*Cocos nucifera* var. *rubescens*) on reducing uric acid levels in mice (*Mus musculus*).

MATERIALS AND METHODS

This study was conducted at the Biology Education Laboratory, Faculty of Teacher Training and Education, Tadulako University, from September to October 2025. The study employed an experimental laboratory research design aimed at determining the effect of red coconut tembuluk extract (*Cocos nucifera* var. *rubescens*) on the reduction of uric acid levels in mice (*Mus musculus*). A total of 28 male mice aged 2–4 months, weighing 20–40 g, and free from physical abnormalities were used as experimental animals. The mice were randomly divided into seven groups consisting of three control groups normal control (KN), negative control (K−), and positive control (K+) and four treatment groups (PI, PII, PIII, and PIV), with three replications. Each group consisted of four mice.Prior to the experiment, the mice were acclimatized for seven days at the Biology Education Laboratory, Universitas Tadulako, to allow adaptation to the new environment and minimize stress. During the acclimatization period, the mice were maintained under standard laboratory conditions and provided with food and water ad libitum.

Procedures

Preparation of Chicken Liver Juice

The inducing agent used in this study was chicken liver prepared in the form of juice. The chicken liver juice was freshly prepared daily for 30 days. The ratio of chicken liver weight to water was 1:3. The dose of chicken liver juice administered orally to mice to induce hyperuricemia was 0.5 mL per 20 g body weight, given once daily.

Preparation of Red Coconut Tembuluk Extract

Red coconut (*Cocos nucifera* var. *rubescens*) tembuluk used in this study was obtained from community plantations in North Maninili Village, South Tinombo District, Parigi Moutong Regency. The harvested red coconut tembuluk was collected and washed thoroughly with clean running water. It was then sliced into small, thin pieces and dried by air circulation in a closed room without direct sunlight exposure for one week to reduce moisture content.

After drying, the red coconut tembuluk was weighed to the required amount (1 kg) and ground into a fine powder using a blender. The powdered material was extracted using the maceration method with 96% ethanol (2 L) for 3 × 24 hours. The macerate was then filtered using a Büchner funnel and filter paper. The resulting filtrate was evaporated using a rotary evaporator at 40°C and 96 rpm. The obtained extract was homogenized using a shaker for 30 minutes and subsequently prepared into extract concentrations of 10%, 20%, 40%, and 80%.

Administration of Test Materials

The test materials were administered orally by gavage, with different treatments applied to each group. A total of seven experimental groups were used, consisting of three control groups—normal control (KN), negative control (K−), and positive control (K+) and four treatment groups (PI, PII, PIII, and PIV). Each group consisted of four mice with three replications. Prior to treatment, the mice were acclimatized for seven days at the Biology Education Laboratory, Universitas Tadulako. On day 7, baseline uric acid levels were measured. From day 8 to day 21, the normal control group (KN) was provided food and water ad libitum, while the negative control (K−), positive control (K+), and all treatment groups (PI–PIV) were induced with lard fat. On day 21, uric acid levels were measured again.

From day 22 to day 28, the positive control group (K+) was administered allopurinol at a dose of 0.026 mg per 20 g body weight. Uric acid levels were measured on day 28 following allopurinol administration. Subsequently, from day 29 to day 35, the normal control (KN) and negative control (K−) groups received food and water ad libitum, while treatment groups PI, PII, PIII, and PIV were administered red coconut tembuluk extract at different concentrations. Treatment group PI received 10% extract, PII received 20%, PIII received 40%, and PIV received 80% extract, administered for seven days. Blood samples were collected on day 35 from mice that had received red coconut tembuluk extract. The final blood sampling was conducted on day 42 for all groups (KN, K−, K+, PI, PII, PIII, and PIV) to evaluate uric acid levels before induction, after lard fat induction, following allopurinol treatment, and after administration of red coconut tembuluk extract. Prior to blood collection, the mice were fasted for eight hours.

