Increased C-fiber Activities to Cold in Adjuvant-monoarthritic Rats was not Accompanied by Increased Expression of TREK1 and CMR1 mRNAs

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[**Aim**] Cold allodynia is an annoying symptom in patients with chronic inflammation. We reported last year that adjuvant-monoarthritic rats were hypersensitive to cold and that the cold response of a few primary afferent fibers increased in these animals. In the current study, we further examined nerve activities and probed molecular mechanism of the observed hypersensitivity to cold.[**Methods**] Persistent inflammation was induced by an injection of complete Freund's adjuvant solution into the tibio-tarsal joint. We carried out single nerve recordings from the sural nerve of control and inflamed animals 2 to 3 weeks after the adjuvant injection. Cold stimuli down to 2°C were applied to the receptive fields using a Peltier thermode. Besides, we measured mRNAs for ion channels possibly responsible to cold transduction, CMR1 and TREK-1, by semi-quantative RT-PCR.[**Results**] The response to cold stimuli in persistently inflamed animals was significantly facilitated in C-low threshold mechanoreceptors and the incidence of cold-sensitive fibers was increased in C-nociceptors. No change was detected in the response of C-cold fibers. The expression levels of CMR1 and TREK-1 mRNAs did not change significantly.[**Conclusion**] These results suggest changed sensitivities to cold of peripheral C-fibers are possibly involved in cold hypersensitivity in persistent inflammation. Expression level of CMR1 and TREK-1 may not be implicated in these changes.