Supplementary Table 1. The Q-PCR primer sequence is summarized in the following table.

Name	Sequence (5'-3')	Application
Flag-u	ggactacaaggacgacgatgac	Shared upstream primer for all the amplifications of Flag-tagged HPV38 E7 mutants, described in Fig. 5I lowest panel
CR1-d	ctcttcaagaactatatcacgaagagtag	Downstream primer for the amplification of the CR1 mutant
CR2-d	tgcttcaatatcctctggaagatca	Downstream primer for the amplification of the full-length HPV38 E7, the CR2, CR1+2 and CR2+3 mutants and the C-terminal truncated mutants (aa 1-50, aa 1-70, aa 1-84)
CR3-d	tcttctcgacaggtgggacac	Downstream primer for the amplification of the CR3 mutant

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Supplementary Figure legends

Supplementary FIG. 1. Dynamics of disruption of actin stress fibers by HPV38 E7.
U2OS cells were sequentially transfected with LifeAct-RFP and GFP or GFP-HPV38 E7 plasmids. At 12h after the second transfection, live imaging of the cells ectopically expressing LifeAct-RFP and GFP or GFP-HPV38E7 was performed under the spinning disk confocal microscope at different time points. The arrows indicate the cells co-expressing LifeAct-RFP and GFP-HPV38E7.

Supplementary FIG. 2. Over-expression of CK2α subunit promotes the dissociation of actin stress fibers
U2OS cells were transfected with the expression plasmids for HA-CK2 α wild-type and HA-CK2 α-4A kinase dead-mutant and 24 h after transfection, cells were fixed and stained with phalloidin-Alexa 568 and anti-HA antibody to visualize actin fibers and the expression of CK2 alpha subunits respectively. Bar is 25 μm.

Supplementary FIG. 3. Association of cutaneous HPV E7s with endogenous eEF1A and their effects on Rho activity and F-actin content.
(A) The binding efficiency of various cutaneous HPV E7s to eEF1A was evaluated by GST pull-down and immunoblotting. (B) U2OS cells were transfected as indicated. 48h after transfection, the Rho activity assay was performed as described in Fig. 2A. (C)

U2OS cells were transfected as indicated. 48h after transfection, the quantification of F-actin/total actin was performed as described in Fig. 1B.

Supplementary FIG. 4. Interaction of HPV38E7 mutants with F-actin.

(A) The lysates of U2OS cells expressing different C-terminal deletion mutants (aa 1-55, aa 1-70, aa 1-84) and the full-length of HPV38 E7 were immunoprecipitated with anti-Flag M2 beads to compare their affinities to F-actin. Quantitative RT-PCR was performed to check the RNA expression level of all the mutants (the lowest panel). (B) Cells were transfected with indicated plasmids and immunoprecipitation and immunoblotting were performed on cell lysates. (C) HPV38 E7 and its mutants were transfected into U2OS cells for immunoprecipitation to examine its binding to F-actin.

Supplementary FIG. 5. eEF1A1 mutant (T430A+V431A) is defective in interacting with HPV38 E7 and F-actin and to promote Rho activation

(A) GST pull-down and immunoblotting were performed to evaluate the interaction of eEF1A1 and its mutant with HPV38 E7. T430 and V431A were targeted for mutagenesis as they are important for the completeness of beta sheet in the C-domain of eEF1A (59). (B) F-actin was incubated with immunoprecipitated F-eEF1A or its mutant (T430A+V431A). Supernatant and pellets were separated by centrifugation and analyzed by immunoblotting. F-actin and its binding proteins appear in the pellets, and G-actin and unbound proteins appear in the supernatant. 2% SDS was included in the reaction mix as the control. (C) U2OS cells were transfected as indicated, and Rho activity assay was performed as previously described in Fig. 2A.

