

at bottom of
Table 7

The specific and non-specific "H" agglutinins of typhi-murium could not be completely exhausted on account of the high titre of the unabsorbed serum.

O = less than 1:100

The results of Table 7 show that typhi-murium removed all "O" type and group agglutinins from 234 serum, as well as from the homologous serum, while culture 234 completely exhausted both typhi-murium serum and its own serum. Accordingly culture 234 must be regarded as identical with typhi-murium, and its antigenic structure should be made up of the same components, viz. "O" antigen IV, V, "H" specific antigen i, "H" non-specific antigen 1, 2, 3.

For fermentation reactions see Table 25.

IV. SALMONELLA INFECTION OF PIGS.

Salmonella infection is fairly common in pigs. Although Salmon and Smith's (1885) interpretation of the significance of S. cholerae-suis as an etiological factor of swine fever is no longer accepted, there can be no doubt that this organism is an important pathogen for pigs and a frequent cause of food-poisoning in man. On comparing the hog-cholera bacillus with other members of the Salmonella group Smith and Moore (1894) found that it fermented dextrose, but no lactose or sucrose, and that it was highly pathogenic for rabbits in very small doses. Kruse (1896) described the hog-cholera bacillus under the name of Bacillus suispestifer and, according to him, Selander regarded this organism as the cause of Danish swine fever.

Preisz (1898) also found Bact. suispestifer pathogenic for pigs and he incriminated it as the cause of "Schweineseuche" (swine-fever) and "Schweineseptikaemie" in Germany.

After Salmon's isolation of the hog-cholera bacillus from the blood and internal organs of most of the cases of swine-fever studied by him, this organism was universally accepted as the cause of this malady until de Schweinitz and Dorset (1904) pointed out that a disease indistinguishable from hog-cholera could be readily produced by injecting healthy pigs with morbid material and body fluids, that had been proved to be free from organisms. Later Dorset, Bolton and McBryde (1905) showed that, while hog cholera could be most readily transmitted by means of inoculations of blood and serum from diseased pigs, the use of cultures of the hog-cholera bacillus only sometimes produced a disease resembling hog-cholera. Whereas pigs infected by means of morbid material from diseased ones could easily transmit swine-fever to in-contact pigs, those that were infected with culture remained practically innocuous for other pigs. They further showed that the causal agent of hog-cholera was contained in the filtrates of the blood and body fluids of sick animals, and that these filtrates, although entirely free from Bact. cholerae-suis, were nevertheless highly infective. They regarded the hog-cholera bacillus merely as an accessory factor in the production of disease.

Bainbridge (1908) divided the members of the paratyphoid group into four sub-groups, *viz.* (1) Paratyphosus-A; (2) Paratyphosus-B which was indistinguishable in its cultural characteristics from aertrycke and suispestifer, but which could be differentiated by means of absorption tests;

(3) aertrycke and suipestifer which were regarded as strains of the same organism indistinguishable from one another; and (4) enteritidis (Gaertner) sub-group easily differentiated from the preceding by means of absorption tests. Later Bainbridge (1911) and Bainbridge and O'Brien (1912) divided paratyphoid bacteria into two groups of separate organisms; the first group they regarded as identical with B. suipestifer, and the second similar to Paratyphosus-B. These workers used agglutination and absorption tests for their identification. The source of Paratyphosus-B was considered to be from cases of paratyphoid fever and carriers, while suipestifer was apparently obtained from contaminated food and cases of food-poisoning. Savage (1912) on the other hand, like Bainbridge (1908), considered that food-poisoning bacilli of the aertrycke type were indistinguishable from suipestifer.

Dammann and Stedefeder (1910) succeeded in infecting healthy pigs either by feeding or by inoculating cultures of B. suipestifer and transmitted swine-fever by means of filtered material. Glässer (1909) found a type of B. suipestifer as the cause of disease in young pigs and called the organism Bac. paratyphi-suis.

Dammann and Stedefeder described B. suipestifer (Voldagsen) as the cause of a disease in young pigs, resembling swine-fever clinically, and known as Ferkel-typhus (suckling pig disease). Although Glässer's bacillus was frequently regarded as identical with the Voldagsen's bacillus it is now known that they differ from each other in that the former is diphasic, while the latter is monophasic, occurring only in the group phase like European cholerae-suis. Both of them differ from suipestifer culturally; but serologically Glässer is indistinguishable from the American variety, whereas Voldagsen resembles the European type.

Jordan (1917), basing his division on the study of recently isolated strains, also divided paratyphoid organisms into four groups:-

(1) Paratyphosus-A which fermented arabinose rapidly and dulcitate slowly, xylose being left unaltered; litmus milk was turned alkaline only after some time.

(2) Paratyphosus-B which rapidly fermented arabinose, dulcitate and xylose, and turned litmus milk alkaline in a very short period.

(3) Suipestifer which fermented xylose rapidly, but arabinose and dulcitate slowly or not at all, i.e. not sooner than after 24 hours incubation.

(4) Enteritidis which was indistinguishable from the Paratyphosus-B group culturally but not serologically.

Jordan and Victorson (1917) used lead acetate agar for the differentiation of the types of paratyphoid bacilli. All enteritidis strains and most Paratyphosus-B strains were found to blacken this medium, while all their suipestifer strains and typical Paratyphosus-A failed to do so. According to Bruce White (1926) Schutze found that the Hirschfeld bacillus and European suipestifer could be distinguished from the American variety by the fact that they readily blackened lead acetate.

In the course of an investigation of swine-fever Uhlenhuth and Hübener (1909) encountered a bacterium which they called Paratyphosus-C bacillus. They found that culturally it was indistinguishable from B. suipestifer, but that it was not agglutinated by either suipestifer or Gaertner serum, while its own serum was without effect on the hog-cholera bacillus. They claimed to have isolated this bacterium from the organs of swine-fever pigs, from sausages, and from human, pig and calf excreta, and regarded it as similar to the organism concerned with calf dysentery.

Heimann (1912) isolated a strain of the so-called paratyphosus-C bacillus from cases of food-poisoning at Hildesheim, following the consumption of infected pork obtained from emergency-slaughtered pigs, but Andrewes and Neave (1921) did not regard the tests employed by Heimann as sufficiently reliable for the recognition of the organisms. Bruce White (1926), on the other hand, identified some of the Hildesheim strains as European hog-cholera bacilli.

During the Great War, and subsequently, several closely related organisms were isolated from cases of paratyphoid fever, especially in Eastern Europe. In 1915 Neukirk (1918) encountered an outbreak of disease in the Turkish army and called the causal organism Erzindjan bacillus. Subsequently several other workers observed a similar type of organism in different localities. Weil and Saxl (1917) isolated them from a number of Russian prisoners suffering from paratyphoid (Walhynian strain). Weil studied another type from Albania, while Dienes and Wagner (1918), on investigating an outbreak of disease among a group of Russian prisoners, encountered several strains which were agglutinated by Voldagsen serum. They regarded their organism as identical with Weil's strain, Neukirk's Erzindjan strain and Uhlenhuth's paratyphosus-C bacillus. Hirschfeld (1919) also investigated an enteric-like disease in the Serbian army and called the causal organism Bacillus paratyphosus-C, in flagrant disregard of the original usage of this term by Uhlenhuth and Hübener (1908). Mackie and Bowen (1919) and Macadam (1919) observed a similar type of organism in Mesopotamia, while Garrow (1920) found it in East Africa. Schutze (1920, 1921) identified an organism isolated in India in 1914 as paratyphosus-C and divided the Salmonella group of organisms into two sub-groups:- (1) Enteritidis (Gaertner) and (2) Paratyphosus-B. He further

divided the latter into four serological types viz. Schottmuller, mutton, Hirschfeld and hog-cholera bacilli. In his mutton type he included the bacillus of swine typhus or animal paratyphosus-B (typhi-murium).

By means of agglutination and absorption tests Bruce White (1926) showed that the Hirschfeld strain and Weil's Albanian strain were identical diphasic organisms, while one of the cultures described by Weil and Saxl proved to be a typical newport strain. The erzindjan strain of Neukirk was also found to be a true Hirschfeld bacillus.

Tenbroeck (1920 a and b) regarded Hirschfeld's bacillus as serologically identical with the American hog-cholera bacillus, but different culturally; whereas the former fermented dulcitol and arabinose and produced hydrogen sulphide, the latter failed to do so. Unlike the hog-cholera bacillus Hirschfeld's organism did not prove to be very pathogenic for rabbits. When these animals were first injected with live Hirschfeld bacilli they were resistant to subsequent inoculations of virulent cholerae-suis. Tenbroeck placed far more reliance on serological tests than on biochemical reactions, and on account of its serum reactions he placed the Hirschfeld bacillus in the hog-cholera group.

Andrewes and Neave (1921) noticed that the Glässer and Voldagsen strains resembled each other culturally but not serologically; Voldagsen serum was completely exhausted of all agglutinins by Glässer, while the specific agglutinins present in Glässer serum were almost entirely unaffected by saturation with Voldagsen. Glässer and Voldagsen did not produce much hydrogen sulphide, whereas this gas was readily formed by (European) suipestifer and paratyphosus-C of Hirschfeld. Andrewes and Neave divided the hog-cholera group of organisms into two sub-groups:- Group 1 comprising

American suipestifer, Glässer's typhi-suis and Hirschfeld's paratyphosus-C, while group 2 was composed of European suipestifer and the Voldagsen strain. They showed that any member of group 1 could exhaust all agglutinins from the serum of any member of group 2, while group 2 strains could not materially reduce the titre of the sera of group 1 strains for members of group 1, although the sera were completely exhausted for the members of group 2.

The various porcine strains of Salmonella of the hog-cholera type, and those closely related human strains to which, regardless of its original usage, the term Bacillus paratyphosus-C is frequently applied; form a group of organisms with very close serological affinities. Although different workers have contributed towards the study of the composition of this group it was Bruce White (1926) who finally divided the members into four well-defined types:-

1. Eastern or Hirschfeld bacillus.
2. American hog-cholera bacillus.
3. European hog-cholera bacillus.
4. Glässer-Voldagsen (Ferkeltyphus) bacillus.

The differential features of these organisms were described by Bruce White (1926) and by Nabarro, White, Dyke and Scott (1929). The specific phases of the diphasic members of the group, viz. Hirschfeld bacillus, American hog-cholera bacillus, and the Ferkeltyphus bacillus (Glässer strain) are indistinguishable; the non-specific phases of the last two are identical, while the European hog-cholera bacillus and the Voldagsen strain differ from them only in so far as they lack any trace of specific phase antigen. The Hirschfeld bacillus differs from all these by the deficiency of its non-specific phase in some of the antigenic components. They all differ from each other biochemically. The Hirschfeld bacillus ferments mannite, dulcitate, arabinose, but not

rhamnose; the hog-cholera bacillus (both European and American) ferments mannite and rhamnose, but not dulcitate and arabinose; while the Ferkel-typhus strains (Glässer-Voldagsen) ferment only arabinose and rhamnose. All strains, excepting the American hog-cholera bacillus, produce hydrogen sulphide.

