Suppression of Type-II Diabetes with Dyslipidemia and Nephropathy by Peels of *Musa cavendish* Fruit

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Abstract *Musa cavendish*, peels has local and traditional use to promote wound healing, hyperglycemia, ulceration etc. The present work investigated the lipid lowering; nephroprotective and glucose lowering properties of ethanolic extract of peels of *Musa cavendish* (EMC) in alloxan-induced diabetic rats. The EMC 250, 500 and 1000 mg/kg/day and the vehicle were administered orally to alloxan-induced diabetic rats (n = 6) for 3 weeks. Changes in plasma glucose, lipid profile along with kidney function before and after treatment with EMC were recorded. The ethanolic extract of peels of *Musa cavendish* reduced blood glucose, serum triglyceride, cholesterol, LDL cholesterol and creatinine levels and improvement in body weight, liver glycogen, serum HDL cholesterol, serum albumin and total protein level when compared with untreated rats. *Musa cavendish* has lipid lowering, nephroprotective and antidiabetic property by regulating glucose uptake in the liver and muscles by restoring the intracellular energy balance.

Keywords *Musa cavendish* · Antidiabetic · Nephropathy · Dyslipidemia

Introduction

Diabetes mellitus (DM) is a distressing chronic disease and has become a serious problem worldwide; the number of diabetic patients around the world is increasing continuously, with an estimated 371 million cases in 2012, which is expected to reach 552 million by 2030 [1, 2]. DM comprises a group of chronic disorders characterized by hyperglycemia due to lack of insulin secretion, which disturbs metabolism of carbohydrate, lipid and protein [3]. If the situation persists, this condition can lead to cardiovascular diseases, blindness, kidney failure and lower limb amputation, etc. Type 2 DM develops due to either insufficient insulin synthesis or an inability of cells to respond to secreted insulin and leads to glucose build-up in the blood.

Dyslipidemia in diabetes has been reported to be largely due to insulin resistance [4–7]. Diabetic dyslipidemia results in the accumulation of excess free fatty acids, which are converted to triglycerides in the liver. This stimulates the secretion of very low-density lipoprotein cholesterol and apoprotein B and correlates well with hepatic fat accumulation [8]. The consequence of fat accumulation increases small, dense low-density lipoprotein cholesterol (LDL-C) and triglycerides levels and decreases high-density lipoprotein cholesterol (HDL-C), which contributes to cardiovascular risk in diabetes. This is evident as high triglyceride; LDL-C levels in conjunction with low HDL level contribute immensely to accelerated atherosclerosis in diabetes [9].

Diabetic nephropathy (DNP) is a major cause of illness and premature death in diabetic patients, largely through accompanying cardiovascular diseases and end-stage renal failure [10]. Diabetes induced by alloxan in rats results in the development of nephropathy similar to early stage clinical DNP [11].

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At present, a number of hypoglycemic agents, including insulin and oral drugs such as sulphonyl ureas, biguanides and glycosidase inhibitors are popularly used in clinics to keep blood glucose at normal level. Unexpectedly, these agents produce serious side effects in the clinical application, such as weight gain, gastrointestinal disturbances, edema, hypoglycemia and insulin resistance [12]. Therefore, it is important to discover alternative therapies that may have less or no side effects [13]. Medicinal plants used by folk medicinal healers are successfully used in many countries to control diabetes, and have become the most important sources for seeking a safe, specific and effective hypoglycemic agent [14, 15]. Moreover, many hypoglycemic components have been obtained from the medicinal plants, mainly including flavonoids, alkaloids, polysaccharides, saponins, terpenoids and unsaturated fatty acids [16]. *Aegle marmelos*, *Allium cepa*, *Aloe vera*, *Azadirachta indica*, *Gymnema sylvestre*, *Momordica charantia*, *Ocimum sanctum*, *Nigella sativa*, *Ocimum sanctum*, *Panax quinquefolius*, *Salacia reticulata*, *Silybum marianum* and *Trigonella foenum-graecum* have shown hypoglycemic and, in some cases, hypolipidemic activities in diabetes [17].

