organs of affected birds, and a slow-growing *Salmonella* that was agglutinated to high titre by both *Schottmuller* and *Voldagsen* sera, was isolated by Lutje from the internal organs of diseased birds. 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. Baars (1929) also found *typhi-murium* as the cause of a disease in 12 persons that had partaken of some smoked goose breast. 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 investigation, he 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. 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.

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.

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, Manninger (1918) described a disease in 1 to 2 weeks old ducks and geese caused by an organism of the *Paratyphus-B* group. Soon afterwards Rettger and Scoville (1920) investigated a most virulent disease (" keel ") in ducklings; there was a mortality of nearly 100 per cent. in a flock of about 3,000, death usually occurring during the first week of life, but occasionally as late as 3 to 4 weeks after hatching. 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 Krumwiede (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 Silbertstein (1934), strain 3123 of the National Collection of Type Cultures, was found to be *enteritidis*. *Anatum* like most other species of *Salmonella,* however,

affects more than one species of animal. Thus, Kauffmann and Silbertstein isolated a strain from the stool of a patient suffering from gastro-enteritis and intermittent fever, and another strain (*anatum var. Muenster*) 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. 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. Fermentation of the food was regarded as an important contributory factor in the genesis of the disease in this outbreak.

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, one 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 Strozze (1931). Another virulent epizootic in ducklings with a death-rate of over 90 per cent. was recorded by Schaaf (1933). *Typhi-murium* was found to be the cause. Infected birds discharged the organisms with their faeces and gave positive agglutination 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 over 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 *pullorum* 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 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 and hen eggs were used as ingredients of the whipped cream. By dipping fresh eggs into a culture of *typhi-murium*, Scott showed that infection might pass 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 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 eggs several cases have been revealed where foods containing infected duck 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 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 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 Rondenkircken (1933), on the other hand, obtained *Gaertner* bacilli in pure culture from the contents of the remainder of a consignment of duck eggs, some of which had been used in the raw state for a potato salad and 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 found that all the patients had partaken of a pudding made from duck 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 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 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, 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 $5\frac{1}{2}$ 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 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 Jansen (1934a, 1934b, 1935, 1936). 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*.

The importance of *Salmonella* infection in both ducks and geese in Germany was also emphasised by Lerche (1936), who found 5·7 per cent. of the duck's eggs offered for sale to be infected. He described an outbreak of food-poisoning in a family that had eaten fried ducks 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 2,000 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.

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, serological 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 organism obtained by MacFadyean from the heart-blood, spleen and pericardium is probably a *Salmonella*, and the outbreak of "pneumo-pericarditis" caused by it is 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. Cultures of the organism isolated from the heart-blood and pericardial fluid proved to be pathogenic for turkeys and guinea-pigs, but not for fowls. It is highly probable that Jowett was also 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). A pure culture of a paratyphoid-like organism was isolated from the heart-blood and pericardial fluid of diseased birds. 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. 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.

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 poults under 5 weeks old. A pure culture of *typhi-murium* was obtained from the heart-blood and internal organs of affected birds. More recently Cherrington Gildow and Moore (1937) investigated four outbreaks of *typhi-murium* infection among poults 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 poults. A large percentage of the hens that produced diseased poults gave positive agglutination reactions with *typhi-murium*. There was a mortality of over 80 per cent. among the poults under 10 days of age. In one outbreak *typhi-murium* was isolated from some dead-in-the-shells poults, 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 poults due to *S. senftenberg*, the fist record in which this organism has been incriminated as the cause of an animal disease. Moreover, infection of turkeys with *S. gallinarum* is comparatively frequent. Two of the 149 outbreaks caused by this bacterium and recorded by me (*vide infra*) involved turkeys only.

(6) Fowls.

Virulent epizootics, like fowl-typhoid and bacillary white diarrhoea, are so common in gallinaceous birds that it is quite possible that some outbreaks due to other types of *Salmonella* have been mistaken for these diseases. In many outbreaks of paratyphoid the scourge affects only very young birds in the same way as *pullorum* disease attacks eggs laid by infected hens, while in other epizootics older birds also suffer severely, and the disease resembles fowl typhoid. The nature of the malady is revealed only when a careful bacteriological examination of the dead birds is made.

One of the first records of a disease that can be interpreted as paratyphoid in fowls is the description by Mazza (1899) of an epizootic among these birds in Italy. A motile, non-indol forming, glucose fermenting organism obtained from the internal organs was found to be pathogenic for fowls and pigeons, but not for rabbits.

But there are very few early descriptions of paratyphoid in fowls, probably on account of the marked resistance of adult birds to infection. Reinholdt (1912) and others have tried to infect fowls, geese, ducks and pigeons with *enteritidis* and *paratyphoid-B* (*typhi-murium*?) both parenterally and per os; fowls proved to be the most resistant. Pfeiler and Rehse (1913) also found that fowls were not very susceptible to paratyphoid infection. They studied the outbreak on a farm of a chronic disease which occurred enzootically with a few deaths reported from time to time and recovered a bacterium from the internal organs of affected birds which they placed in the *Paratyphus-B* group. Nevertheless, although adult fowls may not be very susceptible to natural or artificial infection with certain types of *Salmonella*, numbers of very virulent outbreaks of paratyphoid in chickens are reported periodically. Thus Spray and Doyle (1921) found that outbreaks of a very destructive disease in newly hatched chicks (2 to 4 days old) may be caused by organisms of the paratyphoid-B group, as well as by *S. pullorum*. Edwards (1929) investigated an epizootic affecting over 2,000 very young chicks with a mortality rate of about 25 per cent. *S. pullorum* could not be detected in any of the birds examined, but there was a mixed infection of *typhi-murium* and *anatum* associated partly with coccidiosis. By means of serological tests and post mortem examinations, no carriers could be detected in either the breeding stock or in the survivors.

Later five separate outbreaks of paratyphoid in birds were recorded by McGaughey (1932). Of these three occurred in chicks, one in adult fowls and one in ducks. In one of the chicken epizootics *typhi-murium* was obtained from the heart-blood and internal organs of the dead birds; in another outbreak a non-motile

strain of the *typhi-murium Paratyphi-B* group was isolated from the carcasses, while *enteritidis* was recovered from the third group of chickens. The disease in the adult fowls caused a large number of sudden deaths. McGaughey isolated *typhi-murium* from the internal organs of one bird and *pullorum* from another. In the outbreak affecting the ducks several deaths occurred during the course of a few months. The ovary of one of the ducks examined resembled that of a case infected with *pullorum*, and *S. enteritidis* was obtained from its liver and ovary. It was stated above that Jansen (1936) isolated *enteritidis var. Essen* from the organs of diseased ducks, and also from the yolk sac of chickens that had been living in close association with ducks.

During the course of the routine diagnosis of chick disease, Jungherr and Borden (1934) encountered 5 cases of paratyphoid infection. In two of these the causal agent was found to be *typhi-murium var. storrs*, in two atypical strains of *cholerae-suis*, and in one an atypical strain of *L2*.

In spring of 1936 Schalm (1937) investigated a *pullorum*-like disease that affected several batches of chicks on a Californian farm. The breeding stock had been healthy for a number of years and the farm was free from bacillary white diarrhoea, but deaths were reported in the chicks sold to five different farmers, and about 40 per cent. losses were sustained in 4 to 10 days old birds. *Typhi-murium* was isolated from the heart-blood and organs, and chilling during shipment was considered to be a predisposing cause. The chicks that remained on the breeder's farm developed an apparently chronic form of the disease which affected fewer and much older birds. It was thought that infection of the chickens on the farm took place in the incubator after hatching by means of bacteria present in the faecal matter on the surface of the egg shells. Schalm could not infect 4-day old chickens either by feeding or by intravenous inoculations of cultures of *typhi-murium*.

According to Emmel (1936) different species of *Salmonella* may occur as facultative parasites in the alimentary canal of fowls; by examining the intestinal contents of a number of fowls suffering from enteritis due either to coccidiosis or to worm infestation he claims to have isolated a*ertrycke, paratyphi-A, paratyphi-B, enteritidis, typhi* as well as *pullorum*. The account published by Emmel does not appear to be complete; there are no records given of the methods used for typing the strains, and it is not stated on what grounds the different strains were classified. To me it seems that Emmel's claims cannot be accepted, unless much more information is available than is presented in his report.

The incidence of food poisoning produced by fowl's meat is apparently much lower than that caused by foods prepared from duck and goose meat and eggs. In a review of outbreaks of food-poisoning due to bird meat, Beller (1933) discussed several cases where the meat of aquatic birds was incriminated; he pointed out that fowl and pigeon meat intended for food are always well cooked so that food-poisoning cannot be readily set up even when the meat is infected. From 1923 to 1932 Meyer (1933) studied 50 outbreaks of food-poisoning caused by bird meat. Three hundred people were

affected and there were three deaths; 37 of the outbreaks were due to goose, 3 to duck, 7 to fowl, one to partridge, one to pigeon meat and in one case both goose and fowl meat were incriminated. Although the type of *Salmonella* recovered was not determined in all outbreaks, *typhi-murium* was found to be by far the most common; *enteritidis* was incriminated in a small number of the cases, while "*Paratyphus-B*" and a *newport*-like organism were recovered from one outbreak. The bacteria were generally isolated either from the suspected food, or from the patients, or from both food and patients. As stated above, the importance of ducks as carriers of *Salmonella* infection lies rather in the eggs than in the meat.

Although *S. gallinarum* and *S. pullorum* are generally regarded as non-pathogenic for man, Kauffmann (1934) has described a strain of *gallinarum* (the Duisberg strain) which he isolated from the stools of patients that developed acute symptoms of gastro-enteritis after they had partaken of a salad. The organism resembled *gallinarum* serologically, and was pathogenic for chickens, but its fermentation reactions were atypical.

Apart from infection with *gallinarum* and *pullorum*, I have studied four outbreaks of *Salmonella* infection in fowls in South Africa.

I. The information relating to this outbreak has been furnished by me in another paper (Henning, 1937).

In 1935 a farmer at Amersfoort in the Transvaal sustained serious losses amongst his chickens from what appeared to be an infectious disease. The disease was not investigated and the cause of the mortality remained unknown until the end of 1936, when the malady reappeared and a few affected birds were sent to Onderstepoort for examination. An apparently pure culture, obtained by Mr. J. D. W. A. Coles, head of the Poultry Disease Section, from the heart blood of a 7-day-old chick, was handed to me for identification. The culture was plated and a few isolated colonies were picked. The cultures obtained from these were tested against various agglutinating sera. It was found that the antigenic structure of the organism exhibited an entirely new combination of antigenic components; for this reason, therefore, the germ should be admitted to species rank in compliance with the recommendations of the Salmonella Sub-committee of the Nomenclature Committee of the International Society of Microbiology (1934). The name *Salmonella amersfoort* (Henning, 1937) was given to the organism—after the place of its origin.

