

Title	Effects of TRH and DN-1417 on high potassium-evoked acetylcholine release from rat basal forebrain slices determined directly by radioimmunoassay
Sub Title	
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Publisher	共立薬科大学
Publication year	1989
Jtitle	共立薬科大学研究年報 (The annual report of the Kyoritsu College of Pharmacy). No.34 (1989.) ,p.89- 89
JaLC DOI	
Abstract	
Notes	抄録
Genre	Technical Report
URL	https://koara.lib.keio.ac.jp/xoonips/modules/xoonips/detail.php?koara_id=AN00062898-00000034-0089

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Effects of TRH and DN-1417 on High Potassium-Evoked Acetylcholine Release from Rat Basal Forebrain Slices Determined Directly by Radioimmunoassay

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1. High potassium (50 mM) -evoked acetylcholine (ACh) release from rat basal forebrain slices under conditions without an exogenous choline supply was determined using a radioimmunoassay for ACh.
2. A consistent amount of ACh release was observed at each repetitive stimulation and ACh content in brain slices was not altered by potassium stimulations. These results indicate the existence of a large intracellular releasable ACh store, which is independent of new synthesis from exogenous choline.
3. Atropine, even at a concentration of 10^{-6} M, did not affect the potassium-evoked ACh release. Thus, modulation of ACh release by the muscarinic autoreceptor was not revealed under the conditions employed.
4. Thyrotropin-releasing hormone (TRH, 10^{-4} M) caused a slight and statistically insignificant increase in potassium-evoked ACh release. DN-1417, a TRH analogue, at a concentration of 10^{-4} M significantly increased potassium-evoked ACh release. These findings indicate that DN-1417 is able to enhance ACh output independently of ACh synthesis from exogenous choline.

* 本報告は *Gen. Pharmacol.*, **20** (2), 239—242 (1989) に発表.