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Necropsy Findings, Isolation and Identification of *Salmonella pullorum* in Layer Chickens in Sumedang, Indonesia: A Case Report

(*LAPORAN KASUS: TEMUAN NEKROPSI SERTA ISOLASI DAN IDENTIFIKASI SALMONELLA PULLORUM PADA AYAM PETELUR DI SUMEDANG, INDONESIA*)

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ABSTRACT

Pullorum disease or Chalky feces, caused by *Salmonella pullorum* bacteria, is a highly contagious bacterial infection that significantly affects commercial poultry, particularly in developing countries. This study was aimed to report a case of pullorum disease in commercial layer chickens in Sumedang, West Java, Indonesia. The diagnostic approach included clinical examination based on clinical signs, followed by pathological and microbiological identification. Clinical signs observed were lethargy, white diarrhea (chalky feces) and decreased egg production. Necropsy findings revealed pathological changes including hepatomegaly, splenomegaly, caseous masses in the caecum and joints, oophoritis and white nodules in the heart. Additional anatomy pathological findings included tracheal hyperemia and malacia in the brain. Bacterial isolation and identification were conducted from internal organs using MacConkey Agar, followed by Gram staining and biochemical tests including Kligler's Iron Agar (KIA), Motility Indole Urea (MIU) and Simmons's Citrate Agar. The results confirmed the presence of *S. pullorum* bacteria, characterized by non-motile, Gram-negative bacilli. These findings demonstrate that *S. pullorum* can cause systemic infection affecting multiple organs. Based on pathological and microbiological examinations, it was concluded that the disease case in the layer chicken samples was caused by chronic infection of *S. pullorum*. This report highlights the importance of early diagnosis and improved biosecurity measures to prevent outbreaks, reduce mortality rates and minimize economic losses in Chicken farming.

Keywords: *Salmonella pullorum*; Chalky feces; Layer chicken; Necropsy; Bacterial Isolation

ABSTRAK

Penyakit pullorum atau penyakit berak kapur yang disebabkan oleh bakteri *Salmonella pullorum*, merupakan infeksi bakteri yang sangat menular dan berdampak signifikan pada unggas komersial, khususnya di negara berkembang. Studi ini bertujuan untuk melaporkan kasus penyakit pullorum pada ayam petelur komersial di Sumedang, Jawa Barat, Indonesia. Metode pemeriksaan ayam ini meliputi pemeriksaan klinis yang didasari dari gejala klinis, serta pemeriksaan patologi anatomi yang dilanjut dengan identifikasi mikrobiologi. Gejala klinis meliputi letargi, berak kapur atau diare putih, dan penurunan produksi telur. Hasil nekropsi menunjukkan adanya perubahan patologis berupa hepatomegali, splenomegali, massa kaseosa pada sekum dan sendi, ooforitis, serta nodul putih pada jantung. Temuan patologi anatomi tambahan meliputi hiperemia trakea dan malasia pada otak. Isolasi dan identifikasi bakteri dilakukan dari organ internal menggunakan *MacConkey Agar*, diikuti dengan pewarnaan Gram dan uji biokimia termasuk *Kligler's Iron Agar* (KIA), *Motility Indole Urea* (MIU) dan *Simmons's Citrate Agar*. Hasil pemeriksaan meneguhkan keberadaan *S. pullorum* yang ditandai dengan morfologi basil Gram-negatif non-motil. Temuan ini menegaskan bahwa akteri *S. pullorum* dapat menyebabkan infeksi sistemik yang menyerang berbagai organ. Berdasarkan temuan anatomi patologis dan uji mikrobiologi, dapat disimpulkan bahwa kasus penyakit pada sampel ayam petelur disebabkan oleh infeksi kronis *S. pullorum.* Temuan ini menjadi penting dalam diagnosis dini dan peningkatan biosekuriti untuk mencegah wabah, menekan angka kematian, serta mengurangi kerugian ekonomi dalam peternakan unggas