Morphology and cultural character.—Morphologically, *S. amersfoort* resembles a typical *Salmonella*, and, like it, grows readily on ordinary laboratory media. It is Gram-negative and actively motile. Saline and thermo-agglutination tests, as well as the shape of individual colonies, show that it is smooth.

Biochemical character.—*S. amersfoort* forms acid and gas in glucose, dulcite, mannite, maltose, arabinose, rhamnose, and sorbite; it forms hydrogen sulphide and renders litmus milk alkaline; it does not produce indol.

Pathogenicity.—*S. amersfoort* is pathogenic for chickens and mice; 0·25 c.c. of a 24-hour-old broth culture, given intraperitoneally, kills a 6-week-old chicken in 4 days and 0·05 c.c. kills an adult mouse in 36 hours. *S. amersfoort* was recovered from the heart blood and spleen in each case. But chickens dosed with 1 c.c. of the virulent broth culture remained apparently healthy.

While making an antigenic analysis of *Salmonella amersfoort* I noticed well-marked flocculation occurring between this organism and the sera of organisms that are generally regarded as not even remotely related antigenically.

Bruce White (1929) described three forms of antigenic variation occurring in the genus *Salmonella*: (1) the "H" form—"O" form variation of Weil and Felix (1920), (2) the Smooth form—Rough form variation of Arkwright (1921), and (3) the specific phase—non-specific phase variation of Andrewes (1922). Later Kauffmann and Mitsui (1930) described a new type of phase variation, involving the specific phases of *brandenburg*, *dar-es-salaam* and *Potsdam*; and they called this $\alpha - \beta$—variation. A similar variation has been observed in a number of other types of *Salmonella*, viz. *abortus-bovis* (Bernard, 1935), *hvittingfoss* and *oslo* (Tesdal, 1936, 1937), *bispebjerg* and *typhi* (Kauffmann, 1936a, 1936b), *chester* and *schleissheim* (Kauffmann and Tesdal, 1937). The antigenic structure of these organisms, according to Kauffmann and Tesdal (1937), is given in Table 20B. The inagglutinable (containing Vi antigen) and agglutinable forms of *S. typhi* described by Felix and his co-workers (1934, 1935, 1936) may be regarded as another type of variation. Kauffmann (1935) introduced the terms "V-form" and "W-form" to denote, respectively, the variant containing Vi-antigen and that devoid of it; while Craigie and Brandon (1936), Brown (1936), Scholtens and others showed the effect of bacteriophage on the V – W degradation.

Serology.—For the study of the antigenic structure of *S. amersfoort*, "O" sera, "H" specific and non-specific sera, and mixed "O" and "H" sera, prepared against a number of representative strains of *Salmonella*, were used. Sera prepared against *S. amersfoort* were also used. The sera and agglutinating suspensions were prepared according to the methods described above.

Preliminary tests showed that *amersfoort* gave a well-marked fine granular agglutination with "O" sera containing factors VI and VII of the Kauffmann-White schema (*choleraesuis*, *newport*, *potsdam* and others), while a distinctly coarse floccular agglutination was produced not only with "H" sera containing factors *en* or *e* (*abortus equi*, *brandenburg*, *potsdam*, *dar-es-salaam*, *onderstepoort*, *newport*, *reading* or *anatum*), but also with those containing factor *d* (*stanley*, *muenchen* and *typhi*). However, a much stronger agglutination was produced by sera containing factors *en* than with those containing factor *e* but not *n*.

The culture was again plated on Mason tubes to obtain a number of separate colonies for independent study. After 5 hours' incubation at 37° C. broth cultures of these colonies were tested against *Kunzendorf* and *Binns* group sera as well as against the type sera of

onderstepoort, *newport*, *potsdam* and *typhi*. The results are given in Table 15. It will be noticed that the majority of the cultures agglutinated with *typhi* serum (factor *d*), a number agglutinated with *potsdam* (factors *enlv*), and *newport* (factors *eh*) or *onderstepoort* (factors *eh*) sera, a few agglutinated incompletely with all four sera, being apparently intermediate forms, but no agglutination whatsoever was effected with *Kunzendorf* and *Binns* sera.

TABLE 15.

Thirty colonies grown in broth for 5 hours and tested against 5 different sera.

No. of Colony.	*Typhi* s.	*Newport* or *Onderste-poort* s.	*Potsdam* s.	*Kunzendorf* s.	*Binns* s.
1–16.....	++++	0	0	0	0
17–25.....	0	++++	++++	0	0
26–30.....	+	+	+	0	0

++++ = complete flocculation within 30 minutes.
\+ = partial flocculation after 1 hour.
0 = no flocculation after 18 hours.
In headings to table s. = serum.

These results indicated (1) that the organism occurred only in the type phase and (2) that the culture used was either a mixed one or that it exhibited properties that have hitherto not been described in a member of the *Salmonella* group. In order to settle the matter of the purity of the strain, Dr. J. H. Mason kindly single-celled fresh cultures derived from a colony of each of the two types—i.e. from one colony agglutinating only with sera made against specific factor *d* and from another that flocculated solely with the anti-sera of specific factors *en* and *eh*. After plating the primary cultures obtained from

TABLE 16A.

Twenty-two colonies picked from the plate seeded with growth from the single cell obtained from colony 1, *Table* 15.

No. of Colony.	*Typhi* Serum.	*Onderstepoort* or *Potsdam* Serum.
1 to 21.....	++++	0
22.....	0	++++

the single cells a number of well-isolated colonies were again picked into broth tubes and incubated at 37° C. for 5 hours—in order to reduce the lag phase in the growth the broth tubes were placed in a water-bath at 40° C. for 10 minutes before transferring them to the incubator.

Four single cells (*a b c* and *d*) obtained from colony 1, Table 16B, were now cultivated separately in broth and plated. A number of colonies from each plate were picked into broth, incubated and tested against both *d* and *en* sera. The results are given in Table 17.

TABLE 16B.

Thirty colonies picked from the plate seeded with the broth culture from single cell of colony 17, *Table* 15.

No. of Colony.	*Typhi* Serum.	*Onderstepoort* or *Potsdam* Serum.	Saline Control.
1 to 28	0	++++	0
29 and 30	++++	0	0

TABLE 17.

Single Cell.	No. of Colony.	*Typhi* Serum.	*Onderstepoort* or *Potsdam* Serum.
a	1 to 4	++++	0
a	5 to 12	0	++ --
b	1 to 14	0	++ ++
b	15	++++	0
c	1 to 14	++++	0
c	15	0	++++
d	1 to 10	++++	0

++++ = complete flocculation after 30 minutes.
0 = no flocculation after 18 hours.

Therefore, these results clearly show that *S. amersfoort* is composed of two distinct "H" antigenic complexes, both of which occur in the specified phase; the second (*d*-) antigen, apparently corresponding to the α phase of Kauffmann and Mitsui (1930), is agglutinated with the "H" serum of *typhi* and, as will be shown below, also with specific sera of other *Salmonellas*, *stanley* and *muenchen*, containing specific factor *d*, while the other component, the first (*en*-) antigen, apparently corresponding to the β phase of Kauffmann and Mitsui (1930), is agglutinated solely with *potsdam*, *onderstepoort*, and other sera containing aglutinins for the type factors *en* and *eh* (*vide infra*). Sera containing agglutinins for

factors *en* always give a much stronger flocculation than the anti-sera of factors *eh*. It has also been shown that single cells composed of either the one or other complex constantly give rise to daughter cells some of which resemble the parent cell antigenically, while others have adopted a new antigenic structure entirely different from that present in the parent. The latter daughter cells again give rise to offspring some of which resemble themselves, while others are like the parent. These mutations constantly proceed and cells containing either the one or other antigenic complex continually produce cells of both types, and neither the one nor the other type of cell has been found to breed entirely true.

On single-celling the growth obtained from each of the two types of colonies serially three successive times, both variants constantly appear in the cultures arising from the single cells.

The purity of the culture is therefore beyond dispute; it is the property of the bacterium of giving rise to two distinct types of variants in the specific phase that is responsible for the uncommon behaviour of the culture. The organism apparently does not occur in the non-specific phase.

" O " agglutination.—Cross-agglutination tests were carried out with the heat-stable " O " antigens and " O " sera of the different *Salmonella* type of the Kauffmann-White schema; also with *S. aberdeen* (Smith, 1934), *S. poonae* (Bridges and Scott, 1935) and *S. onderstepoort* (Henning, 1936). The reactions are given in Table 18.

TABLE 18.—*" O " Agglutination.*

	" O "—ANTIGEN.				
	Amersfoort.	*Potsdam.*	*Muenchen.*	*Onderstepoort.*	*Brandenburg.*
UNABSORBED SERA—					
Amersfoort s.	800	800	200	100	0
Potsdam s.	800	800	—	—	—
Muenchen s.	200	—	1,600	—	—
Brandenburg s.	0	—	—	—	1,600
Onderstepoort s.	50	—	—	800	—
ABSORBED SERA—					
Amersfoort s.a.b. Amersfoort	0	0	—	—	—
Amersfoort s.a.b. *Potsdam*	0	0	—	—	—
Amersfoort s.a.b. *Muenchen.*	200	—	0	—	—
Amersfoort s.a.b. *Brandenburg*	800	—	—	—	0
Potsdam s.a.b. *Potsdam*	0	0	—	—	—
Potsdam s.a.b. *Amersfoort*	0	0	—	—	—
Muenchen s.a.b. *Amersfoort.*	0	—	800	—	—

0 = less than 1 : 50. — = not tested.

In this table s. = serum; s.a.b. = serum absorbed by.

The results show that *amersfoort* " O " as well as *potsdam* " O " sera are completely exhausted for the homologous " O " antigen by *amersfoort*. In the same way both sera are exhausted by *potsdam*. The somatic " O " antigen of *amersfoort* must, therefore, be regarded as identical with that of *potsdam*, i.e., it is composed of factors VI, VII.

