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Effect of Boswellia gum and cinnamaldehyde on diabetes induced in male albino rats

Mustafa Elbadrawy[1*], Ghada Tabl[1], Eman E. El-Nahass[1], Menna Harby[1]

[1] Zoology Department, Faculty of Science, Tanta University, Tanta 31527, Egypt.

Running title: Antidiabetic effects of Boswellia and cinnamaldehyde

Corresponding author: Mustafa Tharwat Mustafa ElBadrawy
Zoology Department, Faculty of Science, Tanta University
E-mail: mustafaelbadrawy17@gmail.com Phone: 01004636268

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Abstract:

Diabetes mellitus (DM) is a Chronic metabolic state characterized by hyperglycemia originating from poor insulin production, insulin resistance, or a combination of both. Oxidative stress and inflammation significantly contribute to diabetes etiology and complications. The purpose of this study was to investigate the antidiabetic Boswellia gum and cinnamaldehyde in streptozotocin (STZ)-induced diabetic male albino rats. Both compounds are recognized for their anti-inflammatory effects, antioxidant, and glucose-lowering properties. Forty-two male albino rats with an average weight of 120-150 g were divided into 6 groups (n=7) and treated with Boswellia gum, cinnamaldehyde, or both, following diabetes induction by STZ. Biochemical parameters such as blood glucose, lipid profile, and liver and kidney function markers were assessed, in addition to histopathological examination of pancreatic tissue. The results demonstrated that both Boswellia gum and cinnamaldehyde significantly reduced blood glucose levels and improved lipid profile and liver enzymes, with enhanced effects observed in the combination group. Histological analysis supported the biochemical findings. These results suggest that both agents exhibit protective and therapeutic potential against diabetes-related metabolic alterations.

Keywords: *Boswellia serrata*, Cinnamaldehyde, Type 2 *Diabetes mellitus*, Albino Rats, Antioxidants, Metformin

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1. Introduction:

Diabetes mellitus (DM) is among the most widespread non-communicable and persistent illnesses globally, affecting individuals of all ages and socioeconomic backgrounds. Its prevalence is especially rising in low- and middle-income nations as a result of fast urbanization, inactive lifestyles, and changes in nutritional patterns. According to global estimates, the individuals diagnosed with diabetes is projected to increase dramatically in the forthcoming decades, from 463 million in 2019 to 700 million in 2045 [1]. In Egypt, the burden is especially severe, with Type 2 diabetes mellitus (T2DM) accounting for approximately 15.6% of adults aged 20–79 years, positioning the nation among the top 10 globally in terms of diabetes prevalence [2, 3]. T2DM is distinguished by persistent hyperglycemia caused by a combination of insulin resistance (IR) and relative insulin insufficiency. Unlike type 1 diabetes, which

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outcomes from the autoimmune obliteration of pancreatic beta cells, T2DM typically arises from a complex interplay of hereditary and environmental determinants [4, 5]. Obesity, particularly visceral obesity, is an important risk indicator for insulin resistance and type 2 diabetes mellitus (T2DM). This resistance diminishes cellular sensitivity to insulin, resulting in compromised glucose uptake, increased hepatic glucose production, and dyslipidemia [6, 7].

A central aspect of the T2DM pathogenesis involves oxidative stress, which comes from a disparity between reactive oxygen species (ROS) production and the body's antioxidant defense systems. Hyperglycemia promotes the formation of AGEs and excessive ROS, which trigger damage to cellular constituents, including lipids, proteins, and DNA [8, 9]. Mitochondrial impairment, stress in the endoplasmic reticulum, and persistent low-grade inflammation are intimately linked to oxidative damage and contribute to β-cell dysfunction and apoptosis [10, 11].

These mechanisms not only contribute to glucose dysregulation but also lead to the long-term complications in organs as the liver, kidneys, eyes, and nerves [12].

The liver, a central organ in glucose, lipid, and protein metabolism, is highly susceptible to diabetic injury. In T2DM, the liver may create a range of ailments from non-alcoholic fatty liver disease (NAFLD) to cirrhosis. hepatocellular carcinoma [13]. Diabetic nephropathy, characterized by glomerular sclerosis, basement membrane thickening, and Tubulointerstitial fibrosis, is a prominent cause of end-stage renal failure worldwide [11].

Retinopathy, neuropathy, and macrovascular complications, as cardiovascular disease and stroke, are also major contributors to diabetes-related morbidity and mortality [14].

Despite the availability of numerous pharmacological agents for diabetes management, including metformin, sulfonylureas, insulin, DPP-4 inhibitors, and alpha-glucosidase inhibitors, many patients fail to attain sufficient glycemic control, and the side effects of these therapies further limit their long-term [15,16]. This has driven interest toward alternative therapeutic strategies such as natural products and plant-based interventions, which offer multi-targeted actions with fewer adverse effects [17,18].

One of the promising natural agents under investigation is Boswellia serrata, frequently referred to as frankincense. It has been extensively used in traditional medicine because of its anti-inflammatory properties, antioxidant, and hepatoprotective characteristics [19, 20]. The bioactive components in Boswellia gum, particularly boswellic acids (such as AKBA), have been shown to inhibit pro-inflammatory agents, reduce oxidative stress, and regulate lipid and glucose metabolism [21]. In diabetic models, Boswellia has demonstrated improvements in insulin secretion, serum glucose, LDL/HDL ratio, and hepatic architecture [22]. Its potential extends to nephroprotection and wound healing in diabetic ulcers [10]. Another agent with significant antidiabetic potential is cinnamaldehyde (CA), the major active compound in cinnamon bark oil. CA exhibits a wide array of pharmacological activities, including antibacterial and anti-inflammatory properties, antioxidant, aldose reductase inhibition, and modulation of insulin sensitivity [23, 24]. Studies have demonstrated that CA suppresses oxidative stress, downregulates pro-inflammatory cytokines, and improves glucose uptake by activating insulin signaling mechanisms, including PI3K/Akt and AMPK [25, 26]. Moreover, CA exerts vascular protective effects by preserving endothelial integrity and reducing platelet aggregation and thrombosis risk [27].

Given the distinct yet complementary mechanisms of Boswellia gum and cinnamaldehyde, their combination holds promise as a synergistic strategy to combat hyperglycemia, oxidative damage, and diabetic complications. While both agents have individually shown efficacy in improving glucose metabolism and protecting organs from diabetic injury, few studies have explored their combined therapeutic potential in a single experimental model.

Therefore, the current research aimed to assess the individual and synergistic impacts of Boswellia gum and cinnamaldehyde on STZ-induced type 2 diabetes in male albino rats. The research focuses on assessing their impact on glucose levels, lipid profiles, hepatic and renal function biomarkers, oxidative stress parameters, and histopathological changes in pancreatic tissues. The goal is to determine whether co-administration of these agents offers superior protective and therapeutic benefits compared to monotherapy, thereby providing scientific support for their potential use in integrated diabetes management.

2. Materials and Methods:

Chemicals

All the chemicals and reagents used were obtained commercially and were of analytical grade. Streptozotocin (STZ) was purchased from Sigma-Aldrich. Glucose, alpha amylase, aspartate amino transferase (AST), alanine amino transferase (ALT), and super oxidedismutase (SOD), catalase (CAT) and malondialdehyde (MDA) kits were purchased from Bio diagnostic Company, Egypt. Phosphate buffer saline (PBS), ethanol and other chemicals were purchased from Al-Gomhoria Company, Tanta, Egypt. Metformin (Met) was purchased from local pharmacy in Tanta, Egypt, diluted by PBS and the concentration was adjusted to (150 mg/kg) b wt. in 300 µl for oral administration. Fructose was acquired in the form of white crystals (Safety Misr, Specialized Food Industry Co., Cairo, Egypt). Cinnamaldehyde was acquired as a pale-yellow liquid from Sigma-Aldrich (St. Louis, Missouri, USA), and *Boswellia serrate* was obtained as a white powder from Sigma-Aldrich.

Experimental Animals

Adult male albino rats (120–150 g, 4 weeks old) were obtained from the animal facility at Alexandria University. Rats were housed under standard conditions for 2 weeks for adaptation before starting the experiment. All animal experiments were conducted at the Department of Zoology, Faculty of Science, Tanta University.

Preparation of Boswellia Gum Extract

A simple water-based extraction method was used by dissolving Boswellia gum in water with gentle stirring and filtering the solution to remove undissolved particles.

Experimental Protocols

Diet Composition

Normal diet: 5% fat, 65% carbohydrates, 20.3% proteins, 5% fiber, 3.7% salt mixture, and 1% vitamin mix. High-fat diet (HFD): 46% fat, 24% carbohydrates, 20.3% proteins, 5% fiber, 3.7% salt, and 1% vitamin mix (custom diet composition). Similar approaches and HFD descriptions have been reported [28]. (Exp Diabetes Res 2008; and Research Diets technical datasheet).

