Microbe of the month

Breaking The Chain of Infection

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Compiled by
Helen Loudon IPC Consultant

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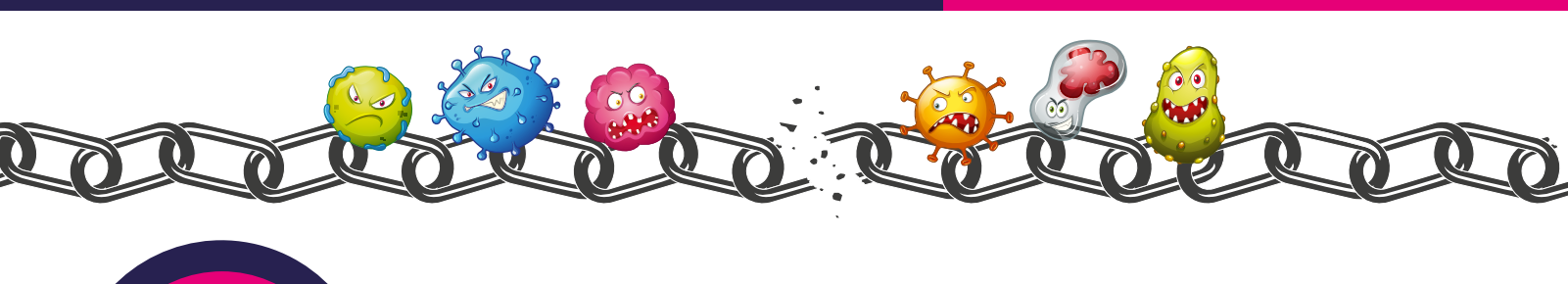


Featured this month:

Microbial Biofilm

A protective bullet-proof vest!

Hello readers!

The aim of the Microbe of the Month newsletter is to help create awareness about microorganisms of clinical importance, in an easy to read and understand format. Each newsletter provides insights into prevalent healthcare-related pathogens and those aspects which are relevant to Infection Prevention and Control (IPC), and Antimicrobial Stewardship (AMS) practice.

Please use this newsletter as a teaching tool in your workplace, share it widely with colleagues and start an 'infectious dialogue' about topical issues in infection control!

The formation of biofilm by microorganisms is an ancient survival strategy.
It enhances microbial survival and reproduction, provides a medium for the transfer of antibiotic-resistant genes, and enables microbial tolerance to environmental threats (e.g., antiseptics and topical antimicrobial dressings).

Biofilm is the primary cause of chronic infections, such as otitis media, those associated with indwelling medical devices (e.g., catheters, surgical implants), infections associated with cystic fibrosis, osteomyelitis, rhinosinusitis, and chronic wound infections.

Biofilm should also be considered synonymously with antimicrobial resistance (AMR) because of its proficiency in transferring resistance genes, as well as its protective function and innate tolerance to antimicrobial agents.

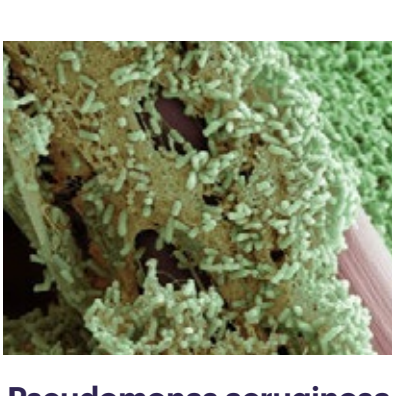
Greater awareness of the existence and consequences of microbial biofilm is crucial to improving hygiene practices and controlling the emergence and spread of antimicrobial resistance in healthcare facilities.[1,2]

Key words: *biofilm structure, extracellular polymeric substance (EPS), virulence, gene transfer, quorum sensing, antimicrobial resistance (AMR).*

WHAT IS A BIOFILM? [3,4,6,7]

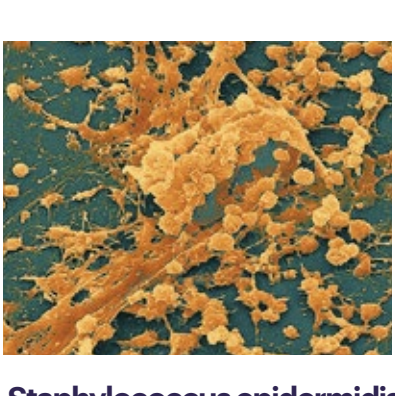
The hallmark of a biofilm is the secretion of a slimy 'extracellular polymeric substance' **(EPS)** by clusters of mixed microbial species. EPS comprises polysaccharides, proteins, lipids and glycoproteins, and serves a variety of purposes, the most important of which is to provide attachment to living or non-living surfaces.

