MORBIDITY AND MORTALITY FACTORS IN KEY DEER (*ODOCOILEUS VIRGINIANUS CLAVIUM*)

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ABSTRACT: The population health of endangered Key deer (*Odocoileus virginianus clavium*) was monitored from 10 February 1986 to 28 September 2000 by necropsy of animals that were killed by vehicles, euthanized because of terminal injuries or disease conditions, or found dead. The predominant mortality factor during the period was collision with motor vehicles; however, several infectious diseases were diagnosed, including infections with *Arcanobacterium pyogenes*, *Haemonchus contortus*, *Salmonella* spp., and *Mycobacterium avium* subsp. *paratuberculosis*. During the period monitored, the only infectious disease that was thought to have affected population dynamics was haemonchosis. Nevertheless, several of the observed diseases have potential to impact viability of the Key deer population under appropriate environmental conditions.

Key words: *Arcanobacterium pyogenes*, *Haemonchus contortus*, Johne's disease, Key deer, *Mycobacterium avium* subsp. *paratuberculosis*, *Odocoileus virginianus clavium*, salmonellosis, white-tailed deer.

INTRODUCTION

Florida Key deer (*Odocoileus virginianus clavium*) are the smallest race of North American white-tailed deer and have been listed as endangered since 1967 because of habitat loss and fragmentation, diminishing numbers, and concern for loss of genetic diversity. Key deer distribution is restricted to approximately 12 islands (keys) of the Lower Florida Keys (Florida USA), with the majority of the population residing on Big Pine and No Name Keys (24°44′N, 81°20′W). Deer numbers were estimated at fewer than 100 animals in the 1940s and 1950s (Allen, 1952; Dickson, 1955; US Fish and Wildlife Service, 1985), but the population had increased by the early 1970s to an estimated 350–400 deer (Silvy, 1975; Klimstra et al., 1978). However, further study indicated that the Key deer population was declining (Humphrey and Bell, 1986).

Records of deer mortality have been kept continuously since 1969 by personnel at the National Key Deer Refuge (NKDR); however, causes of death were assigned to general categories by NKDR staff primarily by case history or circumstantial evidence. The Southeastern Cooperative Wildlife Disease Study (SCWDS) began to assist with assessment of Key deer health in 1986 through examination of samples submitted from road kills or releasable animals examined by NKDR biologists.

In November 1996, an emaciated adult doe was presented to a NKDR biologist for examination and abnormal appearing ileum and associated ileocecal lymph node were submitted to SCWDS. Subsequent histopathology and polymerase chain reaction diagnostic procedures revealed infection with *Mycobacterium avium* subsp. *paratuberculosis* (Quist et al., 2002). The morphologic diagnosis of Johne's disease in this animal provided the impetus for more detailed study of morbidity and mortality factors in Key deer. Reported herein is health-related information for Key deer with emphasis placed on animals examined from February 1997 to September 2000.

MATERIALS AND METHODS

On 10 February 1986, assistance to the NKDR was initiated when a SCWDS field

team went to Big Pine Key to demonstrate basic necropsy and deer herd health evaluation procedures to refuge staff. During this trip, five road-killed Key deer were examined. From that time through November, 1996, an additional 156 Key deer were necropsied by NKDR refuge biologists, and SCWDS received frozen abomasums for stomach worm counts and/or serum samples. Following the discovery of a probable case of Johne's disease in 1996, health monitoring was intensified, and SCWDS veterinarians and biologists made eight site visits from 10 April 1997 to 28 September 2000. In the presentation of mortality results, only necropsy data obtained by veterinarians during the latter period were used.

Key deer killed by vehicles, euthanized because of terminal injuries or conditions, or found dead were held frozen prior to examination and were necropsied by SCWDS field teams during site visits. The quality of carcasses for necropsy ranged from good to marginal. Exceptions were two recently dead deer that were shipped to SCWDS headquarters refrigerated because they were Johne's disease suspects. The age, sex, and body weight were recorded, and the necropsy protocol described by Nettles (1981) was used except many of the specific procedures to recover parasites were omitted in most cases. Physical condition was rated based on muscle mass and body fat (Stockle et al., 1978). Because necropsies were oriented toward determination of cause of morbidity or mortality, diagnostic procedures varied among cases. In most instances, the cause of death was trauma, and additional diagnostic testing was not needed. Follow-up laboratory testing was done when needed. When laboratory tests were required, the appropriate samples were preserved by refrigeration and returned to The University of Georgia's College of Veterinary Medicine (Athens, Georgia, USA) or sent to collaborating laboratories. When abscesses were encountered, swabs of exudate were plated on blood and MacConkey agars. All organisms were identified by API Systems (bio-Merieux Vitek, Inc., Hazelwood, Missouri 63042, USA).

