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Protective Effects of a Bioactive Compound from Eugenia Jambolana Fruit Pulp Against Fructose Induced Insulin Resistance in Rats

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Abstract

Aim: The present study was designed to evaluate the preventive potential of an active principle (FIIc) isolated from the fruit pulp of Eugenia jambolana against fructose-induced insulin resistance in rats.

Methodology: A crude aqueous extract of Eugenia jambolana fruit pulp was subjected to ion-exchange column chromatography to obtain fraction FII, which was further purified to yield FIIc. The purity of FIIc was confirmed by high-performance liquid chromatography (HPLC). FIIc was administered orally to experimental rats at a dose of 15mg/kg body weight for 60 days. Body weight, fasting blood glucose, serum triglycerides, total cholesterol, LDL-C, HDL-C, liver and skeletal muscle glycogen levels, tumor necrosis factor-α (TNF-α), serum insulin, insulin resistance (HOMA-IR), and insulinogenic index were assessed at 30-day intervals over the study period.

Results: Fructose feeding for 60 days resulted in a significant increase ($p < 0.001$) in serum biochemical parameters along with a marked reduction in liver and skeletal muscle glycogen levels in untreated fructose-fed rats. Oral administration of FIIc significantly reduced fasting blood glucose levels ($p < 0.001$) compared with the fructose control group. Significant improvements ($p < 0.001$) were also observed in body weight, lipid profile, and tissue glycogen content following FIIc treatment. Serum TNF-α and insulin levels were restored toward normal values. Additionally, insulin resistance and insulinogenic index showed significant improvement compared with untreated fructose-fed rats.

Conclusion: These findings demonstrate that FIIc exerts a protective effect against fructose-induced insulin resistance, suggesting its potential role in preventing metabolic dysfunction associated with excessive fructose intake.

Keywords: Eugenia jambolana; Insulin resistance; Tumor necrosis factor α; Fructose.

Introduction

Over the past few decades, there has been a substantial rise in per capita fructose consumption, largely due to the widespread use of sucrose and high-fructose corn syrup (HFCS) in the food industry [1,2]. Excessive fructose intake has been associated with several adverse metabolic consequences, including hyperlipidemia, hyperinsulinemia, insulin resistance, hyperuricemia, hypertension, glucose intolerance, and enhanced non-enzymatic fructosylation of proteins [3-5].

Fructose metabolism differs fundamentally from glucose metabolism. Upon ingestion, fructose is rapidly phosphorylated by fructokinase to fructose-1-phosphate, which is subsequently cleaved by aldolase B into glyceraldehyde and dihydroxyacetone phosphate. These intermediates bypass the rate-limiting step of glycolysis catalyzed by phosphofructokinase. Consequently, hepatic fructose metabolism proceeds in a relatively unregulated manner, promoting de novo lipogenesis and increased triglyceride synthesis, thereby rendering fructose more lipogenic than glucose [6-8]. Experimental models employing high-fructose diets have been widely used to induce insulin resistance in rodents. Such models exhibit hallmark features including hyperglycemia, hypertriglyceridemia, and hyperinsulinemia, closely resembling metabolic disturbances observed in humans [10-15].

Therefore, fructose-fed rats provide a suitable and well-established model for investigating potential therapeutic agents targeting insulin resistance. The present study evaluates the effect of a purified active compound (FIIc), isolated from the fruit pulp of Eugenia jambolana, on fructose-induced insulin resistance. Previous investigations have demonstrated the antihyperglycemic, antihyperlipidemic, and organ-protective properties of FIIc in experimental diabetic models [16-20]. However, its role in preventing insulin resistance in a diet-induced prediabetic state has not been previously explored. This study, therefore, aims to elucidate the protective effects of FIIc on insulin resistance, inflammatory markers such as TNF-α, and tissue glycogen levels in fructose-fed rats.

Materials and Methods

Plant material

Fruits of Eugenia jambolana were procured from the Azadpur Mandi herbal market, Delhi, India. Botanical authentication was performed by a qualified taxonomist, and a voucher specimen (No. P-96/7) was deposited for future reference.

Preparation of crude aqueous extract

Fresh fruits were thoroughly washed, and seeds were removed. The fruit pulp was homogenized with distilled water, filtered, centrifuged, and subsequently lyophilized to obtain the crude aqueous extract.

Isolation and Chemical Characterization of FIIc. The lyophilized extract was subjected to DEAE-52 ion-exchange chromatography to isolate fraction FII, which was further purified to obtain FIIc. Purity was confirmed by HPLC analysis. Spectroscopic characterization identified FIIc as α-hydroxy succinamic acid ($C_4H_7O_4N$) [16].

Experimental design

Male Wistar albino rats were randomly assigned to three groups:

i. Chow control

ii. Fructose control

iii. Fructose + FIIc (15mg/kg body weight)

FIIc was administered orally for 60 days. Biochemical and metabolic parameters were evaluated at predetermined intervals.

Analytical methods

Fasting blood glucose, serum lipid profile, insulin, TNF-α, liver and skeletal muscle glycogen, and insulin resistance (HOMA-IR) were assessed using standard biochemical and immunoassay techniques.

Statistical analysis

Data were expressed as mean ± SEM. Statistical significance was determined using analysis of variance (ANOVA), with $p < 0.05$ considered statistically significant.

Results

Glycemic control

Fructose feeding resulted in significant hyperglycemia, whereas FIIc administration markedly reduced fasting blood glucose levels.

Lipidemic control

Serum triglyceride levels were significantly elevated in fructose-fed rats and were effectively normalized following FIIc treatment.

Insulin and TNF-α

Fructose feeding led to significant increases in serum insulin and TNF-α levels. Treatment with FIIc significantly attenuated these elevations and improved insulin sensitivity.

