Effect of Trichostatin A on Histone Deacetylases 1, 2 and 3, p21Cip1/Waf1/Sdi1, p27Kip1, and p57Kip2 Gene Expression in Breast Cancer SK-BR-3 Cell Line

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Abstract

Objective: DNA methylation, the covalent addition of a methyl group to cytosine, and histone modification play an important role in the establishment and maintenance of the program of gene expression. The balance of histone acetylation is determined by the activities of two groups of enzymes including histone acetyltransferases (HATs) and histone deacetylases (HDACs). Histone deacetylation is generally associated with silencing gene expression resulting in several solid tumors. HDAC inhibitors (HDACIs) are the new class of potential anticancer compounds for the treatment of the solid and hematological cancers. The current study was designed to evaluate the effect of trichostatin A (TSA) on histone deacetylases 1, 2 and 3, p21Cip1/Waf1/Sdi1 (p21), p27Kip1 (p27), and p57Kip2 (p57) gene expression in breast cancer SK-BR-3 cell line.
Materials and Methods: The breast cancer SK-BR-3 line was treated with TSA. To determine cell viability, cell apoptosis, and the relative expression level of the genes, MTT assay, cell apoptosis assay, and qRT-PCR were done respectively.
Results: TSA significantly inhibited cell growth, and induced apoptosis. Furthermore, this compound increased p21, p27, and p57 and decreased histone deacetylases 1, 2 and 3 gene expression significantly.
Conclusion: The TSA can reactivate the p21, p27, and p57 through down-regulation of histone deacetylases 1, 2 and 3 gene expression.