Muscle tenderness from exercise: mechanisms?

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When we carry out a bout of intense exercise we can, at times, reach the point where the exercise becomes painful. But as soon as we stop, the pain subsides. However, there is one form of exercise, eccentric exercise, where there is typically no pain immediately after the exercise, but we find ourselves stiff and sore next day. The soreness can persist for 4–5 days, depending on the severity of the exercise.

The reason for the delayed soreness is that, in someone unaccustomed to eccentric exercise, it leads to localized areas of damage in muscle fibres. The present-day view is that the inflammatory response triggered by the damage leads to a sensitization of muscle nociceptors. For a review, see Proske & Morgan (2001). The soreness, referred to as delayed onset muscle soreness (DOMS), has a number of features that distinguish it from other forms of muscle pain. Incidentally, the accompanying sensation of muscle stiffness is the result of a sensitization towards mechanical stimuli that do not evoke any sensations of pain in an unexercised individual. It is for that reason it should be referred to as a tenderness, rather than a soreness. It has led to the view that DOMS is a type of hyperalgesia and is distinct from other kinds of muscle pain such as myositis, where there is typically some chronic pain associated with tonic activity in nociceptors (Berberich et al. 1988). Because of the unusual features of DOMS and because of the debilitating effects DOMS may have on the performance of competing athletes, there has been a need to find out more about the underlying neural mechanisms. Studying pain mechanisms in human subjects is fraught with difficulties, and only limited insight can ever be achieved, especially about central mechanisms. These are circumstances where animal models can play an important role.

This issue of The Journal of Physiology contains the first description of an animal model suitable for the study of neural mechanisms underlying DOMS (Taguchi et al. 2005). The extensor digitorum longus (EDL) muscle of anaesthetized rats was contracted eccentrically numerous times and the animals were then allowed to recover. Interestingly, on recovery, the exercised rats showed no behavioural evidence of muscle tenderness. On day 2 after the exercise, one group of animals was subjected to periods of mechanical compression of the exercised muscle. Only in this group, not in the exercised animals that were not given compressions, was there evidence of up-regulation of c-Fos expression in the spinal dorsal horn, at the level of entry of afferents coming from EDL. The authors conclude that, given the diffuse and dull character of DOMS, it makes it likely to be a sensation mediated by C-fibre afferents.

However, there is evidence that the story of DOMS is not as simple as that. It has been shown that the discomfort experienced from strong mechanical compression of unexercised human calf muscles can be reduced by superimposing 80 Hz vibration on the compression. This is probably an example of the phenomenon ‘rubbing it makes it better’. If this procedure is repeated in someone with the symptoms of DOMS, the vibration exacerbates the discomfort, rather than alleviating it (Weerakkody et al. 2003). The result raises a number of issues. Presumably as a result of the exercise, there has been a change in the central processing of the vibration-evoked afferent signals. Vibration frequencies of up to 120 Hz reduced the pain threshold (Weerakkody et al. 2003). It makes it unlikely that the afferents involved are in the C-fibre range, given the long refractory period for unmyelinated fibres. Other potential candidates for the vibration response are mechanically sensitive group III afferents (Paintal, 1960). It has also been shown that the pain threshold to mechanical pressure in someone with DOMS rises if the large muscle afferents, within the group I–II range, are blocked (Barlas et al. 2000; Weerakkody et al. 2005). It raises the possibility that large muscle afferents can contribute to DOMS and that DOMS could be seen to have features of a secondary hyperalgesia and allodynia. Direct evidence for mechanisms of this kind can now be sought in animal models of the type described by Taguchi et al. (2005).

References