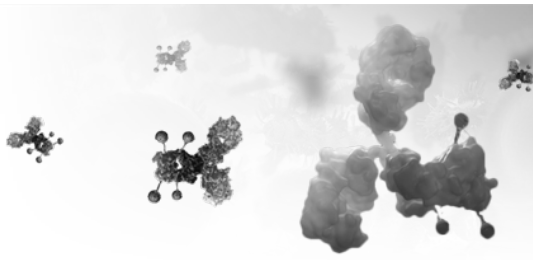

Mouse Anti-Human DLL1 monoclonal antibody, clone NN18 (CABT-ZB901)

This product is for research use only and is not intended for diagnostic use.

PRODUCT INFORMATION

Specificity	It reacts with Human DLL1 It has no cross-reactivity in ELISA with Mouse DLL4/Delta-4, Mouse DLL1/Delta-1, Human DLL4/Delta-4.
Target	DLL1
Immunogen	Recombinant Human DLL1 protein
Isotype	IgG
Source/Host	Mouse
Species Reactivity	Human
Clone	NN18
Purification	Protein A purified
Conjugate	Unconjugated
Applications	ELISA, ELISA(det) We recommend the following for sandwich ELISA (Capture - Detection): CABT-ZB538 - CABT-ZB901 This antibody will detect DLL1 in antibody pair set. [ABPR-ZB114]
Preparation	This antibody was produced from a hybridoma resulting from the fusion of a mouse myeloma with B cells obtained from a mouse immunized with purified, recombinant Human DLL1 / Delta-1. The IgG fraction of the cell culture supernatant was purified by Protein A affinity chromatography.
Format	Purified, Liquid

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Concentration	Lot specific
Size	50 µL, 100 µL, 200 µL, 1 mL
Buffer	PBS
Preservative	None
Storage	This antibody can be stored at 2°C-8°C for one month without detectable loss of activity. Antibody products are stable for twelve months from date of receipt when stored at -20°C to -80°C. Preservative-Free. Avoid repeated freeze-thaw cycles.
Ship	Wet ice

BACKGROUND

Introduction	Delta-like protein 1(DLL1), also known as Delta1, a single-pass type I membrane protein which contains one DSL domain and eight EGF-like domains, acts as a ligand for Notch receptors, and positively regulates T-cell development. DLL1 is proteolytically processed in a similar manner to the Notch receptor, and it has been speculated to participate in bidirectional signaling. The proteolytic processing of DLL1 helps achieve an asymmetry in Notch signaling in initially equivalent myogenic cells and helps sustain the balance between differentiation and self-renewal. Interactions between DLL1 and Notch in trans activate the Notch pathway, whereas DLL1 binding to Notch in cis inhibits Notch signaling. DLL1 undergoes proteolytic processing in its extracellular domain by ADAM10. It had been demonstrated that DLL1 represents a substrate for several other members of the ADAM family. In co-transfected cells, DLL1 is constitutively cleaved by ADAM12, and the N-terminal fragment of DLL1 is released to medium. ADAM12-mediated cleavage of DLL1 is cell density-dependent, takes place in cis orientation, and does not require the presence of the cytoplasmic domain of ADAM12. Full-length DLL1, but not its N- or C-terminal proteolytic fragment, co-immunoprecipitates with ADAM12. By using a Notch reporter construct, we show that DLL1 processing by ADAM12 increases Notch signaling in a cell-autonomous manner. Furthermore, ADAM9 and ADAM17 have the ability to process DLL1. In contrast, ADAM15 does not cleave DLL1, although the two proteins still co-immunoprecipitate with each other. During fetal development, DLL1 is an essential Notch ligand in the vascular endothelium of large arteries to activate Notch1 and maintain arterial identity. DLL1-Notch signaling was required for VEGF receptor expression in fetal arteries.
Keywords	DLL1; delta-like 1 (Drosophila); DL1; Delta

GENE INFORMATION

Synonyms	DLL1; delta-like 1 (Drosophila); DL1; Delta; DELTA1; delta-like protein 1; H-Delta-1; drosophila Delta homolog 1

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UniProt ID	O00548

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