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The prevalence of salmonellas in mink

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SUMMARY

The mean isolation rates of salmonellas from the mesenteric lymph nodes and faeces of healthy mink were 16·7% of 618 animals from three farms and 3·9% of 772 animals from four farms respectively. *Salmonella senftenberg* was the most commonly isolated serotype. *S. typhimurium*, *S. dublin*, *S. livingstone*, *S. menston*, *S. enteritidis*, *S. bredeney* and *S. infantis* were also seen.

The prevalence of salmonellosis in 316 dead mink from 12 farms was 0·6%.

The epidemiological aspects are discussed.

INTRODUCTION

The wide distribution of salmonellas makes their prevalence in mink of interest not only to the mink farmer but also to other agriculturalists and those concerned with public health. Previous surveys have shown a low incidence varying from 0·6% of 3079 autopsies (Momberg-Jorgensen, 1949) to 1·6% of 1602 German mink (Loliger, 1956). These figures are supported by more general surveys where the number of isolations of salmonellas from different species has been reported (Bigland, Wilton, Vance & Carlson, 1962; Karlsson, Rutqvist & Thal, 1963; Hurvell, Lagerquist, Rutqvist & Thal, 1969). The isolations from mink have been low compared with those from cattle, pigs and poultry. *S. typhimurium* was the most commonly isolated serotype.

Outbreaks of salmonellosis among mink are not often reported. During pregnancy, infection with *S. choleraesuis* var. *kunzendorf* causes abortion, metritis and the occasional death (Hartsough, 1946–7). Secondary salmonellosis in mink dying from distemper has been observed by Zimmerman (1962) and by Head (1959) who, in the same paper, also recorded sudden deaths in weaned kits associated with *S. dublin*. He commented that probably salmonellosis only becomes a problem when mink are stressed. This was demonstrated experimentally by Gorham, Cordy & Quortrup (1949), who found that the oral administration of *S. choleraesuis* var. *kunzendorf*, *S. enteritidis* and *S. newport* had no effect on normal animals. However, when the mink were stressed by starving them before dosing with *S. newport*, an occasional animal died. Despite these observations many mink farmers and their advisers still think that salmonellas commonly cause disease in mink. The present work has attempted to assess this impression by determining the prevalence of salmonellas in healthy mink and the prevalence of salmonellosis

Table 1. *Number of animals examined during survey*

Farm	Period 1 Nov. 1970–Jan 1971	Period 2 March 1971	Period 3 Nov. 1972	Total
A	188	—	5	193
B	203	—	—	203
C	174	25	100	299
D	200	—	100	300
Total	765	25	205	995

in the mink received for pathological examination at the Veterinary Investigation Centre, Leeds.

MATERIAL AND METHODS

Carcass survey

Details of the numbers of healthy mink and when they were examined are given in Table 1. A total of 706 fresh and 289 deep frozen carcasses were examined after pelting in November, December, January and March. Although all animals were clinically healthy before slaughter, one batch on farm C had previously failed the iodine agglutination test and could therefore be expected to be suffering from the early stages of Aleutian disease. The survey included male and female kits (6–7 months old) and adults.

Cultures were made from the stomach contents, rectum, gall bladder, mesenteric and gastro-hepatic lymph nodes, but not all sites were cultured in any one batch of animals. This is made clear in the text and Table 2.

The mink received at the Veterinary Investigation Centre, Leeds, during 1969–72 for pathological examination, comprised 73 1-day-old kits from five farms and 316 fully grown animals (known hereafter as 'dead mink') from twelve farms. All were examined bacteriologically and salmonellosis was diagnosed where the infection was septicaemic or where salmonella was isolated from a diseased tissue.

Feeding

During period 1, farms B and C fed the same constituents from the same sources, i.e. broiler offal, day-old chicks, bovine rumens, fish offal and cereals. During period 2, the ration on farm C included bovine livers and excluded the broiler offal. In period 3, the diet was the same as period 1 but the bovine rumens had been replaced by ovine and the sources of the broiler offal, fish and cereal had changed.

Farm A fed a similar diet to farms B and C but the sources were different.

Farm D's sources also differed from the rest and during period 1 did not include any chicken, i.e. diet consisted of ovine rumens, heads, fish offal and cereals. A change had been made by period 3 and the ration included hen offal.

