

Gene Editing and Delivery of Myeloid Cells to Promote Wound Healing

S20-412

Cas9 knockout of Ndr2 in a patient's own dendritic cells, seeded on a hydrogel and applied topically; diabetic wounds closed 5 days earlier than control-cell wounds

MODALITY
Autologous DC therapy

TARGET
Ndr2

ROUTE
Topical hydrogel

PATENT
Pending (US)

Technology

Non-viral, ex vivo CRISPR/Cas9 editing knocks out Ndr2 in dendritic cells from the patient's own blood, driving a pro-angiogenic phenotype that signals to wound fibroblasts via VEGF. The cells are seeded on a hydrogel and laid on the wound bed. The workflow extends to other wound-repair genes.

Unmet need

In **chronic diabetic wounds**, standard care does not correct the impaired angiogenesis that keeps the wound open. Autografting adds donor-site morbidity; allogeneic products carry rejection risk.

Advantages

- Closure 5 days faster than control DCs in diabetic mice (day 16 vs 21)
- Autologous cells from blood: no donor site, no rejection, no surgery
- Regenerative: greater CD31+ vessel density and dermal thickness
- Non-viral ex vivo editing, topical delivery, no injections or grafting

Applications

Lead: **chronic diabetic wounds**, in vivo validated.

Exploratory: surgical wounds; venous and pressure ulcers; burns.

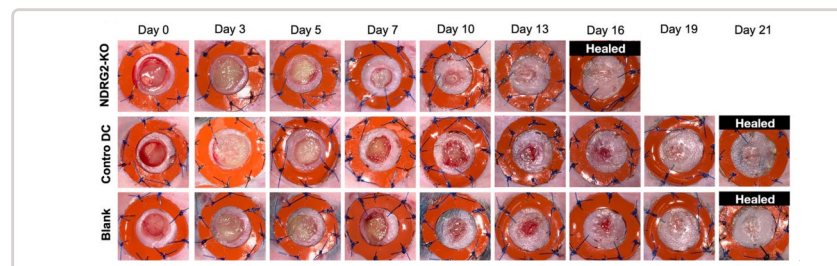


Fig 1. Excisional wounds in diabetic mice. Ndr2-KO dendritic cells in hydrogel reached full re-epithelialization by day 16, 5 days ahead of control DCs and blank hydrogel. ([Henn et al., Nat Commun 2023](#))

DEVELOPMENT STAGE

Discovery In vitro **In vivo** IND-enabling Clinical

Latest: in vivo (diabetic mouse): closure 5 days earlier than control DCs; significantly increased CD31+ vessel density ($P < 0.05$).



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