The Gouldian Finch (*Erythrura gouldiae*) in Health and Disease

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The Gouldian Finch (*Erythrura gouldiae*) is a very popular passerine species within aviculture. Its popularity has resulted in it being almost domesticated with many colour mutations having been established around the world. Despite its secure captive status, this species is suffering from a serious decline in numbers in its native habitat in northern Australia. Excessive cattle grazing and altered fire regimes are thought to be key factors in this decline. Infection with the parasitic mite, *Sternostoma tracheacolum*, is thought to be of secondary importance. In captivity, despite its popularity, it has the reputation of being delicate and susceptible to many diseases. A range of viral, bacterial, mycotic, protozoal, helminthic and arthropodal agents and metabolic disorders have been identified and implicated as contributing to the illness and death of many Gouldian Finches. The presence of many diseases such as mycobacteriosis, cryptosporidiosis, microsporidiosis and avian gastric yeast which are commonly considered as secondary pathogens is discussed. The importance of suitable housing and diet, decreasing environmental stressors and optimizing hygiene are also stressed.

The Gouldian Finch (*Erythrura gouldiae)* is a very popular passerine species of the family *Estrildidae* which will illustrate the husbandry and health issues which face many commonly kept "finch" species. It also is listed as endangered with its numbers continuing to decline in the wild.(1) Hence understanding its biology & captive requirements are both important for its ongoing survival.

Natural History

This species is found in northern Australia from the Kimberley Region in Western Australia across the northern part of the Northern Territory, north-west Queensland to Cape York. It appears to be more numerous in the Northern Territory and Western Australia. Currently, the population is estimated at 2,500 breeding birds, but its numbers are continuing to decline.(1)

Within its range it inhabits open tropical woodland that has a grassy understorey, particularly in hilly areas. They are never found far from water. They feed almost exclusively on half-ripe & ripe grass seed .This consists of annual grasses such as *Sorghum* sp during the dry season & various perennial grasses during the wet season. They will take insects during the breeding season, especially swarming termites, but captive birds seem less interested in livefood. Nesting occurs toward the end of the wet season from January to April and occurs almost exclusively in hollow logs. Average clutch sizes are 5.2, pairs often multiple brood but productivity is typically low at 1.5 fledglings per pair.(1)

Gouldians are very sociable birds gathering in large flocks during the non-breeding period and even nesting proximity in surrounding trees. These breeding birds will often come together daily with loud social calls, often in the late afternoon to bathe, feed or just sit together.(1,2)

Threats to this species appear to be primarily habitat related.(1) Cattle grazing & altered fire regimes are the main concerns, either because cattle prevent grass from producing seed that is essential to the finch, or because the birds need patchy burning and the fire history of the landscape has become too uniform. Infection with the parasitic tracheal mite S*ternostoma tracheacolum* is now considered to be of secondary importance.(1) Mining may also adversely affect birds at a local scale and in the past trapping for the avicultural trade may also have been a factor. Availability of nesting habitat and low breeding success are not thought to be limiting.(1)

Avicultural History

The Gouldian Finch first reached England in 1887 and continental Europe in 1895 (2). It was first bred in Australia prior to 1886. Due to its beautiful, vibrant colouration it has become a firm favourite amongst aviculturists the world over. It has become domesticated with the establishment of many different colour mutations. It has had a reputation as being a very prolific breeder but also as being very delicate if not kept under appropriate conditions.

Avicultural Requirements

Housing

These birds should be kept in a dry, warm environment. Although some aviculturists have successfully kept them in open flight aviaries, the experience of most suggests that they quickly succumb, especially in cool, wet weather.(3,4) In temperate & warmer climates they are best housed in fully enclosed aviaries with only the front open to allow access to sunlight & fresh air. These should have the facility to be covered at night or during inclement weather. The use of opaque building materials such as polycarbonate or glass, will allow light to enter and help warm up the aviary during the day. These birds seem to thrive in hot conditions, even when daytime temperatures reach over 40 degrees Celsius (104 degrees Fahrenheit), but are less able to tolerate cold, damp conditions. Therefore, aviaries should be positioned to receive maximum sunlight exposure, but protect the occupants from the prevailing weather conditions. The floors should be kept as dry as possible and inorganic substrates will help to decrease the effects of pathogens. Suitable aviary furnishings such as perches, cut shrubbery & branches affixed to aviary walls and the planting of seeding grasses can further enhance the birds' environment. As aviary birds, Gouldians are best kept either as single pairs(4) or in colonies of at least three pairs, where their behavioural interactions can be appreciated.(3) In colony situations, breeding results can be enhanced by adding an extra male bird to stimulate the other males within the colony.(3) This species is inoffensive to other birds and so can be kept with other small to medium sized, non aggressive seed-eaters. However, they are more likely to be the victims of stress from other species, even if only subtly, which may predispose them to illness. They really are best kept on their own for this reason in many breeders' and veterinarians' opinions.

These birds can also be housed as single pairs in smaller cages or breeding cabinets.(4) This method is practiced in many parts of the world, particularly in the colder climates where they are kept in temperature-controlled rooms. They are also popular as ornamental pets due to their bright colours and so are kept in many households purely for their aesthetic value.

