

Editorial

Contents

■ Editorial	1
■ Mini review	2
■ Current Trends	5
■ In Profile	9
■ Relaxed Mood	10
■ Bug of the Month	11
■ Did you Know	12
■ Best Practices	13
■ In Focus	17



Mini Review section – Milk and milk products have played significant role in the human food since ancient times. The study of microorganisms that are associated with milk and milk products in all aspects is defined as “Dairy Microbiology”. Milk in addition to being a nutritious food for humans, provides a favourable environment for the growth of microorganisms. Hygienic milk is concerned with the production of clean, wholesome milk that is free from bacteria or other disease causing micro-organisms and maintenance of this condition from farm to the consumers.

Current Trends section – Biofilms are found widely in nature and have been rigorously studied for many years. However, the study of biofilms in relation to health and in particular wounds is a relatively recent development. The National Institutes of Health (NIH) suggest that 80% of human infectious disease is caused by biofilm, usually manifesting as chronic infection. These chronic infections often viewed as benign are in fact insidious and progressive in nature and produce death tolls each year rivaling that of heart disease or cancer, yet clinicians appear to have developed an extremely passive relationship with biofilm disease including those implicated in wound infection.

In Profile Scientist – Howard Martin Temin was an American geneticist and virologist. He discovered reverse transcriptase in the 1970s at the University of Wisconsin–Madison, for which he shared the 1975 Nobel Prize in Physiology or Medicine with Renato Dulbecco and David Baltimore.

Bug of the Month – *Cyclospora cayentanensis* is a coccidian parasite that causes a diarrheal disease called cyclosporiasis in humans and possibly in other primates. *Cyclospora* is generally transmitted when infected feces contaminate food or water. It's unlikely to be transmitted directly from person to person because the *Cyclospora* parasite needs time (days to weeks) after being passed in a bowel movement to become infectious for another person.

Did You Know? – Researchers discover biomarkers that indicate early brain injury in extreme premature infants. Extremely premature infants are at a high risk for brain damage. Researchers have now found possible targets for the early treatment of such damage outside the brain: Bacteria in the gut of premature infants may play a key role. The research team found that the overgrowth of the gastrointestinal tract with the bacterium *Klebsiella* is associated with an increased presence of certain immune cells and the development of neurological damage in premature babies.

Best Practices – In May 2004, the WHA approved the creation of an international alliance to improve patient safety globally; WHO Patient Safety was launched the following October. For the first time, heads of agencies, policy-makers and patient groups from around the world came together to advance attainment of the goal of “First, do no harm” and to reduce the adverse consequences of unsafe health care. The purpose of WHO Patient Safety is to facilitate patient safety policy and practice.

Microbiology of milk and milk products (I)

Introduction



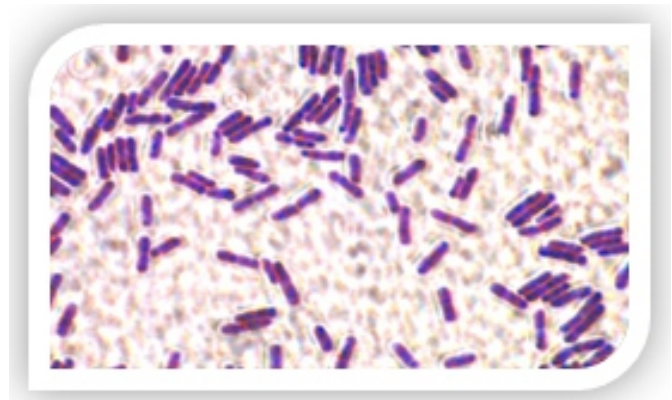
Milk and milk products have played significant role in the human food since ancient times. Milk plays an important role in human diet contributing essential nutrient. The need for milk has given rise to a whole new milk and milk product industry and studies regarding the same. The study of microorganisms that are associated with milk and milk products in all aspects is defined as “Dairy Microbiology”. Milk is described as a whole, fresh, clean, lacteal secretion obtained from the complete milking of healthy milch animal containing the minimum prescribed levels of fat and solids non-fat (SNF). Hygienic milk is concerned with the production of clean, wholesome milk that is free from bacteria or other disease causing micro-organisms and maintenance of this condition from farm to the consumers.

Milk is considered as the most nutritious and complete food for neonates and adult human beings both. Milk in addition to being a nutritious food for humans, provides a favourable environment for the growth of microorganisms. Yeasts, moulds and a broad spectrum of bacteria can grow in milk, particularly at temperatures above 16°C. Microbes can enter milk via the cow, air, feedstuffs, milk handling equipment and the milker. Once microorganisms get into the milk their numbers increase rapidly.

Micro-organisms found in milk

Milk drawn from a healthy milk animal already contains some bacteria. Most of the changes which take place in the flavour and appearance of milk, after it is drawn from udder are the results of the activities of microbes. These microbes are of two types i.e. favourable – which brings favourable changes in flavour & appearance while pathogenic – which may cause diseases. The favourable are carefully propagated while pathogenic (unfavourable) are destroyed to make the milk & its products safe for human consumption.

Bacteria: - Are microscopic, unicellular, occurs in the form of spherical, cylindrical or spiral cells, size 1-5µm. Spore forming bacteria produce trouble in dairy industry because of their resistance to pasteurization and sanitization produces. Greater the bacteriological count in milk, the lower is its bacteriological quality.



The following bacteriological standards of raw milk are suggested as a guide for grading raw milk in India.

SPC/ml (org)	Grade
Not exceeding 2,00,000	Very good
Between 2,00,000 and 10,00,000	Good
Between 10,00,000 and 50,00,000	Fair
Over 50,00,000	Poor

*Pasteurized milk should have a SPC/ml (org) not exceeding 30,000

Bacterial types commonly associated with milk

<i>Pseudomonas</i>	Spoilage
<i>Brucella</i>	Pathogenic
Enterobacteriaceae	Pathogenic and spoilage
Staphylococci	
<i>Staphylococcus aureus</i>	Pathogenic
Streptococcus	
<i>S. agalactiae</i>	Pathogenic
<i>S. thermophilus</i>	Acid fermentation
<i>S. lactis</i>	Acid fermentation
<i>S. lactis</i> -diacetyllic	Flavour production
<i>S. cremoris</i>	Acid fermentation
<i>Leuconostoc lactis</i>	Acid fermentation
<i>Bacillus cereus</i>	Spoilage
Lactobacillus	
<i>L. lactis</i>	Acid production
<i>L. bulgaricus</i>	Acid production
<i>L. acidophilus</i>	Acid production
<i>Propionibacterium</i>	Acid production
<i>Mycobacterium tuberculosis</i>	Pathogenic

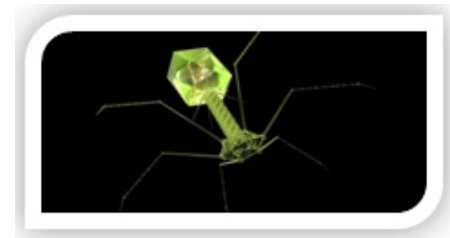
Moulds: - Multi-cellular, at maturity known as Mycelium. Useful in cheese making which is responsible for defects in butter and other milk products. Most spores of moulds are destroyed by pasteurization.



Yeast: - Unicellular, larger than bacteria, egg-shaped single-cell fungus that is only visible with a microscope. Yeasts are introduced into the milk as contamination during the milking process or as recontamination after the heat treatment. Destroyed during pasteurization.

