Enhanced leg exercise endurance with a high-carbohydrate diet and dihydroxyacetone and pyruvate

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STANKO, R. T., R. J. ROBERTSON, R. W. GALBREATH, J. J. REILLY, JR., K. D. GREENAWALT, AND F. L. GOSS. *Enhanced leg exercise endurance with a high-carbohydrate diet and dihydroxyacetone and pyruvate.* J. Appl. Physiol. 69(5): 1651–1656, 1990.—The effects of dietary supplementation of dihydroxyacetone and pyruvate (DHAP) on metabolic responses and endurance capacity during leg exercise were determined in eight untrained males (20–30 yr). During the 7 days before exercise, a high-carbohydrate diet was consumed (70% carbohydrate, 18% protein, 12% fat; 35 kcal/kg body weight). One hundred grams of either Polycose (placebo) or dihydroxyacetone and pyruvate (treatment, 3:1) were substituted for a portion of carbohydrate. Dietary conditions were randomized, and subjects consumed each diet separated by 7–14 days. After each diet, cycle ergometer exercise (70% of peak oxygen consumption) was performed to exhaustion. Biopsy of the vastus lateralis muscle was obtained before and after exercise. Blood samples were drawn through radial artery and femoral vein catheters at rest, after 30 min of exercise, and at exercise termination. Leg endurance was 66 ± 4 and 79 ± 2 min after placebo and DHAP, respectively ($P < 0.01$). Muscle glycogen at rest and exhaustion did not differ between diets. Whole leg arteriovenous glucose difference was greater ($P < 0.05$) for DHAP than for placebo at rest (0.36 ± 0.05 vs. 0.19 ± 0.07 mM) and after 30 min of exercise (1.06 ± 0.14 vs. 0.65 ± 0.10 mM) but did not differ at exhaustion. Plasma free fatty acids, glycerol, and β-hydroxybutyrate were similar during rest and exercise for both diets. Estimated total glucose oxidation during exercise was 165 ± 17 and 203 ± 15 g after placebo and DHAP, respectively ($P < 0.05$). It is concluded that feeding of DHAP for 7 days in conjunction with a high carbohydrate diet enhances leg exercise endurance capacity by increasing glucose extraction by muscle.

leg endurance; high-carbohydrate diet; glucose extraction

IN A PREVIOUS INVESTIGATION (17), it was shown that consumption of dihydroxyacetone and pyruvate (DHAP) as part of a standard diet improved submaximal arm endurance by 20%. The experimental paradigm called for the substitution of 75 g dihydroxyacetone and 25 g pyruvate for an isocaloric amount of carbohydrate (CHO) in the diet over 7 consecutive days. Both arteriovenous glucose difference and blood fractional glucose extraction were higher after dietary consumption of DHAP compared with glucose (placebo). Although it was not directly measured, the greater glucose extraction after DHAP supplementation was assumed to be indicative of enhanced muscle glucose uptake and prolonged oxidation of glucose. Therefore utilization of blood-borne glucose delayed the onset of fatigue, extending submaximal endurance capacity (2).

In addition, resting muscle glycogen concentration was higher during the DHAP compared with the placebo trial before exercise but did not differ between trials at exercise termination (17). The comparatively greater pool of energy that was available from enhanced intramuscular glycogen stores could have also contributed to the prolonged exercise performance in the DHAP trial (3).

Our previous investigation (17) evaluated subjects performing arm exercise after consuming DHAP in a diet containing 55% CHO. The question arises whether DHAP would improve endurance capacity in subjects consuming a high-CHO diet. Consumption of such diets for periods of 3–7 days increases intramuscular glycogen stores and improves submaximal endurance performance in both laboratory and competitive trials (2, 10, 15). Typically, this ergogenic procedure allocates ~70% of the total caloric intake to a mix of simple and complex CHO. As such, one purpose of this investigation was to compare endurance time during submaximal leg exercise in subjects consuming high-CHO diets that do and do not contain DHAP.

