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Original Article

Modulatory Effects of *Passiflora edulis* on Liver Function, Lipid Profile and Adipokines in Fructose-Induced Metabolic Syndrome in Rats

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ABSTRACT

Background: This research explores the restorative effects of *Passiflora edulis* extract counter to liver damage and metabolic disruptions caused by a high-fructose diet in female Wistar rats. **Materials and Methods:** The consumption of fructose resulted in increased levels of hepatic enzymes, signaling liver damage, along with notable changes in the lipid profile, including TG, TC and LDL and HDL all of which are characteristic of metabolic syndrome. Doses of 250, 500 and 1000 mg/kg of the *Passiflora edulis* extract were administered to evaluate its therapeutic potential. **Results:** The results demonstrated that extract meaningfully dropped SGPT and SGOT levels, suggesting liver protection, particularly at the higher doses (500 and 1000 mg/kg). However, the 250 mg/kg dose did not have a substantial outcome on ALP levels, indicating that a higher dosage is needed for optimal liver protection. Additionally, extract improved lipid profiles by falling TG, TC and LDL levels, while boosting HDL, indicating its potential to correct lipid imbalances associated with metabolic syndrome. At higher doses, extract also improved adipokines levels, increasing adiponectin and decreasing leptin and resistin, which are linked to better insulin sensitivity and reduced inflammation. Histopathological analysis showed that extract mitigated liver damage, with the most significant improvement observed at the 1000 mg/kg dose, where the liver tissue appeared almost normal. **Conclusion:** These results suggest that extract has beneficial effects on liver function and metabolic disturbances, predominantly at higher doses, positioning it as a hopeful substance for treating metabolic syndrome-related conditions. Subsequent studies are needed to evaluate the underlying mechanisms and assess the long-term effects of extract in metabolic diseases.

Keywords: Adipolines, Fructose, Hepatic Enzymes, Histopathological Analysis, Lipid Profile, Metabolic Syndrome.

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INTRODUCTION

Metabolic Syndrome (MetS) is a set of metabolic abnormalities including reduced insulin sensitivity, Imbalanced blood lipids, obesity and elevated blood pressure, which significantly increase the risk of developing cardiovascular diseases, chronic hyperglycemia and alcohol-independent fatty liver disease.[1] Among the crucial factors contributing to MetS, the excessive consumption of fructose has emerged as a major dietary factor that induces insulin resistance, alters lipid metabolism and promotes adipose tissue dysfunction.[2,3] The high intake of fructose leads to dysregulated lipid synthesis, increased liver fat accumulation and elevated circulating levels of pro-inflammatory cytokines, ultimately impairing liver function.[4,5]

Natural products, particularly plant-derived bioactive compounds, are being increasingly studied for their potential to modulate metabolic disturbances and mitigate the effects of MetS.[6] One such plant is *Passiflora edulis* (commonly known as passion fruit), which has been reported to possess comprehensive pharmacological effects, including free radical scavengers, reduced inflammation and liver-protecting actions.[7] *Passiflora edulis* is rich in polyphenols, carotenoids and flavonoids, compounds that have shown promise in reducing oxidative stress and improving lipid metabolism.[8] Furthermore, its effects on adipokines, which are secretory factors from adipose tissue and play a vital role in metabolic regulation, remain a subject of growing interest.

Liver function and lipid homeostasis are tightly interconnected with the secretion of adipokines such as leptin, adiponectin and resistin, which regulate energy balance, insulin sensitivity and inflammation.[9] Dysregulation of these adipokines is often observed in MetS and related conditions like fatty liver disease.[10,11] Given the growing body of evidence linking *Passiflora edulis* to

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improvements in metabolic disorders, the present study aims to explore its modulatory effects on liver function, lipid profile and adipokines in a rat model of fructose-induced MetS. Through this investigation, we seek to better understand whether Extract of *Passiflora edulis* (EPE) can mitigate the detrimental metabolic changes induced by high-fructose feeding, potentially offering a natural therapeutic option for managing MetS and its associated complications.

MATERIALS AND METHODS

Drugs and chemicals

Fructose was acquired at a local vendor and standard biochemical kits along with analytical-grade chemicals and reagents were utilized throughout the study.

