Effects of NaHCO₃ loading on acid-base balance, lactate concentration, and performance in racing greyhounds

LYLE D. KESL AND RICHARD L. ENGEN
Department of Physiology and Pharmacology, College of Veterinary Medicine, Iowa State University, Ames, Iowa 50011

Kesl, Lyle D., and Richard L. Engen. Effects of NaHCO₃ loading on acid-base balance, lactate concentration, and performance in racing greyhounds. J. Appl. Physiol. 85(3): 1037–1043, 1998.—This investigation examined the effects of NaHCO₃ loading on lactate concentration ([La]), acid-base balance, and performance for a 603.5-m sprint task. Ten greyhounds completed a NaHCO₃ (300 mg/kg body weight) and control trial in a crossover design. Results are expressed as means ± SE. Presprint differences (P < 0.05) were found for NaHCO₃ vs. control, respectively, for blood pH (7.47 ± 0.01 vs. 7.42 ± 0.01), HCO₃⁻ (28.4 ± 0.4 vs. 23.5 ± 0.3 meq/l), and base excess (5.0 ± 0.3 vs. 0.2 ± 0.3 meq/l). Peak blood [La] increased (P < 0.05) in NaHCO₃ vs. control (20.4 ± 1.6 vs. 16.9 ± 1.3 mM, respectively). Relative to control, NaHCO₃ produced a greater (P < 0.05) reduction in blood base excess (−18.5 ± 1.4 vs. −14.1 ± 0.8 meq/l) and HCO₃⁻ (−17.4 ± 1.2 vs. −12.8 ± 0.7 meq/l) from presprint to postexercise. Postexercise peak muscle H⁺ concentration ([H⁺]) was higher (P < 0.05) in NaHCO₃ vs. control (158.8 ± 8.8 vs. 137.0 ± 5.3 mM, respectively). Muscle [H⁺] recovery half-time (7.2 ± 1.6 vs. 11.3 ± 1.6 min) and time to predose values (22.2 ± 2.4 vs. 32.9 ± 4.0 min) were reduced (P < 0.05) in NaHCO₃ vs. control, respectively. No differences were found in blood [La] recovery curves or performance times. NaHCO₃ increased postexercise blood [La] but did not reduce the muscle or blood acid-base disturbance associated with a 603.5-m sprint or significantly affect performance.

MATERIALS AND METHODS

Animals. All protocols and procedures used in this investigation were reviewed and approved by the Iowa State University Committee on Animal Care. Four weeks before the study began, all animals were placed on a standard commercial dry-food diet (Science Diet Maintenance; Hills Division, Colgate Palmolive, Topeka, KA) and housed in similar inside runs to minimize the influence of previous diet, exercise patterns, or trainer handling. Ten adult greyhounds (4 males, 6 females; mean age, 31 ± 2 mo), all with previous track experience, completed a 603.5-m sprint (a standard industry race distance) after either a control or a NaHCO₃ solution. Animals were randomly assigned in a crossover design, with 7–10 days between trials. Trials were conducted at a consistent time of day and with animals that had fasted overnight.

NaHCO₃-loading regimen. In a preliminary pilot study of five animals that received a dose of NaHCO₃ (300 mg/kg) in a single gastric bolus, arterial blood [HCO₃⁻], pH, and base excess (BE) were elevated (P < 0.05) above predose (PD) levels that began at 30 min postdose and persisted throughout a 4-h sampling period. No overt gastrointestinal or muscular disturbances were noted during preliminary dosing trials.

In the present investigation, the NaHCO₃ solution consisted of a 300 mg/kg body weight dose of NaHCO₃ that was dissolved in 200 ml of warm water and delivered via a gastric tube in a single bolus. The control solution consisted of 200 ml of warm water. Administration of solutions occurred 70 min...
before the sprint trials. Animals sprinted over a 603.5-m distance after a mechanical lure on a 300-m oval track.

