Human Follistatin-related Gene Protein/FLRG Antibody

Antigen Affinity-purified Polyclonal Goat IgG

Catalog Number: AF1288

DESCRIPTION

Species Reactivity	Human
Specificity	Detects Follistatin-related Gene Protein/FLRG in direct ELISAs and Western blots. In direct ELISAs, approximately 40% cross-reactivity with recombinant mouse FLRG is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human FLRG Met27-Val263 Accession # O95633
Endotoxin Level	<0.10 EU per 1 µg of the antibody by the LAL method.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

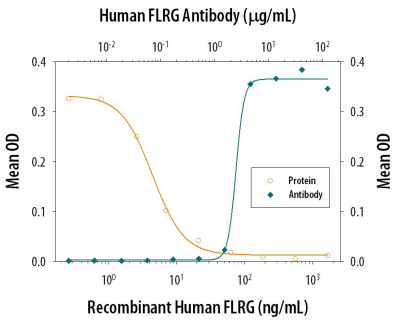
APPLICATIONS

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.*

	Recommended Concentration	Sample
Western Blot	0.1 µg/mL	Recombinant Human Follistatin-related Gene Protein/FLRG (Catalog # 1288-F3)
Neutralization	Measured by its ability to neutralize Follistatin-related Gene Protein/FLRG inhibition of Activin A-dependent hemoglobin expression in the K562 human chronic myelogenous leukemia cell line. The Neutralization Dose (ND_{50}) is typically 1-4 µg/mL in the presence of 0.1 µg/mL Recombinant Human Follistatin-related Gene Protein/FLRG and 7.5 ng/mL Recombinant Human/Mouse/Rat Activin A.	

DATA

Neutralization



Follistatin-related Gene Protein/FLRG Inhibition of Activin A-induced Hemoglobin Expression and Neutralization by Human Follistatin-related Gene Protein/FLRG Antibody. Recombinant Human Follistatin-related Gene Protein/FLRG (Catalog # 1288-F3) inhibits Recombinant Human/Mouse/Rat Activin A (Catalog # 338-AC) induced hemoglobin expression in the K562 human chronic myelogenous leukemia cell line in a dose-dependent manner (orange line), as measured by the psuedoperoxidase activity. Inhibition of Recombinant Human/Mouse/Rat Activin A (7.5 ng/mL) activity elicited by Recombinant Human Follistatin-related Gene Protein/FLRG (0.1 µg/mL) is neutralized (green line) by increasing concentrations of Goat Anti-Human Follistatin-related Gene Protein/FLRG Antigen Affinity-purified Polyclonal Antibody (Catalog # AF1288). The ND_{50} is typically 1-4 µg/mL.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month from date of receipt, 2 to 8 °C, reconstituted. ● 6 months from date of receipt, -20 to -70 °C, reconstituted.

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BACKGROUND

Follistatin-related gene protein (FLRG), also known as follistatin-like 3 (FSTL3) is a glycoprotein belonging to the follistatin-module protein family. Human FLRG cDNA encodes a 263 amino acid (aa) residue protein with a putative 26 aa signal peptide, an N-terminal domain, two cysteine-rich follistatin-like domains (FS) and a C-terminal acidic domain. Compared to follistatin, FLRG lacks the third FS domain found in follistatin. In addition, FLRG also lacks the heparin-binding domain found within the first amino-terminal FS domain of follistatin. Mouse and human FLRG share approximately 83% aa sequence homology. Like follistatin, FLRG has been shown to bind and inhibit the activities of TGF-β family ligands including activin, BMP-2, -6, -7 and GDF-8/myostatin. While both FLRG and follistatin are located in a wide and overlapping range of adult and fetal tissue, their sites of peak expression differ: FLRG most highly in heart, lung, kidney, placenta and testis, while follistatin is highest in ovary and pituitary. The expression of FLRG is upregulated by TGF-β and activin signaling through Smad proteins. Although FLRG is a secreted protein in many cell types, it has also been localized to the nuclear compartment in HeLa, 293 and CHO cells (1-5).

References:

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2. Sidis, Y. *et al.* (2002) Endocrinology **143**:1613.
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4. Hill, J. *et al.* (2002) J. Biol. Chem. **277**:40735.
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