

Anti-NAG-1 (C-terminal specific) (RABBIT) Antibody Peroxidase Conjugated
NAG-1 Antibody Peroxidase Conjugated
Catalog # ASR5824**Specification****Anti-NAG-1 (C-terminal specific) (RABBIT) Antibody Peroxidase Conjugated - Product Information**

Host	Rabbit
Conjugate	Peroxidase (Horseradish)
Target Species	Human
Reactivity	Human, Mouse
Clonality	Polyclonal
Application	WB, IHC, E, I, LCI
Application Note	Anti-NAG-1 (C-terminal specific) antibody has been tested by ELISA and is suitable for IHC and western blotting of human and mouse NAG-1 protein. For detection of NAG-1 in human serum, a sandwich ELISA is suggested using this antibody in combination with anti-NAG-1/GDF15 (N-terminal), H variant or D variant specific antibodies. Specific conditions for reactivity should be optimized by the end user. Expect bands in Western blots of approximately 14 and 28 kDa in size corresponding to NAG-1 monomer and dimer, respectively, using the appropriate cell lysate or extract.
Physical State	Lyophilized
Buffer	0.02 M Potassium Phosphate, 0.15 M Sodium Chloride, pH 7.2
Immunogen	Anti-NAG-1 (C-terminal specific) antibody was prepared by repeated immunizations with a synthetic peptide corresponding to a region near the carboxy terminal end of human NAG-1 protein. A residue of cysteine was added to facilitate coupling to KLH.
Reconstitution Volume	100 µL
Reconstitution Buffer	Restore with deionized water (or equivalent)
Stabilizer	10 mg/mL Bovine Serum Albumin (BSA) - Immunoglobulin and Protease free
Preservative	0.01% (w/v) Gentamicin Sulfate. Do NOT add Sodium Azide!

Anti-NAG-1 (C-terminal specific) (RABBIT) Antibody Peroxidase Conjugated - Additional Information

Gene ID 9518

Other Names

9518

Purity

Anti-NAG-1 (C-terminal specific) was affinity purified from monospecific antiserum by immunoaffinity chromatography. This antibody reacts with the C-terminus of endogenous NAG-1 protein from human and mouse tissues. A BLAST analysis suggests reactivity with NAG-1 from chimpanzee and macaque based on a 100% homology. Partial reactivity is expected against rat based on an 86% homology with the immunizing sequence. Cross-reactivity with NAG-1 from other sources has not been determined.

Storage Condition

Store vial at 4° C prior to restoration. For extended storage aliquot contents and freeze at -20° C or below. Avoid cycles of freezing and thawing. Centrifuge product if not completely clear after standing at room temperature. This product is stable for several weeks at 4° C as an undiluted liquid. Dilute only prior to immediate use.

Precautions Note

This product is for research use only and is not intended for therapeutic or diagnostic applications.

Anti-NAG-1 (C-terminal specific) (RABBIT) Antibody Peroxidase Conjugated - Protein Information

Name GDF15 {ECO:0000303|PubMed:23468844, ECO:0000312|HGNC:HGNC:30142}

Function

Hormone produced in response to various stresses to confer information about those stresses to the brain, and trigger an aversive response, characterized by nausea, vomiting, and/or loss of appetite (PubMed: 23468844, PubMed: 24971956, PubMed: 28846097, PubMed: 28846098, PubMed: 28846099, PubMed: 28953886, PubMed: 29046435, PubMed: 30639358, PubMed: 31875646, PubMed: 33589633, PubMed: 38092039). The aversive response is both required to reduce continuing exposure to those stresses at the time of exposure and to promote avoidance behavior in the future (PubMed: 30639358, PubMed: 33589633, PubMed: 38092039). Acts by binding to its receptor, GFRAL, activating GFRAL-expressing neurons localized in the area postrema and nucleus tractus solitarius of the brainstem (PubMed: 28846097, PubMed: 28846098, PubMed: 28846099, PubMed: 28953886, PubMed: 31535977). It then triggers the activation of neurons localized within the parabrachial nucleus and central amygdala, which constitutes part of the 'emergency circuit' that shapes responses to stressful conditions (PubMed: 28953886). The

