

## Rat MIS RII Antibody

Antigen Affinity-purified Polyclonal Goat IgG Catalog Number: AF1618

DESCRIPTION			
Species Reactivity	Rat		
Specificity	Detects rat MIS RII in direct ELISAs and Western blots. In direct ELISAs, less than 1% cross-reactivity with recombinant human (rh) TGF-β RII, rhTGF-β RIII, recombinant mouse (rm) TGF-β RI, and rmTGF-β RII is observed.		
Source	Polyclonal Goat IgG		
Purification	Antigen Affinity-purified		
Immunogen	S. frugiperda insect ovarian cell line Sf 21-derived recombinant rat MIS RII Pro19-Pro144 Accession # Q62893		
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.		
APPLICATIONS Please Note: Optimal dilution	ons should be determined by each I	aboratory for each application	on. General Protocols are available in the Technical Information section on our website.
		Recommended Concentration	Sample
Western Blot		0.1 μg/mL	Recombinant Rat MIS RII Fc Chimera (Catalog # 1618-MR)
Immunohistochemistry		5-15 μg/mL	Perfusion fixed frozen sections of rat ovary
Blockade of Receptor-ligand Interaction		In a functional ELISA, 0.05-0.2 μg/mL of this antibody will block 50% of the binding of 50 ng/mL of Recombinant Rat MIS RII Fc Chimera (Catalog # 1618-MR) to immobilized Recombinant Human MIS/AMH (Catalog # 1737-MS) coated at 3 μg/mL (100 μL/well). At 2 μg/mL, this antibody will block >90% of the binding.	
PREPARATION AND S	STORAGE		
Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.		

## BACKGROUND

Shipping Stability & Storage

Müllerian inhibiting substance (MIS), also named anti-Müllerian hormone (AMH) is a tissue-specific TGF-β superfamily growth factor. Its expression is restricted to fetal testis, plus postnatal testis and ovary (1). MIS induces Mullerian duct (female reproductive tract) regression during sexual differentiation in the male embryo and has been shown to have a regulatory role in gonads postnatally (1). 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, can be utilized for MIS signaling (1). In contrast, the type II MIS receptor (MIS RII) is unique and does not bind other TGF-β superfamily members (1, 2). 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 Smad8 (1).

The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

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, 2 to 8 °C under sterile conditions after reconstitution.

6 months, -20 to -70 °C under sterile conditions after reconstitution.

The gene for rat MIS RII was isolated separately by two groups working from Sertoli cell and fetal ovary cDNA libraries (3, 4). MIS RII comprises a 557 amino acid (aa) residue type I transmembrane protein with a putative 17 aa signal peptide. Mature MIS RII has a 127 aa cysteine-rich extracellular domain containing 2 potential N-glycosylation sites, a 21 aa transmembrane domain, and a 392 aa cytoplasmic region with a serine/threonine kinase domain (3, 4). Rat MIS RII shares 95% and 82% aa sequence identity with the mouse and human homologues, respectively (5). MIS RII is expressed in the mesenchymal cells surrounding the Mullerian ducts during embryonic development. Postnatally, it is expressed in uterine tissues and rodent Leydig cells, and coexpressed with MIS in the testicular Sertoli and ovarian granulosa cells (1, 6). The expression of MIS RII in the Mullerian mesenchyme is regulated by Wnt7a signaling from nearby epithelium through the canonical Wnt pathway. Wnt7a mutant mice do not express MIS RII, and do not experience Mullerian duct regression (7).

## References:

- 1. Josso, N and N. diClemente (2003) Trends Endo. Met. 14:91.
- 2. Mishna, Y. et al. (1999) Endocrinology 140:2084.
- 3. Baarends, W. *et al.* (1994) Development **120**:189.
- 4. di Clement, N. et al. (1994) Mol. Endocrinol. 8:1006.
- 5. Mishina, Y. et al. (1997) Biochem. Biophys. Res. Comm. 237:741.
- 6. Teixeira, J. et al. (1996) Endocrinology 137:160.
- 7. Hossain, A. and G. Saunders (2003) J. Biol. Chem. 278:26511.

