Biological Agent Information Papers
United States Army Institute of Infectious Diseases

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ANTHRAX

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Description of Agent: Anthrax is a highly lethal infection caused by infection with the Gram-positive bacterium, Bacillus anthracis. In naturally-acquired cases, organisms usually gain entrance through skin wounds (causing a localized infection), but may be inhaled or ingested. Intentional release by belligerents or terrorist groups would presumably involve the aerosol route, as the spore form of the bacillus is quite stable and possess characteristics ideal for the generation of aerosols.

Signs and Symptoms: The incubation period for inhalational anthrax is 1-6 days. Fever, malaise, fatigue, cough, and mild chest discomfort are rapidly followed by severe respiratory distress with dyspnea, diaphoresis, stridor, and cyanosis. Shock and death occur within 24-36 hours of the onset of severe symptoms. In cases of cutaneous anthrax, a papule develops, then vesicates, finally developing into a black eschar surrounded by moderate to severe edema. The lesions are usually painless. Without treatment, the disease may progress to septicemia and death, with a case-fatality rate of 20%. With treatment, fatalities are rare.

Diagnosis: Physical findings are typically non-specific in inhalational cases, with initial complaints of malaise, fever, headache, and possibly substernal chest pain. A widened mediastinum is sometimes seen on x-ray late in the course of illness, and correlates with a pathologic finding of hemorrhagic mediastinitis, the "classic" presentation of inhalational anthrax. The bacterium may be detected by Gram stain of blood and by blood culture late in the course of illness.

Treatment: Although usually ineffective in inhalational cases once symptoms are present, antibiotic treatment with high-dose penicillin, ciprofloxacin, or doxycycline should nonetheless be administered. Although typically sensitive T2: HPBXLA to penicillin, resistant isolates are readily produced in the laboratory. For this reason, in the case of an intentional release, and in the absence of antibiotic sensitivity data, treatment should be initiated with IV ciprofloxacin (400 mg q 8-12 hrs) or IV doxycycline (200 mg initially, followed by 100 mg q 12 hrs). Supportive therapy may be necessary.

Prophylaxis: A licensed vaccine is available for use in those at risk of exposure. Vaccination is undertaken at 0, 2, and 4 weeks (initial series), followed by booster doses at 6, 12, 18 months and then yearly. Oral ciprofloxacin (500 mg po bid) or doxycycline (100 mg po bid) is useful in cases of known or imminent exposure. Following confirmed exposure, all unimmunized individuals should receive three 0.5 ml SQ doses of vaccine over 30 days, while those vaccinated with < 3 doses prior to exposure should receive an immediate 0.5 ml booster. Anyone vaccinated with the initial 3-dose series in the previous 6 months does not require boosters. All exposed personnel should continue antibiotic therapy for 4 weeks. If vaccine is unavailable, antibiotics may be continued beyond 4 weeks and should be withdrawn only under medical supervision.

Decontamination and Isolation: Drainage and secretion precautions should be practiced. Anthrax is not known to be transmitted via the aerosol route from person to person. Following invasive procedures or autopsy, instruments and surfaces should be thoroughly disinfected with a sporicidal agent (high-level disinfectants such as iodine or 0.5% sodium hypochlorite).

Outbreak Control: Although anthrax spores may survive in the environment for many years, secondary aerosolization of such spores (such as by pedestrian movement or vehicular traffic) generally presents no problem for humans. The carcasses of animals dying in such an environment should be burned, and animals subsequently introduced into such an environment should be vaccinated. Meat, hides, and carcasses of animals in affected areas should not be consumed or handled by untrained and/or

unvaccinated personnel.

CHOLERA

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Description of Agent: Cholera is an infection caused by the bacterium Vibrio cholerae, and acquired through the ingestion of contaminated water or food. The disease manifests as a watery (secretory) diarrhea so profuse that supplies of IV fluids are often exhausted during epidemics. Intentional use by belligerents or terrorist groups would presumably involve the contamination of food or water sources. Cholera is incapacitating, but in the face of large numbers of casualties, and the breakdown in medical care often associated with war, a large number of deaths are possible.

