

Recombinant human Wnt7α protein ab116171

1 References

Description

Product name	Recombinant human Wnt7a protein
Biological activity	Determined by its ability to decrease alkaline phosphatase activity in CCL-226 cells when treated with 25 ng/ml of Murine Wnt-3a.
Purity	> 80 % SDS-PAGE. The purity of ab116171 is greater than 80% by SDS-PAGE gel and HPLC analyses.
Endotoxin level	< 1.000 Eu/μg
Expression system	HEK 293 cells
Accession	<u>O00755</u>
Protein length	Full length protein
Animal free	No
Nature	Recombinant
Species	Human
Sequence	LGASII CNKI PGLAPRQRAI CQSRPD AIV IGEGSQMGLD ECQFQFRNGR WNC SALGERT VFGKELKVGS REAAFTYAII AAGVAHAITA ACTQGNLSDC GCDKEKQGQY HRDEGWKWGG CSADIRYGIG FAKVFVDARE IKQNARTLMN LHNNEAGRKI LEENMKLECK CHGVSGSCTT KTCWTTLPQF RELGYVLKDK YNEAVHVEPV RASRNKRPTF LKI KPLSYR KPM DTDLVYI EKSPNYCEED PVTGSGVTQG RACNKTAPQA SGCDLMCCGR GYNTHQYARV WQCNC FHC CYV KCNTCSE RTE MYTCK
Predicted molecular weight	36 kDa
Amino acids	32 to 349

Specifications

Our **Abpromise guarantee** covers the use of **ab116171** in the following tested applications.

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

Applications	SDS-PAGE
	Functional Studies

Form Lyophilized

Preparation and Storage

Stability and Storage Shipped at 4°C. Store at -20°C.
This product is an active protein and may elicit a biological response in vivo, handle with caution.

Reconstitution Reconstitute to a concentration of 0.1 mg/ml.

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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