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Research Article

Comparison of Taurine and Metformin on Paraoxonase Enzyme Activity and Lipid Profiles in Streptozotocin-Induced Diabetic Rats

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Abstract

Diabetes remains a pressing global health challenge, demanding innovative biochemical interventions for effective management. This study evaluates the effects of taurine and metformin on glucose regulation, lipid metabolism, and antioxidant enzyme activity in diabetic rats. Diabetes was induced in mice using a single intraperitoneal dose of streptozotocin (60 mg/kg), and animals with blood glucose levels exceeding 300 mg/dL were considered diabetic. The experimental subjects were systematically classified into four groups: healthy controls, diabetic controls, metformin-treated diabetics, and taurine-treated diabetics. Metformin and taurine were each administered via oral gavage at a dosage of 500 mg/kg/day for a period of one month, beginning seven days post-diabetes induction. Key indicators assessed included changes in body weight, fasting glucose levels, serum lipid components, and paraoxonase enzymatic activity. Metabolic evaluations included fasting blood glucose, body weight, lipid profile parameters, and paraoxonase enzyme activity. Metformin treatment did not significantly alter weight relative to the diabetic group, whereas taurine led to a notable improvement. Hyperglycemia was markedly reduced in both treatment groups compared to diabetic controls. Furthermore, analysis of lipid components revealed significant normalization: triglyceride, cholesterol, and LDL levels decreased, while HDL concentrations increased with both interventions. Antioxidant assessment showed restored paraoxonase activity in taurine- and metformin-treated animals.

These outcomes support the hypothesis that taurine and metformin contribute to the restoration of biochemical equilibrium in diabetic conditions. Both Taurine and metformin can modulate Diabetes-related metabolic disturbances, aiding in physiological stabilization. Both agents promoted glycemic correction, lipid improvement, and enzymatic recovery. Their efficacy suggests promise in supportive metabolic care for diabetes.

Keywords: Taurine; Metformin; Paraoxonase; diabetic; FBS; HDL; Glucose levels.

1. Introduction

Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycemia resulting from defects in insulin secretion, insulin action, or both. Diabetes mellitus is frequently accompanied by a constellation of chronic complications—including cardiovascular dysfunction, renal impairment, neuropathic degeneration, and retinal pathology—that

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collectively erode patient well-being [1, 2]. Among its biochemical hallmarks, lipid metabolism perturbation stands out, often manifesting as dyslipidemia characterized by imbalanced lipoprotein profiles and disrupted lipid transport mechanisms. Dyslipidemia in diabetic patients is characterized by elevated levels of total cholesterol, triglycerides, and low-density lipoprotein (LDL), alongside reduced levels of high-density lipoprotein (HDL) [3].

Paraoxonase (PON1) is an enzyme associated with high-density lipoproteins (HDL) that plays a crucial role in preventing lipid oxidation. Paraoxonase 1 (PON1) facilitates the enzymatic breakdown of structurally diverse xenobiotic compounds—including organophosphates, lactones, and aromatic esters—thereby contributing to the preservation of lipoprotein integrity and cellular membrane stability under oxidative stress conditions. [4]. Studies have shown that PON1 activity is significantly reduced in diabetic patients, contributing to increased oxidative stress and atherogenesis. Upregulating paraoxonase 1 (PON1) enzymatic activity may serve as a targeted therapeutic strategy to counteract oxidative stress and rebalance lipid metabolism in diabetic conditions, owing to its critical role in hydrolyzing pro-oxidant substrates and fortifying lipoprotein structures against oxidative degradation. [5].

Taurine is a sulfur-containing amino acid with multiple physiological roles, including bile salt formation, osmoregulation, membrane stabilization, and antioxidation. Its potential benefits in diabetes management have been attributed to its insulin-like effects, antioxidative properties, and regulatory effects on lipid metabolism[6]. Metformin, on the other hand, is a first-line pharmacological treatment for type 2 diabetes mellitus. Metformin exerts its therapeutic effects predominantly by increasing insulin sensitivity, attenuating hepatic gluconeogenesis, and facilitating glucose uptake in peripheral tissues. Beyond glycemic regulation, it has also been associated with improvements in lipid metabolism and the mitigation of oxidative stress through its antioxidative mechanisms [7].

