Short Commentary

Metabolic and hormonal factors influencing high intensity exercise, recovery, and fatigue

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Abstract

The purpose of the paper is to consider the metabolic mechanisms contributing to high intensity exercise performance, recovery, and fatigue. A further purpose is also to consider the total stress response to exercise by outlining the catecholamine responses to exercise performance. Concentrations of Noradrenaline (NA), Adrenaline (A) and have been found to reach higher values during short duration high intensity performance compared to longer duration low intensity activities. The secretion rates of catecholamines are related to metabolic and psychological stress. Knowledge of the metabolic stress pathways and hormonal interactions are important for training programme design and our understanding of high intensity fatigue mechanisms.

Introduction

During brief, high intensity exercise, rapid changes in metabolism and muscle function occur. This may result in an inability to maintain performance, force or required exercise intensity. These processes collectively contribute to the phenomenon of fatigue [1,2] 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, on a cycle ergometer with either 30 or 60 sec recovery period between each sprint. The test protocol used was similar to that of the Wingate test [3].

Loadings were pre-determined to ensure that each subject would achieve the maximal power output attainable, while pedaling within the range of 150 to 160 rpm.

The results demonstrated 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 highlighted 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 [4]. Ten rugby union backs volunteered to participate in the study. A non-motorized 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 a possible greater acidosis. This may have resulted from the limited time for translocation of Hydrogen Ions (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 [1]. The maximal rate of energy expenditure cannot exceed the activity of the ATP hydrolyzing enzymes (i.e., muscle ATPase activity). Myofibrillar ATPase activity has been determined during maximal static contraction in skinned human muscle fiber to 0.10, 0.27 and 0.41 $mmol.l^{-1}s^{-1}$ in type I, IIA and IIB fibers respectively [5].

Assuming a Q10 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 [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 fibers, respectively. This value approximates to the value observed in mixed muscle during 10s of maximal cycling (15 mmol ATP kg^{-1} dry mass) [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 by limitation in ATP hydrolysis.

The higher degree of PCr depletion [8] and plasma ammonia (NH3) accumulation [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 fibers contain 15 - 20% more PCr than slow - twitch fibers [10] which is in accordance with the higher glycolytic capacity of this fiber type. With the maximal rate of PCr breakdown one would expect complete depletion of PCr within 10 s [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 [11,8]. These first signs of fatigue have been shown to correlate with substantial decreases in muscle PCr. Based on 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 power output decreases after 5 s of maximal cycling even though a considerable portion of PCr remains in the working muscle [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 [11].

Previous studies have demonstrated that the muscle store of total creatine (PCr + creatine) can increase by about 10-20% after oral creatine supplementation [13]. Creatine supplementation was shown to increase performance during high intensity exercise in some studies [14-16] but not in others [17,18].

Post - exercise hypoxanthine [16] and plasma NH3 [15] were reduced following creatine supplementation even though 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 [20] and the in vivo experiments of Wilson et al. (1988) it has been suggested that increases in Pi may contribute to fatigue. Concomitant with the decline in PCr there is 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 [12]. However, creatine supplementation increases pre - exercise PCr [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 [21].

Metabolic stress and high intensity exercise

Total stress response to exercise is reflected by the adrenal medulla response. This may be measured by the accumulation of Adrenaline (A) and Noradrenaline (NA). This increase may be markedly influenced by the effect of physical exertion, cold and emotional factors. Plasma catecholamine levels also increase as a result of both increases in the duration of exercise and the severity of exercise [22]. The plasma concentrations of catecholamines have been shown to increase during different types of aerobic and anaerobic exercises [23].

However, it has been found that plasma adrenaline increase in heavy high intensity exercise may be higher than in aerobic exercise [24].

Different high intensity activities such as sprinting [25] and cycle ergometry [26,27] as well as heavy resistance exercise training [28] have all been found to elevate plasma catecholamine levels. At identical submaximal exercise levels several authors observed [29] a lesser increase in free plasma Adrenaline (A) and Noradrenaline (NA) in endurance trained athletes than in sedentary subjects. Conversely, [30,31] found that endurance trained athletes compared to untrained ones exhibited not only similar plasma NA but also higher plasma A concentrations. Brief high intensity exhaustive exercise has been shown to increase plasma catecholamine levels to values higher than those observed during aerobic activity [29]. Therefore, it may be possible that sprinters exhibit a higher adrenal response to sympathetic activity. It has often been emphasized that feedback mechanisms are important for the hormonal response to exercise [32]. A reduction in plasma glucose levels during exercise gives rise to an increase in the plasma concentration of adrenaline, and it has been postulated that increasing adrenaline secretion may function as a safety backup mechanism, preventing hypoglycemia and lack of substrate in the working muscle [31]. It seems, however, that rapid changes in endocrine and metabolic responses found at the onset and the cessation of exercise, occur too quickly to be explained by feedback mechanisms only. This may be partly explained by the direct stimulation from the motor centers of the brain, the feed - forward or central command mechanisms which are also involved with respiratory and circulatory regulation [33]. These mechanisms will also be active in substrate mobilization during exercise. Physical training may alter the hormonal response of several hormones to a given exercise workload.

Conclusion

The release and rate of release of hormones are controlled in response to specific needs.

The rates of secretion should be related to their metabolic functions and needs of a particular circumstance i.e., whether high or low intensity [34].

Previous experiments have demonstrated the importance of anaerobic glycolysis in the resynthesis of Adenosine Triphosphate (ATP) during high intensity performance. Exercise of this intensity although anaerobic, provides a severe challenge to the control of the cardiovascular system and elevates sympathetic activity. The stimulation of glycogenolysis by adrenaline has been demonstrated, and significant correlations have been observed between ATP resynthesis and plasma adrenaline concentration [25]. These studies have also found that during maximal exercise large concentrations in circulating adrenaline and noradrenaline occur, suggesting increases in the stress response to high intensity performances. Knowledge of fatigue and stress mechanisms are important in the development of high intensity training programmes. This information also provides valuable knowledge in relation to fatigue and recovery profiles during and following high intensity exercise performances.

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