Expression of Upregulated SHH, SMO and GLI1 in the untreated rats and Primary Cultured Retinal Müller Cells

To investigate the effect of exogenous SHH on untreated rats and primary cultured retinal Müller Cells, we performed western blot analysis of retinal samples after two intravitreous injections of PBS, SHH (100μg/ml) or cyclopamine (5μg/ml) every two weeks, as well as primary retinal Müller Cells after treatment with SHH (3μg/ml) or cyclopamine (20μg/ml). Western blot analysis (Figures A–D) revealed that the expression of SHH, SMO and GLI1 in normal rats retinas were rare. Exogenous SHH increased protein expression of SHH, SMO and GLI1 and exogenous cyclopamine reversed that effect. Also, the exogenous SHH increased the expression of SHH, SMO and Gli1 in 5.5mM glucose cultured primary Müller Cells, cyclopamine inhibited the expression of SHH, SMO and GLI1 (Figures E–H).

Figure legends

Expression of Upregulated SHH, SMO and GLI1 in the untreated rats and Primary Cultured Retinal Müller Cells. (A) Western blot analysis of SHH, SMO, and GLI1 expression in normal rats retinas after SHH-N or cyclopamine treatment shows that expression of each was elevated by SHH-N treatment but decreased by cyclopamine treatment. Quantitative analysis of protein expression of SHH (B), SMO (C) and GLI1 (D). (E) Western blot analysis of SHH, SMO, and GLI1 expression in 5.5mM glucose cultured primary Müller Cells after SHH-N or cyclopamine treatment shows that expression of each was elevated by SHH-N treatment but decreased by cyclopamine treatment. Quantitative analysis of protein expression of SHH (F), SMO (G) and GLI1 (H). n=2 per group. ★★★p < .01, ★p < .05, compared with PBS-treated rats and non-treatment Müller Cells. Fold changes are shown as mean ± standard deviation.