The effect of reactive oxygen species on muscle fatigue at room temperature compared to body temperature

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The production of reactive oxygen species (ROS), in particular superoxide anion radicals, plays a significant role in the modulation of muscle function (Clarkson & Urso, 2003). During intense exercise, there is an increased production of ROS, which leads to oxidative stress on muscle. Such stresses are thought to lead to the loss of contractile function, reduction in Ca^{2+} handling and contribution to fatigue (Reid, 2001). Also, it has been shown that increased temperature results in muscle dysfunction due to oxidative stress (van der Poel & Stephenson, 2002).

The present study is concerned with muscle performance during several bouts of fatigue at room temperature compared to body temperature. In particular, the influence of ROS at these temperatures is assessed in terms of onset of fatigue and maximum force.

Small muscle bundles (5 - 10 fibres per bundle) were dissected from the flexor brevis muscle of mice and were subjected to a fatigue protocol at room temperature (25°C) and at body temperature (37°C). Muscles were fatigued until force reached 50 % of the initial force. For each preparation, bundles were subjected to three fatigue runs (R1, R2, R3) allowing adequate time for muscles to recover between each run (45 minutes).

It was observed that there was no significant difference in the time taken for muscles to fatigue to 50% of the maximum force ($T_{1/2}$) at 25°C for R1, R2 and R3. Tiron (20mM), a free radical scavenger, was applied for 30 minutes between R1 and R2 and this treatment had no significant effect on the $T_{1/2}$ of R2 and R3 at 25°C. At 37°C however, $T_{1/2}$ was reduced to 65 ± 6 % for R2 and 26 ± 11 % for R3 compared to the $T_{1/2}$ for R1. When muscle preparations were treated with 20mM tiron, $T_{1/2}$ recovered to 106 ± 16 % for R2 and 103 ± 15 % for R3 compared to the value of R1. These results show that the ROS production has a profound effect on muscle fatigue at 37°C.

At 25°C there was no significant change in the fall of maximum force between each of the fatigue runs. At 37°C however, there was a significant decline in the maximum force when comparing R2 and R3 ($P<0.05$); and R1 and R3 ($P<0.01$). Treatment with tiron significantly reversed the decline of maximum force at 37°C. These results suggest that the production of free radicals at higher temperatures adversely affect the contractile properties, which are in some way responsible for the observed decline in maximum force.

The present study clearly shows that multiple bouts of fatigue at body temperature, in contrast to room temperature, progressively decreases muscle performance in terms of the onset of fatigue and maximum force development. Reduced muscle performance at 37°C appears to be partly caused by an increase in the production of ROS.

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