Adam Chong

STUDENT, BACHELOR OF SCIENCE - ENVIRONMENTAL SCIENCE MAJOR

Brusatol inhibition of Nrf2 Signaling in MDA MB-231 cells reduces resistance to paclitaxel

One of the most prevalent causes of death is cancer, as they can mutate, adapt, and evade different types of checkpoints within the cells and immune system. Because cancer can adapt to different type of treatments, we must approach cancer in another angle. Paclitaxel is a chemical therapeutic agent that interrupts the proliferation of cancer but has heavy side effects when used as a treatment option in humans. Our goal is to find a better treatment that would be less damaging to humans and living organisms, but also provide better efficacy when dealing with cancer. One of the approaches we can take is by extending the utility of paclitaxel by using Brusatol in conjunction with paclitaxel, which is a plant derived natural quassinoid showing potent tumor supressing effects. The purpose of Brusatol is to inhibit a cell signaling pathway that protects both healthy and cancer cells, inhibiting the Nuclear Factor Erythroid-2 (Nrf2) signaling pathway allows Paclitaxel to become more effective when trying to eliminate cancer cells. To examine this MDA MB-231 cells were grown in RPMI 1640 and 10% fetal bovine serum, with 5 different groups, replicated 3 times for each group, where cells were counted 24 hours, 48 hours and 72 hours using fluorescence microscopy to detect for apoptotic cells, viable cells, and early apoptotic/ necrotic cells. Variables were testing using a controlled, Cells with DMSO, paclitaxel, Brusatol and finally Brusatol with paclitaxel. The results reveal that when treated with paclitaxel, Brusatol and combined, there were significant reduction of cells compared to that of the untreated cell. The cell count analysis shows a correlation between treatment and efficacy. From this research we can conclude that Brusatol does extend the utility of paclitaxel on MDA MB-231 cancer cells.

Research Advisor: Dr. Deborah Hemmerling, Professor