The effect of sodium citrate intake on anaerobic performance in normoxia and after sudden ascent to a moderate altitude

B. FERICHE FERNÁNDEZ-CASTANYS, M. DELGADO FERNÁNDEZ, J. ÁLVAREZ GARCÍA

Background. The effect of sodium citrate intake on anaerobic performance in normoxia and acute hypoxia was tested in 17 healthy male subjects.

Methods. The subjects underwent a high-intensity exercise protocol in conditions of normoxia (N) and at 2320 m above the sea level (H). Each condition was combined with the intake of a placebo (P) or sodium citrate (C).

Results. The results obtained showed a drop in the maximum HR (p<0.001), due to the effect of the altitude (185±8 vs 176±8 bpm for N and H under PI conditions and 189±9 vs 178±8 bpm for N and H under C conditions). C caused an increase in the RER (p<0.05) and the maximum Lac (p<0.01). The action of this same factor brought about a drop in the maximum VE (p<0.01) (182.60±21.58 vs 177.38±20.29 L·min⁻¹ in N and 185.71±22.98 vs 179.06±22.91 L·min⁻¹ in H). The interaction of both C and H affected the maximum concentration of lactate obtained (p<0.01), which fell as regards to the corresponding action of both factors separately (14.33±2.94 vs 17.8±2.74 mMol·L⁻¹ with PI and C in N and 15.29±2.15 vs 15.54±2.59 mMol·L⁻¹ in H). There were no significant differences in the length of work time in each of the conditions established.

Conclusions. It would, therefore, seem that in the conditions described, the intake of sodium citrate does not cause appreciable changes in anaerobic performance.

Key words: Alkalosis - Citrates - Anoxia.

Sodium citrate is an alkaliniser used to improve performance capability during high-intensity exercise.¹⁻³ The rise in plasma bicarbonate levels after administering alkaline agents can aid the efflux of hydrogen ions and of lactate from the muscle into the blood.⁴⁻⁸ This mechanism is suggested to allow an improvement in performance during exercise where acidosis may constitute a limiting factor.³,⁹ However, the results obtained in studies in which sodium citrate is used are somewhat contradictory due, in part, to the variability of the dosage, protocols and populations used.¹⁰

During an ascent in altitude, the barometric pressure falls together with the density of the surrounding air. The associated reduction in the availability of oxygen does not necessarily have to compromise the capacity of anaerobic performance, at least during acute rises in altitude.¹¹⁻¹⁴ However, it is possible that, although it is not well defined, the aerobic contribution during high intensity exercise may constitute from 20-40% in 30 sec long exercises up to 65% in those of 2 min.¹⁵,¹⁶ Changes in performance have not been recorded in a 30 sec Wingate test in conditions of sudden hypoxia, although with increases in duration.

Address reprint requests to: B. Feriche, Departamento de Educación Física y Deportiva, Facultad de Ciencias de la Actividad Física y el Deporte, Universidad de Granada, Carrera de Alifarca sn, 18011, Granada, Spain. E-mail: mbelen@ugr.es
up to 45 sec the results are somewhat contradictory. At altitude, the aerobic contributions to the exercise could be altered by the reduction in availability of oxygen in the atmosphere. However, an increase in the anaerobic contribution as a compensatory mechanism is generally reported. The major implication of the anaerobic pathway could increase the concentration of blood lactate for the same work load which would decrease the performance capability in this type of exercise.

This study was designed to determine whether the intake of sodium citrate prior to initiating exercise might improve the capacity to execute a high intensity effort in conditions of normoxia and during acute exposure to moderate altitude (2320 m), where acidosis may be an earlier limiting factor due to the implication of the anaerobic metabolism during the exercise.

Materials and methods

A group of 17 healthy, male physical education students (of 22±1 years of age, 73.03±4.25 kg body mass, 176.1±4.25 cm tall and 4.04±0.67 l·min⁻¹ maximum oxygen consumption) participated voluntarily in our study after giving their consent in writing. This study has been approved by the Research Commission of the Physical Education Department of Granada University.

All subjects carried out a maximal incremental test and a total of four high intensity exercise trials at a constant workload on an electrically braked cycle ergometer (Ergoline 900). The work intensity employed during the high-intensity tests corresponded to the maximum work load reached during a prior maximal incremental test (25 watts a minute at pedal cadence between 60 and 90 rpm) carried out under conditions of normoxia and without an alkaliser. This intensity corresponded to about 112% of VO₂max and resulted in exhaustion between 2 and 4 min. Before starting the trial, the subjects underwent a 5-min warm-up which consisted of pedalling for 1 min at 100 watts (W), 1 min at 120 W, 1 min at 140 W, 1 min at 120 W and 1 min at 100 W, as described in other similar studies. Subsequently, pedalling began at 50 W for 1 min so that going on to the full load (an average of 315±43 W) of the trial would be made easier by acquiring prior pedalling inertia. During the tests the subjects had to work at the maximum pedal cadence that could be obtained and must be maintained in all the trial test. At the moment they could not maintain a minimum pedal cadence of 60 rpm the test was stopped.

