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Product datasheet for RC207796L2V

HSD11B2 (NM_000196) Human Tagged ORF Clone Lentiviral Particle

Product data:

Product Type:	Lentiviral Particles
Product Name:	HSD11B2 (NM_000196) Human Tagged ORF Clone Lentiviral Particle
Symbol:	HSD11B2
Synonyms:	AME; AME1; HSD2; HSD11K; SDR9C3
Mammalian Cell Selection:	None
Vector:	pLenti-C-mGFP (PS100071)
Tag:	mGFP
ACCN:	NM_000196
ORF Size:	1215 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(RC207796).
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_000196.2, NP_000187.2
RefSeq Size:	1939 bp
RefSeq ORF:	1218 bp
Locus ID:	3291
UniProt ID:	P80365
Cytogenetics:	16q22.1
Protein Families:	Druggable Genome
Protein Pathways:	Androgen and estrogen metabolism, C21-Steroid hormone metabolism, Metabolic pathways

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MW: 44.1 kDa

Gene Summary: There are at least two isozymes of the corticosteroid 11-beta-dehydrogenase, a microsomal enzyme complex responsible for the interconversion of cortisol and cortisone. The type I isozyme has both 11-beta-dehydrogenase (cortisol to cortisone) and 11-oxoreductase (cortisone to cortisol) activities. The type II isozyme, encoded by this gene, has only 11-beta-dehydrogenase activity. In aldosterone-selective epithelial tissues such as the kidney, the type II isozyme catalyzes the glucocorticoid cortisol to the inactive metabolite cortisone, thus preventing illicit activation of the mineralocorticoid receptor. In tissues that do not express the mineralocorticoid receptor, such as the placenta and testis, it protects cells from the growth-inhibiting and/or pro-apoptotic effects of cortisol, particularly during embryonic development. Mutations in this gene cause the syndrome of apparent mineralocorticoid excess and hypertension. [provided by RefSeq, Feb 2010]