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Purple Sweet Potato Extract Effects on TNF-a and Gut Injury

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ABSTRACT

Purple sweet potato has antioxidant and anti-inflammatory properties with the potential to reduce TNF-α and serve as a modality to prevent Curling's ulcer. The purpose of this study was to demonstrate the effect of oral administration of purple sweet potato (Ipomoea batatas L.) on serum and tissue TNF-α levels in the stomach and duodenum, as well as gastrointestinal damage, in Wistar rats (Rattus norvegicus) with major burns. This experimental study employed a Randomized Post-Test Only Control Group Design using Wistar rat subjects, where the research sample was divided into a control group (P0) and treatment group 1 (P1). The results showed that the treatment group had lower serum TNF-α levels than the control group ($p = 0.002$). The decrease in tissue TNF-α levels was also statistically significant ($p = 0.049$), indicating that purple sweet potato extract has a local anti-inflammatory effect on the intestinal mucosa. Statistical analysis further revealed that the treatment group had a higher mucosal thickness than the control group, with a mean difference of 270.32 µm ($p < 0.001$). There was no significant difference in villi width between the control and treatment groups. The inflammation lesions parameter showed a significant difference between the groups, with a p value of 0.001. The reduction in TNF-α levels and increase in mucosal thickness suggest that this extract has a protective effect on the intestine under inflammatory conditions caused by burns.

KEYWORDS *Duodenal mucosal thickness, duodenal villi width, gastric and duodenal tissue TNF-α, hemorrhagic lesions, inflammation lesions, major burns, necrotic lesions, serum TNF-α*

INTRODUCTION

Major burns are characterized by extensive tissue damage and able to pose serious risks to survival due to complications such as infection, multi-organ dysfunction, and systemic inflammatory responses. One key mediator implicated in these outcomes is the pro-inflammatory cytokine TNF-α, which exacerbates tissue injury and impairs healing. Gastrointestinal tract damage, often underestimated, emerges as a common complication in burn patients, especially following sepsis and organ failure. Splanchnic hypoperfusion, which may reduce intestinal blood flow by up to 60%, contributes to mucosal ischemia, bacterial translocation, and the development of stress-related ulcers such as Curling's ulcers (McCann et al., 2022; Nielson et al., 2017).

Curling's ulcers, associated with mucosal barrier breakdown, affect approximately 1.5% of major burn patients globally according to recent studies from the American Burn Association, and up to 15% in settings lacking ulcer prophylaxis according to WHO burn injury prevention guidelines. These ulcers are triggered by oxidative stress, inflammatory mediators, and acidic gastric secretions, often accompanied by splanchnic hypoperfusion and compromised gut barrier integrity (Siddiqui et al., 2019; Sinaga et al., 2013). The mortality rate associated with gastrointestinal bleeding in burn patients can reach 40-60% when complicated by sepsis, making prevention strategies critically important. While pharmacologic

interventions—such as PPIs, H2 antagonists, and sucralfate—are the mainstay of treatment, prophylaxis remains vital, especially in patients contraindicated for invasive procedures.

Purple sweet potato (Ipomoea batatas L.) has gained attention due to its rich antioxidant and anti-inflammatory phytochemical profile, particularly anthocyanins and polysaccharides. Recent studies have shown that purple sweet potato polysaccharides (PSPPs) can reduce TNF-α, IL-6, and IL-1β while enhancing anti-inflammatory IL-10 levels, restoring gut microbiota balance, and exerting mucosal healing effects in animal models of colitis and inflammatory bowel disease. However, specific studies investigating purple sweet potato effects in burn-induced gastrointestinal injury remain limited. Previous research has primarily focused on general antioxidant effects in metabolic disorders, with minimal exploration of its therapeutic potential in stress ulcer prevention or burn-related gastrointestinal complications (Sun et al., 2020).

