SUMMARY AND CONCLUSION

According to current estimates, the human population worldwide appears to be in the hub of an epidemic of diabetes. In spite of the great developments that have been made in the understanding and management of diabetes, the disease and disease related complications are increasing persistently. Recent developments in understanding the pathophysiology of the disease process have opened up several new ways to identify and develop novel therapies to conflict the diabetic pandemic. Concurrently, sulphur containing allyl groups are presenting an exciting opportunity for the development of new types of therapeutics.

In the present study 2-allyl amino 4-methyl sulfanyl butyric acid (AMSB) was synthesized in good yield. AMSB was characterized by FTIR, NMR ($^1$H and $^{13}$C) and LCMS. The radical scavenging activity and reducing power assay of AMSB was assessed using DPPH, ABTS and FRAP assay and was found to be 44.1, 34.71 and 41.7 $\mu$g/ml respectively. The synthesized compound showed effective inhibition against $\alpha$-amylase and $\alpha$-glucosidase. AMSB was identified to be a reversible mixed noncompetitive inhibitor of $\alpha$-amylase and $\alpha$-glucosidase. The molecular docking study was carried out to evaluate the specific groove binding properties and affords valuable information of AMSB binding mode in the active site of $\alpha$-glucosidase. AMSB has strong potential to be further investigated as a new lead compound for better management of diabetes.

The male albino wistar rats were randomly divided into four groups with six animals in each group. DM was induced by intraperitoneal injection of streptozotocin (STZ) (55mg/kg). After induction of diabetes, rats were treated with 2-allylamino 4-methyl sulfanyl butyric acid (AMSB) (150 mg/kg body weight) for
45 days. The glucose level was studied after 72 hrs to ratify the development of DM. Before the post treatment the blood glucose level was increased whereas body weight was decreased. The biochemical estimations like lipid profile, fatty acid, phospholipids of plasma and tissues (liver and kidney) and carbohydrate metabolic enzymes were performed. Administration of AMSB resulted in significant reduction in blood glucose, cholesterol, triglycerides, low density lipoprotein (LDL), very low density lipoprotein (VLDL), free fatty acids and phospholipids. In addition, significant elevation in body weight and high density lipoprotein (HDL) was also observed in AMSB treated rats. On the other hand, the activity of hexokinase, glycogen and glycogen synthase were significantly increased and reduction in glucose-6-phosphatase, fructose-1,6-bisphosphatase and glycogen phosphorylase were also observed.

Further the effect of AMSB on protein and glycoprotein components in STZ induced diabetic rats was investigated. On pretreatment, the levels of urea, uric acid, creatinine, glycosylated haemoglobin (HbA1c) and pathophysiological enzymes such as aspartate transaminase (AST), alanine transaminase (ALT) and alkaline phosphatase (ALP) were analyzed and found to be increased. Besides, glycoprotein components such as hexose, hexosamine, fucose and sialic acid in plasma, liver and kidney were significantly elevated, while the protein, plasma insulin and haemoglobin level were found to be decreased. On post treatment, significant reduction were observed in blood urea, serum uric acid, serum creatinine, glycosylated haemoglobin (HbA1c) and glycoprotein components such as hexose, hexosamine, fucose and sialic acid whereas, the level of protein, plasma insulin and haemoglobin was increased.
Oxidative stress plays an important role in the development of DM. the effect of AMSB on the oxidative stress in STZ induced diabetic rats. The oxidative stress was measured in the tissues of pancreas, liver and kidney using antioxidant markers such as lipid peroxidation (LPO) thiobarbituric acid reactive substances (TBARS), hydroperoxide (H₂O₂), superoxide dismutase (SOD), catalase (CAT), reduced glutathione (GSH), glutathione peroxidase (GPx), Glutathione oxidized (GSSG) and GSH/GSSG ratio. After oral administration of AMSB, a significant (P<0.05) reduction in TBARS, H₂O₂ and Glutathione oxidized (GSSG) level were observed in diabetic rats. Significant (P<0.05) elevations were observed in SOD, CAT, GSH and GPx levels in the pancreas, liver and kidney of AMSB treated diabetic rats. Histology studies proved that diabetic rats treated with AMSB showed β-cell regeneration, besides the preservation of architecture and hypertrophy of hepatocytes of liver and kidney. These findings suggest that AMSB showed antioxidative effects in STZ induced diabetic rats.

In conclusion, the use of AMSB offers propitious effects in STZ induced diabetes. The present study gratifies the overall objectives that AMSB may be a plausible therapeutic agent for the treatment of diabetes.