# Evidence Summary: Wound Infection: Biofilms and Sharp Debridement

6 June 2012


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## Question

What is the best available evidence of the effectiveness of *sharp* debridement in the eradication of wound biofilm?

## Clinical Bottom Line

Biofilms display greater vulnerability to antimicrobial intervention within the first 24-48 hours of formation. This finding is particularly applicable to the time period following sharp debridement of a wound.[1]

## Biofilms defined

- The bioburden of an infected wound that fails to respond to treatment and progresses to a chronic wound is likely to involve one or more biofilm/s.[2,3] (Levels IV & II resp.). Along with other bacteria, free-floating, single-cell planktonic bacteria are present on the skin surface in a non-pathogenic relationship with the host. A biofilm is created when bacteria (multiple or single species) adhere to a wound surface and by secreting a mucopolysaccharide substance form a protective exopolymeric matrix (EPS) that encapsulates the biofilm community.[2,3,4] (Level IV; II & III resp.)
- Through this process a previously non-pathogenic and mutually interdependent relationship between the human host and commensal bacteria undergoes a parasitic transformation that results in a self-sustaining cycle of chronicity causing harm to the human host.[4] (Level III)
- The formation of biofilm communities follows a complex and well-coordinated sequence of molecular events designed to maximise the microorganism community's survival and sustainability. *In vitro* tests* have identified the following events associated with the formation of biofilm:[1,3,4,5] (Level II; II; III; IV resp.)
  - Bacteria attaches rapidly to a wound surface
  - Within 2-4 hours forms microcolonies
  - Within 6-12 hours bacterial communities encase themselves in a protective extracellular matrix composed of self-secreted alginates that provides increased resistance to antibiotics and anti-microbial agents.
  - Within 24-48 hours a fully matured biofilm is established.
  - Established biofilm recovers from mechanical disruption within 24 hours.
  - To be effective, intervention aimed at reducing and/or eradicating biofilm must be implemented within this 24 hour "window of opportunity".

** Research has noted structural congruence between the features of biofilms formed from single species in vitro and those formed by mixed species in nature*[6] (Level III)

## Debridement defined

Sharp debridement is the removal of foreign matter and devitalised / necrotic tissue from a wound using either a sterile scalpel or sterile scissors. Debridement aims to reduce and remove infected tissue from a wound until the surrounding healthy tissue is exposed; the application of an appropriate (antimicrobial) intervention follows to encourage the wound healing process.[5,7,8] (Levels IV)

- Sharp debridement can be performed in the homes of clients, a clinic or the hospital bedside. It is performed by a doctor, nurse or podiatrist suitably qualified in the technique. Care to avoid tendon damage involves heightened precaution over joints or feet. In skilled hands, sharp debridement can be a cost and time-efficient method, particularly when compared with surgical debridement which involves a surgeon and the use of a theatre.[8] (Level IV)
- A number of debridement methods are available and clinicians' choice depends upon their knowledge, experience and skill; patient factors including safety and suitability to the form of debridement chosen are also important considerations.[5,8] (Levels IV)

## Therapeutic window of opportunity

A multi-centre laboratory trial evaluated the hypothesis that ***newly formed*** wound biofilms are more susceptible to antimicrobial intervention than are established biofilms. The study found that biofilm resistance to antimicrobial treatment increases within a very short timeframe.[1] (Level II). The main features of the study and its findings are as follows:

- Four models were used to investigate biofilm (*P. aeruginosa* and *S. aureus)* susceptibility over time. Tissue samples were 'debrided' under lab conditions; samples for control conditions were immersed in test tubes containing 10ml of saline solution; samples for intervention experimental conditions were immersed in test tubes containing 10ml

of saline solution with 200µg/ml gentamicin for 24 hours. Bacterial levels were expressed as the average colony forming units per ml (CFU/ml) found in the bacterial suspension.

- The models used were as follows:-
  - An *in vitro* drip-flow biofilm model.
  - A hydro-debridement study.
  - A porcine skin punch biopsy *ex vivo* model.
  - A mouse chronic wound model.
- In all conditions the results showed that after 18 hours *P. aeruginosa* biofilms had developed increased resistance against the antimicrobial agent. This resistance continued to strengthen over time with peak resistance being reached at 48 hours of growth; thereafter antimicrobial treatment was ineffective and no differences in CFU counts of the control biofilms and gentamicin-treated biofilms were detected. *S. aureus* biofilm took a little longer to reach antimicrobial resistance but by 72 hours considerable resistance was noted and by 96 hours it too was completely resistant to the effects of the antimicrobial.
- Biofilm **regrowth** of *P. aeruginosa* following debridement showed an increased resistance to the antimicrobial agent. Susceptibility to the intervention was evident for 6–24 hours post-debridement compared to the 24 hours observed in the initial exposure.
- *S. aureus* biofilm **regrowth** following debridement showed a similar increase in resistance to the antimicrobial agent. Susceptibility to the intervention was evident for 24 hours post-debridement compared to the 48 hours observed in the initial exposure. Complete resistance was noted at 72 hours.[1] (Level II)

## Clinical debridement

- In addition to the animal explants experiments above, a small clinical longitudinal debridement study was conducted using wound tissue samples from three human patients (with their permission); at baseline these samples had significant levels of *P. aeruginosa* (average 5.2 x $10^8$ CFU/5mg bioburden).[1] (Level II). The main features of this arm of the study and its findings are as follows:
  - Within the 24 hours following sharp debridement sensitivity to antibiotic treatment was clearly evident demonstrated by a significant ($p>0.05$) nine-fold reduction in cells in the biofilm community (i.e. the CFU count).
  - In this study, two of the three tissue samples were still sensitive to antibiotics at 48 hours post-debridement but one sample had regained resistance to the antibiotic. At 72 hours all three biofilm samples were almost back to pre-debridement resistance levels.

