WOUND INFECTION

Almost all wounds are colonized with bacteria however this does not mean they are infected?

NO

In fact in general the presence of bacteria in a wound will not inhibit wound healing

This principle is being challenged in that some wounds although not infected show a delay in wound healing

Colonization v Infection

Colonization can be defined as the presence of large numbers of bacteria which cause no ill effect to their host

Infection

Infection occurs if normal tissues by bacteria and destruction of cells occurs, or if the colonizing bacteria produce poisonous materials eg. Toxins, that diffuse into tissue and damage the cells.

WOUND INFECTION

One way bacteria alter the healing process is the presence of endotoxin or lipopolysaccharide from gram-negative bacterial cell walls.

This triggers the release of interleukin-1 tumor necrosis factor α andIL-6,

This stimulates the production of phospholipase A₂ (PLA₂) thought to be responsible for conditions such as septic shock

(Trengrove 2000)

Bacterial Balance

Control mechanisms

- Intact skin is physical barrier
- pH is not conducive to bacterial growth
- Skin secretes fatty acids and antibacterial polypeptides
- Normal flora aide in preventing pathogenic flora from establishing
**Bacterial Burden in Wounds**

- **Contamination**
  - The presence of non-replicating microorganisms within the wound.
- **Colonisation**
  - The presence of replicating microorganisms that do not cause injury to the host.
- **Local Infection**
  - The presence of replicating microorganisms that are beginning to cause local tissue damage.
- **Spreading / Systemic Infection**
  - The presence of replicating microorganisms that are capable of causing injury to the host.

---

**Antibiotic Facts**

- Chemical compounds that either kill or inhibit growth of bacteria (i.e. bactericidal or bacteriostatic)
  - Not viruses or fungi (There are specific antiviral and anti fungal drugs)
- Show selectivity only for certain bacteria
  - Spectrum of action varies from compound to compound (This may be either Narrow or broad)

---

**ANTIBIOTICS: MODES OF ACTIONS**

1. **Inhibition of cell wall synthesis**
   - eg. penicillins, cephalosporins, vancomycin, bacitracin, novobiocin
2. **Inhibition of cell membrane function**
   - eg, polypeptides (polymyxin, colistin), tyrothricin, polyenes (amphotericin, nystatin), lira, keto, fluconazoles, tetrinatine, amorolfine
3. **Inhibition of nucleic acid synthesis**
   - eg actinomycin, mitomycin, colicin, quinolones, griseofulvin, ethambutol, rifamides, isoniazid.
4. **Inhibition by competitive inhibition**
   - eg, sulfonamides, para-aminosalicylate, dapsone, 5-flucytosine, nitrofurantoin
5. **Inhibition of protein synthesis**
   - eg, chloramphenicol, tetracyclines, macrolides, lincosamides, aminoglycosides, linizolid.
Bacterial Burden

Contamination - Infection Continuum

Trengove (1996) found that there was significantly greater chance of failure to heal if four or more groups of bacteria were present in the wound.

Host

Resistance

Bacterial quantity and virulence

Local perfusion

Adhesins

Immunosuppression

Cell Capsules

Diabetes

Biofilms

Medications

Antibiotic Resistance

Clinical Presentation

Acute Wound Infection

or

Severe Chronic Wound Infection

Advancing erythema

Fever

Warmth

Oedema / swelling

Pain

Purulence

“Classic” Signs & Symptoms

Critically Colonized

Delayed healing

Change in color of wound bed

Friable granulation tissue

Absent or abnormal granulation tissue

↑ or abnormal odor

↑ serous drainage

↑ pain at wound site

“Secondary” Signs & Symptoms

“The validity of the clinical signs and symptoms used to identify localized wound infection”

Wound Repair and Regeneration 2001;9(3):178-186

- “Traditional” Signs & Symptoms need not be present for local wound infection to be present in chronic wounds.
- Quantitative tissue biopsy demonstrated that “secondary” signs & symptoms occurred more often than “classic” in chronic wound infections.
- No single sign or symptom is 100% sensitive suggesting that none should be considered crucial or necessary to identify a chronic wound infection.
- Increasing pain and wound breakdown considered sufficient.

Gardner SE, Frantz RA, Doebbeling BN (2001)

Bacterial Biofilms Are Major Barriers For Wound Healing

Presence of bacterial biofilm is a major cause of the chronic inflammation/infection that prevents wounds from healing.