Banana (*Musa cavendish*) is most commonly consumed edible fruit due to its high nutritional contents. In the Indian system of folk medicine, banana is used to treat various diseases and disorders like wound, hyperglycemia, ulcer, dysentery etc. [18]. *Musa cavendish* is called as ‘Sunkela’ in local language (Son-Gold, Kela-Banana; named due to its golden yellow colour and pure quality aspects), having small in size but high in nutritional value due to high carbohydrate contents. Several flavonoids and related compounds (leucocyanidin, quercetin and its 3-*O*-galactoside, 3-*O*-glucoside, and 3-*O*-rhamnosyl glucoside, galocatechin) have been isolated from the unripe pulp and peel of plantain [19–21]. These phytochemicals have shown a protective action against diseases which involve oxidative stress like diabetes, hepatic disorders. Many Indian people residing in rural and tribal areas have a habit of eating the inner peel, in addition to the pulp of banana fruit. One of the objectives of the present investigation was to find out the benefit of eating the inner peel of banana fruit, especially in diabetes. Hence, the present investigation was aimed to evaluate potential of *Musa cavendish* inner fruit peel in the treatment of diabetes and its complications by using alloxan-induced diabetic rats.

Materials and Methods

Experimental Animals

Albino rats of Wistar strain weighing 180–200 g were received from the National Institute of Nutrition (NIN),

Hyderabad, India. Animals of either sex were housed under standard laboratory conditions of 22 ± 3 °C temperature and relative humidity 30 % and 12 h light and dark cycle maintained, free access to standard pellet diet and water ad libitum was allowed to animals. The experimental protocol was approved by the Institutional Animal Ethics Committee (1613/PO/a/12/CPCSEA).

Acute Toxicity Study

The LD50 of the peel extract was tested to determine the safety of the agent according to the guidelines set by the Organization for Economic Cooperation and Development (OECD) No. 423 [22]. The study was carried out in two phases. In the first phase, nine mice were randomized into three groups of three mice per group and administered 100, 600 and 1000 mg/kg of the extract orally. The animals were observed for the first 4 and 24 h for signs of toxicity and mortality. The results of this phase informed the choice of doses for the second phase, in which 2000, 3000 and 5000 mg/kg were administered to another set of three mice per group and these animals were observed for signs of toxicity and mortality for 72 h.

Collection and Authentication of the Plant Materials

Fresh unripe fruits of *Musa cavendish* Linn. (Musaceae) was collected from Satara District, Maharashtra state of India. The specimen was authenticated at Science College Nanded, Maharashtra state, India (25-9/12).

Preparation of Extract

The inner peel of *Musa cavendish* was scraped and shed dried and latter powdered. This powder was then defatted with petroleum ether and extracted with ethanol using Soxhlet extraction. It was then filtered and the solvent was evaporated under vacuum. The ethanolic extract of inner peels of *Musa cavendish* (EMC) stored in a cool place for further use.

Experimental Induction of Diabetes in Rats

Albino Wistar rats of either sex having body weight 180–200 g were selected for the study. The animals were fed with standard diet and water ad libitum. The rats were injected with alloxan monohydrate with a pre-standardized dose of 140 mg/kg i.p. The rats were kept for the next 24 h on 5 % glucose solution to prevent hypoglycemia [23]. After 48 h, rats with plasma glucose above 200 mg/dl (as the sign of establishment of diabetes) were used for further study.

Experimental Design

Antidiabetic Study in Alloxan-Induced Diabetic Rats

After administration of alloxan, blood glucose level at the second day of initial dose indicated the presence of diabetes. Then the animals were divided into different groups and their fasting blood glucose levels were measured. The treatment was started on the same day considering it as 1st day of the study [24–26].

Further assessment of blood glucose level was carried out on 0th, 7th, 14th and 21st days of the study. The treatment protocol design using 42 rats of either sex (36 diabetic surviving rats and 6 normal) was divided into six groups (n = 6) is given below:

Group I: Normal control, received suspension of 1 % gum acacia (1 ml/kg, p.o.).

Group II: Diabetic control, received suspension of 1 % gum acacia solution (1 ml/kg, p.o.)

Group III: Glim, animals of this group received glimepiride (0.09 mg/kg, p.o.).

Group IV: Losar, animals of this group received losartan (2 mg/kg, p.o.).

Group V: EMC 250; received suspension of ethanolic extract of *Musa cavendish* 250 mg/kg, p.o. in 1 % gum acacia.

Group VI: EMC 500; received suspension of ethanolic extract of *Musa cavendish* 500 mg/kg, p.o. in 1 % gum acacia.

