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The effect of citrate loading on exercise performance, acid-base balance and metabolism

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Summary. Nine subjects ($\dot{V}_{O_{2max}}$ 65±2 ml·kg^{-1}·min^{-1}, mean±SEM) were studied on two occasions following ingestion of 500 ml solution containing either sodium citrate (C, 0.300 g·kg^{-1} body mass) or a sodium chloride placebo (P, 0.045 g·kg^{-1} body mass). Exercise began 60 min later and consisted of cycle ergometer exercise performed continuously for 20 min each at power outputs corresponding to 33% and 66% $\dot{V}_{O_{2max}}$, followed by exercise to exhaustion at 95% $\dot{V}_{O_{2max}}$. Pre-exercise arterialized-venous [H$^+$] was lower in C (36.2±0.5 nmol·l^{-1}; pH 7.44) than P (39.4±0.4 nmol·l^{-1}; pH 7.40); the plasma [H$^+$] remained lower and [HCO_3^-] remained higher in C than P throughout exercise and recovery. Exercise time to exhaustion at 95% $\dot{V}_{O_{2max}}$ was similar in C (310±69 s) and P (313±74 s). Cardiorespiratory variables (ventilation, $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$, heart rate) measured during exercise were similar in the two conditions. The plasma [citrate] was higher in C at rest (C, 195±19 µmol·l^{-1}; P, 81±7 µmol·l^{-1}) and throughout exercise and recovery. The plasma [lactate] and [free fatty acid] were not affected by citrate loading but the plasma [glycerol] was lower during exercise in C than P. In conclusion, sodium citrate ingestion had an alkalinizing effect in the plasma but did not improve endurance time during exercise at 95% $\dot{V}_{O_{2max}}$. Furthermore, citrate loading may have prevented the stimulation of lipolysis normally observed with exercise and prevented the stimulation of glycolysis in muscle normally observed in bicarbonate-induced alkalosis.

Key words: Citrate — Alkalosis — Buffering — Performance — Lactate — Glycerol — Free fatty acids

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Introduction

At exercise power outputs greater than 70–80% of maximal aerobic capacity ($\dot{V}_{O_{2max}}$) ATP production is supported by both oxidative phosphorylation and anaerobic glycolysis. The rise in intra- and extracellular concentrations of lactate and hydrogen ions that accompany the increased anaerobic glycolysis have been cited as factors contributing to fatigue (for reviews see Hermansen 1981; Sahlin 1983, 1986).

Increasing the "buffer" reserve of the body was suggested, over 50 years ago, as a mechanism available for combating the deleterious effects of acidosis on performance. Ingestion of plasma alkalinizing agents have been shown to improve performance of certain tasks relative to control conditions. Sodium bicarbonate has often been used as the alkalinizing agent with results in various types of activities showing both improvements (Costill et al. 1984; Goldfinch et al. 1988; Inbar et al. 1983; Jones et al. 1977; Sutton et al. 1981; Wijnen et al. 1984; Wilkes et al. 1983) and no effect on performance with alkalosis (Katz et al. 1984; Kindermann et al. 1977; Kowalchuk et al. 1984; McCartney et al. 1983; Parry-Billings and MacLaren 1986).

Fruit juices (high in potassium citrate) and alkaline salts of citric acid have also been used to raise plasma pH. Hewitt and Calloway (1936) found improvements in swimming performance in events ranging from 100 to 400 m, while endurance running and cycling were improved after alkali ingestion (Dennig 1937). In contrast, Johnson and Black (1953) found no improvement in performance time during a series of 1.5 mile cross-country races. Recently Parry-Billings and MacLaren (1986) observed that alkalosis induced by either sodium bicarbonate, sodium citrate or combined sodium bicarbonate-sodium citrate in-

gestion did not enhance performance during successive 30 s Wingate tests.

