

DESCRIPTION

Source	Human embryonic kidney cell, HEK293-derived human GPVI protein		
	Human GPVI (Gln21-Lys267) Accession # Q9HCN6.4	IEGRMD	Human IgG ₁ (Pro100-Lys330)
	N-terminus		C-terminus
N-terminal Sequence Analysis	Gln21		
Structure / Form	Disulfide-linked homodimer		
Predicted Molecular Mass	54 kDa		

SPECIFICATIONS

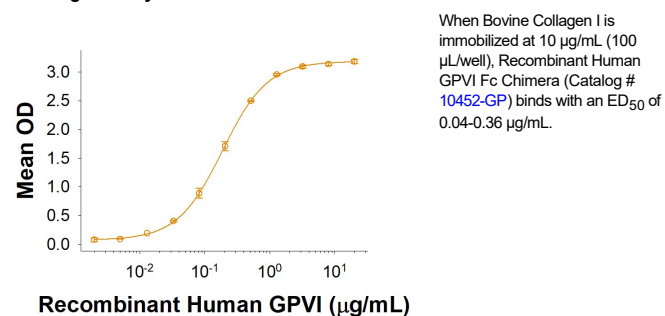
SDS-PAGE	68-76 kDa, under reducing conditions
Activity	Measured by its binding ability in a functional ELISA. When Bovine Collagen I is immobilized at 10 µg/mL (100 µL/well), Recombinant Human GPVI Fc Chimera (Catalog # 10452-GP) binds with an ED ₅₀ of 0.04-0.36 µg/mL.
Endotoxin Level	<0.10 EU per 1 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 with Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

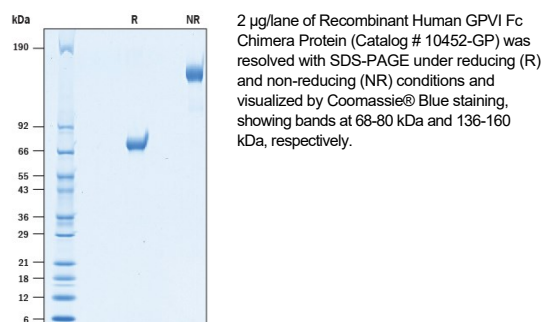
Reconstitution	Reconstitute at 500 µg/mL in PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. • 12 months from date of receipt, -20 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

Binding Activity



SDS-PAGE



BACKGROUND

Glycoprotein VI (GPVI) is a 63 kDa platelet/megakaryocyte-specific type I transmembrane glycoprotein of the immunoglobulin superfamily that is an important collagen receptor and initiator of platelet activation, aggregation and thrombin generation (1, 2). GPVI is also a secondary receptor required for platelet spreading on laminin (3). Human GPVI contains a 20 amino acid (aa) signal sequence, a 247 aa extracellular domain (ECD) that has two C-type Ig-like domains followed by a mucin-like, presumably O-glycosylated Ser-Thr-rich region, a 21 aa transmembrane (TM) domain and a 51 aa cytoplasmic tail that contains calmodulin-binding and SH3 domains. Human GPVI ECD shows 69%, 65% and 70% aa identity with mouse, bovine and canine GPVI ECD, respectively. Two splice variants exist; one is 17 aa shorter in the ECD, while the other diverges at aa 260, creating an inactive monomeric and presumably secreted 681 aa protein (3). GPVI associates with the FcRγ via charged amino acid in the TM domains of GPVI (arginine) and the FcRγ (aspartic acid) (2). Collagen binding by the GPVI Ig-like domains initiates signaling through the FcRγ ITAM sequence (2). Dimerization of GPVI (2:2 with FcRγ) and N-glycosylation greatly enhances collagen binding (5, 6). Type I and III collagens are strong thrombus-forming components in the vascular subendothelium and atherosclerotic plaques (7). GPVI initiates binding to fibrillar collagens under flow conditions, then activates integrin α2β1 which binds collagen more tightly (8). GPVI deficiencies cause only a mild bleeding tendency, probably because integrin α2β1 is able to minimally initiate collagen binding (8). Normal human GPVI concentration can vary widely and affect maximum thrombin generation (9). Engagement of GPVI by collagens or other agonists, including autoantibodies, causes calmodulin-regulated metalloproteinase cleavage of the 57 kDa ECD and depletes surface GPVI (10).

References:

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