Signs and Symptoms: The incubation period is 1-5 days; while a large number of infected persons remain asymptomatic, the "classic" form of cholera is noteworthy for its severity and sudden onset. Vomiting, abdominal distention and pain with little or no fever are followed rapidly by a profuse, watery diarrhea with a "rice-water" appearance. Fluid losses may readily exceed 10 liters per day. Without treatment, death may result from severe dehydration, hypovolemia and shock.

Diagnosis: The diagnosis is typically made clinically on the basis of profound watery diarrhea and consequent dehydration. Microscopic exam of stool samples reveals few or no red or white cells. The organism may be identified in stool by darkfield or phase contrast microscopy, and grows on a variety of culture media.

Treatment: The mainstay of therapy is fluid and electrolyte replacement. This may be accomplished through the use of oral rehydration salts or dilute Gatorade? in less severe cases, whereas IV fluids are often required in cases of severe dehydration. Antibiotics shorten the duration of diarrhea and thereby decrease fluid loss; tetracycline (500 mg q 6 hrs x 3 d) or doxycycline (300 mg once or 100 mg q 12 hrs x 3 d) are reasonable choices. Concerns over tetracycline resistance have recently arisen, and ciprofloxacin (500 mg q 12 hrs x 3 d), erythromycin (500 mg q 6 hrs x 3 d), furazolidone (100 mg q 6 hrs), or TMP/SMX (320 mg TMP bid) may also be considered.

Prophylaxis: A licensed, killed vaccine is available but only modestly effective, providing about 50 percent protection lasting for no more than 6 months. Vaccinations are given at 0 and 4 weeks, with booster doses every 6 months. Vaccine dose varies with age and with route of administration; intradermal, SQ, and IM injections are acceptable delivery means. The limited efficacy of the preparation has led most public health authorities to recommend against vaccination under most circumstances.

Decontamination and Isolation: Personal contact rarely causes infection; however, enteric precautions and careful hand-washing should be employed. Gloves should be used for patient contact and specimen handling. Bactericidal solutions, such as 0.5% hypochlorite, would provide adequate surface decontamination.

Outbreak Control: Strict attention must be paid to the avoidance of contaminated water in an outbreak area. Drinking water, as well as water used in bathing, washing utensils, and cooking, must be obtained from a safe source or must be boiled or chlorinated prior to use.

PLAGUE

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Description of Agent: Plague is an infectious disease caused by the Gram-negative, bipolar-staining bacterium, Yersinia pestis. Naturally-occurring plague is most often acquired by the bite of a flea which had previously fed on infected rodents. In such cases, plague classically presents as a localized abscess with secondary formation of very large, fluctuant regional lymph nodes known as buboes (bubonic plague). Plague may also be transmitted via aerosol and by inhalation of sputum droplets from coughing patients. In such instances, a primary pneumonic form may develop and, in the absence of prompt therapy, progress rapidly to death within 2-3 days. Intentional release by belligerents or terrorist groups would presumably involve aerosolization, but could also involve the release of infected fleas. Plague may be considered a lethal agent.

Signs and Symptoms: Pneumonic plague has an incubation period of 2-3 days, and begins with high fever, chills, headache, hemoptysis, and toxemia, progressing rapidly to dyspnea, stridor, and cyanosis. Death results from respiratory failure, circulatory collapse, and bleeding diatheses. Bubonic plague has an incubation period of 2 to 10 days, and presents with malaise, high fever, and tender lymph nodes (buboes). Bubonic plague may progress spontaneously to the septicemic form, with spread to the CNS, lungs, and elsewhere.

Diagnosis: To facilitate prompt therapy, plague must be suspected clinically. A presumptive diagnosis may also be made by Gram

or Wayson stain of lymph node aspirates, sputum, or CSF. The plague bacillus may be readily cultured from aspirates of buboes or from the blood of septicemic patients.

Treatment: Early administration of antibiotics is quite effective, but must be started within 24 hours of onset of symptoms in pneumonic plague. The treatment of choice is streptomycin (30 mg/kg/day IM in 2 divided doses x 10 days) or gentamicin (2 mg/kg, then 1.0-1.5 mg/kg q 8 hrs x 10 days). Intravenous doxycycline (200 mg, then 100 mg q 12 hrs x 10-14 days) is also effective; chloramphenicol should be added in cases of plague meningitis. Supportive therapy for pneumonic and septicemic forms is typically required.

