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# Epidemiological studies on *Salmonella senftenberg*

## II. Infections in farm animals

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## INTRODUCTION

According to Taylor (1967) and van Oye (1964) the most frequently isolated salmonella serotypes in any one country are characteristic of that country and not liable to extreme changes over short periods of time, although the order will change and some serotypes will disappear and appear in time. Amongst these are the serotypes most capable of animal-to-animal spread, such as *Salmonella typhimurium*, *S. dublin* and *S. choleraesuis*. The less commonly isolated serotypes are more necessarily involved in food cycles with sporadic outbreaks the frequency of which appears to depend on the current level of contamination of animal feed and human foodstuffs. These seldom cause disease on farms (Gibson, 1965) and differ experimentally in their ability to do so (Vestal & Stephens, 1966). This also appears to be true for human disease; Szanton (1957) and Watt *et al.* (1958) have reported infection in infants with *S. oranienberg* and *S. tennessee* without clinical disease, and McCall *et al.* (1966) have reported clinical symptoms to be minimal or absent with other serotypes. The production of clinical symptoms (recognizable 'salmonellosis') is dependent not only on certain characteristics such as age of the individual and dosage but also on intercurrent infection, as seen in hospital outbreaks (Tin Han, Sokal & Neter, 1967, Datta & Pridie, 1960), farm outbreaks (Gibson 1965) or experimental malaria (Kaye, Merselis & Hook, 1965) or coccidiosis (Stephens & Vestal, 1966). Adverse environmental conditions such as cold or lack of feed and water have been shown to affect the clinical disease rate of animals by Previte & Berry (1962) and Bierer & Eleazer (1965), but this was denied by Morrello, Digenio & Baker (1965). The reduction of intercurrent infection, by the use of coccidiostats and tylosin, appears to reduce the attack rate in experimental and field conditions (Bierer, 1961; Ridgway & Ryden, 1966).

Since *S. senftenberg* is so widespread in animal feedstuffs and yet so infrequently isolated from men and animals, it is of some interest to know why it should be isolated at all. While tracing the sources of the animal infections in a series of *S. senftenberg* isolations, reasonably complete histories were obtained from six of the eight farms by visits to the farms and examination of their records maintained by the Veterinary Investigation Service and Veterinary Field Service. Salmonellosis in poultry up to 14 days old is characterized by failure to thrive, inappetence, diarrhoea, and increasing numbers of deaths; after death the carcass has a septicaemic appearance, enlarged congested liver with focal necrosis, some myocarditis and caecal casts.

## FARM INVESTIGATIONS

*Farms B–H*

The disease histories and details of animal feeding on these farms are given in a previous paper (Hobbs & Hugh-Jones, 1969) and the possibility of a feed contamination discussed. In the farms from which *S. senftenberg* had been isolated, no disease was reported from a dairy herd, there was a chronic diarrhoea of unknown cause in a ewe flock, and Gumboro disease, starvation syndrome and omphalitis in each of three poultry flocks. In these instances *S. senftenberg* did not appear to have acted as a primary pathogen, if as a pathogen at all.

*Farm A*

We are fortunate to collect a complete series of post-mortem examination and veterinary field service reports from this farm covering the period from March 1963 to August 1966. All the post-mortem examinations had been done by one laboratory worker.

This farm is the headquarters of a five-farm complex, each farm with a commercial turkey flock; farm A also has a breeding flock in the Poultry Health Scheme, separate from the commercial flock, producing fertile eggs for a hatchery. The commercial farm A flock is about a quarter of a mile from the main buildings and has a separate staff. Originally the farm produced forced spring rhubarb but the market for this product fell and the field buildings were used for raising turkeys. Infection with *S. senftenberg* was limited to the commercial flock at farm A and was not found at any of the other four farms. The birds arrive as day-old poults and are placed in a pair of central brooder houses with a common feed store. The houses are in moderate repair with wooden walls and compacted earth floors; woodshavings are used for litter; both piped and bottled water is available. The roof of the feed stores appeared to be sound. The poults receive company IV 'turkey starter' (Hobbs & Hugh-Jones, 1969) for the first 5 weeks in self-feeders which are supposed to be checked and the faeces removed every day. At 5 weeks old the birds are moved into growing or fattening sheds, and fed on a growing ration of company V white fish meal and grain (soya, maize, barley or oats, vitamin and minerals). They are slaughtered in these sheds when 18–20 weeks old after 24 hr. starvation, but birds from certain hatcheries are returned alive in crates for slaughter elsewhere. Slaughtering starts in mid-April and is continuous until mid-December, when left-over birds are sold locally. Empty houses are cleaned out, sprayed with 'Hydrox' mixed with diesel oil and left empty for no less than 1 week. All the houses are empty over Christmas and the brooder houses are re-stocked in January.

