

# Pathogenicity of *Salmonella* Paratyphi A in Pullets

A.O. Ogunleye<sup>1</sup> A.T.P. Ajuwape<sup>1\*</sup> A.I. Adetosoye<sup>1</sup>  
O.G. Ohore<sup>1</sup>

## Keywords

Chicken – *Salmonella* Paratyphi A – Pathogenicity – Nigeria.

## Summary

The pathogenicity of *Salmonella* Paratyphi A isolated at Debiwise Poultry Farm during a fulminating outbreak was tested. Twenty 16-week-old pullets were inoculated orally with 0.5 ml of  $1.3 \times 10^8$  CFU/bird *Salmonella* Paratyphi A, while 20 others of the same age served as uninfected control. By the fourth day postinoculation (p.i.) dullness, ruffled and unkempt feathers, somnolence, yellowish-green diarrhea, decreased water and feed consumption were observed in the infected birds; mortality was 95% by day 16 p.i. Remarkable pathological lesions were recorded between days 7 and 14 p.i. The liver was moderately enlarged, with multiple necrotic foci, and dark brown to bronze coloration. The kidneys were swollen, with widespread focal pale necrotic areas, while the spleen was slightly enlarged. Histopathologically, the proventriculus showed focal glandular necrosis, mononuclear cell infiltration and moderate perivascular leucocytic infiltration. There was mucosal hemorrhage, matting of intestinal villi with reduction of villous height and presence of epithelial debris in the lumen with increased leucocytic infiltration in the lamina propriae. The liver showed moderate, diffuse congestion of the sinusoid and central veins, as well as multiple foci of necrosis of hepatocytes, mononuclear infiltration and perivascular cellular infiltration. There was diffuse lymphoid depletion in the nodule and around the splenic arterioles as well as throughout the parenchyma. The kidneys were congested with tubular epithelial necrosis characterized by karyorrhexis of the nuclei. The organism was recovered from the liver, spleen, heart, heart blood and bone marrow of infected birds. No clinical sign or gross lesion or pathogen was observed in the negative control.

## INTRODUCTION

Domestic poultry constitute the largest single reservoir of *Salmonella* organisms found in nature (12, 13). Salmonellosis is of major economic importance in terms of losses in farm animals and the disease is endemic in commercial poultry flocks in Nigeria (17, 24). Both morbidity and mortality in salmonellosis are highly variable in chickens and are influenced by age, strain susceptibility, nutrition, flock management, and characteristics of exposure (22). In Nigeria, salmonellosis due to *Salmonella* Gallinarum, Pullorum and Typhimurium have been reported (2, 18, 19). *Salmonella enterica* serotypes Typhi and Paratyphi A, B, C are responsible for typhoid fever and paratyphoid enteric fever, respectively (15, 23). A large outbreak of *Salmonella enterica* serotype Paratyphi B infection caused by goats' milk cheese was reported in France (7). Dhillon et al. (8) in a pathogenicity study recorded 30.7, 7.6 and 15% mortalities for *Salmonella* Pullorum, *Salmonella enteritidis* PT5A and *Salmonella enterica* serotype phage type 4 (chicken-

CA), respectively at a dose of  $1.0 \times 10^6$  CFU in specific pathogen-free chickens. These investigators also measured the weight of the *Salmonella*-infected birds on the first, seventh, fourteenth and twenty first days postinoculation as a means of evaluating the subclinical disease and reported a 1.8-12.6% reduction in weight over a period of 28 days. The present investigation was conducted to evaluate the pathogenicity of *Salmonella* Paratyphi A recovered from a poultry outbreak in Ibadan, Nigeria, in 16-week-old pullets.

