

Product datasheet

Anti-SHP2 antibody ab17753

★★★★☆ 1 Abreviews

Overview

Product name	Anti-SHP2 antibody
Description	Rabbit polyclonal to SHP2
Tested applications	Suitable for: WB, IP, ICC, Neutralising
Species reactivity	Reacts with: Mouse, Rat, Human
Immunogen	Fusion protein, corresponding to amino acids 2-216 of Mouse SHP2.
Positive control	Non stimulated A431 cell lysate.

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C or -80°C. Avoid freeze / thaw cycle.
Storage buffer	Preservative: 0.05% Sodium Azide Constituents: 0.1M Tris glycine. pH 7.0.
Purity	Protein A purified
Purification notes	Purified by affinity chromatography.
Clonality	Polyclonal
Isotype	IgG

Applications

Our [Abpromise guarantee](#) covers the use of **ab17753** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
WB	★★★★☆	Use a concentration of 1 - 4 µg/ml. Detects a band of approximately 64 kDa. Apparent size increases to approximately 70kDa upon tyrosine phosphorylation.
IP		Use at an assay dependent dilution.

Application	Abreviews	Notes
ICC		Use a concentration of 10 µg/ml.
Neutralising		Use at an assay dependent dilution. Does not inhibit phosphotyrosine phosphatase activity.

Target

Function	Acts downstream of various receptor and cytoplasmic protein tyrosine kinases to participate in the signal transduction from the cell surface to the nucleus.
Tissue specificity	Widely expressed, with highest levels in heart, brain, and skeletal muscle.
Involvement in disease	<p>Defects in PTPN11 are the cause of LEOPARD syndrome type 1 (LEOPARD1) [MIM:151100]. It is an autosomal dominant disorder allelic with Noonan syndrome. The acronym LEOPARD stands for lentiginos, electrocardiographic conduction abnormalities, ocular hypertelorism, pulmonic stenosis, abnormalities of genitalia, retardation of growth, and deafness.</p> <p>Defects in PTPN11 are the cause of Noonan syndrome type 1 (NS1) [MIM:163950]. Noonan syndrome (NS) is a disorder characterized by dysmorphic facial features, short stature, hypertelorism, cardiac anomalies, deafness, motor delay, and a bleeding diathesis. Some patients with Noonan syndrome type 1 develop multiple giant cell lesions of the jaw or other bony or soft tissues, which are classified as pigmented villomoduolar synovitis (PVNS) when occurring in the jaw or joints. Note=Mutations in PTPN11 account for more than 50% of the cases. Rarely, NS is associated with juvenile myelomonocytic leukemia (JMML). NS1 inheritance is autosomal dominant.</p> <p>Defects in PTPN11 are a cause of juvenile myelomonocytic leukemia (JMML) [MIM:607785]. JMML is a pediatric myelodysplastic syndrome that constitutes approximately 30% of childhood cases of myelodysplastic syndrome (MDS) and 2% of leukemia. It is characterized by leukocytosis with tissue infiltration and in vitro hypersensitivity of myeloid progenitors to granulocyte-macrophage colony stimulating factor.</p> <p>Defects in PTPN11 are a cause of metachondromatosis (MC) [MIM:156250]. It is a skeletal disorder with radiologic fetarures of both multiple exostoses and Ollier disease, characterized by the presence of multiple enchondromas and osteochondroma-like lesions.</p>
Sequence similarities	<p>Belongs to the protein-tyrosine phosphatase family. Non-receptor class 2 subfamily.</p> <p>Contains 2 SH2 domains.</p> <p>Contains 1 tyrosine-protein phosphatase domain.</p>
Domain	The SH2 domains repress phosphatase activity. Binding of these domains to phosphotyrosine-containing proteins relieves this auto-inhibition, possibly by inducing a conformational change in the enzyme.
Post-translational modifications	Phosphorylated on Tyr-546 and Tyr-584 upon receptor protein tyrosine kinase activation; which creates a binding site for GRB2 and other SH2-containing proteins.
Cellular localization	Cytoplasm.

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