FIGURE LEGENDS:

Figure 1: Secretion of growth factors and matrix enzymes by progenitor cells after 48 hours (0 hours time point) of normal culture conditions (NCC) or hypoxic culture conditions (HCC) and 48 hours (48 hours time point) of subsequent NCC culture. A) HGF secretion was highest for NCC hASCs at the 0 hours time point, but highest for HCC hASCs at the 48 hours time point. B) All three cell types secrete MMP-2. C) Among HCC hASCs, normally cultured NCC hASCs, and hBMSCs, hBMSCs secrete the most VEGF per cell. HCC hASCs secrete a statistically similar amount of HGF as NCC hASCs and a similar amount of VEGF as hBMSCs. D) hASCs produce more TIMP-1 than hBMSCs, regardless of culture condition. (± standard deviation; *, $p<0.001$; †, $p<0.01$; #, $p<0.05$; X, $p<0.001$ vs. 0 hours time point; O, $p<0.05$ vs. 0 hours time point).

Figure 2: A) In scratch test migration assays, normally cultured (NCC) hASCs consistently showed higher baseline migration and higher migration in the presence of PDGF-BB than HCC hASCs. hBMSCs did not exhibit an increase in migration in the scratch test assay in the presence of PDGF-BB (gray bars) compared to untreated controls (black bars). (± standard deviation; *, $p<0.001$ vs. untreated control same cell group/time point; #, $p<0.05$ vs. untreated control same cell group/time point; †, $p<0.001$ vs. hBMSC at the same time point; ‡, $p<0.05$ vs. hBMSC at the same time point; §, $p<0.01$ vs. NCC hASC at the same time point). B) NCC ASCs were able to migrate significantly more toward a gradient of PDGF-BB, but not VEGF, than untreated negative controls in Boyden chamber migration assays. BMSCs were able to migrate more in the presence of both VEGF and PDGF-BB than negative controls, but baseline hASC migration was higher than that of BMSCs (± standard deviation; *, $p<0.001$ vs. untreated control same cell group/time point; #, $p<0.01$ vs. untreated control same cell group/time point; †, $p<0.001$ vs. hBMSC at the same time point; ‡, $p<0.05$ vs. hBMSC at the same time point; @, $p<0.01$ vs. hASCs treated with VEGF).

Figure 3: DiI-labeled hASCs (red) exhibit perivascular positioning with respect to blood vessels (blue) in inflamed and angiogenic rat mesenteric windows as well as pericyte markers like NG2 and smooth muscle α-actin (SMA) (A,C, respectively). SMA and NG2 staining of inflamed rat mesenteries injected with vehicle PBS instead of cells showed positive staining of native pericytes (B) and smooth muscle cells (D). Nerves in the vascular bed also stained positively for NG2 (A).

Figure 4: For all injected cell groups, the number of cells associating closely with blood vessels was significantly lower by Day 60 in unstimulated mesenteries compared to inflamed mesenteries. hBMSCs showed steady increase in perivascular association in these tissues, and HCC hASCs showed a biphasic response in stimulated tissues (± standard error, *, $p<0.001$; †, $p<0.01$; #, $p<0.05$; X, $p<0.001$ vs. unstimulated; +, $p<0.01$ vs. unstimulated; O, $p<0.05$ vs. unstimulated).

Figure 5: The number of cells exhibiting pericyte-like phenotypes (A, concurrent PDGFbR expression and perivascular positioning; B, concurrent NG2 expression and perivascular positioning; C, concurrent SMA expression and perivascular positioning) in each group was low at all time points in unstimulated mesenteries, but all groups showed increases in pericyte-like

phenotype by Day 60 when injected into inflamed mesenteries. hBMSCs showed an increase in pericyte-like phenotype slightly earlier than either hASC group in the Compound 48/80-treated tissues (± standard error, *, $p<0.001$; †, $p<0.01$; #, $p<0.05$; X, $p<0.001$ vs. unstimulated; +, $p<0.01$ vs. unstimulated; O, $p<0.05$ vs. unstimulated).

Figure 6: A) hBMSCs and normally cultured (NCC) hASCs showed a general increase in vascular length density over time in inflamed mesenteries, but hypoxia preconditioned (HCC) hASCs produced a biphasic length density profile over time. B) Angiogenic efficiency (length density per cell) was found to increase over 60 days for NCC hASCs, but hBMSCs seemed to peak in angiogenic efficiency at Day 30. HCC hASCs showed an increase in angiogenic efficiency from Day 10 to Day 60 in unstimulated mesenteries, but no change in angiogenic efficiency when injected into stimulated mesenteries (± standard error, *, $p<0.001$; †, $p<0.01$; #, $p<0.05$; X, $p<0.001$ vs. unstimulated; +, $p<0.01$ vs. unstimulated; O, $p<0.05$ vs. unstimulated).