## MATERIALS AND METHODS

### Experimental chickens

The S and D Aderupoko Farm<sup>®</sup>, Abeokuta hatchery division, donated 150 day-old chicks (Yaffa<sup>®</sup> pullets) out of which 40 pullets were randomly selected for this investigation. The chicks were raised under strict hygienic conditions for 16 weeks at the Teaching and Research Farm University of Ibadan in Nigeria. They were fed *ad libitum* with chicks' mash, without antibiotics, containing 17% crude protein, 2531 metabolizable energy kcal/kg from a day old to the eighth week of age. The growers' mash, containing 18% crude protein, 2448 metabolizable energy kcal/kg, was fed from the ninth week until the end of the investigation. Clean water was provided in troughs *ad libitum*. The chicks were routinely

1. University of Ibadan, Ibadan, Nigeria.

\* Corresponding author

Department of Veterinary Microbiology and Parasitology, University of Ibadan, Ibadan, Nigeria.

E-mail: atpajuwape@yahoo.com

vaccinated using the National Veterinary Research Institute (Vom, Plateau State, Nigeria) vaccines against the Newcastle disease on the first and seventeenth days (intraocular and LaSota vaccines, respectively) and the infectious bursal disease on the ninth day (Gumboro, live attenuated vaccine). Forty pullets were randomly selected and cloaca samples examined bacteriologically for *Salmonella* by standard methods (1, 4, 9).

### *Salmonella* organism

In July 2002 there was a suspected salmonellosis outbreak with high morbidity and mortality in a commercial (Debiwise Poultry) farm near the border of Ibadan Oyo State. Postmortem specimens were taken from the liver, spleen, heart and heart blood of dead chickens for bacteriological investigation (4). The isolates on MacConkey agar were non-lactose fermenting, yellowish glistening colonies and were found to be Gram negative, motile, short rod, oxidase negative, urease negative, citrate negative, indole negative, salicin negative, sucrose negative, glucose positive, sorbitol positive, dulcitol positive, and H<sub>2</sub>S negative (4, 9). A pure colony of *Salmonella* isolate was emulsified with normal saline and tested by the slide agglutination method and was positive for both polyvalent agglutinating antisera O and H (Laboratory Diagnostic Products, UK), and was identified as a *Salmonella* species.

The serological identification was done by inactivating the *Salmonella* isolate as described by Bernard et al. (5). The isolate was inoculated into sterile trypticase soy broth (TSB), and incubated at 37°C overnight. One milliliter of 0.1% formalin was added to 1.0 ml broth culture of *Salmonella* isolate. The mixture was then diluted by another 4.0 ml sterile TSB and incubated at 37°C overnight. The inactivated *Salmonella* isolate was inoculated onto blood agar and MacConkey agar and incubated at 37°C for 48 h. Sera from two weaner rabbits were collected before they were inoculated orally with 1.0 ml of the inactivated *Salmonella* isolate. The pre-inoculation sera were screened using febrile antigen kit (Fortress Diagnostic, Unit 2C Antrum Technology Park, Antrum BT41 IQS Northern Ireland, UK; code No. FEBWC 100, batch No.FC-0507-8) containing *Salmonella* Typhi O, *Salmonella* Typhi H, *Salmonella* Paratyphi AO, *Salmonella* Paratyphi AH, *Salmonella* Paratyphi BO, *Salmonella* Paratyphi BH, *Salmonella* Paratyphi CO and *Salmonella* Paratyphi CH. Positive and negative controls were used for the slide agglutination test. Two weeks after, the sera of the two rabbits were screened for *Salmonella* antibodies by the slide agglutination test using the above febrile antigen kit. The antibody titers to the two antigens (*Salmonella* Paratyphi AH and *Salmonella* Paratyphi AO) that were positive by the slide agglutination test were determined by using the serological diagnosis by Widal reaction previously described by Cruickshank et al. (6) after a slight modification. Briefly, two sets of serial dilutions of each serum sample from the rabbits were constituted in sterile tubes with normal saline (0.85% NaCl), starting from 1:20 to 1:2560, while the ninth tube contained only saline. A volume of 0.5 ml of each of the two slide agglutination test-positive antigens was added to each of the two sets of serially diluted serum from the rabbits. The tubes were incubated at 37°C for 2 h followed by incubation at room temperature for 30 min and examined for agglutination for (H) *Salmonella* Paratyphi AH, while for (O) *Salmonella* Paratyphi AO the tubes were incubated at 37°C for 4 h and kept at 4°C overnight before reading.

