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Research Article

LAMINARIN-A MARINE POLYSACCHARIDE ATTENUATES RENAL AND HEPATIC ALTERATIONS IN HFD/STZ-INDUCED DIABETIC RATS

Navenaa Sri Gowri Pandian and Mirunalini Sankaran

Department of Biochemistry and Biotechnology, Annamalai University,
Annamalai Nagar - 608002, Tamil Nadu, India.

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ABSTRACT

Diabetes mellitus is a widespread metabolic disorder characterized by persistent hyperglycemia, which is a major contributing factor to the development and progression of diabetes-related complications. Laminarin, a β-glucan obtained from brown algae, has demonstrated promising pharmacological properties; however, its protective effect against diabetes-associated hepatic and renal biochemical alterations remains insufficiently explored. The present study was undertaken to investigate whether Laminarin can ameliorate the altered hepatic and renal biochemical parameters in high-fat diet (HFD)/streptozotocin (STZ)-induced type 2 diabetic rats. HFD feeding for four weeks followed by a single intraperitoneal administration of STZ (35 mg/kg body weight) produced significant increases in serum urea, uric acid and creatinine levels, together with elevated activities of AST, ALT and ALP in diabetic control rats. Oral administration of Laminarin at 200 mg/kg body weight for 28 days significantly attenuated the diabetes-induced elevation of these renal and hepatic markers. Metformin (100 mg/kg body weight) treatment also reduced the altered biochemical parameters. Laminarin administration to non-diabetic rats did not produce significant alterations in the assessed parameters compared with normal control rats. we conclude that laminarin ameliorated the activities of hepatic and renal markers in HFD/STZ induced diabetic rats observed from this study and these findings indicate the potential of laminarin for the management of diabetes mellitus.

Keywords: Hepatic enzymes, Insulin resistance, Renal markers, Wistar rats, Pharmacological.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by persistent hyperglycaemia resulting from impaired insulin action and progressive dysfunction of pancreatic β-cells (Goyal *et al*., 2023). Although disturbances in glucose homeostasis represent the principal feature of T2DM, prolonged metabolic dysregulation affects multiple physiological systems and contributes to the development of progressive tissue and organ complications (Fuentes-Barría *et al*., 2026). The sustained elevation of blood glucose is associated with metabolic, cellular and vascular alterations that can compromise the functional integrity of organs involved in glucose metabolism, detoxification and excretion (Hussain, 2025). Consequently, the assessment of diabetes-associated changes beyond glycemic control is important for understanding the progression of systemic complications and for evaluating the efficacy of potential therapeutic interventions (Mahmood *et al*., 2009). Among the organs affected by diabetes, the liver has a central position owing to its involvement in glucose, lipid and protein metabolism. Hepatic insulin resistance and persistent hyperglycaemia disturb normal metabolic processes and may promote hepatocellular stress and functional impairment (Mohamed *et al*., 2016). Diabetes-associated alterations in hepatic metabolism can affect the integrity of hepatocytes and cellular membranes, resulting in the release of intracellular enzymes into the circulation. Serum aspartate aminotransferase (AST), alanine aminotransferase (ALT) and alkaline phosphatase (ALP) are therefore widely used biochemical indicators for evaluating hepatic alterations. Increased activities of these enzymes have been reported in experimental diabetic models and may reflect loss of hepatocellular integrity and altered hepatic function (Thakur *et al*., 2024). The kidney represents another major

*Corresponding Author: Dr. S. Mirunalini, Associate Professor, Department of Biochemistry and Biotechnology, Annamalai University, Annamalai Nagar-608002, Tamil Nadu, India. Email: mirunasankar@gmail.com.

target of diabetes-associated metabolic injury because of its continuous role in filtration and elimination of metabolic waste products (Tuttle *et al*., 2022). Persistent hyperglycaemia can adversely affect renal function through alterations in glomerular and tubular integrity, eventually reducing the efficient clearance of circulating metabolites (Lv *et al*., 2025). Such functional changes can be reflected by alterations in serum urea, uric acid and creatinine, which is commonly, employed biochemical indices for evaluating renal function. Experimental studies have reported increased concentrations of these parameters in diabetic animals, indicating impaired renal handling of metabolic waste products. The simultaneous assessment of hepatic enzyme activities and renal biochemical markers therefore provides a useful approach for characterizing diabetes-associated alterations in these two metabolically important organs.

