

Canary flocks infected with *Salmonella typhimurium*

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Introduction

Of the more than 2000 serotypes of Salmonellae which are probably capable of causing disease in birds and other animals *S. typhimurium* probably accounts for approximately half of the outbreaks of salmonellosis in poultry and other bird species. It is a common pathogen of pigeons and free-living birds. Birds, rodents, pets, and humans can act as carriers of *S. typhimurium* and due to its importance in public health, disease outbreaks due to this organism require a thorough investigation. The purpose of the present report is to describe the clinical and pathological findings in two canary (*Serinus canaria*) flocks experiencing mortalities due to infections with *S. typhimurium*.

Flock 1

Flock 1 was a group of approximately 120 birds that had been shipped by air to a pet shop in Western Australia and had experienced high mortalities. A prevalence of 5-10 deaths per day was reported, deaths began approximately 24 hours after transportation. The main clinical feature of affected birds was rapid onset of depression and a fluffed-plumage appearance. Most deaths occurred within 12 hours, although some birds survived for 2 to 3 days. Abnormalities detected at necropsy in acutely affected canaries were similar. In such cases the birds were in good muscle condition. The most consistent finding was a moderate to marked enlarged reddish-purple spleen and congested intestinal tract. Livers from acutely affected birds were either normal or were mild to moderately swollen and slightly purple. In chronically affected birds there was splenomegaly and the liver was firm and diffusely slightly tanned. Liver and spleen routinely inoculated onto blood agar plates and incubated aerobically at 37°C yielded heavy growths of *S. typhimurium*.

Histological lesions of varying severity were present in the liver, spleen, kidney and heart. In the spleen there was a diffuse infiltrate of macrophages and depletion of lymphocytes. A multifocal nodular and perivascular infiltration of proliferating reticuloendothelial cells forming typhoid nodules accompanied in some areas by scattered lymphocytes and heterophils were present throughout the liver.

Flock 2

Flock 2 was a private aviary flock of canaries, finches and quail. The main presenting complaint was lack of breeding success due to high nestling mortality. In the previous breeding year six breeding pairs had bred successfully to expand the flock to 60 birds, 20 of which were sold to a pet shop. At presentation, mortalities had reduced the flock to 17 birds. There had been no introduction of new canaries or other bird species in the year before presentation. Breeding pairs were observed to eject dead and weak nestlings from nests. Deaths also occurred in the few fledglings that were produced and only two fledglings had survived to maturity. Affected fledgling and adult canaries were observed to rapidly become depressed with fluffed plumage and spent an increased amount of time at or near water containers. The droppings had excess urine and the faeces were either normal in colour and consistency or slightly watery. Deaths occurred within six to 12 hours of the first clinical signs.

Approximately 14 finches, including cordon bleu (*Uraeginthus bengalus*) and orangebreasted waxbills (*Sporaeginthus subflavus*) and four Japanese quail were also originally present in the aviary. Breeding success or deaths were not accurately recorded for the finches. However, deaths had reduced the number of finches and no pairs had successfully raised a clutch to fledging. Deaths or breeding activity had not occurred in the quail

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but they had been observed to eat dead canary nestlings ejected from nests.

At presentation deaths were continuing to occur despite seven days of in-water oxytetracycline therapy². Two dead and two moribund birds in good pectoral muscle condition were submitted for necropsy which revealed discoloured reddish-purple, moderate to marked enlarged splenomegaly and congested intestinal tracts. Livers appeared normal size but were also slightly discoloured reddish-purple. Liver and swabs of intestinal contents were aseptically collected for bacteriology. The cloaca of one hen appeared to be diffusely slightly pale and thickened. Direct microscopic examination of intestinal and cloacal content wet-preparations were normal. Impression smears of liver and spleen were also collected. The visceral organs were fixed in buffered formalin for histopathological examination.

Microscopic examination of Wright's and Gram-stained impression smears demonstrated abundant numbers of Gram-negative bacilli free and within macrophages and a heterophilic inflammatory cell infiltrate. Liver and intestinal contents routinely inoculated onto blood agar plates and incubated at 37°C yielded heavy growths of *S. typhimurium* after 24 hours. The isolate was sensitive to neomycin, gentamycin, ampicillin, norfloxacin, amoxicillin-clavulanic acid, cephelexin, trimethoprim-sulphur, tetracycline and nitrofurantoin.

Histopathological examination demonstrated severe acute to chronic splenitis and hepatitis in all birds examined and a severe necrotising heterophilic cloacitis in one hen.

