

Figure S1 : SHIP1 is dispensable for actin polymerisation in thrombin-stimulated platelets. Wild-type and SHIP1-deficient platelets were activated with thrombin (0.5 IU/ml) in suspension under non-aggregating conditions before isolation of their cytoskeleton to quantify the amount of F-actin. Representative coomassie staining of F-actin and Western blots of actin on whole lysates were shown. Data are expressed as fold increase of resting (0 s) and are means \pm SEM of 3 independent experiments.

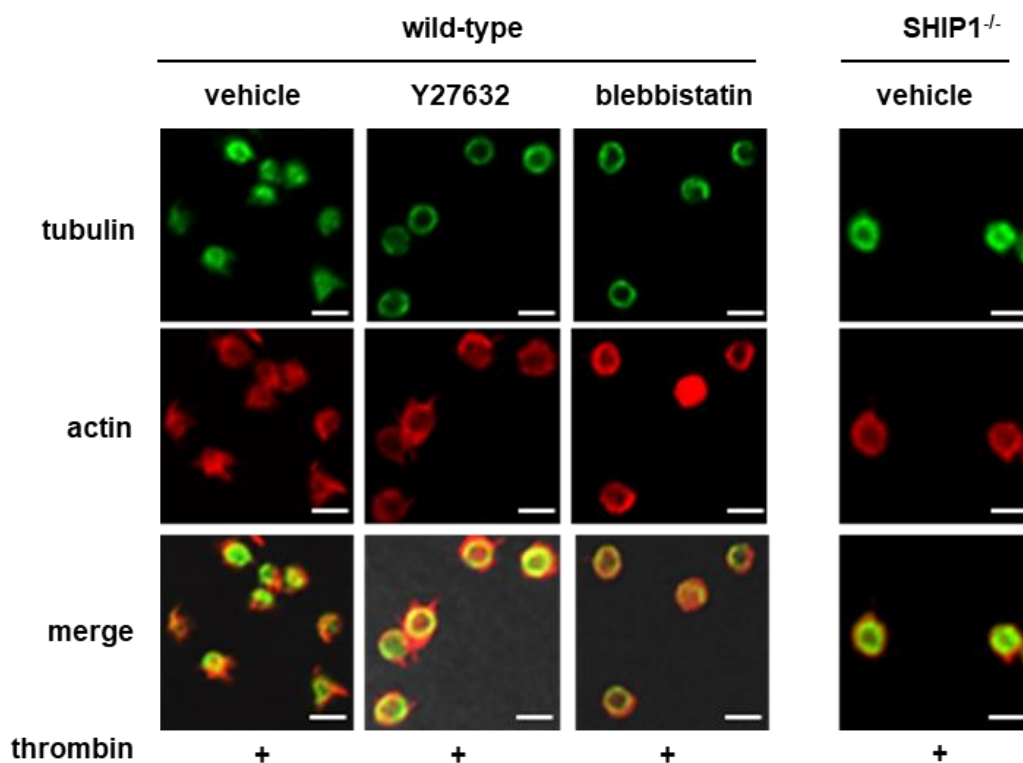


Figure S2 : Inhibition of Rho-kinase or myosin IIA activity affects thrombin-induced internal contraction in platelets. Wild-type and SHIP1-deficient platelets were preincubated with vehicle (DMSO), 20 μ M Y27632 or 10 μ M blebbistatin for 20 min before thrombin stimulation (0.5 IU/ml) for 2 min under non-aggregating suspension conditions. Representative confocal images of platelet microtubule (green) and actin cytoskeleton (red) from 4 independent experiments are shown (scale bar = 2 μ m).

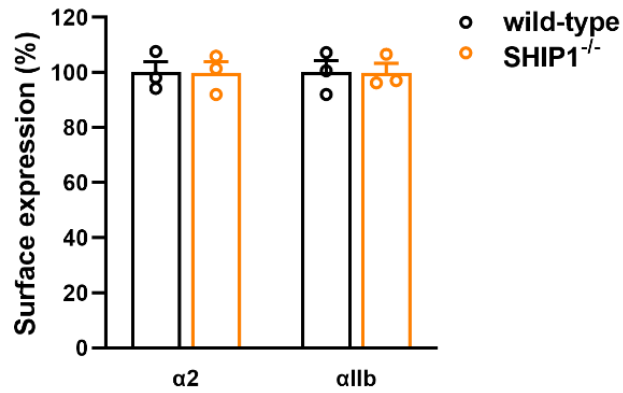


Figure S3 : Similar expression of integrin subunits on the surface of wild-type and SHIP1-deficient platelets. Platelet surface expression of $\alpha 2$ and αIIb integrin subunits was determined by flow cytometry using specific fluorescein isothiocyanate (FITC)-labeled antibodies. Results are expressed as relative percentage of surface expression and are means \pm SEM of 3 independent experiments,

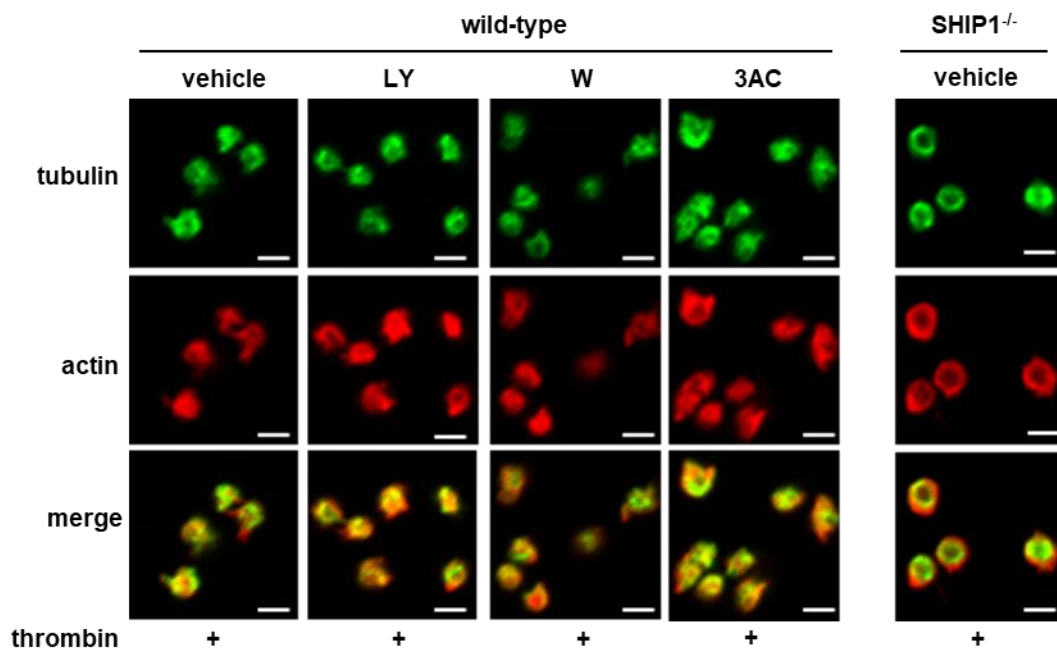


Figure S4 : SHIP1 phosphatase activity is dispensable for internal platelet contraction. Wild-type and SHIP1-deficient platelets were preincubated for 15 min in the presence of vehicle (DMSO), LY294002 (LY, 25 μ M), wortmannin (W, 50 nM) or SHIP1 inhibitor 3AC (100 μ M), then stimulated by thrombin (0.5 IU/ml) in suspension under non-aggregating conditions. Representative confocal images of platelet microtubule (green) and actin cytoskeleton (red) from 4 independent experiments are shown (scale bar = 2 μ m).