

ESCHAREX PROMOTES RAPID DEBRIDEMENT AND MODULATES WOUND HEALING-RELATED GENE EXPRESSION IN A PORCINE CHRONIC WOUND MODEL



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Overview

INTRODUCTION

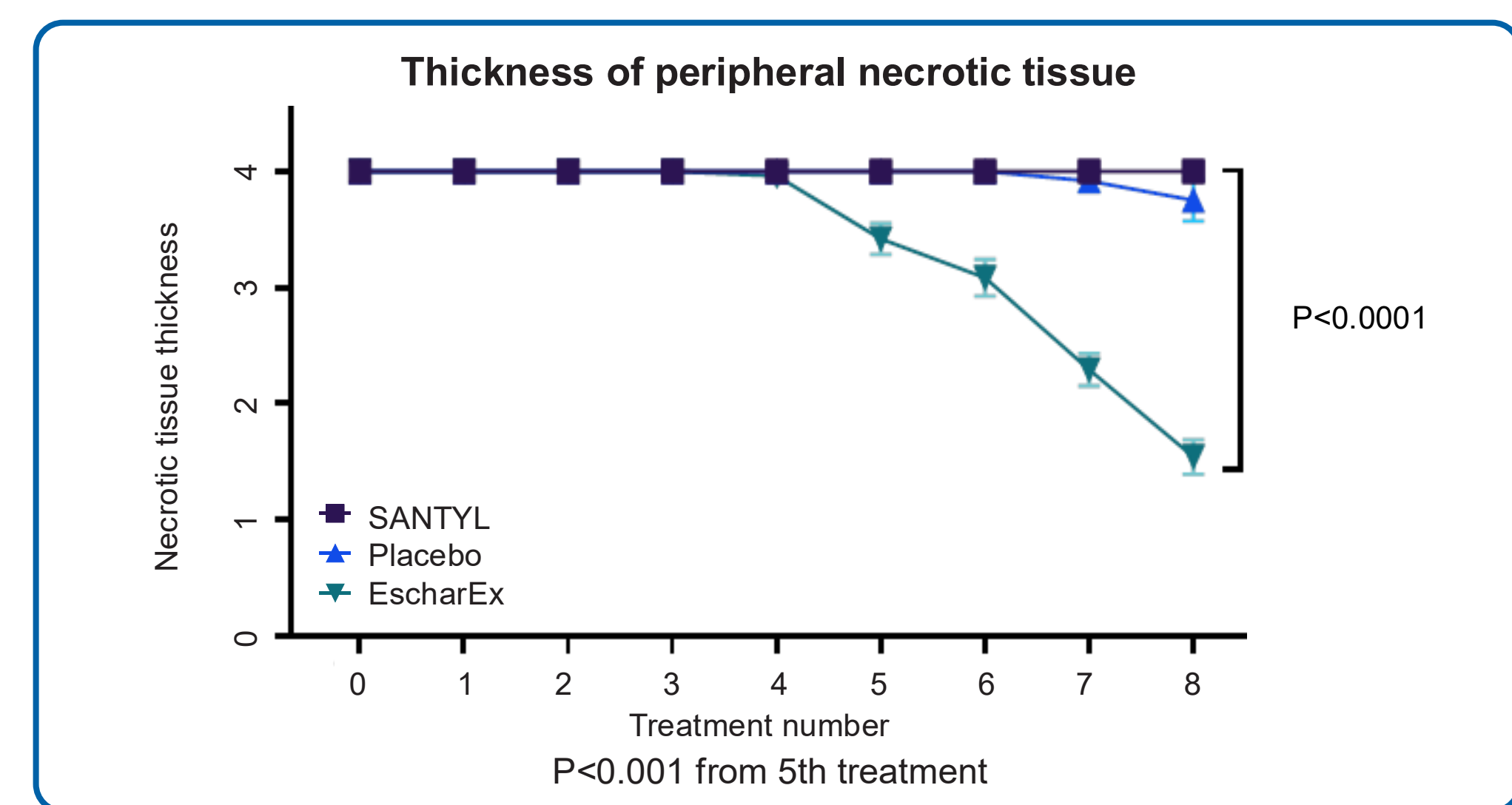
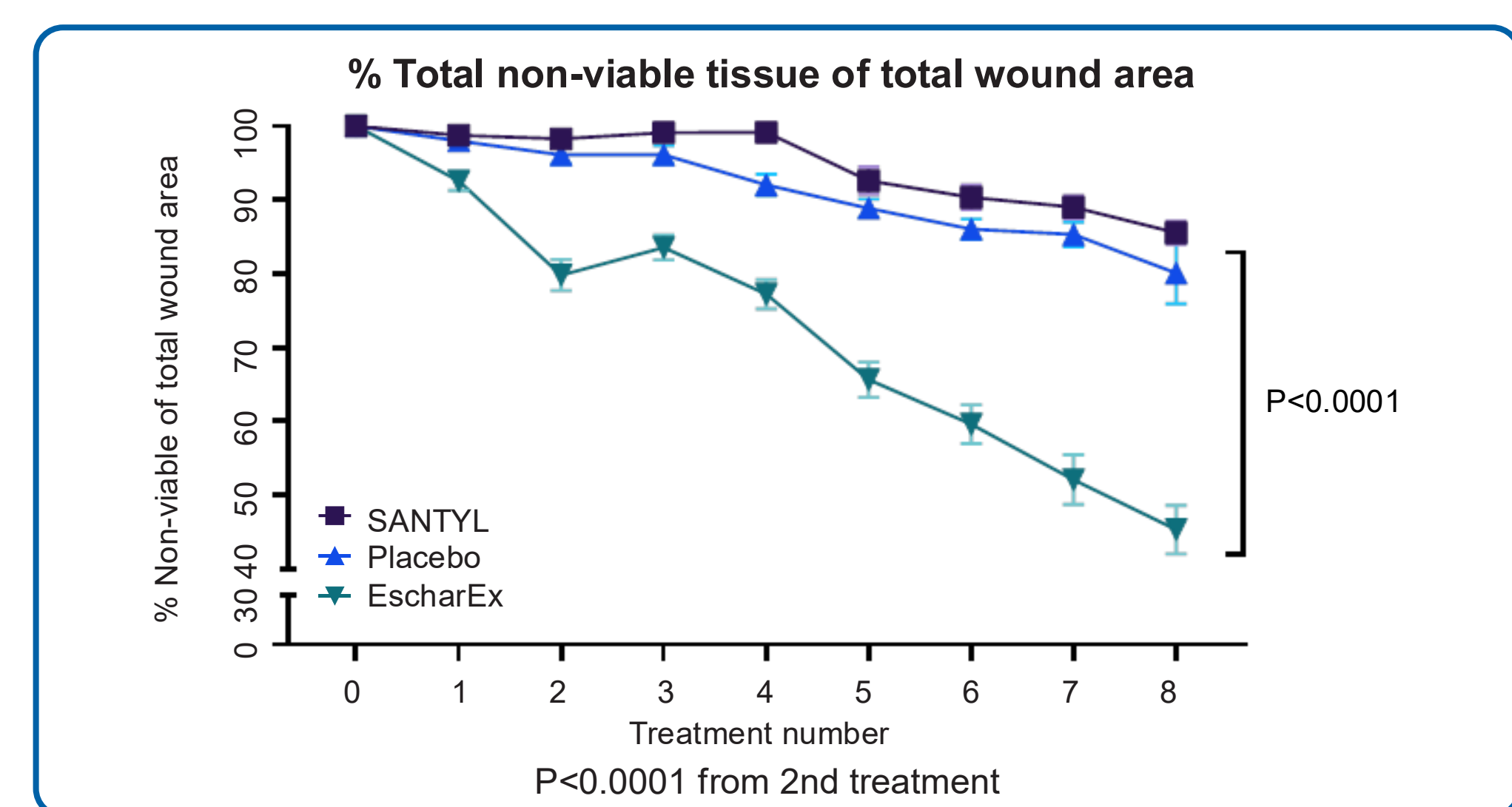
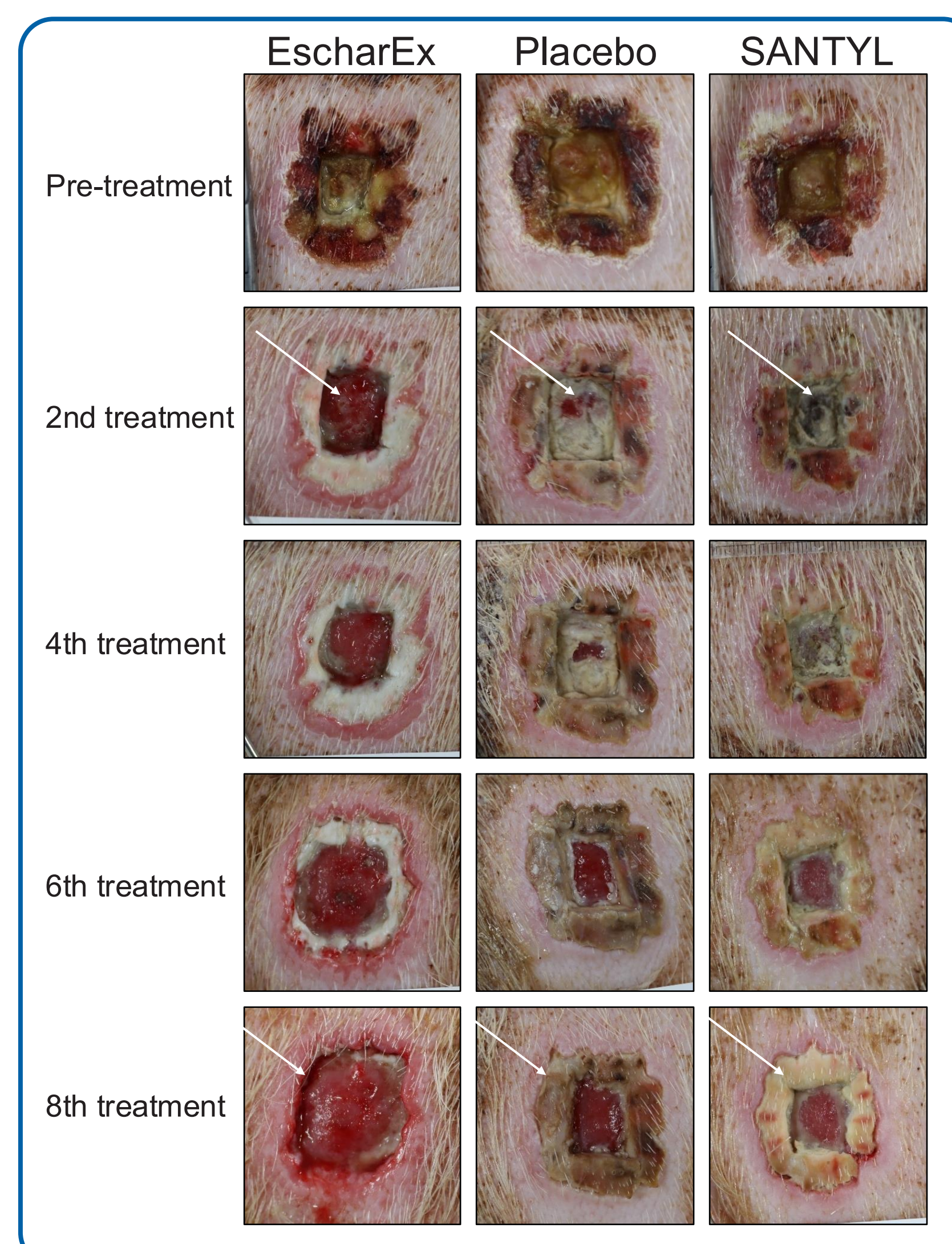
