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# Editorial

## Contents



**Mini Review Section** – MRSA, (***methicillin-resistant staphylococcus aureus***), is a form of bacterial infection that is resistant to numerous antibiotics including methicillin, amoxicillin, penicillin and oxacillin, thus making it challenging to treat the infection successfully.

Often referred to as a superbug, MRSA infection may commence as a minor skin sore, pimple or boil, before becoming serious, potentially harmful and sometimes fatal. Infections with MRSA are more common in people in hospital wards or nursing homes. It can also cause more serious skin infections or infect surgical wounds, the bloodstream, the lungs, bone or the urinary tract.

**Current Trends** – The antiseptic agent polyhexamethylenebiguanide (also known as polihexanide or PHMB) has been used for over 60 years in a wide range of applications from swimming pool sanitisers to preservatives in cosmetics and contact lens solutions. In Europe, it has been available as a wound irrigation fluid for some time.

PHMB is a fast-acting biguanide compound composed of a synthetic mixture of polymers. The compound is structurally similar to the antimicrobial peptides (amps) produced by many cells within the wound, such as keratinocytes and inflammatory neutrophils, where they are thought to help protect against infection. Amps have a broad spectrum of activity against bacteria, viruses and fungi, inducing cell death by disrupting cell membrane integrity.

**In Profile Section – "**Maurice Hilleman" was responsible for developing more than 40 vaccines, including measles, mumps, hepatitis A, hepatitis B, meningitis, pneumonia, *Haemophilus influenzae* bacteria, and rubella. His vaccines have been credited with saving millions of lives and with eradicating common childhood diseases. The measles vaccine alone has prevented approximately one million deaths. Among other accomplishments, he succeeded in characterising and isolating many viruses, including the hepatitis A vaccine in culture.

**Bug of the Month** - *Cronobacter sakazakii* represents a significant risk to the health of neonates. This bacterium is an emerging opportunistic pathogen that is associated with rare but life-threatening cases of meningitis, necrotizing enterocolitis, and sepsis in premature and full-term infants. Infants aged <28 days are considered to be most at risk. Feeding with powdered infant formula (PIF) has been epidemiologically implicated in several clinical cases. Infants should be exclusively breast-fed for the first 6 months of life, and those who are not should be provided with a suitable breast-milk substitute.

**Did You Know?** The preservation of foods by heat has probably been practiced by man since the discovery of fire. Although sterilisation is an absolute term which usually means the complete destruction of all forms of life, substantial food preservation can be achieved by less than complete sterilisation. Pasteurisation is such a treatment: demonstrated that heating liquids, especially wines, to fairly low temperatures, such as 60°C, improved the keeping quality during storage. This low-temperature heat treatment destroyed spoilage organisms, but was low enough so as not to destroy the original characteristics of the liquid being treated.

**Best Practices -** Water purification is the process of removing undesirable chemicals, biological contaminants, suspended solids and gases from contaminated water. The goal is to produce water fit for a specific purpose. Most water is disinfected for human consumption (drinking water), but water purification may also be designed for a variety of other purposes, including fulfilling the requirements of medical, pharmacological, chemical and industrial applications.

All work & no play make Jack a dull boy! We don't forget that ever. Each issue comes with its own bouquet of jokes ……

So go on, explore the information……..

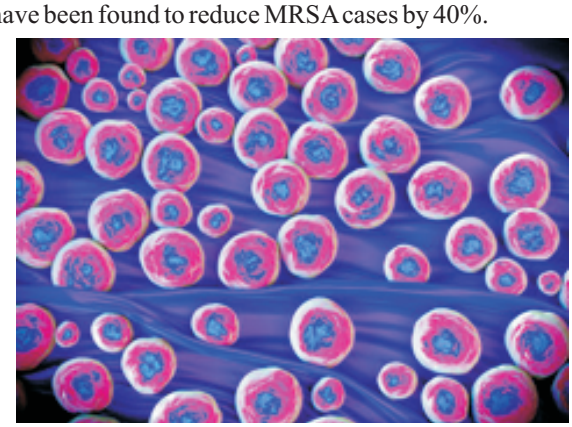
# MRSA: Causes, Symptoms, Prevention and Treatments

MRSA, (***methicillin-resistant staphylococcus aureus***), is a form of bacterial infection that is resistant to numerous antibiotics including methicillin, amoxicillin, penicillin and oxacillin, thus making it challenging to treat the infection successfully.

Often referred to as a superbug, MRSA infection may commence as a minor skin sore, pimple or boil, before becoming serious, potentially harmful and sometimes fatal. Infections with MRSA are more common in people in hospital wards or nursing homes. It can also cause more serious skin infections or infect surgical wounds, the bloodstream, the lungs, bone or the urinary tract.

Here are some key facts about MRSA.

- "Methicillin" represents the semisynthetic penicillin-related antibiotic once effective against *staphylococci* (*staph*). *Staph* bacteria have developed a resistance to penicillin-related antibiotics, including methicillin - these resistant bacteria are called *methicillin-resistant staphylococcus aureus*, or MRSA.
- MRSA is a form of bacterial infection that is resistant to numerous antibiotics.
- MRSA infection can begin as a minor skin sore or pimple and become potentially harmful.
- "Methicillin" represents the antibiotic that was once effective against *staphylococci* (*staph*).
- "*Staphylococcus aureus*" refers to a bacterium that commonly resides inside the nose and human skin.
- Around one in three (33%) people carry staph in their nose, usually without any illness. Two in 100 people carry MRSA.
- MRSA can be divided between health care associated MRSA and community associated MRSA.
- The bacterium that can cause *staph* skin infections can divide every half-hour in optimum conditions. Theoretically, a single cell can form a colony of more than a million cells in 10 hours.
- Annually, there are around 94,360 invasive MRSA infections diagnosed in the US, with 18,650 associated deaths.
- It is estimated that 49-65% of health care associated *S. aureus* infections are caused by methicillin-resistant strains.
- Invasive health care associated MRSA infections declined by 54% between 2005 and 2011.
- Approximately 86% of all invasive MRSA infections are contracted with health care settings.
- Germ-killing soaps and ointments used in intensive care units have been found to reduce MRSA cases by 40%.



Since the 1960s, MRSA has picked up more resistance to different antibiotics. Overuse of antibiotics has increased resistance in MRSA and other infectious bacteria, because resistance genes (the genes that code for resistance) can be passed from bacteria to bacteria.

If a doctor orders a test for bacteria on a specimen of pus, for example, the laboratory will alert the doctor if the test shows MRSA, so that precautions can be taken, and the right treatment can be started. A deadly complication of MRSA is a deep infection, necrotizing fasciitis, which causes rapid spread and destruction of human tissues. Some but not all strains of MRSA are more likely to behave like "flesh-eating bacteria." It is impossible to predict which MRSA infection will be "flesh-eating."

People with higher risk of MRSA infection are those with skin breaks (scrapes, cuts, or surgical wounds) or hospital patients with intravenous lines, burns, or skin ulcers. In addition, MRSA may infect people with weak immune systems (infants, the elderly, people with diabetes or cancer, or HIV-infected individuals) or people with chronic skin diseases (eczema and psoriasis) or chronic illnesses. People with pneumonia (lung infection) due to MRSA can transmit MRSA by droplets produced during coughing. Patients in health-care facilities are often in these risk categories, so special precautions recommended by CDC may be posted on a sign at the room entrance. Examples include "Droplet precautions" -- if the patient has pneumonia, disposable masks, gowns, and gloves must be used by people who enter the room, and they must be taken off before leaving. "Contact precautions" may be posted recommending gowns and gloves only if the patient has skin infection. Precautions must be followed as posted by both health-care professionals and visitors to keep from spreading MRSA to other patients or people at risk of serious infection.The incubation period (time between infection and start of symptoms) is variable and may depend on the particular strain of MRSA and the person's immunity. Most MRSA infections are skin infections that produce the following signs and symptoms:

**Cellulitis**, an infection of the skin or the fat and tissues under the skin, usually starting as small red bumps in the skin. It includes redness, swelling of the tissues, warmth, and tenderness.

**Boils** (pus-filled infections of hair follicles).

**Abscesses** (collections of pus in or under the skin).

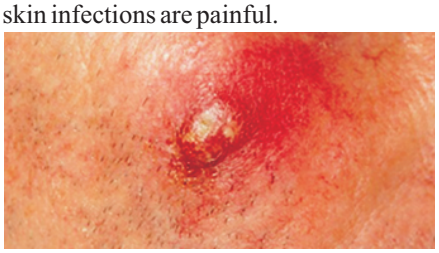
**Sty** (an infection of an oil gland of the eyelid).

**Carbuncles** (infections larger than an abscess, usually with several openings to the skin).

**Impetigo** (a skin infection with pus-filled blisters).

**Rash or skin redness** (skin appears to be reddish or have red-colored areas).

All of these skin infections are painful.



A major problem with MRSA (and occasionally other staph infections) is that occasionally the skin infection can spread to almost any other organ in the body. When this happens, it is a deep or invasive infection that can spread to the blood and infect internal organs. MRSA infections can cause complications such as infection of heart valves (endocarditis), gangrene or death of the soft tissues (necrotizing fasciitis), and bone or joint infections (osteomyelitis or septic arthritis). This can be deadly. Fever, chills, low blood pressure, joint pains, severe headaches, shortness of breath, and rash over most of the body are symptoms of sepsis (blood poisoning), which requires emergency medical attention.

**Causes of MRSA :**

**Ultimately MRSA is caused by bacterium strains that have acquired a resistance to particular antibiotics.**

MRSA can spread from person to person (skin-to-skin contact) and from person to object to person when an individual has active MRSA or is colonized by the bacteria.

Skin-to-skin contact with someone carrying MRSA is not necessary for infection to spread. MRSA bacteria are also able to survive for extensive periods on surfaces and objects including door handles, floors, sinks, taps, cleaning equipment and fabric. One study to determine the survival of resistant *staph* on common hospital surfaces looked at *staph* survival on five materials commonly found in hospital.

