



NETWORKING METABOLITES, DISEASES, AND DRUGS

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Given that most diseases are the result of the breakdown of some cellular processes, a key aim of modern medicine is to establish the relationship between disease phenotypes and the various disruptions in the underlying cellular networks. In the presence of intricate networking of cellular components, the impact of a mutated gene is not limited to its product but also may affect other cellular functions, leading to potential correlations of diseases and drug effects. In this talk, I show that our current understanding of the topology of the human metabolic network can provide insight into potential relationships among often distinct disease phenotypes and drugs. With the known enzyme-disease associations, a human metabolic disease network is constructed, in which nodes are diseases and two diseases are linked if mutated enzymes associated with them catalyze adjacent metabolic reactions. We find that connected disease pairs display higher correlated reaction flux rate, higher coexpression of the corresponding enzyme-encoding genes, and higher comorbidity than those that have no metabolic link between them. Furthermore, a computational analysis of the modeled function of bacterial metabolic networks offers a chance to discover new antibiotic targets and understand the combinatorial drug effects. Thus, the structure and function of metabolic network can have important consequences for disease diagnosis, treatment, and prevention.