Title: Targeting Cancer Metabolism

Speaker

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Chi Van Dang is Director of the Abramson Cancer Center of the University of Pennsylvania and the John H. Glick Professor. His career at Penn started in September 2011 after having been at Johns Hopkins when he was the Johns Hopkins Family Professor in Oncology Research and Vice Dean for Research of Johns Hopkins University School of Medicine. He directed the Hopkins Institute for Cell Engineering and was a Professor of Medicine, Pathology, Oncology, and Cell Biology with joint appointment in Molecular Biology and Genetics. Dr. Dang is Editor-in-Chief of Cancer & Metabolism, a scientific editor of Cancer Discovery and serves on editorial boards of Cancer Research, Clinical Translational Science, Current Cancer Therapy Reviews, eLIFE, Journal of Clinical Investigation, Journal of Molecular Medicine, Genes & Cancer, Molecular and Cellular Biology, Neoplasia, and Oncotargets. He has authored over 200 scientific and medical articles, book chapters and a book. He is a member of the Institute of Medicine of the National Academy of Sciences, American Academy of Arts & Sciences, National Cancer Institute Board of Scientific Advisors, American Society for Clinical Investigation (ASCI) and The Association of American Physicians. He was president of the ASCI (2003). He held an NIH/National Cancer Institute MERIT award, received a number of honors, and sponsored and mentored many NIH K08 physician-scientist awardees, Ph.D. doctorates and post-doctoral fellows. The Dang laboratory has contributed to the understanding of the function of the MYC cancer gene (www.myccancergene.org), which has emerged as a central transcription factor or gene switch in many different human cancers. His laboratory established the first mechanistic link between the MYC cancer gene and cellular energy metabolism, contributing to the concept that genetic alterations in cancers re-program fuel utilization by tumors and render cancers addicted to certain fuel sources. His laboratory is now exploiting these concepts for therapeutic targeting of cancer cell metabolism as a new way to treat cancer.

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ABSTRACT:

Canonical oncogenes and tumor suppressors involved in cell growth and proliferation are intimately linked to metabolic pathways, which in turn affect tumorigenesis. The MYC oncogene regulates gene expression and stimulates metabolism in favor of cell growth and proliferation. Deregulation of MYC in cancers is surmised to stimulate cell growth without extracellular cues, rendering tumorigenic cells addicted to glucose, glutamine, and fatty acids. In this regard, experiments to inhibit key enzymes, such as lactate dehydrogenase A (LDHA) and glutaminase (GLS), have provided proof-of-concept for targeting cancer metabolism. Because glycolysis and glutaminolysis are also used by some normal cells for growth, we also investigated an additional potential therapeutic window through the natural circadian metabolic rhythm of normal cells that is presumed to be disrupted in certain cancer cells. We found that MYC is able to directly perturb circadian transcription factors, disrupts circadian rhythm, and causes sustained metabolic deregulation, which could be exploited for chrono-metabolic therapy.