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DECODING MlrB EXPRESION DURING *Salmonella Typhimurium* MACROPHAGES INFECTION

Folmer, M. Poema - Brovedan, Marco - Checa, Susana K. - Soncini, Fernando C.

Instituto de Biología Molecular y Celular de Rosario (IBR), CONICET-UNR, Rosario, Argentina.

Contacto: poefolmer@gmail.com

Salmonella Typhimurium is an intracellular pathogen known for its ability to cause gastrointestinal and systemic infections. The success of the pathogen relies on its capacity to adapt and survive in hostile environments, including the potential to form biofilms and to thrive within host cells. These adaptive traits are mediated by various virulence genes encoded within specific regions of the genome, such as the *Salmonella* Pathogenicity Islands (SPIs). Our group focuses on the characterization of regulators of the MerR family. In particular, those that control the transition between motile and sessile lifestyles, such as MlrA, a main activator of CsgD, the master regulator of biofilm formation in enterobacteria. In this context, we identified MlrB, which shares 40% identity and 70% similarity with MlrA. MlrB acts as a virulence factor, promoting *Salmonella* survival inside macrophages. *mlrB* expression is induced inside macrophages and under conditions that mimic the intravacuolar environment of host cells, such as low pH, limited magnesium, and the presence of iron, conditions that also activate the *Salmonella* Pathogenicity Island 2 (SPI-2) gene expression. We observed that *mlrB* transcription is under the cascade regulation of the two-component systems PhoP/PhoQ-PmrA/PmrB. We identified a PmrA-binding box within the promoter region of *mlrB*, suggesting a direct control by PmrA/PmrB. The interaction of PmrA to *mlrB* promoter was confirmed by Electrophoretic Mobility Shift Assays (EMSA), highlighting the crucial role of PmrA in fine-tuning the expression of genes essential for *Salmonella* survival in harsh intracellular environments. Regulation of *mlrB* by PmrA underscores a sophisticated network where *Salmonella* integrates multiple environmental signals to adjust its virulence strategy. Understanding these interactions provides insights into *Salmonella* pathogenesis and constitutes a potential target for the development of novel therapeutic agents.

Palabras clave: *Salmonella* – biofilm – virulence factors – MlrA homolog