Tenbroeck (1920 a and b) expressed his surprise at the comparative infrequency of paratyphoid in man caused by the hog-cholera bacillus, while Savage and Bruce White (1925) also remarked upon the rarity of suipestifer food-poisoning in man. They considered that the slight virulence of the organism for man and the massive doses required for setting up an infection are responsible for the low incidence of the disease. According to Krüger (1932b), it was declared by Uhlenhuth (1926), at a meeting of the German Society of Microbiology, that suipestifer bacilli could not be regarded as very pathogenic for man; and it was stated by Ostertag that, although thousands of swine-fever pigs were slaughtered for human consumption, mass infection of man did not occur. Kruger considered that suipestifer frequently lives as a saprophyte in the human body, setting up an infection only when the resistance has been lowered by conditions like appendicitis. Nevertheless, Salmonella infection of porcine origin has been known to cause serious disease in man. Indeed, the number of human cases of infection with cholerae-suis recorded during recent years cannot be treated as insignificant. Apart from the number of outbreaks of paratyphoid fever in man in Eastern Europe, due to the Hirschfeld bacillus, several cases are reported from time to time where the hog-cholera bacillus has been incriminated as the cause of the disease. Krumwiede, Provost and Cooper (1922) recorded an outbreak of paratyphoid fever in four members of a family after eating tapioca pudding.

One of the patients died and S. cholerae-suis was isolated from the liver. It was thought that the source of the infection was pork that contaminated the pudding. Scott (1926) described four outbreaks of infection due to the European hog-cholera bacillus, involving over a hundred persons. In all the cases the source of the infection was traced to prepared meats. A fatal case of septicaemia in man caused by "Bacillus (Salmonella) suipestifer" (America) and resembling typhoid fever was studied by Bauer and McLintock (1929), while two cases of human infection with the American hog-cholera bacillus were reported by Nabarro, Bruce White, Dyke and Scott (1929). Another case of American suipestifer causing disease in man in England was described by Boycott and McNee (1936); the organisms were obtained from blood culture, but although they were diphasic they resembled the European type culturally. Symptoms of malaise, fever, shivers, sweating, pains in different parts of the body and mental disturbances were observed.

Clayton, Milne and Menton (1930) recorded an outbreak of acute gastro-enteritis in eight persons following the ingestion of pork pie. Three of the cases ended fatally; from the intestines of these patients as well as from the stools of the other five, American suipestifer was isolated. Another case of cholerae-suis infection of man was described by Branham, Motyca and Devine (1930), the most outstanding symptoms being intermittent chills, headaches, delirium, fever, cystitis, pyelitis and proctitis. The condition lasted for over 5 weeks and the organism was obtained from blood culture. Kuttner and Zepp (1932, 1933) reported eleven cases of suipestifer infection, mostly in children. Of these ten were due to the European variety, and only one was caused by the American type; symptoms of septicaemia, cystitis, arthritis, broncho-pneumonia and meningitis were

manifested to a varying degree, and in some cases the symptoms were mistaken for those of typhoid fever. Bact. suipestifer was obtained by blood culture from all the patients. All the cases recovered excepting one which ended fatally.

In Germany Kôbe (1930) described two strains of suipestifer obtained from cases of meat-poisoning following the ingestion of pork. The patients showed symptoms of septicaemia with gastro-enteritis, as in typhoid fever. Krüger (1932a) reviewed several outbreaks of paratyphoid fever in which Bact. suipestifer was incriminated as the causal agent. He described one case in which the source of infection was traced to pork sausage; symptoms of pyrexia, pruritus and icterus, lasting for several weeks, were manifested, and Bact. suipestifer (Kunzendorf) was isolated from the blood and urine of the patient.

Giglioli (1930) studied a number of cases of quinine resistant fever in British Guiana and found Hirschfeld bacillus in 72 out the 77 ^{patients} cases examined. The organisms isolated corresponded both culturally and serologically with Hirschfeld's bacillus. More recently D'Hooghe (1932) and Mattlet (1932) described a number of fatal cases of paratyphoid fever in the Belgian Congo where they incriminated Hirschfeld's bacillus as the cause, while Tenbroeck, Li and Yii (1931) recorded five cases of infection in man caused by the same organism in Peiping (China). Materna and Januschke (1925) incriminated cholerae-suis as the cause of purulent meningitis in a man, while Rovitch and Washington (1937) described several cases of suipestifer septicaemia in Negro children.

In South Africa Greenfield and Judd (1936) and Henning and Greenfield (1937) have described an outbreak of

TABLE 10 (after Henning and Greenfield).

Antigen.	UNABSORBED SERA.					ABSORBED SERA.									
	Afri. Aust. serum unabsorbed.	Heidelberg serum unabsorbed.	Kunzen-dorf serum unabsorbed.	Newport Kottbus serum.	Muenchen serum.	Bovis-morbificans serum unabsorbed.	Afri. Aust serum with Afri. Aust.	Afri. Aust. serum with Kunzen-dorf.	Afri. Aust serum absorbed with Heidelberg.	Afri. Aust. serum absorbed with Bovis-morbificans.	Kunzen-dorf serum absorbed with Afri. Aust.	Heidelberg serum absorbed with Afri. Aust.	Bovis-morbificans serum absorbed with Afri. Aust.	Bovis-morbificans serum absorbed with Bovis-morbificans.	Afri. Aust serum absorbed by Heidelberg and Kunzen-dorf.
Afri. Aust. "O".....	800	0	0	1600	800	1600	0	400	800	0	-	-	0	0	400
Afri. Aust. Type.....	6400	6400	0	-	-	6400	0	6400	0	0	-	-	0	0	0
Afri. Aust. Group....	6400	3200	3200	-	-	25600	0	400	3200	0	0	-	0	0	400
Heidelberg "O".....	0	800	-	-	-	-	-	-	-	-	-	800	-	-	-
Heidelberg Type.....	6400	6400	-	-	-	-	-	-	0	-	-	0	-	-	-
Heidelberg Group.....	1600	12800	800	-	-	-	-	-	0	-	-	6400	-	-	-
Kunzen-dorf "O".....	50	-	800	-	-	-	-	-	-	-	800	-	-	-	-
Kunzen-dorf "H".....	3200	-	3200	-	-	-	-	0	-	-	200	-	-	-	-
Bovis-morbificans "O"	800	-	0	-	-	1600	0	-	-	0	-	-	0	0	-
Bovis-morbificans Type	3200	-	-	-	-	6400	0	-	-	0	-	-	0	0	-
Bovis-morbificans Group	6400	-	-	-	-	25600	0	-	-	0	-	-	0	0	-
Newport (Kottbus) "O"	-	-	0	1600	-	-	-	-	-	-	-	-	-	-	-
Muenchen "O".....	-	-	0	-	800	-	-	-	-	-	-	-	-	-	-

0 = less than 1:50.

The organism was originally described by Greenfield and Judd (1936) as a new Salmonella - suipestifer var. Afri. Aust. But later Henning and Greenfield (1937) showed that it is not distinguishable from bovis-morbificans, Base-nau. Cultures of the organism were tested against various "O" and "H" sera. These were agglutinated by "O" sera of organisms containing factors VI and VIII of the Kauffmann-White schema, by group sera and by the type sera of heidelberg and bovis-morbificans. The organism was, therefore, regarded as diphasic.

A culture was plated so as to yield several well-separated colonies after 24 hours incubation; a number of fresh colonies were picked into broth and incubated for 5 to 6 hours at 37°C. The cultures obtained were tested against a pure group serum, e.g. Kunzendorf serum, and also against the type sera of heidelberg and bovis-morbificans. The colonies that occurred in the group phase agglutinated with Kunzendorf serum, while those that occurred in the type phase were flocculated only by heidelberg and bovis-morbificans sera. Group, type and "O" suspensions were now prepared and tested against a number of sera (Table 10). It will be noticed that heidelberg serum agglutinated both the group and type antigens, but not the "O" antigen, that kunzendorf flocculated the group antigen, but neither the type nor the "O" antigen, and that bovis-morbificans serum agglutinated all three antigens up to a very high titre.

After suitable antisera were prepared against Afri. Aust. absorption tests were performed (Table 10). On absorbing Afri. Aust. serum with heidelberg all agglutinins for the type phases of both heidelberg and Afri. Aust. were exhausted, but there was hardly any reduction of the group agglutinins (from 6400 to 3200), and all the "O" agglutinins remained. When this partly absorbed serum was re-absorbed by Kunzendorf a marked reduction of group agglutinins (from

3200 to 400) was effected, but the "O" titre remained unaltered; Kunzendorf also reduced the ^{group}"O" titre of unabsorbed Afri. Aust. serum from 6400 to 400, but it had no effect on the type agglutinins. On the other hand, Afri. Aust. removed all the type, but very little of the group agglutinins from heidelberg serum, and it did not reduce the "O" titre. Moreover, Afri. Aust. absorbed most of the group agglutinins (from 3200 to 200) from Kunzendorf serum without reducing its "O" titre appreciably.

On absorbing bovis-morbificans serum with Afri. Aust. and Afri. Aust. serum with bovis-morbificans all the type, group and "O" agglutinins for both organisms were completely exhausted.

It will be observed that Kunzendorf did not completely exhaust the group agglutinins from Afri. Aust. serum and that Afri. Aust. failed to remove all the group agglutinins from Kunzendorf serum. This occurrence cannot be explained as Afri. Aust. and bovis-morbificans have the same group antigenic factors, and, according to the Kauffmann-White schema, the group antigens of Kunzendorf and bovis-morbificans are identical. There was barely any "O" agglutination between Afri. Aust. and Kunzendorf, indicating that the somatic factor VI of Kunzendorf is either absent or poorly represented in Afri. Aust.

These results clearly show that S. suispestifer var. Afri. Aust. of Greenfield and Judd has the same type antigen as heidelberg and bovis-morbificans (factor r) and a group antigen that corresponds largely with that of Kunzendorf and entirely with that of bovis-morbificans (factors 1, 3, 4, 5), while its somatic "O" antigen resembles that of bovis-morbificans (factors VI, VIII). Moreover, since Afri. Aust. removes all agglutinins, type, group and "O", from bovis-

morbificans serum, and bovis-morbificans exhausts all agglutinins from Afri. Aust. serum there can be no doubt that the two organisms are identical.

