

Effects of a 36-hour fast on human endurance and substrate utilization

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ZINKER, BRADLEY A., KAREN BRITZ, AND GEORGE A. BROOKS. *Effects of a 36-hour fast on human endurance and substrate utilization*. *J. Appl. Physiol.* 69(5): 1849–1855, 1990.— To determine if prolonged fasting affects substrate utilization and endurance time, seven trained men exercised to exhaustion on a cycle ergometer at 50% maximum oxygen consumption ($\dot{V}O_{2\max}$) in an overnight-fasted [postabsorptive (PA)] state and after a 36-h fast (F). Fasting produced significant elevations in the resting concentrations of blood free fatty acids (FFA; 1.16 ± 0.05 vs. 0.56 ± 0.06 mM, F vs. PA, respectively, a 107% increase), β -hydroxybutyrate (β -OH, 2.06 ± 0.66 vs. 0.15 ± 0.06 mM, a 1,270% increase), and glycerol (0.12 ± 0.03 vs. 0.04 ± 0.01 mM, a 200% increase), with a significant decline in glucose (79.79 ± 2.12 vs. 98.88 ± 3.11 mg/dl, a 19% decrease). Exercise in the F trial increased FFA, decreased glucose, and significantly elevated β -OH and glycerol over the PA trial. There was no difference in blood glucose concentration between trials at exhaustion. However, F produced a significant decrement in exercise endurance time compared with the PA trial (88.9 ± 18.3 vs. 144.4 ± 22.6 min, F vs. PA, a 38% decrease). Based on the respiratory exchange ratio, fasting led to a greater utilization of lipids during rest and exercise. It was concluded that 1) a 36-h fast significantly altered substrate utilization at rest and throughout exercise to exhaustion, 2) glucose levels do not appear to be the single determinant of time to exhaustion in submaximal exercise, and 3) despite the apparent sparing of carbohydrate utilization with the 36-h fast, endurance performance was significantly decreased.

exertion; fatigue; fasting; glucose; free fatty acids; β -hydroxybutyrate; lactate

FASTING, because it changes patterns of substrate utilization toward a greater dependence on lipid, has been considered effective in improving submaximal exercise endurance capacity. In 24-h fasted rats, Dohm et al. (6) observed that blood free fatty acid, glycerol and β -hydroxybutyrate levels were increased, whereas the rate of glycogen degradation was decreased in exercising muscle. Exercise endurance performance was increased by 79% as a result of fasting. The superior endurance of fasted rats was explained on the basis of enhanced “glucose-fatty acid cycle” activity (11, 17, 18, 25, 26).

Contrary to results of studies on animals, results on human subjects have demonstrated that fasting decreases exercise endurance. Loy et al. (16) determined that fasting decreased endurance at power outputs eliciting 79 and 86% maximum oxygen consumption ($\dot{V}O_{2\max}$) on a cycle ergometer. Dohm et al. (5) studied humans during treadmill running at a power output requiring 70% $\dot{V}O_{2\max}$ after a 24-h fast. Fasting raised

blood free fatty acids (FFA) and glycerol, but exercise endurance was not assessed. Additionally, Nieman et al. (20) reported similar results with treadmill running at 70% $\dot{V}O_{2\max}$ in human subjects after fasting. Muscle glycogen utilization was apparently not affected. However, endurance capacity was reduced by 45%. In a recent investigation it was determined that exercise to exhaustion at 45% $\dot{V}O_{2\max}$ was unaffected by a 3.5-day fast in comparison with a 14-h fast in untrained male subjects (15).

Because a primary effect of fasting is to shift metabolism to a greater utilization of fats, we reasoned that if endurance performance were to be increased by fasting, the exercise power output should be lowered to a level at which fats could have a more predominant role in the overall energy supply (4, 23). Moreover, if endurance training causes a shift to enhanced fat metabolism during exercise, then we further reasoned that fasting could preferentially benefit endurance in trained subjects. Therefore the purpose of the present investigation was to study the effects of a 36-h fast in moderately trained human subjects exercising at 50% $\dot{V}O_{2\max}$ to exhaustion. Under these conditions, a decrease in submaximal exercise performance was demonstrated after fasting.