Table 2. *Isolation of salmonellas from clinically normal mink on four farms*

Batch	Date	Sex and age	No. of carcasses examined	Percentage of isolations from: Mesenteric lymph node	Gall bladder	Stomach contents	Rectal contents	Serotype
Farm A								
1	Nov. 70	M, kit	49	6·1	0	0	0	1
2	Nov. 70	M, adult	16	43·75	0	6·25	6·25	1, 2, 3, 4
3	Nov. 70	F, adult	23	34·8	ND	ND	ND	1, 2, 3, 4, 7
4	Nov. 70	F, adult	50	26·0	2·0	2·0	4·0	2, 4
5	Dec. 70	F, adult	50	0	0	0	6	1
6	Nov. 72	F, kit	5	40·0	0	ND	40·0	4, 8
	Sub-total		193	17·1	0·6	1·2	4·7	
Farm B								
7	Nov. 70	F, kit	50	ND	2	ND	6·0	5
8	Nov. 70	M, kit	50	ND	0	ND	4·0	5
9	Nov. 70	M, adult	33	ND	0	ND	3·0	5
10	Nov. 70	F, adult	70	ND	0	ND	1·4	1
	Sub-total		203	ND	0·5	ND	3·4	
Farm C								
11	Nov. 70	M, kit	74	ND	5·4	ND	13·5	1, 5
12	Dec. 70	F, adult*	40	0	0	15	0	1
13	Dec. 70	F, kit	25	12·0	8·0	16·0	4·0	1
14	Dec. 70	F, adult	35	8·6	0	17·1	2·9	1
15	Mar. 71	M, adult	25	72	32	28	8	3
16	Nov. 72	Both, kit	50	18	2	6	ND	2, 6
17	Nov. 72	M, kit	50	26	ND	ND	ND	2, 6
	Sub-total		299	20·4	6·0	14·9	7·0	
Farm D								
18	Nov. 70	Both, both	100	ND	0	ND	1	3
19	Jan. 71	F, adult	100	1	ND	ND	0	2
30	Nov. 72	Both, both	100	23	ND	1	ND	2
	Sub-total		300	12	0	1	0·5	
	Total		995	16·7	2·4	6·6	3·9	

Abbreviations: F = female; M = male; * = batch which failed the iodine agglutination test.
Numbers in the serotype column refer to the numbers indicating the serotypes in Table 5, e.g. 3 = *S. dublin*.
ND = not done.

Bacteriological examinations

Alginate swabs were inserted into the stomach, rectum and gall-bladder and transferred to 10 ml. of selenite F broth. Lymph nodes could not be cultured this way and instead a portion (0·5 cm.^{3}) was excised from each node and transferred to selenite F broth. In one experiment with twenty-three cadavers, a portion was placed directly in broth, whilst a further portion was homogenized in the broth before incubation. Selenite F broths were incubated at 43° C. for 18–24 hr. and subcultured on brilliant green agar plates. After a further 24 hr. incubation at 37° C., non-lactose fermenting colonies were selected for serological and biochemical identification. Confirmatory serological typing was carried out by the Central Veterinary Laboratory, Weybridge, and phage typing by the Enteric Reference Laboratory, Colindale.

RESULTS

Isolation from different sites in healthy animals

The observed values for each batch, the mean values for each farm and the overall means are given in Table 2. Isolations from the mesenteric lymph nodes were significantly greater than from any other site ($P < 0{\cdot}001$, Table 3), whilst those from the stomach contents were significantly greater than from the gall bladder and rectal contents ($P < 0{\cdot}01$). There was no significant difference between the recovery rates from the gall bladder and rectal contents. These observations were valid overall but not for the individual batch since between-batch variations were considerable, even where consecutive batches came from the same farm (Table 2). The gastro-hepatic lymph nodes were cultured in two batches (Table 3). The isolation rate (3·0%) was significantly lower than from comparable mesenteric lymph nodes (22·0%).

The statistical analyses in Table 3 have grouped all serotypes and assumed that their prevalence in each site would rank the same. This was true for most but not for *S. senftenberg*, where there was no significant difference between the recovery rate from the mesenteric lymph nodes, stomach contents and rectum (Table 4. $\chi^2 = 2{\cdot}6$ for 2 degrees of freedom).

It was found that homogenizing the mesenteric lymph nodes resulted in an increase in the rate of isolation of salmonellas. Eight isolations were made from 23 nodes in the control group and 13 from 23 homogenized glands. However, since only two isolates were duplicated, it was impossible to make any direct comparison of the two methods. In two animals, different serotypes were isolated from the same node; *S. senftenberg* and *S. dublin* from one and *S. senftenberg* and *S. livingstone* from the other. In all, 19/23 nodes (82·6%) were positive.

