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EPIDEMIOLOGY OF CHRONIC WASTING DISEASE IN CAPTIVE ROCKY MOUNTAIN ELK

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ABSTRACT: Between June 1986 and May 1997, chronic wasting disease (CWD) was the only natural cause of adult mortality among captive Rocky Mountain elk (*Cervus elaphus nelsoni*) held at a wildlife research facility near Fort Collins, Colorado (USA). Of 23 elk that remained in this herd >15 mo, four (17%) developed CWD. All affected elk were unrelated females from the founding cohort, captured as neonates and raised in 1986. The index case was diagnosed in 1989; time intervals between subsequent cases ranged from 13 to 32 mo. Initial age at onset of clinical signs ranged from about 2.9 to 8.1 yr; duration of clinical disease ranged from 5 to 12 mo (mean = 7.5 mo) prior to death. Intraspecific lateral transmission of CWD seemed the most plausible explanation for the epidemic pattern observed; neither periparturient nor maternal transmission appeared necessary to sustain this outbreak. Early detection and elimination of incubating or clinical individuals may have aided in reducing exposure or infection rates as compared to a previous outbreak in the same facility. Transmission routes and rates, pathogenesis, antemortem diagnostic tools, and the potential role of reservoirs or environmental contamination in perpetuating CWD epidemics warrant further investigation.

Key words: *Cervus elaphus nelsoni*, chronic wasting disease, epidemiology, Rocky Mountain elk, transmissible spongiform encephalopathy.

INTRODUCTION

Chronic wasting disease (CWD), a transmissible spongiform encephalopathy of some North American cervid species, was first recognized in captive deer (*Odocoileus* spp.) held at wildlife research facilities in Colorado and Wyoming (USA) (Williams and Young, 1980). Captive Rocky Mountain elk (*Cervus elaphus nelsoni*) from both Colorado and Wyoming facilities were subsequently diagnosed with CWD (Williams and Young, 1982). Clinical and pathologic features of CWD are well-described (Williams and Young, 1980, 1982, 1992, 1993), but several important questions about its epidemiology remain unresolved. Here, we report epidemiological observations made on a captive Rocky Mountain elk herd affected by CWD.

MATERIALS AND METHODS

All observations were made at the Colorado Division of Wildlife's (CDOW) Foothills Wildlife Research Facility (FWRF) (Fort Collins, Colorado, USA; 40°355'N, 105°10'W). This approximately 15 ha facility was built in the 1960's and 1970's to support ongoing research on various aspects of native ruminant ecology, physiology, and management. The FWRF's original populations of deer and elk were infected with CWD (Williams and Young, 1980, 1982, 1992). Details of husbandry and facility management related to CWD at the FWRF prior to 1985 have been described previously (Williams and Young, 1980, 1982, 1992).

In 1985, all cervids residing at the FWRF and its satellite facilities were killed in an attempt to eradicate CWD. Paddocks where affected cervids had resided were treated with 1,000 ppm calcium hypochlorite solution (65% available chlorine) via mobile sprayer and helicopter (Neil, 1985), plowed to a depth of about 0.3 m, and treated a second time; shelters, feed bunkers, and automatic waterers were either replaced or hand cleaned twice with 1,000 ppm calcium hypochlorite solution. Subsequently, the FWRF's perimeter was enclosed by a 2.5 m game-proof woven wire fence that provided spatial buffers of ≥6 m between the perimeter fence and all animal paddocks to preclude fence-line contact between research animals and free-ranging cervids or domestic livestock. Paddocks destined to house the new elk herd also were separated from bighorn sheep (*Ovis canadensis*) and mountain goat (*Oreamnos americanus*) paddocks by ≥1 m