Supplementary FIG. 6. HPV38 E7 induces actin cytoskeleton disruption and enhances cell proliferation in HEK 293

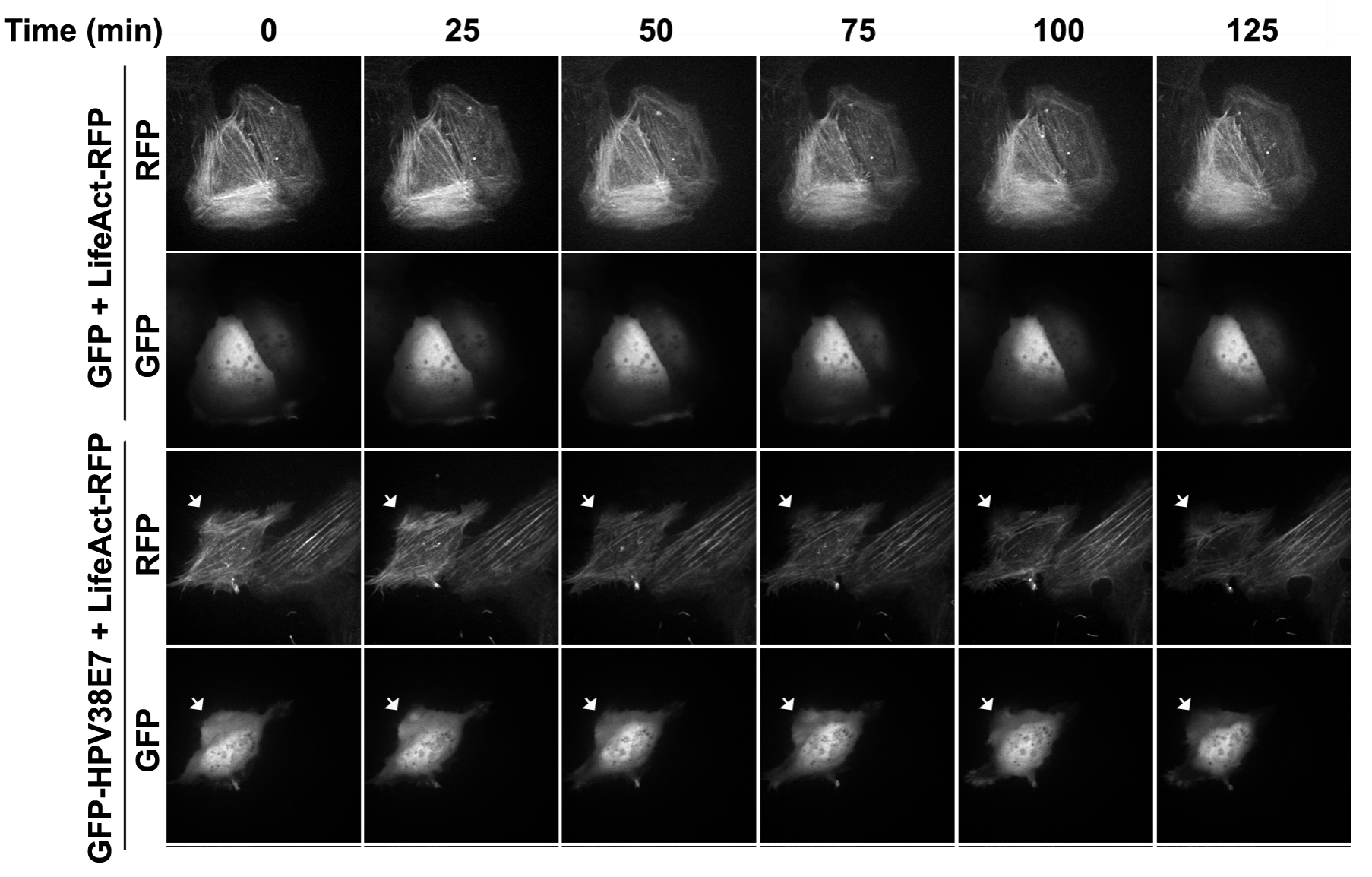
HEK 293 cells stably transfected with vector (293-vector) (A) or stably expressing F-HPV38E7 (293-F-HPV38E7) (B) were treated with indicated kinase inhibitors, and actin fiber staining and cell proliferation were performed. Bar is 25 μm.

