



Dietary fiber of jicama (*Pachyrhizus erosus* L) tuber exerts hepatoprotective effect against high-sugar drinks in mice

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ABSTRACT

Introduction: Jicama (*Pachyrhizus erosus*, family Fabaceae) is a potent medicinal plant. Although extensive studies report the health benefits of jicama extract, few studies have investigated the efficacy of its dietary fiber in preventing metabolic diseases, including liver disease. The present study aimed to elucidate whether dietary fiber obtained from the jicama tuber counteracts the development of liver disease induced by high-sugar drinks.

Methods: Twenty-four adult male mice (DDY strain; 2 months old with bodyweight 22-25 g) were randomized into three groups: normal drink (ND), fed with tap water and standard chow; high-sucrose drink (HSD), fed with a high-sucrose drink and standard chow; and high-sucrose drink plus standard chow with 25% jicama fiber (HSD + JF 25%). After the mice were on their respective diets for ten weeks, the following parameters were measured: body weight, liver weight, malondialdehyde (MDA), histopathological alterations, blood glucose, and serum glutamate-pyruvate transaminase (SGPT).

Results: Mice in the HSD + JF 25% group had significantly lower body weight ($P < 0.01$), liver weight ($P < 0.05$), MDA ($P < 0.01$), blood glucose ($P < 0.01$), and SGPT ($P < 0.01$) compared to those in the HSD group. They also had fewer histopathological alterations in the liver, as demonstrated by a lower proportion of degenerated cells and an overall lower histopathological score than those in the HSD group ($P < 0.05$).

Conclusion: Adding jicama fiber (25% of standard chow) mitigates the increase in blood glucose and body weight and histopathological changes in the liver induced by high-sucrose drinks, showing liver protective activity.

Implication for health policy/practice/research/medical education:

The present study demonstrated that supplementation of fiber extracted from jicama (*P. erosus*) tuber in the diet counteracts liver disease caused by a high-sugar drink in an animal model. It suggests the potential for further investigations in human subjects to determine whether jicama fiber is effective as a dietary supplement to preclude metabolic disorders such as non-alcoholic fatty liver disease (NAFLD).

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Introduction

Jicama (*Pachyrhizus erosus*, Fabaceae) is a medicinally potent plant that provides several health benefits, such as the inhibition of platelet aggregation in humans and, therefore, the prevention of strokes and heart attacks (1). Jicama tuber extract reduced postprandial blood glucose and improved glucose tolerance in rats with streptozotocin-induced diabetes (2). It also inhibited the activity of α -glucosidase and α -amylase, two enzymes responsible for carbohydrate metabolism in the gastrointestinal tract;

the inhibition of α -amylase by jicama was higher than that by acarbose (2). Moreover, jicama could improve insulin sensitivity (3). Another study demonstrated that jicama yogurt reduced blood glucose in mice with diabetes (4). Therefore, consumption of jicama tuber is likely to be beneficial for those with diabetes and other metabolic dysregulation disorders.

Although many studies have reported the health benefits of jicama extract, investigations explicitly focusing on the dietary fiber of jicama tuber are few. Dietary fiber

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extracted from the jicama tuber modulated immune responses in splenocytes *in vitro* (5), which may be due to the polysaccharide pectin found in the jicama tuber (6). Our previous study also revealed that supplementation of jicama fiber effectively counteracted excessive body weight increase in mice fed with a high-sucrose powder (7). Furthermore, jicama fiber could elicit a preventive effect against liver injury in mice (8). We also found that jicama fiber is effective in preventing the development of hyperglycaemia caused by high-fat diet in mice (9). Taken together, those findings suggest that jicama fiber is beneficial to prevent the development of diet-induced metabolic diseases. However, whether jicama fiber can exert a preventive effect against liver degeneration caused by high-sugar drinks remains unknown.

