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MSL3 Antibody (N-term)

Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP17736a

Specification

MSL3 Antibody (N-term) - Product Information

Application	**WB,E**
Primary Accession	Q8N5Y2
Other Accession	Q9WVG9, NP_001180199.1
Reactivity	**Human**
Predicted	**Mouse**
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**Rabbit IgG**
Calculated MW	**59824**
Antigen Region	**28-55**

MSL3 Antibody (N-term) - Additional Information

Gene ID 10943

Other Names
Male-specific lethal 3 homolog, Male-specific lethal-3 homolog 1, Male-specific lethal-3 protein-like 1, MSL3-like 1, MSL3, MSL3L1

Target/Specificity
This MSL3 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 28-55 amino acids from the N-terminal region of human MSL3.

Dilution
WB~~1:1000
E~~Use at an assay dependent concentration.

Format
Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage
Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions
MSL3 Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

MSL3 Antibody (N-term) - Protein Information

Name MSL3 {ECO:0000303|PubMed:16227571, ECO:0000312|HGNC:HGNC:7370}

Function Non-catalytic component of the MSL histone acetyltransferase complex, a multiprotein complex that mediates the majority of histone H4 acetylation at 'Lys-16' (H4K16ac), an epigenetic mark that prevents chromatin compaction (PubMed:16227571, PubMed:16543150, PubMed:20018852, PubMed:20657587, PubMed:20943666, PubMed:21217699, PubMed:30224647, PubMed:33837287). The MSL complex is required for chromosome stability and genome integrity by maintaining homeostatic levels of H4K16ac (PubMed:33837287). The MSL complex is also involved in gene dosage by promoting up-regulation of genes expressed by the X chromosome (By similarity). X up-regulation is required to compensate for autosomal biallelic expression (By similarity). The MSL complex also participates in gene dosage compensation by promoting expression of Tsix non-coding RNA (By similarity). Acts as a histone reader that specifically recognizes and binds histone H4 monomethylated at 'Lys-20' (H4K20Me1) in a DNA-dependent manner and is proposed to be involved in chromosomal targeting of the MSL complex (PubMed:20657587, PubMed:20943666). May play a role X inactivation in females (PubMed:21217699).

Cellular Location
Nucleus.

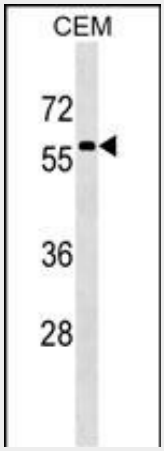
Tissue Location
Expressed in many tissues including liver, pancreas, heart, lung, kidney, skeletal muscle, brain, and placenta, with highest expression in skeletal muscle and heart

MSL3 Antibody (N-term) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

MSL3 Antibody (N-term) - Images



MSL3 Antibody (N-term) (Cat. #AP17736a) western blot analysis in CEM cell line lysates (35ug/lane).This demonstrates the MSL3 antibody detected the MSL3 protein (arrow).

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MSL3 Antibody (N-term) - Background

This gene encodes a nuclear protein that is similar to the product of the Drosophila male-specific lethal-3 gene. The Drosophila protein plays a critical role in a dosage-compensation pathway, which equalizes X-linked gene expression in males and females. Thus, the human protein is thought to play a similar function in chromatin remodeling and transcriptional regulation, and it has been found as part of a complex that is responsible for histone H4 lysine-16 acetylation. This gene can undergo X inactivation. Alternative splicing results in multiple transcript variants. Related pseudogenes have been identified on chromosomes 2, 7 and 8.

MSL3 Antibody (N-term) - References

Smith, E.R., et al. Mol. Cell. Biol. 25(21):9175-9188(2005)
Marin, I., et al. Mol. Biol. Evol. 17(8):1240-1250(2000)
Prakash, S.K., et al. Genomics 59(1):77-84(1999)