



Figure 1. Activation of the EGFR signaling pathway upon EGF binding. Binding of EGF to EGFR results in EGFR dimerization and autophosphorylation (EGFR phosphorylates itself) at various sites on the receptor's intracellular domain. Depending on the specific sites phosphorylated, the downstream effects will be different. Autophosphorylation of specific tyrosine residues results in the activation of the MAPK (MEK/ERK) signaling pathway through the phosphorylation of its downstream components, leading to increased expression of genes that promote cell proliferation and growth. Autophosphorylation of certain tyrosine and serine residues leads to the ubiquitination and internalization of EGFR, followed by either recycling of the receptor back to the cell membrane or lysosome-mediated degradation. EGFR can bind to other ligands besides EGF, and the fate of EGFR after internalization depends on the ligand that induces EGFR signaling, the concentration of ligand present, and the length of time EGFR is stimulated. Internalization and degradation decrease the number of activated receptors on the cell membrane at a given point and attenuate signaling. Several details about EGFR signaling, such as the other pathways, for example the AKT/PKB and STAT5b pathways, which are activated in the presence of EGF, have been omitted in this figure for simplicity.