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DIFFERENCES IN THE METABOLIC AND HORMONAL RESPONSE TO EXERCISE BETWEEN RACING CYCLISTS AND UNTRAINED INDIVIDUALS

BY S. R. BLOOM, R. H. JOHNSON, D. M. PARK, M. J. RENNIE AND W. R. SULAIMAN

From the Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0HS, and University of Glasgow Department of Neurology, Institute of Neurological Sciences, Glasgow G51 4TF

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SUMMARY

1. Six well-trained cyclists and six untrained subjects were studied during and immediately after four successive 7 min periods of exercise at 30, 45, 60 and 75% of their maximal work capacity.

2. Venous blood samples were taken at rest, at the end of each exercise period and 5 min following the end of exercise, for estimation of metabolites in blood and plasma insulin, growth hormone, cortisol and catecholamines.

3. The results showed significant differences in the mobilization and utilization of muscle fuels between the athletically fit cyclists and the untrained group. In the cyclists, glucose, glycerol and free fatty acid concentrations were higher, but lactate, pyruvate and alanine were lower than in the untrained subjects during exercise.

4. Plasma catecholamines rose in both groups during exercise but the rise was significantly less in the racing cyclists. Plasma insulin was depressed to a greater extent in the untrained subjects during exercise and plasma glucagon rose to a greater extent during strenuous exercise and remained elevated after the end of exercise in the untrained group. Plasma human growth hormone rose to a greater extent during exercise and remained elevated after the end of exercise in the untrained group. Plasma cortisol fell at low and moderate exercise rates in both groups, but to a smaller extent in the cyclists. Cortisol values rose at higher workloads and were significantly higher in the cyclists at the end of exercise.

5. It is concluded that there are significant differences in the metabolic and hormonal responses to exercise between athletically trained and untrained individuals, even when the physically fit subjects work at the same percentage of their maximal capacity as the unfit subjects.

INTRODUCTION

Earlier studies of trained athletes and untrained individuals provided evidence of an adaptation of the metabolic and hormonal response to exercise as a result of physical training (Johnson, Walton, Krebs & Williamson, 1969; Rennie, Jennett & Johnson, 1974; Rennie & Johnson, 1974). Separate comparisons of racing cyclists and untrained subjects, and of individuals studied before and after physical training showed that during strenuous exercise in the trained state blood lactate and pyruvate were lower and blood glucose higher than in untrained subjects. Plasma free fatty acids and blood ketone-body concentrations were also lower after exercise, even when trained subjects worked harder (Rennie *et al.* 1974). In addition, plasma insulin and growth hormone concentrations were lower during and after exercise in trained individuals. Interpretation of these results was made difficult by conflicting information on possible changes in other metabolically active hormones such as cortisol, glucagon, noradrenaline and adrenaline. Although plasma catecholamine concentrations may be lower in trained than untrained subjects at a given submaximal workload it has been suggested that the plasma levels of catecholamines are similar during exercise at the same relative workload (Häggendal, 1971). The present study was therefore principally designed to examine plasma catecholamine concentrations in untrained subjects and racing cyclists working at similar relative levels. A sensitive and accurate method of analysis of catecholamines was employed to make this possible. These results have been related to changes in a number of metabolites and hormones, including cortisol and glucagon, in blood taken during the same investigations.

METHODS

Subjects. The investigation was explained to the subjects and their consent obtained. Six racing cyclists (males aged 22–27 years) agreed to take part in the study. They were tested at the end of a racing season during which they had trained by cycling about 200 miles per week and had frequently competed in local and national events. Six control subjects (males aged 25–33 years) who were not in regular athletic training were also studied. Details of the subjects are shown in Table 1.

Procedure. All subjects were studied on two occasions between 17.00 and 19.00 hr. The studies were carried out on two different days with at least 48 hr between them. The subjects were asked not to eat breakfast and to take only a light meal of sandwiches at mid-day, after which they had not eaten. All subjects carried out their normal daily activities before the tests. On the first occasion the work capacity of each subject was assessed by an increasing work-rate test (Spiro, Juniper, Bowman & Edwards, 1974). The subjects exercised on an electrically braked bicycle-ergometer (Elema-Schönander EM 369) at a work load of 16 W at 60 rev/min for 1 min before

