INNSPUB

International Journal of Biosciences | IJB |
ISSN: 2220-6655 (Print); 2222-5234 (Online)
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Vol. 29, Issue: 5, p. 66-73, 2026

RESEARCH PAPER OPEN ACCESS

Protective effects of laminarin on diabetes-associated alterations in glycoprotein components of plasma, liver and kidney in HFD/STZ-induced diabetic male wistar rats

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Key words: Hyperglycemia, Laminarin, Glycoproteins, High-fat diet, Streptozotocin

Received: October 21, 2026 **Accepted:** October 31, 2026 **Published:** November 09, 2026

DOI: https://dx.doi.org/10.12692/ijb/29.5.66-73

ABSTRACT

Diabetes mellitus is a metabolic disorder characterized by persistent hyperglycemia, which contributes to widespread metabolic disturbances and the development of diabetes-associated tissue complications. Among these metabolic alterations, changes in glycoprotein components have gained attention because glycoproteins play important structural and functional roles in cellular recognition, adhesion and tissue homeostasis. In this context, laminarin, a β-glucan derived from brown algae, has demonstrated promising pharmacological properties; however, its effects on diabetes-associated alterations in glycoprotein components remain insufficiently explored. Therefore, the present study was undertaken to investigate the effects of laminarin on glycoprotein components in the plasma, liver and kidney of high-fat diet (HFD)/streptozotocin (STZ)-induced type 2 diabetic rats. To achieve this, diabetes was induced by feeding rats an HFD for four weeks, followed by a single intraperitoneal administration of STZ (35 mg/kg body weight). The diabetic control rats subsequently exhibited significant alterations in glycoprotein components, with increased levels of hexose, hexosamine, fucose and sialic acid in plasma. In contrast, liver and kidney tissues showed increased levels of hexose, hexosamine and fucose, accompanied by decreased sialic acid levels. Oral administration of laminarin (200 mg/kg body weight) for 28 days significantly ameliorated the altered levels of these components in plasma and tissues and was comparable to that of standard metformin. Furthermore, laminarin administration to non-diabetic rats did not produce marked changes in the measured components compared with the normal control group. Overall, these findings suggest that laminarin may help restore glycoprotein homeostasis in HFD/STZ-induced diabetic rats. In light of these beneficial findings, further studies are warranted to explore the potential use of laminarin in clinical trials for alleviating diabetes-associated complications.

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INTRODUCTION

The prevalence of diabetes mellitus (DM) is a significant and growing health problem worldwide. According to the International Diabetes Federation (IDF) approximately one in nine adults aged 20–79 years are currently living with diabetes, with more than four in ten individuals remaining unaware of their condition. This burden is projected to increase further, with approximately 853 million adults expected to be living with diabetes by 2050 (Hossain *et al.*, 2024). Type 2 diabetes mellitus (T2DM) accounts for more than 90% of all diabetes cases, characterized by persistent hyperglycemia arising primarily from insulin resistance and progressive impairment of pancreatic β-cell function. Chronic hyperglycemia induces widespread metabolic disturbances that contribute to diabetes-associated tissue damage (Galicia-Garcia *et al.*, 2020). Among these alterations, disturbances in glycoprotein metabolism have received considerable attention, as glycoproteins are involved in several cellular and extracellular functions and their carbohydrate components may be altered under diabetic conditions (Naseri *et al.*, 2020).

Glycoproteins are important structural and functional components of cell membranes, extracellular matrices and the circulation, where their carbohydrate moieties regulate protein stability, cellular recognition, adhesion, signaling and other biological processes. Alterations in glycoprotein structure and glycosylation can therefore affect cellular communication, protein function and tissue homeostasis (Shivatare *et al.*, 2022). Among the major carbohydrate constituents of glycoproteins, hexoses serve as essential monosaccharide building blocks for glycan structures and contribute to the structural organization and biological properties of glycoconjugates. Hexosamines are important precursors in glycan biosynthesis and participate in the hexosamine biosynthetic pathway, which links cellular glucose availability with protein glycosylation and nutrient sensing (Ertik *et al.*, 2023). Fucose is an important monosaccharide constituent of N- and O-linked glycans and contributes to glycan-mediated molecular recognition and cell–cell or cell–matrix interactions. Sialic acid, which commonly occupies the terminal position of glycan chains, contributes to the surface charge, stability and biological recognition of glycoproteins and regulates interactions between cells and their extracellular environment (He *et al.*, 2024).

