A morphological study of delayed muscle soreness

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Summary. Biopsies, taken up to 1 week postexercise, from the soleus muscles of 5 healthy males (20–34 years old) suffering from pronounced exercise-induced delayed muscle soreness were analyzed morphologically. There was no evidence for ischemic tissue injury or mechanical fibre disruption. However, at the subcellular level frequent myofibrillar disturbances, especially with regard to the Z-bands, were noted. Thus, the contractile machinery of overloaded muscle fibres seemed to be partially distorted several days following exercise.

Most people, especially those engaged in sporadic physical exercise, experience severe muscular discomfort 1–3 days after unusual heavy physical exercise. This condition is termed 'delayed muscle soreness'. The major symptoms are muscular stiffness, tenderness, and pain, especially when making active movements. The signs are firm, tender and weak musculature. The pathophysiology behind this condition is not known, although many theories exist[1–6].

Previous workers using experimental animals have found considerable changes in muscle morphology following excessive exercise[7–9]. As it is known that running downstairs is one type of exercise that gives rise to pronounced delayed muscle soreness in calf muscles, it was decided to investigate whether this type of exercise would cause ischemic necrosis of single muscle fibres or mechanical fibre rupture in humans. Muscle biopsies from 5 healthy males (age 20–34 years) were analyzed by means of morphological techniques. The men were asked to run rapidly 10 times from the 10th floor down the stairs to the ground floor. The only resting periods consisted of those occasions when the subjects went up to the 10th floor by the elevator. They all suffered from intense calf muscle discomfort during the subsequent week, especially during the first 2–3 days following the exercise program. Biopsies were obtained alternatively from the right and left m. soleus by open surgical technique 2 weeks before the exercise and, from the contralateral side, 2 and 7 days after exercise. Each biopsy was initially divided into 2 halves, one of which was prepared for enzyme histochemistry[10], the other for electron microscopy. The biopsy part for electron microscopy was carefully mounted with nails on a cork plate so that the muscle fibres during the fixation remained at their approximate rest length. This was taken to be the length at which the muscular fascia became taut. Prefixation was performed in glutaraldehyde and postfixation in osmium tetroxide. Vestopal was used for plastic embedding.

The muscle fibres, both before and after exercise, as observed in sections specifically stained for oxidative enzymes and myofibrillar adenosine triphosphatase, were always seen tightly packed in well organized fascicles (fig. 1). Neither frequent focal nor diffuse fibre abnormalities were observed in any of the specimens. Furthermore, there was no evidence for presence of regenerating fibres. The relative number of different fibre types and their sizes

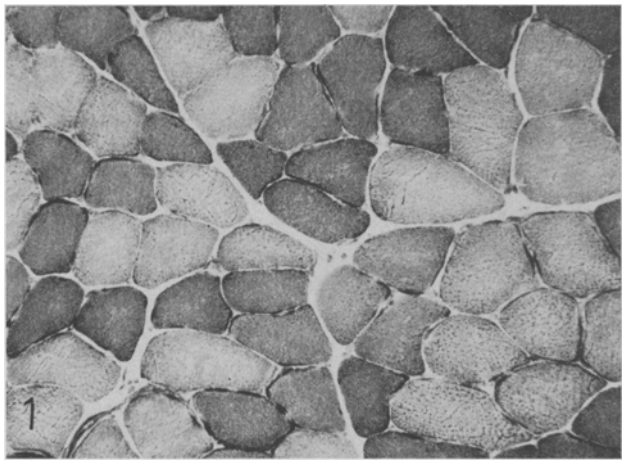


Fig. 1. Soleus muscle biopsy obtained 2 days after unusually heavy physical work when the subject experienced a severe muscle soreness. The section has been treated for visualization of oxidative activity (TPNH). The overall muscle morphology is normal. No necrotic fibres, indicating ischemic tissue damage or total fibre rupture, are seen. × 125.

