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TESTICULAR ATROPHY IN A MULE DEER POPULATION

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ABSTRACT: Monitoring mule deer (*Odocoileus hemionus*) on a former plutonium production site along the Columbia River at the Hanford Site, Washington (USA) revealed 27 (23%) of 116 adult males had unusually shaped, velvet-covered antlers and abnormally developed testicles. We captured 32 males to examine age-class differences and the ratio of affected to unaffected deer and determine whether affected testicles were atrophic or hypoplastic. We found testicular atrophy in most deer with velvet-covered antlers, primarily in animals older than 5 yr. Deer had marked to extreme stages of testicular atrophy, indicating permanent sterility. Decreased serum levels of testosterone and compensatory increased levels of luteinizing hormone and follicle stimulating hormone were detected in all affected males; thus, the gonadotrophic hormonal pathway may have responded to abnormally low levels of testosterone in the affected animals. *Brucella* spp. antibodies in sera were not detected and 9 (90%) of 10 affected animals were seropositive for epizootic hemorrhagic disease virus (EHDV-2) and bluetongue virus (BTV-11) as compared to 12 (63%) of 19 unaffected animals; however, signs of other infectious diseases were not observed. Testicular degeneration generally exceeded that observed with nutritional disorders and poisons in domestic species. Also, severity of the atrophy and apparent lack of other affected tissues suggested that radiation may not be responsible. Testicular atrophy in mule deer has been reported elsewhere; however, neither prevalence has been as high nor occurrence as well confined to a specific geographical area, as that observed at the Hanford Site. Furthermore, no physiological or age-related influences were described. Documenting the status of such variables and examining their relationships to this phenomenon is a crucial step in understanding the reproductive capacity of a wild deer population.

Key words: Antler deformity, androgens, contaminants, mule deer, *Odocoileus hemionus*, testicular atrophy.

INTRODUCTION

Causes of atypical, velvet-covered antlers in mule deer have been extensively studied and were summarized by Brown (1983). Wislocki et al. (1947) described the hormonal control of the antler growth cycle, proposing that gonadal and antler cycles in deer are controlled primarily by the anterior pituitary and testes. Goss (1968) studied inhibition of growth and shedding of antlers by reduced levels of oestradiol and testosterone and supported the theory that annual cycles of antler replacement are coordinated by seasonal fluctuations in sex hormones (Wislocki et al., 1947).

The cause of testicular atrophy in deer presumably is unknown. Cases of atrophic testes in deer (*Odocoileus* spp.) have been reported, but very few demographic or physiologic investigations have been conducted to determine the etiology of the disease (Murphy and Clugston, 1970; DeMartini and Connolly, 1972; Robinette

et al., 1977). Clarke (1916) first documented atypical, permanently velvet-covered antlers in *Odocoileus* sp. of the Pacific slope. Taylor et al. (1964) documented a similar occurrence in white-tailed deer (*O. virginianus*) in Texas (USA), but cause was not determined. They observed bucks with atrophied testes and velvet antlers (7% of adult males killed) to be more prevalent on granite-gravel soil types as compared to non-granite soil types within their study area. The researchers concluded that some toxic agent in the soil might be responsible. Clark (1953) reported that deer with abnormal antler growth and retained velvet (referred to as “cactus bucks”) were present in Arizona (USA) and had testes “the size of an average marble and almost as hard.” Robinette et al. (1977) also reported at least 3 (0.06%) of 4,670 hunter-killed bucks in Utah had atrophied testes.

Several workers have investigated the occurrence and causes of testicular atro-

phy in domestic animals (Ladd, 1985), but none have determined the cause of gonadal atrophy in deer. Possible etiologies for testicular atrophy by direct or indirect (hormonal) pathways include heat stress, brucellosis, phytoestrogens, mycotoxins, vitamin and mineral deficiencies, high doses of heavy metals, organochlorine contamination, genetic factors, and many others (Robbins, 1983; Ladd, 1985; Blanchard et al., 1991; Diekman and Green, 1992; Colburn et al., 1996).

We studied mule deer (*Odocoileus hemionus*) from 1991 through 1994 on the U.S. Department of Energy's 1,450 km² Hanford Site a plutonium production site located in the Columbia Basin of south-central Washington (USA). The Hanford Site has been closed to the public since 1943 when the federal government acquired the land to produce plutonium for atomic weapons. The area previously was occupied by settlers and used for livestock grazing, and later for cultivation agriculture and urbanization (Chatters, 1989). Plutonium production, which ended in 1988, left a legacy of environmental radioactive and chemical waste products. Mule deer studies were conducted as part of the Department of Energy's Wildlife Resources Monitoring Project. The project tracks the status of wildlife populations on the Hanford Site to determine whether Hanford Site operations have affected them. Rocky Mountain mule deer are of interest to wildlife management and contaminant monitoring programs because they serve as an indicator of environmental conditions of Hanford Site operations and provide useful information for current environmental clean-up efforts (Eberhardt and Cadwell, 1983).

