

Letters

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Wound biofilms — are they visible?

Over the past decade a number of publications have drawn attention to the polymicrobial nature of chronic wounds;^{1–3} in particular, the occurrence of bacteria in biofilm colonies and their possible role in ‘chronicity’.^{4,5} Given the broad spectrum of evidence available, it is reasonable to assume that bacteria existing in the phenotypical state of biofilm communities do exist in human wounds and actively contribute to recalcitrance.^{6,7}

Currently, the supportive body of evidence is, to a large degree, circumstantial, as a definitive assay for biofilms *in vivo* has yet to be developed. Transplantation of single organism biofilms grown *in vitro* to *in vivo* animal models are very useful scientifically,⁸ but cannot, in our estimation, be regarded as definitive for the human wound. Biofilms in wounds have taken on the mantle of being a ‘hot topic’, despite the fact that a number of authorities remain reluctant to accept, what is for others, an undeniable reality. It is, therefore, important that clinicians interpret the available evidence objectively, so that the emerging science of ‘biofilmology’ is not placed at risk of ridicule.

The purpose of this letter is to dispel myths and unwarranted assumptions that may have entered the wound care ‘culture’. In doing so, we would like to take this opportunity to present our assessment of the evidence, and clarify what is, and what is not, robust. We would also like to offer some guidance to clinicians on the impact of the evidence on treatments and outcomes.

The increasing interest paid to biofilms has arisen from the seminal work of Costerton and

colleagues, with their scholarly activity emanating from the late 1970s and early 1980s.^{9,10} This group is generally acknowledged to have ‘discovered’ biofilms and established the association of medical biofilms, infections and other chronic conditions, such as cystic fibrosis.^{11–13} Some of the earliest wound-related biofilm publications came in 2001.¹⁴

Since these publications, wound biofilm research has expanded and developed considerably;¹⁵ however, in spite of this attention, there are still many aspects of wound biofilm understanding that require further research. For example, while the existence of biofilms in wounds has been established from experimental models, and by persuasive circumstantial evidence, as yet there is no conclusive clinical/*in vivo* proof. The technology whereby patients’ wounds may be examined in the clinic to establish the presence of biofilms has yet to be developed. This is fundamental to the implementation and interpretation of treatment outcomes.

Despite this paucity of ‘hard’ evidence we have witnessed a number of publications and conference presentations/posters where the authors’ claim to have identified biofilm presence with the naked eye and presented putative photographic evidence in support of their claim. A recent example of this may be seen in Lenselink.¹⁶ While we applaud the authors’ endeavour and energy, we feel we must take issue with their interpretation of the wound bed and the assertion of wound biofilm presence.

Currently, no validation of visual observations with microscopic confirmation of biofilm presence currently exists. It is important that noviciates to

wound care are not misled into making similarly spurious claims. We share the assertions of others that biofilms do exist in wounds and do have an impact on ‘chronicity’, especially in diabetic foot ulcers, leg ulcers and pressure ulcers. Where supportive evidence exists, it is in the form of confocal laser scanning microscopy and not naked eye observation.¹⁷

Although no categorical evidence exists to dispute our position, we further assert that one positive intervention to remove or disrupt biofilms is debridement; in this respect we are not alone.^{18,19} Antimicrobials, especially those delivered topically in sustained release formulations, are generally effective in killing bacteria in planktonic forms. There is some laboratory data in respect of topical antiseptics, including honey, and their impact on biofilm communities,^{20–23} but these are limited in scope. Antibiotics in general have little impact on biofilm colonies, although there are reports of anti-biofilm activity for some antibiotics.^{24–26}

In chronic wounds where perfusion of the wound bed is often compromised, there can be little confidence that blood-borne antibiotics reach the affected area in sufficient concentrations to function efficiently. One possible strategy to circumvent this challenge is that of biofilm-based wound care (BBWC).²⁷ The key to success in this model of care lies in the concurrent, rather than consecutive, use of debridement, topical antiseptic agents and/or selective systemic antibiotics.^{17,18}

In conclusion, we feel that our understanding and clinical interpretation of wounds and biofilms must be based on firm

science and not on enthusiastic and unfounded claims. We suggest that clinical science embraces the concept of biofilms, recognise their potential role in chronicity and advocate that BBWC appears to offer the most appropriate approach to healing in these recalcitrant wounds.

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Combination of chlorhexidine gluconate and PVP in surgical site antisepsis

Surgical site infections can have a deleterious effect on the postoperative recovery of a patient undergoing surgery and, in particular, reconstructive procedures. Much research has been done comparing the efficacy of chlorhexidine and povidone-iodine (PVP), but little is written about the possible synergistic effect of combining both agents in surgical site antisepsis.

Chlorhexidine and PVP are both chemical antiseptics commonly used in preoperative skin cleansing. Chlorhexidine is

bactericidal and bacteriostatic on both Gram positive and Gram negative organisms, while PVP consists of the chemical polyvinylpyrrolidone and iodine. The iodine, which is liberated in solution, is what gives PVP its antiseptic properties; it is a broad spectrum bactericide, and is also effective against various fungi and viruses.

Recent evidence from *in vitro* studies on the sequential application of PVP, followed by chlorhexidine, have demonstrated superior antiseptic effects against a range of pathogens, when used in combination compared with when individually applied.²⁸ This effect has also been supported by two other studies using the same application method, prior to removing central lines and prepping patients for neurosurgery.^{29,30}

This superior protection was attributed to the reduction in superficial and deep incisional infections that were caused primarily by Gram positive skin flora. We feel that this could very well be applied to plastic surgery procedures, which most commonly can be complicated by superficial wound infection.

We currently employ this method of antisepsis at our trust

hospital for routine day cases. Each patient is prepped uniformly, ensuring that the surgical site is covered with the disinfectant and then wiped dry with sterile gauze. The procedures are limited to simple excision of lesions and all patients are followed-up in dressing clinic for wound check.

To date, we have noted no adverse reactions in patients, following the sequential application of both antiseptic agents, neither have other investigators in separate series.

The available literature suggests the possibility of a synergistic effect of combining both agents in surgical site preparation. Clinically, we have observed that combining both do result in a decreased rate of infection. The additional cost is minimal, but there is the benefit of preventing a possible wound infection complication. Also by wiping away the yellow stain left by iodine using chlorhexidine, patients will not feel stigmatised or shocked by the yellow stain.

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The editor welcomes readers' letters.

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