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Contents

Editorial

Well to Jump start with this issue we have the section on 'Mini Review' giving us a description of a variety of airborne infections in susceptible hosts can result from exposures to clinically significant microorganisms released into the air when environmental reservoirs (i.e., soil, water, dust, and decaying organic matter) are disturbed.

Current Trends mentions Vibriosis is a general term referring to an infection by any member of the large group of *Vibrio*, bacteria. ***Vibrio*** is a genus of Gram-negative bacteria possessing a curved-rod shape (comma shape), several species of which can cause foodborne infection, usually associated with eating undercooked seafood. Typically found in saltwater, *Vibrio* species are facultative anaerobes that test positive for oxidase and do not form spores.

Selman Abraham Waksman was a Ukrainian-born, Jewish-American inventor, biochemist and microbiologist whose research into organic substances—largely into organisms that live in soil—and their decomposition

'Relax Your Mood' as usual helps you to unwind, laugh and think about what could fill the void, in track your brains.

Yersinia enterocolitica is a Gram-negative bacillus-shaped bacterium, belonging to the family Enterobacteriaceae. *Y. enterocolitica* infection causes the disease yersiniosis, which is a zoonotic disease occurring in humans, as well as a wide array of animals such as cattle, deer, pigs, and birds.

"Did You Know" Antibiotic resistance is poised to spread globally among bacteria frequently implicated in respiratory and urinary infections in hospital settings, according to new research at Washington University School of Medicine in St. Louis.

Antimicrobial resistance occurs when a microorganism is no longer destroyed or stopped from reproducing by an anti-microbial medicine to which it was originally sensitive - quite simply, "the drugs don't work anymore". In recent years, some common pathogens have demonstrated multi-drug resistance and have caused infections, including those of urinary tract, bloodstream and wounds. Tackling Antimicrobial resistance (AMR) requires action on multiple levels, the prudent use of antibiotics being one of the key actions. 'Best Practices' takes a look at this important approach.

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Airborne Infectious Diseases in Health-Care Facilities

A variety of airborne infections in susceptible hosts can result from exposures to clinically significant microorganisms released into the air when environmental reservoirs (i.e., soil, water, dust, and decaying organic matter) are disturbed. Once these materials are brought indoors into a health-care facility by any of a number of vehicles (e.g., people, air currents, water, construction materials, and equipment), the attendant microorganisms can proliferate in various indoor ecological niches and, if subsequently disbursed into the air, serve as a source for airborne health-care–associated infections.

Respiratory infections can be acquired from exposure to pathogens contained either in droplets or droplet nuclei. Exposure to microorganisms in droplets (e.g., through aerosolized oral and nasal secretions from infected patients) constitutes a form of direct contact transmission. When droplets are produced during a sneeze or cough, a cloud of infectious particles >5 µm in size is expelled, resulting in the potential exposure of susceptible persons within 3 feet of the source person. Examples of pathogens spread in this manner are influenza virus, rhinoviruses, adenoviruses, and respiratory syncytial virus (RSV). Because these agents primarily are transmitted directly and because the droplets tend to fall out of the air quickly, measures to control air flow in a health-care facility (e.g., use of negative pressure rooms) generally are not indicated for preventing the spread of diseases caused by these agents. The spread of airborne infectious diseases via droplet nuclei is a form of indirect transmission. Droplet nuclei are the residuals of droplets that, when suspended in air, subsequently dry and produce particles ranging in size from 1–5 µm. These particles can (a) contain potentially viable microorganisms, (b) be protected by a coat of dry secretions, (c) remain suspended indefinitely in air, and (d) be transported over long distances. The microorganisms in droplet nuclei persist in favourable conditions (e.g., a dry, cool atmosphere with little or no direct exposure to sunlight or other sources of radiation). Pathogenic microorganisms that can be spread via droplet nuclei include *Mycobacterium tuberculosis*, measles virus (i.e., rubeola), and smallpox virus (i.e., variola major). Several environmental pathogens have life-cycle forms that are similar in size to droplet nuclei and may exhibit similar behaviour in the air. The spores of *Aspergillus fumigatus* have a diameter of 2–3.5 µm, with a settling velocity estimated at 0.03 cm/second (or about 1 meter/hour) in still air. With this enhanced buoyancy, the spores, which resist desiccation, can remain airborne indefinitely in air currents and travel far from their source.

Airborne Infectious Diseases in Health-Care Facilities

a. Aspergillosis and Other Fungal Diseases

Aspergillosis is caused by molds belonging to the genus Aspergillus. Aspergillus spp. are prototype health-care acquired pathogens associated with dusty or moist environmental conditions. Aspergillus spp. is ubiquitous, aerobic fungi that occur in soil, water, and decaying vegetation; the organism also survives well in air, dust, and moisture present in health-care facilities. The presence of aspergilli in the health-care facility environment is a substantial extrinsic risk factor for opportunistic invasive aspergillosis (invasive aspergillosis being the most serious form of the disease). Site renovation and construction can disturb Aspergillus-contaminated dust and produce bursts of airborne fungal spores. Increased levels of atmospheric dust and fungal spores have been associated with clusters of health-care acquired infections in immune-compromised patients.

Patient-care items, devices, and equipment can become contaminated with Aspergillus spp. spores and serve as sources of infection if stored in such areas. Most cases of aspergillosis are caused by Aspergillus fumigatus, a thermotolerant/thermophilic fungus capable of growing over a temperature range from 12°C–53°C; optimal growth occurs at approximately 40°C, a temperature inhibitory to most other saprophytic fungi. It can use cellulose or sugars as carbon sources; because its respiratory process requires an ample supply of carbon, decomposing organic matter is an ideal substrate.

Other opportunistic fungi that have been occasionally linked with health-care–associated infections are members of the order Mucorales (e.g., Rhizopus spp.) and miscellaneous moniliaceous molds (e.g., Fusarium spp. and Penicillium spp.). Many of these fungi can proliferate in moist environments (e.g., water-damaged wood and building materials). Some fungi (e.g., Fusarium spp. And Pseudoallescheria spp.) also can be airborne pathogens. As with aspergillosis, a major risk factor for disease caused by any of these pathogens is the host's severe immunosuppression from either underlying disease or immunosuppressive therapy. Infections due Cryptococcus neoformans, Histoplasma capsulatum, or Coccidioides immitis can occur in health-care settings if nearby ground is disturbed and a malfunction of the facility's air-intake components allows these pathogens to enter the ventilation system. C. neoformans is a yeast usually 4 - 8 µm in size. However, viable particles of <2 µm diameter (and thus permissive to alveolar deposition) have been found in soil contaminated with bird droppings, particularly from pigeons.

Substantial numbers of these infectious particles have been associated with chicken coops and the roosts of blackbirds. Several outbreaks of histoplasmosis have been associated with disruption of the environment; construction activities in an endemic area may be a potential risk factor for health-care–acquired airborne infection. C. immitis, with arthrospores of 3–5 µm diameter, has similar potential. Emerging evidence suggests that Pneumocystis carinii, now classified as a fungus, may be spread via airborne, person-to-person transmission. Controlled studies in animals first demonstrated that P. carinii could be spread through the air. More recent studies in health-care settings have detected nucleic acids of P. carinii in air samples from areas frequented or occupied by P. carinii-infected patients but not in control areas that are not occupied by these patients. Clusters of cases have been identified among immune-

compromised patients who had contact with a source patient and with each other. Recent studies have examined the presence of P. carinii DNA in oropharyngeal washings and the nares of infected patients, their direct contacts, and persons with no direct contact. Molecular analysis of the DNA by polymerase chain reaction (PCR) provides evidence for airborne transmission of P. carinii from infected patients to direct contacts, but immunocompetent contacts tend to become transiently colonized rather than infected. The role of colonized persons in the spread of P. carinii pneumonia (PCP) remains to be determined. At present, specific modifications to ventilation systems to control spread of PCP in a health-care facility are not indicated. Current recommendations PCP prophylaxis with PCP-infected patients.

b. Tuberculosis and Other Bacterial Diseases

The bacterium most commonly associated with airborne transmission is Mycobacterium tuberculosis. A comprehensive review of the microbiology and epidemiology of M. tuberculosis and guidelines for tuberculosis (TB) infection control have been published. M. tuberculosis is carried by droplet nuclei generated when persons (primarily adults and adolescents) who have pulmonary or laryngeal TB sneeze, cough, speak, or sing; normal air currents can keep these particles airborne for prolonged periods and spread them throughout a room or building.

However, transmission of TB has occurred from mycobacteria aerosolized during provision of care (e.g., wound/lesion care or during handling of infectious peritoneal dialysis fluid) for extrapulmonary TB patients. Gram-positive cocci (i.e., Staphylococcus aureus, group A beta-hemolytic streptococci), also important health-care–associated pathogens, are resistant to inactivation by drying and can persist in the environment and on environmental surfaces for extended periods. These organisms can be shed from heavily colonized persons and discharged into the air. Airborne dispersal of S. aureus is directly associated with the concentration of the bacterium in the anterior nares. Approximately 10% of healthy carriers will disseminate S. aureus into the air, and some persons become more effective disseminators of S. aureus than others. The dispersal of S. aureus into air can be exacerbated by concurrent viral upper respiratory infection, thereby turning a carrier into a "cloud shedder." Outbreaks of surgical site infections (SSIs) caused by group A beta-hemolytic streptococci have been traced to airborne transmission from colonized operating-room personnel to patients. In these situations, the strain causing the outbreak was recovered from the air in the operating room or on settle plates in a room in which the carrier exercised. S. aureus and group A streptococci have not been linked to airborne transmission outside of operating rooms, burn units, and neonatal nurseries. Transmission of these agents occurs primarily via contact and droplets.

Other gram-positive bacteria linked to airborne transmission include Bacillus spp. which are capable of sporulation as environmental conditions become less favorable to support their growth. Outbreaks and pseudo-outbreaks have been attributed to Bacillus cereus in maternity, pediatric, intensive care, and bronchoscopy units; many of these episodes were secondary to environmental contamination. Gram-negative bacteria rarely are associated with episodes of airborne transmission because they generally require moist environments for persistence and growth. The main exception is Acinetobacter spp., which can withstand the inactivating effects of drying. In one epidemiologic investigation of bloodstream infections among pediatric patients, identical Acinetobacter spp. were cultured from the patients, air, and room air conditioners in a nursery.