Kata-kata kunci: *Salmonella pullorum*; penyakit berak kapur; ayam petelur; nekropsi; isolasi bakteri *Salmonella* sp

INTRODUCTION

Poultry is one of the most popular types of livestock and has promising potential for further development due to its numerous benefits. Poultry plays a significant role in meeting the demand for animal-based food products. According to data from the Indonesian Central Statistics Agency, the average weekly per capita consumption of eggs and chicken meat in Indonesia was 2.152 kg and 0.121 kg, respectively, in 2018 (BPS 2018). Furthermore, in 2023, the poultry population in West Java reached 52,513,258 for laying hens and 770,905,770 for broiler chickens (BPS 2023).

Laying hens in Indonesia are highly susceptible to various diseases that can reduce productivity and cause substantial economic losses. One of the most impactful infectious diseases affecting commercial poultry production is Pullorum Disease (Diyantoro, 2017). The bacterium *Salmonella pullorum* causes this disease and is highly contagious, leading to high mortality rates, particularly in young birds within the first 2–3 weeks of life. In adult poultry, the infection can lead to a significant decline in egg production (Yeakel, 2019). The spread of the disease is often exacerbated by poor sanitation and hygiene practices, which facilitate the transmission of the bacteria among individuals (Nasution *et al*., 2021). Transmission can occur through both direct and indirect contact and may also be vertical, passing from infected hens to their offspring's via the eggs or transovarian transmission (Yeakel, 2019). Infection with *S. pullorum* results in systemic disease, which can be fatal. Infected birds commonly exhibit symptoms such as decreased appetite, white diarrhea, depresssion, and cloacal infections (Shen *et al.*, 2022).

One method to establish a diagnosis

of disease in poultry is through necropsy. Necropsy is a structured and systematic examination of a carcass aimed at identifying the cause of death, confirming a diagnosis, and investigating treatment failure if medication had previously been administered. The term necropsy is derived from the Greek words *nekros*, meaning dead, and *-opsis*, meaning sight (McDonough and Southard, 2017). The necropsy process provides information about the nature of the causative agent, age of the bird and epidemiological characteristics, thereby allowing for a more specific and directed disease diagnosis (Hambal *et al.*, 2019). This article was aimed to report a case of Pullorum Disease in commercial layer chickens in Sumedang, West Java, Indonesia, by presenting the necropsy findings, isolation and identification of *S. pullorum*.

RESEARCH METHODS

Animal Research

The animal used and evaluated in this case study was a single commercial layer hen (*Gallus gallus domesticus*), examined at the Veterinary Teaching Hospital of Universitas Padjadjaran and the Microbiology Laboratory of the Faculty of Medicine at Hegarmanah, Jatinangor Sumedang, West Java. The sample was obtained from a layer poultry farm located in Sumedang Regency. The examined chicken layer was two years old. According to the anamnesis, the farm housed reared approximately 500 layer hens and was experiencing a decline in egg production. The affected hen had been isolated for one month prior to examination due to clinical signs of lethargy and white, chalk-like diarrhea. The housing system was a battery cage type, elevated one meter above the ground, with ammonia levels recorded at approximately 10 ppm which may lead to damage of the respiratory mucosal lining in chickens (Medion, 2021). One week later, two additional hens died in succession, followed by continued mortality within the flock.

