Using hydration response technology dressings in bacteria management

KEY WORDS

- Wound dressing
- Bacteria
- Sequestration
- Wound bioburden
- Hydration response

Wound dressings that are used on infected wounds, or on individuals at risk of infection, should provide a physical barrier preventing microbial ingress. If a medicated dressing is used then broad spectrum antimicrobial activity, and avoidance of cytotoxicity to newly formed tissue cells or periwound skin is important. Furthermore, the dressing should promote a moist wound environment that supports the healing process. Passive mechanisms for the management of wound bioburden include binding or sequestering bacteria into the dressing material.

In this article the authors outline how they investigated the bacterial sequestration and retention capability of a dressing delivering hydration response technology (HRT) (Sorbion Sachet S, Aktiengesellschaft [AG]). The HRT dressing and three competitor test dressings were exposed to *Staphylococcus aureus* or *Pseudomonas aeruginosa*, *in vitro*, over seven days. Bacterial growth resulting from the dressing surface was observed on a broad spectrum growth medium. In a separate experiment, Scanning electron microscopy (SEM) was used to visualise bacteria on the HRT dressing contact surface and within the HRT dressing core.

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Typically, a variety of dressings are used to manage wounds as they progress through the various stages of healing. The basis for dressing choice is usually the provision of an environment that is most conducive to the healing process, relative to the stage of healing. In chronic wounds, a dominant criterion is often the absorption characteristics of a dressing.

However, it should not be forgotten that although efficient absorption characteristics are of vital importance in moderate-to-heavily exuding wounds, ancillary characteristics also have a valuable role to play in support of the healing process. In this article, the authors report on the ability of four test dressings to manage the inflammatory characteristics of a chronic wound through the process of bacterial sequestration.

CHRONIC WOUNDS

A chronic wound is one that does not progress sequentially through the stages of healing within the anticipated time frame (Hermans and Treadwell, 2010). A wound can stall at any stage throughout the healing process, but commonly, a chronic wound is unable to progress past the inflammatory phase.

Chronic inflammation resulting from persistent infection can cause elevated levels of exudate, which act as a transport medium for pro-inflammatory cytokines, proteases and neutrophils

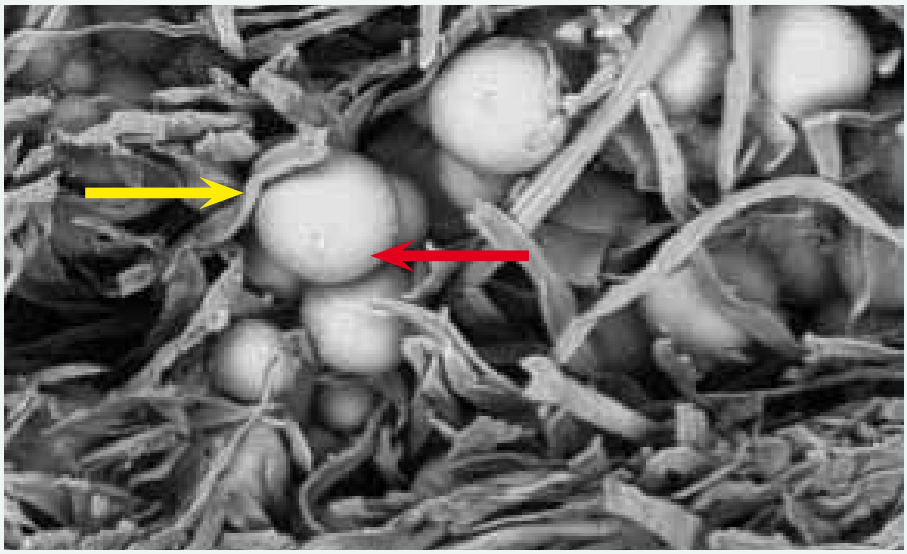

Figure 1: A scanning electron micrograph (SEM) image showing the inner core of a HRT dressing. The red arrow represents the gelling agent, while the yellow arrow represents cellulose fibre.