Induction of T2DM

Rats had an overnight fast and received injections. intraperitoneally with a single STZ dose (30 mg/kg) [29]. To prevent hypoglycemia, 10% glucose solution was provided after 6 hours for the next 48 hours [30]. After 3 days, fasting blood glucose (FBG) was measured using a OneTouch glucometer. Rats with FBG > 200 mg/dL were considered diabetic.

Experimental Groups:

A total of 42 male Rats were allocated randomly to 6 groups (n = 7 per group):

Gp1 (Normal control): received normal diet, Gp2 (Diabetic): STZ-induced diabetic rats, Gp3 (Metformin): Diabetic rats administered Metformin

(150 mg/kg/day) for 8 weeks, Gp4 (Boswellia gum): Diabetic rats administered Boswellia extract (400 mg/kg/day) for 8 weeks, Gp5 (Cinnamaldehyde): Diabetic rats treated with CA (20 mg/kg/day) for 8 weeks GP6: Diabetic rats had administered both B.gum and CA as in Gp4 and Gp5

Sample Collection:

After 8 weeks of treatment, rats were fasted overnight and anesthetized with isoflurane (100%, Pharco, Alexandria, Egypt). Blood was collected via cardiac puncture into EDTA and plain tubes. Plasma and serum were separated and stored at −20°C for biochemical analysis.

Biochemical Analyses:

Fasting Blood Insulin:

Blood specimens were acquired from arterial blood vessels and heart chambers, and then sera were separated by centrifugation for biochemical analysis. Collected via retro-orbital sinus [31], centrifuged at 3000 rpm, 4°C for 10 min. Insulin quantified using ELISA kits [32].

Fasting Blood Glucose

Determined using Spectrum Diagnostics liquizyme reagent [33].

HOMA-IR Index

Calculated from fasting glucose and insulin levels using ELISA kits and enzymatic methods.

Lipid Profile

Total cholesterol [34] , triglycerides [35] , HDL-C, LDL-C, and VLDL-C measured enzymatically; atherogenic index calculated as (TC – HDL-C)/HDL-C.

Liver and Kidney Function Tests

ALT: [36] , AST: [37] , Urea and creatinine: [33] Antioxidant and Oxidative Stress Markers.

Tissue Homogenization

Liver and kidney tissues were homogenized in phosphate buffer solution (pH 7.4), centrifuged at 10,000 rpm, 4°C.

SOD Activity

Measured by the [38] technique, based on inhibition of NBT reduction.

CAT Activity

Ascertained per [39], using H_2O_2 as substrate.

GSH Levels

Estimated using Ellman's reagent (DTNB), Quantifying absorbance at 412 nm.

Histopathological investigation of liver, kidney, and pancreas tissues:

The liver, the kidney, and the pancreas tissue samples were taken and fixed in 10% formalin. After finishing tissue processing in various series of alcohol and xylene, paraffin blocks were prepared. 5 µm sections from paraffin blocks were cut and then stained with hematoxylin and eosin before being examined using a light microscope (Optika light microscope (B-350) to look for significant cellular damage (Bancroft and Gamble, 2008).

3. Results:

Hematological Parameters

RBCs, hemoglobin (Hb), and platelet counts were significantly reduced in diabetic rats compared to all treated and control groups ($P \leq 0.05$). Platelets were significantly elevated in the Cin cohort compared to the Bos cohort.

White Blood Cells and Differentials

WBCs count was significantly reduced in the diabetic cohort compared to the control and treated cohorts. In contrast, monocytes, lymphocytes, and neutrophils were significantly elevated in the diabetic group. Treatments helped restore these parameters towards normal levels.

Fasting Blood Glucose, Insulin, and HOMA-IR

Diabetic rats exhibited a significant elevation in fasting blood glucose (FBS), serum insulin levels, and HOMA-IR index compared to the control cohort ($p \leq 0.001$). The mean FBS in the diabetic cohort was 548.50 ± 55.86 mg/dL, insulin 11.30 ± 2.12

µIU/mL, and HOMA-IR 2.60 ± 0.28. Treatment with Metformin, Boswellia serrata, cinnamaldehyde, and their combination significantly reduced these levels. The combination cohort (Cin+Bos) had the most notable improvement, with FBS 106.00 ± 11.31, insulin 3.70 ± 0.14, and HOMA-IR 0.95 ± 0.07 ($p \leq 0.05$ vs diabetic cohort).

Renal Function Parameters

This demonstrates a significant increase in urea (74.43 ± 9.45 mg/dL) and creatinine (0.894 ± 0.10 mg/dL) levels in the diabetic cohort compared to controls ($p \leq 0.05$). These parameters were significantly improved in all treated groups, particularly in the Boswellia and combination groups. The lowest creatinine was seen in the combination group (0.563 ± 0.17), suggesting nephroprotective effects.

Liver Enzymes (ALT and AST)

Diabetic rats showed significantly elevated ALT (69.33 ± 7.23 U/L) and AST (193.5 ± 15.19 U/L) compared to control rats ($p \leq 0.05$). Treatment with Metformin, Boswellia, cinnamaldehyde, and their combination significantly reduced liver enzymes. Notably, ALT levels dropped to 43.7 ± 4.22 U/L and AST to 107.7 ± 11.12 U/L in the combination group.

Lipid Profile

Diabetic rats had significantly elevated total cholesterol (90.4 ± 9.89 mg/dL), triglycerides (161.1 ± 14.26 mg/dL), and LDL (64.5 ± 6.45 mg/dL), and decreased HDL (28.7 ± 3.11 mg/dL) compared to control rats. All treatment groups showed improvements, with the combination group showing the highest HDL (57.5 ± 5.89 mg/dL) and improved TC, TG, and LDL levels, indicating a synergistic hypolipidemic effect.

Antioxidant Status and Oxidative Stress Markers

Antioxidant markers such as SOD, CAT, GSH, and TAC were significantly reduced in the diabetic group. SOD dropped to 5.54 ± 1.23 U/mg, CAT to 50.87 ± 5.22 U/mg, GSH to 15.12 ± 2.11 ng/mL, and TAC to 0.15 ± 0.05 mmol/L. These markers improved markedly in treated groups, especially in the combination group (SOD 7.56, CAT 68.92, GSH 27, TAC 0.21), with significant differences compared to the diabetic group ($p \leq 0.05$).

Histopathological Observations

Administration of metformin, *B. serrata* & cinnamon, and *B. Serrata* & metformin improved Streptozotocin (STZ) diabetic liver histopathological changes

Liver sections of control rats showed a normal hepatic structure in which polygonal hepatocytes with prominent nuclei were arranged in a radial pattern. Many hepatocytes were binucleated. The hepatic strands alternated with narrow blood sinusoids lined by an endothelial cell layer containing Kupffer cells (Figure 9a). Diabetic rats showed marked hepatic disorganization, including variable degrees of bridging necrosis of hepatocytes, most prominent in centrilobular and mononuclear cellular infiltrates in the lobule, hepatocyte shrinkage, and hepatocyte fragmentation (apoptosis). Also, hepatocyte swelling, vacuolation, or fibrosis appeared. Congested central veins with hemorrhage, intensive degeneration of its epithelial lining, area around the central vein associated with cellular infiltration. Kupffer cells, pyknotic nuclei, and karyolytic ones were seen (Figure 9b). Cinnamon administered to diabetic rats showed a centrilobular pattern of hepatic degeneration with irregularly branched and congested central vein, widening of blood vessels, area of infiltration around the central vein. Binucleated, vacuolated hepatocytes that have pyknotic nuclei, vesicular nuclei, and megakaryocytic nuclei; others show eosinophilia, and kupffer cells also appeared (Figure 9c). *B. Serrata* administered to diabetic rats showed centrilobular regeneration, with the restoration of the central vein, vacuole, sinusoidal spaces, hepatocytes with mild necrosis, and mild infiltration of inflammatory cells associated with steatosis in the rat liver (Figure 9d). Liver sections of diabetic rats administered with metformin and *B. Serrata* &

cinnamon showed partial improvement of the hepatic architecture with minimal tissue degeneration, but there was cellular infiltration still appearing around the wide central vein (Figure 9e & f). After *B. Serrata and metformin* treatment, liver sections showed improvement of more normal hepatic architecture with normal-like hepatocytes, slight widening of the central vein, kupffer cells, and binucleated hepatocytes also appeared. Hepatocytes with pyknotic, vesicular nuclei and megakaryocytic ones were seen (Figure 9g).

