Revision 5

Store at -20C

#6319

Phospho-DRP1 (Ser637) (D3A4) Rabbit mAb

Cell Signaling TECHNOLOGY®

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Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, W-S	R	Endogenous	78-82	Rabbit IgG	#O00429	10059

Product Usage Information

Application	Dilution
Western Blotting	1:1000
Simple Western™	1:10 - 1:50

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.

Specificity/Sensitivity

Phospho-DRP1 (Ser637) (D3A4) Rabbit mAb recognizes endogenous levels of DRP1 protein only when phosphorylated at Ser637.

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic phosphopeptide corresponding to residues surrounding Ser637 of human DRP1 protein.

Background

Dynamin-related protein 1 (DRP1) is a member of the dynamin superfamily of GTPases. Members of this family have diverse cellular functions including vesicle scission, organelle fission, viral resistance, and intracellular trafficking (reviewed in 1). DRP1 affects mitochondrial morphology and is important in mitochondrial and peroxisomal fission in mammalian cells (2-5). The yeast ortholog of DRP1 clusters into a spiral-shaped structure on the mitochondrial membrane at the site of fission (reviewed in 6), and this structure is likely conserved in mammalian cells (3). The division of the mitochondria, which is required for apoptosis, as well as normal cell growth and development is controlled, in part, by the phosphorylation of DRP1 at Ser616 by Cdk1/cyclin B and at Ser637 by protein kinase A (PKA) (reviewed in 6). When phosphorylated at Ser616, DRP1 stimulates mitochondrial fission during mitosis. Conversely, fission is inhibited when DRP1 is phosphorylated at Ser637 (reviewed in 6). Dephosphorylation at Ser637 by calcineurin reverses this inhibition (7). In addition to phosphorylation, sumoylation of DRP1 is also an enhancer of mitochondrial fission (8). Balancing fission and fusion events is essential for proper mitochondrial function. Research studies have demonstrated mitochondrial defects in a variety of neurodegenerative diseases including Alzheimer's disease, Parkinson's disease, and Huntington's disease (reviewed in 6).

Background References

1. Praefcke, G.J. and McMahon, H.T. (2004) *Nat Rev Mol Cell Biol* 5, 133-47.
2. Taguchi, N. et al. (2007) *J Biol Chem* 282, 11521-9.
3. Smirnova, E. et al. (2001) *Mol Biol Cell* 12, 2245-56.
4. Smirnova, E. et al. (1998) *J Cell Biol* 143, 351-8.
5. Koch, A. et al. (2003) *J Biol Chem* 278, 8597-605.
6. Knott, A.B. et al. (2008) *Nat Rev Neurosci* 9, 505-18.
7. Cereghetti, G.M. et al. (2008) *Proc Natl Acad Sci USA* 105, 15803-8.
8. Zunino, R. et al. (2007) *J Cell Sci* 120, 1178-88.

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 **W-S:** Simple Western™

Cross-Reactivity Key

R: Rat

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