Overexpression of HER2 protein and/or amplification of the gene has been reported in 10% to 20% of breast cancers. HER2 gene amplification is responsible for protein overexpression in approximately 90% of breast carcinomas. Although associated with a poor prognosis, HER2 protein overexpression and/or gene amplification are the therapeutic basis of the treatment by humanized monoclonal antibody trastuzumab. A subset of breast cancers (10% to 50%) harboring increased chromosome 17 copy number (polysomy). Although studies have indicated that the presence of polysomy 17 is correlated with tumor aggressiveness— including high histological grade, nuclear atypia, lymphovascular invasion and lymph node metastasis — and hormonal status, the clinical significance of polysomy 17 has not been completely being clear. It has been hypothesized that an increased chromosome 17 copy number without HER2 gene amplification may explain a small percentage of breast carcinomas with HER2 protein overexpression. Moreover, it has become clear that a simple increase of CEP17 copy number does not necessarily represent the presence of multiple copies of chromosome 17.