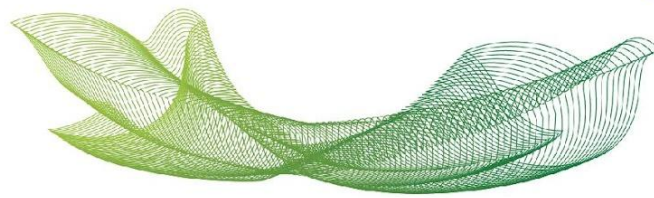




<b>Tipo</b>	Periódico
<b>Título</b>	SdiA, a Quorum-Sensing Regulator, Suppresses Fimbriae Expression, Biofilm Formation, and Quorum-Sensing Signaling Molecules Production in <i>Klebsiella pneumoniae</i>
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<b>Assunto (palavras-chaves)</b>	<i>Klebsiella pneumoniae</i> ; SdiA regulator; Cell Division; Quorum sensing; Type 1 fimbriae; Biofilm
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<b>Resumo</b>	<p><i>Klebsiella pneumoniae</i> is a Gram-negative pathogen that has become a worldwide concern due to the emergence of multidrug-resistant isolates responsible for various invasive infectious diseases. Biofilm formation constitutes a major virulence factor for <i>K. pneumoniae</i> and relies on the expression of fimbrial adhesins and aggregation of bacterial cells on biotic or abiotic surfaces in a coordinated manner. During biofilm aggregation, bacterial cells communicate with each other through inter- or intra-species interactions mediated by signalling molecules, called autoinducers, in a mechanism known as quorum sensing (QS). In most Gram-negative bacteria, intra-species communication typically involves the LuxI/LuxR system: LuxI synthase produces <i>N</i>-acyl homoserine lactones (AHLs) as autoinducers and the LuxR transcription factor is their cognate receptor. However, <i>K. pneumoniae</i> does not produce AHL but encodes SdiA, an orphan LuxR-type receptor that responds to exogenous AHL molecules produced by other bacterial species. While SdiA regulates several cellular processes and the expression of virulence factors in many pathogens, the role of this regulator in <i>K. pneumoniae</i> remains unknown. In this study, we describe the characterization of <i>sdiA</i> mutant strain of <i>K. pneumoniae</i>. The <i>sdiA</i> mutant strain has increased biofilm formation, which correlates with the increased expression of type 1 fimbriae, thus revealing a repressive role of SdiA in fimbriae expression and bacterial cell adherence and aggregation. On the other hand, SdiA acts as a transcriptional activator of cell division machinery assembly in the septum, since cells lacking SdiA regulator exhibited a filamentary shape rather than the typical rod shape. We also show that <i>K. pneumoniae</i> cells lacking SdiA regulator present constant production of QS</p>



	autoinducers at maximum levels, suggesting a putative role for SdiA in the regulation of AI-2 production. Taken together, our results demonstrate that SdiA regulates cell division and the expression of virulence factors such as fimbriae expression, biofilm formation, and production of QS autoinducers in <i>K. pneumoniae</i>
<b>Fomento</b>	Fapesp e CAPES