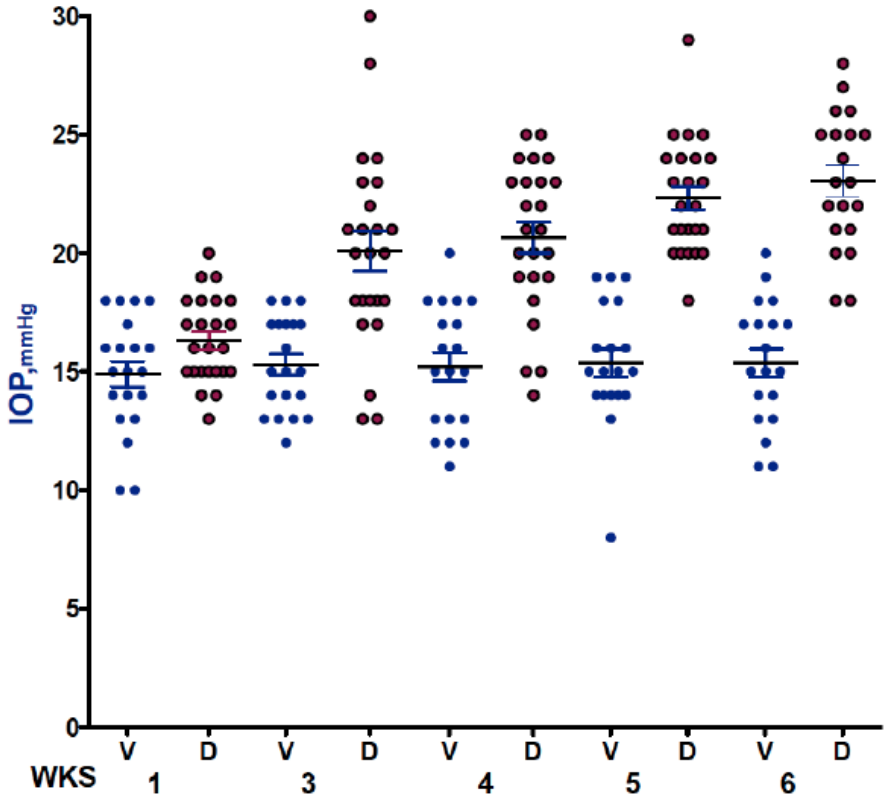
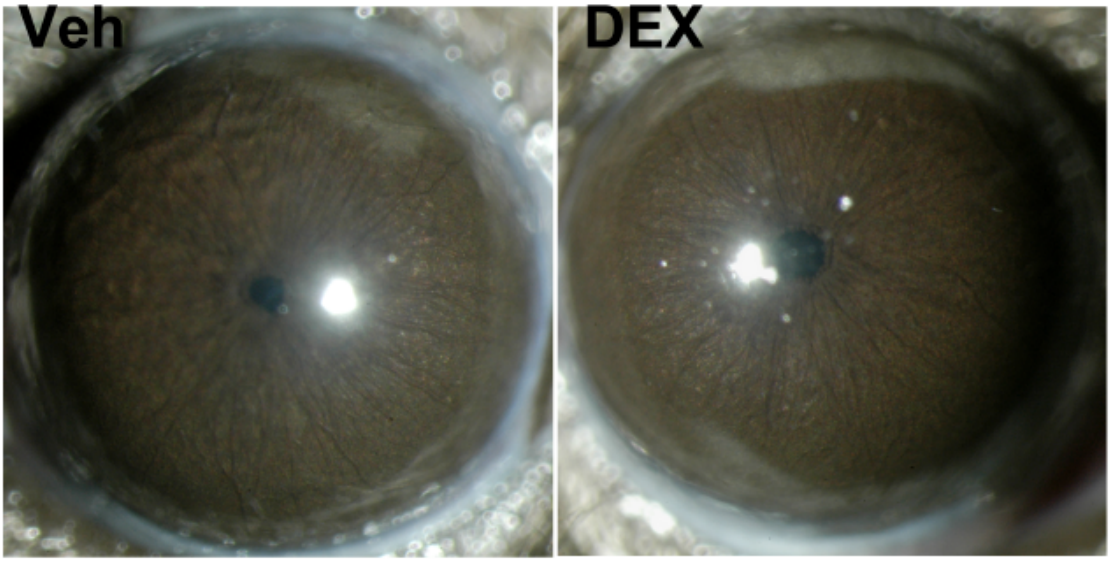
Supplementary Figures

