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The responses of the catecholamines and β-endorphin to brief maximal exercise in man

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Summary. The responses to brief maximal exercise of 10 male subjects have been studied. During 30 s of exercise on a non-motorised treadmill, the mean power output (mean ± SD) was 424.8 ± 41.9 W, peak power 653.3 ± 103.0 W and the distance covered was 167.3 ± 9.7 m. In response to the exercise blood lactate concentrations increased from 0.60 ± 0.26 to 13.46 ± 1.71 mmol·l^{-1} ($p < 0.001$) and blood glucose concentrations from 4.25 ± 0.45 to 5.59 ± 0.67 mmol·l^{-1} ($p < 0.001$). The severe nature of the exercise is indicated by the fall in blood pH from 7.38 ± 0.02 to 7.16 ± 0.07 ($p < 0.001$) and the estimated decrease in plasma volume of 11.5 ± 3.4% ($p < 0.001$). The plasma catecholamine concentrations increased from 2.2 ± 0.6 to 13.4 ± 6.4 nmol·l^{-1} ($p < 0.001$) and 0.2 ± 0.2 to 1.4 ± 0.6 nmol·l^{-1} ($p < 0.001$) for noradrenaline (NA) and adrenaline (AD) respectively. The plasma concentration of the opioid β-endorphin increased in response to the exercise from < 5.0 to 10.2 ± 3.9 p mol·l^{-1}. The post-exercise AD concentrations correlated with those for lactate as well as with changes in pH and the decrease in plasma volume. Post-exercise β-endorphin levels correlated with the peak speed attained during the sprint and the subjects peak power to weight ratio. These results suggest that the increases in plasma adrenaline are related to those factors that reflect the stress of the exercise and the contribution of anaerobic metabolism. In common with other situations that impose stress, β-endorphin concentrations are also increased in response to brief maximal exercise.

Key words: Exercise — Catecholamines — β-endorphin — Blood glucose — Blood lactate

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Introduction

Exercise has been shown to change the plasma concentrations of many hormones. However, the majority of studies have involved exercise of a long duration (see Galbo 1983) whilst the responses to brief maximal exercise have received little attention. Moreover, in those cases where hormonal responses to brief maximal exercise have been studied the responses are often different from those observed in prolonged exercise (Nävari et al. 1985).

The release and rate of release of hormones are controlled in response to specific needs. The rates of secretion should thus be related to their metabolic functions and the needs of a particular circumstance. The increase in sympathetic activity with exercise has been shown to influence muscle and liver glycogenolysis (Galbo 1983) to provide the glucose for further energy production. The natural opioid β-endorphin has also been shown to increase with exercise (Farrell 1985). The metabolic consequences of raised circulating β-endorphin levels are unclear although a wide range of possible affects have been proposed (Allen 1983). Whilst in prolonged exercise hormonal responses appear to be related to the relative intensity of the exercise, what influences the quantitative responses to brief maximal exercise is not known.

We have used a 30 s sprint on a non-motorised treadmill (Cheetham et al. 1986), to investigate the responses of the catecholamines and β-endorphin to brief maximal exercise.

Methods

Ten male subjects volunteered for these experiments which had University Ethical committee approval. The subjects,

whose age (mean ± SD) was 32.3 ± 10.6 years and weight 71.3 ± 6.9 kg, had previous experience of the experimental procedure and were a physically active group with a mean $\dot{V}_{O_2max}$ of 59.5 ± 7.1 ml·kg^{-1}·min^{-1}. The maximal exercise test involved a sprint of 30 s duration on a non-motorised treadmill. The performance of the test was monitored by recording the treadmill belt speed and the horizontal component of the force exerted by the subjects on the belt enabling force, speed, power and distance to be determined (Lakomy 1987). The heart rate of all subjects was monitored throughout the experiment.

The experimental procedure involved a standardised warm-up on the treadmill of two 30 s periods at 2.2 and 2.8 m·s^{-1}. This was followed by 5 min of rest in a recumbent position after which, a venous blood sample (20 ml) was taken from an antecubital vein. The sprint, which began from a rolling start at 2.2 m·s^{-1}, involved the subjects accelerating the treadmill to their maximum abilities and attempting to maintain this speed for 30 s. After the sprint, the subjects returned to the recumbent position where mixed arterialised samples (20 µl) were taken from the thumb at 1 and 5 min post-exercise and a venous sample taken 3 min post-exercise. Further venous samples were taken from 4 of the subjects 10 and 20 min post-exercise.

