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The transition between sessile and motile bacterial lifestyles

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Document Version

Publisher's PDF, also known as Version of record

Publication date:

2012

[Link to publication in University of Groningen/UMCG research database](#)

Citation for published version (APA):

Tsompanidou, E. (2012). The transition between sessile and motile bacterial lifestyles. Groningen: s.n.

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The transition between sessile and motile bacterial lifestyles

1. The *agr* system plays a decisive role in the choice of *S. aureus* cells between a sessile and a motile lifestyle (this thesis).
2. Mutation of the *srtA* gene locus leads to a hyper-spreading phenotype both in *S. aureus* and *S. epidermidis* (this thesis).
3. PSM peptides not only allow *S. aureus* cells to spread to new surfaces that might be richer in nutrients, but they also allow *S. aureus* to compete with other bacterial species that colonize the same surfaces (this thesis).
4. Cells that form strong biofilms are poor spreaders and *vice versa* (this thesis).
5. PSM-mediated spreading plays a major role in the movement of *S. aureus* over biotic surfaces (this thesis).
6. Drugs that target the PSM peptides in combination with cell-surface exposed proteins, such as FnbpA, FnbpB, ClfA and ClfB might decrease the survival of *S. aureus* in the host and limit their transmission both in the community and within a hospital setting (this thesis).
7. The results you obtain with *S. aureus* are seemingly dependent on the part of the world in which you conduct your experiments.
8. Cell wall-associated factors that promote a sessile lifestyle of *S. aureus* antagonize this bacterium's colony spreading motility.
9. Working with *S. aureus* makes you feel like a speeder. You have to be the first one to reach the editors office before 'the others' do.
10. Working with PSMs and trying to get my work published was like a battle between a woman and men.