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RESEARCH ARTICLE

Purple Sweet Potato (*Ipomoea batatas* L.) Mitigates Overtraining-Induced Physiological Stress and Inflammation

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Abstract

Repeated high-intensity physical exercise induces overtraining by generating excess reactive oxygen species (ROS) and activating systemic inflammatory pathways, leading to oxidative damage and impaired physical endurance. In this state, malondialdehyde (MDA) and proinflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor alpha (TNF-α), and transforming growth factor beta (TGF-β) are elevated, while endogenous antioxidant defenses including superoxide dismutase (SOD) are suppressed. This study aimed to investigate the mechanisms underlying purple sweet potato (*Ipomoea batatas L.*) modulation of oxidative stress, inflammatory signaling, and fatigue resistance in an overtrained Wistar rat model. Using a true experimental, randomized controlled pre-test–post-test design conducted with ethical approval from the Research Ethics Committee of the Faculty of Medicine, Universitas Sebelas Maret (No. 14/UN27.06.11/KEP/EC/2025) and in accordance with ARRIVE 2.0 guidelines, 36 male Wistar rats (150 - 250 g) were assigned into 6 groups (n = 6): Normal control, negative control (overtraining only), positive control (vitamin C 1.6 mg/200 g/day), and three treatment groups receiving purple sweet potato at 100, 200, and 300 mg/kg body weight/day. After a 7-day acclimatization period, overtraining was induced on a treadmill (20 m/min, 30 min, 10° incline) for 5 weeks. Plasma levels of MDA, SOD, IL-6, TNF-α, and TGF-β were measured by enzyme-linked immunosorbent assay (ELISA), and fatigue time was assessed by a treadmill exhaustion test. Data were analyzed using paired-samples t-test and one-way ANOVA with Tukey's post hoc test ($p < 0.05$). Purple sweet potato administration dose-dependently reduced MDA levels from 6.07 ± 0.24 to 3.02 ± 0.19 nmol/mL, IL-6 from 81.33 ± 5.27 to 61.66 ± 4.32 pg/mL, TNF-α from 14.30 ± 0.51 to 6.85 ± 0.29 pg/mL, and TGF-β from 13.29 ± 0.46 to 8.07 ± 0.38 pg/mL, while increasing SOD activity from 44.12 ± 3.83 to 71.32 ± 2.75 U/mL at the highest dose (300 mg/kg body weight). Fatigue time increased significantly from 32.14 ± 1.76 min (100 mg/kg body weight) to 45.19 ± 1.64 min (300 mg/kg body weight), approaching normal control values (48.89 ± 1.20 min). All changes were statistically significant ($p < 0.05$) and showed a clear dose-dependent trend. Mechanistically, purple sweet potato likely attenuates exercise-induced oxidative stress through the action of its bioactive anthocyanins and polyphenols, which scavenge ROS, enhance SOD-mediated dismutation of superoxide radicals, and upregulate endogenous antioxidant defenses. Concurrently, these compounds appear to suppress proinflammatory cytokine production by modulating key inflammatory pathways, including NF-κB and related cytokine signaling, thereby

reducing systemic inflammation and protecting muscle and metabolic tissues. In conclusion, purple sweet potato supplementation mitigates oxidative stress and inflammation-related damage while improving antioxidant capacity and endurance in an overtrained rat model, supporting its potential as a natural, mechanism-based intervention for overtraining-related fatigue and inflammation.

Keywords: Oxidative stress, Cytokines, Antioxidant capacity, Exercise fatigue, Animal model

Introduction

Physical exercise plays an important role in maintaining health and physical fitness. However, excessive training without adequate recovery may lead to overtraining syndrome, characterized by decreased physical performance, chronic fatigue, and physiological and biochemical disturbances [1]. Overtraining is a complex condition involving metabolic imbalance, hormonal dysregulation, increased inflammation, and reduced capacity to maintain homeostasis [2]. During exercise, the body undergoes physiological adaptations, including improved mitochondrial function and oxygen utilization. However, excessive training load without sufficient recovery can induce chronic physiological stress, disrupting redox balance. This condition results in increased production of reactive oxygen species (ROS) and decreased antioxidant defense capacity, ultimately triggering oxidative stress and systemic inflammation [3,4].

Oxidative stress plays a central role in overtraining. Increased ROS production, mainly from mitochondrial activity, can cause lipid peroxidation, protein damage, and DNA alterations [5]. Malondialdehyde (MDA) is a widely used biomarker of lipid peroxidation, indicating cellular damage under oxidative stress conditions [5,6]. In contrast, superoxide dismutase (SOD) represents a key endogenous antioxidant enzyme that protects cells from oxidative damage. An increase in MDA levels accompanied by a decrease in SOD activity is a hallmark of oxidative stress in overtraining conditions [7]. In addition to oxidative stress, overtraining is closely associated with inflammatory responses. Excessive ROS can activate nuclear factor kappa B (NF-κB), leading to increased expression of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) [8]. These cytokines contribute to tissue damage, impaired muscle function, and decreased physical performance. Furthermore, chronic inflammation may increase transforming growth factor-beta (TGF-β), which is associated with tissue fibrosis and impaired regeneration [9]. The interaction between oxidative stress and inflammation reflects a disruption of physiological homeostasis during excessive exercise.

Natural antioxidants have been widely investigated as potential strategies to reduce oxidative stress and inflammation. Purple sweet potato (*Ipomoea batatas* L.) is rich in anthocyanins, flavonoids, and phenolic compounds, which exhibit strong antioxidant and anti-inflammatory properties [10]. Anthocyanins have been reported to scavenge free radicals, inhibit NF-κB activation, and enhance endogenous antioxidant defense systems [11]. Previous studies have shown that purple sweet potato extract may increase antioxidant enzyme activity under stress conditions, suggesting its potential to mitigate oxidative damage. Physical performance, particularly fatigue resistance, is an important parameter in overtraining studies. Exhaustion time reflects an organism's ability to sustain physical activity and is influenced by oxidative stress, inflammation, and energy metabolism. A reduction in exhaustion time is a common indicator of overtraining due to impaired muscle function and increased physiological stress [12,13].

Although the antioxidant properties of purple sweet potato have been widely reported, studies investigating its effects on oxidative stress, inflammatory biomarkers (MDA, SOD, IL-6, TNF-α, TGF-β), and physical performance in overtraining conditions remain limited. Therefore, this study aimed to evaluate the effect of purple sweet potato supplementation on oxidative stress, inflammatory biomarkers, and fatigue time in a Wistar rat overtraining model. It is hypothesized that purple sweet potato supplementation reduces oxidative stress and inflammatory responses while improving antioxidant activity and physical endurance.

Materials and methods

Experimental design

This study was a randomized, controlled, in vivo experiment conducted with 36 male Wistar rats (Rattus norvegicus) weighing 150 - 250 g, obtained from a certified supplier of laboratory animals in Solo, Central Java, Indonesia. The animals were housed under standard conditions with a 12-hour light–dark cycle and controlled temperature (22 - 25 °C), with free access to standard rodent chow and drinking water. To minimize stress-related physiological confounders, rats underwent a 1-week acclimatization period prior to the intervention. Following acclimatization, the animals were randomly assigned into 6 experimental groups (n = 6 per group) using a computer-generated randomization table: Normal control (no overtraining, no supplementation), negative control (overtraining only), positive control (overtraining + 1.6 mg/200 g body weight/day vitamin C), and three treatment groups receiving purple sweet potato at doses of 100, 200, and 300 mg/kg body weight/day, respectively. The sample size was determined based on the Federer formula for completely randomized experimental designs: $(t - 1)(n - 1) \geq 15$, where t is the number of groups and n is the number of animals per group. For this study, with $t = 6$ groups, the minimum required sample size per group is: $(6 - 1)(n - 1) \geq 15 \Rightarrow 5(n - 1) \geq 15 \Rightarrow n \geq 4$. Thus, a minimum of 4 animals per group is required. We used 6 animals per group to increase statistical reliability and account for potential biological variability and experimental loss, while adhering to ethical principles of animal reduction. All experimental protocols were approved by the Animal Research Ethics Committee of the Faculty of Medicine, Universitas Sebelas Maret (No. 14/UN27.06.11/KEP/EC/2025) and were conducted in accordance with ARRIVE 2.0 guidelines. To account for circadian variability, all interventions were administered between 08:00 and 12:00, and animals were monitored daily for behavioral distress or significant fluctuations in body weight.

Overtraining protocol

Overtraining was induced using a motorized treadmill exercise regimen (treadmill speed: 20 m/min, 30 min per session, 10° incline), performed five times per week for five consecutive weeks. The protocol was designed to gradually increase physical load while maintaining a consistent intensity to avoid acute injury or excessive exhaustion. On non-exercise days, the rats remained in their cages without additional physical stimulation. The overtraining model was validated by comparing baseline and post-intervention performance and biomarker profiles with those of the normal control group. Physical performance and endurance capacity were evaluated by measuring fatigue time using a treadmill exhaustion test performed at the end of the intervention period. For this test, each rat was placed on the treadmill at a starting speed of 15 m/min, with incremental increases of 2 m/min every 2 min until the animal could no longer maintain pace despite gentle manual encouragement. The time from the start of the treadmill run to complete exhaustion was recorded as fatigue time (min). During the test, rats were observed continuously for signs of inability to continue running, including frequent stumbling, prolonged lying down, or failure to respond to gentle prodding.

