FISH Probes Breast Cancer Research

Approximately 1 in 8 women will be diagnosed with invasive breast cancer in their lifetime, making it one of the most common cancers among women. Once considered a single disease, breast cancer is now understood to include multiple molecular and histological subtypes, each defined by distinct genetic alterations. Amplifications in ERBB2 (HER2), CCND1, MYC, FGFR1, and ZNF217, as well as abnormalities in TOP2A and chromosome 17 (CEP17), are commonly observed, while emerging markers like NECTIN4 are gaining interest in aggressive subtypes such as triple-negative breast cancer. Empire Genomics' FISH probes enable detection of these key genomic changes, supporting accurate tumor characterization and breast cancer research.

Empire Genomics' breast cancer FISH panel is made up of probes that detect gene aberrations frequently found in breast cancer. For more information on how our probes have been used by researchers and clinicians around the world, and to view our extensive biomarker catalog, please visit our website.

Probe Name	Catalog Number	Test Count
ERBB2/CON17 FISH Probe	ERBB2-CHR17-20-OG-R	20
CCND1/CON11 FISH Probe	CCND1-CHR11-20-OG-R	20
NECTIN4/CON1 FISH Probe	NECTIN4-CHR01-20-OG-R	20
MYC FISH Probe	MYC-20-O-R	20
FGFR1 FISH Probe	FGFR1-20-O-R	20
TOP2A/ERBB2/CON17 FISH Probe	TOP2A-ERBB2-CHR17-20-OGA-R	20
ZNF217 FISH Probe	ZNF217-20-O-R	20

ERBB2

The ERBB2 gene, also known as HER2, encodes a receptor tyrosine kinase involved in cell proliferation and survival. Amplification or over expression of ERBB2 occurs in approximately 15–20% of breast cancers and is associated with more aggressive disease, higher recurrence rates, and distinct molecular subtypes. ERBB2 status is routinely assessed by FISH in pathology labs as a key biomarker for tumor characterization and clinical stratification.[1]

CCND1

CCND1 encodes cyclin D1, a protein critical for regulating the G1/S transition in the cell cycle. Amplification of CCND1 occurs in roughly 15% of breast cancers and is especially common in ER-positive and luminal B subtypes. Overexpression of CCND1 can be associated with increased proliferation, resistance to endocrine therapy, and poor prognosis in certain patient populations.[2,3]

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MYC

The MYC oncogene, located at 8q24, is a transcription factor that controls genes involved in cell growth, metabolism, and apoptosis. Amplification of MYC is found in 15–40% of breast cancers and has been correlated with higher tumor grade, increased proliferation, and reduced survival. Its activation contributes to aggressive tumor behavior and may co-occur with other oncogenic events[4]

NECTIN4

NECTIN4 (PVRL4) encodes a cell adhesion molecule overexpressed in several epithelial cancers, including triple-negative breast cancer (TNBC). Its gene is amplified or highly expressed in certain breast tumors, particularly those with aggressive or basal-like phenotypes. As a surface molecule, NECTIN4 has become a key biomarker in breast cancer research for tumor characterization.[5]

FGFR1

FGFR1 (fibroblast growth factor receptor 1), located on 8p11, is amplified in approximately 10% of breast cancers, particularly in estrogen receptor-positive (ER+) tumors. Its amplification leads to activation of mitogenic and anti-apoptotic signaling pathways and has been implicated in endocrine therapy resistance. FGFR1 gain is associated with poor prognosis and may indicate a more proliferative phenotype.[6]

ZNF217

ZNF217 is a zinc finger protein gene located on 20q13, a region frequently amplified in breast cancer. Its amplification has been associated with poor prognosis, tumor progression, and genomic instability. ZNF217 may promote oncogenesis by interfering with apoptosis, promoting immortalization, and deregulating transcription.[7]

References: **1.** Nitta H, et al., Pathol Int. 2016;66(6):313-324. **2.** Jeffreys SA, et al., Front Endocrinol (Lausanne). 2022 Jun 17;13:895729. **3.** Valla M, et al., J Mammary Gland Biol Neoplasia. 2022 Mar;27(1):67-77. **4.** Green, A., Aleskandarany, M., Agarwal, D. et al. Br J Cancer 114, 917–928 (2016) **5.** Challita-Eid PM, Satpayev D, Yang P, et al. Cancer Res. 2016;76(10):3003–3013. **6.** Turner N, Pearson A, Sharpe R, et al. Cancer Res. 2010;70(5):2085–2094. **7.** Collins C, Rommens JM, Kowbel D, et al. Proc Natl Acad Sci U S A. 1998;95(15):8703–8708.

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