the load was increased by 16 W/min until they were unable to continue. On the second occasion, during the main investigation, the subjects worked for four periods of 8 min each at successive workloads fixed at 30, 45, 60 and 75 % of the maximum work rate achieved in the first test. Heart rate was recorded on both occasions using an electrocardiograph (Elema-Schönander Mingograf). Blood samples were taken via an indwelling catheter previously placed in an arm vein. The catheter was flushed with physiological saline between taking samples. Samples were taken at rest, sitting on the ergometer, at the end of each 8 min period of exercise and 5 min after the end of the fourth period. Two samples of blood were taken on each occasion. The first (14 ml.) was divided into a 4 ml. aliquot which was deproteinized with 5 ml. 10 % (w/v) perchloric acid in a weighed tube and a 10 ml. aliquot delivered into a heparinized tube for later separation of plasma. The second sample (20 ml.), for catecholamine assay, was taken immediately after the first and delivered into an ice-cold universal glass bottle containing EDTA (50 mg), sodium metabisulphite (50 mg) and pargyline (0·5 mg). The tube was sealed with a polypropylene cap and inverted six times. All samples were kept in ice-water until the end of the investigation (37 min in all), when the deproteinized extract and blood plasma were separated by centrifugation at 3000 rev/min for 20 min. The plasma for catecholamine assay was centrifuged at 4° C. All separated samples were stored at $-20°$ C until they could be analysed.

The deproteinized extract was used for enzymatic estimation of lactate and pyruvate (Hohorst, Kreutz & Bücher, 1959), acetoacetate and 3-hydroxybutyrate (Williamson, Mellanby & Krebs, 1962), glycerol (Kreutz, 1962), alanine (Yoshida & Freese, 1965) and glucose (Werner, Rey & Wielinger, 1970). The separated heparinized plasma was stored at $-20°$ C for later estimation of cortisol (Usui, Kawamoto & Shimao, 1970), free fatty acids (Dalton & Kowalski, 1967), and immunoreactive insulin (IRI) and human growth hormone (HGH) using Wellcome MR71 standard insulin and MRC standard A growth hormone with charcoal separation methods (Morgan & Lazarow, 1963; Herbert, Lau, Gottlieb & Bleicher, 1965; Hunter & Ganguli, 1971). An aliquot of the plasma had 1000 kallikrein inhibiting units of aprotinin (Trasylol) added for each ml. and was stored for later radio-immunoassay of glucagon. MRC 69/104 glucagon standard made up in glucagon free plasma was used together with a pancreatic glucagon specific antiserum (Bloom, 1974). This assay gives lower basal glucagon values than those reported by other workers (e.g. Unger & Lefebvre, 1972) because interference from non-specific effects (Weir, Turner & Martin, 1973) had been minimized (F. P. Alford and S. R. Bloom, unpublished). The plasma from the second sample was analysed for adrenaline and noradrenaline by a two stage chromatographic purification followed by fluorimetric assay using the trihydroxyindole method (Renzini, Brunori & Valori, 1970). Tracer amounts of noradrenaline [methylene-^{14}C]bitartrate and of [7-^{3}H]adrenaline (Radiochemical Centre, Amersham, Bucks) were added to each sample for radiochemical estimation of recovery from the purification procedure. Mean recovery of noradrenaline and adrenaline were 84 ± 5 % and 82 ± 7 %, respectively. The error of the fluorimetric assay was estimated in twelve identical samples to be ± 6 %. All metabolites and catecholamine samples were analysed within 24 hr of sampling. Statistical analysis was performed using Student's t test and the Mann–Whitney non-parametic U test for small samples (Mann & Whitney, 1947).

RESULTS

Work capacity

The racing cyclists achieved a much greater maximum work output in the increasing work rate test than the untrained subjects. The difference was highly statistically significant ($P < 0{\cdot}001$).

Heart rate

In the increasing work rate rest the racing cyclists had signifficantly lower ($P < 0{\cdot}01$) heart rates at rest and significantly higher ($P < 0{\cdot}01$) heart rates at the maximum achieved workload than the untrained subjects.

TABLE 1. Details of six racing cyclists and six untrained subjects and their response to the exercise capacity test. (All values means ± S.E. of mean.)

	Age (year)	Height (cm)	Weight (kg)	Maximum work rate (W)	Heart rate at rest (beats/min)	Heart rate at maximum work rate (beats/min)
Racing cyclists	26 ± 0·8	175 ± 3·0	69 ± 4·0	342 ± 15	63 ± 7·0	195 ± 1·0
Untrained subjects	29 ± 1·0	184 ± 4·0	74 ± 4·0	236 ± 11	83 ± 4·0	186 ± 2·0
P	n.s.	$P < 0{\cdot}05$	n.s.	$P < 0{\cdot}001$	$P < 0{\cdot}01$	$P < 0{\cdot}01$

When the subjects exercised at the same percentage of their maximum achieved work rate, there were no significant differences in heart rate between the two groups during exercise. However, the cyclists showed a rapid fall in heart rate after exercise so that 1 min after the end of exercise it was much lower than in the untrained subjects ($P < 0{\cdot}001$).

Metabolic changes

Blood glucose. Resting glucose concentrations were similar in the two groups. Glucose concentrations rose during exercise in both groups, but the rise was much greater in the trained subjects and the values for the two groups were significantly different ($P < 0{\cdot}01$) after 16, 24 and 32 min of exercise (45%, 60% and 75% maximum). The values continued to rise after exercise and remained significantly different 5 min later ($P < 0{\cdot}001$).

