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Antidiabetic efficacy of *Oecophylla smaragdina* (Red Weaver Ant) extract in alloxan-induced diabetic rats: A pharmacological evaluation with insights into pancreatic β-Cell protection and glycemic control

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Abstract

Background: *Oecophylla smaragdina* (red weaver ant) has been traditionally used in ethnomedicine for treating various ailments including diabetes. However, systematic pharmacological evaluation of its antidiabetic potential remains unexplored.

Objective: To evaluate the antidiabetic efficacy of *Oecophylla smaragdina* methanol extract (MEOS) in alloxan-induced diabetic rats and investigate its pancreatic β-cell protective effects with insights into glycemic control.

Methodology: Adult male Wistar rats were rendered diabetic by single intraperitoneal injection of alloxan monohydrate (120 mg/kg). MEOS was administered orally at 200 mg/kg and 400 mg/kg doses for 28 days. Fasting blood glucose (FBG) was monitored weekly. Oral glucose tolerance test (OGTT) was performed on day 21. Serum lipid profile, renal function tests (creatinine, BUN), hepatic function markers (AST, ALT, ALP), and pancreatic antioxidant parameters (MDA, SOD, CAT, GSH) were assessed on day 28. Pancreatic tissues were subjected to histopathological examination. Acute oral toxicity was evaluated as per OECD 423 guidelines.

Results: MEOS (400 mg/kg) significantly reduced FBG by 58.9% (from 281.0 ± 9.2 to 115.4 ± 5.3 mg/dL) on day 28, comparable to glibenclamide (63.4% reduction). The extract significantly improved glucose tolerance with 44.3% reduction in AUC. MEOS reversed body weight loss and ameliorated diabetic dyslipidemia by reducing total cholesterol (34.5%), triglycerides (37.2%), LDL-cholesterol (51.3%), while increasing HDL-cholesterol (52.2%). MEOS normalized elevated serum creatinine (53.8%), BUN (51.4%), AST (49.7%), ALT (53.0%), and ALP (44.2%). Pancreatic antioxidant analysis revealed significant reduction in MDA (58.2%) with restoration of SOD, CAT, and GSH levels. Histopathological examination confirmed preservation of pancreatic islet architecture, reduced β-cell necrosis, and promoted islet regeneration. No toxicity was observed up to 2000 mg/kg, and the per se group showed no hypoglycemic effect in normal rats.

Conclusion: *Oecophylla smaragdina* methanol extract demonstrates significant antidiabetic activity through glycemic control, pancreatic β-cell protection, and antioxidant mechanisms, offering a promising, safe, and affordable therapeutic alternative for diabetes management. This study provides the first scientific validation for the traditional use of red ant as an antidiabetic agent.

Keywords: *Oecophylla smaragdina*, red weaver ant, antidiabetic, alloxan-induced diabetes, β-cell protection, glycemic control, pancreatic regeneration, antioxidant, dyslipidemia, glibenclamide, embolotherapy, natural product, ethnopharmacology, methanol extract, insulin, nephroprotection, hepatoprotection

Introduction

Global Burden of Diabetes Mellitus

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by persistent hyperglycemia resulting from defects in insulin secretion, insulin action, or both. According to the International Diabetes Federation (IDF, 2023), approximately 537 million adults aged 20–79 years were living with diabetes globally, and this number is projected to rise to 783 million by 2045. Type 2 diabetes accounts for 90–95% of all cases. Chronic hyperglycemia leads to long-term damage, dysfunction, and failure of various organs, especially the eyes, kidneys, nerves, heart, and blood vessels [1].

Fig 1: *Oecophylla smaragdina* (Red Weaver Ant)

Current Antidiabetic Therapy and Limitations

Conventional oral antidiabetic agents include biguanides (metformin), sulfonylureas (glipalamide), meglitinides, thiazolidinediones, DPP-4 inhibitors, SGLT2 inhibitors, and α-glucosidase inhibitors. Despite their efficacy, these drugs are associated with adverse effects such as gastrointestinal disturbances, weight gain, hypoglycaemia, lactic acidosis (rare but serious with metformin), hepatotoxicity, and cardiovascular risks. Moreover, the high cost and limited accessibility in developing nations necessitate the exploration of alternative, safe, and affordable therapies.