Experimental animals

The female Wistar rats used in this study were supplied by the Zydus Research Centre in Ahmedabad, with each animal weighing between 200 and 250 g. The rats were maintained in a standardized environment with a 12 hr diurnal cycle and specific temperature maintained at 24±1°C and humidity levels between 35% and 60%. They had unrestricted to a pellet diet and water throughout the study. Before the experiment began, the rats underwent a 48 hr acclimatization period. The pre-clinical study was permitted by the Institutional Animal Ethics Committee.

Acute toxicity study

Oral acute toxicity testing was conducted on albino Wistar rats, with weights ranging from 200 and 250 g, adhering to the OECD guideline. Three female rats were administered varying doses of EPE orally. Their responses were monitored daily for 14 days, with observations made at intervals of 0, 30, 60, 120, 180 and 240 min.

Experimental design

The rats were randomly placed into 5 groups, with 6 animals in each group, to evaluate the effects of EPE on fructose-induced conditions. Group I acted as the normal control and was administered distilled water orally. In Group II, rats were provided with a fructose solution in their drinking water, establishing the fructose control condition.[12,13] Groups III, IV and V included rats on a fructose diet that received increasing doses of EPE (250, 500 and 1000 mg/kg, respectively) through oral administration. These groups were set up to investigate the potential dose-dependent effects of EPE on various parameters. A 20% fructose solution was used to induce MetS in all groups, except Group I. Over the 8-week study period, each group received their assigned treatment daily. At the conclusion of the experiment, blood was drawn from the retro-orbital plexus of anesthetized rats using glass capillaries. These samples were collected for subsequent biochemical analysis. After a 15 min clotting period, the blood was centrifuged at 5000 rpm for 20 min to separate the serum, which was then kept at -20°C until further testing. Standard diagnostic kits were used to measure biochemical markers, including lipid profiles and liver enzymes. Serum levels of adiponectin, leptin and resistin were quantified using ELISA.

Histopathology

Following euthanasia, liver tissues from each group were swiftly dissected, rinsed with saline and conserved in 10% phosphate-buffered formalin. The tissues were subsequently fixed in paraffin and thinly sliced into 5 μM thick sections, which were stained with hematoxylin and eosin. The stained sections were inspected using a light microscope and images were captured using an Olympus DP12 camera (Japan) to evaluate any histopathological alterations. The analysis was conducted by a pathologist who was blinded to the group allocations.

Statistical analysis

All results are reported as mean±SEM. Statistical changes between groups were assessed using one-way ANOVA, followed by the Bonferroni post hoc test when applicable, using Prism software (GraphPad). A *p*-value of less than 0.05 was acknowledged as statistically meaningful for all analyses.

RESULTS

Acute oral toxicity

Administering EPE at doses as high as 5000 mg/kg did not result in any signs of toxicity or mortality over a 14-day period. As a result, pharmacological investigations were carried out using EPE at doses corresponding to 1/20th (250 mg/kg), 1/10th (500 mg/kg) and 1/5th (1000 mg/kg) of the maximum dose.

Effect of EPE on liver enzymes against fructose-induced MetS

Fructose consumption in rats caused a marked increase in liver enzymes (SGPT, SGOT and ALP), signaling liver damage and dysfunction. This suggests that excessive fructose intake put strain on the liver, potentially leading to conditions such as fatty liver disease. However, treatment with EPE at doses of 250, 500 and 1000 mg/kg led to noteworthy drops in SGPT and SGOT levels compared to the untreated fructose group, suggesting that EPE helps protect liver function. Notably, the 250 mg/kg dose of EPE did not significantly change ALP levels, suggesting that lower doses may not be effective in addressing all forms of liver dysfunction. In contrast, higher doses of EPE (500 and 1000 mg/kg) resulted in a substantial decline in ALP levels, demonstrating a more pronounced therapeutic effect at these doses (Figure 1).