METHODS

Subjects. Seven healthy male subjects volunteered to participate in this experiment. All were moderately trained cyclists. The subjects were informed of the nature of the study and were free to discontinue it at any time. An informed written consent was obtained from each participant (University of California, Committee for Protection Of Human Subjects, no. 85-6-25). The characteristics of the subjects are presented in Table 1.

To assess $\dot{V}O_{2\max}$, subjects completed a continuous progressive test on a Quinton (model QI-844) electrically braked cycle ergometer. The $\dot{V}O_{2\max}$ test was performed on the cycle ergometer starting at $300 \text{ kg} \cdot \text{m}^{-1} \cdot \text{min}^{-1}$ for 2 min; each subsequent 2 min the work load was stepped up $200 \text{ kg} \cdot \text{m}^{-1} \cdot \text{min}^{-1}$ until the subject was unable to maintain the work load.

Experimental design. Subjects participated in two trials separated by 2 wk. These trials were assigned at random and were started at ~7:00 A.M. A 24-h dietary recall was obtained from each subject. Additionally, subjects were instructed not to exercise 2 days before the testing and, in the case of the postabsorptive trial, to maintain their normal diet. The two trials to exhaustion

TABLE 1. Description of subjects

| Subj No. | Age, yr | Weight, kg | $\dot{V}O_{2\max}$, l/min | $\dot{V}O_{2\max}$, ml·kg ⁻¹ ·min ⁻¹ | Power Output, kg·m ⁻¹ ·min ⁻¹ | Diet Composition, % | | |
|------------|----------|------------|----------------------------|---|---|---------------------|----------|----------|
| | | | | | | CHO | Fat | Protein |
| 1 | 23 | 69.5 | 3.80 | 54.71 | 900 | 40 | 46 | 15 |
| 2 | 22 | 63.9 | 3.95 | 61.78 | 1,000 | 33 | 48 | 19 |
| 3 | 27 | 71.0 | 4.04 | 56.90 | 1,100 | 50 | 30 | 20 |
| 4 | 32 | 66.8 | 3.45 | 51.61 | 900 | 42 | 45 | 13 |
| 5 | 28 | 72.2 | 3.92 | 54.32 | 1,000 | 47 | 39 | 14 |
| 6 | 27 | 66.2 | 3.32 | 50.18 | 800 | 46 | 37 | 16 |
| 7 | 20 | 79.3 | 4.41 | 56.62 | 1,100 | 59 | 29 | 12 |
| Means ± SE | 25.6±1.7 | 69.8±2.1 | 3.8±0.2 | 55.2±1.6 | 971.4±45.4 | 45.3±3.4 | 39.1±3.1 | 15.6±1.2 |

Power output is work load eliciting 50% $\dot{V}O_{2\max}$. CHO, carbohydrate.

were 1) control ad libitum-fed (12-h overnight-fasted postabsorptive, PA) at a work load eliciting ~50% $\dot{V}O_{2\max}$ and 2) 36-h fasted (water-limited, F), also at 50% $\dot{V}O_{2\max}$. Exhaustion was determined voluntarily by the subject and was defined to be that moment when the work load could no longer be maintained with verbal encouragement. The subjects were provided tap water ad libitum throughout the trials, and a fan was utilized to aid in cooling. Pedaling rates were set by a metronome and checked on the ergometer tachometer for consistency among the subjects (range 65–85 rpm).

Respiratory gas exchange was monitored via a continuous-flow open-circuit system. Subjects breathed through a two-way Daniels valve from which expired air was continuously sampled through a heated line at a rate of ~0.48 l/min. The sample was first drawn through an infrared CO₂ analyzer (model CD 3A, Applied Electrochemistry) and then through a fuel-cell O₂ analyzer (model S-3A, Applied Electrochemistry). Both were on-line with an Apple II+ computer and were calibrated before and after each trial with reference gases previously analyzed by the micro-Scholander technique. Inspired gas volumes were measured with a Fleisch pneumotachometer and Validyne pressure transducer (model MP45-1-871) and carrier demodulator (model CD 15). The demodulator also provided an analog signal to the Apple II+ computer that was proportional to the inspired volume. Calculations of the rates of volume of expired air ($\dot{V}E$, l/min), volume of inspired air ($\dot{V}I$, l/min), volume of expired carbon dioxide ($\dot{V}CO_2$, l/min), volume of oxygen consumed ($\dot{V}O_2$, l/min, ml·kg⁻¹·min⁻¹), and the respiratory exchange ratio ($R = \dot{V}CO_2/\dot{V}O_2$) were made by the Haldane transformation. All calculations were completed on-line with the Apple II+ computer. Heart rates were monitored by electrocardiogram (model EK/5A, Burdick).

