Supplemental table 1
Subject characteristics (n=402)

Age (yr)	61 ± 8.8
Sex, men/women	193/209
BMI (kg/m^2)	23 ± 2.8
BP, systolic (mmHg)	131 ± 21
BP, diastolic (mmHg)	78 ± 12
Smoking, yes/no	133/269
LDL-C (mg/dl)	149 ± 28
HDL-C (mg/dl)	55 ± 15
TG (mg/dl)	127 ± 76
LDL size (nm)	26 ± 1.0
MDA-LDL (IU/l)	115 ± 38
Glucose (mg/dl)	96 ± 9.0
Insulin (μU/ml)	7.3 ± 6.2
sLR11 (U)	3.0 ± 1.0
IMT (mm)	0.79 ± 0.2

BP;Blood pressure, LDL-C;LDL-cholesterol, HDL-C;HDL-cholesterol, TG:Triglycerides, MDA-LDL;malondialdehyde LDL.

Supplemental table 2
Univariate analysis of the association of IMT with atherosclerotic risk factors

	r or Mean±SD	P values
Age (yr)	0.41	<0.001
Sex, men/women	0.84±0.18/0.75±0.19	0.001
BMI (kg/m^2)	0.03	0.68
BP, systolic (mmHg)	0.29	<0.001
BP, diastolic (mmHg)	0.28	<0.001
Smoking, yes/no	0.84±0.19/0.79±0.19	0.02
LDL-C(mg/dl)	0.04	0.58
HDL-C (mg/dl)	-0.31	<0.001
TG (mg/dl)	0.19	0.01
LDL size (nm)	-0.21	0.002
MDA-LDL (IU/l)	0.09	0.23
Glucose (mg/dl)	0.10	0.18
Insulin (μU/ml)	0.15	0.04
sLR11 (U)	0.48	<0.001

LDL-C;LDL-cholesterol, HDL-C;HDL-cholesterol, TG:Triglycerides, MDA-LDL;malondialdehyde LDL.

Supplemental table 3
Univariate analysis of the association of sLR11 with atherosclerotic risk factors or IMT

	Men		Women	
	r or Mean±SD	P values	r or Mean±SD	P values
Age (yr)	0.18	0.07	0.11	0.28
BMI (kg/m^2)	0.19	0.06	-0.33	0.74
BP, systolic (mmHg)	0.37	<0.001	0.05	0.60
BP, diastolic (mmHg)	0.35	<0.001	-0.02	0.86
Smoking, yes/no	3.09±0.93 /3.09±1.09	0.99	2.84±1.21 /2.93±0.96	0.78
LDL-C(mg/dl)	0.01	0.94	0.21	0.03
HDL-C (mg/dl)	-0.27	0.008	-0.31	<0.001
TG (mg/dl)	0.02	0.89	0.19	0.05
LDL size (nm)	0.13	0.22	0.23	0.02
MDA-LDL (IU/l)	0.14	0.18	0.16	0.11
Glucose (mg/dl)	0.17	0.10	-0.09	0.35
Insulin (μU/ml)	0.06	0.57	0.21	0.03
IMT (mm)	0.47	<0.001	0.49	<0.001