The primary aim of this study is to evaluate the effects of taurine and metformin on PON1 enzyme activity and lipid profiles in streptozotocin (STZ)-induced diabetic rats. This study hypothesizes that diabetes induces significant changes in PON1 activity and lipid concentrations, including cholesterol, triglycerides, LDL, and HDL. We further hypothesize that administering taurine and metformin will modulate PON1 activity and improve lipid profiles in diabetic rats.

To investigate these hypotheses, taurine and metformin will be administered to streptozotocin-induced diabetic rats, followed by assessment of paraoxonase 1 (PON1) enzymatic activity and serum lipid parameters, including total cholesterol, triglycerides, LDL, and HDL levels. This experimental approach aims to delineate the modulatory effects of these agents on oxidative stress and lipid metabolism, thereby providing mechanistic insights into their potential therapeutic utility in diabetes management.

2. Materials and Methods

2.1. Animal Selection and Housing Conditions

This study utilized forty-eight (48) healthy adult male Sprague-Dawley rats, randomly selected for experimental use. All procedures were approved by the institutional ethics committee and adhered to the guidelines of the National Institutes of Health (NIH) for the care and use of laboratory animals (NIH Publication No. 85-23, revised 1996) [8].

The animals were housed in individually ventilated transparent polycarbonate cages equipped with stainless steel mesh lids. Bedding consisted of sterilized wood shavings, changed twice per week to maintain hygienic conditions. A controlled environment was maintained with a 14-hour light/10-hour dark cycle, ambient temperature of $22 \pm 2°C$, and relative humidity of $55 \pm 2\%$. Rats had ad libitum access to a standardized rodent diet (Beparvar, Tehran, Iran) and drinking water sourced from municipal tap lines, delivered via autoclave-compatible drinking bottles.

Routine monitoring of cage conditions and animal health was carried out throughout the study to ensure optimal welfare.

2.2. Grouping of Experimental Animals

The forty-eight rats were randomly assigned to four groups ($n = 12$ per group) as follows:

- ✓ **Healthy Control Group**: Received standard laboratory diet and drinking water without any pharmacological intervention.

- ✓ **Diabetic Control Group**: Diabetes was induced by a single intraperitoneal injection of streptozotocin (STZ, 60 mg/kg) [9]. Animals subsequently received an oral gavage of physiological saline, equivalent in volume to that of the treatment groups.
- ✓ **Experimental Group 1 (Metformin-treated Diabetic Rats)**: Following STZ-induced diabetes (60 mg/kg, i.p.), rats were treated with metformin (500 mg/kg/day) via oral gavage for one month.
- ✓ **Experimental Group 2 (Taurine-treated Diabetic Rats)**: After STZ induction (60 mg/kg, i.p.), rats were administered taurine (500 mg/kg/day) by oral gavage for one month.

Treatment began seven days after STZ injection to allow for the establishment of hyperglycemia. At the conclusion of the experimental period, body weights were recorded using a calibrated digital scale (Model SF400).

2.3. Induction of Diabetes in Experimental Animals

Diabetes was induced in the rats using streptozotocin. The required amounts of streptozotocin powder were weighed and dissolved in a sodium citrate buffer solution (pH 4.5). A single dose of streptozotocin (60 mg/kg body weight) was administered intraperitoneally to each animal. One week after the streptozotocin injection, blood glucose levels were measured using a glucometer (Accu-Chek Active, Roche, Germany). Rats with blood glucose levels above 300 mg/dL were considered diabetic and included in the study [9].

2.4. Anesthesia and Blood Sample Collection

Before initiating blood sampling, rats were sedated via intraperitoneal administration of ketamine (80 mg/kg) and xylazine (12 mg/kg) to achieve sufficient anesthetic depth and reduce handling-induced stress. Blood samples were obtained via the lateral tail vein using sterile 23-gauge needles, with the site disinfected using 70% ethanol prior to puncture. For each collection, approximately 1 mL of blood was drawn into heparinized microcentrifuge tubes to prevent coagulation. Samples were immediately centrifuged at 3000 rpm for 10 minutes at 4°C to separate plasma, which was then aliquoted and stored at −80°C until biochemical analyses. All procedures were conducted in accordance with institutional ethical guidelines to ensure animal welfare and sample integrity.

