Introduction:

Carcinoma of the breast is one of the leading causes of cancer morbidity and mortality among women worldwide. In the United States alone, there are more than 200,000 newly diagnosed cases of invasive breast cancer and in excess of 40,000 cancer-related deaths each year. When a new diagnosis of breast cancer is made, the most immediate issues for each patient involve what the diagnosis means for her future, whether or not she will survive, and whether therapies beyond primary surgery might be of additional benefit. There has been an encouraging decline in mortality from breast cancer over the past years, which can be attributed to several factors, likely largely related to public education and screening programs that lead to the discovery of the disease at an earlier and more treatable stage. In addition, there have been several significant and important treatment advances, with **improvements in hormonal therapies**, the development of more effective combination chemotherapy regimens, and the development of biologic therapeutics such as **the targeted therapy against the human epidermal growth factor receptor 2 (*HER2*) receptor tyrosine kinase**. This evolution of therapeutic modalities for breast cancer has yielded an increasingly complex array of treatment options, both local and systemic, necessitating the development of some rational way of stratifying patients as to the most appropriate treatment regimen based on an assessment of the likelihood for disease recurrence after completion of local-regional therapy.

Generics and Family History of breast cancer: About 5% to 10% of breast cancers are related to specific inherited mutations. Women are more likely to carry a breast cancer susceptibility gene if they develop breast cancer **before** menopause, have bilateral cancer, have other associated cancers (e.g., ovarian cancer), have a significant family history (i.e., multiple relatives affected before menopause), or belong to certain ethnic groups. About half of women with hereditary breast cancer have mutations in gene *BRCA1* (on chromosome 17q21.3), and an additional one-third have mutations in *BRCA2* (on chromosome 13q12-13). Although their exact role in carcinogenesis and their relative specificity for breast cancer are still being elucidated, **both of these genes are thought to function in DNA repair**. They act as tumor suppressor genes, since cancer arises when both alleles are inactive or defective – one caused by a germ-line mutation and the second by a subsequent somatic mutation.