Administration of metformin, *B. Serrata* & cinnamon, and *B. Serrata* & metformin improved Streptozotocin (STZ) diabetic kidney histopathological changes

The renal cortex of control rats appeared normal, with a normal glomerulus, proximal and distal convoluted tubules lined by cuboidal epithelium, and Bowman's capsule. There were no signs of inflammation (Figure 10a). The kidney sections of diabetic rats showed disorganized, congested, shrunken, and destroyed glomeruli with irregular Bowman's space. The majority of the renal tubules were widened, injured, with degenerated epithelial lining cells, resulting it losing their characteristic appearance and intertubular haemorrhage. It may also be occluded with hyaline casts, mild degeneration of the lining renal epithelial cells, and their contents intermixed with each other (Figure 10 b). Kidney sections of diabetic rats administered cinnamon showed destroyed, shrunken, and congested glomeruli with irregularly destroyed Bowman's capsule, wide Bowman's space. Destroyed renal tubules that lost their typical appearance with distinct vacuolated and highly degenerated lining epithelium; cellular infiltration at the intertubular spaces was observed, and the appearance of cellular hemorrhage at the intertubular spaces. Dilated, congested renal blood vessels, which are engorged with blood (Figure 10c). Kidney sections of diabetic rats administered with *B. Serrata* showed disorganized glomeruli with narrow Bowman's space, damaged renal tubules with undistinguished lining epithelium, which lost their distinctive structure (Figure 10d). After administration of *metformin*, partial improvement in glomeruli with glomerular shrinkage disappeared (Figure 10e). Kidney sections of diabetic rats that were administered with *B. Serrata and cinnamon* also showed partial improvement in glomeruli structure, but accompanied by glomerular shrinkage and intertubular hemorrhage still appear (Figure 10f). *B. Serrata and metformin* administered to diabetic rats showed a normal-like architecture of glomeruli with a normal Bowman's capsule, normal Bowman's space. Most of the renal tubules were improved with a normal appearance, and renal blood vessels were normal (Figure 10g).

Administration of metformin, *B. Serrata* & cinnamon, and *B. Serrata* & metformin improved Streptozotocin (STZ) diabetic pancreas histopathological changes

The control group's pancreas showed a normal cellular composition in the pancreatic islets of Langerhans with rich vascular supply, as well as normal histological findings in pancreatic acinar cells that were arranged in irregular cords with blood capillaries in between (Figure 10a). The pancreas of diabetic rats showed a shrinkage of the normal architecture of the pancreatic islets. The cytoplasm of the cells was vacuolated with pyknotic nuclei, many necrotic cells were seen, and many cells showed hydropic degeneration. Meanwhile, the diabetic rat pancreas had severe cell damage, necrosis, and inflammation, as well as islet shrinkage, degenerated acini cells, and Langerhans islets (Figure 10b). Following *B. Serrata* treatment, the pancreas showed no improvement in cellular architecture (Figure 10c). When diabetic rats were treated with cinnamon extracts, showed partial improvement appeared in cellular architecture as measured by regeneration with restoration of normal-like islet and pancreatic cell population size, a mild interstitial inflammatory cell infiltrate associated with acinar hyperplasia (Figure 10d). Pancreatic cells from Metformin-treated rats showed the most significant improvement, as these cells retain their normal structure and appearance (Figure 10e). With *B. Serrata* and cinnamon, and also with *B. Serrata* and metformin treatment, it showed an improvement of pancreatic tissue with normal architecture of the pancreatic islets. The cytoplasm becomes granulated, the vacuoles of β cells disappear, and nuclei become normal (Figure 10f & g).

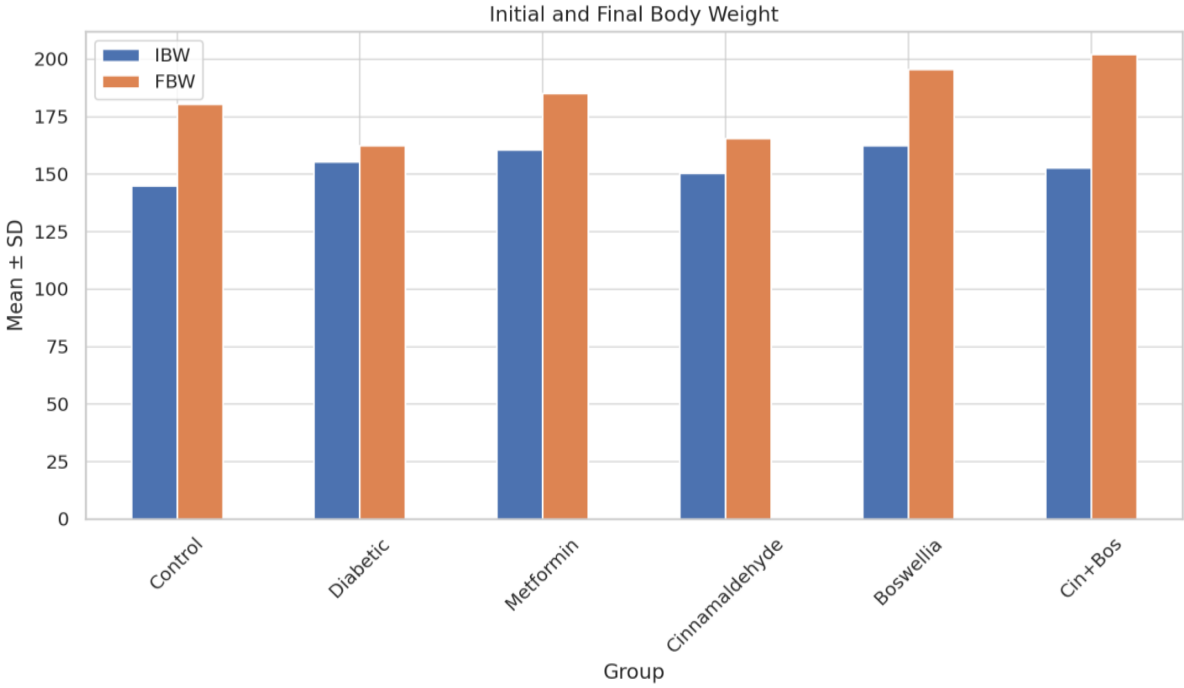

Figure 1. Effect of treatments on body weight changes in control and diabetic rats
Diabetic rats showed reduced weight gain compared to controls. All treatments improved body weight, with the combination (Boswellia + Cinnamaldehyde) producing the most significant increase, suggesting a protective effect against diabetes-induced weight loss.

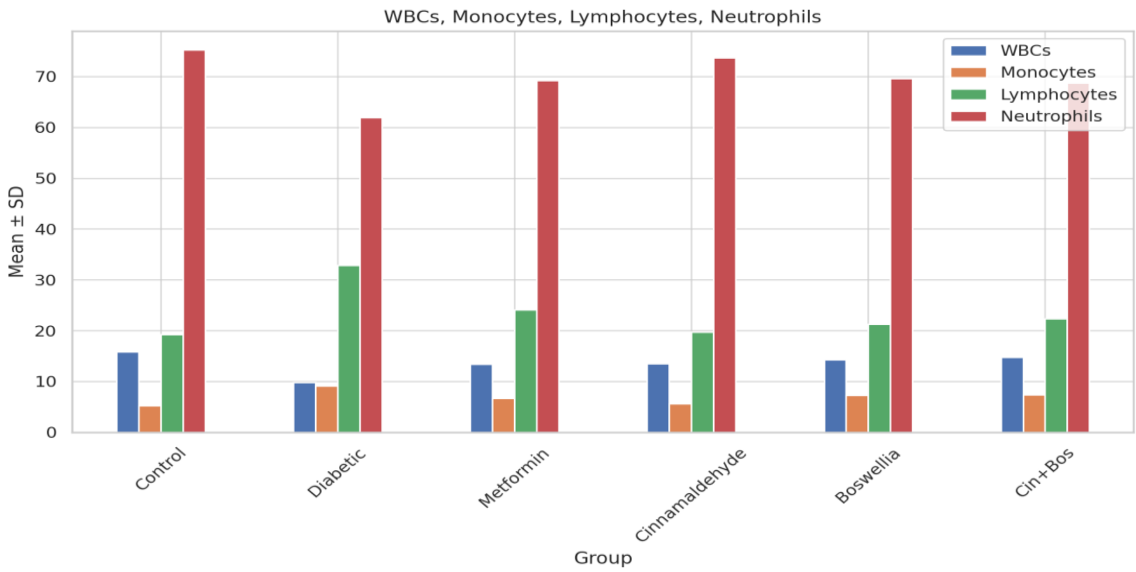

Figure 2. Effect of treatments on white blood cell counts and differentials
Diabetic rats showed leukopenia with elevated lymphocytes and monocytes. Treatments improved WBC counts and normalized differential distribution, with Boswellia and combination therapy showing the best recovery.

