

**SPG15 Antibody**  
**Catalog # ASC10851****Specification**

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**SPG15 Antibody - Product Information**

Application	WB, IF
Primary Accession	<a href="#">Q68DK2</a>
Other Accession	<a href="#">EAW80952</a> , <a href="#">119601358</a>
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	Predicted: 280 kDa; Observed: 260 kDa
Application Notes	KDa SPG15 antibody can be used for detection of SPG15 by Western blot at 1 - 2 µg/mL. For immunofluorescence start at 20 µg/mL.

**SPG15 Antibody - Additional Information**Gene ID **23503****Target/Specificity**

ZFYVE26; Multiple isoforms of SPG15 are known to exist.

**Reconstitution & Storage**

SPG15 antibody can be stored at 4°C for three months and -20°C, stable for up to one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. Antibodies should not be exposed to prolonged high temperatures.

**Precautions**

SPG15 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

**SPG15 Antibody - Protein Information****Name** ZFYVE26**Synonyms** KIAA0321**Function**

Phosphatidylinositol 3-phosphate-binding protein required for the abscission step in cytokinesis: recruited to the midbody during cytokinesis and acts as a regulator of abscission. May also be required for efficient homologous recombination DNA double-strand break repair.

**Cellular Location**

Cytoplasm, cytoskeleton, microtubule organizing center, centrosome. Midbody. Note=Localizes to the centrosome during all stages of the cell cycle. Recruited to the midbody during cytokinesis by KIF13A

### Tissue Location

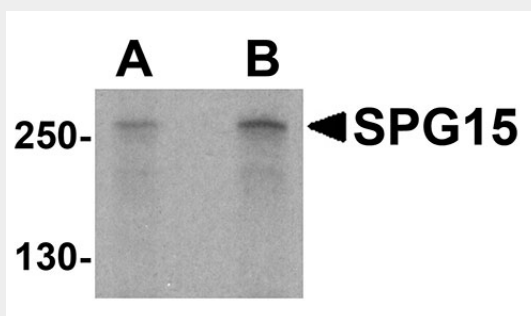
Strongest expression in the adrenal gland, bone marrow, adult brain, fetal brain, lung, placenta, prostate, skeletal muscle, testis, thymus, and retina. Intermediate levels are detected in other structures, including the spinal cord

### SPG15 Antibody - Protocols

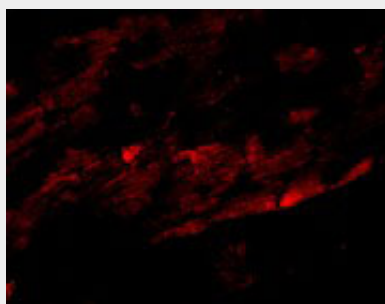
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

### SPG15 Antibody - Images



Western blot analysis of SPG15 in K562 cell lysate with SPG15 antibody at (A) 1 and (B) 2 µg/mL.



Immunofluorescence of SPG15 in Mouse Heart tissue with SPG15 antibody at 20 µg/mL.

### SPG15 Antibody - Background

SPG15 Antibody: Hereditary spastic paraplegias (HSPs) are genetically and phenotypically heterogeneous disorders. Spastic paraplegia with thinning of the corpus callosum (ARHSP-TCC) is a relatively frequent form of complicated hereditary spastic paraplegia in which mental retardation and muscle stiffness at onset are followed by slowly progressive paraparesis and cognitive deterioration. SPG15 is the second gene known to be responsible for ARHSP-TCC in the Italian population. Mutations in this gene are associated with autosomal recessive spastic paraplegia-15. SPG15 encodes a protein containing a FYVE zinc finger binding domain which is thought to target these proteins to membrane lipids through interaction with phospholipids in the membrane. SPG15

mRNA is widely distributed in human tissues, as well as in rat embryos, suggesting a possible role for this protein during embryonic development. SPG15 co-localizes partially with endoplasmic reticulum and endosome markers, suggesting a role in intracellular trafficking.

### **SPG15 Antibody - References**

Hughes CA, Byrne PC, Webb S, et al. SPG15, a new locus for autosomal recessive complicated HSP on chromosome 14q. *Neurology* 2001; 56:1230-3.

Denora PS, Muglia M, Casali C, et al. Spastic paraplegia with thinning of the corpus callosum and white matter abnormalities: further mutations and relative frequency in ZFYVE26/SPG15 in the Italian population. *J. Neurol. Sci.* 2009; 277:22-5.

Hanein S, Martin E, Boukhris A, et al. Identification of the SPG15 gene, encoding spastizin, as a frequent cause of complicated autosomal-recessive spastic paraplegia, including Kjellin syndrome. *Am. J. Hum. Genet.* 2008; 82:992-1002.

Boukhris A, Feki I, Denis E, et al. Spastic paraplegia 15: linkage and clinical description of three Tunisian families. *Mov. Disord.* 2008; 23:429-33.