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Use of Enteral Electrolyte Solutions Containing Different Energy Sources Administered by Continuous Flow via Nasoesophageal Tube in Dogs

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Abstract

This study compared the effects of two hypotonic enteral electrolyte solutions containing different energy sources, administered by nasoesophageal tube in continuous flow, on dogs subjected to water and food restriction. Evaluated parameters included serum concentrations of sodium, potassium, chloride, calcium, magnesium, phosphorus, urea, and creatinine, as well as urinary specific gravity, volume, and excretion of sodium, potassium, chloride, calcium, magnesium, urea, and creatinine. Six healthy adult dogs were experimentally submitted to water and food restriction and used in a 6 × 2 crossover design. All animals received both treatments. The treatments were as follows: ESMalt: 5 g of sodium chloride, 1 g of potassium chloride, 1 g of calcium acetate, 0.2 g of magnesium pidolate, and 9.6 g of maltodextrin diluted in 1000 mL of water (measured osmolarity: 215 mOsm L^{-1}); ESDext: 5 g of sodium chloride, 1 g of potassium chloride, 1 g of calcium acetate, 0.2 g of magnesium pidolate, and 9.6 g of dextrose diluted in 1000 mL of water (measured osmolarity: 243 mOsm L^{-1}). The solutions were administered at a rate of 15 mL kg^{-1} $hour^{-1}$ for four hours in continuous flow. In conclusion, hypotonic enteral electrolyte solutions promoted an increase in urinary volume and a decrease in urinary specific gravity without causing any adverse effects. The animals in the ESMalt group exhibited a higher urinary volume.

Keywords

Canine, Hypovolemia, Biochemical Test, Enteral Fluid Therapy

1. Introduction

Dehydration is one of the most common disorders in routine clinical medicine. This condition is present in many diseases and syndromes that affect dogs and is corrected through hydration with electrolyte solutions [1] [2]. The hydroelectrolytic losses that occur in animals can lead to various alterations in homeostasis, depending on the etiology of the disease, which consequently results in different types of electrolyte and acid-base imbalances. As a result, the composition of the electrolyte solutions used for rehydration can be adjusted according to the patient's biochemical profile.

Diarrhea in dogs can cause significant losses of fluids and electrolytes. These losses can be extensive and, when combined with reduced water intake, may lead to mild to severe dehydration and, in some cases, serious complications such as hypovolemic shock. In addition, these animals may simultaneously present with hyporexia or anorexia, weakness, and hypoglycemia [3].

Parameters commonly used to assess and monitor fluid and electrolyte deficits, in addition to clinical signs, include hematocrit, total protein, urea, creatinine, urinary specific gravity, urinary volume, Na^+, Cl^-, K^+, Ca^{2+}, and blood gas analysis [4]-[6]. Measuring urinary electrolytes, urea, and creatinine is also important for monitoring the treatment being administered. However, it should be noted that the literature on this subject in dogs is scarce.

Oral hydration in dogs has been used successfully to treat mild to moderate dehydration as an alternative to intravenous fluid therapy. Although controlled trials are still limited in veterinary medicine, this type of rehydration has been studied for the treatment of gastroenteritis and kidney disorders, with satisfactory results [7]-[9]. In dogs, this experimental study using enteral hydration via nasogastric tube in continuous flow is a pioneer.

The aim of this study was to evaluate the effects of two hypotonic enteral electrolyte solutions containing different energy sources, administered via nasoesophageal tube in continuous flow for four hours, on urinary specific gravity and volume, as well as blood and urinary electrolyte profiles in dogs submitted to water and food restriction. The hypothesis of this study is that the use of hypotonic enteral electrolyte solutions administered via nasogastric tube in continuous flow will increase blood volume without causing adverse effects.

2. Material and Methods

The experimental procedures were approved by the ethics committee in animal use of Universidade Federal de Viçosa (CEUA/UFV process number 96/15) following the guidelines of Brazilian legislation edited by the National Council for

the Control of Animal Experimentation (CONCEA).