" *H* " *agglutination.*—Flocculation, approximately equivalent in titre to that produced with the homologous antigen, was obtained with the specific sera of *abortus equi*, *potsdam*, *brandenburg*, *dar-es-salaam*, *muenchen* and *typhi*, but a much weaker agglutination resulted when the type of serum of *onderstepoort*, *newport*, *reading* or *anatum* was used for the test. In the same way *amersfoort* " H " serum agglutinated the specific antigens of *abortus equi*, *potsdam*, *brandenburg*, *dar-es-salaam*, *stanley*, *muenchen* and *typhi* almost up to full titre, while its titre for type antigens containing factors *eh* was much lower.

On absorbing *amersfoort* " H " serum with the specific phase of either *potsdam* (factors *enlv*), *brandenburg* (factors *enlv*) or *dar-es-salaam* (factors *enlw*) the titre of the serum for one of the homologous specific antigens (*en-*), β phase, was reduced from 6,400 to approximately 800, while the titre for the other homologous specific antigen (*d-*), α phase, as well as for *stanley*, *muenchen* and *typhi* (factor *d*) remained unaltered. When *abortus equi* (factors *enx*) was used for the absorption, the reduction in titre for the homologous *en* antigen (β phase) was almost complete, but still no noticeable decrease in agglutinins for the homologous *d* antigen (α phase) was effected; a small residue, however, remained which caused an incomplete agglutination with the *en*-variant (β phase) of *amersfoort*. The cause of this flocculation is discussed below.

On the other hand, when *amersfoort* " H " serum was absorbed with either *stanley*, *muenchen* or *typhi* (factor *d*) most of the agglutinins for the one variant (*d-*) α phase, of *amersfoort* were removed, while the titre for the other homologous antigen (*en-*) β phase, remained unaltered (Table 19).

When either *potsdam* or *brandenburg* serum was absorbed with *amersfoort*, all agglutinins for *amersfoort* were removed, but the titre of the serum for *panama* (factors *lv*) and *london* (factors *lv*) was not affected. Moreover, the treated serum still agglutinated the homologous antigen although the flocculation was incomplete and the fluid remained turbid, due, no doubt, to the persistence of *lv* agglutinins in the serum. *Dar-es-salaam* serum behaved in practically the same way, but *amersfoort* is apparently capable of removing all the agglutinins for the homologous antigen from *abortus-equi* serum.

On absorbing either *stanley*, *muenchen* or *typhi* serum with *amersfoort*, most of the agglutinins for the homologous " H " specific antigen were exhausted, *muenchen* serum being exhausted much more completely than either *stanley* or *typhi* serum, while all the agglutinins for the second variant of *amersfoort* (factor *d-*) were removed.

TABLE 19.

"H" SPECIFIC ANTIGEN.

	Amersfoort d.	*Amersfoort* en.	*Typhi.*	*Stanley.*	*Muenchen.*	*Potsdam.*	*Brandenburg.*	*Dar-es-salaam.*	*Abortus-equi.*	*Onderstepoort.*	*Newport.*	*Reading.*	*Anatum.*	*Panama.*	*London.*
UNABSORBED SERA.															
Amersfoort d. s.	12,800	6,400	12,800	12,800	12,800	1,600	3,200	3,200	3,200	200	—	—	—	0	0
Amersfoort en. s.	6,400	6,400	6,400	6,400	6,400	3,200	6,400	3,200	6,400	400	400	400	400	0	0
Typhi s.	12,800	0	12,800	12,800	12,800	—	—	—	—	—	—	—	—	—	—
Stanley type s.	25,600	400	25,600	25,600	25,600	—	—	—	—	—	—	—	—	—	—
Muenchen type s.	6,400	0	6,400	6,400	12,800	—	—	—	—	—	—	—	—	—	—
Potsdam s.	0	6,400	—	—	—	6,400	6,400	6,400	3,200	800	—	—	—	6,400	1,600
Brandenburg s.	0	6,400	—	—	—	6,400	12,800	3,200	3,200	400	—	—	—	6,400	3,200
Dar-es-salaam s.	0	1,600	—	—	—	—	—	3,200	—	—	—	—	—	1,600	—
Onderstepoort s.	0	400	—	—	—	200	—	200	200	1,600	—	—	—	—	—
Abortus-equi s.	—	6,400	—	—	—	1,600	3,200	6,400	3,200	—	—	—	—	—	—
ABSORBED SERUM.															
Amersfoort d. s.a.b. *Amersfoort*	0	0	0	0	0	0	—	—	0	—	—	—	—	—	—
Amersfoort d. s.a.b. *Typhi*	400	6,400	0	—	—	—	—	—	—	—	—	—	—	—	—
Amersfoort d. s.a.b. *Stanley*	400	3,200	—	0	—	—	—	—	—	—	—	—	—	—	—
Amersfoort d. s.a.b. *Muenchen*	400	3,200	—	0	0	—	3,200	3,200	—	—	—	—	—	—	—
Amersfoort en s.a.b. *Potsdam*	6,400	800*	—	—	—	0	—	0	800	—	—	—	—	—	—
Amersfoort en s.a.b. *Brandenburg*	6,400	800*	6,400	—	—	—	0	—	—	—	—	—	—	—	—

0 = less than 1 : 50. * = partial flocculation, fluid remaining turbid.
In this table s. = serum, s.a.b. = serum absorbed by. — = not tested.

TABLE 19 (*continued*).

	Amersfoort d.	*Amersfoort en.*	*Typhi.*	*Stanley.*	*Muenchen.*	*Potsdam.*	*Brandenburg.*	*Dar-es-salaam.*	*Abortus-equi.*	*Onderstepoort.*	*Newport.*	*Reading.*	*Anatum.*	*Panama.*	*London.*
ABSORBED SERUM (*continued*).															
Amersfoort en s.a.b. *Dar-es-salaam*.....	6,400	1,600*	—	6,400	—	—	—	0	—	—	—	—	—	—	—
Amersfoort en s.a.b. *Abortus-equi*.....	6,400	200	—	—	—	—	0	—	0	—	—	—	—	—	—
Typhi s.a.b. *Amersfoort*.	0	0	800	—	—	—	—	—	—	—	—	—	—	—	—
Typhi s.a.b. *typhi*.....	0	0	0	—	—	—	—	—	—	—	—	—	—	—	—
Stanley s.a.b. *Amersfoort*	0	0	—	400	400	—	—	—	—	—	—	—	—	—	—
Stanley s.a.b. *Stanley*...	0	0	—	0	—	—	—	—	—	—	—	—	—	—	—
Muenchen s.a.b. *Amersf.*	0	0	—	0	100	—	—	—	—	—	—	—	—	—	—
Potsdam s.a.b. *Amersf.*.	—	0	—	—	—	6,400*	6,400	6,400	0	—	—	—	—	6,400	3,200
Brandenburg s.a.b. *Amersfoort*.....	—	0	—	—	—	—	6,400	—	—	—	—	—	—	6,400	3,200
Dar-es-salaam s.a.b. *Amersfoort*.....	—	0	—	—	—	800	—	1,600	—	—	—	—	—	1,600	800
Abortus-equi s.a.b. *Amersfoort*.....	0	0	—	—	—	0	0	—	0	—	—	—	—	—	—
Potsdam s.a.b. *Amers.* and then by *Panama*	—	—	—	—	—	100	100	—	—	—	—	—	—	0	0
Brandenburg s.a.b. *Amersfoort*, then by *London*.....	—	—	—	—	—	—	200	—	—	—	—	—	—	0	0
Abortus-equi s.a.b. *Potsdam*.....	—	400	—	—	—	—	0	—	200	—	—	—	—	—	—

0 = less than 1 : 50. * = partial flocculation, fluid remaining turbid.
In this table s. = serum, s.a.b. = serum absorbed by. — = not tested.

Discussion.

These results show that *Salmonella amersfoort* contains two distinct antigenic complexes, the one, β phase, corresponding to factors *en* of *abortus-equi, potsdam, brandenburg* and *dar-es-salaam* plus an additional factor, part of which apparently corresponds to factor *x* of *abortus-equi*; the other complex, α phase, coincides largely with factor *d* of *stanley, muenchen* and *typhi*. The additional factor is probably responsible for the residue of agglutinins left for the first (*en*-) antigen, β phase, after absorbing *amersfoort* serum with *potsdam, brandenburg* or *dar-es-salaam;* but, although factor *x* of *abortus-equi* apparently forms a part of this additional factor, there may be another component which is not present in *abortus-equi*. The fact that *amersfoort* exhausts all agglutinins from *abortus-equi* serum for itself as well as for the homologous specific antigen indicates that *amersfoort* contains all the specific antigenic components of *abortus-equi*, i.e. factors *enx*; but since *abortus-equi* fails to exhaust *amersfoort* serum completely for the homologous first (*en*-) antigen it is possible that this antigen of *amersfoort* contains a minor factor in addition to the *enx* of *abortus-equi*.

After absorbing *amersfoort* serum with either *stanley, muenchen* or *typhi*, a small residue is left which still agglutinates the homologous second (*d*-) antigen, α phase, but not the specific antigen (factor *d*) of either *stanley, muenchen* or *typhi*. It is not quite clear to what this residue can be ascribed; whether it should be regarded as an extra factor in the second (*d*-) antigen, α phase, in addition to factor *d* of *stanley, muenchen* and *typhi*, or whether it can be attributed to a trace of the first (*en*-) antigen, β phase, present in the emulsion of the second (*d*-) antigen, α phase, of *amersfoort* used for the test, is not certain. If the latter explanation holds it is likely that the agglutination occurring in *amersfoort* serum absorbed with *abortus-equi* is likewise due to an overflow of the second (*d*-) antigen, α phase, in the emulsion of the first (*en*-) antigen, β phase, of *amersfoort*.

Neither *abortus-equi, potsdam, brandenburg*, nor *dar-es-salaam* effected any reduction in the titre of *amersfoort* serum for the homologous second (*d*-) antigenic complex, α phase, or for the type phases of *stanley, muenchen* and *typhi*. In the same way neither *stanley, muenchen* nor *typhi* absorbed an appreciable amount of agglutinins from *amersfoort* serum for the homologous first (*en*-) antigen, β phase, or for *abortus-equi, potsdam, brandenburg* and *dar-es-salaam*.

When *potsdam* serum was absorbed by *amersfoort* all agglutinins for both *amersfoort* and *abortus-equi* were completely exhausted, but flocculation to nearly full titre was still effected with the specific phases of *potsdam, brandenburg, panama* and *london*. On reabsorbing the partly absorbed *potsdam*, serum with *panama* (factors *lv*) no appreciable agglutination resulted when specific antigens of *potsdam, brandenburg, panama* and *london* were used. *Amersfoort*, therefore, removed only the agglutinins of factors *en* from the *potsdam* serum, leaving the agglutinins of factors *lv* to the absorbed by *panama*.

