



Staphylococcus aureus alters cell cycle in human and bovine epithelial cells

Ludmila Alekseeva, Lucie Rault, Sintia Almeida da Silva, Fabien Cousin, Patrick Legembre, Valérie Edmond, Vasco Azevedo, Anderson Miyoshi, Sergine Even, Frederic Taieb, et al.

► To cite this version:

Ludmila Alekseeva, Lucie Rault, Sintia Almeida da Silva, Fabien Cousin, Patrick Legembre, et al.. Staphylococcus aureus alters cell cycle in human and bovine epithelial cells. 16. International Symposium on Staphylococci and Staphylococcal Infections, Aug 2014, Chicago, United States. 2014. hal-01209709

HAL Id: hal-01209709

<https://hal.science/hal-01209709>

Submitted on 3 Jun 2020

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Staphylococcus aureus can cause a wide range of infections in humans and warm-blooded animals. The epithelial surface is the principal site of staphylococcal infection. The goal of this work was to investigate the cytopathic effect of *S. aureus* and its ability to affect host cell cycle. Cultures of human cervix cancer HeLa and mammary gland bovine MAC-T epithelial cells were used to study *in vitro* the effects of *S. aureus* strains from human or ruminant origins. Using microscopic examination, we found that *S. aureus* induced an enlargement of the host cells and we observed that *S. aureus* slowed down cell proliferation. Similar effect was observed on both human and bovine cells using the human or the ruminant strains. Microscopic analysis of DAPI-stained cells revealed huge decrease of mitotic indexes in infected HeLa and Mac-T cells, synchronized by Double Thymidine Block. Flow cytometry analysis showed that *S. aureus* induces G2/M phase delay in synchronous HeLa cells. Western blot analysis revealed that the G2/M phase delay was associated with an accumulation of inactive phosphorylated cyclin-dependent kinase Cdk1/cdc2, a key inducer of mitosis entry, and dephosphorylation of core histone H3, an important component of the epigenetic landscape. Altogether, these results suggest that the delay in G2/M phase transition of the infected epithelial cells may be one of the mechanisms employed by *S. aureus* to survive and to propagate inside the host. Further investigations are ongoing to fully understand the mechanisms associated to this type of *S. aureus*-host cell interactions.