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The epidermal growth factor receptor (EGFR) is the founding member of the ErbB family of four structurally related receptor tyrosine kinases. In humans this includes Her1 (EGFR, ErbB1), Her2 (Neu, ErbB2), Her3 (ErbB3), and Her4 (ErbB4).

ErbB-1, also named epidermal growth factor receptor (EGFR). Mutations that lead to EGFR overexpression (known as upregulation) or overactivity have been associated with a number of cancers, including lung cancer, anal cancers and glioblastoma multiforme.

ErbB-2, also named HER2 in humans and neu in rodents. Amplification or over-expression of this gene has been shown to play an important role in the pathogenesis and progression of certain aggressive types of breast cancer and in recent years it has evolved to become an important biomarker and target of therapy for the disease.

ErbB-3, also named HER3. Amplification of this gene and/or overexpression of its protein have been reported in numerous cancers, including prostate, bladder, and breast tumors.

ErbB-4, also named HER4

The gene symbol, ErbB, is derived from the name of a viral oncogene to which these receptors are homologous: Erythroblastic Leukemia Viral Oncogene. Insufficient ErbB signaling in humans is associated with the development of neurodegenerative diseases , such as multiple sclerosis and Alzheimer's Disease. Excessive ErbB signaling is associated with the development of a wide variety of types of solid tumor. ErbB-1 and ErbB-2 are found in many human cancers , and their excessive signaling may be critical factors in the development and malignancy of these tumors.

ErbB-1 is overexpressed in many cancers. Drugs such as panitumumab , cetuximab , gefitinib , erlotinib are used to inhibit it. It has recently been shown that acquired resistance to cetuximab and gefitinib can be linked to hyperactivity of ErbB-3.

ErbB-2 (HER-2) is often overexpressed in breast cancer. The drug Trastuzumab (Herceptin) targets this receptor.