

Novel Therapeutic Anticancer Property of *Vernonia amygdalina* Delile Towards the Treatment of Prostate Cancer



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Biography

Clement recently joined Florida Agricultural & Mechanical University as an Associate Professor of Biology. He received his Ph.D. in Environmental Science with concentration in molecular pharmacology and toxicology. His current research focuses on the assessment of *Vernonia calvoana* (Hook.f.) (VC-H) and *Vernonia amygdalina* Delile (VAD) as anti-cancer agent in the management of prostate cancer, and breast cancer using neoplastic cancer cells and mouse model. In vitro data demonstrated that VC-H crude extract is very effective towards the treatment of patients with breast cancer. From a mechanistic standpoint, we demonstrated that VC-H crude extract treatment reduces cellular viability; induces DNA damage leading to apoptosis accompanied by a secondary necrotic cell in tumor cells. Many of his research contributions in this field of cancer have made international news and have been featured, reported, and highlighted on: NewsRx.com, Nigerian Tribune, MDLinx.com, Pharmacy News & Articles, Mississippi Link, and Clarion-Ledger (Jackson, Mississippi Newsletter).



Prostate cancer is one of the common cancers in males and its incidence keeps increasing globally. Approximately 81% of prostate cancer is diagnosed during the early stage of the disease. The treatment options for prostate care include surgery, radiotherapy, and chemotherapy, but these treatments often have side effects that may result to poor quality of life such as impotence or decrease bowel function. Our central goal is to test the anticancer activity of *Vernonia amygdalina* Delile (an edible medicinal plant that is relatively inexpensive, nontoxic, and virtually without side effects) for the prevention of prostate cancer using human adenocarcinoma (PC-3) cells as a test model. To address our specific goal, PC-3 cells were treated with *Vernonia amygdalina* Delile (VAD). Cell viability and cell morphology was analyzed by acridine orange and propidium iodide (AO/PI) dye using the fluorescent microscope. DNA damage was evaluated by the comet assay. Cell cycle arrest and cell apoptosis was evaluated by Flow Cytometry assessment. Nucleosomal DNA fragmentation was detected by DNA ladder assay. Data obtained from the AO/PI dye assessment indicated that VAD significantly reduced the number of live cells in a dose-dependent manner, showing a gradual increase in the loss of viability in VAD-treated cells. Similar result was previously obtained by the MTT assay. We observed a significant increase in DNA damage in VAD-treated cells compared to the control group. Flow cytometry data showed that VAD induced cell cycle arrest at the G0/G1 checkpoint. Flow cytometry data also showed that VAD induced caspase-3 activation in treated cells compared to the control group. We observed the formation of the DNA ladder in gel electrophoresis by induction of apoptosis in PC-3 cells treated with VAD. These results suggest that inhibition of cancer cell growth, induction of DNA damage, cell cycle arrest at the G0/G1 checkpoint, and apoptosis through caspase-3 activation and nucleosomal DNA fragmentation are involved in the therapeutic efficacy of VAD as anticancer candidate towards the prevention and/or treatment of prostate cancer.

Publications

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