JOURNAL OF
HYGIENE SCIENCES
Committed to the advancement of Clinical & Industrial Disinfection & Microbiology
VOLUME - V ISSUE - V SEP-OCT 2012

Editorial

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This issue of the Journal brings forth not just topics to ponder over, but essentials to read & understand……..

Well, Food as we all know has to be hygienic, whether it is a wholesome meal or simply a desert. Food contamination refers to foods that are spoiled because they either contain microorganisms, such as bacteria or parasites, or toxic substances that make them unfit for consumption. Food contamination can be microbial or environmental, with the former being more common. The term Food-borne diseases, including food borne intoxications and food-borne infections, covers illnesses acquired through the consumption of such contaminated food, and are also frequently referred to as food poisoning. So let's review Food borne diseases & related issues in our Mini review section.

As we all think the health-care environment is rarely implicated in disease transmission, but it does get affected with infections associated with air, water, or other elements of the environment. In our Current Trends section let's have look at what control measures need to be practiced for environmental infection control in health care facilities.

Our In Profile segment covers Francis Crick, the co-discoverer of the structure of the DNA molecule in 1953. He was awarded the Nobel Prize for Physiology or Medicine in 1962 "for their discoveries concerning the molecular structure of nucleic acids and its significance for information transfer in living material". Crick also used the term "central dogma" to summarize an idea that implies that genetic information flow between macromolecules would be essentially one-way i.e. DNA-RNA-Proteins. He received many awards and honours, including the Lasker Award of Basic Medical Research Award (1960) the Royal and Copley medals of the Royal Society (1972 and 1975).

Clostridium tetani is the Bug of the month for this time. *Clostridium tetani* is a rod-shaped, anaerobic bacterium, Gram-positive, and its appearance on a gram stain resembles tennis rackets or drumsticks. *C. tetani* is found as spores in soil or in the gastrointestinal tract of animals and produces a potent biological toxin, **tetanospasmin**, and is the causative agent of tetanus, a disease characterized by painful muscular spasms that can lead to respiratory failure and, in up to 40% of cases, death. In current ongoing research it was discovered that *C. tetani* and other members of the *Clostridium* family could be used for anti-cancer drug delivery process.

Our Did You Know section tells you some cool facts about Mycoplasma. What are they? How Mycoplasma contamination can be detected & prevented? All this questions are answered when you have a quick look at this section.

In general laboratories commonly receive different samples like blood samples or cerebrospinal fluid from patients with pneumonia, meningitis, or unexplained febrile illness. Laboratories may also receive urine, joint fluid, pleural fluid, or other sterile site specimens from these patients. This section of Best Practices provides methods for the isolation and presumptive identification of agents from these normally sterile sites.

"Laughter is the music of the soul" so ease your mind with the humour in our Relax Mood section. Also give a try to the interesting quiz questions in the other part of this section.

Our JHS team thanks all our readers for their continuous support & contribution. Feedback & suggestions are always welcomed.

Food Borne Diseases

INTRODUCTION:

The term Food-borne diseases, including food borne intoxications and food-borne infections, covers illnesses acquired through consumption of contaminated food, and are also frequently referred to as food poisoning.

Worldwide, food-borne diseases are a major health burden leading to high morbidity and mortality. The global burden of infectious diarrhea involves 3-5 billion cases and nearly 1.8 million deaths annually, mainly in young children, caused by contaminated food and water.

According to the CDC (Center for Disease Control), an estimated 76 million cases of food-borne disease are reported annually in the United States with approximately 5000 deaths.

HISTORY:

In India, food poisoning affecting 78 personnel was reported in 1998 by the armed forces at high altitude, wherein *Salmonella enteritidis* was identified as the etiological agent and frozen fowl was the implicated food source for the outbreak. A food poisoning outbreak due to *Salmonella paratyphi A* that affected 33 people, due to vegetarian food was reported from Yavatmal (Maharashtra) in 1995. Two separate food poisoning outbreaks due to *Salmonella weltevreden* and *Salmonella wein* affecting 34 and 10 people respectively, due to non vegetarian food (chicken and fish) were reported from Mangalore in 2008-09.

In 1995, outbreaks due to *Clostridium perfringens* (in which mutton and peas were implicated as the food source) and *Bacillus cereus* (due to a bakery product) were reported, and in 2002, watermelon was implicated as the food source in another outbreak affecting 6 members of a family.

A food poisoning outbreak due to *Yersinia enterocolitica* was reported in 1997 from Tamil Nadu affecting 25 people, in which buttermilk was incriminated as the food source. An outbreak of food-borne botulism due to *Clostridium butyricum* affecting 34 students from a residential school in Gujarat was reported in 1996, and the food sample found to be contaminated was sevu (crisp made from gram flour). An outbreak of *Staphylococcal aureus* food poisoning due to contaminated "bhalla" (a snack made up of potato balls fried in vegetable oil) affected more than 100 children and adults in Madhya Pradesh in 2007.

CHEMICAL FOOD POISONING

Toxic compounds like lectins and glycoalkaloids are naturally present in some vegetables like potatoes and legumes. Many marine toxins produced by dinoflagellates occurring secondarily in molluscs and mussels can lead to food poisoning in humans. Other toxic compounds like pesticides, heavy metals and toxins of fungal or bacterial origin could also contaminate food during manufacture, storage or transportation.

India's production of pesticides was 85,000 metric tonnes in 2004, and rampant use of these chemicals has lead to several short-term and long-term effects. The first report of pesticide poisoning in India was from Kerala in 1958, where over 100 people died after consuming food made from wheat flour contaminated with parathion. In 1997, a food-borne outbreak of organophosphate (malathion) poisoning affected 60 men (and was fatal for one) who ate a communal lunch prepared from food stored in open jute bags which was contaminated with the pesticide sprayed in the kitchen that morning. It is estimated that 51% of food commodities are contaminated with pesticide residues in India.

CLASSIFICATION OF FOOD-BORNE ILLNESSES

- **Food-borne infections** - caused by consuming foods or liquids contaminated with bacteria, viruses, or parasites. These pathogens cause infection by:
  1) Invading and multiplying in the lining of the intestines and/or other tissues
  2) Invading and multiplying in the intestinal tract and releasing a toxin (bacteria only)

- **Food-borne intoxications -** caused by consuming foods or beverages already contaminated with a toxin. Sources of toxins are as follows:
  1) Certain bacteria (pre-formed toxins)
  2) Poisonous chemicals
  3) Natural toxins found in animals, plants

INFECTIONS VERSUS INTOXICATIONS

	INFECTIONS	INTOXICATIONS
Cause	Bacteria / Viruses / Parasites	Toxin
Mechanism	Invade and / or multiply within the lining of the intestines	No invasion or multiplication
Incubation period	Hours to days	Minutes to hours
Symptoms	DiarrhoeaNausea / Vomiting Abdominal cramps± Fever	Vomiting, Nausea, Diarrhoea Double vision Weakness Respiratory failure Numbness Sensory and motor dysfunction
Transmission	Can spread from person-to-person via the faeco-oral route	Not communicable
Factors related to food contamination	Inadequate cooking Croos-contaminatio Poor personal hygiene Bare hand contact	Inadequate cooking Improper holding temperatures

Pathogenesis:

Food-borne illness is typically caused by microorganisms or their toxins, and most often manifests with gastro-intestinal symptoms, which can vary in severity and duration. In addition to food-borne pathogens (bacteria, viruses and parasites), food-borne disease may also be caused by contaminants like heavy metals, chemicals, pesticides and toxic substances present naturally in food like toxic mushrooms, plants, fish or shellfish.

The food-borne diseases due to infectious causes form the

majority of cases, and are largely dependent on the inoculum size or the infective dose of the pathogen. This may be as small as 10 to 100 bacteria or cysts for Shigella, Entero- Haemorrhagic *E. coli* (EHEC), Giardia lamblia and Entamoeba histolytica, requiring minor lapses in hygiene for the faeco-oral transmission. The infective dose for Vibrio cholerae on the other hand is usually 105 – 108, and may be variable for Salmonella sp.

FOOD-BORNE TRANSMISSION OF PATHOGENS AND TOXINS:

Food may become contaminated during production and processing or during food preparation and handling.

1) **Food production and processing**
   Foods, such as fruits and vegetables, may be contaminated if washed or irrigated with water that is contaminated with pathogens from animal or human faeces. Animals naturally harbour many food-borne bacteria in their intestines that can cause illness in humans, but often do not cause illness in the animals. During slaughter, meat and poultry carcasses can become contaminated if they are exposed to small amounts of intestinal contents.

2) **Food preparation and handling**

   Infected individuals - Most food-borne pathogens are shed in the faeces of infected persons and these pathogens may be transferred to others through food via the faecal-oral route. Bacteria present in infected lesions and normal nasal flora may also be transmitted from an infected food-handler to ready-to-eat foods.

   Cross-contamination – Pathogens naturally present in one food may be transferred to other foods during food preparation if same cooking equipment and utensils are used without washing and disinfecting in between, especially in case of ready-to-eat foods.

   Inadequate cooking temperature – With insufficient cooking bacteria can multiply and produce toxins within the food. Many With insufficient cooking bacteria can multiply and produce toxins within the food. Many bacterial toxins are heat stable and may not be destroyed by cooking.

3) **Improper storage**
   Food held or stored at warm (10-50ºC) temperature allows multiplication of pathogens and is an important cause of food borne outbreaks.

