Effects of acute intravenous aldosterone administration on Na\(^{+}\), K\(^{+}\), and water excretion in the horse

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Effects of acute intravenous aldosterone administration on Na\(^{+}\), K\(^{+}\), and water excretion in the horse. J Appl Physiol 92: 135–141, 2002;—The effect of a temporary increase in plasma aldosterone concentration on Na\(^{+}\), K\(^{+}\), and water balance was investigated in four horses. Aldosterone was injected intravenously for 6 h at 20-min intervals (total 5.4 μg/kg body wt). Samples were taken for 24 h before, during, and for 48 h after the treatment. Aldosterone treatment reduced the Na\(^{+}\) loss via urine and feces by 99 and 72%, respectively, later followed by a marked increase in Na\(^{+}\) excretion by both pathways. During the first 6 h after the treatment, fecal K\(^{+}\) excretion was elevated, and the plasma K\(^{+}\) concentration was lowered. Fluid was retained throughout the treatment period and for 12–15 h thereafter. In a second experiment, exercise was performed once after aldosterone treatment and once without prior treatment. Sweat samples were collected, and the composition was not altered after treatment. It was concluded that acute aldosterone injections reduce Na\(^{+}\) losses in both feces and urine but not in sweat. In addition, the feces was shown to be the main excretion pathway of aldosterone.

Herbivores have a low Na\(^{+}\) intake, and the Na\(^{+}\) balance is challenged when Na\(^{+}\) losses increase. Horses may lose large amounts of Na\(^{+}\) during exercise because their sweat, which is iso- or even hypertonic compared with plasma, has a high NaCl content (22). It is well known that in various other species aldosterone reduces Na\(^{+}\) losses via the kidneys and that the reabsorption of Na\(^{+}\) involves systems that exchange either K\(^{+}\) or hydrogen ions for Na\(^{+}\) ions. Aldosterone has also been shown to stimulate Na\(^{+}\) absorption in other parts of the body, e.g., the proximal colon of ponies (6), the salivary parotis glands in sheep (3), and the eccrine sweat glands of humans (7). In contrast to the human sweat gland, the sweat gland of equines is apocrine (10), and it is not known whether these glands are affected by aldosterone. The high Na\(^{+}\) content of horse sweat may indicate that these glands are unresponsive to aldosterone. In fact, in the acute situation sweat Na\(^{+}\) concentration has been shown to increase with duration of sweating (18, 21). However, contradictory results have been reported for the long-term regulation of Na\(^{+}\) secretion in sweat. In one study, a 10-wk training program failed to influence sweat composition in horses (20), whereas McCutcheon and Geor (21) showed that the sweat Na\(^{+}\) concentration was reduced after 8 wk of training.

Studies of plasma aldosterone concentration (PAC) have been made in horses at rest (14) and in ponies (4) and horses (5) after feeding. In athletic horses with a low Na\(^{+}\) intake, we have observed individual plasma aldosterone levels at rest of ~1,300 pmol/l (16). Levels of 500 pmol/l have been reported in connection with short-term exercise (13, 16), and individual levels of 1,000 (26) and 2,900 pmol/l (17) have been observed after more endurance-like exercise. The importance of aldosterone during exercise is not known, but it is probably stimulated by the increased extracellular K\(^{+}\) concentration and involved in the regulation of K\(^{+}\) excretion and reuptake to muscle tissue. It is also possible that it is released secondary to a mechanism intended to stimulate chloride retention (23). However, postexercise increased plasma aldosterone levels have mainly been suggested to be released to reabsorb Na\(^{+}\) (17, 23). We have shown that increased plasma aldosterone levels in horses are associated with a low fecal Na\(^{+}\) concentration and a high K\(^{+}\) concentration (15), but, to our knowledge, the role of aldosterone in the regulation of total fluid balance in horses has never earlier been investigated.

As far as we know, the investigation by Clarke et al. (6) is the only report in which exogenous aldosterone earlier was administered to horses, and information on half-life and excretion patterns of aldosterone in horses is lacking. The routes of excretion of aldosterone and its metabolites seem to vary between different species. In humans, the metabolites are mainly excreted in the urine (11), whereas in rats fecal excretion has been shown to account for almost 70% (19).

The aim of this study was to investigate the effect of a temporary increase in PAC on the total excretion of Na\(^{+}\), K\(^{+}\) and water in horses, as well as on the exercise-induced sweat composition. It was hypothesized that horses would show a Na\(^{+}\)-saving response in feces...
and urine but not in sweat after acute aldosterone injections. In addition, we investigated the excretion pathways of aldosterone before, during, and after aldosterone administration.

