LOCALIZATION AND DYNAMICS OF PROTEINS THAT CONTROL DIVISION-SITE PLACEMENT IN *Bacillus subtilis* DURING SPORULATION.

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During vegetative growth, *B. subtilis* divides symmetrically generating identical daughter cells. This is in part due to the action of the MinCD complex, a division inhibitor which localizes to the cell poles and prevents the polymerization of the main division protein - FtsZ - from happening there. In contrast, during sporulation *B. subtilis* divides asymmetrically, with the septum forming next to one of the cell poles. Our lab has been employing localization of GFP-tagged proteins to try to answer the question of how FtsZ becomes capable of assembling at the cell poles during sporulation. We have previously demonstrated that MinC localization changed during sporulation. The protein gradually left the pole and associated with the new asymmetric septum. Here we show that MinD, the partner of MinC, and DivIVA, the protein responsible for targeting MinCD to the cell poles, also leave the pole for the new asymmetric septum during sporulation. These results suggest that the asymmetric division of sporulation is due to the departure of the division inhibitor from the polar site. To test this hypothesis directly we have co-localized MinC and FtsZ in sporulating cells. Surprisingly, we found that asymmetric FtsZ structures can be detected in cells that still have MinCD at the poles, Thus, FtsZ assembly at the poles is not simply due to the relocalization of the MinCD inhibitor.

Supported by FAPESP