Endocrinology, doi:10.1210/endo-126-4-1996
Endocrinology Vol. 126, No. 4 1996-2002

Effect of the Lectin Concanavalin-A on Calcium-Regulated Adenosine 3',5'-Monophosphate Accumulation in Bovine Parathyroid Cells*

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Abstract

Extracellular calcium (Ca^{2+}) is the major physiological regulator of parathyroid function; high Ca^{2+} decreases PTH secretion as well as reduces cAMP accumulation. There is an increasing body of evidence suggesting the presence of a receptor-like mechanism at the surface of the parathyroid cell which mediates these and other actions of Ca^{2+}. In the present studies we used the lectin Concanavalin-A (Con-A) to investigate the possible role of carbohydrate moieties in the regulation of cAMP metabolism by Ca^{2+} in bovine parathyroid cells, which is thought to involve inhibition of adenylate cyclase via activation of the guanine nucleotide regulatory protein G;. Pretreatment of parathyroid cells with Con-A for 15–60 min significantly reversed the inhibitory effect of high Ca^{2+} on dopamine-stimulated cAMP accumulation, reducing the inhibition at 3 mM Ca^{2+} from 70 ± 3% to 30 ± 3%. This effect was also observed in the absence of preincubation and with concentrations of Con-A as low as 40 μg/ml and was reversed by α-methyl-D-glucoside, a specific antagonist of the lectin. The lectin also reversed the inhibitory effects of Ca^{2+} (2–3 mM) on cAMP accumulation stimulated by isoproterenol and forskolin to a comparable extent. Prostaglandin F^{2a}-induced inhibition of cAMP accumulation (likewise mediated by G;) was, however, not reversed by Con-A, suggesting that the lectin did not have a generalized effect on the cell surface or on receptors inhibiting adenylate cyclase. Moreover, fluoride-induced inhibition of cAMP accumulation was not reversed by Con-A, providing additional evidence that the lectin did not act at or distal to G^i {i.e. modulate G^i, adenylate cyclase, and/or phosphodiesterase). The present study suggests that Con-A may modulate the actions of extracellular Ca^{2+} on parathyroid secretion, possibly modifying the interaction of Ca^{2+} with the cell surface by affecting carbohydrate moieities that seem to be important in the Ca^{2+}-sensing process. The structural element involved in Ca^{2+} sensing in the parathyroid cell may be a glycoprotein or closely associated with glycoproteins with carbohydrate chains containing a-methyl-D-glycoside. (*Endocrinology* **126:** 1996–2002,1990)

Footnotes

* This work was supported by USPHS Grants AM-35796 and AM-41 and institutional funds from the Joslin Diabetes Center.

Received October 9, 1989.