**INTRODUCTION**

*Diabetes mellitus* is described as a group of metabolic disorders in which a person has high blood glucose levels, either because insulin production is inadequate, or because the body cells do not respond properly to insulin or both* (Deepthi et al., 2017). Patients with high blood glucose levels will experience polyuria, polydipsia, and polyphagia (Lal, 2016). Diabetes is categorized into three major types: Type I, Type II and gestational diabetes (CDC, 2017). In India, 69.1 million people are affected by diabetes and India will soon have the second-highest number of cases in the world (Sanjeevaiah et al., 2019). In India, the prevalence of diabetes is maximum in the southern part of the country range from 5 to 17% (Sanjeevaiah et al., 2019). *Diabetes mellitus* is expected to become the seventh important cause of mortality in the globe by 2030 and total deaths from hyperglycemia are predicted to increase by more than 50% in the next 10 years (Mathers and Loncar 2006). Chronic diabetes facilitates oxidative stress and plays a significant role in the pathogenesis of diabetes-related problems (Sreekutty and Mini 2016). In the case of diabetes, a cell can not utilize glucose as an energy source which then remains in the blood and ultimately accumulates in the tiny vessels of the kidney, heart, eyes and, nervous system and damages them (Mathenge et al., 2019).
If left untreated, diabetes can lead to cardiovascular diseases, blindness, and neural disorders (Sanjeevaiah et al., 2019). WHO has identified 21,000 medicinal plants with great medicinal values and have been used for many diseases worldwide. Many of them have been known to possess antihyperglycemic activity and produce natural antioxidants (Belal et al., 2017). The present hypoglycemic drugs have potential toxicity as well as lack of efficacy. Therefore, the ideal antidiabetic drug should have a combination of both antidiabetic and antioxidant activity (Krentz et al., 2017). Antidiabetic drugs are either too expensive or have undesirable complications, including hematological, coma, and liver and kidney disorders. At present, only insulin and oral medicines are available to manage the high blood glucose levels in type-1 diabetes (Hosseini et al., 2015).

Moringa oleifera belongs Moringaceae family. Moringa has high nutritional value with great medicinal properties. Every part of the moringa tree is edible. It has long been consumed by humans. It is an exceptional natural source of digestible proteins, minerals, mainly iron, amino acids, and antioxidants (M Halaby et al., 2013). Moringa has several phytochemicals such as simple sugar (rhamnose), alkaloids, flavonoids, glycosides sterols (Doumas et al., 1973; Jain et al., 2010), and many other components like glucosinolates and isothiocyanates (Bennett et al., 2003; Fahey et al., 2001). These polyphenolic secondary metabolites are natural antioxidants that directly react with superoxide anions and lipid peroxyl radicals and subsequently prevent the series of lipid peroxidation. They perform their free radical scavenging activity by donating the hydrogen or electron to the superoxide anions hence moringa is a natural antioxidant (Rajananth and Kavitha, 2010). It also contains a rarer combination of phenolics compounds, including zeatin, quercetin, kaempferol, and apigenin which are vital and diseases-preventing nutrients (Sreelatha et al., 2011). It is used as antipyretic, hypotensive, anti-inflammatory, hypoglycemic, and hypercholesteremia (Sreelatha et al., 2011). Furthermore, moringa leaves steam and bark have been reported as antidiabetic on hyperglycemic rats, mice, and rabbits (Gupta et al., 2012; Kar et al., 2003; Luangpiom et al., 2013; Manohar et al., 2012). Francis et al. (2004) reported that methanolic moringa fruit extracts were effective against diabetes by the stimulation of insulin secretion and also had cyclooxygenase and lipid peroxidase inhibition activity. However, inadequate information on moringa fruit extract as an antidiabetic agent has been reported. Therefore, the current study was undertaken to evaluate the antidiabetic properties of ethanolic fruit extract of M. oleifera in an alloxan-induced diabetic model.