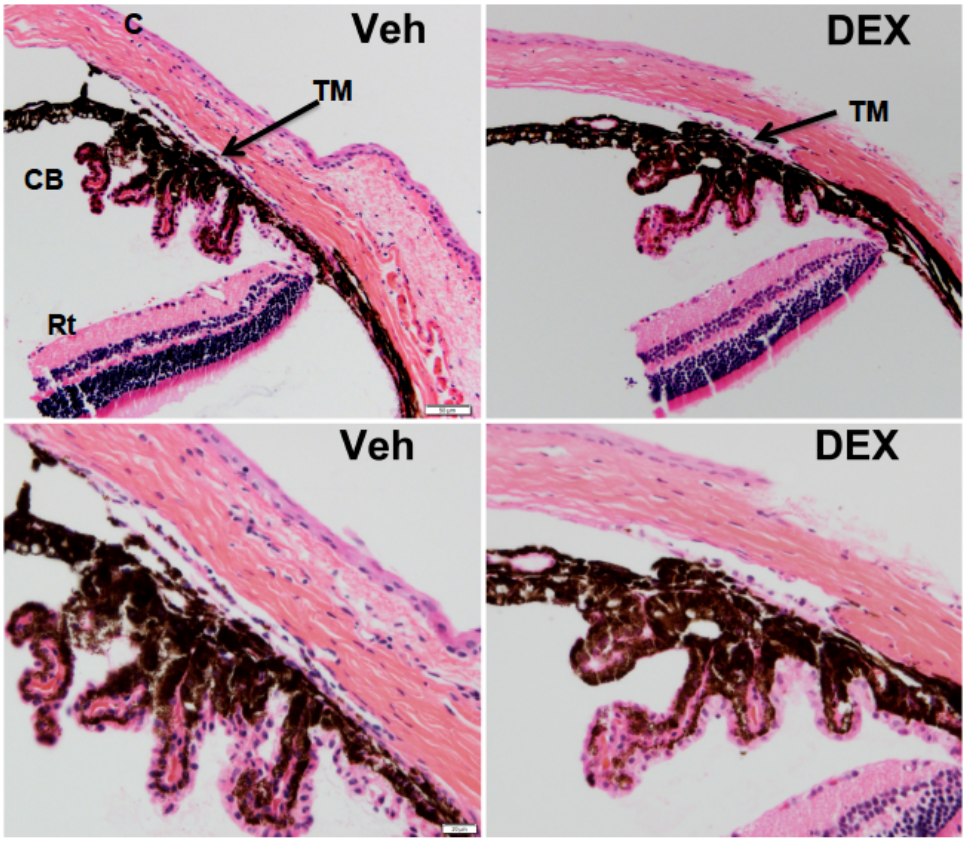


S. Fig 1: Individual IOP measurements of vehicle and dexamethasone-treated mice over the course of treatment. C57BL/6J mice were given vehicle or dexamethasone eye drops for 6 weeks. IOP was measured every week. IOP of each mice is shown here as a scatter plot (the same data are shown in Fig. 1 as a bar graph). IOP from each eye is considered one measurement since each eye responded differently to dexamethasone treatment. This experiment was performed in two groups at different times and data from both groups are combined here. The graph shows data from total 10-12 mice (n=20-24 individual eyes). Dexamethasone elevated IOP significantly at the 3rd week of treatment (15.30 ± 0.4419 mmHg, n=20 in control versus 20.08 ± 0.8447 mmHg, n=24 in dexamethasone-treated mice, t-test; $p<0.0001$). We therefore established IOP

