

# New research findings may help stop age-related macular degeneration at the molecular level

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Researchers at University College London say they have gleaned a key insight into the molecular beginnings of age-related macular degeneration, the No. 1 cause of vision loss in the elderly, by determining how two key proteins interact to naturally prevent the onset of the condition.

In a paper to be published in a forthcoming issue of the [Journal of Biological Chemistry](#), the team reports for the first time how a common blood protein linked to the eye condition reins in another protein that, when produced in vastly increased amounts in the presence of inflammation or infection, can damage the eye.

"By starting to understand these interactions in greater detail, we can begin to devise methods that will ultimately prevent the development of blindness in the elderly," said Zuby Okemefuna, the lead author of the paper to be published Jan. 8.

Age-related [macular degeneration](#), or AMD, is painless but affects the macula, the part of the retina that allows one to see fine detail. One form of the debilitating condition, known as "wet" AMD, occurs when abnormal and fragile blood vessels grow under the macula, leaking blood and fluid and displacing and damaging the macula itself. The second form, "dry" AMD, occurs when light-sensitive cells in the macula slowly break down.

It is believed that both forms start on a common molecular route and then deviate into dry or wet AMD, explained the research leader, Steve Perkins.

"The earliest hallmark of AMD is the appearance of protein, lipid and [zinc](#) deposits under the [retinal pigment](#) epithelial cells," he said, adding that the yellowish deposits, usually discovered by an ophthalmologist, are commonly known as "drusen."

The researchers studied two proteins involved in drusen formation -- blood protein Factor H and a second [blood protein](#) known as C-reactive protein -- and showed that Factor H binds to C-reactive protein when C-reactive protein is present in large amounts, as in the case of infection, to reduce the potentially damaging effects of an overactive immune system.

"In the eye, during the normal processes of aging, cells will die naturally for all sorts of reasons," Okemefuna said. "The blood supply to the eye will bring C-reactive protein with it, and a low level of C-reactive protein activity will enable the normal processes of clearance of dead cells at the retina through mild inflammation. In conditions of high inflammation, the levels of C-reactive protein in the retina will increase dramatically."

Uncontrolled C-reactive protein activity causes damage to the retina, which is followed by more inflammation and then even more damage to the retina, and so forth.

"It's the debris of broken up retinal cells, some of which is caused by this cycle, that is deposited as drusen," Okemefuna said.

The team also found that a genetically different form of Factor H does not bind to the C-reactive protein quite as well as the normal one, making people who carry the modified protein more vulnerable to an

immune system attack in the eye and, thus, drusen buildup.

"In normal individuals, further damage to the [retina](#) by prolonged exposure to high levels of C-reactive protein is prevented by Factor H. C-reactive protein also prevents Factor H from clumping together and initiating the processes that lead to drusen formation," Perkins said. "Both these 'good' activities of Factor H are much reduced in the genetically different form of Factor H."

While there is no known cure for AMD, existing therapies aim to treat the symptoms and delay progression.

"It is interesting how the interaction of these two blood proteins protects the eye during crisis," Perkins said. "The two proteins also can be involved in a rare and often fatal cause of kidney failure in children. We now are better positioned to begin to work out preventative strategies for these diseases."

Provided by American Society for Biochemistry and Molecular Biology

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