### Pathogenicity test

Twenty of the forty randomly selected sixteen-week-old pullets that tested negative for *Salmonella* were housed in one cage designated "experimental birds", while twenty others (control) were

housed in another cage. The weighing balance was used to take the grammetric data of the birds before they were housed. The exercise was repeated on days 7, 14 and 21 postinoculation (p.i.) as a means of evaluating the subclinical disease. A viable count was effected 24 hours after incubation of broth culture of *Salmonella* Paratyphi A at 37°C using the plate count, a method by Miles et al. (16) to determine the concentration of the infective dose. Each experimental bird was fed orally 0.5 ml of the 8 h broth containing  $1.3 \times 10^8$  CFU/ml *Salmonella* Paratyphi A, while the control birds were fed orally with 0.5 ml of sterile TSB each. The two groups were subsequently given growers' rations and water without antibiotics. The birds were observed daily for clinical signs and dead birds were taken for postmortem examination. The only surviving bird was euthanized with chloroform on day 21 p.i. (13).

### Bacteriology and histopathology

Specimens were taken aseptically from the liver, small intestines, spleen, heart and heart blood, and bone marrow of infected birds for bacteriological and histopathological examinations. The bacteriological specimens were stored in the deep freezer at -20°C, in the Department of Veterinary Microbiology and Parasitology at the University of Ibadan, until they were examined by standard methods (1, 4, 9). Histopathological specimens were fixed in 10% formal saline and processed routinely for histopathological examination (21).

### RESULTS

The cultured formalin-inactivated broth culture of the *Salmonella* isolate showed no growth on blood agar and MacConkey agar, respectively, after 48 h incubation. Also the sera from the weaner rabbits were negative when tested with *Salmonella* Typhi O, *Salmonella* Typhi H, *Salmonella* Paratyphi AO, *Salmonella* Paratyphi AH, *Salmonella* Paratyphi BO, *Salmonella* Paratyphi BH, *Salmonella* Paratyphi CO and *Salmonella* Paratyphi CH antigens. However, two weeks after the oral inoculation of the rabbits with formalin-inactivated broth culture of the *Salmonella* isolate, the sera from the inoculated rabbits reacted with only *Salmonella* Paratyphi AO and *Salmonella* Paratyphi AH in the slide agglutination test. The results of the tube agglutination test are presented in Table I. The suspected isolate was identified morphologically, biochemically and serologically as *Salmonella* Paratyphi A.

The experimental birds showed slight inactivity on day 2 p.i. By day 4 p.i. dullness, ruffled and unkempt feather, somnolence, yellowish green diarrhea, decreased water and feed consumption were observed in the infected birds, and the severity increased until day 16 p.i. Mortality in the infected group was 95% by day 16 p.i. The initial mean body weights of infected and control birds were  $0.76 \pm 0.20$  and  $0.78 \pm 0.21$  kg, respectively. However, the weighing scheduled to be repeated on days 7, 14 and 21 p.i. could not be performed because of the rapid course of the disease.

Table I

Results by the tube agglutination test

|          | <i>Salmonella</i> Paratyphi AO<br>(Antibody titer) | <i>Salmonella</i> Paratyphi AH<br>(Antibody titer) |
|----------|--|--|
| Rabbit A | 1:320  | 1:160  |
| Rabbit B | 1:160  | 1:160  |

Remarkable pathological lesions were recorded between days 7 and 14 p.i. The liver was moderately enlarged, with multiple necrotic foci of about 1 mm in diameter, with dark brown to bronze coloration (Figure 1). The kidney was swollen, with widespread focal pale necrotic areas, while the spleen was slightly enlarged. Histopathologically, the proventriculus showed focal glandular necrosis (Figure 2) with mononuclear cell infiltration and moderate perivascular leucocytic infiltration. There was a mucosal hemorrhage and matting of intestinal villi (Figure 3), with reduction of villous height and presence of epithelial debris in the lumen with increased leucocytic infiltration in the lamina propriae. The liver showed moderate, diffuse congestion of the sinusoids and central veins, as well as multiple foci of necrosis of hepatocytes, and mononuclear infiltration and perivascular cellular infiltration. There was diffuse lymphoid depletion in the nodule and around the splenic arterioles (Figure 4) as well as throughout the parenchyma. The kidneys were congested with tubular epithelial necrosis characterized by karyorrhexis of the nuclei (Table II).

