Supplementary Figure Legend

Supplementary Figure 1. Assessment of kallistatin protein levels in non-diabetic, Type 1 and Type 2 diabetic human corneas. (A) Representative images of Western blotting of kallistatin in the corneas were collected from non-diabetic (NDM), Type 1 DM (T1DM), and Type 2 DM (T2DM) donors. (B) Densitometry analysis of kallistatin in the cornea and normalized by GAPDH levels (mean±SEM). NDM (n=2), T1DM (n=3), and T2DM (n=3).

Supplementary Figure 2. Schematics showing that kallistatin inhibits Wnt signaling and delays wound healing in diabetic cornea. Transgenic expression of kallistatin suppressed Wnt signaling and delayed corneal wound healing. Local inhibition of Wnt signaling by kallistatin protein, an LRP6-blocking antibody (2F1), or soluble VLDLR ectodomain (VLN) delayed corneal wound healing. In contrast, ablation of VLDLR resulted in the overactivation of Wnt/β-catenin signaling and accelerated corneal wound healing. Local activation of Wnt signaling in the cornea by lithium chloride (LiCl) or the constitutively active mutant of β-catenin (S37A) accelerated wound healing. APC: *adenomatous polyposis coli*; CKIα: casein kinase Iα; Dvl: Dishevelled; EGFR: epidermal growth factor receptor; Fzd: frizzled receptor; GSK-3β: glycogen synthase kinase 3 beta; LRP5/6: low-density lipoprotein receptor-related protein 5 or 6; TCF/LEF: T-cell factor/lymphoid enhancer factor.