Deer that were being captured, marked, and released by refuge biologists were examined as opportunities became available during each of eight field visits. Each animal was given a brief examination for ectoparasites; representative samples were taken and submitted to the National Veterinary Services Laboratories (NVSL), Ames, Iowa, USA. Blood samples were taken in vacuum tubes for serum sampling. Serum samples were tested for antibodies to bluetongue (BT), epizootic hemorrhagic disease (EHD), bovine virus diarrhea (BVD), infectious bovine rhinotracheitis (IBR), and parainfluenza 3 (PI3) viruses. Samples also were tested for antibodies to *Brucella*, six serovars of *Leptospira*, and *M. avium* subsp. *paratuberculosis*. Commercial immunodiffusion test kits were used for BT and EHD (Veterinary Diagnostic Technology, Wheat Ridge, Colorado, USA) and *M. avium* subsp. *paratuberculosis* (ImmuCell Corp., Portland, Maine, USA). Microneutralization assays (Degegt et al., 2000) were performed for BVD, IBR, and PI3 viruses using the NADL strain of BVD virus and field isolates of IBR and PI3 viruses. The rose bengal plate agglutination and microscopic agglutination tests were used for *Brucella* and *Leptospira*, respectively, according to recommended methods (Office of International Epizootics, 1996).

Evening road counts were conducted monthly by NKDR personnel to serve as a population index for Key deer on Big Pine and No Name keys. A predesignated 73 km route was driven beginning at 10:00 PM. During these road counts, deer seen were recorded, and sex and age (fawn, yearling, adult) were estimated.

RESULTS

Between 13 February 1997 and 28 September 2000, necropsies were performed on 170 of 442 (38.4%) Key deer that were known to have died. There were substantially more males than females in the sample (122 of 170, 71.7%). The age and sex representation in the sample was as follows: <1 yr old: 34 males and 14 females; 1–2 yr old: 26 males and 10 females; and >2 yr old: 62 males and 24 females.

Highway mortality

Of the 170 Key deer carcasses examined, 127 (74.7%) were known or presumed hit by automobiles based on history and/or compatible lesions. Necropsy revealed that 111 of 127 (87.4%) of the road-killed Key deer had no gross lesions other than those attributable to vehicle-related trauma. For these 111 animals, physical condition was rated as excellent for 32 deer, good for 59, fair for 17, and poor for three. The 16 remaining road-killed deer either had debilitating conditions thought to have predisposed them to automobile collision ($n=7$) or they had incidental lesions judged to represent subclinical

health problems (n=9). Debilitating conditions thought to have predisposed the animals to automobile collisions were intracranial abscesses or purulent meningoencephalitis in six adult males, and parasitic gastritis (haemonchosis) in one fawn. The secondary conditions included subclinical purulent dermatitis and/or osteomyelitis in five adult males, subclinical parasitic gastritis caused by *Haemonchus contortus* in three male fawns, and cardiomyopathy due to previous trauma in one adult male.

Chronic purulent infections

Second to vehicle-related mortality, the most frequent health problem encountered was an array of chronic purulent infections. Thirty-three of 170 (19.4%) Key deer examined had either clinical or subclinical bacterial infections, most of which were located in the cranial region. Of the group with chronic purulent infections, 17 (48.5%) had neurologic disease caused by intracranial abscesses and/or purulent meningoencephalitis. As stated in the previous section, six of these deer with neurologic disease were killed by automobiles. Another three drowned, and eight deer either were found dead or were euthanized. All of these 17 deer were adult males, and only one was less than 3 yrs old. Only four of the 17 afflicted deer had normal-appearing heads. The other 13 had missing antlers (n=5), open wounds or exposed cranial bones around the antler pedicels (n=5), atypical antler configuration (n=4), or old scars in the poll region (n=2). Their condition was compatible with what has been previously described as an intracranial abscessation/suppurative meningoencephalitis complex attributable mainly to *Arcanobacterium pyogenes* (Davidson et al., 1990). In Key deer, *A. pyogenes* was isolated in 15 of 16 neurologic cases in which culture was attempted. Other bacteria cultured from the brains or meninges included *Bacteroides* sp. (n=1); *Citrobacter* sp. (n=1); *Corynebacterium* sp. (n=2); *Enterobacter* sp. (n=1); *Pasteurella multocida* (n=1); *Serratia marcesens* (n=1); *Staphyloccus aureus* (n=4); and *Staph. sciuri* (n=1).

