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Authors: McOrist, Steven, Black, Douglas G., Pass, David A., Scott, Peter C., and Marshall, John

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BEAK AND FEATHER DYSTROPHY IN WILD SULPHUR-CRESTED COCKATOOS (*CACATUA GALERITA*)

Steven McOrist,[1] Douglas G. Black,[2] David A. Pass,[3] Peter C. Scott,[4] and John Marshall[5]

ABSTRACT: Three flocks of wild sulphur-crested cockatoos in southeastern Australia had a 10–20% prevalence of feather and beak deformities. In affected birds, the crest, tail, contour and down feathers were lost or reduced in length owing to a "pinching off" 0.5–1 cm above the skin. The underlying skin was discolored brown. Flight feathers appeared normal and could sustain flight. In mildly affected birds, only the crest and down feathers were involved. Histological examination of affected feathers showed necrosis of developing cells in their epidermal collars. The epidermis was moderately hyperplastic and contained numerous large macrophages, with large intracytoplasmic granules. Electron microscopic examination showed these granules to consist of regular lattices of 17–20-nm-diameter particles, resembling parvovirus particles. In some birds the upper beak was enlarged and had longitudinal cracks. Histological examination of the beak showed necrosis of its epidermis. It was concluded that a viral infection may have caused feather and beak dystrophy in these birds.

INTRODUCTION

The occurrence of a feather and beak disease syndrome has been reported in captive sulphur-crested cockatoos, galahs (*Cacatua roseicapilla*) and Major Mitchell cockatoos (*Cacatua leadbeaterii*) (Perry, 1981; Pass and Perry, 1984). The clinical features of this syndrome are loss, deformities and shortening of crest, tail, contour and down feathers combined with enlargement and longitudinal splitting of the beak. The disease has been seen most commonly in young adult birds (Perry, 1981; Pass and Perry, 1984). Light and electron microscopy indicated that these changes were due to necrosis of the feather and beak epidermal structures. Particles resembling parvoviruses were consistently found within affected epidermal tissue (Pass and Perry, 1984).

The sulphur-crested cockatoo forms numerous flocks of 100 to 500 birds in various interspersing habitats throughout eastern Australia (Kikkawa, 1968). This is the first report of significant integumentary disease in wild cockatoos.

MATERIALS AND METHODS

From June 1979 to June 1983, three flocks of wild sulphur-crested cockatoos were observed approximately every 6 mo. The flocks were generally confined to areas of 500–1,000 ha of farmland and natural vegetation in Victoria, Australia. Some affected birds were trapped in aerial nets by licensed operators. Two or three representative cases from each flock were held for examination. Eight affected birds were killed by halothane inhalation and the birds' skin, beak and internal organs examined at necropsy. The birds' age group was estimated from gonadal morphology. Tissue samples were fixed in 10% formol saline, paraffin embedded, sectioned at 4 μm and stained with hematoxylin and eosin. Care was taken to include longitudinal and transverse sections of the base of several feathers. Sectioning of beak samples required prolonged immersion in decalcifying agents before embedding. Feathers and skin tissue from three birds were post-fixed in osmium tetroxide, resin embedded and ultra thin sections were stained with uranyl acetate for electron microscopy. Feather, skin and beak tissues from normal sul-

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[1] Department of Agriculture, Regional Veterinary Laboratory, P.O. Box 483, Bairnsdale, Victoria 3875, Australia.
[2] Springvale Veterinary Clinic, 570 Springvale Road, Springvale South, Victoria 3172, Australia.
[3] School of Veterinary Studies, Murdoch University, Murdoch, Western Australia 6150, Australia.
[4] Department of Agriculture, Veterinary Research Institute, Park Drive, Parkville, Victoria 3052, Australia.
[5] Fairfield Hospital, Fairfield, Victoria 3078, Australia.