In the 290 animals where four sites were cultured, the same serotype was isolated on thirteen occasions from more than one site. The numbers were too small to analyse statistically but there was no suggestion of any correlation between infection of the mesenteric lymph nodes and excretion in the faeces. Occasionally, more than one serotype was isolated from the same animal. Two examples of dual infection of the lymph node have been given above. Others were *S. senftenberg* and

Table 3. *A comparison of the isolation rates from different sites*

	Cultured site	No. of positive isolations	Percentage positive	*F*	χ^2	*P*
290 mink (eight batches)	Mesenteric lymph node	47	16·2	3	41·90	< 0·001
	Stomach contents	25	8·6	2	9·76	< 0·01
	Gall bladder	11	3·8	.	.	n.s.
	Rectal contents	10	3·4	.	.	n.s.
100 mink (two batches)	Mesenteric lymph node	22	22	1	13·9	< 0·001
	Gastro-hepatic lymph node	3	3	.	.	.

F = degrees of freedom. n.s. = not significant.

Table 4. *Prevalence of individual serotypes in those batches in which the serotype occurred and where four sites were cultured*

Serotype	No. of batches	No. of animals examined	Percentage of isolations from: Mesenteric lymph node	Gall bladder	Stomach contents	Rectal contents
S. senftenberg	7	265	4·5	0·75	6·0	3·0
S. typhimurium	3	116	7·8	0	1·7	0
S. dublin	2	41	46·3	19·5	17·1	4·9
S. livingstone	2	66	10·6	1·5	1·5	0
S. enteritidis	1	50	18·0	2·0	4·0	0

S. typhimurium from the mesenteric lymph node, *S. typhimurium* from the mesenteric lymph node *S. senftenberg* from the rectum, and in a third, *S. typhimurium* from the mesenteric lymph node and *S. livingstone* from the stomach contents.

At the beginning of the survey it was thought that the isolation rate from the mesenteric lymph nodes might be increased in kits and in animals suffering from early Aleutian disease. In fact, where animals were examined at comparable times, isolations from kits (14·1% of 99) and adults (15·6% of 289) were similar, and it was clear that any such influences were insignificant compared with between-batch variations.

Salmonellas on the individual farm

On farm A during period 1, *S. typhimurium* (phage type 9), *S. senftenberg*, *S. livingstone*, *S. dublin* and *S. bredeney* were isolated, and from a small batch of five mink during period 3, *S. livingstone* and *S. infantis* were found.

During period 1, farms B and C shared the same serotypes, *S. menston* and *S. senftenberg*, and the same feed supplies. Thereafter, only farm C was examined. A single batch during period 2 revealed *S. dublin* and in period 3 *S. typhimurium* (phage type 1) and *S. enteritidis* (phage type 8) were found.

The prevalence on farm D was low during period 1 but high during period 3. *S. typhimurium* (phage type 3a) and *S. dublin* were isolated in period 1 and *S. typhimurium* (phage type 12a) during period 3.

When batches were examined during consecutive weeks (Table 2, e.g. batches 1–5, 7–9, 11–14), it was usual to find the same serotype in each batch; this was not

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Table 5. *Isolation of serotypes from four mink farms*

Serotype	No. of times serotype isolated from 20 batches	Farms A	B	C	D	No. of farms from which serotype was isolated
1. *S. senftenberg*	9	+	+	+	.	3
2. *S. typhimurium**	7	+	+	+	+	4
3. *S. dublin*	4	+	.	+	+	3
4. *S. livingstone*	4	+	.	.	.	1
5. *S. menston*	4	.	+	+	.	2
6. *S. enteritidis*	2	.	.	+	.	1
7. *S. bredeney*	1	+	.	.	.	1
8. *S. infantis*	1	+	.	.	.	1
Number	.	6	3	5	2	.

+ = isolation; * Not always the same phage type.

necessarily true if several months separated the sampling periods. On farm C, *S. senftenberg* and *S. menston* were isolated during period 1, *S. dublin* during period 2 and *S. enteritidis* and *S. typhimurium* during period 3. On the other hand, on farm A, *S. livingstone* was seen 2 years after the first isolation. Eight serotypes were isolated during the survey, *S. senftenberg* being the most common (Table 5). All these serotypes, with the exception of *S. menston*, were isolated from the mesenteric lymph nodes indicating that infection and not passive carriage had occurred.

The overall prevalence of individual serotypes from each site has not been given since a high recovery rate from a batch (e.g. *S. dublin* from batch 15, Table 2) unduly weights the means and this could be misleading. The method used in Table 5 (i.e. the number of serotypes in 20 batches) is considered to be more valid.

Salmonellas as a cause of disease

Salmonellosis was diagnosed in 0·6% of the 316 dead mink. *S. typhimurium* (*phage* type 1a) was septicaemic in a 6-month-old male that had died suddenly on farm B and *S. dublin* in a mink suffering from urolithiasis on farm C. In addition, five isolations (three of *S. typhimurium* (untypable), one of *S. typhimurium* (phage type 29) and one of *S. dublin*) were made after enrichment cultures, but these were not causing disease.

