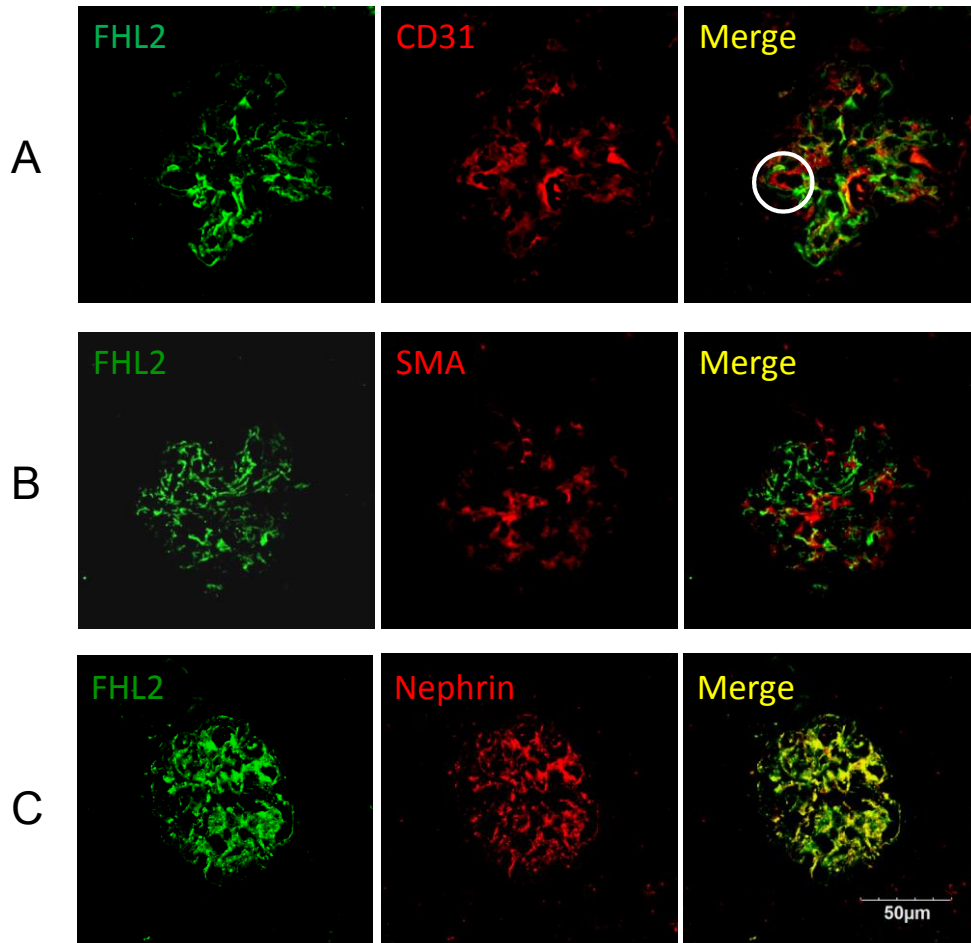


## Supplement data

**Supplement Figure 1.** Immunofluorescence double staining of FHL2 and glomerular cell markers in mouse kidney.



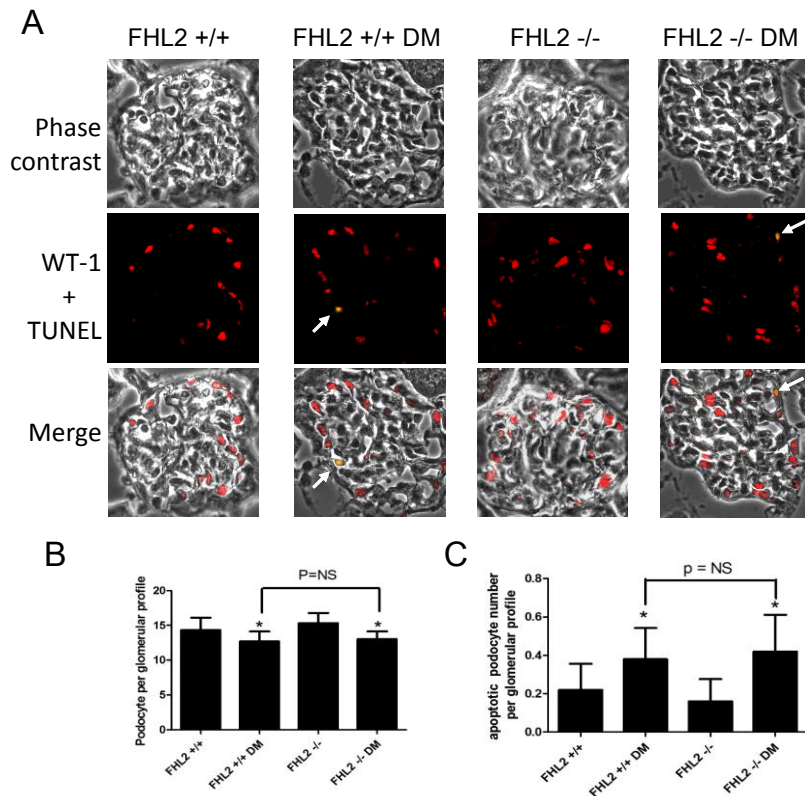
Double staining of FHL2 and three different glomerular cell markers.

Panel A: Both FHL2 and CD31 are stained in granular loop pattern, but they are not co-stained together (note the CD31 locates in the inner loop and FHL2 in the outer loop).

Panel B: Smooth muscle actin (SMA) is stained in typical “pruned shrub” mesangial cell pattern, it is not co-stained with FHL2.

Panel C: FHL2 is well co-stained with podocyte marker nephrin.

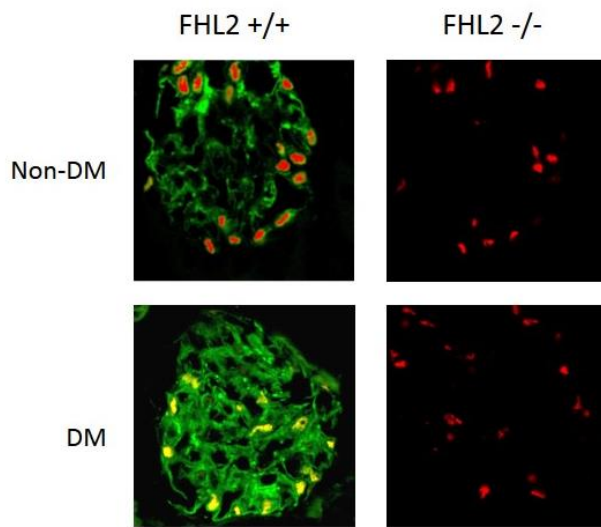
**Supplement Figure 2.** Diabetic FHL2<sup>-/-</sup> and FHL2<sup>+/+</sup> mice had comparable podocyte number and podocyte apoptosis rate.



Panel A: Representative pictures of podocyte number and apoptotic podocyte per glomeruli section. For each mice, 15 glomeruli were randomly selected and podocyte number were calculated. DNA strand breaks in podocytes were identified using the in situ nick/end-labeling assay (TUNEL). Panel B: quantified podocyt numbers per glomerulus of four groups. Panel C: Absolute podocyte apoptosis number is modest but significant increase in mouse diabetes kidney disease model. The two diabetic groups had comparable podocyte apoptosis rate.

\*P<0.05 compare to non-diabetic FHL2<sup>+/+</sup> mice. N= 10 each group.

**Supplement figure 3.** FHL2 nuclear translocation in diabetic kidney disease in mice.



Kidney sections of mice were stained with anti-FHL2 and anti-WT1. In non-DM mice, FHL2 expressed in podocyte cytoplasm (green), WT-1 (red) demarcates podocyte cell nucleus. Induction of diabetes increases FHL2 expression and triggers its nuclear translocation *in-vivo*. As expected, FHL2 is not detected in FHL2  $-/-$  mice.