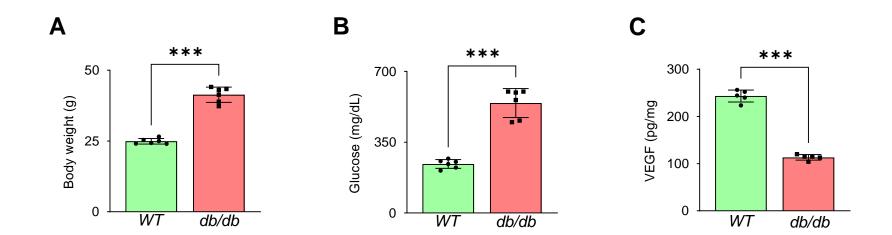
Supplemental Table 1. Primers used in the real-time RT-PCR

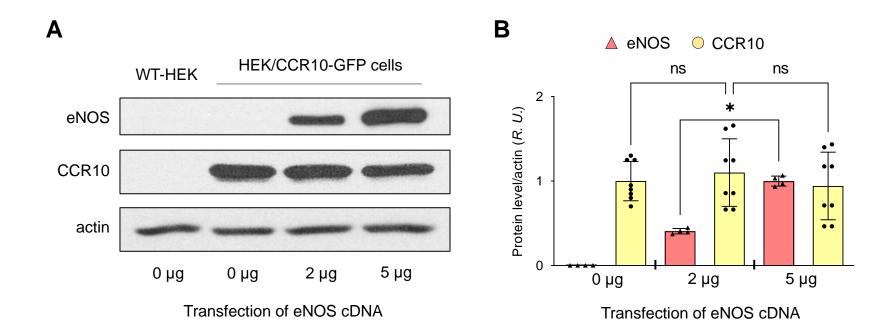
Name	Target	Forward primer	Reverse primer
CCL28	Human	ACTACAACCTCCACCTACCT	GGGCTGACACAGATTCTTCT
CCR10	Human	CACACTTGGTCTCCGTCATC	CTTCACCGTCTGCGTGAG
GAPDH	Human	CTTTGGTATCGTGGAAGGACTC	AGTAGAGGCAGGGATGATGT
CCL27	Mouse	AGCCTCCCGCTGTTACTGT	AGTTTTGCTGTTGGGGGTTT
CCL28	Mouse	ATGAGAGCCTCAGAGGTAAAGA	TGTTCTGTGCTTTCTCGTAGTG
CCR3	Mouse	CCAGCTGTCAGCAGAGTAAA	CTCACCAACAAAGGCGTAGA
CCR10	Mouse	TCGCTGTTTCTGGCTCTAC	AGTCTGCGTGAGGCTTTC
IL-6	Mouse	CGAGAGTCCTTCAGAGAGATACA	CCTTCTGTGACTCCAGCTTATC
Arg1	Mouse	CATGGGCAACCTGTGTCCTT	CGATGTCTTTGGCAGATATGCA
TGF-β1	Mouse	CTTCAGCTCCACAGAGAAGAAC	TGTCCAGGCTCCAGATGTA
TGF-β2	Mouse	TAAAGAGGTCACCCGCGTGCTAAT	ACTGCTTCCCGAATGTCTGACGTA
IL-4	Mouse	ACAGGAGAAGGGACGCCAT	GAAGCCCTACAGACGAGCTCA
IL-13	Mouse	GGAGCTGAGCAACATCACACA	GGTCCTGTAGATGGCATTGCA
IGF-1	Mouse	TCATGTCGTCTTCACACCTCTTCT	CCACACGAACTGAAGAGCAT
Collagenase I	Mouse	CAAGGGAGAGAGTGGTAACAAG	GGGAACCTCTCTTCCTTCTTC
Collagenase III	Mouse	AGGATGGCTCTAAACATAC	CTTGATCAGGACCACCAATATCA
eNOS	Mouse	CCCTCAGGTTCTGTGTTTT	GAGTCAGCCCTGGTAGTAATTG
CD31	Mouse	CACCCATCACTTACCACCTTATG	TGTCTCTGGTGGGCTTATCT
VEGFR2	Mouse	GTCCGAATCCCTGTGAAGTATC	GTGAGTTCATCGCCAACAATC
VE-cadherin	Mouse	CAGTGACAGAGGCCAATTCT	GCCTCCACAGTCAGGTTATAC
GAPDH	Mouse	GGGTGTGAACCACGAGAAATA	GTCATGAGCCCTTCCACAAT

Footnote: All primers were supplied by Integrated DNA Technologies IDT (Coralville, IA).



