

SARM Antibody
Catalog # ASC11503**Specification****SARM Antibody - Product Information**

Application	WB, IF, E
Primary Accession	Q6SZW1
Other Accession	NP_055892 , 154090976
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Application Notes	SARM antibody can be used for detection of SARM by Western blot at 1 - 2 µg/mL. For immunofluorescence start at 20 µg/mL.

SARM Antibody - Additional InformationGene ID **23098****Target/Specificity**

SARM1; At least three alternatively spliced transcript variants encoding distinct isoforms have been observed. SARM antibody recognize the longest isoform.

Reconstitution & Storage

Antibody can be stored at 4°C up to one year. Antibodies should not be exposed to prolonged high temperatures.

Precautions

SARM Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

SARM Antibody - Protein Information**Name** SARM1**Function**

NAD(+) hydrolase, which plays a key role in axonal degeneration following injury by regulating NAD(+) metabolism (PubMed:25908823, PubMed:27671644, PubMed:28334607). 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 (PubMed:15123841, PubMed:16964262, PubMed:20306472, PubMed:25908823). 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 (PubMed:25908823, PubMed:28334607, PubMed:30333228, PubMed:31128467, PubMed:31439792, PubMed:31439793, PubMed:32049506, PubMed:32828421, PubMed:33053563). Also able to hydrolyze NADP(+), but not other NAD(+)-related molecules (PubMed:29395922). Can activate neuronal cell death in response to stress (PubMed:20306472). 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 (PubMed:16964262).

Cellular Location

Cytoplasm. Cell projection, axon {ECO:0000250|UniProtKB:Q6PDS3}. Cell projection, dendrite {ECO:0000250|UniProtKB:Q6PDS3}. Synapse {ECO:0000250|UniProtKB:Q6PDS3}. Mitochondrion Note=Associated with microtubules. {ECO:0000250|UniProtKB:Q6PDS3}

Tissue Location

Predominantly expressed in brain, kidney and liver. Expressed at lower level in placenta.

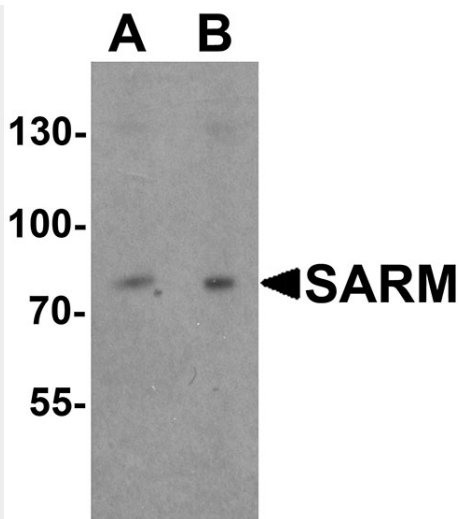
SARM Antibody - Protocols

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

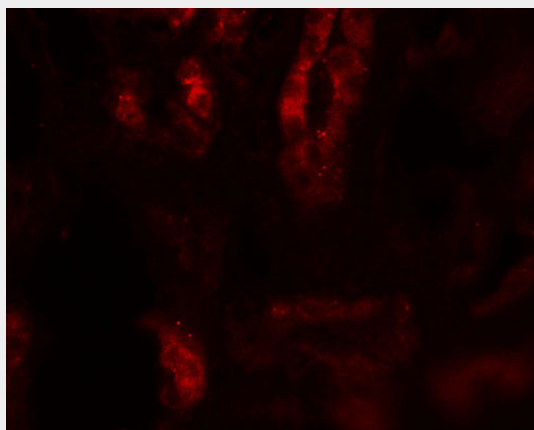
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

SARM Antibody - Images





Western blot analysis of SARM in Daudi cell lysate with SARM antibody at (A) 1 and (B) 2 µg/mL.



Immunofluorescence of SARM in human kidney tissue with SARM antibody at 20 µg/mL.

SARM Antibody - Background

SARM Antibody: Toll-like receptors (TLRs) are signaling molecules that recognize different microbial products during infection and serve as an important link between the innate and adaptive immune responses. SARM (SAM and ARM-containing protein), along with other molecules such as TIRP, TRIF, TIRAP, and MyD88, is thought to serve as an adaptor protein for the TLRs that allows for the activation of downstream kinases and NF-κB, and ultimately the expression of proteins involved in host defense. While SARM has not been conclusively shown to associate directly with TLRs, the presence of a Toll-interleukin-1 (TIR) domain in SARM is consistent with a role as a signaling molecule.

SARM Antibody - References

Vogel SN, Fitzgerald KA, and Fenton MJ. TLRs: differential adapter utilization by toll-like receptors mediates TLR-specific patterns of gene expression. *Mol. Interv.* 2003; 3:466-77.
Takeda K, Kaisho T, and Akira S. Toll-like receptors. *Annu. Rev. Immunol.* 2003; 21:335-76.
Janeway CA Jr and Medzhitov R. Innate immune recognition. *Annu. Rev. Immunol.* 2002; 20:197-216.
O'Neill LAJ, Fitzgerald FA, and Bowie AG. The Toll-IL-1 receptor adaptor family grows to five members. *Trends in Imm.* 2003; 24:286-9.