Synstatin-Mediated Inhibition of Syndecan-1 in Aggressive Hodgkin Lymphoma

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Hodgkin Lymphoma (HL) is a blood cancer that develops in the lymphatic system when a lymphocyte undergoes a malignant transformation and develops a DNA mutation. HL makes up about 1% of all cancer types and is considered a highly curable disease, with a 1-year overall survival (OS) rate of 92% and a 5-year OS rate of 86%. However, around 20% of patients who ailed from HL “experience either progressive disease, refractory disease, or relapse multiple times (< 4 years).” Through this study, I assessed Syndecan-1 (SDC1), a cell surface adhesion molecule that maintains cell morphology and interacts with the surrounding environment of the cell, as a biomarker for Hodgkin Lymphoma. SDC1 expression was analyzed in tissues of poor outcome patients and HL cell lines and compared to expression in good outcome patient samples and normal B-cells. SDC1 was found to be overexpressed in the former cases, suggesting its critical role in cell proliferation, migration, and invasion. Synstatin (SSTN) has been evaluated as a peptide that can inhibit SDC1 activity by mimicking the sequence of SDC1’s ectodomain and competitively blocking SDC1/integrin interaction with cell-matrix receptors. So, I explored the ability of SSTN to inhibit SDC1, thereby assessing its potential to be a drug candidate to treat HL patients with poor prognosis. This study showed that SDC1 is an appropriate diagnostic marker for aggressive HL and that SSTN exerts a dose-dependent, inhibitory effect on HL cell proliferation by competitively inhibiting SDC1 function.

Awards Won:

University of the Sciences in Philadelphia: Tuition Scholarship of $9,250. per year for four years.

University of Arizona: Tuition Scholarship Award