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Authors: Wolhuter, Julie, Bengis, Roy G., Reilly, Brian K., and Cross, Paul C.

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Clinical Demodicosis in African Buffalo (*Syncerus caffer)* in the Kruger National Park

Julie Wolhuter,[1] **Roy G. Bengis,**[2] **Brian K. Reilly,**[1] **and Paul C. Cross**[3,4,5] [1] Department of Nature Conservation, Tshwane University of Technology, Private Bag X 680, Pretoria 0001, South Africa; [2] Veterinary Investigation Centre, PO Box 12, Skukuza 1350, South Africa; [3] Northern Rocky Mountain Science Center, US Geological Survey, 229 AJM Johnson Hall, Bozeman, Montana 59717, USA; [4] Department of Ecology, Montana State University, Bozeman, Montana 59717, USA; [5] Corresponding address: (email: pcross@usgs.gov)

ABSTRACT: We investigated the relationship between prevalence and severity of clinical signs of *Demodex cafferi* infection in free-ranging African buffalo (*Syncerus caffer*) and other factors such as age, sex, pregnancy status, and concomitant infections with bovine tuberculosis (BTB), Rift Valley fever (RVF), and brucellosis (BA). Approximately half of 203 buffalo examined in this study had clinical signs of demodicosis (cutaneous nodules); younger age classes had the highest prevalence and severity of lesions ($\chi^2=21.4$, df=6, $P=0.0015$). Nodules were generally limited to the head and neck region, but in severe cases were present over the entire animal. We found no significant association between clinical severity of the *Demodex* infection and gender, pregnancy status, or infection with BTB, RVF, or BA.

Key words: African buffalo, co-infection, *Demodex cafferi*, Kruger National Park.

The parasitic mite, *Demodex cafferi*, is part of the normal skin fauna of most African buffalo (*Syncerus caffer*) (Dräger and Paine, 1980). However, in certain cases, the mites proliferate within the sebaceous glands of the hair follicle, causing the development of nodular or eczematous skin lesions (Kaufmann, 1996). *Demodex* spp. mites are believed to be relatively benign, although there have been few studies on buffalo (Young and Van Den Heever, 1969; Basson et al., 1970). In cattle, demodectic mange, caused by *Demodex bovis*, is well documented; although the infection may affect the quality of hides for leather production, it is not considered to be of clinical importance. *Demodex* spp. mites have been causally related to secondary pyoderma and sepsis in dogs, and the same may be true for other host species.

There is a single published study that focused on the epidemiology of *Demodex* infection in a free-ranging buffalo population; the study was conducted in Botswana in the late 1970s (Dräger and Paine, 1980). Dräger and Paine (1980) described the typical nodular appearance of this parasitic condition in buffalo and also described severe cases wherein aggregations of confluent nodules resulted in localized skin swelling and secondary bacterial infections. Overall prevalence of clinical signs of infection reported in the Botswana study was 28% ($n=193$). Prevalence of *Demodex* nodules was highest in younger age classes (<2 yr of age), but no significant gender predilection was reported.

The objective of this current study in the Kruger National Park, South Africa was to document and describe the prevalence and intensity of the infection within this subpopulation of buffalo and to compare the results with those found in Botswana. A second objective was to investigate the possible relationship between prevalence and severity of clinical signs caused by *D. cafferi* and other factors such as age, gender, pregnancy status, and concomitant infections with bovine tuberculosis (BTB), Rift Valley fever (RVF), and brucellosis (BA). We hypothesized that co-infections may impact the host immune system and potentially affect the prevalence or severity of *Demodex* lesions.

Demodex nodules have a characteristic appearance and anatomic profile. To

confirm etiology, smears of the expressed contents of the distended sebaceous glands were examined. Biopsy samples for histopathology were taken, whereby the sebaceous lesion was excised with an elliptical, full-thickness skin incision and was closed by routine suturing. Microscopic examination of the mites confirmed the species as *D. cafferi,* the same species described in the Botswana buffalo. Reference specimens were submitted to the Agricultural Research Institute in Pretoria, South Africa (accession number AcY 08/327).

Our study included 203 buffalo captured from May 2003 to August 2004 in two regions of the Kruger National Park: Satara (24°23′S, 31°46′E) and Lower Sabie (25°07′S, 31°55′E). Study animals were dart-anesthetized using a combination of etorphine hydrochloride (M99® Novartis, Isando, South Africa) and azaperone (Stresnil®, Janssen Animal Health, Halfway House, South Africa). After sampling and identifiably marking study animals, anesthesia was reversed using diprenorphine hydrochloride (M5050®, Novartis, Isando, South Africa) (Bengis, 1993). All capture and handling procedures were approved by the University of California at Berkeley (USA) Animal Care and Use Committee (Protocol #R217B).

