

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human MIS/AMH in direct ELISAs. In direct ELISAs, no cross-reactivity with recombinant rat MIS/AMH is observed.
Source	Monoclonal Mouse IgG ₁ Clone # 805531
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human MIS/AMH Leu19-Gln450 (predicted) Accession # P03971
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.

Immunocytochemistry 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

Müllerian inhibiting substance (MIS), also named anti-Müllerian hormone (AMH), is a tissue-specific TGF- β superfamily growth factor. Its expression is restricted to the Sertoli cells of fetal and postnatal testis, and to the granulosa cells of postnatal ovary (1). The human MIS gene encodes a 553 amino acid residue (aa) prepropeptide containing a signal a sequence (1-24), a pro-region (25-455), and the carboxyl-terminal bioactive protein (446-553) (2-4). MIS is synthesized and secreted as a disulfide-linked homodimeric pro-protein. Proteolytic cleavage is required to generate the N-terminal pro-region and the C-terminal bioactive protein, which remain associated in a non-covalent complex. Recombinant C-terminal MIS has been shown to be bioactive. However, the complex with the N-terminal pro-region showed enhanced activity (3, 5). The C-terminal region contains the seven canonical cysteine residues found in TGF- β superfamily members. These cysteine residues are involved in inter- and intra-molecular disulfide bonds, which forms the cysteine knot structure. Human and mouse MIS share 73% and 90% aa sequence identity in their pro-region and C-terminal region, respectively. MIS induces Mullerian duct (female reproductive tract) regression during sexual differentiation in the male embryo (6). Postnatally, MIS has been shown to regulate gonadal functions (1). MIS inhibits Leydig cell proliferation and is a regulator of the initial and cyclic recruitment of ovarian follicles. MIS has also been found to have anti-proliferative effects on breast, ovarian and prostate tumor cells (7-9). Like other TGF- β superfamily members, MIS signals via a heteromeric receptor complex consisting of a type I and a type II receptor serine/threonine kinase. Depending on the cell context, different type I receptors (including Act RIA/ALK2, BMP RIA/ALK3, and BMP RIB/ALK6) that are shared by other TGF- β superfamily members, have been implicated in MIS signaling (10-12). In contrast, the type II MIS receptor (MIS RII) is unique and does not bind other TGF- β superfamily members. Upon ligand binding, MIS RII recruits the non-ligand binding type I receptor into the complex, resulting in phosphorylation the BMP-like signaling pathway effector proteins Smad1, Smad5, and Smad 8 (10-12).

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