The order of test in conditions of normoxia (N) and hypoxia (H) was randomized and were conducted in the biology laboratory of the Physical Activity and Sports Faculty in Granada (690 m above sea level and 717 mmHg of barometric pressure) and the Medical Services Exercise Test Laboratory of the High Performance Centre in Sierra Nevada (2320 m above sea level and 560 mmHg of barometric pressure). At altitude, the tests were conducted during the first 3 or 4 hours after arrival. In all cases, the time interval between tests was a minimum of 48 hrs and a maximum of 72 hrs.

In addition, each altitude condition was combined with either the intake of a placebo (P) or an experimental drink with sodium citrate (C), using a random double blind study design.

The placebo was sodium chloride (NaCl) using a dose of 0.045 g·kg⁻¹ of body mass (bm). The experimental drink contained 0.4 g·kg⁻¹ of sodium citrate (Na₃C₆H₅O₇·2H₂O). Both substances were diluted in 500 ml of distilled water. Saccharine and lemon essence were added in order to mask their flavours and make them indistinguishable as regards taste and smell. Drinks were administered 120 min before exercise commenced to obtain the peak value of pH and base excess as has been described in other studies.

Before beginning the trial, the subjects were connected to an electrocardiograph with monitor (Helige) to record heart rate (HR) and to a gas analyser (cpx of Medical Graphics or Oxycon Sigma the Jaeger for N and H, respectively) to measure, breath by breath, oxygen consumption (VO₂), ventilation (VE) and the respiratory exchange ratio (RER). The data were calculated as average of 10 sec during work. Furthermore, both before and after the test, as well as during min 3 and 5 of passive recovery, capillary blood samples were taken from fingers to determine the lactate concentration in a spectrophotometer (Shimadzu CL 700), using the Boehringer method. The highest blood lactate concentration obtained between the end of the test and the passive recovery was considered as the peak or max value.

The maximum values obtained are expressed as mean and standard deviation (SD). The main effect of a sudden rise to moderate altitude, of the intake of
sodium citrate or of the interaction of both factors at the same time on these parameters was measured using multifactorial analysis of the variance (MANOVA) for repeated measurements. The Newman Keuls post-
hoc was used as retest in those analyses that showed statistical significance. The p level accepted for all comparisons was less than 0.05.

### Results

Mean values, main effect of altitude and sodium citrate and the interaction effect between them both for time to exhaustion (TD), minute VE (l·min⁻¹), and HR (bpm) are displayed in Table I. Although time to exhaustion tended to be greatest under NC conditions no significant differences were found in any conditions (p>0.05). However, VE was significantly reduced at the end of exercise in both altitude conditions (N and H) after citrate intake (p<0.05). Exercise at altitude was completed with a significantly lower HR (p<0.001) at the end of exercise compared with normoxia.

In Table I the peak mean values reached in the VO₂ (l·min⁻¹) and RER in the situations established are also set out. Sodium citrate condition showed an increased RER value at the end of the exercise (p<0.05). The maximum lactate concentration (mMol·l⁻¹) reached under the four experimental conditions are shown in Table I too. The intake of the alkalizer increases the maximum lactate concentration (Lac) reached at the end of the exercise (p<0.01). The interaction analysis obtained when both factors are present (altitude and sodium citrate) only shows an effect on the recorded concentration of maximum Lac. The maximal lactate concentration showed a reduced value under HC condition as reflected when both were analysed separately (p<0.001). The remainder of the variables analysed

