Greetings everyone;

We'll now start our new topic of discussion, debridement with this week's article titled "Regular debridement is the main tool for maintaining a healthy wound bed in most chronic" (from Journal of Wound Care 2009, Vol 18, No 2, pages 54–56), this article is an introduction to biofilms and how sharp debridement is effective and cost-effective in removal and suppression of a biofilm.

Whilst the article can be read on a handheld mobile device (i.e. a smartphone), to view the tables in detail you may need to open it on a desktop device.
This material can be used in multiple ways: For example, treatment nurses are encouraged to read the emailed documents and discuss any questions they may have with the rounding staff from ASWC. Another approach would for the DON/charge nurses to discuss the articles(s) with the treatment nurses and encourage group participation on the topic of interest.

If you would like to add your colleague(s) to the email list please visit www.advantagewoundcare.org and on the left-hand margin you will see “subscribe to our mailing list”. This is an evolving platform; with time we will add other useful features to facilitate continuing education.

Sincerely;

G.S. Dhillon MD PhD

Regular debridement is the main tool for maintaining a healthy wound bed in most chronic

Sharp debridement is the most clinically and cost-effective way of physically removing and suppressing a biofilm. Continued debridement, as part of a multifaceted treatment strategy, will keep the biofilm in a weakened state

chronic wounds; biofilm; sharp debridement

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The concept of a 'healthy wound' seems contradictory, yet only when the wound bed is vital will the affected tissue progress toward dermal regeneration. A 'healthy' wound surface is created when biofilm infection, physiological stress, inflammation, exudate and necrotic tissue are minimised to such an extent that the host, even the compromised host, can dedicate physiological resources toward tissue regeneration.

This can be achieved through debridement, although how this works is not completely understood. Hypotheses include:

- Removing senescent cells[1]
- Balancing bioburden,[2]
- Improving microcirculation[3]
- Normalising the biochemistry.[4]

Debridement can be achieved through modalities such as:

- Appropriate dressings
- Pulse lavage
- Maggot debridement therapy
- Enzymatic debridement
- Autolytic debridement.

However, these modalities do not work specifically on biofilms, whereas surgical or sharp debridement does and is much quicker.[5-9]

Biofilm

Chronic wounds are mired in a chronic inflammatory state exhibiting markedly elevated pro-inflammatory cytokines (interleukin-1, tumour necrosing factor-alpha, gamma interferon), elevated matrix metalloproteases (MMP2, MMP8, MMP9) and excessive neutrophils.[10-14]

This persistent inflammatory state *may* be explained as the consequence of biofilm phenotype bacteria — namely, chronic infection.[15]

Although methods to detect and characterise biofilm in wounds are only now being developed and tested for sensitivity, recent unpublished work by our laboratory is showing that over 80% of all chronic wounds evaluated have biofilm phenotype bacteria and only wounds that are on a positive healing trajectory and are being treated with biofilm-based wound care do not have detectable biofilm phenotype bacteria (Dowd et al., unpublished data).

Our previous papers have explored the ecology, nature and physiological effects of biofilm in wounds,[15,16] so we will not repeat this here.

Biofilm-based wound care

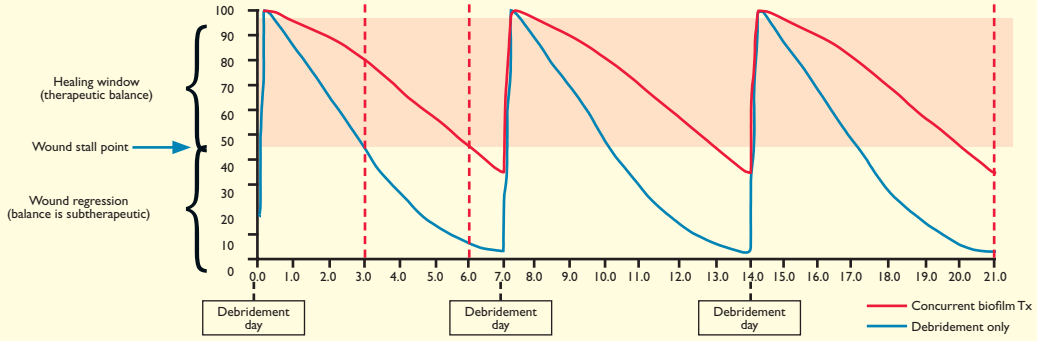
Biofilms are a wound management challenge for four main reasons:

- They are resistant to antibiotics (50 to 1500 fold)[17]
- They are highly resistant to biocides (hydrogen peroxide, acids, bleach)[18]
- They evade the host immune system (white blood cells, antibodies, complement)[19,20]
- They are poorly penetrated by many antibiotics used. [21-24]

