Hydrophobicity removes wound bioburden, aiding healing

Wound infection is one of the main areas of concern in the management of the wound environment. Infection complicates treatment and impedes the healing process by damaging tissue, reducing wound tensile strength and inducing an undesirable inflammatory response.[1-3] More recently, wound dressings (Cutimed Sorbact - BSN medical) have been introduced into clinical practice that reduces bacteria by adsorbing bacteria on the dressing surface through a hydrophobic effect.[4]

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Bacterial colonisation of a wound is normal. Where healing is progressing, adjuncts such as antimicrobials are generally not indicated as this could increase the risk of selection for resistance. A strategy to support healing lies in maintaining host immunological control of the wound environment.[5]

Hydrophobic interaction has been introduced to the array of wound dressings that interact with the surface bioburden. At its heart is the fatty acid DACC (dialkylcarbamoylchloride) that coats dressing fibres. This physical principle provides an interesting mechanism for bacterial binding. Microbes, including fungi, are irreversibly bound through hydrophobic interaction to the DACC coating on the dressing surface, allowing them to be disposed of at dressing change, without clinicians having to resort to 'traditional' antimicrobials.

Both *in vitro* and *in vivo* evidence demonstrates the efficacy of the DACC coating and resulting hydrophobic interaction in reducing the wound bioburden and facilitating healing. *In vitro* evidence indicates that DACC enhances binding of MRSA and *P. aeruginosa* biofilms.[6]

Bacterial adherence and hydrophobicity

The principle of hydrophobic (lacking an affinity for water molecules) interaction is a key mechanism for bacterial attachment. In order for invading pathogens to initiate an infection, they need to adhere to underlying damaged tissues.[7,8] Doyle, in a review of literature, showed there is a relationship between hydrophobicity and infection.[9]

Microbes can attach to exposed extracellular matrix components of a wound by hydrophobic and charge interactions and with receptor-like cell surface proteins called hydrophobins.[10]

Hydrophobic interactions take place when cells expressing cell-surface hydrophobicity come into contact with each other. When two hydrophobic molecules come into contact with each other in an aqueous environment they increase the entropy (the disorder of molecules, or the tendency for a reaction to proceed in a particular direction)[11] and expel water molecules[11,12] between them. In this way, they aggregate and are held together by the surrounding water molecules.

Impact of prolonged inflammation on healing

The physical removal of bacteria from the wound helps to remove the stimulus for continued dysfunctional neutrophil activity. Neutrophils and macrophages are essential to health; they target and destroy pathogenic microbes by phagocytosis and lyosomal enzyme breakdown and play a key role in growth factor production. However, neutrophils can have a negative effect on wound healing; high levels become highly destructive.[13,14]

Sustained neutrophil infiltration prevents wound healing because of the continuing proteolytic and oxidative havoc it wreaks and a hypoxic state will continue[15] chemically signalling further neutrophil recruitment. The destruction of pathogenic organisms reduces the bacterial load and therefore reduces exotoxin levels. However, the death and disruption of bacteria within the wound results in the release of endotoxins and the dumping of cell debris, leading to further inflammatory events locally and possibly systemically, even septic shock.[16] Therefore, treatment modalities that reduce wound bacterial numbers and proliferation rates without inducing bacterial death and the release of these toxins may be preferable to long-term wound health.

The benefits of DACC technology[17]

- Bacterial or fungal resistance does not develop
- No cytotoxicity
- As bacteria are not killed, there are no endotoxins released

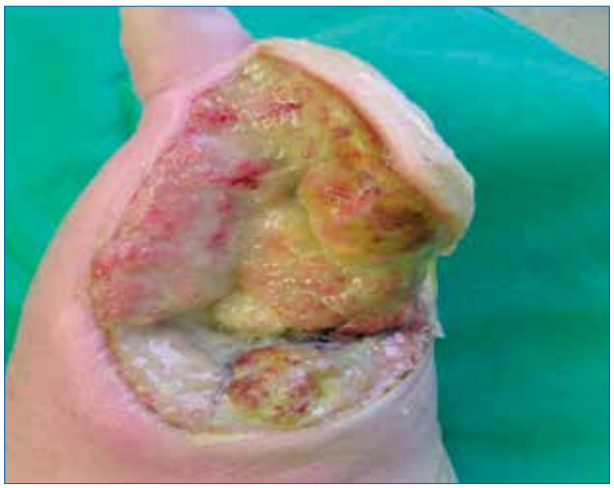

Figure 1: Day 1 (start of treatment)
Wound status on second postoperative day. Large and deep wound area. The wound margins are reddened, with heavy layers of fibrinous necrotic slough on the plantar side, and some superficial fatty tissue and muscle necroses.

- No contraindications
- No risk of allergic reactions.
- No upper binding capacity
- Can bind all common wound pathogens plus toxins
- No systemic absorption so suitable for use of all patients regardless of their age or underlying illnesses
- No cell debris

A technology that can bind bacteria to it rather than just kill it *in situ* represents a distinct paradigm shift from previous approaches to bioburden management. Traditional methods of control that aim to destroy microbes can be problematic as the chemical arsenal developed can turn against the environment they were designed to protect. Patient sensitisation, the development of resistant pathogens, cellular and systemic toxicity and the promotion of extended inflammatory response are all very real issues for the wound care clinician.[5]

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Figure 2: Day 2 (wound dressing)
A Cutimed® Sorbact® ribbon gauze is applied to the wound and covered by a Cutimed® Sorbact® absorbent pad fixed with an elastic gauze bandage. The next dressing change will be required at the following day because of the heavy exudation.

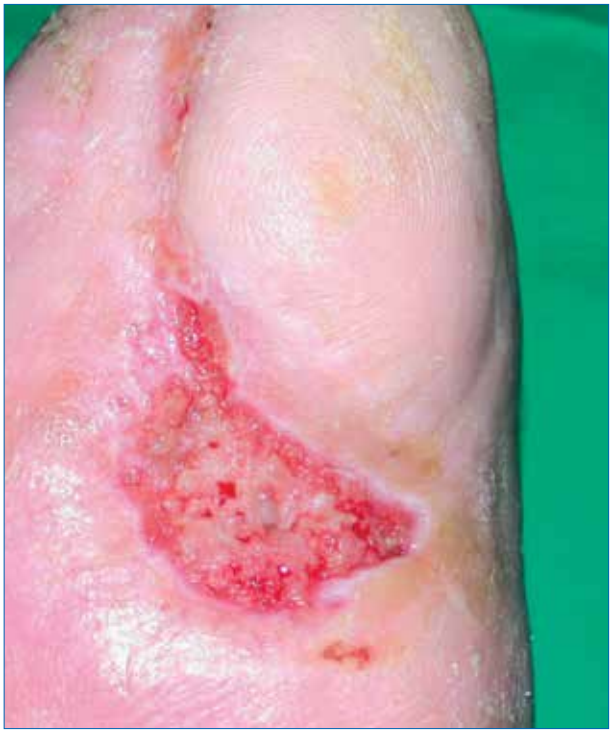

Figure 3: Day 73
With a wound size of 3 x 1.5 cm, the patient is discharged to a course of rehabilitative treatment.

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