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Baicalein increases the expression and reciprocal interplay of RUNX3 and FOXO3a through crosstalk of AMPKα and ERK1/2 signaling pathways in human non-small cell lung cancer cells

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Baicalein, a natural flavonoid obtained from the *Scutellaria baicalensis* root, has been reported to inhibit growth of human lung cancer. However, the detailed mechanism underlying this effect has not been well elucidated. We showed that baicalein significantly inhibited the growth and induced apoptosis of NSCLC in a time- and dose-dependent manner. Baicalein induced RUNX3 and FOXO3a mRNA and protein expression, and increased phosphorylation of AMPKα and ERK1/2. Moreover, the inhibitors of AMPK and ERK1/2 reversed the effect of baicalein on RUNX3 and FOXO3a protein expression. Interestingly, while compound C had little effect on blockade of baicalein-induced phosphorylation of ERK1/2, PD98059 significantly abrogated the baicalein-induced phosphorylation of AMPK . Intriguingly, while silencing of RUNX3 abolished the effect of baicalein on expression of FOXO3a and apoptosis, silencing of FOXO3a significantly attenuated baicalein-reduced cell proliferation. On the contrary, overexpression of FOXO3a restored the effect of baicalein on cell growth inhibition in cells silencing of endogenous FOXO3a gene and enhanced the effect of baicalein on RUNX3 protein expression. Finally, exogenous expression of RUNX3 increased FOXO3a protein and strengthened baicalein-induced phosphorylation of ERK1/2 and. Collectively, our results show that baicalein inhibits growth and induces apoptosis of NSCLC cells through AMPKα- and ERK1/2-mediated increase and interaction of FOXO3a and RUNX3. The crosstalk between AMPK and ERK1/2 signaling pathways, and the reciprocal interplay of FOXO3a and RUNX3 converge on the overall response of baicalein. This study reveals a novel mechanism for regulating FOXO3a and RUNX3 signaling axis in response to baicalein and suggests a new strategy for NSCLC associated targeted therapy.

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