Detecting, removing and treating bacterial biofilms is a crucial component of Wound Bed Preparation.

Gardner SE, Frantz R, Doebbeling BN (2001)
Five stages of biofilm development in *Pseudomonas aeruginosa*. In Stage 1, bacterial cells attach reversibly to the surface. At Stage 2, the cells attach irreversibly, mediated by exopolymeric substances, and lose flagella-driven motility. In Stage 3, the biofilm architecture occurs, forming microcolonies, while in Stage 4 the fully mature biofilm architecture is achieved. In Stage 5, dispersion of single motile cells occurs from the mature biofilm, which ‘seed’ other surfaces, re-initiating the process.

**Electron Micrographs Reveal Biofilms on 60% of Chronic Wounds But <10% of Acute Wounds**

**How Do Bacterial Biofilms Form?**

**How Does The Immunological Response to Biofilms Cause Tissue Damage?**

**Prevention of Biofilm Formation by Silver-Containing Dressings**

**Identification of Different Bacterial Genuses in a Biopsy of a Chronic Pressure Ulcer**

A total of 36 different bacterial genuses were identified by nucleic acid sequences. The % represents the percentage of the total sequences analyzed within the sample. The 8 main of the Bacteria represent 70% and 7 of the 8 are Anaerobes.

Distribution of Aerotolerance of Bacterial Populations in Chronic Wounds

Recommendations

- Routine wound cleansing
- Exudate management

Recommendations

- Thorough cleansing
- Debridement
- Consider topical antimicrobials
- Silver
- Slow release cadexomer iodine
- Exudate management

Atypical Wound Infections

- Insect Bites
- Fish string
- Buruli Ulcers

White-Tailed Spider
Buruli Ulcers

These ulcers were first identified in Australia by MacCallum and others though it was first described in Uganda.
- In 1897 by Sir Albert Cook
- It is an uncommon ulcer in Australia
- However it is a very big problem in Africa
- The Ivory Coast alone has recorded 15,000 cases since 1978

The wounds are caused by an infection from Mycobacterium Ulcerans the bacteria produces a toxin that causes the massive tissue damage. Named mycolactone the toxin secreted by M. ulcerans causes skin cells to become rounder and then die.

Buruli ulcers have an unusual, “clean,” uninfected look – a result of the disruption of normal immune response.

The bacteria is associated with water and the infection is the result of a minor trauma. The resulting wounds are slow to develop however once established can be very necrotic in nature.

The wound is often painless and can occur on any part of the body. Though the ulcers are self-limiting, and will heal over a number of months they may lead to severe joint contractures, and disfigurement.

Some reports suggest the possibility of transmission by insects.

The treatment usually involves major surgical debridement and skin grafting.

There is no antibiotic treatment yet that has been shown to destroy the bacteria. The latest research involves the use of dihydofolate reductase inhibitor with Dapsone, Rifampacin and Dapsone with some small studies showing encouraging results.

Clinical indications
- Local heat
- Pyrexia
- Erythema
- Increased pain
- Increased exudate
- Frank pus
- Lab tests – WCC, ESR, CRP, etc

Confirmed/identified by
- Microscopy
- Culture
- Serology
Principles of Antibiotic Use
(From: The Antibiotic Guidelines)

- **General**
  - Used only where benefits scientifically demonstrable
  - Use narrowest spectrum agent to cover likely pathogen(s)
  - Single drugs used unless proven that combination therapy required
  - Dose high enough to ensure efficacy & minimise risk of resistance without toxicity

- **Therapy**
  - Therapy based on culture (directed therapy) or known common pathogens & their resistance patterns (empirical therapy)
  - Duration as short as possible (not >7 days unless proof that extended therapy needed)

- **Prophylaxis**
  - Choice based on known or likely target pathogens
  - Duration as short as possible

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Using Topical Antiseptics

- Infection risk or colonisation as indicator for use
- Consider if systemic therapy necessary
- Therapy selection based on:
  - Risk
  - Therapeutic goal
  - Understanding of chemical properties
  - Delivery vehicle
- Consider antibacterial dressings

---

Antiseptics

- Effectiveness as a bactericidal/bacteriostatic must be balanced against likely degree of damage to human healthy tissue.
- Kamora demonstrated that antiseptics bind to organic substances in the wound thus failing to penetrate at levels sufficient to produce antimicrobial activity.
- Fleming (1890) stated “it is essential in the estimation of the value of an antiseptic to study its effect on the tissues rather than its effect on bacteria.”
- Lineweaver, Branemark, Van Der Broek, Rodeheaver & Leaper all demonstrated the ill effects of antiseptics on tissue.

