

Supplementary Materials

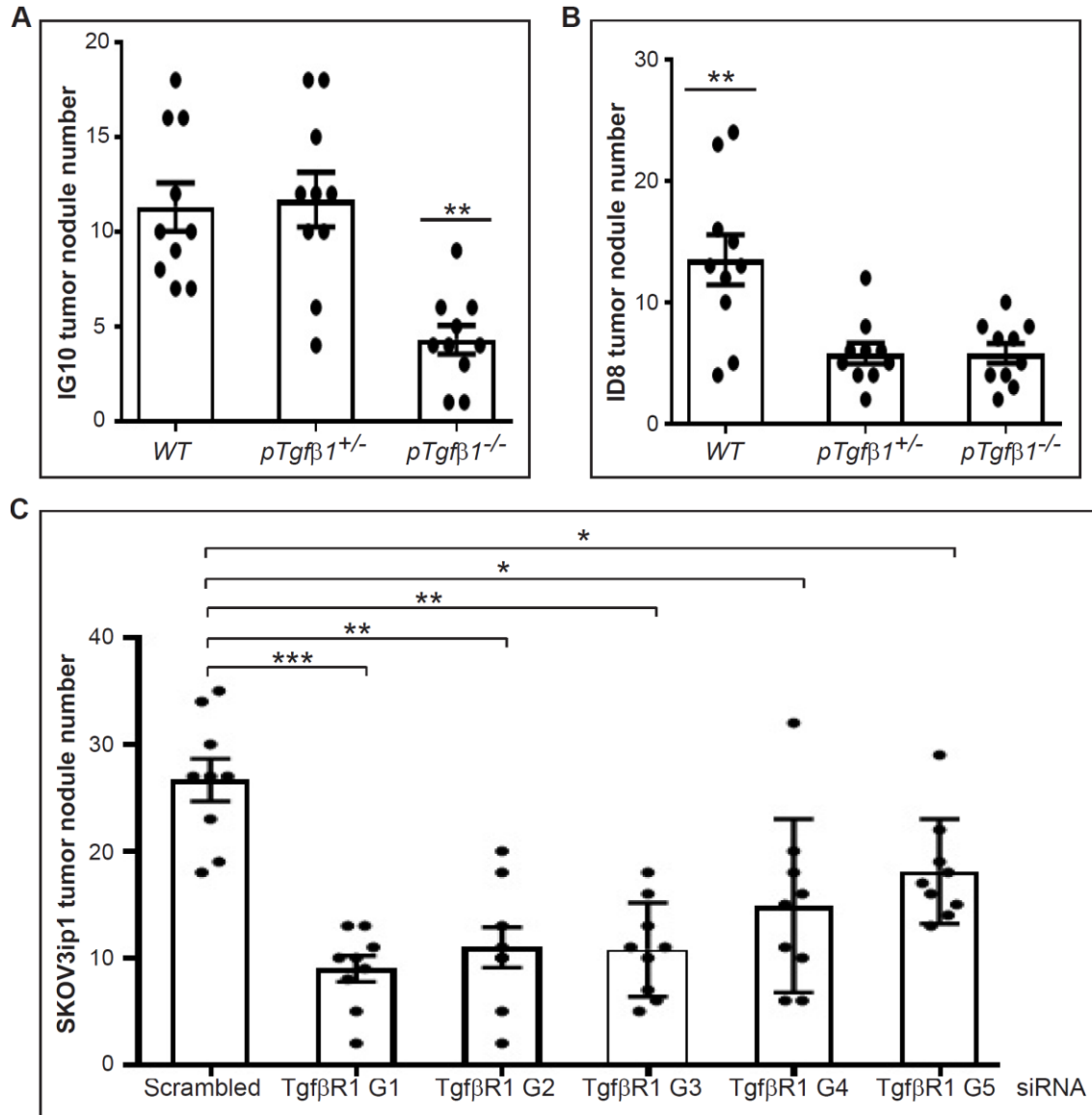


Figure S1: Effect of platelet Tgfβ1 and ovarian cancer cell TgfβR1 on the number of tumor nodules in orthotopic murine models of ovarian cancer. The number of tumor nodules induced by (A) IG10 and (B) ID8 murine ovarian cancer cells in WT, *pTgfβ1^{+/-}*, or *pTgfβ1^{-/-}* mice. (C) The number of tumor nodules induced by SKOV3ip human ovarian cancer cells in nude mice treated with hTgfβR1 siRNA at different time points. Mice treated with scrambled siRNA served as controls. The ANOVA analysis was performed and corresponding *p* values of the F-test were 0.002 (A), 0.004 (B), and <0.00001 (C). Student's t-test is carried out for statistical analysis and significance levels indicated are as follows: *p* < 0.05 for *, *p* < 0.01 for **, *p* < 0.001 for ***. Averaged data are presented as the mean ± SEM.