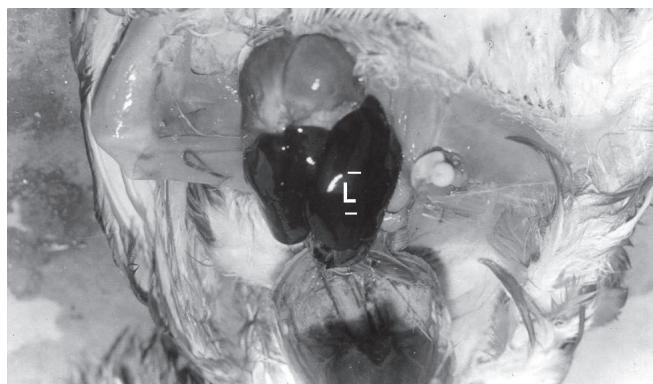
The organism was recovered from the liver, spleen, heart and heart blood, and bone marrow of the infected birds. No clinical sign or gross lesion or pathogen was observed from the negative control.

## DISCUSSION

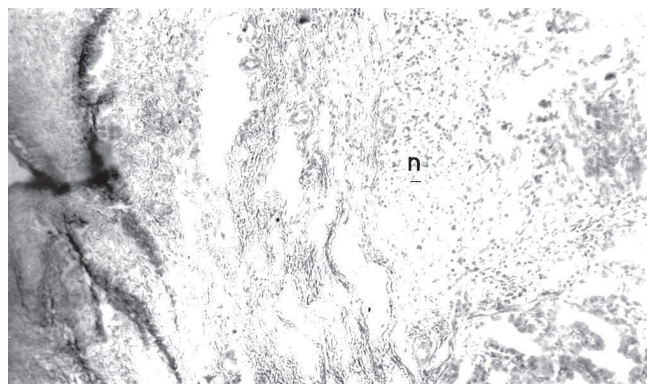
It was not possible to monitor the body weight of the experimental birds beyond the initial data because of the fulminating disease produced by *Salmonella Paratyphi A*. This was unlike the 1.8–12.6% reduction in weight recorded over a period of 28 days p.i. by Dhillon et al. (8). In an earlier study these authors recorded 30.7, 7.6

and 15% mortality for *Salmonella Pullorum*, *Salmonella enteritidis* PT5A and *Salmonella enteritidis* PT4 (chicken CA), respectively, at a dose of  $1.0 \times 10^6$  CFU/ml in specific pathogen-free chickens. This is lower than the 95% recorded in the present investigation. These findings confirm the earlier report that both morbidity and mortality in salmonellosis are highly variable in chickens and are influenced by age, strain susceptibility, nutrition, flock management, and characteristics of exposure (22). *Salmonella* species are known to vary in virulence. For instance *Salmonella enteritidis* PT4 is reported to produce higher mortalities than other *Salmonella enteritidis* phage types (3, 10). The higher mortality found in the current investigation compared to the values recorded elsewhere (8) suggests that the *Salmonella Paratyphi A* may be more virulent than *Salmonella Pullorum* and *Salmonella enteritidis* PT5A. Hence, the organism could be more devastating as experienced in the flock from which it was recovered during the outbreak.

