**„ClpAP protease as a controlling factor of the *parDE* toxin-antitoxin system activation of RK2 plasmid"**
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The activity of type II toxin-antitoxin systems (TA), which are responsible for many important features of bacterial cells, is based on the differences between toxin and antitoxin stabilities. In all type II TA systems antitoxins lability results from bacterial protease activity. Here, I investigated how particular *Escherichia coli* cytosolic proteases, namely, Lon, ClpAP, ClpXP, and ClpYQ, affect the stability of both the toxin and antitoxin components of the *parDE* system from the broad host range plasmid RK2. The results of my *in vivo* and *in vitro* experiments show that the ParD antitoxin is degraded by the ClpAP protease, and dsDNA stimulates this process. The ParE toxin is not degraded by any of these proteases and can therefore cause growth inhibition of plasmid-free cells after an unequal plasmid distribution during cell division, this process is called post-segregation killing (PSK). I also demonstrate that the ParE toxin interaction with ParD prevents antitoxin proteolysis by ClpAP; however, this interaction does not prevent the ClpAP interaction with ParD. I show that ClpAP protease homologs from *Caulobacter crescentus* and *Pseudomonas putida* bacteria affect plasmid stability. During *in vitro* proteolysis assays I also showed that ClpAP proteases from *C. crescentus* and *P. putida* species efficiently degrades ParD anti-toxin. All the experiments I made, indicating that ClpAP is a universal activator of the *parDE* system and that ParD is a universal substrate for ClpAP. Furthermore, the *parDE* system is a universal TA system responsible for maintaining the RK2 plasmid in host cells by activating PSK.