Most studies have been conducted overseas with limited exploration in Indonesia, despite purple sweet potatoes being a locally abundant commodity with potential for developing evidence-based complementary medicine. Despite promising findings, there is limited research exploring the prophylactic role of anthocyanin-rich natural agents against gastrointestinal injury in burn models, particularly in the Indonesian context (Ermis et al., 2023; Serafim et al., 2021; Izalin et al., 2016). The use of local ingredients such as purple sweet potatoes represents an innovation in complementary therapy with low costs, high availability, and potential to support evidence-based approaches to traditional medicine in Indonesia.

Based on these gaps, this research aims to investigate the effect of oral administration of purple sweet potato tuber extract on TNF-α levels and gastrointestinal tract integrity in Wistar rats (Rattus norvegicus) subjected to major burns. This research contributes to developing cost-effective, locally-sourced therapeutic options for burn management while providing scientific evidence for traditional medicine applications in Indonesian healthcare settings.

METHOD

This study was a randomized post-test only control group design research using Wistar rats divided into two groups: a control group (P0) and an intervention group (P1). The inclusion criteria for the Wistar rats were male, able to eat and active, aged between two to three months, and weighing 180–220 grams. Dropout rats were those that died during the research. Each group consisted of 14 rats. The P1 group was given purple sweet potato extract, prepared by maceration using ethanol, at a dose of 1 gram/kgBW/day for one month. All Wistar rats were given pellet food containing protein (20%), fat (5%), carbohydrates (45%), fiber (5%), vitamins, and minerals, at 12–20 grams per day.

The extract was made using purple sweet potatoes (Ipomoea batatas L.) sourced from a single location: Bukian Village, Payangan District, Gianyar Regency, with a total weight of 3 kg. The tubers were thoroughly washed and thinly sliced to facilitate drying. The slices were oven-dried at 60°C until completely dehydrated, then ground into a fine powder using a blender. Extraction was performed via maceration. Two kilograms of the powdered tuber were soaked in 96% ethanol (3 L) for 72 hours (3 × 24 hours). The mixture was then filtered using Whatman filter paper. The resulting filtrate was evaporated using a rotary evaporator at a heating bath temperature of 40°C to obtain the purple sweet potato tuber extract. If not used immediately, the extract was stored in a refrigerator at approximately 4°C.

After one month, the rats were given a burn injury covering 25% of the total body surface area (TBSA). The rats had been anesthetized previously using intraperitoneal ketamine (90 mg/kg). The back of the necks of the rats was shaved up to the tail. The burn injury was induced using a steel ingot heated in boiling water at 100°C and applied to the shaved area, creating a third-degree burn injury. Blood samples were then taken from the Wistar rats to examine TNF-α levels, and the rats were euthanized to collect gastrointestinal tract samples for TNF-α level analysis and histopathological imaging examination.

Data were analyzed using SPSS version 25 to evaluate the effects of oral purple sweet potato (Ipomoea batatas L.) extract on serum and tissue TNF-α levels, as well as gastrointestinal histopathological changes in Wistar rats with major burns. Descriptive statistics summarized each variable such as serum and tissue TNF-α, duodenal mucosal thickness, villi width, and gastric lesions (necrosis, hemorrhage, inflammation) using mean, SD, median, and range.

Normality was assessed using the Shapiro-Wilk test ($p > 0.05$ = normal). Homogeneity of variance was tested with Levene's test for normally distributed data ($p > 0.05$ = homogeneous). Comparative tests between groups depended on these results: normally distributed and homogeneous data were analyzed using Independent T-test, while non-homogeneous data used the corrected T-test. Non-normally distributed data were tested with the Mann-Whitney U test. All statistical tests considered $p \leq 0.05$ as significant, ensuring valid test selection based on the data characteristics.