- Bacterial cells encased in biofilm EPS are different than free-floating, planktonically growing bacteria.
- Benign or commensal biofilms include those found on the skin or in the gastrointestinal tract. Commensal biofilms protect the human body from infection and disease.
- 'Benign biofilms' can modify their behaviour to become 'pathogenic' or 'virulent' – upregulating their genetic and biochemical processes to actively protect the polymicrobial communities within them (i.e., a type of 'biological bunker').
- The protective EPS provides a strong foundation and a 'house-like' structure which completely envelops the microorganisms, and is highly resistant to penetration by antimicrobial agents and the immune system (e.g., phagocytic leukocytes and antibodies).
- A biofilm structure has water channels that bring nutrients and water to, and waste away from, the lower layers of the biofilm.
- Biofilms increase the opportunity for the transfer of antimicrobial-resistance genes carried on mobile genetic elements, such as plasmids.
- Genetic transfer can even occur between bacteria of different species, which increases the potential for virulent, multi-drug-resistant and persistent infections.
- The nutritional, oxygen and metabolic requirements of microbes are reduced in biofilm. This is termed 'metabolic quiescence' (a type of hibernation) – promoting the inclusion of anaerobic species, as well as the ability to tolerate the effects of topical and systemic antimicrobial agents which would otherwise be quite lethal to planktonic (free-floating) bacteria.
- This leads to more rapid and excessive development of biofilm and EPS, the excessive production of degrading enzymes (matrix metalloproteinases or MMPs), enhanced generation of 'signalling' molecules (known as 'quorum sensing'), microbial proliferation and dissemination.
- **Collectively, these strategic behaviours stimulate an inflammatory immune response and predispose to the development of infection.**



Pseudomonas aeruginosa biofilm on a toothbrush bristle.

MICROBIAL BIOFILMS are thought to be the root cause of approximately 80% of all infections in humans, and most medical device-related infections.[2]



Staphylococcus epidermidis biofilm on the lumen of a vascular catheter.

HOW DO BIOFILMS FORM?

Biofilm formation is dynamic and typically involves the following stages:

STAGE 01 – **Reversible surface attachment:** microorganisms are commonly perceived to be free-floating and solitary (i.e., planktonic). However, under natural and moist conditions microorganisms tend to attach to surfaces and eventually form biofilms. The initial attachment is reversible.

STAGE 02 – **Irreversible (permanent) surface attachment:** as the bacteria multiply, they secrete EPS and become more firmly attached (sessile). They also differentiate, changing gene expression patterns in ways that promote survival. This is usually the result of a type of bacterial communication known as 'quorum sensing'.

STAGE 03 – **Biofilm growth and differentiation** culminating in a mature biofilm community with characteristic structural features such as water channels and towering clusters of cells.

STAGE 04 – **Detachment and dispersion of biofilm cells or fragments** which form new biofilm microcolonies.

Watch this video (5 mins) on Biofilm Formation
https://www.youtube.com/watch?v=Aa8WE2LOOcQ

