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Produktinformation



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Lieferung & Zahlungsart

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Zuschläge

- Mindermengenzuschlag
- 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](https://www.linkedin.com/company/szaboscandic) 

Mouse anti-JAM-1, clone BV16 (Monoclonal)

Clone no. BV16

MONOSAN

Product name	Mouse anti-JAM-1, clone BV16 (Monoclonal)
Host	Mouse
Applications	IHC-fr,FC,IF,IP,WB
Species reactivity	human
Conjugate	-
Immunogen	Unknown or proprietary to MONOSAN and/or its suppliers
Isotype	IgG1
Clonality	Monoclonal
Clone number	BV16
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

FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES

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Additional info

The monoclonal antibody BV16 recognizes the human junction adhesion molecule (JAM)-A. Together with JAM-C (JAM-2) and JAM-B (VE-JAM or JAM-3), JAM-A belongs to a family of adhesion proteins with a V-C2 immunoglobulin domain organization and their molecular weight is about 30-40 kDa. JAMs are important for a variety of cellular processes, including tight junction assembly, leukocyte transmigration, platelet activation, angiogenesis and virus binding. JAM-A is expressed by endothelial and epithelial cells, platelets, neutrophils, monocytes, lymphocytes and erythrocytes. Like all other JAMs, JAM-A plays an important role in tight junctions where it is involved in cell-to-cell adhesion through homophilic interaction. It codistributes with other tight junction components as ZO-1, 7H6 antigen, cingulin and occludin. JAM-A also plays an important role in leukocyte transmigration. Leukocyte transmigration can be blocked by antibodies and by soluble JAM-A/Fc fusion proteins. Homophilic JAM-A interactions between leukocytes and the endothelium but also heterophilic interactions of JAM-A with the beta2-integrin leukocyte function-associated antigen-1 (LFA-1) are considered to actively guide leukocytes during transmigration. Several studies imply a role for JAM-A in the initiation of atherosclerosis since JAM-A is upregulated on early atherosclerotic endothelium. The adhesion of activated platelets on the activated endothelium is mediated by homophilic interactions of JAM-A.

References

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2. Luo, Y et al Invest Ophthalmol Vis Sci 2006, 47: 3644
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4. Vetrano S et al. Gastroenterol 2008; 135: 173
5. -

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