

BLOOD VALUES OF CAPTIVE BEIRA ANTELOPE (*DORCATRAGUS MEGALOTIS*) PRIOR TO AND DURING AN OUTBREAK OF FIBRINOUS PLEUROPNEUMONIA SYNDROME (FPFS)

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Abstract: Currently the only captive population of beira antelope (*Dorcatragus megalotis*) is held at the Al Wabra Wildlife Preservation, Qatar. An outbreak of a severe respiratory disease—fibrinous pleuropneumonia syndrome, most likely caused by *Mycoplasma ovipneumoniae*—led to a marked population decline. Reactive systemic inflammatory (AA) amyloidosis was noted as a chronic manifestation of the disease. Blood samples had been collected for biochemistry and hematology baseline values prior to the outbreak. Population-level changes were analyzed before and during the course of the outbreak in selected blood parameters (white blood cells [WBC], blood urea nitrogen [BUN], and creatinine). The annual population WBC increased and decreased concurrently with the population size, with a significant correlation between the two measures ($R = 0.92$; $P = 0.001$). Both BUN and creatinine values were higher during the outbreak. These values peaked at the same time as mortality, which was 1 yr after the WBC peak. These changes were interpreted as the transition from an acute disease with a primary respiratory manifestation into a chronic condition where renal amyloidosis led to chronic renal failure and death. Also, elevated liver values in diseased animals were attributed to amyloidosis. Parallels to a literature report on a lung disease complex caused by *M. ovipneumoniae* in bighorn sheep (*Ovis canadensis*) were found. Trends in population-level blood values of the beira antelopes implicate amyloidosis as a significant, long-term consequence of the putative *Mycoplasma* infection.

Keywords: Beira antelope, blood, *Dorcatragus megalotis*, fibrinous pleuropneumonia syndrome, *Mycoplasma*, population.

INTRODUCTION

Beira antelope are listed as vulnerable by the International Union for the Conservation of Nature Red List and are native to the Horn of Africa.¹⁹ Currently, the only captive population is maintained at Al Wabra Wildlife Preservation (AWWP) in Qatar.^{12,13} A severe respiratory epidemic that started in winter 2005–2006 led to a marked population decline. Clinical signs of respiratory disease including nasal discharge, coughing, and forced respiration, and rales on auscultation were noted.²⁵ Morbidity reached 100% in 2007 (and stayed at that level in future years) while mortality was 32.8 and 46.2% in 2006 and 2007, respectively.²¹ Daily monitoring of clinical signs demonstrated a seasonal influence, with more signs during colder months.² Gross

necropsy findings included fibrinous adhesions within the thoracic and the abdominal cavity, effusions, lung consolidations, liver consolidations, and congested or hemorrhagic kidneys.²⁵ Histologically, pulmonary emphysema, bronchopneumonia, and interstitial pneumonia as well as interstitial nephritis and fibrosis were diagnosed most often.²⁵ In animals that initially seemed to recover from the respiratory disease, immunohistochemistry revealed inflammatory (AA) amyloidosis in spleen, liver, kidneys, lymph nodes, and gut, suggesting chronic inflammatory processes.²¹ All samples of beira antelope tested negative for bovine herpesvirus 1 and bovine viral diarrhea virus.²⁰ Very low adenovirus-3 titers were detected. Positive antibody titers of parainfluenza-3 virus (PI-3) and bovine respiratory syncytial virus (BRSV) were attributed to a former vaccination with PI-3 virus, BRSV virus, and *Pasteurella haemolytica* (Bovigripp RSP plus®, Intervet, Unterschleissheim, 85716, Germany).²⁰

Initially, *Mycoplasma capricolum capripneumonia* (Mccp)—the etiologic agent of contagious caprine pleuropneumonia (CCPP)—was suspected to be the cause of the outbreak because it causes a similar pathology in domestic goats and other

