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Target Raf/MEK/ERK kinase cascade for cancer therapy

Ras/Raf/MEK/ERK signaling plays a critical role in cell proliferation, differentiation and survival. Its dysfunction leads to developmental diseases and cancers. Recent studies have shown that hyperactive Ras/Raf/MEK/ERK signaling exists in >40% human cancers, which functions as a driver therapy, both Raf and MEK inhibitors have been developed and applied to clinical treatment. Although these inhibitors achieved a good efficacy in the short-term treatment of some BRAF (V600E)-positive cancers, cancers with genetic alterations on Ras or upstream exhibit intrinsic resistance to Raf/MEK inhibitors, appealing a complete understanding of molecular mechanisms that control Raf/MEK/ERK kinase cascade. In current studies, we employed molecular biology, biochemistry, cell biology and tumor xenograft model to dissect molecular details by which Raf kinase activates MEK and in turn MEK triggers ERK under both normal and pathological conditions. Our findings provided a novel target for the design of next generation of Raf/MEK inhibitors.

Biography

Jiancheng Hu has received his PhD from University of Colorado Denver in 2007. He has then worked at Washington University in St. Louis and Howard Hughes Medical Institute as a Post-doctoral Research Fellow. In 2014, he has joined National Cancer Centre Singapore where he has served as the Principal Investigator at the Laboratory of Cancer Signaling. Currently, he is an Assistant Professor in Cancer and Stem Cell Program, Duke-NUS Medical School, National University of Singapore. His research interests include the regulatory mechanism of Raf kinase and other oncogenic protein kinases under normal/pathological conditions, molecular basis that underlie intrinsic and acquired resistance of kinase inhibitors in clinic treatment of cancers and the development of novel kinase inhibitors.

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