

**Note:** Centrifuge before opening to ensure complete recovery of vial contents.

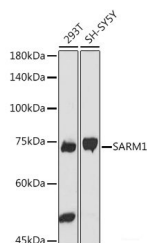
## Description

<b>Reactivity</b>	Human
<b>Immunogen</b>	A synthetic peptide of human SARM1
<b>Host</b>	Rabbit
<b>Isotype</b>	IgG
<b>Purification</b>	Affinity purification
<b>Conjugation</b>	Unconjugated
<b>Formulation</b>	PBS with 0.05% proclin300,50% glycerol,pH7.3.

## Applications Recommended Dilution

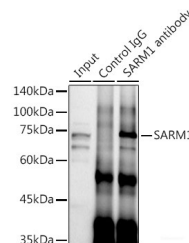
<b>WB</b>	1:500-1:2000
<b>IP</b>	1:50-1:200

## Data



Western blot analysis of extracts of various cell lines using SARM1 Polyclonal Antibody at 1:1000 dilution.

**Observed Mw:73KDa**  
**Calculated Mw:79kDa**



Immunoprecipitation analysis of 300ug extracts of SH-SY5Y cells using 3ug SARM1 Polyclonal Antibody. Western blot was performed from the immunoprecipitate using SARM1 Polyclonal Antibody at a dilution of 1:1000.

## Preparation & Storage

**Storage** Store at -20°C. Avoid freeze/thaw cycles.

## Background

NAD(+) hydrolase, which plays a key role in axonal degeneration following injury by regulating NAD(+) metabolism. Acts as a negative regulator of MYD88- and TRIF-dependent toll-like receptor signaling pathway by promoting Wallerian degeneration, an injury-induced form of programmed subcellular death which involves degeneration of an axon distal to the injury site. Wallerian degeneration is triggered by NAD(+) depletion: in response to injury, SARM1 is activated and catalyzes cleavage of NAD(+) into ADP-D-ribose (ADPR, cyclic ADPR (cADPR and nicotinamide; NAD(+) cleavage promoting cytoskeletal degradation and axon destruction. Also able to hydrolyze NADP(+), but not other NAD(+)-related molecules. Can activate neuronal cell death in response to stress. Regulates dendritic arborization through the MAPK4-JNK pathway (By similarity. Involved in innate immune response: inhibits both TICAM1/TRIF- and MYD88-dependent activation of JUN/AP-1, TRIF-dependent activation of NF-kappa-B and IRF3, and the phosphorylation of MAPK14/p38.

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