Group VII: EMC 1000; received suspension of ethanolic extract of *Musa cavendish* 1000 mg/kg, p.o. in 1 % gum acacia.

Evaluation of Blood Glucose Level

Evaluation of the antidiabetic effect of EMC was performed by blood glucose estimation which was carried out at 0th, 7th, 14th and 21st day of treatment. Determination of blood glucose level was performed using (glucose oxidase/peroxidase) standard diagnostic kits from Crest Diagnostic, India.

Evaluation of Glycogen Content in Liver

In order to find out the effect of EMC on the storage of glucose, glycogen content of liver was estimated at the end of the experimental period. Animals were sacrificed with an overdose of urethane. The liver was removed and glycogen content was measured according to the standard method [27].

Change in Body Weight

Effect of EMC on body weight in diabetic rats was assessed throughout the study period, i.e. at 0th, 7th, 14th and 21st day of treatment [28].

Assessment of Dyslipidemic Activity and Cardiovascular Risk Indices

Effect of EMC on lipid levels of rats was determined by estimating all lipid parameters. Blood was collected at the end of the study period, i.e. on the 21st day of treatment and serum triglyceride, cholesterol, LDL cholesterol, and HDL cholesterol were determined by using standard diagnostic kits from Crest Diagnostics, India.

Cardiac index [29], Atherogenic index [30] and Coronary artery index, which are termed as cardiovascular risk indices, were calculated as per the following formulae.

$$\text{Cardiac index: } \frac{\text{Total cholesterol}}{\text{HDL cholesterol}}$$

$$\text{Atherogenic index: } \frac{(\text{Total cholesterol})}{(\text{HDL cholesterol})} - \text{HDL cholesterol}$$

$$\text{Coronary artery index: } \frac{\text{LDL Cholesterol}}{\text{HDL cholesterol}}$$

Assessment of Nephroprotective Effect

Biochemical Analysis

A possible effect of EMC on diabetic kidney disease was assessed by using estimation of various serum markers like serum creatinine, albumin and total protein at the end of the study period, i.e. on the 21st day of treatment. Losar group was not considered for kidney function. Estimation was carried out by using standard diagnostic kits from Crest Diagnostic, India.

Histopathological Study

Histological study of effect of EMC on the kidney was performed on the 21st day of treatment to confirm the possible nephroprotective action. Kidneys were removed from the sacrificed animals, washed with distilled water and kept in 10 % formalin solution. Kidney tissues were fixed in 10 % formalin and embedded in paraffin. Rotary microtome was used to cut the kidney tissues into 5 µm thick sections. Later on, sections were treated with xylene and ethanol for deparaffinization and further stained with hematoxylin & eosin (H&E) and then examined microscopically.

Statistical Analysis

The data obtained was treated statistically by using analysis of variance (ANOVA) followed by Dunnett's test to detect any significant difference among different means, with level of significance set at $p < 0.05$. The results were expressed as mean $\pm$ SEM.

Results

Acute Toxicity

The acute oral toxicity test showed normal behavior of the treated animals. There was no mortality observed at a high dose of 5000 mg/kg. Hence the 1/10th of the safer dose was selected as a therapeutic dose (250, 500 and 1000 mg/kg p.o.).

Plasma Glucose Estimation

Figure 1 depicts the effect of ethanolic extract of *Musa cavendish* on plasma glucose level in alloxan-induced diabetic rats. Alloxan-induced diabetic animals demonstrated significant rise in plasma glucose level ($p < 0.01$) while, diabetic animals treated with EMC 250, 500 and 1000 showed a significant reduction in plasma glucose at 14th ($p < 0.05$) and 21st day ($p < 0.01$).

Lipid Profile

Diabetic animals demonstrated significant escalation in the levels of serum triglyceride, serum cholesterol, serum LDL cholesterol and drop in the level of serum HDL cholesterol ($p < 0.01$). Diabetic animals treated with both EMC-500 and EMC-1000 depicted significant reduction in the serum triglyceride level ($p < 0.05$) while, only EMC-1000 demonstrated marked reduction ($p < 0.05$) in the level of

serum cholesterol, serum LDL cholesterol ($p < 0.01$) and improvement in the serum HDL cholesterol level (Fig. 2).