Ahlborg et al. (1) have shown that fractional glucose extraction by muscle is less in leg than in arm exercise. One possible mechanism by which DHAP enhances exercise endurance is to increase muscle fractional extraction of glucose (17). Therefore the question arises whether DHAP enhances endurance capacity during leg exercise, which involves muscle with lower capacity for fractional glucose extraction than arm muscle (1). As such, this investigation examined the ergogenic effect of DHAP on endurance performance using submaximal leg exercise.

METHODS

Subjects. Eight male physically active but untrained university students who were free of known disease were used as subjects. On average they were 23.6 (± 0.9 SE) yr old, weighed 78.2 ± 3.6 kg, and had a peak oxygen consumption ($\dot{V}O_2$) on a cycle ergometer of 3.7 ± 0.3 l/min. All experimental procedures were approved by the University of Pittsburgh's Institutional Review Board for Human Subjects Experimentation. Subjects were in-

formed of the possible side effects of the experiment and gave their written consent to participate.

Experimental design. A double-blind repeated measures experimental design was used, with each subject receiving a placebo and a treatment diet. The order of presentation of the placebo and treatment diets was counterbalanced. All the food of the representative diets was given to the subjects at the Clinical Research Center of the University of Pittsburgh but was consumed on an outpatient basis for 7 days before exercise testing. Both diets provided 35 kcal/kg body weight and were composed of 70% CHO, 18% protein, and 12% fat. The treatment diet (DHAP) contained 75 g dihydroxyacetone and 25 g sodium pyruvate (Chemical Dynamics, South Plainfield, NJ). On average, DHAP accounted for 15% of calories and did not exceed 400 kcal in a given 24-h period. This supplemental amount was selected because, in previous studies in rats (16) and humans (17), feeding DHAP as ~15% of daily calories has been shown to increase body glycogen stores. At the time of this study, sodium pyruvate was the only form of pyruvate available for human consumption. To avoid excess sodium intake, maximum daily consumption of sodium pyruvate was 25 g. Consequently, a 3:1 mixture of DHAP was used, and 75 g dihydroxyacetone and 25 g pyruvate were consumed daily. One hundred calories of DHAP were added to 8 oz water, sweetened with Nutrasweet, and ingested four times daily. The placebo drink contained an isocaloric amount of Polycose. Randomization of the diets was performed by the chief clinical dietitian of the Clinical Research Center. The subjects and all personnel associated with experimental testing were unaware of the contents of the diets. On their first visit to the Clinical Research Center, subjects were given either the placebo or the treatment diet. The first diet was consumed during three daily meals over a 1-wk period. After completion of the first diet and after a 10- to 12-h postabsorptive period, the subjects performed leg exercise to determine submaximal endurance capacity. Seven to fourteen days after the first experimental trial was completed the second diet was initiated. This diet was consumed during three meals over a 1-wk period. At the completion of the second diet, leg exercise to determine endurance capacity was again undertaken. The subjects avoided prolonged exhaustive exercise and alcohol intake during the study.

TABLE 1. *Peak $\dot{V}O_2$, treatment sequence, and endurance capacity during prolonged leg exercise*

Subject No.	Peak $\dot{V}O_2$, l/min	Diet Sequence	Endurance Time After Placebo Diet, min	Endurance Time After DHAP Diet, min
1	4.1	P:T	60	76
2	2.7	T:P	70	80
3	3.1	P:T	52	78
4	4.2	P:T	77	85
5	4.3	T:P	62	67
6	3.3	T:P	60	80
7	2.8	T:P	61	83
8	5.2	P:T	84	80
Mean ± SE	3.7 ± 0.3		66 ± 4	79 ± 2*

P, placebo diet; T, DHAP treatment diet. * $P < 0.01$ vs. placebo.

None of the subjects was receiving medications or anabolic steroids.

