Electrolyte supplementation after prolonged moderate-intensity exercise results in decreased plasma [TCO₂] in Standardbreds

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Abstract
The present study used the physicochemical approach to characterize the changes in acid–base status that occur in Standardbreds after post-exercise electrolyte supplementation. Jugular venous blood was sampled from six conditioned Standardbreds on two separate occasions, at rest and for 24 h following a competitive exercise test (CET) designed to simulate the speed and endurance test of a 3-day event. After the CETs, horses were given water *ad libitum* and either a hypotonic commercial electrolyte solution, via nasogastric tube followed by a typical hay/grain meal, or a hay/grain meal alone. The electrolyte supplementation resulted in 2.2 mmol l⁻¹ decreased plasma [TCO₂] during the recovery period as compared with control. The primary contributor to the decreased [TCO₂] with electrolyte supplementation was a decreased strong ion difference ([SID]), as a result of the non-significant increase in plasma [Cl⁻]. Additionally, electrolyte supplementation resulted in faster restoration of hydration status compared with control, as evidenced by faster recovery of plasma [protein] and total weak acid concentration ([Atot]). It is concluded that oral administration of a hypotonic electrolyte solution after prolonged moderate-intensity exercise diminishes the post-exercise alkalosis, and that recovery of hydration status is still incomplete 24 h after exercise when no electrolytes are given. Thus, supplementation with electrolytes according to estimated sweat losses may attenuate post-exercise increases in plasma [TCO₂], which is of significant practical interest to the horse racing community, as a testing threshold of greater than 37 mmol l⁻¹ is used by many racing jurisdictions to determine whether a horse has been administered an alkalinizing agent.

Keywords: equine; TCO₂; electrolyte; exercise; H⁺; Stewart

Introduction
In horses, both prolonged submaximal exercise and brief vigorous exercise are associated with fluid and electrolyte shifts into and out of the plasma compartment and depletion of total body water and electrolytes that persists well into the recovery period. Prolonged submaximal exercise is also associated with the development of a mild systemic alkalosis that results from extracellular Cl⁻ loss in excess of Na⁺ loss as a result of thermoregulatory sweating. In order to enhance recovery, horses are commonly supplemented with electrolyte solutions both during and after exercise. Because electrolyte mixtures contain strong ions and affect hydration status, it is expected that electrolyte supplementation should have direct effects on plasma acid–base balance. According to the physicochemical approach, the independent variables that determine plasma acid–base status are the concentration of strong ions in solution - defined as the strong ion difference ([SID]), the partial pressure of carbon dioxide (PCO₂) and the concentration of weak acids in solution - defined as the total weak acid concentration ([Atot]). Thus, the dependent acid–base variables - [H⁺], bicarbonate concentration ([HCO₃⁻]) and total carbon dioxide concentration ([TCO₂]) - only change when one or more of the independent variables are altered.

There are few studies of acid–base effects of electrolyte supplementation during recovery from exercise in horses. Szucsik et al. determined that administration of seven different commercial electrolyte supplements,
as hypertonic pastes prior to exercise, had no effect on plasma concentrations of electrolytes, TCO$_2$ or plasma protein concentration ([PP]). The amounts of electrolytes given were relatively small, however (1.1–6.5 g Na, 0.9–3.7 g K and 2.6–13 g Cl), corresponding to only c. 21 sweat loss$^7$, and the time course was limited to only two samples post-exercise. When a greater amount of total electrolytes (20.2 g Na, 9.0 g K and 31.1 g Cl) was given as an isotonic electrolyte/water solution, increases in plasma [Cl$^{-}$] and [Na$^{+}$] (as compared with control), and no changes in plasma [K$^{+}$] or [PP], were seen$^4$; however, the effects on acid–base state were not studied. Thus, the acute time course of changes in acid–base state in response to electrolyte supplementation has not been characterized.

