Original Article

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Effect of *Mucuna Pruriens* Seed Extract on Glycaemic Control, Inflammation and Lipid Profile in Type 2 Diabetes-induced Rats: An Experimental Study

Pharmacology Section

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ABSTRACT

Introduction: Type 2 Diabetes Mellitus (T2DM) represents a major global health challenge, with rapidly increasing prevalence and significant morbidity due to associated metabolic and inflammatory complications. India bears a substantial share of this burden, with a large and growing population affected by the disease. In addition to persistent hyperglycaemia, type 2 diabetes is characterised by chronic low-grade inflammation and dyslipidaemia, which contribute to insulin resistance and disease progression. Therapeutic agents capable of targeting multiple metabolic abnormalities simultaneously may offer improved disease management. *Mucuna pruriens*, a medicinal plant containing bioactive compounds such as L-3,4-Dihydroxyphenylalanine (L-DOPA), flavonoids and polyphenols, has been reported to possess antioxidant and anti-inflammatory properties.

Aim: To evaluate the effect of *Mucuna pruriens* seed extract on body weight, blood glucose levels, inflammatory markers and lipid profile in High-Fat Diet (HFD) and low-dose streptozotocin-induced T2DM in male Wistar rats.

Materials and Methods: The present experimental animal study was conducted at the Department of Pharmacology, KAHER's Jawaharlal Nehru Medical College, Belagavi, Karnataka, India, from March 2024 to March 2025. Adult male Wistar rats (200±20 g) were used. Type 2 diabetes was induced by feeding a HFD for four weeks followed by intraperitoneal administration of streptozotocin (30 mg/kg). Animals with fasting blood glucose ≥200 mg/dL were included. Rats were divided into four groups (Normal control n=6; Diabetic control n=8; Metformin 180 mg/kg n=8; *Mucuna pruriens* 200 mg/kg n=8). Treatments were administered orally for seven weeks. Body weight and fasting blood glucose were monitored periodically. Serum inflammatory markers {Tumor Necrosis Factor-alpha (TNF-α) and Interleukin-6 (IL-6)} and lipid parameters were estimated at the end of the study. Statistical analysis was performed using One-way Analysis of Variance (ANOVA) followed by Tukey's post-hoc test.

Results: Diabetic control animals developed marked hyperglycaemia (311.1±3.8 mg/dL). Treatment with *Mucuna pruriens* (200 mg/kg) significantly reduced fasting blood glucose levels to 167.5±15.4 mg/dL compared with diabetic control (311.1±3.8 mg/dL; $p<0.001$) and metformin (240.8±19.8 mg/dL; $p<0.001$). Significant reductions in inflammatory markers were observed, with IL-6 showing significant improvement compared with metformin ($p=0.004$), while TNF-α reduction was comparable between groups. Improvement in lipid parameters, including increased HDL and reduced LDL cholesterol and triglycerides, was also observed.

Conclusion: *Mucuna pruriens* seed extract demonstrated significant improvement in glycaemic control, inflammatory cytokines and lipid abnormalities in experimental T2DM. The extract showed greater reduction in blood glucose and inflammatory markers compared with metformin, suggesting potential adjunct therapeutic value in managing metabolic disturbances associated with type 2 diabetes.

Keywords: Cytokines, High-fat diet, Hyperglycaemia, Insulin Resistance, Streptozotocin, Wistar rats

INTRODUCTION

The global prevalence of T2DM continues to rise, making it a major public health concern. According to the International Diabetes Federation, approximately 589 million adults (aged 20-79 years) were living with diabetes worldwide in 2024, and this number is projected to rise to around 853 million by 2050 [1]. India contributes significantly to this burden, with recent estimates from the Indian Council of Medical Research-India Diabetes (ICMR-INDIAB) study indicating that in 2021, over 101 million individuals had diabetes and nearly 136 million had prediabetes [2]. The rising prevalence is largely driven by urbanisation, sedentary lifestyle, obesity and population ageing, particularly in low and middle-income countries, posing a significant public health and economic burden [3].

Alterations in body weight, particularly excess adiposity, play a crucial role in the development of metabolic imbalance. Expansion of adipose tissue leads to dysregulated secretion of adipokines and free fatty acids, which impair insulin signalling and promote insulin resistance, thereby contributing to the onset of T2DM [4,5].