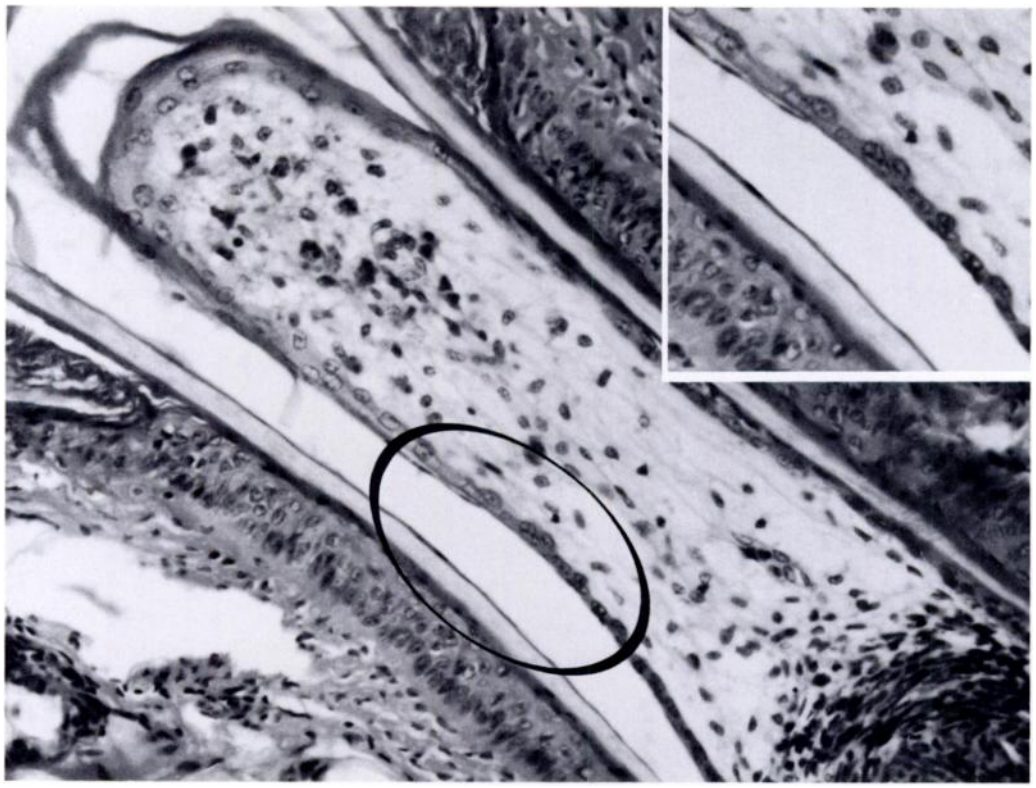

FIGURE 1. Normal sulphur-crested cockatoo feather. ×300, H&E. Inset (circled): Normal epidermal collar. ×500, H&E.

phur-crested cockatoos were available for comparison.

RESULTS

The three flocks examined consisted entirely of sulphur-crested cockatoos with the flock size averaging 500 birds (range 200–1,000). In all flocks, the proportion of affected birds declined during the 4-yr study period from 20% to 10%. Affected birds could fly normally, but were distinguishable by the loss of crest feathers and the brown discoloration of their normally white skin. Captured diseased birds were young adults and showed no abnormal behavior or activity, although they were observed to be low in the feeding order. Severely affected birds showed loss of their crest, tail, contour and down feathers. Remaining feathers were reduced in length due to a "pinching off" of these feathers 0.5–1 cm above the skin. Thus the birds' body covering consisted of short stumps of feathers with much bare skin evident. The body skin had diffuse dirty brown discoloration. The flight feathers were normal. In mildly affected birds, the changes described were confined to the head and the down feathers. All affected birds had mild to moderate upper beak enlargement. There were several longitudinal cracks evident in each beak.

Examination of longitudinal sections of the base of affected feathers showed necrosis of many individual epithelial cells within epidermal collars. Developing cells within this epidermis had enlarged nuclei, loss of cell outlines and improper align-

