SEROLOGIC SURVEILLANCE FOR SELECTED VIRAL AGENTS IN CAPTIVE AND FREE-RANGING POPULATIONS OF ARABIAN ORYX (*ORYX LEUCORYX*) FROM SAUDI ARABIA AND THE UNITED ARAB EMIRATES

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ABSTRACT: A total of 294 sera collected between 1999 and 2001 from eight captive and one free-ranging herds of Arabian oryx (*Oryx leucoryx*) distributed in Saudi Arabia (SA) and the United Arab Emirates (UAE) were assayed for antibodies against 13 selected viral agents. Arabian oryx have been exposed to bluetongue virus (BTV), epizootic hemorrhagic disease virus (EHDV), rinderpest virus (RPV), bovine respiratory syncytial virus (BRSV), bovine adenovirus 3 (BAV-3), cervid herpesvirus-1, foot-and-mouth disease virus, equine herpesvirus 9, and bovine viral diarrhea virus. The high seroprevalence to BTV and EHDV in the UAE and SA indicates that Arabian oryx are likely to be susceptible to infection by these viruses and therefore could act as a source of virus to vectors during the infective stage of infection. Moreover, antibodies were detected against RPV and BRSV in sera from SA and against BAV-3 in sera from the UAE. No antibodies were found against bovine herpesvirus-1, caprine herpesvirus-1, enzootic bovine leucosis virus, and peste des petits ruminants virus. On the basis of these results, caution should be applied when considering translocation of Arabian oryx, and only those proven to be free of infectious agents that might present a risk to other species should be moved.
   Key words: Arabian oryx, *Oryx leucoryx,* Saudi Arabia, serologic status, United Arab Emirates, viral agents.

INTRODUCTION

The Arabian oryx (*Oryx leucoryx*) is one of six surviving species within the subfamily Hippotraginae (Wilson and Reeder, 1993). Although excessive hunting resulted in the extinction of this species in the wild by the early 1970s (Henderson, 1974), reintroduction of captive-bred animals to their former ranges has been carried out successfully in Oman in 1982 and Saudi Arabia (SA) in 1990 and 1995 (Ostrowski et al., 1998; Spalton et al., 1999).

There is evidence that Arabian oryx are susceptible to some agents that affect livestock (Kock and Hawkey, 1988; Flamand, 1999; Ostrowski et al., 2002); therefore, there is always a risk that relocated animals can either become infected with agents already present or introduce new and novel pathogenic agents into naive areas (Woodford, 1989; Woodford and Rossiter, 1994). Obtaining up-to-date information on the serologic status of these animals and occurrence of diseases in oryx that affect domesticated animals is important for the health management of captive Arabian oryx herds and for reintroduction programs (Greth et al., 1992).

The objective of this study was to determine the prevalence of antibodies against 13 selected viral agents in 294 captive and free-ranging Arabian oryx living in the United Arab Emirates (UAE) and SA. This study is particularly relevant because it concerns reintroduction projects of a threatened species into several Middle Eastern countries.

MATERIALS AND METHODS

Two hundred ninety-four blood samples were collected from eight captive herds (*n*=278) and one free-ranging herd (*n*=16) of Arabian oryx in SA (*n*=98) and the UAE (*n*=196) between 1999 and 2001 (Table 1). The number of serologic reactors was calculated

TABLE 1.   Herd size and number of samples collected at different locations from Saudi Arabia and the United Arab Emirates between 1999 and 2001.

Location	Herd size	Sample size
Saudi Arabia		
A[a]	40	20
F1+F2[a]	200	62
Mahazat as-Sayd	450	16
United Arab Emirates		
Jarf	300	55
Sea Palace	65	20
Reef	180	15
Ghantoot	120	48
Bida Khalifa	8	8
Bani Yas	350	50
Total	1,713	294

[a] Located in the National Wildlife Research Center; herd F1 includes the offspring of herd A, and F2 consists of the offspring of F1; only F1 and F2 are in direct contact.

FIGURE 1.   Distribution of the Arabian oryx collection sites in Saudi Arabia (SA) and the United Arab Emirates (UAE) (●).

separately for each location, because the herds had no contact to each other.

In SA, samples were collected at the National Wildlife Research Center (NWRC; 21°15′N, 40°41′E) near Taif (Fig. 1) and in the reserve of Mahazat as-Sayd (28°15′N, 41°40′E). The Arabian oryx herd of the NWRC was created in 1986 as part of the national oryx conservation plan. Because of a bovine tuberculosis outbreak that occurred in 1986 and 1987, the founder group (generation A) had been kept isolated in one area of the NWRC with no contact in following generations (F1, F2). The F1 generation was removed from their dams immediately after birth. Thus, from an epidemiologic aspect, we considered A and F1+F2 as two separate herds. The animals had no direct contact with other ungulate species.

Mahazat as-Sayd, a 2,240 km² protected area located about 200 km northeast of the NWRC, was fenced in 1989 to exclude poachers and grazing livestock. The free-ranging herd of Arabian oryx of the reserve was founded from 72 captive-born animals from the F1+F2 herd at NWRC and elsewhere; animals were reintroduced between 1990 and 1993. Reintroduced animals and their offspring are never returned to the NWRC. Apart from a population of reintroduced Arabian sand gazelles (*Gazella subgutturosa marica*; Nayerul Haque and Smith, 1995), the free-ranging Arabian oryx herd has no direct contact with other ungulate species. The approximate herd size for Mahazat as-Sayd during 1999 and 2001 was estimated with an exponential model, starting with 350 animals at
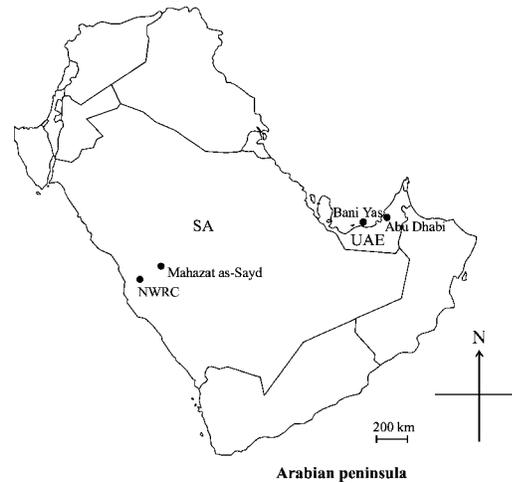
the end of 1997 (Ostrowski et al., 1998) and ending with 650 animals at the beginning of 2003.

