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Factors Involved in Genetic Alteration Leading to Generation of Cancer Cells in Humans

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Perspective

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DESCRIPTION

Breast cancer is a complex illness although the aetiology of breast cancer is poorly understood; genes and hormones seem to be important contributors. Breast epithelial cells that line the ducts or lobules of the breast undergo a sequence of molecular alterations as breast cancer first manifests as a genetic metamorphosis. Increased lifetime oestrogen exposure is considered to be a significant risk factor, with other growth factors, high levels of endogenous oestrogens appear to hasten the progression of breast cancer at numerous phases from the initial mutation to tumour metastasis. Exogenous oestrogen exposure and early life occurrences such exposure to viruses, radiation, and toxins in the environment are crucial additional pathways for the development of breast cancer.

The genetic alteration of a single cell advances depending on environmental factors from local benign hyperplasia to atypia and carcinoma *in situ*, and finally ends with an invasive tumour capable of metastasizing. Invasive or infiltrating carcinomas, in which tumour cells penetrate the breast tissue and may spread to other parts of the body, and *in situ* carcinomas, in which tumour cells remain contained to the ducts or lobules and show no indication of invasion. Indolent disease refers to cancers that can develop in the breast over an extended period of time and includes both *in situ* carcinomas and invasive tumours.

Both inherited and acquired genetic flaws may cause the molecular cell alterations that lead to breast cancer. Just 10% to 15% of breast cancers are actually genetically related, meaning they are handed down from either parent. This is significant to keep in mind. The BRCA1 and BRCA2 mutations, which are characterized by abnormalities in tumour suppressor activity, are the best documented of these inherited breast cancer syndromes. The remaining 85% to 90% are random somatic mutations, like enhanced expression of the HER2/neu and p53 genes.

Progesterone Receptors (PRs) and/or Estrogen Receptors (ERs) are expressed on the cell surfaces of up to 70% of breast tumours. Estrogen plays a role in the development of breast cancer in at least two different ways: first, by binding to estrogen receptors on cell surfaces and activating signalling pathways that promote cell growth; and second, by forming toxic metabolites that can disrupt DNA and possibly lead to the expression of oncogenes or the deactivation of tumour suppressor genes. While progesterone's function is still unclear, estrogen is known to be a powerful breast cell mitogen. Male breast cancers also benefit from estrogen, and 90% of them have ERs.

Both moderately and powerfully estrogenic metabolites can be produced by the oxidative metabolism of estrogens. The moderately estrogenic 2-hydroxyestrones (2-OHEs) may provide protection against the more potent, cancer-causing 16-hydroxyestrones (16-OHEs). Strong catechololic estrogen metabolites have the potential to produce reactive oxygen species, which in turn cause free radicals to damage DNA. In order to inform recommendations and track interventions aimed at promoting the protective 2-OHE pathway, some integrative practitioners measure urine estrogen metabolites to determine the ratio of 2-OHEs to 16-OHEs.

Estrogen levels, especially estradiol, have been reported to be elevated in breast tumours in postmenopausal women. Breast tumour oestrogens are either taken up from the bloodstream or locally synthesised from other hormones. Sulfatases and aromatases, two enzyme systems, work together to create estrogen in tumour cells. Estrone is converted to estradiol by sulphates enzymes, and androgens are converted to estrogens in fatty tissue by aromatase enzymes. Currently, a key target for chemoprevention intervention is the aromatase pathway.

By removing damaged, precancerous cells before they can grow, the immune system significantly contributes to the regulation of carcinogenesis. On the other hand, persistent immune system overstimulation has been linked to the development of cancer. According to a review, cyclooxygenase-2 (COX-2), an enzyme linked to the inflammatory process, is overexpressed in both ductal carcinomas *in situ* and invasive breast cancers. As a result of this circumstance, the breast tumor's estrogen levels have grown along with aromatase expression. Moreover, COX-2 may contribute to the development and spread of tumours. Long-term usage of Nonsteroidal Anti-Inflammatory Medicines (NSAIDs) lowers the incidence of breast cancer, according to population studies.