Because mule deer have been protected from hunting on the Hanford Site for approximately 50 yr, the herd has developed unique population characteristics, including a large number of old animals and large-antlered males, in contrast to other herds in the semi-arid regions of the northwestern United States. The deer

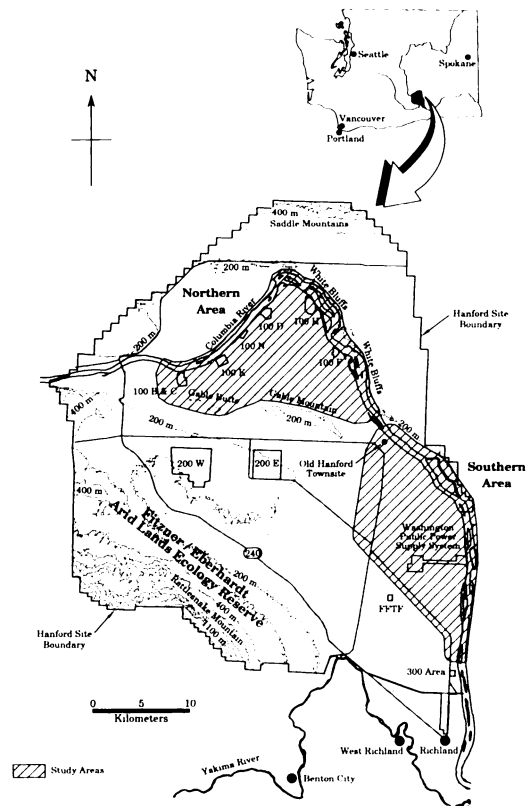


FIGURE 1. Map of the Hanford Site and its location in southeastern Washington, showing northern and southern study areas and the Fitzner/Eberhardt Arid Land Ecology Reserve.

herd at the Hanford Site provides an opportunity for comparison to other more heavily harvested herds. For this study, our initial objective was to collect baseline data describing herd composition and pertinent characteristics of males with testicular atrophy.

MATERIALS AND METHODS

We examined deer along an approximately 150 km² portion of the Hanford Site bordering the Columbia River in Benton and Grant counties, Washington (USA; 46°40'N, 119°20'W; Fig. 1). In general, the area is characterized by shrub-steppe vegetation dominated by big sagebrush (*Artemisia tridentata*) and Sandberg's bluegrass (*Poa sandbergii*) (Daubenmire, 1970; Downs et al., 1993), with approximately 16 cm of annual precipitation (Hoitink and Burk, 1994). The climate consists of hot dry summers and relatively cool winters when the bulk of annual precipitation occurs.

For comparative purposes, the study region was divided into north and south study areas. The southern area generally is unaltered by Hanford Site-related activities and is characterized by sand dunes, abandoned farm fields, and early successional shrub-steppe habitat recovering from a large wildfire in 1985. Plant communities in the dunes region are dominated by rabbitbrush (*Chrysothamnus* spp.) and bitterbrush (*Purshia tridentata*) (Downs et al., 1993). The northern study site contains six inactive nuclear production reactor sites, abandoned agricultural fields, and scattered patches of shrub-steppe habitat.

The Columbia River supports riparian habitat and riverine islands commonly used by resident mule deer. Shoreline vegetation along the Hanford Reach consists of a narrow zone of broad-leaved deciduous trees and shrubs intermingled with a variety of perennial grasses and forbs (Sackschewsky et al., 1992; Downs et al., 1993). The riparian zone tends to remain green throughout the hot dry summer months.

Fifty-four mule deer were captured in drive nets (Beasom et al., 1980) at several locations along the Hanford Site bordering the Columbia River in February and March 1991, 1992, and 1993. In 1994, we captured 26 male deer using a CODA net gun (CODA Enterprises, Mesa, Arizona, USA) fired from a hovering helicopter. The captured animals were slung in a cargo net for transport to staging areas where animals were processed. Chase and handling time for these animals ranged from 1 to 3 min and 20 to 45 min, respectively. For all deer captured, we measured incisor 1 lengths, estimated age, noted general health, fastened a solar-powered transmitter (Advanced Telemetry Systems, Isanti, Minnesota, USA) to the ear of adult males, and when available, collected antler samples for radiological analyses. We removed a canine tooth from 29 males and submitted them to Matson's Laboratory (Milltown, Montana, USA) for age determination by cementum annuli analysis (Erickson and Seliger, 1969).

During 1993 and 1994, blood was collected from the jugular vein of each deer and placed in glass tubes. Blood samples were stored on ice in a cooler until that evening when sera and blood constituents were separated by centrifugation and immediately frozen at -20°C . Results of all serum analyses and testicular weights were tested for significant differences using the non-parametric multi-response permutation procedure (Mielke, 1991).

