Effect of sodium citrate ingestion on oxygen debt & exercise endurance during supramaximal exercise

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Background & objectives: Administration of alkalinizing agents has been shown to improve performance in high intensity exercise. The present investigation was undertaken to determine whether sodium citrate ingestion enhances supramaximal endurance performance on a cycle ergometer and to assess its effect on oxygen debt incurred for the same supramaximal energy output.

Methods: Ten untrained, healthy, males performed acute intense bicycle ergometry exercise in two sessions viz., without (control) and with sodium citrate ingestion (experimental) in a dose of 0.5 g/kg body weight. Pre-exercise O₂ consumption, pulse rate (PR) and respiratory rate (RR)/minute were recorded before both sessions. Exhaustion time (ET) was noted and work done calculated in the control session. Post-exercise PR and RR/min were also recorded in both sessions of study. Venous blood samples were drawn post-exercise and analysed for pH and lactate. O₂ debt incurred was calculated in both sessions.

Results: Work output in supramaximal exercise averaged 69.40±15.31 Watts in the control session. None of the subjects in the experimental session complained of fatigue even when the exhaustion time noted in the control session was reached. Post-exercise tachycardia and tachypnoea were both significantly less (P<0.05 and P<0.001 respectively) in subjects after the experimental session as compared to the control. O₂ debt incurred and lowering of blood pH were also significantly less (P<0.001) in the post-citrate phase. However, serum lactate increased significantly (P<0.05) in subjects after the experimental session.

Interpretation & conclusion: Ingestion of sodium citrate prior to supramaximal exercise resulted in a reduction in post exercise O₂ debt incurred. Blood pH was almost normal despite a higher serum lactate concentration. This probably led to an enhancement in exercise performance.

Key words Alkalosis - endurance - exercise - lactate - oxygen debt - sodium citrate - work output

Anaerobic glycolysis serves as the primary energy source during supramaximal exercise1,2. During the process of anaerobic glycolysis, lactate production is accompanied by an increase in muscle3 and blood4 hydrogen ion concentration (H⁺). The decrease in pH causes impaired release of calcium from the sarcoplasmic reticulum of the muscle and Ca²⁺ ion binding leading to impairment of contractile mechanism efficiency5 and this has frequently been implicated as the major cause of fatigue.

It has been postulated6 that prevention of this increase in blood hydrogen ion concentration may have an ergogenic potential. An artificially induced alkalosis...
prior to exercise increases the rate of efflux of lactate and its dissociated hydrogen ions into the blood during exercise, reduces intracellular acidosis and enhances the time to fatigue. However, conflicting results regarding exercise performance and endurance time following buffer ingestion have been reported by various authors.

Several studies have shown that sodium bicarbonate (NaHCO$_3$) or sodium citrate can be used to this effect. The high dosages of NaHCO$_3$ (0.3 - 0.5 g/kg body weight) required to enhance exercise performance are associated with a major drawback of causing considerable gastrointestinal (GI) disturbance. A dose of 0.5 g/kg body weight of sodium citrate has been implicated as being the most appropriate for greatest improvements during anaerobic cycle ergometer performance. The advantage of this treatment over the use of NaHCO$_3$ is that it is well tolerated and reportedly does not cause any GI disturbance.

It is well established that O$_2$ consumption remains elevated for some period after exercise, a phenomenon that has been termed as excess post exercise oxygen consumption (EPOC). The amount of extra O$_2$ consumed is proportionate to the extent to which the energy demands during exertion exceeded the capacity for aerobic synthesis of energy stores, i.e., the extent to which an O$_2$ debt was incurred. To the best of our knowledge, no previous study has compared the effect of sodium citrate ingestion on O$_2$ debt incurred at the end of a single bout of supramaximal exercise performed under conditions similar to the present study.

The present study was undertaken to ascertain whether sodium citrate ingestion enhances supramaximal endurance performance on a cycle ergometer and to observe its effect on O$_2$ debt incurred for the same supramaximal energy output.

**Material & Methods**

**Subjects:** The study was done at the Department of Physiology GSVM Medical College, Kanpur. Ten, healthy, untrained males participated in this study. The subjects were randomly selected from the batch of first year MBBS students. All subjects participated in regular physical activity although not in events of a specifically anaerobic type. All individuals provided written informed consent before the start of the experiment. The study was approved by the ethics committee of the institute. The physical characteristics of the subjects were (mean ± SD): age, 19.30±0.95 yr; height, 1.69±0.03 m; weight, 60.60±7.80 kg; body surface area (BSA), 1.70±0.11 m$^2$.