Effective debridement is a critical prerequisite for timely wound healing. Collagenase-based enzymatic debridement is widely used but is often associated with gradual and slow non-viable tissue removal, which may delay wound closure. EscharEx[®] is a novel bromelain-based enzymatic debridement agent developed to accelerate the breakdown of non-viable tissue while promoting a wound environment conducive to healing. In this study, we explored debridement efficacy and the molecular mode of action of EscharEx compared with placebo and SANTYL in a porcine chronic wound model.

METHODS

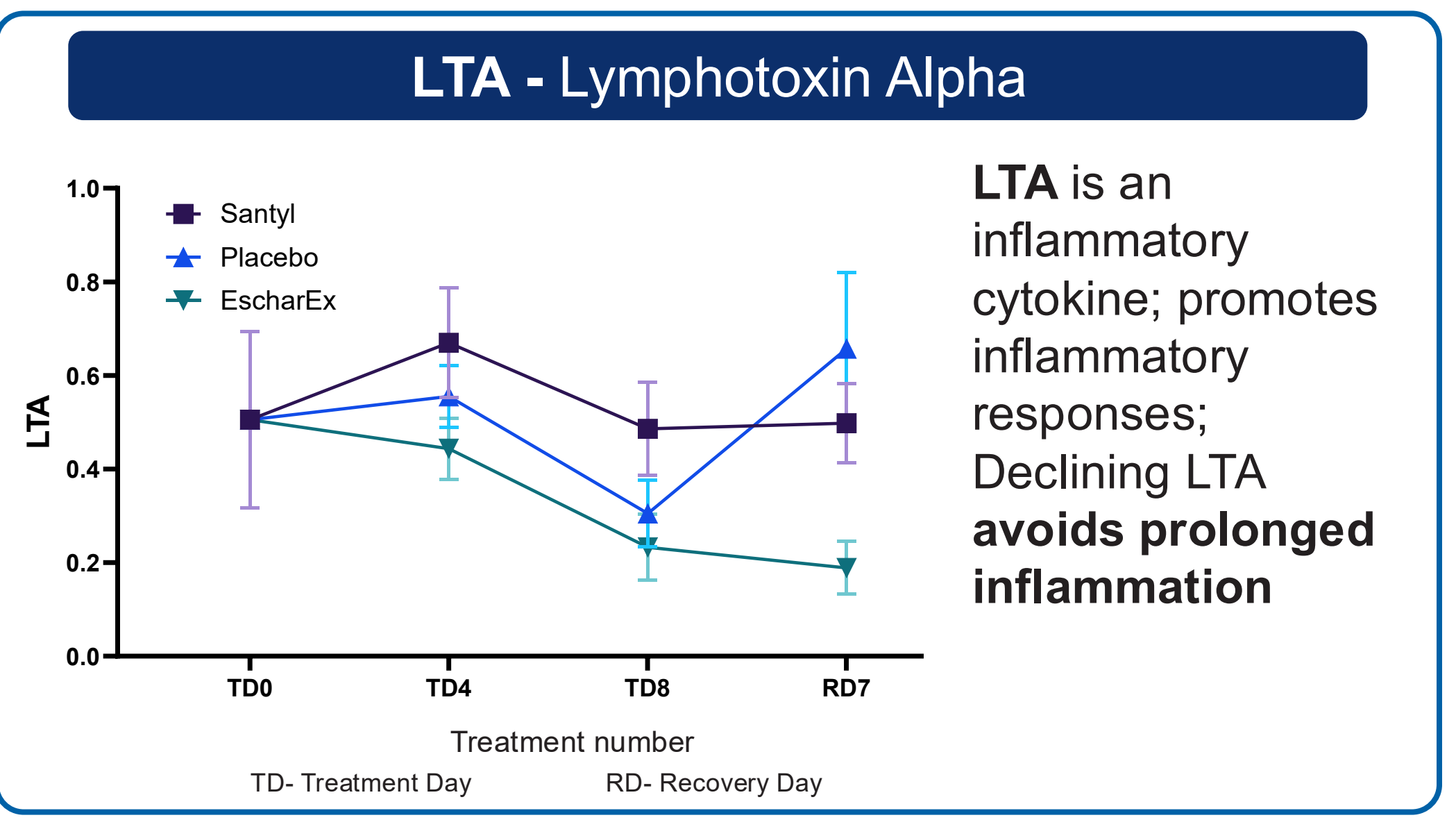
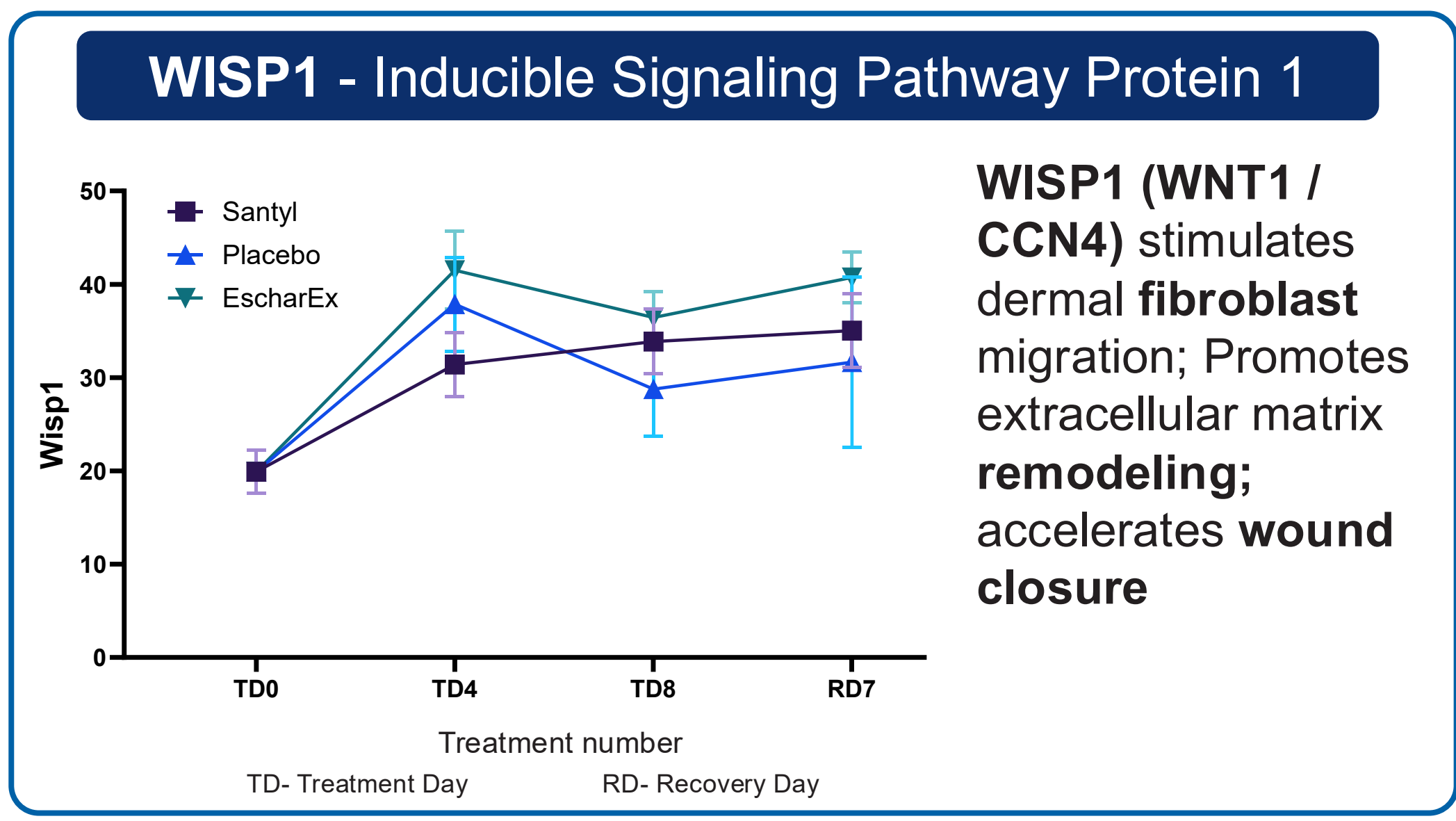
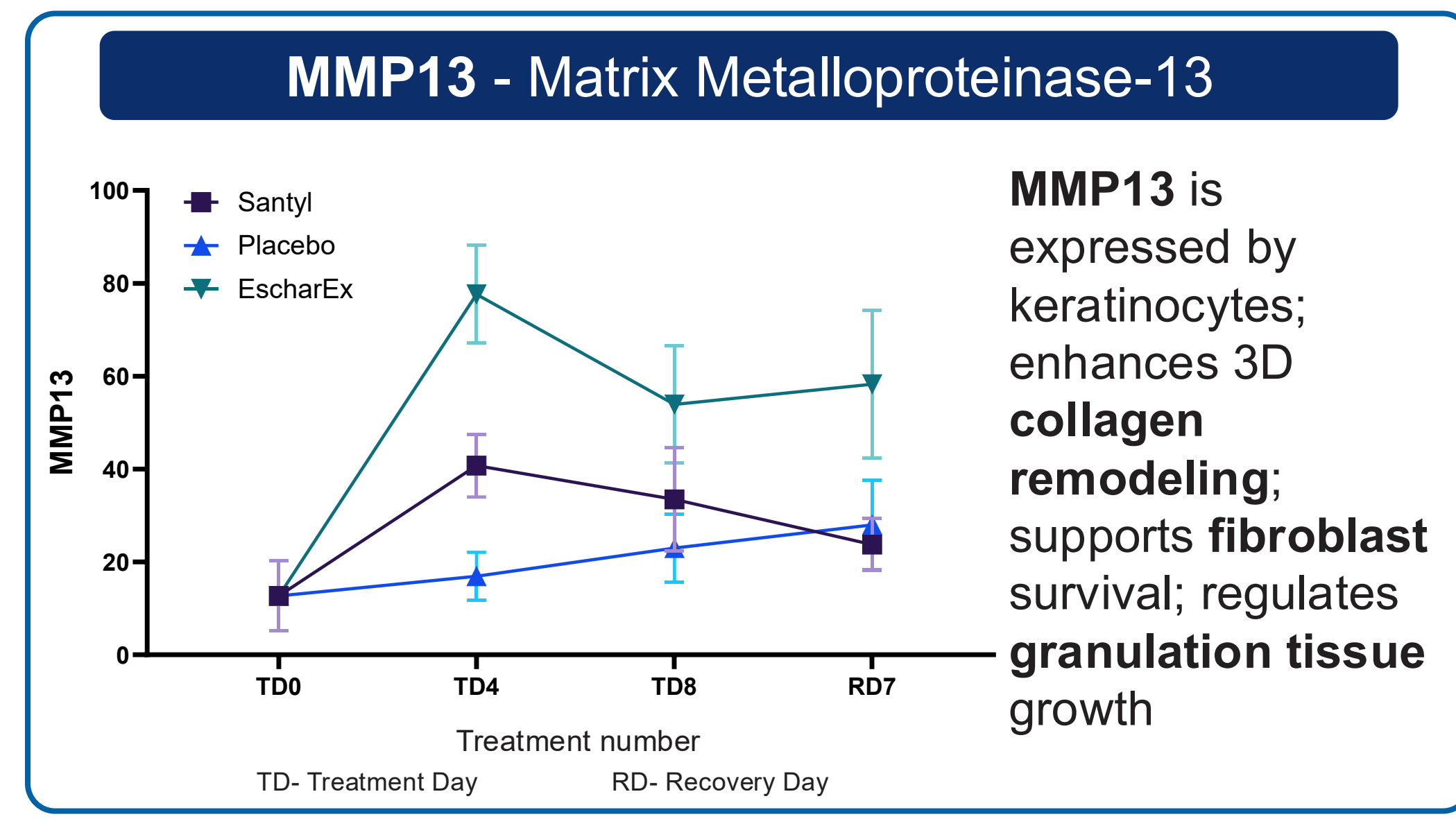
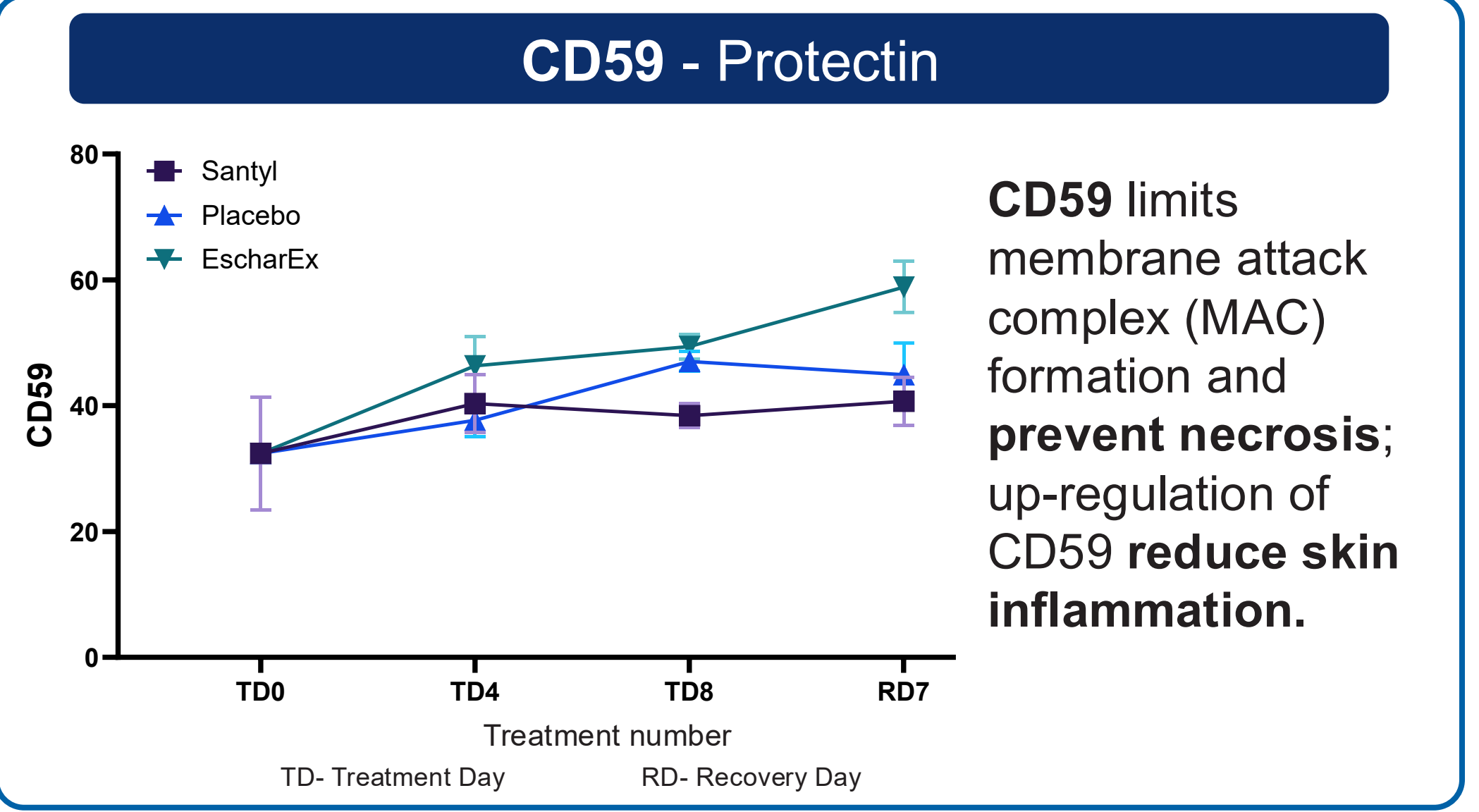