Preparation of purple sweet potato simplicia

Purple sweet potato (*Ipomoea batatas L.*) tubers were processed into simplicia (dried herbal material) at the Pharmacy Laboratory of Muhammadiyah University of Surakarta (UMS). The raw tubers were initially washed thoroughly with running tap water to remove soil, debris, and surface contaminants. After washing, the tubers were inspected, and damaged, discolored, or externally abnormal specimens were discarded. The selected tubers were then cut into slices approximately 0.5 cm thick to ensure uniform drying and consistent exposure to heat. Drying was carried out in a controlled-temperature drying oven at 40 °C for 8 h to reduce moisture content while preserving bioactive constituents and minimizing thermal degradation. The dried slices were ground into a fine powder using a laboratory grinder and subsequently sieved through a #60 mesh sieve (pore size ≈ 250 μm) to obtain a homogeneous simplicia powder free from large particles or aggregates (**Figure 1**). The resulting powder was stored in light-protected, airtight containers at room temperature until use. For administration, the purple sweet potato simplicia was prepared as an aqueous suspension in distilled water at the appropriate concentrations (100, 200 and 300 mg/kg body weight per day) and given orally by gavage between 08:00 and 09:00 daily throughout the experimental period. Vitamin C

(ascorbic acid) was administered in a similar schedule and volume as the positive control to maintain methodological consistency.

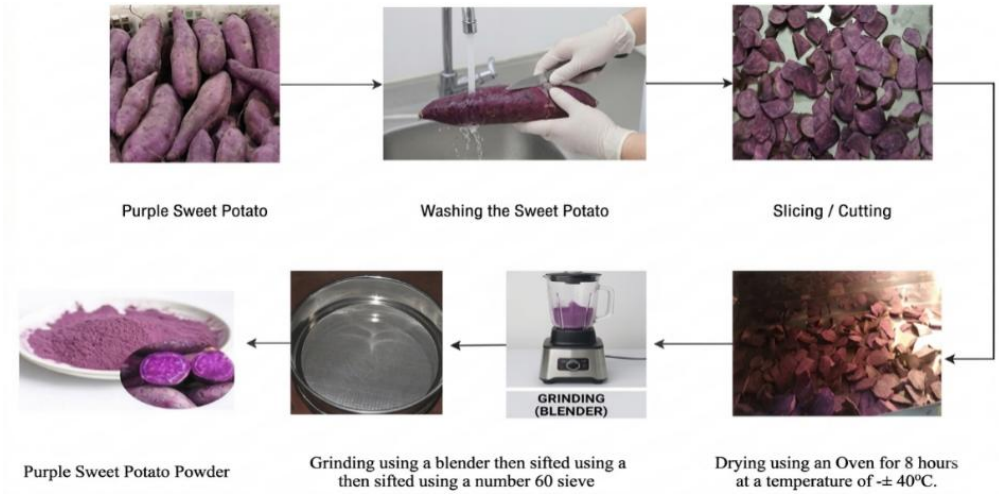


Figure 1 Purple sweet potato simplicia process.

ELISA Assay

At the end of the five-week treatment period, the rats were fasted for 8 h, anesthetized (e.g., by intraperitoneal injection of ketamine–xylazine or according to institutional protocol), and euthanized by exsanguination. Blood samples were collected via cardiac puncture into EDTA-containing tubes and centrifuged at 3,000 rpm for 10 min at 4 °C to obtain plasma. Selected tissue samples (e.g., skeletal muscle and/or liver, depending on the protocol) were rapidly excised, rinsed with ice-cold saline, weighed, snap-frozen in liquid nitrogen, and stored at −80 °C until further analysis. Malondialdehyde (MDA) levels in plasma and/or tissue homogenates were measured as an index of lipid peroxidation and oxidative stress using a commercial ELISA kit (Fine Test, Cat. No. ER1878), following the manufacturer's instructions. Superoxide dismutase (SOD) activity was determined using a commercial ELISA kit (Fine Test, Cat. No. ER1950), with results expressed as units per milliliter (U/mL) or per milligram of protein. Inflammatory biomarkers interleukin-6 (IL-6), tumor necrosis factor alpha (TNF-α), and transforming growth factor beta (TGF-β) were quantified in plasma using commercially available ELISA kits (Fine Test; IL-6: Cat. No. ER0042, TNF-α: Cat. No. ER1393, TGF-β: Cat. No. ER1378) according to the manufacturers' instructions. Absorbance was measured using a microplate reader at the specified wavelengths, and cytokine concentrations were calculated from standard curves generated from the provided reference standards.

Statistical analysis

All quantitative data are presented as mean ± standard deviation (mean ± SD). Data distribution was assessed using the Shapiro–Wilk test, and homogeneity of variances was evaluated using Levene's test. Given the relatively small sample size, normality assessment was interpreted cautiously and supported by visual inspection Q–Q plots. Between-group differences were analyzed using one-way ANOVA, followed by Tukey's Honestly Significant Difference (HSD) test for post hoc comparisons. Statistical analyses were conducted using standard IBM SPSS Statistics for Windows, Version 27.0, with significance set at $p < 0.05$. The graph visualization was created using GraphPad Prism.

Results and discussion

Purple sweet potato attenuates oxidative stress

In the healthy groups, malondialdehyde (MDA) levels remained stable between pretest and posttest (2.12 ± 0.49 vs 2.02 ± 0.18; $p = 0.66$), indicating the absence of significant oxidative stress. In contrast, the negative control group (C-, overtraining without treatment) exhibited a markedly elevated MDA level, increasing from 2.23 ± 0.50 to 11.25 ± 0.35 ($p < 0.001$), which

reflects robust lipid peroxidation secondary to overproduction of ROS during overtraining. Administration of purple sweet potato reduced MDA levels in a clear dose-dependent manner. Post-test MDA values were 6.07 ± 0.24 nmol/mL (P1, 100 mg/kgBW), 4.16 ± 0.14 nmol/mL (P2, 200 mg/kgBW), and 3.02 ± 0.19 nmol/mL (P3, 300 mg/kgBW), all significantly lower than the overtrained untreated group ($p < 0.05$). The reduction in the highest-dose group (P3) approached levels observed in the normal control, with no statistically significant difference, suggesting near-normalization of oxidative stress (**Figure 2(A)**). These findings indicate that the anthocyanin- and flavonoid-rich purple sweet potato effectively suppresses lipid peroxidation by scavenging ROS and reducing oxidative damage [6].

In the C- group, SOD activity declined dramatically after overtraining, decreasing from 85.29 ± 3.83 to 19.85 ± 2.75 U/mL ($p < 0.001$), reflecting exhaustion of the endogenous antioxidant defense system due to chronic free radical exposure. By contrast, daily administration of purple sweet potato significantly restored SOD activity in a dose-dependent fashion: 44.12 ± 3.83 U/mL (P1), 61.02 ± 2.75 U/mL (P2), and 71.32 ± 2.75 U/mL (P3; $p < 0.001$). Although SOD activity in P3 remained statistically lower than in the normal control (mean difference = 9.56; $p = 0.001$), it was markedly higher than in the untreated overtrained group (**Figure 2(B)**). The progressive recovery of SOD suggests that the phenolic compounds and anthocyanins in purple sweet potato may activate the Nrf2 signaling pathway, thereby upregulating antioxidant enzyme expression, including SOD [11].

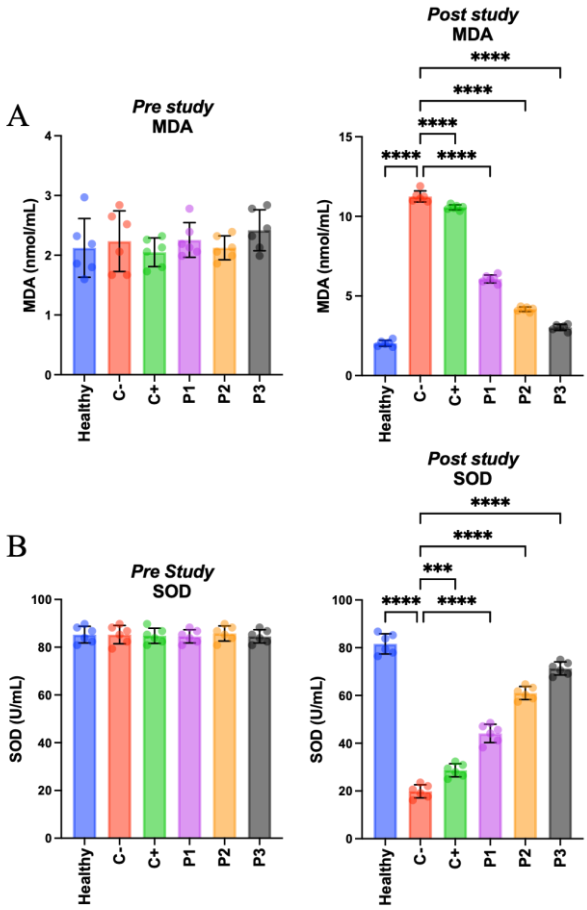


Figure 2 Effects of purple sweet potato on oxidative stress markers in overtrained Wistar rats. (A) Plasma malondialdehyde (MDA) levels across experimental groups in the pre- and post-study. (B) Superoxide dismutase (SOD) activity in the treatment groups in the pre- and post-study. Data are presented as mean ± SD. Different letters indicate significant differences (*$p < 0.05$; ** $p < 0.01$; ***$p < 0.001$; ****$p < 0.0001$) by Tukey's post hoc test. P1 (Purple sweet potato administration of 100 mg/kg body weight/day); P2 (Purple sweet potato administration of 200 mg/kg body weight/day); P3 (Purple sweet potato administration of 300 mg/kg body weight/day).

