Epigenetic Therapy for Liver Cancer: The Effect of 5-azacytidine on the Expression of Tumor Suppressor Genes p15INK4b, p16INK4a, and SOCS-1

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This study examines epigenetic therapy as a treatment for slowing the growth of cancerous liver cells. More specifically, epigenetic changes such as the abnormal patterning of DNA hypo- and hypermethylation are known to be characteristics of cancerous cells and play critical roles in the regulation of the disorder. Hypermethylation, in particular, in the promoter regions of tumor suppressor genes is a hallmark of human tumors and leads to the transcriptional silencing of critical defense proteins responsible for tumor cell invasion, cell cycle control, DNA repair and other processes where silencing would lead to the spread of cancer. Thus in investigating epigenetic therapy as a treatment for cancer, this experiment tested the effect of 5-azacytidine (a DNA methyltransferase inhibitor on the growth of liver cancer by comparing cell counts, cellular viability rates, and examining the expression of tumor suppressor genes p15INK4b, p16INK4a, and SOCS-1 through RT-PCR and gel electrophoresis. The results (cell counts & images) show that 5-azacytidine (at 1.5uM = 3uM dosage) does not serve an important role in minimizing the growth of liver cancer cells; there was no statistically significant difference between treated and control cell counts and viability rates. Gel electrophoresis displayed the overall expression of p15INK4b and p16INK4a in the WB311 treated and control cells as absent except for the presence of a few random, faded bands. Bands from the SOCS-1 gene, though, appeared clearly with the treated DNA band appearing thicker than the control. I believe that 5-azacytidine may hold the potential to increase the expression of certain tumor suppressor genes but does not heavily influence the translation process whereby these genes produce proteins for cancer defense.