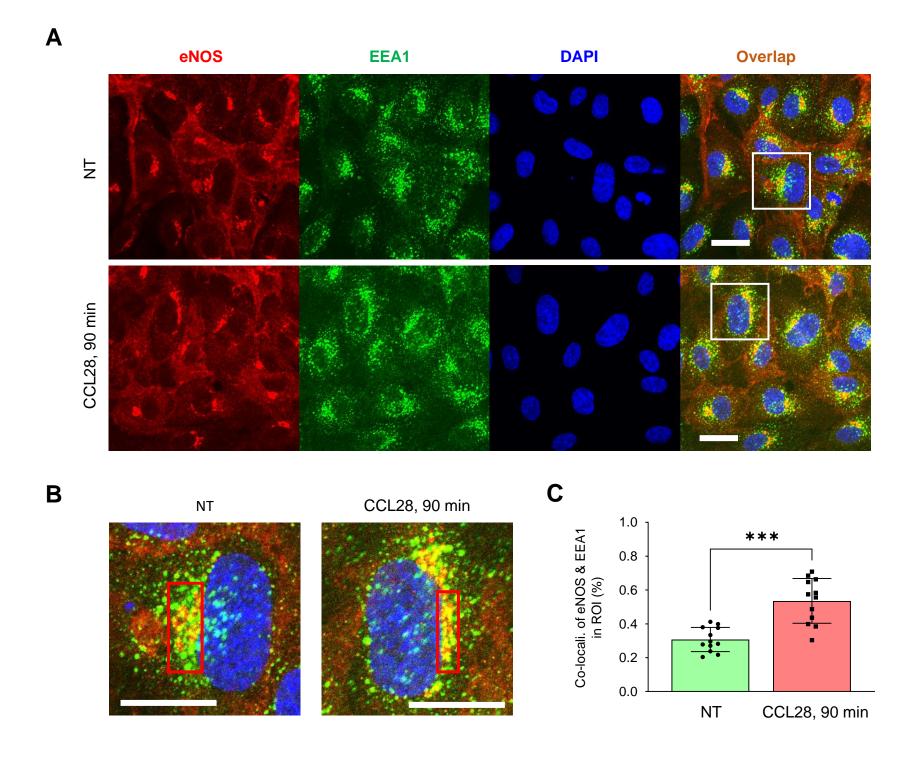
Supplemental Figure 1. Comparison of body weight, blood glucose level and VEGF level in dorsal skin of WT and diabetic db/db mice.

(A) Body weight of WT mice and db/db mice. Mice age: 9-12 weeks. (B) Higher blood glucose level in db/db mice (without fasting) by a blood glucose meter (Contour, Parsippany, NJ), compared to WT mice. (C) Reduced VEGF level in dorsal skin of db/db mice, compared to WT mice. Mouse dorsal was collected and prepared for ELISA measurement, as described in **RESEARCH DESIGN AND METHODS**. Data are presented as means \pm SD. ***P < 0.001.



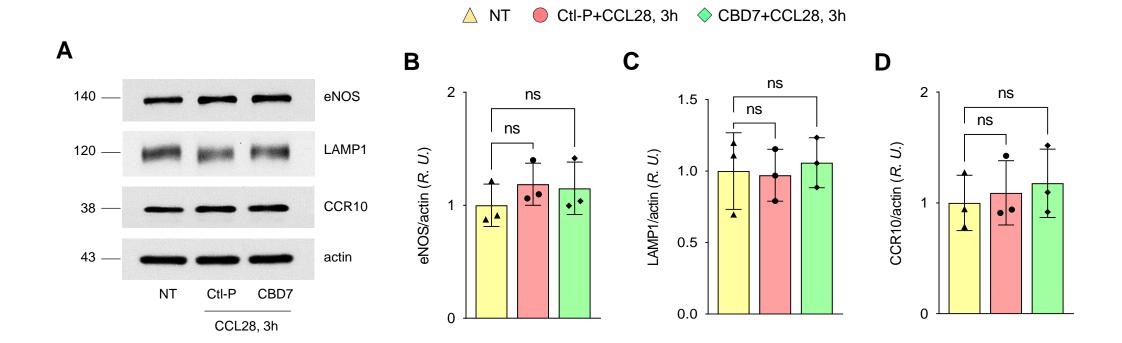
Supplemental Figure 2. Effect of overexpression of eNOS on CCR10 level in HEK/eNOS-GFP cells.

(**A**) After 48 hrs transfection of different doses of eNOS cDNA in stable HEK/CCR10-GFP cells, the cells were then collected and prepared for Western blot. Details can be seen in RESEARCH DESIGN AND METHODS. The blots were then probed for eNOS (top panel), CCR10 (middle panel) and loading control actin (bottom panel). (**B**) Normalized expression levels of eNOS and CCR10 vs. actin in (**A**). ns, not significant. Data are presented as means \pm SD. ns, not significant. *P < 0.05. R. U., relative unit. Note that CCR10 level does not change after different doses of eNOS cDNA transfection in HEK cells.



