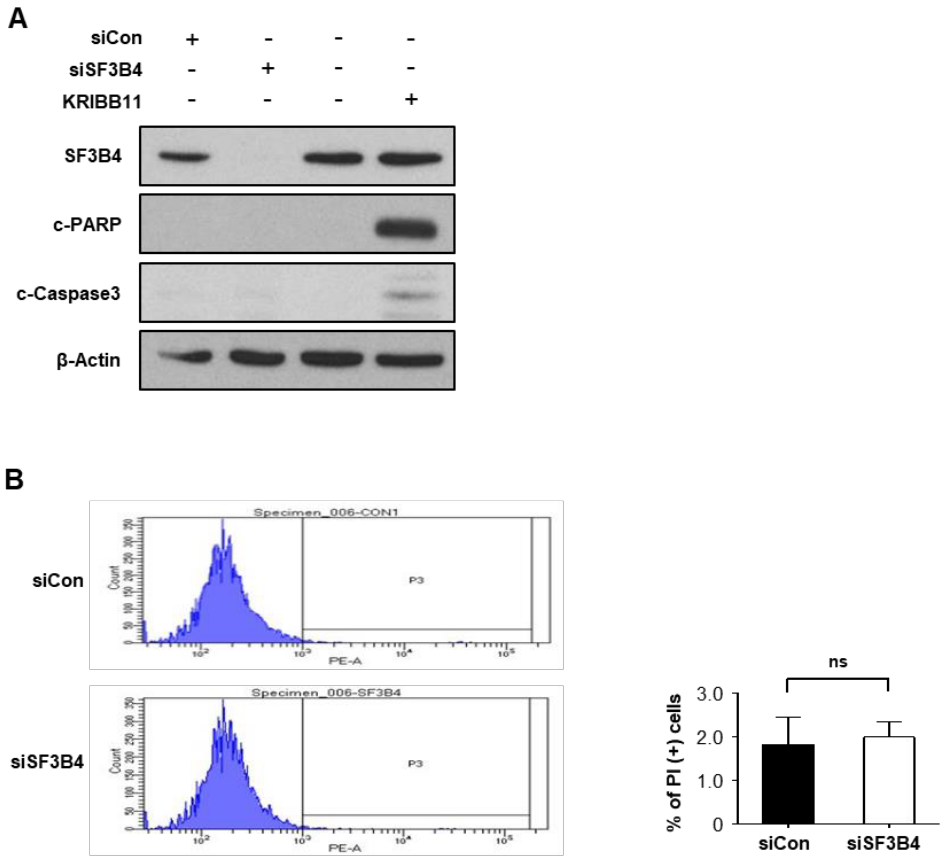
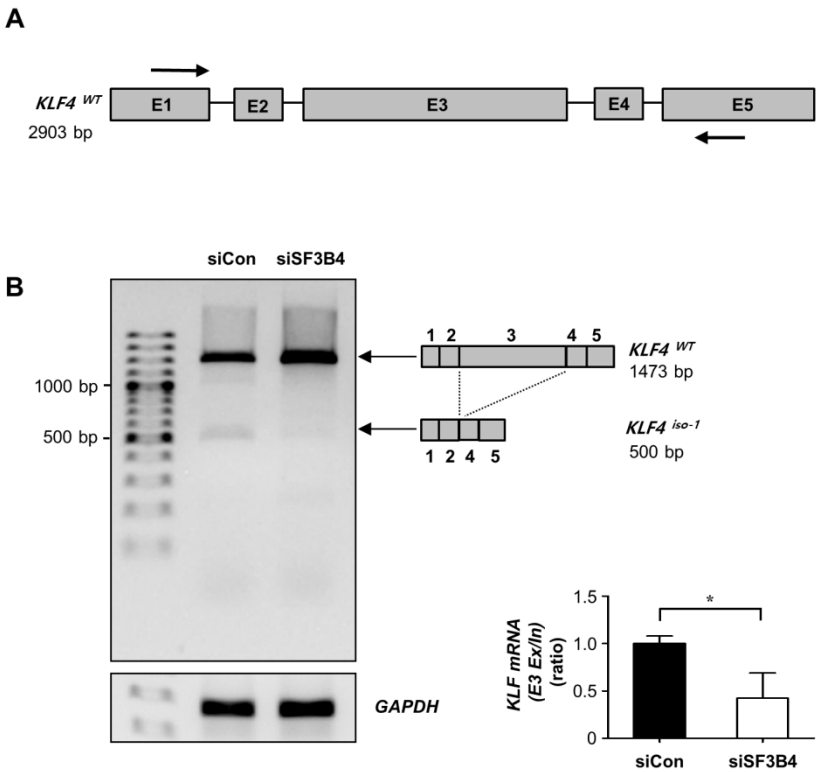


Supplementary Fig. S1. The effect of SF3B4 depletion on the induction of apoptosis and necrosis in A549 cells. (A) After knockdown of SF3B4 using SF3B4 siRNA (siSF3B4, 100 nM), the apoptosis was determined by western blotting for cleaved PARP (c-PARP) and cleaved caspase 3 (c-caspase 3). As a positive control, the lysates from A549 cells treated with KRIBB11 (5 μM), which was shown to induce apoptosis in our previous study (Yoo et al., 2021), were loaded together. siCon, control siRNA. (B) FACS analysis following propidium iodide (PI) staining of control and SF3B4-depleted A549 cells (left). The population of PI positive cells was determined from three independent experiments (right). Data are presented as the mean ± SD. ns, non-significant.

REFERENCE

Yoo, K., Yun, H.H., Jung, S.Y., and Lee, J.H. (2021). KRIBB11 induces apoptosis in A172 glioblastoma cells via MULE-dependent degradation of MCL-1. Molecules *26*, 4165.



Supplementary Fig. S2. The effect of SF3B4 depletion on the splicing of *KLF4* mRNA in A549 cells. (A) Schematic diagram of five exons (E1-E5) of *KL4F* mRNA. The positions of the primer sets for PCR are marked with arrows. (B) After knockdown of SF3B4, RT-PCR was performed using the primer sets covering exon 1 and exon 5 of *KLF4* mRNA as previously described (Shen et al., 2018). The PCR products were analyzed by 1% agarose gel electrophoresis (left). The assumed bands for wild type (wt) and isoform 1 (iso 1) of *KLF* mRNA were indicated on the right. GADPH was provided as a loading control. The intensities of the lower and upper bands were calculated and presented as a ratio of exon 3 (E3) exclusion/inclusion (Ex/In) as a relative value in which the value from the control cells is designated as 1.0 (right). Values are presented as the mean ± SD of three independent experiments. $*P < 0.05$.

Supplementary Table S1. The primer sequences used for qRT-PCR in this study

Gene	Forward (5'→ 3')	Reverse (5'→ 3')
SF3B4	CTATTCCTTGGACCAATCAG	GGACCAACATAAAAAAGAAA
UBE4B	CCAAAGAAGCTGTTGGACCAACTG	GGGTGTCCATCAGAGGGTCTC TG
p53	GCCATCTACAAGCAGTCACAG	TCATCCAAATACTCCACACGC
MDM2	AGCTTCTCTGTGAAAGAGCACAG	ATGGCGTCCCTGTAGATTCA
p27	GAGAAGCACTGCAGAGACAT	ATGCGTGTCCTCAGAGTTAG
p21	GGAAGGGACACACAAGAAGAA	TCCTTGTTCCGCTGCTAATC
β-Actin	AGTACTCCGTGTGGATCGGC	GCTGATCCACATCTGCTGGA