<table>
<thead>
<tr>
<th>Parameters</th>
<th>N-PI</th>
<th>H-PI</th>
<th>N-C</th>
<th>H-C</th>
<th>Factor effect (posthoc p value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TD (sec)</td>
<td>167.8±29.42</td>
<td>169.94±33.57</td>
<td>176±32.78</td>
<td>161.82±33.95</td>
<td>Pa = 0.0997; Pb = 0.0980; Pc &gt;0.05</td>
</tr>
<tr>
<td>VE (l·min⁻¹)</td>
<td>182.59±21.58</td>
<td>185.70±22.97</td>
<td>177.38±20.29</td>
<td>179.26±22.90</td>
<td>Pa = 0.4958; Pb = 0.0180; Pc &gt;0.05 ***</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>185±8</td>
<td>176±8</td>
<td>185±9</td>
<td>178±8</td>
<td>Pa = 0.0002; Pb = 0.3899; Pc&lt;0.05</td>
</tr>
<tr>
<td>VO₂ (l·min⁻¹)</td>
<td>3.94±0.52</td>
<td>3.85±0.53</td>
<td>3.88±0.48</td>
<td>3.92±0.55</td>
<td>Pa = 0.6091; Pb = 0.8288; Pc&gt;0.05 CCC</td>
</tr>
<tr>
<td>VCO₂ (l·min⁻¹)</td>
<td>5.78±0.67</td>
<td>5.79±0.81</td>
<td>5.97±0.73</td>
<td>6.08±0.8</td>
<td>Pa = 0.6026; Pb = 0.0007; Pc&lt;0.05 CCC</td>
</tr>
<tr>
<td>RER</td>
<td>1.47±0.11</td>
<td>1.51±0.13</td>
<td>1.54±0.12</td>
<td>1.55±0.07</td>
<td>Pa = 0.4695; Pb = 0.0119; Pc&gt;0.05 CCC ###</td>
</tr>
<tr>
<td>Lac (mMol·l⁻¹)</td>
<td>14.33±2.94</td>
<td>15.29±2.15</td>
<td>17.80±2.74</td>
<td>15.54±2.59</td>
<td>Pa = 0.2389; Pb = 0.0023; Pc&lt;0.001</td>
</tr>
</tbody>
</table>

Posthoc p value in bold: pa = altitude effect; pb = citrate effect; pc = interaction effect.
show no effect caused by the interaction of altitude with sodium citrate at the intensities of work described.

**Discussion and conclusions**

The results of this study show no independent or combined effect of altitude or sodium citrate ingestion on the ability to complete a high intensity exercise test. This suggests that probably the level of acidosis reached may not be the only factor causing fatigue during high intensity exercise.

It has been reported that respiratory alkalosis induced by a rise in altitude may prove beneficial in high-intensity activities by improving the conditions in which the muscle works. In the present study, pH was not directly measured. However, there is evidence in the literature that citrate would affect pH. We obtained previously in our laboratory that both the intake of sodium citrate and, above all, the rise to altitude increased the alkalinity of blood (p<0.001) before, during and after a maximum incremental exercise. However, the present results show no improvement in the exhaustion or changes in physiological (VO₂ and VE) or metabolic (Lac and RER) variables despite significant changes in alkalinity found in other studies and supporting previous data examining these variables at altitude (Fig. 1).

No effect of altitude on the maximum concentration of lactate reached during the tests has been observed. At altitude, a significant increase in lactate concentration after short-term high-intensity exercises has not always been seen. Some studies suggest that the level of circulating catecholamines reached during physical exercise could be an indicator of the speed at which glycogen is used up in the muscle and of the formation of lactate. However, the levels of adrenaline and noradrenaline recorded before or after carrying out a supramaximum effort at altitude are not always higher but can even be reduced. In our study, any maintenance or decline of the sympathetic stimulus after acute exposure to altitude may explain the absence of significant changes in VE and Lac that was observed when the altitude factor is analysed. Furthermore, the maintenance of a similar VO₂ between both conditions of N and H during the execution of this protocol indicates that hypoxia does not significantly affect aerobic participation during high-intensity exercise, as has been shown in other studies.

The decline in peak HR that we observed at altitude has been previously reported. Again, it is possible that the previously mentioned reduction in the peak of catecholamines, together with a possible drop in the response of the β-receptors, are responsible for the reduction in HR while carrying out high-intensity work in conditions of sudden hypoxia.

Many studies show that sodium citrate is one of the best ergogenic aids in 1 to 7 min-long anaerobic-type exercising. However, our results, as with those of other researchers, do not register this ergogenic effect in a high-intensity stable-load test. We should point out that in conditions of normoxia, although not a significant result, our subjects were capable of maintaining the work load for around 9 sec longer after consuming sodium citrate than after taking the placebo, which would constitute a decisive time difference in any competition (p=0.09). We are not exactly sure why some individuals benefit and others do not from the intake of this alkaliser in conditions of N as opposed to those of H. Six subjects improved their performance at N (Fig. 2), four at H (Fig. 3) and only two did it in both conditions when PI and C were compared. Linossier et al., who also recorded great
individual variability in the response to this alkaliizer (1-40%), attributed it to the possible difference in the level of physical condition of the subjects analysed in their sample.