Blood sampling procedures. Blood samples (10 ml) were taken at rest, during, and after each test. A cannula (¼ in catheter, model 18.5 G × 1, Delmed) was inserted into a forearm vein. Three samples were obtained before exercise. The first of these was taken ~5 min after insertion of the catheter while the subject was lying supine, the second was taken 10 min after the first, and the last was obtained after the subject had been sitting on the cycle ergometer for 5 min and before exercise initiation. Sampling during the exercise bout was every 30 min and at the point of exhaustion. Subjects were

then asked to lie in the supine position, and a final sample was drawn 10 min after exercise.

A portion (2 ml) of each blood sample was deproteinized in 6% HClO₄, and the remainder was added to heparinized tubes for FFA determinations.

Analytic procedures. Perchloric acid extracts were neutralized by the addition of KOH. The neutralized extracts were assayed enzymatically for β-hydroxybutyrate (β-OH) (30), glycerol (29), glucose (Glucostat, Worthington Biochemical, Freehold, NJ), and lactate (12). FFA levels were determined on plasma by the single-extraction method (7).

Statistical methods. Data from rest to recovery were analyzed using paired and nonpaired *t* tests, one-way and two-way analyses of variance, and analyses of covariance (ANCOVA). Paired and nonpaired *t* tests were utilized because the subjects did not all stop after the same number of samples had been drawn. Scheffe's test was used to identify significant mean differences across trials when significant *F* ratios were obtained in the analysis of variance. Results are expressed as means ± SE. The level of significant difference chosen was *P* < 0.05 for differences between and within trials.

RESULTS

Physiological measures. Characteristics of subjects are presented in Table 1. The experimental work loads elicited 53.1 and 54.0% of $\dot{V}O_{2\max}$ (PA vs. F, *P* > 0.05, respectively). The average body weights before experimentation were not significantly different ($\dot{V}O_{2\max}$, PA, or F trial, Table 1). Moreover, there were no differences in weight loss between trials, F vs. PA. The heart rate response was significantly higher in the F trial, (150.3 ± 5.7 vs. 160.0 ± 5.6 beats/min, PA vs. F, respectively, on exhaustion, *P* < 0.05).

Respiratory measures. $\dot{V}O_2$ values during exercise were similar between trials (2.0 ± 0.1 l/min). Within 30 min and to 60 min of exercise, there was a significant decrease in *R* during the F trial compared with the PA trial (*P* < 0.05, Fig. 1). However, at exhaustion this difference disappeared. Within each trial the *R* significantly declined from initiation of the work bout to exhaustion, *P* < 0.05 (Fig. 1).

Time to exhaustion. Endurance decreased 38% after the fast in comparison with the PA trial (88.9 ± 18.5 vs. 144.4 ± 22.6 min, F vs. PA, respectively, *P* < 0.05). After

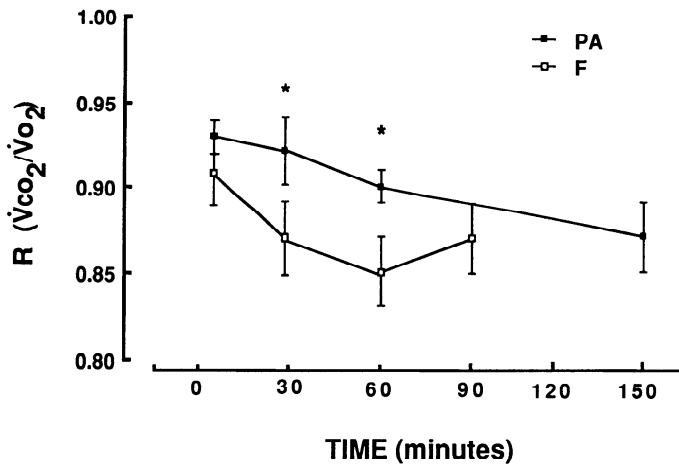


FIG. 1. Ventilatory respiratory exchange ratio (R) as a function of time at initiation of exercise, 30 min, 60 min, and exhaustion, for all subjects individually. Power output elicited 50% of maximum oxygen consumption ($\dot{V}O_{2max}$), under postabsorptive (PA) and 36-h-fasted (F) conditions. * Significant difference between means (PA vs. F, $P < 0.05$).