**MATERIALS AND METHODS**

**Experimental mice**

Twenty-four male adult Swiss albino mice were used in the experiment. Experimental mice were 12 weeks old and weighed 30 to 35 gm at the initial period of the investigation. Animals were obtained from the animal house of Mahavir Cancer Sansthan & Research Centre, Patna, India (CPCSEA Reg-No.1129/bc/07/CPCSEA). They were kept under hygienic conditions in well-ventilated chambers at 23 ± 2°C and 50 ± 10% relative humidity with a 12h photoperiod. They were maintained on the laboratory-prepared food, including multigrain bread, soaked grams, green vegetables, and drinking water *ad libitum*. They were divided into four groups (Group I to IV), each consisting of four animals. Each group was housed in distinct polypropylene cages (26 x 19 x 13 cm). All animal groups were adapted under the laboratory housing conditions for 10 days before the beginning of the experiment. All the experimental animals were accompanied as per guidelines of the Committee for control and supervision of experiments on animals (CPCSEA), New Delhi, India. Ethical approval was obtained from the Institutional Animal Ethics Committee (IAEC) of the institute with the IAEC No 2020/IE-27/08/20.

**Preparation of medicinal plant extract**

Fresh fruits of *M. oleifera* were washed, cut into small parts, dried in the incubator at 37°C for 20 days, and ground to powder. Fruit powder was soaked in absolute ethanol for 48 hours. It was then dried through rotary evaporators (Buchi, R-3, Switzerland) at 60°C and 300 psi. The obtained extract was then stored in an airtight bottle. A fresh solution has been prepared from this stock by dissolving 150 mg moringa extract into 5% ethanol. This extract of *M. oleifera* was administered orally (150 mg/kg/body weight/day) to the diabetic mice.

**Induction of diabetes**

Diabetes was induced in animals by alloxan, purchased from Loba Chemie Pvt. Ltd., Mumbai (CAS No-22244-11-3, batch No. 6209707). Twelve mice were fasted overnight and were injected with a single dose of alloxan, intraperitoneally, with a dose of 110 mg/kg body weight. Mice were allowed free access to the food and water *ad libitum*. After 3 days of alloxan administration, the hyperglycemic condition was estimated by testing their fasting blood glucose levels. The blood samples were collected from the tip of the tail. The blood glucose level was estimated by one touch glucometer [Infobia Co. Ltd., Korea]. Mice having a blood glucose level equal to 198 mg/dl were considered diabetic mice.
Experimental plan
Experimental animals were randomly divided into four groups and each group consisted of four animals. Twelve animals were used for the preparation of the diabetic model and four mice were run along with the experiment as the control.
Group I: Untreated controls received food and water only.
Group II: Diabetic controls (Alloxan administered); mice were left without any treatment throughout the experiment for auto-recovery.
Group III: Diabetic (Alloxan administrated) received food and water only.
Group IV: Diabetic group; administration of the ethanolic fruit extract of moringa, @ dose of 150 mg/kg body weight/day orally once a day for 84 days.
Mice of groups I and II were sacrificed along with group IV after the accomplishment of the entire experiment.
Group III was sacrificed after 15 days to evaluate the effects of alloxan.

Biochemical estimation
Mice were sacrificed by the cervical dislocation process after the accomplishment of the experiment. Blood was collected by the orbital puncture. The serum was separated from the blood through the centrifugation method, rpm at 3000 g for 15 minutes at room temperature. The collected serum was used for the biochemical assays. Biochemical parameter tests were completed by the standard commercial kit - Coral clinical system with the help of a spectrophotometer (UV-10, Thermo Scientific, USA). The serum glucose level was estimated by GOD/POD method (Trinder, 1969). Reitman and Frankel (1957) method was used for the estimation of liver markers enzyme serum glutamic-oxaloacetic transaminase (SGOT) and glutamate pyruvate transaminase (SGPT). Jendrassik-Grofs BM (1938) method was used to estimate total bilirubin. Urea was estimated by the method of Fawcett and Scott (1960) and uric acid through the method of Fossati (1980), while creatinine was measured by the method of Bonsnes and Taussky (1945).

Hormonal assay
Estimation of Insulin levels was performed through the Enzyme-Linked Immunosorbert Assay (ELISA) method (Cal biotech, Inc.1935 Cordell Ct., El Cajon’s, 92020 USA, Lot No Ins5296). The final readings were taken through the ELISA reader- AM 2100, Thermo Fisher, USA, and the generated data were interpreted.

Histopathological assessment
The mice were dissected and their tissues were taken out for the histopathological study. The small segments of the liver, kidney, and pancreas tissues were fixed into Tissue preservative (10%) Formalin for 24 hours.