Blood lactate, pyruvate and alanine. There were no significant differences in the resting values of these metabolites between the two groups. Mild exercise (30%) caused a significant ($P < 0{\cdot}05$) rise in the metabolites in

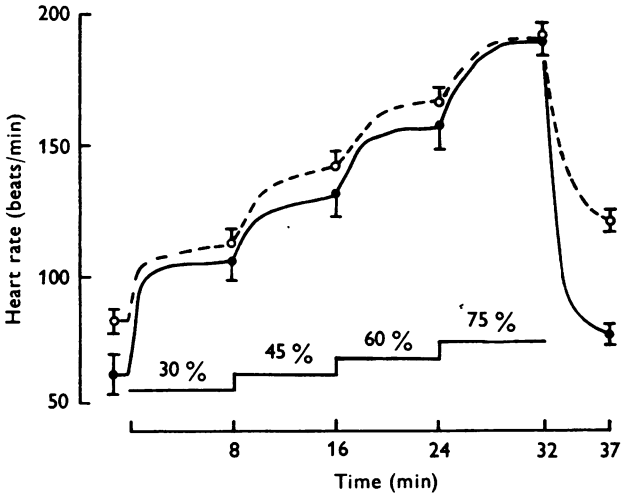

Fig. 1. Effect of graded exercises on heart rate (beats/min) in six racing cyclists (●——●) and six untrained subjects (○- - -○). All values means ± S.E. of mean. The step bar above the horizontal time axis shows the percentage work load of the maximum work rate in the first test.

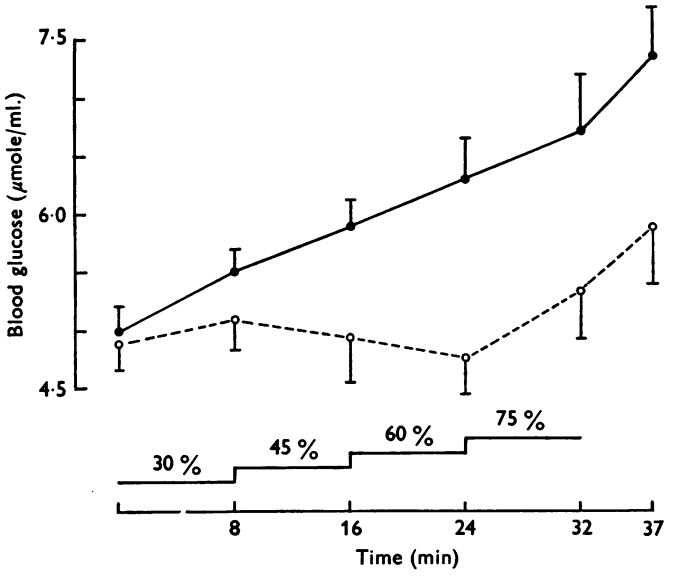

Fig. 2. Effect of graded exercise on blood glucose (μmole/ml.) in six racing cyclists (●——●) and six untrained subjects (○- - -○). All values means ± S.E. of mean. The horizontal axis and bar are described in the legend of Fig. 1.

the untrained subjects but not in the cyclists. The blood concentrations of lactate, pyruvate and alanine were significantly greater ($P < 0{\cdot}05$) in the untrained subjects after 8 min. Pyruvate and alanine concentrations in both groups of subjects and lactate concentrations in the untrained subjects continued to rise during exercise at greater loads. The racing cyclists showed little change in blood lactate until the end of the last period

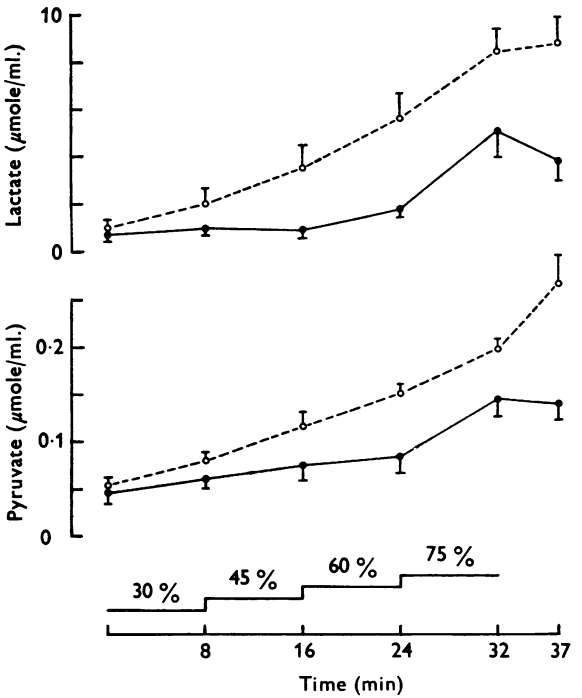

Fig. 3. Effects of graded exercise on blood lactate and pyruvate (μmole/ml.) in six racing cyclists (●——●) and six untrained subjects (○- - -○). All values means ± S.E. of mean. The horizontal axis and bar are described in the legend of Fig. 1.