METHODS

Animals and diets. Four Standardbred geldings were used in the study (body wt 453–550 kg; age 4–8 yr). A fifth gelding was used for a pilot study (body wt 492 kg; age 7 yr). All horses were housed in the experimental stalls for at least 3 wk before the experiment. The horses were kept in individual boxes (10 m²) and could move freely within their boxes throughout the experiment. On days during which no blood samples were taken, they had an outdoor session in sandy paddocks for 4 h/day. The horses were fed at 0600, 1200, 1600, and 2100 h. The diet consisted of 14.3 g grass hay–kg body wt⁻¹•day⁻¹ and 1.9 g concentrates–kg body wt⁻¹•day⁻¹, corresponding to the Swedish recommendations for metabolic energy and protein supply for horses performing light exercise (13.6 MJ/100 kg body wt, 76 g digestible protein/100 kg body wt). The concentrates included 20.2% oats, 10.3% wheat germ, 4.1% rapeseed oil, 3.15% NaCl, and 0.15% protein/100 kg body wt). The concentrate solution included 82.3% oats, 10.3% wheat germ, 4.1% rapeseed oil, 3.15% NaCl, and 0.15% protein/100 kg body wt). The daily Na⁺ intake (hay and concentrates) was 29 mg/kg body wt, which corresponds to ~150% of the suggested maintenance requirement (24). The daily K⁺ intake was 162 mg/kg body wt. Daily amounts of hay and concentrates were evenly distributed between meals. The horses were fed the experimental diet for 12 days before the study began. All horses were treated for intestinal parasites 10 days before the study began (Ivomec, Merial, London, UK). Water was available ad libitum (not in the paddocks) from automatic water vessels (flow 8 l/min), and intake was measured with flowmeters. The contribution of Na⁺ and K⁺ from the water was negligible. The horses were exercised on a treadmill every third day for 12 days before the experiment (5 min walk, 10 min trot at 5 m/s; 2 min walk; 1 min trot at 8 m/s and 2.5% incline; 1 min walk; 1 min trot at 8 m/s and 2.5% incline; and 5 min walk). The study took place during September and October in 1997. The average daily outdoor temperature and relative humidity decreased from 6.0 to 3.7°C and from 92 to 85%, respectively, during this period. The study was approved by the Local Ethics Committee of Uppsala.

Pilot study. A pilot study was conducted in one gelding for 3.5 days immediately before the experiment. The aim of this study was to determine the effects of jugular injections of aldosterone on the plasma concentration of aldosterone and the urinary Na⁺ excretion. The injected dose was intended to give a plasma aldosterone level around or above 1300 pmol/l, which have been observed in athletic horses at rest (16). In addition, plasma K⁺ (pK) was measured, and an electrocardiogram was recorded to register any effects of a reduced pK (for a description of the methods, see below). Day 1 was a control day, and aldosterone was injected on day 2 between 0600 and 1200 h. The aldosterone solution was prepared by dissolving 2.66 mg (5.4 µg/kg body wt) aldosterone (β-aldosterone, Sigma-Aldrich, St. Louis, MO) in 3 ml of ethanol (98%) and 92 ml of saline (0.9% NaCl). A 5-ml amount of this solution was injected via a jugular catheter every 20 min. Blood samples (12 ml) were taken via a jugular catheter before every injection as well as every hour between 1200 and 1800 h and every third hour between 1800 and 0300 h. An extension tube was attached to the catheter to facilitate injections and blood sampling.

The PAC increased from a level of 100–300 to 600–1,200 pmol/l during the first 3 h of the injection period and reached 1,800–2,900 pmol/l during the last 3 h. The PAC dropped rapidly after the injections ceased and was already down to 200–300 pmol/l 2 h later. The pK decreased from 4.0 mmol/l before the injections to 3.1 mmol/l after 6 h. There were no abnormalities in the electrocardiogram during the injection period and for at least 3 h afterward. Urinary Na⁺ excretion was low throughout the last 3 h of the injection period and was still low at 2100 h. However, the pattern of Na⁺ excretion was similar on day 1, suggesting that it was under the influence of a diurnal rhythm. Therefore, it was decided to conduct the aldosterone injections between 0000 and 0600 h instead.

