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Predominant contribution of the G protein-mediated mechanism to NaF-induced vascular contractions in diabetic rats: association with an increased level of G(qalpha) expression.

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Abstract

The purpose of this study was to determine the mechanism responsible for alterations in NaF-induced contractions of blood vessels from streptozotocin-induced diabetic rats. In the presence of AlCl(3), NaF (>/=7.5 mM) produced significantly greater contractions in diabetic aorta and mesenteric artery compared with age-matched controls. Pretreatment with 1 microM nifedipine eliminated the enhanced contractile responses of diabetic vessels to NaF, resulting in no difference in the magnitude of NaF-induced contractions between control and diabetic vessels. In the presence of 100 microM deferoxamine, an Al(3+) chelator, NaF-induced contractions of diabetic vessels were markedly attenuated, whereas only the responses to lower concentrations of NaF were reduced in control vessels. No significant difference was found in the peak amplitude of transient contractions induced by 10 microM cyclopiazonic acid between control and diabetic vessels. The addition of 10 microM okadaic acid produced attenuated contractions in diabetic vessels. These findings indicate no involvement of the inhibitory effects of NaF on endoplasmic reticular Ca(2+)-pump ATPase and protein phosphatases in the genesis of the enhanced responsiveness of diabetic vessels to NaF. Western blot analysis showed a 2.5-fold increase in the expression of G(qalpha) in diabetic aortic membranes. In contrast, the G(ialpha) level was modestly decreased and the G(salpha) and G(betagamma) levels were unchanged in diabetes. The present results suggest that enhanced vascular contractions to NaF in diabetes is attributed predominantly to a G protein-mediated Ca(2+) channel activation that results from markedly increased G(qalpha) expression in vascular tissues under this pathological state.

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