(75% maximum) of exercise. The differences between the groups were significant at 16, 24 and 32 min of exercise. The metabolite concentrations continued to rise immediately after exercise in the untrained subjects but fell in the racing cyclists, so that the differences in concentrations between the two groups of each metabolite 5 min after exercise had become more significant ($P < 0{\cdot}001$).

Blood glycerol. The glycerol concentrations at rest were similar in both groups. Mild exercise (30%) caused an immediate rise in the blood glycerol level in the cyclists and the value continued to rise at successive workloads. In the untrained subjects glycerol levels remained almost unchanged

during exercise at the 30 % and 45 % work rate but increased progressively at 60 % and 75 %. However, the increases were much greater in the racing cyclists and the glycerol levels were significantly different between the two groups at the end of 8 min ($P < 0{\cdot}05$), 16 min ($P < 0{\cdot}01$), 24 min ($P < 0{\cdot}001$) and 32 min ($P < 0{\cdot}001$) of exercise. In both groups glycerol values continued to rise immediately after exercise and remained significantly different 5 min after the end of exercise ($P < 0{\cdot}001$).

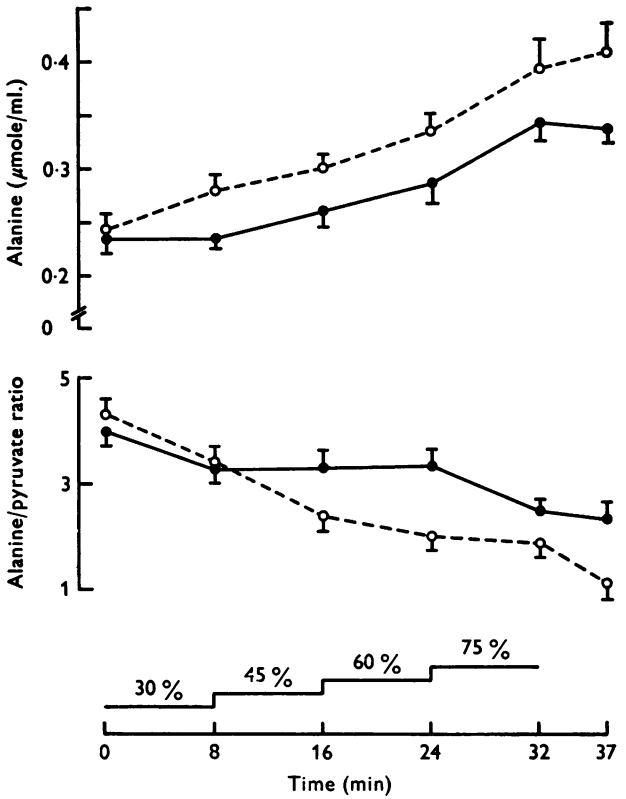

Fig. 4. Effects of graded exercise on blood alanine (μmole/ml.) and alanine–pyruvate ratio in six racing cyclists (●——●) and six untrained subjects (○- - -○). All values means ± S.E. of mean. The horizontal axis and bar are described in the legend of Fig. 1.

Plasma free fatty acids. No significant difference in the concentration of plasma free fatty acids was observed between the groups of subjects at rest. In both groups the exercise programme caused an initial fall in plasma free fatty acids. The untrained subjects showed a further slight fall in plasma concentration in the second exercise period (45 %) and then a rise in the third and fourth periods (60 and 75 %) brought the free fatty

acids concentration just above the resting value. Plasma free fatty acids concentration in the racing cyclists rose rapidly above the levels in the untrained subjects during the second and third 8 min periods of the exercise, and remained steady during the last period. The values of plasma free fatty acids in the untrained subjects were significantly lower than those in the racing cyclists at 16, 24 (both $P < 0{\cdot}05$) and 32 min of exercise ($P < 0{\cdot}01$). Plasma free fatty acids levels 5 min after the end of exercise had risen markedly in both groups of subjects, but the difference in free fatty acids concentrations remained significant ($P < 0{\cdot}01$).