Discussion

This report describes the clinical and pathological findings present in two flocks of canaries which experienced high mortalities due to *S. typhimurium* infection. The history, clinical signs and lesions described here for birds in Flocks 1 and 2 were consistent with a diagnosis of bacterial infection. The differential diagnoses for such lesions in canaries and other small passerine birds should include other bacterial infections such as *E. coli* and other Enterobacteriaceae. For many such cases with similar histories and clinical presentations antibiotics are instituted by aviculturists, pet shop owners or veterinarians either before or without establishing a definitive diagnosis. However, due to its importance in public health, a diagnosis of Salmonellae infection should be ruled out in flocks of canaries and finches experiencing similar signs described here. Indeed, it was later learnt that a worker at the pet shop had developed diarrhoea and was diagnosed with suspected salmonellosis recovering uneventfully with medical treatment. Unfortunately, the diagnosis was not confirmed but it is possible that canaries were a source of infection.

Despite being probably a relatively common disease in passerine aviary birds there seems to be few reports describing lesions of Salmonellae infections in canaries. Arthritis and osteomyelitis due to *S. heidelberg* infection (Panigrahy & Gilmore 1983) and subcutaneous granulomas due to *S. saint paul* infection (Reinhard et al., 1988) have been reported in canaries. In other aviary-bird species, acute disease and lesions similar to those described here have been reported for *S. typhimurium* infection in zebra finches (*Poephila guttata*) which were shown to excrete the organism for at least 22 days after experimental infection (Sato & Aoyagi 1996). The lesions induced included necrotic foci in the liver (Sato and Aoyagi 1996). Strains of *S. typhimurium* originating from finches can be infective and pathogenic to other bird species (Sato and Aoyagi 1996; Sato et al., 1996).

The finding of severe necrotising cloacitis in one hen canary from the second flock indicates that the strain of *S. typhimurium* isolated may have a predilection for this part of the alimentary tract and that vertical transmission, as occurs in chickens, may have been responsible for the nestling mortalities. In chickens oral, intravaginal or intra-cloacal inoculation of *S. enteritidis* results in colonisation of the cloaca and oviduct and transmission occurs by eggshell contamination either as a consequence of descending or ascending infections from colonised ovarian or oviduct tissues or colonised vaginal and cloacal tissues, respectively (Miyamoto et al., 1997; Keller et al., 1995). Male birds can shed Salmonellae in semen and contaminated semen can transmit Salmonellae to susceptible hens (Reiber et al., 1995a,b). Virgin birds may be more susceptible to Salmonellae colonisation than previously mated hens which indicates that mating activity may offer some form of protection against Salmonellae colonisation in the reproductive tract (Reiber & Conner, 1995).

In chicks hatching from contaminated eggs and horizontally infected chicks, Salmonellae colonises the alimentary tract during the first few days or weeks of life and susceptibility to infection decreases with age. Colonisation

²Tricon® Apex, Laboratories.

of the alimentary tract can be rapid and in contaminated hatcheries, *S. typhimurium* can reach the gut of chicks hatching from Salmonella-free eggs before they are removed from the hatcher (Cason et al., 1994). Once infection is established, Salmonellae are probably shed intermittently in the faeces for the life of the bird.

The stresses involved with transportation probably played some role in the disease outbreak in Flock 1. Prevention of similar outbreaks would probably be very difficult because the absence of clinical signs of ill-health may not correlate with shedding of Salmonellae by carrier birds and the lack of a history of stress in Flock 2 indicates that there may be some degree of unpredictability as to when outbreaks occur. Although, minor outbreaks associated with various stresses may occur more frequently without being diagnosed.

Probably the best method of eradicating Salmonellae from infected flocks such as Flock 2 is complete de-stocking and disinfection. However, many aviculturists are reluctant to do this and besides there is a risk of reintroducing Salmonellae-infected birds from other sources. In an outbreak situation the use of appropriate antibiotic medication will probably not eradicate carriers of infection. However, appropriate management revision such as improved hygiene and disinfection procedures and cessation of breeding activity may minimise ongoing losses. Prophylactic competitive exclusion using nonpathogenic enteric bacteria such as *Lactobacillus* can increase the resistance of birds to colonisation by Salmonellae (Watkins & Miller 1983) and for reasons described above is probably best instituted when eggs are hatching. Vaccines for *S. typhimurium* are available for use mainly in pigeons. However, the preventive use of salmonella vaccines in pigeons and probably other bird species cannot be justified because, although they may reduce the severity of clinical signs, probably none can offer full protection from all strains present in nature (Uytendaele et al., 1991).

For aviary flocks of passerine birds it is also noteworthy that mealworms (*Alphitobius diaperinus*) can act as reservoirs of *S. typhimurium* infection and can excrete the bacteria in their faeces for at least 28 days after having fed on Salmonellae contaminated feed (McAllister et al., 1994). Transstadial transmission probably also occurs. Ingestion of just one infected mealworm adult or larva can be enough to cause enteric infection in 1-day-old chicks (McAllister et al., 1994).

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