2.5. Measurement of Serum Glucose Levels using the Photometric Method

Serum glucose levels in animals were measured using a kit from Pars Azmoon and the Perestige24i autoanalyzer. Initially, the device was calibrated with a commercial calibrator sample. The glucose levels were then measured using the Pars Azmoon glucose kit through an enzymatic colorimetric method. The test is based on the principle that glucose, in the presence of the glucose oxidase enzyme, produces hydrogen peroxide, which reacts with phenol and 4-aminoantipyrine in the presence of the peroxidase enzyme to form a quinoneimine. The amount of quinoneimine formed is measured photometrically at 546 nm, correlating directly with the glucose concentration.

2.6. Measurement of Lipid Profile

To determine the lipid profile (cholesterol, triglycerides, HDL, and LDL), the method described by (S Aborhyem et al.2016)was employed [10]:

2.7. Measurement of Serum Triglycerides

Serum triglyceride concentrations were measured using a commercially available enzymatic colorimetric kit (Pars Azmoon, Tehran, Iran) on the Perestige24i autoanalyzer. Prior to analysis, the instrument was calibrated using a standardized reference sample. The assay operates through a series of enzymatic reactions: triglycerides are initially hydrolyzed by lipoprotein lipase to release glycerol, which is then phosphorylated by glycerol kinase and oxidized by glycerol phosphate oxidase. The resulting hydrogen peroxide interacts with 4-aminoantipyrine and chlorophenol in the presence of peroxidase, yielding a quinoneimine chromophore. The intensity of this colored compound, measured spectrophotometrically at 546 nm, directly correlates with the triglyceride concentration in the specimen.

2.8. Measurement of Serum Cholesterol in Rats

Serum cholesterol levels in rats were determined using the Pars Azmoon diagnostic kit, which utilizes the

enzymatic-colorimetric CHOD-PAP method. This technique differentiates between esterified and free cholesterol, with the former enzymatically hydrolyzed by cholesterol esterase to yield free cholesterol. The liberated cholesterol is then oxidized by cholesterol oxidase, producing hydrogen peroxide. In the presence of peroxidase, this hydrogen peroxide reacts with phenol and 4-aminoantipyrine to form a red quinoneimine dye. The absorbance of the chromogenic product, quantified spectrophotometrically at 546 nm, serves as a direct indicator of the cholesterol concentration present in the analyzed serum sample.

2.9. Measurement of Low-Density Lipoprotein Cholesterol (LDL-C) in Rat Serum

The reference method for measuring LDL cholesterol uses ultracentrifugation, which requires specialized equipment and is time-consuming. Therefore, it is not commonly used in routine laboratories. Instead, the Pishtaz Teb diagnostic kit was employed to measure LDL cholesterol levels (Rafaei, 2022) [11].

Each lipoprotein has distinct physical and chemical properties and reacts differently with detergents. This method leverages these differences by combining two detergents. In the first reaction, Detergent 1 alters the structure of all lipoproteins except LDL, including chylomicrons, HDL, and VLDL. In the presence of Detergent 1, cholesterol oxidase and cholesterol esterase act on these lipoproteins, excluding LDL. In the second reaction, Detergent 2 accelerates the reaction and enhances the color development from the remaining LDL from the first reaction.

The resulting product has a maximum absorbance at 600 nm, which can be measured photometrically and is directly proportional to LDL cholesterol concentration. Additionally, LDL cholesterol can be calculated using the following formula, provided that triglyceride (TG) levels do not exceed 400 mg/dL.

2.10. Measurement of High-Density Lipoprotein Cholesterol (HDL-C) in Rat Serum

Serum HDL cholesterol levels were measured using the Pishtaz Teb diagnostic kit based on an enzymatic-colorimetric reaction. The principle of this method involves accelerating the reaction of cholesterol oxidase with unesterified HDL cholesterol and solubilizing HDL with a specific detergent. In the first step, high-affinity substances prevent other lipoproteins (VLDL, LDL, and chylomicrons) from participating in the enzymatic reaction. In the second step, a surfactant selectively accelerates the reactions between enzymatic reagents and HDL cholesterol.

