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Role of fusion genes in human cancers

Chromosome rearrangement is some of the key features of human malignancies. Recently, we discovered a panel of cancer-specific fusion genes human cancers. Among these fusion genes, MAN2A1-FER and SLC45A2-AMACR appear to activate oncogenic pathways and lead to spontaneous cancer development in the animals. Fusion of MAN2A1-FER generates a constitutively activated tyrosine protein kinase. The fusion translocates FER kinase from the cytoplasm to Golgi apparatus. The fusion protein ectopically phosphorylates the N-terminal domain of EGFR and several growth factor receptors. MAN2A1-FER has been found in a variety of human malignancies. It transforms immortalized cell lines into highly aggressive cancer cells. Expression of MAN2A1-FER increases cancer cell proliferation, invasion, and metastasis. Hydrodynamic tail vein injection of MAN2A1-FER expressing vectors produces spontaneous liver cancer in animals. Targeting at MAN2A1-FER or other fusion genes using small molecules, immunogenic reagents and genomic approach showed the effective killing of cancer cells containing these fusion genes both *in vitro* and *in vivo*. Thus, fusion gene targeting holds promise for effective treatment of human cancers.

Biography

Jianhua Luo has been studying molecular pathology related to human malignancies in the last 28 years. Currently, he is a Professor of Pathology and Director of High Throughput Genome Center at the University of Pittsburgh. In the last 17 years, he has been largely focusing on the genetic and molecular mechanism of human prostate and hepatocellular carcinomas. In this period, his group has identified and characterized several genes that are related to prostate cancer and hepatocellular carcinoma, including SAPC, myopodin, CSR1, GPx3, ITGA7, MCM7, MCM8, MT1h, and GPC3. He has characterized several signaling pathways that play the critical role in prostate cancer development, including Myopodin-ILK-MCM7 inhibitory signaling, myopodin-zyxin motility inhibition pathway, CSR1-CPSF3, CSR1-SF3A3, and CSR1-XIAP apoptotic pathways, MT1h-EHMT1 epigenomic signaling, ITGA7-HtrA2 tumor suppression pathway, GPx3-PIG3 cell death pathway, AR-MCM7, MCM7-SF3B3, and MCM8-cyclin D1 oncogenic pathways. He proposed prostate cancer field effect in 2002. He is one of the pioneers in utilizing high throughput gene expression and genome analyses to analyze field effects in prostate cancer and liver cancer. He is also the first in using methylation array and whole genome methylation sequencing to analyze prostate cancer.

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