Necropsy

The sample chicken was slaughtered in accordance with pathological standards by severing the three essential channels: the digestive tract, respiratory tract, and major blood vessels. The carcass was then placed on a necropsy table, and feathers in the thoracic and abdominal regions were removed. Inspection began with an examination of the feathers, skin and mucosal surfaces of all external and accessible internal organs. An incision was made at the caudal end of the sternum bone, followed by skin reflection cranially to expose the musculus pectoralis major. Dislocation of the coxofemoral joint was performed to facilitate the necropsy process. The costal cartilages (costochondral junctions) were cut to allow evaluation of the air sacs. The thoracic cavity was then fully opened to examine the visceral organs. The heart was assessed through a longitudinal incision to observe potential myocardial hypertrophy or other abnormalities. Further inspection and surface incision were carried out on the spleen, liver and gallbladder to detect any pathological changes. The trachea, bronchi and lungs were collected for evaluation of necrosis, hemorrhage and other abnormalities, followed by a flotation test (lung flotation test) to assess pulmonary conditions. Examination of the gastrointestinal tract was conducted from the esophagus to the cloacae. The lumen was opened and inspected for hemorrhages, obstructions, imternal parasites infestations and other abnormalities. Additionally, the urogenital organs, nervous system and joints were examined. The cranium bones were opened by sawing to allow evaluation of the brain for any signs of neurological lesions or abnormalities (Davis and Morishita, 2006).

Isolation and Identification of Bacteria

Isolation of *Salmonella* sp., was carried out by swabbing and inoculating internal organs of the layer chicken that exhibited anatomical pathological changes onto Mac Conkey Agar (Oxoid CM0115®, Thermo Fisher Sientific, Massachusetts, USA). The colonies obtained (Figure 2) were then subjected to Gram staining, followed by a

series of biochemical tests using *Kligler's Iron Agar* (KIA) (Oxoid CM0033®, Thermo Fisher Scientific, Massachusetts, USA), *Motility-Indole-Urea* (MIU) (Motility Indole Urea Medium®, Thermo Fisher Scientific, Massachusetts, USA) and *Simmons' Citrate Agar* (SCA) (Oxoid CM0155®, Thermo Fisher Scientific, Massachusetts, USA) to confirm the identity of the bacteria (Tortora *et al.*,2018).

RESULT AND DISCUSSION

Gross Pathological Examination

Gross pathological findings obtained from the necropsy of layer chicken samples indicated a single primary etiology, namely a systemic bacterial infection (bacteremia) caused by *Salmonella* spp., which affected the respiratory, digestive and reproductive systems. The characteristics of the lesions (Figure 1) were consistent with salmonellosis, including *caseous* masses in the chicken caecum, yellow foci on the liver surface, ovarian infla-mmation (oophoritis), splenomegaly, hepato-megaly and the presence of caseous exudate in various organs (Penn State University, 2023). In addition, hyperemia of the trachea and necrotic malacia in the brain were also observed.

Salmonellosis is a zoonotic bacterial disease caused by *Salmonella* spp., a genus of Gram-negative, rod-shaped, generally motile, non-spore-forming bacteria. According to Paiva *et al*. (2009), only *S. gallinarum* and *S. pullorum* are non-flagellated, non-motile and obligate pathogens. This infection commonly affects the gastrointestinal tract of poultry such as chickens, ducks and turkeys. It is associated with high morbidity and mortality rates, especially in neonatal (1–7 days) to pre-juvenile (7–21 days) age groups. Clinical manifestations include chalky-white diarrhea adhering to the cloacae, lethargy, drooping wings, ruffled feathers, decreased feed intake and decreased egg production. Gross pathological lesions typically found include:

Marked Hyperemia and Congestion in Trachea. The recruitment of neutrophils and macrophages, along with the production of pro-inflammatory cytokines such as interleukin-6 (IL-6), IL-1β and Tumor Necrosis Factor-α (TNF-α), plays a critical role in the host's innate immune response against pathogens. While this innate immune activation helps the host resist infection, it also leads to infiltration of inflammatory cells and tissue injury, which may result in pulmonary swe-lling and acute hemorrhage. *Salmonella pullorum* has been shown to trigger an excessive pro-inflammatory response, which is largely responsible for the development of pulmonary lesions.

The release of inflammatory cytokines stimulates excessive mucus production by goblet cells in the trachea. This process is further enhanced by goblet cell metaplasia induced by IL-13 and IL-17, leading to mucus hypersecretion and purulent exudetion, thus resulting in a mucoid trachea.

