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Manipulating the SLIT2-ROBO2-EID1-EP300 pathway to overcome fibrosis

Researchers at Stanford University have identified EP300 modulators as effective treatments for reducing skin scarring, demonstrating significant regeneration of hair follicles, sweat glands, and connective tissue architecture.

Skin wounds invariably heal by developing fibrotic scar tissue. Although scarring restores skin integrity, the resulting fibrosis may lead to devastating disfigurement, growth restriction, and permanent functional loss. There are many clinical and consumer options available for scar treatment, such as topicals, injectables, laser treatments, and surgical revision. While they offer improvement in appearance and flexibility, none effectively prevent or reverse the underlying fibrotic process.

Stanford researchers have discovered a novel method using either EP300 inhibitors or SLIT2 protein to prevent skin scarring. Using a mouse model of wound healing, they found that a single application of EP300 inhibitor or SLIT2 immediately following wounding dramatically reduces scarring. Additionally, unlike bare untreated wounds, treated wounds show clear regrowth of skin appendages, such as hair follicles and sweat glands. Using a machine learning algorithm for quantitative assessment, they found that the connective tissue architecture in treated wounds strongly resembles that of unwounded skin. Importantly, the treatments do not delay wound closure. Targeting the SLIT2-ROBO2-EID1-EP300 pathway can revolutionize scar treatment and skin regeneration.

Stage of Development
Preclinical

Applications

- Scar prevention
- Other fibroses of the skin and body

Advantages

- Reduced scarring
- Improved connective tissue
- Improved scar appearance
- Regrowth of functional skin elements (hair follicles and sweat glands)
- No delay of wound closure
- No competing technology

Patents

- Published Application: WO2026015476

Innovators

- Derrick Wan
- Dayan Li
- Michelle Griffin
- Michael Longaker

Licensing Contact

Inyoung Lee

Licensing Manager, Life Sciences

Email