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Treatment of Metastatic and Non-Metastatic Breast Cancer with Nonionic Surfactant Vesicles Containing Doxorubicin Hydrochloride

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Introduction

Despite its numerous negative effects, Doxorubicin (Dox) is still a popular breast cancer treatment. As a result, a combination of chemotherapy is being developed in order to reduce the dose of doxorubicin in the doxorubicin regimen. The goal of this study is to see how Doxorubicin (dox) and Areca Extract (AE) work together on T47D human breast cancer cells. The MTT assay was used to determine the cytotoxic activity. To determine the effects of the combination treatment, the Combination Index (CI) was generated (synergistic, additive or antagonistic). On T47D cells, the combination of dox (6-22nM) with AE (8-30g/ml) had a synergistic (CI0.9) or additive (CI0.9-1.1) impact.

On CI0.5, the effective dox-AE combination was 6 nM - 8 g/ml. The induction of apoptosis by AE alone and in conjunction with dox was demonstrated using the double staining method. Moreover, immunocytochemistry revealed the production of Bax and caspase-3 proteins, which are involved in apoptosis. The combination of AE and Dox boosted Caspase-3 expression but did not increase Bax expression. As a result, AE improved the efficacy of doxorubicin against T47D cells.

Hormone Replacement Therapy (HRT) is a high-cost, poorly-secured treatment for oestrogen insufficiency and postmenopausal symptoms. Phytoestrogens are one type of alternative therapy. Flavones, flavonol, flavone, and polimethoxyflavone are all phytoestrogens found in banana peels. The goal of this study was to see if Banana Peel Extract (BPE) had an estrogenic effect on the development of the mammary gland in ovariectomized rats. *In vivo* and in silico experiments were used to investigate estrogenic effects. Female Sprague-dawley rats aged 50 days were ovariectomized for the in vivo experiment. At 70 days of age, 12 rats were given BPE 500 mg/kgBB and 1000 mg/kgBB, 5 rats were given estradiol 2g/day, and the rest of the rats were given CMC-Na 0.5 percent and euthanized two weeks later. At the age of 70 days, both the ovariectomized and non-ovariectomized rats were slaughtered. The in silico investigation looked at myricetin and oestrogen receptor alpha (ER-) molecular docking. The results of an in vivo investigation revealed that 1000 mg/kgBW BPE dramatically increased c-Myc expression and ovariectomized rat mammary gland development. Meanwhile, molecular docking revealed a hydrogen bond contact between the bioactive chemical in BPE and the Estrogen Receptor (ER), though it is weaker than the oestrogen and ER relationship. In conclusion, BPE can behave as an oestrogen agonist, causing c-Myc expression to increase.

Primary MEC in the hepatobiliary system is extremely rare, and the salivary glands are the most common location of MEC. As a result, the classification of salivary gland tumors is based on its degree of aggressiveness. MEC can be classified as low differentiated, moderately differentiated, or high differentiated, according to the 2017 WHO classification of head and neck cancers. The liver MEC in this case is mostly poor differentiated, which explains the patient's quick deterioration. Furthermore, in primary liver tumors, MEC can be defined as a specific form of biliary carcinoma. For clinical staging, it might relate to intrahepatic cholangio carcinoma. In this example, the tumor demonstrated vascular invasion and regional lymph node metastases, but no distant metastasis. T2N1M0 was the clinical stage.

MiR-4295 regulates FLT1 expression, since overexpression of miR-4295 in human umbilical vein endothelial cells (HUVEC) dramatically increases FLT1 protein levels. Propranolol inhibits the expression of fms-associated tyrosine kinase 1 (FLT1) through inhibiting miR-4295, a tumor suppressor. FLT1 is a VEGF/PIGF receptor with a protein binding activity. It is found on the surface of vascular endothelial cells and is involved in angiogenesis and cancer progression.

The vast accumulated information on carcinogenesis and molecular marker-status-based treatments has vastly expanded the options of cancer treatment regimens by using both prognostic and predictive molecular markers to satiate the obsessive quest for the targeted treatment, as seen from the trenches of public health. The use of molecular markers is an effective way for determining the stage and grade of breast cancer, as well as evaluating therapy responses. Indeed, prognostic and predictive molecular markers are helpful in deciding which patients should receive normal medical treatment, predicting the course of cancer, and predicting the likelihood of recurrence following treatment. Simultaneously, predictive markers indicate a specific medical treatment, allowing doctors to choose the optimal treatment for a specific form of breast cancer based on molecular marker status.

This study examines the conundrum of current technologies and principles for detecting molecular markers, therapeutic mechanism of action, and cancer development and recurrence prediction. Furthermore, despite the fact that the traditional markers, namely hormone receptors and human epidermal growth factor receptor2 (HER2), are the coveted molecular markers of therapeutic prominence in the prognosis of breast cancer, the used marker-status aids in suggesting oncologists/physicians for the selection of an appropriate treatment method, the used marker-status aids in suggesting oncologists/physicians for the selection of an appropriate treatment method. This study gives a unified narrative of therapeutically important molecular markers utilized for detection as well as the long-term benefits of breast cancer management.

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