

Medaysis Enable Innovation

Rabbit Anti-Arginase-1/ARG-1 [MD167R]: RM0279, EM0279RTU7

Intended Use: For Research Use Only

Description: Arginase is a manganese metalloenzyme that catalyzes the hydrolysis of arginine to generate ornithine and urea. Arginiase I and II are isoenzymes which differ in subcellular localization, regulation, and possibly function. Arginase I is a cytosolic enzyme, which is expressed mainly in the liver as part of the urea cycle, whereas arginase II is a mitochondrial protein found in a variety of tissues. Antibody to ARG-1 labels hepatocytes in normal tissues and granulocytes in peripheral blood. ARG-1 is a sensitive and specific marker for identification of hepatocellular carcinoma.

Specifications:

Clone: MD167R
Source: Rabbit
Isotype: IgG
Reactivity: Human

Immunogen: Recombinant human ARG1 protein fragment aa300-400

Localization: Cytoplasm, nucleus

Formulation: Purified antibody in PBS pH7.4, containing BSA and ≤ 0.09% sodium azide (NaN3)

Storage: Store at 2°-8°C Applications: IHC, WB

Package:

Description	Catalog No.	Size	
Arginase-1/ARG-1 Concentrated	RM0279	1 ml	
Arginase-1/ARG-1 Prediluted	RM0279RTU7	7 ml	

IHC Procedure*:

Positive Control Tissue: HCC, normal liver

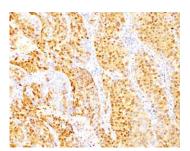
Concentrated Dilution: 25-100

Pretreatment: Tris EDTA pH9.0, 15 minutes using Pressure Cooker, or 30-60 minutes

using 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 HCC liver stained with anti-Arginase-1 using DAB

References:

- Arginase-1 is a novel immunohistochemical marker of hepatocellular differentiation. Ordóñez NG. Adv Anat Pathol 21:285-90, 2014.
- 2. Liver-specific knockout of arginase-1 leads to a profound phenotype similar to inducible whole body arginase-1 deficiency. Ballantyne LL, et al. Mol Genet Metab Rep 9:54-60, 2016.
- 3. Inducible arginase 1 deficiency in mice leads to hyperargininemia and altered amino acid metabolism. Sin YY, et al. PLoS One 8:e80001, 2013.

Doc. 100-RM0279

Rev. B

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