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Effect of Purple Sweet Potato (Ipomoea batatas L.) Leaf Extract on AST and ALT Enzyme in CCl4-Induced Rats

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Abstract

This study investigates the effect of sweet potato leaf extract on AST and ALT enzyme levels in rats with liver damage induced by CCl4 (carbon tetrachloride). Liver damage, often caused by toxins like CCl4, is a significant health concern, and AST and ALT are key biomarkers used to assess liver function. Sweet potato leaves, known for their antioxidant and anti-inflammatory properties, may offer therapeutic benefits for liver protection. This study aims to evaluate the potential of sweet potato leaf extract in mitigating liver damage by examining its effect on liver enzymes in CCl4-induced rats. Twenty-four male Wistar rats were divided into six groups. The normal control group received no treatment, the positive control group was treated with silymarin (50 mg/kg body weight orally per day), and the negative control group received CCl4 (1 mL/kg body weight intraperitoneally twice a week) to induce liver damage. The experimental groups received sweet potato leaf extract at doses of 250 mg/kg, 500 mg/kg, and 750 mg/kg body weight orally. All treatments were administered daily for three weeks. The CCl4-induced group showed significantly higher AST and ALT levels compared to the normal control group. However, rats treated with sweet potato leaf extract demonstrated a significant reduction in both enzyme levels, with the highest dose showing the most improvement in liver function. Statistical analysis confirmed that the extract had a dose-dependent effect. Sweet potato leaf extract has a hepatoprotective effect, potentially reducing AST and ALT levels in rats with CCl4-induced liver damage. This study supports its potential as a natural remedy for liver protection, warranting further research for clinical applications.

Keywords: Sweet Potato Leaf Extract, AST, ALT, Liver Damage, CCl4, Hepatoprotective

Introduction

The liver is a vital organ that plays a central role in maintaining systemic homeostasis. It is involved in diverse metabolic functions, including the synthesis of plasma proteins, glycogen storage, detoxification of harmful substances, and bile production for fat digestion.[1] Furthermore, the liver regulates hormonal balance and drug metabolism. Liver dysfunction, therefore, can lead to systemic complications and is associated with significant morbidity and mortality.[2, 3]

One of the major causes of liver injury is exposure to hepatotoxic chemicals such as carbon tetrachloride (CCl_4). This compound is widely used in animal models to mimic chemically induced liver damage due to its ability to generate free radicals that destroy hepatocytes.[4] CCl_4 is metabolized in the liver by cytochrome P450 enzymes into trichloromethyl radicals, which then react with oxygen to form peroxyl radicals. These radicals initiate lipid peroxidation in hepatocyte membranes, leading to cellular necrosis and inflammation.[5-7]

The use of CCl_4-induced hepatotoxicity models is well-established in experimental research due to their ability to simulate both acute and chronic liver injury. The toxic effects of CCl_4 are typically marked by a significant increase in serum levels of liver enzymes such as aspartate aminotransferase (AST) and alanine aminotransferase (ALT), which serve as critical indicators of hepatocellular damage.[8, 9] This model provides a robust framework for evaluating the hepatoprotective potential of therapeutic agents, particularly plant-based compounds.

AST and ALT are the most frequently used biomarkers for assessing liver injury. These enzymes are released into the bloodstream when hepatocyte membranes are compromised. ALT is more liver-specific, whereas AST is also present in cardiac and skeletal muscle tissues.[10,11] Elevated serum levels of these enzymes often reflect necrosis or oxidative stress within the liver. Therefore, reductions in AST and ALT levels can serve as important markers of hepatoprotection.