Supplementary FIG. 7. HPV38 E7 mutant defective for pRb binding has a weaker ability to enhance keratinocyte proliferation.

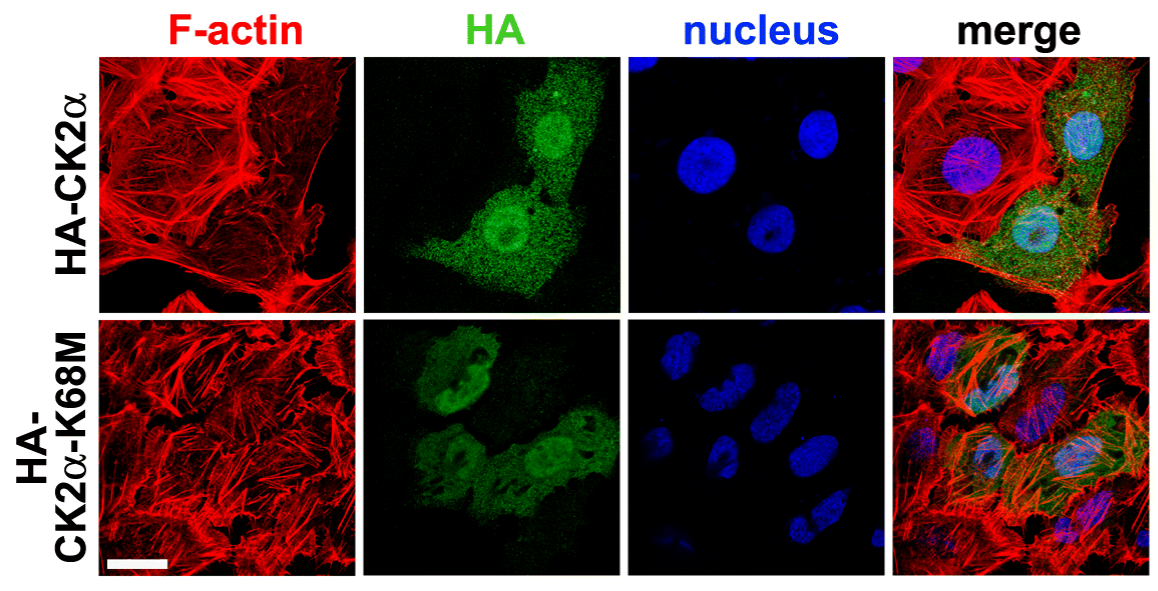
Human primary keratinocytes were transduced by vector pLXSN, HPV38 E7 or its pRb binding defective mutant (C24G+E26G) were monitored for proliferation as described in FIG. 9.

Supplementary FIG. 8. eEF1A has no effect on ERK phosphorylation.