Measurement and interpretation of acid–base status is important in clinical practice and in the racing community to determine whether horses have been administered alkalinizing substances for the purpose of performance enhancement. Therefore, the purpose of the present study was to detail the time course and magnitude of the changes in all plasma constituents that determine acid–base state in horses, after oral administration of a commercially available electrolyte solution post-exercise, and to employ the physico-chemical approach to describe the resulting acid–base disturbances. It was hypothesized that oral administration of a hypotonic electrolyte solution after prolonged moderate-intensity exercise would result in (1) reduced plasma [H$^{+}$] and [TCO$_2$] and (2) enhanced recovery of hydration status, as compared with a control when no electrolytes are given.

**Methods**

**Animals**

Six Standardbred geldings (body mass 464 ± 10 kg; age 5–12 years) from the University of Guelph research herd were used. The study took place in June and July, and horses underwent a 4- to 6-week diet and exercise acclimation period during which they were housed in individual box stalls with 7 h of paddock turnout during the day. Horses were exercise conditioned 5 days/week on a high-speed treadmill (SATO, Sweden) and outdoor exerciser (Odyssey Performance Trainer, Campbellville, Ont., Canada), until able to comfortably perform a 60-min competitive exercise test (CET)$^{13,14}$ on a high-speed treadmill intended to significantly decrease muscle glycogen content$^5$ and result in total body water losses of 8–10 l. The CET was designed to simulate the second day (speed and endurance test - classic format) of a one-star CCI 3-day event and includes the following phases: 10-min walk (1.7 m s$^{-1}$), 10-min trot (3.7 m s$^{-1}$), 2-min gallop (10.0 m s$^{-1}$), 20-min trot (3.7 m s$^{-1}$), 10-min walk (1.7 m s$^{-1}$), 8-min canter (8.0 m s$^{-1}$) and 30-min walk (1.7 m s$^{-1}$).

The horses were maintained on a diet consisting of oats, DCAB (Na + K - Cl = −54.6 meq kg$^{-1}$) twice daily and mixed grass hay (DCAB = 368.8 meq kg$^{-1}$) thrice daily, with free access to water and a salt block. The amount of feed given was increased over this acclimation period such that during the final 2 weeks the horses were receiving 4 kg sweet feed and 6 kg hay daily (dietary DCAB = 200.4 meq kg$^{-1}$), and there were no significant changes in the body masses of the horses during this time. The animal care and use procedures were approved by the University of Guelph Animal Care Committee and performed in accordance with the Guidelines of the Canadian Council on Animal Care.

**Experimental protocol**

The study consisted of an electrolyte treatment and control, thus each horse performed the CET twice in randomized order, separated by an 8- to 10-day interval$^4,14$ during which time exercise conditioning was maintained. The CET was performed in a climate-controlled treadmill room with conditions intending to approximate those of a typical summer day in a temperate climate (temperature c. 25°C and humidity c. 70%). On both sampling days beginning at 7 am, the hair coat over the jugular vein, 10–20 cm below the mandible, was clipped short to the skin on both sides of the neck. Each jugular vein catheterization site was aseptically prepared for insertion of catheters. EMLA cream (2.5% lidocaine and 2.5% prilocaine; Astra Pharma, Mississauga, Ont., Canada), was applied topically 25–30 min before insertion of catheters to desensitize the skin. Local anaesthetic (2% Xylocaine; Astra Pharma) was injected subcutaneously to complete the anaesthesia. Catheters (14 gauge, 5.25 in.; Angiocath, Becton-Dickinson, Mississauga, Ont., Canada) were inserted anterograde into the left and right jugular veins, secured with tape and stitched to the skin. Four-way stopcocks with 50 cm extensions were attached to the catheters for ease of blood sampling. Patency of the catheters was maintained with sterile heparinized 0.9% NaCl (2000 IU1$^{-1}$ NaCl).

