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**Fig. S1**  CRP dose response for α-granule secretion and CRP peptide control test on cardiomyocyte viability. (A) Washed platelets ($2 \times 10^8$/mL) from WT mice were stimulated with increasing concentrations of CRP (0.1–50 µg/mL) and monitored for α-granule secretion by measuring surface CD62P levels. Response curves are expressed as percentage of maximal Geo-mean values, are shown, and represent two independent experiments. Gray circle inset denotes chosen CRP concentration (10 µg/mL) throughout the article. (B) Cardiomyocytes, pretreated with releasates from vehicle-treated (control) WT platelets or CRP-peptide (10 µg/mL), were subjected to ischemic injury and analyzed for changes in cell viability, with nonischemic (oxygenated control) samples monitored at 0- and 240-minute time points. Data mean ± SD, n = 3.

**Fig. S2**  Quantification of platelet dense granule marker, ATP, in fractionated platelet releasates and the effect of adenosine on cardiomyocyte viability during ischemia. (A) Washed platelets ($2 \times 10^8$/mL) from WT mice were treated with vehicle (control) or 10 µg/mL CRP (stimulated) for 15 minutes at 37°C. The isolated releasate was left untreated (unfiltered) or fractionated using a 3-kDa MW cutoff with centrifugation at 14,000g for 20 minutes. The filtered (<3 kDa) and retained (>3 kDa) fractions were readjusted to their original volume and, together with unfiltered samples, analyzed for ATP luminescence using luciferin–luciferase (Chrono-log, United States) on a microplate reader (Tecan Infinite M200Pro, Switzerland). Post measurement, 1 nmol of ATP standard was added as a reference value for absolute quantification of ATP. Data are mean ± SD; n = 3, *p < 0.05, **p < 0.001. Two-way ANOVA with Bonferroni post hoc test. (B) Cardiomyocytes pretreated with vehicle (Tyrode's) or 50 µM adenosine were subjected to ischemic injury and monitored for changes in cell viability, with nonischemic (oxygenated control) samples monitored at 0- and 240-minute time points. Data mean ± SD; n = 3, ns (nonsignificant).
The CXCR4 and TGFBR inhibitors, AMD3100 and SB431542, effectively block the protective effect of recombinant SDF-1α and TGF-β1. (A, B) Cardiomyocytes were pretreated with vehicle control (0.1% DMSO) and (A) 0.5 µM AMD3100 (CXCR4 antagonist) or (B) 1 µM SB431542 (TGFBR1 antagonist) for 30 minutes, prior to preconditioning with recombinant SDF-1α (100 ng/mL) or TGF-β1 (100 ng/mL), respectively. Cardiomyocytes were then monitored for changes in viability every 60 minutes for up to 240 minutes of ischemic injury, with nonischemic (oxygenated control) samples monitored at 0- and 240-minute time points. (A) and (B) are representative of two independent experiments.

PKC inhibition with BIM I does not alter basal responses of cardiomyocytes during ischemic injury. Cardiomyocytes were pretreated with vehicle control (0.1% DMSO) or 10 µM BIM I (PKC inhibitor) for 30 minutes and then monitored for changes in cell viability for up to 240 minutes of ischemic injury. Figure shown is representative of two independent experiments.