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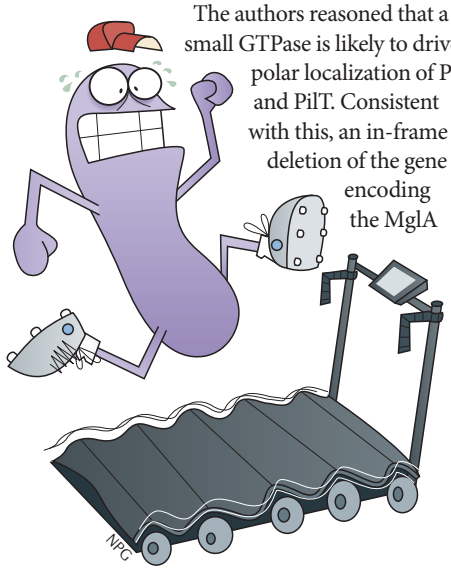
BACTERIAL PHYSIOLOGY

SofG and BacP keep bacteria moving

For many bacteria, the principles of directed motility resemble those of eukaryotes: the cell must become polarized and form a leading and a lagging edge. In the rod-shaped species *Myxococcus xanthus*, type IV pilus (T4P)-dependent motility involves the polar localization of the T4P proteins PilB and PilT to the leading and lagging edges (or poles), respectively, and this differential localization depends on the small GTPase MglA. Now, Søgaard-Andersen and colleagues solve another piece of the polarity puzzle, revealing that, prior to MglA activity, the small GTPase SofG and the cytoskeletal protein BacP ensure that PilB and PilT are first recruited to the same pole.



The authors reasoned that a small GTPase is likely to drive polar localization of PilB and PilT. Consistent with this, an in-frame deletion of the gene encoding the MglA paralogue SofG (Δ*sofG*) caused defects in T4P-dependent motility. The authors also observed that GTPase activity was substantially reduced following mutation of the SofG GTPase domain (specifically, an Arg111Ala substitution) and that this GTPase mutant could not compensate for the motility defect associated with the loss of SofG; this highlights a key role for SofG GTPase activity in this process.

Despite the defects in motility, Δ*sofG* cells showed normal accumulation of T4P proteins and assembled T4Ps; however, PilB and PilT displayed defective localization at the two cell poles. Similarly to PilB and PilT, SofG was found to localize within ~1.4 μm of the nearest pole, suggesting that SofG functions upstream of the two Pil proteins to promote their polar localization. The GTPase function of SofG was important for this process, as PilB and PilT also showed defective polar localization in the Arg111Ala SofG mutant. Furthermore, PilT, which is integral for T4P retraction, did not dynamically relocalize during migration reversal in Δ*sofG* cells.

Interestingly, time-lapse analysis showed that SofG shuttled between the subpolar region and the nearest pole, so the authors postulated that it might interact with another protein in the pole to ensure polar PilB and PilT localization. Pull-down experiments identified the cytoskeletal protein BacP — a bactofilin — as a candidate, and electron microscopy confirmed that the two proteins directly interact. Consistent with this, Δ*bacP* cells had defective T4P-dependent motility, similarly to Δ*sofG* cells. Moreover, although wild-type cells contained large patches of BacP filaments at the subpolar regions, these patches were absent in Δ*bacP* cells. Importantly, SofG showed diffuse localization in Δ*bacP* cells, and the localization of PilB and PilT resembled that observed in Δ*sofG* bacteria. So, the authors conclude that BacP acts upstream of SofG to promote correct PilB and PilB localization.

On the basis of their findings, the authors propose that BacP first assembles into filament patches at the subpolar regions to recruit SofG. In turn, SofG shuttles along the filaments from the subpolar region to the pole in a GTPase-dependent manner, recruiting PilB and PilT to the pole. MglA subsequently sorts the two proteins to opposite poles to establish their correct polarity.

Rachel David

ORIGINAL RESEARCH PAPER Bulyha, I. *et al.* Two small GTPases act in concert with the bactofilin cytoskeleton to regulate dynamic bacterial cell polarity. *Dev. Cell* 11 Apr 2013 (doi:10.1016/j.devcel.2013.02.017)