Diet

As mentioned previously these birds are primarily seed-eaters so a varied dry seed diet has been recommended. This usually consists of a variety of millets & plain canary seed, with small amounts

of hulled oats & oil seeds during the colder time of the year. This should be supplemented with non-toxic half-ripe seeding grasses of a size suitable to the birds. These will vary in different parts of the world, so it would be best for keepers to become familiar with the varieties suitable in their locality. The same can be said of green food. This species will eat domesticated varieties of leafy vegetables, particularly if shredded, and also wild varieties such as chickweed, dandelions and flowering heads of milk thistle. The important factors to consider when feeding seeding grasses or greens to birds is to ensure that they are collected from uncontaminated sources, that they are fresh & not infected by fungi etc, that they are fed soon after collection and that they be fed in gradually increasing amounts rather than in large amounts irregularly which can lead to gastrointestinal disorders. Any uneaten portions should be removed from the enclosure before being allowed to go mouldy. This species prefers to feed from a raised feeding platform and seldom takes food from the ground. This equates to their wild habit of clambering about tall grasses taking the seeding heads.

Gouldian finches also show a strong liking for grits, both soluble & insoluble. They are particularly keen on sources of calcium such as cuttlebone & baked chicken eggshells which they devour during the breeding season & have been observed eating charcoal in the wild. There is some debate in the veterinary literature about the suitability of feeding finches grit items & the risks of gastrointestinal impactions. Suffice it to say that if grits are fed, the birds should always be monitored for signs of pica or illness at which time the grits should be removed.

There is also some evidence to suggest that Gouldian Finches are not capable of producing their own Vitamin C (3,5) so adding ascorbic acid to their diet may be of benefit.

Soft food preparations such as egg and biscuit mixes as fed to canaries may be of benefit particularly during periods of higher protein requirements such as during breeding and moulting. However, these foods are frought with dangers such as risk of *Salmonella sp* poisoning & spoilage issues. A much better suggestion would be to include a dry pelleted or crumble manufactured for breeding finches. If manufactured by a reputable producer, these possess the perceived higher protein requirements, have a broad range of amino acids, vitamins, minerals & trace elements and hopefully are manufactured pathogen-free. However, determining the nutritional requirements of passerines is still in its infancy, so these foods at best are good guesses. Many estrildid passerines are conservative feeders and Gouldian Finches are no exception so converting these birds onto synthetic diets can be even more challenging than it is for small psittacines. However, the continued domestication of this species should make it more adaptable to new food items.

Livefood can also be fed to this species but the concensus of opinion amongst aviculturists the world over is that it is not required for this species and that most individuals will ignore it. Termites have been the best accepted of these in Australia, particularly when they swarm.

Breeding

Pairs usually nest in artificial nest boxes. They are poor nest builders so boxes are usually lined with dry grasses provided by the aviculturist. Some pairs add to this themselves but feathers are not used to line the nest. The best nests are dome-shaped whilst the less well constructed nests may be little more than cup-shaped. Hens typically lay from five to seven white eggs, sometimes more. At times several hens may lay in the same nest. Egg incubation, which lasts fourteen days, commences with the laying of the fifth egg. It is shared by both sexes during the day, but the female usually incubates alone at night. Chicks are born naked. The adults dispose of the eggshell but do not carry out any great degree of nest hygiene. The chicks' eyes open between the seventh and tenth days of life. The primary feathers erupt at the eleventh or twelfth day. The female generally ceases

brooding the young when they are approximately ten days old, which can result in the loss of the chicks on cool nights. Thus, this is a critical period in the development of the young. The chicks fledge after approximately twenty days. The fledged juveniles begin to moult at six to eight weeks of age and normally finish moulting by three to four months old, but this can be delayed for months. Some birds bred late in the season do not achieve adult colouration for up to eighteen months. The period of juvenile moult is the second critical period in the life of the Gouldian Finch. It seems that warmth, a varied diet and minimal stress are important in allowing birds to moult quickly and with maximum survivability.

Some breeders foster Gouldian finches under the domesticated Bengalese (Society) Finch *(Lonchura domestica).* This can result in more chicks reared per pair but issues of health (eg *Cochlosoma* sp., *Campylobacter* sp., microsporidiosis infection) and imprinting need to be addressed.

Gouldian Finch Medicine

Viral Diseases

Several viral diseases have been reported to occur in finches in general, and in Gouldian Finches specifically.

Avian polyomavirus (APV) has been diagnosed in several passerine species, but Gouldian finches tend to be very susceptible (8-15). Signs of the disease include early nestling mortality, ejection of chicks from the nest, poor fledgling growth, lack of vigour, additional disease problems, abnormal beak and feather growth and delayed or incomplete moulting. Blood in the droppings has also been noted on some occasions. Birds which survived one outbreak were undersized, had dirty plumage and were slow to fledge. Many of these birds had an abnormally long & tubular lower mandible. Flocks which experience losses during one breeding season may not experience any during subsequent seasons. Surviving birds have been shown to have moderate levels of antibody that will neutralize a lovebird- derived APV. It has been suggested that this virus may be responsible for predisposing young Gouldians to other chronic diseases.

