Researchers create first model for retina receptors

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A team of scientists at the University of Oklahoma Health Sciences Center has created the first genetic research model for a microscopic part of the eye that when missing causes blindness. The research appears in a recent issue of the *Journal of Biological Chemistry*.

The team led by OU scientists at Dean McGee Eye Institute also includes researchers from Harvard Medical School. The group is studying how diabetes and insulin receptors affect the eye, and in many cases cause blindness. In diabetes, the insulin receptors malfunction and scientists have yet to figure out why.

"Our hope is to test drug compounds and therapeutic agents to see if they can prolong the life of the receptor cells and either delay or prevent blindness. Therapies could include a pill or gene therapy to activate the malfunctioning receptor," said Raju Rajala, Ph.D., principal investigator on the project.

Rajala said researchers expect to have some form of therapy available within 15 years.

They are focusing on an insulin receptor in the eye's rods, which are part of the retina. The rods translate what we see into electric signals to the brain. When the receptors aren't working, blindness occurs.

To learn more about how the receptors work and how proteins and insulin play a role in their function, scientists needed a research model to

test their ideas. With the new model at OU, scientists hope to find ways to significantly delay blindness or prevent it, especially in patients with diabetic retinopathy.

Diabetic retinopathy is the most common diabetic eye disease and a leading cause of blindness in American adults. It is caused by changes in the blood vessels of the retina.
In some people with diabetic retinopathy, blood vessels may swell and leak fluid. In other people, abnormal new blood vessels grow on the surface of the retina. A healthy retina is necessary for good vision.

"We are looking for clues to understand the progression of diabetic retinopathy so we can eventually stop it," Rajala said. "We still don't understand why the receptors malfunction or what their defense mechanism is. We needed a model to understand this process and now we have one."

Source: University of Oklahoma