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InnoCyto Inc.

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Anti-Human VEGF (Aflibercept Biosimilar)

Catalog Number:	500401, 500402, 500403
Size:	1 mg, 5 mg, 20 mg
Target Name:	VEGF
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	Aflibercept
Application:	Neutralization, Intracellular Flow cytometry, animal model study
Reactivity:	Human
Format:	Liquid
Product Description:	Anti-Human VEGF (Aflibercept Biosimilar)
Isotype:	Human IgG1-Fc
Clonality:	Recombinant
Species specificity:	Human
Purity:	>95% by reducing SDS-PAGE
Grade:	In vivo
Min Sample Size:	1 mg
Storage Conditions:	4ºC
Maximal Shelf Life:	12 months
Synonyms:	VEGFR, VEGFR1, VEGFR2

BACKGROUND INFORMATION

Aflibercept is a recombinant fusion protein specifically engineered to act as a decoy receptor for vascular endothelial growth factors (VEGFs). Structurally, the molecule combines the ligand-binding domains of two distinct human receptors, VEGFR-1 (Flt-1) and VEGFR-2 (KDR), fused to the Fc portion of human immunoglobulin G1 (IgG1). More precisely, Aflibercept contains the extracellular domain 2 of VEGFR-1 and domain 3 of VEGFR-2, which together form a high-affinity binding site for VEGF ligands. The resultant fusion protein has a molecular mass of approximately 115 kilodaltons (kDa) and is produced in mammalian expression systems such as Chinese Hamster Ovary (CHO) cells to ensure proper folding, disulfide bonding, and glycosylation.

The Fc region of Aflibercept serves several critical structural and functional purposes. It provides molecular stability, confers dimerization through disulfide bridges to form a Y-shaped homodimer, and extends the molecule's circulatory half-life via interactions with neonatal Fc receptors (FcRn). The fusion of the VEGF-binding domains to this Fc backbone effectively produces a hybrid receptor that retains the natural ligand-binding specificity of VEGFR molecules while gaining the pharmacokinetic benefits typical of antibodies. Each Aflibercept dimer can bind multiple molecules of VEGF, acting as a soluble trap with high binding affinity

in the picomolar range.

Functionally, Aflibercept acts as a potent VEGF antagonist by sequestering VEGF-A, VEGF-B, and placental growth factor (PlGF) before these ligands can interact with their receptors on endothelial cells. By intercepting VEGF signaling, the molecule effectively prevents receptor dimerization and downstream activation of signaling pathways such as MAPK and PI3K-AKT, which are involved in endothelial cell proliferation, survival, and permeability. This molecular sequestration mechanism makes Aflibercept an important biological model for studying angiogenesis regulation, receptor-ligand affinity optimization, and engineered protein-receptor interactions in molecular biology and biochemistry.

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