Recombinant Mouse GP6/GPVI (C-6His)

Catalog Number: PKSM041437

Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

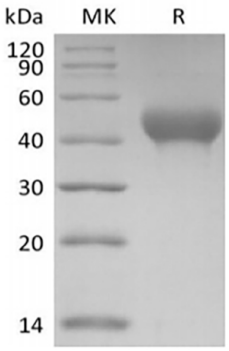
Description

Species	Mouse
Source	HEK293 Cells-derived Mouse GP6/GPVI protein Gln22-Lys265, with an C-terminal His
Calculated MW	27.8 kDa
Observed MW	40-60 kDa
Accession	B2RR15
Bio-activity	Not validated for activity

Properties

Purity	> 95 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μg of the protein as determined by the LAL method.
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80 ℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from a 0.2 μm filtered solution of PBS, pH 7.4. Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 95 % as determined by reducing 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. GPVI is also a secondary receptor required for platelet spreading on laminin. GPVI associates with the Fc receptor gamma -chain via charged aa in the TM domains of GPVI (arginine) and the FcR gamma (aspartic acid). Collagen binding by the GPVI Ig-like domains initiates signaling through the FcR gamma ITAM sequence. Dimerization of GPVI (2:2 with FcR gamma) and N-glycosylation greatly enhances collagen binding. Type I and III collagens are strong thrombus-forming components in the vascular subendothelium and atherosclerotic plaques. GPVI initiates binding to fibrillar collagens under flow conditions, then activates integrin alpha 2 beta 1 which binds collagen more tightly. GPVI deficiencies cause only a mild bleeding tendency, probably because integrin alpha 2 beta 1 is able to minimally initiate collagen binding. Normal human GPVI concentration can vary widely and affect maximum thrombin generation. Engagement of GPVI by collagens or other agonists, including autoantibodies, causes calmodulin-regulated metalloproteinase cleavage of the 57 kDa ECD and depletes surface GPVI.