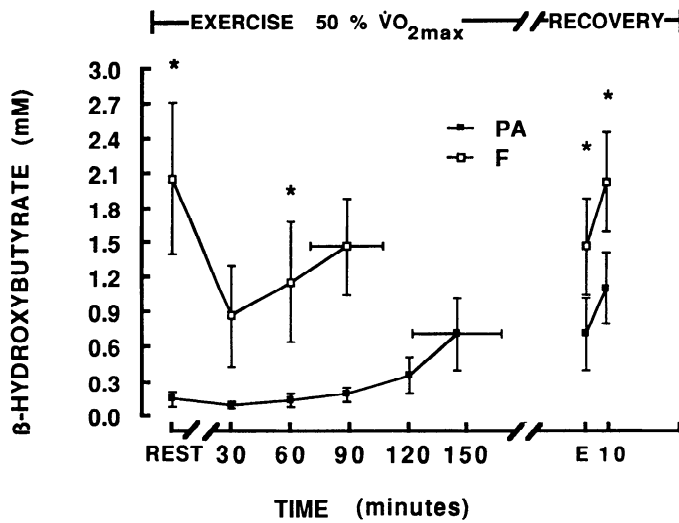


FIG. 2. Blood β -hydroxybutyrate (β -OH) concentration at rest, during exercise to exhaustion at 50% of $\dot{V}O_{2max}$, and 10 min postrecovery under PA and F conditions. E, exhaustion time point. * Significant difference between means (PA vs. F, $P < 0.05$). SE vertical bars are included for each sampling point, and horizontal bars are shown for time to exhaustion.

exercise in the F condition, subjects reported a common feeling of dizziness and a decreasing ability to concentrate on the task at hand.

Blood metabolites. Because, as determined by the analyses of variance, blood metabolite levels in the three resting blood samples were not significantly different, the resting values were pooled and averaged for each trial.

Plasma β -OH. Resting β -OH values after the 36-h fast increased 12-fold over PA rest levels ($P < 0.05$, Fig. 2). In the transition between rest and exercise, β -OH levels declined initially in both trials. However, after this initial fall, β -OH values increased throughout both trials and were significantly greater in the F trial than in the PA trial ($P < 0.05$), except at the 30-min time point (Fig. 2). After 10 min of recovery, the β -OH values rose sharply and significantly within both trials ($P < 0.05$), although the differences between the trials remained (F vs. PA, P

< 0.05). ANCOVA was utilized to factor out the differences in the resting concentrations of β -OH between the F and PA trials. The ANCOVA revealed no significant differences in the exhaustion and recovery β -OH values between trials ($P > 0.05$).

Plasma glycerol. After 36 h of fasting, resting glycerol levels were elevated twofold over those of the PA trial ($P < 0.05$, Fig. 3). Throughout the F trial as well as on exhaustion, glycerol levels were significantly greater than in the PA trial ($P < 0.05$). However, glycerol concentrations increased in a linear manner with time during both trials. After 10 min of recovery, the glycerol levels decreased, but remained substantially and significantly above rest values, and the significant difference between trials remained during recovery. Again, when resting differences due to the fast were factored out, there were no differences at exhaustion or during recovery between trials (ANCOVA, $P > 0.05$).

Plasma free fatty acids. The 36-h fast significantly elevated plasma FFA concentrations 107% at rest (F vs. PA, $P < 0.05$, Fig. 4). During exercise and at exhaustion, the FFA levels were greater in the F trial than in the PA trial ($P < 0.05$). Moreover, during both trials FFA levels tended to increase from resting levels during exercise. After 10 min of recovery, both groups showed large increases in FFA levels; FFA rose 54% after exercise in the F condition, whereas they rose 70% after exercise in the PA condition ($P < 0.05$). ANCOVA indicated that after exercise the FFA levels rose more with the PA trial than with the F trial ($P < 0.05$). However, a paired t test indicated that the rate of rise of FFA from exercise onset to exhaustion was similar between treatments ($P > 0.05$).