A pre-exercise blood sample was taken at 8 am, and then the CET was performed. Immediately upon completion of the final canter, an ‘end of exercise’ blood sample was taken, following which the horse walked for 10 min. Then, 20 min after cessation of exercise, the horse either (1) was nasogastrically administered a commercially available electrolyte solution (Perform’N Win, Buckeye Nutrition, Dalton, OH) according to manufacturer’s direction for an 8 l sweat loss (12 g Na, 24 g Cl and 9 g K), in 8 l water (osmolality = 212 mOsm kg$^{-1}$) or (2) stood in stocks for equivalent amount of time. Within 20 min of cessation of exercise, the horses were given 2 kg sweet feed and 2 kg hay (0 min of recovery), with access to water ad libitum. Horses were given
Acid–base balance after electrolyte supplementation

2 kg sweet feed and 2 kg hay at 6 h of recovery, and 2 kg hay at 12 h of recovery. Blood samples were taken at 20- to 60-min intervals up to 8 h of recovery and again at 24 h of recovery, and horses remained in their stalls for the duration of sampling.

Sample analysis

Each blood sample was collected into 7 ml lithium heparinized vacutainers and immediately analysed for plasma pH, the partial pressures of carbon dioxide (pCO₂) and oxygen (pO₂), and the plasma concentrations of Na⁺, Cl⁻, K⁺, Ca²⁺ and lactate⁻ using a Nova Stat Profile 9+ (NOVA Biomedical, Waltham, MA). Haematocrit (Hct) was measured by conductivity, and [HCO₃⁻] and total carbon dioxide (TCO₂) concentrations were calculated using the Henderson–Hasselbach equation by the Nova Stat Profile 9+. Blood was then transferred into two 1.5 ml Eppendorf centrifuge tubes and centrifuged for 5 min at 15 000 × g to separate the plasma. ([PP]) was determined (CV = 0.83%) by refractometry (Atogo clinical refractometer model SPR-T2; Atago, Tokyo, Japan).

Calculations

Plasma [H⁺] was calculated using the measured pH such that:

\[ \text{pH} = - \log [\text{H}^+] \]

Plasma ([SID]) was calculated as the sum of the plasma concentrations of the strong cations minus the strong anions, such that:

\[ [\text{SID}] = [\text{Na}^+] + [\text{K}^+] - [\text{Cl}^-] - [\text{lactate}^-] \]

In practice, the concentrations of the divalent cations and anions (Ca²⁺, Mg²⁺, PO₄³⁻ and SO₄²⁻) are small and the sum of their charges close to zero and can be ignored. The plasma concentration of weak ions ([A_tot]) was calculated by multiplying the [PP] (in g dl⁻¹) by 2.24. Calculations of dependent acid–base parameters (pH, [H⁺], [HCO₃⁻], TCO₂) were made by AcidBasics II software (©2003, PD Watson), using the following equation:

\[
\begin{align*}
[H^+] + (K_A + [\text{SID}])[H^+]^3 \\
+ K_A([\text{SID}] - [A_{\text{tot}}]) - (K_C \times p\text{CO}_2 + K_W)[H^+]^2 \\
- K_A(K_C \times p\text{CO}_2 + K_W) + (K_3 \times K_C \times p\text{CO}_2)[H^+] \\
- K_A \times K_3 \times K_C \times p\text{CO}_2) \\
= 0
\end{align*}
\]

where \( K_W \), \( K_A \), \( K_3 \) and \( K_C \) are the equilibrium constants for dissociations of water, weak acids, carbonic acid and bicarbonate, respectively.

\[
\begin{align*}
K_W &= 4.4 \times 10^{-14} \text{ (eq l}^{-1}\text{)} \\
K_A &= 2.22 \times 10^{-7} \text{ (eq l}^{-1}\text{)} \\
K_3 &= 5.76 \times 10^{-11} \text{ (eq l}^{-1}\text{)} \\
K_C &= 2.45 \times 10^{-11} \text{ (eq l}^{-1}\text{)}^2 \text{/mm Hg}
\end{align*}
\]

The contributions of the independent variables \([A_{\text{tot}}]\) and \(p\text{CO}_2\) to the dependent variable \([H^+]\) were determined by holding two of either [SID], pCO₂ or \([A_{\text{tot}}]\) constant while calculating \([H^+]\) in response to changes in the third independent variable. The contribution of the [SID] to the changes in \([H^+]\) was calculated by determining the contributions of pCO₂ and \([A_{\text{tot}}]\), and then subtracting these from the measured change in \([H^+]\).