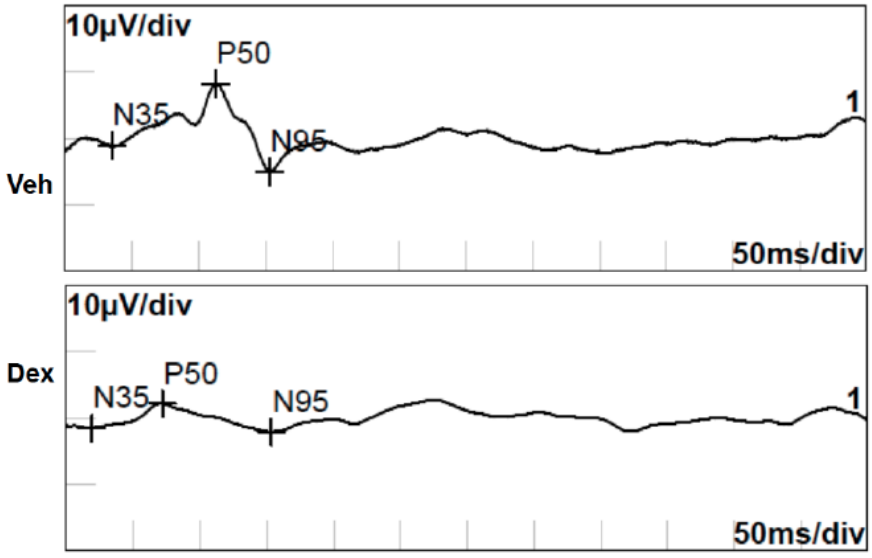
elevation by 4mmHg as statistically significant ocular hypertension in our mice (IOP of 20mmHg or more). Some of the dexamethasone treated mice did not show ocular hypertension until four weeks of treatment. At 5-weeks of dexamethasone treatment, 23 out of 24 eyes demonstrated IOP elevation. At 6-weeks of treatment, 18 out of 20 dexamethasone-treated eyes showed IOP elevation. It should be noted that two mice were sacrificed during this week. We also observed that a dexamethasone-treated eye with normal IOP of 18mmHg at 5 weeks of treatment had elevated IOP (23mmHg) at 6 weeks of treatment. In addition, two dexamethasone-treated eyes with normal IOP (18mmHg) at 6 weeks had elevated IOP (20 and 25 mmHg) at 5-weeks. These data suggest that mice with no IOP elevation (less than 20mmhg) may be outliers for that particular day's experimental settings. Thus, we conclude that dexamethasone treatment elevates IOP in most of the mice (90-95%)".



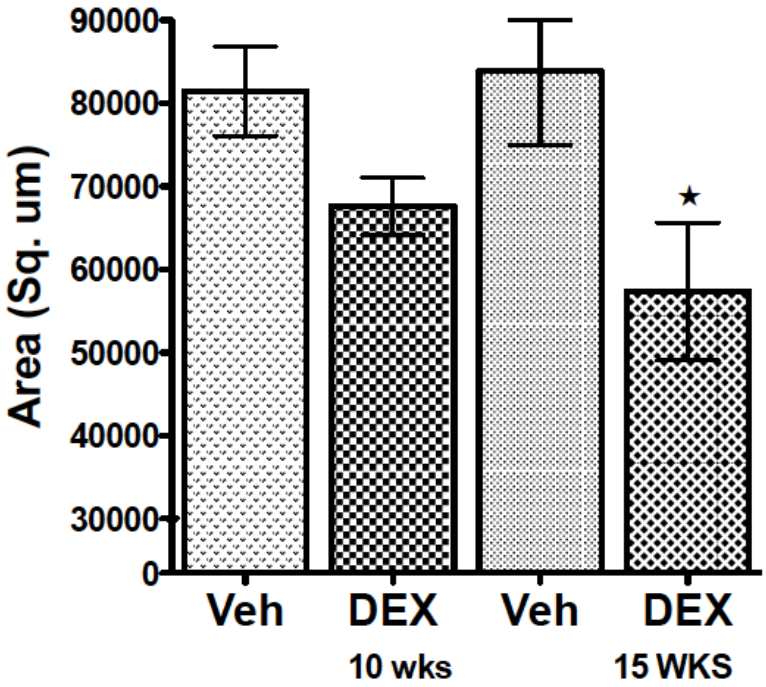
S. Fig. 2: Slit lamp examination of anterior chamber structures in 3-weeks vehicle and dexamethasone-treated mice. Slit lamp examination of the anterior chamber revealed no abnormalities in the iris, cornea, and lens. None of the mice treated with vehicle or dexamethasone showed cataract or other gross eye abnormalities.



