Mechanism of Cell Cycle Inhibition by FIP200

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Technology description

The disclosure related to a novel protein inhibitor for focal adhesion kinase (FAK), which binds to FAK directly and inhibits its kinase activity and associated cellular functions.
Cancer is considered to be a disorder of cell cycle function. Furthermore, cell cycle deregulation is commonly seen during tumor development. Cornell researchers have discovered that FIP200 affects critical regulators of the cell cycle: cyclin D1 and p21. As a result, FIP200 inhibits G1-S phase progression, proliferation, and clonogenic survival in human breast cancer cells.
FIP200 functions by inhibiting Focal Adhesion Kinase (FAK) activity and downstream cellular functions, including cell adhesion, spreading, and motility in fibroblasts. FIP200 also induces G1 arrest, by increasing p21 and decreasing cyclin D1 protein levels in breast cancer cells. Furthermore, FIP200 can interact with exogenous and endogenous p53 protein and significantly increase its half-life compared to the control cells. Normally p53 functions to induce apoptosis and it is directly mutated in 50% of all human cancers. Even in the majority of tumors with wild type p53, p53 activity is affected due to poor stabilization or increased degradation of wild type p53.

Application area

FIP200 inhibits G1-S phase progression, proliferation, and clonogenic survival in human breast cancer cells and may play such an important role in other cancer cells.
FIP200-induced G1 arrest may contribute to potential new anti-tumor treatments.
Mechanisms of p53 stabilization by FIP200 merits further investigation and may reveal new therapeutic strategies for p53 stabilization in cancer cells.

Institution

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