The ergogenic effects of alkalinizing agents on exercise performance have not been demonstrated conclusively. While the effects of sodium bicarbonate ingestion have been well documented, information on the effects of citrate ingestion on performance and metabolism is lacking. Citrate occurs in many foods and could provide a natural means of alkalinizing the body. An increase in plasma citrate levels could also raise the cytosolic citrate concentration and thereby potentiate the inhibitory effects of ATP on phosphofructokinase to inhibit the rate of glycolysis (Newsholme and Leech 1983) and slow the rate of glucose and glycogen utilization. The present study examined the effects of citrate ingestion on the plasma citrate concentration, plasma acid-base status, and exercise performance using a protocol that has previously been shown to improve performance with sodium bicarbonate ingestion (Jones et al. 1977; Sutton et al. 1981).

Materials and methods

Nine active university students [age, 23±1 years (mean±SEM); height, 1.77±0.003 m; mass, 68.4±2.9 kg; $\dot{V}_{O_{2max}}$, 4422±182 ml·min^{-1}] volunteered for the study after being given a detailed description of the procedures and possible risks and side effects. The study was approved by the University of Waterloo Office of Human Research, and written consent was obtained from all subjects.

Each subject was studied on three occasions: one preliminary session during which he performed a progressive exercise test to exhaustion (20 W·min^{-1} ramp protocol) to determine his $\dot{V}_{O_{2max}}$ and to assess appropriate workloads for the submaximal exercise studies, and two experimental sessions performed after oral ingestion of 500 ml of a glucose-free, orange-flavoured drink containing either sodium chloride as a placebo (P; dose, 0.045 g·kg^{-1} body mass) or sodium citrate (C; dose, 0.300 g·kg^{-1} body mass). Exercise was performed on an electrically braked cycle ergometer (Quinton Instruments Model 870).

The exercise protocol used in the present study was similar to that of Jones and coworkers (1977). Subjects reported to the laboratory after a light breakfast. After approximately 30 min rest the subject ingested one of two flavoured drinks. Exercise began 60 min post-ingestion and consisted of continuous cycle ergometer exercise at power outputs corresponding to 33% $\dot{V}_{O_{2max}}$ for 20 min, 66% $\dot{V}_{O_{2max}}$ for 20 min, and 95% $\dot{V}_{O_{2max}}$ until exhaustion (Fig. 1). Recovery was followed for an additional 20 min post-exercise.

Ventilation, O_2 uptake ($\dot{V}_{O_2}$) and CO_2 output ($\dot{V}_{CO_2}$) were monitored between 7–10 min and 17–20 min during each of the 33% and 66% $\dot{V}_{O_{2max}}$ power outputs, and throughout the 95% $\dot{V}_{O_{2max}}$ power output. Ventilation and gas exchange were monitored using an open circuit gas analysis system and minute values were calculated for consecutive 30 s intervals using methods described by Hughson and coworkers (1980). Ventilation was measured by pneumotachograph (Hewlett-Packard

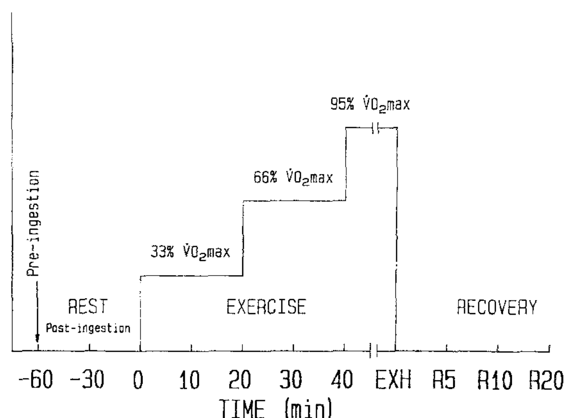

Fig. 1. Schematic representation of the protocol. EXH = time a exhaustion from 95% $\dot{V}_{O_{2max}}$; R5, R10, R20 = 5, 10, 20 min of post-exercise recovery

47303A) and O_2 and CO_2 concentrations in expired air were measured by a solid oxide electrochemical cell (Applied Electrochemistry Inc. S-3A O_2 analyzer) and infrared CO_2 analyzer (Beckman Instruments LB-2 CO_2 analyzer), respectively. Expired air was sampled from a 7.0 L mixing chamber and expired gas concentrations were matched to ventilation with an appropriate volume delay (Hughson et al. 1980).