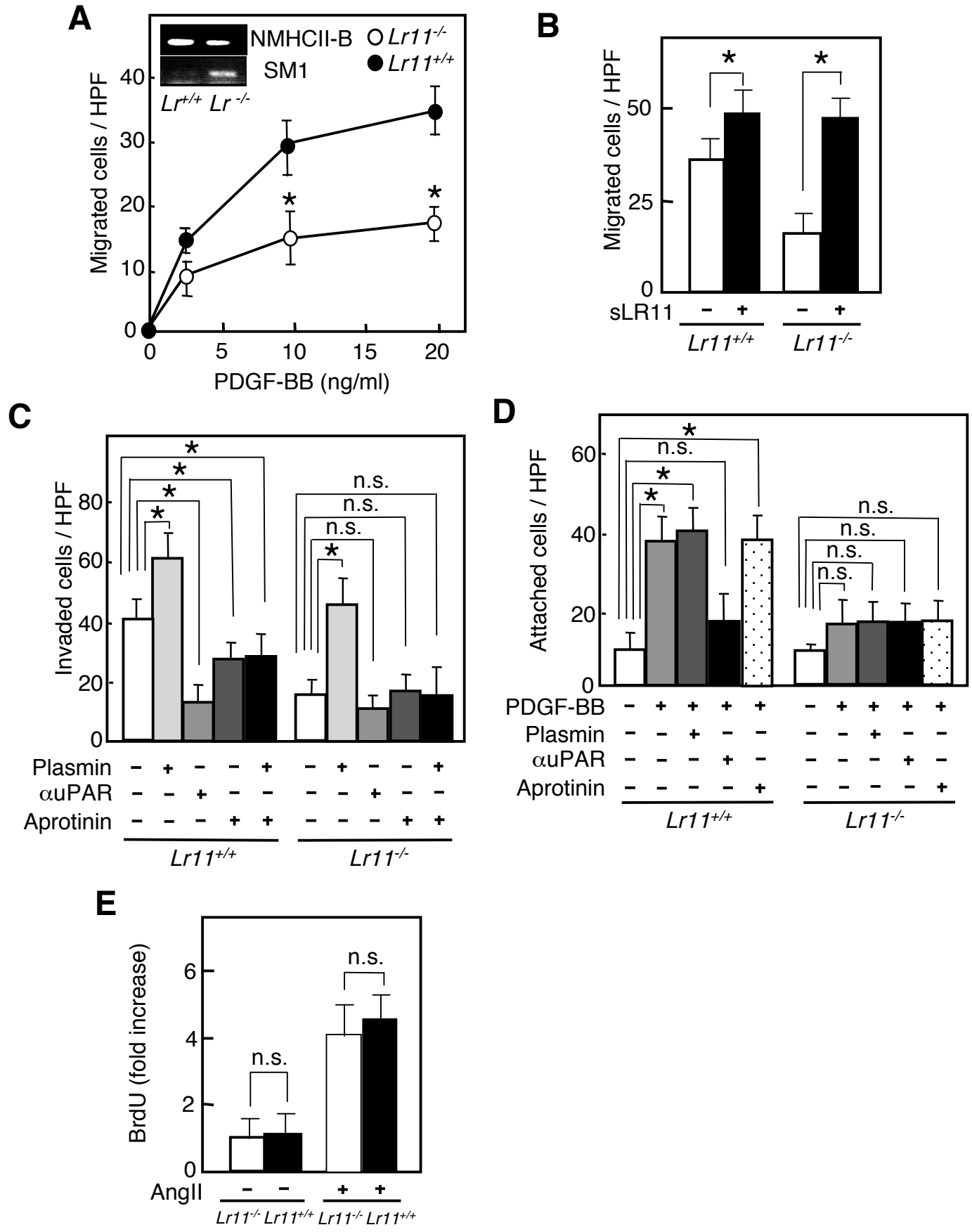
LDL-C;LDL-cholesterol, HDL-C;HDL-cholesterol, TG:Triglycerides, MDA-LDL;malondialdehyde LDL.

Supplemental table 4
Multivariate assessment of the effect of atherosclerotic risk factors or IMT on sLR11

	OR (95%CI)	P values
Men		
BP, per 10mmHg increase		
systolic	1.03 (0.70-1.51)	0.88
diastolic	1.20 (0.59-2.45)	0.62
HDL-C, per 10mg/dl decrease	0.99 (0.96-1.03)	0.65
IMT, per 0.1mm increase	1.59 (1.20-2.12)	0.001
Women		
TG, per 10mg/dl increase	1.01 (0.91-1.13)	0.81
LDL size	0.57 (0.29-1.14)	0.11
Insulin	1.06 (0.94-1.19)	0.34
LDL-C, per 10mg/dl increase	1.08 (0.83-1.41)	0.57
HDL-C, per 10mg/dl decrease	1.27 (0.80-2.02)	0.31
IMT, per 0.1mm increase	1.47 (1.13-1.92)	0.005

LDL-C;LDL-cholesterol, HDL-C;HDL-cholesterol, TG:Triglycerides, OR, odds ratio.

