Objectives

1. Terminal Learning Objective.

Given the background information regarding geographical distribution; epidemiology; and unique pathogenesis of military significant disease agents, discuss the military impact of microbiological public health and biological warfare threats in accordance with (IAW) lesson references.

2. Enabling Learning Objectives.

a. Discuss the considerations for assessing disease risk for travelers IAW lesson content.

b. Discuss the microorganism(s) responsible for causing specific microbial diseases of military significance IAW lesson content.

c. Discuss the disease, geographical distribution, mode of transmission, clinical manifestations, and/or laboratory diagnosis caused by specified microbial diseases of military significance IAW lesson content.

d. State the purpose and characteristics of biological warfare IAW lesson content.

e. State likely agents, potential threats, and delivery mechanisms of biological warfare IAW lesson content.

f. State the principles for determining whether or not a disease outbreak is likely to be a biological warfare attack IAW lesson content.

g. Discuss the current medical microbiology laboratory detection and/or diagnosis of microbial agents of military significance, including biological warfare agents IAW lesson content.

This Mimeo supersedes M 54W6LD-1M/F, 09/93.
A. Considerations for assessing disease risk for travelers\(^1\).

1. Incidence and prevalence of endemic disease.
   a. Endemic diseases recognized by local populations.
   b. Recent outbreaks of disease in the region.
   c. Diseases encountered during previous travel to area.

   a. Symptomatic vs. asymptomatic infections.
   b. Severity of illness and medical complications
   c. Incubation period and potential impact on accomplishing travel objectives.
   d. Hospitalization and medical evacuation requirements.

3. Risk factors associated with specific travel activity.
   a. Climate factors.
   b. Seasonality of diseases.
   c. Topographic considerations.
   d. Level of sanitation.
   e. Urban vs. rural exposures.
   f. Contact with local populations.

4. Environmental conditions.
   a. Intensity and duration of exposure to vectors or etiologic agents.
   b. Animal hosts in the area.

5. Preventive measures.
   a. Field sanitation and personal hygiene.
   b. Immunization for specific diseases.

\(^1\) Strickland--Hunter's Tropical Medicine, 7th ed., 1991, pg. 1048-1067.
c. Chemoprophylaxis against specific diseases.
d. Vector control and personal protective measures.

B. Diseases Presenting as Diarrhea and Dysentery—usually the leading cause of incapacitation during troop deployments.

1. Clinical manifestations/symptoms are as follows:
   a. Acute onset with watery diarrhea, vomiting and abdominal pain.
   b. Dehydration.
   c. Cramps.
   d. Fever.
   e. Dysentery (stools with blood, pus and mucus).
   f. Anorexia.

2. Mode of transmission—ingestion of water and food contaminated by feces of patients, carriers, and animals that are infected.

3. Etiological agents and disease name.
   a. Vibrio cholera (various serogroups)—Cholera: massive fluid loss due to the effects of toxins on the intestinal lining.

   b. Escherichia coli—intestinal infections with toxin production (also known as "Traveler's Diarrhea").
      (1) Enterotoxigenic Escherichia coli (ETEC)—acute, watery diarrhea, dehydration.
      (2) Enteroinvasive Escherichia coli (EIEC)—dysentery-like diarrhea.
      (3) Enteropathogenic E. coli (EPEC)—protracted, relapsing watery diarrhea.
      (4) Enterohemorrhagic E. coli (EHEC)—grossly bloody diarrhea.

   c. Shigella species, (serogroups A, B, C and D)—Shigellosis, bacillary dysentery.

   d. Campylobacter jejuni—direct contact, ingestion of contaminated meat, raw milk, and water.

   e. Salmonella species (approximately 2,000 serologic types)—Salmonellosis (non-typhoid fever, enteric fever, Salmonella "food poisoning").

   f. Rotavirus—ranges from subclinical to mild infection to acute
self-limiting illness lasting 3-8 days or to severe gastroenteritis.

g. Norwalk virus--shellfish and plankton natural reservoir; lasts 24 to 60 hours.

h. Entamoeba histolytic--Amebiasis (amebic dysentery, amebic colitis, amebic liver abscess); oral ingestion of food or water contaminated with human feces containing cysts; intestinal infection often benign (commensal) with few or no symptoms; hepatic and systemic lesions.

i. Giardia lamblia--Giardiasis (giardia enteritis, lambliasis); beaver and other wild or domestic animals.

   (1) Acute stage.

   (a) Explosive diarrhea with epigastric discomfort.

   (b) Bloating and belching.

   (2) Chronic or subacute stage.