Study design: The experimental trial was conducted in the municipality of Viçosa, Minas Gerais, at an altitude of approximately 640 meters, latitude −20˚73'56.03" and longitude −42˚85'76.62". The average temperature during the experimental period was 21.7˚C ± 3.1˚C, with relative humidity ranging from 43.6% to 89.6%. Six healthy Golden Retrievers (four males and two females), aged between two and six years, with a mean body weight of 33.5 kg and a body condition score of 3 [10], were included in the study. Clinical and laboratory evaluations were performed before the experimental period. Twelve hours prior to the start of hydration, the animals were placed in individual stalls with restricted access to food and water. The dogs were randomly assigned to two groups using a 6 × 2 crossover design, with three-day intervals between treatment cycles. The ESMalt group received an electrolyte solution containing 5 g of NaCl, 1 g of KCl, 1 g of calcium acetate, 0.2 g of magnesium pidolate, and 9.6 g of maltodextrin diluted in 1000 mL of water (measured osmolarity: 215 mOsm L^{-1}). The ESDext group received an electrolyte solution containing the same base composition, replacing maltodextrin with 9.6 g of dextrose (measured osmolarity: 243 mOsm L^{-1}) (**Table 1**).

Table 1. Composition and osmolarity of enteral electrolyte solutions.

Composition	ESMalt	ESDext
Sodium chloride	5 g	5 g
Potassium chloride	1 g	1 g
Calcium acetate	1 g	1 g
Magnesium pidolate	0.2 g	0.2 g
Maltodextrium	9.6 g	-
Dextrose	-	9.6 g
Osmolarity	215 mOsm L^{-1}	243 mOsm L^{-1}

The solutions were administered using nasoesophageal tubes −100 cm in length and 3 mm in diameter (Long nasogastric tube No. 8—Embramed, São Paulo, SP)—inserted into the right nostril and secured with superglue (Superbonder®). All solutions were administered at a rate of 15 mL kg^{-1} $hour^{-1}$ for four hours (replacement phase).

Collection of biological samples and laboratory evaluations: For laboratory testing, blood samples were collected by jugular venipuncture into vacuum tubes without anticoagulant (Vacuum II—10 mL, siliconized), to obtain serum for the measurement of magnesium (colorimetric method; Bioclin® kit), urea (UV enzymatic method; Bioclin® kit), and creatinine (colorimetric method; Bioclin® kit) using an automated biochemistry analyzer (HumaStar 300—Human, Itabira, MG). Additional venous blood samples were collected using sterile syringes containing lithium heparin (Pico50—Radiometer Medical ApS, Copenhagen, Denmark) to assess sodium, potassium, ionized calcium, and chloride levels (ABL80 FLEX—

Radiometer).

Urine volume was measured using a closed system and calculated with a graduated beaker. Urinary specific gravity was assessed using a refractometer. Urine samples (10 mL) were collected directly from the tube for analysis of sodium and potassium (Flame Photometer B462—Micronal, São Paulo, SP), and chloride, total calcium, magnesium, urea, and creatinine (HumaStar 300—Human).

Laboratory evaluations were conducted at the following time points: T-12h: immediately before food and water restriction; T0h: end of the restriction period, immediately before fluid therapy; T4h: end of the fluid therapy period; T8h: four hours after the end of fluid therapy.

Statistical Analyses: Descriptive statistics were used to calculate the means (±) and standard deviations of all variables. Data were assessed using the Lilliefors and Cochran & Bartlett tests to verify normality and homogeneity of variances, respectively. If ANOVA assumptions were met, variance analysis was performed; otherwise, non-parametric tests (Kruskal-Wallis and Wilcoxon) were used. Statistical analysis was performed using Minitab 17.0 (Minitab Inc., Pennsylvania, USA).

3. Results

There was a significant difference ($P < 0.05$) in plasma sodium values only in the animals of the ESDext group at T8h compared to T0h (Table 2). Urinary sodium concentrations ($urNa^+$) also differed significantly over time in both groups ($P < 0.05$) (Table 3). There was an increase in $urNa^+$ levels from T0h (end of the dehydration period), reaching the highest values at T8h in animals from both the ESDext and ESMalt groups.

Table 2. Mean values and standard deviations of sodium, potassium, chloride, ionized calcium, magnesium, phosphorus, urea, and creatinine in the blood of dogs dehydrated by water restriction and subjected to two continuous-flow enteral fluid therapy protocols.

