Pressure ulcer research at the TU/e : how far are we from clinical practice?

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How far are we from clinical practice?

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Introduction

Pressure ulcers are a serious health and financial problem. Prevalence figures are very high: 20% in general hospitals up to 29% in nursing homes. Pressure ulcers can initiate either at the skin layer (superficial ulcers) or within deeper tissues (deep pressure ulcers) after sustained compressive loading. These two types of ulcers have different etiologies, which are both not well understood.

The main problem associated with deep pressure ulcers is their early detection, since they are not visible at the skin layer. For superficial ulcers the assessment of high-risk patients is essential, in order to deliver the appropriate preventive measures to the patients.

Objectives

To understand the mechanisms associated with pressure ulcer development and to develop an early detection method for deep pressure ulcers and a risk-assessment tool for superficial ulcers.

Model systems

The pressure ulcer research at the TU/e is divided into five projects; three focusing on muscle tissue (deep pressure ulcers) and two focusing on skin tissue.

Muscle

Theories explaining the damage to muscle tissue after compressive loading include ischemia, reperfusion damage and sustained deformation of cells. In order to study the separate contributions of these factors two model systems were developed.

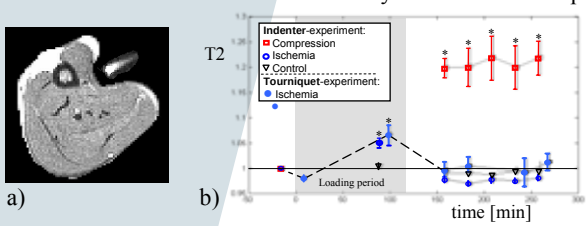

Figure 1. *In-vivo animal model (a) to study the separate and combined effect of deformation and ischemia (b). (Stekelenburg)*

An *in-vivo* rat model was used in which the tibialis anterior was loaded with an indenter (fig 1). Tissue status was measured before, during and after loading using several MRI techniques.

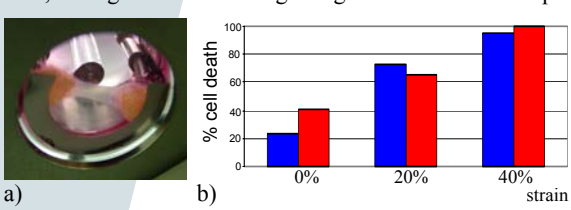

Figure 2. *In-vitro tissue-engineered muscle model (a) to study the separate and combined effect of deformation and hypoxia (b) in a controlled manner. (Gawlitta)*

An *in-vitro* model was used to study the different effects of ischemia (e.g. hypoxia, acidification) and deformation in more detail using fluorescence microscopy (fig 2). Results from both models demonstrated that cellular deformation played a more important role in early damage development compared to ischemia, contrary to the most adhered hypothesis that ischemia is the main trigger. To understand the role of ischemia and deformation and, in addition, of changing tissue properties a numerical model is developed (fig 3).

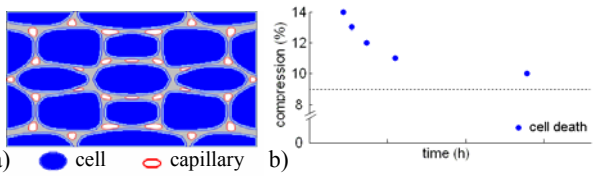

Figure 3. *Numerical model of muscle cells (a) and the effect of compression on cell death (b). (Ceelen)*

This model can provide information on changing parameters which cannot be obtained from *in-vivo* or *in-vitro* experiments.

Skin

For the development of an objective risk assessment tool the release of damage markers, which can be measured at the skin surface, is being investigated using an *in-vitro* skin model (fig 4).

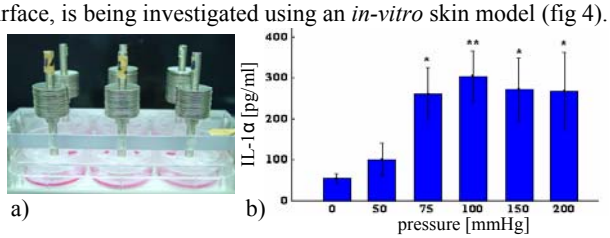

Figure 4. *In-vitro skin model (a) to study the effect of deformation on release of damage marker IL-1α (b). (Bronneberg)*

An increase of interleukin-1α (IL-1α) was measured in the surrounding medium after compressive loading. To better understand the distribution and transport of IL-1α in the skin-sample a numerical diffusion model is developed (fig 5).

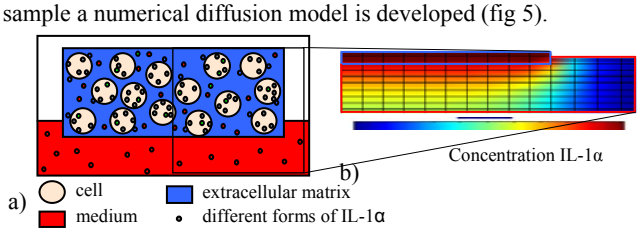

Figure 5. *Schematic of distribution of IL-1α in sample (a) and diffusion from sample to surrounding medium (b). (Cornelissen)*

Conclusion

Outcomes of our research on deep pressure ulcers will be used in the development of new European guidelines for treatment. The feasibility of an early detection method for deep ulcers, based on the measurement of damage markers in blood, is currently being investigated. A clinical study is being prepared to test cytokine (e.g IL-1α) release in patients with and without skin damage.