Mechanism	Location	Illness	Stool M/E	Examples
Non-inflammatory (enterotoxin)	Proximal small intestine	Watery diarrhoea	No faecal leukocytes	*Vibrio cholerae*, ETEC, EAggEC, *Cl. perfringens*, *Bacillus cereus*, *Staph aureus*, rotavirus, norovirus, enteric adenoviruses, *Giardia lamblia*, Cryptosporidium, Cyclospora, Microsporidia
Inflammatory (invasion / cytotoxin)	Colon / distal small intestine	Dysentery / inflammatory diarrhoea	PMN faecal leukocytes	Shigella, Salmonella, *C. jejuni*, EHEC, *enterocolitica*, *Vibrio parahaemolyticus*, *Cl. difficile*, *E. histolytica*
Penetrating	Distal small intestine	Enteric fever	Mono-nuclear faecal leukocytes	*Salmonella typhi*, *Y. enterocolitica*, *Campylobacter fetus*

BACTERIAL FOOD POISONING:

I.P.	Cause	Symptoms	Common foods
1-6 hours	*Staph aureus*	Nausea, Vomiting, Diarrhoea	Ham, poultry, potato / egg salad, mayonnaise, cream pastries
	Bacillus cereus	Nausea, Vomiting, Diarrhoea	Fried rice
8-16 hours	*Cl. perfringens*	Abdominal cramps, diarrhoea (vomiting rare)	Beef, poultry, legumes, gravies
	B. cereus		Meats, vegetables, dried beans, cereals
>16 hours	*Vibrio cholerae*	Watery diarrhoea	Shell-fish
	ETEC	Watery diarrhoea	Salad, cheese, meats, water
	EHEC	Bloody diarrhoea	Beef, salami, raw milk / vegetables, apple juice
	Salmonella sp	Inflammatory diarrhoea	Beef, poultry, eggs, diary products
	Campylobacter jejuni	Inflammatory diarrhoea	Poultry, raw milk
	Shigella sp	Dysentery	Potato / egg salad, lettuce, raw eggs
	V. parahaemolyticus	Dysentery	Molluscs, crustaceans

SOME IMPORTANT FOOD-BORNE PATHOGENS, TOXINS AND CHEMICALS PATHOGENS:

Bacteria		
Aeromonas hydrophila	Entero Toxigenic *E. coli* (ETEC)	Salmonella (non Typhi) sps
Bacillus cereus	Entero Pathogenic *E. coli* (EPEC)	Shigella spp
Brucella spp	Entero-Haemorrhagic *E. coli* (EHEC)	*Staphylococcus aureus*
Campylobacter spp	Entero-Invasive *E. coli* (EIEC)	*Vibrio cholerae* O1 and O139
Clostridium botulinum	*Listeria monocytogenes*	*Vibrio parahaemolyticus*
Clostridium perfringens	*Salmonella typhi*	*Vibrio vulnificus*
Escherichia coli spp	*S. paratyphi*	*Yersinia enterocolitica*
Viruses		
Hepatitis A virus	Norovirus	Rotavirus
Hepatitis E virus	Poliovirus	
Protozoa		
Cryptosporidium spp	*Entamoeba histolytica*	*Toxoplasma gondii*
Cyclospora cayetanensis	*Giardia lamblia*	
Trematodes		
Clonorchis sinensis	*Fasciolopsis buski*	*Opisthorchis viverrin*
Fasciola hepatica	*Opisthorchis felineus*	*Paragonimus westermani*
Cestodes		
Diphyllobothrium spp	Echinococcus spp	*Hymenolepis nana*
Taenia solium / saginata		
Nematodes		
Anisakis spp	*Ascaris lumbricoides*	*Trichinella spiralis*
Trichuris trichiura		
Toxins		
Marine biotoxins	Tetrodotoxin (pufferfish)	Pyrrolizidine alka
Ciguatera poisoning bean poisoning)	Mushroom toxins	Phytohaemagglutinin (red kidney
Shellfish toxins (paralytic, neurotoxic, diarrhoeal, amnesic)	Mycotoxins (e.g. aflatoxins)	Grayanotoxin (honey intoxication)
Scombroid poisoning/ histamine	Plant toxicants	
Chemicals		
Pesticides (organophos-phates, antimony	Radionuclides	Nitrites (food preservatives)
Toxic metals (cadmium, copper, lead, mercury, tin)	Fluoride	Sodium hydroxide
Polychlorinated biphenyls	Zinc	Monosodium glutamate

LABORATORY DIAGNOSIS OF FOODBORNE ILLNESSES

The main objectives of laboratory analysis during food-borne outbreak investigations are to

(1) Confirm the clinical diagnosis by isolation of causative agent from human specimens

(2) Ensure proper identification of the disease, and

(3) Determine if the same causative agent is present in implicated food sources, using relevant epidemiological markers like biotyping, serotyping, antimicrobial susceptibility profile, phage typing, plasmid profile, pulsed field gel electrophoresis, PCR, etc.

Most food-borne infections are diagnosed through the identification of the pathogen in stool collected from infected persons. Vomitus has also been used to detect certain organisms and confirm the aetiology. Blood samples are recommended for cases with systemic involvement.

Stool Specimens

Proper collection and transport of stool specimens requires the appropriate transport medium (modified Cary-Blair medium), and encouraging ill persons to submit a stool specimen.

Vomitus / gastric aspirate can also be tested for organisms and toxins, and should be collected as soon as possible after onset of illness. Instruct the patient to vomit directly into a sterile specimen container, such as a screw-capped bottle (or a urine specimen container). If this is not possible, ask the patient to vomit in a clean container, bowl or plastic bag and transfer the vomitus to the screw-capped container with a clean spoon. Place the cap securely on the container and seal the lid with tape.

Food specimens

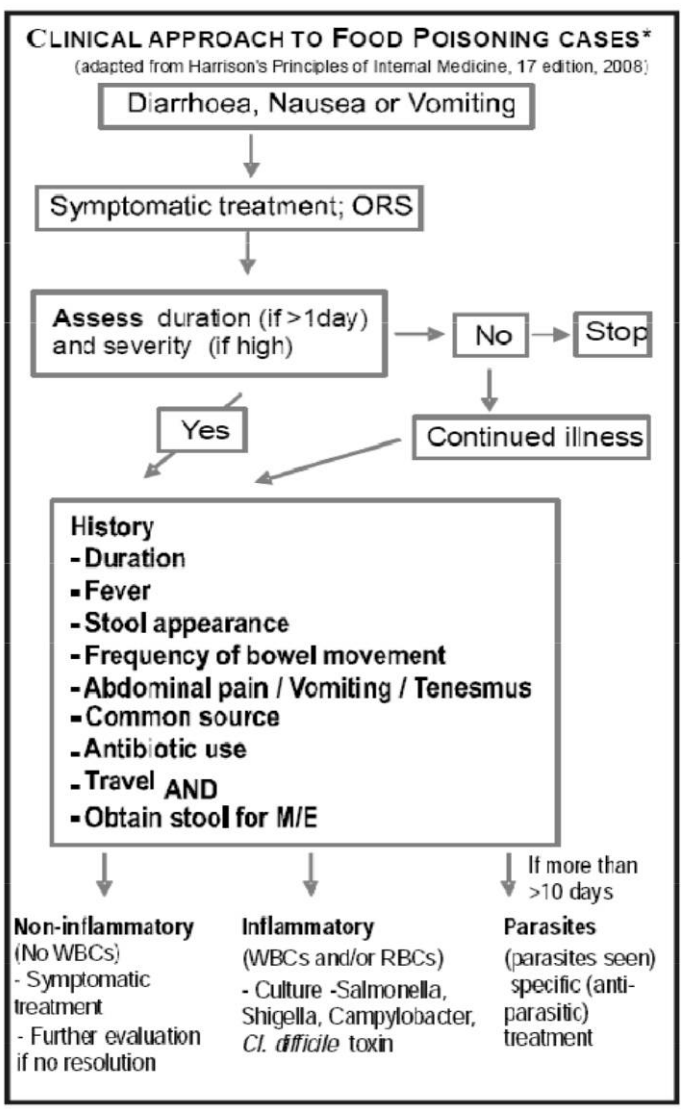
Microbiological analysis of food supports the epidemiological investigation of a food-borne disease outbreak. The purpose of testing is to isolate and identify pathogenic micro-organisms in food samples, which have been implicated in the outbreak. Samples collected as part of the investigation should be treated as official samples and should be collected in a manner that reflects the food as it was prepared, served, or used in preparation of the suspected meal.

Food samples must be collected using aseptic techniques and appropriate containers. Samples must be refrigerated during storage and transport and must arrive at the food microbiology laboratory within three days of collection. Samples collected frozen should be stored and transported frozen on dry ice.

- Whenever possible, food samples should be submitted in the original container as contamination of a sample may occur during manipulation.
- Samples that cannot be shipped in their original container should be collected aseptically using sterile and leak-proof collection containers.
- Representative sample of the solid food item should be taken from the geometric centre as well as several other locations in the food item.
- Stir or shake the liquid food item and pour or ladle the sample into the sterile leak proof container.
- Collection of an adequate amount of the food sample (minimum of 100 grams).
- Containers should be filled not more than 75% of their capacity and sealed.
- Food samples should be placed in vaccine carrier with ice packs.
- Sample labelling should include the following: name and type of product, brand of product, product manufacturer and code or lot number, collected by, date, time, and place of collection, and establishment's name.

Labelled specimen containers should be placed in a zip-lock bag and sealed. Cold chain should be maintained during transport by sending sample in vaccine carrier with ice packs, avoid freezing. Investigation forms should be filled for each specimen obtained along with relevant clinical details.

The collection of prepared food samples for outbreak investigation does not have medico legal implications and do not fall under the Prevention of Food Adulteration Act, 1954.



