SUPPLEMENTARY DATA

Supplementary Table 1. Plasma blood glucose, triglycerides, total cholesterol, HDL and LDL cholesterol levels in hindlimb ischemia study.

	Non-Diabetic Wildtype		Diabetic Wildtype		Diabetic SR-BI$^{-/-}$	
	PBS	**rHDL**	**PBS**	**rHDL**	**PBS**	**rHDL**
Blood Glucose (mM)	12.28±0.50	12.35±0.74	27.40±0.85*	27.54±1.44*	29.29±0.84*	30.49±1.06*
Triglycerides (mg/mL)	0.55±0.06	0.55±0.05	1.11±0.15*	1.31±0.41*	1.87±0.43*	2.81±0.39*,$^{\#}$
Total Cholesterol (mg/mL)	1.38±0.08	1.37±0.08	2.25±0.18	2.52±0.18	4.36±0.58$^{\#}$	4.71±0.60$^{\#}$
HDL Cholesterol (mg/mL)	1.49±0.06	1.48±0.05	1.68±0.07	1.76±0.14	2.13±0.15	2.21±0.14
LDL Cholesterol (mg/mL)	0.10±0.01	0.24±0.04	0.57±0.19	0.86±0.31	2.23±0.43$^{\#}$	2.50±0.51$^{\#}$

*$p<0.05$ vs. Non-Diabetic Wildtype animals.
$^{\#}p<0.05$ relative to Wildtype Diabetic animals.

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SUPPLEMENTARY DATA

Supplementary Table 2. Plasma blood glucose, triglycerides, total cholesterol, HDL and LDL cholesterol levels in wound healing study.

	Non-Diabetic	Diabetic	
	Wildtype	**Wildtype**	**SR-BI$^{-/-}$**
Blood Glucose (mM)	12.76±0.76	28.46±1.99*	28.31±1.81*
Triglycerides (mg/mL)	0.41±0.03	1.47±0.36	2.02±0.54
Total Cholesterol (mg/mL)	1.35±0.05	3.76±0.68*	4.54±0.26*
HDL Cholesterol (mg/mL)	1.23±0.02	2.07±0.21*	2.40±0.10*
LDL Cholesterol (mg/mL)	0.18±0.07	1.59±0.95	2.14±0.26

*$p<0.05$ vs. Non-Diabetic Wildtype animals.

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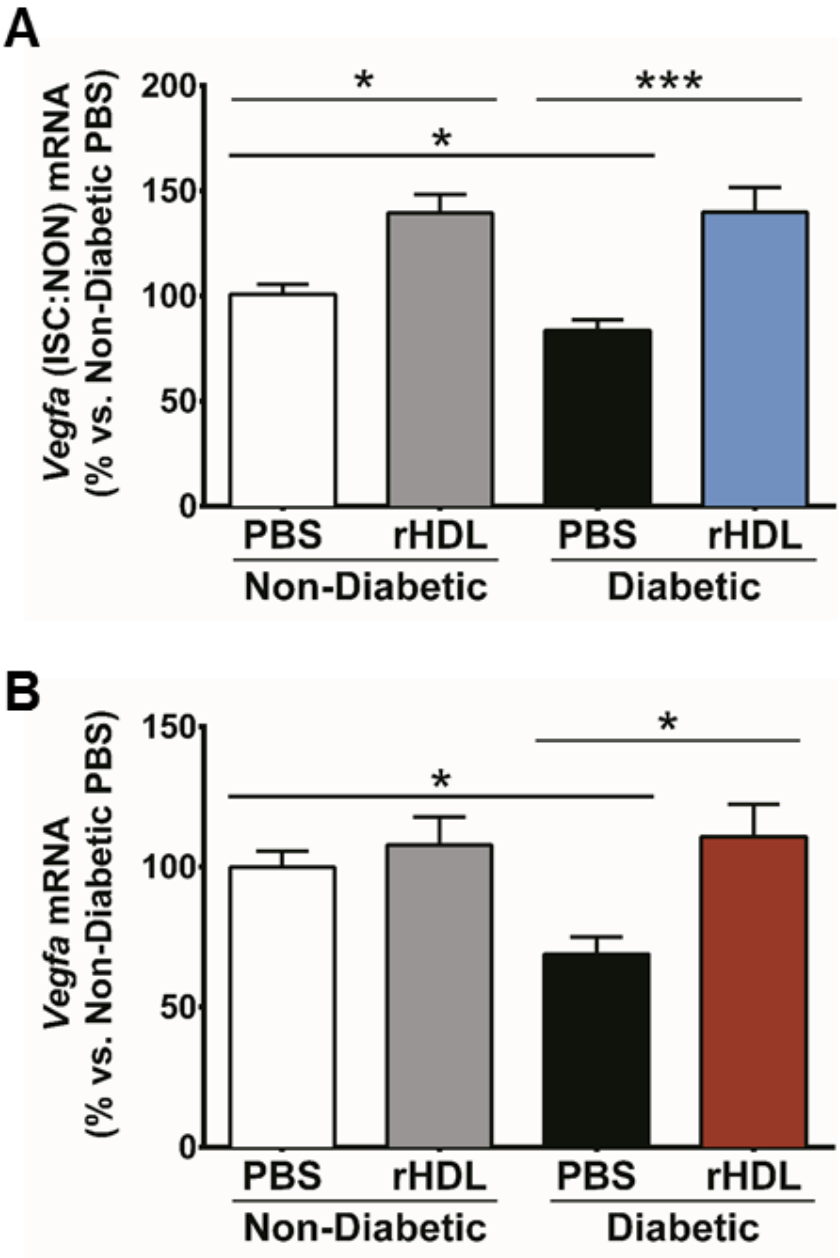
SUPPLEMENTARY DATA