   (a) Loose to mushy stools, intermittent diarrhea.

   (b) Constipation.

   (c) Malnutrition.

4. Incubation period--few hours (8-24 hrs) to several days (1-5 days) or several months.

   a. Food poisoning (Salmonellosis)--12 to 24 hours.

   b. Enteric fever--10 to 20 days.

5. Epidemiology.

   a. Widespread throughout the world with individual agents having specified geographic distribution.

   b. Among travelers returning from third world countries or who have eaten contaminated food or drank contaminated water.

   c. Seasonal--higher incidence with increased temperatures and humidity--Vibrio cholera, Shigella, Giardia lambia.

   d. Rotavirus--infants (6-12 months) winter season.

   e. Increased rates in areas with poor sanitation, slum conditions, poor personal hygiene practices--Entamoeba histolytic and Giardia lamblia.

6. Laboratory diagnosis.

   a. Laboratory isolation of etiological agent on selective and differential media from fecal clinical specimens (MacConkey, Hektoen, XLD, SS, TCBS, Sorbitol-MacConkey).
b. Slide agglutination for Salmonella and Shigella, Campylobacter jejuni, Vibrio cholera, and Rotavirus.

c. Wet mounts examination for motility—Giardia lamblia, Entamoeba histolytic.

d. ELISA and EIA—Rotavirus, Norwalk virus, Giardia lamblia and others.

e. Immunofluorescence—E. coli, Giardia lamblia, and others.

f. Direct fecal smear—Entamoeba histolytic, Giardia lamblia and Campylobacter jejuni (gram stain).

g. Concentration methods (FEA, MIF)—for parasites.

h. String test—Giardia lamblia.

i. Nucleic acid probes for toxin genes of E. coli.

7. Prevention and control.

a. Avoid employing local persons of third world countries as food handlers.

b. Enforce sanitation regulations.

c. Heat treatment and pasteurization of animal foods—Salmonella species.

d. Education of populations and personnel with respect to proper personal hygiene, food and fecal sanitation, and food handling.

e. Immediately remove anyone from kitchen duties who becomes ill and/or infected.

f. Enforce water supply maintenance regulations.

g. Immerse fresh vegetables and fruit in a solution of chlorinated water made by adding one-fourth cup of chlorine bleach to each gallon of water. Tear off the outer leaves of lettuce and break up the head so that all parts of the leaves are exposed to the chlorinated water. Leave the vegetables and fruit immersed for at least 10 minutes, then rack to drain. DO NOT RINSE. The chlorine odor and taste will soon dissipate. Remember that the organism is invisible to the eye and ONLY
chlorine will kill it.\textsuperscript{2}

8. Treatment.

a. Oral rehydration and intravenous electrolyte fluid replacement when necessary.

b. Antibiotic therapy--Erythromycin, Chloramphenicol.

c. Quinacrine, Metronidazole, Furazolidone--\textit{Entamoeba histolytica} and \textit{Giardia lamblia}.

### Diseases Presenting as Diarrhea and Dysentery

<table>
<thead>
<tr>
<th>Agent</th>
<th>Mode of Transmission</th>
<th>Clinical Manifestations</th>
<th>Laboratory Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Vibrio cholerae</em></td>
<td>oral ingestion of contaminated water and food</td>
<td>profuse, watery diarrhea; rapid dehydration</td>
<td>culture ID, slide agglutination</td>
</tr>
<tr>
<td><em>Enterotoxigenic Escherichia coli</em> (ETEC)</td>
<td></td>
<td>acute, watery diarrhea; dehydration</td>
<td>culture ID, Sorbitol-MacConkey</td>
</tr>
<tr>
<td><em>Enteroinvasive Escherichia coli</em> (EIEC)</td>
<td></td>
<td>dysentery</td>
<td>serologic ID</td>
</tr>
<tr>
<td><em>Enteropathogenic Escherichia coli</em> (EPEC)</td>
<td></td>
<td>protracted, relapsing diarrhea</td>
<td>Nucleic acid probes to toxin</td>
</tr>
<tr>
<td><em>Enterohemorrhagic Escherichia coli</em> (EHEC)</td>
<td></td>
<td>grossly bloody diarrhea</td>
<td></td>
</tr>
<tr>
<td><em>Shigella spp.</em> groups A,B,C,D</td>
<td></td>
<td>dysentery</td>
<td>culture ID</td>
</tr>
<tr>
<td><em>Campylobacter jejuni</em></td>
<td>contaminated meat, raw milk, water</td>
<td></td>
<td>slide agglutination</td>
</tr>
<tr>
<td><em>Salmonella spp.</em> non-typhoidal enteric fever, food poisoning</td>
<td>oral ingestion of contaminated food and water</td>
<td>diarrhea</td>
<td></td>
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<tr>
<td><em>Rotavirus</em></td>
<td></td>
<td>severe gastroenteritis</td>
<td>slide aggl., EIA/ELISA</td>
</tr>
<tr>
<td><em>Norwalk virus</em></td>
<td></td>
<td>diarrhea</td>
<td>EIA/ELISA</td>
</tr>
<tr>
<td><em>Entamoeba histolytic</em></td>
<td></td>
<td>amoebic dysentery</td>
<td>wet mount FEA, MIF, IFA</td>
</tr>
<tr>
<td><em>Giardia lamblia</em></td>
<td></td>
<td>explosive diarrhea, intermittent diarrhea, constipation</td>
<td>wet mount FA, MIF, EIA/ELISA, IFA, string test</td>
</tr>
</tbody>
</table>
C. Diseases Presenting as Fever.

