Oridonin

Oridonin is purified from the Chinese herb Rabdosia rubescens and considered to exert extensive anticancer effects on human tumorigenesis.

A study examined the antitumor function of oridonin in Central neurocytoma (CN) cells, and investigated the underlying molecular mechanism. An MTT assay suggested that treatment with oridonin was able to significantly inhibit the proliferation of CN cells. The annexin V-fluorescein isothiocyanate/propidium iodide assay and western blot analysis demonstrated that oridonin was able to induce apoptosis and alter the expression of apoptosis-associated proteins by downregulating anti-apoptotic protein, B-cell lymphoma-2 (Bcl-2), and upregulating pro-apoptosis proteins, Bcl-2-like protein 4, cleaved caspase-3 and cleaved poly(ADP-ribose) polymerase 1. Subsequently, the Wnt/β-catenin signaling pathway was examined. Western blot analysis indicated that oridonin markedly decreased the expression of β-catenin, cyclin D1 and v-myc avian myelocytomatosis viral oncogene homolog. Furthermore, β-catenin was silenced by small interference RNA or overexpressed in CN cells, and the effect on cell proliferation was examined. The results indicated that silencing of β-catenin enhanced the inhibitory effect of oridonin on cell growth, whereas the overexpression of β-catenin attenuated this effect. These data indicated that oridonin inhibited proliferation and induced apoptosis to exert its antitumor activity in CN cells by repressing Wnt/β-catenin signaling. Therefore, the present study suggested that oridonin might be an effective adjuvant agent, and that the Wnt/β-catenin signaling pathway may be a potent target for the therapy in CN [1].

In a study, Zhang et al. systemically investigated the role of Oridonin in tumor growth and the underlying mechanisms in human glioma. We found that Oridonin inhibited cell proliferations in a dose— and time—dependent manner in both glioma U87 and U251 cells. Moreover, these anti—cancer effects were also confirmed in a mouse model bearing glioma. Furthermore, cell cycle arrest in S phase was observed in Oridonin—mediated growth inhibition by flow cytometry. Cell cycle arrest in S phase led to eventual cell apoptosis, as revealed by Hoechst 33342 staining and annexin V/PI double—staining. The cell apoptosis might be accomplished through a mitochondrial manner. In all, we were the first to our knowledge to report that Oridonin could exert anti—cancer effects on tumor growth in human glioma by inducing cell cycle arrest and eventual cell apoptosis. The identification of Oridonin as a critical mediator of glioma growth may potentiate Oridonin as a novel therapeutic strategies in glioma treatments [2].

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2)
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