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Delving Ks-01 as a Novel Therapeutic Strategy in Treating Breast Cancer

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Introduction

Cancer cells have an increased need for cholesterol, which is required for cell membrane integrity. Cholesterol accumulation has been described in various malignancies including breast cancer. Cholesterol has also been known to be the precursor of estrogen and vitamin D, both of which play a key role in the histology of breast cancer. Thus, depleting the cholesterol levels in cancer cells is a proposed innovative strategy to treat cancer. Therefore, novel cholesterol-depleting compounds are currently being investigated. KS-01 is a cyclic amylose oligomer composed of glucose units. It solubilizes the cholesterol and is proven to be toxicologically benign in humans. This led us to hypothesize that it might deplete cholesterol from cancer cells and may prove to be a clinically useful compound. Our work provides preliminary experimental evidences to support this hypothesis. We identified the potency of KS-01 in vitro against two breast cancer cell lines: MCF-7 (Estrogen positive, ER+), MDA-MB-231 (Estrogen negative, ER-) and compared the results against two normal Cell lines MRC-5 (Normal Human Lung Fibroblasts) and HEK-293 (Normal human embryonic kidney cells) using cytotoxic, apoptosis and cholesterol based assays. KS-01 treatment reduced intracellular cholesterol resulting in significant breast cancer cell growth inhibition through apoptosis.

The results hold true for both ER+ and ER-. These data suggest that KS-01 can prevent cholesterol accumulation in breast cancer cells and is a promising new anticancer agent. Breast cancer is cancer that develops from breast tissue Signs of breast cancer may include a lump in the breast, a change in breast shape, dimpling of the skin, fluid coming from the nipple, a newly inverted nipple, or a red or scaly patch of skin. In those with distant spread of the disease, there may be bone pain, swollen lymph nodes, shortness of breath, or yellow skin. Risk factors for developing breast cancer include obesity, a lack of physical exercise, alcoholism, hormone replacement therapy during menopause, ionizing radiation, an early age at first menstruation, having children late in life or not at all, older age, having a prior history of breast cancer, and a family history of breast cancer.

Genetics

Genetics is believed to be the primary cause of 5–10% of all cases. Women whose mother was diagnosed before 50 have an increased risk of 1.7 and those whose mothers were diagnosed at age 50 or after have an increased risk of 1.4. In those with zero, one or two affected relatives, the risk of breast cancer before the age of 80 is 7.8%, 13.3%, and 21.1% with a subsequent mortality from the disease of 2.3%, 4.2%, and 7.6% respectively. n those with a first degree relative with the disease the risk of breast cancer between the age of 40 and 50 is double that of the general population. Other genetic predispositions include the density of the breast tissue and hormonal levels. Women with dense breast tissue are more likely to get tumors and are less likely to be diagnosed with breast cancer – because the dense tissue makes tumors less visible on mammograms. Furthermore, women with naturally high estrogen and progesterone levels are also at higher risk for tumor development.

Medical conditions

The major causes of sporadic breast cancer are associated with hormone levels. Breast cancer is promoted by estrogen. This hormone activates the development of breast throughout puberty, menstrual cycles and pregnancy. The imbalance between estrogen and progesterone during the menstrual phages causes cell proliferation. Moreover, oxidative metabolites of estrogen can increase DNA damage and mutations. Repeated cycling and the impairment of repair process can transform a normal cell into pre-malignant and eventually malignant cell through mutation. During the premalignant stage, high proliferation of stromal cells can be activated by estrogen to support the development of breast cancer. During the ligand binding activation, the ER can regulate gene expression by interacting with estrogen response elements within the promoter of specific genes. The expression and activation of ER due to lack of estrogen can be stimulated by extracellular signals. Interestingly, the ER directly binding with the several proteins, including growth factor receptors, can promote the expression of genes related to cell growth and survival.

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