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Dietary intervention and training in swimmers

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Summary. To ascertain if muscle damage occurred in swimmers as a result of high-intensity training and to find if fluid and dietary manipulation could affect muscle damage, we studied 40 members of the University of Florida swimming team using creatine kinase (CK) and lactic dehydrogenase (LDH) as markers of muscle damage during a 6-month period of intensive training. During this time, training intensity, fluid intake during exercise and dietary supplementation were all modified one by one to examine their individual effects. During a control period of 4 weeks, all swimmers drank water before and during (120 min) workouts. CK in men at the end of this period averaged 315, SD 122 (normal $< 170\ IU \cdot l^{-1}$). Half of the swimmers were then given 500 ml of a glucose-electrolyte solution (GES) (Na 21 $mmol \cdot l^{-1}$, Cl 13 $mmol \cdot l^{-1}$, K 2.5 $mmol \cdot l^{-1}$, PO_4 5 $mmol \cdot l^{-1}$ and glucose 6%) before workouts and twice at intervals during the workout, while half continued to drink the same volume of water. One week after division into fluid groups, the workout intensity was increased by about 10%. Another week later CK had increased to 500, SD 180 $IU \cdot l^{-1}$ in swimmers drinking water, but fell to 280, SD 105 $IU \cdot l^{-1}$ in those drinking GES ($P < 0.05$). The second phase of the study began after a 4-week control period during which all athletes drank water before and during workouts. The swimmers were divided into four matched groups. Group I drank water before and during workouts and 250 ml of a 16% sucrose solution after; group II drank water before and during exercise and 250 ml of a milk protein supplement (MPS) containing 15 g lactalbumin and 16% sugar afterwards; group III drank GES before and during and the sucrose drink after exercise; group IV drank GES before and during and the MPS drink after exercise. Then during a 6-week period, the intensity of exercise was progressively increased by 25%. CK increased 61% ($P < 0.01$) in group I men, while it fell 12% ($P < 0.05$) in groups II and III, and 41% ($P < 0.01$) in group IV. In women, CK in group I increased 18% ($P < 0.05$); in group II it decreased 3.5%, in group III was unchanged, and in group IV declined 12.6% ($P < 0.05$). The final phase of the study was performed on 8 olympic swimmers who performed identical workouts each Saturday for 4 weeks. The 1st week they ingested water before and during exercise and the 16% sucrose solution afterwards. The 2nd week the GES solution was consumed before and during exercise and the sucrose solution afterwards. The 3rd week water was consumed before and during and MPS afterward and the 4th week GES before and during and MPS afterwards. Determination of CK and LDH before, immediately after, and at intervals afterwards showed that CK and LDH increased less when GES was the test fluid during exercise than when water was consumed. Recovery, as judged by return of CK and LDH to control values was more rapid when MPS was the post-exercise fluid than when the sucrose solution was given.

Key words: Exercise - Muscle damage - Creatine kinase - Lactic dehydrogenase - Recovery

Introduction

It is generally accepted by coaches and athletes that high-intensity training is necessary if an athlete is to reach a level of competence to allow successful competition on an international level (Forbes 1985). In pursuing competitive goals, however, an athlete may overtrain and cause muscle damage (Ahlborg et al. 1974; Davies and White 1981; Hikida et al. 1983; Newham et al. 1984), which can inhibit progress toward this goal.

Utilizing creatine kinase (CK) as an index of muscle damage (Newham et al. 1983; Troup et al. 1989; Vaananan et al. 1986) we studied the University of Florida swimming team to determine whether there was evidence of muscle damage induced by overwork (Friden

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et al. 1983). We then used fluid and dietary interventions to determine whether we could accelerate the rate of muscle recovery during the interval between training sessions so that the swimmer, with muscles completely recovered from the previous bout of exercise, could train hard consistently, and thus accelerate training and, hopefully, improve performance.

