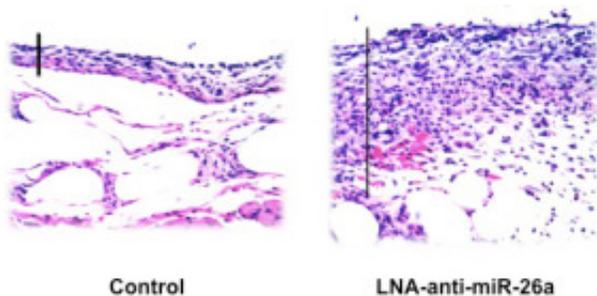


Silencing a microRNA may keep the doctor away for diabetic wounds

Patients with diabetes are frequently afflicted with impaired wound healing that may progress into chronic, diabetic ulcers, often leading to complications including limb amputation with increased risk of morbidity and mortality. These ulcers occur not only in the later stages of diabetes but are also seen in newly diagnosed patients. Indeed, the diabetic foot is the most common cause of non-traumatic lower extremity amputations in the US.



Local delivery of inhibitors to microRNA-26a accelerates diabetic wound healing and granulation tissue thickness by increasing angiogenesis.

Accumulating studies demonstrate that angiogenesis, or the formation of blood vessels from pre-existing ones, may promote wound healing. However, angiogenesis is impaired in diabetic patients and the mechanisms controlling this process are not fully understood.

In this paper, we identify a unique role for a small non-coding RNA, termed microRNA-26a (miR-26a), in regulating the development of diabetic wound healing. Specifically, our findings indicate that miR-26a expression is increased in wounds of diabetic mice. Therapeutic silencing of miR-26a by local intra-dermal delivery effectively promoted wound closure and was associated with favorable phenotype features including increased granulation tissues, induction of Smad1 signaling in endothelial cells, and markedly increased angiogenesis. Interestingly, local silencing of miR-26a had no effect on dermal leukocyte inflammation, highlighting the importance of blood vessel growth for diabetic wound closure.

Collectively, these findings indicate that therapeutic inhibition of miR-26a may provide a promising treatment for diabetic subjects by restoring angiogenesis for dermal wound healing, and provide new therapeutic targets and strategies to ameliorate complications from diabetic wounds.

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