RESULT AND DISCUSSION

The subjects' characteristics can be observed in Table 1. The mean serum TNF-α level in the control group was 185.02 ± 78.88 ng/ml, higher than the treatment group, which had a mean of 108.96 ± 39.55 ng/ml. Data distribution in both groups was non-normal ($p = 0.029$ and 0.015). TNF-α levels in gastric and duodenal tissues were also higher in the control group (66.26 ± 34.66 ng/ml) compared to the treatment group (44.41 ± 18.92 ng/ml), with both datasets showing normal distribution ($p = 0.115$ and 0.971). The duodenal mucosal thickness was greater in the treatment group (1450.45 ± 81.24 µm) compared to the control (1180.13 ± 179.39 µm), with both showing normal distribution. In contrast, villus width data were non-normally distributed in the control group ($p < 0.001$) but normally distributed in the treatment group ($p = 0.101$).

Mucosal necrosis and hemorrhagic lesions showed non-normal distribution in both groups. Inflammatory lesions were normally distributed in the control group ($p = 0.111$), but not in the treatment group ($p = 0.018$). Overall, most variables did not follow a normal distribution, thus warranting the use of non-parametric statistical tests for further analysis. Homogeneity testing was conducted on variables with normally distributed data. Levene's test showed that TNF-α tissue levels had a p-value of 0.089, indicating homogeneous variance between groups ($p > 0.05$). In contrast, duodenal mucosal thickness had a p-value of 0.024, suggesting non-homogeneous variance ($p < 0.05$).

Comparative analysis revealed significant differences in several research variables between the control and treatment groups. For serum TNF-α levels, the control group had a mean of 185.02 ± 78.88 ng/ml, while the treatment group showed a lower mean of 108.96 ± 39.55 ng/ml. The Mann-Whitney test indicated a significant difference with a p-value of 0.002,

suggesting that oral administration of purple sweet potato tuber extract effectively reduced serum TNF-α levels. For tissue TNF-α levels, the control group had a mean of 66.26 ± 34.66 ng/ml, while the treatment group showed a lower mean of 44.41 ± 18.92 ng/ml. Independent T-Test analysis showed a mean difference of 21.84 ng/ml with a 95% confidence interval (0.14 – 43.54 ng/ml) and a standard error of 10.55 ng/ml. This difference was statistically significant (p = 0.049), indicating that the extract also significantly reduced tissue TNF-α levels.

Regarding mucosal thickness, the control group had a mean of 1180.13 ± 179.39 µm, while the treatment group had a higher mean of 1450.45 ± 81.24 µm. Independent T-Test analysis revealed a mean difference of 270.32 µm with a 95% confidence interval (162.13 – 378.50 µm) and a standard error of 52.63 µm. This difference was significant (p < 0.001), indicating that the extract significantly increased mucosal thickness. For villi width, the control group had a mean of 161.48 ± 160.75 µm, whereas the treatment group showed a lower mean of 106.25 ± 21.51 µm. The Mann-Whitney test showed no significant difference (p = 0.129), suggesting that the extract had no significant effect on villi width. The Mann-Whitney test showed a significant difference in mucosal necrosis lesions. The control group had a mean of 1.53 ± 0.43, while the treatment group was lower at 0.40 ± 0.16 (p < 0.001), indicating a significant reduction in necrosis severity with treatment.

For hemorrhagic lesions, the control group had a mean of 1.16 ± 0.45, compared to 0.23 ± 0.21 in the treatment group. The difference was statistically significant (p < 0.001), indicating a marked reduction in hemorrhagic damage following treatment. Similarly, for inflammatory lesions, the control group had a mean of 1.51 ± 0.26, while the treatment group was lower at 1.01 ± 0.38. This difference was also significant (p = 0.001), demonstrating a notable anti-inflammatory effect of the extract.