Reduced insulin sensitivity results in decreased peripheral glucose uptake and increased hepatic glucose output, leading to persistent hyperglycaemia. Chronic hyperglycaemia further exacerbates β-cell dysfunction through glucotoxic mechanisms, thereby accelerating disease progression [3,5]. Hence, improvement in glycaemic status remains a central objective in diabetes management.

In addition to metabolic dysregulation, T2DM is increasingly recognised as a state of chronic low-grade inflammation. Hyperglycaemia and oxidative stress activate intracellular signalling pathways, including Nuclear Factor-Kappa B (NF-κB), leading to increased production of pro-inflammatory cytokines such as TNF-α and IL-6. These cytokines impair insulin receptor signalling and further aggravate insulin resistance, thereby contributing to disease progression [6,7].

Dyslipidaemia is a common feature of T2DM and is characterised by elevated triglycerides, increased low-density lipoprotein cholesterol and reduced high-density lipoprotein cholesterol. Excess circulating free fatty acids and lipid intermediates such as diacylglycerol and ceramides accumulate in insulin-sensitive tissues, where they interfere with insulin signalling pathways by activating serine kinases and impairing insulin receptor substrate function. These alterations lead to reduced glucose uptake and increased hepatic glucose production, thereby worsening insulin resistance. In addition, lipid-induced oxidative stress and inflammatory responses further contribute to metabolic dysfunction, highlighting the importance of therapies targeting both metabolic and lipid disturbances [8].

Experimental induction of T2DM using a HFD followed by low-dose streptozotocin produces a model that closely resembles the pathophysiology of human T2DM. HFD induces insulin resistance by promoting obesity, lipotoxicity and metabolic imbalance, while low-dose streptozotocin causes partial pancreatic β-cell dysfunction. The combination of these two factors mimics the dual defects of insulin resistance and impaired insulin secretion observed in human disease. This model is therefore widely used for evaluating pharmacological agents targeting both metabolic and inflammatory pathways [9].

Mucuna pruriens (family *Fabaceae*) is a medicinal plant traditionally used in the management of metabolic disorders. Its seeds contain bioactive constituents such as L-DOPA, flavonoids and polyphenolic compounds. These constituents have been reported to exert antioxidant and anti-inflammatory effects and may improve glucose homeostasis and insulin sensitivity through modulation of oxidative stress and inflammatory pathways. In addition, L-DOPA may influence glucose metabolism via dopaminergic mechanisms, while flavonoids and polyphenols contribute to improved insulin action and reduced cytokine-mediated insulin resistance. Despite these potential mechanisms, comprehensive evaluation of the combined effects of *Mucuna pruriens* on glycaemic control, inflammatory mediators and lipid abnormalities in T2DM remains limited [10,11].

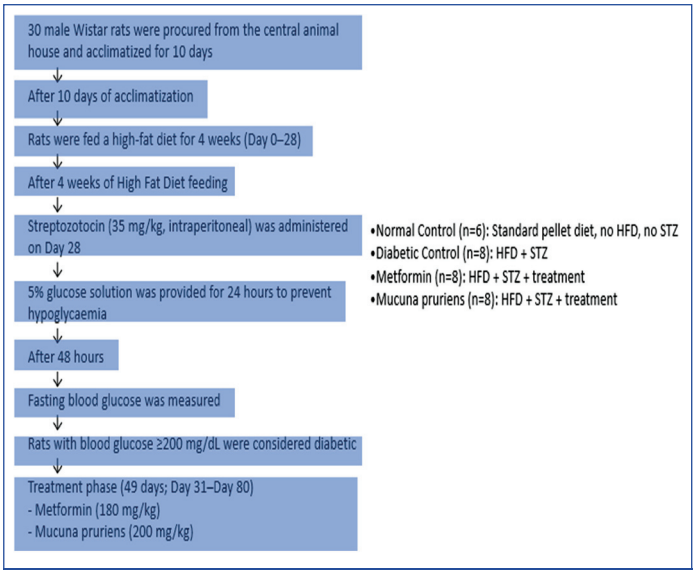
Previous experimental studies [12,13] suggest potential metabolic regulatory activity of *Mucuna pruriens*; however, comprehensive evaluation of its effects on body weight, glycaemic control, inflammatory mediators and lipid abnormalities remains limited. Therefore, the present study was undertaken to evaluate the effect of *Mucuna pruriens* seed extract on body weight, blood glucose levels, inflammatory markers and lipid profile in HFD and low-dose streptozotocin-induced T2DM in male Wistar rats. It was hypothesised that *Mucuna pruriens* seed extract would improve glycaemic control, reduce inflammatory cytokines and ameliorate dyslipidaemia in this experimental model.