Chronic purulent infections also were found in locations other than the central nervous system, viz., poll region (n=22); retro-orbital tissues (n=6); middle ear (n=2); mandible (n=2); fascial planes and ligamentum nuchae of neck (n=4); lungs (n=4); and distal leg joints and tendon sheaths (n=4). Many of these abscesses contained large amounts of creamy yellow-green pus and resulted in extreme debilitation or death. Chronic purulent infections affecting regions other than the central nervous system were responsible for the death or euthanasia of 11 animals. In total, *A. pyogenes*, along with a variety of other bacteria, was isolated from lesions in 27 of the 33 Key deer with chronic purulent infections. All but one of the 33 Key deer afflicted with chronic purulent infections were males. Of the 32 infected males, 22 had evidence of trauma to the head region such as missing antlers, open wounds or scars around the antlers, or atypical antlers.

Haemonchosis

A third mortality factor observed was the malnutrition/parasitism syndrome in fawns (<1 yr old) associated with large stomach worm (*H. contortus*) infection. This condition was observed only in the winter of 1997. Five emaciated fawns (born in the Spring 1996) had roughened hair coats, pale body tissues, and thin, watery blood. Ascites and submandibular edema also were seen in two of these animals. In addition to these deer, which died or were euthanized, a sixth fawn killed by a vehicle in 1997 also had characteristic lesions. Worm burdens for five clinically ill fawns ranged from 15.4 to 352 adult nematodes per kg of body weight (BW) (mean 135 worms/kg BW). Three of the five affected fawns had infections that exceeded the 75 worms/kg BW pathogenic threshold guideline proposed previously (Davidson et al., 1980). One emaciated

TABLE 1. Burden of *Haemonchus contortus* in Key deer fawns

Season/Year	fawns examined	*Haemonchus* mean, ±SD (range)	Body wt (kg) mean, ±SD (range)	
Winter 1997	6*	1,140, ±1,202 (60–2,980)	10.8, ±3.13 (6.4–15.0)	128.9, ±147.6 (4.0–352.2)
Fall 1997	4	1,050, ±937 (380–2,420)	13.1, ±4.22 (9.6–19.1)	89.5, ±89.9 (27.2–221.8)
Winter 1998	1	140, – (–)	18.2, – (–)	9.9, – (–)
Fall 1998	10	124, ±129 (0–440)	14.8, ±3.96 (9.5–19.8)	7.9, ±6.76 (0–22.3)
Winter 1999	7	266, ±221 (40–620)	20.91, ±2.62 (18.6–26.4)	13.0, ±10.55 (1.5–29.6)
Fall 1999	3	267, ±94 (180–330)	14.9, ±2.92 (12.7–18.2)	18.0, ±6.29 (12.6–24.9)
Winter 2000	3	280, ±197 (60–440)	13, ±4.58 (8.2–17.3)	19.2, ±10.34 (7.3–25.5)
Late summer 2000	6	37, ±29 (0–60)	11.4, ±3.25 (7.3–13.6)	3.2, ±2.88 (0–7.3)

* Five of six fawns had clinical disease

FIGURE 1. Average deer seen on USFWS monthly road counts (bars) and annual Key deer mortality (line) on Big Pine and No Name keys, 1976–2000.

fawn treated with ivermectin prior to death did not have nematodes.

Abomasal parasite burdens in fawns that died during the winter and fall sampling periods are presented in Table 1. Worm numbers were highest in winter and fall 1997. By winter of 1998, few fawns were present in the population as evidenced by the single fawn available for necropsy. Key deer census counts made by refuge biologists showed that the fawn:doe ratio declined from 0.28:1 in October 1997 to 0.07:1 in March 1998 (NKDR, unpubl. data). This appearance of clinical haemonchosis corresponded with the peak numbers for annual known mortality and total numbers of Key deer seen in census routes (Fig. 1).

Haemonchus contortus was shown to be endemic in the Key deer population during the first 10 yr of monitoring as it was found routinely in abomasa submitted by NKDR biologists; however, pathogenic levels were rarely encountered. *Haemonchus contortus* burdens exceeded 1,000 worms in only one fawn and one adult deer during this period, and both animals were rated in poor condition. Between February 1986 and November 1996, mean *H. contortus* burdens in 55 adult Key deer and 19 Key deer fawns were 141 worms (SD±164.9, range 0–1,140 worms) and 217 (SD±553.1, range 0–2,760 worms), respectively.