The only other record of a Salmonella obtained from a pig is that of Robinson and Martinaglia (1932) when they described an organism isolated from a pig at Onderstepoort. A description of the antigenic structure of this organism, strain 192, was not attempted by them, but its antigenic analysis was subsequently performed by ~~the~~^{the} author (Table 8). It was noticed that strain 192 was agglutinated far better by typhi-murium than by cholerae-suis serum. The agglutination obtained with the latter serum was purely floccular, while with the former the agglutination was both granular and floccular, suggesting the existence of a closer relationship between strain 192 and typhi-murium than between it and cholerae-suis. Moreover, strain 192 was found to be diphasic; its type phase colonies were agglutinated by typhi-murium type serum, while the group phase colonies were flocculated by both typhi-murium and cholerae-suis group sera. Accordingly agglutination and absorption tests were performed as shown in Table 8, mixed "O" and "H" type and group sera being used for the tests. The results show that typhi-murium removed all agglutinins ("O", "H" type and "H" group) from 192 serum as well as from the homologous serum; culture 192 also completely exhausted both typhi-murium serum and its own serum. Accordingly it was evident that culture 192 and typhi-murium were composed of the same antigenic structure, and that they both contained the following antigenic factors of the Kauffmann-White schema:- "O" IV, V, "H" specific 1, "H" non-specific 1, 2, 3.

TABLE 8.

Antigen.	<u>Typhi-murium</u> s.a.b. <u>typhi-murium.</u>	<u>Typhi-murium</u> s.a.b. 192	192 s.a.b. <u>typhi-murium.</u>	192 s.a.b. 192.	<u>Typhi-murium</u> s. unab- sorbed	192 s. unab- sorbed.
<u>Typhi-murium</u> "O"	0	0	0	0	800	1600
<u>Typhi-murium</u> "H" type	100	100	0	0	100,000	6400
<u>Typhi-murium</u> "H" group	0	0	0	0	25,000	3200
192 - "O"	0	0	0	0	800	1600
192 - "H" type	100	100	0	0	100,000	6400
192 - "H" group	0	0	0	0	25,000	6400

0 = less than 1:100; s = serum; a.b. = absorbed by. The "H" type titre of typhi-murium was so high (1:100,000) that it was impossible to remove a small residue (1:100) of the agglutinin.

In 1933 Dr. Robinson and myself isolated another strain of Salmonella (culture 168) from the blood of pigs suffering from a septicaemic disease in the Cape Province. This organism was readily agglutinated by cholerae-suis serum and was found to occur entirely in the group phase. Accordingly, absorption tests were conducted as shown in Table 9.

TABLE 9.

Antigen	Cholerae-suis (European) s.a.b. Cholerae-suis (European).	Cholerae-suis (European) s.a.b. 168.	168 s.a.b. Cholerae-suis (European).	168 s.a.b. 168.	168 s.a.b. Cholerae-suis (America).	Cholerae-suis (America) s.a.b. 168.	Cholerae-suis (America) s.a.b. Cholerae-suis (America).	Cholerae-suis (European) s. unab-sorbed.	168 serum unab-sorbed.	Cholerae-suis (America) serum unabsorbed.
Cholerae-suis (European) "O"	0	0	0	0				800	1600	
Cholerae-suis (European) "H"	0	0	0	0				6400	12800	
168 - "O"	0	0	0	0	0	0	0	800	1600	800
168 - "H"	0	0	0	0	0	0	0	6400	12800	6400
Cholerae-suis (America) "O"					0	0	0		1600	800
Cholerae-suis (America) "H" type					0	3200	0		0	3200
Cholerae-suis (America) "H" group					0	0	0		12800	6400

0 = less than 1:100

s = serum

a.b. = absorbed by

The results show that culture 168 removed all agglutinins from cholerae-suis (European) serum as well as from the homologous serum, and that cholerae-suis (European) exhausted both 168 serum and its own serum. Absorption tests were also carried out with the diphasic American hog-cholera bacillus and its serum. It was found that culture 168, while completely removing all the "O" and group agglutinins from cholerae-suis serum, left the "H" specific titre almost unaltered. On the other hand cholerae-suis (America) completely exhausted all the agglutinins ("O" and "H" non-specific) from 168 serum. The results, therefore, showed that culture 168 was devoid of an "H" specific antigen and that it contained the same antigenic components as cholerae-suis (European), viz. the following factors of the Kauffmann-White schema:- "O", VI, VII. "H" non-specific 1, 3, 4, 5. "H" specific nil.

Six other strains, cultures 365, 380, 381, 382, 383, 384, isolated from the blood of pigs during an outbreak of swine fever in the Transvaal were also studied serologically. With strain 365 complete mirror absorption tests were carried out as in the case of culture 168 (Table 9) and exactly similar results were obtained. With the other five strains one-sided absorption tests were performed, using both European and American hog-cholera sera. Whereas all the agglutinins ("O" and "H" non-specific) were removed from European hog-cholera serum the "H" specific agglutinins of the American suipestifer serum were left unabsorbed. All six strains were found to be monophasic.

These results, therefore, clearly show that strains 365, 380, 381, 382, 383, 384 resemble cholerae-suis (European) antigenically, containing the same antigenic factors assigned to strain 168.

For fermentation tests see Table 25.

Murray (1934) cites several different workers who have isolated cholerae-suis from the faeces of a small percentage of apparently healthy pigs. He states, however, that he has been unable to demonstrate the presence of suipestifer in normal pigs.

V. SALMONELLA INFECTION OF EQUINES.

In horses infection with S. abortus-equi is undoubtedly the most common disease caused by the genus Salmonella. More than forty years ago Kilborne (1893) and Smith (1893) studied an outbreak of abortions in mares and isolated a non-lactose fermenting organism of the hog-cholera group from the vaginal discharges of the affected animals. This organism was found to be pathogenic for rabbits and was regarded as the cause of the abortions; on cultivation it formed a membranous growth with wrinkled edges on the agar.

Subsequently several different workers investigated outbreaks of infectious abortion in mares caused ostensibly by the same bacterium studied by Kilborne and Smith. Good and Corbett (1913) studied a very serious epizootic in Kentucky due to organisms of the enteritidis hog-cholera group, which produced nearly 100 per cent abortions. Intravenous inoculations of cultures of this organism caused abortions in mares within 10 days. About the same time Meyer and Boerner (1913), de Jong (1913), Dassonville and Riviere (1913), van Heelsbergen (1914) and Schofield (1914) also described epizootics of abortion in mares due to Bact. abortus-equi. Later MacFadyean and Edwards (1917) discussed the relationship of infectious abortion in mares and joint-ill in foals, while Miessner and Berge (1917) and Murray (1919) also incriminated abortus-equi as the etiological agent of outbreaks of abortion in mares.

Apart from causing abortions in equines this organism has been found ~~to be~~ responsible for pyaemic arthritis, joint-ill, abscessation and tendo-vaginitis. While studying the etiology of infectious arthritis in colts in America, Good and Smith (1914) isolated from the pus of the joints a bacterium which resembled the causal agent of infectious abortion in mares; but from the affected synovia of one foal they obtained only streptococci. In the outbreaks of pyaemic arthritis in foals investigated by Schofield (1914) Gram-negative bacteria were isolated in pure culture from the synovia of the affected joints - in a few cases only, the cultures yielded a mixed growth of Gram-negative bacteria and streptococci. The former were regarded as closely related to the bacterium of contagious abortion in mares. In Germany Miessner and Berge (1917) ascribed the cause of a severe epizootic of abortion in a stud to a paratyphoid organism, which was isolated from the stomach and intestines of dead fetuses; they pointed out that the majority of the foals ^{which} ~~that~~ were born alive on the affected farm developed joint-ill, but streptococci were regarded as the most important etiological agent, paratyphoid organisms being obtained from only one case. In a comprehensive study of contagious abortion in mares and joint-ill in foals, MacFadyean and Edwards (1917) found Bact. abortus-equi as the most common cause of the two diseases. They isolated this organism from the heart-blood and internal organs of several of the aborted fetuses, and also from the joints of a number of foals affected with joint-ill. Some of the horses that were immunised with abortus-equi for the purpose of serum production developed arthritis. Magnusson (1919) on the other hand, considered an organism, which he called Bact. viscosum equi, as the most common cause of joint-ill in foals.

In South Africa Martinaglia (1929) ~~has~~ described several cases of tendo-vaginitis in adult horses due to abortus-equi following horsesickness immunisation. Out of twelve cases studied in 1922, nine yielded pure cultures of abortus-equi, while in the remaining three a mixed infection of this organism and a streptococcus was found. One animal, a stallion, was affected with orchitis due to abortus-equi. In 1925 a similar condition appeared in mules, also after immunisation against horsesickness.

Seymour (1936) also incriminated abortus-equi as the cause of an outbreak of pyosepticaemia in foals, while Fujimura and Hoshi (1936) described outbreaks of contagious abortion and cases of abscessation in equines due to this organism. Moreover, they reported a case of abortus-equi infection in man.

Although the antigenic structure of the organism incriminated in these outbreaks is not clearly given, there seems to be very little doubt that abortus-equi, or a very closely related bacterium, was responsible for most of the cases. The strains isolated by Martinaglia were described as actively motile; but only one of these, culture 219, was kept. When this strain was finally received by me it was found to have lost all its properties of motility. After preparing antisera, agglutination and absorption tests were performed with this organism and abortus-equi WH2. The results of these tests showed conclusively that culture 219 and abortus-equi WH2 had the same somatic antigen; culture 219 removed all the "O" agglutinins from abortus-equi WH2 serum as well as from the homologous serum, ~~On the other hand,~~ ^{while} abortus-equi WH2 completely exhausted the "O" agglutinins from both sera. As culture 219 was non-motile its serum was devoid of "H" agglutinins and it left the "H" agglutinins of abortus-equi WH2 ^{Serum} unaltered.

Apart from abortus-equi infection other types of Salmonella are sometimes responsible for outbreaks of disease in solipeas. Thus, Moulin and Amichau (1918), Combes (1918) and Urbain, Stocanne and Chaillot (1929) described epizootics in horses due to paratyphoid bacilli. Graham, Reynolds and Hill (1919) studied a virulent outbreak of disease in a shipment of horses and mules due to enteritidis. Meissner incriminated typhi-murium as the cause of a disease in foals and obtained this organism as well as abortus-equi from mares that had aborted. Moreover, Lutje (1930) isolated both enteritidis and typhi-murium from equines affected with abortion, and he obtained enteritidis from foals exposed to infection with calves. Standfuss (1925) and Lehr (1928) isolated paratyphoid organisms from horses that were slaughtered in emergency. Other workers like Baumann and Gratzl (1931) and Arnberger (1931) described outbreaks of gastro-enteritis in horses due to typhi-murium, while Edwards (1934) investigated an epizootic of infectious colitis in 3 to 7 months old foals caused by the same organism. Cernczubov, Filipovic and Stavel (1937) claim to have isolated five strains of typhi-murium and four of paratyphi-B from diseases in horses.