Experimental design. The experiment was conducted over 14 days. Blood samples were taken, and the total outputs of urine and feces were collected for 84 h (3.5 days) starting at 0000 on day 1 in two of the horses and 24 h later in the other two horses. Aldosterone injections started 24 h after the horses commenced the experiment. During the first 24 h of the experiment, no treatment was given (control day). The diurnal PAC is mainly dependent on the Na⁺ balance of the horse (16). In addition, K⁺ excretion may be affected by feeding frequency (16). A single control day, as used in the present study, can therefore be justified because all horses were in positive Na⁺ balance before the experiment and were fed according to a very precise schedule throughout the study.

The aldosterone solution was prepared by dissolving 24 mg of aldosterone in 8 ml ethanol. Individual solutions were then prepared as described for the pilot study. In all horses, a catheter was introduced into one of the jugular veins, which was, after careful flushing and cleaning between each injection, also used for the blood sampling. This procedure had been tested during the pilot study. Separate catheters were not used because it was presumed that at some point during the experiment the catheter would fail and have to be replaced while still having access to an undamaged jugular vein. An extension tube was attached to the catheter to facilitate blood sampling and cause a minimal amount of disturbance to the horse. A 5-ml amount of the individual aldosterone solution was injected every 20 min between 0000 and 0600 h. Blood samples were taken hourly between 0000 and 0600 h and every third hour between 0600 and 2400 h on all days. The feces were collected with a device consisting of a bag hanging under the tail just below the anus, attached to a girth. The bag was emptied manually at least every 60 min. The feces were collected into samples corresponding to 3-h periods and stored at −20°C. Urine was collected in a bottle hanging in front of the hind legs. The bottle was emptied as soon as urine was passed. The horses were adjusted to the collecting device during the 3-wk period preceding the experiment. The horses were hand walked for 20 min during the collection days. Samples of saliva were taken at 1200 h the day before the injections and on the day of the injection (i.e., 6 h after the last injection). The saliva was absorbed by pressing a piece of filter paper against the mucous membrane under the tongue. This paper was then placed in a tube with deionized water (0.8 ml). Before-and-after weighings of the paper-tube-water combination allowed us to calculate the amount of saliva collected. We were then able to determine the levels of Na⁺ and K⁺ in the dissolved saliva.

On days 10 and 14, all four horses were exercised on the treadmill. On each day, two of the horses had been earlier treated with aldosterone (Eₐ), whereas the other two had not (E₀). The amount of aldosterone injected and the duration of the injections were the same as earlier described. The Eₐ was performed 3 h after the last aldosterone injection was given, by the time when the maximal effect was observed during the
experiment at rest. Because only one horse at a time could exercise on the treadmill, the start of the injection period was 2300, 0000, 0100, and 0200 h in the four horses, respectively. The horses were exercised at the same time of the day on E_a. The main purpose of the exercise test was to initiate and stimulate substantial sweat secretion, and therefore a low-intensity test was performed, covering 7,020 meters (heart rate and hematocrit ~130 beats/min and 46%, respectively). The exercise consisted of a 5-min walk (1.7 m/s) followed by a 20-min trot (5 m/s) and another 5-min walk (1.7 m/s). Sweat was collected every fifth minute during the trot. It was collected from the abdomen with absorbent filter paper in a nonventilated capsule attached to the skin surface by a girth. The sweat samples were treated in the same way as the samples of saliva. Blood samples were taken every hour after the start of exercise; and 15, 45, and 120 min postexercise. The horses were weighed before and after exercise. Fluid loss was calculated as the body weight loss minus the fecal excretion. No urine was excreted while the horses were exercising. The heart rate was recorded during exercise (Min- gograf 410, Siemens Elema, Solna, Sweden), and rectal temperature was measured with a digital thermometer before and after exercise.