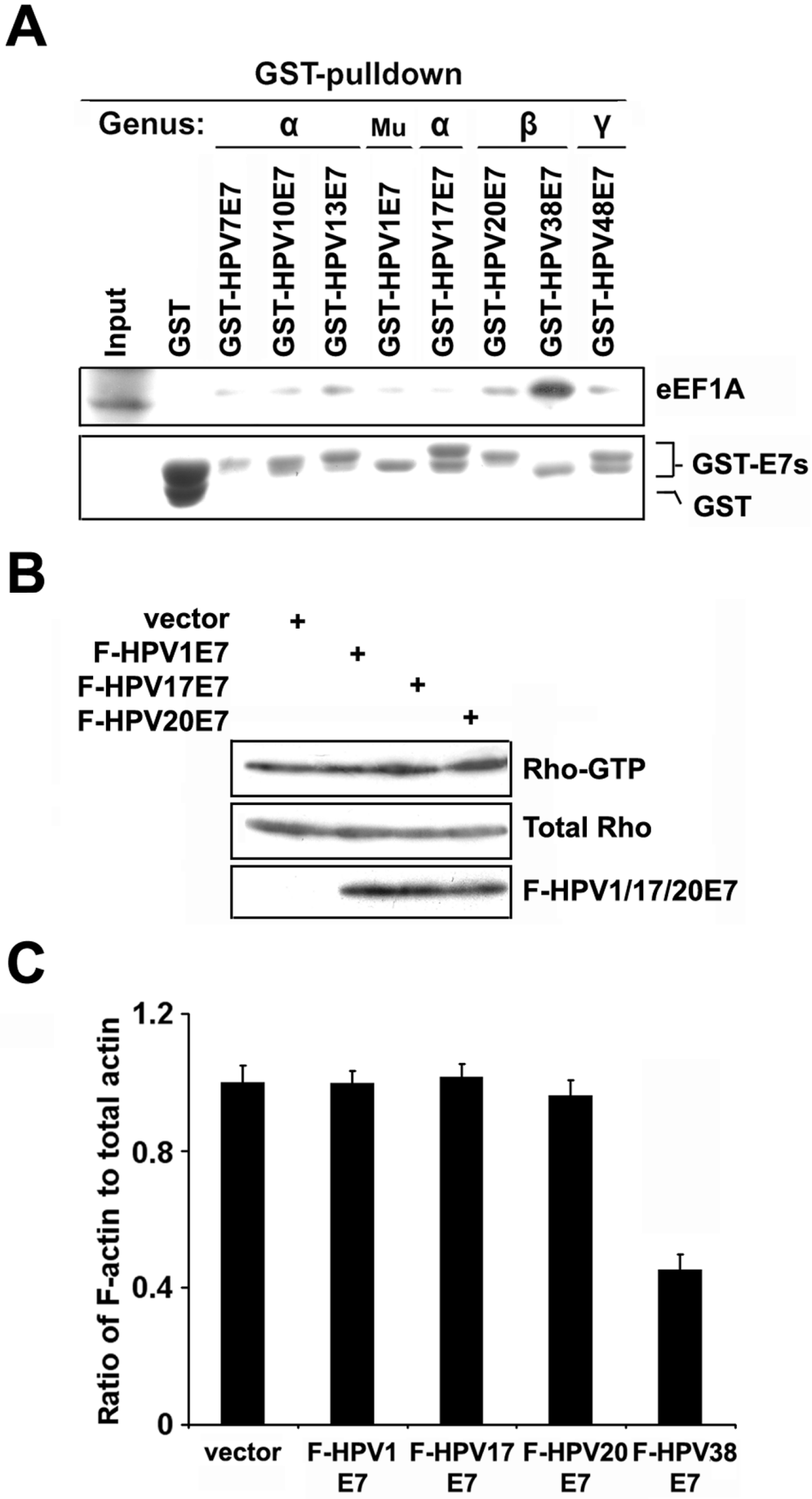
U2OS cells were transfected with empty vector or F-eEF1A1 expression plasmid, and 24 h later, cells were serum starved for treated for 18 hrs. Cells were then put back to a 10% serum-containing medium and lysed at indicated time points for immunoblotting.



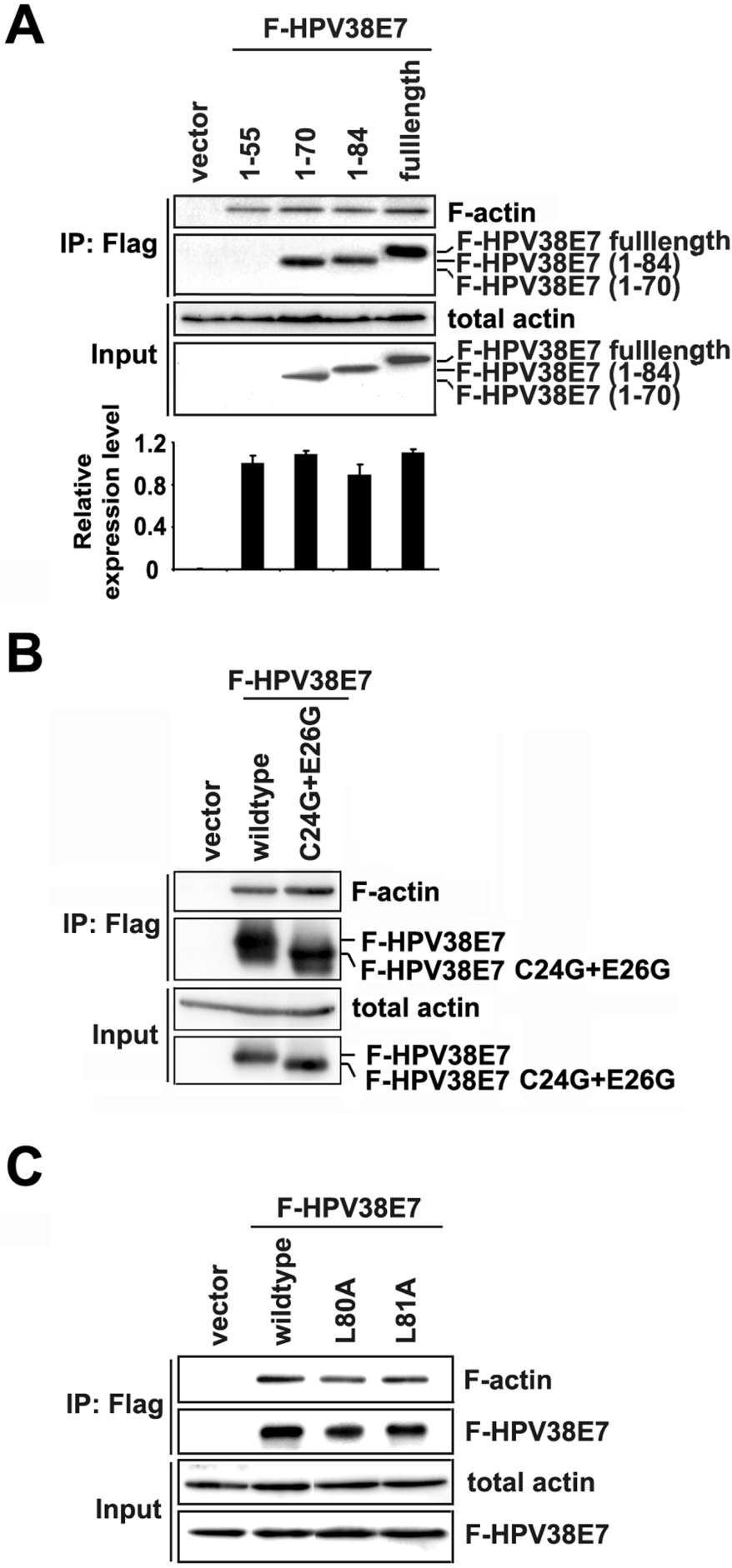
Supplementary Figure 1. Yue et al.