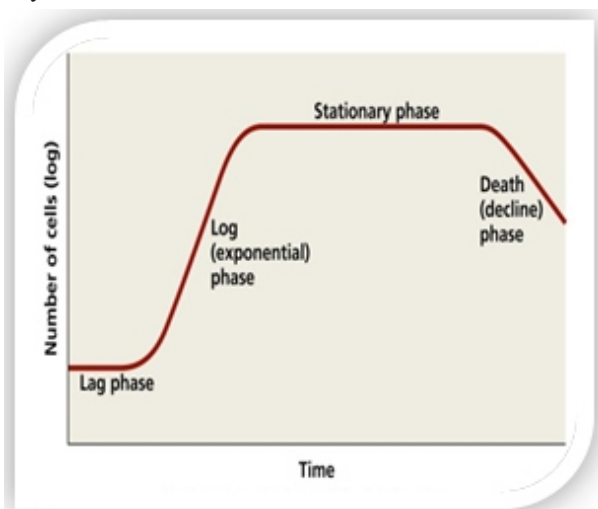


Viruses: - Are ultra-microscopic forms of like can be destroyed by pasteurization or higher heat treatment.



Growth of Micro-organisms

Bacteria multiply during production and holding of milk, depending on storage time and conditions. The changes take place in the physico-chemical properties of milk are result of the activities of the individual microbial cells during their period of growth and reproduction or of substances produced during such activity.



Stages of growth

- Initial stationary phase
- Lag phase (Phase of adjustment)
- Accelerated growth phase (log phase)
- Maximum stationary phase
- Phase of accelerated death

Factors Influencing Growth

- Food supply – Milk and its products are good food source, provides all food requirements.
- Moisture – Milk contains adequate moisture to development.
- Air – Supplies O₂ to aerobic bacteria and moulds.
- Acidity or pH – Preferably range 5.6 to 7.5.
- Preservatives – Check growth depending upon concentration.
- Light – More or less harmful.
- Concentration – High sucrose or salt content check growth.
- Temperature – Important means for controlling growth.
- According to their optimum growth temperature, bacteria can be classified into :
 - Psychotropic – can grow at refrigeration temp. 5°C-7°C.
 - Mesophilic – can grow at temp. 20°C-40°C.
 - Thermophilic – can grow at temp. above 50°C.

Products of Microbial Growth:-

- Enzymes
- Decomposition products (fats, proteins, sugars)
- Pigments
- Toxins
- Miscellaneous changes

Results of Microbial Growth in Milk:-

- Souring: - Most common, due to transformation of lactose into lactic acid & other volatile acids & compounds, principally by lactic acid bacteria.
- Souring & gassiness: - Caused by coli group, indicates contamination of milk and its products.
- Aroma production: - Due to production of desirable flavour compounds (diacetyl).
- Proteolysis: - Protein decomposition leading to unpleasant odour.
- Ropiness: - Long threads of milk are formed while pouring. Mainly Alkaligenous viscus.
- Sweet curdling: - Due to production of a remain like enzyme curdles milk without souring.

Destruction of Micro-organisms:-

May be done by following means:

- Heat – Most widely used method: Pasteurization & sterilization.
- Ionizing radiation – Such as ultraviolet rays etc.
- High frequency sound waves – Supersonic and ultrasonic.
- Electricity – Microbes are destroyed actually by heat generated.
- Pressure – Should be about 600 times greater than atmospheric pressure.
- Chemicals – Includes acids, alkalis, hydrogen peroxide, halogens etc.

Action of Microbes on Milk

Microbial growth can be controlled by cooling the milk. Most microorganisms reproduce slowly in colder environments. Cooling milk also slows chemical deterioration. The temperature of freshly drawn milk is about 38°C. Bacteria multiply very

rapidly in warm milk and milk sours rapidly if held at these temperatures. If the milk is not cooled and is stored in the shade at an average air temperature of 16°C, the temperature of the milk will only have fallen to 28°C after 3 hours. Cooling the milk with running water will reduce the temperature to 16°C after 1 hour. At this temperature bacterial growth will be reduced and enzyme activity retarded. Thus, milk will keep longer if cooled.

Natural souring of milk may be advantageous: for example, in smallholder butter-making, the acid developed assists in the extraction of fat during churning. The low pH retards growth of lipolytic and proteolytic bacteria and therefore protects the fat and protein in the milk. The acidity of the milk also inhibits the growth of pathogens. It does not, however, retard the growth of molds. Naturally soured milk is used to make many products, e.g. yoghurt, sour cream, ripened buttermilk and cheese. These products provide ways of preserving milk and are also pleasant to consume. They are produced by the action of fermentative bacteria on lactose and are more readily digested than fresh milk. The initial microflora of raw milk reflects directly microbial contamination during production. The microflora in milk when it leaves the farm is determined by the temperature to which it has been cooled and the temperature at which it has been stored. The initial bacterial count of milk may range from less than 1000 cells/ml to 106/ml. High counts (more than 105/ml) are evidence of poor production hygiene.

Milk Pasteurisation

Pasteurisation is the process used to destroy bacteria in milk. In pasteurisation, the milk is heated to a temperature sufficient to kill pathogenic bacteria, but well below its boiling point. This also kills many non-pathogenic organisms and thereby extends the storage stability of the milk.

In pasteurizing, two types of processes can be used: slow and rapid. Slow pasteurization uses pasteurization temperatures for several minutes; e.g., typical temperature–time combinations are 63 to 65°C over 30 minutes or 75°C over 8 to 10 minutes. Rapid, high or flash pasteurization uses numerous time/temperature combinations are recommended but the most usual is 72°C for 15 seconds followed by rapid cooling to below 10°C. It is carried out as a continuous process using a plate heat-exchanger to heat the milk and a holding section to ensure that the milk is completely pasteurised. Milk is normally pasteurised prior to sale as liquid

milk. Pasteurisation is used to reduce the microbial counts in milk for cheesemaking, and cream is pasteurised prior to tempering for butter making in some factories. Batch pasteurisation is used where milk quantities are too small to justify the use of a plate heat exchanger.

In batch pasteurisation, fixed quantities of milk are heated to 63°C and held at this temperature for 30 minutes. The milk is then cooled to 5°C and packed. The lower temperature used for batch pasteurisation means that a longer time is required to complete the process 30 minutes at 63°C, compared with 15 seconds at 72°C.

Effects of pasteurisation on milk

Pasteurisation reduces the cream layer, since some of the fat globule membrane constituents are denatured. This inhibits clustering of the fat globules and consequently reduces the extent of creaming. However, pasteurisation does not reduce the fat content of milk. Pasteurisation has little effect on the nutritive value of milk. The major nutrients are not altered. There is some loss of vitamin C and B group vitamins, but this is insignificant. The process kills many fermentative organisms as well as pathogens. Microorganisms that survive pasteurisation are putrefactive. Although pasteurised milk has a storage stability of 2 to 3 days, subsequent deterioration is caused by putrefactive organisms. Thus, pasteurised milk will putrefy rather than develop acidity. In rural milk processing, many processes depend on the development of acidity, and hence pasteurisation may not be appropriate.

Milk sterilisation

In pasteurisation, milk receives mild heat treatment to reduce the number of bacteria present. In sterilisation, milk is subjected to severe heat treatment that ensures almost complete destruction of the microbial population. The product is then said to be commercially sterile. Time/temperature treatments of above 100°C for 15 to 40 minutes are used. The product has a longer shelf life than pasteurised milk. Another method of sterilisation is ultra-heat treatment, or UHT. In this system, milk is heated under pressure to about 140°C for 4 seconds. The product is virtually sterile. However, it retains more of the properties of fresh milk than conventionally sterilised milk.

Wound Infection and Modern Biocides Contd...

Biofilm's Defenses (Resistance)

The survivability of biofilm is a result of adaptation strategies developed over millions of years. These strategies together with brief explanations of their mechanisms may be found in Table 3.

