Evaluating the Viability of Multiple Carcinogenic Cell Lines to Novel Chemotherapeutic TU100 with the Effects of Ascorbate on ROS Generation, a Second Year Study

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This investigation evaluates how sodium ascorbate impacts the cytotoxic effect of novel chermotherapeutic TU100 to multiple transformed/non-transformed cell lines via means of ROS production and di-ethidium bromide translation. Analysis utilized the organic fluorescent matrix of the Cell Titer Blue Viability Assay and ROS production through the di-hydro ethidium assay. It is hypothesized that TU100 will have a negative chemotoxic effect on cell viability of the transformed cell lines with enhanced cytotoxic efficacy in combination with sodium ascorbate. The most significant decrease in relative fluorescence/viability occurred between the range of drug concentration from 0uM to 40uM TU100 while the greatest increase in efficacy from ascorbate-enhanced ROS production occurred ~15uM TU100. The resulting dramatic decrease in cell proliferation supports the hypothesis that TU100 generates the ROS that break the EtBr-EtBr bond allowing singular EtBr to intercalate into DNA, a unique mechanism of cytotoxic action that inhibits transformed cells' altered metabolic pathways. Through investigation of ROS production via di-hydro ethidium, the data uniquely indicates that production of reactive oxygen species is the initial mechanism of cytotoxic action for the drug. The data suggests that sodium ascorbate is effective at producing greater ROS, thus magnifying the cytotoxic effect of the drug. Data from cell lines with/with out the p27 tumor suppressor gene indicates that TU100 has the ability to preferentially target carcinogenic cells while not damaging healthy tissue. Therefore, the results indicate that under augmentation of sodium ascorbate, TU100 represents a fundamental advancement in the progress of chemotherapeutic molecular synthesis that warrants further study.

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