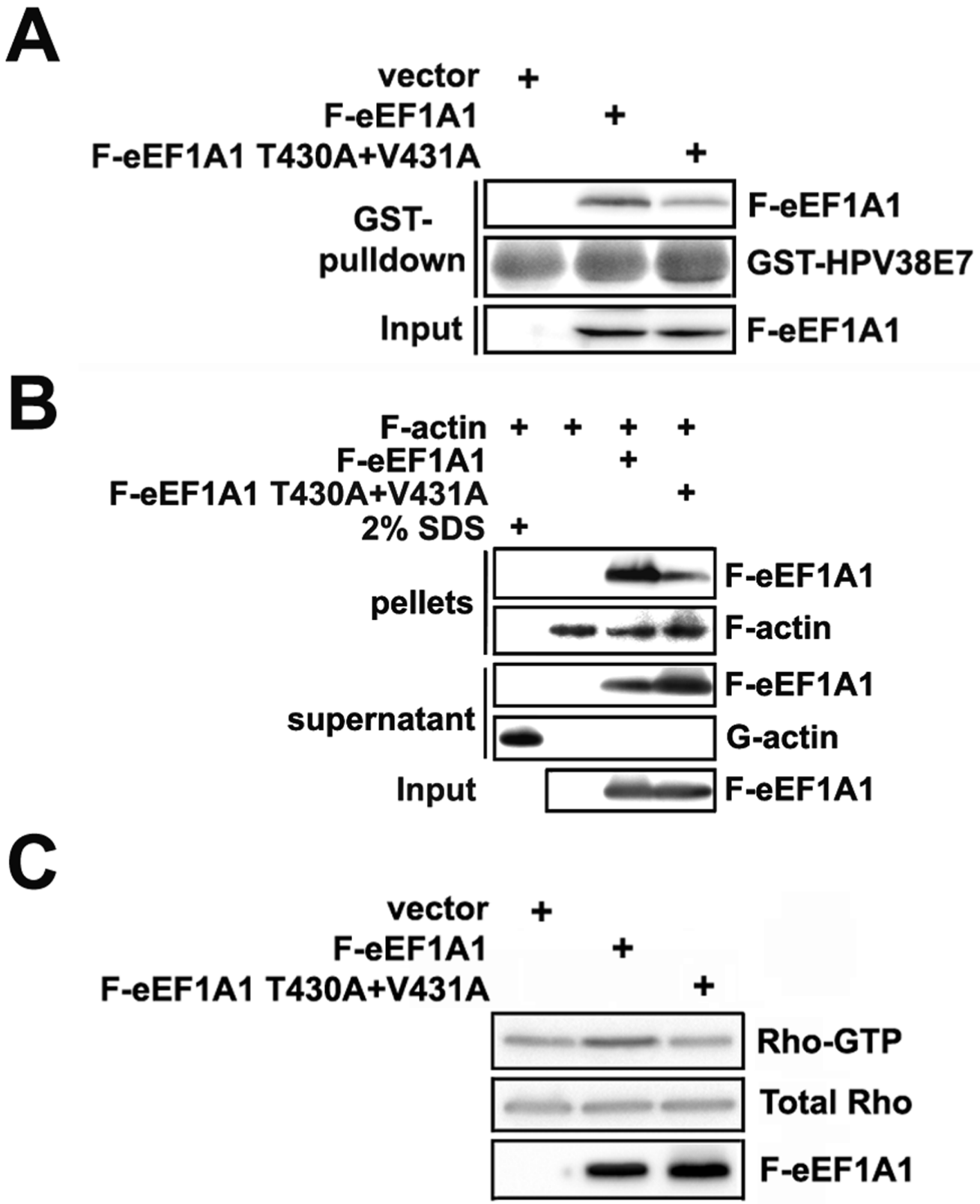
Supplementary Figure 2. Yue et al.