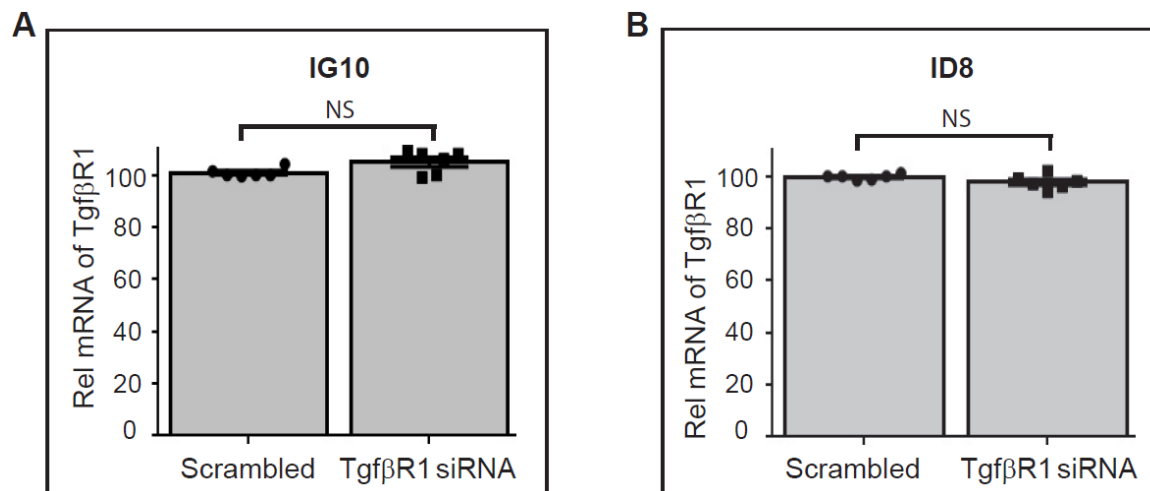


Figure S2: Quantification of TgfβR1 mRNA level by qRT-PCR in IG10 and ID8 murine ovarian cancer cells incubated with scrambled siRNA or human specific hTgfβR1 siRNA *in vitro* for 48 hours (n=6). For IG10, the relative mRNA levels in cells treated with scrambled siRNA or hTgfβR1 siRNA were 100.85 ± 0.72 and 105.05 ± 1.79 , respectively and their difference was statistically insignificant ($p = 0.07$). For ID8, the relative mRNA levels in cells treated with scrambled siRNA or hTgfβR1 siRNA were 99.70 ± 0.43 and 97.67 ± 1.12 , respectively and their difference was also statistically insignificant ($p = 0.14$).