Exercise testing. Before the experimental trials, peak $\dot{V}O_2$ was determined during leg exercise on a Monarck cycle ergometer (model 864) equipped with toeclips. This test was used to establish the power output equivalent of 70% peak $\dot{V}O_2$ that was used during the experimental trials. The initial power output for the leg peak $\dot{V}O_2$ test protocol was 24.5 W, with the power output increased in 24.5-W increments every 3 min. The crank rate was 70 revolutions/min, with cadence established by an electric metronome. Total body $\dot{V}O_2$ and respiratory exchange ratio were determined every minute during exercise. The test was terminated when the subject was unable to continue exercise, as indicated by a failure to maintain a cranking rate of 70 revolutions/min for a consecutive 15 s. Peak values (Table 1) were determined when $\dot{V}O_2$ leveled off in the presence of increasing power output. In all tests of $\dot{V}O_2$, peak respiratory exchange ratio exceeded 1.1.

During the experimental trials the subjects were seated on the cycle ergometer. The ergometer was calibrated before each experiment. During all tests, ambient temperature and relative humidity were maintained at 20–22°C and 50%, respectively. All tests were started at 0900 h. Ad libitum consumption of water was allowed throughout the exercise trial. Subjects exercised at 70% of peak $\dot{V}O_2$ until the test was terminated because of exhaustion. The 70% relative metabolic rate for the exercise trials was chosen because during pilot experiments it was determined that this exercise intensity could be sustained for extended periods of time. Consistency of $\dot{V}O_2$ over the time course of exercise was determined every 10 min by respiratory gas analysis. Exhaustion was defined as the inability to maintain a crank rate of 70 revolutions/min for 15 consecutive s as determined by two observers. Subjects were regularly instructed to maintain the crank rate and continue exercise as long as possible. Forceful motivation was not used. Endurance times were not revealed to the subjects. All methods of time measurement, such as wristwatches and wall clocks, were removed during the exercise trials. Subjects exercised continuously except during a 90-s rest period every 30 min when blood samples were obtained and blood flow measurements were performed.

Cardiorespiratory measures. Heart rate was measured every 10 min during exercise by use of the R-R intervals on an electrocardiogram. Metabolic responses were determined by standard techniques of open-circuit spirometry. Inspired ventilation was measured with a Parkinson Cowan CD-4 gasometer. The concentrations of CO_2 and O_2 in expired air were measured with a Beckman LB-2 CO_2 analyzer and an Applied Electrochemistry S-3A O_2 analyzer. The analyzers and gasometer were integrated into a laboratory computer (Apple IIe) for calculation of $\dot{V}O_2$, carbon dioxide production ($\dot{V}CO_2$), and respiratory exchange ratio. The analyzers were regularly calibrated with previously standardized gases. The dry gasometer was calibrated against a Tissot spirometer.

Fuel oxidation. Glucose and lipid oxidation were estimated using the equations (7)

glucose oxidation (g/min)

$$= 4.55\ \dot{V}CO_2 - 3.21\ \dot{V}O_2 - 2.87\ N_2$$

$$\text{lipid oxidation (g/min)} = 1.67\ (\dot{V}O_2 - \dot{V}CO_2) - 1.92\ N_2$$

where N_2 is urinary nitrogen excretion. Subjects were assumed to be in ~1-g positive nitrogen balance because weight did not change during the study. Subjects consumed 20 g N_2 daily, and therefore nitrogen excretion was estimated to be ~0.01 g N_2/min. $\dot{V}O_2$ and $\dot{V}CO_2$ were determined for 1 min every 10 min. Fuel oxidation was calculated for 1 min, assumed to be constant for 10 min, and recorded for 10-min intervals. Because exercise duration varied but all of the subjects exercised for at least 50 min during both exercise trials, data are presented in 10-min intervals and for the total exercise period.