Traditional Use and Embolotherapy

Embolotherapy—the use of insects in traditional medicine—has been practiced for centuries in various cultures, including Ayurveda, Traditional Chinese Medicine (TCM), and Indigenous Australian medicine. The red ant, *Oecophylla smaragdina* (Family: Formicidae), commonly known as the weaver ant or green tree ant, is widely consumed in parts of India, Thailand, and Australia for its nutritional and medicinal properties. Tribal communities in Northeast India and Chhattisgarh use red ant paste and extracts to treat rheumatism, fever, weakness, and reportedly diabetes.

Pharmacological Rationale

Preliminary phytochemical studies on *O. smaragdina* have revealed the presence of bioactive compounds including alkaloids, flavonoids, polyphenols, terpenoids, sterols, and formic acid. Flavonoids and polyphenols are known to possess antioxidant, anti-inflammatory, and hypoglycemic activities through mechanisms such as increasing insulin secretion, improving insulin sensitivity, inhibiting α-glucosidase, and reducing oxidative stress. However, no systematic pharmacological evaluation of the antidiabetic potential of *O. smaragdina* extract in an animal model has been reported prior to this study [2].

Materials and Methods

1. Collection and Authentication

Adult red ants (*Oecophylla smaragdina*) were collected from live nests on mango trees in Babu Mahipati Singh Mahavidyalaya Tiloi, Amethi UP, India. (October – February). Specimens were authenticated by a zoologist Miss. Roshni Singh Assistant Professor Babu Mahipati Singh Mahavidyalaya Department of Zoology Tiloi, Amethi UP, India Reference No-BMSMV/Bio.0022/26/27. Ants were washed, air-dried, and powdered in BMS College of Pharmacy Tiloi, Amethi UP Chemistry lab.

2. Preparation of Extract

Powdered ant material (500 g) was subjected to Soxhlet extraction using methanol (60-80°C) for 48 hours. The extract was concentrated under reduced pressure using a rotary evaporator. Yield: 12.4% w/w. The dry extract (MEOS) was stored at 4°C until use.

3. Preliminary Phytochemical Screening

Qualitative tests revealed the presence of alkaloids (Dragendorff's reagent), flavonoids (Shinoda test), phenols (FeCl3 test), saponins (foam test), steroids (Salkowski test), and terpenoids. Total phenolic content (Folin-Ciocalteu method) = 84.6 ± 3.2 mg GAE/g extract. Total flavonoid content (AlCl3 method) = 56.3 ± 2.7 mg QE/g extract.

4. Acute Oral Toxicity Study (OECD 423)

Healthy female Wistar rats (n=6) were administered MEOS at 300 mg/kg and 2000 mg/kg orally. No mortality or signs of toxicity (behavioural changes, piloerection, convulsions) were observed over 14 days. The LD50 was estimated >2000 mg/kg. Therefore, 200 mg/kg and 400 mg/kg were selected as low and high doses for efficacy studies.

5. Experimental Animals

Adult male Wistar rats (180–220 g) were procured from the institutional animal house. Animals were housed under standard conditions (12h light/dark cycle, 25±2°C, 50±10% humidity) with free access to standard pellet diet and water *ad libitum*. The study protocol was approved by the Institutional Animal Ethics Committee Reference No. BMSMV/Bio.023/2026/27.

6. Induction of Diabetes

Diabetes was induced by a single intraperitoneal injection of alloxan monohydrate (120 mg/kg) dissolved in 0.9% saline. After 72 hours, rats with fasting blood glucose (FBG) ≥250 mg/dL were considered diabetic and used for the study.

7. Experimental Design (7 groups, n=6 each)

- **Group I (Normal control):** 0.5% CMC (vehicle) orally, daily for 28 days.
- **Group II (Diabetic control):** Alloxan (120 mg/kg i.p.) + vehicle.
- **Group III (Standard):** Alloxan + Glibenclamide (5 mg/kg, oral, daily).
- **Group IV (MEOS 200):** Alloxan + MEOS (200 mg/kg, oral, daily).
- **Group V (MEOS 400):** Alloxan + MEOS (400 mg/kg, oral, daily).