The high-fat diet (HFD) and streptozotocin (STZ)-induced model is widely used to investigate type 2 diabetes mellitus in experimental animals. HFD feeding promotes insulin resistance, whereas low-dose STZ induces partial pancreatic β-cell dysfunction, resulting in hyperglycemia and metabolic abnormalities resembling important features of T2DM (Skovsø, 2014). Metformin, a widely used antihyperglycemic drug, primarily reduces hepatic glucose production and is commonly employed as a reference treatment in experimental diabetes studies (Hunter *et al.*, 2018). Although metformin is an established treatment for T2DM, an increasing research attention is being directed towards bioactive compounds from natural sources as potential therapeutic candidates for managing T2DM and its associated metabolic disturbances, with the aim of identifying effective agents that may offer fewer adverse effects than currently available antidiabetic drugs. Brown algae represent an important source of marine bioactive compounds and contain a variety of biologically active substances, including polysaccharides, polyphenols, carotenoids and omega-3 fatty acids (Zhu *et al.*, 2024). Among these compounds, laminarin is a storage polysaccharide predominantly composed of glucose units linked through β-(1→3) glycosidic bonds, with some β-(1→6) linkages. Laminarin and its oligosaccharides have attracted considerable research interest because of their reported antioxidant, anti-inflammatory, immunomodulatory and other biological activities (Navenaa Sri and Mirunalini, 2025). Previous studies have also explored the potential of laminarin in metabolic health, including its possible role in regulating glucose metabolism and

diabetes-associated disturbances. Its biocompatibility and biodegradability have further increased interest in its potential applications in biomedical and nutraceutical research (Karuppusamy *et al.*, 2022). However, the effects of laminarin on diabetes-associated alterations in glycoprotein components remain insufficiently explored.

The aim of the present study was to experimentally validate the laminarin in the management of diabetes and diabetes-induced complications on glycoprotein components in plasma, liver and kidney of HFD/STZ-induced experimental rats which was comparable with standard metformin.

MATERIALS AND METHODS

Chemicals

Laminarin was obtained from TCI Chemicals Co., Ltd., India, while streptozotocin (STZ) and metformin were purchased from Sigma-Aldrich, India. All other chemicals, reagents, and solvents used throughout the study were of analytical grade and procured from Hi-Media Laboratories Pvt. Ltd. and S.D. Fine Chemicals, Mumbai, India.

Animals and ethical approval

Male albino Wistar rats, 7–8 weeks of age and weighing 180–220 g, were procured from Mass Biotech, Chennai, India. The animals were housed in the Central Animal House facility of Rajah Muthaiah Medical College (RMMC), Annamalai University, under controlled laboratory conditions. The housing temperature was maintained at 22–24 °C with a 12 h light/dark cycle. Standard pellet diet and drinking water were provided ad libitum throughout the experimental period. All animal procedures were conducted in accordance with the guidelines of the Institutional Animal Ethics Committee (IAEC). The experimental protocol was approved by the IAEC under Proposal No. IAEC/1395/12/24.

Induction and treatment protocol

Following a one-week acclimatization period, the animals were randomly assigned to five experimental groups comprising two non-diabetic groups (n= 6 each) and three diabetic groups (n = 10 each). Animals assigned to the normal control and laminarin-alone groups received a normal pellet diet (NPD) containing 13% fat, 63% carbohydrate, and 24% protein. The remaining animals were maintained on a high-fat diet (HFD) for four weeks. The HFD was freshly prepared in the laboratory each week that contained 35.2 g fat (beef tallow), 17.7 g protein, and 34.5 g carbohydrate.

At the end of the dietary induction period, HFD-fed animals were fasted overnight for 12 h and administered a single intraperitoneal injection of STZ (35 mg/kg body weight), freshly prepared in ice-cold 0.1 M citrate buffer (pH 4.5), for the induction of diabetes (Srinivasan *et al.*, 2005; Nambirajan *et al.*, 2018). Rats in the non-diabetic groups received an equivalent volume of sodium citrate buffer. Following STZ administration, the animals were provided with 20% glucose solution overnight to minimize the risk of STZ-induced hypoglycaemia. After 72 h, fasting blood glucose (FBG) was determined using tail-tip venous blood with a glucose analyzer (Accu-Chek Performa, Roche, Germany). Animals exhibiting FBG concentrations above 250 mg/dL were considered diabetic and included in the study. This study received approval from the Institutional Animal Ethics Committee with approval number IAEC/1395/12/24.