Effect of EPE on lipid profiles against fructose-induced MetS

The findings show that rats fed with fructose experienced significant disruptions in their lipid profiles, marked by increased levels of Triglycerides (TG), Total Cholesterol (TC) and Low-Density Lipoprotein (LDL), together with a fall in High-Density Lipoprotein (HDL) levels. These lipid imbalances are characteristic of MetS and suggest that the fructose-treated rats were in a state of dyslipidemia, a recognized risk factor for cardiovascular diseases. However, administration of EPE (at doses of 250, 500 and 1000 mg/kg) led to substantial improvements in lipid levels, with reductions in TC, TG and LDL and an increase in HDL. These results suggest that EPE may help reverse the lipid abnormalities induced by excessive fructose consumption, indicating its potential to mitigate the metabolic disruptions linked to MetS. This underscores EPE's therapeutic potential for addressing lipid-related issues in conditions like MetS, where lipid imbalances are a key factor in disease development (Figure 2).

Effect of EPE on adiponectin, resistin and leptin against fructose-induced MetS

In fructose-fed control rats, significant changes in adiponectin, leptin and resistin levels were observed, with a decrease in adiponectin and an increase in leptin and resistin, indicating metabolic disturbances linked to insulin resistance and inflammation. When treated with EPE at doses of 500 mg/kg and 1000 mg/kg, rats showed notable improvements, including an upsurge in adiponectin and a drop in leptin and resistin

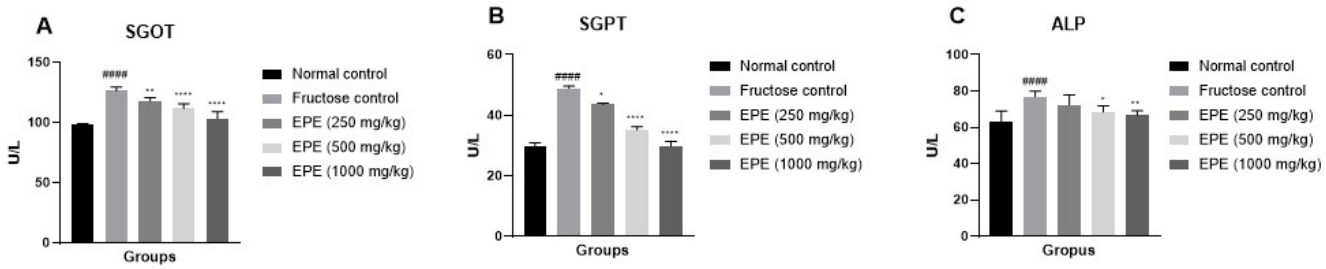

Figure 1: Impact of EPE on (A) SGOT (B) SGPT and (C) ALP in fructose-induced MetS. Values are stated as mean±SEM; n=6 $^{\#\#\#\#}p<0.0001$ relative to normal control; $^{*}p<0.05$, $^{**}p<0.01$, $^{****}p<0.0001$ relative to fructose control.

