Opposite expression of securin and γ-H2AX regulates baicalein-induced cancer cell death.

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**Abstract**

Securin and γ-H2AX have been shown to regulate cell survival and genomic stability. However, it is still unknown how the expression and regulation of these proteins is altered following treatment with baicalein, a natural flavonoid extracted from the *Scutellaria baicalensis* root. In the present study, we investigate the possible roles of securin and γ-H2AX in baicalein-induced cancer cell death. Baicalein reduced cell viability in a variety of human cancer cell lines, including bladder, cervical, colon, and lung cancer cells. Interestingly, baicalein treatment (40-80 µM for 24 h) markedly inhibited securin expression, while the levels of γ-H2AX were elevated. Abnormal spindle formation and chromosomal segregation were induced by baicalein. Furthermore, wild type HCT116 cancer cells had a higher incidence of cytotoxicity and apoptosis than securin-null HCT116 cells following treatment with baicalein. In contrast, baicalein increased the levels of γ-H2AX to a similar extent in both cell types. Transfection with H2AX siRNA further increased baicalein-induced cell death. Additionally, blockade of the AKT pathway by treatment with wortmannin or AKT shRNA lowered the levels of γ-H2AX and enhanced cytotoxicity in baicalein-treated cells. Taken together, our findings suggest that the opposing effects of baicalein on securin and γ-H2AX levels may be involved in the regulation of cell viability and genomic stability by this compound.

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