Coccidian oocyst and nematode egg counts of free-ranging African buffalo (*Syncerus caffer*) in the Kruger National Park, South Africa

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ABSTRACT

Faecal specimens collected in the Kruger National Park from 103 African buffaloes (*Syncerus caffer*) up to 1 year old and 283 buffaloes older than 1 year were examined for the presence of coccidian oocysts and nematode eggs. Most specimens from animals older than 1 year had negative coccidian oocyst counts. Positive specimens from younger animals had significantly higher coccidian oocyst counts than those from older animals. No such difference was found for nematode egg counts.

Key words: African buffalo, coccidia, Kruger National Park, nematodes, South Africa, *Syncerus caffer.*

INTRODUCTION

The gastrointestinal parasites of free-ranging buffalo (*Syncerus caffer*) in the Kruger National Park (KNP; between 22°31′ and 25°31′S, 30°45′ and 32°00′E) have been documented by several authors, but quantitative studies are few[1,4,14]. Coccidiosis has been mentioned as affecting buffaloes in the KNP, but no details were given[14]. However, a group of wild-caught buffalo calves held in pens for experimental purposes in the KNP developed severe coccidiosis and required treatment (V de Vos, South African National Parks, pers. comm., 1996). Coccidiosis in livestock usually occurs in young animals (generally <6 months old) in intensive production systems; older animals normally develop a degree of immunity[13].

The coccidian species infecting African buffaloes have not been documented[4]. Although coccidia tend to be host-specific, this is not unequivocally so. It is known that at least some of the approximately 15 *Eimeria* species described from domestic cattle can also infect water buffaloes (*Bubalis bubalis*)[9] and American bison (*Bison bison*)[10].

Nine nematode species have been recorded from the gastrointestinal tract of buffaloes in the KNP: *Agriostomum gorgonis, Cooperia fülleborni, Haemonchus bedfordi, Haemonchus contortus, Oesophagostomum radiatum, Parabronema skrjabini, Tricho-*

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strongylus axei, Trichostrongylus deflexus and *Trichuris globulosa*[1,4]. With the exception of schistosomes, indications are that free-ranging buffaloes may not harbour large helminth burdens[4]. Three adult buffaloes from the KNP harboured a mean of 1361 (range 148–2017) and a calf 244 gastrointestinal nematodes. The 3 adults yielded 100 eggs per gram of faeces (EPG) but none were present in the calf's faeces (J Boomker, I G Horak, Faculty of Veterinary Science, University of Pretoria, pers. comm., 1999). Six adult buffaloes from Hluhluwe-Umfolozi Park, KwaZulu-Natal, South Africa, harboured a mean of 2136 (range 40–8603) and 2 calves 40 and 1800 gastrointestinal nematodes; all faecal specimens had negative egg counts (J Boomker, I G Horak, Faculty of Veterinary Science, University of Pretoria, pers. comm., 1999). Considering the diversity of nematode species collected and the size of the host, the individual burdens are insignificant and probably do not constitute a threat to the buffalo's health.

Bovine tuberculosis has recently been reported in buffaloes and other mammal species in the KNP[2,7]. A randomised epidemiological survey was carried out during September and October 1998 to assess the prevalence and distribution of infection in the KNP buffalo population. Collection sites were spread throughout the Park. Faeces collected from these animals were used primarily to assess levels of infection with coccidia, but nematode egg counts were also made.

Determining individual nematode loads was beyond the scope of this investigation.

MATERIALS AND METHODS

The buffaloes collected were assigned to well-defined age classes[11]. Faecal specimens were collected from 386 buffaloes culled during the survey and examined for the presence of coccidian oocysts and helminth eggs. Specimens from buffaloes up to 1 year old ($n = 103$) and those older than 1 year ($n = 283$) were analysed separately.