1. Malaria.
   a. Clinical manifestations--fever and chills.
   b. Mode of transmission--bite of infected female Anopheles mosquito.
   c. Etiological agents.
      (1) Plasmodium falciparum.
      (2) Plasmodium vivax.
      (3) Plasmodium ovale.
      (4) Plasmodium malariae.
   d. Incubation period--12 to 30 days.
   e. Epidemiology--primarily tropical areas.
   f. Laboratory diagnosis--demonstration of intracellular parasitic forms in blood films by Giemsa or Wright staining.
   g. Prevention and control.
      (1) Mosquito control.
      (2) Prophylaxis, nets, and insect repellents.

2. Typhoid fever and paratyphoid fever (typhus abdominalis and enteric fever).
   a. Clinical manifestations.
      (1) Fever.
      (2) Malaise.
      (3) Rose spots on trunk.
      (4) Intestinal hemorrhage.
   b. Mode of transmission--fecal to oral (both patients and chronic carriers).
   c. Etiological Agent--Salmonella typhi and Salmonella paratyphi.
   d. Incubation period--1-3 weeks.
   e. Epidemiology.
      (1) Widespread communicable disease in countries where sanitary conditions are poor.
      (2) Public health problem in Central and South America, Africa, Asia and western Pacific.
(3) Tropics and subtropics—seasonal, peak in hot, dry months of the year.

f. Laboratory diagnosis.

(1) Laboratory isolation of etiological agent on selective and differential media from blood, urine, and fecal clinical specimens.

(2) Fourfold rise in agglutination titer in paired sera.

g. Prevention and control.

(1) Maintain safe water supplies and effective sewage systems.

(2) Surveillance of chronic carriers and their occupational activities as food handlers.

(3) Immunization.

h. Treatment—Chloramphenicol.

3. Rocky Mountain spotted fever; Boutonneuse fever; Rickettsialpox; Epidemic typhus; Murine typhus; Scrub typhus; Trench fever; and Q-fever.

a. Clinical manifestations.

(1) Sudden onset of fever.

(2) Myalgias—muscle pain.

(3) Arthralgias—severe pain in joints(s).

(4) Rash.

b. Etiological agents and mode of transmission.

(1) Rocky Mountain spotted fever—Rickettsia rickettsii, bites of infected ticks.

(2) Boutonneuse fever—Rickettsia conorii, bites of infected ticks.

(3) Rickettsialpox—Rickettsia akari, infected mites.

(4) Epidemic typhus—Rickettsia prowazekii, infected body lice.

(5) Murine typhus—Rickettsia typhi, infected fleas.

(6) Scrub typh—Rickettsia tsutsugamushi, infected chiggers.

(7) Trench fever—Rochalimaea quintana, infected body lice.

(8) Q-fever—Coxiella burnetii, contracted from domestic
animals by inhalation of infectious aerosols.

c. Incubation period--1 to 2 weeks, commonly 12 days.

d. Epidemiology.

(1) Rocky mountain spotted fever--limited to Western Hemisphere (infrequent in Rocky Mountains).
(2) Boutonneuse fever--Africa, Mediterranean Countries.
(3) Rickettsialpox--small outbreaks in urban tenements.
(4) Epidemic typhus--highlands of Africa, Central and South America.
(5) Murine typhus--worldwide.
(6) Scrub typhus--Western Pacific countries.
(7) Trench fever--Mexico.
(8) Q-fever--worldwide, sporadic and outbreak situations.

e. Laboratory diagnosis.

(1) Indirect fluorescent antibody tests.
(2) ELISA and EIA.

f. Prevention and control.