Total body water loss during the CET was determined as the change in body mass after accounting for faecal losses.

Plasma osmolality was calculated according to the formula of Brownlow and Hutchins for equine plasma such that:

\[
\text{Osmolality (mOsm kg}^{-1}\text{)} = 1.86([\text{Na}^+] + [\text{K}^+]) \\
+ [\text{Glucose}] + [\text{Lactate}^-] \\
+ 9
\]

Statistics analysis

Data are presented as mean ± SE. The changes over time were assessed by one–way repeated measures ANOVA and the differences between treatments during the recovery period were assessed by two–way repeated measures ANOVA. When a significant \( F \) – ratio was obtained, means were compared using the all pairwise multiple comparison procedure of Holm–Sidak. Statistical significance was accepted when \( P \leq 0.05 \) at a power of 0.8.

Results

Ambient temperature and humidity during the CET were 23.7 ± 0.2°C and 69.3 ± 1.9%, respectively. Total body water loss during the CET was 8.4 ± 0.2 l.

All horses consumed all the feed offered by the 24-h sample. Every horse finished all of the oats given by 20-min post–feeding and had consumed all the hay given by 3–4 h post–feeding.

Independent variables and electrolytes

The plasma electrolytes are shown in Fig. 1. Plasma \([\text{Na}^+]\) (Fig. 1a) was increased from pre–exercise during the initial 20–60 min of the recovery period for both the trials, with no difference between treatments
 Plasma $[\text{Ca}^{2+}]$ was decreased at the end of exercise and until 40 min of recovery, with no differences between trials ($P = 0.361$) (Table 1). Plasma $[\text{lactate}^-]$ was increased at the end of exercise to $3.4 \pm 0.9$ and $3.4 \pm 1.3 \text{mmol}^{-1}$ in the electrolyte and control trials, respectively, with no differences between trials ($P = 0.539$) (Table 1).

The time course of changes in independent acid-base variables is shown in Fig. 2. There were no differences from pre-exercise in plasma $[\text{SID}]$ (Fig. 2a) for either trial; however, plasma $[\text{SID}]$ in the electrolyte trial was significantly lower than control ($P = 0.015$). Plasma PCO$_2$ (Fig. 2b) was decreased at the end of exercise and increased during the initial 20–60 min of recovery in both trials, with no differences between trials ($P = 0.108$). Plasma $[\text{A}_{\text{tot}}]$ (Fig. 2c) was increased from pre-exercise at the end of exercise until 60 min of recovery in the electrolyte trial and until the end of sampling in the control trial. There was a trend ($P = 0.109$) for a lower $[\text{A}_{\text{tot}}]$ during the recovery phase of the electrolyte trial.

**Dependent variables**

Plasma $[\text{H}^+]$ (Fig. 3) was decreased from pre-exercise at the end of exercise and increased during 40–60 min of recovery for both trials. There was a trend ($P = 0.065$) towards higher plasma $[\text{H}^+]$ in the electrolyte trial. When the contributions of the independent variables to plasma $[\text{H}^+]$ were determined (data not shown), the decrease in $[\text{H}^+]$ at the end of exercise was entirely due to the decreased PCO$_2$, while increases in $[\text{A}_{\text{tot}}]$ and PCO$_2$ were the primary contributors to the increase in $[\text{H}^+]$ during early recovery in both trials. The primary contributor to the trend towards increased $[\text{H}^+]$ during 120–480 min of recovery in the electrolyte trial was the decreased $[\text{SID}]$.