Venous samples were analysed for blood pH immediately following collection. Four aliquots were then taken, two of which, with the mixed arterialised samples, were analysed for glucose and lactate by enzymatic methods (Maughan 1982). The second two were used to determine blood haemoglobin and haematocrit values in order to calculate the percentage change in plasma volume (Dill and Costill 1974). The remaining blood was centrifuged at 3° C and the plasma stored at −25° C after the addition of 200 µl of an anti-oxidant solution (100 mmol·l^{-1} of reduced glutathione and EGTA adjusted to pH 6.5). Plasma catecholamines were determined by HPLC using electrochemical detection (Davies et al. 1981).

Plasma immunoreactive β-endorphin concentrations were determined using a two step assay. β-endorphin was first extracted from the plasma sample using Sepharose linked rabbit anti-β-endorphin antibodies. The bound fraction was eluted and assayed by a radioimmunoassay for β-endorphin. Cross reactivity with N-acetyl β-endorphin was 100%, with β-lipotropin <5% and with ACTH <0.01% (Immuno Nuclear Corporation, Stillwater, Minnesota, USA). The minimum detectable plasma β-endorphin concentration was 5 pmol·l^{-1}.

The statistical evaluation of the results was carried out using the Pearson product-moment correlation, a Student's *t*-test for paired data or where stated a Mann-Whitney test.

Results

The performance results for the 30 s sprint, as reflected by the treadmill belt speed, are shown in Fig. 1. This shows the initial acceleration from a rolling start, to reach a maximum speed after 4.7 ± 1.5 s and the subsequent decline in speed as fatigue occurs. The results of the other aspects of performance measured are shown in Table 1.

The maximum heart rate reached during the 30 s was 173 ± 13 beats per minute however, this was not as high as the 188 ± 9 beats per minute ($p < 0.001$) attained during the incremental grade test on a motorised treadmill used to determine $\dot{V}_{O_2max}$. Of the ten subjects, the maximum heart rate of eight was recorded between 10 and 20 s after the sprint rather than during the sprint itself. Blood pH fell from a pre-exercise value of 7.38 ± 0.02 to 7.16 ± 0.07 after the sprint, a mean fall of 0.23 whilst plasma volume fell by 11.5 ± 3.4%. The increases in blood lactate and glucose are shown in Table 2. Plasma AD and NA concentrations also increased in response to the sprint, there being a 7 fold increase in AD from 0.2 ± 0.2 to 1.4 ± 0.6 nmol·l^{-1} and a 6 fold increase in NA from 2.2 ± 0.6 to 13.4 ± 6.4 nmol·l^{-1}.

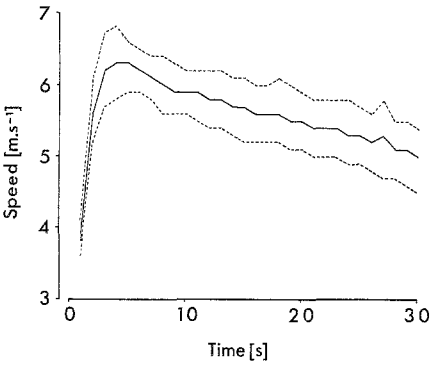

Fig. 1. The belt speed during a 30 s sprint on a non-motorised treadmill ($n = 10$, mean ± SD)

Table 1. Performance results of a 30 s sprint on a non-motorised treadmill ($n = 10$). Fatigue is expressed as the (peak speed − end speed)/peak speed * 100

	Mean	SD
Peak power (W)	653.3	103.0
Peak power/Wt (W·kg^{-1})	9.2	1.4
Mean power (W)	424.8	41.9
Peak speed (m·s^{-1})	6.4	0.3
Distance (m)	167.3	9.7
% fatigue	22.8	6.5

