

BLM (B-4): sc-365753

BACKGROUND

Bloom's syndrome is an autosomal recessive disorder characterized by pre- and post-natal growth deficiencies, sun sensitivity, immunodeficiency and a predisposition to various cancers. The gene responsible for Bloom's syndrome, BLM, encodes a protein homologous to the RecQ helicase of *E. coli* and is mutated in most Bloom's syndrome patients. One characteristic of Bloom's syndrome is an increased frequency of sister chromatid exchange (SCE). BLM has been shown to unwind G₄ DNA, and a failure of this function is thought to be responsible for the increased rate of SCE. BLM is known to be translocated to the nucleus, where its ATPase activity is stimulated by both single- and double-stranded DNA. Mutations in the yeast SGS1, a homolog of BLM, are known to cause mitotic hyperrecombination similar to that observed in Bloom's cells.

CHROMOSOMAL LOCATION

Genetic locus: BLM (human) mapping to 15q26.1; Blm (mouse) mapping to 7 D3.

SOURCE

BLM (B-4) is a mouse monoclonal antibody raised against amino acids 1118-1417 of BLM of human origin.

PRODUCT

Each vial contains 200 µg IgG_{2b} kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.
BLM (B-4) is available conjugated to agarose (sc-365753 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; to HRP (sc-365753 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-365753 PE), fluorescein (sc-365753 FITC), Alexa Fluor® 488 (sc-365753 AF488), Alexa Fluor® 546 (sc-365753 AF546), Alexa Fluor® 594 (sc-365753 AF594) or Alexa Fluor® 647 (sc-365753 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor® 680 (sc-365753 AF680) or Alexa Fluor® 790 (sc-365753 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

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APPLICATIONS

BLM (B-4) is recommended for detection of BLM 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 BLM siRNA (h): sc-29808, BLM siRNA (m): sc-29809, BLM shRNA Plasmid (h): sc-29808-SH, BLM shRNA Plasmid (m): sc-29809-SH, BLM shRNA (h) Lentiviral Particles: sc-29808-V and BLM shRNA (m) Lentiviral Particles: sc-29809-V.

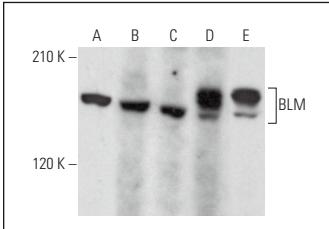
Molecular Weight of BLM: 180 kDa.

Positive Controls: Jurkat whole cell lysate: sc-2204, NIH/3T3 whole cell lysate: sc-2210 or Raji whole cell lysate: sc-364236.

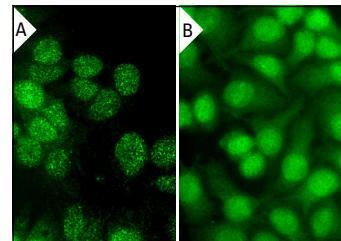
STORAGE

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

DATA



BLM (B-4): sc-365753. Western blot analysis of BLM expression in Jurkat (**A**), Raji (**B**), NIH/3T3 (**C**), NRK (**D**) and RAT2 (**E**) whole cell lysates.



BLM (B-4): sc-365753. Immunofluorescence staining of formalin-fixed Hep G2 cells showing nuclear localization (**A**). Immunofluorescence staining of formalin-fixed HeLa cells showing nuclear and cytoplasmic localization (**B**).

SELECT PRODUCT CITATIONS

- Shen, J., et al. 2019. PARPi triggers the STING-dependent immune response and enhances the therapeutic efficacy of immune checkpoint blockade independent of BRCA1/2. *Cancer Res.* 79: 311-319.
- Chen, T.I., et al. 2019. Hepatitis C virus NS3 protein plays a dual role in WRN-mediated repair of non-homologous end joining. *J. Virol.* 93: e01273-19.
- Townsend, A., et al. 2021. DCAF14 promotes stalled fork stability to maintain genome integrity. *Cell Rep.* 34: 108669.
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- Xiao, H., et al. 2022. Increased resection at DSBs in G₂-phase is a unique phenotype associated with DNA-PK_{cs} defects that is not shared by other factors of c-NHEJ. *Cells* 11: 2099.
- Guh, C.Y., et al. 2022. XPF activates break-induced telomere synthesis. *Nat. Commun.* 13: 5781.
- Shukla, V., et al. 2022. TET deficiency perturbs mature B cell homeostasis and promotes oncogenesis associated with accumulation of G-quadruplex and R-loop structures. *Nat. Immunol.* 23: 99-108.
- Ovejero, S., et al. 2022. The BLM helicase is a new therapeutic target in multiple myeloma involved in replication stress survival and drug resistance. *Front. Immunol.* 13: 983181.
- Maresca, C., et al. 2023. PARP1 allows proper telomere replication through TRF1 poly(ADP-ribosylation) and helicase recruitment. *Commun. Biol.* 6: 234.
- Liu, H., et al. 2023. Cancer-associated SMARCAL1 loss-of-function mutations promote alternative lengthening of telomeres and tumorigenesis in telomerase-negative glioblastoma cells. *Neuro Oncol.* 25: 1563-1575.

RESEARCH USE

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