

Recombinant human AMH

Catalog No: #AG0035

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Description

Product Name	Recombinant human AMH
Host Species	HEK293
Purification	> 95% by Tris-Bis PAGE; > 95% by SEC-HPLC
Immunogen Description	Ala453-Arg560
Target Name	AMH
Other Names	AMH; MIF; MIS; Mullerian hormone; muellerian-inhibiting factor; muellerian-inhibiting substance; Mullerian hormone; Mullerian inhibiting factor; Mullerian inhibiting substance
Accession No.	Uniprot:P03971 Gene ID:268
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GeneID	268
Target Species	human
Calculated MW	5.7 KDa
Tag Info	additional amino acid free
Formulation	0.22 µm filtered solution of PBS, pH 7.4.
Storage	Aliquot and store at -80°C. Avoid repeated freeze/thaw cycles.

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 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 of the BMP-like signaling pathway effector proteins Smad1, Smad5 and Smad 8 (10,12).

Note: This product is for in vitro research use only