Frequent consumption of high-sugar beverages, including those with high-fructose corn syrup, has been correlated with the development of metabolic disorders in children and adolescents (10). Likewise, a cohort study revealed that consumption of sugary drinks is correlated with the prevalence of cardiovascular disease (11). Long-term consumption of sugary drinks increased the incidence of type 2 diabetes in young adults (12) and the development of liver diseases, especially non-alcoholic fatty liver disease (NAFLD) (13,14). The present study aimed to determine whether dietary fiber extracted from the jicama tuber is therapeutic against liver disease induced by high-sugar drinks. We hypothesized that dietary jicama fiber supplementation may prevent histopathological damage and oxidative stress in the liver caused by high-sugar drinks.

Materials and Methods

Sample collection and authentication

The jicama tubers were obtained from a farm field in Kuranji, Padang, West Sumatra. A certified plant taxonomist authenticated the species identity, and a voucher specimen was deposited in the Herbarium ANDA, Andalas University (ID: 062/K-ID/ANDA/1/2022).

Extraction of dietary fiber

Jicama tubers were washed with tap water, peeled, and subsequently ground using an electric grater for 20 minutes. The fiber was extracted from the samples in distilled water according to the procedures described elsewhere (5,7). The powder of the fiber obtained from the extraction was used for the experiment.

Experimental animal model and treatments

We used adult male albino mice ($n = 24$; DDY strain; 2 months old with bodyweight 22–25 g), purchased from Balai Veteriner Baso, Bukittingi, West Sumatra. The mice were kept individually in cages (one mouse/cage), acclimatized in the animal room for seven days (25–26°C, 68% humidity, 12-hour light/dark cycle) and fed with a

standard chow diet (RATBIO: Citra Ina Fedmill, Jakarta-Indonesia) and tap water *ad libitum*. After acclimatization, the mice were randomized into three groups: normal drink (ND), fed with tap water and standard chow; high-sucrose drink (HSD), fed with a high-sucrose drink and standard chow; and high-sucrose drink plus standard chow diet with 25% jicama fiber (HSD + JF 25%). The high-sucrose drink was 30% sucrose (Sigma-Aldrich, Burlington, USA) in distilled water (gram/volume) and provided in the water bottle *ad libitum*. The HSD + JF 25% diet was prepared by combining jicama fiber with standard chow at a ratio of 25g jicama fiber/75 g standard chow. The dose of jicama fiber was based on our previous studies (7–9). All animal handling and uses in this study were performed per guidelines established by the Committee of Ethical Code for Research of Faculty of Medicine, Andalas University (Approval No.528/ UN.16.2/ KEP-FK/2021).

Monitoring of blood glucose and body weight

Blood glucose levels were measured biweekly at 9 a.m. when the mice were in the fed state. The blood sample was drawn from the tail tip and the glucose concentration was determined using an automatic glucometer (AGM-4000; All Medicus Co. Ltd.). Body weight was measured twice, namely, on the first day of treatment and at the end of treatment (week 10), using a sensitive digital balance for animals (PCE-BDM 1.5, Benchtop; PCE Instruments).

Measurement of serum glutamate-pyruvate transaminase (SGPT) in the blood

At the end of the experiment, animals were sacrificed through dislocation of the cervical vertebrae. Blood was drawn by cardiac puncture and immediately centrifuged at 4000 rpm for 10 minutes. Plasma was separated into sterile microcentrifuge tubes and kept at -80°C until used for SGPT measurement. The SGPT measurement was conducted using an alanine aminotransferase 1 mouse ELISA kit (BioVision, San Francisco, USA) according to the manufacturer's instructions. The determination of absorbance at the wavelength 365 nm was conducted by using UV-vis spectrophotometer (Bio-Rad UV-visible spectrometer SmartSpec™ Plus, California, USA).

