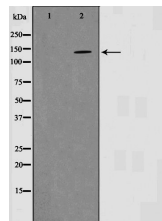




#24269

**Catalog Number:** 24269-1, 24269-2**Amount:** 50µg/50µl, 100µg/100µl**Swiss-Prot No. :** P00533**Form of Antibody:** Rabbit IgG in phosphate buffered saline (without Mg<sup>2+</sup> and Ca<sup>2+</sup>), pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol.**Storage/Stability:** Store at -20°C/1 year**Immunogen:** The antiserum was produced against synthesized peptide derived from Human EGFR**Purification:** The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.**Specificity/Sensitivity:** EGFR Antibody detects endogenous levels of total EGFR**Reactivity:** Human, Mouse, Rat**Applications:** Predicted MW: 135, 170kd WB: 1:500-2000

Western blot analysis on SK-OV3 cell lysate using EGFR Antibody

**Background :** EGFR is a receptor tyrosine kinase. Receptor for epidermal growth factor (EGF) and related growth factors including TGF- $\alpha$ , amphiregulin, betacellulin, heparin-binding EGF-like growth factor, GP30 and vaccinia virus growth factor. Is involved in the control of cell growth and differentiation. . A single-pass transmembrane tyrosine kinase. Ligand binding to this receptor results in receptor dimerization, autophosphorylation (in trans), activation of various downstream signaling molecules and lysosomal degradation. Can be phosphorylated and activated by Src. Activated EGFR binds the SH2 domain of phospholipase C- $\gamma$  (PLC- $\gamma$ ), activating PLC- $\gamma$ -mediated downstream signaling. Phosphorylated EGFR binds Cbl, leading to its ubiquitination and degradation. Grb2 and SHC bind to phospho-EGFR and are involved in the activation of MAP kinase signaling pathways. Phosphorylation on Ser and Thr residues is thought to represent a mechanism for attenuation of EGFR kinase activity. Overexpressed in breast, head and neck cancers, correlating with poor survival. Activating somatic mutations seen in lung cancer, corresponding to minority of patients with strong response to EGFR inhibitor Iressa (gefitinib). Mutations and amplification also seen in glioblastoma, and upregulation seen in colon cancer and neoplasms. In xenografts, inhibitors synergized with cytotoxic drugs in inhibition of many tumor types.