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wild ungulates. An outbreak of CCPP in wild goats (*Capra aegagrus*), Nubian ibex (*Capra ibex nubiana*), Laristan mouflon (*Ovis orientalis laristanica*), and gerenuk (*Litocranius walleri*) at AWWP was confirmed 1 yr before the outbreak of respiratory disease in the beira antelope.¹ As a consequence to the Mccp outbreak in the other wild ungulates in 2005, all beira antelope were vaccinated against Mccp with a commercial vaccine (Caprivax®, Kenya Veterinary Vaccine Production Institute, Nairobi, 00200 Kenya). In a nonspecific PCR, 12 of 26 tissue samples from beira antelope (46%) were positive for *Mycoplasma* spp., but Mccp could not be detected in any sample. The disease syndrome was termed fibrinous pleuropneumonia syndrome (FPPS).¹⁴ At first, sequencing of the *Mycoplasma* spp. was not possible, but later PCR for *Mycoplasma ovipneumoniae* was repeatedly positive from tissue samples of deceased beira antelope.²¹ Isolation and culture of *M. ovipneumoniae* was possible in 2010. Treatment of affected animals with FPPS was symptomatic and supportive with different protocols of antibiotics, NSAIDs, glucocorticoids, mucolytics, para-immunity⁹ inducer, vitamins² and fluids.²⁵ In many cases it was possible to achieve a temporary improvement, but relapses were common and the animals were chronically affected for a prolonged time until they either died or were euthanized because of welfare reasons. After isolation and culturing *M. ovipneumoniae*, an autogenous vaccine was developed. Three young beira antelope were vaccinated but still died from the disease. After that, three newborn beira antelope were hand-reared with colostrum from vaccinated domestic goats. These animals did not develop clinical disease.

As part of the beira antelope health management plan, blood samples from clinically healthy beira antelope had been collected before the outbreak (2001–2005) for baseline values and are reported here. The current work is a retrospective evaluation correlating mortality with clinical pathology data. Population-level changes in selected blood parameters (white blood cell [WBC], blood urea nitrogen [BUN], creatinine) before and during the course of the epidemic were analyzed. The aim of this study was to examine whether trends in population blood values could show a correlation with population size and mortality and document the long-term consequences of the disease.

MATERIALS AND METHODS

Information about the population development and mortality (2001–2009) was generated from

the AWWP stock list. The mortality was calculated as the percentage of individuals that died within 1 yr out of all individuals that were ever alive within that year. Data on morbidity over time were not considered for this study because clinical signs were very variable; basically, clinical signs were observed, at different frequencies, in all animals at some time or other.^{2,25} Biochemistry baseline values were established from laboratory results of the individual records of beira antelope. Blood samples were taken using manual restraint or anesthesia with 3 mg/kg ketamine (Ketamidor®, Richter Pharma AG, Wels, 4600, Austria) and 0.08 mg/kg medetomidine (Domitor®, Pfizer GmbH, Karlsruhe, 76139, Germany) given either by hand injection or blow pipe.²² For manual restraint and hand injection, the animals were led through a narrow corridor into a transport box. The animals were restrained with a blanket to enable blood sampling from the jugular vein.¹² Only blood values from animals without documented clinical health problems at the time of blood collection (2001–2005) were used for the baseline values. If multiple blood samples from an individual were taken, average values from this individual were used.

For biochemistry values, blood samples collected into either lithium-heparin or serum clot tubes were centrifuged at 1.000 g for 10 min. Afterwards, the plasma-serum was removed and sent to the Vet Med Labor GmbH, Division of IDEXX Laboratories, Ludwigsburg, 71636, Germany. BUN, creatinine, glucose, total protein, aspartate aminotransferase (ASAT), gamma-glutamyltransferase (GGT), alkaline phosphatase, glutamate dehydrogenase (GLDH), creatine kinase, total bilirubin, cholesterol, β -hydroxybutyrate, sodium, chloride, potassium, calcium, inorganic phosphate, magnesium, and iron were analyzed with a Modular® Analytics EVO analyzer (Roche Diagnostics, Basel, 4070, Switzerland). Zinc and copper were measured using an atomic absorption spectrophotometer (Spectra AA 800, Varian, Mulgrave, Victoria, 3170 Australia). To measure selenium, the atomic absorption spectrophotometer Spectra AA 110–220/880 (Varian, Mulgrave, Victoria, 3170, Australia) was used.