Blood samples were obtained for analysis of blood gases, pH and metabolite concentrations at rest immediately prior to ingesting the flavoured drink, at 30 and 60 min post-ingestion; during exercise at 10 and 20 min at each of the 33% and 66% $\dot{V}_{O_{2max}}$ power outputs and at exhaustion from 95% $\dot{V}_{O_{2max}}$; and at 5, 10 and 20 min post-exercise. Blood was drawn from an indwelling catheter (Angiocath, 21 gauge) in a dorsal hand vein; the blood was "arterialized" by warming the hand in an electric heating pad. One blood sample was drawn anaerobically and analyzed for blood pH, PO_2 and PCO_2 by electrode (Radiometer ABL30 Acid-Base Analyzer). The bicarbonate anion concentration ($[HCO_3^-]$) was calculated using the Henderson-Hasselbalch equation (Severinghaus 1966). A second blood sample was drawn and an aliquot was removed and deproteinized in cold perchloric acid and assayed for lactate, glucose, and glycerol using fluorometric procedures (Lowry and Passoneau 1972); citrate was measured according to Toftegaard Nielsen (1976). The serum obtained from the remaining sample was assayed for free fatty acids (FFA) using fluorometric procedures (Miles et al. 1983). Hemoglobin concentration represented the average of three measurements determined by standard cyanomethemoglobin procedures. Micro hematocrit was measured in triplicate by centrifugation.

Statistical analysis consisted of repeated measures analysis of variance with main effects of subject and treatment repeated over time for all variables except endurance time which was treated by a paired t-test. The values are presented as mean±SEM.

Results

Administration of sodium citrate (C) was associated with a lower plasma $[H^+]$ ($p < 0.002$) and elevated $[HCO_3^-]$ ($p < 0.0001$) in the post-ingestion period than placebo (P) (Fig. 2). The $[H^+]$

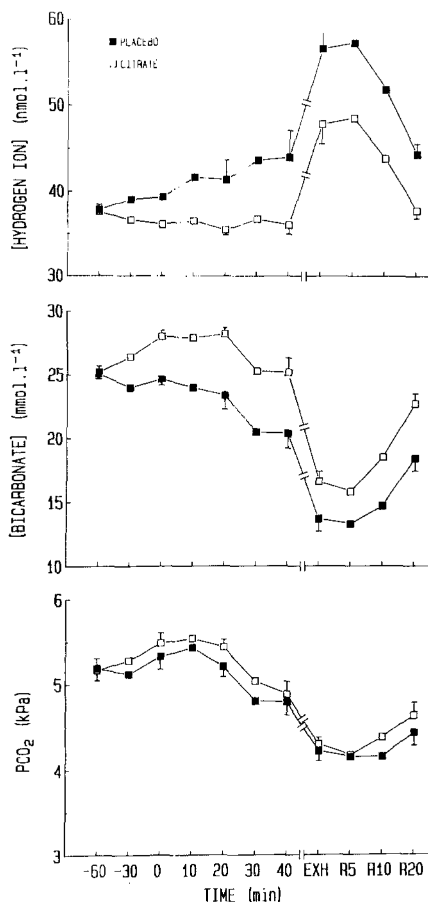

Fig. 2. The plasma concentration of hydrogen ions and bicarbonate ions, and PCO_2 (mean ± SEM) at rest, during exercise at 33% and 66% $\dot{V}_{O_{2max}}$, at exhaustion at 95% $\dot{V}_{O_{2max}}$ and recovery from exercise following the ingestion of sodium citrate and the sodium chloride placebo

and $[HCO_3^-]$ remained at pre-exercise levels during exercise at 33% $\dot{V}_{O_{2max}}$ but with increasing exercise intensity the $[H^+]$ increased ($p<0.01$) and $[HCO_3^-]$ decreased ($p<0.01$) from pre-exercise values. Differences between C and P remained throughout exercise and recovery ($p<0.05$).