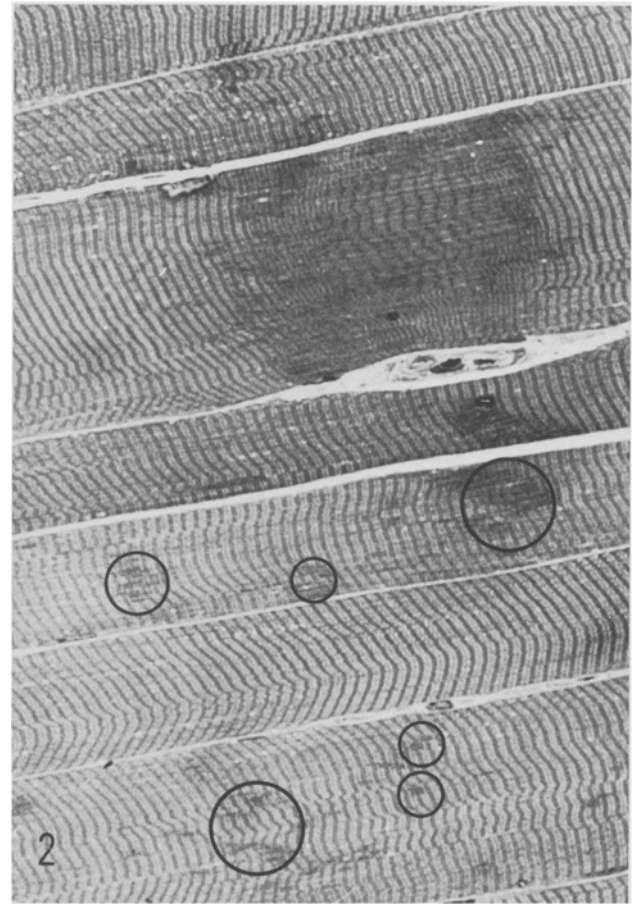


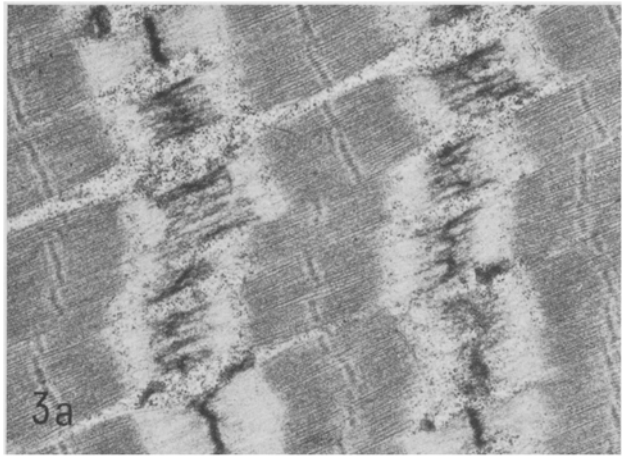
Fig. 2. Toluidine blue stained 1-µm-thick survey plastic section showing striated muscle fibres from the same biopsy as in fig. 1. The striation pattern is at places disturbed. A segment of 1 fibre (upper half of the figure) is severely disorganized. Other fibres show frequent focal disturbances, some of which are encircled. × 500.

remained unchanged after exercise. In summary, these results indicate that there were no signs at cellular level either of ischemic fibre necrosis or of fibre rupture in the specimens from the sore muscles. Similar findings have been made by other workers[5].

However, at subcellular level abnormalities were seen in the biopsies obtained 2 days postexercise. Frequent focal disturbances of the characteristic cross-striated band pattern were revealed in semithin (1-µm-thick) toluidine-blue stained survey sections of plastic embedded specimens (fig. 2). The total area showing focal disturbances in preparations obtained 2 days postexercise was estimated, by means of morphometric methods, to be at least 3 times larger than in comparable sections from control muscles as well as in specimens obtained 7 days after exercise. At the ultrastructural level the disturbances were found to originate from the myofibrillar Z-band which showed a marked broadening, streaming and, at places, total disruption (fig. 3). In some cases only 1 single Z-band of 1 myofibril was affected (fig. 3a), whilst there was also evidence for involvement of several sarcomeres and myofibrils (fig. 3b). The myofilamentous material in sarcomeres adjacent to the affected Z-bands was either supercontracted or disorganized and out of register. The Z-bands, which normally have a regular and complex fine structure[11], were often observed to have gaps in their lattice pattern even in areas where neither obvious broadening nor streaming was seen. The present findings indicate that a much greater Z-band disorganization occurs in biopsies obtained 2 days postexercise than those obtained prior to exercise. Other workers have reported some Z-band streaming in healthy individuals who have not undergone any preceeding conscious excessive physical effort[12]. The immediate interpretation of the findings is that the high myofibrillar tension developed during activation of the contractile material, i.e. the interdigitating arrays of thin and thick myofilaments, has resulted in some mechanical disruption of the Z-bands. These Z-bands, or in 3-dimensional terms, the Z-discs, connect adjacent sarcomeres to each other. The findings therefore indicate that the Z-bands during overloading constitute a weak link in the myofibrillar contractile chain. A disruption of myofibrils may result in a formation of protein components (such as globular proteins and degraded Z-proteins) and subsequent releasing of protein-bound ions which may cause oedema and thereby give rise to exercise-induced muscle soreness.

The appearance of delayed muscle soreness has mainly been connected with eccentric work[2,5,13], which is an important component in most exercises, for example, running downstairs. This type of work causes greater tension per active motor unit than corresponding concentric work and may therefore increase the risk for mechanical damage to the myofibrillar material as has been observed in the present work (the Z-discs).

Our results are not necessarily direct proof for the theory of mechanical Z-disc disruption as we only have data obtained 2 and 7 days postexercise and no biopsies were taken immediately after the work. Therefore, the structural disturbances may also be secondary resulting from an activation of lysosomal enzymes[8,9,14], bringing about a concomitant inflammation[6,13,15]. However, it does appear that the overloaded muscle fibres seem to have their contractile machinery partially distorted. As these fibres are probably predisposed to a total fibre rupture the risk for a serious rupture of a larger part of the muscle might be increased.



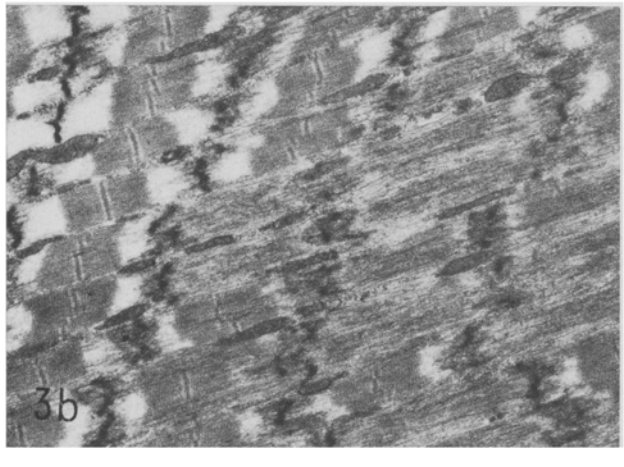


Fig. 3. Electron micrographs showing fibres from the specimen shown above (fig. 2). The cross-striated myofibrillar band pattern is disturbed at places. Z-band broadening and streaming is seen comprising either only single Z-bands (a), or several sarcomeres and myofibrils (b). The myofilamentous material in sarcomeres adjacent to the affected Z-bands, is supercontracted or disorganized, $a \times 12{,}000$; $b \times 7500$.

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