abcam

ab110296

Anti-ACADM/MCAD antibody [3B7BH7]

Key facts

Isotype	IgG1
Host species	Mouse
Storage buffer	pH: 7.5 Preservative: 0.02% Sodium azide Constituents: 99.98% HEPES buffered saline
Form	Liquid
Clonality	Monoclonal
Immunogen	The exact immunogen used to generate this antibody is proprietary information.
Clone number	3B7BH7
Purification technique	Precipitation Ammonium Sulphate
Light chain type	kappa
Concentration	1 mg/mL The concentration of this product may be batch-dependent Batch concentration finder →
Purification notes	Produced in vitro using hybridomas grown in serum-free medium, and then purified by biochemical fractionation.

Reactivity data

IHC-P

Tested

Species	Human
Dilution info	1/1000

Notes	Perform heat-mediated antigen retrieval via the pressure cooker method before commencing with IHC staining protocol.

Expected

Species	Mouse, Rat
Dilution info	Use at an assay dependent concentration.
Notes	-

Flow Cyt

Tested

Species	Human
Dilution info	1 µg/mL
Notes	ab170190 - Mouse monoclonal IgG1, is suitable for use as an isotype control with this antibody.

Expected

Species	Mouse, Rat
Dilution info	Use at an assay dependent concentration.
Notes	-

WB

Tested

Species	Human
Dilution info	0.125 µg/mL
Notes	-

Species	Mouse
Dilution info	0.125 µg/mL
Notes	-

Species	Rat

Dilution info	0.125 µg/mL
Notes	-

ICC/IF

Tested

Species	Human
Dilution info	1 µg/mL
Notes	-

Expected

Species	Mouse, Rat
Dilution info	Use at an assay dependent concentration.
Notes	-

Target data

See full target information ACADM

Function

Medium-chain specific acyl-CoA dehydrogenase is one of the acyl-CoA dehydrogenases that catalyze the first step of mitochondrial fatty acid beta-oxidation (FAO), breaking down fatty acids into acetyl-CoA and allowing the production of energy from fats (PubMed:1970566, PubMed:21237683, PubMed:2251268, PubMed:8823175). The first step of FAO consists in the proR-proR stereospecific alpha, beta-dehydrogenation of fatty acyl-CoA thioesters using the electron transfer flavoprotein (ETF) as their physiologic electron acceptor, resulting in the formation of trans-2-enoyl-CoA ((2E)-enoyl-CoA) (PubMed:2251268). ETF is the electron acceptor that transfers electrons to the main mitochondrial respiratory chain via ETF-ubiquinone oxidoreductase (ETF dehydrogenase) (PubMed:15159392, PubMed:25416781). Among the different mitochondrial acyl-CoA dehydrogenases, medium-chain specific acyl-CoA dehydrogenase has preference for fatty acyl-CoAs with saturated 6 to 12 carbons long primary chains, making it but can also catalyze longer chains such as C14 and C16 (PubMed:1970566, PubMed:21237683, PubMed:2251268, PubMed:8823175).

Storage

Shipped at conditions	Blue Ice

Appropriate short-term storage conditions	+4°C
Appropriate long-term storage conditions	+4°C
Storage information	Do Not Freeze

Notes

MCAD (medium-chain acyl-CoA dehydrogenase) is an oxidoreductase enzyme of the mitochondrial fatty acid beta-oxidation pathway that is specific for acyl chain lengths of 4 to 16. It also utilizes the electron transfer flavoprotein (ETF) as electron acceptor that transfers the electrons to the main mitochondrial respiratory chain via ETF-ubiquinone oxidoreductase (ETF dehydrogenase).

Want a custom formulation?

This antibody clone is manufactured by Abcam. If you require a custom buffer formulation or conjugation for your experiments, please contact orders@abcam.com

Supplementary info

This supplementary information is collated from multiple sources and compiled automatically.