Endurance time at 95% $\dot{V}_{O_{2max}}$ was not affected by citrate ingestion; the time to exhaustion was 310±69 s (C) and 313±74 s (P). Ventilation, $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$, and respiratory exchange ratio (RER) were similar in C and P ($p<0.05$, Table 1).

The pre-ingestion plasma [citrate] was 46±6 μmol·l^{-1}. Citrate loading raised the [citrate] to 195±19 μmol·l^{-1} ($p<0.01$) during the 60 min before exercise (Fig. 3). The [citrate] decreased during exercise at 33% $\dot{V}_{O_{2max}}$, and decreased only slightly in the remaining exercise and recovery. Although the [citrate] increased post-ingestion with P, it remained lower ($p<0.0006$) than C

Table 1. Ventilation, gas exchange and heart rate during exercise following ingestion of sodium chloride (P) and sodium citrate (C)

		% $\dot{V}_{O_{2max}}$				
		33		66		95
	Cond.	10	20	10	20	Exhaustion
Ventilation	P	35.5	32.1	60.4	62.8	121.7
(l·min^{-1})		(3.9)	(1.4)	(3.2)	(3.7)	(4.8)
	C	31.6	31.6	60.4	62.4	119.2
		(1.8)	(1.8)	(5.3)	(4.1)	(5.9)
O_2 intake	P	1785	1805	3131	3188	4285
(ml·min^{-1})		(69)	(51)	(144)	(108)	(151)
	C	1786	1804	3129	3151	4285
		(83)	(95)	(165)	(143)	(183)
CO_2 output	P	1568	1560	2877	2918	4698
(ml·min^{-1})		(69)	(46)	(115)	(104)	(160)
	C	1590	1576	2951	2899	4714
		(85)	(86)	(189)	(138)	(166)
RER	P	0.88	0.87	0.93	0.92	1.10
		(0.02)	(0.02)	(0.01)	(0.01)	(0.02)
	C	0.89	0.88	0.95	0.92	1.10
		(0.02)	(0.02)	(0.03)	(0.02)	(0.02)
Heart rate	P	121	127	169	177	197
(bt·min^{-1})		(6)	(8)	(8)	(8)	(5)
	C	120	125	166	173	193
		(6)	(8)	(10)	(10)	(5)

Values are mean (±SEM)

throughout the test; the peak [citrate] was 101±6 μmol·l^{-1} at 30 min post-ingestion.

The plasma [lactate] increased ($p<0.01$) during exercise at 66% and 95% $\dot{V}_{O_{2max}}$; the [lactate] at exhaustion was 12.8±0.9 mmol·l^{-1} (C) and 10.5±0.6 mmol·l^{-1} (P) (Fig. 4). No differences were observed in [lactate] between the two conditions ($p>0.05$).

The plasma [glycerol] increased ($p<0.05$) with increasing intensity of exercise; the [glycerol] was

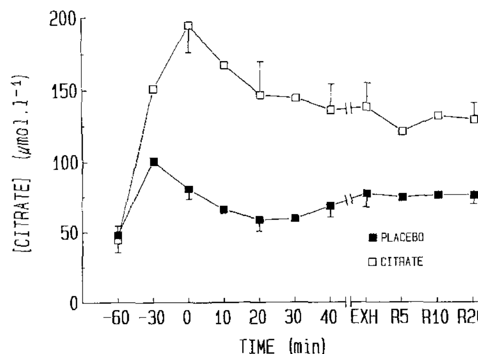

Fig. 3. The plasma citrate concentration (mean ± SEM) at rest, during exercise and recovery following ingestion of sodium citrate and the sodium chloride placebo

