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Título: *Corynebacterium diphtheriae* OxyR CONTRIBUTES TO FIBRINOGEN, FIBRONECTIN AND COLLAGEN-BINDING, SURVIVAL IN HUMAN RESPIRATORY CELLS AND LETHALITY OF *Caenorhabditis elegans*

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Resumo

The ability to maintain intracellular concentrations of toxic reactive oxygen species (ROS) within safe limits is essential for all aerobic organisms, requiring the constitutive expression of ROS scavenging systems. OxyR is one of the most well-known systems that might be also involved in bacterial virulence, contributing for biofilm formation, toxin production and antimicrobial resistance of some bacterial species. *Corynebacterium diphtheriae* is the main causative agent of diphtheria, a communicable disease that affects primarily the respiratory tract. This pathogen has also been related with invasive infections, such as endocarditis, osteomyelitis and catheter-related infections. It is known that *C. diphtheriae* OxyR (DIP1421) functions as a catalase repressor by a mechanism independent of H_2O_2-induced stress. Nevertheless, the OxyR involvement in *C. diphtheriae* virulence remains unclear. Presently, the role of OxyR in diphtheria bacilli virulence was investigated through the construction of an *oxy*R-interrupted strain (LDCIC-L2). The LDCIC-L2 strain was tested for the abilities to resist to antimicrobial and oxidative agents, bind to fibrinogen, fibronectin and collagen, form biofilm on hydrophilic (glass) and hydrophobic (polystyrene) surfaces, adhere to and survive within human respiratory cells (HEp-2 cells), and to kill the nematode *Caenorhabditis elegans*. The *oxy*R interruption caused a significant reduction in the bacterial binding activity to the plasma and extracellular matrix proteins. However, it did not affect the biofilm formation on abiotic surfaces or the antimicrobial susceptibility profile of *C. diphtheriae*. The LDCIC-L2 also showed a decreased in both the lethality of *C. elegans* and the survival in the intracytoplasmatic compartment of HEp-2 cells. In conclusion, the role of OxyR in *C. diphtheriae* virulence seems not to be solely related to the regulation of oxidative stress defense genes, but is also associated with other clinically relevant processes that allow this pathogen to persist in the host tissues, despite inflammatory and cellular responses.

Palavras-chaves: *Caenorhabditis elegans*; *Corynebacterium diphtheriae*; OxyR; extracellular matrix; HEp-2 cells.

Agências de fomento: CNPq, CAPES, FAPERJ, SR-2 UERJ. *Bolsista do CNPq – Brasil.