Docket #: S21-299

Use of CD36 inhibitors for the prevention of skin scarring

Stanford inventors have developed a novel method of using CD36 inhibitors to prevent and reduce skin scarring. They found that the protein JUN, a major driver of tissue scarring in many organs including the lung and skin, initiates fibrosis via CD36 in fibroblasts. Preclinically, the inventors have shown that CD36 inhibitors can successfully reduce scarring by reducing JUN mediated skin fibrosis.

There is no current therapeutic strategy that can prevent or reduce fibrotic process. This novel method of using CD36 inhibitors can be an effective treatment for patients suffering from skin scarring. It can reduce scarring, improve scar appearance, and allow regrowth of skin elements. These are highly significant treatment outcomes for patients. Moreover, this method also has potential for treating fibrosis in other important organs, such as lung and liver.

Stage of development
Research – in vivo

Applications

- Skin fibrosis
- Fibrosis in lung, liver

Advantages

- Effective

Publications

- Griffin, M.F. et al. JUN promotes hypertrophic skin scarring via CD36 in preclinical in vitro and in vivo models. *Science Translational Medicine* (2021)

Patents

- Published Application: WO2023028304

Licensing Contact

Eileen Lee

Email