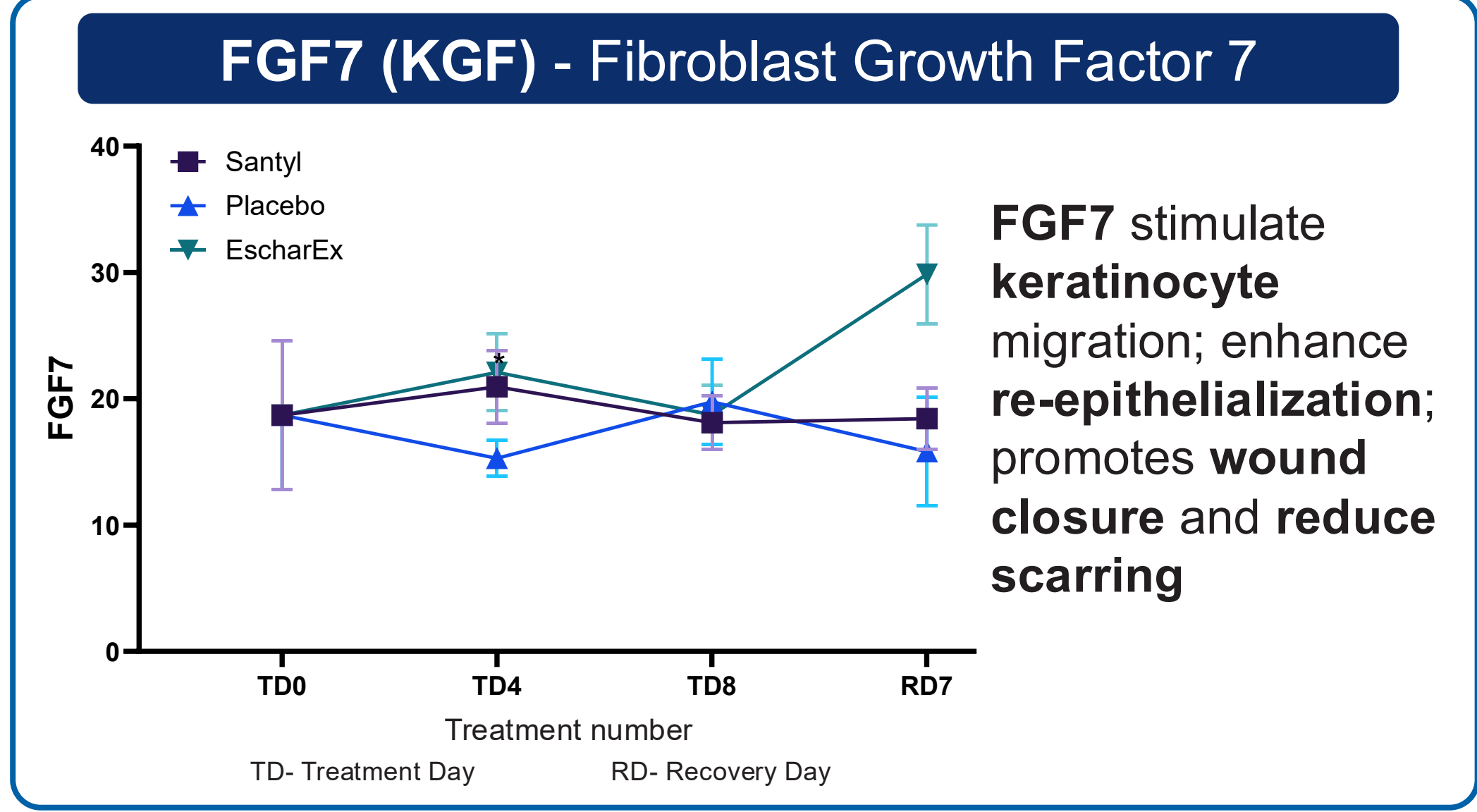
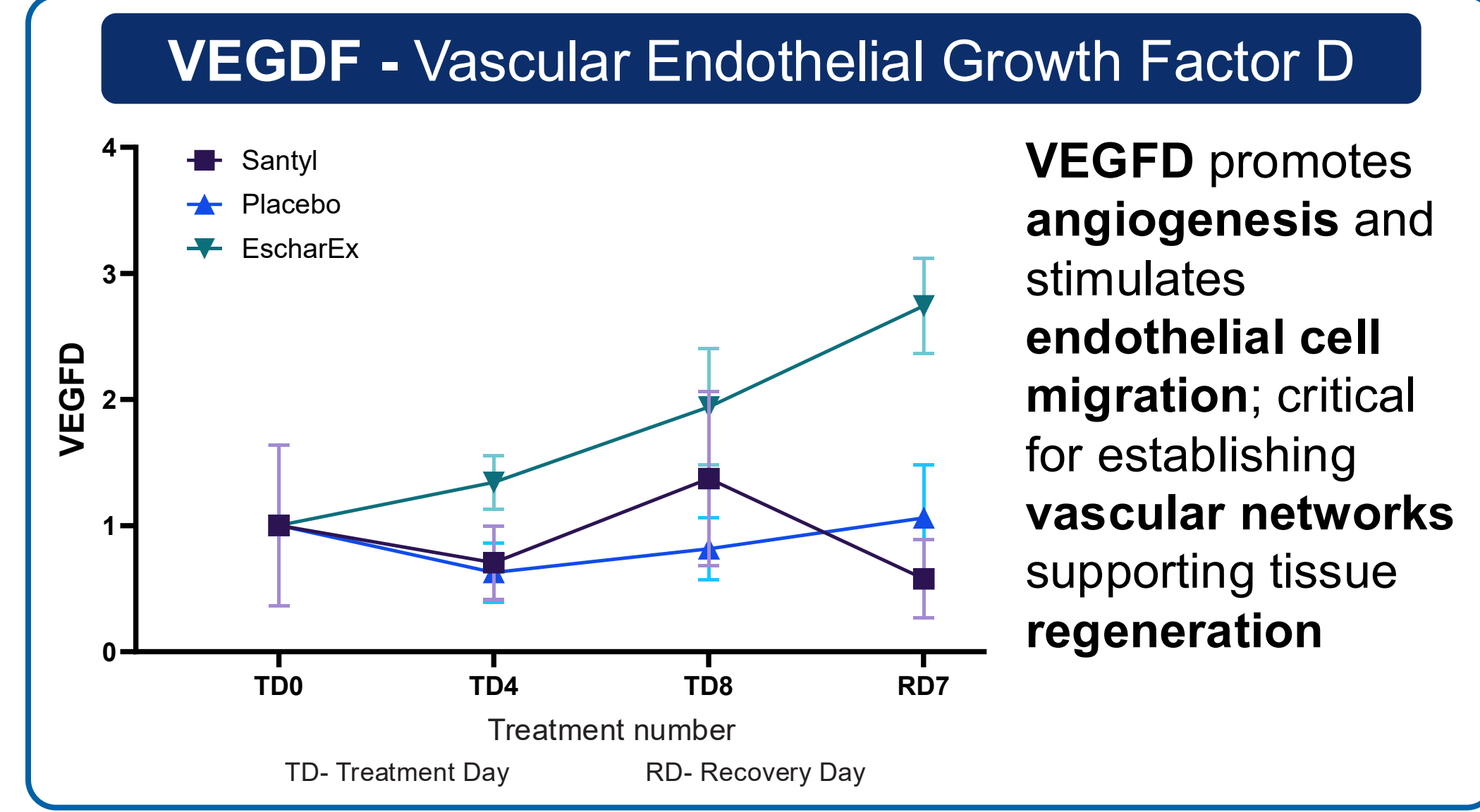
Twelve full-thickness hard-to-heal wounds were created on the dorsal side of 4 pigs by skin excision combined with doxorubicin injection on days 1 and 6 post-wounding. The wound bed was characterized by a distinct central slough and peripheral full-thickness necrotic skin. Twenty days after model establishment, wounds were treated with EscharEx, SANTYL, or gel vehicle (placebo control). Treatments were applied once daily for 8 days. Debridement progression was assessed using blinded clinical scoring, quantitative image analysis, and histological evaluation. Serial wound biopsies were collected at multiple time points and analyzed by RNA sequencing.

Debridement Results

In the chronic wound model, peripheral non-viable tissue was generated by intradermal injection of doxorubicin and consisted of full-thickness necrotic skin. In the center of the wound, all skin layers were removed, resulting in slough formation adherent to the wound bed. EscharEx removed both slough and full-thickness necrotic skin significantly more rapidly than placebo and SANTYL.



Modulation of Wound Healing



Conclusions

EscharEx demonstrated:

- **Superior debridement:** Effective removal of various types of non-viable tissue commonly found across different chronic wound etiologies, outperforming SANTYL and placebo
- **Modulation of key biological pathways supporting wound healing** evident by gene expression changes associated with:
 - Necrosis prevention
 - Angiogenesis and granulation tissue formation
 - Migration and survival of fibroblasts, keratinocytes and endothelial cells
 - Modulation of inflammation
 - Remodeling of collagen and extracellular matrix