Cardiovascular Risk Indices

Diabetic animals revealed a significant increase, whereas treatment with EMC 250, 500 and 1000 mg/kg, p.o. demonstrated a marked reversal in atherogenic, cardiac and coronary artery indices when compared with diabetic animals (Fig. 3). These results are comparable to that of the groups treated with glimepiride and losartan.

Liver Glycogen Analysis

Figure 4 represents the effect of EMC on liver glycogen content. Untreated diabetic animals demonstrated significant decrease in the liver glycogen content in comparison to control group. EMC-500 and EMC-1000 produced a significant rise in liver glycogen level in diabetic animals.

Changes in Body Weight

Figure 5 represents the changes in the body weight of alloxan-induced diabetic rats during the 21 day study period. During this period, untreated diabetic animals demonstrated marked reduction in the body weight (-21.62 ± 2.38 g). Treatment with EMC showed significant improvement in body weight at the dose of 500 and 1000 mg/kg, p.o. as indicated by the small reduction in body weight (1.32 ± 0.86 g) as compared to diabetic animals and which is comparable to glimepiride-treated animals.

Kidney Function Performance Biochemical Analysis

Figure 6 depicts the effect of EMC on various kidney markers. Diabetic animals showed a significant increment in serum creatinine and fall in serum albumin and total

Fig. 1 Effect of EMC on plasma glucose level in alloxan-induced diabetic rats. Bars carrying different superscripts for each profile are significantly different (* $p < 0.05$; ** $p < 0.01$). Values are

statistically significant at $p < 0.05$. Significance determined by ANOVA followed by Dunnett's test. Diabetic animals treated with EMC 1000 showed significant activity at day 14 and day 21

Fig. 2 Effect of EMC on serum triglyceride, cholesterol, LDL and HDL cholesterol parameters in alloxan-induced diabetic rats after oral administration for 21 days. Values are Mean $\pm$ SEM. Bars carrying different superscripts for each profile are significantly different (* $p < 0.05$; ** $p < 0.01$). Values are statistically significant at ($p < 0.05$). Significance determined by ANOVA followed by

Dunnnett's test. EMC-500 demonstrated significant decrease in serum triglyceride and LDL cholesterol level. EMC-1000 depicted significant reduction in serum triglyceride, cholesterol and LDL cholesterol, moreover, significant improvement in serum HDL cholesterol level was seen

Fig. 3 Effect on atherogenic, cardiac and coronary artery index in alloxan-induced diabetic rats after oral administration of EMC for 21 days. Values are Mean $\pm$ SEM. Bars carrying different superscripts for each profile are significantly different (* $p < 0.05$;

** $p < 0.01$). Values are statistically significant at ($p < 0.05$). Significance determined by ANOVA followed by Dunnnett's test. All the treatment groups demonstrated significant decrease in atherogenic, cardiac and coronary artery index

protein levels ($p < 0.01$). Diabetic animals treated with EMC-500 and EMC-1000 demonstrated a marked decrease in the serum creatinine level ($p < 0.01$), which is comparable to glimepiride. Similarly EMC-500 and EMC-1000 also caused a marked increase in serum albumin level in diabetic animals, however, only EMC-1000 demonstrated a remarkable rise in the level of serum total protein ($p < 0.01$).

Histopathological Study

Figure 7 summarizes the effect of EMC and glimepiride on renal structures of rats. Alloxan (untreated diabetic animals) caused significant marks of glomerulosclerosis (nephritis) and hyalinization. Similar to that of glimepiride-

treated rats, diabetic animals treated with EMC-500 and EMC-1000 demonstrated significant improvement from glomerulosclerosis and hyalinization showing almost normal architecture.

Discussion

Blood Glucose

Uncontrolled DM is mostly associated with complications like dyslipidemia and co-morbidities such as obesity, nephropathy and hypertension. In the present study, the antidiabetic potential of EMC was assessed in alloxan-induced diabetes and its complications. Classical symptoms

Fig. 4 Effect on liver glycogen content in alloxan-induced diabetic rats after oral administration of ethanolic extract of *Musa cavendish* for 21 days. Values are Mean $\pm$ SEM. Bars carrying different superscripts for each profile are significantly different (* $p < 0.05$; ** $p < 0.01$). Values are statistically significant at ($p < 0.05$). Significance determined by ANOVA followed by Dunnett's test. EMC-500 and EMC-1000 demonstrated a significant increase in liver glycogen content when compared with diabetic animals

of DM (type 2) were witnessed in untreated alloxan-induced diabetic rats, i.e. hyperglycemia, severe loss of body weight etc. as compared to the control group. Treatment with EMC for 21 days demonstrated a significant reduction ($p < 0.01$) in elevated levels of plasma glucose in diabetic

rats. This is indicative of its antihyperglycemic activity. Further, its beneficial effects in DM were witnessed from its effect on glycogen content (discussed separately).

