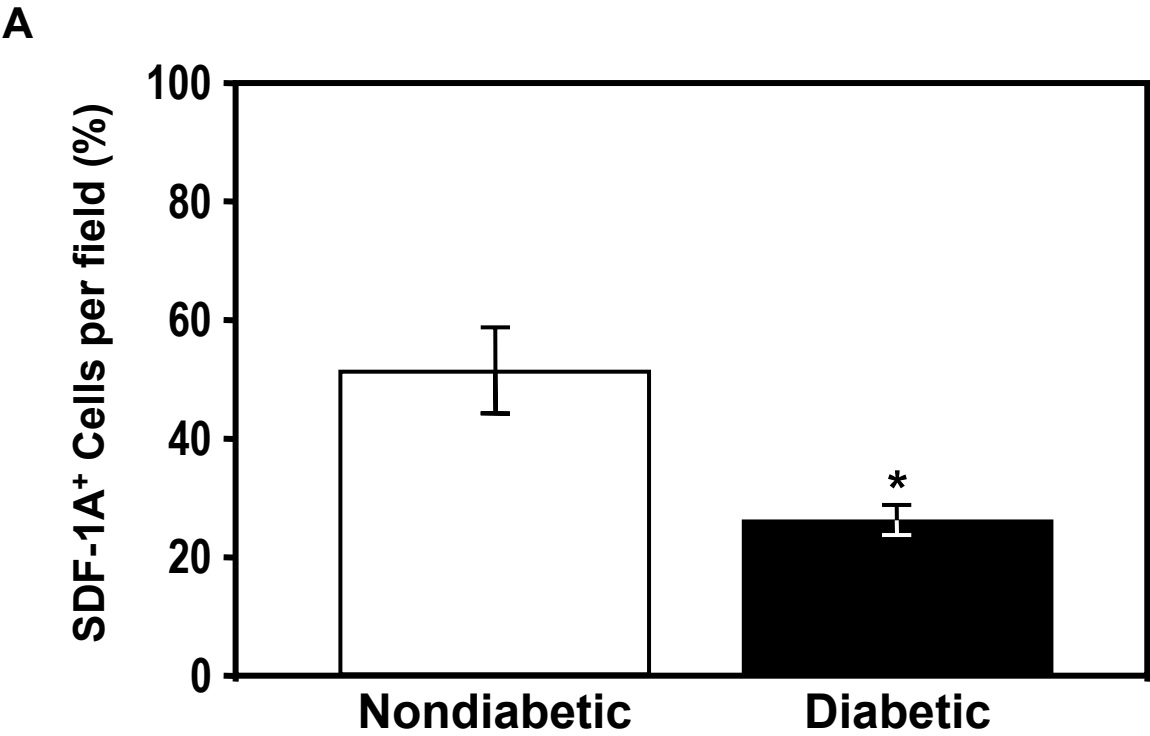
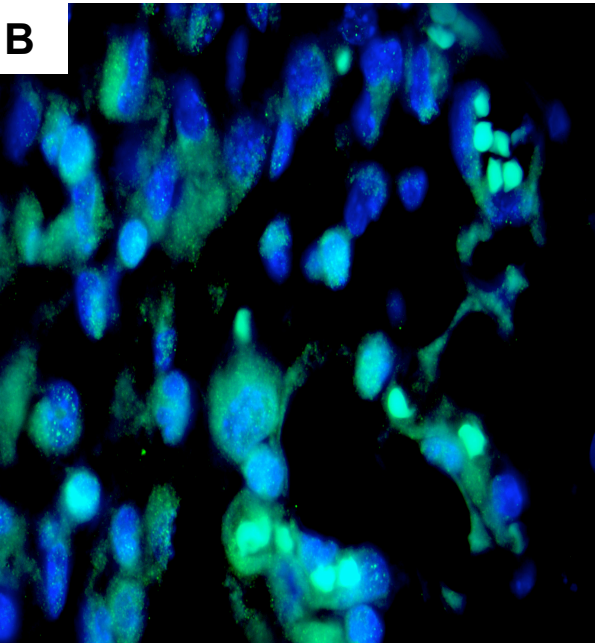


