AVIAN DERMATOLOGY - NON-FEATHER PLUCKING DISEASES IN BIRDS

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INTRODUCTION

Although feather-picking is the commonest dermatological complaint seen in caged psittacine birds, there are many other causes of avian skin and feather disease encountered in practice. A full understanding of the structure and function of avian skin and feathers and of these other diseases is necessary as part of the logical diagnostic approach and rule-outs in any feather-picking case, and it should be appreciated that some diseases may or may not also be associated with feather picking.

ANATOMY

Avian skin is relatively thin with the epidermis consisting of only 2-10 cell layers. The dermis has a rich blood supply especially in more specialised area of skin such as the wattles. Beneath the dermis is elastic tissue and connective tissue which allows movement and attachment of underlying structures, but compared to mammalian skin is quite closely attached to these, making it relatively immobile. There are no sweat glands; instead heat is lost via the respiratory tract and by radiation from featherless areas. The brood patch is a featherless area that can occur in both sexes and is either seasonal or permanent depending on species. It allows close contact between the egg and the parent during incubation. Not all species have a brood patch, for example pelicans, penguins, boobies and gannets do not and instead cradle the eggs on their feet when incubating. The feet have shield-like plates of keratin named scutes. The foot has various digital pads to aid grip.

The uropygial gland or preen gland is bilobed gland that secrets lipoid sebaceous material that is spread by the beak over the plumage during preening. The gland lies dorsally near the tip of the tail. The size varies between the bird species. The gland is absent in the ostrich, Amazon parrot and pionus parrots. Beak conformation varies amongst the different bird species. The outer layer of the beak is formed by a thick stratum corneum. The dermis contains collagen and elastin fibres and is closely attached to the periosteum that blends into the bone.

Feathers

Feathers function not only to facilitate flight but also insulate, waterproof and protect the bird. Courtship, camouflage and display are other functions. Feathers are keratinized epidermis derived from follicles in the dermis. They are set in feather tracts known as pterylae, and the featherless tracts between them are known as apterylae. Not all species have apterylae, e.g penguins. These bare tracts can easily be mistaken as lesions or lacerations as the skin is so thin, but they are also useful for visualisation of underlying structures such as the jugular vein, without the need for plucking feathers. There are various types of feathers with different appearances and functions including contour, down, powder downs and filoplumes. The basic structure of a flight feather is a hollow shaft (rachis) and base (calamus). The feather vane is comprised of filaments called barbs which extend on either side of the rachis. The barbs are zippered together by rows of small hooks called barbules.

Contour feathers can be divided into:

Flight feathers (remiges) and tail feathers (retrices): primary flight feathers (9-16) attached to the metacarpal and phalangeal bones. The secondary flight feathers (6-40) are attached to the ulna. There are 12 tail feathers and 2-7 alular feathers. Feathers that cover the bases of the remiges and retrices are called coverts.

Body feathers: these feathers form a continuous waterproof layer over the body.
Down feathers cover the body below the contour feathers and provide isolation and buoyancy. Powder down feathers shed a fine waxy powder by continuously growing and the barbs at the tip breaking off. It helps maintain feather condition and water proof feathers. Natal down feathers cover the whole of a newly hatched chick until the first moult.

Moulting

New feathers developing in the dermal papilla force the shedding of old feathers above it. The shedding and replacement of feathers occurs at least once a year usually after breeding but can occur more frequent. The duration varies between species and between wild and captive populations. The moult starts with the inner primary feathers, followed by outer primary feathers and secondary feathers.

Birds in temperate zones moult prior to the change of seasons while tropical birds moult gradually and continually. Thyroid hormones are important in normal feather development but are not a stimulus for moulting. Changing of photoperiod and temperature are presumed to influence hormonal factors and induce moulting. In the northern hemisphere captive passerines moult between May and December for about 5-8 weeks. Canaries moult for 8-10 weeks between May and September. Parrots moult throughout the year. This varies from a continuous moult to 3 times a (e.g. budgerigars). The effects of moulting include cessation of ovulation and singing. Pruritus can occur and also a cyclic osteoporosis, making bones more fragile. The feathers are pulled out by the bird or fall out naturally.

