

**ARG57957**  
**anti-ACMSD antibody**Package: 100 µl  
Store at: -20°C

### Summary

Product Description	Rabbit Polyclonal antibody recognizes ACMSD
Tested Reactivity	Ms, Rat
Tested Application	WB
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Target Name	ACMSD
Species	Human
Immunogen	Recombinant fusion protein corresponding to aa. 1-336 of Human ACMSD (NP_612199.2).
Conjugation	Un-conjugated
Alternate Names	EC 4.1.1.45; Picolinate carboxylase; 2-amino-3-carboxymuconate-6-semialdehyde decarboxylase

### Application Instructions

Application table	Application	Dilution
	WB	1:500 - 1:2000
Application Note	* The dilutions indicate recommended starting dilutions and the optimal dilutions or concentrations should be determined by the scientist.	
Positive Control	Mouse kidney	
Observed Size	38 kDa	

### Properties

Form	Liquid
Purification	Affinity purified.
Buffer	PBS (pH 7.3), 0.02% Sodium azide and 50% Glycerol.
Preservative	0.02% Sodium azide
Stabilizer	50% Glycerol
Storage instruction	For continuous use, store undiluted antibody at 2-8°C for up to a week. For long-term storage, aliquot and store at -20°C. Storage in frost free freezers is not recommended. Avoid repeated freeze/thaw cycles. Suggest spin the vial prior to opening. The antibody solution should be gently mixed before use.
Note	For laboratory research only, not for drug, diagnostic or other use.

Bioinformation

Gene Symbol	ACMSD
Gene Full Name	aminocarboxymuconate semialdehyde decarboxylase
Background	The neuronal excitotoxin quinolinate is an intermediate in the de novo synthesis pathway of NAD from tryptophan, and has been implicated in the pathogenesis of several neurodegenerative disorders. Quinolinate is derived from alpha-amino-beta-carboxy-muconate-epsilon-semialdehyde (ACMS). ACMSD (ACMS decarboxylase; EC 4.1.1.45) can divert ACMS to a benign catabolite and thus prevent the accumulation of quinolinate from ACMS.[supplied by OMIM, Oct 2004]
Function	Converts alpha-amino-beta-carboxymuconate-epsilon-semialdehyde (ACMS) to alpha-aminomuconate semialdehyde (AMS). ACMS can be converted non-enzymatically to quinolate (QA), a key precursor of NAD, and a potent endogenous excitotoxin of neuronal cells which is implicated in the pathogenesis of various neurodegenerative disorders. In the presence of ACMSD, ACMS is converted to AMS, a benign catabolite. ACMSD ultimately controls the metabolic fate of tryptophan catabolism along the kynurenine pathway. [UniProt]
Calculated Mw	38 kDa

Images