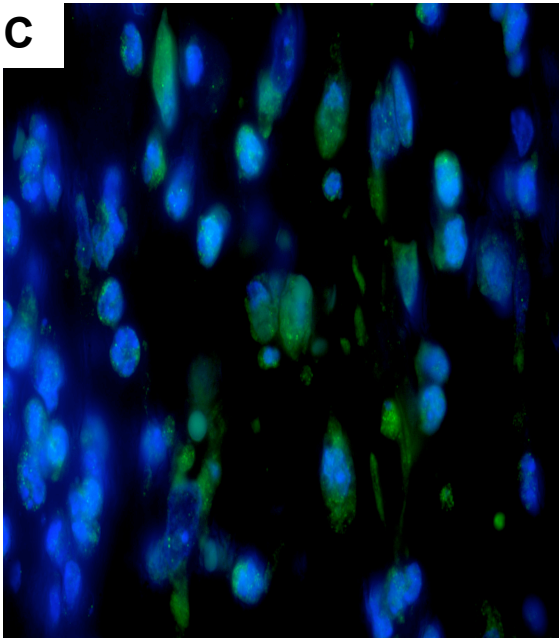
Supplemental Figure 1



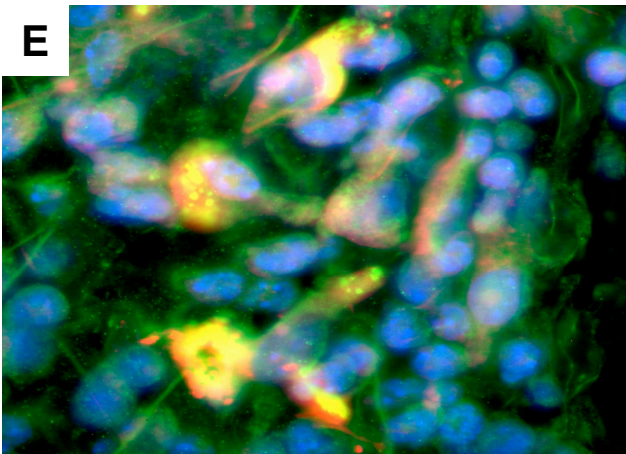
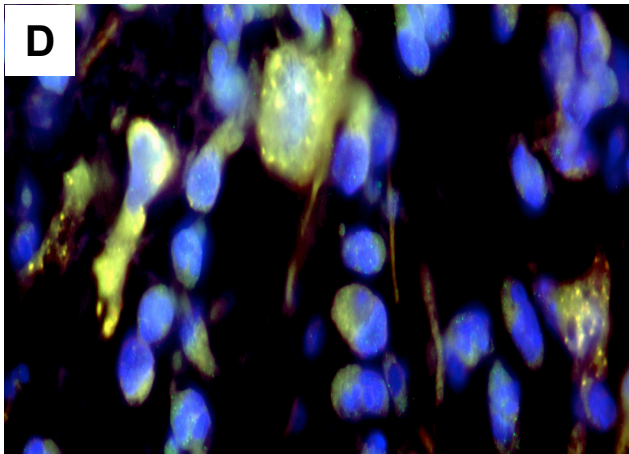
