THOMPSON RIVERS UNIVERSITY | Risk & Safety Services

Exposure Protocols

Contents

Introduction

Within this binder are a list of some of the hazards you, or other researchers in this lab, could conceivably be exposed to during the course of your research. Please note that they are not listed alphabetically. This was done in order to facilitate simple adding and removal of organisms as required.

The lists and information within are not meant to be alarmist or to frighten. Rather, their purpose is to inform and help direct emergency responses to exposure events. Not every exposure will result in infection or illness. Some exposures will carry a higher risk of infection than others and this will be dependent on:

- the virulence of the organism(s) and/or nature of the material(s) in question;
- how much is known about the organism(s) and or material(s) with which work is being carried out;
- mode(s) of transmission;
- route of exposure;
- the required infectious dose of the pathogen(s) or suspected pathogen(s) in question, and if possible, an estimation as to whether or not that threshold was reached.

Deciding whether or not you are at risk for infection after an exposure is not up to you – a professional medical assessment is required to determine this. For this reason, regardless of perceived risk, in the event of any exposure with any of the materials that are contained in this binder, remain calm and follow the post-exposure protocols listed below:

1) Remove any contaminated PPE and personal effects immediately;

2) Evacuate yourself from the area in which the exposure took place;

3) Wash any potentially exposed skin/mucous membrane/hair areas thoroughly with soap and running water for 10 to 15 minutes;

4) Encourage bleeding if exposure involves a sharps injury or puncture and keep washing for a minimum of 15 minutes;

5) Yourself or someone else must inform your supervisor, senior laboratory staff, or the biosafety officer of the exposure;

6) Get immediate medical attention.

The flow chart on the following pages is to assist you and your supervisor (and your medical team – emergency room physician, family physician or public health office if required) in managing exposure risks and treatment methods following an exposure.

Please note that you **MUST REPORT** ANY AND ALL EXPOSURES.

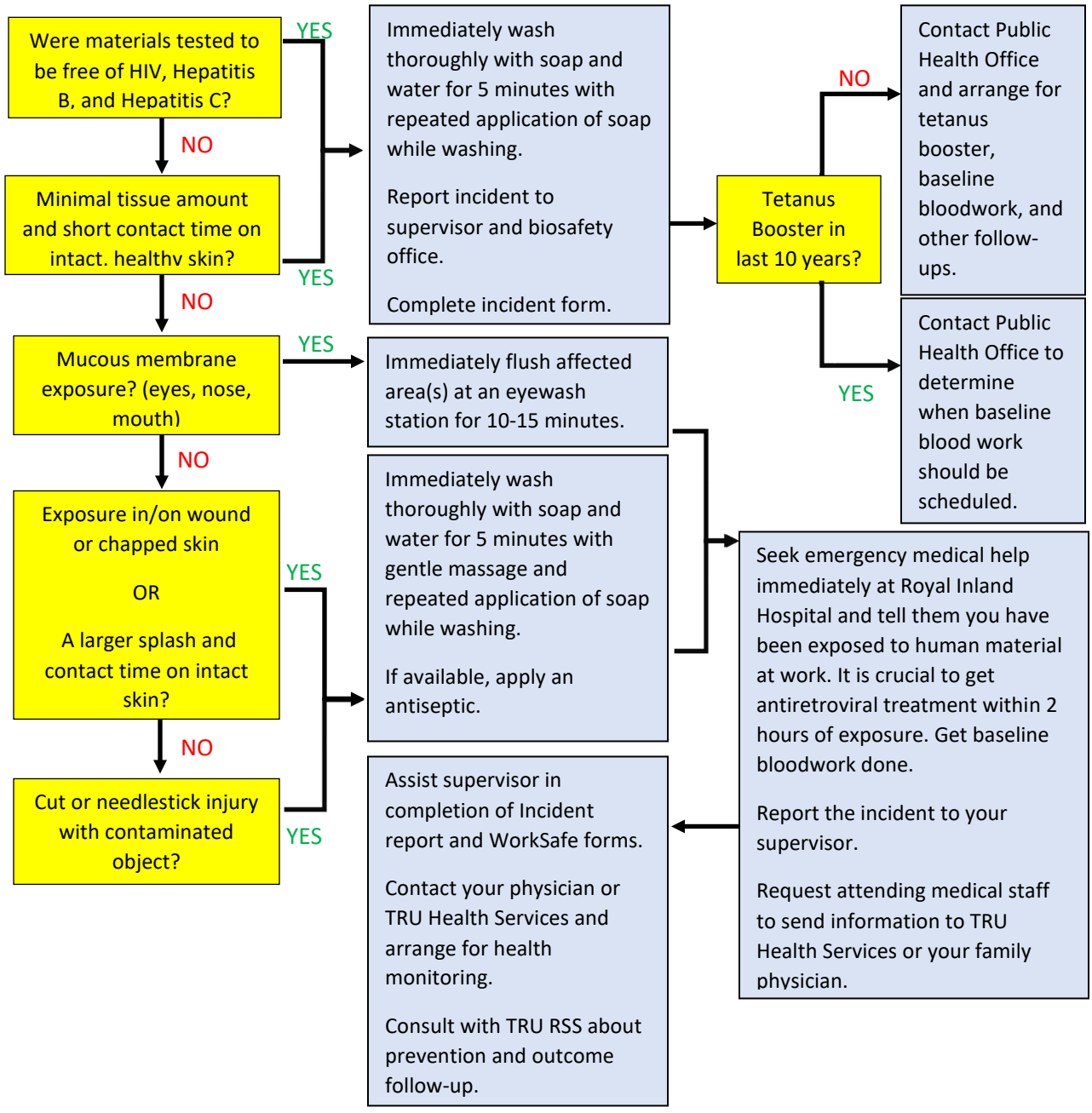
Reporting exposures and completing an incident report, allows us - collectively you, your supervisor, your colleagues, and the university community – to learn from the incident and possibly make policy or behavioral change requirements in order to prevent similar future incidents.

Another important reason for reporting exposures is to prevent exposure of other individuals following the incident. In other words, if an exposed person is infectious and does not know, or does know and is not instructed in the proper way to prevent infecting others, additional infectious events will not be prevented.

A third reason that illustrates why reporting an exposure is important, is that after an infectious exposure to many of the risks listed in this binder, serious signs or symptoms of infection may take weeks, months, or years to present themselves. If an exposure is not reported, not only may exposed individuals not get needed acute medical treatment, but they may also have more difficulty in the future, securing critical medical support if/when infection manifests itself symptomatically.

Any questions or concerns about the information within this binder can be directed to the TRU Biosafety Officer or RSS: safety@tru.ca

Flowchart – First Aid and Medical Response Protocol for Human Blood and/or Tissues and MDRO Incidents



IMPORTANT:
NO MATTER YOUR COURSE OF ACTION
Take the information sheets within this binder that corresponds with the organism(s) and/or material(s) to which you may have been exposed to the administering medical authorities.

Pathogen identification, Mode of Transmission, Incubation Period, Period of Communicability, Infectious Dose, Typical Presenting Symptoms, Mode(s) of Decontamination, and Emergency Response:

Primary Blood Products

Medical Surveillance for Primary Blood Products

Human Immunodeficiency Virus (HIV);

Hepatitis B Virus (HBV);

Hepatitis C Virus (BVC);

Treponema pallidum

Pathogen Identification	**Human Immunodeficiency virus (HIV)**	**Hepatitis B Virus (HBV)**	**Hepatitis C Virus (HCV)**	***Treponema pallidum***
Mode(s) of Transmission	HIV is transmitted either by exposure to mucosa or parenterally via contaminated needles.	HBV is transmitted by percutaneous or mucosal exposure to infected blood or other body fluid.	Mainly parenterally by contaminated needles and other sharps.	T. pallidum is transmitted by percutaneous or mucosal exposure to infected blood or other body fluid.
Incubation Period	Variable. Commonly the time from infection to the development of detectable antibodies is generally 1 to 3 months; however, the time from HIV infection to diagnosis of AIDS had an observed range of less than 1 year to 15 years or longer.	Usually 24-180 days (average 60-90 days). The variation depends on the amount of virus in the inoculum, mode of transmission, and other host factors.	Ranges from 2 to 12 weeks.	Syphilis acquired via mucosal exposure has an incubation of 10-90 days, although it is usually 21 days. Secondary venereal syphilis usually arises 6 weeks to 6 months post infection. Tertiary venereal syphilis has its onset months to years after initial infection. The primary stage of endemic syphilis arises approximately 2 to 4 weeks after inoculation. Secondary stage occurs 3 to 6 months post-inoculation and tertiary stage occurs as early as 6 months or as late as several years after initial symptoms.
Period of Communicability	Early after HIV infection and is thought to last throughout the life of the infected individual. Infectiousness is related to viral load.	All persons who are Hepatitis B antibody positive are potentially infectious, and blood can be infectious for several weeks before the onset of clinical symptoms.	All infected persons are potentially infectious.	If present in a blood sample, it may survive for as long as 120 hours at 4°C. Otherwise it may not be viable, but caution must still be exercised.
Infectious Dose(s)	Unknown.	Unknown.	Unknown.	57 organisms by injection.

Typical Presenting Symptoms	**Acute infection** is accompanied by non-specific "flu-like" and "mononucleosis-like" symptoms such as myalgia, arthralgia, diarrhoea, nausea, vomiting, headache, hepatosplenomegaly, weight loss, and neurological symptoms. **Typically, when the patient's CD4+ T-cell count falls below 500 cells/µL, syndromes indicative of depressed cell mediated immunity can appear**. Examples include orophayngeal and recurrent vulvovaginal candidiasis, bacillary angiomatosis, recurrent or multidermatomal herpes zoster, listeriosis, infections due to *Rhodococcus equi*, pelvic inflammatory disease, oral hairy leukoplakia associated with Epstein-Barr virus, cervical dysplasia, long lasting diarrhoea, idiopathic thrombocytopenic purpura, and peripheral neuropathy**. Late-stage disease** refers to the period when the patient's CD4+ T-cell count falls below 200 cells/µL. The loss of the integrity of cell-mediated immune responses allows ubiquitous environmental organisms with limited virulence to become life threatening pathogens. Examples of conditions (as set out by the US Centers for Disease Control and Prevention) include candidiasis of bronchi, trachea, lungs or oesophagus, invasive cervical cancer, coccidioidomycosis, cryptococcosis, cryptosporidiosis, cytomegalovirus disease (other than liver, spleen, or nodes), cytomegalovirus retinitis (with loss of vision), HIV-related encephalopathy, herpes simplex, histoplasmosis, isosporiasis, Kaposi's sarcoma,	***Acute hepatitis B infection*****:** Persons with acute hepatitis B infection may be asymptomatic or present with a clinical picture varying from mild to severe hepatitis. Persons with symptomatic acute HBV infections can show signs and symptoms that include nausea, abdominal pain, vomiting, fever, jaundice, dark urine, changes in stool colour, and hepatomegaly or splenomegaly as well as signs of liver dysfunction. ***Chronic hepatitis B infection*****:** Defined as the persistence of Hepatitis B antibodies for more than 6 months. Persons with chronic HBV infection may be asymptomatic or may suffer from symptoms such as fatigue, anorexia, nausea, abdominal discomfort and liver dysfunction. They are at substantially increased risk for developing chronic liver diseases, including cirrhosis of the liver and primary hepatocellular carcinoma.	***Acute HCV infection***: Asymptomatic in most patients (60-75%). The syndrome of acute hepatitis is often preceded or accompanied by symptoms of fatigue, myalgia, low-grade fever, right upper quadrant pain, nausea, vomiting, jaundice, mild hepatosplenomegaly, maculopapular rash, and arthralgia. These symptoms may last for 2 to 12 weeks. ***Chronic HCV infection*****:** malaise, nausea, abdominal pain and pruritis. Fluctuating alanine transferase levels are characteristic. The late sequelae of chronic HCV infection include serious health consequences such as chronic hepatitis, cirrhosis, and hepatocellular carcinoma. If cirrhosis develops, patients may experience jaundice, splenomegaly, ascites, oesophageal varices, and hepatic encephalopathy. Extrahepatic manifestations are uncommon but may include mixed essential cryo-globulinaemia, membranous or membrano-proliferative glomerulo-nephritis, non-Hodgkin's lymphoma, Sjorgren's syndrome, lichen planus, and porphyria cutanea tarda.	If mucosal, acute infection can manifest as a painless chancre and regional lymphadenopathy. Acute parenteral infection may be asymptomatic Clinical manifestations of the secondary stage include a symmetric maculopapular rash involving the palms and soles, generalized lymphadenopathy, fever, malaise, raised lesions (condylomata lata), and alopecia. Tertiary syphilis may include gummatous syphillis where gummatous lesions may form on any organ or tissue, cardiovascular syphilis, which usually manifests as aortic disease and neurosyphillis. Neurosyphilis can manifest as acute syphilitic meningitis, meningovascular syphilis or as paresis or tabes doraslis.

	Burkitt's lymphoma, immunoblastic lymphoma, primary lymphoma of the brain, *Myco-bacterium avium* complex, *Myco-bacterium tuberculosis*, *Pneumocystis jirovecii* pneumonia, recurrent pneumonia, progressive multifocal leukoencephalopathy, recurrent salmonella septicaemia, toxoplasmosis of the brain, and wasting syndrome due to HIV.			
Mode(s) of Decontamination	HIV is susceptible to fresh 2% glutaraldehyde, 2% Jodopax (detergent and iodine), hypochlorite, iodine, phenolics, and to a lesser extent 70% ethanol, NaOH, isopropanol, and Ultraviolet (UV) light.	Treatment of HBV diluted in phosphate buffered saline with 1% non-ionic detergent (Triton X-100) plus 0.3% tri-n-butyl-phosphate leads to HBV inactivation. HBV is also inactivated by formaldehyde, glutaraldehyde, sodium hypochlorite (5,000 ppm available chlorine), quaternary ammonium compounds, and alcohols (70-80%). Moist heat at 98°C for 1 minute will partially inactivate HBV in a 1:10 serum dilution. Incubation at 60°C for 10 hours (pasteurisation) will also inactivate HVB.	HCV RNA is readily degraded by 2% glutaraldehyde when added to biological samples at 37°C, and soaking laboratory equipment in 3% glutaraldehyde is effective at limiting HCV transmission. Phenolic compounds (0.4 to 3%) are effective at inhibiting HCV binding and infectivity in VERO cell cultures. Furthermore, treatment of HCV diluted in phosphate buffered saline with 1% non-ionic detergent (Triton X-100) plus 0.3% tri-n-butyl-phosphate leads to inactivation. Also pasteurization is effective.	Susceptible to 70% ethanol, 2% glutaraldehyde, 1% sodium hypochlorite.
Emergency Response	If exposure is suspected wash area thoroughly and encourage bleeding if, sharps or puncture injury. Emergency post-exposure prophylaxis (PEP) must be sought immediately and initiated within 72 hours of the suspected exposure event. HIV postexposure prophylaxis regimens are based on the nature of the exposure. The majority of HIV exposures will warrant a two drug regimen, using 2 NRTIs or 1 NRTI and 1 NtRTI. Combinations include: zidovudine (ZDV) and lamivudine (3CT) or emtricitabine (FTC); stavudine (d4T) and 3TC or FTC; and	Following exposure to HBV the affected area should be washed immediately with soap and water. Seek emergency medial attention. Mucous membranes and conjunctivae should be irrigated thoroughly with water. If the material involved is known to contain HBV or be positive for Hepatitis B antibodies then hepatitis B immunoglobulin (HBIG) should be given, ideally within 48 hours of exposure. Seven drugs are licensed in the United States for treatment of HBV infection: interferon-α, pegylated interferon α-	If exposure is suspected, suspected wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Medical surveillance is recommended before initiating any HCV treatment. There is no demonstrated benefit associated with rapid PEP and HCV. If HCV exposure is suspected, an HCV RNA antibody test will be carried out shortly after exposure and then again after 12 weeks. If	If exposure is suspected, suspected wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Medical surveillance is recommended. Penicillin provides the most effective treatment for all stages of disease cause by *Treponema pallidum*. Those who are allergic to penicillin can take tetracycline, doxycycline, or erythromycin. Ceftriaxone may be considered as an alternative for treatment of early syphilis in pregnancy.

	tenofovir (TDF) and 3TC or FTC. The addition of a third or fourth drug should be considered for exposures that pose an increased risk of transmission. The preferred drugs in this case are proteinase inhibitors such as lopinavir/ritonavir (LPV/RTV).	2a, lamivudine, adefovir, entecavir, telbivudine, and tenofovir. Previously unimmunised adults exposed to HBsAg positive blood should receive HBIG as soon as possible as well as immunization with HB vaccine unless natural immunity can be confirmed. Two types of HB vaccine have been licensed and shown to be highly effective against all subtypes of HBV. The first, prepared from plasma from HBsAg-positive persons, is still widely used. The second is synthesised using recombinant DNA.	observed to be positive, treatment may be utilized and may consist of mono-therapy with pegylated interferon (addition of polyethylene glycol to interferon-α) and combined therapy of pegylated interferon with ribavirin, or standard interferon with ribavirin, are common methods of treating HCV infection.	Chloramphenicol may also be used to treat neurosyphilis.
Suggested Reference: http://www.phac-aspc.gc.ca/lab-bio/res/psds-ftss/index-eng.php.				

Primary Blood Products - continued

Human T-Lymphotropic Virus (HTLV);
Leptospira interrogans;
Creutzfedlt-Jacob agent, Kuru agent

Pathogen Identification	**Human T-Lymphotropic Virus (HTLV)**	***Leptospira interrogans***	**Creutzfedlt-Jacob agent, Kuru agent**
Mode(s) of Transmission	Infections can occur from blood and mucosal exposure.	Direct contact with infected urine or tissues; occasionally through ingestion of contaminated food or by inhalation of droplet aerosols of contaminated fluids.	The mode of transmission of most cases is unknown.
Incubation Period	Unknown.	Usually 10 days with a range of 4-19 days.	15 months to 2 years.
Period of Communicability	Unknown. Human-to-human transmission is possible.	Direct transmission from person to person is rare; leptospires may be excreted in urine for usually 1 month but has been observed as long as 11 months after the acute illness.	CNS and other tissues are infectious throughout symptomatic illness; lymphoid and other organs probably infectious before signs of illness appear.
Infectious Dose(s)	Unknown.	Unknown.	Unknown.
Typical Presenting Symptoms	Acute HTLV infection is rarely suspected or diagnosed. Adult T-cell leukemia or lymphoma (ATLL) occurs in 1-2% of those infected. Symptoms present 20-30 years after infection. Of those who develop ATLL, more than two thirds develop leukemia while the remainder develop lymphoma. Prognosis is approximately 1 year after development of ATLL. ATLL has five types: asymptomatic, pre-leukemic, chronic/smouldering, lymphoma, and acute. The smouldering type presents with skin lesions and involvement of the bone marrow. The chronic stage is associated with elevated circulating leukemic cells and usually progresses to the acute type within two	Fever, headache, chills, severe malaise, vomiting, myalgia and conjunctival suffusion; occasionally meningitis, rash and uveitis; sometimes jaundice, renal insufficiency, anemia and hemorrhage of the skin; clinical illness lasts 3 days to few weeks, often biphasic; may have asymptomatic infection; low case fatality rate but increases with age.	Insidious onset of confusion, progressive dementia, myoclonic jerks with spasticity, wasting and coma; slight elevation of CSF proteins; death usually occurs in less than 1 year. CNS disease with cerebellar ataxia, incoordination, tremors, rigidity, progressive wasting and death within 3-9 months.

	years. The acute phase is an aggressive form of leukemia and is accompanied by hypocalcaemia, elevated lactate dehydrogenase (LDH), skin lesions, lymphadenopathy, lymphomatous meningitis, lytic bone lesions, spleen or liver involvement and immune-deficiency. HTLV-1-associated myelopathy/tropical spastic paraparesis (HAM/TSP) has a shorter latency period than ATLL. HAM/TSP is a progressive and chronic myelopathy, with preferential damage to the thoracic spinal cord. Symptoms include muscle weakness of lower limbs, hyperreflexia, sphincter disorders, impotence, sensory disturbances and lower back pain.		
Mode(s) of Decontamination	Susceptible to 1% sodium hypochlorite, 2% glutaraldehyde, 4% chlorhexidine, 70% ethanol, 0.3% hydrogen peroxide, iodophores, phenolics, and quaternary ammonium compounds. Can be inactivated by steam sterilization at 121ºC for a minimum of 15 minutes and UV light.	Susceptible to 1% sodium hypochlorite, 70% ethanol, glutaraldehyde, formaldehyde. Sensitive to moist heat (121° C for at least 15 min).	Resistance to commonly used disinfectants is well recognized: formaldehyde, glutaraldehyde, ethanol, and iodine. Immersion in undiluted bleach (60,000 ppm available chlorine) for 1 hour is only partially effective. Disinfection should be carried out using 1N sodium hydroxide at room temperature for 1 hour (shorter treatments have occasionally not inactivated the pathogen). Resistant to ultraviolet and ionizing radiation, ultrasonication, nucleases, boiling, heat; autoclaving - 15 to 30 min at 121°C or 132°C will not effectively inactivate pathogen, 1 hour at 132°C is recommended). Contaminated electrodes stored in ethanol-formalin for several years were found to cause CJD in chimpanzee.
Emergency Response	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. No established therapy. Combination therapy of Zidovudine (AZT) and interferon alpha (INF-α) is used with some success to improve prognosis.	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms of illness; confirm serologically; isolation of leptospires from blood, CSF or urine. Doxycycline treatment within 4 days of onset, combination of amoxycillin and erythromycin can be effective; resistant to penicillin prophylaxis.	If exposure is suspected, wash area thoroughly with NaOH and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms of illness - clinical signs - diagnosis based on EEG, histopathological findings, transmission to animals from tissue samples from suspected specimens.

Pathogen identification, Mode of Transmission, Incubation Period, Period of Communicability, Infectious Dose, Typical Presenting Symptoms, Mode(s) of Decontamination, and Emergency Response:

Primary Breast Milk

Medical Surveillance Breast Milk

Human Immunodeficiency Virus (HIV);
Human T-Lymphotropic Virus (HTLV);
Cytomegalovirus (CMV);
Creutzfedlt-Jacob agent, Kuru agent

Pathogen Identification	**Human Immunodeficiency Virus (HIV)**	**Human T-Lymphotropic Virus (HTLV)**	**Cytomegalovirus (CMV)**	**Creutzfedlt-Jacob agent, Kuru agent**
Mode(s) of Transmission	HIV is transmitted either by exposure to mucosa or parenterally via contaminated needles. Blood, semen, vaginal secretions, cerebrospinal fluid, synovial fluid, peritoneal fluid, pleural fluid, pericardial fluid, amniotic fluid, other specimens containing visible blood, breast milk, unscreened or inadequately treated blood products, and infected human tissues are all potential sources of HIV.	Lab acquired infections have been reported and Infections can occur from mucosal exposure as well as accidental parenteral inoculation. Blood samples and bodily fluids (i.e. CSF, breast milk, blood etc.) are potential sources of HTLV.	Inhalation of concentrated aerosolized materials, droplet exposure of mucous membranes of the eyes, nose, or mouth, ingestion, accidental parenteral inoculation are the primary hazards associated with herpes viruses including Cytomegalovirus. No lab acquired infections have been reported. Urine, blood, breast milk, tears, stool, semen, respiratory secretions (nasopharyngeal secretions, saliva, throat washings, and bronchoalveolar lavage fluid) and cervical secretions are all potential sources of CMV infection.	The mode of transmission of most cases is unknown. Accidental parenteral inoculation; risk of infection from aerosols, droplets, and exposure of intact skin, gastric and mucous membranes is not known. No documented laboratory-associated infections with spongiform encephalopathies however, consequences of infection are grave. Infections have been observed via blood, CSF, and breast milk in animal studies.
Incubation Period	Variable. Commonly the time from infection to the development of detectable antibodies is generally 1 to 3 months; however, the time from HIV infection to diagnosis of AIDS had an observed range of less than 1 year to 15 years or longer.	Unknown.	Worldwide - Cytomegalovirus (CMV) is a universally distributed pathogen with approximately 40-100% of the world's population having CMV antibody present in blood as evidence of infection. Incubation period is generally 1-4 months post-CMV infection.	15 months to 2 years.

