IMMUNOLOGICAL FEATURES OF THE REPAIR PROCESS

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Currently, many antioxidants, catalase inhibitors, superoxide dismutase and xanthine oxidase inhibitors, as well as a new class of agents called 21-aminosteroids with antioxidant properties, are used for clinical purposes.

T cells also play an important role in the repair process. They accumulate in the area of damage, causing a shift in the helper/suppressor ratio towards T helpers. Monocytes/macrophages activated by T-helper mediators secrete fibrogenic cytokines that cause fibroblast proliferation and collagen synthesis.

Immunological disorders play an important role in the development of hypertrophic scars after burns. These disorders arise as a result of impaired biosynthesis of certain cytokines and lead to massive infiltration of the skin by activated T lymphocytes. These cells, along with macrophages, enhance the expression of IL-2, which actively influences the proliferation and maturation of certain cells.

It has been established that abnormal expression of HLA DR and ICAM-1 (CD-54), as well as CD-36, occurs in keratinocytes and fibroblasts in hypertrophic scars. When studying the cytokine profile of T-cell clones located in the dermis and epidermis of hypertrophic scars, it was found that in the active phase, these scars are heavily infiltrated by Type 0 and Type 1 lymphocytes, which produced large amounts of IFN-gamma and small

amounts of IL-4. CD4+ TCR and CD-8 TCR a/b clones participated equally in the secretion of interferon. In the remission stage of the scar, the level of interferon was 4-6 times lower.

Thus, the systematic study of the molecular mechanisms involved in the restoration of skin lost as a result of burns offers hope for the development of physicochemical treatment methods based on computer modelling and control of the molecular physiology of inflammation and repair as applied to each individual patient.