Pathway-selective *l*-isoproterenol potentiation of pyramidal cell activation in field CA1 of the rat hippocampal slice, a poster presented at the Annual Meeting of the Society of Neuroscience (1995) by Cooper, D., Dahl, D., Epling, G., Nejtek, V., Moushegian, G. School of Human Development and Communication Sciences, University of Texas at Dallas, Richardson, Texas.

Results of an in-vitro study of the locus of adrenergic potentiation in rat hippocampus.

Description:
Norepinephrine of *l*- isoproterenol (a Beta- adrenergic agonist) produces potentiation of pyramidal cell activation by the Schaffer Collateral pathway (SC) in hippocampal field CA1. Whether this potentiation is due to direct effects of adrenergic agonists on pyramidal cells, or indirectly via modulation of SC is not known.
The locus of adrenergic potentiation was investigated in rat hippocampal slice. Independent pathways activating field CA1 pyramidal cells were stimulated by monopolar electrodes placed in mid-*striatum radiatum* (to activate SC) and in the heavily mylinated perforant pathway (PP) proximal to the hippocampal fissure. After Colbert and Levy, field potentiation profiles and baclofen were employed to verify isolation of the SC and PP. Intracellular recordings of excitatory postsynaptic potentials (EPSPs) were taken to alternating SC and PP stimulation. Activation of pyramidal cells by the weak PP to field CA1 was enhanced by application of bicuculline methiodide (a Gamma amino butyric acid agonist [1.0 microM])
Concentrations of *l*-isoproterinol in the range of 500 nM - 1.0 uM produced significant potentiation of SC evoked EPSPs, but were without effect on PP-evoked EPSPs. *In no experiment did PP-evoked EPSPs undergo potentiation in parallel to potentiation of the SC.* Initial EPSPs of only a few mV frequently potentiated to well beyond action potential threshold.
These results are consistent with the hypothesis that adrenergic potentiation of pyramidal cell activation in field CA1 probably involves a pre-synaptic locus.

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