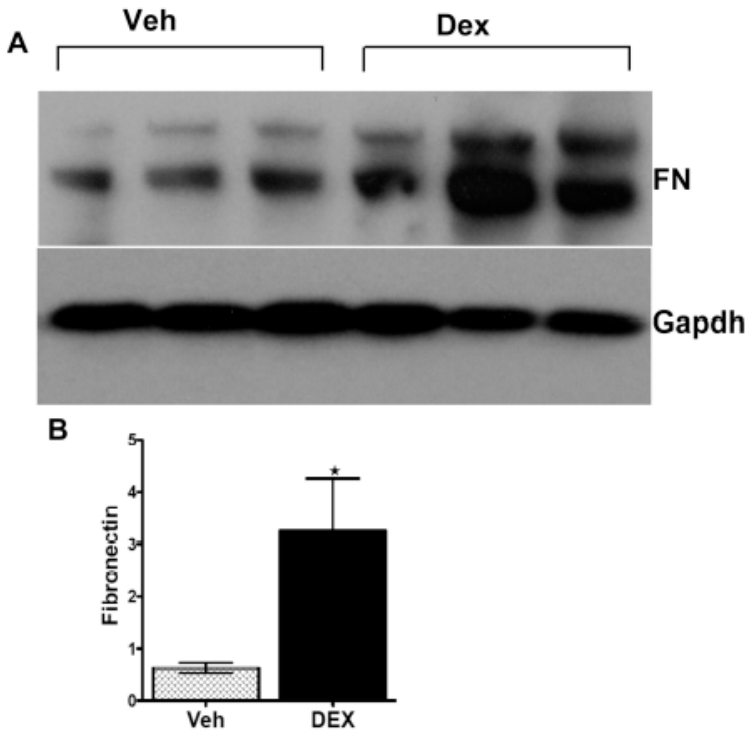
S. Fig. 3: Open iridocorneal angle in dexamethasone-treated mice. H and E staining of paraffin-embedded anterior chamber sections in 3-weeks treated mice revealed that dexamethasone-treated mice show an open iridocorneal angle. Top panel shows iridocorneal angle at lower magnification (scale bar = 50 micron). Bottom panel shows higher magnification of iridocorneal angle (scale bar = 20 microns). n=4. Arrow shows TM. C=cornea; Rt=retina; CB=ciliary body. No abnormalities were observed in vehicle or dexamethasone-treated tissues.



