

Florida Museum of Natural History, College of Liberal Arts and Sciences

GENETICS RESEARCH PROVIDES INSIGHTS ABOUT FLOWERING PLANTS

FLOWERING PLANTS HAVE EVOLVED AT EXPLOSIVE RATES THROUGHOUT HISTORY, YET SCIENTISTS SINCE CHARLES DARWIN HAVE BEEN FACED WITH THE GREAT BIOLOGICAL MYSTERY OF HOW THEY ORIGINATED.

A new University of Florida study published in December in the *Proceedings of the National Academy of Sciences* presents the deepest insight to the genes that made up the first flower, the common ancestor of all flowering plants, and how those genes have changed over time.

“Our survival depends on products we get from the flower — grains, fruits and many other materials,” said Doug Soltis, UF distinguished professor of biology and project co-investigator. “Crop improvement is so important,

but you don’t understand how a flower is put together unless you have a reference point — you can’t modify what you can’t understand.”

After nearly 10 years of research funded by the National Science Foundation, scientists from the Florida Museum of Natural History, the UF Department of Biology and the UF Genetics Institute are bringing the study to a close.

“There are 350,000 species of flowering plants (or angiosperms), and they serve as the foundation of nearly all



Jeff Gage

of Earth’s ecosystems, yet we don’t know how the flower originated,” said Pam Soltis, UF distinguished professor, Florida Museum of Natural

History curator and project co-investigator. “We now know the origin of many of the genes responsible for making a flower and how those genes

College of Medicine

PROTEIN MAY EXPLAIN ROLE OF DIET IN AGING

RESTRICTING CALORIES EXTENDS LIFE AND SLOWS A RANGE OF AGE-RELATED DISORDERS IN MICE, RATS AND OTHER ORGANISMS. BUT EVEN AFTER EIGHT DECADES OF RESEARCH ON THE SUBJECT, SCIENTISTS ARE STILL UNCLEAR JUST HOW CALORIC RESTRICTION EXERTS ITS AGE-BATTLING INFLUENCE.

Now, for the first time in mammals, researchers at the University of Florida and the University of Wisconsin-Madison have sleuthed out the role of a key player in the process, using age-related hearing loss as an example. The protein in question, called Sirt3, could provide a new target for anti-aging drug therapies. The findings were reported in the journal *Cell* in November.

The researchers found that when Sirt3 is absent, caloric restriction loses its anti-aging powers. They uncovered details of how the protein, an enzyme found primarily in mitochondria — the energy-producing centers of cells — wards off cell death by maintaining an environment that combats destructive chemicals.

“Knocking it out seems to be very negative for

mitochondrial function and allows the accumulation of oxidative stress and damage to neurons and other cells,” said Christiaan Leeuwenburgh, chief of UF’s Biology of Aging division. “That’s an important clue about the role that Sirt3 plays in protecting cells from age-related damage.”

Age-related hearing loss is the most common sensory disorder among the elderly, affecting more than 40 percent of people older than 65 and projected to affect 28 million Americans by 2030, according to the Department of Health and Human Services.

The disorder is marked by the death of sensory hair and nerve cells in the inner ear. While those cells are long-lived, they do not regenerate, so their demise means permanent loss of hearing. But all is not lost, since the environment in which those cells reside can be remodeled over time as damaged organelles such as mitochondria get replaced. Caloric restriction helps to rescue those damaged cells by reducing oxidative damage.

Having previously shown that restricting the diet induces expression of the protein Sirt3 in the inner ear, the researchers now

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have changed during the history of angiosperms.”

A 2009 UF study traced the origin of flowers using genetic data for the avocado

(a representative of one of the early lineages of flowering plants) and a well-known plant in genetics research, *Arabidopsis thaliana*. The new study includes additional comparisons with a water lily, California poppy and cycad (a gymnosperm or non-flowering seed plant) and shows how the first flowers evolved from pre-existing genetic programs in gymnosperm cones.

“We have a much better understanding of the flower than we did 10 years ago and it’s a huge improvement,” Doug Soltis said. “We don’t know every pathway, but we have a much better handle on what makes those parts tick.”

Typical angiosperms have flowers with four organs: sepals (typically green), petals

(typically colorful), stamens (male organs, which produce pollen) and carpels (female organs, which produce eggs). But in the earliest flowers, the distinct borders between their floral organs fade to a blur. The flowers of early angiosperms have organs that merge into each other — for example, a stamen of a water lily produces pollen but it may also be petal-like and colorful.

“Our study found that the floral organs of basal angiosperms merge not only in appearance but also in their underlying genetic pathways,” Pam Soltis said. “During evolution, the timing and location of where these genes act have become restricted, ultimately producing flowers with separate and

distinguishable flower parts.”

“These missing links are incredibly important,” Doug Soltis said. “They are our key to the past.”

The study was a collaboration of researchers at UF, Penn State, the University of Georgia and the University at Buffalo. The first author on the paper is Andre Chanderbali, a postdoctoral student at the Florida Museum and the UF Department Of Biology.

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show that Sirt3 aids caloric restriction by combating some of the chemical changes that play a major role in the process of aging.

The enzyme belongs to a class of compounds called sirtuins that are known to have anti-aging effects in lower organisms including yeast and flies. Until now, however, there wasn’t clear evidence that the effect extends to mammals.

“This is a major step in terms of understanding aging retardation by dietary restriction — it doesn’t work without

Sirt3,” said Shinichi Someya, of the University of Wisconsin-Madison.

In normal mice, lowering calorie intake to 75 percent of a regular diet reduced hearing loss, but in Sirt3-deficient mice, dietary restriction had no such effect. Further, after caloric restriction, mice lacking Sirt3 lost more cellular structures vital for hearing — sensory hair and nerve cells in the ear — than did normal mice on a similarly restricted diet.

Corresponding with that observation, the researchers found that while caloric restriction reduced oxidative

damage to DNA in inner ear cells in normal mice, it did not have that effect in mice that lacked Sirt3.

Effects seen in the ear were also observed in brain and liver tissue, suggesting that Sirt3 might have a role well beyond age-related hearing loss, and a potential benefit in cardiovascular and neurological diseases.

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