No salmonellas were isolated from the 73 1-day-old kits.

DISCUSSION

Salmonellas are prevalent in mink but clinical salmonellosis is uncommon. These conclusions were expected since mink thrive on a diet of raw offals which often contains salmonellas. The assumption that infection originates mainly from the food is supported by our observations. Farms B and C shared the same feed during period 1 and also the same serotypes, and on farm C when the diet changed so did the serotypes. The contaminated constituents have not yet been identified, but since *S. senftenberg*, *S. livingstone*, *S. menston*, *S. enteritidis*, *S. bredeney* and

S. infantis are all more commonly found in birds than animals (Sojka & Field, 1970), broiler offal and, in the case of *S. menston*, day-old chicks were the probable sources of infection for these serotypes. The flocks from which the day-old chicks came were known to be infected with *S. menston*. *S. dublin* probably originated from ruminant offals, but the origin of the ubiquitous *S. typhimurium* must remain uncertain. The contribution of the cereals and fish is thought to have been insignificant, although Hobbs & Hugh-Jones (1969) suspected that white fish meal was the source of the outbreak of *S. senftenberg* in domestic animals and humans which they investigated. Wild birds might be a further source of infection, but surveys have shown neither the number nor the prevalence of serotypes found in mink (Goodchild & Tucker, 1968).

The survey measured the prevalence of salmonellas in healthy mink on four farms during the autumn and winter. Observations were not made at other times as carcasses were unavailable, but this was not a serious drawback since on all farms the dietary constituents and their sources tend to remain the same throughout the year; it is the proportions which vary. The incidence of *Salmonella* infection therefore depends on the frequency and degree of contamination in feedstuffs; the amounts that are collected; how long they are fed; the proportion of contaminated constituents in the total diet; the invasiveness of the salmonellas and the length of time for which mink remain carriers after infection. The incidence of avian serotypes in mink might be fairly constant since the poultry industry is becoming vertically integrated and individual organizations can often be identified by their salmonellas. Mink receiving poultry offal tend to be regularly challenged by the same serotypes.

On comparing the prevalence in different sites it was found that the recovery rate from the mesenteric lymph nodes was significantly higher than from any other site. This suggests that mink do become carriers but for how long is not known. The absence of correlation between the recovery rate from the mesenteric lymph nodes and the faeces suggests that the excretion rate from carriers is low or non-existent. The increased recovery rate from the mesenteric lymph nodes was true for most serotypes but not for *S. senftenberg*, a serotype which does not appear to be particularly invasive in mink.

The prevalence in the gall bladder and gastro-hepatic lymph nodes was low, whilst the recovery rate from the faeces was significantly lower than from the stomach contents or mesenteric lymph nodes but still high compared with other domestic species. Williams-Smith (1971) has listed the frequency with which salmonellas have been isolated from the faeces of adult healthy domestic animals in Britain. The highest figures of 2·5 and 2·0% were for turkeys and geese respectively but the list did not include mink. The faecal recovery rate from these mink was 3·9%. The reason for the lower recovery rate from the rectum than from the stomach contents is not known.

Comparisons with other surveys are not easy since it is not always clear from accounts which sites were cultured and what methods were used but the prevalence of 0·6% for clinical salmonellosis in the present survey is similar to that quoted by Momberg-Jorgensen (1949). The effect of stress in precipitating salmonellosis

may have been over emphasized. Two common causes of death in mink are chronic Aleutian disease and the stress/starvation syndrome. Prolonged stress occurs in both but secondary salmonellosis is uncommon and other factors may be more important. For example, the association between distemper and secondary salmonellosis (Head, 1959; Zimmerman, 1962) is probably due to the viral leucopenia reducing the resistance of the animal to bacterial infection and we have concluded that, with the exception of *S. choleraesuis* var. *kunzendorf*, the pathogenicity of salmonellas for even young mink is doubtful.

Whether mink are an important epidemiological source of salmonellas for people and livestock is still uncertain, but clearly their carcasses and excreta are potentially dangerous. Mink carcasses are usually disposed of at meat rendering plants and certainly if the hygiene in any of these plants was poor, they could be an important source of contamination for meat meals. The excreta lie in the mink sheds for up to 1 year and are then usually spread on agricultural land. To our knowledge, this has not been associated with subsequent outbreaks of salmonellosis in grazing animals but there would appear to be a potential danger and this will depend on whether salmonellas can survive for long periods in mink faeces. This we are investigating.

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