SANTA CRUZ BIOTECHNOLOGY, INC.

DNA Ligase IV (D-8): sc-271299

BACKGROUND

The X-ray repair cross-complementing protein XRCC4 and DNA Ligase IV are essential for repairing double-strand breaks in DNA. These proteins form a critical complex consisting of two molecules of each protein that preferentially bind DNA with nicks or broken ends. As an obligate accessory molecule, XRCC4 binds to DNA Ligase IV and enhances its joining activity. The XRCC4/DNA Ligase IV complex is also involved in V(D)J recombination. V(D)J recombination occurs in normal development of the adaptive immune system and involves the formation of a double-strand break intermediate. Deletions of either DNA Ligase IV or XRCC4 inhibit the completion of V(D)J recombination, resulting in a high incidence of apoptosis in the developing nervous system and a block in B and T cell maturation.

CHROMOSOMAL LOCATION

Genetic locus: LIG4 (human) mapping to 13q33.3; Lig4 (mouse) mapping to 8 A1.1.

SOURCE

DNA Ligase IV (D-8) is a mouse monoclonal antibody raised against amino acids 545-844 mapping at the C-terminus of DNA Ligase IV of human origin.

PRODUCT

Each vial contains 200 µg IgG_{2a} kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

DNA Ligase IV (D-8) is available conjugated to agarose (sc-271299 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; to HRP (sc-271299 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-271299 PE), fluorescein (sc-271299 FITC), Alexa Fluor® 488 (sc-271299 AF488), Alexa Fluor® 546 (sc-271299 AF546), Alexa Fluor® 594 (sc-271299 AF594) or Alexa Fluor® 647 (sc-271299 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor® 680 (sc-271299 AF680) or Alexa Fluor® 790 (sc-271299 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

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APPLICATIONS

DNA Ligase IV (D-8) is recommended for detection of DNA Ligase IV of mouse, rat and human origin by Western Blotting (starting dilution 1:100, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for DNA Ligase IV siRNA (h): sc-37394, DNA Ligase IV siRNA (m): sc-72113, DNA Ligase IV shRNA Plasmid (h): sc-37394-SH, DNA Ligase IV shRNA Plasmid (m): sc-72113-SH, DNA Ligase IV shRNA (h) Lentiviral Particles: sc-37394-V and DNA Ligase IV shRNA (m) Lentiviral Particles: sc-72113-V.

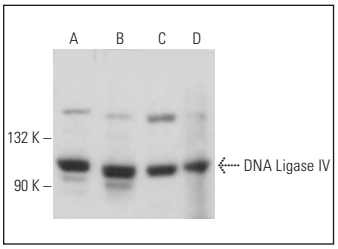
Molecular Weight of DNA Ligase IV: 96 kDa.

Positive Controls: JAR cell lysate: sc-2276, HeLa whole cell lysate: sc-2200 or Ramos whole cell lysate: sc-2216.

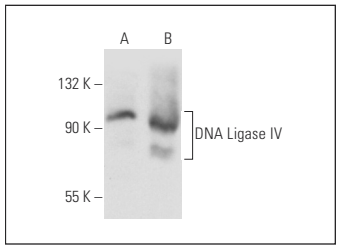
STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

DATA



DNA Ligase IV (D-8): sc-271299. Western blot analysis of DNA Ligase IV expression in HeLa (**A**), JAR (**B**) and KNRK (**C**) whole cell lysates and rat thymus tissue extract (**D**).



DNA Ligase IV (D-8): sc-271299. Western blot analysis of DNA Ligase IV expression in HeLa (**A**) and Ramos (**B**) whole cell lysates.

SELECT PRODUCT CITATIONS

1. Kim, M.R., et al. 2015. TGFβ1 protects cells from γ-IR by enhancing the activity of the NHEJ repair pathway. Mol. Cancer Res. 13: 319-329.
2. Chakraborty, A., et al. 2016. Classical non-homologous end-joining pathway utilizes nascent RNA for error-free double-strand break repair of transcribed genes. Nat. Commun. 7: 13049.
3. Hromas, R., et al. 2017. The endonuclease EEPD1 mediates synthetic lethality in Rad52-depleted BRCA1 mutant breast cancer cells. Breast Cancer Res. 19: 122.
4. Bhargava, R., et al. 2018. C-NHEJ without indels is robust and requires synergistic function of distinct XLF domains. Nat. Commun. 9: 2484.
5. Singh, V., et al. 2019. Azoospermic infertility is associated with altered expression of DNA repair genes. DNA Repair 75: 39-47.
6. Mladenov, E., et al. 2020. Strong suppression of gene conversion with increasing DNA double-strand break load delimited by 53BP1 and Rad52. Nucleic Acids Res. 48: 1905-1924.
7. Park, Y.J., et al. 2020. Ribosomal Protein S3 is a novel negative regulator of non-homologous end joining repair of DNA double-strand breaks. FASEB J. 34: 8102-8113.
8. Noh, S.E., et al. 2020. Inhibition of non-homologous end joining of gamma ray-induced DNA double-strand breaks by cAMP signaling in lung cancer cells. Sci. Rep. 10: 14455.
9. Olmedo-Pelayo, J., et al. 2020. Canonical non-homologous end-joining promotes genome mutagenesis and translocations induced by transcription-associated DNA topoisomerase 2 activity. Nucleic Acids Res. 48: 9147-9160.

RESEARCH USE

For research use only, not for use in diagnostic procedures.