Role of narL gene in the pathogenesis of Salmonella Typhimurium

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Abstract

Salmonella Typhimurium (STM) is a facultative anaerobe of zoonotic importance and one of the causative agents of non-typhoidal salmonellosis (NTS). During infection, STM must adapt to the changes in oxygen concentration encountered in the crucial niches of host like gut lumen and intramacrophage environments. But being a chemo-organoheterotroph, STM is capable of obtaining its energy from organic sources via redox reactions. NarL, a transcription factor and the response regulator of the two-component regulatory system NarX/L, gets activated under nitrate rich anaerobic condition. Upon activation, it upregulates the nitrate reduction during anaerobic respiration. However, in this study, we observed a significant attenuation of virulence in the narL-knockout strain of STM, while the respective morphotypes got rescued upon genetic complementation. Along with motility and biofilm forming ability, the mutant strain displayed reduced intracellular replication in either intestinal epithelial cells or monocyte-derived macrophages of poultry origin. Further, in vivo competitive assay in the murine model showed that wild type STM significantly outcompeted its isogenic narL null mutant.

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