Blood chemistry measurements. Before exercise, a 16-gauge 6.35-cm FEP Radiopaque catheter was inserted retrograde through the groin into the common femoral vein such that the tip was approximately positioned at the inguinal ligament. Such positioning enabled sampling of venous blood drainage for the whole leg. A catheter was also placed in the radial artery of the ipsilateral arm. The venous catheter was stabilized with three 4-0 silk sutures and tape. The catheter, pelvis, and proximal 20 cm of left thigh were then further stabilized with Coban wrap (3M, Minneapolis, MN). The arterial catheter was secured with tape. Exercise was discontinued for 90 s every 30 min for blood sampling. Patency of the catheters was maintained with saline flush. Despite the excessive leg movement at 70 revolutions/min, only one venous catheter became temporarily dislodged and nonfunctional during the study. Arterial and venous blood was analyzed for pyruvate, lactate, glucose, free fatty acids, glycerol, and β-hydroxybutyrate. Glucose (9), lactate (8), pyruvate (8), glycerol (18), and β-hydroxybutyrate (19) were analyzed in duplicate in whole blood by enzymatic techniques. Plasma free fatty acids were determined by enzymatic techniques (NEFAC, Wako Pure Chemical, Osaka, Japan). Biochemical profile and complete blood count were determined by standard laboratory techniques.

Muscle glycogen. Before exercise and at exhaustion, a 10-mg biopsy of the vastus lateralis muscle was obtained with a Trucut biopsy needle. For each biopsy, a 5-mm incision was made in the skin after 0.5–1.0 ml xylocaine was injected subcutaneously to induce local anesthesia. The biopsy sample was immediately frozen in liquid nitrogen and stored at −10°C. The sample was later analyzed for glycogen concentration (13).

Blood flow. Blood flow in the whole leg was measured by bioelectrical impedance (14). Impedance was measured with a Minnesota Impedance Cardiograph (Surcom, Minneapolis, MN) with electrodes applied to the leg at thigh and ankle by means of 1.0-cm tape around the circumference of the leg. Impedance recordings were made before exercise, for 30 s before and 30 s during the intermittent rest periods, and at exhaustion of each exercise trial. Blood flow was calculated according to the equation

$$\Delta V = P \times (L/Z_0)^2 \times dZ/dt \times T$$

where, ΔV is blood volume of each pulse (in ml), P is resistivity of blood (in $\Omega \cdot$cm), L is distance between sensing electrodes (in cm), Z_0 is baseline impedance (in Ω), dZ/dt is maximal rate of change of impedance (in Ω/s), and T is ventricular ejection time.

Statistical analysis. Differences in exercise performance capacity and total fuel oxidation between experimental trials were determined with a paired t test (6). Changes in blood chemistry, muscle glycogen, and intervals of fuel oxidation were examined with a two-way analysis of variance (condition × time) with repeated measures on both factors. Significant main effects and interactions were evaluated with Scheffé's post hoc test (12). Responses were not affected by order of treatment. There were eight subjects in all evaluations.

RESULTS

Submaximal endurance capacity during leg exercise was 66 ± 4 and 79 ± 2 min during the placebo trial and the DHAP trial, respectively ($P < 0.01$; Table 1). Seven of eight subjects increased endurance capacity after consumption of DHAP.

Resting muscle glycogen concentration was 112 ± 23 and 108 ± 6 mmol/kg after consumption of the treatment diet and the placebo diet, respectively (P = NS; Fig. 1). Muscle glycogen concentration decreased over the time course of exercise and did not differ between experimental trials at the point of exhaustion.

Arterial and venous glucose concentrations are presented in Table 2. Arteriovenous glucose difference (glucose extraction) was greater ($P < 0.05$) before exercise and after 30 min of exercise during the treatment compared with the placebo trial. Differences were not noted between conditions at exhaustion. Fractional glucose extraction (arteriovenous glucose concentration/arterial glucose concentration; Fig. 2) was also greater ($P < 0.05$) before and after 30 min of exercise during the treatment