Additional immune responses include the accumulation of neutrophils, which contributes to extracellular matrix degradation, hyperplasia of mucous glands, and increased mucus production. This is accompanied by decreased ciliary clearance and direct epithelial damage in the respiratory tract. Moreover, neutrophils release elastase, which degrades elastin, thereby compromising alveolar wall integrity. This leads to the destruction of alveolar and capillary epi-thelial cells, resulting in capillary rupture and subsequent pulmonary hyperemia and con-gestion (Gramegna *et al.*, 2017; Cheng *et al.*, 2020).

Splenomegaly, Hepatomegaly, Oophoritis, and Intestinal Hemorrhage. *Salmonella* can reach the intestines through oral ingestion (horizontal trans-mission) from contaminated environments, feed or water sources. Its incubation period is approximately 7–14 days. *Salmonella* adheres to intestinal epithelial cells using fimbrial adhesins. Entry into the intestinal mucosa is facilitated by M-cells in the Peyer's patches, followed by internalization by dendritic cells

and uptake by enterocytes. This process is mediated by effector proteins associated with virulence genes. The M-cells absorb and actively transport *Salmonella* across the epithelial barrier by transcytosis.

The uptake of bacterial antigens by M-cells is critical in initiating both mucosal and systemic immune responses. *Salmonella* antigens are delivered to mononuclear phagocytes, including dendritic cells and macrophages. Although macrophages can internalize *Salmonella,* they are unable to eliminate the bacteria, as *Salmonella* can inhibit the fusion of phagosomes with secondary lysosomes, this is an essential mechanism used by macrophages to destroy intracellular pathogens. This ability enhances the intracellular survival of the bacteria.

Salmonella replicates within macrophages in a specialized compartment known as the *Salmonell*a-containing vacuole (SCV). Eventually, it disseminates to mesenteric lymph nodes, leading to bacteremia and systemic invasion of organs such as the liver, spleen, ovaries and gallbladder. Massive activation of the innate immune system (neutrophils and macrophages) leads to the formation of necrotic foci in the liver, which appear as yellowish spots. Neutrophilic infiltration also induces lymphocyte hyper-plasia and vascular dilation in the white and red pulp of the spleen, resulting in splenomegaly. Intestinal hemorrhage occurs due to neutrophil and macrophage infiltration and subsequent inflammation of enterocytes. This is mediated by pro-inflammatory cytokines, including IL-8, TNF-α, and IL-1β, which damage the intestinal mucosal capillary walls and result in vascular leakage.

Neutrophils and macrophages may also infect granulosa cells and theca cells within ovarian follicles, thereby impairing follicular development. This disrupts follicular maturation and leads to follicular atresia (Shaji *et al*., 2023). Neutrophils and macrophages may also infect granulosa cells and theca cells within ovarian follicles, thereby impairing follicular development. This disrupts follicular maturation and leads to follicular atresia (Shaji *et al*., 2023).

White Nodules in the Heart. According to Kumari *et al*. (2013), pathological findings in the heart due to *Salmonella* infection include mild to moderate congestion and hemorrhagic lesions, accompanied by the presence of white nodules. These lesions are attributed to the infiltration of hetero-phils, lymphocytes, and macrophages, indi-cating an active inflamatory response. Additionally, myocardial steatosis may occur as a secondary effect of hepatic necrosis. Hepatocellular stress resulting from liver damage can disrupt lipid metabolism, leading to impaired triglyceride synthesis. Consequently, lipid accumulates within the myocardium, contributing to the development of fatty degeneration in cardiac tissue.

Caseous lesion in the joints. The activity of neutrophils and macrophages contributes to the formation of epithelial cells and multinucleated giant cells, which are also supported by mechanisms involving reactive oxygen species (ROS) and bacterial toxins. These factors lead to coagulative necrosis, in which the lipid membranes of necrotic cells solidify, resulting in the formation of white, cheese-like masses, this lesions referred to as caseous lesions, particularly in the joints.

