Revision 1

Store at -20C

#14649

DNA Ligase IV (D5N5N) Rabbit mAb

Cell Signaling TECHNOLOGY®

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Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W	H	Endogenous	100	Rabbit IgG	#P49917	3981

Product Usage Information

Application	Dilution
Western Blotting	1:1000

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

DNA Ligase IV (D5N5N) Rabbit mAb recognizes endogenous levels of total DNA ligase IV protein.

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Leu771 of human DNA ligase IV protein.

Background

DNA double-strand breaks (DSBs) are potentially hazardous lesions that can be induced by ionizing radiation (IR), radiomimetic chemicals, or DNA replication inhibitors. Cells detect and repair DSBs through two distinct but partly overlapping signaling pathways, nonhomologous end joining (NHEJ) and homologous recombination (HR). DNA repair through the HR pathway is restricted to S and G2 phases of the cell cycle, while NHEJ can occur during any cell cycle phase. Defects in both pathways have been associated with human disease, including cancer (1).

DNA repair through the NHEJ pathway involves a core group of proteins that includes the Ku heterodimer, DNA-PKcs, DNA ligase IV, XRCC4, and XLF. XLF interacts with XRCC4 and promotes the ligation of DNA strands by DNA ligase IV and the ligase cofactor XRCC4. The ATP-dependent ligation of free DNA ends is the final step in the NHEJ repair pathway (2). Research studies suggest that XLF and XRCC4 proteins form complexes that bridge DNA breaks earlier in the NHEJ pathway (3). Additional studies indicate that localization of XRCC4 to the nucleus and levels of XRCC4 protein are both regulated by DNA ligase IV (4). Mutations in the corresponding *LIG4* gene are associated with LIG4 syndrome, a disorder characterized by immunodeficiency and developmental growth delay. Cells isolated from patients diagnosed with LIG4 syndrome display typical cell cycle checkpoint activity, but aberrant rejoining of DNA double strand breaks (5,6).

Background References

1. Hartlerode, A.J. and Scully, R. (2009) *Biochem J* 423, 157-68.
2. Tsai, C.J. et al. (2007) *Proc Natl Acad Sci U S A* 104, 7851-6.
3. Andres, S.N. et al. (2012) *Nucleic Acids Res* 40, 1868-78.
4. Francis, D.B. et al. (2014) *DNA Repair (Amst)* 21, 36-42.
5. O'Driscoll, M. et al. (2001) *Mol Cell* 8, 1175-85.
6. O'Driscoll, M. et al. (2004) *DNA Repair (Amst)* 3, 1227-35.

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

Cross-Reactivity Key

H: Human

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Limited Uses

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