PACLITAXEL–RESISTANT HUMAN BREAST DUCTAL CARCINOMA CELLS ARE V. AMYGDALINA-SENSITIVE IN VITRO

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Abstract: Cancer of the breast is the most commonly diagnosed non-skin cancer and second leading cause of cancer-related deaths in women. Breast cancer represents 15% of new cases of all cancers. An estimated 178,000 women will be diagnosed with invasive breast cancer and 40,460 women will die from the disease this year in the U.S. More than one-half of all breast are estrogen receptor-positive (ER+). Tamoxifen (TAM), an anti-estrogen drug, is one of the most effective chemotherapies for this type of breast cancer. For other types that are estrogen receptor-negative (ER-), Paclitaxel or Taxol (TAX), and anti-microtubule agent, is effective. Health disparities exist in breast cancer mortality; although the incidence of breast cancer is highest in White Women (WW), African American women (AAW) have higher mortality rate than other racial or ethnic groups in the U.S. The gap, or disparity, has even widened in recent years. One of the reported reasons for the breast cancer disparity is that AAW are more likely to be diagnosed with ER- breast cancer (more aggressive breast cancer, with less treatment options) than other ethnic groups. Therefore, there is an urgent need for the discovery and development of agent(s) efficacious against ER- breast cancer to close or eliminate the breast cancer disparity gap. *Vernonia amygdalina* (VA) is increasingly emerging as a very strong candidate. VA may be used alone or in combination (adjuvant) with known drugs. The human ductal carcinoma cells (BT549) have been reported to express little or no estrogen receptor-alpha (ER-α), and thus represent a suitable model to study estrogen-independent cell growth. The effects of VA have not been previously studied in BT549. Therefore, the objective of this study was to assess the growth inhibitory activity of VA, paclitaxel and combination in these cells. The BT549 cells were propagated in tissue culture plates with RPMI-1640 supplemented with 10% FBS and 1% penicillin-streptomycin at 37°C in a 95% air/5% CO₂ humidified incubator. Mitosis was determined by DNA synthesis assays and confirmed cell counts using a hemacytometer. Exposure of BT549 to increasing concentrations of aqueous VA abrogated cell growth in a concentration-dependent fashion: VA at concentrations of 10, 100, and 1000 µg/ml inhibited BT549 cell viability (growth) by 20%, 25% (p<0.05), and 50% (p<0.05) respectively compared to the controls. Increasing concentrations of Taxol alone and in combination with VA did not have any effect on DNA synthesis. This data suggests that these cells are not sensitive to Taxol and that ER- breast cancer cells patients may benefit from VA consumption.