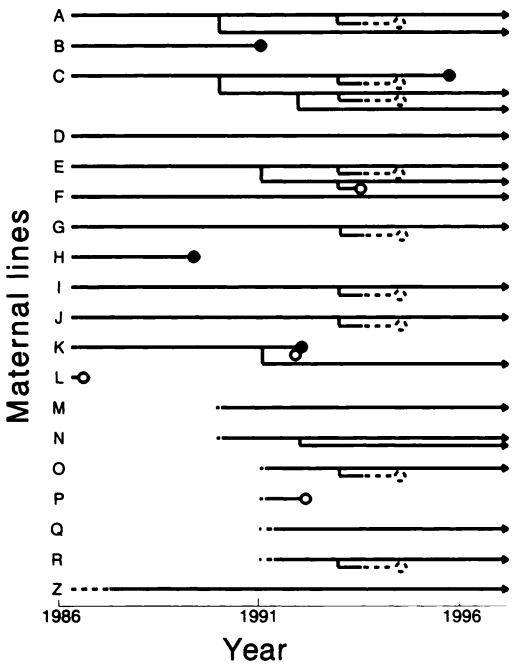

FIGURE 1. Between June 1986 and May 1997, 36 elk representing 19 maternal lineages entered a captive herd at the Foothills Wildlife Research Facility (FWRF). Four of these died or were euthanized on site (○) and nine others were removed from the herd (broken ○) before reaching 15 mo of age; none showed signs or pathology of CWD. Of 23 elk that remained in the FWRF herd >15 mo, chronic wasting disease was confirmed in four (17%) (●). Years are represented in a biological context and consequently begin in June; broken lines indicate time periods and events occurring outside the FWRF.

spatial buffers. The future elk paddocks were left fallow for >12 mo. No hoofed stock other than elk used these paddocks after 1986.

The FWRF's second Rocky Mountain elk herd was started in June 1986 with 12 <1-wk-old wild-born female calves (Fig. 1). All of these calves were captured in Rocky Mountain National Park, Colorado (40°10′ to 40°33′N, 105°30′ to 105°58′W) during June 1986. Additions to the herd after 1986 included births of two calves in 1990, two in 1991, three in 1992, and 10 in 1993 (Fig. 1). We also acquired a dam-raised yearling male in 1987, as well as two calves in 1990 and four calves in 1991 (Fig. 1); additions not born on site originated in Colorado, but outside Larimer County. In all, 19 different maternal lineages were represented in this herd between 1986 and 1997.

Twenty of the calves were bottle-raised using undiluted evaporated milk in protocols described previously (Wild and Miller, 1991; Wild et al., 1994); 18 of these were raised on site, and two entered the FWRF herd after weaning at about 4 mo of age. The other 16 elk were dam-raised, all but one on-site. After weaning, elk were maintained on alfalfa (long stem or cubed), grass hay, and a pelleted high-energy supplement (Baker and Hobbs, 1985) as prescribed under established feeding protocols for respective age and sex classes (Miller, 1990); fresh water and mineralized salt blocks were provided ad libitum. Natural forage dominated by buffalograss (*Buchloe dactyloides*), blue gramma (*Chondrosom gracilis*), cheatgrass brome (*Bromus tectorum*), and kochia (*Kochia scoparia*) was available in paddocks where elk were housed. Aside from evaporated milk, no animal-derived proteins were included in elk diets.

This elk herd was divided among four 2 to 3 ha paddocks in various assemblages between 1986 and 1997; although subgroupings were somewhat random and dynamic, we tended to house smaller individuals together and to segregate males and castrated males from females. All elk were observed daily by animal caretakers and weighed (±0.5 kg) at about monthly intervals. In addition to routine handling, most individuals were used in one or more research experiments between 1986 and 1997.

We either observed initial health problems or were notified by caretakers. Initial problems included behavioral changes or unexplained or persistent weight loss. Elk showing these signs, consistent with early stages of CWD, were subsequently regarded as "CWD suspects"; such individuals were intermittently placed in 25 m^2 isolation pens for observation and further diagnostic evaluation. Once we made a clinical diagnosis of CWD, the affected animal was killed by lethal injection or electrocution under deep anesthesia; we used intravenous pentobarbital sodium solution (Beuthanasia®-D Special; Schering-Plough Animal Health Corporation, Kenilworth, New Jersey, USA) or a combination of embutramide, mebezonium iodide, and tetracaine hydrochloride (HCl) (T-61®; Hoechst-Roussel Agri-Vet Company, Somerville, New Jersey, USA) for lethal injections and used xylazine HCl (≥2 mg/kg intramuscularly; multiple sources) to induce deep anesthesia prior to electrocution.

We performed complete necropsies on all elk that died and collected representative tissues from all major organ systems. Tissues were fixed in 10% neutral buffered formalin, and subsamples of brain and spleen also were stored at −70 C. Fixed tissues were subsequently embedded in paraffin, sectioned at 5 to 6 μm, and stained with hematoxylin and eosin (H&E). Fresh brain from one case also was ex-

amined via negative-stain electron microscopy for presence of scrapie-associated fibrils (SAFs) (Merz et al., 1981). Carcasses were either incinerated or buried on site.

