Diabetes mellitus (DM) is probably one of the oldest diseases known to mankind. It was first reported in Egyptian manuscript about 3000 years ago. In 1936, the distinction between type 1 and type 2 DM was clearly established. Type 2 DM was first described as a component of metabolic syndrome in 1988. Type 2 DM (formerly known as non-insulin dependent DM) is the most common form of DM characterized by persistent hyperglycemia, insulin resistance, and relative insulin deficiency. The initiation and progression of Type 2 DM results from the interactions between genetic, environmental and behavioral risk factors.

Type 2 diabetes mellitus (DM) is a chronic metabolic disorder and its prevalence has been increasing alarmingly all over the world. As a result of this trend, it is becoming an epidemic in some countries of the world with the number of people affected is expected to double in the next decade due to increase in ageing population, thereby adding to the already existing burden for healthcare providers, especially in developed countries. Screening and diagnosis is still based on World Health Organization (WHO) and American Diabetes Association (ADA) criteria which include both clinical and laboratory parameters. No cure has yet been found for the disease; however, treatment modalities include lifestyle modifications, treatment of obesity, oral hypoglycemic agents and insulin sensitizers like metformin and biguanides that reduce insulin resistance. Other effective medications include non-sulfonylurea secretory analogues, thiazolidinediones, alpha glucosidase inhibitors and insulin.
Intensive research into the pathophysiology of type 2 DM has led to the introduction of new medications and more recently several metal complexes of vanadium and zinc have synthesized and evaluated for their efficacy in treating diabetes. In the present study, we have synthesized a zinc complex using morin and evaluated its antidiabetic properties in HFD-STZ induced type 2 diabetes in experimental rats.

The following are the summary of the present study

- A novel zinc-morin complex was synthesized and characterized using spectral studies such as FTIR, $^{13}$CNMR, $^1$HNMR and Mass spectra.
- Acute toxicity studies revealed that zinc-morin complex up to 500mg/Kg.b.w did not elicit any adverse effects in the control group of rats.
- Based on the periodical assessment of fasting blood glucose levels, the optimum dose of zinc-morin complex was fixed as 5 mg/kg body weight/rat/day orally for 30 days.
- Oral administration of zinc-morin complex improves oral glucose, insulin tolerance and HOMA-IR in experimental diabetic rats, which indicates the positive impact of zinc-morin complex on glucose homeostasis.
- Zinc-morin complex modulates the activity of key enzymes of carbohydrate and glycogen metabolism and improves the
glycogen content in hepatic tissues of experimental diabetic rats thereby maintains normoglycemia.

- Zinc-morin complex significantly improves the levels of fasting blood glucose, plasma insulin, glycosylated hemoglobin, plasma protein, blood urea, serum creatinine, uric acid and reduce the activity of serum marker enzymes such as AST, ALT, ALP indicating the antidiabetic as well as tissue protective nature of zinc-morin complex.

- Zinc-morin complex improves the levels of adipokines such as adiponectin, leptin and TNF-α which indicates the positive impact of zinc-morin complex on glucose homeostasis.

- Zinc-morin has stimulated the pancreatic insulin secretion in RINm5F pancreatic beta cells.

- Zinc-morin complex restore the antioxidant status, inflammatory responses and the physiology of pancreatic islets of experimental diabetic rats thereby preserving the beta cell insulin content and Glucose stimulated insulin secretion, which is evidenced from the plasma insulin level.

- The histopathological, ultrastructural and immunohistochemical observations made on the pancreatic tissue of high fat diet- low dose streptozotocin induced diabetes in rats evidenced the tissue protective nature of zinc-morin complex.
The histopathological, ultrastructural, PAS staining, Masson trichrome and toludine blue staining in the liver tissue of high fat diet - low dose streptozotocin induced diabetes in rats also evidenced the tissue protective and antidyslipidemic property of zinc-morin complex.

Zinc-morin complex facilitates 2-Deoxy glucose uptake in skeletal muscles of experimental diabetic rats and improves muscle glycogen content.

Zinc-morin complex significantly improves the uptake of glucose in rat L6 myotubes by increasing the translocation of GLUT4 to the plasma membrane.

Zinc-morin complex enhances the uptake of glucose in rat L6 myotubes independent of insulin and through the stimulation of PPARγ and activation of PI 3-Kinase signaling pathway.

Zinc-morin complex enhances glucose uptake in FFA-induced insulin resistant in rat L6 myotubes.

Zinc-morin complex improves insulin sensitivity in FFA-induced insulin resistant rat L6 myotubes by reducing the accumulation of lipid metabolites, which are capable of suppressing the action of insulin via increasing the phosphorylation of Ser(307) on IRS-1.

In conclusion, the above studies hopefully emerged with an obvious portrayal of the antidiabetic, antidyslipidemic, antioxidant, and anti-inflammatory properties of zinc-morincomplex in experimental type 2
diabetes and the efficacy was comparable with metformin, a standard drug widely used for the treatment of type 2 diabetes. The improved actions in the diabetic rats treated with zinc-morin complex might be due to the zinc-morin complex mediated activation of PPARγ and PI3-kinase in peripheral tissues and especially in skeletal muscle, which constitutes more than 50% of the total body mass. Thus, the results of the present study established the scientific evidence for the non toxic as well as the antidiabetic properties of zinc-morin complex.