Whole blood collected into ethylenediaminetetraacetic acid was used for hematology and was analyzed in the in-house laboratory. Packed cell volume was measured using a microhematocrit centrifuge at 765 g (Compur Microspin, Bayer Diagnostics, Leverkusen, 51368, Germany). A total WBC count was obtained by using BD Unopette System Tests (BD Franklin Lakes, New

Table 1. Biochemistry values from clinically healthy captive beira antelope (*Dorcatragus megalotis*) from Al Wabra Wildlife Preservation, Qatar. Ranges from domestic sheep and goats¹⁷ are provided for comparison.

Analyte	<i>n</i>	Mean (SD)	Min	Max	RI robust Box-Cox	Normal range ovine	Normal range caprine
Blood urea nitrogen (mg/dl)	22	18.48 (3.85) ^a	10.50	23.99	7.84–27.73	8.0–20.0	10.0–20.0
Creatinine (mg/dl)	20	1.00 (0.13) ^b	0.77	1.27	0.71–1.28	1.2–1.9	1.0–1.8
Glucose (mg/dl)	23	118.36 (34.05) ^c	49.18	174.92	46.84–192.76	50–80	50–75
Total protein (g/dl)	22	5.58 (0.41) ^d	4.95	6.3	4.73–6.51	6.0–7.9	6.4–7.0
Aspartate aminotransferase (U/L)*	21	69.0 (34.8) ^e	42	157.2	34.2–514.2	60–280	167–513
Gamma-glutamyltransferase (U/L)	20	25.8 (12.0) ^f	13.2	52.8	10.8–66.6	20–52	20–56
Alkaline phosphatase (U/L)	10	387.6 (362.4)	29.4	1129.8	5.4–2532	68–387	93–387
Glutamate dehydrogenase (U/L)*	19	5.4 (1.8) ^g	2.4	9.6	1.8–10.2	20	—
Creatine kinase (U/L)	20	207.6 (109.8)	42	432.6	34.2–514.2	8.1–12.9	08.8–8.9
Total bilirubin (mg/dl)	21	0.19 (0.09) ^h	0.10	0.40	0.08–0.41	0.1–0.5	0–0.1
Cholesterol (mg/dl)	20	70.98 (2 7.99)	39	136	33.90–157.78	52–76	80–130
β-hydroxybutyrate (mg/dl)*	18	2.92 (0.58)	1.75	4.08	1.672–4.211	5.73 ± 0.42	—

* Data not normally distributed.

^a Significantly different from values in ill animals (29.56 [21.19]; $P < 0.001$).

^b Significantly different from values in ill animals (2.36 [2.16]; P [nonparametric] = 0.002).

^c Significantly different from values in ill animals (145.24 [70.26]; $P = 0.015$).

^d Significantly different from values in ill animals (6.42 [1.29]; $P < 0.001$).

^e Significantly different from values in ill animals (303.3 [380.8]; P [nonparametric] < 0.001).

^f Significantly different from values in ill animals (103.6 [110.4]; P [nonparametric] < 0.001).

^g Significantly different from values in ill animals (922.1 [1421.6]; P [nonparametric] < 0.001).

^h Significantly different from values in ill animals (1.01 [2.67]; P [nonparametric] < 0.001).

Jersey, 07417, USA) and a hemocytometer (Paul Marienfeld GmbH & Co. KG, Lauda-Königshafen, 97922, Germany). Differential blood counts were made from blood smears stained with Diff Quick® Fixative Solution (Medion Diagnostics AG, Duedingen, 3186, Switzerland).

For statistical analysis, Reference Value Advisor (RefValAdv; National Veterinary School, Toulouse, 87614, France)¹⁰ an add-in for Microsoft Excel® (Microsoft Corporation, Redmond, Washington, 98052, USA) was used. This software considers the standard procedures accepted for the establishment of reference values including handling small sample sizes, Box-Cox transformation, outlier exclusion by Dixon-Reed and Tukey's tests, and reference interval calculation with the robust method.¹⁰ For several analytes the sample size was small ($n < 20$); therefore, the baseline intervals might not be representative. For hematology, only the mean, standard deviation, and minimum and maximum values were calculated because the sample size was smaller than 10.

