New Zealand Society of Animal Production online archive

This paper is from the New Zealand Society for Animal Production online archive. NZSAP holds a regular annual conference in June or July each year for the presentation of technical and applied topics in animal production. NZSAP plays an important role as a forum fostering research in all areas of animal production including production systems, nutrition, meat science, animal welfare, wool science, animal breeding and genetics.

An invitation is extended to all those involved in the field of animal production to apply for membership of the New Zealand Society of Animal Production at our website www.nzsap.org.nz

View All Proceedings   **Next Conference**   **Join NZSAP**

The New Zealand Society of Animal Production in publishing the conference proceedings is engaged in disseminating information, not rendering professional advice or services. The views expressed herein do not necessarily represent the views of the New Zealand Society of Animal Production and the New Zealand Society of Animal Production expressly disclaims any form of liability with respect to anything done or omitted to be done in reliance upon the contents of these proceedings.

Plasma potassium – an indicator of protein catabolism in deer?

N.B. JOPSON, P.F. FENNESSY, AND J.M. THOMPSON[1]

AgResearch, Invermay Agricultural Centre, Private Bag 50034, Mosgiel, New Zealand.

ABSTRACT

Elevated plasma potassium (K$^+$) concentrations well outside the normal range for deer have been reported during the rut. The effects of season and nutrition on plasma K$^+$ concentration were examined in two experiments in farmed red and fallow deer. In Experiment 1 castrate and entire red stags were fed *ad libitum* on pasture and blood sampled fortnightly over 11 months. Except for a transitory peak at the beginning of Experiment 1, red entires and castrates showed no abnormal plasma K$^+$ concentrations over the 11 month period. In Experiment 2, a castrate group of fallow bucks were fed to lose a similar amount of live weight as entires over the rut. When plasma K$^+$ concentration was expressed as a function of changes in muscle weight there was a significant negative relationship (P<0.05) in the castrates. There was no relationship in the entires, although the measure of change in muscle weight could have been confounded by changes in water content of the musculature over the rut. The occurrence of hyperkalaemia in deer was variable. Further work is required to clarify the conditions that lead to its expression.

Keywords: red deer; fallow deer; potassium; hyperkalaemia; fasting; muscle catabolism.

INTRODUCTION

Plasma potassium (K$^+$) concentrations in deer generally lie between 3.5 and 5.4 mmol/l (calculated from mean ± 95% confidence intervals; Wilson and Pauli, 1983). In other mammalian species elevated plasma K$^+$ concentrations (hyperkalaemia) produce electrocardiographic abnormalities that may result in cardiac arrest (Tasker, 1980). McAllum (1985) reported elevated plasma K$^+$ concentrations in wild-captured red deer suffering from post-capture myopathy. Jopson and Fennessy (1992) reported hyperkalaemia in red stags during the rut and postulated that this was a function of protein catabolism in years of poor pre-rut nutrition. Thus the elevated levels in these red stags during the rut may indicate an adaptation to periods when plasma K$^+$ rises above normal levels, but the nature and function of such an adaptation is unknown.

Therefore the hypothesis (Jopson and Fennessy, 1992) that hyperkalaemia in red deer was a result of muscle catabolism over the rut was tested in two experiments. Experiment 1 examined the seasonal pattern of plasma K$^+$ in castrate and entire red deer (*Cervus elaphus*) fed *ad libitum* on pasture. Experiment 2 investigated the relationship of plasma K$^+$ concentration to muscle catabolism in castrate and entire fallow deer (*Dama dama*) that underwent a substantial live weight loss using computed tomography (CT) to define changes in the musculature.

MATERIALS AND METHODS

Experiment 1: Eight two year old red deer stags (mean live weight 127.6 kg, SD 8.9) were randomly allocated to castration and entire groups. Castrations were performed on 7 November 1989. The stags were group fed *ad libitum* on pasture throughout the experiment and weighed and blood sampled fortnightly for 11 months (7 November 1989 to 9 October 1990).

Experiment 2: Eight adult fallow deer bucks (mean live weight 70 kg, SD 11) were randomly assigned to two treatment groups, namely entire bucks fed *ad libitum* through the rut (n=4, ENT) and castrate bucks (castrated on 1 February 1991) fed *ad libitum* until 2 April and then fed at the level consumed by the entires in the previous week (n=4, castrate restriction, CR). The intention was that CR bucks underwent an equivalent live weight loss to the ENT bucks. However CR bucks failed to lose as much weight as ENT bucks and therefore, intake for CR bucks was further restricted to 1.9 kg DM/head/week from 9 May for a further four weeks. Blood samples were collected weekly from 26 February until 3 June 1991. Bucks were CT scanned to estimate body composition on five occasions (Jopson *et al.,* 1997).