Methods

Forty swimmers, 20 men and 20 women, members of the University of Florida swimming team, served as subjects. Characteristics of the swimmers are shown in Table 1. The study was begun 6 weeks after the start of autumn training and was carried out in several phases. First, all swimmers were started on a workout schedule which was constant for each day of the week. Two workouts of 2 h duration were performed each day. Blood was obtained each Tuesday immediately preceding the afternoon workout. After 4 weeks of training at a constant intensity, the swimmers were divided into two matched groups (Table 1). Groups I and II drank water; groups III and IV drank a glucose-electrolyte solution (GES). The groups were formed by the swimming coaches according to the event the swimmer participated in and the ability of the swimmer. The groups were formed without knowledge of which fluid they would consume. One group drank 500 ml GES (Na 21 $mmol \cdot l^{-1}$, Cl 13 $mmol \cdot l^{-1}$, K 2.5 $mmol \cdot l^{-1}$, PO_4 5 $mmol \cdot l^{-1}$ glucose 6%), immediately before each workout and again after 45 and 70 min of swimming; the other group continued to drink water, on the same schedule, as had all 40 swimmers before the division. One week after the division, the intensity of training was increased by 10% and then kept constant for 3 more weeks. During these training sessions rectal temperature of the swimmers was monitored at 5–10 min intervals with an indwelling thermistor (YSI 400) and sweat loss estimated by weight loss plus fluid ingested over the course of the exercise session.

During part of each workout swimmers were tethered to a weight by means of a line extended over a pully in the ceiling of the building. Intensity of the exercise was varied by increasing the weight lifted or by increasing the number of lifts, or both. Intensity of exercise was also increased by increasing the number of laps each subject swam or by increasing the swimming speed.

For the second phase of the study the swimmers, again following 4 weeks of constant intensity work drinking water before and during exercise as in phase 1, were divided into four matched groups (see Table 1). Group I drank 500 ml water immediately before and another 1000 ml during exercise and drank 500 ml of an orange-flavored drink containing 16% sucrose immediately after each training session. Group II drank water before and during exercise as previously described but drank 250–500 ml of the milk protein supplement (MPS) containing 15 g protein (lactalbumin) and 16% sugar immediately after each exercise session. Group III drank 500 ml GES before and 1000 ml during exercise and drank the fruit-flavored sucrose drink immediately afterwards. Group IV drank GES before and during exercise as described above and drank MPS immediately afterwards. The post-exercise fruit-flavored drink contained 16% sucrose so that the carbohydrate load given immediately after exercise was approximately the same for all subjects. Blood for CK determination was again obtained each Tuesday before the afternoon workout.

Intensity of exercise remained constant for 1 week following division into the four study groups and was then increased three times during a period of 4 weeks.

Eight swimmers, all world class, participated in the final phase of the study. Blood for control CK and lactic dehydrogenase (LDH) measurements was obtained at 0730 hours each Saturday, approximately 12 h after the conclusion of the previous workout. A standard 2.5 h workout was then performed and blood was drawn immediately after the workout, and again 3, 8 and 22 h after completion of the workout.

The 1st week swimmers drank 500 ml water before exercising and drank water ad lib throughout the workout. The sucrose supplement was given afterwards; a regular training table diet was consumed.

The 2nd week, a workout identical, as nearly as possible, with the one carried out the previous Saturday, was performed. GES (500 ml) was taken immediately before the workout and ad lib during exercise. Again the sucrose supplement was given afterwards and, as before, a training table diet was consumed.

The 3rd week, the same workout was performed as the one performed the previous 2 weeks. Water was given, 500 ml before and ad lib throughout the workout and MPS, either 250 or 500 ml, immediately after and again twice later in the day. A training table diet was again taken.

The fourth study was similar to the previous three except GES was consumed before and during exercise and MPS was consumed immediately afterward.

Blood samples were allowed to clot and were then separated and analyzed immediately for CK (Newham et al. 1983) and LDH. Statistical analysis was carried out using analysis of covariance for multiple observations (Winer 1971). The purpose, benefits and risks were explained before the experiment began and informed consent was obtained from all participants.

