10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

NAGA Polyclonal Antibody
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP57346

Specification

NAGA Polyclonal Antibody - Product Information

Application	**WB, IHC-P, IHC-F, IF, ICC**
Primary Accession	P17050
Reactivity	**Rat, Pig, Dog, Bovine**
Host	**Rabbit**
Clonality	**Polyclonal**
Calculated MW	**47 KDa**
Physical State	**Liquid**
Immunogen	**KLH conjugated synthetic peptide derived from human NAGA**
Epitope Specificity	**101-200/411**
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	**Lysosome.**
SUBUNIT	**Homodimer.**
DISEASE	**Schindler disease (SCHIND) [MIM:609241]: Form of NAGA deficiency characterized by early-onset neuroaxonal dystrophy and neurological signs (convulsion during fever, epilepsy, psychomotor retardation and hypotonia). NAGA deficiency is typically classified in three main phenotypes: NAGA deficiency type I (Schindler disease or Schindler disease type I) with severe manifestations; NAGA deficiency type II (Kanzazi disease or Schindler disease type II) which is mild; NAGA deficiency type III (Schindler disease type III) characterized by mild-to-moderate neurologic manifestations. NAGA deficiency results in the increased urinary excretion of glycopeptides and oligosaccharides containing alpha-N-acetylgalactosaminyl moieties. Inheritance is autosomal recessive. Note: The disease is caused by mutations affecting the gene represented in this entry. Ref.13 Ref.15 Kanzaki disease (KANZD) [MIM:609242]: Autosomal recessive disorder characterized by late-onset, angiokeratoma corporis diffusum and mild intellectual impairment.**

Important Note	**Note: The disease is caused by mutations affecting the gene represented in this entry.** **This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.**

Background Descriptions
NAGA encodes the lysosomal enzyme alpha-N-acetylgalactosaminidase, which cleaves alpha-N-acetylgalactosaminyl moieties from glycoconjugates. Mutations in NAGA have been identified as the cause of Schindler disease types I and II (type II also known as Kanzaki disease). [provided by RefSeq, Jul 2008]

NAGA Polyclonal Antibody - Additional Information

Gene ID 4668

Other Names
Alpha-N-acetylgalactosaminidase, 3.2.1.49, Alpha-galactosidase B, NAGA (HGNC:7631)

Dilution
WB~~1:1000<br \>IHC-P~~N/A<br \>IHC-F~~N/A<br \>IF~~1:50~200<br \>ICC~~N/A

Storage
Store at -20 ℃ 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 ℃.

NAGA Polyclonal Antibody - Protein Information

Name NAGA (HGNC:7631)

Function
Removes terminal alpha-N-acetylgalactosamine residues from glycolipids and glycopeptides. Required for the breakdown of glycolipids.

Cellular Location
Lysosome.

NAGA Polyclonal Antibody - Protocols

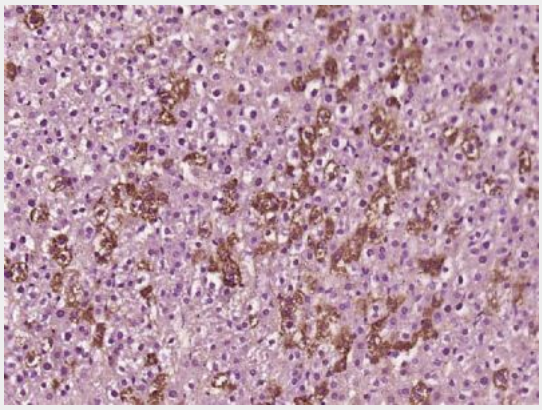
Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence

10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

- Immunoprecipitation
- Flow Cytomety
- Cell Culture

NAGA Polyclonal Antibody - Images



Paraformaldehyde-fixed, paraffin embedded (Rat liver); Antigen retrieval by boiling in sodium citrate buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minutes; Blocking buffer (normal goat serum) at 37°C for 30min; Antibody incubation with (NAGA) Polyclonal Antibody, Unconjugated (bs-19001R) at 1:500 overnight at 4°C, followed by a conjugated secondary (sp-0023) for 20 minutes and DAB staining.