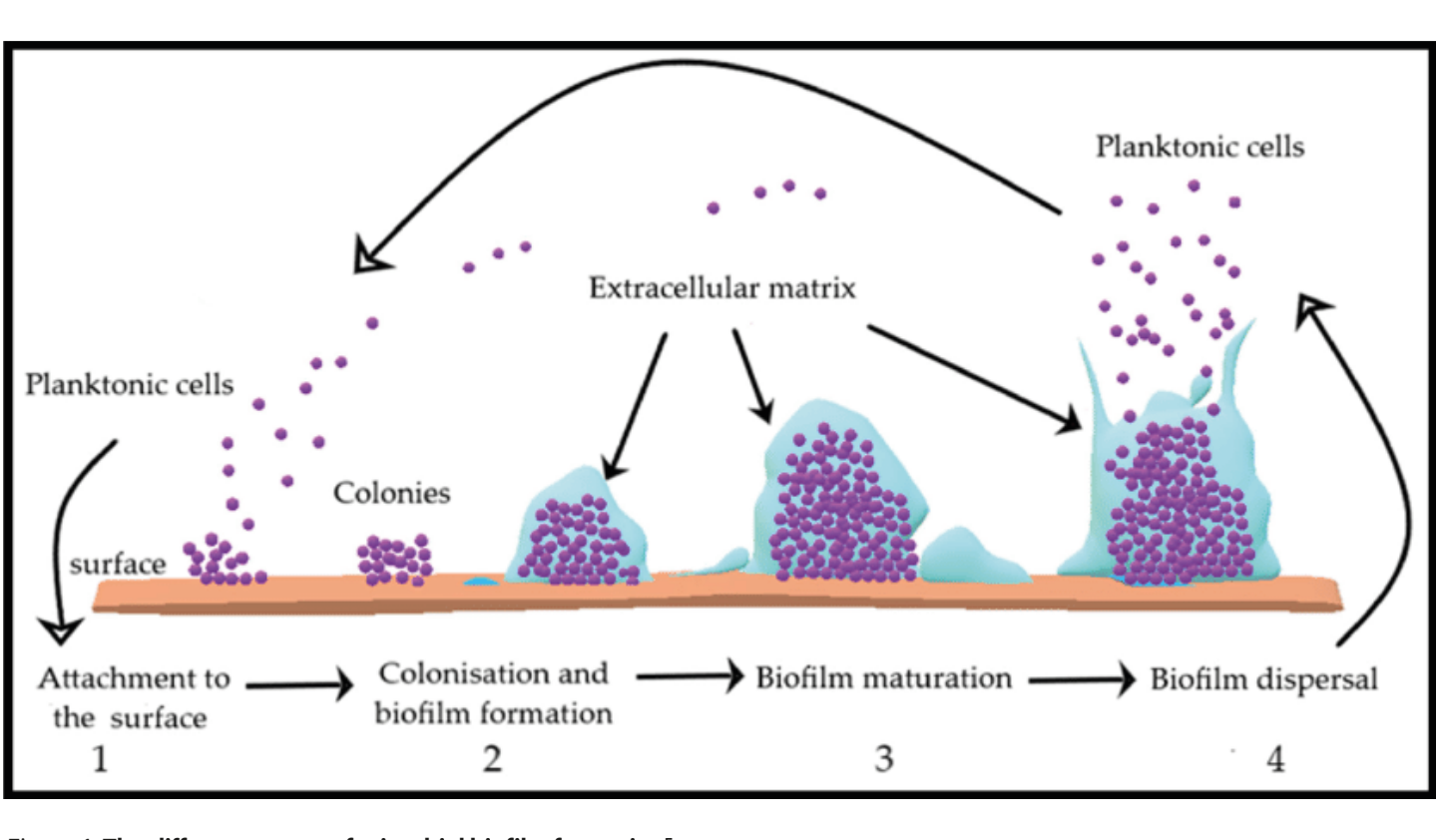


Figure 1. **The different stages of microbial biofilm formation**[5]

THE RELEVANCE OF BIOFILM IN SEPTIC AND CHRONIC WOUNDS [1,3,4,6,7]

Wound hygiene is a fundamental aspect of care for all patients with an open wound **and it should be assumed that all hard-to-heal wounds contain biofilm**. However, wound biofilms are difficult to visualise macroscopically - slough, debris and fibrin layers may be visually mistaken for biofilm.

Biofilms delay wound healing by eliciting a chronic inflammatory response - this predisposes to increased exudate production and tissue breakdown.

CLINICAL SIGNS OF BIOFILM ACTIVITY

- Delayed healing despite optimal wound management and support of underlying patient comorbidities
- Failure or recalcitrance to appropriate topical and/or systemic antimicrobial treatment
- Increased exudate/moisture
- Low-level chronic inflammation
- Peri-wound erythema and skin breakdown
- Poor granulation or friable, bleeding hypergranulation
- Covert signs of infection

EPS is a mechanical barrier to antiseptics, antibiotics and immune cells, which decreases their effectiveness and may result in treatment failure.
Ongoing local debridement (using a method appropriate for the wound type and patient's risk profile) and mechanical cleansing (including topical wound 'soaks' for 10-15mins) with a biofilm-active agent will disrupt biofilm structures and remove pro-inflammatory proteases and debris.

In septic and heavily colonised wounds, antimicrobial dressings alone are not sufficient to disrupt and remove biofilm. They should be changed timeously, and used as a supportive measure to control local bioburden and slow down the inevitable re-formation of biofilm in between dressings.

This could, in turn, reduce antibiotic usage in wound care.[8]