Anti-hyperglycemic effect of EMC may be due to the presence of phyto-constituents like tannins, saponins and flavonoids [20, 21]. The phytochemical investigation of EMC showed the presence of tannins, alkaloids and flavonoids (i.e. gallic acid) [19, 31]. These substances are frequently implicated in its anti-hyperglycemic effects [32–35]. Gallic acid is one of the active constituents of *Musa cavendish* inner peel which increases peripheral glucose utilization [19, 36]. Inhibition of dietary absorption of glucose or improvement in insulin production from pancreatic beta cells may be responsible for antidiabetic effect. Fully mature banana, which hold the delphinidin, anthocyanins and cyaniding, and catecholamine may also contribute to the antidiabetic effect [37].

Dyslipidemia

Chronic DM is mostly associated with derangement of lipid metabolism and enhanced activity of the hormone

Fig. 5 Effect of EMC on changes in body weight after oral administration for 21 days in alloxan-induced diabetic rats. Values are Mean $\pm$ SEM. Lines carrying different superscripts for each profile are significantly different (* $p < 0.05$; ** $p < 0.01$). Values are

statistically significant at ($p < 0.05$). Significance was determined by ANOVA followed by Dunnett's test. Significant improvement in body weight was seen in the animals treated with EMC-500 and EMC-1000

Fig. 6 Effect on serum creatinine, albumin and total protein level in alloxan-induced diabetic rats after oral administration of EMC for 21 days. Values are Mean $\pm$ SEM. Bars carrying different superscripts for each profile are significantly different (* $p < 0.05$; ** $p < 0.01$). Values are statistically significant at ($p < 0.05$).

Significance determined by ANOVA followed by Dunnett's test. EMC-500 demonstrated significant decrease in serum creatinine and an increase in serum albumin level. EMC-1000 revealed significant reduction in serum creatinine and improvement in serum albumin and total protein level when compared with the diabetic animals

Fig. 7 Effect on histology of kidney in alloxan-induced diabetic rats after oral administration of ethanolic extract of *Musa cavendish* for 21 days. Photomicrographs revealing the renal structures of rats at $\times 100$. **a** Control group kidney, notice the normal appearance of the glomerular capillaries (arrow), showing normal epithelium. **b** Diabetic-control group kidney, the glomerular capillaries are widened, irregular (arrow). Tubular epithelium is also affected (arrowhead). **c** D + Glim group kidney, the histological features are relatively improved compared to non-treated diabetic group. The glomerular

capillaries retain their normal size and appearance (arrow). **d** D + EMC-250 group kidney showing mark of glomerulosclerosis (nephritis) and hyalinization (arrow). **e** D + EMC-500 group kidney, the glomerular capillaries are widened, irregular showing mild inflammation (arrow). **f** D + EMC-1000 group kidney, the histological features greatly improved and are nearly back to normal structure of glomerular capillaries (arrow) showing normalized epithelium (arrow head)

sensitive lipases, which increase the lipolysis and releases more free fatty acids in the circulation. Increased fatty acid concentration also augments the β -oxidation of fatty acids, producing more acetyl-CoA, and cholesterol, which further enhances the development of atherosclerosis in DM [10, 38, 39]. Diabetic animals showed significant increase in the level of triglycerides, serum cholesterol, LDL cholesterol and decrease in HDL-cholesterol levels, while, administration of EMC demonstrated significant and dose dependant reduction in levels of serum triglyceride, serum cholesterol, LDL-cholesterol, and improvement in HDL-cholesterol levels. This effect is suggestive of its utility in DM with hyperlipidemic complication. EMC contains phenolic compounds, tannins and saponins, which may