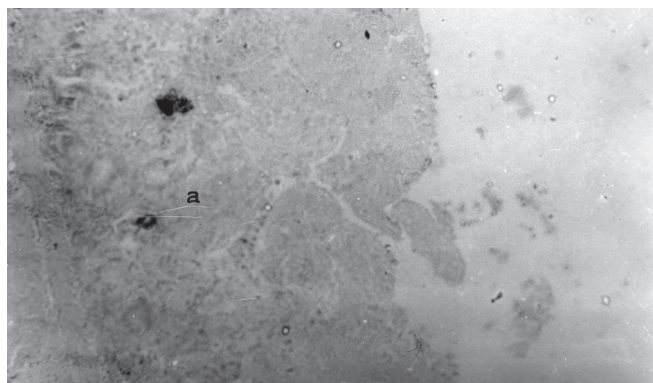
Enteric fever in humans is caused by *Salmonella Typhi* and occasionally by *Salmonella Paratyphi A*, *Salmonella Schottmuelleri* and *Salmonella Hirschfeldii*. The typhoid fever is caused by *Salmonella Typhi*, while the paratyphoid fever is caused by *Salmonella Paratyphi A*, *Salmonella Schottmuelleri* and *Salmonella Hirschfeldii* (7, 15, 20, 23). The clinical features of the paratyphoid fever and typhoid fever are similar but the former is a milder disease (11). In Nigeria, salmonellosis due to *Salmonella Gallinarum*, *Salmonella Pullorum* and *Salmonella Typhimurium* have been reported in poultry (2, 18, 19). However, *Salmonella Paratyphi A* has not been previously reported. It produced a fulminating disease in infected pullets, as found in this study, similar to the clinical observations made in infected birds at Debiwise Poultry Farm in Nigeria. Similarly,



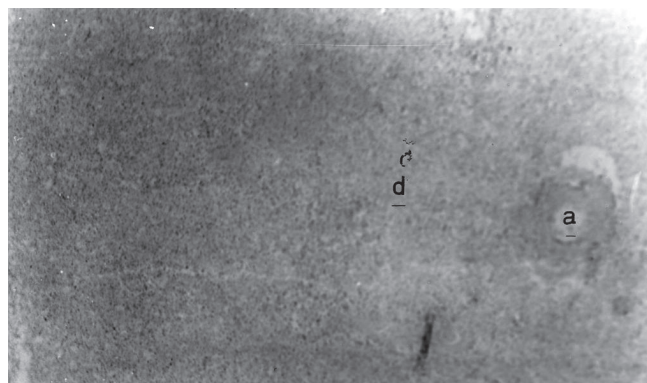
**Figure 1:** Typical bronze color gross lesion of the liver (L) of a chicken from the infected group.



**Figure 2:** Histological section of the proventriculus of an experimental bird showing severe glandular necrosis (n) (x 250).



**Figure 3:** Histological section of the intestine of an experimental bird showing matting of villi, reduction of villous height and glandular necrosis (a) (x 250).



**Figure 4:** Histological section of the spleen from an experimental bird showing severe lymphoid depletion throughout the parenchyma (d) and splenic arteriole (a) (x 250).

Table II

Histopathological findings in dead pullets infected (orally) with 0.5 ml of  $1.3 \times 10^8$  CFU of *Salmonella Paratyphi A*

| Organ          | Lesions  | Severity (num. of pullets affected) |         |        |          |
|----------------|--|-------------------------------------|---------|--------|----------|
| Liver          | Congestion of the sinusoids / central veins                  |                                     | ++ (7)  | – (12) | – (1)*   |
|                | Hepatocyte necrosis  | +++ (10)                            | ++ (5)  | – (4)  | – (1)*   |
|                | Perivascular / parenchymal mononuclear cellular infiltration | +++ (10)                            | ++ (5)  | – (4)  | + (1)*   |
| Intestines     | Villous stunting   | +++ (6)                             |         | – (13) | +++ (1)* |
|                | Increased leucocytic infiltration of lamina propriae         | +++ (6)                             |         | – (13) | +++ (1)* |
|                | Focal area of mucosa hemorrhages                             |                                     | + (3)   | – (16) | + (1)*   |
| Spleen         | Lymphoid hypoplasia around splenic arterioles                |                                     | ++ (12) | – (7)  | + (1)*   |
|                | Lymphoid hypoplasia throughout the parenchyma                |                                     | ++ (2)  | – (17) | – (1)*   |
| Proventriculus | Glandular necrosis / mononuclear cell infiltration           |                                     | + (9)   | – (10) | + (1)*   |
| Kidneys        | Congestion of interstitial vessels                           | +++ (4)                             | ++ (6)  | – (9)  | + (1)*   |
|                | Tubular epithelial necrosis                                  |                                     | ++ (2)  | – (17) | ++ (1)*  |
| Heart          | Congestion of coronary vessels                               |                                     |         | – (18) | + (1)*   |

– Absent

+ Weak

++ Moderate

+++ Marked

\* Lesion observed in the last bird (euthanized) that survived until day 21 postinoculation

a large outbreak of *Salmonella enterica* serotype Paratyphi B infection caused by goats' milk cheese was reported in France (7).