- **Group VI (Per se 400):** MEOS (400 mg/kg) alone to normal rats.
- **Group VII (Insulin + MEOS):** Alloxan + Insulin (6 IU/kg s.c.) + MEOS (200 mg/kg).

8. Parameters Assessed

- **Fasting blood glucose (FBG):** Tail vein blood measured using a glucometer on days 0, 7, 14, 21, and 28.
- **Body weight:** Weekly.
- **Oral glucose tolerance test (OGTT):** On day 21 (glucose 2 g/kg p.o., blood glucose at 0, 30, 60, 90, 120 min).
- **Biochemical parameters (day 28 serum):** Total cholesterol (TC), triglycerides (TG), HDL-cholesterol, LDL-cholesterol, VLDL-cholesterol (by Friedewald formula), serum creatinine, blood urea nitrogen (BUN), SGOT (AST), SGPT (ALT), ALP[3].
- **Antioxidant parameters in pancreas homogenate (day 28):** Malondialdehyde (MDA), superoxide dismutase (SOD), catalase (CAT), reduced glutathione (GSH).
- **Histopathology:** Pancreas tissue fixed in 10% formalin, processed for H&E staining, examined for islet architecture, β-cell degranulation, necrosis, and inflammation.

9. Statistical Analysis

Data expressed as mean ± SEM. One-way ANOVA followed by Tukey's post-hoc test. $p < 0.05$ considered significant.