But apart from causing disease in equines Salmonella infection of horses may lead to serious outbreaks of food-poisoning in countries where horse flesh is used for human food. Thus, during the Great War and the years immediately following ^{it} more than 25 per cent of all outbreaks of meat-poisoning in Germany were traced to horse meat; ^{on the other hand} while the incidence of gastro-enteritis from this source has been very low during recent years (Meyer, 1934, 1936). In 1923 Meyer recorded 19 outbreaks and in 1932 only one that could be ascribed to this cause. Kuppelmayr (1924) described 47 outbreaks of food-poisoning, involving

5440 cases and causing 63 deaths, all traced to the consumption of infected horse meat. Elkelas (1925) recorded 61 outbreaks of meat-poisoning in Germany during 1923, involving 3093 persons and causing 20 deaths; the majority of these cases were due to horse-meat. Glage (1916) studied an epizootic of food-poisoning due to horse-meat; 392 persons were affected and there were 2 deaths. Organisms of the paratyphus-B group were isolated from the suspected meat and from the stools of the patients. Infection of man following the consumption of horse-meat was also described by Muller (1921). Clarenburg (1931) described two outbreaks of food-poisoning in Holland where the cause of infection was horse-meat. Uhlenhuth (1926) isolated typhimurium from patients who were suffering from acute gastroenteritis following the consumption of horse-meat, while Kauffmann and Silberstein (1934) obtained anatum var. Muenster from a person who had developed food-poisoning after a meal containing raw horse-meat. Several other outbreaks of Salmonella food-poisoning in man resulting from the ingestion of horse-meat has been studied in the Reichsgesundheitsamt in Germany. Many of these have followed the consumption of meat from animals slaughtered in emergency.

Recently my colleague, Mr. R. Clark, investigated an outbreak of purulent arthritis in foals in the ^{Orange} Free State. He obtained pus from the affected joints of one foal and made cultures on agar slants; the growths obtained were submitted to me for identification. These were plated on MacConkey's bile salt agar and yielded pure cultures of a non-lactose fermenting bacterium which looked like a salmonella. Several of the single colonies obtained were tested against various "O", type and group sera. They were all agglutinated by the "O" sera of group B the Kauffmann-

White schema, and it was at first thought that the organism was probably abortus-equi. But, on further testing, it was found that some of the colonies were agglutinated by typhi-murium type serum, while others were flocculated by a pure group serum, like that of cholerae-suis var. Kunzendorf. The organism (culture 478), therefore, was diphasic. Accordingly, antisera were prepared against it for the purpose of carrying out agglutination and absorption tests.

These tests (Table 26) show that typhi-murium removes all "O", "H"-type and "H"-group agglutinins from 478 serum, while 473, although completely exhausting the "H" agglutinins from typhi-murium^{serum}, merely reduced the "O" titre of the serum from 800 to approximately 200. Culture 478 also absorbed all the type and group agglutinins from aberdeen serum without altering the "O" titre, and aberdeen exhausted all the "H" agglutinins from 478 serum, but failed to reduce its "O" agglutinin content. On the other hand, culture 478 removed all the agglutinins ("O", type and group) from both storrs and copenhagen sera, while both storrs and copenhagen completely exhausted 478 serum. The results of these tests, therefore, showed that culture 478 is identical with Salmonella typhi-murium var. storrs (Edwards, 1935), or S. typhi-murium var. copenhagen (Kauffman, 1934), containing the following antigenic formula:- "O" 1V, "H"-specific 1, "H"-non-specific 1, 2, 3. The fermentation reactions of culture 478 are given below (Table 25).

Strains of Salmonella typhi-murium devoid of "O" factor V were first described by Landsteiner and Levine (1932) when they studied the Binns strain of Schutze. Later Kauffmann (1935a) recorded 16 variants of typhi-murium which contained "O" factor 1V, but not factor V, and he called these variants typhi-murium var. Copenhagen. About the same time

(1934)
 Jungherr and Wilcox^h obtained from pigeons a strain of typhi-murium which reacted atypically with maltose; an antigenic analysis of this organism made by Edwards (1935)^h showed that it is lacking in "O" factor V. Edwards called the variant typhi-murium var. Storrs. Hohn and Hermann (1937) also recorded an outbreak of disease in pigeons due to the 1V-variant of typhi-murium, while Hoffmann and Edwards (1937) studied an infection in rabbits caused by the same type of organism. Moreover, several cases of infection in man due to strains of typhi-murium devoid of "O" factor V have been described by Zahn (1935).

Both Edwards and Kauffmann found that the 1V-variants exhibited biochemical reactions that are not typical for typhi-murium and that strains from different localities did not always react in the same way.

Edwards (1938) points out that all the recorded outbreaks of disease due to 1V-variants of typhi-murium have occurred in man, pigeons and rabbits; no 1V-variants were found among typhi-murium cultures obtained from horses, sheep, guinea-pigs, rats, mice, turkeys, chickens, ducks and canaries. The strain of typhi-murium var. Copenhagen (Storrs) described by me is, therefore, the first record of this organism obtained from a horse.

742
TABLE 26A

Antigen	ABSORBED SERA.												UNABSORBED SERA								
	478 serum absorbed by:-						Storrs serum absorbed by:-		Copenhagen 659 serum absorbed by:-		typhi-murium serum absorbed by:-		abortus-equi serum absorbed by:-		Aberdeen serum ab. by:-						
	478	Storrs	Copen- hagen 659	typhi- murium	Abor- tus- equi	Aber- deen	478	Storrs	478	Copen- hagen	478	typhi- murium	478	Abor- tus- equi	478	478	478	Storrs	Copen- hagen	typhi- murium	Abor- tus- equi
478-"O"	0	0	0	0	0	800	0	0	0	0	0	0	0	0	0	800	1600	1600	800	800	0
478-type	0	0	0	0	12800	0	0	0	0	0	0	0	0	0	0	12800	6400	12800	25600		50,000
478-group	0	0	0	0	3200	0	0	0	0	0	0	0	0	0	0	3200	25600	6400	25600		6,400
Storrs-"O"	0	0	-	-	-	-	0	0	-	-	-	-	-	-	-	800	1600	-	-	-	-
Storrs-type	0	0	-	-	-	-	0	0	-	-	-	-	-	-	-	12800	6400	-	-	-	-
Storrs-group	0	0	-	-	-	-	0	0	-	-	-	-	-	-	-	3200	25600	-	-	-	-
Copenhagen "O"	0	-	0	-	-	-	0	0	0	0	-	-	-	-	-	800	-	1600	-	-	-
Copenhagen-type	0	-	0	-	-	-	0	0	0	0	-	-	-	-	-	12800	-	12800	-	-	-
Copenhagen group	0	-	0	-	-	-	0	0	0	0	-	-	-	-	-	3200	-	6400	-	-	-
typhi-murium "O"	0	-	-	0	-	-	-	-	-	-	200-400	0	-	-	-	800	-	-	800	-	-
typhi-murium-type	0	-	-	100	-	-	-	-	-	-	100	100	-	-	-	12800	-	-	25600	-	-
typhi-murium-group	0	-	-	100	-	-	-	-	-	-	100	100	-	-	-	3200	-	-	25600	-	-
Abortus-equi "O"	0	-	-	0	0	-	-	-	-	-	-	-	0	0	-	1600	-	-	-	1600	-
Abortus-equi "H"	0	-	-	0	0	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Aberdeen "O"	0	-	-	-	-	0	-	-	-	-	-	-	-	-	400	0	-	-	-	-	400
Aberdeen type	0	-	-	-	-	0	-	-	-	-	-	-	-	-	100	12800	-	-	-	-	50,000
Aberdeen group	0	-	-	-	-	0	-	-	-	-	-	-	-	-	0	3200	-	-	-	-	6,400

VI. SALMONELLA INFECTION IN BIRDS.

Infection of birds with different types of Salmonella is much more varied and widespread than in mammals, and the losses sustained through this group of organisms are probably far greater than those resulting from any other cause. Epizootics in fowls due to S. gallinarum are extremely common in some countries; in South Africa, fowl typhoid is without doubt the most serious infectious disease of fowls, while in Europe and America Pullorum disease seems to be more important. Epizootics due to Salmonellas other than gallinarum and pullorum, although less common, may nevertheless be responsible for serious losses in all species of domestic birds. It is with a discussion of these diseases that this part of my paper is chiefly concerned. Although no authentic cases of paratyphoid in pigeons, ducks and geese have so far been recorded in South Africa, my discussion will not be complete unless the literature relating to disease in these birds is duly reviewed. Moreover, Salmonella infection, other than that due to gallinarum and pullorum occurs apparently more frequently in them than in gallinaceous birds. The extensive literature relating to fowl typhoid and pullorum disease is not discussed in this paper; it has been fully reviewed by a number of different workers.

Salmonella infection is most common, and also most serious, in very young birds. Adult birds usually suffer from a chronic form of the disease with lesions in the ovary, testes, joints, liver and spleen; whereas in young birds septicaemia and enteritis with changes in the internal organs are more frequently observed. The infection may be the cause of serious losses in the affected ^{flocks} ~~herds~~ and of food-poisoning in man, either through the medium of infected meat

or eggs. Several different species of birds may be affected, and a number of different types of Salmonella have been incriminated as etiological agents.

In a recent review Schaaf (1936) mentioned typhimurium, enteritidis, anatum, cholerae-suis and abortus-equi as the causes of paratyphoid in birds; while Edwards (1936, 1937) has found oranienburg as the cause of an infection in quail and Senftenberg responsible for a disease in turkeys. Recently I have recorded an outbreak in chickens due to Salmonella amersfoort (Henning, 1937). Typhimurium seems to be the most common cause, with enteritidis next in importance; the other organisms are only rarely found.

Natural infection usually occurs by means of food or water contaminated with the excreta of infected animals or birds, but transmission may also take place through the medium of the egg which has obtained the infection in the ovary or oviduct, or which has been contaminated by means of infected faeces. Sometimes the embryo is dead in the shell as a result of the infection, but generally the newly hatched birds develop the disease during the first few days of life. There are several predisposing factors like bad hygiene, improper feeding and infestation by parasites which favour infection; the dirty habits of water birds, probably account for the frequency of paratyphoid in ducks and geese, as well as the number of outbreaks of food-poisoning that result from the ingestion of food-stuffs containing their eggs or meat as ingredients.

