*Salmonella enterica* are Gram negative, nontoxic bacteria that cause disease in several hosts including humans, cattle, pigs, and chickens.\(^1\)

**Cattle**

Cattle are a reservoir of non-typhoidal *S. enterica* serovars (NTS), which can survive and spread within the bovine lymphatic system.\(^1,4\)

Peripheral lymph nodes of cattle are small and difficult to remove during large scale beef production, and can get incorporated into ground beef products.

Salmonella virulence factors and pathogenesis in cattle

Consumption of undercooked meat leads to human non-typhoidal salmonellosis. NTS serovars are estimated to cause 78,000 illnesses, 59,000 deaths, and 4.3 million DALYs lost per year worldwide.\(^5\)

There are currently no effective vaccines to control *Salmonella* in cattle. Existing vaccines confer limited serotype-specific protection.

The aim of this research was to study *Salmonella* pathogenesis in cattle, a naturally affected host species, to better understand the host and bacteriological factors involved in pathogenesis and protection and ultimately inform the design of new vaccines and other intervention strategies for cattle and limit zoonoses.

*Salmonella* Dublin SD3246 was transformed with pPV25.10 to make the bacteria GFP+. GFP+ *S. Dublin* were easily identified by flow cytometry and the plasmid was stably maintained within SD3246, even in the absence of ampicillin for selection.

Calves were challenged orally with GFP+ *S. Dublin* to mimic the natural route of infection.

Calves infected with GFP+ *S. Dublin* showed clinical signs post-infection, such as pyrexia, as expected in calves challenged with wild-type *S. Dublin*.

Host-pathogen interactions were studied in the distal ileum and mesenteric lymph nodes (LN). Where are *Salmonella* during infection?

*S. Dublin* were predominantly extracellular in both the distal ileal mucosa as well as in mesenteric lymph nodes.

What are the consequences of infection for infected bovine cells?

*S. Dublin*-infected cells (GFP+) in the distal ileal mucosa and in mesenteric lymph nodes expressed higher levels of MHCI compared to uninfected cells (GFP-) in the same tissues as well as cells from cognate tissues of uninfected calves.

Thus, the *S. Dublin*-infected cells were macrophage-like antigen presenting cells.

**Almost all** murine infection models are pathogen driven, with pathogen adaptations leading to host disease. In contrast, cattle are a reservoir of non-typhoidal *Salmonella* serovars. For example, *S. Dublin* causes systemic typhoid-like disease and can lead to abortions and *S. Typhimurium* causes acute self-limiting enteritis. Interestingly, chicken-specific *Salmonella* Gallinarum is avirulent in cattle but can invade the bovine gut.

Most of our knowledge about host-pathogen interactions comes from the *mouse typhoid model* of salmonellosis.

Which bovine cells do *Salmonella* infect?

Infected cells in the distal ileal mucosa and mesenteric lymph nodes were identified by flow cytometry using the GFP+ signal from *S. Dublin* and were found to express MHCI, CD40, CD80, CD86, CD1b and CD11c.

However, they did not express CD1.

Thus, the *S. Dublin*-infected cells were macrophage-like antigen presenting cells.

What are consequences of infection for *Salmonella*? *S. Dublin* successfully invaded bovine blood monocyte-derived macrophages and survived within them up to 24 h, with a drop in viability after 6 h but did not actively replicate within them. The rate of replication decreased gradually over 6 h as observed by fluorescence dilution.\(^7\)

References