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Dysregulated metabolic pathways in cancer cell growth and drug resistance

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The metabolic properties of cancer cells are different from those of normal cells. Energy production in cancer cells is abnormally dependent on aerobic glycolysis. In addition to the dependency on glycolysis, cancer cells have other atypical metabolic characteristics such as increased fatty acid synthesis and increased rates of glutamine metabolism. Emerging evidence shows that many features characteristic to cancer cells, such as dysregulated Warburg-like glucose metabolism, fatty acid synthesis and glutaminolysis are linked to therapeutic resistance in cancer treatment. Therefore, targeting cellular metabolism may improve the response to cancer therapeutics and the combination of chemotherapeutic drugs with cellular metabolism inhibitors may represent a promising strategy to overcome drug resistance in cancer therapy. By targeting the metabolic pathways that import, catabolize, and synthesize essential cellular components, drug resistant cancer cells can often be resensitized to anti-cancer treatments. The specificity and efficacy of agents directed at the unique aspects of cancer metabolism is expected to be high; and may, when in utilized in combination with more traditional therapeutics, present a pathway to surmount resistance within tumors that no longer respond to current forms of treatment.

Biography

Ming Tan is an Associate Professor, Chief of Center for Cell Death and Metabolism, and an Endowed Scholar at Mitchell Cancer Institute, University of South Alabama. Upon earning his Ph.D. degree in cancer biology from the University of Texas MD Anderson Cancer Center, Tan continued his research on the mechanisms of oncogene-induced cancer progression and therapeutic resistance as a postdoctoral fellow. He joined the faculty at Mitchell Cancer Institute at the University of South Alabama in 2007, where he conducted research on metabolism and cancer, mechanism of cancer cell therapeutic resistance, invasion and metastasis, and microRNA in cancer development.

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