Purple sweet potato suppresses systemic inflammatory cytokines

Systemic inflammation was evidenced by a substantial increase in TNF-α levels in the negative control group, rising from 5.18 ± 0.29 to 21.04 ± 0.16 pg/mL ($p < 0.001$) after overtraining. Purple sweet potato supplementation significantly attenuated TNF-α levels, with post-test values of 14.30 ± 0.51 pg/mL (P1), 9.24 ± 0.41 pg/mL (P2), and 6.85 ± 0.29 pg/mL (P3). The P3 group exhibited the smallest deviation from the normal control (mean difference = 1.51; $p < 0.001$), indicating a strong anti-inflammatory effect (Figure 3A). The dose-dependent suppression of TNF-α suggests that purple sweet potato bioactives interfere with NF-κB activation, a central pathway in proinflammatory cytokine production.

IL-6 levels in the negative control group surged from 49.00 ± 5.79 to 115.66 ± 3.93 pg/mL ($p < 0.001$), reflecting intense systemic inflammatory activation. Purple sweet potato administration, particularly at the highest dose, markedly reduced IL-6 levels, with the P3 group reaching 61.66 ± 4.32 pg/mL. This value was not statistically different from the normal control (mean difference = 5.00; $p = 0.768$), indicating effective suppression of the proinflammatory cytokine cascade (**Figure 3(B)**). The reduction in IL-6 further supports the role of purple sweet potato in dampening the systemic inflammatory response induced by overtraining.

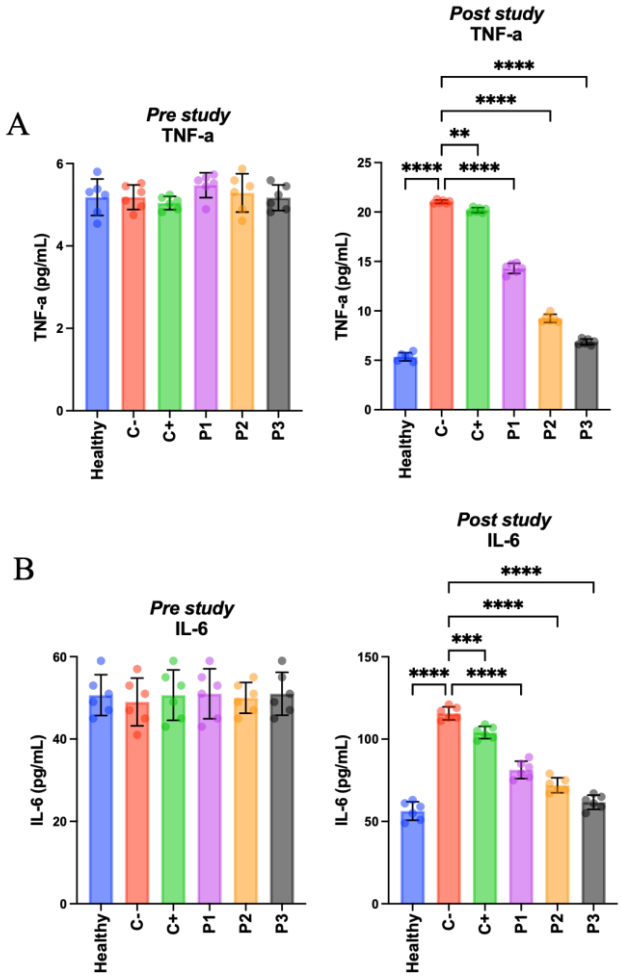


Figure 3 Effects of purple sweet potato on the systemic inflammatory cytokines in overtrained Wistar rats. (A) Plasma TNF-a levels across experimental groups in the pre- and post-study. (B) IL-6 levels in the treatment groups in the pre- and post-study. Data are presented as mean ± SD. Different letters indicate significant differences (*$p < 0.05$; ** $p < 0.01$; ***$p < 0.001$; ****$p < 0.0001$) by Tukey's post hoc test. P1 (Purple sweet potato administration of 100 mg/kg body weight/day); P2 (Purple sweet potato administration of 200 mg/kg body weight/day); P3 (Purple sweet potato administration of 300 mg/kg body weight/day).

Overtraining also induced a dramatic elevation in TGF-β levels, increasing from 4.04 ± 0.39 to 36.29 ± 0.65 pg/mL ($p < 0.001$) in the C-group, suggesting a predisposition toward fibrogenic processes. Purple sweet potato supplementation exerted a dose-dependent protective effect, lowering TGF-β to 13.29 ± 0.46 pg/mL (P1), 9.98 ± 0.31 pg/mL (P2), and 8.07 ± 0.38 pg/mL (P3). Although TGF-β in P3 remained significantly higher than in the normal control (mean difference = 3.45; $p < 0.001$), the marked reduction indicates a strong attenuation of the fibrosis-associated signaling pathway, likely through decreased oxidative stress and chronic inflammation both key activators of TGF-β (**Figure 4**).

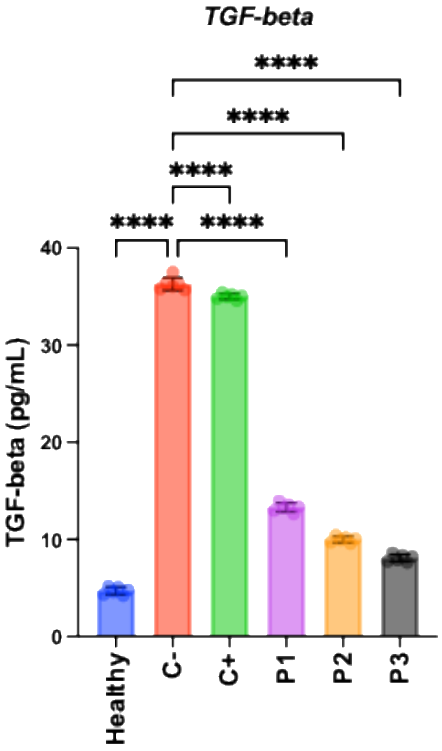


Figure 4 Effects of purple sweet potato on the growth factor TGF-beta in overtrained Wistar rats. Data are presented as mean ± SD. Different letters indicate significant differences (*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$; ****$p < 0.0001$) by Tukey's post hoc test. P1 (Purple sweet potato administration of 100 mg/kg body weight/day); P2 (Purple sweet potato administration of 200 mg/kg body weight/day); P3 (Purple sweet potato administration of 300 mg/kg body weight/day).

Purple sweet potato improves fatigue time and endurance capacity

The negative control group exhibited a severe decline in fatigue time, dropping to 12.07 ± 1.11 min compared with the normal control (48.89 ± 1.20 min; $p < 0.001$), consistent with overtraining-induced performance impairment. Administration of purple sweet potato significantly increased fatigue time, with values of 32.14 ± 1.76 min (P1), 40.82 ± 0.49 min (P2), and 45.19 ± 1.64 min (P3) (**Figure 5**). The fatigue time in P3 approached that of the normal control, indicating a substantial restoration of physical endurance. This improvement is likely attributable to the combined effects of reduced oxidative stress, attenuated systemic inflammation, and possibly enhanced mitochondrial function, all of which contribute to better skeletal muscle performance and delay in fatigue onset.

