

## Product datasheet for **RC211069L4V**

### **FUT3 (NM\_000149) Human Tagged ORF Clone Lentiviral Particle**

#### **Product data:**

Product Type:	Lentiviral Particles
Product Name:	FUT3 (NM_000149) Human Tagged ORF Clone Lentiviral Particle
Symbol:	FUT3
Synonyms:	CD174; FT3B; FucT-III; LE; Les
Mammalian Cell Selection:	Puromycin
Vector:	pLenti-C-mGFP-P2A-Puro (PS100093)
Tag:	mGFP
ACCN:	NM_000149
ORF Size:	1083 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(RC211069).
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_000149.1</a>
RefSeq Size:	2607 bp
RefSeq ORF:	1086 bp
Locus ID:	2525
UniProt ID:	<a href="#">P21217</a>
Cytogenetics:	19p13.3
Protein Pathways:	Glycosphingolipid biosynthesis - lacto and neolacto series, Metabolic pathways
MW:	42.1 kDa



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**Gene Summary:**

The Lewis histo-blood group system comprises a set of fucosylated glycosphingolipids that are synthesized by exocrine epithelial cells and circulate in body fluids. The glycosphingolipids function in embryogenesis, tissue differentiation, tumor metastasis, inflammation, and bacterial adhesion. They are secondarily absorbed to red blood cells giving rise to their Lewis phenotype. This gene is a member of the fucosyltransferase family, which catalyzes the addition of fucose to precursor polysaccharides in the last step of Lewis antigen biosynthesis. It encodes an enzyme with alpha(1,3)-fucosyltransferase and alpha(1,4)-fucosyltransferase activities. Mutations in this gene are responsible for the majority of Lewis antigen-negative phenotypes. Differences in the expression of this gene are associated with host susceptibility to viral infection. [provided by RefSeq, Aug 2020]