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High Intensity Exercise Metabolism and Muscle Function: Implications for Performance

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During brief, high intensity exercise, rapid changes in metabolism and muscle function occur. This may ultimately result in an inability to maintain performance, force or required exercise intensity. These processes collectively contribute to the phenomenon of fatigue Hermanson [1], Wootton and Williams [2] investigated the influence of recovery duration on repeated maximal sprints. The exercise task was randomly assigned and consisted of five 6-sec maximal sprint bouts, with either 30 or 60-sec recovery periods between each sprint. The test protocol used was similar to that of the Wingate test Bar-Or et al. [3]. Loadings were pre-determined to ensure that each subject would achieve the maximal power output attainable, while pedalling within the range of 150 to 160 rpm. The results showed that the capacity to perform repeated 6-sec bouts of maximal exercise on cycle ergometers was markedly influenced by the preceding number of sprints. The study also demonstrated that muscle contraction was dependent on the ability to recover muscle performance following brief maximal intensity exercise. Effects of recovery duration on performance and fatigue during multiple treadmill sprints was investigated by Holmyard et al. [4]. Ten rugby union backs volunteered to participate in the study.

A non-motorised treadmill was used for the sprint tests which allowed the subjects to run at unrestricted speeds. Fatigue was recorded as a decrease in running speed.

The experimental protocol consisted of ten 6-sec maximal sprints, with either a 30-sec or 60-sec recovery period between each successive sprint. The results obtained showed that performance during brief duration, multiple treadmill sprinting was affected by both the recovery interval and by the preceding number of sprints. With 30-sec recovery only 5 sprints could be performed before fatigue influenced power outputs. Alternatively, 60-sec recovery duration enabled power outputs to be maintained throughout the duration of testing. The larger decrease in performance observed with the 30-sec recovery interval may be due to an incomplete resynthesis of PC and also a possible greater acidosis. This may have resulted from the limited time for translocation of H^+ from the muscle to blood. It has been suggested that H^+ causes fatigue by either inhibiting energy provision from anaerobic glycolysis through moderating the activity of phosphofructokinase (PFK) or by affecting the contractile mechanism itself Hermanson [1]. The maximal rate of energy expenditure cannot exceed the activity of the ATP hydrolysing enzymes (ie muscle ATPase activity). Myofibrillar ATPase activity has been determined during maximal static contraction in skinned human muscle fibre to 0.10, 0.27 and 0.41 $mmol.l^{-1}s^{-1}$ in type I, IIA and IIB fibres respectively, Stienen et al. [5]. Assuming a Q_{10} of 2, 3.3l of H_2O per kg^{-1} dry mass of muscle and 2.7 times higher energy turnover during maximal dynamic exercise than static contraction Potma et al. [6] it can be calculated that maximal ATP expenditure is 6.5, 17.6 and 26.6 mmol ATP kg^{-1} dry mass in type I, IIA and IIB fibres respectively. This value approximates to the value observed in mixed muscle during 10s of maximal cycling (15 mmol ATP kg^{-1} dry mass; Jones et al. [7]. It therefore seems plausible that the release of energy during short bursts of activity (<5 s) is not limited by the rate of ATP supply but rather by limitation in ATP hydrolysis.

The higher degree of PCr depletion Hiroven et al. [8] and plasma NH_3 accumulation Hageloch et al. [9] during the initial phase of sprinting in sprint trained subjects support this contention. The amount of energy that can be produced from PCr is rather small and is limited by the intramuscular stores of PCr. Fast twitch fibres contain 15 - 20% more PCr than slow - twitch fibres Soderlund et al. [10] which is in accordance with the higher glycolytic capacity of this fibre type. With the maximal rate of PCr breakdown one would expect complete depletion of PCr within 10s Jones et al. [7]. However, PCr breakdown can contribute to ATP generation for more than 20 s because ATP is supplied from other energy sources and because energy expenditure decreases after a few seconds of contraction. Following 10 s of maximal exercise the power output decreases Nevill et al. [11] Hiroven et al. [8]. These first signs of fatigue have been shown to correlate with substantial decreases in muscle PCr. On the basis of thermodynamic considerations the maximum rate of PCr breakdown and therefore ATP generation would fall when the PCr content decreases. Availability of PCr may therefore be a limiting factor for power output even before the muscle content of PCr is totally depleted. This may partly explain why the power output decreases after 5 s of maximal cycling despite the fact that a considerable portion of PCr remains in the working muscle Sahlin et al. [12]. Maximal force is related to muscle PCr both during contraction and the recovery period. Similarly, after maximal cycling, peak power is restored with a similar time course as PCr Nevill et al. [11]. Recent studies have demonstrated that the muscle store of total creatine (PCr+Creatine) can increase by about 10-20% after oral creatine supplementation Harris et al. [13]. Creatine supplementation was shown to increase performance during high intensity exercise in some studies Balsom et al. [14] Greenhaff et al. [15] Earnest et al. [16] but not in others Barnett et al. [17] Deutekom et al. [18].

Post-exercise hypoxanthine Balsom et al. [14] and plasma NH_3 Greenhaff et al. [15] were reduced following creatine supplementation despite the fact that there was an increase in work performed. These findings support the hypothesis that limitations to energy supply are a major cause of fatigue during high intensity exercise. Based on the in vitro experiments of Cooke et al. [19] and the in vivo experiments of Wilson et al. [20] it has been suggested that increases in Pi may contribute to fatigue. Concomitant with the decline in PCr there is almost a stoichiometric increase in Pi and the observed correlation between PCr and force

during exercise and recovery may therefore be an effect of increased Pi and not energy deficiency per se Sahlin et al. [12]. However, creatine supplementation increases pre - exercise PCr Harris et al. [13] and therefore one could expect augmented release of Pi and an earlier onset of fatigue. The finding that performance is improved following creatine supplementation cannot be reconciled with the hypothesis that increases in Pi is a major cause of fatigue Woledge [21].

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