

An epidemiological study of the incidence of salmonellas in pigs

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(Received 9 August 1971)

SUMMARY

The incidence of salmonellas in pigs was studied in five farms and a bacon factory.

Persistence and spread of salmonella excretion in pigs in a breeding establishment is described. Salmonella excretor boars and sows were responsible for the spread and perpetuation of infection in the farm. The possibility of spreading salmonella infection between farms through the distribution of excretor pigs was studied. Infection persisted and was related to the initial state of excretion of the pigs while at the farm of origin.

The importance of feeding stuffs as a source of salmonella infection in pigs is discussed. Specially prepared heat treated pellets fed to the pigs prevented the introduction of salmonellas.

INTRODUCTION

The wide distribution of salmonellas in nature and their importance as pathogens in animals and men are well established facts. The role of animals as a source of these organisms is recognized (Hobbs, 1961; van Oye, 1964; Greatorex, 1966). Pigs are considered to be an important reservoir (Buxton, 1957). Pig meat and meat products have often been found associated with food poisoning incidents in man (Bevan Jones, Farkas, Ghosh & Hobbs, 1964).

Salmonellosis occurs either as overt clinical infection or as symptomless excretion in man and animals. *Salmonella choleraesuis* and *S. typhimurium* are mainly responsible for clinical disease in pigs but many other serotypes are excreted in the faeces without symptoms, although localization often occurs in lymph nodes of young animals. It has been recognized that symptomless excretion is of potential public health importance although its incidence may not be known by veterinarians.

Earlier surveys included examination of pooled mesenteric lymph nodes from normal pigs at abattoirs (Hormaeche & Salsamendi, 1936, 1939; Scott, 1940; Medical Research Council, 1947). Smith & Buxton (1951) stressed the risk of contamination of carcasses and abattoir premises in a study of faecal samples from pigs at slaughter. Many workers have searched for the source of infection in pigs. Galton, Smith, McElrath & Hardy (1954) demonstrated the importance of abattoir lairages in cross-infection. Later work by McDonagh & Smith (1958) and Bevan Jones *et al.* (1964) showed the relation between build up of infection and duration of stay of pigs in lairages. Shotts, Martin & Galton (1962) demonstrated a consider-

able increase of excretion of salmonellas in pigs during transportation and subsequent holding at abattoir lairages until slaughter.

Newell and his colleagues (1959) noted examination of caecal contents from normal pigs at abattoirs gave higher isolation rates of salmonellas than caecal swabs. These workers traced the source of infection back to the farm of origin of infected pigs and were able to isolate the same serotypes from pigs and feeding meals.

Others have also investigated the source of infection of pigs on farms (Galton *et al.* 1954; Leistner, Johantges, Deibel & Niven, 1961). Intensive farming methods involve large numbers of susceptible animals being reared together on feed concentrate. A considerable proportion of this is derived from animal sources. In Holland, Edel & Kampelmacher (1970) isolated salmonellas from 30% of healthy pigs at slaughterhouses and in Bulgaria 7–31% of pigs were found to be symptomless excretors (Dimitrov *et al.* 1970).

In a study of pigs, mainly conducted in south-west England (PHLS Working Group, Skovgaard & Nielsen, 1972), 5637 samples of caecal faeces and 2483 of mesenteric lymph nodes were examined. Faecal incidence of salmonellas ranged from 2 to 13% with an average of 6%. Of the mesenteric glands, 5% were positive for salmonellas and when both were examined the proportion of infected pigs was twice that obtained by examination of one site only.

The work presented here is concerned with some of the factors associated with the epidemiology of salmonellosis in pigs, including persistence of symptomless excretion, dissemination and sources of infection within the farm, and potential risk of spread between farms through distribution of excretor animals. Investigations were conducted at five farms and one bacon factory.

THE FARMS

Farm A was an intensive pig breeding unit in the west of England, which started with a foundation stock of 6-week-old pigs delivered by hysterectomy from large White and Landrace sows. These animals were received in batches of 22–150 and were housed in new buildings. Two outbreaks of clinical salmonellosis occurred at this farm (Heard, Linton, Penny & Wilson, 1965). The first incident involved two pigs aged 12 weeks. One died after a short illness and the other was destroyed. *S. typhimurium* phage type 1 was isolated from both pigs post-mortem. Rectal swabs of the remaining 62 pigs in the same house showed 44 positive for *S. typhimurium* phage type 1. This organism was also cultured from 22/36 rectal swabs taken at random from pigs in other houses.