At the NWRC, animals were bled during the annual winter prophylaxis operations. They were individually kept in a 40-m² capture pen before blood sampling. Males were immobilized with xylazine (Rompun®, Bayer, Leverkusen, Germany) according to Ancrenaz (1994) with the use of a pneumatic dart gun (GUT-50, Telinject, Germany). Females were handled and blood-sampled without being tranquilized. At Mahazat as-Sayd, blood of Arabian oryx were opportunistically sampled when captured to fix identification collars. They were immobilized with a combination of etorphine (M99®, 4.5–5 mg/animal, Grampian Pharmaceuticals, Dundee, UK) and xylazine (12–25 mg/animal). Animals were reversed with diprenorphine (M5050®, 2–3 mg/mg etorphine iv, Grampian Pharmaceuticals) and atipamezole (Antisedan®, 0.2–0.3 mg/mg xylazine iv, Farmos Group, Turku, Finland) at Mahazat as-Sayd and with atipamezole alone at NWRC.

In the UAE, sampling was performed at five different locations (Jarf, Sea Palace, Reef, Ghantoot, and Bida Khalifa) in the vicinity of Abu Dhabi city (24°29′N, 54°22′E) and at a sixth site on Sir Bani Yas Island (24°19′N, 52°36′E; Fig. 1). These six herds comprise between eight and 350 captive animals living in large fenced enclosures varying from 1–30 km². Because there have been no movements of animals into the herds, these are six different herds from an epidemiologic point of view. All oryx from the UAE were immobilized according to Ancrenaz (1994). Sera from all animals

bled in SA and the UAE were stored at −20 C before assay. All animals sampled in SA and the UAE were adult (3.5–10 yr).

A constant 1:5 dilution of each serum was assayed with serogroup-specific competitive enzyme-linked immunosorbent assay (C-ELISA) for the presence of antibodies to rinderpest virus (RPV) and peste des petits ruminants virus (PPRV; Anderson et al., 1991), bluetongue virus (BTV; Anderson, 1984), and epizootic hemorrhagic disease virus (EHDV; Thevasagayam et al., 1995). Positive sera specific for each virus being tested and a negative serum were included as controls for each ELISA and on each test plate. Sera giving percent inhibition values equal to or greater than 50% were recorded positive.

The Eli-vet bovine respiratory syncytial virus (BRSV) and bovine adenovirus-3 (BAV-3) assays (Sanofi, Cera GmbH, Düsseldorf, Germany) were used for the detection of antibodies against BRSV and BAV-3, respectively. Briefly, specific antigen was adsorbed onto ELISA microplate wells. Wells without viral antigens were used to test the conspecificity of the assay. Bovine antibodies specific to the corresponding virus-type antigens were detected by a monoclonal anti-bovine IgG_1 antibody coupled with peroxidase. Because Arabian oryx belong to the family Bovidae, a high level of cross-reactivity with anti-bovine IgG_1 was assumed.

Four cytopathic strains of bovine viral diarrhea virus (BVDV: SH9/11, Grub 313/83, NADL, and Osloss) were used in microneutralization tests (NT; Frey and Liess, 1971; Frölich and Hofmann, 1995) for the detection of anti-BVDV antibodies. All tests were performed in 96-well microtiter plates (Nunc-Gibco, Paisley, Renfrewshire, UK) with 100 $TCID_{50}$ (dose infecting 50% of the inoculated tissue culture cell) BVDV per 100-μl well and twofold serum dilutions. The NT was performed for 1 hr at 37 C. Subsequently, 3×10^5 cells/ml were seeded into each well. Georgia bovine kidney cells (American Type Culture Collection, Rockville, Maryland, USA) and Dulbecco's modified eagle medium (DMEM, Life Technologie, Berlin, Germany) with 5% fetal bovine calf serum (FCS) were used for verification of BVDV. Four days later, the formalin-fixed cell cultures were evaluated for the presence of cytopathic effects (Frost et al., 1990), and antibody titers were calculated according to Spearmann and Kärber (1985). Titers of more than 1:4 were considered positive (Malmquist, 1968). Neutralization tests were performed twice for each serum, and the mean titer was calculated. Virus, cell, and FCS controls were included in each test.

The 3ABC ELISA used was designed to detect antibodies against the foot-and-mouth disease virus (FMDV) polyprotein 3ABC in exotic species, including oryx. The assay uses glutathione S-transferase expressed 3ABC as the antigen, prepared according to the methods of De Diego et al. (1997) and Mackay et al. (1998). This assay uses conjugated protein G, which is known to bind to the antibody molecules of a wide variety of species (Kronvall, 1973; Akerstrom et al., 1985). Briefly, GST-3ABC antigen, optimally diluted in carbonate-bicarbonate buffer (pH 9.6), was added to all test wells of columns 2 and 3, 5 and 6, 8 and 9, and 11 and 12 of ELISA plates (Maxisorp Immuno-plate, Nunc-Gibco). Columns 1, 4, 7, and 10 (negative antigen control wells) received 50 μl/well of carbonate-bicarbonate buffer alone. Plates were incubated at ambient temperature (18–22 C) overnight in a humidity chamber. Sera, diluted 1:200 in PBS containing 0.05% Tween 20 (PBST), 3% commercial unsweetened organic soya milk, 1% normal horse serum (blocking buffer), and 1% *Escherichia coli* sonicate, were allowed to stand at room temperature for 1 hr before addition to the ELISA plates. The ELISA plates were washed three times with PBS. Fifty microliters of diluted test sera were then added to triplicate wells of each row (e.g., A1, A2, and A3). Plates were incubated at 37 C for 1 hr on an orbital shaker. Control sera on each plate included a strong and weak FMDV-specific positive convalescent serum, a negative bovine serum, a strong FMDV-positive serum, and a negative gemsbok (*Oryx gazella*) serum. Plates were washed as before, and 50 μl of protein G horseradish peroxidase conjugate, optimally diluted in blocking buffer, was added to each well. Plates were incubated as before. After washing, 50 μl/well of chromogen/substrate (orthophenylene diamine/H_2O_2) were added to each well. The color reaction was stopped after 15 min by addition of an equal volume of 1.25 M H_2SO_4. Absorbance values were determined spectrophotometrically at 492 nm. Results for test and control sera were expressed as the mean optical density (OD_{492}) for the two wells with GST-3ABC antigen minus the OD_{492} of the well containing only buffer. The net OD_{492} of every test sample and of the weak positive control were then divided by the net OD_{492} of the strong positive bovine control, resulting in the test/positive (t/p) ratio. This procedure adjusts results for plate-to-plate and for between-test variation. Test sera giving t/p ratios equal to or greater than that of the weak positive control on the same plate were considered positive.

