

Cytokine 2000 Jun;12(6):671-6

**Diminished interleukin 6 (IL-6) production during scarless human fetal wound repair.**

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Fetal wound healing is characterized by minimal inflammation and scarless repair. IL-6 stimulates inflammation in postnatal wound healing. We hypothesized that fetal skin has a diminished IL-6 response and that exogenous IL-6 will result in scar formation. Human adult or fetal skin was placed subcutaneously in SCID mice and incisionally wounded.

Wounds were excised after 4, 12, 24 or 72 h for IL-6 mRNA quantification by RT-PCR. In other grafts, 5 microgram of IL-6 was injected at wounding and then harvested at 7 days for analysis of scar formation. IL-6 production was examined in primary cultures of human fetal or adult dermal fibroblasts incubated for 8 h with 0, 0.1, 1 or 10 ng/ml of PDGF-BB. IL-6 mRNA was detected 4 h after wounding in fetal and adult wounds, but by 12 h there was no IL-6 mRNA in the fetal wounds.

Adult wounds had IL-6 mRNA persisting to 72 h. IL-6 administration to fetal wounds resulted in scar formation. Fetal fibroblasts produced less IL-6 protein and mRNA at all points examined ( $P < 0.01$  vs adult). Diminished production of inflammatory cytokines such as IL-6 may be responsible for the lack of inflammation seen during fetal wound healing. Diminished inflammation may provide a permissive environment for scarless wound healing.

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PMID: 10843743 [PubMed]