To analyze differences between healthy animals and animals considered clinically ill, data of animals noted as clinically ill were collated (one value per individual; in case of multiple values available for an individual, an average was calculated). After testing for normal distribution by Kolmogorov-Smirnov test, differences between

baseline values and those of ill animals were either assessed by a parametric test (t -test) or a nonparametric test (Mann-Whitney U -test).

To analyze trends in blood values of the entire beira population at AWWP over time, available blood values of all animals of the stock list were included, independent of their health status at the time of blood collection. The proportion of animals sampled varied between years. If several blood samples had been taken from an individual during 1 yr, average values for that individual were calculated first; from these individual data the population average was subsequently calculated. Comparisons between years were performed using Kruskal-Wallis tests. If these tests indicated significant differences, individual pairwise comparisons were performed between years using Mann-Whitney U -tests with Sidak adjustment for multiple testing. Correlations between data per year were tested by Pearson's correlation coefficient (after confirming normal distribution). All statistical evaluations were performed in SPSS (21.0; SPSS Inc., Chicago, Illinois 60606-6412, USA), with the significance level set to 0.05.

RESULTS

Biochemical baseline values (Table 1) and baseline values for electrolytes and minerals (Table 2) were established for beira antelope.

Table 2. Electrolytes and mineral content values from clinically healthy captive beira antelope (*Dorcatragus megalotis*) from Al Wabra Wildlife Preservation, Qatar. Ranges from domestic sheep and goats are provided for comparison.^{17,28}

Analyte	<i>n</i>	Mean (SD)	Min	Max	RI robust Box-Cox	Normal range ovine	Normal range caprine
Sodium (mmol/L)	17	155.10 (1.81)	151.5	158	151.42–159.34	139–152	142–155
Chloride (mmol/L)	21	114.42 (3.08)	108	120	107.55–120.74	95–103	99–110.3
Potassium (mmol/L)	19	4.55 (0.31)	3.9	5.25	3.9–5.26	3.9–5.4	3.5–6.7
Calcium (mg/dl)	22	10.41 (0.69)	8.87	11.67	8.74–11.67	11.5–12.8	8.9–11.7
Phosphate (mg/dl)	23	8.36 (3.34)	3.00	13.32	1.21–15.70	5.0–7.3	4.2–9.1
Magnesium (mg/dl)	20	2.38 (0.34)	1.82	2.94	1.53–4.18	2.2–2.8	2.8–3.6
Zinc (µg/dl)	21	113.88 (39.82)	58.8	199.8	49.275–211.54	80–120	65–270
Copper (µg/dl)	23	101.53 (26.18)	61	161	55.99–165.35	70–200	70–120
Selenium (µg/L)	21	139.2 (33.10)	87	191	71.59–213.47	80–400	80–200
Iron (µg/dl)	16	218.19 (55.08)	132	310	109.40–354.52	166–222	—

Variations due to age and gender were not assessed because of the small sample size. For hematology, only a few values were available from clinically healthy animals; therefore, only mean and standard deviation were compiled (Table 3).

There were differences in WBC, BUN, and creatinine between baseline values and values of clinically ill animals. For all three analytes, values of clinically ill animals were significantly elevated (Tables 1, 3). Additionally, clinically ill animals had significantly lower total protein and significantly higher glucose, ASAT, GGT, GLDH, and total bilirubin values (Table 1) whereas other analytes investigated did not differ significantly between clinically ill and healthy animals.

Evaluation of the stock list revealed that the beira population at AWWP increased from 12 animals alive in 2000 to 73 animals in 2006. With the outbreak of FPPS the population size declined significantly to 26 animals in 2009. The annual population WBC increased and decreased concurrently with the population size (Fig. 1), with a significant correlation between the two measures ($R=0.92$; $P=0.001$) (Fig. 2). Although the peak in mortality was 1 yr later than the peak in popula-

tion and WBC (Fig. 1), mortality was significantly correlated with the WBC ($R=0.775$; $P=0.014$).