Blood lactate and glucose. Resting blood lactate concentrations were similar at rest and during exercise but tended to be higher throughout exercise in the F trial (Fig. 5). At exhaustion, subjects in the F trial had significantly higher blood lactate levels than subjects in the PA trial ($P < 0.05$).

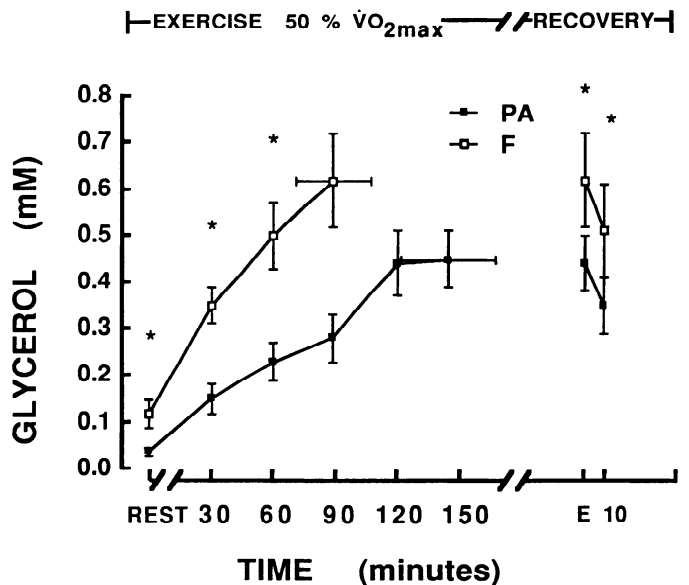


FIG. 3. Blood glycerol concentrations at rest, during exercise to exhaustion at 50% $\dot{V}O_{2max}$, and 10-min postrecovery under PA and F conditions. SE bars as in Fig. 2. * Significant difference between means (PA vs. F, $P < 0.05$).

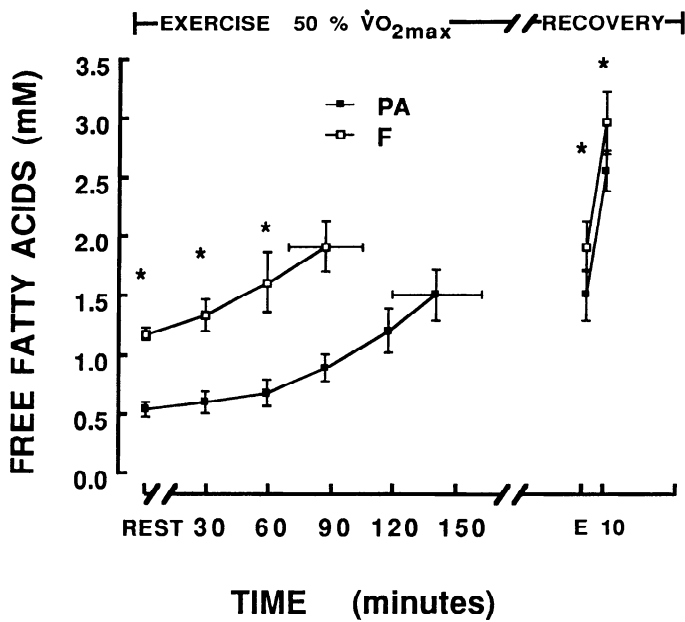


FIG. 4. Plasma free fatty acid (FFA) concentration at rest, during exercise to exhaustion at 50% $\dot{V}O_{2max}$, and 10-min postrecovery under PA and F conditions. E, exhaustion time point. SE bars as in Fig. 2. * Significant difference between means (PA vs. F, $P < 0.05$).