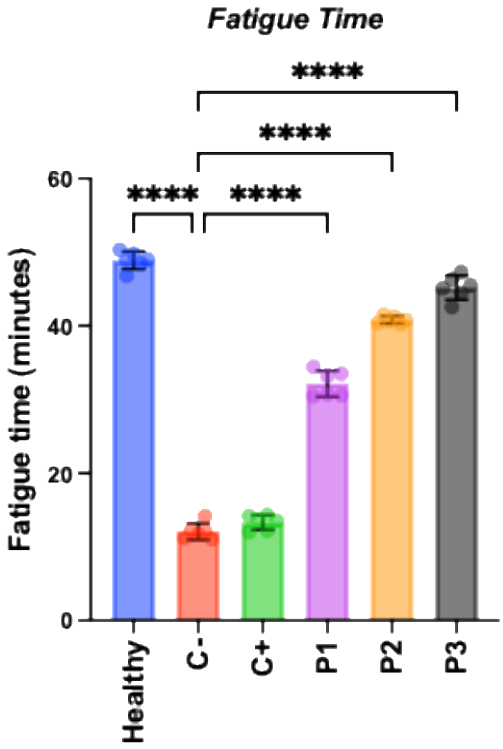


Figure 5 Effects of purple sweet potato on fatigue time in an overtrained Wistar rat model. Treadmill exhaustion time (min) is shown for the normal control, negative control (C-, overtraining), positive control (C+, vitamin C), and treatment groups of purple sweet potato (P1 - P3). Data are presented as mean ± SD. Different letters indicate significant differences (*$p < 0.05$; ** $p < 0.01$; ***$p < 0.001$; ****$p < 0.0001$) by Tukey's post hoc test.

Histopathological analysis of the Liver

Histopathological examination of the liver revealed clear morphological differences among groups. The normal control group showed preserved hepatic architecture, with normal hepatocyte cords, intact sinusoidal spaces, and no obvious inflammatory cell infiltration. In contrast, the negative control group demonstrated marked hepatic injury, characterized by inflammatory cell infiltration, hepatocyte degeneration, focal necrosis, and sinusoidal dilation. These findings indicate substantial liver damage associated with overtraining-induced oxidative stress and inflammation. In group P1, partial histological improvement was observed, although mild inflammatory infiltration persisted. Group P2 showed a more pronounced protective effect, with relatively intact hepatocyte structure and reduced inflammatory changes. The most prominent recovery was observed in group P3, in which liver histology approached that of the normal control group, with near-complete restoration of hepatic architecture (**Figure 6**). Overall, these histopathological findings are consistent with the biochemical data and support the hepatoprotective effect of purple sweet potato, likely mediated through suppression of ROS production, inhibition of NF-κB–dependent inflammatory signaling, and activation of the Nrf2 antioxidant pathway, which enhances endogenous antioxidant enzyme expression.

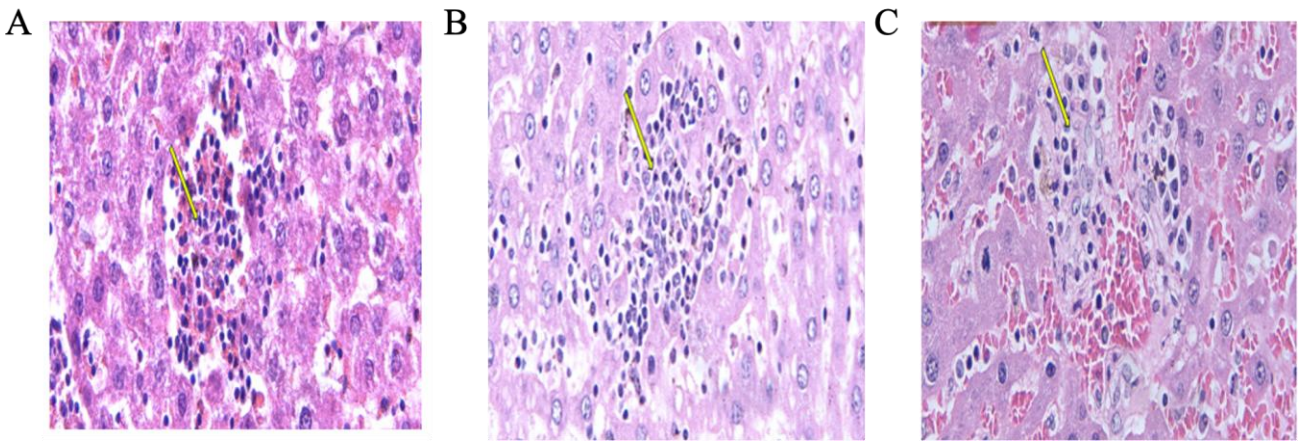


Figure 6 Representative histopathological images of liver tissue in overtrained rats treated with purple sweet potato. (A) P1 groups, (B) P2 groups, and (C) P3 groups. The yellow arrow indicates a macrophage in filtration.

Metabolite profiling of purple sweet potato

GC-MS analysis of dried sweet potato powder revealed a complex chemical profile characterized predominantly by lipid-derived constituents, including fatty acid esters, long-chain alcohols, fatty acid derivatives, and oxygenated hydrocarbons (**Figure 7**). The chromatogram showed multiple peaks distributed across the retention time range, indicating the presence of both early-eluting low-molecular-weight compounds and later-eluting hydrophobic metabolites. The identified compounds were mainly associated with nonpolar or moderately polar chemical classes, suggesting that the GC-MS method preferentially captured volatile and semi-volatile constituents from the dried powder. The most frequently detected compounds belonged to the fatty acid and ester classes, including methyl and ethyl esters of long-chain fatty acids, such as octadecanoic acid, decanoic acid, and dodecanoic acid. Several peaks were also assigned to long-chain alcohols, including ethyl oleyl alcohol-like annotations and related unsaturated alcohols. In addition, phytol-related compounds, cyclic alcohols, and minor terpenoid-like structures were observed, indicating the presence of secondary metabolites that may contribute to the functional properties of sweet potato powder. Overall, the profile suggests that drying preserved a broad spectrum of extractable metabolites, with a strong enrichment in hydrophobic bioactive constituents. The abundance of unsaturated lipid derivatives, especially linoleate- and oleate-related compounds, is notable because these molecules are often associated with nutritional value and potential antioxidant or membrane-stabilizing activity. The detection of phytol-like and other oxygenated terpenoid compounds further supports the presence of plant-derived bioactive compounds in the dried sweet potato matrix. Taken together, the GC-MS profile indicates that dried sweet potato powder contains a chemically diverse mixture of compounds that may contribute to its nutritional and functional characteristics.

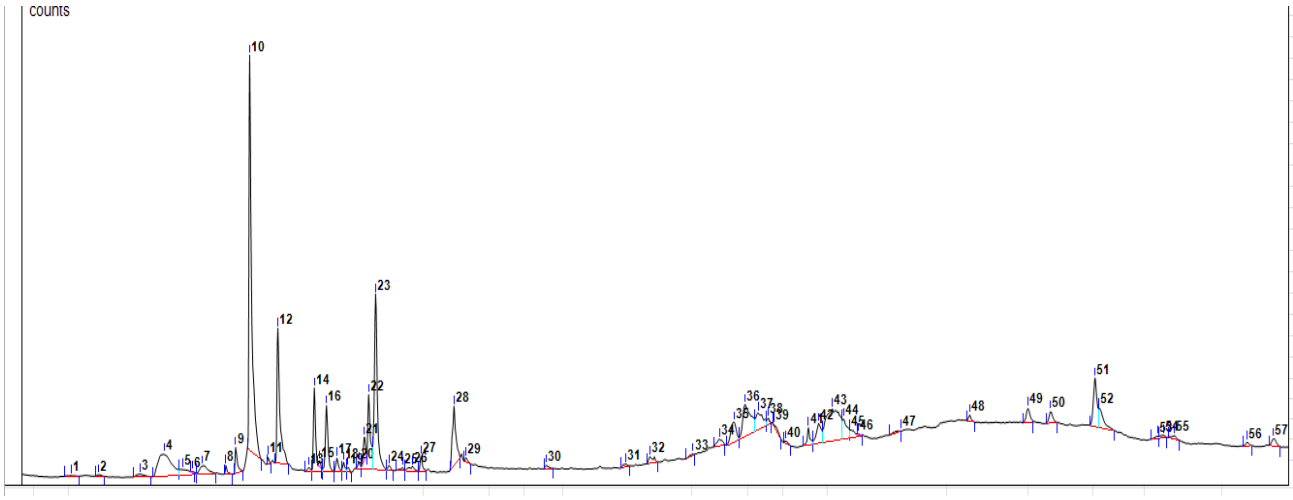


Figure 7 Representative Gas Chromatography-Mass Spectrometry (GC-MS) total ion chromatogram of purple sweet potato. The chromatogram displays the elution profile of volatile and semi-volatile metabolites detected in the sample. Numbered peaks correspond to the compounds identified in **Table S1**, which include various fatty acid derivatives, alcohols, and esterified lipids.

Overtraining induction significantly increased MDA levels in the negative control group compared to normal controls (11.25 ± 0.35 vs 2.02 ± 0.18 nmol/mL; $p < 0.001$), which confirms the occurrence of lipid peroxidation due to increased ROS. Administration of purple sweet potato decreased MDA levels in a dose-responsive manner, with the largest decrease in P3 (300 mg/kgBW) approaching normal control levels (3.02 ± 0.19 nmol/mL; $p = 0.027$). This finding is consistent with the role of anthocyanins as potent antioxidants that scavenge free radicals and reduce lipid peroxidation, leading to lower malondialdehyde (MDA) production. This has been observed across dietary anthocyanin interventions, which show significant reductions in MDA and enhanced antioxidant capacity.

