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- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

F. +43(0)1 489 3961-7

mail@szabo-scandic.com

www.szabo-scandic.com

linkedin.com/company/szaboscandic

Product datasheet MON3053

Mouse anti-JAM-1, clone M.Ab.F11 (Monoclonal)

Clone no. M.Ab.F11

MONOSAN

Product name	Mouse anti-JAM-1, clone M.Ab.F11 (Monoclonal)
Host	Mouse
Applications	FC,FUNC,ELISA,IF,IP,IHC-P,WB
Species reactivity	human
Conjugate	-
Immunogen	Unknown or proprietery to MONOSAN and/or its suppliers
Isotype	IgG1
Clonality	Monoclonal
Clone number	M.Ab.F11
Size	1 ml
Concentration	100 ug/ ml
Format	-
Storage buffer	PBS with 0.1% BSA and 0.02% sodium azide
Storage until expiry date	2-8°C

Mouse anti-JAM-1, clone M.Ab.F11 (Monoclonal)

Clone no. M.Ab.F11

MONOSAN

Additional info

The monoclonal antibody M.Ab.F11 recognizes junctional adhesion molecule-A (JAM-A) also known as the human platelet F11-Receptor (F11R) or JAM-1. JAM-A is a surface glycoprotein duplex (32 and 35 kDa) belongingto the immunoglobulin superfamily found on the surface of human platelets and at intercellular junctions of endothelial cells and epithelial cells. JAM-A belongs together with JAM-C (JAM-2) and JAM-B (VE-JAM or JAM-3) to a family of adhesion proteins with a V-C2 immunoglobulin domain organization. JAM-A plays an important role in tight junctions where it is involved in cell-to-cell adhesion through homophilic interactions. It co-distributes with other tight junction components such as ZO-1, 7H6 antigen, cingulin and occludin. Moreover, JAM-A plays a role in platelet aggregation, secretion, adhesion, spreading.
 In the platelet, JAM-A is a membrane protein involved in 2 distinct processes initiated on the platelet surface. Namely,, antibody-induced platelet aggregation and secretion both dependent on FcgammaRII and GPIIb/IIIa integrin, a process that may be involved in pathophysiological processes associated with certain thrombocytopenias and secondly, antibody mediated platelet adhesion independent from FcgammaRII or- fibrinogen receptor that appears to play a role in physiological processes associated with platelet adhesion and aggregation. A physiological role for the JAM-A protein was demonstrated by its phosphorylation after the stimulation of platelets by thrombin and collagen. A pathophysiological role for the JAM-A was revealed by demonstrating the presence of JAM-A antibodies in patients with thrombocytopenia. Adhesion of platelets through JAM-A resulted in events characteristic of the action of cell adhesion molecules. Recent data suggests a role for JAM-A in the adhesion of platelets to cytokine-inflamed endothelial cells and thus in thrombosis and atherosclerosis induced in non-denuded blood vessels by inflammatory processes.

References

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2. Naik, U et al Biochem J 1995, 310: 155
3. Wang; F et al. Biochem J 1995; 311: 401
4. Sobocka M et al. Blood 2000; 95: 2600
5. Babinska A et al. Thromb Haemost 2002; 87: 712

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