Title: Role of thalamic ventral posterolateral nucleus histamine H_2 and opiate receptors in modulation of formalin-induced muscle pain in rats

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PII: S1734-1140(17)30191-3
DOI: http://dx.doi.org/doi:10.1016/j.pharep.2017.05.001
Reference: PHAREP 719

To appear in:

Received date: 9-3-2017
Revised date: 18-4-2017
Accepted date: 5-5-2017

Please cite this article as: {http://dx.doi.org/

<AT>Role of thalamic ventral posterolateral nucleus histamine H_2 and opiate receptors in modulation of formalin-induced muscle pain in rats
<!--<RunningTitle>thalamic ventral posterolateral nucleus and muscle pain</RunningTitle>-->
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<ABS-HEAD>Highlights► • Intramuscular injection of formalin produced paw licking and paw flinching. ► • Ranitidine prevented histamine-, dimaprit- and morphine-antinociception. ► • Naloxone blocked morphine-, histamine-, and dimaprit-induced antinociception. ► • Histamine H_2 and opiate receptors modulate muscle pain at the PVL level.
 <ABS-HEAD>Abstract
<ABS-P><ST>Background</ST> Histamine and opiate systems contribute to supraspinal processing of pain. In the present study, we investigated the effects of microinjection of histamine and agonists and antagonists of histamine H_2 and opiate receptors into the thalamic ventral posterolateral nucleus on muscle pain in rats.
<ABS-P>
<ABS-P><ST>Methods</ST> The thalamic ventral posterolateral nuclei were bilaterally implanted with two guide cannulas. Muscle pain was induced by intramuscular injection of a diluted formalin solution (2.5%, 50 μl) into the belly of gastrocnemius muscle, and pain-related behaviors including paw licking duration and paw flinching number were recorded at five-min blocks for 60 min.
<ABS-P><ST>Results</ST> Formalin produced a biphasic pattern of pain-related behaviors. Ranitidine (a histamine H_2 receptor antagonist) alone did not affect pain intensity, whereas it prevented the antinociceptive activities of histamine, dimaprit (a histamine H_2 receptor agonist) and morphine (an opiate receptor agonist). Naloxone (an opiate receptor antagonist) alone increased pain, and inhibited histamine-, dimaprit-, and morphine-induced antinociception. Locomotor activity was not changed with these chemicals.
<ABS-P><ST>Conclusions</ST> Our results showed an interaction between histamine H_2 and opiate receptors at the thalamic ventral posterolateral nucleus in modulation of muscle pain.
<KWD>Keywords: Histamine; Histamine H_2 receptor; Muscle pain; Opiate receptor; Thalamic ventral posterolateral nucleus

Introduction

Histamine through H_1, H_2, H_3, and newly H_4 receptors participates in processing of learning and memory, feeding regulation, arousal induction and pain modulation [1-4]. Activation of H_1 and H_2 receptors at submedius (Sm) and ventral posteromedial (VPM) nuclei of the thalamus and inhibition of H_3 receptor at VPM, anterior cingulated cortex (ACC), primary somatosensory cortex (PSC) and dentate gyrus (DG) suppress pain responses [5-9]. Mu-, delta-, and kappa-opioid receptors exert potent actions on local peripheral, spinal and supraspinal modulation of pain [10]. These receptors also imply to explore the supraspinal mediating effects of various neurotransmitters such as noradrenalin, dopamine, glutamate and histamine [5, 11-13]. At this