

Product datasheet for **RC210683L4V**

PYGL (NM_002863) Human Tagged ORF Clone Lentiviral Particle

Product data:

Product Type:	Lentiviral Particles
Product Name:	PYGL (NM_002863) Human Tagged ORF Clone Lentiviral Particle
Symbol:	PYGL
Synonyms:	GSD6
Mammalian Cell Selection:	Puromycin
Vector:	pLenti-C-mGFP-P2A-Puro (PS100093)
Tag:	mGFP
ACCN:	NM_002863
ORF Size:	2541 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(RC210683).
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_002863.3
RefSeq Size:	2859 bp
RefSeq ORF:	2544 bp
Locus ID:	5836
UniProt ID:	P06737
Cytogenetics:	14q22.1
Domains:	phosphorylase
Protein Families:	Druggable Genome



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Protein Pathways: Insulin signaling pathway, Starch and sucrose metabolism

MW: 97.1 kDa

Gene Summary: This gene encodes a homodimeric protein that catalyses the cleavage of alpha-1,4-glycosidic bonds to release glucose-1-phosphate from liver glycogen stores. This protein switches from inactive phosphorylase B to active phosphorylase A by phosphorylation of serine residue 15. Activity of this enzyme is further regulated by multiple allosteric effectors and hormonal controls. Humans have three glycogen phosphorylase genes that encode distinct isozymes that are primarily expressed in liver, brain and muscle, respectively. The liver isozyme serves the glyceamic demands of the body in general while the brain and muscle isozymes supply just those tissues. In glycogen storage disease type VI, also known as Hers disease, mutations in liver glycogen phosphorylase inhibit the conversion of glycogen to glucose and results in moderate hypoglycemia, mild ketosis, growth retardation and hepatomegaly. Alternative splicing results in multiple transcript variants encoding different isoforms.[provided by RefSeq, Feb 2011]