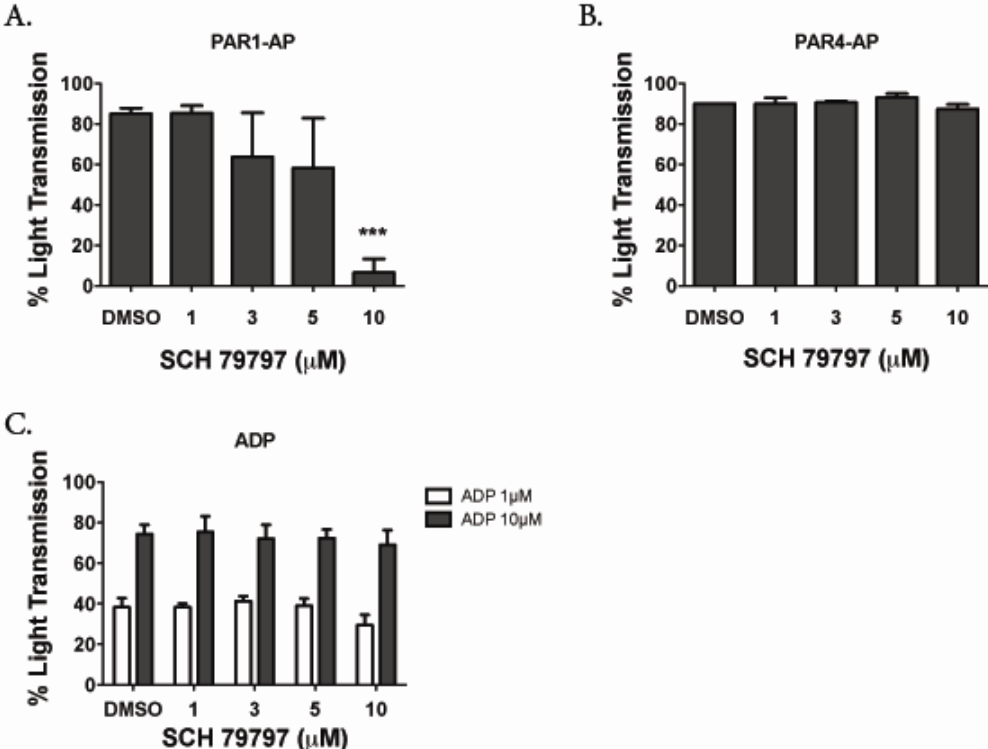
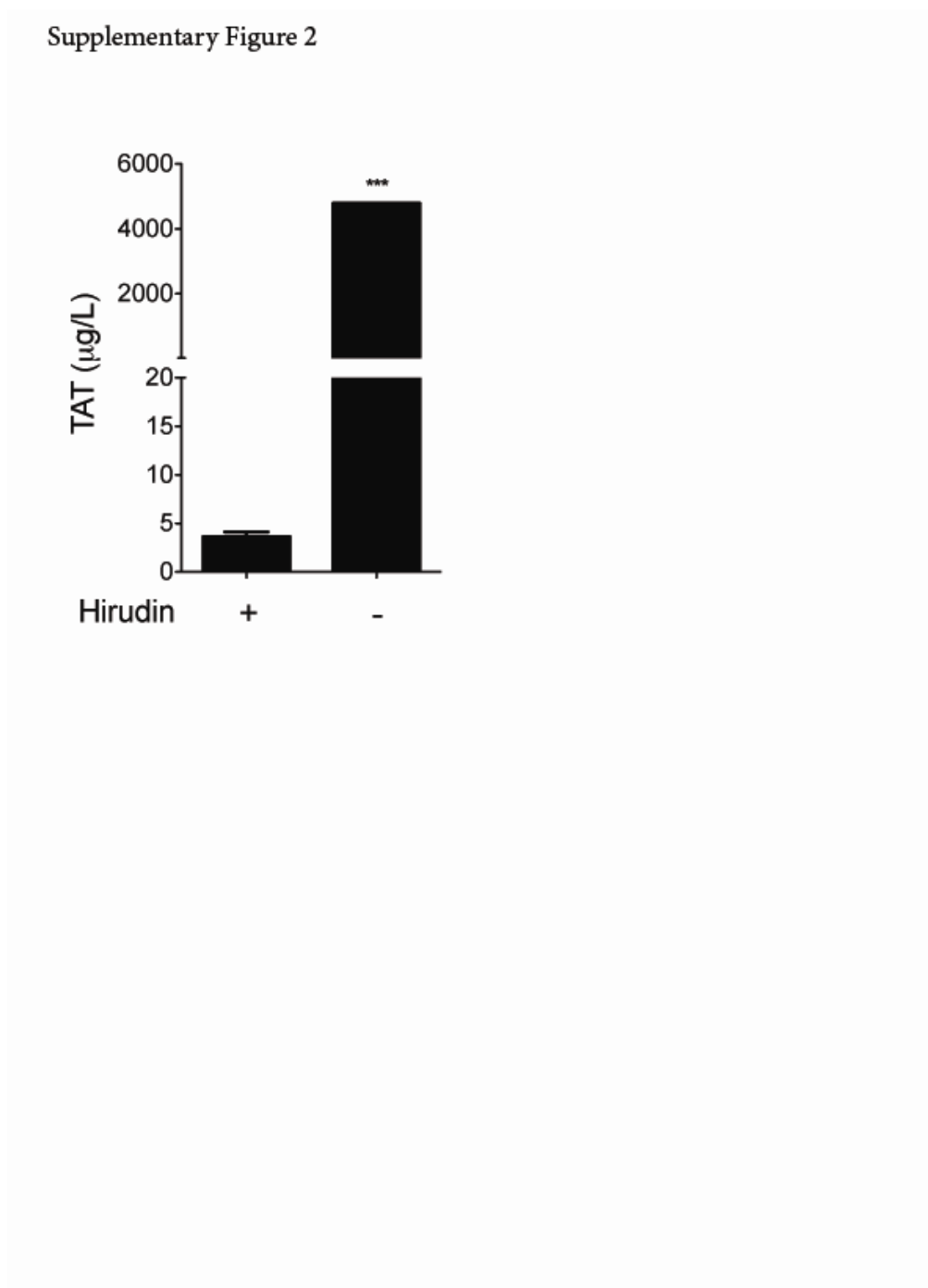