Parameters	Groups	Evaluation times			
		T-12h	T0h	T4h	T8h
Sodium (mmol L^{-1})	ESMalt	147 ± 3.1^{Aa}	145.4 ± 5.9^{Aa}	149 ± 1.9^{Aa}	149 ± 0.7^{Aa}
	ESDext	146.8 ± 3^{Aab}	145.2 ± 2.1^{Ab}	148.2 ± 1.1^{Aab}	149.8 ± 1.3^{Aa}
Potassium (mmol L^{-1})	ESMalt	4.2 ± 0.4^{Ab}	4.7 ± 0.1^{Aa}	4.3 ± 0.2^{Aab}	3.7 ± 0.2^{Ac}
	ESDext	4.1 ± 0.2^{Aab}	4.5 ± 0.2^{Aa}	4 ± 0.3^{Ab}	3.8 ± 0.2^{Ab}
Chloride (mmol L^{-1})	ESMalt	112.9 ± 3.8^{Ab}	121.3 ± 2.2^{Aa}	118 ± 3.1^{Aa}	111.8 ± 1.5^{Ab}
	ESDext	114.8 ± 8.1^{Abc}	119.5 ± 4.3^{Aa}	116.2 ± 2.3^{Aabc}	112.2 ± 1.6^{Ac}
Ionized calcium (mmol dL^{-1})	ESMalt	1.4 ± 0.1^{Aa}	1.4 ± 0.1^{Aa}	1.4 ± 0.0^{Aa}	1.4 ± 0.0^{Aa}
	ESDext	1.4 ± 0.1^{Aa}	1.4 ± 0.1^{Aa}	1.4 ± 0.1^{Aa}	1.4 ± 0.0^{Aa}
Magnesium (mg dL^{-1})	ESMalt	2.0 ± 0.6^{Aa}	2.0 ± 0.6^{Aa}	1.8 ± 0.6^{Aa}	1.3 ± 0.5^{Aa}
	SEdext	2.2 ± 1^{Aa}	2.2 ± 1^{Aa}	2.2 ± 0.7^{Aa}	2.1 ± 0.9^{Aa}
Phosphorus (mg dL^{-1})	ESMalt	3.4 ± 0.2^{Aa}	3.8 ± 0.5^{Aa}	2.9 ± 0.5^{Aa}	3.1 ± 0.6^{Aa}
	ESDext	3.4 ± 0.3^{Aa}	3.6 ± 0.5^{Aa}	3.1 ± 0.8^{Aa}	3.4 ± 0.5^{Aa}

Continued

Urea (mg dL^{-1})	ESMalt	31.2 ± 7.1^{Aab}	36.8 ± 2.8^{Aa}	27.2 ± 4.5^{Ab}	22 ± 4.1^{Ab}
	ESDext	32.8 ± 2.2^{Aab}	35.4 ± 3.6^{Aa}	30.4 ± 5.4^{Aab}	24 ± 7.5^{Ab}
Creatinine (mg dL^{-1})	ESMalt	1.2 ± 0.1^{Aa}	1.2 ± 0.1^{Aa}	1.1 ± 0.1^{Aa}	1.1 ± 0.1^{Aa}
	ESDext	1.1 ± 0.2^{Aa}	1.1 ± 0.1^{Aa}	1.1 ± 0.1^{Aa}	1.1 ± 0.1^{Aa}

Mean values followed by different capital letters in the same column or by different lowercase letters in the same row differ from each other ($P < 0.05$).

Plasma potassium levels also showed significant differences ($P < 0.05$) in both groups throughout the experimental period (Table 2), with an increase observed at the end of the water and food fasting period (T0h). At T4h and T8h, potassium concentrations gradually decreased in both groups. Urinary potassium values (urK^+) remained unchanged in the ESDext group throughout the experimental period ($P > 0.05$), despite a slight decrease observed at T8h (Table 3). Conversely, in the ESMalt group, urK^+ levels gradually declined at T4h and reached significantly lower values at T8h ($P < 0.05$).

Table 3. Mean values and standard deviations of urine specific gravity and volume (mL), sodium (mmol L^{-1}), potassium (mmol L^{-1}), chloride (mmol L^{-1}), calcium (mg dL^{-1}), magnesium (mg dL^{-1}), urea (mg dL^{-1}), and creatinine (mg dL^{-1}) in the urine of dogs dehydrated by water restriction and subjected to two continuous-flow enteral fluid therapy protocols.

