

**DESCRIPTION**

**Source** Chinese Hamster Ovary cell line, CHO-derived  
Ala112-Arg227  
Accession # P49767

**N-terminal Sequence Analysis** Ala112

**Structure / Form** Disulfide-linked homodimer

**Predicted Molecular Mass** 13 kDa

**SPECIFICATIONS**

**SDS-PAGE** 13-20 kDa, reducing conditions

**Activity** Measured in a cell proliferation assay using HMVEC human microvascular endothelial cells. Marconcini, L. *et al.* (1999) Proc. Natl. Acad. Sci. USA **96**:9671.  
The ED<sub>50</sub> for this effect is 1.5-9 ng/mL.

**Endotoxin Level** <0.10 EU per 1 µg of the protein by the LAL method.

**Purity** >95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

**Formulation** Lyophilized from a 0.2 µm filtered solution in HCl. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

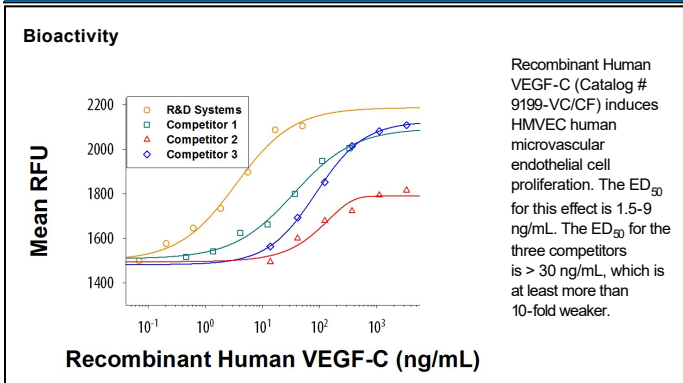
**Reconstitution** Reconstitute at 250 µg/mL in 4 mM HCl.

**Shipping** The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

**Stability & Storage** **Use a manual defrost freezer and avoid repeated freeze-thaw cycles.**

- 12 months from date of receipt, -20 to -70 °C as supplied.
- 1 month, 2 to 8 °C under sterile conditions after reconstitution.
- 3 months, -20 to -70 °C under sterile conditions after reconstitution.

**DATA**



**BACKGROUND**

Vascular endothelial growth factor C (VEGF-C) and VEGF-D constitute a subfamily of the angiogenic VEGF angiogenic factors (1). VEGF-C is synthesized as a 58 kDa molecule that consists of a VEGF homology domain (VHD) flanked by N- and C-terminal propeptides. The proprotein undergoes covalent homodimerization and stepwise proteolytic processing to generate ligands with increasing affinity for VEGF R3/Fit-4 (2-4). Fully processed VEGF-C containing just the 21 kDa VHD can additionally bind and activate VEGF R2/KDR/Fik-1 (2, 4). Fully processed human VEGF-C shares 98% amino acid sequence identity with mouse and rat VEGF-C. VEGF-C interactions with VEGF R3 are critical for lymphangiogenesis (5-8). VEGF-C and VEGF R3 are usually co-expressed at sites with lymphatic vessel sprouting, in the embryo, and in various pathological conditions. Over-expression of VEGF-C in tumor cells induces tumoral lymphatic hyperplasia, resulting in enhanced lymph flow and metastasis to regional lymph nodes (9-12). It also induces physiological and intratumoral neoangiogenesis and vessel sprouting through interactions with VEGF R2 (8, 13, 14).

**References:**

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