

pMXs- Puro Retroviral Vector

CATALOG NUMBER: RTV-012

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. Cell Biolabs' pMXs-Puro retroviral vector is based on Moloney murine leukemia virus (MMLV). 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, MMLV LTRs, package signal and MCS for cloning of your gene of interest (Figure 1).

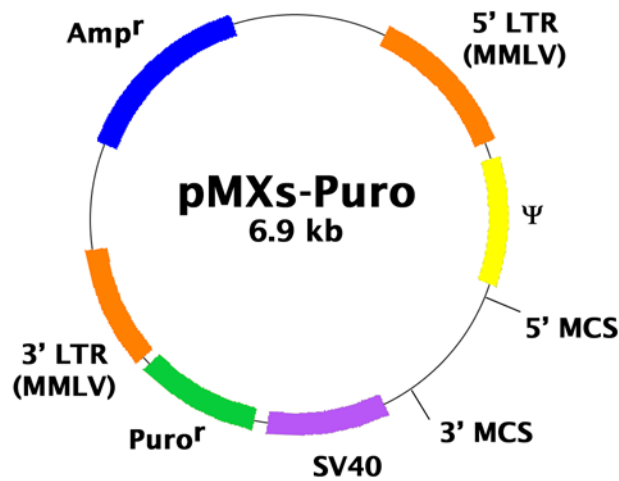


Figure 1. Schematic representation of pMXs-Puro retroviral vector.

5'-MCS:

- Enzyme Sites: 5'-PacI, BamHI, EcoRI-3'
- MCS Sequence: TTAATTAAGGATCCAGTGTGGTGGTACGGGAATTC AAGCTTGATC

3'-MCS:

- Enzyme Sites: 5'-EcoRI, XhoI, NotI-3'
- MCS Sequence:
GGCGGAATTCCAGCTGAGCGCCGGTTCGCTACCATTACCAGTTGGTCTGGTGTCAAAAA
TAATAATAACCGGGCAGGCCATGTCTGCCCGTATTTCGCGTAAGGAAATCCATTATGT
ACTATTTAAACTCGAGCGGCCGCCAGCACAGTGGTTCGAC---SV40---puro-GTCGAC---

Note: For optimal expression, both 5' MCS and 3' MCS should be used to clone gene of interest and replace the stuffer sequence (partial LacZ) between them.

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. Li, Y.Y. *et al.* (2017). Exome and genome sequencing of nasopharynx cancer identifies NF- κ B pathway activating mutations. *Nat Commun.* **8**:14121. doi: 10.1038/ncomms14121.
2. Mori, S. *et al.* (2017). Human Papillomavirus 16 E6 Upregulates APOBEC3B via the TEAD Transcription Factor. *J Virol.* **91**(6). pii: e02413-16. doi: 10.1128/JVI.02413-16.
3. Furuyama, W. *et al.* (2016). Fc γ -receptor IIa-mediated Src Signaling Pathway is Essential for the Antibody-Dependent Enhancement of Ebola Virus Infection. *PLOS Pathogens.* **12**(12):e1006139. doi: 10.1371/journal.ppat.1006139.
4. Yamashita, S. *et al.* (2016). Mitochondrial division occurs concurrently with autophagosome formation but independently of Dro1 during mitophagy. *J. Cell Biol.* **215**:649-665.
5. Arisawa, K. *et al.* (2016). Saturated fatty acid in the phospholipid monolayer contributes to the formation of large lipid droplets. *Biochem. Biophys. Res. Comm.* **480**:641-647.
6. Jin, W. J. *et al.* (2016). Notch2 signaling promotes osteoclast resorption via activation of PYK2. *Cell Signal.* **28**:357-365. Hedberg, M. L. *et al.* (2015). Genetic landscape of metastatic and recurrent head and neck squamous cell carcinoma. *J Clin Invest.* doi:10.1172/JCI82066.
7. Yeon, J. T. *et al.* (2015). Arginase 1 is a negative regulator of osteoclast differentiation. *Amino Acids.* doi:10.1007/s00726-015-2112-0.
8. Yeon, J. T. *et al.* (2015). KCNK1 inhibits osteoclastogenesis by blocking the Ca²⁺ oscillation and JNK-NFATc1 signaling axis. *J Cell Sci.* **128**:3411-3419.
9. Mori, S. *et al.* (2015). Identification of APOBEC3B promoter elements responsible for activation by human papillomavirus type 16 E6. *Biochem Biophys Res Commun.* doi:10.1016/j.bbrc.2015.03.068.
10. Lui, V. W. *et al.* (2014). Frequent mutation of receptor protein tyrosine phosphatases provides a mechanism for STAT3 hyperactivation in head and neck cancer. *Proc Natl Acad Sci U S A.* **111**:1114-1119.
11. Chan, E. C. *et al.* (2014). Mastocytosis associated with a rare germline KIT K509I mutation displays a well-differentiated mast cell phenotype. *J Allergy Clin Immunol.* **134**:178-187.

12. Chmielecki, J. et al. (2014). Comprehensive genomic profiling of pancreatic acinar cell carcinomas identifies recurrent RAF fusions and frequent inactivation of DNA repair genes. *Cancer Discov.* **4**:1398-1405.
13. Parikh, C. et al. (2012). Disruption of PH–kinase domain interactions leads to oncogenic activation of AKT in human cancers. *PNAS.* **109**:19368-19373.
14. Iwai, A. et al. (2010). Influenza A virus polymerase inhibits type I interferon induction by binding to interferon β promoter stimulator 1. *J. Biol. Chem.* **285**:32064-32074

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