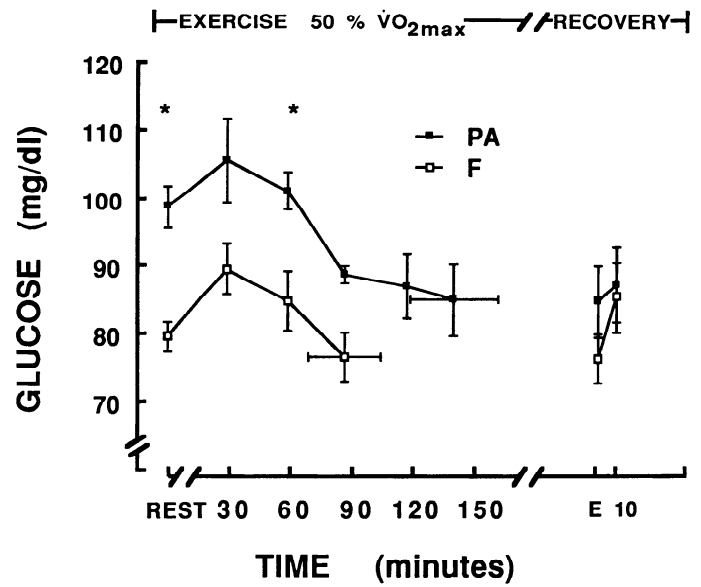


FIG. 6. Blood glucose concentration at rest, during exercise to exhaustion at 50% $\dot{V}O_{2max}$, and 10-min postrecovery under PA and F conditions. SE bars as in Fig. 2. * Significant difference between means (PA vs. F, $P < 0.05$).

DISCUSSION

On the basis of the present results, it is apparent that a 36-h fast decreases exercise endurance capacity in moderately trained male cyclists. It was determined that fasting increases FFA and ketone body utilization while sparing carbohydrates, but this response was insufficient in maintaining or enhancing exercise endurance. Fasting of this duration does not seem to be a viable means of increasing exercise endurance. In this regard our results are consistent with previous reports on humans (16, 20) but are contrary to results of work on rats (6).

Respiratory gas exchange. Oxygen consumption data make it clear that both fasted and control trials elicited essentially the same metabolic response, with similar $\dot{V}O_2$ values. The gas exchange R values were alike at the beginning and end of exercise but different during exercise (Fig. 1). Based on the respiratory gas exchange ratio, we estimate that during the PA trial, the initial percentage of fuel derived from fat was 27% and that from carbohydrate 73% (assuming a minimal protein contribution). During exercise in the PA trial, this fuel mixture gradually became greater in fat and less in carbohydrate. At 30 min of exercise, 28% of the fuel was obtained from fat and 72% from carbohydrate, whereas at 60 min the relative contributions were 34% from fat and 66% from carbohydrate in the PA trial. On exhaustion in the PA trial, the fuel mixture was 43% fat and 57% carbohydrate (Fig. 1). During the fasted trial the fuel mixture percentages obtained from combustion of fat and carbohydrate were ~32 and 68% at exercise initiation, 43 and 57% at 30 min, 51 and 48% at 60 min, and 43 and 57% upon exhaustion, respectively (Fig. 1). Therefore during the fasted trial, lipids were apparently being utilized to a greater extent than in the PA trial, and this was significant at 30 and 60 min.

Although the gas exchange R was depressed by fasting, the data still indicate a significant utilization of carbo-

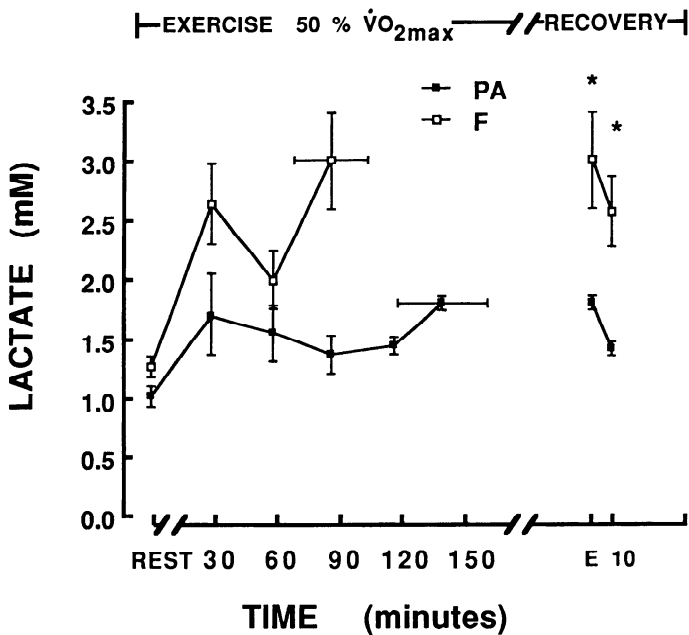


FIG. 5. Blood lactate concentration at rest, during exercise to exhaustion at 50% $\dot{V}O_{2max}$, and 10-min postrecovery under PA and F conditions. SE bars as in Fig. 2. * Significant difference between means (PA vs. F, $P < 0.05$).

