Northern Regional Meeting of the London Mathematical Society
&
Workshop on Mathematics of Human Biology

Abstracts of Communications

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Complex dynamics of cellular transcriptional response: how do cells get on the fast lane?

Eytan Domany[a]

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The talk will start with a very basic introduction aimed at the non-expert.

In response to external stimuli, cells adjust their behavior to a changing environment - for example, they start to divide or migrate. In order to perform these actions, the protein content of the cell must change. To accomplish this, a cell must modify the levels at which the genes that code for these proteins are transcribed. These transcriptional responses to extracellular stimuli are regulated by tuning the rates of transcript production and degradation. I present here the results of a study aimed at deducing the dynamics of these two processes from measurements of the transcriptome, and to elucidate the operational strategy behind this dynamics.

By combining a simple theoretical model of transcription with simultaneous measurements of time-dependent precursor mRNA and mature mRNA abundances, we were able to infer unexpected complex stimulation-induced time-dependent transcript production and degradation. In particular, we found that production of many transcripts was characterized by a large dynamic range, which allowed these genes to exhibit an unexpectedly strong and brief pulse of production, thereby accelerating their induction. Surprisingly, we found that the widely used assumption of close correspondence between mRNA abundance and production pro les is incorrect: timing of mRNA maxima does not allow inference of the production pulse. Finally, we discovered that mRNA degradation is regulated in a precisely timed and transcript speci c manner.

References

[1] Coupled pre-mRNA and mRNA dynamics unveil operational strategies underlying transcriptional responses to stimuli. Amit Zeisel, Wolfgang J Kostler, Natali Molotski et al (2011) Molecular Systems Biology 7: 529

A mathematical modeling study of neutrophil dynamics in response to chemotherapy and G-CSF

Michael Mackey[a]

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We have used a mathematical model of the combined dynamics of the hematopoietic stem cells and the di erentiated neutrophil progeny to examine the e ects of periodic

chemotherapy in generating neutropenia, and the corresponding response of this system to granulocyte colony stimulating factor given to counteract the neutropenia. We find that there is a significant period of chemotherapy delivery that induces resonance in the system (at a period twice the average neutrophil lifespan from commitment to death) and a corresponding neutropenia, suggesting that myelosuppressive protocols should avoid this period to minimize hematopoietic damage. The response to G-CSF is highly variable. All of these results, seemingly mysterious initially, are easily understood through an analysis of the dynamical equations and have significant potential impact in clinical practice. I will discuss these results as well as extensions of this research currently underway.

Empirical approaches to the application of mathematical techniques in health technologies

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Mathematical modeling of ageing is built in this paper around research and development activities in cooperation with pharmaceutical companies and hospitals. The interaction of *dirty data* with appropriate mathematical techniques is exemplified mainly with applications to health technologies in endocrinology and oncology. The emphasis is more on old techniques in new situations than on new techniques, though there are references to some novel approaches to modeling.

Workshop: Abstracts of Communications

A ne Re ection Groups for Tiling applications: Knot theory and Modeling of DNA Site-Speci c Recombination

Modeling the kinetics of physiological variables as a dynamical system

Maria Zakynthinaky[a]

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This talk will present a model (Stirling et al., Bul. Math. Biol. 67 (5), 2005) of the underlying dynamics of the kinetics of a physiological variable s (where let s be the heart rate or the rate of change of Oxygen uptake) in response to movement. Assuming that s is a function of time t and intensity v, i.e. s=s(v,t) the model is given in the form of a set of nonlinear coupled vector elds for the second derivative of s with respect to time and the rst derivative of v with respect to time. The model not only provides a perfect r to raw physiological time series data (examples will be presented) but also has the power to estimate the time dependency of the physiological demand and also give predictions regarding the behavior of the physiological system under intensities for which no data exist (such as for example very high intensities). A physiological justi cation of the model will be presented and the results of a linear stability analysis of the model will also be discussed.

References

[1] Stirling et al., Bul. Math. Biol. 67 (5), 2005.

Title: Time-frequency analysis in biomedical data analysis

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The aim of this talk is to present an overview of time-frequency analysis, i.e., techniques to unravel frequency and time information within a non-stationary signal whose frequency content varies over time. For such a signal, the traditional Fourier analysis of frequency loses its e ectiveness and hence one needs to seek a way to capture the time-varying nature of the frequencies occurred in a signal. In this talk, we introduces the audience the highlights to the related theory and demonstrate its usefulness in biomedical applications.

For your Notes

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