The experimental animals were divided into five distinct groups to evaluate the therapeutic effects of the treatments. Group I (n = 6) served as the control group, with rats maintained on a normal pellet diet (NPD). Group II (n = 10) consisted of diabetic model rats, which were fed a 40% high-fat diet (HFD) and induced via a single intraperitoneal injection of streptozotocin at a dose of 35 mg/kg body weight (b.w.). Group III (n = 10) comprised diabetic rats that were orally administered laminarin at a dose of 200 mg/kg b.w., while Group IV (n = 10) included diabetic rats treated with the standard drug metformin at a dose of 100 mg/kg b.w. Finally, Group V (n = 6) served as a treatment control group, where normal rats were fed an NPD and administered laminarin alone at a dose of 200 mg/kg b.w.

Laminarin and metformin were freshly dissolved in distilled water and administered orally once daily for 28 days. Body weight, food intake and water intake were recorded weekly throughout the experimental period. At the end of the experimental period, the animals were fasted overnight and anaesthetized with ketamine hydrochloride (24 mg/kg body weight; intramuscular administration). Blood samples were collected by cardiac puncture into EDTA-coated tubes. Following blood collection, the animals were sacrificed, and the tissues were carefully excised and processed for subsequent biochemical and histopathological analyses.

Biochemical analysis

For the analysis of glycoprotein components, tissue samples were first defatted and a known quantity of the resulting tissue was homogenized in 3.0 mL of 2N hydrochloric acid (HCl). The suspension was hydrolyzed by heating at 90 °C for 4 h. After cooling to room temperature, the hydrolysate was neutralized with 3.0 mL of 2N sodium hydroxide (NaOH). The resulting preparation was used for the estimation of hexose, hexosamine, fucose and sialic acid. Total hexose content in plasma and tissue samples was determined according to the method of Niebes (1972), while hexosamine was estimated following the procedure described by Wagner (1979). Fucose and sialic acid concentrations were determined according to the methods of Dische and Shettles (1948) and Warren (1959), respectively.

Statistical analysis

Results are presented as mean ± standard deviation (SD). All biochemical parameters were performed using SPSS software, version 27.0 (SPSS, Chicago, IL). The assumptions of homogeneity of variances across the experimental groups were assessed using Levene's test. Differences between the experimental groups were evaluated by one-way ANOVA. Welch's ANOVA and the Brown-Forsythe test were additionally performed as robust tests of equality of means, considering the unequal group sizes. Post hoc pairwise comparisons were performed using the Games-Howell test. A p-value < 0.05 was considered statistically significant.

RESULTS

Effects of laminarin on plasma glycoprotein components of experimental rats

The levels of plasma glycoprotein components in control and experimental rats are illustrated in Fig. 1. Diabetic control rats exhibited significantly elevated levels of hexose, hexosamine, fucose and sialic acid compared with normal control rats. Treatment with laminarin (200 mg/kg body weight) significantly reduced the levels of all four glycoprotein components compared with diabetic control rats, with changes comparable to those observed in the metformin-treated group. In non-diabetic rats, administration of laminarin alone did not produce any marked alteration in plasma glycoprotein components compared with normal control rats.

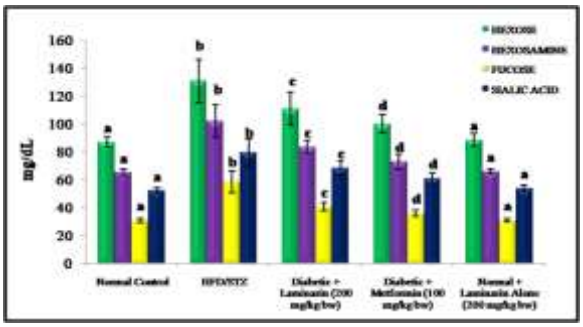


Fig. 1. Effect of laminarin on plasma glycoprotein components of control and experimental rats

Data are expressed as mean ± SD (n = 6 for Non-diabetic groups (I); n = 10 for diabetic groups (II–V)). Statistical analysis was performed using one-way ANOVA followed by Duncan's Multiple Range Test (DMRT). Superscript letters (a–d) indicate DMRT homogeneous subsets. Means sharing at least one common superscript letter are not significantly different, whereas means with no common superscript letters differ significantly at $p < 0.05$.

