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Short Article

World Cancer 2021: CyH enhances NSCLC cell sensitivity to Gefitinib by mediating EGFR activation and PD-L1 expression - Xudong Tang - Guangdong Medical University, China

Xudong Tang

Guangdong Medical University, China

Abstract

In our previous study, we have isolated cytochalasin H (CyH) from endophytic fungus derived from mangrove plant in Zhanjiang. Recently, epidermal growth factor receptor (EGFR) activation and programmed cell death 1 ligand (PD-L1) expression have been demonstrated to mediate non-small cell lung cancer (NSCLC) resistance to Gefitinib. Here, we further investigated the effect of CyH on EGFR activation and PD-L1 expression and Gefitinib sensitivity in NSCLC cell lines, A549 (wild-type EGFR), HCC827 (EGFR mutation), and NCI-H1975 (dual EGFR mutations and acquired Gefitinib resistance). Our results showed that CyH significantly inhibited EGFR activation and PD-L1 expression in A549, HCC827, and NCI-H1975 cells. Additionally, CyH dramatically promoted the inhibitory effect of Gefitinib on proliferation of A549 and HCC827 cells, and enhanced NCI-H1975 cell sensitivity to Gefitinib ($P<0.05$). Moreover, CyH increased the inhibitory effect of Gefitinib on EGFR activation and PD-L1 expression in A549, HCC827 and NCI-H1975 cells. Mechanically, CyH was found to inhibit the activation of JAK3-STAT signaling pathway. Taken together, our findings suggest that CyH enhances NSCLC cell sensitivity to Gefitinib by mediating EGFR activation and PD-L1 expression, which may be related to the inhibition of JAK3-STAT signaling pathway.

Biography

Xudong, PhD, MD, Professor. She is the associate director of Institute of Biochemistry and Molecular Biology. She has published more than 70 papers in Chinese and English journals.