Period of Communicability	Early after HIV infection and is thought to last throughout the life of the infected individual. Infectiousness is related to viral load.	Unknown period of communicability, but, human-to-human transmission is possible.	CMV virus can persist in body fluids such as urine, saliva, and seminal fluids for many years, or can remain dormant until reactivation of latent infection. Transmission occurs through direct contact with body fluids from symptomatic or asymptomatic persons excreting the virus, thus infection may be transmitted between humans and from adults to children through childbirth and breastfeeding.	CNS and other tissues are infectious throughout symptomatic illness; lymphoid and other organs probably infectious before signs of illness appear.
Infectious Dose(s)	Unknown	Unknown.	Unknown	Unknown.
Typical Presenting Symptoms	Acute infection is accompanied by non-specific "flu-like" and "mononucleosis-like" symptoms such as myalgia, arthralgia, diarrhoea, nausea, vomiting, headache, hepato-splenomegaly, weight loss, and neurological symptoms. Typically, when the patient's CD4+ T-cell count falls below 500 cells/µL, syndromes indicative of depressed cell mediated immunity can appear. Examples include oropharyngeal and recurrent vulvovaginal candidiasis, bacillary angiomatosis, recurrent or multidermatomal herpes zoster, listeriosis, infections due to *Rhodococcus equi*, pelvic inflammatory disease, oral hairy leukoplakia associated with Epstein-Barr virus, cervical dysplasia, long lasting diarrhoea, idiopathic thrombocytopenic purpura, and peripheral neuropathy. Late-stage disease refers to the period when the patient's CD4+ T-cell count falls below 200 cells/µL. The loss of the	HTLV-1 primarily causes adult T-cell leukemia/ lymphoma and topical spastic parapareis/HTLV-1 associated myelopathy. It also causes uveitis, infective dermatitis and lymphadenitis. HTLV-2 is a less pathogenic strain and has been associated with milder neurological disorders and chronic pulmonary infections. HTLV-3 and HTLV-4 have not been associated with specific illnesses. Acute HTLV infection is rarely suspected or diagnosed. Adult T-cell leukemia or lymphoma (ATLL) occurs in 1-2% of those infected. Symptoms present 20-30 years after infection. Of those who develop ATLL, more than two thirds develop leukemia while the remainder develop lymphoma. Prognosis is approximately 1 year after development of ATLL. ATLL has five types: asymptomatic, pre-leukemic, chronic/smouldering,	CMV infection is common and usually asymptomatic in healthy children and adults, but can cause severe disease in newborns and immuno-compromised children or adults. CMV is the most common cause of congenital infection, affecting 0.2-2.4% of all infants, and also of viral-based mental retardation and hearing deficit in children of developing countries. Infections are often recurrent, caused by reactivation of latent virus (especially in immunocompromised patients such as bone marrow or other transplant recipients), reinfection may also occur due to the antigenic diversity of the virus. Infection may cause a mononucleosis-like-syndrome with prolonged fever (lasting 2-3 weeks), malaise, atypical	Insidious onset of confusion, progressive dementia, myoclonic jerks with spasticity, wasting and coma; slight elevation of CSF proteins; death usually occurs in less than 1 year. CNS disease with cerebellar ataxia, incoordination, tremors, rigidity, progressive wasting and death within 3-9 months.

	integrity of cell-mediated immune responses allows ubiquitous environmental organisms with limited virulence to become life threatening pathogens. Examples of conditions (as set out by the US Centers for Disease Control and Prevention) include candidiasis of bronchi, trachea, lungs or oesophagus, invasive cervical cancer, coccidioidomycosis, cryptococcosis, cryptosporidiosis, cytomegalovirus disease (other than liver, spleen, or nodes), cytomegalovirus retinitis (with loss of vision), HIV-related encephalopathy, herpes simplex, histoplasmosis, isosporiasis, Kaposi's sarcoma, Burkitt's lymphoma, immunoblastic lymphoma, primary lymphoma of the brain, *Myco-bacterium avium* complex, *Myco-bacterium tuberculosis*, *Pneumocystis jirovecii* pneumonia, recurrent pneumonia, progressive multifocal leuko-encephalopathy, recurrent salmonella septicaemia, toxoplasmosis of the brain, and wasting syndrome due to HIV.	lymphoma, and acute. The smouldering type presents with skin lesions and involvement of the bone marrow. The chronic stage is associated with elevated circulating leukemic cells and usually progresses to the acute type within two years. The acute phase is an aggressive form of leukemia and is accompanied by hypocalcaemia, elevated lactate dehydrogenase (LDH), skin lesions, lymphadeno-pathy, lymphomatous meningitis, lytic bone lesions, spleen or liver involvement and immune-deficiency. HTLV-1-associated myelopathy/tropical spastic paraparesis (HAM/TSP) has a shorter latency period than ATLL. HAM/TSP is a progressive and chronic myelopathy, with preferential damage to the thoracic spinal cord. Symptoms include muscle weakness of lower limbs, hyperreflexia, sphincter disorders, impotence, sensory disturbances and lower back pain.	lymphocytosis, cervical lymphadenitis, mild hepatitis, and encephalitis. Both primary CMV infection, or reactivation can cause serious illness in immuno-suppressed patients (for example patients with AIDS, patients with leukemia or lymphoma who are receiving chemotherapy, and recipients of organ transplants on immuno-suppressive therapy). The major symptoms of the disease in organ transplant recipients include fever, myalgia, malaise, arthralgia, leucopenia, thrombo-cytopenia, and hepatitis. Pneumonitis, caused by CMV infection in bone marrow transplant recipients is associated with significant morbidity and mortality (50-90%). CMV infection in HIV patients may spread to visceral organs, resulting in chorioretinitis, gastrointestinal infections (esophagitis, gastritis, and ulcerative colitis), polyradiculo-myelopathy, and neurological disorders.	
Mode(s) of Decontamination	HIV is susceptible to fresh 2% glutaraldehyde, 2% Jodopax (detergent and iodine), hypochlorite, iodine, phenolics, and to a lesser extent 70% ethanol, NaOH, isopropanol, and Ultraviolet (UV) light. HIV can remain viable in blood in syringes at room temperature for 42 days, and in blood and cerebrospinal fluid from autopsies for up to 11 days. Although drying in	Susceptible to 1% sodium hypochlorite, 2% glutaraldehyde, 4% chlorhexidine, 70% ethanol, 0.3% hydrogen peroxide, iodophores, phenolics, and quaternary ammonium compounds. Can be inactivated by steam sterilization at 121°C for a minimum of 15 minutes and UV light. HTLV-1 and HTLV-2 can survive in stored blood for 8-9 days.	Cytomegalovirus has been shown to be susceptible to 7.5% povidone-iodine. Although not much information is available on disinfectants specific for CMV, most herpes viruses are susceptible to 30% ethanol and isopropanol, 1% sodium hypochlorite, formaldehyde,	Resistance to commonly used disinfectants is well recognized: formaldehyde, glutaraldehyde, ethanol, and iodine. Immersion in undiluted bleach (60,000 ppm available chlorine) for 1 hour is only partially effective. Disinfection should be carried out using 1N sodium hydroxide at room temperature for 1 hour (shorter

	the environment is known to cause a rapid reduction in HIV concentration, under experimental conditions, Cell-free HIV dried onto a glass coverslip in 10% serum can survive for longer than 7 days, depending on the initial titre.		0.12% ortho- phenylphenol, and 0.04% glutaraldehyde. Inactivated by heat (56 °C for 30 min), low pH, UV light, cycles of freezing/thawing. Cytomegalovirus can survive on dry inanimate surfaces (persistence varies from only a few hours up to 7 days); blanket for 2 hours; plexiglass for 4-8 hours.	treatments have occasionally not inactivated the pathogen). Resistant to ultraviolet and ionizing radiation, ultrasonication, nucleases, boiling, heat; autoclaving - 15 to 30 min at 121°C or 132°C will not effectively inactivate pathogen, 1 hour at 132°C is recommended). Contaminated electrodes stored in ethanol-formalin for several years were found to cause CJD in chimpanzee.
Emergency Response	If exposure is suspected wash area thoroughly and encourage bleeding if, sharps or puncture injury. Emergency post-exposure prophylaxis (PEP) must be sought immediately and initiated within 72 hours of the suspected exposure event. HIV postexposure prophylaxis regimens are based on the nature of the exposure. The majority of HIV exposures will warrant a two drug regimen, using 2 NRTIs or 1 NRTI and 1 NtRTI. Combinations include: zidovudine (ZDV) and lamivudine (3CT) or emtricitabine (FTC); stavudine (d4T) and 3TC or FTC; and tenofovir (TDF) and 3TC or FTC. The addition of a third or fourth drug should be considered for exposures that pose an increased risk of transmission. The preferred drugs in this case are proteinase inhibitors such as lopinavir/ritonavir (LPV/RTV). There is no available vaccine for HIV infection.	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. No established therapy available. Monitor for symptoms. HTLV is detected in blood serum by identifying antibodies using ELISA. Other methods of detection include particle agglutination assays and Western Blotting. For the identification of specific viral sequences, PCR is used. Limited therapy is available for HTLV infections. ATL treated with chemotherapy. Zidovudine (AZT) and alpha interferon have shown some response and improved the ATL prognosis. Other treatments are currently under investigation including arsenic, trioxide, proteasome inhibitors, retenoids, angiogenesis inhibitors and cellular immunotherapy. Antiretroviral treatments using lamivudine and high dose interferon alpha and interferon beta is used for HTLV-1-associated myelopathy/tropical spastic parparesis. Uveitis is treated with topical and systemic corticoids to improve sight. Infective dermatitis is	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Since CMV infection resolves spontaneously in normal healthy patients, antiviral treatment is not usually recommended. If exposed individual is chronically immuno-compromised, Intravenous ganciclovir or oral valganciclovir are the first line drugs for therapy for CMV infection. Intravenous cidofovir and foscarnet are used as alternatives. Once symptomatic improvement has been achieved, these drugs can also be used for chronic maintenance therapy in patients with CMV retinitis or neurological disease. No vaccine is licensed for prevention of cytomegalovirus infection.	If exposure is suspected, wash area thoroughly with NaOH and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms of illness - clinical signs - diagnosis based on EEG, histopathological findings, transmission to animals from tissue samples from suspected specimens.

		treated with antibiotics. There is no available vaccine for HTLV 1 or 2.		

Suggested Reference: http://www.phac-aspc.gc.ca/lab-bio/res/psds-ftss/index-eng.php.

Pathogen identification, Mode of Transmission, Incubation Period, Period of Communicability, Infectious Dose, Typical Presenting Symptoms, Mode(s) of Decontamination, and Emergency Response:

Laboratory Bacterial Inventory – Bacteria

Medical Surveillance Inventory Bacteria

Streptococcus Group A (*S. pyogenes*);

Staphylococcus intermedius;

Staphylococcus hyicus;

Streptococcus Group B (*S. agalactiae*)

Pathogen Identification	***Streptococcus* Group A (*S. pyogenes*)**	***Staphylococcus intermedius***	***Staphylococcus hyicus***	***Streptococcus* Group B (*S.agalactiae*)**
Mode(s) of Transmission	Transmission via respiratory droplets, hand contact with nasal discharge and skin contact with impetigo lesions are the most important modes of transmission. The pathogen can be found in its carrier state in the anus, vagina, skin and pharynx and contact with these surfaces can spread the infection. Inhalation of infectious aerosols and contamination of mucocutaneous lesions are the primary laboratory hazards associated with working with this pathogen. The bacterium can survive on a dry surface for 3 days to 6.5 months. Necrotizing fasciitis is usually because of contamination of skin lesions or wounds with the infectious agent.	Inhalation of infectious aerosols, contamination of mucocutaneous lesions, and accidental inoculation are the primary hazards associated with working with this pathogen. Direct contact with infectious material and objects may also be a source of transmission.	Contamination of mucocutaneous lesions, and accidental inoculation are the primary hazards associated with working with this pathogen. Contact between exposed intact and inflamed skin and infectious material and contaminated objects can also be a source of transmission.	Transmission is possible through contact with contaminated objects and/or surfaces. Accidental parenteral inoculation, ingestion, inhalation of infectious aerosols and direct contact are the primary hazards when working with this pathogen. *Streptococcus agalactiae* has been isolated from blood, cerebrospinal fluid, joint fluid, peritoneal fluid, pleural fluid, bone, vaginal, throat and rectal cultures, aerosols and feces. This bacterium has been found to survive months in dry dust in buildings.
Incubation Period	Usually 1 to 3 days.	Variable and indefinite, however most commonly incubation is 4-10 days.	Unknown due to lack of available data.	Unknown.
Period of Communicability	If untreated, patients with streptococcal pharyngitis are infective during the acute phase of the illness, usually 7-10 days, and for one week afterwards; however, if antibiotics are used, the infective period is reduced to 24 hours. The bacterium can remain in the body in its carrier state without causing	As long as a purulent lesion is present or as long as the carrier state exists.	Unknown due to lack of available data.	Variable and indefinite in some cases as symptom-free humans carry the pathogen in their throat, genitourinary tract and their rectum.

	illness in the host for weeks or months and is transmissible in this state.			
Infectious Dose(s)	Unknown	Unknown.	Unknown in humans.	Unknown.
Typical Presenting Symptoms	This bacterium is responsible for a wide array of infections. It can cause streptococcal sore throat which is characterized by fever, enlarged tonsils, tonsillar exudate, sensitive cervical lymph nodes and malaise. If untreated, strep throat can last 7-10 days. Scarlet fever (pink-red rash and fever) as well as impetigo (infection of the superficial layers of skin) and pneumonia are also caused by this bacterium. Septicaemia, otitis media, mastitis, cellulitis, erysipelas, myositis, osteomyelitis, septic arthritis, meningitis, endocarditis, pericarditis, and neonatal infections are all less common infections due to *S. pyogenes*. Streptococcal toxic shock syndrome, acute rheumatic fever (joint inflammation, carditis and CNS complications), post-streptococcal glomerulonephritis (inflammation, hematuriia, fever, edema, hypertension, urinary sediment abnormalties and severe kidney pain) and necrotizing fasciitis (rapid and progressive infection of subcutaneous tissue, massive systematic inflammation, hemorrhagic bullae, crepitus and tissue destruction) are some of the more serious complications involving *S.pyogenes* infections.	This bacterium is responsible for a wide array of infections. It may cause fever, tissue inflammation, tissue swelling (edema), purulent skin infections, skin abscesses, cellulitis, wound infections, nail bed infections, gastroenteritis, bacteremia, otitis, septic arthritis, endocarditis, sinusitis, mastoiditis, pneumonia, meningitis, and brain abscesses. *S. intermedius* also produces exotoxins, which if ingested, can cause symptoms similar to that of food-poisoning including abdominal cramping, diarrhea, vomiting, loss of appetite, fever, weakness, nausea, and headaches.	This bacterium is responsible for a wide array of infections. It has been implicated as a causative agent in toxic shock syndrome, cellulitis, bacteremia, septicemia, and spondylodiscitis. Based on animal models, it may also cause vomiting, purulent skin infection, and wound infection. Symptoms may vary and *S. hyicus* infection has been mistaken as acute *S. aureus* infection.	Infection symptoms depend on the part of the body that is infected. Bacteremia and sepsis can manifest as fever, chills, and low alertness. Pulmonary infections can present as pneumonia, the symptoms of which can include: fever and chills, cough, rapid breathing or difficulty breathing, and chest pain. Skin and soft tissue can manifest as swelling, redness, pain, and elevated temperature. Infected areas may also be full of pus or other drainage. If the infection involves bones or joints, it may present as fever, chills, swelling and stiffness that prevents normal joint range of motion.
Mode(s) of Decontamination	Susceptible to 1% sodium hypochlorite, 4% formaldehyde, 2% glutaraldehyde, 70% ethanol, 70% propanol, 2% peracetic acid, 3-6% hydrogen peroxide and 0.16% iodine.	Susceptible to 4% chlorhexidine in 70% ethanol, 6.5% sodium hypochlorite, 0.3% stabilized glutaraldehyde in 70% ethanol, 0.3% stabilized glutaraldehyde in distilled	10% sodium hypochlorite solution. Bacteria are susceptible to moist heat (121°C for at least 15	Susceptible to 2-5% phenol, 1% sodium hypochlorite, 4% formaldehyde, 2% glutaraldehyde, 70% ethanol, 70% propanol, 2% peracetic acid, 3-6% hydrogen

	Bacteria are susceptible to moist heat (121°C for at least 15 minutes) and dry heat (170°C for at least 1 hour). The bacterium can survive on a dry surface for 3 days to 6.5 months.	water, and 1% formaldehyde solutions. Bacteria are susceptible to moist heat (121°C for at least 15 minutes) and dry heat (170°C for at least 1 hour).	minutes) and dry heat (170°C for at least 1 hour).	peroxide, and iodine. Sensitive to moist heat at 55°C for 30 minutes. Bacteria are also susceptible to moist heat (121°C for at least 15 minutes) and dry heat (160-170°C for at least 1 hour). Fecal streptococci can be inactivated by ozone. This bacterium has been found to survive months in dry dust in buildings.
Emergency Response	If exposure is suspected wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms. Confirm infection by bacteriological and serological testing, latex bead agglutination, fluorescent antibody staining or ELISA. Appropriate antibiotic treatment is necessary for a *S. pyogenes* infection. Penicillin is used for respiratory tract infections (pharyngitis) and macrolides or lincosamides are used if there are allergies. Clindamycin may be used in cases of necrotizing fasciitis and surgical debridement of the affected area is necessary. *S. pyogenes* infections are susceptible to a variety of drugs: β-lactams such as penicillin, as well as erythromycin, clindamycin, imipenem, rifampin, vanomycin, macrolides and lincomycin; however, certain strains of the bacterium have been found to resistant to macrolides, lincomycin, chloramphenicol, tetracyclines and cotrimoxazole.	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Infections with this bacterium are often mistaken as S. aureus infections. It can be differentiated via a negative or weak maltose test, a positive arylamidase test, delayed mannitol fermentation, via Polymerase Chain Reaction identification, Ribotyping, Pulse-field gel electrophoresis, Pyrolysis-mass spectrometry, Multilocus enzyme electrophoresis. Some antibiotic resistance has been also observed in some clinical settings. Many Staphylococcus species are β-lactam resistant and therefore the use of penicillins, ampicillin, and amoxicillin, which are all β-lactam antibiotics in a clinical setting may not be effective. Resistance to tetracycline, streptomycin, and methicillin has also been observed.	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Infections with this bacterium can be mistaken as *S. aureus* infections. It can often be reliably differentiated and identified via 16S rRNA or nuc gene analysis. Penicillin has been observed to be an effective antibiotic in *S. hyicus* infection treatment.	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms and confirm by culture of blood or CSF. Serology can be used to determine the Lancefield group. Susceptible to penicillin or a combination of ampicillin and an aminoglycoside. *Streptococcus agalactiae* is also sensitive to vancomycin, ciprofloxacin, clindamycin, erythromycin, cotrimoxazole, and ceftriaxone. It is generally resistant to macrolides and clindamycin resistance has also occurred. No vaccine is currently available for *S. agalactiae*.

Suggested Reference: http://www.phac-aspc.gc.ca/lab-bio/res/psds-ftss/index-eng.php.

Medical Surveillance Inventory – Bacteria (cont)

Streptococcus Group C (*S. zooepidemicus*, *S. equi*, and *S. dysgalactiae*);

Streptococcus Group D;

Streptococcus pneumoniae;

Candida albicans

Pathogen Identification	***Streptococcus* Group C (*S. zooepidemicus, S. equi, and S. dysgalactiae*)**	***Streptococcus* Group D**	***Streptococcus pneumoniae***	***Candida albicans***
Mode(s) of Transmission	Inhalation of infectious aerosols, accidental parenteral inoculation, and contamination of mucocutaneous lesions are the primary hazards associated with working with this pathogen. Direct contact with infectious persons, materials, and objects can also facilitate transmission	Inhalation of infectious aerosols, contamination of mucocutaneous lesions, and accidental inoculation are the primary hazards associated with working with this pathogen. Direct contact with infectious persons, materials, and objects can also facilitate transmission. Blood, urine, wound samples, and feces are all potential carriers of this bacterium. In addition, Enterococci can grow and survive in harsh environments, and can persist almost anywhere including soil, plants, water, and food. Can survive 5 days to 4 months on dry inanimate surfaces.	Infectious cells can be disseminated via microaerosol droplets created by coughing or sneezing, or person-person oral contact can also facilitate infection. Person to person transmission is common, but infection is infrequent as healthy individuals carry *S. pneumoniae* in the nasopharyngeal region without any presence of infection. Sputum, nasal or throat swabs, blood, cerebrospinal fluids, and respiratory secretions can all be infectious. Primary laboratory hazards include: accidental inhalation of aerosols containing bacterial cells, accidental inoculation, direct mucous-to-person contact. *Streptococcus* spp. can survive in dental plaque for up to 7 days, in dust for up to 20 days, on glass for 1 – 11 days, and up to 180 days in frozen fish.	Most infections result from the patient's own flora, rather than from cross infection. Although rare, nosocomial transmission has also been reported to occur from inanimate surface, from hands of health care workers or between patients. In laboratories, the primary hazards are skin contact with contaminated personnel, surfaces, and cultures. Also through accidental parenteral inoculation and trauma of cutaneous barrier. Epithelial scrapings, lesion exudates, sputum, brochoalveolar lavage, and blood can all be infectious. *C. albicans* can survive on inanimate surfaces for 24 hours to 120 days, and on palms for about 45 minutes. *C. albicans* has been isolated from bed-sheets, cots, and wash-basins of nurseries, and it has also been found to be able to survive and grow in distilled water at room temperature. The fungus can survive on drying in darkness for 5 hours, and 1 hour if also exposed to light.
Incubation Period	Unknown.	Unknown.	Unknown, but speculated to be 1 to 3 days.	Unknown.

Period of Communicability	If untreated, patients with streptococcal pharyngitis are infective during the acute phase of the illness, usually 7-10 days, and for one week afterwards; however, if antibiotics are used, the infective period is reduced to 24 hours. The bacterium can remain in the body in its carrier state without causing illness in the host for weeks or months and is transmissible in this state.	Unknown, but likely for as long as persistence of infection or symptoms, but symptoms not required.	In humans, these bacteria are often located in the upper respiratory tracts of healthy individuals where they colonize and multiply. Communicable period is therefore variable. May be communicable via coughing or sneezing, likely for as long as persistence of infection or symptoms, but symptoms not required.	Although rare, person to person transmission can occur.
Infectious Dose(s)	Unknown	Unknown.	Unknown for humans. Mice developed sepsis or pneumonia when infected with 10^7 or 10^8 cfu, and after infection with 10^4 bacteria, *S. pneumoniae* are able to cross the blood-brain barrier.	Unknown.
Typical Presenting Symptoms	This bacterium is responsible for a wide array of infections. It can cause acute illness which is characterized by pharyngitis leading to sore throat, fever, enlarged tonsils, tonsillar exudate, sensitive cervical lymph nodes and malaise. Skin and soft-tissue infections including pyroderma, cellulitis, wound infections, abcesses, erysipelas, and necrotizing fasciitis can also result from dermal exposure and infection. Septicaemia, septic arthritis, endophthalmitis, myositis, osteomyelitis, pneumonitis, meningitis, endocarditis, nephritis, glomerulonephritis, and sinusitis can also result from infection with Group C streptococcal species. Streptococcal toxic shock-like syndrome consisting initially of dizziness, confusion, and the development of large patches of reddened skin which can lead to	Enterococci infection will vary according to the site of infection. If bloodstream infection (bacteremia or septicemia), fever, elevated heart rate, and malaise are common. In severe cases shock can result which is indicated by dizziness, confusion, hypotension, weakness and fainting that can lead to life threatening conditions. Urinary tract infections present as pain or burning during urination, back pain, difficulty urinating, frequent urination, or fever. Wound, and soft tissue infections may be red with warm skin and swelling, pain, and may or may not have pus or pus drainage. They are also associated with endocarditis and in some cases pneumonia which is characterized by fever, coughing, shaking chills, and shortness of	*S. pneumonia* colonizes in the mucosal surfaces of the nasopharynx and upper respiratory airway, and symptoms of inflammation appear as the bacteria migrate into the sterile parts of the airway. It is the most common etiological agent of community-acquired pneumonia and otitis media, and the second most prevalent cause of bacterial meningitis in Israel (*Neisseria meningitides* being the first). Early symptoms include shaking chills and high fever, and coughs producing pink to rusty coloured sputum. If left untreated, sustained fever and pleuritic pain will develop, as	*C. albicans* is a commensal pathogen as it is a member of the gastrointestinal, oropharyngeal and female genital flora. However, it is also an opportunistic pathogen in humans, as it can cause disease in immunodeficient and immuno-competent individuals that can be life-threatening. The most frequent clinical form is thrush/oral candidiasis, where infection can be observed on the tongue, palate or other mucosal surfaces and is characterized by single or multiple, ragged white patches. Esophageal candidiasis is manifested by inflammatory patches that develop on the esophagus, causing painful swallowing and substernal chest pain. In immuno-compromised patients (such as those with HIV infection), similar lesions can also occur on the

	hypotension, necrotizing fasciitis, and eventual multi organ system failure (respiratory, renal, and liver).	breath, especially when climbing stairs.	well as sinusitis, endocarditis, arthritis, and peritonitis. When pneumococci migrate to the lungs, they can cause pneumonia, or can enter the blood stream and cause bacteremia or septicemia. Uncontrolled colonization of *S. pneumonia* in the lung, meninges, or middle ear will cause pneumococcal lysis, which can trigger inflammation. Mortality rate of pneumococcal pneumonia is 5 – 10% despite antimicrobial treatment, a higher mortality rate as been observed in patients with pneumococcal bacteremia, as it has been approximately 25 – 29% in the past four decades. *S pneumoniae* infection is an important cause of bacterial co-infection in patients with influenza and can increase the morbidity and mortality in these patients.	small intestine and stomach. Chronic mucocutaneous candidiasis is a rare genetic disease, which occurs in individuals with defects in immune response against *Candida*. It involves chronic infections of the skin, hair, face, scalp and hands, and can further disseminate to deeper tissues and major body organs such as kidneys, heart and brain, which may lead to septicimea (candidemia – *Candida* in blood) and death. Infections of the nail (paronychial and onychomycotic candidosis), superficial invasion of mucous membranes, cutaneous infections of the macerated skin (in crural folds, diaper area in infants), eye infections such as endophthalmitis are also examples of infections caused by *C. albicans.*
Mode(s) of Decontamination	Susceptible to 1% sodium hypochlorite, 4% formaldehyde, 2% glutaraldehyde, 70% ethanol, 70% propanol, 2% peracetic acid, 3-6% hydrogen peroxide and 0.16% iodine. Bacteria are susceptible to moist heat (121°C for at least 15 minutes) and dry heat (170°C for at least 1 hour).	Susceptible to 70% isopropyl alcohol, 70% ethanol, 0.041% sodium hypochlorite, phenolic and quaternary ammonia compounds, and glutaraldehyde. Resistant to 3% hydrogen peroxide. Enterococci are killed by temperatures in excess of 80°C.	Exposure to 0.5% glutaraldehyde, 1% sodium hypochlorite, iodines, 70% ethanol, and formaldehyde (effective at higher temperatures than 20˚C) have been shown to disinfect *S. pneumonia*. Cells can be inactivated by heat suspension in a water bath at 56˚C for 30 minutes.	*Candida albicans* strains can be killed effectively with sodium hyphochlorite (5% and 0.5%), iodine (2%) and potassium iodide (4%) within 30 seconds. Chlorhexidine acetate (0.5%) is able to completely kill *C. albicans* strains within 5 minutes. *C. albicans* strains are resistant to calcium hydroxide. *C. albicans* isolates are also susceptible to 70% ethanol, 0.5% ecodiol and a combination of 1.2% sodium hyphochlorite and 0.5% ecodiol. UV light has been shown to reduce fungal load, but is ineffective in killing the

				yeast completely. Most microorganisms are also inactivated by moist heat (121°C for 15 min- 30 min).
Emergency Response	If exposure is suspected wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms. Confirm infection by bacteriological and serological testing, latex bead agglutination, fluorescent antibody staining or ELISA. Appropriate antibiotic treatment is necessary for a Group C Streptococcus infection. Antibiotic effectiveness may vary from case to case and condition to condition. Group C Streptococcal species have demonstrated susceptibility to a variety of drugs: β-lactams such as penicillin, as well as erythromycin, clindamycin, imipenem, rifampin, vanomycin, macrolides and lincomycin; however, certain strains of the bacterium have been found to resistant to macrolides, lincomycin, chloramphenicol, tetracyclines and cotrimoxazole.	If exposure is suspected wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms. Diagnosis is via isolation of enterococci from clinical specimens. Confirm infection by bacteriological and serological testing, latex bead agglutination, fluorescent antibody staining or ELISA. Treatment with penicillin or ampicillin for infections such as urinary tract infection, peritonitis, and wound infections. Combination therapy of a cell wall- active agent (penicillin, ampicillin or vancomycin) and an aminoglycoside is required for the treatment of endocarditis and possibly meningitis. Most strains remain susceptible to penicillin, ampicillin, and vancomycin**.** Strains resistant to β-lactams, aminoglycosides and, increasingly, vancomycin have been described. Strains have also been identified which carry genetic elements conferring resistance to chloramphenicol, tetracyclines, macrolides, lincosamides, quinolones, and streptogramins.	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms. Gram-negative stains or bacteriology studies of direct smears from nasal fluid, fluid from lesions, or areas of inflammation can be used to detect symptomatic or asymptomatic infections. ELISA, PCR fingerprinting analysis, and radiography techniques are also useful for diagnosis. Administer appropriate drug treatment. Inflammation caused by pneumococcal lysis makes the treatment of pneumococcal diseases less effective with antibiotics alone, and even highly effective bactericidals such as β-lactam may actually enhance the harmful effects of the disease in some cases. Susceptible to penicillin, tetracycline, cefotaxime, levofloxacin, erythromycin, and fluoroquinolones, especially moxifloxacin and gatifloxacin. The bacteria may display full susceptibility to telithromycin, vancomycin, and linezolid. Multi-drug resistant *S. pneumoniae* is emerging. It displays high resistance to penicillin, as well as to erythromycin,	If exposure is suspected, wash area thoroughly and encourage bleeding if sharps or puncture injury. Seek immediate emergency medical attention. Appropriate biotherapy and ongoing surveillance are recommended. Direct examination of the fungus or the fungus in culture in the clinical specimen can confirm the presence of infection if key characteristics (size and shape of yeast, presence of pseudophypahe, blastoconidia, chlamydospores, and absence of arthroconidia and capsule) are observed. Other methods include biochemical tests, serological methods such as RIA and ELISA, and molecular biology methods such as REA (restriction enzyme analysis) PCR, and PGFE (pulse-field gel electrophoresis). Eliminating predisposing factors such as administration of antibiotics, steroids, and immuno-suppressants; humidity, local maceration, vaginal pH, removal of infected catheter can help in resolving infections. Susceptibility has been shown for amphotericin B, nystatin, flucytosine, the azoles, echinocandins, and combination drug therapies. Topical and oral azoles such as butoconazole, clotrimazole, triazole, and econazole lipogel can be used against vaginal candidiasis. Systemic antifungals such as fluconazole or itraconazole can be used to treat mucocutaneous candida infections. Voriconazole, and

			cefotaxime, levofloxacin, tetracycline, TMP/SMX, β-lactam agents, macrolides, chloramphenicol, and ceftriaxone, with fluoroquinolone-resistant strains being resistant to ciprofloxacin and levofloxacin treatments. Recent widespread use of a pneumococcal capsular polysaccharide (CPS) conjugate vaccine reduces the incidences of carriage of the bacteria in children, and, as a result, herd immunity has also been observed. Protection, however, is serotype-specific, but development of promising vaccines are focusing on viral pneumococcal proteins that are common to all serotypes. Pneumococcal vaccination is recommended in Canada for infants less than 2 years of age, adults over 65 years and others at high risk of invasive pneumococcal disease. Currently available vaccines are the pneumococcal conjugate vaccine (PCV7) which is effective against 7 serotypes, and it has been shown to be effective for children less than 2 years of age. The pneumococcal polysaccharide vaccine (PPV23) is effective against 23 serotypes.	echinocandins can be effective against cutaneous candidiasis although an azole is preferred, and invasive candidiasis can be treated with caspofungin, or lipid formulations of amphotericin B. Posaconazole is used to treat oral, but not systemic candidiasis. Resistance of *C. albicans* to fluconazole has been associated with repeated use of this drug, particularly in immuno-suppressed patients who are taking this drug chronically for prophylaxis. Resistance to echinocandins has also been reported.

