Tipo	Periódico
Título	Pneumococcal surface protein A (PspA) prevents killing of Streptococcus pneumoniae by indolicidin
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Programa/Curso (s)	Programa de Pós-Graduação Stricto Sensu em Ciências da Saúde
DOI	0.1038/s41598-024-73564-9.
Assunto (palavras chaves)	Streptococcus pneumoniae; Cationic peptide; Immune evasion; Indolicidin; Pneumococcal surface protein A.
Idioma	Inglês
Fonte	Título do periódico: Scientific Reports ISSN: 2045-2322 Volume/Número/Paginação/Ano: 14(1):23517, 2024
Data da publicação	10/2024
Formato da produção	digital
Resumo	Pneumococcal surface protein A (PspA) is an important virulence factor in Streptococcus pneumoniae that binds to lactoferrin and protects the bacterium from the bactericidal action of lactoferricins-cationic peptides released upon lactoferrin proteolysis. The present study investigated if PspA can prevent killing by another cationic peptide, indolicidin. PspA-negative pneumococci were more sensitive to indolicidin-induced killing than bacteria expressing PspA, suggesting that PspA prevents the bactericidal action of indolicidin. Similarly, chemical removal of choline-binding proteins increased sensitivity to indolicidin. The absence of capsule and PspA had an additive effect on pneumococcal killing by the AMP. Furthermore, anti-PspA antibodies enhanced the bactericidal effect of indolicidin on pneumococci, while addition of soluble PspA fragments competitively inhibited indolicidin action. Previous in silico analysis suggests a possible interaction between PspA and indolicidin. Thus, we hypothesize that PspA acts by sequestering indolicidin and preventing it from reaching the bacterial membrane. A specific interaction between PspA and indolicidin was demonstrated by mass spectrometry, confirming that PspA can actively bind to the AMP. These results reinforce the vaccine potential of PspA and suggest a possible mechanism of innate immune evasion employed by pneumococci, which involves binding to cationic peptides and hindering their ability to damage the bacterial membranes.
Fomento	FAPESP (processos 2020/11037-6 e 2023/10579-8); Swedish Research Council (VR grants 2021–06050 and 2022 − 01296) e Capes (bolsas 88887.901630/2023-00 e 88887.148363/2017-0).