Factors affecting moulting:

- Nutritional deficits such as hypovitaminosis A
- Reduced protein levels result in poor feather condition and 'fret marks'.
- Stress or fear can induce a stress moult.
- Reduction to shorter periods of light (especially after prolonged periods of light) induces moulting (through stimulation of the pituitary gland).
- Ectoparasites can cause abnormal moulting patterns.
- Hypothyroidism leads to retardation of feather growth.

Synthesis of thyroid hormones is similar to that in mammals. TSH from the pituitary gland stimulates the uptake of iodide and the production of thyroglobulin and also stimulates the hydrolysis of thyroglobulin and release of T_3 and T_4. In birds they both have a short half-life (diurnal pattern) making interpretation of a single serum sample difficult. The normal T_4 levels in birds are much lower than in mammals.

VIRAL DISEASE

PBFD (Psittacine beak and Feather Disease). Psittacine beak and feather disease is an important viral infection of parrots. It is caused by a DNA circovirus (BFDV) that can remain stable in the environment for up to a year. Single stranded DNA viruses such as BFDV readily undergo recombination and can therefore evolve rapidly, and molecular studies on BFDV genomes (full genome analysis) have revealed many strains that can have different effects and significance in different species. The virus is shed in feather dander and faeces and is transmitted by inhalation or ingestion. Vertical transmission is also possible (Rahaus et al., 2008). The virus favours rapidly dividing cells and the clinical signs are related to its effects on dividing tissues and the targeted systems are the epithelial cells, the gastrointestinal tract and the immune system. Birds that are exposed to the virus can develop a wide variety of clinical signs and have a varying incubation period. Latent infections are also possible.

In the chronic or classical form birds develop lesions between six months and three years of age. Rapidly growing feathers affected first (e.g. the powder down feathers). Feathers grow abnormally and can appear untidy, are dysplastic with retained sheaths, haemorrhage can occur within the pulp cavity, feather shafts can fracture and eventually complete feather loss can occur. Colour changes of the feathers are also common, e.g green birds develop patchy yellow feathers, blue birds develop patchy white feathers and African Grey parraots can develop red feathers. There can also be hyperkeratosis, overgrowth or fracture of the beak, and oral ulceration, typically seen in cockatoos. Some texts report the beak as being shiny or glossy as there is no powder down. Secondary infections are common as the disease is immunosuppressive. Typical examples include aspergillosis and chlamydiosis.

The acute form generally affects young birds during first feather formation and is characterised by depression ± diarrhoea followed by the rapid development of dystrophic feathers. These birds can also develop anaemia and a leukopaenia. An acute form is also commonly seen in cockatoo and African grey parrot nestlings. This typically occurs by sixteen weeks of age and in many cases within a month of purchase of a young parrot. This form is characterised by severe leukopaenia (on a blood smear you do not see hardly any white blood cells), depression, regurgitation and death, before feather lesions develop. Occasionally fret marks on the feathers may be seen. Often the only sign is sudden death. Birds can also suffer paresis from internal secondary pathogens. The strain affecting lorikeets can appear to be less pathogenic and apparent recovery is possible, but they are still shedding virus and may often relapse.

Budgerigars commonly carry PBFD and juveniles can exhibit normal feathering except for complete absence of primary and secondary flight feathers or become severely alopecic. Mild cases survive and can re grow feathers at the first moult (12 weeks old). The same signs occur with polyoma virus infection in budgerigars although in the UK PBFD appears to be more common. Polyoma is more likely to lead to nestling death with leucopaenia, anaemia and widespread haemorrhages.

Diagnosis is based on a PCR for the virus. This can be taken from pulpy feathers which can be gently plucked or from a blood sample, but the sensitivity is lower. It is present in the leucocytes and severely leucopaenic birds may have a false negative blood test. These tests are available commercially and some birds may have a negative test report sent to the new owners. HA and HI tests are also available. Sadly the bird may have been exposed to birds which are PBFD positive after this test result. If you are suspicious but have a negative test then a bone marrow biopsy can be taken for PCR. If the bird has died then the bursa of Fabricius in the cloaca or a skin biopsy may reveal the basophillc intracytoplasmic inclusions or lymphoid depletion typical of this infection. Any suspect

carcases should be kept frozen as a PCR can be performed on frozen tissue. Positive birds that are healthy should be quarantined and re tested in 90 days to see if they have cleared the virus.