(1) Vaccination.
(2) Avoidance of vector infested areas.
(3) Vector removal.

g. Treatment--Tetracycline and Chloramphenicol.


a. Clinical manifestations.

(1) Sudden onset with high fever (40°C) for 3-5 days.
(2) Severe muscle pain ("break bone fever").
(3) Myalgia and arthralgia.
(4) Rash.
(5) Encephalitis.

b. Mode of transmission--bite of infectious Flaviviridae mosquitoes, principally *Aedes aegypti*.
c. Etiological agent--four dengue viruses (types 1-4) are antigenically closely related and constitute a distinct subgroup within the flavivirus genus (group B arboviruses) of the Flaviviridae family.

d. Incubation period--3 to 14 days, commonly 7-10 days.

e. Epidemiology.

(1) South-East Asia (types 1, 2, 3 and 4)-- southern China and Hainin, Vietnam, Laos, Cambodia, Thailand, Burma, India, Sri Lanka, Indonesia, Philippines, Malaysia and Singapore, New Guinea, Bangladesh, Nepal, and Taiwan.

(2) Pacific (type 2).

(3) West Africa (types 1 and 2).

(4) East Africa (type 2).

(5) Caribbean (types 1 and 4).

(6) Americas (types 2 and 3)--Mexico, Central America, Venezuela, Colombia, Equador, Brazil, Bolivia and Paraguay.

f. Laboratory diagnosis.

(1) Virus isolation from blood during acute febrile stage by inoculation of mosquitoes or cell cultures.

(2) Fourfold or greater increase in antibody titer in infected individuals.

(3) Antigen detection or IgM antibody capture ELISA.

(4) Neutralization test.

(5) Hemagglutination--inhibition test.

(6) Complement--fixation test.

(7) Indirect fluorescent antibody test.

g. Prevention and control.

(1) Education of people on personal measures for destroying breeding sites and protecting against day-biting mosquitoes.

(2) Vector control.

(3) Insecticide spraying.

(4) Experimental vaccine.

h. Treatment.
(1) Treatment is symptomatic only.

(2) Careful management of fluid and electrolyte balance.

5. Korean Hemorrhagic fever (KHF) and Hantavirus Pulmonary Syndrome (HPS)--Epidemic hemorrhagic fever, Hemorrhagic nephrosonephritis (HNN), Hemorrhagic fever with renal syndrome (HFRS).

a. Clinical manifestations.

(1) Abrupt onset of fever (3-8 days).

(2) Hemorrhagic fever with renal syndrome.

(3) Respiratory illness--flu-like symptoms, capillaries ooze fluid into the lungs.

b. Mode of transmission--aerosol transmission from rodent excreta. Virus present in urine, feces and saliva of infected asymptomatic rodents; highest virus concentration found in lungs. Airborne transmission by infected dust particles.

c. Etiological agent--Hantaviruses (a genus of the Bunyaviridae family).

d. Incubation period--usually 12-16 days after a rodent bite.

e. Epidemiology.

(1) KHF--major public health problem in Korea, China and Japan--increased incidence during May-June and October-November.

(2) European former USSR.

(3) Scandinavia, Greece, Yugoslavia, Balkans, Belgium, and France.

(4) HPS--Southwestern U.S.A.

(5) Intimate contact with rodents, such as in agricultural areas with high human and rodent population densities, during military campaigns or exercises (trench warfare and bivouacs in the open), or crowded urban housing.

f. Laboratory diagnosis.

(1) Demonstration of specific antibodies using IFA or ELISA.

(2) Virus isolation and serologic tests for antibody (IgM antibody capture ELISA, neutralization).

g. Prevention and control.

(1) Exclude rodents from houses and other buildings in endemic areas; improve food storage.

(2) Prevention of contact (removal from area, insecticides,
screening, rodent-proofing) between susceptible human or animal and source of virus (infected arthropod or vertebrae).

(3) Experimental vaccine.

h. Treatment.

(1) Treatment is symptomatic only.

(2) Maintain fluid and electrolyte balance.

6. Viral Encephalitis--Venezuelan Equine encephalitis (VEE), Japanese encephalitis (JE), Eastern equine encephalitis (EEE), Western equine encephalitis (WEE), Murray Valley encephalitis (MVE) (Australian encephalitis), St. Louis encephalitis (SLE), Rocío encephalitis, California encephalitis, La Crosse encephalitis (LAC).

a. Clinical manifestation.

(1) Acute inflammatory viral disease, short duration involving brain, spinal cord and meninges.