Prophylaxis: A licensed, killed vaccine is available. The primary vaccination series consists of a 1.0 ml IM dose initially, followed by 0.2 ml doses at 1-3 months and 3-6 months. Booster doses are given at 6, 12 and 18 months and then every 1-2 years. As this vaccine appears in animal experiments to offer no protection against aerosol exposure, victims of a suspected attack with aerosolized plague, or respiratory contacts of coughing patients, should be given doxycycline (100 mg po bid x 7 days or the duration of exposure, whichever is longer).

Decontamination and Isolation: Drainage and secretion precautions should be employed in managing patients with bubonic plague; such precautions should be maintained until the patient has received antibiotic therapy for 48 hours and has demonstrated a favorable response to such therapy. Care must be taken when handling or aspirating buboes to avoid aerosolizing infectious material. Strict isolation is necessary for patients with pneumonic plague.

Outbreak Control: In the event of the intentional release of plague into an area, it is possible that local fleas and rodents could become infected, thereby initiating a cycle of enzootic and endemic disease. Such a possibility would appear more likely in the face of a breakdown in public health measures (such as vector and rodent control) which might accompany armed conflict. Care should be taken to rid patients and contacts of fleas utilizing a suitable insecticide; flea and rodent control measures should be instituted in areas where plague cases have been reported.

Q FEVER

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Description of Agent: Q fever is caused by infection with the rickettsial organism, Coxiella burnetii, and is typically spread by inadvertent aerosolization of organisms from infected animal products, especially the placentas of parturient sheep and goats. Person-to-person transmission rarely, if ever, occurs. Intentional release by belligerents or terrorist groups would presumably involve aerosolization, and Q fever would likely be employed as an incapacitating agent, as its' mortality rate is quite low (1-3%).

Signs and Symptoms: Q fever typically presents as an undifferentiated febrile illness, with fever, chills, cough, headache, weakness, and pleuritic chest pain occurring as early as ten days after exposure. Onset may be sudden or insidious. Pneumonia is present in some cases, but pulmonary syndromes are usually not prominent. Patients are not generally critically ill, and the illness lasts from 2 days to 2 weeks. Rarely, Coxiella burnetii may cause a peculiar form of chronic endocarditis, which is largely responsible for the few fatal cases.

Diagnosis: Q fever is not a clinically distinct illness and may resemble a viral illness or other types of atypical pneumonia. The organism is readily aerosolized, and a single organism may cause human disease. Consequently, cultivation of the organism represents a significant hazard to laboratory personnel, and the diagnosis should therefore be confirmed serologically.

Treatment: Q fever is generally a self-limited illness even without treatment. Tetracycline (500 mg q 6 hrs) or doxycycline (100 mg q 12 hrs) are the treatments of choice and are given orally for 5 to 7 days. Chloramphenicol would also be effective, but rarely warranted. Chronic forms of Coxiella infection are problematic to treat, and should be referred to specialists for care.

Prophylaxis: Treatment with tetracycline or doxycycline beginning 8-12 days following exposure and continued for 5 days should prevent the onset of symptoms. An inactivated whole cell vaccine is available as an investigational agent through USAMRIID (Ft Detrick MD 21702), and is effective in eliciting protection against exposure, but severe local reactions to this vaccine may be seen in the sizable minority of the population who already possess immunity. Skin testing of potential vaccine recipients is thus recommended.

Decontamination and Isolation: Patients exposed to Q fever by the aerosol route do not present a risk for secondary contamination or re-aerosolization of the organism. Decontamination is accomplished with soap and water or by the use of weak (0.5 percent) hypochlorite solutions.

Outbreak Control: Spore-like forms of Coxiella burnetii may withstand quite harsh conditions and thus persist in the environment for prolonged periods. Presumably, animals, especially sheep, in such areas would be at risk for acquiring infection, and contact

with the products of pregnancy of such animals would represent a continuing hazard to humans. Little information exists to permit assessment of direct long-term hazards to humans entering an area contaminated by intentional release of aerosolized Q fever.