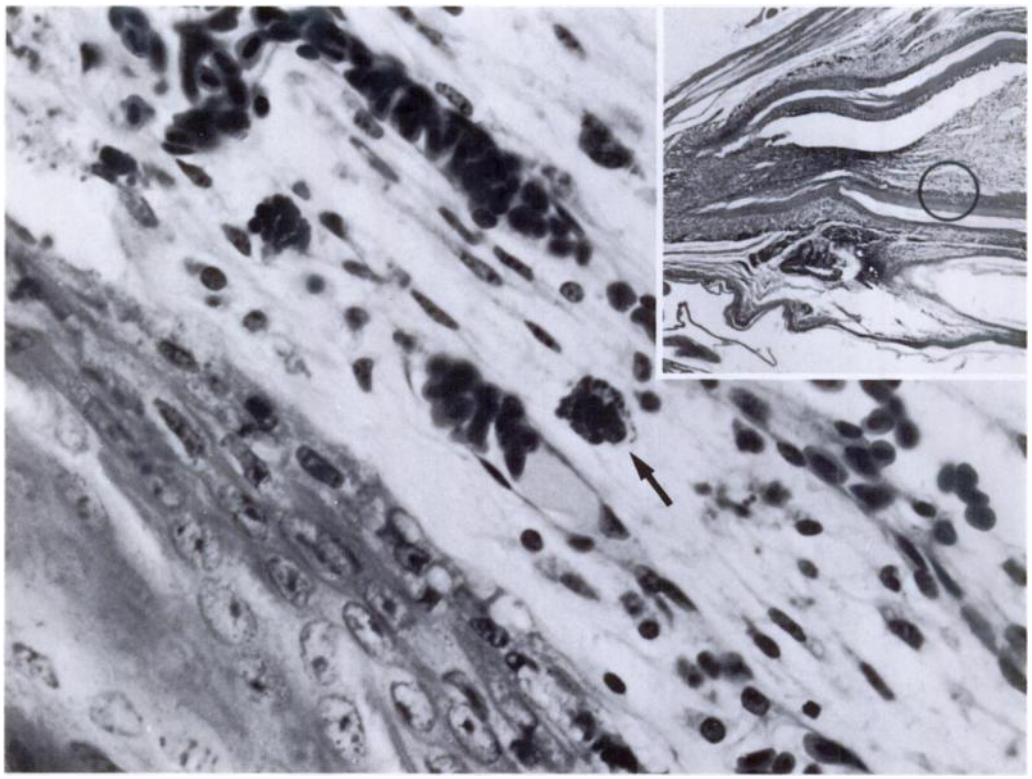

FIGURE 2. Affected sulphur-crested cockatoo feather follicle with necrosis and hyperplasia of epidermal collar and macrophage-like cell containing intracytoplasmic granules in pulp (arrow). ×700, H&E. Inset: Affected sulphur-crested cockatoo feather. ×20, H&E.

ment to the basal membrane (Fig. 1). There was moderate epidermal hyperplasia within these affected epidermal collars (Fig. 1). The changes noted were verified by comparison to a normal cockatoo feather epidermal collar (Fig. 2). The affected feathers also had numerous macrophage-like cells, with round nuclei, and several purple intracytoplasmic granules, within the epidermal collar and the pulp (Fig. 1). The pulp of some feathers contained hemorrhages and numerous heterophils. A thick layer of brown melanin (Fig. 3) was seen below the epidermis–dermis junction of diseased birds' skin. This layer did not continue into the feathers. Examination of the beak sections showed similar necrosis of the proliferative basal epidermis. This necrosis was associated with separation of the overlying laminae from the epidermis. There was some hyperkeratosis, and disorganization of the overlying laminae.

Ultrastructural examination of the inclusion material within the large macrophage-like cells in the epidermal collars and pulp of affected feathers revealed a regular lattice of particles 17–20 nm in diameter (Figs. 4 and 5).

Internal examination of the eight birds revealed that four were males and four females. No gross or microscopic lesions were detected in the internal organs.

DISCUSSION

The light and electron microscopic findings are consistent with a viral infection of the feather follicle. The morphol-

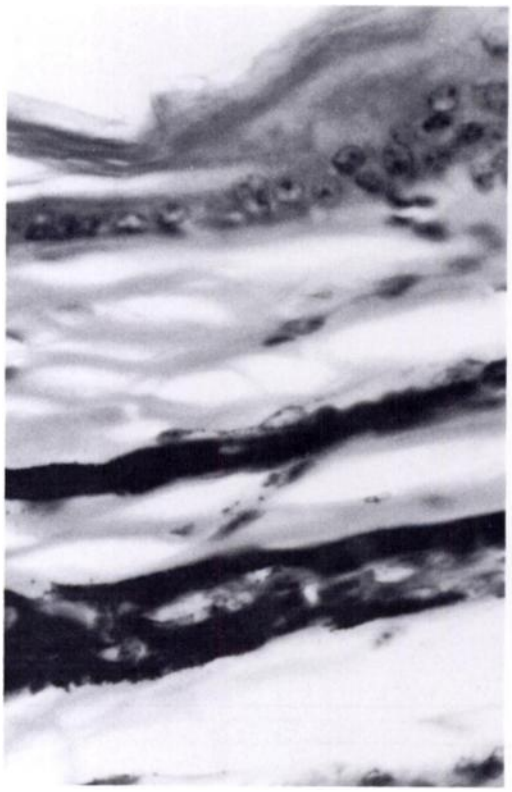

FIGURE 3. Melanin layer in the dermis of an affected sulphur-crested cockatoo. ×500, H&E.