Parameters	Groups	Evaluation times			
		T-12h	T0h	T4h	T8h
Urine specific gravity	ESMalt	1035.6 ± 3.6^{Aab}	1040 ± 0.4^{Aa}	1015.6 ± 15.6^{Ab}	1011.6 ± 1.7^{Ab}
	ESDext	1024.4 ± 11.3^{Ab}	1040 ± 0.0^{Aa}	1017.8 ± 6.3^{Ab}	1018 ± 7.6^{Ab}
Urine volume	ESMalt	10 ± 0.0^{Ac}	36 ± 37.1^{Abc}	479.2 ± 237^{Aab}	684 ± 337^{Aa}
	ESDext	10 ± 0.0^{Ac}	70 ± 134.2^{Abc}	366 ± 139^{Aab}	484 ± 268.2^{Aa}
Urine sodium ($urNa^+$)	ESMalt	40.4 ± 49.1^{Ab}	91 ± 41.4^{Aab}	73.8 ± 53^{Aab}	118.2 ± 34.3^{Aa}
	ESDext	30.2 ± 35.6^{Ab}	84.8 ± 45.1^{Ab}	76 ± 36^{Ab}	167.2 ± 61.3^{Aa}
Urine potassium (urK^+)	ESMalt	99 ± 34.8^{Aab}	158 ± 72.1^{Aa}	50.8 ± 62.8^{Aab}	19.4 ± 7.2^{Ab}
	ESDext	113.6 ± 43^{Aa}	97.4 ± 40^{Aa}	100 ± 31.24^{Aa}	77 ± 33.9^{Aa}
Urine chloride ($urCl^-$)	ESMalt	136.8 ± 83.9^{Aa}	232.8 ± 121.5^{Aa}	166.4 ± 125.9^{Aa}	177.6 ± 53.4^{Aa}
	ESDext	99.2 ± 73.2^{Aa}	156.8 ± 110.4^{Aa}	204 ± 90.2^{Aa}	247.2 ± 37.7^{Aa}
Urine calcium ($urCa^{++}$)	ESMalt	$2,3 \pm 1,0^{Aa}$	$2,1 \pm 0,7^{Aa}$	$2,7 \pm 1,7^{Aa}$	$3,7 \pm 2,4^{Aa}$
	ESDext	2.2 ± 1^{Aa}	2.8 ± 1.6^{Aa}	2.65 ± 1.2^{Aa}	2.9 ± 1^{Aa}
Urine magnesium ($urMg^{++}$)	ESMalt	2.4 ± 1.7^{Aa}	3.7 ± 2.2^{Aa}	1.7 ± 1.5^{Aa}	1.4 ± 0.6^{Aa}
	ESDext	1.7 ± 1.9^{Aa}	2.5 ± 2.7^{Aa}	1.5 ± 1.4^{Aa}	1.2 ± 0.7^{Aa}
Urine urea (urUrea)	ESMalt	3959 ± 1062^{Aa}	5717 ± 765^{Aa}	1816 ± 1451^{Ab}	758 ± 240^{Ab}
	ESDext	2969 ± 1762^{Aab}	5878 ± 1264^{Aa}	1616 ± 725^{Ab}	1302 ± 880^{Ab}
Urine creatinine (urCrea)	ESMalt	106 ± 91.3^{Aa}	156.5 ± 128.3^{Aa}	67.3 ± 61.5^{Aa}	32 ± 16.6^{Aa}
	ESDext	152 ± 114.5^{Aab}	243 ± 38.6^{Aa}	71.5 ± 27.8^{Ab}	88 ± 45.9^{Aab}

Mean values followed by different capital letters in the same column or by different lowercase letters in the same row differ from each other ($P < 0.05$).

Similar to sodium and potassium, plasma chloride concentrations differed significantly ($P < 0.05$) between groups throughout the experimental period (**Table 2**). Following the water and food fasting period (T0h), a slight increase was observed, followed by a significant decrease at T8h ($P < 0.05$). Urinary chloride ($urCl^-$) concentrations did not differ significantly ($P > 0.05$) between or within groups over time (**Table 3**).