The fact that *amersfoort* almost completely exhausted *muenchen* serum for the homologous specific antigen suggests that the second (*d*-) factor, α phase, is similar to the specific phase (factor *d*) of *muenchen*; the small residues of agglutinins left in *stanley* and *typhi* sera for their homologous specific antigens after absorption with *amersfoort* cannot be explained at present.

Summary and Conclusions.

A new type of pathogenic *Salmonella* for the fowl has been described. Its somatic "O" antigen corresponds with factors VI, VII of *potsdam*. It occurs only in the specific phase, but its flagellar "H" antigen contains at least two distinct and separate antigenic complexes, which commonly occur in organisms that are not even remotely related. The one complex (the first, *en*-, antigen, β phase of Kauffmann and Mitsui) contains factors *enx*, which represent also the factors of the specific phase of *abortus-equi*. The other complex (the second, *d*-, antigen, α phase of Kauffmann and Mitsui) contains factor *d*, which comprises the type phase of *stanley*, *muenchen* and *typhi*.

Single cells containing factors *enx*, on multiplying, constantly yield variants containing factor *d* as well as offspring that retain antigenic complex *enx*. In the same way single cells containing apparently only specific factor *d* will bring forth new cells, most of which retain the parental antigenic structure, but a small proportion of the progeny will acquire specific factors *enx* instead of *d*.

When a broth culture of *amersfoort* in either the *enx* or *d* phase and in an apparently pure form, is used for the preparation of sera, agglutinins of approximately the same titre for both variants are produced in the sera. The purity of the phase culture must be judged by the agglutination test, using heterologous sera which contain agglutinins either against factors *enx* or *d*.

As a result of the information given above the following antigenic structure is proposed for *Salmonella amersfoort*:

Somatic "O" antigen—VI, VII.

Flagellar "H" antigen—

(1) α phase of Kauffmann and Mitsui—*d*-

(2) β phasse of Kauffmann and Mitsui—*enx*.

II. During the course of 1937 an outbreak of a fatal disease occurred among a group of adult fowls on a farm near Onderstepoort. The symptoms and lesions presented were indistinguishable from those of an ordinary virulent outbreak of fowl typhoid. The disease was investigated by my colleague, Mr. J. D. W. A. Coles, who made spleen cultures on agar from three birds; the cultures were handed to me for identification and I spread seed material from each culture on to MacConkey's bile-salt agar in Mason tubes. After 24 hours' incubation both small and large non-lactose fermenting colonies appeared in two of the Mason tubes. Some of these colonies were picked and mixed separately with drops of a *gallinarum* serum

dilution on glass slides. All the small colonies tested were readily agglutinated by the *gallinarum* serum, but all the large colonies failed to react with this serum. A pure culture of the small colonies was obtained and labelled *culture* 360. Several of the large colonies were now tested against various " O ", type and group sera. No agglutination, whatsoever occurred with any group serum, but some of the colonies flocculated when mixed with *typhi* and *stanley* type sera, while others were agglutinated by type sera containing factors *enx*, *enlv*, *enlw* and *eh* (*abortus-equi*, *potsdam*, *dar-es-salaam*, *onderstepoort*). All the colonies tested were agglutinated by " O " sera containing factors VI, VII (*cholerae-suis*, *potsdam*). Moreover, all the colonies tested were flocculated by *amersfoort* " O " and " H " mixed sera (factors VI, VII and *d—enx*). It appeared, therefore, from these preliminary tests that the organisms from the large colonies, labelled *culture* 359, were related to *amersfoort*, and a rabbit was immunised for the production of antiserum.

When culture 359 was plated so as to give several well separated single colonies, like *amersfoort*, some of these were found to agglutinate only with a type serum containing factors *enx*, *enlv*, *enlw or eh* (*abortus-equi*, *potsdam*, *dar-es-salaam* or *reading*), while others were flocculated only by type sera containing factor *d* (*typhi*, *stanley* and *muenchen*). In order to make sure that the culture used was unquestionably pure it was single-celled. It was found that the single-cell obtained from the colony that was agglutinated by type sera containing factors *enx* etc. produced daughter organisms which, on sub-cultivation, gave rise to colonies some of which agglutinated with *enx* sera, while others (about 12 per cent.) were agglutinated by type sera containing factor *d*. Moreover, the single-cell procured from the colony that was flocculated by type sera containing factor *d* yielded bacilli, which on sub-cultivation produced colonies occurring in both the phases (*d* and *enx*). Some of the colonies (about 90 per cent.) were agglutinated only by sera containing factor *d*, while a smaller number were agglutinated by type sera containing factors *enx*, *enlv*, *enlw* or *eh*. The bacilli of culture 359, therefore, also occurred in two specific phases, the α and β phases of Kauffmann and Mitsui (1930), the organisms which occurred in the one phase constantly dissociating into bacilli which were present in both phases. As the organism occurred only in the specific phase the dissociation was confined to that phase; non-specific variants were not encountered at any time.

In order to settle the identity of culture 359, agglutination and absorption tests were carried out as shown in Table 20A. After absorption tests had been performed with *amersfoort* the identity of the strain was determined, and no further tests were performed.

The results of Table 20A show that *amersfoort* absorbed all the agglutinins (" O ", " H " *d*-type and " H " *enx* type) from the homologous serum as well as from 359 serum; on the other hand, 359 completely exhausted both its own serum and *amersfoort* serum. *Amersfoort* (culture 336) and culture 359 should, therefore, be regarded as identical. But the original *amersfoort* (culture 336), was obtained in pure culture from dead chickens during a virulent outbreak of a septicaemic disease in very young chickens at

Amersfoort, while the present strain, *culture* 359, was isolated in conjunction with *gallinarum* (*vide infra*) from adult fowls suffering from a fowl typhoid-like disease. In the epizootic discussed above (1) and in my previous paper (Henning, 1937), *amersfoort* 336 was apparently the sole cause of the mortality in the chicks; but in the present outbreak it is not quite clear whether *amersfoort* 359 or *gallinarum* 360 was the primary cause of the disease. The probability is that *gallinarum* 360 was the more important etiological agent, and that *amersfoort* 359 gained admission into the body after its resistance had been lowered by fowl typhoid.

TABLE 20A.

Antigen.	*Amersfoort* 336 Serum Absorbed by *Amersfoort*.	*Amersfoort* 336 Serum Absorbed by 359.	*Amersfoort* 336 Serum Unabsorbed.	359 Serum Absorbed by *Amersfoort* 336.	359 Serum Absorbed by 359.	359 Serum Unabsorbed.
Amersfoort "O"	0	0	800	0	0	1,600
Amersfoort "H" *d* (*a*)	0	0	12,800	0	0	12,800
Amersfoort "H" *en* (*β*)	0	0	6,400	0	0	12,800
359– "O"	0	0	800	0	0	1,600
359– "H" (*a*)	0	0	12,800	0	0	12,800
359—*en* (*β*)	0	0	6,400	0	0	12,800

0 = less than 1 : 100.

Whereas the organisms comprising *amersfoort* culture 359 were motile, those of *culture* 360, obtained from the small colonies, were non-motile. *Culture* 360 was tested, therefore, both serologically and by means of fermentation reactions. The latter are given below (Table 25) and are typical for *gallinarum*. Agglutination and absorption tests were carried out with culture 560 and *gallinarum* 43, obtained from the National Collection of Type Cultures, and the serum of the latter. An antiserum for culture 360 was not prepared, but a one-sided absorption was carried out and it was found that, like *gallinarum* 43, *culture* 360, completely removed all the agglutinins from the serum of *gallinarum* 43. Moreover, the latter serum agglutinated the "O" antigen of culture 360 up to full titre (1:1600). Both serologically and by means of fermentation reactions (vide infra), therefore, culture 360 resembled *gallinarum* 43, and it should be regarded as a strain of *gallinarum*.

The fermentation reactions of *amersfoort* culture 359 are also given below (Table 25).

For purposes of comparison Table 20B is included in order to show the antigenic structure of different organisms that occur in the *a* and *β* phases of Kauffmann and Mitsui.

TABLE 20B.

Organisms known to show α-β phase variation in their specific phases (partly after Kauffmann and Tesdal, 1937).

	"O" Antigen.	H. Specific *a* Phase.	Antigen *β* Phase.	Author.	Origin.
S. Abortus-bovis	I, IV	*b*	*enx*	Bernard (1935)	Aborting cows.
S. Schleissheim	IV	*b*	*Z5*	Kauffmann & Tesdal (1937)	Bovine.
S. hvittingfoss	XVI	*b*	*enx*	Tesdal (1936)	Gestro-enteritis in man.
S. Chester	IV, V (XII)	*eh*	*enx*	Kauffman & Tesdal (1937)	Gastro-enteritis in man.
S. Brandenburg	IV (XII)	*lv*	*en*	Kauffmann & Mitsui (1930)	Gastro-enteritis in man.
S. Bispebjerg	IV (XII)	*a*	*enx*	Kauffmann (1936)a	Gastro-enteritis in man.
S. Abortis-equi	IV (XII)*	?	*enx*	—	Aborting mares.
Potsdam	VI, VII (XII)	*lv*	*en*	Kauffmann & Mitsui (1930)	Gastro-enteritis in man.
S. Oslo	VI, VII (XII)	*a*	*enx*	Tesdal (1937)	Gastro-enteritis in man.
S. Amersfoort	VI, VII (XII)	*d*	*enx*	Henning (1937)	Septicaemic disease in fowls.
S. Typhi	IX (XII)	*d*	*j*	Kauffmann (1936b)	—
S. Dar-es-salaam	I, IX (XII)	lw	en	Kauffmann & Mitsui (1930)	Pyrexia.

* Kauffmann & Tesdal (1937) consider that *S. abortus-equi* will probably also be found to show a–β variation at times. This was confirmed by Edwards and Bruner (1939).

III. During spring of 1936 a very virulent epizootic occurred in a few day-old chicks at the School of Agriculture near Potchefstroom. Some of these chickens were forwarded to Onderstepoort for investigation and were examined by Mr. Coles, Chief of the Section of Poultry Diseases. Heart-blood and spleen cultures made by him yielded a pure growth of a gram-negative bacterium that was handed to me for identification. The organism was found to be very actively motile, and it did not ferment lactose. When it was tested against various " O ", type and group, sera by means of slide agglutination, it was readily agglutinated by " O " sera containing factors IV and V, by *typhi-murium* type serum and by group sera. The organism was also found to be di-phasic, and the culture was labelled 357. A rabbit was immunised with a killed saline suspension of a fresh agar culture, and a good serum was obtained. As the preliminary test indicated that culture 357 is probably related to *typhi-murium*, cross-agglutination and absorption tests were first performed with this organism (Table 20c).