The high-fat diet (HFD)/streptozotocin (STZ) model is frequently employed for experimental induction of T2DM because it incorporates dietary metabolic stress with pancreatic β-cell dysfunction (Fryklund *et al*., 2026). HFD feeding promotes insulin resistance and associated metabolic abnormalities, whereas subsequent administration of STZ induces partial β-cell impairment and persistent hyperglycaemia. The resulting diabetic state is accompanied by alterations in biochemical parameters associated with hepatic and renal function (Zhang *et al*., 2008). The increasing prevalence of diabetes-associated complications has encouraged the investigation of naturally derived compounds with multifunctional biological activities (Chahrour *et al*., 2025). Marine algae represent a valuable source of structurally diverse polysaccharides with reported antioxidant, anti-inflammatory, metabolic and other pharmacological properties. Among these bioactive constituents, Laminarin, a water-soluble β-glucan predominantly obtained from brown algae, has attracted considerable interest because of its diverse biological activities. Laminarin is primarily composed of β-(1→3)-linked glucose residues with β-(1→6) branching, and studies have reported its antioxidant, anti-inflammatory, antidiabetic and other health-promoting properties (Karuppusamy *et al*., 2022; Navenaa sri and Mirunalini, 2025). The reported biological properties of Laminarin provide a rationale for investigating its potential against metabolic complications associated with T2DM. Its reported antidiabetic activity, together with antioxidant and anti-inflammatory properties, may be relevant to the tissue disturbances accompanying persistent diabetic conditions. Nevertheless, the effects of Laminarin on the biochemical indices reflecting both hepatic and renal functional alterations in an HFD/STZ-induced T2DM model remain insufficiently characterized. A combined assessment of hepatic enzymes and renal function markers may therefore provide a broader understanding of its protective potential against diabetes-associated organ dysfunction. Therefore, the present study was undertaken to evaluate the effect of Laminarin on hepatic and renal biochemical alterations in HFD/STZ-induced T2DM rats by assessing serum AST, ALT and ALP activities as indicators of hepatic function, together with serum urea, uric acid and creatinine as indicators of renal function.

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.

Experimental study design 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 (Vatandoust *et al*., 2018). This study received approval from the Institutional Animal Ethics Committee with approval number IAEC/1395/12/24. The experimental groups were assigned as follows: Group I (n = 6), normal control rats

receiving NPD; Group II (n = 10), HFD/STZ-induced diabetic control rats; Group III (n = 10), diabetic rats treated with laminarin (200 mg/kg body weight); Group IV (n = 10), diabetic rats treated with metformin (100 mg/kg body weight); and Group V (n = 6), non-diabetic rats receiving laminarin alone (200 mg/kg body weight). 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

Estimation of renal marker enzymes

Serum urea (Martinek, 1969), uric acid (Fossatti *et al*., 1980) and creatinine (Jaffe, 1886) concentrations were determined using commercially available diagnostic kits (Merck Diagnostic Ltd., India) according to the respective spectrophotometric procedures. The concentrations of all three parameters were expressed as mg/dL of serum.

Estimation of hepatic marker enzymes

AST and ALT activities were determined according to the method of King (1965), while ALP activity was estimated following the method of King (1965). The enzyme activities were expressed as U/L of serum.