The resulting product exhibits maximum absorbance at 600 nm, which can be measured photometrically and is directly proportional to the HDL cholesterol concentration.

2.11. Statistical Analysis Methods

The raw data obtained from the experiments were analyzed using SPSS software version 23. The normality of data distribution was assessed using the one-sample Kolmogorov-Smirnov test. Given the normal distribution, a one-way analysis of variance (ANOVA) followed by Tukey's post hoc test was employed for data analysis. Results were expressed as mean ± standard deviation (SD), with significance set at $p < 0.05$. Histograms were generated using GraphPad software version 7.8.

3. Result and discussion

3.1. Body Weight Variations and Treatment Effects in Experimental Groups

As shown in **Figure 1**, ANOVA and Tukey's post hoc test revealed a significant reduction in mean body weight in the diabetic group compared with healthy controls ($p < 0.001$). A similar reduction was observed in the metformin-treated diabetic group when compared to the control group ($p = 0.019$). However, no significant difference was found between the metformin-treated and untreated diabetic groups ($p > 0.05$), indicating that metformin treatment did not substantially reverse diabetes-induced weight loss. In contrast, the diabetic group receiving taurine showed a statistically significant increase in mean body weight compared to untreated diabetic mice ($p = 0.038$), suggesting a potential restorative influence. No other comparisons produced significant differences ($p > 0.05$).

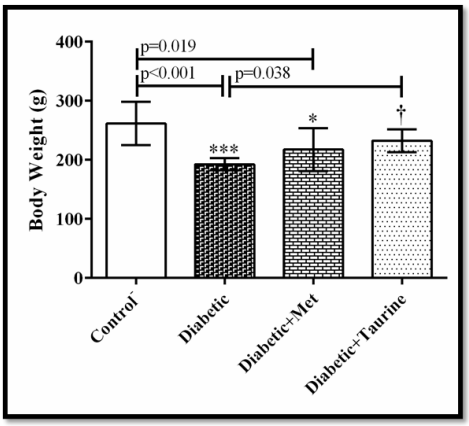

Figure 1. Results about the mean body weight in the study groups. Data are presented as Mean ± SD. The symbol * indicates a difference compared to the control group, and the symbol † indicates a difference compared to the diabetic group.

These results illustrate the physiological disruption triggered by streptozotocin-induced diabetes, which often leads to weight loss due to compromised glucose utilization and the degradation of essential protein stores. The weight reduction observed in both the diabetic and metformin-treated groups supports this interpretation. Although metformin is known to reduce blood glucose levels by limiting hepatic glucose production and improving peripheral glucose uptake, its impact on body weight remains limited under the conditions of this study. The absence of a significant difference in body weight between metformin-treated and untreated diabetic animals suggests that metformin's metabolic action primarily affects glucose regulation rather than body mass [2, 12, 13].

In contrast, taurine treatment led to a significant increase in body weight in diabetic mice. This effect appears to stem from taurine's role in maintaining energy balance, promoting anabolic activity, and facilitating more efficient glucose utilization. By helping regulate metabolic stability, taurine may reduce tissue catabolism and mitigate the weight loss commonly associated with diabetic conditions. These outcomes highlight taurine's capacity to safeguard both nutritional balance and metabolic stability during physiological disruption caused by diabetes[14]. Despite its efficacy in lowering blood glucose, metformin did not significantly alter the mean body weight in treated diabetic mice compared with untreated diabetic mice. This may be due to its multifaceted effects on metabolic processes that do not necessarily translate into immediate weight changes [15, 16].

Previous studies have shown that metformin can reduce weight by increasing energy expenditure and altering lipid metabolism [17]. Furthermore, metformin has been shown to affect the gut microbiome, which plays a significant role in metabolic health and weight regulation [18]. Studies also indicate that metformin's influence on the central nervous system can alter appetite regulation, contributing to its effects on body weight [19, 20].

