DELAYED ANTLER DEVELOPMENT AND SEXUAL MATURITY AMONG YEARLING MALE WHITE-TAILED DEER[a]

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Abstract: Yearling male white-tailed deer (*Odocoileus virginianus*) at Mammoth Cave National Park, Kentucky, had bony protuberances covered with hair in place of bone antlers. The testes were subfunctional but did contain type-A spermatogonia. The seminal vesicles were not producing seminal plasma. These abnormal deer had significantly smaller body and endocrine gland weights than normal yearling male deer. Since these abnormalities were not observed in older male deer, we considered the condition to be transitory. Moreover we believe that the abnormalities were caused by a hormone deficiency between the anterior pituitary and the testes. Malnutrition brought on by chronic overuse of forage plants was a contributing influence.

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Abnormal retention of antler velvet associated with testicular degeneration has been reported in white-tailed deer in Texas (Taylor et al. 1964) and in Columbian black-tailed deer (*Odocoileus hemionus columbianus*) in California (DeMartini and Connolly 1975). We observed a different type of antler abnormality that was associated with hypogonadism in yearling male white-tailed deer in the Mammoth Cave National Park deer herd, Mammoth Cave, Kentucky. These deer lacked bone antlers and possessed subfunctional testes. These abnormalities have not previously been reported; this paper describes these abnormalities and hypothesizes their causes.

PROCEDURE

Deer were collected by shooting in 1969-71 and by trapping in 1973-74. No deer were collected in 1972. The deer were aged by tooth eruption (Severinghaus 1949), which permits positive separation of yearlings from fawns and adults. Only male deer approximately 15 to 19 months old examined from September through January were included in this report; this is the period that yearling males normally develop bone antlers and spermatogenesis is initiated.

Antler morphogenesis was recorded for all yearling males sampled. Whole body weights were recorded for the deer that were necropsied. The thyroids, adrenals, seminal vesicles, epididymides, and testes were excised, trimmed free of their adexa, and weighed to the nearest 0.01 g. In September 1974, a male lacking normal antler development was trapped and then hemicastrated before being collared, ear tagged, and released. All tissues were fixed in AFA, Bouin's fluid, or 10 percent buffered formalin. Tissue section 5 μm thick were prepared and stained with hematoxylin and counterstained with Orange G or eosin before histological evaluation. Diameters of round seminiferous tubules were obtained (20 per animal) by averaging two right angle measurements. The ratio of seminiferous tubule to interstitial tissue was obtained by scanning the testes systematically and scoring 300 random points after the method of Chalkley (1943). The heights of the adrenal cortex were obtained from tissue sections taken from the middle of the gland. Two measurements, one dorsally and one ventrally, were taken at the midline of the sections with an eye micrometer. Sagittal sections of the pituitary were taken from the middle of the gland and were stained with hematoxylin and counterstained with eosin. We compared differences between the abnormal and normal groups for body and endocrine gland weights, seminiferous tubule diameters, adrenal cortex thickness, and the ratio of tubuler to interstitial tissue by the t-test (Steel and Torrie 1960).

RESULTS

The ratios of abnormal to normal yearling males were 3 out of 8 (1969), 4 out of 15 (1970), 1 out of 5 (1971), 3 out of 12 (1973), and 0 out of 30 (1974), totaling 11 abnormal out of 70 (15.7% males of this age class) examined.

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Antler Development

The abnormal deer in this study characteristically had small bony protuberances approximately 1 cm high arising from the forehead at the locations where antlers would normally arise in deer of this age (Fig. 1). These protuberances, covered with skin and hair, were continuous with the frontal bones of the skull and are called pedicle primordia. Their presence is normal in male fawns before the development of true (deciduous) antlers. The normal group was characterized by the presence of single-point antlers from 0.9-4.0 cm long and lacking a basal burr.

Body and Gland Weights

The abnormal deer (x = 36 kg) weighed significantly less ($P < 0.05$) than the normal deer (x = 47 kg) (Table 1). The mean testes and seminal vesicle weights

Table 1. Comparison of means of body and endocrine gland weights, percent tubular tissue, and seminiferous tubule diameters in yearling male deer, Mammoth Cave National Park, Kentucky.

Antler classification	*Body weights ± S.D. (kg)*	*Testis weights ± S.D. (g)*	*Seminal vesicle weights ± S.D. (g)*	*Adrenal weights ± S.D. (g)*	*Thyroid weights ± S.D. (g)*	*Seminiferous tubule diameters ± S.D. (μm)*	*Percent tubular tissue ± S.D.*
Pedicle primordia	35.78±6.99(10)*	10.97± 3.40(10)	2.11±0.82(10)	4.0 ±1.11(10)	2.34±0.59(10)	98.75±11.65(10)	67.4±5.66(10)
Bone antlers	47.09±8.40(25)	45.34±23.61(22)	5.59±3.56(22)	4.70±1.38(22)	3.18±1.24(21)	155.00±23.33(22)	74.6±4.08(22)

*Number in parentheses represents sample size.