Antibodies against the following three alphaherpesviruses were compared: bovine herpesvirus-1 (BHV-1; Cooper-type strain, USA), caprine herpesvirus-1 (CapHV-1; E/CH), and

the Moredun strain of cervid herpesvirus (HVC-1). A standard NT (Ackermann et al., 1986) was used for the detection of anti-herpesvirus antibodies. All tests were performed in 96-well microtiter plates. Georgia bovine kidney cells and DMEM with 5% fetal bovine serum were used for the propagation of all herpesviruses. In an initial screening procedure, 50 µl of 1:4 serum dilutions were incubated in duplicate with approximately 70 plaque-forming units (PFU) in 50 µl DMEM/well for 1 hr at 37 C. Subsequently, 3×10^4 cells/100 µl were seeded into each well. After 3 hr, 200 µl of 1.6% carboxymethylcellulose in DMEM were added. In the second assay, the positive sera were titrated in twofold dilutions. The procedure was as described above, however four wells were used for each dilution. The cells were examined for the presence of cytopathic effects after 2 days for BHV-1 and CapHV-1 and 5 days for HVC-1. Virus, cell, and FCS controls were included in each test, and antibody titers were calculated according to Horzinek (1985). Titers were expressed as the reciprocal of the highest dilution of serum exhibiting 50% inhibition of cytopathic effects. Titers of 1:4 or more were considered positive (Ek-Kommonen et al., 1982). The NT was performed twice for each serum, and the mean titer was calculated.

A standard NT, as described by Fukushi et al. (1997), was used for the detection of antibodies against equine herpesvirus-9 (EHV-9). All tests were performed in 24-well plates (Nunc-Gibco). Equine dermis cells (ZBV, BFA für Viruskrankheiten der Tiere, Insel Riems, Germany) and DMEM with 10% fetal bovine serum were used for the propagation of herpesvirus. In an initial screening procedure, 100 µl of 1:4 heat-inactivated (30 min at 56 C) serum dilutions were incubated with approximately 70 PFU in 100 µl DMEM for 1 hr at 37 C. Subsequently, 1×10^5 cells/200 µl were seeded into each well. After 3 hr, 400 µl of 1.6% carboxymethylcellulose in DMEM was added. After 1 day, the cells were examined for the presence of cytopathic effects. Plaques were stained with Giemsa solution and counted. In a second step, the positive sera were titrated in twofold dilutions. Antibody titers were expressed as the highest serum dilution producing greater than 50% reduction in plaque counts. Titers of 1:10 or more were considered positive. All samples were tested twice in this assay, and the mean titer was calculated. Virus, cell, and FCS controls were included in each test.

The CHEKIT-leucosis ELISA (Dr. Bommeli AG, Liebefeld-Bern, Switzerland) was used for the detection of antibodies against enzootic bovine leucosis virus (EBLV). Briefly, the wells of the CHEKIT-leucotest microplates were precoated with inactivated virus antigen (positive antigen) and blank antigen (negative antigen). Antibodies against EBLV in a serum sample are bound by the virus antigen on the leucotest microplate. The formation of immunocomplex elements is detected by means of the anti-bovine immunoglobulin peroxidase conjugate, staining the CHEKIT chromogen blue-green. Because Arabian oryx belong to the family Bovidae, a high level of cross-reactivity with anti-bovine IgG_1 was assumed. Nonspecific antibodies are evenly bound to negative antigen and positive antigen. The net extinction of a sample is equal to the positive antigen extinction minus the negative antigen extinction of this sample. The intensity of the color corresponds to the antibody titer of the sample. Therefore, the samples must be prepared only in one dilution (1:40). Diagnostic evaluation depends on the spectrophotometric comparison of the color with controls.

For all tests in this study, a negative control serum was used. However, positive control sera were not available for most of the tests. Therefore, detection of antibodies against a closely related virus cannot be excluded.

The prevalence and the 95% confidence interval (CI) were calculated separately for each virus strain and each location. The population sizes were known, so the 95% CIs were corrected for the respective population sizes according to Burstein (1975). Because of the generally small number of samples, the prevalence of antibodies recorded for each viral agent in different years was not compared. However, the seroprevalence at different locations was compared for each virus strain between the three SA locations and between the six UAE locations. Interdependencies between pairs of categorical or binary variables were tested by the chi-square test (exact version). Adjusted standardized residuals were used to identify the locations responsible for significant differences (Everitt, 1977). More frequently than expected positive samples (standardized residual of >1.96) or less frequently than expected positive samples (standardized residual of <−1.96) in a strain's contingency table (seropositivity, location) are indicated by a + or − sign, respectively (Tables 2, 3). The significance level was generally set to α=0.05. SPSS 9.0 (SPSS Inc., Chicago, Illinois, USA) served for the statistical calculations, except for the Burstein correction.

RESULTS

Table 2 records the results obtained for the Arabian oryx sera collected in SA. A

TABLE 2.   Prevalence of antibodies[a] to 16 viral agents in sera of captive (A, F1, F2) and free-ranging (Mahazat as-Sayd) Arabian oryx from three different locations[b] in Saudi Arabia.

Agent[c]	A		F1+F2		Mahazat as-Sayd
	P/T[c]	CI	P/T	CI	P/T
BTV	9/20	0.29–0.62	28/58	0.37–0.60 +	0/16 −
EHDV	10/20	0.32–0.66	24/58	0.30–0.53	0/16 −
BRSV	2/20	0.03–0.26	15/62	0.16–0.35 +	0/16 −
RPV	3/20	0.06–0.32	16/58	0.18–0.39	0/16
BAV-3	1/20	0.01–0.19	2/62	0.01–0.10	0/16
BVDV (NADL)	0/20		5/62	0.03–0.16	0/16
BVDV (Grub)	0/20		5/62	0.03–0.16	0/16
BVDV (SH9/11)	1/20	0.01–0.19	5/62	0.03–0.16	0/16
BVDV (Osloss)	0/20		0/62		0/16
FMDV	0/20		0/58		0/16
HVC-1	0/20		0/59		0/15
BHV-1	0/19		0/59		0/15
CapHV-1	0/20		0/62		0/16
EHV-9	0/20		0/59		0/16
EBLV	0/20		0/62		0/16
PPRV	0/20		0/58		0/16

[a] P/T = number of positive reactors/number of samples tested; CI = 95% confidence interval; + = more than expected positive samples; − = fewer than expected positive samples.

[b] Only individuals from F1 and F2 are in direct contact.

