

**DESCRIPTION**

**Source** Chinese Hamster Ovary cell line, CHO-derived human FGL2 protein  
Asp32-Pro439, with a C-terminal 6-His tag  
Accession # Q14314

**N-terminal Sequence Analysis** Asp32

**Predicted Molecular Mass** 48 kDa

**SPECIFICATIONS**

**SDS-PAGE** 52-79 kDa, under reducing conditions.

**Activity** Measured by its ability to inhibit anti-CD3-induced proliferation of stimulated human T cells.  
The ED<sub>50</sub> for this effect is 1-8 µg/mL.

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

**Purity** >80%, 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 with Trehalose. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

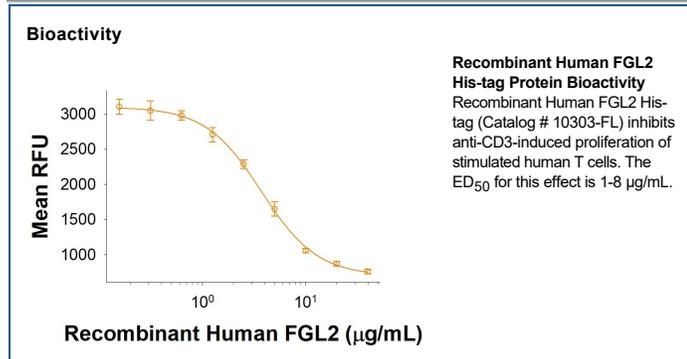
**Reconstitution** Reconstitute at 500 µg/mL in PBS.

**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**



**BACKGROUND**

FGL2 (fibrinogen-like protein 2), also called fibroleukin, is a 64-70 kDa secreted glycoprotein of the Fibrinogen-like superfamily. It has prothrombinase activity and also promotes T regulatory (Treg) activity (1-6). The human FGL2 gene encodes a 439 amino acid (aa) protein that contains a 23 aa signal sequence and a 416 aa mature sequence with a coiled-coil region and a fibronectin C-terminal homology domain or FRED (1, 2). A 260-280 kDa FGL2 complex is thought to be a tetramer formed by covalent disulfide linkage of dimers that are associated via coiled-coil interactions (2, 3). Mature human FGL2 shares 79% aa identity with mouse and rat FGL2. FGL2 appears to have two modes of action. One mode involves its prothrombinase activity, which requires calcium and acidic phospholipids (4). This mode is thought to be active during hepatitis viral infections when FGL2, produced by macrophages in response to IFN-gamma, induces hepatic apoptosis and fibrin deposition (7). In addition, FGL2 produced by endothelial cells in response to TNF-alpha within cardiac xenografts or allografts promotes coagulation during acute vascular rejection (7-9). A second mode of action involves soluble (not phospholipid-associated) FGL2 and is independent of prothrombinase activity (2). Soluble FGL2 is required for Treg function, and directly suppresses DC, T, and B cell immune reactivity; consequently, some FGL2-deficient mice develop autoimmune glomerulonephritis (5, 6). *In vitro*, soluble FGL2 can skew T cell polarization toward Th2 and inhibit proliferation of stimulated T cells and maturation of DC (6). In pregnancy, fetal trophoblast cells secrete FGL2. The immune suppressive mode of FGL2 may prevent early fetal loss; however, the procoagulant mode is thought to mediate infection-triggered abortion (10). In the central nervous system (CNS), FGL2 was shown to be highly expressed in glioma stem cells and primary glioblastoma cells and may serve as a critical immune oncology target (11).

**References:**

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