MATERIALS AND METHODS

The present experimental animal study was conducted in the Department of Pharmacology, KAHER's Jawaharlal Nehru Medical College, Belagavi, Karnataka, India, from March 2024 to March 2025, after approval from the Institutional Animal Ethics Committee (CPCSEA Reg.No.: 627/PO/Re/S/02/CPCSEA). All procedures were performed in accordance with CPCSEA guidelines for care and use of laboratory animals [Table/Fig-1] [14].

Study Procedure

Experimental animals: A total of 30 adult male Wistar rats weighing 200-250 g was used in the study and were divided into four groups: normal control (n=6), diabetic control (n=8), metformin-treated (n=8) and *Mucuna pruriens*-treated (n=8). Animals were maintained under controlled environmental conditions (22±2°C, 12-hour light/dark cycle, relative humidity 50-60%) with free access to standard pellet diet and water. Animals were acclimatised for 10 days before initiation of the experiment [Table/Fig-1].



[Table/Fig-1]: Schematic representation of experimental design and treatment protocol.

Preparation of *Mucuna pruriens* extract: *Mucuna pruriens* seed extract was obtained from natural remedies, Bengaluru, India, in the form of a dried ethanolic extract powder. The use of ethanolic seed extract in experimental studies has been reported previously [15,16]. The extract was reconstituted in distilled water, which served as the vehicle to obtain the required concentration and administered orally using an oral gavage to the animals. The required dose was calculated based on body weight (mg/kg) and was freshly prepared prior to administration. The extract was procured from Natural Remedies, Bengaluru, India; however, detailed information regarding the extraction solvent and phytochemical standardisation (e.g., L-DOPA content) was not provided by the manufacturer, and no independent analysis was performed in the present study.

High-Fat Diet (HFD) Composition: The HFD was formulated to induce insulin resistance and consisted of powdered normal pellet diet (365 g/kg), lard (310 g/kg), casein (250 g/kg), cholesterol (10 g/kg), vitamin and mineral mix (60 g/kg), DL-methionine (3 g/kg), yeast powder (1 g/kg), and sodium chloride (1 g/kg). The composition was adapted from previously described HFD formulations used in experimental models of insulin resistance [9].

Induction of Type 2 Diabetes Mellitus (T2DM): Insulin resistance was induced by feeding rats a HFD for four weeks [9,17]. The normal control group was not administered the HFD. Animals in the normal control group received a standard pellet diet throughout the study period. Subsequently, streptozotocin (35 mg/kg, intraperitoneal) was administered after being freshly prepared in cold citrate buffer (pH 4.5) and used within 15 minutes of preparation [9,17]. To prevent early hypoglycaemia, 5% glucose solution was provided ad libitum for 24 hours after injection. Fasting blood glucose was measured after 48 hours and animals with glucose ≥200 mg/dL were considered diabetic [3,9].

Experimental timeline: The experimental timeline of the study is illustrated in [Table/Fig-1]. Treatment was initiated on Day 31 and continued for seven weeks (49 days), (corresponding to Day 31-Day 80 of the study).

Experimental groups and drug administration: A total of 30 adult male Wistar rats weighing 200-250 g was used in the study were randomly allocated into four experimental groups using a simple randomisation method: and were divided into four groups: normal control (n=6), diabetic control (n=8), metformin-treated (n=8) and *Mucuna pruriens* treated (n=8). All animals included in the study were accounted for throughout the experimental period, and no mortality or attrition was observed.

The number of animals in each group was determined based on Institutional Animal Ethics Committee approval, adhering to

the principle of reduction to minimise animal use, while ensuring adequate statistical power for analysis.

- Normal control-received vehicle;
- Diabetic control-received vehicle;
- Standard group-metformin 180 mg/kg orally [18];
- Test group- *Mucuna pruriens* seed extract 200 mg/kg orally [19,20].

The dose of metformin (180 mg/kg) was derived based on conversion of the maximum human therapeutic dose to the equivalent rat dose using the Allometric scaling based on Body Surface Area (BSA) normalisation method [18]. The dose of *Mucuna pruriens* (200 mg/kg) was selected based on previously published experimental studies demonstrating its antidiabetic, anti-inflammatory, and metabolic effects in rodent models, and was considered an effective dose within the commonly reported therapeutic range [19,20].