Suggested Reference: http://www.phac-aspc.gc.ca/lab-bio/res/psds-ftss/index-eng.php.

Medical Surveillance Inventory – Bacteria (cont)

Listeria monocytogenes;
Nocardia spp.;
Streptococcus salivarius (S. viridans);
Pseudomonas aeruginosa

Pathogen Identification	***Listeria monocytogenes***	***Nocardia spp.***	***Streptococcus salivarius (S. viridans)***	***Pseudomonas aeruginosa.***
Mode(s) of Transmission	The predominant mode of *L. monocytogenes* transmission is by ingestion of contaminated food. *L. monocytogenes* can also be transmitted transplacentally from mother to child during pregnancy and via the birth canal during birth. Direct contact with diseased animals may lead to transmission to farmers and veterinarians during the birthing of domestic farm animals. Nosocomial infections and person-to-person transmission (excluding vertical) are recognised but rare. *L. monocytogenes* is commonly found in nature, particularly in association with soil. Blood, cerebrospinal fluid, faeces, placenta, skin lesions, pus, amniotic fluid, menstrual blood, lochia, respiratory secretions, meconium, gastric aspirate, animal tissues/specimens, and infected organs such as brain and liver are all potential sources of infection. Accidental autoinoculation, exposure to the tissues of experimentally infected animals, and *L. monocytogenes* cultures, are the primary hazards associated with working with this pathogen.	Inhalation of infectious aerosols, dust, contamination of mucocutaneous lesions, and accidental parental inoculation are the primary hazards associated with working with this pathogen. *Nocardia* have been isolated from sputum, sinus tissues, bronchial aspirates, pus, and rarely from urine and blood.	Inhalation of infectious aerosols, contamination of mucocutaneous lesions, and accidental inoculation are the primary hazards associated with working with this pathogen. Direct contact with infectious persons, materials, and objects can also facilitate transmission. Blood, peritoneal fluid, cerebrospinal fluid, and oropharyngeal secretions are all potential carriers of this bacterium. Studies in the 1930s and 1940s suggest that *S. salivarius* can survive on drinking glass rims and utensils for at least a couple of days; however, genetic identification was not available at this time, and it is unknown whether species-specific identification was possible.	*P.aeruginosa* have been found to survive within droplet nuclei and can remain in aerosols for long periods of time, thus there is evidence of potential airborne transmission. Contact with contaminated water is also a major route, but since the oral infectious dose is thought to be very high, routes that pose the greatest health risk are skin exposure (for example, in contaminated hot tub water) and lung exposure from inhaling aerosols discharged from infected respiratory tracts. The bacterial can often enter the body through injuries and wounds. The use of contaminated mechanical respiratory ventilators in hospital settings is also a common source of nosocomial infections. Inhalation of infectious aerosols, contamination of mucocutaneous lesions, and accidental inoculation are the primary hazards associated with working with this pathogen. *Pseudomonas* can survive for months on dry surfaces and inanimate objects, and are one of the bacteria most frequently isolated from patients with nosocomial infections; humidity can improve persistence. Growth

				observed in distilled water can survive up to months with minimal nutrients.
Incubation Period	Can vary depending on the mode of transmission and dose received, but typically ranges from 1 to 4 weeks, and can be as high as several months. Febrile gastroenteritis as a result of *L. monocytogenes* has a short incubation period, typically 18 to 20 hours.	Unknown.	Unknown.	Varies according to infection, eye infection can appear 24 – 72 hours after infection.
Period of Communicability	Unknown, however person to person transmission, while recognized as possible, is rare.	The bacterium is not transmitted from human to human but rather through external contact with it in the environment.	Unknown, but human to human transmission has been recorded.	Spread of infection from person-to-person is speculated to be highly possible during infection, especially amongst cystic fibrosis patients.
Infectious Dose(s)	The approximate infective dose of *L. monocytogenes* is estimated to be 10 to 100 million colony forming units (CFU) in healthy hosts, and only 0.1 to 10 million CFU in individuals at high risk of infection.	Unknown.	Unknown.	Unknown.
Typical Presenting Symptoms	Although relatively rare, human listeriosis is often severe and mortality rates can approach 50%. Certain factors predispose individuals to infection with *L. monocytogenes*, such as neonates, pregnancy, leukemia, Hodgkin's disease, diabetes mellitus, alcoholism or cirrhosis and immunosuppressive or cytostatic therapy. Most commonly, listeria causes a mild febrile illness however, several types of listeriosis disease manifestations are recognised; for instance, listeriosis in pregnancy, listeriosis of the central nervous system (CNS), febrile gastroenteritis, glandular listeriosis, local listeriosis, typhoid listeriosis, and atypical listeriosis. *Listeriosis in pregnancy*: Occurs mostly during the third trimester, and is characterised by a "flu like" illness	Nocardial infections are predominantly opportunistic infections. As such, patients who are immunosuppressed (transplant patients and patients with HIV, systematic lupus erythematosis, chronic granulomatous disease, malignancies, trauma, or intravenous catheters) are at higher risk of contracting nocardiosis. Nocardial infections occur in three main forms: pulmonary, systemic, and cutaneous. Pulmonary infections usually cause acute, chronic, or relapsing broncho-pneumonia that sometimes spreads to cavities and pleura. Symptoms include coughing, dyspnea, and fever. Pulmonary infection can lead to systemic or neurologic complications such as meningitis and brain abscesses.	*S. salivarius* has been associated with a variety of infections. The most common reports refer to meningitis, and bacteraemia. Other cases include pericarditis, spontaneous bacterial peritonitis, acute jejunitis, pancreatic abscess, multimicrobial endocarditis, early neonatal sepsis, sinusitis, endophthalmitits, bullous impetigo and femoral osteitis. It must be noted, however, that although *S. salivarius* frequently enter the bloodstream, infections with *S. salivarius* are rare due to their low virulence. Many patients with *S. salivarius*bacteraemia have predisposing local factors	As opportunistic pathogens, *Pseudomonas* sp. often invades the host tissue and cause infection and bacteremia in immuno-compromised hosts (e.g., HIV/AIDS, cystic fibrosis, bronchiectasis, and severe chronic obstructive pulmonary disease, burns, malignancy, or diabetes mellitus). The common site of infection is the lower respiratory tract, and severity ranges from colonization without immunological response to severe necrotizing broncho-pneumonia; such severe infection in patients with cystic fibrosis is almost impossible to eradicate once established in the airways. Pseudomonal pneumonia often develops from oro-pharyngeal contamination or secondary bacteremia, and is also a common cause of nosocomial ventilator-related

	with symptoms such as fever, chills, malaise, arthralgia, back pain, and diarrhoea. In many cases the infection is subclinical or unapparent; however, intrauterine infection of the foetus can lead to foetal death, spontaneous abortion, premature delivery, or the birth of a foetus that dies shortly after birth. Surviving newborns with listeriosis are often classified as "early onset" or "late onset". Early onset neonatal listeriosis due to transplacental infection often presents as pneumonia and/or sepsis. Severe disease can result in widespread granulomas (granulomatosis infantisepticum). Late onset neonatal listeriosis is said to occur from infection during birth, with neonates showing symptoms of meningitis one to several weeks after birth. In both early and late onset neonatal listeriosis, the mortality rate ranges from 20 to 30%. *Listeriosis of the CNS*: Meningitis is the most frequently recognised listerial infection. Common symptoms of listeriosis of the CNS include high fever, nuchal rigidity, tremor and/or ataxia, and seizures. The most common form of non-meningitic form of CNS listeriosis is encephalitis involving the brainstem (rhombencephalitis)F*ebrile gastroenteritis*: A non-invasive form of listeriosis that manifests as symptoms typical of gastroenteritis, for example, fever, diarrhoea, and vomiting. *Glandular listeriosis*: Resembles infectious mononucleosis with swelling of the salivary glands and nuchal lymph nodes. *Local listeriosis*: Can manifest as papules and pustules on the hands and	*Nocardia* brain abscesses are commonly found in the brain stem, basal ganglia, and cerebral cortex, and the mortality rate for patients who develop brain abscesses is about 50%. Systemic infections of *Norcardia* are rare and commonly occur in immuno-compromised individuals. Cutaneous infection, called mycetoma, is characterized by pustules, fever, tender lymphadenitis in local lymph nodes, abscess, and yellow-white grainy discharge. Mycetoma can affect the underlying bone and is most common in adult males who walk bare foot or who have burn injuries. With appropriate antibiotic treatment, it is usually healed within a few weeks, though it may take up to 3 months for full clearance.	such as mucosal disruption and/or serious underlying diseases, such as malignancy or liver cirrhosis.	pneumonia in intensive care settings. Infections also include endocarditis, osteomyelitis, urinary tract infections, gastrointestinal infections, meningitis, and, commonly, septicaemia. *P. aeruginosa* is the most common agent associated with infection and inflammation during contact lens wear. The bacteria colonize on lenses and produce proteases to kill or invade corneal cells, an infection that can lead to scarring and vision loss. The species is also the most virulent with a mortality rate of 30%, which can be higher depending on predisposing conditions. *P. aeruginosa* can also readily colonize on open wounds, causing infections, abscesses, and sepsis, with edema and/or discoloration of unburned skin at wound margins and green pigment in subcutaneous fat. *P. aeruginosa* is also associated with swimmer's ear (otitis externa). Other *Pseudo-monas* species are also opportunistic; however, cases of infection are rare.

	arms following direct contact with infectious material, and can be accompanied by constitutional symptoms (fever, myalgia, and/or headache). *Typhoid listeriosis*: Characterised by high fever and is particularly frequent in immunocompromised individuals. *Atypical listeriosis*: Rare cases of have been described with symptoms such as endocarditis, purulent (mononuclear) pleural exudates, pneumonia, urethritis, and abscesses.			
Mode(s) of Decontamination	At room temperature, *L. monocytogenes* is susceptible to sodium hypochlorite, iodophor compounds, and quaternary ammonium compounds. Five to 10-fold higher concentrations of the above compounds are required at 4°C. *L. monocytogenes* can be inactivated by ozone, high pressure (500MPa), and high temperatures (at least 70°C for 2 minutes). *L. monocytogenes* can tolerate cold temperature environments well, and can survive at low pH.	Susceptible to 2-5% phenol, 1% sodium hypochlorite, 4% formaldehyde, 2% glutaraldehyde, 70% ethanol, 70% propanol, 2% peracetic acid, 3-6% hydrogen peroxide, and iodine. Bacteria are susceptible to moist heat (121°C for at least 15 minutes) and dry heat (160-170°C for at least 1 hour). It is able to remain viable for 8 hours at 50°C.	Susceptible to 5.25% sodium hypochlorite, and cresophene (30% paramonochlorophenol, 5% thymol, 0.1% dexamethasone), 21% alcohol, and 2.0% chlorohexidine. Streptococcal species are inactivated at low pH. Bacteria are susceptible to moist heat (121°C for at least 15 minutes) and dry heat (170°C for at least 1 hour).	Susceptibility has been shown for 1% sodium hypochlorite, 70% ethanol, 2% glutaraldehyde, and formaldehyde; however, it has been found to be resistant to disinfectants that are used to treat drinking water such as chlorine, chloramines, ozone, and iodine. Certain adapted stains have been found to be able to grow in disinfectants; however, isopropyl alcohol 4% v/v or ethyl alcohol 6% v/v are effective disinfectants. Inactivation and sterilization using moist heat should be at 121°C for 15 minutes or longer, dry heat at 170-250 °C or higher for 30 minutes or more.
Emergency Response	If exposure is suspected wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms. Listeriosis can be diagnosed in the laboratory by cultivation of the organism, and demonstration of the infectious agent or its products in tissues or body fluids. Several commercially available kits exist for the detection of *L. monocytogenes*. These rapid procedures are based on ELISA and	If exposure is suspected wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms. ELISA for detection of *Nocardia* -specific mAbs,PCR, sequence-based identification methods, and microscopic observation can be used to identify the presence of species in the host Appropriate antibiotic therapy is used to treat clinical disease caused by this bacterium. In	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms. Confirm infection using gram-stain, followed by isolation of the organism from blood or cerebrospinal fluid culture. PCR has also been used to identify	If exposure is suspected, wash area thoroughly and encourage bleeding if sharps or puncture injury. Seek immediate emergency medical attention. Appropriate biotherapy and ongoing surveillance are recommended. Aminoglycoside with β-lactam penicillin is usually the first line of treatment. Aggressive treatment can avoid development of chronic infection. Wounds should be cleaned with surgical detergent disinfectants and/or topical

	PCR technology; however, none have been validated for use as a diagnostic tool. Treatment for human listeriosis with ampicillin or amoxicillin together with gentamicin is the primary choice of therapy. The recommended course of treatment is ampicillin for 2 to 4 weeks. The addition of gentamicin for 2 weeks should be considered for immunocompromised patients. An alternative therapy for individuals allergic to β-lactams is intravenous co-trimoxazole. Susceptible to most broad spectrum and gram-positive spectrum antibiotics, except the cephalosporins are active against *L. monocytogenes in vitro*. *In vivo*, the most active are ampicillin and amoxicillin.	some cases of mycetoma, surgical drainage may be needed. Most species are susceptible to trimethoprim-sulfamethoxazole, amikacin, ampicillin, carbenicillin, broad-spectrum cephalosporins, minocycline, erythromycin, ciprofloxacin, clindamycin, imipenem, and cotrimoxazol, but resistant to penicillin and antituberculous, and antifungal agents.	*S. salivarius* in clinical samples. Antibiotic therapy, typically with ceftriaxone, amoxicillin, and/or vancomycin. Treatment is delivered depending on the manifestation of the infection, for example patients suffering from meningitis due to *S.salivarius* may require mechanical ventilation. Sensitive to various antibiotics, including ciprofloxacin, levofloxacin, metronidazole, penicillin, amoxicillin, ceftriaxone, clindamycin, rifampicin, gentamycin, cefuroxime, moxifloxacin, ceftoxime, and vancomycin. Certain strains of *S. salivarius* have shown partial resistance to penicillin, ceftriaxone, erythromycin, and meropenem.	antibacterial ointments, such as mupirocin. *Pseudomonas* spp. are resistant to many antibiotics. Susceptibility to extended-spectrum penicillins (such as ticarcillin, azlocillin, and piperacillin), aminoglycosides, cephalosporins, fluoroquinolones, polymixins, and the monobactams. Multi-drug resistant strains are emerging, such as against carbenicillin, cephalosporins, ceftazidime, and ciprofloxacin.
Suggested Reference: http://www.phac-aspc.gc.ca/lab-bio/res/psds-ftss/index-eng.php.				

Medical Surveillance Inventory – Bacteria (cont)

Proteus spp.;
Bacillus cereus;
Citrobacter diversus;
Enterobacter spp. (*E. cloacae, E. aerogenes*)

Pathogen Identification	***Proteus spp***	***Bacillus cereus***	***Citrobacter diversus.***	***Enterobacter* spp. (*E. cloacae, E. aerogenes)***
Mode(s) of Transmission	*Proteus* spp. are part of the human intestinal flora and can cause infection upon leaving this location. They may also be transmitted through ingestion or by accidental parenteral inoculation. The specific, natural mode of transmission, however, has not been identified. In the laboratory, accidental contact with aerosols, parenteral inoculation, trauma of the cutaneous or mucocutaneous barriers, and/or ingestion of contaminated material are all infectious risk factors. Urine tract, wound, blood, and in some cases, CSF, stool, tears, and respiratory fluid may all be infectious sources of this group of pathogens. *Proteus* spp. survive only for a few days on inanimate surfaces; and only 1 to 2 days in the case of *P. vulgaris*. They also survive well within the environment in soil, water, and sewage.	The primary mode of transmission is via the accidental ingestion of *B. cereus* contaminated materials. It forms spores and spreads easily. In hospitals, *B. cereus* can also be transmitted via contact with contaminated fabrics. In the laboratory, accidental parenteral inoculation, trauma of the cutaneous or mucocutaneous barriers, and/or ingestion of contaminated material are all infectious risk factors. Lab samples at risk for infectious material contamination may include stool samples, food specimens, and soil samples. *B. cereus* survives in soil and on vegetation, and is generally heat-resistant and thus may survive thermal food processing with or without injury to cells.	Transmission is possible through accidental ingestion or parenteral inoculation with contaminated materials. Person-to-person transmission is also possible. Human feces, brain abscesses, cerebral fluids, laboratory mice, eye, urine, intestines, umbilicus, skins pustules, hands, environmental sources (soil, water) are possible sources of infectious contamination.	Transmission is through direct or indirect contact of infected specimens with mucous membranes, parenteral inoculation, trauma of the cutaneous or mucocutaneous barriers, aerosols, and ingestion. Direct or indirect contact of mucosal surfaces with infectious agent (e.g. bacteria can transfer from contaminated hands or contaminated urinals) or, in the case of endogenous flora, through transfer to adjacent, susceptible, sterile body sites. Entero-bacteriaceae can also be spread through the fecal-oral route. Infected urine, feces, respiratory secretions, wound exudates, blood, water, soil, and plants are all potential sources of infectious contamination.
Incubation Period	Unknown.	The diarrheal form of *B cereus* has an onset period of 8-16 h while the emetic form has an onset period of 1-6 h. Recovery is usually complete in 24 h.	Unknown in adults. In neonates time ranges from hours to days.	Unknown.
Period of Communicability	*Proteus* spp. are not known to be transmitted from person-to-person.	Not known to be transmitted from person-to-person.	Unknown.	Variable. In some cases, the bacteria can be present as resident flora in the absence of symptoms and can be

				transmitted for as long as the carrier state persists.
Infectious Dose(s)	Unknown.	In diarrheal illness, the toxin responsible is produced by organisms in the small intestine and infective dose is 10^4-10^9 cells per gram of food. The emetic toxin is preformed and indigested in food (about 10^5-10^8 cells per gram in order to produce sufficient toxin).	Via ingestion: Approximately 10^7 CFU/mL. Parenteral inoculation dose unknown.	Unknown, however, approximately 1000 cells have been considered infectious, similar to the infectious dose of the pathogenic bacteria *Neisseria meningitidis* , *Escherichia coli* O157, and *Listeria monocytogenes* 4b.
Typical Presenting Symptoms	Generally, affect the upper urinary tract (common site of infection), causing infections such as urolithiasis (stone formation in kidney or bladder), cystitis, and acute pyelonephritis. Rare cases of bacteraemia, associated with UTIs, with *Proteus* spp. have also been reported. Other infections include septicaemia and wound infections. After attachment and colonization within the urinary tract, *Proteus* spp. release urease, which catalyzes the conversion of urea into ammonia and CO_2. This causes a decrease in the urine pH and may eventually lead to the formation of kidney or bladder stones. *P. mirabilis* causes the most infections among all *Proteus* spp.	*B. cereus* causes self-limiting (24-48 hours) food-poisoning syndromes (a diarrheal type and an emetic type), opportunistic infections and is associated with clinical infections such as endophthalmitis and other ocular infections. The diarrheal form of *B. cereus* food poisoning is characterized by abdominal cramps, profuse watery diarrhea, and rectal tenesmus, and, occasionally, fever and vomiting. The emetic form of B. *cereus* food poisoning is characterized by nausea, vomiting, and malaise, occasionally with diarrhea. *B. cereus* can cause wound infections, bacteremia, septicaemia, meningitis, pneumonia, central nervous system infections, endocarditis, pericarditis, respiratory infections, and peripheral infections. Infection in immunocompromised individuals can be life-threatening. *B. cereus*strains which harbour a plasmid bearing *B. anthracis*-like virulence factors can cause severe pneumonia in immuno-competent people.	*Citrobacter* normally cause urinary tract infections, blood stream infections, intra abdominal sepsis, brain abscesses, and pneumonia and other neonatal infection, such as meningitis, neonatal sepsis, joint infection or general bacteremia. CNS infections are more common for infants under 2 months old than for older children or immunocompromised adult patients, but rare cases have been reported. May cause neonatal meningitis that can lead to brain abscesses. *Citrobacter* infections can be fatal, with 33-48 % overall death rates, and 30% for neonates. Infant survivors may experience significant damage to CNS, including profound retardation, hemiparesis, seizures, etc.	*Enterobacter* spp., particularly *E. aerogenes* and *E. cloacae,* have been associated with nosocomial outbreaks, and are considered opportunistic pathogens. *Enterobacter* spp. can cause numerous infections, including cerebral abscess (mental process inhibition, decreases speech, sensation and motor skills, changes in vision and personality and vomiting), pneumonia (cough, fever, shaking chills, shortness of breath, especially when climbing stairs), meningitis (fever, cold hands and feet, drowsiness, confusion, irritability, severe muscle pain, blotchy skin, sever headache, and stiff neck), septicemia (fecer and shaking chills, or a very low body temperature, decreased urination, rapid pulse, rapid breathing, nausea and vomiting, and diarrhea), wound infection (increased pain, redness, swelling, and warmth around the area, red streaking extending from the area, pus drainage, and fever), urinary tract infection (upper back and side (flank) pain high fever shaking and chills nausea, vomiting, pelvic pressure lower abdomen discomfort frequent,

				painful urination blood in urine), and abdominal cavity/intestinal infections (tenderness, pain, bloating, nausea and vomiting, diarrhea, constipation, loss of appetite). In addition, *Enterobacter* spp. have been noted in intravascular device-related infections, and surgical site infections (primarily postoperative or related to devices such as biliary stents). Many species can cause extra-intestinal infections, for example, *Enterobacter sakazakii,* has been associated with brain abscesses in infants and with meningitis. Mortality rates for bacterial meningitis range from 40-80%.
Mode(s) of Decontamination	Generally susceptible to a number of disinfectants including phenolic compounds, hypochlorites (1% sodium hypochlorite), alcohols (70% ethanol), formaldehyde (18.5 g/L; 5% formalin in water), glutaraldehyde, and iodines (0.075 g/L). Bacteria are generally sensitive to moist heat (121 °C for at least 15 minutes) and dry heat (160 to 170 °C for at least 1 hour).	Gluteraldehyde is a chemical agent used to sterilize bacillus-contaminated material. Spores can be killed by 1% sodium hypochlorite, paracetic acid, activated hydrogen peroxide, chlorine dioxide, formaldehyde, iodine, acids, alkali. These chemical agents should be highly concentrated and required greater time of contact to kill spores. Oazolidinones are also effective antibacterial agents for *B. cereus*. *B. cereus* can be inactivated by pulse electric field in 0.15 % NaCl solution. *B. cereus* spores can be resistant to heat and radiation, but heating at 100°C for 5 minutes results in cellular damage to the membranes and ribosomes. Gamma irradiation at 2-5 kGy is required to inactivate *B. cereus* cells.	Phenolic disinfectants, 1% sodium hypochlorite, 70% ethanol, formaldehyde, glutaraldehyde, iodophore and paracetic acid are effective against *Citrobacter*. Ethanol (0.41M), chlorhexidine detergent scrub, hexachlorophene or iodophor preparations and bleach may also be effective. 90% of the *Citrobacter* organisms may be killed after 15 minutes at 230 MPa. *Citrobacter* are also inactivated by UV, microwave, gamma radiation, moist heat (121°C for at least 20 min) and dry heat (165-170°C for 2 h).	While information specific to *Enterobacter* spp. is not available, most species in the family Entero-bacteriaceae are susceptible to 70-80 % ethanol and most vegetative bacteria are also susceptible to 1% sodium hypochlorite, glutaraldehyde, formaldehyde, iodines, hydrogen peroxide, peracetic acid, and quaternary ammonium compounds. *Enterobacter sakazakii* have been shown to be inactivated by pulsed electric fields and high hydrostatic pressure. While additional information specific to *Enterobacter* spp. is unavailable, most vegetative bacteria can be inactivated by moist heat (121 °C for 15 min- 30 min) and dry heat (160-170 °C for 1-2 hours).

Emergency Response	If exposure is suspected, wash area thoroughly and encourage bleeding if sharps or puncture injury. Seek immediate emergency medical attention. *Proteus* spp. can be diagnosed by isolation and differentiation with chromogenic media (i.e. by means of cultured organisms from urine and bloods samples). Administer appropriate antibiotic therapy where necessary. Other than that, treatment is mainly for symptoms. *Proteus* spp. are generally susceptible to broad-spectrum cephalosporins, aminoglycosides, and imipenem. *P. mirabilis* is also susceptible to trimethoprim-sulfamethoxazole, ampicillin, amoxicillin, and piperacillin. *P. vulgaris* and *P. penneri* are also susceptible to cefoxitin, cefepime, and aztreonam. *P. mirabilis* is resistant to nitrofurantoin. Resistance to ciprofloxacin may develop with unrestricted use.*P. vulgaris* and *P. penneri* are resistant to piperacillin, amoxicillin, ampicillin, cefoperazone, cefuroxime, and cefazolin. *P. penneri* is more resistant to penicillin than *P. vulgaris*. Resistance to β-lactamases among *Proteus* spp. is emerging. Carbapenem resistance, including pan-resistant isolates, have been described in India.	If exposure is suspected, wash area thoroughly and encourage bleeding if sharps or puncture injury. Seek immediate emergency medical attention. Monitor for symptoms. *B. cereus* strains can be isolated and grown in laboratory media at 37°C. Specimen isolated from contaminated human stool can be grown with tryptic soy broth with polymyxin. The organism can be isolated in *B. cereus*medium, i.e. in mannitol, egg yolk, polymyxin B agar (MEYP) or polymyxin B, egg yolk, mannitol, bromthymol blue agar (PEMBA). Immunological assays, polymerase chain reaction and biological tests, have been used to detect the enterotoxin activity of *B. cereus*. Isolation of greater than 10^5 organisms/g from contaminated food can confirm *B. cereus* contamination. Administer appropriate drug therapy with supportive treatment. Oral rehydration therapy is the treatment for acute food poisoning syndromes, and antibiotics are seldom required. Patients are given corticosteroids and antibiotics as a first line treatment for eye infections from *B. cereus*. Whenever gram-positive rods are discovered in the blood or the cerebrospinal fluid of an immunocompromised patient with clinical signs of infection, the empiric antibiotic treatment should cover *B. cereus* (*B. cereus* is usually sensitive to clindamycin, aminoglycosides, vancomycin, chloramphenicol, and erythromycin). In cases of an acute non-inflammatory infectious diarrhea, a pharmacologic prophylaxis with bismuth subsalicylate in a dose of two	If exposure is suspected, wash area thoroughly and encourage bleeding if sharps or puncture injury. Seek immediate emergency medical attention. Monitor for symptoms. *Citrobacter* can be detected by serological methods, molecular methods, or polymerase chain reaction (PCR) generated DNA finger prints from mother to child. Infection can also be confirmed by other bacteriological and serological means. Administer appropriate drug therapy. *Citrobacter* spp. are susceptible to aminoglycosides, chloramphenicol, imipenim/cilastatin, trimetoprim, and trimetoprim/sulfamethazole. Resistance has been shown against cephalosporins, ceftazidime , pipera-cillintazobactam, antipseudomona penicillins, ampicillin, cephalothin, and carbenicillin. Prophylaxis consists of antibiotics such as amoxicillin and a beta-lactamase inhibitor.	If exposure is suspected, wash area thoroughly and encourage bleeding if sharps or puncture injury. Seek immediate emergency medical attention. Monitor for symptoms. Stool specimen should be observed for presence of blood or mucous. *Enterobacter* species can be isolated by plating into MacConkey agar, eosin methylene blue agar or blood agar. PCR assays for the detection and identification of *Enterobacter* spp. have also been developed. Administer appropriate antibiotics accounting for local antimicrobial susceptibility patterns. Most *Entero-bacter* spp. are susceptible to cefepime, aminoglycosides, fluoroquinolones, and trimethoprim-sulfamethoxazoleTigecycline has been shown effective in vitro. E*nterobacter spp.* are resistant to ampicillin; first- and second-generation cephalosporins; and cephalothin.

		tablets four times daily with meals and at bedtime may be useful. Duration of use should not exceed 3 weeks. *B. cereus* is susceptible to imipenem and vancomycin, and most strains are sensitive to chloramphenicol, aminoglycosides, ciprofloxacin, erythromycin, and gentamicin. Some strains were moderately sensitive to clindamycin and tetracycline. Clindamycin with gentamicin, given early, is the best treatment for ophthalmic infections from *B. cereus*. *B. cereus* produce large amounts of β lactamase and are resistant to penicillin, ampicillin, cephalosporins, trimethoprim.		
Suggested Reference: http://www.phac-aspc.gc.ca/lab-bio/res/psds-ftss/index-eng.php.				

Medical Surveillance Inventory – Bacteria (cont)

Haemophilus influenzae;

Pasteurella sp.;

Salmonella enterica spp.;

Staphylococcus aureus (MRSA, MSSA, VISA, hVISA, VRSA)

Pathogen Identification	***Haemophilus influenzae***	***Pasteurella sp.***	***Salmonella enterica* spp.**	***Staphylococcus aureus* (MRSA, MSSA, VISA, hVISA, VRSA)**
Mode(s) of Transmission	Respiratory droplet transmission as well as via contact with discharge from nose and throat during infectious periods. The portal of entry is most commonly the nasopharynx. Bacteria may be in the cerebrospinal fluid, serum, urine, blood, pleural fluid, joint fluid and middle ear aspirates. Lab acquired infections are caused by inhalation, autoinoculation, or ingestion. does not survive long term in the environment, but can survive more than 18 hrs in mucous and 12 hrs on plastic.	Transmission occurs primarily by bites and scratches of infected animals, animal licks on injury site, respiratory droplets, and infected meat. Cockroaches may spread pasteurellosis if they come in contact with food. Laboratory hazards include laboratory animal bites, exposure to infectious aerosols, contact with broken cutaneous or mucocutaneous barriers, and parenteral inoculation. Infected bite wounds and abscesses, pus, bronchial secretion, CSF, and blood may contain *Pasteurella* spp. *P. multocida* may survive in air (5% after 45 min), in distilled water and ocean water (14 days at 4°C, less than 24 hours at 37°C), and in pig slurry (3 days at 4°C and 6 days at 37°C). It may also survive in blood.	Primary hazards when working with *Salmonella enterica* are accidental parenteral inoculation, exposure of non-intact mucocutaneous and cutaneous barriers, and ingestion. The risk associated with aerosol exposure is not yet known. Human infection usually occurs when consuming contaminated foods and water, contact with infected feces, as well as contact with infective animals, animal feed, or humans. Foods that pose a higher risk include meat, poultry, milk products, and egg products. In hospitals, the bacteria have been spread by personnel in pediatric wards, either on their hands or on inadequately disinfected scopes. Flies can infect foods which can also be a risk for transmission to humans. Serotype Choleraesuis can survive in wet swine feces for at least 3 months and in dry swine feces for at least 13 months. Serotype Dublin can survive in feces spread on concrete, rubber, and polyester for almost six years. Serotype	Ingestion of food containing enterotoxins. Vertical transmission during vaginal delivery is uncommon. Person-to-person transmission occurs through contact with a purulent lesion or with a carrier. Unsanitary conditions and crowded community settings increase exposure to *S. aureus*. Infection may be spread from person-to-person through health care workers or patients. Nasal colonization can lead to auto-infection. Trauma of cutaneous barrier, parenteral inoculation, ingestion of infected material, and contact with aerosols are the primary lab associated hazards. Direct contact with open cuts and lesions of skin is also a risk. May become carrier by touching contaminated surfaces and objects with intact epidermis. Infective stages may be present in CSF, joint aspirates, blood, abscesses, aerosols, faeces, and urine. Survives on carcasses and organs (up to 42 days), floors (less than 7 days), glass (46 hours), sunlight (17 hours), UV (7 hours), meat products (60 days), coins (up to 7 days), skin (30 minutes to 38 days). Depending on colony size, *S. aureus* can survive on fabrics from days to months.

			Typhimurium can survive in cattle slurry for 19-60 days, cattle manure for 48 days, soil for 231 days, and water for up to 152 days. Flies have been shown to excrete certain serotypes for 8 days and bed bugs can excrete bacilli for up to 21 days. Certain serotypes have been shown to survive on fingertips for up to 80 minutes, depending on the inoculum size. *Salmonella* serotypes have been found to live up to 63 days on lettuce, 231 days on parsley, 32 weeks in pecans, 10 months on refrigerated cheddar cheese, 9 months in butter, up to 63 days in frozen yogurt, and up to 20 weeks on frozen minced beef and chicken.	
Incubation Period	2-4 days.	Depends on site of infection, but generally less than 24 hours.	For non-typhoidal salmonellosis, the incubation period is variable, depends on the inoculum size, and usually ranges between 5 and 72 hours. For typhoid fever, the incubation period can be between 3 and 60 days, although most infections occur 7-14 days after contamination. The incubation period for typhoid fever is highly variable and depends on inoculum size, host susceptibility, and the bacterial strain.	If ingested, 30 minutes to 8 hours is expected, otherwise symptoms may appear 1 to 10 days post infection. However, people may be colonized intermittently or for months-years before a symptomatic infection occurs, if at all.
Period of Communicability	Not highly contagious. Secondary infection may occur in the case of particularly close contact with infected individuals. Not communicable 48 h after initiation of efficient antibiotic treatment.	*Pasteurella* spp. may be transmitted during colonization. Human-to-human communicability is suspected, but not confirmed.	Humans can spread the disease for as long as they shed the bacterium in their feces. Certain carriers shed the bacteria for years and 5 % of patients recovering from non-typhoidal salmonellosis can shed the bacteria for 20 weeks. Animals	Communicable period is as long as a purulent lesion is present or carrier state persists.

			can have a latent or carrier state where they excrete the organism briefly, intermittently or persistently.	
Infectious Dose(s)	Unknown.	Unknown.	Unknown for parental inoculation. For ingestion, the infectious dose varies with the serotype. For non-typhoidal salmonellosis, the infectious dose is approximately 10^3 bacilli. For enteric fever, the infectious dose is about 10^5 bacilli by ingestion. Patients with achlorhydria, depressed cell-mediated immunity, or who are elderly may become infected with at a lower infectious dose. The infectious dose may also be dependent on the level of acidity in the patient's stomach.	At least 100,000 organisms in humans.
Typical Presenting Symptoms	Infection with *Haemophilus influenzae* (type b) can cause meningitis (50% of all cases - adults and children), epiglottitis (17%), pneumonia (15%), septic arthritis (8%), cellulitis (6%), osteomyelitis (2%), or generalized bacteremia (2%). A small proportion of children (0.5-3%) children will have asymptomatic infections. Meningitis may begin as minor upper respiratory infection. Symptoms include behavioural or mental status change, fever, vomiting, headaches, and signs of meningeal irritation such as bulging fontanelle in infants, or stiff neck in older children or adults. In adults, complete recovery is common with treatment, but for children there is a 2-5% mortality rate, even with treatment. Sequelae such as hearing loss, mental retardation, seizures, vision loss, and motor and speech	50% of pasteurellosis cases in Europe and America are infected bites or scratches from animals, usually cats and dogs 20-80% of cat bites and 3-18% dog bites will be infected. 3-48h after infection, substantial local cellulitis, sometimes accompanied with low fever, appear. If this infection progresses, subcutaneous abscess, osteomyelitis, pneumonia, endocarditis, septic arthritis, pericarditis, brain abscess, liver abscess, renal abscess, bacteremia/septicaemia, conjunctivitis, and lymphangitis may develop. 25% of the cases present as respiratory tract diseases, which may include epiglottitis, sinusitis, tracheobronchitis, pneumonia, empyema, and pulmonary abscesses. Rarely, it may also present as malakoplakia and as a granulomatous pulmonary lesion. The remainder of cases appear as urinary	*Salmonella enterica* can cause four different clinical manifestations: gastroenteritis, bacteremia, enteric fever, and an asymptomatic carrier state. It is more common in children under the age of 5, adults 20-30 year olds, and patients 70 years or older. Gastroenteritis: Gastroenteritis or "food poisoning" is usually characterized by sudden nausea, vomiting, abdominal cramps, diarrhea, headache chills and fever up to 39 ºC. The symptoms can be mild to severe and may last between 5-7 days. The Typhimurium serotype is the most common cause of gastroenteritis and there are an estimated 1.3 billion cases and 3 million deaths annually (1.4 million cases and 600 deaths in	*Staphylococcus aureus* is an opportunistic pathogen that can cause a variety of self-limiting to life-threatening diseases in humans. The bacteria are a leading cause of food poisoning, resulting from the consumption of food contaminated with enterotoxins. Staphylococcal food intoxication involves rapid onset of nausea, vomiting, abdominal pain, cramps, and diarrhea. Symptoms usually resolve after 24 hours. Animal bites can result in local infections, cellulitis, erythema, tenderness, mild fever, adenopathy, and lymphangitis (rarely). Scalded skin syndrome is caused by exfoliative toxins secreted on the epidermis and mostly affects neonates and young children. Other skin conditions caused by Staphylococcal exfoliative toxins include blisters, skin loss, pimples, furuncles, impetigo, folliculitis, abscesses, poor temperature

	delay may develop in 15-30% of cases. Epiglotitis is an acute infection of the upper airway that causes oedema and inflammation of the epiglottis and adjacent tissues and may lead to complete airway obstruction in hours. Symptoms include severe sore throat and fever. Speaking, swallowing or breathing may be impaired and respiration may be noisy. To assist with breathing, patients may adopt a tripod or sniffing posture. Airways obstruction results in a 5-10% mortality rate. Cellulitis often affects the face, head or neck. Cellulitis causes localized tissue inflammation and may lead to proptosis, loss of visual acuity, limitation of extraoccular movement and death. 12-25% of affected children may have concomitant meningitis. Other illnesses associated with infection include pneumonia (pulmonary infection with purulent excretion), osteomyelitis (bone infection), septic arthritis (joint infection) and pericaditis (infection of the pericardial membrane surrounding the heart).	tract infections, sepsis, or meningitis. Pasteurellosis may lead to death with an overall mortality rate of 30% among reported cases.	the US alone) due to non-typhoidal *Salmonella*. In well resourced countries with low levels of invasive complications, the mortality rate due to non-typhoidal *Salmonella* is lower then 1%; however, in developing countries, the mortality rate can be as high as 24%. Bacteremia: Bacteremia occurs in 3-10% of individuals infected with *Salmonella enterica*and certain serotypes (particularly serotype Choleraesuis) have higher mortality rates. Immunosuppressed individuals and patients with comorbid medical conditions (e.g. HIV-AIDS, diabetes, mellitus, malignancy, corrhosis, chronic granulomatous disease, sickle cell disease, lymphoproliferative disease, or collagen vascular disease) have a higher risk of developing bacteremia due to a *Salmonella* infection. Bacteremia can cause septic shock; endocarditis, especially in patients older then 50 or with heart conditions; infection of the aorta, especially in patients with pre-existing atherosclerotic disease; liver, spleen, and biliary tract infections in patients with underlying structural abnormalities; mesenteric lymphadenitis; osteomyelitis in long bones and vertebrae; urinary tract infection; pneumonia; pulmonary abscess; brain abscess; subdural and epidural empyema; meningitis; CNS infections (rarely); and death. Enteric fever: Also known	control, fluid loss, and secondary infection. *S. aureus* can also cause necrotizing fasciitis in immuno-compromised individuals, although this is very rare. Necrotizing fasciitis is life-threatening and causes severe morbidity. Certain strains of *S. aureus* produce the superantigen TSST-1, which is responsible for 75% of toxic shock syndrome (TSS) cases. The clinical presentation of TSS is severe and acute symptoms include high fever, vascular collapse, vomiting, diarrhea, myalgia, hypotension, erythematous rash, desquamation, and involvement of at least 3 organs. Mortality is very high and death can occur within 2 hours. Deep infections include endocarditis, peritonitis, necrotizing pneumonia, bacteremia, meningitis, osteomyelitis, septic arthritis, and infections of bones, joints and organs.

			as typhoid fever, this infection is caused by serotypes Typhi and Paratyphi. Enteric fever is characterized by fever (rising within 72 hours after the onset of illness) and headache, brachycardia, faint rose-colored rash on the abdomen and chest, anorexia, abdominal pain, myalgias, malaise, diarrhea (more common in children) or constipation (more common in adults), hepatosplenomegaly, segmental ileus, meningismus, and neuropsychiatric manifestations. Less common symptoms are sore throat, cough, and bloody diarrhoea. Complications include myocarditis, encephalopathy, intravascular coagulation, infections of the biliary tree and intestinal tract, urinary tract infection, and metastatic lesions in bone, joints, liver, and meninges. The most severe complication (occurs in about 3% of patients) is haemorrhage due to perforations of the terminal ileum of proximal colon walls. If untreated, the fever can last for weeks; however, with proper antimicrobial therapy, patients usually recover within 10-14 days. The disease is milder in children and, if treated, has a mortality rate of less then 1%; untreated cases can have a mortality rate greater then 10 %.	
Mode(s) of Decontamination	Phenolic disinfectants, 1% sodium hypochlorite, 70% ethanol, formaldehyde, glutaraldehyde, iodophore and peracedic acid are	Phenolic disinfectants, 1% sodium hypochlorite, 70% ethanol, formaldehyde, glutaraldehyde, iodophore, and peracetic acid are	Gram negative bacteria are susceptible to 2-5% phenol, 1% sodium hypochlorite, 4% formaldehyde, 2%	Susceptible to 70% ethanol, clorhexidine, 1% hypochlorite, 70% ethanol, formaldehyde, glutaraldehyde, iodophore, and peracetic acid are

	effective disinfectants. Inactivated by UV, microwave, and gamma radiation, moist heat (121°C for at least 20 min), and dry heat (165-170°C for 2 h.	effective against *Pasterella* spp. *Pasteurella* spp. are inactivated by UV, microwave, gamma radiation, moist heat (121°C for at least 20 min), and dry heat (165-170°C for 2 h).	glutaraldehyde, 70% ethanol, 70% propanol, 2% peracetic acid, 3-6% hydrogen peroxide, quaternary ammonium compounds and iodophors; however, *Salmonella* spp. is resistant to nitrites. Susceptible to moist heat (121°C for at least 15 minutes) and dry heat (170°C for at least 1 hour). *Salmonella* spp. can also be disinfected with ozone.	effective against *Serratia* sp.; also inactivated by UV, microwave, gamma radiation, moist heat (121°C for at least 20 min), and dry heat (165-170°C for 2 h).
Emergency Response	If exposure is suspected, wash area thoroughly and encourage bleeding if sharps or puncture injury. Seek immediate emergency medical attention. The primary treatment for *Haemophilus influenzae* is appropriate antibiotics. Intravenous antibiotics are often required but depending on illness, oral administration may follow for 7-10 days. If airways are blocked, more invasive procedures may be indicated. Susceptible to chloramphenicol and third generation cephalosporins (e.g. cefotaxime, ceftriaxone, and cefuroxime). Resistance has been shown for ampicillin, co-trimoxazole, clarithromycin, tetracycline, chloramphenicol, and rifampicin. Currently, vaccination targets the PRP antigen and is effective for Hibbut not other *Haemophilus influenzae* serotpyes. Vaccine is usually given between 2 months and five years of age. Rifampin prophylaxis is indicated for direct contacts, as directed by a doctor. Pregnant women should not receive prophylactic treatment.	If exposure is suspected, wash area thoroughly and encourage bleeding if sharps or puncture injury. Seek immediate emergency medical attention. Monitor for symptoms and confirm bacteriologically and by PCR. Give appropriate antibiotic therapy. Sensitive to penicillins and doxycycline. Antibiotics such as penicillin or derivative are given with clinical observation of infected bites.	If exposure is suspected, wash area thoroughly and encourage bleeding if sharps or puncture injury. Seek immediate emergency medical attention. Confirm diagnosis by isolation from stool or blood and by serotyping to identify the serotype. Treatment depends on the clinical symptoms presented by the patient. Gastrotenteritis: Fluid and electrolyte replacement as well as control of the nausea and vomiting are the usual treatments for these symptoms. Antibiotic treatment is not usually used; however, it may be necessary for neonates, children, the elderly, and the immunosuppressed, in which case ciproflaxin, co-trimoxazole, ampicillin, and cephalosporins may be used. Bacteremia: Antibiotic treatment is used to treat bacteremia (e.g. ciproflaxin, co-trimoxazole, ampicillin, or cephalosporins), especially for neonates, children, the elderly, and the immunosuppressed. Asymptomatic carrier state:	If exposure is suspected, wash area thoroughly and encourage bleeding if sharps or puncture injury. Seek immediate emergency medical attention. Appropriate biotherapy and ongoing surveillance are recommended.

			Carriers can be treated with ciproflaxin in order to reduce the spread of the infectious agent. Susceptible to chloramphenicol, ciproflaxin, amoxicillin, co-trimoxazole, trimethprim-sulfonamid, cephalosporins and norfloxacin. Some resistance to chloramphenicol has been reported and, in 1989, 32% of strains were multi-drug resistant.	

Suggested Reference: http://www.phac-aspc.gc.ca/lab-bio/res/psds-ftss/index-eng.php.

Medical Surveillance Inventory – Bacteria (cont)

Bordatella bronchiseptica;

Serratia sp.;

Klebsiella spp.;

Vancomycin-resistant Enterococci (VRE)

Pathogen Identification	***Bordetella bronchiseptica***	***Serratia sp.***	***Klebsiella spp.***	**Vancomycin-resistant Enterococci (VRE)**
Mode(s) of Transmission	Transmission can occur through direct contact with respiratory secretions, fomites, or inhalation of infected aerosol. Primary lab hazards include parenteral inoculation, contact with non-intact cutaneous and mucocutaneous barriers, and exposure of mucous membranes to infectious aerosols. *Bordetella* species survive only for a few hours in respiratory secretions. *B. bronchiseptica* has been shown to survive for 24 weeks in phosphate-buffered saline and lake water at 10°C and at 37°C, without any nutritional supplement. *B. bronchiseptica* can also survive in soil for 45 days.	Ingestion of contaminated foods and direct contact. Nosocomial transmission may occur by hand contact from hospital personnel and other patients. Accidental parenteral inoculation, droplets exposure of mucous membrane, infectious aerosols, and ingestion and contact with non-intact mucocutaneous and cutaneous barriers. Fomites may also spread *Serratia*. *S. marcescens* may survive from 3 days to 2 month on dry, inanimate surfaces, and 5 weeks on dry floor. The organism may survive less than 4 days in a blood bag under aerobic conditions and 20 days in semi-anaerobic/ anaerobic conditions. It has been also reported to survive in contact lens disinfectant (with chlorheximide), double-distilled water, non-medicated hand soap, but no duration has been reported for those cases. *Serratia* spp. are found in feces, wound exudates, respiratory specimen, blood, eye culture, and urine.	*Klebsiella* spp. can be transmitted through skin contact with environmentally contaminated surfaces and/or objects; examples include Loofah sponges, medical equipment, sewage, and blood products. Fecal transmission has also been suggested for some cases of bacteremia caused by *Klebsiella* spp. *K. rhinoscleromatis* can be transmitted from person-to-person via airborne secretions; however, prolonged contact with infected individuals is required for infection. *K. granulomatis* are sexually transmitted. They may also be vertically transmitted (from mother to child) or by accidental inoculation. Transmission rates between partners are low (<50%) compared to other sexually transmitted diseases. Sources for clinical samples of *Klebsiella* spp. primarily include samples from respiratory tract (RT; nasopharyngeal samples) and	Inhalation of infectious aerosols, contamination of mucocutaneous lesions, and accidental inoculation are the primary hazards associated with working with this pathogen. Direct contact with infectious persons, materials, and objects can also facilitate transmission. Blood, urine, wound samples, and feces are all potential carriers of this bacterium. In addition, Enterococci can grow and survive in harsh environments, and can persist almost anywhere including soil, plants, water, and food. Can survive 5 days to 4 months on dry inanimate surfaces.

			urinary tract (UT), and blood. Laboratory hazards include direct contact of mucosal membranes with contaminated surfaces and/or object, and inhalation of infectious airborne secretions, accidental parenteral inoculation and/or ingestion.	
Incubation Period	Unknown; however, one report described a 5-year-old girl who became sick 10-12 days after being exposed to an infected rabbit. Other human infections with this pathogen have been reported but none of the cases was incubation period described.	Unknown.	Not clearly understood. According to some sources, the incubation period for *K. granulomatis* is usually 1 to 6 weeks.	Unknown.
Period of Communicability	Unknown, but spread of infection from person-to-person via respiratory droplets is possible during infection.	*Serratia sp. may be transmissible between people but rates are unknown.*	Members of *Klebsiella* spp. can be transmitted from person-to-person; however, the communicability period is unknown. Approximately one-third of people carry *Klebsiellae* in their stools; detection rates according to different studies vary from 5% to 36%. Detection rates in nasopharynx vary from 1% to 6%. Hospital personnel have been shown to frequently carry *Klebsiellae* on their hands.	Unknown, but likely for as long as persistence of infection or symptoms, but symptoms not required.
Infectious Dose(s)	Unknown.	Unknown.	Unknown. According to one source, 10^8 *Klebsiella* organisms per gram of feces are required to produce damage.	Unknown.
Typical Presenting Symptoms	*B. bronchiseptica* is primarily a respiratory tract pathogen found in a variety of animals. It causes atrophic rhinitis in swine, kennel cough in dogs, with nasal	*Serratia* sp. are opportunistic pathogens and are one of the ten most common causes of bacteremia in North America. They are responsible for a variety of	*Klebsiella* spp. have been identified as important common pathogens for nosocomial pneumonia (7 to 14% of all cases), septicaemia	Enterococci infection will vary according to the site of infection. If bloodstream infection (bacteremia or septicemia), fever, elevated heart rate, and malaise are common. In

	turbinate inflammation and turbinate atrophy. It has been implicated as an infrequent cause of infection in humans, primarily in immuno-compromised patients exposed to infected animals, although infections have also been reported in immunocompetent individuals as well. Woolfrey et al., 1991 described 25 patients with *B. bronchiseptica* infections, 56% of whom had an immuno-compromising factor and all except 3 had an exposure to an infected animal. These individuals presented with varied clinical manifestations such as nosocomial tracheobronchitis, acute maxillary sinusitis, peritonitis, septicemia and bacteremia in immuno-compromised individuals and whooping cough/pertussis-like disease in healthy individuals. Many *B. bronchiseptica* infections have been reported in human immunodeficiency virus-infected patients, as Dworkin et al., 1999 described 9 patients with *B. bronchiseptica* infections, all of whom had at least one AIDS defining condition. These patients presented with pneumonia, sinusitis or bronchitis. Two cases of meningitis with *B. bronchiseptica* have also been reported. Severe cases of illness due to B. *bronchiseptica* are often found to be co-infections with toxigenic *Pasteurella multocida*.	infections, including bacteremia, pneumonia, intravenous infections, osteomyelitis, endocarditis, and rarely, endogenous and exogenous endophthalmitis. Symptoms of infection may include heachache, fever, chills, hypotension, vomiting, respiratory distress, erythema, ocular pain, periorbital swelling, cellulitis, ear aches, ear discharge, and hypopyon (pus in the eyes). The mortality rate from bacteremia due to *Serratia* sp. 6 months after infection is 37%. *Serratia* infections in neonates are frequent (11-15% in neonatal intensive care unit) and may include bloodstream infection (42%), conjunctivitis (26%), pneumonia (13%), urinary tract infection (8%), meningitis (7%), and surgical site infections. Other infections in infants are documented (otitis externa, enterocolitis and omphalitis, gastroenteritis, septic arthritis, and intraperitoneal infection/abcess), but are rare. Risk factors include birth weight, use of mechanical ventilation, and gestational age (under 37 weeks are at greater risk). The mortality rate in neonates is 44%.	(4 to 15%), urinary tract infection (UTIs; 6 to 17%), wound infections (2 to 4%), intensive care unit (ICU) infections (4 to 17%), and neonatal septicaemias (3 to 20%). *Klebsiella* spp. can also cause bacteremias and hepatic infections, and have been isolated from a number of unusual infection, including endocarditis, primary gas-containing mediastinal abscess, peritonitis, acute cholecystitis, crepitant myonecrosis, pyomyositis, necrotizing fasciitis, psoas muscle abscess, fascial space infections of the head and neck, and septic arthritis. They are also important opportunistic pathogens, particularly among the immuno-compromised. Respiratory disease: Infection of the upper lobe is more common. Symptoms include: fevers, chills, and leukocytosis with red currant jelly-like sputum. Rare complications include lung infection involving necrosis and sloughing of the entire lobe. Chronic nose inflammation has also been observed as ozena, or rhinoscleroma. Central nervous system (CNS) infections including meningitis and cerebral abscesses. Clinical symptoms include: headaches, fever, altered consciousness,	severe cases shock can result which is indicated by dizziness, confusion, hypotension, weakness and fainting that can lead to life threatening conditions. Urinary tract infections present as pain or burning during urination, back pain, difficulty urinating, frequent urination, or fever. Wound, and soft tissue infections may be red with warm skin and swelling, pain, and may or may not have pus or pus drainage. They are also associated with endocarditis and in some cases pneumonia which is characterized by fever, coughing, shaking chills, and shortness of breath, especially when climbing stairs.

			seizures, and septic shock. Can also cause urinary tract infections, pyogenic liver abscesses (symptoms including fever, right-upper-quadrant pain, nausea, vomiting, diarrhoea or abdominal pain, and leukocytosis. Abscesses occur predominantly in the right lobe and are solitary.) and donovanosis or granuloma, a chronic ulcerative disease that primarily affects the genitalia. Symptoms include development of small papule or ulcer at the site of inoculation that later develop into large red ulcers (lesions) that extend along the moist folds of the genitalia.	
Mode(s) of Decontamination	Information specific to *B. bronciseptica* is not available, but most vegetative bacteria have been shown to be susceptible to low concentrations of chlorine (<1ppm), 70% ethanol, phenolics such as orthophenylphenol and ortho-benzyl-para-chlorophenol, 2% aqueous glutaraldehyde, peracetic acid (0.001% to 0.2%). Information specific to *B. bronchiseptica* in not available, but most vegetative bacteria can be inactivated by moist heat (121°C for 15 min- 30 min) and dry heat (160-170°C for 1-2 hours).	Phenolic disinfectants, 1% sodium hypochlorite, 70% ethanol, formaldehyde, glutaraldehyde, iodophore, and peracetic acid are effective against *Serratia* spp. *Serratia* spp. are inactivated by UV, microwave, gamma radiation, moist heat (121°C for at least 20 min), and dry heat (165-170°C for 2 h).	Gram-negative bacteria are generally susceptible to a number of disinfectants, including phenolic compounds, hypochlorites (1% sodium hypochlorite), alcohols (70% ethanol), formaldehyde (18.5 g/L; 5% formalin in water), glutaraldehyde, and iodines (0.075 g/L). Reduction in the growth and metabolic activity of *K. pneumoniae* at temperatures >35 °C has been reported. Significant growth reduction has been demonstrated at 60 °C; however, the bacteria still show some metabolic activity (i.e. not completely inactivated). Bacteria can be inactivated by moist heat	Susceptible to 70% isopropyl alcohol, 70% ethanol, 0.041% sodium hypochlorite, phenolic and quaternary ammonia compounds, and glutaraldehyde. Resistant to 3% hydrogen peroxide. Enterococci are killed by temperatures in excess of 80°C.

			(121°C for 15 min- 30 min) and dry heat (160-170°C for 1-2 hours).	
Emergency Response	If exposure is suspected, wash area thoroughly and encourage bleeding if sharps or puncture injury. Seek immediate emergency medical attention. Monitor for symptoms. Diagnosis can be made by culturing the bacteria from clinical specimens, or via molecular methods such as PCR. MacConkey agar or Regan-Lowe agar (RL agar) media can be used for culturing *B. bronchiseptica* from clinical specimens. Since *B. bronchiseptica* is sensitive to cephalexin, cephalexin is replaced by methicillin or oxacillin in the RL agar media to allow its growth. Administer appropriate drug therapy. No specific guidelines have been described for the treatment of *B. bronchiseptica*. Patients are generally treated with aminoglycosides, extended-spectrum third-generation penicillin, tetracycline, and susceptibility to quinolone and trimethoprim-sulfamethoxazole is variable but it can generally be administered to children as oral treatment. Antibiotics course of 2 to 4 weeks is recommended to treat the disease. Longer courses, of up to 6 months, have been required for treatment in some patients. No vaccines or prophylaxis are available for humans. Susceptible *in vitro* to aminoglycosides (amikacin, gentamicin, and tobramycin), penicillins such as azlocillin, mezlocillin, piperacillin, and ticarcillin, broad septrum cephalosporins, tetracyclines, erythromycin, choramphenicol, and	If exposure is suspected, wash area thoroughly and encourage bleeding if sharps or puncture injury. Seek immediate emergency medical attention. Appropriate biotherapy and ongoing surveillance are recommended. *Serratia* spp. are usually susceptible to aminoglycosides, fluoroquinolones, and co-trimazole. Many *Serratia* spp. isolates (39-73%) are resistant to gentamicin. They are all resistant to penicillins and cephalosporin.	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek immediate medical attention. Monitor for symptoms. Other tests include isolating strains of the bacteria or typing different isolates. This is often necessary for investigation of endemic and epidemic nosocomial infections and also for epidemiological investigations from the environment. *Klebsiellae* can be isolated by growth in media. Although there are specific chromogenic media available for isolating these bacteria from specific samples, *Klebsiellae* grow well on blood and non-differential media. Biotyping and serotyping are two common forms of typing methods used for typing *Klebsiella* spp. Serotyping is the most widely used technique for typing these bacteria which involves detection of capsular antigens by means of antisera. Serotyping tests include: quelling reaction, immunofluorescence, double-diffusion gel precipitation, counter-current immuno-electrophoreses, *Staphylococcus* coagulation, and latex coagulation methods.	If exposure is suspected wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms. Diagnosis is via isolation of enterococci from clinical specimens. Confirm infection by bacteriological and serological testing, latex bead agglutination, fluorescent antibody staining or ELISA. Treatment with penicillin or ampicillin for infections such as urinary tract infection, peritonitis, and wound infections. Combination therapy of a cell wall- active agent (penicillin, ampicillin or vancomycin) and an aminoglycoside is required for the treatment of endocarditis and possibly meningitis. Although this most strains remain susceptible to some antibiotics, this strain is resistant to vancomycin. Some strains have also been identified which carry genetic elements conferring resistance to chloramphenicol, tetracyclines, macrolides, lincosamides, quinolones, and streptogramins.

	trimethoprim-sulfamethoxazole. *B. bronchiseptica* strains are usually resistant to streptomycin, primary penicillins such as penicillin G and ampicillin, miocycline, erythromycin, and ceftriaxone.		Biotyping is not preferred due to the large number of reactions and the amount of time required to complete these tests. Molecular typing methods are also being developed, although they are not commonly used. These include: plasmid analysis, ribotyping, PFGE, and random amplified polymorphic DNA analysis, all of which have been successfully used to track strains epidemiologically. Administer appropriate antibiotic therapy where necessary. *Klebsiella* spp. are known to show resistance to penicillins, especially ampicillin and carbenicillin. Since more and more strains of *Klebsiella* spp. appear to be developing and harbouring extended-spectrum beta-lactamases (ESBLs), cephalosporinases, and carbapenemases, resistance of *Klebsiella* spp. to current antibiotics appears to be increasing. According to results from some studies in Europe and USA, ranges of susceptibility were as follows: ceftazidime (92-95%), ceftriaxone (96-98%), cefotaxime (96%), piperacillin-tazobactam (90-97%), imipeneum (98-100%), gentamicin (95-96%), amikacin (98-99%), triethoprimsulfamethoxazole (SXT) (88-90%). Resistance values tend to be higher for	

			strains isolated from ICU patients compared to non-ICU patients. Pan-resistant isolates have been identified in the Indian subcontinent.	

Suggested Reference: http://www.phac-aspc.gc.ca/lab-bio/res/psds-ftss/index-eng.php.

Pathogen identification, Mode of Transmission, Incubation Period, Period of Communicability, Infectious Dose, Typical Presenting Symptoms, Mode(s) of Decontamination, and Emergency Response:

Untreated Sewage

Medical Surveillance Sewage Bacteria

Legionella pneumophila;

Mycobacterium spp.;

Shigella spp.;

Helicobacter pylori

Pathogen Identification	***Legionella pneumophila***	***Mycobacterium spp.***	***Shigella spp.***	***Helicobacter pylori***
Mode(s) of Transmission	Can be transmitted by aerosols and aspiration of contaminated water and sewage. Lab infection hazards include accidental ingestion of the pathogen, direct contact of open wounds/site of injury with the pathogen, exposure to aerosols, and accidental parenteral inoculation. Sources of *L. pneumophila* in the laboratory include expectorated sputum, lower respiratory specimens, pleural fluid, blood, pericardial fluid, kidney, liver, spleen, myocardium and soft tissues. Other sources include water samples from water systems and fresh water sources.	Nosocomial, direct contact with a contaminated environment. Mycobacteria are able to survive for weeks to months on inanimate objects if protected from sunlight. NTM species are widely distributed in nature and have been found in sewage, natural water, tap water, soil, water used in showers and surgical solutions. NTM can be isolated from sputa, exudates from lesions, tissues, environmental samples (soil, water), and from wounds. MAC has also been isolated from blood, and stool specimens of infected individuals. Primary lab infection hazards include ingestion of the pathogen, direct contact of open wounds/ site of injury with the pathogen, exposure to aerosols, and accidental parenteral inoculation.	Organisms are spread through the fecal-oral route, and transmission is typically through one of three mechanisms: ingestion of contaminated foods (washed with fecally contaminated water, or handled with poor hygiene, commonly in tossed salads, chicken, and shellfish); drinking contaminated water (or in swimming pools); or by person-to-person contact by anal sexual contact. Spread of infection linked to flies has also been recorded. Organisms can also be found in stool samples, but rarely in blood lab samples. The primary lab infection hazard is accidental ingestion, however, direct contact of open wounds/site of injury with the pathogen, exposure to aerosols, and accidental parenteral inoculation are also potential hazards. *Shigella* species have been recently identified to be the most frequently identified agent of laboratory-acquired infections because of their high	The exact route of transmission is unknown, but acquisition is likely to occur during childhood through faecal–oral or oral–oral contact or during gastrointestinal tract transit disorders. Transmission may also occur through food-borne, airborne, or waterborne pathways, as the sewage system has been found to be an agent of dissemination. In its coccoid form, it can survive up to one year in a river-water microcosm, and remains culturable for more than 10 days in 4˚C water. Lab infection hazards include accidental ingestion of the pathogen, direct contact of open wounds/site of injury with the pathogen, exposure to aerosols, and accidental parenteral inoculation. *H. pylori* may be located in the oral cavity, gastrointestinal and hepatobiliary regions of infected mammals and birds. They can also be found in tissues of the small intestine, saliva, gastric juice, and faeces.

			virulence and low infectious dose.	
Incubation Period	Incubation period for Legionnaires' disease is 2-14 days. Pontiac fever has an incubation period of 30 to 90 hours (with 24-48 hours being most common).	Infection with *M. marinum* has an incubation period of about 2-3 weeks.	Ranges from 1 – 7 days. Acute diarrhea can develop within 1 – 2 days. Symptoms and shigellosis may occur within 12 – 50 hours.	Unclear as symptoms usually do not appear until adulthood and observable symptoms may never develop (known as silent infections). Major symptoms such as abdominal pain, heartburn, and nausea have been observed 3 – 4 days after ingestion of the bacteria.
Period of Communicability	No person-to-person transfer has been documented. No documentation of zoonotic (animal-to-human) or animal-to-animal transmission cases have been described.	Not generally transferred from human to human. Transmission is dependent on accidental ingestion or contact with contaminated material source(s).	Agents begin to be shed in feces 4 weeks after infection, and it is communicable as long as the organisms are present in excrement. Although rare, asymptomatic carriers can also spread the infection for up to some months.	Variable and indefinite. Transmission between people usually occurs through oral-oral routes. Carriers can be infectious and symptom free.
Infectious Dose(s)	Unknown.	Unknown.	Infection can result from ingestion of 10 – 200 organisms.	Unknown for humans. Infection in the Rhesus monkey occurred with a minimum of 10^4 *H. pylori* bacteria intake by orogastrical inoculation.
Typical Presenting Symptoms	*L. pneumophila* infection can cause Legionnaires' disease, a severe form of pneumonia. The symptoms of Legionnaire's disease include confusion, headache, diarrhoea, abdominal pain, fever, chills, and myalgia as well as a non-productive cough. Mortality rate is reported to be 15-25%. Pontiac fever is a non-pneumonic form of *L. pneumophila* infection. Symptoms are flu-like, including fever, tiredness, myalgia, headache, sore throat, nausea, and cough may or may not be present. Pontiac fever is self limited and requires no hospitalization or antibiotic therapies. There are no	Non-tuberculous mycobacteria (NTM) infections occur mainly in immunosuppressed individuals, although immunocompetent patients can also be affected. Non tuberculous mycobacteria cause many different diseases in humans: Pulmonary disease: Pulmonary diseases are caused mainly by *M. kansasii*, MAC, and rarely by *M. malmoense, M. xenopi, M. asiaticum*, *M. simiae* and *M. szulga*. Pulmonary disease may show different clinical patterns: tuberculosis like infiltrates, nodular bronchiectasis, solitary nodules, and diffuse infiltrates.	Ingested pathogens can survive gastric acidity and cause illness by infecting the colonic mucosa and multiplying in the colonic epithelial cells, and spreading laterally to adjacent cells. Infection may be mild and asymptomatic, but it is most commonly characterized by acute intestinal infections upon ingestion, resulting in mild watery diarrhea to severe inflammatory bacillary dysentery or shigellosis, manifested by severe abdominal cramps, nausea and vomiting, fever, tenesmus, anorexia, and stool containing	*H. pylori* are not invasive, but colonize in the human stomach's antral region and gastric mucosal surfaces where they release pathogenic proteins that induce cell injury and inflammation. This can result in clinical symptoms of infection, such as duodenal ulcer and gastric adenocarcinoma. Other common illnesses as a result of infection include gastroenteritis, diffuse antral gastritis, and gastric carcinoma. *H. pylori* is a Class I human carcinogen according to the World Health Organization. Infection can last a lifetime in the host if not properly treated,

	reported deaths associated with Pontiac fever. Although up to 85-90% of cases can be attributed to *L. pneumophila*, approximately half of the remaining 49 species in the genus have caused disease in humans. It is conjectured that up to 40% of cases involving these rare pathogens, attributed to species and groups other than *L. pneumophila* sero-group 1, are missed clinically if commercially available urine antigen detection kits, which target only *L. pneumophila* sero-group 1, are used exclusively for diagnosis. Sources of *L. pneumophila* in the laboratory include expectorated sputum, lower respiratory specimens, pleural fluid, blood, pericardial fluid, kidney, liver, spleen, myocardium and soft tissues. Other sources include water samples from water systems and fresh water sources.	Lymphadenitis: caused mainly by MAC, *M. scrofulaceum*, *M. haemophilum*, *M. fortuitum, M. kansasii*. Cutaneous and soft tissue infections (skin ulcers): Cutaneous or skin infections may be associated with *M. marinum*, *M. ulcerans*, *M. fortuitum*, and *M. chelonei* . *M. marinum*can cause cutaneous infection on exposure of broken skin to contaminated freshwater fish tanks. The disease is characterized by a formation of a single papulonodular lesion confined to one extremity, which with time may become ulcerative. *M. ulcerans* causes cutaneous skin ulcers which may vary from a localized nodule to widespread ulcerative or non-ulcerative disease including osteomyelitis. Other infections associated with NTM include enteritis, musculoskeletal disease, bursitis, CNS disease, corneal infections, and otitis media. Disseminated infection and bacteremia may also occur. Disseminated infection occurs mainly in immuno-suppresed individuals, and may involve liver, spleen, bone marrow. Patients with disseminated infection present with nonspecific symptoms such as fever, malaise, weight loss, anorexia, abdominal pain and night sweats.	blood and mucus. Further complications include Reiter's syndrome which has been associated with *S. flexneri*, severe dehydration, intestinal perforation, toxic mega colon, bacteremia, toxaemia, septicaemia, seizures, toxic encephalopathy with headache and alterations of consciousness, septic shock and convulsions (very rare), and haemolytic uremic syndrome, which have been linked to Shiga toxin (a potent cytotoxin produced by *S. dysenteriae* that can also cause other neurotoxic effects). Virulence of *Shigella* is temperature-regulated, as organisms are able to invade HeLa cells at 37°C, and cannot do so *in vitro* at 30°C. Infections are usually self-limiting, but can become life-threatening in immunocompromised patients or if not properly treated. Severity of infection depends on the host, dose, and serotype. *S. dysenteriae* is the most pathogenic species, with a fatality rate up to 20%, whereas *S. sonnei* usually cause mild forms of shigellosis.	causing chronic gastritis which can lead to peptic gastroduodenal ulcer disease. The rate of mortality varies with country and age, but is generally low, being around 2 – 4%.
Mode(s) of Decontamination	*L. pneumophila* is susceptible to 1% solutions of sodium hypochlorite, 2% phenol, 2% gluteraldehyde, isopropyl alcohol, and formaldehyde. *L. pneumophila* can be inactivated in water by UV light, and temperatures of 80 °C	The vegetative state is susceptible to disinfectants such as 70% ethanol, 0.1% sodium hypochlorite, and 0.1N NaOH. Spores may be resistant to disinfectants. Toxins are inactivated (more than 99.7%) by 20 minutes exposure to 3 mg/L free	Susceptible to 1% sodium hypochlorite, 70% ethanol, 2% glutaraldehyde, iodines, phenolics, and formaldehyde . Organisms can be heat-killed by steaming using an autoclave for 1 hour at 100˚C under	Readily inactivated by free chlorine, iodine treatments can inhibit its vacuolation toxin activity, thus practices used for treating drinking water, apart from ozonolysis, should be sufficient for disinfecting *H.*

	for 0.4 minutes when in its aqueous environment. Autoclave conditions of 121 °C for 15 minutes. *L. pneumophila* is found naturally in most fresh water sources, including lakes, ponds, and rivers. It is also found in cooling towers, plumbing systems, water heaters, and warm water spas *L. pneumophila* was found to survive for up to 139 days in distilled water and 415 days in tap water. Can persist outside of the host in biofilms like those formed in potable and health care facility water systems > 98 days. Clinical specimens will usually support the survival of *L. pneumophila* for up to 1 week.	available chlorine (FAC; similar to the military disinfection procedure), and 84% inactivated by a treatment of 20 minutes at 0.4 mg/L FAC (similar to municipal water treatment procedures). Toxin is destroyed after heating for 5 minutes at greater than 85°C. Toxins are detoxified in air within 12 hours and following exposure to sunlight within 1 to 3 hours. Spores are highly resistant to heat and desiccation; therefore, it is recommended to sterilize with dry heat (2 hours at 160°C) by autoclaving (20 minutes at 121°C, 1 atm pressure) and/or by irradiation. Survives well in soil, water and agricultural products.	normal atmospheric pressure. Can survive up to months on dry surfaces, up to 10 days in citric juices and carbonated soft drinks, several days on contaminated vegetables, over 3 hours on fingers, 2 – 28 days on metal utensils at 15°C or 0 – 13 days at 37°C, in feces for 12 days at 25°C, and water for under 3 days. Growth is possible at 25°C – 37°C and bacteria can survive at 5°C on MacConkey agar. Flies can carry *Shigella*for up to 20 – 24 days.	*pylori* as well. Exposing *H. pylori* to 1.1mg/L residual chlorine for 45 minutes is enough to eradicate the pathogen (biocidal properties of chlorine are optimized at lower pH levels, such as around <pH 7 or 6). Inactivated by low pressure UV light at fluences (UV dose) of less than 8 mJ/cm^2. Bacteria can be heat-killed by incubating at 70˚C for 10 minutes, followed by 95˚C for 5 minutes. As culturing *H. pylori* in the laboratory is difficult as it needs adequate conditions of desiccation, air supply, and temperature, it is likely that it does not survive well outside of its host; however, in its coccoid form, it can survive up to one year in a river-water microcosm, and remains culturable for more than 10 days in 4˚C water.
Emergency Response	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek immediate medical attention. Monitor for symptoms. Diagnosis can be confirmed via identification of *L. pneumophila*, often isolated from respiratory secretions, by culturing, immunofluorescent staining, urine antigen tests, PCR or serologic tests. Respiratory fluoroquinolones and the newer macrolides are used to treat *L. pneumophila* pneumonia. Treatment typically lasts 7-10 days or in the case of immunosuppressed patients, 21 days. Pontiac fever usually does not require antimicrobial therapy. *L. pneumophila* is susceptible to erythromyin, clarithromycin,	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek immediate medical attention. Monitor for symptoms. Diagnosis of NTM infection can be done via culture of clinical specimens and identification using phenotypic characteristics (growth rate, colony pigmentation and biochemical tests); histopathological examination to demonstrate the presence of granuloma in aspirates or biopsies; serotyping methods; isoenzyme and protein electro-pherogram based methods; and PCR, DNA fingerprinting and identification using gene probes. **A c**ombination of several antibiotics	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek immediate medical attention. Monitor for symptoms. Serological testing of stool isolates can distinguish and confirm serogroups. Administer appropriate drug therapy. Oral rehydration or electrolyte replacement in dehydrated patients can lead to recovery within days. Antibiotics usually are not needed in mild cases, but should be administered for infections involving *S. dysenteriae*. Antimicrobials may reduce duration of	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek immediate medical attention. Monitor for symptoms. Presence of *H. pylori* can be confirmed by culture, blood antigen detection, urease detection, or bacterial metabolite detection in the infected individual's breath. Administer appropriate drug therapy. *H. pylori* is rapidly developing antibiotic resistance, so antibiotics can be applied with a proton pump inhibitor or a bismuth compound. Such dual, triple or quadruple treatments have been found to be more effective than

	azithromycin, tetracycline, moxifloxin, and levofloxacin. Aminoglycosides such as gentamicin, kanamycin and streptomycin are active against *L. pneumophila*; however, there are mutant strains that show resistance to streptomycin. *Beta*-lactam antibiotics (i.e. penicillin, cephalosporins) are ineffective against *L. pneumophila*.	over long periods of time is recommended for treatment of NTM infections. The most important antibiotics used in antimyco-bacterial therapy include: rifampin, isoniazid, ethambutol, macrolides (clarithromycin, azithromycin), quinolones (ciprofloxacin, moxifloxacin, gatifloxacin), aminoglycosides (streptomycin, amikacin) and linezolid. Surgery may be useful in removing debridement in soft tissue diseases caused by NTM species, and in managing cervical lymphadenitis. Surgery can also be used along with antibiotic therapy to reduce the bacterial load and to cure life threatening symptoms such as hemoptysis. Combinational drug therapy is used as different species are susceptible and resistant to different drugs. Drug susceptibility tests are performed on isolated organisms to guide proper therapy. *M. kansasii* is susceptible to first line tuberculosis drugs (rifampin, isoniazid, pyrazinamide and ethambutol). *M. marinum* is resistant to pyrazinamide, and MAC organisms, unlike *M. kansasii*, are resistant to first line tuberculosis drugs.	infection, carriage state of the patient, and mortality. Other treatments aids for severe cases include mechanical ventilation, anticonvelsants, and inotropics.	administering one antibiotic alone. Sensitivity has been established for clarithromycin, amoxicillin, tetracycline, imipenem, cefaclor, minocycline, simethicone, gabexate mesilate, and ketoconazole. Strains have been found to show resistance to antibodies such as clarithromycin, erythromycin, ofloxacin, and metronidazole, and show low levels of resistance to tetracycline, amoxicillin, fluoroquinolones, and rifabutin. Omeprazole, clarithromycin, and metronidazole can be administered if early symptoms of infection such as heartburn, nausea, or severe epigastric cramps are experienced. Recombinant urease (rUrease) and parenteral vaccine containing *H. pylori* antigens (CagA, VacA, and NAP) in combination with aluminum hydroxide as an adjuvant have been found to be effective vaccines against *H. pylori*, although they cannot prevent re-infection.
Suggested Reference: http://www.phac-aspc.gc.ca/lab-bio/res/psds-ftss/index-eng.php.				

Medical Surveillance Sewage Bacteria – (cont)

Escherichia coli (enterotoxigenic);
Escherichia coli (uropathogenic);
Escherichia coli (entero-hemorrhagic),
Escherichia coli (septicemia causing)

Pathogen Identification	***Escherichia coli* (enterotoxigenic)**	***Escherichia coli* (uropathogenic)**	**Escherichia coli, (entero-hemorrhagic)**	***Escherichia coli* (septicemia causing)**
Mode(s) of Transmission	Ingestion of contaminated food or other materials; fecal-oral transmission. The primary lab infection hazard is accidental ingestion, however, direct contact of open wounds/site of injury with the pathogen, exposure to aerosols, and accidental parenteral inoculation are also potential hazards. Sources of entero-toxigenic *E. coli,* in the laboratory include contaminated food products, faeces, and sewage.	Ingestion of contaminated food or other materials; fecal-oral transmission. The primary lab infection hazard is accidental ingestion, however, direct contact of open wounds/site of injury with the pathogen, exposure to aerosols, and accidental parenteral inoculation are also potential hazards. Sources of entero-toxigenic *E. coli,* in the laboratory include contaminated food products, faeces, and sewage.	Ingestion of contaminated food or other materials; fecal-oral transmission; person-to-person transmission (extremely high). The primary lab infection hazard is accidental ingestion, however, direct contact of open wounds/site of injury with the pathogen, exposure to aerosols, and accidental parenteral inoculation are also potential hazards. Sources of entero-hemorrhagic *E. coli,* in the laboratory include contaminated food products, faeces, and sewage.	Ingestion of contaminated food or other materials; fecal-oral transmission. The primary lab infection hazard is accidental ingestion, however, direct contact of open wounds/site of injury with the pathogen, exposure to aerosols, and accidental parenteral inoculation are also potential hazards. Sources of entero-toxigenic *E. coli,* in the laboratory include contaminated food products, faeces, and sewage.
Incubation Period	Between 14 and 30 hours.	3-8 days.	2-8 days.	Unknown.
Period of Communicability	Can last from days to months.	Can be transferred from human to human via heterosexual intercourse as well as via accidental ingestion or contact with contaminated material source(s).	Can last from days to months.	Not transferred from human to human. Transmission is dependent on contact with contaminated material source(s).
Infectious Dose(s)	The infectious dose of ETEC in adult is estimated to be at least 10^8organisms, but the young, the elderly and the infirm may be susceptible to lower numbers.	Unknown.	Appears to have low infectious dose, may be similar to that of *Shigella* spp.,10 organisms by ingestion.	Unknown.

Typical Presenting Symptoms	Patients with ETEC enteritis usually have an abrupt onset of watery diarrhea that does not contain blood, pus, or mucus (nondysenteric). The diarrhea is usually mild to moderate in severity, but some patients may have severe fluid loss. Low-grade fever, nausea, and abdominal pain may also be present. Dehydration may become severe or life threatening in neonates and children, necessitating aggressive fluid and electrolyte replacement. A self-limited course, with resolution in 2-5 days, is most common in adult travelers who acquire the disease, though some strains of the organism may produce a disease lasting much longer, with a median duration of illness of 7 days. There are an estimated 800,000 deaths each year due to ETEC.	Burning during urination, frequent urges to urinate with very little passage of urine, foul smelling, cloudy, or bloody urine, fever, chills, pelvic pain in women, rectal pain in men, and pain in the lower back, abdomen, hips, or flank.	Hemorrhagic colitis, intestinal disease accompanied by cramps and abdominal pain; initially watery, followed by bloody diarrhea; low grade fever; last about 8 days; 5-10% of hemorrhagic colitis victims may develop hemolytic uremic syndrome (HUS); affects all ages, higher death rates occur in elderly and young; can cause thrombo-cytopenic purpura (TTP) in elderly.	Bacteraemia which can lead to septicemia (symptoms: fever, a rapid heart rate, shaking chills, low blood pressure, gastrointestinal symptoms (abdominal pain, nausea, vomiting, and diarrhea), rapid breathing, and/or confusion).
Mode(s) of Decontamination	Susceptible to a combination of 2,2-dibromo-2-cyanoacetamide (DBA) with sodium iodide (20:80 parts), iodine, 2 % glutaraldehyde, quaternary ammonium (20°C, 0.5 min), hypochlorite (0.525%, 20°C, 0.5 min), phenolic (20°C, 0.5 min), and ethyl alcohol (70%, 20°C, 0.5 min). Ozone can inactivate *E. coli*. *E. coli* are also sensitive to heat treatment, especially at temperatures of 70°C or higher. Can survive for 1.5 hours to 16 months on dry inanimate surfaces.	Susceptible to a combination of 2,2-dibromo-2-cyanoacetamide (DBA) with sodium iodide (20:80 parts), iodine, 2 % glutaraldehyde, quaternary ammonium (20°C, 0.5 min), hypochlorite (0.525%, 20°C, 0.5 min), phenolics (20°C, 0.5 min), and ethyl alcohol (70%, 20°C, 0.5 min). *E. coli* are also sensitive to heat treatment, especially at temperatures of 70°C or higher. Can survive for 1.5 hours to 16 months on dry inanimate surfaces.	Susceptible to many disinfectants - 1% sodium hypochlorite, 70% ethanol, phenolics, glutaraldehyde, iodines, formaldehyde. Heat sensitive, inactivated by moist heat (121° C for at least 15 min) and dry heat (160-170° C for at least 1 hour). Survives well in contaminated feces and soil, only small reduction in organism number over 2 months	Susceptible to a combination of 2,2-dibromo-2-cyanoacetamide (DBA) with sodium iodide (20:80 parts), iodine, 2 % glutaraldehyde, quaternary ammonium (20°C, 0.5 min), hypochlorite (0.525%, 20°C, 0.5 min), phenolics (20°C, 0.5 min), and ethyl alcohol (70%, 20°C, 0.5 min). *E. coli* are also sensitive to heat treatment, especially at temperatures of 70°C or higher. Can survive for 1.5 hours to 16 months on dry inanimate surfaces.
Emergency Response	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek immediate medical attention. Monitor for symptoms. Stool culture is a common method used to identify *E. coli*. ETEC can be detected using non-radioactively labeled oligonucleotides	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek immediate medical attention. Monitor for symptoms. Strain identification by molecular and biochemical means is necessary to determine proper antibiotic	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek immediate medical attention. Monitor for symptoms; confirm bacteriologically, DNA probe to detect Verotoxins VT1 and	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Sepsis is a genuine medical emergency. Seek immediate medical attention. Monitor for symptoms. Adequate organ perfusion should be ensured

	DNA probes and PCR targeted against the LT and ST genes. ELISA can also be used to detect LT and ST. Treatment with trimethoprim/sulfamethoxazole (TMP-SMX) or quinolones reduces the duration of diarrhea. Treatment of fluid and electrolyte loss is usually achieved through oral rehydration. The use of the World Health Organization Oral Rehydration Salts (ORS) solution has been recommended. Intravenous rehydration may be necessary for infants, individuals with excessive vomiting, or those with severe dehydration. Bismuth subsalicylate may decrease the amount of diarrhea and the duration of disease. Antimicrobial therapy is generally not indicated, because of the self-limited nature of this disease. Susceptible to carbapenem, fosfomycin-trometanol, nitrofurantoin, and bovine apo-lactoferrin. *E. coli* can be resistant to chloramphenicol, β lactams, nalidixic acid, ampicillin, and ciprofloxacin. Fluoroquinolones such as ciprofloxacin enhance toxin production.	treatment. Antibiotic treatment, typically with trimethoprim /sulfamethoxazole, ciprofloxacin, Fosfomycin, Nirtofurantoin, Levofloxacinm Cephalexin, Ceftrialone, Azithromycin, and Doxycycline may be effective for eradication of the infecting strain. However, there is documentation of increases in antibiotic resistance, allergic reaction to certain pharmaceuticals, alteration of normal gut flora, and failure to prevent recurrent infections.	VT2. Electrolyte fluid therapy; antibiotics may be administered in very severe cases. Sensitive to a wide spectrum of antibiotics.	and hypotension should be managed with with intravenous administration of supplementary fluids. Ventilatory support should be provided for patients with progressive hypoxia, hypercapnia, altered sensorium, or respiratory muscle fatigue. Blood glucose must be maintained and if present, disseminated intravascular coagulation must be managed via transfusion. Strain identification by molecular and biochemical means is necessary to determine proper antibiotic treatment. Antibiotics should initially be given intravenously. Ideally, antibiotic treatment should start within an hour of diagnosis to reduce the risk of serious complications or death. Intravenous antibiotics are usually replaced by tablets after two to four days.
Suggested Reference: http://www.phac-aspc.gc.ca/lab-bio/res/psds-ftss/index-eng.php.				

Medical Surveillance Sewage Bacteria – (cont)

Staphylococcus aureus (MRSA, MSSA, VISA, hVISA, VRSA);
Salmonella enterica spp.;
Aeormonas hydrophilia;
Campylobacter coli.

Pathogen Identification	***Staphylococcus aureus*, (MRSA, MSSA, VISA, hVISA, VRSA)**	***Salmonella enterica* spp.**	***Aeromonas hydrophila***	***Campylobacter coli***
Mode(s) of Transmission	Ingestion of food containing enterotoxins. Vertical transmission during vaginal delivery is uncommon. Person-to-person transmission occurs through contact with a purulent lesion or with a carrier. Unsanitary conditions and crowded community settings increase exposure to *S. aureus*. Infection may be spread from person-to-person through health care workers or patients. Nasal colonization can lead to auto-infection. Lab infection hazards include accidental ingestion of the pathogen, direct contact of open wounds/site of injury with the pathogen, exposure to aerosols, and accidental parenteral inoculation. Laboratory sources of infection include, but are not limited to: CSF, joint aspirates, blood, abscesses, aerosols, faeces, urine, and contaminated water sources.	Human infection usually occurs when consuming contaminated foods and water, contact with infected feces, as well as contact with infective animals, animal feed, or humans. Foods that pose a higher risk include meat, poultry, milk products, and egg products. In hospitals, the bacteria have been spread by personnel in pediatric wards, either on their hands or on inadequately disinfected scopes. Flies can infect foods which can also be a risk for transmission to humans. Primary lab infection hazards include ingestion of the pathogen, direct contact of open wounds/ site of injury with the pathogen, exposure to aerosols, and accidental parenteral inoculation. All *Salmonella enterica* subspecies (with the exception of serotype Typhi) are found in blood, urine, feces, food and feed and environmental materials. Serotype Typhi is found in blood, urine, feces and bile.	Infection is spread via fecal-oral transmission during direct ingestion or drinking of contaminated water or foods. Infection can also be transmitted by eating contaminated meat, dairy, shrimp, or fish. Primary lab infection hazards include ingestion of the pathogen, direct contact of open wounds/ site of injury with the pathogen, exposure to aerosols, and accidental parenteral inoculation. The bacteria have been isolated from feces, sputum, urine, bile, pus, surgical wounds, medicinal leeches, normal flora, spleen, pleural fluid, placenta, skin lesion, throat, gall bladder, hospital water supplies, dialysis fluids, and blood plasma products.	Oral ingestion of bacteria from contaminated food, contaminated drinking water, and sewage. Contact with animals and their feces is also a source of infection. Primary lab infection hazards include: accidental ingestion, direct contact of open wounds/site of injury with the pathogen, exposure to aerosols, and accidental parenteral inoculation. *Campylobacter* can survive for many weeks in water at 4°C, but only a few days above 15°C, and for 2-10 hours when exposed to drying. Nearly all water sources are contaminated with *C. coli*.
Incubation Period	Onset of symptoms after consuming contaminated food is usually 30 minutes to 8 hours. Colonies of *S.*	For non-typhoidal salmonellosis, the incubation period is variable, depends on the inoculum size, and	The reported incubation period for Aeromonas-associated diarrhea is 1 to 2 days.	1 to 10 days.

	aureus can be carried for an undetermined amount of time; some individuals may carry it chronically, and some may carry it intermittently.	usually ranges between 5 and 72 hours. For typhoid fever, the incubation period can be between 3 and 60 days, although most infections occur 7-14 days after contamination. The incubation period for typhoid fever is highly variable and depends on inoculum size, host susceptibility, and the bacterial strain .	Aeromonas' infections contracted via recreational sporting activities, such as swimming occur as early as 24h post exposure. Cellulitis is the most frequent soft tissue infection and is usually accompanied by systemic signs developing within 8 to 48 h. The length of time from initial *A. hydrophila* infection to bacteremia ranges from 1 to 38 days.	
Period of Communicability	Communicable period is as long as a purulent lesion is present or carrier state persists.	Humans can spread the disease for as long as they shed the bacterium in their feces. Certain carriers shed the bacteria for years and 5 % of patients recovering from non-typhoidal salmonellosis can shed the bacteria for 20 weeks. Animals can have a latent or carrier state where they excrete the organism briefly, intermittently or persistently.	It can be transferred from human-to-human by contact with infected wounds, feces or blood. The pathogen can also be transferred during sports; especially when played in muddy environments involving transfer of infected soil.	Not generally transferred from human to human. Transmission is dependent on accidental ingestion or contact with contaminated material source(s).
Infectious Dose(s)	At least 100,000 organisms in humans.	The infectious dose varies with the serotype. For non-typhoidal salmonellosis, the infectious dose is approximately 10^3 bacilli. For enteric fever, the infectious dose is about 10^5 bacilli by ingestion. Patients with achlorhydria, depressed cell-mediated immunity, or who are elderly may become infected with at a lower infectious dose. The infectious dose may also be dependent on the level of acidity in the patient's stomach.	The infectious dose for humans and animals is greater than 10^{10} organisms.	As low as 500 organisms by ingestion. One volunteer study found that 9000 bacteria were required to infect 50 percent of subjects.
Typical Presenting Symptoms	*Staphylococcus aureus* is an opportunistic pathogen that can cause a variety of self-limiting to life-threatening diseases in humans. The bacteria are a leading cause of food poisoning, resulting from the	*Salmonella enterica* can cause four different clinical manifestations: gastroenteritis, bacteremia, enteric fever, and an asymptomatic carrier state. It is more common in children	Infection with *Aeromonas hydrophila* can result in gastrointestinal or non-gastrointestinal complications. Symptoms of gastrointestinal infection range from watery	A major agent of gastroenteritis and acute enterocolitis in humans, and also of acute diarrheal illnesses in developed countries. Typical symptoms include watery diarrhea that may contain red or

	consumption of food contaminated with enterotoxins. Staphylococcal food intoxication involves rapid onset of nausea, vomiting, abdominal pain, cramps, and diarrhea. Symptoms usually resolve after 24 hours. Animal bites can result in local infections, cellulitis, erythema, tenderness, mild fever, adenopathy, and lymphangitis (rarely). Scalded skin syndrome is caused by exfoliative toxins secreted on the epidermis and mostly affects neonates and young children. Other skin conditions caused by Staphylococcal exfoliative toxins include blisters, skin loss, pimples, furuncles, impetigo, folliculitis, abscesses, poor temperature control, fluid loss, and secondary infection. *S. aureus* can also cause necrotizing fasciitis in immunocompromised individuals, although this is very rare. Necrotizing fasciitis is life-threatening and causes severe morbidity. Certain strains of *S. aureus* produce the superantigen TSST-1, which is responsible for 75% of toxic shock syndrome (TSS) cases. The clinical presentation of TSS is severe and acute symptoms include high fever, vascular collapse, vomiting, diarrhea, myalgia, hypotension, erythematous rash, desquamation, and involvement of at least 3 organs. Mortality is very high and death can occur within 2 hours. Toxic shock syndrome is associated with vaginal colonization with toxin-producing *S. aureus* during menstruation, complications with staphylococcal infection at other sites, or complications of surgical procedures. Deep infections include endocarditis, peritonitis, necrotizing pneumonia, bacteremia, meningitis,	under the age of 5, adults 20-30 year olds, and patients 70 years or older. Gastroenteritis: Gastroenteritis or "food poisoning" is usually characterized by sudden nausea, vomiting, abdominal cramps, diarrhea, headache chills and fever up to 39 ºC. The symptoms can be mild to severe and may last between 5-7 days. The Typhimurium serotype is the most common cause of gastroenteritis and there are an estimated 1.3 billion cases and 3 million deaths annually (1.4 million cases and 600 deaths in the US alone) due to non-typhoidal *Salmonella*. In well resourced countries with low levels of invasive complications, the mortality rate due to non-typhoidal*Salmonella* is lower then 1%; however, in developing countries, the mortality rate can be as high as 24%. Bacteremia: Bacteremia occurs in 3-10% of individuals infected with *Salmonella enterica*and certain serotypes (particularly serotype Choleraesuis) have higher mortality rates. Immunosuppressed individuals and patients with comorbid medical conditions (e.g. HIV-AIDS, diabetes, mellitus, malignancy, corrhosis, chronic granulomatous disease, sickle cell disease, lymphoproliferative disease, or collagen vascular disease) have a higher risk of developing bacteremia due to a *Salmonella* infection. Bacteremia can cause septic shock; endocarditis, especially in patients older then 50 or with heart conditions; infection of the aorta, especially in patients with pre-	diarrhea to dysenteric or bloody diarrhea. Chronic infection is also possible. Non-gastrointestinal complications that may arise subsequent to *A. hydrophila* infection include hemolytic syndrome and kidney disease, cellulitis, wound and soft-tissue infection, meningitis, bacteremia and septicemia, ocular infections, pneumonia and respiratory tract infections, urinary tract infection in neonates, osteomyelitis, peritonitis and acute cholecystitis. Severe infection can result from non resolved intermittent diarrhea, which can occur months after the initial infection. *A. hydrophila* can cause disease in aquatic animals, such as red leg disease in frogs which is caused by endotoxin and haemolysin produced by the bacteria and can be fatal.	white blood cells, inflammatory enterocolitis, abdominal pain, fever, malaise, nausea and vomiting. Symptoms usually last for about a week, with relapses occurring in 5-10% of cases if untreated, and persistent symptoms may be observed in immuno-compromised patients. A large number of campylobacter infections are asymptomatic; however, although the illness is generally mild, many complications can be preceded by enteritis, including bacteremia, hepatitis, cholecystitis, pancreatitis, urinary tract infection, abortion, myocarditis and meningitis. In developing countries where infections are endemic, the majority of symptomatic cases occur in young children.

	osteomyelitis, septic arthritis, and infections of bones, joints and organs.	existing atherosclerotic disease; liver, spleen, and biliary tract infections in patients with underlying structural abnormalities; mesenteric lymphadenitis; osteomyelitis in long bones and vertebrae; urinary tract infection; pneumonia; pulmonary abscess; brain abscess; subdural and epidural empyema; meningitis; CNS infections (rarely); and death. Enteric fever: Also known as typhoid fever, this infection is caused by serotypes Typhi and Paratyphi. Enteric fever is characterized by fever (rising within 72 hours after the onset of illness) and headache, brachycardia, faint rose-colored rash on the abdomen and chest, anorexia, abdominal pain, myalgias, malaise, diarrhea (more common in children) or constipation (more common in adults), hepatosplenomegaly, segmental ileus, meningismus, and neuropsychiatric manifestations. Less common symptoms are sore throat, cough, and bloody diarrhoea. Complications include myocarditis, encephalopathy, intravascular coagulation, infections of the biliary tree and intestinal tract, urinary tract infection, and metastatic lesions in bone, joints, liver, and meninges. The most severe complication (occurs in about 3% of patients) is haemorrhage due to perforations of the terminal ileum of proximal colon walls. If untreated, the fever can last for weeks; however, with proper antimicrobial therapy, patients usually recover within 10-14 days. The disease is milder in children and, if treated, has a mortality rate of		

		less then 1%; untreated cases can have a mortality rate greater then 10%.		
Mode(s) of Decontamination	Susceptible to 70% ethanol, clorhexidine, 1% sodium hypochlorite, 2% glutaraldehyde, 0.25% benzalkonium chloride, and formaldehyde. *Staphylococcus aureus* can grow in a pH of 4.2 to 9.3 and in salt concentrations of up to 15%. Enterotoxins are resistant to temperatures that would destroy the bacilli. Sensitive to dry heat treatment of 160-170ºC for at least an hour, but not to moist heat treatment. Survives on carcasses and organs (up to 42 days), floors (less than 7 days), glass (46 hours), sunlight (17 hours), UV (7 hours), meat products (60 days), coins (up to 7 days), skin (30 minutes to 38 days) (citation needed). Depending on colony size, *S. aureus* can survive on fabrics from days to months.	Gram negative bacteria are susceptible to 2-5% phenol, 1% sodium hypochlorite, 4% formaldehyde, 2% glutaraldehyde, 70% ethanol, 70% propanol, 2% peracetic acid, 3-6% hydrogen peroxide, quaternary ammonium compounds and iodophors; however, *Salmonella* spp. is resistant to nitrites. Susceptible to moist heat (121°C for at least 15 minutes) and dry heat (170°C for at least 1 hour). *Salmonella* spp. can also be disinfected with ozone. Serotype Choleraesuis can survive in wet swine feces for at least 3 months and in dry swine feces for at least 13 months. Serotype Dublin can survive in feces spread on concrete, rubber, and polyester for almost six years. Serotype Typhimurium can survive in cattle slurry for 19-60 days, cattle manure for 48 days, soil for 231 days, and water for up to 152 days. Flies have been shown to excrete certain serotypes for 8 days and bed bugs can excrete bacilli for up to 21 days. Certain serotypes have been shown to survive on fingertips for up to 80 minutes, depending on the inoculum size. *Salmonella* serotypes have been found to live up to 63 days on lettuce, 231 days on parsley, 32 weeks in pecans, 10 months on refrigerated cheddar cheese, 9 months in butter, up to 63 days in frozen yogurt, and up to 20 weeks on frozen minced beef and chicken.	Susceptibility has been shown for 1% sodium hypochlorite, 2% glutaraldehyde, 70% ethanol, iodines, phenolics, and formaldehyde. It is also sensitive to silver in water and free chlorine. Exposure to temperatures of 62ºC or greater for more that a few minutes is lethal to *A. hydrophila*. The bacteria can be inactivated by moist heat (121°C for 15 min - 30 min) and dry heat (160-170°C for 1-2 hours). The bacteria can also be inactivated by ultrasonic waves delivered under high pressure (200 KPa) and elevated temperatures (40 °C). Simultaneous sonication at temperature higher than 70°C and 0.5 MPa is also effective. *Aeromonas hydrophila* can survive in large volumes of open water, ground water, soil, as well as polluted lakes and rivers and sewage.	Inactivation can be achieved by using >1.5% concentration of NaCl. A related pathogen *C. jejuni* is susceptible to 10 mg/L iodophor, 1:50 000 quaternary ammonium compound, 0.15% phenolic compound, 70% ethyl alcohol or 0.125% glutaraldehyde all with a contact time of 1 minute or 5mg/L of hypochlorite with a contact time of 5 minutes. Inactivated by heat (70°C for 1 min), hydrostatic pressure (450 MPa at 15°C for 30 s), and pH levels below 5.0 and above 9.0. *Campylobacter* cells can enter a viable but nonculturable state (VBNC) when subject to stress. This is thought to improve their survival in the environment, as it has been observed to survive freezing for several months in frozen poultry, minced meat, and other cold food products. *Campylobacter* can survive for many weeks in water at 4°C, but only a few days above 15°C, and for 2-10 hours when exposed to drying.

Emergency Response	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek immediate medical attention. Monitor for symptoms. In outbreak settings, food poisoning can be diagnosed on clinical grounds with food cultured for *S. aureus*. Toxic shock syndrome can be indicated with a clinical diagnosis and isolation of *S. aureus* strain, TSST-1, or enterotoxins B or C. This can be achieved using ELISA, reverse passive latex agglutination, or PCR. Scalded skin syndrome can be diagnosed clinically, with presence of Nikolsky's sign and identification of *S. aureus* retrieved from the infection site. Bacteremia and deep site infections are confirmed with direct microscopic examination of clinical specimen. Treatment of abscesses usually does not need antibiotic therapy; appropriate drainage is usually sufficient. Proper antibiotic therapy is required for more serious infections. Many strains of Staphylococcus aureus have increasing resistance to multiple antibiotic classes. Methicillin resistant strains are common causes of nosocomial infection. Increasing resistance to vancomycin is being documented in many hospitals.	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek immediate medical attention. Monitor for symptoms. Confirm diagnosis by isolation from stool or blood and by serotyping to identify the serotype. Treatment depends on the clinical symptoms presented by the patient. Gastrotenteritis: Fluid and electrolyte replacement as well as control of the nausea and vomiting are the usual treatments for these symptoms. Antibiotic treatment is not usually used; however, it may be necessary for neonates, children, the elderly, and the immunosuppressed, in which case ciproflaxin, co-trimoxazole, ampicillin, and cephalosporins may be used. Bacteremia: Antibiotic treatment is used to treat bacteremia (e.g. ciproflaxin, co-trimoxazole, ampicillin, or cephalosporins), especially for neonates, children, the elderly, and the immunosuppressed. Enteric fever: Chloramphenicol is the most common antibiotic used for enteric fever although ampicillin, trimethoprim-sulfonamid, cephalosporins, ciproflaxin, and norfloxacin are also being used to treat the disease. Asymptomatic carrier state: Carriers can be treated with ciproflaxin in order to reduce the spread of the infectious agent. Susceptible to chloramphenicol, ciproflaxin, amoxicillin, co-trimoxazole, trimethprim-sulfonamid, cephalosporins and norfloxacin. Some resistance to chloramphenicol has been reported	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek immediate medical attention. Monitor for symptoms. The bacteria can be detected by PCR. *Aeromonas* spp. samples can be isolated and confirmed on ampicillin dextrin agar forming yellow colour colonies. Confirmation by laboratory tests can be performed by oxidase test (positive), triple sugar iron agar (glucose fermenting), or Biolog detection methods. Administration of antibiotics such as ciprofloxacin, co-trimoxazole, Gentamicin, or Amikacin. Intravenous fluid replacement in combination with oral antibiotics is effective in lessening infection symptoms. Sensitivity has been confirmed for fluoroquinolones (ciproflaxin), aminoglycosides (except streptomycin), tetracycline, chloramphenicol, carbapenems, polymyxin, streptomycin, gentamicin, and trimethoprim-sulfametho-xazole. Almost all *Aeromonas* spp. are resistant to penicillin, ampicillin, amoxicillin, ticarcillin, carbenicillin, and cephalothin.	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek immediate medical attention. Monitor for symptoms. Campylobacter infection can be confirmed by culturing and identification of bacteria in stool. Recent Campylobacter infections can be identified using serologic tests. Administer proper antimicrobial therapy. Treatment is primarily supportive as most infections are self-limiting. Antibiotic therapy may be required in more serious cases particularly in young, elderly or immunocompromised patients. Erythromycin is the drug of choice for treating Campylobacter gastroenteritis. Susceptible to macrolides, fluoroquinolones, aminoglycosides, chloramphenicol, nitrofurantoin and tetracycline. Antibiotic resistant strains are emerging particularly to fluoroquinolones and erythromycin. Antibiotic resistance strains are emerging particularly to fluoroquinolones, macrolides, trimethoprim, beta lactam antibiotics, including penicillin and most cephalosporins, as well as to tetracycline, quinolone and kanamycin.

		and, in 1989, 32% of strains were multi-drug resistant.		

Suggested Reference: http://www.phac-aspc.gc.ca/lab-bio/res/psds-ftss/index-eng.php.

Medical Surveillance Sewage Bacteria – (cont)

Clostridium difficile;
Clostridium botulinum;
Yersinia enteracolitica;
Campylobacter jejuni

Pathogen Identification	***Clostridium difficile***	***Clostridium botulinum***	***Yersinia enterocolitica***	***Campylobacter jejuni***
Mode(s) of Transmission	Transmission mainly occurs through the fecal-oral route, via contaminated foods, fomites, or hands. *C. difficile* overgrowth and toxin production can occur in immuno-compromised patients from their natural flora. Nosocomial transmission has also been reported, where infections were transmitted via hospital staff and contaminated equipment. The primary lab infection hazard is accidental ingestion of the pathogen or associated toxins, however, direct contact of open wounds/site of injury with the pathogen, exposure to aerosols, and accidental parenteral inoculation are also potential hazards. Laboratory samples at risk of infection include, but are not limited to: Human fecal and excretion samples, contaminated food products, sewage, soil, and bowel luminal contents or tissue.	*Food borne botulism*: Ingestion of contaminated food containing toxin. Infection is commonly associated with commercially processed foods that had undergone poor processing, storage, and improper preservation. *Wound botulism*: Contamination of wounds with spores of neurotoxin producing Clostridium species and is seen almost exclusively in injection drug users, particularly those who partake in injection of black-tar heroin into skin tissue. *Intestinal (infant) botulism*: Ingestion of spores. Sources include honey and infant milk powder. *Adult infectious botulism*: Ingestion of clostridial spores, rather than toxin, which then colonize the gut to produce their neurotoxin directly in the gut. *Iatrogenic botulism*: Side effect of injection of purified toxin. *Inhalational botulism*: Occurs due to absorption of botulinum toxin by the mucous membrane of the nose. The primary lab infection hazards are accidental ingestion of the pathogen or associated toxins, direct contact of open wounds/	Human-to-human transmission has been reported rarely in schools, daycares and hospitals. Nosocomial infections and blood transfusion related infections by this bacterium have been reported. Fecal-oral transmission from animal-to-human or consumption of contaminated foods (raw pork products, undercooked pork, tofu and unpasteurized milk have been shown to be a source of outbreaks) and untreated water are also common modes of transmission. Also found in untreated sewage. The primary lab infection hazards are accidental ingestion of the pathogen, direct contact of open wounds/ site of injury with the pathogen, exposure to aerosols, and accidental parenteral inoculation.	Oral ingestion of bacteria from contaminated food, contaminated drinking water. Contact with animals and their feces is also a source of infection. Primary lab infection hazards include: accidental ingestion, direct contact of open wounds/site of injury with the pathogen, exposure to aerosols, and accidental parenteral inoculation. *Campylobacter* can survive for many weeks in water at 4°C, but only a few days above 15°C.

		site of injury with the pathogen, exposure to aerosols, and accidental parenteral inoculation.		
Incubation Period	5-10 days with a range of 1 day to weeks following antibiotic treatment for AAD (antibiotic associated diarrhea).	The shorter the incubation period, the more severe the disease and the higher the case fatality rate. *Food-borne botulism*: Usually 12 to 72 hrs after ingestion of toxin, depending on the dose. *Wound botulism*: The median period is 7 days. *Adult infectious botulism:* Unknown. *Intestinal botulism*: Unknown. *Inhalational botulism*: Not well defined, but it is longer than for food borne botulism, and is estimated at 12-80 hours.	The incubation period for this bacterium is between 3-10 days.	1 to 10 days.
Period of Communicability	Indefinite. Low risk of transmission from person-to-person, although nosocomial transmission from contaminated hands, instruments such as endoscopes, and the environment have been reported. Asymptomatic patients can also act as a reservoir for the transmission of this pathogen within the hospital.	Not generally transferred from human to human. Transmission is dependent on accidental ingestion or contact with contaminated material source(s).	Variable. Although rare, the disease can be spread from human-to-human and bacteria can still be present in stool weeks after the clinical symptoms have ceased.	Not generally transferred from human to human. Transmission is dependent on accidental ingestion or contact with contaminated material source(s).
Infectious Dose(s)	Unknown.	Cells/spores are not normally toxic for healthy adults. Botulinum toxin is the most potent toxin known, with an estimated oral or injected toxic dose (serotype A) of 0.001 µg/kg body weight, and an estimated lethal dose by inhalation exposure in humans of approximately 0.07 µg/kg body weight. Type A toxin is more potent than types B and E and causes the longest lasting disease.	The infectious dose is of 10^8 bacteria or more orally.	As low as 500 organisms by ingestion. One volunteer study found that 9000 bacteria were required to infect 50 percent of subjects.
Typical Presenting Symptoms	*C. difficile* is the main cause of nosocomial antibiotic- associated diarrhea. Antibiotics or any other procedures that disrupt the normal intestinal microbiota can lead to the	Rare but serious paralytic disease, caused by a neurotoxin formed during the growth of the spore-forming bacterium *C. botulinum* (or rarely, *C. argentinense, C.*	*Yersinia enterocolitica* infection is characterized by enteritis, enterocolitis (particularly in children), fever (39°C), watery stools,	*Campylobacter jejuni* cause gastroenteritis, with the most common symptom being diarrhea (sometimes bloody) that lasts 2-10 days, as well as mild to severe

	overgrowth and production of toxin by *C. difficile*, which leads to clinical manifestations of infection. The diarrhea may range from a few days of intestinal fluid loss to life-threatening pseudo-membranous colitis. In diarrhea without pseudo-membranous colitis (PMC), feces have a foul odor and are not bloody. Abdominal pain with or without pyrexia may also be present along with diarrhea. PMC is associated with intense inflammation of the colon and formation of pseudo-membranes on the intestinal mucosal surface. Patients with PMC also have more systemic side effects. Rare extracolonic manifestations of *C. difficile* infection include bacteremia, intra-abdominal abscess, osteomyelitis, visceral abscess, empyema, toxic megacolon, colonic perforation, and reactive arthritis. The production of exotoxins A and B facilitates tissue damage, which results in cell necrosis and ulceration, diarrhea and fluid secretion, and colitis. The strain with ribotype 027, also known as North America Pulsotype (NAP) 1, is one of the most pathogenic types of *C. difficile,* and the severity of this strain is based on its unusually high levels of toxin A and B production, its production of a third toxin known as the binary toxin, and its resistance to fluoroquinolone antibiotics. Another new hypervirulent *C. difficile* strain 078 (NAP7/8), has been associated with severe diarrhea and high mortality rates have been reported from The Netherlands and other countries, including Canada.	*butyricum, or C. baratii*). This neurotoxin binds to the neuromuscular junction and blocks excitatory synaptic transmission by inhibiting acetylcholine release, causing (flaccid) paralysis, and sometimes fatal respiratory failure. The fatality rate of botulism is 5 to 10%. *Food-borne botulism*: The classic form of botulism is caused by the ingestion of preformed toxin in contaminated food. Symptoms include double vision, drooping eyelids (ptosis), slurred speech, difficulty swallowing and muscle weakness that is symmetric and descends through the body (first shoulders are affected, then upper arms, lower arms, thighs, calves, etc.). Death is usually due to respiratory failure and may occur as soon as 24 hours after onset of symptoms. *Wound botulism*: Occurs by contamination of a wound with spores from neurotoxin-producing *Clostridium* species in the environment and subsequent germination of these spores and production of toxin in the anaerobic milieu of an abscess. The toxin is released into the bloodstream and symptoms may take up to 2 weeks to appear. *Intestinal (infant) botulism*: Results almost exclusively from spore ingestion and subsequent growth and toxin production in the intestine, affecting infants under 1-year-old. The first clinical sign is usually constipation, but this disease has a wide spectrum of clinical severity, ranging from mild illness with	abdominal pain and acute mesenteric lymphadenitis (which may mimic appendicitis). In some cases, acute terminal ileitis and enteric fever can occur (violent and relatively acute epigastric or abdominal pain caused by penetration of larvae through the stomach or lower small intestine mucosa, especially the ileum. Nausea, vomiting, and fever may occur). 1-3 weeks after the initial clinical symptoms, reactive arthritis (joint pain and swelling) and erythema nodosum (reddish, painful, tender lumps most commonly located in the front of the legs. The tender lumps, or nodules, of erythema nodosum range in size from one to five centimeters. The nodular swelling is caused by inflammation in the fatty layer of skin) may occur which can last about 6 months after infection. In rare instances, complications can include meningitis (symptoms: can include fever, cold hands and feet, vomiting, severe headache, stiff neck, severe muscle pain, pale blotchy skin, rash confusion, sleepiness, and seizures), endophthalmitis (progressive deterioration of vision, light sensitivity, pain and swelling around the eye), conjunctivitis (symptoms can include: redness in white of eye or eyelid, increased amount of	abdominal pain, fever, malaise, nausea and vomiting. Symptoms last for about a week but relapses occur in 5-10% of untreated cases. Although a large number of campylobacter infections are asymptomatic and mild, many complications have been reported in young children and immuno-compromised patients, including bacteremia, hepatitis, cholecystitis, pancreatitis, abortion, myocarditis and meningitis. *C. jejuni* has been associated with post-infection sequelae, most commonly Guillain-Barré syndrome and reactive arthritis. All strains of *C. jejuni* possess a gene coding for cytolethal distending toxin, however not all strains produce it. The role of these toxins in disease is not known. Motility is required for full virulence, and some effectors associated with virulence are secreted through the flagellum.

