IDENTIFYING POTENTIAL ROLES FOR SIN3A AS A METASTASIS SUPPRESSOR IN E1B55K-DELETED AD INFECTED CELLS

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Synopsis:

The main goal of this project is to identify ways in which the gene Sin3a, a known metastasis suppressor in triple negative breast cancer cell lines, can suppress metastasis in other cell lines treated with E1B55K-deleted virus. We do this as another step into identifying therapies and additional treatment options for patients with metastatic disease, as they currently have limited treatment options available to them.
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Worldwide, there were 14.1 million new cases of cancer and 8.2 million deaths due to cancer reported in 2012. It is estimated that by the year 2030, 13 million people will die from cancer. Metastasis is a complex process that occurs when cancer cells travel to and grow in distant parts of the body, away from the original site of cancerous growth. One main contributor to the survival of cancer cells are genes that are associated with metastasis. These genes are able to be regulated due to chromatin-modification complexes. The study of metastasis is critical because it is the cause of most cancer deaths and there is a lack of treatment options available for patients with metastatic disease. Adenoviruses show promising capabilities for oncolytic viral gene therapy for cancer and specifically as a vector for vaccine delivery. One of the chromatin-modification complexes that have been shown to be involved in metastasis is the Switch-Independent 3 (SIN3) chromatin modification complex. Based on previous published data that demonstrates that the Adenovirus early region 1B55K (E1B55K) protein interacts with SIN3A, we hypothesize that SIN3A will be reduced in wild type infected cells and stabilized in E1B55K-deleted virus infected cells. Since SIN3A is reduced in metastatic cancer, a reduction in the levels of this product by Adenovirus might point to viral factors that are capable of enhancing metastasis. Here we infect human cancer cell lines, including Hela and A549 cell lines and use western Blot and immunoprecipitation assays to detect the presence of SIN3A.