For each buffalo, nodules on the left-hand side were counted through direct observation and palpation of the skin surface. Nodules were assigned to one of three anatomic regions; head and neck, shoulder and thorax, or abdomen and hindquarter. Pregnancy status of females >3 yr of age was determined by rectal palpation. Exposure to viral and bacterial agents was determined by a modified gamma interferon (IFNg) BOVIGAM™ Prionics AG, Zurich, Switzerland assay for BTB, by standard complement fixation and serum agglutination tests for brucellosis, and by enzyme-linked immunosorbent assay for RVF.

For statistical analysis, Pearson's chi-square tests were conducted using R version 2.5 (R Development Core Team, 2007). Due to issues with sample size, only main effects were tested. Animals were placed into age classes representing the calves and juvenile animals (0–2 yr), subadults (3–4 yr), prime adults (5–8 yr), and older adults (9+ yr) using tooth eruption, body size, and horn shape and wear (Grimsdell, 1973; Sinclair, 1977). In addition, the severity of demodicosis was graded as follows; zero, low (1–20 nodules), and high (20+ nodules).

Lesions in the Kruger buffalo subpopulation were similar to those described in Botswana buffalo by Dräger and Paine (1980). Dermal nodules were 5–20 mm in diameter and represented distended sebaceous glands in the hair follicles. They contained inflammatory cells in a matrix of collagen and numerous demodex mites in all stages of development. During microscopic inspection of expressed material from various lesions, it was found that, as the nodules aged, cells and parasites underwent degeneration and necrosis, forming an amorphous mass of debris; epithelial cells become keratinized. The prevalence of demodicosis was 49% (n=203; 95% CI=0.42, 0.56). No statistically significant difference in the prevalence of clinical demodicosis was detected between our two study areas (χ^2=0.037, df=1). Therefore, we combined data from both areas in subsequent statistical analyses.

There was an association between age class and the severity of infection (χ^2=21.4, df=6, P=0.0015; Fig. 1). Lesions were present on the head and neck region (44%; 95% CI=0.37, 0.51) more often than on the shoulder and thorax (13%; 95% CI=0.09, 0.18) or abdomen and hindquarters (11%; 95% CI=0.07, 0.16). Statistically, there appeared to be no gender predilection to infection status (χ^2=0.021, df=1, P=0.88) or severity (χ^2=0.595, df=2, P=0.74), and no significant association between pregnancy and the prevalence or severity of *Demodex* lesions (prevalence: χ^2=0.056, df=1, P=0.81, n=71; severity: χ^2=2.14, df=2,

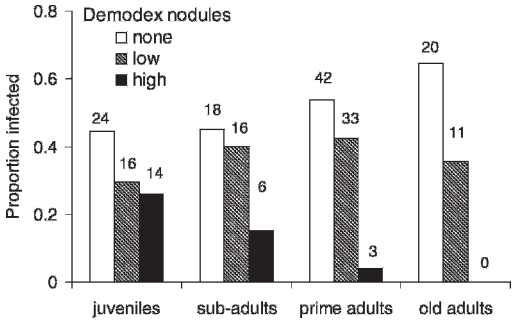

FIGURE 1. The proportion of 203 African Buffalo classified with zero (none), low, or high numbers of demodex-related nodules in each age class. Numbers above the bars indicate the sample size.

$P=0.34$) was seen. In addition, no significant correlation was detected between prevalence or severity of *Demodex* lesions and BTB ($n=196$), CA ($n=195$) or RVF ($n=117$) status; BTB and prevalence: $\chi^2=0.004$, df=1, $P=0.95$; BTB and severity: $\chi^2=0.096$, df=2, $P=0.95$; RVF and prevalence: $\chi^2=0.303$, df=1, $P=0.58$; RVF and severity: $\chi^2=2.06$, df=2, $P=0.35$; BA and prevalence: $\chi^2=0.001$, df=1, $P=0.97$; or BA and severity: $\chi^2=0.34$, df=2, $P=0.84$.

Our finding that the majority of nodules were recorded on the head and neck region in the young age class of buffalo is in agreement with the findings of Dräger and Paine (1980) and may reflect transmission of *Demodex* from mother to offspring during nursing, as described for other species (Muller and Kirk, 1969). However, the overall prevalence of clinical lesions in the Kruger buffalo study (49%) was much higher than those reported by Dräger and Paine (1980) in the Botswana study population (28%), which may be due to a number of factors such as climate, buffalo genetics, parasite strains, or sampling bias. The age-related decrease in prevalence and severity of *D. cafferi* lesions may be indicative of a progressive, successful immune response or of greater survival of animals that have superior genetic immunocompetence.

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