BIOFILMS

Building biofilms with DNA

DNA released from dead bacteria forms an important part of bacterial biofilms. In a paper that is available online in *Molecular Microbiology*, Thomas and colleagues describe a fratricidal mechanism in *Enterococcus faecalis* that leads to the lysis of a fraction of the bacterial population to provide a source of DNA for biofilm formation.

Cell death during *Staphylococcus aureus* biofilm formation has been postulated to be an altruistic process, but the mechanism used by *E. faecalis* is unknown. In previous work, Thomas and colleagues had determined that the protease gelatinase (GelE) was important in bacterial cell death during *E. faecalis* biofilm formation, and they were interested in determining the nature of the cell death involved. The production of GelE is upregulated in response to the quorum sensing factor gelatinase biosynthesis-activating pheromone (GBAP). However, the authors found that in stationary phase, about 15% of the bacteria did not respond to GBAP and therefore did not make GelE or the co-transcribed protease SprE.

In co-culture experiments, the authors showed that only bacteria that produce GelE can lyse other bacteria. Interestingly, bacteria that do not produce SprE could lyse prey bacteria more efficiently than wild-type bacteria, pointing to a modulating function for SprE.

Previous observations had implicated autolysins, proteins that are involved in the breakdown of peptidoglycan, as targets of GelE. *In vitro* experiments comparing wild-type bacteria and GelE mutants revealed that the autolysin AtlA is processed by GelE. As further evidence of a role for AtlA in the formation of biofilms, the authors found that biofilm formation and the release of extracellular DNA were affected similarly by a disruption of *atlA* and a deletion of *gelE*. However, *altA* mutants were still lysed by wild-type bacteria. The authors speculated that the release of AtlA by the wild-type bacteria was responsible for this lysis, and went on to show that bacteria deficient in AtlA were unable to lyse *atlA* mutants. Furthermore, GelE can process AtlA *in vitro*. SprE can also break down and activate AtlA, but the autolysin fragments generated by GelE and SprE are different. The GelE-treated AtlA had a decreased cell-binding affinity compared with SprE-treated AtlA, but when AtlA was first treated with SprE further treatment with GelE did not influence the binding characteristics of AtlA.

Based on these findings, the authors propose a model in which the quorum signal GBAP activates the production of both GelE and SprE. SprE acts as an immunity factor and prevents the deleterious effects of GelE. The bacteria that do not respond to GBAP form a target-susceptible subfraction that can be lysed by the activity of the GelE-cleaved AtlA, thereby releasing extracellular DNA, a valuable component that is needed for biofilm formation.

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> “*In vitro* experiments comparing wild-type bacteria and GelE mutants revealed that the autolysin AtlA is processed by GelE.”

ORIGINAL RESEARCH PAPER Thomas, V. C. *et al.* A fratricidal mechanism is responsible for eDNA release and contributes to biofilm development of *Enterococcus faecalis*. *Mol. Microbiol.* 21 April 2009 (doi: 10:1111/j.1365-2958.2009.06703.x)

FURTHER READING Claverys, J.-P. & Håverstein, L. S. Cannibalism and fratricide: mechanisms and raisons d’être. *Nature Rev. Microbiol.* **5**, 221–229 (2007)