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MiR-155 Promotes Proliferation and Epithelial-mesenchymal Transition (EMT) and Inhibits Apoptosis of Cervical Cancer Cells via Regulating ING4

Min Du[1], Jibin Ye[2], Jianjun Jiang[2], Lan Huang[2], Shan Chen[2], Dayuan Yu[2] and Chuan Zhang[2,*]

[1]Department of Palliative Treatment, The Affiliated Cancer Hospital of Xinjiang Medical University, Wulumuqi, 83000, Xinjiang, China
[2]Palliative Medicine Department, West China School of Public Health and West China Fourth Hospital Sichuan University. No. 18, Section 3, Renmin South Road, Chengdu, Sichuan Province, 610041, China

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ABSTRACT Cervical cancer, the fourth common gynecologic malignancy, causes huge menace to female health. This work studied the role of miR-155 and ING4 in changes in proliferation, and apoptosis in epithelial–mesenchymal transition (EMT) of cervical cancer cells. RNA levels of miR-155 and ING4 were assessed with RT-qPCR. MiR-155 inhibition and ING4 overexpression were achieved through transfection methods, whose expressions were evaluated by RT-qPCR. CCK-8 and flow cytometry measured cell viabilities and apoptosis, respectively. RT-qPCR was also implemented for examining expressions of E-cadherin, Snail and N-cadherin. MiR-155 was highly expressed in cervical cancer cells while its suppression retarded cell viabilities and EMT but enhanced the apoptosis. In contrast, ING4 expressions were significantly decreased in cervical cancer cells. Overexpression of ING4 blocked cell viabilities and EMT but promoted apoptosis. Moreover, overexpressed ING4 sharply inhibited cell viabilities and EMT and enhanced apoptosis. MiR-155 might accelerate proliferation, EMT and suppress apoptosis in cervical cancer cells in vitro while ING4 played an antipodal role. Further studies are needed for gaining comprehensive knowledge of miR-155 and ING4 in the future.