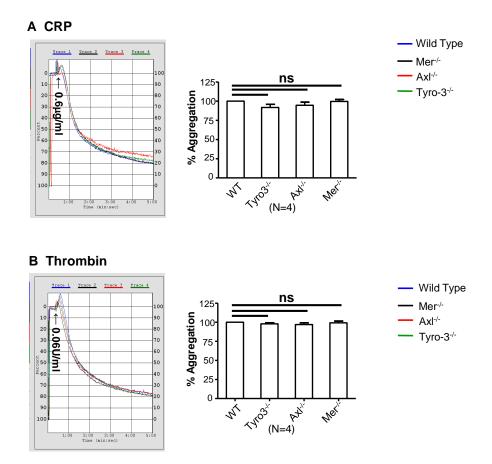
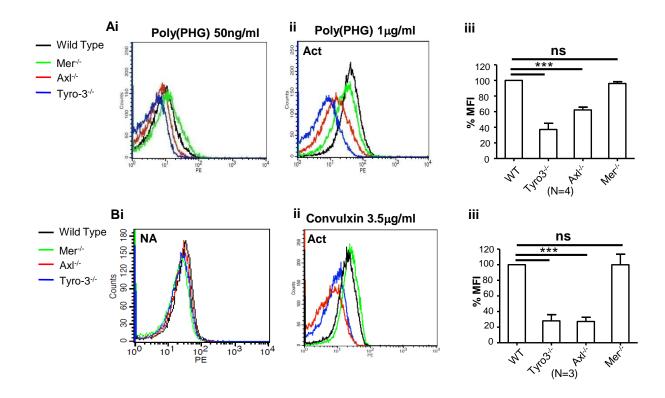
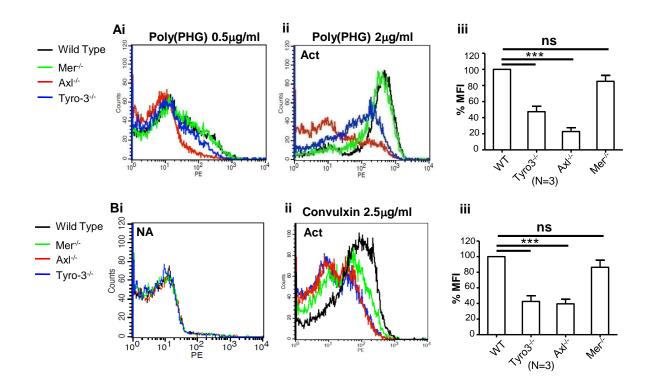
Supplemental figure I.



<u>Supplemental figure I.</u> The defect in aggregation of TAM single knockout platelets were compromised when stimulated by high concentration of agonists. (A) CRP($0.6\mu g$ mL⁻¹)-induced aggregation(n=4) and (B) Thrombin(0.06U mL⁻¹)-induced aggregation(n=4); mean \pm SEM, NS = not significant. One-way ANOVA followed by dunnett's multiple comparison test.



<u>Supplemental figure II</u>. JON/A binding is decreased on Tyro3^{-/-} and AxI^{-/-} platelets on in response to poly(PHG) and convulxin. Platelets from wild type, Tyro3^{-/-}, AxI^{-/-} 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 \pm SEM, n=3, NS = not significant, *** P<0.001. One-way ANOVA followed by dunnett's multiple comparison test.



<u>Supplemental figure III</u>. The deficiency of Tyro3 and AxI inhibits P-selectin expression on platelet surface in response to poly(PHG) and convulxin. Platelets from wild type, Tyro3-/-, AxI-/- 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 \pm SEM, n=3, NS = not significant, *** P<0.001. One-way ANOVA followed by dunnett's multiple comparison test.