TABLE 20c.

Antigen.	*Typhi-murium* (Glasgow) Serum Absorbed by *Typhi-murium*.	*Typhi-murium* (Glasgow) Serum Absorbed by 357.	*Typhi-murium* (Glasgow) Serum Unabsorbed.	357 Serum Absorbed by *Typhi-murium*.	357 Serum Absorbed by 357.	357 Serum Unabsorbed.
Typhi-murium " O "	0	0	800	0	0	800
Typhi-murium type	100	100	100,000	0	0	25,600
Typhi-murium group	100	100	50,000	0	0	6,400
357—" O "	0	0	800	0	0	800
357—type	100	100	100,000	0	0	25,600
357—group	100	100	50,000	0	0	6,400

0 = 1 : 100. On account of the high titre of the *typhi-murium* type and group serum a small residue (1 : 100) of unabsorbed agglutinins were left after the absorption.

The results of Table 20c clearly show that the antigenic structures of culture 357 and *typhi-murium* (Glasgow) are identical; culture 357 removed all the agglutinins (" O ", type and group) from *typhi-murium* (Glasgow) serum, as well as from the homologous serum, while *typhi-murium* (Glasgow) completely exhausted both its own serum and 357 serum.

The agglutination (titre 1:100) which is recorded in the absorbed sera in columns 2 and 3 of Table 20c is attributed to the high titre of the unabsorbed serum; as stated above, sera of very high agglutination titres are very unwieldy for absorption tests, because it is extremely difficult to remove the last trace of agglutinin, even when the homologous antigen is used for the absorption.

After several strains of pure culture of *typhi-murium* (357) were obtained from a number of the chickens, this organism was considered to be the etiological agent of the epizootic, and an attempt was made to determine the source of the infection. As the first deaths took place only a few days after hatching, it was thought that the infection was probably obtained from the breeding hens through the eggs. Two successive slide agglutination tests were performed with the blood of the breeding stock; but both tests were negative and no carriers could be found among the hens. In the case of ducks Warrack and Dalling (1933) observed that infected eggs were laid only when the titre of the affected birds was high and that the agglutination titre of the sera obtained from reactors dropped considerably during the course of the laying season. Whether the same condition holds for fowls cannot be stated at present, and it is not certain whether the existence of carriers escaped notice on account of the lateness of the tests– serological tests were performed only some weeks after *typhi-murium* had been proved to be the cause of the epizootic. No eggs were available for examination for *typhi-murium* infection, and *Salmonellas* could not be detected in the ovaries of any of the hens examined. The source of the infection, therefore, still remains obscure.

IV. During the course of an investigation of another fowl typhoid-like epizootic among adult birds, Mr. Coles again made agar cultures from the heart-blood and spleen of the affected birds, and handed these to me for further study. The cultures (three in number) did not appear to be pure, and some seed material from each one was thinly spread on MacConkey's bile-salt agar in Mason tubes. One of the cultures yielded only lactose-fermenting colonies and was discarded; but from both the others several large and small non-lactose fermenting colonies were obtained, suggesting the existence of a mixed infection. The small colonies were readily agglutinated by *gallinarum* serum; a few of these were picked, cultured and labelled culture 415. The large colonies were tested against various "O", type and group serum dilutions on glass slides. A distinct fine granular agglutination was obtained with the "O" sera of *senftenberg* and *anatum*; coarse floccules were produced by group sera (e.g. *cholerae-suis* var. *Kunzendorf* serum) and by *newport*, *reading*, *onderstepoort* and *anatum* type sera. Some of the large colonies were sub-cultured and labelled *culture* 414, and a rabbit was immunised with it. The preliminary tests showed that *culture* 414 was related partly to *senftenberg* and *anatum* on account of its "O" antigen and partly to *newport*, *reading*, *onderstepoort* and *anatum* on account of its "H" specific antigen, and that it was di-phasic. Cross agglutination and absorption tests were, therefore, performed, first with *culture* 414, *anatum* and *senftenberg* (Table 20D).

The results of Table 20D show that culture 414 has the same antigenic structure as *anatum*. *Culture* 414 completely absorbed all the agglutinins ("O", type and group) from *anatum* serum, as well as from the homologous serum, while *anatum* completely exhausted the sera of *culture* 414 and of itself. *Anatum* var. *muenster* completely exhausted both the "O" and specific agglutinins from 414 serum, but it merely reduced the non-specific titre from 6,400 to 1,600. *Culture* 414 should, therefore, be regarded as a strain af *anatum*.

Antigen.	*Anatum* S. ab. by *Anatum*.	*Anatum* S. ab. by 414.	*Anatum* S. Unabsorbed.	414 S. ab. *Anatum*.
Anatum "O"	0	0	400	0
Anatum type	0	0	3,200	0
Anatum group	0	0	6,400	0
414—"O"	0	0	400	0
414—type	0	0	3,200	0
414—group	0	0	6,400	0
L_2—O	—	—	—	—
L_2—type	—	—	—	—
L_2—group	—	—	—	—
Kottbus "O"	—	—	—	—
Kottbus type	—	—	—	—
Kottbus group	—	—	—	—
Anatum v. *Muenster* "O"	—	—	—	—
Anatum v. *Muenster* type	—	—	—	—
Anatum v. *Muenster* group	—	—	—	—
Senftenberg "O"	—	—	—	—

TABLE 20D.

414 S. ab. 414.	414 S. Un-absorbed.	L_2 S. ab. 414.	L_2 S. Un-absorbed.	*Kottbus* Specific S. ab. 414.	*Kottbus* Specific Unabsorbed.
0	800	—	—	—	—
0	12,800	—	—	—	—
0	6,400	—	—	—	—
0	800	0	800	0	0
0	12,800	—	0	0	3,200
0	6,400	—	1,600	0	800
—	800	0	800	—	—
—	0	—	320	—	—
—	6,400	0	6,400	—	—
—	0	—	—	1,600	1,600
—	3,200	—	—	0	3,200
—	1,600	—	—	800	3,200
—	800	—	—	—	—
—	6,400	—	—	—	—
—	1,600	—	—	—	—
—	200	—	—	—	—

0 = < 1 : 100, S. = Serum, ab. = absorbed by.

414 S. ab. L_2.	414 S. ab. *Kottbus.*	414 S. a.b. *Anatum* v. *Muenster.*	414 S. ab. *Senftenberg.*	*Senftenberg* S. ab. 414.	*Senftenberg* S. Un-absorbed.
—	—	—	—	—	—
—	—	—	—	—	—
—	—	—	—	—	—
0	800	0	400	0	400
12,800	0	0	—	—	—
200	800	1,600	—	—	—
0	0	—	—	—	—
0	—	—	—	—	—
0	—	—	—	—	—
—	0	—	—	—	—
—	0	—	—	—	—
—	0	—	—	—	—
—	—	0	—	—	—
—	—	0	—	—	—
—	—	0	—	—	—
—	—	—	—	400	800

159-160b 159-160c

Culture 414, obtained from the small colonies, was tested against *gallinarum* 43 serum and was agglutinated by it to full titre (1:1,600); it also completely absorbed *gallinarum* 43 serum, showing that it contained the same antigenic components as *gallinarum*. The fermentation tests given below (Table 25) are also typical for *gallinarum*. *Culture* 415 should, therefore, be regarded as a strain of *gallinarum*. Antiserum for *culture* 415 was not prepared and the absorption test performed was one-sided.

In this outbreak also it is not certain whether *anatum culture* 414 or *gallinarum* 415 was the primary cause of the disease. *Salmonellas* were found in two cultures only—three were made—and both contained *anatum* as well as *gallinarum*. On account of the frequency of *gallinarum* infection, however, and on account of the predominence of *gallinarum* colonies in the first subcultures made, it seems probable that the organism of fowl typhoid was the main etiological agent in this outbreak.

In addition to these outbreaks of *Salmonella* infection in poultry, 149 epizootics of fowl typhoid and 55 outbreaks of disease in young chicks were also investigated. In most of the cases heart-blood and spleen cultures were made by Mr. J. D. W. A. Coles and submitted to me for identification. Some of the cultures were made by me personally, and *culture* 206 was obtained by Dr. Martinaglia from one of a number of chicks thought to be affected with bacillary white diarrhoea. On plating *culture* 206 I noticed that both large and small colonies appeared on the agar within 24 hours. As large and small colonies have frequently been observed in cultures of *pullorum* that were unquestionably pure, especially after allowing the cultures to stand a day or two at room temperature, the existence of a mixed infection was not suspected; nevertheless, a few of the small, and some of the large colonies were picked and cultured separately. The large colonies yielded a dense growth of actively motile organisms, while the small colonies gave rise to a much poorer growth of non-motile bacteria. In order to determine the purity of the two cultures, my colleague, Dr. J. H. Mason, kindly undertook to single-cell them; the growths obtained from the single-cells were labelled *culture* 207 and *culture* 208 for the non-motile and motile bacteria respectively.

The two cultures were now tested against various " O ", type and group sera. *Culture* 207 was agglutinated only by " O " sera containing factor IX, while *culture* 208, which proved to be monophasic, was flocculated by " H " sera containing factor *d*, as well as by " O " sera with factor IX.

With a view to carrying out complete absorption tests, antisera were prepared against both *culture* 207 and 208. But as *culture* 208 was agglutinated with sera containing " O " factor IX and " H "-specific factor *d*, it was evident that the organisms comprising the culture were closely related to *S. typhi*, which contains both these components. Absorption tests were, therefore, performed with *S. typhi*, as shown in Table 21.

The results of these tests clearly show that *culture* 208 absorbs all agglutinins (" O " and " H ") from *S. typhi* serum, as well as from its own serum, while *S. typhi* completely exhausts both the

homologous serum and 208 serum. *Culture* 208, therefore, has the same antigenic structure as *S. typhi*, and should be regarded as a strain of this organism. *S. typhi* is not regarded as a pathogen for poultry, and it is not known to be carried by fowls. It is true that Emmel (1936) claims to have isolated *S. typhi*, *S. paratyphi-A*, *S. paratyphi-B* and other *salmonellas* from the intestinal contents of fowls suffering from enteritis due to coccidiosis or verminosis, but he gives no information on what basis the organisms were recognised, and his conclusions require confirmation before they can be accepted. For comparison see biochemical tests in Table 25.

