

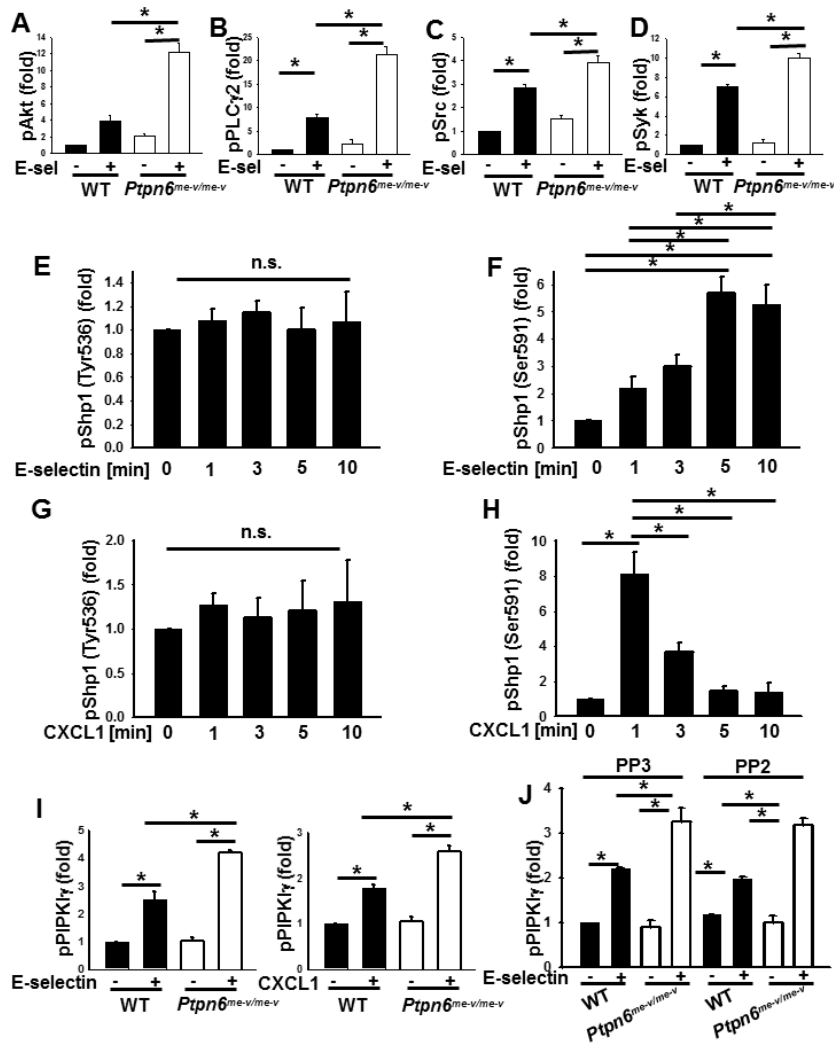
Supplemental Material

Crosstalk between Shp1 and PIPKI γ controls leukocyte recruitment.

Anika Stadtmann,^{*,†} Helena Block,^{*,†} Stephanie Volmering,^{*,†} Clare Abram,[‡] Charlotte Sohlbach,^{*,†} Mark Boras,^{*,†} Clifford A. Lowell,[‡] and Alexander Zarbock^{*,†}

^{*} Department of Anesthesiology, Intensive Care and Pain Medicine, University of Muenster, Albert-Schweitzer-Campus 1, 48149 Muenster, Germany, [†] Max-Planck Institute for Molecular Biomedicine, Roentgenstr. 20, 48149 Muenster, Germany, [‡] Department of Laboratory Medicine and the Program in Immunology, University of California, 505 Parnassus Avenue, San Francisco, CA94143-0100, USA,

Figure S1



Supplementary Figure 1: Densitometric analysis of Immunoprecipitations and Western Blots. Densitometric quantification of immunoprecipitations and Western Blots comparing the phosphorylation of Akt (A), PLC γ 2 (B), Src kinases (C) and Syk (D) following E-selectin stimulation in WT and *Ptpn6^{me-v/me-v}* (n=3) neutrophils shown in Figure 2G. Densitometric quantification of Western Blots comparing the phosphorylation of the Shp1 (Ser591 or Tyr536) following E-selectin (E+F) or CXCL1 (G+H) stimulation in WT neutrophils shown in Figure 5A-D. Densitometric quantification of Western blots (shown in Figure 6A+B) of immunoprecipitations of PIPKI γ in unstimulated, E-selectin and CXCL1 stimulated wild type and *Ptpn6^{me-v/me-v}* neutrophils showing the phosphorylation of PIPKI γ in the absence (I+J) or presence (J) of a Src family kinase inhibitor.