Introduction

In contrast to physiological wound repair non-healing wounds are characterized by an imbalance of the underlying molecular processes resulting in an elongated inflammatory phase and severe tissue damage. As several studies have shown, the exudates of chronic wounds contain elevated levels of inflammatory immune modulators, e.g. the cytokines IL-1β, IL-6 and IL-8, leading to tissue damage and impairing healing [1,2]. Hence, reduction of these mediators is a suitable way to promote normal healing. Studies have shown that collagen is able to bind significant amounts of cytokines [3,4]. We investigated the influence of a collagen-dressing* (figure 1) on the concentration of IL-8 and IL-6 in a cell-based inflammation model.

Results

TNF-α had no significant effect on HaCaT keratinocyte viability or proliferation but lead to a distinct increase in the release of the inflammatory cytokines IL-6 and IL-8 in vitro. The collagen-dressing alone neither had an effect on cell viability and proliferation nor did it induce the expression of IL-6 and IL-8. However, the use of the collagen sample on the TNF-α-stimulated HaCaT-cells led to a significant decrease in the amount of unbound IL-8 in the supernatant (figure 3) and a minor reduction in the concentration of IL-6 (figure 4).

Discussion

Collagen dressings should be able to improve the healing outcome of chronic wounds by decreasing the excessive concentrations of inflammatory mediators. Using a cell-based inflammation model it could be shown that collagen* directly influences the amount of IL-6 and IL-8 released by TNF-α-stimulated HaCaT-cells most likely by binding these mediators as well as acting directly on the TNF-α present by reducing its concentration.

References