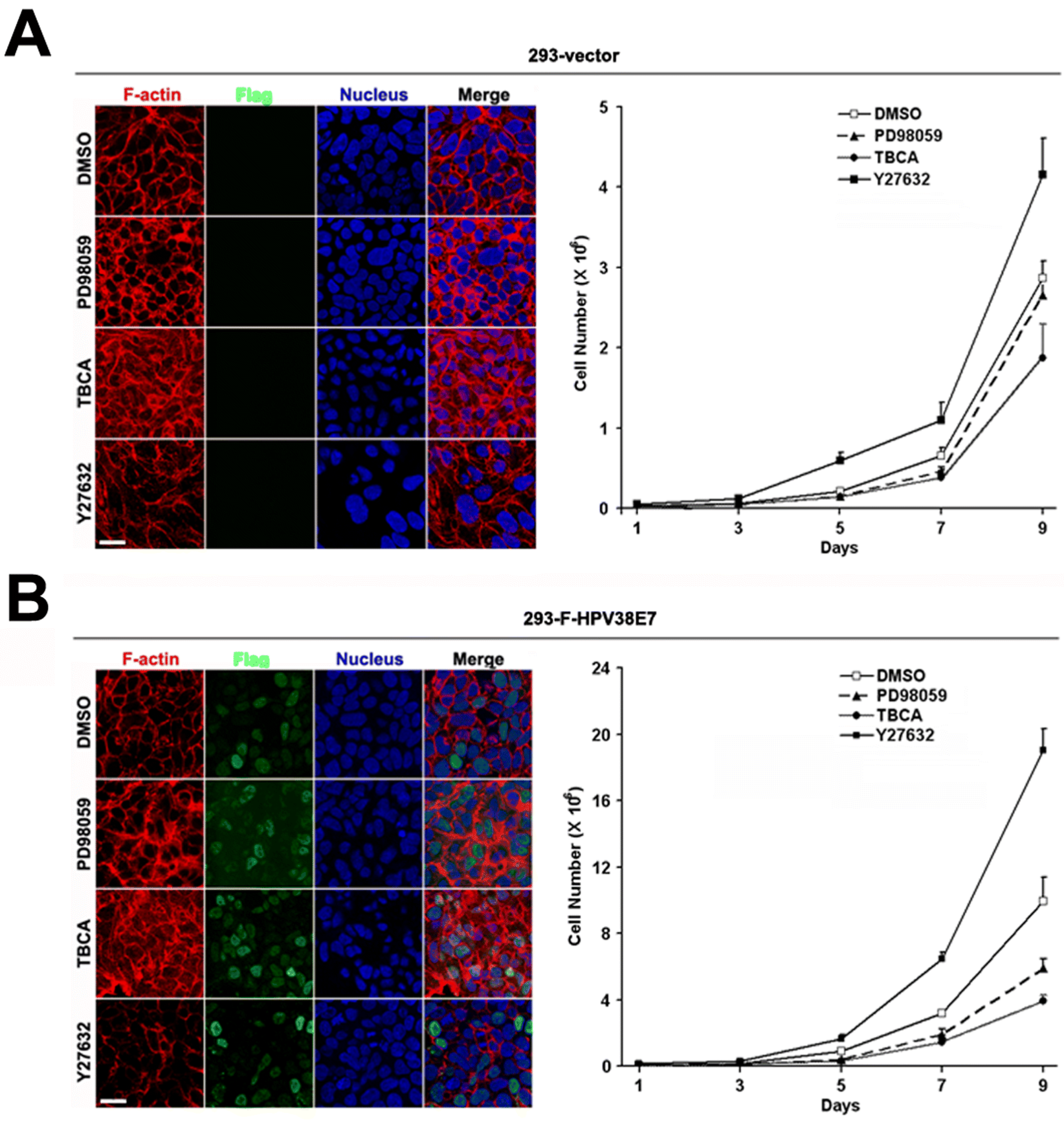
Supplementary Figure 3. Yue et al.



