DinB (A-9): sc-166667



The Power to Question

BACKGROUND

Problems in DNA replication may lead to breaks in the replication fork, and recombinational reactions occur to restore the integrity of the fork via strandinvasion of the broken chromosome with its homologous strand. If this happens within repeated DNA sequences, genetic rearrangements may be produced. The bacterial UmuC/DinB family consists of bypass polymerases that are responsible for translesion DNA synthesis. DinB, also referred to as DNA polyermase IV or DNA polymerase κ , is an SOS-inducible, error-prone DNA polymerase that plays a role in DNA damage-induced mutagenesis by preferentially making frameshift mutations. DinB is uniquely and highly expressed in the adrenal cortex and testis, as well as in a variety of other tissues. p53 regulates DinB and exposure to various DNA-damaging agents causes an upregulation of DinB.

REFERENCES

- Silvian, L.F., et al. 2001. Crystal structure of a DinB family error-prone DNA polymerase from Sulfolobus solfataricus. Nat. Struct. Biol. 8: 984-989.
- Zhou, B.L., et al. 2001. Crystal structure of a DinB lesion bypass DNA polymerase catalytic fragment reveals a classic polymerase catalytic domain. Mol. Cell 8: 427-437.
- 3. Velasco-Miguel, S., et al. 2003. Constitutive and regulated expression of the mouse DinB (Pol- κ) gene encoding DNA polymerase κ . DNA Repair 2: 91-106.

CHROMOSOMAL LOCATION

Genetic locus: POLK (human) mapping to 5q13.3; Polk (mouse) mapping to 13 D1.

SOURCE

DinB (A-9) is a mouse monoclonal antibody raised against amino acids 131-310 mapping near the N-terminus of DinB of human origin.

PRODUCT

Each vial contains 200 μ g lgG $_1$ kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin. Also available as TransCruz reagent for Gel Supershift and ChIP applications, sc-166667 X, 200 μ g/0.1 ml.

DinB (A-9) is available conjugated to agarose (sc-166667 AC), 500 $\mu g/0.25$ ml agarose in 1 ml, for IP; to HRP (sc-166667 HRP), 200 $\mu g/ml$, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-166667 PE), fluorescein (sc-166667 FITC), Alexa Fluor® 488 (sc-166667 AF488), Alexa Fluor® 546 (sc-166667 AF546), Alexa Fluor® 594 (sc-166667 AF594) or Alexa Fluor® 647 (sc-166667 AF647), 200 $\mu g/ml$, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor® 680 (sc-166667 AF680) or Alexa Fluor® 790 (sc-166667 AF790), 200 $\mu g/ml$, for Near-Infrared (NIR) WB, IF and FCM.

Alexa Fluor® is a trademark of Molecular Probes, Inc., Oregon, USA

STORAGE

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

APPLICATIONS

DinB (A-9) is recommended for detection of all DinB isoforms of mouse, rat and human origin by Western Blotting (starting dilution 1:100, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g 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).

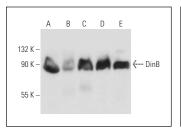
Suitable for use as control antibody for DinB siRNA (h): sc-60537, DinB siRNA (m): sc-60538, DinB shRNA Plasmid (h): sc-60537-SH, DinB shRNA Plasmid (m): sc-60538-SH, DinB shRNA (h) Lentiviral Particles: sc-60537-V and DinB shRNA (m) Lentiviral Particles: sc-60538-V.

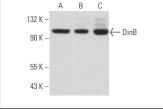
DinB (A-9) X TransCruz antibody is recommended for Gel Supershift and ChIP applications.

Molecular Weight of DinB: 99 kDa.

Positive Controls: HeLa nuclear extract: sc-2120, F9 cell lysate: sc-2245 or PC-12 cell lysate: sc-2250.

DATA





DinB (A-9): sc-166667. Western blot analysis of DinB expression in F9 (A), PC-12 (B), DU 145 (C) and COLO 320DM (D) whole cell lysates and HeLa nuclear

DinB (A-9): sc-166667. Western blot analysis of DinB expression in F9 ($\bf A$), MDA-MB-231 ($\bf B$) and CCRF-CEM ($\bf C$) whole cell lysates.

SELECT PRODUCT CITATIONS

- 1. Ω i, Y., et al. 2016. DNA polymerase κ is a key cellular factor for the formation of covalently closed circular DNA of hepatitis B virus. PLoS Pathog. 12: e1005893.
- 2. Tonzi, P., et al. 2018. Translesion polymerase κ -dependent DNA synthesis underlies replication fork recovery. Elife 7: e41426.
- 3. Thakar, T., et al. 2020. Ubiquitinated-PCNA protects replication forks from DNA2-mediated degradation by regulating Okazaki fragment maturation and chromatin assembly. Nat. Commun. 11: 2147.
- Coleman, K.E., et al. 2022. USP1-trapping lesions as a source of DNA replication stress and genomic instability. Nat. Commun. 13: 1740.
- Kanao, R., et al. 2022. RFWD3 and translesion DNA polymerases contribute to PCNA modification-dependent DNA damage tolerance. Life Sci. Alliance 5: e202201584.

RESEARCH USE

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