It is only in hindsight that it could be suggested that the presence of *S. senftenberg* contributed to the ill-health of turkey poults on farm A. Frequently the post-mortem picture was of a non-specific nature, typical of 'chilling' and 'not starting'. During 1963 aspergillosis was diagnosed twice in growers and poults. In April 1964 the flock was tested serologically for *S. pullorum* with negative results. *S. senftenberg* was isolated for the first time in August 1964. The initial deaths had

been normal among the 2000 day-old poults but rapidly increased from the tenth to the fourteenth day. Furazolidone treatment was started, the deaths tapered off with a 6% mortality and the birds reared satisfactorily. Aspergillosis was diagnosed on the basis of mycotic abscesses in the lungs and air sacs and *S. senftenberg* was isolated from the swollen livers of all the four dead birds examined.

In January 1965, 2600 day-old poults arrived and by the fifth day 147 birds had died. Post-mortem examination showed that they had not been eating; the lungs and kidneys were congested; the gall bladders distended; the yolk sacs were being absorbed and, besides *Escherichia coli*, *S. senftenberg* was isolated from the yolk sacs. The initial diagnosis was 'delay in starting to eat'. The hatchery was visited by the Veterinary Field Service and found to have 'the highest standards of hygiene and organization'. However, the hatch providing these birds was made up of 2600 eggs from farm E in Scotland and 1484 of the hatchery's own eggs (*S. senftenberg* was isolated from farm E for the first time in April 1965). These eggs were incubated in separate machines and 49% and 68% hatched respectively. Another farm received 480 poults from the second day's hatch and suffered an over-all 43% mortality with forty-six dying in the first 4 days. Unfortunately none of these birds were available for bacteriological examination. It seemed probable that the hatchery might have obtained infected eggs from farm E, but other factors could have been involved.

Late in February 1965, 1350 day-old poults were mixed with 1650 week-old poults from the same hatchery and the birds began to die, although on a furazolidone-supplemented diet. Of the 10 week-old birds examined, one had an incorrectly absorbed yolk sac, and the others congested lungs and kidneys, distended gall bladders and empty gizzards; such lesions are associated with birds 'not starting'. *E. coli* was recovered from the lungs and kidney, but the intestines were not examined bacteriologically. This flock was seen again when 5 weeks old and proved to be unthrifty and unevenly grown. Seven were examined out of the forty-one that had died; the carcasses were rather wet and congested and the gizzards were a little enlarged; five had air-sacculitis, thin-walled intestines, and enlarged and congested kidneys. *S. senftenberg* was isolated from the intestines. It was noted at the time that the respiratory lesions were insufficient to have caused death. The hatchery was visited again but no problems had been reported with the poults from the same hatch on other growing farms.

In March a delivery of 5600 day-old poults from a different hatchery arrived; 3600 went to another farm and remained healthy, while 2000 stayed on farm A. They were cared for by the same poultry man as the February birds and were in the same group of buildings. The poults were severely debeaked on day 12 and 3 days later 287 birds died (see Fig. 1), although furazolidone was added to their food from day 13. The poultryman reported that the birds were found dead with no apparent symptoms. Of the ten 15-day-old poults examined, three showed pale areas on the gizzard muscles, one had small 'brownish' kidneys, one only had food in its crop and their intestines were flaccid; four had swollen livers and inflamed kidneys, and the rest congested kidneys only. *S. senftenberg* was isolated from all three livers examined. When seen by a Field Officer on day 23 there were no ailing

birds in the flocks. It seemed that the severe debeaking of the flock with resultant tissue damage, shock and depriving the poults of food and water, caused an abnormally large number of deaths with post-mortem lesions similar to those of salmonellosis.