Samples of animal feeding stuffs and stool specimens from farm personnel were negative for salmonellas. All animal food was treated for 10 days with nitrofurans (nitrofurazone 25%, furazolidone 3.6%) and the 62 pigs were re-examined. Thirty-eight were still positive for the same serotype. Further clinical cases occurred 6 months later when six pigs died over a period of 15 days. *S. typhimurium* phage type 1 was again isolated at post-mortem. No other clinical infections were recorded after treatment with nitrofurans but many pigs remained symptomless salmonella excretors.

Farm A comprised several subunits, sow yard, boar yard, farrowing houses, performance test houses, rearing houses, feed and equipment stores and office. The different pig houses were divided into pens each for one sow (in farrowing house), one to four boars or 8 to 16 pigs. The pens were fitted with troughs and a mains water point. In the sow yard vasectomized boars were allowed to mix with the sows. The sows were taken to the boar yard for service, remaining about 3 hr. Pigs were moved from house to house and pen to pen as they grew and this contributed to the spread of infection.

When infection persisted structural changes were made aimed at controlling spread of disease. Communal dung channels were abolished to prevent faecal contamination between adjacent pens in each pig house. Where possible, under-floor drainage was constructed. Gradient of floors was adjusted to ensure rapid removal of excreta.

Finished and culled pigs from farm A were sent to the bacon factory about a mile away by lorry which was thoroughly disinfected before and after delivery. Pigs were killed without delay to avoid infection of lairages.

The feed used on Farm A at the beginning of the investigation was 'jumbo nuts' and later specially treated pellets.

Farm B was a semi-intensive farm mainly used for fattening a stock of about 300 middle and large white pigs of varying ages and unknown origin. Animals were housed in brick buildings divided into pens for ten or more pigs. The standard of hygiene was reasonably good. Fish meal and barley meal with mineral mixture was fed to the animals. Mains water was available for drinking.

Farms C and D were semi-intensive premises dating from 1965 and the foundation stock of sows came from Farm A. Other stock was obtained from unrelated sources. Details of these farms were similar to Farm B.

Farm E was a minimum disease pig unit. Reared pigs (10–50 including sows and boars) were produced. Specially prepared pellets were fed to these animals.

MATERIALS AND METHODS

Farms

Fresh faecal samples (mostly composite) were collected from floors of the different houses. These were taken with wooden spatulas (one per sample) into waxed containers (Mono). Many were kept at room temperature overnight and examined next day. About 3 g. of well-mixed faeces was suspended in 15 ml. of selenite F broth (Leifson, 1936) and 15 ml. of tetrathionate broth (Rolfe, 1946). Enrichment media were incubated at 37° C. for 24 hr. and subcultured on deoxycholate sucrose citrate and bismuth sulphite agars. Second subcultures were made from negative samples after 72 hr. incubation. One or more suspicious colonies were picked into tubes of Gillies I and II media (Gillies, 1956) and lysine broth and onto MacConkey agar for purity and lactose fermentation. Early in the investigation negative samples were re-examined but the practice was discontinued, as it was found unrewarding. Slide agglutination was performed from Gillies slopes and cultures were finally submitted to the Salmonella Reference Laboratory for identification.

Bacon factory

Mixed caecal and rectal samples were collected from individual Farm A pigs. These were examined by the laboratory attached to the factory. Cultures thought to be salmonellas were sent to the Food Hygiene Laboratory for checking.

At the same time at the bacon factory a parallel survey was conducted on pigs from other farms in various parts of England. Mixed caecal and rectal samples were collected from each of 15–20 pig carcasses at weekly intervals. The pigs were 4 months to 4 years old.

RESULTS

Farm A

This was studied for 2 years. During this period 413/773 (53%) of faecal samples were positive for salmonellas. The serotypes isolated were *S. bredeney* (148 cultures), *S. durban* (43), *S. heidelberg* (92), *S. manchester* (1), *S. typhimurium* phage type 1 (24), phage type 3a (83), phage type 23 (2) and untypable (17), and three unidentified salmonellas.