(paired) for the abnormal and normal groups were 11.0 and 45.3 g, and 2.1 and 5.6 g, respectively. These differences were significant ($P < 0.05$). Mean weights of the adrenals (paired) were 4.0 g (abnormal) vs. 4.7 g (normal), and of the thyroids 2.3 g (abnormal) vs. 3.2 g (normal). These differences were also significant ($P < 0.05$).

Testicular Morphology

Definitive germ cells (type-A spermatogonia) were present in the testes of all abnormal deer. However, in 9 of these deer spermatogenesis was arrested, as no cells had differentiated beyond the type-A spermatogonia (Fig. 2). The lumen of the seminiferous tubules were closed. Asynchronized spermatogenesis was observed in 1 male collected in January (Fig. 3). In this specimen elongate spermatids and primary spermatocytes were present but no round spermatids. The lumens of the tubules were not completely open. Spermatozoa were present in the epididymis. Only the abnormal male hemicastrated in September showed signs of normal spermatogenesis. Spermatogenesis had advanced to the primary spermatocyte stage of development. The lumens of the tubules were beginning to open. This animal had the largest testis weight of any abnormal animal, 12 g. Spermatogenesis in the normal males killed in September had advanced to either the round or elongated spermatid stage. Normal deer killed from October through January were sexually mature.

Seminiferous tubule diameters were significantly less ($P < 0.05$) in the abnormal group (x = 98.8 μm than in the controls (x = 155.0 μ) (Table 1), moreover the percentage of tubular tissue in the abnormal group (x = 67.4%) was also significantly less ($P < 0.05$) than in the normal deer (x = 74.6%).

The morphology of the Leydig cells differed between the 2 groups. Nuclei of the abnormal group were small and fusiform or spindle-shaped. The nuclear membrane was crenated and the nuclear material was clumped along the nuclear membrane (Fig. 4). In contrast, nuclei of the normal deer were large and round, the nuclear membrane was not crenated and the nuclear material was not clumped along the nuclear membrane (Fig. 5).

Thyroid Morphology

No differences in thyroid morphology were observed between the 2 groups. Wide variations existed in the follicle epithelium within individuals of each group. Flattened cuboidal and low columnar cells characterized the follicle epthelium. Follicle sizes ranged from small to large. Large accumulations of coloid within large follicles indicated a hypothroidal condition (Fig. 6) among both groups of deer.

Adrenal Morphology

Histologically, no differences were observed between the two groups. All three layers of the cortex, zona glomerulosa, zona fasciculata, and zona recticularis, were distinguishable, and the mean thickness ± the standard deviation for the cortex was 2.10 mm ±

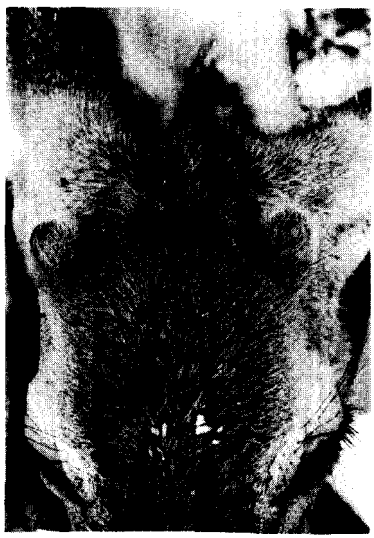

Fig. 1. Hair and skin covering the pedicle primordia characterizes antler develop among abnormal yearling male deer at Mammoth Cave National Park, Kentucky.

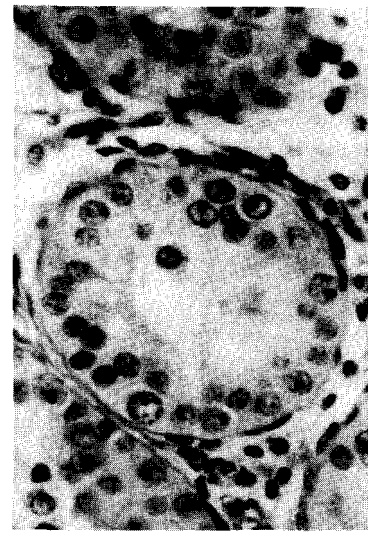

Fig. 2. Cross section of seminiferous tubule taken from an abnormal yearling male during the peak breeding season. Tubule shows only type-A spermatogonia and Sertoli cells.