In May 3000 day-old poults arrived from the same hatchery as the January and February birds and they were immediately started on a furazolidone-supplemented diet. Deaths occurred as shown in Fig. 1 and at 1 month old the flock was seen to be unthrifty, with birds of different sizes, but with no symptoms noticed by the poultryman other than sudden deaths. A post-mortem examination of birds

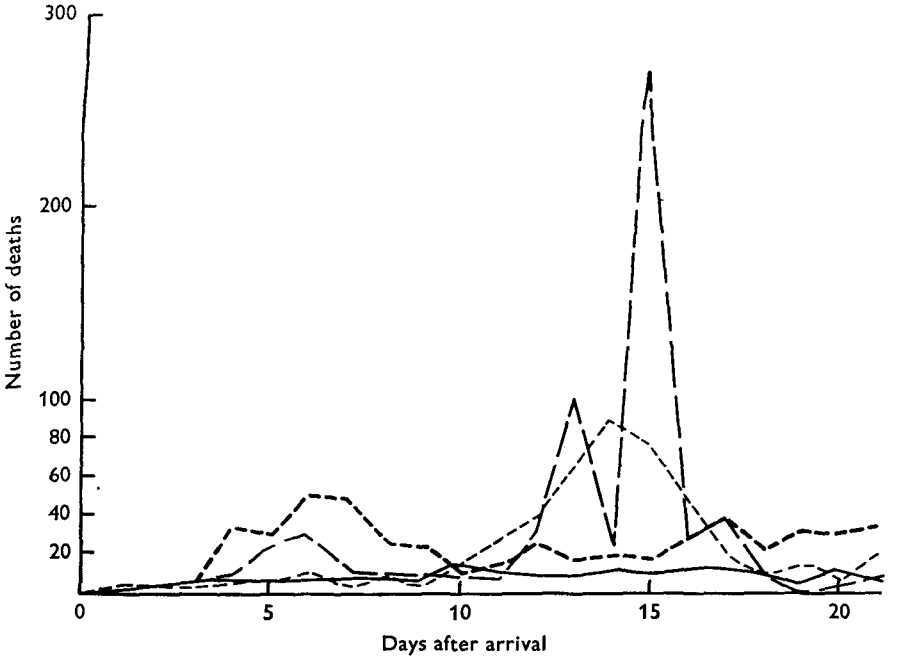


Fig. 1. Deaths in batches of day-old poults at farm A.
—— —— 2000 which arrived 1 March 1965; mortality 30·8 %.
– – – – – 3000 which arrived 10 May 1965; mortality 16·2 %.
– – – – – 2000 which arrived 7 July 1965; mortality 22·5 %.
———— 2828 which arrived 21 July 1965; mortality 5·5 %.

13 days old showed them to have a mild enteritis, diarrhoea, enlarged gall-bladders and congested livers; in birds dying at 21 days there was also congestion of the lung and kidney and diarrhoea; one of the eight birds examined had a curled tongue. *S. senftenberg* was isolated from the intestines of the 13-day-old carcasses but not from those of the 21-day-old birds. The hatchery was visited again and it was observed also that birds from the same hatch sent to other farms had remained healthy. A year later, when the farm was visited, the commercial flock showed chronic respiratory symptoms, and considering the earlier history of this flock, it may be assumed that there had been a constant respiratory problem on this farm.

In June 1965 *S. senftenberg* was isolated again from week-old poults that had 'failed to start', from which time the manager ceased to send carcasses for examina-

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tion, although deaths continued to occur. However, in late July the normal number of deaths occurred in a new batch of poults. When the brooder house was re-stocked after the 1965 Christmas holiday, normal death-rates occurred among the birds, and *S. senftenberg* was not recovered from any dead birds. The known isolations of *S. senftenberg* occurred from August 1964 to June 1965 from birds in both brooder houses, and in August and September 1966 *S. senftenberg* was recovered from the earth floors under the existing litter by the Food Hygiene Laboratory. Unfortunately the source of infection on this farm was not discovered.

## DISCUSSION

All these isolations of *S. senftenberg* suggest that it is usually found by chance as a result of the bacteriological examination of animals with other disease conditions. *S. senftenberg* is an accidental finding. But under circumstances of extreme 'stress', such as debeaking very young turkey poults, it may contribute to the subsequent mortality if not actually produce salmonellosis. It may also aid other secondary pathogens or disease conditions, such as a chronic low-level respiratory problem, or cold brooder conditions, in producing a non-specific but significant number of deaths in poults, but of itself not be pathogenic enough to give such deaths the characteristics of a 'disease'.

## SUMMARY

Between August 1964 and November 1965 *Salmonella senftenberg* was isolated from poultry, sheep and cattle on eight farms in England and Scotland. From an analysis of the case records its presence would appear to be incidental; but it may contribute to poultry mortality by acting in conjunction with other intercurrent infections or following stressful events such as severe debeaking or cold brooder conditions.

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