All treatments were administered once daily for seven weeks using oral gavage [21]. The treatment duration of seven weeks was selected to allow adequate evaluation of glycaemic, inflammatory and lipid parameters and is within the range of treatment durations previously employed in experimental studies of *Mucuna pruriens* and diabetic rodent models. The dosing volume was maintained at 10 mL/kg body weight, and distilled water was used as the vehicle [22].

Blood sample collection and biochemical analysis: At the end of the experimental period, animals were fasted overnight and anesthetised under Thiopentone sodium (40mg/kg IP). Blood samples were collected by cardiac puncture for estimation of lipid profile and inflammatory markers. Measurements were performed on Day 49 of treatment (corresponding to Day 80 of the study).

Following blood collection, animals were euthanised by overdose of thiopentone sodium 120 mg/kg Intra peritoneal route.

Measurement of body weight: Body weight was recorded at baseline, after diabetes confirmation and weekly thereafter using a digital weighing balance.

Estimation of fasting blood glucose: blood samples were collected from tail vein after overnight fasting and glucose levels were measured using glucometer method [23].

Estimation of lipid profile: At the end of the treatment period, serum total cholesterol, triglycerides, High-Density Lipoprotein-cholesterol and Low-Density Lipoprotein-cholesterol were determined using enzymatic colorimetric kits [24].

Estimation of inflammatory markers: serum TNF-α, IL-6 levels was estimated using Enzyme-Linked Immunosorbent Assay (ELISA) kits according to manufacturer protocol [25].

STATISTICAL ANALYSIS

Data were expressed as mean±Standard Deviation (SD). For parameters measured repeatedly over time (body weight and fasting blood glucose), statistical analysis was performed using Two-way Repeated Measures ANOVA (RM ANOVA) to evaluate the effects of time and treatment, followed by Tukey's multiple comparisons post-hoc test. For parameters measured at a single time point (serum inflammatory markers including TNF-α and IL-6, and lipid profile parameters including total cholesterol, triglycerides, LDL, and HDL), statistical analysis was performed using One-way ANOVA followed by Tukey's post-hoc test. A p-value of <0.05 was considered statistically significant. All analyses were performed using GraphPad Prism version 8.0.1 (GraphPad Software, USA).

RESULTS

Body weight: Baseline body weight was comparable between normal and diabetic control groups (p=0.4794). Following induction, diabetic control animals showed significant weight loss compared to normal control on Day 21 (p<0.0001) and Day 49 of treatment (Day 80) (p<0.0001).

Metformin significantly improved body weight compared to diabetic control on Day 28 (p=0.0026) and Day 49 of treatment (Day 80) (p<0.0001), but not on Day 21 (p=0.0954).

Mucuna pruriens (MP200) significantly improved body weight on Day 28 (p=0.0026), Day 21 (p=0.0033), and Day 49 of treatment (Day 80) (p<0.0001). No significant difference was observed between metformin and MP200 (p=0.4461) [Table/Fig-2].

Body weight (g) at different time points				
Groups	Day 0	28th day after HFD	21st Day of treatment	Day 49 of treatment
Normal control	216.7±10.8	236.7±10.8	241.7±10.8	246.7±10.8
Diabetic control	208.8±8.3 *ns (p=0.4794)	228.8±8.3 *ns (p=0.4794)	194.4±9.4* *(p<0.0001)	184.4±9.4* *(p<0.0001)
Metformin	198.4±13.6 *ns (p=0.0662) #ns (p=0.303)	203.5±12.8*# *(p=0.0011) #(p=0.0026)	207.3±10.7* *(p=0.0005) #ns (p=0.0954)	212.9±7.7*# *(p=0.0006) #(p<0.0001)
Mucuna pruriens 200mg/kg	204.1±9.9 *ns (p=0.1787) #ns (p=0.746) $ns (p=0.7691)	210.3±8.2*# *(p=0.0033) #(p=0.0026) $ns (p=0.6058)	213.3±7.6*# *(p=0.0021) #(p=0.0033) $ns (p=0.5856)	218±5.5*# *(p=0.0025) #(p<0.0001) $ns (p=0.4461)