Four preliminary samples taken at random from floors of the sow yard, farrowing house I, performance test house I and gully trap, receiving effluent from the various pig houses, contained salmonellas. The serotypes were *S. bredeney*, *S. durban*, *S. heidelberg* and *S. typhimurium* phage type 3a. A fortnight later 16 pen samples and four drain swabs from gully traps were taken. Six pen samples and one drain swab were positive. The serotypes isolated were *S. bredeney*, *S. heidelberg* and *S. typhimurium*. Further detailed surveys of the different pig houses were carried out.

Many boars excreted more than one serotype, e.g. *S. bredeney*, *S. typhimurium* and *S. heidelberg*. On repeated examination certain boars were found to excrete salmonellas regularly. Others were intermittent excretors.

Table 1 shows the incidence and serotypes of salmonellas grown from faeces from pig houses at Farm A.

Table 1. *Incidence and serotypes of salmonellas grown from faeces from pig houses at Farm A*

Source	No. of samples		Serotypes						
	Ex- amined	Positive	T						
			B	D	H	M	1	3a	23
Sow house	342	223 (65)	+	+	+	+	+	+	–
Boar house	235	137 (58)	+	–	+	–	+	+	+
Farrowing houses I and II	73	25 (34)	+	–	+	–	+	+	–
Performance test houses I and II	69	8 (12)	+	–	+	–	–	+	–
Rearing houses early and final	31	13 (41)	+	+	+	–	–	+	–

(Figures in parentheses are percentages)

R = *S. bredeney*; D = *S. durban*; H = *S. heidelberg*; M = *S. manchester*;

T = *S. typhimurium* (phage types indicated underneath).

Young boars, in groups of eight per pen, were kept in the performance test houses I and II for periods of 14–19 weeks according to their rate of food conversion and weight gain. Pen samples were taken within 2–3 days of their arrival and continued at weekly intervals.

Young boars transferred from the performance test houses, which had a lower incidence of salmonella, into the boar yard were negative during the first few days. After using the central passage and after contact with excretors amongst older boars retained in the herd, the young boars began to excrete salmonellas. Therefore, there was continuity of infection. Batches of in-pig sows were examined within 48 hr. of arrival in the farrowing house.

Piglets were weaned at about 6 weeks and were moved to the early rearing house for about 10 weeks. Boars of good potential were moved to the performance test house, while gilts and rejected boars went to the final rearing house.

A salmonella eradication scheme was instituted 2 years after the start of the investigation. All pigs housed in infected pens were slaughtered. Pens were thoroughly disinfected and restocked with animals from salmonella-free pens. Subsequent examination of 88 faecal samples from all houses collected on two occasions gave only one positive – *S. californica*, a serotype not previously found. Six months later all 121 faecal specimens examined on three occasions were negative for salmonellas.

Farm B

Preliminary examination of pen samples from five pens showed 1/5 specimens positive; *S. schwarzengrund* was isolated. Later 14 samples were examined from 14 pens, including those first investigated. *S. schwarzengrund* was again found in the same pen.

A boar known to be an intermittent excretor of *S. bredeney* was introduced into the herd from Farm A. The animal was first isolated for 6 weeks and faeces collected daily for the first 6 days; the specimen taken on the fourth day contained *S. bredeney*. After isolation the boar was allowed to mix with the rest of the herd including pigs from the pen positive for *S. schwarzengrund*. Four weeks later samples were examined from the boar and from 11 other pens. The boar was negative but the incidence of salmonella excretion had increased in the other pigs. Six pen samples contained *S. schwarzengrund* but *S. bredeney* was not found.

Farm C and Farm D

Foundation stocks for both farms came from Farm A. Sampling at Farm C began about 10 months after arrival of pigs. Fifty-eight faeces were tested on three occasions (8, 10 and 40 samples) at four-week intervals. Salmonellas were not isolated at Farm C. Eleven faeces samples collected from pens 5 months after arrival of pigs at Farm D contained *S. bredeney* and an unidentified salmonella in four of the samples. *S. bredeney* and an unidentified serotype were found in the sow yard at Farm A.