(Wolcott et al, 2008; Moore, 2010). Excessive levels of proteases can hinder wound closure by causing maceration (Nelson, 1997) and excoriation (Vowden and Vowden, 2003).

In addition to harbouring a high bacterial burden, exudate may also consist of bacteria-produced biofilm material, both of which prevent or delay wound healing and have been linked to malodour (Bowler et al, 1999a). The production of wound exudate can vary in terms of the quantity, viscosity and clarity between different wound types, but also at different stages of healing.

MICROBIAL SEQUESTRATION

Microbial sequestration is the ability of a wound dressing to 'bind' and immobilise wound exudate and microorganisms. The risk of clinical infection is reduced if bacterial sequestration is of a magnitude that maintains a wound bioburden 'balance' in favour of the host. Wound dressings may need to be changed less frequently when they demonstrate a strong and prolonged ability to sequester microorganisms. By sequestering microorganisms, the dressing locks them away from the wound bed, thus lowering the bacterial burden and, therefore, encouraging healing (Newman et al, 2006).

Different dressing structures vary in their ability to sequester and retain microorganisms (Bowler et al, 1999b; Tachi et al, 2004). One study found a vast improvement in an infected pressure ulcer that was seen following the application of HRT — previously, the wound had failed to respond to systemic and topical applications of antimicrobials (Evans, 2010). Previous evaluations have shown that the use of HRT to manage exudate resulted in reduced periwound maceration (Cutting, 2009).

HRT

An innovative wound dressing utilising HRT exploits the interactive response of two components — mechanically modified cellulose fibres and selected gelling agents (*Figure 1*), contained within an outer polypropylene cover. This technology is designed to interact with the wound environment, managing wound exudate while concurrently avoiding desiccation of the wound bed, or saturation of the periwound skin (Sharp, 2010).

An appropriately hydrated wound supports keratinocyte migration, re-epithelialisation and, finally, wound closure. HRT remains effective under compression equal to standard sub-bandage pressure (40mmHg) (Kwon-Lee, 2010).

A study reported on 20 chronic non-healing wounds that were treated with HRT dressings and found that dressings

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stayed in situ without slipping, were easy to remove, acceptable to the patients, comfortable to wear and would be chosen by the clinicians for future use (Ling, 2011). The dressing also reduced malodour, debrided the wound, decreased infection and encouraged healing (as determined by a decrease in wound area). In particular, it is claimed that HRT can sequester and retain microorganisms within the dressing. In support of these dressing performance characteristics, Kwon-Lee (2010) has stated that HRT dressings remain effective when used in association with compression therapy.

This paper provides the first report of an in vitro study that directly examined the sequestration and retention of bacteria within a HRT dressing.

The objectives of this study were to:

- Demonstrate the ability of HRT to sequester *S. aureus* and *P. aeruginosa*
- Compare the ability of HRT and the three test dressings to sequester and retain microorganisms
- Visualise *P. aeruginosa* within a HRT dressing.

METHODS

Experiment 1

Test dressings comprised:

- HRT dressing
- Test dressing 1 — superabsorbent dressing with a non-woven contact layer
- Test dressing 2 - superabsorbent polymer particle dressing with a polyethylene contact layer
- Gauze dressing.

Bacteria were cultured into Solution A (142mmol NaCl, 2.5mmol CaCl) to a concentration of 2.0x106 cells/ml. Over the seven-day test period, a total volume of 105ml of the inoculum was added to each dressing. Twelve wound dressings were placed into trays containing 15ml of inoculated Solution A and the trays were incubated at 37°C for 24 hours. Trays were re-infected daily with 15ml of freshly inoculated Solution A.

Sampling

Samples were collected on days 1, 3, 5, and 7 (N=3). After 24 hours incubation, three dressings were removed from the initial inoculation trays and transferred to sterile agar plates using sterile tweezers. Dressings were left in place on the agar, covered and incubated for 24 hours. After incubation, dressings were removed from the agar and discarded. The agar plates were re-incubated for a further 24 hours.