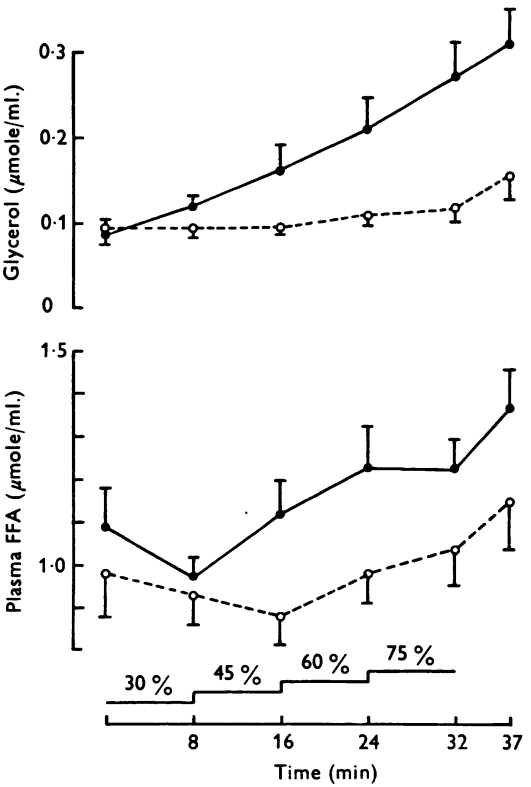

Fig. 5. Effects graded exercise on blood glycerol (μmole/ml.) and plasma free fatty acids, FFA (μmole/ml.) in six racing cyclists (●——●) and six untrained subjects (○- - -○). All values means ± S.E. of mean. The horizontal axis and bar are described in the legend of Fig. 1.

Blood ketone bodies. The difference in ketone bodies at rest was not significant. Blood ketone-bodies fell during exercise by similar amounts in both groups and rose slightly 5 min afterwards. The post-exercise rise was significantly greater in the untrained subjects than in the cyclists (untrained 59%, cyclists 35%; (means) $P < 0{\cdot}05$).

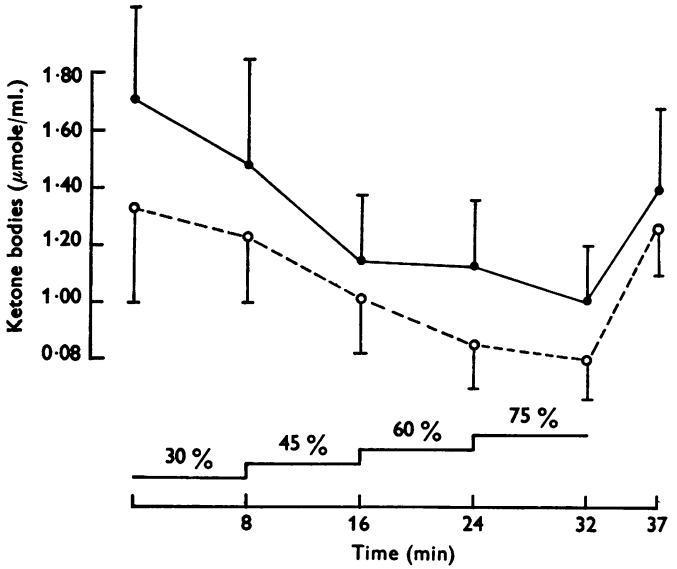

Fig. 6. Effect of graded exercise on blood ketone-bodies (μmole/ml.) in six racing cyclists (●——●) and six untrained subjects (○- - -○). All values means ± S.E. of mean. The horizontal axis and bar are described in the legend of Fig. 1.

Hormonal changes

Plasma cetecholamines. Resting concentrations of noradrenaline were lower ($P < 0{\cdot}05$) in the racing cyclists but there was no significant difference in adrenaline concentrations between the two groups. In both groups noradrenaline concentrations increased disproportionately as the subjects exercised against the increased loads. The rise was smaller in the cyclists, however, and they had significantly lower plasma noradrenaline concentrations at the end of each work period ($P < 0{\cdot}01$). The untrained subjects also showed a greater rise in adrenaline at the two highest work loads. The cyclists had only small increases in adrenaline concentration during exercise except at the highest work load when the value was significantly lower than in the untrained subjects. In both groups catecholamine concentrations fell rapidly after exercise to values at 5 min afterwards of about one third of the maximum values at the end of exercise. The adrenaline and noradrenaline concentrations remained significantly higher in the untrained group compared to the cyclists ($P < 0{\cdot}02$).

Plasma immunoreactive insulin. Resting values of plasma insulin concentrations were similar in the racing cyclists and in the untrained individuals. During exercise insulin concentration decreased progressively

with increasing work rate in the untrained subjects but there was no fall in the cyclists so that the differences during exercise were significant ($P < 0{\cdot}01$). Plasma insulin values rose very markedly 5 min after exercise in the untrained subjects but the rise was much less in the cyclists, the difference being highly significant ($P < 0{\cdot}001$).

