Atovaquone Targets STAT3 in Ovarian Cancer Spheroids

Kayli E. Neil

University of New Hampshire, Durham

Follow this and additional works at: https://scholars.unh.edu/honors

Recommended Citation
https://scholars.unh.edu/honors/503

This Senior Honors Thesis is brought to you for free and open access by the Student Scholarship at University of New Hampshire Scholars' Repository. It has been accepted for inclusion in Honors Theses and Capstones by an authorized administrator of University of New Hampshire Scholars' Repository. For more information, please contact Scholarly.Communication@unh.edu.
Atovaquone Targets STAT3 in Ovarian Cancer Spheroids

Senior Honors Thesis
Kayli Neil and Sarah Walker
May 2020

University of New Hampshire, Durham, NH
College of Life Sciences and Agriculture
Department of Molecular, Cellular, and Biomedical Sciences

Walker Lab, Rudman Hall, Room 152
46 College Road, Durham, NH 03824

Contact: kneil9716@gmail.com or ken1009@wildcats.unh.edu

ABSTRACT

Ovarian cancer remains a deadly disease for countless women. Recent evidence demonstrates that ovarian cancer cell clusters, spheroids, are important in promoting ovarian recurrence and metastasis. Often these spheroids are resistant to therapy. Therefore, we were interested in identifying drugs that could target ovarian cancer spheroids. Analysis of gene expression identified the STAT3 signaling pathway as a pathway enriched in 3D growing ovarian cancer cells. STAT3 is a transcription factor that is activated by tyrosine phosphorylation in about 70% of high grade serous ovarian cancers. Using shRNA targeting STAT3 and the upstream signaling molecule, GP130, we found that reduction of both STAT3 and its upstream signaling molecule GP130 reduced the growth of spheroids. This suggested that targeting STAT3 or an upstream signaling molecule might be a viable treatment option. Atovaquone, used normally for the treatment of malaria or the prevention of pneumocystic pneumonia, inhibits STAT3 activation by inhibiting the expression of GP130 on the cell surface. Therefore, showing that atovaquone has potential in decreasing viability in ovarian cancer cells.