RESULTS AND DISCUSSION

The results of this study on the effect of red coconut tembuluk extract (*Cocos nucifera* var. *rubescens*) on uric

acid reduction in mice (*Mus musculus*) showed the mean blood uric acid levels across the experimental groups, including the normal control group (KN), positive control group (K+), and negative control group (K−). Treatment group 1 (PI) received the extract at 10%, treatment group 2 (PII) at 20%, treatment group 3 (PIII) at 40%, and treatment group 4 (PIV) at 80%. These results are presented in Figure 1.

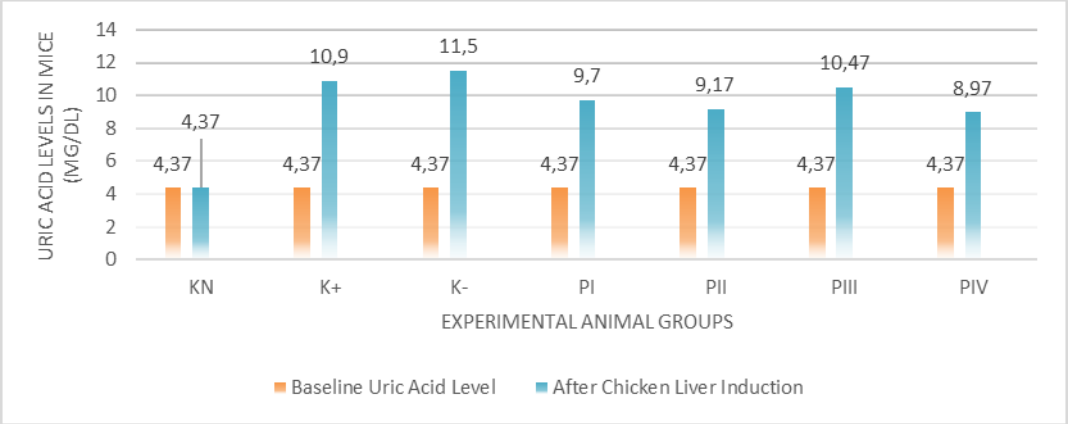

Figure 1. Mean uric acid levels before and after chicken liver induction.

Based on the data presented in Figure 1, the mean uric acid levels in mice before and after chicken liver induction can be observed. After induction with chicken liver, mice exhibited a marked increase in mean uric acid levels. In the positive control group (K+), the mean uric acid level increased from 4.37 mg/dL before induction to 10.9 mg/dL after induction. Similarly, in the negative control group (K−), the mean uric acid level increased from 4.37 mg/dL to 11.5 mg/dL. In treatment group 1 (PI), the mean uric acid level increased from 4.37 mg/dL before induction to 9.7 mg/dL after induction. In treatment group 2 (PII), the mean uric acid level increased from 4.37 mg/dL to 9.17 mg/dL. In treatment group 3 (PIII), the mean uric acid level increased from 4.37 mg/dL to 10.47 mg/dL, while in treatment group 4 (PIV), the mean uric acid level increased from 4.37 mg/dL to 8.97 mg/dL after induction.