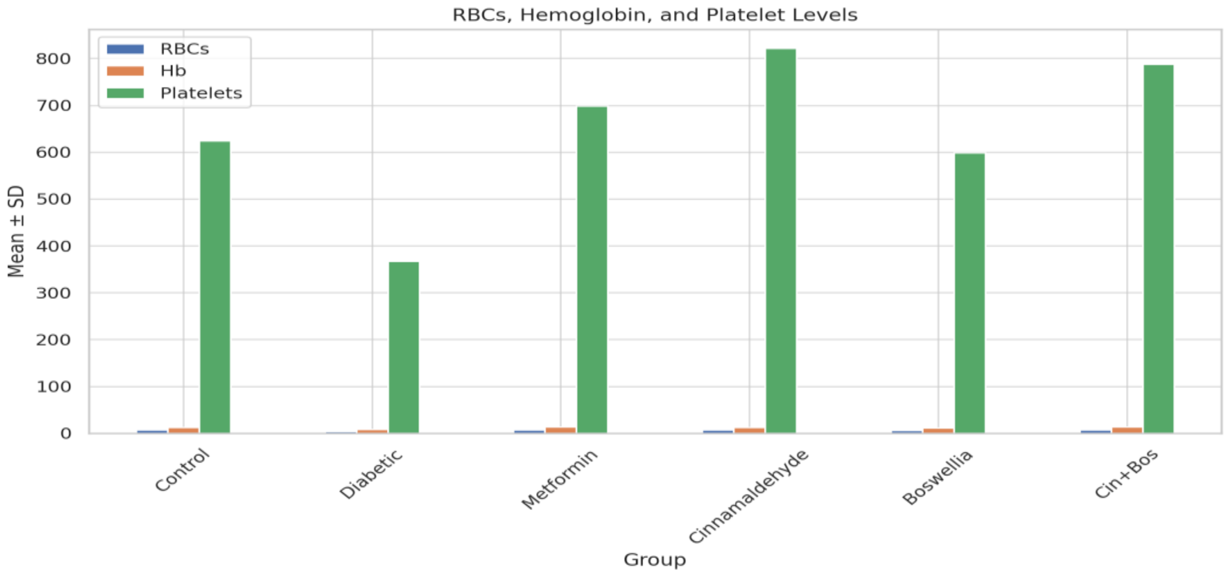

Figure 3. Effect of treatments on red blood cells, hemoglobin, and platelet count

Diabetes significantly reduced RBCs, Hb, and platelet counts. Treatments restored hematological parameters, especially in the combination group, indicating hematoprotective effects.

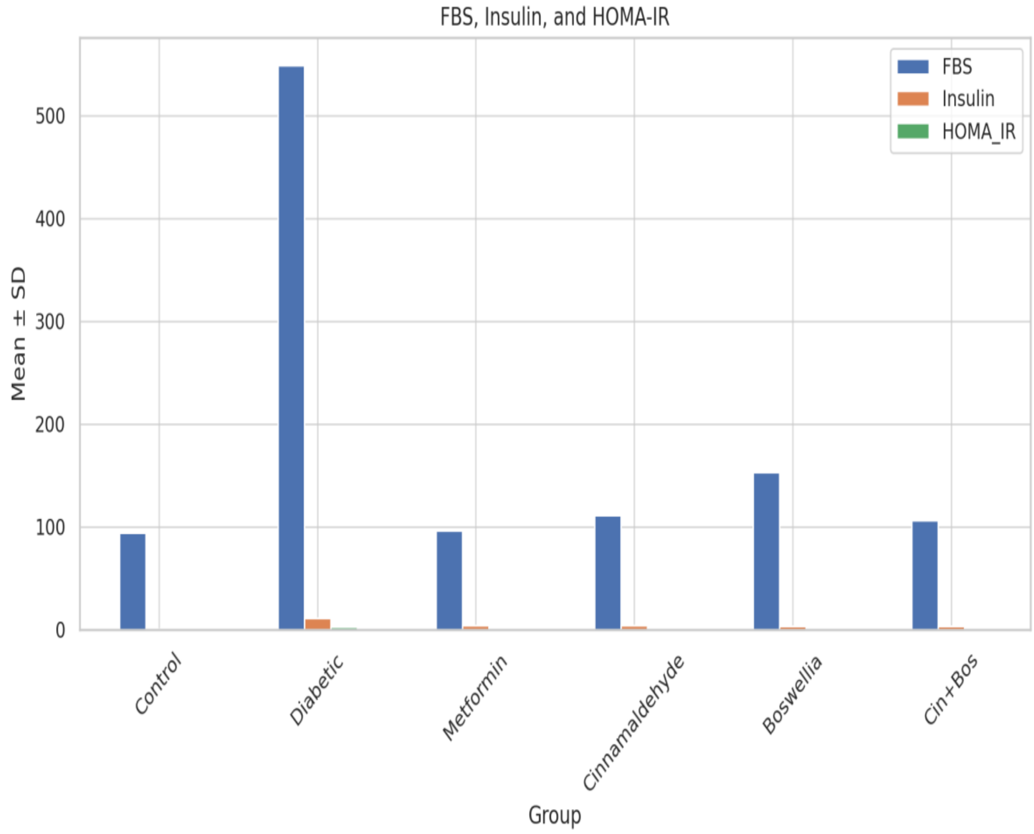

Figure 4. Effect of treatments on fasting blood glucose, insulin levels, and HOMA-IR index

Diabetic rats exhibited severe hyperglycemia and insulin resistance. All treatments improved glucose metabolism, with the combination group showing the greatest hypoglycemic and insulin-sensitizing effect.

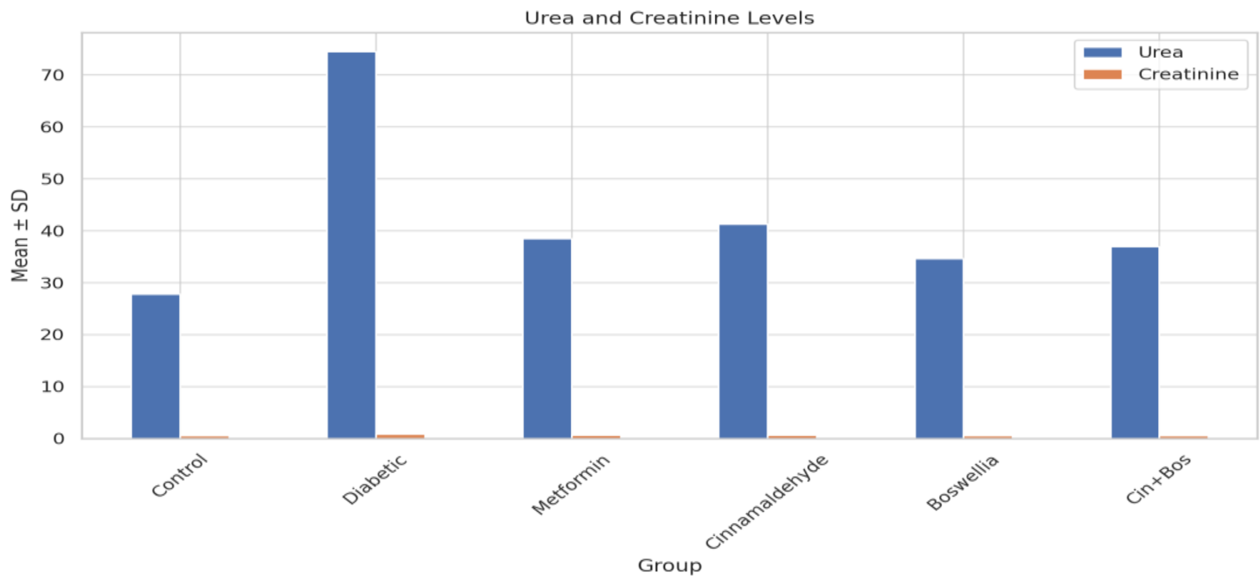

Figure 5. Effect of treatments on renal function (urea and creatinine).

Diabetes caused significant elevation of urea and creatinine levels, indicating renal impairment. Boswellia and the combination groups markedly improved renal function, highlighting nephroprotective effects.