Strategies	Mechanisms
Extracellular polymeric substance (EPS)	Constructed by the bacteria of the biofilm to protect the community from desiccation, predators, immune cells, and toxins. The components of the EPS can include pathogen and host polysaccharides, proteins, and nucleic acids. The chemical structure of the EPS may also work to prevent some antimicrobials from entering the biofilm.
Enzymatic Protection	Metabolically active cells are able to produce enzymes such as catalase or beta lactamase that can neutralize biocides and antibiotics and shield the inner members of the community.
Altered microenvironments	By-products of the biofilm create acidic and hypoxic areas which produce slow growth and diversify the ecology of the biofilm.
Plastic phenotype	Biofilms have a dramatically different expression of proteins. Up to 50% of the outer membrane proteins are different from their planktonic counterparts, which demonstrates the phenotypic heterogeneity that can be found within a species.
Heterogeneity	When combined with slower growth, heterogeneity makes most antibiotics less effective.
Quorum sensing	Where groups of bacteria are present, cell-to-cell signaling takes place. The bacterial pheromones facilitate cooperation or result in competitive antagonism, which work together to yield a climax biofilm community that is best suited for the stresses and nutrients of the wound environment.
Evasion of Host Defenses	Most chronic infections are firmly entrenched within the host. Complement pathways, antibodies and even white blood cells have been found to be very ineffective against biofilm.

Imaging studies, including light and electron microscopy of samples from 50 wounds, demonstrated that 60% of chronic wounds possess biofilm, whereas 16 acute wounds failed to show significant biofilm. The chronic wounds healed in over 3 months (a delayed wound healing trajectory), whereas all the acute wounds healed within 3 weeks. This suggests that not only is biofilm present but it may impair healing. A biofilm model may explain many of the clinical challenges that can make wound care so intricate and complex. It has been established that chronic wounds become "stuck" in a chronic inflammatory state. This chronic inflammation is defined at a molecular level by increases in macrophage-derived MMPs 2 and 9 and neutrophil-derived MMP 8 and elastase. At the cellular level, excessive neutrophils predominate within the wound bed. The presence of biofilm on the surface of the wound can explain the molecular and cellular findings in chronic wounds. Differences in opinion of the value of antibiotics in acute and chronic wound care may be found. When antibiotics are used as a single agent, they fail to "heal" a chronic wound the vast majority of times. Clinically, what is often seen following antibiotic administration is a short-term improvement in the wound, that is followed by a subsequent deterioration or recalcitrance. This is possibly due to failure of the antibiotic to

reduce the bioburden to a level at which the host defenses can prevail, resulting in reconstruction of the biofilm and enhanced resistance. Clinical support for biofilm's role in impaired healing is demonstrated by a retrospective study which showed that wounds treated with anti-biofilm strategies were more likely to heal when compared to those treated by standard care methods. The results provide good working explanations for what is seen clinically in wound care.

Biofilm-Based Wound Management

Suppressing wound biofilm while managing the other known barriers to wound healing (pressure, poor perfusion, poor nutrition, etc.) holds the potential to radically advance wound healing.

Chronic wounds are often managed using a single strategy (e.g. enzyme, topical antiseptic, or a specialty dressing) at a time. Early progress may be observed but often healing is stalled and another strategy is applied. Sequential strategies often result in failure to close the wound.

Using a biofilm model to explain the organization of wound

bacteria, it becomes clear that a single strategy is unlikely to succeed. Biofilms are polymicrobial with important interspecies synergies along with the ability to control their environment through modifications of their protective matrix. This has led dentistry and many other industries to adopt a multiple concurrent strategy in managing biofilms. Dentistry has managed biofilm (dental plaque) successfully over several decades. This has resulted in the well-known daily regimen of: debridement (brushing) at the same time applying an anti-biofilm substance, namely toothpaste. These anti-biofilm agents block reattachment, impair EPS formation, or are biocidal, killing the community members of the plaque. For more recalcitrant plaques, harsher biocides are applied through oral rinses and aggressive debridement can be carried out through flossing, ultrasonic debridement, or professional cleaning. This process of suppression, which will continue throughout our lifetime, does not aim to eradicate the biofilm but to suppress it below a level that would cause periodontal disease. The same principles seem reasonable when applied to managing wound biofilm. It is important to note that as biofilm reconstitutes itself and before it has formed a stable climax community, it is much more susceptible to antimicrobials. Frequent debridement sets the stage for treating agents to be more effective.

Debridement provides a cornerstone in the management of chronic wounds and evidence demonstrates that frequent debridement improves wound healing. However, in most wounds, when slough or biofilm is removed from the surface, it rapidly reconstitutes itself on the surface within 24 hours. Clinically, what is seen is a cleanbleeding wound bed postdebridement one day but the next day the slough that was removed the day before debridement is seen on the wound bed. In the laboratory, it takes biofilm about 24 hours to re-establish the biomass of the community.

Topical antiseptics, such as silver and honey provide some evidence of their value in managing biofilm. Empirically, the authors have noted that iodine preparations, particularly cadexomer, also possess the capability to manage biofilm infection. The goal is not eradication but to get multiple different strategies producing significant stress to the biofilm at the same time.

It is recognized that biofilm demonstrates increased resistance to antibiotics, biocides and host defenses. However, when used concomitantly with frequent debridement and other topical agents that impair biofilm defenses, antibiotics can be more successful. Clinical medicine has found that for biofilm diseases such as osteomyelitis and endocarditis, higher doses of antibiotics for longer periods of time are more successful. In a chronic wound, use of antibiotics as a single agent struggles to suppress biofilm, but when used in conjunction with the other strategies indicated above, does show significant impact in healing wounds. Because wound biofilms are resistant to antibiotics and host defenses, clinicians struggle to manage successfully many chronic wounds. Aggressively targeting wound biofilm suppresses the bioburden over a period of time to a level at which the host immune response will prevail and resolve the chronic wound.

Silver

Silver-based products are extensively used in wound care (Klasen 2000a, 2000b; Demling and De Santi, 2001; Clarke, 2003), with

skin discolouration (argyria) and irritation being the only visible side effects (White, 2002). It is thought that silver has a number of antimicrobial modes of action (Thurman and Gerba, 1989; Russell and Hugo, 1994). However, questions have been raised over the long-term use of these dressings, especially in infants (Denyer, 2009a; 2009b).

Recently, there have been concerns about silver toxicity (Parsons et al, 2005; Burd et al, 2007), and the systemic uptake and deposition of silver in organs have been noted in a number of studies (Wan et al, 1991; Denyer, 2009a; Wang et al, 2009). To date, the pathological consequences of this are unknown. Added to this, there are fears about the emergence of silver resistance (Percival et al, 2005; Loh et al, 2009). It would seem that, in academic circles at least, questions exist over its continued widespread clinical use. This has been further enhanced by questions about its cost-effectiveness (Bergin and Wraight, 2006; Chaby et al, 2007; Michaels et al, 2009), which in some areas has led to product restrictions.

Iodine

Iodine-based products have been used in wound care for many years. Like all antiseptics, iodine simultaneously affects multiple sites in microbial cells, resulting in cell disruption and death (Cooper, 2007). However, not only have its antimicrobial efficacy and chemical stability been debated, but also its toxicity to host tissues and the ensuing effect on patient comfort (Kramer, 1999; Wilson et al, 2005). It has been found that providone-iodine is not as effective as some other biocides in eradicating *Staphylococcus epidermis* within *in vitro* biofilms (Presterl et al, 2007). Cadexomer iodine provides sufficient iodine for biofilm suppression without causing significant damage to the host (Akiyama et al, 2004; Rhoads et al, 2008) but pain has been reported as a side effect of its use (Hansson, 1998).

Chlorhexidine

Chlorhexidine has been used clinically for about 50 years (Russell, 2002). It is active against gram-negative organisms such as *Pseudomonas aeruginosa* and gram-positive organisms such as *Staphylococcus aureus* and *Escherichia coli*, although methicillin-resistant *Staphylococcus aureus* (MRSA) resistance has been recorded (Cookson, 2000). Chlorhexidine appears to be relatively safe, with little effect on the healing process.