Plasma $[\text{TCO}_2]$ (Fig. 4a) at pre-exercise was $37.3 \pm 0.7$ and $37.4 \pm 0.5 \text{mmol}^{-1}$ for the electrolyte and control trials, respectively. Plasma $[\text{TCO}_2]$ was decreased at the end of exercise in both trials, but increased at 20 min of recovery in the control trial only ($40.0 \pm 0.5 \text{mmol}^{-1}$). There was a significant ($P = 0.033$) treatment effect such that plasma $[\text{TCO}_2]$ was lower during recovery in the electrolyte, compared with the control, trial. The contributions of the independent variables to the change in plasma $[\text{TCO}_2]$ are shown in Fig. 4b and 4c. The main contributor to the decreased $[\text{TCO}_2]$ at the end of exercise in both trials was the increased $[\text{A}_{\text{tot}}]$, with decreased PCO$_2$ and $[\text{SID}]$ contributing to a lesser extent. The increased $[\text{TCO}_2]$ at 20 min of recovery in the control trial (Fig. 4b) was entirely due to increased $[\text{SID}]$, while the overall decreased $[\text{TCO}_2]$ in the electrolyte trial (Fig. 4c) was primarily due to increased $[\text{A}_{\text{tot}}]$ during the initial 60 min of recovery and due to a decreased $[\text{SID}]$ during the latter 120–480 min of recovery.
Acid–base balance after electrolyte supplementation

Table 1

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>Electrolyte Control</th>
<th>Electrolyte Control</th>
<th>Electrolyte Control</th>
<th>Electrolyte Control</th>
<th>Electrolyte Control</th>
<th>Electrolyte Control</th>
<th>Electrolyte Control</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>[Hct]</td>
<td>[Ca²⁺]</td>
<td>[Lactate]</td>
<td>[Glucose]</td>
<td>[PP]</td>
<td>PO₂</td>
<td></td>
</tr>
<tr>
<td>End</td>
<td>7.50</td>
<td>0.01*</td>
<td>31.0</td>
<td>0.01*</td>
<td>30.4</td>
<td>0.01*</td>
<td>30.3</td>
</tr>
<tr>
<td>180</td>
<td>7.44</td>
<td>0.01*</td>
<td>35.5</td>
<td>0.01*</td>
<td>34.6</td>
<td>0.01*</td>
<td>33.5</td>
</tr>
<tr>
<td>240</td>
<td>7.44</td>
<td>0.01*</td>
<td>34.9</td>
<td>0.01*</td>
<td>34.5</td>
<td>0.01*</td>
<td>33.6</td>
</tr>
<tr>
<td>300</td>
<td>7.45</td>
<td>0.01*</td>
<td>36.9</td>
<td>0.01*</td>
<td>37.2</td>
<td>0.01*</td>
<td>36.8</td>
</tr>
<tr>
<td>360</td>
<td>7.43</td>
<td>0.01*</td>
<td>34.2</td>
<td>0.01*</td>
<td>35.7</td>
<td>0.01*</td>
<td>34.5</td>
</tr>
<tr>
<td>480</td>
<td>7.43</td>
<td>0.01*</td>
<td>34.8</td>
<td>0.01*</td>
<td>35.0</td>
<td>0.01*</td>
<td>34.2</td>
</tr>
<tr>
<td>1440</td>
<td>7.45</td>
<td>0.01*</td>
<td>35.0</td>
<td>0.01*</td>
<td>35.7</td>
<td>0.01*</td>
<td>35.0</td>
</tr>
</tbody>
</table>

*Significantly different from baseline (pre-exercise) time point for the control and electrolyte trials, respectively. Values are mean ± SE in mmol/l, except Hct in per cent, [PP] in g/dl and PO₂ in mm Hg.
decreased at the end of exercise, and was significantly lower in the electrolyte trial ($P = 0.033$).

