

Anti-DLL4 Antibody (40557)

Product Details

Ig Type:	Rabbit IgG
Reactivity:	Human
Conjugation:	Unconjugated
Clone:	40557
Purification:	Protein A

Applications

Application:	ELISA,ELISA(Det)
Recommended	ELISA: 1:5000-1:10000; ELISA(Det): 1:1000-1:10000

Properties

Stability & Storage:	Store at 2°C-8°C for 1 month. Store at -20°C or -80°C for 12 months. Avoid repeated freeze-thaw cycles. Preservative-Free.
Shipping:	Shipping with blue ice.

Antigen Details

Immunogen:	Recombinant Protein: Human DLL4 protein (TMPY-01321)
Antigen Species:	Human
Synonyms:	δ -like 4 (Drosophila);delta-like 4 (Drosophila);Delta-like 4;h δ 2;hdelta2; δ -like 4
Biology Area:	Cancer Drug Targets, Notch Pathway

Research Background

Delta-like protein 4 (DLL4, Delta4), a type I membrane-bound Notch ligand, is one of five known Notch ligands in mammals and interacts predominantly with Notch 1, which has a key role in vascular development. Recent studies yield substantial insights into the role of DLL4 in angiogenesis. DLL4 is induced by vascular endothelial growth factor (VEGF) and acts downstream of VEGF as a 'brake' on VEGF-induced vessel growth, forming an autoregulatory negative feedback loop inactivating VEGF. DLL4 is downstream of VEGF signaling and its activation triggers a negative feedback that restrains the effects of VEGF. Attenuation of DLL4/Notch signaling results in chaotic vascular network with excessive branching and sprouting. DLL4 is widely distributed in tissues other than vessels including many malignancies. Furthermore, the molecule is internalized on binding its receptor and often transported to the nucleus. In pathological conditions, such as cancer, DLL4 is up-regulated strongly in the tumour vasculature. Blockade of DLL4-mediated Notch signaling strikingly increases nonproductive angiogenesis, but significantly inhibits tumor growth in preclinical mouse models. In preclinical studies, blocking of DLL4/Notch signaling is associated with a paradoxical increase in tumor vessel density, yet causes marked growth inhibition due to functionally defective vasculature. Thus, DLL4 blockade holds promise as an additional strategy for angiogenesis-based cancer therapy.

Inhibitor · Natural Compounds · Compound Libraries · Recombinant Proteins

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