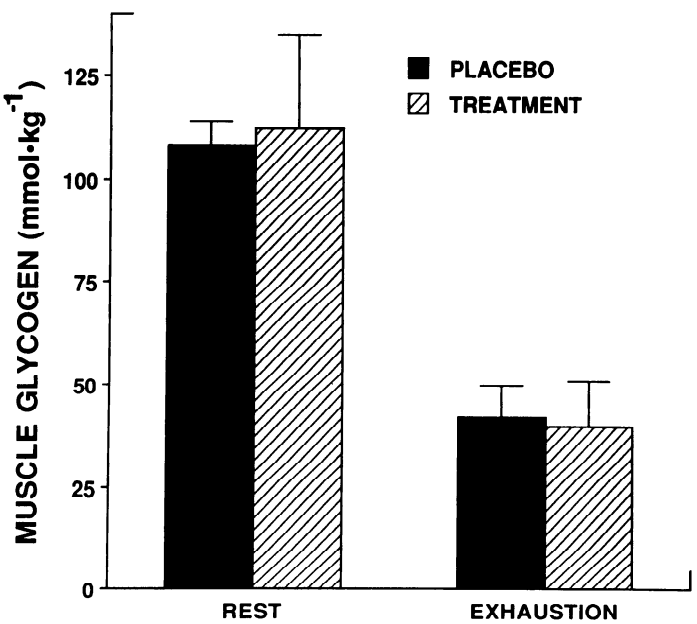

FIG. 1. Glycogen concentration of vastus lateralis muscle before and after exhaustive leg exercise in subjects consuming glucose polymer (placebo) or DHAP (treatment) for 1 wk. Values are means ± SE.

TABLE 2. *Blood glucose concentrations during rest and exercise*

	Glucose, mM		
	Rest	30 min	Exhaustion
Arterial			
Placebo	4.31±0.17	4.47±0.23	4.40±0.26
Treatment	4.56±0.10	4.60±0.23	4.16±0.41
Venous			
Placebo	4.12±0.16	3.82±0.21	3.53±0.26
Treatment	4.20±0.10	3.54±0.19	3.09±0.28
Arteriovenous			
Placebo	0.19±0.07	0.65±0.10	0.87±0.11
Treatment	0.36±0.05*	1.06±0.14*	1.07±0.16

Values are means ± SE. * $P < 0.05$ vs. placebo.

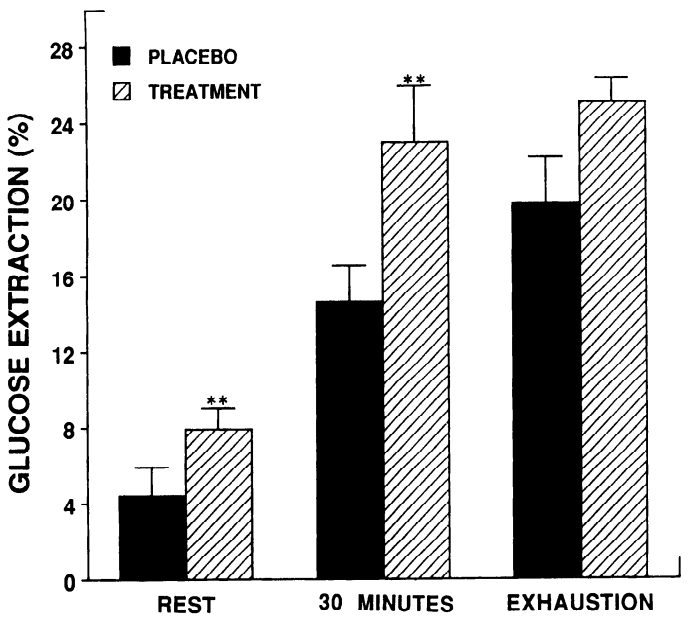

FIG. 2. Fractional extraction of glucose across 1 leg at rest, after 30 min of exercise, and at exhaustion in subjects performing leg exercise. Percent extraction of glucose was determined by dividing the arteriovenous concentration of glucose by arterial glucose concentration. Values are means ± SE. ** $P < 0.05$ between means for a given time period.

compared with the placebo trial.

Despite the consumption of 25 g pyruvate daily for 7 days, arterial and venous lactate and pyruvate concentrations and arteriovenous differences (Table 3) were similar after consumption of the placebo and treatment diets during both rest and exercise.