- 100% smooth cotton (clothing)
- 100% cotton terry (towels and wash cloths)
- 60% cotton-40% polyester blend (scrub suits, lab coats and clothing)
- 100% polyester (privacy drapes, curtains and clothing)
- 100% polypropylene plastic (splash aprons).

Swatches of fabric were injected with 10,000 to 100,000 colony-forming units (CFU) of the microorganism and observed daily. Results showed *S. aureus* survived on the materials for the following number of days:

- Cotton: 4-21 days
- Terry: 2-14 days
- Polyester blend: 1-3 days
- Polyester: 1-40 days
- Polypropylene: 40-greater than 51.

These results conclude the need for thorough contact control and meticulous disinfection procedures to limit spread of bacteria.

**Health care-associated MRSA**

MRSA frequently causes illness in people with a compromised immune system who interact with or reside in hospitals and health care facilities. This is referred to as health care-associated MRSA (or hospital-acquired MRSA) and often occurs for one of the following reasons.

- A break in the skin barrier, such as a surgical wound, burn, catheter or intravenous line that allows bacteria to enter the body
- Older age, comorbidities or multiple complex health issues, and weakened immune systems due to a specific health condition or the use of medications that lower immune function
- The simple fact that hospitals and health care facilities are visited by large numbers of people, both patients and staff, providing an environment for bacteria to easily spread from person to person or person to object to person.

Those with a weakened immune system can include:

- Patients in hospital for a long period of time.
- Patients on kidney dialysis (hemodialysis).
- Open wounds.
- A catheter or intravenous drip inserted.
- Burns or cuts to the skin surface.
- Severe skin conditions.
- Frequent antibiotics as part of their treatment.
- Patients receiving cancer treatment or specific medications that affect immune function.
- Those who inject illegal drugs.
- Individuals who have had surgery within a year of being back in hospital.

**Estimates suggest that 49-65% of health care-associated *S. aureus* infections are caused by methicillin-resistant strains.**

According to the Centers for Disease Control and Prevention (CDC) study, invasive health care-associated MRSA infections declined 54% between 2005 and 2011, with 30,800 fewer severe MRSA infections and 9,000 fewer deaths.

A similar study conducted by the National Healthcare Safety Network (NHSN) found that rates of health care-associated MRSA bloodstream infections fell nearly 50% from 1997-2007.

The average age of a person with health care-associated MRSA was 68.

**Community-associated MRSA :**

Community-associated MRSA is contracted external to the hospital and is less widespread compared with health care-associated MRSA. Factors that cause increased risk of developing community-associated MRSA include:

- Living in an environment with a lot of people; military bases, jail, on-campus housing.
- Regular skin-to-skin interaction for example in contact or collision sports such as rugby, ice hockey, soccer and basketball.
- Cuts or grazes to the skin or regular injection of drugs.
- Contaminated surfaces.
- Unhygienic facilities or lack of personal hygiene.
- Previous antibiotics use.

**How is a MRSA infection transmitted or spread?**

There are two major ways people become infected with MRSA. The first is physical contact with someone who is either infected or is a carrier (people who are not infected but are colonized with the bacteria on their body) of MRSA. The second way is for people to physically contact MRSA from objects such as door handles, floors, sinks, or towels that have been touched by a MRSA-infected person or carrier. Normal skin tissue in people usually does not allow MRSA infection to develop; however, if there are cuts, abrasions, or other breaks in the skin such as psoriasis (a chronic inflammatory skin disease with dry patches, redness, and white scales), MRSA (or any S. aureus) may proliferate. Many otherwise healthy individuals, especially children and young adults, do not notice small skin imperfections or scrapes and may be lax in taking precautions about skin contacts. This is the likely reason MRSA outbreaks occur in diverse types of people such as school team players (like football players or wrestlers), dormitory residents, and armed-services personnel in constant close contact.

**What tests do health-care professionals use to diagnose a MRSA infection?**

Most doctors start with a complete history and physical exam of the patient to identify any skin changes that may be due to MRSA,

especially if the patient or caretaker mentions a close association with a person who has been diagnosed with MRSA. If possible, a sample of pus from a wound, blood, or urine is sent to a microbiology lab and cultured for *S. aureus*. Deep infections (such as bone) may require removal of a piece of tissue for testing (biopsy). If *S. aureus* is isolated (grown on a petri plate), the bacteria are then exposed to different antibiotics, including methicillin. *S. aureus* bacteria that grow well when methicillin is in the culture are termed MRSA, and the patient is diagnosed as MRSA-infected. Often there is no material to culture, and doctors treat the person with antibiotics that kill MRSA as well as more common bacteria until more information is available. This is called empiric therapy, meaning that doctors make their best guess on what bacteria are likely to be the cause of infection, until the bacteria have been definitively identified.

Some hospitals may screen patients for carrying MRSA, so that precautions can be taken to avoid spreading MRSA. The same procedure is done by swabbing the skin or nose. These tests help distinguish MRSA infections from other skin changes that often appear initially similar to MRSA, such as spider bites or skin changes that occur with Lyme disease. Many MRSA infections get mistaken for a spider bite. This can cause delayed or incorrect treatment and progression of the MRSA infection.

There are rapid screening tests that can detect the presence of MRSA DNA material (polymerase chain reaction, PCR) in a blood sample in as little as two hours. The test is able to determine whether the genetic material is from MRSA or from less dangerous forms of staph bacteria. It may allow hospitals to start precautions early. It may also allow doctors to quickly tailor the antibiotics to only what is needed; this reduces unnecessary antibiotic use and helps reduce antibiotic resistance. It also may reduce side effects and costs of unnecessary antibiotics. These tests cannot be used alone for the diagnosis of a MRSA infection. They do not provide important details about the antibiotics to which the specific strain is susceptible.

**Treatment and prevention of MRSA :**

If MRSA is diagnosed, treatment will vary subject to the following factors:

- Type of infection
- Location of the infection
- Severity of the symptoms
- Antibiotics to which the specific strain of MRSA responds.

Management of MRSA skin and soft tissue infections may include:

- Pus drainage from lesion
- Drained material sent for culture and susceptibility testing
- Patients being provided with information on wound care and hygiene
- Antimicrobial therapy (in cases of possible cellulitis without abscess).

Treatment options for MRSA skin and soft tissue infections may include:

- Clindamycin
- Tetracycline drugs - Doxycycline and Minocycline
- Trimethoprim and Sulfamethoxazole
- Rifampin
- Linezolid.

Research suggests that certain probiotic strains may help reduce susceptibility to active infection with MRSA. Reduced diversity and strength of the gut microflora leaves us vulnerable to opportunistic infections, while *Lactobacillus* species such as *paracasei*, and *L. acidophilus*, as well as *Bifidobacteria animalis* subsp l*actis* have been seen to offer a degree of protection against MRSA.

**What If you have an MRSA infection, you may need treatment with particular antibiotics.**

**Decolonisation** : If MRSA screening shows that you just carry MRSA on your skin, you'll need decolonisation treatment to remove the bacteria. Decolonisation involves using antibacterial products, such as a body wash or powder, to remove the MRSA bacteria from your skin. During the decolonisation process, you should wash every day, ideally using a fresh towel to dry yourself each time. You should also wear a new set of clothes each day and try to change your bedding on a daily basis. The resulting laundry should be washed at a high temperature separately from other people's clothes and bedding. An antibacterial cream can be used to remove MRSA from inside your nose, and an antibacterial shampoo can be used to remove it from your scalp. These products should be used one or more times a day for five days. You do not have to be admitted to hospital for treatment. You can do it at home before you go into hospital.

**Treating skin and soft tissue infections :** Minor skin and soft tissue infections, such as smaller boils or abscesses, may only require a treatment called incision and drainage. Incision and drainage involves piercing the tip of the boil or abscess with a sterile needle or scalpel to drain the pus and allow the affected area to heal. Before the procedure, you're likely to be given a local anaesthetic to numb the affected area. More extensive skin infections, such as cellulitis, will usually require a course of antibiotic tablets. You're likely to be given a course of antibiotic injections if you develop a skin or soft tissue infection in hospital and you're more vulnerable to the effects of the infection. This might be because you have a serious underlying condition.

**Treating invasive infections :** If you become infected with MRSA in hospital, it's likely that you'll need to be transferred to an isolation room. This reduces the risk of the bacteria spreading to other patients and infecting them. You may be placed in a room by yourself or in a small ward with other people who have an MRSA infection. You should still be able to have visitors, but it's very important that they clean their hands thoroughly before and after visiting you and before and after touching you.

For more serious, invasive MSRA infections, treatment will involve a course of antibiotic injections that could last several weeks. A combination of different antibiotics may be used.

Click on the links below for more information on treating some types of invasive infection that can be caused by MRSA:

- treating blood poisoning (sepsis)
- treating bursitis
- treating endocarditis
- treating osteomyelitis
- treating pneumonia
- treating septic arthritis

**Treating urinary tract infections (UTIs) :** Doctors, nurses and other health care providers have the following measures in place to prevent MRSA infections:

- **Hand cleanliness** - using soap and water or alcohol-based hand rub between caring for patients
- **Hospital rooms and equipment** - ensuring thorough cleaning
- **Keeping patients with MRSA separate from other patients** - either in a single room or shared with another person who has MRSA

- **Health care providers clothing** - wearing gloves and gown over clothing while caring for MRSA patients
- **Visitor clothing** - wearing of gloves and gowns
- **Disposal and cleanliness** - visitors and hospital providers removing and disposing of gowns and gloves after exiting the patient's room and washing hands thoroughly
- **Access to common areas** - patients with MRSA will be asked to limit movement around the hospital, avoid gift shops or cafeteria and stay in their room. The only exception to this rule is to visit other areas for treatment and tests
- **MRSA swabbing** - to identify if some non-MRSA patients have MRSA on their skin.