[c] BTV = bluetongue virus; EHDV = epizootic hemorrhagic disease virus; BRSV = bovine respiratory syncytial virus; RPV = rinderpest virus; BAV-3 = bovine adenovirus 3; BVDV = bovine viral diarrhea virus; FMDV = foot-and-mouth disease virus; HVC-1 = cervid herpesvirus 1; BHV-1 = bovine herpesvirus 1; CapHV-1 = caprine herpesvirus 1; EHV-1 = equine herpesvirus 1; EBLV = enzootic bovine leucosis virus; PPRV = peste des petits ruminants virus.

high prevalence of antibodies was recorded against the two arthropod-borne viruses BTV and EHDV and against RPV and BRSV in herds A and F1+F2, whereas no antibodies against these four viruses were observed in the herd from Mahazat as-Sayd (Table 2). A low prevalence of antibodies was detected among captive Arabian oryx from SA against the BVDV strains SH9/11, NADL, and Grub 313/83. The NT titers varied between 1:19 and 1:150 against SH9/11, between 1:13 and 1:298 against NADL, and between 1:9 and 1:89 against Grub 313/83. No antibodies against Osloss strain were found in any of the sera tested. Because of the low number of positive samples for all three strains, a statistical comparison was not performed. A low seroprevalence for BAV-3 was only recorded in herd A (one of 20) and herd F1+F2 (two of 62). No antibodies were detected against the four different herpesviruses (HVC-1, BHV-1, CapHV-1, and EHV-9), FMDV, EBLV,

and PPRV. Significantly different seroprevalences were found between herds A, F1+F2, and Mahazat as-Sayd for BRSV (P=0.040), BTV (P=0.002), and EHDV (P=0.003). More seroreactors than expected were found for BRSV and BTV in herd F1+F2, and fewer than expected were found in the Mahazat as-Sayd herd. Fewer than expected reactors were also observed for EHDV in the Mahazat as-Sayd herd (Table 2).

Table 3 records the results obtained for the Arabian oryx sera collected in the UAE. Antibodies against BTV and EHDV were recorded for each year of sampling and at all locations except Reef (where no antibodies were detected against EHDV), whereas antibodies against BRSV were only observed in herds from Jarf (one of 55), Sea Palace (four of 20), and Reef (one of 15). Unlike SA, no antibodies were detected against RPV. Likewise, no antibodies were detected against the four strains of BVDV tested. A low prevalence of an-

TABLE 3.   Prevalence of antibodies[a] to 16 viral agents in sera of captive Arabian oryx from six different locations[b] in the United Arab Emirates.

Agent[c]	Jarf		Sea Palace		Reef		Ghantoot		Bida Khalifa		Bani Yas	
	P/T[c]	CI	P/T	CI	P/T	CI	P/T	CI	P/T	CI	P/T	CI
BTV	17/55	0.20–0.44 +	3/19	0.05–0.36	2/15	0.02–0.39	5/48	0.05–0.20	1/7	0.05–0.32	3/50	0.02–0.16 –
EHDV	31/55	0.44–0.69 +	1/19	0.01–0.23 –	0/15	–	7/48	0.08–0.25 –	3/7	0.26–0.61	31/50	0.48–0.73 +
BRSV	1/55	0.00–0.09	4/20	0.08–0.40 +	1/15	0.00–0.31	0/48		0/7		0/50	
RPV	0/55		0/19		0/15		0/48		0/7		0/50	
BAV-3	1/55	0.00–0.09	0/20		0/15		11/48	0.14–0.34	0/7		3/50	0.02–0.16
BVDV (NADL)	0/55		0/20		0/15		0/48		0/8		0/50	
BVDV (Grub)	0/55		0/20		0/15		0/48		0/8		0/50	
BVDV (SH9/11)	0/55		0/20		0/15		0/48		0/8		0/50	
BVDV (Osloss)	0/55		0/20		0/15		0/48		0/8		0/50	
FMDV	0/55		0/19		0/15		2/48	0.01–0.12	0/7		0/50	
HVC-1	0/55		0/20		0/15		1/48	0.00–0.09	0/8		2/49	0.01–0.13
BHV-1	0/55		0/20		0/15		0/47		0/8		0/48	
CapHV-1	0/55		0/20		0/15		0/48		0/8		0/48	
EHV-9	0/55		0/20		0/15		0/47		0/7		1/49	0.00–0.10
EBLV	0/55		0/20		0/15		0/48		0/7		0/50	
PPRV	0/55		0/19		0/15		0/48		0/7		0/50	

[a] P/T = number of positive reactors/number of samples tested; CI = 95% confidence interval; + = more than expected positive samples; – = fewer than expected positive samples.
[b] Individuals from the different locations are not in direct contact.
[c] BTV = bluetongue virus; EHDV = epizootic hemorrhagic disease virus; BRSV = bovine respiratory syncytial virus; RPV = rinderpest virus; BAV-3 = bovine adenovirus 3; BVDV = bovine viral diarrhea virus; FMDV = foot-and-mouth disease virus; HVC-1 = cervid herpesvirus 1; BHV-1 = bovine herpesvirus 1; CapHV-1 = caprine herpesvirus 1; EHV-1 = equine herpesvirus 1; EBLV = enzootic bovine leucosis virus; PPRV = peste des petits ruminants virus.

tibodies was detected against HVC-1 (Ghantoot: one of 48, Bani Yas: two of 49), EHV-9 (Bani Yas: one of 49), and FMDV (Ghantoot: two of 48). Antibodies against BAV-3 were only recorded in herds from Jarf (one of 55), Ghantoot (11 of 48), and Bani Yas (three of 50). No antibodies were detected against EBLV and PPRV. Among the six UAE locations, seroprevalence was significantly different for BRSV ($P=0.002$), BTV ($P=0.016$), and EHDV ($P<0.001$). More seroreactors than expected were found for EHDV in the Jarf and Bani Yas herds, and fewer than expected were observed in Sea Palace, Reef, and Ghantoot herds. For BTV, more seroreactors than expected were found in the Jarf herd, and fewer than expected were observed in the Bani Yas herd. For BRSV, only in one herd (Sea Palace) were more seroreactors than expected found.

DISCUSSION

Bluetongue (BT) and epizootic hemorrhagic disease (EHD) are infectious but noncontagious arthropod-borne viral diseases of domesticated and wild ruminants. Bluetongue virus is the prototype virus of the genus *Orbivirus* within the family *Reoviridae.* Twenty-four serologically distinct types have been identified. The viruses are considered to be endemic in the Middle East, tropical and subtropical Africa, Australia, and America, although excursions of BTV outside of these areas do occasionally occur (Mellor and Wittman, 2002). Clinical disease is characterized by extensive edema and hemorrhage. Viremia in sheep and cattle have been reported to be as long as 54 days (Koumbati et al., 1999) and 112 days (Du Toit, 1962), respectively, although in most animals, the duration of viremia is considerably less. Epizootic hemorrhagic disease virus is another member of the genus *Orbivirus* comprising at least eight serotypes. Clinical signs of EHD are usually less dramatic than BT in domesticated ruminants, although in white-tailed deer (*Odocoileus virginianus*), disease can be severe (Fischer et al.,

1995). Viremia has been reported to persist for up to 28 days in cattle (Gibbs and Lawman, 1977), whereas in white-tailed deer, it has been detected >50 days (Quist et al., 1997). Serologic evidence shows the virus to be widespread, occurring in the same geographic areas as BTV (Thevasagayam, 1998). Both of these viruses are transmitted by *Culicoides* biting midges; therefore, infection is usually confined to those areas in which competent vectors abound (e.g., *Culicoides imicola* in the Middle East; Mellor and Boorman, 1995).

