

PRESENTATION OF RESULTS OF MOVING GRID FINITE-ELEMENT ANALYSES ON A PLASTIC MECHANOCHEMICAL CONTINUUM MODEL FOR DERMAL WOUND HEALING

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The healing of full-thickness dermal wounds involves a complicated sequence of spatially and temporally coordinated processes that can be classified roughly into four consecutive, partly overlapping phases: haemostasis, inflammation, proliferation, and remodelling [1]. One of the key processes that takes place during the proliferative phase is contraction of the wound. During this process the wound boundaries are drawn inwards by biomechanical mechanisms so that the size of the injury is reduced. This phenomenon is an important and intrinsic feature in the healing process and it is usually beneficial when it is well-balanced. If this balance is disrupted however, then this may cause delayed and / or impaired healing in the case of insufficient contraction, while excessive contraction may induce the development of substantial scarring and contractures (i.e. a permanent shortening of scar tissue that results in deformity). Even though intensive research over the last few decades has produced much knowledge about the biomechanical mechanisms underlying wound contraction and its associated pathologies, there is much that remains understood incompletely about these mechanisms and the etiology of the pathologies that may develop.

In order to gain more insight into the mechanisms underlying wound contraction and the development of contractures, we have developed a new deterministic mechanochemical continuum model. This model allows a detailed evaluation of the effects of strong interactions between changing mechanical structures and some of the most important biological entities involved in the contraction of full-thickness dermal wounds. This evaluation is accomplished by describing the interactions between generic immune cells, (myo-)fibroblasts, two generic types of growth factor, immature and mature extracellular matrix material and a mechanical force balance as a result of the physico-chemical properties of the extracellular matrix and the cell-generated traction / pulling forces. The plastic evolution of

the dermal tissue as a consequence of remodelling of the dermal tissue by (myo-)fibroblasts is incorporated in the model by using the ‘morphoelasticity’ theory developed by Cameron Hall to model biological plasticity [2]. This theory gives a description of how the ‘zero stress state’ of a material (i.e. the configuration of a material such that all residual stresses are relieved) changes in response to remodelling and provides the necessary evolution equations for the elastic strain.

We will present results from moving grid finite-element analyses performed on the model and subsequently draw conclusions about the influence of different processes described by the model, on the contraction of dermal wounds and the development of contractures. Contractures often develop in burn wounds that cover substantial areas and therefore the application of the model to these wounds *in vivo* may suggest strategies for controlling contraction and the development of contractures [3].

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