Venous concentrations of free fatty acids, β-hydroxybutyrate, and glycerol before and during the experimental trials are presented in Table 4. Although these venous fuel substrate concentrations increased with exercise time, differences were not found among experimental trials at rest, after 30 min of exercise, and at exhaustion for any of these variables. Arteriovenous differences for these fuel substrates were also unchanged (data not presented).

Respiratory exchange ratio and estimates of glucose and lipid oxidation are presented in Table 5. Respiratory exchange ratio did not differ between experimental trials. Total glucose oxidation increased by 20% with DHAP ($P < 0.05$). Total lipid oxidation was similar after both

TABLE 3. *Blood lactate and pyruvate concentrations during rest and exercise*

	Rest	30 min	Exhaustion
		Lactate, mM	
Arterial			
Placebo	1.235±0.140	2.741±0.383	2.807±0.334
Treatment	1.144±0.142	2.758±0.398	2.614±0.305
Venous			
Placebo	1.282±0.121	3.046±0.423	2.900±0.324
Treatment	1.282±0.095	3.182±0.459	2.829±0.387
Arteriovenous			
Placebo	−0.047±0.055	−0.305±0.085	−0.093±0.094
Treatment	−0.138±0.054	−0.424±0.111	−0.215±0.088
		Pyruvate, mM	
Arterial			
Placebo	0.028±0.005	0.054±0.012	0.066±0.010
Treatment	0.021±0.003	0.055±0.010	0.073±0.016
Venous			
Placebo	0.023±0.003	0.072±0.014	0.095±0.014
Treatment	0.019±0.002	0.077±0.014	0.111±0.021
Arteriovenous			
Placebo	0.005±0.002	−0.018±0.006	−0.029±0.007
Treatment	0.002±0.002	−0.022±0.006	−0.038±0.008

Values are means ± SE.

TABLE 4. *Fuel substrate concentration in venous blood*

	Diet	Rest	30 min	Exhaustion
Glycerol, μM	Placebo	0.14±0.01	0.24±0.02	0.44±0.03
	Treatment	0.16±0.02	0.26±0.02	0.43±0.03
β-Hydroxybutyrate, μM	Placebo	0.08±0.02	0.05±0.01	0.12±0.03
	Treatment	0.06±0.02	0.04±0.01	0.11±0.02
Free fatty acids, meq/l	Placebo	0.46±0.09	0.51±0.17	0.64±0.15
	Treatment	0.35±0.05	0.43±0.12	0.62±0.10

Values are means ± SE.

TABLE 5. *Respiratory exchange ratio and glucose and lipid oxidation*

Time, min	Diet	RER	Glucose, g	Lipid, g
0–10	Placebo	0.94±0.01	26.9±1.5	2.9±0.3
	Treatment	0.96±0.01	30.6±1.3	1.8±0.4
10–20	Placebo	0.91±0.01	25.2±2.5	3.6±0.4
	Treatment	0.92±0.01	25.7±1.7	3.1±0.3
20–30	Placebo	0.90±0.01	24.9±2.2	3.9±0.3
	Treatment	0.93±0.01	26.7±1.8	3.3±0.3
30–40	Placebo	0.90±0.01	23.1±2.0	4.4±0.4
	Treatment	0.94±0.01	25.8±2.2	3.3±0.5
40–50	Placebo	0.93±0.01	27.8±2.4	3.2±0.4
	Treatment	0.94±0.01	29.1±2.2	2.8±0.3
0–50	Placebo	0.92±0.01	128±10	18.0±1.3
	Treatment	0.94±0.01	140±8	14.3±1.5
Total	Placebo	0.92±0.01	165±17	23.3±2.6
	Treatment	0.94±0.01	203±15*	22.2±1.3

Values are means ± SE. RER, respiratory exchange ratio. Total, oxidation of glucose or lipid summed over the entire exercise trial (placebo, 66 min; treatment, 79 min). * $P < 0.05$ vs. placebo.

diets. Glucose and lipid oxidation did not differ between diets at any individual sampling period during exercise.