Effects of laminarin on glycoprotein components in the liver of experimental rats

The levels of glycoprotein components in the liver of control and experimental rats are presented in Fig. 2. Diabetic control rats exhibited significantly increased levels of hexose, hexosamine and fucose, whereas sialic acid levels were markedly reduced compared with normal control rats. Treatment with laminarin

(200 mg/kg body weight) significantly attenuated the elevated levels of hexose, hexosamine and fucose and restored hepatic sialic acid levels toward normal. Similar changes were observed in the metformin-treated group. Furthermore, the levels of hepatic glycoprotein components in non-diabetic rats treated with laminarin alone remained comparable to those of normal control rats.

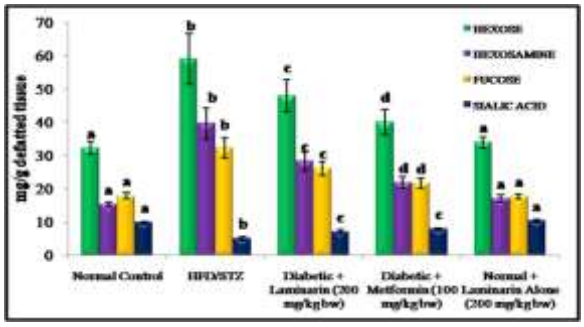


Fig. 2. Effect of laminarin on liver tissue glycoprotein components of control and experimental rats

Data are expressed as mean ± SD (n = 6 for Non-diabetic groups (I); n = 10 for diabetic groups (II–V)). Statistical analysis was performed using one-way ANOVA followed by Duncan's Multiple Range Test (DMRT). Superscript letters (a–d) indicate DMRT homogeneous subsets. Means sharing at least one common superscript letter are not significantly different, whereas means with no common superscript letters differ significantly at $p < 0.05$.

Effects of laminarin on glycoprotein components in the kidney of experimental rats

The levels of glycoprotein components in the kidney of control and experimental rats are presented in Fig. 3. Diabetic control rats exhibited significantly increased levels of hexose, hexosamine and fucose, accompanied by a significant decrease in sialic acid levels compared with normal control rats. Treatment with laminarin (200 mg/kg body weight) significantly reduced the elevated levels of hexose, hexosamine and fucose and increased sialic acid levels toward normal. Similar changes were observed in the metformin-treated group. In non-diabetic rats, laminarin administration alone did not markedly alter the levels of kidney glycoprotein components compared with normal control rats.

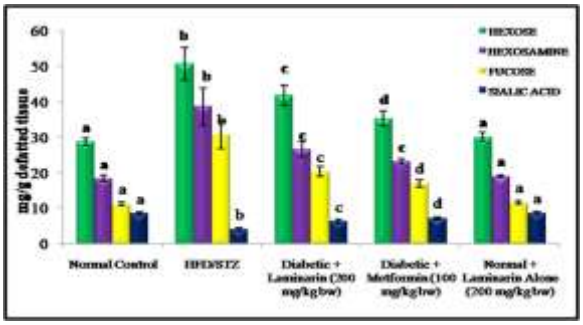


Fig. 3. Effect of laminarin on kidney tissue glycoprotein components of control and experimental rats

Data are expressed as mean ± SD (n = 6 for Non-diabetic groups (I); n = 10 for diabetic groups (II–V)). Statistical analysis was performed using one-way ANOVA followed by Duncan's Multiple Range Test (DMRT). Superscript letters (a–d) indicate DMRT homogeneous subsets. Means sharing at least one common superscript letter are not significantly different, whereas means with no common superscript letters differ significantly at $p < 0.05$.

DISCUSSION

Glycoproteins are essential structural and functional components of cellular membranes, extracellular matrices and circulating proteins. Their carbohydrate moieties contribute to protein stability, cell recognition, adhesion and intracellular signaling. Persistent hyperglycemia in type 2 diabetes mellitus (T2DM) can disturb carbohydrate metabolism and glycoprotein homeostasis, potentially affecting normal cellular and tissue functions (Sundaram and Muthu, 2024). In the present study, HFD/STZ-induced diabetic rats exhibited marked alterations in hexose, hexosamine, fucose and sialic acid levels in plasma, liver and kidney. Treatment with laminarin (200 mg/kg body weight) ameliorated these alterations, with changes generally approaching those observed in normal control rats.

