Study finds lack of VEGF can cause defects similar to dry macular degeneration

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Scientists at Schepens Eye Research Institute have found that when the eye is missing a diffusible form of vascular endothelial growth factor (VEGF), i.e. one that when secreted can reach other cells at a distance, the retina shows defects similar to "dry" macular degeneration, also called geographic atrophy (GA). This finding, published in the November 3, 2009 print edition of PNAS (*Proceedings of the National Academy of Sciences*), not only increases the understanding of the causes of this blinding disease, but it may also impact the use of anti-VEGF drugs, such as Lucentis, which are designed to neutralize VEGF in eyes with "wet" macular degeneration.

"These results are significant for several reasons. We know little about what causes GA or how to treat it. Our discovery may be an important piece of the puzzle. It shows that reduced VEGF from the retinal pigment epithelium (RPE), RPE, the bottommost layer of the retina, to the choriocapillaris (CC) - the small blood vessels beneath retina-- leads to degeneration of the CC. Therefore, the continuous blockage of VEGF may contribute to the development of or a worsening of GA," says Patricia D'Amore, principal investigator of the study and senior scientist at Schepens.

VEGF is a protein that stimulates the growth of new blood vessels. The eye produces several different forms of VEGF that differ in their size and their ability to move away from the producing cell.

Age-related macular degeneration (AMD) is a disease that destroys the

macula, the central part of the retina responsible for detailed vision needed for reading, driving and face recognition. It comes in two types—"wet" and "dry." In wet AMD, a pathological overproduction of VEGF leads to the development of abnormal blood vessels, which leak and damage the retina. Wet AMD can be treated with some success with anti-VEGF drugs that block abnormal blood vessel growth and leakage. Dry macular degeneration develops less rapidly, and is related to an accumulation of debris under the retina that can advance to GA where RPE and underlying vessels are lost.

Knowing that the RPE in the adult produced VEGF, the Schepens team hypothesized that in a healthy individual ,the RPE produces forms of VEGF that, when secreted, can move away from the RPE and reach the underlying CC to support its function and survival. The CC vessels are extremely important as they supply the photoreceptors (the light- and color-sensitive cells in the macula) with oxygen and nutrients necessary for vision.

In the PNAS study, the researchers tested their hypothesis using a genetic mouse model in which the RPE produced a form of VEGF that was unable to diffuse. As the mice aged, they began to display an age-dependent degeneration of both the CC and RPE, culminating with the death of photoreceptors and vision loss, similar to that observed in GA,.

The next step in the research, according to the first author Dr. Magali Saint-Geniez, is to determine if this model can be used to investigate the role of RPE-CC interaction in AMD and to design new therapies.

Source: Schepens Eye Research Institute

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