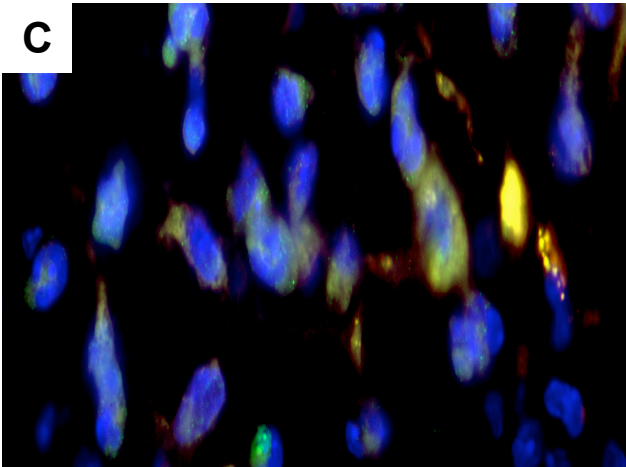
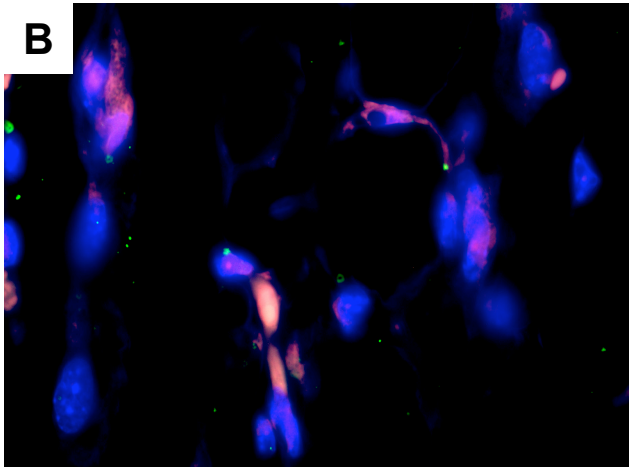
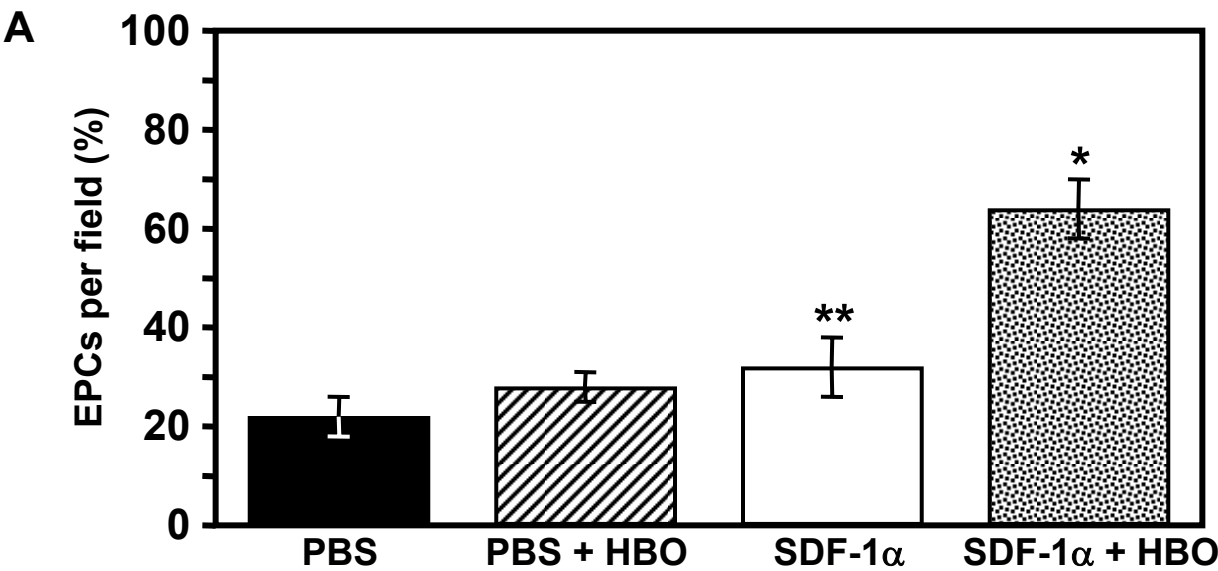
Nondiabetic