(2) Stupor, disorientation and coma.

b. Mode of transmission--by the bite of infective mosquitoes; mosquitoes acquire infection from wild birds.

c. Etiological agent--each disease is caused by a specific virus in one of three groups.

(1) EEE and WEE are alphaviruses.

(2) JE, MVE, SLE, and Rocío are flaviviruses.

(3) LAC, California encephalitis, Jamestown Canyon and snowshoe hare viruses in the California group of bunyaviruses.

d. Incubation period--usually 5-15 days.

e. Epidemiology.

(1) Seasonal--summer and early fall.

(2) VEE--South America.

(3) JE--western Pacific Islands (Japan, Philippines, eastern Asia from Korea to Indonesia, China and India, Malaysia.

(4) MVE--northern and western Australia and New Guinea, Kalimantan.

(5) SLE, EEE, WEE--Trinidad, Jamaica, Panama and Brazil, Argentina, Surinam, Uruguay.

(6) Rocío--Brazil.

f. Laboratory diagnosis.
(1) Demonstration of specific IgM in acute phase serum or CSF.

(2) Antibody rises between early and late specimens of serum by neutralization, complement-fixation, Hemagglutination-inhibition, FA, ELISA tests.

g. Prevention and control.
(1) VEE--vaccination of equines.
(2) Insecticides.
(3) Mosquito control measures.

h. Treatment.
(1) Antipyretics and analgesics for fever.
(2) Treat symptomatically.
(3) Maintain fluid and electrolyte balance.

7. Sandfly Fever--a group of arboviral diseases.

a. Clinical manifestations.
(1) Fever of 101 to 103°F for 3 to 4 days.
(2) Headache, malaise, and nausea.
(3) Retrobulbar pain on motion of the eyes.
(4) Pain in the back and limbs.
(5) Prolonged mental depression may precede complete recovery.

b. Mode of transmission--bite of an infected sandfly.
(1) The common sandfly is a small, hairy, blood-sucking midge which bites at night and has a limited flight range.
(2) Sandflies become infective about one week after biting an infected person and remain infective during their lifespan of about one month.

c. Etiological agent--the sandfly fever group of viruses; several related immunologic types.

d. Incubation period--usually 3-4 days, rarely less, and up to 6 days.
e. Epidemiology--those parts of Europe, Africa, and Asia where the sandfly vector lives. Also found in Central and South America where closely related viral agents are present.

1. A disease of tropical and subtropical areas associated with:
   a. Long periods of hot, dry weather in Europe and Africa.
   b. Rainforests in the tropics of the Western Hemisphere.

2. Seasonal between April and October.

3. Prone to appear as a disease of troops and travelers from non-endemic areas.

f. Laboratory diagnosis.

1. Diagnosis confirmed by detection of specific IgM.

2. By titer rise in serologic tests.

3. By isolation of virus from blood in newborn mice or in cell culture.

g. Prevention and control.