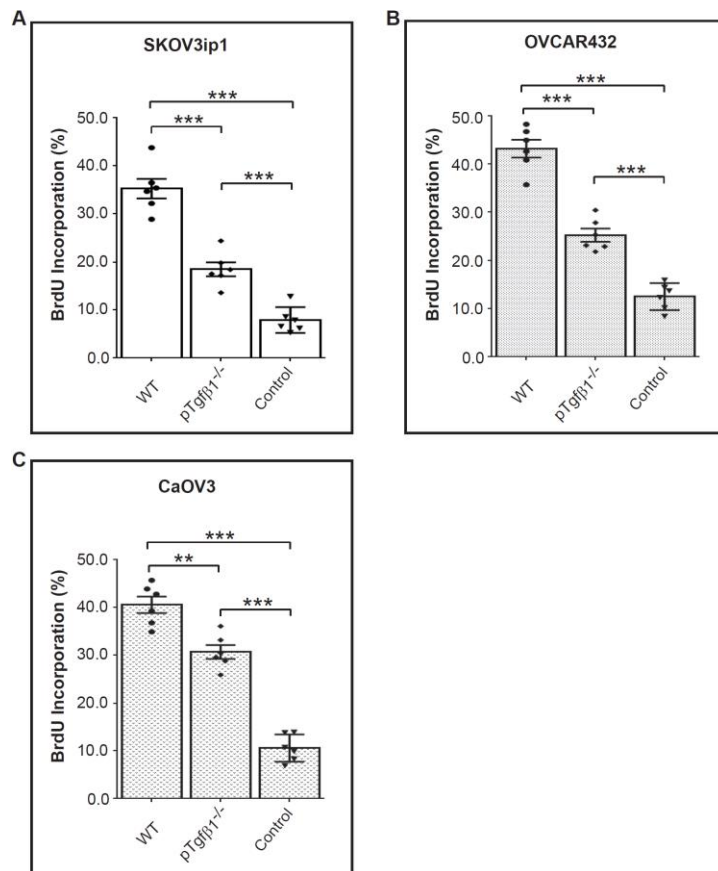


Figure S3: Effect of platelet Tgfβ1 on proliferation of human ovarian cancer cells *in vitro*. Platelets isolated from whole blood of mice were coincubated with human ovarian cancer cells (SKOV3ip1, OVCAR432, and CaOV3) and cell proliferation rate was measured by quantifying BrdU incorporations (A) Proliferation rate of SKOV3ip1 cells coincubated with platelets isolated from WT or *pTgfβ1^{-/-}* mice. SKOV3ip1 cells incubated in buffer were used as controls. (B) Proliferation rate of OVCAR432 cells coincubated with platelets isolated from WT or *pTgfβ1^{-/-}* mice. OVCAR432 cells incubated in buffer were used as controls. (C) Proliferation rate of CaOV3 cells coincubated with platelets isolated from WT or *pTgfβ1^{-/-}* mice. CaOV3 cells incubated in buffer were used as controls. n=3 mice per each genotype and each assay was performed in duplicate. The ANOVA analysis was performed on the results and all of the *p* values of the F-test were < 0.00001. Student's t-test was carried out for statistical analysis and significance levels were as follows: *p* < 0.05 for *, *p* < 0.01 for **, *p* < 0.001 for ***. Averaged data are presented as the mean ± SEM. WT platelets increased BrdU incorporation by 4.5 folds in SKOV3ip1 cells (no platelets: 7.9% ± 1.1% vs WT platelets: 35.3% ± 2.0%, *p* < 0.001); 3.5 folds in OVCAR432 cells (no platelets: 12.5% ± 1.1% vs WT platelets: 43.1% ± 1.9%, *p* < 0.001); and 3.8 folds for CaOV3 (no platelets: 10.6% ± 1.2% vs WT platelets: 40.6% ± 1.7%, *p* < 0.001). *pTgfβ1^{-/-}* platelets increased BrdU incorporation by 2.3 folds in SKOV3ip1 cells (no platelets: 7.9% ± 1.1% vs *pTgfβ1^{-/-}* platelets: 18.5% ± 1.5%, *p* < 0.001); 2.0 folds in OVCAR432 cells (no platelets: 12.5% ± 1.1% vs *pTgfβ1^{-/-}* platelets: 25.2% ± 1.4%, *p* < 0.001); and 2.9 folds for CaOV3 (no platelets: 10.6% ± 1.2% vs *pTgfβ1^{-/-}* platelets: 30.7% ± 1.5%, *p* < 0.001). *pTgfβ1^{-/-}* platelets reduced the WT platelet-induced proliferation rate in SKOV3ip1 by 61.2% (*p* < 0.001), in OVCAR432 cells by 58.5% (*p* < 0.001), and in CaOV3 cells by 32.9% (*p* = 0.02).