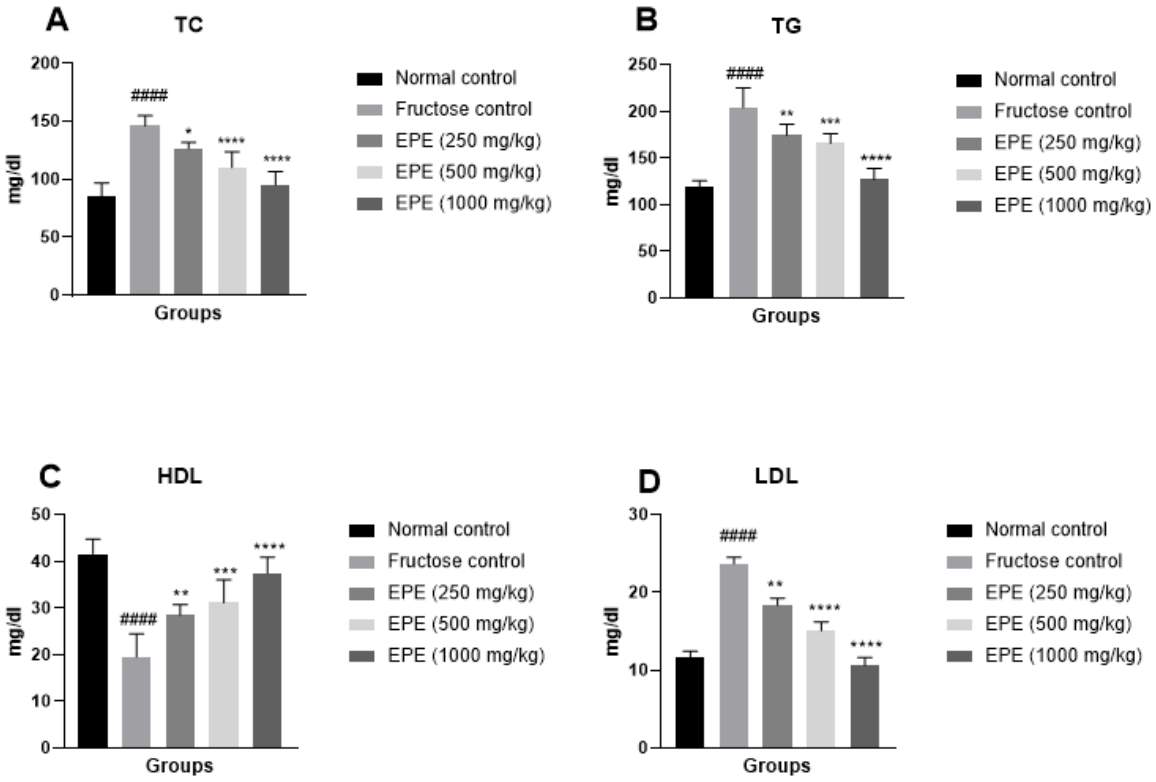

Figure 2: Impact of EPE on (A) TC (B) TG (C) HDL and (D) LDL in fructose-induced MetS. Values are stated as mean±SEM; n=6 $^{\#\#\#\#}p<0.0001$ relative to normal control; $^{*}p<0.05$, $^{**}p<0.01$, $^{***}p<0.001$, $^{****}p<0.0001$ relative to fructose control.

levels, suggesting that EPE helps restore a balanced adipokine profile and may improve metabolic health. However, the 250 mg/kg dose of EPE did not significantly alter leptin and resistin levels, indicating that a higher dose may be more effective in modulating these adipokines and improving metabolic outcomes in fructose-fed rats (Figure 3).

Histopathology study

In normal control rats, hepatocytes exhibited a healthy appearance with a typical radial arrangement, indicative of normal liver function. In contrast, fructose-fed control rats displayed significant vacuolization in hepatocytes, a hallmark of glycogen infiltration, leading to the development of fatty liver. This suggests that the fructose diet caused metabolic disturbances that resulted in lipid accumulation and liver damage. When treated with EPE, rats at the 250 mg/kg dose showed a reduction in vacuolization and only mild fatty liver, indicating some protective effects against liver damage, though not fully restoring normal liver function. At the 500 mg/kg dose, treatment resulted in mild vacuolization, but the hepatocytes appeared largely normal, reflecting a more pronounced improvement in liver health. The most significant effect was seen at the 1000 mg/kg dose, where the liver cells appeared completely normal, with no visible signs of vacuolization or fatty liver. These findings suggest that EPE has a dose-dependent protective effect on liver health, particularly in counteracting the hepatic damage induced by a fructose-rich diet, with higher doses providing more effective protection and restoration of normal liver structure (Figure 4).

DISCUSSION

This study explores the impact of EPE on liver enzymes and metabolic disturbances triggered by a high-fructose diet, offering significant insights into its potential therapeutic benefits. The observed rise in liver enzymes (SGPT, SGOT and ALP) in fructose-fed rats is consistent with previous research linking excessive fructose intake to liver dysfunction and the development of fatty liver disease.[14,15] The notable decrease in SGPT and SGOT levels following EPE administration at doses of 500 and 1000 mg/kg supports previous findings on the hepatoprotective