TREATMENT:

Initial treatment of patients with food poisoning should focus on assessment and reversal of dehydration, either through oral rehydration therapy (ORT) especially in children, or through IV fluids in seriously dehydrated cases.

Specific treatment in case of pesticide poisoning with chelating agents may be done based on epidemiological and clinical features, under medical supervision.

The earlier standard Oral Rehydration Salts (ORS) provided a solution containing 90 mEq/l of sodium with a total osmolarity of 311 mOsm/l. In 2003, the "improved" ORS having lower osmolarity was formulated by reducing the solution's glucose and salt concentrations. Because of the improved effectiveness of reduced osmolarity ORS solution, especially for children with acute, non-cholera diarrhoea, WHO and UNICEF now recommend that countries use and manufacture the following formulation in place of the previously recommended ORS solution.

REDUCED OSMOLARITY ORS FORMULATION

Preparation of ORS: Mix the following in one liter of clean drinking water:

Formula	grams/ litre
Sodium chloride	2.6
Glucose, anhydrous	13.5
Potassium chloride	1.5
Trisodium citrate, dihydrate	2.9

Constituents	mmol/ litre	Acceptable Range
Sodium	75	60-90
Chloride	65	50-80
Glucose, anhydrous	75	= Na but ≤111 mmol/l
Potassium	20	15-25
Citrate	10	8-12
Total Osmolarity	245	200-310 mmol/l

PREVENTION:

Hazard Analysis and Critical Control Point (HACCP) is a systematic preventive approach to food safety that addresses physical, chemical and biological hazards as a means of prevention rather than finished product inspection. HACCP is used in the food industry to identify potential food safety hazards, so that key actions can be taken at these Critical Control Points (CCPs). The system is used in the food industry at all stages of food production and preparation processes including packaging, distribution, etc.

HACCP is an effective approach to food safety and protecting public health. Apart from food contamination, transmission of infection occurs by direct contact, favoured by the habits and customs of people, improper storage and handling of cooked food is equally responsible for food-borne illnesses, as during storage, especially at ambient temperatures (28- 38 degree C) there is higher risk of multiplication of pathogenic organisms.

Food safety education is a critical pre-requisite to prevent food-borne outbreaks by education of food-handlers and the community about proper practices in cooking and storage of food, and personal hygiene. Hand washing is one of the key interventions, not just by food handlers, but also by the community at large. Environmental measures include discouraging sewage farming for growing vegetables and fruits.

PRINCIPLES OF HAZARD ANALYSIS AND CRITICAL CONTROL POINT (HACCP)

1. **Analyze hazards -** Potential hazards associated with a food and measures to control those hazards (biological, e.g. a microbe; chemical, e.g. a toxin; or physical, e.g. ground glass or metal fragments) are identified.

2. **Identify critical control points -** These are points in a food's production - from its raw state through processing and shipping to consumption by the consumer - at which the potential hazard can be controlled or eliminated. Examples are cooking, cooling, packaging, and metal detection.

3. **Establish preventive measures with critical limits for each control point** - For a cooked food, for example, this might include setting the minimum cooking temperature and time required to ensure the elimination of any harmful microbes.

4. **Establish procedures to monitor the critical control points -** Such procedures include determining how and who should monitor the cooking time and temperature.

5. **Establish corrective actions when monitoring shows that a critical limit has not been met** - For example, reprocessing or disposing of food if the minimum cooking temperature is not met.

6. **Establish procedures to verify that the system is working properly** - For example, testing time-and-temperature recording devices to verify that a cooking unit is working properly.

7. **Establish effective record keeping for documentation -** This would include records of hazards and their control methods, monitoring of safety requirements and action taken to correct potential problems.

REFERENCES:

1. CD Alert/newsletter of the National Centre for Disease Control (formerly known as NICD), Directorate General of Health Services.
2. www.niaid.nih.gov
3. www.cdc.gov
4. www.who.int/foodsafety/foodbornediseases

Environment Services in Healthcare Facilities

Introduction:

The health-care facility environment is rarely implicated in disease transmission, except among patients who are immune compromised. Nonetheless, inadvertent exposures to environmental pathogens (e.g., *Aspergillus* spp. and *Legionella* spp.) or airborne pathogens (e.g., *Mycobacterium tuberculosis* and varicella-zoster virus) can result in adverse patient outcomes and cause illness among health-care workers. Environmental infection-control strategies and engineering controls can effectively prevent these infections. The incidence of health-care-associated infections and pseudo-outbreaks can be minimized by 1) appropriate use of cleaners and disinfectants; 2) appropriate maintenance of medical equipment; 3) adherence to water-quality standards for hemodialysis, and to ventilation standards for specialized care environments (e.g., airborne infection isolation rooms, protective environments, or operating rooms); and 4) prompt management of water intrusion into the facility.

- This article contains recommendations for environmental infection control in health-care facilities, describing control measures for preventing infections associated with air, water, or other elements of the environment. These recommendations represent the views of different divisions within CDC's National Center for Infectious Diseases and the Healthcare Infection Control Practices Advisory Committee (HICPAC), a 12-member group that advises CDC on concerns related to the surveillance, prevention, and control of health-care--associated infections, primarily in U.S. health-care facilities. In 1999, HICPAC's infection-control focus was expanded from acute-care hospitals to all venues where health care is provided (e.g., outpatient surgical centers, urgent care centers, clinics, outpatient dialysis centers, physicians' offices, and skilled nursing facilities). This is applicable to majority of health-care facilities in the United States. This report is intended for use primarily by infection-control practitioners, epidemiologists, employee health and safety personnel, engineers, facility managers, information systems professionals, administrators, environmental service professionals, and architects. Key recommendations include infection-control impact of ventilation system and water system performance;
- establishment of a multidisciplinary team to conduct infection-control risk assessment;
- use of dust-control procedures and barriers during construction, repair, renovation, or demolition;
- environmental infection-control measures for special areas with patients at high risk;
- use of airborne-particle sampling to monitor the effectiveness of air filtration and dust-control measures;
- procedures to prevent airborne contamination in operating rooms when infectious tuberculosis (TB) patients require surgery;
- guidance for recovering from water-system disruptions, water leaks, and natural disasters (e.g., flooding);
- infection-control concepts for equipment using water from main lines (e.g., water systems for hemodialysis, ice machines, hydrotherapy equipment, dental unit water lines, and automated endoscope reprocessors);
- environmental surface cleaning and disinfection strategies with respect to antibiotic-resistant microorganisms;
- use of animals in health care for activities and therapy;
- managing the presence of service animals in health-care facilities;
- infection-control strategies for when animals receive treatment in human health-care facilities;
- a call to reinstate the practice of inactivating amplified cultures and stocks of microorganisms onsite during medical waste treatment.

Recommendations ---Environmental Services

Rating Categories

Recommendations are rated according to the following categories:

Category IA. Strongly recommended for implementation and strongly supported by well-designed experimental, clinical, or epidemiologic studies.

Category IB. Strongly recommended for implementation and supported by certain experimental, clinical, or epidemiologic studies and a strong theoretic rationale.

Category IC. Required by state or federal regulation, or representing an established association standard. (Note: Abbreviations for governing agencies and regulatory citations are listed where appropriate. Recommendations from regulations adopted at state levels are also noted. Recommendations from AIA guidelines cite the appropriate sections of the standards.)

Category II. Suggested for implementation and supported by suggestive clinical or epidemiologic studies, or a theoretic rationale.

I. Cleaning and Disinfecting Strategies for Environmental Surfaces in Patient-Care Areas

A. Select effective disinfectants, and use them in accordance with the manufacturer's instructions. Category IC

B. Follow manufacturers' instructions for cleaning and maintaining noncritical medical equipment. Category II

C. In the absence of a manufacturer's cleaning instructions, follow certain procedures.
   1. Clean noncritical medical equipment surfaces with a cleaner/disinfectant. This may be followed by application of an effective disinfectant (depending on the nature of the surface and the degree of contamination), in accordance with germicide label instructions. Category II
   2. Do not use alcohol to disinfect large environmental surfaces. Category II
   3. Use barrier protective coverings as appropriate for noncritical surfaces that are 1) touched frequently with gloved hands during the delivery of patient care; 2) likely to become contaminated with blood or body substances; or 3) difficult to clean (e.g., computer keyboards). Category II

D. Keep housekeeping surfaces (e.g., floors, walls, tabletops) visibly clean on a regular basis and clean up spills promptly. Category II
   1. Use a one-step process and an effective hospital detergent/disinfectant designed for general housekeeping purposes in patient-care areas where 1) uncertainty exists as to the nature of the soil on the surfaces (e.g., blood or body fluid contamination versus routine dust or dirt); or 2) uncertainty exists regarding the presence of multidrug resistant organisms on such surfaces. Category II
   2. Detergent and water are adequate for cleaning surfaces in non patient-care areas (e.g., administrative offices). Category II
   3. Clean and disinfect high-touch surfaces (e.g., doorknobs,

bed rails, light switches, and surfaces in and around toilets in patients' rooms) on a more frequent schedule than minimal touch housekeeping surfaces. Category II

4. Clean walls, blinds, and window curtains in patient-care areas when they are visibly dusty or soiled. Category II

E. Follow proper procedures for effective uses of mops, cloths, and solutions. Category II
   1. Prepare cleaning solutions daily or as needed, and replace with fresh solution frequently according to facility policies and procedures. Category II
   2. Change the mop head at the beginning of each day and also as required by facility policy, or after cleaning up large spills of blood or other body substances. Category II
   3. Clean mops and cloths after use and allow drying before reuse; or use single-use, disposable mop heads and cloths. Category II