Blood and muscle sampling. Immediately before dosing, a 5-ml PD arterial blood sample was obtained from the left femoral artery. After a local anesthetic (2% lidocaine, Elkins-Sinn, Cherry Hill, NJ) was injected under the skin, a skin incision was made over the midsection of the right biceps femoris muscle. A PD sample of muscle tissue (75–100 mg) was surgically removed, immediately frozen (within 2–3 s of excision), and stored in liquid nitrogen until it was analyzed. The incision site was sutured, and the appropriate trial solution was administered. An incision was made over the midsection of the left biceps femoris for postexercise muscle biopsies and was sutured to facilitate rapid postexercise sampling. Animals remained in a pen until 10 min before the scheduled sprint time (60 min postdose).

At 10 min before the scheduled sprint time, a 5-ml pre-sprint (PS) arterial blood sample and muscle biopsy were obtained from the left femoral artery and right biceps femoris muscle, respectively. The incision site was sutured, and the animal was exercised at 70 min postdose. The 10-min difference between PS sampling and the sprint trial was necessary to facilitate collection of PS samples and to minimize bleeding at the arterial puncture site.

Animals sprinted over a 603.5-m distance and were caught within 10–15 s for postexercise sampling. Postexercise muscle biopsies were obtained from the left biceps femoris at 30 s and at 5, 10, 20, and 30 min postspurt. Arterial blood samples (5 ml) were obtained at 2, 5, 10, 15, 20, and 30 min postspurt via a 20-gauge, 2-in. radiopaque Teflon catheter (Angiocath; Deseret Medical, Becton Dickinson, Sandy, UT) placed in the right femoral artery. The animal remained in lateral recumbency throughout the recovery sampling period, after which the catheter was removed and the incision site was sutured.

Blood-gas and blood-lactate analysis. Blood samples (3 ml) were collected in heparinized glass syringes, capped, and placed on ice until analysis of arterial PO2 (PaO2), arterial PCO2 (PaCO2), and pH. All blood-gas measurements were determined within 60 min of collection with the use of a model 813 pH/blood-gas analyzer (Instrumentation Laboratories, Lexington, MA).

Blood-gas analysis was determined in an autocalibration mode, in which the original calibration settings were automatically reset between each sample. Blood [HCO3\(^{-}\)] and BE were calculated from the PaCO2 and pH measurements. One milliliter of whole blood was deproteinized in 2 ml of cold 8% perchloric acid and centrifuged (4,000 g for 10 min). The supernatant was stored at −5°C until determination of [La] by spectrophotometric analysis (19) (Spectronic 70; Analytical Systems Division, Bausch and Lomb), at the conclusion of the study. Hemoglobin concentration [Hb] was determined by the cyanmethemoglobin method (9), and packed cell volume (PCV) was determined by the microhematocrit method.

Muscle pH and La determination. All visible blood and connective tissue were removed, and muscle samples were weighed on a Mettler AE 163 microbalance (Mettler Instrument, Highstown, NJ) before analysis. During processing, samples remained frozen. Intracellular muscle pH was determined by the homogenate technique as described by Costill et al. (3). The pH of the homogenate was measured at 38°C with a Radiometer BMS 3MK2 Blood Micro System (Radiometer, Copenhagen, Denmark) coupled to a PHM-73 pH/blood-gas monitor that was calibrated every fifth sample by using a two-point (6.840 and 7.383) calibration procedure. Muscle [La] was determined by fluorometric enzymatic analysis (Ratio 2 Flurometer, Farrand Optical, Valhalla, NY) and expressed as millimoles per kilogram wet weight (19). All assay samples and standards were analyzed in duplicate. All samples from a specific subject were analyzed within the same assay batch at the conclusion of the study to decrease interassay variance.

Statistical analysis. Treatment effects were tested by using a two-factor ANOVA (treatment × sample interval) with repeated measures on sampling intervals. The effects of NaHCO3 ingestion were assessed by analyzing the PD and PS samples. Exercise effects were assessed by analysis of the PS and the immediate postexercise sampling interval. Recovery was assessed by analysis of the recovery time intervals. A Student-Newman-Keuls multiple-range test procedure was used to identify significant mean differences.