Table 2. Blood glucose and lactate concentrations in venous samples (pre and 3 min post) and mixed arterialised samples (1 and 5 min post) before and after a 30 s sprint on a non-motorised treadmill ($n = 10$, $*p < 0.001$ paired *t* test, post vs pre)

		Pre	Post		
			1 min	3 min	5 min
Glucose (mmol·l^{-1})	mean	4.25	5.22	5.59*	6.05
	SD	0.45	0.72	0.67	0.76
Lactate (mmol·l^{-1})	mean	0.60	11.10	13.46*	15.56
	SD	0.26	1.16	1.71	1.69

The pre-exercise concentrations of β-endorphin in plasma were below the limits of sensitivity of the assay (less than 5.0 pmol·l^{-1}, the mean resting value calculated from data reviewed in Farrell (1985) was 3.2±1.5 pmol·l^{-1}). However, the levels increased by at least 2 fold as the mean post-exercise concentration was 10.2±3.9 pmol·l^{-1}.

In the 4 subjects from whom further samples were taken, β-endorphin concentrations fell to 8.6±1.7 pmol·l^{-1} after 10 min and 5.5±1.0 pmol·l^{-1} after 20 min (see Fig. 2). A comparison of these results with those of the 10 subjects (Mann-Whitney test) indicated that there was no fall in β-endorphin concentrations after 10 min but that a significant fall had occured by 20 min post-exercise ($p<0.05$).

Correlations were found between post exercise AD levels and both post exercise blood lactate ($r=0.701, p<0.05$) and blood pH ($r=-0.708$, $p<0.05$). A correlation was also found between the percentage decrease in plasma volume and the post exercise AD ($r=0.690, p<0.05$). The post-exercise plasma concentrations of β-endorphin were related to the decrease in blood pH ($r=-0.620$, $p<0.05$) and to two aspects of the performance of the sprint. The correlation between β-endorphin and the peak power to weight ratio of the subjects being $r=0.808$ ($p<0.01$) and the peak speed the subjects attained being $r=0.708$ ($p<0.05$) (see Fig. 3).

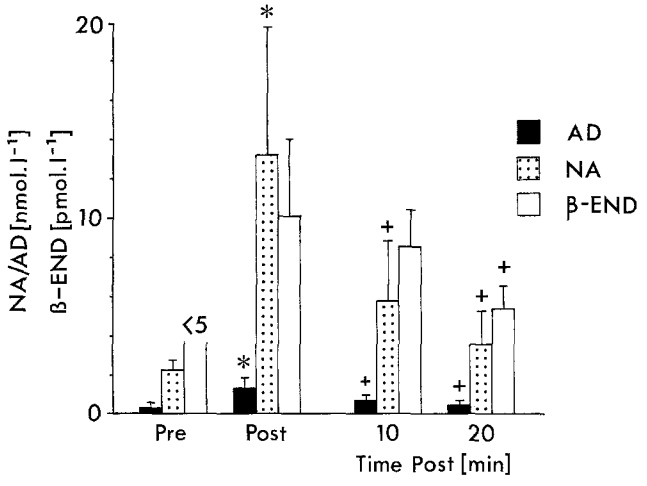

Fig. 2. Plasma AD, NA and β-endorphin concentrations (mean±SD) before and after a 30 s sprint on a non-motorised treadmill (*$p<0.001$ for paired t test pre vs post where $n=10$ and +$p<0.05$ for Mann-Whitney test post vs 10 and 20 min post where $n=10$ and 4)

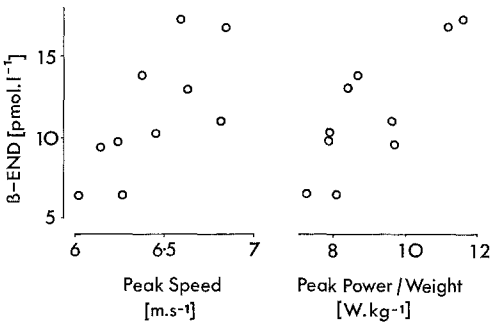

Fig. 3. The correlations between the post-exercise β-endorphin levels and the peak power to weight ratio ($r=0.808$, $p<0.05$) and the peak speed ($r=0.708$, $p<0.05$) for a 30 s sprint on a non-motorised treadmill ($n=10$)

Discussion

The intensity of the exercise during the 30 s sprint is evident from the 22 fold rise in the concentration of blood lactate. These high levels reflect the large contribution made by anaerobic metabolism and represent the utilisation of about 25% of the available skeletal muscle glycogen stores in just 30 s (Cheetham et al. 1986).