**Study protocol:** Each of the subjects underwent two test sessions, one without sodium citrate ingestion (control) and one with sodium citrate ingestion (experimental). On the day of testing, the subjects reported to the laboratory 1 h post prandial (breakfast). Pre-exercise pulse rate (PR), respiratory rate (RR) and O$_2$ consumption/min were recorded. PR was recorded using pulse recording probe fitted to the left index finger and connecting it to the physiograph (Biodevices, India). RR recording was made by attaching a nasal probe in the left nostril and connecting it to a physiograph to record the inspiratory and expiratory cycles (Biodevices, India). Oxygen consumption was measured on a double benedict Roth spirometer (Inco., India). The subjects then carried out acute intense exercise on a friction type bicycle ergometer (Inco., India) at a fixed frictional force of 2 kg. Exercise tests comprised pedalling at a speed which they could manage. The time to exhaustion and total number of pedal revolutions were noted and the exercise terminated. Pulse and respiratory rates and O$_2$ consumption were recorded and 2 ml of venous blood was collected from the median cubital vein. The subjects were then allowed to rest for 2 h following which they ingested trisodium citrate in a dose of 0.5 g/kg body wt given as 200 ml of low energy, water-based flavoured drink (to mask the taste). At 2 h post ingestion the pedal revolution rate was fixed with the help of a tachometer and the exercise protocol was repeated till the exhaustion time was reached. PR, RR and O$_2$ consumption were recorded and blood collected on completion of exercise.

**Blood analysis:** All samples were analysed for blood pH and serum lactate. Blood pH was determined using a combined electrode type of pH meter (model CK-61, Elico, India). Serum lactate was determined enzymatically by colorimetric method (Spectrocolorimeter-103, Systronics, India) at room temperature using a cuvette with 1 cm light path at 550 nm.
**Calculations : work done:** The individual work output in the subjects was calculated by using the following formula\(^{17}\):

\[
\text{Work done (Watts)} = \frac{F(g) \times 2 \pi r n \text{ (cm)} \times 980}{10^7 \times t \text{ (sec)}}
\]

Where \(F\) = Frictional force

\(2 \pi r\) = wheel circumference of ergometer

\(n\) = number of revolution/minute

\(t\) = exhaustion time

\(O_2\) debt: The total post exercise \(O_2\) consumption was spirometrically recorded until it fell down to the pre exercise \(O_2\) consumption recorded in the same subject (A). The pre-exercise \(O_2\) consumption/min was multiplied with the duration in minutes to obtain the value for pre-exercise \(O_2\) consumption in that duration in which the total post-exercise \(O_2\) consumption was recorded (B). The \(O_2\) debt was then calculated as Oxygen Debt = A-B.

**Statistical analysis:** The pre and post-exercise data in both session of the study were compared by applying Student’s paired ‘t’ test and \(P<0.05\) being considered as significant.

**Results**

**Pre-exercise parameters:** Pre-exercise pulse and respiratory rates as well as \(O_2\) consumption did not show significant difference in the control and experimental sessions (Table).

**Exercise performance:** The subjects performed cycle ergometry for a mean duration of 8.70±2.87 min (exhaustion time) at a mean pedal revolution rate of 124.90±27.68 rev/min in control session. The work output in this session was 69.40±15.31 Watts which presumably depended on their individual endurance. None of the subjects complained of a feeling of exhaustion during performance of the pre-fixed limit of supramaximal exercise after citrate ingestion as compared to exercise without citrate ingestion.

**Post-exercise blood and cardio-respiratory parameters:** The increase in post exercise PR and RR following citrate ingestion was significantly lower when compared to that in the control session. In the experimental group a statistically significant decline (\(P<0.001\)) in \(O_2\) debt incurred was seen as compared to the control group. The decline in blood \(pH\) was also statistically less (\(P<0.001\)) in the experimental group. However, the post exercise serum lactate levels were significantly (\(P<0.05\)) more in subjects who had ingested citrate (Table).

**Discussion**

Sodium citrate has been used as an ergogenic aid to enhance exercise performance\(^{2,7,14}\). The exercise protocol of the present study is different from that of earlier studies\(^{7,9-12,14}\) where either graded exercise was used or the exhaustion time noted before and after citrate administration. The experimental design used in our study ensured that each subject carried out identical exercise under both trial session. This was achieved by keeping the exercise parameters \(viz.,\) work output and pedal revolution rate fixed in both sessions of the study.