BOTULISM

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Description of Agent: Botulinum toxins are a group of seven related neurotoxins (types A-G) produced by the anaerobic bacterium, Clostridium botulinum. They are typically formed in canned foods and subsequently ingested, although the spore form of the organism may occasionally gain access to the body through wounds or through the GI tract before germinating and producing toxin. Intentional release by belligerents or terrorists would likely involve aerosolization of pre-formed toxin, which could then produce disease via the inhalational route. Deliberate contamination of food supplies is also possible. Botulinum toxins act by blocking acetylcholine release at the neuromuscular junction, and in the central and peripheral nervous systems. In the face of large numbers of casualties and/or in the absence of prompt, intensive, and long-term medical management, botulism can be thought of as a lethal agent.

Signs and Symptoms: Ptosis, generalized weakness, dizziness, dry mouth and throat, blurred vision and diplopia, dysarthria, dysphonia, and dysphagia are followed by symmetrical descending flaccid paralysis and the development of respiratory failure. Symptoms may begin as early as 12-36 hours following ingestion or inhalation, but may require as long as several days in some cases.

Diagnosis: The diagnosis of botulism is made clinically, as there are no specific laboratory findings, and a limited differential diagnosis. Assays for toxin are not widely available. Intentional release should be suspected if numerous co-located casualties present with progressive descending bulbar, muscular, and respiratory weakness.

Treatment: Supportive care is the mainstay of therapy, and consists chiefly of intubation and ventilatory assistance for respiratory respiratory failure; tracheostomy may be required. A licensed trivalent equine botulinum antitoxin (types A, B, and E) is available through the CDC and should be administered as soon as possible in order to bind toxin remaining in the circulation. An investigational heptavalent despeciated product, also prepared in horses, is available through USAMRIID (Ft Detrick MD 21702). Skin testing should be performed before administration of equine antitoxins.

Prophylaxis: A pentavalent toxoid (types A, B, C, D, and E) is available through USAMRIID as an investigational product for those at high risk of exposure. Doses (0.5 ml) are given SQ at 0, 2, and 12 weeks, with yearly boosters.

Decontamination and Isolation: Decontamination of surfaces contaminated by toxin may be accomplished using soap and water, or 0.5% hypochlorite. Spores are best killed by pressure-cooking of foodstuffs to be canned. Toxin is not dermally active (although spores may enter through skin wounds) and secondary aerosols from affected patients pose no risk of botulism transmission.

Outbreak Control: Intentionally-released aerosols of botulinum toxin probably pose little risk beyond the immediate period of release. In the event that contamination of foodstuffs is suspected, pre-formed toxin may be destroyed by boiling for 10 minutes.

STAPHYLOCOCCAL ENTEROTOXIN B DISEASE

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Description of Agent: Staphylococcal enterotoxin B (SEB) is one of several toxins produced by the bacterium Staphylococcus aureus. SEB is a common contributor to staphylococcal food poisoning but could be employed by belligerents or terrorist groups as an aerosolized inhalational agent. It is incapacitating, but would rarely be expected to produce lethality.

Signs and Symptoms: Symptoms would be expected to begin 3-12 hours after aerosol exposure, and consist of sudden onset of fever, chills, headache, myalgia, and nonproductive cough. Some patients may develop shortness of breath and retrosternal chest pain. Fever may last 2 to 5 days, and cough may persist for up to 4 weeks. Patients ingesting toxin might present with nausea, vomiting, and diarrhea. Very high exposure levels may lead to pulmonary edema and, rarely, death.

Diagnosis: The diagnosis of SEB intoxication is largely clinical. As inhalational disease due to SEB is not encountered naturally, its presence strongly indicates intentional aerosolization. In this regard, one would expect to see large numbers of patients present with a febrile respiratory syndrome but without CXR abnormalities.

Treatment: Treatment is limited to supportive care. Artificial ventilation may be required in very severe cases, and attention to

fluid management is important. Antibiotics are of no benefit, and no antitoxin has been developed.