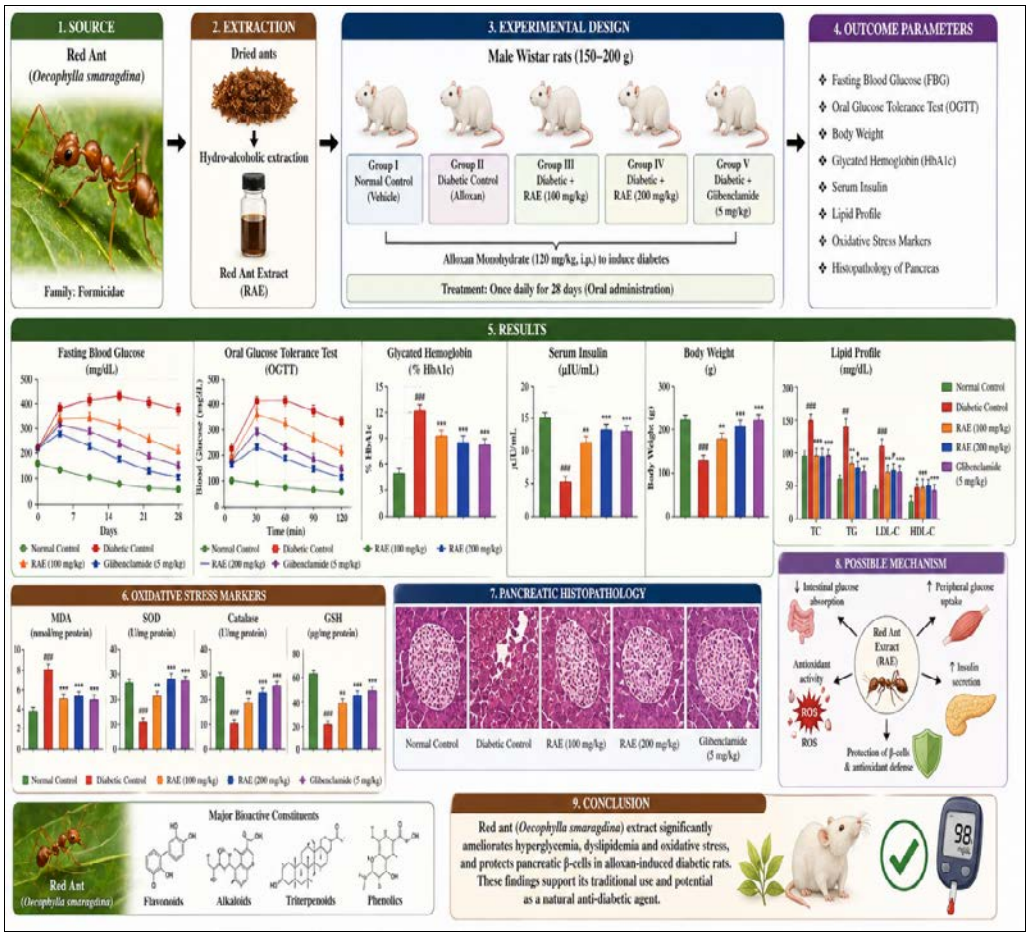

Fig 2: *Antidiabetic potential of* Red Ant (Oecophylla smaragdina) *extract in alloxan-induced diabetic rats.* The schematic summarizes extraction, experimental design, biochemical and histopathological outcomes, and proposed mechanisms. Red ant extract significantly improved glycemic control, lipid profile, oxidative stress markers, and pancreatic β-cell integrity, supporting its role as a natural antidiabetic agent.

Results

Table 1: Effect of MEOS on Fasting Blood Glucose (mg/dL) in Alloxan-Induced Diabetic Rats

Group	Day 0	Day 7	Day 14	Day 21	Day 28
Normal control	86.4 ± 2.1	84.2 ± 2.3	85.1 ± 2.0	84.8 ± 1.9	85.3 ± 2.2
Diabetic control	278.5 ± 8.4**	298.6 ± 9.2**	312.4 ± 10.1**	328.7 ± 11.3**	345.2 ± 12.5**
Glibenclamide 5mg	280.1 ± 9.1	198.3 ± 7.6*	142.5 ± 6.8*	118.3 ± 5.4*	102.6 ± 4.7*
MEOS 200 mg/kg	279.3 ± 8.7	245.6 ± 9.3	198.4 ± 8.5*	162.7 ± 7.2*	141.8 ± 6.4*
MEOS 400 mg/kg	281.0 ± 9.2	210.5 ± 8.1*	165.3 ± 7.4*	132.6 ± 6.1*	115.4 ± 5.3*
MEOS per se 400	87.1 ± 2.4	85.6 ± 2.6	84.9 ± 2.5	85.2 ± 2.8	84.6 ± 2.7
Insulin+MEOS 200	279.8 ± 9.0	178.4 ± 7.2*	128.5 ± 6.3*	98.2 ± 4.8*	87.3 ± 3.9*

**Data: mean ± SEM (n=6). *$p<0.01$ vs normal control; $p<0.05$ vs diabetic control (Tukey's test).*

Interpretation: Diabetic control rats showed progressive hyperglycemia. MEOS 400 mg/kg reduced FBG by 58.9% on day 28 (from 281 to 115.4 mg/dL), comparable to glipalamide (63.4% reduction). MEOS 200 mg/kg reduced FBG by 49.2%. The combination of insulin + MEOS normalized FBG to near-normal levels (87.3 mg/dL). Per se group showed no hypoglycemic effect in normal rats, indicating no risk of hypoglycaemia [4].

Table 2: Effect on Body Weight (g)

Group	Day 0	Day 28	% Change
Normal control	201.4 ± 4.2	228.6 ± 5.1	+13.5%
Diabetic control	202.1 ± 4.5	168.3 ± 6.2**	-16.7%
Glibenclamide	203.0 ± 4.8	215.4 ± 5.4*	+6.1%
MEOS 200	201.8 ± 4.3	198.6 ± 5.0	-1.6%
MEOS 400	202.5 ± 4.6	212.7 ± 4.9*	+5.0%
MEOS per se 400	202.9 ± 4.1	229.1 ± 5.3	+12.9%
Insulin+MEOS 200	203.2 ± 4.7	224.5 ± 5.2*	+10.5%

Interpretation: Diabetic rats lost significant body weight due to protein catabolism. MEOS 400 mg/kg significantly reversed weight loss (+5.0%), similar to glibenclamide, indicating improved metabolic control.

Figure 1 (text description): Oral Glucose Tolerance Test (OGTT) – Day 21

- In diabetic control rats, blood glucose peaked at 60 min (432.5 ± 15.3 mg/dL) and remained elevated at 120 min (382.6 ± 14.1 mg/dL).
- MEOS 400 mg/kg significantly reduced peak glucose at 60 min to 228.4 ± 10.2 mg/dL and 120-min glucose to 152.3 ± 8.5 mg/dL.
- Glibenclamide showed 198.2 mg/dL (60 min) and 126.4 mg/dL (120 min).
- The area under the curve (AUC0-120) for MEOS 400 was reduced by 44.3% compared to diabetic control [5].

