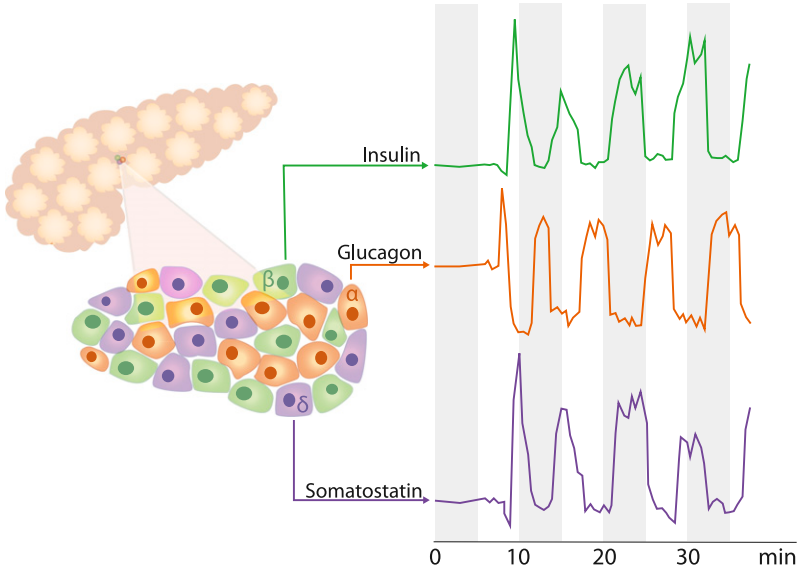

FIG. 1. Schematic representation of the pulsatile nature and temporal relationships of insulin, glucagon, and somatostatin secretion by glucose-stimulated human β-cells. Hormone concentration tracings from Ref. 5 with permission.

in the pathogenesis of type 2 diabetes may not apply to all forms of this disorder, but support for the hypothesis is provided by the fact that among the several common genetic variations associated with type 2 diabetes, β-cell α_2A-adrenergic receptor is overexpressed, potentially mediating suppression of insulin secretion (14). Moreover, epidemiological data show that β-cell dysfunction, loss of pulsatile insulin secretion, and insulin resistance are frequently observed in relatives of patients with type 2 diabetes (15). Finally, the findings by Matveyenko et al. may have therapeutic implications. If confirmed, the findings imply that increasing circulating insulin levels by secretagogues or administration of long-acting insulin preparations may contribute to the development of tissue insulin resistance, and the possibility of providing insulin in a pulsatile manner should be explored (16).

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