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Antiseptics- When to Use

- Hand washing prior to performing aseptic procedures (95% reduction in skin bacteria)
- Skin preparation before passing an instrument
- Immediate decontamination of an acute wound, when the bacterial inoculum is likely to be high
- For skin breaches, orthopaedic pins, peritoneal/IV catheters
- As specified by the manufacturers
- Patients with reduced immune system function
- Prior to skin grafting

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Antiseptics with Limited Place in Therapy

- Many antiseptic cytotoxic effects outweigh antibacterial effects including:
  - Toxicity to fibroblasts
  - Occlude microcirculation
  - Retard collagen deposition
  - Oxygen embolus risk (peroxides)
  - Cause localised oedema, hypernatraemia, hyperthermia, burns (hypochlorites)
- Some are feeble antiseptics
- Includes: Hypochlorites, peroxides, phenolics, mercurochrome, pot permanganate

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Tissue Toxicity

The most cytotoxic products included those which contained silver or Chlorhexidine & Paraffin Cream™ a moisturizer which contains the preservative Chlorocresol.

Essential Oils with Antimicrobial Effect

- Oregano
- Tea Tree
- Mint
- Sandalwood
- Clove
- Nigella Sativa
- Onion
- Lavender
- Lemon
- Eucalyptus
- Peppermint
- Cinnamon
- Clove
- Thyme

Tea-Tree Oil

- Evidence of antiseptic activity – used for this for many years
  - Some research looking at use as alternative to chlorhexidine for oral microorganisms
  - More effective than other oils against MRSA & VRE

- New evidence for other activity
  - Wound cooling (Melaleuca hydrogel)
  - Inhibit in vitro growth of human melanoma cells

- Some problems with contact dermatitis
  - Limited activity
  - Essential oils extracted containing range of chemicals

  Caution is needed as there have been some studies indicating toxicity

Tea-tree Oil Components

MELALEUCA ALTERNIFOLIA (Myrtaceae) “Tea-Tree” 1,4-CINEOLE Leaf 0.001 ppm 1,8-CINEOLE Leaf 260 - 16,000 ppm ALLOAROMADENDRENE Leaf 45 - 112 ppm ALLYL-HEXANOATE Leaf 0.001 ppm ALPHA-BULNESENE Leaf 36 ppm ALPHA-CADINENE Leaf 143 - 358 ppm ALPHA-COPENE Leaf 10 - 25 ppm ALPHA-CUBELENE Leaf 4 - 11 ppm ALPHA-GURJUNENE Leaf 23 - 56 ppm ALPHA-AURONE Leaf 8.01 - 30 ppm ALPHA-P-DIMETHYLSTYRENE Leaf 0.001 ppm 1,8-CINEOLE Leaf 7 - 18 ppm ALPHA-PHELLANDRENE Leaf 10 - 50 ppm ALPHA-PINENE Leaf 200 - 700 ppm ALPHA-TERPINENE Leaf 100 - 4.375 ppm ALPHA-TERPINOL Leaf 180 - 902 ppm ALFA-TERPINEOL Leaf 364 ppm ALPHA-THUJENE Leaf 76 ppm AROMADENDRENE Leaf 235 - 675 ppm BETA-ELEME Leaf 0.001 ppm BETA-PHELLANDRENE Leaf 75 ppm BETA-PINENE Leaf 59 - 950 ppm CALAMENENE Leaf 10 - 25 ppm CAMPHENE Leaf 0.001 ppm CAMPHOR Leaf 0.001 ppm CARYOPHYLLENE Leaf 0.001 - 154 ppm CYMENENE Leaf 120 ppm CYMENENE Leaf 10.000 - 25,000 ppm CYMENINE Leaf 1.154 - 3,000 ppm HEXANOL Leaf 0.001 ppm HYMULENE Leaf 0.001 - 12 ppm LIMONENE Leaf 100 - 250 ppm LINOCOL Leaf 10 - 25 ppm MENTHAHATRIENES Leaf 0.001 ppm MYRICE Leaf 52 - 130 ppm NEROL Leaf 0.001 ppm P-CYMEN-8-OL Leaf 15 - 32 ppm P-CYMENINE Leaf 300 - 2,850 ppm PIPERITOL Leaf 7 - 18 ppm PIPERITONE Leaf 8 - 20 ppm SABINE Leaf 12 - 30 ppm TERPINEN-1-OL Leaf 40 - 100 ppm TERPINEN-4-OL Leaf 2.941 - 11,205 ppm TERPINOLENE Leaf 238 - 6,125 ppm VIRIDIFLORENE Leaf 103 - 257 ppm