**What can be done to prevent community-associated MRSA?**

The following actions can reduce risk of community-associated MRSA outside of hospitals

- Regular hand-washing
- Keeping fingernails short
- Avoiding sharing products such as soaps, lotions, creams and cosmetics with others
- Avoiding sharing unwashed towels
- Avoiding sharing personal items such as razors, nail files, toothbrushes, combs or hairbrushes.

**Wounds infected with MRSA should be kept clean and covered with clean, dry bandages until healed to prevent the spread of infection to others.**

Never attempt to drain the infection yourself - this could make the infection worse or spread to other people. If antibiotics are prescribed, make sure the full dosage is taken for the duration recommended by your doctor, even if the infection is getting better. Only stop taking medication if your doctor advises you to.

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# PHMB in wound management

Wound infection results from the complex interaction between an individual's immune system, the condition of the wound and the number and virulence of bacteria present .Underlying medical problems such as poor blood supply, hypoxia and metabolic disorders are also contributing factors. If bacterial species are allowed to flourish, the states of colonisation, critical colonisation, or wound infection will occur. This is not only costly to the patient, but also has serious financial and legal implications for healthcare providers. Reducing the risk of infection through effective management of wound bioburden is thus an essential aspect of wound care.

**Effects of bioburden**

Although there are no clinical studies on the impact of specific microorganisms on the healing process, clinicians agree that infection causes serious delays in healing as a result of the expression of bacterial virulence. These factors are believed to damage the wound bed in a variety of ways:

a) Microorganisms consume nutrients and oxygen required for wound repair
b) Protease virulence determinants (e.g. Elastase) damage the extracellular matrix
c) White cell function is impaired by the release of short-chain fatty acids produced by anaerobes. Endotoxins stimulate production of interleukins: tumor necrosing factor and matrix metalloproteinases (mmps).
d) Free oxygen radical production increases
e) Imbalances occur between mmps and tissue inhibitors of metalloproteinases
f) Fibroblast production is decreased or delayed, collagen disorganised and scar strength decreased.

Topical antibiotics, which are linked to bacterial resistance, should be avoided. Adjuvant topical antimicrobial dressings may be used to help reduce the wound bioburden.

However, critical colonisation and localised, subclinical infection remain an issue and are significant contributors to prolonged wound healing. In recent years, topical antimicrobial agents have become the first line of treatment in managing bacterial burden, particularly in chronic wounds. Current opinion suggests that the ideal antimicrobial is:

- Associated with minimal systemic absorption
- Effective against likely contaminants and pathogens
- Fast-acting, with prolonged residual activity after a single dose
- Inexpensive
- Incapable of promoting bacterial resistance
- Non-carcinogenic and non-teratogenic (i.e. Does not cause DNA damage, which could result in carcinoma or foetal abnormality) to host cells
- Non-toxic
- Widely available

Antimicrobial dressings should be capable of bactericidal activity against both planktonic bacteria and those in biofilm colonies. In addition, the active substances must be contained in a delivery system that would normally, although not exclusively, be a contact dressing, which can be left in contact with the wound for 12 hours or more and remain active for the duration of wear time.

**PHMB**

The antiseptic agent polyhexamethylenebiguanide (also known as polihexanide or PHMB) has been used for over 60 years in a wide range of applications from swimming pool sanitisers to preservatives in cosmetics and contact lens solutions. In Europe, it has been available as a wound irrigation fluid for some time.

PHMB is a fast-acting biguanide compound composed of a synthetic mixture of polymers. The compound is structurally similar to the antimicrobial peptides (amps) produced by many cells within the wound, such as keratinocytes and inflammatory neutrophils, where they are thought to help protect against infection. Amps have a broad spectrum of activity against bacteria, viruses and fungi, inducing cell death by disrupting cell membrane integrity.

The structural similarities to AMP mean that PHMB can infiltrate bacterial cell membranes and kill bacteria in a similar way. However, PHMB does not interfere with the proteins that make up animal cell membranes. It, therefore, has a specific antimicrobial action that does not affect animal cell integrity. It is thought that, once it has adhered to the target cell membranes, PHMB causes them to leak potassium ions and other dissolved ions from the cytoplasm, resulting in cell death. PHMB has an effect on both planktonic bacteria and those in biofilms. Its action on the bacterial cell membrane also means that the efflux pump (a mechanism used by many bacterial cells to remove toxins) is unable to remove the antiseptic, so intracellular bactericidal concentrations are maintained. Once inside the cell, there is evidence that PHMB binds to DNA and other nucleic acids, suggesting it may also damage or inactivate bacterial DNA.

Studies have shown that PHMB is effective in vitro, while clinical studies indicate it has a broad spectrum of activity, including against human immunodeficiency virus (HIV). Testing has demonstrated that exposure to PHMB causes viral cells to clump together, forming aggregates. This prevents invasion into the host cells, making PHMB a potent antiviral treatment in wound care.

However, studies have shown that the product is safe in clinical use. Schnuch et al (2000; 2007) demonstrated that in trials including 3529 patients, skin sensitisation to PHMB is low (approximately 0.5%), even when the tested concentrations (2.5% and 5%) were 5–10 times that normally used in wound applications. Comparative tests of PHMB's biocompatibility (measurement of an antiseptic agent's activity in relation to its cytotoxicity) against other commonly used therapies have demonstrated its superiority to chlorhexidine, povidone-iodine, triclosan, silver and sulpadiazine. In addition, no known resistance to PHMB has been reported, most likely owing to its rapid and non-specific bactericidal activity.

**PHMB-based wound care**

Recently, PHMB has been successfully incorporated into a range of wound products with various formats. These products offer the clinician alternative methods of using PHMB in bioburden management. These products include:

Solutions and gels, infection control dressings which are impregnated with 0.2% PHMB, gauze dressings, Antimicrobial

Foam, which has a higher percentage of PHMB impregnated 0.5%.

PHMB molecule has been chemically bound to the base material, providing it with antimicrobial properties when in contact with wound moisture. The product, therefore, protects against the development of wound infection by decreasing the bacterial load in the dressing and prevents bacterial penetration through the dressing.

In PHMB-donating products, the active component is not chemically bound to the dressing material, and so can be delivered into the wound and periwound tissues. Here, the dressing is a carrier for a wider antimicrobial activity as it donates the PHMB into the wound.

Wound care products incorporating PHMB have been shown to have positive effects on wound healing. In vitro and in vivo studies have shown that, in some of these products, the influence of PHMB:

- Reduces wound pain rapidly and effectively
- Reduces wound malodour
- Increases formation of granulation tissue
- Increases keratinocyte and fibroblast activity
- Reduces slough within the wound
- Helps remove non-viable tissue

The success of PHMB has resulted in its recommendations as the primary antimicrobial in many European countries and has prompted the publication of a UK consensus review.

**Conclusion**

PHMB appears to meet the criteria for an ideal antimicrobial agent and is available in presentations that provide clinicians with effective wound-care modalities for most clinical scenarios. Clinical use, both in the UK and the wider healthcare community, has shown PHMB-based wound-care products to be effective options for managing wound colonisation and infection and, so, deserve closer scrutiny.

**KEY POINTS**

-PHMB has proven broad antimicrobial action and anti-fungal activity
-PHMB has minimum blood/protein inactivation (reduction of effect on mucous membranes owing to presence of mucin)
-PHMB has a sustained, post-application effect
-PHMB has an established promotion of wound healing and additional anti-inflammatory properties

PHMB is a heterodisperse mixture of polymers and is a synthetic compound. The basic molecular chain of PHMB can be repeated from two to 30 times, with increasing polymer chain length correlating with increasing antiseptic/ antimicrobial efficacy.

Amps are important in innate immune response and are produced by the majority of living organisms. They have a broad spectrum of activity against bacteria, viruses and fungi, and have been suggested as therapeutic alternatives to antibiotics. Amps are positively-charged molecules that bind to bacterial cell membranes and induce cell lysis by destroying membrane integrity, in a similar way that penicillin and cephalosporin antibiotics do. Amps are produced by many cells within the wound, such as keratinocytes and inflammatory neutrophils, where they are thought to play a role in protection against infection.

Molecular structure and mode of action of PHMB

The structural similarities between amps and PHMB mean that the latter can insert into bacterial cell membranes and kill bacteria in a similar way to amps. While it is unclear what the precise action of PHMB on bacteria is, the primary targets appear to be the outer and cytoplasmic membranes. PHMB is thought to adhere to and disrupt target cell membranes, causing them to leak potassium ions and other cytosolic components, which results in cell death. Studies indicate that PHMB does not form association with the neutral phospholipids that populate animal cell membranes, however, it does strongly interact with a key component of bacterial membranes, the acidic phosphatidylglycerol (PG). There is also evidence that some of the compound's antibacterial effects follow its penetration into target cells. In 1984, Broxton et al reported that maximal bactericidal activity occurs under conditions that promote rapid transportation of PHMB to the cytoplasm and cytoplasmic membrane. It has since been demonstrated that PHMB binds to DNA and other nucleic acids and precipitates them from aqueous solution. This suggests it may damage or inactivate bacterial DNA.

Comparative tests of PHMB's biocompatibility (measurement of an antiseptic/ antimicrobial agent's activity in relation to its cytotoxicity) against other commonly used therapies have demonstrated its superiority to chlorhexidine, povidone-iodine, triclosan, silver and sulfadiazine.

In wound care specifically, PHMB has previously been demonstrated to block Pseudomonas aeruginosa-induced infection (Cazzaniga et al, 2000) and prevent its degradation of wound fluid and skin proteins in vitro (Werthen et al, 2004). It can also kill a diverse range of bacteria and fungi.

PHMB can have a positive effect on tissue proliferation. In a laboratory study, cultures of normal human keratinocytes, fibroblasts and hacat-cells (human adult high calcium low temperature keratinocytes) were exposed to varying concentrations of PHMB and the results observed. It was found that concentrations between 0.2–2µg/ml had a significant proliferative effect on keratatinocytes. In concentrations greater than 2µg/ml, a dose-dependent decrease in cell proliferation was noted, thus there is a critical point at which it stops being beneficial and becomes damaging.

