عنوان مقاله:

Valproic acid inhibits cell proliferation and PD-L۱ mediated tumor immune escape throughtargeting CIP۲A and c-MYC/PI۳K/Akt/mTOR signalling molecules in breast cancer

محل انتشار:

پانزدهمین کنگره بین المللی سرطان پستان (سال: 1400)

تعداد صفحات اصل مقاله: 1

نویسندگان:

Elahe Zeinali - *Department of Immunology and Faculty of Medicine, Birjand University of Medical Sciences, Birjand, Iran*

Vahid Bagheri - *Cellular and Molecular Research Center, Birjand University of Medical Sciences, Birjand, Iran*

Esmaeil Rostami - *Department of Laboratory Sciences, Faculty of Para- Medicine, Sabzevar University of Medical Sciences, Sabzevar, Iran*

Gholamreza Anani Sarab - *Cellular & Molecular Research Center, Birjand University of Medical Sciences, Birjand, Iran.*

خلاصه مقاله:

Resistant cells are a critical problem that reducetreatment efficacy of breast cancer. Nowadays,CIP۲A and PD-L۱ are considered as theraputicalchallenges in breast cancer, because ofresponsible for drug resistance and immuneevasion respectively. Hence, identifying agents tosuppress these factors is great of interest.Specifically, epigenetic drugs can be an effectiveapproach to alter the behavior of genes. Althoughvalproic acid (VPA) as a HDAC inhibitor hascertain anticancer properties but molecularmechanism of VPA in breast cancer cells remainsto be explored. In this study, we investigated drugeffects and molecular mechanisms of VPA,particularly its effect on CIP۲A and PD-L۱ inbreast cancer MCF-۷ cell line.In this study, MCF-۷ cells were treated withvarious concentration of VPA for ۲۴ h, ۴۸ h, and۷۲ h. The rate of cell viability was measured byMTT assay. Finally, gene expressions of CIP۲A,c-MYC, PI۳K, Akt, mTOR and PD-L۱ wereanalyzed by real time PCR and ΔCT method.

کلمات کلیدی:

Valproic acid (VPA);histone deacetylase inhibitor(HDACi); CIP۲A, PD-L۱; breast cancer

لینک ثابت مقاله در پایگاه سیویلیکا: