Online Data Supplement

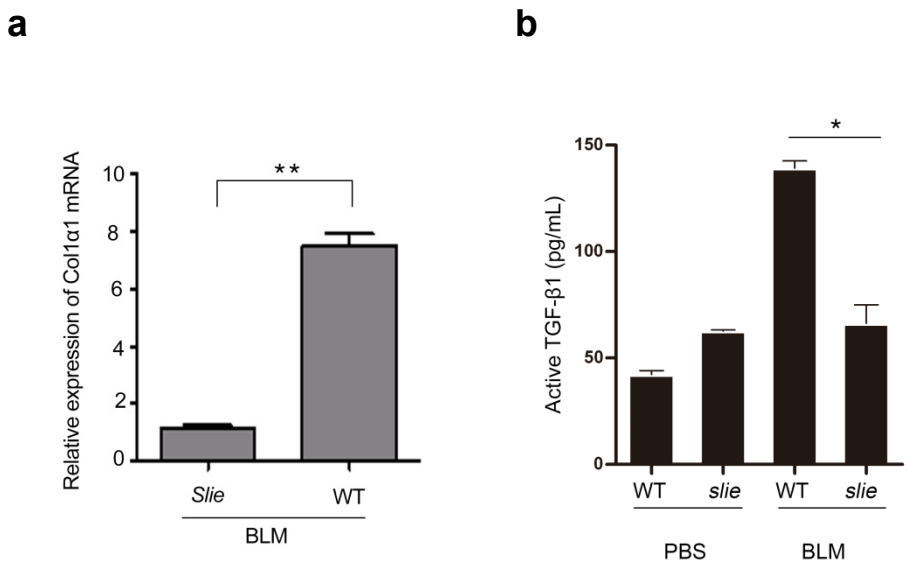

Figure S1 The expression levels of fibrotic markers in the lung of DDR2 mutant mice. Wild type or *Ddr2* deficient (*slie*) mice were intranasally administered bleomycin solution. 14 days post injury challenge, the lung tissues and broncho-alveolar lavage fluid (BALF) were harvested separately. (**a**) qPCR analysis of collagen I (*col1α1*) mRNA expression in lung tissues. The experiment was performed independently for at least three times. *Gapdh* was used as reference genes. The relative *col1α1* expression level in *slie* mice was set at 1. **, $p<0.001$. (**b**) ELISA assay of the concentration of active form of TGF-β1 in BALF.

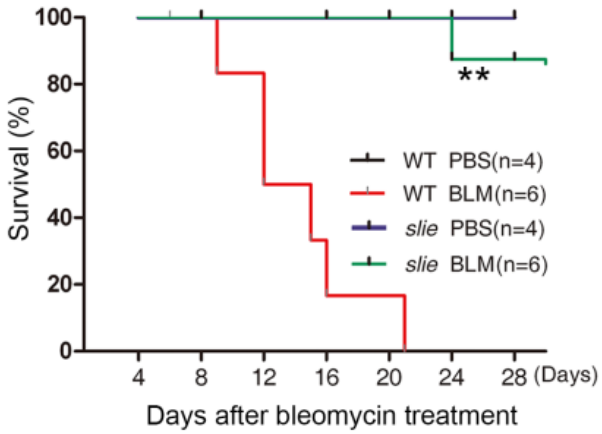

Figure S2 DDR2 deficiency confers the mice survival advantage in response to high dose of bleomycin. Wild type or *slie* mutant mice received high dose of bleomycin (25 mg/kg) treatment and the life span of each mouse was documented. Kaplan-Meier survival curves were plotted. **, $p<0.01$.