Supplementary Figures to Lee et al. “The contribution of thrombin-induced platelet activation to thrombus growth is diminished under pathological blood shear conditions” (Thromb Haemost 2012; 107.2)

Supplementary Figure 1



Supplementary Figure 1: Specific inhibition of PAR1 on human platelets by SCH-79797. Aggregation of human platelets in platelet-rich plasma was induced by a PAR1-activating peptide (PAR1-AP; 10 μ M TFLLR), a PAR4-activating peptide (PAR4-AP; 100 μ M AYPGKF), or ADP (1 or 10 μ M) in the absence or presence of the PAR1 antagonist SCH-79797 at the indicated concentrations. Data are mean \pm sem; N = 5-8. *** = P < 0.001 versus vehicle control (one way ANOVA with Dunnett's test for multiple comparisons). Note the selective inhibition of PAR1-AP-induced platelet aggregation at 10 μ M SCH-79797.



Supplementary Figure 2: Thrombin production is abolished by *in vivo* treatment with hirudin. Mice were treated with either hirudin (50 mg/kg, i.v.) or vehicle control (volume matched normal saline) 10 mins prior to the performance of the *ex vivo* whole blood flow assay. Whole blood from these mice was collected at the completion of the flow experiment and plasma TAT levels assessed via ELISA. Data are mean \pm sem; N = 3. ***, P < 0.001 (unpaired, two-tailed t-test).