The effect of Microcarrier-delivered Keratinocytes and Single-stage Matriderm on Wound Contraction and Epithelialisation

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Introduction

Delayed burn wound healing is associated with scarring and contracture formation which may result in functional disability and psychological distress for patients. Specialist long-term management also has significant healthcare costs. The application of sprayed autologous keratinocytes (SAK) to accelerate burn wound healing is well established. The use of gelatin microcarrier beads (Cultisphere G) to culture and deliver autologous keratinocytes (AK) to a wound bed negates the use of murine feeder cells or proteolytic enzymes which may temporarily inhibit cell proliferation, attachment and migration. The aim of this pre-clinical study was to determine the effect of microcarrier-delivered keratinocytes and Matriderm (dermal regeneration template) on wound contraction and epithelialisation.

Methods

Eighteen full-thickness wounds were created in a porcine model of wound repair. Six control wounds were treated with single-stage Matriderm and a widely meshed split skin graft (1:6). The remaining wounds were treated with Matriderm and either AK cultured on Cultisphere G microcarriers (n=6) or SAK (n=6). The study was terminated and the wounds were excised at 21 days. Histological analyses and digital photograph analyses of all wounds were performed.

Results

All wounds showed similar rates of reduction in contraction. Control wounds demonstrated neo-epithelialisation over the entire wound surface at 21 days. Wounds treated with microcarrier-delivered keratinocytes or SAK showed a central area without overlying epithelium surrounded by neo-epithelium on all sides. This distinct pattern indicates that neo-epithelium has been formed as a result of epithelial migration from the wound edges.

Conclusion

AK delivery on microcarriers or as a spray, in conjunction with Matriderm alone, shows potential in minimising wound contraction in vivo. However, AK delivery on microcarriers or as a spray, in conjunction with Matriderm alone, is incapable of wound epithelialisation which may be explained by the lack of application of autologous fibroblasts in the treatment wounds.