F. After the last surgical procedure of the day or night, wet vacuum or mop operating room floors with a single-use mop and an effective hospital disinfectant. Category IB

G. Do not use mats with tacky surfaces at the entrances to operating rooms or infection-control suites. Category IB

H. Use appropriate dusting methods for patient-care areas designated for immune compromised patients (e.g., HSCT patients). Category IB
   1. Wet-dust horizontal surfaces daily by moistening a cloth with a small amount of an effective hospital detergent/disinfectant. Category IB
   2. Avoid dusting methods that disperse dust (e.g., feather-dusting). Category IB

I. Keep vacuums in good repair and equip vacuums with HEPA filters for use areas with patients at risk. Category IB

J. Close the doors of immune compromised patients' rooms when vacuuming, waxing, or buffing corridor floors to minimize exposure to airborne dust. Category IB

K. When performing low- or intermediate-level disinfection of environmental surfaces in nurseries and neonatal units, avoid unnecessary exposure of neonates to disinfectant residues on these surfaces by using effective germicides in accordance with manufacturers' instructions and safety advisories. Category IB, IC
   1. Do not use phenolics or any other chemical germicide to disinfect bassinets or incubators during an infant's stay. Category IB
   2. Rinse disinfectant-treated surfaces, especially those treated with phenolics, with water Category IB

II. Cleaning Spills of Blood and Body Substances

A. Promptly clean and decontaminate spills of blood or other potentially infectious materials Category IB, IC

B. Follow proper procedures for site decontamination of spills of blood or blood-containing body fluids. Category IC
   1. Use protective gloves and other PPE appropriate for this task. Category IC
   2. If the spill contains large amounts of blood or body fluids, clean the visible matter with disposable absorbent material, and discard the used cleaning materials in appropriate, labeled containers. Category IC
   3. Swab the area with a cloth or paper towels moderately wetted with disinfectant, and allow the surface to dry. Category IC

C. Use effective biocides for use as hospital disinfectants and labeled tuberculocidal to decontaminate spills of blood and other body fluids. Category IC

D. Sodium hypochlorite product is preferred.
   1. Use a 1:100 dilution (500--615 ppm available chlorine) to decontaminate nonporous surfaces after cleaning a spill of either blood or body fluids in patient-care settings. Category IB
   2. If a spill involves large amounts of blood or body fluids, or if a blood or culture spill occurs in the laboratory, use a 1:10 dilution (5,000--6,150 ppm available chlorine) for the first application of germicide before cleaning. Category IB

III. Carpeting and Cloth Furnishings

A. Vacuum carpeting in public areas of health-care facilities and in general patient-care areas regularly with well-maintained equipment designed to minimize dust dispersion Category II

B. Periodically perform a thorough, deep cleaning of carpeting as determined by facility policy by using a method that minimizes the production of aerosols and leaves little or no residue. Category II

C. Avoid use of carpeting in high-traffic zones in patient-care areas or where spills are likely (e.g., burn therapy units, operating rooms, laboratories, or intensive care units). Category IB

D. Follow appropriate procedures for managing spills on carpeting.
   1. Spot-clean blood or body substance spills promptly. Category IC
   2. If a spill occurs on carpet tiles, replace any tiles contaminated by blood and body fluids or body substances. Category IC

E. Thoroughly dry the wet carpets to prevent the growth of fungi; replace carpeting that remains wet after 72 hours. Category IB

F. No recommendation is offered regarding the routine use of fungicidal or bactericidal treatments for carpeting in public areas of a health-care facility or in general patient-care areas.

G. Do not use carpeting in hallways and patient rooms in areas housing immune suppressed patients (e.g., PE areas). Category IB

H. Avoid using upholstered furniture and furnishings in high-risk patient-care areas and in areas with increased potential for body substance contamination (e.g., pediatrics units) Category II

I. No recommendation is offered regarding whether upholstered furniture and furnishings should be avoided in general patient-care areas. Unresolved issue
   1. Maintain upholstered furniture in good repair. Category II
   2. Maintain the surface integrity of the upholstery by repairing tears and holes. Category II
   3. If upholstered furniture in a patient's room requires cleaning to remove visible soil or body substance contamination, move that item to a maintenance area where it can be adequately cleaned with a process appropriate for the type of upholstery and nature of the soil. Category II

IV. Flowers and Plants in Patient-Care Areas

A. Flowers and potted plants need not be restricted from areas for immune competent patients. Category II

B. Designate care and maintenance of flowers and potted plants to staff not directly involved with patient care. Category II

C. If plant or flower care by patient-care staff is unavoidable, instruct the staff to wear gloves when handling plants and flowers and perform hand hygiene after glove removal. Category II

D. Do not allow fresh or dried flowers, or potted plants, in patient-care areas for immune suppressed patients. Category

II

V. Pest Control

A. Develop pest-control strategies, with emphasis on kitchens, cafeterias, laundries, central sterile supply areas, operating rooms, loading docks, construction activities, and other areas prone to infestations. Category II

B. Install screens on all windows that open to the outside; keep screens in good repair. Category IB

C. Contract for routine pest control service by a credentialed pest-control specialist who will tailor the application to the needs of a health-care facility. Category II

D. Place laboratory specimens (e.g., fixed sputum smears) in covered containers for overnight storage. Category II

VI. Special Pathogens

A. Use appropriate hand hygiene, PPE (e.g., gloves), and isolation precautions during cleaning and disinfecting procedures. Category IB

B. Use standard cleaning and disinfection protocols to control environmental contamination with antibiotic-resistant, gram-positive cocci (e.g., methicillin-resistant *Staphylococcus aureus*, vancomycin intermediate sensitive *Staphylococcus aureus*, or vancomycin-resistant *Enterococcus*. Category IB

1. Pay close attention to cleaning and disinfection of high-touch surfaces in patient-care areas (e.g., bed rails, carts, charts, bedside commodes, bed rails, doorknobs, or faucet handles). Category IB
2. Ensure compliance by housekeeping staff with cleaning and disinfection procedures Category IB
3. Use effective chemicals/biocides appropriate for the surface to be disinfected as specified by the manufacturer's instructions. Category IB, IC
4. When contact precautions are indicated for patient care, use disposable patient-care items (e.g., blood pressure cuffs) wherever possible to minimize cross-contamination with multiple-resistant microorganisms. Category IB
5. Follow these same surface-cleaning and disinfecting measures for managing the environment of VRSA patients. Category II

C. Environmental-surface culturing can be used to verify the efficacy of hospital policies and procedures before and after cleaning and disinfecting rooms that house patients with VRE. Category II

1. Obtain prior approval from infection-control staff and the clinical laboratory before performing environmental-surface culturing. Category II
2. Infection-control staff, with clinical laboratory staff consultation, must supervise all environmental culturing. Category II

D. Thoroughly clean and disinfect environmental and medical equipment surfaces on a regular basis by using effective disinfectants in accordance with manufacturers' instructions. Category IB, IC

E. Advise families, visitors, and patients regarding the importance of hand hygiene to minimize the spread of body substance contamination (e.g., respiratory secretions or fecal matter) to surfaces. Category II

F. Do not use high-level disinfectants like glutaraldehyde on environmental surfaces; such use is inconsistent with label instructions because of the toxicity of the chemicals. Category IC

G. Because no products are specific for inactivating *Clostridium difficile* spores, use hypochlorite-based products for disinfection of environmental surfaces in accordance with guidance from the scientific literature in those patient-care areas where surveillance and epidemiology indicate ongoing transmission of *C. difficile*. Category II

H. Apply odourless and colourless disinfectants to control environmental contamination with respiratory and enteric viruses in pediatric-care units and care areas for immune compromised patients. Category IC

I. Clean surfaces that have been contaminated with body substances; perform disinfection on cleaned surfaces with an effective disinfectant in accordance with the manufacturer's instructions. Category IC

J. Use disposable barrier coverings as appropriate to minimize surface contamination. Category II

K. Develop and maintain cleaning and disinfection procedures in patient-care areas to control environmental contamination with agents of Creutzfeldt-Jakob disease (CJD). Category II

1. In the absence of contamination with central nervous system tissue, extraordinary measures (e.g., use of 2N sodium hydroxide [NaOH] or applying full-strength sodium hypochlorite) are not needed for routine cleaning or terminal disinfection of a room housing a confirmed or suspected CJD patient. Category II
2. Do not use aldehydes to eradicate CJD, as they might fix CJD proteins on the instrument and environment surfaces.
3. After removing gross tissue from the surface, use either 1N NaOH or a sodium hypochlorite solution to decontaminate operating room or autopsy surfaces with central nervous system or cerebral spinal fluid contamination from a diagnosed or suspected CJD patient. Category II
   a. The contact time for the chemical used during this process should be 30 min--1 hour
   b. Blot up the chemical with absorbent material and rinse the treated surface thoroughly with water.
   c. Discard the used, absorbent material into appropriate waste containers.
3. Use disposable, impervious covers to minimize body substance contamination to autopsy tables and surfaces. Category II

L. Use standard procedures for containment, cleaning, and decontamination of blood spills on surfaces as previously described (Environmental Services: II) Category IC

1. Wear PPE appropriate for a surface decontamination and cleaning task. Category IC
2. Discard used PPE by using routine disposal procedures or decontaminate reusable PPE as appropriate. Category IC

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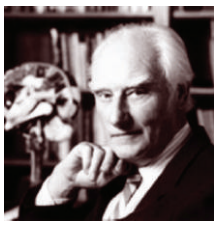

Francis Crick

Born: 8 June 1916, England
Nationality: British
Fields: Physics, Molecular biology
Known for: DNA structure, consciousness
Notable awards: Nobel Prize for Physiology or Medicine (1962)
Died: 28 July 2004(2004-07-28) (aged 88)

Francis Harry Compton Crick, (8 June 1916 – 28 July 2004) was an English molecular biologist, biophysicist, and neuroscientist, and most noted for being a co-discoverer of the structure of the DNA molecule in 1953 together with James D. Watson. He, Watson, and Maurice Wilkins were jointly awarded the 1962 Nobel Prize for Physiology or Medicine "for their discoveries concerning the molecular structure of nucleic acids and its significance for information transfer in living material".