The results of this study confirm that excessive treadmill exercise at 20 m/min for 30 min with a 10° incline for 5 weeks can induce a state of oxidative stress, characterized by increased accumulation of reactive oxygen species (ROS) in skeletal muscle and other tissues. Strenuous or prolonged exercise has been shown to elevate ROS production through enhanced mitochondrial respiration and activation of ROS-generating enzymes such as NADPH oxidase and xanthine oxidase, leading to disruption of redox homeostasis and activation of systemic inflammatory pathways. Elevated ROS and inflammatory signaling stimulate pro-inflammatory cytokine expression and impair mitochondrial function, ultimately accelerating lipid peroxidation and the formation of malondialdehyde (MDA), a biomarker of oxidative stress, in animal models of intense physical training and overtraining [12,16].

The purple sweet potato variety Antin 2 used in this study contains high levels of anthocyanins (252.13 mg/100 g), along with flavonoids, polyphenols, and vitamin C. These bioactive compounds have been widely studied as antioxidants with the ability to reduce reactive oxygen species (ROS) through scavenging mechanisms and enhance endogenous antioxidant capacity. Dietary anthocyanins and other polyphenolic phytochemicals have been shown to exert antioxidant and anti-inflammatory effects by modulating stress and inflammatory biomarkers in animal and human studies, including reductions in oxidative stress markers and pro-inflammatory mediators [17].

Another study has shown that anthocyanins and other polyphenolic compounds derived from pigmented plant sources exhibit significant antioxidant activity, including strong free radical scavenging and inhibition of lipid peroxidation in vivo and in dietary intervention models. Dietary anthocyanins have also been reported to enhance endogenous antioxidant defense mechanisms, such as increasing activities of superoxide dismutase (SOD), glutathione peroxidase (GPx), and other antioxidant enzymes, thereby mitigating oxidative stress

and reducing pro-oxidant biomarkers such as malondialdehyde (MDA) in animal models [18,19].

The results of this study confirm MDA's position as the most reliable biomarker for detecting oxidative damage due to overtraining. This study adds to the evidence that MDA provides an accurate measure of lipid peroxidation and can serve as an indicator of the effectiveness of antioxidant interventions, particularly those derived from natural ingredients such as purple sweet potatoes. Scientific knowledge regarding MDA has advanced rapidly over the last decade and has been further strengthened by recent studies examining the relationships among oxidative stress, inflammation, and the body's antioxidant capacity. Through this study, a more comprehensive understanding was obtained: increased MDA levels are a logical consequence of redox imbalance in *overtraining conditions,* while decreased MDA levels indicate the restoration of this balance. Thus, the results of this study enrich the epistemological construction regarding the role of MDA as a primary biomarker of oxidative stress, while also providing a scientific contribution to the use of purple sweet potato content as an effective intervention to reduce oxidative damage in *overtraining conditions.* These findings strengthen the direction of future research on functional nutrition and natural ingredient-based recovery strategies in the context of exercise physiology.

SOD levels. Overtraining significantly decreased SOD activity in negative controls (19.85 ± 2.75 U/mL) compared to controls (81.61 ± 4.23 U/mL; $p < 0.001$). Purple sweet potato intervention significantly and gradually increased SOD in a dose-dependent manner, with the highest value at P3 (71.32 ± 2.75 U/mL; $p < 0.001$). This increase indicates activation of the endogenous antioxidant system, possibly via the Nrf2 pathway, which induces the expression of antioxidant enzymes. Dietary polyphenols and plant-derived compounds have been shown to activate the Nrf2 transcription factor and enhance the expression of antioxidant enzymes, thus improving antioxidant capacity and increasing the activities of antioxidant enzymes such as SOD in experimental models of oxidative stress [20,21].

Superoxide dismutase (SOD) is the primary antioxidant enzyme that converts superoxide radicals into hydrogen peroxide and oxygen. Under overtraining conditions, increased production of reactive oxygen species (ROS) is an inevitable consequence of elevated aerobic metabolism, increased electron transport chain activity, and greater radical generation. In this situation, SOD works harder to neutralize superoxide. Consequently, if oxidative stress persists and overwhelms the endogenous defense mechanisms, functional enzyme levels may decrease or fail to compensate. Recent literature suggests that while acute bouts of high-intensity exercise initially elevate antioxidant enzyme activity as a compensatory response, prolonged or excessive high-intensity training can lead to sustained oxidative stress that may blunt or reduce antioxidant enzyme responses, including SOD, in certain contexts of overreaching or inadequate recovery [16,22].

Previous studies have demonstrated that exercise-induced increases in reactive oxygen species (ROS) stimulate adaptive antioxidant responses via the Nrf2 signaling pathway, which enhances the expression and activity of endogenous antioxidant enzymes, including superoxide dismutase isoforms (SOD1, SOD2). However, when ROS production exceeds the capacity of antioxidant defenses during prolonged or excessive training, redox imbalance and functional suppression of antioxidant enzyme activity can occur, contributing to oxidative fatigue and mitochondrial dysfunction [23]. Biological fatigue, or biological exhaustion caused by excessive exercise, is indicated by the continuous accumulation of ROS, which impairs SOD function. The pre-test results in this study, which showed relatively homogeneous SOD levels across all groups (84.55 - 85.78 U/mL), indicate that the baseline conditions of all groups were similar before treatment began, allowing differences after the intervention to be interpreted more validly. In group P1, SOD levels decreased from 84.55 ± 2.75 to 44.12 ± 3.83 with a difference of 40.42 ± 2.02. Meanwhile, in group P2, SOD levels decreased less than in group P1, namely from 85.78 ± 3.17 to 61.02 ± 2.75, with a difference of 24.75 ± 3.40. In group P3, there was another decrease in SOD levels, namely from 84.55 ± 2.75 to 71.32 ± 2.75 with a difference of 13.23 ± 3.47. It can be concluded that a dose of 300 mg *of Ipomoea batatas* is the most effective compared to the other groups. This can happen because *Ipomoea batatas* is rich in anthocyanins, which

have strong antioxidant activity. Ability This helps neutralize radical free and reduce oxidative stress.

The theory of SOD as a biomarker of oxidative stress stems from extensive biochemical and exercise physiology research. The concept of oxidative stress is crucial for understanding fatigue, maladaptation, and tissue damage resulting from excessive physical activity; it reflects an imbalance between the production of reactive oxygen species (ROS) and the capacity of antioxidant defenses to neutralize them. Because superoxide dismutase is a primary antioxidant enzyme that directly converts superoxide radicals into less harmful molecules, changes in its activity are widely used to assess oxidative stress status and antioxidant defense capacity. Several modern studies indicate that although moderate training can enhance antioxidant enzyme activities as part of physiological adaptation, excessive or prolonged high-intensity exercise may overwhelm the antioxidant system, leading to attenuated SOD responses or shifts in redox balance, reflecting maladaptive responses to oxidative stress [24,25]. Therefore, when the negative control group in this study showed decreased SOD after overtraining, the findings align with established literature on oxidative stress and its impact on antioxidant defenses.

Compared with the negative control group that did not receive antioxidant intervention, the purple sweet potato treatment group showed increased SOD activity at 200 and 300 mg/kgBW. Post-test data from this study also showed that the purple sweet potato treatment group had increased SOD activity. This increase indicates that the polyphenols and anthocyanins in purple sweet potatoes can modulate the redox response and enhance SOD enzymatic activity following oxidative stress induced by excessive exercise. Based on the theory that exogenous antioxidants can affect the expression of endogenous antioxidant genes through a transcriptional mechanism involving nuclear factors. erythroid 2–related factor 2 (Nrf2).

One important point in understanding the results of this study is the anthocyanin content in purple sweet potatoes, which reaches 252.13 mg/100 g. As a bioactive compound, anthocyanins have the ability to capture free radicals, inhibit NADPH oxidase activity, and prevent ROS from forming in mitochondria. Because the radical load that must be handled is significantly reduced, this mechanism directly helps increase SOD activity. New studies show that regular consumption of anthocyanins can improve SOD function through the hormesis mechanism (mild stimulation that triggers stronger antioxidant adaptation). Therefore, the dose of 200 - 300 mg/kgBW that showed the best response in this study may be the balance point between antioxidant supply and triggering biological adaptations needed to increase SOD.

The positive control group containing vitamin C also showed an increase in SOD activity, but this increase was smaller than in the purple sweet potato treatment group. Vitamin C primarily acts as a direct free radical scavenger and can moderately support endogenous antioxidant systems, including SOD, through pathways such as Nrf2, although its effect is generally weaker than that of anthocyanins and other polyphenols [26]. From an epistemological perspective, it is reasonable that the increase in SOD in the positive control group was not as large as in the purple sweet potato treatment group in this study, because vitamin C has a more limited mechanism of action to increase SOD activity compared to polyphenolic antioxidants.