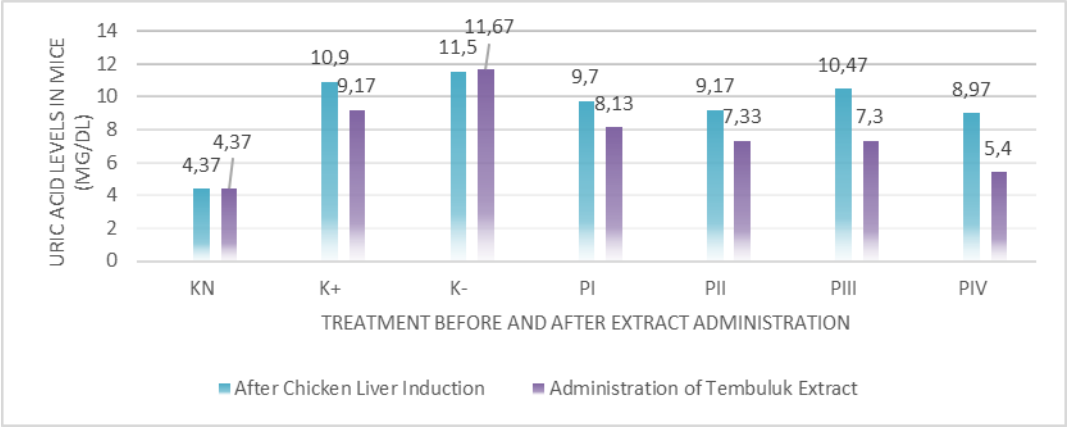

Figure 2. Mean uric acid levels before and after administration of red coconut tembuluk extract (*Cocos nucifera* var. *rubescens*).

Based on the data presented in Figure 2, the mean uric acid levels in mice before and after administration of red coconut tembuluk extract (*Cocos nucifera* var. *rubescens*) indicate a reduction in uric acid levels following extract treatment. In treatment group 1 (PI), the mean uric acid level decreased from 9.7 mg/dL before extract administration to 8.13 mg/dL after administration of the extract at a concentration of 10%. In treatment group 2 (PII), the mean uric acid level decreased from 9.17 mg/dL to 7.33 mg/dL after administration of the extract at a concentration of 20%. In treatment group 3 (PIII), the mean uric acid level decreased from 10.47 mg/dL before treatment to 7.30 mg/dL after administration of the extract at a concentration of 40%. In treatment group 4 (PIV), the mean uric acid level decreased from 8.97 mg/dL to 5.40 mg/dL after administration of the extract at a concentration of 80%.

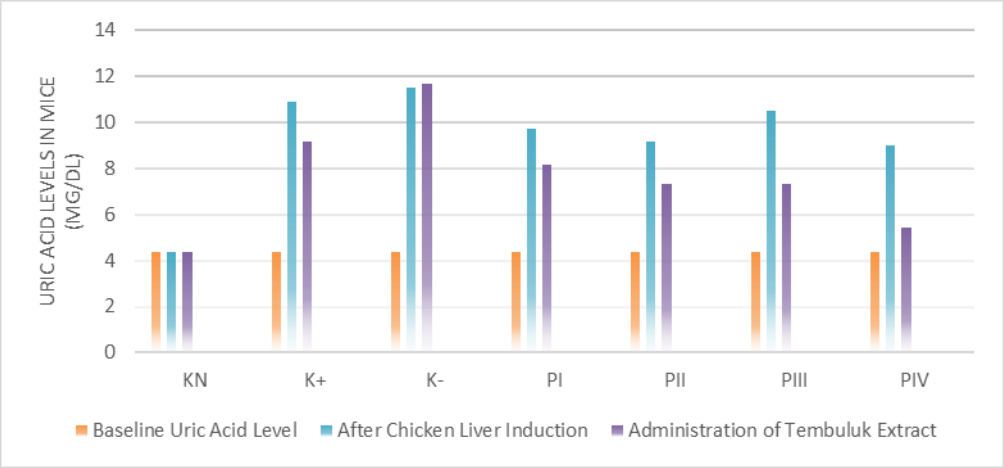

Figure 3. Comparative diagram of mean uric acid levels in mice before chicken liver induction, after chicken liver induction, and after administration of red coconut tembuluk extract (*Cocos nucifera* var. *rubescens*).