Physical removal and suppression of biofilm reformation is therefore a necessary part of the wound management regimen. Experience and the literature have demonstrated that debridement is the most effective modality to achieve this.[25-28]

Debridement can also facilitate an opportunity for antibiotic intervention: it physically disrupts the biofilm, which then must reconstitute itself. To do this, the biofilm has to reattach to the host surface, becoming metabolically active. This increases the rate of proliferation and synthesis, in turn increasing nutrient uptake. This presents a healing window of opportunity for clinicians as biofilm phenotypes are much more susceptible to antibiotics and biocides in the first 24–72 hours (depending on community species) due to energy and nutrient expenditure in the growth phase and the immaturity of the protective extracellular polymeric substance (EPS) matrix.[17,18]

Fig 1. Graphical illustration of the effects of biofilm-based wound care



The goal of biofilm-based wound care is to ensure the therapy maintains its balance within the healing window, as described in the text. It can be assumed from this figure that, without concurrent strategy, the frequency of debridement could be increased to keep the wound from falling below the healing stall point. Debridement remains the primary tool for ensuring the wound stays above the stall point

Although debridement can remove the vast majority of biofilm phenotype bacteria, complete sterilisation of an active wound is unlikely, even with the most vigorous debridement, as bacteria have been shown to integrate around deep capillaries[29] and, based on both *in vitro* within 24 hours[30] and *in vivo* within 48 hours[31] evidence, extensive biofilm can reform in wounds very rapidly.

To take advantage of the healing wound, advanced and rapid diagnostic methods are required to identify the bacteria and their potential antibiotic susceptibilities. These diagnostic methods are now being utilised effectively in our clinic.[32]

Thus, a powerful combination of debridement, rapid molecular diagnosis of infecting agents, topical application of treating agents, and systemic antibiotics give a multiple concurrent strategy that we are finding to highly beneficial clinically.

As well as physically removing biofilm, temporarily disrupting its colony defences and forcing it to become more susceptible to antibiotics, biocides and host immunity,[17,18] debridement will also prepare the wound bed by opening all undermining and tunnels, removing all devitalised and poorly perfused tissue, and shaping the wound topography. The resulting smooth, well-perfused wound bed will inhibit biofilm adhesion. Maintenance debridement prevents re-establishment.

However, complete eradication of biofilm with debridement is not possible. Continued maintenance debridement is able to keep wound biofilm in a weakened and susceptible state. Fig 1 is provided in part to illustrate this. Two healing profiles are shown:

- Healing profile with debridement alone
- Healing profile using debridement with multiple concurrent strategies to inhibit the reformation of biofilm.

In this illustration, which is based on our clinical model for wound care, maintenance debridement alone keeps the wound balance in favour of the host (healing) for approximately 43% of the days between visits, although the rate of healing immediately post-debridement is more rapid. However, in this scenario, the wound is capable of regressing in 57% of the days between visits as the balance shifts back to favour the biofilm. When debridement is combined with multiple concurrent strategies to further inhibit the biofilm's recovery, the healing window (clinical opportunity) remains open for 86% of the days between visits; furthermore, the net rate of healing is also augmented. In this multiple concurrent strategy, only 14% of the days between visits are capable of regression.

While these are not absolute numbers relative to every wound, the order and separation of the rates for single versus multiple concurrent biofilm strategies are in alignment with what we see in the clinic. Regardless, the primary intent of Fig 1 is to illustrate the strategy of biofilm-based wound care in a multifaceted approach; this maximises the wound's healing potential.

Cost-effectiveness

The premise of this article is that debridement is pivotal to chronic wound healing. It represents a minor portion of the total US wound-care budget: $188 million dollars in 2005, less than 0.8% of the total amount spent on wounds.[33] It has also been demonstrated, both scientifically and experientially, to decrease back-end costs such as antibiotics, hospitalisation, amputation and death.[8,16] In rela-

tion to the expense of other treatments that have not been proven as broadly effective,[34,35] debridement would appear to be the most cost-effective option.

Conclusion

Biofilm is an important, and until recently, an unrecognised barrier to chronic wound healing. Clinical experience demonstrates that frequent disruption of biofilm, through debridement, forces the biofilm phenotype community to continually reattach to the host, reform its extracellular polymeric substance, and increase its metabolic activity for cell division, synthesis and colony activity. Each of these factors provides a clinical opportunity as the biofilm is more vulnerable to antibiotics and selective biocides during the recovery phase of the biofilm post-debridement. Debridement as part of a multiple concurrent strategy for wound care is an effective tool for the suppression of biofilm. ■

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