Fig S1

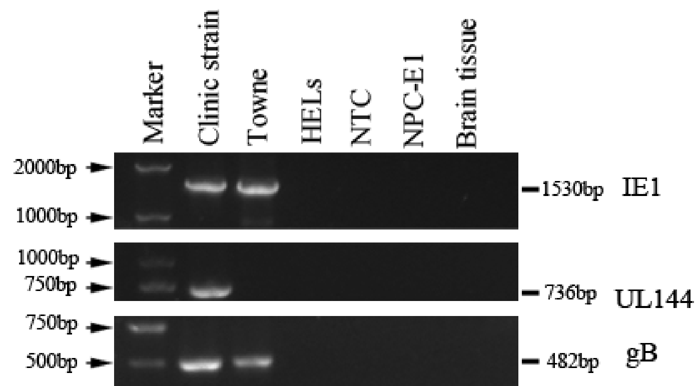

Fig.S1 HCMV infection was excluded from the brain tissue and isolated NPCs. Obtained brain tissue and the isolated NPCs at P3 before using and freezing was examined for the presence of HCMV DNA by PCR. DNA was extracted from the brain tissue and NPCs. Specific primers for IE1 (forward 5’CTGTATGTGACCCATGTGCTT3’, reverse 5’GGGGCAACTTCCTCTATCTCA3’, product size is 1530bp), UL144 (clinic HCMV isolate specific, forward 5’CGTATTACAAACCGCGGAGAGGAT3’, reverse 5’CTCAGACACGGTTCCGTAAAGTG-3’, product size is 736bp) and gB (forward 5’GGAAACGTGTCCGTCTTTGA3’, reverse 5’ GAGTAGCAGCGTCCTGGCGA3’, product size is 482bp), were applied for detection of HCMV DNA, Towne virus and clinical strain (TR) were used as positive control, and regular cultured HELs used as negative control; none template control (NTC) used as a negative control for the PCR reaction system. No HCMV DNA was detected and representatives (the brain tissue and the isolated NPC-E1) are shown.

Fig S2

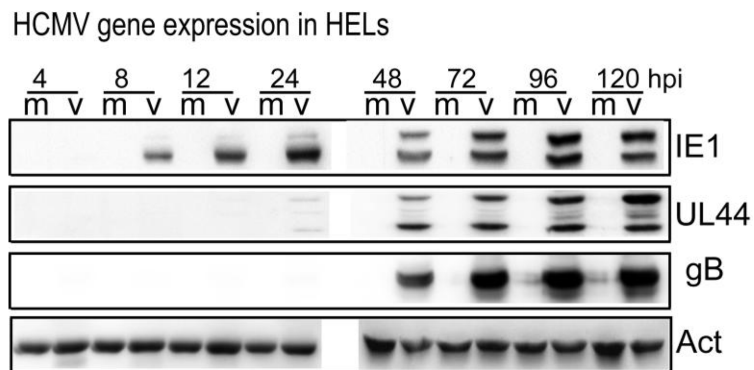

Fig. S2 Viral gene expression and timing in HELs. Serum starved HELs were reseeded, infected with HCMV (Towne) at an MOI of 3 as described (Luo et al 2006，2007), cells were harvested at the indicated times pi. The representatives of IE (IE1/IE2), E (UL44) and L (gB) stages were detected and results are shown. The specific antigens are indicated at right.