KLF5 promotes cervical cancer proliferation, migration and invasion in a manner partly dependent on TNFRSF11a expression

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Supplemental Figures

Figure S1

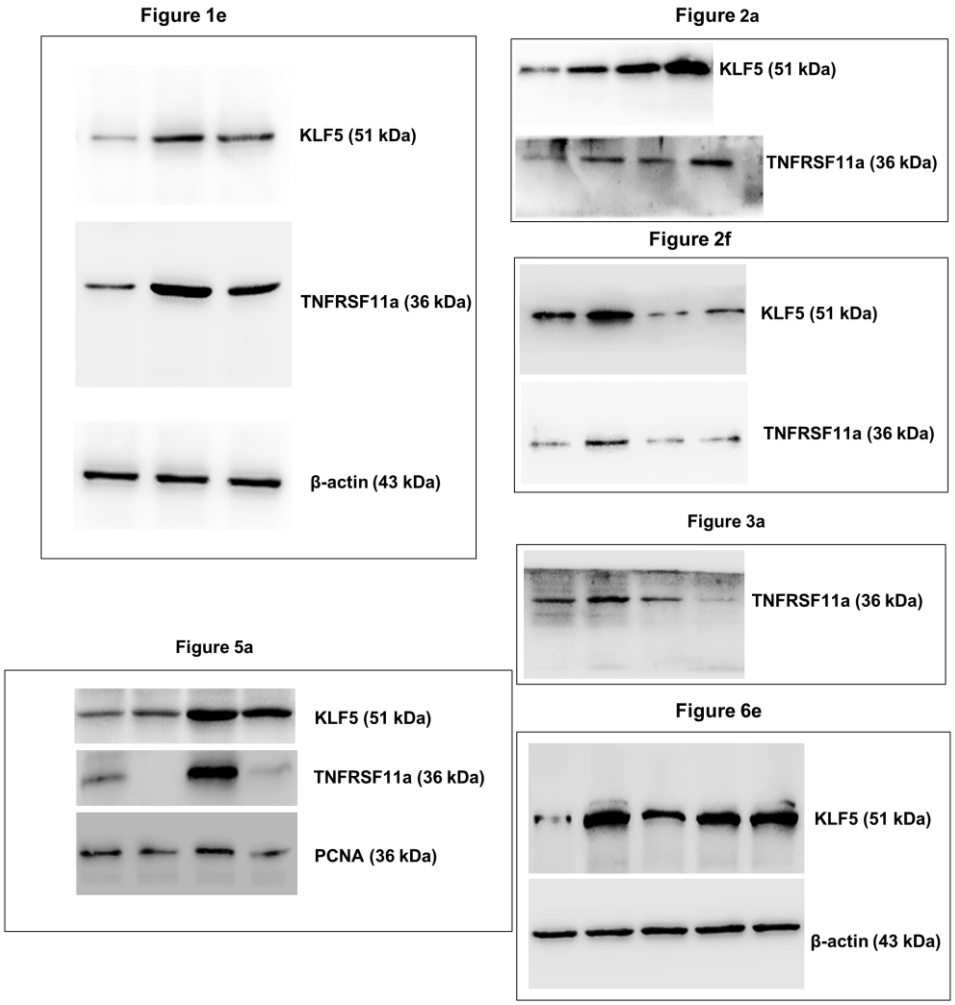

Fig. S1. Full-width of membrane with a protein marker besides the each box of the original blots used in Figures 1–6. The molecular size is shown as indicated. Panels shown in this supplemental figure correspond to those in the main article.

Figure S2

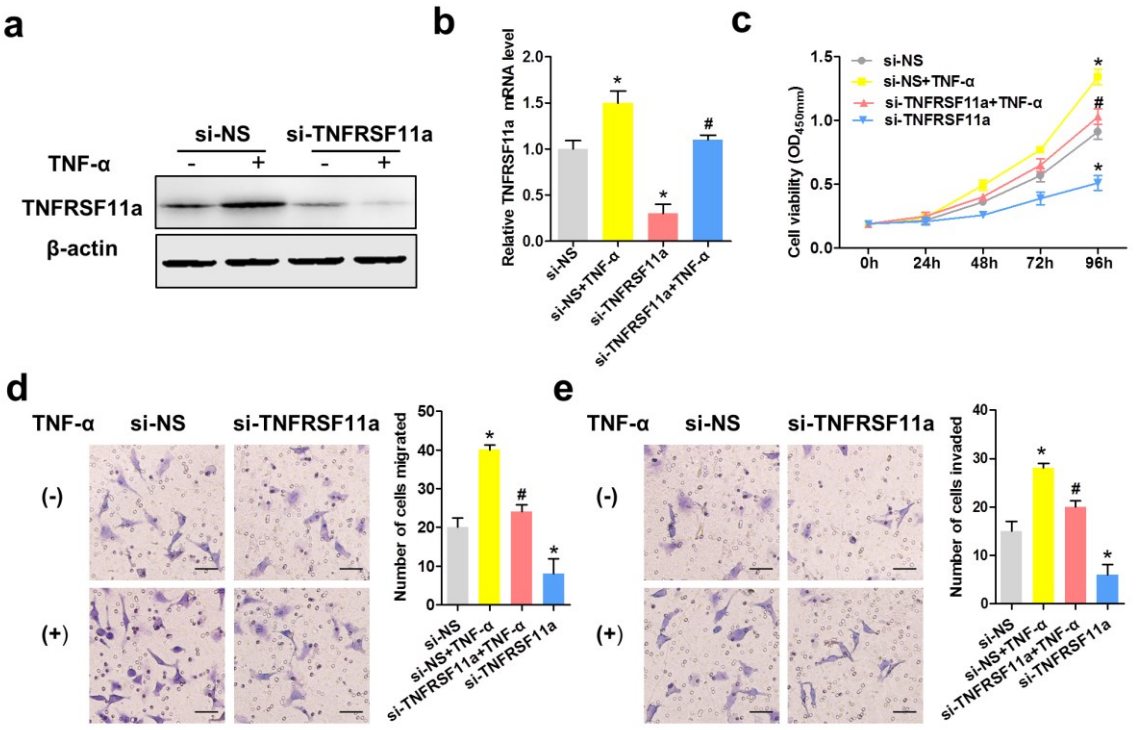

Fig. S2. SiHa cells were transfected with non-specific short interfering RNA (siRNA; si-NS) or tumour necrosis factor receptor superfamily member 11a (TNFRSF11a)-specific siRNA (si-TNFRSF11a) for 24 h and subsequently treated or not with tumour necrosis factor (TNF)-α. TNFRSF11a protein and mRNA expression were analysed by western blotting (A) and quantitative real-time polymerase chain reaction (B). *P<0.05, **P<0.01 vs. the green fluorescent protein adenovirus (Ad-GFP) group. (C) In SiHa cells, stable TNFRSF11a knockdown significantly inhibited cell growth relative to the si-NS treatment during a 96-h period, as measured by the CCK-8 assay. Data represent the means ± standard errors of the means (SEM; n=3). *P<0.05 vs. the si-NS group, $^{\#}$P<0.05 vs. the TNF-α group. TNFRSF11a knockdown altered SiHa cell migration (E) and invasion (F), as shown in Transwell assays. Data represent the means ± SEM (n=3). *P<0.05 vs. the si-NS group, $^{\#}$P<0.05 vs. the TNF-α group.