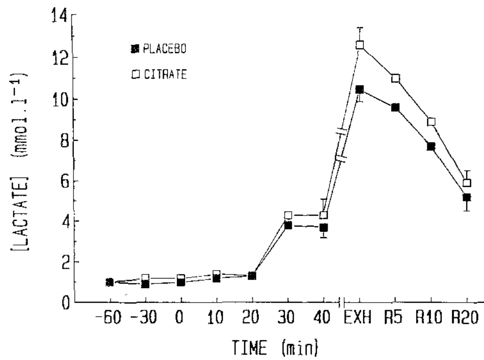

Fig. 4. The plasma lactate concentration (mean ± SEM) at rest, during exercise and recovery following ingestion of sodium citrate and the sodium chloride placebo

Table 2. Hematocrit and hemoglobin concentration at rest and after 20 min at each exercise intensity following ingestion of sodium chloride (P) and sodium citrate (C)

	Cond.	post-ingestion 0	post-ingestion 60	% $\dot{V}_{O_{2max}}$ 33	% $\dot{V}_{O_{2max}}$ 66	% $\dot{V}_{O_{2max}}$ 95
Hematocrit (%)	P	41.9 (0.4)	43.0 (0.5)	43.9 (0.6)	45.2 (0.6)	46.7 (0.7)
	C	42.2 (0.8)	42.3 (0.8)	42.9 (0.5)	44.2 (0.7)	45.9 (0.5)
Hemoglobin g L^{-1}	P	1.47 (0.04)	1.52 (0.04)	1.57 (0.05)	1.64 (0.04)	1.67 (0.05)
	C	1.45 (0.03)	1.47 (0.04)	1.51 (0.04)	1.57 (0.04)	1.59 (0.04)

Values are mean (± SEM)

greater in P than C ($p < 0.05$, Fig. 5). The plasma [free fatty acid] remained at rest levels throughout exercise (Fig. 5); no differences were observed between P and C ($p > 0.05$). The plasma [glucose] remained at rest levels during exercise at 33% and 66% $\dot{V}_{O_{2max}}$ but increased ($p < 0.05$) during exercise to exhaustion at 95% $\dot{V}_{O_{2max}}$ with no differences between P and C ($p > 0.05$, Fig. 5).

The [hemoglobin] and hematocrit increased ($p < 0.05$) with increasing exercise intensity. During exercise the [hemoglobin] and hematocrit were lower in C than P ($p < 0.05$, Table 2).

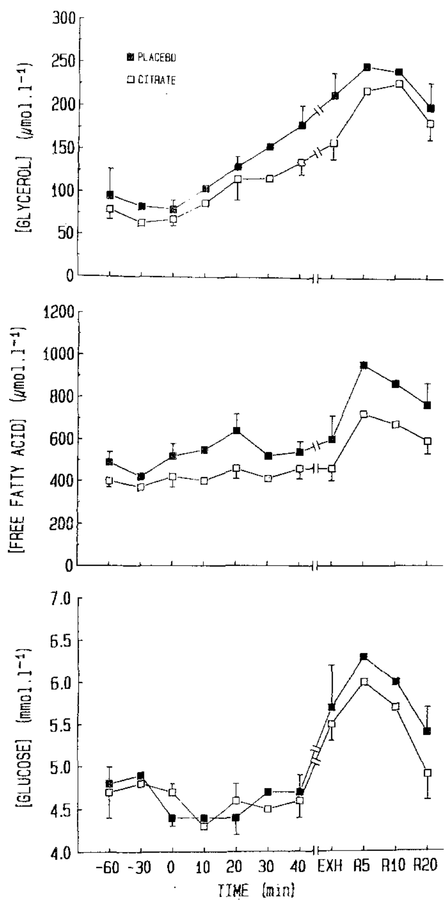

Fig. 5. The plasma concentration of glycerol, free fatty acids and glucose (mean ± SEM) at rest, during exercise and recovery following ingestion of sodium citrate and the sodium chloride placebo