Circulating levels of AD and NA increased in response to the exercise test, the magnitude of the responses being similar to those after 30 s of maximal exercise on a cycle ergometer (MacDonald et al. 1983). During graded exercise the increases in the catecholamines appear to be related to the intensity as measured by $\dot{V}_{O_{2max}}$ and to the duration of the exercise (Lehmann et al. 1983). The factors influencing catecholamine concentrations in response to brief maximal exercise are less clear. Variations in sympathetic tone will affect levels of AD and NA released from the adrenal medulla. One of the factors that has been shown to influence the central control of sympathetic tone is a change in muscle osmolarity and/or potassium (Tibes et al. 1976). Interestingly, in the present study a correlation was found between post-exercise AD and the decrease in plasma volume. With sustained muscle contraction, vascular sympathetic nerve activity increases in proportion to the tension developed (Saito et al. 1986). If this also occures in dynamic exercise, then changes in the amount or degree of sympathetic innervation should be reflected by variations in the overflow of the transmitter NA into the circulation.

Differences in the response to maximal exercise have been demonstrated between individuals with different training backgrounds. Ohkuwa et al. (1984) have shown that a sprint trained group of runners had higher AD, NA and lactate con-

centrations after performing a 400 m sprint than an untrained group of subjects, even though the duration of the exercise was longer for the untrained group. They proposed that the differences in fibre types between the groups may contribute to the variations in lactate, the sprint group having a greater proportion of fast twitch (glycolytic) fibres. The findings in the present experiments of correlations between post-exercise concentrations of lactate and AD and between AD and blood pH would be consistent with this view. In addition, a correlation between circulating AD and degree of glycolysis has been demonstrated for female subjects undertaking this same type of high intensity exercise (Cheetham et al. 1986).

The post-exercise hyperglycaemia seen in the present study can not be explained by changes in plasma volume. The magnitude of the change in blood glucose being over twice that of the change in plasma volume (32% and 11.5% respectively). The experiments of Lavoie et al. (1987) have indicated that liver glycogenolysis is responsible for the post-exercise increase in glucose after brief supramaximal exercise. The post-exercise concentrations of AD and NA are such that direct stimulation should occur even though an increase in glucagon has also been demonstrated (Näveri et al. 1985).

Whilst catecholamine concentrations have been shown to be related to the exercise intensity (as % $\dot{V}_{O_{2max}}$, Lehmann et al. 1983), β-endorphin concentrations do not appear to be related to the intensity of exercise as measured by $\dot{V}_{O_{2max}}$ (reviewed in Farrell 1985). Inter-individual variations in responses have been demonstrated and De Meirleir et al. (1986) have proposed that both metabolic and hormonal influences affect β-endorphin release. These increases in circulating catecholamines and β-endorphin may not be dissociated. In rats, β_2 agonists produce a dose dependent increase in circulating β-endorphin (Berkenbosch et al. 1981). Influence in the opposite direction is seen in man where naloxone augments the exercise induced increase in circulating AD and NA (Grossman et al. 1984). The β-endorphin response to brief maximal exercise (mean post-exercise plasma levels 10.2 pmol·l^{-1}) was lower than most reported post-exercise value (see Farrell 1985). However, the duration of the exercise in all these cases was longer than the 30 s used in the present study. Thus, duration would appear to be one factor in determining the magnitude of the β-endorphin response. In the present study correlations were found between post-exercise β-endorphin concentrations and two measurements of performance, the peak power to weight ratio and peak speed (see Fig. 3). Thus, higher peak speeds or higher peak power to weight ratios correspond to higher post-exercise β-endorphin levels. This relationship is supported by the finding that a training regimen that improved performance also increased β-endorphin release (Carr et al. 1981).

In summary, the present study contributes to a description of the β-endorphin response to exercise and the demonstrates that brief maximal exercise will increase circulating concentrations. However, the role of β-endorphin in exercise is unclear and requires further investigation.

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