RESULTS

Between June 1986 and May 1997, CWD was the only natural cause of adult mortality in elk at the FWRF. Of the 36 captive elk that entered the FWRF herd over this period, two died and two were killed before reaching 15 mo of age. One of these deaths was attributed to *Clostridium sordellii* infection and another to unspecified bacteremia; one healthy calf was killed shortly after birth along with its CWD-affected dam, and a 14-mo-old castrated male was killed after sustaining a pelvic fracture. Nine other elk were removed from the herd at about 6 mo of age and subsequently killed as part of another study conducted in an off-site isolation facility (Fig. 1). None of these juveniles showed signs or pathology associated with CWD. Of the 23 elk that remained in this herd >15 mo, four (17%) developed CWD (Fig. 1).

All affected elk were females and were members of the founding cohort captured and raised in 1986. The index case was confirmed by histopathology in September 1989, followed by confirmed cases in May 1991, June 1992, and February 1995. In each case, the affected elk had been classified as a CWD suspect for 5 to 12 mo prior to euthanasia (mean = 7.5 mo) (Fig. 1). Initial age at onset of clinical signs ranged from about 2.9 to 8.1 yr (Fig. 1). The time intervals between cases (date of death to date of death) were 19, 13, and 32 mo (mean = 21 mo) (Fig. 1, 2).

Early clinical signs were weight loss (Fig. 2) and subtle behavioral changes. Although marked seasonal trends in elk body weights were observed annually (Fig. 2), CWD-affected elk were unable to regain lost weight even when provided with ad libitum quantities of high-quality feed. Isolation, anorexia, repetitive behaviors, hyperesthesia, and intractability were the most consistent behavioral changes we observed. Three of the four cases were terminated before severe signs developed; the index case was allowed to progress, and that animal showed relatively severe emaciation, marked behavioral changes, excessive salivation, odontoprisis, fine tremors, and mild ataxia prior to death.

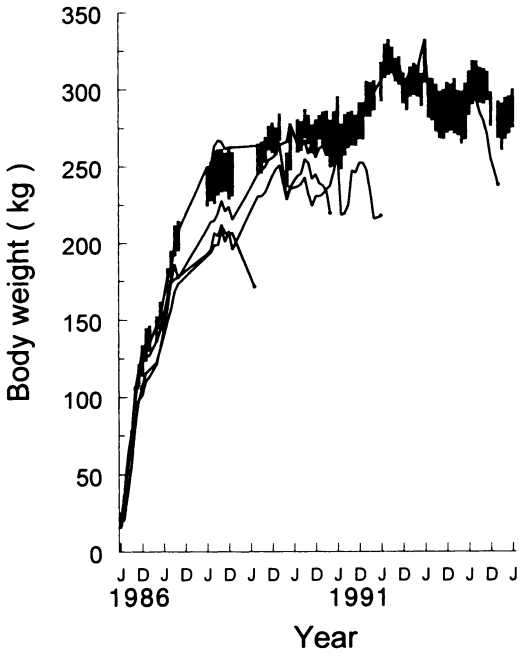

FIGURE 2. Persistent weight loss accompanied other clinical signs of chronic wasting disease in elk affected between 1986 and 1995. Weight dynamics of affected individuals (black) generally followed average 1986 cohort trends (gray) until 6 to 12 mo prior to euthanasia (●). Gray bars are ±2 standard errors of mean weights of unaffected 1986 cohort members.

The primary gross lesions were mild to severe emaciation accompanied by reduction or generalized absence of subcutaneous, visceral and skeletal adipose tissue. One female was pregnant with a single fetus and another had delivered a small but normal calf 4 days prior to death.

Our clinical diagnosis of CWD was confirmed by histopathology in all four cases. The predominant and most consistent lesions were in the brain and spinal cord. Spongiform encephalopathy, characterized by microcavitation primarily of the gray matter with single or multiple intracyto-

plasmic vacuoles in neuronal perikarya and neuronal degeneration, was found in neural tissues; lesions were distributed as previously described (Williams and Young, 1993). The severity and distribution of lesions did not vary appreciably among cases, despite some observed differences in the stage of clinical disease at euthanasia. Electron microscopy demonstrated SAFs in brain tissue from the single case examined.

DISCUSSION

Chronic wasting disease has been the predominant natural source of mortality in the FWRF's captive elk herds for over two decades. Since 1986, CWD was the sole source of adult mortality in this captive elk herd. Similarly, CWD accounted for five of seven (71%) natural deaths (or humane terminations) among adults in the original elk herd maintained at the FWRF between 1976 and 1985 (Williams and Young, 1982; CDOW, unpubl. data) (Fig. 3). We believe comparisons of these two natural, independent, and largely uncomplicated CWD outbreaks in the FWRF's captive elk offer valuable insights into the epidemiology of CWD in elk.