Farm E

Batches of 6-week-old pigs produced by hysterectomy on this unit formed the foundation stock for Farm A. Six faecal samples from sows and litters on four occasions were examined with negative results.

Bacon factory

Mixed rectal and caecal faeces from 1053 pigs from Farm A were cultured. Three hundred and thirty-four (32%) contained salmonellas; serotypes isolated were *S. bredeney*, *S. durban*, *S. heidelberg*, *S. orion* and *S. typhimurium* phage types 1 and 3a. *S. orion* had been found on Farm A at an earlier date.

Pigs from other sources were also examined at the bacon factory. Results of faecal cultures of animals from various farms in different counties were compared with those from Farm A. Thirty-four out of 586 (6%) specimens contained salmonellas (Table 2). Fourteen serotypes were found: *S. anatum*, *S.alachua*, *S. bredeney*, *S. canoga*, *S. give*, *S. heidelberg*, *S. litchfield*, *S. livingstone*, *S. manchester*, *S. newport*, *S. panama*, *S. senftenberg*, *S. stanley* and *S. typhimurium* phage types, 9, 12a, U84 and U184.

Information on housing and feeding methods at the farms was not obtained. It was known, however, that pelleting and heat treatment of feeds as used on Farm A were not applied elsewhere in the locality. It is of interest that many serotypes isolated were different to those found on Farm A. Feeding stuffs were suspected as the source of these strains.

DISCUSSION

Symptomless excretor animals are potentially important in the spread of infection. In the present investigation it was observed that the distribution of symptomless excretors from pig-breeding farms resulted in the transmission of infection to rearing establishments.

Faecal samples from pens were more reliable specimens for salmonella isolation than rectal swabs from single pigs (Bevan Jones *et al.* 1964). Rectal swabs provide only a scanty amount of faeces for culture owing to contraction of the sphincter muscles, possibly more pronounced in nervous animals.

During the 2-year investigation, four different serotypes were persistently found on one farm: *S. bredeney*, *S. durban*, *S. heidelberg* and *S. typhimurium* phage types 1, 3a, 23 and untypable. *S. manchester* was isolated once from the sow house and *S. typhimurium* phage type 23 twice from the boar house. Other serotypes were fairly regularly cultured. With the exception of *S. durban* and *S. manchester*, all serotypes isolated on Farm A have been found in animal feeding stuffs (Taylor *et al.* 1965).

Although salmonellas were not isolated from feeds on the farm during the investigation, feeds were thought to be a probable source of infection at some earlier time. Measures were taken to prevent re-introduction of salmonellas by feeding pellets prepared at high temperatures. Absence of new serotypes during the 2-year period suggest that this may have been successful. The comparative

study at the bacon factory of faecal samples from pigs reared on different feeds at other farms showed the existence of other sources of salmonellas entering the factory (Table 2). Edel, Guinée, van Schothorst & Kampelmacher (1967) found that the use of pelleted feeds controlled infection. The high cost of pellets may be offset by a saving in food material.

Although the introduction of further infection into Farm A was prevented, the programme of breeding necessitated movement of pigs and encouraged dissemination and perpetuation of infection through the various pig houses. Symptomless excretors, mainly sows and boars, were responsible for the spread of disease, as they were retained for lengthy periods on Farm A. Most positive samples came from sow and boar yards (Table 1). Sows and vasectomized boars mixed freely. Sows were moved around frequently and vasectomized boars were regularly positive for salmonellas.

In the boar yard the central passage used for exercise probably helped to spread infection. Sows retained for service with boars might become infected or pass on infection. Some boars with excellent genetic potential were found to be regular excretors. Young boars could be infected by older boars through stay in contaminated environments.

