

## Product datasheet for MR216034L3V

## 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

## Glrx2 (NM\_001038592) Mouse Tagged ORF Clone Lentiviral Particle

**Product data:** 

**Product Type:** Lentiviral Particles

**Product Name:** Glrx2 (NM\_001038592) Mouse Tagged ORF Clone Lentiviral Particle

Symbol: Glrx2

**Synonyms:** 1700010P22Rik; AI645710; Grx2

Mammalian Cell

Selection:

Puromycin

**Vector:** pLenti-C-Myc-DDK-P2A-Puro (PS100092)

Tag: Myc-DDK

**ACCN:** NM\_001038592

ORF Size: 468 bp

**ORF Nucleotide** 

The ORF insert of this clone is exactly the same as(MR216034).

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:** NM 001038592.1, NP 001033681.1

 RefSeq Size:
 3603 bp

 RefSeq ORF:
 471 bp

 Locus ID:
 69367

 UniProt ID:
 Q923X4

Cytogenetics: 1 62.53 cM







## **Gene Summary:**

Glutathione-dependent oxidoreductase that facilitates the maintenance of mitochondrial redox homeostasis upon induction of apoptosis by oxidative stress. Involved in response to hydrogen peroxide and regulation of apoptosis caused by oxidative stress. Acts as a very efficient catalyst of monothiol reactions because of its high affinity for protein glutathione-mixed disulfides. Can receive electrons not only from glutathione (GSH), but also from thioredoxin reductase supporting both monothiol and dithiol reactions. Efficiently catalyzes both glutathionylation and deglutathionylation of mitochondrial complex I, which in turn regulates the superoxide production by the complex. Overexpression decreases the susceptibility to apoptosis and prevents loss of cardiolipin and cytochrome c release. [UniProtKB/Swiss-Prot Function]