The most consistent post mortem lesion is a swollen, pale liver which in many cases has been mottled by recent haemmorhages. On histopathology, hepatocellular necrosis with intranuclear inclusions can be noted. Myocarditis is another common finding in passerines. Lymphoid depletion and or macrophage hyperplasia of the spleen, gizzard epithelial necrosis and mononuclear cell infiltration in the kidneys and intestines have also been described. Large clear to basophophilic intranuclear inclusion bodies can be found in infected organs. In some peracute infections of nestlings, no histopathologic changes could be found. Evidence of secondary mycotic, bacterial and/or parasitic infections may also be found. Electron microscopic examination of the intranuclear inclusions reveal discrete, round to icosahedral, electron-dense particles measuring 45-50 nm in diameter, typical of avian polyomavirus. Currently available PCR tests do not appear consistently suitable for detecting polyomavirus in passerines. Although APV DNA was detected in the tisues of one finch with psittacine-derived APV PCR primers, other studies suggest that another significantly different virus may also infect these birds.

As with many viral diseases there is no known treatment, however supportive care, good stock selection and optimized housing, feeding & hygiene practices are all important. Suitable disinfectants include sodium hypochlorite, chlorine dioxide and phenolics. Resting of breeding birds may also stop shedding of the virus, as has been experienced with budgies. Treating any

concurrent secondary infections may also help reduce the impact of an outbreak, although many birds die before any treatment can be instituted. One author has vaccinated the adults of an affected flock, after which neonatal and juvenile mortalities ceased. O.1ml of the avian polyomavirus vaccine (Biomune APVV, Biomune, Lenexa, Kansas, USA) was administered by shallow intramuscular injection. No adverse reactions to the vaccine were noted.(8) The suitability of this vaccine for passerines needs further investigation.

Herpesvirus- like lesions have been responsible for epidemics of conjunctivitis with respiratory distress and a mortality rate of up to 70-100% in Europe, in African and Australian finches, including Gouldian finches.(14-16) Basophilic intranuclear inclusions have been noted in the hugely swollen nuclei of conjunctival, oesophageal and tracheal epithelial cells. Electron microscopy has revealed cytomegalovirus-like particles. In a review of 8 cases involving 25 finches in the USA from 4 outdoor aviaries, clinical signs included ocular discharge, weakness and occasionally a head tilt. (16) Variably increased mortality was noted (25-100%) as was sudden death. Gross & microscopic lesions included conjunctivitis, rhinitis, sinusitis, tracheitis, bronchitis and oesophagitis associated with hypertrophied cytomegalic epithelial cells containing intranuclear inclusions. Herpesvirus particles were seen in the nucleus and occasionally in the cytoplasm of affected tissues by electron microscopy. In a separate case, a bird with viral hepatitis was characterized by eosinophilic intranuclear inclusions suggestive of herpes virus but no further confirmation was made. No treatment or prevention is known. One outbreak occurred four weeks after the introduction of wild-caught waxbills. Strict hygiene and quarantine measures should be instituted in an outbreak to prevent further spread of the infection.

Paramyxovirus III has been associated with head tilts, circling, torticollis and other vestibular signs in the finch .(18) Affected birds may also exhibit depression and variable degrees of weight loss. The birds can be carriers for months before clinical signs are noted. Diagnosis is presumptively based on the clinical signs. Serology and virus isolation are required to confirm the diagnosis. Necropsy findings tend to be non specific but histopathological findings tend to be characterized by a severe pancreatitis. Lesions may also be seen in the inner and middle ear which should always be examined in birds exhibiting CNS signs. A common differential for similar clinical signs is vitamin E deficiency caused by feeding rancid cod liver oil, as well as other infectious, toxic and metabolic causes. PMV type III has been documented in the USA in Gouldian Finches (8). Again there is no specific treatment.

Eastern Equine Encephalomyelitis virus has also been reported to cause nervous signs such as paresis and death in Gouldian Finches kept outdoors in Florida. (8,15,19). Brain and spinal cord should be histologically examined in suspect cases. Protection from insect vectors and supportive care will help stop the effects of this virus in areas where it is endemic in the wild bird population.

Other viral diseases will no doubt be found in passerines in the future, as more specimens are submitted for veterinary treatment, necropsy and histopathological examination. For example, lesions resembling those caused by Proventricular Dilatation Disease have been described in Gouldian Finches. These include a lymphocytic ganglioneuritis seen in the ventriculus, oesophagus/crop or proventriculus of two birds.(8)

Bacterial Diseases

A variety of bacteria have been found to cause illness and death in Gouldian finches, either alone or together with other pathogens.(8,10,11,13-15,20) The lesions found are consistent with generalised

septicaemia or bacteraemia or more specifically may infect particular organs such as the liver, intestine, lungs or reproductive organs. Large numbers of coliform or gram positive organisms are not normally found in the gastrointestinal and respiratory tracts of healthy passerines (21)