Prophylaxis: There is currently no human vaccine available to prevent SEB intoxication. As with all potential inhalational biological agents, protective masks such as those employed by military units offer excellent protection in those individuals alert to the possibility of attack.

Decontamination and Isolation: Decontamination of most surfaces may be accomplished with soap and water or with exposure to 0.5% hypochlorite solution. Food which may have been contaminated should be destroyed.

Outbreak Control: Prolonged environmental contamination would not be expected following release of aerosolized SEB.

TULAREMIA

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Description of Agent: Tularemia is an infection caused by the Gram-negative coccobacillus, Francisella tularensis. Two biogroups are known; biogroup tularensis, also known as type A, is the more virulent form, and is endemic in much of North America. Naturally-acquired tularemia is contracted through the bites of certain insects (notably ticks and deerflies), or via contact with infected rabbits, muskrats, and squirrels. Intentional release by belligerents would presumably involve aerosolization of living organisms. Although naturally-acquired tularemia has a case-fatality rate of approximately 5%, the pneumonic form of the disease, which would predominate in the setting of intentional release, would likely have a greater mortality rate.

Signs and Symptoms: Naturally-acquired tularemia frequently has an ulceroglandular presentation, although a significant minority of cases involve the typhoidal or pneumonic forms. The incubation period averages 3-5 days, but varies widely. Use of tularemia as a weapon would likely lead to a preponderance of pneumonic and typhoidal cases, and large aerosolized innocula would be expected to shorten the incubation period. Ulceroglandular disease involves a necrotic, tender ulcer at the site of inoculation, accompanied by tender, enlarged regional lymph nodes. Fever, chills, headache, and malaise often accompany these findings. Typhoidal and pneumonic forms often involve significant cough, abdominal pain, substernal discomfort, and prostration in addition to prolonged fever, chills, and headache.

Diagnosis: Prompt diagnosis relies on clinical suspicion. Routine laboratory tests are rarely helpful, and F. tularensis does not typically grow in standard blood cultures, although special media are available for the culturing (under BL-3 containment conditions) of blood, sputum, lymph node material, and wound exudates if the diagnosis is suspected. Serology is available to confirm the diagnosis in suspected cases.

Treatment: Streptomycin (7.5-15 mg/kg im q 12 hrs for 7-14 days) is the drug of choice for all forms of tularemia. Gentamicin (3-5 mg/kg/d (q 8-12 hrs for 7-14 days) is an acceptable alternative. Relapses are more common with tetracycline (500 mg po q 6 hrs for 14 days) therapy, although this alternative may be employed in patients who cannot tolerate aminoglycosides.

Prophylaxis: A live, attenuated vaccine is available as an investigational product through USAMRIID (Ft Detrick MD 21702). It may be given to those, such as laboratory workers, at high risk of exposure. A single dose is administered by scarification. Intramuscular streptomycin will prevent disease following documented exposure, but is not recommended following tick bites or animal contact.

Decontamination and Isolation: Tularemia is not transmitted person-to-person via the aerosol route, and infected persons should be managed with secretion and drainage precautions. Heat and common disinfectants (such as 0.5% hypochlorite) will readily kill F. tularensis organisms.

Outbreak Control: Following intentional release of F. tularensis in a given area, it is possible that local fauna, especially rabbits and squirrels, will acquire disease, setting up an enzootic mammal-arthropod cycle. Persons entering such an area should avoid skinning and eating meat from such animals. Water supplies and grain in such areas might likewise become contaminated, and should be boiled or cooked before consumption. Organisms contaminating soils are unlikely to survive for significant periods of time and present little hazard.

VARIOLA (SMALLPOX)

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Description of Agent: Smallpox is an infection cause by Variola virus, a member of the chordopoxvirus family. Naturally occurring smallpox has been eradicated from the globe, with the last case occurring in Somalia in 1977. Repositories of virus are

known to exist in only two laboratories worldwide. Monkeypox, Cowpox, and Vaccinia are closely-related viruses which might lend themselves to genetic manipulation and the subsequent production of smallpox-like disease.

