Supplemental figure I. The defect in aggregation of TAM single knockout platelets were compromised when stimulated by high concentration of agonists. (A) CRP(0.6µg mL⁻¹)-induced aggregation(n=4) and (B) Thrombin(0.06U mL⁻¹)-induced aggregation(n=4); mean ± SEM, NS = not significant. One-way ANOVA followed by dunnett's multiple comparison test.
Supplemental figure II. JON/A binding is decreased on Tyro3−/− and Axl−/− platelets on in response to poly(PHG) and convulxin. Platelets from wild type, Tyro3−/−, Axl−/− or Mer−/− mice were stimulated with poly(PHG) (Ai-Aiii) and convulxin (Bi-Biii), followed by incubation with PE-labeled JON/A antibody. The samples were analyzed by flow cytometry. Mean ± SEM, n=3, NS = not significant, *** P<0.001. One-way ANOVA followed by dunnett's multiple comparison test.
Supplemental figure III. The deficiency of Tyro3 and Axl inhibits P-selectin expression on platelet surface in response to poly(PHG) and convulxin. Platelets from wild type, Tyro3−/−, Axl−/− or Mer−/− mice were stimulated with poly(PHG) (Ai-Aiii) and convulxin (Bi-Biii), followed by incubation with PE-labeled anti-P-selectin antibody. The samples were analyzed by flow cytometry. Mean ± SEM, n=3, NS = not significant, *** P<0.001. One-way ANOVA followed by dunnett's multiple comparison test.