The peak in population BUN and creatinine was at the same time as the peak in mortality (Fig. 3); correspondingly, correlations between mortality and both BUN and creatinine were significant ($R=0.794$; $P=0.011$ and $R=0.721$; $P=0.028$ respectively) as well as the correlation between BUN and creatinine ($R=0.738$; $P=0.023$). This peak in mortality, population BUN, and creatinine occurred 1 yr later than the peak in population WBC (Figs. 1, 3); there were neither a correlation between WBC and BUN ($R=0.545$; $P=0.129$) nor between WBC and creatinine ($R=0.420$; $P=0.260$). A Kruskal-Wallis test indicated a significant difference for WBC ($P<0.001$) and creatinine ($P=0.002$) between years but not for BUN ($P=0.263$). Pair-wise comparisons with adjustment for multiple testing indicated that for WBC, the increase between 2004 and 2006 and the decreases between 2006 and 2008, 2006 and 2009, and 2007 and 2009 were significant (always $P<0.0015$). For creatinine, the increases between 2002 and 2007, 2005 and 2007, and 2006 and 2007 were significant (always $P<0.0015$).

Table 3. Hematologic values from clinically healthy captive beira antelope (*Dorcatragus megalotis*) from Al Wabra Wildlife Preservation, Qatar. Ranges from domestic sheep and goats are provided for comparison.²⁸

Variable	<i>n</i>	Mean (SD)	Min	Max	Normal range ovine	Normal range caprine
Hematocrit (%)	9	52 (5.7)	44.5	60	27–45	22–38
Red blood cells ($10^6/\mu\text{l}$)	9	9.1 (1.37)	6.85	11.1	9–15	8–18
White blood cells ($10^3/\mu\text{l}$)	7	3.68 (0.48) ^a	2.88	4.35	4–12	4–13
Lobulated neutrophils (%)	8	81.06 (7.13)	73	89	10–50	30–48
Lymphocytes (%)	8	16.8 (6.4)	9	25	40–75	50–70
Monocytes (%)	6	2.42 (1.28)	1.5	5	—	0–4

^aSignificantly different from values in ill animals (8.60 [5.67]; P [nonparametric] <0.001).

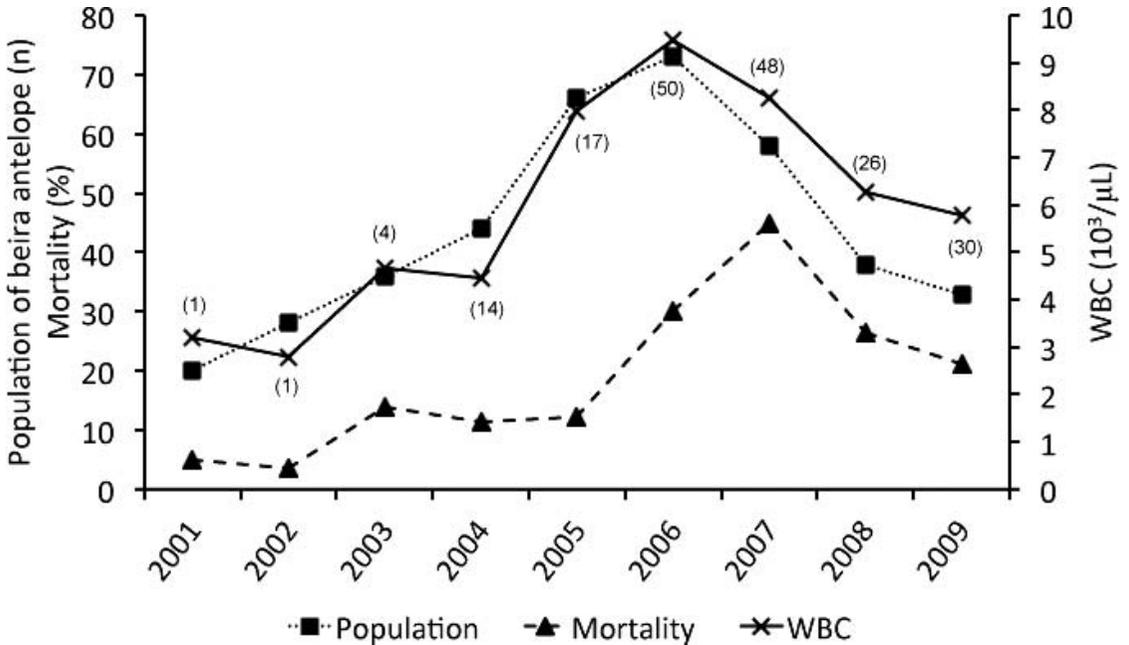


Figure 1. Mean white blood cell (WBC) count from 2001–2009 of the entire beira antelope (*Dorcatragus megalotis*) population at Al Wabra Wildlife Preservation, Qatar as compared to population size and mortality. If several blood samples had been taken from an individual during 1 yr, averages for that individual were calculated first; from these individual data the population average was calculated subsequently. The number of individuals with blood samples evaluated for WBC in a year is indicated in brackets.

