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Cullin 7 Rabbit pAb

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Catalog # AP59153

Product Information

Application	WB, IHC-P, IHC-F, IF, E
Primary Accession	Q14999
Predicted	Human, Mouse, Rat, Dog, Horse, Sheep
Host	Rabbit
Clonality	Polyclonal
Calculated MW	191161
Physical State	Liquid
Immunogen	KLH conjugated synthetic peptide derived from human Cullin 7
Epitope Specificity	1251-1400/1698
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SUBCELLULAR LOCATION	Cytoplasm.
SIMILARITY	Belongs to the cullin family. Contains 1 DOC domain.
SUBUNIT	Part of a SCF-like complex consisting of CUL7, RBX1, SKP1, FBXW8 and GLMN isoform 1. Interacts with a complex of SKP1 and FBXW8, but not with SKP1 alone. Interacts with CUL9. Interacts with FBXW8; interaction is mutually exclusive of binding to CUL9 or TP53. Interacts with TP53; the interaction preferentially involves tetrameric and dimeric TP53. The CUL7-CUL9 heterodimer seems to interact specifically with TP53. Interacts with CUL1; the interactions seems to be mediated by FBXW8 (By similarity). Interacts with SV40 Large T antigen; this interaction seems to inhibit CUL7. Component of a SCF-like complex composed of SV40 Large T antigen, CUL7, SKP1, RBX1, and FBXW8. Interacts with OBSL1.
DISEASE	Defects in CUL7 are the cause of 3M syndrome type 1 (3M1) [MIM:273750]. An autosomal recessive disorder characterized by severe pre- and postnatal growth retardation, facial dysmorphism, large head circumference, and normal intelligence and endocrine function. Skeletal changes include long slender tubular bones and tall vertebral bodies.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	Component of a probable SCF-like E3 ubiquitin-protein ligase complex, which mediates the ubiquitination and subsequent proteasomal degradation of target proteins. Probably plays a role in the degradation of proteins involved in endothelial proliferation and/or differentiation (By similarity). Seems not to promote polyubiquitination and proteasomal degradation of TP53. In vitro, complexes of CUL7 with either CUL9 or FBXW8 or TP53 contain E3 ubiquitin-protein ligase activity. Involvement in disease: Defects in CUL7 are the cause of 3M syndrome type 1 (3M1). An autosomal recessive disorder characterized by severe pre- and postnatal growth retardation, facial dysmorphism, large head circumference, and normal intelligence and endocrine function. Skeletal changes include long slender tubular bones and tall vertebral bodies.

Additional Information

Gene ID	9820
Other Names	Cullin-7, CUL-7, CUL7, KIAA0076
Target/Specificity	Highly expressed in fetal kidney and adult skeletal muscle. Also abundant in fetal brain, as well as in adult pancreas, kidney, placenta and heart. Detected in trophoblasts, lymphoblasts, osteoblasts, chondrocytes and skin fibroblasts.
Dilution	WB=1:500-2000,IHC-P=1:100-500,IHC-F=1:100-500,IF=1:100-500,ELISA=1:5000-10000
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

Protein Information

Name	CUL7
Synonyms	KIAA0076
Function	Core component of the 3M and Cul7-RING(FBXW8) complexes, which mediate the ubiquitination and subsequent proteasomal degradation of target proteins (PubMed:12481031, PubMed:12904573, PubMed:21572988, PubMed:21737058, PubMed:24793695, PubMed:35982156). Core component of the 3M complex, a complex required to regulate microtubule dynamics and genome integrity (PubMed:21572988, PubMed:21737058, PubMed:24793695). It is unclear how the 3M complex regulates microtubules, it could act by controlling the level of a microtubule stabilizer (PubMed:24793695). The Cul7-RING(FBXW8) complex alone lacks ubiquitination activity and does not promote polyubiquitination and proteasomal degradation of p53/TP53 (PubMed:16547496, PubMed:17332328, PubMed:35982156). However it mediates recruitment of p53/TP53 for ubiquitination by neddylated CUL1-RBX1 (PubMed:35982156). Interaction with CUL9 is required to inhibit CUL9 activity and ubiquitination of BIRC5 (PubMed:24793696). The Cul7-RING(FBXW8) complex also mediates ubiquitination and consequent degradation of target proteins such as GORASP1, IRS1 and MAP4K1/HPK1 (PubMed:21572988, PubMed:24362026). Ubiquitination of GORASP1 regulates Golgi morphogenesis and dendrite patterning in brain (PubMed:21572988). Mediates ubiquitination and degradation of IRS1 in a mTOR-dependent manner: the Cul7-RING(FBXW8) complex recognizes and binds IRS1 previously phosphorylated by S6 kinase (RPS6KB1 or RPS6KB2) (PubMed:18498745). The Cul7-RING(FBXW8) complex also mediates ubiquitination of MAP4K1/HPK1: recognizes and binds autophosphorylated MAP4K1/HPK1, leading to its degradation, thereby affecting cell proliferation and differentiation (PubMed:24362026). Acts as a regulator in trophoblast cell epithelial-mesenchymal transition and placental development (PubMed:20139075). While the Cul7-RING(FBXW8) and the 3M complexes are associated and involved in common processes, CUL7 and the Cul7-RING(FBXW8) complex may have additional functions. Probably plays a role in the degradation of proteins involved in endothelial proliferation and/or differentiation.
Cellular Location	Cytoplasm. Cytoplasm, cytoskeleton, microtubule organizing center,

	centrosome. Cytoplasm, perinuclear region. Golgi apparatus. Note=Colocalizes with FBXW8 at the Golgi apparatus in neurons; localization to Golgi is mediated by OBSL1. During mitosis, localizes to the mitotic apparatus (PubMed:24793695). CCDC8 is required for centrosomal location (PubMed:24793695)
Tissue Location	Highly expressed in fetal kidney and adult skeletal muscle. Also abundant in fetal brain, as well as in adult pancreas, kidney, placenta and heart. Detected in trophoblasts, lymphoblasts, osteoblasts, chondrocytes and skin fibroblasts

Background

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