A modified McMaster technique was employed for quantification[12]. Four grams of faeces were placed in a beaker and 56 mℓ of 40 % sucrose solution were added and the contents stirred well. The mixture was poured into an electric food blender and thoroughly mixed in 3 short bursts (1 second each), at 2 second intervals. The mixture was poured into a beaker, 5–6 drops of amyl alcohol were added (to eliminate air bubbles), the mixture was swirled and allowed to settle for 2 minutes (until air bubbles no longer rose to the surface). The mixture was again swirled and using a Pasteur pipette and bulb, 2 McMaster slides were filled with the mixture, and allowed to stand for 2 minutes. Coccidian oocysts and nematode eggs in each of the 3 chambers were counted under the ×10 objective of a standard microscope. The total number of oocysts and eggs counted, divided by the number of chambers (6) and multiplied by 100 gave the number of oocysts (OPG) or eggs (EPG) per gram of faeces.

RESULTS

The results are summarised in Table 1. The majority of specimens from buffaloes ≤1 year old (64/103) and >1 year old (166/283) yielded no coccidian oocysts. Younger animals tended to have higher counts; the differences were significant ($\chi^2 = 16.177215$; df = 4, $P = 0.00279$) (the counts for OPG ranges >251 were pooled). The mean oocyst count of positive specimens from animals ≤1 year old was 363.3 (SD = 703.6, maximum 2867), while that from animals >1 year old was 102.1 (SD = 226.4, maximum 1800).

The differences between nematode egg

Table 1: **The number and percentage** (in brackets) **of faecal specimens collected from African buffaloes up to and older than 1 year, respectively, containing specific numbers of coccidian oocysts per gram of faeces** (OPG) **and helminth eggs per gram of faeces** (EPG).

	Age class			
	≤1 year old (*n* = 103)		>1 year old (*n* = 283)	
OPG/EPG range	**Oocysts**	**Eggs**	**Oocysts**	**Eggs**
0	44 (42.7)	33 (32.0)	166 (58.7)	113 (39.9)
1–50	31 (30.1)	36 (35.0)	82 (29.0)	76 (26.9)
51–100	9 (8.7)	12 (11.7)	14 (4.9)	32 (11.3)
101–250	9 (8.7)	17 (16.5)	14 (4.9)	40 (14.1)
251–500	2 (1.9)	3 (2.9)	4 (1.4)	19 (6.7)
501–1000	2 (1.9)	1 (1.0)	1 (0.4)	3 (1.1)
>1000	6 (5.8)	1 (1.0)	2 (0.7)	–

counts of specimens from the 2 age classes were not significant (χ^2 = 4.164391, df = 4, P = 0.384215) (the counts for EPG ranges >251 were pooled). The mean EPG of positive specimens from animals ≤1 year old was 94.5 (SD = 172.9, maximum 1150), while that from animals >1 year old was 120.6 (SD = 143.4, maximum 867).

DISCUSSION

In livestock, a few oocysts per gram of faeces usually indicate a low-grade infection, 50–100 OPG a moderate infection and 100–1000 OPG or more a high-grade infection[13]. By these criteria, mean oocyst counts from both younger and older buffaloes would indicate a high-grade infection. The mere presence of oocysts does not necessarily indicate clinical coccidiosis, and the OPG counts reported may not be of significance in free-ranging buffaloes. The situation would change completely if these animals were captured and placed in a confined area.

Quantification of coccidian infections in free-ranging wildlife appears to be rare. Oocyst counts in blue wildebeest (*Connochaetes taurinus*) calves (1–7 months of age) in the KNP generally were considerably higher than in older animals[6]. The lower counts in older animals are probably an indication of immunity, but may also be due to the larger volume of faeces excreted by those animals and hence dilution of the oocysts. Relatively low counts were recorded in American bison in Montana, USA, where oocysts were recovered from only 5 of 31 bison[10]. The mean OPG in yearling bison heifers was 123 (range 8–621) and 26.4 (range 0–169) in yearling bulls.

The nematode egg counts are more difficult to interpret. Negative counts have been reported from buffaloes known to be infected with nematodes (J Boomker, I G Horak, Faculty of Veterinary Science, University of Pretoria, pers. comm., 1999).

