Rat anti Mouse-MCP-1, clone ECE.2 (Monoclonal)

Clone no. ECE.2

MONOSAN

Product name	Rat anti Mouse-MCP-1, clone ECE.2 (Monoclonal)
Host	Rat
Applications	IHC-fr,WB
Species reactivity	mouse
Conjugate	-
Immunogen	Unknown or proprietery to MONOSAN and/or its suppliers
Isotype	IgG1
Clonality	Monoclonal
Clone number	ECE.2
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

Product datasheet MON7035

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Additional info

The monoclonal antibody ECE.2 recognizes mouse monocyte chemoattractant protein 1 (MCP-1). The murine JE gene encodes the monocyte-specific cytokine monocyte chemotactic protein 1 (MCP- 1). MCP-1 is a CC chemokine of 76 amino acids (~11 kDa) and is chemotactic for monocytes and basophils but not neutrophils and eosinophils. MCP-1 is expressed by smooth muscle cells (SMC), macrophages, endothelial cells, keratinocytes and fibroblasts in response to inflammatory stimuli such as interleukin 1Î² and tumor necrosis factor α. MCP-1 has been implicated in a variety of inflammatory processes, including inflammatory bowel disease, rheumatoid arthritis, asthma, nephritis, and parasitic and viral infections. MCP-1 antigen is not detected in the endothelium or SMC of normal arteries. MCP-1 has also been shown to exhibit biological activities other than chemotaxis. It can induce the proliferation and activation of killer cells known as CHAK (CC-Chemokine-activated killer) MCP-1 signals via the CCR2 receptor, and is critical for aneurysm formation because of its stability to recruit leukocytes. These leukocytes produce extracellular matrix-degrading MMPs, thereby inductin aortic remodelling and dilatation. Interleukin-6 is also involved in this amplification loop accelerating vascular inflammation. MCP-/- mice display significantly delayed wound re-epithelialization, and also delayed wound angiogenesis.

References

1. Zoja; C et al. J Am Soc Nephrol 1997; 8: 720
2 Kimura, Y et al Eur J Pharmacol 2008, 584: 415
3. Tieu; B et al. J Clin Invest 2009; 119: 3637
4. -
5. -

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