We subsequently sedated animals with 3 to 5 ml of a 1:3 mixture of xylazine-HCL (Rompun-100 mg/ml, Miles Inc., Shawnee Mission, Kansas, USA) and ketamine-HCL (Ketaset-100 mg/ml, Fort Dodge Laboratories, Inc.,

Fort Dodge, Iowa, USA) intramuscularly and surgically removed one or both testicles from 12 affected bucks. We also removed the left testicle from six apparently normal males. Immobilized animals were given 5 ml of yohimbine-HCL intravenously (5 mg/ml-Antagonil, Wildlife Lab. Inc., Fort Collins, Colorado, USA) to reverse the actions of xylazine-HCL and subsequently were released near the capture site (Mech et al., 1985; Kreeger et al., 1986). Gross and microscopic examinations were made of the testes. Testes were measured, photographed, and weighed before and after removal of the epididymis, vascular plexes, and tunics. On occasion, radio-equipped bucks were reported to be as traffic mortalities. For these bucks, samples of brain, hypothalamus, pituitary, thyroid, lymph node, heart, lung, liver, adrenal gland, and spleen were collected and prepared for microscopic examination. Specimens were fixed in 10% formalin, paraffin embedded, sectioned at $5\text{ }\mu\text{m}$, and stained with hematoxylin and eosin. We stained sections of one testicle with periodic-acid schiff (PAS), trichrome, Prussian blue, and Congo red (Thompson, 1966). Levels of follicle stimulating hormone (FSH), luteinizing hormone (LH) and testosterone (T) were determined using radioimmunoassay as described by Berndtson et al. (1974) and Nett et al. (1975, 1979). Sera were tested by the University of Georgia (Southeast Cooperative Wildlife Disease Study, College of Veterinary Medicine, Athens, Georgia, USA) for antibodies against epizootic hemorrhagic disease virus and bluetongue virus by agar gel immunodiffusion (AGID) and serum neutralization (Stallknecht et al., 1995). Serum chemistry was conducted using an automated system (Boehringer Mannheim/Hitachi 747, Boehringer Mannheim Corporation, Diagnostics Lab System Division, Indianapolis, Indiana, USA) by Phoenix Central Laboratory (Everett, Washington, USA). Thyroxine (T_4) levels were determined using monoclonal solid phase radioimmunoassay (Becton-Dickinson, Mississauga, Ontario, Canada). Sera were tested for *Brucella* spp. antibodies using the standard buffered acidic plate antigen test (MacMillan, 1990) by the Washington State Department of Agriculture (Olympia, Washington, USA).

Because permanently velvet-covered antlers are characteristic of males with testicular atrophy, we examined frequencies of affected to unaffected males when antlers were normally calcified. We observed deer during random capture events, while tracking radio-transmitted males and when conducting systematic roadside observations. We obtained an initial estimate of the proportion of affected animals during the random capture events (February

and March 1991 and 1992). Routine ground radio-tracking of several deer residing on the Hanford Site provided an opportunity to estimate the frequency of affected males for fall and winter 1992 and 1993. For long-term monitoring, we conducted 10 roadside surveys from August through December 1994 along approximately 60 km of road beginning in the southern area and ending in the northern area. Bucks were visually examined with binoculars and/or spotting scopes and antlers were determined to be calcified or velvet covered. Roadside surveys also were conducted in 1994 and 1995 on the Fitzner/Eberhardt Arid Lands Ecology (ALE) Reserve (46°40'N 119°50'W), a portion of the Hanford Site set aside for research and education since 1967 (Fig. 1).

Age data were grouped, reflecting the ages of each animal at the time of initial capture. Because capture efforts were biased toward affected animals, the proportion of animals that exhibited permanently velvet-covered antlers was adjusted to reflect the mean proportion of affected animals observed during 1994 roadside surveys.

RESULTS

Four (22%) of 18 adult males captured in 1991 and 1992 had velvet-covered antlers and lacked normally developed testicles. Based on observations while radio-tracking male deer in fall and early winter 1992 and 1993, 13 (15%) of 88 bucks exhibited atypical, velvet-covered antlers. Systematic roadside observations in 1994 indicated $23 \pm 3\%$ ($\bar{x} \pm \text{S.E.}$, $n = 116$) of the male population was affected. Although sample sizes were low when grouped by region, no difference in the frequency of affected animals was observed between the northern and southern study regions. None of the male deer observed during fall and winter 1994 and 1995 had velvet-covered antlers on the ALE Reserve. However, we know that at least one deer exhibiting velvet-covered antlers and small testicles was harvested from property adjacent to the Reserve.

Based on the ages of 17 apparently normal males with calcified antlers and 12 males with atypical velvet-covered antlers, only the relatively older animals (mostly >5 yr) were affected (Fig. 2). Affected animals were not observed in the 1- to 2-yr

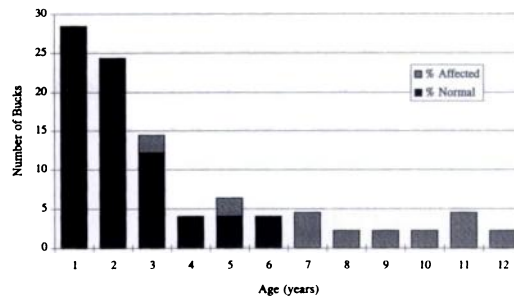


FIGURE 2. Histogram showing percentage of normal Hanford Site male deer and those with atypical, velvet-covered antlers by age group.

age group; three (22%) of 14 of the 3- to 5-yr age group were affected, and all the ≥ 7 -yr age groups were affected.