In light of the side effects and hepatotoxicity associated with conventional therapies, researchers are increasingly turning to plant-based natural products as safer alternatives. One such plant with promising hepatoprotective properties is the purple sweet potato (*Ipomoea batatas* L.), particularly its leaves, which are rich in bioactive compounds such as flavonoids, and polyphenols.[12-14] Purple sweet potato leaves have long been utilized in traditional medicine due to their strong antioxidant and anti-inflammatory properties.The extract of purple sweet potato leaves is known to contain flavonoid, which can scavenge free radicals and inhibit lipid peroxidation. Additionally, its phenolic compounds are capable of suppressing inflammatory mediators such as TNF-α and IL-6, which are known contributors to hepatic inflammation. This makes the plant a strong candidate for further exploration as a hepatoprotective agent.[15, 16]

Previous in vivo studies have shown that purple sweet potato leaf extract can reduce AST and ALT levels in animals exposed to hepatotoxins such as ethanol However, its effect on liver injury specifically induced by CCl_4 has not been thoroughly investigated. CCl_4-induced damage is particularly complex and chronic, necessitating rigorous evaluation of any therapeutic agent claiming hepatoprotective activity.[17-19]

This study aims to evaluate the hepatoprotective effect of purple sweet potato leaf extract on serum AST and ALT levels in male Wistar rats induced with CCl_4. The central objective is to assess whether administration of the extract can significantly attenuate the elevation of these liver enzymes, thereby indicating a protective effect on liver tissue. Additionally, this study seeks to identify the optimal dose that yields the maximum protective benefit.

This research holds both scientific and clinical relevance given the high prevalence of liver diseases caused by toxic exposure and drug-induced injury. If proven effective, purple sweet potato leaf extract could be developed into a safe, affordable, and accessible phytopharmaceutical agent. This is particularly important in resource-limited settings where access to conventional liver treatments is constrained.

To date, there remains a paucity of systematic investigations into the effect of purple sweet potato leaf extract on specific hepatic biomarkers such as AST and ALT in CCl_4-induced models. Prior studies have mostly

focused on the tubers or generalized antioxidant properties, rather than evaluating its specific hepatoprotective effects.[20] Thus, this study addresses a critical research gap that warrants further exploration.

This study offers novelty by investigating the hepatoprotective potential of purple sweet potato *leaves*, a plant part less commonly studied compared to its tubers. It is also one of the few studies to use a CCl_4-induced rat model to quantitatively assess the extract's impact on key liver enzymes, contributing original data to the fields of pharmacology and toxicology.

Experimental Section

This experimental study utilized a total of twenty-four healthy male Wistar rats (Rattus norvegicus), aged 8–10 weeks and weighing between 180–200 grams. All animals were obtained from a certified laboratory animal facility and acclimatized for one week under standard laboratory conditions. These included a controlled temperature of 22–24°C, a 12-hour light/dark cycle, and ad libitum access to commercial pellet feed and clean drinking water. The animals were observed daily during the acclimatization period to ensure uniform health and to minimize environmental stress before the treatment phase began.

Following acclimatization, the animals were randomly divided into six groups (n = 4 per group): (1) Normal control group (no treatment), (2) Negative control group (received only CCl_4), (3) Positive control group (received silymarin at 50 mg/kg body weight daily), (4) Low-dose group (received purple sweet potato leaf extract at 250 mg/kg body weight daily), (5) Medium-dose group (500 mg/kg body weight daily), and (6) High-dose group (750 mg/kg body weight daily). The extract and silymarin treatments were administered orally using a gastric gavage once daily for three weeks.

To induce liver injury, a hepatotoxic solution of carbon tetrachloride (CCl_4) was prepared by mixing pure CCl_4 with olive oil in a 1:1 ratio. This solution was administered intraperitoneally at a dose of 1 mL/kg body weight, twice a week for three weeks, to all groups except the normal control. The negative control group received CCl_4 only, whereas the positive control group was co-treated with silymarin. The three experimental groups received CCl_4 alongside their respective doses of purple sweet potato leaf extract. All interventions were conducted consistently for 21 consecutive days.