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 AND DISCUSSION

Figure 1 illustrates the effect of Laminarin on serum urea, uric acid and creatinine levels in control and experimental rats. The diabetic control group (Group II) showed a significant increase in serum urea, uric acid and creatinine levels compared with the normal control group (Group I). Treatment with Laminarin (200 mg/kg body weight) significantly decreased the elevated levels of all three renal markers compared with the diabetic control group. Metformin (100 mg/kg body weight) treatment also significantly reduced the levels of these markers compared with the diabetic control group. The Laminarin-alone group (Group V) showed no significant difference in serum urea, uric acid and creatinine levels compared with the normal control group. Figure 2 illustrates the activities of ALP, ALT and AST in control and experimental rats. The diabetic control group (Group II) showed a significant increase in the activities of all three hepatic marker enzymes compared with the normal control group (Group I). Treatment with laminarin at 200 mg/kg body weight (Group III) significantly reduced the elevated activities of ALP, ALT and AST compared with the diabetic control group. Metformin treatment (Group IV) also significantly decreased the activities of these enzymes compared with Group II. The laminarin-alone group (Group V) showed enzyme activities comparable to those of the normal control group. Assessment of renal markers and hepatic enzyme markers prior the administration of laminarin revealed the organ damage occurred due to high-fat diet and streptozotocin induction, which potentiated diabetes-induced organ damage.

Under normal conditions, urea, uric acid and creatinine are metabolic waste products eliminated mainly by the kidneys. Urea arises from protein metabolism, uric acid from purine metabolism and creatinine from muscle creatine metabolism (AlSuhaymi, 2024). In diabetes, persistent hyperglycaemia, oxidative stress and inflammation can cause glomerular and tubular injury, thereby reducing renal clearance of these waste products. Consequently, their serum levels increases, indicating impaired renal function associated with diabetic kidney injury (Ajiboye *et al*., 2026). In our study, the diabetic group exhibited significant surge in the levels of serum urea, uric acid and creatinine upon high-fat diet and streptozotocin administration. However, laminarin (200 mg/kg) and metformin (100 mg/kg) treatment restored the above said parameters to near normalcy. These findings evidently show that laminarin standardize the disturbed renal markers in diabetic rats by exerting its significant antihyperglycemic effects (Al-Malki and El Rabey, 2015). In the normophysiology conditions, AST and ALT participate primarily in amino acid metabolism and the transfer of amino groups, while ALP is a membrane-associated enzyme involved in phosphate metabolism and is particularly associated with the canalicular and biliary membranes of the liver (Jogu *et al*., 2026).

Under diabetic conditions, persistent hyperglycaemia and insulin resistance disturb hepatic glucose and lipid metabolism, increasing fatty-acid influx and hepatic lipid accumulation. These metabolic abnormalities promote mitochondrial dysfunction, excessive generation of reactive oxygen species and inflammatory responses, resulting in oxidative damage to hepatocytes and disruption of cellular membranes. Consequently, intracellular AST and ALT leak into the circulation, leading to increased serum activities (Rehman *et al*., 2023). Diabetes-associated hepatic and biliary alterations may also increase circulating ALP activity.

