Titulo : IDENTIFICATION OF A COMPENSATORY BEHAVIOR ASSOCIATED TO CURLI FIMBRIAE IN A Cah-DEFICIENT O157 SHIGA TOXIN-PRODUCING STRAIN DURING BIOFILM FORMATION ON ABIOTIC SURFACES

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Resumo :

Shiga toxin-producing *Escherichia coli* (STEC) are important food-borne pathogens associated with human diseases including mild diarrhea, hemorrhagic colitis and hemolytic uremic syndrome. Ruminants, especially cattle, are the major reservoir of STEC strains. In addition to Shiga toxin, STEC strains can present different virulence factors like several types of adhesins, autotransporter proteins, and other toxins. Biofilm formation has been described as an important characteristic for maintenance and persistence of STEC in the environment and in the host. Moreover, formation of biofilms on various surfaces can serve as an important source and/or vehicle of contamination Several structures such as curli fimbriae and autotransporter protein Cah have been described associated to biofilm formation in STEC. The aim of this study was to evaluate the expression of curli fimbriae gene (*csgA*) during biofilm formation processes on glass and polystyrene surfaces by a *cah* gene-deficient O157 STEC strain (Δ*cah*) that presented greater interaction with these abiotic surfaces compared to the wild-type strain. The wild-type and mutant STEC strains were submitted to interaction assays with glass and polystyrene surfaces for extraction and purification of RNA. Expression of *csgA* gene was assessed by qRT-PCR. Overexpression of curli fimbria by the Δ*cah* strain was detected on both surfaces compared to the wild-type strain. In the Δ*cah* strain expression of *csgA* was 5-fold and 30-fold higher on glass and polystyrene surfaces, respectively. The results obtained suggest that curli fimbriae is deeply related to the compensatory behavior of this Δ*cah* STEC strain during biofilm formation on glass and polystyrene surfaces. Further studies are necessary to determine if this behavior is associated to any gene that regulates curli synthesis and to understand how this compensatory mechanism is triggered.

Palavras chaves: biofilm, Shiga toxin-producing *E. coli*, O157:H7, Cah, curli fimbriae

Agencia de Fomento: CNPq