From a consumer's viewpoint, efforts should be made to reduce the spread of this organism in poultry and poultry products in Nigeria in light of the findings of this investigation. The paratyphoid is a zoonotic disease; food products from infected birds pose a risk to human health, and thus hinder public acceptance of poultry products in general, as was the case in the United States of America, Prince Edward Island and Canada (1, 12).

The involvement of *Salmonella Paratyphi A* in poultry disease outbreaks is of public health concern against the backdrop of food safety since this organism has been incriminated in human typhoid (1, 7, 15, 23). This is because *Salmonella Paratyphi A* affects humans; hence infected birds can spread the disease to exposed human beings. *Salmonella Paratyphi A* has been incriminated in human typhoid fever in Nigeria (Akingbola, pers. commun.). It is therefore possible that this organism might have been introduced to the poultry farm from infected farm attendants or contaminated feed and/or water. The fish pond located on the farm may have contributed to the outbreak because a recent report linked aquacultural practices to reservoirs of *Salmonella enterica* Paratyphi B with resultant gastroenteritis in humans (14). Hence it is suggested that livestock farm workers be screened for both typhoid and paratyphoid infections regularly to reduce the incidence of typhoid fever in humans and livestock.

### Acknowledgments

The authors wish to thank the management of S and D, Aderupoko Farm, Abeokuta, Ogun State, Nigeria. The day-old chicks used for this work were supplied through the farm manager, Dr Ajayi.

The Happy Day Veterinary Konsult Oyo, Oyo State of Nigeria, supplied the chicks' mash used for the first three weeks.

### REFERENCES

1. ABOUZEED Y.M., HARIHARAN H., POPPE C., KIBENGE F.S.B., 2000. Characterization of *Salmonella* isolates from beef, cattle, broiler chickens and human sources in Prince Edward Island. *Comp. Immunol. Microbiol. Infect. Dis.*, **23**: 253-266.
2. ADENIRAN G.A., OYEJIDE A., ALASA M.Y., 1990. Serological survey for pullorum disease in four States of Nigeria. *Trop. Vet.*, **8**: 61-64.
3. BARROW P.A., 1991. Experimental infection of chicken with *Salmonella enteritidis*. *Avian Pathol.*, **20**: 145-153.
4. BARROW G.H., FELTHAM R.K.A., 1993. Cowan and Steel's manual for identification of medical bacteria, 3rd Edn. Cambridge, UK, Cambridge University Press, 331 p.
5. BERNARD D.D., RENATO D., HERMAN N.E., HEROLD G., 1980. Microbiology including immunology and molecular genetics, 3rd Edn. Philadelphia, PA, USA, Herper and Row Publishers, 1270 p.
6. CRUICKSHANK R., DUGUID J.P., MARMION B.P., SWAN R.H.A., 1975. Medical microbiology, 12th Edn. London, UK, Churchill Livingstone, p. 417-418.
7. DESENCLOS J.-C., BOUVET P., BENZ-LEMOINE E., GRIMONT F., DESQUEYROUX H., REBIERE I., GRIMONT P.A., 1996. Large outbreak of *Salmonella enterica* serotype Paratyphi B infection caused by a goats' milk cheese, France, 1993: A case finding and epidemiological study. *BMJ*, **312**: 91-94.
8. DHILLON A.S., SHIVAPRASAD H.L., ROY P., ALISANTOSA B., SCHABERG D., BANDLI D., JOHNSON S., 2001. Pathogenicity of environmental origin salmonellas in specific pathogen free chick. *Poult. Sci.*, **80**: 1323-1328.
9. EWING W.H., 1986. The genus *Salmonella*. In: Edward and Ewings' Identification of *Enterobacteriaceae*, 4th Edn. New York, NY, USA, Elsevier Science, p. 181-340.