Aerosols generated from showers and faucets may potentially contain legionellae and other gram-negative waterborne bacteria (e.g., Pseudomonas aeruginosa). Exposure to these organisms is through direct inhalation.

c. Airborne Viral Diseases

Some human viruses are transmitted from person to person via droplet aerosols, but very few viruses are consistently airborne in transmission (i.e., are routinely suspended in an infective state in air and capable of spreading great distances), and health-care–associated outbreaks of airborne viral disease are limited to a few agents. Consequently, infection-control measures used to prevent spread of these viral diseases in health-care facilities primarily involve patient isolation, vaccination of susceptible persons, and antiviral therapy as appropriate rather than measures to control air flow or quality. Infections caused by VZV frequently are described in health-care facilities. Health-care–associated airborne outbreaks of VZV infections from patients with primary infection and disseminated zoster have been documented; patients with localized zoster have, on rare occasions, also served as source patients for outbreaks in health-care facilities. VZV infection can be prevented by vaccination, although patients who develop a rash within 6 weeks of receiving varicella vaccine or who develop breakthrough varicella following exposure should be considered contagious.

Viruses whose major mode of transmission is via droplet contact rarely have caused clusters of infections in group settings through airborne routes. The factors facilitating airborne distribution of these viruses in an infective state are unknown, but a presumed requirement is a source patient in the early stage of infection that is shedding large numbers of viral particles into the air. Airborne transmission of measles has been documented in health-care facilities. In addition, institutional outbreaks of influenza virus infections have occurred predominantly in nursing homes, and less frequently in medical and neonatal intensive care units, chronic-care areas, HSCT units, and pediatric wards. Some evidence supports airborne transmission of influenza viruses by droplet nuclei, and case clusters in pediatric wards suggest that droplet nuclei may play a role in transmitting certain respiratory pathogens (e.g., adenoviruses and respiratory syncytial virus [RSV]). Some evidence also supports airborne transmission of enteric viruses. An outbreak of a Norwalk-like virus infection involving more than 600 staff personnel over a 3-week period was investigated in a Toronto, Ontario hospital in 1985; common sources (e.g., food and water) were ruled out during the investigation, leaving airborne spread as the most likely mode of transmission. Smallpox virus, a potential agent of bioterrorism, is spread predominantly via direct contact with infectious droplets, but it also can be associated with airborne transmission. A German hospital study from 1970 documented the ability of this virus to spread over considerable distances and cause infection at low doses in a well-vaccinated population; factors potentially

facilitating transmission in this situation included a patient with cough and an extensive rash, indoor air with low relative humidity, and faulty ventilation patterns resulting from hospital design (e.g., open windows). Smallpox patients with extensive rash are more likely to have lesions present on mucous membranes and therefore have greater potential to disseminate virus into the air. In addition to the smallpox transmission in Germany, two cases of laboratory-acquired smallpox virus infection in the United Kingdom in 1978 also were thought to be caused by airborne transmission. Airborne transmission may play a role in the natural spread of hantaviruses and certain hemorrhagic fever viruses (e.g., Ebola, Marburg, and Lassa), but evidence for airborne spread of these agents in health-care facilities is inconclusive. Although hantaviruses can be transmitted when aerosolized from rodent excreta, person-to-person spread of hantavirus infection from source patients has not occurred in health-care facilities. Lassa virus transmission via aerosols has been demonstrated in the laboratory and incriminated in health-care–associated infections in Africa, but airborne spread of this agent in hospitals in developed nations likely is inefficient. Yellow fever is considered to be a viral hemorrhagic fever agent with high aerosol infectivity potential, but health-care–associated transmission of this virus has not been described. Viral hemorrhagic fever diseases primarily occur after direct exposure to infected blood and body fluids, and the use of standard and droplet precautions prevents transmission early in the course of these illnesses. However, whether these viruses can persist in droplet nuclei that might remain after droplet production from coughs or vomiting in the latter stages of illness is unknown. Although the use of a negative-pressure room is not required during the early stages of illness, its use might be prudent at the time of hospitalization to avoid the need for subsequent patient transfer. Current CDC guidelines recommend negative-pressure rooms with anterooms for patients with hemorrhagic fever and use of HEPA respirators by persons entering these rooms when the patient has prominent cough, vomiting, diarrhea, or hemorrhage. Face shields or goggles will help to prevent mucous-membrane exposure to potentially-aerosolized infectious material in these situations. If an anteroom is not available, portable, industrial-grade high efficiency particulate air (HEPA) filter units can be used to provide the equivalent of additional air changes per hour (ACH).

Recommendations—

I. Air-Handling Systems in Health-Care Facilities

A. Use AIA guidelines as minimum standards where state or local regulations are not in place for design and construction of ventilation systems in new or renovated health-care facilities. Ensure that existing structures continue to meet the specifications in effect at the time of construction.

B. Monitor ventilation systems in accordance with engineers' and manufacturers' recommendations to ensure preventive engineering, optimal performance for removal of particulates, and elimination of excess moisture.

1. Ensure that heating, ventilation, air conditioning (HVAC) filters are properly installed and maintained to prevent air leakages and dust overloads.
2. Monitor areas with special ventilation requirements (e.g., AII or PE) for ACH, filtration, and pressure differentials.
   a. Develop and implement a maintenance schedule for ACH, pressure differentials, and filtration efficiencies using facility-specific data as part of the multidisciplinary risk assessment. Take into account the age and reliability of the system.
   b. Document these parameters, especially the pressure differentials.
3. Engineer humidity controls into the HVAC system and monitor the controls to ensure proper moisture removal.
   a. Locate duct humidifiers upstream from the final filters.
   b. Incorporate a water-removal mechanism into the system.
   c. Locate all duct takeoffs sufficiently down-stream from the humidifier so that moisture is completely absorbed.
4. Incorporate steam humidifiers, if possible, to reduce potential for microbial proliferation within the system, and avoid use of cool mist humidifiers. Category II
5. Ensure that air intakes and exhaust outlets are located properly in construction of new facilities and renovation of existing facilities.
   a. Locate exhaust outlets >25 ft. from air-intake systems.
   b. Locate outdoor air intakes >6 ft. above ground or >3 ft. above roof level.
   c. Locate exhaust outlets from contaminated areas above roof level to minimize recirculation of exhausted air.
6. Maintain air intakes and inspect filters periodically to ensure proper operation.
7. Bag dust-filled filters immediately upon removal to prevent dispersion of dust and fungal spores during transport within the facility.
   a. Seal or close the bag containing the discarded filter.
   b. Discard spent filters as regular solid waste, regardless of the area from which they were removed.
8. Remove bird roosts and nests near air intakes to prevent mites and fungal spores from entering the ventilation system.
9. Prevent dust accumulation by cleaning air-duct grilles in accordance with facility specific procedures and schedules when rooms are not occupied by patients.
10. Periodically measure output to monitor system function; clean ventilation ducts as part of routine HVAC maintenance to ensure optimum performance.

C. Use portable, industrial-grade HEPA filter units capable of filtration rates in the range of 300–800 ft3/min. to augment removal of respirable particles as needed. Category II 1. Select portable HEPA filters that can recirculate all or nearly all of the room air and provide the equivalent of >12 ACH.

2. Portable HEPA filter units previously placed in construction zones can be used later in patient-care areas, provided all internal and external surfaces are cleaned, and the filter's performance verified by appropriate particle testing.
3. Situate portable HEPA units with the advice of facility engineers to ensure that all room air is filtered.
4. Ensure that fresh-air requirements for the area are met.

D. Follow appropriate procedures for use of areas with through-the-wall ventilation units.
1. Do not use such areas as PE rooms.
2. Do not use a room with a through-the-wall ventilation unit as an AII room unless it can be demonstrated that all required AII engineering controls required are met.

E. Conduct an infection-control risk assessment (ICRA) and provide an adequate number of AII and PE rooms (if required) or other areas to meet the needs of the patient population.

F. When UVGI is used as a supplemental engineering control, install fixtures 1) on the wall near the ceiling or suspended from the ceiling as an upper air unit; 2) in the air-return duct of an AII room; or 3) in designated enclosed areas or booths for sputum induction.

G. Seal windows in buildings with centralized HVAC systems and especially with PE areas.

H. Keep emergency doors and exits from PE rooms closed except during an emergency; equip emergency doors and exits with alarms.

I. Develop a contingency plan for backup capacity in the event of a general power failure.713 Category IC (Joint Commission on Accreditation of Healthcare Organizations [JCAHO]: Environment of Care [EC]
1. Emphasize restoration of proper air quality and ventilation conditions in AII rooms, PE rooms, operating rooms, emergency departments, and intensive care units.
2. Deploy infection-control procedures to protect occupants until power and systems functions are restored.

J. Do not shut down HVAC systems in patient-care areas except for maintenance, repair, testing of emergency backup capacity, or new construction.
1. Coordinate HVAC system maintenance with infection-control staff to allow for relocation of immunocompromised patients if necessary.
2. Provide backup emergency power and air-handling and pressurization systems to maintain filtration, constant ACH, and pressure differentials in PE rooms, AII rooms, operating rooms, and other critical-care areas.
3. For areas not served by installed emergency ventilation and backup systems, use portable units and monitor ventilation parameters and patients in those areas.
4. Coordinate system startups with infection-control staff to protect patients in PE rooms from bursts of fungal spores.
5. Allow sufficient time for ACH to clean the air once the system is operational.

K. HVAC systems serving offices and administration areas may be shut down for energy conservation purposes, but the shutdown must not alter or adversely affect pressure differentials maintained in laboratories or critical-care areas with specific ventilation requirements (i.e., PE rooms, AII rooms, operating rooms).

L. Whenever possible, avoid inactivating or shutting down the entire HVAC system at one time, especially in acute-care facilities.

M. Whenever feasible, design and install fixed backup ventilation systems for new or renovated construction for PE rooms, AII rooms, operating rooms, and other critical care areas identified by ICRA.

II. Construction, Renovation, Remediation, Repair, and Demolition

A. Establish a multidisciplinary team that includes infection-control staff to coordinate demolition, construction, and renovation projects and consider proactive preventive measures at the inception; produce and maintain summary statements of the team's activities.

B. Educate both the construction team and the health-care staff in immunocompromised patient-care areas regarding the airborne infection risks associated with construction projects, dispersal of fungal spores during such activities, and methods to control the dissemination of fungal spores.