Analysis. The blood samples were collected in lithium-heparinized tubes kept on ice until centrifugation and thereafter were stored at −20°C. The hematocrit was determined in duplicate by centrifuging blood in capillary tubes (12,000 rpm, ALC microhematocrit centrifuge, Milan, Italy). The total plasma protein concentration (TPP) was measured with a refractometer (Cambridge Instruments, Buffalo, NY). Plasma, feed, and fecal Na⁺ and K⁺ concentrations were measured by use of an ion-selective electrode method (System E2A electrolyte analyzer, Beckman Instruments, Brea, CA). Na⁺ and K⁺ concentrations in urine, saliva, and sweat were determined by flame photometry (Auto Cal Flame photometer, Instrumental Lab 943, Milan, Italy). The PAC was determined after extraction of fat and proteins (acetone and petroleum ether extraction) by use of a commercially available RIA kit (Coat-a-Count, aldosterone, DPC, Los Angeles, CA). The samples for the standard curve were extracted in the same way. The quality control was run using MultiCalc software version 2.0 (Wallac, Turku, Finland). The within-assay variation was 6%, and the between-assay variation was <10%. Feces and feed samples were dried (65°C, 24 h), milled (1 mm), ashed (600°C, 12 h), and dissolved in 1 M HNO₃ (1 h). After neutralization of the samples (1 M NH₃), the Na⁺ and K⁺ concentrations were measured. Evaluation of this method has shown that the results are similar to those obtained with fresh samples boiled in HNO₃ and perchloric acid. Analysis of aldosterone in feces was made on the dried and milled samples. A 0.25-g sample was dissolved in 5 ml of phosphate buffer (including 0.1% gelatin and 0.1% bovine albumin, pH 7.4) and extracted twice with 3 ml of CH₂Cl₂. The extraction was evaporated (37°C) and analyzed with the same RIA as described above (sample A). A conjugate of aldosterone may be split under extraction at pH 1, and aldosterone is released in the free form (2). Therefore, a 0.5-ml amount of 32 N HCl was added to the residue remaining after the first extraction protocol, and this sample (pH 1) was extracted twice with 3 and 2 ml of CH₂Cl₂, respectively, and was then evaporated and analyzed with the RIA (sample B). The urine (2 ml) was analyzed in the same way as the feces except that during the second extraction protocol (sample B) the sample was extracted twice with 3 and 1 ml of CH₂Cl₂, respectively.

Statistical analysis. Values are presented as means ± SE. All data were subjected to analysis of variance (GLM procedure in the Statistical Analysis Systems package, SAS Institute, Cary, NC). Values of urinary Na⁺ and K⁺ excretion were logarithmically transformed to get a normal distribution of data before the analysis. For analysis of urine, feces, and blood variables at rest, the following model was used

\[
Y_{ijk} = \mu + \alpha_i + \beta_j + \gamma_{k} + (\beta\gamma)_{jk} + e_{ijk}
\]

where \(Y_{ijk}\) is the observation, \(\mu\) is the mean value, \(\alpha_i\) is the effect of animal, \(\beta_j\) is the effect of day, \(\gamma_{k}\) is the effect of sample (time), \((\beta\gamma)_{jk}\) is the effect of interaction between day and sample, and \(e_{ijk}\) is the residuals; \(e_{ijk} \sim \text{IND}(0, \sigma^2)\). Data from the exercise tests were analyzed by use of the same model, although \(\beta\) is the effect of treatment (E_a or E_0). Differences within a treatment or day were tested for significance via a paired t-test. The significance level was set at \(P < 0.05\).

RESULTS

Effects on urinary composition and excretion. The urinary Na⁺ excretion every 6 h was between 10 and 46 mmol on the control day. The excretion was lowered for 6 h after the treatment compared with the corresponding period on the control day and then increased dramatically from 12 h after the treatment stopped and was high until 30 h after the treatment (Fig. 1). The urinary K⁺ excretion per every 6 h was between 520 and 960 mmol on the control day and was not significantly altered after aldosterone treatment. The Na⁺/K⁺ ratio of urine was decreased for 6 h after the treatment and was then increased 12–24 h after the treatment compared with the same periods on the control day. There was a decrease in the mass of urine in the time period between 1200 and 1800 (Table 1), and the total mass of urine from the start of the treatment and until 12 h after the treatment was also decreased compared with the same period on the control day.

Effects on fecal excretion. The fecal concentration of Na⁺ tended to be lower (\(P = 0.056–0.10\)) 6–18 h after the treatment period compared with the same period
on the control day, and the Na\(^+\) excretion tended \((P = 0.058)\) to be lower 9–12 h after the treatment period (Fig. 2). The Na\(^+\) concentration was increased from 24 h after the treatment period and throughout the study, and the Na\(^+\) excretion was increased from 27 h after the treatment period and periodically during the rest of the study. There was an increase in fecal K\(^+\) excretion for 3 h after the treatment stopped, and then the K\(^+\) concentration decreased from 21 h after the treatment stopped and throughout the study (Fig. 2). The Na\(^+\)/K\(^+\) ratio of feces was increased from 24 h after the treatment stopped compared with the control day. The water content of the feces increased occasionally after the treatment (Table 2), but there were no differences in the total fecal excretion (day 1: 18.8 ± 3.6 kg, day 2: 17.7 ± 1.1 kg and day 3: 17.1 ± 1.3 kg).