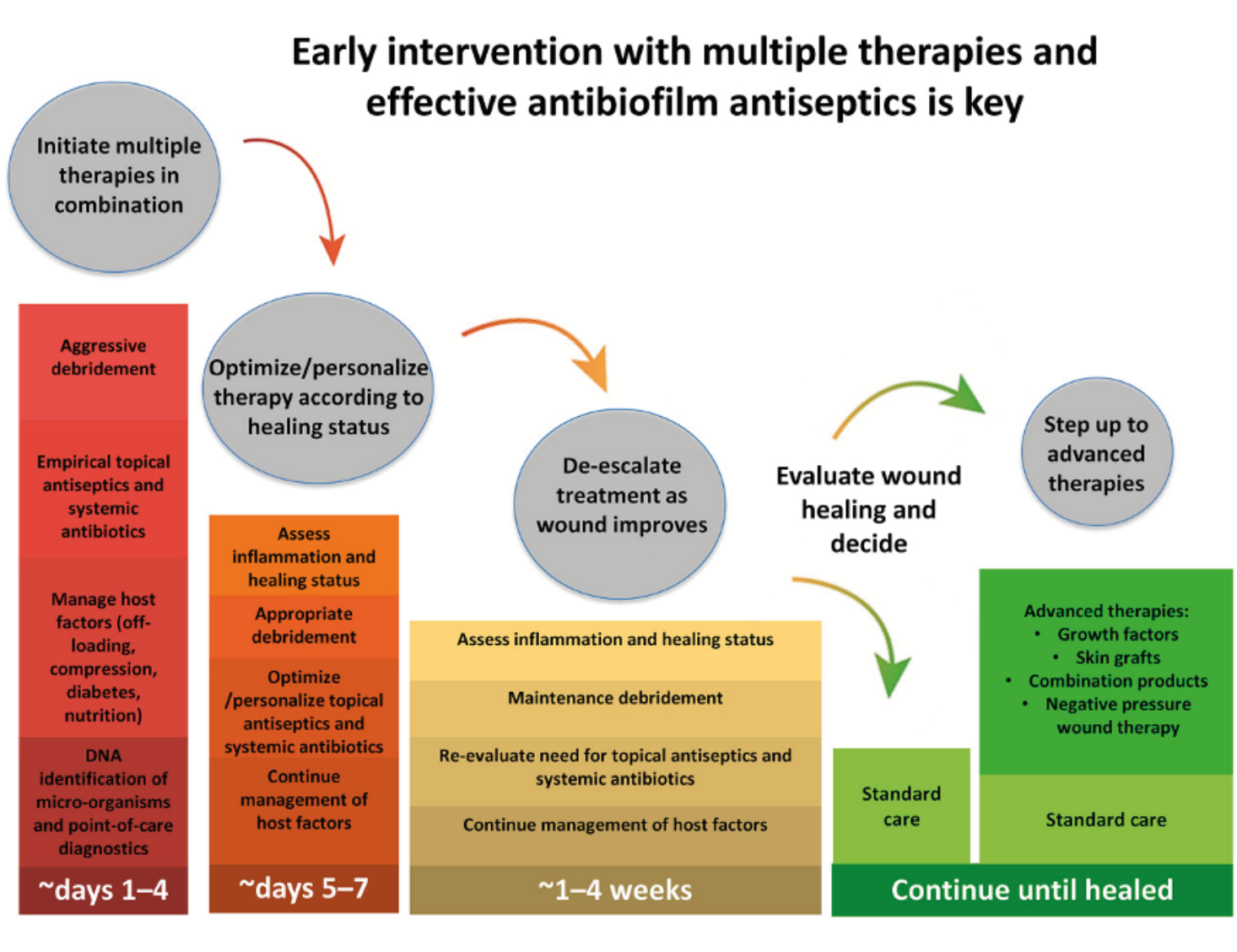


Figure 2. **The step-down/step-up approach to biofilm-based wound care**[6]

THE BOTTOM LINE... [6,7,8]

- **Microbial biofilms are everywhere**, especially in warm, dark and moisture-rich environments.
- **Biofilms have a high affinity for plastics, invasive medical devices, implants and non-healing wounds.**
- The polymicrobial nature of biofilm increases infection virulence and complicates treatment.
- Avoid the use of invasive devices where possible, and practice strict aseptic techniques for their insertion and aftercare. Remove vascular lines and drains at the earliest opportunity.
- Chronic wounds with high exudate levels, or the presence of slough or infection, present ideal conditions for the development of biofilm.
- Care plans should always include wound bed preparation (WBP) measures such as local debridement, the use of dressings which promote a moist wound healing environment but absorb excess exudate and the use of topical antimicrobial (not antibiotic) agents which are able to disrupt and reduce biofilm structures in septic and heavily colonised wounds.
- Examples of 'biofilm-active' microbicidal wound cleansers are hypochlorous acid, polyhexanide biguanide (PHMB) and povidone iodine.
- Practice '**standard precautions**' (i.e., face shields, gloves, plastic disposable aprons, etc.) for contact with blood, body fluids, secretions, mucous membranes, and contaminated items such as drainage tubes.
- Always ensure that patient care equipment is properly cleaned and disinfected prior to re-use on another patient.
- Apply alcohol-based hand rub frequently during tasks when hands are not visibly soiled, and after touching a patient, their environment, or their possessions.
- Wash hands promptly after contact with infective material, and always after glove removal.
- Clean and disinfect environmental surfaces with a sodium hypochlorite-based **detergent-based** cleaner, using colour-coded cloths and cleaning equipment.

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