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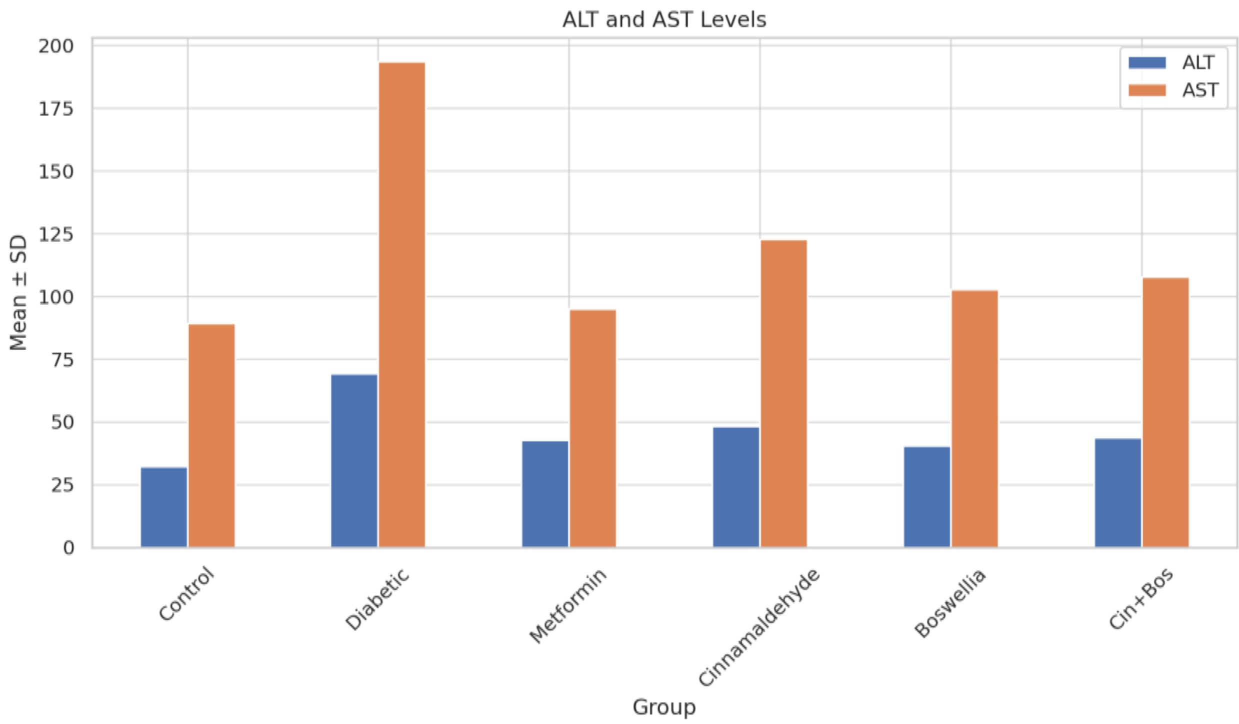
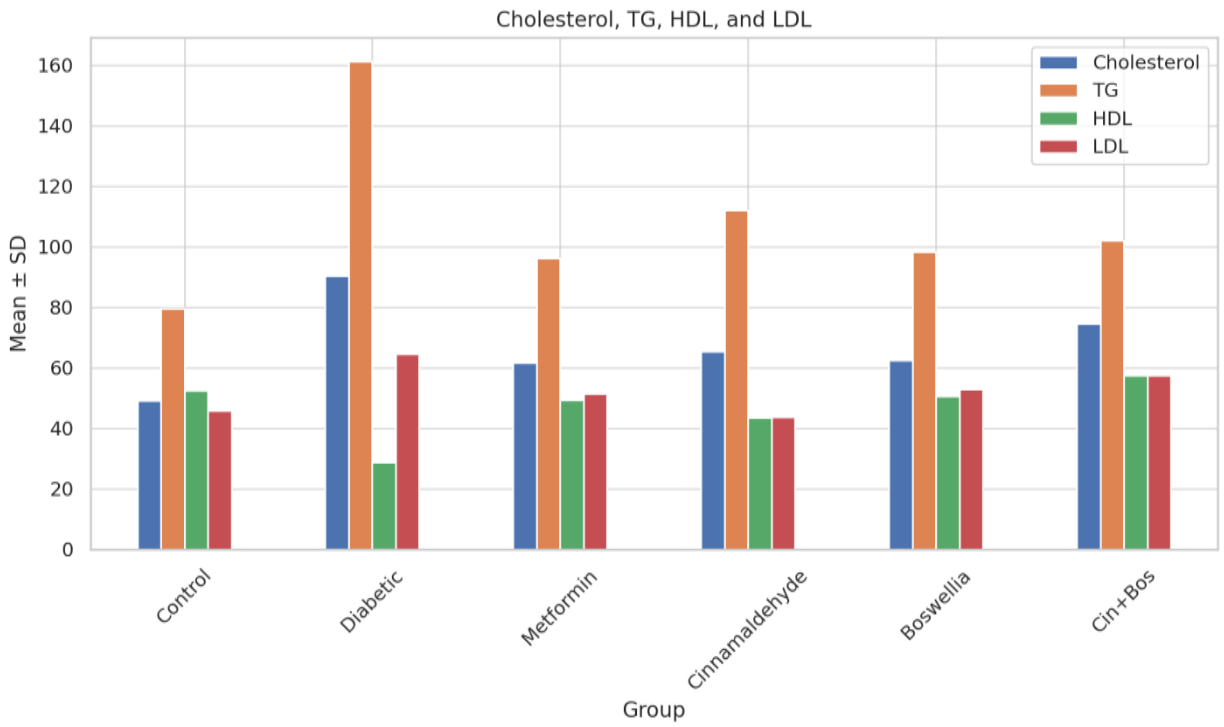


Figure 6. Effect of treatments on liver enzymes (ALT, AST).

Comment: Diabetic rats had elevated ALT and AST, reflecting liver injury. Treatments reduced enzyme levels, with the combination group demonstrating the strongest hepatoprotective effect



).Figure 7. Effect of treatments on lipid profile (TC, TG, HDL, LDL
Diabetic rats developed dyslipidemia, including elevated TC, TG, LDL, and reduced HDL. Treatments improved lipid parameters, with the combination group restoring HDL levels and reducing atherogenic lipids most effectively

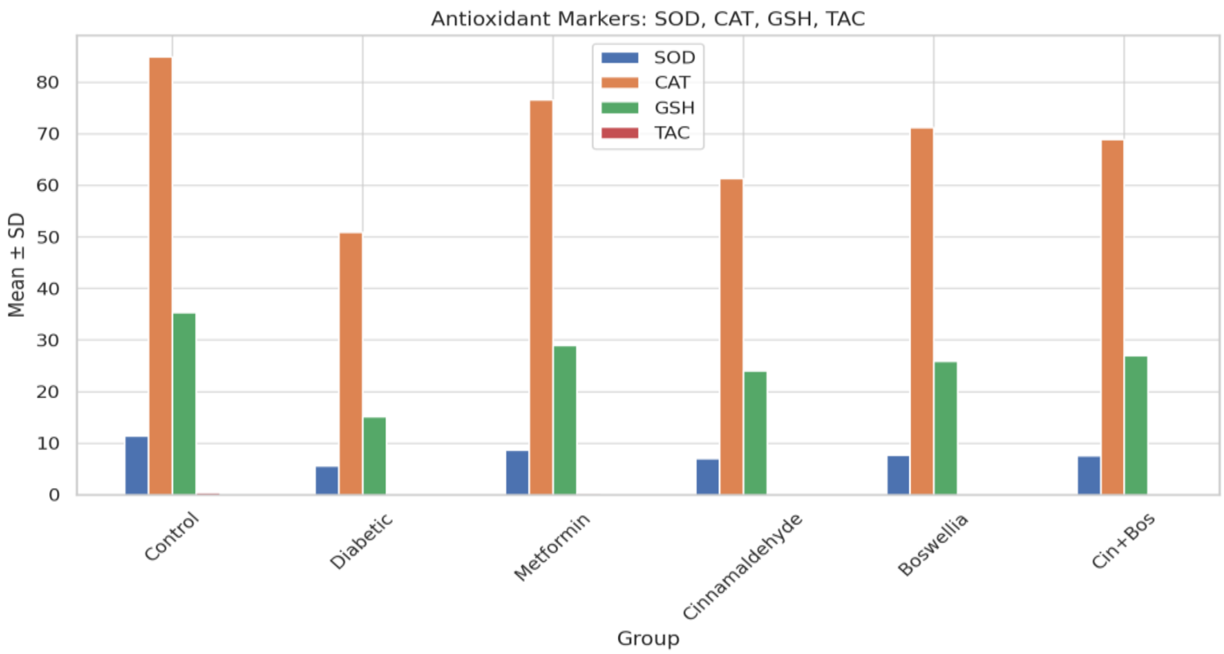


Figure 8. Effect of treatments on antioxidant and oxidative stress markers (SOD, CAT, GSH, TAC)
Antioxidant defenses were impaired in diabetic rats. Treatments enhanced antioxidant activity, particularly in the combination group, which showed the closest values to the control.

