associate with glial sprouts and blooms. (C, D) These cells were not concentrated at the base where glia exited the retina (arrow). Scale bars indicate $50 \,\mu$ m.

Figure 11. Large glial membranes were most prominent in neovascular AMD retinas.

Retina wholemounts stained for GFAP (red) are shown. Tiled images taken at 10x are shown from representative (A) non-AMD retina, (B) early AMD, (C) geographic atrophy, and (D) neovascular AMD. In all images, arrows indicate some of the preretinal glia. Images were taken with the fovea centered. (E) Graphs show the average percentage of posterior pole tiled images covered with $GFAP^+$ cells on the vitreal surface. (F) Graphs show the average percentage of the ILM covered by preretinal $GFAP^+$ structures. As shown, there was a significant increase in the $GFAP^+$ cells on the vitreal side of the ILM in neovascular AMD in the posterior pole but not outside this area. Scale bars indicate 200 µm. Asterisks indicate significant difference compared to the non-AMD group.

Supplemental Figure 1. A glial sprout on the retinal surface. A three dimensional rendering of a retina stained for GFAP (red), UEA lectin (blue), and IBA-1 (green) demonstrates the location of this glial sprout above a retinal vessel. IBA-1⁺ cells also associated with glial sprouts. The connection between individual sprouts is also noteworthy.

Supplemental Figure 2. Video of 3D rendered GS/vimentin and GFAP. A three dimensional rendering of a retina stained for GFAP (red), UEA lectin (blue), vimentin (yellow), and GS (green) demonstrates the GS/vimentin double-positive cells atop of a normal astrocyte template with no GFAP⁺ sprouts.

Supplementary Figure 3. The effect of AMD treatment on preretinal glial cells. Retinas in the neovascular AMD group were divided by treatment and the average percentage of the ILM