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## Interactions of toxins from *S.aureus* toxin-antitoxin systems with membranes

Toxin-antitoxin systems are genetic modules widely spread in bacterial genomes. They contain a toxin, which stops the bacteria's growth and an antitoxin, which inhibits toxin's expression. Under normal conditions, the antitoxin is produced concomitantly with the toxin, so the toxin is not translated. Under a stress, the antitoxin is not produced anymore, so the toxin can be translated, act on the bacteria and stop its growth when overexpressed. The role of these systems is still elusive. So far, they have been shown to be involved in the maintenance of mobile genetic elements, growth adaptation to environmental stresses or antibiotic persistence.

*Staphylococcus aureus* is a major human pathogen and one of the first cause of hospital-acquired infections. Several type I toxin-antitoxin systems have been discovered in *S. aureus* genome. These systems are thought to be involved in *S. aureus* pathogenicity.

Notably, three of these toxins have been studied [1-3]: they are small cationic peptides structured in alpha helix. They show hemolytic activity against erythrocytes, while they do not exhibit effective antibacterial activity. How to explain this specificity of action?

We aim here to investigate their structures and the interactions between these toxins and membranes when overexpressed inside the bacteria. We also study interactions with model membranes in order to gain insight about the selectivity of these toxins for mammalian versus bacterial cell membranes and so about their mechanisms of action and roles in *S.aureus* virulence.

[1] Sayed, N., Nonin-Lecomte, S., Réty, S. & Felden, B. Journal of Biological Chemistry, 287 (2012) 43454–43463.

[2] Pinel-Marie, M.-L., Brielle, R. & Felden, B. Cell Reports, 7 (2014) 424–435.

[3] Germain-Amiot, N. et al. Nucleic Acids Research, 47 (2019) 1759–1773.

### Session

Molecular interactions at the membrane surface

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