

HYPERKALEMIA IN FREE-RANGING WHITE-TAILED DEER (*ODOCOILEUS VIRGINIANUS*)

Elizabeth M. Stringer,^{1,4} Suzanne Kennedy-Stoskopf,¹ M. Colter Chitwood,²
Jeffrey R. Thompson,³ and Christopher S. DePerno²

¹ North Carolina State University, College of Veterinary Medicine, 4700 Hillsborough Street, Raleigh, North Carolina 27606, USA

² North Carolina State University, Department of Forestry and Environmental Resources, Fisheries, Wildlife, and Conservation Biology, 110 Brooks Avenue, Raleigh, North Carolina 27607, USA

³ SAS Institute Inc., World Headquarters, SAS Campus Drive, Cary, North Carolina 27513, USA

⁴ Corresponding author (email: Betsy_Stringer@ncsu.edu)

ABSTRACT: Sixty adult and yearling female white-tailed deer (*Odocoileus virginianus*) were collected in July 2008 ($n=30$) and March 2009 ($n=30$) from eastern North Carolina as part of a population health assessment. During July 2008, standard serum analyses revealed hyperkalemia in all deer sampled. In March, the effect of processing time as a possible source of the hyperkalemia was investigated. For a subset of deer ($n=10$), blood tubes were centrifuged and processed at four time points (0, 30, 60, and 120 min) postcollection. Delayed centrifugation and plasma separation did not affect potassium (K^+) concentration over time, indicating that a shift in intracellular K^+ did not occur and the hyperkalemia was not due to improper sample handling. Potassium levels were negatively correlated with age and varied across collection periods. Also, K^+ levels were positively correlated with glucose and not correlated with creatine kinase (CK). No single variable indicated a strong enough relationship to explain the hyperkalemia in the study.

Key words: Centrifugation, creatine kinase, glucose, hyperkalemia, *Odocoileus virginianus*, potassium, pseudohyperkalemia, white-tailed deer.

INTRODUCTION

Many reference ranges have been reported for potassium (K^+) in free-ranging cervids (Table 1; Wilber and Robinson, 1958; White and Cook, 1974; Seal et al., 1978; Kie et al., 1983; Waid and Warren, 1984; DeLiberto et al., 1989; DelGiudice et al., 1992). The International Species Information System (ISIS), widely used as a reference for captive populations, reports a mean K^+ of 4.3 ± 0.9 milliequivalents (mEq)/l ($n=61$; ISIS, 2002) for white-tailed deer (*Odocoileus virginianus*). Although K^+ levels are relatively constant for most mammals, reports of hyperkalemia in free-ranging white-tailed deer occur in the literature, with values reaching as high as 14.0 mEq/l (Wilber and Robinson, 1958).

A large intracellular pool of K^+ exists, compared with that of extracellular K^+ (Engelking, 2004), and a shift in K^+ distribution can increase plasma K^+ concentration. As K^+ levels reach 5.5 mEq/l, arrhythmias can occur due to an increase in the rate of repolarization of the action

potential and worsen as levels rise to 7.0 mEq/l (Smith et al., 1985). Hyperkalemia of 8–9 mEq/l can cause fluid shifts and profound electrocardiographic abnormalities, including ventricular fibrillation and asystole (Smith et al., 1985; Jopson and Fennessy, 1992; Carlson, 1997). Hyperkalemia most often occurs following a change in renal excretion of K^+ , as in acute renal failure, Addison's disease, or hypovolemia (Carlson, 1997).

Hyperkalemia also is observed with hemolysis or prolonged storage of blood samples prior to separation of serum from the erythrocytes (Carlson, 1997). A study in humans showed that delayed centrifugation and serum separation was one of the main causes of pseudohyperkalemia, a condition in which the in vitro blood K^+ concentration is elevated, but the in vivo concentration is normal (Kapoor et al., 2009). Species with high intraerythrocyte K^+ levels (e.g., domestic horses, pigs, and cattle) can have significant errors in their blood K^+ value due to improper handling of samples (Carlson, 1997). Although the

TABLE 1. Published reference values (mEq/l) for K^+ in free-ranging white-tailed deer.