possibly act by inhibiting cholesterol synthesis and increased excretion of cholesterol [40]. A possible mechanism for this lipid lowering activity of EMC may involve an increase uptake and utilization of glucose leading to gain of glycemic control as witnessed from its effect on liver glycogen content which is noticeably increased with EMC treatment (11.42 ± 0.91 mg/g). Proper glycemic control may lead to inhibition of lipolysis or cholesterol esterase, activation of fatty acid synthase, and production of triglyceride precursors such as acetyl-CoA and glycerol phosphate [41]. Saponins and flavonoids have demonstrated anti-hyperlipidemic potentials by binding with cholesterol in the intestinal lumen (as cholesterol is less readily absorbed) and increasing lipoprotein lipase activity

which helps in removal of LDL and chylomicrons from circulation [42]. Hence, EMC appears to be useful to treat DM and associated complications owing to its anti-hyperglycemic and lipid lowering effect. The abridged atherogenic, cardiac and coronary artery indices demonstrated its potential in the management of risk of cardiovascular complications that accompanied diabetes. This may be attributed to the presence of flavonoids and phenolic compounds, which reduce cardiovascular risk in DM [43].

Liver Glycogen Content

Insulin is the main regulator of glycogenesis in muscle and liver; insulin deficiency in DM leads to decrease in the hepatic and skeletal muscle glycogen content [44]. One possible effect of antidiabetic drugs can be improvement in glycogenesis that ultimately causes control of blood sugar level [45]. Supplementation of the EMC for 21 days in diabetic rats resulted in significant and dose dependant elevation in the liver glycogen content. This is suggestive of improvement of glycogenesis process in the liver is one of the possible ways of antidiabetic action of EMC.

Change in Body Weight

A significant reduction in body weight is one of the characteristic features of uncontrolled DM. Administration of EMC for 21 days have produced a beneficial effect by increasing the body weight of diabetic animals. These results specify reduction in protein catabolism through increasing glucose utilization. During uncompensated diabetes, there is a decrease in body mass due to energy deficit and the cellular catabolic process characterized by glycogenolysis, lipolysis and proteolysis [46, 47]. EMC treatment reversed diabetic proteolytic conditions, which was evident from its positive effect on serum total protein and increased body weight (1.32 ± 0.86 g) of treated animals.

Nephroprotective

Diabetes produces qualitative and quantitative changes in the composition of the glomerular basement membrane which undergoes accelerated glycosylation and further rearrangement to form advanced glycation end-products (AGEs), which cause endothelial dysfunction. Hyperglycemia increases the expression of transforming growth factor beta (TGF β) in the glomeruli and matrix protein, which is specifically stimulated by cytokines. TGF β may contribute to both the cellular hypertrophy and enhanced collagen synthesis observed in DNP [48]. Increased protein catabolism with inflow of amino acids to liver occurs in DM, which feed gluconeogenesis and accelerate

ureagenesis, resulting in hypo-proteinemia and hypo-albuminemia [49]. Hyperglycemia causes elevation in the levels of serum creatinine and reduction in serum total protein and albumin, which are considered as significant markers of renal dysfunction [50–53]. In the present work, EMC treatment demonstrated marked hike in the serum total protein and albumin level, whereas, fall in the levels of serum creatinine. EMC attenuates the progression of renal damage in alloxan-induced diabetic rats, which is further evident from the histological investigations. Marked reversal of glomerulosclerosis and recovery of damaged tubular epithelium observed in EMC-treated rats supports renoprotective actions of EMC.

Conclusion

Ethanollic extracts of *Musa cavendish* demonstrated marked plasma glucose lowering effect in alloxan-induced diabetic rats. Severe decline in glycogen content was the landmark observation of untreated diabetic rats. This was notably reversed with EMC-500 and EMC-1000, which is comparable to glimepiride. In agreement with these effects, body weight of EMC-500 and EMC-1000 treated animals was also increased which is otherwise remarkably lessened in untreated diabetic rats. EMC further demonstrated marked reduction in the levels of serum triglyceride, cholesterol, serum LDL cholesterol and an improvement in the serum HDL cholesterol level. The findings of the present study demonstrated a nephroprotective effect of *Musa cavendish* on the development of alloxan-induced DNP in rats by reducing in serum creatinine and hike in serum albumin and total protein levels. By considering these findings of the present study, it can be concluded that ethanollic extract of *Musa cavendish* fruit peel hibernates dyslipidemia, and nephropathy in diabetic rats.

Compliance with Ethical Standards

Conflict of Interest Authors have no conflicts of interest.

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