Crick was an important theoretical molecular biologist and played a crucial role in research related to revealing the genetic code. He is widely known for use of the term "central dogma" to summarize an idea that genetic information flow in cells is essentially one-way, from DNA to RNA to protein.

Early life and education: Francis Harry Compton Crick was the first son of Harry Crick (1887–1948) and Annie Elizabeth Crick, née Wilkins, (1879–1955). He was born and raised in Weston Favell. He was educated at Northampton Grammar School and, after the age of 14, Mill Hill School in London (on scholarship), where he studied mathematics, physics, and chemistry. He shared the Walter Knox Prize for Chemistry on Foundation Day, Friday, 7 July 1933.

At the age of 21, Crick earned a B.Sc. degree in physics from University College London. Crick had failed to gain a place at a Cambridge college, probably through failing their requirement for Latin. Crick later became a PhD student and Honorary Fellow of Gonville and Caius College and mainly worked at the Cavendish Laboratory and the Medical Research Council (MRC) Laboratory of Molecular Biology in Cambridge. He was also an Honorary Fellow of Churchill College and of University College, London.

Crick began a Ph.D. research project on measuring viscosity of water at high temperatures in the laboratory of physicist Edward Neville da Costa Andrade at University College, London, but with the outbreak of World War II, Crick was deflected from a possible career in physics.

RESEARCH: Crick was interested in two fundamental unsolved problems of biology: **how molecules make the transition from the non-living to the living, and how the brain makes a conscious mind.** It was at this time of Crick's transition from physics to biology. It was clear in theory that covalent bonds in biological molecules could provide the structural stability needed to hold genetic information in cells. It only remained as an exercise of experimental biology to discover exactly which molecule was the genetic molecule. In Crick's view, Charles Darwin's theory of evolution by natural selection, George Mendel's genetics and knowledge of the molecular basis of genetics, when combined, revealed the secret of life.

It was clear that some macromolecule such as a protein was likely to be the genetic molecule. However, it was well known that proteins are structural and functional macromolecules, some of which carry out enzymatic reactions of cells. In the 1940s, some evidence had been found pointing to another macromolecule, DNA, the other major component of chromosomes, as a candidate genetic molecule.

However, other evidence was interpreted as suggesting that DNA was structurally uninteresting and possibly just a molecular scaffold for the apparently more interesting protein molecules. Crick was in the right place, in the right frame of mind, at the right time (1949), to join Max Perutz's project at Cambridge University, and he began to work on the X-ray crystallography of proteins. X-ray crystallography theoretically offered the opportunity to reveal the molecular structure of large molecules like proteins and DNA, but there were serious technical problems then preventing X-ray crystallography from being applicable to such large molecules.

Late in 1951, Crick started working with James D. Watson at Cavendish Laboratory at the University of Cambridge, England. Watson and Crick together developed a model for a helical structure of DNA, which they published in 1953. For this and subsequent work they were jointly awarded the Nobel Prize in Physiology or Medicine in 1962 with Maurice Wilkins.

Watson and Crick first made helical models with the phosphates at the center of the helices. Of great importance to the model building effort of Watson and Crick was Rosalind Franklin's understanding of basic chemistry, which indicated that the hydrophilic phosphate-containing backbones of the nucleotide chains of DNA should be positioned so as to interact with water molecules on the outside of the molecule while the hydrophobic bases should be packed into the core. During their model building, Crick and Watson learned that an antiparallel orientation of the two nucleotide chain backbones worked best to orient the base pairs in the centre of a double helix.

Another key to finding the correct structure of DNA was the so-called Chargaff ratios, experimentally determined ratios of the nucleotide subunits of DNA: the amount of guanine is equal to cytosine and the amount of adenine is equal to thymine. The significance of these ratios for the structure of DNA were not recognized until Watson, persisting in building structural models, realized that A:T and C:G pairs are structurally similar. In particular, the length of each base pair is the same these base pairs are held together by hydrogen bonds. After the discovery of the hydrogen bonded A:T and C:G pairs, Watson and Crick soon had their anti-parallel, double helical model of DNA, with the hydrogen bonds at the core of the helix providing a way to "unzip" the two complementary strands for easy replication.

In 1954, at the age of 37, Crick completed his Ph.D. Thesis: "X-Ray Diffraction: Polypeptides and Proteins" and received his degree. In 1956, Crick wrote an informal paper about the genetic coding problem for the small group of scientists in Gamow's RNA group. In this article, Crick reviewed the evidence supporting the idea that there was a common set of about 20 amino acids used to synthesize proteins. Crick proposed that there was a corresponding set of small "adaptor molecules" that would hydrogen bond to short sequences of a nucleic acid, and also link to one of the amino acids. Crick also used the term "central dogma" to summarize an idea that implies that genetic information flow between macromolecules would be essentially one-way: **DNA → RNA → Protein**

Proof that the genetic code is a degenerate triplet code finally came from genetics experiments, some of which were performed by Crick.

HONOURS: In addition to his third share of the 1962 Nobel prize for Physiology or Medicine, he received many awards and honours, including the Lasker Award of Basic Medical Research Award (1960) the Royal and Copley medals of the Royal Society (1972 and 1975), Order of Merit (on 27 November 1991).

Personal Life: Father: Harry Crick, Mother: Annie Elizabeth Wilkins Crick, Brother: A.F. Crick (medical doctor), Wife: Ruth Doreen Dodd Crick, Son: Michael F. C. Crick (scientist), Wife: Odile Crick, Daughter: Gabrielle A. Crick, Daughter: Jacqueline M. T. Crick.

Recognition:

Francis Crick Prize Lectures: The Francis Crick Lecture was established in 2003.The lecture is delivered annually in any field of biological sciences, with preference given to the areas in which Francis Crick himself worked.

Francis Crick Institute: The Francis Crick Institute is a planned £660,000,000 biomedical research centre to be located in London, UK. The Francis Crick Institute is a partnership between Cancer Research UK, Imperial College London, King's College London, the Medical Research Council, University College London (UCL) and the Welcome Trust. Once completed in 2015, it will be the biggest centre for biomedical research and innovation in Europe.

Francis Crick Graduate Lectures: The University of Cambridge Graduate School of Biological, Medical and Veterinary Sciences hosts The Francis Crick Graduate Lectures.

DEATH: Crick died of colon canceron 28 July 2004 at the University of California San Diego(UCSD) Thornton Hospital in La Jolla.

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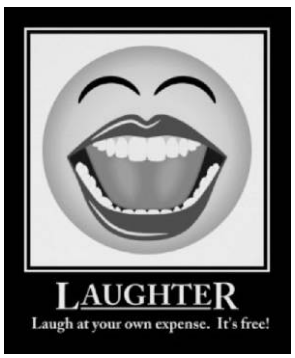
Enjoy the humour



A man was granted two wishes by God,
He asked for the best drink & the best woman ever.
Next moment he got mineral water & Mother Teresa.

There are 3 kinds of men in this world.
Some remain single and make wonders happen.
Some have girlfriends and see wonders happen.
Rests get married and wonder what happened!

Wives are magicians.
They can change anything into an argument.

Why do women live a Better, Longer & Peaceful Life, as compared to men?
A very INTELLIGENT student replied: "Because Women don't have a wife!"

COOL MESSAGE BY A WIFE:
Dear Mother-in-law, Don't teach me how to handle my children.
I am living with one of yours and he needs a lot of improvement!?

I WILL THINK ABOUT IT:
When a married man says, I'll think about it - what he really means is that he doesn't know his wife's opinion yet.

TALKING IN SLEEP:
A lady says to her doctor: "My husband has a habit of talking in his sleep! What should I give him to cure it?"
The doctor replies: "Give him an opportunity to speak when he's awake!

TRACK YOUR BRAIN

1. Which enzyme produced by Group A streptococci is called "spreading factor," an enzyme important in skin and soft tissue infection?
   A. Streptokinase
   B. Hyaluronidase
   C. M Protein
   D. Deoxyribonuclease C
   E. None of the above

2. Which of the following virulence factors of *E. coli* is important for attachment to host epithelial cells in the pathogenesis of urinary tract infections?
   A. Aerobactin
   B. Alpha hemolysin
   C. Urease
   D. K1 antigen
   E. Pili

3. Infections caused by anaerobes are:
   A. Usually confined to the abdomen
   B. Never seen in the lung because of its excellent blood supply
   C. Mixed
   D. Rapidly progressive

4. Non fermentative gram negative bacilli are:
   A. Strict aerobes
   B. Catalase negative
   C. Beta-hemolytic on sheep blood agar
   D. Spore forming

5. Which of the following is not a gram-negative bug?
   A. *Clostridium perfringens*
   B. *Vibrio cholerae*
   C. *Escherichia coli*
   D. *Bordetella pertussis*

6. Which of the following microorganisms are not matched correctly with the appropriate isolation media?
   A. Fungi - Sabourand's agar
   B. *Neisseria gonorrhoeae* – Pink colonies media
   C. *Haemophilus influenzae* – Chocolate agar
   D. *Mycobacterium tuberculosis* - Lowenstein-Jensen agar

7. Chagas' disease is commonly treated with Nifurtimox and is linked to the ____ microorganism.
   A. Naegleria
   B. *Schistosoma*
   C. *Wucheria bancrofti*
   D. *Trypanosoma cruzi*

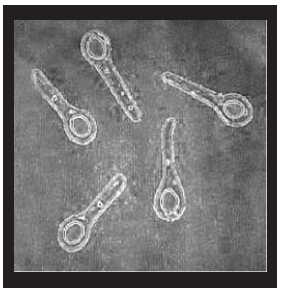
8. Which of the following is not a DNA virus?
   A. Adenovirus
   B. Calicivirus
   C. Papovirus
   D. Poxvirus

9. The Tzanck test is not used on which of the following viruses?
   A. VZV
   B. HSV-2
   C. HHV-8
   D. HSV-1

10. Which of the following microorganisms has not been linked to UTI's?
    A. *E. coli*
    B. *Pseudomonas*
    C. *Klebsiella*
    D. *Haemophilus*

ANSWERS: 1. D, 2. E, 3. C, 4. A, 5. A, 6. B, 7. D, 8. B, 9. C, 10. D.