Supplementary Figure 1. rHDL rescues diabetes-induced suppression of VEGF expression *in vivo*. (A) Femoral artery ligation was done on non-diabetic and diabetic C57B1/6J mice (n=9-10/group). Mice received *i.v.* injections of rHDL (200 μg/mouse) or PBS (vehicle) on alternate days following ligation until sacrifice. *Vegfa* mRNA levels, expressed as a ratio of ischemic (ISC):non-ischemic (NON) hindlimb, normalized to *36B4*. (B) Two full thickness wounds were created on non-diabetic and diabetic C57B1/6J mice (n=11/group). Mice received daily topical applications of rHDL (50 μg/wound) or PBS. *Vegfa* mRNA levels, normalized to *36B4*. Results are expressed as mean±SEM. *p<0.05, ***p<0.001.



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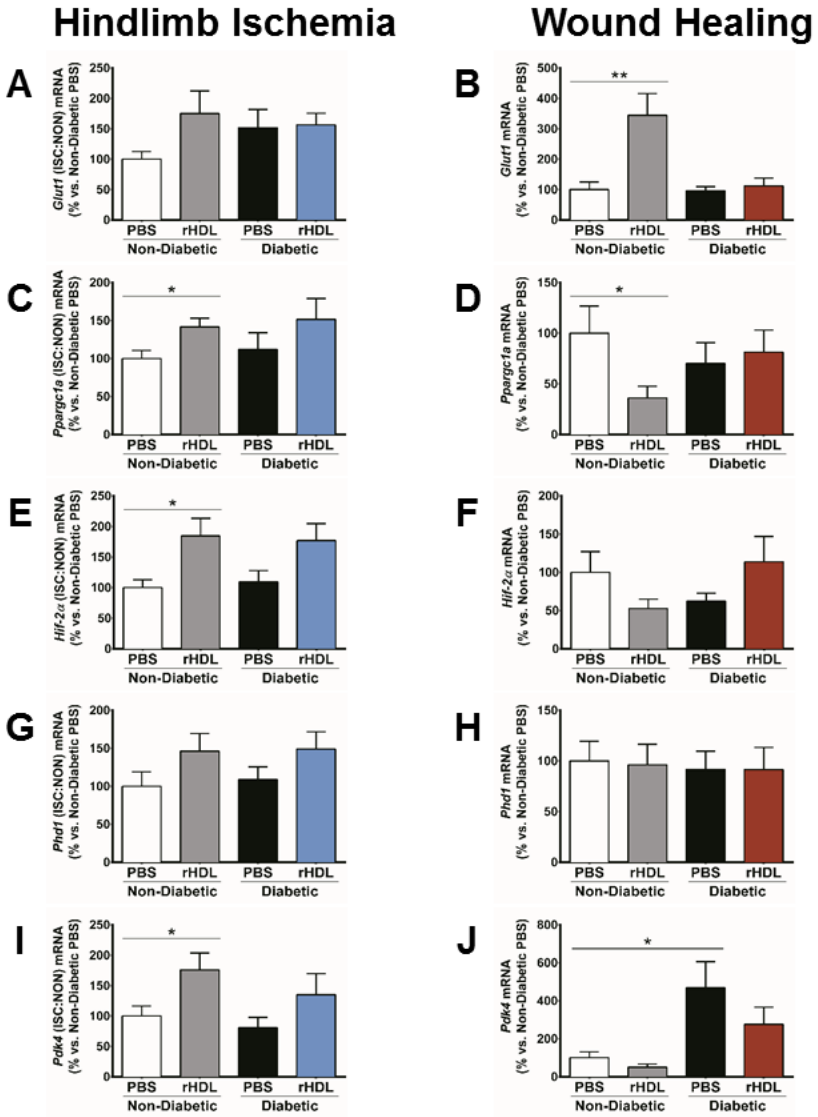
SUPPLEMENTARY DATA