From an epistemological perspective, this study advances scientific understanding of how SOD functions as a key indicator of the body's resilience to oxidative stress. The significantly different SOD activity levels across groups suggest that this biomarker is highly sensitive for identifying physiological changes induced by exercise and nutritional interventions. Furthermore, these findings support the notion that increased SOD indicates restoration of redox homeostasis, while decreased SOD indicates long-term oxidative stress that exceeds the body's defenses. Overall, purple sweet potato anthocyanins have been shown to aid the recovery of the endogenous antioxidant system, making them potential candidates for mitigating oxidative stress induced by excessive exercise.

TNF-α levels. TNF-α levels increased sharply in the negative control (21.04 ± 0.16 pg/mL) compared to the control (5.35 ± 0.41 pg/mL; $p < 0.001$), indicating systemic inflammatory activation. Purple sweet potato administration significantly reduced TNF-α at all doses, with the most optimal effect at P3 (6.85 ± 0.29 pg/mL; $p < 0.001$). This decrease indicates that anthocyanins inhibit the NF-κB pathway, which is known to suppress cytokine transcription. proinflammatory [27]. After *overtraining,* over the course of five weeks, the negative

control group showed a sharp spike in TNF-α, increasing to 21.04 pg /dL (5.18 ± 0.29: 20.19 ± 0.25 pg/dL). This increase in levels provides strong empirical evidence that the *overtraining model* successfully induces severe oxidative stress and systemic inflammation. These findings are consistent with study of Meeusen *et al.* [1] showed that prolonged, intense physical exercise increases ROS, which then activate the NF-κB pathway and trigger massive TNF-α secretion. Another study demonstrated that overtraining triggers increases in pro-inflammatory cytokines, including tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), and interleukin-1β, in an established treadmill overtraining mouse model. The exhaustive exercise protocol led to systemic inflammation, impaired endurance, and maladaptive physiological responses, confirming that chronic high-intensity exercise can disrupt homeostasis. Thus, the empirical data from this study support the inflammatory theory of strenuous exercise and reinforce the use of TNF-α as a sensitive biomarker for assessing homeostatic disturbances induced by chronic physical stress [28]. In the treatment group given purple sweet potato variety Antin 2, the dynamics of TNF-α changes showed a clearer pattern when the phytochemical content of this local plant was placed in the context of its molecular mechanisms. The purple sweet potatoes used in the study had a very high anthocyanin content of 252.13 mg/100 g, as well as significant levels of phenols, flavonoids, and vitamin C. These contents provide the biological basis for the TNF-α-lowering effect. The P1 treatment group at 100 mg/kgBW showed an increase in TNF-α to 14.30 pg/dL, a value that was still much lower than that of the negative control group. This phenomenon indicates that even at the lowest dose, anthocyanins and other phytochemicals contained in purple sweet potatoes have a protective effect against inflammation. The biological pathway underlying this phenomenon can be explained by the antioxidant properties of anthocyanins, which break the chain of free radical reactions and inhibit NF-κB activation.

Another study has shown that anthocyanins and related polyphenolic compounds can attenuate pro-inflammatory responses by modulating the NF-κB signaling pathway, including suppression of NF-κB nuclear translocation and downstream cytokine expression such as TNF-α. This modulation occurs partly through stabilization of IκB and inhibition of the phosphorylation events that lead to NF-κB activation, resulting in reduced pro-inflammatory gene expression in cellular and animal models of inflammation. Thus, the observed changes in the P1 group can be interpreted as an empirical manifestation of the molecular mechanisms described in the literature regarding anthocyanin-mediated inhibition of NF-κB signaling and pro-inflammatory cytokine expression [29,30].

In group P2, which received a dose of 200 mg/kgBW, the TNF-α-lowering effect appeared even stronger, with post- overtraining TNF-α levels reaching only 9.24 pg/dL. This more significant decrease indicates a dose-response relationship between anthocyanins and the modulation of inflammation. Another study reported that anthocyanins can attenuate NLRP3 inflammasome activation and reduce associated inflammatory markers by suppressing ROS-mediated inflammasome signaling. In a clinical intervention, anthocyanin supplementation markedly decreased expression of NLRP3 inflammasome components and pro-inflammatory cytokines, highlighting the ability of anthocyanins to modulate inflammatory pathways linked to oxidative stress [31]. In other words, the pattern that emerged in the P2 group was not only a numerical decrease, but also a representation of a gradual intensification of the biological mechanisms at work.

In group P3, the group with the highest dose (300 mg/kgBW), TNF-α levels after overtraining reached only 6.85 pg/dL, a value very close to the pre-treatment normal. The highest anthocyanin dose appears to provide almost complete protection against inflammation. Another body of research indicates that, beyond their roles as antioxidants and anti-inflammatory agents, anthocyanins can also influence gene expression by modulating epigenetic mechanisms, such as alterations in DNA methylation, histone modifications, and non-coding RNA pathways, which, in turn, can affect the transcription of pro-inflammatory genes and signaling pathways. Thus, the molecular changes observed in the P3 group align with evidence that purple sweet potato anthocyanins act at multiple biological levels to mitigate oxidative stress and inflammation [32].

The consistent pattern of TNF-α reduction from P1 to P3 supports the view that anthocyanins from purple

sweet potatoes have a strong causal role in inhibiting inflammation. This data pattern does not stand alone; it is integrated with changes in other biomarkers, such as decreased MDA and increased SOD, in the same group. A decrease in MDA indicates reduced oxidative stress, while an increase in SOD indicates a stronger antioxidant defense system. In the epistemology of coherence, such a pattern is considered a strong indicator of scientific truth because all data support the same conclusion, and there is no contradiction between variables. Research by Khoo *et al.* [11] also showed that anthocyanins can increase endogenous antioxidant capacity, thereby reducing TNF-α levels, a natural effect of improved redox stability.

Including phenol, flavonoid, and vitamin C content in data interpretation adds to the epistemic richness of this discussion. Phenols and flavonoids are known to possess potent antioxidant activity, while vitamin C acts as an essential cofactor in antioxidant regeneration. The presence of these compounds synergistically enhances the effects of anthocyanins. The inclusion of phenol, flavonoid, and vitamin C content in data interpretation adds to the epistemic richness of this discussion. Phenolic compounds and flavonoids are known for their potent antioxidant and anti-inflammatory activities, while vitamin C enhances antioxidant regeneration and redox balance. The presence of these multiple bioactive components can act synergistically, resulting in greater overall antioxidative and immunomodulatory effects compared to individual compounds alone. Evidence from recent systematic reviews indicates that dietary flavonoids and vitamin C interact to enhance antioxidant defense and control pro-inflammatory signaling, supporting the notion that the reduction in TNF-α observed in this study reflects the combined actions of these phytochemicals rather than the effect of a single molecule [33].

The results of this study demonstrate that anthocyanins are not simply antioxidant compounds, but biological agents with multifunctional capabilities that can modulate inflammatory pathways, ameliorate oxidative stress, and restore cellular homeostasis. However, critical epistemology also demands that knowledge not be accepted as absolute. These results raise new questions about the limits of effective dosage, the mechanisms of phytochemical interactions, and the potential translation of these findings to humans. The results of this study provide a significant epistemic contribution to the development of functional nutrition and sports immunology. The new knowledge generated is that local Indonesian purple sweet potato varieties, with their high anthocyanin content, have great potential as agents for modulating inflammation induced by strenuous exercise. However, this knowledge should also be viewed as part of a scientific journey that still needs to be expanded and further tested.

The results of the study showed that *overtraining* triggered a sharp increase in several inflammatory and fibrotic mediators in the untreated group, whereas the group receiving purple sweet potatoes (high in anthocyanins) showed a restraint in the increase in TGF-β. The results of the study obtained measurements of TGF beta levels before the control group 4.29 ± 0.39 pg/dL, the negative control group 4.04 ± 0.39 pg/dL, the positive control 4.31 ± 0.44 pg/dL, treatment group 1 (100 mg) 4.25 ± 0.52 pg/dL, treatment group 2 (200 mg) 4.27 ± 0.62 pg/dL and treatment group 3 (300 mg) 4.20 ± 0.59 pg/dL. Measurement of TGF beta levels after the control group 4.69 ± 0.39 pg/dL, negative control group 36.29 ± 0.65 pg/dL, positive control 35.01 ± 0.29 pg/dL, treatment group 1 (100 mg) 13.29 ± 0.46 pg/dL, treatment group 2 (200 mg) 9.98 ± 0.31 pg/dL and treatment group 3 (300 mg) 8.07 ± 0.38 pg/dL.

TGF-β is a pleiotropic cytokine that plays a central role in regulating inflammation, tissue repair, and fibrotic processes. Upon tissue stress or injury, such as that induced by intense or repeated exercise, TGF-β signaling can become activated in response to cellular damage and increased reactive oxygen species (ROS). Active TGF-β engages its canonical signaling cascade, in which ligand binding to its receptors leads to phosphorylation of receptor-regulated Smad proteins (Smad2/3), their association with Smad4, and subsequent translocation into the nucleus to regulate transcription of genes involved in extracellular matrix production and fibrosis. While TGF-β signaling contributes to normal wound healing and tissue remodeling, excessive and prolonged activation has been associated with pathological fibrosis and maladaptive responses in skeletal and cardiac muscle tissues [34,35].