Table 1: *Comparing body weight between groups*

Group	Initial body weight (IBW)		Final body weight (FBW)		P.value
	Mean	SD	Mean	SD	
Control	145.0	4.82	180.3	3.06	0.0126*
Diabetic	155.3	3.59	162.5	5.85	0.2717
Metformine	160.5	3.29	185.0	4.65	0.0016*
Cinemaldhyde	150.3	3.59	165.5	5.85	0.0177*
Boswillia serrata	162.5	3.11	195.5	3.11	0.0002*
Cin+Bos	152.8	2.63	202.0	4.79	0.0001*
P.value	0.0062* P1*, P2*		0.0116* P3*, P4*, P5*		------

*P ≤ 0.05 is considered significant P1: Bos vs Control,DM,Cin,Cin+Bos P2:Met vs control, Cin,Cin+Bos P3: Cin+Bos vs Control, DM, Met, Cin P4: Bos vs Control, DM, Met, Cin P5:Met vs DM, Cin

Table 2: *Comparing RBC, HB, and platelets between groups*

Group	R.B.Cs ($\times 10^6/\mu L$)		Hb (g/dL)		Platelets ($\times 10^3/\mu L$)	
	Mean	SD	Mean	SD	Mean	SD
Control	6.90	0.45	12.07	0.83	623.75	101.9
Diabetic	3.55	0.28	7.85	0.60	367.35	42.3
Metformine	7.33	0.48	13.12	0.63	698.25	95.3
Cinemaldhyde	6.95	0.28	13.00	0.3	821.25	100.5
Boswillia serrata	6.12	1.35	11.91	2.5	598.75	85.3
Cin+Bos	7.02	0.52	13.43	1.06	787.5	112.6
P.value	0.0320* P1*		0.0019* P1*		0.0429* P1* , P2*	

*P ≤ 0.05 is considered significant P1: DM vs Control, Met, Cin, Bos, Cin+Bos P2:Cin vs Bos

Table 3: *Comparing other blood cells between groups*

Group	W.B.Cs ($\times10^3/\mu L$)		Monocytes (%)		Lymphocytes (%)		Neutrophils (%)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Control	15.87	1.04	5.27	0.59	19.25	2.26	75.2	0.85
Diabetic	9.80	0.65	9.13	0.32	32.80	3.86	61.92	0.76
Metformine	13.38	2.1	6.71	0.88	24.05	2.57	69.25	1.7
Cinemaldhyde	13.51	0.98	5.62	1.49	19.71	13.55	73.70	0.34
Boswillia serrata	14.32	3.7	7.32	1.54	21.25	4.2	69.55	2.67
Cin+Bos	14.74	2.05	7.40	1.06	22.35	8.16	68.75	6.13
P.value	0.0478* P1*, P2*		0.0384* P2*, P3*		0.0301* P2*		0.0056* P4*,P5*	

*P ≤ 0.05 is considered significant P1: Control vs DM, Met,Cin P2:DM vs control, Met, Cin, Bos, Cin+Bos P3: Control vs DM, Cin+Bos P4: Control vs DM, Met,Cin,Bos, Cin+Bos P5:Cin vs DM,Met,Bos

Table 4: *Comparing FBS, Insulin, and Homa IR between groups*

Group	FBS (mg/dl)		Insulin		Homa IR	
	Mean	SD	Mean	SD	Mean	SD
Control	94.50	9.19	0.92	0.00	1.30	0.14
Diabetic	548.50	55.86	11.30	2.12	2.60	0.28
Metformine	96.00	22.62	3.95	0.21	0.95	0.21
Cinemaldhyde	111.50	19.09	4.40	0.28	1.20	0.14
Boswillia serrata	153.00	49.49	3.10	0.28	1.15	0.21
Cin+Bos	106.00	11.31	3.70	0.14	0.95	0.07
P.value	< 0.001* P1*		< 0.001* P1*		0.002* P1*	

*P ≤ 0.05 is considered significant. P1: DM vs Control, Met, Cin, Bos, Cin+Bos

Table 5: *Comparing renal functions between groups*

Group	**Urea**		**Creatinine**	
	Mean	SD	Mean	SD
Control	27.75	6.23	0.523	0.23
Diabetic	74.43	9.45	0.894	0.10
Metformine	38.5	7.11	0.649	0.11
Cinemaldhyde	41.23	6.81	0.612	0.15
Boswillia serrata	34.6	5.78	0.598	0.21
Cin+Bos	36.9	4.34	0.563	0.17
P.value	0.0078* P1*		0.0116* P1*	

*P ≤ 0.05 is considered significant. P1: DM vs Control, Met, Cin, Bos, Cin+Bos

Table 6: *Comparing liver functions between groups*

Group	**ALT** (U/L)		**AST** (U/L)	
	Mean	SD	Mean	SD
Control	32.33	3.45	89.2	9.15
Diabetic	69.33	7.23	193.5	15.19
Metformine	42.7	5.11	95.1	9.89
Cinemaldhyde	48.21	6.27	122.7	12.45
Boswillia serrata	40.6	4.13	102.8	10.57
Cin+Bos	43.7	4.22	107.7	11.12
P.value	0.0078* P1*		0.0116* P1*, P2*	

*P ≤ 0.05 is considered significant P1: DM vs Control, Met, Cin, Bos, Cin+Bos

P2: Cin vs Control, Met

Table 7: *Comparing Lipid profiles between groups*

Group	CH (mg/dl)		TG (mg/dl)		HDL (mg/dl)		LDL (mg/dl)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Control	49.2	5.12	79.5	8.23	52.5	5.45	45.7	4.44
Diabetic	90.4	9.89	161.1	14.26	28.7	3.11	64.5	6.45
Metformine	61.7	6.45	96.2	9.46	49.3	5.10	51.5	5.32
Cinemaldhyde	65.3	6.24	112.1	11.29	43.5	4.78	43.6	4.33
Boswillia serrata	62.5	5.56	98.2	8.90	50.5	5.55	52.8	5.22
Cin+Bos	74.5	3.16	102	10.23	57.5	5.89	57.4	5.93
P.value	0.0205* P1*, P2*		0.0410* P1*, P3*		0.0109* P1*		0.0056* P4* , P5*	

*P ≤ 0.05 is considered P1: DM vs Control, Met, Cin, Bos, Cin+Bos P2: Control vs DM, Cin+Bos P3: Control vs DM, Cin, Bos, Cin+Bos P4: DM vs Control, Met, Cin P5: Control vs Cin+Bos

Table 8: *Comparing antioxidant markers between groups*

Group	SOD (U/mg)		CAT (U/mg)		GSH (ng/ml)		TAC (mmol/L)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Control	11.39	2.11	84.86	7.34	35.35	3.44	0.39	0.11
Diabetic	5.54	1.23	50.87	5.22	15.12	2.11	0.15	0.05
Metformine	8.66	1.99	76.58	7.19	28.94	3.10	0.28	0.10
Cinemaldhyde	7.01	1.45	61.3	5.98	24	2.82	0.20	0.08
Boswillia serrata	7.63	2.22	71.21	7.11	25.9	2.91	0.24	0.09
Cin+Bos	7.56	1.67	68.92	6.94	27	3.51	0.21	0.07
P.value	0.0141* P1*		0.0028* P2*, P3*		0.0119* P4*, P5*		0.0056* P1*	

*P ≤ 0.05 is considered P1: Control vs DM P2: Control vs DM, Cin, Cin+Bos P3: DM vs Control, Met, Bos, Cin+Bos P4: DM vs Control, Met, Cin, Bos, Cin+Bos P5: Control vs DM, Cin, Bos, Cin+Bos