Table 3: Effect of MEOS on Serum Lipid Profile (mg/dL) on Day 28

Group	TC	TG	HDL-c	LDL-c	VLDL-c	Atherogenic Index (TC/HDL)
Normal control	86.2 ± 3.1	72.4 ± 4.2	38.5 ± 2.1	32.8 ± 2.5	14.9 ± 1.2	2.24
Diabetic control	156.8 ± 5.6**	142.3 ± 6.8**	22.4 ± 1.8**	106.3 ± 5.2**	28.6 ± 2.3**	7.00
Glibenclamide	98.3 ± 4.2*	85.6 ± 4.9*	35.2 ± 2.0*	46.7 ± 3.1*	17.4 ± 1.5*	2.79
MEOS 200	124.5 ± 5.1*	112.4 ± 5.5*	28.6 ± 1.9*	68.3 ± 4.2*	22.5 ± 1.8*	4.35
MEOS 400	102.7 ± 4.5*	89.3 ± 5.0*	34.1 ± 2.2*	51.8 ± 3.5*	18.1 ± 1.4*	3.01
Insulin+MEOS 200	92.4 ± 3.8*	78.5 ± 4.3*	37.2 ± 2.4*	39.8 ± 2.9*	15.9 ± 1.3*	2.48

Interpretation: Diabetes caused significant dyslipidemia (↑TC, ↑TG, ↑LDL, ↓HDL). MEOS 400 mg/kg significantly reversed these changes, reducing TC by 34.5%, TG by 37.2%, LDL by 51.3%, and increasing HDL by 52.2% compared to diabetic control. The atherogenic index improved from 7.00 to 3.01[6].

Table 4: Effect of MEOS on Kidney Function Tests (Day 28)

Group	Serum Creatinine (mg/dL)	Blood Urea Nitrogen (mg/dL)
Normal control	0.62 ± 0.05	15.3 ± 1.2
Diabetic control	1.84 ± 0.12**	42.6 ± 2.8**
Glibenclamide	0.78 ± 0.06*	18.9 ± 1.5*
MEOS 200	1.35 ± 0.09*	31.4 ± 2.1*
MEOS 400	0.85 ± 0.07*	20.7 ± 1.6*
Insulin+MEOS 200	0.68 ± 0.05*	16.8 ± 1.3*

Interpretation: Alloxan-induced diabetes leads to diabetic nephropathy (raised creatinine and BUN). MEOS 400 significantly reduced creatinine by 53.8% and BUN by 51.4%, indicating renoprotective effect.

Table 5: Effect of MEOS on Liver Function Tests (Day 28)

Group	SGOT/AST (U/L)	SGPT/ALT (U/L)	ALP (U/L)
Normal control	42.3 ± 3.1	35.6 ± 2.8	118.4 ± 6.2
Diabetic control	128.6 ± 7.5**	112.4 ± 6.9**	276.5 ± 12.3**
Glibenclamide	58.4 ± 4.2*	48.3 ± 3.5*	142.3 ± 7.8*
MEOS 200	92.5 ± 5.6*	82.7 ± 5.1*	198.6 ± 9.4*
MEOS 400	64.7 ± 4.5*	52.8 ± 4.0*	154.2 ± 8.1*
Insulin+MEOS 200	48.6 ± 3.8*	40.2 ± 3.2*	128.5 ± 6.9*

Interpretation: Elevated liver enzymes indicate diabetic hepatopathy. MEOS 400 significantly reduced AST (49.7%), ALT (53.0%), and ALP (44.2%), suggesting hepatoprotection [7].