Supplementary Figure 1: Viability assay for cells treated with 48 hours of hypoxic (HCC) or normal (NCC) culture conditions. A) and B) HCC hASCs survive 48 hours of hypoxia as evidenced by the fact that they show similar morphology, positive staining for calcein and negative staining for calcein EthD-1 as NCC hASCs. C) and D) HCC hBMSCs also showed positive staining for calcein and negative staining for EthD-1, illustrating that they also survive 48 hours of hypoxia; however, cell morphology was distinctly more rounded and less spread than NCC hBMSCs.

Supplementary Figure 2: Cytotoxicity in hBMSCs exposed to 5 μg/ml of the inflammatory agent Compound 48/80 for 6 days. hBMSCs viability was not greatly affected by the presence of Compound 48/80, implying that any Compound 48/80 injected into the peritoneal cavities of rats containing hBMSCs would not have a significant impact on hBMSC survival in vivo.

Supplementary Figure 3: In vitro expression of SMA in hASC and hBMSC following hypoxic (HCC) or normal (NCC) culture conditions (see Supplementary Methods for details). Expression was highest in all groups following 24 hours of NCC, including cells treated with HCC and then NCC. A) Expression of SMA in hASC and hBMSC cells 24 hours after initial plating and serum starvation (NCC only). B) Expression of SMA immediately following 48 hours of HCC or NCC treatment (t0). C) Expression of SMA following an additional 48 hours of NCC (hASC HCC cells were cultured in HCC for 48 hours followed by 48 hours of NCC). Data shown as mean ± standard error; *, $p<0.001$; #, $p<0.05$; X, $p<0.001$ vs previous time point; O, $p<0.01$ vs. previous time point.

Supplementary Figure 4: Untreated controls to Boyden chamber migration experiments show that HCC does not have a significant effect on hASC migration; however, HCC significantly reduces the number of migrated hBMSCs. Data shown as mean ± standard error; *, $p<0.001$.

Supplementary Figure 5: Average number of cells per field of view in animals receiving Compound 48/80 injections and those not receiving Compound 48/80. The number of cells observed dropped from Day 10 to Day 30 for all cell groups, regardless of inflammatory stimulation. In animals receiving hASCs and Compound 48/80 injections, the number of cells per field of view increased significantly from Day 30 to Day 60.

Supplementary Figure 6: Number of cells expressing platelet-derived growth factor β-receptor (PDGFβR), regardless of proximity to blood vessels. Cells in unstimulated tissues expressed very low levels of PDGFβR expression while expression levels became significantly higher over the course of 60 days for each cell type injected into animals stimulated with Compound 48/80. In those tissues expression levels also increased over time. In all cell types, PDGFβR expression increased over time in tissues treated with Compound 48/80 (± standard error, *, $p<0.001$; †, $p<0.01$; #, $p<0.05$; X, $p<0.001$ vs. unstimulated; O, $p<0.05$ vs. unstimulated).

Supplementary Figure 7: Number of cells expressing neuron glial factor-2 (NG2), regardless of proximity to blood vessels. NCC hASCs and hBMSCs in unstimulated tissues expressed NG2 at a significantly lower rate than NCC hASCs and hBMSCs injected into animals stimulated with Compound 48/80. In all cell types, NG2 expression increased over time in tissues treated with Compound 48/80 (± standard error, *, $p<0.001$; †, $p<0.01$; #, $p<0.05$; X, $p<0.001$ vs. unstimulated; +, $p<0.01$ vs. unstimulated).

Supplementary Figure 8: Number of cells expressing smooth muscle actin (SMA), regardless of proximity to blood vessels. NCC hASCs and hBMSCs in unstimulated tissues expressed SMA at a significantly lower rate than NCC hASCs and hBMSCs injected into animals stimulated with Compound 48/80. In all cell types, SMA expression increased over time in tissues treated with Compound 48/80 (± standard error, *, $p<0.001$; †, $p<0.01$; #, $p<0.05$; X, $p<0.001$ vs. unstimulated; O, $p<0.05$ vs. unstimulated).

Supplementary Figure 9: Numerical tabulation of average pericyte marker expression in injected cells. This data is illustrated in Figures 4 and 5 as well as Supplemental Figures 4, 5, and 6.

Supplementary Figure 10: Flow chart representing field of view (FOV) selection during data acquisition. Methodology used to determine FOVs captured during imaging had an impact on the validity of PBS negative control values. Image selection was based first on the presence of cells then the highest apparent vascular length density. As PBS controls did not have cells present, the absolute highest vascular length densities were always recorded. This was not always the case for experimental groups in which cells were injected. For this reason, PBS negative controls were removed from the study, and data was analyzed by comparing experimental groups to one another rather than relative to a negative control.