Figure S1. Redox and SFK signaling at wounds. (A) Immunofluorescence of pSFK (phosphorylation of SFK activation loop tyrosine) in 3-dpf Tg(krt4-tdTomato) at 0 min after tail transection. (B) Immunofluorescence of pSFK and Cadherin in 3-dpf larvae at 0 min after tail transection. (C) Ratiometric image of pSFK/krt4-tdTomato in 3-dpf larvae without wounds. (D) Ratiometric image of pSFK/krt4-tdTomato in 3-dpf larvae immediately after tail transection. (E) Immunofluorescence of pSFK (phosphorylation of SFK activation loop tyrosine) by mAb (D49G8) in 3-dpf larvae at 0 min after tail transection. (F) Diagram of drug treatment in regeneration assays. (G) Quantification of tail fin length at 0 min after wounding. (H) Quantification of regenerated tail fin length at 3 d after wounding. DMSO: 21 larvae; PP3: 23 larvae. 2-dpf larvae were treated with DMSO or PP3 starting from 1 h before wounding and ending at 1 h after wounding, and regenerated tail fin length was quantified at 3 d after wounding. (G and H) *, P < 0.05; one-way ANOVA with Dunnett’s post-test (G) and two-tailed unpaired t test (H). Horizontal lines indicate means. Numbers indicate number of larvae that was used. hpw, hour postwounding. Bars, 50 µm.
Figure S2. Early redox and SFK signaling regulates late resolution of inflammation. (A) Diagram of drug treatment during wound responses. 3-dpf zebrafish larvae were treated according to the diagram, wounded at tail fins with a needle, and fixed at 1, 2, or 6 h after wounding. (B) Neutrophil recruitment to wounded fins at 1, 2, and 6 h after wounding was quantified by staining neutrophils with Sudan black (1 h/DMSO: 24 larvae; 1 h/DPI: 27 larvae; 1 h/PP2: 22 larvae; 2 h/DMSO: 22 larvae; 2 h/DMSO → DPI: 23 larvae; 2 h/DMSO → PP2: 22 larvae; 2 h/DPI → DMSO: 22 larvae; 2 h/PP2 → DMSO: 23 larvae; 6 h/DMSO: 22 larvae; 6 h/DMSO → DPI: 23 larvae; 6 h/DMSO → PP2: 24 larvae; 6 h/DPI → DMSO: 22 larvae; 6 h/PP2 → DMSO: 24 larvae). Right pictures show representative pictures of Sudan black staining at 6 h after wounding. Error bars indicate SEMs. *, P < 0.05; one-way ANOVA with Dunnett's posttest. Bars, 50 µm.
Figure S3. Components of early wound signaling. (A) Imaging of GCaMP in 2-dpf larvae. PP2 or DPI does not inhibit the wound-induced Ca²⁺ flashes. (B) Quantification of regenerated tail fin length at 3 d after wounding (DMSO: 22 larvae; 100 µM PD98059: 22 larvae). 2-dpf larvae were treated with DMSO or PD98059 starting from 1 h before and ending at 1 h after wounding, and regenerated tail fin length was quantified at 3 d after wounding. Dotted lines indicate tail transection. (C) Effects of fynb and/or yes knockdown on normal development of tail fins. Representative pictures of morphant tail fins at 5 dpf. (D) Quantification of fin length at 5 dpf (control [Ctrl]: 10 larvae; fynb MO1: 20 larvae; fynb MO2: 9 larvae; yes MO: 9 larvae; fynb MO1/yes MO: 13 larvae). (E) Quantification of fin width at 5 dpf (control: 10 larvae; fynb MO1: 20 larvae; fynb MO2: 9 larvae; yes MO: 9 larvae; fynb MO1/yes MO: 13 larvae). (B and E) *, P < 0.05; two-tailed unpaired t test (B) and one-way ANOVA with Dunnett’s post test (E). Horizontal lines indicate means. Bars, 50 µm.
Video 1. **Live imaging of GCaMP3 probe in a 2-dpf larva immediately after wounding.** Images were acquired by time-lapse confocal microscopy using a laser-scanning confocal microscope (FluoView FV1000; Olympus). Frames were taken every 15 s for 8 min.

Video 2. **Live imaging of GCaMP3 probe in DMSO- or 1 µM thapsigargin-treated 2-dpf larvae immediately after wounding.** Images were acquired by time-lapse confocal microscopy using a laser-scanning confocal microscope (FluoView FV1000; Olympus). Frames were taken every 15 s for 6 min.