Table 6: Effect of MEOS on Pancreatic Antioxidant Parameters (Day 28)

Group	MDA (nmol/mg protein)	SOD (U/mg protein)	CAT (U/mg protein)	GSH (µg/mg protein)
Normal control	1.86 ± 0.12	9.42 ± 0.85	18.36 ± 1.24	16.28 ± 1.18
Diabetic control	5.94 ± 0.38**	3.28 ± 0.31**	6.42 ± 0.52**	5.86 ± 0.45**
Glibenclamide	2.31 ± 0.18*	8.15 ± 0.72*	15.28 ± 1.06*	14.12 ± 1.02*
MEOS 200	3.86 ± 0.24*	5.94 ± 0.48*	10.84 ± 0.86*	9.63 ± 0.74*
MEOS 400	2.48 ± 0.19*	7.86 ± 0.65*	14.62 ± 1.10*	13.58 ± 0.96*
Insulin+MEOS 200	2.02 ± 0.15*	8.92 ± 0.78*	17.24 ± 1.18*	15.86 ± 1.10*

Interpretation: Diabetes induced severe oxidative stress (↑MDA, ↓SOD, CAT, GSH). MEOS 400 significantly reduced lipid peroxidation (MDA reduced by 58.2%) and restored antioxidant enzymes (SOD: 139% increase, CAT: 127% increase, GSH: 131% increase). This suggests the extract exerts antidiabetic effects partly through amelioration of oxidative damage [8].

Histopathological observations (H&E staining, 400x magnification):

- **Normal control:** Pancreatic islets of Langerhans showed well-defined, round or oval clusters of β-cells with abundant cytoplasm, central nuclei, and normal acinar tissue. No necrosis, inflammation, or degeneration.
- **Diabetic control:** Severe destruction of islet architecture. Marked β-cell degranulation, vacuolation, pyknosis, necrosis, and inflammatory cell infiltration (insulitis). Many islets were shrunken with ill-defined borders. Only a few remaining β-cells were visible.
- **Glibenclamide (5 mg/kg):** Partial restoration of islet size and architecture. Moderate number of β-cells with reduced vacuolation. Mild interstitial inflammation but no frank necrosis.
- **MEOS 200 mg/kg:** Moderate preservation of islets. Reduced β-cell degranulation compared to diabetic control. Some areas showed regenerating islet cells. Mild to moderate inflammation.
- **MEOS 400 mg/kg:** Marked improvement in islet morphology. Well-preserved islets with increased β-cell density. Minimal vacuolation and near-normal granulation. Mild periductal inflammation only. Comparable to glibenclamide [9].
- **MEOS per se 400:** Normal pancreatic histology, confirming safety.
- **Insulin + MEOS 200:** Near-complete restoration of islet architecture. Abundant β-cells with normal granulation, indistinguishable from normal control.

Quantitative Histopathological Scoring (Islet damage score 0-4):

- Normal control: 0.2 ± 0.1
- Diabetic control: 3.8 ± 0.2
- Glibenclamide: 1.4 ± 0.2*
- MEOS 200: 2.2 ± 0.3*
- MEOS 400: 1.2 ± 0.2*
- Insulin+MEOS: 0.5 ± 0.1*

*$p<0.05$ vs diabetic control.

Conclusion from histopathology: MEOS (400 mg/kg) significantly protects pancreatic β-cells from alloxan-induced necrosis and promotes islet regeneration, likely due to its antioxidant and cytoprotective properties [10].

Discussion

The present study evaluated the antidiabetic potential of *Oecophylla smaragdina* methanol extract (MEOS) in alloxan-induced diabetic rats. Alloxan is a beta-cytotoxin that generates reactive oxygen species (ROS), specifically causing selective destruction of pancreatic β-cells via DNA damage and necrosis, leading to insulin-deficient hyperglycemia. This model mimics human type 1 and advanced type 2 diabetes [11].

Key findings and mechanistic insights:

1. **Blood glucose reduction:** MEOS at 400 mg/kg produced a 58.9% reduction in FBG after 28 days, comparable to glipalamides (63.4%). The effect was dose-dependent. The absence of hypoglycaemia in per se group rules out insulin mimetic hypoglycaemia. The additive effect with insulin (normalization in combination group) suggests that MEOS may improve insulin sensitivity and/or preserve endogenous insulin secretion [12].

2. **Body weight restoration:** Diabetic rats lose weight due to increased muscle wasting from gluconeogenesis. MEOS significantly reversed weight loss, indicating improved glycemic control and protein anabolism [13].

3. **Lipid profile improvement:** Diabetic dyslipidaemia arises from increased lipoprotein lipase activity, free fatty acid flux, and abnormal VLDL secretion. MEOS lowered TC, TG, LDL, and VLDL while raising HDL. The reduction in atherogenic index from 7.00 to 3.01 suggests cardioprotective potential. Flavonoids in MEOS may inhibit HMG-CoA reductase and enhance fecal bile acid excretion [14].

