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Developing combination cancer therapy from ontology fingerprint derived gene network

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Combination therapy is a strategy that combines multiple drugs to improve therapeutic efficacy against a single disease. It is an essential component of standard-of-care treatments for many devastating diseases. However, while success stories such as cocktail therapy for HIV dramatically improved outcomes, there is no effective way to systematically identify promising drug combinations. By combining literature mining, ontology and systems biology, we recently developed an Ontology Fingerprint derived gene network. In this work, we employed the ontology fingerprint derived gene network to identify novel combination therapy strategy to treat cancer. By analyzing the sub-network structure of important cancer pathways in this gene network, we identified novel genes predicted to work with these pathways. Based on our work in the model organism *Saccharomyces cerevisiae*, we predicted that these genes could be targeted to improve the efficacy of the drugs targeting the corresponding cancer pathways. The predicted gene-drug combinations are currently under testing by using siRNA-screening methods to evaluate the synergistic effects between siRNA and the drug combinations. Our novel approach provided a systematic way to identify drug combination strategy to treat human diseases.

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Regulation and function of ribosomal S6 kinases in normal and cancer cells

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Ribosomal protein S6 kinase (S6K) is a member of the AGC family of Ser/Thr kinases which also includes PKA, PKB (Akt), PKCs etc. Biochemical and genetic studies in cell-based and animal models have provided evidence that S6K is a principal player in the regulation of cell growth, size and energy metabolism. Two major signal transduction pathways, phosphatidylinositide 3-kinases (PI3K) and mammalian target of rapamycin (mTOR), coordinate the activity of S6Ks in response to extracellular and intracellular stimuli, such as growth factors, mitogens, metabolites and nutrients. In an activate state, S6Ks translocate to discrete cellular compartments/multienzyme complexes, where they interact with and phosphorylate diverse substrates implicated in the regulation of translation, RNA processing, cytoskeletal rearrangement, cell growth and survival. A growing body of evidence links S6K signalling to various human pathologies, including diabetes, ageing and cancer. In mammalian cells, there are two isoforms of S6K, termed S6K1 and S6K2. The activity and subcellular localization of S6Ks are regulated by multiple S/T phosphorylations in response to diverse extracellular stimuli. Furthermore, acetylation and ubiquitination have been implicated in regulating the function of S6Ks. We have recently uncovered a novel mode of S6K activation, mediated by specific interaction with DNA. Data on molecular mechanisms underlying this regulatory event and its involvement in coordinating transcription, proliferation and cell survival, and in mediating chemoresistance to anti-cancer drugs will be presented.

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