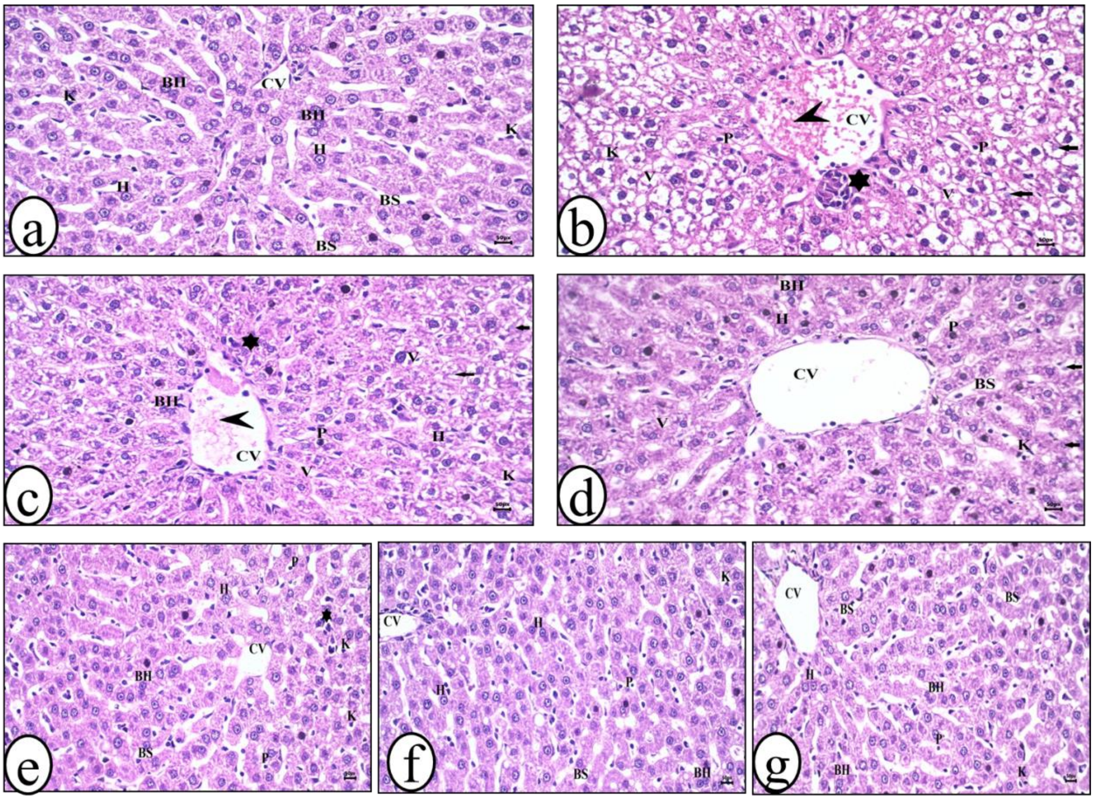


Figure 9: Photomicrographs of liver sections stained with H&E showing: a) Normal control rat liver appeared with normal hepatocytes (arrow) with normal radial arrangements around hepatic cords. Binucleated hepatocytes (BH), Kupffer cells (K), and narrow blood sinusoids lined by an endothelial cell layer (BS). b) Diabetic rat liver showing marked hepatic disorganization, including vacuolation in the cytoplasm of hepatocytes appeared as indistinct clear vacuoles (V), indicates glycogen infiltration in diabetes. Congested central veins (CV) with hemorrhage (head arrow), intensive degeneration of its epithelial lining, and an area around the central vein associated with cellular infiltration (star). Kupffer cells (K), pyknotic nuclei, and karyolytic ones were seen (arrow). c) Liver of diabetic rat treated with cinnamon showed a centrilobular pattern of hepatic degeneration with irregularly branched and congested central vein (CV), widening of blood vessels, area of infiltration around the central vein (star). Binucleated (BH), vacuolated hepatocytes (V) that have pyknotic nuclei (P), vesicular nuclei, and megakaryocytic nuclei; others show eosinophilia (arrow). Kupffer cells also appeared (K). d) *B. Serrata* administered to diabetic rats showed centrilobular regeneration, with the restoration of the central vein (CV), vacuole, sinusoidal spaces (BS), hepatocytes with mild necrosis, and mild infiltration of inflammatory cells (H). e,f) Liver sections of diabetic rats administered with metformin and also *B. Serrata &* cinnamon showed partial improvement of the hepatic architecture with minimal tissue degeneration (H), but there is cellular infiltration still appearing (star) around the wide central vein (CV). g) *B. Serrata and metformin-treated* liver showed improvement of the more normal hepatocytes (H) with mild vacuolation of hepatocytes (V), slight widening of the central vein (CV), kupffer cells (K), binucleated hepatocytes (BH), and Hepatocytes with pyknotic nuclei (P) also appeared (x400).

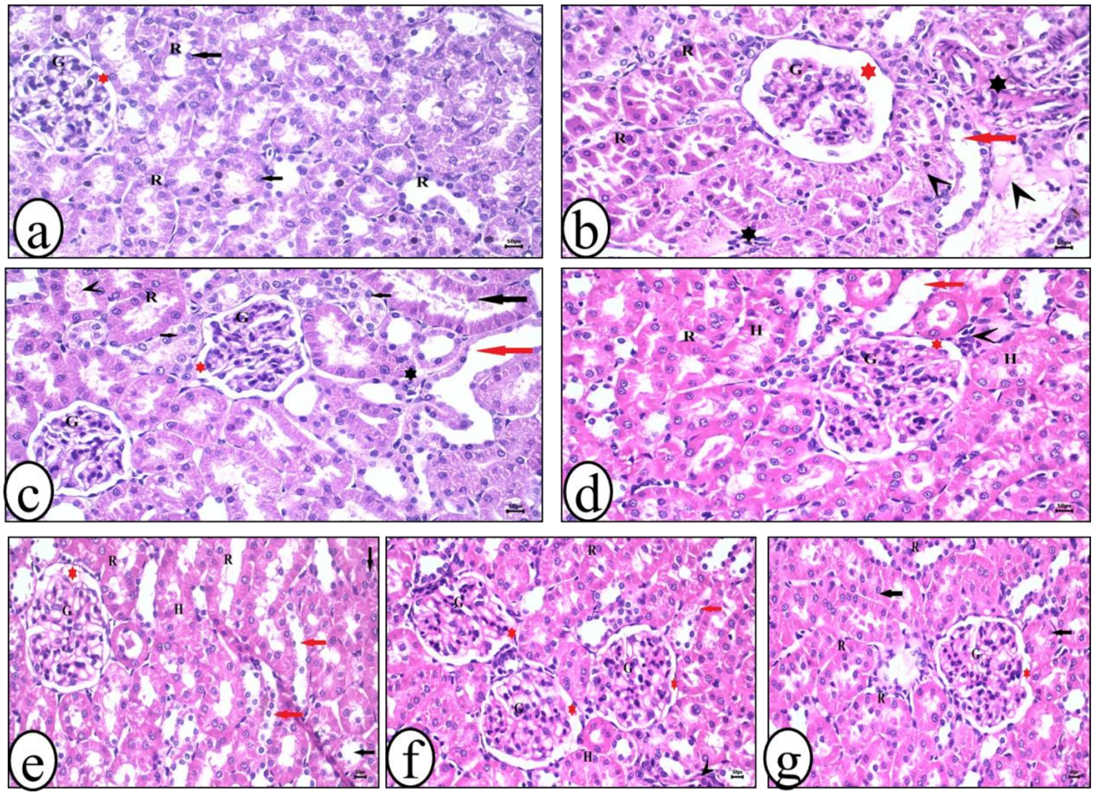


Figure 10: Photomicrographs of kidney sections stained with H&E showing: a) Normal control rats appeared with a normal glomerulus (G), proximal and distal convoluted tubules lined by cuboidal epithelium (R), and Bowman's capsule with normal space (red star); there were no signs of inflammation. b) Diabetic rats' kidney sections showed disorganized, congested, shrunken, and destroyed glomeruli (G) with irregular Bowman's space (red star). The majority of the renal tubules were widened (red arrow), injured with degenerated epithelial lining cells, resulting it losing their characteristic appearance with intertubular haemorrhage. Dilated, congested renal blood vessels, which are engorged with blood (head arrow), also may occlude with hyaline casts and interlobular infiltration (black star). c) Kidney sections of diabetic rats administered cinnamon showed destroyed, shrunken, and congested glomeruli (G) with irregularly destructed Bowman's capsule, wide Bowman's space (red star). Destroyed renal tubules that lost their typical appearance with distinct vacuolated and highly degenerated lining epithelium (red arrow); cellular infiltration at the intertubular spaces was observed (black star), and appearance of cellular hemorrhage at the intertubular spaces (head arrow). d) Kidney sections of diabetic rats administered with *B. Serrata* showed disorganized glomeruli (G) with narrow Bowman's space (red star), damaged renal tubules with undistinguished lining epithelium, which lost their distinctive structure (red arrow). e) After administration of metformin, partial improvement in glomeruli with glomerular shrinkage disappearing (G). f) Kidney sections of diabetic rats that were administered with *B. Serrata and cinnamon* also showed partial improvement in glomeruli structure (G), but accompanied by glomerular shrinkage and intertubular hemorrhage still appear (arrowhead). g) *B. Serrata and metformin* administered to diabetic rats showed a normal-like architecture of glomeruli (G) with a normal Bowman's capsule (red star). Most of the renal tubules were improved with normal appearance (black arrow), and renal blood vessels (BV) were normal (x 400).

