Scientists identify an innate function of vitamin E

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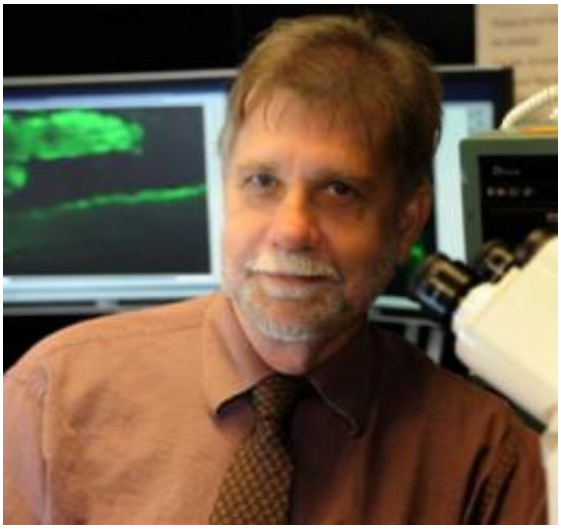

Dr. Paul McNeil, cell biologist at Georgia Health Sciences University, has discovered one of the innate functions of vitamin E. Credit: Phil Jones/GHSU photographer

It's rubbed on the skin to reduce signs of aging and consumed by athletes to improve endurance but scientists now have the first evidence of one of vitamin E's normal body functions.

The powerful antioxidant found in most foods helps repair tears in the plasma membranes that protect cells from outside forces and screen what enters and exits, Georgia Health Sciences University researchers report in the journal *Nature Communications*.

Everyday activities such as eating and exercise can tear the plasma membrane and the new research shows that vitamin E is essential to repair. Without repair of muscle cells, for example, muscles eventually waste away and die in a process similar to what occurs in muscular dystrophy. Muscle weakness also is a common complaint in diabetes, another condition associated with inadequate plasma membrane repair.

"Without any special effort we consume vitamin E every day and we don't even know what it does in our bodies," said Dr. Paul McNeil, GHSU cell biologist and the study's corresponding author. He now feels confident about at least one of its jobs.

Century-old animal studies linked vitamin E deficiency to muscle problems but how that happens remained a mystery until now, McNeil said. His understanding that a lack of membrane repair caused muscle wasting and death prompted McNeil to look at vitamin E.

Vitamin E appears to aid repair in several ways. As an antioxidant, it helps eliminate destructive byproducts from the body's use of oxygen that impede repair. Because it's lipid-soluble, vitamin E can actually insert itself into the membrane to prevent free radicals from attacking. It also can help keep phospholipids, a major membrane component, compliant so they can better repair after a tear.

For example, exercise causes the cell powerhouse, the mitochondria, to burn a lot more oxygen than normal. "As an unavoidable consequence you produce reactive oxygen species," McNeil said. The physical force of exercise tears the membrane. Vitamin E enables adequate plasma membrane repair despite the oxidant challenge and keeps the situation in check.

When he mimicked what happens with exercise by using hydrogen peroxide to produce free radicals, he found that tears in skeletal muscle

cells would not heal unless pretreated with vitamin E.

Next steps, which will be aided by two recent National Institutes of Health grants, include examining membrane repair in vitamin E-deficient animals.

McNeil also wants to further examine membrane repair failure in diabetes. Former GHSU graduate student Dr. Amber C. Howard showed in a recent paper in the journal Diabetes that cells taken from animal models of types 1 and 2 diabetes have faulty repair mechanisms. Howard found high glucose was a culprit by soaking cells in a high-glucose solution for eight to 12 weeks, during which time they developed a repair defect. It's also well documented that reactive oxygen species levels are elevated in diabetes.

The *Nature Communications* paper showed that vitamin E treatment in an animal model of diabetes restored some membrane repair ability. Also, an analogue of the most biologically active form of vitamin E significantly reversed membrane repair deficits caused by high glucose and increased cell survival after tearing cells in culture.

Now McNeil wants to know if he can prevent the development of advanced glycation end products – a sugar that high glucose adds to proteins that his lab has shown can also impede membrane repair – in the animal models of diabetes. The researchers have a drug that at least in cultured animal cells, prevents repair defects from advanced glycation end products.

Provided by Georgia Health Sciences University

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