Treatment is supportive, including treatment for secondary pathogens such as *Aspergillu*s spp and *Chlamydia* spp. Some work using avian interferon has led to an increased survival rate but the product is not available commercially. Mammalian interferon is of no use. Control is through proper quarantine with PPE and fogging wirh F10. Screen all birds prior to entry to a collection or if a pet bird once it is at home. Birds should be contained in separate air spaces to limit disease transmission.

Avian Polyoma virus (Budgerigar fledgling disease). Avian polyoma virus (APV) is a DNA papovavirus that affects all parrots and passerine birds. Typically budgerigars are clinically affected but it has also been reported as a cause of morbidity and mortality in other psittacines such as juvenile macaws, eclectus parrots and conures . Caiques, ring-necked parakeets, and lovebirds may be older, up to 1 year of age when affected. APV can aslo cause mortality in nestling passerines such as Gouldian finches (Alley et al., 2013). The virus is shed in the faeces, feather dust and secretions. Transmission is via inhalation or ingestion. Vertical transmission may be possible, but transmission to chicks can be horizontal as the virus is shed in crop secretions.

Clinical signs in budgerigars include neonatal death (can have abdominal distension, subcutaneous haemorrhage and reduced down and contour feathers). If neonates survive greater than 15 days they will lose tail and flight feathers and are known as 'French moulters'. PBFD can cause similar signs and concurrent infection of budgerigars with both avian polyomavirus and PBFD have been reported (Ramis et al., 1998). If they survive the feather loss resolves after several months. Recovered birds are carriers and shed when stressed. In other parrots, sudden death occurs or death after depression, weight loss, diarrhoea, regurgitation, subcutaneous haemorrhage, dyspnoea and polyuria. A chronic form manifests as weight loss, intermittent anorexia, polyuria, recurrent infections, poor feather formation and neurological disease. It is unknown but suspected that persistent infections occur in large parrots similar to the disease in budgerigars. Typically polyoma virus is more unusual in other parrots and typically very young birds only. In passerine birds, acute death in fledglings and adults can occur. Beak abnormalities and feather dystrophies may also be present. The psittacine PCR does not work in finches.

The diagnosis is made by PCR on blood or cloacal swabs, and to some extent clinically. On post mortem examination there is hepatic necrosis, bursal lymphoid depletion, membranous glomerulopathy, basophilic intranuclear and intracytoplasmic inclusion bodies in feather follicles and renal tissue. Control is dependent on good quarantine with closed flocks. In budgerigars the recommendation is to stop breeding and clean out facilities for 3 – 6 months. In other parrots, identifying shedders and removing them may be economically feasible. There is a vaccine in the USA. Parrots of low economic value should be housed in a separate air space away from more valuable tested (negative) parrots.

Poxvirus. Most poxviruses are species specific and transmitted by biting insects. Wet pox (diphtheroid form) occurs in mucosa (oral cavity and trachea). An acute septicaemic form also exists. Cutaneous or dry pox causes nodules, papules or vesicles on the skin. The cutaneous or dry pox is seen more commonly in raptors and songbirds as nodular lesions on feet, eyes or face. Canaries and finches develop lesions on the face, mouth and feet and develop severe pulmonary complications. Agapornis (lovebird) poxvirus causes lesions on the face, oral and nasal cavity and axilla, shoulder and abdomen. The lesions are discoloured, pruritic and secondary bacterial infection can develop. Budgerigar

poxvirus is considered apathogenic. Amazon poxvirus has a self-limiting cutaneous form and a diphtheric form with high mortality. Diagnosis is through histology or impression smears (intracytoplasmatic eosinophilic inclusion bodies). Infection is generally self-limiting (3-4 weeks) and treatment will include supportive care and antibiotics for secondary bacterial infection. A vaccine (Columbovac PMV/Pox, Fort Dodge) is available for pigeons. Control of insects is also important.

Papilloma virus.Papilloma virus is associated with benign epithelial tumours. Therapy depends on their location (cloaca, oral cavity or skin). Papillomas may reoccur after removal and spontaneous remission has also been described.

