Investigation of the Effect of 5-Aza-2 '-Deoxycytidine in Comparison to and in Combination with Trichostatin A on p16INK4a, p14ARF, p15INK4b Gene Expression, Cell Growth Inhibition and Apoptosis Induction in Colon Cancer Caco-2 Cell Line

Sanaei Masumeh, Kavoosi Fraidoon, Ghasemzadeh Vahid

The cell cycle is divided into four phases, G1, G2, S, and M phase. The mammalian cell cycle is controlled and governed by the kinase complexes including cyclin and the cyclin-dependent kinase (CDK), cyclin-CDK complexes. The activity of the complexes is regulated by cyclin-dependent kinase inhibitors (CDKIs), the INK4, and the CDK interacting protein/kinase inhibitory protein (CIP/KIP) families. Promoter hypermethylation and histone deacetylation of CDKIs have been reported in several cancers. These changes can be reversed by DNA demethylating agents, such as decitabine, 5-Aza-2′-deoxycytidine (5-Aza-CdR), and histone deacetylase inhibitors (HDACIs), such as trichostatin A. Previously, we reported the effect of 5-Aza-CdR and trichostatin A (TSA) on hepatocellular carcinoma (HCC). The present study aimed to investigate the effect of 5-Aza-CdR in comparison to and in combination with trichostatin A on p16INK4a, p14ARF, p15INK4b genes expression, cell growth inhibition and apoptosis induction in colon cancer Caco-2 cell line.

Keywords: Colonic neoplasms, cyclin-dependent kinase inhibitors, decitabine, trichostatin A