ID: 245

Critical Inhibitory Role of Linker Histone H1 in Self-Renewal of Cancer Cells

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Objective: Linker histone H1, with seven variants in human somatic cells, plays crucial roles in chromatin remodeling, nucleosome spacing and DNA methylation. H1 is one of the epigenetic regulators that inhibit cancer cell proliferation, while determines hierarchies in tumour population. It has been shown that chromatin configuration compatible with proliferation is only observed in cells with stable repression of H1, and that induced expression of H1 resulted in silencing of oncogenic and self-renewal genes. Clinical studies revealed differential expression of H1in cells derived from breast and kidney carcinoma, glioma, melanoma, and hepatocellular carcinoma, as H1 expression was high and low in cancer stem cells and differentiated cells, respectively. Other research has also determined that down regulation of each H1 variant in breast cancer cells resulted in altered gene expression in a variant specific manner, with H1.2 and H1.4 knock downs being most deleterious. In addition, it has been reported that H1.2 is responsible for apoptosis induction in leukemia cells, and such effect had been attributed to its structural homology with proteins secreted by activated macrophages and their tumoricidal activity.

Keywords: Histone H1, Cancer cell, Self-renewal, Epigenetic regulation

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