

## **Salmonellosis in Birds in New Zealand**

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Australia and New Zealand both battled long and hard with pullorum disease for more than 20 years during the 1940's and 50's before finally eradicating it from commercial flocks in the 1960's using a test and slaughter policy. About this time the first cases of Salmonella Typhimurium disease were seen in birds and animals and it was not long before this became the most prevalent serovar in New Zealand (Salisbury, 1957). Since then a variety of salmonella serovars have been isolated from birds including *S. Enteriditis*, *S. Hadar*, *S. Havana*, *S. Agona*, *S. Anatum*, *S. Derby*, *S. Heidelberg*, *S. Infantis*, *S. London*, *S. Mbandaka* and *S. Sentfenberg* (Clark *et al*, 2002), but none of these have been responsible for major epidemics in birds or other animal species this country.

The situation in the northern hemisphere has been a little different with *S.Typhimurium* accounting for most isolations from poultry and other birds in the 1960–70's followed by *S. Enteriditis* which emerged as the commonest isolate in poultry in the last 10-15 years. There have also been reports of many ill and dead finches and sparrows found associated with winter feeding stations throughout Scotland and England during the 1990's (Pennycott *et al*, 1998). In these cases several different phage types of *S.Typhimurium* and occasionally *E. coli* O86 were isolated from the affected birds (Pennycott *et al*, 2002). Similar deaths associated with garden feeders have been reported in the United States and Canada since 1988, mostly involving pine siskins (*Carduelis pinus*), and common redpolls (*Carduelis flammea*) and less often evening grosbeaks (*Coccothraustes vespertinus*), house sparrows (*Passer domesticus*) and American goldfinches (*Carduelis tristis*). The main phage type of salmonella involved in these cases was *S.Typhimurium DT40* (Mikaelian *et al*, 1997, Pennycott, 2001).

### **The Salmonella Typhimurium DT160 outbreak in New Zealand**

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The first isolation of Salmonella Typhimurium DT160 was from a human case of salmonellosis that appeared in Christchurch in November 1998. Further human cases occurred in the same region in November 1999 and by December 2000 the organism had spread to people throughout the country requiring a 20% hospitalisation rate. By 2001, infection with *S.Typhimurium DT 160* had increased to account for 34% of all cases of human salmonellosis.

Mass mortalities of several hundreds of birds were first observed in rural areas around Christchurch in June 2000 and in the Manawatu region in August (Alley *et al*, 2002). These mortalities were often associated with grain silos where there was spilled grain or they occurred in suburban areas around garden feeding stations. In addition to sparrows, lesser numbers of greenfinches, goldfinches, chaffinches and occasionally blackbirds were affected. A small but significant mortality occurred in silvereyes (*Zosterops lateralis*) particularly around feeding stations in Christchurch. The only mortalities confirmed in psittacine birds (kaka, *Nestor meridionalis* and kakariki, *Cyanoramphus novaezelandiae novaezelandiae*) were in captive situations in the North Island where the birds had contact with sparrows or their droppings. Two captive cockatoos died in Christchurch after eating dead or dying sparrows that had entered their aviary.

Affected birds were usually in good body condition indicating an acute disease. If they were found alive they exhibited lethargy, reluctance to fly and often appeared ‘dazed’ and sometimes ataxic making them easy prey for cats which regularly left them on the doorstep or brought them into the house. The most consistent lesion observed was a multifocal hepatitis and acute necrotising splenitis with encephalitis present in more than half the cases. Pale necrotic plaques and superficial ulcerative lesions were seen in the crop in about 40% of cases suggesting that this may be the site of entry of the organisms but this could not be proven experimentally when typical septicaemic lesions were produced 9 days after inoculation with  $10^3$  cfu of organisms. Excretion began within 24 hrs of inoculation and continued until euthanasia at 10 days post inoculation (Connolly *et al*, 2006). Fortunately, the prevalence of this disease in both birds and mammals has reduced markedly in subsequent years but cases of DT 160 are still seen in birds every year and this organism currently comprises about 10 – 20% of human cases of salmonellosis.

### **A Salmonella outbreak in an Endangered Species**

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Hihi or stitchbirds (*Notiomystis cinta*) are currently managed intensively on some offshore and mainland islands in New Zealand since they declined to near extinction following European colonization. They are maintained by supplementary feeding, nest box provision and parasite control and subject to regular health screenings prior to translocation. During February this year, six freshly dead birds were discovered in close proximity to each other having died suddenly in good body condition. Histopathological examination showed acute septicaemic lesions typical of *S.Typhimurium* infection and microbiology revealed heavy growths of a new strain; DT195 from all the affected birds. Although in total only 9 bodies were found, it was estimated that the mortality rate may have been as high as 30% leaving insufficient birds available for planned translocations in the coming year.

As with *S.Typhimurium* DT 160, the DNA type of the new DT 195 was identical to that of a human strain of the same organism isolated from a person with salmonellosis in the upper North Island shortly before the outbreak commenced. Since the island is regularly visited by groups of tourists it seems highly likely that this outbreak in hihi resulted from human contamination of the island environment. Volunteers from overseas had been helping with bird management on the island but those volunteers who were tested subsequently were negative for salmonella carriage.

### **Conclusion**

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Like many infectious diseases, salmonellosis in birds is constantly changing and evolving new infection patterns. The introduction of new strains and opportunities for infection have often been provided by humans involved in the intensive management of both captive and free-living avian species. This may be difficult for the birds but it is likely to provide work for microbiologists and pathologists for many years to come.

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