Samples from farrowing houses, where sows were kept singly in pens, and from early and final rearing houses showed a lower incidence of infection than those in the other houses. Young animals are regarded as being more susceptible to

Table 2. Incidence and serotypes of salmonellas from caecal and rectal contents of pigs at the bacon factory

Source of pigs	No. of pigs		Salmonella serotypes	Source of pigs	No. of pigs		Salmonella serotypes
	Ex-aminated	Posi-tive			Ex-aminated	Posi-tive	
Berks.	10	0	—	Herts	6	0	—
Bristol	22	1	<i>stanley</i>	Kent	53	5	<i>panama</i>
Cornwall	45	1	<i>anatum</i>				<i>typhimurium</i> U 84
Devon	31	0	—	Norfolk	8	1	unident.
Dorset	21	0	—	Somerset	98	12	<i>bredeney</i> <i>livingstone</i>
Essex	42	3	<i>give</i> <i>typhimurium</i> 9				<i>panama</i> <i>typhimurium</i> U 184
Glos.	46	4	<i>litchfield</i> <i>newport</i> <i>typhimurium</i> 12a	Suffolk	13	0	—
				Surrey	9	2	<i>manchester</i>
Hants	26	4	<i>alachua</i> <i>canoga</i> <i>senftenberg</i> <i>typhimurium</i>	Sussex	26	0	—
				Wilts	30	0	—
				Unknown	100	1	<i>heidelberg</i>

Unident. = unidentified.

The figures under the *S. typhimurium* strains indicate phage types.

infection (Buxton, 1957) and the absence of clinical disease among young piglets may be explained by the fact that they were kept in separate houses and did not come into contact with older boars and sows except for a short period after birth. Colostrum immunity might also be acquired.

Infection by the movement of excretor pigs from farm to farm was demonstrated by transference of animals from Farm A to Farm D which was newly constructed and housing pigs for the first time.

Foundation stock from Farm D included a boar regularly excreting *S. bredeney*, *S. heidelberg* and *S. typhimurium* and a number of sows excreting unidentified salmonellas. Subsequent tests showed infection with *S. bredeney* and unidentified strains persisted not only in boars and sows but also in offspring. Stress factors must be taken account of during transport as these may encourage excretion of salmonellas (Williams & Newell, 1970).

Spread of infection through excretor pigs was also recorded on Farm B. Initial examination showed that samples from one pen contained *S. schwarzengrund*. Isolation of this serotype from animal feed was reported by Taylor *et al.* (1965). In the investigation described, serotypes found in pigs on the farm and in the bacon factory corresponded to those isolated from feeding stuffs. Another survey in the south west of the United Kingdom has given similar results (PHLS Working Group *et al.* 1972). The situation in Britain was contrasted in this study with that in Denmark where feed ingredients of animal origin are terminally heat treated to eliminate salmonellas. This has reduced the prevalence of types other than *S. typhimurium* in pigs in Denmark, as compared with England. Contaminated feeding stuffs are known to initiate symptomless excretion in pigs (Smith, 1960). Meat may become contaminated by faeces during slaughter and it is then a potential vehicle for infecting man.

Stool samples from farm personnel and rodents were negative for salmonellas. The role of wild animals and birds as primary infection sources is debatable. They are usually accepted as being victims of a contaminated environment.

The more animals excreting salmonellas when they leave the farm the greater the build up of infection during transport and more widespread the environmental contamination in the abattoir. The longer the holding period of animals before slaughter the greater is the danger of spread of disease (Galton *et al.* 1954; McDonagh & Smith, 1958; Anderson, Galbraith & Taylor, 1961; Bevan Jones *et al.* 1964).

Implementing effective control measures against human and animal salmonellosis involves taking adequate steps to prevent transmission of infection through animals, detecting and eliminating symptomless excretors from farms and preventing introduction of fresh infection, for example, through contaminated feeding stuffs. The institution of efficient feed sterilization methods could contribute to this control.

I wish to express my gratitude and thanks to Dr J. C. Greatorex and Dr Betty C. Hobbs for their guidance and helpful advice during the planning and execution of the work, and its preparation for publication.

I am grateful to Dr E. S. Anderson and the staff of the Enteric Reference Laboratory, Colindale for the phage-typing of the *S. typhimurium* cultures and to Dr Joan Taylor and the staff of the Salmonella Reference Laboratory, Colindale for serotyping many salmonella cultures.

I am grateful also to Dr R. W. S. Harvey for help in correcting the manuscript and to my colleagues in the Food Hygiene Laboratory for their help in many ways.

Thanks are also due to the staff of various farms and abattoirs from which samples were collected.

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