C. Incorporate mandatory adherence agreements for infection control into construction contracts, with penalties for noncompliance and mechanisms to ensure timely correction of problems.

D. Establish and maintain surveillance for airborne environmental disease (e.g., aspergillosis) as appropriate during construction, renovation, repair, and demolition activities to ensure the health and safety of immune-compromised patients.
1. Using active surveillance, monitor for airborne fungal infections in immune-compromised patients.
2. Periodically review the facility's microbiologic, histopathologic, and postmortem data to identify additional cases.
3. If cases of aspergillosis or other health-care–associated airborne fungal infections occur, aggressively pursue the diagnosis with tissue biopsies and cultures as feasible.

E. Implement infection-control measures relevant to construction, renovation, maintenance, demolition, and repair.
1. Before the project gets underway, perform an ICRA to define the scope of the project and the need for barrier measures.
   a. Determine if immunocompromised patients may be at risk for exposure to fungal spores from dust generated during the project.
   b. Develop a contingency plan to prevent such exposures.

2. Implement infection-control measures for external demolition and construction activities.
    a. Determine if the facility can operate temporarily on recirculated air; if feasible, seal off adjacent air intakes.
    b. If this is not possible or practical, check the low-efficiency (roughing) filter banks frequently and replace as needed to avoid buildup of particulates.
    c. Seal windows and reduce wherever possible other sources of outside air intrusion (e.g., open doors in stairwells and corridors), especially in PE areas.
3. Avoid damaging the underground water distribution system (i.e., buried pipes) to prevent soil and dust contamination of the water.
4. Implement infection-control measures for internal construction activities.
    a. Construct barriers to prevent dust from construction areas from entering patient-care areas; ensure that barriers are impermeable to fungal spores and in compliance with local fire codes. b. Block and seal off return air vents if rigid barriers are used for containment.
    c. Implement dust control measures on surfaces and by diverting pedestrian traffic away from work zones.
    d. Relocate patients whose rooms are adjacent to work zones, depending upon their immune status, the scope of the project, the potential for generation of dust or water aerosols, and the methods used to control these aerosols.
5. Perform those engineering and work-site related infection-control measures as needed for internal construction, repairs, and renovations.
    a. Ensure proper operation of the air-handling system in the affected area after erection of barriers and before the room or area is set to negative pressure.
    b. Create and maintain negative air pressure in work zones adjacent to patient-care areas and ensure that required engineering controls are maintained.
    c. Monitor negative air flow inside rigid barriers.
    d. Monitor barriers and ensure the integrity of the construction barriers; repair gaps or breaks in barrier joints.
    e. Seal windows in work zones if practical; use window chutes for disposal of large pieces of debris as needed, but ensure that the negative pressure differential for the area is maintained.
    f. Direct pedestrian traffic from construction zones away from patient-care areas to minimize the dispersion of dust.
    g. Provide construction crews with 1) designated entrances, corridors, and elevators whenever practical; 2) essential services [e.g., toilet facilities], and convenience services [e.g., vending machines]; 3) protective clothing [e.g., coveralls, footgear, and headgear] for travel to patient-care areas; and 4) a space or anteroom for changing clothing and storing equipment.
    h. Clean work zones and their entrances daily by 1) wet-wiping tools and tool carts before their removal from the work zone; 2) placing mats with tacky surfaces inside the entrance; and 3) covering debris and securing this covering before removing debris from the work zone. i. In patient-care areas, for major repairs that include removal of ceiling tiles and disruption of the space above the false ceiling, use plastic sheets or prefabricated plastic units to contain dust; use a negative pressure system within this enclosure to remove dust; and either pass air through an industrial grade, portable HEPA filter capable of filtration rates ranging from 300–800 ft3/min., or exhaust air directly to the outside. Upon completion of the project, clean the work zone according to facility procedures, and install barrier curtains to contain dust and debris before removal of rigid barriers.
    k. Flush the water system to clear sediment from pipes to minimize waterborne microorganism proliferation.
    l. Restore appropriate ACH, humidity, and pressure differential; clean or replace air filters; dispose of spent filters.

F. Use airborne-particle sampling as a tool to evaluate barrier integrity.

G. Commission the HVAC system for newly constructed health-care facilities and renovated spaces before occupancy and use, with emphasis on ensuring proper ventilation for operating rooms.

H. No recommendation is offered on routine microbiologic air sampling before, during, or after construction or before or during occupancy of areas housing immune-compromised patients. If a case of health-care–acquired aspergillosis or other opportunistic environmental airborne fungal disease occurs during or immediately after construction, implement appropriate follow-up measures.
    1. Review pressure differential monitoring documentation to verify that pressure differentials in the construction zone and in PE rooms were appropriate for their settings.
    2. Implement corrective engineering measures to restore proper pressure differentials as needed.
    3. Conduct a prospective search for additional cases and intensify retrospective epidemiologic review of the hospital's medical and laboratory records.
    4. If there is no evidence of ongoing transmission, continue routine maintenance in the area to prevent health-care–acquired fungal disease.

J. If there is epidemiologic evidence of ongoing transmission of fungal disease, conduct an environmental assessment to determine and eliminate the source.
    1. Collect environmental samples from potential sources of airborne fungal spores, preferably using a high-volume air sampler rather than settle plates.

2. If either an environmental source of airborne fungi or an engineering problem with filtration or pressure differentials is identified, promptly perform corrective measures to eliminate the source and route of entry.
3. Use an EPA-registered anti-fungal biocide (e.g., copper-8-quinolinolate) for decontaminating structural materials.
4. If an environmental source of airborne fungi is not identified, review infection control measures, including engineering controls, to identify potential areas for correction or improvement.
5. If possible, perform molecular subtyping of Aspergillus spp. isolated from patients and the environment to establish strain identities.

K. If air-supply systems to high-risk areas (e.g., PE rooms) are not optimal, use portable, industrial-grade HEPA filters on a temporary basis until rooms with optimal air-handling systems become available.

III. Infection-Control and Ventilation Requirements for PE Rooms

A. Minimize exposures of severely immunocompromised patients (e.g., solid organ transplant patients or allogeneic neutropenic patients) to activities that might cause aerosolization of fungal spores (e.g., vacuuming or disruption of ceiling tiles).

B. Minimize the length of time that immunocompromised patients in PE are outside their rooms for diagnostic procedures and other activities.

C. Provide respiratory protection for severely immune-compromised patients when they must leave PE for diagnostic studies and other activities; consult the most recent revision of CDC's Guidelines for Prevention of Health-Care–Associated Pneumonia for information regarding the appropriate type of respiratory protection.

Incorporate ventilation engineering specifications and dust-controlling processes into the planning and construction of new PE units.

1. Install central or point-of-use HEPA filters for supply (incoming) air.
2. Ensure that rooms are well sealed by 1) properly constructing windows, doors, and intake and exhaust ports; 2) maintaining ceilings that are smooth and free of fissures, open joints, and crevices; 3) sealing walls above and below the ceiling, and 4) monitoring for leakage and making necessary repairs.
3. Ventilate the room to maintain >12 ACH.
4. Locate air supply and exhaust grilles so that clean, filtered air enters from one side of the room, flows across the patient's bed, and exits from the opposite side of the room.
5. Maintain positive room air pressure (>2.5 Pa [0.01-inch water gauge]) in relation to the corridor.
6. Maintain airflow patterns and monitor these on a daily basis by using permanently installed visual means of detecting airflow in new or renovated construction, or using other visual methods (e.g., flutter strips, or smoke tubes) in existing PE units. Document the monitoring results.
7. Install self-closing devices on all room exit doors in protective environments.

E. Do not use laminar air flow systems in newly constructed PE rooms.

F. Take measures to protect immunocompromised patients who would benefit from a PE room and who also have an airborne infectious disease (e.g., acute VZV infection or tuberculosis).

1. Ensure that the patient's room is designed to maintain positive pressure.
2. Use an anteroom to ensure appropriate air balance relationships and provide independent exhaust of contaminated air to the outside, or place a HEPA filter in the exhaust duct if the return air must be recirculated.
3. If an anteroom is not available, place the patient in AII and use portable, industrial grade HEPA filters to enhance filtration of spores in the room.

G. Maintain backup ventilation equipment (e.g., portable units for fans or filters) for emergency provision of ventilation requirements for PE areas and take immediate steps to restore the fixed ventilation system function.

IV. Infection-Control and Ventilation Requirements for AII Rooms

A. Incorporate certain specifications into the planning, and construction or renovation of AII units.

1. Maintain continuous negative air pressure (2.5 Pa [0.01-inch water gauge]) in relation to the air pressure in the corridor; monitor air pressure periodically, preferably daily, with audible manometers or smoke tubes at the door (for existing AII rooms) or with a permanently installed visual monitoring mechanism. Document the results of monitoring.
2. Ensure that rooms are well-sealed by properly constructing windows, doors, and air intake and exhaust ports; when monitoring indicates air leakage, locate the leak and make necessary repairs.
3. Install self-closing devices on all AII room exit doors.
4. Provide ventilation to ensure >12 ACH for renovated rooms and new rooms, and >6 ACH for existing AII rooms. Direct exhaust air to the outside, away from air-intake and populated areas. If this is not practical, air from the room can be recirculated after passing through a HEPA filter.4, 120 Category IC.

B. Where supplemental engineering controls for air cleaning are indicated from a risk assessment of the AII area, install UVGI units in the exhaust air ducts of the HVAC system to supplement HEPA filtration or install UVGI fixtures on or near the ceiling to irradiate upper room air.4 Category II.

C. Implement environmental infection-control measures for persons with known or suspected airborne infectious diseases.

1. Use AII rooms for patients with or suspected of having an airborne infection who also require cough-inducing procedures, or use an enclosed booth that is engineered to provide 1>12 ACH; 2) air supply and exhaust rate sufficient to maintain a 2.5 Pa [0.01-inch water gauge] negative pressure difference with respect to all surrounding spaces with an exhaust rate of >50 ft3/min.; and 3) air exhausted directly outside away from air intakes and traffic or exhausted after HEPA filtration prior to recirculation.
2. Although airborne spread of viral hemorrhagic fever (VHF) has not been documented in a health-care setting, prudence dictates placing a VHF patient in an AII room, preferably with an anteroom to reduce the risk of occupational exposure to aerosolized infectious material in blood, vomitus, liquid stool, and respiratory secretions present in large amounts during the end stage of a patient's illness.

a. If an anteroom is not available, use portable, industrial-grade HEPA filters in the patient's room to provide additional ACH equivalents for removing airborne particulates.

b. Ensure that health-care workers wear face shields or goggles with appropriate respirators when entering the rooms of VHF patients with prominent cough, vomiting, diarrhea, or hemorrhage.