		gradual onset, to sudden infant death due to respiratory failure. With appropriate intensive care, almost 100% of infants with botulism make a full recovery. Infants with botulism are lethargic, feed poorly, have a weakened cry, exhibit ptosis, and floppy neck, and may progress to generalised flaccidity and respiratory compromise. *Adult infectious botulism*: Rare. Caused by the intestinal colonization of *C. botulinum* / other neurotoxin producing species, followed by *in vivo* toxin production in a manner similar to infant botulism. Patients often have a history of immuno-compromise, abdominal surgery, bowel disease, or recent antibiotic therapy. *Inhalational botulism*: Is not a naturally occurring disease, but has occurred in laboratory workers due to inhalation of aerosolized toxin. Inhalational botulism leads to neurological symptoms similar to those of food-borne botulism, but with a longer incubation period. *Iatrogenic botulism*: Side effects resulting from the therapeutic intramuscular injection of Botox (purified, diluted A neurotoxin). Characterized by clinical weakness and electro-physiological abnormalities.	tears, thick yellow discharge on eyelashes after sleep, green or white discharge from eye, itchy eyes, burning eyes, blurred vision, increased sensitivity to light), myocarditis (chest pain, arrhythmias, shortness of breath, fluid retention, joint swelling, fatigue), pneumonia (symptoms: cough with or without mucus, fever, shaking, chills, and shortness of breath, especially during physical exertion), pulmonary abscess (fever and chills, rapid heart rate, profuse sweating, prolonged period of poor health, loss of appetite, weight loss, chest pain, deep cough that may produce foul smelling of bloody sputum), hepatitis (abdominal pain, dark urine, fever, joint pain, lossof appetite, nausea and vomiting, weakness and fatigue, yellowing of skin and whites of eyes - jaundice), cholangitis (chills, fever, abdominal pain, clay coloured stools, dark urine, nausea and vomiting, jaundice), peritonitis (abdominal pain, bloating, fever, nausea and vomiting, loss of appetite, diarrhea, low urine output, thirst), glomerulo-nephritis (blood or excess protein in urine, high blood pressure, frequent night time urination, bubbly or foamy urine, abdominal pain, frequent nosebleeds), urethritis (burning on urination, frequent urination with only small amounts of urine passed on each occasion,	

			anal or oral infections, urgent need to urinate, bloody or yellowish discharge from the penis, blood in urine, urethral itching), cellulitis (pain, inflammation, fast growing sore or rash, tight, glossy, swollen skin appearance, pus filled center, fever), haemolytic anaemia (low RBC, dark urine, jaundice, heart murmur, increased heart rate, enlarged spleen, enlarged liver), thyroiditis (fatigue, depression, cold intolerance, weight gain, dry skin and hair, muscle cramps, constipation, decreased concentration and sleepiness, leg swelling, puffy eyes; severe symptoms include a slow heart rate, low body temperature, heart failure and coma), pharyngitis (sore throat, sneezing, cough, headache, fatigue, body aches, chills, fever), and septicaemia (symptoms: fever, a rapid heart rate, shaking chills, low blood pressure, gastrointestinal symptoms (abdominal pain, nausea, vomiting, and diarrhea), rapid breathing, and/or confusion). The infection is a greater health risk for immuno-suppressed individuals and if untreated, the mortality rate due to septicaemia can be up to 50%.	
Mode(s) of Decontamination	Spores are generally resistant to disinfection. *Clostridium* spores are resistant to ethyl and propyl alcohols. High level disinfectants such as 2%	The vegetative state is susceptible to disinfectants such as 70% ethanol, 0.1% sodium hypochlorite, and 0.1N NaOH. Spores may be	Susceptible to 2-5% phenol, 1% sodium hypochlorite, 70% ethanol, 4% formaldehyde, 2% glutaraldehyde, 2% peracetic	*C. jejuni* is susceptible to 10 mg/L iodophor, 1:50 000 quaternary ammonium compound, 0.15% phenolic

	glutaraldehyde can kill spores within 20 minutes. 8% formaldehyde and 20 ppm sodium hypochlorite are also effective against bacterial spores. Spores of the genus *Clostridium* are generally heat resistant and can withstand temperature of 116°C for 3 hours, whereas their vegetative cells can be rapidly killed by temperatures of only 55-65°C. Most spores can be inactivated by moist heat at 121°C for 15-30 minutes. *C. difficile* is able to survive in soil, sewage, meat, and vegetables.	resistant to disinfectants. Toxins are inactivated (more than 99.7%) by 20 minutes exposure to 3 mg/L free available chlorine (FAC; similar to the military disinfection procedure), and 84% inactivated by a treatment of 20 minutes at 0.4 mg/L FAC (similar to municipal water treatment procedures). Toxin is destroyed after heating for 5 minutes at greater than 85°C. Toxins are detoxified in air within 12 hours and following exposure to sunlight within 1 to 3 hours. Spores are highly resistant to heat and desiccation; therefore, it is recommended to sterilize with dry heat (2 hours at 160°C) by autoclaving (20 minutes at 121°C, 1 atm pressure) and/or by irradiation. Survives well in soil, water and agricultural products.	acid, 3-6% hydrogen peroxide and 0.16% iodine. Bacteria are sensitive to moist heat (121°C for at least 12 minutes) and dry heat (170°C for 1 hour). The bacterium can survive 448 days in water between -4 and 8°C, and 10 days in water between 20 and 30°C. It can live up to 10 days in soil and cattle manure between -4 and 30°C.	compound, 70% ethyl alcohol or 0.125% glutaraldehyde all with a contact time of 1 minute or 5mg/L of hypochlorite with a contact time of 5 minutes. Inactivated by heat (70°C for 1 min), hydrostatic pressure (450 MPa at 15°C for 30 s) and gamma irradiation. *Campylobacter* cells can enter a viable but nonculturable state (VBNC) when subject to stress. This is thought to improve their survival in the environment, as it has been observed to survive freezing for several months in frozen poultry, minced meat, and other cold food products. *Campylobacter* can survive for many weeks in water at 4°C, but only a few days in water above 15°C.
Emergency Response	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek immediate medical attention. Monitor for symptoms. Detection of toxins in stool specimens is the Gold Standard test for diagnosis of *C. difficile* infection. Detection of toxins is done using cell culture assays, or Enzyme Immunoassay (EIA). Diagnosis can also be done by culturing the bacteria on appropriate media such as egg yolk agar-based media or blood agar media. Two different approaches are commonly used for typing of *C. difficile*strains and include PCR-ribotyping and pulse field gel electrophoresis (PFGE). Treatment should be supportive with rebalancing of fluid levels and electrolytes. Antibiotic treatment	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek immediate medical attention. Monitor for symptoms. Since botulism is a life threatening condition, a rapid diagnosis is essential and may require testing to differentiate botulism from other neurological diseases. *Food borne botulism*: Can be diagnosed by demonstration of toxin in serum, stool, gastric aspirate or implicated food, or by culture of *C. botulinum* from a patient's gastric aspirate or stool in a clinical case. The mouse bioassay is the most reliable method for detection of botulinum. *Wound botulism*: Can be diagnosed by demonstration of toxin in serum, or by positive wound	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek immediate medical attention. Monitor for symptoms. The bacterium can be isolated in stool, tissue samples, blood and pus. PCR and ELISA can also be used to diagnose the disease. Although infections by *Yersinia enterocolitica* are usually self limiting, antibiotic treatment is necessary for severe or complicated cases. The antibiotics commonly used are gentamicin, cotrimoxazole, and ciprofloxacin. Surgery may be required to treat acute terminal ileitis. *Yersinia enterocolitica* is susceptible to chloramphenicol,	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek immediate medical attention. Monitor for symptoms. Campylobacter infection can be confirmed by culturing and identification of bacteria in stool. Recent Campylobacter infections can be identified using serologic tests. Administer proper antimicrobial therapy. Treatment is primarily supportive as most infections are self-limiting. Antibiotic therapy may be required in more serious cases particularly in young, elderly or immunocompromised patients. Erythromycin is the drug of choice for treating Campylobacter gastroenteritis. Generally

	should be discontinued for antibiotic associated diarrhoea (AAD) infection. For patients who do not respond to drug withdrawal or are present with systemic illness, oral metronidazole is used for treatment. Oral vancomycin has been shown to be more effective than metronidazole in treating recurrent infections. Susceptible to metronidazole and oral vancomycin. *C. difficile* also demonstrates sensitivity to penicillins and cephalosporins *in vitro*, but these drugs are not used for treatment because they can be destroyed by β-lactamases/cephalosporinases produced by other intestinal bacteria and are, thus, ineffective for treatment in vivo.	culture. *Adult infectious botulism*: Can be diagnosed by demonstration of *C. botulinum* (or other neurotoxin producing species) and/or toxins in a patient's faeces or in autopsy specimens. *Intestinal (infant) botulism:* Since the toxin is rarely found in the sera of infants, faeces should be examined. An ELISA has been developed for the detection of A and B toxins in children's faecal samples. *Inhalational botulism*: Aerosolized toxin can not usually be identified in serum or faeces, but may be detected by ELISA from nasal swabs. *Iatrogenic botulism*: Should be suspected if patient has recently received Botox. An immuno-PCR assay capable of detecting neurotoxin type A in the femtogram range has been developed. Susceptible to penicillin, metronidazole, clindamycin, cephalothin, cefoxitin, cefotaxime, chloramphenicol, tetracycline, erythromycin, rifampin, and vancomycin (with some strain variation). Usually resistant to the aminoglycosides, and may be resistant to tetracyclines and cephalosporins (with some strain variation). Also resistant to nalidixic acid and sulpha-methoxazole-trimethoprim (SMX-TMP).	fluoroquinolones, gentamicin, tetracycline, and trimethoprim-sulfa-methoxazole. It is generally resistant to penicillin and its derivatives and to narrow spectrum cephalosporins.	susceptible to macrolides, fluoroquinolones, aminoglycosides, chloramphenicol, nitrofurantoin, gentamicin, and tetracycline. Antibiotic resistance strains are emerging particularly to fluoroquinolones, macrolides, trimethoprim, beta lactam antibiotics, including penicillin and most cephalosporins, as well as to tetracycline, quinolone and kanamycin.

Suggested Reference: http://www.phac-aspc.gc.ca/lab-bio/res/psds-ftss/index-eng.php.

Medical Surveillance Sewage Bacteria – (cont)

Clostridium perfringens;

Klebsiella spp.;

Actineobacter spp.

Pathogen Identification	***Clostridium perfringens***	***Klebsiella* spp.**	***Acineobacter* spp.**
Mode(s) of Transmission	*Food Poisoning*: Food-borne illness acquired by ingestion of large number of *C. perfringens* vegetative cells present in the food. Food sources are usually cooked meat, vegetables, fish or poultry dishes which have been stored at ambient temperatures for a long time after cooking. *Enteritis Necroticans*: Ingestion of contaminated pork meat. *Gas Gangrene/ Anaerobic Cellulitis*: Infection can occur through contamination of wounds (fractures, bullet wounds) with dirt or any foreign material contaminated with *C. perfringens*. Primary laboratory hazards include: accidental ingestion of the enterotoxin, direct contact of open wounds/site of injury with the pathogen, accidental parenteral inoculation of the toxin. Sources can include: human feces, suspect food in a food borne illness, blood, bowel luminal contents or tissue from the involved bowel in case of enteritis necroticans, wound exudates, and sewage.	*Klebsiella* spp. can be transmitted through skin contact with environmentally contaminated surfaces and/or objects; examples include Loofah sponges, medical equipment, sewage, and blood products. Fecal transmission has also been suggested for some cases of bacteremia caused by *Klebsiella* spp. *K. rhinoscleromatis* can be transmitted from person-to-person via airborne secretions; however, prolonged contact with infected individuals is required for infection. *K. granulomatis* are sexually transmitted. They may also be vertically transmitted (from mother to child) or by accidental inoculation. Transmission rates between partners are low (<50%) compared to other sexually transmitted diseases. Laboratory hazards include direct contact of mucosal membranes with contaminated surfaces and/or object, and inhalation of infectious airborne secretions, accidental parenteral inoculation and/or ingestion.	*Acineobacter* spp. can be transmitted through skin contact with environmentally contaminated surfaces and/or objects. *Acinetobacter* spp. are ubiquitous inhabitants of soil, water, and sewage environments. Infection is most commonly associated with contact with wounds and burns or inhalation by susceptible individuals. Primary lab infection hazards include: accidental ingestion, direct contact of open wounds/site of injury with the pathogen, exposure to aerosols, and accidental parenteral inoculation. *Acinetobacter* has been isolated from 97% of natural surface water samples in numbers of up to 100/ml. The organisms have been found to represent 1.0–5.5% of the HPC flora in drinking-water samples and have been isolated from 5–92% of distribution water samples. Nosocomial infections with this bacteria are not uncommon.
Incubation Period	*Food Poisoning*: 8-24 hours. *Gas Gangrene*: 1-4 days after the injury, but may also start within 10 hours.	Not clearly understood. According to some sources, the incubation period for *K. granulomatis* is usually 1 to 6 weeks.	Unknown.
Period of Communicability	Not transferred from human to human. Transmission is dependent on contact with contaminated material source(s) .	Members of *Klebsiella* spp. can be transmitted from person-to-person; however, the communicability period is unknown. Approximately one-third of people carry *Klebsiellae* in their stools; detection rates according to different studies vary from 5% to 36%. Detection rates in nasopharynx vary from 1% to 6%. Hospital personnel	Not transferred from human to human. Transmission is dependent on contact with contaminated material source(s).

		have been shown to frequently carry *Klebsiellae* on their hands.	
Infectious Dose(s)	Unknown.	Unknown. According to one source, 10^8 *Klebsiella* organisms per gram of feces are required to produce damage.	Unknown.
Typical Presenting Symptoms	Clostridial Food Poisoning: Food poisoning can be caused by *C. perfringens* entero-toxin (CPE) produced by *C. perfringens* spores in the small intestine, which can germinate in foods such as meat and poultry. In the United States consumption of large amounts of *C. perfringens* is considered an important cause of watery diarrhea. Main symptoms of the disease are nausea, abdominal pain, and diarrhea. The disease is usually mild and self-limiting in healthy individuals, with symptoms resolving within 24 hours. Clostridial Myonecrosis (Gas Gangrene): *C. perfringens* is the most common cause of clostridial myonecrosis. The disease involves breakdown of muscle tissue due to the action of potent exotoxins, alpha and theta, produced by the bacteria. It is manifested by severe pain, edema, tenderness and pallor, followed by discoloration and hemorrhagic bullae, and production of gas at the site of wound. Systemic manifestations of the disease include shock, renal failure, hypotension, bacteremia with intravascular hemolysis leading to coma and death. Clostridial Cellulitis: *C. perfringens* is the most common cause of clostridial cellulitis, which is often associated with local trauma or recent surgery. Infection is less systemic than in clostridial myonecrosis, with localized infection and associated skin and soft tissue necrosis, but sparing of the fascia and deep muscles. Enteritis Necrotican (pigbel): Enteritis necroticans is a life threatening infection involving ischemic necrosis of the jejunum. The often fatal disease is caused by *C. perfringens* type C, and is marked by hemorrhagic, inflammatory, or ischemic necrosis of the jejunum. Most cases occur sporadically, during outbreatks, or in underdeveloped countries. CNS manifestations of C. perfringens infection: CNS diseases due to *C. perfringens*infection are rare. The main	*Klebsiella* spp. have been identified as important common pathogens for nosocomial pneumonia (7 to 14% of all cases), septicaemia (4 to 15%), urinary tract infection (UTIs; 6 to 17%), wound infections (2 to 4%), intensive care unit (ICU) infections (4 to 17%), and neonatal septicaemias (3 to 20%). *Klebsiella* spp. can also cause bacteremias and hepatic infections, and have been isolated from a number of unusual infection, including endocarditis, primary gas-containing mediastinal abscess, peritonitis, acute cholecystitis, crepitant myonecrosis, pyomyositis, necrotizing fasciitis, psoas muscle abscess, fascial space infections of the head and neck, and septic arthritis. Respiratory disease: Infection of the upper lobe is more common. Symptoms include: fevers, chills, and leukocytosis with red currant jelly-like sputum. Rare complications include lung infection involving necrosis and sloughing of the entire lobe. Chronic nose inflammation has also been observed. Central nervous system (CNS) infections: meningitis and brain abscesses. Clinical symptoms include: headaches, fever, altered conciousness, seizures, and septic shock. Can also cause donovanosis or granuloma, a chronic ulcerative disease that primarily affects the genitalia. Symptoms include development of small papule or ulcer at the site of inoculation that later develop into large red ulcers (lesions) that extend along the moist folds of the genitalia.	Opportunistic pathogens that may cause urinary tract infections (symptoms: upper back and side (flank) pain, fever, shaking and chills, nausea, vomiting, pelvic pressure, lower abdomen discomfort, frequent, painful urination, and blood in urine), pneumonia (symptoms: cough with or without mucus, fever, shaking chills, and shortness of breath, especially during physical exertion), bacteraemia which can lead to septicemia (symptoms: fever, a rapid heart rate, shaking chills, low blood pressure, gastrointestinal symptoms (abdominal pain, nausea, vomiting, and diarrhea), rapid breathing, and/or confusion), secondary meningitis (symptoms: headache, stiff neck, and fever. In infants, these symptoms may be difficult to detect. Other symptoms include vomiting, nausea, photophobia (sensitivity to light), confusion, sleepiness, and seizures) and wound infections (symptoms: increased pain, swelling, redness, and drainage).

	manifestations of *C. perfringens* infection in CNS are meningitis and encephalitis. Clinical symptoms of the two diseases are similar and include tiredness, fever, headache, vomiting, hypersensitivity to light or noise, neck stiffness, or impaired consciousness and coma.		
Mode(s) of Decontamination	Spores are resistant to most disinfectants and, when susceptible, they require longer contact time. *Clostridium* spores are resistant to ethyl and propyl alcohols, chlorine dioxide. Spores of clostridium species can be killed by high level disinfectants such as 2% aqueous glutaraldehyde within 3 hours, and 8% formaldehyde. Spores are highly resistant to both heat, and gamma-irradiation. Enterotoxin is heat labile and can be inactivated by heat treatment at 60ºC for 5 minutes. Vegetative cells can be rapidly killed by dry heat at 160-170°C for 1-2 hours or moist heat at 121°C for 15 min- 30 min. Spores can survive in soil, crevices, food, decaying vegetation, marine sediments, sewage, internal cavities and in the anaerobic conditions inside the meat rolls, animal carcasses, feces, dehydrated and cooked food.	Gram-negative bacteria are generally susceptible to a number of disinfectants, including phenolic compounds, hypochlorites (1% sodium hypochlorite), alcohols (70% ethanol), formaldehyde (18.5 g/L; 5% formalin in water), glutaraldehyde, and iodines (0.075 g/L). Reduction in the growth and metabolic activity of *K. pneumoniae* at temperatures >35 °C has been reported. Significant growth reduction has been demonstrated at 60 °C; however, the bacteria still show some metabolic activity (i.e. not completely inactivated). Bacteria are also sensitive to moist heat and dry heat. *Klebsiella* spp. grow rapidly on surfaces of potatoes and lettuce with counts exceeding 10^3 organisms per g of surface. They have been found in Loofah sponges made from vegetable gourds. They also survive well within wood and sawdust. They do not grow well on human skin and generally exists in infected individuals and/or surfaces, and the environment; surface water, sewage, soil, and on plants, where they can survive for extended periods of time.	10% bleach, 70% ethanol, and 2% glutaraldehyde solutions are all effective surface decontaminants. Acineobacter spp. can also be inactivated by moist heat (15 minutes at 121°C) and dry heat (1 hour at 160-170°C). Outside of the host, Acineobacter spp. can survive for long periods of time outside of susceptible hosts on dry surfaceas, in water, soil, and in sewage.
Emergency Response	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek immediate medical attention. Monitor for symptoms. Diagnosis is based mainly on clinical symptoms. Food borne illness: Diagnosis consists of: 1) culture and characterization of the bacteria including Gram-stain; 2) PCR amplification of the enterotoxin (cpe) gene, as toxin production is associated with its presence; and 4) detection of CPE in feces through toxin assay, cell culture assay, ELISA or RPLA (reverse-phase latex agglutination). Enteritis Necroticans: Diagnosis consists of direct Gram stain of specimens from symptomatic patients, and culture and characterization of the bacteria. Typing can be done using PCR assay for the cpa and	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek immediate medical attention. Monitor for symptoms. Other tests include isolating strains of the bacteria or typing different isolates. This is often necessary for investigation of endemic and epidemic nosocomial infections and also for epidemiological investigations from the environment. *Klebsiellae* can be isolated by growth in media. Although there are specific chromogenic media available for isolating these bacteria from specific samples,*Klebsiellae* grow well on blood and non-differential media. Biotyping and serotyping are two common forms of typing methods used for typing *Klebsiella* spp. Serotyping is the most widely	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek immediate medical attention. Monitor for symptoms. Strain identification by molecular and biochemical means is necessary to determine proper antibiotic treatment. *Acineobacter spp.* commonly presents resistance to multiple antimicrobial agents, occasionally including carbapenems and polymyxins, and hence, it is considered the paradigm of multidrug-resistant (MDR) or pandrug-resistant (PDR) bacterium. MDR *A. baumannii* is a rapidly emerging pathogen, especially in the intensive care setting, causing infections including bacteremia, pneumonia/ventilator-associated pneumonia

	cpb genes, which code for a- and b-toxins, respectively. Gas gangrene/Anaerobic cellulitis: Diagnosis consists of direct Gram stain smear of the wound for the presence of short chains of large, fat gram positive rods with blunt ends from symptomatic patients. Food poisoning: Self-limiting disease. Therapy in mainly supportive; bowel resection may be required for very severe cases. Gas Gangrene: Treatment mainly involves excision of all devitalized tissue in conjunction with antibiotic therapy with a combination of penicillin and clindamycin or tetracycline, which appear most effective based on animal models. *In vitro,* chloramphenicol, metronidazole, and several cephalosporins are active against *C. perfringens*. There have been a few reports of successful results using hyperbaric oxygenation in adjunctive therapy. Anaerobic Cellulitis: Surgical debridement of the tissue and antibiotic therapy with penicillin or clindamycin. In case of drug resistance to clindamycin, second line antibacterial agents such as vancomycin can be used. Vaccination against CPB toxin of *C. perfringens* type C which causes Enteritis Necroticans was reported to decrease the incidence of the disease in New Guinea**.**	used technique for typing these bacteria which involves detection of capsular antigens by means of antisera. Serotyping tests include: quelling reaction, immunofluorescence, double-diffusion gel precipitation, counter-current immunoelectrophoreses, *Staphylococcus* coagulation, and latex coagulation methods. Biotyping is not preferred due to the large number of reactions and the amount of time required to complete these tests. Molecular typing methods are also being developed, although they are not commonly used. These include: plasmid analysis, ribotyping, PFGE, and random amplified polymorphic DNA analysis, all of which have been successfully used to track strains epidemio-logically. Administer appropriate antibiotic therapy where necessary.	(VAP), meningitis, urinary tract infection, central venous catheter-related infection, and wound infection.

Medical Surveillance Sewage Viruses

Norovirus;

Adenovirus serotypes 1-5, and 7;

Adenovirus serotypes 40 and 41;

Hepatitis A Virus

Pathogen Identification	**Norovirus (Norwalk virus)**	**Adenovirus serotypes 1, 2, 3, 4, 5, and 7 (excluding serotypes 40 and 41)**	**Adenovirus (serotypes 40 and 41)**	**Hepatitis A Virus (HAV)**
Mode(s) of Transmission	Norovirus transmission is usually person to person through the fecal-oral route. It can also be transmitted through the environment, contaminated surfaces, food, water, fomites, and aerosols. Laboratory hazards include Ingestion, exposure of mucous membranes to infective aerosols, accidentally ingestion, and accidental parenteral inoculation. Noroviruses have a protein capsid protecting it from the environment. They can survive in seawater, groundwater, fresh water, soil, and inanimate surfaces for an unknown period of time. Norwalk virus cannot be cultivated under laboratory conditions as of yet. Feline calicivirus, another member of this family, with similar structure, can survive on glass surfaces for 21-28 days at room temperature and for longer periods at 4°C. At 37°C, feline calicivirus survives over 24 hours.	Respiratory and fecal-oral routes. Infection can also spread through contaminated fomites, fingers, ophthalmic solutions, and airborne particulates. Laboratory hazards include Ingestion, exposure of mucous membranes to infective aerosols, accidental ingestion, and accidental parenteral inoculation. Adenoviruses are very stable in the environment and persist for 7 days to 3 months on dry inanimate surfaces. They can also survive for weeks in tap water, sewage effluent and sea water. Adenovirus type 2 can survive on common environmental surfaces for up to 8 weeks at room temperature.	The virus is transmitted via the fecal-oral route. Laboratory hazards include Ingestion, exposure of mucous membranes to infective aerosols, accidental ingestion, and accidental parenteral inoculation. Most serotypes are stable at 36 °C for a week, for several weeks at room temperature, and for several months at 4 °C. Adenoviruses are very stable in the environment and persist for 7 days to 3 months on dry inanimate surfaces. They can also survive for many days in tap water, sewage effluent, and sea water.	HAV transmission usually occurs via the faecal-oral route. The most common reported source of HAV infection is household or other close contact with an infected person. Other potential sources of infection include men having sex with men, travel to countries where HAV is endemic, and from contaminated drugs and needles following illicit drug use. Consumption of contaminated food and water are infrequent modes of transmission, and transmission by transfusion of blood or blood products is even rarer. Laboratory hazards include Ingestion, exposure of mucous membranes to infective aerosols, accidental ingestion, and accidental parenteral inoculation. HAV is exceptionally stable at ambient temperature and at low pH, thus allowing it to survive in the environment and to be transmitted via contaminated foods and drinking water.
Incubation Period	Onset of symptoms usually takes 15-48 hours from the time of infection.	Approximately 2 to 14 days.	3 to 10 days.	Average of 28 to 30 days (range of 15 to 50 days).

