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Product datasheet for RC202172L2V

Cytochrome P450 Reductase (POR) (NM_000941) Human Tagged ORF Clone Lentiviral Particle

Product data:

Product Type:	Lentiviral Particles
Product Name:	Cytochrome P450 Reductase (POR) (NM_000941) Human Tagged ORF Clone Lentiviral Particle
Symbol:	Cytochrome P450 Reductase
Synonyms:	CPR; CYPOR; P450R
Mammalian Cell Selection:	None
Vector:	pLenti-C-mGFP (PS100071)
Tag:	mGFP
ACCN:	NM_000941
ORF Size:	2040 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(RC202172).
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_000941.2, NP_000932.1
RefSeq Size:	2509 bp
RefSeq ORF:	2043 bp
Locus ID:	5447
UniProt ID:	P16435
Cytogenetics:	7q11.23
Domains:	flavodoxin, NAD_binding_1, FAD_binding_1

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Protein Families:	Druggable Genome, P450, Transmembrane
MW:	77.1 kDa
Gene Summary:	This gene encodes an endoplasmic reticulum membrane oxidoreductase that is essential for multiple metabolic processes, including reactions catalyzed by cytochrome P450 proteins for metabolism of steroid hormones, drugs and xenobiotics. The encoded protein has a flavin adenine dinucleotide (FAD)-binding domain and a flavodoxin-like domain which bind two cofactors, FAD and FMN, that allow it to donate electrons directly from NADPH to all microsomal P450 enzymes. Mutations in this gene cause a complex set of disorders, including apparent combined P450C17 and P450C21 deficiency, amenorrhea and disordered steroidogenesis, congenital adrenal hyperplasia and Antley-Bixler syndrome, that resemble those caused by defects in steroid metabolizing enzymes such as aromatase, 21-hydroxylase, and 17 alpha-hydroxylase. [provided by RefSeq, Aug 2020]