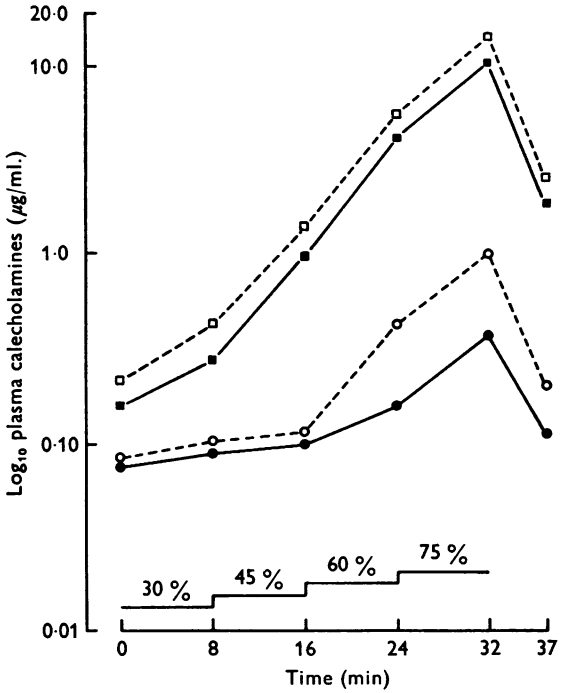

Fig. 7. Effects of graded exercise on plasma catecholamines (μg/ml.) in six racing cyclists (noradrenaline, ■——■; adrenaline, ●——●) and six untrained subjects (noradrenaline, □---□; adrenaline, ○---○). All values means. Note log. ordinate for plasma catecholamines. The horizontal axis and bar are described in the legend of Fig. 1.

Plasma glucagon. Resting glucagon concentrations were slightly higher in the untrained subjects and the difference was marginally significant ($P < 0{\cdot}1$). The response to exercise was similar in pattern in both groups with a slight fall in glucagon concentration at the end of the 60% and 75% work periods and also 5 min after exercise. Plasma glucagon was significantly higher in the untrained subjects throughout exercise and 5 min afterwards ($P < 0{\cdot}01$).

Plasma human growth hormone. Resting values of growth hormone were similar in both groups. However, during exercise the plasma human growth hormone concentration increased more rapidly in the untrained subjects

than in the cyclists. The values were significantly higher at 8 min ($P < 0{\cdot}05$) and throughout the remainder of the exercise period ($P < 0{\cdot}001$). The growth hormone concentration fell after exercise in the cyclists but continued to rise in the untrained subjects and the difference 5 min after the end of exercise was highly significant ($P < 0{\cdot}001$).

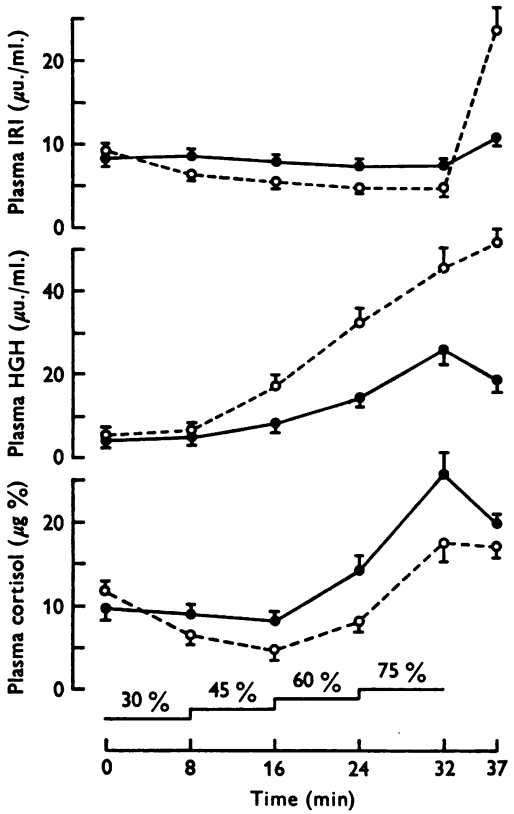

Fig. 8. Effects of graded exercise on plasma immunoreactive insulin, IRI, (μu./ml.); plasma human growth hormone, HGH (μu./ml.); and plasma cortisol (μg 100/ml.); in six racing cyclists (●——●) and six untrained subjects (○---○). All values are ± S.E. of mean. The horizontal axis and bar are described in the legend of Fig. 1.

Plasma cortisol. Cortisol concentration at rest was similar in both groups. During exercise cortisol levels fell at 8 and 16 min in the untrained subjects but then rose above resting in all subjects at 32 min. The rise was significantly greater ($P < 0{\cdot}01$) in the racing cyclists. In all subjects the plasma cortisol concentrations fell 5 min after exercise, the values for the untrained subjects falling less.