In addition to CWD's role in adult mortality, there are other striking similarities between the original and recent CWD outbreaks in the FWRF elk herds. In each outbreak, the index case occurred about 3 yr after the herd was established. Subsequent cases arose 2 to 3 yr later, with additional cases 2 to 2.5 yr thereafter (Figs. 1, 3). Single cohorts of maternally (and probably paternally) unrelated individuals accounted for most CWD cases in the initial outbreak and all cases in the recent outbreak. Ages of affected individuals ranged from about 3 to 7 yr in the 1976 cohort and about 3 to 8 yr in the 1986 cohort.

Differences between the original and recent outbreaks are perhaps equally revealing with respect to epidemiology. The original FWRF elk herd was composed of individuals from four other captive facilities, three of which had housed deer with CWD (Williams and Young, 1982, 1992); individuals from all three of the latter sources were affected. One of the affected individuals in the original outbreak was probably infected at another facility: this female entered the FWRF herd as an adult in 1978 and succumbed to CWD about 12 mo later. In the recent outbreak, all but one of the animals in the herd entered as calves and all affected elk were wild-born but raised at the FWRF.

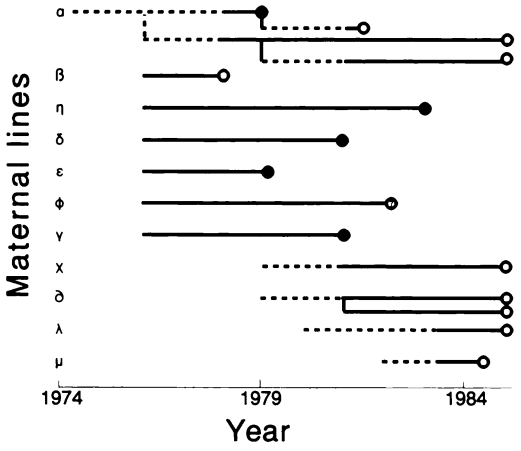

FIGURE 3. Chronic wasting disease (●) also affected 5 of 14 (36%) elk that remained >12 mo in the original elk herd maintained at the Foothills Wildlife Research Facility (FWRF) between 1976 and 1985. Years are represented in a biological context and consequently begin in June; broken lines indicate time periods and events occurring outside the FWRF, and open circles represent natural deaths or euthanasias. Data are from Williams and Young (1982) and unpublished FWRF records; the ultimate fate of one animal (?) could not be determined from available records.

Prior to 1985, mule deer were a potential source of infection for elk at the FWRF (Williams and Young, 1982). Although the original source of CWD remains undetermined (Williams and Young, 1992; Spraker et al., 1997), cases in deer initially preceded cases in elk by at least a decade at the FWRF (Williams and Young, 1980, 1982). In contrast, facility management practices since 1986 precluded contact between deer and elk. Moreover, elk were the only cervids housed at

the FWRF between June 1986 and July 1990, and CWD did not recur in FWRF deer until October 1994 (CDOW, unpubl. data).

Differences in estimated overall incidence rates for these two outbreaks (17% from 1986 to 1997 vs. 36% from 1976 to 1985) may be attributable to herd management practices. In addition to segregating elk from deer since 1986, we also promoted earlier detection and removal of clinical cases. Other aspects of animal husbandry, including nutrition and sanitation, were also improved over the last 11 yr. Although the present outbreak is probably ongoing, some combination of these actions may have influenced recent CWD incidence.

Our observations provide compelling circumstantial evidence for lateral transmission of CWD among elk. We believe the most plausible explanation for the epidemic pattern observed in this captive elk herd (Fig. 1) is animal-to-animal transmission of CWD. It appears that each affected elk infected one or two other individuals before dying, with an average period of about 21 mo (range 13 to 32 mo) between cases. A similar pattern was evident in the original FWRF elk herd, where cases clustered about 24 mo apart (Fig. 3). The first three cases in the recent outbreak occurred among the three smallest members of the 1986 cohort; our tendency to group elk by size may have increased contact among these individuals. Lateral transmission is admittedly not the only possible mechanism for explaining the epidemic patterns we observed. Some combination of maternal transmission and point- or common-source exposure could have led to one or more of these cases. Lack of animal protein in elk diets, however, made exposure to contaminated feed highly unlikely as an important underlying mechanism for CWD transmission.

