

Mouse Syk Antibody (Center)

Affinity Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP16152c

Specification

Mouse Syk Antibody (Center) - Product Information

Application WB,E
Primary Accession P48025

Other Accession <u>Q64725</u>, <u>F1N9Y5</u>, <u>NP_035648.2</u>

Reactivity
Predicted
Chicken, Rat
Host
Clonality
Polyclonal
Isotype
Calculated MW
Antigen Region

Human
Chicken, Rat
Rabbit
Rabbit
Polyclonal
Rabbit IgG
71376
195-223

Mouse Syk Antibody (Center) - Additional Information

Gene ID 20963

Other Names

Tyrosine-protein kinase SYK, Spleen tyrosine kinase, Syk, ptk72, Sykb

Target/Specificity

This Mouse Syk antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 195-223 amino acids from the Central region of mouse Syk.

Dilution

WB~~1:1000

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

Mouse Syk Antibody (Center) is for research use only and not for use in diagnostic or therapeutic procedures.

Mouse Syk Antibody (Center) - Protein Information

Name Syk

Synonyms ptk72, Sykb



Function Non-receptor tyrosine kinase which mediates signal transduction downstream of a variety of transmembrane receptors including classical immunoreceptors like the B-cell receptor (BCR). Regulates several biological processes including innate and adaptive immunity, cell adhesion, osteoclast maturation, platelet activation and vascular development (PubMed: 33782605). Assembles into signaling complexes with activated receptors at the plasma membrane via interaction between its SH2 domains and the receptor tyrosine- phosphorylated ITAM domains. The association with the receptor can also be indirect and mediated by adapter proteins containing ITAM or partial hemITAM domains. The phosphorylation of the ITAM domains is generally mediated by SRC subfamily kinases upon engagement of the receptor. More rarely signal transduction via SYK could be ITAM-independent. Direct downstream effectors phosphorylated by SYK include DEPTOR, VAV1, PLCG1, PI-3-kinase, LCP2 and BLNK. Initially identified as essential in B-cell receptor (BCR) signaling, it is necessary for the maturation of B-cells most probably at the pro-B to pre-B transition. Activated upon BCR engagement, it phosphorylates and activates BLNK an adapter linking the activated BCR to downstream signaling adapters and effectors. It also phosphorylates and activates PLCG1 and the PKC signaling pathway. It also phosphorylates BTK and regulates its activity in B-cell antigen receptor (BCR)-coupled signaling. In addition to its function downstream of BCR also plays a role in T-cell receptor signaling. Plays also a crucial role in the innate immune response to fungal, bacterial and viral pathogens. It is for instance activated by the membrane lectin CLEC7A. Upon stimulation by fungal proteins, CLEC7A together with SYK activates immune cells inducing the production of ROS. Also activates the inflammasome and NF-kappa-B-mediated transcription of chemokines and cytokines in presence of pathogens. Regulates neutrophil degranulation and phagocytosis through activation of the MAPK signaling cascade. Required for the stimulation of neutrophil phagocytosis by IL15 (By similarity). Also mediates the activation of dendritic cells by cell necrosis stimuli. Also involved in mast cells activation. Involved in interleukin-3/IL3-mediated signaling pathway in basophils (PubMed: 19098920). Also functions downstream of receptors mediating cell adhesion. Relays for instance, integrin-mediated neutrophils and macrophages activation and P-selectin receptor/SELPG-mediated recruitment of leukocytes to inflammatory loci. Also plays a role in non-immune processes. It is for instance involved in vascular development where it may regulate blood and lymphatic vascular separation. It is also required for osteoclast development and function. Functions in the activation of platelets by collagen, mediating PLCG2 phosphorylation and activation. May be coupled to the collagen receptor by the ITAM domain-containing FCER1G. Also activated by the membrane lectin CLEC1B that is required for activation of platelets by PDPN/podoplanin. Involved in platelet adhesion being activated by ITGB3 engaged by fibringen. Together with CEACAM20, enhances production of the cytokine CXCL8/IL-8 via the NFKB pathway and may thus have a role in the intestinal immune response (PubMed:26195794).

Cellular Location

Cell membrane. Cytoplasm, cytosol. Cytoplasmic vesicle, phagosome

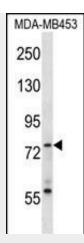
Mouse Syk Antibody (Center) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

Mouse Syk Antibody (Center) - Images





Mouse Syk Antibody (Center) (Cat. #AP16152c) western blot analysis in MDA-MB453 cell line lysates (35ug/lane). This demonstrates the Syk antibody detected the Syk protein (arrow).

Mouse Syk Antibody (Center) - Background

Positive effector of BCR-stimulated responses. Couples the B-cell antigen receptor (BCR) to the mobilization of calcium ion either through a phosphoinositide 3-kinase-dependent pathway, when not phosphorylated on tyrosines of the linker region, or through a phospholipase C-gamma-dependent pathway, when phosphorylated on Tyr-342 and Tyr-346. Thus the differential phosphorylation of Syk can determine the pathway by which BCR is coupled to the regulation of intracellular calcium ion. Phosphorylates USP25 and regulates its intracellular levels (By similarity).

Mouse Syk Antibody (Center) - References

Heizmann, B., et al. Proc. Natl. Acad. Sci. U.S.A. 107(43):18563-18568(2010) Fernandez, P.L., et al. J. Biol. Chem. 285(43):32844-32851(2010) Zou, W., et al. J. Cell. Sci. 123 (PT 17), 2955-2963 (2010) : Falet, H., et al. J. Exp. Med. 207(9):1967-1979(2010) Dierks, C., et al. Cancer Res. 70(15):6193-6204(2010)