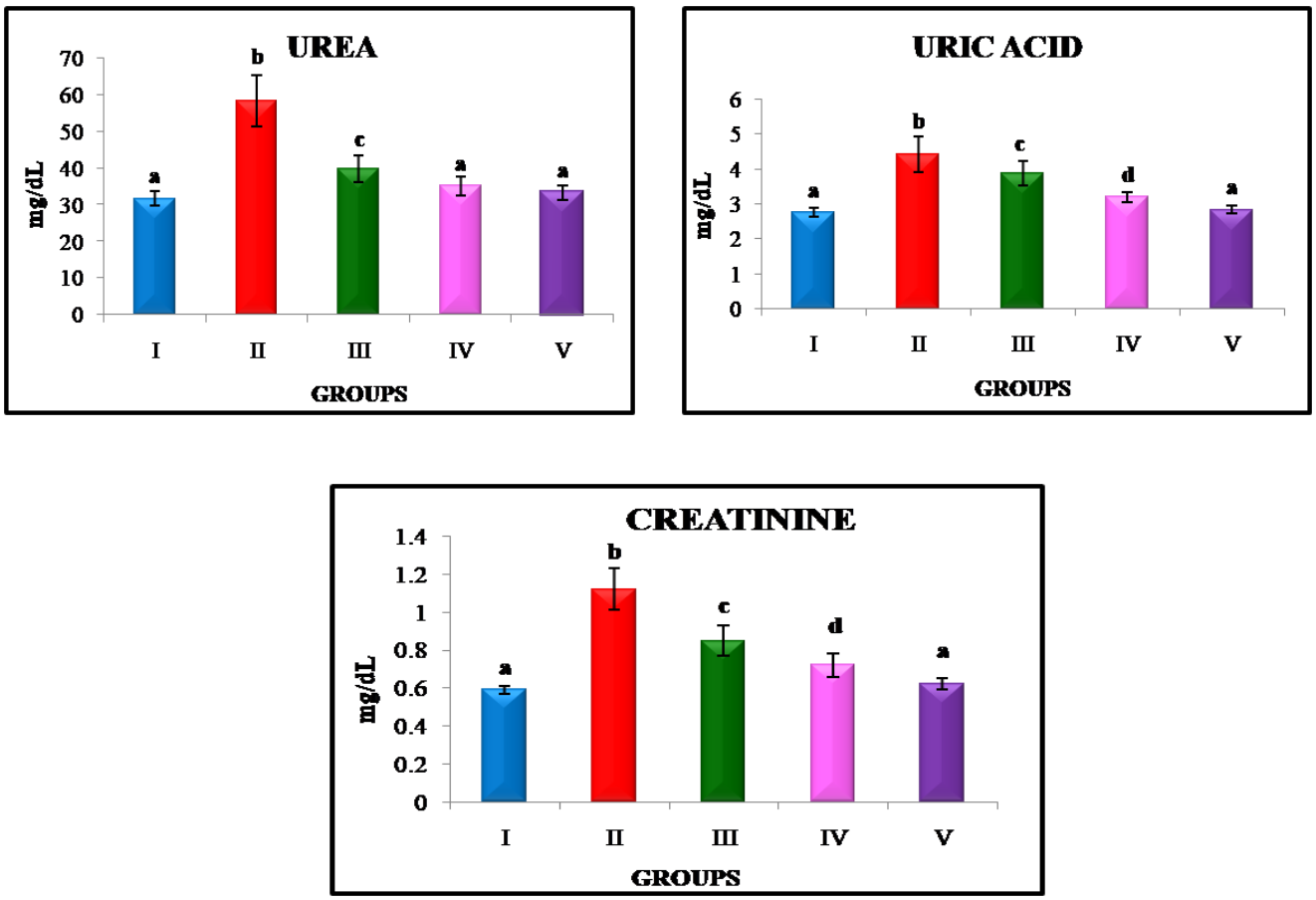


Figure 1. Effects of laminarin on serum urea, uric acid and creatinine of control and experimental rats.

