AKB 16 Biological Networks

Time: Wednesday 14:00–15:15

Invited Talk

AKB 16.1 Wed 14:00 ZEU 255 Biological Networks: Design Principles of Robust Information Processing •MARKUS KOLLMANN — University of Freiburg, Institute of Physics, Hermann-Herder-Str. 3, 79104 Freiburg

Cellular biochemical networks have to function in a noisy environment using imperfect components. In particular, networks involved in gene regulation or signal transduction allow only for small output tolerances and the underlying network structures can be expected to have undergone evolution for inherent robustness against perturbations. We combined theoretical and experimental analysis to investigate an optimal design for the signalling network of bacterial chemotaxis, one of the most thoroughly studied signalling networks in biology. We experimentally determined the extent of intercellular variations in expression levels of chemotaxis proteins and use computer simulations to quantify the robustness of several hypothetical chemotaxis pathway topologies to such gene expression noise. We demonstrate that the experimentally established topology of the chemotaxis network in Escherichia coli is one of the smallest sufficiently robust structures, allowing accurate chemotactic response for almost all individuals within a population. Our results suggest that this pathway has evolved to show an optimal chemotactic performance while minimising the cost of resources associated with high levels of protein expression. Moreover, the underlying topological design principles compensating for intercellular variations seem to be highly conserved among bacterial chemosensory systems.

\Zitat{1}{M. Kollmann, L. Lovdok, K. Bartholome, J. Timmer, and V. Sourjik, Nature, in press }

AKB 16.2 Wed 14:30 ZEU 255

Locating overlapping dense subgraphs in gene association networks and identifying novel functional units among these groups •ILLES J. FARKAS, GERGELY PALLA, IMRE DERENYI, and TAMAS VICSEK — Biol. Phys. Res. Group of HAS and Dept. of Biol. Phys., Eotvos Univ., H-1117 Budapest, Pazmany P. stny. 1A, Hungary

The identification of the groups of proteins performing the diverse tasks in a cell is crucial to our understanding of cellular networks. In the yeast, S. cerevisiae, known physically interacting groups of proteins (complexes) strongly overlap. The total number of proteins in them by far underestimates their total size (from Refs. [1,2] the ratio is 2750/8932 and 1355/2676), thus, all functional groups of proteins, both physically interacting and other, are likely to share many of their members with other groups. However, most current community search methods exclude overlaps. With the aim to discover both novel functions of individual proteins and novel functional units in gene association networks we combine (i) a search for overlapping dense subgraphs based on the Clique Percolation Method (CPM) [3,4], which explicitly allows overlaps among the groups, and (ii) the verification and characterization of the identified groups of nodes (genes) by annotation tools listing known functions [5].

Guldener, U., et.al. Nucl. Acids Res. 33, D364-368 (2005).

[2] Gavin, A. C., et.al. Nature 415, 141-147 (2002).

[3] Derenyi, I., et.al. Phys. Rev. Lett. 94, 160202 (2005).

[4] Palla, G., et.al. Nature 435, 814-818 (2005),

http://angel.elte.hu/clustering

[5] The Gene Ontology Consortium. Nature Genetics 25, 25-29 (2000).

AKB 16.3 Wed 14:45 ZEU 255

Architecture of Randomly Evolving Idiotypic Networks •Holger Schmidtchen and Ulrich Behn – Institut für Theoretische Physik, Universität Leipzig, POB 100 920, 04009 Leipzig

B-Lymphocytes express on their surface receptors (antibodies) of a given specifity (idiotype). Crosslinking these receptors by complementary structures, antigens or antibodies, stimulates the lymphocyte. Thus a large functional network of interacting lymphocytes, the idiotypic network, emerges. Idiotypic networks conceived by Niels Jerne 30 years ago, experience a renewed interest, e.g. in the context of autoimmune diseases. In a previously proposed minimalistic model [1] idiotypes are represented by bitstrings. The population dynamics of the idiotype clones is reduced to a zero-one scheme. An idiotype survives only if it meets enough but not too much complementary structures. We investigate the random evolution of the network towards a highly organized functional architecture which is driven by the influx of new idiotypes, randomly generated in the bone marrow. The vertices can be classified into different groups,

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which are clearly distinguished, e.g., by the mean life time of the occupied vertices. They include densely connected core groups and peripheral groups of isolated vertices, resembling central and peripheral part of the biological network. We found the building principles of the observed patterns and propose a description of their architecture, which are easily transferable to other patterns and applicable to different system sizes. [1] M. Brede, U. Behn, Patterns in randomly evolving networks: Idiotypic networks, Phys. Rev. E 67, 031920 (2003)

AKB 16.4 Wed 15:00 ZEU 255

Robustness and evolvability of genetic networks - • STEFAN BRAUNEWELL and STEFAN BORNHOLDT — Institute for Theoretical Physics, University of Bremen, Otto-Hahn-Allee, 28359 Bremen, Germany

The molecular biological networks that control the processes of a living cell are required to be robust: They simply have to be stable against perturbations to ensure the survival of the organism [1]. On the other hand, organisms are highly evolvable and have proven quite flexible in the course of biological evolution. Therefore, also the genetic control networks should be flexible under evolution [2]. Is robustness evolvable and does network robustness affect evolvability? We here study these questions in the framework of a simple discrete dynamical network model. We develop a new method for modeling noise in genetic networks and use a non-stochastic approach that allows for exact results.

[1] K. Klemm and S. Bornholdt, Topology of biological networks and reliability of information processing, Proc. Natl. Acad. Sci. USA (2005), in press.

[2] S. Bornholdt and K. Sneppen, Neutral mutations and punctuated equilibrium in evolving genetic networks, Phys. Rev. Lett. 81 (1998) 236.