Purple sweet potato (Ipomoea batatas L.) leaf extract was prepared through ethyl acetate maceration. Dried leaf powder was macerated in ethyl acetate, filtered, and evaporated under reduced pressure using a rotary evaporator. The concentrated extract was stored in sealed containers at 4°C and freshly reconstituted in distilled water before oral administration. The dose selection was based on prior toxicological screening and literature demonstrating potential antioxidant and hepatoprotective effects.

At the end of the treatment period, all animals were fasted overnight. Blood samples were collected under light ether anesthesia via the retro-orbital plexus using sterile glass capillary tubes. The samples were transferred to serum separator tubes and centrifuged at 3000 rpm for 10 minutes to isolate the serum. The serum was then analyzed for levels of liver enzymes aspartate aminotransferase (AST) and alanine aminotransferase (ALT).

Biochemical analysis of AST and ALT was performed using fully automated clinical chemistry analyzers. Specifically, instruments were used *BioSystems BTS* analyzers. These devices measure enzyme activity in serum through spectrophotometric methods based on absorbance changes in standardized reagent reactions. The tests were conducted in duplicate to ensure reliability and accuracy, and all procedures followed the manufacturer's standard operating protocols. Data obtained were subjected to statistical analysis to evaluate the significance of enzyme level changes between experimental groupsrequire ethical approval must mention the authority that provided approval and the corresponding ethical approval code.

Results and Discussion

This study investigated the hepatoprotective effects of purple sweet potato (Ipomoea batatas L.) leaf extract against carbon tetrachloride (CCl_4)-induced liver damage by assessing serum AST and ALT enzyme levels. The data were collected from six groups: Normal Control, Negative Control (CCl_4 only), Positive Control (silymarin 50 mg/kg BW), and three experimental groups receiving the extract at low (250 mg/kg BW), medium (500 mg/kg BW), and high (750 mg/kg BW) doses. Blood samples were collected at the end of the third week of treatment, and enzyme levels were analyzed using a BioSystems BTS clinical chemistry analyzer.

The descriptive statistics of AST and ALT levels are summarized in the table below. The Negative Control group, which received only CCl_4 without any protective treatment, exhibited the highest mean AST (297.20 ± 71.43 U/L) and ALT (70.63 ± 4.68 U/L) levels, indicating significant liver damage due to hepatotoxin exposure. In contrast, the Normal Control group displayed the lowest enzyme levels, with a mean AST of 136.01 ± 8.93 U/L and ALT of 31.34 ± 9.50 U/L.

Treatment with silymarin (Positive Control group) resulted in a reduction of both enzymes, with AST and ALT levels at 163.45 ± 6.54 U/L and 46.78 ± 13.26 U/L, respectively. These values indicate a protective effect against CCl_4-induced hepatotoxicity. Similar trends were observed in the groups treated with purple sweet potato leaf extract. The high-dose group (750 mg/kg) demonstrated the most prominent hepatoprotection among the experimental groups, with AST reduced to 146.35 ± 6.24 U/L and ALT to 50.78 ± 4.58 U/L, closely resembling the silymarin group.

The medium-dose group (500 mg/kg) also showed substantial reductions in AST (166.89 ± 12.27 U/L) and ALT (53.78 ± 5.48 U/L). While the low-dose group (250 mg/kg) presented moderately elevated enzyme levels compared to medium and high doses, it still exhibited better outcomes than the negative control, with AST at 207.83 ± 24.03 U/L and ALT at 61.48 ± 4.74 U/L.

Table 1. Mean and Standard Deviation of AST and ALT Levels Across Treatment Groups

Group	ASTMean (U/L)	AST SD	ALT Mean (U/L)	ALT SD	n
Normal	136.01	8.93	31.34	9.50	4
Negative Control	297.20	71.43	70.63	4.68	4
Positive Control	163.45	6.54	46.78	13.26	4
Low Dose (250 mg)	207.83	24.03	61.48	4.74	4
Medium Dose (500 mg)	166.89	12.27	53.78	5.48	4
High Dose (750 mg)	146.35	6.24	50.78	4.58	4

Statistical analysis using one-way ANOVA showed significant differences between treatment groups for both AST ($F = 13.17$, $p < 0.001$) and ALT ($F = 11.43$, $p < 0.001$), indicating that the treatments had a significant effect on liver enzyme levels.

