

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse Fetuin A/AHSG in direct ELISAs and Western blots. In these formats, approximately 5% cross-reactivity with recombinant human Fetuin A/AHSG is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse Fetuin A/AHSG (R&D Systems, Catalog # 1563-PI) Ala19-Ile345 Accession # NP_038493
Conjugate	Alexa Fluor 488 Excitation Wavelength: 488 nm Emission Wavelength: 515-545 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

Western Blot Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

Mouse Fetuin A, also known as α_2 -Heremans-Schmid glycoprotein, is encoded by the AHSG gene. It has been also called "countertrypsin" because of its ability to inhibit trypsin (1). It is a major plasma protein and a member of the cystatin superfamily of protease inhibitors (2, 3). It is expressed by hepatocytes, the principal cell source, and by monocyte/macrophages (4). The major form of plasma Fetuin A corresponds to two disulfide bond-linked chains derived from the single chain (5). Fetuin-A has a number of functions. It is a negative acute-phase protein with normal circulating levels in adults (300-600 µg/mL), which fall significantly (30-50%) during injury and infection (5). It enhances entry of cationic inhibitors into macrophages (6). It inhibits both insulin receptor autophosphorylation and undesirable calcification (7, 8).

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