Miscellaneous mortality factors

There were two Key deer with enteric infections. Salmonellosis was diagnosed in a 10 mo old male that was found in a weakened condition and died within minutes after capture. Emaciation, dehydration, and diarrhea were noted. Histopathology of intestinal tract revealed necrosis of the villi, congestion, and neutrophil infiltration in the ileum. *Salmonella enterica* serotype *hartford* was isolated from intestinal contents.

A 2.5 yr old doe captured on 16 July 1998 was emaciated and had chronic diarrhea. A rectal swab taken at the time of capture yielded *S. enterica* serotype *weltevreden* (group E-1). The deer was taken to a veterinarian for treatment, but its condition deteriorated, and euthanasia was performed 4 days later. *Salmonella enterica* serotype *kralendyl* was isolated at necropsy. *Mycobacterium avium* subsp. *paratuberculosis* also was isolated from this doe (Quist et al., 2002).

Three deer (two adult does, one male fawn) were killed by dogs, and one adult doe apparently drowned with no predisposing problem. Two deer had cardiac insufficiency. Vegetative valvular endocarditis was diagnosed in a 1 yr old male and a ventricular septal defect was found in a 3.5 yr old male. There were two deer that died of toxicoses of undetermined cause. Individual deer died because of fence entanglement ($n=1$); trap entanglement ($n=1$); and chronic, post-traumatic urethral obstruction ($n=1$). Euthanasia was performed on a debilitated adult doe with a chronic fracture of the elbow joint and an adult doe with a widely metastasized neuroendocrine tumor.

Nonclinical parasitism

In addition to *H. contortus*, other parasites seen included two additional nematode parasite species (*Dictyocaulus viviparus* and *Trichuris* sp.); three ectoparasite species (*Ixodes scapularis*, *Solenopotes binipilosus*, and *Damalinia* sp.); and one protozoan parasite (*Sarcocystis* sp.). Chewing lice, *Damalinia* sp., were seen occasionally in large numbers on young deer that were in poor condition attributed to malnutrition and/or haemonchosis. This parasite is an undescribed species seen in Key deer previously (J. Mertins, NVSL, pers. comm.). The other ectoparasite encountered was the black-legged tick, *I. scapularis*. Three deer were infested with either one or two ticks per animal. Fleas were observed on Key deer that frequented areas near cats and dogs; however, samples were not obtained for identification.

Serology

All adult Key deer (>1 yr old) were serum test negative for antibodies to *Brucella abortus* ($n=99$), BVD virus ($n=108$), IBR virus ($n=106$), PI3 virus ($n=106$), and *M. avium* subsp. *paratuberculosis* ($n=122$). Nineteen of 113 (16.8%) Key deer were positive for bluetongue virus antibodies, and 17 of 111 (15.0%) Key deer were positive for EHD virus antibodies. Of the 21 animals that had either BTV or EHDV antibodies, 15 reacted to both tests. *Leptospira* serology tests for 109 adult Key deer yielded the following results: two deer had 1:100 titers against serovar bratislava; one deer had a 1:100 titer against serovar canicola; 14 deer had titers against serovar gryppotyphosa (eight at 1:100, four at 1:200, one at 1:400, one at 1:6,400); four deer had 1:100 titers against serovar hardjo; one deer had a 1:100 titer against serovar icterohemorrhagiae; and one deer had a 1:100 titer against serovar pomona.

DISCUSSION

Highway mortality was a major cause of death in the Key deer population. However, an accurate estimate of its importance relative to other types of mortality is difficult to obtain. Our necropsy results, which indicated that highway mortality composed approximately 75% of the deaths in the sample, probably over-represented this type of mortality. In contrast, NKDR records estimate highway mortality at approximately 65% of observed deaths

(NKDR, unpubl. data). Highway mortality represented even a lower percentage (50%) in recent (NKDR, unpubl. data) and older (Hardin, 1974) radiotelemetry studies conducted with Key deer. The 50% estimate for highway mortality may be more accurate for two reasons. First, there is an inherent bias towards highway mortality because it is the most likely mortality event to be observed. Second, dead deer discovered in remote locations often were decomposed and unsuitable for necropsy. These decomposed deer were more likely to have died of causes other than highway collision, but often were classified as mortality of "unknown" cause.

