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## Modulation of ompW gene expression by the OmpR regulator protein of Yersinia enterocolitica O:9

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**Background:** *Yersinia enterocolitica* is a human enteropathogen – the causative agent of yersiniosis, a zoonotic infectious disease of the gastrointestinal tract. Successful host infection by *Y. enterocolitica* largely depends on the coordinated expression of a multitude of virulence factors and genes involved in the adaptation to the specific environmental niches. It is well documented that the OmpR protein plays a key role in the regulation of genes expression during the bacterial infection process. Our latest proteomic findings showed that OmpR exerts an effect on the abundance of numbers of outer membrane proteins (OMPs), including OmpW production. OmpW belongs to the OmpW/AlkL family of OMPs present in all gram-negative bacteria and participates in resistance to a broad range of chemically diverse quaternary cationic compounds, that are used agriculturally, medically or industrially as cationic surfactants, antiseptics and herbicides. According to the proteomic data we postulated that OmpR could directly or indirectly regulates the transcription of *ompW*. The aim of this study was to reveal the role of OmpR in the regulation of *ompW* expression.
**Material/methods:** The level of OmpW synthesis in the wild and Δ*ompR* mutant strain was determined by differential label-free quantitative proteomic analysis. To examine whether the expression of *ompW* is under the control of OmpR, the chromosomal transcriptional fusion *ompW-lacZYA'* was constructed in the wild-type strain and the Δ*ompR* mutant derivative. The gel electrophoresis mobility shift assay (EMSA) was used to detect OmpR complexes with the regulator sequences of *ompW.*
**Results:** Mass spectrometry analysis indicated that upon growth at 37°C, the OmpR-negative strain exhibited a 2- (at high osmolarity) and 4-fold increase (at low pH) in the level of the OmpW protein. Based on measurements of β-galactosidase activity we found higher *ompW* expression in the *ompR* mutant strain compared to the wild type strain. This upregulation was absent following complementation with the wild-type *ompR* allele on a plasmid. These data confirmed the OmpR-dependent negative regulation of *ompW* gene. Inspection of the regulatory region of *ompW* showed one putative OmpR binding site with 50% identity to the *E. coli* consensus sequence, and 60% identity to the *Yersinia* spp. consensus sequence. Furthermore, electrophoretic mobility shift assays showed that OmpR binds to the promoter region of *ompW,* indicating that this factor directly regulates the transcription of this gene.
**Conclusions:** Our results revealed the involvement of OmpR in the direct, negative regulation of OmpW expression in *Y. enterocolitica.*

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