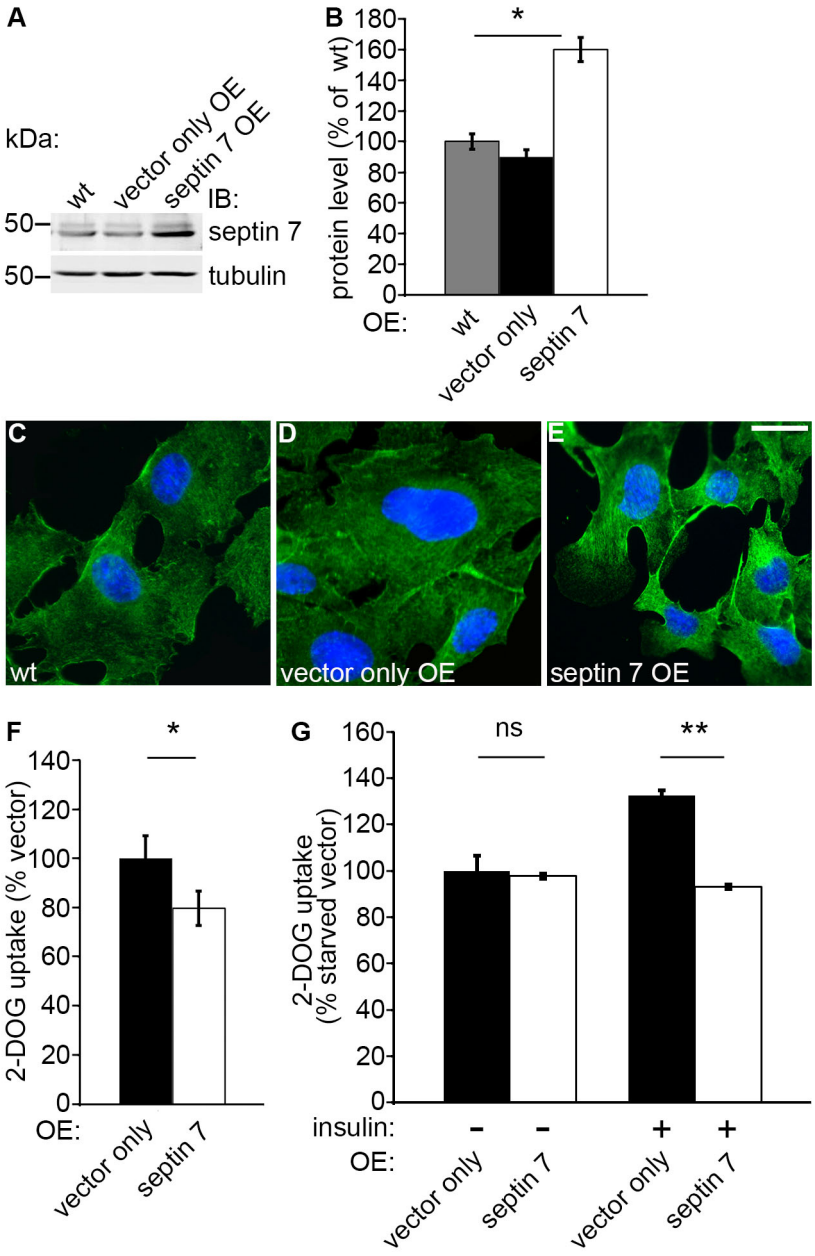

Wasik *et al*. Supplementary Figure S1. Septin 7 overexpression decreases basal and insulin-stimulated glucose uptake in mouse podocytes. (A) Mouse podocytes were transfected with pcDNA 3.1 vector (vector only OE) or pcDNA 3.1- septin 7 vector (septin 7 OE). Immunoblotting confirms septin 7 overexpression. Tubulin is included as a loading control. (B) Quantification of the septin 7 level in (a) indicates that septin 7 level is increased by 60% in septin 7 overexpressing podocytes compared to vector only -transfected cells. (C to E) Immunofluorescence images of mouse podocytes stained for septin 7 show increased expression of septin 7 in septin-7 overexpressing podocytes (E) compared to the wild-type (wt) (C) and control vector transfected podocytes (vector only OE) (D). (F) Overexpression of septin 7 decreases the glucose uptake activity of mouse podocytes by 20% compared to the control siRNA transfected cells (set to 100%) under basal

conditions. (G) Overexpression of septin 7 does not change the glucose uptake activity of serum starved mouse podocytes (septin 7 OE, - insulin). Glucose uptake activity of the control and serum starved cells is set to 100% (vector only, - insulin). After insulin stimulation, the increase in glucose uptake is 33% in control podocytes (vector only, + insulin). In septin 7 overexpressing podocytes insulin does not increase the glucose uptake activity (septin 7 OE, + insulin) compared to the control, serum starved cells (vector only, - insulin). In A, 50 µg of lysates from wild-type (wt), control (vector only OE) and septin 7 overexpressing (septin 7 OE) podocytes were separated by SDS-PAGE and immunoblotted with anti-septin 7 and anti-tubulin IgGs. In C to E, cells were fixed with 2% PFA, labeled with anti-septin 7 IgG, and examined by fluorescence microscopy. In F to G mouse podocytes were transfected with pcDNA 3.1 (vector only OE) or pcDNA 3.1-septin 7 vector (septin 7 OE). Glucose uptake was measured as described in Materials and Methods in basal state (culture medium containing 10% FCS) (E) or after serum starvation (-) and treatment with 20 nM insulin (+) (F). Scale bar, 20 µm. Bars show the mean and error bars STDEV of three independent experiments, Student's t-test. * $p<0.05$, ** $p<0.01$

	normoalbuminuric (*n* = 4)	**macroalbuminuric (*n* = 5)**	**P**
Male/female	4/0	5/0	NS
Age (years)	35.0 ± 2.2	46.9 ± 5.1	< 0.01
Diabetes duration (years)	12.2 ± 2.1	33.4 ± 5.9	< 0.001
$HbA1_c$ (%)	7.7 ± 0.5	7.8 ± 1.1	NS
Triglycerides (mmol/l)	1.3 ± 0.4	1.4 ± 0.9	NS
HDL cholesterol (mmol/l)	1.5 ± 1.2	1.4 ± 0.3	NS
LDL cholesterol (mmol/l)	2.1 ± 1.1	1.8 ± 0.7	NS

Wasik *et al*. Supplementary Table S1. Characteristics of the normoalbuminuric and macroalbuminuric patients with type 1 diabetes. The patients were participants of the Finnish Diabetic Nephropathy Study (FinnDiane). The study protocol was approved by the local Ethics Committee (Helsinki University Central Hospital), and all participants gave an informed consent to participate in the study. Urinary albumin excretion rate (AER) was defined as normal (<30 mg/24 h), microalbuminuria (≥30, <300 mg/24 h), and macroalbuminuria (≥300 mg/24 h). Fasting glucose values were measured using a Hemocue device (Hemocue, Helsinki, Finland), and serum lipids were determined with a Konelab analyzer (Thermo Scientific, Vantaa, Finland). Other biochemical analyses were performed in an accredited hospital laboratory (HUSLAB, Helsinki, Finland). Data are presented as mean ± standard deviation. P values are from student's t-test and from $\chi 2$ test. NS, not significant.