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Product datasheet for RC200508L2V

ATP citrate lyase (ACLY) (NM_001096) Human Tagged ORF Clone Lentiviral Particle

Product data:

Product Type:	Lentiviral Particles
Product Name:	ATP citrate lyase (ACLY) (NM_001096) Human Tagged ORF Clone Lentiviral Particle
Symbol:	ATP citrate lyase
Synonyms:	ACL; ATPCL; CLATP
Mammalian Cell Selection:	None
Vector:	pLenti-C-mGFP (PS100071)
Tag:	mGFP
ACCN:	NM_001096
ORF Size:	3303 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(RC200508).
OTI Disclaimer:	The molecular sequence of this clone aligns with the gene accession number as a point of reference only. However, individual transcript sequences of the same gene can differ through naturally occurring variations (e.g. polymorphisms), each with its own valid existence. This clone is substantially in agreement with the reference, but a complete review of all prevailing variants is recommended prior to use. More info
OTI Annotation:	This clone was engineered to express the complete ORF with an expression tag. Expression varies depending on the nature of the gene.
RefSeq:	NM_001096.2
RefSeq Size:	4450 bp
RefSeq ORF:	3306 bp
Locus ID:	47
UniProt ID:	P53396
Cytogenetics:	17q21.2
Domains:	CoA_binding, ligase-CoA
Protein Families:	Druggable Genome

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Protein Pathways: Citrate cycle (TCA cycle), Metabolic pathways

MW: 120.8 kDa

Gene Summary: ATP citrate lyase is the primary enzyme responsible for the synthesis of cytosolic acetyl-CoA in many tissues. The enzyme is a tetramer (relative molecular weight approximately 440,000) of apparently identical subunits. It catalyzes the formation of acetyl-CoA and oxaloacetate from citrate and CoA with a concomitant hydrolysis of ATP to ADP and phosphate. The product, acetyl-CoA, serves several important biosynthetic pathways, including lipogenesis and cholesterogenesis. In nervous tissue, ATP citrate-lyase may be involved in the biosynthesis of acetylcholine. Multiple transcript variants encoding distinct isoforms have been identified for this gene. [provided by RefSeq, Dec 2014]