
Sodium bicarbonate ingestion does not restore the decrement in high-intensity exercise capacity induced by a 27 h Fast

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Summary. A 24 h fast reduces the time to exhaustion at 100 % of $\dot{V}O_2$ max relative to the 4 h post-absorptive condition. A consequence of fasting is a metabolic acidosis, which may be a cause of the premature fatigue. The purpose of this investigation was to orally administer sodium bicarbonate in combination with a one day (27 h) fast and determine whether exercise capacity was restored to that of the fed condition (3h post-absorptive). Six healthy men (mean \pm SD: age 26 ± 3 years; mass $68,3 \pm 6,0$ kg; height $1,75 \pm 0,04$ m; and $\dot{V}O_2$ max $55,4 \pm 7$ ml/kg/min) participated after giving their written consent. Subjects exercised to exhaustion at 98 ± 2 % of $\dot{V}O_2$ max on 4 occasions separated by seven days. Prior to two trials (FT and FC) subjects fasted for 27 h while prior to the other two trials subjects maintained their normal diet and were 3 h post-absorptive (NDT and NDC). On one of the fasted trials (FT) and one of the normal diet trials (NDT) subjects ingested 3,6 mmol NaHCO₃ per kilogram of body mass beginning three hours prior to exercise. On the remaining fasted (FC) and normal diet (NDC) trials subjects ingested a placebo, 3,0 mmol CaCO₃/kg body mass, beginning three hours prior to exercise. Exercise duration after both FC and FT was significantly lower than that attained after both NDC and NDT (200 ± 14 , 162 ± 14 , 200 ± 15 , and 169 ± 19 s for NDC, FT, NDT, and FC; $p < 0,001$). In conclusion, sodium bicarbonate ingestion after a 27 h fast did not restore high-intensity exercise capacity to that attained in the 3h post-absorptive condition, suggesting that a fasting-induced acidosis is not a significant component in the reduced performance in the fasted state.

Key words: sodium bicarbonate, high-intensity exercise, fasted trials, normal diet.

Резюме. Протягом 24 годин швидко скорочується час виснаження, практично на 100 % відновлюється показник $\dot{V}O_2$ max по відношенню до 4 годин постпоглинаючого стану. Наслідком такого стану може бути метаболічний ацидоз, який є причиною передчасної втоми. Мета цього дослідження – регуляція рівня бікарбонату натрію завдяки пероральному прийому протягом одного дня (24 години) з метою прискорення та визначення толерантності відновлення організму після фізичного навантаження у поствідновлювальному періоді (3 години після завершення). У дослідженнях брали участь шість здорових чоловіків за особистою письмовою згодою.

У ході дослідження проводилось вивчення підвищення відновлення організму після тривалого фізичного навантаження на фоні прийому спеціальних бікарбонат натрію складових фармакологічних препаратів по відношенню до результатів осіб, які приймали препарати типу «плацебо» і знаходились у звичайному для них (щоденному) режимі харчування.

Ключові слова: бікарбонат натрію, фізичні навантаження високої інтенсивності, обмежені випробування, звичайний режим харчування.

Introduction

Short term (~24 h) fasting results in a large increase in circulating concentrations of free fatty acids and ketone bodies (Loy et al. 1986, Nieman et al. 1987, Gleeson et al. 1988) as well as a significant reduction in hepatic glycogen stores (Nilsson and Hultman, 1973). The effect of such

a fast on skeletal muscle glycogen stores, however, is less clear with some authors reporting a decline in muscle glycogen following fasting and others reporting no effect. For example, Hultman (1967) reported that a 24 h fast reduced muscle glycogen levels by 25 % and this is supported by Coyle, Coggan et al. (1985) who reported that

muscle glycogen levels were 42 % lower immediately prior to exercise in subjects who fasted for 16 hours when compared to the glycogen levels attained when subjects fasted for 12 h and ingested a 145g carbohydrate feeding 4h prior to the muscle biopsy. In contrast, Maughan and Williams (1978) and Loy et al. (1986) reported that a 24 h fast had no significant effect on muscle glycogen levels. Nonetheless, in humans, short term fasting has been shown to have a detrimental effect on the performance of prolonged exercise of moderate intensity (Pequignot et al. 1980, Loy et al. 1986, Nieman et al. 1987, Maughan and Gleeson 1988).

