

Supplementary data

Figure S-1. Viral incorporation and cellular processing of the EGF-chimeric Env proteins (E1 to E6). Viral incorporation was analysed by Western blotting (WB) of the virions (**A** and **B**). Cellular processing was examined by immunoprecipitation (IP) and subsequent Western blotting of the cell lysates (**C** and **D**). E1 to E6 indicate either virions or lysates from the TELCeB6 cells stably transfected with each of the EGF-chimeric *env* plasmids and cultured at 32°C. SDS-gels of 8% polyacrylamide were used. Rabbit anti-human EGF (Santa Cruz Biotechnology) and anti-R peptide were used at 1:500 and 1:1,000 dilutions, respectively, for Western blotting. The lower portion of the blot for panel A was used for detection of the Gag capsid protein (CA). Pr, Env precursor.

An SU protein was detected only in the virions from cells transfected with the E3 *env* plasmid (**A**). It had a larger molecular size than the wild-type SU protein (lane MO) (**A**) and reacted with the anti-EGF (**B**). When the cell lysates were subjected to immunoprecipitation and Western blotting using anti-SU, all the EGF-chimeric Env proteins appeared as two bands (**C**). When the cell lysates were immunoprecipitated by anti-SU and subjected to Western blotting using anti-R peptide, only the upper band was detectable for the E3 Env protein, while for other EGF-chimeric Env proteins, both upper and lower

bands were detectable (**D**). These results indicate that the E3 Env precursor undergoes proteolytic processing to the SU and TM proteins, but that other Env precursors do not. The lower bands in lanes E1, E2, E4, E5, and E6 probably represent underglycosylated Env precursors.

Table S-I. Transduction titers of the E3 vector for various cell lines. NE13 cell line was derived from NIH 3T3 cells after stable transfection with an expression plasmid for the EGF receptor (pCO12-EGFR) (Velu, T.J. *et al.* (1987) *Science*, **238**, 1408-1410). 3T3-E and NE13-E were prepared by infection of NIH 3T3 and NE13 cells, respectively, with Mo-MLV.

The E3 vector transduced mouse NIH 3T3 cells with a similar titer to that of the MO vector. Similar to the MO vector, transduction by the E3 vector was inhibited in NIH 3T3 cells chronically infected with Mo-MLV (3T3-E), indicating that the E3 protein retained affinity towards the ecotropic MLV receptor (mCAT1) and the ability to carry out post-receptor binding function. The MO vector transduced NE13 cells with a similar titer to that for NIH 3T3 cells, whereas the E3 vector transduced NE13 cells with a much lower titer (1.8%) than that for the NIH 3T3 cells. Transduction of NE13 cells by the E3 vector was completely interfered by Mo-MLV (NE13-E), indicating that mCAT1-independent transduction did not occur. These results suggest the presence of a transduction-inhibitory interaction between the E3 protein and the EGF receptor on NE13 cells.

Figure S-1

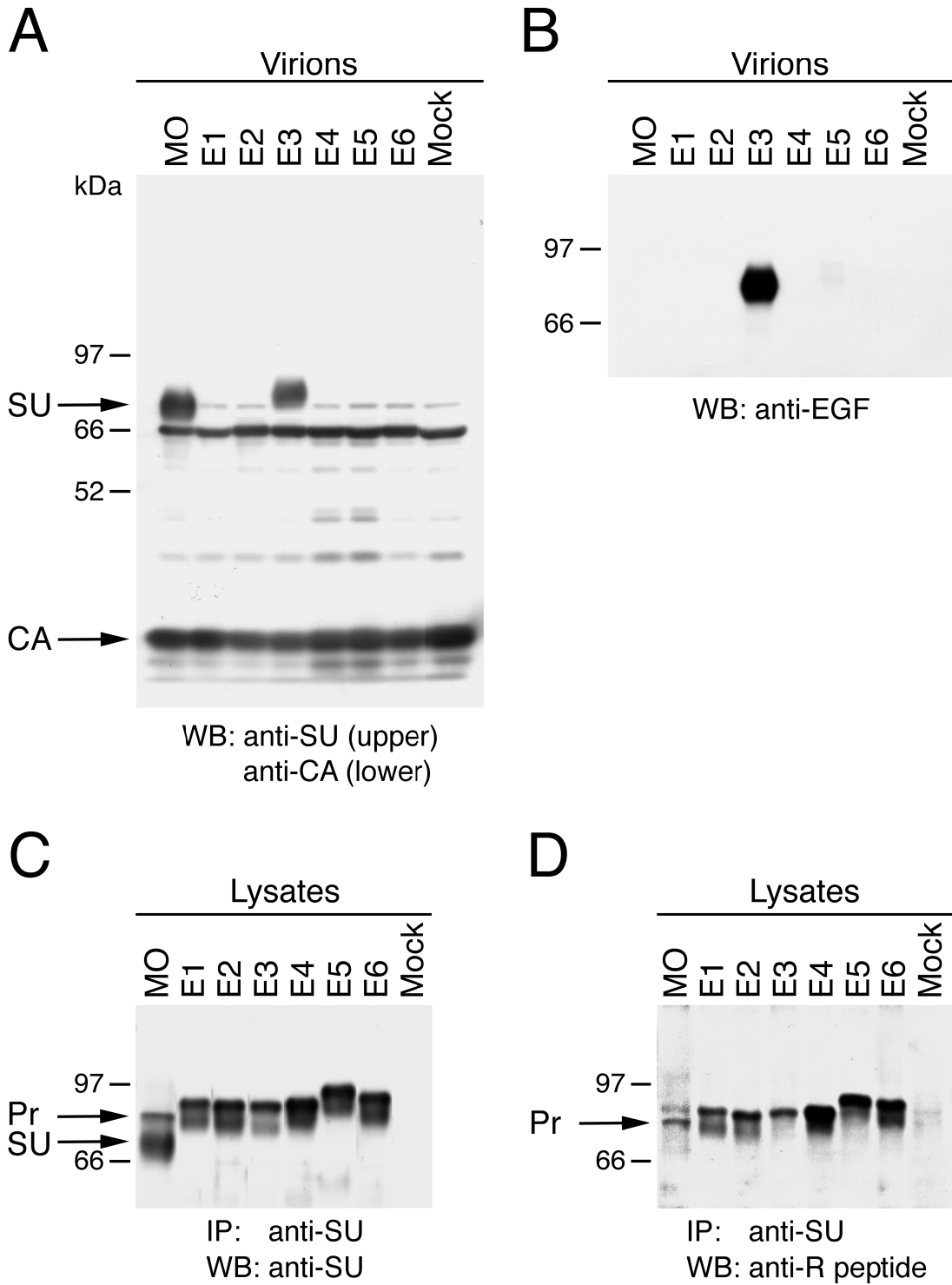


Table S-I. Transduction titers of the E3 vector for various cell lines

Env ^a	Transduction titer (CFU/ml) for the cell line			
	NIH 3T3	3T3-E	NE13	NE13-E
E3	4.1×10^6	<10	7.5×10^4	<10
MO	1.7×10^7	<10	7.4×10^6	<10

^a Env protein which pseudotyped the *lacZ* vector.