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SARM1 / SARM Antibody (clone Sarmy-1)

Mouse Monoclonal Antibody
Catalog # ALS11212

Specification

SARM1 / SARM Antibody (clone Sarmy-1) - Product Information

Application	**IHC-P, IP**
Primary Accession	Q6SZW1
Reactivity	**Human**
Host	**Mouse**
Clonality	**Monoclonal**
Calculated MW	**79kDa KDa**
Dilution	**IHC-P~~N/A** **IP~~N/A**

SARM1 / SARM Antibody (clone Sarmy-1) - Additional Information

Gene ID 23098

Other Names
Sterile alpha and TIR motif-containing protein 1, Sterile alpha and Armadillo repeat protein, Sterile alpha motif domain-containing protein 2, MyD88-5, SAM domain-containing protein 2, Tir-1 homolog, SARM1, KIAA0524, SAMD2, SARM

Target/Specificity
Recognizes human SARM.

Reconstitution & Storage
Long term: -70°C; Short term: +4°C

Precautions
SARM1 / SARM Antibody (clone Sarmy-1) is for research use only and not for use in diagnostic or therapeutic procedures.

SARM1 / SARM Antibody (clone Sarmy-1) - 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.

SARM1 / SARM Antibody (clone Sarmy-1) - 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

SARM1 / SARM Antibody (clone Sarmy-1) - Images

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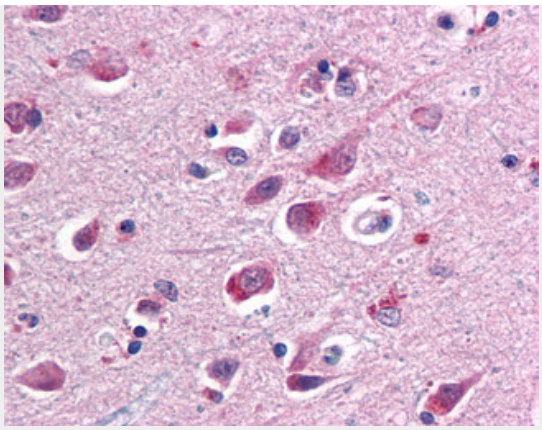

Anti-SARM1 / SARM antibody IHC of human brain, cortex.

SARM1 / SARM Antibody (clone Sarmy-1) - Background

Negative regulator of MYD88- and TRIF-dependent toll- like receptor signaling pathway which plays a pivotal role in activating axonal degeneration following injury. Promotes Wallerian degeneration an injury-induced axonal death pathway which involves degeneration of an axon distal to the injury site. Can activate neuronal death in response to stress. Regulates dendritic arborization through the MAPK4-JNK pathway. 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.

SARM1 / SARM Antibody (clone Sarmy-1) - References

Mink M.,et al.Genomics 74:234-244(2001).
Bousson J.-C.,et al.Submitted (OCT-2003) to the EMBL/GenBank/DDBJ databases.
Nagase T.,et al.DNA Res. 5:31-39(1998).
Liberati N.T.,et al.Proc. Natl. Acad. Sci. U.S.A. 101:6593-6598(2004).
Carty M.,et al.Nat. Immunol. 7:1074-1081(2006).