[Table/Fig-2]: Effect of *mucuna pruriens* seed extract on body weight (g) at different time. Points in High-Fat Diet (HFD) and Low-Dose Streptozotocin-Induced Diabetic Rats
Values are expressed as Mean±SD (n=6 in normal control and n=8 in all other groups). Statistical analysis done by using Two-way repeated measures ANOVA followed by Tukey's post-hoc test.
(*) indicates p<0.05-groups compared with Normal Control (NC);
(#) indicates p<0.05-treatment groups compared with Diabetic Control;
($) indicates p<0.05-*Mucuna pruriens* 200mg/kg compared with Metformin.
ns indicates- Non-significant values
Day 0 and Day 28 after HFD represent pre-treatment measurements. Any statistically significant differences observed at these time points reflect baseline biological variability and not treatment effects.

Blood Glucose Levels

Baseline glucose levels were comparable (p=0.7731). Diabetic control animals showed significantly elevated glucose levels at 48 hours, Day 21, and Day 49 of treatment (Day 80) compared to normal control (p<0.0001 for all).

Metformin significantly reduced glucose levels compared to diabetic control at 48 hours (p<0.0001), Day 21 (p=0.0013), and Day 49 of treatment (Day 80) (p<0.0001).

MP200 also significantly reduced glucose levels at 48 hours (p=0.0005), Day 21 (p<0.0001), and Day 49 of treatment (Day 80) (p<0.0001). MP200 showed a significantly greater reduction compared to metformin at Day 21 and Day 49 of treatment (Day 80) (p<0.0001 for both) [Table/Fig-3].

Blood Glucose Levels (mg/dL) at different Time Points				
Groups	Day 0	After 48 hours of induction	21st day of treatment	Day 49 of treatment
Normal control	98.5±2.4	113.2±1.5	116.2±1.5	116.7±2.2
Diabetic control	99.8±2.4 *ns(p=0.7731)	291±3.5* *(p<0.0001)	295.3±3.2* *(p<0.0001)	311.1±3.8 * *(p<0.0001)
Metformin	96.3±5.5 *ns(p=0.74) #ns(p=0.4032)	317.3±10.9*# *(p<0.0001) #(p<0.0001)	270±11.3*# *(p≤0.0001) #(p=0.0013)	240.8±19.8*# *(p<0.0001) #(p<0.0001)
Mucuna pruriens 200mg/kg	96.9±12.5 *ns(p=0.9831) #ns(p=0.9165) $ns(p=0.9992)	311.8±22.8*# *(p<0.0001) #(p=0.0005) $ns(p=0.9251)	190.9±16.3*#$ *(p<0.0001) #(p<0.0001) $(p<0.0001)	167.5±15.4*#$ *(p=0.0001) #(p<0.0001) $(p<0.0001)

[Table/Fig-3]: Effect of *mucuna pruriens* seed extract on serum fasting blood glucose levels (mg/dL) in High-Fat Diet (HFD) and low-dose streptozotocin-induced diabetic rats at different time points.
Values are expressed as mean±SD (n=6 in normal control and n=8 in all other groups). Statistical analysis done by using Two-way repeated measures ANOVA followed by Tukey's post-hoc test.
(*) indicates p<0.05 - groups compared with normal control;
(#) indicates p<0.05 – treatment groups compared with diabetic control;
($) indicates p<0.05-*Mucuna pruriens* 200 mg/kg compared with Metformin.
ns indicates- Non-significant values
Differences observed after diabetes induction occurred prior to treatment initiation and reflect inter-animal variability in response to HFD-STZ induction rather than treatment effects.

Inflammatory Markers

TNF-α and IL-6 levels were significantly elevated in diabetic control compared to normal control (p<0.001).

Both metformin and MP200 significantly reduced TNF-α compared to diabetic control (p<0.001), with no significant difference between them (p=1.000).

For IL-6, both treatments reduced levels compared to diabetic control (metformin: p=0.001; MP200: p<0.001). MP200 showed a significantly greater reduction compared to metformin (p=0.004) [Table/Fig-4].