Discussion

The present study examined the effects of sodium citrate loading on acid-base balance, metabolism and performance during submaximal and near-maximal exercise. The results demonstrated that compared to ingestion of a placebo (sodium chloride), sodium citrate loading (0.300 g·kg^{-1} body mass) had a significant alkalinizing effect in the plasma, did not improve endurance performance during heavy exercise, and reduced the plasma [glycerol] but did not effect that plasma concentration of free fatty acids, glucose or lactate.

The significance of plasma alkalinization on exercise performance is controversial as studies report both improvements (Bouissou et al. 1988; Costill et al. 1984; Goldfinch et al. 1987; Hewitt and Callaway 1936; Inbar et al. 1983; Jones et al. 1977; Sutton et al. 1981; Wilkes et al. 1984) and no effects (Johnson and Black 1953; Katz et al. 1984; Kindermann et al. 1977; Kowalchuk et al. 1984; McCartney et al. 1983; Parry-Billings and MacLaren 1986; Wijnen et al. 1984) on exercise performance with alkalosis. Early studies used either sodium bicarbonate or fruit juices (high in potassium citrate) and alkaline salts of citric acid to induce a state of alkalosis. Dennig (1931, 1937); Dill (1932) and their coworkers demonstrated that running time to exhaustion was pro-

longed if a state of alkalosis was established before the exercise. They also demonstrated that the ability to neutralize lactic acid and accumulate an O_2 debt was enhanced with alkalosis and impaired during acidosis. Alkali ingestion was shown to improve swimming performance in events ranging from 100 to 440 yd in distance (Hewitt and Callaway 1936) and endurance running and cycling (Dennig 1937). Johnson and Black (1953) used a similar alkalinizing protocol to Dennig (1937) but found no improvement in running time during a series of 1.5 mile cross-country races.

Recently, sodium bicarbonate (approximately 0.2–0.3 $g \cdot kg^{-1}$ body mass) has become the preferred alkalinizing agent. In heavy exercise lasting only 30 s Inbar and coworkers (1983) observed small but significant improvements in mean power output from 8.75 $W \cdot kg^{-1}$ to 8.86 $W \cdot kg^{-1}$ during a Wingate cycling test, whereas McCartney and coworkers (1983), using a similar protocol, found no effect on mean power output or total work performed after bicarbonate ingestion. Parry-Billings and MacLaren (1986) demonstrated that bicarbonate or citrate ingestion produced only a small 1–3% increase in total work during three successive Wingate tests. Bouissou and coworkers (1988) demonstrated that time to exhaustion while cycling at 125% $\dot{V}_{O_{2\,max}}$ improved approximately 20% (75.3 s vs 61.5 s) during alkalosis, however Katz and coworkers (1984) used an identical protocol but found that the time to exhaustion (approximately 100 s) was not affected by alkalosis.