Reference	K^+ Range (mEq/l)	Mean K^+ (mEq/l)	No.	Method of capture	Sample
Wilber and Robinson, 1958	7.9–14.0	10.4	10	Neck shot	Plasma
White and Cook, 1974	4.2–9.9	6.8	79	Neck shot	Serum
Seal et al., 1978	n/a ^a	4.5	32	Chemical immobilization	Serum
Kie et al., 1983	n/a	8.3	48	Shot	Serum
Waid and Warren, 1984	n/a	7.7	74	Neck shot	Serum
DeLiberto et al., 1989	7.1–11.2	n/a	55	Neck or head shot	Serum
DelGiudice et al., 1992	4.3–9.2	n/a	59	Chemical immobilization	Serum

^a n/a = Data not in reference.

intraerythrocytic K^+ content has not been described for cervids, it is likely similar to other ungulates. Further, K^+ can be released from leukocytes or platelets following clot formation, especially in animals with a marked leukocytosis or thrombocytosis (Smith et al., 1985; Reimann et al., 1989; Carlson, 1997). Therefore, it has been suggested that K^+ concentration is determined most accurately by measuring plasma rather than serum (Reimann et al., 1989).

Improper sample handling can include incomplete clot formation prior to centrifugation, inadequate centrifugation, improper separation of serum from the clot during pipetting, prolonged storage of blood samples prior to separation of serum or plasma from the erythrocytes, sample contamination, or inadvertent thawing and refreezing of samples. Hence, the objectives of this study were to investigate whether time to centrifugation impacted K^+ concentration and to determine if the severe hyperkalemia observed in a population of white-tailed deer was a pseudo-hyperkalemia or a true hyperkalemia.

MATERIALS AND METHODS

The study was conducted at Hofmann Forest, a 78,000-acre (31,565-ha) tract of contiguous pocosin habitat in the coastal plain of North Carolina (34°50'N, 77°18'W). The forest was owned and managed by North Carolina State Natural Resources Foundation and intensively managed for loblolly pine (*Pinus taeda*) production. Pocosins are characterized by deep, acidic, nutrient deficient,

sandy, or peat soils (Richardson et al., 1981). Dominant vegetation (Christensen et al., 1981; Richardson et al., 1981) included loblolly pine and pond pine (*Pinus serotina*) and a dense shrub layer comprised of titi (*Cyrilla racemiflora*), sweetbay (*Magnolia virginiana*), redbay (*Persea borbonia*), inkberry (*Ilex glabra*), and greenbrier (*Smilax* spp.). Undisturbed pocosin habitat contains low crude protein, phosphorus, boron, and calcium, which could affect deer body maintenance (Sossaman and Weber, 1974).

Sixty adult, female white-tailed deer (*O. virginianus*) were collected in July 2008 ($n=30$) and March 2009 ($n=30$) as part of a population health assessment. Collections were approved by the North Carolina Wildlife Resources Commission and the North Carolina State University Institutional Animal Care and Use Committee (08-082-O).

Deer were head shot with high-powered rifles at night. Within minutes of collapse, blood was collected via cardiac puncture. A vacutainer system was used to collect blood into ethylenediaminetetraacetic acid (EDTA) and then serum tubes. Samples were stored on ice at a central processing station until hematocrits were measured and serum tubes were centrifuged. Blood processing occurred from 2 to 6 hr after collection. Serum and whole blood samples were stored frozen at -80°C . Serum was later analyzed by Antech Diagnostics (Chapel Hill, North Carolina, USA) on an Olympus AU5400 (Olympus America Inc., Melville, New York, USA), which used ion-selective probes for K^+ measurement.

Because July blood samples were stored for 2 to 6 hr prior to processing, the authors hypothesized that delayed centrifugation was the cause of the hyperkalemia due to a shift in intracellular K^+ . Therefore, in March 2009, K^+ was analyzed over time in a subset of 10 deer (eight females, two males). Plasma was used for this portion of the study, so the first tube

