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Feline Immunodeficiency Virus (FIV) Based Vectors and Cell Lines

The FELIX vector system, like the PHOENIX MLV-based packaging system, produces high-titre retroviral particles capable of stably transducing a wide variety of target cells with a gene of interest. Whereas the utility of the PHOENIX system was limited by its inability to transduce non-dividing cells, the FELIX vector system efficiently transfers genes to most cell types, dividing or nondividing. As the FELIX vectors are based on Feline Immunodeficiency Virus (FIV), they can deliver genes to non-dividing primary cells, as well as to dividing cells and cell lines. In contrast to adenoviral and lipid based gene delivery systems, genes transferred by FIV are stably integrated into the target cell genome and passed on to subsequent generations of the target cell. FELIX vectors can transfer inserts of up to 8kB - almost double the capacity of an AAV vector. The FELIX system can be used under BSL2 conditions as the vectors have been engineered to maximize safety. The FELIX vector system includes transfer vectors which carry the gene of interest, a vector which produces the FIV structural proteins, and a third vector which produces VSV-G envelope. The system also includes 293T cells, T-antigen transformed human embryonic kidney cells, which, due to their transfectability, serve as an ideal viral producer line.

Applications

- Stable transfer of genes of up to 8kB in size to non-dividing or dividing cells for in vitro studies or in vivo gene therapy.

Advantages

- Ability to transduce non-dividing and terminally differentiated cells.
- Gene of interest is permanently transferred to target cell

- Three plasmid system provides safety by minimizing the chances of recombination.
- Derived from FIV, which is not known to be a human pathogen.
- Felix vectors can accommodate inserts up to 8kB.
- CTE (Cytoplasmic Transport Element)-based vectors allow elimination of all viral proteins except Gag-Pol.
- FELIX vectors are not packaged by HIV, preventing vector mobilization in human patients.

Publications

- Curran, et al. Molecular Therapy (1): 31-38, 2000.

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