Common Antiseptics

- Hypochlorites
  - Dakins Solution, Eusol
  - thought to dissolve necrotic tissue
  - highly toxic to fibroblasts
  - occludes microcirculation
  - retards deposition of collagen
  - can cause localised oedema, hypernatraemia, hyperthermia, burns
  - chemically instable
  - rapidly deactivated by organic material

Tissue Pre-exposure

24 hours post-exposure

The effect of antiseptics on the healing wound: a study using the rabbit ear chamber

The effects of several antiseptic agents on prisional tissue were studied using rabbit ear chambers. This method is used to study and quantify the effects on the microcirculation of the ear. All the agents used caused adverse effects, but in the case of hypochlorites, Dakins and Chlorate, T. Blood flow in the capillary circulation of the prisional tissue and the process of repair seems to be substantially delayed. A 1.4% sodium thiavent fluid was used in these experiments. It was found to be effective in reducing the toxic effect on the microcirculation of the ear.
Tissue Perfusion Units of Flux by Laser Doppler (n=10 in each category; medians & ranges)

<table>
<thead>
<tr>
<th>Time</th>
<th>Saline</th>
<th>Eusol</th>
<th>Povidone</th>
<th>Iodine</th>
<th>Povidone Iodine</th>
<th>Hydrogen Peroxide</th>
<th>Chlorhexidine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>47(40-50)</td>
<td>46(38-40)</td>
<td>28(24-32)</td>
<td>44(44-48)</td>
<td>30(26-34)</td>
<td>28(24-32)</td>
<td>28(24-32)</td>
</tr>
<tr>
<td>1 Min</td>
<td>44.5(42-47)</td>
<td>4(0-10)</td>
<td>4(0-10)</td>
<td>13.4(2-26)</td>
<td>42(40-48)</td>
<td>9(0-20)</td>
<td>9(0-20)</td>
</tr>
<tr>
<td>5 Min</td>
<td>47(44-50)</td>
<td>0.5(0-4)</td>
<td>3(0-6)</td>
<td>15(6-22)</td>
<td>47(42-50)</td>
<td>26(22-28)</td>
<td>26(22-28)</td>
</tr>
<tr>
<td>10 Min</td>
<td>46.5(45-50)</td>
<td>1.0(0-10)</td>
<td>6(0-10)</td>
<td>20(18-30)</td>
<td>50(46-56)</td>
<td>26(22-38)</td>
<td>26(22-38)</td>
</tr>
<tr>
<td>30 Min</td>
<td>46(46-49)</td>
<td>1.5(0-6)</td>
<td>3(0-12)</td>
<td>24(22-26)</td>
<td>52(48-55)</td>
<td>39(30-44)</td>
<td>39(30-44)</td>
</tr>
<tr>
<td>60 Min</td>
<td>47(44-52)</td>
<td>0.5(0-4)</td>
<td>4(0-21)</td>
<td>25(23-30)</td>
<td>49(46-56)</td>
<td>30(26-34)</td>
<td>30(26-34)</td>
</tr>
<tr>
<td>24 Hrs</td>
<td>2(0-10)</td>
<td>15(8-24)</td>
<td>28(22-32)</td>
<td>48(42-50)</td>
<td>42(39-50)</td>
<td>40(30-48)</td>
<td>40(30-48)</td>
</tr>
</tbody>
</table>

SS. Brennan & DJ Leaper

**Common Antiseptics**

- **Hydrogen Peroxide**
  - Decomposes to oxygen and water
  - Oxygen bubbles may physically loosen debris
  - Very little anti-microbial activity
  - Highly toxic to fibroblasts
  - Risk of oxygen emboli
  - No place as a wound antiseptic

- **Chlorhexidine**
  - Low toxicity to granulation tissue
  - Skin sensitivity reasonably common
  - Deactivated by organic material
  - Principally used on intact skin (e.g., in surgical scrubs)

- **Savlon**
  - Chlorhexidine and cetrimide
  - Cetrimide has surfactant properties therefore useful in removing debris in acute wounds
  - Cetrimide highly toxic to fibroblasts

**Common Antiseptics Iodine**

- Iodine in its various forms has been used as a topical antiseptic since 1840.
- The newer forms of iodophores have been used since the 1950s. Most of these new forms combine iodine in a complex with a polymer.
- E.g., Povidone, Cadexomer these slowly release the iodine.
- Iodine is active against bacteria, mycobacteria, fungi, protozoa, and viruses. There is no evidence of resistance to iodine.

