Researchers discover mechanism that limits scar formation

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Researchers from the University of Illinois at Chicago have discovered that an unexpected cellular response plays an important role in breaking down and inhibiting the formation of excess scar tissue in wound healing.

Their study was published online this week in Nature Cell Biology.

When an organism suffers severe injury, specialized cells are "recruited" to the wound site that rapidly produce extracellular matrix proteins such as collagen to provide structural support to the tissue, according to Lester Lau, professor of biochemistry and molecular biology at the UIC College of Medicine and principal investigator in the study.

Joon-Il Jun, a postdoctoral fellow working in Lau's lab and first author of the paper, found that fibroblasts recruited to the site of skin wounds were entering a state of reproductive dormancy, or cell-cycle arrest, called senescence.

This was quite unexpected, Jun said. Until now senescence was believed to occur in cells that suffered DNA damage -- to prevent them from proliferating and, possibly, becoming cancerous.

He discovered that the senescent fibroblasts were making proteins that degraded the extracellular matrix and accelerated the breakdown of collagen. The senescent cells also stopped making collagen.

"The accumulation of senescent cells in the wound has the biological effect of inhibiting the formation of excess scar tissue," Jun said.

Jun also discovered that a protein called CCN1 is responsible for turning on the senescent state in fibroblasts. He was able to show that in mice with a mutated, non-functional form of CCN1, the fibroblasts at the site of a skin wound did not become senescent, and the wound developed excessive scar tissue.

Further, Jun was able to "rescue" the mutated mice by applying CCN1 protein topically to the skin wound, triggering fibroblast senescence and limiting the formation of scar tissue.

The discovery that senescence is a normal wound-healing response in the skin; that senescence in the wound serves an anti-fibrotic function; and that CCN1 is the critical protein that controls this process may prove important in understanding a wide range of pathological conditions related to tissue scarring, said Lau.

"For example, chronic injury to the liver from a number of causes, including viral infections, alcoholism, diabetes and obesity, leads to fibrosis and may progress to cirrhosis," Lau said. "After a heart attack, accumulation of scar tissue in the heart impairs its ability to pump efficiently."

The ability to control the formation of scar tissue, or fibrosis, has important implications for future therapies for treating wound-healing disorders, including organ damage where functional tissue is replaced with scar tissue, Lau said.

Provided by University of Illinois at Chicago

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