10. GAST R.K., BENSON S.T., 1995. The comparative virulence for chicks of *Salmonella enteritidis* phage type 4 isolates and isolates of phage types commonly found in poultry in the United States. *Avian Dis.*, **39**: 567-574.
11. HOOK E.W., 1979. *Salmonella* species (including typhoid fever). In: Mandell G.L., Douglas Jr R.G., Bennet J.E., Eds, Principles and practices of infectious diseases. New York, NY, USA, Wiley, p. 1256-1269.
12. JEMMI T., DANUSER J., GRIOT C., 2000. Zoonosis as a risk when handling livestock or animal products. *Schweiz. Arch. Tierheilkd.*, **142**: 665-671.
13. JORDAN F.T.W., PATTISON M., 1999. Poultry diseases. London, UK, W.B. Sanders.
14. LEVINGS R.S., LIGHTFOOT D., PARTRIDGE S.R., HALL R.M., DJORDJEVIC S.P., 2006. Aquariums as reservoirs for multidrug-resistance *Salmonella Paratyphi B*. *Emerging infect. Dis.*, **12**: 507-510.
15. LIU S.-L., SANDERSON K.E., 1995. The chromosome of *Salmonella Paratyphi A* is inverted by recombination between *rrnH* and *rrnG*. *J. Bacteriol.*, **177**: 6585-6592.
16. MILES A.A., MISRA S.S., IRWIN J.O., 1938. The estimation of the bactericidal power of the blood. *J. Hyg. Comb.*, **38**: 732-748.
17. MOLOKWU J.U., SHUAIBU Y., BANYIGI S.A., 1989. Concurrent outbreak of salmonellosis, chronic respiratory diseases and Newcastle disease in a broiler flock. *Zariya Vet.*, **4**: 123-126.
18. OBOEGBULEM S.I., ORAJAKA L.J., OKOYE J.O.A., EROJIKWE E.E., 1980. Fowl typhoid outbreak in university laying flocks: Case report. *Nig. vet. J.*, **9**: 24-26.
19. OKOYE J.A.O., EROJIKWE E., 1988. An outbreak of fowl paratyphoid in Nigeria. *Zariya vet.*, **3**: 90-92.
20. OJO M.O., 1993. Manual of pathogenic bacteria. Ibadan, Nigeria, Shanesson, 412 p.
21. RAPHAEL S.S., 1976. Lynch's medical laboratory technology, 3rd Edn. Philadelphia, PA, USA, WB Saunders, p. 876-933.
22. SNOEYENBOS G.H., 1978. Pullorum disease. In: Hofstad M.S., Calnek B.W., Helmboldt C.F., Reid W.M., Yoder Jr H.W., Eds, Diseases of poultry, 7th Edn. Ames, IA, USA, Iowa State University Press, p. 80-100.
23. THRELFALL E.J., SKINNER J.A., WARD L.R., 2001. Detection of decreased *in vitro* susceptibility to ciprofloxacin in *Salmonella enterica* serotypes Typhi and Paratyphi A. *J. Antimicrob. Chemother.*, **48**: 735-748.
24. UGOCHUKWU E.I., 1982. Post vaccination disease outbreak. *Nigerian vet. J.*, **11**: 24-28.

Accepté le 27.10.2006

## Résumé

**Ogunleye A.O., Ajuwape A.T.P., Adetosoye A.I., Ohore O.G.**  
Pathogénicité de *Salmonella Paratyphi A* chez des poulettes