Bacteria of the family Enterobacteriaceae are frequently isolated. *Escherichia coli* causing colisepticaemia has been responsible for a variety of clinical signs. Lethargy, anorexia, fluffed appearance, diarrhea, polyuria, chronic weight loss, dyspnoea, reproductive disorders including metritis and egg-related peritonitis, and nestling mortality have all been described(8,10,18,20). In one case of enteric colibacillosis, the passage of whole seeds in the droppings were noted before death.(8) Post mortem lesions can vary but serofibrinous inflammation of several body organs, particularly of the liver & kidney is one of the more common lesions. Granuloma formation may also be noted. In nesting birds, infection can occur at any time from egg formation, incubation through to hatching time due to faecal contamination. Youngsters infected during the first few days of life can suffer enteritis from enterotoxin production. The chicks can quickly dehydrate and become cachexic. The nests become dirty, wet and yellowish. The brooding female also appears wet. Youngsters may die within the first week of life. Antibiotic choice should be based on culture and sensitivity results and general aviary hygiene and management practices should be improved. The administration of avian lactobacilli to lower intestinal pH and facilitate the colonization of normal gut flora may also be of benefit.

Salmonellosis has also been diagnosed in these birds. *Salmonella typhimurium* was cultured from birds suffering from granulomatous ingluvitis and/or enteritis and gastrointestinal ulcers. Other cases reveal small, yellow focal bacterial granulomas on the liver and spleen. Sometimes focal necrosis in the heart, lung and pectoral muscle can occur. Swollen joints and arthritic lesions as seen in racing pigeons have not been described in Gouldians to date but arthritic lesions have been seen in canaries.(15) Transmission is primarily through the oral route but egg transmission is also possible. Contaminated dust from faeces and feathers may aid aerosol spread(10). Rodents, free ranging birds and insects may act as vectors. (11, 20) Affected birds are usually too ill to respond to treatment but identifying appropriate antibiotics can help save cagemates. The possibility of lifelong shedders post infection means that affected birds should be culled, although this is controversial.

Klebsiella spp and *Enterobacter* spp are other Enterobacteriacea which may be primary or secondary pathogens.

Pseudomonas spp. have been responsible for severe necrotizing or perforating enteritis lesions, some of which have resulted in peritonitis. The organism may cause foul smelling diarrhea or mucopurulent pneumonia and air sacculitis. (10) Histologic findings include moderate to heavy infiltrates of lymphocytes, plasma cells, and heterophils within the lamina propria.(8) Infections may originate from contaminated drinking water systems, or poorly prepared soaked seed or soft food. Concurrent environmental stressors are also important. Many strains are resistant to commonly used antibiotics so culture and sensitivity are advised

Campylobacter spp. have been isolated from several ill birds. *C. f*etus *var. jejuni* have been associated with the production of pale voluminous droppings and nestling mortality.(22)The pale faecal colour is due to te presence of undigested starch. Society *or* Bengalese Finches *Lonchura domestica* are implicated as asymptomatic carriers, but when they are used as foster parents for Gouldian Finch chicks, the chicks suffer. Culture and sensitivity are required for diagnosis and appropriate treatment but erythromycin appears to be one of the most consistently successful antibiotics. European investigators have suggested that bolstering the immune system by feeding

increased animal protein, mineral and vitamins in the form of “soft food” may protect against repeated infections.(22) The role of any fostering also needs to be reconsidered.

Enterococcus has been associated with enteritis and tracheitis, pneumonia and air sac infections. It needs to be differentiated from infections caused by the respiratory mite *Sternostoma tracheacolum*. It is resistant to many antibiotics.

Staphylococcus aureus has caused pododermatitis in some Gouldian Finches.(8). It has been associated with respiratory and gastrointestinal disease in Zebra Finches (*Taeniopygia [Poephila] guttata*), with affected birds showing septicaemia and arterial thrombosis leading to ischaemic necrosis (11)

Lactobacillus was recovered in heavy growth from the affected lungs of one bird.(8)

Other bacterial isolates taken from ill Gouldian Finches include *Corynebacterium, Moraxella-like sp, Streptococcus* and *Yersinia pseudotuberculosis* (particularly in winter in rodent infested aviaries).Yersiniosis is characterized by enteritis and hepatic and splenic multifocal abscessation(20). Affected birds exhibit dyspnoea, diarrhea or just acute deaths.(10) Sick birds respond poorly to treatment but appropriate antibiosis and management changes will halt the progress of the disease.

