

An outbreak of Salmonellosis in an Ostrich chick rearing facility

Brett Gartrell, BVSc(Hons) MACVSc(Avian Health)
School of Zoology, University of Tasmania

Introduction

This is a case report of the diagnosis and management of an outbreak of Salmonellosis on a commercial ostrich farm. The emphasis of the report is on the characteristics of the bacteria. *Salmonella* species are capable of infecting all vertebrate species and thus have importance to all aspects of avian practice as well as public health.

Farm Overview

The farm was set up with the purpose of incubating eggs and raising chicks for a number of small ostrich farmers in the district. Other farm operations included a single large paddock for sub-adults and twenty pairs of adult ostriches in half-acre paddocks. The incubator and hatchery were in one building though in separate rooms. One managed the chicks and the incubator and hatchery. This person always monitored the eggs prior to any chick care.

There were three separate chick sheds with runs in a poorly drained and muddy area. Chicks were locked up at night and put into the yards each morning for the day.

Recent farm history

For the previous years of 1995 and 1996 there had been greater than 60% chick mortality which a local veterinarian had diagnosed through post-mortems and histopathology as possible "Ostrich fading syndrome".^{5,17} In 1997 I was asked to give a second opinion on the problem. Approximately ten post mortems, with associated histopathology and bacterial culture and sensitivity, were carried out. This revealed a variety of disease syndromes including enteritis and hepatitis, which cultured *Escherichia coli* of varying antibiotic susceptibility and degenerative myopathy with histological signs of Vitamin E deficiency.

The chick mortality reduced to less than five percent in 1997 following changes to the husbandry and nutrition of chicks.

Recent changes to chick management

Lucerne chaff was removed from the chicks' early diet. The basal diet was changed to higher quality pelleted diet.

Chicks were begun on a batch in, batch out system^{5,17} and general biosecurity was tightened. Routine disinfection of chick sheds was begun.

All chick runs were freshly turfed in an attempt to reduce the mud in the chick runs.

Case history

This particular problem began when two, three-week old ostrich chicks were found dead after a twenty-four hour period of general depression and lethargy.

On gross post mortem a miliary nodular hepatitis and splenitis and a diffuse enteritis was evident in both chicks. The hepatic and splenic nodules were white to cream in colour and ranged from 0.5-3.0mm in diameter.

Cultures revealed a strong growth of *Salmonella typhimurium*.

Salmonella characteristics

There are approximately 2000 species of Salmonella divided into 5 subgenera. Most vertebrates can be infected or colonised by Salmonella, although host susceptibility varies and some strains are host adapted.^{3,6,13,14,15}

There is a spectrum of pathogenicity of Salmonella strains. This spectrum ranges from low virulence strains, which are only likely to cause gastro-intestinal colonisation unless the mucosal barrier is damaged, to high virulence strains, which actively penetrate the gastrointestinal mucosa and can cause a primary septicaemia.^{6,12,16}

The common sequela of infection is a carrier state. i.e. subclinical gastrointestinal colonisation, with the possibility of recurring septicaemia in times of stress or disease.^{6,16}

Propagation can occur outside the host in the right environmental conditions and Salmonella can persist for up to 21 months in dry faeces and feather dust.^{2,3,6,8,9}

Multiple antibiotic resistance and the existence of L-forms are common.^{6,16}

Transmission

The main route of transmission is oral ingestion but respiratory inhalation of contaminated faecal and feather dust can also cause infection.^{2,4,6,16}

Egg transmission is possible and can experimentally occur with as few as 10 organisms.^{6,9}

Vectors of Salmonella include people, rodents, wild birds and insects.^{3,4} Studies in poultry sheds have shown the organism to spread through a naïve shed in as little as a week and become endemic.^{2,4,7,8,9,11,16}

Pathogenesis

Like all Enterobacteriaceae, Salmonella can produce endotoxins but disease is more often associated with direct infection. Even non-pathogenic strains can cause disease if the gut mucosa is disrupted¹². Septicaemia occurs and the organisms colonise any body tissues encountered. The host usually produces a non-specific purulent inflammation in response to parenchymal infection. In birds, this manifests as the caseous nodules seen on gross post mortems.^{6,16}

Carriers

Chronic infections either result in intestinal colonisation and intermittent shedding without clinical signs or the birds can have intermittent septicaemia and clinical signs. These birds suffer progressive organ involvement in the infection and usually the end result is chronic CNS and joint infections.^{6,13,14,15,16}

Carriers can be created by antibiotic treatment, which creates a dilemma of whether or not to treat. Most authors recommend treatment in companion animals based on public health grounds⁶. However, a recommendation for treatment in food producing animals is less certain.

People can be carriers of Salmonella and serve as reservoirs of infection.^{3,6}

Treatment

All chicks on this farm were treated with Tribissen (trimethoprim and sulfadiazine) water medication in drinking water at a dose rate of 5mls per litre for 7 days.

Three weekly faecal cultures from each chick batch were carried out following treatment. All batches had positive results at some time in post treatment sampling.

Culling of the culture positive birds was advised but this advice was ignored. A second course of antibiotic treatment was carried out. The farm refused further bacterial culture.

