The work embedded in this thesis encompasses the functional elucidation of the tumor suppressor p53, potentiation of chemotherapeutic effect by combination therapy and exploration of the alternative modes of cell death in breast cancer cells. p53 is involved in distinct functions at the cellular level, namely regulation of normal cell growth and division, gene transcription, DNA repair, chemosensitivity and genomic stability. Cancer development is attributed either to p53 deletion, p53 mutation or aberrant p53 function. Breast cancer is the most common malignancy among females worldwide and among high penetrance genes, p53 was the first tumor suppressor gene linked to hereditary breast cancer. However the functional attributes of p53 in breast cancer chemotherapy are yet not clear. This thesis describes the efforts to elucidate the role of p53 in breast cancer cells through development of an isogenic model system. Also, the enhancement of therapeutic potential by combination chemotherapy and the events that occur in heterogeneous breast cancer during chemotherapy induced propagation of injury through bystander cytotoxicity have been elucidated. Hence, necessary background literature is provided on p53, breast cancer and its chemotherapy.