

DESCRIPTION

Source Mouse myeloma cell line, NS0-derived
His20-Pro187, with a C-terminal 6-His tag
Accession # NP_808457

N-terminal Sequence Analysis His20

Predicted Molecular Mass 20 kDa

SPECIFICATIONS

SDS-PAGE 29-38 kDa, reducing conditions

Activity Measured by its ability to inhibit anti-CD3 antibody induced IFN-gamma secretion by human peripheral blood mononuclear cells (PBMC).
The ED₅₀ for this effect is 0.5-2.5 µg/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 PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 200 µg/mL in PBS.

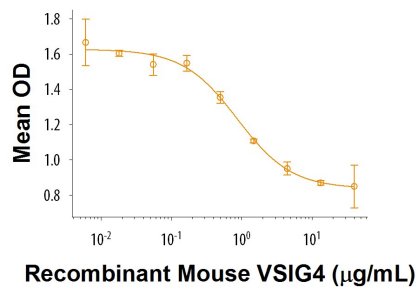
Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

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.

DATA

Bioactivity



Recombinant Mouse VSIG4 (Catalog # 9747-VS) inhibits IFN-γ secretion by human peripheral blood mononuclear cells in the presence of anti-CD3 antibody. The ED₅₀ for this effect is 0.5-2.5 µg/mL.

BACKGROUND

VSIG4 (Vset and immunoglobulin domain containing 4), also known as complement receptor immunoglobulin (CRIg) and Z39IG, is a 45 kDa, type I transmembrane protein of the B7 family within the Ig superfamily that is expressed only in tissue resident macrophages (1-4). The gene is located on the X chromosome (2). Mouse VSIG4 is synthesized as a 280 amino acid (aa) precursor that contains a signal sequence, an IgV-type immunological domain, one potential N-linked glycosylation site, and a single transmembrane domain (4). The IgV domain of mouse VSIG4 shares 86% and 80% aa sequence identity with the IgV domains of rat and human VSIG4, respectively. VSIG4 is specifically expressed on macrophages in the thymic medulla, peritoneum, alveoli, synovia, adipose and heart, liver Kupffer cells, placental Hofbauer cells, and atherosclerotic foam cells (1-4, 6-9). It is absent on infiltrating macrophages (8). VSIG4 is a complement receptor that binds C3b and iC3b fragments, internalizes them to recycling endosomes, and is recycled to the cell surface (4, 6). It contributes significantly to innate immunity by binding and phagocytosis of complement opsonized invading pathogens (4, 8, 10). Binding of either native or recombinant soluble VSIG4 to C3b inhibits complement amplification through the alternative, but not classical, pathway (10, 11). VSIG4 is also a negative regulator of mouse and human T cell activation (2). Although VSIG4 engagement may activate NFκB and thus be proinflammatory in some cases, many of its activities are important in resolving, rather than initiating, inflammation (1, 2, 7, 10, 11). There is emerging evidence in human conditions that VSIG-4 may be a valuable biomarker in infection and immunity, inflammatory conditions and cancer prognosis (12-14).

References:

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