After the final incubation, agar plates were visualised and photographed to show where bacteria had grown underneath and surrounding the dressing. When no growth was visible, the agar plate was re-inoculated for a further 24 hours in order to demonstrate that no active agent was released from the dressing. The experiment was repeated with the four test dressings.

Experiment 2

A working culture of *P. aeruginosa* was prepared in tryptic soy broth (TSB) to a concentration of 2.0x106 cells/ml. Seventy-five ml of inoculated TSB were added to six 10x10cm^2 trays and 75ml of sterile TSB was transferred to the remaining three trays. The HRT dressings were placed on top of the inoculated solutions (contact layer down) and covered by the tray lids.

The trays and dressings were then incubated at 37°C. Three of the trays containing the inoculum were sampled after 10 minutes and the remaining six trays (three containing inoculum and three containing sterile TSB) were sampled at 48 hours. Following incubation, 1cm^2 sections of the lower polypropylene (PP) layer and the inner gel layer of each dressing were dissected using sterile scissors and tweezers. Samples were allowed to dry in air and then visualised using a TM3000 SEM (Hitachi).

All reagents and equipment were obtained from Fisher Scientific, Loughborough, UK, unless otherwise stated.

RESULTS

Experiment 1

Confluent growth was seen under the gauze and test dressing two from Day 1. Minimal growth was seen under test dressing 1 and under the HRT dressing (*Figure 2*). A similar pattern was observed at Day 3. From Day 5, less growth was visible under the HRT dressings than the other test dressings. Confluent growth was seen under the test dressings and

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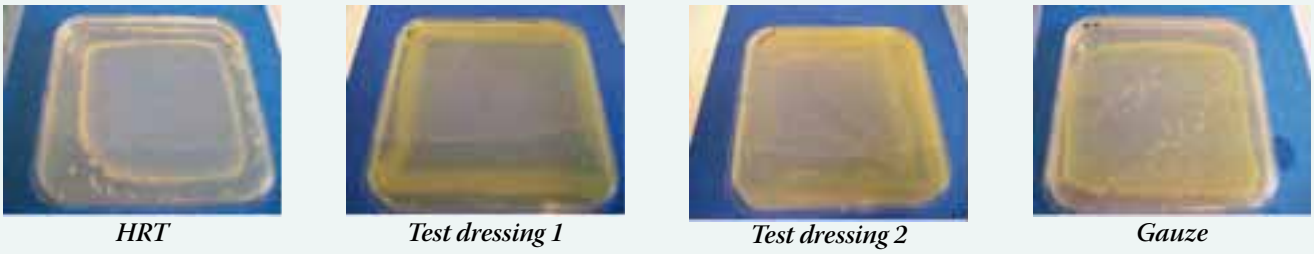

Figure 2: Photos of agar plates, following the removal of dressings that had been allowed to absorb P. aeruginosa *inoculum for one day.*

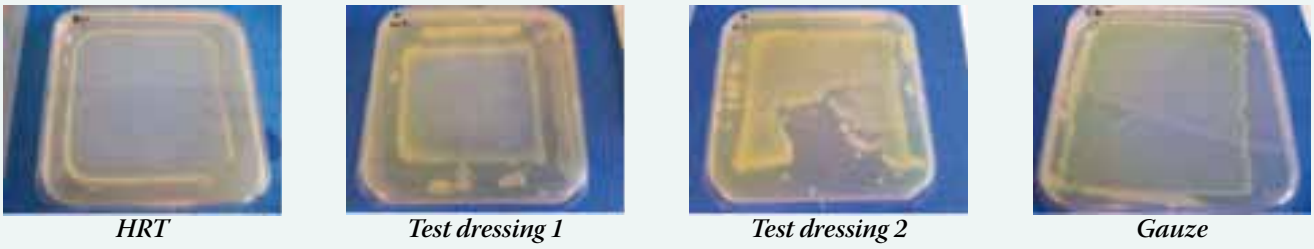

Figure 3: Photos of agar plates, following the removal of dressings that had been allowed to absorb P. aeruginosa *inoculum for seven days.*

gauze on Day 7 (*Figure 3*). There was some growth around the edge of all the test dressings.

