Medical Xpress

Scientists identify gene that could hold the key to muscle repair

April 18 2011

(PhysOrg.com) -- Researchers have long questioned why patients with Duchenne muscular dystrophy (DMD) tend to manage well through childhood and adolescence, yet succumb to their disease in early adulthood, or why elderly people who lose muscle strength following bed rest find it difficult or impossible to regain. Now, researchers at the National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS), part of the National Institutes of Health, are beginning to find answers in a specialized population of cells called satellite cells. Their findings, reported in the journal *Genes & Development*, suggest a potential therapeutic target for conditions where muscle deterioration threatens life or quality of life.

Key to the development of skeletal muscle of the embryo and fetus, satellite cells continue to actively increase muscle mass through infancy. After that, they decrease in number and become quiescent, or inactive, until they are activated by injury or degeneration to proliferate. The process, which enables the body to repair damaged muscle, works quite well — to a point, says Vittorio Sartorelli, M.D., senior investigator in the NIAMS Laboratory of Muscle Stem Cells and Gene Regulation and lead author of the study.

For example, when a young person experiences muscle loss after a period of inactivity, muscle rebuilds as soon as activity is resumed. However, in the elderly, muscles lose that capacity. Similarly, in patients with DMD, the initial phases of muscle degeneration are effectively counteracted by the ability of satellite cells to regenerate.

"That is why people can survive until they are 20 years old without much of a problem, but, at a certain point, satellite cells stop proliferating," said Dr. Sartorelli. "That is the point at which the patient will start developing weakness and problems that will ultimately lead to death."

Suspecting a genetic switch that might turn off satellite cell proliferation in these circumstances, the scientists looked to a gene called Ezh2, known to keep the activity of other genes in check. When they genetically inactivated Ezh2 in satellite cells of laboratory mice, the mice failed to repair muscle damage caused by traumatic injury — satellite cells could not proliferate.

Ezh2 expression is known to decline during aging, and the new research in mice suggests that therapies to activate Ezh2 and promote satellite cell proliferation might eventually play a role in treating degenerative muscle diseases.

"We will not be able to cure the muscular dystrophies with this approach because the mutation in the gene that causes the diseases would remain. But certainly, if we can extend the period in which the satellite cells proliferate and compensate for the underlying defect, we might increase the lifespan of people with muscular dystrophy. We could certainly increase their quality of life," said Dr. Sartorelli.

Likewise, in the elderly, tweaking the gene in satellite cells would not increase their lifespan, but could increase their quality of life by helping to prevent falls and enabling them to move and walk better and go about their daily activities.

Dr. Sartorelli cautions that while the identification of Ezh2's role is a crucial step, any therapies are still many years away.

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Provided by National Institutes of Health

Citation: Scientists identify gene that could hold the key to muscle repair (2011, April 18) retrieved 3 May 2024 from https://medicalxpress.com/news/2011-04-scientists-gene-key-muscle.html

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