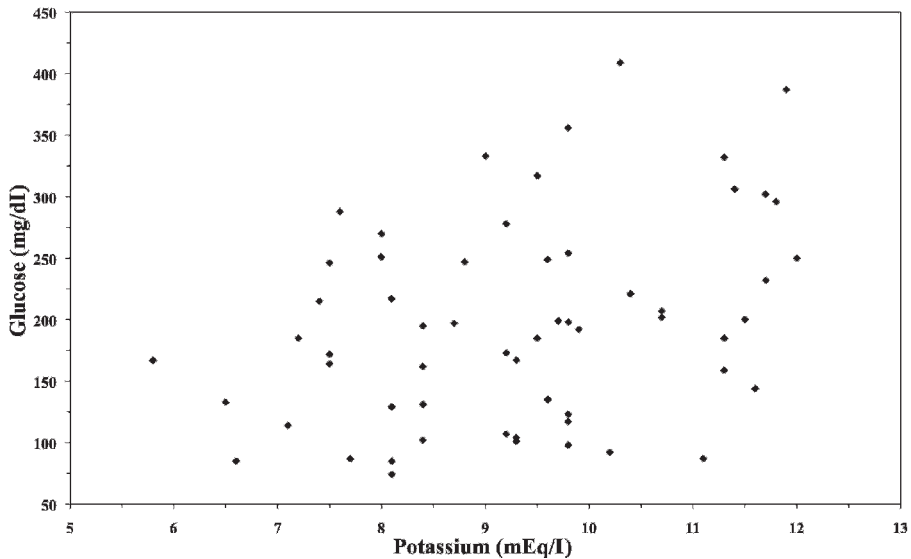


FIGURE 1. Relationship between serum K^+ (mEq/l) and glucose (mg/dl) in 59 white-tailed deer collected at Hofmann Forest, North Carolina, July 2008 and March 2009.

could be centrifuged immediately in the field. Lithium heparin (i.e., plasma) tubes were filled prior to EDTA and serum tubes. The initial heparin tube was spun within 10 min of the animal's death (i.e., time zero). Subsequent heparin tubes were centrifuged and processed at 30, 60, and 120 min postcollection. All plasma samples were separated immediately after centrifugation, frozen, and later analyzed by Antech Diagnostics. For all March deer sampled, EDTA and serum tubes were processed within 2 to 6 hr of collection as previously described.

All carcasses were necropsied to evaluate body condition and examined for evidence of infectious disease (Chitwood, 2010). Age of the deer was estimated by cementum annuli analysis of extracted incisor teeth (Matson's Lab, Milltown, Montana, USA).

Statistical analyses were performed using JMP 7.0 computer software (SAS Institute, Cary, North Carolina, USA). Nonparametric Spearman correlation coefficients were used to compare age, K^+ , creatine kinase (CK), and glucose values. To analyze K^+ over time, a nonparametric Page test for ordered alternatives was conducted, and a *t*-test was used to examine effects of seasonality. Alpha (α) was set at 0.05.

RESULTS

We collected 59 blood samples, 30 in July and 29 in March. A sample was not

obtained from one deer in March due to difficulty reaching the animal. In July, one blood sample was considered hemolyzed by the diagnostic lab, with a 1+ hemolysis grading, but was retained for analysis.

Hyperkalemia (as defined by the ISIS reference range) was observed in all deer sampled. Serum K^+ values averaged 9.3 ± 1.6 mEq/l (range=5.8–12.0 mEq/l). Nine animals required a second shot to kill. To determine if delay in death increased K^+ levels, these deer were examined as a subgroup; their K^+ averaged 9.4 ± 1.6 mEq/l (range=7.4–12.0 mEq/l).

The relationship of K^+ to glucose, as a possible indicator of acute stress, was examined. Glucose values averaged 197 ± 82.6 mg/dl (range=74–409 mg/dl) and were similar to published references for captive and free-ranging white-tailed deer (captive: 142 ± 59 mg/dl, $n=84$ [ISIS, 2002]; free-ranging: 60–320 mg/dl, $n=118$ [Klinger et al., 1986]). Potassium levels were positively correlated with glucose ($r=0.323$, $P=0.013$; Fig. 1).

Creatine kinase values averaged 262 ± 266 international units (IU)/l (range=63–1,883 IU/l) and were within published reference ranges for captive and

