Revision 1

Store at -20C

#12532

Siva-1 Antibody

Cell Signaling TECHNOLOGY®

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For Research Use Only. Not for Use in Diagnostic Procedures.

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, IP	H	Endogenous	19	Rabbit	#O15304	10572

Product Usage Information

Application	Dilution
Western Blotting	1:1000
Immunoprecipitation	1:50

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA and 50% glycerol. Store at –20°C. Do not aliquot the antibody.

Specificity/Sensitivity

Siva-1 Antibody recognizes endogenous levels of total Siva-1 protein. This antibody does not cross-react with Siva-2. This antibody cross-reacts with a protein of unknown origin at ~70 kDa.

Source / Purification

Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Pro71 of human Siva-1 protein. Antibodies are purified by protein A and peptide affinity chromatography.

Background

First identified as a pro-apoptotic protein that binds the cytoplasmic tail of the TNF receptor superfamily member CD27 (1), Siva-1 also binds several other TNFR family members including glucocorticoid-induced tumor necrosis factor receptor (GITR) and OX40 (1-3), as well as anti-apoptotic Bcl-2 family members Bcl-xL and Bcl-2 (4,5). Siva-1 is composed of a central death domain homology region, a C-terminal box-B-like ring finger followed by a zinc finger-like domain, and a unique N-terminal amphipathic helical region (SAH) (1,4). Studies have demonstrated that Siva-1 has the ability to induce cell death via both the extrinsic and intrinsic apoptotic pathways (1-8). The SAH domain of Siva-1 is responsible for the inhibition of the pro-survival activities of Bcl-xL and Bcl-2, leading to caspase-mediated cell death (4,5,8). Siva-1 plays a role in T cell signaling and homeostasis by inhibiting NF-κB activity, also resulting in apoptotic cell death (7,9). An alternative splice variant of Siva-1, Siva-2, lacks part of the SAH and death domains and is less effective at inducing apoptosis (1,2,5,8). Studies in xenografts have shown that down-regulation of Siva-1 inhibits tumorigenesis in response to p53 activation (10). Down-regulation of Siva-1 may also play a role in tumor metastasis through its regulation of the epithelial-mesenchymal transition (EMT) and cell migration (11). Overexpression of Siva-1 is implicated in several pathological conditions including acute ischemic injury (12) and Coxsackievirus infection (13).

Background References

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6. Cao, C. et al. (2001) *J Biol Chem* 276, 11465-8.
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8. Py, B. et al. (2004) *J Immunol* 172, 4008-17.
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10. Du, W. et al. (2009) *Cell Death Differ* 16, 1493-504.
11. Li, N. et al. (2011) *Proc Natl Acad Sci U S A* 108, 12851-6.
12. Padanilam, B.J. et al. (1998) *Kidney Int* 54, 1967-75.
13. Henke, A. et al. (2000) *J Virol* 74, 4284-90.

Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Western Blot Buffer

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

Applications Key

W: Western Blotting **IP:** Immunoprecipitation

Cross-Reactivity Key

H: Human

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Limited Uses