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Rhein induces apoptosis and autophagy in human and rat glioma cells and mediates cell differentiation by ERK inhibition

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Highlights

- Anticancer potentials of Rhein, was assessed on the rat F98 glioma cells.
- Rhein induced cell cycle arrest, caspase mediated apoptosis.
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Rhein treatment did not alter the phosphorylated MAPKs activation including p-38, JNK and NF-κB, transcription unit.

- Rhein significantly inhibited ERK1/2 activation in F98 glioma cells.

Abstract

In this study, we investigated the anticancer potentials of Rhein, an anthraquinone derivative of most commonly used Chinese rhubarb on the rat F98 glioma cells. The experimental studies revealed that Rhein induced cell cycle arrest, caspase mediated apoptosis. It results in the formation of intracellular acidic vesicles in cytoplasm, leading to autophagy. Differentiation of viable cells towards elongation of matured astrocytes was proved by monitoring dramatic changes in morphological characteristics as well as identified from the elevation of glial fibrillary acidic protein (GFAP) expression. Rhein treatment did not alter the phosphorylated MAPKs activation including p-38, JNK and NF-κB, transcription unit whereas rhein significantly inhibited ERK1/2 activation in F98 glioma cells. PD98059, a specific inhibitor for ERK activation imitates rhein effects on morphology and expressions of GFAP but did not help to induce any apoptosis or autophagy. Collective data exhibited that potentials of rhein in anti-cancer property in ERK-independent apoptosis and autophagy in association with downregulated ERK-dependent differentiation process of glioma cell lines.

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Keywords

Glioma; Rhein; GFAP; ERK pathway

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