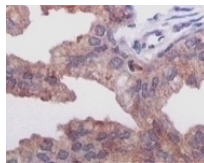
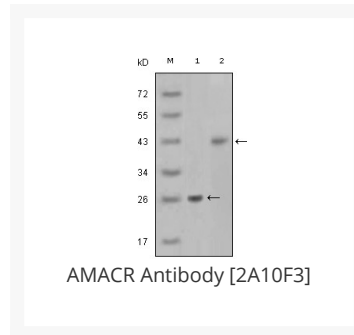




AMACR Antibody [2A10F3]

Cat. No.: 32-107



Immunohistochemical analysis of paraffin - embedded human prostate adenocarcinoma tissue showing cytoplasmic location using AMACR antibody with DAB staining.

Ψ Specifications

HOST SPECIES:	Mouse
SPECIES REACTIVITY:	Human
IMMUNOGEN:	Purified truncated recombinant AMACR-His fusion protein expressed in E. Coli strain BL21 (DE3).
TESTED APPLICATIONS:	ELISA, IHC, WB
APPLICATIONS:	Western Blot:1:500 - 1:2,000 IHC(P):1:500 - 1:2,000 ELISA:Propose dilution 1:10,000. Determining optimal working dilutions by titration test.

CLONALITY:	Monoclonal
ISOTYPE:	IgG2b
CONJUGATE:	Unconjugated
BUFFER:	Ascitic fluid containing 0.03% sodium azide.
STORAGE CONDITIONS:	AMACR monoclonal antibody can be stored at -20 °C, stable for one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. Antibodies should not be exposed to prolonged high temperatures.

Additional Info

OFFICIAL SYMBOL:	AMACR
ALTERNATE NAMES:	2-methylacyl-CoA racemaseRM, RACE, CBAS4, AMACRD
ACCESSION NO.:	Q9UHK6
PROTEIN GI NO.:	313104070
GENE ID:	23600
USER NOTE:	Optimal dilutions for each application to be determined by the researcher.

Background and References

BACKGROUND:	AMACR (alpha-methylacyl-CoA racemase) has been recently described as prostate cancer-specific gene that encodes a protein involved in the beta-oxidation of branched chain fatty acids. Expression of AMACR protein is found in prostatic adenocarcinoma but not in benign prostatic tissue. It stains premalignant lesions of prostate: high-grade prostatic intraepithelial neoplasia (PIN) and atypical adenomatous hyperplasia. AMACR can be used as a positive marker for PIN. Defects in AMACR are the cause of congenital bile acid synthesis defect type 4 (CBAS4); also known as cholestasis, intrahepatic, with defective conversion of trihydroxycoprostanic acid to cholic acid or trihydroxycoprostanic acid in bile. Clinical features include neonatal jaundice, intrahepatic cholestasis, bile duct deficiency and absence of cholic acid from bile.
REFERENCES:	1) Chen Q. Watson JT. Marengo SR. et al. Cancer Lett. 2006, Dec 8, 244 (2):274-88.Epub 2006 Feb 23.
	2) Epstein JI. Herawi M. J Urol. 2006, Mar, 175 (3 Pt 1):820-34. Review.

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