Conclusions
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Conclusions with Respect to 5-HT and Diabetes Mellitus:

5-HT produces dose dependent hyperglycemia that may be mediated through specific 5-HT$_{2A}$ and 5-HT$_3$ receptors. These receptors appear to be present peripherally and alteration in their activity may be involved in diabetes mellitus.

5-HT$_{2A}$ receptor antagonist sarpogrelate and 5-HT$_3$ receptor antagonist ondansetron produce beneficial effect in streptozotocin-induced type 1 diabetes mellitus. Sarpogrelate produces more beneficial effects in diabetes particularly with respect to cardiac function. This may be due to release of insulin from pancreas.

5-HT$_{2A}$ receptors are involved in glucose transport mechanisms and increase in glucose transporters in cardiomyocytes by sarpogrelate may be independent of insulin. It is possible that 5-HT$_{2A}$ receptor inhibition causes an increase in the biogenesis of glucose transporters and thereby increasing the glucose transport in cardiomyocytes.

It is suggested that 5-HT$_{2A}$ receptors may be looked upon as a novel target to develop anti-diabetic drugs to prevent the cardiovascular complications associated with diabetes mellitus considering:

(a) The close association between 5-HT$_{2A}$ receptors and glucose transporters,

(b) The beneficial effects of 5-HT$_{2A}$ receptor antagonists like sarpogrelate in cardiovascular abnormalities and,

(c) The link between cardiac dysfunction and glucose metabolism.
Conclusions with Respect to Dopamine and Diabetes Mellitus:

Like 5-HT, dopamine also produces hyperglycemia. The mechanism of DA-induced hyperglycemia remains unclear because specific DA agonists or antagonists failed to elicit alteration in glucose levels.

Chronic treatment with fenoldopam produces beneficial effects in both type 1 and type 2 diabetes mellitus in rats. Fenoldopam is more effective in type 2 than in type 1 STZ-induced diabetic rats with respect to metabolic and renal functions. Fenoldopam improves insulin sensitivity indicating interlink between dopaminergic system and insulin sensitivity.

Hyperinsulinemia causes down-regulation of D1 receptor function in proximal convoluted tubules and lowering of plasma insulin levels leads to the restoration of renal D1 receptor function and increase in sodium excretion. Thus, insulin seems to play a significant role in the regulation of renal functions through D1 receptors.