The necropsy sample was heavily represented by male Key deer (72%), which could be attributed to their much more mobile behavior, as demonstrated by telemetry and mark-recapture studies (NKDR, unpubl. data). Greater mobility would make males more vulnerable to highway mortality. However, previous studies have shown that the fetal sex ratio of Key deer is strongly biased toward males. Hardin (1974) found a male:female fetal sex ratio of 1.45:1 (59% males) in the examination of 26 does. Thus, the skewed sex ratio also helps explain the preponderance of male deer as well.

The majority of deer killed on the roads were normal animals in good to excellent condition, and we concluded that overall health of the Key deer population was acceptable during the period monitored. However, this survey did provide evidence that infectious diseases and parasites were impacting the herd. Although the chronic purulent infections in adult male deer were more prevalent than haemonchosis in fawns, we believe that the latter has a greater potential to affect deer population numbers. Poor fawn survival has been linked to stomach worm infections in combination with poor nutrition in previous studies (Prestwood et al., 1973; Eve and Kellogg, 1977; Davidson et al., 1980). We believe that the precipitous drop in 1997 Key deer fawn crop was caused by this problem.

The abomasal parasite count (APC) has been a routine parameter used to gauge the health and nutritional status of white-tailed deer herds in the southeastern United States (Eve and Kellogg, 1977). The APC technique was developed from deer herds that were infected with multiple species of abomasal parasites, viz., *Haemonchus, Mazammastrongylus, Ostertagia,* and *Trichostrongylus,* and mean total worm burdens of >1,500 worms per deer (n=5 or more deer) were considered predictive of deer over-population in relation to nutritional capacity of the habitat. Based on our observations of Key deer, it appears that traditional APC guidelines would not be useful in predicting nutritional status for white-tailed deer when a population is infected only with *H. contortus*. *Haemonchus contortus* has been shown to stimulate an immune response that results in much lower worm numbers in animals that survive initial infection (McGhee et al., 1981). Thus, intensities of infection of >1,500 *H. contortus* per adult deer are unlikely to occur; however, the Key deer data do reinforce the concept that the presence of even low intensities of *H. contortus* in adult deer should provide reason to suspect problems in fawns (Davidson et al., 1980). In addition, the long-term presence of this parasite in Key deer demonstrates its capacity to be maintained without support from domestic cattle, sheep, or goats.

Chronic purulent infections caused by *A. pyogenes* are strongly associated with adult male deer, which are present in the Key deer population in surplus to their biologic need for reproduction. Therefore, the impact of these infections on Key deer reproduction and population viability appears minimal at present. Nevertheless, the chronic purulent infection syndrome associated with *A. pyogenes* is important in the day-to-day management of this urban deer population because of the behavioral changes and chronic debilitation caused in

fected deer and the public's reaction to ese sick animals. Because of this disease ndrome, refuge personnel are forced to end many hours reacting to situations inlving affected deer.

Confirmation of Johne's disease in Key er provides an unusual opportunity to luate the capability of white-tailed deer maintain *M. avium* subsp. *paratuberlosis*. Cattle and sheep are not present the area, and goats, when present, are re. Current data neither confirm or rete that there is a herd-based problem th this disease. Although there were unirmly negative results from the serologic sting, this is not surprising. Only one er had physical evidence of Johne's dis-, and serologic tests for Johne's disease e of limited value in early infection (Cols, 1996). A long-term surveillance effort s been initiated that includes evaluation deer examined in this study for clinical ane's disease.

Although the endangered Key deer is a ique race of white-tailed deer, the Key er population serves as a model for what y occur in other environmentally coned urban white-tailed deer populations. e current Key deer population probably s reached or slightly exceeded the longm carrying capacity of the available bitat and death losses are beneficial to set reproduction and maintain populan stability. The total protection of the y deer herd has resulted in a system ereby human-induced vehicle collisions a major mortality pressure acting upon Key deer population, but more "nat- mortality factors caused by infectious eases (haemonchosis, *A. pyogenes* intions, Johne's disease, salmonellosis) rently appear to be important as well. rthermore, there is no guarantee that population dynamics of the Key deer l will remain stable, and there is con- n that a change in environmental conons could enhance an infectious dise. For example, concentration of Key r at illegal feeding sites created by local idents could enhance spread of the aforementioned infectious agents. Although a relationship between artificial feeding and infectious agents is difficult to prove, the diseases in question are potentially capable of causing greater impact on the Key deer population and should be watched carefully.

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