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Persistent ERK activation by the histamine H4 receptor in spinal neurons underlies chronic itch

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Transient extracellular signal-regulated kinase (ERK) activation through H1 receptor in the spinal cord triggers histamine-induced acute itch. However, whether persistent ERK activation plays an important role in chronic itch development remains unclear. This study investigated the role of spinal ERK activation in chronic itch. Results showed that repetitive 2,4-dinitrofluorobenzene (DNFB) painting in the nape of mice evoked not only induced scratching but also sustained, spontaneous scratching. In addition, DNFB induced itching rather than nociception, as demonstrated using a cheek model. Furthermore, ERK was persistently activated in the spinal cord of DNFB treated- mice and intrathecal inhibition of pERK suppressed both the spontaneous itching and ERK activation. ERK activation was observed in neurons but not in glia cells as chronic itch developed. Finally, DNFB-induced spontaneous itching behavior and ERK activation were largely inhibited by the histamine H4 receptor antagonist JNJ7777120, but not by H1 receptor antagonist chlorpheniramine. Our results indicate that persistent ERK activation via the histamine H4 receptor in spinal neurons underlies DNFB-induced chronic itch.

Biography

Ling Zhang is a Professor and Principle Investigator at Department of Neurobiology, Tongji University School of Medicine. She has received her PhD degree at Fudan University in 2005. After that, she pursued her Postdoctoral Fellowship at Pain Medical Center of Brigham and Women's Hospital, Harvard Medical School.

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