Serum Inflammatory markers (pg/mL) on day 49 of the treatment		
Groups	**Serum TNF-α**	**Serum IL-6**
Normal control	36.3±3	26.8±3.3
Diabetic control	138.3±24.6* *(p<0.001)	383.2±48* *(p<0.001)
Metformin	64.4±34.3# *ns(p=0.396) #(p<0.001)	290±29.5*# *(p<0.001) #(p= 0.001)
Mucuna pruriens 200	43.5±12.9# *ns(p=0.396) #(p<0.001) $ns(p=1.000)	207.7±33.8*#$ *(p<0.001) #(p<0.001) $(p=0.004)

[Table/Fig-4]: Effect of *mucuna pruriens* seed extract on serum inflammatory markers (TNF-α, IL-6) in High-Fat Diet (HFD) and low-dose streptozotocin-induced diabetic rats on day 49 of the treatment.
Values are expressed as mean±SD (Normal Control, n=6; other groups, n=8). Statistical analysis done by using One-way ANOVA followed by Tukey's post-hoc test.
(*) indicates p<0.05-groups compared with normal control;
(#) indicates p<0.05-treatment groups compared with diabetic control;
($) indicates p<0.05-*Mucuna pruriens* 200mg/kg compared with Metformin.
ns indicates- Non-significant values.

Lipid Profile

Diabetic control animals exhibited significant dyslipidaemia compared to normal control (p<0.001 for all parameters).

Both metformin and MP200 significantly improved lipid parameters compared to diabetic control (p<0.001 for all).

Compared to metformin, MP200 showed no significant difference in total cholesterol (p=1.000) and HDL (p=1.000), but produced significantly greater reductions in LDL (p<0.001) and triglycerides (p<0.001) [Table/Fig-5].

Serum Lipid Profile Parameters(mg/dL) on day 49 of the treatment				
Groups	**Serum HDL**	**Serum LDL**	**Serum triglycerides**	**Serum cholesterol**
Normal control	28±5.5	14±3.6	73.5±5	85.5±8.7
Diabetic control	16.9±2* *(p≤0.001)	72±1.9* *(p<0.001)	191.6±2.3* *(p<0.001)	175.9±9.1* *(p<0.001)
Metformin	24.5±2.8# *ns(p=1.000) #(p=0.002)	58.9±5.5*# *(p<0.001) # (p<0.001)	162.5±5.3*# *(p<0.001) # (p<0.001)	127.5±10.4*# *(p<0.001) # (p<0.001)
Mucuna pruriens 200	26.5±3.9# *ns(p=1.000) #(p<0.001) $ns(p=1.000)	47±4.9*#$ *(p<0.001) #(p<0.001) $ (p<0.001)	144.6±3.2*#$ *(p<0.001) #(p<0.001) $ (p<0.001)	132±10.6*# *(p<0.001) #(p<0.001) $ns(p=1.000)

[Table/Fig-5]: Effect of *mucuna pruriens* seed extract on lipid profile parameters (HDL, LDL, Triglycerides, and Total Cholesterol) in High-Fat Diet (HFD) and low-dose. Streptozotocin-Induced diabetic rats on day 49 of the treatment
Values are expressed as mean±SD (Normal control, n=6; other groups, n=8). Statistical analysis done by using One-way ANOVA followed by Tukey's post-hoc test.
(*) indicates p<0.05-groups compared with normal control;
(#) indicates p<0.05-treatment groups compared with diabetic control;
($) indicates p<0.05-*Mucuna pruriens* 200mg/kg compared with Metformin.
ns indicates- Non-significant values.

DISCUSSION

The present study evaluated the metabolic and inflammatory effects of *Mucuna pruriens* seed extract in a HFD and low-dose streptozotocin-induced model of T2DM. The findings demonstrated improvement in body weight, glycaemic status, inflammatory cytokines and lipid parameters following treatment.

Reduction in body weight observed in the diabetic control group following diabetes induction is a recognised feature of uncontrolled diabetes resulting from impaired glucose utilisation and increased protein catabolism. Similar alterations in body weight have been reported in experimental diabetes models induced by HFD and streptozotocin administration [9,17]. In the present study, treatment with *Mucuna pruriens* partially prevented the decline in body weight compared with diabetic controls. Improvement in body weight may reflect enhanced metabolic efficiency and better utilisation of circulating nutrients following treatment [10,21]. Although the diabetic groups were fed a HFD, they subsequently developed diabetes following streptozotocin administration. Diabetes is associated with reduced body weight due to impaired glucose utilisation and increased breakdown of fat and protein stores. As a result, the diabetic animals showed weight loss despite prior exposure to a HFD. In contrast, the Normal Control group did not develop diabetes and therefore continued normal growth throughout the study period, resulting in a higher body weight compared to the diabetic groups.