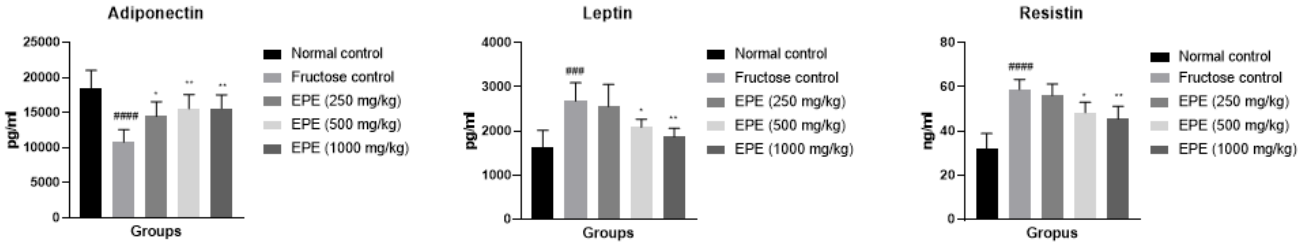

Figure 3: Impact of EPE on (A) Adiponectin (B) Leptin and (C) Resistin in fructose-induced MetS. Values are stated as mean±SEM; $n=6$ $^{###}p<0.001$, $^{####}p<0.0001$ relative to normal control; $^{*}p<0.05$, $^{**}p<0.01$, relative to fructose control.

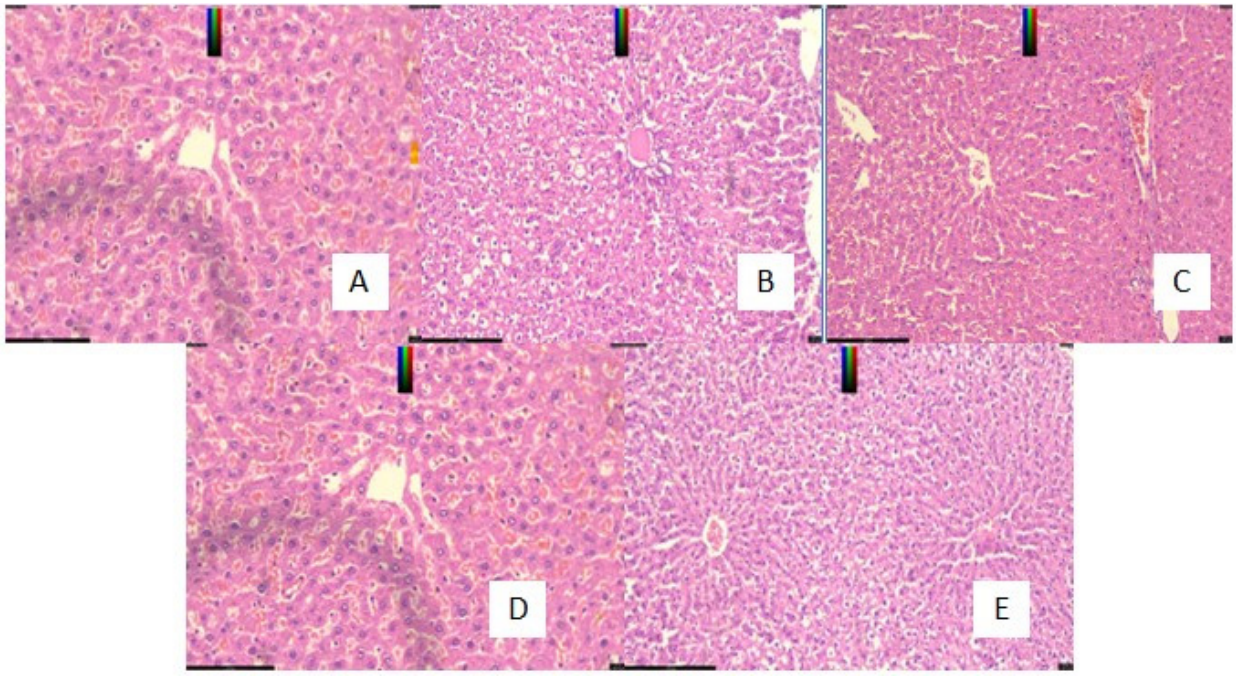

Figure 4: Light microscopy of liver tissue from rats (A) Normal control (B) Fructose control (C) EPE (250 mg/kg) (D) EPE (500 mg/kg) and (E) EPE (1000 mg/kg).

properties of various plant extracts in reducing elevated liver enzyme levels.[16] However, the lack of significant change in ALP levels with the 250 mg/kg dose contrasts with studies suggesting that lower doses of extract of *Passiflora* spp. may be insufficient in fully addressing certain forms of diabetes induced liver dysfunction[17] underscoring the need for dose optimization to achieve the desired therapeutic effect.

