Chiu et al., https://doi.org/10.1083/jcb.201702013

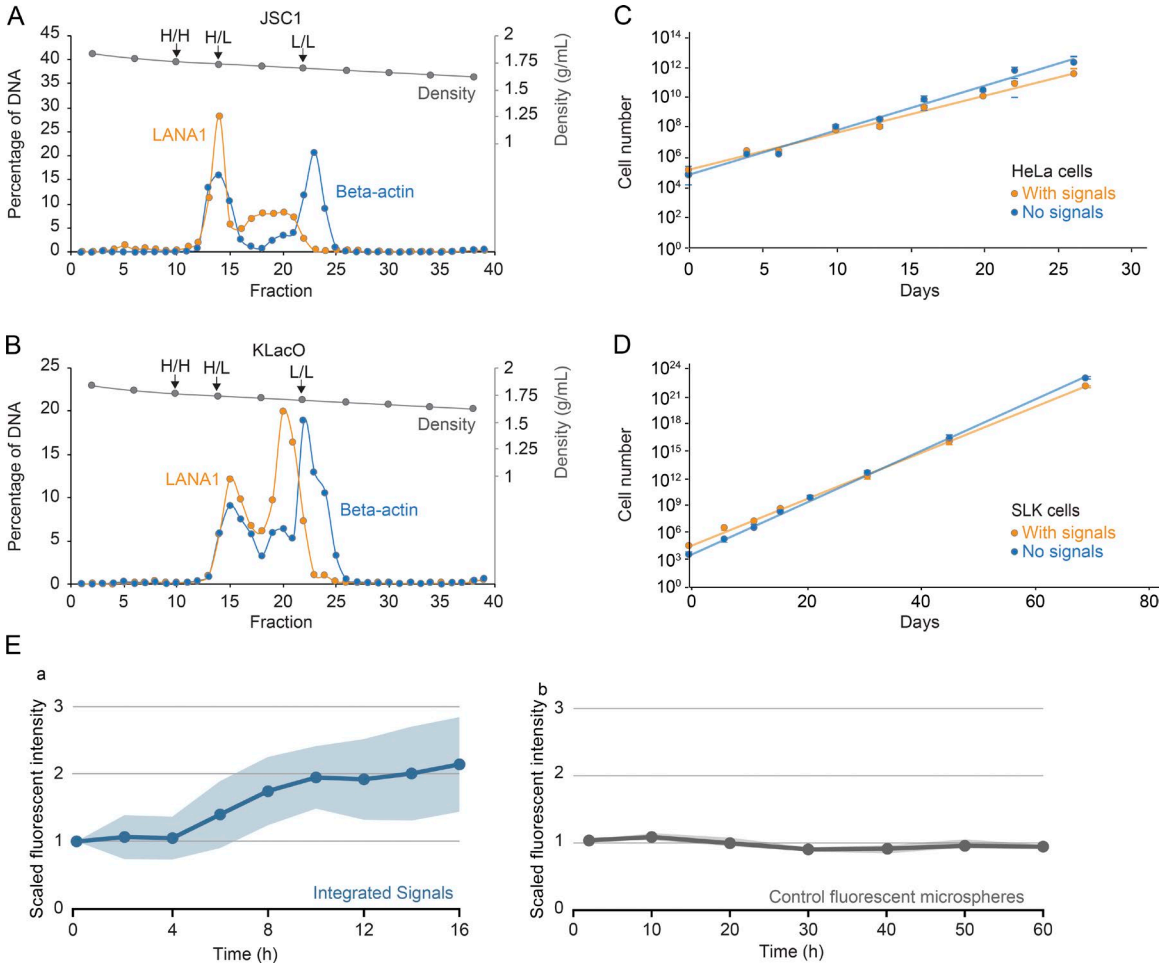
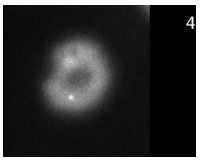
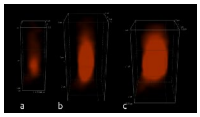


Figure S1. **Distributions of KSHV genomes in PEL cells; synthesis of KSHV and KLacO plasmids is regulated in a licensed manner.** JSC1 (A) and HeLa/KLacO (B) cells were cultured in BrdU and deoxycytidine for 11 h, and total DNA was fractionated in a CsCl gradient. Samples from each fraction were precipitated and used as templates in qPCR to detect sequences encoding β-actin (blue line) and LANA1 (yellow line) as markers of cellular and KSHV DNA, respectively. Results are presented as the proportion of total template molecules detected for each probe. The density (black line) was determined by measuring the refractive index of every fourth fraction; expected approximate densities for heavy-heavy (HH), heavy-light (HL), and light-light (LL) DNAs are indicated. KSHV DNA has higher G+C content than human DNA (Russo et al., 1996) and therefore a higher buoyant density when unsubstituted (one independent experiment for each JSC1 and HeLa/kLacO). (C and D) The proportion of cells with zero KLacO signals was determined by FISH and used to calculate the total number of cells with and without signals at each point along growth curves for HeLa (C) and SLK (D) clones. Cells retaining signals did not grow faster than cells that had lost signals. Data from each clone were pooled within each cell type (HeLa, three clones; SLK, two clones); error bars on each point indicate SD. (E) Integrated KSHV DNA doubles in intensities during S phase. (a) The sum of intensities of 18 integrated lacO signals followed for 60 h in three independent experiments was determined with CAPS and scaled for progression from G1 through G2, with intensities at the beginning of G1 set to 1, showing that signal intensities are close to twofold increased in S phase and maintained through G2. The temporal scaling necessitated signals being binned; the SE for these bins is shaded in blue. (b) Fluorescent microspheres 0.1 µm in diameter were embedded at the surface of the culture plates with poly-L-lysine, followed, and measured over time along with cells harboring miniKSHV with CAPS. Their intensities changed less than 10% (six beads each measured over seven time points).



Video 1. **The consecutive uncompressed *z*-planes for several of the compressed images shown in Fig. 3 (A) and Fig. 8 (B).**



Video 2. **Rotations about the 3D volumes shown in Fig. 4.**

Provided online are three Excel tables and a zip file containing seven files relating to the simulations. Table S1 is a summary of the distributions of KLacO signals in G2 through M phase. Table S2 is a summary of the distributions of miniKSHV signals in SLK/LANA1 cells in G2 through M phase. Table S3 is a summary of the distributions of miniKSHV signals and the sum of their intensities in G2 in SLK/LANA1/AThook cells through G1 phase in their daughter cells (A) and a summary of the distributions of miniKSHV signals in SLK/LANA1/AThook cells in G2 through M phase (B).

Reference

Russo, J.J., R.A. Bohenzky, M.C. Chien, J. Chen, M. Yan, D. Maddalena, J.P. Parry, D. Peruzzi, I.S. Edelman, Y. Chang, and P.S. Moore. 1996. Nucleotide sequence of the Kaposi sarcoma-associated herpesvirus (HHV8). *Proc. Natl. Acad. Sci. USA*. 93:14862–14867. http://dx.doi.org/10.1073/pnas.93.25.14862