Resting blood glucose concentrations in the F trial were significantly reduced from those in the PA trial (79.8 ± 2.1 vs. 98.9 ± 3.1 mg/dl, F vs. PA, respectively, $P < 0.05$, Fig. 6). Glucose levels were not significantly different at any other time point during or after exercise, except at the 60-min time point when the F trial was less than the PA trial ($P < 0.05$). Considering the differences between resting and end-exercise blood glucose concentrations, there were no differences between conditions in the decline of blood glucose concentration that accompanied fatigue.

hydrate during exercise. Previously (16, 20) it has been shown that 24–27 h of fasting had no major effects on skeletal muscle glycogen utilization rates during exercise to exhaustion.

The above values were computed assuming minimal contribution of amino acids to the substrate supply. Increased amino acid oxidation during fasting would have inflated the gas exchange R (compared with fat combustion), and thus this effect, if present, would have reduced the apparent differences in R between trials.

Although the results suggest that a greater amount of fat was combusted during the F trial, the difference from the PA trial was not great. There are several plausible mechanisms for this small difference. First, it could have been that the rate of intramuscular fat metabolism was too low to meet the energy turnover demand. Second, it may have been that the fatty-acyl oxidase pathway was substrate saturated, and thus additional increases in FFA concentrations were unable to increase the rate of FFA oxidation. Third, it is possible that there could have been an increase in catecholamine secretion as subjects became progressively more hypoglycemic during exercise. This would have stimulated muscle and liver glycogenolysis as well as liver gluconeogenesis, thus increasing carbohydrate metabolism (21, 24). Fourth, it may be that fatigue of type I muscle fibers resulted in increased recruitment of the less oxidative type II fibers to maintain the exercise power output (28). Finally, there could have been increased amino acid metabolism, which would have increased the R (compared with fat) and given the appearance of carbohydrate oxidation.

Of these possibilities, effects of elevated catecholamines on glycogenolytic and gluconeogenic rates are supported by several sets of observations. As noted previously (Figs. 5 and 6), blood glucose was lower and blood lactate was higher in the F condition. An elevation in catecholamines, increasing the supply of gluconeogenic (Cori cycle) precursor and accelerating glycogenolysis within the active muscle, would be an appropriate response to lowered glucose levels during exercise in the F condition. Additionally, mobilization of glycogen from active as well as inactive muscles could have supplied lactate for use as a fuel source (3).

β -OH. The lowering of β -OH on the initiation of exercise in the F trial can be interpreted to indicate an increased uptake and utilization of β -OH to generate energy equivalents through oxidation in the skeletal muscle. This interpretation is further supported by the response during recovery in which the concentration of β -OH increased 38% during the first 10 min after exercise. These results suggest increased ketone turnover and oxidation during exercise, particularly in the F trial with the β -OH levels significantly greater than in the PA trial. Consistent with previous investigations, the 36-h fast elevated the β -OH concentration over 12 times (1, 10). However, the results of the ANCOVA suggest that fasting did not significantly affect the increment in β -OH concentration during exercise and that the effects of exercise were superimposed on the effects of 36-h of fasting. It has previously been determined by others that ketone body oxidation increases two- to fourfold during exercise in the 3-day fasted state (1). Ketone concentra-

tions in the range indicated by our β -OH values have been shown to inhibit glucose uptake by resting muscle (1, 10, 18, 25).

Glycerol. The increase in blood glycerol concentration throughout exercise was expected, based on previous results (5, 14, 16, 27). Unfortunately, the ability of humans to utilize this glycerol as a gluconeogenic precursor is probably not as great as that of the rat (8, 17, 22, 28). This could be one of the primary reasons why the fasted rats (6) were successful in increasing endurance time, whereas we did not observe this in our subjects.

