Figure S1. Derivatives #5, #3 and #8 formed cell clumps of KSHV-infected BC2 PEL cells and uninfected Ramos cells. KSHV-infected BC2 and uninfected Ramos cells were cultured with 20 μM of derivative #3, #5 or #8 for 24 h. Phase-contrast images were obtained using an inverted microscope. The images of BCBL1 and BC3 which are same as the images in Fig. 3F in the manuscript are presented for comparison. Kaposi's sarcoma-associated herpesvirus.

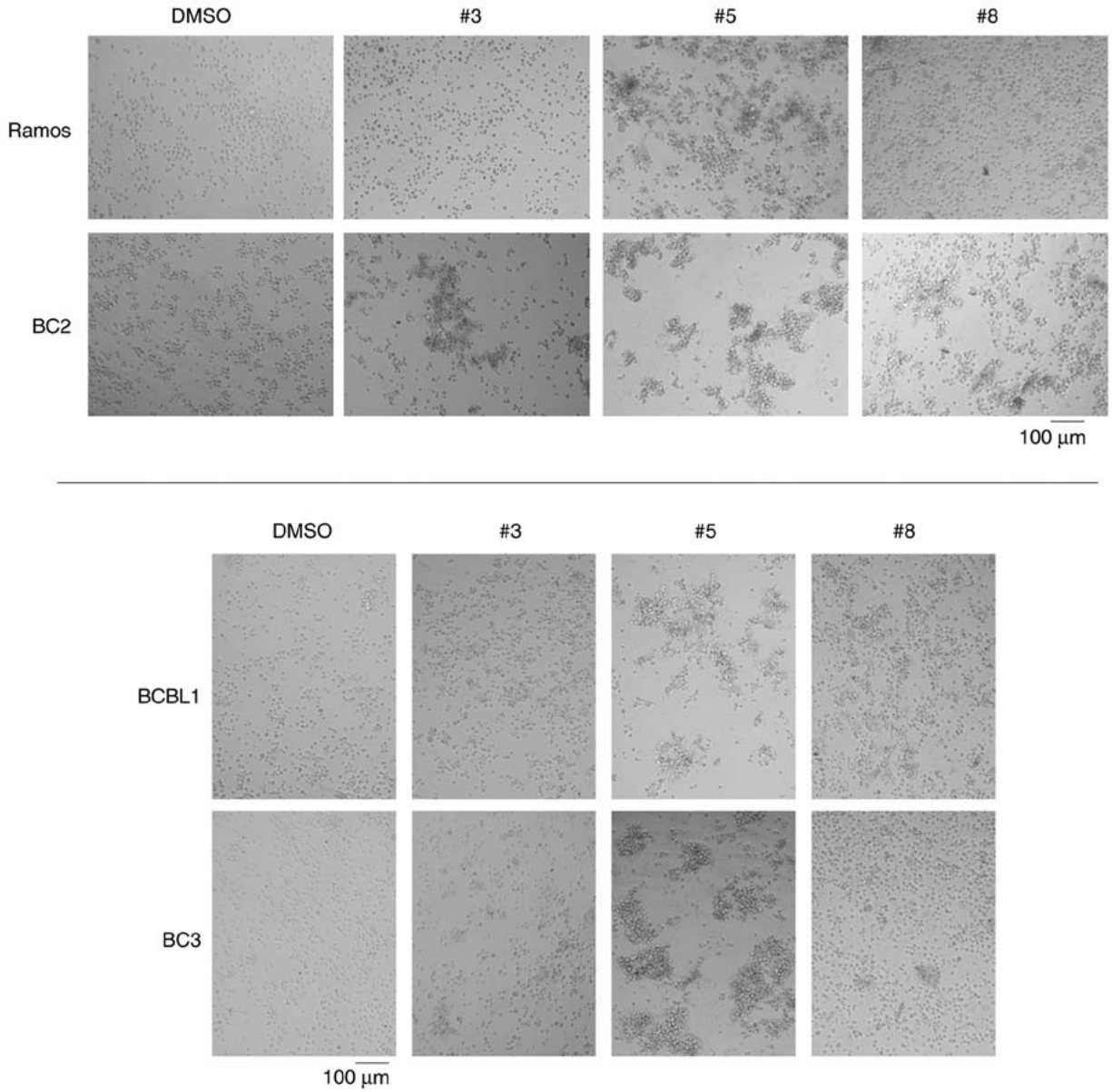

Figure S2. Derivative #5 does not affect STAT3 and NF-κB signaling in PEL cells. (A) KSHV-infected PEL cells (HBL6, BCBL1, BC2, BC3 and JSC1) and KSHV-uninfected B cells (DG75, Ramos and Raji) were incubated with 20 μM of derivative #5 for 9 h, and cell extracts were subjected to western blot analysis using anti-phosphorylated STAT3 [anti-p-STAT3 (T705)], as well as anti-IκBα and anti-NF-κB antibodies. Anti-actin antibodies were also used to assess the actin levels, which served as a loading control. (B) HBL6 PEL cells exhibited a low-expression of β-catenin compared with the other PEL cell lines. Cell extracts of DG75, BC2, BC3 and BC3 cells were subjected to western blot analysis using anti-β-catenin antibody. The western blotting experiments the results of which are depicted were performed twice using independent samples. PEL, primary effusion lymphoma; Kaposi's sarcoma-associated herpesvirus.