The values for ionized calcium, magnesium, phosphorus, and creatinine in the blood showed no significant differences ($P > 0.05$) between or within groups during the experimental period (**Table 2**). Similarly, urinary calcium ($urCa^{2+}$) and magnesium ($urMg^{2+}$) concentrations did not differ significantly between or within groups (**Table 3**). Urinary creatinine (urCrea) in the ESDext group decreased at T4h, while serum and urinary urea levels decreased in both groups at T4h and T8h (**Tables 2** and **3**).

Urinary volume and specific gravity exhibited similar behavior and showed no significant difference between groups ($P > 0.05$). However, beginning at T4h, there was a significant reduction in urinary specific gravity ($P < 0.05$) and a significant increase in urinary volume ($P < 0.05$) in both groups (**Table 3**).

4. Discussion

A slight increase in plasma sodium levels was observed in animals from both groups, beginning at T4h and peaking at T8h. This increase, which persisted after the end of fluid therapy (T8h), is attributable to the sodium content in the electrolyte solution administered. The presence of carbohydrates in the two electrolyte solutions may have contributed to this increase, as according to Reece *et al.* [11], the presence of these substances in the electrolyte solution enhances sodium absorption. Despite the slight increase in plasma sodium values in the animals of both groups, they remained within the reference range established by Dibartola [2].

There was also an increase in urinary sodium ($urNa^+$) in animals from both groups, reaching the highest values at T8h (**Table 3**). This increase indicates that in dehydrated animals without serum or plasma hyponatremia, the amount of sodium in the rehydration electrolyte solution should not exceed five grams per liter or should be even less. Sodium excess and edema can develop iatrogenically from the excessive administration of sodium-containing fluids in patients with severely compromised renal function. The most serious risk is cerebral hemorrhage, caused by the shrinkage of brain cells due to high sodium concentrations, leading to vascular rupture. Additionally, hypernatremia can cause muscle spasms, seizures, and coma [12]. This statement is corroborated by the composition of the enteral electrolyte solution for rehydrating children recommended by the WHO/UNICEF since 2005, which contains 4.3 grams of sodium per liter. Lima *et al.* [13] in calves and Dias *et al.* [14] in adult horses also recommend using 4 g of NaCl per liter of solution. Perhaps in dogs 4 grams of NaCl per liter of solution is the appropriate amount. However, in patients with dehydration and decreased serum

or plasma sodium, the amount of sodium in the electrolyte solution can be increased, depending on the intensity of hyponatremia.

The decrease in blood volume resulting from the period of water restriction was the cause of the slight increase in plasma potassium observed at T0h (**Table 2**). This elevation may be associated with hypovolemia, which, according to Carlson and Bruss [15], can lead to metabolic acidosis. As a compensatory mechanism, hydrogen ions (H^+) are transported into cells for buffering, while potassium ions are translocated to the extracellular space to maintain electroneutrality, thereby increasing serum or plasma potassium concentrations. Given that the increase at T0h was mild, it is assumed that any acidosis present was also of low intensity. This assumption is supported by the slight decrease in base excess reported in these animals by Dantas *et al.* [16].

The decrease recorded in plasma potassium at T4h and T8h in the animals from both groups (**Table 2**) was the result of blood volume expansion, associated with the absorption of carbohydrates present in the enteral electrolyte solution, which causes potassium to be translocated into the cells, as mentioned by Tannen [17].

Even though the carbohydrate-containing enteral electrolyte solutions used in this trial contained 1 g of KCl per liter, they were unable to increase blood potassium concentration (**Table 2**), indicating that in cases of prolonged fluid therapy and especially in animals with hypokalemia, monitoring blood potassium is essential, as it may be necessary to increase the potassium concentration in the solution.

Urinary potassium values (urK^+) remained unchanged throughout the experimental phase in the animals in the ESDext group ($P > 0.05$), despite a slight decrease recorded at T8h (**Table 3**). On the other hand, those given the ESMalt treatment showed a decrease in urinary potassium values at T4h, which became significant at T8h ($P < 0.05$). As the animals in the ESMalt group had the highest urinary volume (**Table 3**), it is possible that this greater urine output caused dilution and increased potassium excretion.