RESULTS

Biochemical analysis
Effect on glucose level
The serum glucose levels in mice (Group I, Control) were stable throughout the experimental period. On the other hand, in the alloxan-induced diabetic group (Group II), there was a significant rise in the serum glucose levels (p<0.0001) in contrast to Group I. However, diabetic mice, which were left without any treatment for 12 weeks for auto restoration, did not exhibit any natural restoration in their hyperglycemic state. Pancreatic diameter decreased significantly (P<0.0001) in the diabetic model (Group II) and the diabetic control (Group III) relative to the control (Group I) (Fig. 1).

Effect on the serum insulin level
Serum insulin levels significantly lowered down (p<0.0001) in both diabetic models (Group II) and diabetic control in comparison to normal control (Group I). However, the serum insulin levels markedly increased in moringa treated group of mice. while the diabetic control group (Group III) restored the insulin levels up to 12 weeks (Fig. 2).

Effect on pancreas size
The diameter of pancreas was significantly (p<0.0001) decreased in both diabetic model (Group II) and diabetic control (Group III) as compared to the control (Group I). It may happen due to free radical regeneration of alloxan and cause necrosis of Pancreas. However, in the moringa treated group (Group VI), there was a significant (p<0.0001) increase in the pancreas diameter as compared to the diabetic model and diabetic control both (Fig. 3).

Serum level of hepatic marker enzymes
The results showed that alloxan-induced (Group II) had markedly (p<0.0001) elevation in the serum levels of hepatic marker enzymes (SGOT, SGPT) and significant (P<0.0001) increase in the pancreas diameter as compared to the diabetic model (Group II) and diabetic control (Group III). However, the serum glucose levels markedly increased in moringa treated group of mice. while the diabetic control group (Group III) restored the insulin levels up to 12 weeks (Fig. 2).
bilirubin, SGPT and SGOT, as compared to the normal control group (Group I). However, in an alloxan-induced diabetic control group of mice (Group III), which were kept for auto-recovery for 12 weeks after alloxan treatment, had a significant (p<0.0001) decrease in the SGPT, SGOT levels, but the bilirubin levels non-significantly changed in comparison to the diabetic mice. But, after the administration with ethanolic moringa fruit extract with a dose of 150 mg/kg body weight per day for 12 weeks on diabetic mice, showed the defensive effect against diabetic induced hepatotoxicity as there was a significant (p<0.0001) reduction in the levels of SGPT, SGOT and bilirubin levels in comparison to the diabetic control and diabetic mice (Table 1).

Serum level of renal marker enzyme

The mean value of renal marker enzyme creatinine urea, and uric acid, were markedly (p<0.0005) elevated in the diabetic (group II) and diabetic control (group III) in contrast to the normal control mice. However, the alloxan-induced diabetic control group (Group III), which was kept for auto-recovery for 12 weeks after alloxan-induced, had a significant (p<0.05) reduction in the urea, but uric acid and creatinine levels had a non-significant change in comparison to the diabetic model. Group II but, after treatment with moringa ethanolic fruit extract over 12 weeks on diabetic model mice, it showed the defensive effect against diabetes-induced kidney damage. Creatinine, urea and uric acid were significantly (p<0.001) reduced in comparison to the diabetic control and diabetic group (Table 2).

**Table 1. Effect of ethanolic extract of moringa fruit on liver marker enzyme**

<table>
<thead>
<tr>
<th>Group</th>
<th>Control (Group I)</th>
<th>Diabetic model (Group II)</th>
<th>Diabetic control (Group III)</th>
<th>Mo 12 w treated (Group IV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SGPT (U/mL)</td>
<td>25.25±1.79</td>
<td>192.0±4.20***</td>
<td>164.0±4.52**</td>
<td>55.50±2.10**</td>
</tr>
<tr>
<td>SGOT (U/mL)</td>
<td>28.75±0.59</td>
<td>206.3±6.303***</td>
<td>131.5±4.36**</td>
<td>62.58±1.81**</td>
</tr>
<tr>
<td>Bilirubin (mg/dl)</td>
<td>0.64±0.04</td>
<td>1.95±0.15***</td>
<td>1.97±0.08***</td>
<td>0.72±0.11***</td>
</tr>
</tbody>
</table>