Clostridium tetani



Classification:

Kingdom	: Bacteria
Phylum	: Firmicutes
Class	: Clostridia
Order	: Clostridiales
Family	: Clostridiaceae
Genus	: *Clostridium*
Species	: ***C. tetani***
Binomial Name	: *Clostridium tetani*

CHARACTERISTICS:

Clostridium tetani is a rod-shaped, anaerobic bacterium of the genus *Clostridium*. Like other *Clostridium* species, it is Gram-positive, and its appearance on a gram stain resembles tennis rackets or drumsticks. *C. tetani* is found as spores in soil or in the gastrointestinal tract of animals. *C. tetani* produces a potent biological toxin**, tetanospasmin**, and is the causative agent of tetanus, a disease characterized by painful muscular spasms that can lead to respiratory failure and, in up to 40% of cases, death.

During vegetative growth, the organism cannot survive in the presence of oxygen, is heat-sensitive and exhibits flagellar motility. As the bacterium matures, it develops a terminal spore, which gives the organism its characteristic appearance. *C. tetani* spores are extremely hardy as they are resistant to heat and most antiseptics. The spores are distributed widely in manure treated soils and can also be found on human skin and in contaminated heroin.

COOL FACTS:

A) One very interesting thing about *C. tetani* is that it produces four clinical types of tetanus. Incubation period ranges from 3-21 days, with an average of about a week. It has a fatality rate of 30%.

1) **Generalized** – (80%) (Most common type) Toxins get distributed via lymphatic and vascular system and spread more widely and affect more nerves. First symptom is the characteristic lock jaw. It spreads and begins to affect the rest of your muscle, starting with the neck and moving to your back. This generalized muscle rigidity comes with reflex spasms as your body tries to respond to various stimuli. These spasms can cause fractures, tendon rupture and respiratory failure. Death from tetanus results from respiratory failure and cardiovascular instability. Other symptoms caused by autonomic dysfunction may include fever, sweating and high blood pressure. Recovery can take months but is usually complete unless complications occur.
2) **Localized** – Very uncommon. Patients with this clinical type experience muscle rigidity close to the site of injury. These contractions can persist for many weeks before disappearing.
3) **Cephalic** – Form of localized disease that affects cranial nerves. It can happen after ear infections or head injuries. It affects cranial nerves so it can affect the muscles in your face (eyelid, tongue, lips, etc).
4) **Neonatal** – Form of generalized tetanus that occurs in newborn infants. Usually happens when the umbilical cord is cut with an unsterile instrument. There are some cultures where it is ritualistic to apply cow dung to the already cut umbilical cords of newborn infants. Very common in developing countries.

B) Another cool fact about *C. tetani* has to do with the common myth about rusty objects. Contrary to common belief, it is not true that wounds caused by rusty objects, such as nails, cause tetanus. The fact is that it is not likely that the rusted object that has caused the wound will be carrying *C. tetani* bacterium. The object will only increase the chance of infection because of the open wound that it has created.

TOXICITY:

C. tetani usually enters a host through a wound to the skin, then it replicates. Once an infection is established, *C. tetani* produces **two exotoxins, tetanolysin and tetanospasmin.** Eleven strains of *C. tetani* have been identified, which differ primarily in flagellar antigens and in their ability to produce tetanospasmin. The genes for toxin production are encoded on a plasmid which is present in all toxigenic strains, and all strains that are capable of producing toxin produce identical toxins.

Tetanolysin serves no known benefit to *C. tetani*. Tetanospasmin is a neurotoxin that causes the clinical manifestations of tetanus. Tetanus toxin is generated in living bacteria, and is released when the bacteria lyse, such as during spore germination or vegetative growth. A minimal amount of spore germination and vegetative cell growth are required for toxin production.

On the basis of weight, tetanospasmin is one of the most potent toxins known. The estimated minimum human lethal dose is 2.5ng per kg of body weight, or 175 ng in a 70 kg (154 lb) human. The only toxins more lethal to mice are botulinum toxin, produced by close relative *Clostridium botulinum* and the exotoxin produced by *Corynebacterium diphtheriae*, the causative agent of diphtheria.

Tetanospasmin is a zinc-dependent metalloproteinase that is structurally similar to botulinum toxin, but with different effects. *C. tetani* synthesizes tetanospasmin as a single 150kDa polypeptide progenitor toxin that is then cleaved by a protease into two fragments; fragment A (a 50kDa "light chain") and fragment B (a 100 kDa "heavy chain") which remain connected via a disulfide bridge. Cleavage of the progenitor toxin into A and B fragments can be induced artificially by trypsin.

TOXIN ACTION:

Tetanospasmin released in the wound is absorbed into the circulation and reaches the ends of motor neurons all over the body. The toxin acts at several sites within the central nervous system, including nerve terminals, the spinal cord, and brain, and within the sympathetic nervous system. By binding to peripheral motor neuron terminals, the toxin enters the nerve axons, and is transported across synaptic junctions to the nerve-cell body in the brain stem and spinal cord by retrograde intraneuronal transport, until it reaches the central nervous system, where it rapidly binds to gangliosides at the presynaptic membrane of inhibitory motor nerve endings.

The clinical manifestations of tetanus are caused when tetanus toxin blocks inhibitory impulses, by interfering with the release of neurotransmitters, including glycine and gamma-aminobutyric acid. These inhibitory neurotransmitters inhibit the alpha motor neurons. With diminished inhibition, the resting firing rate of the alpha motor neuron increases, producing

rigidity, unopposed muscle contraction and spasm. Characteristic features are risus sardonicus (a rigid smile), trismus (commonly known as "lock-jaw"), and opisthotonus (rigid, arched back). Seizures may occur, and the autonomic nervous system may also be affected. Tetanospasmin appears to prevent the release of neurotransmitters by selectively cleaving a component of synaptic vesicles called synaptobrevin II. Loss of inhibition also affects preganglionic sympathetic neurons in the lateral gray matter of the spinal cord and produces sympathetic hyperactivity and high circulating catecholamine levels. Hypertension and tachycardia alternating with hypotension and bradycardia may develop.

TREATMENT TO TETANUS:

The goals of treatment in patients with tetanus include stopping the production of toxin within the wound, neutralizing unbound toxin, controlling disease manifestations, and managing complications.

Stopping toxin production

Antimicrobials are used to decrease the number of vegetative forms (source of toxin) of *C tetani*in the wound. The current antimicrobial drug of choice is metronidazole, with penicillin as an alternative treatment. A higher survival rate was obtained with metronidazole than with penicillin in one nonrandomized trial. Less CNS excitation is seen with metronidazole than with penicillin. Other antimicrobials that have been used include clindamycin, erythromycin, tetracycline, and vancomycin.

Neutralizing unbound toxin

Human tetanus immunoglobulin (TIG) is recommended for treatment. A single total dose of 3000-6000 U is recommended for children and adults. The optimal dose has not yet been established, but doses as low as 500 U have been effective in infants with tetanus neonatorum.

Available preparations must be intramuscularly administered at the time of diagnosis to prevent further circulating toxin from reaching the CNS. Some authorities recommend infiltration of part of the dose locally around the wound, although the efficacy of this approach has not been proven.

The TIG formulation available in the United States is not licensed or appropriate for intravenous or intrathecal use. In countries in which TIG is not available, equine tetanus antitoxin may be available.

Tetanus antitoxin is administered as a single dose of 50,000-100,000 U after appropriate testing for sensitivity and desensitization, if necessary. Part of this dose (20,000 U) should be intravenously administered.

Controlling disease manifestations

A benzodiazepine should be used to produce sedation, decrease rigidity, and control spasms (midazolam administered intravenously at 5-15 mg/h is suitable). If the spasms are not controlled with benzodiazepines, long-term neuromuscular blockade is required.

Managing complications

Specific therapy for autonomic system complications and control of spasms should be initiated. Sympathetic hyperactivity is treated with combined alpha and beta blockade or morphine. Epidural blockade with local anesthetics may be needed. Hypotension requires fluid replacement and dopamine or nor epinephrine administration. Parasympathetic over activity is rare, but if bradycardia is sustained, a pacemaker may be needed. Clinical tetanus does not induce immunity against future attacks; therefore, all patients should be fully immunized with tetanus toxoid during the convalescent period.