TABLE 21.

Antigen.	*Typhi* Serum Absorbed by *Typhi*.	*Typhi* Serum Absorbed by 208.	*Typhi* Serum Un-absorbed.	208 Serum Absorbed by *Typhi*.	208 Serum Absorbed by 208.	208 Serum Un-absorbed.
Typhi "O".....	0	0	3,200	0	0	1,600
Typhi "H".....	0	0	25,600	0	0	12,800
208—"O".....	0	0	3,200	0	0	1,600
208—"H".....	0	0	25,600	0	0	25,600

0 = less than 1 : 100.

As *culture* 207 was readily agglutinated by "O" sera containing factor IX, and as *pullorum* infection was suspected, absorption tests were performed with *S. pullorum*, which like *culture* 207 is non-motile. The results, which are given in Table 22, show that *culture* 207 absorbs all the "O" agglutinins from *pullorum* serum and that *pullorum* completely exhausts the serum of *culture* 207. But *pullorum* and *gallinarum* have the same somatic antigenic components, so that it is not possible to determine by means of a serological test alone to which of these two types *culture* 207 belongs; a final differentiation can be made only by means of fermentation tests (Table 25). According to those tests *culture* 207 corresponds to *Salmonella pullorum*.

TABLE 22.

Antigen.	*Pullorum* Serum Absorbed by *Pullorum*.	*Pullorum* Serum Absorbed by 207.	*Pullorum* Serum Un-absorbed.	207 Serum Absorbed by *Pullorum*.	207 Serum Absorbed by 207.	207 Serum Un-absorbed.
Pullorum "O"...	0	0	3,200	0	0	3,200
207—"O".....	0	0	3,200	0	0	3,200

0 = less than 1 : 50.
Pullorum = *Pullorum* Bb 26 of the N.C. of type cultures.

Discussion.

Of the 139 outbreaks of fowl typhoid 137 cultures were obtained from fowls and two from turkeys. Against four of these, antisera were prepared for the purpose of performing absorption tests with

strains of *gallinarum* and *pullorum* obtained from the National Collection of Type Cultures of the Lister Institute. In Table 23 the results are given which were obtained with *culture* 29; the results obtained with the other three cultures (249, 314 and 340) are similar, but are not given. Table 23 shows that *culture* 29 absorbs all the " O " agglutinins from *gallinarum* serum, as well as from the homologous serum, while *gallinarum* exhausts both its own serum and 29 serum. Similar results were obtained when *pullorum* serum was substituted for *gallinarum* serum, and *pullorum* cultures used for the absorption tests instead of *gallinarum*. *Cultures* 29, 249, 314 and 340, therefore, resemble both *gallinarum* and *pullorum* serologically, but their fermentation reactions (Table 25) corresponded to those of *gallinarum*, so that they should be regarded as strains of *gallinarum*. The other 135 cultures were used for unilateral absorption tests of *gallinarum* serum, and were found to remove all the " O " agglutinins from the serum; the fermentation reactions of all these cultures also resembled those of *gallinarum*.

TABLE 23.

Antigen.	*Gallinarum* Serum a.b. *Gallinarum.*	*Gallinarum* s. a.b. 29.	*Gallinarum* s. Unabsorbed.	29 s. a.b. *Gallinarum.*	29 s. a.b. 29.	29 s. Unabsorbed.
Gallinarum " O "	0	0	1,600	0	0	1,600
29—" O "	0	0	1,600	0	0	1,600

Gallinarum = *gallinarum* 416 of N.C. of type cultures.
0 = less than 1 in 50.
a.b. = absorbed by.
s. = serum.

A study was also made of 55 cultures obtained from a number of few day old chicks suffering from an acute disease, and from the ovaries of hens that gave a positive agglutination test for *pullorum*. Against three of these, cultures 317, 322, and 436, antisera were prepared for absorption tests. The results, which are given in Table 24, show that *culture* 317 removes all the " O " agglutinins from *pullorum* serum and from the homologous serum, while *pullorum* also completely exhausts both these sera. Similar results were also obtained with cultures 322 and 436 and *pullorum*. When *gallinarum* was substituted for *pullorum* identical results were obtained, so that identification of the cultures could not be made entirely on the basis of the serological test—fermentation tests were necessary for the complete differentiation between *gallinarum* and *pullorum*; these are given in Table 25. Unilateral absorption tests were performed with the other 52 cultures and *pullorum* or *gallinarum* serum, resulting in the complete absorption of the sera. But when fermentation tests were carried out, it was found that the reactions of 42 of the cultures resembled those of pullorum, while the other 10 corresponded to *gallinarum*.

TABLE 24.

Antigen.	*Pullorum* Serum Absorbed by *Pullorum*.	*Pullorum* Serum Absorbed by 317.	*Pullorum* Serum Un-absorbed.	317 Serum Absorbed by *Pullorum*.	317 Serum Absorbed by 317.	317 Serum Un-absorbed.
Pullorum " O "...	0	0	3,200	0	0	1,600
317—" O ".....	0	0	3,200	0	0	1,600

Pullorum = *Pullorum* Bb. 26.
0 = less than 1 in 50.

Recently I have studied a culture of *pullorum* isolated from the spleen of a duck by a colleague, Mr. Haig.

According to the fermentation reactions, therefore, 45 of the cultures from few day old chicks and infected ovaries of adult hens should be regarded as *pullorum*, while the other 10 cultures fall under *gallinarum*. The clinical symptoms and lesions presented by the chicks from which *pullorum* cultures were isolated did not differ materially from those which yielded cultures of *gallinarum*. A diagnosis of infection with either *gallinarum* or *pullorum* in very young chicks should, therefore, not be made, unless fermentation tests have been carried out, as well as serological tests.

The fermentation reactions given in Table 25 include the rhamnose test of Bitter, Weigmann and Habs (1926), the glycerin-fuchsin-broth test of Stern (1916) and the *d*-tartrate test of Jordan and Harmon (1928). By using solid agar media advised by Jordan and Harmon more clear-cut results were obtained than with the fluid media of Silberstein (1931); in positive reactions the colour of the (phenol-red) was changed yellow by the acid formed. The the extent to which this discolouration of the agar occurred varied even with different strains of the same organism; stab cultures were made and the discolouration started from the inoculum, spreading from this point in all directions. In some cases barely a quarter of the medium was changed, while in others as much as a half or three-quarters had turned yellow.

Fifty of the strains labelled *dublin* gave negative tests with Bitter's rhamnose and Stern's glycerin-fuchsin-broth; with the d-tartrate test of Jordan and Harmon the indicator was changed yellow about half-way down the tubes (+ +) in 14 cultures, and about one quarter down the tube (+) in the remaining 36 cultures—47 of the cultures were not tested with these media. Out of 97 cultures, 76 were arabinose negative and 21 were positive after 5 days incubation; 79 were rhamnose positive and 18 were negative after 48 hours incubation. But the rhamnose was generally fermented after 4 or 5 days in the incubator. The antigenic structure of all the 97 strains was identical with that of *S. enteritidis* var. *dublin*.

TABLE 26.

Summary of results obtained with the antigenic analysis of 318 *strains of* Salmonellas *isolated from domestic animals and birds in South Africa.*

Type of *Salmonella*.	No. of Strains Studied.	Origin of Organisms.	"O"—Antigen.	"H"—Antigen.		
				Type (Specific).		Group (non-specific).
				a—Phase.	*β*—Phase.	
Enteritidis var. *dublin*	97*	96 Calves and 1 human	IX, (XII)	*gp*	—	—
Enteritidis	3	Calves	IX, (XII)	*gom*	—	—
Typhi	1	Chick	IX, (XII)	*d*	(*j*?)	—
Gallinarum	149	139 Adult fowls, 10 few day old chicks	IX, (XII)	—	—	—
Pullorum	45	42 Chicks, 3 ovaries of hens	IX, (XII)	—	—	—
Abortus-equi	1†	Adult horse (tendo-vaginitis)	IV, (XII)	—	(*enx.*)	—
Typhi-murium	10	2 Calves, 2 canaries, 1 finches, 1 canary-food, 1 pig, 1 chickens, 1 sheep, 1 rabbits	IV, V (XII)	*i*	—	1, 2, 3.
Typhi-murium var. *Copenhagen* (*Storrs*)	1	Foal (purulent arthritis)	IV, (XII)	*i*	—	1, 2, 3.
Cholerae-suis (Kunzendorf)	6	Pigs	VI, VII (XII)	—	—	1, 3, 4, 5.
Bovis-morbificans	1	Pork	VI, VIII	*r*	—	1, 3, 4, 5.
Anatum	1	Fowls	X, III	*eh*	—	1, 4, 6.
Onderstepoort	1	Sheep	XIV	*eh*	—	1, 4, 5.
Amersfoort	2	1 Chickens, 1 adult fowls	VI, VII, (XII)	*d*	*enx*	—

* Two of these strains were non-motile and were, therefore, devoid of H-specific factors *gp*.

† The one strain studied had lost its motility and, therefore, did not contain an H—antigen, factors *enx*.

O—factor (XII) has been recently added by Kauffmann (1935b) and is supposed to occur in a number of different species that are not related.

Since the completion of this paper 10 more strains of *dublin* have been isolated from calves and 17 more strains of *gallinarum* have been obtained from outbreaks of fowl typhoid. In their antigenic structure and biochemical reactions the *dublin* strains correspond to those *dublin* strains described above and the reactions of the *gallinarum* strains resembled those of the *gallinarum* cultures given above. The total number of *dublin* strains therefore, should be 107 and of *gallinarum* 166, and the total number of *Salmonellas* 345.

For comparison five stock strains of *dublin* were studied. Of these Pesch 256, Cambridge 1 and Topley were positive with Stern's glycerin-fuschin-broth, while *dublin* (Knox) and *Paracoli* (Savage) 255 gave a negative reaction. All five reacted negatively with Bitter's rhamnose and positively with Jordan and Harmon's d-tartrate. On comparing the antigenic structure of Cambridge 1 and Topley with that of *dublin* (Knox) by means of agglutination and absorption tests the three cultures were found to be identical; both Cambridge 1 and Topley completely exhausted *dublin* (Knox) serum.