TGF-β values before treatment showed a very stable range. Pre -treatment values were at 4.29 ± 0.39 pg/dL in the control group, 4.04 ± 0.39 pg/dL in the

negative control group, 4.31 ± 0.44 pg/dL in the positive control group, 4.25 ± 0.52 pg/dL at a dose of 100 mg, 4.27 ± 0.62 pg/dL at a dose of 200 mg, and 4.20 ± 0.59 pg/dL at a dose of 300 mg. This uniformity indicates that the initial physiological conditions between groups were at the same level so that subsequent changes can be interpreted as the effect of exposure to intense exercise and differences in purple sweet potato administration.

After intense exercise, the no-intervention group showed a very striking spike in TGF-β. The negative control group increased from 4.04 to 36.29 pg/dL with a difference of −32.25 ± 0.63 pg/dL, while the positive control group increased from 4.31 to 35.01 pg/dL with a difference of −30.70 ± 0.67 pg/dL. This more than eightfold increase indicates a strong activation of the TGF-β pathway. TGF-β is a multifunctional cytokine that is released in response to tissue damage and inflammatory stimuli and plays a central role in regulating the balance between normal wound healing and fibrotic responses. It can be secreted by multiple cell types, including macrophages and fibroblasts, and upon activation, engages the canonical Smad2/3 signaling cascade to regulate gene expression related to extracellular matrix production and fibrosis. Sustained and excessive activation of the TGF-β/Smad pathway under prolonged injury or stress has been linked to pathological fibrotic changes in various tissues, suggesting that persistently elevated TGF-β after intense exercise could contribute to maladaptive tissue remodeling if not properly resolved. These mechanisms have been described in recent literature on TGF-β signaling and tissue fibrosis [34,36].

Changes in the treatment group showed a much lower increase pattern and showed a clear relationship between the anthocyanin dose and the degree of inhibition of TGF-β increase. At a dose of 100 mg, the initial value of 4.25 pg/dL increased to 13.29 pg/dL, resulting in a difference of −9.04 ± 0.87 pg/dL. Although the increase still occurred, the amount was much lower than the negative control. At a dose of 200 mg, the initial value of 4.27 increased to 9.98 pg/dL, resulting in a difference of −5.71 ± 0.78 pg/dL. At a dose of 300 mg, the initial value of 4.20 increased to 8.07 pg/dL, a difference of −3.86 ± 0.58 pg/dL, the lowest increase among all groups.

Anthocyanins exhibit a dose-dependent biological response, where higher concentrations produce stronger effects. The main anthocyanins in purple sweet potatoes, cyanidin, peonidin, and pelargonidin, are potent antioxidants that neutralize reactive oxygen species (ROS) through hydrogen donation and electron transfer. By reducing ROS, anthocyanins indirectly suppress activation of pro-inflammatory pathways such as NF-κB, which interact with the TGF-β signaling cascade. This explains why higher doses of purple sweet potato anthocyanins more effectively decrease TGF-β activation and inflammatory responses in overtrained models [37].

Anthocyanins suppress pro-inflammatory cytokines, including IL-6 and TNF-α, which act as secondary stimulants for TGF-β activation. By reducing these cytokines through antioxidant and NF-κB–modulating effects, anthocyanins indirectly attenuate TGF-β signaling. This mechanism explains the observed dose-dependent pattern, in which higher anthocyanin doses result in smaller increases in TGF-β [38]. The effects of purple sweet potato bioactive compounds are not only related to antioxidant capacity but also the ability to modulate the expression of fibrosis -related genes. Recent literature indicates that anthocyanins can act as epigenetic modulators, influencing gene expression by altering DNA methylation, histone modifications, and non-coding RNA profiles. These epigenetic effects have been observed in studies showing that anthocyanins alter histone acetylation and miRNA expression, which, in turn, can modulate inflammatory and signaling pathways, including those involved in cytokine expression. Thus, the observed suppression of TGF-β increases at higher anthocyanin doses in the P3 group aligns with evidence that anthocyanins can regulate gene expression through multiple epigenetic mechanisms [32].

Overall, the TGF-β elevation figures show a significant difference between groups. The increase from approximately four to over 35 pg /dL in the untreated group indicates extreme inflammatory and fibrotic activation. In contrast, the increase at the 300 mg dose was only from 4.20 to 8.07 pg /dL, indicating that the regulation of tissue repair pathways is more controlled and adaptive. Epistemologically, this numerical pattern illustrates the internal coherence between the basic concepts of TGF-β molecular biology, oxidative stress theory, and the anti-inflammatory effects of anthocyanins. The knowledge built from the

relationship between numerical data and biological theory indicates that the inhibition of TGF-β elevation by purple sweet potato is not a coincidental phenomenon, but rather a process with a strong mechanistic basis. The consistency of the dose- response pattern strengthens the validity of the claim that purple sweet potato constituents play a significant role in preventing excessive activation of fibrogenic pathways.

IL-6 levels. In line with TNF-α, IL-6 levels increased significantly in the negative control (115.66 ± 3.93 pg/mL) compared to the control (56.33 ± 5.60 pg/mL; $p < 0.001$). Purple sweet potato administration decreased IL-6 in a dose-responsive manner, with P3 showing values close to the control (61.66 ± 4.32 pg/mL; $p = 0.025$). This decrease in IL-6 is relevant to the role of anthocyanins in modulating oxidative stress-induced inflammation in liver and muscle tissues [39]. Anthocyanins in purple sweet potatoes (cyanidin, peonidin, pelargonidin) reduce oxidative stress and inhibit NF-κB activation, lowering IL-6 production while enhancing endogenous antioxidants, such as SOD, thereby protecting muscle cells from exercise-induced inflammation [38].

The role of anthocyanins in controlling IL-6 is even stronger at higher doses. At a dose of 300 mg, the increase in IL-6 was only about 10 picograms per deciliter, significantly lower than in the negative control group, which increased by more than 66 picograms per deciliter. Anthocyanins in purple sweet potatoes can reduce inflammatory responses by lowering oxidative stress and inhibiting pro-inflammatory pathways, such as NF-κB. This leads to decreased expression of pro-inflammatory cytokines, such as IL-6 and TNF-α, explaining why higher anthocyanin doses were associated with a smaller increase in IL-6 [40].

IL-6 acts as an early marker of sensitivity to excessive exercise stress. A sharp increase in IL-6 in the control group reflects a non-adaptive inflammatory response, while anthocyanins suppress this increase, shifting the response toward a safer, more adaptive pattern. The consistent dose-response effects and significant p-values confirm that anthocyanins reliably modulate IL-6. Integrating numerical data, inflammation theory, and antioxidant biochemistry provides a framework for understanding and predicting muscle responses to stress and phytochemical interventions, making IL-6 a key indicator of how purple sweet potato compounds guide the body's adaptive response to exercise.

Overtraining reduced exhaustion time in the control group, while purple sweet potato supplementation dose-dependently increased endurance, with the highest dose nearly restoring normal performance. These effects are linked to reduced oxidative stress and inflammation, improved antioxidant capacity, and more efficient mitochondrial energy metabolism, which delays fatigue by counteracting ROS-induced mitochondrial impairment [41,42].

Repeated intense exercise increases metabolic demand in skeletal muscle, leading to excessive ROS that overwhelm antioxidant defenses. High ROS levels cause oxidative damage to lipids, proteins, and DNA, disrupt calcium handling and mitochondrial function, and lead to muscle fatigue. Lipid peroxidation, indicated by elevated MDA, damages membranes and accelerates exhaustion, explaining the very short performance times in overtrained control groups [43], [44]. In overtrained control groups, MDA increased while SOD activity decreased, indicating that antioxidant defenses were overwhelmed. Reduced SOD allowed ROS to accumulate, causing oxidative damage, impairing muscle contraction and energy metabolism, and accelerating fatigue, which explains the very short exhaustion times observed [45].

Inflammatory cytokines, particularly IL-6, contribute to fatigue during intense exercise. In overtrained controls, IL-6 rose sharply, triggering glycogen breakdown, shifting metabolism toward anaerobic pathways, and accelerating energy depletion, which leads to faster fatigue. IL-6 thus serves as a sensitive marker of metabolic stress and the onset of exercise-induced fatigue [46-48]. TNF-α worsens fatigue by impairing muscle contractile function, disrupting extracellular matrix integrity, altering sarcomere structure, and disrupting calcium-dependent excitation-contraction coupling, thereby reducing force generation and accelerating fatigue during systemic inflammation and exercise stress [49]. TGF-β also showed a significant increase in the negative control group, from approximately 4 to over 36 pg/dL. Excessive TGF-β signaling has been strongly associated with fibrotic changes in skeletal muscle, characterized by increased extracellular matrix deposition, myofibroblast activation, and reduced tissue elasticity.

Elevated TGF-β activates the canonical Smad pathway, particularly Smad2 and Smad3, which translocate to the nucleus to stimulate profibrotic gene expression and collagen synthesis, contributing to muscle stiffness and impaired contractile function. Such structural alterations can reduce muscle mechanical efficiency and accelerate fatigue, supporting the hypothesis that repeated intense exercise induces significant tissue damage and fibrotic signaling in this model [50,51].