Levels of liver marker enzyme in different treated groups (control and treated group). Values are mentioned as mean ± SEM, n=6. Statistical significance level was checked through the one-way ANOVA, followed by Tukey’s test with multiple comparisons. The p-values of diabetic model group***(p<0.0001) compared with the normal control group; Mo 12 W treated group with control group (p<0.05).
Histopathological assessment of the hepatic tissue
The liver section of normal control mice (Group I) showed the normal architecture of the central vein lined by endothelial cells, hepatic cells with a well-defined nucleus, and normal sinusoids. Cytoplasmic material was also normal. (Fig. 4A). The liver section of the alloxan diabetic mouse (group II) showed significant deterioration of the hepatocyte and central vein with the broken or irregular pattern of endothelial cells with frequent vacuolisations (Fig. 4B). The hepatic section of the diabetic control (group III) showed a slight articulation of the central vein, the portal vein, but vacuolisations in the cytoplasmic material were still persistent. There was mild amelioration observed in the hepatic tissue in the diabetic control group. It may be due to the regeneration property of the liver (Fig. 4C). The ethanolic extract of moringa fruit administration on the diabetic model, liver section showed a significant normal hepatocellular architecture which was well organized. The central vein showed necrotic nuclei or hetrochromatized nuclei with significant restoration. Hepatic cells were normal with cytoplasmic materials and sinusoids (Fig. 4D).

Histopathological assessment of renal tissue
In this histopathological examination, the control mice kidney section showed the normal structure of renal parenchyma and glomerulus in Bowman’s capsule, Proximal convoluted tubules (PCT) and Distal convoluted tubules (DCT) (Group I) (Fig. 5A). The alloxan-induced mice (Group II) kidney section showed degeneration in the glomerulus; vacuolations in Bowman’s capsule along with the distal and proximal convoluted tubules, dilation in renal tubules (Fig. 5B). The diabetic control (Group III) showed degeneration in the nephrocytes, thickening of the glomerular basement membrane, and cytoplasmic vacuolations which indicate auto-improvement up to twelve weeks (Fig. 5C). Bowman’s capsule, glomerulus, and the distal and proximal convoluted tubules showed the normal architecture after administration of moringa fruit extract for 12 weeks upon alloxan-induced diabetic mice in comparison to the diabetic mice and diabetic model mice both (Fig. 5D).

Histopathological study of the pancreatic tissue
In this histopathological study, the pancreatic section of control (Group I) showed the normal structure of acini and islets of Langerhans. Islets showing compact spherical mass with intact interlobular connective tissue (Fig. 6A). The alloxan-induced (Group II) pancreatic section showed necrosis and vacuolization in islets of langerhans, pancreatic acini are not well organized. The number and size of islets are also reduced as com-

<table>
<thead>
<tr>
<th>Group</th>
<th>Control (Group I)</th>
<th>Diabetic model (Group IV)</th>
<th>Diabetic control (Group III)</th>
<th>MO 12w treated (Group IV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea (mg/dl)</td>
<td>22.78±0.54</td>
<td>40.13±1.73***</td>
<td>48.73±0.67***</td>
<td>28.59±2.58***</td>
</tr>
<tr>
<td>Uric acid (mg/dl)</td>
<td>2.75±0.34</td>
<td>9.37±0.66**</td>
<td>8.95±0.53**</td>
<td>3.65±0.29***</td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>1.15±0.07</td>
<td>1.63±0.16**</td>
<td>1.80±0.26**</td>
<td>0.57±0.07**</td>
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Table 2. Effect of the ethanolic extract of moringa fruit extract in kidney marker enzyme

Levels of liver marker enzyme in different treated groups (control and treated group). Values are mentioned as mean ± SEM, (n=6). Statistical significance level was analyzed by the one-way ANOVA, followed by Tukey’s test with multiple comparisons. The p-values of diabetic model group*** (p<0.0001) compared with the control group; MO 12 W treated group with the control group (p<0.05).