However, results from studies are insufficient to draw conclusions about its use on open wounds. In addition, there are concerns about the safety of additives frequently used in chlorhexidine-based preparations to modify their handling properties. More human trials need be performed to assess its efficacy and long-term safety (White et al, 2001; Main, 2008)

Honey

In recent years, there has been resurgence in interest in honey-based products for bioburden management (White, 2002). The exact mode of action of honey is not yet fully understood. However, it is hyperosmolar and, thus, restricts the availability of environmental water to bacteria and other organisms (Molan, 2001), leading to cell disruption and death. However, this effect is lessened as the honey becomes more diluted by wound exudate (Molan, 1999). A secondary action is the release of hydrogen peroxide as the honey is diluted by exudate (Molan and Betts, 2004). However, some honeys, particularly *Leptospermum* or manuka varieties, have been found to retain their bactericidal

properties even without the presence of hydrogen peroxide (Cooper et al, 2002a; 2002b), which is thought to be associated with a phytochemical component (Karayil et al, 1998; Molan, 2002). The antibacterial properties of honey, therefore, vary according to its source.

The Dilemma

Careful and objective review of the literature suggests that the use of many antiseptics in wound management must be subject to a risk-benefit assessment of possible local toxicity and beneficial antibacterial action (Brennan and Leaper, 1985). In short, it is advised that, before use, the beneficial antimicrobial effects and bioavailability should be weighed against any possible cellular toxicity (Wilson et al, 2005).

Given the widespread availability of antimicrobial products, factors likely to influence selection include:

- Clinician familiarity
- Availability, cost, and reimbursement issues
- Ease of use and implications for pattern of care
- Efficacy and safety (WUWHS, 2008)

As there appears to be concern about the safety and efficacy of commonly used and familiar antimicrobial products, clinicians need to cast the net wider and search for alternative safe, effective and efficient products.

The antiseptic agent polyhexamethylene biguanide (also known as polyhexanide 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 (Sorensen et al, 2003; Ousey and McIntosh, 2009). AMPs have a broad spectrum of activity against bacteria, viruses and fungi, inducing cell death by disrupting cell membrane integrity (Ikeda et al, 1983; Ikeda et al, 1984; Moore and Gray, 2007;).

The structural similarities to AMP mean that PHMB can infiltrate bacterial cell membranes and kill bacteria in a similar way (Moore and Gray, 2007). 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 (Davies et al, 1968; Davies and Field, 1969; Broxton et al, 1984a; Yasuda et al, 2003; Gilbert, 2006), resulting in cell death. PHMB has an effect on both planktonic bacteria and those in biofilms (Seipp et al, 2005; Pietsch and Kraft, 2006; Harbs and Siebert, 2007). 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 (Kingsley et al, 2009). 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 (Allen et al, 2004).

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) (Wérthen et al, 2004; Krebs et al, 2005). 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 (Pinto et al, 2009).

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 sulphadiazine (Müller and Kramer, 2008). In addition, no known resistance to PHMB has been reported, most likely owing to its rapid and non-specific bactericidal activity (Moore and Gray, 2007).

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 (Daeschlein et al, 2007; Galitz et al, 2009)
- Reduces wound malodour (Daeschlein et al, 2007)
- Increases formation of granulation tissue (Mueller and Krebsbach, 2008)
- Increases keratinocyte and fibroblast activity (Wiegand et al, 2008a)
- Reduces slough within the wound (Mueller and Krebsbach, 2008)
- Reduces MMP-induced periwound breakdown (Cazzaniga et al, 2002; Werthen et al, 2004)
- Helps remove non-viable tissue (Kaehn, 2009)

The success of PHMB has resulted in its recommendations as the primary antimicrobial in many European countries (Dissemond et al, 2010) and has prompted the publication of a UK consensus review (Wounds UK, 2010).

Bibliography:

- Allen MJ, Morby AP, White GF (2004) Cooperativity in the binding of the cationic biocide polyhexamethylene biguanide to nucleic acids. *Biochem Biophys Res Commun* 318(2): 397–404
- Ansorg RA, Azem T, Fabry WH, Rath PM (2002) Influence of mucin on the activity of the antiseptic Lavasept against *Staphylococcus aureus*. *Chemotherapy* 48(3): 129–33
- Bergin SM, Wraight P (2006) Silver based wound dressings and topical agents for treating diabetic foot ulcers. *Cochrane Database Syst Rev Issue 1 Art. No.: CD005082*. DOI:10.1002/14651858.CD005082.pub2
- Best Practice Statement (2011) The Use of Topical Antimicrobial Agents in Wound Management. Wounds UK, London. Available at: <http://www.wounds-uk.com/best-practice-statements/bestpractice-statement-the-use-of-topical-antisepticantimicrobialagents-in-wound-management-2nd-edition>