Postexercise [H\(^{+}\)] and [La] were characterized in both muscle and blood by nonlinear, least squares regression analysis to the function [H\(^{+}\)] or [La] = PD concentration + [(maximal Δ in H\(^{+}\) or La)/exp\(^{-b t}\)], where Δ is change, b is rate constant, and t is time. Correlation coefficients were used to describe the relationship between actual data and the monoeponential curve fit. The restoration of [La] and [H\(^{+}\)] to PD levels was characterized by calculation of a half-time (t\(_{1/2}\)) and a t\(_{D}\). The t\(_{D}\) was defined as the time required to return the concentration to one-half of the difference between the peak postexercise and PD concentrations. The t\(_{D}\) was defined as the time required to return the concentration to PD levels.

In vivo muscle buffer capacity was calculated from the PS and 30-s-postspurt muscle pH and [La] values. The ratio of the change (PS to postspurt) in muscle [La] to muscle pH, expressed in units of millimoles La per kilogram pH unit or "Slykes" was used to estimate muscle buffer capacity (33). A paired t-test procedure was used to compare t\(_{1/2}\), t\(_{D}\), muscle buffer capacity, and performance sprint times between trials. All statistical analysis involving pH measurements was performed with [H\(^{+}\)]. The level of probability was set at P < 0.05 to reject the null hypothesis. All results are expressed as means ± SE.

RESULTS

Administration of the NaHCO3 solution produced significant (P < 0.05) increases in arterial blood pH, BE, and HCO3\(^{-}\) (Table 1). There were small but significant (P < 0.05) increases in muscle pH, Hb, and PCV.

Table 1. Select arterial blood and muscle parameters before (predose) and after (presprint) a 300 mg/kg gastric bolus of NaHCO3 and control solution

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control</th>
<th>NaHCO3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Predose</td>
<td>Presprint</td>
</tr>
<tr>
<td>Blood pH</td>
<td>7.42±0.01</td>
<td>7.42±0.01</td>
</tr>
<tr>
<td>BE, meq/l</td>
<td>0.8±0.3</td>
<td>0.2±0.3</td>
</tr>
<tr>
<td>HCO3(^{-}), meq/l</td>
<td>24.5±0.3</td>
<td>23.5±0.3</td>
</tr>
<tr>
<td>PaO2, Torr</td>
<td>94.4±1.9</td>
<td>92.9±2.0</td>
</tr>
<tr>
<td>PaCO2, Torr</td>
<td>39.4±0.6</td>
<td>37.5±0.6</td>
</tr>
<tr>
<td>Blood [La], mM</td>
<td>19.0±0.0</td>
<td>21.2±0.2</td>
</tr>
<tr>
<td>Muscles pH</td>
<td>7.08±0.02</td>
<td>7.11±0.01*</td>
</tr>
<tr>
<td>Muscle [La], mmol/kg wet wt</td>
<td>3.0±0.3</td>
<td>3.2±0.3</td>
</tr>
<tr>
<td>[Hb], g/dl</td>
<td>18.9±0.7</td>
<td>20.6±0.6*</td>
</tr>
<tr>
<td>PCV, %</td>
<td>53.6±1.6</td>
<td>56.6±1.5*</td>
</tr>
</tbody>
</table>

Values are means ± SE for 10 animals. BE, base excess; HCO3\(^{-}\), blood bicarbonate; PaO2, arterial PO2; PaCO2, arterial PCO2; blood [La], blood lactate concentration; Hb, hemoglobin concentration; PCV, packed cell volume. *Significant difference from predose to presprint, P < 0.05. †Significant difference between NaHCO3 and control, P < 0.05.
from PD to PS in both trials. Resting muscle [La], blood [La], PaCO2, and PaO2 were not affected by NaHCO3 ingestion. Significant (P < 0.05) increases in both blood (Fig. 1) and muscle (Fig. 2) [La] occurred in both trials over the sprint task. The highest measured blood [La], which occurred 2 min after exercise in both trials, was significantly (P < 0.05) higher in the NaHCO3 trial vs. control (20.4 ± 1.6 vs. 16.9 ± 1.3 mM, respectively). The highest muscle [La], which occurred 30 s after exercise, was similar in both trials (17.7 ± 1.8 vs. 16.2 ± 1.6 mmol/kg wet wt for NaHCO3 and control, respectively).

