

DESCRIPTION

Source Mouse myeloma cell line, NS0-derived
Glu22-Val202, with a C-terminal 6-His tag
Accession # P05452

N-terminal Sequence Analysis Glu22

Predicted Molecular Mass 21.0 kDa (monomer)

SPECIFICATIONS

SDS-PAGE 25 kDa, reducing conditions

Activity Measured by its binding ability in a functional ELISA.
When rhHGF (Catalog # 294-HGN) is immobilized at 5 µg/mL, the concentration of rhCLEC3B that produces 50% of the optimal binding response is found to be approximately 1-5 µg/mL.

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

Purity >95%, by SDS-PAGE under reducing conditions and visualized by silver stain.

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

PREPARATION AND STORAGE

Reconstitution Reconstitute at 100 µg/mL in PBS.

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.

BACKGROUND

CLEC3B (C-type lectin domain family 3 member B), also known as Tetranectin, is a 17 kDa O-glycosylated member of the C-type lectin superfamily (1, 2). Mature human CLEC3B consists of an α-helical coiled-coil region followed by one C-type lectin domain (CTLD) (3, 4). It shares 81% amino acid sequence identity with mouse and rat CLEC3B. CLEC3B associates into non-covalent homotrimers, although it was named Tetranectin based on the proposal that it formed tetramers (4, 5). CLEC3B is secreted by endocrine, epithelial, endothelial, and mesenchymal cells including several hematopoietic cell types (6, 7). It shows binding selectivity for heparan sulfate, fucoidan, and chondroitin sulfates A, B, and C (8). CLEC3B binds the Kringle domain-containing proteins Plasminogen, tPA, and HGF, and it enhances the tPA-mediated activation of Plasminogen (5, 9). It also reduces the ability of Angiostatin (a Plasminogen fragment) to inhibit vascular endothelial cell proliferation (10). In mouse, CLEC3B is involved in the development and repair of muscle, spine, and skin (11 - 13). CLEC3B is upregulated in stromal cells surrounding various tumors but not in the tumor cells themselves (3, 14 - 16). It is concentrated in the extracellular matrix at the leading edge of malignant tumors, a pattern that overlaps that of Plasminogen (3, 14, 16).

References:

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