1. Prevention of sandfly bites by use of repellents, particularly after sundown.

2. Eradication of sandflies with insecticides.

3. Isolation of infected persons from sandfly vectors with mosquito nets or very fine screening.

h. Specific treatment--none.
<table>
<thead>
<tr>
<th>Agent</th>
<th>Mode of Transmission</th>
<th>Clinical Manifestations</th>
<th>Laboratory Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malaria: <em>Plasmodium</em> spp.--<em>P. vivax, P. ovale, P. malariae, P. falciparum</em></td>
<td>bite of female Anopheles mosquito, blood transfusion, needlestick</td>
<td>moderate fever to paroxysms, intense chills, high fever, sweating, malaise, myalgia</td>
<td>Giemsa or Wright stained blood films, intracellular parasites, immunologic tests</td>
</tr>
<tr>
<td><em>Salmonella typhi</em>--typhoid fever</td>
<td>fecal contaminated food &amp; water</td>
<td>fever, malaise, intestinal hemorrhage</td>
<td>culture ID, serological ID</td>
</tr>
<tr>
<td>Rocky Mountain Spotted Fever--<em>Rickettsia rickettsii</em></td>
<td>bite of infected tick</td>
<td>sudden onset of fever,</td>
<td>indirect FA,</td>
</tr>
<tr>
<td>Boutonneuse fever--<em>Rickettsia conorii</em></td>
<td>bite of infected tick</td>
<td>myalgia, rash,</td>
<td>detection of antibodies,</td>
</tr>
<tr>
<td>Rickettsialpox--<em>Rickettsia akari</em></td>
<td>infected mites</td>
<td>arthralgia</td>
<td>EIA/ELISA</td>
</tr>
<tr>
<td>Epidemic typhus--<em>Rickettsia prowazekii</em></td>
<td>infected body lice</td>
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<td></td>
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<tr>
<td>Murine typhus--<em>Rickettsia typhi</em></td>
<td>infected fleas</td>
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<td></td>
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<tr>
<td>Scrub typhus--<em>Rickettsia tsutsugamushi</em></td>
<td>chiggers</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trench fever--<em>Rochalimaea quintana</em></td>
<td>body lice</td>
<td></td>
<td></td>
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<tr>
<td>Q-fever--<em>Coxiella burnetii</em></td>
<td>aerosols from infected animals</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dengue fever--<em>Flavivirus</em></td>
<td>bite from <em>Aedes</em> mosquito</td>
<td>fever, rash, encephalitis</td>
<td>EIA/ELISA, viral isolation</td>
</tr>
<tr>
<td>Korean Hemorrhagic Fever--<em>Hantavirus</em></td>
<td>aerosols from rodent excreta</td>
<td>fever, renal failure, hemorrhagic fever</td>
<td>EIA/ELISA, IFA, viral isolation in cell culture</td>
</tr>
<tr>
<td>Viral encephalitis: alphavirus, flavivirus, bunyavirus</td>
<td>bite from infected mosquito, virus found in wild birds</td>
<td>acute inflammatory viral disease involving CNS stupor, coma, disorientation</td>
<td>specific IgM in serum, CSF during acute phase CF, HF, Fā, EIA/ELISA</td>
</tr>
<tr>
<td>Sandfly fever virus group</td>
<td>bite from infected sandfly</td>
<td>fever, headache, malaise, nausea, pain behind eyes, pain in back and limbs</td>
<td>specific IgM, virus isolation in cell culture</td>
</tr>
</tbody>
</table>
D. Diseases Presenting as Cutaneous Lesions.

1. Gambian trypanosomiasis or West African sleeping sickness, Rhodesian trypanosomiasis or East African sleeping sickness.
   a. Clinical manifestations.
      (1) Chancre at site of inoculation.
      (2) Lymphadenopathy--cervical lymph nodes.
      (3) Somnolence--lethargy (sleeping sickness).
      (4) Severe neurologic disturbance.
   b. Mode of transmission--infected humans, game and domestic animals.
   c. Etiological agents.
      (1) Trypanosoma brucei.
      (2) Trypanosoma brucei gambiense.
      (3) Trypanosoma brucei rhodesiense.
   d. Incubation period--3 days to 3 weeks.
   e. Epidemiology.
      (1) Trypanosoma b. gambiense--West Africa from Senegal south to Angola and east to Lake Victoria.
      (2) Trypanosoma b. rhodesiense--East Africa from Sudan south to Mozambique and west to Lake Victoria.
      (3) Disease transmitted by male and female flies of the genus Glossina (tsetse flies) or congenitally.
   f. Laboratory diagnosis.
      (1) Demonstration of trypanosomes in blood, lymph node aspirate or cerebrospinal fluid
      (2) Cultivation in an artificial medium.
      (3) Animal inoculation.
      (4) Immuno diagnosis.
   g. Prevention and control.
      (1) Clearing of vegetation that provides habitats for the flies.
(2) Residual insecticide application.

(3) Active surveillance.

(4) Prompt diagnosis and treatment.

(5) Proper wear of uniform and use of other personal protective measures (that is, insecticides).

h. Treatment.

(1) Early stages--Suramin and Pentamidine.

(2) Late stages--Melarsoprol.

2. Leishmaniasis (visceral leishmaniasis or Kala-azar; cutaneous leishmaniasis, mucocutaneous leishmaniasis).

a. Clinical manifestations.

(1) Fever.

(2) Weight loss.

(3) Splenomegaly.

b. Mode of transmission--transmitted to humans by sandflies from a wide variety of reservoir hosts.

c. Etiological agents--\textit{Leishmania} spp.

d. Incubation period--week up to many months.

e. Epidemiology.

(1) Cutaneous--\textit{Leishmania tropica}.

(a) Rural areas of hot, humid regions of developing countries.

(b) Latin America, along Atlantic coast of Central America and South America.

(c) Mediterranean Sea, Middle East, Afghanistan, and Southern Russia.

(2) Mucocutaneous--\textit{Leishmania braziliensis}.

(a) Brazil, south of Amazon.

(b) Columbia.

(c) Central America.

(3) Visceral and bone marrow--\textit{Leishmania donovani}.