Jones and coworkers (1977) used an exercise protocol similar to that used in the present study and demonstrated that endurance time at 95% $\dot{V}_{O_{2\,max}}$ was increased from 270 s in control ($CaCO_3$) to 440 s in alkalosis ($NaHCO_3$) and decreased to 160 s during acidosis (NH_4Cl). In a similar study these same authors reported endurance times of 274 s (control), 326 s (alkalosis) and 188 s (acidosis) (Sutton et al. 1981). The plasma $[H^+]$ measured immediately prior to the start of exercise in the two studies were approximately 36 $nmol \cdot l^{-1}$ (alkalosis) and 42 $nmol \cdot l^{-1}$ (control) (Jones et al. 1977; Sutton et al. 1981). In the present study, the plasma $[H^+]$ measured immediately before exercise was 36 $nmol \cdot l^{-1}$ (C, alkalosis) and 39 $nmol \cdot l^{-1}$ (P, control) (Fig. 2), similar to the values reported by Jones, Sutton and coworkers (1977, 1981). Thus in spite of comparable acid-base changes prior to exercise in the present study, endurance time was not improved during alkalosis. Although the reasons for this difference are not known, it may partly be explained by the differences between the agents used to induce the alkalosis. Unlike bicarbonate, citrate is an important substrate and cofactor for metabolism in various metabolic pathways: it is a metabolic intermediate in the citric acid cycle, it transports acetyl CoA units from the mitochondrion into the cytosol for fatty acid synthesis, it is a metabolic regulator and inhibits the glycolytic enzyme 6-phosphofructokinase by potentiating the inhibitory effects of ATP (Newsholme and Leech 1983) and it may play a role in excitation-contraction coupling in skeletal muscle by reducing the membrane potential for contraction threshold (Dulhunty 1988). It is impossible to determine if any of the above were important in affecting performance in the present study. The results of the present study suggest that unlike bicarbonate-induced alkalosis (Jones et al. 1977; Sutton et al. 1981), citrate-induced alkalosis failed to improve performance during high intensity cycling requiring near maximal aerobic power.

In the present study citrate was administered as the tri-sodium salt (Na_3 Citrate). Citrate was rapidly absorbed into the bloodstream as demonstrated by the increase in plasma [citrate] after 30 min ingestion (Fig. 3). The increase in plasma [citrate] observed with the placebo reflects the expected change due to the small quantity of citrate ingested in the orange-flavoured drink. Whereas the plasma [citrate] continued to increase throughout the pre-exercise period in C, the [citrate] decreased between 30 and 60 min post-ingestion in P (Fig. 3).

The mechanism responsible for the alkalosis is explained using the physico-chemical principles reviewed by Stewart (1983). Sodium citrate does not exist in molecular form in body fluids but dissociates into individual ions, $sodium^+$ and $citrate^{3-}$. As the citrate anion, but not the sodium cation, is removed from plasma, the plasma [strong ion difference] ([SID] = (sum of the strong cations) − (sum of the strong anions)) increases (Stewart 1983); that is, the ratio of strong cations to strong anions increases and there is an electrical charge imbalance. Electrical neutrality must be maintained and this is met by a fall in $[H^+]$ and an increase in $[HCO_3^-]$ (Stewart 1983). Thus the fall in $[H^+]$ (or increase in $[HCO_3^-]$) induces the state of alkalosis observed in the present study.

It was hypothesized that in addition to the effects of sodium citrate ingestion on acid-base balance, citrate may also inhibit glycolysis in the working muscle. It is assumed that citrate is able

to cross the sarcolemma into the cytosol, although the mechanism by which this occurs is not known. A specific tricarboxylate carrier is required to transport citrate across the mitochondrial membrane (Newsholme and Leech 1983) but we are not aware that such a carrier is present on the cell membrane. It is possible that citrate may cross the membrane at a slow rate by diffusion. Evidence that citrate was removed from the plasma, possibly into skeletal muscle, is given by the fact that an alkalosis developed in the plasma in the citrate condition (see above).

The muscle [lactate] was not measured but the plasma [lactate] measured during exercise and recovery was not different between C and P. The plasma [lactate] represents a balance between efflux from lactate producing tissues (active skeletal muscle) and uptake by lactate consuming tissues (liver, kidney, heart, and active and inactive skeletal muscle). Faster rates of lactate efflux have been observed from the isolated working gastrocnemius of the dog (Hirche et al. 1975) and frog sartorius muscle (Mainwood and Worsley-Brown 1975) when the extracellular pH and [HCO_3^-] were elevated. A higher plasma [lactate] is often observed during exercise with alkalosis compared to control or acidotic conditions (Inbar et al. 1983; Jones et al. 1977; Katz et al. 1984; McCartney et al. 1983; Sutton et al. 1981; Wijnen et al. 1984; Wilkes et al. 1983), while a higher muscle [lactate] has been reported after exercise in alkalosis (Bouissou et al. 1988; Sutton et al. 1981). Thus unlike bicarbonate-induced alkalosis where both lactate production and lactate efflux may be enhanced, the similar plasma [lactate] found during C and P in the present study suggests that citrate-induced alkalosis failed to stimulate these processes (i.e. lactate production and/or efflux) in excess of that observed in control conditions. Although this suggests that citrate may have had an effect on glycolysis and lactate production, additional studies must be performed to determine whether the intramuscular [citrate] and [lactate] were affected by citrate ingestion.