Measurement of liver weight and liver index

The liver was weighed upon sacrifice and subsequently fixed in 10% formalin for histopathological examination. The liver index was calculated with the following formula:

$$\text{Liver index} = \frac{\text{Liver weight}}{\text{body weight}} \times 100\%$$

Measurement of malondialdehyde (MDA) in the liver tissue

A 0.5-g sample of liver tissue was homogenized in phosphate-buffered saline (PBS) before being centrifuged

(2000 rpm for 10 minutes at 4°C). The supernatant was collected and used for detection of MDA using a lipid peroxidation assay kit (Sigma-Aldrich, Burlington, USA). The procedure for the assay was performed as per the instructions described in the product kit. The absorbance of samples at wavelength 532 nm was determined by using a UV-Vis spectrophotometer.

Histological examination of liver

The 10% formalin-fixed samples of liver tissue were dehydrated with graded alcohol before being subjected to tissue processing and hematoxylin-eosin staining according to the procedures as described elsewhere (15). The histopathological observations were conducted by using a microscope (Olympus CX21, Tokyo, Japan). The normal and degenerated cells were counted based on the microscopic picture taken at 400× magnification. The histopathological alterations of the liver were analyzed semi-quantitatively using the Manja Roenigk scoring system as previously described (15).

Statistical analysis

Data are presented as mean \pm standard error (SE). Quantitative data were analyzed by ANOVA followed by Duncan's new multiple range test. Statistical significance was set at $P < 0.05$. All analyses were carried out by using SPSS version 26.

Results

Mice fed with a high-sucrose drink (HSD group and HSD + JF 25% group) exhibited higher blood glucose levels starting from the second week of treatment compared to those fed with the tap water drink (ND group) (Figure 1A). However, after six weeks of treatment, the mice in the HSD + JF 25% group showed a markedly lower blood glucose level that was not significantly different from that of the ND group. Furthermore, after 10 weeks, the mice in the HSD group had a significantly higher body weight compared to those in the ND and HSD + JF 25% groups. Body weight was not significantly different between ND and HSD + JF 25% groups (Figure 1B and C).

After 10 weeks, the livers of the mice in the HSD group were heavier than those of mice in the other two groups (Figure 2A), while the weights of the livers of mice in the HSD + JF 25% group were not significantly different when compared to those in the ND group. No significant differences in liver index values were detected (Figure 2B), but liver weights tended to be lower in mice in the HSD + JF 25% group compared to the other two groups.

Mice in the HSD group had significantly elevated SGPT as compared with those in other groups. However, the SGPT level of mice in the HSD + JF 25% group was comparable to the ND group (Figure 3A).

Mice in the HSD group exhibited significantly higher liver MDA compared to that in the ND and HSD + JF 25%