The effects of short term fasting on high-intensity exercise capacity are not well documented. At fatigue during high-intensity exercise muscle glycogen (in samples of mixed fibre type) remains considerably higher than the level believed to limit the contribution of glycogenolysis to energy provision (Hermansen 1981). In addition, Katz et al. (1986) calculated that blood borne glucose provides only 1 % of the carbohydrate utilized during exercise to fatigue at 100 % of $\dot{V}O_2$ max. Based on these findings, the reduction in hepatic glycogen stores and possible reduction in muscle glycogen stores associated with a 24h fast should have little effect on muscle energy provision during high-intensity exercise. However, Gleeson et al. (1988) reported that, when compared to the 4 h post absorptive condition, a 24 h fast reduced exercise duration at a power-output corresponding to 100 % of maximum oxygen uptake ($\dot{V}O_2$ max) by 13 %. An additional consequence of the 24 h fast in the investigation of Gleeson et al. (1988) was lower plasma HCO_3^- and blood base excess (BE) prior to exercise relative to the values obtained in the 4 h post-absorptive condition. Because alterations in extracellular acid-base status influence the rate of lactate/ H^+ efflux from muscle (Hirche et al. 1975), and under some circumstances influence high-intensity exercise capacity (Jones et al. 1977, Sutton et al. 1981, Costill et al. 1984), it is possible that the alterations in acid-base status associated with fasting was a cause of fatigue in the investigation of Gleeson et al. (1988).

The purpose of this investigation was to determine whether the ingestion of sodium bicarbonate, designed to reverse a fasting-induced acidosis, after fasting for 24 hours would restore high-intensity exercise capacity to that observed in the fed condition.

Methods

Subjects. Six healthy men (mean±SD: age 26 ± 3 years; mass $68,3 \pm 6,0$ kg; height

$1,75 \pm 0,04$ m; $\dot{V}O_2$ max $55,4 \pm 7$ ml/kg/min) participated in this investigation after being informed of the risks involved and giving their written consent. All subjects participated in some form of regular physical activity. This investigation was approved by the local ethics committee before its initiation.

Preliminary Testing. Each subjects $\dot{V}O_2$ max was determined using a discontinuous protocol on an electrically braked cycle ergometer. $\dot{V}O_2$ max was verified approximately two days after the initial assessment. Seven days prior to the initial experimental trial each subject participated in a familiarization trial which was identical to the experimental trials except that there was no treatment administration and fewer blood samples were obtained.

Experimental Testing. On four separate occasions separated by seven days each subject exercised to exhaustion on an electrically braked cycle ergometer at a power output equivalent to 98 ± 2 % (mean±SE) of $\dot{V}O_2$ max. Subjects maintained a pedal cadence of 75–80 revolutions/minute. Exhaustion was considered the point at which the subject could no longer maintain a pedal cadence of 50 revolutions/minute.

Prior to two of the trials (FT and FC) subjects fasted for 27 h while prior to the remaining two trials subjects maintained their normal diet and were 3 h post-absorptive (NDT and NDC). On one of the fasted trials (FT) and one of the normal diet trials (NDT) subjects were on the Test condition and ingested 3,6 mmol $NaHCO_3$ per kilogram of body mass (0,3 g/kg body mass) over two hours beginning three hours prior to exercise. On the remaining fasted (FC) and normal diet (NDC) trials subjects were on the Control condition and ingested 3,0 mmol $CaCO_3$ /kg body mass (0,3g/kg body mass) as placebo over two hours beginning three hours prior to exercise. Treatment order was randomized and $NaHCO_3$ and $CaCO_3$ administration was double blind: $NaHCO_3$ and $CaCO_3$ were administered in gelatin capsules. The final meal before both the fasted and normal diet conditions was 3,15 MJ of a complete nutritional liquid (Ensure Plus, Abbott Laboratories Ltd, Queensborough, Kent ME11 5E) consisting of 53 % carbohydrate, 16,7 % protein, and 30 % fat. In each case the final meal was administered after a 10 h overnight fast, and subjects were instructed to consume the same final meal at the same time on the preceding evening. All trials were performed between 11:00 and 13:00 hours and each subject performed his four trials at the same time of day. Subjects maintained their normal pattern of physical activity and

diet between trials and abstained from strenuous exercise and alcohol consumption for 48 hours preceding each trial.