Fig. 4. Photograph of mice liver stained with hematoxylin and eosin (H&E 500x). (A) Liver section of control mice with regular arrangement of hepatocytes (H) and central vein (CV), sinusoids (S) and kupffer (K) cells (H&E 400x). (B) Section of alloxan-induced mice liver showing degenerated hepatocytes (H), bile duct (BD), and kupffer cells (KF) (H&E 400x). (C) Section of alloxan-induced diabetic control mice liver with mild degeneration and vacuolization in the hepatocytes (H) due to alloxan (H&E 400x). (D) Section of liver administered with moringa fruit extract upon diabetic mice showing significant improvement in the hepatic histoarchitecture along with the central vein (CV) and hepatic cells (H), sinusoids (S) (H&E 400x)
pared with the control (Fig. 6B). The diabetic control mice (Group III) also showed degeneration in acini and islets of Langerhans, dilation of the intralobular duct, reduction in number and size of islets. It does not show auto-restoration (Fig. 6C). The ethanolic moringa fruit extract administration upon alloxan-induced diabetic mice pancreatic section showed significant restoration in the islets of Langerhans, normal acini, and an intralobular duct. The number and size of islets of Langerhans significantly increased as compared with the diabetic control. It shows the regeneration of islands with β cells (Fig. 6D).

DISCUSSION

Antidiabetic properties of *M. oleifera* have been reported previously by different authors (Bamagous et al., 2018; Abd El Latif et al., 2014; Villarruel-Lopez et al., 2018; Tang et al., 2017). As it is well known that fruits of *M. oleifera* are generally consumed as a vegetable, so this study is very important because it deciphers the antidiabetic effect of fruit extract on the current animal model. Therefore, the assessment and implementation of the hypoglycemic effect of Moringa fruits create a benchmark for managing diabetes.

