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Can Androgen Deficiency Promote an Outbreak of Psoroptic Mange Mites in Male Deer?

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ABSTRACT: Repeated outbreaks of infection by psoroptic mange mites (*Psoroptes equi* var. *ovis*) affecting most regions of the body and legs were observed in several male white-tailed deer (*Odocoileus virginianus*) suffering from hypoandrogenism (e.g., castrates, animals treated with antiandrogens or postprime). The massive infection was characterized by a severe alopecia and skin inflammation and began usually in early winter. One or two spray treatments of a 1% solution of Lindane was usually sufficient to eliminate the clinical signs of the disease and to restore a healthy hair coat. Neither healthy male or female deer have ever exhibited any external signs of this disease nor has the parasite been detected in the scrapings of their skin. We propose a possible relationship between the hormonal status of these animals and their resistance to this parasitic mite infection.

Key words: *Psoroptes equi* var. *ovis*, mange mite, white-tailed deer, *Odocoileus virginianus*, male, androgens, immunology, resistance.

Infections with psoroptic mange mites has been described in domestic livestock as well as in wild animals (Sweatmen, 1971). *Psoroptes cervinus* and *Psoroptes ovis* have been observed on bighorn sheep (*Ovis canadensis*) (Sweatman, 1971; Lange et al., 1980) and *Psoroptes cervinus* has been reported in wapiti (*Cervus elaphus canadensis*) (Sweatman, 1971; Thorne et al., 1982). Infection with *Psoroptes cuniculi* has been observed in wild and captive populations of white-tailed deer (*Odocoileus virginianus*) and mule deer (*Odocoileus hemionus*) (Roberts et al., 1970; Rollor et al., 1978; Strickland et al., 1981; Schmitt et al., 1982).

The clinical signs of disease caused by mites are depilation, exudation and pruritus. The feeding activity of the parasite causes the superficial layers of epidermis to break up which induces irritation and inflammation. This is accompanied by exudation of lymph and serous fluid that form layers of crusts and scabs. The acutely infected skin is red and usually loses hair over the affected areas (Sweatman, 1971). A secondary bacterial infection often causes a purulent exudate from the skin (Thorne et al., 1982).

In the wapiti, infection was observed on the neck, body and the upper legs (Sweatman, 1971); in white-tailed deer and mule deer *Psoroptes* spp. was reported only around the pedicle and in the deep regions of the auditory canal, but not in other dermal parts (Roberts et al., 1970; Kellogg et al., 1971; Strickland et al., 1981; Schmitt et al., 1982).

Because the disease errupts almost exclusively in winter (Sweatman, 1971), massive loss of hair can cause death of the host by exposure to cold (Honess and Winter, 1956; G. A. Bubenik, pers. obs.). On the other hand, ear infection, which produces a greenish-brown waxy material plugging an ear canal, can cause a pyogenic otitis (Rollor et al., 1978). Transmission of psoroptic mites by a direct body contact or from common rubbing posts has been reported by Sweatman (1971). However, an indirect transmission of mites deposited on vehicles and people is also being considered possible (Thorne et al., 1982).

During the last 10 yr, numerous infections of *Psoroptes equi* var. *ovis* have been observed in captive adult white-tailed deer bucks born and kept at the University of Guelph Deer Research Station in Cruikston Park near Cambridge (Ontario, Canada). The species of mites was identified by K. W. Wu from Agriculture Canada (Biosystematic Research Center, Ottawa, Ontario, Canada K1A 0C6). All male deer were housed individually in 5 × 30 m pens which were separated by a wall made of particle board; these were attached 20 cm from the ground to cedar posts, so nasal

contact between animals was possible. Females and fawns were kept in one large breeding enclosure (1 ha) adjacent to individual pens of males.

In contrast to previous reports describing the psoroptic mites on white-tailed deer (Roberts et al., 1970; Kellogg et al., 1971; Strickland et al., 1981), diseased skin was observed not only around the pedicle and in the auditory canal, but also on other parts of the body and legs. Signs of infection usually started in the skin right below the antler coronet (burr). This area has a higher humidity and warmer temperature than skin in other parts of the head. From there the mites spread anteriorly into frontal and nasal areas, laterally into the cheek regions and the auditory canal and posteriorly into the skin of the neck and the shoulders. Secondary infection (possibly the result of licking) developed later in the groin region from where it spread into abdominal areas.