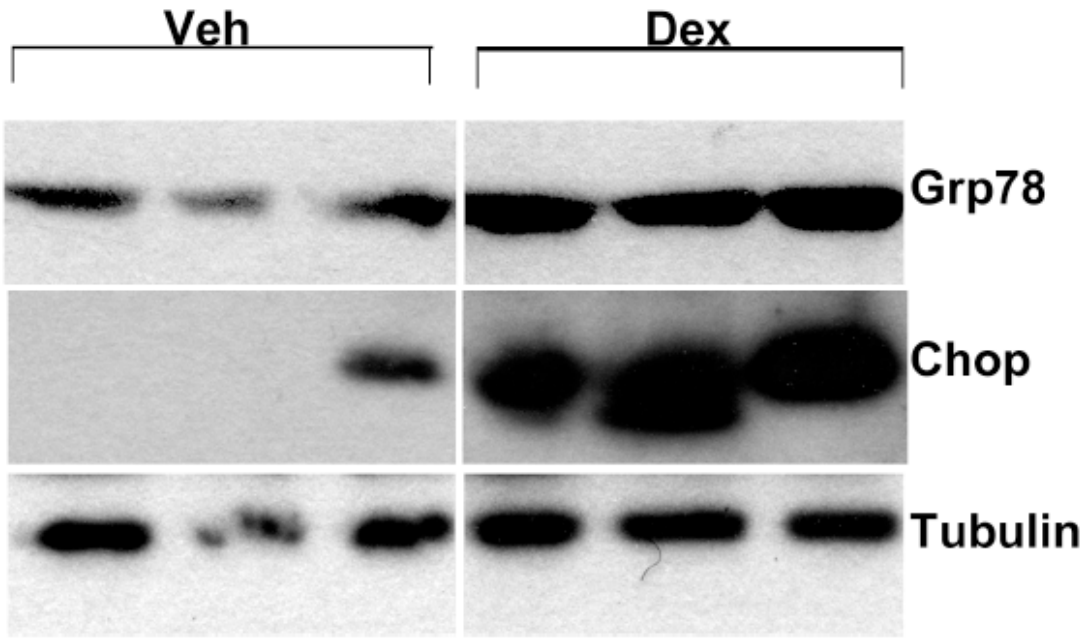
S. Fig. 4: Pattern ERG in vehicle and dexamethasone-treated mice. A representative pattern ERG in vehicle and dexamethasone-treated mice is shown. P50-N95 indicates a specific function of RGCs. In vehicle-treated mice (top panel) P50-N95 was 13.2 microvolts, which is decreased dramatically to 6.5 microvolts in dexamethasone-treated mice.



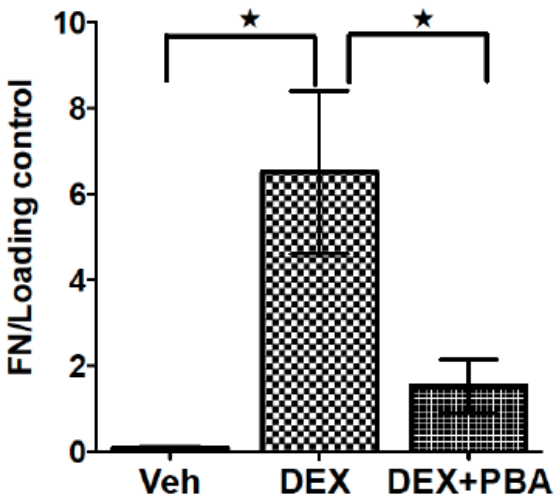
S. Fig. 5: Optic nerve area is reduced significantly in dexamethasone-treated mice compared to vehicle-treated mice. Optic nerve area stained with PPD was measured using Image J at 10 and 15-weeks in vehicle and dexamethasone-treated mice We observed that 10 and 15 weeks of dexamethasone treatment reduces optic nerve area significantly compared to vehicle-treated mice. n= 8-10. Data are mean ± SEM. statistical analysis by t-test, $*P < 0.05$.



