The results and conclusions of the present dissertation can be summarised as follows:

1. Alloxan-induced diabetes caused a decrease in the activity of monoamine oxidase with a concomitant increase in the activity of Na\(^+\)K\(^+\)ATPase in brain regions at early time intervals. A reversal of the effect was seen with insulin administration to the diabetic rats.

2. Following thyroidectomy, the monoamine oxidase activity decreased at early time intervals, whereas the Na\(^+\)K\(^+\)ATPase activity increased in all the regions of the brain. T\(_3\) treatment to thyroidectomised rats increased the monoamine oxidase activity, whereas the Na\(^+\)K\(^+\)ATPase activity was decreased.

3. 6-Aminonicotinamide (6-AN) administration to control rats showed a decrease in monoamine oxidase activity at all time intervals, whereas the Na\(^+\)K\(^+\)-ATPase activity was found to increase. However, a regional variation in the activity of MAO and Na\(^+\)K\(^+\)ATPase was observed after 6-AN administration.
4. A substantial percentage of monoamine oxidase activity (about 35 per cent) was found in the soluble fraction of rat brain homogenates. The enzyme was not inhibited in vivo by high doses of MAO A and MAO B inhibitors, namely clorgyline and deprenyl (12 mg/kg for 2 hr). Monoamine oxidase activity associated with the soluble fraction did not show the usual biphasic dose-response kinetics with clorgyline, when kynuramine was used as the substrate. The data show some evidence for the presence of a new form of MAO associated with the soluble fraction of the cell, having properties different from the two well known mitochondrial monoamine oxidases A and B.

5. The mitochondrial and the cytosolic (MAO S) monoamine oxidase were purified 220 and 120 fold respectively from the rat brain. The comparative study of the properties revealed that the mitochondrial and the cytosolic monoamine oxidases are two different enzyme entities.

6. In vitro studies on purified Na⁺K⁺-ATPase revealed that aldehydes derived from oxidative deamination of the MAO catalysed reaction, may regulate Na⁺K⁺-ATPase activity in brain.