Diabetic

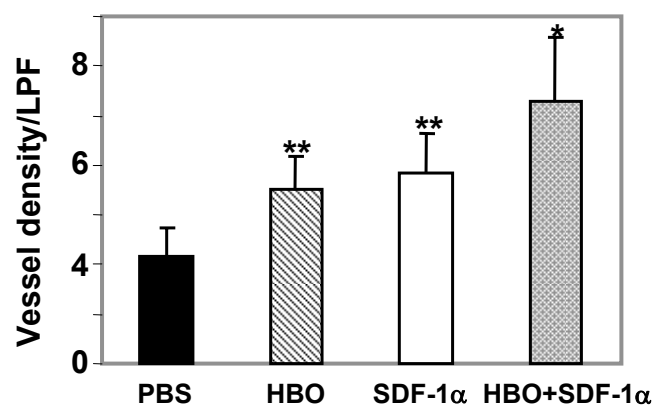


Supplemental Figure 2

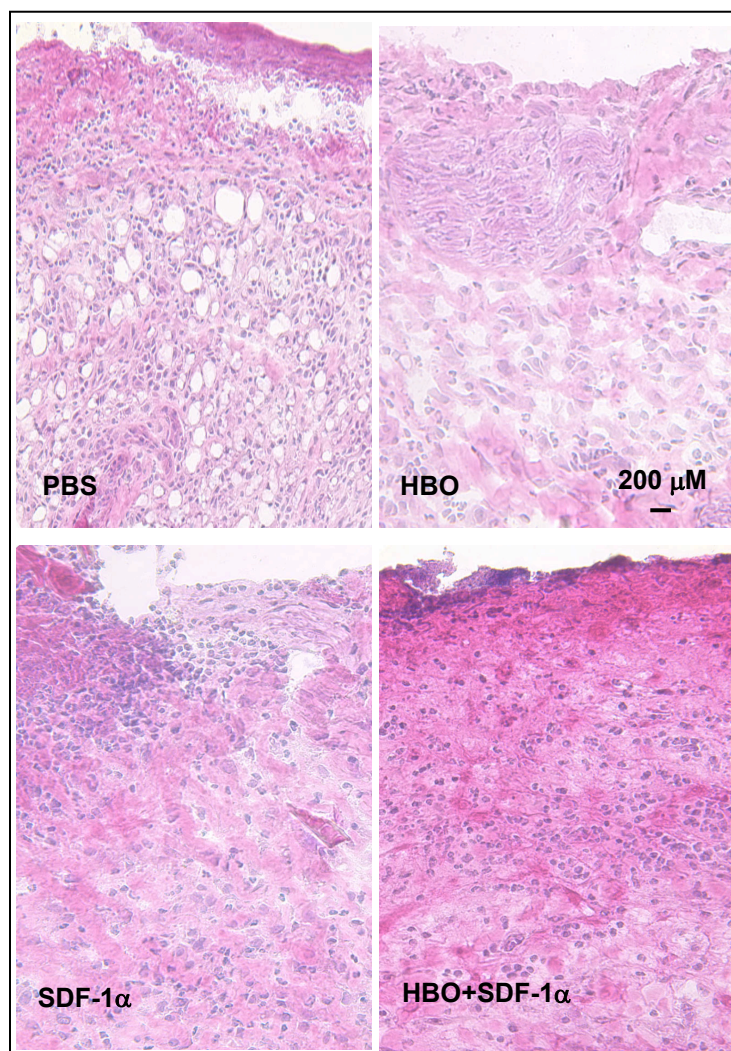


Supplemental Figure 3

A



B



Supplemental Figure 1.

Decreased numbers of SDF-1 α ⁺ expressing cells in peripheral wounds of diabetic mice.

(A) Wounds were examined for SDF-1 α ⁺ expression in diabetic (n=5) and nondiabetic (n=5) mice by fluorescence immunostaining 24 h post-wounding and 10 d after STZ treatment. For each animal, the percentage of cells expressing SDF-1 α was quantified relative to the total wound cellularity in 5 serial cross-sections per wound, counting 10 random high power fields (HPF) at 100X magnification. Wounds harvested from diabetic mice demonstrated significantly fewer cells expressing SDF-1 α as compared to nondiabetic controls. * $P < .005$. (B-C) Representative SDF-1 α ⁺ staining of wound sections of nondiabetic (B) and diabetic (C) animals are shown. Sections (5 μ m thick) were stained for cells with anti-SDF-1 α antibody and Alexa 488-conjugated secondary antibody (green). Nuclei were counterstained with Hoescht dye (blue).

Supplemental Figure 2.

Impaired EPC homing to wound tissue in diabetes is reversed by cutaneous administration of SDF-1 α . 4 groups (n=5 per group) of wounded diabetic mice were treated with daily wound injections of either SDF-1 α or PBS \pm daily HBO. After 3 days of treatment, wounds were harvested and analyzed by fluorescent immunostaining of tissue sections (5 μ m thick) with anti-CXCR4 antibody with Alexa 488-conjugated secondary antibody (green) and anti-VEGFR2 antibody with PE-conjugated secondary antibody (red). Nuclei were counterstained with Hoescht dye (blue). EPC were identified as cells double-labeled with CXCR4 and VEGFR2. (A) Quantitation of EPCs in PBS controls, PBS + HBO, SDF-1 α , and SDF-1 α + HBO treated diabetic mice are

shown. For each animal, 10 random high-power fields (100X magnification) from 5 serial cross-sections were analyzed and the percentage of cells expressing both CXCR4 and VEGFR2 was quantified relative to the total wound cellularity. All data are expressed as mean \pm SEM based on three experiments. SDF-1 α + HBO treated mice had a significant rise in the percentage of endothelial progenitor cells compared to PBS controls, PBS + HBO, and SDF-1 α treated animals (* P <.05). SDF-1 α treated animals had a significant increase in tissue EPC when compared to PBS controls (** P <.05). HBO did not significantly change the percentage of EPC within wounds. (B-E) Representative wound sections of PBS controls (B), PBS + HBO (C), SDF-1 α (D) and SDF-1 α + HBO (E) are shown.

Supplemental Figure 3.

(A) Blood vessel density in healing wounds. Vessels were stained with anti-VEGFR2-FITC in wounded tissue sections. For each sample, 10 random low-power fields (LPF, X20) from 5 serial cross-sections were analyzed and the number of vessels was counted.

(B) H&E staining show overall cellularity and stromagenesis in healing wounds at d 6. SDF1 α +HBO treatment strongly enhances stromagenesis compared to other groups.