

DESCRIPTION

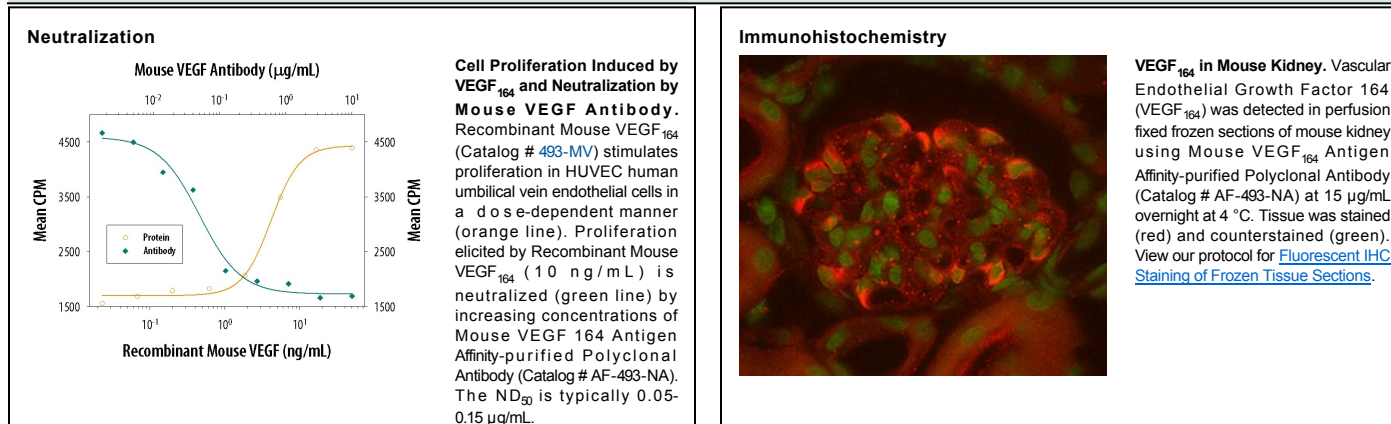
Species Reactivity	Mouse
Specificity	Detects mouse VEGF in ELISAs and Western blots. In sandwich immunoassays, less than 0.04% cross-reactivity with recombinant human (rh) VEGF ₁₂₁ , rhVEGF ₁₆₅ , and rhVEGF/PlGF is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	<i>S. frugiperda</i> insect ovarian cell line Sf 21-derived recombinant mouse VEGF ₁₆₄ (R&D Systems, Catalog # 493-MV) Ala27-Arg190 Accession # AAA40547
Endotoxin Level	<0.10 EU per 1 µg of the antibody by the LAL method.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 µm filtered solution in PBS.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
Western Blot	0.1 µg/mL	Recombinant Mouse VEGF ₁₆₄ (Catalog # 493-MV)
Immunohistochemistry	5-15 µg/mL	See Below
Mouse VEGF Sandwich Immunoassay		Reagent
ELISA Capture	0.2-0.8 µg/mL	Mouse VEGF ₁₆₄ Antibody (Catalog # AF-493-NA)
ELISA Detection Standard	0.1-0.4 µg/mL	Mouse VEGF Biotinylated Antibody (Catalog # BAF493) Recombinant Mouse VEGF ₁₆₄ (Catalog # 493-MV)
Neutralization		Measured by its ability to neutralize VEGF ₁₆₄ -induced proliferation in HUVEC human umbilical vein endothelial cells. Conn, G. <i>et al.</i> (1990) Proc. Natl. Acad. Sci USA 87 :1323. The Neutralization Dose (ND ₅₀) is typically 0.05-0.15 µg/mL in the presence of 10 ng/mL Recombinant Mouse VEGF ₁₆₄ .

DATA



PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> • 12 months from date of receipt, -20 to -70 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Vascular endothelial growth factor (VEGF or VEGF-A), also known as vascular permeability factor (VPF), is a potent mediator of both angiogenesis and vasculogenesis in the fetus and adult (1 - 3). It is a member of the PDGF family that is characterized by a cystine knot structure formed by eight conserved cysteine residues (4). Alternately spliced isoforms of 121, 145, 165, 183, 189, and 206 amino acids (aa) have been identified in humans, with 120, 164 and 188 aa isoforms found in mouse (2, 4). Isoforms other than VEGF₁₂₀ and VEGF₁₂₁ contain basic heparin-binding regions and are not freely diffusible (4). Mouse VEGF₁₆₄ shares 97% aa sequence identity with corresponding regions of rat, 89% with human and porcine, 88% with bovine, and 90% with feline, equine and canine VEGF, respectively. VEGF binds the type I transmembrane receptor tyrosine kinases VEGF R1 (also called Flt-1) and VEGF R2 (Flk-1/KDR) on endothelial cells (4). Although affinity is highest for binding to VEGF R1, VEGF R2 appears to be the primary mediator of VEGF angiogenic activity (3, 4). Human VEGF₁₆₅ binds the semaphorin receptor, Neuropilin-1 and promotes complex formation with VEGF R2 (5). VEGF is required during embryogenesis to regulate the proliferation, migration, and survival of endothelial cells (3, 4). In adults, VEGF functions mainly in wound healing and the female reproductive cycle (3). Pathologically, it is involved in tumor angiogenesis and vascular leakage (6, 7). Circulating VEGF levels correlate with disease activity in autoimmune diseases such as rheumatoid arthritis, multiple sclerosis and systemic lupus erythematosus (8). VEGF is induced by hypoxia and cytokines such as IL-1, IL-6, IL-8, oncostatin M and TNF- α (3, 4, 9).

References:

1. Breier, G. *et al.* (1992) *Development* **114**:521.
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6. Weis, S.M. and D.A. Cheresh (2005) *Nature* **437**:497.
7. Thurston, G. (2002) *J. Anat.* **200**:575.
8. Carvalho, J.F. *et al.* (2007) *J. Clin. Immunol.* **27**:246.
9. Angelo, L.S. and R. Kurzrock (2007) *Clin. Cancer Res.* **13**:2825.