

Baicalein induces proliferation inhibition in B16F10 melanoma cells by generating reactive oxygen species via 12-lipoxygenase

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摘要

Abstract

In a previous study, we demonstrated that baicalein induces hydroxyl radical formation in human platelets but the mechanisms are unclear. Herein, we show, using an electron spin resonance technique, that baicalein also induces hydroxyl radical formation in B16F10 melanoma cells in a dose-dependent manner. Baicalein produced superoxide anions in the presence of an iron chelator and superoxide dismutase (SOD) inhibitor. We suggest that superoxide anions produced by baicalein were promptly converted to hydroxyl radicals through SOD and the Fenton reaction in B16F10 melanoma cells. According to Western blotting results, the 12-LOX protein was expressed in B16F10 melanoma cells, but baicalein had no effect on 12-LOX expression. Decreases in 12-LOX protein expression and hydroxyl radical signals occurred in a 12-LOX small interfering RNA knockdown protein group compared with the baicalein control. In the MTT assay, we also found that baicalein caused a reduction in cellular viability, which was reversed by the addition of ROS scavengers. On the basis of these data, we conclude that ROS formation catalyzed by 12-LOX is one possible mechanism of growth inhibition by baicalein in B16F10 melanoma cells.