Leinco Technologies, Inc.
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Anti-Mouse CD309 (VEGFR2) [Clone DC101] — Purified *in vivo* GOLD™ Functional Grade

Hybridoma Monoclonal Antibody

Product Information

Product No.:	V173
Clone:	DC101
Isotype:	Rat Rat IgG1 κ
Storage:	Sterile 2-8°C

Product Description

Specificity:
DC101 activity is directed against VEGFR-2.

Antigen Distribution:
VEGFR-2 is widely expressed by vascular endothelial cells, some vascular tumors, carcinomas, malignant melanomas, and lymphomas. Certain leukemia cells express functional VEGFR on the cell surface.

Background:
Vascular endothelial growth factors (VEGF) and VEGF receptors (VEGFR) play an essential role in angiogenesis[1]. There are three VEGFRs: VEGFR-1, VEGFR-2, and VEGFR-3. VEGFR-1 and VEGFR-2 are responsible for angiogenesis, and VEGFR-3 affects lymphogenesis. In the pathogenesis of diseases including diabetes mellitus, rheumatoid arthritis, and cancer, new blood vessel formation is highjacked. Changes at the VEGF/VEGFR-2 axis are particularly potent at allowing VEGF-induced proliferation, migration, and vascular endothelial cell differentiation during tumor angiogenesis. Additionally, VEGFR-2 is upregulated in tumor vascular endothelial cells, and VEGF levels are associated with poor prognosis and resistance to chemotherapy. Consequently, the VEGF/VEGFR axis is a prime anti-cancer target.

DC101 greatly reduces melanoma tumor growth and cell proliferation in murine mouse models without adverse effects as well as promotes tumor vessel normalization[2]. Additionally, DC101 therapy enhances immune cell penetration of melanoma cells by increasing the proportion of $CD19^+$ B cells, $CD11c^+$ dendritic cells, and $CD3^+$ and $CD8^+$ T cells. DC101 treatment also increases expression of PD-1 and PD-L1 in $CD45^+$ immune cells and tumors. Additionally, DC101 directly inhibits angiogenesis *in vivo*, and, in tumors, reduces xenograft tumor growth, decreases endothelial cells and microvessel density, and increases tumor cell apoptosis[3].

DC101 binds to an extracellular, ligand-binding domain on the amino-terminal of VEGFR-2, thereby blocking ligand binding and preventing $VEGF_{165}$-induced receptor phosphorylation[4]. DC101 has been

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used in Cy5.5-, FITC, and HYNIC-labeled chitosan conjugates to study VEGFR-2 expression in ischemia[5].

Known Reactivity Species:
Mouse

Format:
in vivo GOLD™, Purified in vivo Functional Grade

Immunogen:
Recombinant full-length Mouse VEGFR2 protein

Formulation
This monoclonal antibody is aseptically packaged and formulated in 0.01 M phosphate buffered saline (150 mM NaCl) PBS pH 7.2 - 7.4 with no carrier protein, potassium, calcium or preservatives added. Due to inherent biochemical properties of antibodies, certain products may be prone to precipitation over time. Precipitation may be removed by aseptic centrifugation and/or filtration.

Purity
≥95% monomer by analytical SEC, >95% by SDS Page

Endotoxin
< 1.0 EU/mg as determined by the LAL method

Storage and Stability
Functional grade preclinical antibodies may be stored sterile as received at 2-8°C for up to one month. For longer term storage, aseptically aliquot in working volumes without diluting and store at ≤ -70°C. **Avoid Repeated Freeze Thaw Cycles.**

Product Preparation
Functional grade preclinical antibodies are manufactured in an animal free facility using *in vitro* cell culture techniques and are purified by a multi-step process including the use of protein A or G to assure extremely low levels of endotoxins, leachable protein A or aggregates.
Other Applications Reported in Literature:

FA,
WB

Country of Origin
USA

References
1. Spratlin J. Curr Oncol Rep. 13(2):97-102. 2011.
2. Wang Z, Shi X, Zhao Y, et al. Biochem Biophys Res Commun. 661:10-20. 2023.
3. Prewett M, Huber J, Li Y, et al. Cancer Res. 59(20):5209-5218. 1999.
4. Patent EP1602668A1:

https://patentimages.storage.googleapis.com/10/da/cb/f945064c422659/EP1602668A1.pdf
5. Lee CM, Kim EM, Cheong SJ, et al. J Biomed Mater Res A. 92(4):1510-1517. 2010.
6. Rockwell P, Neufeld G, Glassman A, et al. Mol Cell Differ. 3(1): 91–109. 1995.

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