

Experimental Infection of Egg-laying Hens with *Salmonella enterica* Serovar Enteritidis Phage Type 4 and its Three Mutants

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Abstract :

The emergence of *Salmonella enterica* serovar Enteritidis (*S. Enteritidis*) during the past three decades as major contaminant of eggs and other poultry products caused a surge in human infections. This could have been mediated in part by emerging *S. Enteritidis* strains with enhanced virulence. The overall pathogenicity of *Salmonella* is controlled by numerous genes. To assess the role of a few specific genes thought to contribute to the pathogenicity of *S. Enteritidis* in egg laying hens, we conducted an experimental infection of egg-laying hens, with a wild type (WT) *S. Enteritidis* phage type 4 strain and three mutants (M1, M2, M3). These mutants were produced from the wild type by the inactivation of the *prgH*, *SEN4287*, and *tyrR* genes, respectively. We observed that the WT and the M1 mutant had shorter durations of fecal shedding and faster clearance from internal organs of the infected hens than the M2 and M3 mutants. The isolation rates of the wild type *S. Enteritidis* and the mutants were highest from the ceca, moderately high from the liver and spleen, and lowest from the ovaries of the infected hens. Hep-2 cells attachment assay revealed attenuated attachment for the M1 mutant while the M3 mutant seemed to have enhanced attachment. Colonization of tissues of the infected hens by M1 mutant appears to have been attenuated.

Key Word :

egg-laying hens, experimental infection, mutants, *Salmonella* Enteritidis, pathogenicity

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