(a) Central and southern Brazil.
(b) Kenya.
(c) Eastern India.
(d) Pacific coast of Central America.

f. Laboratory diagnosis--identification of organism in tissue impression smears, exudates, or culture of flagellated forms from infected tissue.

g. Prevention and control--prevent human contact with sandflies.
   (1) Control or elimination of animal reservoirs.
   (2) Insecticide spraying.
   (3) Insect repellant and appropriate clothing.

h. Treatment--Pentavalent antimonial agents.


   a. Clinical manifestations.
      (1) Sudden onset with fever.
      (2) Cutaneous lesions--5 to 20 percent fatality rate, mildest mortality rate; 90 percent of human infections seen with this symptom.
      (3) Respiratory infection--rapid fatality rate, highest mortality rate, death within 24 hours without treatment; 5 percent of human infections seen with mild upper respiratory involvement and productive cough.
      (4) Gastrointestinal infection--rare disease, low mortality rate; 5 percent of human infections seen with this symptom.

   b. Mode of transmission--contaminated soil, water, animal hides, and by-products of infected domestic livestock and wild herbivores.

   c. Etiological agent--Bacillus anthracis: gram-positive sporeforming bacillus. (Spores are quite stable and they are resistant to many environmental stresses, including some disinfectants and decontamination procedure.)

   d. Incubation period--2 to 7 days; most cases occur within 48 hours after exposure.

   e. Epidemiology.
      (1) Worldwide; where alkaline soils are common.
      (2) Transportation of contaminated products of domestic animals (wool, mohair, bone meal) produces unsuspected infections and an unknowingly increased hazard of
contaminating areas and herds of livestock.

(3) Infections by ingestion or inhalation occur more frequently in colder climates.

(4) Cutaneous anthrax--cattle-producing countries (Iran); intestinal anthrax--Africa.

(5) Seasonal--dry season high prevalence.

f. Laboratory diagnosis--culture and identification of gram-positive rod from blood, tissue and fomites.

g. Prevention and control.

(1) Incineration of infected carcasses in situ

(2) Immunization of domestic livestock and persons in endemic areas (Vaccine was used in Southwest Asia during Operation Desert Storm.)

h. Treatment--Penicillin (resistant strains are easily produced/induced)

4. Leptospirosis (Weil's disease in its severe form; canecutter's, swineherd's).

a. Clinical manifestations.

(1) Acute onset of--severe headaches, muscle pain, fever and rash.

(2) Severe cases--meningitis, bleeding renal failure, and hepatitis.

b. Mode of transmission.

(1) Rodents, livestock, and wild animals mammals or marsupials by direct contact with infected urine or tissues.

(2) Indirect contact through contaminated surface waters, mud or earth.

c. Etiological agent--One of the serovars of Leptospira interrogans.

d. Incubation period--usually 10 days, range of 4-19 days.

e. Epidemiology.

(1) Worldwide, usually sporadic, seasonal and geographic.

(2) Mainly domestic livestock and rodents in temperate climates.
(3) Greatest public health importance in Southeast Asia and Latin America, eastern and southern Europe, Australia, and New Zealand.

f. Laboratory diagnosis—growth, isolation of serovar Leptospira from blood, urine or CSF.

g. Prevention and control.
   (1) Immunization of livestock and dogs.
   (2) Protective clothing.
   (3) Education and awareness of public, physicians and veterinarians.
   (4) Chemoprophylaxis.

h. Treatment.
   (1) Antibiotic therapy.
   (2) Supportive measures for renal or other organ failure.
### Diseases Presenting as Cutaneous Lesions

<table>
<thead>
<tr>
<th>Agent</th>
<th>Mode of Transmission</th>
<th>Clinical Manifestations</th>
<th>Laboratory Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trypanosoma brucei gambiense</td>
<td>infected humans,</td>
<td>chancr at site of bite, lymphadenopathy, severe CNS disturbance, lethargy (sleeping sickness)</td>
<td>immunodiagnosis, microscopic exam of blood film, lymph node aspirates, CSF</td>
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<td></td>
<td>game animals,</td>
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<td>Trypanosoma brucei rhodesiense</td>
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<td>fever, weight loss, splenomegaly</td>
<td>staining and exam of tissue smears, tissue culture</td>
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<td>sandflies from</td>
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<td>wide variety of</td>
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<td>reservoir hosts</td>
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<td>Leishmania tropica</td>
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<td>sudden onset with fever, cutaneous, respiratory or gastrointestinal infection</td>
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<td>soil, water,</td>
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<td>and wild animals</td>
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<td>Leishmania braziliensis</td>
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<td>Leptospira interrogans</td>
<td>infected urine or</td>
<td>acute onset of severe headache, fever, rash, meningitis, hepatitis, bleeding renal failure</td>
<td>growth and isolation of Leptospira from blood, urine, or CSF</td>
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<td>tissues of rodents,</td>
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<td>water</td>
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</table>

