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The role of centrosomal protein TAX1BP2 in the pathogenesis of hepatocellular carcinoma

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Emerging evidence suggests that supernumerary centrosomes drive chromosomal instability and is linked to oncogenesis. We have recently characterised a novel cellular centrosomal protein, which we named TAX1BP2 and have shown to play an important role in centrosome duplication. TAX1BP2 is targeted by human T cell leukaemia virus (HTLV-I) to create chromosome instability and aneuploidy. While aneuploidy is frequently observed in human liver cancer, we hypothesize that dysregulation of TAX1BP2 plays a role in the hepatocarcinogenesis. Using quantitative RT-PCR, our data has suggested that TAX1BP2 was frequently lost in human HCC. The underexpression of TAX1BP2 transcript was significantly associated with shorter overall survival rates, thus a poorer prognosis, in HCC patients. Furthermore, we demonstrated that TAX1BP2 is regulated by CDK2 kinase and the regulation of TAX1BP2 by CDK2 is tightly linked to the upregulation of tumor suppressor, p53. Thus, we propose that TAX1BP2 is a novel tumor suppressor in liver cancer.

Biography

Yick-Pang Ching has studied hepatocellular carcinoma for more than 10 years, during which he has authored more than 60 peer-reviewed papers, including paper in Nature Cell Biology, Cancer Research and Hepatology. He has served on the editorial boards for the PLoS One, and the International Journal of Oncology.

Low frequency KRAS mutations in colorectal cancer patients and the presence of multiple mutations in oncogenic drivers in non-small cell lung cancer patients

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Intra-tumor heterogeneity can confound mutational status in oncodriver genes and challenge targeted cancer therapy strategies. Ultra-deep sequencing can detect low-frequency and expanded clonal mutations in primary tumors to better inform treatment decisions. KRAS coding exons in 61 treatment naive colorectal cancer (CRC) tumors were sequenced, with KRAS, EGFR, ALK, and MET in lung tumors from 3 Chinese non-small cell lung cancer (NSCLC) patients.

Forty-two percent of CRC patients (28/61) harbored mutations in the KRAS active domain, eleven of which (18%) not detected by Sanger sequencing; 5/11 had frequencies <10%, and 12 patients harbored >1 mutation. Low frequency KRAS active (G12R) and EGFR kinase domain mutations (G719A) were identified in one NSCLC patient. Multiple low frequency mutations in KRAS, EGFR, and MET and ALK gene copy number increases were found in a second NSCLC patient. A third NSCLC patient showed EML4-ALK fusion with ALK, EGFR, and MET mutations. Within the same patient, multiple low frequency mutations occurred within a gene.

A complex pattern of intrinsic low frequency driver mutations in well-known tumor oncogenes may exist prior to treatment, resulting in resistance to targeted therapies. Ultra-deep sequencing can characterize intra-tumor heterogeneity and identify such mutations to ultimately impact treatment decisions.

Biography

Yihong Yao is director and head of Pharmacogenomics and Bioinformatics Group at MedImmune, LLC. The focus of Dr. Yao's group is the utilization of cutting edge genomics and genetics approaches to develop pharmacodynamics and predictive diagnostic markers to understand disease linkage and to identify the right patients that might respond (or not respond) to therapeutic interventions. The other areas of interest in Yao's group include: to unveil potential key drivers in cancer, respiratory and inflammatory diseases, and to understand the role of miRNAs in disease pathogenesis.

He received a bachelor's degree in Biochemistry from Fu Dan University in 1988. He received a master's degree in Bioinformatics from Boston University. He completed his doctorate in Biochemistry and Biophysics from the University of Kansas in Lawrence, Kansas. He conducted his postdoctoral research at Johns Hopkins Medical School in Baltimore, Maryland. He has authored over 50 peer-reviewed publications, edited two books and has over 15 patents.