Malacia Necrosis in The Brain. *Salmonella* invasion can lead to systemic dissemination via the bloodstream (bacteremia), triggering the release of endotoxins such as lipopolysaccharide (LPS). This stimulates the activation and recruitment of neutrophils and macrophages, along with the release of inflammatory mediators including TNF-α, IL-1β, and IL-6. These inflammatory responses contribute to damage to neural cells, including neurons, oligodendrocytes and astrocytes. The interaction with the blood brain barrier (BBB) further exacerbates this process by increasing the expression of chemokines such as IL-8, promoting leukocyte infiltration into the central nervous system. Neuronal injury results in the degradation of the extracellular matrix proteins and the onset of necrosis.

Table 1. Observation of *Salmonella* spp., from liver samples and cultural and biochemical media

Medium/Test	Observation	Interpretation	Reference
MacConkeyAgar (MCA)	Pale white, round, convex colonies with surrounding yellow zone (non-lactose fermenter)	Presumptive *Salmonella* spp.	Wulandari and Suryani (2008)
Kligler's Iron Agar (KIA)	Slant: red (alkaline); Butt: yellow (acid); H_2S: positive (black precipitate); Gas: negative	Glucose fermenter, non-lactose/sucrose fermenter, H_2S producer	Anjung (2016)
Simmons' Citrate Agar	Medium turned blue from green	Citrate utilization positive	Himedia (2019)
Motility Test (MIU)	No spreading growth from stab line	Non-motile	Paiva *et al*. (2009)
Indole Test (MIU)	Yellow ring, no red layer	Indole negative	Sridevi and Konada (2007)
Urea Hydrolysis (MIU)	Medium remained yellow	Urease negative	Mahmudah *et al.* (2016)

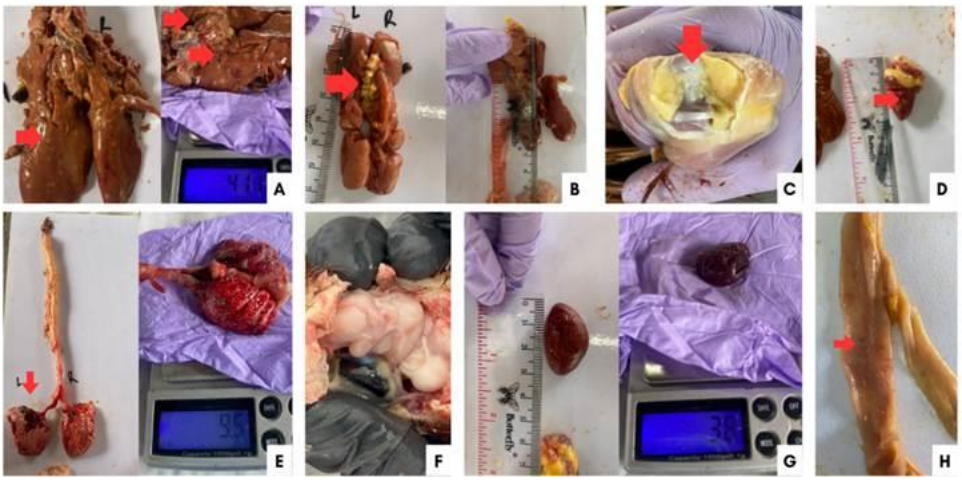

Figure 1. (A) Yellow foci in the liver (red arrow); (B) Underdevelop ovary/egg follicle (red arrow); (C) Caseous lesion in the joint (red arrow); (D) White foci on the heart (red arrow); (E) Hyperemia and congestion in the trachea (red arrow); (F) Malacia necrosis in the brain (red); (G) Splenomegaly; (H) Hemorrhage in the small intestine (red arrow)

This process is mediated in part by macro-phages through the induction of Receptor-Interacting Protein Kinase 1 (RIPK1) and RIPK3 signaling pathways in the brain. It leads to liquefactive necrosis and ulti-mately results in malacia (Yu *et al*., 2024; Van Sorge *et al*., 2011).