The increase in plasma chloride observed in both groups at T0h was caused by hypovolemia resulting from food and water restriction (**Table 2**). The presence of hyperchloremia without a proportional increase in sodium is consistent with hyperchloremic metabolic acidosis [18]. As in the present study, at T0h, the lowest plasma sodium values were detected in the ESMalt group (145.4 ± 5.9 mmol L^{-1}) and the ESDext group (145.2 ± 2.1 mmol L^{-1}), it can be inferred that this event occurred. The chloride concentration increases in this type of acidosis due to proportionally lower losses of chloride compared to bicarbonate and increased renal chloride reabsorption in response to decreased blood bicarbonate [19].

Following the initiation of enteral electrolyte solution administration, a decrease in plasma chloride levels was observed at T4h, although not statistically significant ($P > 0.05$), which persisted until T8h ($P < 0.05$), returning to baseline values (**Table 2**). This result demonstrated that the enteral electrolyte solutions contained adequate amounts of chloride. This is supported by the behavior of uri-

nary chloride ($urCl^-$), which did not show any difference ($P > 0.05$) between or within groups during the experimental phase (Table 3).

The values of ionized calcium, magnesium, phosphorus, and creatinine in the blood showed no significant differences ($P > 0.05$) between or within groups (Table 2). Furthermore, these values remained within the normal range recommended by Kaneko *et al.* [20]. Similarly, there were no differences ($P > 0.05$) in urinary calcium and magnesium concentrations between groups and across time points (Table 3).

As shown in Table 2, the urea values showed no differences between groups ($P > 0.05$), but differences were detected over time ($P < 0.05$). As a result of the dehydration protocol, urea levels increased at T0h in both groups. Using a dehydration protocol, Balbinot *et al.* [21] also found elevated urea levels, justifying the increase as a consequence of dehydration.

In the animals that received the maltodextrin-containing solution (ESMalt), there was a significant decrease ($P < 0.05$) in urea at T4h and T8h, while in the ESDext group, the decrease was only significant at T8h. This suggests that the ESMalt solution provided faster blood volume expansion, as the effect was observed earlier (T4h) compared to the ESDext group (T8h). This may be explained by the lower osmolarity of ESMalt (215 mOsm L^{-1}) compared to ESDext (243 mOsm L^{-1}), as mentioned by Rautanen *et al.* [22] and Ribeiro Filho *et al.* [23], who stated that the lower the osmolarity, the greater the intestinal absorption and resulting expansion of volemia. Therefore, enteral electrolyte solutions with lower osmolarity are more effective for rehydration and electrolyte replacement, as they attract less water into the intestinal lumen and are absorbed in greater volume.

Urinary urea concentrations differed ($P < 0.05$) only over time in both groups (Table 3), showing a similar pattern: values increased at T0h due to dehydration, then decreased at T4h and T8h as a result of rehydration and increased urine production, which led to urea dilution. Urinary creatinine showed a similar pattern, increasing at T0h and decreasing at T4h and T8h (Table 3), confirming the effects of the dehydration protocol and rehydration.

Urine specific gravity and volume differed ($P < 0.05$) only when comparing timepoints within each group. At T0h, both groups showed increased specific gravity due to water deprivation, validating the dehydration protocol. After fluid therapy (T4h), there was a marked decrease in specific gravity, persisting through T8h in both groups ($P < 0.05$). Concurrently, urinary volume increased significantly from T4h to T8h ($P < 0.05$) in both groups, likely due to increased glomerular filtration resulting from blood volume expansion. Although not statistically different between groups ($P > 0.05$), the ESMalt group produced 30% more urine at T4h and 41% more at T8h compared to ESDext, confirming superior intestinal absorption of the maltodextrin solution.

The behavior of these two variables confirms that absorption and blood volume expansion induced by hypotonic electrolyte solutions administered enterally are rapid and effective, supporting the findings of Reineke *et al.* [8] and Forbes *et al.*

[9], who reported satisfactory results using this modality for gastroenteritis, renal disorders, and preoperative care in veterinary medicine.

It can be concluded that hypotonic enteral electrolyte solutions containing maltodextrin (ESMalt) and dextrose (ESDext) promote an increase in urinary volume and a reduction in urine specific gravity, with a more pronounced diuretic effect observed in the ESMalt group. In addition, no adverse effects were observed in animals from either group. These findings support the efficacy of hypotonic enteral electrolyte solutions supplemented with energy sources as valuable therapeutic options for hydration in dogs.

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Data

The datasets generated for this study are available on request to the corresponding author.

Conflicts of Interest

The authors have no competing or conflict of interest in submitting this article.

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