Introduction

Sulfur Mustard (SM) is a vesicant (blistering) agent that was widely used in WWII, and was more recently used in Iraq by Saddam Hussein. The mechanisms by which sulfur mustard creates injury are thought to involve proteases (and likely other inflammatory agents), which illustrates significant similarity in the hemostasis of chemical and thermal burns.

Experimental dressing

Experimental dressing. Previous research (reported at previous WHS meetings including 2006 and 2007) described the NIMBUS-SAP dressing system (Quick-Med Technology, Inc), characterized by laboratory assays. The dressing system was shown to have broad antimicrobial activity as tested per ATCC method 100-1999. Additionally, protease inhibition at high levels was demonstrated using Accucase and TNO-212 assays.

Evaluation of experimental dressings. Systemic FT (cell in use) and/or topical antimicrobial/antiseptic treatment with various topical dressings (Afshar, MD, and others) skin models demonstrated the dramatic effects to reduce wound size, proteinase activity, and collagen levels, and to enhance wound healing.

Results

Injury Model. In order to assess wound healing, a reproducible injury model was devised. Figure 3 shows the desanguination of cell proliferation generated by the two final candidate exposure amounts — either 20 or 10 μl of 40 or 40 μl of 20 μm of CEsS (2-chloroethylethyl sulfide), applied onto a Whatman244 filter disk that was placed atop the live well (initial tissue construct). The larger dose was selected because it achieved an injury that more accurately reproduced the basement membrane detachment characteristic of SM injury, although cell proliferation measured by MTS assay on the same average result for both exposures. Figure 4 shows histology for uninjured vs. exposed tissue constructs.

Testing details

Treatment groups assessed were as follows:

- Treated with NIMBUS-SAP with Doxy (doxycycline and vitamins)
- Treated with NIMBUS-SAP with doxy and EGF (epidermal growth factor)
- Treated with NIMBUS-SAP with doxy and EGF (vitamins)
- Treated with NIMBUS-SAP with doxy and EGF treatment solution, in which the concentration of active agents was 1% of EGF, 500 μg/ml doxycycline and 100μg/ml each of vitamin C and vitamin E analogs.

Cellular Proliferation, per MTS assay demonstrated that CEES exposure has caused no decrease in the metabolic activity of the exposed uninjured control tissues (Figure 5). A statistically significant upregulation of 10-15% was shown between exposed control and both exposed treated groups (DOXY+EGF and DOXY). No significant difference was noted between the three treated groups (DOXY+EGF vs. DOXY). These results have warranted progress to an animal exposure model to substantiate these results in vivo. Animal tests involve a well-characterized weanling pig model, developed by and tested at Battelle Memorial Institute.

Conclusions

The results presented showed that the NIMBUS-SAP antimicrobial dressing system significantly improved healing during the 3 day exposure period tested in vitro using Epiderm EFT-400 tissue model. Promounced vascularization due to the CEES injury, which manifested as epidermal-dermal separation, was mitigated by application of NIMBUS-SAP treatments. These results have warranted a further animal exposure model to substantiate these results in vivo. Animal tests involve a well-characterized weanling pig model, developed by and tested at Battelle Memorial Institute.

References


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