- Brennan SS, Leaper DJ (1985) The effects of antiseptics on the healing wound: a study using the rabbit ear chamber. *Br J Surg* 72(10): 780–2
- Broxton P, Woodcock PM, Heatley F, Gilbert P (1984a) Interaction of some polyhexamethylene biguanides and membrane phospholipids in *Escherichia coli*. *J Appl Bacteriol* 57(1): 115–24
- Burd A, Kwok CH, Hung SC, Chan HS, Gu H, Lam WK, Huang L (2007) A comparative study of the cytotoxicity of silver-based dressings in monolayer cell, tissue explant and animal models. *Wound Repair Regen* 15(1): 94–104
- Cazzaniga A, Serralta V, Davis S, Orr R, Eaglstein W, Mertz PM (2002) The effect of an antimicrobial gauze dressing impregnated with 0.2-percent polyhexamethylene biguanide as a barrier to prevent *Pseudomonas aeruginosa* wound invasion. *Wounds* 14(5): 169–76
- Chaby G, Senet P, Vaneau M, Martel P, Gauillaume JC et al (2007) Dressings for acute and chronic wounds: a systematic review. *Arch Dermatol* 143(10): 1297–304
- Clarke S (2003) The silver solution. *Journal of Community Nursing* 17(1): 25–8
- Cookson BD (2000) Methicillin-resistant *Staphylococcus aureus* in the community: new battlefronts, or are the battles lost? *Infect Control Hosp Epidemiol* 21(6): 398–403
- Cooper RA (2004) A review of the evidence for the use of topical antimicrobial agents in wound care. *WorldWide Wounds*. Available online at: <http://tinyurl.com/76m6ctg>
- Cooper RA (2007) Iodine revisited. *Int Wound J* 4(2): 124–37
- Cooper RA, Halas E, Molan PC (2002b) The efficacy of honey in inhibiting strains of *Pseudomonas aeruginosa* from infected burns. *J Burn Care Rehabil* 23(6): 366–70
- Cooper RA, Molan PC, Harding KG (2002a) The sensitivity to honey of Gram-positive cocci of clinical significance isolated from wounds. *J Appl Microbiol* 93(5): 857–63
- Daeschlein G, Assadian O, Bruck JC, Meinel C, Kramer A et al (2007) Feasibility and clinical applicability of polyhexanide for treatment of second-degree burn wounds. *Skin Pharmacol Physiol* 20(6): 292–6
- Davies A, Bentley M, Field BS (1968) Comparison of the action of vantocil, cetrinide and chlorhexidine on *Escherichia coli* and its spheroplasts and the protoplasts of Gram-positive bacteria. *J Appl Bacteriol* 31(4): 448–61
- Davies A, Field BS (1969) Action of biguanides, phenols and detergents on *Escherichia coli* and its spheroplasts. *J Appl Bacteriol* 32(2): 233–43
- Demling R, DeSanti L (2001) The role of silver in wound healing: part 1. Effects of silver on wound management. *Wounds: A Compendium of Clinical Research and Practice* 13(1 Suppl A): 4–15
- Denyer J (2009a) Epidermal bullosa and silver absorption in paediatrics. Presentation at Wounds UK Conference, 11 November 2009, Harrogate
- Denyer J (2009b) Management of the infant with epidermolysis bullosa. *Infant* 5(6): 168–71
- Dissemmond J, Gerber V, Kramer A, Riepe G, Strohal R et al (2010) A practice-oriented expert recommendation for treatment of critically colonised and locally infected wounds using polyhexanide. *J Tissue Viability* 19(3): 106–15
- Dow G (2001) Infection in chronic wounds. In: Krasner DL, Rodeheaver, GT, Sibbald RG eds. *Chronic wound care: a clinical source book for healthcare professionals* (3rd edn). HMP Communications Wayne, PA: 385–356
- Dowsett C, Edwards-Jones V, Davies S (2004) Infection control for wound bed preparation. *Br J Community Nurs* 9(9s): 12–17
- Eberlein Th, Wild Th (2008) Approaches to therapy and an outlook on improving the clinical data situation, with a special focus on the polyhexanide-containing Suprasorb X + PHMB Hydrobalance dressing. EWMA conference extended abstract. Available online at: <http://tinyurl.com/cfs6rak>
- Edwards R, Harding KG (2004) Bacteria and wound healing. *Curr Opin Infect Dis* 17(2): 91–6
- European Wound Management Association (2006) Position Document: Management of wound infection. MEP Ltd, London
- Galitz C, Hämmerle G, Signer M, Traber J, Eberlein T et al (2009) Polyhexanide versus silver wound dressings: first interim results of a controlled, randomized, prospective, multicentric study. Poster presentation. European Wound Management Association (EWMA) Conference, Helsinki. Available at: <http://tinyurl.com/737n6go>
- Gilbert P (2006) Avoiding the resistance pitfall in infection control. Does the use of antiseptic products contribute to the spread of antibiotic resistance? *Ostomy Wound Manage* 52(10A Suppl): 1S–3S
- Harbs N, Siebert J (2007) In vitro efficacy of octenidine and polyhexanide against biofilms composed of *Pseudomonas aeruginosa*. *GMS Krankenhaushyg Interdiszpl* 2(2): Doc.45 (20071228)
- Hansson C (1998) The effects of cadexomer iodine paste in the treatment of venous leg ulcers compared with hydrocolloid dressing and paraffin gauze dressing. Cadexomer Iodine Study Group. *Int J Dermatol* 37(5): 390–6
- Hunt TK, Hopf HW (1997) Wound healing and wound infection. What surgeons and anaesthesiologists can do. *Surg Clin North Am* 77(3): 587–606
- Ikeda T, Ledwith A, Bamford CH, Hann RA (1984) Interaction of a polymeric biguanide biocide with phospholipid membranes. *Biochim Biophys Acta* 769(1): 57–66
- Ikeda T, Tazuke S, Watanabe M (1983) Interaction of biologically active molecules with phospholipid membranes. I. Fluorescence depolarization studies on the effect of polymeric biocide bearing biguanide groups in the main chain. *Biochim Biophys Acta* 735(3): 380–6
- Karayil S, Deshpande SD, Koppikar GV (1998) Effect of honey on multidrug resistant organisms and its synergistic action with three common antibiotics. *J Postgrad Med* 44(4): 93–6

Howard Martin Temin

Howard Temin was born on December 10, 1934 in Philadelphia, Pennsylvania, United States of America, the second of three sons of Annette and Henry Temin. His father was an attorney, and his mother has been continually active in civic affairs, especially educational ones. His older brother, Michael, is also an attorney in Philadelphia, and younger brother, Peter, is a Professor of Economics at the Massachusetts Institute of Technology, Cambridge, Mass.

He received elementary and high school education in the public schools of Philadelphia. His specific interest in biological research was focused by summers (1949-1952) spent in a program for high school students at the Jackson Laboratory in Bar Harbor, Maine, and a summer (1953) spent at the Institute for Cancer Research in Philadelphia. He attended Swarthmore College from 1951 to 1955, majoring and minoring in biology in the honors program. After another summer (1955) at the Jackson Laboratory, he became a graduate student in biology at the California Institute of Technology in Pasadena, California, majoring in experimental embryology. His doctoral thesis was on Rous sarcoma virus. Much of his early work on this virus was carried out with the dose collaboration of Dr. Harry Rubin, then a postdoctoral fellow in Professor Dulbecco's laboratory. After finishing his Ph.D. degree in 1959, he remained for an additional year in Professor Dulbecco's laboratory as a postdoctoral fellow. In that year, he performed the experiments that led to the formulation in the same year of the provirus hypothesis for Rous sarcoma virus.

In 1960, he moved to Madison as an Assistant Professor in the McArdle Laboratory for Cancer Research, which is also the Department of Oncology, in the Medical School, The University of Wisconsin-Madison. His first laboratory was in

the basement, with a sump in my tissue culture lab and with steam pipes for the entire building in my biochemistry lab. Here he performed the experiments that led in 1964 to my formulating the DNA provirus hypothesis. In the fall of 1964, the entire department moved to a new building. He became successively Associate Professor, Full Professor, Wisconsin Alumni Research Foundation Professor of Cancer Research and in 1974, American Cancer Society Professor of Viral Oncology and Cell Biology. From 1964 to 1974, he also held a Research Career Development Award from the National Cancer Institute.

During his first years at Wisconsin, he worked with only two technicians. His first postdoctoral fellow joined me in 1963, and my first graduate student, in 1965. I had no more than two or three postdoctoral fellows and graduate students at one time until about 1968.

During the late 1960's, about half of his time was spent in studying the control of multiplication of uninfected and Rous sarcoma virus-infected cells in culture. This work led to my appreciation of the role of specific serum factors in the control of cell multiplication and the demonstration that a multiplication-stimulating factor in calf serum for chicken fibroblasts was the same as somatomedin.

He served on the editorial boards of several journals, including the *Journal of Cellular Physiology*, the *Journal of Virology*, and the *Proceedings of the National Academy of Sciences U.S.A.* He has also been a member of the Virology Study Section of the National Institutes of Health.

Since the general acceptance of the DNA provirus hypothesis in 1970, he has received many honors, including the Warren Triennial Prize, the Pap Award of the Papanicolaou Institute, Miami, Florida; the Bertner Award, M. D. Anderson Hospital and Tumor Institute, Houston, Texas; the U. S. Steel Foundation Award in Molecular Biology, National Academy of Sciences U.S.A.; the American Chemical Society Award in Enzyme Chemistry; the Griffuel Prize, Association Development Recherche Cancer, Villejuif, France; the G.H.A. Clowes Award, American Association for Cancer Research; the Gairdner International Award (with David Baltimore); the Albert Lasker Award in Basic Medical Research; and honorary degrees from Swarthmore College and New York Medical College. I have also presented several honorary lectures. He is a fellow of the American Academy of Arts and Sciences and a member of the National Academy of Sciences, U.S.A.

In 1962 he married Rayla Greenberg of Brooklyn, New York, a population geneticist. She has been a constant source of support and warmth. They have two daughters, Sarah Beth and Miriam.

Jokes



My wife told me she needs more space
I said no problem
and locked her
out of the house



Doctor: You should take at least 10 Glasses of water every day.
Patient: It is Impossible.
Doctor: Why?
Patient: I have only 4 Glasses at home..!



How do you know you're ugly?
If you always get handed the camera for **group photos**.

One Humour A Day Keeps the Boredom Away:

I asked my new **Girlfriend** what sort of books she's interested in..



She said:
Cheque books.



Boy: Where are you going now?
Girl: For Suicide..
Boy: Then, Why so much Make-up?
Girl: You Idiot... Tomorrow My Photo will come in Newspaper..!

Girl friend: "where is my birthday present?"

Boy friend: "can you see a red color car on the roadside?"