In contrast to the negative control group, the treatment group given purple sweet potato showed a gradual increase in fatigue time as the dose increased. At a dose of 100 mg, fatigue time increased to 32.14 min, then to 40.82 min at 200 mg, and reached 45.19 min at 300 mg, approaching the normal control group. This pattern suggests that anthocyanins in purple sweet potato improve physical endurance by reducing exercise-induced oxidative stress and inflammation, enhancing mitochondrial energy metabolism, and increasing endogenous antioxidant defenses. Mechanistically, anthocyanins are known to modulate redox balance and inflammatory signaling by activating the Nrf2 pathway, which induces antioxidant enzymes (SOD), and by inhibiting the NF-κB pathway, leading to reduced expression of pro-inflammatory cytokines such as IL-6 and TNF-α, processes that together help protect muscle cells from fatigue-related damage and improve performance outcomes [52,53].

The decrease in IL-6, TNF-alpha, and TGF-beta levels in the treatment group is evidence that anthocyanins are effective in reducing systemic physiological stress. By reducing inflammation and increasing antioxidant capacity, the internal muscle environment becomes more stable. This stability allows muscles to maintain contractile function longer and directly increases fatigue time. This supports the finding that increased fatigue resistance is a logical consequence of improved metabolic, redox, and structural homeostasis. Epistemologically, fatigue time in this study can be interpreted as a comprehensive indicator that reflects not only physical performance but also the body's internal biological condition. By treating fatigue as a multifactorial phenomenon and placing inflammatory and redox biomarkers as the main elements, fatigue time becomes a multifactorial indicator that reflects the organism's physiological balance. When nutritional interventions, such as anthocyanins, improve this homeostasis, the increase in fatigue time is a systemic manifestation of these improvements. Thus, the physiological mechanisms underlying the increase in physical endurance in the treatment group are not only local to the muscles but are comprehensive and integrated within the body's metabolic and inflammatory systems. In addition, a limitation of this study was that we used male rats. Sex is a well-established variable that influences oxidative stress responses, inflammatory signaling, and exercise tolerance through hormonal regulation, including estrogen's antioxidant effects and metabolic pathways. Excluding females limits the generalizability of our findings to both sexes and may obscure sex-specific responses to purple sweet potato supplementation. This decision was made to reduce biological variability and confounding from hormonal fluctuations, but future studies should include female animals to evaluate sex-dependent effects.

Overall, these results indicate that purple sweet potato has a protective effect against oxidative stress and inflammation induced by overtraining, as evidenced by decreased MDA, TNF-α, and IL-6 and increased SOD, with direct implications for improving physical endurance. Liver histopathology findings showing improved hepatocyte structure at high doses strengthen the mechanistic evidence that modulation of the ROS–Nrf2–NF-κB pathway plays a key role in purple sweet potato's hepatoprotective effects.

Conclusions

Administration of purple sweet potatoes (*Ipomoea batatas* L.) has been shown to reduce oxidative stress and inflammatory biomarkers in Wistar rats subjected to an overtraining model. The protective effects were demonstrated by decreased levels of MDA, TNF-α, IL-6, and TGF-β, alongside increased SOD activity. These findings suggest that purple sweet potatoes have potential as a natural intervention to mitigate the adverse effects of excessive physical exercise.

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Declaration of generative AI in scientific writing

The authors used generative artificial intelligence (AI) tools to improve the clarity and language of this manuscript. All content was carefully reviewed and verified by the authors, who take full responsibility for the final version of the manuscript.

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Supplementary Materials

Table S1 GC-MS-identified compounds in purple sweet potato powder, with retention time, tentative compound assignment, chemical class, and relative peak area.

No.	Retention time (min)	Compound name	Chemical class	Relative area (%)
1	5.29	Ethyl iso-olchidole	Alcohol/derivative	0.00
2	5.99	[1-(3-Ethoxypropyl)-2-octonoic acid, 2-hexyl-, methyl ester]	Fatty acid ester	0.13
3	7.10	4-Methyl-4-butyldecenoic acid	Fatty acid derivative	0.47
4	7.28	Maltose	Carbohydrate	5.47
5	7.32	2-Methylpropyl nonadecanoate	Fatty acid ester	1.12
6	8.45	2-Methylpropyl nonadecanoate	Fatty acid ester	0.03
7	8.57	Cyclopropanedecanoic acid, 2-octyl-, methyl ester	Fatty acid ester	0.05
8	9.03	α-Ocimene	Terpenoid hydrocarbon	0.30
9	9.30	2-Nonenethioic acid	Sulfur-containing compound	0.05
10	9.94	2-Pentanone, 4-methyl-2-[3,4,4,5-tetramethyl-…]	Ketone derivative	18.90
11	10.46	Ethyl iso-olchidole	Alcohol/derivative	0.21
12	10.67	2-Propanone, 1-(1,1-dimethyl-2,3,4,5-tetrahydro-1H-1-benzocyclo…)	Ketone derivative	0.15
13	11.53	1-Heptadecanol	Long-chain alcohol	0.26
14	11.89	1,6,6-Trimethyl-bicyclo[3.1.1]hept-2-ene	Terpenoid hydrocarbon	1.90
15	11.82	1-Heptadecanol	Long-chain alcohol	0.18
16	11.82	2-Isopropyl-5-dimethylcyclohex-2-enone	Cyclic ketone	0.30
17	12.14	1-Heptadecanol	Long-chain alcohol	0.42
18	12.44	5-Methyl-7-tetradecen-1-ol acetate	Ester/alcohol derivative	0.23
19	12.55	Ethyl iso-olchidole	Alcohol/derivative	0.10
20	12.69	1H-Benzocycloheptene, 2,3,4,5,6,7-hexahydro-1,1,4,4-tetramethyl-, cis-	Hydrocarbon	0.92
21	13.04	1H-Eicosanedioic acid	Fatty acid	0.10
22	13.69	1-Nonylpropyl-5,5-dimethylcyclopentane-2-enone	Cyclic ketone	4.13
23	13.94	1-Heptadecanol	Long-chain alcohol	0.50
24	14.49	1-Heptadecanol	Long-chain alcohol	0.25
25	14.99	1-Heptadecanol	Long-chain alcohol	0.25
26	15.14	3-Methyl-1,2-benzodioxole	Aromatic heterocycle	0.25
27	15.35	3-Tetradecen-1-ol	Unsaturated alcohol	0.22
28	15.37	trans-13-Octadecenoic acid	Unsaturated fatty acid	3.90

No.	Retention time (min)	Compound name	Chemical class	Relative area (%)
29	15.87	Ethyl iso-olchidole	Alcohol/derivative	0.22
30	17.79	Ethyl iso-olchidole	Alcohol/derivative	0.23
31	20.05	Hexadecanoic acid, 1-(hydroxymethyl)-1,2-ethanediyl ester	Fatty acid ester	0.60
32	20.64	Dodecanoic acid, 1,2-propanediyl ester	Fatty acid ester	0.06
33	21.20	Dodecanoic acid, 1,2-propanediyl ester	Fatty acid ester	0.04
34	21.72	Dodecanoic acid, 1,2-propanediyl ester	Fatty acid ester	0.06
35	23.06	Dodecanoic acid, 1,2-propanediyl ester	Fatty acid ester	0.04
36	23.86	Dodecanoic acid, 1,2-propanediyl ester	Fatty acid ester	0.04
37	24.67	Ethyl iso-olchidole	Alcohol/derivative	0.00
38	24.87	Ethyl iso-olchidole	Alcohol/derivative	0.00
39	25.00	Hexadecanoic acid, 1-(hydroxymethyl)-1,2-ethanediyl ester	Fatty acid ester	0.00
40	25.43	Dodecanoic acid, 1,2-propanediyl ester	Fatty acid ester	0.87
41	25.59	Ethyl iso-olchidole	Alcohol/derivative	0.31
42	27.01	Ethyl iso-olchidole	Alcohol/derivative	0.24
43	27.11	Ethyl iso-olchidole	Alcohol/derivative	0.21
44	27.91	Ethyl iso-olchidole	Alcohol/derivative	0.99
45	28.19	Ethyl iso-olchidole	Alcohol/derivative	0.31
46	29.01	Ethyl iso-olchidole	Alcohol/derivative	0.31
47	31.04	Ethyl iso-olchidole	Alcohol/derivative	0.20
48	32.21	2-Stenol	Sterol-related compound	3.24
49	32.32	Ethyl iso-olchidole	Alcohol/derivative	1.68
50	32.87	Ethyl iso-olchidole	Alcohol/derivative	0.00
51	34.01	Hexadecanoic acid, 1-(hydroxymethyl)-1,2-ethanediyl ester	Fatty acid ester	0.23
52	34.80	Ethyl iso-olchidole	Alcohol/derivative	0.22
53	36.23	Ethyl iso-olchidole	Alcohol/derivative	0.31
54	36.23	Ethyl iso-olchidole	Alcohol/derivative	0.23