Blood flow at rest was 533 ± 11 and 531 ± 13 ml/min after feeding of the placebo diet and DHAP diet, respectively (P = NS). After 30 min of exercise, blood flow was 914 ± 45 and 924 ± 36 ml/min during the placebo trial and the DHAP trial, respectively (P = NS). At exhaustion, blood flow was 948 ± 22 and 990 ± 10 ml/min

during the placebo trial and the DHAP trial, respectively (P = NS).

Two subjects developed borborygmi and diarrhea with DHAP feeding but were able to perform the exercise trial without difficulty. No other side effects were associated with the placebo or treatment diets.

DISCUSSION

Dietary consumption of DHAP as a portion of a high-CHO diet for 1 wk increased leg exercise endurance by 20% in untrained male subjects. Before exercise, arteriovenous difference and fractional glucose extraction were greater for the DHAP than for the placebo diet. These responses persisted during exercise. Indirect estimate of fuel oxidation suggests that the ergogenic effect of a high-CHO diet containing DHAP was likely due to an increased availability of glucose for oxidation by exercising muscle.

After ingestion of a 70% CHO diet containing DHAP, subjects in the present study experienced the identical improvement in endurance capacity as subjects who performed arm exercise after consumption of a 55% CHO diet supplemented with DHAP (17). Therefore the effect of DHAP on endurance capacity was evident despite consumption of a placebo diet known to be ergogenic in nature.

Increased muscle glucose extraction may in part explain the increase in endurance capacity secondary to feeding of DHAP. After dietary consumption of DHAP, arteriovenous glucose difference was 89 and 63% greater at rest and after 30 min of exercise, respectively, compared with the placebo trial. Fractional extraction of glucose increased by 80 and 57% at rest and after 30 min of exercise, respectively, with the DHAP diet. Multiplication of arterial glucose concentration, fractional extraction of glucose, and blood flow yields the following glucose extraction (in mmol glucose/min): (*1*) rest, placebo 0.009, DHAP 0.196; (*2*) 30 min, placebo 0.665, DHAP 0.914; and (*3*) exhaustion, placebo 0.813, DHAP 1.009. Glycogen utilization during exercise was nearly identical between the placebo and DHAP conditions (Fig. 1). Therefore an increase in muscle extraction of glucose likely accounted for the 20% increase in glucose oxidation during the DHAP trial.

The effect of DHAP on glucose extraction was greatest at rest. During exercise, glucose extraction was remarkably similar after leg and arm exercise after DHAP supplementation (17). With leg exercise, arteriovenous glucose difference was 0.36, 1.06, and 1.07 mM at rest, during exercise, and at exhaustion, respectively. With arm exercise, arteriovenous glucose difference was 0.60, 1.00, and 0.97 mM before, during, and after exercise, respectively (17). Also, with leg exercise, fractional extraction of glucose was 7.9, 23.0, and 25.1% at rest, during exercise, and at exhaustion, respectively. With arm exercise, fractional extraction of glucose was 11.1, 22.5, and 24.3% before, during, and after exercise, respectively. Therefore the effects of DHAP on muscle glucose extraction are present for diets containing normal and high amounts of CHO.

Resting muscle glycogen concentration was not differentially influenced by feeding DHAP. This is in contrast to our previous report (17) involving prolonged exercise, in which resting muscle glycogen concentration was 30% higher after DHAP was fed compared with an isocaloric amount of glucose. However, the two investigations differ with respect to the percentage of CHO in the diet. The previous investigation employed a placebo diet containing 55% of kilocalories as CHO. A "glycogen-loading" effect would not be expected after this placebo diet. In contrast, the diets used in the present study contained CHO as 70% of the total daily caloric intake. In this case, both the placebo and DHAP diets appear to have provided a glycogen-loading effect. Therefore the experimental paradigm of the present study probably controlled for a differential contribution of muscle glycogen to the total energy yield during prolonged exercise. Although not evaluated in our two studies, differences in muscle fiber types between subjects might explain the glycogen concentrations that we observed. Because arterial glucose did not differ between dietary conditions, the ergogenic effect after the DHAP diet is likely attributable to the greater extraction of blood-borne glucose.