Wound Care and the 'At-risk' patient. It is known that patients with chronic wounds are often older and have a higher incidence of comorbidities, such as heart disease, diabetes mellitus, peripheral arterial disease and neuropathy, among others. Therefore, these preexisting conditions would classify a patient as 'at risk' of developing a foot ulcer. These patients require expert care from appropriate members of the multidisciplinary team. Healthcare professionals that are active at this level of wound care education need to be able to critically analyse the evidence, in order to recognise its validity and reliability and then demonstrate the ability to implement it into treatment planning and care pathways. It is also essential that they understand the importance of interprofessional and multidisciplinary working. The diabetic Foot Diabetes is known to increase the complications in wounds and in a recent study it has been

reported that patients with diabetes mellitus who have foot ulcers are at an increased risk of mortality compared with people who have diabetes and do not have a foot ulceration, with mortality rates being similar to patients suffering with common types of cancer. Diabetes is currently recognised as one of the leading causes of morbidity and mortality in the UK (Jefcoate and Harding, 2003; Bilous and Donnelly, 2010). Diabetes is a condition that can further affect the microvascular and macrovascular system (Bilous and Donnelly, 2010). The context and cost of diabetes in healthcare Diabetes care being used to treat diabetic foot problems (Roberts, 2006; NICE, 2011).The main cause of non-traumatic lower limb amputations is diabetic foot ulceration (Clayton and Elasy, 2009) and it is estimated that £252m is being spent annually on amputations (NICE, 2011). Recent figures reported by NICE (2011) suggest that each year in the UK, around 5,000 people with diabetes undergo leg, foot or toe amputations — equivalent to 100 occurring each week (NICE, 2011). The NICE clinical guidelines for prevention and management of type 2 Diabetes (NICE, 2004), report figures produced by Neil et al (1989), Walters et al (1992), Kumar et al (1994) and, more recently, Shakher and Stevens (2011), indicating that 20–40% of people with diabetes are estimated to have neuropathy and about 5% have a foot ulcer. Recognising those people at risk of ulceration is, therefore, crucial. Essentially, foot ulceration occurs due to neurological, vascular and/ or mechanical force problems.

Wound bed preparation, infection Andexudate management-the use of antimicrobials in wound care is not a new phenomenon. The current trend is looking to reduce the amount of antibiotics used as antibiotic-resistant bacteria, such as Methicillin-resistant Staphylococcus aureus (MRSA), are on the increase (Kingsley, 2009). Wound care, therefore, needs to focus on recognising the early stages of infection. All chronic wounds are contaminated with bacteria to some degree and low levels of bacteria can, in certain circumstances, facilitate healing (De Haan et al, 1974; Pollack, 1984; Kingsley, 2009). When the bacterial burden of the wound overtakes the host response, this is when clinical signs of bacteria are noted. Bacterial burden -the following terms indicate the bacterial numbers in a wound: Colonisation-Colonised wounds contain multiplying bacteria but the host does not have obvious clinical symptoms, nor is the healing of the wound affected by their presence. All wounds have a level of bacterial burden.

Critical Colonisation- Critically colonised wounds require a reduction in the bacterial burden if the wound is to heal. Chronic wounds are often critically colonised and this may be identified clinically when the wound margins fail to change. Critical colonisation may appear as a dull brick red colour with an increase in serous exudate. Sloughy fibrous tissue may also be present, which requires debridement and can be an early indicator of possible signs of localised infection. Local and systemic infection this is recognised as cellulitis, erythema, oedema, localised heat, pain and limited function, and may include increased discharge, wound breakdown, slough and odour. Localised infection would be less than 2cm and systemic infection greater than 2cm. Infection is better treated with antibiotics, but can be accompanied by topical antimicrobials. Septicaemia is characterised by chills, high fever, rapid breathing, rapid heart rate and the person appearing very ill. Wound healing focuses on wound exudate levels and viscosity (Vowden and Vowden, 2004; Gray et al, 2005). The higher the viscosity and exudate levels, the higher the incidence of infection.

Polyhexamethylenebiguanide (PHMB) Antimicrobial effectiveness has been demonstrated on *Acanthamoebapolyphaga*, *Acastellanii*, and *A hatchetti*. Additional effectiveness was demonstrated for PHMB use in water treatment. Barker and colleagues tested the effect of PHMB on *Legionella pneumophila*. This bacterium causes Legionnaire's disease and can be found in water systems, air conditioning machinery, and cooling towers.

Gilbert and colleagues have performed numerous studies on bacteria, especially those that form biofilms, such as *Klebsiella pneumoniae*. In studying biofilms produced from *E coli* and *S epidermidis*, they noted that those compounds with higher activity against planktonic bacteria, including PHMB, were also the most effective agents against sessile bacteria found within biofilms. They suggested that the differences in effects of concentration of PHMB on planktonic versus sessile bacteria was due to either the mechanism of action or the number or disposition of cationic binding sites. Kramer have studied the effects of various antiseptics including PHMB on fibroblast proliferation and cytotoxicity. They noted that while octenidine-based products retarded wound healing, PHMB promoted contraction and aided wound closure significantly more than octenidine and placebo.

The mechanism of action of PHMB has been described in a number of articles. Broxton et al demonstrated that maximal activity of the PHMB occurs at between pH 5–6 and that initially the biocide interacts with the surface of the bacteria and then is transferred to the cytoplasm and cytoplasmic membrane. Ikeda and colleagues showed that the cationic PHMB had little effect on neutral phospholipids in the bacterial membrane—its effect was mainly on the acidic negatively charged species where it induced aggregation leading to increased fluidity and permeability. This results in the release of lipopolysaccharides from the outer membrane, potassium ion efflux, and eventual organism death.

Clinically, PHMB has been used as a perioperative cleansing agent,in mouth wash, in ophthalmology, and as a topical wash. A combination of oral terbinafine and topical ciclopirox and PHMB were used to successfully treat a deep fungal infection (*Trichophyton mentagrophytes*) of the throat. Petrou-Binder describes the germicidal effects of PHMB (Lavasept 0.02%) as eye drops prior to cataract surgery. It was well tolerated with low tissue response and minimal patient discomfort.

**Biosynthesized Cellulose Wound Dressing—Antimicrobial (BWD-PHMB)**

Biosynthesized cellulose wound dressings (xcell Cellulose Wound Dressing and xcell Cellulose Wound Dressing Antimicrobial) were developed to maintain a moist wound environment without causing maceration, reduce pain, and enable autolytic debridement. This is possible because the dressings effectively absorb exudate and hydrate dry areas of a wound different from other dressings that have only a single function.

A 49-patient, multicenter, controlled, randomized clinical study was conducted to demonstrate effectiveness of BWD

compared to standard of care on venous leg ulcers. Significantly more autolytic debridement, significantly reduced pain, and cleaner wound margins were demonstrated after the 12-week study period. Improved rate of wound closure, as demonstrated by increased epithelialization and granulation tissue, was also noted.

The antimicrobial version of BWD (BWD-PHMB) contains cellulose, water, and 0.3% polyhexamethylenebiguanide (PHMB). BWD-PHMB is indicated for use on partial- and full-thickness wounds. It is designed to cover a wound or burn, absorb areas of wound exudate, and provide a moist wound environment that supports autolytic debridement of nonviable tissue. The dressing may be used on moderately exuding, nonexuding, and dry wounds. It also protects against abrasion, desiccation, and external contamination. The moist environment has a cooling effect that has demonstrated a significant reduction of pain.

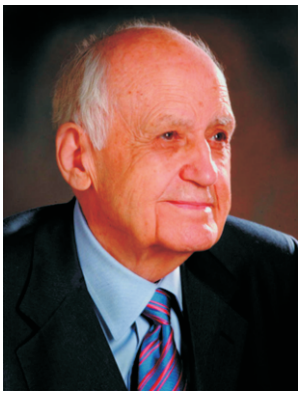
Preclinical efficacy testing. BWD-PHMB demonstrates it effectiveness against a variety of organisms. Following a modified American Association of Textile Chemists and Colorists (AATCC) Method 100, samples were incubated with approximately 106 CFU/ml of the various challenge organisms. After 24 hours, a second count was made to determine the reduction in the number of organisms present. Results indicated 99.9% reduction of MRSA, *Escherichia coli*, *Enterococcus faecalis*, *Bacillus subtilis*, and *Candida albicans* within the 24-hour period.

Release of PHMB from BWD-PHMB. A study was performed to demonstrate the release of PHMB from BWD-PHMB. Five sterile 3.5-in x 3.5-in samples were used. One quarter of the dressing was used to determine the initial PHMB concentration in each dressing using UV-Vis (Ultraviolet-Visible) Spectroscopy (Genesys™ 10 UV, ThermoSpectronic, Rochester, NY) at a wavelength of 234 nm. The remainder of the sample was weighed and placed into 20 times its weight in filtered water. At various times, including 0.5, 1, 2, 3, 4, 5, 6, and 24 h, the solution was assayed for PHMB concentration. At the 24-h time the dressing was removed from the tray, weighed, and an extract was taken and assayed for PHMB concentration.

**Systemic antibiotics were not given in conjunction with the use of BWD-PHMB to ensure bacterial reductions were solely due to the PHMB.**
The organisms identified included methicillin-resistant *Staphylococcus aureus* (MRSA), *Staphylococcus aureus*, *Pseudomonas aeruginosa*, *Proteus mirabilis*, Diphtheroid gram-positive rods, beta hemolytic *Streptococcus B*, *Enterobacter aerogenes*, mixed skin flora, and *Enterococcus sp*. The most common was Staphylococcus (including MRSA) and Pseudomonas.

Case Reports
Case 1. A 58-year old woman presented with a full-thickness draining wound over the dorsal foot secondary to an incision. The patient's wound extended to the level of tendon and was recalcitrant to topical gels, ointments, foam dressings, silver dressings, and moist saline gauze. Past medical history was significant for Hodgkin's disease, heart valve replacement, pacemaker, hemolyticanemia, and chemo and radiation therapy for breast cancer, which was on-going at the time of presentation. After 3 weeks of treatment with a papain-urea ointment (Panafil®, Healthpoint, Fort Worth, Tex), the majority of fibrotic tissue was removed although the wound did not decrease in size. The patient was then placed exclusively on BWD-PHMB for approximately 4 weeks with the dressing being changed once a week. The wound rapidly improved and progressed to complete closure during this time period.

