

## Product datasheet for **RC219065L4V**

### Factor IX (F9) (NM\_000133) Human Tagged ORF Clone Lentiviral Particle

#### Product data:

Product Type:	Lentiviral Particles
Product Name:	Factor IX (F9) (NM_000133) Human Tagged ORF Clone Lentiviral Particle
Symbol:	Factor IX
Synonyms:	F9 p22; FIX; HEMB; P19; PTC; THPH8
Mammalian Cell Selection:	Puromycin
Vector:	pLenti-C-mGFP-P2A-Puro (PS100093)
Tag:	mGFP
ACCN:	NM_000133
ORF Size:	1383 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(RC219065).
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. <a href="#">More info</a>
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:	<a href="#">NM_000133.2</a>
RefSeq Size:	2804 bp
RefSeq ORF:	1386 bp
Locus ID:	2158
UniProt ID:	<a href="#">P00740</a>
Cytogenetics:	Xq27.1
Domains:	GLA, Tryp_SPc, EGF_CA, EGF, EGF
Protein Families:	Druggable Genome, Protease, Secreted Protein



[View online »](#)

**Protein Pathways:** Complement and coagulation cascades

**MW:** 51.78 kDa

**Gene Summary:** This gene encodes vitamin K-dependent coagulation factor IX that circulates in the blood as an inactive zymogen. This factor is converted to an active form by factor XIa, which excises the activation peptide and thus generates a heavy chain and a light chain held together by one or more disulfide bonds. The role of this activated factor IX in the blood coagulation cascade is to activate factor X to its active form through interactions with Ca<sup>2+</sup> ions, membrane phospholipids, and factor VIII. Alterations of this gene, including point mutations, insertions and deletions, cause factor IX deficiency, which is a recessive X-linked disorder, also called hemophilia B or Christmas disease. Alternative splicing results in multiple transcript variants encoding different isoforms that may undergo similar proteolytic processing. [provided by RefSeq, Sep 2015]