It was observed that none of the subjects complained of a feeling of exhaustion at the end of the experimental session when the exhaustion time noted in the control session for that subject was reached. This implies that sodium citrate ingestion improves the endurance power of the subjects and this is in close agreement to another studies\(^{18,19}\) using sodium bicarbonate. Inadequate buffer dosages, lack of established alkalosis prior to exercise

**Table. Changes in blood and cardiorespiratory parameters of subjects in control (without sodium citrate) and experimental (with sodium citrate) sessions**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Sessions</th>
<th>Control</th>
<th>Experimental</th>
</tr>
</thead>
<tbody>
<tr>
<td>PR/min</td>
<td>Pre-exercise</td>
<td>105.30±11.57</td>
<td>111.40±14.24</td>
</tr>
<tr>
<td></td>
<td>Post-exercise</td>
<td>184.20±25.91</td>
<td>159.80±22.26 #</td>
</tr>
<tr>
<td>RR/min</td>
<td>Pre-exercise</td>
<td>20.90±3.03</td>
<td>21.70±4.60</td>
</tr>
<tr>
<td></td>
<td>Post-exercise</td>
<td>43.80±5.79</td>
<td>29.30±4.56 *</td>
</tr>
<tr>
<td>(O_2) consumption ml/min</td>
<td>Pre-exercise</td>
<td>279±49.09</td>
<td>279±49.09</td>
</tr>
<tr>
<td>(O_2) debt (l)</td>
<td>Post-exercise</td>
<td>7.77±0.96</td>
<td>4.65±0.66 *</td>
</tr>
<tr>
<td>Blood (pH)</td>
<td>Post-exercise</td>
<td>7.19±0.04</td>
<td>7.36±0.04 *</td>
</tr>
<tr>
<td>Serum lactate (mmol/l)</td>
<td>Post-exercise</td>
<td>12.73±2.03</td>
<td>14.49±1.83 #</td>
</tr>
</tbody>
</table>

\(n = 10\), values are mean ± SD; \(P<0.05; *<0.001\) compared to control
or use of non anaerobic low intensity exercise may have been the critical factors in studies which showed no improvements in performance\textsuperscript{9,12,20}. Following buffer ingestion the exercise should begin approximately after 100-120 minute to obtain positive results\textsuperscript{20}. It is possible that in these studies the absorption time between ingestion and initiation of exercise was not adequate to induce sufficient alkalosis.

Very high intensity, short duration exercise utilises stored muscle creatine phosphate as the primary energy source\textsuperscript{21} and hence is unaffected by alkalosis. Supramaximal exercise performances of 45 sec to 15 min duration would show improvements following induced alkalosis\textsuperscript{22}. In the present study, exhaustion time in the control session ranged from 3.5-14 min. This could explain the improvement seen in exercise performance in subjects in the experimental session.

The supramaximal exercise produced sufficient degree of anaerobism as suggested by high values of O\textsubscript{2} debt in subjects in the control session. The anaerobic changes also produced remarkable reflexogenic acceleration of post exercise PR and RR in this session. Citrate intake mitigated the anaerobic status of the subjects presumably because of more efficient buffering as well as respiratory compensation as suggested by significant reduction of oxygen debt and post exercise PR and RR in subjects in the experimental session.

Blood pH remained very close to normal in subjects in the experimental session compared to the control session suggesting that sodium citrate ingestion had produced significant plasma alkalinisation prior to exercising and hence was able to combat the fall in blood pH and also increase the time to onset of fatigue. The higher amounts of lactate seen in the experimental session are similar to those seen by others using NaHCO\textsubscript{3} as a buffer\textsuperscript{23}. The transport of lactate across the cell membrane has been shown to occur predominantly through the pH sensitive monocarboxylate transporter\textsuperscript{24}. Maintaining a higher pH during exercise may have allowed this transport mechanism to facilitate a greater lactate flux of the contracting tissue. Since citrate is metabolised at several points with the release of HCO\textsubscript{3}\textsuperscript{-}, it may result in a slower release of HCO\textsubscript{3}\textsuperscript{-} into the blood, hence elevating blood HCO\textsubscript{3}\textsuperscript{-} and allowing greater anaerobic performance to be undertaken. However, the exact mechanism of action of sodium citrate still needs to be elucidated.

The present study shows that sodium citrate acts as a performance enhancer in that the subjects were able to maintain the same work output for the same duration as in the control session without experiencing fatigue or exhaustion. The metabolic changes and reduction in post-exercise O\textsubscript{2} debt also suggest the usefulness of sodium citrate in bringing about improvement in exercise endurance during supramaximal exercise.

References


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