- **Povidone Iodine**
  - Betadine (10% P-I)
  - Wide spectrum of activity
  - Inactivated by body fluids
  - 5% solution causes cessation of blood flow
  - Toxic to fibroblasts
  - Risk of systemic absorption
  - In an acute wound it is usually left in place for 3-6 minutes and then washed off with clean water.
  - In a chronic wound dilute 1 in 10/1 in 20 before use

- **Acetic Acid**
  - 5% (vinegar)
  - Action due only to its physiological unacceptable pH
  - Two clinical trials have shown efficacy in treating Pseudomonas, due to acidic pH
  - Topical wash for 10 minutes twice a day.
  - Toxic in dilution to fibroblasts
  - Recent research has shown that a 3-4% aqueous solution of Citric Acid is as effective in reducing pseudomonas as Acetic Acid with less risk of tissue damage
Cadexomer Iodine Dressings (Iodosorb)

- Absorbent - forms gel with exudate
- Releases iodine as gel forms
- Pulses iodine at 0.1% (not cytotoxic)
- For sloughy/infected wounds
- Iodine may stimulate growth factors

0.9% Iodine immobilised in cadexomer is slowly released from the cadexomer to an iodine free environment in the presence of exudate (the wound)

- The Iodine (I₂) will move across a concentration gradient until an equilibrium is established between IODOSORB® and the wound bed.

- Once in the wound bed, the I₂ will convert to I⁻ as it kills micro-organisms. When all the I₂ has been converted to I⁻ there will be a noticeable colour change which will indicate that it is time to change IODOSORB®.

Characteristics:
- 0.9% iodine in a polysaccharide base
- Very absorbent (powder for wetter wounds)
- Sustained slow release of ‘safe’ iodine
- Reduces the pH of the wound, enhancing antimicrobial effect
- ‘Kick’ starts chronic wound healing
- Converts to a gel resembling ‘mashed potato’ or ‘marzipan paste’
**Cadexomer Iodine (Iodosorb)**

**Common errors:**
- Failure to check for Iodine/shellfish sensitivities (Hashimoto’s thyroiditis, Graves disease)
- Weekly maximum dose must not exceed 150gm
- Change to paste when powder begins to crust up
- Failure to remove all product before reapplication
- Failure to warn patient that some pain may be experienced

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**SILVER CONTAINING DRESSINGS**

Silver has been used for many years in particular in the treatment of burns as a Silver Sulphadiazine Cream. This cream has also been applied to some wounds.

The difficulty is the a cream is formulated to be applied to intact skin. When applied to a wound it encourages the development of muscilagenous slough.

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**Inadine**

**INADINE® PVP-I Non-Adherent Dressing** consists of a low adherent knitted viscose fabric impregnated with a polyethylene glycol (PEG) base containing 10% Povidone Iodine; equivalent to 1.0% available iodine.

**INADINE® dressings** are designed to protect the wound, even if infected. **INADINE®** is indicated for the management of ulcerative wounds and may also be used for the prevention of infection in minor burns and minor traumatic skin loss injuries.

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**SILVER CONTAINING DRESSINGS**

In recent year a range of dressings that Contain or combine Silver into their Structure have been released. They include:
- **High Density Polyethylene dressings** [Acticoat]
- **Foam Dressing** [Acticoat Moisture Absorbing]
- **Foam with Silicone Adhesive** [Mepilex Ag]
- **Alginate Dressing** [Acticoat Absorbent,]
- **Hydroactive Dressing** [Biatain Ag]
- **Hydrofibre Dressing** [Aquacel Ag]
- **Tulle Dressing** [Atrauman Ag]
Silver Dressings

The ACTICOAT® family are a unique range of antimicrobial barrier dressings for use over partial, full thickness and acute wounds. Unique Patented Silver technology: SILCRYST Nanocrystalline Silver Antimicrobial protection Effective barrier to over 150 wound pathogens¹ Faster kill rates, longer wear times

ACTICOAT Moisture Control (with SILCRYST™ Nanocrystals) is a 3-layer dressing consisting of the following: A nanocrystalline silver-coated polyurethane layer, a white polyurethane foam layer and a blue waterproof polyurethane film layer. ACTICOAT Moisture Control provides an effective barrier to bacterial penetration. In the presence of exudate the dressing will help maintain a moist wound environment. ACTICOAT Moisture Control may be left in place over a wound for up to 7 days.