Table 1. Characteristics of athletes

Group	Men/women	Surface area (m^2)	Height (m)	Age (years)
I	5/5	1.76 (0.05)	1.69 (12)	20.0 (1.4)
		1.68 (0.05)	1.59 (16)	20.0 (1.8)
II	5/5	1.79 (0.09)	1.77 (18)	20.2 (1.1)
		1.63 (0.07)	1.59 (12)	20.6 (1.2)
III	5/5	1.71 (0.06)	1.73 (17)	21.0 (1.1)
		1.64 (0.01)	1.64 (16)	19.6 (1.0)
IV	5/5	1.80 (0.08)	1.68 (14)	21.8 (2.3)
		1.67 (0.06)	1.58 (15)	21.0 (2.2)

Values are mean (SD)

Results

Data for men for the first phase of the study are shown in Fig. 1. It is apparent that CK was stable until a 10% increase in exercise intensity was instituted. In swimmers drinking water, a 72% rise in CK followed increased intensity while in swimmers drinking GES, CK gradually declined. The difference between the two groups following increased intensity exercise is significant ($P<0.05$).

Rise in rectal temperature during the first 45 min of exercise in men drinking water was 0.044, SD 0.004° $C \cdot min^{-1}$. Temperature then remained relatively constant or slowly declined as swimming pace decreased. Sweat loss over the entire 2-h workout (weight loss plus fluid consumed) in men drinking water was 27, SD 4 $ml \cdot min^{-1}$. In the group of men drinking GES, temperature increased 0.034, SD 0.003° $C \cdot min^{-1}$ and sweating occurred at a rate of 19.7, SD 2.5 $ml \cdot min^{-1}$. Differences in rate of temperature rise and sweat loss between the two groups were both significant ($P<0.05$). In women drinking water, rectal temperature increased at a rate of 0.038, SD 0.001° $C \cdot min^{-1}$ and sweat was

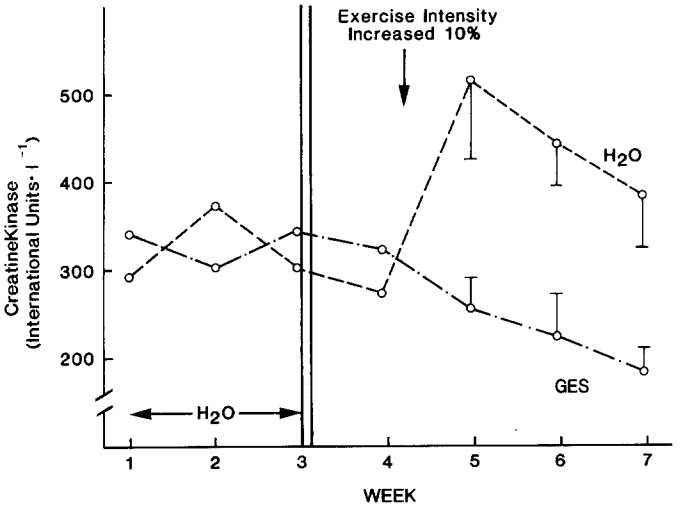

Fig. 1. The effect of exercise intensity and fluid replacement on creatine kinase in 20 athletes of the men's swimming team. All data points are mean values. H_2O, Water; GES, glucose-electrolyte solution

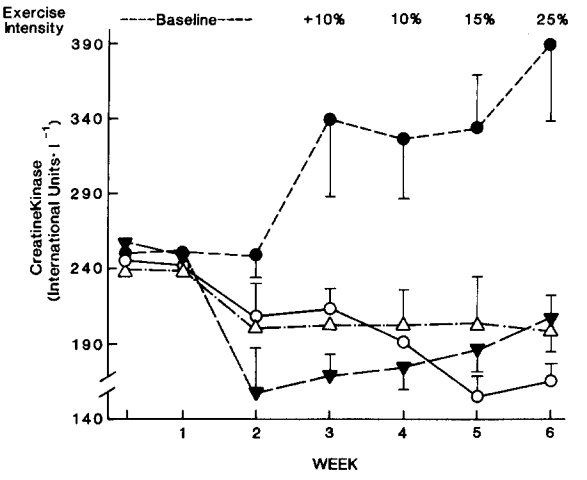

Fig. 3. Change in creatine kinase with increasing exercise intensity in men only comparing effects of GES vs H_2O and MPS vs sucrose. For symbols see legend to Fig. 2

