Theses

Ulf W. Liebal, Regulation of the general stress response of *Bacillus subtilis*

Major new insights

1. Different stressors activate the stressosome signalling protein complex identically. Thus, according to the stressosome, a stress of 3% ethanol is as stressfull as 488 mM of NaCl.

2. Structures of truncated icosahedra (vertices as proteins) allow a characteristic collapse of three dimers. This collapse hypothesis of stressosome activation explains the information transfer from the stress sensor to the output protein.

3. Discrimination of differential equation models of the general stress response suggests a protease that degrades the reporter protein. This protease model reproduces the stimulus-independent transient activation of the stress response.

Independent confirmation of knowledge

1. Geometric models show that truncated icosahedra are optimally constructed from dimers. In these models, sixty tetrahedra coincide in their structural arrangement with the arrangements of proteins in the stressosome.

2. A cellular automaton confirms the stimulating effect of phosphorylated RsbR on the kinase activity of RsbT by reproducing different experimental data sets.

3. The fit of the cellular automaton of the stressosome to experimental data is optimal, if the phosphatase RsbX is specific for RsbS-P during low and medium stress, and dephosphorylation of RsbR-P is magnitudes lower.

4. Stress reception of the stressosome leads to structural changes and the activation of a signalling molecule. The slow deactivation of the stressosome repeatedly activates the signalling molecule and decouples the reponse from the signal duration.