Antibodies against BTV and EHDV have been recorded in several wild ruminants, including members of the tribe Hippotragini (i.e., roan antelope, *Hippotragus equinus*; Formenty et al., 1994; Thevasagayam, 1998), sable antelope (*Hippotragus niger*; Anderson and Rowe, 1998; Thevasagayam, 1998), and beisa oryx (*O. gazella beisa*; Davies and Walker, 1974). Our report describes significant levels of antibodies against BTV and EHDV in Arabian oryx. In a previous serologic survey in a herd of the NWRC, only one of 78 animals tested was reported seropositive (Greth et al., 1992). Although no evidence of either BTV or EHDV infection has been reported for Arabian oryx, the results here indicate that this species, like other ruminants, is susceptible to infection. Lack of seroreactors against BTV and EHDV in Arabian oryx from Mahazat as-Sayd compared with those from the NWRC is consistent with the hypothesis that these viruses have not circulated in the free-ranging population. We suggest that the high ambient temperature, intense solar radiations, desiccating conditions, and rare precipitation (average annual rainfall < 100 mm) prevailing in Mahazat as-Sayd might impose significant abiotic limitations to the development of transmitting *Culicoides* sp. vectors. The differences of exposure between the herds in the UAE might be a result of habitat modification through the establishment of forests by irrigation (Jarf, Bani Yas) that could favor development of vectors. Although BTV and EHDV are not

considered a major threat to Arabian oryx, they might act as a virus source during any viremic period. Therefore, only Arabian oryx that are not infected with BTV should be considered for translocation and release (Flamand, 1999).

Bovine respiratory syncytial virus belongs to the genus *Pneumovirus* within the family *Paramyxoviridae.* Infection of wild ruminants might be subclinical or result in pneumonia (Spraker and Collins, 1986; Foreyt and Evermann, 1988). Serologic and virologic evidence of BRSV infections have been found in a variety of wild ruminant species throughout the world. However, this has not been the case for Arabian oryx or related species, such as roan and sable antelope or gemsbok, studied previously (Van Campen and Early, 2001). Therefore, this is the first report indicating exposure to BRSV in herds of Arabian oryx. The high number of seropositive reactors for BRSV in Sea Palace (UAE) might be explained by the close proximity of the herd to humans and subsequent interaction. Handling of animals could play a role in the transmission of the virus (Berthiaume et al., 1973; Jacobs and Edington, 1975).

Rinderpest virus is classified in the genus *Morbillivirus* in the family *Paramyxoviridae* (Kingsbury et al., 1978). The virus is highly contagious and causes an acute to subacute disease of mammals in the order Artiodactyla. In the past, the disease was characterized by necrosis and erosions in the gastrointestinal tract that results in severe diarrhea, dehydration, and death. Morbidity and mortality rates often exceeded 90%. Recently however, inapparent infections have been more common in cattle (Rossiter, 1996). The spread of RPV is almost exclusively by contact between infected and susceptible animals (Rossiter, 1994). Wild ungulates exhibit a wide range of clinical signs, ranging from very severe in kudu (*Tragelaphus strepsiceros*), African buffalo (*Syncerus caffer*), giraffe (*Giraffa camelopardalis*), and eland (*Tragelaphus oryx*) to mild or

nonspecific signs in impala (*Aepyceros melampus*; Rossiter, 1994). Beisa oryx are reported to be moderately susceptible to the disease (Rossiter et al., 1983), which is probably true for Arabian oryx as well (Rossiter, 1994; Flamand, 1999). For that reason, all Arabian oryx are vaccinated at least once in their life against RPV at the NWRC. Because the test used in this study also detects postvaccine antibodies, the presence of antibodies in the two NWRC populations is therefore likely to reflect postvaccine seroconversion. However, knowing that all oryx have been vaccinated, it is surprising that only a few seropositive reactors were found, particularly because in cattle, vaccinal antibodies are considered to provide lifelong immunity. This might be related to an inadequately preserved vaccine. No RPV-seropositive Arabian oryx from Mahazat-as Sayd were found because none of them have been vaccinated. Absence of antibodies in herds from the UAE might be because not all herds of Arabian oryx are vaccinated against RPV and because livestock living in the vicinity of the herds are presently vaccinated against RPV.

Bovine adenovirus-3 is a member of the family *Adenoviridae.* Viruses are shed in a variety of ways, including coughing. The disease usually starts with respiratory and enteric signs; fever and anorexia might be observed (Woods, 2001). Low seroprevalence for BAV-3 was found in herd A and herd F1+F2 from SA and in the Jarf, Ghantoot, and Bani Yas herds in the UAE. Only one previous report of antibodies against BAV-3 was found in oryx and involved captive scimitar-horned oryx (*Oryx dammah*) in the United Kingdom (Frölich and Flach, 1998). Therefore, this is the first report that indicates exposure of Arabian oryx to BAV-3.

Bovine viral diarrhea/mucosal disease is an acute, highly contagious disease that is characterized mainly by pyrexia, nasal discharge, erosions of the alimentary mucosa, and diarrhea (Malmquist, 1968). The virus is a member of the genus *Pestivirus* within

the family *Flaviviridae.* Antibodies against BVDV have been reported in many wild species and affect a wide range of hosts. Infection of wild ruminants can result in either acute disease or a subclinical infection (Van Campen et al., 2001). A high prevalence of antibodies against the NADL strain has been recorded in three other related species of Hippotraginae: gemsbok and sable and roan antelope (Hamblin and Hedger, 1979; Soine et al., 1992). Ours is the first study to demonstrate exposure of Arabian oryx to BVDV. However, positive sera were only found in SA in herds A and F1+F2 from the NWRC.

Foot-and-mouth disease is a highly contagious acute viral infection of ruminants and pigs. The virus belongs to the family *Picornaviridae* and is the only member of the genus *Aphthovirus.* Seven serologically distinct serotypes have been identified. Transmission is usually by direct contact, but the virus can also be transmitted via fomites and over considerable distances by the airborne route. Their geographic distribution is heterogeneous, and four of the seven identified serotypes are or have been prevalent in the Middle East (types O, A, C, and Asia 1), although there have been incursions of a fifth (SAT 1; Hedger, 1981; Thomson, 1994). Clinical signs include vesicular lesions and erosions in the mouth, tongue, lips, and nose. Foot lesions occur in the interdigital space and around the coronary band. Young animals can die as a result of myocarditis, often referred to as "tiger-heart disease." Virus-neutralizing antibodies against different FMDV serotypes have been reported in sable and roan antelope and gemsbok (Condy et al., 1969). More recently, an outbreak caused by FMDV serotype O was reported in captive Arabian oryx in 2001 by the National Commission for Wildlife Protection, Bahrain. Morbidity was 100%, and mortality reached 60%. Two animals aborted and 42 died (Ostrowski and Anajariyah, 2002). The two Arabian oryx that were seropositive in 2001 had been negative the previ-

ous year. Because the 3ABC ELISA only detects antibodies produced against actively replicating virus, it is probable that these two animals were infected during the 2001 FMDV type O outbreak in the UAE (Anonymous, 2002). Therefore, it is recommended that captive Arabian oryx should be vaccinated annually against the FMDV serotypes that are usually prevalent in the Middle East. In addition, the continued vaccination of susceptible domestic livestock in and around reserves in which the Arabian oryx are being reintroduced is important (Thomson, 1994; Flamand, 1999).

