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Regulation of Growth Hormone

Inhibitory Control of Growth Hormone Secretion by Somatostatin in Rat Pituitary GC Cells: sst_2 but Not sst_1 Receptors Are Coupled to Inhibition of Single-Cell Intracellular Free Calcium Concentrations

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- Growth hormone
- Somatotropes
- GC cell line

Abstract

Rat pituitary tumor cells (GC cells) exhibit spontaneous oscillations of intracellular free calcium concentration ($[Ca^{2+}]_i$) that allow continuous release of growth hormone (GH). Of the somatostatin (SRIH) receptor subtypes (sst receptors) mediating SRIH action, sst_1 and sst_2 receptors are highly expressed by GC cell membranes. In the present study, the effects of sst_1 or sst_2 receptor activation on single-cell $[Ca^{2+}]_i$ were investigated in GC cells by confocal fluorescence microscopy. In addition, the effects of sst_1 or sst_2 receptor activation on GH secretion were also studied. Our results demonstrate that SRIH decreases $[Ca^{2+}]_i$ baseline and almost completely blocks Ca^{2+} transients through activation of sst_2 but not of sst_1 receptors. In contrast, SRIH effectively inhibits GH secretion through activation of both sst_1 and sst_2 receptors. Blocking Ca^{2+} transients is less efficient than SRIH to inhibit GH release. The cyclic octapeptide, CYN-154806, antagonizes sst_2 receptors at $[Ca^{2+}]_i$ since it abolishes the sst_2 receptor-mediated inhibition of $[Ca^{2+}]_i$ without affecting single-cell Ca^{2+} signals. On the other hand, CYN-154806 alone potently inhibits GH secretion through the involvement of pertussis toxin-sensitive G proteins. In conclusion, the present results demonstrate that SRIH inhibition of GH release in GC cells involves mechanisms either dependent or independent on SRIH modulation of $[Ca^{2+}]_i$. The implications of CYN-154806 inhibition of GH secretion are discussed.

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