**Effects on plasma variables, saliva, and water intake.** The mean PAC increased from levels of 95–385 pmol/l during the control day to 2,880–5,420 pmol/l during the treatment (Fig. 3). Aldosterone concentrations at control levels were reached again 3–6 h after the treatment had ended. There were no changes in the plasma Na\(^+\) concentration (pNa), except for the last sample taken during the treatment period (Fig. 3). The pK dropped after 5 h of treatment with aldosterone, and the lowest level (3.2 ± 0.4 mmol/l) was reached 3 h after the treatment ended (Fig. 3). Normal pK was reached 9 h after the treatment period. TPP dropped temporarily 2 h after the aldosterone injections started but was normal during the following 2 h (Fig. 3). TPP dropped again 5 h after the treatment started and was decreased by −4% in almost all samples until 15 h after the treatment period. The composition of saliva was not affected by aldosterone treatment (Table 3). Water intake during every 3-h period starting at 0000 h, 1.3 ± 0.5, 6.3 ± 2 (fed at 1200 h), 6.8 ± 0.5 (fed at 1600 h), 0.5 ± 0.3, and 3.5 ± 0.6 (fed at 2100 h) liters on the control day, and there were no significant changes in water intake during the experiment.

**Exercise.** The PAC increased after aldosterone treatment to levels similar to those during the experiment at rest. PAC dropped at the start of EA and was not statistically significantly different from E0. The fluid loss during exercise was 3.5 ± 0.3 and 3.9 ± 1.8 kg on E0 and EA, respectively (not significant). The Na\(^+\) and K\(^+\) concentration of sweat was not affected by aldosterone treatment or duration of exercise (Fig. 4). However, the Na\(^+\)/K\(^+\) ratio was lower after 20 min of exercise in EA compared with E0 (Fig. 4).

**Excretion of aldosterone.** The excretion of aldosterone took place predominately via the feces, both under control conditions and after treatment with aldosterone (Fig. 5). There was a small increase in the urinary excretion after the treatment, but the amount was negligible compared with the fecal excretion.

**DISCUSSION**

In accordance with our hypothesis, it was found that horses show a Na\(^+\)-saving response after acute aldoste-
Aldosterone injections with a decrease in both fecal and urinary output without any clear effects on sweat composition. Aldosterone was also found to play a role in the regulation of fluid balance shown by a reduced urine volume and a decrease in TPP. The first Na⁺-saving effects of aldosterone were observed at the end of the treatment period, and the Na⁺ loss via both urine and feces was ultimately reduced by 99% (for 6 h after the treatment) and 72% (from 9 to 12 h posttreatment), respectively. To our knowledge, this is the first time that the effect of aldosterone on the urinary excretion in horses has been demonstrated. Furthermore, the regulation of Na⁺ balance by fecal excretion has been shown to be of the same magnitude as urinary excretion. The effects of aldosterone treatment on the fecal excretion of Na⁺ is in accordance with results from Clarke et al. (6), who showed that the fecal Na⁺ excretion rate decreased and the K⁺ excretion rate increased within 8 h of aldosterone treatment. In their study, increased Na⁺ absorption was observed in all regions of the equine colon. These authors showed that this was due to both electroneutral and electrogenic processes, although the latter (i.e., increased Na-K-ATPase activity) was considered more likely within this time period. The exact mechanisms behind the increased K⁺ excretion after aldosterone treatment have not been demonstrated, and Clarke et al. (6) discussed the possibilities of secretory processes, passive transportation along a gradient, or even an inhibition of colonic K⁺ absorption.

The results from the present study and an earlier study on Standardbred horses with extremely low Na⁺ intake and high PAC (16) show that the fecal Na⁺ concentration can be reduced to \( \overline{50} \) mmol/g dry weight. This may also be the minimal fecal Na⁺ excretion rate for this breed. The nonsignificant reduction in fecal Na⁺ excretion (72%, \( P = 0.058 \)) after the treatment could be explained by the comparatively low rate of fecal Na⁺ excretion (15) under the control conditions and a limited potential to reduce it further. We presume that the response could have been stronger if the Na⁺ intake level had been higher rather than only 150% of the maintenance requirement used here, because excretion can increase with intake (15, 16).

