Repair of damage tissue is a fundamental biological process to restore the barrier function of the skin. Wound healing involves a complex interplay of numerous cell types, modulation of soluble factors, extracellular matrix (ECM), and blood elements. Wound healing goes through a linear series of overlapping events including cell proliferation, migration, ECM deposition, resolution and remodeling. Each phase is dominated by particular cell types, as well as secreted soluble factors (cytokines and chemokines). The healing process is almost never perfect, and often an excessive activity is seen, which can result in formation of a permanent scar, ultimately causing organ failure and even death. Impaired wounds enter a stage of prolonged inflammation and reparative processes leading to excessive connective tissue deposition and scar formation. Such pathological tissue repair not only affects the skin but can also be seen in internal organs. Fibroblasts, the main producer of ECM, are constantly communicating with keratinocytes and inflammatory cells by different cytokines and growth factors to orchestrate the healing process. Fibroblasts are stimulated by the cytokine TGF-β, from e.g. immune cells, to increase deposition of ECM and we hypothesize that keratinocytes secrete other cytokines, such as IL-1α, to counteract this effect. Thus, in this thesis we explored the effects of keratinocytes and IL-1α on gene and protein expression as well as signaling pathways in fibroblasts stimulated by the pro-fibrotic factor TGF-β. Our results add to the understanding of how fibroblasts respond to keratinocytes and soluble factors during tissue repair.