Revision 3

Store at -20C

#2742

BLM Antibody

Cell Signaling TECHNOLOGY®

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For Research Use Only. Not for Use in Diagnostic Procedures.

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W	H	Endogenous	190	Rabbit	#P54132	641

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 and 50% glycerol. Store at –20°C. Do not aliquot the antibody.

Specificity/Sensitivity

BLM Antibody detects endogenous levels of total BLM protein. Bands of unknown origin may be detected at approximately 220, 90 and 80 kDa.

Source / Purification

Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Arg85 of human BLM. Antibodies are purified by peptide affinity chromatography.

Background

BLM, a member of the RecQ family of DNA helicases, is part of the BRCA1-associated genome surveillance complex (BASC) that responds to DNA damage, stalled replication forks and S phase arrest (1-4). Phosphorylation of BLM helicase at Thr99 and Thr122 occurs in response to genotoxic stress (4), and phosphorylation of Ser144 appears to be important in regulating chromosome stability during mitosis (5). Typical BLM protein resides in the nucleus and forms part of a dynamic protein complex that acts in response to DNA damage during specific periods of the cell cycle (6). Although RecQ helicases are rarely considered as essential enzymes, they function at the interface between DNA recombination and repair and are required for global genome stability maintenance. Mutations in BLM helicase are responsible for development of Bloom Syndrome, a recessive genetic disorder clinically characterized by short stature, immunodeficiency and elevated risk of malignancy (7). Similar alterations to genes encoding the related RecQ helicases RecQ4 and WRN also result in recessive genetic disorders associated with genomic instability (8,9). Cells from Bloom Syndrome patients exhibit genomic instability and increased frequency of sister chromatid exchange (10).

Background References

1. Wang, Y. et al. (2000) *Genes Dev.* 14, 927-939.
2. Langland, G. et al. (2002) *Cancer Res.* 62, 2766-2770.
3. Sengupta, S. et al. (2003) *EMBO J.* 22, 1210-1222.
4. Davies, S.L. et al. (2004) *Mol. Cell. Biol.* 24, 1279-1291.
5. Leng, M. et al. (2006) *Proc. Natl. Acad. Sci. USA* 103, 11485-11490.
6. Bischof, O. et al. (2001) *J. Cell Biol.* 153, 367-380.
7. van Brabant, A.J. et al. (2000) *Annu. Rev. Genomics Hum. Genet.* 1, 409-459.
8. Kitao, S. et al. (1999) *Nat. Genet.* 22, 82-84.
9. Yu, C.E. et al. (1996) *Science* 272, 258-262.
10. Chaganti, R.S. et al. (1974) *Proc. Natl. Acad. Sci. USA* 71, 4508-4512.

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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