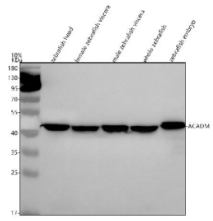
Zebrafish Acadm Antibody / Mcad (RZ1034)

Catalog No.	Formulation	Size
RZ1034	0.5mg/ml if reconstituted with 0.2ml sterile DI water	100 ug

Bulk quote request

Availability	2-3 weeks
Species Reactivity	Zebrafish
Format	Antigen affinity purified
Host	Rabbit
Clonality	Polyclonal (rabbit origin)
Isotype	Rabbit Ig
Purity	Antigen affinity chromatography
Buffer	Lyophilized from 1X PBS with 2% Trehalose
UniProt	A2CG95
Applications	Western blot: 0.5-1 ug/ml
Limitations	This Zebrafish Acadm antibody is available for research use only.



Zebrafish Acadm Antibody WB. Western blot analysis of Acadm protein using Zebrafish Acadm antibody and 1) zebrafish head, 2) female zebrafish viscera, 3) male zebrafish viscera, 4) whole zebrafish and 5) zebrafish embryo tissue lysate. Predicted molecular weight ~46 kDa.

Description

Zebrafish (Danio rerio) Acadm antibody recognizes medium-chain acyl-CoA dehydrogenase, also known as Mcad, a mitochondrial enzyme encoded by the zebrafish acadm gene located on chromosome 17. Acadm is a core component of the fatty acid beta-oxidation pathway and catalyzes the first dehydrogenation step in the breakdown of medium-chain fatty acyl-CoA substrates. This reaction supports energy production by generating FADH2 for electron transport chain activity and initiating the cyclic degradation of fatty acids into acetyl-CoA. In Danio rerio, Acadm is expressed broadly throughout embryogenesis, with strong enrichment in tissues requiring high mitochondrial activity, including developing heart, skeletal muscle, brain, notochord, and early endoderm-derived organs such as liver and pancreas. Subcellular localization is

strictly mitochondrial, where Acadm integrates into beta-oxidation metabolic networks.

Medium-chain acyl-CoA dehydrogenase is essential for energy homeostasis during early development. Zebrafish embryos depend on fatty acid metabolism as they transition from yolk-derived lipid stores toward autonomous metabolic regulation. Acadm supports ATP production required for cell proliferation, morphogenetic movements, and organ formation. Because fatty acid oxidation supplies both ATP and metabolic intermediates for biosynthetic pathways, Acadm influences developmental events such as cardiac morphogenesis, muscle differentiation, and neural maturation. Disruption of acadm function reduces mitochondrial fatty acid utilization, leading to energy deficits that impair tissue growth, reduce locomotor activity, and alter metabolic signaling pathways.

Developmental studies indicate that Acadm contributes to metabolic patterning across germ layers. In the developing heart, Acadm supports early mitochondrial maturation and contractile function by providing sustained ATP generation. In muscle tissues, its activity is required for myofibril organization and maintenance of mitochondrial membrane potential. Neural tissues also rely on Acadm-mediated beta-oxidation to support axonal growth, neurotransmission-related metabolism, and redox balance. Because metabolic state influences cell fate decisions, Acadm-dependent pathways help coordinate signaling networks such as AMPK, PPAR, and mTOR that regulate growth and differentiation.

Zebrafish models of metabolic deficiency have shown that reduced Acadm activity leads to lipid accumulation, impaired mitochondrial respiration, increased reactive oxygen species, and developmental delays. These phenotypes parallel aspects of human MCAD deficiency, a fatty acid oxidation disorder characterized by metabolic instability. Although zebrafish do not fully replicate the human disease, they offer a valuable system for studying conserved metabolic mechanisms and testing interventions that affect mitochondrial function. Acadm also participates in adaptive responses to nutrient limitation, hypoxia, and oxidative stress, processes commonly examined in zebrafish embryos exposed to environmental or genetic perturbation.

At the molecular level, Mcad forms homotetrameric complexes within the mitochondrial matrix and interacts with electron transfer flavoprotein to pass electrons into the respiratory chain. Isoform variation may arise through differential regulation of acadm during distinct developmental stages, allowing tissues with unique metabolic demands to fine-tune beta-oxidation capacity. Acadm integrates with broader metabolic pathways involving TCA cycle flux, ketone body formation, and redox maintenance, linking its enzymatic function directly to developmental energy dynamics.

This Zebrafish Acadm antibody is suitable for detecting medium-chain acyl-CoA dehydrogenase in research focused on mitochondrial metabolism, beta-oxidation pathways, cardiac and muscle development, neural maturation, metabolic stress responses, and early energy regulation in zebrafish. It supports studies examining mitochondrial dysfunction, fatty acid utilization, and developmental phenotypes resulting from altered metabolic capacity. NSJ Bioreagents provides this reagent within its zebrafish and metabolic biology antibody portfolio.

This Zebrafish antibody is part of a broader Zebrafish / Danio rerio antibody panel offered by NSJ Bioreagents.

Application Notes

Optimal dilution of the Zebrafish Acadm antibody should be determined by the researcher.

Immunogen

A synthetic peptide corresponding to a sequence at the C-terminus of zebrafish Acadm protein was used as the immunogen for the Zebrafish Acadm antibody.

Storage

After reconstitution, the Zebrafish Acadm antibody can be stored for up to one month at 4oC. For long-term, aliquot and store at -20oC. Avoid repeated freezing and thawing.

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