activation. Plasma leucine concentration tended to be higher for the HP condition but during the second hour of HP exercise there was a 12% decrease ($P < 0.05$), which coincided with a 21% decrease in leucine oxidation ($P < 0.05$). There was a 34% reduction in whole body protein synthesis during exercise ($P < 0.005$) from 126 μmol kg$^{-1}$ h$^{-1}$, and diet had no further effect. The results suggest that increased dietary protein does not stimulate BCAA oxidative capacity in skeletal muscle. The higher leucine oxidation in the HP condition may be due either: to the increased plasma leucine concentration being sufficient to increase flux through the muscle oxidative pathway without activation of BCKADH; or possibly to an increase in liver BCKADH activation as occurs in rats. The results provide evidence that the body’s capacity to utilize dietary amino acids for protein synthesis is not enhanced by a high protein diet.

**Muscle temperature and short-term power output following prolonged exercise in humans**

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In the present study we examined the effect of prolonged high intensity exercise (45 min; 80% $V_{O_{2 \max}}$; 120 rev min$^{-1}$) on the instantaneous peak power (IPP) measured immediately after prolonged exercise and in a second series of experiments after 6 min of recovery in eight cyclists. IPP was measured during the log extension phase by means of special pedals instrumented with strain gauges. During these experiments the cycle ergometer was switched to operate in an isokinetic mode so that pedalling rate was controlled at 120 rev min$^{-1}$. This rate is close to the optimum velocity for power output in this form of exercise (Sargeant & Beelen, 1993). No significant difference in IPP, immediately after the prior exercise (1466 ± 220 W) was found when compared with IPP measured under control conditions (1471 ± 209 W). This finding would suggest that the 45 min exercise did not induce fatigue. However, as a consequence of the prior exercise muscle temperature of *m. vastus lateralis* (Tm) measured at a depth of 3 cm increased by 3.5 ± 0.8 °C, thus expected power at this pedal rate should be in the value of 15–20% higher (Sargeant, 1987).

In parallel experiments we allowed subjects a freely chosen warm up before the control measurements. This resulted in an increase of 2.5 ± 0.6 °C in Tm and a significantly higher IPP (1767 ± 207, $P < 0.01$). If this value is used as the appropriate control then there is a significant fatigue of at least 17% immediately after the 45 min exercise. IPP after the 45 min exercise is probably a combined effect of fatigue, which decreased power output, and an increased muscle temperature, which increased power output.

If a component of fatigue was present, IPP would be expected to increase when the subjects were allowed a 6 min recovery period before the subsequent 25 s sprint in the parallel experiment. Although Tm decreased by 0.9 ± 0.5 °C at 3 cm depth during the 6 min rest period, there was nevertheless a 9% recovery of IPP (1632 ± 292, $P < 0.05$) when compared with IPP immediately after the 45 min exercise, but this recovery was not complete. Still a significant ($P < 0.05$) 8% lower IPP value was found when compared to IPP measured after the free chosen warm up (at which time muscle temperatures were virtually identical).

In contrast to the rapid recovery from the fatigue induced by short duration prior exercise (Sargeant & Beelen, 1993), in these experiments where fatigue is induced by prolonged prior exercise the recovery rate of IPP also seems prolonged.

Approval for this study was obtained from the Ethical Committee of AMC, Amsterdam, The Netherlands.

**REFERENCES**


**Effect of branched-chain amino acid supplementation on performance during prolonged exercise**


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Wagenmakers and colleagues observed a deterioration of performance and a large increase in the exercise-induced hyperammonemia in patients with McArdle’s disease (a muscle glycogen breakdown defect) when oral supplements of branched-chain amino acids (BCAA) were given (Wagenmakers et al. 1990). It was hypothesized that an increase in the rate of BCAA oxidation in muscle puts a drain on the carbon flux in the citric acid cycle and, therefore, may lead to premature fatigue in patients with McArdle’s disease and in healthy individuals during prolonged exercise leading to glycogen depletion. The present study, therefore, was carried out to investigate whether BCAA supplementation affects performance and enhances the increase in plasma ammonia concentration during prolonged exercise in healthy endurance trained athletes. Ethical committee approval was obtained. Subjects (n = 10) were studied in three tests during cycle ergometer exercise until exhaustion at 70–75% of their maximal power output, while receiving one of three drinks. Drinks were given double blind and in random order 2 ml (kg body weight)$^{50}$ at the beginning of exercise and then each 15 min. All drinks contained 6% sucrose with or without (placebo)
Biochemical markers of muscle injury in man. The plasma ratio of myoglobin over fatty acid-binding protein distinguishes myocardial from skeletal muscle damage

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Myoglobin (88 kD) and fatty acid-binding protein (FABP, 16 kD) are low molecular mass, cytoplasmic proteins abundantly present in heart and skeletal muscle. Both proteins are released into the blood upon muscle cell damage (Kleine et al. 1992; Stone et al. 1975). In a group of patients with acute myocardial infarction (AMI) (nine males, one female; age 57 ± 10 years, mean ± s.d.) and a group of patients undergoing aortic surgery (eight males, one female; age 65 ± 7 years, mean ± s.d.) we studied whether the ratio of the plasma or serum concentrations of myoglobin and FABP can be used to discriminate between heart and skeletal muscle injury.

In intact human heart tissue, obtained from autopsy, the ratio of myoglobin over FABP was found to be 4.6 ± 0.8 (mean ± s.d., five cases), while among various skeletal muscles this ratio was found to vary from 21 to 73. In patients with AMI the plasma levels of each of the proteins was raised significantly within a few hours after the onset of first symptoms. Over 24 h after AMI the ratio of myoglobin over FABP in plasma was 4.4 ± 1.4 (mean ± s.d.; n = 33, nine patients), which is in agreement with the ratio found in heart tissue. In patients who underwent aortic surgery, the serum levels of each of the proteins was raised between 12 and 24 h after surgery, with the ratio of myoglobin over FABP amounting to 45 ± 22 (mean ± s.d.; n = 26, nine patients). In addition, in one AMI patient who was defibrillated at arrival in the coronary care unit 1 h after onset of symptoms, the ratio of myoglobin over FABP increased from 10 at about 3 h after onset of symptoms to about 50 at 24 h after onset of symptoms.

From these data we conclude that the ratio of the released amounts of myoglobin over FABP after muscle damage corresponds with the ratio of these proteins in the tissue of origin, and that assessment of this ratio is useful to discriminate between myocardial and skeletal muscle cell injury.

This study was approved by the ethical committee of the Academic Hospital, Maastricht, The Netherlands.

REFERENCES


Surface electromyography and fatigue during low intensity exercise

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Studies on localized muscle fatigue during exercise of low intensity are of importance in view of prevention of musculoskeletal disorders. Fatigue development, as measured by reduction in performance capacity, is often indicated by changes in the power spectrum of the electromyogram (EMG). This study will survey the reliability of the changes in power spectrum parameters mean power frequency (MPF) and centre frequency (CF) as indicators of fatigue caused by low intensity exercise of the triceps surae muscle.

After approval of the local ethics committee, ten males volunteered in this experiment in which continuous isometric plantar flexion at 5-8% (target level) of the