Marked seasonal differences were found in faecal egg counts for impalas (*Aepyceros melampus*) from Nylsvley Nature Reserve, Northern Province: egg counts were at a high level from February to April, but a subsequent decrease was followed by a marked rise in November and December[5]. This coincided with marked seasonal inhibition in the development of the nematode species parasitising the impalas.

In blue wildebeest in the KNP, faecal egg counts were highest in 1–6-month-old animals, and the counts in 7–12-month-old animals were in turn higher than those in 13–18-month-old animals[6]. Although the decrease in faecal egg count in older animals may be due to increased faecal volume, it is more likely that it is due to increased resistance to infestation resulting in a decrease of egg output by female worms. This hypothesis is supported by the fact that, although the total accumulated burdens of the 3 major nematode parasites were reasonably similar during each 6-month period from birth until wildebeests reached 18 months of age, the faecal egg counts decreased progressively during each of the 3 periods.

Fluctuations in faecal egg counts in kudus (*Tragelaphus strepsiceros*) in the KNP were unrelated to the seasonal abundance of worms and little correlation (0.07) was found between faecal worm egg count and total worm burden[3]. The size of nematode burdens in wildlife should not be estimated on the basis of faecal egg counts[3], and as with livestock, autopsy remains the best method.[12]

Buffaloes are known to utilise large home ranges[8], which reduces the probability of ingesting infective stages of parasites. They do concentrate at waterholes, however, which may be the foci where infection occurs. The survey was carried out at the end of the dry season, when ambient temperatures were already high; these are the worst possible conditions for the survival of eggs and oocysts. Conversely, stress induced by poor quality grazing at that time of the year may make the buffaloes more susceptible to infection.

REFERENCES

1. Basson P A, McCully R M, Kruger S P, Van Niekerk J W, Young E, De Vos V 1970 Parasitic and other diseases of the African buffalo in the Kruger National Park. *Onderstepoort Journal of Veterinary Research* 37: 11–28
2. Bengis R G, Kriek N P J, Keet D F, Raath J P, De Vos V, Huchzermeyer H F A K 1996 An outbreak of bovine tuberculosis in a free-living African buffalo (*Syncerus caffer* Sparrman) population in the Kruger National Park: a preliminary report. *Onderstepoort Journal of Veterinary Research* 63: 15–18
3. Boomker J, Horak I G, De Vos V 1989 Parasites of South African wildlife. IV. Helminths of kudu, *Tragelaphus strepsiceros*, in the Kruger National Park. *Onderstepoort Journal of Veterinary Research* 56: 111–121
4. Boomker J, Horak I G, Penzhorn B L, Keet D F 1996 Parasites of African buffaloes: a documentation. In Penzhorn B L (ed.) *Proceedings of a Symposium on the African Buffalo as a Game Ranch Animal*. Wildlife Group of the South African Veterinary Association, Onderstepoort: 90–103
5. Horak I G 1978 Parasites of domestic and wild animals in South Africa. X. Helminths in impala. *Onderstepoort Journal of Veterinary Research* 45: 221–228
6. Horak I G, De Vos V, Brown M R 1983 Parasites of domestic and wild animals in South Africa. XVI. Helminth and arthropod parasites of blue and black wildebeest (*Connochaetes taurinus* and *Connochaetes gnou*).

Onderstepoort Journal of Veterinary Research 50: 243–255

7. Keet D F, Kriek N P J, Penrith M-L, Michel A, Huchzermeyer H 1996 Tuberculosis in buffaloes (*Syncerus caffer*) in the Kruger National Park: spread of the disease to other species. *Onderstepoort Journal of Veterinary Research* 63: 239–244

8. Krüger J 1996 Socio-ecology of the African buffalo (*Syncerus caffer*): implications for game ranching. In Penzhorn B L (ed.) *Proceedings of a Symposium on the African Buffalo as a Game Ranch Animal.* Wildlife Group of the South African Veterinary Association, Onderstepoort: 1–5

9. Levine N D 1985 *Veterinary protozoology.* Iowa State University Press, Ames, Iowa

10. Penzhorn B L, Knapp S E, Speer C A 1994 Enteric coccidia in free-ranging American bison (*Bison bison*) in Montana. *Journal of Wildlife Diseases* 30: 267–269