Some studies establish a relationship between the increase in the net production of lactic acid with the improvement in energy production capacity from anaerobic glycolysis and the duration of the effort.20 This relationship could justify the absence of an improvement in performance in those studies that use exercise protocols of very short duration although, as occurred in our study, the majority register a significant increase in the concentration of maximum lactate during the series in which the alkaliizer was taken.3 20, 39-42 The citrate is a phosphofructokinase (PFK) inhibitor. It is possible that the effect of sodium citrate has on the lactate kinetics may be affected by the fact that the latter blocks the (PFK). The latter block of PFK by citrate ingestion could restrict the use of the glycolytic mechanisms but allows a greater formation of lactic acid in those exercises whose duration (more than about 180 sec) is long enough for this limitation to disappear,1 due suppression of an inhibitor.

Our results also show an effect of sodium citrate on the maximal VE. The majority of studies reviewed do not show an alteration in the cardiorespiratory parameters due to the effect of this factor.24, 43 However, other studies,1, 4, 44 also registered attenuated ventilatory dynamics after the intake of the alkaliizer. It is possible that a central control prevails over the peripheral regulatory mechanisms controlling VE, keeping it at a reduced level when faced with the increase in the state of the blood pH due to the alkaliization with sodium citrate. It is also possible that the drop in the VE is due to a reduction in the sympathetic activity, a phenomenon referred to earlier.

The observation of a higher maximum respiratory ratio in conditions of alkaliization responds to the increase in blood pH values and is in accordance with the lactic acid response obtained in this study. The increase (not shown in this work), observed in the production of carbon dioxide (CO₂) as a consequence of a greater participation of the blood bicarbonate in the buffering mechanisms, also observed in other research work,43 may be responsible for the differences observed in the behaviour of the RER during the tests. However, the fact that at high work intensities, CO₂ dynamics is conditioned by the different buffer metabolic reactions speeds on the lactate by the blood bicarbonate may cause alterations in CO₂ and, therefore, in the ventilatory response, as observed by our group and which responds to still-unknown mechanisms.3 45

Contrary to our expectations, we observed in our study that there is a significant reduction in the maximum accumulation of lactate during high-intensity exercise when both factors, citrate and altitude, are present. Due to the effect that both factors may have on the acid-base state of the blood, it was expected that when both occurred at the same time, they would induce a greater alkaliization and, therefore, a greater increase in the maximum concentration of lactic acid. In fact, some studies show a significant increase in the production of lactate at altitude with work at 90% of the maximum VO₂ when the rise is combined with
the intake of sodium bicarbonate. However, Hausswirth et al. did not observe changes in the pH and lactate dynamics in conditions of hypoxia and induced alkalosis as opposed to those of normoxia and placebo, respectively. In this same study, though, the intake of sodium citrate in conditions of normoxia did improve the capacity of maintaining an isometric contraction in time. It seems difficult to combine all these results with those of this study due to the differences in populations, designs and protocols in the various pieces of research work. It is possible that at such high work intensities as those in our research (about 112% of the $\text{VO}_{2\text{max}}$), altitude interferes in the production mechanism of lactic acid due to the reduction in the sympathetic response. On the other hand, the production of lactate can also be reduced due to the effect that the sodium citrate has on the PFK. However, although both factors (altitude and citrate), particularly sodium citrate, improve the exit of the lactate from the muscle into the blood stream, it is possible, corroborating the results obtained in the preliminary study, that when both factors appear at the same time, this diffusion mechanism becomes saturated. In this case, it is possible that the subjects cannot obtain enough energy from a deteriorated anaerobic system combined with a limited aerobic contribution at altitude by reduction of oxygen availability.

Therefore, if the decrease in the extracellular pH were the limiting factor on performance, fatigue should appear at the same level of blood lactate in the different conditions established. However, the absence of significant differences in performance among the tests shows that, at least at high intensities of exercising, performance capacity is probably not exclusively conditioned by the alkaline reserve level nor by the state in extracellular pH reached. Unfortunately, we did not measure blood gases in this study and this conclusion needs to be clarified in future studies.

Acknowledgements.—The author thanks Dr Tim Cable for his collaboration at the Centre for Sport and Exercise Sciences (Liverpool John Moores University), the CAR of Sierra Nevada (CSFD), the Department of Physical Education and the Sciences Faculty of the Physical Activity and Sport of Granada (Spain) in this study.

References


27. Feriche B. Estudio de la eficacia del citrato sódico sobre la mejora del rendimiento y tolerancia a las cargas de trabajo en condiciones de normoxia e hipoxia subita a una altitud moderada. Doctoral thesis. Universidad de Granada, Spain, 1998.