La pathogénicité de *Salmonella Paratyphi A*, isolée dans l'élevage de volailles de Debiwise lors d'un foyer aigu, a été testée. Vingt poulettes âgées de seize semaines ont été inoculées oralement avec  $1,3 \times 10^8$  UFC/volaille de *Salmonella Paratyphi A*, et vingt autres du même âge ont servi de témoins non infectés. Quatre jours après l'inoculation, les animaux infectés ont présenté de l'apathie, des plumes ébouriffées ou entremêlées, de la somnolence, des diarrhées vert jaunâtre, une baisse de la prise d'eau et d'aliments ; la mortalité a été de 95 p. 100, seize jours après l'inoculation. Des lésions importantes ont été enregistrées entre les septième et quatorzième jours après l'inoculation. Le foie était hypertrophié, d'aspect brun sombre à bronze, et avait de multiples lésions nécrotiques. Les reins étaient inflammés, avec des zones nécrosées étendues claires et focales, et la rate avait légèrement grossi. A l'analyse histopathologique, le proventricule a présenté des nécroses glandulaires focales, une infiltration des cellules mononucléaires et une infiltration leucocytaire périvasculaire modérée. Une hémorragie des muqueuses a été observée, ainsi qu'un ternissement des villosités intestinales avec réduction de leur taille, et la présence de débris épithéliaux dans la lumière avec augmentation de l'infiltration leucocytaire dans la lamina propria. Dans le foie, une congestion modérée et diffuse de la sinusoiide et de la veine centrale était présente, ainsi que de nombreux foyers de nécroses hépatocytaires, des infiltrations mononucléaires, et cellulaires et périvasculaires. Il y avait une déplétion lymphoïde diffuse dans le nodule et autour des artérioles liénales, ainsi que sur tout le parenchyme. Les reins étaient congestionnés avec des zones de nécrose épithéliale tubulaire caractérisées par une caryrrhexis des noyaux. *Salmonella* a été trouvée dans le foie, la rate, le cœur, le sang du cœur et la moelle osseuse des oiseaux infectés. Ni signe clinique, ni lésion macroscopique, ni agent pathogène n'ont été observés chez les témoins.

**Mots-clés :** Poulet – *Salmonella Paratyphi A* – Pouvoir pathogène – Nigeria.

## Resumen

**Ogunleye A.O., Ajuwape A.T.P., Adetosoye A.I., Ohore O.G.**  
Patogenicidad de *Salmonella Paratyphi A* para pollas

Se examinó la patogenicidad de *Salmonella Paratyphi A*, aislada de la Cría de Debiwise durante un brote fulminante. Veinte pollas de 16 semanas fueron inoculadas oralmente con  $1,3 \times 10^8$  UFC/ave de *Salmonella Paratyphi A*, mientras que otras 20 pollas de la misma edad sirvieron como controles no infectados. Al día cuatro post inoculación (p.i.) se observaron mareos, plumas erizas y mal cuidadas, somnolencia, diarrea verde-amarilla, disminución en el consumo de agua y alimento en las aves infectadas; la mortalidad fue de 95% al día 16 p.i. Importantes lesiones patológicas se registraron entre los días 7 y 14 p.i. El hígado estaba moderadamente aumentado, con focos necróticos múltiples y coloración bronce a bronce oscuro. Los riñones estaban hinchados, con zonas necróticas focales dispersas, mientras que el bazo estaba ligeramente aumentado. Histopatológicamente, el proventriculo mostró necrosis glandular focal, infiltraciones de células mononucleares e infiltraciones leucocíticas peri vasculares moderadas. Se observó una hemorragia en las mucosas, aplastamiento de las vellosidades intestinales con reducción de la altura de las vellosidades y presencia de restos epiteliales en el lumen con infiltración leucocítica aumentada en la lámina propia. El hígado mostró congestión difusa moderada de las venas central y sinusoidal, así como múltiples focos de necrosis de hepatocitos, infiltración mononuclear e infiltración celular peri vascular. Se observó una reducción linfoide difusa en el nódulo y alrededor de las arteriolas del bazo, así como en el parénquima. Los riñones estaban congestionados con necrosis epitelial tubular, caracterizada por cariorexis del núcleo. El organismo se recuperó en el hígado, bazo, corazón, sangre del corazón y médula ósea de las aves infectadas. No se observaron signos clínicos o lesiones macroscópicas o patógenos en las aves control negativas.

**Palabras clave:** Pollo – *Salmonella Paratyphi A* – Patogenicidad – Nigeria.