Blood flow at rest and during exercise was similar after feeding of the placebo and DHAP diets. Therefore increases arteriovenous glucose differences after consumption of DHAP probably represent actual increases in glucose extraction from blood by muscle. Irrespective of blood flow, fractional extraction of glucose was increased before and during exercise. Although resistivity of blood may vary with viscosity (11), hematocrit changes with exercise were similar after both diets were fed. Therefore comparison of blood flow between trials was considered valid. The indirect electrical impedance technique has only been validated by comparison with cardiac output dye techniques during rest and exercise (5). However, the nearly identical blood flow values of trials at rest, during exercise, and at exhaustion in the present study suggest that blood flow probably did not differentially affect the metabolic parameters investigated.

Gas exchange was not measured continuously during exercise trials and therefore cannot be used to conclusively determine fuel oxidation. Nevertheless, the estimation of glucose and fat oxidation provides some insight into the effect of DHAP on fuel utilization during exercise. At each 10-min sampling period during exercise, glucose oxidation did not differ between the DHAP trial (mean 90.5 ± 1.1%) and the placebo trial (mean 88.0 ± 1.1%). Fat oxidation between trials also did not differ at these sampling periods. However, total glucose oxidation was 20% greater ($P < 0.05$) with DHAP, as was endurance time.

Venous concentrations of glycerol, β-hydroxybutyrate, and free fatty acids were the same between the DHAP and placebo trials. These findings, in conjunction with the effect of DHAP on fat oxidation, suggest that energy derived from lipolysis was not influenced to a great extent by dietary DHAP supplementation. These observations, which are in agreement with those seen during arm exercise (17), provide evidence that DHAP does not impair free fatty acid mobilization during prolonged submaximal exercise.

Although detailed investigations of the effect of DHAP

on glucose oxidation before and during exercise remain to be performed, our data provide evidence for speculation concerning the mechanism of enhanced exercise endurance with DHAP feeding. Consumption of DHAP results in increased muscle glucose extraction before and during exhaustive exercise. A greater glucose extraction may spare muscle glycogen in exercising muscle. Increased glucose extraction and/or spared muscle glycogen provide fuel for prolongation of exercise endurance capacity. Our finding that the rate of glucose oxidation does not significantly increase during exercise with DHAP does not invalidate this hypothesis. Coyle et al. (4) have reported enhancement of endurance capacity with CHO feeding during exercise where subjects shifted from glycogen to glucose as the primary source of total CHO oxidation. However, an increased rate of CHO oxidation was not found. DHAP may not increase the rate of glucose oxidation during exercise but may prolong glucose oxidation and subsequently extend exercise endurance. The finding that DHAP supplementation significantly increases both total glucose oxidation and exercise endurance time by 20% seems to support this conclusion.

The results of the present study coupled with our previous study of arm exercise (17) establish that submaximal endurance capacity can be increased after dietary supplementation of DHAP for 7 days. DHAP supplementation increases fractional extraction of glucose identically in both leg and arm muscle. Therefore, given the similarities in metabolic properties of human skeletal muscle, DHAP probably increases glucose availability to all skeletal muscle in a more or less equal manner.

It is concluded that dietary intake of DHAP enhances leg exercise endurance capacity. Feeding of these three-carbon compounds increases glucose extraction by muscle during submaximal leg exercise after consumption of a diet containing 70% CHO.

The authors express appreciation to Judith E. Arch for time and dedicated service. They also thank Drs. Robert H. Miller, Arthur L. Hecker, and Jeffrey Falkel for assistance.

This work was funded in part by the National Institutes of Health Division of Research Resources General Clinical Research Center Grant RR-000056.

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Received 8 December 1989; accepted in final form 29 June 1990.

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