

We have compared the coupling mechanisms of rat renal alpha 1A- and alpha 1B-like adrenoceptors to inositol phosphate formation. The experiments were performed in parallel in native renal tissue preparations and in those where alpha 1B-adrenoceptors had been inactivated by treatment with 10 $\mu\text{mol/l}$ chloroethylclonidine for 30 min at 37 degrees C; renal slices were used in most experiments but isolated renal cells were also used in some cases. The Ca^{2+} chelating agent, EGTA (5 mmol/l), reduced noradrenaline-stimulated inositol phosphate formation in native but enhanced it in chloroethylclonidine-treated renal slices. The inhibitory effect of EGTA was not mimicked by 100 nmol/l nifedipine. Inactivation of 87% of cellular G_i by 16-20 h treatment with 500 ng/ml pertussis toxin did not significantly affect noradrenaline-stimulated inositol phosphate formation in isolated renal cells but abolished the inhibitory effect of chloroethylclonidine. The adenylate cyclase activator, forskolin (20 $\mu\text{mol/l}$), inhibited noradrenaline-stimulated inositol phosphate formation in native and chloroethylclonidine-treated slices, and the inhibitory effects of chloroethylclonidine treatment and forskolin were additive. Verdich JM, Tangen JD, Harris L, Platt SN, Lampe JW, and the NINDS rtPA Stroke Trial. The efficacy and safety of tissue plasminogen activator in the management of acute ischemic stroke. *N Engl J Med* 2000;342:1214-24. Toma H, Tateishi J, Oba J, Aoyama N, Iwamoto H, Kasama Y, Okada M, Masai Y, Takaku Y, Takahara T, and the NINDS rtPA Stroke Study Group. A randomized controlled trial of a combination of rtPA and aspirin (tissue plasminogen activator-aspirin) in the treatment of acute ischemic stroke. *N Engl J Med* 2000;342:1248-57.

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