Before the experimental trials each subject reported to the laboratory and his nude body weight was obtained. The subject's hand was then immersed in water of 42°C for 10 min to arterialize the venous blood. This method has previously been validated by Forster et al. (Forster, Dempsey et al. 1972). A 21g butterfly cannula was then inserted into a vein on the dorsal surface of the subject's hand. This allowed for sampling of arterialized-venous blood immediately before ingestion of the final meal on the fasted (-27h) condition, before ingestion of the NaHCO₃ and CaCO₃ on the fasted condition (-3h), before ingestion of the final meal and NaHCO₃ and CaCO₃ on the Normal Diet condition (-3h), immediately prior to exercise (0), and at 2, 4, 6, 10, and 15 min after exercise. The cannula was kept patent between samples by flushing with a small volume of isotonic saline.

Analytical Methods. At each sampling point 2,5 ml of blood for determination of selected metabolites was obtained and dispensed into a tube containing K₃EDTA. In duplicate, 100 ul of this blood was deproteinized in 1 ml of 0,3 N perchloric acid, mixed, placed in iced water, and subsequently centrifuged: blood glucose concentration was determined on this sample using the glucose oxidase method (Boehringer Corporation, Lewes, UK) while blood lactate, alanine, beta-hydroxybutyrate (B-HB) concentrations were determined using the methods of Maughan (1982). An additional aliquot (1ml) of this blood was placed in an empty tube, centrifuged and the plasma drawn off for the determination of glycerol concentration (Boobis and Maughan 1983).

Statistical Analysis. Endurance capacity data was analyzed by one-way ANOVA for repeated measures. All other data were analyzed by three-way ANOVA (Bicarbonate or placebo X fasted or fed X time) with repeated measures on the time factor. Following a significant main effect or interaction, the Newman-Keuls post-hoc analysis was used to determine the location of differences between pairs of means. Differences for all analyses were considered significant if they achieved the probability level of 0.05. Descriptive data of the subjects are presented as mean±SD. All other data in text, tables, and figures are presented as mean±SEM. Standard error bars on the figures have been removed for clarity.

Results

Exercise Capacity. Exercise duration after both FC and FT was significantly lower than

that attained after both NDC and NDT (200 ± 14, 162 ± 14, 200 ± 15, and 169 ± 19 s for NDC, FT, NDT, and FC; $p < 0,001$). The magnitude of the reduction as a result of fasting was 16 ± 5 % when subjects were on the Control condition (FC vs. NDC) and 19 ± 3 % when subjects were on the Test condition (FT vs. NDT).

Body Mass. The 27 h fast resulted in a greater body weight reduction than the normal diet (0,27 ± 0,10, -0,47 ± 0,24, 0,42 ± 0,50, -0,90 ± 0,33 kg for NDC, FT, NDT, FC; $p < 0,003$; $p = 0,121$ for FT vs. NDC; $p = 0,051$ for FT vs. NDT; $p = 0,01$ for FC vs. NDC; and $p = 0,004$ for FC vs. NDT).

Effects of Fasting on Selected Metabolites.

Fasting resulted in a significant change in the profile of the blood metabolites. There was a 13 % reduction ($p = 0,0008$) and a 14,0 % reduction ($p = 0,0012$) in the glucose concentration from -27h to -3 h and from -27h to 0, respectively. The alanine concentration was significantly lower by 17,8 % ($p = 0,0100$) and by 14,8 % ($p = 0,0341$) from -27h to -3h and from -27h to time 0, respectively. The lactate concentration was reduced by 31 % ($p = 0,0276$) from -27h to -3 h but was not reduced from -27h at 0 h. Beta hydroxybutyrate was elevated by 22,6 % (NS) and by 85 % ($p = 0,0008$) at -3h and 0h relative to -27h. Finally, glycerol was elevated by 25 % at -3h ($p = 0,0378$) and by 64,8 % at 0 hours ($p = 0,0002$) relative to -27h

Pre and Post-Exercise Metabolites. A main effect of the exercise, in combination with the dietary history, was observed with glucose being significantly lower in the fasted condition than the fed condition ($p = 0,01$). There was a significant interaction for nutritional condition (fasted or fed) vs. time ($p = 0,0016$) for the glucose concentration. However, subsequent Newman-Keuls post-hoc analysis found no pairwise differences at any time point between fasted and fed conditions.