### Water consumption, plasma osmolality and other variables

Calculated plasma osmolality (Fig. 5) was increased at the end of exercise, and from 20 to 60 min and from 20 to 240 min of recovery in the control and electrolyte trials, respectively. Plasma osmolality decreased throughout the recovery period in the control trial such that it was significantly lower than pre-exercise from 360–420 min of recovery; however, there was no difference between trials ($P = 0.104$). Total water intake (including the 8 l given nasogastrically) was significantly greater in the electrolyte trial, such that at 24 h of recovery (Fig. 6) was $49.5 \pm 4.2$ and $34.8 \pm 2.2$ l in the electrolyte and control trials, respectively ($P = 0.018$). Total water intake over the entire time course of the trials also showed a treatment effect, with total intake in the electrolyte trial significantly greater than the control ($P = 0.014$).

[PP] was increased from pre-exercise at the end of exercise until 60 min of recovery in the electrolyte trial, and until the end of sampling in the control trial, with no difference between treatments ($P = 0.169$) (Table 1). Hct was increased at the end of exercise and remained increased until 60 min of recovery in both trials, with no difference between trials ($P = 0.348$) (Table 1). Plasma PO$_2$ was increased at the end of exercise in both trials, with no difference between trials ($P = 0.901$) (Table 1). Plasma [Glucose] was increased from the end of exercise until 180 min of recovery, with no difference between trials ($P = 0.399$) (Table 1).

### Discussion

This study appears to be the first to detail the time course of acute changes in plasma-dependent and independent acid-base variables in response to post-exercise electrolyte supplementation in horses.
The electrolytes were administered according to manufacturer's directions to replace the approximate sweat losses during exercise, and both the control and electrolyte treatments were designed to imitate recovery protocols typical of the industry and thus included meal feeding with water *ad libitum*. The nasogastric administration of a hypotonic commercial electrolyte solution in 8 l of water, followed by a typical hay and grain meal, resulted in decreased plasma \([\text{TCO}_2]\) during the recovery period as compared with the control. The primary contributor to the decreased \([\text{TCO}_2]\) with electrolyte supplementation was a decreased \([\text{SID}]\), as a result of the non-significant increase in plasma \([\text{Cl}^-]\).

**Independent variables**

Despite the fact that none of the individual strong ions exhibited a treatment effect of electrolyte administration, plasma \([\text{SID}]\) was lower during recovery in the electrolyte trial. This result illustrates the importance of determining the independent variables as opposed to looking only at individual plasma electrolyte concentrations. In both trials, plasma \([\text{Na}^+]\) was increased and plasma \([\text{K}^+]\) was decreased during the initial 60 and 120 min of recovery, respectively, but their concentrations did not differ with electrolyte treatment. This is in agreement with previous studies that found no change in plasma \([\text{Na}^+]\) or \([\text{K}^+]\) with either isotonic or hypertonic electrolyte supplementation when free access to water was provided. This is in large part due to the large distribution volume for supplemented electrolytes, comprising c. 1001 of extracellular fluid and 2001 of intracellular fluid. The increase in plasma \([\text{Na}^+]\) during early recovery in both trials coincided with increases in \([\text{PP}]\) and Hct, and thus is likely a result of a cyclical fluid shift between the plasma compartment and the gastrointestinal tract as a result of feeding. The decrease in plasma \([\text{K}^+]\) during early recovery has been demonstrated previously after moderate- and high-intensity exercise, and can be explained by high rates of \(\text{Na}^+/\text{K}^+\)-ATPase activity in previously contracting muscles. Plasma \([\text{Cl}^-]\) in the present study did not differ from pre-exercise throughout the electrolyte trial, but was significantly decreased from pre-exercise to 300 to 480 min of recovery in the control trial, indicating that electrolyte supplementation resulted in maintenance of plasma \(\text{Cl}^-\) homeostasis and effectively replaced sweat \(\text{Cl}^-\) losses. Equine sweat contains a high \([\text{Cl}^-]\), and decreases in plasma \([\text{Cl}^-]\) have been demonstrated after endurance and moderate- and high-intensity exercise. The maintenance of plasma \([\text{Cl}^-]\) in the present study resulted in an overall lower \([\text{SID}]\) with electrolyte treatment, and provides evidence that supplementation with adequate electrolytes diminishes the post-exercise alkalosis that results when sweat losses of \(\text{Cl}^-\) exceed those of \(\text{Na}^+\).