Postexercise blood and muscle [La] values were used to characterize the time course of recovery to preexercise conditions (Figs. 1 and 2, respectively). The t1/2 for blood [La] was similar for control compared with the NaHCO3 trial (14.5 ± 0.9 vs. 13.9 ± 1.8 min, respectively). There were no differences in blood [La] tPD values for control compared with the NaHCO3 trial (31.7 ± 2.5 vs. 35.4 ± 4.8 min, respectively). Postexercise muscle [La] t1/2 values (10.8 ± 1.6 vs. 14.5 ± 1.7 min, control vs. NaHCO3, respectively) and tPD values (38.4 ± 5.3 vs. 40.3 ± 4.4 min, control vs. NaHCO3, respectively) were similar.

In both trials, blood and muscle [H+] increased significantly (P < 0.05) during the exercise task. The 2-min postexercise blood [H+] (Fig. 3) was similar for NaHCO3 and control (53.6 ± 2.4 vs. 56.4 ± 2.0 nM, respectively). There was no differences in blood [H+] tPD values for control compared with the NaHCO3 trial (31.0 ± 2.5 vs. 35.8 ± 3.0 min, respectively). Postexercise muscle [H+] t1/2 values (10.8 ± 1.6 vs. 14.5 ± 1.7 min, control vs. NaHCO3, respectively) and tPD values (38.4 ± 5.3 vs. 40.3 ± 4.4 min, control vs. NaHCO3, respectively) were similar.

In addition, the calculated in vivo muscle buffer capacity was similar in both trials (45.1 ± 5.5 vs. 52.4 ± 6.3 mmol La·kg−1·pH−1 for NaHCO3 vs. control, respectively). There were no differences in postexercise blood [H+] t1/2 values (9.7 ± 1.4 vs. 9.5 ± 1.4 min for NaHCO3 and control, respectively) or tPD values (20.4 ± 2.5 vs. 20.8 ± 2.7 min for NaHCO3 and control, respectively). The NaHCO3 trial postexercise muscle [H+] t1/2 (7.2 ± 1.2 min) and tPD values (22.2 ± 2.4 min) were significantly (P < 0.05) reduced compared with the control trial (11.3 ± 1.6 and 32.9 ± 4.0 min for t1/2 and tPD values, respectively).

In addition, the calculated in vivo muscle buffer capacity was similar in both trials (45.1 ± 5.5 vs. 52.4 ± 6.3 mmol La·kg−1·pH−1 for NaHCO3 vs. control, respectively).
Both trials produced significant (P < 0.05) declines in $\text{PaCO}_2$, BE, and HCO$_3^-$ and increases in $\text{PaO}_2$, $[\text{Hb}]$, and PCV from PS to 2 min postexercise, with values returning toward preexercise levels throughout the remainder of the recovery period (Table 2). The NaHCO$_3$ trial, however, produced a significantly (P < 0.05) greater peak reduction in arterial BE ($-18.5 \pm 1.4$ vs. $-14.1 \pm 0.8$ meq/l) and HCO$_3^-$ ($-17.4 \pm 1.2$ vs. $-12.8 \pm 0.7$ meq/l) from PS to the initial postexercise sampling point (Fig. 5). This difference was only observed at the initial sampling interval, with no significant differences throughout the remainder of the recovery period.

Mean performance sprint times for the 603.5-m sprint task were not significantly different between the NaHCO$_3$ and control trials (46.29 ± 1.1 vs. 47.76 ± 0.9 s, respectively), although 6 of the 10 animals had reductions in individual sprint times. The magnitude of change in blood HCO$_3^-$ and pH at the PS sample in the NaHCO$_3$ trial and control trial was not related to the change in individual performance times ($r = 0.54$, $P = 0.10$, and $r = 0.25$, $P = 0.10$ for HCO$_3^-$ and pH, respectively). There was a trend for animals with lower control trial muscle buffer capacity to exhibit greater reductions in sprint times during the NaHCO$_3$ trial ($r = 0.55$, $P = 0.09$).