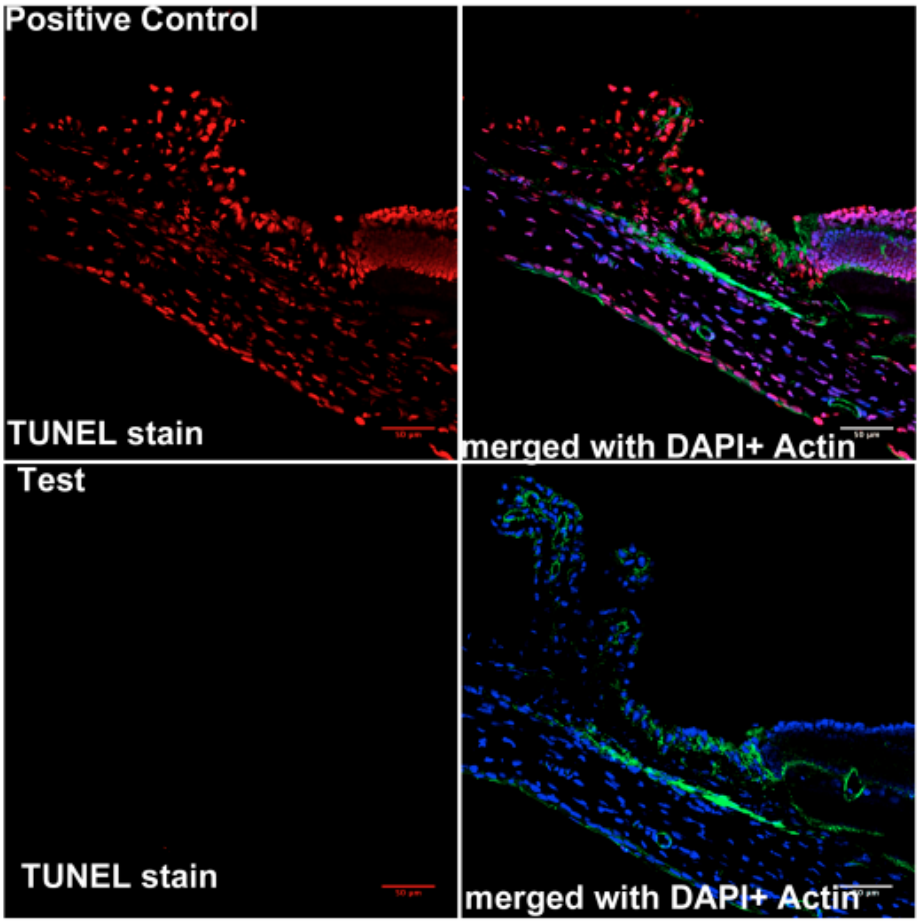
S. Fig. 6: Dexamethasone treatment increases fibronectin in the iridocorneal angle tissues. Western blot **(A)** and densitometric analyses **(B)** of fibronectin in 1-week vehicle and dexamethasone-treated mice show that dexamethasone significantly increases fibronectin levels in the iridocorneal angle tissues. Gapdh was used as a loading control. n=3 in each group, $p<0.05$.



S. Fig. 7: Dexamethasone treatment for 8-weeks increases ER stress in the iridocorneal angle tissues. Western blot analysis for Grp78 and Chop in the iridocorneal angle tissues at 8-weeks vehicle and dexamethasone-treated mice. Blots were run on the same gel but bands were cut and represented in the order presented. Tubulin was used as a loading control. Dexamethasone increased Grp78 and Chop compared to vehicle treated mice.



S. Fig. 8: PBA treatment reduces dexamethasone-induced elevated fibronectin levels. Densitometric analysis of Western blots for fibronectin in the iridocorneal angle tissues was normalized to loading control (Gapdh). Dexamethasone alone significantly increased fibronectin levels compared to vehicle treated mice. However, mice treated concomitantly with PBA and dexamethasone together demonstrated reduced fibronectin levels in the iridocorneal angle tissues.



S. Fig. 9: Examination of TM cell death in dexamethasone-treated mice. TUNEL stain was performed to examine TM cell death in dexamethasone-treated anterior chamber. Top panel represents a positive control in which the anterior chamber was treated with DNase1 (left side image shows TUNEL stain and right side image shows tunnel merged with DAPI and actin stain). Bottom panel shows tunnel stain in dexamethasone-treated anterior chamber. No cell death was observed in dexamethasone-treated anterior chamber structures.