Acticoat Flex 3 and 7

Acticoat Flex consists of a single layer of knitted polyester to ensure ultimate flexibility and comfort during wear time for the patient.

- Highly conformable and flexible
- One-way stretch improves patient mobility
- Open-weave allows fluid and exudate migration
- Pre-moisten the dressing for acute and low exuding wounds

Acticoat Flex is a knitted polyester weave which conforms to anatomical areas to maximise dressing contact with the wound. The dressing moves with the patient to facilitate a comfortable duration of wear and low wound adherence helps to minimise trauma on removal.

ALLEVYN® Ag Gentle & Ag Gentle Border

- Antimicrobial Hydrocellular Foam Dressing with Soft or Silicone Gel Adhesive for Low to Moderately Exuding Wounds
- Pricing comparable to ALLEVYN Ag
- All products in box of 10

ALLEVYN Ag Gentle & Ag Gentle Border
Silver Dressings

Mepilex Ag combines the unique features of Safetac technology with the bacteria reducing power of silver. Mepilex Ag goes to work quickly, inactivating wound pathogens within 30 minutes and for up to 7 days. At dressing removal, Mepilex Ag does not stick to the wound or strip surrounding skin, minimising patient pain and wound trauma.

*Silver Containing Dressings*

**Biatain** Absorbent polymer dressing containing silver. The silver is released into the wound as exudate is absorbed.

**Aquacel** is a hydrofibre of sodium carboxy-methylcellulose with the silver chemically attached to some of the polymer as Silver carboxy methylcellulose. As with Aquacel the hydrofibre absorb exudate into its structure and forms a gel sequentially releasing the silver.

**Silvercel** Antimicrobial Alginate Dressing is a sterile, non-woven pad composed of high tensile strength alginate, carboxymethylcellulose (CMC) and Silver coated fibers. SILVERCEL Antimicrobial Alginate Dressing contains elemental silver (8%) as a sustained release formulation.

**Newer Antiseptics**

**Prontosan** is a solution containing Polyhexanide a Biguanid antiseptic related to Chlorhexidine and Undecylenamidopropyl Betaine a surfactant.
Polyhexamethylene biguanide (PHMB)

Polyhexamethylene biguanide (PHMB) is a polymeric cationic antimicrobial agent that has been deployed in consumer applications for over 40 years. Whilst it shares many attributes with the simpler cationic agents it has additional action mechanisms that render it unique amongst this generic class of antimicrobials.

The toxicity profile of both the biguanides and the polymeric biguanides is excellent. Neither molecule is a primary skin irritant nor a hypersensitising agent. With respect to the deployment of PHMB as part of a wound care system there is little or no evidence to suggest that this would lead to the emergence of PHMB resistant. Use of the agent within a barrier wound dressing such as Kerlix AMD would impair the growth and penetration through the dressing of adventitious pathogens both from the environment to the dressed wound.

Undecylenamidopropyl Betaine

Betaine is a very mild, active surfactant with a dual water and oil solubility. Betaine is exceptionally mild. The action is to reduce surface tension and allow wound contaminants to lift.

In respect to its action on Biofilm this is based on one in-vitro study by H.-M. Seipp Efficacy of Various Wound Irritants against Biofilms. This lab study Examined the effect of Saline, Ringers solution and Prontosan on Biofilm on the surface of silicone tubes.

Flaminal

Flaminal is available as two hydrogels with a high alginate content which are promoted for the reduction of bacterial growth in wounds.
Flaminal

Flaminal® contains lactoperoxidase which is an enzyme extracted from milk and acts as an important natural antimicrobial (Banks et al, 1986). It has been shown to be bacteriostatic against Gram-positive organisms and exhibits pH-dependent bactericidal action against Gram-negative organisms in the presence of hydrogen peroxide and thiocyanate. (Richard White: Wounds UK, 2006, Vol 2, No 3)

Sorbact

Sorbact® - mechanism of action

- Bacteria and fungus bind to surfaces via hydrophobic interaction.

