

## **Product datasheet for MR219346L4V**

### OriGene Technologies, Inc.

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# Aff1 (NM\_133919) Mouse Tagged ORF Clone Lentiviral Particle

### **Product data:**

Product Type: Lentiviral Particles

**Product Name:** Aff1 (NM\_133919) Mouse Tagged ORF Clone Lentiviral Particle

Symbol: Aff

**Synonyms:** 9630032B01Rik; Af; Af4; AW319193; Mllt; Mllt2h; R; Rob

Mammalian Cell

Selection:

Puromycin

**Vector:** pLenti-C-mGFP-P2A-Puro (PS100093)

Tag: mGFP

**ACCN:** NM\_133919 **ORF Size:** 3654 bp

**ORF Nucleotide** 

The ORF insert of this clone is exactly the same as(MR219346).

Sequence:
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.

**RefSeg:** NM 133919.3, NP 598680.3

RefSeq Size:8323 bpRefSeq ORF:3657 bpLocus ID:17355

**Cytogenetics:** 5 50.45 cM







### **Gene Summary:**

This gene encodes a member of the AF4/ lymphoid nuclear protein related to the Fragile X E syndrome (FRAXE) family of proteins, which have been implicated in human childhood lymphoblastic leukemia, fragile chromosome X intellectual disability, and ataxia. It is the prevalent mixed-lineage leukemia fusion gene associated with spontaneous acute lymphoblastic leukemia. Members of this family have three conserved domains: an N-terminal homology domain, an AF4/ lymphoid nuclear protein domain, and a C-terminal homology domain. Knockout of the mouse gene by homologous recombination severely affects early events in lymphopoiesis, including precursor proliferation or recruitment, but is dispensable for terminal differentiation. In addition, an autosomal dominant missense mutation results in several phenotypes including ataxia and adult-onset Purkinje cell loss in the cerebellum, indicating a role in Purkinje cell maintenance and function. Alternative splicing results in multiple transcript variants. [provided by RefSeq, Jul 2017]