Prevention and control

An autogenous vaccine was prepared by a commercial laboratory from cultured organisms and given to all birds on the property twice at a one-month interval. Vaccination in poultry has been shown to reduce the incidence of carrier infections and also to reduce the number of Salmonella organisms shed by carriers.^{1,10,11,15} Autogenous vaccines are recommended over generic Salmonella vaccines.^{1,10} Vaccination does not provide reliable prevention of infection or aid birds suffering from the septicaemic disease.^{1,10}

Disinfection of all shed flooring with glutaraldehydes was commenced at weekly intervals.

All new eggs introduced were wiped with a halogenated tertiary amine disinfectant. It was recommended that no new eggs or birds be brought onto the property for a month but again this advice was refused.

Rodent control measures were introduced.

Sources of infection?

The turf laid in chick runs was underlain and fertilised with chicken manure. This is the most likely source of infection as the outbreak occurred in the two weeks following turving and occurred in chicks in this area.

However, transmission via imported eggs into the incubator and hatchery is possible but cultures of these rooms was consistently negative for Salmonella.

The possibility of a rodent and wild bird reservoir is also possible, as is the possibility of carrier adults on property. Despite numerous bacterial cultures in the preceding three years, there had been no previous positive cultures of Salmonella in either chicks or adults.

Mistakes made

Eggs were still brought in during treatment and control period increasing the risk of treatment failure if off-farm birds had been the source of infection.

Follow-up cultures were not made following the second round of treatment. Culture positive birds should have been culled and disposed of to prevent further spread on the farm.

The public health risks were ignored in this case. Staff were not made aware of the disease outbreak or the risks to themselves, contrary to my advice.

There was no notification of the relevant government authorities, who would have had better power to ensure both staff and public were not put at risk. In this case the farm went broke in the following year. I remain uncertain as to the ultimate fate of the chicks, some of which were likely to be carriers of a pathogenic strain of Salmonella.

References

1. Arora AK, Sandhu KS, Sodhi SS. 1998. *Comparative studies on efficacy of different vaccines against fowl typhoid in chickens*. Indian Journal of Animal Sciences. 68(4):297-299
2. Byrd JA, Corrier DE, Deloach JR, Nisbet DJ, Stanker LH. 1998. *Horizontal transmission of Salmonella typhimurium in broiler chicks* Journal of Applied Poultry Research. 7(1):75-80
3. Clark RM, Geldreich EE, Fox KR, Rice EW, Johnson CH, Goodrich JA, Barnick JA, Abdesaken F. 1996. *Tracking a Salmonella serovar typhimurium outbreak in Gideon, Missouri - role of contaminant propagation modelling*. Aqua (Oxford). 45(4):171-183
4. Davies RH, Wray C. 1996. *Persistence of Salmonella enteritidis in poultry units and poultry food*. British Poultry Science. 37(3):589-596
5. Doneley RJ 1997. *Ostrich paediatrics*. Association of Avian Veterinarians - Australian Committee, Annual Conference Proceedings 1997, Perth WA. 1-20.
6. Gerlach H. 1994. *Bacteria in Avian Medicine: Principles and Applications* eds. Ritchie B.W., Harrison G.J. and Harrison L.R. Wingers Publishing, Lake Worth, Florida.
7. Hinz KH, Legutko P, Schroeter A, Lehmacher W, Hartung M. 1996. *Prevalence of motile Salmonella in egg-laying hens at the end of the laying period*. Zentralblatt Fur Veterinarmedizin - Reihe B. 43(1):23-33
8. Holt PS, Mitchell BW, Gast RK. 1998. *Airborne horizontal transmission of Salmonella enteritidis in molted laying chickens*. Avian Diseases. 42(1):45-52

9. Leach SA, Williams A, Davies AC, Wilson J, Marsh PD, Humphrey TJ. 1999. *Aerosol route enhances the contamination of intact eggs and muscle of experimentally infected laying hens by Salmonella typhimurium*. FEMS Microbiology Letters. 171(2):203-207
10. Methner U, Barrow PA, Martin G, Meyer H. 1997. *Comparative study of the protective effect against Salmonella colonisation in newly hatched spf chickens using live, attenuated Salmonella vaccine strains, wild-type Salmonella strains or a competitive exclusion product*. International Journal of Food Microbiology. 35(3):223-230
11. Methner U, Koch H, Meyer H. 1995. *Model for testing efficacy of control measures against Salmonella infections in poultry*. Deutsche Tierärztliche Wochenschrift. 102(6):225-228
12. Qin ZR, Fukata T, Baba E, Arakawa A. 1995. *Effect of Eimeria tenella infection on Salmonella enteritidis infection in chickens*. Poultry Science. 74(1):1-7
13. Raidal SR 1998. *Salmonellosis in two canary (Serinus canaria) flocks*. Australian Veterinary Practitioner. 28(2):58
14. Sato Y, Aoyagi T. 1996. *Infectivity and persistence of Salmonella typhimurium for zebra finches (Poephila guttata) isolated from the same species*. Journal of Veterinary Medical Science. 58(9):845-848
15. Siegel HS. 1995. *Stress, strains and resistance*. British Poultry Science. 36(1):3-22
16. Suzuki S. 1994. *Pathogenicity of Salmonella enteritidis in poultry*. International Journal of Food Microbiology. 21(1-2):89-105
17. Tully TN, Shane SM. 1996. *Husbandry practices as related to infectious and parasitic diseases of farmed ratites*. Bulletin de l'Office International des Epizooties. 15(1):73-89