

## OriGene Technologies, Inc.

9620 Medical Center Drive, Ste 200 Rockville, MD 20850, US Phone: +1-888-267-4436 https://www.origene.com techsupport@origene.com EU: info-de@origene.com CN: techsupport@origene.cn

## Product datasheet for RC206409L4V

## SLC19A3 (NM\_025243) Human Tagged ORF Clone Lentiviral Particle

## **Product data:**

Product Type:	Lentiviral Particles
Product Name:	SLC19A3 (NM_025243) Human Tagged ORF Clone Lentiviral Particle
Symbol:	SLC19A3
Synonyms:	BBGD; THMD2; thTr-2; THTR2
Mammalian Cell Selection:	Puromycin
Vector:	pLenti-C-mGFP-P2A-Puro (PS100093)
Tag:	mGFP
ACCN:	NM_025243
ORF Size:	1488 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(RC206409).
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. <u>More info</u>
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 025243.3</u>
RefSeq Size:	3775 bp
RefSeq ORF:	1491 bp
Locus ID:	80704
UniProt ID:	Q9BZV2
Cytogenetics:	2q36.3
Domains:	Folate_carrier
Protein Families:	Transmembrane



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	SLC19A3 (NM_025243) Human Tagged ORF Clone Lentiviral Particle – RC206409L4V
MW:	55.7 kDa
Gene Summary:	This gene encodes a ubiquitously expressed transmembrane thiamine transporter that lacks folate transport activity. Mutations in this gene cause biotin-responsive basal ganglia disease (BBGD); a recessive disorder manifested in childhood that progresses to chronic encephalopathy, dystonia, quadriparesis, and death if untreated. Patients with BBGD have bilateral necrosis in the head of the caudate nucleus and in the putamen. Administration of high doses of biotin in the early progression of the disorder eliminates pathological symptoms while delayed treatment results in residual paraparesis, mild cognitive disability, or dystonia. Administration of thiamine is ineffective in the treatment of this disorder. Experiments have failed to show that this protein can transport biotin. Mutations in this gene also cause a Wernicke's-like encephalopathy.[provided by RefSeq, Jan 2010]

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