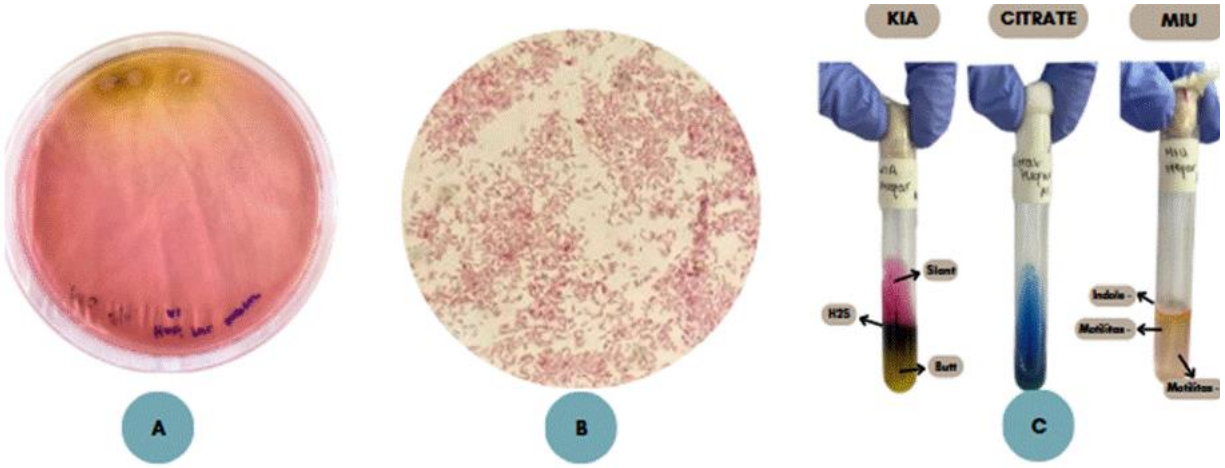

Figure 2. (A) Colonies appeared translucent white, round, convex, and caused the agar surrounding the colonies to turn yellow; (B) Gram staining revealed Gram-negative bacilli; (C) KIA test results showed a red (alkaline) slant, yellow (acidic) butt, and positive H_2S production; citrate test indicated a color change to blue; and MIU test results were negative for motility, indole, and urease.

Microbiological and Biochemical Examination

The microbiological and biochemical examination results were presented in Table 1. The isolation of bacterial colonies on Mac Conkey Agar showed non-lactose fermenting colonies (pale white), a typical characteristic of *Salmonella* spp., which generally appear as round, convex, and transparent colonies with diameters between 2–4 mm (Wulandari and Suryani, 2008).

On Kligler's Iron Agar (KIA), the observed reaction of a red slant and yellow butt with Hydrogen Sulfide (H_2S) production supports the identification of *Salmonella* spp. The alkaline slant indicates the inability to ferment lactose and sucrose, while the acidic butt confirms glucose fermentation. The presence of H_2S (blackening) is a hallmark of many *Salmonella* strains (Anjung, 2016).
The Simmons' Citrate Agar (SCA) turning blue indicates a positive citrate utilization test, which is consistent with the metabolic capability of *Salmonella* to use citrate as the sole carbon source (Himedia, 2019).

The MIU test showed negative results for motility, indole, and urease. While many *S. enterica* serovars are motile, *S. gallinarum* and *S. pullorum* are exceptions, as they are non-flagellated and non-motile (Paiva *et al.*, 2009). The negative indole result is due to the inability of the bacteria to metabolize tryptophan into indole (Sridevi and Konada, 2007), while the absence of urease activity further supports *Salmonella* identification (Mahmudah *et al.*, 2016).

