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Structure of Human Complication Disease Network and its Dynamics

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DESCRIPTION

At the molecular or phenotypic level, diseases are not unrelated to one another. Comorbidities are identified from mental patient records by the co-occurrence frequency of disease pairings in patients more frequently than expected. The comorbidity network derived from medical records examines the Type 2 diabetes chronic illness progression trend. From the medicare records, the phenotypic disease network with comorbidity patterns is also extracted, and in particular, the dynamic progression patterns are examined. Significant molecular biology applications, such as protein interaction networks, regulatory networks, metabolic networks, RNA networks, genetic networks, and kinase-substrate networks, are inspired by advances in complex networks. Numerous potential uses for these biological networks in treating human disease exist. The motifs, in particular, may be used to describe the local characteristics of large-scale networks. It has been demonstrated that motifs in many biological networks contain information about functionality. In phenotypic disease networks, genetics is further taken into account along the way. The comorbidity correlation, which is taken from the medical histories of 1.5 million people, is used to evaluate the extent of genetic overlap between diseases. Similar to this, the number of common genes from the human illness network positively correlates with the symptom-based similarity between two diseases. The term “diseasome” refers to the projection of such network-based connections between illness genes and disease phenotypic characteristics. To study the dynamics of biological networks, system biology approaches that combine computational models with experimental data are introduced concurrently. The quantitative modelling methods, usually using networks provide detailed descriptions of biochemical processes in differential equations. This modeling method requires a good knowledge of biological mechanisms and kinetic parameters, limiting its applicability to small and well-characterized networks. In contrast, qualitative modeling approaches are primarily based on network structure and do not require information on motion parameters. However, as a quantitative model, it allows the analysis of important functional properties of large-scale networks, such as input-output relationships, feedback loops, and signaling pathways. However, studies on the dynamics of complex disease systems have so far been lacking due to the complex disease course and lack of experimental data. However, an important overlooked medical data set is the interconnected structure of disease complications. We construct a unique human disease/complication network that expresses the causal relationship between upstream diseases and downstream complications. Systematically examine the structure of the network and pay attention to disease modules. Given the complexity of network dynamics, we further explore motifs that can be used to map the dynamics of disease systems. This work is not only useful for understanding the collectively occurring patterns of specific groups of diseases, but also provides a quantitative framework for studying dynamic course behavior in complex disease systems.

CONCLUSION

Tangles are unforeseen sequelae caused by infections, treatments, or methods. Collect networks of human disease complaints from clinical information and examine tissue quality. We can see that the organizational modules are overwhelmed by infection classes. The relationships between modules are thoroughly clarified. Three key issues have been identified by organizations. We further reproduce elements of the topic using Boolean Eigen models. Each theme corresponds to a specific and unique behaviour that may be useful in disease situations, for example, creating fleeting movements or monitoring responses to fluctuating external stimuli.

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CONFLICT OF INTEREST

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