# COMPUTATIONAL BIOLOGY WEBINAR @ IMSc

# WHY COUSINS ARE MORE SIMILAR THAN MOTHER-DAUGHTERS: IMPLICATIONS FOR CELL CYCLE REGULATION


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Recent developments in microscopy techniques have allowed probing of cellular dynamics at an unprecedented resolution and throughput. For example, these advances are now allowing us to study the phenomenon of cellular proliferation at the single cell level, rather than the population dynamics of millions of cells. However, interpreting the inevitably noisy datasets associated with such single cell measurements is a fundamental challenge and provides an exciting opportunity for developing physical models in combination with statistical inference. Here I will present work where we combined time-lapse microscopy and Bayesian inference to uncover surprising correlations in the division and death times of colon cancer cells closely related by lineage, both before and during chemotherapy treatment. These correlations could not be explained using simple protein production-degradation models that are currently believed to underlie cell fate control. We then developed a stochastic model explaining how the observed correlations can arise from oscillatory mechanisms underlying cell cycle control. Our model was able to recapitulate the data only with specific oscillation periods that fit measured circadian rhythms, suggesting that cell to cell heterogeneity in cell cycle progression rates may arise from circadian control over the cell cycle. Finally, I will discuss some new experiments and theory we are developing to further investigate the role of the circadian clock in cellular proliferation, both in cancer as well as in stem cells.


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