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 RC208752L1V

BCL10 (NM_003921) Human Tagged ORF Clone Lentiviral Particle

Product data:

Product Type:	Lentiviral Particles
Product Name:	BCL10 (NM_003921) Human Tagged ORF Clone Lentiviral Particle
Symbol:	BCL10
Synonyms:	c-E10; CARMEN; CIPER; CLAP; IMD37; mE10
Mammalian Cell Selection:	None
Vector:	pLenti-C-Myc-DDK (PS100064)
Tag:	Myc-DDK
ACCN:	NM_003921
ORF Size:	699 bp
ORF Nucleotide Sequence:	`The ORF insert of this clone is exactly the same as(RC208752).`
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_003921.3
RefSeq Size:	3118 bp
RefSeq ORF:	702 bp
Locus ID:	8915
UniProt ID:	O95999
Cytogenetics:	1p22.3
Domains:	CARD
Protein Families:	Druggable Genome

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Protein Pathways: B cell receptor signaling pathway, T cell receptor signaling pathway

MW: 26.3 kDa

Gene Summary: This gene was identified by its translocation in a case of mucosa-associated lymphoid tissue (MALT) lymphoma. The protein encoded by this gene contains a caspase recruitment domain (CARD), and has been shown to induce apoptosis and to activate NF-kappaB. This protein is reported to interact with other CARD domain containing proteins including CARD9, 10, 11 and 14, which are thought to function as upstream regulators in NF-kappaB signaling. This protein is found to form a complex with MALT1, a protein encoded by another gene known to be translocated in MALT lymphoma. MALT1 and this protein are thought to synergize in the activation of NF-kappaB, and the deregulation of either of them may contribute to the same pathogenetic process that leads to the malignancy. Alternative splicing results in multiple transcript variants. [provided by RefSeq, Mar 2016]