GDF15-GFRAL signal induces expression of genes involved in metabolism, such as lipid metabolism in adipose tissues (PubMed:31402172). Required for avoidance behavior in response to food allergens: induced downstream of mast cell activation to promote aversion and minimize harmful effects of exposure to noxious substances (By similarity). In addition to suppress appetite, also promotes weight loss by enhancing energy expenditure in muscle: acts by increasing calcium futile cycling in muscle (By similarity). Contributes to the effect of metformin, an anti-diabetic drug, on appetite reduction and weight loss: produced in the kidney in response to metformin treatment, thereby activating the GDF15-GFRAL response, leading to reduced appetite and weight (PubMed:31875646, PubMed:37060902). The contribution of GDF15 to weight loss following metformin treatment is however limited and subject to discussion (PubMed:36001956). Produced in response to anticancer drugs, such as camptothecin or cisplatin, promoting nausea, vomiting and contributing to malnutrition (By similarity). Overproduced in many cancers, promoting anorexia in cancer (cachexia) (PubMed:32661391). Responsible for the risk of nausea and vomiting during pregnancy: high levels of GDF15 during pregnancy, mostly originating from the fetus, are associated with increased nausea and vomiting (PubMed:38092039). Maternal sensitivity to nausea is probably determined by pre-pregnancy exposure to GDF15, women with naturally high level of GDF15 being less susceptible to nausea than women with low levels of GDF15 before pregnancy (PubMed:38092039). Promotes metabolic adaptation in response to systemic inflammation caused by bacterial and viral infections in order to promote tissue tolerance and prevent tissue damage (PubMed:31402172). Required for tissue tolerance in response to myocardial infarction by acting as an inhibitor of leukocyte integrin activation, thereby protecting against cardiac rupture (By similarity). Inhibits growth hormone signaling on hepatocytes (By similarity).

Cellular Location

Secreted Note=Secreted in the plasma.

Tissue Location

Detected in plasma (at protein level) (PubMed:28572090, PubMed:29046435). Highly expressed in placenta, with lower levels in prostate and colon and some expression in kidney (PubMed:37060902, PubMed:9348093).

Anti-NAG-1 (C-terminal specific) (RABBIT) Antibody Peroxidase Conjugated - 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](#)

Anti-NAG-1 (C-terminal specific) (RABBIT) Antibody Peroxidase Conjugated - Images

Anti-NAG-1 (C-terminal specific) (RABBIT) Antibody Peroxidase Conjugated - Background

Non-steroidal anti-inflammatory drug (NSAID) activated gene (NAG-1) is a member of the

transforming growth factor-beta (TGF-beta) superfamily. NAG-1 is also known as Macrophage Inhibitory Cytokine-1 (MIC-1), Growth Differentiation Factor 15 (GDF15), Placental Bone Morphogenetic Protein (PLAB), or Prostate Derived Factor (PDF). NAG-1 is expressed in human placenta, prostate and colon. It possesses antitumorigenic and proapoptotic activities. NAG-1 expression is dramatically increased in inflammation, injury and malignancy. Increase of NAG-1 expression is a feature of many cancers including breast, colon, pancreas and prostate. In a number of studies, NAG-1 expression was increased by a number of NSAIDs. This increase in expression may correlate with the chemopreventive effect NSAIDs seem to have with certain cancers. NAG-1 expression is also induced by PPAR gamma ligands and by several dietary compounds such as conjugated linoleic acids (CLAs), naturally occurring fatty acids in ruminant food products, indoles, epicatechin gallate, and genistein. Induced expression of NAG-1 results in stimulation of apoptosis and inhibition of cell growth. Inhibition of NAG-1 induced expression by small interference RNA (siRNA) results in repression of induced apoptosis. NAG-1 expression is regulated by a number of transcription factors such as ERG-1 and Sp1. EGR-1 may be necessary for NSAID-induced NAG-1 expression. The study of expression of NAG-1 proteins, including variants, is important to define their potential role as serum biomarkers for cancer diagnosis, treatment monitoring, epidemiology study, and nutrition surveys.