Cells beneath the skin explain differences in healing

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A microscopic comparison between normal skin (left) and scarred skin. Credit: Yale University

Differences in the cells that give skin its resilience and strength during wound repair may explain why individuals heal differently, according to a new Yale study published Nov. 23 in the journal *Science*.
Fibroblasts, the **cells** that form the protein structure beneath the surface of the **skin**, were once thought to be fairly uniform in their function. However, the new study found that subsets of fibroblasts may explain why skin regeneration is less robust in older people and how certain types of scars form.

"These subsets of cells may explain different healing potentials in different people," said senior author Valerie Horsley, associate professor of molecular, cellular, and developmental biology.

Horsley and first author Brett Shook studied the genetics of fibroblasts and looked at their effects in mice and humans and found multiple differences in how these cells respond to injury and changes that occur during aging. For instance, after injury, a subset of fibroblasts that normally produce adipocytes, or fat cells, start forming **scar tissue** to repair the skin tissue. Interestingly, say the researchers, these cells are reduced in human skin compared to mouse skin, which may explain why scars are more prevalent in people than mice.

The study also identified the essential role of immune cells called macrophages in not only fighting infection but also healing wounds and scar formation. Macrophages that appear at the peak of tissue regeneration can selectively signal to a subset of fibroblasts. Human scar tissue contains more **macrophages** and **fibroblast** subsets and both populations are reduced in poorly healing wounds from aged mice.

"By identifying functionally distinct populations of skin cells and revealing the signals that control their behavior, we hope to develop new targeted therapies for the treatment of defective wound healing and scar formation," Shook said.

**More information:** Brett A. Shook et al. Myofibroblast proliferation and heterogeneity are supported by macrophages during skin repair,

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