functional significance of this finding remains to be determined.

One caveat of the current study is that lymph and blood flow, as well as nervous connections to the lymph node, were severed, which might have had unpredictable effects on lymphocyte activity. However, in the near future, intravital imaging of lymphoid tissues might be possible. The ability to follow immune cells in living tissues over time and in three dimensions, combined with currently available fluorescence-based assays of lymphocyte function, will make this a powerful new tool for immunologists.

Jennifer Bell

References and links

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WEB SITES

Michael Cahalan's lab: http://crt.biomol.uci.edu

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γδ T CELLS

Stressed skin

The skin is the largest organ of the human body, and owing to its unique interface with the external environment, it is highly susceptible to injury. The process of wound repair is complex and poorly understood, but in a recent paper in *Science*, Jameson *et al.* propose that dendritic epidermal T cells (DETCs) that bear a γδ T-cell receptor (TCR) might have an important role.

In contrast to most peripheral T-cell populations, a large proportion of those in epithelial tissues express γδ TCRs, rather than αβ TCRs. In particular, DETCs express the Vγ3Vδ1 TCR, which recognizes antigens expressed by stressed keratinocytes. The functional importance of the interaction between DETCs and keratinocytes was tested in a mouse model of wound healing, and confirmed in skin organ culture (SOC) assays.

After full-thickness wounding of mouse back skin, TCRδ$^{-/-}$ mice, which lack γδ T cells, had a 2–3-day delay in wound closure and reduced epithelial hyperthickening compared with wild-type C57BL/6 mice. Cell proliferation was also reduced in the wounds of TCRδ$^{-/-}$ mice compared with wild-type mice, which indicates that γδ T cells in the skin have a role in the proliferation of keratinocytes that leads to re-epithelialization and, hence, wound repair.

How might DETCs influence the repair process? In a PCR assay for keratinocyte growth factors (KGFs), fibroblast growth factor-7 (FGF7) messenger RNA could be detected in DETCs from wounded, but not non-wounded, skin, and FGF10 mRNA was detected in DETCs of wounded skin from C57BL/6 and *Fgf7*$^{-/-}$ mice, but not wounded TCRδ$^{-/-}$ mice. These data indicate that γδTCR^{+} DETCs produce KGFs, such as FGF7 and FGF10, in response to wounding.

These results were confirmed by an *in vitro* SOC assay, which showed that it is the absence of KGF-producing DETCs in TCRδ$^{-/-}$ mice that is responsible for the defective wound healing. The addition of FGF7 or activated DETCs to wounded skin from TCRδ$^{-/-}$ mice restored the rate of wound closure and of keratinocyte proliferation to that of skin cultures from wild-type mice.

Given that DETCs are considered to be a prototype intraepithelial γδ T-cell population, is their involvement in wound healing shared by γδ T-cell populations in other epithelial tissues? If this is the case, investigation of their function might be relevant to a wide range of 'epithelial' diseases, such as inflammatory bowel disease and asthma.

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WEB SITE

Wendy Havran's lab: http://www.scripps.edu/imm/havran/