The epidemiology of CWD most closely resembles that of scrapie in domestic sheep and goats (Greig, 1940; Dickinson et al., 1974; Pálsson, 1979; Hourrigan and Klingsporn, 1996), where both lateral and maternal transmission apparently occur. Although maternal transmission has been widely regarded as the most important mode for scrapie transmission (Pattison et al., 1972; Dickinson et al., 1974; Hourrigan and Klingsporn, 1996), interpretation of supporting data remains somewhat equivocal (Ridley and Baker, 1995; Hunter et al., 1996). Based on our observations, periparturient contact appeared unnecessary for CWD transmission among elk; moreover, maternal transmission was clearly not required to maintain CWD in these herds. The nine elk affected during the two FWRF outbreaks were all unrelated. No calving occurred among the 1986 cohort prior to 1990, and neither of the first two elk affected from that cohort had ever calved (Fig. 1). None of the elk affected during the original outbreak calved on site, and the sole calf from that herd was born 2 yr after the first two CWD cases occurred. Since 1986, two calves born to females that eventually developed CWD have remained clinically normal (Fig. 1).

We recognize that the possibility of maternal transmission cannot be completely discounted by our observations. Calves in the 1986 cohort were captured from a free-ranging elk population later recognized as having been infected with CWD since at least 1981 (Spraker et al., 1997). However, because the prevalence of clinical CWD in that source population is presently <1% (M. W. Miller, unpubl. data), the probability that 36% of the calves captured in 1986 were born to infected dams is exceedingly low. Moreover, the solitary nature of cervids at parturition, combined with relatively high neonatal mortality rates in the wild, tend to diminish the importance of maternal transmission in maintaining CWD in modeled deer and elk populations (M. W. Miller, unpubl. data). Consequently, we believe lateral transmission predominates CWD epidemiology and that occurrences of maternal transmission are largely special cases of

lateral transmission between infected dams and their offspring.

Although perhaps unnecessary to maintain these CWD outbreaks, maternal transmission could explain the recurrence of CWD in the FWRF elk herd via inclusion of a single dam-infected calf among the 1986 cohort. Because single cohorts of elk were almost exclusively involved in both CWD outbreaks at the FWRF, we also acknowledge the possibility that an unidentified point source of infection could have independently given rise to these outbreaks. We believe this latter explanation is relatively implausible because the incubation period would have to range from about 1.5 to 9 yr to cover all cases observed. Alternatively, environmental contamination (Greig, 1940; Pálsson, 1979; Sigurdarson, 1991) or some unrecognized reservoir (e.g., hay mites; Wishiewski et al., 1996) could have been a factor in infecting captive elk with CWD. If an environmental source was involved, then it apparently survived our extensive disinfection procedures in 1985 and would likely hamper elimination of CWD from the FWRF (and other affected facilities) in the future.

Assuming lateral transmission occurs, and using the interval between cases or case clusters as an estimate, the incubation period of CWD in elk averaged about 26 mo; data from individual cases suggest a potential range of about 18 to 36 mo. The report of a free-ranging 21-mo-old elk succumbing to CWD (Spraker et al., 1997) is consistent with this estimated incubation period. Similarly, mule deer fawns inoculated intracranially with affected deer brain tissue developed clinical CWD 18 to 24 mo postinoculation (E. S. Williams, unpubl. data). Based on our observations, the clinical course of CWD in captive elk may exceed 12 mo; in our estimation, three of the four elk that we killed as CWD suspects would have survived for several more mo before succumbing naturally. Although the duration of clinical disease was reportedly somewhat shorter (mean about 3 mo) in the original outbreak (Williams and Young, 1982) than observed here (mean about 7.5 mo), we attribute this difference to improved recognition of the early signs of CWD in elk since 1986.

Based on data and observations gathered at the FWRF since 1976, we suggest the following as plausible hypotheses of CWD epidemiology in elk: The CWD agent probably enters an elk via oral exposure to infectious secretions or excretions (e.g., saliva, feces, urine). After an incubation period of 1.5 to 3 yr, elk become clinically affected; most elk succumb <12 mo after initial clinical signs appear, but some may survive with clinical disease >12 mo. Clinically affected elk transmit CWD to other individuals; the route of agent shedding is presently unknown, but the rate of shedding likely increases as clinical signs progress. Both young and adult elk appear susceptible to CWD infection. Neither periparturient nor maternal transmission of CWD are necessary to sustain an epidemic in an elk herd. Maternal transmission is probably a special case of lateral transmission in that calves born to infectious dams may be more likely than herdmates to become infected via intensive calf-dam interactions. Effective means of eliminating CWD from captive elk populations remain elusive, but early detection and elimination of incubating or clinically affected individuals may reduce infection rates. The epidemiology of CWD in cervids appears unique in some respects from that of other transmissible spongiform encephalopathies. Transmission routes and rates, pathogenesis, antemortem diagnostic tools, and the potential role of reservoirs or environmental contamination in perpetuating CWD epidemics warrant further investigation.

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