SUMMARY

1) The neurotransmitter content observed in hypothalamus of diabetic rats showed an increased adrenergic activity and a decreased serotoninergic activity. The brain stem adrenergic activity remained the same except epinephrine content was increased. Serotonergic activity also was increased. The cerebral cortex of diabetic rats showed an increased adrenergic and serotonergic activity. The dopaminergic activity of corpus striatum showed an increased dopamine content. Serotonin and other amines of cerebellum remained unaffected by diabetes.

2) The α-2 adrenergic receptors of hypothalamus showed an increased affinity. Serotonergic receptors of hypothalamus switched to more of S1 receptors in diabetic rats.

3) The brain stem α-2 adrenergic receptors and serotonergic receptors showed decreased affinity for their agonists during diabetes.

4) The cerebral cortex of diabetic rats showed more of S2 receptors than their controls.

5) The guanine nucleotide regulated affinity changes in diabetic hypothalamus is impaired. This keep the diabetic hypothalamus α2 adrenergic receptors in high affinity receptor state. An altered Gi function is proposed in diabetic rats.
6) The hypothalamic $\alpha_1$ adrenergic receptors increased in number and a decreased phospholipase C activity coincided with this change. These two changes may be related.

7) In the brain stem, the guanine nucleotide and sodium ion mediated affinity changes are intact but their effect on the $\alpha_2$ receptors to keep all the receptors in $\alpha_2L$ state may be an adaptation for the increased epinephrine content.

Together all the changes in the $\alpha_2$ receptor affinity with $5-HT_1$ receptor increase in hypothalamus indicate a possible role for hypothalamic neurons in producing streptozotocin-induced diabetic state. But it is yet to clearly demonstrate that it was these changes that produce diabetes. It is also concluded that treatment with insulin did not reverse the neurotransmitters and their receptor changes. This clearly indicate that in diabetics the glucose level may reverse to normal with treatment of insulin, but the metabolic disturbances and behavioral changes affected is not reversed. A better cure is awaited in the coming years where brain neurotransmitter receptor-hormonal network will be given more importance.