When two hydrophobic particles come in direct contact the bind together with the binding force of the surrounding water molecules = Hydrophobic interaction

Green Wound Healing

Non-antibiotic
Non-antiseptic
Anti-microbial Wound Management

What makes Sorbact® unique?

Sorbact uses the same binding process that bacteria and fungus use to bind to surfaces, hydrophobic interaction.

Sorbact applied directly on the wound surface.

Pathogenic microorganisms bind to the Sorbact surface and become inactivated.

The bound microorganisms are removed when the dressing is changed.
Common wound pathogens

- Staph. aureus
- Psuedomonas
- E. coli
- Streptococcus
- Candida albicans

Hydrophobic properties

Hydrophobicity of microorganisms

- Enterococci 94%
- Staph. aureus 90%
- E. coli 85%
- Candida albicans 84%

Areas of use

- All wounds regardless of etiology, exudate level and wound condition.
- Fungal infections: skin folds and in between toes.
- Microbial prophylaxis on acute, chronic, traumatic and post surgical wounds.

Sorbact® areas of use

- Infected Wounds
- Critically colonized
- Hard to heal wounds
- Prevention

Fungal infection in skin folds
- Athletes foot

Frequency of dressing changes

**Recommended frequency of dressing changes**

- Infected wounds: Initially once daily
- Collonized/Critically colonized wounds: According to exudate level every 2 – 3 days

The frequency will be decreased according to wound status improvement

- "Clean" wounds: 2 times/week or when needed

Other antiseptics

- Honey
  - Antibacterial effect, also anti-fungal
    - bacteriostatic
    - tested against (at different dilutions)
      - staph aureus spp (inc MRSA), pseudomonas spp with inhibition of both
  - Daily dressings usual
    - may be up to tds depending on exudate
  - Contains H2O2
    - hydrogen peroxide in "slow-release" form due to enzyme action (antiseptic)
Honey positives
- High osmolarity
  - reduce oedema and maceration
  - unsuitable environment for bacterial growth
- Low pH
  - inhibit cell growth
- Produce enzymes
  - may promote slough separation
- May reduce odour
- Inexpensive??
- Easily obtained
- Easy to apply
- May reduce pain
- Anti-inflammatory??

Honey negatives
- Infection not biggest problem in chronic wounds
- Doesn’t address underlying causes
  - vascular problems
  - pressure, shear, etc
  - diabetes
- Anti-bacterial activity can vary by up to 100-fold from one batch to next
- Pain in some patients
- Hydrogen peroxide not recommended in wound care
  - Feeble antiseptic and may cause oxygen emboli
- May contain bacteria or spores if unsterilised

Other antiseptics

<table>
<thead>
<tr>
<th>Product</th>
<th>Type</th>
<th>Cytotoxicity</th>
<th>Biofilm effect</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Super oxidized with Hypochlorous Acid (HOCl) and Sodium Hypochloride (NaOCl)</td>
<td>antimicrobial</td>
<td>Some</td>
<td></td>
<td>Comes in a spray-on format solution and gel</td>
</tr>
<tr>
<td>Octenidine dihydrochloride (OCT)</td>
<td>Surfactant</td>
<td></td>
<td>Some</td>
<td>Comes in a gel and irritation preparation that can be used together or separately</td>
</tr>
<tr>
<td>PHMB / Betaine</td>
<td>Surfactant</td>
<td></td>
<td>Some</td>
<td>There is little or no evidence to suggest that this would lead to the emergence of PHMB resistant.</td>
</tr>
</tbody>
</table>

Topical antibiotics

Topical Antibiotics are used in wounds
- Silver Sulphadiazine in burns and in wounds
- Problems: Delivery method a cream
- Mixed with a Hydrogel this alters the base
- Metronidazole: an anti anaerobic antibiotic
- Problems used as crushed tablets
- Mupiricin: a specific topical antibiotic with no similar Compounds used systemically or orally

Resources
- AWMA Guideline "Bacterial impact on wound healing: From contamination to infection"
- EWMA Guideline "Management of wound infection"
- TIME resources
Conclusion

Despite the use of many other antiseptics in a wide range of situations evidence supporting their efficacy in the treatment of wound infection is more limited. Clinicians will use newer products however it is important that further good clinical research to be undertaken and published to validate their use in wound management.