PARASITES

Mites.Mites include *Cnemidocoptes* that causes scaly legs and face. These mites are normally located on featherless areas and their burrowing activity stimulates hyperplasia and hyperkeratosis. They are most commonly seen in budgerigars causing beak deformities and leg lesions in canaries. The mites spend their entire life cycle on the bird and are transmitted from bird to bird.

Dermanyssus spp. (red mites)are usually visible at night on the bird and spend the day free living in the cage or nest. They are primarily bloodsucking mites causing anaemia and death in fledglings. A white sheet placed over the cage at night will enable to visualise the mites the following morning. They can get on to humans causing irritation.

Lice.Lice may cause feather damage and irritation if in high numbers. This occurs when the bird is debilitated or due to poor husbandry. Lice appear to be host specific and cluster in a specific area of the body. The complete life cycle occurs on the host.

Giardia. Giardia a protozoan has been described as a cause for feather plucking in cockatiels and budgerigars due to hypersensitivity, or possibly a malabsorption/nutritional effect . A fresh direct faecal smear can be examined for trophozoites. Metronidazole at 30mg/kg PO SID for 7-10 days is the treatment of choice.

BACTERIAL AND FUNGAL DISEASE

Primary bacterial and fungal disease of the skin is less common. Bacteria isolated from skin infection in birds include *Staphylococcus sp., Mycobacterium, Mycoplasma* and *Aeromonas. Candida* has been associated with hypovitaminosis-A. *Microsporum, Trichophyton*, *Cryptococcus neoformans* and malassezia have been reported.

Ulcerative dermatitis in Lovebirds.The exact aetiology is unknown and may be associated with poor nutrition, giardia, *Agapornis* poxvirus, viral infection and bacterial infection. The birds mutilate along the patagial membrane, axilla, shoulder and neck. Diagnosis would include dietary history, blood samples for health profiles and screening for avian viruses. A swab for bacterial culture and skin biopsy can be taken from the lesions. Treatment depends on the diagnosis. Surgical debridement and closure may be indicated if the wounds do not heal with supportive care. A collar may have to be placed concurrent with therapy.

NUTRITIONAL FACTORS

Chronic malnutrition commonly presents as deterioration in the integument and plumage when malnutrition has reached severe levels. Hypovitaminosis A affects moulting and causes hyperkeratosis and glandular metaplasia of the uropygial gland. Reduced protein levels result in poor feather condition and 'fret marks'. Lysine, methionine and cysteine are essential amino acids necessary for feather quality, growth and pigmentation. They are deficient in all-seed diets. Severe deficiencies are required before feathers are affected. All changes are potentially reversible but owners should always be advised that improvements will take up to 18 months as new feathers will need to be produced.

HYPO- AND HYPERTHYROIDISM

Hypothyroidism leads to retardation of feather growth. True hypothyroidism is often misdiagnosed, and a low blood thyroid level is not definitive for a diagnosis in pet birds. Thyroid hormone levels fluctuate throughout the day and many general disease conditions can also lead to lower circulating blood thyroid levels. A positive response to thyroxine is also not a definitive diagnosis, which necessitates a TSH stimulation test. The use of the TSH stimulation test has been reported in parrots at 1.0 IU/kg IM and a repeat blood sample is taken 6 hours later for T_4 level. The T_4 should at least double following administration of TSH. L-thyroxine at a dose of 0.02mg/kg orally once or twice daily has been used for confirmed cases of hypothyroidism. Hypothyroid birds typically are chronically obese and have a marked lipaemia. Hyperthyroidism can cause PU/PD, regurgitation, tachycardia, weight loss, convulsions and death.

BROWN CERE HYPERTROPHY

This condition is seen in older female budgerigars with hyperplasia of the cornified layer of the cere or in males with testicular tumours. Intervention is not required unless it is growing towards the skull or occluding the nares. Hypertrophied material can be scraped away.

CUTANEOUS GROWTHS

Cysts.Follicular cysts are commonly seen in canaries with keratinaceous debris accumulating in an occluded feather follicle or a deformed feather within a follicle. They are now considered to be basal cell tumours or feather folliculomas a benign neoplasm. They occur most commonly on the wing and along the back. Treatment of a small isolated lesion is not warranted. If there are multiple lesions or the lesion is causing discomfort it can be surgically removed using an electrosurgery unit. Feather cysts are likely to re-occur.