Testicular weights (without plexus, epididymis, and tunics) from 11 animals with atypical, velvet-covered antlers had a mean of 4.4 g (range 0.8 to 6.5 g) compared with 10.4 g (range 6.7 to 16.1 g) for testes collected from six apparently normal animals ($P \leq 0.001$). On histological examination, four of the six bucks with normal antlers were essentially normal for the non-breeding season (Fig. 3a). These four animals were determined to be 1-yr-old. Testes from the other two males with normal antlers had slight tubular and leydig cell atrophy with small amounts of associated interstitial lymphocytic inflammation (Fig. 3b). Within areas of atrophy, and occasionally other interstitial foci, small aggregates of monocytes and histocytes were present. These animals were 3- and 4-yr-old.

Testes collected from 11 of the 12 males with atypical velvet-covered antlers had marked (Fig. 3c) to extreme (Fig. 3d) tubular and leydig cell atrophy. Within this tubular and leydig cell atrophy there were generally only remnants of seminiferous tubules remaining, with absence of most spermatocytes, spermatogonia, and sertoli cells. Interstitial tissues in two of the extremely atrophied testicles contained degenerative changes including moderate amounts of granular olive-green pigment, generally extracellular, which was non-refractile, non-polarizing, iron negative, and

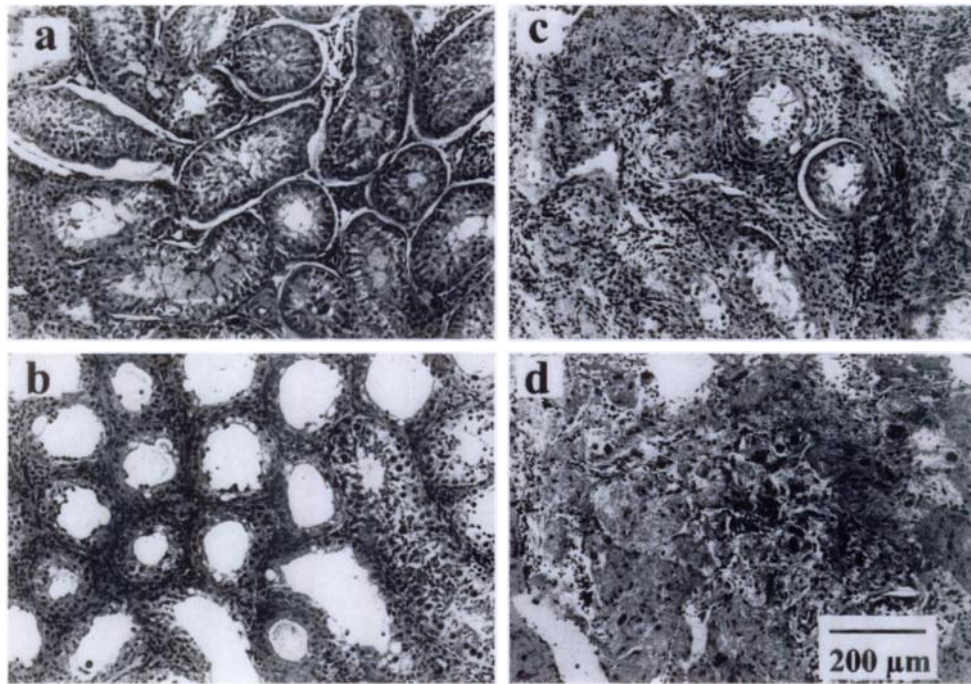


FIGURE 3. Hematoxylin and eosin-stained sections of (a) normal testis from a 1-yr-old deer; (b) testis from a 3-yr-old deer with slight atrophy; (c) testis from a 7-yr-old deer with marked atrophy; and (d) testis from an 8-yr-old deer with extreme atrophy.

stained slightly orange with the PAS reaction. Eosinophilic bands (collagenous material) were Congo red negative, lightly PAS positive, and stained blue with the trichrome stain. These degenerative changes were considered secondary to the atrophy. One of the 12 animals with velvet-covered antlers (3-yr-old) had marked infarction of the testes with extensive central coagulative necrosis. Surrounding the coagulative necrosis were a narrow zone of severely degenerative tubules, small amounts of hemorrhage and small foci of dystrophic calcification.

Although age differences were not observed between those animals with marked testicular atrophy and those with extreme atrophy, the average age of these two groups combined was approximately 8 (range = 3- to 12-yr-old). Other tissues (pituitary, brain, thyroid, heart, lung, adrenal gland, spleen, and liver) from two affected animals that were incidentally killed by vehicles during this study were micro-

scopically normal and tissue weights were typical for deer (Anderson et al., 1974).