Serologic surveys performed in a variety of ungulates have revealed the presence of alphaherpesviruses (Nettleton et al., 1988; Frölich, 1996). Such viruses include BHV-1, which causes infectious bovine rhinotracheitis and pustular vulvovaginitis in cattle (Ludwig and Gregersen, 1986), HVC-1 isolated from red deer (*Cervus elaphus*; Jenny and Wessman, 1973; Inglis et al., 1983), the rangifer herpesvirus isolated from reindeer (*Rangifer tarandus*; Ek-Kommonen et al., 1986), and CapHV-1 isolated from goats (Engels et al., 1992). Experimental studies suggest that these viruses are quite host specific (Reid, 1994). Clinical signs in ungulates include conjunctivitis, lacrimation, and corneal lesions. Ulceration of the nares and serous or purulent nasal discharge can also occur. Direct contact is normally required for natural transmission of herpesviruses in ungulates (Inglis et al., 1983; Reid et al., 1986; Nettleton et al., 1988). Serologic reactors against alphaherpesviruses have been identified in roan and sable antelope and gemsbok (Hedger and Hamblin 1978; Hamblin and Hedger, 1982). This is the first report indicating exposure of Arabian oryx to alphaherpesviruses (HVC-1), with all three seropositive individuals originating from the UAE. Antibodies against HVC-1 in the three Arabian oryx tested might be specific but could also be a result of either nonspecific reactions or cross-reactions with other alphaherpesviruses as

yet unidentified that are specific to the Arabian oryx.

Equine herpesvirus-9 was first identified following an outbreak of acute encephalitis in captive Thomson's gazelles (*Gazella thomsoni*) in Japan (Fukushi et al., 1997), although the natural host remains unknown. This is the first report of the possible exposure of Arabian oryx to EHV-9. The one seropositive animal originated from the UAE.

The results presented here show that Arabian oryx have been exposed to BTV, EHDV, RPV, BRSV, BAV-3, HVC-1, FMDV, EHV-9, and BVDV or closely related viruses. Besides vaccination (rinderpest, FMD), these responses could reflect either an earlier incursion of virus into the population, continual exposure of a non-susceptible species, or a coincidental transmission from another species. The high antibody prevalence detected against BTV and EHDV in both UAE and SA oryx indicates that Arabian oryx are likely susceptible to both viruses and that if they become viremic, they could act as a source of virus to vectors. Absence of seroreactors against various viruses in animals from Mahazat as-Sayd could be explained by lack of transmission, the low density of the desert population, or both. Before consideration is given to relocation and reintroduction of oryx, it is critical to know the infection status. However, serology is the first step in determining the potential significance that pathogens could play. More work is needed to understand the origin, pathogenesis, and importance of these pathogens in Arabian oryx.

LITERATURE CITED

ACKERMANN, M., A. E. MELTZER, H. MCDONAGH, L. BRUCKNER, H. K. MÜLLER, AND U. KIHM. 1986. Stellen nichtbovine Paarhufer ein IBR-Virusreservoir dar? Schweizer Archiv für Tierheilkunde 128: 557–573.

AKERSTROM, B. T., T. BRODIN, K. REIS, AND L. BJORK. 1985. Protein G: A powerful tool for binding and detection of monoclonal and polyclonal antibodies. Journal of Immunology 135: 2589–2592.

ANCRENAZ, M. 1994. Use of atipamezole to reverse xylazine tranquilization in captive Arabian oryx (*Oryx leucoryx*). Journal of Wildlife Diseases 30: 592–595.

ANDERSON, E. C., AND L. W. ROWE. 1998. The prevalence of antibody to the viruses of bovine diarrhoea, bovine herpes virus 1, Rift Valley fever, ephemeral fever and bluetongue and to *Leptospira* sp in free-ranging wildlife in Zimbabwe. Epidemiology and Infection 121: 441–449.

ANDERSON, J. 1984. Use of monoclonal antibody in a blocking ELISA to detect group specific antibodies to bluetongue virus. Journal of Immunological Methods 74: 139–149.

———, J. A. MCKAY, AND R. N. BUTCHER. 1991. The use of monoclonal antibodies in competitive ELISA for the detection of antibodies to rinderpest and peste des petits ruminants viruses. IAEA-TECDOC-623, sero-monitoring of rinderpest throughout Africa: Phase one. *In* Proceedings of the International Atomic Energy Agency, Bingerville, Cote d'Ivoire, 19–23 November 1990, pp. 43–53.

ANONYMOUS. 2002. OIE world health in 2001, part 2. Tables on animal health status and disease control methods. Office International des Epizooties, Paris, France, pp. 666–667.

BERTHIAUME, L., J. JONCAS, G. BOULAY, AND V. PAVILANIS. 1973. Serological evidence of respiratory syncytial virus infection in sheep. The Veterinary Record 93: 337–338.

BURSTEIN, H. 1975. Finite population correction for binomial confidence limits. Journal of the American Statistical Association 70: 67–69.

CONDY, J. B., K. A. J. HERNIMAN, AND R. S. HEDGER. 1969. Foot-and-mouth disease in wildlife in Rhodesia and other African territories. Journal of Comparative Pathology 79: 27–31.

DAVIES, F. G., AND A. R. WALKER. 1974. The distribution in Kenya of bluetongue diseases and antibody, and the *Culicoides* vector. Journal of Hygiene 72: 265–272.

DE DIEGO, M., E. BROCCHI, D. MACKAY, AND F. DE SIMONE. 1997. The use of the non-structural polyprotein 3ABC of FMD virus as a diagnostic antigen in ELISA to differentiate infected from vaccinated cattle. Archives of Virology 142: 2021–2033.

DU TOIT, R. M. 1962. Bluetongue–recent advances in research. The role played by bovines in the transmission of bluetongue in sheep. Preliminary communication. Journal of the South African Veterinary Medical Association 33: 483–490.

EK-KOMMONEN, C., P. VEIJALAINEN, M. RANTALA, AND E. NEUVONEN. 1982. Neutralizing antibodies to bovine herpesvirus 1 in reindeer. Acta Veterinaria Scandinavia 23: 565–569.

———, S. PELKONEN, AND P. F. NETTLETON. 1986. Isolation of a herpesvirus serologically related to bovine herpesvirus 1 from a reindeer (*Rangifer*

tarandus). Acta Veterinaria Scandinavia 27: 299–301.