Alloxan was used as a diabetogenic for the selective destruction of β cells (Lenzen, 2008). After the induction of diabetes, serum glucose levels were significantly raised in comparison to the normal mice, also reported by (Durašević et al., 2019). However, after the treatment of ethanolic moringa fruit extract for twelve weeks, the glucose level of diabetic mice significantly reduced and almost in a normal range of fasting serum glucose level. Similar results have been reported, with the help of an aqueous extract of leaves of moringa in Wistar rats (Jaiswal et al., 2009), methanolic moringa extract in alloxan-induced diabetic mice (Olayaki et al., 2015). Another study was made by Hemant et al. (2013) who observed that ethanolic pod extract in alloxan diabetic rats with a concentration of 200 mg/kg
showed antidiabetic properties by increasing the action of insulin. In the present study, insulin secretion from the pancreatic β-cell was significantly (p<0.0001) reduced in group III from 6.8±0.42 to 1.13±0.08 in the alloxan-induced diabetic mice in contrast to the normal control group of mice, while after administration with the ethnic moringa fruit extracts. The serum insulin levels in diabetic mice were significantly (p<0.0001) elevated from 1.13±0.08±7.94±0.45 after the treatment with moringa extract. Basyony et al. (2016) also found that there was a significant reduction in the insulin levels in alloxan-treated mice but, after the administration of moringa leaves for 30 days, there was a significant increase in the insulin levels in diabetic mice. Isolated N-Benzyl thiocarbamates, benzyl nitriles, N-benzyl carbamates, and a benzyl ester from methanol extract of moringa fruit powder were reported to stimulate the insulin release from the rodent's pancreatic β-cells. Ethanolic moringa leaf extract elevates insulin levels in diabetic mice, confirmed by (Tang et al., 2017). Many authors (Karthikesan et al., 2010; Vergara-Jimenez et al., 2017) conclude that chlorogenic acid found in moringa plays an important role in maintaining the glucose levels by inhibiting the glucose 6-phosphate translocase in diabetic rats by reducing glycogenolysis and gluconeogenesis. *M. oleifera* fruit extract inhibits the enzymatic activity of alpha-amylase and alphaglucosidase in the intestine, which prevents the breakdown of the complex sugar into simple sugar, thus preventing the hyperglycemic condition (Kamalrudin et al., 2018). The liver plays an important role to balance the normal glucose levels in the blood, which regulates the insulin secretion and release of the inflammatory cytokines (Bamagous et al., 2018). Elevated levels of serum hepatic marker enzymes are the indicators of hepatic damage, where the enzymes are released into the bloodstream (Annadurai et al., 2012; Geleta and Mackonen, 2015). Alloxan-induced diabetic mice also showed similar results, proving the hepatic damage. Agarwal et al. (2012) also found a marked elevation in the levels of serum liver marker enzymes such as bilirubin, SGPT, and SGOT. In the alloxan-induced diabetic mice. However, oral administration of ethnic moringa fruit extract for 12 weeks in diabetic mice showed a substantial reduction in serum hepatic marker enzyme levels compared to the diabetic control. This finding is in agreement with (Bamagous et al., 2018). Furthermore, moringa shows hepatoprotective activity against acetaminophen (Aly et al., 2020), alcohol-induced liver disorders (Muzumbukila et al., 2019), and cadmium toxicity (Toppo et al., 2015). This liver injury may be restored due to the antioxidant property of *M. oleifera*, which inhibits the oxidative stress associated with liver injury (Alhakmani et al., 2013). In the present study, the level of urea and uric acid significantly (p<0.0001) increased in diabetic control (Group III) range from 22.78±0.54 to 48.73±0.67 and 2.75±0.34 to 8.95±0.53, respectively. The level of creatinine significantly (p<0.0001) increased from 1.63±0.16 to 1.80±0.28 in diabetic control (Group III) as compared to the control mice. Elevation of the enzyme due to the oxidative stress of alloxan. Additionally, our results showed marked improvement (p<0.0001) in the renal marker enzymes after the administration of ethnicolic moringa fruit extract in diabetic mice for twelve weeks. This finding is in agreement with (Al-Malki and El Rabey, 2015). As a result of histopathology, one important finding was observed: the size from 160.7±2.61 to 113.3±0.84 of islets of Langerhans in diabetic control mice was significantly (p<0.0001) decreased in comparison to the normal control group of mice. Moreover, degeneration of pancreatic β-cells and vacuolizations was observed in diabetic albino rats, which were earlier reported by (Elkotby et al., 2018). Whereas, in the moringa administered group, there was a significant increase (p<0.0001) in size from 113.3±0.84 to 137.3±3.17 of the islets of Langerhans with well-arranged pancreatic β-cells. Apart from that, it almost achieved its normal architecture and these results decipher that ethanolic moringa fruit extract is involved in pancreatic cell regeneration. In another study, Gupta et al. (2012) also found the restoration in histoarchitecture of pancreatic tissue through the methanolic extract of moringa leaves extract. Moringa is a good source of antioxidants and valuable therapeutic nutrients like flavonoids, terpenoids, kaempferol, and quercetin. Therefore, it may be capable of repairing and regenerating injured pancreatic tissue and β-cell in alloxan-induced diabetic mice. These components are strong antioxidants and great free radical scavengers (Abd El Latif et al., 2014; Moyo et al., 2012; Tende et al., 2011). In the present study, the elevated serum hepatic marker showed a direct correlation with our histopathological study. In alloxan-induced diabetic mice showed degenerated hepatocytes, bile duct, and kupffer cells with degenerated nuclei and enlarged sinusoids. Lucchesi et al. (2015) also observed a similar pattern in their study, in the diabetic rat, which liver shows similar morphology same as chronic fatty liver in diabetic human patients Ethanolic moringa fruit extract effectively ameliorates hepatic tissue damage in 12 weeks. (Omodanisi et al., 2017). Alloxan-induced oxidative stress also damages the renal tissue especially causing vacuolation in the Bowman’s capsule and glomerulus. This is in agreement with the study on the effect of *Salvadora persica* leaf extracts in alloxanized rats (Alrasheed, 2020). Ethanolic moringa fruit ameliorates the bowman’s capsule, glomerulus and distal convoluted tubules and shows normal histoarchitecture of the kidney. This is consistent with another finding who reported significant
alterations in the renal tissue in the diabetic rat after the treatment with the methanolic moringa extract (Kandasamy and Ashok Kumar 2013; Omodanisi et al. 2017).

Conclusion

Generally, people prefer the moringa fruit in their diet over the moringa leaves and moringa fruit extract is less explored as an anti glycemic agent. Based on the experimental results, it was concluded that the fruit extract of *M. oleifera* provided potentially protection against hyperglycemia and its complications. It maintained the blood glucose levels, normalized the insulin level, and protected and rejuvenated the pancreatic islets, liver, and kidney tissue. It also improves carbohydrate metabolism, including restoring the integrity and function of the pancreatic β-cells along with the glucose uptake and utilization. Moringa extract maintains glucose homeostasis. Findings suggest that *M. oleifera* fruit extract is a potential antidiabetic drug that can reduce blood glucose levels and diabetic-associated problems.

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Conflict of interest

The authors declare that they have no conflict of interest.

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