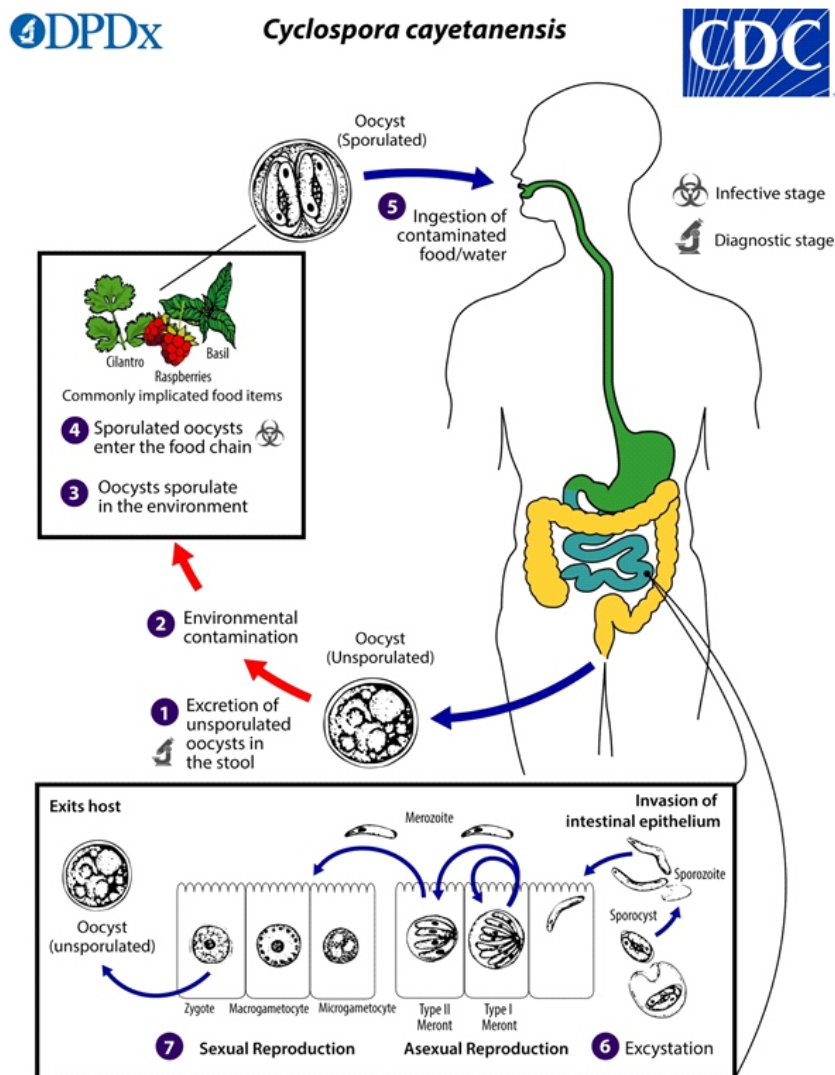
Girl friend with excitement: "Wow!"

..
..

Boy friend: "I have bought a same color nail polish for you!!" 🤔



Cyclospora cayetanensis – A Food-Borne and Waterborne Parasite



When freshly passed in stools, the oocyst is not infective (1) (thus, direct fecal-oral transmission cannot occur; this differentiates *Cyclospora* from another important coccidian parasite, *Cryptosporidium*). In the environment (2), sporulation occurs after days or weeks at temperatures between 22°C to 32°C, resulting in division of the sporont into two sporocysts, each containing two elongate sporozoites (3). The sporulated oocysts can contaminate fresh produce and water (4) which are then ingested (5). The oocysts excyst in the gastrointestinal tract, freeing the sporozoites, which invade the epithelial cells of the small intestine (6). Inside the cells they undergo asexual multiplication into type I and type II meronts. Merozoites from type I meronts likely remain in the asexual cycle, while merozoites from type II meronts undergo sexual development into macrogametocytes and microgametocytes upon invasion of another host cell. Fertilization occurs, and the zygote develops to an oocyst which is released from the host cell and shed in the stool (7). Several aspects of intracellular replication and development are still unknown, and the potential mechanisms of contamination of food and water are still under investigation.

Hosts

Humans appear to be the only major host for *C. cayetanensis*.

Occasionally, cysts are recovered from animal feces, but it is likely that this represents spurious passage following coprophagy.

Geographic Distribution

Cyclosporiasis has been reported in many countries, but is most common in tropical and subtropical areas. In the United States, the majority of cases are reported during the spring and summer months. Outbreaks have been identified nearly every year since the mid-1990s.

Clinical Presentation

After an average incubation period of one week, symptomatic infections typically manifest as watery diarrhea of varying severity. Other manifestations include complications of dysentery, further abdominal symptoms, and sometimes non-specific systemic symptoms (e.g. headache, low-grade fever). Untreated infections typically last for 10–12 weeks and may follow a relapsing course. The duration of symptoms and associated weight loss are greater in individuals with HIV or possibly other immunosuppressive conditions. Infections can be asymptomatic in disease-endemic regions.

Gut bacteria influence brain development

Researchers discover biomarkers that indicate early brain injury in extreme premature infants

Extremely premature infants are at a high risk for brain damage. Researchers have now found possible targets for the early treatment of such damage outside the brain: Bacteria in the gut of premature infants may play a key role. The research team found that the overgrowth of the gastrointestinal tract with the bacterium *Klebsiella* is associated with an increased presence of certain immune cells and the development of neurological damage in premature babies.

Complex interplay: the gut-immune-brain axis

The early development of the gut, the brain and the immune system are closely interrelated. Researchers refer to this as the gut-immune-brain axis. Bacteria in the gut cooperate with the immune system, which in turn monitors gut microbes and develops appropriate responses to them. In addition, the gut is in contact with the brain via the vagus nerve as well as via the immune system. "We investigated the role this axis plays in the brain development of extreme preterm infants," says the first author of the study, David Seki. "The microorganisms of the gut microbiome -- which is a vital collection of hundreds of species of bacteria, fungi, viruses and other microbes -- are in equilibrium in healthy people. However, especially in premature babies, whose immune system and microbiome have not been able to develop fully, shifts are quite likely to occur. These shifts may result in negative effects on the brain," explains the microbiologist and immunologist.

Patterns in the microbiome provide clues to brain damage

"In fact, we have been able to identify certain patterns in the microbiome and immune response that are clearly linked to the progression and severity of brain injury," adds David Berry, microbiologist and head of the research group at the Centre for Microbiology and Environmental Systems Science (CMESS) at the University of Vienna as well as Operational Director of the Joint Microbiome Facility of the Medical University of Vienna and University of Vienna. "Crucially, such patterns often show up prior to changes in the brain. This suggests a critical time window during which brain damage of extremely premature infants may be prevented from worsening or even avoided."

Comprehensive study of the development of extremely premature infants

Starting points for the development of appropriate therapies are provided by the biomarkers that the interdisciplinary team was able to identify. "Our data show that excessive growth of the

bacterium *Klebsiella* and the associated elevated T-cell levels can apparently exacerbate brain damage," explains Lukas Wisgrill, Neonatologist from the Division of Neonatology, Pediatric Intensive Care Medicine and Neuropediatrics at the Department of Pediatric and Adolescent Medicine at the Medical University of Vienna. "We were able to track down these patterns because, for a very specific group of newborns, for the first time we explored in detail how the gut microbiome, the immune system and the brain develop and how they interact in this process," he adds. The study monitored a total of 60 premature infants, born before 28 weeks gestation and weighing less than 1 kilogram, for several weeks or even months. Using state-of-the-art methods -- the team examined the microbiome using 16S rRNA gene sequencing, among other methods -- the researchers analysed blood and stool samples, brain wave recordings (e.g. aEEG) and MRI images of the infants' brains.