3.2. Fasting Blood Sugar (FBS) Levels and Their Modulation Across Study Groups

As illustrated in **Figure 2**, the diabetic group exhibited a significant elevation in mean fasting blood sugar (FBS) levels compared to the healthy control group ($p < 0.001$). Similarly, FBS levels remained elevated in the diabetic groups treated with metformin and taurine relative to controls ($p < 0.001$). However, treatment with metformin and taurine led to a notable reduction in FBS levels compared to the untreated diabetic group ($p < 0.001$), with no statistically significant difference between the two treatment modalities ($p > 0.05$).

These observations reflect the pathophysiological effects of streptozotocin-induced beta-cell damage, leading to impaired insulin secretion and subsequent hyperglycemia. The elevation of FBS in diabetic groups affirms the disruption of pancreatic function, while the glucose-lowering response to both treatments suggests therapeutic efficacy in restoring glycemic control.

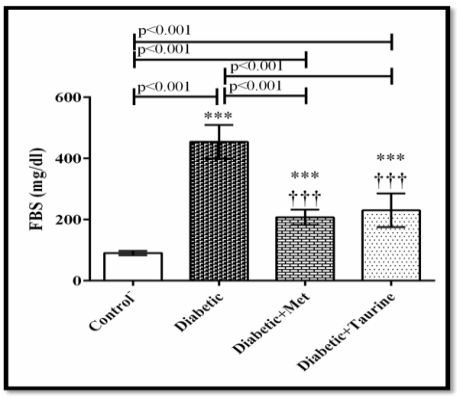

Figure 2. Results on mean fasting blood sugar (FBS) levels across the study groups: Data are presented as Mean ± SD. The symbol * indicates a difference compared to the control group, and the symbol † indicates a difference compared to the diabetic group.

Metformin's ability to lower fasting glucose levels is primarily due to its multifaceted actions on hepatic glucose

production and peripheral glucose uptake. The engagement of AMP-activated protein kinase (AMPK) plays a pivotal role in these mechanisms by suppressing hepatic glucose synthesis and enhancing glucose uptake in skeletal muscle cells. This mechanism aligns with established evidence underscoring metformin's role as a first-line agent in the management of type 2 diabetes [21-23].

In parallel, taurine also demonstrated a hypoglycemic effect, likely mediated through its antioxidant activity, preservation of beta-cell integrity, and insulin-mimetic properties. Taurine's capacity to regulate oxidative homeostasis and enhance cellular stability during hyperglycemia underscores its therapeutic potential for managing blood glucose dynamics. Experimental data suggest that taurine can enhance pancreatic function and improve metabolic stability, offering a complementary approach to conventional diabetic therapies.

The findings from this study emphasize the effectiveness of both metformin and taurine in reducing FBS levels and mitigating hyperglycemia. Taurine helps maintain oxidative equilibrium and fortify cellular defense mechanisms in conditions of elevated blood glucose, underscoring its role in glycemic regulation. Additionally, taurine exhibited significant hypoglycemic effects by protecting pancreatic beta cells and mimicking insulin activity [24, 25]. The current study supports the notion that taurine supplementation may improve insulin regulation and glycemic control in diabetic conditions. Evidence from experimental models suggests that taurine enhances pancreatic function and stabilizes blood glucose levels, potentially through its role in maintaining redox balance and cellular resilience under hyperglycemic stress. These outcomes reinforce taurine's potential as a metabolic modulator in diabetic conditions, highlighting its capacity to counteract key biochemical disturbances associated with the disease [26].

3.3. Lipid Profile Disruption and the Therapeutic Influence of Metformin and Taurine in Diabetic Models

Quantitative analysis revealed significant disturbances in lipid parameters among diabetic subjects when compared to non-diabetic controls, as shown in **Figure 3. A–C**. Diabetic mice displayed elevated levels of triglycerides (TG), total cholesterol, and low-density lipoprotein (LDL), while high-density lipoprotein (HDL) concentrations were substantially reduced ($p < 0.001$ for all). These findings reflect the shift in lipid metabolism induced by insulin deficiency and reinforce the characteristic dyslipidemia associated with streptozotocin toxicity.