Avian mycobacteriosis (*Mycobacterium avium* complex) appears to be overrepresented in Gouldian Finches compared to other passerines. (23,24) As with other avian species, this still presents as a chronic disease, with mainly alimentary and liver involvement,(24,25) although respiratory lesions can occur.(26) Sudden death may be the only presesenting sign, although affected birds are usually thin. Enlargement of the liver and spleen are often seen with grey foci being occasionally seen in the lungs.(24-26) It is important to note that mycobacteriosis in passerines usually produces diffuse granulomatous, non- caseous lesions rather than discrete tubercles as found in galliforms.(24-26) As well as sporadically infecting individual birds, an outbreak involving 10 of 17 birds has also been recorded.(24) One author describes the presence of mycobacteria within macrophages of the dermis in some finches which have exhibited feather loss on the head and/or face.(8) Acid-fast organisms were found within these dermal macrophages, although no dermal granulomas were seen. These birds also exhibited diffuse enlargements of the spleen and liver, sometimes associated with pallor. Ante mortem haematology performed on one bird revealed a mild leokocytosis (total WBC 18,000). Weight loss was typically seen in most affected birds. Two birds exhibited head tilts and seizuring and one bird showed diarrhoea with faecal pasting.
Most cases from finches are diagnosed by the presence of acid-fast bacilli within macrophage cytoplasm. Attempts to culture from these samples is often unrewarding, despite the use of a variety of specialist mycobacterial media (8,24),. The potential zoonotic risks associated with mycobacterial infections need to be considered and the practicalities of long term multi-pronged treatments assessed before any treatment courses are begun. Removal of birds from soil floored aviaries would be one important way of minimizing the impact of this disease within a collection. Also harvesting the eggs from the survivors of an affected flock and fostering these under less susceptible finches has also been suggested. This would only be successful if the health integrity of the fostering birds could be assured.

Fungal Diseases

Candidiasis of the pharynx, crop and endoventriculus has been commonly observed in Gouldian Finches.(8, 10-15,27) Typical lesions include raised and thickened yellow-to-white areas on the mucosal surfaces giving rise to the "Turkish-towel" lesions described in many texts. Affected birds usually exhibit anorexia, weight loss, regurgitation, diarrhea, passage of whole seeds in droppings and crop stasis. In one study, candidiasis was responsible for a fatality rate of 35% over an 18 month period in Gouldian nestlings. (27) This disease may be seen in association with other infectious agents and is considered a disease of the immunocompromised but its possible role as a primary pathogen in this species has also been raised (27). Samples taken from the crop or cloaca or faeces may reveal large numbers of budding yeasts. Be wary of the presence of non-budding yeasts which may be dietary in origin and not of pathological significance. Predisposing causes such as prolonged antibiotic therapy, malnutrition, spoiled food and stress all need to be addressed.

A syndrome of baldness and facial feather loss is commonly seen in Gouldians. As well as the mycobacterial aetiology discussed previously, it more commonly has a fungal cause, with hyphae demonstrable in the skin.(8) A mild scaliness of the skin can also be seen. Beak necrosis in this species has also been attributable to fungal infections. The base and surface of the upper beaks appear flaky and are white or yellow in colour. Fungal hyphae have been demonstrated in the beak matrix by special staining.

Treatments for these conditions include most of the commonly used antifungals.(8) Nystatin at 100 000 IU per litre of water and/or 200 000 IU/kg soft food for 10 days; ketoconazole at 30 mg/kg PO BID or 200mg/litre of water or 250mg/kg softfood for 2 weeks, fluconazole at 150 mg per litre of water OR 5-10 mg/kg PO SID in orange juice OR 10mg per 100 cc's of soft food or flucytosine at 250 mg/kg PO every 12 hours for 14-17 days (28) or itraconazole at 10 mg/kg PO every 12 hours for 21 days. One study found that blood levels of itraconazole well above the therapeutic threshold were achieved and had no ill effects in the Gouldians treated.(27). This was achieved by adding 0.1ml of water to 100mg itraconazole and mixing to a paste. This was left overnight. In the morning it was shaken up with 5ml olive oil and pourd into 100 gm seed and thoroughly mixed. This mix was fed to twenty birds first thing each morning and no ore seed given until all treated seed was eaten. This treatment was given over four weeks with no sign of toxicity and with elimination of the candida from subsequent faecal sampling. Diluted unscented chlorhexidine solution (eg Nolvasan 2% solution) added daily to the drinking water may help to prevent further infections during an outbreak of gastrointestinal disease or during periods of increased susceptibility (28). Miconazole can also be used topically. An outbreak of candidiasis was controlled by one author simply by increasing the minimum ambient temperature to 24 degrees C (75degrees F)(8)

Megabacteria or Avian Gastric Yeast (AGY) are novel ascomycetous yeasts that inhabit the isthmus connecting the proventriculus to the ventriculus.(29) They live on the surface of the isthmic glands but in large numbers seem to invade between the glands, invade the transitional koilin of the isthmus and the koilin of the ventriculus. AGY have been diagnosed in many passerine species including Gouldian finches, either alone or in conjunction with other pathogens.(8,30,31). Their presence is associated with increased gastric pH and abnormal digestion. It is uncertain whether they are primary invaders which caused increased gastric pH and maldigestion, or whether some other factor causes increased gastric pH and hence a favourable environment in which the organism can proliferate.(14) Affected birds exhibit depression, pass undigested seed in the droppings, become progressively emaciated and debilitated, polyphagic, regurgitate and may become anaemic. Organisms are often, but not always, shed in the faeces