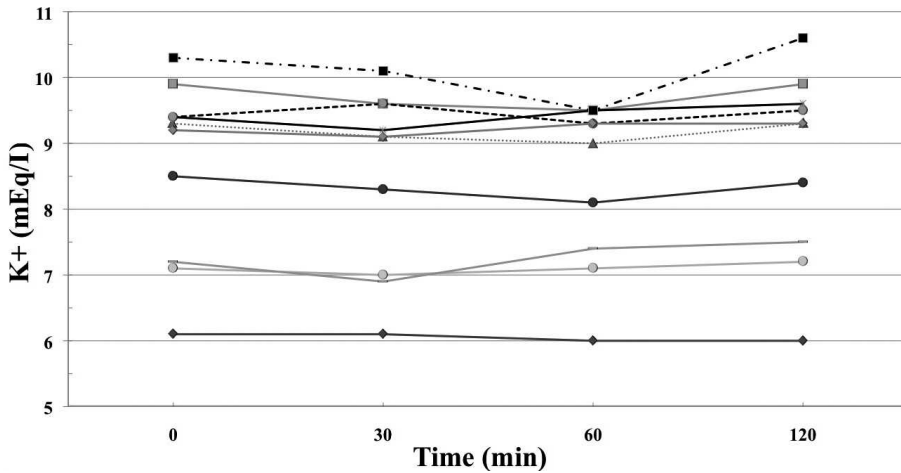


FIGURE 2. Plasma potassium values in mEq/l at time zero, 30, 60, and 120 min in a subset of white-tailed deer ($n=10$) collected at Hofmann Forest, North Carolina, March 2009.

free-ranging white-tailed deer (captive: 611 ± 988 IU/l, $n=42$ [ISIS, 2002]; free ranging: 513 ± 105 IU/l, $n=40$ [Seal et al., 1978]). Creatine kinase did not correlate to K^+ ($r=0.1713$, $P=0.195$).

Age of collected deer averaged 4 yr (range=1–10 yr). Potassium was negatively correlated with age ($r=-0.2731$, $P=0.0363$). Also, K^+ differed between seasons ($P=0.037$); July K^+ averaged 9.7 ± 1.6 mEq/l (range=5.8–12.0 mEq/l, $n=30$), and March K^+ averaged 8.9 ± 1.3 mEq/l (range=6.5–11.9 mEq/l, $n=29$). Regardless of the seasonal difference, both means were elevated.

In the subset of deer from March ($n=10$), plasma K^+ values over time averaged 8.6 ± 1.3 mEq/l (range=6.0–10.6 mEq/l) but did not increase between time zero and 120 min (Page test: $L=258.5$, $P=0.105$; Fig. 2).

DISCUSSION

Potassium is the most abundant intracellular cation (Engelking, 2004) with 98% of the exchangeable K^+ located intracellularly (Carlson, 1997). The resulting membrane potential is critical for cardiac and neuromuscular excitability, and hyperkalemia decreases this potential (Carlson,

1997). Extracellular K^+ concentrations are tightly regulated, and an acute rise stimulates secretion of insulin, epinephrine, and aldosterone, which subsequently activates sodium potassium adenosine triphosphatase (Na^+/K^+ ATPase) to allow movement of K^+ back into cells (Engelking, 2004). This quickly returns the extracellular K^+ concentration to normal, thus the tight reference ranges traditionally reported in mammals. Why or how this mechanism might differ in free-ranging cervids is unknown.

In our study, delayed centrifugation and plasma separation did not affect the K^+ concentration over time, indicating that no shift in intracellular K^+ occurred. Additionally, collection into lithium heparin tubes reduced any contribution of K^+ released from platelets during clot formation (Reimann et al., 1989). Thus, the white-tailed deer at Hofmann forest exhibited a true hyperkalemia, not a pseudohyperkalemia due to improper sample handling. Further, EDTA-coated blood tubes can contaminate subsequently filled tubes when using a vacutainer system (Calam and Cooper, 1982). The K_2 -EDTA tubes were used in this study. However, heparin tubes were filled prior to EDTA tubes, as recommended by Calam and

Cooper (1982), so the plasma hyperkalemia was not an artifact of EDTA contamination.

Another potential artifactual source of hyperkalemia is leakage from cells postmortem (Jashnani et al., 2010). Potassium concentrations rise in plasma and vitreous humor after death, and the latter is used to estimate the postmortem interval in human forensic investigations (Jashnani et al., 2010). Data from Zaugg and Kinsel (1997) showed that postmortem vitreous K^+ concentrations can be used to estimate the time of death in elk and mule deer. However, in the current study, the time from death to sample collection was less than 10 min, so postmortem leakage is unlikely to be the cause of the hyperkalemia.