Of the three strains which corresponded antigenically to *S. enteritidis* all gave a positive reaction to Bitter's rhamnose, two (cultures 290 and 418) reacted positively with Stern's fuchsin-broth and one (culture 216) failed to change it. All three cultures were positive with Jordan and Harmon's medium. The three stock strains, M.7. and D.5, and Weybridge gave a positive reaction with Bitter's rhamnose, Stern's fuchsin-broth and Jordan and Harmon's d-tartrate.

Of the 13 strains which were antigenically identical with *typhi-murium* (Glasgow) all were Stern and d-tartrate positive, twelve were Bitter positive and one (culture 357) was Bitter negative. Eight of the cultures were inosite positive and two (strains 357 and 502) were negative; nine were positive and one (strain 357) was negative with rhamnose, while all the ten tested fermented arabinose. Culture 357 was both rhamnose and inosite negative. Moreover, a complete mirror absorption test performed with culture 357 (Table 20c) showed without doubt that it is a strain of *typhi-murium*. Of the *typhi-murium* stock strains tested all five were Bitter and d-tartrate positive, three (Mutton 74, Glasgow and Weybridge) were Stern positive, while two (Binns and Breslau) were Stern negative.

The one strain of *typhi-murium* var. *Copenhagen-Storrs-* (culture 478) studied gave positive Bitter, Stern and d-tartrate reactions. It fermented both inosite and maltose, but not arabinose; while the stock strain *Storrs* 19500 fermented arabinose and inosite, but not maltose; it was also Bitter and d-tartrate positive, but Stern negative. The two strains of *Copenhagen* (659 and 1147) both fermented maltose and arabinose but not inosite; both were Bitter, Stern and d-tartrate positive. Antigenically, however, *Copenhagen* (*Storrs*) 478, *Storrs* 19500 and the two strains of *Copenhagen* were identical (Table 12).

For comparison 5 stock strains of *paratyphi-B* were included in the test. Of these three (D.C., Grey and Odense) were Stern positive and two (Schottmuller and Lowestoft) were negative; only one (Grey) was Bitter positive, the other four were negative. All five reacted negatively with Jordan and Harmon's d-tartrate.

The six strains which resembled *cholerae-suis* (Kunzendorf) antigenically were all Bitter and d-tartrate positive but Stern negative.

There was only one culture (strain 391) which had the same antigenic structure as *bovis-morbificans*. Like the original culture

of Basenau it was Bitter, Stern and d-tartrate positive, but the strain of Sladden and Scott differed from it on account of its negative Bitter reaction.

The strain of *anatum* (culture 414) studied gave a positive reaction with Bitter, Stern and d-tartrate, resembling, therefore, the two stock strains (3701 and 3702).

Like *abortus-equi* W.H.2, the one strain (culture 219) studied reacted positively to Bitter and d-tartrate, but negatively to Stern's glycerin-fuchsin-broth. It was found to be anaerogenic.

Both strains of *amersfoort* and the one of *onderstepoort* were positive with Stern, Bitter and d-tartrate.

All the forty *gallinarum* strains tested gave a negative reaction with Bitter and Stern, but a positive one with Jordan and Harmon's d-tartrate. Out of 139 strains 119 fermented arabinose, while 20 failed to do so after 4 days. All the cultures fermented rhamnose, but in the case of 17 of the strains the fermentation was delayed. All were dulcite and maltose positive.

The fifteen strains of *pullorum* tested all failed to react on Bitter, Stern or d-tartrate. Thirty-seven of the 45 strains fermented arabinose, but 8 failed to do so. All the 45 strains fermented rhamnose, but in 10 the reaction was delayed. All were dulcite and maltose negative, and all 45 strains were anaerogenic. (Recently a few aerogenic strains were isolated.)

For the differentiation of *S. gallinarum* and *S. pullorum* the cysteine-gelatin medium of Hinshaw and Rettger (1936) was tried, but the organisms failed to grow in the medium so that no change could be detected in the tubes. The cysteine used was freshly prepared.

Only one strain of *S. typhi* was studied. The original culture, obtained from a chicken by Martinaglia, was found to be composed of a mixture of two organisms when I received it; the one non-motile and the other motile. After " single-celling ", the non-motile culture was labelled " strain 207 " and the motile one " strain 208 ". The former resembled *pullorum* and *gallinarum* antigenically (Table 22) and *pullorum* biochemically; while culture 208 was found to have the same antigenic structure as *typhi* (Table 21). The fermentation reactions of the latter, however, did not altogether correspond to those of the stock strains of *typhi* employed. Like *typhi*, culture 208 was Bitter and Stern negative, and d-tartrate positive. Four years ago culture 208 fermented dulcite slowly, and maltose within 24 hours, but at present it fails to ferment dulcite and the fermentation of maltose is delayed for about five days. All the stock strains of *typhi* used fermented maltose within 24 hours, but failed to ferment dulcite. The fermentation reactions of culture 208 are, therefore, not quite typical of *typhi*, but its antigenic structure is identical with that of this bacterium. When it was found that a change had occurred in the fermentation reactions of culture 208 after four years, the serological tests were repeated. The results of these tests show that the antigenic structure of culture 208 has remained unaltered, and that it still resembles *typhi*.

When the biochemical reactions of the different types of *Salmonellas* studied are compared with their serological reactions very marked differences may be exhibited by several strains belonging to the same serological type. For example, some striking variations in their fermentation reactions were manifested by the four strains of *typhi-murium var. storrs* (*Copenhagen*) studied; the one strain 478 described by me and the other 3 stock strains. All four gave positive Bitter and d-tartrate tests; three (*Storrs* 478 and *Copenhagen* 659 and 1147) were Stern positive and fermented maltose, while one (*Storrs* 19500) gave a negative reaction to these tests. Two (*Storrs* 478 and 19500) were inosite positive and two (*Copenhagen* 659 and 1147) were negative. All four were antigenically identical (Table 12).

Analogous variations have been observed in the case of *typhi-murium*, *paratyphi-B*, *enteritidis*, *enteritidis* var. *dublin*, *bovis-morbificans* and, to a lesser degree with *typhi*, *pullorum* and *gallinarum*.

SUMMARY.

Altogether 318 strains of *Salmonella* from different species of animals have been studied. The antigenic analysis obtained is summarised in Table 26 and the biochemical reactions are given in Table 25.

The results recorded in Tables 25 and 26 therefore, show that the biochemical reactions of *Salmonellas*, although very useful as complementary tests, cannot be solely relied upon for the identification of the type or species of organism. Many of the types which were indistinguishable on serological grounds were found to differ in their fermentation reactions, whereas others which differed markedly in their antigenic composition showed identical biochemical reactions. For the recognition of the type of *Salmonella* studied, reliance can be placed only on the antigenic analysis of the bacterium by means of carefully performed serological tests.

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Type of Organism.	No. of Cultures Tested.	Bitter.	Stern.	d. Tartrate (Jordan & Harmon).	No. of Cultures Tested.
Enteritidis var. *dublin*	50	50	50—	36+ 14+++	97
Dublin 154	1	—	—	+	1
Dublin 170	1	—	—	++	1
Dublin 175	1	—	—	++	1
Dublin 283	1	—	—	+	1
Dublin 295	1	—	—	++	1
Dublin 303	1	—	—	+	1
Dublin 329	1	—	—	+	1
Dublin 342	1	—	—	+	1
Dublin 441	1	—	—	+	1
Dublin 445	1	—	—	+	1
Dublin Pesch. 258	1	—	++	++	1
Dublin Knox (2206A)	1	—	—	++	1
Dublin Cambridge 1	1	—	++	++	1
Dublin Topley	1	—	++	+	0
Paracoli savage 255	1	—	—	++	—
Enteritidis 216	1	+	—	++	1
Enteritidis 290	1	+	++++	++++	1
Enteritidis 418	1	+	++++	+	1
Enteritidis M. 7	1	+	++++	++	1
Enteritidis D. 5	1	+	++++	+	0
Enteritidis Weybridge	1	+	++++	+	0
Enteritidis Jena	1	+	++++	++	0
Enteritidis var. *Rostock* 3747	1	—	—	+	0
Enteritidis var. *Moscow*	1	+	—	+	0
Enteritidis var. *Blegdam*	1	+	—	++	0
Enteritidis var. *Essen*	3	+	++++	++	0
Typhi	2	—	—	++	2
Typhi 208	1	—	—	++	1
Typhi Ty2	1	—	—	++	1
Typhi-murium	13	12+ 1—	13	5 + 8++++	10
Typhi-murium 190	1	+	++++	++++	1
Typhi-murium 192	1	+	++++	++++	1
Typhi-murium 176	1	+	++	+	1
Typhi-murium 177	1	+	++++	+	1
Typhi-murium 357	1	—	++++	+	1
Typhi-murium 502	1	+	++	++	1
Typhi-murium 234	1	+	++++	+	1
Typhi-murium var. *Storrs* 478. *Copenhagen*	1	+	++++	+	1
Typhi-murium V. *Storrs* 19500	1	+	—	+	1
Typhi-murium V. *Copenhagen* 659	1	+	++++	+	1
Typhi-murium V. *Copenhagen* 1147	1	+	++++	+	1
Typhi-murium (*Glasgow*)	1	+	++++	+	1
Typhi murium (*Breslau*)	1	+	—	+	1
Typhi-murium (*Mutton*) 74	1	+	+	+	1

TABLE 25.

