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第二十八屆生物醫學聯合學術年會 **ABSTRACT FORM** （正本）

黃芩素促進口腔癌細胞 $p27^{kip}$ 降解的機制

Baicalein Enhances $p27^{kip}$ Degradation in Oral Cancer Cells

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Backgrounds:

While most studies in cancer cell lines report that baicalein causes cell cycle arrest by increasing the expression of $p27^{kip}$, we found that in our preliminary data, the $p27^{kip}$ was decreased rather than increased in baicalein treated oral cancer cells, HSC-3. The reduction of $p27^{kip}$ was concomitant to the reduction of cyclinD1 and CDK4 as well as phosphorylated Rb (p-Rb), indicating its association with phosphrylation of Rb and growth inhibition. Thus, the purpose of the study is to elucidate how baicalein regulates $p27^{kip}$ expression in the oral cancer cell model.

Methods and Results:

Using cell cycle analysis, we found that baicalein treated cells was arrested at S phase at 24hr treatment. Data by Western Blot showed that $p27^{kip}$ was decreased at 12 and 24hr whereas phosphorylated $p27^{kip}$ (p-p27) was increased in baicalein treated cells. The reduction of $p27^{kip}$ in baicalein treated cells was time-correlated to the decrease of Akt and increase of p-Akt. This suggests that baicalein induces the phosphorylation of $p27^{kip}$, which is mediated by activation of Akt. It is known that the increase of p-p27 eventually leads to the degradation of $p27^{kip}$ and thus, decrease the expression of $p27^{kip}$. Interestingly, phosphorylated GSK-3β (p-GSK-3β) was also increased at the time when p-Akt was increased and $p27^{kip}$ was decreased, suggesting that GSK-3β pathway might be also involved in the modulation of $p27^{kip}$.

Conclusion:

Our data indicates that baicalein modulates the degradation of $p27^{kip}$ through Akt pathway in HSC-3. GSK-3β, the downstream protein molecule of Akt pathway might be an indirect effect of Akt on regulating the expression of $p27^{kip}$. Downregulation of $p27^{kip}$ has been reported to correlate to the malignancy and prognosis of oral cancers. The impact of baicalein on the reduction of $p27^{kip}$ in oral cancer cells shall be determined in terms of migration and invasion, in addition to its effect on growth inhibition.

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