Serological tests for *Brucella* spp. antibodies collected from nine affected animals were negative. Neutralizing antibodies in sera were detected against all of the EHD viruses (EHDV-1, -2) and blue-tongue viruses (BTV-2, -10, -11, -13, -17) and all but one of 21 deer that tested seropositive for EHDV were seropositive for BTV. Nine (90%) of 10 affected deer had antibodies against BTV-11 and EHDV-2. Six (55%) of 11 unaffected 1-yr-old males and six (86%) of seven unaffected males ages 3- to 5-yr-old also were seropositive for these two viruses. With the exception of blood urea-nitrogen (BUN), serum chemistry results from 12 affected and 20 apparently normal animals showed no differences between the groups and were essentially normal for *Odocoileus* spp. (Seal et al., 1981; Wallmo, 1981). Blood urea-nitrogen levels were significantly lower ($P \leq 0.10$) in affected animals. Alkaline phos-

TABLE 1. Results of serum hormone assays from mule deer bucks with affected and normal antlers in 1993–1994 at Hanford Site, Washington.

Antler condition	Hormone ^a		
	Testosterone	Luteinizing hormone	Follicle stimulating hormone
Normal (<i>n</i> = 20)	0.63 ± 0.18 ^b (0.09–3.36)	0.22 ± 0.03 (0.08–0.53)	23.32 ± 4.42 (9.46–99.61)
Affected (<i>n</i> = 12)	0.22 ± 0.07 (0.03–0.87)	3.87 ± 0.56 (1.05–6.84)	149.66 ± 32.78 (76.90–504.70)

^a ng/ml serum.^b Mean ± standard error (range).

phatase values were slightly elevated in normal animals. Mean testosterone (T) levels in affected animals were considerably lower than in the normal animals (Table 1). Levels of follicle stimulating hormone (FSH) and luteinizing hormone (LH) were six and 17 times greater, respectively, in affected animals than in normal animals. All androgen constituents were significantly ($P \leq 0.01$) different between affected and normal animals.

Field technicians closely observed most of the living normal radio-tagged bucks each year (September through January, 1992–1995), when antlers are typically calcified. None of the radio-tagged animals found to be normal during capture events were observed with velvet-covered antlers during the rutting season. However, one previously normal male captured in 1992 was harvested on lands adjacent to the Hanford Site in 1995 and exhibited velvet-covered two-point antlers. The hunter noted the testicles as being very small. In 1994, field technicians had observed the buck as a notably large four-point.

DISCUSSION

The epidemiologic data and microscopic lesions indicated that testicular atrophy was probably due to a disease or diseases rather than failure of the testes to develop. The 1-yr-old males essentially were normal, intermediate lesions were present in a 3-yr-old buck, and severely degenerative/atrophic testes occurred in the 5- to 12-yr-old males. If we assumed that the bucks

we examined were representative of deer in their respective age groups, progressive testicular degeneration occurred in this deer population.

The prevalence of testicular atrophy in Hanford Site adult male deer (23%) is greater than that reported anywhere else. This value, however, is difficult to compare with other mule deer populations because of differences in demography. Although some deer hunting occurs along the boundaries of the Hanford Site, the age-class distribution of our study population is unlike most deer populations where 5-yr-old males are a rarity (Zeigler, 1978; Mendin and Anderson, 1979; Story and Kitchings, 1994). Furthermore, <1% of affected animals observed on the ALE Reserve may be correlated with fewer old-aged animals found there because of the relatively high hunting pressure on adjacent lands compared with Columbia River portions of the Hanford Site. Age-class information is crucial for further understanding and relating this phenomenon to other wild mule deer populations. Although Blanchard et al. (1991) discuss senile testicular atrophy as a condition that varies by species, this condition has not been reported in captive deer herds containing 5- to 10-yr-old males. Steinhoff (1957) recorded antler status for a captive mule deer buck for all 18 yr of life and found typical calcification of the antlers up to 17 yr of life. At that point, antlers developed as “small and decadent, shaped

like a pin cushion with many small velvet-covered protuberances."

Because reproductive effects often occur in the younger, more sensitive animals, it is possible that some unknown event that took place when the affected animals were young resulted in testicular atrophy being incidentally expressed at older ages. However, we observed one radio-tagged deer that changed from normal to affected in 1995. Climatic conditions on the Hanford Site were relatively mild in 1995 and did not indicate excessive thermal stress or a shortage of forage. Sargent et al. (1994) studied thermoregulation by mule deer on the ALE Reserve and found body temperatures to be similar to those reported for deer elsewhere.