**DISCUSSION**

In this investigation, NaHCO$_3$ ingestion did not significantly affect the performance time in greyhounds.

---

**Table 2. Postsprint arterial blood-gas parameters, Hb, and PCV after NaHCO$_3$ and control**

<table>
<thead>
<tr>
<th>Parameter/Condition</th>
<th>2</th>
<th>5</th>
<th>10</th>
<th>20</th>
<th>30</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\text{PaCO}_2$, Torr</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>25.1 ± 1.4*</td>
<td>23.1 ± 1.9*</td>
<td>25.6 ± 1.9*</td>
<td>31.2 ± 1.9</td>
<td>34.3 ± 1.5</td>
</tr>
<tr>
<td>NaHCO$_3$</td>
<td>24.6 ± 1.9*</td>
<td>25.4 ± 1.8*</td>
<td>29.9 ± 1.9*</td>
<td>35.3 ± 1.2</td>
<td>38.1 ± 0.7</td>
</tr>
<tr>
<td>$\text{PaO}_2$, Torr</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>106.8 ± 2.5*</td>
<td>108.4 ± 1.6*</td>
<td>100.0 ± 2.8</td>
<td>86.9 ± 3.6</td>
<td>86.5 ± 3.3</td>
</tr>
<tr>
<td>NaHCO$_3$</td>
<td>109.1 ± 1.8*</td>
<td>105.1 ± 3.3*</td>
<td>91.4 ± 1.9</td>
<td>81.5 ± 3.1</td>
<td>80.7 ± 2.3</td>
</tr>
<tr>
<td>HCO$_3^-$, meq/l</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>10.7 ± 0.6*</td>
<td>10.5 ± 1.6*</td>
<td>13.6 ± 1.6*</td>
<td>20.3 ± 1.4</td>
<td>23.3 ± 0.5</td>
</tr>
<tr>
<td>NaHCO$_3$</td>
<td>11.1 ± 1.1*</td>
<td>12.5 ± 1.5*</td>
<td>16.9 ± 2.0*</td>
<td>23.3 ± 1.4</td>
<td>26.3 ± 0.8</td>
</tr>
<tr>
<td>BE, meq/l</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>-14.0 ± 0.8*</td>
<td>-13.7 ± 1.5*</td>
<td>-9.6 ± 2.0*</td>
<td>-1.6 ± 1.4</td>
<td>12 ± 0.4</td>
</tr>
<tr>
<td>NaHCO$_3$</td>
<td>-13.0 ± 1.2*</td>
<td>-11.4 ± 1.8*</td>
<td>-6.6 ± 2.5*</td>
<td>0.3 ± 1.6</td>
<td>3.4 ± 0.9</td>
</tr>
<tr>
<td>$[\text{Hb}]$, g/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>21.6 ± 0.4*</td>
<td>21.2 ± 0.4</td>
<td>20.3 ± 0.4</td>
<td>18.4 ± 0.5*</td>
<td>17.8 ± 0.5*</td>
</tr>
<tr>
<td>NaHCO$_3$</td>
<td>21.8 ± 0.6*</td>
<td>21.5 ± 0.5</td>
<td>20.4 ± 0.7</td>
<td>19.1 ± 0.5*</td>
<td>18.4 ± 0.5*</td>
</tr>
<tr>
<td>PCV, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>59.3 ± 1.4*</td>
<td>60.0 ± 1.7*</td>
<td>58.0 ± 1.7</td>
<td>52.5 ± 1.5*</td>
<td>50.4 ± 1.7*</td>
</tr>
<tr>
<td>NaHCO$_3$</td>
<td>59.1 ± 1.7*</td>
<td>58.9 ± 1.5*</td>
<td>57.3 ± 1.9</td>
<td>52.6 ± 1.7</td>
<td>50.3 ± 1.7*</td>
</tr>
</tbody>
</table>

Values are means ± SE for 10 animals. *Significant difference from presprint, $P < 0.05$. 

---

Fig. 4. Postexercise muscle $[\text{H}^+]$. Data points represent means ± SE for 10 animals; n, no. of data points. PD represents predose $[\text{H}^+]$. Correlation coefficients are shown for monoeponential curve fit. *Significant ($P < 0.05$) difference for control vs. NaHCO$_3$.