Blood sampling and electrolyte measurement: The animals were mechanically restrained and blood samples collected by jugular venepunture into lithium heparinised tubes. Blood samples were stored at 4°C for no more than 30 minutes before centrifugation to ensure minimal K$^+$ leakage from red blood cells (on CT scanning weeks the time between collection and processing exceeded 30 minutes and these samples were not analysed for K$^+$ concentration). Plasma samples were stored at -18°C until assayed.

Plasma K$^+$ concentrations were determined for Experiment 1 using a flame photometer (Radiometer, FLM3 Flame Photometer, Copenhagen, Denmark). For Experiment 2, K$^+$ concentrations were measured using an AVL 984 Electrolyte Analyser (AVL Biomedical Instruments, AL8207 Schaffhausen, Switzerland), which operated on the principle of ion-selective electrodes.
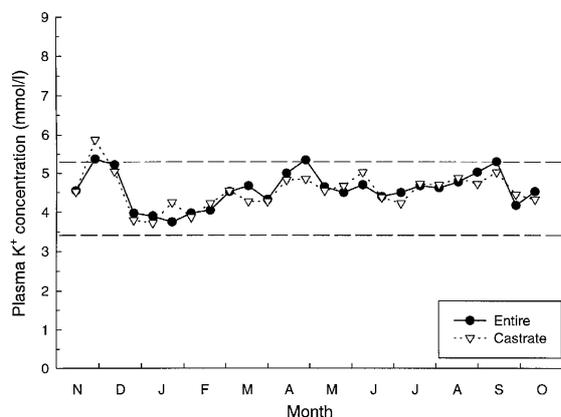
[1] Animal Science Department, University of New England, Armidale, NSW 2351, Australia.

Statistical analysis: In Experiment 2 the mean rate of change in muscle (g/day) was calculated by the increment in muscle weight on time for individual animals over the 3 or 4 week period between CT scannings. Mean K^+ concentrations were calculated over the corresponding periods. Analysis of K^+ concentration used a mixed model which contained terms for treatment, rate of change in muscle and the first order interaction. Animal within treatment was included as a random effect.

RESULTS

Experiment 1: Plasma K^+ concentration (Figure 1) only rose above the normal range (indicated by dashed lines) on one occasion throughout the entire experiment. For entire stags this measure peaked at 5.9 mmol/l on 28 November, but returned within the normal range at the next sampling. This peak coincided with a transitory peak in plasma K^+ on the second and third samples for the castrate stags. For the remainder of this experiment, plasma K^+ levels were stable at normal levels for both treatment groups.
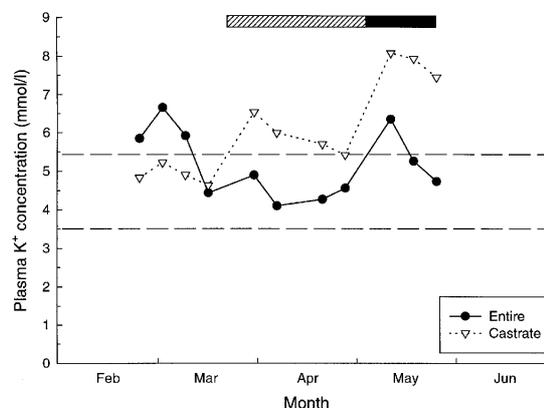
FIGURE 1: Seasonal variation in plasma K^+ concentration in entire and castrated red deer stags. Dashed lines indicate the normal range for plasma K^+ in red deer (mean ± 95% confidence limits; Wilson and Pauli, 1983).

FIGURE 2: Plasma K^+ concentration in *ad libitum* fed entire and restricted castrate fallow bucks. Castrates were fed to lose an equivalent amount of live weight as entires over the rut. Dashed lines indicate the normal range for plasma K^+ in red deer. Hatched bar indicates the group-pair feeding period. Solid bar indicates the restriction period applied only to castrates.