Post-hoc Tukey HSD analysis revealed that the Negative Control group was significantly different ($p < 0.05$) from all other groups in both AST and ALT levels. This confirms the effectiveness of both the positive control (silymarin) and the purple sweet potato leaf extract treatments in mitigating liver enzyme elevation induced by CCl_4. Additionally, the medium and high doses of the extract were statistically comparable to the silymarin group, suggesting similar hepatoprotective efficacy.

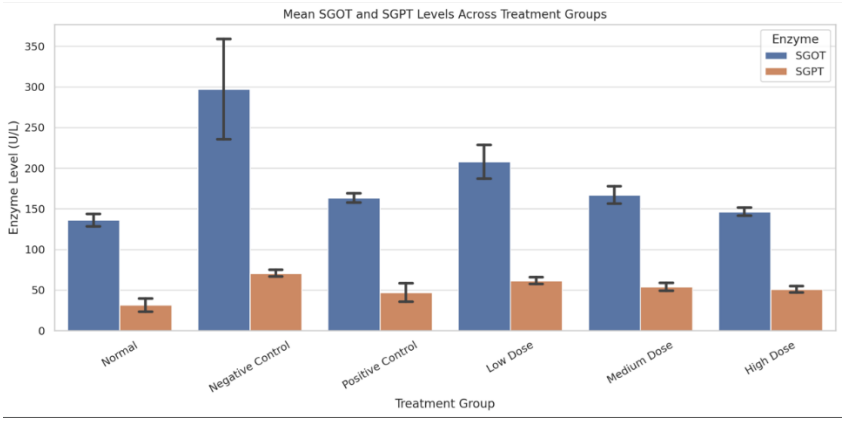

Figure 1. Mean AST (SGOT) and ALT (SGPT) Levels Across Treatment Groups

In summary, administration of purple sweet potato leaf extract effectively attenuated liver enzyme elevations induced by CCl_4 in a dose-dependent manner. The high dose was the most effective among the extract-treated groups, with results approaching those of the established hepatoprotective agent silymarin

The findings of this study demonstrate that purple sweet potato (Ipomoea batatas L.) leaf extract has a dose-dependent hepatoprotective effect in rats subjected to CCl_4-induced liver injury. The extract significantly reduced serum levels of AST and ALT, two well-established biomarkers of hepatic damage. These results are consistent with the hypothesis that phytochemicals with antioxidant and anti-inflammatory properties can mitigate chemical-induced hepatotoxicity.

Carbon tetrachloride (CCl_4) is a classic hepatotoxin that induces oxidative stress through the formation of reactive oxygen species (ROS) and lipid peroxidation, leading to hepatocyte necrosis and fibrosis (Jain et al., 2022). When metabolized by cytochrome P450 enzymes in the liver, CCl_4 produces trichloromethyl and peroxyl radicals that disrupt cellular membranes. These mechanisms explain the elevated AST and ALT levels observed in the negative control group of this study.

The significant reduction in liver enzymes observed in rats treated with purple sweet potato leaf extract supports previous reports indicating the antioxidant capacity of Ipomoea batatas leaves. The strong antioxidant capacity of purple sweet potato leaves is primarily attributed to their high content of polyphenols and flavonoids, which are known to neutralize reactive oxygen species and inhibit lipid peroxidation.[17] These compounds likely played a critical role in maintaining hepatocyte membrane stability and preventing oxidative liver damage in this study.

