

Title	Determination of acetylcholine release in the striatum of anesthetized rats using in vivo microdialysis and a radioimmunoassay
Sub Title	
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Publisher	共立薬科大学
Publication year	1991
Jtitle	共立薬科大学研究年報 (The annual report of the Kyoritsu College of Pharmacy). No.36 (1991.) ,p.50- 50
JaLC DOI	
Abstract	
Notes	抄録
Genre	Technical Report
URL	https://koara.lib.keio.ac.jp/xoonips/modules/xoonips/detail.php?koara_id=AN00062898-00000036-0050

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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 ($1 \mu M$) 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.

* 本報告は *J. Neurochem.* 57, 882—887 (1991) に発表.