## Maurice Ralph Hilleman



| | |
|---|---|
| Born | August 30, 1919, Miles City, Montana |
| Died | April 11, 2005 (aged 85), Philadelphia, Pennsylvania |
| Cause of death | Cancer |
| Nationality | United States |
| Occupation | Microbiologist, vaccinologist |
| Known for | Developing several importantvaccines |
| Awards | Robert Koch Prize (Gold, 1989)<br>Albert B. Sabin Gold Medal (1997)<br>Prince Mahidol Award (2002) |

Maurice Hilleman was responsible for developing more than 40 vaccines, including measles, mumps, hepatitis A, hepatitis B, meningitis, pneumonia, *Haemophilus influenzae* bacteria, and rubella. His vaccines have been credited with saving millions of lives and with eradicating common childhood diseases. The measles vaccine alone has prevented approximately one million deaths. Among other accomplishments, he succeeded in characterising and isolating many viruses, including the hepatitis A vaccine in culture.

Despite Hilleman's many breakthroughs in immunology and vaccinology, he has never been a household name. Anthony Fauci, director of the US National Institute of Allergy and Infectious Diseases, said Hilleman had "little use for self credit." Dr Fauci told the *BMJ* that Hilleman's contributions were "the best kept secret among the lay public. If you look at the whole field of vaccinology, nobody was more influential."

Hilleman's interest in microbiology and science had its roots in his childhood. Born in 1919, he grew up during the Great Depression on a farm in the southeastern plains of Montana. To help his family through the Depression, he needed to be economical and tenacious. It was a building block he later used for keeping his focus.

After the Depression, he entered Montana State University on a full scholarship. In a 1999 issue of*Immunological Reviews,* he described Montana State as a "no-nonsense institution where professors taught and where teaching assistants, other than laboratory aides, did not exist." He gained a bachelor's degree in microbiology and chemistry.

His graduate education at the University of Chicago reinforced his independence and self reliance. It was a tough environment, in which Hilleman said you would either "sink or swim." In 1944 he was awarded a PhD in microbiology and chemistry. Hilleman told his professors at Chicago that he was going into industry, where he thought he would be best positioned not only for conducting research, but also for ensuring and expediting clinical applications. His professors told him that he belonged in academia and that they had not trained him for a career in industry. Hilleman strongly disagreed, maintaining that academic institutions lacked the resources to move scientific innovations forward and to market.

Paul Offit, chief of infectious diseases at the Children's Hospital of Philadelphia, told the *BMJ*, "His commitment was to make something useful and convert it to clinical use. Maurice's genius was in developing vaccines, reliably reproducing them, and he was in charge of all pharmaceutical facets from research to the marketplace." Hilleman felt that scientists had a responsibility to provide a return on knowledge gained in the laboratory.

In 1944 he joined the virus laboratories of E R Squibb & Sons in New Brunswick, New Jersey, where he developed a vaccine against Japanese B encephalitis, urgently needed to immunise troops fighting in the Pacific.

Hilleman characterised several viruses and identified changes that could result when a virus mutated. This concept, which he worked out while at the Walter Reed Institute of Army Research, helped prevent a huge pandemic of Hong Kong flu in 1957. Learning that the flu was a new strain, 40 million doses of vaccine were rapidly made available in the United States.

He joined Merck on New Year's Eve, 1957, as director of a new department of virus and cell biology research. Under Hilleman's aegis, by 1984 Merck had garnered 37 product licences, with an additional three vaccines ready for development. He retired from Merck at age 65, but stayed on as a consultant.

Hilleman's style of working was icono-clastic. Dr. Offit said, "To give you an example of how he worked, in 1963, [when his daughter had the classic symptoms of the mumps,] he swabbed the back of his daughter's throat, brought it to the lab to culture, and by 1967, there was a vaccine." He added, "Today's regulation would preclude that from happening... If Maurice was alive today, I doubt he would be able to be Maurice. He was a very strong willed person and a person like him could face a high level of inertia."

During his more than 60 years in basic and applied research, he earned a reputation as an often harsh, impatient fellow who tangled with industry and government bureaucracies. Hilleman defended his pushy and prickly behaviour, which offended some colleagues and coworkers, as crucial for science to advance. He argued that politics, not science, determined which breakthroughs were brought to the marketplace.

Hilleman received many honours, including a special lifetime achievement award from the World Health Organization.

He leaves his second wife, Lorraine; two daughters; and five grandchildren.

*Maurice Hilleman, microbiologist Philadelphia, United States (b Miles City, Montana, 1919), died from cancer on 11 April 2005.*

# JOKES

Soul1: How did u die?
Soul2: Due 2 cold U?
S1: I doubted my wife with a man & searched my house, found none, felt guilty & suicided
S2: Ha ha i was in d Fridge..

Love Law: Newton in comedy jokey mood- "Love can neither be created, Nor be destroyed. Only it can b transferred from 1 girlfriend to another girlfriend, with some loss of money and time.

Explosive comedy:
Santa gives dictation test for students,
last bench students said v r not able to hear sir..
Santa said ok i will write on board.!

A vegetarian Guy looked at my burger and said, " You know, a sheep died so you could have that burger."
I looked at her salad and responded, " May be she died because you keep eating all her food!

Four guys
1 from Harward:
1 Oxford
1 Texas
&
a Sardar from Pujab university
1 common question:
What is the fastest thing in world?
Oxford: Light
Harvard: Thought
Texas: Blink of an eye
Sardar: Its loose motions, because last night I was lying in my bed & before I could blink, think or turn on the lights, it was over!

Question:
Why most of the engineering students
Can't clear all subjects in 1st attempt..?
?
?
?
Answer:
Smooth roads never make good drivers,
Clear sky never makes good pilots
&
Clearing all subjects in the 1st attempt,
Never makes good engineers.

Difference between Ignorance & self control?
When u c mirror & u don't laugh at yourself, that is ignorance!
&
When i look at u & i don't laugh, thats called self control:-)

Can we do romance in the midnight today?
I'm in a good mood:)
Just a little bit of kissing and biting!!
Reply me soon, yours Loving Mosquito.

Teacher : What do you call a person who keeps on talking when people are no longer interested?
Pupil : A teacher

2 Guys Were Following 2 Girls
Both Girls Took Rakhi & Tied To Their Hands.
1st Guy To Second-What Will We Do Now?
2nd Guy-U Marry My Sis, I Will Marry Ur Sis

Height of confidence
Once many professors were called and asked to sit in an airplane.
After they sat. They were informed that the plane is made by their students.
All of them ran and got out of plane except one. People asked him the reason
He said,"If it's made by my students it will not even start."

KID :- Why some of ur hair are white dad ?
DAD : - Every time a son make his dad unhappy, one of his father's hair turns white .....
KID :- Now understand why grandpa's hairs are all white.

A student is talking to his teacher.
Student: 'Would you punish me for something I didn`t do?'
Teacher: 'Of course not.'
Student: 'Good, because I haven't done my homework.'

Judge: Y U've stolen money 4m dis man?
Sardar: My lord I've nt stolen money. He jst gave it 2 me
Judge: Whn He gave U money ?
Sardar: Whn I showd him gun

Santa: whats diffrence between Seniors & Juniors?
Banta: samundra k najdik rehte wo seniors (sea+nears)
&
jo Zoo k najdik rehte wo Juniors (Zoo+nears)

# *Cronobacter sakazakii*

**Introduction:**

*Cronobacter sakazakii* represents a significant risk to the health of neonates. This bacterium is an emerging opportunistic pathogen that is associated with rare but life-threatening cases of meningitis, necrotizing enterocolitis, and sepsis in premature and full-term infants. Infants aged <28 days are considered to be most at risk. Feeding with powdered infant formula (PIF) has been epidemiologically implicated in several clinical cases. Infants should be exclusively breast-fed for the first 6 months of life, and those who are not should be provided with a suitable breast-milk substitute. PIF is not a sterile product; to reduce the risk of infection, the reconstitution of powdered formula should be undertaken by caregivers using good hygienic measures and in accordance with the product manufacturer's food safety guidelines.

*Cronobacter sakazakii* is a motile peritrichous, gram-negative bacillus. The organism, which was initially referred to as "yellow-pigmented cloacae," was reclassified as *Cronobacter "sakazakii"* in 1980 on the basis of differences in DNA-DNA hybridization, biochemical reactions, pigment production, and antibiotic susceptibility, compared with *Enterobacter cloacae*. Recent studies have demonstrated that *C. sakazakii* is a genomically heterogeneous and, therefore, poorly defined species.

*C. sakazakii* is regarded as an emerging opportunistic human pathogen and the etiological agent of life-threatening bacterial infections in infants. *C. sakazakii* was first implicated in a case of neonatal meningitis in 1958, when an outbreak in England resulted in the deaths of 2 infants. Although the incidence of *C. sakazakii* infection is low, the prognosis is poor, and infection is associated with significant morbidity and mortality. Powdered infant formula (PIF) products have been shown to contain *C. sakazakii* and have been epidemiologically linked to several clinical cases. Related coliforms, such as *Citrobacter diversus*, have also been isolated from PIF. Like *C. sakazakii*, these organisms can cause invasive infections. There is also evidence from surveillance activities that low-level contamination of PIF with *Salmonella* species has led to cases of disease in infants. Recalls of infant formula contaminated with *C. sakazakii* have occurred in the United States and Europe. This has resulted in increased efforts to implement appropriate strategies to reduce the health risks associated with the use of PIF.