Taken together, the combination of non-lactose fermenting colonies on MCA, H_2S-positive KIA reaction, citrate positivity and negative indole/urease/motility results strongly indicates *S. enterica* subsp. enterica serovar Gallinarum biovar Pullorum (*S. pullorum*). This strain causes Pullorum Disease, characterized clinically by white diarrhea, lameness due to joint inflammation and high mortality in young chicks (Yeakel, 2024).

CONCLUSION

Based on pathological anatomical findings and microbiological tests, it was concluded that the disease case in the layer chicken samples was caused by a chronic infection of *S. pullorum*.

SUGGESTION

This study should be further developed using the polymerase chain reaction (PCR) method to confirm the diagnosis. This

bacterial infection is already present in Indonesia and causes significant economic losses to chicken farmers, highlighting the need for effective prevention and control measures against salmonellosis..

CONFLICT OF INTEREST

The authors declare no conflict of interest related to this study and case report.

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REFERENCES

Anjung MUK. 2016. Identification of *Salmonella* sp., contamination and isolation of bacteriophage as biocontrol in post-harvest handling of vannamei shrimp (*Litopenaeus vannamei*) [*Magister Thesis*]. Bandar Lampung. Universitas Lampung.

BPS [Badan Pusat Statistik], 2018. Rata-rata konsumsi per kapita seminggu beberapa macam bahan makanan penting, 2007-2018. Jakarta: BPS Indonesia. Viewed 15 Mei 2025, from https://www.bps.go.id/id/ statistics-table/1/OTUwIzE=/rata-rata-konsu-msi-per-kapita-seminggu-beberapa-macam-bahan-makanan-penting--2007-2023.html

BPS [Badan Pusat Statistik], 2023. Populasi ternak unggas di Jawa Barat (ekor), 2023. Jakarta: BPS Indonesia, viewed 15 Mei 2025, from https://jabar.bps.go.id/id/statistics-table/2/MjU4IzI=/populasi-ternak-unggas-di-jawa-barat.html

Cheng Y, Sihua Z, Lu Q, Zhang W, Wen G, Luo Q, Shao H, Zhang T. 2020. Evaluation of young chickens challenged with aerosolized *Salmonella pullorum*. *Avian Pathology* 49(5): 507–514.

Davis MF, Morishita TY. 2006. Poultry necropsy basics. *The Poultry Site*. Viewed 6 September 2006, fromm https://www.thepoultrysite.com/articles/poultry-necropsy-basics

Dewi R.P, Wahyuni A, Wibowo M., 2019. qIsolation, Identification, and Serotyping A vibacterium paragallinarum from Commercial Layer with Snot Symptoms *Jurnal Sain Veteriner* 37(2): 115–122.

Diyantoro WS. 2017. Detection of *Salmonella pullorum* and *Mycoplasma gallisepticum* antibodies in broiler chicks (DOC) sold by several companies in Lamongan Regency. *Jurnal Argo Veteriner* 5:.152–157.

Fitri NL, Setiawan D, Hidayatullah M. 2021. Detection of coccidiosis in layer chickens in Sesaot, Narmada District, West Lombok Regency. *Media Kedokteran Hewan* 32(3),97–104.

Gramegna A, Amati F, Terranova L, Sotgiu G, Tarsia P, Miglietta D, Calderazzo MA, Aliberti S, Blasi F. 2017. Neutrophil elastase in bronchiectasis. *Respiratory Research* 18:1–13.

Hambal M, Hasanuddin Z, Zein MSA, Sugito, Armansyah T. 2019. The use of necropsy in diagnosing poultry diseases. *International Journal of Poultry Science* 18(10): 475–480.

Himedia. 2019. *Technical Data*. *Simmons Citrate Agar*. Himedia Laboratories, Thane, Maharashtra, India.

Ihsan B, Abdiani IM, Imran 2018. Detection and identification of *Salmonella* spp., bacteria in milkfish sold at Gusher Market, Tarakan City. *Jurnal Harpodon Borneo* 11(10),46–51.

