Biophysics and Systems Biology Seminar Series

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"The mechanistic origin of cell-size homeostasis in bacteria"

Abstract: Evolutionarily divergent bacteria share a common phenomenological strategy for cell-size homeostasis under steady-state conditions. In the presence of inherent physiological stochasticity, cells following this "adder" principle gradually return to their steady-state size by adding a constant volume between birth and division regardless of size at birth. The identification of the "adder" represented a major shift in our understanding of cell-size homeostasis, but its mechanism has been unknown despite intense efforts. I will explain that adder is a direct consequence of two underlying processes: (1) constant production of division proteins/precursors that track the rest of the proteome, and (2) accumulation of these biomolecules to a fixed threshold number. This mechanism is naturally robust to static growth inhibition, due to balanced biosynthesis of division factors that mirrors elongation of the cell. Leveraging this finding, we are able to "break" adder in one-billion years divergent Escherichia coli and Bacillus subtilis in a predicted manner by generating dynamic oscillations in the concentration of the division proteins. In contrast, periodic induction of replication initiator proteins caused oscillations in initiation mass, but did not alter division size. Finally, I will show how to "restore" the adder phenotype in slow growth conditions, in which E. coli is known to deviate from adder. Together the mechanisms of bacterial cell-size homeostasis we identified are general and robust, allowing us not only to break but also to restore the adder phenotype in a predictive manner under all growth conditions.

Thursday, October 25th, 2018 at 10:00AM
Natural Sciences 2, Room 1201

Hosts: Matt Bovyn and Professor Albert Siryaporn

If you're interested in meeting with Dr. Jun, please contact Albert Siryaporn (asirya@uci.edu)