3. Place smallpox patients in negative pressure rooms at the onset of their illness, preferably using a room with an anteroom if available.

D. No recommendation is offered regarding negative pressure or isolation rooms for patients with Pneumocystis carinii pneumonia.

E. Maintain back-up ventilation equipment (e.g., portable units for fans or filters) for emergency provision of ventilation requirements for AII rooms and take immediate steps to restore the fixed ventilation system function.

V. Infection-Control and Ventilation Requirements for Operating Rooms

A. Implement environmental infection-control and ventilation measures for operating rooms.

1. Maintain positive-pressure ventilation with respect to corridors and adjacent areas.
2. Maintain >15 ACH, of which >3 ACH should be fresh air.
3. Filter all recirculated and fresh air through the appropriate filters, providing 90% efficiency (dust-spot testing) at a minimum.
4. In rooms not engineered for horizontal laminar airflow, introduce air at the ceiling and exhaust air near the floor.
5. Do not use UV lights to prevent surgical-site infections.
6. Keep operating room doors closed except for the passage of equipment, personnel, and patients, and limit entry to essential personnel.

B. Follow precautionary procedures for TB patients who also require emergency surgery. Use an N95 respirator approved by the National Institute for Occupational Safety and Health (NIOSH) without exhalation valves in the operating room. (Occupational Safety and Health Administration [OSHA]; 29 Code of Federal Regulations [CFR].

2. Intubate the patient in either the AII room or the operating room; if intubating the patient in the operating room, do not allow the doors to open until 99% of the airborne contaminants are removed (Appendix B, Table B.1).4, 358 Category IB
3. When anesthetizing a patient with confirmed or suspected TB, place a bacterial filter between the anesthesia circuit and patient's airway to prevent contamination of anesthesia equipment or discharge of tubercle bacilli into the ambient air.
4. Extubate and allow the patient to recover in an AII room.4, 358 Category IB
5. If the patient has to be extubated in the operating room, allow adequate time for ACH to clean 99% of airborne particles from the air (Appendix B, Table B.1) because extubation is a cough-producing procedure.

C. Use portable, industrial-grade HEPA filters temporarily for supplemental air cleaning during intubation and extubation for infectious TB patients who require surgery.

1. Position the units appropriately so that all room air passes through the filter; obtain engineering consultation to determine the appropriate placement of the unit.
2. Switch the portable unit off during the surgical procedure.
3. Provide fresh air as per ventilation standards for operating rooms; portable units do not meet the requirements for the number of fresh ACH.

D. If possible, schedule infectious TB patients as the last surgical cases of the day to maximize the time available for removal of airborne contamination.

E. No recommendation is offered for performing orthopedic implant operations in rooms supplied with laminar airflow.

F. Maintain backup ventilation equipment (e.g., portable units for fans or filters) for emergency provision of ventilation requirements for operating rooms, and take immediate steps to restore the fixed ventilation system function.

VI. Other Potential Infectious Aerosol Hazards in Health-Care Facilities.

A. In settings where surgical lasers are used, wear appropriate personal protective equipment, including N95 or N100 respirators, to minimize exposure to laser plumes.

B. Use central wall suction units with in-line filters to evacuate minimal laser plumes.

C. Use a mechanical smoke evacuation system with a high-efficiency filter to manage the generation of large amounts of laser plume, when ablating tissue infected with human papilloma virus (HPV) or performing procedures on a patient with extrapulmonary TB.

Vibrio Illness (Vibriosis)

Vibrio is a genus of Gram-negative bacteria possessing a curved-rod shape (comma shape), several species of which can cause foodborne infection, usually associated with eating undercooked seafood. Typically found in saltwater, *Vibrio* species are facultative anaerobes that test positive for oxidase and do not form spores. All members of the genus are motile and have polar flagella with sheaths.

Several species of *Vibrio* are pathogens.[6] Most disease-causing strains are associated with gastroenteritis, but can also infect open wounds and cause septicemia. They can be carried by numerous marine animals, such as crabs or prawns, and have been known to cause fatal infections in humans during exposure. Pathogenic *Vibrio* species include *V. cholerae* (the causative agent of cholera), *V. parahaemolyticus*, and *V. vulnificus.*

Vibrios are bacteria that occur naturally in estuarine and marine waters worldwide.*Vibrio* are in the same family of bacteria that cause cholera. There are over 80 species of *Vibrio* bacteria. *Vibrio vulnificus, Vibrio parahaemolyticus, Vibrio fluvialis, Vibrio mimicus,* and *Vibrio alginolyticus* are some of the species known to cause infection when people are exposed by open wounds or punctures that occur while swimming, wading, crabbing, or fishing. Not all strains of *Vibrios*cause human illness.

Vibrios are naturally-occurring bacteria, and pollution is not considered a factor for finding these bacteria in surface waters. The most significant factors for finding them in surface waters are temperature, salinity (a measure of how much salt is in the water), and chlorophyll. *Vibrio* bacteria are not commonly found in the winter when water temperatures are low, but may be common in the summer and early fall when water temperatures are warm.

Vibrio vulnificus (V. vulnificus) and *Vibrio parahaemolyticus (V. parahaemolyticus)* are bacteria that occur naturally in warm coastal areas, such as the Gulf of Mexico. These bacteria are found in higher concentrations in the summer months when water gets warmer.

Vibriosis is a disease caused by an infection with bacteria of the *Vibrio* genus, most commonly *Vibrio parahemolyticus* or *Vibrio vulnificus.*

Vibrios typically cause disease in people who eat contaminated seafood.

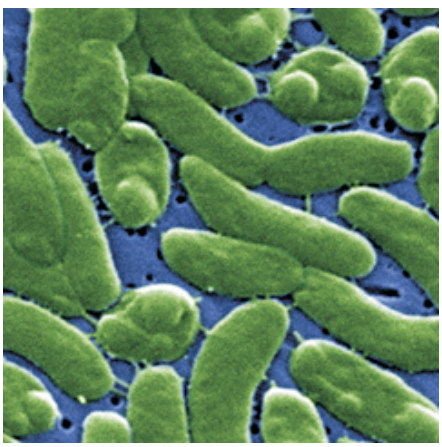
- *V. parahaemolyticus* typically causes non-bloody diarrhea.
- In persons with liver disease, cancer, or another immune-compromising condition, *V. vulnificus* typically infects the bloodstream, causing a life-threatening illness. About half of *V. vulnificus* bloodstream infections are fatal, and death can occur within two days. In addition to transmission by raw shellfish, *V. vulnificus* can enter the body via a wound that is exposed to warm seawater.

Description

Vibriosis is a general term referring to an infection by any member of the large group of *Vibrio*, bacteria.

Alternate names include non-cholera *Vibrio* infection, *Vibrio parahemolyticus* infection, and *Vibrio vulnificus* infection. Non-cholera Vibrio species are uncommon but important enteric bacterial pathogens, causing an estimated 8,000 infections, 185 hospitalizations, and 57 deaths in the United States (US) each year. Vibrio species are natural inhabitants of marine coastal and estuarine environments, and their populations increase dramatically during the warm summer months. In the US, V. *parahaemolyticus* is the most commonly reported Vibrio infection, but V. *vulnificus* is associated with severe morbidity and mortality.

Consuming raw, undercooked, or cross-contaminated seafood, especially shellfish, is the most common cause of non-cholera vibriosis, but exposing wounds to contaminated warm seawater can also cause skin or soft tissue Vibrio infection. There is no national Healthy People 2010 target objective for non-cholera vibriosis.



Vibrio parahaemolyticus is a bacterium in the same family as those that cause cholera and *Vibrio vulnificus*. It lives in brackish saltwater and causes gastrointestinal illness in humans. *V. parahaemolyticus* naturally inhabits coastal waters in the United States and Canada and is present in higher concentrations during summer; it is a halophilic, or salt-requiring organism.

V. *parahaemolyticus* infection causes acute gastroenteritis with fever that usually occurs after an incubation period of 24 hours. Symptoms usually last 1 to 7 days and are often self-limited. Symptoms of *Vibrio parahaemolyticus* infection may include:

- Watery diarrhea
- Abdominal cramps
- Nausea
- Vomiting
- Fever
- Headache
- Bloody diarrhea

In contrast, V. *vulnificus* causes septicemia in persons with immunocompromising conditions, chronic liver disease, and alcoholism. Fifty percent of such patients with septicemia die, and the case-fatality rate exceeds 90% among patients who become hypotensive.

Raw oysters are the usual source, although other seafood can carry the bacteria.

Vibrio parahemolyticus causes severe diarrhea. *Vibrio vulnificus* may cause diarrhea, but in persons with an underlying disease it may cause severe blood infections (septicemia or blood poisoning). V. parahaemolyticus and V. vulnificus generally are not passed person-to-person. Contact of a wound with seawater or contaminated seafood can lead to a *Vibrio vulnificus* skin infection.

Exposure to contaminated water during natural disasters such as

hurricanes has resulted in wound infections. Transmission of infection person to person has not been reported. People with liver disease, low gastric acidity, and immunodeficiency have increased susceptibility to infection with Vibrio species.

Causes and symptoms

Vibriosis is caused by eating seafood contaminated with *Vibrio parahemolyticus* or *Vibrio vulnificus*. These bacteria damage the inner wall of the intestine, which causes diarrhea and related symptoms. *Vibrio vulnificus* can get through the intestinal wall and into the bloodstream. Persons at risk for severe, often fatal vibriosis include those with liver disease (cirrhosis), excess iron (hemochromatosis), thalassemia (a blood disorder), **AIDS**, diabetes, or those who are immunosuppressed.

Symptoms of intestinal infection occur within two days of eating contaminated seafood. Symptoms last for two to 10 days and include watery diarrhea, abdominal cramps, nausea, vomiting, **headache**, and possibly **fever**. Symptoms of a blood infection develop one to two days after eating contaminated seafood, and include fever, chills, low blood pressure, and large fluid-filled blisters on the arms or legs. Similar blisters can also be produced by a *Vibrio vulnificus* skin infection.