Although the causes of testicular degeneration in domestic animals are numerous (McEntee, 1990), the degree of severity in the older bucks generally exceeded that observed with nutritional disorders and, for the most part, poisons. Robinette et al. (1977) observed that the lack of velvet shedding among yearling mule deer in Utah (USA) was associated with their physical condition. Seven percent of the hunter-killed yearlings in 1949, which followed the most severe winter of their study, retained most of their velvet. However, atrophic testes were not associated with the deer exhibiting velvet-covered antlers. No differences in pelage or general body condition were observed between affected and normal animals captured in this study. Results of serum chemistry analyses were essentially normal and there was no evidence of brucellosis in the bucks, indicating this disease process was not responsible for the atrophic testes.

Difference between affected and normal animal serum alkaline phosphatase levels likely is related to antlerogenesis. Increased levels of alkaline phosphatase in serum presumably play a role in the antler shedding and growing processes (Molello et al., 1963). The lower alkaline phosphatase values are probably because affected animals do not shed their antlers. Com-

paring alkaline phosphatase values between affected and normal animals also contains significant age-related differences that have been inversely correlated with serum levels of alkaline phosphatase (Seal et al., 1978).

Mean serum hormone levels of FSH and LH found in the affected animals were many times higher than those found in normal animals. The levels demonstrate proper feedback responses of the gonadopituitary axis to extremely low levels of T found in affected animals. These results suggest testicular tissues have been directly affected by the causative agent rather than by altered hormonal pathways. This hypothesis could be tested by monitoring year-round fluctuations in androgen levels.

We do not regard exposure to known sources of radiation as the likely cause of testicular atrophy in this deer population based on the severity of the lesions and because of the absence of effects in other tissues (Wilkinson, 1969; Fajardo, 1982; Jones and Hunt, 1983), however, effects of radiation on deer has not been specifically studied experimentally. Also, because sera levels of thyroxine suggest no difference between affected and normal animals and they are essentially the same as normal values reported for *Odocoileus* spp., alteration of the thyroid caused by radio-iodine deposition is doubtful. Although exposure to other unknown radioactive substances is possible, radiation exposure measurements on the Hanford Site indicate the deer should not be exposed to levels that would produce changes in testicular functions (International Atomic Energy Agency, 1992; Woodruff and Hanf, 1992).

There appeared to be a positive correlation between age and seroprevalance of BTV or EHDV. Most animals exhibiting testicular atrophy were relatively old males and the majority of them were seropositive. Notably, one affected animal with extreme degeneration of the testes did not have antibodies for BTV or EHDV. Although signs typical of hemorrhagic dis-

ease have never been observed in the deer at Hanford Site, these viruses might be important in the etiology of testicular atrophy in *Odocoileus* spp. Short-term experimental studies of EHDV and BTV in deer have demonstrated striking changes in vascular endothelium and virus replication may initiate intravascular thrombosis (Karstad and Trainer, 1967; Tsai and Karstad, 1973). Because testicular arteries are relatively long and thin, EHDV or BTV could result in reduced or completely blocked blood flow to the testes, producing degenerative or infarcted tissues.

Although several possible causative agents have been considered, the cause of the testicular atrophy in this deer herd remains unknown. As with any epidemiologic investigation, all aspects of herd health should be considered. Of continued interest is the phytoestrogenic activities of legumes such as *Astragalus* spp. and *Melilotus* spp., which are known to produce significant quantities of estrogen-like compounds (Saloniemi et al., 1995) and are common within areas of the Hanford Site occupied by affected deer. Wildfires and arid climatic conditions may be significant factors in the potentiation of phytoestrogenic compound (Livingston, 1978). Aflatoxins naturally produced by fungal species that grow on many plants also are known to exist in this region and have been shown to act directly on testicular tissues, presumably by inhibiting early steps of the steroidogenic pathway (Fenske and Fink-Gremmels, 1990). Additional tissue samples will be obtained to further compare gross morphology of selected organs and examine levels of various environmental contaminants, including heavy metals, pesticides, herbicides and fungicides.