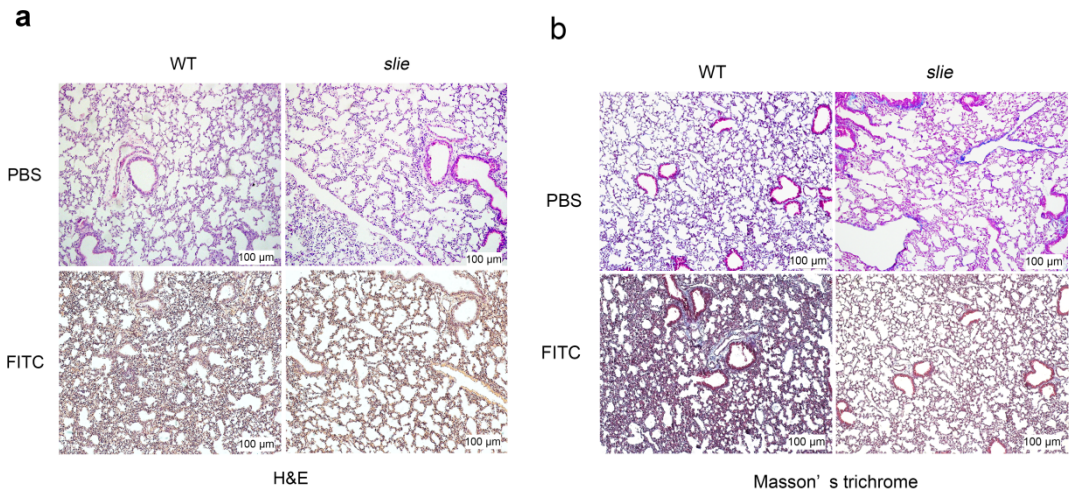

Figure S3 DDR2 mutant mice are protected from FITC-driven lung fibrosis. Wild type or *slie* mice were intranasally administered FITC solution (6 mg/kg BW) or an equal volume of PBS (five mice for each group). Lung tissues were harvested at day 28 and then subjected to H&E staining (**a**) and Masson's trichrome staining (**b**), respectively. Representative images of the staining are shown. Scale bars, 100 μm.

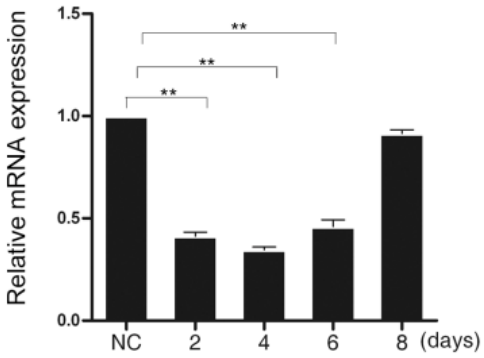

Figure S4 The duration of *in vivo* knockdown of DDR2. C57BL/6 mice were intranasally administered 2'-OMe-modified siRNA and at various time points after siRNA treatment, the mouse lungs were obtained for qPCR analysis of DDR2 mRNA expression. The experiment was performed independently for at least three times. *Gapdh* was used as reference genes. The relative DDR2 expression level in control siRNA-treated mice was set at 1. **, $p<0.001$.

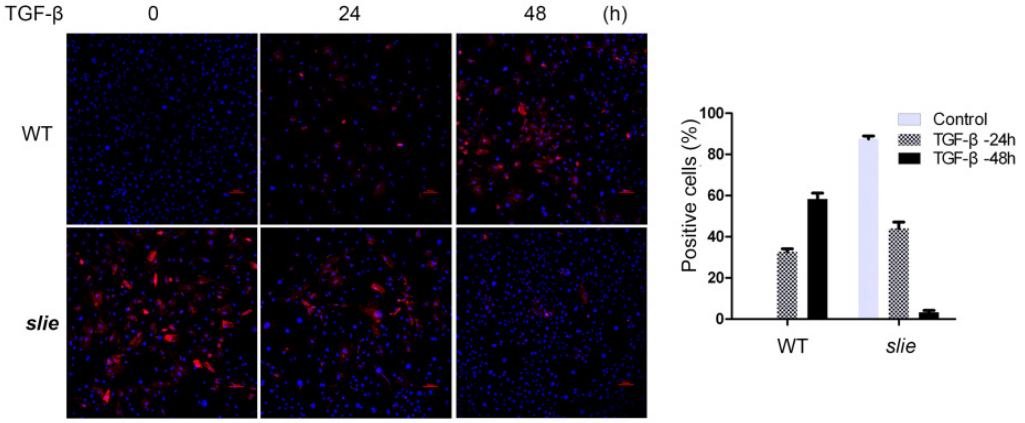

Figure S5 DDR2-deficient lung fibroblasts are refractory to TGF-β1-induced expression of α-SMA. Primary lung fibroblasts from control or *slie* mice were stimulated with TGF-β1 for 24 or 48 h and the treated cells were then subjected to immunofluorescence staining of α-SMA. The right represents the percentage of α-SMA positive cells (10 fields per group).

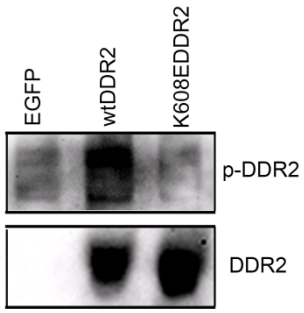

Figure S6 DDR2 kinase-dead mutant is resistant to collagen-induced activation. The lentiviral constructs expressing either wild-type DDR2 or the kinase-dead mutant form of DDR2 (K608E) were transiently transfected into HEK293T cells. 24 h after transfection, the cells were stimulated with 10 μg/mL colagen I for 2 h. For detection of phosphorylated DDR2, the cell lysates were immunoprecipitated with anti-DDR2 and then subjected to immunoblot with anti-phosphotyrosine.