**Infections Caused By *C. Sakazakii***

*C. sakazakii* infections are an important cause of life-threatening meningitis, septicemia, and necrotizing enterocolitis in infants. Premature and low-birth-weight infants and those aged <28 days are considered to be more at risk than are older infants. Clinical presentation includes meningitis (complicated by ventriculitis, brain abscess, cerebral infarction, and cyst formation), bacteremia, and necrotizing entercolitis. A CT scan of the skull should be considered early during the management of symptomatic infected infants, because it nearly always reveals abnormalities, including cystic changes, abscesses, fluid collection, dilated ventricles, and infarctions. *C. sakazakii* has also been identified in the stool or urine of asymptomatic infants, and stool carriage has been demonstrated for up to 18 weeks.

There have been few reports of *C. sakazakii* infection in adults, and it is not usually life threatening. Indeed, most adults with reported*E. sakazakii* infections had serious underlying diseases, such as malignancies. *C. sakazakii* infections continue to be more common in neonates and infants, among whom they are usually associated with a poor prognosis. Mortality rates of 33%–80% have been reported. *C. sakazakii* infections are also associated with significant morbidity. Most children who survive *Enterobacter*-associated meningitis (94%) develop irreversible neurological sequelae resulting in quadriplegia, developmental impedance, and impaired sight and hearing. These sequelae are frequently attributed to secondary cerebral infarcts.

**Reservoirs of *C. Sakazakii***

The natural habitat of *C. sakazakii* is currently unknown. This bacterium can be found in the environment and in food. The organism's natural habitat may be on plant material, and this may account for the organism's isolation from dry herbs and spices. Kandhai et al. isolated *E. sakazakii* from milk powder manufacturing facilities (8 of 9 samples) and household vacuum cleaners (5 of 16 samples), thus confirming its ubiquitous distribution. *C. sakazakii* has also been isolated from milk powders, cheese products, baby foods, minced beef, sausage meat, and vegetables. In addition, the isolation of *C. sakazakii* from animal sources—Mexican fruit flies and from the gut of stable fly larvae—has been documented. The organism was not detected in other environmental settings, including surface water, soil, mud, rotting wood, grain, bird droppings, domestic animals, cattle, or cows' milk. *C. sakazakii* has also been isolated from a wide range of clinical sources, including CSF, blood, bone marrow, sputum, urine, inflamed appendix tissue, intestinal and respiratory tracts, eye, ear, wounds, and feces. This organism has also been isolated from the hospital environment.

**Mode of Transmission**

The sources of *C. sakazakii* and its vehicles of transmission are not always clear. Although the organism has been detected in multiple food sources, a strong association has been found only with PIF. Intrinsic and extrinsic contamination of PIF with *C. sakazakii* can occur. Intrinsic contamination results from the introduction of the organism to the PIF at some stage during the manufacturing process. In contrast, extrinsic contamination may result from the use of contaminated utensils, such as blenders and spoons, in the preparation of PIF.

Several investigations into the presence of *C. sakazakii* in PIF have been performed. Muytjens et al. examined 141 different powdered formulas from 35 countries and isolated *C. sakazakii* at levels ranging from 0.36 to 66 cfu per 100 g from 20 formula samples from 13 countries. Simmons et al. isolated *C. sakazakii* (8 cfu per 100 g) from PIF in association with an outbreak in Memphis, Tennessee. Biering et al. isolated *C. sakazakii* from 5 different lot numbers of unopened packages of PIF after an outbreak of neonatal meningitis in Iceland. A Canadian survey that investigated the incidence of *C. sakazakii* in PIF isolated the organism from 8 of 120 cans from 5 different manufacturers.

Van Acker et al. reported an outbreak of *C. sakazakii* infection involving 12 infants who had necrotizing enterocolitis in 1998 in Belgium; *C. sakazakii* was isolated from liquid formula prepared from PIF. In Belgium in 2002, an infant died of *C. sakazakii*–associated meningitis after consuming a commercial PIF. The product was withdrawn after the detection of low levels of *C. sakazakii* in the implicated infant formula. In New Zealand in July 2004, a premature infant contracted *C. sakazakii* meningitis and died. The subsequent investigation found that 4 other babies in the neonatal intensive care unit were colonized with this organism, but none became unwell. The investigation attributed the source of the organism to PIF used in the nursery. Most recently, another PIF was withdrawn after a possible link to 5 cases of presumed *C. sakazakii* infection in premature infants in France in 2004 that led to the death of 2 infants.

**Pathogenicity and Virulence Factors**

In mammalian tissue culture, the organism can attach to intestinal cells and survive internally in macrophages. However, the specific bacterial adhesins and host cell receptors involved in these processes are unknown. Some strains of *C. sakazakii* produce capsular material, and how this material contributes to macrophage evasion remains to be determined. Furthermore, this capsule may also provide protection for the organism, facilitating its survival in desiccated environments.

*C. sakazakii* can attach to plastics and silicon rubber surfaces and grow in a biofilm. Enteral feeding tubes and feeding-bottle teats can harbor the bacterium in large numbers.

Biofilm formation may also be a factor associated with altered susceptibility to antimicrobials.

No reports have investigated the dose-response relationship in *E. sakazakii* infection. Using a suckling mouse model, Pagotto et al. determined a minimum lethal dose for 18 *E. sakazakii* isolates (9 clinical, 8 food, and 1 type strain). They challenged newborn mice orally and intraperitoneally and showed that all isolates administered intraperitoneally were lethal at $1 \times 10^8$ cfu. For 2 strains, the minimum lethal dose was $1 \times 10^5$ cfu. Two of 18 isolates were lethal when administered orally. The authors concluded that the minimum lethal dose in neonates would most likely require an unusually high number of viable cells (an event likely to occur if reconstituted formula is held at an inappropriate temperature over time). This study also suggested the possibility of enterotoxin production. However, the importance of putative enterotoxin production remains unclear, because neither the genes encoding the putative toxin nor the protein itself have been identified as of yet. Furthermore, the possibility of differences in virulence among isolates cannot be ruled out. Future research should address these issues and provide a better understanding of the mechanism(s) of pathogenesis.

The thermotolerance of *C. sakazakii* has been determined by a number of researchers. Although there are significant discrepancies regarding the degree of heat resistance, *C. sakazakii* appears to be more thermotolerant than other Enterobacteriaceae cultured from dairy products.

Whether this variation in thermoresistance correlates with the genetic diversity of *C. sakazakii* remains to be established.

**Antimicrobial Resistance**

*C. sakazakii* is naturally resistant to all macrolides, lincomycin, clindamycin, streptogramins, rifampicin, fusidic acid, and fosfomycin. It is susceptible to some antibiotics, including tetracyclines, aminoglycosides, numerous β-lactams, chloramphenicol, antifolates, and quinolones. *E. sakazakii* infections have been traditionally treated with ampicillin-gentamicin or ampicillin-chloramphenicol. However, resistance to ampicillin has emerged owing to the acquisition of transposable elements and the production of β-lactamases *Cronobacter* species are known to be capable of inactivating broad-spectrum penicillins and cephalosporins through the production of β-lactamase enzymes. This situation also appears to be increasing among isolates of *C. sakazakii*. Consequently, consideration should be given to the use of carbapenems or the newer cephalosporins in combination with a second agent, such as an aminoglycoside. The use of trimethoprim-sulfamethoxazole may also be useful.

**Laboratory Detection of *C. Sakazakii***

Reliable detection of *C. sakazakii* poses a major challenge to PIF manufacturers; thus, the action to be taken on its identification is important. PIF is not a sterile product, and current Codex Alimentarius Commission specifications for PIF permit 1–10 coliform bacteria per gram of formula. It should be noted that *C.* belongs to this group of organisms. Nevertheless, PIF manufacturers implement a policy of zero tolerance for both *Salmonella* and *Listeria* species in products. Current drafting of microbiological specifications for *C. sakazakii* is under consideration by the International Committee for the Microbiological Safety of Food and the Codex Alimentarius Commission.

**Culture and Biochemical-Based Identification**

The US Food and Drug Administration—recommended procedure for the isolation of this organism uses standard isolation methods for Enterobacteriaceae, with additional selection for yellow-pigmented organisms and subsequent biochemical identification. Furthermore, this protocol is only selective for Enterobacteriacae, is not specific for *C. sakazakii*, and requires 5 days to complete.

Muytjens et al. first identified α-glucosidase enzyme activity in 129 isolates (100%) of *C. sakazakii*. Farmer et al. confirmed this finding, identifying 53 of 57 *C. sakazakii* strains positive for α-glucosidase activity. To facilitate the detection of *C. sakazakii*, culture media—including Druggan-Forsythe-Iversen, Oh-Kang, and Leuscher-Baird-Donald-Cox agar—have been recently formulated that exploit this key biochemical characteristic. However, α-glucosidase activity is not solely restricted to *C. sakazakii*.

**Molecular Detection**

As a means of improving the detection of *C. sakazakii*, molecular-based methods are being developed. Seo and Brackett described a quantitative real-time PCR technique in which primers and a TaqMan probe were developed to target the macromolecular synthesis operon of *C. sakazakii*. The method could detect 100 cfu/mL in reconstituted PIF without an enrichment step. More recently, Liu et al. developed 2 real-time PCR assays based on TaqMan and SYBR green technology. Both of these assays used primers that target the 16-23S rRNA spacer region and could detect 1.1 cfu of *C. sakazakii* per 100 g of infant formula after a 25-h enrichment.

**Public Health Significance of *C. Sakazakii* and Food Safety**

Infants and young children are particularly vulnerable to foodborne infections. Therefore, the microbiological safety of infant and follow-up formula is of utmost importance. Caregivers in hospital neonatal units should be continuously alerted to the fact that PIF is not a sterile product and that, therefore, the use of hygienic measures during preparation and reconstitution are essential.