Notably, the hepatoprotective effect was most prominent at the highest dose (750 mg/kg), where AST and ALT levels were nearly comparable to those in the silymarin-treated group. Silymarin, derived from Silybum marianum, is a well-established reference compound known for its antioxidant and anti-fibrotic effects in liver injury models.[21] The similarity in outcomes suggests that the purple sweet potato leaf extract may offer comparable benefits as a natural alternative.

Previous studies have demonstrated that flavonoids and polyphenols not only neutralize reactive oxygen species (ROS) but also modulate key pro-inflammatory signaling pathways, including NF-κB and MAPK, which play a central role in liver inflammation.[20, 21] Inhibition of these pathways leads to reduced cytokine production, decreased leukocyte infiltration, and attenuation of fibrosis—hallmarks commonly observed in CCl_4-induced hepatic injury. Therefore, the anti-inflammatory and antioxidant mechanisms associated with these phytochemicals may further explain the hepatoprotective effects observed in this study.

The dose-dependent pattern of enzyme reduction reinforces the importance of optimal dosing in phytomedicine. While low doses (250 mg/kg) yielded some reduction in enzyme levels, the moderate (500

mg/kg) and high (750 mg/kg) doses provided statistically and clinically more relevant improvements. This pattern suggests that the efficacy of polyphenol-rich plant extracts is closely linked to bioavailability and cumulative antioxidant activity.[22-24]

The use of AST and ALT as primary indicators of hepatic injury is well-supported in toxicological and clinical hepatology literature. These enzymes are released into circulation when hepatocyte membranes are compromised. The observed normalization of AST and ALT levels in treated groups is a strong indicator of hepatocyte protection and repair, which aligns with the histopathological observations in similar studies.[5, 10]

Moreover, the study outcomes corroborate existing literature on the protective role of other plant-based antioxidants such as curcumin, resveratrol, and green tea catechins, which also modulate liver enzyme activity in hepatotoxin-exposed animals.[9, 10] These findings underscore the potential of dietary phytochemicals as low-toxicity, high-efficacy agents in liver disease prevention and management.

It is important to highlight that the purple sweet potato leaf extract showed hepatoprotective effects comparable to silymarin without observable toxicity. This is consistent with reports that Ipomoea batatas leaves are non-toxic and even used as dietary vegetables in many parts of Asia and Africa.[13, 14] This enhances the translational potential of the extract for therapeutic applications in humans.

The findings of this study contribute to the growing body of research advocating for the integration of traditional plant knowledge with modern pharmacological approaches. While silymarin remains the gold standard for hepatoprotection, the accessibility, affordability, and dietary compatibility of purple sweet potato leaves offer significant advantages, especially in low-resource settings.

Despite promising results, limitations of this study must be acknowledged. The sample size per group was modest, and only biochemical markers were assessed without histological confirmation. Future studies should include liver histopathology, oxidative stress biomarkers (e.g., MDA, SOD), and pro-inflammatory cytokines to better elucidate the mechanisms involved.

Conclusions

This study demonstrates that purple sweet potato (Ipomoea batatas L.) leaf extract exerts significant hepatoprotective effects in a CCl_4-induced liver injury model in rats, evidenced by the dose-dependent reduction in serum SGOT and SGPT levels. Among the treatment groups, the highest dose of 750 mg/kg body weight exhibited enzyme-lowering effects comparable to those of the standard hepatoprotective agent silymarin, indicating the therapeutic potential of this plant-based intervention. These findings highlight the role ofantioxidant-rich extracts in mitigating oxidative and inflammatory hepatic damage and support the feasibility of utilizing purple sweet potato leaves as a natural, accessible alternative in hepatoprotection strategies. Given its safety profile and bioactivity, further research should focus on isolating active constituents, exploring molecular mechanisms, and conducting clinical trials to evaluate its translational application in liver disease management.

Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this paper. The authors affirm that there are no personal, financial, or professional relationships that could be perceived as inappropriately influencing the interpretation or presentation of the research results.

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