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Resumo	Although originally known as an opportunistic pathogen, *Klebsiella pneumoniae* has been considered a worldwide health threat nowadays due to the emergence of hypervirulent and antibiotic-resistant strains capable of causing severe infections not only on immunocompromised patients but also on healthy individuals. Fimbriae is an essential virulence factor for *K. pneumoniae*, especially in urinary tract infections (UTIs), because it allows the pathogen to adhere and invade urothelial cells and to form biofilms on biotic and abiotic surfaces. The importance of fimbriae for *K. pneumoniae* pathogenicity is highlighted by the large number of fimbrial gene clusters on the bacterium genome, which requires a coordinated and finely adjusted system to control the synthesis of these structures. In this work, we describe KpfR as a new transcriptional repressor of fimbrial expression in *K. pneumoniae* and discuss its role in the bacterium pathogenicity. *K. pneumoniae* with disrupted *kpfR* gene exhibited a hyperfimbriated phenotype with enhanced biofilm formation and greater adhesion to and replication within epithelial host cells. Nonetheless, the mutant strain was attenuated for colonization of the bladder in a murine model of urinary tract infection. These results indicate that KpfR is an important transcriptional repressor that, by negatively controlling the expression of fimbriae, prevents *K. pneumoniae* from having a hyperfimbriated phenotype and from being recognized and eliminated by the host immune system.
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