Revision 1

Store at -20C

#12651

PDGF Receptor Activation Antibody Sampler Kit

1 Kit (8 x 20 microliters)

Cell Signaling TECHNOLOGY®

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For Research Use Only. Not for Use in Diagnostic Procedures.

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Phospho-PDGF Receptor β (Tyr751) (C63G6) Rabbit mAb	4549	20 µl	190 kDa	Rabbit IgG
PDGF Receptor β (28E1) Rabbit mAb	3169	20 µl	190 kDa	Rabbit IgG
Phospho-SHP-2 (Tyr542) Antibody	3751	20 µl	72 kDa	Rabbit
SHP-2 (D50F2) Rabbit mAb	3397	20 µl	72 kDa	Rabbit IgG
Phospho-Akt (Ser473) (D9E) XP® Rabbit mAb	4060	20 µl	60 kDa	Rabbit IgG
Akt (pan) (C67E7) Rabbit mAb	4691	20 µl	60 kDa	Rabbit IgG
Phospho-p44/42 MAPK (Erk1/2) (Thr202/Tyr204) (D13.14.4E) XP® Rabbit mAb	4370	20 µl	44, 42 kDa	Rabbit IgG
p44/42 MAPK (Erk1/2) (137F5) Rabbit mAb	4695	20 µl	42, 44 kDa	Rabbit IgG
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

Description

The PDGF Receptor Activation Antibody Sampler Kit provides an economical means to evaluate the activation status of multiple members of the PDGF receptor pathway, including SHP-2, Akt, and p44/42 MAPK (Erk1/2). The kit includes enough antibody to perform two western blot experiments per primary antibody.

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.

Background

Platelet derived growth factor (PDGF) family proteins form dimers (PDGF AA, PDGF AB, PDGF BB, PDGF CC, and PDGF DD) that bind receptor tyrosine kinases PDGF receptor α (PDGFRα) and PDGF receptor β (PDGFRβ) in a specific pattern. PDGFRβ homodimers bind PDGF BB and DD homodimers and the PDGF AB heterodimer. Heteromeric receptor PDGF α/β binds PDGF B, C, and D homodimers and the PDGF AB heterodimer (1). Ligand binding induces PDGF receptor dimerization and autophosphorylation, followed by binding and activation of cytoplasmic SH2 domain-containing signal transduction molecules, such as GRB2, Src, GAP, PI3 kinase, PLCγ, and NCK. Activated PDGF receptors initiate signaling pathways that control cell growth, actin reorganization, migration, and differentiation (2). PDGFRβ kinase-insert region residue Tyr751 forms the PI3 kinase docking site, and phosphorylation of PDGFRβ at this site inhibits the association between the SH2 domain of the PI3 kinase p85 subunit and PDGFRβ (3,4).

SHP-2 (PTPN11) is a nonreceptor protein tyrosine phosphatase that participates in signaling pathways that control cell growth, differentiation, migration, and death (5). Activation of SHP-2 and its association with Gab1 is critical for sustained Erk activation downstream of growth factor receptors and cytokines (6). Phosphorylation of SHP-2 at Tyr542 and Tyr580 in response to growth factor receptor activation is thought to relieve basal inhibition and stimulate SHP-2 tyrosine phosphatase activity (7,8).

Insulin and various growth/survival factors activate Akt, a kinase that acts in a wortmannin-sensitive pathway involving PI3 kinase to help control survival and apoptosis (9-11). Akt is activated by phospholipid binding and activation loop phosphorylation at Thr308 by PDK1 (12) and by phosphorylation within the carboxy terminus at Ser473.

The p44/42 MAPK (Erk1/2) signaling pathway is activated in response to extracellular stimuli including mitogens, growth factors, and cytokines (13-15). Research suggests that this pathway is an important target in cancer diagnosis and treatment (16). External stimuli lead to activation of a kinase cascade that results in the activation of p44 and p42 by a MAP kinase. MEK1 and MEK2 activate p44 and p42 through phosphorylation of activation loop residues Thr202/Tyr204 and Thr185/Tyr187, respectively.

Clinical studies describe PDGF expression in a number of different solid tumors, from glioblastomas to prostate carcinomas. The biological role of PDGF signaling in these tumors varies from autocrine stimulation of cancer cell growth to more subtle paracrine interactions involving adjacent stroma and even angiogenesis. Targeting PDGF signaling may be an effective way for tumor treatment (17).

Background References

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