Chapter - 6

Summary & Conclusion
Summary

- STZ administered rats developed characteristic features of diabetes such as polyuria, polydipsia and an increase in food intake. Diabetic rats showed decrease in serum insulin levels with concomitant increase in blood glucose levels. Clorgyline treatment had no effect on these parameters.

- MAO-A protein expression did not alter however, MAO-A activity was found to be increased in the heart of diabetic rats. CLG treatment showed significant reduction in MAO-A activity without altering its protein expression.

- CLG treatment did not show hepatotoxicity in control and diabetic rats.

- Serum markers of myocardial injury such as Troponin I, CK-MB and CK-NAC were elevated in diabetic rats and were normalized after MAO-A inhibition.

- Assessment of hemodynamic and electrocardiographic parameters showed that MAO-A inhibition could reinstate cardiac dysfunction in diabetic rats.

- Increased MAO-A activity contributed to oxidative stress and mitochondrial damage in diabetic hearts.

- MAO-A inhibition prevented diabetes induced cardiac cell death.

- Diabetes-induced myocardial morphological changes and cardiac fibrosis were ameliorated after MAO-A inhibition.
**Conclusion**

In conclusion, our findings support MAO-A as an important source of ROS that contributes to oxidative stress in DCM. Furthermore, prevention of cardiac contractile dysfunction, apoptosis as well as fibrosis by a specific inhibitor of MAO-A, clorgyline, suggest that an increase in cardiac MAO-A activity could play a major role in the progression of DCM. Thus we propose that MAO -A may be a promising pharmaceutical target for the cardio protection in diabetes.

Schematic representation summarizing the mechanism of action of MAO-A in diabetic cardiomyopathy.