

Product datasheet for MR208943L4V

OriGene Technologies, Inc.

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Prkag2 (NM_001170556) Mouse Tagged ORF Clone Lentiviral Particle

Product data:

Product Type: Lentiviral Particles

Product Name: Prkag2 (NM 001170556) Mouse Tagged ORF Clone Lentiviral Particle

Symbol: Prkag2

Synonyms: 2410051C13Rik; AAKG; AAKG2; Al854673; H91620p; WPWS

Mammalian Cell

Selection:

Puromycin

Vector: pLenti-C-mGFP-P2A-Puro (PS100093)

Tag: mGFP

ACCN: NM_001170556

ORF Size: 1701 bp

ORF Nucleotide

The ORF insert of this clone is exactly the same as(MR208943).

Sequence:

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: <u>NM 001170556.1</u>, <u>NP 001164027.1</u>

 RefSeq Size:
 2258 bp

 RefSeq ORF:
 981 bp

 Locus ID:
 108099

 UniProt ID:
 Q91WG5

Cytogenetics: 5 A3







Gene Summary:

AMP/ATP-binding subunit of AMP-activated protein kinase (AMPK), an energy sensor protein kinase that plays a key role in regulating cellular energy metabolism. In response to reduction of intracellular ATP levels, AMPK activates energy-producing pathways and inhibits energy-consuming processes: inhibits protein, carbohydrate and lipid biosynthesis, as well as cell growth and proliferation. AMPK acts via direct phosphorylation of metabolic enzymes, and by longer-term effects via phosphorylation of transcription regulators. Also acts as a regulator of cellular polarity by remodeling the actin cytoskeleton; probably by indirectly activating myosin. Gamma non-catalytic subunit mediates binding to AMP, ADP and ATP, leading to activate or inhibit AMPK: AMP-binding results in allosteric activation of alpha catalytic subunit (PRKAA1 or PRKAA2) both by inducing phosphorylation and preventing dephosphorylation of catalytic subunits. ADP also stimulates phosphorylation, without stimulating already phosphorylated catalytic subunit. ATP promotes dephosphorylation of catalytic subunit, rendering the AMPK enzyme inactive (By similarity).[UniProtKB/Swiss-Prot Function]