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LITERATURE CITED

- ANDERSON, A. E., D. E. MEDIN, AND D. C. BOWDEN. 1974. Growth and morphometry of the Carcass, selected bones, organs, and glands of mule deer. Wildlife Monographs Number 39, The Wildlife Society, Washington, D.C., 122 pp.
- BEASOM, S. L., W. EVANS, AND L. TEMPLE. 1980. The drive net for capturing western big game. The Journal of Wildlife Management 44: 478–483.
- BERNDTSON, W. E., B. W. PICKETT, AND T. M. NETT. 1974. Reproductive physiology of stallion. IV. Seasonal changes in the testosterone concentration of peripheral plasma. Journal of Reproductive Fertility 39: 115–118.
- BLANCHARD, T. L., D. D. VARNER, K. N. BRETZLAFF, AND R. G. ELMORE. 1991. The causes and pathologic changes of testicular degeneration in large animals. Veterinary Medicine 531–536.
- BROWN, R. D. (editor). 1983. Antler development in Cervidae. Caesar Kleberg Wildlife Research Institute, Texas A&M University, Kingsville, Texas, 468 pp.
- CHATTERS, J. 1989. Hanford cultural resources management plan. PNL-6942. Pacific Northwest Laboratory, Richland, Washington, 64 pp.
- CLARKE, F. C. 1916. Malformed antlers of deer. California Fish and Game 2: 119–123.
- CLARK, E. D. 1953. A study of the behavior and movements of the Tucson mountain mule deer. M.S. thesis, University of Arizona, Tucson, Arizona, 27 pp.
- COLBURN, T., D. DUMANOSKI, AND J. P. MYERS. 1996. Our stolen future. Penguin, New York, New York, 395 pp.
- DAUBENMIRE, R. 1970. Steppe vegetation of Washington. Technical Bulletin 62, Washington Agricultural Experiment Station, Washington State University, Pullman, Washington, 102 pp.
- DEMARTINI, J. C., AND G. E. CONNOLLY. 1972. Testicular atrophy in Columbia black-tailed deer in California. Journal of Wildlife Diseases 11: 101–106.
- DIEKMAN, M. A., AND M. L. GREEN. 1992. Mycotoxins and reproduction in domestic livestock. Journal of Animal Science 70: 1615–1627.
- DOWNS, J. L., W. H. RICKARD, C. A. BRANDT, L. L. CADWELL, C. E. CUSHING, D. R. GEIST, R. M. MAZAIIKA, D. A. NEITZEL, M. R. SACKSCHEWSKY, AND J. J. NUGENT. 1993. Habitat types on the Hanford Site: Wildlife and plant species of con-

- cern. PNL-8942, Pacific Northwest Laboratory, Richland, Washington, 64 pp.
- EBERHARDT, L. E., AND L. L. CADWELL. 1983. Radio-telemetry as an aid to environmental contaminant evaluation of mobile wildlife species. *Journal of Environmental Monitoring and Assessment* 5: 283–289.
- ERICKSON, J. A., AND W. G. SELIGER. 1969. Efficient sectioning of incisors for establishing ages of mule deer. *The Journal of Wildlife Management* 33: 384–388.
- FAJARDO, L. F. 1982. Pathology of radiation injury. MASSON Publishing USA, Inc., New York, New York, pp. 129–146.
- FENSKE, M., AND J. FINK-GREMMEIS. 1990. Effects of fungal metabolites on testosterone secretion in vitro. *Archives of Toxicology* 64: 72–75.
- GOSS, R. J. 1968. Inhibition of growth and shedding of antlers by sex hormones. *Nature* 220: 83–85.
- HOITINK, D. J., AND K. W. BURK. 1994. Climatological data summary 1993 with historical data. PNL-9809, Pacific Northwest Laboratory, Richland, Washington, 373 pp.
- INTERNATIONAL ATOMIC ENERGY AGENCY. 1992. Effects of ionizing radiation on plants and animals at levels implied by current radiation protection standards. Technical Report 332, International Atomic Energy Agency, Vienna, Austria, 74 pp.
- JONES, T. C., AND R. D. HUNT. 1983. Veterinary pathology. 5th edition. Lea and Febiger, Philadelphia, Pennsylvania, 1792 pp.
- KARSTAD, L., AND D. O. TRAINER. 1967. Histopathology of experimental bluetongue disease of white-tailed deer. *The Canadian Veterinary Journal* 8: 247–254.
- KREEGER, T., G. D. DEL GIUDICE, U. S. SEAL, AND P. D. KARNS. 1986. Immobilization of white-tailed deer with xylazine hydrochloride and ketamine hydrochloride and antagonism by tolazoline hydrochloride. *Journal of Wildlife Diseases* 22: 407–412.
- LADD, P. W. 1985. The male genital system. In *Pathology of domestic animals*. K. V. F. Jubb, P. C. Kennedy, and N. Palmer (eds.). 3rd edition, Volume 3. Academic Press, Inc., New York, New York, pp. 409–459.
- LIVINGSTON, A. L. 1978. Forage plant estrogens. *Journal of Toxicology and Environmental Health* 4: 301–324.
- MACMILLAN, A. 1990. Conventional serological tests. In K. Nielsen and J. R. Duncan (eds.). *Animal brucellosis*. CRC Press, Boca Raton, Florida, pp. 153–197.
- MCENTEE, K. 1990. Reproductive pathology of domestic animals. Academic Press, New York, New York, 401 pp.
- MECH, L. D., G. D. DEL GIUDICE, P. D. KARNS, AND U. S. SEAL. 1985. Yohimbine as an antagonist to xylazine hydrochloride-ketamine hydrochloride immobilization of white-tailed deer. *Journal of Wildlife Diseases* 21: 405–410.
- MENDIN, D. E., AND A. E. ANDERSON. 1979. Modeling the dynamics of a Colorado mule deer population. *Wildlife Monograph Number 68*, The Wildlife Society, Washington, D.C., 77 pp.
- MIELKE, P. W. 1991. The application of multivariate permutation methods based on distance functions in the earth sciences. *Earth-Science-Reviews* 31: 55–71.
- MOLELLO, J. A., C. P. EPLING, AND R. W. DAVIS. 1963. Histochemistry of the deer antler. *American Journal of Veterinary Research* 24: 573–579.
- MURPHY, B. D., AND R. E. CLUGSTON. 1970. Bilateral testicular degeneration in a wild mule deer (*Odocoileus hemionus*). *Journal of Wildlife Diseases* 7: 67–69.
- NETT, T. M., D. W. HOLTAN, AND V. L. ESTERGREEN. 1975. Serum levels of estrogens, luteinizing hormone and prolactin in pregnant mares. *Journal of Reproductive Fertility Supplement* 23: 457–462.
- , B. W. PICKETT, AND E. L. SQUIRES. 1979. Effects of equimate (ICI-81008) on levels of luteinizing hormone, follicle-stimulating hormone, and progesterone during the estrous cycle of the mare. *Journal of Animal Science* 48: 69–75.
- ROBBINS, C. T. 1983. *Wildlife feeding and nutrition*. Academic Press, Orlando, Florida, 55 pp.
- ROBINETTE, W. L., N. V. HANCOCK, AND D. A. JONES. 1977. The oak creek mule deer herd in Utah. *Utah State Division of Wildlife Resources*, Salt Lake City, Utah, 148 pp.
- SACKSCHEWSKY, M. R., D. S. LANDEEN, J. L. DOWNS, W. R. RICKARD, AND G. I. BAIRD. 1992. Vascular plants of the Hanford Site. WHC-EP-0554, Westinghouse Hanford Company, Richland, Washington, 343 pp.
- SALONIEMI, H., K. WAHALA, P. KURKI-NYKANEN, K. KALLELA, AND I. SASSTAMOINEN. 1995. Phytoestrogen content and estrogenic effect of legume fodder. *Proceedings of the Society for Experimental Biology and Medicine* 208: 13–17.
- SARGENT, G. A., L. E. EBERHARDT, AND J. M. PEEK. 1994. Thermoregulation by mule deer (*Odocoileus hemionus*) in arid rangelands of southcentral Washington. *Journal of Mammology* 75: 536–544.
- SEAL, U. S., L. J. VERME, AND J. J. OZOGA. 1981. Physiologic values. In *Diseases and parasites of white-tailed deer*. W. R. Davidson, F. A. Hayes, V. F. Nettles, and F. E. Kellogg (eds.). *Southeastern Cooperative Wildlife Disease Study*, The University of Georgia, Athens, Georgia, pp. 17–34.
- , M. E. NELSON, L. D. MECH, AND R. L. HOSKINSON. 1978. Metabolic differences among deer. *The Journal of Wildlife Management* 42: 746–754.
- STALLKNECHT, D. E., V. F. NETTLES, E. A. ROLLOR