In treatment group 1 (PI), the mean uric acid level in mice was 4.37 mg/dL before chicken liver induction and increased to 9.7 mg/dL after induction. Following administration of red coconut tembuluk extract at a concentration of 10%, the uric acid level decreased to 8.13 mg/dL. In treatment group 2 (PII), the mean uric acid level increased from 4.37 mg/dL before induction to 9.17 mg/dL after induction, and subsequently decreased to 7.33 mg/dL after administration of the extract at a concentration of 20%. In treatment group 3 (PIII), the mean uric acid level increased from 4.37 mg/dL before induction to 10.47 mg/dL after induction, and then decreased to 7.30 mg/dL following administration of the extract at a concentration of 40%. In treatment group 4 (PIV), the mean uric acid level increased from 4.37 mg/dL to 8.97 mg/dL after induction, and decreased to 5.40 mg/dL after administration of the extract at a concentration of 80%. In the positive control group (K+), the mean uric acid level increased from 4.37 mg/dL before induction to 10.9 mg/dL after chicken liver induction. After treatment with allopurinol at a dose of 0.026 mg, the uric acid level decreased to 9.17 mg/dL. In the negative control group (K−), the mean uric acid level increased from 4.37 mg/dL before induction to 11.5 mg/dL after induction. Meanwhile, the normal control group (KN) maintained a mean uric acid level of 4.37 mg/dL. Subsequently, the data were analyzed using a one-way ANOVA (Analysis of Variance) to determine the effect of red coconut tembuluk extract on uric acid levels in chicken liver–induced mice. The results of the analysis are presented in the analysis of variance table (Table 1).

Table 1. Results of the analysis of variance (ANOVA) of uric acid levels in mice.

SK	db	Sum of Squares	Mean Square	F value	F table
Treatment	6	93	15,5	12,231	2,836
Galat	14	17,7155556	1,2651804		
Total	20	110,7155556			

Based on the data presented in Table 1, the null hypothesis (H_0) was rejected and the alternative hypothesis (H_1) was accepted, indicating that red coconut tembuluk extract had a significant effect on reducing uric acid levels in chicken liver–induced mice. Subsequently, a Least Significant Difference (LSD) test at a 5% significance level was conducted to determine which treatments differed significantly from each other. The results of the LSD test are presented in Table 2. Based on the results of the Least Significant Difference (LSD) test, an LSD value of 1.9551 was obtained. The findings indicate that uric acid levels in chicken liver–induced mice treated with red coconut tembuluk extract showed significant differences among treatments. Therefore, the second alternative hypothesis (H1) was accepted, indicating that there is an effective concentration of red coconut tembuluk extract capable of reducing uric acid levels in mice after chicken liver induction.

Table 2. Least Significant Difference (LSD) test of uric acid levels in chicken liver–induced mice treated with red coconut tembuluk extract.

Treatment	Treatment Mean	Mean						LSD 0,05
K Normal	4,37							
K+	9,17	4,7*						
K-	11,67	6,7*	2*					
PI	8,13	3,7*	1,1^	3,1*				1,9551
PII	7,33	3,1*	1,7^	3,7*	0,6^			
PIII	7,3	3,0*	1,8^	3,8*	0,7^	0,1^		
PIV	5,4	1,1*	3,7*	5,7*	2,6*	2*	1,9^	

Notes:
** indicates a statistically significant difference ($p < 0.05$)*
^ indicates no statistically significant difference

Discussion

An increase in blood uric acid levels was observed in the mice. This increase was evident from the average blood uric acid level in the negative control group (K−), which was induced with chicken liver juice, reaching 11.5 mg/dL, a value higher than that of the other groups. Following induction with chicken liver juice, blood uric acid levels increased in all treatment groups. Chicken liver juice was used as a hyperuricemia-inducing agent because it is a purine-rich food that can elevate uric acid levels after administration (Wikantyasning et al., 2024). Gouty arthritis, or gout, is a form of joint inflammation caused by the deposition of uric acid crystals in the joints. This disease results from disturbances in purine metabolism. Purines are compounds derived from protein metabolism in the body that are subsequently converted into uric acid. Gouty arthritis is the third most common type of arthritis after osteoarthritis and other inflammatory joint disorders. This condition can significantly affect patients' quality of life due to recurrent joint pain. Gout is typically characterized by repeated episodes of acute arthritis, associated with the formation of large monosodium urate crystals. These conditions may lead to nodal deformities (joint damage) and impaired kidney function due to excessive purine consumption. Consequently, the affected joints become painful, swollen, and inflamed.