The plasma [glycerol] was lower in alkalosis compared to control but no significant differences were found in the plasma [free fatty acid] between the two conditions (Fig. 5). As the plasma [glycerol] provides an index of the rate of lipolysis occurring in adipose tissue (Newsholme and Leech 1983), the lower plasma [glycerol] found during alkalosis in the present study implies that the rate of lipolysis was lower. These results differ somewhat from those reported by Jones and coworkers (1977) who demonstrated that while the plasma [glycerol] and [free fatty acid] were lower during acidosis, no differences were observed between the alkalosis and control conditions. The differences between the two studies are difficult to explain since citrate has not been shown to have antilipolytic effects in adipose tissue. However, in a recent study Bouissou and coworkers (1988) observed that at exhaustion from heavy exercise the increases in the concentration of adrenaline and noradrenaline were lower during alkalosis than in control conditions. Catecholamines are the major lipolytic hormones in human adipose tissue (Newsholme and Leech 1983). Catecholamines were not measured, however lower plasma catecholamine levels could explain the lower plasma [glycerol] found during alkalosis in the present study.

Plasma [free fatty acid] was not significantly different between C and P; however, the concentration was somewhat lower in C as would be expected given the lower [glycerol]. Of importance in the interpretation of these data is the measurement of respiratory exchange ratio. There was no significant difference in respiratory exchange ratio in C and P suggesting similar metabolic breakdown of carbohydrate and free fatty acids. It is possible that alkalinization did lower plasma catecholamines (Bouissou et al. 1988), and that both lipolysis and fatty acid re-esterification were reduced relative to control conditions (Brooks et al. 1982). Thus lipolysis may not have been stimulated to the same extent following citrate ingestion as occurred during control conditions but plasma fatty acid levels were similar in both conditions.

The plasma [hemoglobin] and hematocrit increased with increasing intensity of exercise but were lower in alkalosis compared to control (Table 2). An increase in both [hemoglobin] and hematocrit occurs as plasma volume is lost from the vascular compartment due to an increase in vascular hydrostatic pressure and/or extravascular osmotic pressure. The slower increase in [hemoglobin] and hematocrit observed in alkalosis may be due to a slower loss of plasma volume associated with the ingestion of more osmotically active molecules in alkalosis; approximately 71 mmol sodium citrate were ingested ($3*71=213$ mmol sodium) compared to 54 mmol sodium chloride ($1*54=54$ mmol sodium). Although sodium citrate ingestion may have slowed the loss of plasma from the vascular compartment, this was not important in prolonging cycling time during heavy exercise.

In summary, the ingestion of sodium citrate

(0.300 $g \cdot kg^{-1}$ body mass) compared to sodium chloride (0.045 $g \cdot kg^{-1}$ body mass) induced a decrease in plasma $[H^+]$ and an increase in the plasma $[HCO_3^-]$ similar to that observed with a bicarbonate-induced alkalosis (Jones et al. 1977; Sutton et al. 1981). However the time to exhaustion at 95% $\dot{V}_{O_{2max}}$ was not prolonged following citrate ingestion. The plasma concentration of lactate, free fatty acids and glucose were not affected by citrate ingestion but the [glycerol] was lower during alkalosis.

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