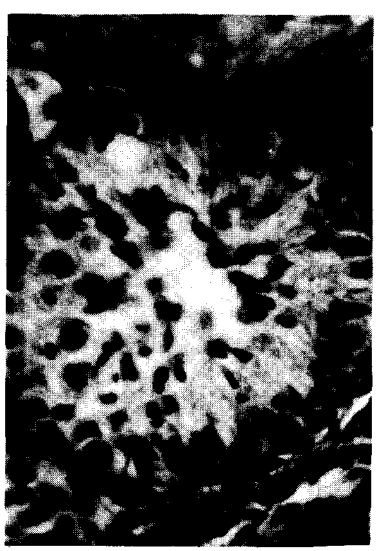

Fig. 3. Cross section of a seminiferous tubule taken from an abnormal yearling male late in the breeding season. Tubule shows asynchronized spermatogenesis.

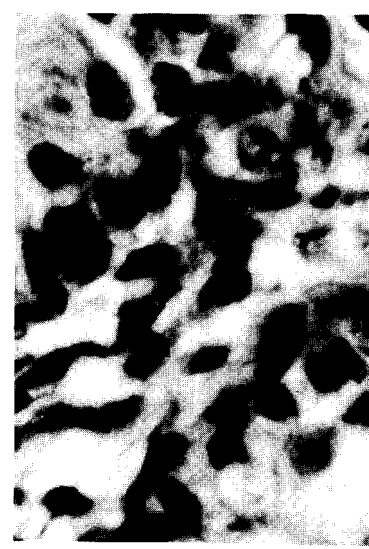

Fig. 4. Nuclei of Leydig cells from abnormal yearling male deer are characterized by small size and fusiform or spindle shape. The nuclear membrane is crenated.

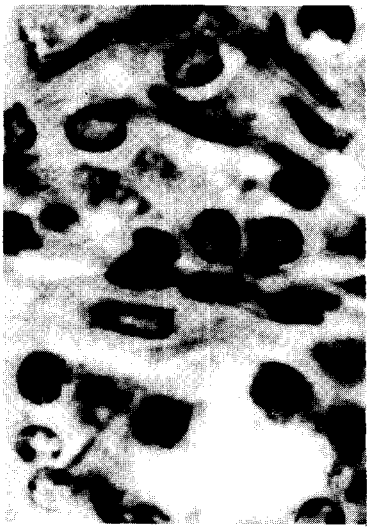

Fig. 5. Nuclei of Leydig cells from normal deer are characterized by large size and round configuration.

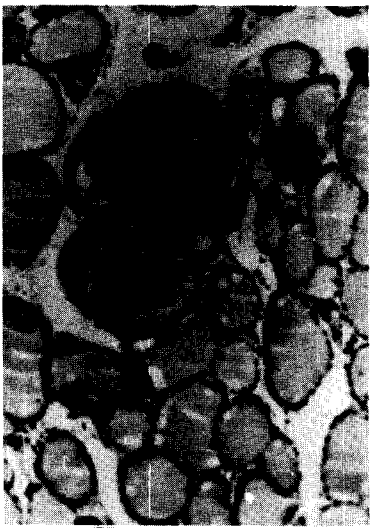

Fig. 6 The large follicles indicate a hypothyroid condition for both the normal and abnormal yearling male deer.

0.55 for the abnormal antler group and 1.98 mm $\pm$ 0.35 for the control group. The difference between groups was not significant ($P > 0.05$).

Anterior Pituitary

Acidophilic and basophilic cell populations were observed in the anterior pituitary; the acidophilic types were more numerous. Qualitative evaluation of the gland showed little difference between these 2 cell types among and between the abnormal and normal groups. No lesions or tumors in the gland were observed, and no morphological differences were observed in the other 2 sections of the gland.

Seminal Vesicle Morphology

Although seminal plasma was not being produced or stored in the abnormal deer, the cellular components of the seminal vesicles appeared to be similar between the 2 groups.

DISCUSSION

The antler and testicular abnormalities described in the present study differ from those reported in white-tailed deer from the central mineral region of Texas (Taylor et al. 1964) and in Columbian black-tailed deer from California (DeMartini and Connolly 1975). All these animals were sterile (type-A spermatogonia were absent). Antlers were present but they were velvet-covered even during the breeding season. These abnormalities were not restricted to yearlings. The etiology of these two abnormalities was not determined. In our study the abnormal animals were not sterile since type-A spermatogonia were present. Bone antlers failed to develop in the spring and the antler morphology was regarded as a manifestation of fawn antler growth carried over into the yearling age class. We believe that the lack of bone antlers and delayed sexual maturity were transitory since they were not identified in animals older than the yearling age class. To test the hypothesis that these conditions are transitory, we planned to trap, mark, and release back into the population a sample of such abnormal deer. One deer, the hemicastrate, was marked and released but was never recaptured. Before any additional animals could be marked and released, the project was terminated.