The levels of protein-bound hexose and hexosamine were significantly elevated in the plasma, liver and kidney of diabetic rats. Hexose residues contribute to the structural organization of glycoproteins, whereas hexosamine residues serve as important substrates for glycan biosynthesis (Paneque *et al.*, 2023). Chronic hyperglycemia increases intracellular glucose availability and may alter the hexosamine biosynthetic pathway,

which generates UDP-N-acetylglucosamine for protein glycosylation. Dysregulation of this pathway and associated changes in protein glycosylation have been implicated in insulin resistance and diabetic tissue complications (Qi *et al.*, 2025). The increased hexose and hexosamine levels observed in the present study may therefore reflect disturbed carbohydrate utilization and glycoprotein metabolism during diabetes. Laminarin treatment significantly reduced these elevated levels in plasma and both tissues, suggesting an improvement in diabetes-associated metabolic disturbances. Similar alterations in glycoprotein components and their amelioration following treatment with chrysin have been reported in HFD/STZ diabetic rats (Sundaram and Muthu, 2024). The response observed with laminarin was also comparable in direction to that of metformin, a standard antihyperglycemic agent.

Fucose is a constituent of N-linked and O-linked glycans and contributes to glycoprotein-mediated cellular recognition, adhesion and signaling (Schneider *et al.*, 2017). In the present investigation, diabetic rats showed significantly increased fucose levels in plasma, liver and kidney, indicating an alteration in glycoprotein carbohydrate composition. This change may be associated with altered glycoprotein synthesis, turnover or fucosylation under diabetic conditions. However, fucosyltransferase and fucosidase activities were not measured in this study, the precise mechanism responsible for the increased fucose levels remains to be established. Laminarin administration significantly attenuated the diabetes-associated elevation of fucose in plasma and tissues, suggesting a beneficial effect on the altered glycoprotein profile.

These findings are consistent with the study conducted by Veeramani *et al.* (2012) in experimental diabetes, in which elevated plasma and tissue fucose levels were ameliorated by treatment with *Cardiospermum halicacabum* leaf extract.

Sialic acid is commonly present at the terminal position of glycan chains and contributes to the surface charge, stability and biological recognition of glycoproteins (Soares *et al.*, 2021). Unlike hexose, hexosamine and fucose, its distribution differed between plasma and tissues in the present study. Diabetic rats exhibited increased plasma sialic acid levels, accompanied by decreased concentrations in liver and kidney tissues. This opposing pattern suggests that diabetes alters the distribution of sialylated glycoproteins between the circulation and tissues. Increased circulating sialic acid may reflect changes in glycoprotein levels or the release of sialic-acid-containing molecules, whereas the reduced tissue concentrations may be associated with altered synthesis, glycosylation or degradation (Sujithra *et al.*, 2019). Comparable alterations in plasma and tissue sialic acid have been reported in experimental diabetic models demonstrated by Ramalingam *et al.* (2020). Notably, laminarin treatment reduced plasma sialic acid toward the control level while restoring liver and kidney sialic acid concentrations. This response suggests that laminarin may help normalize diabetes-associated disturbances in sialic acid metabolism and tissue glycoprotein composition.

The effects of laminarin on the measured glycoprotein components were generally comparable to those observed with metformin. Furthermore, the levels of these components in non-diabetic rats receiving laminarin alone remained close to those of the normal control group, suggesting that laminarin did not substantially disturb the measured glycoprotein profile under the experimental conditions. Taken together, the results of the present study demonstrate that laminarin significantly ameliorated the diabetes-induced alterations in glycoprotein carbohydrate components in plasma, liver and kidney. These beneficial effects may be associated with improved glucose homeostasis and the regulation of glycoprotein metabolism under diabetic conditions.

CONCLUSION

In conclusion, the findings of the present study suggest that laminarin may have beneficial effects on diabetes-associated alterations in glycoprotein metabolism in HFD/STZ-induced diabetic rats, as evidenced by the normalization of hexose,

hexosamine, fucose and sialic acid levels in the plasma, liver and kidney. These effects were generally comparable to those observed with metformin without ant toxic effects suggesting that laminarin may help ameliorate disturbances in glycoprotein homeostasis associated with diabetes mellitus. Moreover, these findings provide a basis for further investigation into the potential use of laminarin as an adjuvant therapeutic agent for managing diabetes mellitus and its associated complications.

ACKNOWLEDGEMENTS

The authors acknowledge Department of Biochemistry and Biotechnology, Annamalai University for providing the necessary animal house, laboratory facilities and technical support to conduct this study.

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