PRODUCT INFORMATION

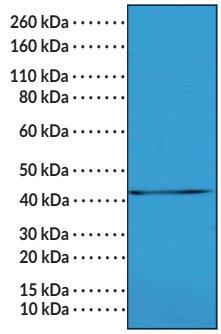
α-SMA (N-Term) Rabbit Monoclonal Antibody (Clone RM253)

Item No. 32204

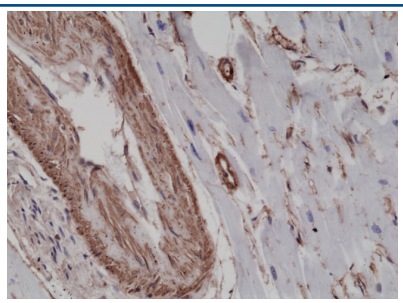
Overview and Properties

Contents:	This vial contains 100 µl of protein A-affinity purified monoclonal antibody.
Synonyms:	α-Actin 2, Smooth Muscle, ACTA2, α-Smooth Muscle Actin
Immunogen:	Synthetic peptide corresponding to the N-terminus of human α-SMA
Species Reactivity:	(+) Human, mouse
Cross Reactivity:	(+) α-SMA
Form:	Liquid
Storage:	-20°C (as supplied)
Stability:	≥1 year
Storage Buffer:	PBS with 50% glycerol, 1% BSA, and 0.09% sodium azide
Concentration:	50 µg/ml
Clone:	RM253
Host:	Rabbit
Isotype:	IgG
Applications:	Immunohistochemistry (IHC) and Western blot (WB); the recommended starting dilution for IHC is 1:1,1000-1:2,500 and 1:1,1000-1:2,000 for WB. Other applications were not tested, therefore optimal working concentration/dilution should be determined empirically.

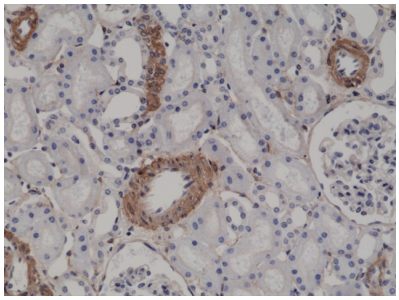
Images



WB of mouse heart tissue lysate using α-SMA (N-term) Rabbit Monoclonal Antibody (Clone RM253) at a dilution of 1:1,000.



Immunohistochemical staining of formalin-fixed and paraffin-embedded human heart tissue using α-SMA (N-term) Rabbit Monoclonal Antibody (Clone RM253) at a dilution of 1:2,500.



Immunohistochemical staining of formalin-fixed and paraffin-embedded human kidney tissue using α-SMA (N-term) Rabbit Monoclonal Antibody (Clone RM253) at a dilution of 1:2,500.

CAYMAN CHEMICAL
1180 EAST ELLSWORTH RD
ANN ARBOR, MI 48108 · USA
PHONE: [800] 364-9897
[734] 971-3335
FAX: [734] 971-3640
CUSTSERV@CAYMANCHEM.COM
WWW.CAYMANCHEM.COM

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Description

α-Smooth muscle actin (α-SMA) is a cytoskeletal protein encoded by *ACTA2* in humans and a component of the smooth muscle cell (SMC) contractile unit.[1] It is expressed in the cytosol of SMCs throughout the vascular system where it polymerizes to form the thin filaments of the SMC contractile unit. α-SMA is also expressed in pericytes and myofibroblasts.[2] *Acta2* expression is upregulated in, and positively correlates with enhanced migration and contraction of, hepatic stellate cells, which are precursors to myofibroblasts, in a rat model of carbon tetrachloride-induced liver injury. Missense mutations in *ACTA2* cause approximately 14% of inherited ascending thoracic aortic aneurysms and dissections (TAAD) *via* induction of defective SMC contraction, aortic medial degeneration, and SMC hyperplasia or disarray.[3] Mutations in *ACTA2* are also positively correlated with a predisposition for various additional vascular diseases including premature coronary artery disease, stroke, and Moyamoya disease.[4] Cayman's α-SMA (N-Term) Rabbit Monoclonal Antibody (Clone RM253) can be used for immunohistochemistry (IHC) and Western blot (WB) applications.

References

1. Karimi, A. and Milewicz, D.M. Structure of the elastin-contractile units in the thoracic aorta and how genes that cause thoracic aortic aneurysms and dissections disrupt this structure. *Can. J. Cardiol.* **32(1)**, 26-34 (2016).
2. Rockey, D.C., Weymouth, N., and Shi, Z. Smooth muscle α actin (*Acta2*) and myofibroblast function during hepatic wound healing. *PloS One* **8(10)**, e77166 (2013).
3. Cho, D.-C., Pannu, H., Tran-Fadulu, V., *et al*. Mutations in smooth muscle alpha-actin (*ACTA2*) lead to thoracic aortic aneurysms and dissections. *Nat. Genet.* **39(12)**, 1448-1493 (2007).
4. Guo, D.-C., Papke, C.L., Tran-Fadulu, V., *et al*. Mutations in smooth muscle alpha-actin (*ACTA2*) cause coronary artery disease, stroke, and Moyamoya disease, along with thoracic aortic disease. *Am. J. Hum. Genet.* **84(5)**, 617-627 (2009).

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