After exercise in both trials there was a significant decrease in glycerol level from that at exhaustion. This drop in blood glycerol could have been due to increased clearance associated with an elevated blood flow to the liver upon cessation of exercise. A rapid increase in FFA levels during recovery supports this interpretation of continued lipolysis after exercise. During the F trial there was a significantly higher glycerol concentration above that observed in the PA trial at this recovery time point. We attribute this finding to increased triglyceride utilization during the F trial.

FFA. In the present investigation, plasma FFA content after a 36-h fast was significantly higher at rest and during exercise than after an overnight fast. Therefore it can be concluded that a 36-h fast is sufficiently long to increase FFA mobilization. Arterial FFA concentration is one of the factors that limit the ability to utilize lipid in muscle (4). Thus it can be surmised that there was an increased utilization of FFA during the F trial. The gas exchange R data support this contention.

The 10-min recovery values demonstrated a significant increase in FFA concentration above exhaustion for both trials. This increase indicated that the release of FFA by the adipose and muscle declined more slowly than did the extraction by muscle and other tissue. Because of the sudden cessation of exercise, the previously active musculature no longer cleared FFA at a high rate, and thus the FFA levels increased.

Lactate. In the present experiments, resting lactate levels did not differ significantly between trials. However, after 30 min of exercise in the F trial, blood lactate doubled. This was not observed in the PA trial.

Blood lactate levels were not significantly different between trials until the exhaustion time point, when the F trial was significantly greater. The increased lactate could have been due to recruitment of a greater mass of higher-threshold glycolytic fast-twitch fibers as the population of slow-twitch fibers became increasingly glycogen depleted and fatigued. Loy et al. (16) observed a significant increase in lactate concentration concomitant to a decrease in skeletal muscle glycogen after a 24-h fast and exercise to exhaustion. Additionally, a catecholamine response associated with the onset of hypoglycemia could have accelerated muscle glycogenolysis (16, 20). Moreover, increased norepinephrine levels in the F trial could have decreased lactate clearance by shunting blood away from the liver (16, 20).

Glucose. The nadir of glucose concentration was associated with exhaustion. However, there was no significant difference between the two trials in blood glucose values, except in the F trial the nadir was reached much

sooner. It is possible that there is a basal level of glucose concentration that, if approached, will lead to termination of exercise. At that time either the exercise must stop, or the lack of glucose supply to the brain will result in symptoms of hypoglycemia (9). Individuals subjected to either a 36-h or an overnight fast appeared to have one common low level of glucose concentration that could not be reduced further.

Although blood glucose levels seem to have been important in determining endurance, we note that in the PA trial the blood glucose value at exhaustion (85.1 ± 5.3 mg/dl) was higher than the resting level before the F trial (79.8 ± 2.1 mg/dl). Thus if this difference in blood glucose level were critical for exercise, the F trial should not even have been possible. Therefore factors other than low levels of blood glucose must have contributed to fatigue. Low skeletal muscle glycogen is a prime candidate for inducing exhaustion in both trials (1, 6, 13, 16). Indeed, previous investigators (16) determined that low blood glucose values in combination with decreased skeletal muscle glycogen availability seemed to be the primary causes of fatigue. Such an effect could quite possibly have been amplified in the present study, because we utilized a 36-h fast compared with the shorter 24-h fast of Loy et al. (16). In our experiments, it might have been that the glucose turnover and liver as well as skeletal muscle glycogen degradation rates during the fasted trial were decreased because of the already depleted intramuscular and liver glycogen stores. Additionally, elevated levels of FFA may have led to the inhibition of pyruvate catabolism to acetyl-CoA (25).

After 10 min of recovery in both trials, both glucose values began to increase. This increase in blood glucose was likely due to a continued production of glucose via liver gluconeogenesis and to a fall in glucose utilization with the end of exercise.

Conclusions. In conclusion, 36-h fasting decreases the capacity of humans to endure moderate intensity exercise. Fasting did apparently increase FFA and ketone utilization, but these were insufficient to compensate for decreased carbohydrate reserves. Prolonged fasting of 36 h is not a recommended aid to increase endurance in humans.

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