PIF has been fed to millions of infants for years, and it constitutes the majority of infant formula used worldwide. This product is formulated to mimic the nutritional profile of human breast milk. Because PIF is not a sterile product, it is an excellent medium to support bacterial growth. Bovine milk is an essential ingredient of PIF and a potential source of bacteria that are pathogenic to humans. On occasion, bacterial pathogens have been cultured from PIF, including *Citrobacter, Enterobacter, Klebsiella, Staphylococcus, Streptococcus*, and *Yersinia* species. Should *C. sakazakii* multiply in PIF, it can result in infection. A definitive link between the presence of *C. sakazakii* in an unopened can of PIF and an outbreak of infection has been reported. Cases of invasive *C. sakazakii* infection have recently been added to the list of notifiable diseases in New Zealand, after the death of an infant due to *C. sakazakii* meningitis in July 2004. These actions highlight the importance of this opportunistic pathogen and the risk posed to vulnerable infants.

The World Health Organization recommends that infants should be exclusively breast-fed for the first 6 months of life. Infants who are not breast-fed should be provided with a suitable breast milk substitute, formulated in accordance with Codex Alimentarius Commission standards. To reduce the risk of infection in infants fed PIF, recommendations have been made for the preparation and storage of PIF.

Manufacturers of PIF are being encouraged to develop a greater range of commercially sterile alternative formula products for high-risk groups. In addition, formula manufacturers must implement strategies aimed at reducing the risks of product contamination. Controlling the initial populations of *C. sakazakii* during the production of PIF and avoiding postprocessing contamination, using suitable microbiological approaches, will have a positive effect. Data from surveys showed that *C. sakazakii* can be cultured at various frequencies in samples of PIF, from the manufacturing facility, and from environmental sources. However, the true frequency of contamination is unknown, making it difficult to quantify the level of risk to vulnerable groups. The role of the broader infant food chain and of dairy animals and their environment as sources of contamination has not been investigated. Standardized analytical approaches are necessary to ensure product safety. The European Food Safety Authority has recommended the introduction of a performance objective for PIF and follow-up formula that is aimed specifically at low levels of *Salmonella* and *C. sakazakii* (e.g., absence in 1, 10, or 100 kg).

**Future Needs**

Current research on *C. sakazakii* has focused on the elimination of this coliform from PIF. Investigations into thermal resistance, osmotic tolerance, exopolysaccharide production, and pathogenicity, among others, have been performed, and attempts have been made to identify environmental reservoirs. Only 1 study has suggested the possible existence of an enterotoxin produced by *C. sakazakii* on the basis of an animal model. Other virulence factors remain to be identified. Furthermore, why infection can occur in all age groups but is more frequent among full-term infants and neonates remains to be understood. Physicians and other care givers must advocate breast-feeding as the preferred means of feeding infants. Where this is not possible, hygienic practices for the preparation of PIF in both the home and hospitals should be carefully followed. These guidelines will contribute toward the minimization of risk. Increasing the awareness of *C. sakazakii* infection among medical personnel and the continuous education of all care givers to the potential threats posed by this organism will be essential to protect infants at high risk.

# Milk pasteurisation and safety: a brief history and update

**Introduction**

The preservation of foods by heat has probably been practiced by man since the discovery of fire. Although sterilisation is an absolute term which usually means the complete destruction of all forms of life, substantial food preservation can be achieved by less than complete sterilisation. Pasteurisation is such a treatment: the name is derived from that of Louis Pasteur, whose discoveries in the 1860s and 1870s demonstrated that heating liquids, especially wines, to fairly low temperatures, such as 60°C, improved the keeping quality during storage. This low-temperature heat treatment destroyed spoilage organisms, but was low enough so as not to destroy the original characteristics of the liquid being treated.

The early history of pasteurisation was reviewed in detail by Westhoff and much of the information below is taken from that paper. The International Dairy Federation has developed a monograph on pasteurised milk which covers all aspects of pasteurization.

**Current issues**

Despite the improvement in the quality of the milk supply in the USA through research, education, standards development, evaluation and certification activities, there are still occasional outbreaks of milk-borne diseases, even though these comprise less than 1% of the reported disease outbreaks associated with contaminated food and water. This only emphasises the need for continued vigilance at every stage of milk utilisation, from production to distribution and consumption.

***Mycobacterium tuberculosis* complex**

Pulmonary tuberculosis has been one of the great scourges of humankind; numerous studies have demonstrated the presence of mycobacteria in milk. Classification of acid-fast bacilli isolated from raw milk has identified *M. tuberculosis, M. bovis, M. smegmatis, M. avium* and *M. fortuitum,* as well as other acid-fast bacilli such as *Nocardia.* The *M. tuberculosis* complex also includes *M. africanum* and *M. microti.* Consumption of raw milk contaminated with pathogenic mycobacteria has been associated with human disease; humans are extremely susceptible to disease from *M. bovis*. Although other avenues of environmental exposure, such as contaminated soil or water supplies, may account for some cases of human disease, transmission of mycobacteria from raw milk appears to be the most likely route of exposure.

The presence of mycobacteria in the milk of cows subclinically infected with tuberculosis was once a major public health issue. Reports have indicated that children under 15 years of age are most susceptible to infection, with resultant lesions in cervical and abdominal lymph nodes (76).

Major international bovine tuberculosis eradication campaigns resulted in virtual elimination of *M. bovis* from cattle herds in the USA; the incidence is presently estimated at 0.003%. The adoption of milk pasteurisation standards aided in the eradication process. Therefore, the discovery that tuberculosis has not been eradicated but is reappearing in the USA as outbreaks of multiple drug-resistant M. *tuberculosis* has been particularly unexpected. Seventy-three patients with microbiologically documented *M. bovis* infections have been identified over a recent twelve-year period (1980-1991); 80% of the patients were of Hispanic origin. The immigration of high risk populations and human immunodeficiency virus infection has apparently reversed the annual 5% decline in the incidence of tuberculosis in the USA. Deterrents to tuberculosis eradication have been identified as being infected cervid herds (elk, especially, and bison) in the USA and Canada (46, 52). In addition, a recent resurgence of bovine tuberculosis in the livestock industry of Mexico and the ongoing importation of steers from Mexico into the USA has prompted a review of proper methods for management of colostrum and milk from infected cows. *M. bovis* remains endemic in beef and dairy cattle herds in Mexico. This resurgence of a 'forgotten' disease re-emphasises the importance of careful handling practices for milk and milk products.

Other mycobacterial agents isolated from raw milk which have been classified as known human pathogens are *M. smegmatis, M. avium* and *M. intracellulare.* Although the environment serves as the major source of human exposure to these pathogens, animals may also serve as a reservoir for human infection. Rapid-growing acid-fast bacteria such as *M. smegmatis* have been isolated from cows with mastitis. Similar to other forms of mastitis, mycobacterial intramammary infections are characterised by abnormal mammary secretions, oedema and localized inflammation of affected quarters. There has been speculation that mycobacterial intramammary infections occur secondary to severe clinical mastitis, and that these mycobacteria may be more accurately described as opportunistic pathogens. Reports have associated this type of mastitis with the introduction of mycobacteria into the teat canal as a contaminant during antibiotic therapy for other mastitic organisms compounded by poor sanitation.

*Mycobacterium paratuberculosis,* the mycobacterium responsible for paratuberculosis (Johne's disease) in ruminants, has been implicated as the pathogen which causes Crohn's disease in humans. Of the common clinical signs shared by para tuberculosis and Crohn's disease, the most significant is the localised intestinal inflammation found in both disorders. The consumption of milk or dairy products has been cited as one possible source of human exposure to *M. paratuberculosis* since the presence of paratuberculosis DNA has been documented in cow milk obtained from retail markets in Great Britain. Cows with clinical paratuberculosis have been found to shed *M. paratuberculosis* in milk, albeit in low numbers, and the organism has been isolated from mammary tissue and regional lymph nodes. However, there is no evidence to date to indicate that viable *M. paratuberculosis* can be cultured from milk after pasteurisation.

The presence of mycobacterial pathogens in raw milk suggests a potential public health hazard. The use of raw milk in the production of cheese and other dairy products has further exacerbated the problem as mycobacteria have been cultured from aged cheeses.

There is a wide belief today that pasteurisation of raw milk adequately kills any contaminating mycobacteria which may be present, making the milk safe for human consumption.

Further studies have demonstrated that after pasteurisation of raw milk contaminated with *M. tuberculosis,* no growth could be detected on selective growth medium. Strains of *M. avium* and *M. fortuitum* were isolated from homogenised, flash-pasteurised milk samples, although there was speculation that contamination during the processing procedure may have been responsible. Other studies have shown that pasteurisation of raw milk either by the 'holder' method (63.5°C, 30 min) or the high-temperature, short-time method (HTST: 71.7°C, 15 s) was effective in destroying these strains of mycobacteria. Laboratory simulation of either method is difficult, and experimental methods vary widely among laboratories. Examples of this include studies conducted using a test-tube model to simulate the holding vessel during heat inactivation: with this model, treatment of raw milk experimentally inoculated with various strains of mycobacteria at 63.5°C for 30 min completely inactivated *M. bovis* and *M. fortuitum,* but some survival of *M. avium, M. intracellulare* and *M. kansasii* was evident. Similarly, *M. paratuberculosis* survived in milk when held at either 63.5°C for 3 0 min or 72°C for 15 s using the test-tube model. Improvement of the laboratory-scale pasteuriser system to simulate an HTST heat-exchanger as used in commercial systems decreased the numbers of viable *M. paratuberculosis* cultured from milk compared to the test-tube model, yet small numbers still survived (<1%).

More recently, studies were conducted to evaluate heat inactivation of *M. paratuberculosis* in raw milk by the holder test-tube method and a method using a flow-through laboratory-scale pasteuriser system designed by a commercial manufacturer. Results from these studies show that *M. paratuberculosis* survived heat treatment at 65°C for 30 min using the test-tube model; however, treatment of milk at 72°C for 15 s in the flow-through pasteuriser unit effectively killed all bacteria present. These studies demonstrate that the vigorous mixing induced by turbulent flow of the milk during pasteurisation, as occurs in commercial operations, is essential to ensure a uniform temperature for complete destruction of contaminating *M. paratuberculosis.*

Contamination of raw milk with mycobacteria is seemingly unavoidable, even under the most sanitary conditions, since many strains are ubiquitous in the environment. Heat treatment of raw milk using current commercial pasteurisation protocols appears to ensure adequate destruction of contaminating mycobacteria which may be present. Therefore, transmission of viable mycobacteria to humans through pasteurised dairy products seems unlikely: pasteurisation minimises the threat of mycobacteria as causative factors in human disease.