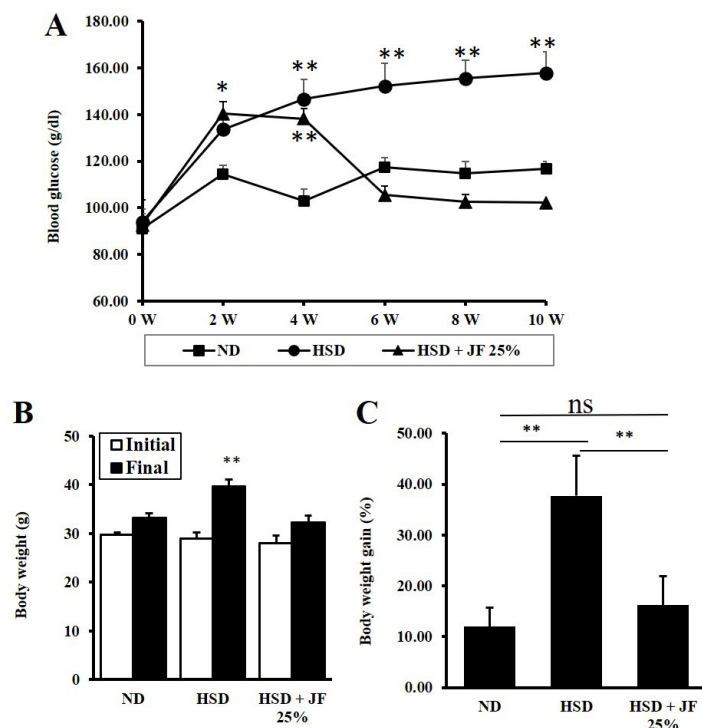


Figure 1. Effect of dietary jicama fiber on mouse blood glucose and body weight. (A) Biweekly blood glucose measurement; (B) Body weight at the beginning and the end of the diet treatments; (C) Body weight gain for ten weeks of treatment presented as percent increase. ND (normal tap water drink); HSD (high-sucrose drink); HSD + JF 25% (25% of jicama fiber in the diet). * and ** indicate statistically significant differences among groups at $P < 0.05$ and $P < 0.01$, respectively; ns (not significant).

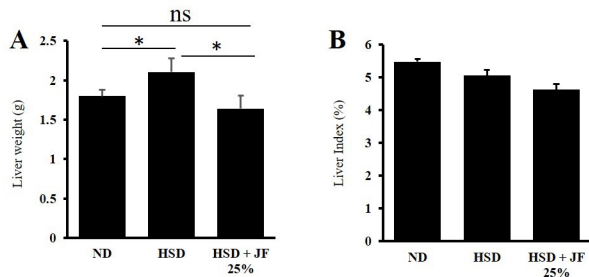


Figure 2. Effect of dietary jicama fiber on mouse liver weight and liver index. (A) Liver weight as determined at the end of treatment; (B) Liver index presented as the ratio of liver weight to total body weight. ND (normal tap water drink); HSD (high-sucrose drink); JF 25% (25% of jicama fiber in the diet); * indicates statistically significant differences between groups at $P < 0.05$; ns (not significant).

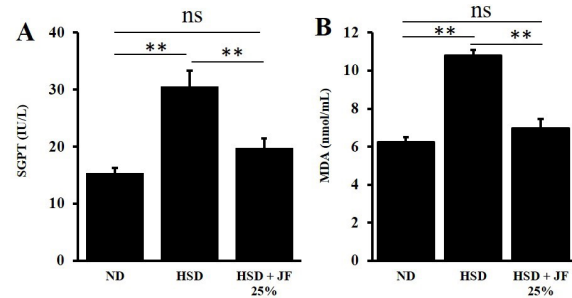


Figure 3. Effect of dietary jicama fiber on mouse serum glutamate-pyruvate transaminase (SGPT) levels and liver malondialdehyde (MDA) levels. (A) Level of SGPT in blood plasma; (B) Level of MDA in liver tissue. ND (normal tap water drink); HSD (high-sucrose drink); JF 25% (25% of jicama fiber in the diet); ** indicates statistically significant differences between groups at $P < 0.01$; ns (not significant).

groups. In contrast, there were no significant differences in liver MDA between the HSD + JF 25% group and the ND group (Figure 3B).

Mice in the HSD group experienced severe histopathological damage as indicated by degeneration of hepatocytes and the wall of the central vein (Figure 4A). In contrast, the histopathological alterations in the HSD + JF 25% and ND groups were lower. Microvesicles in the liver were also elevated in the HSD group but lower in the ND and HSD JF 25% groups.

Most hepatocytes were degenerated in the HSD group, whereas fewer were degenerated in the ND and HSD + JF 25% groups (Figure 4B). However, the magnitude of the degeneration remained markedly higher in the HSD + JF 25% group when compared to that of ND group.

The histopathological scoring data (Manja Roelingk score; Figure 4C) also indicated that the overall histopathological damages were significantly higher in the HSD group compared to those in the ND group and HSD + JF 25% group. Moreover, the injuries were also significantly different between HSD + JF 25% group and the ND group.