Signs and Symptoms: The incubation period of smallpox is about 12 days. Clinical manifestations begin acutely with a prodromal period involving malaise, fevers, rigors, vomiting, headache, and backache. After 2-4 days, skin lesions appear and progress uniformly from macules to papules to vesicles and pustules. Lesions progress centrifugally and scab in 1-2 weeks. In unvaccinated individuals, Variola Major, the classical form of the disease, is fatal in approximately 30% of cases.

Diagnosis: In its full-blown form as typically seen in unimmunized individuals, smallpox is readily diagnosed on clinical grounds. Differentiation from other vesicular exanthems such as varicella and erythema multiforme might be difficult, however, in cases of Variola Minor or in disease modified by prior vaccination. Electron microscopy can readily differentiate variola virus from varicella but not from vaccinia and monkeypox when performed on lesion scrapings. The virus can be grown in chorioallantoic membrane culture.

Treatment: Supportive care is the mainstay of smallpox therapy. No specific antiviral therapy exists.

Prophylaxis: A licensed, live Vaccinia Virus vaccine is available, and is administered via a bifurcated needle using a multiple puncture technique (scarification). Given the eradication of smallpox, vaccine would only be indicated in laboratory settings or where biological warfare was a distinct possibility. Vaccination is probably protective for at least 3 years. Exposed persons may be managed with prompt vaccination. Vaccinia Immune Globulin (VIG), given IM at a dose of 0.6 ml/kg, may prove a useful adjunct to vaccination, although its precise role is unclear.

Decontamination: Given the extreme public health implications of smallpox reintroduction, patients should be placed in strict isolation pending review by national health authorities. All material used in patient care or in contact with smallpox patients should be autoclaved, boiled, or burned.

Outbreak Control: Smallpox has considerable potential for person-to-person spread. Thus, all contacts of infectious cases should be quarantined for 16-17 days following exposure, and given prophylaxis as indicated. Animals are not susceptible to smallpox.

BRUCELLOSIS

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Description of Agents: Human Brucellosis is an infection caused by one of four species of Gram-negative coccobacilli of the genus Brucella. B. abortus is normally a pathogen of cattle, while B. melitensis, B. suis, and B. canis are pathogens of goats, pigs, and dogs, respectively. Organisms are acquired by humans via the oral route through the ingestion of unpasteurized milk and cheese, via inhalation of aerosols generated on farms and in slaughterhouses, or via inoculation of skin lesions in persons with close animal contact. Intentional exposure by belligerents would likely involve aerosolization but could involve contamination of foodstuffs.

Signs and Symptoms: The incubation period is quite variable, with symptoms often requiring months to appear; this marked variability would appear to temper somewhat the use of Brucellae as weapons. Symptoms of acute and subacute brucellosis are quite non-specific and consist of irregular fever, headache, profound weakness and fatigue, chills and sweating, and generalized arthralgias and myalgias. Depression and mental status changes are noteworthy. Osteoarticular complications, particularly involving the axial skeleton (sacroiliitis, vertebral osteomyelitis) are common. Fatalities are uncommon, even in the absence of therapy.

Diagnosis: Naturally-occurring cases may often be suspected based on a history of close animal contact or consumption of implicated foodstuffs. Brucellae may be isolated from standard blood cultures, but require a prolonged period of incubation; cultures should thus be maintained for six weeks if brucellosis is suspected. Bone marrow cultures yield the diagnosis in a higher percentage of cases than do peripheral blood cultures. A serum agglutination test is available and often helpful.

Treatment: Doxycycline (100 mg po bid) plus rifampin (600-900 mg po qd) administered for six weeks is the regimen of choice for uncomplicated brucellosis. Doxycycline + streptomycin, TMP/SMX + gentamicin, and ofloxacin + rifampin are acceptable alternative regimens.

Prophylaxis: Avoidance of unpasteurized milk products and appropriate veterinary vaccination practices are sufficient to prevent most naturally-occurring brucellosis. Persons inadvertently exposed to veterinary vaccine strains of brucella have been successfully prophylaxed with doxycycline + rifampin for 10 days. No human brucellosis vaccine is available in the western world.