CURRENT RESEARCH:

In a 2002 study on anti-cancer drug forms, it was discovered that *C. tetani* and other members of the *Clostridium* family could be used for tumor-specific drug delivery. More specifically, the spores of *C. tetani* and other members of the genus would be used in this anti-cancer drug delivery process. It was found that the presence of hypoxia, which are components of solid tumors that result from a lack of sufficient blood supply, were dramatically reducing the efficiency of anti-cancer techniques, such as surgery, chemotherapy, and radiation. The use of genetically recombined spores of *C. tetani* that lack the pathogenicity of normal *C. tetani* (due to the tetanus toxin) species, were used to deliver toxic elements specific to the tumor cells. Upon administration to the target tumor, these bacteria colonize specific regions of the hypoxia and can be genetically transformed to produce enzymes like cytosine deaminase and tumor necrosis factor alpha. This protein delivery process via the use of *Clostridium* bacterium is thought to be very safe because specific antibiotics can be administered at any point to get rid of the specific bacteria. This study has provided an alternative technique to cancer treatment that involves the delivery of anti-cancer proteins to a specific tumor site.

A 2008 study in Kano, Nigeria sought to determine the susceptibility of *Clostridium tetani* to various antibiotics. Soil was collected from five different locations and cultured under anaerobic conditions to observe the number of samples which contained *Clostridium tetani* spores. The bacterium was observed in 60% of the soil samples. The samples were treated with amoxicillin, chloramphenicol, tetracycline, erythromycin, augmentin, co–trimoxazole, metronidazol, penicillin V, gentamycin, cloxacillin, sparfloxacin and ciprofloxacin to determine antibiotic susceptibility. The most effective antibiotic in preventing colony growth was observed to be sparfloxacin, with erythromycin, tetracycline, gentamycin, chloramphenicol, metronidazole and ciprofloxacin also preventing growth. The remaining antibiotics appeared to be inaffective against *Clostridium tetani*.

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Mycoplasma

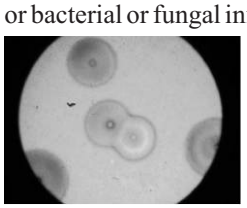
Mycoplasmas are the smallest free-living organisms that, unlike other bacteria, lack a cell wall. The outer layer is instead, a three layered membrane containing sterols. Diameters of these organisms may range from 0.2-0.3 µm and, due to their plasticity, are able to pass through the pores of a 0.2 micron filter with applied pressure. Because the morphology of Mycoplasmas are pleomorphic, they occur as two different structural forms during a life cycle: coccoidal, a spherical or spheroidal shape, and filamentous, resembling rods.

Because Mycoplasma lack a cell wall, the organisms are poorly stained, if at all, by bacterial stains. With the exception of *M. hyorhinis*, most Mycoplasma can be cultivated using standardized and Mycoplasma agar formulations, as well as in broth media, although growth is slow. When grown on agar, the colonies have a "fried-egg" appearance since the colony center grows into the and appears more dense than the rest of the colony.

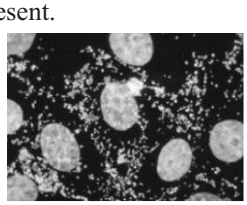
Unless it is specifically tested for, cultures contaminated by Mycoplasma often remain undetected since there are no obvious signs of contamination, like the destruction of host cells. The main source of mycoplasma infection, in the majority of cases, is cross contamination by a previously mycoplasma-infected cell culture which is used in the same laboratory. Mycoplasmas were surprising hardy despite their lack of a cell well. They survived on surfaces, even in operating laminar flow hoods, for as long as six days. The following clues may help signal deterioration of a culture affected by Mycoplasma contamination:

- Interference with the rate of cell growth
- Changes in cell morphology
- Aberrations in chromosomes
- Altered DNA, RNA, and protein synthesis
- Induced cell transformation

Several methods exist to test for the presence of Mycoplasma. One is the Hoechst 33258 DNA staining method. This technique is rapid (less than 30 minutes), but requires heavy contamination (10^6 mycoplasma/ml) to produce a clear positive result. If the suspect cells are co-incubated for 2–4 days with an "indicator" cell line (such as 3T3) that is particularly suitable for demonstration of positive staining, then sensitivity can be substantially increased. Cell cultures are stained with Hoechst 33258, a fluorescent stain, which binds specifically to DNA. When viewed with fluorescent microscopy, uncontaminated cultures have a low cytoplasm/nucleus ratio, while the nuclei and extranuclear mycoplasmal DNA fluoresce in infected cells. The advantages to using the Hoechst 33258 DNA stain include detection of the non-cultivable strain *M. hyorhinis*. However, this method has low sensitivity and fails to detect low titres. It may be difficult to differentiate Mycoplasma from disintegrating nuclei, or bacterial or fungal infections, if present.



Typical Mycoplasma colonies with the classic "fried egg" morphology



DNA fluorochrome staining of Vero cells infected with Mycoplasma hyorhinis

Other methods of detection include the culture method in which growth is observed on standardized agar or in broth media with the exception of *M. hyorhinis*. Mycoplasma detection kits and PCR may also be used to detect Mycoplasma contamination. CELLshipper® kit is a direct DNA fluorochrome staining kit specifically developed by Bionique Testing Laboratories, Inc. for the detection of mycoplasma contamination in cell cultures.

Some common organisms that cause cell culture contamination include *M. hyorhinis* porcine, *M. arginini* bovine, and *M. orales M. fermentans* natural hosts are humans. Unfortunately, in the event of contaminated cultures, the best course of action is to discard the culture, as it can be difficult to completely eradicate the organism. If the cell line is irreplaceable, however, antibiotics such as ciprofloxacin may be used to eliminate the Mycoplasma. Prophylactic use of antibiotics as a preventative for any type of contamination is not recommended, as they may mask contamination and cause it to remain undetected, particularly if resistance is a problem or if the antibiotic is bacteriostatic instead of bacteriocidal. It is observed that cell cultures routinely grown in antibiotic containing media had 10-fold higher rates of mycoplasma contamination (72%) than cultures grown antibiotic-free (7%).Laboratories, documented the antibiotic resistance of mycoplasma isolates from infected cell cultures: streptomycin 88%, kanamycin 73%, gentamicin 80%. As a result, there is an absence of visible signs to indicate the culture has been contaminated with mycoplasma. Had antibiotics not been used, then the other easier to detect contaminants would have quickly taken over the culture, been observed and the culture discarded. Hence the use of antibiotics should be limited to contaminated cultures, and only when discarding is out of the question. addition, rigid aseptic technique is a must to prevent Mycoplasma infection since poor technique may become habit-forming.

All basal media and salt solutions are required to be tested for the presence of Mycoplasma as an assurance that, these products will not bring Mycoplasma into customers' labs. The quickest and most reliable way to eliminate mycoplasma is to autoclave the contaminated cultures and any bottles of media or sterile reagents that were used with them. Autoclaving is the only guaranteed method for mycoplasma elimination. Afterwards, replace them with fresh culture stocks which have been certified as mycoplasma-free.

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Isolation and Presumptive Identification of Bacterial Agents from Normally Sterile Sites

In general laboratories commonly receive blood samples or cerebrospinal fluid from patients with pneumonia, meningitis, or unexplained febrile illness. Laboratories may also receive urine, joint fluid, pleural fluid, or other sterile site specimens from these patients. This section of the laboratory manual provides methods for the isolation and presumptive identification of agents from these normally sterile sites. Pathogens included in this laboratory manual that could be isolated from normally sterile sites are *Haemophilus influenzae, Neisseria meningitidis, Salmonella* serotype Typhi, and *Streptococcus pneumoniae*. Personnel who are at risk for the routine exposure to aerosolized *N. meningitides* should strongly consider vaccination. The risk of infection when working in the laboratory with *H. influenzae* and *S. pneumoniae* is very low and it is not required that laboratorians receive vaccination against these organisms. However, least two good vaccines (oral and injection) are available for S. typhi, and laboratorians should ensure that their vaccination status remains current. After bacteria are recovered from normally sterile sites, the isolates require confirmatory identification; isolates received by a reference laboratory (*e.g.*, for antimicrobial susceptibility testing) must also undergo confirmatory testing.

Blood cultures

Laboratory personnel handling blood culture specimens must be able to identify culture bottles that may have bacterial growth, isolate bacteria on solid media, and subculture isolates. Provisional identification of an isolate will often be possible on the basis of colony morphology and the microscopic appearance of a Gram stained specimen.

Several variables affect the sensitivity of blood cultures: the number of collections, the volume of each collection, and the steps taken to inhibit or neutralize bactericidal properties of blood may vary with the age of the patient. As stated in the section on specimen collection, blood cultures from young children should be diluted to 1–2 ml of blood in 20 ml of broth (1:10 to 1:20), whereas blood cultures from adults should be diluted to 5–10 ml of blood in 50 ml of broth (1:5 to 1:10). Ideally, the blood samples should be processed in a bacteriology laboratory as soon as possible after collection (*i.e.*, within 2 hours).

Inoculation of primary culture media

Blood should be cultured in a tryptone-based soy broth (commonly referred to as "Trypticase" or "tryptic" soy broth [TSB]) or brain heart infusion with a supplement, such as haematin or sodium polyanetholesulfonate (SPS). If only one blood-culture bottle is used, it should contain TSB. Neutralization of normal bactericidal properties of blood and potential antimicrobial agents is accomplished by adding chemical inhibitors such as 0.025% SPS to culture media and by diluting the blood. SPS, which has anticoagulant, antiphagocytic, anti complementary, and antilysozymal activity, **may be inhibitory if used in higher concentrations**, but it is important to use. The blood-culture bottles should be inoculated directly with blood and should be vented before incubation at 35°– 37°C. Venting is accomplished by inserting a sterile cotton-plugged needle into the diaphragm (*i.e.*, rubber part) of the blood-culture bottle. Adding growth supplements, such as IsoVitaleX or Vitox, to blood culture bottles to help support the growth of *H. influenzae* is appropriate; however, if resources are limited, a laboratory would benefit more by using this costly resource to supplement chocolate agar medium.