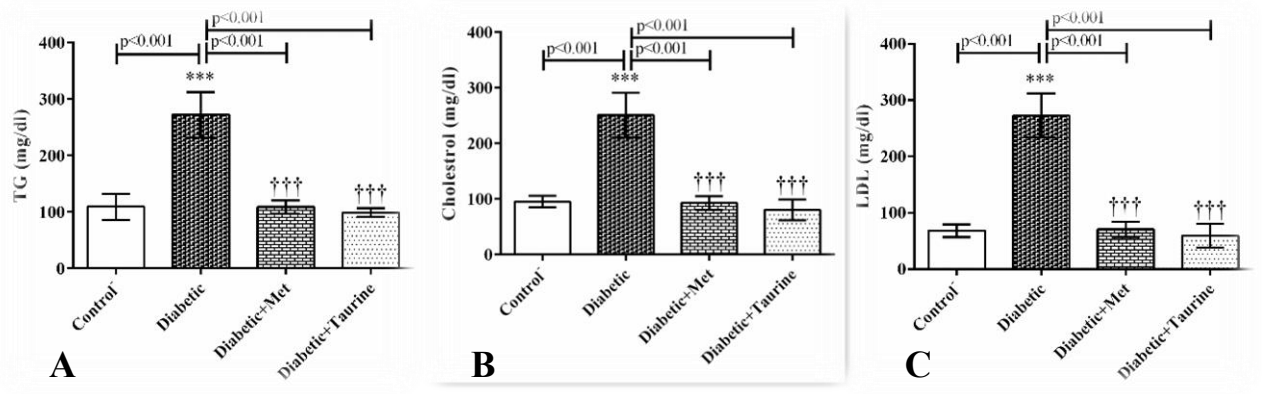

Figure 3. Lipid Profile Alterations Across Experimental Groups. The A panel presents triglyceride (TG) concentrations, showing significantly elevated levels in the diabetic group and notable reductions following metformin and taurine treatment. The B panel illustrates high-density lipoprotein (HDL) levels, showing reduced HDL in diabetic subjects and higher levels in treated groups. The C image displays total cholesterol changes, with significantly higher levels in the diabetic group and reductions observed with both therapeutic interventions. Data are expressed as Mean ± SD. Symbols * and *** represent significant differences versus the control group at $p < 0.05$ and $p < 0.001$, respectively. Symbols † and ††† denote differences compared to the diabetic group at $p < 0.05$ and $p < 0.001$, respectively.

In **Figure 3. A**, TG levels in diabetic animals were markedly increased, likely due to unrestricted mobilization of fatty acids and their hepatic conversion under insulin-deficient conditions. Both metformin and taurine administration resulted in a statistically significant reduction in TG levels compared with untreated diabetic mice ($p < 0.001$). However, the reductions were comparable between the two treatment groups ($p > 0.05$), suggesting similar capacities to curb hypertriglyceridemia.

Figure 3. B depicts total cholesterol fluctuations, with diabetic animals exhibiting significantly higher levels than healthy controls. This elevation is consistent with impaired cholesterol uptake and increased cholesterol biosynthesis in insulin-resistant states. Treatment with metformin and taurine significantly lowered cholesterol concentrations ($p < 0.001$), with no significant distinction between groups ($p > 0.05$), indicating their parallel effectiveness in normalizing this parameter.

Changes in **Figure 3. C** show a sharp rise in LDL concentrations and a reduction in HDL concentrations in diabetic subjects ($p < 0.001$ for both), reflecting an imbalance in hepatic lipid transport and reduced receptor activity. Both interventions significantly reduced LDL levels ($p < 0.001$) and improved HDL concentrations relative to the diabetic group ($p = 0.001$ for metformin; $p = 0.025$ for taurine). However, HDL values in taurine-treated animals remained statistically lower than those of healthy controls ($p = 0.025$), and the difference between treatment groups was not significant ($p > 0.05$).

