

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human GPVI in Western blots.
Source	Polyclonal Sheep IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell-line NS0-derived recombinant human GPVI Gln21-Lys267 Accession # Q9HCN6
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with BSA as a carrier protein. See Certificate of Analysis for details.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
Western Blot	0.1 µg/mL	Recombinant Human GPVI (Catalog # 3627-GP)

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	<p>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</p> <ul style="list-style-type: none"> • 12 months from date of receipt, -20 to -70 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 6 months, -20 to -70 °C under sterile conditions after reconstitution.

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 Fc receptor γ -chain via charged aa in the TM domains of GPVI (arginine) and the FcR γ (aspartic acid) (2). Collagen binding by the GPVI Ig-like domains initiates signaling through the FcR γ ITAM sequence (2). Dimerization of GPVI (2:2 with FcR γ) 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 $\alpha_2\beta_1$ which binds collagen more tightly (8). GPVI deficiencies cause only a mild bleeding tendency, probably because integrin $\alpha_2\beta_1$ 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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