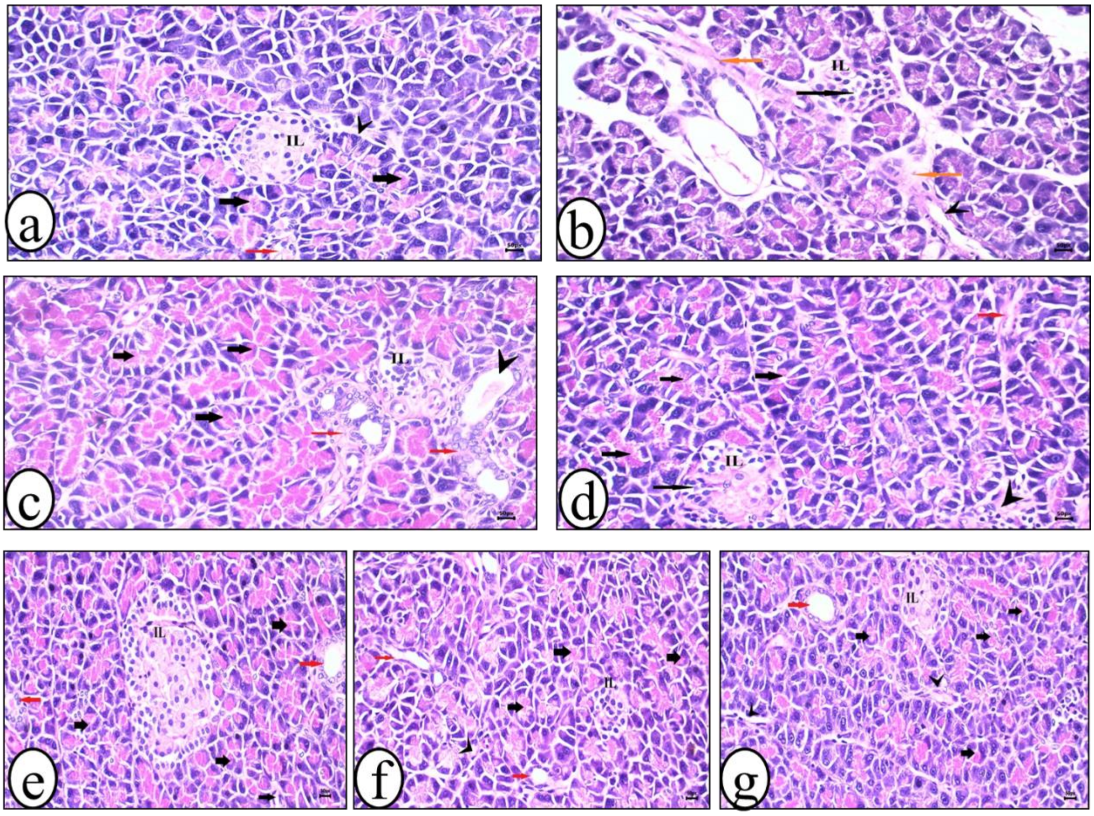


Figure 11: Photomicrographs of pancreas sections stained with H&E. a) Pancreas of normal control rat showing normal cellular composition in the normal-sized islets of Langerhans (IL) with rich vascular supply as well as pale rounded and ovoid normal exocrine acinar cells β-cells in the center (thick arrows), embedded in exocrine portion of pancreas that were arranged in irregular cords with a normal intralobular duct (arrowheads), and normal blood vessels in between (thin red arrows). b) Pancreas section of diabetic rats showing shrinkage of islets of Langerhans (IL) with severe cell damage, inflammation, degeneration and necrosis of exocrine acini cells (thick arrows), the cytoplasm appeared vacuolated (V) with pyknotic nuclei (P), and many cells showed hydropic degeneration where its nucleus appeared densely basophilic and karyolysis is evident (arrow), and severely dilated and congested blood vessels with thick vascular walls (thin red arrows). c) Pancreas sections treated with *B. Serrata* showed no improvement in cellular architecture (black arrow). d) Pancreas sections treated with cinnamon extracts showed partial improvement appeared in cellular architecture as measured by regeneration with restoration of normal-like islet (IL) and pancreatic cell population size, a mild interstitial inflammatory cell infiltrate associated with acinar hyperplasia (black arrow). e) Pancreas sections treated with Metformin showed the most significant improvement of normal islets of Langerhans (IL) with their normal pale large round to ovoid-shaped containing cells that were embedded in the exocrine portion of the pancreas. Acinar cells retain their normal structure and appearance (arrow). f) Pancreas section treated with *B. Serrata* and cinnamon showed improvement of pancreatic tissue with normal architecture of the pancreatic islets (IL). The cytoplasm becomes granulated, the vacuoles of β cells disappear, and nuclei become normal (black arrow). g) Pancreas section of diabetic rat treated with *B. Serrata* and metformin showing well-defined, normally sized islets of Langerhans (IL), but some degeneration of proliferated β cell populations in the center between normal pyramidal acidophilic pancreatic acini (thick arrow) were noticed with normal ducts (red arrow) and normal vascular structure (head arrow) (x400).

4. Discussion:

DM is a multifactorial disease distinguished by persistent hyperglycemia owing to insulin resistance and/or deficiency in insulin secretion. The chronic elevation of blood glucose induces oxidative stress and systemic inflammation, which contribute to a broad range of vascular and metabolic complications involving essential organs, including the liver, kidneys, and pancreas [40]. In this study, the administration of *Boswellia serrata* extract and cinnamaldehyde (CA) individually and in combination demonstrated significant improvements in metabolic, biochemical, and histological characteristics in STZ-male rats with induced diabetes.

The experimental model used in this study successfully mimicked the pathophysiological characteristics of type 2 diabetes through lipid-rich diet administration followed by low-dose STZ injection. This model induced hyperglycemia, insulin resistance, and dyslipidemia, along with signs of hepatic and renal impairment. These alterations are consistent with other studies demonstrating that STZ induces oxidative stress and β-cell dysfunction [29, 30].

A significant reduction in fasting blood glucose (FBG) and HOMA-IR index was observed in all treatment cohorts, especially in the cohort administered the Boswellia and CA combination. This impact may be ascribed to the capacity of both medicines to augment insulin secretion, rehabilitate pancreatic β-cell function, and boost insulin sensitivity. Boswellia's triterpenes may stimulate insulin release and reduce glucose absorption, while cinnamaldehyde activates insulin receptor substrates and glucose transporters such as GLUT4 [25, 41]. Moreover, cinnamaldehyde has been reported to stimulate AMPK and PI3K/Akt signaling pathways, promoting glucose uptake in peripheral tissues and reducing hepatic gluconeogenesis [23]. These molecular mechanisms are consistent with the observed biochemical improvement in glucose homeostasis.

The results revealed that diabetic rats exhibited significantly decreased antioxidant enzyme activities (SOD, CAT, GSH), indicating increased oxidative stress. Treatment with Boswellia and CA reversed these changes, likely due to their well-established antioxidant properties. Boswellia has been shown to scavenge free radicals, enhance endogenous antioxidant systems, and downregulate pro-oxidant gene expression [19] .CA also protects against ROS by preserving mitochondrial integrity and modulating redox-sensitive signaling cascades [42].

Inflammation is another critical component of diabetic pathology, contributing to β-cell dysfunction and insulin resistance. Both agents possess anti-inflammatory activity through the inhibition of nuclear factor-kappa B (NF-κB), tumor necrosis factor-alpha (TNF-α), and interleukin-6 (IL-6). This dual antioxidant and anti-inflammatory action likely underpins their therapeutic efficacy [43].

Diabetic rats exhibited classical dyslipidemia characterized by elevated total cholesterol, triglycerides, and LDL-C, along with decreased HDL-C. Treatment with Boswellia and CA significantly restored lipid balance. Boswellia may exert lipid-lowering effects through the modulation of lipid metabolism enzymes and cholesterol absorption, as supported by earlier findings [22] . CA, on the other hand, enhances lipid clearance and reduces hepatic lipid accumulation, possibly via PPAR-α and PPAR-γ activation [44] . These changes reduce the atherogenic risk index, making these agents potentially useful for cardiovascular risk reduction in diabetic patients [45].

Increased serum ALT, AST, urea, and creatinine levels in diabetic rats indicate hepatic and renal dysfunction. These alterations were significantly ameliorated by Boswellia and CA, reflecting their hepato- and nephron-protective actions. Boswellia reduces hepatic inflammation and fibrosis through inhibition of collagen synthesis and fibrosis. CA exhibits renal protection by attenuating glomerular injury, reducing oxidative DNA damage, and inhibiting apoptosis in kidney tissue.

Histological results confirmed these findings, with Boswellia and CA preventing cellular degeneration and necrosis in liver and kidney tissues. The combination group showed the greatest preservation of tissue integrity, supporting the biochemical data.

Changes in RBCs, WBCs, Hb, and platelets are common in diabetic conditions due to glycation and oxidative stress. Boswellia and CA were able to normalize these values, likely due to their antioxidant roles in protecting red blood cells from hemolysis and maintaining bone marrow function. Notably, the combination group restored RBC and platelet levels closer to normal, highlighting its immunomodulatory potential [46, 47].

One of the most remarkable findings was the enhanced efficacy of the combination group. Co-administration of Boswellia and CA resulted in greater improvements than either agent alone across all parameters-glucose metabolism, lipid profile, organ function, oxidative stress markers, and histological recovery. This suggests a synergistic interaction, likely due to the complementary mechanisms of both agents. Such combination therapies may allow dose reduction and minimize side effects while maximizing efficacy.

5. Conclusion:

The findings of this experimental study strongly suggest that Boswellia serrata and cinnamaldehyde-whether administered individually or in combination—offer promising therapeutic effects in the management of type 2 diabetes mellitus (T2DM). Both agents demonstrated potent hypoglycemic, antioxidant, anti-inflammatory, and organ-protective effects.

Treatment with either agent led to significant improvements in fasting blood glucose, insulin sensitivity (HOMA-IR), lipid profiles, and hepato-renal function markers, with enhanced antioxidant enzyme activities (SOD, CAT, GSH, TAC). Histopathological examination confirmed tissue protection and structural improvement in the pancreas, liver, and kidney.

Most notably, the combination therapy of Boswellia and cinnamaldehyde produced superior outcomes across nearly all evaluated parameters, suggesting a synergistic mechanism that could support the development of novel plant-based adjuvant therapies for T2DM.

In conclusion, these findings pave the way for further preclinical and clinical trials to explore the efficacy, safety, and pharmacological potential of Boswellia and cinnamaldehyde as complementary or alternative treatments for diabetes and its associated complications.

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