These results validate the ability of both metformin and taurine to counteract lipid dysregulation induced by experimental diabetes. Metformin supports lipid regulation by initiating intracellular energy pathways that limit fat accumulation and adjust cholesterol production dynamics (27, 28). This activation initiates a cascade that inhibits fatty acid synthesis in hepatic tissues, accelerates lipid oxidation, and suppresses endogenous cholesterol production by inhibiting HMG-CoA reductase activity. It also enhances lipoprotein lipase activity, facilitating triglyceride clearance [27, 28].

Taurine, although mechanistically distinct, showed comparable outcomes. Taurine may exert its lipid-regulatory effects through a synergy of cellular antioxidant activity, enhancement of insulin-mediated signaling, and direct involvement in hepatic mechanisms that govern lipid transport and metabolism (31). Taurine is thought to reduce lipid accumulation by regulating bile acid metabolism and suppressing enzymes involved in cholesterol and fatty acid synthesis. The observed increases in HDL levels following taurine administration further underscore its role in promoting reverse cholesterol transport and mitigating cardiovascular risk [26, 29].

3.6. Paraoxonase Enzyme Activity and Its Modulation Across Study Groups

According to **Figure 1-3**, the findings indicate a significant decrease in the mean paraoxonase enzyme activity in the diabetic group compared to the healthy control group ($p < 0.001$). This reduction aligns with previous research attributing diminished paraoxonase activity in diabetic states to heightened oxidative stress and structural changes in HDL particles. Such alterations compromise HDL's protective role and may enhance susceptibility to cardiovascular complications associated with diabetes.

The diabetic groups receiving metformin and taurine demonstrated significant increases in paraoxonase enzyme activity compared with the untreated diabetic group ($p < 0.001$), suggesting a restorative effect of both agents. Notably, no statistically significant difference was observed between the metformin- and taurine-treated groups ($p < 0.05$), indicating comparable efficacy in enhancing paraoxonase function.

These therapeutic improvements may be linked to both treatments' capacity to counteract oxidative stress and to influence lipid metabolism positively. Metformin and taurine have been associated with the upregulation of antioxidant defenses, thereby supporting HDL-mediated inhibition of LDL oxidation. Enhanced paraoxonase activity contributes to HDL preservation. Preservation of HDL functionality may help mitigate cardiovascular risks associated with diabetes [30-32]. The biochemical differences observed in this study highlight metformin and taurine as among the best candidates for enhancing systemic antioxidant defense pathways. The elevation in paraoxonase activity reflects a noteworthy dimension of their pharmacological profile, underscoring their utility in counteracting oxidative imbalances frequently encountered in diabetic conditions [33-35].

4. Conclusion

This research uncovers the distinct roles of taurine and metformin in countering diabetes-related disruptions in biochemical balance. In diabetic rat models, treatment with these compounds resulted in significant reductions in blood glucose, total cholesterol, triglycerides, and LDL levels, alongside an elevation in HDL and paraoxonase enzyme activity. These shifts indicate that taurine and metformin can help counteract the metabolic disruptions caused by diabetes.

Looking ahead, an in-depth exploration of the cellular and molecular pathways affected by each agent is essential to decode their precise biological actions. It is also critical to conduct long-term assessments to determine safety, tolerability, and clinical relevance in human subjects. Additionally, investigating the combined administration of taurine and metformin may uncover synergistic effects that enhance their efficacy, potentially opening doors to more refined and powerful treatment strategies. These insights could ultimately aid in the design of more precise interventions for diabetes care.

Ethics approval

This study was performed in line with the principles of the Declaration of Helsinki. The Ethics Committee of Dezful University of Medical Sciences approved.

Informed consent was obtained from all individual participants included in the study. The ethics committee of Dezful University of Medical Sciences approved and supervised the conduct of this study in accordance with the IR ethics code "DUMS.REC.1397.0078".

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Conflict of interest

The authors declare that they have none.

Data availability

Data sharing does not apply to this article, as no datasets were generated or analyzed during the current study

Authors Contributions

All authors contributed to the study conception and design. Material preparation, data collection, and analysis were performed by all authors. All authors read and approved the final manuscript.

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Using artificial intelligence chatbots

There was no use of artificial intelligence in the making of this article.

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