The post-exercise alanine concentration was significantly higher at all time points ($p < 0,0001$) compared with the pre-exercise time point, irrespective of fasting or 3 h post-prandial condition.

There was a significant nutritional condition (fasted or fed) X time effect for the lactate concentration; however, no significant differences between pairs of means were observed. There was a trend for a main effect ($p = 0,0672$) of a lower concentration of lactate observed in the fasted condition compared with the fed condition.

There was a significant Nutritional Condition (fasted or fed) X Time effect for beta hydroxybutyrate; however, the Newman-Keuls

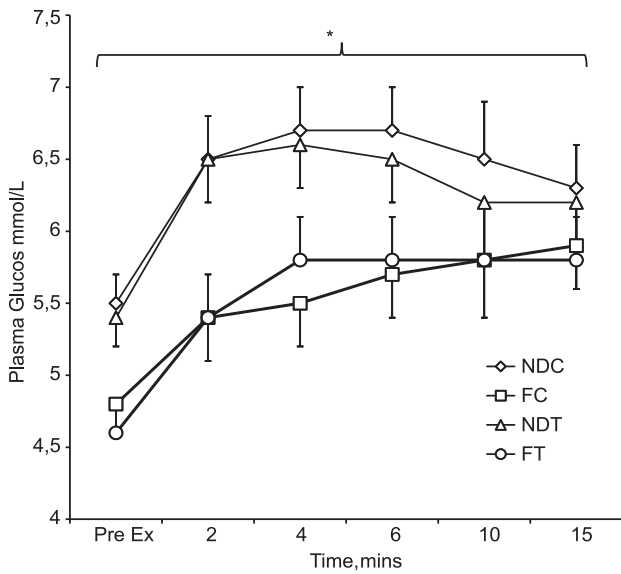


Figure 1. Glucose concentration (mmol/L) pre exercise (Pre Ex) and 2, 4, 6, 10, and 15 minutes after exhaustion. Data are presented as mean \pm SEM. Asterisk denotes a significant main effect between fasted and normal diet conditions ($p < 0,01$)

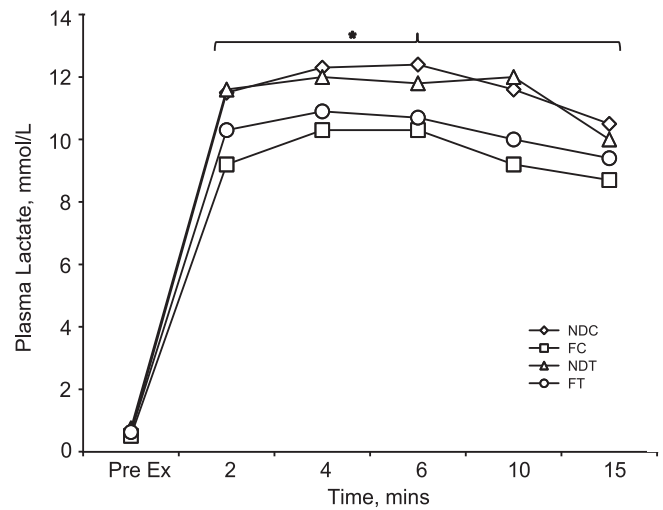


Figure 3. Lactate concentration (mmol/L) pre exercise (Pre Ex) and 2, 4, 6, 10, and 15 minutes after exhaustion. Error bars have been omitted for clarity. Asterisk denotes a significant time effect vs pre-exercise ($p < 0,05$)

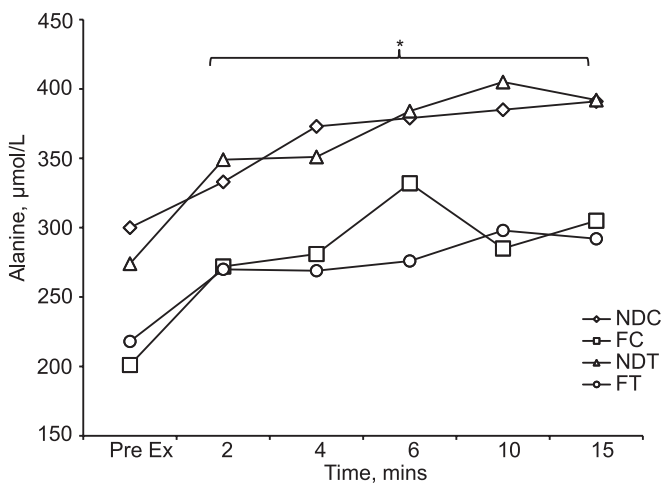


Figure 2. Alanine concentration ($\mu\text{mol/L}$) pre exercise (Pre Ex) and 2, 4, 6, 10, and 15 minutes after exhaustion. Error bars have been omitted for clarity. Asterisk denotes a significant time effect vs. pre-exercise ($p < 0,0001$)

