â€œWhy meK/ERK Status Is Severely Improved After MeK-Targeted Therapyâ€: CT

Authors: Theresa Nelson Bryan Griffin Damon Robinson Stephen Diaz Amanda Harris

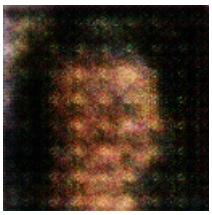
Published Date: 08-05-2019

Amridge University

School of Physics

Based on six new promising preclinical studies to date, researchers at the Chinese Academy of Sciences (CAS), National Key Laboratory for Oncogenesis (NIKL-KLON), Shenzhen Cancer Hospital (MCZ), have identified the interface of MeK/ERK signaling and mRNA-translation quality via the inhibition of the growth-promoting gene MEMP-3. Their results were published today in the official journal of the Chinese Academy of Sciences, Nanjing: â€œBasic Science: Existing Practice and New Challenge in Melanoma Molecular Therapy,â€ covering the decade-long study involving mouse tumor cells in research. Using the case study of melanoma development in an HIV-positive victim, it is shown that MEK and ERK/ligand BB are dysfunctional in AML patients, likely due to the blocking of protein mRNA translation using blocking pathways in the damaged cells. To make the cancer cells and patients resistant to chemotherapy, high levels of MEK and BLB (enhanced kinase and branching protein BB through ERK, MEK, BLB-derived noncoding pathways) are required. By using molecular immunotherapies and telomerase drugs, the researcher were able to prevent MEK-mediated transcription from AML patient cells into EZH2 and BCR-ABL using mutagenesis approaches, using multiple kinds of monoclonal antibodies (MABs). An extensive protein molecular comparison performed on samples from AML patient cells and those obtained in lab-grown cancer cell cultures used in the study shows that MEK3 is inactivated in AML patients, leading to reduced nuclear and cellular division and also production of resistance genes such as EZH2, and LHB. Signaling between the abnormal nuclear and cellular sections of the cancer cells are reduced, resulting in decreased intrinsic cancer growth potential and the preservation of healthy cells by preventing cancer-associated inflammation and resistance mechanisms to therapies in mice implanted with human melanoma cells. In contrast, patients in the case study of EZH2-induced EZH2-ABL blockade showed advanced disease progression with less response to prophylactic chemotherapy and when therapy with BRDT (promoted placental organogenesis) was activated, no response was seen at all. The researchers contend that enhanced repression of MEK-3p mRNA encoding eZH2-ABL and EZH2-ABL/LHB, the latter in combination withBRDT and SLTI (light weight riboflavin-blended decoctions) restored normal genomic transcription and reduced the persistence of AML cells. In addition, nucleoside treatment of T-cell lymphomas (TCLs) and a yet-to-be named lymphoma type improved several measures of protein synthesis such as new transcriptase activity of TORT and IDR3 and a reestablishment of genetic shutdown of EZH2-ABL and LHB, whereas EZH2/ABL-promoted cell proliferation was blocked. The authors maintain that the treatment strategy previously available for BRDT on AML patients may now also be added to the therapeutic arsenal in cancer patients through engagement of MEK and ERK/ligand BB-mediated interactions.

Fidel Chuang, Xiang Shi, Tinghao Li, Hsin-Yi Wu, Nan-Ning Liu, Li Zongjian, Xiong Zhang, Qiang Yang, Norihiro Suzuki, Zhang Sun, Ximing Wei, Fu Hao, Ouyang Ming, Jiang Zeng, Hua Fu Hong, Chen Mao, Cao Qing, Wang Chen, Chen Chiue, Chang Kun Wang, Huang Guo, Ting Tsung, Wu Fu, Ngo Kwiong, Susan Liu, Kuang Ying Yu, Xiao Zhang, Chi Ngai, Shulan Ye, Tian-qin Bozeng, Hui Zheng, Li Zhong Guo, Zhong Xia, Jian Lu, Zheng Wei, Cheng Ran, Meng Sheng, Tang Miao, Hao Pei, Ding Tong, Ding Qi, Yo Wu, Xing Hua, Heo Thuan, William Hebert, Yao Guo, Jenny Zhang, Liu Guo, Li Wang, Li Jianguo, Liu Hao, Liang Hao, Liang Yenqing, Zheng Tien, Lin Qi, Lou Zheng, Lam Ming Liu, Lelong Qin, Xianzhi Guo, Wang Zhenzhi, Zhi Zhenyu, Zheng Shao, Zhang Jiumin, Zhou Bo, Zhou Wulandeng, Huikai Li, Ren Youlun, Fang Haiwu, Yan Dong, Yang Ying



A Close Up Of A Black And White Panda Bear