Liver and skeletal muscle glycogen

A significant reduction in tissue glycogen content was observed in fructose-fed rats. FIIc treatment restored liver and skeletal muscle glycogen levels toward normal values.

Discussion

Chronic fructose consumption induces insulin resistance characterized by hyperglycemia, dyslipidemia, and hyperinsulinemia. The present findings demonstrate that FIIc effectively ameliorates these metabolic disturbances, likely through enhancement of insulin sensitivity and suppression of fructose-induced lipogenesis. Restoration of tissue glycogen content further supports improved peripheral insulin action.

Conclusion

FIIc significantly prevents fructose-induced hyperglycemia, hyperinsulinemia, hypertriglyceridemia, and insulin resistance. Its beneficial effects on inflammatory markers and tissue glycogen levels highlight its potential as a preventive therapeutic agent against insulin resistance and related metabolic disorders.

References

1. Bray George A, Samara J Nielsen, Barry M (2004) Consumption of High-Fructose Corn Syrup in Beverages May Play a Role in the Epidemic of Obesity. American Journal of Clinical Nutrition 79(4): 537-543.

2. Softic Samir, Kimber L, Julien B, Michael J (2020) Fructose and Hepatic Insulin Resistance. Critical Reviews in Clinical Laboratory Sciences 57(5): 308-322.

3. Thorburn AW, Lesley H, Storlien A, Sam K, Edward W (1989) Fructose-Induced in Vivo Insulin Resistance and Elevated Plasma Triglyceride Levels in Rats. American Journal of Clinical Nutrition 49(6): 1155-1163.

4. Reddy SS, R Karuna, R Baskar, D Saralakumari (1993) Prevention of Insulin Resistance by Ingesting Aqueous Extract of Ocimum sanctum in Fructose-Fed Rats. Hormone and Metabolic Research 25(1): 44-47.

5. Dills W (1993) Protein Fructosylation: Fructose and the Maillard Reaction. American Journal of Clinical Nutrition 58: 779S-787S.

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6. Elliott S, Nancy L, James S, Karen T, Peter J (2002) Fructose, Weight Gain, and the Insulin Resistance Syndrome. American Journal of Clinical Nutrition 76(5): 911-922.
7. Jeppesen J, YI Chen, Min Y, Peter S, Ann Coulston, et al. (1995) Postprandial Triglyceride and Retinyl Ester Responses to Oral Fat: Effects of Fructose. American Journal of Clinical Nutrition 61(4): 787-791.
8. Abraha A, SM Humphreys, ML Clark, DR Matthews, KN Frayn (1998) Acute Effect of Fructose on Postprandial Lipemia in Diabetic and Nondiabetic Subjects. British Journal of Nutrition 80(2): 169-175.
9. Arner P (2001) Free Fatty Acids—Do They Play a Central Role in Type 2 Diabetes? Diabetes, Obesity and Metabolism 3(1): 11-19.
10. Zavaroni I, Yung D, Gerald M (1982) Studies of the Mechanism of Fructose-Induced Hypertriglyceridemia in the Rat. Metabolism 31(11): 1077-1183.
11. Sleder J, Yung D, Michael J, Gerald M (1981) Hyperinsulinemia in Fructose-Induced Hypertriglyceridemia in the Rat. Metabolism 30(3): 303-305.
12. Matthews D, Jonathan P, Andrew S, Bruce A, Donald F, Rodney C (1985) Homeostasis Model Assessment: Insulin Resistance and β-Cell Function from Fasting Plasma Glucose and Insulin Concentrations in Man. Diabetologia 28(7): 412-419.
13. Reaven Gerald M (1988) Role of Insulin Resistance in Human Disease. Diabetes 37(12): 1595- 1607.
14. DeFronzo R Ele F (1991) Insulin Resistance: A Multifaceted Syndrome Responsible for NIDDM, Obesity, Hypertension, Dyslipidemia, and Atherosclerotic Cardiovascular Disease." Diabetes Care 14(3): 173-194.
15. Katsurada A, Nobuhiro I, Hiroshi F, Y Matsumura, N Nishimoto, et al. (1990) Effects of Nutrients and Hormones on Transcriptional and Post-Transcriptional Regulation of Acetyl-CoA Carboxylase in Rat Liver. European Journal of Biochemistry 190(2): 435-441.
16. Tanwar, Reenu S, SB Sharma, KM Prabhu (2016) *In Vivo* Assessment of Antidiabetic and Antioxidative Potential of a Natural Phytochemical Isolated from Fruit Pulp of Eugenia jambolana. Redox Report 21(1): 1-7.
17. Tanwar R, SB Sharma, UR Singh, KM Prabhu (2010) Attenuation of Renal Dysfunction by an Antihyperglycemic Compound Isolated from Eugenia jambolana in Streptozotocin-Induced Diabetic Rats. Indian Journal of Biochemistry & Biophysics 47(2): 83-89.
18. Tanwar R, SB Sharma, UR Singh, KM Prabhu (2011) Anti-Atherosclerotic Potential of an Active Principle Isolated from Eugenia jambolana in Streptozotocin-Induced Diabetic Rats. Evidence-Based Complementary and Alternative Medicine.
19. Grover JK, Shilpa Y, Varsha V (2002) Medicinal Plants of India with Antidiabetic Potential. Journal of Ethnopharmacology 81(1): 81-100.
20. Vikrant V, JK Grover, N Tandon, SS Rathi, N Gupta (2001) Treatment with Extracts of Momordica charantia and Eugenia jambolana Prevents Hyperglycemia and Hyperinsulinemia in Fructose-Fed Rats. Journal of Ethnopharmacology 76(2): 139-143.

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