where they can be identified on faecal smears either directly or via Gram's stain as large cigar-shaped, unevenly stained, gram positive rods, much larger than any other bacteria.(29-31) The organism cannot be consistently cultured at this stage. In older birds a chronic wasting syndrome is seen whereas in less immunologically competent individuals death can be rapid, especially if copathogens exist. Some birds shed the organism with no clinical signs of disease (29,30) Necropsy reveals a distended proventriculus, often covered in a white mucus. Evidence of mucosal petechial haemmorhage may be seen. The gizzard mucosa may also be thin. Proventricular impression smears will reveal large numbers of organisms, even when none can be found in the faeces antemortem. Histopathology is characterized by lymphoplasmacytic inflammation of the surrounding mucosa and less commonly with koilin dysplasia and ulceration of the isthmus and ventriculus.(29-31)The only medication that has been moderately successful has been amphotericin given orally at 100-300 mg/kg PO(29-31) for up to 30 days. It appears to be less effective when used to treat flocks in water/food than when given orally to individual birds. More recently fluconazole has shown some promise. Acidifying the drinking water has also been found to help and one trial in Israel using a Lactobacillus suspension sourced from a cockatoo and gavaged to budgies over 2 days led to a decrease in faecal shedding of megabacteria (30). Acidification of the GI tract with dilute HCL (a one molar/L HCl solution is mixed at a rate of 30ml/pint of drinking water)(31) has also been reported but has not met with much success in this author's hands. Any secondary infections need to be treated concurrently. Recovery can be slow as normal gut function returns. Some birds do not respond to treatment. These birds should be euthanised.

Parasitic Diseases

The commonest parasitic problem in Gouldian Finches is respiratory infections with the air sac mite *Sternostoma tracheacolum*.(8,10,11,13-15) This mite has been found in both captive and wild populations(1,3). Mites are found in the trachea, syrinx, lungs and air sacs. Affected birds exhibit wheezing, sneezing, depression and weight loss. Tracheal transillumination may reveal mites as small, dark moving spots in the lumen. Often, an initial diagnosis is based on clinical signs. Secondary bacterial infections often complicate the illness and can prove fatal. Transmission is considered to occur by direct contact or through respiratory secretions. The use of Bengalese finches as foster parents to break the cycle has been mooted, given the foster species' decreased susceptibility to this mite.(11) However, given the other disease problems associated with this practice, other husbandry practices may be indicated. Avoiding housing Gouldians with other susceptible species (eg canaries) and treating birds in quarantine may also be worthwhile. Treatment includes ivermectin or moxidectin administered topically or orally at doses of 200-800 ug/kg), placement of dichlorvos pest strips near affected birds (allow adequate ventilation) and spraying birds with pyrethrin insecticide spray.(8,10,11) It may be worth warning owners that killing the mites may cause complications as the mite may lodge in parts of the airway. In the past, one treatment involved placing the affected bird in a paper bag containing carbaryl powder & shaking the bag containing the poor unsuspecting patient! If it survived the treatment it was usually cured!!

Parasitic nematodes & cestodes are occasionally found to infect this species, but less commonly than in other finches(10,15,32). Ascarids tend to cause weight loss and sudden death and primarily infect the small intestine. *Acuaria* spp. infect the ventricular koilin lining and may result in wasting and the passage of undigested seed in the droppings. Cestode infections tend to cause diarrhea, emaciation, increased appetite and death due to intestinal obstruction. Diagnosis of these parasites is via faecal floatation or direct faecal examination, where the characteristic ova may be detected, but cestode proglottids may be intermittently shed. Anthelmintics such as ivermectin/moxidectins (200-800 ug/kg), benzimidazoles (fenbendazole,[100-227mg/kg] oxfendazole[40-390 mg/kg]),

levamisole (42-78 mg/kg)are most commonly used for the ascarids.(32,33) Praziquantel (Droncit Tapewormer, Bayer Australia Ltd, Pymble, NSW) dosed at 10-22.7 mg/kg is the most effective treatment for cestodes, although benzimidazoles are partially effective.(33) These doses are for direct dosing to the crop which is the most effective method of administration, but impractical in large flock situations. Caution should be used with some of these drugs. Benzimidazoles have been associated with feather abnormalities in moulting pigeons and levamisole has a narrow safety margin. The author does not recommend the higher levamisole dose. (33)Beware of medicating in excessively hot weather or when breeding. Parasite control involves improving aviary hygiene, eliminating moisture from the aviary substrate to inhibit survival/delay embryonation of ova and removal of intermediate hosts such as insects from the aviary environment.

Urinary tract trematodiasis was diagnosed in six birds originating from Hawaii.(8) Multiple fluke profiles were found in the dilated collecting ducts, tubules, ureters and the cloaca. The tubules showed signs of mineralization and necrosis. Signs of bacteraemia and septicaemia were found in several birds. Deaths were not reported following fenbendazole treatments at 50 mg/kg, whereas deaths did occur with praziquantel at 45 mg/kg. The exact identity, method of transmission and availability of intermediate host is unknown but a snail or other intermediate host is usually required.