Lerche (1936) considers that 5.7 per cent. of the ducks' eggs sold in Germany ^{are} may be infected with Salmonellas. Frequently the shell is contaminated with infected faeces and under favourable conditions the organisms penetrate from the shell into the interior of the egg; but although the yolk is an excellent culture medium, the albumen of the fresh egg is strongly bactericidal (Lachtschenko, 1909, Rettger and Sperry, 1912, and Scott, 1930). This germicidal action, however, deteriorates when the egg becomes stale and when it

is exposed to warm, moist weather for more than two weeks, the organisms may penetrate into the interior and increase in number; this increase occurs only when some yolk has diffused into the albumen. The most dangerous source of infection is food which contains duck's eggs as an ingredient, and in which the organisms can readily multiply, e.g. creams, custards, puddings and "Hackfleisch" that have not been sufficiently heated during the preparation; mayonnaise is too acid for bacterial growth and is, therefore, less dangerous. The danger of eating duck's eggs in the raw state is obvious, but even frying or boiling may not be sufficient to kill the organisms in an infected egg. Lerche (1936) considers that after 5 minutes boiling the temperature of the yolk of a duck's egg may not be much more than 40° C., while Bruns and Fromme (1934) state that after boiling an egg in the shell for 3½ minutes the temperature in the interior is only 28°C., but after 5 minutes boiling it rises to about 65°C.

(1) Pigeons.

The first record of a disease in birds caused by a Salmonella is given by Moore (1895) in his description of a severe epizootic in pigeons due to a bacillus of the hog-cholera group. The organism was recovered from the heart-blood and internal organs of affected birds; it was found to be pathogenic for rabbits, mice and guinea-pigs, and it fermented dextrose, but not lactose or saccharose. Salmon (1904) also described a rapidly fatal disease in pigeons, due to an organism of the "enteritidis group". The suspected bacterium was obtained from the internal organs of affected birds, and it was found to be pathogenic for small laboratory animals as well as ^{for} pigeons. Enteritis was the most outstanding lesion recorded. Another outbreak of pigeon paratyphoid was described by Zingle (1914), when he investigated a mortality among military birds at Strassburg. No definite symptoms were described, but the birds lost their condition in spite of good food. After death the liver was found to be enlarged

and yellowish-grey, while numerous greyish nodules of varying sizes were scattered throughout the pectoral muscles, liver and kidneys. Organisms of the Paratyphus-B group (typhi-murium?-) were obtained in pure culture from the heart-blood and organs of diseased birds, but it was not quite clear whether this infection was primary or secondary as the birds were affected simultaneously with pigeon-pox. The invasion of the body by paratyphoid organisms under certain abnormal conditions is explained by Cash and Doan (1931). They have found that latent infections with typhi-murium become seemingly active under adverse conditions; they have reported an apparently spontaneous development of a fatal disease in under-nourished pigeons associated with myeloid hyperplasia of the bone-marrow, an increase of the myeloid elements of the blood, and necrotic foci in the liver, spleen, kidney and bone-marrow, Typhi-murium was regularly recovered from the heart-blood and organs. A similar disease was set up experimentally in normal pigeons by the inoculation of liver emulsions and cultures of the organisms.

Subsequently several other workers described outbreaks of Salmonella infection in pigeons. Thus, Reitsma (1924) studied an epizootic in Holland, as a result of which the pigeons developed an ulcerative enteritis and became very much emaciated; a pure culture of an organism, labelled "B. paratyphus-B" (typhi-murium) was obtained from the liver of the affected birds. Sahaya and Willems (1927) recorded a chronic and an acute form of the disease affecting adult and young birds respectively. The adult pigeons were usually afflicted with a severe arthritis and swelling of the joints, associated with softening and atrophy of the pectoral muscles, while young birds suffered mostly from acute enteritis, About 65 percent. of the young birds died from the disease, and most of the survivors scoured for several months on end. The latter apparently remained carriers and generally transmitted the infection in turn to their off-spring. The most important

lesions recorded were caseous nodules in the lungs, small white nodules in the liver, ulceration of the mucous membrane of the intestine and distension of the joints with a purulent or caseo-purulent material. A Salmonella, which was not identified, was isolated from the pus of the joints in the chronic cases and from the heart-blood of the young birds. This organism was found to be pathogenic for guinea-pigs, rabbits, mice and pigeons. On investigating the cause of a serious epizootic among a group of young squabs, Beaudette (1926 b) found typhi-murium in the heart blood, internal organs and unabsorbed yolk of the young birds. The sick birds showed nervous symptoms, like inco-ordination of movements and convulsions, and diarrhoea; the lesions were swelling of the liver and lungs, catarrhal enteritis and inflammation of the proventriculus. The birds had been kept under very unhygienic conditions, which were regarded as a predisposing factor.

Several outbreaks of paratyphoid in pigeons from widespread areas in Germany were studied by Beck and Meyer (1927). The cause was ascribed to typhi-murium (Breslau) and the disease affected old birds as well as young ones. The latter usually suffered from an acute form associated with loss of appetite, severe thirst, listlessness, diarrhoea and pneumonia. In older birds the symptoms were more sub-acute or chronic; there were symptoms of general weakness, involuntary drooping of the wings, paralysis of the limbs and swelling of the joints. The mortality was generally very high. A post-mortem examination revealed atrophy of the pectoral muscles associated with the presence of several elongated greyish-yellow nodules, and a yellow caseous exudate in the enlarged joints; there were also numerous small, necrotic nodules in the lungs and liver, and several ulcers on the mucous membrane of the intestine; the testes, ovary and oviduct were frequently affected, Beck (1929) considered that the etiological agent of pigeon paratyphoid resembled typhi-murium (Breslau)

serologically, and that adult birds were much less susceptible than young ones; the latter could be readily infected parenterally or otherwise. Berge (1929) regarded paratyphoid as one of the most important diseases of pigeons in Germany -- of 103 birds examined by him 22.6 per cent. were found to be infected with typhi-murium (Breslau). Young birds commonly suffer from an acute form of the disease with symptoms of progressive weakness, listlessness, loss of appetite, thirst, severe diarrhoea and pneumonia; the outstanding lesions are septicæmia, tumour splenis, disseminated necrotic nodules in the liver, acute enteritis, and pneumonia with several greyish-yellow nodules scattered throughout the lungs. In older birds the condition is generally chronic and the symptoms may last for several weeks; the joints are swollen and there is paralysis of the muscles of locomotion and flight; the carcass is usually emaciated, the pectoral and leg muscles are atrophied, there is swelling of the internal organs and numerous small necrotic nodules have developed on the mucous membrane of the small intestine; in cases of enlarged joints there is a purulent or caseous exudate in the joint cavity. Emmel (1929) also found Schottmuller (typhi-murium ?) in practically pure culture in the exudates obtained from the swollen joints of pigeons examined by him.

By examining a flock of over 8000 pigeons suffering from weakness of the wings and swelling of the joints Brunett (1930) found a straw coloured exudate in the joint swellings and abnormalities in the ovaries resembling those of pullorum disease. Typhi-murium was isolated from the joints as well as from the ovaries.

Recently Jungherr and Wilcox (1934) investigated the cause of a disease in a flock of about 1500 pigeons in which there was an annual loss of about 20 percent. They incriminated an atypical non-maltose fermenting variant of typhi-murium as the etiological agent. They found a widespread sensitisation to the variant in the breeding stock, and called attention to the misleading cross-agglutination which was

obtained between this organism and Pullorum, and therefore the possible diagnosis of bacillary white diarrhoea in unusual hosts. The lesions seen were tumour splenis, peritonitis and arthritis. In some cases typhi-murium could not be obtained from reacting squabs, while at other times the organisms were isolated from birds that failed to react serologically. Edwards (1935 b) studied the same variant from three widely separated areas and found the "O" antigen, like that of abortus equi, lacking in factor V of the Kauffmann-White schema. The variant was noticed to be non-maltose fermenting and negative to the Bitter test; it appeared to be similar to typhi-murium var. Copenhagen of Kauffmann (1935 a). Edwards labelled the organism S. typhi-murium var. Storrs. A similar organism, obtained from a case of purulent arthritis in a foal, is described by me above.

Lesbouyries and Verge (1932) described pigeon paratyphoid in France and Cernaiianu and Popovici (1933) in Rumania, while Ismail Abu Bakr Khalifa (1935) studied an epizootic in Egypt due to typhi-murium. More recently Shirlaw and Ganapathy Iyer (1937) have recorded an outbreak of pigeon septicaemia in India caused by what they called a "Gaertner infection". Soon after a number of birds had been inoculated with fowl-pox vaccine they developed symptoms of acute enteritis and fever from which they died. It is not possible to recognise the type of Salmonella incriminated from the description given.

That infection of pigeons with Salmonella may lead to serious outbreak of food-poisoning in men is illustrated by the description of Clarenburg and Dornickx (1932) of an epizootic which involved 20 persons in the military hospital at the Hague. The source of the infection was traced to pudding made largely from pigeons' eggs. The patients showed symptoms of fever, diarrhoea, vomiting and

gastro-enteritis. S. typhi-murium was isolated from the pudding, and from the blood, faeces and urine of some of the patients; the sera of the affected persons also agglutinated cultures of the Salmonella found. On investigation, it was ascertained that the flock of pigeons from which the eggs originated were suffering from paratyphoid. Moreover, typhi-murium was recovered from eggs laid by these birds.

Although several outbreaks of a Septicaemic disease in pigeons have been reported in South Africa from time to time the cause has remained obscure until recently when Henning and Haig (1938) studied an epizootic of squabs in which a Salmonella was found to be the cause. (The outbreak was studied after the completion of this paper).

The affected birds suffered from loss of appetite acute diarrhoea with green evacuations and rapid loss of condition. The most important lesions observed were enlargement of the spleen and liver and acute catarrhal enteritis. The affected flock was composed of over 200 birds of which 24 have died from the disease. Heart-blood, spleen and liver cultures yielded a pure growth of a non-lactose fermenting, Gram-negative motile bacterium (culture 548). On testing this bacterium against various "O", type and group sera of different groups of Salmonella, it was agglutinated by "O" sera containing factor IV of the Kauffmann-White Schema, by type sera containing factor 1 and by group sera. This suggested that the organism is related to typhi-murium. Agglutination and absorption tests were, therefore, performed with different varieties of typhi-murium. The results are given in Table 26 B.

The results of Table 26 B show that typhi-murium absorbed all the agglutinins, "O" type and group, from

548 serum as well as from its own serum, but that culture 548 merely reduced the "0" titre of typhi-murium serum from 3200 to 1600. Culture 548 removed all the "0" agglutinins from its own serum but failed to exhaust a small portion of type and group agglutinins from both its own and typhi-murium serum. This is attributed to the reduction of its motility which occurs on subcultivation on solid agar.