Kumari D, Mishra SK, Lather D., 2013. Pathomicrobial studies on *Salmonella*

gallinarum infection in broiler chickens. *Veterinary World* 6(10): 725.

Mahmudah R, Baharuddin M, Sappewali S. 2016. Identification of thermophilic bacterial isolates from Lejja hot springs, Soppeng Regency. *Al-Kimia* 4(1): 31–42.

McDonough SP, Southard, TL. 2017. Necropsy techniques for examining diseased animals. *Veterinary Pathology* 54(1): 8–20.

Medion. 2022. Strategies for controlling ammonia in the coop. Viewed 1 Mei 2022, from https://www.medion.co.id/info-medion/strategi-mengendalikan-amonia-di-kandang/

Nasution EZJ, Juwita RP, Karo YK, Agustia H. 2021. Serological detection of *Salmonella pullorum* in poultry in Aceh Province in 2019. *Buletin Veteriner (Medan)* 1: 5–10.

Paiva J, Cavallini J, Silva M, Almeida M, Ângela HL, Junior AB. 2009. Molecular differentiation of *Salmonella gallinarum* and *Salmonella pullorum* by RFLP of *fliC* gene from Brazilian isolates. *Brazilian Journal of Poultry Science* 11(4): 271–275. doi:10.1590/s1516-635x2009000400009

Paiva JB, Penha-Filho RAC, Argüello YMS, Berchieri Jr A, Lemos MVF, Barrow PA. 2009b. Defective mutants of *Salmonella enterica* serovar *gallinarum* in cobalamin biosynthesis are avi rulent in chickens. *Brazilian Journal of Microbiology* 40: 495–504.

Penn State University. 2023. *Salmonellosis – clinical signs and prevention strategies in poultry*. The Poultry Site. Viewed 29 September 2025, dari https://www.thepoultrysite.com/articles/salmonellosis

Retnani EB, Rahmawati DN. 2021. Prevalence of tapeworm infection in traditional laying hens in Panekan Dis-trict, Magetan Regency. *Jurnal Kedokteran Veteriner* 9(1):1–6.

Shaji S, Selvaraj RK. Shanmugasundaram R. 2023. Salmonella infection in poultry: a review on the pathogen and control strategies. *Microorganisms* 11(11): 2814.

Shen X, Zhang A, Gu J, Zhao R, Pan X, Dai Y, Yin L, Zhang Q, Hu X, Wang H. 2022. Evaluating *Salmonella pullorum* dissemination and shedding patterns and antibody production in infected chickens. *BipMeical Centre Veterinary Research* 18: 240.

Sridevi M, Konada VM. 2007. Bioproduction of indole acetic acid by *Rhizobium* strains isolated from root nodules of green manure crop, *Sesbania sesban* (L.) Merr. *Iranian Journal of Biotechnology* 5(3): 178–182.

Wulandari S, Suryani L. 2008. Detection of Salmonella bacteria in fried chicken sold in food stalls and sensitivity patterns to various antibiotics. *Mutiara Medika* 8(2): 101–106.

Tortora GJ, Funke BR, Case CL. 2018. *Microbiology: An Introduction*. 13th ed. London. Pearson.

Van Sorge NM, Zialcita PA, Browne SH, Quach D, Guiney DG, Doran KS. 2011. Penetration and activation of brain endothelium by *Salmonella enterica* serovar *Typhimurium*. *Journal of Infectious Diseases* 203(3): 401–405.

Yeakel SD. 2019. *Pullorum disease in poulltry*. *Veterinary Manual*. Viewed 8 Mei 2025, from https://www.Msdvetmanual.com/poultry/salmonelloses/pullorum-disease-in-poultry

Yu X, Yuan J, Shi L, Dai S., Yue L, Yan M. 2024. Necroptosis in bacterial infections. *Frontiers in Immunology* 15: 1394857.