Supplementary Figure 2. rHDL has no effect on markers of glucose metabolism in diabetic animals.

Hindlimb ischemia: Femoral artery ligation was done on non-diabetic and diabetic C57B1/6J mice (n=9-10/group). Mice received *i.v.* injections of rHDL (200 μg/mouse) or PBS (vehicle) on alternate days following ligation until sacrifice. Wound healing: Two full thickness wounds were created on non-diabetic and diabetic C57B1/6J mice (n= 11/group). Mice received daily topical applications of rHDL (50 μg/wound) or PBS (vehicle). (A & B) *Glutl*, (C & D) *Ppargcla*, (E & F) *Hif-2*α_2 (G & H) *Phdl* and (I & J) *Pdk4* mRNA levels, normalized to *36B4*. For the hindlimb tissues, genes are expressed as a ratio of ischemic (ISC)non-ischemic (NON) hindlimb. Results are expressed as mean±SEM. *p<0.05. **p<0.01.



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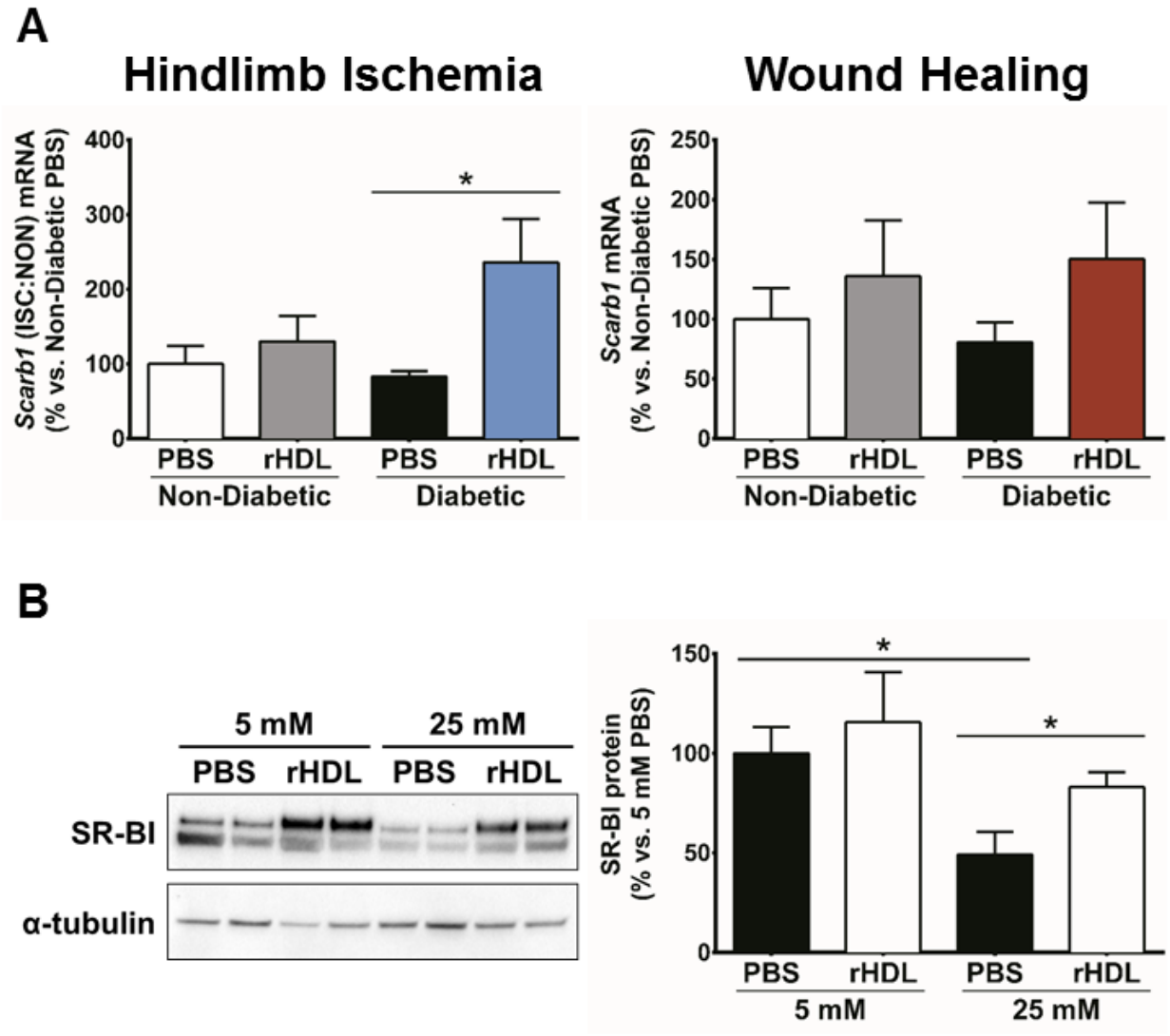
SUPPLEMENTARY DATA