Hyperkalemia can occur transiently in animals with massive muscle necrosis or in vigorous short-term exercise (Smith et al., 1985; Carlson, 1997). Creatine kinase, a group of isoenzymes, increases dramatically during anaerobic conditions associated with muscle exertion. The enzyme utilizes stored creatine phosphate to provide phosphates to convert adenosine diphosphate to ATP and provide energy for muscle contractions (Cardinet, 1997). The deer in this study were head shot at night while standing still and were likely under little stress prior to death. Creatine kinase values were within published reference ranges, and CK was not significantly correlated to K^+ . Therefore, significant muscle damage was unlikely to be the cause of hyperkalemia in this free-ranging white-tailed deer population.

Acute stress due to handling, capture technique, or shock is often reported as the underlying cause of hyperkalemia in exotic hoofstock (Wilber and Robinson, 1958; Kock et al., 1987; DeLiberto et al., 1989; Jopson et al., 1997). However, results are variable and conflicting. Marco and Lavin (1999) detected no difference in K^+ levels between physically restrained or chemically immobilized red deer, but hyperkalemia was attributed to stress or

physical exertion. Kocan et al. (1981) compared physically restrained and chemically immobilized white-tailed deer and were unable to detect an effect on K^+ . They concluded that only minor changes in blood parameters occurred if the capture conditions induced only minimal excitement. DeNicola and Swihart (1997) noted that cortisol levels were lowest for head-shot deer, intermediate for chemically immobilized deer, and highest for deer captured with drop-nets.

In the absence of cortisol values, elevated glucose levels have been used to evaluate stress indirectly. Glucocorticoids convert stored glycogen in the liver and muscle to glucose (Kaneko, 1997). Blood glucose concentrations reflect the equilibrium between the rates of entry and removal of glucose in circulation. This is a dynamic process, and many factors influence glucose levels such as recent food intake and exercise. Consequently, blood glucose levels vary constantly, which explains the relatively wide range of glucose concentrations that are accepted for normal reference ranges.

A natural physiologic relationship exists between K^+ and glucose. Cells shrink during periods of high extracellular glucose concentration, causing K^+ extrusion from the cells (Engelking, 2004). Deer in this study were not hyperglycemic, based on mean reference values, so physiologic stress was probably not a factor. However, we detected a weak positive correlation between K^+ and glucose. A clinically significant relationship between the two variables is tenuous at best and probably does not explain the elevated levels of K^+ in circulation.

Studies on seasonality of K^+ in deer are contradictory. DeLiberto et al. (1989) reported that K^+ values were highest in the summer, while several studies have shown no seasonal variation in K^+ (Seal et al., 1978; Kie et al., 1983; Waid and Warren, 1984; Jopson et al., 1997). Del-Giudice et al. (1992) investigated seasonal effects on blood parameters of white-

tailed deer and reported the most probable cause of hyperkalemia during winter was a partial renal shutdown associated with reduced circulatory volume. We detected a difference in K^+ values between the July and March groups. However, this seasonal difference was not clinically significant as both means were considered elevated.

Hyperkalemia can be potentiated by the kidney's mechanism for conserving nitrogen, causing a secondary elevation of K^+ in circulation (Smith et al., 1985; Jopson and Fennessy, 1992; Jopson et al., 1997). White-tailed deer, like cattle and sheep, can recycle urea and utilize it for protein synthesis (Robbins et al., 1974). Urea is recycled from the blood, back to the gastrointestinal tract, and is hydrolyzed by bacteria to ammonia and carbon dioxide (Robbins et al., 1974). Caribou (*Rangifer tarandus*) respond to a reduction in protein intake by reducing their glomerular filtration rate (GFR), presumably to retain nitrogen (Valtonen, 1979). If white-tailed deer respond in a similar manner, the decreased GFR could account for the observed hyperkalemia, which may be evident during times of inadequate nitrogen intake (Robbins et al., 1974). Thus, the hyperkalemia in this study may reflect the inherent poor soil conditions and forage quality of the pocosin habitat. The relative absence of hyperkalemia in captive white-tailed deer may be a reflection of their access to a high-quality diet year-round.

Many factors may contribute to the hyperkalemia seen in this population of deer, including renal excretion, seasonality, poor forage quality, and method of capture. However, no single variable in our study indicated a strong enough relationship to explain the hyperkalemia, including time to sample centrifugation. How white-tailed deer tolerate these severe elevations in K^+ , which could cause profound cardiac arrhythmias and death in other species, is unknown. Further work is needed to investigate the underlying cause

of the hyperkalemia, how deer tolerate the elevations, and the clinical significance of the condition.

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