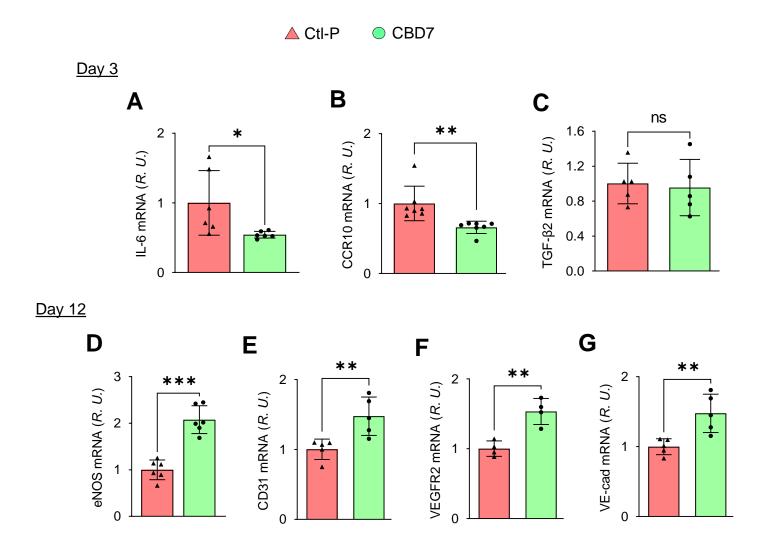
Supplemental Figure 3. eNOS translocates to early endosome antigen 1 (EEA1) in HDMVECs following CCL28 treatment.

(A) After 2 hrs serum-starvation, near-confluent HDMVECs were further not treated (NT) or treated with 500 ng/ml CCL28 for 90 min at 37°C. The cells were then fixed and prepared for confocal microscopy. Under NT (top panel), eNOS (red) was expressed mainly on cell-cell contacts, while showed less co-localization with EEA1 (green). After CCL28 treatment for 90 min (bottom panel), less eNOS were observed in cell-cell contacts, while showed more co-localization (yellow) with EEA1 near perinuclear regions. (B) Enlarged image of cells in the white boxes in A were indicated. (C) Co-localization percentage of EEA1 and eNOS in the region of interest (ROI; red box) was obtained according to software of the Zeiss 880 Confocal microscope. DAPI (blue), nuclear marker. Bar, 10 μm. Similar results were repeated in at least 4 independent experiments. Data are presented as means ± SD. ***P < 0.001. R. U., relative unit Note that co-localization of eNOS and EEA1 was greatly enhanced in ECs after CCL28 treatment for 90 min, compared to NT.



Supplemental Figure 4. Pre-IP protein levels in HDMVECs in the same conditions as Figure 5C.

(A) After serum-starvation and 1 hr pretreatment with serum-free medium alone, with 50 μ M Myr- control peptide (Ctl-P) or CBD7 peptide, confluent HDMVECs were further not treated (NT) or treated with 500 ng/ml CCL28 for 3 hrs min at 37°C. The cells were collected and prepared for Western blotting (10 μ g of cell lysates per condition). The blots were then probed for eNOS, LAMP1, CCR10 and actin. (**B-D**) Normalized values of eNOS (**B**), LAMP1 (**C**) and CCR10 (**D**). The ratio between protein versus actin under NT was set as 1. Note that all the protein levels did not show any significant change under indicated conditions. Data are presented as means \pm SD. ns, not significant. *R. U.*, relative unit.



Supplemental Figure 5. mRNA levels of genes in db/db mouse wounds following treatment with Myr-CBD7 peptide.

Mouse dorsal wounds were collected at indicated times and prepared for real-time RT-PCR. Reduced mRNA levels of IL-6 (**A**), CCR10 (**B**), while no change for TGF- β 2 (**C**) in dorsal wounds in *db/db* mice after 3 days of treatment with 50 μ M Myr-CBD7, compared with control peptide (Ctl-P). ns, not significant. Increased mRNA levels of EC markers eNOS (**D**), CD31 (**E**), VEGFR2 (**F**; n=4) and VE-cadherin (**G**) decreased TGF- β 1 (**F**) were obtained, respectively, by real-time RT-PCR, in db/db mouse wounds on day 12 following treatment with Myr-CBD7, compared with control peptide. Data are presented as means \pm SD. ns, not significant, *P < 0.05, **P < 0.01, ***P < 0.001. R. U., relative unit.