Supplementary Figure 3. rHDL augments SR-BI expression. (A) Hindlimb ischemia: Femoral artery ligation was done on non-diabetic and diabetic C57B1/6J mice (n=9-10/group). Mice received *i.v.* injections of rHDL (200 μg/mouse) or PBS (vehicle) on alternate days following ligation until sacrifice. Wound healing: Two full thickness wounds were created on non-diabetic and diabetic C57B1/6J mice (n=11/group). Mice received daily topical applications of rHDL (50 μg/wound) or PBS. *Scarbl* mRNA levels, normalized to *36B4*. For the hindlimb tissues, genes are expressed as a ratio of ischemic (ISC):non-ischemic (NON) hindlimb. (B) HCAECs were treated with rHDL (20 μM, white bars) or PBS (vehicle, black bars) for 18 h prior to 48 h glucose exposure (5-25 m.M) then utilized to measure SR-BI protein levels. Even protein loading confirmed with α-tubulin. Results are expressed as mean±SEM. *p<0.05.



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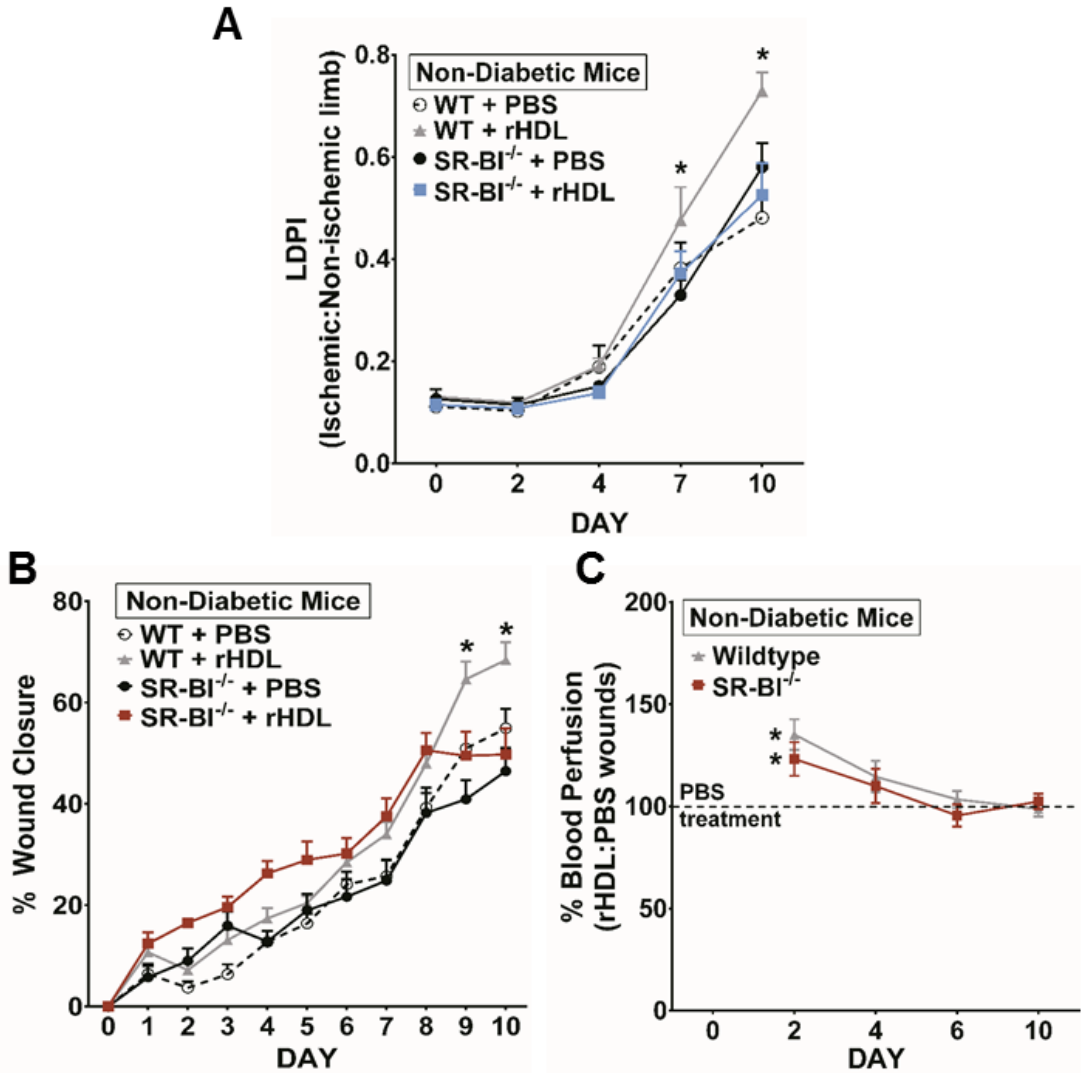
SUPPLEMENTARY DATA

Supplementary Figure 4. The role of SR-BI in rHDL-induced augmentation of ischemia-driven angiogenesis and wound healing. (A) Femoral artery ligation was performed on non-diabetic wildtype (WT) and SR-BI$^{-/-}$ littermates (n=9-10/group). Mice received *i.v.