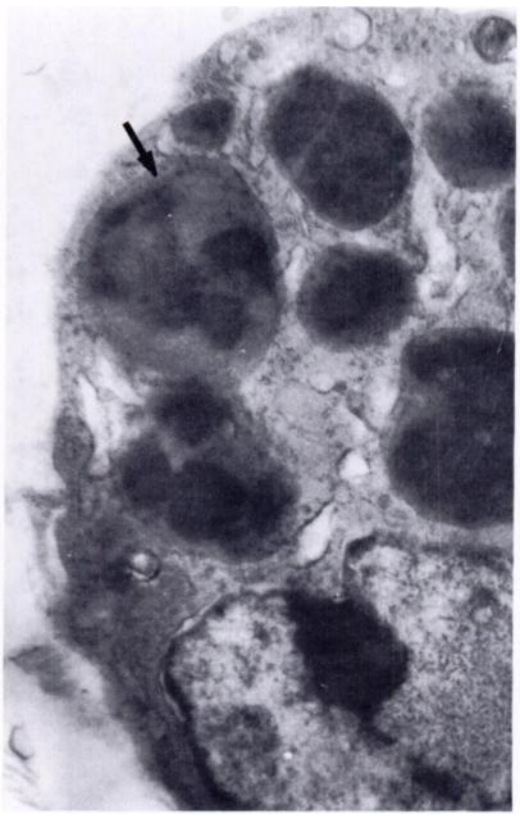

FIGURE 4. Macrophage-like cell containing intracytoplasmic inclusion bodies (arrow) in the feather of an affected sulphur-crested cockatoo. ×40,000.

ogy of the particles seen in the feathers was similar to that seen in mammalian and avian parvovirus infections (Gough et al., 1981; Pospischil, 1981).

A similar disease has been diagnosed commonly in captive Australian cockatoos (Perry, 1981; Pass and Perry, 1984) and the morphology of particles seen in feathers of those birds was also suggestive of a parvovirus infection (Pass and Perry, 1984). Wild birds have formed the major source of supply for captive bird keepers; therefore the disease may have been present in both wild and captive populations for some time.

Although the lesions in wild and captive birds are similar, melanosis of the bare skin appears to be greater in wild birds. Melanosis is probably associated with increased exposure of skin to sunlight, and exposure would be greater to wild birds than to captive birds. Affected captive birds also frequently have curled or clubbed feathers and feathers with retained sheaths (Perry, 1981). These deformities were not evident in the wild birds examined, but could occur in other affected flocks.

The formation and maturation of avian feathers is a complex developmental procedure, with the basal dermal papilla and epidermal collar differentiating into the rachis, barbs and other feather components (Lucas and Stettenheim, 1972), thus damage to the developing epidermal tissues of the feather would be likely to cause serious deformity of the feathers. It appears that the necrosis and dysplasia of the

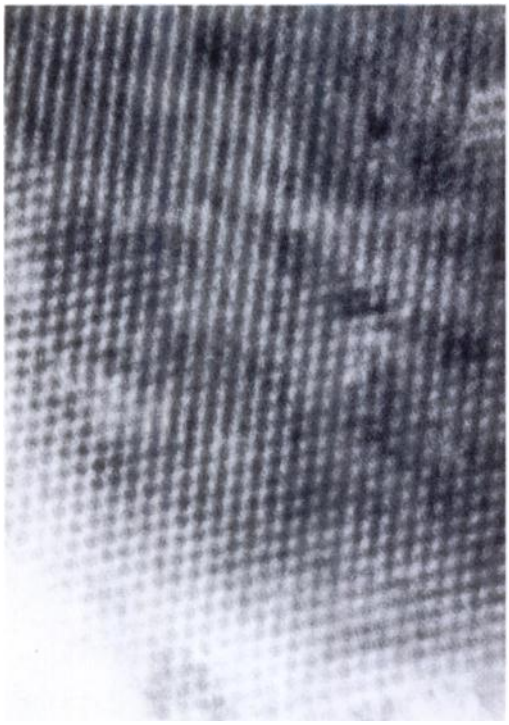

FIGURE 5. Higher magnification of virus particles in crystalline array, in an inclusion body in a macrophage-like cell. ×118,000.

feather epidermis of these cockatoos has resulted in a marked shortening of the feathers close to their basal origins, indicating that the necrosis has caused a cessation of feather growth.

Avian beak tissues mature from a stratified squamous epidermis, with a hard thick superficial corneum (Lucas and Stettenheim, 1972). Necrosis within some areas of the proliferative layers of the epidermis of the beak and the disproportionate growth of some areas of the corneum may have led to the longitudinal splits evident grossly in the beaks of affected cockatoos.

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