Discussion

In the present study, supplementation of dietary fiber, extracted from jicama tuber, could exert a preventive effect against pathological alterations in the liver caused by chronic consumption of the high-sugar drinks in mice. Jicama fiber inhibited the secretion of SGPT from the liver and the accumulation of MDA in the liver. Moreover, the magnitude of structural damage of the liver tissues caused

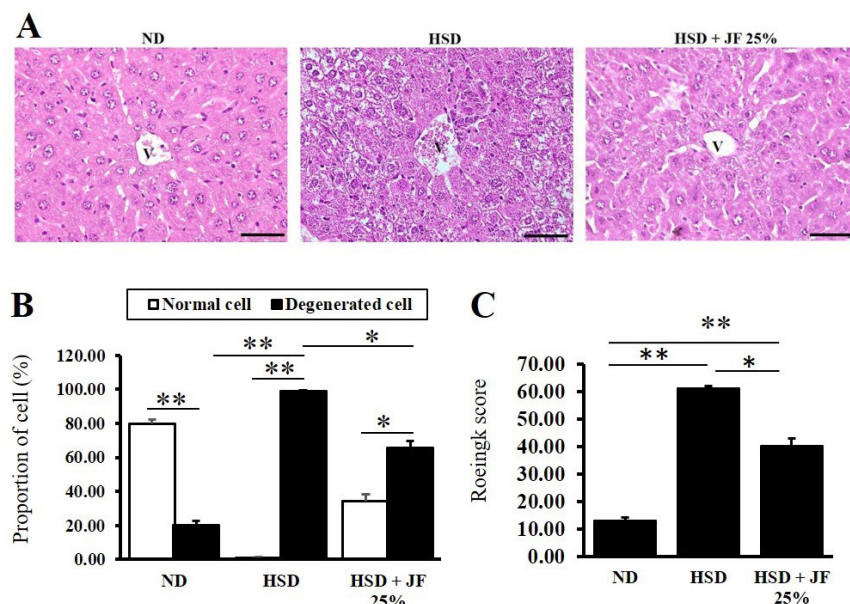


Figure 4. Effect of dietary jicama fiber on the histopathological alterations in mouse liver. (A) Microscopic features of mouse liver; (B) Proportion of normal and degenerated liver cells (C) Level of histopathological damage based on Roelingk score. ND (normal tap water drink); HSD (high-sucrose drink); JF 25% (25% of jicama fiber in the diet); v (central vein); * and ** indicate statistically significant differences at $P < 0.05$ and $P < 0.01$, respectively. Scale bars in A = 20 μ m.

by the high-sucrose drink was significantly lower in mice fed with jicama fiber.

A chronic intake of high-sugar drinks is associated with the hyperglycemic state and the subsequent development of diabetes mellitus and liver diseases (16,17). The liver plays a pivotal role in the regulation of blood glucose homeostasis (18), and an excessive intake of sugars increases the risk of NAFLD (19). In the present research, hyperglycemia induced by high-sugar drinks was reduced by jicama fiber supplementation within six weeks of treatment. This counteractive effect was sustained for four weeks, until the conclusion of the experiment. Thus, the hepatoprotective effect of jicama fiber might be due to its ability to suppress the elevation of blood glucose induced by high-sugar drinks.

We also found that weight gain induced by high-sugar drinks in mice was reduced by the supplementation of dietary jicama fiber. Likewise, jicama fiber also mitigated the increase in liver weight induced by high-sugar drinks. Excessive increase in body weight leading to obesity could be caused by a marked increase in adiposity, including central and ectopic adiposity (20). Our previous studies have shown that jicama fiber, especially at the dose of 25% of the diet, reduces the central and ectopic adiposity induced by a high-sugar diet (7,8). Another report also suggested that the accumulation of fat in the liver in the mice fed with an atherogenic diet could be attenuated by the supplementation of guar gum, a water-soluble dietary fiber (21). Taken together, our data and the existing literature indicate that the protective effects of dietary fiber, including jicama fiber, could be due to the prevention of adiposity.

In our study, supplementation of jicama fiber in the diet effectively prevented the plasma SGPT elevation caused by the high-sucrose drink. SGPT is a hepatic enzyme that is excessively secreted into the blood when there is extensive degeneration of liver tissue (22). Excessive consumption of high-sugar drinks promotes liver degeneration through hyperglycemia-induced glucotoxicity (23,24). Accordingly, an improvement in glycemic profile could lead to prevention of glucotoxicity, thereby reducing the SGPT increase in blood plasma (24). Studies in humans found that dietary fiber consumption is associated with improved glycemic control and reduction of plasma SGPT level (25). Accordingly, we suggest that jicama fiber may prevent the increase in SGPT induced by high-sucrose drinks through sustained glycemic control. However, this remains to be confirmed.