* injections of rHDL (200 μg/mouse) or PBS (vehicle) on alternate days following ligation. Laser Doppler Perfusion Index (LDPI) was determined based on the ratio of ischemic:non-ischemic hindlimb. White circles, non-diabetic WT PBS-infused mice: grey triangles, non-diabetic WT rHDL-infused mice; black circles, non-diabetic SR-BI$^{-/-}$ PBS-infused mice: and blue squares, non-diabetic SR-BI$^{-/-}$ rHDL-infused mice. (B) Two full thickness wounds were created on WT and SR-BI$^{-/-}$ littermates (n=11/group). Mice received daily topical applications of rHDL (50 μg/wound) or PBS. Wound area was calculated from the average of three daily diameter measurements along the x, y and z-axes. Wound closure is expressed as a percentage of initial wound area at Day 0. White circles, non-diabetic WT PBS-treated wound: grey triangles, non-diabetic WT rHDL-treated wound: black circles, non-diabetic SR-BI$^{-/-}$ PBS-treated wound; and red squares, nondiabetic SR-BI$^{-/-}$ rHDL-treated wound. (C) rHDL:PBS wound blood flow perfusion ratio was determined using laser Doppler imaging in non-diabetic wildtype (grey triangles) and SR-BI$^{-/-}$ (red squares) mice. Results are expressed as mean±SEM. *p<0.05, vs. respective PBS control mice.



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