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THE ROLE OF VIRAL LATENCY IN THE EPIZOOTIOLOGY OF CONTAGIOUS ECTHYMA IN ALASKA

By

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The results of this project will be submitted for publication in the Journal of Wildlife Diseases.* The attached manuscript constitutes the final report for this job.

ABSTRACT

In an effort to determine if latency plays a role in the epizootiology of contagious ecthyma (CE), the corticosteroid dexamethasone was injected intramuscularly into 2 adult Dall sheep (Ovis dalli) to simulate adrenal stress. Both of these animals had experienced clinical CE 5 years earlier. No CE lesions developed during the 23-day experimental period, nor was there any significant rise in CE antibody titer.

Key words: Alaska, contagious ecthyma, epizootiology, viral latency.

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INTRODUCTION

Contagious ecthyma (CE) is a viral disease that has erupted in both wild and captive Dall sheep in Alaska over the past several years. In the 2 epizootics which were intensively studied, there was no direct evidence regarding the source of the virus which had precipitated the outbreak (Dieterich et al. 1981, Zarnke et al. 1983). The most recent example occurred in a band of Dall sheep in Denali National Park during September-December 1981 (Zarnke et al. 1983). Three sheep either succumbed to the disease or were sacrificed due to the severity of the infection. Lack of understanding of the cause of these outbreaks precludes any meaningful attempt at human intervention.

Outbreaks of CE in wild sheep have occurred in other parts of North America under similar circumstances where there is no obvious source of the virus (Hebert et al. 1977, Lance et al. 1981).

Researchers who studied these other epizootics have hypothesized that latent CE infections which have subsequently been reactivated may have been the source of the virus (Hebert et al. 1977, Lance 1980). Natural stimulation of the adrenal cortex has been proposed as the mechanism by which this reactivation has been manifested (Lance 1980). Experimental corticosteroid treatment may be capable of reactivating latent CE infection in domestic sheep (Buddle 1981). This technique has proven successful in similar reactivation studies of latent herpes virus infections in members of the Bovidae (Sheffy and Davies 1972, Karstad et al. 1974). The purpose of the current study was to determine if CE could be reactivated in Dall sheep by subjecting them to simulated adrenocortical stress.

MATERIALS AND METHODS

Two adult female Dall sheep (Ovis dalli) were moved into isolation facilities at the University of Alaska-Fairbanks on November 30, 1982. After allowing a week for acclimation to the new surroundings, treatment was begun. For 5 consecutive days,

the sheep received 9 mg of dexamethasone, 1.5 gm dihydrostreptomycin, and 1,200,000 units penicillin G procaine by intramuscular injection. The purpose of the dexamethasone was to simulate stress, and the antibiotics were administered to combat bacterial infections. The animals were closely examined twice per week for the subsequent 3 weeks in search of potential CE lesions. An 8 ml blood sample was collected at the time of each treatment and examination. Sera were tested by means of the complement fixation test (Erickson et al. 1975) for evidence of an increase in CE titer by personnel at the National Veterinary Services Laboratory (United States Department of Agriculture, Ames, Iowa 50010, USA). Differential blood cell counts were also performed on each sample. At the end of the examination period, the animals were necropsied, and sections of the following tissues were collected for both virus isolation attempts and for histological examination: heart, liver, lung, spleen, lymph node, kidney, and tongue. Specimens for virus isolation were stored in 50% glycerin and were tested by personnel of the National Veterinary Services Laboratory. Tissue suspensions were inoculated into embryonic ovine kidney and VERO-M cell cultures for virus isolation. After 2 serial passages, the cultures were examined by means of fluorescent antibody microscopy. Specimens for histology were preserved in 10% formalin, sectioned, mounted on glass slides, and stained with hematoxylin and eosin.

RESULTS

The effect of dexamethasone injection on the hematopoietic system was typical of that seen in other ruminants. Twenty-four hours after the initial injection, both animals exhibited leukocytosis, neutrophilia, lymphocytopenia, eosinopenia, monocytopenia, and basopenia. By the end of the experimental period (23 days after the initial treatment), blood values had essentially returned to pre-treatment levels (Schalm 1975).

The CE antibody of animal B rose from an undetectable level to a titer of 5 during the one-week acclimation period preceding initiation of the corticosteroid treatment regime. The titer rose to 10 on the second and third day of treatment, fell to 5 on the fourth, fifth, seventh, ninth, and eleventh days of the experiment, and then fell to undetectable levels for the remainder of the study.

No oral, hoof, or other lesions were observed on either animal during the experimental period. At necropsy, one small (1 mm diameter and raised 0.5 mm), pink, granular lesion was found directly above the right hoof of animal A. Histologic examination of sections of this lesion revealed deep rete pegs, inflammatory cell infiltration, and minimal ballooning degeneration in the periphery. Virus isolation attempts revealed no evidence that this lesion was related to CE infection, nor was

virus detected in any other tissues. There were no specific abnormalities of internal organs other than a mild discoloration of the adrenal cortex caused by administration of the dexamethasone.

DISCUSSION

The 2 sheep involved in this study were members of a captive band of both ewes and rams which had experienced an outbreak of CE during 1977 (Dieterich et al. 1981). Both ewes had exhibited mild clinical signs of CE, with small crusty lesions on the lips. In July 1977, animal A had a neutralizing titer of 8; animal B was 2. Both had titers of 2 in May 1978. Antibody was undetectable in August 1979 and again immediately preceding the experimental period. The minor apparent fluctuations in titer for animal B during the current experiment are believed to be within the realm of sensitivity of the complement fixation test and are therefore considered to be unrelated to the actual reactivation of a latent CE infection.

The basic approach utilized in the present study has proven effective in reactivating latent infections of herpes family viruses (Sheffy and Davies 1972, Karstad et al. 1974). However, the failure to exacerbate clinical symptoms of CE by means of dexamethasone injection should not be interpreted as precluding the possibility that viral latency plays a role in the epizootiology of CE. Herpes viruses are rather easily reactivated. Perhaps a more severe or more natural stimulus is required to reactivate a latent CE infection. Annual physiologic cycles may also play a role; natural outbreaks in Alaska have occurred during summer and autumn months.

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