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Author: WINGATE, DAVID B.

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POXVIRUS INFECTION OF THE WHITE-TAILED TROPICBIRD *(Phaethon lepturus)* IN BERMUDA

DAVID B. WINGATE, Department of Agriculture and Fisheries, Hamilton, Bermuda.
I.K. BARKER,[1] Department of Pathology, Ontario Veterinary College, Guelph, Ontario, Canada N1G 2W1.
N.W. KING, New England Regional Primate Research Center, Harvard Medical School, Southborough, Massachusetts 01772, USA.

Abstract: Lesions caused by an avian poxvirus were identified on the face and nares of fledgling white-tailed tropicbirds *(Phaethon lepturus catesbyi)* in the natural environment on Bermuda. Between 1958 and 1978, 6 of 81 fledglings found off the nest and unable to fly at departure time had lesions suggestive of poxvirus infection. More detailed nest-site surveys from 1974 to 1978 indicated an overall prevalence of less than 0.5%, involving the fledgling population only.

INTRODUCTION

Although avian poxvirus infection is well known from a wide variety of wild birds,[6,7] it has been recorded only rarely as a natural infection of shorebirds, waterfowl and seabirds, including the dunlin,[2] Canada Goose[1] and murre.[4,5] A single record of poxvirus infection in red-tailed tropicbirds *(Phaethon rubricauda)* on Midway Island is available.[8]

This paper describes cutaneous poxvirus infection in fledgling white-tailed tropic birds and reports observations on its prevalence and significance in a colony of over 3000 breeding pairs in Bermuda, where the breeding biology of the species has been described by Gross[3] and Wingate.[9,10]

MATERIALS AND METHODS

Records of observations by the senior author between 1958 and 1978, of fledgling *P. lepturus* found off the nest and unable to fly at the time of departure, together with the results of a more intensive breeding success survey carried out between 1974 and 1978 on approximately 180 active nest sites on the predator-free Castle Harbour Island Nature Reserve, were reviewed. Formalin-fixed specimens of tissues removed from the facies and nares of birds afflicted by cutaneous tumors in 1978 were processed routinely for light and electron microscopy.

RESULTS

Between 1958 and 1978, 81 fledgling tropicbirds from nests throughout Bermuda were found off the nest and unable to fly at the time of departure, when parental feeding at the nest has ceased. Six of these fledglings had lesions attributable to poxvirus infection. Yearly prevalence was 2/8 (1958); 1/5 (1959); 1/2 (1973); 2/17 (1978) and 0/49 in the other 16 years combined. The other non-viable fledglings were victims of nest predation; washed out of nests by storms; deformed; or emaciated, probably due to mortality of one or both of the parent birds.

Observations from the nest survey of the Castle Harbour Islands between 1974 and 1978 revealed poxvirus infection in only two out of five years viz. 0/121 chicks in 1974; 0/103 in 1975; 0/121 in 1976; 1/125 in 1977 and 1/120 in 1978. This represents an infection rate of 0.8%

[1] To whom requests for reprints should be sent.

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in 1977 and 1978 and about 0.3% over the entire survey period.

Chicks developing lesions on the nest were first observed to have tumours 1 to 2 cm in diameter on the skin of the forehead or eye by the time they were half fledged, about 3 to 6 weeks of age. These rapidly increased in size to approximately 1/3 the size of the head. Lesions on fledged chicks were observed on the head, nares, base of the bill, eye, carpus and back. Frequently the lesions were ulcerated, bleeding and sufficiently large and strategically located to have obscured vision or impaired respiration (Figs. 1, 2). Affected birds found off the nest are usually waterlogged and moribund, or emaciated and wandering on land, although while they are on the nest and fed by the parent they remain otherwise healthy. One chick, collected while still being fed at the full-fledged stage, had a body weight of 470 g, normal for a healthy chick at this stage.

Tumorous lesions submitted for examination had an irregular ulcerated surface covered with scabs, and on cut surface were fibrous and pale grey with superficial hemorrhage. Histologic examination revealed masses of hyperplastic epithelium thrown into papilliform structures. Hypertrophic cells contained large eosinophilic intracytoplasmic inclusion bodies. Superficial epithelium, and some deeper cells were necrotic, and the surface of the lesion was covered by a scab composed of blood and necrotic debris. The supporting connective tissue stroma was highly vascular and relatively loose, with moderately heavy heterophil and mononuclear cell infiltrations in some areas.

Ultrastructural observation revealed immature viruses within spherical vacuoles about 380 nm in diameter, and mature poxvirus particles about 350 × 175 nm with a typical dumbell-shaped

FIGURE 1. Fledgling tropicbird with large ocular poxvirus lesion. Note the blood-staining of feathers on the breast.

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FIGURE 2. Ulcerated poxvirus lesion at the nares of a fledgling tropicbird, found wandering off the nest at the time of departure.

nucleoid and complex envelope, in the cytoplasm of epithelial cells. In some cases, mature poxviruses appeared to be budding into the lumen of large membrane-bound vacuoles in the cytoplasm.

DISCUSSION

Poxvirus infection, although generally of low prevalence, may be a significant component of fledgling mortality in this species, accounting for about 7% of the non-viable birds intercepted after departure from the nest. Most lesions observed were sufficient to handicap or prevent fledglings from flying and those on the head certainly prevented them from feeding since binocular vision is required for capturing their prey of fish and squid, which is accomplished by diving from a height of approximately 30 m. Pox infection appeared to be of high prevalence among red-tailed tropicbirds on Midway Island (19/115 chicks and 2/15 adults banded; 4/9 dead chicks, 0/1 dead adults) and the lesions described resembled those illustrated here.[3] Diphtheritic pox, affecting tracheal and bronchial epithelium, was diagnosed by us in a Bermuda white-tailed tropicbird in 1979, after this paper was originally prepared.

The mode of spread of this virus is uncertain. Only nestlings and fledglings have been observed to be infected in Bermuda. Established breeding pairs invariably return to the same nest site each year but the nest sites are not visited for approximately 7 months between one breeding season and the next. During this non-breeding period, both adults and fledglings are widely dispersed over the Sargasso Sea. There is no physical contact among the birds except at the nest site during the nesting season. As the condition seems invariably to be fatal to infected fledglings, the adult population is apparently either not susceptible or immune. Infection may be carried over from one cohort to the next in the nest environment, gaining access from the nest substrate at sites of trauma, or mucocutaneous junction. Mallophaga are common on all adult tropicbirds examined and might transmit the virus. *Culex fatigans*, *Aedes sollicitans* or other blood sucking arthropods present in the environment also could be involved in transmission. The low prevalence of infection may imply that fomite transmission, rather than arthropod-borne transmission, is most probable.

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