Several risk factors contribute to the development of gouty arthritis, including excessive purine intake, alcohol consumption, stress, use of certain medications, obesity, hypertension, and genetic predisposition. Excessive purine intake causes difficulty in renal excretion of uric acid, leading to its accumulation in the joints. Normal purine requirements range from 500–1000 mg per day. Purine intake is considered low if it is below 500 mg/day and excessive if it exceeds 1000 mg/day (Irmawati et al., 2023). Preventive measures to control elevated blood uric acid levels include dietary regulation by limiting high-purine foods, ensuring adequate vitamin and mineral intake, engaging in regular physical activity, smoking cessation, and stress management. In addition, pharmacological therapy may be administered under medical supervision. As an alternative, herbal medicine may also be used to help reduce blood uric acid levels (Wulandari & Yulianto, 2021; Patyawargana & Falah, 2021). The results presented in Figure 2 show that the average blood uric acid level in the positive control group (K+) was lower than that in the negative control group (K−), indicating that allopurinol administration effectively reduced uric acid levels. Allopurinol is a urate-lowering drug that works by inhibiting the xanthine oxidase enzyme. By broadly suppressing xanthine oxidase activity, allopurinol can effectively control and reduce blood uric acid levels (Jannah et al., 2024). Allopurinol belongs to the same class of xanthine oxidase inhibitors as febuxostat and also exhibits renoprotective effects. However, dose adjustment is required in hyperuricemic patients with chronic kidney disease because allopurinol is excreted entirely through the kidneys. The initial dose of allopurinol is typically 50–100 mg/day and may be gradually increased to 200–300 mg/day over a period of 2–5 weeks until serum uric acid levels decrease to below 6 mg/dL (Choliq et al., 2025).

Several bioactive compounds are known to reduce uric acid levels, including flavonoids, alkaloids, saponins, tannins, and other secondary metabolites. Flavonoids are polyphenolic compounds classified as secondary metabolites in plants. Numerous pharmacological activities of flavonoids have been reported, such as antioxidant, anti-inflammatory, and anticancer effects. Flavonoids are known to reduce blood uric acid levels by acting as antioxidants that scavenge free radicals. Furthermore, flavonoids can inhibit the activity of xanthine oxidase and xanthine dehydrogenase, thereby suppressing uric acid synthesis. Adequate vitamin C intake is also believed to play a role in preventing hyperuricemia and its progression to gout and hyperuricemia-associated nephropathy (Ernis et al., 2020; Abdulkadir et al., 2022). One natural source with potential uric acid–lowering activity is red coconut tembuluk (*Cocos nucifera* var. *rubescens*). Anggraini et al. (2024) reported that young coconut water has potential uric acid–lowering effects due to its L-arginine content, which exhibits strong anti-inflammatory and antioxidant activities that protect tissues from damage. L-arginine may inhibit inflammation and prevent further tissue damage caused by excessive reactive oxygen

species (ROS) generated during uric acid crystallization. The L-arginine content in coconut water has also been shown to significantly reduce free radicals and exhibit antioxidant activity (Zulaikhah, 2019). Similarly, red coconut tembuluk may serve as a natural agent for reducing uric acid levels in mice.

CONCLUSIONS

Based on the results of this study, it can be concluded that red coconut tembuluk extract has a significant effect on reducing uric acid levels in mice induced with chicken liver. The most effective concentration of red coconut tembuluk extract in lowering uric acid levels was 80% (PIV), which resulted in an average uric acid level of 5.4 mg/dL.

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Competing Interests: The authors declare that there are no competing interests.

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