Targeted Therapy for Triple-Negative Breast Cancer: A Comprehensive Review of Emerging Strategies

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Description

Triple-Negative Breast Cancer (TNBC) is a very aggressive subtype of breast cancer that is distinguished by the lack of Human Epidermal Growth Factor Receptor 2 (HER2), Progesterone Receptors (PR) and Estrogen Receptors (ER). TNBC's lack of receptors renders it resistant to HER2-targeted therapies and hormone therapy, which poses a major management problem. Effective targeted therapy for TNBC is therefore desperately needed. In this paper, new approaches to targeted therapy for TNBC are reviewed with an emphasis on their mechanisms, clinical utility and future directions.

TNBC makes up 10%-20% of all breast cancer cases and is more common in African American and younger women. With a range of genetic and epigenetic changes, it is typified by considerable molecular heterogeneity. These comprise changes to pathways like PI3K/AKT/mTOR and MAPK, as well as mutations in genes like *TP53* and *BRCA1/2*. When compared to other breast cancer subtypes, TNBC has a worse prognosis and a greater recurrence rate due to its high molecular variety and lack of specific receptors, which make treatment more difficult. Inhibitors of Poly (Adp-Ribose) Polymerase (PARP) have demonstrated potential in the treatment of Triple Negative Breast Cancer (TNBC), especially in individuals with *BRCA1/2* mutations. An enzyme called PARP is essential in DNA repair; when it is inhibited, DNA damage builds up and eventually results in cancer cell death. A major development in the treatment of *BRCA*-mutated breast cancer has been the approval of PARP inhibitors such as olaparib and talazoparib. Improved Progression-Free Survival (PFS) in patients with TNBC who carry a BRCA mutation has been the result of clinical trials demonstrating their effectiveness.

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TNBC biology expands, more tailored and efficient treatments will be made available, giving individuals fighting this aggressive illness newfound hope.