11. Pienaar U de V 1969 Observations on the developmental biology, growth and some aspects of the population ecology of African buffalo (*Syncerus caffer*) in the Kruger National Park. *Koedoe* 12: 28–52

12. Reinecke R K 1983 *Veterinary helminthology.* Butterworths, Durban, Pretoria

13. Speer C A 1999 Coccidiosis. In Howard J L, Smith R A (eds) *Current veterinary therapy: food animal practice* (4th edn). W B Saunders, Philadelphia: 411–420

14. Young E, Van den Heever L W 1969 The African buffalo as a source of food and by-products. *Journal of the South African Veterinary Medical Association* 40: 83–88

Book review — Boekresensie

Breeding for Disease Resistance in Farm Animals (2nd edition)

Edited by R F E Axford, S C Bishop, F W Nicholas and J B Owen

1999. CABI Publishing, Wallingford, hard cover, 424 pp. Price £75 (US$140). ISBN 085199-325-7.

It needs to be said at the outset that the 20 chapters that make up this book are clearly written and reasonably comprehensive, albeit probably of more interest to specialists in infectious and parasitic diseases than general practitioners. On the other hand, for anyone (such as the reviewer) with the serious hope that genetic resistance to infectious diseases of animals holds imminent solutions to intractable modern-day problems, this volume makes sobering reading. The conclusion one reaches is that, although genetic resistance has much to offer in the longer term, the immediate prospects for real progress in disease control using this approach are much more limited. The reason is quite simply that not enough is known about the fundamental principles involved in the exploitation of what is, superficially, a basic observation, *i.e.* that resistance to infectious and parasitic diseases is generally just as heritable and influenced by quantitative trait loci (QTL) as are production traits.

This point is illustrated by scrapie, where it has long been known that different species (sheep and goats) and breeds within those species differ remarkably in their susceptibility to the disease. For that reason the genetics of scrapie have been extensively studied for many years. The frequencies of genetically-determined variation in the prion protein (PrP) of different breeds of sheep and their effects on scrapie prevalence are well known. This information has been used to alter PrP frequencies (and therefore the rate at which scrapie occurs in endemic situations) by the use of rams with appropriate genotypes. However, this approach may, conversely, result in unwanted effects, such as the loss of desirable breed characteristics, selection of rare scrapie strains that are not inhibited by the sheep genotype selected for, or a selective advantage for the agents maintained by 'carrier' sheep. According to Hunter in the chapter on transmissible spongiform encephalopathies, there is evidence from mouse and modelling studies for the existence of scrapie carriers. In practical terms, therefore, although sheep breeders are provided with new opportunities for controlling this much-studied disease, the longer-term implications are uncertain. Interestingly, cattle do not have the same heterogeneity in their PrP genes, and consequently cattle breeds do not vary noticeably in their susceptibility to bovine spongiform encephalopathy.

Most practical progress in breeding for genetic resistance seems to have been made in respect of parasitic conditions (*e.g.* helminthosis, ovine cutaneous myiasis and tick infestation in Australia) and production diseases. The latter comprise a complex of mostly undefined conditions that inhibit livestock productivity. For these diseases there is a growing realisation that the effective use of genetic resistance has to be integrated into a range of other control options to produce effective strategies.

The compendium is divided into 5 parts: principles and methods, parasites and vectors, bacteria, viruses and subviruses, and production diseases. Each part is made up of 3–5 chapters. The overview chapters on principles and methods (*e.g.* genetic maps, markers and QTLs; the immune system and the major histocompatibility complex) are more lucidly explained than is usually the case with this type of publication. Conversely, despite the wide range of animal diseases addressed, there is not much new in this volume and the impression is conveyed of another rehash. Certainly, fundamental issues that the book purports to address such as 'trying to lessen the impact of ecological perturbations involved in modern pharmaceutical intervention' are not dealt with seriously.

Nevertheless, for anyone with an interest in breeding for genetic resistance, and the principles underlying the approach, this book provides a good overview.

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