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Erratum

The following was originally published in Volume 25, Number 5, pp. 809–817 (DOI: https://doi.org/10.3727/096504016X14799180778233). In the original article Figure 1 contained duplicate images in parts C and D. The corrected version of the figure is shown here, and the figure has been replaced with the corrected version in the original published article in the online site (https://www.ingentaconnect.com/contentone/cog/or/2017/00000025/00000005/art00017).

Kallistatin Suppresses Cell Proliferation and Invasion and Promotes Apoptosis in Cervical Cancer Through Blocking NF-κB Signaling

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Kallistatin has been recognized as an endogenous angiogenesis inhibitor and exerts pleiotropic effects in inhibiting tumor growth, migration, apoptosis, and inflammation. The purpose of the present study was to investigate the potential role and mechanisms of kallistatin in cervical cancer. We demonstrated that kallistatin effectively inhibited cell proliferation and enhanced apoptosis in a dose-dependent manner. Additionally, kallistatin suppressed migration and invasion activities and markedly reduced the expression of matrix-degrading metalloproteinases, progelatinase (MMP-2), MMP-9, and urokinase-type PA (uPA). Kallistatin reversed the epithelial–mesenchymal transition (EMT) and caused the upregulation of epithelial markers such as E-cadherin and inhibited mesenchymal markers such as N-cadherin and vimentin. Moreover, kallistatin led to a marked decrease in the expression of vascular endothelial growth factor (VEGF) and HIF-1α. In a xenograft mouse model, kallistatin treatment reduced tumor growth. Importantly, kallistatin strikingly impeded NF-κB activation by suppressing IκBκ degradation and the level of phosphorylation of p65. Interestingly, similar to kallistatin, treatment with PDTC (an inhibitor of NF-κB) also attenuated cell invasion and migration. Taken together, these findings suggest that kallistatin suppresses cervical cancer cell proliferation, migration, and EMT and promotes cell apoptosis by blocking the NF-kB signaling pathway, suggesting that kallistatin may be a novel therapeutic target for cervical cancer treatment.

Key words: Cervical cancer; Kallistatin; Migration; Apoptosis; NF-κB signaling

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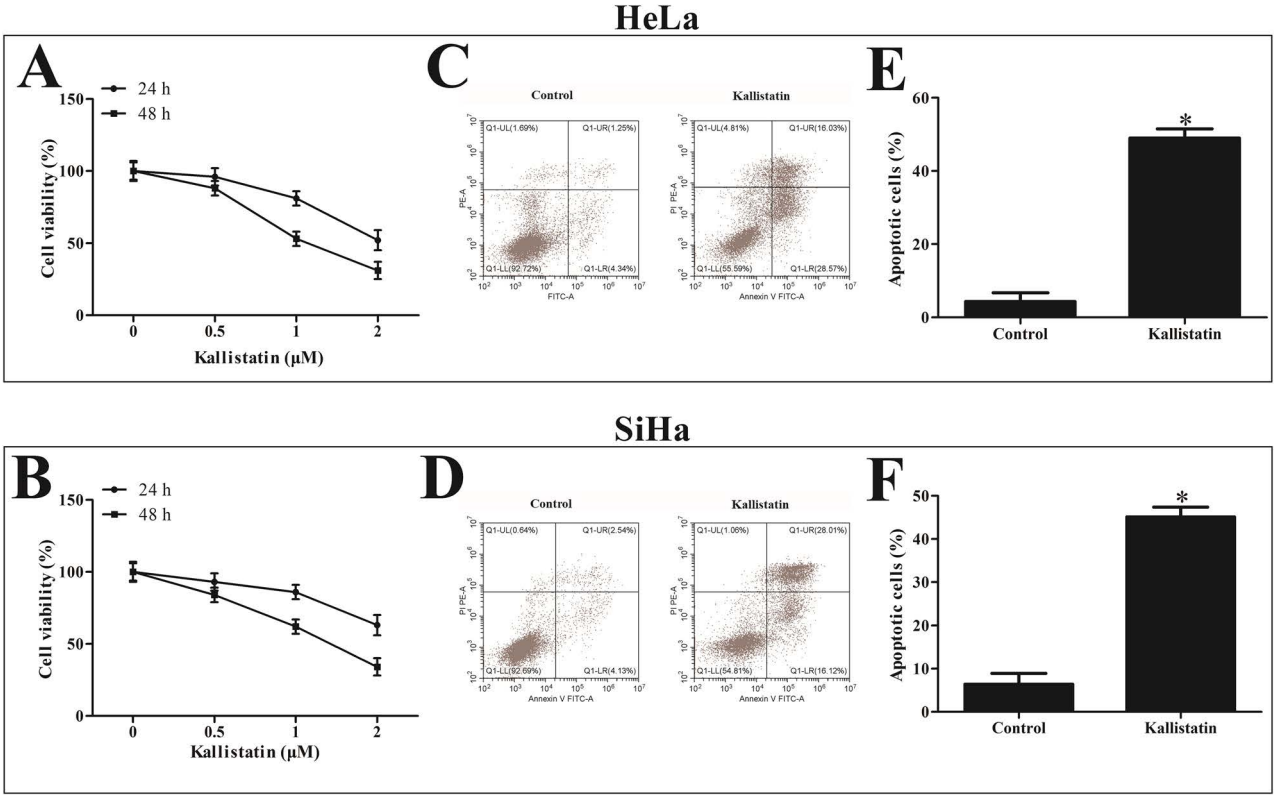

Figure 1. The effect of kallistatin on cell viability and apoptosis in human cervical cancer cells. HeLa (A) and SiHa (B) cells were treated with various concentrations of kallistatin for 24 and 48 h. Cell viability was assessed with the MTT assay. (C, D) Apoptotic cells were analyzed by flow cytometry with annexin V–FITC/PI staining. (E, F) Quantitative analyses of apoptotic populations following kallistatin treatment. Data are presented as the mean ± SD of at least three independent experiments. *$p < 0.05$ versus Control.