Activity summary	ACADM also known as MCAD (medium-chain acyl-CoA dehydrogenase) is an enzyme that plays a significant role in fatty acid metabolism. It weighs approximately 44 kDa and expresses mainly in the liver heart and skeletal muscles. Functions of MCAD involve catalyzing the initial step in the mitochondrial fatty acid β-oxidation spiral specifically for the medium-chain fatty acids. This enzyme helps in the conversion of fatty acyl-CoA to trans-enoyl-CoA through dehydrogenation.
Biological function summary	The enzyme facilitates energy production by breaking down medium-chain fatty acids within mitochondria. MCAD operates as a homotetramer complex where each subunit significantly contributes to its overall function. Such enzymatic activity is important for providing energy particularly when glycogen stores are low. Its efficient operation during fasting states indicates its importance in metabolic homeostasis.
Pathways	Medium-chain acyl-CoA dehydrogenase (MCAD) is integral to the mitochondrial fatty acid beta-oxidation pathway. This pathway is a primary route for fatty acid catabolism ultimately leading to energy production in the form of ATP. MCAD's activity also relates to other fatty acid oxidation enzymes like VLCAD (very-long-chain acyl-CoA dehydrogenase) and SCAD (short-chain acyl-CoA dehydrogenase) which operate on different chain-length fatty acids cooperating to maintain energy balance.
Associated diseases and disorders	MCAD deficiency is a common metabolic disorder that impairs the normal breakdown of fatty acids. This condition results in the accumulation of fatty acid intermediates leading to hypoketotic hypoglycemia during fasting periods. Individuals with MCAD deficiency may experience lethargy vomiting and seizures. The disorder connects with other enzymes such as LCHAD (long-chain 3-

hydroxyacyl-CoA dehydrogenase) involved in similar pathways and deficiencies can result in related metabolic dysfunctions.

Product promise

Tested

We have tested this species and application combination and it works. It is covered by our product promise.

Expected

We have not tested this specific species and application combination in-house, but expect it will work. It is covered by our product promise.

Predicted

This species and application combination has not been tested, but we predict it will work based on strong homology. However, this combination is not covered by our product promise.

Not recommended

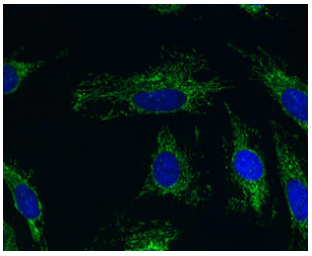
We do not recommend this combination. It is not covered by our product promise.

We are dedicated to supporting your work with high quality reagents and we are here for you every step of the way should you need us.

In the unlikely event of one of our products not working as expected, you are covered by our product promise.

Full details and terms and conditions can be found here:
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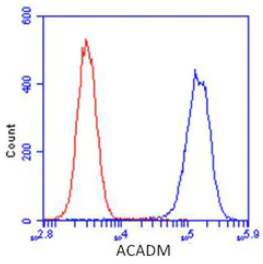
6 product images



Immunocytochemistry/ Immunofluorescence - Anti-ACADM/MCAD antibody [3B7BH7] (ab110296)

ACADM/MCAD Immunocytochemistry/ Immunofluorescence staining using mouse Anti-ACADM/MCAD antibody

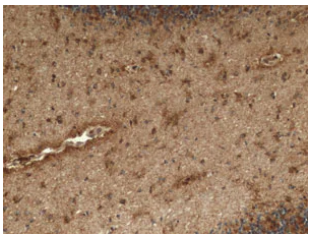
Immunocytochemistry image of ab110296 stained Human HeLa cells. The cells were paraformaldehyde fixed (4%, 20 minutes) and Triton X-100 permeabilized (0.1%, 15 minutes). The cells were incubated with ab110296 (1 µg/ml) for 2 hours at room temperature or over night at 4°C. The secondary antibody was (green) Alexa Fluor® 488 goat anti-mouse IgG (H+L) used at a 1/1000 dilution for 1 hour. 10% Goat serum was used as the blocking agent for all blocking steps. DAPI was used to stain the cell nuclei (blue). Target protein locates mainly in mitochondria.



Flow Cytometry - Anti-ACADM/MCAD antibody [3B7BH7] (ab110296)

ACADM/MCAD Flow Cytometry staining using mouse Anti-ACADM/MCAD antibody

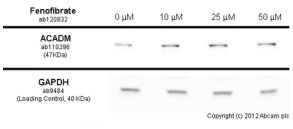
HL-60 cells were stained with 1 µg/mL ab110296 (blue) or an equal amount of an isotype control antibody (red) and analyzed by flow cytometry.



Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) - Anti-ACADM/MCAD antibody [3B7BH7] (ab110296)

ACADM/MCAD Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) staining using mouse Anti-ACADM/MCAD antibody

ACADM/MCAD immunohistochemistry in Human cerebellum visualized with ab110296 at 1/1000. MCAD immunoactivity is most intense in neuronal cell bodies, most notably in the large Purkinje cells. Note the distinctive subcellular localization of MCAD immunoreactivity in the Purkinje cell bodies.



Western blot - Anti-ACADM/MCAD antibody [3B7BH7] (ab110296)

ACADM/MCAD Western blot staining using mouse Anti-ACADM/MCAD antibody

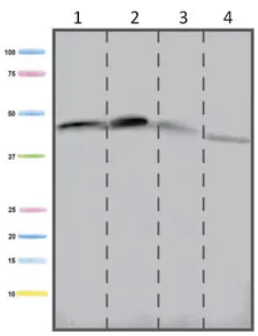
HL-60 cells were incubated at 37°C for 24h with vehicle control (0 µM) and different concentrations of fenofibrate (ab120832). Increased expression of ACADM/MCAD in HL-60 cells correlates with an increase in fenofibrate concentration, as described in literature.
Whole cell lysates were prepared with RIPA buffer (containing protease inhibitors and sodium orthovanadate), 10µg of each were loaded on the gel and the WB was run under reducing conditions. After transfer the membrane was blocked for an hour using 5% BSA before being incubated with ab110296 at 1 µg/ml and ab9484 at 1 µg/ml overnight at 4°C. Antibody binding was detected using an anti-mouse antibody conjugated to HRP (ab97040) at 1/10000 dilution and visualised using ECL development solution.

All lanes:
Western blot - Anti-ACADM/MCAD antibody [3B7BH7] (ab110296)

Developed using the ECL technique.

Performed under reducing conditions.

Predicted band size: 47 kDa



Western blot - Anti-ACADM/MCAD antibody [3B7BH7] (ab110296)

All lanes:
Western blot - Anti-ACADM/MCAD antibody [3B7BH7] (ab110296) at 0.125 µg/mL

Lane 1:
HepG2 cell lysates at 15 µg

Lane 2:
HeLa cell lysates at 15 µg

Lane 3:
H9C2 (rat cells) lysates at 15 µg

Lane 4:
MEF (mouse cells) lysates at 15 µg

Predicted band size: 47 kDa

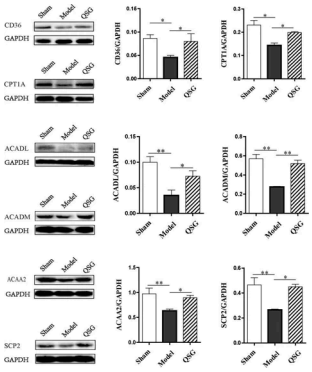


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Western blot - Anti-ACADM/MCAD antibody [3B7BH7] (ab110296)

ACADM/MCAD Western blot staining using mouse Anti-ACADM/MCAD antibody

ACADM/MCAD western blot using anti-ACADM/MCAD antibody [3B7BH7] ab110296. Publication image and figure legend from Gao, K., Zhang, J., et al., 2020, Chin Med, PubMed 32158496.

ab110296 was used in this publication in western blot. This may not be the same as the application(s) guaranteed by Abcam. For a full list of applications guaranteed by Abcam for ab110296 please see the product overview.

Effects of QSG on FAT/CD36-CPT1-FAO pathway in HF rats after AMI. The protein expressional levels of CD36, CPT1A, ACADL, ACADM, ACAA2 and SCP2 in the three groups were significantly decreased in the model group compared with the sham group. After treatment with QSG, cardiac CD36, CPT1A, ACADL, ACADM, ACAA2 and SCP2 levels were all significantly increased. n = 4 per group. Values are mean ± SE. Asterisks indicates significant differences. *p < 0.05, **p < 0.01

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