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Determination of Acetylcholine Release in the Striatum of Anesthetized Rats Using In Vivo Microdialysis and a Radioimmunoassay*

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A vertical-type in vivo microdialysis probe and a sensitive, specific radioimmunoassay (RIA) were used to study the mechanism of acetylcholine (ACh) release in the striatum of anesthetized rats. Without the use of physostigmine, a cholinesterase inhibitor, our RIA could still detect the amount of ACh present in the perfusate (5.6±0.6 fmol/min, n=16). Tetrodotoxin (1 μM) produced a significant decrease in the amount of ACh collected in the perfusate, suggesting that basal ACh determined under the present experimental conditions was related to cholinergic neural activity. Atropine (0.1—1 μM) applied topically via the dialysis probe did not affect the amount of ACh recovered in the perfusate in the absence of physostigmine. Addition of physostigmine (10 μM) to the perfusion fluid produced about a 100-fold increase in the amount of ACh collected. In the presence of physostigmine, topical administration of atropine and pirenzepine (0.01—1 μM) through a dialysis probe produced a further three- to fourfold increase in ACh output, whereas a slight increase was produced by AF-DX 116 at the highest concentration (1 μM). These results indicate that presynaptic modulation of ACh release in the striatum does not occur under basal conditions, and that presynaptic M_1 muscarinic receptors are involved in the modulation of ACh release when the ACh concentration is raised under certain conditions.

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