**Supplemental Figure S1:** To confirm the specificity of the antibodies, immunohistochemical staining with anti type I, II, IV and VI collagens of samples from a human donor eye containing vitreous body (cv), retina (re), choroid (ch) and sclera (sc) was performed. We observed specific and different staining patterns for the different anti-collagen antibodies: type I collagen was observed in the sclera and faintly in the choroid, type II collagen in the vitreous body, type IV collagen in retinal blood vessel walls (arrows) and in the choroid, and type VI collagen in the sclera and to a lesser extent in the choroid. Controls in which the primary antibody was omitted, and controls in which an isotype control (goat or rabbit IgG) was used, were negative. Bars: 50 μm.

![Supplemental Figure S1](http://iovs.arvojournals.org/pdfaccess.ashx?url=/data/journals/iovs/934564/)

**Supplemental Figure S2:** Western blot analysis was used to confirm the specificity of the antibodies against the different types of collagen. Retinal Müller cells (MIO-M1) were harvested in denaturation buffer and proteins were analyzed by SDS-PAGE using 7.5% running gel. Collagen bands could be appreciated at different heights for the different types of collagen: type I collagen at 140 kDa, type IV collagen at 210 kDa and type VI collagen at 120 kDa.

![Supplemental Figure S2](http://iovs.arvojournals.org/pdfaccess.ashx?url=/data/journals/iovs/934564/)
Supplemental Figure S3: Schematic diagram of a proposed theory on the pathogenesis of epiretinal membrane. The initial trigger for Müller cell activation could be a combination of increased stiffness of the vitreoretinal interface and posterior vitreous detachment (PVD). The former could be the result of age-related changes including the accumulation of advanced glycation end products (AGEs). The latter may induce focal traction to Müller cells and add to the process of cell dehiscence and migration. Müller cells inside the retina are surrounded by a compliant extracellular matrix and will sense a stiffer matrix when migrating onto the internal limiting membrane of the retina. This, in combination with transforming growth factor beta (TGF-β) stimulation will contribute to the process of Müller cell transdifferentiation into a myofibroblast phenotype and production of extracellular matrix forming the idiopathic epiretinal membrane.