Methicillin-resistant *Staphylococcus aureus* (MRSA) are major hospital pathogens that can be involved in life-threatening infections. In addition to the *S. aureus* ability to acquire antimicrobial resistances, they present a plethora of virulence factors; some of which enable them to escape the host immune system by subverting phagocytosis, reactive oxygen species and complement activation. MRSA clones are usually defined by SCCmeC typing, MLST and spa polymorphisms. Recently, we have performed a large epidemiological panel study, in Rio de Janeiro State, with 600 MRSA isolates, which reveals clonal complex 5 (CC5) as the predominant MRSA nowadays. Among CC5 isolates, ST105-SCCmeCII-t002 prevailed, accounting for about 50%, followed by ST5-SCCmeCIV-t002 (approx. 20%). The aim of this study was to compare CC5 MRSA clones in a phagocytosis model, bacterial survival after interaction with monocytes, and a virulence model using *Caenorhabditis elegans*. Phagocytosis assays were carried out using fluorescent-marked bacteria and the monocytes lineage THP-1 (MOI 10) and then measured by flow-cytometry. Additionally, after 30 min-interaction with monocytes, the survival of extracellular bacteria was determined by CFU counting. CC5 strains were used to feed adult *C. elegans* and nematode survival curves determined by daily counting for 3 days, using inverted microscopy. The genome sequencing of CC5 isolates was carried out using Illumina MiSeq system. Our data revealed a drop of 91.7% (*p*=0.0005) in the phagocytosis rate for the dominant ST105-SCCmeCII-t002 in relation to ST5-SCCmeCIV-t002. Also, the survival rate of ST105-SCCmeCII-t002, after interaction with monocytes, was 10-fold higher than that of ST5-SCCmeCIV-t002 (*p*=0.0422). Finally, no significant difference was observed in the *C. elegans* survival curves for ST105 and ST5 strains. Taken together, these results suggest that, although both clones showed comparable virulence in the nematoid model, ST105-SCCmeCII-t002 appear to evolved toward increased capability of immune evasion, which may have contributed for their success as a hospital pathogen. In fact, a non-conservative mutation, occurred only in ST105-SCCmeCII-t002 genomes, in the *aur* gene encoding aureolysin, a protease that cleaves host factors of the immune system, might be implicated in these evasion mechanisms.

**KEYWORDS:** Immune evasion; Phagocytosis; *Staphylococcus aureus*; Virulence

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