When 548 serum was absorbed with either typhi-murium var. Storrs or typhi-murium var. copenhagen. All the "0" agglutinins were removed for typhi-murium, typhi-murium var. Storrs, typhi-murium var. Copenhagen and for itself.

According to these results, therefore, culture 548 contains the same type and group antigens as typhi-murium and the same "0" antigen as typhi-murium var. Copenhagen (Storrs). Its antigenic formula should be "0" = IV, type 1 group 1,2,3.

An outbreak of pyo-arthritis in foals caused by the IV-variant of typhi-murium is described on page 73.

TABLE 26 B.

Antigen	548 serum unabsorbed	<u>Typhi-murium</u> serum unabsorbed	548 serum ^x absorbed by 548	548 serum absorbed by <u>typhi-murium</u>	<u>typhi-murium</u> absorbed by 548	548 serum ^x absorbed by <u>typhi-murium</u> var. <u>Storrs</u> .	548 serum absorbed by <u>typhi-murium</u> var. <u>Copenhagen</u> .
548 "O"	1600	3200	0	0	0	0	0
548 type	6400	100,000	100	0	100		
548 group	100,000	50,000	200	0	200		
<u>typhi-murium</u> "O"	1,600	3,200	0	0	1,600	0	0
" type	6,400	100,000	100	0	100		
" group	100,000	25,000	200	0	200		
<u>typhi-murium</u> var. <u>Storrs</u> "O"	1,600					0	0
<u>typhi-murium</u> v. <u>Copenhagen</u> "O"	1,600					0	0

^x Strain 548, although undoubtedly mobile, lost a great deal of its motility when it was grown on saliva agar. It, therefore, failed to absorb all the "H" agglutinins from its own serum as well as from typhi-murium serum.

(2) Canaries.

Canaries seem to be particularly susceptible to Salmonella infection. They usually contract a very virulent form of the disease which may account for very severe losses in both young and adult birds. Joest (1906) was probably the first to draw attention to the occurrence of a disease in canaries caused by the enteric group of bacteria. Another early record of an epizootic apparently due to a Salmonella is that of Gilruth (1910). Apart from symptoms of drowsiness and listlessness, the birds did not appear to be sick; but death was often sudden, with tumor splenis as the most important lesion. A bacterium isolated from the heartblood was found to be pathogenic for mice, rabbits, guinea-pigs and canaries. About the same time Pfeiler (1911) incriminated an organism of the Paratyphi-B group, obtained from blood culture, as the cause of a virulent outbreak of diarrhoea among a group of well-bred canaries. After death, lesions of acute enteritis, peritonitis and tumor splenis were revealed. A somewhat similar outbreak was recorded by Lutje (1924).

Later Beaudette (1926 a), Beaudette and Edwards (1926), and Harkins, (1926) also described virulent epizootics in canaries in which organisms of the Paratyphoid -B group were incriminated as the etiological agent. Beaudette and Edwards (1926) studied two outbreaks in which birds of all ages were affected with severe diarrhoea associated with an increase in the amount of urates excreted; an organism which

resembled typhi-murium serologically was obtained from the heart-blood and internal organs. The outbreak described by Harkins (1926) involved over 200 imported canaries with a mortality of over 75 percent. The premises into which the birds were introduced were well-kept and clean and none of the local birds became affected; but the imported canaries arrived in soiled wooden cages, which probably played a predisposing part in setting up the infection. The sick birds showed symptoms of listlessness, inappetance and diarrhoea, and death was always very rapid.

In South Africa, Martinaglia (1929) recorded two outbreaks of paratyphoid in canaries in which typhi-murium was the cause, and in 1933 I investigated a very virulent epizootic in an aviary comprising about 200 well-bred canaries. The most important symptoms were drowsiness and diarrhoea, and the course of the disease was always very rapid, with a mortality of over 95 per cent. The most important lesions observed were hydro-pericardium, enteritis, tumor splenis and swelling of the liver. A gram-negative, non-lactose fermenting organism was obtained in pure culture from the heart-blood and spleen of all the birds examined. Cultures of this organism were readily agglutinated by typhi-murium serum and the organism was found to be di-phasic. A mixed serum was prepared by injecting a rabbit five times with a suspension of the canary strain (culture 176) in saline. Agglutination and absorption tests were then performed as shown in Table II.

TABLE II.

Antigen	<u>Typhi-</u> <u>murium</u> S. a.b. <u>typhi-</u> <u>murium.</u>	<u>Typhi-</u> <u>murium</u> S. a.b. 176	<u>Typhi-</u> <u>murium</u> S.un. a-b.	176 S. a.b. <u>typhi-</u> <u>murium</u>	176 S. a.b. 176	176 S. un.
Typhi-murium "O"	0	0	1600	0	0	800
Typhi-murium "H" type	0	0	50000	0	0	12800
Typhi-murium "H" group	0	0	25000	0	0	6400
176 "O"	0	0	1600	0	0	800
176 type	0	0	50000	0	0	12800
176 group	0	0	25000	0	0	6400

S = serum ; a.b. = absorbed by; unab = unabsorbed;

0 = no agglutination at 1 in 100.

The results of Table II show that culture 176 removed all agglutinins ("O", type and group) from typhi-murium serum as well as from 176 serum, while typhi-murium completely exhausted both 176 serum and the homologous serum. Culture, 176, therefore, resembles typhi-murium serologically and contains the same antigenic factors; it should be regarded as ~~a strain of~~ typhi-murium.

On investigating the source of the infection, I found that losses commenced to occur soon after the owner had changed the food supply. Several samples of grain used by the owner for feeding were obtained and inoculated into enrichment media e.g. tetrathionate broth. After 24 hours incubation a loopful from each tube was spread onto a Mason tube of MacConkey's bile-salt agar. A few translucent, non-lactose fermenting colonies were observed on one of the tubes; some of these were picked and tested against drops of a typhi-murium serum dilution on a glass slide; the result was a coarse floccular agglutination and typhi-murium was suspected. The remainder of ^{two of} ~~a couple of~~ the positive colonies ^{was} ~~were~~ subcultivated until a pure culture (culture 177) was obtained. Culture 177 was found to be diphasic, and, like

culture 176, it exhausted all agglutinins from the serum of culture 176, as well as from the serum of typhi-murium Glasgow. Accordingly, culture 177, like culture 176, should be regarded as a strain of typhi-murium. A one-sided absorption was considered sufficient in the case of this test.

Although the presence of typhi-murium in the grain may explain the origin of the infection, the possibility of the grain becoming contaminated by attendants handling it after the outbreak among the canaries cannot be excluded. It may be of interest to mention that Jones and Wright (1938) described an outbreak of typhi-murium food-poisoning in man due to contamination of food with the excrete of mice.

Culture 153, obtained from one of a number of finches that were dying from a septicaemic disease in an aviary, was also studied. By testing it with the same method used for culture 176 it was found to be diphasic and to exhibit the same antigenic characters as cultures 176 and 177. It was, therefore, also labelled typhi-murium.

(3) Geese.

Outside Germany there is very little information available regarding the incidence of paratyphoid infection in geese. According to the classical monograph of Hubener (1910) seventeen outbreaks of food-poisoning traced to birds' meat have been recorded in Germany during the period 1903 to 1908; of these 14 outbreaks were due to goose meat, one to duck, one to fowl and, in the case of one, the species of bird was not mentioned. Nine of the outbreaks were ascribed to paratyphoid organisms; of these, eight were caused by goose meat and one was due to the meat of a sick hen.

One of the first records of paratyphoid in geese is that of Pfeiler (1919) when he described a virulent epizootic among 9-week old birds with symptoms of septicaemia and swelling of the head and eyes. Heart-blood and organ cultures yielded a pure growth of an organism of the Paratyphi-B group (typhi-murium?). Earlier in the year cultures of typhi-murium were used for the eradication of mice and there

was a suspicion that geese obtained the infection from the mice. Later Weissgerber and Müller (1922), Lutje (1924) and Burghoffer (1927) described similar epizootics among young geese. The geese studied by Weissgerber and Müller manifested symptoms of diarrhoea, swelling of the eyelids, weakness and convulsions followed by death. The lesions recorded were swelling of the liver and spleen, hæmorrhagic enteritis, epicarditis and hydropericardium. An organism *which resembled both* ~~thought to be partly resembling~~ Paratyphus-B and partly suipestifer was obtained from the heart-blood and organs of affected birds. Very heavy losses were recorded by Lutje among goslings that showed symptoms of fever, listlessness, diarrhoea with lesions of tumor splenis, enteritis, and miliary necrotic nodules in the liver; a slow-growing Salmonella, that was agglutinated to high titre by both Schottmuller and Voldagsen sera, was isolated from the internal organs. Burghoffer investigated a septicaemic disease among 1 and 2 week old goslings and incriminated Bact. enteritidis Breslau (typhi-murium) as the causal agent. Apparently the young birds became infected after hatching as the blood of the laying hens gave a negative serological test with typhi-murium. Experimentally the bacterium isolated was found to be pathogenic for very young geese only, birds from 4 to 6 weeks old being completely refractory to artificial infection.

After the recognition of members of the genus Salmonella as etiological agents of disease in geese, several outbreaks of food-poisoning in man have been traced to goose meat or even to goose eggs. Thus Hohn and Becker (1927) reported a number of outbreaks of food-poisoning in man where foodstuffs, like salads and sausages, which contained either goose eggs or goose meat as ingredients, were incriminated. The symptoms in some of the cases resembled those of typhoid fever, while other cases were typical of typhi-murium infection with vomiting and diarrhoea as the chief symptoms. There were two deaths, and typhi-murium was recovered from some of the suspected salads, as well as from the stools of the

patients. Baars (1929) also found typhi-murium as the cause of a disease in 12 persons that had partaken of some smoked goose breast. Symptoms of diarrhoea, vomiting and colic, lasting from 1 to 10 days, were shown by all the patients. The organisms were isolated from the stools of the patients as well as from the suspected meat.

Later Baars (1931) described another outbreak of meat-poisoning in a family of three due to Breslau infected goose meat. The meat was preserved in brine for a week before it was used. On investigating, ^{he} it was discovered that the goose from which the meat was obtained originated from the same farm as the birds that were responsible for the previous outbreak. It was thought that the infection was introduced onto the farm by means of a number of geese imported from Poland; these geese were suffering from gastro-enteritis when they arrived. Smoking and salting of the meat did not destroy the organisms, but rather caused their enrichment. Baars considered that freshly cooked or fried meats are less dangerous as the organisms are not very resistant to high temperatures.

