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PRODUCT INFORMATION



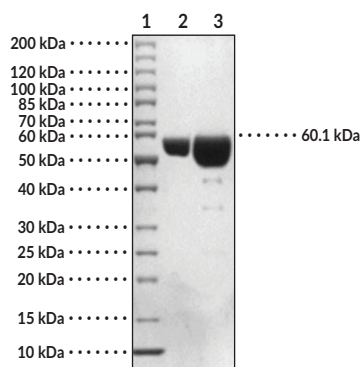
SHP-2 (human, recombinant)

Item No. 42002

Overview and Properties

Synonyms:	Protein Tyrosine Phosphatase 1D, Protein Tyrosine Phosphatase 2C, PTP-1D, PTP-2C, PTPN11, SH-PTP2, SH-PTP3, Src Homology Region 2 Domain-containing Phosphatase-2, Tyrosine-protein Phosphatase Non-Receptor Type 11
Source:	Recombinant human SHP-2 expressed in <i>E. coli</i>
Amino Acids:	1-525
Uniprot No.:	Q06124
Molecular Weight:	60.1 kDa
Storage:	-80°C (as supplied); avoid repetitive freeze/thaw cycles
Stability:	≥1 year
Supplied in:	20 mM Tris-HCl, pH 8.5, with 3 mM TCEP and 150 mM sodium chloride
Protein Concentration:	<i>batch specific</i> mg/ml

Images



Lane 1: MW Markers

Lane 2: SHP-2 (2 µg, reduced)

Lane 3: SHP-2 (10 µg, non-reduced)

SDS-PAGE Analysis of SHP-2. This protein has a calculated molecular weight of 60.1 kDa.

WARNING
THIS PRODUCT IS FOR RESEARCH ONLY - NOT FOR HUMAN OR VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

SAFETY DATA
This material should be considered hazardous until further information becomes available. Do not ingest, inhale, get in eyes, on skin, or on clothing. Wash thoroughly after handling. Before use, the user must review the [complete](#) Safety Data Sheet, which has been sent via email to your institution.

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PRODUCT INFORMATION



Description

Src homology region 2 domain-containing phosphatase-2 (SHP-2) is a non-receptor protein tyrosine phosphatase (PTP) and a member of the PTP family.¹ It is composed of an N-terminal Src homology region 2 (SH2) domain, a second SH2 domain, a PTP domain, a disordered C-terminal tail, which can serve as a binding site for adaptor proteins when phosphorylated, and a proline-rich sequence.² Under basal conditions, the N-terminal SH2 domain binds to and inhibits the PTP domain, but disassociates in the presence of phosphorylated tyrosine residues on activated receptor tyrosine kinases or adaptor proteins to which it can also bind.^{2,3} SHP-2 is ubiquitously expressed and is involved in both positive and negative regulation of growth factor-, cytokine-, interferon-, or insulin-induced signaling pathways by dephosphorylating receptors, signaling intermediates, or kinases.¹ SHP-2 binds phosphorylated programmed cell death protein 1 (PD-1) and decreases the levels of CD69 on the surface of, and IL-2 secretion from, T cell receptor-activated Jurkat T cells.⁴ Conditional knockout of *Ptpn11*, the gene expressing SHP-2, in myeloid cells, but not in mature T cells, reduces tumor volume, increases the tumor draining lymph node levels of effector and central memory T cells, and decreases the splenic levels of myeloid-derived suppressor cells (MDSCs), in a B16/F10 melanoma mouse xenograft model.⁵ Germline and somatic mutations in *PTPN11* have been found in patients with both juvenile myelomonocytic leukemia (JMML) and Noonan syndrome, an autosomal developmental disorder characterized by facial abnormalities, decreased height, cardiac defects, such as pulmonary valve stenosis or hypertrophic cardiomyopathy, and skeletal malformation, and in a smaller percentage of patients with myelodysplastic syndrome (MDS) or acute myeloid leukemia (AML).³ Cayman's SHP-2 (human, recombinant) protein consists of 525 amino acids and has a calculated molecular weight of 60.1 kDa.

References

1. Qu, C.K. The SHP-2 tyrosine phosphatase: Signaling mechanisms and biological functions. *Cell Res.* **10**(4), 279-288 (2000).
2. Barford, D. and Neel, B.G. Revealing mechanisms for SH2 domain mediated regulation of the protein tyrosine phosphatase SHP-2. *Structure* **6**(3), 249-254 (1998).
3. Tartaglia, M., Niemeyer, C.M., Fragale, A., *et al.* Somatic mutations in *PTPN11* in juvenile myelomonocytic leukemia, myelodysplastic syndromes and acute myeloid leukemia. *Nat. Genet.* **34**(2), 148-150 (2003).
4. Marasco, M., Berteotti, A., Weyershaeuser, J., *et al.* Molecular mechanism of SHP2 activation by PD-1 stimulation. *Sci. Adv.* **6**(5), 4458 (2020).
5. Christofides, A., Katopodi, X.L., Cao, C., *et al.* SHP-2 and PD-1-SHP-2 signaling regulate myeloid cell differentiation and antitumor responses. *Nat. Immunol.* **24**(1), 55-68 (2023).

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