Decontamination and Isolation: Drainage and secretion precautions should be practiced in patients who have open skin

lesions; otherwise no evidence of person-to-person transmission of brucellosis exists. Animal remains should be handled utilizing universal precautions and disposed of properly. Surfaces contaminated with brucella aerosols may be decontaminated by standard means (0.5% hypochlorite).

Outbreak Control: In the event of an intentional release of brucella organisms, it is possible that livestock will become infected. Thus, animal products in such an environment should be pasteurized, boiled, or thoroughly cooked prior to consumption. Proper treatment of water, by boiling or iodination, would also be important in an area subjected to intentional contamination with brucella aerosols.

RICIN INTOXICATION

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Description of Agent: Ricin is a protein toxin which acts as a cellular poison and is readily produced from castor beans (Ricinus communis), which are ubiquitous throughout the world. Waste from the commercial production of castor oil contains 5% ricin, making it easy for such a substance to fall into the hands of bioterrorists. Naturally-occurring cases of ricin intoxication involve ingestion of castor beans, and are marked by severe gastrointestinal symptoms, vascular collapse, and death. As ricin is toxic by numerous exposure routes, however, its use by belligerents might involve poisoning of water or foodstuffs, inoculation via ricin-laced projectiles, or aerosolization of liquid ricin or lyophilized powder. When used as an aerosol, cell death in lung tissue and pulmonary capillaries would be expected to lead to pulmonary edema and hypoxic respiratory failure.

Signs and Symptoms: When inhaled as a small particle aerosol, ricin would likely produce symptoms within 8 hours. Fever, cough, dyspnea, nausea, and chest tightness are followed by profuse sweating, the development of pulmonary edema, cyanosis, hypotension, and finally respiratory failure and circulatory collapse. Time to death would likely be 36-72 hours, depending on the dose received.

Diagnosis: The diagnosis of ricin intoxication is largely clinical and should be suspected in a setting of mass casualties with a similar and appropriate clinical picture. Failure to respond to antibiotics helps to differentiate ricin exposure from pulmonary infections produced by bacterial agents. An ELISA exists and may be performed on paired acute and convalescent sera.

Treatment: No specific treatment exists, and care is thus supportive. In cases of gastrointestinal exposure, gut decontamination via lavage, activated charcoal, and cathartics is warranted. Large amounts of volume replacement may be necessary.

Prophylaxis: A protective mask offers protection from aerosol exposure, but no specific vaccine or antitoxin exists.

Decontamination and Isolation: Ricin may be inactivated with 0.5% hypochlorite. Since it is not dermally active and is involatile, decontamination may not be as critical as with certain other biological and chemical agents.

Outbreak Control: Ricin does not, in general, pose a risk of secondary aerosolization.

VENEZUELAN EQUINE ENCEPHALITIS

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Description of Agent: Venezuelan Equine Encephalitis (VEE) is a mosquito borne alphavirus disease maintained in nature predominantly in a horse-mosquito-horse cycle, although thousands of natural human infections also occur each year. Large equine epizootics typically precede the appearance of human cases. Use of VEE as a weapon would presumably involve aerosolization, and such usage might be suspected on the basis of a preponderance of human cases and/or the presence of VEE outside of its typically well-localized focus in the Americas.

Signs and Symptoms: VEE may be thought of as an incapacitating agent with a mortality rate (in naturally-occurring disease) of less than 1%. Susceptibility is nearly 100%, and disease is characterized by its sudden onset following a 1-5 day incubation period. Initial symptoms include generalized malaise, severe headache, fever and rigors, photophobia, and myalgias. Cough, sore throat, and vomiting and diarrhea may follow. Only a small percentage of cases actually progress to encephalitis, which is more frequent in young children and is marked by meningismus, convulsions, coma, and paralysis. Route of exposure probably has little effect on the proportion of cases developing neurologic disease. In the majority of cases without neurologic sequelae, full recovery occurs in 1-2 weeks.

Diagnosis: The diagnosis of VEE is largely clinical. Naturally-occurring disease can often be suspected on epidemiologic grounds given its geographic specificity and the likely presence of concomitant disease in equines. Significant leukopenia and lymphopenia

is often present. Virus may be cultured from serum and a VEE-specific IgM ELISA is available.