In humans, changes in sweat composition have been observed within 4–8 h of treatment with aldosterone (8, 12). Grand et al. (12) also showed that the Na⁺/K⁺ ratio was reduced after 8 h and remained reduced up to 14 h posttreatment, although the urinary ratio had returned to control levels. The present study indicates that the equine sweat gland is not responsive to an acute intravenous aldosterone treatment. This is also in accor-

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### Table 3. Na⁺ and K⁺ concentration and Na⁺-to-K⁺ ratio in saliva under control conditions and after treatment with aldosterone

<table>
<thead>
<tr>
<th></th>
<th>Na⁺, mmol/l</th>
<th>K⁺, mmol/l</th>
<th>Na⁺-to-K⁺ Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>8 ± 1</td>
<td>19 ± 2</td>
<td>0.4 ± 0.1</td>
</tr>
<tr>
<td>Aldosterone</td>
<td>8 ± 1</td>
<td>21 ± 2</td>
<td>0.4 ± 0.1</td>
</tr>
</tbody>
</table>

Values are means ± SE; \( n = 4 \) horses.
dance with a study on eccrine glands of the cat pad, which did not respond to aldosterone treatment (9). In humans, it is well known that athletes and persons adapted to hot climates have lower sweat Na⁺ concentrations than normal persons and that this is mediated by aldosterone. Given the recent results from McCutcheon and Geor (21) and Lindinger et al. (18), it cannot be ruled out that aldosterone may affect sweat Na⁺ concentration during longer periods of exercise training in horses. The lack of any salivary response could be related to the rate of saliva secretion. The saliva samples were taken after a period during which the horses had not been eating, and under these conditions the salivary flow is low, as well as the electrolyte concentration (1). At low electrolyte concentrations, alterations in saliva composition may be difficult to detect.

At rest, treatment with aldosterone did not alter the pNa but decreased the pK and TPP. The reduction in TPP indicated that the plasma volume had expanded by ~1 liter. This increase is also equivalent to the reduction in the urine volume observed some hours later. If we presume that the plasma volume was 20 liters at the start of the experiment, then the increase in plasma volume should theoretically have decreased the pK from ~4.2 to 4.0 mmol/l. The remaining decrease or loss (20 mmol) was probably due to the increased fecal excretion (76 mmol) and/or an intracellular shift. The change in TPP contradicts the results of Clarke et al. (6), who reported that aldosterone treatment had no effect on either serum Na⁺ or TPP. However, they also reported a small decrease in serum K⁺ at the end of the experimental period. The difference in results between studies might be explained by the slightly higher dose used in the present study (5.4 μg/kg body wt in 6 h vs. 4 μg/kg body wt 3 h).
wt in 8 h) or to the fact that the Shetland ponies used by Clarke et al. (6) respond differently to aldosterone compared with Standardbred trotters.

It has been suggested that the PAC may be an important determinant of digesta water content in the colon of hindgut fermenter species (6). In rabbits, the diurnal variation in PAC and the cycle of soft and hard feces are associated, and exogenous aldosterone causes colonic changes that are similar to those observed when hard feces are produced (27). However, contrary to the prediction of this hypothesis, the fecal water content in the present study was not decreased. Nor did a study on horses whose various Na⁺ intakes resulted in different levels of diurnal PACs reveal any differences in fecal dry matter content (16).

To our knowledge, there are no earlier reports on the metabolism and excretion of aldosterone in the horse. Aldosterone is generally considered as a steroid with a half-life of ~20 min. In the present study, the plasma concentration decreased from 4,400 to 700 pmol 3 h posttreatment, indicating a half-life of ~1 h in these animals. Maybe the clearance rate was limited in this study and a more rapid clearance might be the case if the initial level is lower than the 4,400 pmol observed here. The fecal route was found to be the major excretion pathway for aldosterone. In control conditions, the fecal excretion of aldosterone was ~5,000 times the urinary excretion. There was a rapid, but comparatively small, response in the urinary excretion after treatment, probably due to the high PAC and an increased filtration. Sex differences in the metabolism of aldosterone have been reported in rats (25), and, because only geldings were used in the present study, it is uncertain whether our results can be conveyed to stallions or mares. In castrated male rats, the rate of biliary aldosterone excretion was markedly increased compared with intact males, and androgens were suggested to play an important role in regulating the hepatic metabolism of aldosterone and the clearance rate from the plasma (25).

Malin Connysson and Eva Werner offered assistance during the experiment, and Gunilla Druge-Boholm conducted the aldosterone analysis. Leif Eklund, Ann-Margret Hedberg, and Conny Karlsson placed their horses at our disposal during the experiment. All of the above persons deserve our sincere thanks.

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