- III, AND E. W. HOWERTH. 1995. Epizootic hemorrhagic disease virus and bluetongue virus serotype distribution in white-tailed deer in Georgia. *Journal of Wildlife Diseases* 31: 331–338.
- STORY, J. D., AND J. T. KITCHINGS. 1994. White-tailed deer (*Odocoileus virginianus*) on the Department of Energy's Oak Ridge Reservation: 1982 status report. ORNL/TM-6803/S5, Oak Ridge National Laboratory, Oak Ridge, Tennessee, 20 pp.
- STEINHOFF, H. 1957. THE BUCK WITH 2 POINTS. *Colorado Outdoors* 6: 28–29.
- TAYLOR, D. O. N., J. W. THOMAS, AND R. G. MARBURGER. 1964. Abnormal antler growth associated with hypogonadism in white-tailed deer in Texas. *American Journal of Veterinary Research* 25: 179–185.
- TSAI, K., AND L. KARSTAD. 1973. The pathogenesis of epizootic hemorrhagic disease in deer. *American Journal of Pathology* 70: 379–400.
- THOMPSON, S. W. 1966. Selected histochemical and histopathological methods. Charles C. Thomas, Springfield, Illinois, 1,639 pp.
- WALLMO, O. C. 1981. Mule and black-tailed deer of North America, University of Nebraska Press, Lincoln, Nebraska, 605 pp.
- WILKINSON, J. S. 1969. Endocrine disease. In *Textbook of veterinary clinical pathology*. W. Medway, J. E. Prier, and J. S. Wilkinson (eds). Williams and Wilkins, Baltimore, Maryland, pp. 181–204.
- WISLOCKI, G. B., J. C. AUB, AND C. M. WALDO. 1947. The effect of gonadectomy and the administration of testosterone propionate on the growth of antlers in male and female deer. *Endocrinology* 40: 202–224.
- WOODRUFF, R. K., AND R. W. HANF (editors). 1992. Hanford site environmental report for calendar year 1991. PNL-8148, Pacific Northwest Laboratory, Richland, Washington, 188 pp.
- ZEIGLER, D. L. 1978. The Okanogan mule deer herd. Washington Department of Game, Biology Bulletin Number 15., Olympia, Washington, 106 pp.

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