SUPPLEMENTAL FIGURE LEGENDS

FIGURE S1. Differential expression of *miR-29c* in control db/m vs. diabetic db/db mice. Northern blot analysis shows representative results of *miR-29c* expression in several tissues from control db/m and diabetic db/db mice. U6 snRNA serves as the loading control.

FIGURE S2. Upregulation of *miR-29c* expression in streptozotocin (STZ)-induced mouse model of **DN**. Northern blot analysis shows representative results from kidney *miR-29c* expression in controls and STZ-induced diabetic mice (n=3). U6 snRNA serves as a loading control.

FIGURE S3. **Upregulation of** *miR-29c* **expression in kidney microvascular endothelial cells.** Real-time qPCR analysis shows *miR-29c* expression in high-glucose (HG)-treated (24 hrs) cultured kidney microvascular endothelial cells as compared to normal glucose (NG) conditions. Measured transcript levels were normalized to U6 snRNA expression. Samples were run in triplicate. Data are shown as mean±SE.

FIGURE S4. **Downregulation of Spry1 expression by miR-29c overexpression.** Lenti-CAG and LentimiR-29c-infected podocytes were treated with normal glucose (NG) or high glucose (HG) for 36 hrs. Spry1 expression in total cell lysates was analyzed by Western blot. GAPDH serves as a loading control.

FIGURE S5. Effect of miR-29c on podocyte apoptosis. Apoptotic cells were labeled with annexin V-FITC and Sytox Red and analyzed by FACS. Representative data are shown from three independent experiments. The percentages of early apoptotic cells (annexin-positive and Sytox-negative) are shown which are also presented as graphs in Figures 5*A* and 5*C*. *A*. Lenti-CAG and Lenti-miR-29c podocytes were transfected with miR-29c inhibitor. *B*, Control podocytes were transfected with Spry1 siRNA (siSpry1, 30 nM) and/or miR-29c inhibitor as indicated, and treated with HG (25mM) for 36hrs.

FIGURE S6. **Knockdown of Spry1 expression by Spry1 siRNA.** Cultured podocytes were transfected with Spry1 siRNA by Lipofectamine 2000. Knockdown efficiency of Spry1 at the protein level was verified by Western blot. GAPDH serves as the loading control.

FIGURE S7. Knockdown of miR-29c in different tissues of *db/db* mice with miR-29c antisense oligonucleotides (miR-29c ASO). Real-time qPCR analysis shows *miR-29c* expression in the indicated tissues following treatment with miR-29c ASO. NS: non significant.



Supplemental Figure-1



Supplemental Figure-2



Supplemental Figure-3



Supplemental Figure-4





Supplemental Figure-5

B



Supplemental Figure-6



Supplemental Figure-7

	mouse kidney microvascular endothelial cells				mouse cultured podocytes			
miRNA ID	NG	HG (6hrs)	HG (12hrs)	HG (24hrs)	NG	HG (6hrs)	HG (12hrs)	HG (24hrs)
mmu-miR-192	90	112	123	190	133	217	120	492
mmu-miR-29c	67	185	1,092	693	292	369	421	417
mmu-miR-26b	1,132	1,113	1,633	1,914	4,826	5,116	6,849	8,336
mmu-miR-1196	162	244	223	411	282	485	540	793
mmu-miR-1195	286	328	811	741	580	842	805	750
mmu-miR-200b	95	158	280	503	1,871	2,698	4,367	3,756
mmu-let-7g	2,162	2,213	3,299	3,269	935	1,090	1,797	2,071
mmu-miR-200a	212	121	450	547	322	807	1,057	990
mmu-miR-705	243	160	342	336	11,928	7,127	16,002	20,979

Supplementary TABLE 1. **Temporal expression profiles of differentially expressed miRNAs in high glucose-treated kidney microvascular endothelial cells and cultured podocytes.** The relative expression of the upregulated miRNAs at indicated time points are shown. All data are expressed as the mean signal intensity. NG: normal glucose; HG: high glucose.