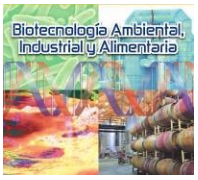
Poster



Involvement of sensor histidine kinases in general stress response of Sphingopyxis granuli strain TFA

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ABSTRACT

Motivation: In their natural habitat, bacteria face constantly changing conditions that can portraiy a challenge to their survival. To overcome this problem, bacteria are able to react to their medium and adapt accordingly to it. One of these responses is the so-called General Stress Response (GSR), an unspecific response to a myriad of different stress signals.

Sphingopyxis granuli strain TFA is an alphaproteobacteria with the ability to use tetralin, an organic solvent with industrial applications, as a carbon and energy source. It is also the first facultative anaerobe described within its genus. These characteristics made TFA an interesting strain from a biotechnological point of view.

In alphaproteobacterial, the regulatory network of the GSR is composed at its most basic level by a sigma factor (EcfG), an anti-sigma factor (NepR), an anti-anti-sigma factor (PhyR) and a sensor histidine kinase that targets the anti-anti-sigma factor. In TFA, EcfG, NepR and PhyR are duplicated and a total of four putative sensor histidine kinases have been found within TFA genome. Previous works by de Dios et al have partially described the regulatory network of the GSR at to the point of the histidine kinases. Therefore, in this work we aim to unravel role that two of these histidine kinases, SGRAN_1655 and SGRAN_2544, have in the regulation of the GSR in TFA.

Methods: The experimental approach for this work consist in the obtention of deletion mutants in the SGRAN_2544 and SGRAN_1165 by the method described by Martínez-García and de Lorenzo. Once the mutants were obtained, We examined their response to several stresses, like high sodium chloride concentration, high heavy metals concentration, hydrogen peroxide treatment and desiccation, by both checking the effect on their growth and quantifying the activity of lacZ fusions to a GSR reporter gene, such as nepR2.

Results: We have been able to succesfully obtain a deletion mutant of one of the two target genes, SGRAN_2544 gene.

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