Catecholaminergic Neurons and the Regulation of Glycemia: The Importance of Hindbrain-Hypothalamic Interactions

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Normal variations of blood glucose in healthy mammals are directed back into a relatively narrow range (5-7mM) by a series of endocrine mechanisms that primarily involve insulin, glucagon, epinephrine, and glucocorticoid. These control mechanisms are compromised in people with diabetes mellitus, either because of failed B-cell function (type 1; T1D), or from insulin resistance and increasingly poor B-cell function (type 2; T2D). With T1D—and now more commonly in T2D—insulin therapy is the treatment of choice. While effective at reducing hyperglycemia, the difficulty of delivering appropriate rates and doses of insulin in real time means that patients can be exposed to bouts of hypoglycemia, which if frequent or unrecognized, lead to serious complications. Brain mechanisms play a primary role in controlling epinephrine, glucocorticoid and glucagon secretion during hypoglycemia. I will discuss recent results that broadly address the structure and function of the brain networks involved with glycemic control, including the importance of hindbrain catecholaminergic projections to the hypothalamus, and the role of the ventromedial nucleus of the hypothalamus. I will also discuss evidence that distinct but somewhat overlapping brain networks are responsible for the counterregulatory responses that follow hypoglycemia with either a rapid-onset rate (the more common form in experimental models) and a slow-onset rate (probably the clinically more prevalent form).