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Disruption of Cell Size Homeostasis in (p)ppGpp-deficient *Escherichia coli*

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Under constant environmental conditions, the processes of cell growth and cell cycle progression are coordinated to ensure that cells of the Gram-negative bacterium *Escherichia coli* divide at midcell after adding a constant volume between birth and division. As long as these processes are properly coordinated, cells maintain size homeostasis, exhibiting very little variation in the size of dividing and newborn daughter cells.

Precisely how cells maintain homeostasis is not fully understood, but the signaling nucleotides guanosine pentaphosphate or tetraphosphate ((p)ppGpp), appear to play a critical role. (p)ppGpp is a global regulator of biosynthetic pathways that is synthesized in response to nutritional stress. The intracellular concentration of (p)ppGpp rises following carbon, nitrogen, phosphorus, and iron deprivation, or reductions in fatty acid synthesis, which triggers a rapid and global downregulation of ribosomal and transfer RNA synthesis to slow down biosynthetic pathways. This is accompanied by (p)ppGpp-dependent modulation of DNA replication initiation, elongation, and cell division.

While (p)ppGpp is studied as a mediator of stress responses, cells unable to synthesize (p)ppGpp ((p)ppGpp^0) exhibit a number of phenotypes under relatively stress-free conditions, including an average increase in cell size and disruption of size homeostasis. To further our understanding of how (p)ppGpp acts to maintain size homeostasis, we will analyze various cellular parameters of (p)ppGpp^0 cells, using live-cell imaging and cell segmentation software to quantify single-cell growth rates, cell size at birth and division, and to characterize the positioning and dynamics of the cell division machinery.