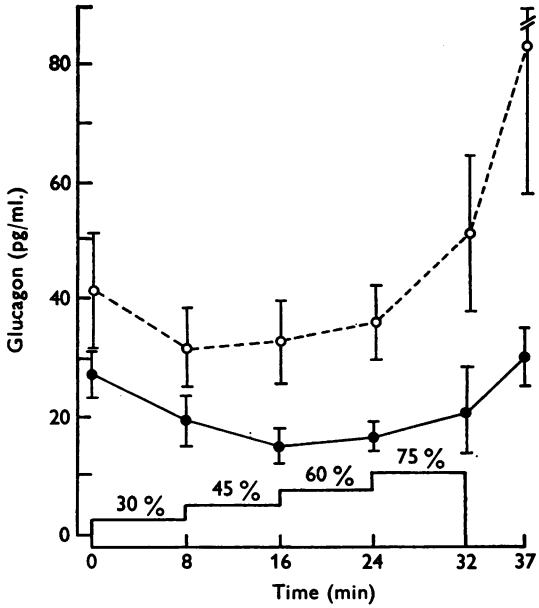

Fig. 9. Effect of graded exercise on plasma glucagon (pg/ml.) in six racing cyclists (●——●) and six untrained subjects (○---○). All values are ± S.E. of mean. The horizontal axis and bar are described in the legend of Fig. 1.

DISCUSSION

Both groups of subjects achieved similar heart rates when they worked at each level of exercise during the second investigation, suggesting that there were no differences in the relative work loads chosen for the two groups (Åstrand, Cuddy, Saltin & Stenberg, 1964; Spiro *et al.* 1974). The heart rates of all subjects at the end of the last period of exercise (194 ± 6 beats/min) suggest that the work loads were close to the aerobic maxima for the subjects (Åstrand *et al.* 1964), the cyclists having a greater exercise capacity.

Resting values of blood glucose, lactate and pyruvate were similar in the two groups and within the ranges previously described (Rennie *et al.* 1974). However, in both groups the free fatty acids and blood ketone-body concentrations were higher at rest than observed in previous investigations (Rennie & Johnson, 1974), perhaps because the present studies were carried out from 17.00 hr instead of after an overnight fast. The rise in blood glucose during exercise at work rates greater than 20 % of $\dot{V}_{O_2}$ has been noted by other workers (Wahren, Ahlborg, Felig & Jorfeldt, 1971). The present results show that trained athletes have a more marked increase

in blood glucose than untrained persons during exercise at similar relative workloads. The greater rise of glucose in the trained cyclists cannot be explained on the basis of increased catecholamine or glucagon levels, since the plasma concentration of both noradrenaline and adrenaline and also of glucagon was less in the racing cyclists than in the untrained controls. Adipose tissue from trained animals shows greater sensitivity to the lipolytic stimulation of catecholamines (Parizkova & Stankova, 1964; Gollnick & Williams, 1969) and the lipogenic actions of insulin. Athletic training may increase the sensitivity of other metabolic processes, including glycogenolysis, to hormonal activity.

The lower concentrations of lactate and pyruvate in the blood of the racing cyclists compared to that of untrained subjects working at the same relative intensity confirms previous findings (Saltin & Karlsson, 1971; Rennie *et al.* 1974). Saltin & Karlsson (1971) showed that the lower lactate and pyruvate did not depend upon a lower rate of glycogen consumption in trained subjects since rates of muscle glycogenolysis are similar in trained and untrained subjects working at the same relative work load. Evidence exists, however, that muscle alanine aminotransferase, which utilizes pyruvate as substrate and generates alanine, shows increased activity as an adaptation to exercise training (Molé, Baldwin, Terjung & Holloszy, 1973). The present results indicate that blood levels of alanine were lower in trained subjects than in untrained subjects exercising at the same relative intensity. The alanine–pyruvate ratio was also lower in the trained subjects than in the untrained subjects, suggesting that lower blood lactate and pyruvate observed during exercise in the trained state are unlikely to be solely due to increased rates of alanine formation in muscle. Liver blood-flow decreases during exercise, the fall being less in athletically trained subjects (Rowell, 1969) and this may promote relatively greater gluconeogenesis from substrates in the blood (Krebs & Yoshida, 1963; Exton, 1972).

There were also marked differences between the well-trained and the untrained subjects in blood-borne fat metabolites. The higher concentrations of glycerol (Steinberg, 1963) and plasma free fatty acids during exercise in the well-trained subjects support the suggestion that a greater degree of lipolysis occurred in the athletes. In previous studies of runners, racing cyclists and untrained persons we found lower levels of free fatty acids in the well-trained subjects during and after exercise and this was attributed to increased utilization (Johnson *et al.* 1969; Rennie & Johnson, 1974; Rennie *et al.* 1974). The increase of plasma free fatty acids appears to depend on exercise intensity, and it may be that despite care taken to equalize the relative intensity of work done by monitoring the heart rate and oxygen consumption of the subjects during a previous investigation

(Rennie *et al.* 1974) the results represented changes appropriate to a relatively lower level of exercise for the racing cyclists.

