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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 ( $5.6 \pm 0.6$  fmol/min,  $n=16$ ).

Tetrodotoxin ( $1 \mu M$ ) 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 ( $0.1-1 \mu M$ ) 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 ( $10 \mu M$ ) 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 ( $0.01-1 \mu M$ ) 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 ( $1 \mu M$ ). 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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