Mean hematocrit of the healthy animals ($n = 9$) before the outbreak of respiratory disease was $52.0 \pm 5.7\%$ (Table 3). Population hematocrit values during the course of the disease declined from 47.5% in 2006 to 41.9% in 2009, but the difference between population hematocrit values

before and after the outbreak was not significant ($P = 0.291$).

DISCUSSION

This study is a retrospective evaluation correlating mortality with clinical pathology data. The major limitations of this study were the low sample size and different immobilization techniques used to collect the blood. Hematologic values should be interpreted with caution because of the low sample size; therefore, baseline intervals were not calculated. Additionally, the data from after the outbreak do not represent a balanced sample of the whole population but an opportunistic sample of all animals from which blood had been taken as part of veterinary procedures. But as such, these are typical, practical constraints when treating captive wild animals over long periods of time, and the presented information may still be considered relevant for zoo veterinarians.

Baseline biochemistry values for total protein, creatinine, sodium, potassium, chloride, phosphorous, magnesium, calcium, and ASAT of the beira antelope were comparable with values of other captive nondomestic ruminants^{15,27} as well as domestic sheep and goats.¹⁷ The GGT, GLDH,

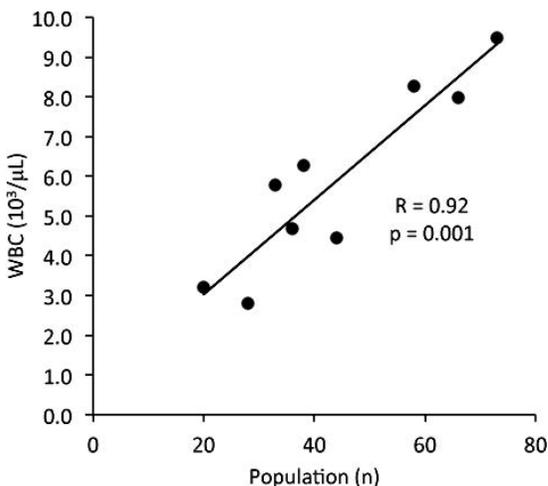


Figure 2. The correlation of the yearly mean white blood cell count of the beira population including healthy and diseased animals with the population size (n).