**Conclusions**

Since pathogenic micro-organisms are readily isolated from raw milk, many State health departments, the United States Food and Drug Administration and the International Dairy Federation strongly recommend that unpasteurised milk should not be drunk or used in the manufacture of any dairy product and specifically not in cheese manufacture. Disease outbreaks from raw milk are usually associated with children visiting a dairy farm and drinking the raw milk. Disease outbreaks associated with cheese made from unpasteurized milk indicate that the 60 days of ripening required before distribution may not be sufficient to completely eliminate pathogens such as mycobacteria, *Salmonella, Listeria* and *E. coli* 0157:H7. Pasteurised milk is usually considered pathogen-free with the exception of the spores of *Bacillus cereus,* if present in large numbers. When a milk-borne disease outbreak occurs, the cause is usually either post-pasteurisation contamination or improper processing. The dairy industry and public health regulators must remain vigilant to ensure that all measures are taken to prevent the entry and multiplication of pathogenic micro-organisms during the handling and processing of milk and milk products to prevent any pathogen-associated illness.

# Best practices-in chemical water disinfection

Water purification is the process of removing undesirable chemicals, biological contaminants, suspended solids and gases from contaminated water. The goal is to produce water fit for a specific purpose. Most water is disinfected for human consumption (drinking water), but water purification may also be designed for a variety of other purposes, including fulfilling the requirements of medical, pharmacological, chemical and industrial applications.



The standards for drinking water quality are typically set by governments or by international standards. These standards usually include minimum and maximum concentrations of contaminants, depending on the intended purpose of water use.

Chemical and microbiological analysis, while expensive, are the only way to obtain the information necessary for deciding on the appropriate method of purification.

Disinfection is accomplished both by filtering out harmful micro-organisms and also by adding disinfectant chemicals. Water is disinfected to kill any pathogens which pass through the filters and to provide a residual dose of disinfectant to kill or inactivate potentially harmful micro-organisms in the storage and distribution systems. Possible pathogens include viruses, bacteria, including *Salmonella*, *Cholera*, *Campylobacter* and *Shigella*, and protozoa, including *Giardia lamblia* and other *cryptosporidia*. Following the introduction of any chemical disinfecting agent, the water is usually held in temporary storage – often called a contact tank or clear well to allow the disinfecting action to complete.

The most common disinfection method involves some form of chlorine. Chlorine is a strong oxidant that rapidly kills many harmful micro-organisms. Because chlorine is a toxic gas, there is a danger of a release associated with its use. This problem is avoided by the use of sodium hypochlorite, which is a relatively inexpensive solution used in household bleach that releases free chlorine when dissolved in water. Chlorine solutions can be generated on site by electrolyzing common salt solutions. A solid form, calcium hypochlorite, releases chlorine on contact with water. Handling the solid, however, requires greater routine human contact through opening bags and pouring than the use of gas cylinders or bleach which are more easily automated. The generation of liquid sodium hypochlorite is both inexpensive and safer than the use of gas or solid chlorine.

All forms of chlorine are widely used, despite their respective drawbacks. One drawback is that chlorine from any source reacts with natural organic compounds in the water to form potentially harmful chemical by-products. These byproducts, trihalomethanes (THMs) and haloacetic acids (HAAs), are both carcinogenic in large quantities and are regulated by the United States Environmental Protection Agency (EPA) and the Drinking Water Inspectorate in the UK. The formation of THMs and haloacetic acids may be minimized by effective removal of as many organics from the water as possible prior to chlorine addition. Although chlorine is effective in killing bacteria, it has limited effectiveness against protozoa that form cysts in water (*Giardia lamblia* and *Cryptosporidium*, both of which are pathogenic).

Chloro-isocyanurate compounds are smartly used for emergency chlorination of drinking water. For routine treatment of public water supplies, there is little or no use of other disinfectants. Some chemicals, such as chloro-isocyanurate compounds are widely used as a stable source of chlorine for the disinfection of swimming pools and in the food industry, Sodium dichloroisocyanurate is used for temporary emergency disinfection applications as a source of free available chlorine in the form of hypochlorous acid (HOCl) with the attendant residual formation of cyanuric acid from its addition to water. The WHO is currently preparing guideline text on Sodium dichloroisocyanurate for inclusion in their future 4th edition of their Guidelines for Drinking Water Quality. In their background document for development of Guidelines for Drinking-water Quality the WHO advised that "The amounts of sodium dichloroisocyanurate used should be the lowest consistent with adequate disinfection, and the concentrations of cyanuric acid should be managed to be kept as low as is reasonably possible.

**Process-Chlorination**

**Advantages** -Well understood disinfectant capability. Established dosing technology. Stable residual in clean networks. Potential for using chlorine for both primary disinfection and distribution, makes for straightforward application.

**Limitations** -Chlorination by-products and taste and odour issues can affect acceptability. Ineffective against Cryptosporidium. By-product formation during distribution. Loss of residual in distribution systems with long residence times.

**Chlorination-** Chlorine will oxidize both iron and manganese to their insoluble forms. The higher the chlorine residual in the water, the faster the reaction is. For typical iron and manganese removal needs, first you treat the water with an initial chlorine residual of 5 to 10 mg/L, then filter the insoluble iron and manganese formed and then finally dechlorinate the water to an acceptable residual for domestic use. Doses beyond 10 mg/L can causes excess concentrations of total trihalomethanes (TTHMs), which can cause adverse health conditions. The dechlorination uses a reducing agent such as sodium bisulphate to remove the excess chlorine. Usually a reaction basin is added after the dechlorination process.

Requires an additional dechlorination step to the water treatment process.

Always add powder to water and never water to the powder. Sodium hypochlorite deteriorates very rapidly (60 to 90 days), especially when exposed to light, and so it should be stored in a cool, dry, dark area FOR YOUR OWN SAFETY You must wear rubber gloves, a rubber apron, and nose and eye protection when you are working with sodium hypochlorite

Contact time – The longer the microorganisms are in contact with the free chlorine, the more effective the disinfection.

**pH of water** – The effectiveness of free chlorine is maximized at pH levels between 6.0 and 7. As chlorine solutions are highly alkaline (i.e. high pH), the dose of chlorine solution increases the pH of the well water and can diminish the effectiveness of the free chlorine. Therefore, it is important to follow the concentration range requirements for free chlorine set out.

**Temperature** – The effectiveness of free chlorine can change with temperature. Free chlorine is more effective at higher temperatures.

**Interfering substances** – Available chlorine will be used up by any inorganic and organic compounds in the well water which will reduce the concentration of free chlorine. Biofilm - Biofilm is a slimy substance that attaches to sides of wells and pumping equipment. The slime consists primarily of nuisance microbes (e.g. iron oxidizing bacteria and sulphatereducing bacteria) that can shield pathogens from the oxidizing action of the free chlorine and reduce the amount of available chlorine. The oxidizing action of the chlorine solution kills bacteria, viruses, protozoans and some protozoal cysts.

Household bleaches typically contain 3 to 6 percent available chlorine. Industrial strength commercial bleach and swimming pool products can contain 10 to 12 percent available chlorine. The unstable nature of sodium hypochlorite makes it sensitive to temperature and light and therefore it has a limited shelf life. For example, sodium hypochlorite degrades extremely rapidly in the hot, sunlit cab of a truck.
Requirements and Best Management Practices - All chlorine products utilized for potable water use must be either fresh unscented bleach or must meet the NSF International Standard 60 for Drinking Water Treatment Chemicals – Health Effects, or an equivalent standard ● Avoid using scented bleach or products such as swimming pool chlorine that typically contain additives such as surfactants, thickeners, stabilizers, perfumes, UV inhibitors, algaecides or other additives as they can impair the quality of the water and aquifer after disinfection and are not designed for potable water use ● Always check product labels to verify product contents and manufacturer's suggested usage as well as Material Safety Data Sheets (MSDS) ● Chlorine products should always be stored in a cool, dry and dark environment.

**EFFECTIVENESS OF CHLORINE-FREE CHLORINE & RESIDUAL**

When a chlorine solution is first added to water the available chlorine will react with substances in the water, and on the surfaces inside. During this reaction, some of the available chlorine is used up by organic and inorganic matter and can no longer kill pathogens and disinfect. The remaining available chlorine is the free chlorine residual that can effectively react to any pathogens. Free chlorine residual consists of two main compounds: hypochlorous acid (HOCl) and hypochlorite ion ($OCl^-$ ). Hypochlorous acid is much more effective (80 to 200 times better) at killing pathogens than the hypochlorite ion.

Examples of common materials or properties that reduce the free chlorine concentration are: ● Alkalinity ● Hydrogen sulphide ($H_2S$) ● Methane ($CH_4$) ● Iron ● Manganese ● Biofilm (iron oxidizing bacteria and sulphate-reducing bacteria) ● Silt ● Clay
Therefore, additional cleaning or additional chlorine solution may be needed to meet the required free chlorine residual concentration range.

**Best Management Practice –** Adjusting the pH of the Chlorine Solution It is important to control the pH to maximize the amount of hypochlorous acid available to kill pathogens. There are several commercial acid products on the market that can lower the pH of water that will be used to make the chlorine solution. Any of the acid products used in the process must not impair the quality of the water in the well or the aquifer and should meet NSF International Standards for potable water or an equivalent standard. Carefully follow the manufacturer's instructions when adding any acids to the water.

## Highlights of the coming issue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Contents

**Mini Review**
A Special Fondness for Lactobacilli.

**Current Trends**
Current trends in hand rub formulations.

**In Profile**
Werner Arber.

**Bug of the Month**
*Shigella sonnei.*

**Did You Know**
Genetically engineered crops: Experiences and prospects.

**Best Practices**
To prevent candidiasis in healthcare.

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