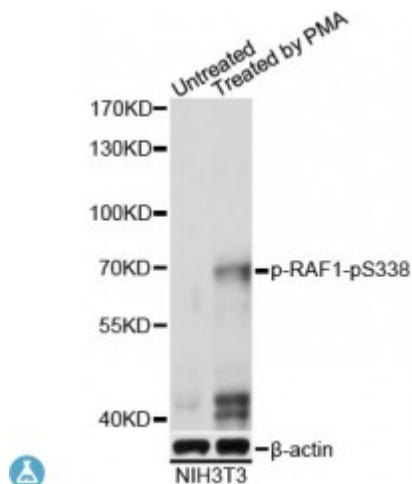


Anti-Phospho-RAF1-(S338) Antibody



Description

This gene is the cellular homolog of viral raf gene (v-raf). The encoded protein is a MAP kinase kinase kinase (MAP3K), which functions downstream of the Ras family of membrane associated GTPases to which it binds directly. Once activated, the cellular RAF1 protein can phosphorylate to activate the dual specificity protein kinases MEK1 and MEK2, which in turn phosphorylate to activate the serine/threonine specific protein kinases, ERK1 and ERK2. Activated ERKs are pleiotropic effectors of cell physiology and play an important role in the control of gene expression involved in the cell division cycle, apoptosis, cell differentiation and cell migration. Mutations in this gene are associated with Noonan syndrome 5 and LEOPARD syndrome 2.

Model	STJ117871
Host	Rabbit
Reactivity	Human, Mouse, Rat
Applications	WB
Immunogen	A phospho specific peptide corresponding to residues surrounding pS338 of human RAF1
Gene ID	5894
Gene Symbol	RAF1
Dilution range	WB 1:500 - 1:2000
Tissue Specificity	In skeletal muscle, isoform 1 is more abundant than isoform 2
Purification	Affinity purification
Note	For Research Use Only (RUO).

Protein Name	RAF proto-oncogene serine/threonine-protein kinase
Molecular Weight	73.052 kDa
Clonality	Polyclonal
Conjugation	Unconjugated
Isotype	IgG
Formulation	PBS with 0.02% sodium azide, 50% glycerol, pH7.3.
Storage Instruction	Store at -20C. Avoid freeze / thaw cycles.
Database Links	HGNC:9829OMIM:164760Reactome:R-HSA-2672351
Alternative Names	RAF proto-oncogene serine/threonine-protein kinase
Function	Serine/threonine-protein kinase that acts as a regulatory link between the membrane-associated Ras GTPases and the MAPK/ERK cascade, and this critical regulatory link functions as a switch determining cell fate decisions including proliferation, differentiation, apoptosis, survival and oncogenic transformation, RAF1 activation initiates a mitogen-activated protein kinase (MAPK) cascade that comprises a sequential phosphorylation of the dual-specific MAPK kinases (MAP2K1/MEK1 and MAP2K2/MEK2) and the extracellular signal-regulated kinases (MAPK3/ERK1 and MAPK1/ERK2), The phosphorylated form of RAF1 (on residues Ser-338 and Ser-339, by PAK1) phosphorylates BAD/Bcl2-antagonist of cell death at 'Ser-75', Phosphorylates adenylyl cyclases: ADCY2, ADCY5 and ADCY6, resulting in their activation, Phosphorylates PPP1R12A resulting in inhibition of the phosphatase activity, Phosphorylates TNNT2/cardiac muscle troponin T, Can promote NF-kB activation and inhibit signal transducers involved in motility (ROCK2), apoptosis (MAP3K5/ASK1 and STK3/MST2), proliferation and angiogenesis (RB1), Can protect cells from apoptosis also by translocating to the mitochondria where it binds BCL2 and displaces BAD/Bcl2-antagonist of cell death, Regulates Rho signaling and migration, and is required for normal wound healing, Plays a role in the oncogenic transformation of epithelial cells via repression of the TJ protein, occludin (OCLN) by inducing the up-regulation of a transcriptional repressor SNAI2/SLUG, which induces down-regulation of OCLN, Restricts caspase activation in response to selected stimuli, notably Fas stimulation, pathogen-mediated macrophage apoptosis, and erythroid differentiation,
Cellular Localization	Cytoplasm, Cell membrane, Mitochondrion, Nucleus,
Post-translational Modifications	Phosphorylation at Thr-269, Ser-338, Tyr-341, Thr-491 and Ser-494 results in its activation, Phosphorylation at Ser-29, Ser-43, Ser-289, Ser-296, Ser-301 and Ser-642 by MAPK1/ERK2 results in its inactivation, Phosphorylation at Ser-259 induces the interaction with YWHAZ and inactivates kinase activity, Dephosphorylation of Ser-259 by the complex containing protein phosphatase 1, SHOC2 and M-Ras/MRAS relieves inactivation, leading to stimulate RAF1 activity, Phosphorylation at Ser-338 by PAK1 and PAK5 and Ser-339 by PAK1 is required for its mitochondrial localization, Phosphorylation at Ser-621 in response to growth factor treatment stabilizes the protein, possibly by preventing proteasomal degradation, Phosphorylation at Ser-289, Ser-296, Ser-301, Ser-338 and Ser-621 are somehow linked to the methylation potential of cells, Treatment of cells with HGF in the presence of the methylation inhibitor 5'-methylthioadenosine (MTA) results in increased phosphorylation

at Ser-338 and Ser-621 and decreased phosphorylation at Ser-296, Ser-301 and Ser-338, Dephosphorylation at Ser-338 by PPP5C results in a activity decrease,

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