Fig. 5. Pre- to postsprint change in arterial blood base excess (BE) and HCO$_3^-$; Data represent means ± SE for 10 animals. *Significant ($P < 0.05$) difference for control vs. NaHCO$_3$.
sprinting over a 603.5-m distance. The NaHCO₃ trials produced significantly greater 2-min postexercise blood [La] and 30-s muscle [H⁺]. The NaHCO₃ trial did not reduce the intracellular acid-base disturbance associated with the exercise task based on muscle buffer capacity determinations, although the NaHCO₃ trial was associated with an accelerated rate of muscle acid-base restoration based on a significant reduction in the muscle [H⁺]t₁/₂ and tₚD values.

The potential ergogenic effect of NaHCO₃-loading regimens has been attributed to an enhancement of extracellular buffer reserve. This increased extracellular buffer reserve may subsequently affect rates of La⁻ and/or H⁺ efflux (10, 20, 21), minimizing the intracellular pH disturbance and thus reducing pH-related impairment of muscle function (4, 29, 32). The effectiveness of NaHCO₃ regimens may be related to the degree of alkalosis induced before exercise. In a meta-analysis (22) of human NaHCO₃-loading studies, a mean increase in blood pH of 0.07 ± 0.02 and HCO₃⁻ of 5.3 ± 1.4 mM was reported for investigations that used a 300 mg/kg dose. These responses were slightly higher than the 0.06 ± 0.005-unit increase in pH and 4.4 ± 0.4 meq/l increase in HCO₃⁻ observed in the present study, although these are interspecies comparisons.

In a meta-analytic review (22) of 35 human studies, 27 reported higher postexercise blood [La] after NaHCO₃-loading regimens. An increased blood [La] is usually interpreted as an indicator of increased anaerobic metabolism and/or muscle La efflux. In the present investigation, the 2-min postexercise blood [La] was significantly increased after NaHCO₃ loading. This difference, however, was only transient because blood [La] values from 5 to 30 min postexercise were similar based on similar t₁/₂ and tₚD values.

Postexercise muscle [La] was similar in both trials. In other investigations involving human subjects, metabolic alkalosis has been associated with an increase in peak postexercise muscle [La] (1, 31) or no difference (2), although these investigations employed a variety of exercise tasks. In the present study, the increase in peak blood [La] coupled with a similar muscle [La] may suggest an increased production of La⁻ in the NaHCO₃ trial; however, conclusions based on [La] are tenuous because muscle [La] is the net result of production, clearance, and oxidation.

The effect of NaHCO₃ ingestion on postexercise blood pH has also varied, with some investigators reporting higher postexercise blood pH (2, 8, 11, 13, 15, 16, 25, 34) or no treatment differences (1, 23). The postexercise blood pH difference between NaHCO₃ and control trials appears to parallel the preexercise difference produced by the NaHCO₃ dosing protocol (22). In this investigation, on the basis of similar peak blood [H⁺], PS-to-postprint change in [H⁺]t₁/₂ and tₚD values, there was no difference in the magnitude of disturbance or rate of recovery of blood acid-base balance. A greater peak reduction in arterial blood HCO₃⁻ and BE in the NaHCO₃ trial, however, suggests that greater blood H⁺ buffering occurred during exercise and in the initial recovery period. Similar results have been reported in both individual investigations (1, 11) and a recent review (22). A similar postexercise blood pH, coupled with the greater reduction in blood HCO₃⁻ and BE and increased blood [La], suggests that the buffer capacity of the arterial blood was enhanced in the NaHCO₃ trial.

