# Study suggests new approach to common cause of blindness

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Researchers at the University of North Carolina at Chapel Hill School of Medicine in collaboration with lead investigators at the University of Kentucky have identified a new target for the diagnosis and treatment of age-related macular degeneration, the most common cause of blindness in older Americans.

In a study published by the journal *Nature*, the researchers demonstrate that blocking the activity of a specific protein - called CCR3 -- can reduce the abnormal blood vessel growth that leads to macular degeneration. Furthermore, targeting this new protein may prove to be safer and more effective than the current treatment for the disease, which is directed at a protein called vascular endothelial growth factor or "VEGF."

The discovery -- made in mouse models and cultured human cells --may also enable physicians to catch the disease in its earliest stages, before blood vessels have fully infiltrated and destroyed the central portion of the eye's retina -- an area known as the macula -- to cause vision loss.

"It would be much better to prevent the disease in the first place," said study co-author and principal investigator of the UNC study site, Mary Elizabeth Hartnett, M.D., a professor of ophthalmology in the UNC School of Medicine. "An exciting implication of this study was that the CCR3 protein could be detected in early abnormal blood vessel growth, giving us the opportunity to prevent structural damage to the retina and preserve vision."

Age-related macular degeneration (AMD) affects 30 to 50 million people globally, and that number is expected to double in the next decade as the baby boomer generation ages. The disease is currently treated with drugs that block the effects of VEGF, a growth factor that promotes the growth of abnormal blood vessels. However, because this factor is also involved in the growth and health of normal blood vessels, concerns have been raised about the safety of its long-term use. To date, however, these anti-VEGF agents have been found to be safe.

Thus, the investigators sought to identify a new target for treatment that is specific to AMD. They detected the presence of the CCR3 protein in eye tissue from humans with AMD but not in that of individuals of similar age who did not have the disease. When they blocked CCR3, either with drugs or through genetic engineering, they saw a decrease in the generation of abnormal blood vessels. Drugs targeting CCR3 were significantly more effective than those targeting VEGF, meaning this could represent a new therapy for the two-thirds of patients that do not respond to current treatment.

The researchers now may look to see if levels of the protein can be detected in the bloodstream in order to identify people who are at risk of developing the disease. They also plan to search for genetic changes in the CCR3 gene in patients with AMD to better understand its causes.

Source: University of North Carolina School of Medicine (news : web)