Squamous cell carcinoma. These are common malignant tumours seen on the skin and oral cavity of budgerigars. They can be locally invasive and need surgical removal. Cryosurgery can be used.

Polyfolliculitis.Multiple feather shafts appear from the same follicle and generally no systemic effects are noted. The cause is not known but one should screen for aetiologies for skin and feather disorders.

Lipomas. Lipomas are benign proliferation of lipocytes commonly seen in budgerigars in the subcutis of the sternum and abdominal skin. Obesity and advancing age predispose birds to developing lipomas. The bird should be placed on an appropriate diet and allowed plenty of exercise.

Xanthomas. Xanthomas are locally invasive benign masses consistent of foamy macrophages, multinucleated giant cells and cholesterol crystals. They are common in budgerigars and to a less degree in cockatiels. Yellow friable tissue is distributed over dorsal areas of the bird or over underlying conditions. Feed low fat and protein diet when possible. Some xanthomas are thyroid responsive (0.1mg tablet L-thyroxine in 120ml drinking water).

Epitheliolymphosarcoma. These can present as gradually enlarging, crusty, pruritic skin lesions that do not respond to therapy.

FOOT DISEASES

Constricted toe syndrome. This occurs when an encircling fibrous scab or necrotic tissue inducing an avascular necrosis of the distal extremity. The aetiology is unknown and possible causes include low humidity and egg-related strictures. To relieve the stricture multiple incisions at right angles are made over the fibrous stricture.

Bumblefoot.Bumble foot or pododermatitis can be classified into three grades according to the changes in appearance of the plantar surface of the foot, i.e. metatarsal pad and digital pads. An alternative 5 grade system can also be used.

Type I :mild and localised with either flattening of the epithelium or hypertrophy of the epithelium to form a corn. There may or may not be swelling and heat. Good prognosis.
Type II: can be a degeneration of type I or caused by a puncture to the foot. Bacteria such as *Staphylococcus aureus* are isolated but *Escherichia coli, Corynebacterium* and *Pseudomonas* have also been isolated. The foot will be hot and swollen. Fair to good prognosis.
Type III: the infection will involve ligaments, tendons and joints. Guarded to poor prognosis.

Bumble foot in waterfowl is caused by chronic wear, pressure or abrasions generally due to poor substrate and/or obesity. In raptors lack of exercise, inadequate perches or surfaces and increased weight bearing on a foot are causes for bumble foot. Puncture wounds caused by a claw or prey can also lead to bumble foot. In parrots poor perches, obesity and hypovitaminosis A lead to bumble foot. Treatment will depend on the grade of bumble foot and in mild cases may only need changes in husbandry and in severe cases surgery. Corrective husbandry will include supplementing the diet with vitamins and minerals, covering the perch with astroturf, reducing the bird's weight and increase exercise. Feet can be bathed or the perches soaked with dilute chlorhexidine or povidine-iodine. Bandaging can range from a corn plaster to ball or doughnut bandages. Antibiotic therapy should be based on culture and sensitivity, lincomycin (50mg/kg BID PO) or cloxacillin (250mg/kg BID PO) have been suggested. Removal of the scab and debridement of infected tissue under general anaesthesia will be needed in more advanced cases. Radiograph should be taken to assess joints and bones.

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Table 1. Treatment options for ectoparasites

Agent	Trade name	Use	Dose
High cis permethrin	Harker's Louse powder	Mites, lice and fleas	Powder applied throughout plumage and repeated every 2-3 weeks Licensed for pigeons
Fipronil	Frontline	Mites, flies, flies and ticks	Apply frontline spray to cotton bud and apply to head, under wings and base of tail. Repeat every 2-4 weeks.
Ivermectin	Panomec	Mites and myiasis	0.2mg/kg given by injection or orally at 2-4 weeks interval. Topically diluted with propylene glycol or in the water for canaries at 1mg/l drinking water. Toxicity has been reported in finches.
Environmental Control			
Cypermethrin	Dy-Sect (Deosan)	Mites	Dilute to 2%
Malathion	Duramitex (Harkers)	Mites	Dilute 0.93% paint or spray on perches
Permethrin/pyriproxyfen **Methoprene/Permethrin**	Indorex (Virbac) Zodiac + (Birdcare co.)	Mites, ticks and fleas Fleas and mites	Spray Spray