The present study utilizing direct measurement of plasma catecholamine in man showed that at rest there were significantly lower plasma concentrations of noradrenaline in the well-trained subjects, but no significant difference in adrenaline. Both noradrenaline and adrenaline increased during exercise as has been reported by other workers (Euler, 1969; Häggendal, Hartley & Saltin, 1970; Hartley, Mason, Hogen, Jones, Kotchen, Mougey, Wherry, Pennington & Ricketts, 1972). Hartley and co-workers (1972) demonstrated lower levels of noradrenaline at rest and during severe exercise after a 7-week training programme. However, they were unable to demonstrate differences in adrenaline at any workload. In the present study a rise in adrenaline was only detectable at high work levels, when there were lower concentrations of plasma adrenaline and noradrenaline in well-trained individuals working at the same relative work levels as untrained persons. As catecholamines were lower in the athletes in whom fat mobilization was greater, it is possible that there is a greater sensitivity of the tissues of trained athletes to circulating catecholamines.

Insulin is higher at rest in untrained individuals compared to well-trained subjects (Björntorp, DeJounge, Sjöstrom & Sullivan, 1970; Björntorp, Fahlén, Grimby, Gustafson, Holm, Renstrom & Scherstén, 1972; Rennie & Johnson, 1974; Rennie *et al.* 1974). It falls during exercise (Devlin, 1963; Pruett, 1970) and the present results show that the fall is greater in untrained subjects compared with the racing cyclists, so that the cyclists had higher insulin levels than the untrained subjects throughout exercise. The fall of insulin is apparently not related to changes in blood glucose, which rose in both groups. At rest adrenaline infusion depresses insulin secretion (Porte, 1967) and the exercise-induced depression of plasma insulin concentration is abolished by alpha-blocking agents such as phentolamine (Brisson, Malaisse-Lagae & Malaisse, 1971), suggesting that the effect during exercise is mediated by adrenergic mechanisms. This is supported by the present results which show a greater fall in plasma insulin in the untrained subjects who have a larger increase in plasma catecholamines. It has been suggested that the lipolytic effect of endogenous catecholamines during exercise is mediated through inhibition of insulin release (Lefebvre, Luyckx & Federspil, 1972). The present observations of a greater suppression of insulin, and lower degree of lipolysis indicated by lower blood glycerol and plasma free fatty acids concentrations in the untrained subjects make this difficult to accept.

Exercise causes a rise in plasma glucagon (Böttger, Schlein, Faloona, Knochel & Unger, 1972), and we have found that concentrations are

greater during and after exercise in untrained subjects. At rest, stimuli of glucagon release include a drop in plasma free fatty acids and glucose, and rises in alanine and lactate (Unger & Lefebvre, 1972). It is possible that the greater levels of glucagon in the untrained subjects during exercise are related to their greater concentrations of catecholamines since these are potent stimuli of glucagon release (Leclerq-Meyer, Brisson & Malaisse, 1971; Samols, Tyler & Kajinuma, 1971). Glucagon, the hormone of energy need (Luyckx & Lefebvre, 1973), promotes mobilization of fatty acids and glucose as metabolic fuels. The lower levels of glucagon in the athletes may be related to a greater sensitivity to the hormone's metabolic effects in these subjects, as suggested for other hormones.

The results also confirm previous findings of a smaller rise in plasma growth hormone in well-trained subjects compared with unfit individuals. As there was no fall in blood glucose in either group during the present investigation hypoglycaemia cannot be the cause of growth hormone release as suggested by Hunter and co-workers (Hunter, Fonseka & Passmore, 1965). Infusion of lactate may stimulate growth hormone secretion (Sutton, Young, Lazarus, Hickie & Maksvytis, 1969), and the greater blood lactate concentration in unfit subjects might explain their greater increase of human growth hormone during exercise. The smaller rise in human growth hormone in the racing cyclists may also be related to their lower catecholamine levels (Blackard & Hubbell, 1970).

Changes in plasma cortisol have previously been demonstrated during and after exercise. Moderate exercise for 10 min has been shown to decrease cortisol levels in man (Cornil, de Coster, Copinschi & Franckson, 1965). Sutton and co-workers (1969) and Few (1974) showed an initial fall of plasma cortisol followed by a rise as exercise continued, and the present results show a similar pattern. The initial fall might be the result of uptake of cortisol by muscle (Cornil *et al.* 1965; Wahlqvist, Kaijser, Lassers, Löw & Carlson, 1972; Few, 1974). Sutton and co-workers (1969) were unable to demonstrate any significant difference in cortisol levels between fit and unfit subjects but our results, showing significantly higher values of plasma cortisol during strenuous exercise in well-trained individuals than in untrained persons working at the same relative load, agree with the findings of Hartley *et al.* (1972). The differences in cortisol concentrations observed during the workloads of high intensity were probably due to greater secretion of cortisol by the racing cyclists (Frenkl & Csalay, 1962).

The present results confirm that metabolic and hormonal changes during exercise are different in athletically trained and untrained individuals and the differences persist even when the subjects work at the same relative level (Johnson *et al.* 1969; Rennie & Johnson, 1974; Rennie *et al.*

1974). In addition, these observations indicate that at the same relative levels of exercise-trained athletes have lower levels of circulating catecholamines and glucagon and higher levels of insulin and cortisol than untrained subjects.

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