

## Human GPVI VHH (S\_D4)



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## Properties

<b>Product type:</b>	Bi-head VHH
<b>Catalog number:</b>	G-003
<b>Clone number:</b>	34_1_Suleyka_D4 (S_D4)
<b>Immunogen:</b>	Purified recombinant human glycoprotein VI (GPVI)
<b>Reacts with:</b>	Human glycoprotein VI (GPVI)
<b>Tested applications:</b>	Blocking VHH, ELISA, Flow cytometry
<b>Source:</b>	Monoclonal VHH ( <i>Llama glama</i> ), purified from HEK293-E 253 cells using affinity chromatography
<b>Clonality:</b>	Monoclonal
<b>Purity:</b>	Rmp-Protein A purification, 99%
<b>Storage buffer:</b>	PBS
<b>Form:</b>	Liquid
<b>Concentration:</b>	4.0 mg/ml
<b>Storage:</b>	Store at -80°C

## Products

Cat. No.	Target	Clone	Form	Applications	Size
G-003	Human GPVI	S_D4	Purified	blocking VHH ELISA, FC,	250 µg

## Description

Glycoprotein VI (GPVI, GP6) is a platelet membrane glycoprotein of the immunoglobulin superfamily and a receptor for collagen involved in collagen-induced platelet adhesion and activation. Ligand binding to GPVI initiates migration to lipid rafts and subsequent dimerization of GPVI, the formation of a signaling complex with the FcR gamma chain, and the recruitment of downstream signaling proteins, including Src family kinases Fyn and Lyn and the adapter protein LAT. Activation of this signaling pathway results in thrombus formation via the activation of phospholipase C gamma 2. Transduction of signals by GPVI is mediated in an immunoreceptor-based manner and involves its immune-receptor tyrosine-based activation motif (ITAM).<sup>1,3</sup> In addition to collagen or collagen-related peptides, GPVI was reported to also bind other ligands, including fibrinogen and fibrin, resulting in the support of growth and stabilization of the thrombus.<sup>2,4</sup>

Different inherited and acquired disease-causing variants of GPVI are known, which include mutations in the *gp6* gene causing the bleeding disorder platelet-type 11 (BDPLT11), a mild to moderate bleeding disorder characterized by defective platelet activation and aggregation in response to collagen. Also, platelets may be deficient of GPVI due to inherited or acquired loss of the protein, the latter through i.e. autoantibody-induced receptor shedding.<sup>5</sup>

## Use, storage and stability

The human GPVI bi-head (S\_D4) VHH is suitable for use in ELISA and flow cytometry studies. In addition, it acts as blocking agent of collagen-induced platelet activation: in a flow cytometry-based platelet activation test, S\_D4 blocks GPVI-mediated fibrinogen binding and P-selectin expression as markers for integrin  $\alpha$ IIb $\beta$ 3 activation and platelet granule secretion, and it blocks collagen-induced platelet aggregation in light transmission aggregometry studies.

S\_D4 is different in sequence from GPVI VHH (S\_D2; Cat. No. G-002), but similar in its blocking action.

After thawing, the VHH should be stored in appropriate small aliquots at -20°C or -80°C (stable for at least 6 months) or at 4°C for short time storage.

## References

1. Watson SP, Gibbins J. Collagen receptor signalling in platelets: extending the role of the ITAM. *Immunol Today*. 1998; 19: 260-264. Doi: 10.1016/s0167-5699(98)01267-5.
2. Rayes J, Watson SP, Nieswandt B. Functional significance of the platelet immune receptors GPVI and CLEC-2. *J Clin Invest*. 2019; 129: 12-23. doi: 10.1172/JCI122955.
3. Bodin S, Tronchère H, Payrastre B. Lipid rafts are critical membrane domains in blood platelet activation processes. *Biochim Biophys Acta*. 2003; 1610: 247-257. doi: 10.1016/s0005-2736(03)00022-1.
4. Mangin PH, Onselaer MB, Receveur N, *et al*. Immobilized fibrinogen activates human platelets through glycoprotein VI. *Haematologica*. 2018; 103: 898–907. doi: 0.3324/haematol.2017.182972.
5. Nurden AT. Clinical significance of altered collagen-receptor functioning in platelets with emphasis on glycoprotein VI. *Blood Rev*. 2019; 38: 100592. doi: 10.1016/j.blre.2019.100592.

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