Antler failure among yearling males implies a deficiency in hormone production by the testes, anterior pituitary, or both. Normal antler development is dependent on the following sequence of events. The luteinizing hormone releasing factor (LHRF) of the hypothalamus stimulates the release into the bloodstream of luteinizing hormone (LH) by the adenohypophysis. LH in turn stimulates the Leydig cells of the testis to produce the androgen, testosterone. The testosterone then primes the antler sites. The absence of testosterone for initial priming of the antler site prevents antler growth (Wislocki et al. 1947). This explains why males castrated when immature will remain antlerless throughout their lives. That testosterone production was impaired in the antlerless group was evidenced by both the abnormal Leydig cell morphology, and the low seminal vesicle weights. Seminal vesicle weights have been shown to accurately reflect testosterone production (Turner 1966). Malnutrition in bulls interfered with testosterone production by reducing the concentration of LHRF in the hypothalamus and consequently reducing the quantity of LH released (Leathem 1970). LH injection in these animals elicited a rapid testosterone response within the testis.

By thyroidectomy of a 2-month-old male that subsequently developed "a good set of antlers" as a yearling, Wislocki et al. (1947) demonstrated that antlers can develop in the absence of thyroid influence. Studies by Hoffman and Robinson (1966) and Silver et al. (1969) suggest that a hypothyroid state in the winter is normal for white-tailed deer.

Small body weights, hyposecretion of the gonadotropic hormones [follicle stimulating hormone (FSH) and luteinizing hormone (LH)], or combinations of both may have resulted in the aspermatogenic testes. Impaired body and testicular growth and delayed puberty occurred when young bulls received only 60 percent of a recommended total digestible nutrient diet (Van Demark et al. 1964). These bulls were underfed from 8 weeks of age and when sampled at 12 and 16 months of age, were 4-8 months or more behind control bulls in testicular growth, semen volume, and total sperm production. Mann (1974) stated that underfeeding affects young males undergoing sexual maturation by suppressing the endocrine activity of the testes and, consequently, there is a growth arrest and diminished secretory activity of male accessory sex glands. Courot et al. (1970) reported that spermatogenesis is more closely related to body weight than to age. All the males in the Mammoth Cave study had progressed from the definitive prepubertal gonocytes, or primitive germ cells, to type-A spermatogonia and all but two were arrested at this stage, even though spermatogenesis in rats may progress up to the pachytene stage without being under the influence of either FSH or LH (Vilar 1968). However, the separate actions of FSH and LH in the initiation and maintenance of spermatogenesis is unknown (Vilar 1973). Our data suggest that LH was suppressed (small weights of the seminal vesicles), but we have no evidence suggesting the suppression of FSH.

Froelich's syndrome (Smith and Jones 1957), which is caused by a hypopituitary and produces demasculinizing and feminizing of the male, can be ruled out because the characteristics of this disease, such as accumulation of subcutaneous fat, scanty and fine hair, and thin skin, were not observed in the Mammoth Cave deer. Because there was no difference in the height of the cortex between the two groups we concluded that the gland was not hypofunctional for the adrenocorticotrophin hormone. A hypofunctional gland would have reduced the thickness of the cortex, particularly the zona fasticulata (Turner 1966).

We hypothesize that malnutrition was responsible for the absence of bone antlers and delayed puberty. In a deer nutrition study, male fawns fed either a low energy or low protein ration from 6 to 18 months of age exhibited symptoms similar to those observed in our study (McEwen et al. 1957). Unfortunately, the gonads were not examined and there were no available data on the fertility of these animals. As yearlings, the underfed deer were extremely stunted and did not develop antlers although all had visible antlers at 2½ years regardless of diet. Since we did not observe any antlerless males beyond the yearling age class at Mammoth Cave, we assumed that the abnormal yearling males developed antlers during their third year of life.

Malnutrition and the subsequent abnormalities may have been caused by the Mammoth Cave deer herd exceeding the carrying capacity of the area. Since the establishment of the park in the early 1940's, the herd has grown without any major restraints. Because it is a U.S. National Park, deer hunting has been prohibited. The only removal has been for restocking other areas in Kentucky; some poaching has also occurred. Unfortunately, no population figures are available. Mammoth Cave, at its time of purchase, was largely farmland. Since that time, plant succession has progressed from old field to forests, gradually reducing the amount of favorable deer habitat. The available forage area is considered overbrowsed, exceeding the prescribed "safe" browsing limits on both preferred and other forage species (Troublefield 1976).

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