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5-Aza-2'-deoxycytidine may influence the proliferation and apoptosis of cervical cancer cells via demethylation in a dose- and time-dependent manner

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ABSTRACT. The methylation of tumor suppressor genes has been shown to be involved in many human cancers. 5-Aza-2'-deoxycytidine (5-Aza-CdR) can reactivate the expression of methylated tumor suppressor genes. In our study, 2 human cervical cancer cell lines, HeLa and SiHa, were treated with different concentrations (20, 10, 5, and 2.5 μM) of 5-Aza-CdR for 24, 48, and 72 h. After incubation, cells were analyzed by 3-(4,5-dimethyl-2-thiazolyl)-2,5-diphenyl-2H-tetrazolium bromide assay and flow cytometry. The expression of *RASSF1A* and *APAF-1* was detected by RT-PCR. 5-Aza-CdR inhibited the growth of HeLa and SiHa cells at different concentrations. The strongest inhibition and apoptosis rates were obtained after incubation for 72 h (5.63 ± 1.38 and

8.24 ± 2.40%, respectively). No significant difference in the expression of *RASSF1A* was found upon drug treatment, while *APAF-1* expression increased in HeLa cells after treatment (0.790 ± 0.056%). Our results suggest that the tumor-suppressive effect of 5-Aza-CdR may result from the reactivation of silenced *APAF-1* through demethylation.

Key words: 5-Aza-2'-deoxycytidine; Cervical cancer; *APAF-1*; DNA methylation; *RASSF1A*