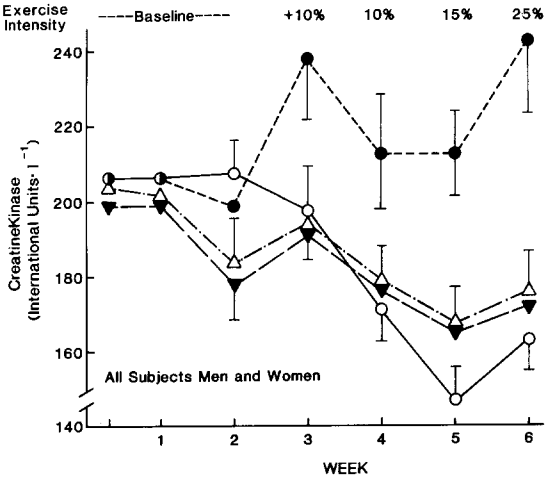

Fig. 2. Change in creatine kinase with increasing exercise intensity in 40 swimmers, 20 men and 20 women, comparing the effects of glucose-electrolyte solution (GES) vs water and milk protein supplement (MPS) vs sucrose. Group I ● H_2O-sucrose; group II ▼ H_2O-MPS; group III △ GES-sucrose; group IV ○ GES-MPS

lost at 24, SD 4.7 ml·min^{-1}. In women drinking GES temperature rose at a rate of 0.03, SD 0.003° C·min^{-1} and sweat was elaborated at 18.3, SD 2.5 ml·min^{-1}. Differences between the two groups of women for rate of temperature rise and sweat formation were both statistically significant ($P<0.05$).

In the next phase of the study the swimmers were divided into four matched groups. Data for all subjects, both men and women, are shown in Fig. 2. CK had become stable over a course of 4 weeks before the athletes were divided into separate study groups. After blood had been obtained at week 1, the four fluid regimens were started. At week 2 intensity of exercise was increased an estimated 10%; at week 4 it was increased an additional 5% and at week 5 it was increased again by an estimated 10% so that during the 6 weeks, intensity of exercise was increased approximately 25%. It is apparent that the response of CK to exercise was different in the water-sucrose-treated group compared with any of the other three groups. In group I, CK increased 25% in response to an increased work load, while in groups II and III it decreased 15%, and in group IV, CK declined 23%. Group I is different from groups II, III and IV with a confidence level of $P<0.01$. Group IV is different from groups II and III with a confidence level of $P<0.05$.

Data for men only are shown in Fig. 3. Again control data are shown to the left and changes in CK for each week during ingestion of test beverages are shown to the right. While changes during the experimental period are essentially the same as those shown for the entire group in Fig. 2, it is apparent that the change in men was greater than that observed for the group as a whole. The rise in CK in men following the initial 10% increase in intensity of exercise was 37% ($P<0.05$). The rise over the entire 6 weeks was 150 IU·l or 61% ($P<0.01$). In contrast, in each of the three supplemented groups there was a significant decrease in CK during the 6-week experimental period. In the cases of groups II and III, CK decreased 30 IU·l^{-1} or 12% while in group IV, which received both GES and MPS, CK decreased 82 IU·l^{-1} or 41% even though the work performed had increased by 25%. Group I is different from groups II, III and IV with a confidence level of $P<0.05$.

Figure 4 shows the same information for women swimmers. While the changes again are similar to those observed in men, it is apparent that the magnitude of change in CK in the women is much less than it was in the men. Statistically, the CK measured in group IV was significantly ($P<0.05$) different than that of group I. It is of interest to note that the effect of GES in men

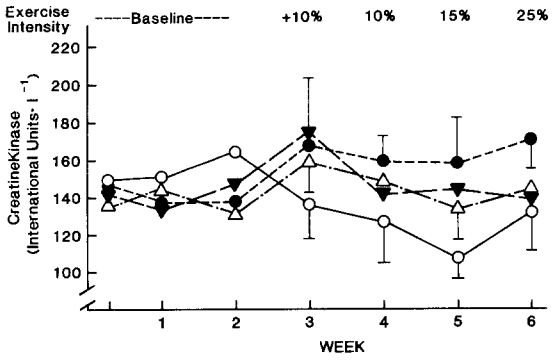

Fig. 4. Change in creatine kinase with increasing exercise intensity in women only comparing the effects of GES vs H_2O and MPS vs sucrose. For symbols see legend to Fig. 2

