

背側縫線核由来セロトニンおよび5-HT_{1A}
受容体アゴニストのSM-3997による
海馬CA₁ニューロンの抑制

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INHIBITION OF HIPPOCAMPAL CA₁ NEURONS BY 5-
HYDROXYTRYPTAMINE, DERIVED FROM THE DORSAL RAPHE NUCLEUS
AND THE 5-HYDROXYTRYPTAMINE_{1A} AGONIST SM-3997

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ABSTRACT Electrophysiological studies, using chloral hydrate-anesthetized rats, were undertaken to determine whether hippocampal pyramidal neurons, receiving input from the medial septal nucleus, were affected by 5-hydroxytryptamine (5-HT) derived from the dorsal raphe nucleus. The pyramidal neurons in the CA₁ region of the hippocampus were classified into short- and long-latency neurons, based on their response to stimulation of the medial septal nucleus. Microiontophoretically applied atropine inhibited the generation of spikes upon stimulation of the medial septal nucleus in short-latency neurons, but had no effect on long-latency neurons. In the short-latency neurons, the stimulation-induced spikes of the medial septal nucleus were inhibited by conditioning stimuli applied to the dorsal raphe nucleus and iontophoretic application of 5-HT and the 5-HT_{1A} agonists, SM-3997 (3a α , 4 β , 7 β , 7a α - hexahydro-2-(4-(4-(2-pyrimidinyl)-1-piperazinyl)-butyl)-4,7-methano-1H-isoindole-1,3(2H)-dione dihydrogen citrate) and 8-OH-DPAT (8-hydroxy-2-(di-n-propylamino) tetralin). The conditioning effect of the dorsal raphe nucleus was antagonized by methysergide. However, in the long-latency neurons, the spikes elicited by stimulation of the medial septal nucleus were not affected by the conditioning stimulation of the dorsal raphe nucleus, or iontophoretically applied 5-HT. These results indicate that 5-HT, originating in the dorsal raphe nucleus inhibited hippocampal pyramidal neurons receiving

cholinergic input from the medial septal nucleus, but not those receiving non-cholinergic input from the medial septal nucleus. The drug SM-3997 inhibited the activity of hippocampal pyramidal neurons, that receive excitatory cholinergic input from the medial septal nucleus by acting on 5-HT_{1A} receptors.

抄録 内側中隔野より入力を受ける海馬錐体ニューロンが腹側縫線核 (DR) 由来セロトニンの影響を受けるか否かを検討する目的で、ラットを用いた電気生理学的研究を行った。CA₁ 錐体ニューロンは、内側中隔野 (MS) 刺激により誘発される反応の潜時により単潜時と長潜時ニューロンに分類された。イオントホレーシス法により投与したアトロピンは単潜時ニューロンの発火を抑制したが、長潜時ニューロンには影響しなかった。単潜時ニューロンにおいて、MS刺激誘発スパイクは、DR条件刺激により抑制されるとともに、セロトニンおよび5-HT_{1A} アゴニスト SM-3997 の投与により抑制された。一方、長潜時ニューロンはこれらの処置により影響を受なかった。以上の結果は、SM-3997 がMSよりアセチルコリン性入力を受ける海馬錐体ニューロンに対して、5-HT_{1A} 受容体を介し抑制作用を発現することを示唆する。

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