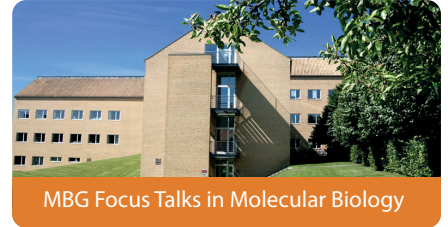


# MBG FOCUS TALK

hosted by Ditlev Brodersen



**Monday 20 June 2022, 11.00 am**

1870-816 Faculty club

**Asc. Prof. Clare Kirkpatrick**

Department of Biochemistry and Molecular Biology, SDU

## **Another three-component variation on the toxin-antitoxin theme: the transcription factor HigC and its role in the DNA damage response**

In the model bacterium *Caulobacter crescentus*, the DNA damage response repressor LexA controls transcription of a number of genes, including several toxin-antitoxin systems. For most of these it is unclear whether they play any functional role in the DNA damage response. Deletion of the toxin HigB in a *lexA* mutant background improves its growth and its resistance to DNA damaging antibiotics, suggesting that the HigBA toxin-antitoxin system at least is involved in the DNA damage response. However, this improvement was conditional on whether the toxin alone or the whole TA system was deleted, suggesting that other regulatory factors are involved. We identified a putative transcription factor immediately downstream of *higBA*, reminiscent of other three-component TA systems where the transcription factor regulates expression of the TA system's promoter. This transcription factor (HigC) did indeed regulate the *higBA* promoter, but unusually was able to affect DNA-damaging antibiotic resistance in a strain where the HigB toxin was absent, showing that it must exert some effect independently of the HigBA TA system. HigC localizes to the cell membrane and forward genetic analysis suggested that HigC may affect cell envelope stability, potentially through the cell wall amidase AmiC. This unusual factor therefore connects the DNA damage and cell envelope stress responses in *Caulobacter*.

**All welcome**

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