Concerning lipid profiles, fructose-induced dyslipidemia in rats, marked by elevated TG, TC and LDL levels and a reduction in HDL, reflects the metabolic disturbances observed in other models of MetS.[18] The improvements in lipid profiles following EPE treatment, especially the reductions in TC, TG and LDL, along with an increase in HDL, are consistent with studies indicating that natural compounds can correct lipid imbalances linked to MetS.[19] These results highlight the potential of EPE as an effective agent in addressing lipid abnormalities associated with MetS.

As for adipokine levels, fructose-fed rats showed altered metabolic markers, including decreased adiponectin and increased leptin and resistin levels, which are indicative of insulin resistance and inflammation.[20,21] The beneficial effects of EPE, especially at higher doses, in increasing adiponectin levels and decreasing leptin and resistin, support previous findings suggesting that dietary and plant-based interventions can help restore a balanced adipokine profile and improve metabolic health.[22] However, the lack of significant changes with the 250 mg/kg dose points to the dose-dependent nature of EPE's effects, suggesting that higher doses may be necessary to achieve more substantial metabolic improvements.

Histopathological analysis revealed significant liver damage in fructose-fed rats, including vacuolization and fatty liver, consistent with prior studies showing fructose-induced hepatic injury.[14] The protective effects of EPE, particularly at the 500 mg/kg and 1000 mg/kg doses, in alleviating liver damage and restoring normal liver structure, are in line with earlier research on the hepatoprotective benefits of various natural plants and compounds.[16,23,24]

CONCLUSION

In conclusion, this study demonstrates the restorative effects of EPE against MetS persuaded by a fructose-rich diet. EPE treatment, particularly at higher doses, significantly reduced liver enzyme levels, improved liver histopathology and reversed liver dysfunction in a dose-dependent manner. The absence of significant effects at lower doses suggests that higher doses are more effective in mitigating liver damage. Moreover, EPE treatment improved lipid profiles, including reductions in triglycerides, total cholesterol and LDL, while increasing HDL, highlighting its potential to address lipid imbalances linked to MetS. Additionally, EPE positively influenced adipokine levels by increasing adiponectin and decreasing leptin and resistin, which could improve insulin sensitivity and reduce inflammation. These findings emphasize the therapeutic potential of EPE for managing liver dysfunction and metabolic abnormalities, particularly in conditions such as MetS. Further investigation is needed to understand its mechanisms and long-term effects on metabolic diseases.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ABBREVIATIONS

ALP: Alkaline Phosphatase; **ELISA:** Enzyme-Linked Immunosorbent Assay; **EPE:** Extract of *Passiflora edulis;* **HDL:** High-Density Lipoprotein; **LDL:** Low-Density Lipoprotein; **MetS:** Metabolic Syndrome; **OECD:** Organisation for Economic Co-operation and Development; **SEM:** Standard Error of the Mean; **SGOT:** Serum Glutamate Oxaloacetate Transaminase; **SGPT:** Serum Glutamate Pyruvate Transaminase; **TG:** Triglycerides.

FUNDING

This study was conducted without any external or institutional funding support.

SUMMARY

This study highlights the therapeutic potential of EPE in mitigating MetS induced by a fructose. High-dose EPE treatment significantly improved liver function, as evidenced by reduced liver enzyme levels, enhanced histopathological profiles and reversal of liver dysfunction in a dose-dependent manner. While lower doses showed minimal impact, higher doses were effective in ameliorating liver damage. EPE also improved lipid metabolism by lowering triglycerides, total cholesterol and LDL, while elevating HDL levels. Additionally, it modulated adipokines by increasing adiponectin and reducing leptin and resistin, suggesting enhanced insulin sensitivity and reduced inflammation. These findings underscore EPE's potential in managing liver dysfunction and metabolic disturbances associated with MetS, warranting further exploration of its mechanisms and long-term efficacy.

ETHICAL APPROVAL

The research protocol (SVU/DP/IAEC/2021/08/47) was approved by the Institutional Animal Ethics Committee of SBKS MI & RC and adhered the standard guidelines.

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