Table 3 Comparative Analysis of Between Subject Groups' Variables

Variables	Groups		Mean difference	IC95%	Standard error	p-value
	Control	Treatment				
Serum TNF-α	185,02 ± 78,88	108,96 ± 39,55	-	-	-	0,002*[b]
Tissue TNF-α	66,26 ± 34,66	44,41 ± 18,92	21,84	0,14 – 43,54	10,55	0,049*[a]
Duodenum mucosal thickness	1180,13 ± 179,39	1450,45 ± 81,24	270,32	162,13 – 378,50	52,63	<0,001*[a]
Diodenum villi width	161,48 ± 160,75	106,25 ± 21,51	-	-	-	0,129[b]
Gastric nectrotic lesion	1,53 ± 0,43	0,40 ± 0,16	-	-	-	<0,001*[b]
Gastric hemorrhagic lesion	1,16 ± 0,45	0,23 ± 0,21	-	-	-	<0,001*[b]
Gastric inflammatory lesion	1,51 ± 0,26	1,01 ± 0,38	-	-	-	0,001*[b]

*Analysis using [a]*Independent T-Test*; [b]Mann-Whitney. Results are significant if p-value ≤0,05.

Figure 1 presents the histopathological analysis of Wistar rat intestines following a major burn model. Intestinal mucosal structures were observed using hematoxylin-eosin (HE) staining, with mucosal thickness and villi width measured as key indicators of tissue integrity. The image shows that the treatment group exhibited greater mucosal thickness compared to the control group, with measurements ranging from 146.11 to 1606.03 micrometers. Additionally, villi width was assessed as another morphological parameter. Although inter-sample variation was observed, the measurements indicated differences in intestinal villi structure in the treatment group, with a range of 107.36 to 146.29 micrometers.

Observations revealed that the control group exhibited reduced mucosal thickness compared to the treatment group, with measurements ranging from 1092.43 to 845.91 micrometers. This thinner mucosa suggests a more pronounced inflammatory impact on the intestinal tissue due to the major burn injury, potentially contributing to increased mucosal permeability and gastrointestinal dysfunction. Additionally, intestinal villi width was measured, ranging from 107.36 to 123.28 micrometers. The relatively larger villi width compared to thetreatment group may indicate morphological alterations associated with inflammation and oxidative stress following the burn injury.

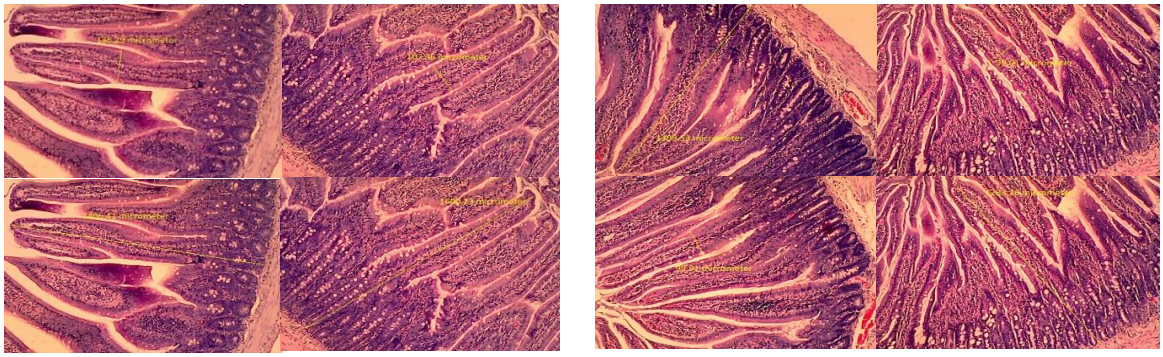

Figure 1. Duodenum Mucosal Thickness and Villi Width in Treatment Group (left) and Control Group (right)

Figure 2 presents the histopathological analysis of gastric lesions in the control group of Wistar rats that experienced major burns without intervention using purple sweet potato (Ipomoea batatas L.) tuber extract. The histological findings reveal significant gastric mucosal damage, characterized by epithelial integrity loss, tissue edema, inflammatory cell infiltration, and submucosal hemorrhage. Major burn injuries are known to trigger a systemic inflammatory response that contributes to burn-induced gastric injury—a pathological condition resulting from elevated levels of pro-inflammatory cytokines such as TNF-α, which impair the gastric mucosal barrier and increase the risk of gastrointestinal bleeding. Gastric lesions such as necrosis, hemorrhage, and inflammation appeared more extensive and severe in the control group, likely associated with increased oxidative stress due to uncontrolled free radical production. The damaged gastric mucosa exhibited degenerative changes, including pyknotic nuclei and cytoplasmic vacuolization, indicating ongoing epithelial cell apoptosis.

