

pMCs-IRES-GFP Retroviral Vector

CATALOG NUMBER: RTV-040

STORAGE: -20°C

QUANTITY AND CONCENTRATION: 10 µg at 0.25 µg/µL in TE

Background

Retroviruses are efficient tools for delivering heritable genes into the genome of dividing cells. Most retrovirus vectors including pBABE and pMXs are based on Moloney murine leukemia virus (MMLV). MMLV-based vectors usually are silenced in immature cells including embryonic carcinoma (EC) cells and embryonic stem (ES) cells, and possibly hematopoietic stem cells. PCC4-cell-passaged myeloproliferative sarcoma virus (PCMV) are mutants of MMLV and can stably express genes in immature cells including ES cells.

Cell Biolabs' pMCs-IRES-GFP retroviral vector (also known as pMCs-IG) includes hybrid LTRs containing elements from both MMLV and PCMV, and it's capable of expressing genes in both EC and ES cells. The vector provides the viral package signal, transcription and processing elements, and MCS for cloning of a target gene. The viral *env* gene, produced by the package cell line, encodes the envelope protein, which determines the viral infectivity range. Transfection into a package cell line produces high-titer, replication-incompetent viruses. In addition to transfer and expression of exogenous genes in mammalian cells, recently, retroviruses have been used to express silencing RNAs (siRNA) to decrease the expression of target genes both *in vitro* and *in vivo*.

The vector contains the ampicillin-resistance gene, LTRs, package signal and MCS for cloning of your gene of interest (Figure 1).

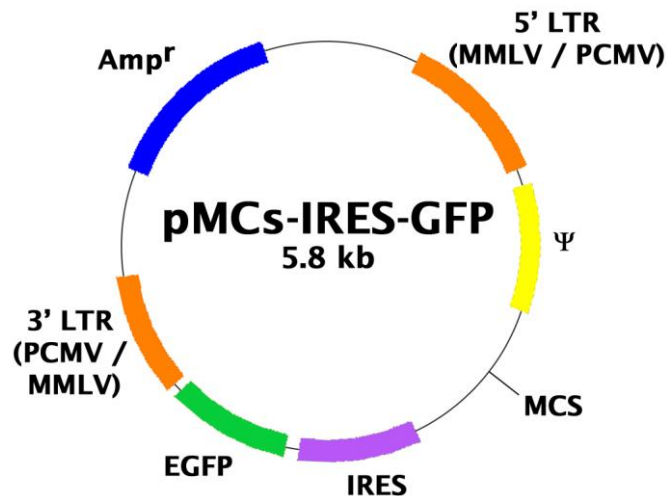


Figure 1. Schematic representation of pMCs-IRES-GFP retroviral vector.

MCS:

- Enzyme Sites: 5'-BamHI, EcoRI, XhoI, NotI, SnaBI-3'
- MCS Sequence:
TTAAGGATCCCAGTGTGGTGGTACGGGAATTCCTGCAGGCCTCGAGGGCCGGCGCGC
CGCGGCCGCTACGTAAATT---IRES---GFP---

Safety Consideration

Remember that you will be working with samples containing infectious virus. Follow the recommended NIH guidelines for all materials containing BSL-2 organisms. Always wear gloves, use filtered tips and work under a biosafety hood.

References

1. Kitamura T., *et al.*, (2003) *Exp. Hematol.* **31**, 1007-1014.

Recent Product Citations

1. Akamatsu, M. *et al.* (2019). Conversion of antigen-specific effector/memory T cells into Foxp3-expressing Treg cells by inhibition of CDK8/19. *Sci Immunol.* **4**(40). pii: eaaw2707. doi: 10.1126/sciimmunol.aaw2707.
2. Shirai, A., *et al.* (2017). Impact of nucleic acid and methylated H3K9 binding activities of Suv39h1 on its heterochromatin assembly. *Elife.* **6**. pii: e25317. doi: 10.7554/eLife.25317.
3. Muramatsu, D. *et al.* (2016). Pericentric H3K9me3 formation by HP1 interaction-defective histone methyltransferase Suv39h1. *Cell Struct. and Funct.* doi:10.1247/csf.16013.
4. Zhao, C. *et al.* (2016). Mice lacking the intracellular cation channel TRIC-B have compromised collagen production and impaired bone mineralization. *Sci. Signal.* doi:10.1126/scisignal.aad9055.
5. Nakaya, Y., *et al.* (2014). Efficacy of NS-018, a potent and selective JAK2/Src inhibitor, in primary cells and mouse models of myeloproliferative neoplasms. *Blood Cancer J.* **4**: e174.
6. Malicet, C. *et al.* (2011). Distinct properties of human HMGN5 reveal a rapidly evolving but functionally conserved nucleosome binding protein. *Mol. Cell. Biol.* **31**:2742-2755.

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