Two outbreaks were recorded by Pressler (1930); the one involving four persons after a meal of pies that contained goose liver; Breslau (typhi-murium) was recovered from the stools of the patients and from what remained of the goose liver, but no infection could be detected in any of the remaining geese of the flock or in the persons that had handled the meat. The other outbreak affected a number of adults and a few children in a "Kinderheim"; they had eaten pies made from goose meat. During the same year Kolbe (1930) also described two epizootics of meat-poisoning resulting from the ingestion of goose meat; symptoms of vomiting, diarrhoea, fever and body pains set in about 12 hours after the meal, and typhi-murium was incriminated as the cause.

On account of the increase in the number of cases of gastro-enteritis in man traced to goose meat, the carcasses

of all suspicious-looking birds are now seized and condemned for human food in Germany. Out of 87 condemned carcasses of geese that had been suffering from fowl cholera, Hüsgen (1931) obtained typhi-murium from 11 and enteritidis from 1. Transportation of the birds was considered to reduce their resistance so that infection could readily have taken place. Hüsgen also reported two outbreaks of food-poisoning due to goose liver and meat infected with typhi-murium, and in 1930 he investigated a severe epizootic of paratyphoid in geese. The outstanding symptoms described in the geese were listlessness and diarrhoea, associated with numerous necrotic nodules in the liver.

During three months of 1932 Wundram and Schönberg (1932) examined 182 goose carcasses in Berlin and isolated typhi-murium from 44. The affected birds were emaciated and showed marked pathological changes in their internal organs, and their skins were reddened. The same workers also reported 6 outbreaks of food-poisoning, involving 16 persons, caused by goose meat and liver infected with typhi-murium. About the same time Bornstedt and Fiedler (1932) examined 828 geese imported from Poland and Lithuania; of these 182 had died and showed either lesions of fowl cholera or verminosis. Of 144 sick geese suffering either from transport injuries or symptoms of fowl cholera, 12 gave a positive agglutination reaction with typhi-murium; from the faeces of five of these S. typhi-murium was isolated. They suggested that S. typhi-murium probably occurs as a saprophyte in the bodies of geese, becoming invasive only when the animal's resistance has been lowered by factors like disease, injury and transportation.

As far as South Africa is concerned no cases of geese infected with Salmonella have so far been recorded.

(4) Ducks.

From the public health aspect Salmonella infection in ducks is particularly dangerous because the organisms may occur in the eggs of infected birds as well as in the meat.

Moreover, paratyphoid is far more common in ducks and geese than in gallinaceous birds; Lecoq (quoted by Scott, 1930) considered the constant association of water birds with ponds and mud pools, which are sometimes contaminated with infected excreta, as the cause of the frequency of disease in them. In his account of paratyphoid infection in aquatic birds, Manning (1918) described a disease in 1 to 3 weeks old ducks and geese caused by an organism of the Paratyphus-B group. The main symptoms recorded were drowsiness, loss of appetite, a purulent conjunctivitis, septicaemia and enteritis; death occurred in 2 to 3 days' time and the lesions observed were tumor splenis, sometimes necrotic foci in the liver and spleen, enteritis and pericarditis. Soon afterwards Rettger and Scoville (1920) investigated a most virulent disease ("keel") in ducklings; there was a mortality of nearly 100 percent in a flock of about 3000, death usually occurring during the first week of life, but occasionally as late as 3 to 4 weeks after hatching. The only symptoms observed were listlessness, weakness and intense thirst; after drinking the ducklings drew themselves up, staggered, "keeled" and died. There were no definite lesions, but a Salmonella was readily obtained from the heart-blood and organs of the young birds and also from the ovaries of two adult ducks and the abdominal cyst of one. The investigators considered that the infection was probably transmitted from the ovaries of diseased hens through the egg to the chick, and they named the organism isolated Bacterium anatum, a new species. But Cooper and Brumwiede (1924), Edwards and Rettger (1924, 1927), and Kauffman and Silberstein (1934) found that only some of the strains of Salmonella labelled anatum could be included under the new name as the others resembled typhi-murium serologically; actually one of the strains studied by Kauffmann and Silberstein (1934), strain 3123 of the National Collection of Type Cultures, was found to be enteritidis. Anatum like most other species of Salmonella, however, ~~does not~~ ^{at} affect ^{more than} only one species of animal. Thus, Kauffman and Silberstein isolated a strain from the stool of a patient suffering from gastro-enteritis and

intermittent fever, and another strain (anatum var Huenster) from a person that had developed meat-poisoning after eating raw horse meat; they also described a third strain of human origin obtained from Kristensen. Edwards (1935^a) incriminated anatum as the etiological agent of an epizootic in chickens, and I (vide infra) isolated it from adult fowls.

Subsequently several different workers have recorded epizootics in ducks due to Salmonellas. Doyle (1927) recorded a severe outbreak among chicks and young ducks due to typhi-murium; the source of the infection remained obscure, but the food was suspected. In 1929 Gaiger and Davies (1930) investigated the first known outbreak of "keel" disease in Great Britain. The disease was so virulent that over 4000 ducklings from 2 to 17 days old were lost. The main symptoms manifested were loss of appetite, catarrh of the nose and eyes, incoordination of movements and loss of equilibrium; finally the ducklings fell down unable to rise, "keeling" with their legs in the air. There was a mortality of over 80 per cent. and the recovered birds remained ailing for several weeks. Anatum was obtained from a number of the birds examined. All the deaths occurred on a farm to which the young ducks were moved after hatching, while those that remained behind on the breeding farm remained healthy. It was apparent, therefore, that the infection took place after hatching and that the eggs and incubators were clean. It was suggested by Gaiger and Davies that anatum, which they regarded as a common inhabitant of the alimentary canal of ducks, is normally not pathogenic for these birds, but that it becomes pathogenic only when certain predisposing factors operate. Fermentation of the food was regarded as an important contributory factor in the genesis of the disease in the outbreak under consideration.

Pallaske (1930) described a disease in ducks associated with pathological changes in the ovaries of hens and the testes of drakes; the cause was found to be S. enteritidis Gaertner. Hole (1932) encountered three epizootics in young

ducklings, due to enteritidis and the other two to typhi-murium; infection was thought to have occurred through the egg. Acute and sub-acute enzootics in young ducks and geese with a mortality of 96 per cent were described by Strozzi (1931). Another virulent epizootic in ducklings with a death-rate of over 90 per cent. was recorded by Schaaf (1933). The symptoms manifested were loss of appetite, diarrhoea, with soiling of the cloacal feathers, swelling of the eye-lids and a purulent conjunctivitis. There were lesions of tumor splenis, swelling of the liver, enteritis and septicaemia; the yolk-sacs were about as large as a pea and contained a yellowish mortar-like material. Typhi-murium was found to be the cause. Infected birds discharged the organisms with their faeces and gave positive agglutinations reactions with these bacteria. Natural infection was thought to have resulted from the ingestion of food or water contaminated with infected excreta. Moreover, the vitality and resistance of the birds were considerably reduced by transportation for long distances by rail.

In England Dalling and Warrack (1932), McGaughey (1932), and Warrack and Dalling (1933) have shown that adult ducks may sometimes harbour S. typhi-murium or S. enteritidis, and that breeding birds with diseased ovaries are liable to lay infected eggs, which often fail to hatch; should the infected eggs hatch an epizootic of paratyphoid will probably occur among the newly-hatched birds. In this disease, therefore, as in Bacillary White Diarrhoea, the infecting agent is transmitted from the adult bird through the egg to its progeny. The presence of Salmonellas in the eggs laid by infected birds was demonstrated by these workers. Moreover, those ducks which laid eggs infected with either typhi-murium or enteritidis produced the corresponding agglutinins in their sera, and, as with pullorum infected hens, they could usually be detected by means of a serological test. Warrack and Dalling noticed that the eggs/^{laid}were infected only when the titre of the affected bird was high, and the agglutination titre of the sera

obtained from reactors dropped considerably during the course of the laying season. In the outbreak investigated by McGaughey, several deaths occurred among adult ducks during the course of months. The liver and ovary of one bird, which showed lesions resembling those of Fullorum disease, yielded enteritidis on culture.

But healthy ducklings may acquire the infection from outside sources, e.g. infected eggs may introduce the infection into the incubator and so produce the disease in subsequent hatchings. Moreover, the infection may also be picked up from contaminated soil, food or water.

Scott (1930) considered that eggs may be responsible for many mysterious cases of Salmonella food-poisoning in which none of the common articles could be incriminated. He mentioned seven outbreaks where duck's eggs were suspected, but not proved, to be the cause of the disease, and he alluded to a monograph of Lecoq (1906) in which several outbreaks of bacterial food-poisoning due to whipped cream were described; both duck's and hen's eggs were used as ingredients of the whipped cream. By dipping fresh eggs into a culture of typhi-murium, Scott showed that infection was ^{might pass} possible through the shell, provided the eggs were kept in the room for at least two weeks; both yolk and albumen became infected. But he found that part of the shell must remain moist for the penetration of the bacteria; if the culture was allowed to dry on the shell, infection failed. The bactericidal action of fresh albumen prevented growth, but, as the eggs became stale, the multiplication of the Salmonella was marked and the eggs became badly infected. The infected eggs showed no outward sign of infection and might have been mistaken for normal eggs.

Later Scott (1932) described three widely-separated outbreaks of acute gastro-enteritis in man due to eggs infected with typhi-murium; there was one death. The organisms were recovered from the stools of a number of patients and from the organs of one. Duck's eggs, fried and raw, were

imputed and the suspicion was confirmed by the discovery of typhi-murium infected eggs from the corresponding flocks. The infected birds were recognised by means of serological tests and typhi-murium was isolated from the spleen, ovary, oviduct and intestines of some of the reactors.

Since the discovery by Scott and Dalling and Warrack of the transmission of Salmonella infection by means of duck's eggs several cases have been revealed where foods containing infected duck's eggs as ingredients have been incriminated as responsible for outbreaks of food-poisoning in man. Thus, Fromme (1933) and Willführ, Fromme and Bruns (1933) described 25 outbreaks of gastro-enteritis in Germany, traced to duck's eggs infected either with typhi-murium or enteritidis; there were 143 cases and 2 deaths. In three of the outbreaks Salmonellas were discovered in the food, and in one it was possible to isolate typhi-murium from the faeces of two ducks and from the egg-shells of another. Furth and Klein (1933) recorded two epizootics of food-poisoning in large homes caused by vanilla pudding and potato salad containing duck's eggs as ingredients; altogether 140 cases were involved. In one outbreak typhi-murium, and in the other Gaertner bacilli, were isolated from the stools of the patients. The faeces of some of the ducks, from which the eggs for one of the establishments originated, yielded aertrycke on cultivation, but the examination of the contents of over a hundred eggs from a suspected flock failed to yield Salmonellas. These organisms were, however, obtained from the shells of three of the eggs examined. It was, therefore, thought that the infection was produced by the bacteria present on the shells. Müller and Rodenkärcken (1933), on the other hand, obtained Gaertner bacilli in pure culture from the contents of the remainder of a consignment of duck's eggs, some of which had been used in the raw state for a potato salad, were responsible for an outbreak of food-poisoning.