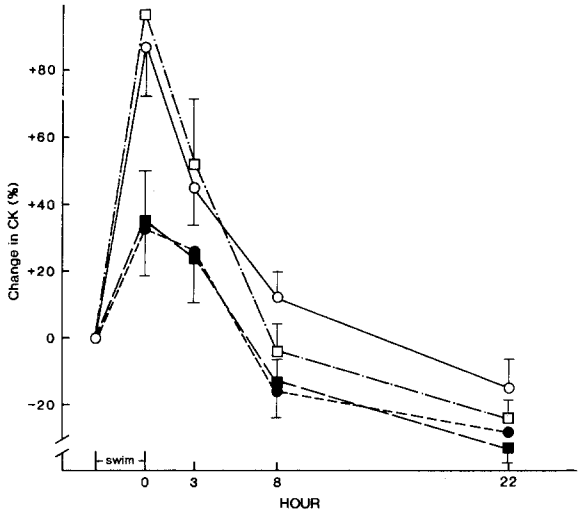

Fig. 5. The effect of vigorous exercise and the modifying effect of GES, MPS or both on the serum creatine kinase in 8 elite swimmers. For symbols see legend to Fig. 2

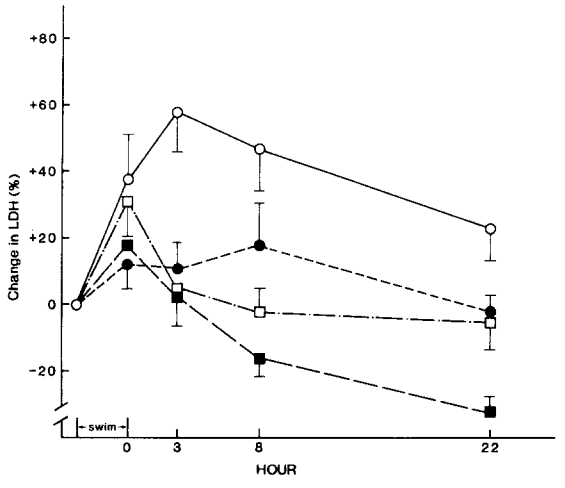

Fig. 6. Effect of vigorous exercise and the modifying effect of GES or MPS or both on the serum lactate dehydrogenase (LDH) in 8 elite swimmers. Week I ● H_2O-sucrose; week II ○ GES sucrose; week III □ H_2O-MPS, week IV ■ GES-MPS

was much greater than in women ($P<0.05$). Further, the reduction in CK of the GES-treated group as the work load increased was greater in men than in women $P<0.05$. The reason for this sex difference is unclear.

Figure 5 shows changes in CK concentration which occurred during the final phase of the study. It is apparent that CK rose far more dramatically immediately after exercise when water was the fluid used before and during exercise than when GES was the test fluid. It is also apparent that recovery was incomplete 8 h after exercise during the control water trial, while CK had returned to pre-exercise levels when either GES or MPS was used. The concentrations of CK in serum during week 2 and 4 are significantly different ($P<0.01$) from week 1 and week 3 at 0 h, and at 3 h ($P<0.05$). Week 2 and 4 values are different from week 1 at both 8 and 22 h ($P<0.05$), while week 3 is different ($P<0.05$) from week 1 at both 8 and at 22 h ($P<0.08$).

Figure 6 shows changes in LDH concentration with exercise as influenced by MPS and GES. We measured LDH, another intracellular enzyme which leaks from the cell as a result of injury, because it gives information about size and severity of injury. The molecular weight of LDH is much greater than that of CK and therefore kinetics of leak and clearance would be different (Newham et al. 1984).

It is again apparent that LDH increased far more during water trials than during GES trials and that recovery, as indicated by LDH, was more rapid when MPS was used. The combination of GES and a high protein supplement was most effective in allowing complete recovery of LDH. Week 2 and 4 values were significantly different ($P<0.01$) from week 1 at 0, 3, 8, and 22 h, while week 3 was significantly different ($P<0.01$) from week 1 at 3, 8 and 22 h.