ENGELS, M., M. PALATINI, A. E. MELTZER, U. PROBST, U. KIHM, AND U. ACKERMANN. 1992. Interactions of bovine and caprine herpesviruses with natural and foreign hosts. Veterinary Microbiology 33: 69–78.

EVERITT, B. S. 1977. The analysis of contingency tables. Chapman and Hall, London, UK, 128 pp.

FISCHER, J. R., L. P. HANSEN, J. R. TURK, M. A. MILLER, W. H. FALES, AND H. S. GOSSER. 1995. An epizootic of hemorrhagic disease in white-tailed deer (*Odocoileus virginianus*) in Missouri: Necropsy findings and population impact. Journal of Wildlife Diseases 31: 30–36.

FLAMAND, J. R. B. 1999. Medical aspects of Arabian oryx reintroduction. *In* Zoo & Wild Animal Medicine, M. E. Fowler and R. E. Miller (eds.). W. B. Saunders Company, Philadelphia, Pennsylvania, pp. 687–698.

FOREYT, W. J., AND J. F. EVERMANN. 1988. Response of vaccinated and unvaccinated bighorn sheep (*Ovis canadensis canadensis*) to experimental respiratory syncytial virus challenge. Journal of Wildlife Diseases 24: 356–359.

FORMENTY, F., J. DOMENECH, F. LAUGINIE, M. OUATTARA, S. DIAWARA, J. P. RAATH, D. GROBLER, Y. LEFORBAN, AND A. ANGBA. 1994. Epidemiological survey of bluetongue in sheep, cattle and various species of wildlife in Cote d'lvoire. Revue Scientifique et Technique Office International des Epizooties 13: 737–751.

FREY, H. R., AND B. LIESS. 1971. Vermehrungskinetik und Verwendbarkeit eines stark zytopathogenen Virusdiarrhoe/Mucosal Disease—Virusstammes für diagnostische Untersuchungen mit der Mikrotitermethode. Zentralblatt für Veterinärmedizin 18: 61–71.

FRÖLICH, K. 1996. Seroepizootiologic investigations of herpesviruses in free-ranging and captive deer (*Cervidae*) in Germany. Journal of Zoo and Wildlife Medicine 27: 241–247.

———, AND E. FLACH. 1998. Long-term viral serology of semi-free-living and captive ungulates. Journal of Zoo and Wildlife Medicine 29: 165–170.

———, AND M. HOFMANN. 1995. Isolation of bovine viral diarrhea virus-like pestiviruses from roe deer (*Capreolus capreolus*). Journal of Wildlife Diseases 31: 243–246.

FROST, J. W., I. WESTESTPHÄLING, AND H. KRAUSS. 1990. Seroepidemiologische Untersuchung bei Schafen in Süd- und Mittelhessen zur Verbreitung von Antikörpern gegen Border-Disease/BVD-Virus. Tierärztliche Umschau 46: 533–536.

FUKUSHI, H., T. TOMITA, A. TANIGUCHI, Y. OCHIAI, R. KIRISAWA, T. MATSUMURA, T. YANAI, T. MASEGI, T. YAMAGUCHI, AND K. HIRAI. 1997. Gazelle herpesvirus 1: A new neurotropic herpesvirus immunologically related to equine herpesvirus 1. Virology 227: 34–44.

GIBBS, E. P. J., AND M. J. P. LAWMAN. 1977. Infection of British deer and farm animals with epizootic haemorrhagic disease of deer virus. Journal of Comparative Pathology 87: 335–342.

GRETH, A., D. CALVEZ, M. VASSART, AND P.-C. LEFEVRE. 1992. Serological survey for bovine bacterial and viral pathogens in captive Arabian oryx (*Oryx leucoryx* Pallas, 1776). Revue Scientifique et Technique Office International des Epizooties 11: 1163–1168.

HAMBLIN, C., AND R. S. HEDGER. 1979. The prevalence of antibodies to ovine diarrhoea/mucosal disease virus in African wildlife. Comparative Immunology. Microbiology and Infectious Diseases 2: 295–303.

———, AND ———. 1982. Prevalence of neutralizing antibodies to bovid herpesvirus 2 in African wildlife. Journal of Wildlife Diseases 18: 429–436.

HEDGER, R. S. 1981. Foot-and-mouth disease. *In* Infectious diseases of wild mammals, J. W. Davis, L. H. Karstad, and D. O. Trainer (eds.). Iowa State University Press, Ames, Iowa, pp. 87–96.

———, AND C. HAMBLIN. 1978. Neutralising antibodies to bovid herpesvirus 1 (infectious bovine rhinotracheitous/infectious pustular vulvo-vaginitis) in African wildlife with special reference to the Cape Buffalo (*Syncerus caffer*). Journal of Comparative Pathology 88: 211–218.

HENDERSON, D. S. 1974. Were they the last Arabian oryx? Oryx 12: 347–350.

HORZINEK, M. C. 1985. Kompendium der allgemeinen Virologie, 2. Aufl. Verlag Paul Parey, Berlin, Germany, 159 pp.

INGLIS, D. M., J. M. BOWIE, M. J. ALLAN, AND P. F. NETTLETON. 1983. Ocular disease in red deer calves associated with a herpesvirus infection. The Veterinary Record 113: 182–183.

JACOBS, J. W., AND N. EDINGTON. 1975. Experimental infection of calves with respiratory syncytial virus. Research in Veterinary Science 18: 299–306.

JENNY, E. W., AND S. J. WESSMAN. 1973. Microtiter serology. Methods for bovine virology: IBR-NT (microtiter). *In* Serologic microtiter techniques for diagnostic virology, E. W. Jenny and S. J. Wessman (eds.). Veterinary Services Diagnostic Laboratory, Ames, Iowa, pp. 6–7.

KINGSBURY, D. W., M. A. BRATT, P. W. CHOPPIN, R. P. HANSON, Y. HOSAKA, V. MEULEN, E. NORRBY, W. PLOWRIGHT, R. ROTT, AND W. H. WUNNER. 1978. *Paramyxoviridae.* Intervirology 10: 137–152.

KOCK, R. A., AND C. M. HAWKEY. 1988. Veterinary aspects of the Hippotraginae. *In* Conservation and biology of desert antelopes, A. Dixon and D. Jones (eds.). Christopher Helm Ltd., Bromley, UK, pp. 75–89.

KOUMBATI, M., O. MANGANA, K. KOMIKOU, P. S. MELLOR, AND O. PAPADOPOULOS. 1999. Duration of bluetongue viraemia and serological responses in experimentally infected European breeds of sheep and goats. Veterinary Microbiology 64: 277–285.

KRONVALL, G. 1973. A surface component in group A, C and G streptococci with non-immune reactivity for immunoglobulin G. Journal of Immunology 111: 1401–1406.