A variety of protozoan parasites have been found to infect Gouldian Finches. Coccidia, primarily belonging to the Isospora and Eimeria genera, have been found in many passerines, although the exact species found in Gouldian Finches has not been recorded.(8) Birds passing oocysts may not have clinical signs, but concurrent stress may result in disease. Affected birds show lethargy, weight loss, diarrhea, malaena, dehydration. Transmission is via the ingestion of sporulated oocysts, which require warm, moist environments in which to sporulate and become infective.(10,11,32) Toltrazuril at 6mg/kg bodyweight administered in water (32) appears to be the most effective treatment but coccidiostats such as amprolium and sulphonamides have also been used. Amprolium is useful in preventative treatment programs at a dosage of 120mg/L drinking water daily for5-7 days.(32) Minimizing stressors and eliminating moisture from the substrate are important environmental management tools.

Microsporidia , in particular *Encephalitozoon hellem*, have been identified in a two week old nestling which was presented for necropsy following an outbreak of nestling mortalities within a facility. (34) Interestingly, mortalities only occurred in nestlings which were fostered under Bengalese finches, whereas those reared by their parents were unaffected. Gross necropsy lesions revealed a thickened crop wall due to a concurrent *Candida spp*. infection but little else. Histologically, segmental regions of the small intestinal mucosa were markedly thickened by large numbers of densely packed microsporidia, which distended the epithelial cell lumina. The organisms were mainly in cells along the villous apices, but also in the crypts and in other cells in the lamina propria and also in the small intestine lumen. There was very little associated inflammatory response. Electron microscopy and molecular studies defined the organism as *Encephalitozoon hellem.* Due to the small size of the organism and the lack of host response, this organism can be difficult to diagnose, especially if samples are partly autolysed. Microsporidiosis is considered a disease of the immunocompromised patient(35), including youngsters. It is thus often found in concert with other diseases. Treatment in the case described above involved dosing the Bengalese foster parents with Netobimin at 140mg powder mixed in 400ml of drinking water daily for 5 days, afterwhich mortalities ceased. It is believed that netobomin is metabolized to albendazole in the gut which is the effective agent.

Cryptosporidiosis has also been diagnosed in these birds, but unlike the case in mammals and other avian species, lesions occur primarily in the proventriculus and not in the intestinal tract.(36) In addition, finch isolates of cryptosporidia show distinct genetic differences to other known cryptosporidial species, whilst maintaining homology amongst themselves.(37) This suggests a new species. Histological findings include necrosis and hyperplasia of proventricular glandular epithelial cells associated with a myriad of cryptosporidia attached to the cell surface. Clinically, the birds show decreasing body weight, yellowish droppings and the passage of undigested seed in the stool. Cryptosporidiosis has traditionally been linked to underlying immunosuppression(38), so the possibility of concurrent disease especially viral or poor management affecting immune status need to be considered. Treatment of this disease is difficult but azithromycin, roxithromycin and paromomycin have all met with some success in other species.(36)

Flagellated protozoal infections also occur. *Cochlosoma* spp. cause disease in young birds less than 12 weeks of age.(10,13-15, 39, 40) Clinical signs include lethargy, depression and the passing of moist, bulky droppings, leading to dehydration and death. Sometimes, undigested seed can be seen in the droppings. One study identified the parasite as *Cochlosoma anatis*-like(40). High mortalities occur among nestlings, whereas adult birds are usually inapparently infected, although their droppings may be bulkier and moister than normal. Gross necropsy usually only reveals dehydration. Histologically, the organisms are consistently found along the rectal & cloacal mucosa(39-40). Again, Gouldian chicks seem to be mostly affected when fostered under Bengalese finches, which act as carriers. Transmission is thought to be acquired orally by direct feeding. Ante mortem diagnosis is via the examination of wet mounts of fresh faecal material which reveal the presence of numerous motile flagellates, each with 6 anterior flagella and a helicoidal, anterior ventral sucker. Effective treatments in adult carrier birds include metronidazole (30mg/kg by crop gavage once(40), carnidazole (0.5mg/15 gm bodyweight, repeated 2 and 4 weeks later)(15) and ronidazole in water at 60mg/L for 7 days (40)but only carnidazole has been confirmed in juveniles at a dose of 0.25mg per 7gm bodyweight.(15) Environmental control is by implementing strict hygiene practices. The organism can survive in damp areas around water but is sensitive to dessication and most disinfectants. Removing moisture from the environment and regular cleaning of water dishes is important. Prevention is also based on prophylactically treating or faecal testing all new birds brought into the aviary, particularly inapparent carriers such as Bengalese and Parrot finches (*Erythrura spp.)*(39-40).

Trichomoniasis is only rarely diagnosed in Gouldian finches. Infected birds may exhibit gagging, regurgitation, respiratory distress, nasal discharge, diarrhoea and weight loss(8,10). This organism has also been associated with sinusitis, oesophagitis and encephalitis in Gouldian finches(41). It is usually identified in warm wet mounts of fresh crop contents where it is seen as a circular to oval, motile, tumbling organism which propels itself in a jerky fashion with its four anterior flagella and an undulating membrane which creates a wave-like appearance along the cell surface. Transmission is by direct oral contact or through the drinking water. Dimetridazole, carnidazole, ronidazole and metronidazole are some of the treatments found to be successful.