An estimated 4500 cases of *V. parahaemolyticus* infection occur each year in the United States. However, the number of cases reported to CDC is much lower because surveillance is complicated by underreporting. Laboratories rarely use the selective medium that is necessary to identify this organism, and it is likely that many cases are undetected. To improve our ability to monitor trends, infections caused by *V. parahaemolyticus* and other *Vibrio* species became nationally notifiable in 2007. State health departments report cases to CDC, and these reports are summarized annually.

Vibrio organisms can be isolated from cultures of stool, wound, or blood. For isolation from stool, use of a selective medium that has thiosulfate, citrate, bile salts, and sucrose (TCBS agar) is recommended. If there is clinical suspicion for infection with this organism, the microbiology laboratory should be notified so that they will perform cultures using this medium. A physician should suspect *V. parahaemolyticus* infection if a patient has watery diarrhea and has eaten raw or undercooked seafood, especially oysters, or when a wound infection occurs after exposure to seawater.

Treatment is not necessary in most cases of *V. parahaemolyticus* infection. There is no evidence that antibiotic treatment decreases the severity or the length of the illness. Patients should drink plenty of liquids to replace fluids lost through diarrhea. In severe or prolonged illnesses, antibiotics such as tetracycline or ciprofloxicin can be used. The choice of antibiotics should be based on antimicrobial susceptibilities of the organism.

Most infections caused by *V. parahaemolyticus* in the United States can be prevented by thoroughly cooking seafood, especially oysters. Wound infections can be prevented by avoiding exposure of open wounds to warm seawater. When an outbreak is traced to an oyster bed, health officials recommend closing the oyster bed until conditions are less favorable for *V. parahaemolyticus*.

Timely, voluntary reporting of *V. parahaemolyticus* infections to state health departments and to regional offices of the Food and Drug Administration (FDA) will help collaborative efforts to improve investigation of these infections. Regional FDA specialists with expert knowledge about shellfish assist state officials with tracebacks of shellfish. When notified rapidly about cases, officials can sample harvest waters to discover possible sources of infection and may close oyster beds. Ongoing research may help us to predict environmental or other factors that increase the chance that oysters carry *Vibrios*.

Prevention

Contamination with *Vibrio* bacteria does not change the look, smell, or taste of the seafood.

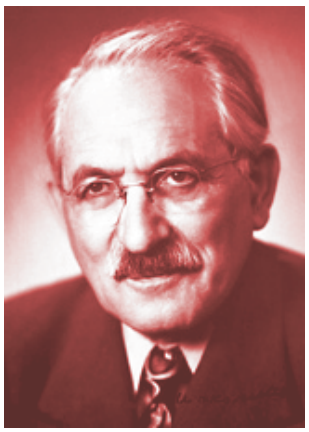
Here are some tips for preventing Vibrio species (non-cholera) infections, particularly among immunocompromised patients, including those with underlying liver disease:

- Do not eat raw oysters or other raw shellfish.
- Cook shellfish (oysters, clams, mussels) thoroughly.
- For shellfish in the shell, either a) boil until the shells open and continue boiling for 5 more minutes, or b) steam until the shells open and then continue cooking for 9 more minutes. Do not eat those shellfish that do not open during cooking.
- Boil shucked oysters at least 3 minutes or fry them in oil at least 10 minutes at 375°F.
- Avoid cross-contamination of cooked seafood and other foods with raw seafood and juices from raw seafood.
- Eat shellfish promptly after cooking and refrigerate leftovers.
- Avoid exposure of open wounds or broken skin to warm salt or brackish water and raw shellfish/seafood drippings.
- Wear protective clothing (e.g., gloves) when handling raw shellfish.

Reference:

1. Austin B. Vibrios as causal agents of zoonoses. Vet Microbiol 2009; 140:310-7.
2. Altekruse SF, Bishop RD, Baldy LM, et al. *Vibrio* gastroenteritis in the US Gulf of Mexico region: the role of raw oysters. Epidemiol Infect 2000;124:489-95.
3. Daniels NA, MacKinnon L, Bishop R, et al. *Vibrio* parahaemolyticus infections in the United States, 1973–1998. J Infect Dis 2000;181:1661-6.
4. Dechet AM, Yu PA, Koram N, Painter J. Nonfoodborne Vibrio infections: an important cause of morbidity and mortality in the United States, 1997–2006. Clin Infect Dis 2008;46:970-6.
5. Horseman MA, Surani S. A comprehensive review of *Vibrio* vulnificus: an important cause of severe sepsis and skin and soft-tissue infection. Int J Infect Dis 2010;15:e157-66.
6. Pickering LK, Baker C, Kimberlin DW, Long SS, editors. Red book: 2009 report of the committee on infectious diseases. 28th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2009. American Academy of Pediatrics. Other vibrio infections. In:; p. 729-30.
7. Scallan E, Hoekstra RM, Angulo FJ, et al. Foodborne illness acquired in the United States—major athogens. Emerg Infect Dis 2011 Jan; 17(1):7-15.
8. Tan, Wen-Si; Muhamad Yunos, Nina Yusrina; Tan, Pui-Wan; Mohamad, Nur Izzati; Adrian, Tan-Guan-Sheng; Yin, Wai-Fong; Chan, Kok-Gan (8 July 2014). "Characterisation of a Marine Bacterium Vibrio Brasiliensis T33 Producing N-acyl Homoserine Lactone Quorum Sensing Molecules". *Sensors* **14** (7): 12104–12113.
9. Thompson FL, Gevers D, Thompson CC, Dawyndt P, Naser S, Hoste B, Munn CB, Swings J (2005). "Phylogeny and Molecular Identification of Vibrios on the Basis of Multilocus Sequence Analysis". *Applied and Environmental Microbiology* **71** (9): 5107–5115.

Selman A. Waksman



Born
Selman Abraham Waksman July 22, 1888 Nova Pryluka, near Kiev, Ukraine, Russian Empire
Died
August 16, 1973 (aged 85) Woods Hole, Barnstable County, Massachusetts, United States
Residence
Woods Hole, Barnstable County, Massachusetts, United States

Citizenship
United States of America (After 1916)
Fields
Biochemistry and Microbiology
Notable awards
Nobel Prize in Physiology or Medicine (1952)
Leeuwenhoek Medal (1950)
Spouse
Deborah B. Mitnik (1 child) (died 1974)

Selman Abraham Waksman was born in Priluka, near Kiev, Russia, on July 22nd, 1888, as the son of Jacob Waksman and Fradia London. He received his early education primarily from private tutors, and completed his school training in Odessa in an evening school and with private tutors. He obtained his matriculation diploma in 1910 from the Fifth Gymnasium in Odessa as an extern, and left for the United States immediately afterwards.

In the autumn of 1911 he entered Rutgers College, having won a State Scholarship the previous spring. He received his B.Sc. degree in Agriculture from Rutgers in 1915. He was then appointed research assistant in soil bacteriology under Dr. J. G. Lipman at the New Jersey Agricultural Experiment Station, and was allowed to continue graduate work at Rutgers, obtaining his M.Sc. degree in 1916. In the same year, he became a naturalized United States citizen and was appointed a Research Fellow at the University of California where he received his Ph.D. in Biochemistry in 1918.

He was invited by Dr. Lipman to return to Rutgers, where he received an appointment as microbiologist at the Experiment Station and as Lecturer in Soil Microbiology at the University. He was appointed Associate Professor in 1925 and Professor in 1930. When the Department of Microbiology was organized in 1940, he became Professor of Microbiology and Head of the Department. In 1949, he was appointed Director of the Institute of Microbiology. He retired in 1958. However, he has a laboratory and office at the Institute to continue a limited amount of research and considerable writing and lecturing.

Apart from his activities at Rutgers, he was invited to organize a division of Marine Bacteriology at the Woods Hole Oceanographic Institution in 1931; he was also appointed marine bacteriologist at the same institution, where he served until 1942. He was then elected as a Trustee, and later a Life Trustee. On various occasions, he held industrial positions for limited periods of time and served as consultant to industrial laboratories, government and other scientific institutions.

Professor Waksman's fields of work include, in chronological order, the microbiological population of the soil, sulphur oxidation by bacteria, microorganisms and soil fertility; decomposition of plant and animal residues, nature and formation of humus; occurrence of bacteria in the sea and their role in marine processes; production and nature of antibiotic substances; taxonomy, physiology, and biochemistry of the actinomycetes. He has published more than 400 scientific papers and has written, alone or with others, 18 books.

He has isolated, together with his students and associates, a number of new antibiotics, including actinomycin (1940), clavacin, streptothricin (1942), streptomycin (1943), grisein (1946), neomycin (1948), fradicin, candicidin, candidin, and others. Two of these, streptomycin and neomycin, have found extensive application in the treatment of numerous infectious diseases of men, animals and plants. They have been covered by patents, that on streptomycin having been recently listed as one of the ten «patents that shaped the world».

Professor Waksman holds honorary doctor's degrees in medicine, science, agriculture, law or letters from the Universities of Liège, Athens, Pavia, Madrid, Strasbourg, Jerusalem, Göttingen, Perugia, Keio (Japan) and several American universities and colleges. He is a member, honorary member or fellow of a number of scientific societies in the USA, France, Sweden, Mexico, India, Germany, Brazil, Spain, and Israel. He is a Former President of the American Society for Microbiology.

His work in the field of microbiology has been recognized by numerous scientific and other societies in the USA, Denmark, The Netherlands, Canada, Sweden, Japan, Israel, Italy, Spain, and Turkey. In 1950 he was made Commander of the French Légion d'Honneur, and in 1952 he was voted as one of «the most outstanding 100 people in the world today» (Little, Brown & Co.).

In 1949, the Trustees of Rutgers University voted to establish an Institute of Microbiology and made Professor Waksman its first Director. The larger portion of the funds derived from the royalties obtained from streptomycin and neomycin have been assigned for the building and support of this Institute, which is being used for research and advanced teaching on a doctorate and post-doctorate level in microbiology. Out of the small portion of the royalties assigned to him personally, Dr. and Mrs. Waksman established the «Foundation for Microbiology», for the support of research and publications in the field of microbiology at various institutions of the world. Professor Waksman continues as President of this Foundation. He and his wife have also established a scholarship for an immigrant student, or the son or daughter of an immigrant, at Rutgers University, and Mrs. Waksman has established a music scholarship at Douglass College, Rutgers University.