LUDWIG, H., AND J. P. GREGERSEN. 1986. Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis: BHV-1 infections. Revue Scientifique et Technique Office International des Epizooties 5: 869–878.

MACKAY, D. K. J., M. A. FORSYTH, P. R. DAVIES, A. BERLINZANI, G. J. BELSHAM, M. FLINT, AND M. D. RYAN. 1998. Differentiating infection from vaccination in foot-and-mouth disease using a panel of recombinant non-structural proteins in ELISA. Vaccine 16: 446–459.

MALMQUIST, W. A. 1968. Bovine viral diarrhea—Mucosal disease. Etiology, pathogenesis and applied immunity. Journal of the American Veterinary Medical Association 156: 763–770.

MELLOR, P. S., AND J. BOORMAN. 1995. The transmission and geographical spread of African horse sickness and bluetongue viruses. Annals of Tropical Medicine and Parasitology 89: 1–15.

———, AND E. J. WITTMANN. 2002. Bluetongue virus in the Mediterranean Basin 1998–2001. Veterinary Journal 164: 20–37.

NAYERUL HAQUE, M., AND T. SMITH. 1995. Reintroduction of Arabian sand gazelle *Gazella subgutturosa marica* in Saudi Arabia. Biological Conservation 76: 203–207.

NETTLETON, P. F., E. THIRY, H. REID, AND P. P. PASTORET. 1988. Herpesvirus infections in Cervidae. Revue Scientifique et Technique Office International des Epizooties 7: 977–988.

OSTROWSKI, S., AND S. ANAJARIYAH. 2002. 2001 Middle East Arabian oryx disease survey. National Wildlife Research Center, Taif, Saudi Arabia, 33 pp.

———, E. BEDIN, D. M. LENAIN, AND A. H. ABUZINADA. 1998. Ten years of Arabian oryx conservation breeding in Saudi Arabia—Achievements and regional perspectives. Oryx 32: 209–222.

———, S. ANAJARIYAH, E. M. KAMP, AND E. BEDIN. 2002. Isolation of *Brucella melitensis* from an Arabian oryx (*Oryx leucoryx*). The Veterinary Record 150: 186–188.

QUIST, C. F., E. W. HOWERTH, D. E. STALLKNECHT, J. BROWN, T. PISELL, AND V. F. NETTLES. 1997. Host defense responses associated with experimental hemorrhagic diseases in white-tailed deer. Journal of Wildlife Diseases 33: 584–599.

REID, H. W. 1994. Deer herpesviruses. *In* Management and diseases of deer, T. L. Alexander and D. Buxton (eds.). Veterinary Deer Society Publication, London, UK, pp. 170–171.

———, D. BUXTON, I. POW, AND J. FINLAYSON. 1986. Malignant catarrhal fever: Experimental transmission of the "sheep-associated" form of the disease from cattle, deer, rabbits, and hamsters. Research in Veterinary Science 41: 76–81.

ROSSITER, P. B. 1994. Rinderpest. *In* Infectious diseases of livestock, Vol. 2, J. A. W. Coetzer, G. R. Thomson, and R. C. Tustin (eds.). Oxford University Press, Cape Town, South Africa, pp. 735–757.

———. 1996. The world without rinderpest: Epidemiological and clinical features of rinderpest in the 1990s. *In* Proceedings of the FAO technical consultation on the global rinderpest eradication programme, Rome, Italy, 22–24 July. FAO Animal Production and Health Paper, 129 pp.

———, L. KARSTAD, D. M. JENETT, T. YAMAMOTO, A. H. DARDIRI, AND E. Z. MUSHI. 1983. Neutralising antibodies to rinderpest virus in wild animal sera collected in Kenya between 1970–1981. Preventive Veterinary Medicine 1: 257–264.

SOINE, C., G. UATANAUA, AND K. R. DEPNER. 1992. Prevalence of antibodies to bovine diarrhoea virus in Namibian wildlife. Tropical Animal Health and Production 24: 125–126.

SPALTON, J. A., M. W. LAWRENCE, AND S. A. BREND. 1999. Arabian oryx in Oman: Successes and setbacks. Oryx 33: 168–175.

SPEARMANN, R., AND G. KÄRBER. 1985. Das Virus als Partikel. *In* Kompendium der allgemeinen Virologie, 2nd Edition, M. C. Horzinek (ed.). Paul Parey, Berlin, Germany, pp. 22–23.

SPRAKER, T. R., AND J. K. COLLINS. 1986. Isolation and serologic evidence of a respiratory syncytial virus in bighorn sheep from Colorado. Journal of Wildlife Diseases 22: 416–418.

THEVASAGAYAM, J. A. 1998. The epizootic haemorrhagic disease virus serogroup. PhD Thesis, University of Hertfordshire, Hertfordshire, UK, 152 pp.

———, P. P. MERTENS, J. N. BURROUGHS, AND J. ANDERSON. 1995. Competitive ELISA for the detection of antibodies against epizootic haemorrhagic disease of deer virus. Journal of Virological Methods 55: 417–425.

THOMSON, G. R. 1994. Foot and mouth disease. *In* Infectious diseases of livestock, J. A. W. Coetzer, G. R. Thomson, and R. C. Tustin (eds.). Oxford University Press, Cape Town, South Africa, pp. 825–852.

VAN CAMPEN, H., AND G. EARLY. 2001. Orthomyxovirus and paramyxovirus infections. *In* Infectious diseases of wild mammals, E. S. Williams and I. K. Barker (eds.). Manson Publishing/Veterinary Press, London, UK, pp. 271–279.

———, K. FRÖLICH, AND M. HOFMANN. 2001. Pes-

tivirus infections. *In* Infectious diseases of wild mammals, E. S. Williams and I. K. Barker (eds.). Manson Publishing/Veterinary Press, London, UK, pp. 232–237.

WILSON, D. E., AND D. M. REEDER. 1993. Mammal species of the world—A taxonomic and geographic reference, 2nd Edition. Smithsonian Institution Press, Washington, D.C., 1206 pp.

WOODFORD, M. H. 1989. Veterinary aspects of the reintroduction of the Arabian oryx into Saudi Arabia. *In* Proceedings of the First Symposium, National Commission for Wildlife Conservation and Development [Publication 3], A. H. Abuzin-ada, P. D. Goriup, and I. A. Nader (eds.), NCWCD, Riyadh, Saudi Arabia, pp. 393–399.

———, AND P. B. ROSSITER. 1994. Disease risks associated with wildlife translocation projects. *In* Creative conservation: Interactive management of wild and captive animals, P. J. S. Olney, G. M. Mace, and A. T. C. Feistner (eds.). Chapman and Hall, London, UK, pp. 178–200.

WOODS, L. W. 2001. Adenoviral diseases. *In* Infectious diseases of wild mammals, E. S. Williams and I. K. Barker (eds.). Manson Publishing/Veterinary Press, London, UK, pp. 202–212.