Figure S1.

Albumin (mg/dl) normalized to creatinine (mg/dl)

control                   25 ng DT          200 ng DT

*  **  ***                   *  ***          *  ***

days after DT
Figure S2
Figure S3.
Supplemental Figures

**Figure S1. Normalized albumin loss in the urine.** Y-axis, urinary albumin concentration (mg/dl) normalized to creatinine (mg/dl). Mice on day 7 after 200ng DT displayed oliguric renal failure with a marked decrease in urine creatinine (14+/-4 mg/dl as compared to 56+/-12 mg/dl on day 5) and resultant increase in the urine albumin/creatinine ratio. *p<0.05; **p<0.01; ***p<0.001 (vs. control)

**Figure S2. Glomerulosclerosis, crescent formation and tubular injury in mice with >40% Podocyte Loss.**

A. 50ng DT is sufficient to trigger progressive glomerular damage. a, untreated control; b, 1 week after a single dose of 50ng injection; c, 4 weeks after a single dose of 50ng DT administration. d, e, and f are higher magnifications of the boxed glomeruli in a, b, and c, respectively, showing glomerulosclerosis (arrow).

B. Anti-αSMA immunostaining is only detected at the vascular pole (asterisk) in the control glomerulus and DTR staining is restricted to normal podocytes (arrow, left panel), while wide spread αSMA signal is present in the fibrocellular crescent 4 weeks after 100 ng DT treatment. Note αSMA+/DTR+ cells are found in the crescent outside of the glomerular tuft (arrow), indicating that podocytes contributed to crescent formation.

**Figure S3.** RTK array identified two receptor kinases that were differentially phosphorylated after DT treatment (pPDGFRα at short exposure and pAxl at longer
exposure). 200ug microsomal protein was loaded to each filter, two controls and two treated samples were processed side by side. Only one pair of the array is shown.