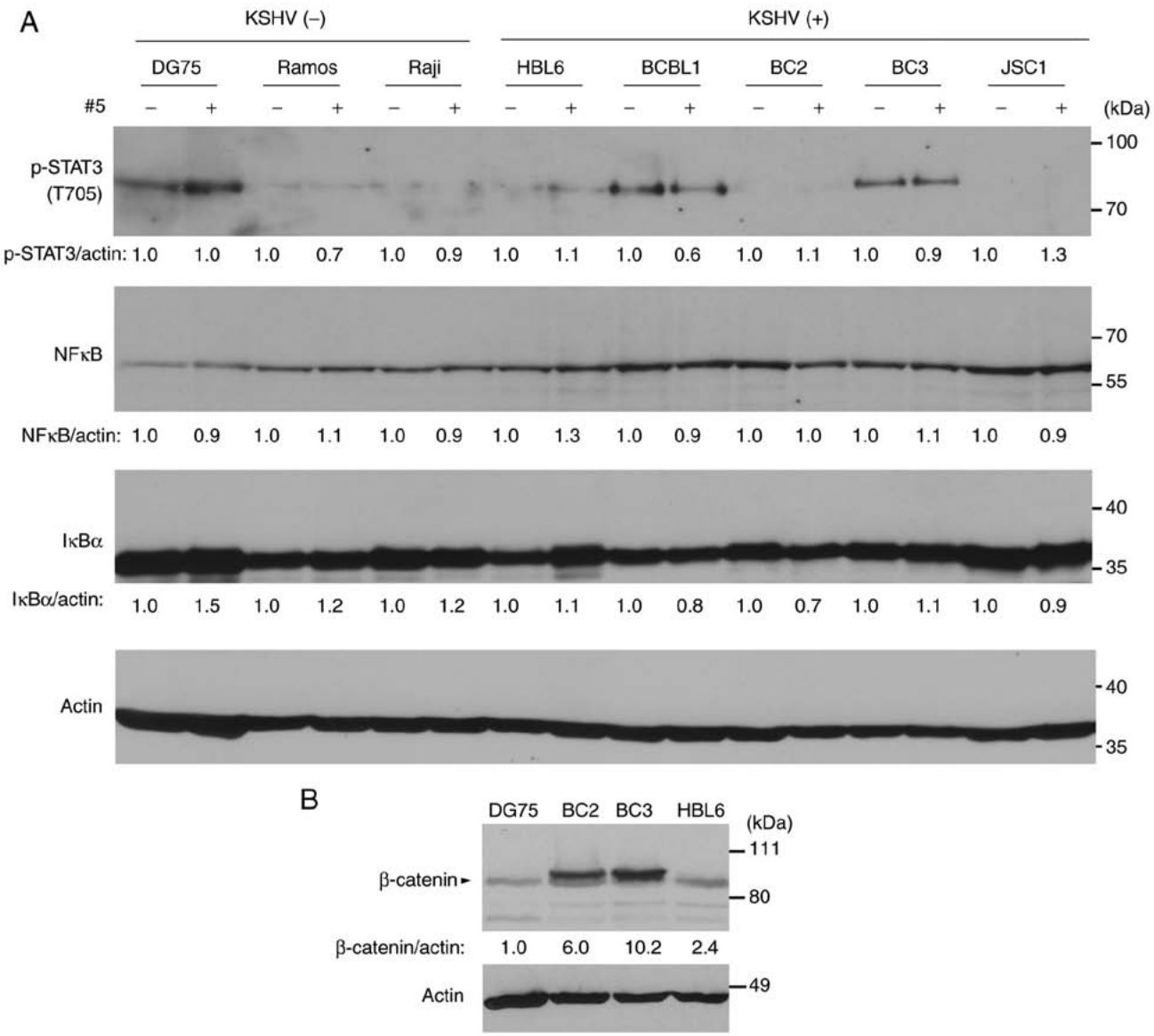

Figure S3. Quantitative analysis of β-catenin expression in derivative #5-treated PEL cells (BCBL1 and BC3). Representative blots (obtained fromwestern blot analysis performed three times) of the quantitative data shown in Fig. 4E are presented. KSHV-infected PEL cells (BCBL1 and BC3) were incubated with 20 μM derivative #5 (or DMSO) in the presence of 50 μg/ml cycloheximide for 0 or 6 h. Cell lysates prepared by three independent experiments were examined using western blot analysis with anti-β-catenin and anti-GAPDH antibodies. PEL, primary effusion lymphoma; Kaposi's sarcoma-associated herpesvirus.