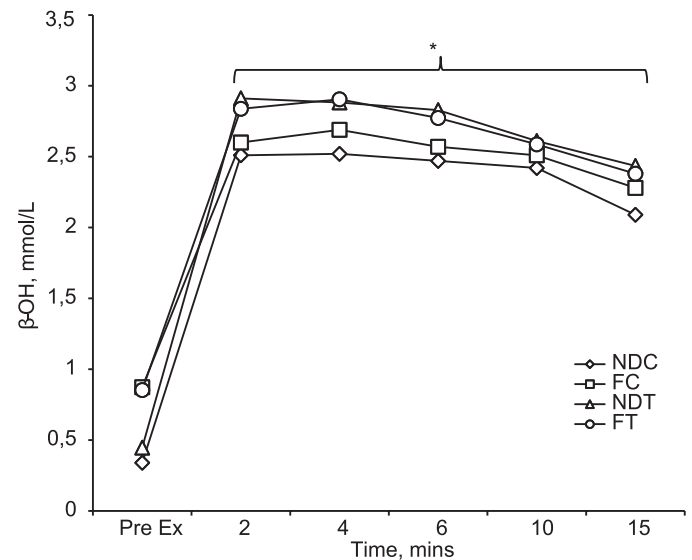


Figure 4. Beta-hydroxybutyrate concentration (mmol/L) pre exercise (Pre Ex) and 2, 4, 6, 10, and 15 minutes after exhaustion. Error bars have been omitted for clarity. Asterisk denotes a significant time effect ($p < 0,05$)

analysis revealed no pairwise significant differences. However, the pre-exercise beta hydroxybutyrate was 118 % higher for the fasted conditions than the fed conditions ($p = 0,191$).

There was a significant Nutritional Condition (fasted or fed) X Time effect for glycerol but the Newman-Keuls analysis did not reveal any pairwise significant differences. There was a significant fasting main effect with the values for the fasted

condition being significantly higher than for the fed condition ($p = 0,0013$).

Discussion

The major finding of the present investigation was that the ingestion of sodium bicarbonate did not reverse the negative effects of fasting on high-intensity exercise capacity. Further, sodium bicarbonate ingestion did not improve time to fatigue at 100 %

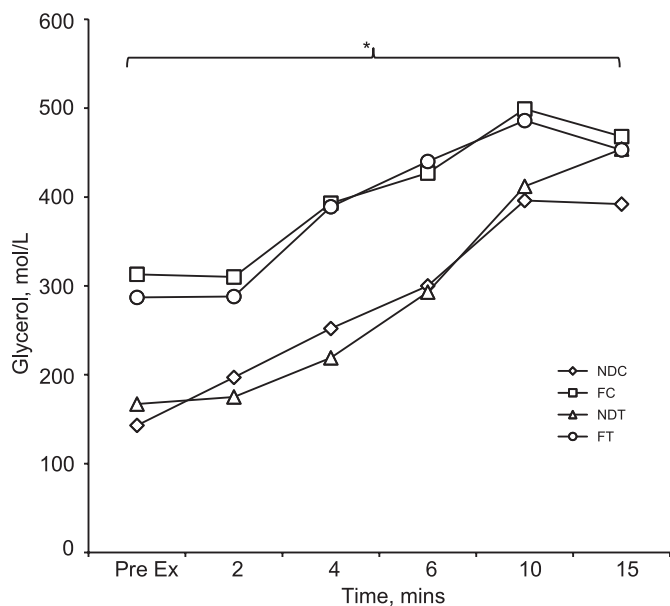


Figure 5. Glycerol concentration ($\mu\text{mol/L}$) pre exercise (Pre Ex) and 2, 4, 6, 10, and 15 minutes after exhaustion. Error bars have been omitted for clarity. Asterisk reveals a significant fasting vs. normal diet main effect ($p < 0,001$)

of $\dot{V}O_2\text{max}$ in the fed condition. Thus, it is likely that factors other than circulatory acidosis are responsible for fatigue during high-intensity exercise in the fasted condition. Additionally, because sodium bicarbonate ingestion did not improve exercise capacity in the fed condition, circulatory acidosis does not appear to be the cause of fatigue during exercise at $\sim 100\%$ of $\dot{V}O_2\text{max}$ in the fed condition.

