

ULK3 Antibody (C-term)
Purified Mouse Monoclonal Antibody (Mab)
Catalog # AW5082

Specification

ULK3 Antibody (C-term) - Product Information

Application	WB,E
Primary Accession	Q6PHR2
Reactivity	Human, Mouse, Rat
Host	Mouse
Clonality	monoclonal
Calculated MW	H=53,24;M=53,24;Rat=53,24 KDa
Isotype	IgG1,k
Antigen Source	HUMAN

ULK3 Antibody (C-term) - Additional Information

Gene ID 25989

Antigen Region
435-468

Other Names
Serine/threonine-protein kinase ULK3, Unc-51-like kinase 3, ULK3

Dilution
WB~~ 1:1000

Target/Specificity
This ULK3 antibody is generated from a mouse immunized with a KLH conjugated synthetic peptide between 435-468 amino acids from the C-terminal region of human ULK3.

Format
Purified monoclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein G column, followed by dialysis against PBS.

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
ULK3 Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

ULK3 Antibody (C-term) - Protein Information

Name ULK3

Function

Serine/threonine protein kinase that acts as a regulator of Sonic hedgehog (SHH) signaling and autophagy. Acts as a negative regulator of SHH signaling in the absence of SHH ligand: interacts with SUFU, thereby inactivating the protein kinase activity and preventing phosphorylation of GLI proteins (GLI1, GLI2 and/or GLI3). Positively regulates SHH signaling in the presence of SHH: dissociates from SUFU, autophosphorylates and mediates phosphorylation of GLI2, activating it and promoting its nuclear translocation. Phosphorylates in vitro GLI2, as well as GLI1 and GLI3, although less efficiently. Also acts as a regulator of autophagy: following cellular senescence, able to induce autophagy.

Cellular Location

Cytoplasm. Note=Localizes to pre-autophagosomal structure during cellular senescence

Tissue Location

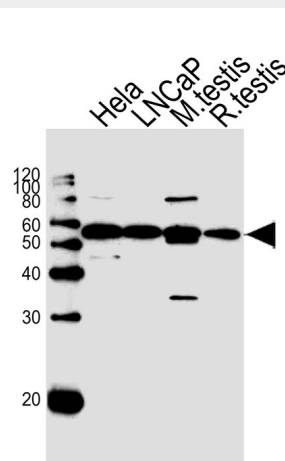
Widely expressed. Highest levels observed in fetal brain. In adult tissues, high levels in brain, liver and kidney, moderate levels in testis and adrenal gland and low levels in heart, lung, stomach, thymus, prostate and placenta. In the brain, highest expression in the hippocampus, high levels also detected in the cerebellum, olfactory bulb and optic nerve. In the central nervous system, lowest levels in the spinal cord

ULK3 Antibody (C-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 Cytometry](#)
- [Cell Culture](#)

ULK3 Antibody (C-term) - Images



Western blot analysis of lysates from HeLa, LNCaP cell line, mouse testis and rat testis tissue lysate (from left to right), using ULK3 Antibody (C-term) (Cat. #AW5082). AW5082 was diluted at

1:1000 at each lane. A goat anti-mouse IgG H&L(HRP) at 1:10000 dilution was used as the secondary antibody.

ULK3 Antibody (C-term) - Background

Serine/threonine protein kinase that acts as a regulator of Sonic hedgehog (SHH) signaling and autophagy. Acts as a negative regulator of SHH signaling in the absence of SHH ligand: interacts with SUFU, thereby inactivating the protein kinase activity and preventing phosphorylation of GLI proteins (GLI1, GLI2 and/or GLI3). Positively regulates SHH signaling in the presence of SHH: dissociates from SUFU, autophosphorylates and mediates phosphorylation of GLI2, activating it and promoting its nuclear translocation. Phosphorylates in vitro GLI2, as well as GLI1 and GLI3, although less efficiently. Also acts as a regulator of autophagy: following cellular senescence, able to induce autophagy.

ULK3 Antibody (C-term) - References

Ota T.,et al.Nat. Genet. 36:40-45(2004).
Bechtel S.,et al.BMC Genomics 8:399-399(2007).
Zody M.C.,et al.Nature 440:671-675(2006).
Mural R.J.,et al.Submitted (SEP-2005) to the EMBL/GenBank/DDBJ databases.
Daub H.,et al.Mol. Cell 31:438-448(2008).