The dangers of bacterial colonization in wounds are well understood by caregivers — in fact the modern peripatetic wound care team is part of a vast international network of wound care professionals working to prevent and treat infections. The challenge of resistant bacteria is that they do not respond as expected to the safety measures normally implemented in a wound care setting. While these interventions may have been effective for previous infections, they are no longer sufficient. The need for more effective techniques to prevent infection of wounds.

Mechanism of resistance generation to antiseptic agents

The mechanism of bacterial resistance depends on the mode of action of the antiseptic agent. While bacteria do not develop resistance to antibiotics, they do develop resistance to antiseptics. This is because antiseptics are exposed at high concentrations, which are more than enough to inhibit bacterial growth. The mechanism of resistance is similar to both antibiotics and antiseptics.

Antiseptics: diversity of action

There are a wide variety of antiseptic agents that have been utilized as topical antimicrobials, and as active ingredients for antimicrobial wound dressings. Not only below are some small-molecule antiseptic agents.

Bactericidal activity

Many of our small-molecule antiseptic agents are characterized by their activity against bacteria. Our agents are designed to inhibit the growth of bacteria, and to kill the bacteria that are already present. This is achieved by a variety of mechanisms, including the inhibition of bacterial enzymes, the disruption of bacterial membranes, and the inhibition of bacterial replication.

Antibiotic agents typically target specific metabolic processes. Defective cells are killed by the antibiotic, which inactivates the metabolic pathway. However, bacteria are able to acquire resistance to these agents by altering the antibiotic target site, or by developing alternative pathways. These processes are fundamental to the development of antibiotic resistance.

Antiseptic agents

Antiseptic agents can be categorized into two groups: those that kill bacteria, and those that inhibit bacterial growth. The classification of these agents is based on their mode of action, and the level of activity required to achieve a therapeutic effect.

Silver agents

Silver agents represent the bulk of the market for the US. Silver based antimicrobials exist in various chemical forms, including metallic silver (Ag), silver hydrosol (Ag2O), silver nitrate (AgNO3), silver chloride (AgCl), silver citrate (Ag3Cit3), silver thiosulfate (Ag3St3), silver sulfadiazine, silver sulfacetamide, and silver sulfadiazine with metronidazole. The same is true for silver compounds as they are fundamental to the development of antibiotic resistance.

Cationic agents

Cationic agents include a range of agents that can be chemically distinguished through the number of charge sites resolved on the molecular (or molecular analog) for peptides and proteins for peptides and proteins. Monomeric quaternary ammonium compounds are known for their activity against Gram-negative bacteria. The smallest molecules are known for their activity against Gram-positive bacteria, while the larger molecules are known for their activity against Gram-negative bacteria. The most active of these compounds is the quaternary ammonium compound, which is used in a variety of formulations.

Figure 4 (above). Determination of microbial activity of NIMBUS surface and efficacy of NIMBUS rinses. The evaluation changes to bacterial resistance to NIMBUS biocide with the step-by-step adaptation training of E. coli. E. coli culture in the active surface of the NIMBUS dressing. Sequential assessment of the minimum inhibitory concentration (MIC) for NIMBUS rinses was conducted to determine the selection vector was created by exposing serial passages of bacteria to the NIMBUS rinses. The MIC for NIMBUS rinses was determined by an automatic and propagated into near inoculum. Exposure to the treated substance was repeated for 24 hours. This resulted in stepwise exposure to E. coli and did not become resistant to NIMBUS after a prolonged and repeated exposure.

References

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2005 viability of E. coli was ± 94% at an concentration of 250 µg in mm. After repeated exposure to NIMBUS biocide after step-by-step adaptation training of E. coli, the E. coli became resistant to NIMBUS after a prolonged and repeated exposure.

Figure 5 (left). SEM imaging of E. coli on untreated wound dressing and on NIMBUS treated wound dressing. NIMBUS does not penetrate into the cell in order to exert antibacterial activity but deactivates the cell wall without inducing cell death. As demonstrated by the high resolution SEM images in Figure 5, the chemistry of the cell wall is relatively immature, so the generation of resistance to this mechanism is extremely unlikely.