Professor Waksman's wife is Deborah B. Mitnik. They have one son, Byron H. Waksman, M.D., who was a Research Associate at Massachusetts General Hospital, Boston, and Assistant Professor at Harvard University Medical School, and more recently Professor of Microbiology at Yale University Medical School, and two grandchildren, Nan and Peter.

JOKES

In bio practical:
Examiner: Tell me the name of this bird by seeing its legs only?
Sardar: I don't know.
Examiner: You are failed, what's your name?
Sardar: See my legs & tell my name

The Santa Claus at the mall was very surprised when a young lady about twenty years old walked up and sat on his lap. Doesn't usually take requests from adults, but she smiled very nicely at him, he asked her, "What do you want for Christmas?" "Something for my mother, please." said the young lady. "Something for your mother? Well, that's very thoughtful of you," smiled Santa. "What do you want me to bring her?" Without blinking she replied, "A son-in-law!"

Wife: You always carry my photo in your handbag to the office. Why?
Darling : When there is a problem, no matter how impossible, I look at your picture and the problem disappears.
Wife: You see, how miraculous and powerful I am for you?
Darling : Yes, I see your picture and say to myself, "What other problem Can there be greater than this one?

Having "WIFE" Is A
Part Of Living...
But
Having "GIRLFRIEND"
Along With The "WIFE" Is
Art Of Living

Wife: honey, what r u looking 4?
Husband: nothing
Wife: why have u been reading our marriage certificate 4 an hour ?
Husband: i was just looking 4 the expiry date

A woman went shopping. At cash counter she opened her purse to pay.
The cashier noticed a TV remote in her purse.
He cud'nt control his curiosity n asked "Do u always carry ur TV remote with u?"
She replied " No, not always, but my husband refused to accompany me for shopping today..
The story continues....
The shopkeeper laughs and takes back all the items that lady had purchased.
Shocked at this act, she asks the shopkeeper what is he doing.
He said your husband has blocked your credit card.
MORAL : Respect the hobbies of your husband.
Story continues....
Wife took out his husbands credit card from purse and uses it to clear all the bills.
Unfortunately he didn't block his own card.
Moral:..... Dont underestimate the power of a WIFE.

A man received message from his neighbour.
Sorry sir I am using your wife.
I am using day and night.
I am using when u r not present at home.
In fact I am using more than U R using.
I confess this because now I feel very much guilt.
Hope U will accept my sincere apologies.
Man went home and had a big fight with his wife.
Few minutes later he received another message.
Sorry Sir spelling / auto correct mistake ...
it's not wife but WIFI.

Man outside phone booth: Excuse me !!
You are holding the phone since 20 mins.
& haven't spoken a word..!!!
Man inside: I'm talking to my wife

A famous inspirational speaker said:
"Best years of my life were spent in the arms of a woman, who wasn't my wife"
Audience was in shock and silence..
He added: "she was my mother"
A big round of applause & laughter!
A very daring husband tried to crack this at home
After a dinner, he said loudly to his wife in the kitchen:
"Best years of my life were spent in the arms of a woman, who wasn't my wife"
standing for a moment, trying to recall the second line of that speaker
by the time he gained his senses,
he was on a hospital bed,
recovering from burns of boiling water!
Moral: don't copy if u can't paste!

A successful man is one who makes more money than his wife can spend.
A successful woman is one who can find such a man.

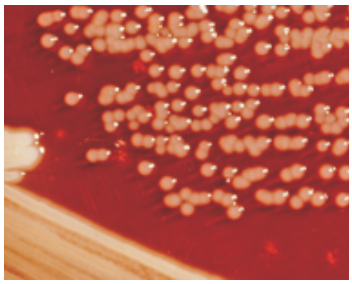
True saying....
Women never dress up to impress man,
She dress up to irritate other women.

Husband : I found Aladin's lamp today.
Wife : wow, what did u ask for darling ??
Husband : I asked him to increase your brain ten times..
Wife : oh..darling..luv u so much.. :-*
Did he do that ??
Husband : He laughed and said multiplication doesn't apply on zero.

Position of husband is like a split A.C.
No matter how loud he is outside,
but inside the house,
he is designed to remain silent, cool & controlled by remote.

A man in Hell asked Devil:
Can I make a call to my Wife?
After making call he asked how much to pay.
Devil : Nothing, Hell to hell is Free.

Yersinia enterocolitica



Kingdom: Bacteria
Phylum: Proteobacteria
Class: Gamma Proteobacteria
Order: Enterobacteriales
Family: Enterobacteriaceae
Genus: Yersinia
Species: *Y. enterocolitica*

Yersinia enterocolitica is a Gram-negative bacillus-shaped bacterium, belonging to the family Enterobacteriaceae. *Y. enterocolitica* infection causes the disease yersiniosis, which is a zoonotic disease occurring in humans, as well as a wide array of animals such as cattle, deer, pigs, and birds. Many of these animals recover from the disease and become asymptomatic carriers. It infects the host by sticking to the cells of the host using trimeric autotransporter adhesins (TAAs).

The genus *Yersinia* includes 11 species: *Y. pestis, Y. pseudotuberculosis, Y. enterocolitica, Y. frederiksenii, Y. intermedia, Y. kristensenii, Y. bercovieri, Y. mollaretii, Y. rohdei, Y. aldovae* and *Y. ruckeri*. Among them, only *Y. pestis, Y. pseudotuberculosis*, and certain strains of *Y. enterocolitica* are of pathogenic importance for humans and certain warm-blooded animals, whereas the other species are of environmental origin and may, at best, act as opportunists. However, *Yersinia* strains can be isolated from clinical materials, so have to be identified at the species level.

Y. enterocolitica is a heterogeneous group of strains, which are traditionally classified by biotyping into six biogroups on the basis of phenotypic characteristics, and by serotyping into more than 57 O serogroups, on the basis of their O (lipopolysaccharide or LPS) surface antigen. Five of the six biogroups (1B and 2–5) are regarded as pathogens. However, only a few of these serogroups have been associated with disease in either humans or animals. Strains that belong to serogroups O:3 (biogroup 4), O:5,27 (biogroups 2 and 3), O:8 (biogroup 1B), and O:9 (biogroup 2) are most frequently isolated worldwide from human samples. However, the most important *Y. enterocolitica* serogroup in many European countries is serogroup O:3 followed by O:9, whereas the serogroup O:8 is mainly detected in the United States.

Y. enterocolitica is widespread in nature, occurring in reservoirs ranging from the intestinal tracts of numerous mammals, avian species, cold-blooded species, and even from terrestrial and aquatic niches. Most environmental isolates are avirulent; however, isolates recovered from porcine sources contain human pathogenic serogroups. In addition, dogs, sheep, wild rodents, and environmental water may also be a reservoir of pathogenic *Y. enterocolitica* strains. Human pathogenic strains are usually confined to the intestinal tract and lead to enteritis/diarrhea.

Signs and symptoms

The portal of entry is the gastrointestinal tract. The organism is acquired usually by insufficiently cooked pork or contaminated water, meat, or milk. Acute *Y. enterocolitica* infections usually lead to mild self-limiting enterocolitis or terminal ileitis and adenitis in humans. Symptoms may include watery or bloody diarrhea and fever, resembling appendicitis or salmonellosis or shigellosis. After oral uptake, *Yersinia* species replicate in the terminal ileum and invade Peyer's patches. From here they can disseminate further to mesenteric lymph nodes causing lymphadenopathy. This condition can be confused with appendicitis, so is called pseudoappendicitis. In immunosuppressed individuals, they can disseminate from the gut to liver and spleen and form abscesses. Because *Yersinia* species are siderophilic (iron-loving) bacteria, people with hereditary hemochromatosis (a disease resulting in high body iron levels) are more susceptible to infection with *Yersinia* (and other siderophilic bacteria). In fact, the most common contaminant of stored blood is *Y. enterocolitica*.

Treatment

Yersiniosis is usually self-limiting and does not require treatment. For severe infections (sepsis, focal infection) especially if associated with immunosuppression, the recommended regimen includes doxycycline in combination with an aminoglycoside. Other antibiotics active against *Y. enterocolitica* include trimethoprim-sulfamethoxasole, fluoroquinolones, ceftriaxone, and chloramphenicol. *Y. enterocolitica* is usually resistant to penicillin G, ampicillin, and cephalotin due to beta-lactamase production.

Prognosis

Y. enterocolitica infections are sometimes followed by chronic inflammatory diseases such as arthritis, erythema nodosum, and reactive arthritis. This is most likely because of some immune-mediated mechanism.

Y. enterocolitica seems to be associated with autoimmune Graves-Basedow thyroiditis. Whilst indirect evidence exists, direct causative evidence is limited, and *Y. enterocolitica* is probably not a major cause of this disease, but may contribute to the development of thyroid autoimmunity arising for other reasons in genetically susceptible individuals. *Y. enterocolitica* infection has also been suggested to not be the cause of autoimmune thyroid disease, but rather is only an associated condition, with both having a shared inherited susceptibility. More recently, the role for *Y. enterocolitica* has been disputed.

Prevention

To reduce the risk of yersiniosis, take these precautions:

- Don't serve or eat raw or undercooked meat.
- Drink and serve only pasteurized milk and milk products.
- Wash hands with soap and water particularly before eating and preparing food; before touching infants or their toys, bottles, or pacifiers; and after contact with animals or handling raw meat.
- Use separate cutting boards for meat and other foods.
- Clean all cutting boards, countertops, and utensils with soap and hot water after preparing raw meat.
- Always cook meat thoroughly before you eat it, especially pork products.
- Dispose of animal feces and sanitize anything they have touched.
- Avoid drinking directly from natural water sources such as ponds and mountain streams, particularly if the water is near farmland where cattle, pigs, or goats are raised.
- As you care for a family member who has diarrhea, remember to wash your hands thoroughly before touching other people and before handling food.

If your pet dog or cat has diarrhea, wash your hands frequently as you care for it, and check with your veterinarian about treatment and/or contagiousness.