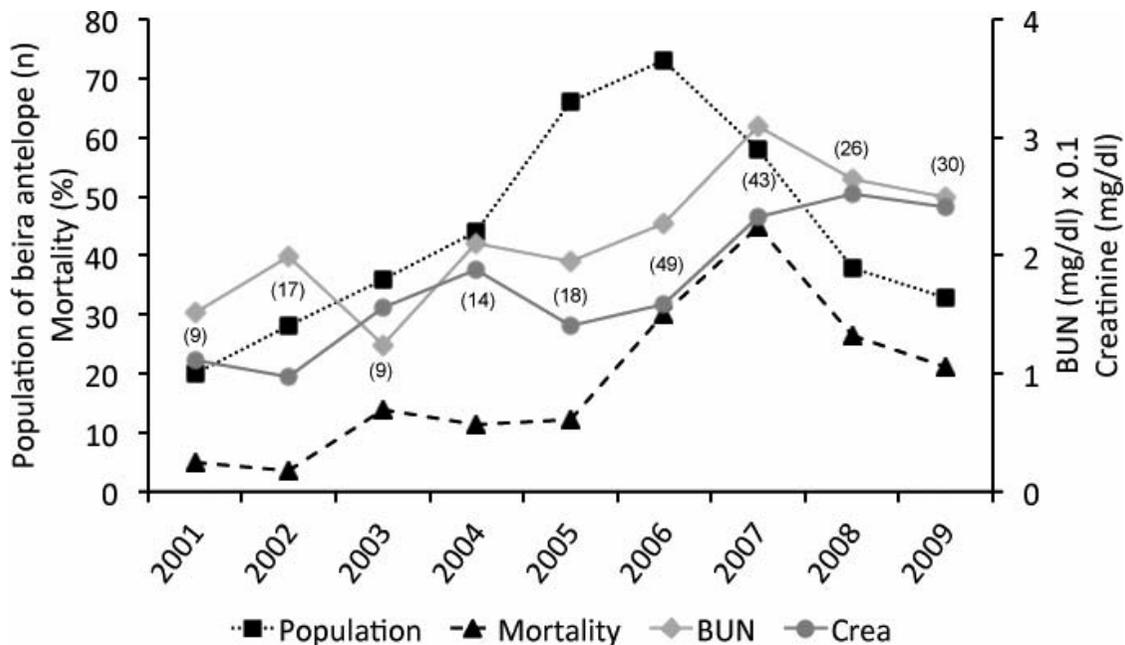


Figure 3. Trends in yearly mean blood urea nitrogen (BUN) values (multiplied by 0.1 for visualization) of the entire beira population (*Dorcatragus megalotis*) at Al Wabra Wildlife Preservation and yearly mean creatinine values as compared to population size and mortality. If multiple blood samples from an individual were taken in the same year, the average of this individual was used to calculate the mean of the population subsequently. The number of individuals with blood samples evaluated for BUN and creatinine in a year is indicated in brackets.

total bilirubin, cholesterol, and β -hydroxybutyrate were comparable with values of domestic sheep and goats.¹⁷

When comparing the baseline values to published reference ranges for domestic sheep and goats, BUN, glucose, creatine kinase, and ALP were higher than expected.¹⁷ This difference in BUN between nondomestic and domestic ruminants has been noted by other authors.^{7,15,17} Reasons for these results may include the stress of restraint in nondomestic animals, the ability to collect preprandial samples from domestic ruminants, differences in methodology in measuring the samples, or any combination of these.¹⁵ Nevertheless, baseline BUN levels in beira antelope provided a useful diagnostic tool because clinically ill animals had significantly higher BUN values. In particular, variation in BUN levels was significantly correlated with variation in creatinine, both on the level of annual population means in this study and on the level of measurements in individual animals as previously published.²⁵ This corroborates that biochemistry indicated impaired kidney function and not other causes wherein BUN or creatinine might increase unrelated to each other. Glucose levels in general are higher in wild than in domestic ruminants.¹⁵

Elevated glucose levels have been associated with release of adrenaline, cortisol, and glucagon due to stress and trauma.³⁰ The glucose levels of the beira antelope were also higher than in domestic sheep and goats¹⁷ but similar to other wild ruminants;^{15,27} therefore, it was more likely due to capture or due to anesthesia with medetomidine, as α 2-agonists have a hyperglycemic effect.²⁹ Creatine kinase and ALP were elevated compared to domestic sheep and goats,¹⁷ which is also thought to be due to capture.

Using blood values of the entire population only has a limited use as a prognostic tool for population health. Even so, such data seem to be useful to demonstrate trends in population health and long-term consequences of a health problem. The baseline values established in this report may help to monitor the beira population more closely. However, to monitor the health of individuals, it is recommended to compare blood values not only with established baseline values but also to establish databases of serial blood analyses from individuals to reduce the influence of interindividual differences.⁴

Compared to the population WBC, peaks in population BUN and creatinine occurred 1 yr later, at the same time as the peak in mortality.