## Clinical application of lab findings

- During the initial formation stages of bacterial biofilm cells show high levels of activity (cell motility) while building up the biofilm colony; once formed, the cells' motility diminishes and slows to a level that is unable to be detected by antibodies; this important defence strategy protects the biofilm community from destruction once it has matured but leaves itself exposed during the initial formation period. Antibiotics can only target cells that are active and it is this rationale that is exploited by the recommendation that antimicrobial intervention be commenced within the first 24 hours following sharp debridement of the wound.[1,6] (Levels II & III resp.) Underlying this recommendation is the assumption that biofilm will recur and that preventative treatment at the early stage of post-debridement may eliminate the need for tertiary intervention of a biofilm that has reformed and re-established in the wound.
- While evidence from these studies requires replication on a larger scale the results reported here are based on sound laboratory research and demonstrate that the first 24 to 48 hours following sharp debridement are the most crucial in biofilm management strategies. Biofilm-forming cells are at their most vulnerable to antimicrobials during this timeframe; this also applies to biofilm regrowth post-debridement but with an added urgency as some re-growth bacterial cells have shown earlier resistance at six hours in some cases.[1,5,6] (Levels II, IV, III resp.)

## Biofilm management strategies

A number of approaches designed to disrupt biofilm infrastructure are currently being investigated. Amongst these are:

- Aggressive sharp debridement of the wound slough and the underlying tissue that contains biofilm is recognised as a key intervention at the beginning of treatment and as a continuing maintenance strategy as biofilm cells reform quickly once disrupted; this intervention represents an important prevention strategy to stop regrowth biofilm.[1,2,3,4] (Levels II, IV, II, III resp.).
- Following debridement the wound is dressed with an appropriate antimicrobial dressing.[1,2,3,4] (Levels II, IV, II, III resp.).
- The first 24 hours after ***sharp*** debridement or the first 24 hours of an initial biofilm development, provides a therapeutic window for the application of topical antimicrobials. Cells involved in early biofilm (re)formation demonstrate increased sensitivity to antimicrobials and anti-biofilm agents at this time. These findings have been demonstrated *in vitro* using porcine and mouse models and also *in vivo* using venous leg ulcer samples from humans.[1] (Level II)
- In an established biofilm prevention and eradication through the use of antibiotics and topical antimicrobials is

largely ineffective due to the protective EPS matrix;[2] (Level IV); the use of povidone iodine however is showing some promise[9] (Level IV), as is silver-containing hydrofibre.[10] (Level III)

## Characteristics of the Evidence

This evidence summary is based on a structure search of the literature and selected evidence-based health care databases. The evidence in this summary comes from:

- A laboratory controlled study that examined the relationship between sharp debridement and time-dependent therapeutic intervention.[1] (Level II)
- A position paper of the Australian Wound Management Association (AWMA).[2] (Level IV)
- A study of biofilm-based wound management in 190 subjects with critical limb ischaemia.[3] (Level II)
- A paper that presents a hypothesis that attributes wound biofilm formation to an impotent initial immune response that perpetuates inflammation and chronicity.[4] (Level III)
- An educational summary of the application of iodine in wounds.[5] (Level IV)
- A paper that translates the ecological characteristics of microbial biofilm into a model that describes the inherent molecular genetics.[6] (Level III)
- A review that summarises a number of debriding techniques and reported on a multi-centre, randomised controlled trial.[7] (Level IV)
- A review explaining the different methods of wound debridement and the systematic assessment of chronic wounds.[8] (Level IV)
- An educational summary of the application of iodine in wounds.[9] (Level IV)
- A lab-based study that reported the eradication of wound biofilm using a silver hydrofibre wound dressing.[10] (Level III)

## Best Practice Recommendations

The same practice and principles of good wound care are applicable to biofilm-based wound care.

Biofilm-based wound care emphasises the importance of the following practices:

- Debride the wound to remove wound slough and the underlying tissue that contains the biofilm. (Grade A)
- Following debridement, dress the wound with an antibacterial barrier dressing that prevents planktonic bacteria from rapidly reforming biofilm colonies on the wound. (Grade B)
- Debride on a regular basis in order to create an optimal molecular environment in the wound. (Grade B)
- Prevent the reformation of biofilms by instigating anti-microbial intervention within the first 24 hours of biofilm (re)formation. (Grade B)
- Remain informed of the latest developments in the emerging knowledge and treatment of biofilms. Currently investigations indicate promising results with the following two approaches:
  - The use of nanocrystalline silver dressings in the prevention of biofilm formation. (Grade B)
  - The use of sustained release cadexomer iodine to kill bacteria biofilm. (Grade B)

## References


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10. Percival S. Assessing the effect of an antimicrobial hydrofiber wound dressing on biofilms. Wound Repair Regen 2008; 1:52–57. (Level III)


## Audit Criteria

1. The wound is debrided and wound slough as well as the underlying tissue that contains the biofilm is removed.
2. Within the first 24 hours following initial debridement and after maintenance debridement, the wound is dressed with an antibacterial dressing.
3. The antibacterial dressing selected is based on current evidence of effectiveness in respect to biofilms e.g. nanocrystalline silver or sustained release cadexomer or povidone iodine.
4. The wound is debrided on a regular basis and newly developing biofilm is removed.
5. The organisation's policy on the management of chronic wounds reflects current best practice i.e. it is updated on an annual basis.

**Keywords:** Wound; biofilm; sharp debridement; colony forming units (CFU); antimicrobial resistance