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Salmonella Pullorum: Now You See Me, Now You Don't — The Paradox of Detection in Chicken Infection Models

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Salmonella enterica serovar Pullorum (*S.* Pullorum) remains one of the most enigmatic host-adapted bacterial pathogens of poultry. Although pullorum disease is considered controlled or eradicated in many parts of the world, experimental and field evidence consistently demonstrate that *S.* Pullorum possesses an extraordinary ability to persist within its avian host while evading conventional detection methods. This paradox is particularly evident in chicken infection models, where birds show pathological, immunological, or serological responses despite failure to recover viable bacteria by standard culture.

Unlike broad-host-range serovars such as *S.* Typhimurium or *S.* Enteritidis, which are optimized for transmission and intestinal colonization across multiple hosts, *S.* Pullorum is strictly adapted to chickens and has undergone extensive genome degradation, including loss of flagellar genes, metabolic pathways, and pro-inflammatory virulence traits (Parkhill et al., 2001; Thomson et al., 2008). This evolutionary specialization favors low-inflammatory, intracellular persistence rather than efficient horizontal spread. As a consequence, *S.* Pullorum frequently establishes infection within macrophages in systemic organs such as the spleen and liver, particularly during early life stages, while remaining largely undetectable in the intestinal tract (Barrow & Freitas Neto, 2011; Wigley et al., 2001).

Numerous experimental infection studies have shown that bacterial counts of *S.* Pullorum decline rapidly after the

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acute phase of infection, often falling below the limit of detection of culture-based methods, even though histopathological lesions, immune cell infiltration, and cytokine responses persist (Berndt et al., 2007; Wigley et al., 2001). This apparent discrepancy has sometimes been interpreted as bacterial clearance; however, accumulating evidence suggests that it instead reflects a disconnect between culturability and persistence. Host immune pressure, nutrient limitation, and intracellular confinement likely push *S.* Pullorum into physiological states characterized by extremely low replication rates or reduced culturability, a phenomenon consistent with viable but non-culturable states described in other host-adapted *Salmonella* serovars (Pinto et al., 2015).

Furthermore, *S.* Pullorum displays inefficient fecal shedding compared with enteric serovars, which severely limits detection through cloacal swabs or environmental sampling in both experimental and field settings (Barrow & Freitas Neto, 2011; Shivaprasad, 2000). Its tendency to localize in immune-privileged or poorly sampled niches, including the reproductive tract, further complicates recovery and contributes to vertical transmission in the absence of detectable horizontal spread. Consequently, negative bacteriology does not equate to absence of infection but reflects the stealth strategy of this pathogen.

The implications for experimental design are substantial. Chicken infection models that rely exclusively on culture-based endpoints risk underestimating persistence, misinterpreting host resistance, or overstating the attenuation of mutant strains. Several studies have demonstrated that molecular detection methods, serology, and immunological readouts can reveal ongoing host–pathogen interactions long after bacteria become non-recoverable by culture (Berndt et al., 2007; Wigley et al., 2001). Thus, failure to isolate *S.* Pullorum should be interpreted as a biological feature of infection rather than an experimental limitation, and should be explicitly acknowledged in study conclusions. Integration of culture-independent approaches, including targeted qPCR, transcriptomic profiling, and emerging spatially resolved -omics technologies, may provide complementary tools to detect low-burden intracellular reservoirs and help distinguish true bacterial clearance from states of non-culturability.

This detection paradox also has broader epidemiological implications. Surveillance systems that rely primarily on bacteriological isolation may create a false sense of eradication, overlooking silent reservoirs maintained through vertical transmission or subclinical carriage. Sporadic re-emergence of pullorum disease in supposedly free regions suggests that *S.* Pullorum may persist below the radar of routine diagnostics, mirroring the challenges observed in experimental models (Shivaprasad & Barrow, 2013).

In this context, *S.* Pullorum exemplifies a broader class of host-adapted pathogens for which invisibility is not synonymous with elimination. Its ability to induce disease, shape host immunity, and persist intracellularly while remaining undetectable challenges traditional definitions of infection success and failure. Recognizing that “now you see me, now you don’t” is an intrinsic feature of *S.* Pullorum biology is essential for accurate interpretation of infection models, effective surveillance, and a realistic understanding of pathogen control in poultry systems.

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Conflict of Interest

The author declares no conflicts of interest.

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