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Myc-p53 interactions illustrate a paradigm for coupled oncogene-tumor suppressor dynamic control of cancer development

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Myc and p53 are both transcription factors, each affecting the expression of thousands of genes. Two recent census of human cancer genes list Myc and p53 in the top groups of frequently amplified and mutated genes, respectively. Myc is an oncogene that promotes cell proliferation and inhibits cell differentiation, whereas p53 is a tumor suppressor gene that inhibits proliferation and promotes differentiation. We have summarized the published literature on pathways of interactions between Myc and p53; and we show that most of the pathways form negative feedback loops (i.e., Myc upregulates p53 activity while p53 downregulates Myc). In many cancers, however, positive feedback interactions (i.e., mutual antagonism) between Myc and p53 may predominate. I will discuss how one can quantify the strengths of the positive and negative feedback loops, thereby allowing us to make predictions on how to control Myc activity. I will also give other examples of oncogene-tumor suppressor gene interactions and argue for the proposal that cancer therapy should be designed in the context of controlling the network of interactions between oncogenes and their associated tumor suppressor genes.

Biography

Aguda obtained his Ph.D. in Chemistry (Chemical Physics Program) and was a faculty member of a few Canadian and US universities before he re-focused his research program on cancer systems biology and moved to NCI in 2010. Some of his substantial contributions include the development of predictive network and kinetic models of mammalian cell cycle checkpoints such as the Restriction Point and the G2 DNA Damage Checkpoint Pathway. He and Professor Avner Friedman wrote the graduate-level book *Models of Cellular Regulation* published in 2008 by Oxford University Press. At the NCI, He works in the field of network pharmacology.

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