Research continues with two studies

The study, which is an inter-university clusterproject under the joint leadership by Angelika Berger (Medical University of Vienna) and David Berry (University of Vienna), is the starting point for a research project that will investigate the microbiome and its significance for the neurological development of prematurely born children even more thoroughly. In addition, the researchers will continue to follow the children of the initial study. "How the children's motoric and cognitive skills develop only becomes apparent over several years," explains Angelika Berger. "We aim to understand how this very early development of the gut-immune-brain axis plays out in the long term." The most important cooperation partners for the project are already on board: "The children's parents have supported us in the study with great interest and openness," says David Seki. "Ultimately, this is the only reason we were able to gain these important insights. We are very grateful for that."

Reference

1. David Seki, Margareta Mayer, Bela Hausmann, Petra Pjevac, Vito Giordano, Katharina Goeral, Lukas Unterasinger, Katrin Klebermaß-Schrehof, Kim De Paepe, Tom Van de Wiele, Andreas Spittler, Gregor Kasprian, Benedikt Warth, Angelika Berger, David Berry, Lukas Wisgrill. Aberrant gut-microbiota-immune-brain axis development in premature neonates with brain damage. *Cell Host & Microbe*, 2021; DOI: 10.1016/j.chom.2021.08.004

WHO GUIDELINES FOR SAFE SURGERY Contd...

Recommendations to achieve 10 basic and essential objectives in any surgery.

(1) To correct patient at the correct site.

- Before induction of anaesthesia, a member of the team should confirm that the patient is correctly identified, usually verbally with the patient or family member and with an identity bracelet or other appropriate means of physical identification. Identity should be confirmed from not just the name but also a second identifier (e.g. date of birth, address, hospital number).
- A team member should confirm that the patient has given informed consent for the procedure and should confirm the correct site and procedure with the patient.
- The surgeon performing the operation should mark the site of surgery in cases involving laterality or multiple structures or levels (e.g. a finger, toe, skin lesion, vertebra). Both the anaesthetist and the nurse should check the site to confirm that it has been marked by the surgeon performing the operation and reconcile the mark with the information in the patient's records. The mark should be unambiguous, clearly visible and usually made with a permanent marker so that it does not come off during site preparation. The type of mark can be determined locally (signing, initialling or placing an arrow at the site). A cross or 'X' should be avoided, however, as this has been misinterpreted to mean that the site is the one not to be operated on.
- As a final safety check, the operating team should collectively verify the correct patient, site and procedure during a 'time out' or pause immediately before skin incision. The surgeon should state out loud the patient's name, the operation to be performed, and the side and site of surgery. The nurse and anaesthetist should confirm that the information is correct.
- As a final safety check, the operating team should collectively verify the correct patient, site and procedure during a 'time out' or pause immediately before skin incision. The surgeon should state out loud the patient's name, the operation to be performed, and the side and site of surgery. The nurse and anaesthetist should confirm that the information is correct.

(2) To methods known to prevent harm from administration of anaesthetics, while protecting the patient from pain.

- The first and most important component of peri-anaesthetic care is the continuous presence of a vigilant, professionally trained anaesthesia provider. If an emergency requires the brief temporary absence of the primary anaesthetist, judgement must be exercised in comparing the threat of an emergency to the risk of the anaesthetized patient's condition and in selecting the clinician left responsible for anaesthesia during the temporary absence.
- Supplemental oxygen should be supplied for all patients undergoing general anaesthesia. Tissue oxygenation and perfusion should be monitored continuously using a pulse oximeter with a variable-pitch pulse tone loud enough to be heard throughout the operating room.
- The adequacy of the airways and of ventilation should be monitored continuously by observation and auscultation. Whenever mechanical ventilation is employed, a disconnect alarm should be used.
- Circulation should be monitored continuously by auscultation or palpation of the heart beat or by a display of the heart rate on a cardiac monitor or pulse oximeter.
- Arterial blood pressure should be determined at least every 5 minutes and more frequently if indicated by clinical circumstances.
- A means of measuring body temperature should be available and used at frequent intervals where clinically indicated (e.g. prolonged or complex anaesthesia, children).
- The depth of anaesthesia (degree of unconsciousness) should be assessed regularly by clinical observation.

(3) To recognize and effectively prepare for life threatening loss of airway or respiratory function.

- All patients should undergo an objective evaluation of their airway before induction of anaesthesia, even when intubation is not anticipated, in order to identify potential difficulties in airway management.
- The anaesthetist should have a planned strategy for managing the airways and be prepared to execute it, even if airway loss is not anticipated.

- When the anaesthetist suspects a difficult airway, assistance during induction should be immediately available and a backup plan for airway management should be clearly identified.
 - When a patient is known to have a difficult airway, alternative methods of anaesthesia should be considered, including regional anaesthesia or awake intubation under local anaesthetic.
 - All anaesthetists should maintain their airway management skills and be familiar with and proficient in the multiple strategies for dealing with difficult airways.
 - After intubation, the anaesthetist should always confirm endotracheal placement by listening for breath sounds as well as gastric ventilation and monitoring the patient's oxygenation with a pulse oximeter.
 - Patients undergoing elective surgery should be fasting prior to anaesthesia. Those at risk of aspiration should be pre-treated to reduce gastric secretion and increase pH.
- (4) To recognize and effectively prepare for risk of high blood loss.**
- Before inducing anaesthesia, the anaesthetist should consider the possibility of large-volume blood loss, and, if it is a significant risk, should prepare appropriately. If the risk is unknown, the anaesthetist should communicate with the surgeon regarding its potential occurrence.
 - Before skin incision, the team should discuss the risk for large volume blood loss and, if it is significant, ensure that appropriate intravenous access is established.
- (5) To avoid inducing an allergic or adverse drug reaction for which the patient is known to be at significant risk.**
- Anaesthetists should fully understand the pharmacology of the medication they prescribe and administer, including its toxicity.
 - Every patient to whom any drug is administered must first be identified clearly and explicitly by the person administering the drug.
 - A complete drug history, including information on allergies and other hypersensitivity reactions, should be obtained before administration of any medication.
 - Medications should be appropriately labelled, confirmed and rechecked before administration, particularly if they are drawn into syringes.
 - Before any drug is administered on behalf of another health provider, explicit communication should take place to ensure that the two have a shared understanding of the indications, potential contraindications and any other relevant information.
- (6) To consistently use methods known to minimize the risk for surgical site infection.**
- Prophylactic antibiotics should be used routinely in all clean-contaminated surgical cases and considered for use in any clean surgical case. When antibiotics are given prophylactically to prevent infection, they should be administered within 1 hour of incision at a dose and with an antimicrobial spectrum that is effective against the pathogens likely to contaminate the procedure. Before skin incision, the team should confirm that prophylactic antibiotics were given within the past 60 minutes. (When vancomycin is used, infusion should be completed within 1 hour of skin incision.)
 - Every facility should have a routine sterilization process that includes means for verifying the sterility of all surgical instruments, devices and materials. Indicators should be used to determine sterility and checked before equipment is introduced onto the sterile field. Before induction of anaesthesia, the nurse or other person responsible for preparing the surgical trays should confirm the sterility of the instruments by evaluating the sterility indicators and should communicate any problems to the surgeon and anaesthetist.
 - Redosing with prophylactic antibiotics should be considered if the surgical procedure lasts more than 4 hours or if there is evidence of excessive intraoperative bleeding. (When vancomycin is used as the prophylactic agent, there is no need for redosing in operations lasting less than 10 hours.)
 - Antibiotics used for prophylaxis should be discontinued within 24 hours of the procedure.
 - Hair should not be removed unless it will interfere with the operation. If hair is removed, it should be clipped less than 2 hours before the operation. Shaving is not recommended as it increases the risk for surgical site infection.
 - Surgical patients should receive oxygen throughout the perioperative period according to individual requirements.
 - Measures to maintain core normothermia should be taken throughout the perioperative period.
 - The skin of all surgical patients should be prepared with an appropriate antiseptic agent before surgery.

