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Suppression of noradrenaline synthesis in sympathetic nerves by carbidopa, an inhibitor of peripheral dopa decarboxylase

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Abstract

IT is generally believed that noradrenaline synthesis in postganglionic sympathetic nerves can be suppressed by drugs which inhibit the activities of tyrosine hydroxylase or dopamine β-oxidase[1,2], but not by inhibition of aromatic 1-amino acid decarboxylase (AAAD; dopa decarboxylase). We present here evidence that the administration to rats of carbidopa (MK-486; α-methyl-L-dopa-hydrazine), a peripherally acting inhibitor of AAAD[3], not only blocks the decarboxylation of exogenous L-dopa (for example, in capillary endothelia within the central nervous system, liver, and kidney[4,5]), but also suppresses the decarboxylation of endogenous dopa formed from ^{3}H-tyrosine within cardiac sympathetic nerve terminals. As a consequence, the drug inhibits noradrenaline synthesis in this tissue.

Access options