Supplemental figure 1, Jiang, et al.



Supplemental figure 1, Jiang, et al.

Figure legend

(A) Migration activity of *Lr11*$^{+/+}$ and *Lr11*$^{-/-}$ SMCs in the presence of various concentrations of PDGF-BB were analyzed using a Boyden chamber. The products of NMHCII-B and SM1 amplified using RT-PCR in *Lr11*$^{+/+}$ and *Lr11*$^{-/-}$ SMCs, respectively, are shown in the inset. Data are presented as mean±SD (n = 6). *P<0.05. The *Lr11*$^{-/-}$ SMCs showed decreased levels of NMHCII-B, but increased levels of SM1 expression compared to wild-type SMCs. *Lr11*$^{-/-}$ SMCs showed less PDGF-BB–induced migration than *Lr11*$^{+/+}$ SMCs, as the number of migrated *Lr11*$^{-/-}$ SMCs at 20 ng/ml PDGF-BB was 51% of that of *Lr11*$^{+/+}$ SMCs. (B) Effect of sLR11 on the PDGF-BB (10 ng/ml) -induced migration activity of *Lr11*$^{+/+}$ or *Lr11*$^{-/-}$ SMCs. SMCs were incubated with recombinant sLR11 (1 μg/ml) for 24 h before migration analyses. Data are presented as mean±SD (n = 6). *P<0.05. In the presence of recombinant sLR11, the decreased PDGF-mediated migration activity of *Lr11*$^{-/-}$ SMCs was restored almost to that of *Lr11*$^{+/+}$ SMCs. (C) Effect of blocking the uPAR/uPA/plasmin system on the PDGF-BB (10 ng/ml) -induced invasion activity of *Lr11*$^{+/+}$ or *Lr11*$^{-/-}$ SMCs. SMCs were incubated with plasmin (10 nM), anti-uPAR antibody (10 μg/ml), or aprotinin (100 KIU/ml) for 24 h before invasion analyses. Data are presented as mean±SD (n = 6). *P<0.05. n.s., not significant. Collagen gel invasion was significantly decreased in *Lr11*$^{-/-}$ SMCs compared to *Lr11*$^{+/+}$ SMCs; although plasmin increased the PDGF-induced invasion activity of *Lr11*$^{-/-}$ SMCs, the resulting activity level in *Lr11*$^{-/-}$ SMCs was still only 71 % of that of *Lr11*$^{+/+}$ SMCs. Neutralization of uPAR using a blocking antibody or aprotinin, a plasmin inhibitor, did not significantly reduce the invasion activity of *Lr11*$^{-/-}$ cells, and the invasion with plasmin plus aprotinin was similar to that with aprotinin alone in both cell types. (D) Effects of blocking the uPAR/uPA/plasmin system on the PDGF-BB (10 ng/ml) -induced attachment of *Lr11*$^{+/+}$ or *Lr11*$^{-/-}$ SMCs. SMCs were incubated with plasmin (10 nM), anti-uPAR antibody (10 μg/ml), or aprotinin (100 KIU/ml) for 24 h before attachment analyses. Data are presented as mean±SD (n = 6). *P<0.05. n.s., not significant. The attachment of *Lr11*$^{-/-}$ SMCs in the presence of PDGF-BB was reduced, and the activity was not recovered by addition of plasmin. (E) Proliferation activity of *Lr11*$^{+/+}$ and *Lr11*$^{-/-}$ SMCs in the presence or absence of 1μM AngII for 8 h before BrdU incorporation analyses. Data are presented as mean±SD (n = 3). n.s., not significant. There were no significant difference in BrdU incorporation between *Lr11*$^{+/+}$ and *Lr11*$^{-/-}$ SMCs in the presence or absence of recombinant AngII.