Identifying positive blood culture bottles

Blood-culture bottles should be examined at 14–17 hours and then every day for up to 7 days. Any turbidity or lysis of erythrocytes may be indicative of growth, and subcultures should be made immediately. Because *H. influenzae, N. meningitidis,* and *S. pneumoniae* are fragile organisms, **subcultures should be routinely performed after 14–17 hours of incubation, again at 48 hours, and again at day 7, regardless of the appearance of the blood-culture bottles** because the absence of turbidity does not always correlate with the absence of bacterial growth. Before sub culturing, swirls the bottle to mix the contents.

Subculture

Subcultures are made by first disinfecting the surface of the blood-culture bottle diaphragm with alcohol and a povidone-iodine swab, and then aspirating a small volume (*i.e.*, 0.5 ml) with a syringe and needle from the blood-culture bottle and inoculating the agar media with the fluid. If the bottle has a screw-cap, open the bottle and take the fluid using sterile technique (*i.e.*, flaming the bottle mouth upon opening and closing the cap). Ordinarily, both chocolate agar plates and blood agar plates are used for subculture. **When only one agar plate is used, it should be chocolate agar, because chocolate agar contains the X and V growth factors needed for *H.influenzae,* whereas blood agar does not.** If a blood specimen is received from a patient with a primary diagnosis of fever of unknown origin, if typhoid is suspected symptomatically, or if a Gram stain of blood-culture broth reveals gram negative bacilli, add a total of 3–4 loopfuls of the blood culture onto MacConkey agar (MAC) in addition to chocolate agar and/or blood agar. Incubate the media with suspect pathogens at 35°–37°C in a 5% CO2 atmosphere (incubator or candle-extinction jar). Because *N. meningitidis* grows well in a humid atmosphere, if an infection with *N. meningitidis* is suspected, laboratories may choose to add a shallow pan of water to the bottom of the incubator or add a dampened paper towel to the candle-extinction jar; the moisture source should be changed regularly (*e.g.*, daily) to prevent contamination with molds. If the laboratory has the resources to support the use of a third plate for subculture, MacConkey agar should be used, particularly when the specimen was obtained from a patient with fever of unknown origin (when typhoid fever [*S. typhi*] or blood stream infection by gram-negative rods of other species [*e.g.*, *E. coli, Klebsiella,* etc.] may be suspected). Chocolate agar should be periodically confirmed to support growth of *H. influenzae.* The agar plates should be streaked, and incubated for up to 48 hours. The MAC and blood plates for *S. typhi* should be incubated for 18–24 hours at 35°–37°C. When bacterial growth has been confirmed by subculture of the blood-culture bottle, the bottle no longer requires incubation. The bottle should be disposed of according to safety procedures.

Presumptive identification of isolates from sterile-site specimens

Because the primary purpose of this section of the manual is to aid in the identification of *N. meningitidis, S. pneumoniae, H.*

influenzae, and *S. typhi* from sterile-site specimens, the methods described here will not apply to the identification of other bacterial agents (of pneumonia and meningitis) of clinical importance that are more rarely encountered. Microbiologists should refer to clinical microbiology manuals (*e.g.*, the American Society for Microbiology's *Manual of Clinical Microbiology,* the WHO's *Manual for the Laboratory Investigations of Acute Enteric Infections,* the *Clinical Microbiology Procedures Manual, Basic Laboratory Procedures in Clinical Microbiology* [WHO 2001] or a medical microbiology manual or textbook for procedures to identify other bacteria. Presumptive identification of *N. meningitidis, H. influenzae,* and *S. pneumoniae* can be made on the basis of the growth on blood agar and chocolate agar and on the basis of the microscopic morphology of the organisms.

N. meningitidis grows on blood agar, whereas *H. influenzae* will not grow without supplements (found in chocolate agar).When grown on chocolate agar, *H. influenzae* and *N. meningitidis* look similar; the two organisms can be distinguished on the agar plate by the pungent smell of indole from *H. influenzae*.

The following procedures should be followed to prepare a dried smear for Gram stain of pure culture.

a) Place one drop of physiological saline or distilled water on an alcohol-rinsed and dried slide.

b) With a flamed and cooled, sterile inoculating needle or loop, touch the center of the bacterial colony.

c) Prepare a smear from the colony by adding the bacteria from the inoculating loop to the physiological saline or distilled water drop with a gentle tap. Use the loop to mix the organisms into suspension.

d) Spread the suspension and allow it to dry, either by air (approximately ten minutes) or incubator.

Upon microscopic examination, organisms that are gram-positive will appear violet, while gram-negative organisms will appear pink. The staining further enables the laboratorian to see morphology of the bacteria.

Presumptive identification of *H. influenzae*

H. influenzae appears as large, flat, colorless-to-grey opaque colonies on chocolate agar. No hemolysis or discoloration of the medium is apparent. Encapsulated strains appear more mucoidal than non-encapsulated strains, which appear as compact greyish colonies. Gram staining will yield small, gram-negative bacilli or coccobacilli.

Presumptive identification of *N. meningitidis*

On blood agar plates, young colonies of *N. meningitidis* are round, smooth, moist, glistening and convex, with a clearly defined edge. Some colonies appear to coalesce with other nearby colonies. Growth of *N. meningitidis* on blood agar is greyish and unpigmented; older cultures become more opaquely grey and sometimes cause the underlying agar to turn dark. Well-separated colonies can grow from about 1 mm in diameter in 18 hours to as large as 4 mm, with a somewhat undulating edge, after several days. Gram staining will yield a gram negative, coffee-bean-shaped diplococcus.

Presumptive identification of *S. pneumoniae*

S. pneumoniae appears as small, greyish, moist (sometimes mucoid), watery colonies with a greenish zone of hemolysis surrounding them on blood agar and chocolate agar. The degree of mucoidness of *S. pneumonia* colonies is dependent on the freshness of the medium and the incubation atmosphere. Some serotypes appear more mucoid than others, and the fresher the medium, the more mucoid the cultures appear.

Young pneumococcal colonies appear raised, similar to viridans streptococci. Differentiating pneumococci from viridans streptococci on chocolate agar is difficult. However, a hand lens or microscope (30X-50X) is a useful aid in differentiating pneumococci from hemolytic viridans streptococci, which also produce a greenish zone of hemolysis on a blood- or chocolate agar plate. However, as the culture ages 24-48 hours, the colonies become flattened and the central part of each colony becomes depressed. This does not occur with the viridans streptococci.

Another type of colony that might appear on the culture plate along with *S. pneumoniae* is *Staphylococcus aureus* (or another *Staphylococcus* species). Two types of colonies will grow on the 5% sheep blood trypticase soy agar medium: the dull gray flat colony surrounded by a greenish zone of hemolysis will be *S. pneumoniae* and the yellowish colony with no hemolytic action will be *S. aureus.* Gram staining of *S. pneumoniae* will reveal a gram positive diplococci or chain of cocci.

Presumptive identification of *Salmonella* ser.Typhi

Salmonella ser. Typhi grows on both blood agar and chocolate agar; on these media, *S. typhi* colonies are grayish, transparent to opaque, glistening (shiny) and usually >1 mm in diameter. On MacConkey agar (MAC), *S. typhi* colonies appear as colorless non fermenters. (Colonies of S. Paratyphi A, S. Paratyphi B, and S. Paratyphi C and most other *Salmonella* serotypes look similar to those of *S. typhi* on these media.) Gram staining of *Salmonella* serotypes will reveal gram-negative bacilli.

Presumptive identification of *Haemophilus influenzae, Neisseria meningitidis,* and *Streptococcus pneumonia*

Growth on		Gram stain morphology	Presumptive identification
Chocolate agar	Sheep blood agar		
+	+	gram-negative diplococci	*N. meningitidis*
+	+	gram-positive cocci or diplococci	*S. pneumoniae*
+	-	small, gram-negative pleomorphic coccobacilli	*H. influenzae*

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(1) www.who.ntm/drugresistance/Publications (2) www.ncbi.nlm.nih.gov

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TST0750	Triosept	Bactericidal, fungicidal and virucidal	Personal hand hygiene

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MLE0750	Microlyse – C	Bactericidal, fungicidal and virucidal	For floor mopping and surface disinfection
NST0750	Nusept – C	Bactericidal, fungicidal and virucidal	For surface disinfection and general purpose disinfection
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Industrial: In Pharmaceutical industry, Food & beverage industry, Hotel industry etc. General surface disinfection. Eliminating biofilms.

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Surgical, post operative, non surgical dressing	Use undiluted, once a day/alternate day
Surgical bath/Sitz bath	Add 50 mL of NUSEPT™ in 1 L of water & use
Antisepsis during minor incisions, Scopy, Catheterization, first aid, cuts, bites, stings etc	Use undiluted
Chronic wound management (diabetic foot, pressure and venous leg ulcers)	Use undiluted
Burn wound management (Only for 1st and 2nd Degree burns, chemical burns)	Use 100 mL NUSEPT™ in 1 L sterile water for both washing (with 1 minute contact time) and dressing of burn wound (Dressing must be changed everyday/ alternate days or as directed)
Midwifery, nursery & sickroom	Use undiluted
Intra-operative irrigation	Use 50 mL NUSEPT™ in 1 L sterile water
General hard surface disinfection	Add 100 mL of NUSEPT™ in 1 L of water and gently mop the floor or surfaces

Highlights of the coming issue

Printed and 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.**