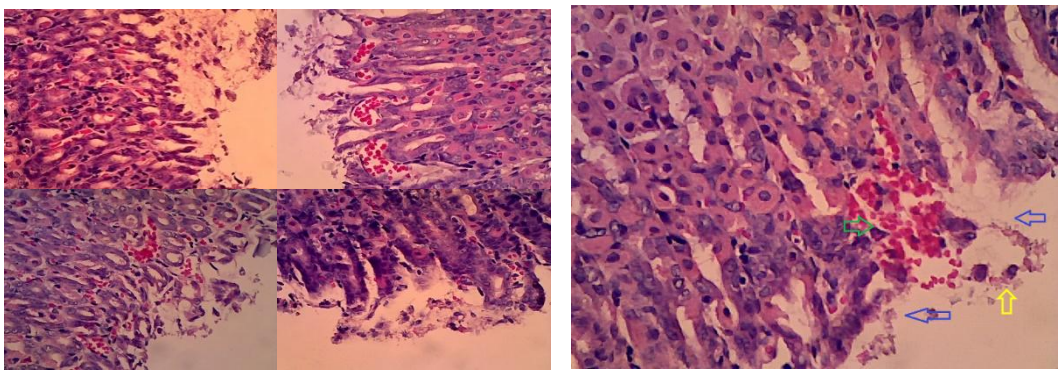

Figure 2. Gastric lesion of control group. Green arrow → hemorrhagic lesion; blue arrows → necrotic lesion; yellow arrow → inflammatory lesion

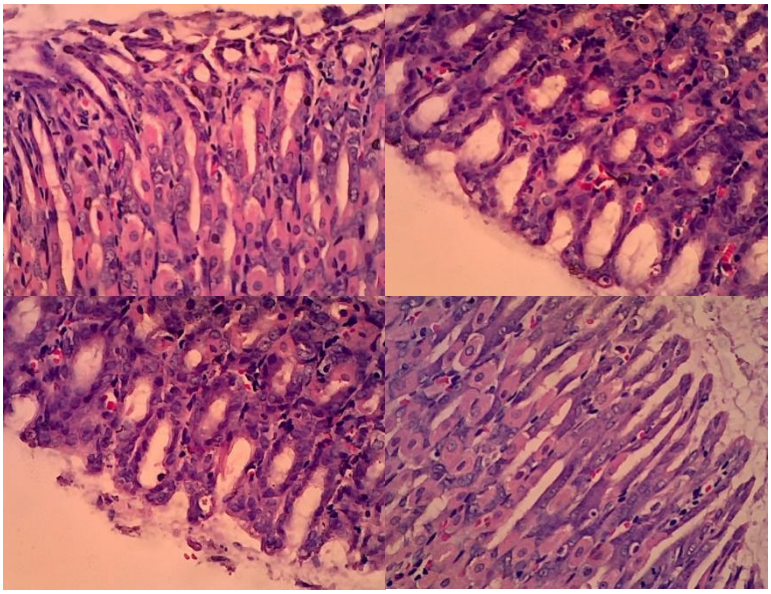

Figure 3. Treatment Group Gastric Lesions

Figure 3 illustrates the histopathological analysis of gastric lesions in the treatment group that received oral administration of purple sweet potato (Ipomoea batatas L.) tuber extract following major burn injury. Overall, the image demonstrates a more preserved gastric mucosal structure compared to the control group, marked by more intact epithelial layers, reduced inflammatory cell infiltration, and decreased tissue edema. The mucosal layer appears better organized, with epithelial cells maintaining their polarity, suggesting that the extract contributed to preserving gastric mucosal integrity against the systemic inflammatory effects triggered by severe burns. The extent of epithelial destruction is visibly lower than in the control group, with fewer areas showing vacuolization and cellular degeneration. These findings indicate that bioactive compounds in the extract, such as anthocyanins and other

phenolic substances, may play a protective role by mitigating oxidative stress and inflammatory responses that lead to mucosal injury.< 0.001)

