IL-1α as an early marker for superficial pressure ulcers

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D. Bronneberg, C.V.C. Bouten, C.W.J. Oomens, P.M. v. Kemenade, F.P.T. Baaijens

Department of Biomedical Engineering, Eindhoven University of Technology, The Netherlands.

Introduction

Pressure ulcers are localized areas of degenerating tissue in skin and underlying tissue resulting from prolonged mechanical loading. Superficial pressure ulcers initiate in the skin and can occur as a consequence of sustained compression. For effective prevention of these ulcers it is essential to identify a discriminative biological marker that signals early tissue damage before the onset of irreversible tissue breakdown. Il-1α is widely accepted as a marker for screening the damaging effects of chemical irritants [1,2,3]. The present study evaluates the potential of IL-1α as a marker for compression-induced skin damage using a commercially available *in vitro* engineered epidermal model.

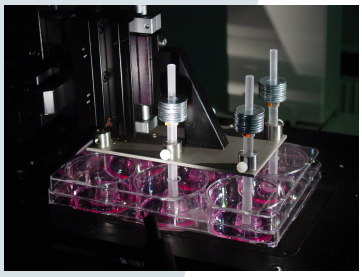
Material and methods

Skin model

The EpiDerm^{TM} (MatTek, Ashland, USA) model is used in this study. This epidermal model consists of normal, human-derived epidermal keratinocytes (NHEK), which have been cultured to form a multilayered, highly differentiated model of the human epidermis. Ultrastructurally, the EpiDerm^{TM} skin model closely parallels human skin *in vivo*.

Compression

Clinically relevant pressures of 6.7 and 13.3 kPa were topically applied to EpiDerm^{TM} samples (d=8 mm) in a compression device using indenters (d=5 mm) and additional weights for a period of 2 hours at 37 °C and 5% CO_2 (figure 1). After loading, the samples were incubated for another 3 hours. Unloaded samples and samples loaded with a disc (d=5 mm) of negligible weight were used as control.



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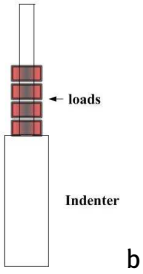

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Figure 1. *An image of the compression device with EpiDerm^{TM} samples (a), and of the indenter and the additional weights (b).*

Marker analysis

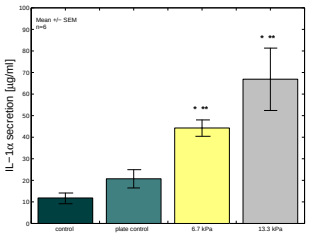
After the compression experiment, medium underneath the EpiDerm^{TM} samples was collected from each well and stored at -20 °C for extracellular IL-1α analysis. The IL-1α present in the medium was measured by means of a commercially available enzyme-linked immunosorbent assay kit (Quantikine, R&D systems, Uithoorn, NL).

Damage assessment

The tissue viability of the EpiDerm^{TM} samples was examined by a MTT assay (MatTek, Ashland, USA), and the tissue morphology was examined histologically (H&E).

Results

Topical pressure application resulted in a significant increase in the IL-1α secretion (figure 2a). It even seems that high pressure application (13.3 kPa) results in a higher secretion of IL-1α compared to low pressure application (6.7 kPa). Furthermore, no tissue damage was observed in the EpiDerm^{TM} samples (figure 2a and 3) upon compression.



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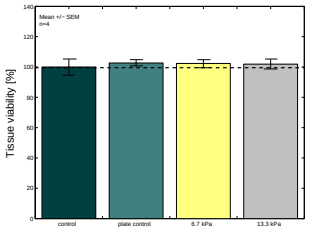

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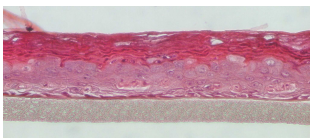
Figure 2. *The IL-1α secretion (a) and the normalized tissue viability (b) of the studied experimental groups. *, p < 0.05 versus control; **, p < 0.05 versus plate control.*



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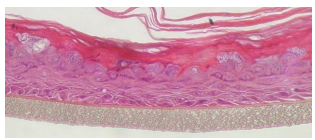

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Figure 3. *Histological appearance of a control sample (a) and of a sample exposed to high pressure (13.3 kPa) (b). The tissue morphology of the other experimental groups are similar to the displayed histological sections.*

Discussion

These results indicate that IL-1α is a promising marker for compression-induced skin damage before the onset of irreversible tissue breakdown and motivate further investigation to evaluate its potential in pressure ulcer prevention. Future research will focus on evaluating other damage markers (e.g. pro-inflammatory mediators) to increase the understanding of the pressure ulcer etiology.

References:

[1] Gibbs, S., *et al*: *Exp Dermatol* 2002; 11: 217-223

[2] Muller-Decker, K., *et al*: *Toxicol Appl Pharmacol* 1994; 127: 99-108

[3] Corsini, E., *et al*: *Toxicol Appl Pharmacol* 1996; 138: 268-274