FIGURE 3: Mean plasma K^+ concentration relative to mean daily change in muscle for *ad libitum* fed entire and restricted castrate fallow bucks. Castrates were fed to lose an equivalent amount of live weight as entires over the rut. Dashed lines indicate the normal range for plasma K^+ in red deer. Regression lines were estimated for castrate and entire deer from a model which included treatment, muscle change, treatment *x* muscle change and animal(treatment).
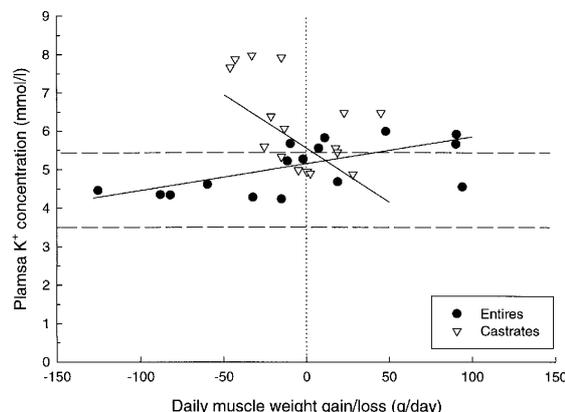

Experiment 2: The mean plasma K^+ concentration for ENT and CR bucks exceeded the normal range on several occasions (Figure 2). The ENT group mean was elevated over the first two weeks of blood sampling, but returned to near the normal range by week 3 (March). Thereafter plasma K^+ concentration remained within the normal range for the ENT group with the exception of a peak of 6.4 mmol/l on 14 May. Plasma K^+ concentrations in CR bucks were within the normal range during *ad libitum* feeding, but rose above this when undergoing live weight loss due to feed restriction. Concentrations reached 6.8 mmol/l in late March and approached nearly twice the normal plasma K^+ concentration in mid May.

When plasma K^+ concentrations were analysed as a function of muscle accretion or mobilisation, there was a significant interaction (P<0.05) between change in muscle weight and treatment, whereby CR bucks with higher rates

of muscle loss had the higher plasma K^+ concentrations (Figure 3). In contrast, there was no relationship (P>0.05) between muscle change and plasma K^+ concentration in entire bucks over the range of muscle change observed.

DISCUSSION

The transitory peaks observed in castrates and entires at the start of Experiment 1 were consistent with a short term behavioural response to the sampling procedure. Severe stresses (Rose *et al.,* 1970; Hofmeyr *et al.,* 1973; Hirche *et al.,* 1980) have been shown to cause elevations in plasma K^+ concentrations in other mammalian species. After the first three samplings, the stags appeared to habituate to the stress of the blood sampling procedure and plasma K^+ concentrations fell within the normal range. In this experiment there was clearly no seasonal trend in

plasma K$^+$ concentrations indicating that the hyperkalaemia reported through the rut by Jopson and Fennessy (1992) was perhaps not a regular feature of the rut. Rather, it was most likely a function of the specific circumstances that prevailed with those particular stags at that time.

The results in Experiment 2 clearly reveal hyperkalaemia in deer as reported by Jopson and Fennessy (1992), although it was restricted to the CR group. When expressed as a function of muscle catabolism, ENT bucks lost more muscle than CR bucks without displaying hyperkalaemia. The hypothesis that the hyperkalaemia was a function of extreme protein catabolism was challenged, when results were examined in isolation. However, previous studies have reported an appreciable increase in live weight in red stags going into the rut after food intake had declined (Fennessy *et al.,* 1997). Much of this increase in live weight may be accounted for by an increase in water content of the musculature over the rut (Tan and Fennessy, 1981). This phenomenon would appear to be specific to entire stags as in the same experiment the change in water content was not evident in the musculature of castrates. The difference in water content was only reported for the 'steroid responsive' muscles, but was of sufficient magnitude to account for the difference in muscle weight change reported in ENT bucks. Therefore, the rate of change in muscle weight may not have accurately reflected the accretion or catabolism of protein. In the CR bucks, where changes in muscle weight were unlikely to be confounded by water content, there was a clear relationship between catabolism of muscle and hyperkalaemia. It is possible that ENT bucks may not have experienced sufficient protein catabolism to trigger the hyperkalaemia. Clearly this explanation needs to be tested in deer and other species undergoing severe protein catabolism.

