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Anti-Human VEGFR-2 (Ramucirumab Biosimilar)

Catalog Number:	505001, 505002, 505003
Size:	1 mg, 5 mg, 20 mg
Target Name:	VEGFR2, VEGF-R2, KDR, Tyrosin kinase growth factor receptor, homolog of FLK1 (FLK1), VEGFR
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	Ramucirumab
Application:	Flow cytometry, animal model study
Reactivity:	Human
Format:	Liquid
Product Description:	Anti-Human VEGFR-2 (Ramucirumab Biosimilar)
Isotype:	Human IgG1
Clonality:	Recombinant
Immunogen:	Human VEGFR2
Species specificity:	Human
Purity:	>95% by reducing SDS-PAGE
Grade:	In vivo
Storage Conditions:	4ºC
Maximal Shelf Life:	12 months
Antibody Type:	Recombinant
Reactivity:	Human

BACKGROUND INFORMATION

Ramucirumab is a fully human monoclonal antibody that belongs to the immunoglobulin G1 (IgG1) subclass and is engineered to bind with high specificity to vascular endothelial growth factor receptor 2 (VEGFR-2), also known as kinase insert domain receptor (KDR). Structurally, Ramucirumab is a glycoprotein with an approximate molecular weight of 147 kilodaltons (kDa). The molecule consists of two identical heavy chains and two identical light chains interconnected by disulfide bonds, forming the canonical Y-shaped antibody configuration typical of IgG molecules. It is produced using recombinant DNA technology in mammalian expression systems such as Chinese Hamster Ovary (CHO) cells to ensure proper folding, disulfide linkage formation, and glycosylation compatible with human proteins.

The variable regions (VH and VL) of Ramucirumab define its antigen-binding site, composed of complementarity-determining regions (CDRs) that mediate specific, high-affinity recognition of the extracellular domain of VEGFR-2. This region corresponds to the receptor’s ligand-binding site for vascular endothelial growth factor A (VEGF-A), VEGF-C, and VEGF-D. By binding to this epitope,

Ramucirumab sterically blocks the interaction between VEGFR-2 and its ligands, preventing receptor dimerization and autophosphorylation events required for downstream signaling activation. This inhibition disrupts intracellular pathways, primarily the MAPK (mitogen-activated protein kinase) and PI3K-AKT cascades, that regulate endothelial cell proliferation, migration, and survival in experimental systems investigating angiogenic control.

The Fc (fragment crystallizable) region of Ramucirumab is derived from human IgG1, providing structural stability and an extended half-life through neonatal Fc receptor (FcRn)–mediated recycling. It also permits limited engagement with Fc gamma receptors (FcγRs), enabling potential antibody-dependent cellular cytotoxicity (ADCC) under certain conditions, though this is not the primary mechanism. Overall, Ramucirumab exemplifies rational antibody design, combining precise receptor targeting, stable IgG architecture, and defined signaling blockade, to model ligand–receptor regulation and angiogenic signaling mechanisms at the molecular level.

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