The antimicrobial agent should be selected on the basis of its ability to decrease the microbial count of the skin rapidly and its persistent efficacy throughout the operation.

- Surgical hand antisepsis should be assured with an antimicrobial soap. The hands and forearms should be scrubbed for 2–5 minutes. If the hands are physically clean, an alcohol-based hand antiseptic agent can be used for antisepsis.
- The operating team should cover their hair and wear sterile gowns and sterile gloves during the operation.

(7) To prevent inadvertent retention of instruments and sponges in surgical wounds.

- A full count of sponges, needles, sharps, instruments and miscellaneous items (any other item used during the procedure that is at risk of being left within a body cavity) should be performed when the peritoneal, retroperitoneal, pelvic or thoracic cavity is entered.
- The surgeon should perform a methodical wound exploration before closure of any anatomical cavity or the surgical site.
- Counts should be done for any procedure in which sponges, sharps, miscellaneous items or instruments could be retained in the patient. These counts must be performed at least at the beginning and end of every eligible case.
- Counts should be recorded, with the names and positions of the personnel performing the counts and a clear statement of whether the final tally was correct. The results of this tally should be clearly communicated to the surgeon.

(8) To secure and accurately identify all surgical specimens

- The team should confirm that all surgical specimens are correctly labelled with the identity of the patient, the specimen name and location (site and side) from which the specimen was obtained, by having one team member read the specimen label aloud and another verbally confirming agreement.

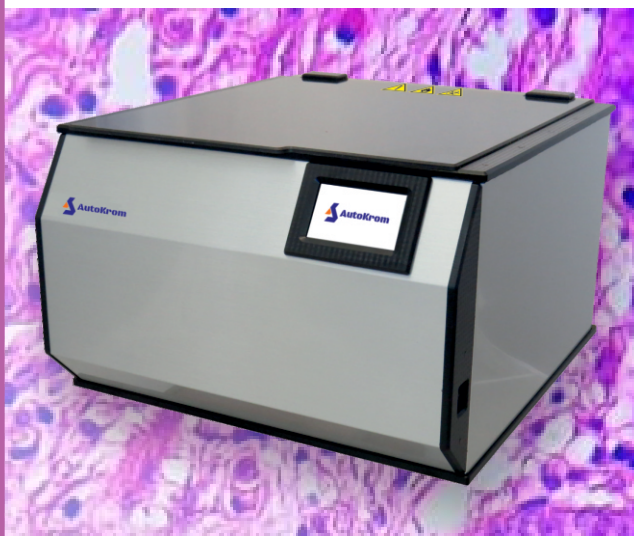
(9) To effectively communicate and exchange critical information for the safe conduct of the operation.

- Before skin incision, the surgeon should ensure that team members, in particular nurses, anaesthetists, and surgical assistants are aware of the critical steps of the procedure to be performed, the risk for heavy blood loss, any special equipment needed (such as instruments, implants,

intraoperative imaging, frozen section pathology) and any likely deviation from routine practice. The nurse(s) should inform the team members about any critical safety concerns and the lack of availability or preparation of any special equipment. The anaesthetist should inform the team about any critical safety concerns, in particular any difficulty in preparing for resuscitation after heavy blood loss or patient comorbidities that add risk to the anaesthesia.

- In cases of bilaterality, multiple body parts (e.g. fingers or toes) and multiple levels (e.g. spine) or when intraoperative decisions on the extent of surgical resection are to be made in conjunction with radiographic imaging, the team should confirm that the necessary imaging is available and displayed in the operating room.
- Before the patient leaves the room, the surgeon should inform team members of any alterations that were made to the procedure performed, any problems that may occur in the postoperative period and essential postoperative plans (which might include antibiotics, venous thromboembolism prophylaxis, oral intake or drain and wound care). The anaesthetist should summarize the clinical condition of the patient during the operation and any other instructions needed to ensure a safe recovery. The nurse should notify the team of any additional concerns recognized during the operation or for recovery. An accurate, complete, signed surgical record should be maintained. All patient records should be:
 - clear: the patient clearly identified by his or her name and hospital number on each page, written legibly or typed and each entry signed, dated and timed;
 - objective: opinions should be based on recorded facts;
 - contemporary: notes should be written as soon as possible after an event;
 - tamper-proof: attempts to amend records should be immediately apparent; if computerized systems are used, they should record the date and author of any notes and track any amendments;
 - original: records should not be altered or amended once an entry is complete. If a mistake is noticed, amendments or corrections may be added and clearly identified as such. If a change is made to the record, it should be signed and dated, and a note should explain why the change was made.

- Information recorded by the surgeon in the operation note should include, at a minimum, the name of the main procedure performed and any secondary procedures, the names of any assistants, the details of the procedure and the intraoperative blood loss. The information recorded by the anaesthetist should include, at a minimum, intraoperative vital sign parameters recorded at regular intervals, medications and fluids administered intraoperatively and any intraoperative events or periods of patient instability. The information recorded by the nursing team should include, at a minimum, sponge, needle, sharps and instrument counts, the names and positions of the personnel performing the counts, instruments and sponges specifically left inside the patient, any action taken in the event of a count discrepancy, and, if no count was performed, the reasons for not conducting a count. The complete operation record should therefore include the names of all team members involved.
- (10) To Hospitals and public health systems will establish routine surveillance of surgical capacity, volume and results.**
- For surgical surveillance at the national level, the following data should be collected systematically by WHO Member States:
 - number of operating rooms,
 - number of surgical procedures performed in an operating room,
 - number of trained surgeons and number of trained anaesthetists,
 - day-of-surgery mortality rate and
 - postoperative in-hospital mortality rate.
 - For surgical surveillance at hospital and practitioner levels, the following data should be collected systemically by facilities and clinicians:
 - Day-of-surgery mortality rate,
 - Postoperative in-hospital mortality rate.



BENEFITS

- ~ Enhances contrast in microscopic images.
- ~ Highlights structural details of biological tissues for true differentiation and distinction.
- ~ Enhances cytoplasmic clarity and transparency.
- ~ Enhanced ease and speed of preparation.
- ~ No compromise on reproducibility.

AUTOMATED SLIDE STAINER

STAINING MADE FASTER & EASIER

Intensify Microscopy with Clarity...!

Published by D.G. Tripathi, Edited by Akshaya Tendulkar for and on behalf of Tulip Diagnostics (P) Ltd., Tulip House, Dr. Antonio Do Rego Bagh, Alto Santacruz, Bambolim Complex, Post Office Goa-403 202, India. Fax: 0832 2458544, Website: www.tulipgroup.com.

AEROBOO
AUTOMATIC FLEXIBLE ENDOSCOPE REPROCESSOR

The new concept is designed to:

- Optimize your workflow
- Provide you with hygienic results at the highest level
- Ensure the safety of your patients and employees
- Protect sensitive, high-tech endoscope instruments

“Effective Reprocessing is Key to Patient Safety in Endoscopy”

-World Gastroenterology Organization

BioShields
Applying Science In Disinfection

Highlights of the coming issue

JOURNAL OF HYGIENE SCIENCES
Committed to the advancement of Clinical & Industrial Disinfection & Microbiology

Contents

- Mini Review**
The Microbiology of milk and milk products (issue II).
- Current Trends**
The Quat Advantage: Quaternary Ammonium Chloride and Its Advantages in Healthcare Facilities.
- In Profile**
Robert Koch.
- Bug of the Month**
Burkholderia mallei.
- Did You Know**
Your Stomach Acid Can Dissolve a Razor Blade: True or False?
- Best Practices**
Minimize Risk of Surgical Site Infection.

Microexpress BioShields 2021 www.tulipgroup.com

Microexpress
Quick Reliable Microbiology

BioShields
Applying Science In Disinfection