

Product datasheet

Human Wnt7 α peptide ab113305

Overview

Product name Human Wnt7a peptide

Description

Nature Recombinant

Amino Acid Sequence

Species Human

Specifications

Our [Abpromise guarantee](#) covers the use of **ab113305** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Applications Blocking - Blocking peptide for Anti-Wnt7a antibody ([ab100792](#))

Form Liquid

Additional notes This is the blocking peptide for [ab100792](#).

- First try to dissolve a small amount of peptide in either water or buffer. The more charged residues on a peptide, the more soluble it is in aqueous solutions.
- If the peptide doesn't dissolve try an organic solvent e.g. DMSO, then dilute using water or buffer.
- Consider that any solvent used must be compatible with your assay. If a peptide does not dissolve and you need to recover it, lyophilise to remove the solvent.
- Gentle warming and sonication can effectively aid peptide solubilisation. If the solution is cloudy or has gelled the peptide may be in suspension rather than solubilised.
- Peptides containing cysteine are easily oxidised, so should be prepared in solution just prior to use.

Preparation and Storage

Stability and Storage Shipped at 4°C. Upon delivery aliquot and store at -20°C or -80°C. Avoid repeated freeze / thaw cycles.

Information available upon request.

General Info

Function	Ligand for members of the frizzled family of seven transmembrane receptors. Probable developmental protein. Signaling by Wnt-7a allows sexually dimorphic development of the mullerian ducts.
Tissue specificity	Expression is restricted to placenta, kidney, testis, uterus, fetal lung, and fetal and adult brain.
Involvement in disease	Defects in WNT7A are the cause of limb/pelvis-hypoplasia/aplasia syndrome (LPHAS) [MIM:276820]; also known as absence of ulna and fibula with severe limb deficiency. LPHAS is a limb-malformation disorder characterized by various degrees of limb aplasia/hypoplasia and joint dysplasia. Defects in WNT7A are a cause of Fuhrmann syndrome (FUHRS) [MIM:228930]; also known as fibular aplasia or hypoplasia femoral bowing and poly- syn- and oligodactyly. Fuhrmann syndrome is a distinct limb-malformation disorder characterized also by various degrees of limb aplasia/hypoplasia and joint dysplasia.
Sequence similarities	Belongs to the Wnt family.
Cellular localization	Secreted > extracellular space > extracellular matrix.

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