Persistent hyperglycaemia in the diabetic control group confirmed successful induction of experimental diabetes. Administration of *Mucuna pruriens* resulted in a marked reduction in fasting blood glucose levels and produced greater glycaemic improvement compared with metformin in the present under the present experimental conditions. Improved glycaemic control in experimental diabetes has been associated with increased peripheral glucose uptake and reduced hepatic glucose production [4,5]. Previous experimental studies have also demonstrated antihyperglycemic activity of *Mucuna pruriens* seed extract in diabetic animal models, supporting its potential role in glucose regulation [10,19,20].

Type 2 diabetes is increasingly recognised as a chronic inflammatory condition characterised by elevated cytokines such as TNF-α and IL-6. These mediators impair insulin receptor signalling and contribute to insulin resistance [6,8]. In the present study, diabetic animals showed markedly elevated inflammatory markers, whereas treatment with *Mucuna pruriens* reduced circulating cytokine levels. This observation suggests attenuation of inflammatory processes associated with metabolic dysfunction. The anti-inflammatory effects of plant-derived compounds rich in flavonoids and polyphenols have been reported to improve metabolic homeostasis in experimental diabetes [6,10].

The diabetic control group exhibited characteristic diabetic dyslipidaemia, including elevated LDL cholesterol, triglycerides and total cholesterol with reduced HDL cholesterol. Such lipid abnormalities are frequently observed in insulin-resistant states and contribute to increased cardiovascular risk [8]. Treatment with *Mucuna pruriens* improved lipid parameters compared with diabetic controls, indicating favourable effects on lipid metabolism. Similar lipid-lowering effects of plant-derived therapeutic agents have been reported in experimental studies and may be related to improved insulin sensitivity and regulation of hepatic lipid synthesis [10,19]. The metabolic improvements observed with MP200 may be attributed to its bioactive constituents, particularly L-DOPA and polyphenolic compounds. L-DOPA, a precursor of dopamine, may influence glucose homeostasis through dopaminergic signalling, which has been associated with modulation of insulin secretion, peripheral glucose utilisation, and hepatic glucose output [26].

In parallel, the reduction in pro-inflammatory cytokines (IL-6 and TNF-α) suggests that *Mucuna pruriens* exerts anti-inflammatory effects, potentially through down-regulation of NF-κB-mediated signalling pathways [8,19]. NF-κB activation is a key driver of cytokine production and insulin resistance; thus, its inhibition may improve insulin sensitivity and contribute to the observed glycaemic improvement. The concurrent improvements in lipid parameters (LDL and triglycerides) may be secondary to enhanced insulin action and modulation of hepatic lipid metabolism, mechanisms that are known to contribute to improved lipid homeostasis in diabetes [12,27].

Collectively, these findings suggest that *Mucuna pruriens* may exerts antidiabetic effects primarily through dopaminergic regulation

and suppression of inflammatory signalling, which is consistent with the reductions in blood glucose, IL-6, TNF-α, and atherogenic lipids observed in the present study. Improvement in glycaemic status along with reduction in inflammatory cytokines and correction of lipid abnormalities indicates that the extract may influence several pathways involved in metabolic regulation.

Limitation(s)

The present study was conducted in an experimental animal model, and therefore direct extrapolation of the findings to humans should be made with caution. The study was limited by the absence of histopathological evaluation, which could have provided additional insight into tissue-level changes. Furthermore, detailed mechanistic investigations, including molecular or signalling pathway analyses, were not performed, limiting the understanding of the underlying mechanisms of action. In addition, the duration of the present study was relatively short and may not fully reflect long-term metabolic and inflammatory changes associated with chronic diabetes. Further studies incorporating histopathological assessment, mechanistic evaluation, and longer duration are required to better establish the therapeutic potential of *Mucuna pruriens*.

CONCLUSION(S)

Mucuna pruriens seed extract (200 mg/kg) demonstrated improvement in body weight, glycaemic control, inflammatory cytokines and lipid parameters in HFD and low-dose streptozotocin-induced T2DM in rats. The extract produced greater reduction in blood glucose, TNF-α and IL-6 levels compared with the diabetic control group and showed favourable effects on lipid profile. These findings suggest that *Mucuna pruriens* may have potential as an adjunct therapeutic agent in the management of metabolic disturbances associated with T2DM.

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