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DATA SHEET

Mouse Anti-CD204/MSR1/SR-A [MD405]: MC0664

Intended Use: For Research Use Only

Description: The scavenger receptor class A (SR-A), also known as Macrophage Scavenger receptor types I and II (MSR1) or CD204, are members of the class A macrophage scavenger receptor family. These proteins bind large quantities of modified lipoproteins and promote endocytosis. SR-A plays a crucial role in lipid metabolism by mediating the uptake of modified LDL, which is significant for understanding atherosclerosis and other lipid-related diseases. SR-A expression occurs predominantly in the liver, placenta, and brain. Upregulation of SR-A in infiltrating myeloid cells may mediate clearance of specific damage signals in response to tissue injury, including ischemic stroke. SR-A germline mutations are also associated with increased prostate cancer susceptibility in some patient cohorts. SR-A contributes to various macrophage-associated pathological processes, including atherosclerosis and Alzheimer′s disease, as well as host defense and cellular adhesion.

Specifications

Clone:	MD405
Source:	Mouse
Isotype:	IgG2a/κ
Reactivity:	Human
Immunogen:	Human SR-A protein aa 61-250
Localization:	Cytoplasm
Formulation:	Antibody in PBS pH7.4, containing BSA and ≤ 0.09% sodium azide (NaN3)
Storage:	Store at 2°- 8°C
Applications:	IHC, ELISA, IF, IP, WB

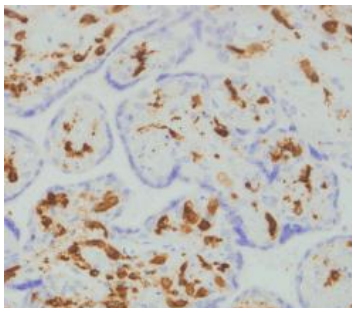
Package:

Description	Catalog No.	Size
CD204/MSR1/SR-A Concentrated	MC0664	1 ml

IHC Procedure*

Positive Control Tissue:	Liver, placenta, thymus, spleen, colon
Concentrated Dilution:	25-200
Pretreatment:	Tris EDTA pH9.0, 15 minutes Pressure Cooker or 30-60 minutes water bath at 95°-99°C
Incubation Time and Temp:	30-60 minutes @ RT
Detection:	Refer to the detection system manual

* Result should be confirmed by an established diagnostic procedure.



FFPE human placenta stained with anti-CD204 using DAB

References:

1. The Presence and Potential Role of ALDH1A2 in the Glioblastoma Microenvironment. Stephanie Sanders, et al. Cells. Sep 20;10(9):2485, 2021.
2. MAFB prevents excess inflammation after ischemic stroke by accelerating clearance of damage signals through MSR1. Takashi Shichita, et al. Nat Med. Jun;23(6):723-732, 2017. doi: 10.1038/nm.4312.
3. Mycobacterium leprae intracellular survival relies on cholesterol accumulation in infected macrophages: a potential target for new drugs for leprosy treatment. Katherine A Mattos, et al. Cell Microbiol. Jun;16(6):797-815, 2014. doi: 10.1111/cmi.12279.

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