It is possible that fasting reduces the pH of the intramuscular compartment. Larson et al. (1991) reported that the increase in the hydrogen ion concentration in muscle with intense exercise was significantly correlated ($r = 0,84$) with the duration of the fast prior to exercise. Therefore, it appears plausible that the premature fatigue induced by fasting was due to intramuscular acidosis that persisted despite sodium bicarbonate ingestion. Muscle pH and buffering capacity data were not obtained in the present investigation. However, if the alterations in acid-base status induced by the 27h fast negatively impaired intramuscular acid-base status, it is possible that the 3h period prior to exercise was not of sufficient duration and/or the induced alkalosis on treatment FT was not of sufficient magnitude to reverse these alterations. Thus, it is possible that intramuscular pH and/or buffering capacity were reduced prior to exercise on both of the fasting trials relative to the trials carried out in the post-absorptive condition and that this was a cause of the reduced exercise capacity.

Muscle glycogen data were not obtained in the present investigation. However, it is possible that muscle glycogen levels were lower in the fasted conditions when compared to the normal diet condition (Hultman 1967; Coyle et al. 1985). A difference in muscle glycogen between conditions would be important during prolonged exercise as the depletion of muscle glycogen stores is highly related to the onset of fatigue (Bergstrom et al. 1967). However, because muscle glycogen levels (measured in mixed fibre samples) remain high at the point of fatigue, the availability of muscle glycogen is not believed to be important to the performance of high-intensity exercise (Hermansen 1981). The data obtained from mixed fiber biopsy samples may be misleading; however, as the glycogenolytic (Harris et al. 1976) and glycolytic (Essen et al. 1975) capacities as well as the ability to generate power (Faulkner et al. 1986) are markedly higher in type II than in type I fibers. Greenhaff et al. (1991) have reported that during intense electrical stimulation to fatigue the glycogenolytic rate in type II fibers was 20 times higher than in type I fibres. In addition, these investigators suggested that because of the high glycogenolytic rate of type II fibres, reduced glycogen availability may limit the rate of ATP turnover in these fibres and therefore result in fatigue during high-intensity contraction. Thus, if muscle glycogen levels were lower in the fasted when compared with the normal diet condition prior to exercise, it is possible that reduced muscle glycogen availability in type II fibres was the cause of the lowered endurance capacity in FT and FC as compared to NDT and NDC. Supporting the contention that reduced carbohydrate availability can limit the performance of high-intensity exercise are investigations which suggest that a low dietary carbohydrate intake will impair exercise performance when compared to a moderate or high-carbohydrate intake (Maughan and Poole 1981; Greenhaff et al. 1987; Greenhaff et al. 1988a; Greenhaff et al. 1988b; Horswill et al. 1990; Davis et al. 1997; Balsom et al. 1999).

Short term fasting is also associated with a reduction in carbohydrate availability. Nilsson and Hultman (1973) have reported that a 24 h fast will reduce hepatic glycogen to 10-25 % of the normal level. The decreased glucose availability would certainly influence the ability to perform prolonged moderate intensity exercise as this type of exercise, if adequate fluid is ingested, has been shown to be dependent on the ability to maintain a high rate of carbohydrate supply and oxidation (Coggan and Coyle 1987;1988). However, for the performance of high-intensity exercise of short duration,

the supply of blood glucose to the working muscles appears to be of minimal importance (Katz et al. 1986). Katz and co-workers (1986) have suggested that above 75 % of $\dot{V}O_2$ max glucose uptake by the working musculature exceeds utilization. These investigators have calculated that during exercise at a workload requiring 100 % of $\dot{V}O_2$ max, blood glucose provides approximately 1 % of the carbohydrate utilized with muscle glycogen providing the other 99 %. Thus, the reduced liver glycogen stores probably did not limit muscle substrate availability to muscle during exercise in the fasted condition.