Discussion

Several authors (Ahlborg 1974; Decombaz et al. 1979; Dohm et al. 1977, 1982, 1985; Millward et al. 1982; Rennie et al. 1981) have shown that protein is utilized for energy during vigorous exercise. These investigators, with the exception of Ahlborg, have also shown that leucine is utilized by direct oxidation. Dohm et al. (1977) have shown that the enzymes necessary for oxidation of leucine are increased by endurance training and suggest that this is a compensatory adaptation which helps ensure an energy supply as stores of carbohydrate are depleted. While the amount of protein catabolized during exercise is in dispute, Dohm et al. (1977) found that during a 16-km run, completed in approximately 1 h, athletes catabolized, on average, 37 g protein which accounted for approximately 18% of the calories expended.

Millward et al. (1982), in examining utilization of protein for production of energy during exercise, found that generous supplementation with carbohydrate during exercise had a sparing effect on utilization of protein while depletion of carbohydrate accelerated catabolism of protein. Other investigators (Decombaz 1979;

Dohm et al. 1985; Rennie et al. 1981) have also found greater depletion of amino acids when it is necessary to form pyruvate from amino acid precursors to sustain availability of carbohydrate during prolonged exercise.

If the magnitude of catabolism of protein reported during exercise is even reasonably accurate, large amino acid deficits, such as reported by Decombaz (1979) following endurance exercise, could limit the rate of recovery of muscle in the intra-exercise periods during training. The adaptation to greater use of protein for production of energy during exercise has even more serious implications for the elite athlete and for less gifted athletes who are working very hard in an attempt to become competitive. As their use of protein for energy increases, the amino acids available for muscle repair and synthesis would conversely diminish.

Previous work has shown that GES ingested before and during exercise prevent the depletion of vascular volume which occurs with massive sweating (Hawk 1904; Hommen 1985; Nadel 1984). Nose et al. (1988) have shown that salt containing solutions taken after volume depletion induced by exercise-thermal sweating are able to correct the reduction in blood volume while the same quantity of water is ineffective. When vascular volume is maintained, optimal cardiac output (Hommen 1985; Nadel et al. 1980; Nadel 1983), adequate perfusion of skin for heat dissipation, and muscle for metabolic processes (Hommen 1985; Nadel 1985), as well as heat removal results. It seemed reasonable, then, to postulate that GES, which spares protein breakdown by supplying adequate carbohydrate, maintains vascular volume and, therefore, cardiac output by replacing salt and water losses. In addition, it decreases the rate of rise of body temperature and, thus, the rate of sweat loss may protect from, or at least ameliorate, the muscle damage occurring with high-intensity exercise. Likewise, a protein supplement of high biological activity which replaces the essential amino acids catabolized during exercise might hasten the rate of repair of exercise-damaged muscle. This would promote more complete recovery before the next scheduled exercise session, and thereby allow the athlete to train hard consistently and make more rapid progress in developing strength, endurance and skill.

The present data show clearly that elevation of CK at a standard point after exercise tracked well with changes in exercise intensity in swimmers who drank water before and during exercise and drank a sucrose beverage immediately on completion of their workouts. In sharp contrast, CK increased only slightly with a 10% increase in intensity of exercise in the three groups who used a GES or MPS, or a combination of the two.

Again when CK and LDH were followed serially during a 24-h period immediately preceding exercise and at intervals after the exercise, the rise in the concentration of intracellular enzymes in serum was greatly attenuated by the use of GES immediately before and during exercise. Consumption of MPS immediately after and at intervals during the day was associated with a more rapid decline in the concentration of intracellular enzymes during the recovery period.

The results suggest that GES, ingested before and during exercise, protects muscle from exercise-induced damage. We believe this is because protein breakdown is diminished by the carbohydrate load (Millward et al. 1982) and because the electrolytes in the replacement fluid support maintenance of the extracellular fluid-blood volume allowing maintenance of the cardiac output with resultant good perfusion of muscle and skin which allows good heat removal. Replacing salt and water losses with water alone accomplishes none of these objectives. With less muscle damage, as indicated by CK and LDH levels, repair is more nearly complete before the next training session. An essential amino acid drink, such as MPS used in these studies, consumed immediately after exercise, replaces the amino acids lost to catabolism, allows more rapid repair and, thus, more complete recovery before the next training session. Use of a GES and MPS makes the goal of improved athletic performance more attainable as training can be accelerated more safely and the risk of athletic injury substantially reduced.

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