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Author	鈴木, 岳之(Suzuki, Takeshi) 藤本, 和子(Fujimoto, Kazuko) 大畑, 尚代(Ohata, Hisayo) 川島, 紘一郎(Kawashima, Koichiro)
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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

Takeshi SUZUKI, Kazuko FUJIMOTO, Hisayo OOHATA
and Koichiro KAWASHIMA

鈴木岳之，藤本和子，大畑尚代，川島紘一郎

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.

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