A high glucose environment could lead to a marked increase in oxidative stress (26). In our study, an increase in hepatic MDA level, as a marker of oxidative stress, was effectively prevented by the supplementation of jicama fiber. Dietary fiber has been suggested to exert antioxidant effects, thereby reducing oxidative stress (27). Hence, the counteractive effect exerted by the jicama fiber against the

increase of MDA induced by the high-sugar drink could be associated with its antioxidant effect. Further studies on the antioxidant properties of jicama fiber are needed to confirm this speculation.

In this study, histopathological alterations of the liver induced by the high-sugar drink were partially prevented by the supplementation of jicama fiber in the diet. Dietary fiber reduces sugar diffusion from the lumen to the brush-border layer in the small intestine, attenuating the absorption of sugar and the related glycaemic responses, and thereby protecting the liver (28). Polysaccharides from *Acacia senegal* decreased the levels of intestinal sodium-glucose transporter 1 (SGLT1) in the membrane vesicle of the jejunal brush border, thereby reducing the detrimental effects of high-sugar drinks, such as liver damage (29). Thus, we suggest that in addition to its antioxidant effect, the hepatoprotective effect of jicama fiber may be linked to its counteractive action against glucose absorption in the intestine.

Products of fiber fermentation in the large intestine, including short-chain fatty acids (SCFAs), exert various beneficial effects on the body's health. For instance, polysaccharides derived from noni fruits, including dietary fiber, alleviated oxidative stress in the liver by modulating microbiome composition and SCFA levels and sustaining the integrity of intestinal cells (30). Inulin, a water-soluble dietary fiber that is also found in the jicama tuber, reduced inflammation in the liver through a mechanism involving modulation of macrophages by SCFAs in mice (31). Moreover, increasing dietary fiber intake also has been associated with the prevention of fibrosis in mouse liver by modulating intestinal microflora and immune responses (21,31).

There were some limitations of our present study. We did not determine the inhibitory effect of jicama fiber on glucose absorption in the intestine. Moreover, we did not investigate the inflammatory markers accompanying high-sugar diet-induced liver degeneration. Consequently, we could not provide the mechanistic evidence demonstrating whether jicama fiber acts directly or indirectly to protect the liver from the detrimental effect of high-sugar drinks. Our study was carried out for a relatively short period of treatment (10 weeks). Thus, it remains unknown whether the hepatoprotective effect of jicama fiber would be sustained for a longer time without side effects. Furthermore, we did not measure SCFAs (butyrate, acetate, and propionate) in the ceca or blood plasma. As a result, it is not known whether jicama fiber supplementation increases the production of SCFAs and provides subsequent beneficial outcomes to the liver in animals fed high-sugar drinks.

Conclusion

The present study revealed that supplementation of jicama (*P. erosus*) fiber in the diet is protective against the effects

of high-sugar drinks on the liver. The results observed in mice may reflect a similar response in humans suffering from NAFLD induced by high-sugar drinks. Hence, frequent consumption of jicama fiber might mitigate the NAFLD-associated health problems caused by high-sugar drinks.

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Authors' contribution

PS, RM, and RO designed and performed the experiments and collected the data. PS and RM analysed the data. PS and RSR prepared the manuscript. All authors read and approved the manuscript for publication.

Conflict of interests

There is no conflict of interest.

Ethical considerations

All protocols applied for this research were approved by the Committee of Ethical Code for Research of Faculty of Medicine, Andalas University (Approval No.528/UN.16.2/ KEP-FK/2021). All authors have inspected carefully and cleared the issues related to misconduct, plagiarism, fabrication, and redundancy associated with the manuscript.

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