This unusually massive infection of body areas, not described previously in white-tailed deer, was observed only in bucks with impaired production and action of androgens. The first cases were observed in two adult bucks treated with an antiandrogen cyproterone acetate (CA) (Schering AG., D-1000, Berlin 65, Federal Republic of Germany) which blocks the central as well as the peripheral action of androgens (Bubenik et al., 1975). Several years later, two other bucks treated with CA (Bubenik et al., 1987) developed a natural infection with psoroptic mites. After the termination of the treatment with CA these animals apparently became resistant again to psoroptic mites. In addition to the CA-treated animals, two castrated white-tailed deer used in several endocrine studies (Bubenik, 1983; Morris and Bubenik, 1983) were repeatedly infected in each of five consecutive winters.

Finally, two postprime bucks (aged at 10-, and at 11- to 12-yr-old, respectively), whose testosterone levels in the rutting season were well below the average concentration (Bubenik and Schams, 1986) had to be treated for infection with psoroptic mites. Alternatively, signs of this skin disease were not observed in two castrated bucks which were treated with testosterone in the fall in order to polish and cast overgrown antlers (Bubenik, 1983). Otherwise, healthy skin in castrates occurred only during the first winter after orchiectomy which was performed late in September.

In contrast to these cases, clinical signs of the disease caused by this mite infection were not observed in any female white-tailed deer; infection was not detected in bucks treated in September with CA for only 2 to 3 wk.

In non-castrated bucks a very mild infection (usually just in the deeper parts of the auditory canal) was observed only in deer that were under severe stress because of bacterial or viral disease (Bubenik and Brownlee, 1987) or a repeated long-term experimental blood sampling (Bubenik, 1986). Healthy, vigorous bucks were never affected, despite being exposed to nasal contact with deer that were heavily infested with mites.

The exact mode of initial transfer of the parasite to our deer was never determined. Domestic livestock (cows and sheep) kept in the nearby farm might have been a source of the mites, but other origins of infection also must be considered because the experimental transfer between wild and domestic ruminants has not been successful (Hepworth and Thomas, 1962).

The initial signs of the skin disease were usually first observed in late December. Cold and humid weather with temperatures near 5 C appeared to facilitate the spread of the disease to new sides on the body. Alternatively, dry, frosty conditions seemed to reduce the spread of mites (G. A. Bubenik, pers. obs.).

As soon as the infection became obvious in the deer (e.g., alopecia, erythemia and exudate) a vigorous treatment was initiated. The obvious sign of hairless patches on the skin indicates that the infection is already established in much larger sur-

rounding areas. After the first detection of clinical signs of infection (usually using binoculars) the infected buck was tranquilized with xylazine hydrochloride (Rompun, Haver-Lockhart, Bayvet Division, Mississauga, Ontario, Canada L4K 1G4) (Bubenik, 1982) and affected areas of skin were treated with 1% solution of Lindane (Pfizer, Dorval, Quebec, Canada H9R 4V2) applied first from a spray bottle and then rubbed in with rubber gloves. Usually twice as large an area was treated as appeared macroscopically to be infected.

In most cases two treatments 2 or 3 wk apart were sufficient to restore healthy skin for the rest of the year. Because of the topical use, concentration of Lindane was much higher than recommended for the whole body dip. The skin of treated deer healed relatively quickly and a new hair growth was observed about 2 weeks after the treatment. However in some severe cases, where the detection was made late, up to four treatments were necessary to eliminate the signs of the disease as determined by a careful examination of the affected areas using a magnifying glass. However, as no microscopic investigation of the skin has been performed, mites might have survived in inaccessible areas (such as the deeper parts of the auditory canal).

That sex hormones can influence the function of the immune system has been well established. Dehydroepiandrosterone was found to have anti-autoimmune properties (Schwartz et al., 1984) and estradiol (E_2) was reported to depress the function of the thymic lymphocytes (Grossmann et al., 1983). Conversely, dihydrotestosterone (DHT) possibly enhances function of thymic lymphocytes (Grossman and Roselle, 1983). In addition, specific E_2 receptors have been detected in the reticuloendothelial cell of the thymus (Grossman and Roselle, 1983).

In addition to the effect of androgens mediated by the thymus, male sexual hormones also are known as anabolic hormones which improve the utilization of nutritional resources (Turner and Bagnara, 1974). Decrease of androgen levels may then affect the immunoresistance by a reduction of certain vital metabolic pathways.

In view of these findings, it can be speculated that the severe psoroptic infection in some captive male white-tailed deer may be a result of their hypoandrogenesis that caused either a decrease in immunoresponsiveness of the skin or in alteration of the skin ecology favoring the proliferation of the mites.

The relationship between the levels of sexual hormones and the mite infection in wild animals deserves further study. Presently, there is no explanation why female deer never exhibited any external signs of the disease (such as the hairless patches or red skin with exudate) or how the thymic stimulation could influence the resistance of the deer against these skin parasites. Further studies exploring whether "an androgen supplement" could prevent spread of this infection in a stressed population or if androgen treatment may cure already heavily infected bucks are warranted.

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