On investigating the cause of an epizootic of gastro-

enteritis among a number of guests at a wedding party on a farm in Germany, Mieszner and Köser (1934) found that all the patients had partaken of a pudding made from duck's eggs. From the ovaries of two ducks owned by the host, from an egg laid by one and from the faeces of another, typhi-murium was isolated. During the period 1931 to 1934 Bruns and Fromme (1934) studied 50 outbreaks of food-poisoning in Western Germany caused by foods containing duck's eggs, prepared mostly in the form of mayonnaise. There were 253 cases and 6 deaths, and either typhi-murium or enteritidis was incriminated. Zeug (1935) also drew attention to the increasing prevalence of food-poisoning in the industrial areas of Western Germany due to foods prepared from duck's eggs; mayonnaise, potato salads, puddings and Hackfleisch were most frequently responsible. Typhi-murium was regarded as the chief cause. Zeug has pointed out that, although no definite clinical symptoms may be observed in the birds that lay infected eggs, egg-laying generally decreases, and pathological changes develop in the ovaries and oviducts. Salmonellas are usually present in these lesions, from which they find their way into the interior of the egg. But infection sometimes occurs by contamination of the egg-shell with infected faeces. As shown by Scott (1930 - vide supra), Salmonellas may penetrate through the shell into the interior of the egg, under certain conditions. If the shell-contaminated eggs are soon cooked, no harm is likely to result; but should they be kept for some time (~~e.g. in the shop~~), serious infection may follow their use. The heating to which eggs are generally subjected is not enough to destroy the organisms present in an infected egg. After 5½ minutes boiling infected eggs may still contain live organisms, but 6 minutes boiling is usually sufficient to kill all the bacteria.

Clarenburg and Pot (1935) also described a severe outbreak of gastro-enteritis in 4 families, involving 9 persons. Symptoms of diarrhoea, vomiting and fever appeared

soon after the people had eaten cream puffs supplied by the same baker. Typhi-murium was isolated from the cream puffs, and from the stools and urine of some of the patients. Duck's eggs were used as ingredients of the puffs, but all the eggs examined from the suspected ducks gave negative results for Salmonella. Six of the ducks, however, gave positive serological tests for typhi-murium, and this organism was isolated from the faeces of one. When the reacting ducks were killed, they showed lesions of chronic oophoritis, and from the ovaries of two of them typhi-murium was obtained in pure culture. Similar bacteria were also isolated from apparently normal looking yolks present in the ovaries.

Recently epizootics in ducks, due to infection with either Gaertner or typhi murium, have been observed fairly frequently in Holland, where the disease has been studied by a number of investigators, especially by Jansen (1934a, 1934b 1935, 1936). From an area badly infected with tuberculosis he obtained about 100 ducks for examination; 34 of these were infected with tuberculosis and 6 showed lesions of chronic oophoritis with either enteritidis or typhi-murium present in the ovaries. A number of birds were infected with both tuberculosis and paratyphoid. In a virulent outbreak among young ducklings with lesions of enteritis and swelling of the liver, he isolated enteritidis bacilli of the Moscow type from the internal organs of affected birds. By testing a number of suspected birds serologically, he found a few affected with oophoritis in which the reaction was negative, though in some positive cases there was no evidence of oophoritis, Generally, however, the ovary was affected when a positive reaction had been obtained.

Jansen has also noticed that a large percentage of ovary-infected ducks lay infected eggs, which frequently cause epizootics of paratyphoid among newly hatched ducklings. But he has also recorded a number of outbreaks in young birds where the eggs could not be incriminated.

In five outbreaks studied by Jansen in 1936, three

were found to be due to enteritidis var. Essen, one to typhi-murium and one to a mixture of the two organisms. In the latter case typhi-murium was obtained from the heart-blood liver and yolk of the young birds, while a small percentage of the adults was infected with essen as well as typhi-murium. In one of the outbreaks no losses occurred during the first few weeks of life, but at about three weeks several acute cases of enteritis set in, a number of which ended fatally; infection through the egg can, therefore, be excluded. The other four epizootics probably occurred through the egg, as the birds started dying during the first few days of life. In the cases caused by typhi-murium there were no outstanding lesions, apart from degeneration and yellow discoloration of the liver; there was a certain amount of unabsorbed yolk in some of the birds, and organisms were obtained from the heart-blood, liver and yolk. The essen infected birds also did not show any marked pathological changes; the liver was coloured yellow, the yolk was partly or completely absorbed and the organisms were isolated from the internal organs.

The importance of Salmonella infection in both ducks and geese in Germany was also emphasised by Lerche (1936). He found 5.7 per cent of the duck's eggs offered for sale to be infected, and he described an outbreak of food-poisoning in a family that had eaten fried duck's eggs. Typhi-murium was isolated from the stools of the patients and from the eggs.

I have not had an opportunity of studying Salmonella infection of ducks in South Africa, but in 1931 Dunning (1934) investigated a virulent epizootic of ducklings in the Cape Peninsula. At least 50 per cent of a flock of 2000 birds died at ages varying from 5 to 23 days. "Keel" disease was tentatively diagnosed. Fourteen newly hatched ducklings taken from the infected farm were removed to fresh, clean premises and kept under observation. All died from 5 to 29 days after hatching. A bacillus obtained in pure culture from

the organs of affected birds was tested biochemically and was found to react like S. enteritidis. The evidence collected by Dunning suggested that the eggs were infected at the time they were placed in the incubator; the owner of the infected farm (Farm A) sold 6 dozen eggs to another farmer living on Farm B (for breeding purposes), with the exception of a few all the birds that were hatched out from these eggs died from a disease that resembled the one on Farm A. Some time after the outbreak Dunning found that several of the surviving ducklings on Farm A remained weak and unthrifty, apparently suffering from a chronic form of the disease.

Early in 1932 Coles also investigated a virulent epizootic of ducklings in the Transvaal, and isolated a Gram-negative, non-lactose fermenting bacterium from the affected birds. Fermentation tests carried out with this organism resembled those obtained with typhi-murium.

As the cultures made from the organisms isolated from both outbreaks were discarded serologically tests could not be performed.

(5) Turkeys.

About 45 years ago MacFadyean (1893) described a disease in turkeys which he called "epizootic pneumo-pericarditis". The main lesions mentioned were marked pericarditis and croupous pneumonia, and numerous short, motile bacteria were found in the pericardial fluid, heart-blood, spleen and lungs. Cultures of this organism were pathogenic for turkeys and rabbits, while guinea-pigs and pigeons were more resistant; fowls and calves appeared to be unsusceptible. The organism described by MacFadyean is probably a Salmonella, and the outbreak of "pneumo-pericarditis" caused by it the first record of paratyphoid among turkeys. The etiology of the disease "pneumo-enteritis", described by Dodd (1905), is less apparent. The organism incriminated was a non-motile bacterium of the "fowl-cholera" type, obtained in pure culture from the heart-blood and lungs. In South Africa, Jowett

(1908) investigated a highly fatal disease in turkeys, which he also called "pneumo-pericarditis" after the condition described by MacFadyean; pericarditis and pneumonia were also the most important lesions, and a "bi-polar staining" bacterium was isolated from the heart-blood, pericardial fluid and internal organs of affected birds. Cultures of this organism proved to be pathogenic for turkeys and guinea-pigs, but not for fowls. It is highly probable that Jowett was dealing with an outbreak of paratyphoid. However, the first authentic record of an epizootic in turkeys, in which a Salmonella was recognised as the causal agent, is that of Pfaff (1921). Apart from listlessness and loss of appetite, the birds did not show any definite symptoms of disease, and death usually occurred within a day or two. The following lesions were observed:- pericarditis, hydropericardium, tumor splenis, greyish-yellow necrotic nodules in the heart muscle and lungs, and patchy swelling of the intestinal mucosa. A pure culture of a paratyphoid-like organism was isolated from the heart-blood and pericardial fluid. Cultures of this bacterium proved to be pathogenic for turkeys and several small laboratory animals.

Later, several other investigators studied outbreaks of paratyphoid in turkeys. Rettger, Plastridge and Cameron (1933) investigated outbreaks of recurrent deaths among young poults on two different farms; the greatest losses occurred among birds that were less than 10 days old, but deaths were also observed as late as 6 weeks after hatching. The affected poults crowded together as if they were chilled, and many that appeared quite normal in the evening were found dead in the morning. Diarrhoea was the most common symptom, and after death lesions of enteritis and swelling of the liver were observed; in many cases the caecal contents were of a cheesy appearance. A pure culture of typhi-murium was obtained from the heart-blood and internal organs and it was thought that the unhygienic conditions under which the birds were kept

on the one farm accounted for the ease with which the disease became established; infected poultts probably conveyed the disease from this farm to birds that were kept on an apparently clean farm.

According to Lee, Holm and Murray (1936) no serious losses were known to occur in turkeys in the State of Iowa prior to 1934. In May of that year a very virulent disease, with a mortality of over 90 per cent., appeared in young poultts under 5 weeks old. The birds were unthrifty, weak and drowsy and sometimes suffered from diarrhoea. The lesions were inconstant, the most common ones being hydropericardium, swelling of the liver, hyperplasia of the bone-marrow and enteritis with caseous caecal contents. A pure culture of typhi-murium was obtained from the heart-blood and internal organs. More recently Cherrington Gildow and Moore (1937) investigated four outbreaks of typhi-murium infection among poultts in widely separated areas. In three of the outbreaks the disease appeared before the birds were a week old, suggesting that the infection was probably transmitted, like pullorum disease, from infected hens through the eggs to the poultts. A large percentage of the hens that produced diseased poultts gave positive agglutination reactions with typhi-murium. The most important symptoms and lesions were weakness, emaciation, pneumonia, watery or coagulated yolk-sacs, occasionally enteritis with a cheesy material in the caeca; the liver was yellow or mottled and the kidneys pale; there was a mortality of over 80 per cent, among the poultts under 10 days of age. In one outbreak typhi-murium was isolated from some dead-in-the-shell poultts, and in another from the ovaries and yolk of some of the reacting hens; in some cases, however, no organisms could be cultivated from the abnormal ovaries of reacting hens.

But outbreaks of paratyphoid in turkeys may be caused by Salmonellas other than typhi-murium. Edwards(1937) has described an epizootic in poultts due to S. senftenberg,