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Brk (phospho Tyr447) Polyclonal Antibody

Catalog # AP67469

Product Information

Application	WB, IF, ICC, E
Primary Accession	Q13882
Reactivity	Human, Rat, Mouse
Host	Rabbit
Clonality	Polyclonal
Calculated MW	51834

Additional Information

Gene ID	5753
Other Names	PTK6; BRK; Protein-tyrosine kinase 6; Breast tumor kinase; Tyrosine-protein kinase BRK
Dilution	WB~~Western Blot: 1/500 - 1/2000. Immunofluorescence: 1/200 - 1/1000. ELISA: 1/5000. Not yet tested in other applications. IF~~1:50~200 ICC~~N/A E~~N/A
Format	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.
Storage Conditions	-20℃

Protein Information

Name	PTK6
Synonyms	BRK
Function	Non-receptor tyrosine-protein kinase implicated in the regulation of a variety of signaling pathways that control the differentiation and maintenance of normal epithelia, as well as tumor growth. Function seems to be context dependent and differ depending on cell type, as well as its intracellular localization. A number of potential nuclear and cytoplasmic substrates have been identified. These include the RNA-binding proteins: KHDRBS1/SAM68, KHDRBS2/SLM1, KHDRBS3/SLM2 and SFPQ/PSF; transcription factors: STAT3 and STAT5A/B and a variety of signaling molecules: ARHGAP35/p190RhoGAP, PXN/paxillin, BTK/ATK, STAP2/BKS. Phosphorylates the GTPase-activating protein ARAP1 following EGF stimulation which enhances EGFR signaling by delaying EGFR down-regulation (PubMed:20554524). Also associates with a variety of proteins that are likely upstream of PTK6 in various signaling pathways, or for which PTK6 may play an adapter-like role. These proteins include ADAM15, EGFR, ERBB2, ERBB3 and IRS4. In normal or

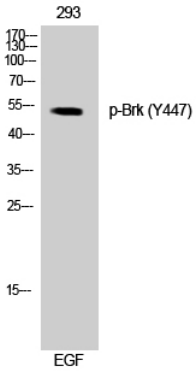
	non-tumorigenic tissues, PTK6 promotes cellular differentiation and apoptosis. In tumors PTK6 contributes to cancer progression by sensitizing cells to mitogenic signals and enhancing proliferation, anchorage-independent survival and migration/invasion. Association with EGFR, ERBB2, ERBB3 may contribute to mammary tumor development and growth through enhancement of EGF-induced signaling via BTK/AKT and PI3 kinase. Contributes to migration and proliferation by contributing to EGF-mediated phosphorylation of ARHGAP35/p190RhoGAP, which promotes association with RASA1/p120RasGAP, inactivating RhoA while activating RAS. EGF stimulation resulted in phosphorylation of PNX/Paxillin by PTK6 and activation of RAC1 via CRK/CrKII, thereby promoting migration and invasion. PTK6 activates STAT3 and STAT5B to promote proliferation. Nuclear PTK6 may be important for regulating growth in normal epithelia, while cytoplasmic PTK6 might activate oncogenic signaling pathways.
Cellular Location	Cytoplasm. Nucleus. Cell projection, ruffle. Membrane. Note=Colocalizes with KHDRBS1, KHDRBS2 or KHDRBS3, within the nucleus. Nuclear localization in epithelial cells of normal prostate but cytoplasmic localization in cancer prostate
Tissue Location	Epithelia-specific. Very high level in colon and high levels in small intestine and prostate, and low levels in some fetal tissues. Not expressed in breast or ovarian tissue but expressed in high percentage of breast and ovarian cancers. Also overexpressed in some metastatic melanomas, lymphomas, colon cancers, squamous cell carcinomas and prostate cancers. Also found in melanocytes. Not expressed in heart, brain, placenta, lung, liver, skeletal muscle, kidney and pancreas. Isoform 2 is present in prostate epithelial cell lines derived from normal prostate and prostate adenocarcinomas, as well as in a variety of cell lines.

Background

Non-receptor tyrosine-protein kinase implicated in the regulation of a variety of signaling pathways that control the differentiation and maintenance of normal epithelia, as well as tumor growth. Function seems to be context dependent and differ depending on cell type, as well as its intracellular localization. A number of potential nuclear and cytoplasmic substrates have been identified. These include the RNA-binding proteins: KHDRBS1/SAM68, KHDRBS2/SLM1, KHDRBS3/SLM2 and SFPQ/PSF; transcription factors: STAT3 and STAT5A/B and a variety of signaling molecules: ARHGAP35/p190RhoGAP, PXN/paxillin, BTK/ATK, STAP2/BKS. Associates also with a variety of proteins that are likely upstream of PTK6 in various signaling pathways, or for which PTK6 may play an adapter-like role. These proteins include ADAM15, EGFR, ERBB2, ERBB3 and IRS4. In normal or non-tumorigenic tissues, PTK6 promotes cellular differentiation and apoptosis. In tumors PTK6 contributes to cancer progression by sensitizing cells to mitogenic signals and enhancing proliferation, anchorage- independent survival and migration/invasion. Association with EGFR, ERBB2, ERBB3 may contribute to mammary tumor development and growth through enhancement of EGF-induced signaling via BTK/AKT and PI3 kinase. Contributes to migration and proliferation by contributing to EGF-mediated phosphorylation of ARHGAP35/p190RhoGAP, which promotes association with RASA1/p120RasGAP, inactivating RhoA while activating RAS. EGF stimulation resulted in phosphorylation of PNX/Paxillin by PTK6 and activation of RAC1 via CRK/CrKII, thereby promoting migration and invasion. PTK6 activates STAT3 and STAT5B to promote proliferation. Nuclear PTK6 may be important for regulating growth in normal epithelia, while cytoplasmic PTK6 might activate oncogenic signaling pathways.

Images

Western Blot analysis of 293 cells using Phospho-Brk (Y447) Polyclonal Antibody



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