E. coli growth and this inhibition was reversed by the addition of free iron. In agreement with previous results, the presence of TL had no effect on *P. aeruginosa* growth. For fungi, the authors examined the effect of TL on the growth of a mutant strain of *A. nidulans* that is deficient in siderophore biosynthesis and showed that the presence of TL significantly inhibited growth.

One member of the lipocalin family, neutrophil gelatinase-associated lipocalin (NGAL), was already known to scavenge siderophores. This paper shows that TL also mops up siderophores, thereby inhibiting bacterial growth; unlike NGAL, however, which only binds to bacterial catecholate siderophores, TL binds to both catecholate and hydroxamate siderophores, and can inhibit both bacterial and fungal growth.

Sheilagh Clarkson

References and links

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CELLULAR MICROBIOLOGY

Ping-pong cell-fate control

"Dr Jacobs-Wagner has observed a critical step in how cells control their development. Although her discovery was made in a…bacterium, it's likely that similar processes also control development in higher organisms, including humans. These findings will likely lead to a better understanding of both normal and abnormal human development" said Marion Zatz, Ph.D., chief of the developmental and cellular processes branch at the National Institute of General Medical Sciences, which partially funded the work.

New research published in *Cell* has linked initiation of a developmental programme in the model bacterium *Caulobacter crescentus* to the completion of cytokinesis.

Generating different cell types isn't just important for some bacteria — in development, eukaryotes use asymmetric cell division to generate a diverse array of cell types. *Caulobacter* divides asymmetrically at each cell division to produce a sessile stalked cell and a smaller motile swamer cell. The swarmer cell undergoes a swarmer-progeny-specific (SwaPS) developmental programme to differentiate into a stalked cell. Inhibition of cytokinesis blocks the initiation of SwaPS development.

Two histidine kinases, DivJ and PleC, which function through the shared response regulator DivK, had already been implicated in coupling SwaPS development with cell division. DivJ phosphorylates DivK, while PleC promotes DivK dephosphorylation, either directly or indirectly. All three regulators are localized in the *Caulobacter* cell — PleC at the flagellar pole, DivJ at the stalked pole and DivK at both poles, with the localization of DivK modulated by DivJ and PleC. After cytokinesis, DivK is no longer present at the flagellar pole. The latest report from the Jacobs-Wagner laboratory examines how these proteins are localized to specific locations and whether localization affects SwaPS development.

Mutating PleC disrupts SwaPS development, possibly by preventing DivK release from the flagellar cell pole. A mutant form of DivK ($DivK_{D90G}$) that suppressed a *pleC* knockout mutant was not localized at the flagellar pole of the nascent swarmer cell. By depleting FtsZ, and therefore disrupting cell division, Matroule *et al*. showed that DivK–GFP was only released from the flagellar pole when cell division proceeded. If DivK is an intrinsic part of the control mechanism it should be possible to undergo SwaPS without cytokinesis if DivK is not present at the flagellar pole — this was the case for the $DivK_{D90G}$ mutant.

If DivK release is the switch, how does the cell sense that cytokinesis is complete and release DivK from the flagellar pole? Using a mutant form of PleC, which had phosphatase activity but lacked kinase activity, Matroule *et al*. showed that PleC is primarily a DivK~P phosphatase *in vivo*. Fluorescence resonance energy transfer (FRET) analysis showed that DivJ and PleC physically interact with DivK at the poles — DivJ/DivK interact at the nascent stalked cell pole, and PleC/DivK interact at the nascent flagellar pole. Finally, fluorescence photobleaching revealed that DivK shuttles from one pole to the other by diffusion. *Caulobacter* monitors communication between the cell poles through 'ping-pong' shuttling of DivK and DivK~P.

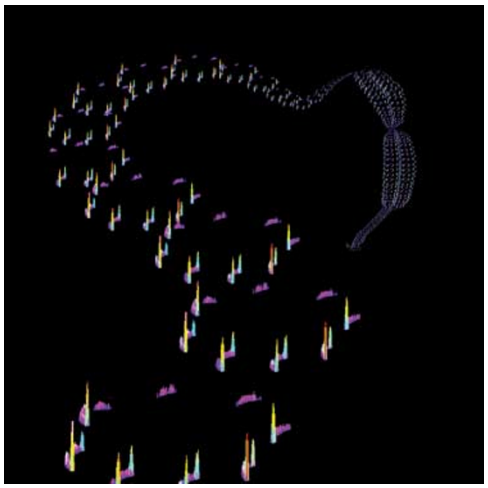

Image courtesy of Christine Jacobs-Wagner.

Keeping DivK~P at the flagellar pole inhibits SwaPS development. Cytokinesis separates the daughter cells, preventing DivK~P (produced by DivJ kinase activity at the stalked pole) from shuttling to the flagellar pole. DivK~P at the flagellar pole is completely dephosphorylated by PleC and released into the cytoplasm, and SwaPS development proceeds, but how DivK itself controls cell fate still needs to be resolved.

In *Caulobacter*, cytokinesis triggers cell development — this might be the first true bacterial checkpoint to be identified. It might also be a paradigm for developmental controls that function at cell poles from bacteria to man.

Susan Jones

References and links

ORIGINAL RESEARCH PAPER Matroule, J.-Y. *et al*. Cytokinesis monitoring during development: rapid pole-to-pole shuttling of a signaling protein by localized kinase and phosphatase in *Caulobacter*. *Cell* **118**, 579–590 (2004)

WEB SITE

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