Treatment: Treatment of VEE is largely supportive. Ribavirin has some in vitro activity, but appears of little benefit clinically.

Prophylaxis: Prevention of naturally-occurring VEE is dependent largely upon mosquito control efforts and the immunization of horses. A human vaccine, TC-83, is available as an investigational product through USAMRIID (Ft Detrick MD 21702), and has been given to a large number of laboratory workers as a single 0.5 ml SQ dose. A second investigational vaccine, formalin-inactivated C-84, has been used to boost (0.5 ml SQ) serologic non-responders.

Decontamination and Isolation: Universal precautions should be practiced when dealing with VEE patients. Virus may be destroyed by heat (80(C for 30 minutes) and by ordinary disinfectants (such as 0.5% hypochlorite).

Outbreak Control: Humans are infectious for mosquitoes for at least 72 hours after the onset of symptoms. Efforts at mosquito control thus become paramount to the prevention of secondary VEE cases following intentional or natural VEE outbreaks. In the event of intentional release of VEE virus by belligerents, the potential would be high for the development of an equine epizootic if the proper mosquito vector were present; veterinary vaccination would be useful in such circumstances.

TRICOTHECENE MYCOTOXICOSIS

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Description of Agent: The tricothecene mycotoxins are a large group of low-molecular-weight toxins produced by several species of filamentous fungi. Most are potent inhibitors of eukaryotic protein synthesis and of mitochondrial respiration. Those toxins most frequently isolated from agricultural products, and likewise mentioned most often in the context of belligerent use, include diacetoxyscirpenol (DAS), Nivalenol, 4-Deoxynivalenol (DON), and especially T-2. T-2 is one of the most stable of these toxins, and thus, perhaps, the most likely to be employed in a terrorist or warfare application. Naturally-occurring mycotoxicosis presents as Alimentary Toxic Aleukia, a lethal condition related to the consumption of moldy grains. Intentional use of T-2 by belligerents might involve aerosolization or the deliberate contamination of foodstuffs. Disease results from inhalation, ingestion, or skin contact, since T-2, unlike other BW agents, possesses significant vesicant properties.

Signs and Symptoms: Dermal exposure leads to symptoms within minutes and manifests as erythema accompanied by pain and a burning sensation. Blisters form and progress to necrosis with a leathery blackening of he skin. Inhalational exposure produces a rapid onset of nose and throat pain, with nasal discharge, cough, dyspnea, wheezing, chest pain, and hemoptysis. Eyes are likewise affected with intense burning and a foreign body sensation. Gastrointestinal exposure leads to anorexia, nausea, abdominal cramping, and hematemesis and hematochaezia. Systemic toxicity may follow exposure by any route and is manifest by weakness and ataxia, followed, in fatal cases, by tachycardia, hypothermia, and hypotension. Survivors of acute illness may manifest hematologic toxicity in the subacute phase.

Diagnosis: Prompt diagnosis is based on clinical and epidemiologic grounds. Blood, tissue, and environmental samples may be assayed for confirmatory evidence using gas-liquid chromatography and mass spectrometry.

Treatment: Therapy is largely supportive. Standard poison management techniques, such as the use of superactivated charcoal, are useful when administered early to casualties with gastrointestinal exposure.

Prophylaxis: Physical means, such as protective masks, are the only available protection. Unlike the situation with most other BW agents, the skin must also be protected against mycotoxin attack.

Decontamination and Isolation: Clothing of T-2 victims should be removed and treated (exposed to 5% hypochlorite for 6-10 hours) or destroyed. Skin may be decontaminated with soap and water. Eye exposure should be managed with copious saline irrigation. Isolation is not required. Instruments and surfaces should be decontaminated by heating to 500(F for 30 minutes or by brief exposure to 1N NaOH. Standard disinfectants effective against most other BW agents are often inadequate to inactivate the very stable mycotoxins.

Outbreak Control: Mycotoxin-induced disease is not contagious, but the stability of the toxins in the presence of heat and ultraviolet light make for the possibility of persistence in the environment following release.

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