Discussion

This study found that oral administration of purple sweet potato (Ipomoea batatas L.) extract significantly reduced both serum and tissue TNF-α levels in rats with major burn injury. This anti-inflammatory effect is attributed to its high anthocyanin content, which suppresses NF-κB and MAPK signaling pathways, thereby reducing inflammatory mediator release. Given TNF-α's central role in systemic inflammatory responses post-burn, its reduction suggests a protective modulation of immune activity (Kano et al., 2005).

Moreover, the extract enhanced duodenal mucosal thickness ($p < 0.001$) without significantly affecting villus width ($p = 0.129$). Anthocyanins are known to support epithelial regeneration and gut barrier integrity by influencing growth factor expression and tight junction stability. Although villus morphology remained largely unchanged, this could be due to the short treatment duration or delayed morphologic adaptation (Izalin et al., 2016; Alam et al., 2022).

The extract appears to protect intestinal structure and mitigate inflammatory damage post-burn, likely through combined antioxidant and immunomodulatory effects. These findings highlight its potential as an adjunctive therapy in severe burn management (Yahfoufi et al., 2018; Taherkhani et al., 2021).

Burn-induced gastrointestinal mucosal necrosis is a result of oxidative stress and systemic inflammation disrupting epithelial integrity, increasing the risk of bacterial translocation. In this study, mucosal necrosis scores were significantly reduced in the treatment group compared to controls (1.53 ± 0.43 vs 0.40 ± 0.16; $p<0.001$), reflecting the extract's protective role. Anthocyanins in purple sweet potato, particularly cyanidin and peonidin have demonstrated antioxidative and NF-κB-inhibiting properties (de los Ángeles Rosell et al., 2024), while also enhancing antioxidant enzymes such as Hmox1 and Gclc to maintain mucosal structure (Esatbeyoglu et al., 2017).

Hemorrhagic lesions, commonly linked to increased vascular permeability and endothelial damage, were also mitigated (control: 1.16 ± 0.45 vs treatment: 0.23 ± 0.21; $p<0.001$), suggesting microvascular protection. This supports findings by Rosell, who reported reduced MDA levels post-extract, and aligns with evidence of anthocyanin-induced vascular stability (de los Ángeles Rosell et al., 2024).

Inflammatory lesion scores were significantly lower in the treatment group (1.01 ± 0.38 vs 1.51 ± 0.26; $p=0.001$), indicating suppression of macrophage overactivation. This corresponds with prior studies showing anthocyanin-mediated downregulation of TNF-α, iNOS, and COX-2 (Kang et al., 2018; Jiang et al., 2022).

CONCLUSION

This research demonstrates that oral administration of purple sweet potato extract significantly reduces both serum and tissue TNF-α levels in burn-injured rats, while improving intestinal mucosal integrity through increased mucosal thickness and reduced gastrointestinal lesions. The extract shows promise as an adjuvant therapy for reducing systemic inflammation and preserving intestinal integrity post-burn, offering a cost-effective, locally-sourced

therapeutic option for burn management in Indonesian healthcare settings. Future clinical trials (RCTs) are needed to confirm efficacy and safety in humans, with additional research exploring mechanisms involving IL-6, CRP, NF-κB, epithelial junction proteins (e.g., occludin, claudin-1), and gut microbiota modulation to optimize dosage and application in burn care and gastrointestinal protection.

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