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Trichostatin A Induced Bcl-2 Protein Level Decrease Mediated A549/CDDP Cells Apoptosis by Mitochondria Pathway

Jun WU, Chengping HU

摘要

Background and objective The use of platinum-based combination chemotherpy remains the standard treatment for non-small cell lung cancer. However, the resistance to platinum limits further treatment clinically. Trichostatin A (TSA) is one of histone deacetylase (HDAC) inhibitors. It inhibits tumor cell proliferation and acts as a chemosensitizer. The aim of this study is to investigate the action mechanism of TSA on cisplatin-resistant human lung adenocarcinoma cell line A549/CDDP. Methods Cytotoxicity and cell viability was assayed by Neutral Red method. Morphologic assessment of apoptosis was determined by fluorescence microscope; cell cycle and mitochondrial membrane potential were detected by flow cytometry. In addition, A549/CDDP cells were transfected with Bcl-2 expression Vector and siRNA-bcl-2. Results A549/CDDP cells treated with TSA showed apparently cytotoxicity, IC50 of TSA was (446.59±27.32) nmol/L. The growth curve showed the ratio of growth decreased with the increase of concentration of TSA. The apoptosis appeared 24 hours after treated by (125-500) nmol/L TSA, morphologic changes including nuclear chromatin condensation. Fluorescence strength was observed with fluorescence microscope. Treated by TSA, mitochondrial membrane potential was decreased and cells were arrested at S phase. Western blotting analyses showed that the levels of Bcl-2 decreased, while expression of Bax increased. Simultaneously caspase-3 was activated. Over expression of Bcl-2 can inhibit TSA-induced A549/CDDP cell apoptosis, while the decrease of Bcl-2 enhanced the sensitivity of A549/CDDP cell to TSA. Conclusion TSA induce A549/CDDP cell apoptosis by mitochondria pathway.

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ABOUT THE AUTHORS

Jun WU

Chengping HU

thoracic CANCER
主编
Qinghua Zhou
Yan Sun
www.thoraciccancer.net

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