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JOURNAL ARTICLE

Atrial natriuretic factor inhibits the phosphorylation of protein kinase C in plasma membrane preparations of cultured Leydig tumor cells

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The peptide hormone atrial natriuretic factor (ANF) exerts its effect in a receptor-mediated fashion and the membrane-bound form of guanylate cyclase represents a biologically active ANF receptor; thus, cGMP has been considered a second messenger of ANF. To understand the mechanisms of ANF action, we have studied its effect on protein phosphorylation in the plasma membrane preparations of murine Leydig tumor (MA-10) cells, which overexpress guanylate cyclase-coupled ANF receptor molecules in high density. After pretreatment of the plasma membranes with ANF (100 nM), a marked decrease in phosphorylation of the 78-kDa protein kinase C (PKC) and the 240-kDa protein was observed. Phosphorylation of the 78-kDa PKC was also inhibited by cGMP (0.1 mM); however, phosphorylation of the 240-kDa protein was not affected by cGMP. The quantitative analyses, as determined by densitometric scanning, revealed that both ANF and cGMP inhibited phosphorylation of the 78-kDa PKC by approximately 75% and 45%, respectively. The inhibitory effect of ANF on phosphorylation of the 240-kDa protein was almost 90%, but cGMP did not show any discernible effect on its phosphorylation in plasma membranes of MA-10 cells. Phosphorylation of the 78-kDa PKC was stimulated by Ca2+ and phospholipids, and it immunologically cross-reacted with antiserum against brain PKC. Furthermore, in these plasma membrane preparations, the 78-kDa PKC was immunoprecipitated and its phosphorylation was inhibited by ANF. These data provide evidence for a new signal transduction mechanism of ANF that negatively regulates phosphorylation of the 78-kDa PKC and the 240-kDa protein in a cGMP-dependent and -independent manner in Leydig cells.

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