In both trials, plasma \(\text{PCO}_2\) was decreased at the end of exercise, probably due to an increased alveolar ventilation as a result of muscular exercise, and increased in the initial 40–60 min of recovery. An increase in glycolytic activity post-feeding may account for the increased \(\text{PCO}_2\) during early recovery, as Stutz et al. found an increase in venous \(\text{PCO}_2\) 1 h after the feeding of a mixed hay and grain meal following exercise.

Electrolyte administration resulted in faster restoration of hydration status compared with control, as...
evidenced by faster recovery of [PP] and [Atot]. Plasma [A\text{tot}] and [PP] returned to pre-exercise by 120 min of recovery, when horses were administered the hypotonic electrolyte solution prior to consuming their typical post-exercise meals and water ad libitum. In contrast, when the horses were provided with their normal meals and water ad libitum (control), [PP] and [A\text{tot}] remained increased from pre-exercise throughout the remainder of the sampling period. Total water intake was significantly greater over the 24-h recovery period when electrolytes were given, even though the supplement was hypotonic. Similarly, Sosa Leon et al.\textsuperscript{25} found that horses administered a hypertonic electrolyte paste after furosemide-induced dehydration had higher water consumption and lower [PP] during the 6-h recovery period. In contrast to humans, equine sweat is hypertonic to plasma,\textsuperscript{7} resulting in an exercise-induced plasma dehydration that is isotonic to hypotonic. Thus, when electrolytes lost in sweat are not replaced, there is no increase in plasma osmolality to stimulate a thirst response. Additionally, if water alone is given after exercise, this reduces plasma tonicity, resulting in greater suppression of thirst and excretion of the water with additional electrolytes in the urine.\textsuperscript{8,26} Indeed, in the present study, plasma osmolality was increased up to 4 h after electrolyte administration, while in the control trial plasma osmolality decreased throughout the recovery period such that it was significantly lower than pre-exercise by 6 h of recovery. In agreement with this, our data demonstrate that electrolyte supplementation after exercise is required for full recovery of hydration status, and that recovery of hydration status is still incomplete 24 h after exercise when no electrolytes are given.

The presence of dextrose in the electrolyte supplement serves as a direct source of glucose to provide cellular energy to subserve increased rates of epithelial transport of Na\textsuperscript{+} and water across the small intestine.\textsuperscript{27,28} The amount of dextrose administered, even with 8 l of supplement, was insufficient to result in a glycaemic effect compared with control, indicating an appropriate dextrose concentration (31 mmol l\textsuperscript{-1}) of the Perform’N Win supplement.

Dependent variables

This appears to be the first study to show that supplementation with a hypotonic electrolyte solution post-exercise, in addition to a typical hay/grain meal and water ad libitum, resulted in decreased plasma [TCO\textsubscript{2}] during the recovery period as compared with water ad libitum and a typical hay/grain meal alone. When the physicochemical determinants of the dependent variables were quantified (Fig. 4b and 4c), the primary contributor to the decreased plasma [TCO\textsubscript{2}] with electrolyte administration was a decreased plasma [SID] from 2 to 8 h of recovery. The overall lower plasma [SID] in the electrolyte trial can be entirely attributed to a tendency towards increased plasma [Cl\textsuperscript{-}] during recovery compared with control. An increased plasma [A\text{tot}] during early recovery also contributed to decreased [TCO\textsubscript{2}] in the electrolyte trial; however, [A\text{tot}] returned to pre-exercise by 120 min of recovery. In contrast, an increased [A\text{tot}] throughout the control trial actually contributed a slight acidifying effect throughout recovery. Finally, despite an overall decreased PCO\textsubscript{2} with electrolyte supplementation, PCO\textsubscript{2} had little effect on plasma [TCO\textsubscript{2}] during either the electrolyte or control trial.