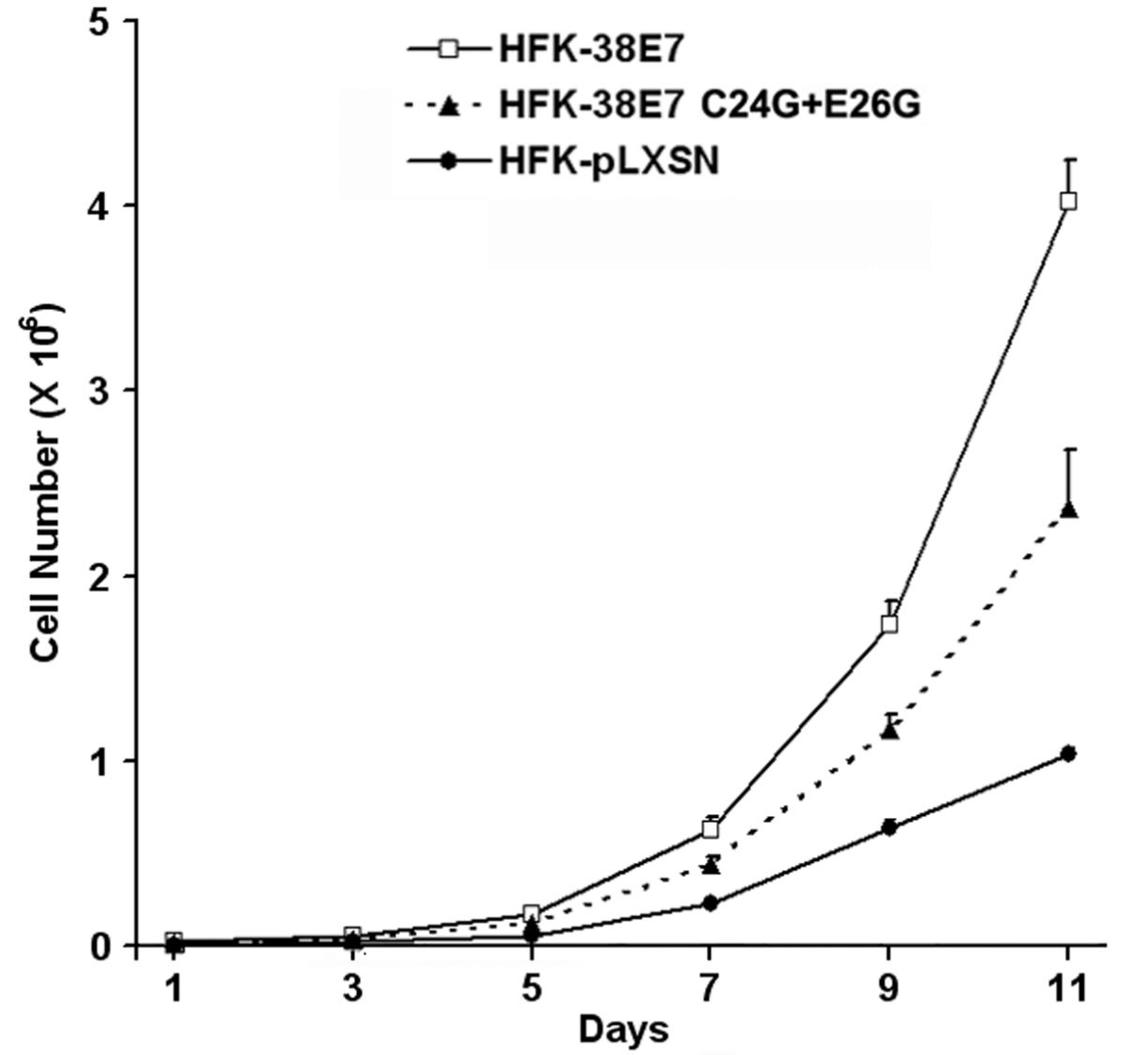
Supplementary Figure 4. Yue et al.



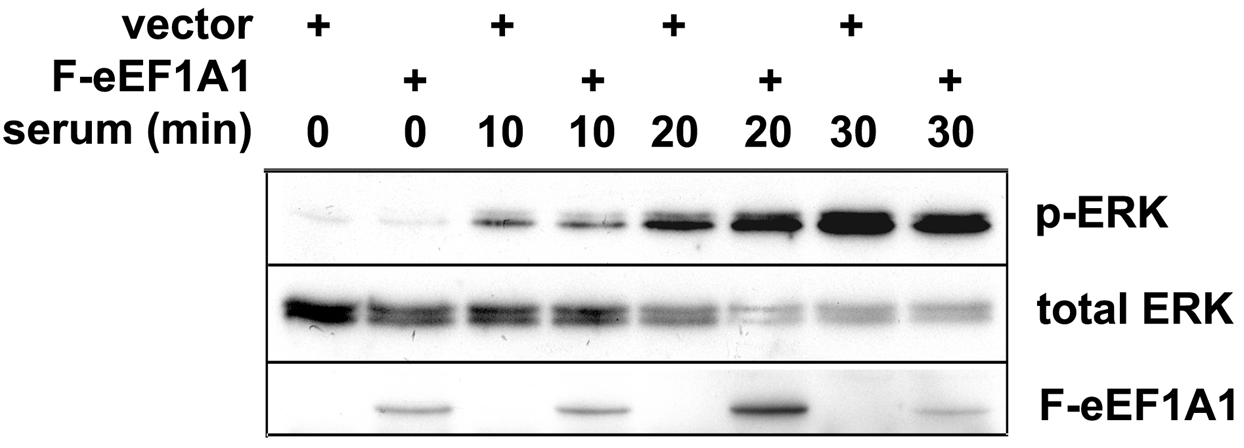
Supplementary Figure 5. Yue et al.



Supplementary Figure 6. Yue et al.



Supplementary Figure 7. Yue et al.



Supplementary Figure 8. Yue et al.