Period of Communicability	Individuals can spread virus particles without showing symptoms of disease. Those who have recovered from symptoms and those who are asymptomatic can shed infectious virus particles up to three weeks after exposure.	Children shed non enteric adenovirus in throat and stool samples for 3 to 6 weeks following lower respiratory infection or generalized illness. Chance of transmission is high in crowded and closed settings such as day cares, boarding schools and long-term care facilities. Transmission between family members is common. In rare cases, virus shedding may last for 18 months or longer.	Low communicability between close contacts in the same household. Virus shedding takes place during the acute stage of the disease, since enteric adenoviruses are rarely recovered from stool samples more than a few weeks after recovery from gastroenteritis. Asymptomatic individuals (mainly children) shed adenoviruses in their stool.	Maximum infectivity occurs in the latter half of incubation and continues a few days after the onset of jaundice. Chronic shedding of HAV in faeces does not occur.
Infectious Dose(s)	Less than 10 virions.	Inhalation of as few as 5 adenovirus particles can cause disease in susceptible individuals. The National Institutes of Health lists the infectious dose for adenovirus serotype 7 as >150 viral units, administered as nasal drops.	Unknown.	Unknown.
Typical Presenting Symptoms	Norovirus infection causes acute gastroenteritis, characterized by rapid onset of nausea, vomiting, diarrhea, abdominal cramps, abdominal pain, mucus in stool, malaise, headache, and fever. The infection is usually resolved within 12 to 60 hours, although it can last up to 120 days in elderly, young children or immunocompromised individuals, and these groups are at greatest risk for mortality and increased morbidity. Up to 30% of Norovirus infections are asymptomatic, however, these individuals are able to transmit the virus.	Adenovirus cause generally mild respiratory tract infections which are self-limiting and generally asymptomatic despite virologic and serologic proof of infection, and only around 45% of infections are manifested by disease. It is a major agent of acute respiratory disease, mainly caused by serotypes 4 and 7, and is characterized by fever, rhinitis, pharyngitis, cough, and conjunctivitis. Other common illnesses can be observed in the respiratory tract, gastrointestinal tract, and eyes (acute follicular conjunctivitis). Common diseases caused by various adenovirus serotypes are: Childhood febrile illness and pharyngo-conjunctival fever (1-3, 5, 7), pneumonia (1-3, 5, 7, 14), pertussis-like illness (1-3, 5, 19, 21), kerato-conjunctivitis (3, 8, 9, 19, 37), acute hemorrhagic cystitis (11), upper respiratory	Adenovirus serotypes 40 and 41 cause acute gastroenteritis primarily in children. Symptoms may include fever, diarrhea, vomiting, and abdominal pain, and last for approximately 10 days. Respiratory symptoms can occur in some individuals. The disease is usually self-limiting in immunocompetent individuals; however rare fatalities can occur in immuno-compromised individuals. Asymptomatic infections are common, particularly in children.	HAV only causes acute hepatitis and is not associated with chronic liver disease. Most individuals infected with HAV develop non-specific constitutional signs and symptoms followed by gastrointestinal symptoms. Symptoms include fever, malaise, anorexia, nausea, abdominal discomfort, dark urine, and jaundice. The disease course typically lasts less than 2 months. In rare cases, HAV can cause severe cases of fulminant hepatitis with fatal outcomes in otherwise healthy adults.

		illness and hepatitis (1-3, 5, 7), and lower respiratory illness (3, 4, 7, 21).		
Mode(s) of Decontamination	Formulation R-82 for 10 minutes, 5-10% sodium hypochlorite for 10-20 minutes, peracetic acid or glutaraldehyde for 5 minutes. Noroviruses are inactivated by temperatures of 71.3°C for 1 minute. It can survive at pH 2.7 for at least 3 hours. UV radiation may have intermediate effect.	Adenoviruses are resistant to lipid disinfectants, but are inactivated by formaldehyde and chlorine. They can be inactivated by contact with 1:5 dilution of bleach for 1 minute or 2 minutes contact with alcohol-based hand gels. Adenovirus can be inactivated by heat: heating to 56 °C for 30 min, 60 °C for 2 min, and autoclaving will destroy infectivity.	Adenoviruses are resistant to lipid disinfectants, but are inactivated by formaldehyde and chlorine. Adenoviruses can be inactivated by contact with 1:5 dilution of bleach for 1-2 minutes, and by contact with alcohol-based hand gels. Adenovirus can be inactivated by heat: heating to 56 °C for 30 min, 60 °C for 2 min, and autoclaving will destroy infectivity. Adenovirus serotype 40 is also sensitive to UV radiation.	HAV is inactivated by 1 % sodium hypochlorite, formulations of quaternary ammonium compounds and HCl, and 2 % glutaraldehyde. Inactivation of HAV can be achieved by heating to greater than 85°C for 1 minute.
Emergency Response	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms and confirm clinically. Viral RNA can be detected in stool samples using reverse transcription PCR. ELISA is also available for identifying noroviruses. No specific therapy other than rest, oral rehydration and intravenous electrolyte replacement. No immunization currently available.	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms and confirm clinically. Infected cells can be observed by microscopy, and adenoviruses can be detected using immuno-fluorescence, enzyme-linked immunoassay, or PCR for antigen detection. No formally approved effective antiviral agents exist for treatment of adenoviral infections. Illness is generally self-limiting and treatment is supportive. It has been suggested that immuno-compromised patients may require drug treatment with cidofovir or other antiviral drugs. A vaccine for adenovirus strains 4 and 7 was developed but is no longer in production (economic reasons).	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms of gastrointestinal and/or respiratory illness. Enteric adenovirus infection can be detected by electron microscopy, agglutination tests, enzyme-linked immuno-sorbent assay, or by PCR. Illness is generally self-limiting. Treatment is primarily oral rehydration or, in serious cases, intravenous rehydration. Reports indicate that cidofovir may be effective against adenoviruses; however, no controlled trials have been performed so far, and the drug is not currently licensed for use.	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms of disease. Viral infection can be detected via RT- PCR, radio-immunoassay, ELISA, immunoblotting, and dot-blot gold filtration. No specific treatment for HAV infection has been shown to be effective. Bed rest and balanced nutrition are recommended, as is the avoidance of alcohol or other hepatotoxins. Several inactivated HAV vaccines have been shown to be effective (highly immunogenic and safe). Individuals who are known to have been exposed to HAV should be administered HAV immune globulin (0.02ml/kg body weight) within two weeks of the initial exposure. Inactivated HAV vaccines are highly effective in

				pre-exposure prophylaxis for laboratory workers and travellers to HAV endemic areas. There is also some evidence for effective post-exposure prophylaxis with HAV vaccine.
Suggested Reference: http://www.phac-aspc.gc.ca/lab-bio/res/psds-ftss/index-eng.php.				

Medical Surveillance Sewage Viruses – (cont)

Hepatitis E Virus;
Coxsackievirus;
Human Rotavirus

Pathogen Identification	**Hepatitis E Virus (HEV)**	**Coxsackievirus**	**Human Rotavirus**
Mode(s) of Transmission	Four modes of transmission of HEV infection have been reported: faecal-oral transmission, food-borne transmission, blood-borne transmission, and vertical transmission. The most common mode of transmission of HEV, also responsible for the majority of the HEV infection outbreaks, is through the faecal-oral route, usually by ingestion of contaminated water. Potential exists for food-borne transmission and some cases have been observed where consumption of raw or uncooked meat from wild boar and deer has led to HEV infection. Blood-borne transmission is rare but has been documented in some cases involving blood transfusions. Some cases of vertical (perinatal) transmission from mother-to- child have been documented, particularly in India, but this is considered to be of minor importance as a mode of transmission for HEV and more investigation is required. Person-to- person transmission and secondary household cases are uncommon, particularly in epidemic (poor hygienic) conditions. In non-endemic regions, where autochthonous cases have been observed, zoonotic transmission has been considered as the likely mode of transmission, but more investigation is required. Laboratory hazards include exposure of mucous membranes to infective aerosols, accidental ingestion, and accidental parenteral inoculation. Since HEV survives the conditions within the intestinal tract, it is considered relatively stable in acidic and mild alkaline conditions. Since HEV mainly spreads	Infection occurs through contact with infective secretions or excretions, and subsequent autoinoculation of mouth, nose, or eyes. It is possible that ingestion of contaminated water may contribute to infection. Intranasal and aerosol transmission are possible for some variants through contaminated respiratory secretions. Laboratory hazards include exposure of mucous membranes to infective aerosols, accidental ingestion, and accidental parenteral inoculation.	The most common mode of transmission for HRV is through faecal-oral spread, either from person-to-person or contact with contaminated environmental surfaces. The possibility of spread through faecally contaminated food and water also exists. Transmission through respiratory droplets has also been suggested; however, more investigation is required. Laboratory hazards include exposure of mucous membranes to infective aerosols, accidental ingestion, and accidental parenteral inoculation.

	through the faecal-oral route, it must be relatively stable under environmental conditions, possibly similar to HAV (i.e. in water and sewage for long periods).		
Incubation Period	Incubation period for HEV infection in humans ranges from 15-60 days with a mean 40 days.	The incubation period for HRV infection is about 1-3 days.	1-3 days
Period of Communicability	Unknown. Person-to-person transmission has been documented but appears to be uncommon. HEV has been detected in the stools of infected patients after the onset of illness (jaundice) for up to 14 days. Maximal HEV shedding occurs during the incubation period and during early acute stage of the disease.	Human-to-human transfer can occur readily through oral, ocular, respiratory, or faecal-oral routes. Incubation period varies with clinical course.	Person-to-person transmission appears to be fairly common through the faecal-oral route. Rotavirus shedding rate is the highest during the diarrhoeal stage of the disease, which occurs during the first 2-5 days of illness.
Infectious Dose(s)	Unknown.	Unknown; however, 15-50 TCID has been shown to be infective in adult volunteers.	Theoretically, a single infectious virus particle is capable of causing infection, although more than one infectious virus particle is generally required. The median infectious dose for rotavirus is 5.597.
Typical Presenting Symptoms	The disease caused by HEV is generally self-limiting with symptoms typical of acute viral hepatitis including, jaundice, malaise, anorexia, abdominal pain, nausea, fever, diarrhea, discoloured stool and/or urine, and hepatomegaly. Anicteric hepatitis and cholestasis are also observed in some cases. Mortality rate due to infection by hepatitis E have been reported to be as high as 1 %; however, the mortality rate may reach up to 20 % in pregnant women with each passing trimester, making HEV infection the most severe hepatitis in pregnancy of all recognized hepatitis viruses. Analysis of serum specimens collected from volunteer blood donors shows that the prevalence of HEV varies from region-to-region but is higher in endemic countries/regions as compared to developed countries. Hepatitis caused by HEV is clinically indistinguishable from hepatitis A disease.	Majority of infections are asymptomatic and self-limiting; however, infections may lead to a variety of rare conditions, some of which are life threatening. Clinical course can vary within and between strains of Coxsackievirus. Coxsackievirus group A associated conditions: hand-foot-and-mouth disease, herpangina, acute lymphatic or nodular pharyngitis, aseptic meningitis, paralysis, exanthema, hand-foot-and-mouth disease (A10, A16), pneumonitis of infants, “common cold”, hepatitis, infantile diarrhoea, acute hemorrhagic conjunctivitis (A24). Coxsackievirus group B associated conditions: diabetes, pleurodynia, aseptic meningitis, paralysis, severe systemic infection in infants, meningoencephalitis, myocarditis, pericarditis, upper respiratory illness and pneumonia, rash, hepatitis, and pancreatitis,. Undifferentiated febrile illness and viral parkinsonism may also be associated with Coxsackievirus group B infection; however, these are more controversial. Most common conditions requiring hospitalization are: Hand-foot-and-mouth disease: characterized by fever and vesicles on the mouth and extremities, sore throat, fever, and anorexia. Usually self limiting and requires only symptomatic treatment.	HRV predominantly attacks enterocytes, which are mature villous epithelial cells in the small intestine. The disease caused by HRV is self-limiting in general, lasting for about 4-7 days, with symptoms similar to those caused by other gastrointestinal agents, although the symptoms of HRV infections are usually more severe. These include fever, vomiting, and non-bloody diarrhoea (often watery and explosive). This usually leads to mild to severe dehydration (usually isotonic in nature); electrolyte imbalance; and in prolonged cases, to secondary disaccharidase deficiency. Subsequent gastroenteritis infections tend to be less severe compared to previous infections. A temporal association of rotavirus infection with a variety of disease conditions has been described, including upper and lower respiratory infection, intussusception, and others. An etiologic association of rotavirus infection with necrotizing enterocolitis, hemorrhagic gastroenteritis, and pneumatosis intestinalis in infancy has been suggested. Detection of rotavirus RNA in cerebrospinal fluid of patients with gastroenteritis suggests that neurological disorders such as

		Aseptic meningitis/meningoencephalitis: Most common clinical syndrome associated with enteroviruses, resulting in medical attention. The virus causes nonbacterial inflammation of the meninges associated with fever, headache, photophobia, and with no apparent parenchymal involvement. The infection can progress to meningoencephalitis with infection of the parenchyma, characterized by disturbed state of consciousness, focal neurologic signs, and seizures. While severe meningeal disease is usually self limiting, deaths have been reported. Myocarditis and dilated cardiomyopathy: Myocarditis is an inflammation of the myocardium associated with damage that is unrelated to ischemic injury, if infection /inflammation persist the syndrome may progress to dilated cardiomyopathy (in which the heart is enlarged), potentially leading to heart failure [very rare].	convulsions may be associated with HRV infection, but this has not been confirmed.
Mode(s) of Decontamination	HEV is susceptible to iodinated disinfectants (0.075g/L or 1 % iodine). It may also be sensitive to hypochlorites (1 % sodium hypochlorite), formaldehyde (18.5 g/L; 5 % formalin in water), and glutaraldehyde. HEV is more heat labile than *Hepatitis A virus* (HAV) and most strains can be inactivated at temperature ≥60°C for 15 minutes or more[12]. The heat sensitivity of HEV, however, depends on the heating conditions. Hepatitis E in PBS is inactivated quickly at 60°C, but in an albumin solution is inactivated more slowly. When HEV is added to freeze-dried fibrinogen containing stabilizers and subjected to dry heat, it is inactivated to below detection limit within 24 hours at 80°C, but is inactivated more slowly at 60°C. It is also susceptible to low storage temperatures (between -70°C and +8°C).	Sensitive to formaldehyde, gluteraldehyde, strong acids, sodium hypochlorite (bleach), and free residual chlorine. Sensitivity is dependent on sufficient concentration, pH, and contact time and is reduced in presence of extraneous organic materials. Infectious viruses are usually resistant to many common laboratory disinfectants, including 70% ethanol, isopropanol, dilute Lysol, and quaternary ammonium compounds. Insensitive to lipid solvents, including ether and chloroform. Stable in many detergents at ambient temperatures. Sensitive to UV mediated inactivation. Drying conditions reduce viral titres, the degree of which is dependent on the porosity of the surface and presence of extraneous organic matter. Most are readily inactivated at 42 °C, but stability and heat resistance is increased in the presence of sulphydral reducing agents and magnesium cations. No antiviral medications are currently approved. Although Pleconaril® has been used in clinical trials to treat severe Coxsackievirus infections, these have shown no important effects on morbidity or mortality.	HRV, either in suspension or on inanimate surfaces, is susceptible to glutaraldehyde (2%); chlorinated disinfectants (>20,000 p.p.m. chlorine); iodinated disinfectants (>10,000 p.p.m. iodine); combinations of quaternary ammonium compounds with alcohols (>40%), some acids (HCl), some bases (sodium metasilicate); and combinations of phenolic compounds with strong anionic surfactants. Longer exposure times are required for disinfecting contaminated surfaces as compared to contaminated suspensions/solutions. HRV has also been shown to be very susceptible to Lysol brand disinfectants (79% ethyl alcohol, 0.1% *o*-phenylphenol). Other disinfectants include formalin (2%) and sodium hypochlorite (2%). HRV is susceptible to strong acidic pH (<3.0). It is also susceptible to heating above 50 °C (for 30 minutes), but is stabilized in 2M magnesium sulphate.
Emergency Response	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms of disease. Confirm using serological or nucleic acid tests, and by exclusion of hepatitis A and B viruses. Serological tests involve enzyme-linked immunosorbent assays (i.e. ELISA) for the detection of antibodies to	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms; confirm by serology, virus isolation, or PCR from lesions or nasopharyngeal and fecal specimens. The use of viral culture is declining as not all serotypes will grow well in culture. Molecular typing has largely replaced serotyping. No specific treatment is available. No general vaccine	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms. Electron microscopy is still the gold standard diagnostic technique for HRV; however, it is slightly less sensitive and more expensive than some new diagnostic techniques. These include enzyme-linked immunosorbent assays (ELISAs)

	HEV (IgM, IgA, and IgG), and nucleic acid tests involve reverse transcription-polymerase chain reaction (RT-PCR) assays for the detection of HEV RNA in serum, bile, and/or faecal samples. Rest is recommended for infected individuals, since no specific treatment currently available.	available; however, one is under development for Coxsackievirus induced heart disease.	and latex agglutination assays used for the detection of HRV (group A) antigen in stool samples. Supportive therapy is needed to prevent dehydration by replacement of fluid and electrolyte losses. This can be done using the World Health Organization (WHO) formulation or other commercial formulations, or through intravenous fluids in cases of severe diarrhoea, intractable vomiting, acidosis, and/or shock accompany the illness. Resumption of normal diet should be promoted after rehydration. In the past, the rhesus-human rotavirus reassortment-tetravalent vaccine (Rotashield) had been recommended for use by the US Advisory Committee on Immunization Practices (ACIP), but its use was suspended in 1999. Health Canada has approved RotaTeq and Rotarix vaccines in Canada; however, they are not part of the routine immunization programs that are currently available. RotaTeq is a live, oral, human-bovine, reassortment rotavirus vaccine developed from a strain of bovine rotavirus, approved by the US Food and Drug Administration (FDA). Rotarix is a live, oral human rotavirus vaccine developed from the most common strain of human rotavirus, also approved by FDA.

Medical Surveillance Sewage – Other

Giardia lamblia;

Cryptosporidum parvum;

Schistosoma spp.;

Naegleria fowleri

Pathogen Identification	***Giardia lamblia***	***Cryptosporidium parvum***	***Schistosoma* spp.**	***Naegleria fowleri***
Mode(s) of Transmission	The infectious cysts of *G. lamblia* are excreted in large numbers in feces of infected persons, and they contaminate hands, drinking water, swimming pool, and food. They can be transmitted by the fecal-oral route, or contaminated food and water. Sexual behaviours that aid in transmission of *G. lamblia* include oral-anal, genital-anal, and digital anal contact. Contaminated soil and fomites can also contain infective cysts. Cysts can survive in cold water for several weeks to months. At 4°C *Giardia lamblia* cysts can survive 11 weeks in water, seven weeks in soil, and one week in cattle feces. They remain infective for a significantly shorter period at warmer temperatures - i.e. 25°C. Trophozoites do not survive in the environment. Laboratory hazards include ingestion and possible aerosol and inoculation infection with contaminated water, faeces, and other materials.	Transmitted through the fecal-oral route, direct contact with infected humans or animals, contaminated food or water and aerosols. Laboratory hazards include ingestion and possible aerosol and inoculation infection. Outside of a host, *C. parvum* survive for 6 months at 20°C in the environment.	Transmission occurs in water contaminated with feces or urine. Free swimming cercariae directly penetrate through the skin to infect humans. Laboratory hazards include direct contact with contaminated fluids, ingestion and possible aerosol and inoculation infection. Cercariae may survive in water for about 2 days.	*N. fowleri* enters the nasal passage, carried in contaminated water, then penetrates through the mucosal layer and travels along the olfactory nerve to the brain. Ingestion of contaminated water does not lead to PAM. *N. fowleri* can survive in water at temperature up to 45°C and at pH 4.6 - 9.5. Primary laboratory hazards include aerosol inhalation and mucocutaneous and parenteral inoculation.
Incubation Period	7 to 14 days; however, the time from ingestion of the cysts to detection of the cysts may be longer than the incubation period, which can result in the stool sample being negative at the time of the onset of symptoms.	7 to 10 days.	Cercariae reach the portal venous system several days post-infection. 4-6 weeks usually pass before egg production begins. Toxemic schistosomiasis may develop 6-8 weeks post infection. Urinary	The first symptoms appear 1-7 days after infection, and death by PAM may occur 7-10 days after infection.

			schistosomiasis may develop 10-12 weeks post infection. Adult schistosomes may live 20 to 30 years.	
Period of Communicability	Can be transmitted from person-to-person, especially persons with poor fecal-oral hygiene, causing epidemics.	Highly contagious. Human-to-human transmission is common. Oocysts can be excreted up to 50 days after cessation of diarrhea.	Not directly transmitted from person to person.	Not transmitted from person-to-person.
Infectious Dose(s)	Human volunteers have been experimentally infected with as few as 10 cysts.	The median infectious dose in healthy adult volunteers is 132 oocysts. However, the infectious dose for humans is as low as 1-5 oocysts. Infectious dose is dependent on the immune status of the host, with immunodeficient persons being much more susceptible.	Unknown.	Unknown.
Typical Presenting Symptoms	The majority of infections are asymptomatic. In symptomatic individuals, patients may experience nausea, chills, low grade fever, epigastric pain and sudden onset of watery diarrhea. Diarrhea is often explosive and presents a foul smell without the presence of blood, gas, bloating, mucus, or cellular exudate. Most infections resolve spontaneously within six weeks. Chronic infections can occur and diarrhea leads to dehydration, malabsorption, weight lost and impaired pancreatic function. Chronic infections can last from months to years. *G. lamblia* are usually found in the upper small intestine, but can be found in the gall bladder and in biliary drainage.	*C. parvum* occurs worldwide and is ubiquitous in the environment. Cryptosporidiosis is in the top five most common causes of infectious diarrhea around the globe. Prevalence varies based on climate and level of development, accounting for 0.1-2% of diarrheal illness in cooler and developed areas and 0.5-10% in warmer and developing countries. Settings involving close contact with infected persons, including day-care centres, which increase transmission. Outbreaks have been associated with contaminated food, drinking, and recreational water. One outbreak linked to contaminated drinking water affected over 400,000 individuals in Milwaukee, Wisconsin.	Symptoms are related to the amount and location of eggs in the human host. Any species of *Schistosoma* can cause acute schistosomiasis. Acute schistosomiasis is characterized by cercarial dermatitis and Katayama syndrome. Cercarial dermatitis is unusual among individuals exposed for the first time such as visitors and migrants, and is rare among endemic populations, travellers or migrants. The clinical presentation of acute schistosomiasis includes fever, headache, generalized myalgia, abdominal pain, vertigo, vomiting, bloody diarrhea, and fatigue. When eggs become trapped in tissue, and they fail to clear, they can produce chronic schistosomiasis. The chronic disease, which is more	*N. fowleri* is the causative agent of primary amoebic meningoencephilitis (PAM). PAM is an acute, fulminating, rapidly fatal disease that is often observed after exposure to fresh water, with symptoms such as sore throat, blocked nasal passages, fever, vomiting, stiff neck, confusion, and abnormal behaviour. Three to four days after the onset of the initial symptoms, mental confusion and coma occur. Death usually occurs 3-4 days after coma. Time from infection to death is 7-10 days. Mortality rate is estimated at greater than 95%.

			prevalent in endemic areas, is due to a granulomatous response to parasitic eggs. Clinical presentation includes inflammation, hyperplasia, ulceration, and occult blood in feces. It is possible that there is an association between chronic schistosomiasis and colorectal cancer. When eggs are deposited in the liver, symptoms can include portal hypertension, hepato-splenomegaly, liver fibrosis, hepatic coma, ascites, and esophogeal varices. The latter are late stages of pipestem fibrosis with liver cell function relatively preserved early in the disease course. *S. japonicum* and *S. mansoni* are primarily associated with hepatic and intestinal pathology, including diarrhoea, abdominal pain, and hepato-splenomegaly. Urinary schistosomiasis is caused by *S. haematobium*, and chronic *S. hematobium* infection is the major risk factor for urinary tract carcinoma in Africa. The clinical presentation includes dysuria, hematuria, proteinuria, calcification in the bladder, obstruction of the ureter, renal colic, hydronephrosis, and renal failure. Secondary infection may occur with urinary schistosomiasis. It is associated with genital disease in 1 out of 3 infected women. *S. haematobium* is a leading cause of bladder	

			cancer. Lung and CNS involvement can occur, producing lesions in the brain (*S. japonicum and S. mekongi*) and spinal cord (*S. mansoni* and *S. haematobium*). Clinical manifestations of cerebral schistosomiasis include seizure, headache, acute encephalopathy, hemiparesis, and hemianopsia. Worms may survive for 30 years in the host.	
Mode(s) of Decontamination	Cysts are relatively resistant to chlorination but *Giardia* cysts in water can be inactivated with 4 mg/L of chlorine after 60 min at 5°C, at pH levels 6, 7 and 8. A chlorine concentration of 8 mg/L will inactivate *Giardia* cysts at pH 6 and 7 after contact for 10 min, and at pH 8 after 30 min. Also, at 25°C, *Giardia*cysts will be killed when exposed to 1.5 mg/L of chlorine for 10 min at pH 6. 6% H_2O_2can be used as a surface disinfectant or in disinfection of spills. *Giardia* is inactivated by exposure to UV light (10 JM^{-2}). Cysts are relatively resistant to ozonolysis. Cysts are susceptible to boiling and freezing.	*C. parvum* is susceptible to high concentration (> 6%) of hydrogen peroxide and ethylene oxide, ozone. It is resistant to low concentration of hydrogen peroxide, peracetic acid, sodium hypochlorite, phenolic, quaternary ammonium compound, 2% glutaraldehyde, ortho-phtalaldehyde, and 70% ethanol. Inactivated by moist heat (e.g. 121°C for 18 minutes), freezing (-70°C for seconds or -20°C for 24 hours), desiccation, and UV light. Use of "absolute" 1 µm filters.	2% glutaraldehyde, sodium hypochlorite, 70% ethanol. Sensitive to low temperatures; *S. japonicum* does not develop at temperatures lower than 15.4°C.	*N. fowleri* is susceptible to NaCl at concentrations greater then 1%, w/v. *N. fowleri* is susceptible to chlorine at concentrations of 0.5 and 1.0 mg/L, ozone, and Deciquam 222. Heating water to 50°C for 5 minutes will kill all forms of the amoebae. Both amoeba and cysts can tolerate temperature of 65°C for 1-3 minutes and temperatures below 20°C inhibit reproduction. Degradation occurs when temperatures reach below 10°C. Dehydration is lethal to *N. fowleri.*
Emergency Response	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms. Since the incubation time may be shorter than the period required for detection of the cysts in the stool, giardiasis should not be eliminated as a diagnostic possibility if a single stool specimen is negative at the onset of symptoms. Since *Giardia* is excreted	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms. Detection usually by direct microscopic observation of oocysts in stool specimens. Nucleic acid and antigen detection methods have also been developed. Illness is generally self-limiting in immuno-competent patients.	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Confirm through direct observation of eggs in stool or urine (*S. haematobium* and rarely *S. mansoni*). If a patient exhibits the clinical symptoms but lacks eggs in faeces or urine, a biopsy of the	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms. Identification is done by microscopic examination of CSF for presence of amoebic organism. Molecular biology techniques such as PCR and real-time PCR have been recently developed for detecting *N.*

	intermittently, in 50% to 70% of cases, *Giardia* will be detected from a single stool specimen and in 90% of cases after three specimens. In general, the initial diagnosis is made by identification of trophozoites or cysts in stool specimens, duodenal fluid, or small bowel tissue by direct microscopic examination. Other techniques such as detecting soluble stool antigens using enzyme immunoassay (EIA) or polymerase chain reaction technique are also used. Dehydration and electrolyte abnormalities should be treated symptomatically. In immunocompetent hosts, infection is self-limited; drug therapy can shorten the duration of symptoms and prevent transmission. Metronidazole, tinidazole, and nitazoxanide are indicated for first line treatment. Susceptible to metronidazole, tinidazole, nitazoxanide, quinacrine, furazolidone, paramomycin, and albendazole.	Rehydration and electrolyte therapy may be used in cases with severe diarrhea. Nitazoxanide is approved for treatment of cryptosporidiosis in children aged 1 to 10 years in the USA. It has also showed promise in immuno-compromised individuals. Immuno-compromised patients are often treated with paromomycin, letrazuril and azithromycin. Highly active antiretroviral therapy (HAART) is currently considered the best treatment option for life-threatening cryptosporidiosis in AIDS patients. Susceptible to nitazoxanide.	bladder or rectal mucosa can be performed. ELISA and PCR can be used, as well as a Kato-Katz thick smear. If too few eggs are excreted, the miracidium-hatching method can be employed. Praziquantel is the primary drug used against schistosomiasis. Artemether is not used where malaria is present to prevent *Plasmodium* from developing resistance. Many strains are resistant to oxamniquine. Resistance to praziquantel has been documented in laboratory strains and in the field.	*fowleri*. Treatment of PAM is rarely successful and depends on prompt diagnosis and administration of medication. The usual course of treatment involves amphotericin B administered in combination with rifampin and other antifungals. *N. fowleri* is susceptible to amphotericin B, which is often used in combination with rifampin, orindazol, miconazol, sulisoxazole, or chloramphenicol. Miltefosine and voriconazole has also been found to be effective against infection. Resistance of *Naegleria* spp. has been shown against fluconazole and itraconazole. This area remains a growing concern when repeated doses are administered, especially in endemic regions.
Suggested Reference: http://www.phac-aspc.gc.ca/lab-bio/res/psds-ftss/index-eng.php.				

Pathogen identification, Mode of Transmission, Incubation Period, Period of Communicability, Infectious Dose, Typical Presenting Symptoms, Mode(s) of Decontamination, and Emergency Response:

Untreated Sewage

Medical Surveillance for New Biologic Materials

Streptomyces spp.;

Pathogen Identification	Streptomyces spp.			
Mode(s) of Transmission	Streptomyces can be transmitted by exposure to contaminated soil. Contact with abraded or broken tissue carries higher risk. Aerosol infection is also a risk.			
Incubation Period	Unknown.			
Period of Communicability	Unknown.			
Infectious Dose(s)	Unknown although pre-existing conditions such as immunodeficiency, Lupus, or various cancers as well as the presence of invasive medical prostheses may increase infection risk.			
Typical Presenting Symptoms	Infection mostly manifest as chronic skin lesions characterized as mycetoma. However, lung-abscess pneumonitis, brain abscesses, septicemia, hypersensitivity pneumonitis, and general pulmonary infection are possible.			
Mode(s) of Decontamination	20% Sodium hypochlorite solution for 10 minutes, 24 hours of incubation in 70% isopropyl alcohol, and moist heat (121°C for at least 15 minutes).			

Emergency Response	If exposure is suspected, wash area thoroughly and encourage bleeding if, sharps or puncture injury. Seek emergency medical attention. Monitor for symptoms. Confirm infection through bacteriological and serological testing. Infection confirmation by 16S ribosomal RNA amplification and sequencing may be necessary. Resistance to penicillin, β-lactam, macrolide, glycopeptide, tetracycline, cephalosporin, and fluoroquinolone antibiotics has been documented.			
Suggested reference: https://www.ncbi.nlm.nih.gov/pubmed/17369139				