It is possible that the reduced carbohydrate intake prior to exercise in the fasted condition resulted in a reduction in cerebral glycogen and glucose prior to exercise (Dalsgaard 2006). Brain glycogenolysis supports neuronal activity and it has been suggested that the astrocyte glycogen level could reach critically low levels and result in central fatigue (Dalsgaard 2006). Indeed Nybo et al. (2003) have reported that prolonged exercise without carbohydrate supplementation led to a reduced central activation of muscle (indicative of central fatigue) when compared to the central activation when carbohydrate was fed during exercise. Thus, it appears that a 27 hour fast results in a reduction in carbohydrate availability directly and through lowered carbohydrate metabolites and that this may result in premature fatigue through CNS mechanisms. Future investigations should evaluate whether central fatigue is a plausible mechanism for the fatigue that occurs due to a 24 hour fast using electrical stimulation studies to evaluate central activation failure.

Hypohydration is another possible contributing factor to the decreased exercise capacity with a 27 hour fast. Indeed, studies examining Ramadan fasting, which is the Muslim practice of fasting during daylight hours, suggest that hypohydration occurs as a result of a disruption in normal eating and drinking habits (Leiper et al. 2003). As discussed by Leiper et al. (Leiper et al. 2003) in situations where the usual eating and drinking patterns are disrupted, there is greater reliance on physiological stimuli directly related to body water deficits. Significant hypohydration (2 %) may occur prior to the individual sensing the need to ingest fluid. However, hypohydration was likely not a significant contributor to the reduction in exercise capacity in the present investigation as Armstrong et al. (Armstrong et al. 1985) reported only a 3 % impairment in 1500 m running performance when a body weight deficit of 1,9 % was incurred through the use of diuretics. The 1500 m is run at an intensity that approximates $\dot{V}O_2$ max. The fasting induced decrement in body weight in the present

investigation was only 1.3 % for the FC condition and 0,7 % for the FT condition while the overall reduction in exercise capacity was 16 % for FC and 19 % for FT.

Another potential contributor to reduced high-intensity exercise capacity is reduced pyruvate dehydrogenase activity. Pyruvate dehydrogenase (PDH) catalyzes the conversion of pyruvate to acetyl co-A and thus irreversible conversion of glucose and glucose 6-phosphate to an essential component of the citric-acid cycle. Thus, the optimal functioning of pyruvate dehydrogenase is extremely important for the optimal use of muscle glycogen and blood glucose via aerobic metabolism. The aerobic contribution to energy metabolism during exercise that lasts 2 min is ~65 % (Medbo and Tabata 1989). A very small amount of this energy is provided by fat with almost all of it being provided by carbohydrate and almost entirely by muscle glycogen (Katz, Broberg et al. 1986). Thus, any reduction in PDH activity would reduce the ATP derived from the aerobic oxidation of glucose or glycogen.

Spriet et al. (2004), reported that the activation of pyruvate dehydrogenase was significantly lower after a 40 h fast (by ~55-60%) when compared to the pre-fast level. Thus, it is plausible that the 27 hour fast resulted in impaired PDH activation and that this resulted in impaired aerobic energy provision from carbohydrate and contributed to the reduced exercise capacity seen in the fasted conditions. The mechanism for the impaired PDH activity appears to be through increased activation of pyruvate dehydrogenase kinase-4 which acts to reversibly phosphorylate PDH (Peters, Harris et al. 2001; Spriet, Tunstall et al. 2004).

Interestingly, Pichard et al. (1988) reported that fasting (for 48 hours) resulted in a reduction in the resting phosphocreatine/phosphate, the Phosphocreatine/ATP, the free energy change of ATP hydrolysis. Further, ADP levels were elevated in the fasted rats compared to the control rats. Thus, although the mechanism has not been elucidated, it appears that energy metabolism is impaired and that there is an imbalance between ATP production and utilization even in the resting non-exercise condition.

In summary, the ingestion of sodium bicarbonate did not restore the high-intensity exercise capacity impaired by a 27 hour fast. Thus, circulatory acidosis does not appear to be the cause of fatigue during high-intensity exercise as a result of 27 hour fast. The contributing factors to fatigue as a result of a one day fast remain to be elucidated but intramuscular acidosis, reduced muscle glycogen

content in type II fibres, reduced cerebral glycogen and/or glucose availability, hypohydration, and/

or reduced PDH activity are plausible but unproven explanations for the reduction in exercise capacity.

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