Little information has been reported concerning effects of NaHCO₃-loading regimens on muscle pH. Rupp et al. (28) reported no treatment difference in pre- or postexercise muscle pH after a cycling task to exhaustion. In a series of five, 1-min cycling bouts, muscle pH immediately before the fifth bout was significantly higher in the NaHCO₃ trial compared with that in control trials, although the pH after the fifth bout (which continued to exhaustion) was similar in both trials (2). In the present investigation, the muscle [H⁺] at 30 s postexercise was significantly higher in the NaHCO₃ trial. An increased muscle [H⁺] during an exercise task over a fixed distance could occur if H⁺ production increased and/or there was a decrease in muscle buffering mechanisms. The increase in blood [La] coupled with no significant change in muscle [La] during the NaHCO₃ trial does suggest that La⁻, and thus H⁺ production, may have been higher in the NaHCO₃ trial. The greater reduction in blood BE and HCO₃⁻ from the PS to the 2-min-postexercise sample also suggests that more H⁺ ions were generated during the NaHCO₃ trial. If muscle buffering mechanisms, including the rate of H⁺ efflux, did not increase proportionally, then muscle [H⁺] could conceivably be higher.

The increased muscle [H⁺] coupled with no difference in muscle [La] suggests that NaHCO₃ ingestion did not effectively enhance intracellular muscle buffer capacity during exercise and the initial recovery period. The reduction in postexercise muscle [H⁺]t₁/₂ and tₚD in the NaHCO₃ trial suggests that NaHCO₃ ingestion reduces the time required to reach the preexercise acid-base status. However, this accelerated muscle acid-base restoration may be a consequence of the initially higher [H⁺] in the NaHCO₃ trial, although comparisons of muscle pH recovery after various levels of exercise induced pH disturbance have not been reported. The acceleration of muscle acid-base restoration may be beneficial when greyhounds are exposed to repeated bouts of exercise, as shown by positive effects on performance reported in investigations of humans that used repeated bout protocols (2, 7, 17). Despite the accelerated time course of muscle acid-base recovery, if NaHCO₃ administration increases the initial postexercise muscle [H⁺], its use in reducing the occurrence and/or severity of pH-induced postexercise muscle trauma is questionable and requires further investigation.

The lack of a significant effect on performance may be related to the metabolic demands of the exercise task chosen, the total dosage and time course of the NaHCO₃-loading regimen, and the potential variability in individual subject responses to ingestion of NaHCO₃. In this investigation, the mean performance time was reduced by 1.47 s, and 6 of the 10 animals had reductions in individual sprint times. Using the control trial mean performance time of 47.76 s, a reduction of
1.47 s is equivalent to a distance of ~18.6 m. In many races, a distance of <18.6 m separates the majority of the competitors, and certainly those animals placing first, second, or third. A treatment effect appears most often in those investigations that used a test criteria involving exercise time to exhaustion as opposed to a performance time for a specified exercise task (22). In this investigation, a greater muscle acid-base disturbance in the NaHCO$_3$ trial without a concomitant decrement in performance suggests that the acid-base disturbances which occur in this type of exercise task may not be limiting to muscle function. In addition, a preexisting high muscle buffer capacity and/or anaerobic capacity may minimize the ergogenic potential of NaHCO$_3$ ingestion, although improved performance in trained animals has also been reported (8, 34). In the present study, an inverse relationship (P = 0.09) was observed between control skeletal muscle buffer capacity and the individual changes in sprint times; this supports the concept that NaHCO$_3$ may be more beneficial in those athletes who exhibit lower muscle buffer capacities. Finally, animals in this investigation were exposed to a single NaHCO$_3$ dose vs. a training regimen with NaHCO$_3$ supplementation.

In summary, a 300 mg/kg dose of NaHCO$_3$ given 70 min before exercise increased postexercise blood [La] but did not reduce the magnitude of the muscle or blood acid-base disturbance associated with a 603.5-m sprint task or significantly affect performance times. In addition, postexercise muscle [H$^+$]$_{t_{0.5}}$ and t$_{50}$ values were reduced in the NaHCO$_3$ trial. This accelerated muscle acid-base restoration may be important when animals are exposed to repeated bouts of exercise.

Address for reprint requests: R. Engen, 2038 Dept. of Veterinary Physiology and Pharmacology, College of Veterinary Medicine, Iowa State Univ., Ames, IA 50011.

Received 2 Jul 1997; accepted in final form 1 May 1998.

REFERENCES


29. Sahlin, K. Effects of acidosis on energy metabolism and force generation in skeletal muscle. In: Biochemistry of Exercise,


