SUPPLEMENTAL FIGURE LEGENDS

Supplemental Figure S1. FLNA association with SNARES mediated by GPVI and thrombin signaling. Human platelets were activated with collagen-related peptide (CRP 1 μg/mL) **(A)** or thrombin (0.1 U) **(B)** prior to solubilization with NP40 lysis buffer. Co-immunoprecipitation (co-IP) was performed on resting and activated platelets by incubating the lysates with an anti-FLNA antibody. Co-IPs were resolved by SDS-PAGE, and probed with antibodies against FLNA, STX11 and SNAP23. The negative control co-IP (IgG) is also shown.

Supplemental Figure S2. FLNA association with SNARES is potentiated by amplification pathways. Human platelets were activated with TRAP-6 (10 μM) in the presence (activated+inhibitors) or absence (activated) of inhibitors (RGDS peptide, 150 μM, apyrase, 1 U/mL, indomethacin, 5 μM) prior to solubilization with NP-40 lysis buffer. Co-immunoprecipitation (co-IP) was performed on resting and activated platelets by incubating the lysates with an anti-FLNA antibody. Co-IPs were resolved by SDS-PAGE, and probed with antibodies against FLNA, STX11 and SNAP23. The negative control co-IP (IgG) is also shown.

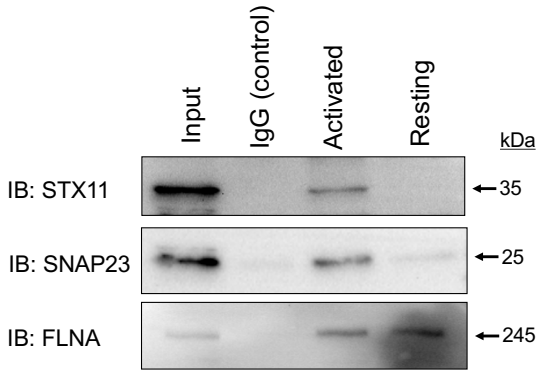
Supplemental Figure S3. The association between SNAP23 and STX11 is diminished in the absence of FLNA. A. Platelets from control (control) and platelet-specific conditional *Flna*-knockout (platelet-specific KO) mice were activated with thrombin (0.1 U) prior to solubilization with NP40 lysis buffer. Co-immunoprecipitation (co-IP) was performed using an anti-SNAP23 antibody. Co-IPs were resolved by SDS-PAGE and probed with antibody against STX11. The negative control co-IP (IgG) is also shown. **B.** Kidneys were harvested from control (control) and platelet-specific conditional *Flna*-knockout (platelet-specific KO) mice. Tissue lysates were prepared and homogenized with RIPA buffer (50 mM Tris-HCl, pH=7.4; 1% (v/v) Triton X-100; 0.2% (w/v) sodium deoxycholate; 0.2% (w/v) sodium dodecylsulfate (SDS), 1 mM sodium EDTA in the presence of protease/phosphatase inhibitors). Tissue and cell debris was removed by centrifugation and the protein concentration was determined using a BCA assay. Equal amounts of protein from control and platelet specific KO mice was resolved by SDS-PAGE and blotted for FLNA. GAPDH is shown as a loading control.

Supplemental Figure S4. ATP release in response to PMA is unaffected by FLNA. Platelets from control (red bars) and FLNA-null (Platelet-specific KO, blue bars) platelets were treated with the phorbol ester PMA (100 nM) for 5 mins and the resultant ATP secretion, as measured by luminescence, using the Chronolume luciferase reagent. Data are mean ± SEM (ns, not significant, based on Student's t-test).

Supplemental Figure S5. ATP release in response to CRP is unaffected by scavenging of external ADP. Platelets from control (red bars) and FLNA-null (Platelet-specific KO, blue bars) platelets were activated with CRP 1 μg/mL in absence or presence of the ADP scavenger apyrase (1 U/mL) for 5 mins and the resultant ATP secretion, as measured by luminescence, using the Chronolume luciferase reagent. Data are mean ± SD.

Supplemental Figure S1

A.



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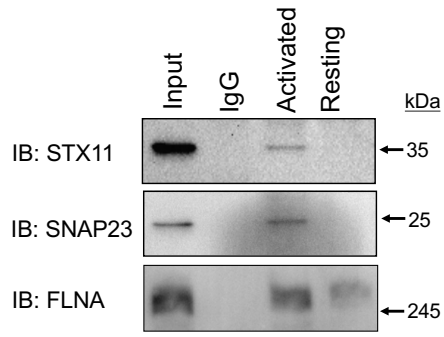
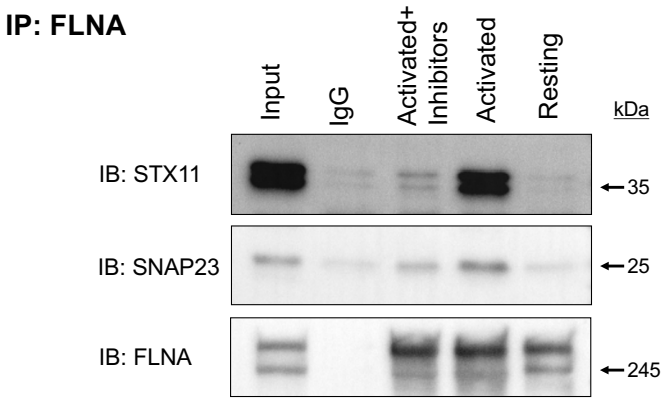
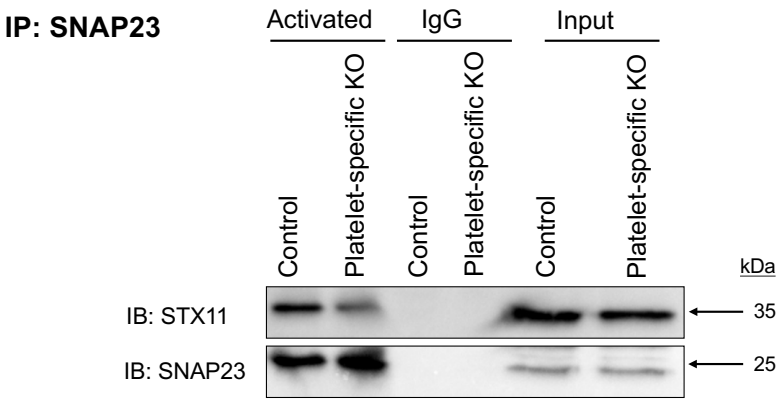


Supplemental Figure S2



Supplemental Figure S3

A.



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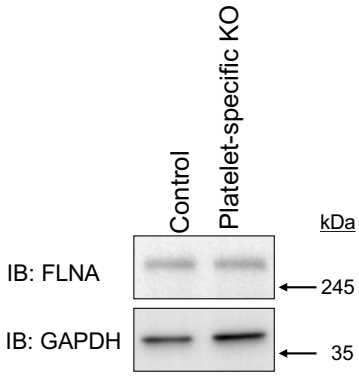
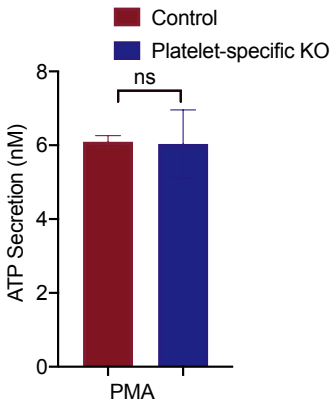


Supplemental Figure S4



Supplemental Figure S5