Supplemental figure 2, Jiang, et al.

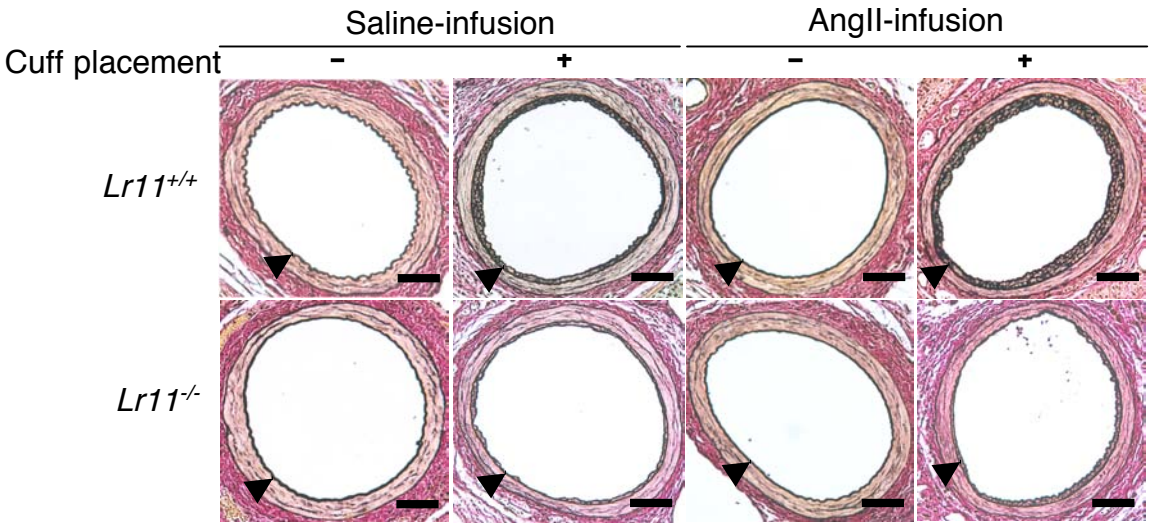


Figure legend

EVG-stained sections of femoral arteries with or without cuff placement in the presence of AngII or saline infusion in *Lr11*$^{+/+}$ or *Lr11*$^{-/-}$ mice. Arrowheads indicate the internal elastic layers. Scale bar, 50 μm. Systemic blood pressure values four weeks after cuff placement were significantly higher in the AngII-infused mice than those in the saline-infused mice (119±7 mmHg vs 106±7 mmHg for *Lr11*$^{+/+}$ mice, and 121±7 mmHg vs 110 ±6 mmHg for *Lr11*$^{-/-}$ mice, respectively, $P<0.05$, n=8 for each group).

Supplemental figure 3, Jiang, et al.

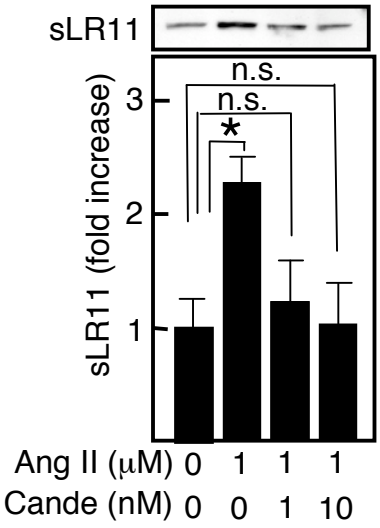


Figure legend

Effect of ARB on the AngII-dependent increase in sLR11 in rabbit SMCs. Conditioned media collected for 12 h in the presence or absence of AngII with/without candesartan (cande) at the indicated concentrations were subjected to immunoblot analysis using anti-LR11 (~250kDa) antibody. Photograph shown is representative of 3 independent experiments. Data ware presented as mean±SD (n = 3). *P<0.05, n.s., not significant. The AngII–mediated increase in sLR11 levels was blocked by ARB candesartan.