This could be interpreted as the transition from an acute disease with a primary respiratory manifestation into a chronic condition where renal amyloidosis plays an important role. The change in renal values indicates that the previously reported amyloidosis was having an observable clinical effect. Supportive treatment led to survival of the acute respiratory disease, but most of these animals never recovered fully and remained chronically ill because of chronic kidney disease. Reactive systemic AA amyloidosis in animals is most-often caused by deposits of the acute phase protein serum amyloid A; these deposits are induced by chronic inflammatory or neoplastic conditions.³² In the beira antelope it was thought to be secondary due to the inflammatory stimulus induced by the respiratory disease.²¹ Animals with renal amyloidosis frequently die from renal failure.³²

In addition to differences in renal values between healthy and affected animals, liver values were similarly elevated. AST, GGT, total bilirubin, and GLDH were significantly higher in affected animals. These findings indicate that the amyloidosis noted histologically in the liver²¹ may have been causing impaired liver function. The association of chronic pulmonary disease with amyloidosis is described multiple times and seems to be an important complication in small domestic and wild ruminants.^{18,23,33,35}

Although WBC and mortality decreased, BUN and creatinine continued to be high in the population blood values. This indicates a severe compromise of kidney function in a large proportion of the animals. Population blood values thus implicate that amyloidosis is a significant, long-term complication of the putative *Mycoplasma* infection of the population. Also, population hematocrit values seemed to decline during the course of the lung disease; this may have been associated with amyloid deposits in the kidneys resulting in anemia of chronic kidney disease or anemia of chronic inflammatory disease similar to other mammals.^{16,28}

Parallels to these observations are found in an outbreak of lung disease complex in captive bighorn sheep (*O. canadensis*).³⁵ Pneumonia of bighorn sheep is a dramatic disease of high morbidity and mortality. At necropsy, lung consolidation, abscesses, fibrinous pleuritis, fibrinous pericarditis, and lungworms were found.³⁴ Amyloidosis was also seen and considered to be secondary to chronic inflammatory and suppurative processes.³⁵ An increased susceptibility of bighorn sheep to amyloidosis was suspected.¹¹ In

these animals leukocytosis, neutrophilia, lymphopenia, and eosinopenia were seen. A second hematologic trend was anemia.³⁵ The etiology of the disease has been debated since its initial discovery, but *M. ovipneumoniae* most completely satisfies the criteria for a causal role in the disease, unlike *Mannheimia haemolytica* and other *Pasteurellaceae* or lungworms.⁵ Domestic sheep are suspected to transfer the pathogen to bighorn sheep populations.³¹

In the beira antelope the population size correlated highly significantly with the WBC count, and population growth occurred concurrently with a rise in WBC count. It could be hypothesized that the increase in population size facilitated the outbreak of FPPS. The decline of the population to a lower level, however, did not lead to recovery of the population. Even though the outbreak and severity of infectious diseases may be density-dependent,^{3,6} the infectious diseases might persist even when herd size reduced to carrying capacity in captivity.²⁴ Such an overshooting reaction represents one of the reasons for the stochastic extinction risk faced by small populations, such as groups of captive species. With *Mycoplasma*, the challenge of clinically healthy carriers aggravates the problem.²⁶ Although antibiotic therapy can bring clinical improvements, it rarely eliminates the organism.²⁶ Carrier status could not be demonstrated in beira antelopes because *Mycoplasma* was only found in the tissue of dead animals. In bighorn sheep pneumonia, chronic carriers lead to spreading of the infection.⁵ Stagnation, decline, and even extirpation of metapopulations of bighorn sheep were seen in conjunction with bighorn sheep pneumonia.^{8,31}

In other species culling would be recommended to eliminate *Mycoplasma* from a population. As AWWP keeps the only captive population of beira antelope, this was not considered as an option. In the beira antelope it was attempted to create a *Mycoplasma*-free population with hygiene management, sentinel animals, prophylactic antibiotic treatment, regular health checks, vaccinations with an autogenous vaccine, and hand-raising of young animals with colostrum from vaccinated goats. Despite all efforts, the disease could not be stopped and the population decline in beira antelope at AWWP is still ongoing. With treatment attempts the acute respiratory disease does seem to transform into a chronic condition, but elimination of the disease seems unlikely. It was speculated that high population numbers of beira antelopes had facilitated the outbreak.²⁴ Although

population size declined, persistence of the infectious disease is most probable.

CONCLUSIONS

In the captive beira antelope population at AWWP, FPPS has significant, long-term effects leading to a decline in population. Animals that survive acute respiratory disease frequently go on to develop chronic, long-term manifestations of amyloidosis such as renal failure and impaired liver function secondary to chronic inflammatory disease.

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