The literature is limited with respect to previous studies on the effects of electrolyte supplementation on equine acid-base balance. Szucsik et al.\textsuperscript{12} reported that administration of seven different commercial electrolyte supplements as hypertonic pastes prior to a simulated race test had no effect on plasma [TCO\textsubscript{2}]; however, the amounts of electrolytes given corresponded to only c. 21 sweat loss, and the time course was limited to only two post-exercise samples at 60 and 90 min of recovery. In contrast, when horses were administered a greater amount of electrolytes (51 g Na, 24 g K and 99 g Cl) as a hypertonic paste after a furosemide-induced dehydration (mean losses of water, Na, K and Cl were 181, 40 g, 20 g and 75 g, respectively), plasma [H\textsuperscript{+}] and [HCO\textsubscript{3}]\textsuperscript{-} had returned to pre-furosemide levels by 2 h post-administration, while a plasma alkalosis persisted in the water-treated control horses.\textsuperscript{25} In the present study, coinciding with the decreased [TCO\textsubscript{2}], plasma [H\textsuperscript{+}] also showed a tendency to be increased in the electrolyte horses as compared with a more alkalotic control, and this was entirely due to decreases in [SID].

An improved understanding of the effects of post-exercise electrolyte supplementation on acid-base status is of practical interest to the racing community. A plasma [TCO\textsubscript{2}] testing threshold of greater than 37 mmol l\textsuperscript{-1} is used by many racing jurisdictions to determine whether a horse has been administered an alkalinizing agent for the purpose of performance enhancement (see the 2007 paper by Lindinger and Waller\textsuperscript{11}). Interestingly, pre-exercise plasma [TCO\textsubscript{2}] for the electrolyte and control trials in the present study was 37.3 ± 0.7 and 37.4 ± 0.5 mmol l\textsuperscript{-1}, respectively, with these horses fed a typical racehorse diet. In the control trial, which was intended to imitate a typical recovery protocol in the Standardbred racing industry, plasma [TCO\textsubscript{2}] was above 39 mmol l\textsuperscript{-1} for the first hour of recovery, and remained above 37 mmol l\textsuperscript{-1} for the entire duration of sampling. Accordingly, the results of this study suggest that some horses may naturally demonstrate [TCO\textsubscript{2}] in excess of the testing threshold, even when no alkalinizing substances have been given. Indeed, a previous study by this laboratory
Acid–base balance after electrolyte supplementation

also found that Standardbreds exhibited increased plasma \([\text{TCO}_2]\) 90–120 min after short-duration, high-intensity exercise, due to decreases in plasma \([\text{Cl}^-]\). It is concluded that sweat-induced losses of \(\text{Cl}^-\) can significantly increase \([\text{TCO}_2]\) by as much as 2 mmol \(\text{l}^{-1}\) with an 81 dehyration. This is also an important concern with dehydration due to equine transport\(^9\), excitement\(^10\) and/or high ambient temperatures\(^11\). Additionally, supplementation with electrolytes according to estimated sweat losses attenuates decreases in plasma \([\text{Cl}^-]\) and results in decreased \([\text{TCO}_2]\) compared with occasions when no electrolytes are given.

Summary and conclusions

The present study quantified the magnitude and time course of the main physicochemical determinants of acid–base status in Standardbreds after post-exercise electrolyte administration, and compared them with a control recovery protocol. The nasogastric administration of a hypotonic commercial electrolyte solution, followed by a typical hay and grain meal, resulted in decreased plasma \([\text{TCO}_2]\) during the recovery period as compared with control. The primary contributor to the decreased \([\text{TCO}_2]\) with electrolyte supplementation was a decreased [SID], as a result of non-significant increases in plasma \([\text{Cl}^-]\). It is concluded that oral administration of a hypotonic electrolyte solution after prolonged moderate-intensity exercise diminishes the post-exercise alkalosis, and that recovery of hydration status is still incomplete 24 h after exercise when no electrolytes are given.

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