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Introduction and objectives:
Salmonella Genomic Island 1 (SGI1) is a multidrug resistance integrative mobilizable element specifically mobilized *in trans* by IncC and IncA conjugative plasmids. SGI1 hijacks the plasmid conjugation apparatus to be mobilized. However, these mobile elements are incompatible in a bacterial population. This incompatibility is caused by SGI1 transient replication that involves, besides IncC/IncA factors, the SGI1 regulatory region *sgaCD* (1, 2). SgaCD are functional homologues of the IncC master activator AcaCD proteins. Unlike *acaCD*, the regulation of *sgaCD* expression has not been characterized yet.
The identification of LexA binding motifs on SGI1, especially in the putative promoter region of *sgaCD*, suggested it may constitute a SOS response-regulated region. Moreover, plasmid entry by conjugation as single-strand DNA in recipient bacteria is known to activate transiently the SOS response (3). Therefore, in the present study we assessed if the entry of an IncC plasmid by conjugation could lead to transient activation of the SOS response, resulting in the *sgaCD* expression and other SGI1 genes under its control.
Materials and methods:
We performed β-galactosidase reporter assay to quantify the SOS response activation in transconjugant cells during conjugation based on the model of Baharoglu et al. (3). We also used β-galactosidase reporter assay to quantify *sgaCD* promoter activity in different conditions and using different genetic backgrounds. We realized electromobility shift assays to confirm the physical interaction between LexA and its potential binding site in *sgaCD* promoter. Finally, we performed a RT-qPCR assay to explore the impact of the SOS response on the expression of SGI1 genes.
Results, discussion and conclusion:
We showed that the IncC conjugative entry activates the SOS response. Molecular experiments confirmed that the LexA binding site in the *sgaCD* promoter region is functional, resulting in the SOS-dependent control of *sgaCD* expression. Furthermore, we also showed that nearly all conjugative transfer and maintenance genes harboured by SGI1 are induced by the SOS response activation directly and/or in a SgaCD dependent manner.
Further work is ongoing to confirm the role of the SOS response in the molecular crosstalk between SGI1 and IncC/IncA plasmids through conjugation and maintenance experiments.

Mots clés : SGI1 - SOS response - IncC plasmid - Multidrug resistance.

Références :
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