In the case of the HRT dressing, this growth occurred beyond the line of the ultrasonic seal of the outer polypropylene (PP) layer. This was thought to represent bacteria that were in contact with the PP outer layer, but were not subject to the effects of HRT, since HRT provides the unique action of the dressing's inner core.

Fresh inocula were added to the agar plates that had not demonstrated bacterial growth. Growth was seen under all of the dressings after a further overnight incubation at 37°C, demonstrating that the dressings had not released any antimicrobial agents.

Experiment 2

Bacteria were not visible on the contact PP layer of the untreated dressings or on the dressings that had been immersed in *P. aeruginosa* bacterial inoculum for 10 minutes or 48 hours (*Figure 4*). The inner gel layer of the control sample was smooth and regular, while the test samples appeared to be covered by a thick, irregular substance (*Figure 5*), suspected to be of bacterial origin. The substance that appeared on the 48-hour sample appeared thicker and more developed than the covering on the 10-minute sample.

DISCUSSION

The HRT dressing was able to manage a bacterial bioburden over a seven-day period. The structure of the dressing's inner core provides close proximity between the gelling agents and the cellulose fibres (Cutting, 2009). It is considered that bacteria bind to the inner core. SEM images demonstrate that bacteria did not attach to the outer PP layer of the dressings (*Figure 4*), suggesting that they are drawn towards the inner core and sequestered. The finding that dressings can manage a bacterial burden for up to seven days implies that once bound to the inner core materials the bacteria are not released.

By holding exudate that contains microorganism within the dressing the risk of microbial transfer between the wounds of patients and healthcare practitioners is reduced (Evans, 2010). This, in turn, decreases the risk of cross-contamination between patients by healthcare practitioners. Bacterial growth following re-inoculation of the agar demonstrated that HRT dressings did not release active antimicrobial agents.

Bacteria held within the inner core of a dressing are prevented from re-contaminating the wound bed and thus actively decrease the wound bioburden without the use of an antimicrobial agent,

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Negative *10 minutes* *48 hours*

Figure 4: The outer PP contact layer of the HRT dressing after inoculation with a P. aeruginosa *inoculum.*

Negative *10 minutes* *48 hours*

Figure 5: The fibrous components of the inner layer of the HRT dressing after inoculation with P. aeruginosa.

thus avoiding the risk of inducing cell toxicity. The absence of an active antimicrobial agent within the dressing also avoids the risk of bacteria selecting for antimicrobial resistance. Selection for resistance occurs when certain strains of bacteria develop a tolerance to specific antibiotics to which they once were susceptible. This passive mechanism of bacterial sequestration avoids leaving behind untreated resistant populations that can thrive.

The absence of cellular toxicity associated with the use of HRT dressings supports active healing of the wound. HRT dressings can be used throughout the wound-healing process and are most effective when used in the presence of wound exudate. This means that a HRT dressing has the potential to decrease costs compared with the use of multiple wound dressings throughout the lifetime of a wound (Ling, 2011).

The multiple performance attributes of HRT, not necessarily limited to those reported here, suggest that HRT is a multi-functional wound dressing. The management of bacteria through sequestration has been demonstrated in the laboratory and this activity appears to be supported in clinical use.

When the HRT dressings, which had been previously exposed to bacterial inocula, were placed on agar plates, the area under the dressing remained clear. This finding was reported for the full seven days of the experiment, indicating that HRT dressings have a potential wear time of up to seven days.

The HRT dressing demonstrated superior sustained bacterial sequestration capability in comparison with the other test dressings.

CONCLUSION

The bacterial management of HRT is the result of the sequestering capability of the hydrodynamic core, while the bacterial growth on the agar following re-inoculation of the agar demonstrated that there was no release of an active antimicrobial agent from the HRT dressing. The physical forces that draw exudate and bacteria into the HRT dressing core enable the mechanism to actively reduce the bioburden at the wound surface. WUK