Animal Model of Delayed Onset Muscle Soreness

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[Aim] Eccentric muscular contraction (ECC) is known to cause delayed onset muscle soreness (DOMS) in humans. The similar exercise has been used in rats to induce DOMS, however, existence of tenderness has not been clarified. We examined sensitivity to mechanical stimulation of muscle by behavioral and immunohistochemical studies after ECC in rats. **[Methods]** Extensor digitorum longus muscle was given repetitive ECC by electrical stimulation of nerve innervating the muscle. Mechanical withdrawal threshold was measured with Randall-Selitto apparatus before, 1, 2, 3 and 7 days after the exercise. C-Fos immunoreactivity (Fos-ir) in the dorsal horn of the spinal cord was examined on the second day after ECC. **[Results]** Withdrawal threshold to the mechanical stimulation of the muscle significantly decreased and reached the lowest value 2 days after ECC from 96.6 g to 72.9 g ($P < 0.001$). The number of Fos-ir neurons in the superficial dorsal horn, which is known to transmit nociceptive information to the upper brain at L4 of the spinal segment significantly increased in rats with mechanical stimulation to the exercised muscle when compared with groups having no ECC and/or mechanical stimulation. ($P < 0.001$). **[Conclusion]** These results suggest that tenderness of the eccentrically exercised muscle in rats exists, and that this animal model is useful for investigating the mechanism of DOMS.

Genetic Subtype and Functional Characteristics of T-type Ca^{2+} Channel in Mouse Embryonic Hearts

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[Aim] T-type Ca^{2+} channels are implicated in cardiac cell growth under physiological development and pathological status. We analyzed the subtype expression of T-type channel during development and after myocardial infarction (MI) by functional and molecular studies. **[Method]** T-type currents ($I_{Ca\text{-}T}$) were recorded in ventricular myocytes from mice of E9.5 and E18 by a patch clamp method. Expression of subtype gene of T-type channel, α1G and α1H, in cardiac ventricle from E9.5, E18 and adult, and in hypertrophied region 4 weeks after MI were quantified by a real time PCR. **[Result]** $I_{Ca\text{-}T}$ were detected at E9.5 and at E18 with same current densities. There were no differences in voltage dependence of activation and time dependency of recovery from inactivation between $I_{Ca\text{-}T}$ at E9.5 and that at E18, while $I_{Ca\text{-}T}$ at E9.5 was inactivated at more negative potential than that at E18. Low dose of Ni^{+} (30μM) blocked $I_{Ca\text{-}T}$ both at E9.5 and at E18, the inhibition was comparable to those described for cloned a1H channel. Expression of α1H mRNA was abundant at E9.5, but decrease from E18 to adulthood. However, α1G mRNA was substantially expressed both at E18 and at adulthood. In the remodeled region after MI, α1H mRNA expression was remarkably increased about 3-folds v.s. sham, while α1G mRNA expression was unaffected. **[Conclusion]** The dominant gene subtype of T-type channel is α1H in early cardiac development. Moreover, α1H subtype generates functional T-type channel during embryonic period. Recapturation of a fetal gene program (α1H dominance) is included in remodeled hearts after MI.