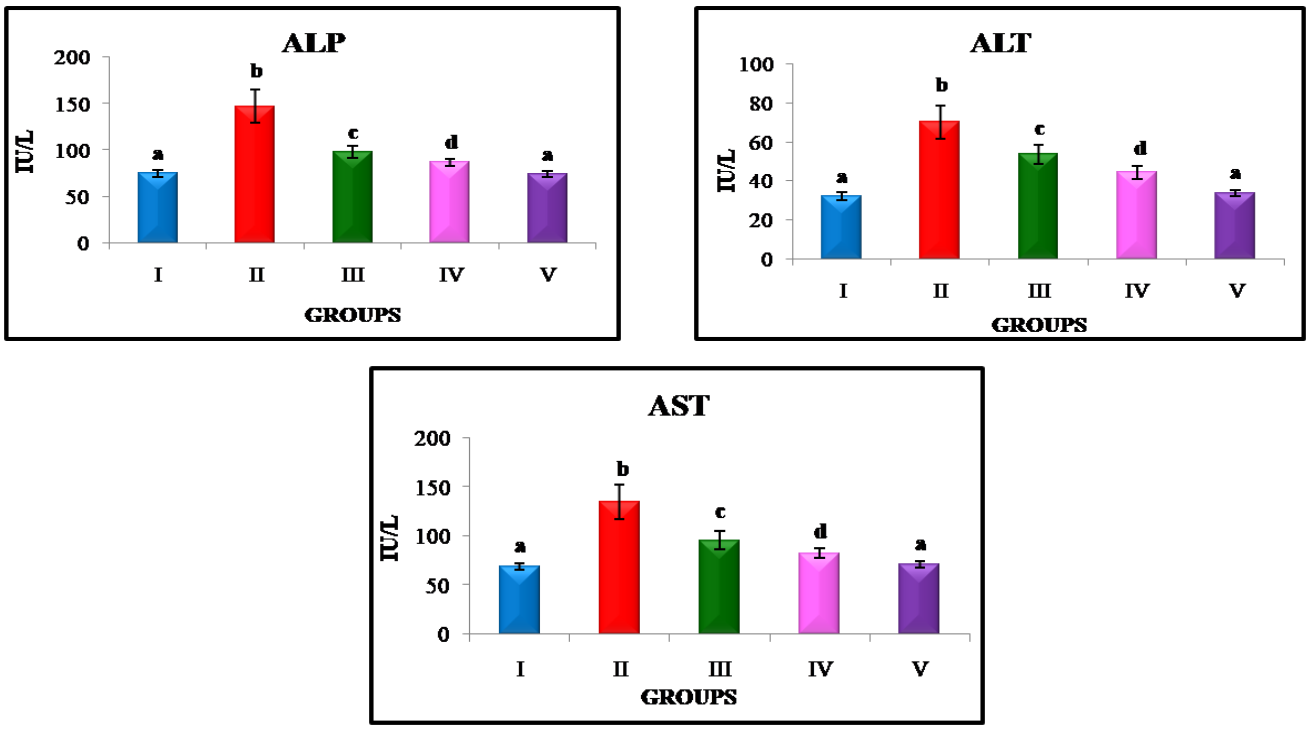


Figure 2. Effects of laminarin on hepatic enzymes of control and experimental rats.

Thus, the elevation of serum AST, ALT and ALP in diabetic animals can be considered indicative of diabetes-associated hepatic dysfunction arising from metabolic stress, oxidative damage and inflammation (Ghanbari *et al*., 2016). In our study, a significant elevation in AST, ALT and ALP was observed in diabetic rats. Upon oral administration of laminarin (200 mg/kg) and metformin (100 mg/kg) to diabetes treated groups has significantly restored the levels to near normal compared to normal control rats. In agreement with these findings, Abu *et al*. (2023) reported significantly elevated serum AST, ALT and ALP activities in STZ-induced diabetic rats, whereas treatment with *Cucumis sativus* fruit ethanol extract and metformin markedly reduced these alterations and restored the enzyme activities towards normal levels (Abu *et al*., 2023).

CONCLUSION

The upshot of the present study demonstrates that oral treatment of laminarin significantly attenuated the alterations in renal and hepatic biochemical markers in HFD/STZ-induced diabetic rats, restoring their levels towards normal. These findings suggest that laminarin may have potential as a marine-derived bioactive agent for mitigating diabetes-associated renal and hepatic disturbances and to develop as an antihyperglycemic agent for the management of diabetes mellitus. Further studies are in progress to elicit the exact mechanisms responsible for the protective effects of Laminarin against diabetes-induced organ damage.

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CONFLICT OF INTERESTS

The authors declare no conflict of interest

ETHICS APPROVAL

Not applicable

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AI TOOL DECLARATION

The authors declare that no AI and related tools are used to write the scientific content of this manuscript.

DATA AVAILABILITY

Data will be available on request

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