The extended period of hyperkalaemia in CR bucks prompts two questions. Firstly, why does the renal system not clear the excessive amounts of K$^+$ from the plasma? One hypothesis is that the high concentrations of plasma K$^+$ are a consequence of a mechanism for the conservation of nitrogen by the kidneys. Evidence in support of this postulate is that reindeer kidneys have been shown to respond to low protein intake with a fall in the glomerular filtration rate (GFR), presumably to retain nitrogen in the form of urea for recycling (Valtonen, 1979). A reduction in GFR could result in the elevated plasma K$^+$ if it was being released into the system through muscle catabolism.

Another hypothesis relates to the structure of the kidney itself. While data for red and fallow deer are limited, reindeer have been shown to have a thick renal cortex in relation to medulla depth, and require ten times the antidiuretic hormone to cause diuresis, when compared to sheep (Valtonen and Eriksson, 1977). These two factors indicate that reindeer, and potentially other deer species, have a poor ability to concentrate urine or excrete a solute load.

Secondly, how is the animal able to tolerate a hyperkalaemia that would potentially be lethal in other mammals? The answer to this question is unknown. It is tempting to speculate that the hyperkalaemia may be a relatively frequent occurrence in deer undergoing severe weight loss and so resulted in natural selection for tolerance to hyperkalaemia.

CONCLUSIONS

The present experiments did not support the hypothesis that hyperkalaemia was a natural occurrence in rutting males undergoing loss in muscle weight. However, there was one occurrence in castrates undergoing severe nutritional stress where plasma K$^+$ concentration was a good predictor of muscle loss. In entires, the relationship between K$^+$ concentration and muscle mobilisation may be confounded by changes in water content of the muscle. Stress at sampling may possibly confound the K$^+$ concentration in animals unaccustomed to the blood sampling procedure. The hypothesis that hyperkalaemia is associated with true protein catabolism requires testing.

ACKNOWLEDGEMENTS

REFERENCES

Fennessy, P.F.; Thompson, J.M.; Suttie, J.M. 1991. Season and growth strategy in red deer: Evolutionary implications and nutritional management. Pp 495-501. *In:* Wildlife production: Conservation and Sustainable Development, L.A. Renecker; R.J. Hudson (eds.), University of Alaska, Fairbanks.

Hirche, H.; Schumacher, E.; Hagemann, H. 1980. Extracellular K$^+$ concentration and K$^+$ balance of the gastrocnemius muscle of the dog during exercise. *Pflugers Archiv* **387:** 231-237.

Hofmeyr, J.M.; Louw, G.N.; du Preez, J.S. 1973. Incipient capture myopathy as revealed by blood chemistry of chased zebras. *Madoqua (Series 1)* **7:** 45-50.

Jopson, N.B.; Fennessy, P.F. 1992. A pattern of plasma potassium concentrations in adult red deer stags. Pp 429-435 *In:* The Biology of Deer, R.D. Brown (ed.), Springer-Verlag, New York.

Jopson, N.B.; Thompson, J.M.; Fennessy, P.F. 1997. Tissue mobilisation rates in male fallow deer (*Dama dama*) as determined by Computed Tomography: the effects of natural and enforced food restriction. *Animal Science* (accepted).

McAllum, H.J.F. 1985. Stress and postcapture myopathy in red deer. Pp. 65-72 *In:* Biology of Deer Production, P.F. Fennessy; K.R. Drew (eds.), The Royal Society of New Zealand, Bulletin 22.

Rose, L.I.; Carroll, D.R.; Lowe, S.L.; Peterson, E.W.; Cooper, K.H. 1970. Serum electrolyte changes after marathon running. *Journal of Applied Physiology* **29:** 449-451.

Tan, G.Y.; Fennessy, P.F. 1981. The effect of castration on some muscles of red deer (*Cervus elaphus L.*). *New Zealand Journal of Agricultural Research* **24:** 1-3.

Tasker, J.B. 1980. Fluids, electrolytes and acid-base balance. Pp 401-446 *In:* Clinical Biochemistry of Domestic Animals, J.J. Kaneko (ed.), Academic Press, New York.

Valtonen, M. 1979. Renal responses of reindeer to high and low protein diet and sodium supplement. *Journal of the Scientific Agricultural Society of Finland* **51:** 387-419.

Valtonen, M. and Eriksson, L. 1977. Responses of Reindeer to water loading, water restriction and ADH. *Acta Physiologica Scandinavia* **100:** 340-346.

Wilson, P.R.; Pauli, J.V. 1983. Blood constituents of farmed red deer (*Cervus elaphus*). II. Biochemical values. *New Zealand Veterinary Journal* **31:** 1-3.