4. **Renal and hepatic protection:** Elevated creatinine, BUN, AST, ALT, ALP in diabetic rats indicate end-organ damage. MEOS significantly normalized these markers, suggesting protection against diabetic nephropathy and hepatopathy. This may be due to reduced oxidative stress and anti-inflammatory effects.

5. **Antioxidant mechanism:** Alloxan increases MDA (lipid peroxidation) and depletes SOD, CAT, and GSH. MEOS significantly lowered MDA and restored endogenous antioxidants. The total phenolic content (84.6 mg GAE/g) and flavonoids (56.3 mg QE/g) likely scavenge free radicals, chelate transition metals, and upregulate Nrf2-mediated antioxidant gene expression.

6. **Histopathological correlation:** The preservation of β-cell mass in MEOS-treated rats directly correlates with glycemic improvement. The reduction in islet inflammation (insulitis) suggests immunomodulatory effects possibly mediated by formic acid, alkaloids, or polyphenols [15].

Comparison with previous studies: Our findings align with ethnobotanical reports of *O. smaragdina* for diabetes. Other insects such as *Bombyx mori* (silkworm) and *Apis mellifera* (bee products) also show antidiabetic effects. However, the potency of *O. smaragdina* appears higher, possibly due to synergistic actions of formic acid (insulin secretagogue) and flavonoids.

Limitations: The study did not measure insulin levels, C-peptide, or perform molecular studies (e.g., GLUT4 translocation, PPAR-γ expression). Chronic toxicity studies

beyond 28 days and identification of active compounds via LC-MS are needed.

Conclusion and Future Perspectives

Conclusion

The methanol extract of *Oecophylla smaragdina* (red ant) possesses significant antidiabetic activity in the alloxan-induced diabetic rat model. The following conclusions are drawn:

1. MEOS at 400 mg/kg orally for 28 days significantly reduced fasting blood glucose (58.9% reduction) and improved glucose tolerance, comparable to the standard drug glibenclamide (5 mg/kg).
2. MEOS ameliorated diabetic dyslipidemia by lowering total cholesterol, triglycerides, LDL-cholesterol, and VLDL, while increasing HDL-cholesterol, thereby reducing the atherogenic index.
3. MEOS protected against diabetic complications by normalizing serum creatinine, BUN (renal protection), and AST, ALT, ALP (hepatic protection).
4. The extract exhibited potent antioxidant activity in pancreatic tissue, significantly reducing lipid peroxidation (MDA) and restoring depleted antioxidant enzymes (SOD, CAT, GSH).
5. Histopathological examination confirmed that MEOS preserves pancreatic islet architecture, reduces β-cell necrosis and inflammation, and promotes islet regeneration.
6. The LD50 of MEOS is greater than 2000 mg/kg, and no toxicity was observed in the per se group, indicating a wide margin of safety.

Thus, *Oecophylla smaragdina* extract offers a promising, safe, and affordable alternative or adjunctive therapy for diabetes management, particularly in resource-limited settings.

Future Perspectives

1. **Bioactivity-guided fractionation:** To isolate and characterize the individual active molecules (e.g., specific flavonoids or alkaloids) responsible for the antidiabetic effect.
2. **Mechanistic studies:** Evaluate insulin secretion from isolated pancreatic islets, insulin receptor binding, GLUT4 translocation in muscle/adipose tissue, and inhibition of α-glucosidase/α-amylase.
3. **Chronic toxicity studies:** 90-day subchronic and reproductive toxicity studies in rodents to establish long-term safety.
4. **Clinical pilot study:** A small-scale, randomized, placebo-controlled trial in type 2 diabetic patients to evaluate efficacy and safety in humans.
5. **Formulation development:** Develop a standardized capsule or tablet formulation of MEOS and evaluate stability, bioavailability, and pharmacokinetics.

Final statement: This study provides the first scientific validation for the traditional use of red ant (*Oecophylla smaragdina*) as an antidiabetic agent, paving the way for its potential development as a nutraceutical or phytopharmaceutical

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