External parasites can also be found in this species. The birds are susceptible to lice and a range of mites(8,10,11,15). Infections with *Knemidocoptes* sp. may be seen as hyperkeratotic lesions of the feet & beak base. Treatments with pyrethrin- based or ivermectin or moxidectin products are usually successful. Eliminating immunosuppressive stressors is also important. Control of arthropods off the host may be achieved with the use of a variety of surface sprays suitable for birds.

Non-Infectious Conditions

Amyloidosis is not uncommonly encountered in Gouldian Finches. (8,10) Clinical signs may range from respiratory distress and stertor due to liver enlargement, abdominal distention, chronic ill-thrift to sudden death. Usually mature birds are affected. Gross necropsy findings may include a swollen, firm, pale to bronze liver, often pushing onto the air sacs and viscera. Amyloid deposits may also be seen in other organs such as the kidney and spleen. Occasionally no lesions are apparent grossly but amyloid deposits can be confirmed histologically. Amyloid deposition appears to be the result of a chronic disease process resulting in the formation of degradation products (amyloid A) from acute phase, reactant proteins. It is usually the result of chronic infections, especially viral and bacterial causes. One survey showed that in 12 of 15 affected birds, concurrent bacterial infections were diagnosed(8). In some circumstances the causative infection can no longer be identified. Where multiple cases are diagnosed in one collection over a period of time, it would be prudent to investigate the health status of the flock more thoroughly, particularly in birds beginning to show earlier stages of liver enlargement. Unfortunately, the lesions appear to be irreversible and no successful treatment is known.

“Egg binding” and dystocia are also encountered in this species during the breeding season. They usually result from birds which are trying to lay eggs under non ideal conditions. Causes are multifactorial(42). Patient factors include age (too young or old), oviduct damage or deficiencies, malformed eggs, obesity and lack of exercise and allowing hens to lay too many clutches. Environmental factors include sudden cold weather, breeding hens out of season, various nutritional deficiencies (calcium and/or vitamin D deficiency; selenium/vitamin E deficiency); other concurrent stressors eg disease, excess disturbance by other birds or vermin etc. Affected birds are seen fluffed, often on the cage floor, reluctant to perch, weak, swollen vent, laboured respiration, persistant tail wagging or paresis of the legs. These birds often present as medical emergencies. The shock needs to be reversed. Sometimes the bird just needs to be kept in a heated cage of 32-35°C. Dehydration may be reversed by giving warmed fluids either orally, subcutaneously or intramuscularly and the administration of calcium gluconate by intramuscular injection. Often the birds are so weak that any attempt to inject, gavage or excessively handle them results in their death. In these cases, placing the bird in the heated cage with a shallow water dish for humidity may be the best first step. If the bird appears to be stabilized then further hands-on treatment can be instituted at the veterinarian’s discretion.

A host of other illnesses have been diagnosed in this species sporadically. Goitre, suspected vitamin D toxicosis (calcification of renal and other tissues); vitamin D deficiency (rickets); yolk peritonitis; head trauma and retinal atrophy have all been reported.(8,10) One case of suspected vitamin E deficiency was seen in a bird suffering from acute dyspnoea, pulmonary and cerebellar oedema and severe muscular degeneration. Various neoplasms including lymphosarcoma have also been seen.(8)

Other Comments regarding Gouldian Finch Medicine

The Gouldian Finch appears to be an avian enigma. On the one hand it can be an extremely prolific avian species. Testament to this is the large numbers kept and bred throughout the world, and the myriad of colour mutations (at least 6480 genotypes in Europe alone)(4) that have been produced and its high degree of domestication. On the other hand, its numbers in the wild are dwindling(1) and it appears to be highly susceptible to diseases which are commonly linked to immunosuppression. Diseases such as mycobacteriosis, cryptosporidiosis, candidiasis, microsporidiosis are all examples of this. In addition, many necropsy findings reveal the presence

of more than one pathologic agent. It may be that the immune system of this species is not as efficient as it might otherwise be. This may be a genetic factor, which has been exacerbated by the breeding of colour mutations and the resultant deleterious effects of inbreeding. Certainly some lines of colour mutations seem to be more susceptible. For example, some strains of the blue-backed mutation in Australia. On the other hand, it may merely be a reflection that this is species is constantly under stress as a result of the way in which it is commonly kept in captivity. Certainly its love of heat has been well documented and the environments found in most countries of the world where this species is kept do not match that of its habitat of origin. Housing the birds in environmentally-controlled situations certainly seems to help, and is practiced by some. However, many breeders to not want the stigma of producing "soft" birds, which won't adapt as well to the normal temperatures found in their households or aviary environments. Further investigations into the strength of this species' immune response compared to other passerines would be worthwhile.

Whenever dealing with these birds, it is even more important to emphasize the need to decrease the number of stressors in their environment, and optimize their housing, feeding and aviary hygiene. Understanding the bird and how it has evolved are essential for success with this species. For example their need for warmth, their need for social interaction with conspecifics and their susceptibility during the moulting period are all important factors.

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