Common bacteria on verge of becoming antibiotic-resistant superbugs

Antibiotic resistance is poised to spread globally among bacteria frequently implicated in respiratory and urinary infections in hospital settings, according to new research at Washington University School of Medicine in St. Louis.

The study shows that two genes that confer resistance against a particularly strong class of antibiotics can be shared easily among a family of bacteria responsible for a significant portion of hospital-associated infections.

Drug-resistant germs in the same family of bacteria recently infected several patients at two Los Angeles hospitals. The infections have been linked to medical scopes believed to have been contaminated with bacteria that can resist carbapenems, potent antibiotics that are supposed to be used only in gravely ill patients or those infected by resistant bacteria.

"Carbapenems are one of our last resorts for treating bacterial infections, what we use when nothing else works," said senior author Gautam Dantas, PhD, associate professor of pathology and immunology. "Given what we know now, I don't think it's overstating the case to say that for certain types of infections, we may be looking at the start of the post-antibiotic era, a time when most of the antibiotics we rely on to treat bacterial infections are no longer effective."

Dantas and other experts recommend strictly limiting the usage of carbapenems to cases in which no other treatments can help.

The study, conducted by researchers at Washington University, Barnes-Jewish Hospital and the National University of Sciences and Technology in Pakistan, is available online in Emerging Infectious Diseases.

The researchers studied a family of bacteria called Enterobacteriaceae, which includes *E. coli*, *Klebsiella pneumoniae* and *Enterobacter*. Some strains of these bacteria do not cause illness and can help keep the body healthy. But in people with weakened immune systems, infections with carbapenem-resistant versions of these bacteria can be deadly.

The Centers for Disease Control and Prevention named carbapenem-resistant Enterobacteriaceae as one of the three most urgent threats among emerging forms of antibiotic-resistant disease. Studies have shown the fatality rate for these infections is above 50 percent in patients with weakened immune systems.

Two genes are primarily responsible for carbapenem-resistant versions of these disease-causing bacteria. One gene, KPC, was detected in New York in 2001 and quickly spread around most of the world, with the exception of India, Pakistan and other South Asian countries. This gene was present in the bacteria that recently contaminated medical equipment in a Los Angeles hospital where two patients died.

A second carbapenem resistance gene, NDM-1, was identified in 2006 in New Delhi, India. It was soon detected throughout South Asia, and most patients infected by bacteria with NDM-1 have had an epidemiological link to South Asian countries.

Dantas and his collaborators were curious about why the two resistance genes seemed to be geographically exclusive. For the study, they compared the genomes of carbapenem-resistant bacteria isolated in the United States with those of carbapenem-resistant bacteria isolated in Pakistan.

Based on the apparent geographic exclusivity of the two resistance genes, the scientists expected to find that bacteria from the two regions were genetically different. Such differences could explain why the two resistance genes weren't intermingling. But the researchers' results showed otherwise. The bacteria's high genetic similarity suggests that the antibiotic resistance genes could be shared easily between bacteria from the two geographic regions.

The researchers also sequenced a special portion of bacterial genetic material called plasmids. Most of a bacteria's DNA is found in its chromosome, but bacteria also have many extra, smaller and circular bits of DNA known as plasmids that easily can pass from one bacterial strain to another. A plasmid is like a bacterial gene delivery truck; it is the primary way antibiotic resistance genes spread between bacteria.

The researchers identified a few key instances in which the plasmids carrying NDM-1 or KPC were nearly identical, meaning they easily could facilitate the spread of antibiotic resistance between disease-causing bacteria found in the United States and South Asia. Recent evidence suggests that this intermingling already may be happening in parts of China.

“Our findings also suggest it's going to get easier for strains of these bacteria that are not yet resistant to pick up a gene that lets them survive carbapenem treatment," Dantas said. "Typically, that's not going to be a problem for most of us, but as drug-resistant forms of Enterobacteriaceae become more widespread, the odds will increase that we'll pass one of these superbugs on to a friend with a weakened immune system who can really be hurt by them."

Tackling antibiotic resistance through hand hygiene practices

Antimicrobial resistance occurs when a microorganism is no longer destroyed or stopped from reproducing by an anti-microbial medicine to which it was originally sensitive - quite simply, "the drugs don't work anymore". In recent years, some common pathogens have demonstrated multi-drug resistance and have caused infections, including those of urinary tract, bloodstream and wounds.

More recently, microbes have started to show resistance to a group of antibiotics called carbepenemases; yet another group of antibiotics no longer work as effectively to clear infections. The number of antibiotics that can treat patients is shrinking, and there are rare cases of microbes that have become resistant to almost all antibiotics in use, potentially taking us back to a pre-antibiotic age.

Tackling Antimicrobial resistance (AMR) requires action on multiple levels, the prudent use of antibiotics being one of the key actions.

The importance of infection prevention and control in general and hand hygiene in particular in preventing the spread of microorganisms has been repeatedly highlighted. One way to control AMR is ensuring that resistant microorganisms are not spread via the hands of (mainly) healthcare workers, and do not have the opportunity to invade vulnerable patients' bodies. Hand hygiene helps to make this potential threat into an avoidable one.

Hand hygiene undertaken at the right time - the WHO's 5 Moments for Hand Hygiene - prevents the spread of resistant or sensitive organisms that can be present on or in patients or in our environment.

Antibiotic Resistance

When exposed to antibiotics, bacteria change to reduce or eliminate their susceptibility to a specific antibiotic. By developing resistance, the bacteria become more difficult or even impossible to treat. Any time bacteria are exposed to an antibiotic, some organisms are unaffected while others die. These resistant strains then multiply and become more prevalent. Overexposure or improper antibiotic use promotes bacterial resistance. Antibiotics are only effective against bacteria, not viruses.

Emergence of resistant organisms

Staphylococcus aureus

Staphylococcus aureus causes a variety of infections, from superficial skin infections to deep tissue infections or more life-threatening infections such as pneumonia, sepsis, and endocarditis. Treatment is with semi-synthetic penicillins and a wide range of antibiotic agents.

Methicillin-resistant Staphylococcus aureus (MRSA) is a strain that is no longer sensitive to methicillin (oxacillin/nafcillin) due to alterations in penicillin-binding proteins located in bacterial cell walls. MRSA is not more virulent than methicillin-sensitive S. aureus (MSSA), but may be more difficult to treat due to limited antibiotic choices. The majority of MRSA isolates are also resistant to most other antibiotics, necessitating the use of the glycopeptide antibiotic, vancomycin.

Methicillin-resistant Staphylococcus aureus

Methicillin-resistant Staphylococcus aureus first emerged in England in 1960, soon after methicillin was introduced for the treatment of penicillin-resistant staphylococci. Colonization with MRSA may occur in the nares, axillae, chronic wounds or pressure ulcer surfaces, perineum, around gastrostomy and tracheostomy sites, and in the sputum or urine.

One of the most common sites of colonization in both patients and employees is the nose (anterior nares). As with susceptible strains of S. aureus, personnel may become colonized with MRSA but rarely develop infections.

MRSA has become a prevalent nosocomial pathogen in hospitals; the most important reservoirs of MRSA are infected or colonized patients. Although hospital personnel can serve as reservoirs for MRSA and may harbor the organism for many months, they have been more commonly identified as a link for transmission between colonized or infected patients. The main mode of transmission is via hands (especially healthcare workers' hands) which may become contaminated by contact with a) colonized or infected patients, b) colonized or infected body sites of the personnel themselves, or c) devices, items, or environmental surfaces contaminated with body fluids containing MRSA.

Vancomycin-resistant Staphylococcus aureus

In June 2002, the first clinical isolate of S. aureus that is fully resistant to vancomycin (VRSA) was reported in the United States. The vancomycin-resistant genetic material from a co-infecting enterococci strain apparently had transferred to Staphylococcus aureus within the patient. The patient was successfully treated with trimethoprim sulfamethoxazole and the isolate was susceptible to several other drugs.

In the healthcare setting, the CDC recommends a patient with VRSA be placed in a private room and have dedicated patient care items. Health care workers providing care to such patients must follow Contact Precautions.

Enterococcus

Enterococci are part of the normal flora found in the gastrointestinal and female genital tracts. Most enterococcal infections have been attributed to endogenous flora within the individual patient. However, patient-to-patient transmission can and does occur via direct contact, or indirectly via hands of personnel, contaminated equipment or environmental surfaces.

In the past, all enterococci were sensitive to antibiotics such as ampicillin and vancomycin.

Vancomycin-resistant Enterococcus (VRE)

Vancomycin-resistant enterococci (VRE) are no more virulent than antimicrobial-sensitive enterococci but VRE poses distinct

problems. These include the lack of available antibacterials and the possibility that vancomycin-resistant genes may be transferred to other gram-positive microorganisms.

Infections by multidrug-resistant organisms (MDROs) are increasing worldwide.

Prevention of spread and control of MDROs in health-care settings are critical and urgent as the number of antibiotics available to treat these infections is extremely limited and development of new antibiotics is not forthcoming in the foreseeable future. Worldwide, the most common bacteria causing health-care associated infections (HAIs) are:

MRSA **M**ethicillin **r**esistant ***S****taphyloccous* ***a****ureus*

VRE **V**ancomycin **r**esistant ***E****nterococci* spp.

ESBL **E**xtended-**s**pectrum **b**eta (β)-**l**actamase gram-negative organisms

CRE **C**arbapenems **r**esistant **E**nterobacteriaceae

MRAB **M**ulti-**r**esistant ***A****cinetobacter* ***b****aumannii*

The emergence of resistance in these microorganisms has mainly been caused by an inappropriate use of antibiotics in general and use of broad spectrum antibiotics in particular.

In addition the spread of MDROs in health-care settings is common and occurs mostly via health-care workers' (HCWs) contaminated hands, contaminated items/equipment and environment often leading to outbreaks and serious infections especially in critically ill patients.

Therefore, implementation of standard precautions for *all* patients *all* the time is key to preventing spread of all microorganisms and MDROs in particular. Hand hygiene performance according to recommendations is the most important measure among standard precautions.

