Is secondary hyperalgesia mediated by type I AMHs?

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Secondary hyperalgesia refers to the increase in pain sensitivity spreading beyond the site of injury. It can be induced experimentally by transcutaneous high frequency electrical stimulation[1](HFS). Secondary hyperalgesia seems characterized by enhanced pain to mechanical but not heat stimuli[2] and likely mediate by type-I nociceptors. It has been shown that when long duration heat stimuli are used the thresholds of type-I and type-II nociceptors are similar[3]. Therefore one may expect that if type-I mediate secondary hyperalgesia, long duration heat stimuli delivered above the type-II heat threshold, will elicit increased heat pain sensitivity in the area of secondary hyperalgesia. Here we investigate the intensity of perception elicited by sustained heat stimuli after HFS. Mechanical and long duration heat (30 seconds) stimuli were delivered on both forearms before and 20 minutes after HFS. Intensity of perception was instantaneously recorded during heat stimuli with an analogic rating scale box. While HFS induced a significant increase of the perception of mechanical stimulation, we only observed a significant enhancement of the perception of heat stimuli in the first 4 seconds of the long duration stimulation. This heat hyperalgesia does not seem compatible with the response profil of type-I nociceptors to sustained heat stimuli.

1: (HFS, LaMotte et al., 1991; Klein et al, 2004)
2: (Ali et al. 1996),
3: Treede et al. (1998)