Fermentation reactions of the different types of Salmonella *studied.*

Glucose.	Lactose.	Dulcite.	Saccharose.	Mannite.	Maltose.	Arabinose.	Rhamnose.	Inulin.	Inosite.
+	—	+	—	+	+	21+ 76—	79+ 18—	—	—
+	—	+	—	+	+	—	+	—	—
+	—	+	—	+	+	—	+	—	—
+	—	+	—	+	+	—	+	—	—
+	—	+	—	+	+	—	+	—	—
+	—	+	—	+	+	—	+	—	—
+	—	+	—	+	+	+	+	—	—
+	—	+	—	+	+	+	+	—	—
+	—	+	—	+	+	—	—	—	—
+	—	+	—	+	+	—	—	—	—
0	0	0	0	0	0	0	0	0	0
+	—	+	—	+	+	—	+	—	—
+	—	+	—	+	+	—	+	—	—
0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0
+	—	+	—	+	+	+	+	—	—
+	—	+	—	+	+	+	+	—	—
+	—	+	—	+	+	+	+	—	—
+	—	+	—	+	+	+	+	—	—
0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0
⊥	—	—	—	⊥	⊥	—	—	—	—
⊥	—	⊥*	—	⊥	⊥	—	—	—	—
⊥	—	—	—	⊥	⊥	—	—	—	—
+	—	+	—	+	+	+	9 + 1—	—	8 + 2—
+	—	+	—	+	+	+	+	—	+
+	—	+	—	+	+	+	+	0	0
+	—	+	—	+	+	+	+	—	+
+	—	+	—	+	+	+	—	—	+
+	—	+	—	+	+	+	+	—	—
+	—	+	—	+	+	+	+	—	+
+	—	+	+	+	+	—	+	—	+
+	—	+	—	+	—	+	+	—	+
+	—	+	—	+	+	+	+	—	—
+	—	+	—	+	+	+	+	—	—
+	—	+	—	+	+	+	+	—	+
+	—	+	—	+	+	+	+	—	+
+	—	+	—	+	+	+	+	—	+

169-170a 169-170 b 169-170c

Salicin.	Galactose.	Sorbite.	Laevulose.	H_2S Production.	Indol.	Litmus Milk.	Motility.	Species of Animal or Source from which Culture was obtained.
—	+	+	+	+	—	Alk	94+ 3 —	Calves 96, Human 1.
—	+	+	+	+	—	Alk	+	Calf.
—	+	+	+	+	—	Alk	+	Calf.
—	+	+	+	+	—	Alk	+	Calf.
—	+	+	+	+	—	Alk	—	Calf.
—	+	+	+	+	—	Alk	+	Calf.
—	+	+	+	+	—	Alk	+	Calf.
—	+	+	+	+	—	Alk	+	Calf.
—	+	+	+	+	—	Alk	+	Calf.
—	+	+	+	+	—	Alk	+	Calf.
—	+	+	+	+	—	Alk	+	Calf.
0	0	0	0	0	0	0	0	0
—	+	+	+	+	—	Alk	+	0
—	+	+	+	+	—	Alk	+	Calf.
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
—	+	+	+	+	—	Alk	+	Calf.
—	+	+	+	+	—	Alk	+	Calf.
—	+	+	+	+	—	Alk	+	Calf.
—	+	+	+	+	—	Alk	+	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	+	0	0	+	0
—	⊥	⊥	⊥	+	—	Neut.	+	0
—	⊥	⊥	⊥	+	—	Neut.	+	Chicken.
—	⊥	⊥	⊥	+	—	Neut.	+	0
—	+	+	+	+	—	Alk	+	0
—	+	+	+	+	—	Alk	+	Calf.
—	+	+	+	+	—	Alk	+	Pig.
—	+	+	+	+	—	Alk	+	Canary.
—	+	+	+	+	—	Alk	+	Grain for canary.
—	+	+	+	+	—	Alk	+	Chickens.
—	+	+	+	+	—	Alk	+	Calf.
—	+	+	+	+	—	Alk	+	Sheep.
—	+	+	+	+	—	Alk	+	Foal (arthritis).
—	+	+	+	+	—	Alk	+	0
—	+	+	+	+	—	Alk	+	0
—	+	+	+	+	—	Alk	+	0
—	+	+	+	+	—	Alk	+	0
—	+	+	+	+	—	Alk	+	0
—	+	+	+	+	—	Alk	+	0

169-170b

169-170c

Type of Organism.	No. of Cultures Tested.	Bitter.	Stern.	d. Tartrate (Jordan & Harmon).	No. of Cultures Tested.
Typhi-murium (*Weybridge*)	1	+	++++	+	0
Typhi-murium (*Binns*)	1	+	—	+	0
Paratyphi B. (*Schotmuller*)	1	—	—	—	0
Paratyphi B. D.C.	1	—	++++	—	0
Paratyphi B. (*Lowestoft*)	1	—	—	—	0
Paratyphi B. (*Grey*)	1	+	+	—	0
Paratyphi B. var. *Odense* 8085	1	—	+	—	0
Cholerae-suis 168 (*Kunzendorf*)	1	+	—	+	1
Cholerae-suis 365 (*Kunzendorf*)	1	+	—	+	1
Cholerae-suis 381	1	+	—	+	1
Cholerae-suis 382	1	+	—	+	1
Cholerae-suis 383	1	+	—	+	1
Cholerae-suis 384	1	+	—	+	1
Cholerae-suis S.F. 8	1	+	—	+	1
Cholerae-suis (*Kunzendorf*)	1	+	—	+	0
Cholerae-suis (*America*)	1	+	—	++++	0
Bovis-morbificans 391	1	+	++++	+	1
Bovis-morbificans (*Basenau*)	1	+	++++	++++	0
Bovis-morbificans (*Sladden-Scott*)	1	—	++++	++++	0
Amersfoort 336	1	+	++++	+	1
Amersfoort 359	1	+	++++	++++	1
Onderstepoort 282	1	+	++++	++++	1
Anatum 414	1	+	++++	+	1
Anatum 3701	1	+	++++	0	1
Anatum 3702	1	+	+++	0	0
Abortus-equi 219	1	+	—	+	1
Abortus-equi W.H. 2	1	+	—	+	0
Gallinarum	40	0	0	++	149
Gallinarum 29	1	—	—	++	1
Gallinarum 360	1	—	—	++	1
Gallinarum 415	1	—	—	++	1
Gallinarum 249	1	—	—	++	1
Gallinarum 324	1	—	—	++	1
Gallinarum 340	1	—	—	++	1
Pullorum	15	—	—	—	45
Pullorum 207	1	—	—	—	1
Pullorum 317	1	—	—	—	1
Pullorum 322	1	—	—	—	1
Pullorum 337	1	—	—	—	1
Pullorum 436	1	—	—	—	1

Stern : +, ++ a
d-tartrate : +, ++
+ = Positive ; ⊥
⊥* = acid, but no

171-172b →

TABLE 25 (*continued*).

Glucose.	Lactose.	Dulcite.	Saccha-rose.	Mannite.	Maltose.	Arabi-nose.	Rham-nose.	Inulin.	Inosite.
0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0
+	—	—	—	+	+	—	+	—	—
+	—	—	—	+	+	—	+	—	—
+	—	—	—	+	+	—	+	—	—
+	—	—	—	+	+	—	+	—	—
+	—	—	—	+	+	—	+	—	—
+	—	—	—	+	+	—	+	—	—
+	—	—	—	+	+	—	+	—	—
0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0
+	—	+	—	+	+	+	+	—	+
0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0
+	—	+	—	+	+	+	+	—	—
+	—	+	—	+	+	+	+	—	—
+	—	+	—	+	+	+	+	—	—
+	—	+	—	+	+	+	+	—	—
+	—	+	—	+	+	+	+	—	—
0	0	0	0	0	0	0	0	0	0
⊥	—	⊥*	—	⊥	⊥	⊥	⊥	—	—
0	0	0	0	0	0	0	0	0	0
⊥	—	⊥	—	⊥	⊥	129⊥ 20—	132⊥ 17⊥d	—	—
⊥	—	⊥	—	⊥	⊥	⊥	⊥	—	—
⊥	—	⊥	—	⊥	⊥	⊥	⊥	—	—
⊥	—	⊥	—	⊥	⊥	⊥	⊥	—	—
⊥	—	⊥	—	⊥	⊥	⊥	⊥	—	—
⊥	—	⊥	—	⊥	⊥	—	⊥	—	—
⊥	—	⊥	—	⊥	⊥	⊥	⊥	—	—
⊥	—	—	—	⊥	—	37⊥ 8 —	35⊥ 10⊥d	—	—
⊥	—	—	—	⊥	—	⊥	⊥	—	—
⊥	—	—	—	⊥	—	—	⊥d	—	—
⊥	—	—	—	⊥	—	—	⊥d	—	—
⊥	—	—	—	⊥	—	⊥	⊥d	—	—
⊥	—	—	—	⊥	—	⊥	⊥	—	—

Stern : +, ++ and +++ indicate different degrees of discoloration, +++ being the most deeply dark lilac colour.
d-tartrate : +, ++ and +++ indicate that ¼, ½ and ¾ respectively of the medium has been changed yellow.
+ = Positive ; ⊥ = acid, but no gas is formed ; ⊥d = delayed fermentation.
⊥* = acid, but no gas formed 4 years ago, negative now.

171–172a

171–172c

Salicin.	Galactose.	Sorbite.	Laevulose.	H_2S Production.	Indol.	Litmus Milk.	Motility.	Species of Animal or Source from which Culture was obtained.
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
—	+	+	+	+	—	Alk	+	Pig (Septicaemie).
—	+	+	+	+	—	Alk	+	Pig (swine fever).
—	+	+	+	+	—	Alk	+	,, ,,
—	+	+	+	+	—	Alk	+	,, ,,
—	+	+	+	+	—	Alk	+	,, ,,
—	+	+	+	+	—	Alk	+	,, ,,
—	+	+	+	+	—	Alk	+	Nat. Coll. of Type Cultures.
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
—	+	+	+	+	—	Alk	+	Pork.
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
—	+	+	+	+	—	Alk	+	Chickens.
—	+	+	+	+	—	Alk	+	Fowls.
—	+	+	+	+	—	Alk	+	Sheep.
—	+	+	+	+	—	Alk	+	Fowl.
—	+	+	+	+	—	Alk	+	0
0	0	0	0	0	0	0	0	0
—	⊥	⊥	⊥	+	—	Alk	—	Horse.
0	0	0	0	0	0	0	0	0
—	⊥	⊥[d]	⊥	+	—	Alk	—	Fowls 137, Turkeys 2, Chickens 10.
—	⊥	⊥	⊥	+	—	Alk	—	Fowl.
—	⊥	⊥	⊥	+	—	Alk	—	Fowl.
—	⊥	⊥	⊥	+	—	Alk	—	Fowl.
—	⊥	⊥	⊥	+	—	Alk	—	5 day old chickens.
—	⊥	⊥	⊥	+	—	Alk	—	Hen.
—	⊥	⊥	⊥	+	—	Alk	—	Fowl.
—	⊥	⊥[d]	⊥	+	—	Neut.	—	Chickens and ovaries of hens.
—	⊥	⊥	⊥	+	—	Neut.	—	Chicken.
—	⊥	⊥	⊥[d]	+	—	Neut.	—	Chickens and ovary of hen.
—	⊥	⊥	⊥[d]	+	—	Neut.	—	Chicken (7 day).
—	⊥	⊥	⊥[d]	+	—	Neut.	—	Chick.
—	⊥	⊥	⊥[d]	+	—	Neut.	—	Chicks.

dark lilac colour.
yellow.

171-172c

171-172b

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