Health care associated infections (HAIs)

Health care associated infections are drawing increasing attention from patients, insurers, governments and regulatory bodies. This is not only because of the magnitude of the problem in terms of the associated morbidity, mortality and cost of treatment, but also due to the growing recognition that most of these are preventable. The medical community is witnessing in tandem unprecedented advancements in the understanding of pathophysiology of infectious diseases and the global spread of multi-drug resistant infections in health care set-ups. These factors, compounded by the paucity of availability of new antimicrobials have necessitated a re-look into the role of basic practices of infection prevention in modern day health care. There is now undisputed evidence that strict adherence to hand hygiene reduces the risk of cross-transmission of infections. With "Clean Care is Safer Care" as a prime agenda of the global initiative of WHO on patient safety programmes, it is time for developing countries to formulate the much-needed policies for implementation of basic infection prevention practices in health care set-ups.

Normal flora of hands

There are two types of microbes colonizing hands: the resident flora, which consists of microorganisms residing under the superficial cells of the stratum corneum and the transient flora, which colonizes the superficial layers of the skin, and is more amenable to removal by routine hand hygiene. Transient microorganisms survive, but do not usually multiply on the skin. They are often acquired by health care workers (HCWs) during direct contact with patients or their nearby contaminated environmental surfaces and are the organisms most frequently associated with HCAIs.

Colonization of hands with pathogens and their role in transmission

The hands of HCWs are commonly colonized with pathogens like methicillin resistant S. aureus (MRSA), vancomycin resistant Enterococcus (VRE), MDR-Gram Negative bacteria (GNBs), Candida spp. and Clostridium difficile, which can survive for as long as 150 h. Approximately 106 skin epithelial cells containing viable microorganisms are shed daily from the normal skin, which can contaminate the gowns, bed linen, bedside furniture, and other objects in the patient's immediate environment. Hand carriage of resistant pathogens has repeatedly been shown to be associated with nosocomial infections. The highest rates of hand contamination are reported from critical care areas, which also report most cases of cross-transmission. The hands may become contaminated by merely touching the patent's intact skin or inanimate objects in patients' rooms or during the "clean" procedures like recording blood pressure

Importance of hand hygiene

Proper hand hygiene is the single most important, simplest, and least expensive means of reducing the prevalence of HAIs and the spread of antimicrobial resistance. Several studies have demonstrated that handwashing virtally eradicates the carriage of MRSA which invariably occurs on the hands of HCPs working in ICUs. An increase in handwashing compliance has been found to be accompanied by a fall in MRSA rates. The hand hygiene liason group identified nine controlled studies, all of which showed significant reductions in infection related outcomes, even in settings with a high infection rates in critically ill patients. Transmission of Health-care-associated *Klebsiella* sp. has also been documented to reduce with improvement in hand hygiene. The evidence suggests that adherence to hand hygiene practices has significantly reduced the rates of acquisition of pathogens on hands and has ultimately reduced the rates of HAIs in a hospital.

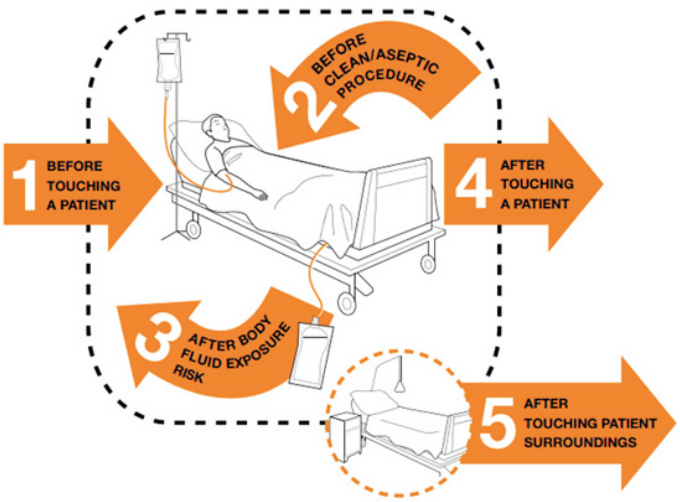
Indications for hand hygiene during patient care

Wash hands with soap and water when (*i*) visibly dirty or contaminated with proteinaceous material, blood, or other body fluids and if exposure to *Bacillus anthracis* is suspected or proven (since the physical action of washing and rinsing hands in such circumstances is recommended because alcohols, chlorhexidine, iodophors, and other antiseptic agents have poor activity against spores); (*ii*) After using a restroom, wash hands with a non-antimicrobial soap and water or with an antimicrobial soap and water; and (*iii*) before and after having food.

In all other clinical situations described below, when hands are not visibly soiled, an alcohol-based hand rub should be used routinely for decontaminating hands. (*i*) Before having direct contact with patients. (*ii*) Before donning sterile gloves when inserting a central intravascular catheter. (*iii*) Before inserting indwelling urinary catheters, peripheral vascular catheters, or other invasive devices that do not require a surgical procedure. (*iv*) After contact with a patient's intact skin (*e.g.*, when taking a pulse or blood pressure or lifting a patient). (*v*) After contact with body fluids or excretions, mucous membranes, nonintact skin, and wound dressings if hands are not visibly soiled. (*vi*) After

contact with inanimate objects (including medical equipment) in the immediate vicinity of the patient. (*vii*) After removing gloves. (*viii*) If moving from a contaminated body site to a clean body site during patient care.

The WHO "SAVE LIVES: Clean Your Hands" programme reinforces the "My 5 Moments for Hand Hygiene" approach as key to protect the patients, HCWs and the health-care environment against the spread of pathogens and thus reduce HAIs. This approach encourages HCWs to clean their hands: before touching a patient, before clean/aseptic procedures, after body fluid exposure/risk, after touching a patient and after touching patient surroundings.



Methods used to improve hand hygiene compliance

Multimodal strategies have been shown to be more successful in improving rates of adherence with hand hygiene in HCWs than single interventions. Targeted, multi-faceted approaches focusing on system change, administrative support, motivation, availability of alcohol-based hand rubs, training and intensive education of HCWs and reminders in the workplace have been recommended for improvement in hand hygiene.

Recent studies support the fact that interactive educational programmes combined with free availability of hand disinfectants significantly increased the hand hygiene compliance. The four member States of the European Union, which implemented National Hand Hygiene Campaigns found the following strategies to be extremely useful in their countries: Governmental support, the use of indicators for hand hygiene benchmarking, developing national surveillance systems for auditing alcohol based hand rub consumption and auditing hand hygiene compliance[4]. Trampuz *et al* advocated simple training sessions for HCWs to be held in each ward to introduce the advantage of alcohol hand rubs over hand washing.

Other factors like positive role modeling (hand hygiene behaviour of senior practitioners) and the use of performance indicators also remarkably improve adherence to hand hygiene. There should be adequate supply of hand hygiene products, lotions and creams, disposable towels and facilities for hand washing, where necessary. Alcohol hand rubs should be available at the point of care in sufficient quantities. It needs to be emphasized that wearing gloves does not replace the need for hand hygiene and that contamination may occur during glove removal. Studies by Pitet showed a remarkable and long lasting improvement in hand hygiene compliance using a multimodal strategy, which has been adopted by the first Global Patient Safety Challenge of WHO to develop hand hygiene strategies. The availability of individual, pocket carried bottles also increased compliance.

Apart from this, all hospitals should have a dynamic infection control team, robust surveillance system, adequate staff to disseminate evidence-based knowledge in an easily comprehensible way to all cadres of staff. At a more local or regional level, there is a need for institutional frameworks or programmes to deal with HAIs.

According to WHO, convincing evidence that improved hand hygiene practices lead to a reduction of infections caused by multidrug resistant bacteria in health facilities has been presented in a new report. For example, when hand hygiene compliance in health facilities increases from poor (<60%) to excellent (90%), there can be a 24% reduction in methicillin-resistant *Staphylococcus aureus* (MRSA) acquisition.

Bibliography:

1. Hand hygiene: Back to the basics of infection control, Indian J Med Res. 2011 Nov; 134(5): 611–620. Purva Mathur.
2. World Hand Hygiene Day: Focus on Antimicrobial Resistance: Centers for Disease Control and Prevention (CDC) sent this bulletin at 05/05/2014 02:06 PM EDT
3. CDC. Recommendations for Preventing the Spread of Vancomycin Resistance. MMWR, 1995; Vol44/No.RR-12.
4. CDC. Recommendations for Preventing Transmission of Infections Among Chronic
5. Hemodialysis Patients. MMWR,2001;Vol.50/No.RR-5.
6. Position Paper on the Control of Methicillin-resistant Staphylococcus aureus in Hospitals and Long-term Care Facilities. 1991. Alabama Department of Public Health.
7. Guidelines for Prevention and Control of Antibiotic Resistant Organisms: Focus on Methicillin-Resistant Staphyloccus aureus (MRSA) and Vancomycin-Resistant Enterococcus (VRE). 1997. Michigan Society for Infection Control.
8. Guideline for the Management of Antimicrobial Resistant Microorganisms in Minnesota Long-Term Care Facilities. 2000. Minnesota Department of Health.
9. Report of the Iowa Antibiotic Resistance Task Force: A Public Health Guide. 1999. Iowa Antibiotic Resistance Task Force.
10. Guidelines for Control of Antibiotic Resistant Organisms. 1999. Florida Department of Health.
11. A Public Health Guide for the Management of Patients with Antibiotic Resistant Organisms in a Variety of Health Care Settings. 1998. Department of Health and Family Services,
12. Wisconsin Bureau of Public Health Communicable Disease Section.
13. CDC Website, www.cdc.gov/ncidod/hip/aresist/mrsahcw.htm MRSA: Information for Healthcare Personnel. 2002.
14. CDC, Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA./APIC/IDSA Hand Hygiene Task Force. Guideline for Hand Hygiene in Health-Care Settings, MMWR, 2002; Vol. 51/No. RR-16.
15. CDC, *Staphylococcus aureus* Resistant to Vancomycin — United States, 2002. MMWR, 2002; Vol. 51/No. 26; 565-567.

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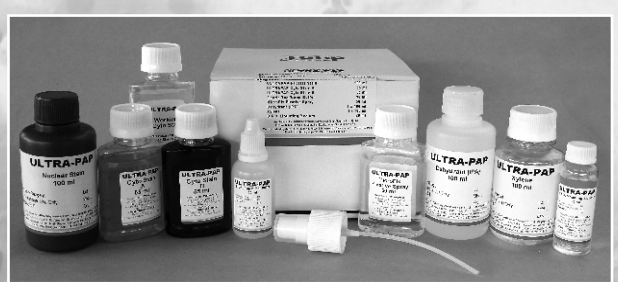

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