### B. Other.

1. Plague, the "Black Death" pest or peste.
   a. Clinical manifestations.
      (1) Bubonic plague--Inflammatory enlargement of the lymph nodes.
      (2) Systemic plague--Intense bacteremia with symptoms of profound toxemia.
      (3) Pneumonic plague--Rapidly progressive areas of pulmonary consolidation, with high fever, chills and pain.
other mammals.

c. Etiological agent: *Yersinia pestis*, a gram negative bacillus.

2. Hepatitis A.
   a. Clinical manifestations--Jaundice.
   b. Mode of transmission--fecal-oral.
   c. Etiological agent--Hepatitis A virus (HAV).
   d. Incubation period--15 to 50 days.

3. Hepatitis B virus (Type B hepatitis, serum sickness).
   a. Clinical manifestations.
      (1) May be asymptomatic.
      (2) Onset with anorexia, abdominal discomfort, nausea and vomiting, arthralgia and rash to jaundice.
      (3) Dark urine, pale stools, palpable liver.
   
   b. Mode of transmission.
      (1) Primarily human blood and serum-derived fluids.
      (2) Contaminated needles, syringes; percutaneous (IV, IM, subcutaneous or intradermal) and permucosal exposure to infective body fluids.
   
   c. Etiological agent--hepatitis B virus.
   d. Incubation period--usually 45 to 180 days.
   e. Epidemiology.
      (1) Low prevalence areas--parenteral inoculation of blood or blood products.
      (2) Nonpercutaneous (close contacts including venereal spread) high-prevalence--perinatal transmission from infected mother to neonate.
      (3) Chronic carriers--defined as viremic or antigenic for more than 6 months.
   
   f. Laboratory diagnosis--Immuno diagnostic tests: Primarily anti-HBc or anti-HBs, high-titered anti-HBc IgM, positive tests for HBSAg or HBV DNA [HBS = surface antigen of hepatitis virus; HBc = core antigen of hepatitis virus].
      (1) HBSAg--first marker to be detected.
(2) HBeAg--virus replication marker.
(3) HBV DNA--virus replication marker.
(4) Anti-HBc IgM--diagnostic of recent acute Hepatitis B infection.
(5) Anti-HBc--indicates current or previous HBV infection; is not associated with recovery from or immunity to HBV.

g. Prevention and control.
   (1) General measures.
      (a) Exclude contact with HBV-infected blood and secretions.
      (b) Minimize needle-stick by scrupulous technique.
   (2) Passive prophylaxis--Intramuscular injection of hepatitis B immune globulin within 7 days of exposure.
   (3) Active prophylaxis--Sub-unit vaccine and/or course of 3 intramuscular injections with booster every 5 years.

h. Treatment.
   (1) Antiviral and immunomodulating agents.
   (2) Alpha-interferon.
   (3) ARA-A and acyclovir alone.

   a. Etiological agents *C. trachomatis*.
   b. Clinical manifestations.
      (1) Trachoma--the primary cause of blindness of third world countries.
      (2) Inclusion conjunctivitis (TRIC agent).
      (3) Primary cause of nongonococcal urethritis (the most common sexually transmitted bacterial pathogen).
      (4) Lymphogranuloma venereum (LGV).
   c. Mode of transmission--person to person transfer by direct contact.
d. Incubation period.
(1) Trachoma--unknown, probably greater than 5-12 days.
(2) Genital--5 to 14 days or longer; females may be asymptomatic.
(3) LGV--3 to 30 days.
e. Epidemiology--worldwide; North and Sub-Saharan Africa and Southeast Asia; sexually transmitted infections.
f. Laboratory diagnosis.
(1) Isolation from involved site by tissue culture in McCoy cells.
(2) Antigen detection by EIA or FA.
g. Treatment--Tetracycline, doxycycline, or erythromycin.

5. Rabies--primarily a disease of animals, almost always fatal viral encephalomyelitis.
a. Clinical manifestations.
(1) Spasms of muscles used in swallowing.
(2) Delirium and convulsions.
b. Mode of transmission--bite or scratch of rabid animal.
c. Etiological agent--Rabies virus (Rhabdovirus).
d. Laboratory diagnosis--fluorescent antibody staining of brain tissue.

6. Tuberculosis--a mycobacterial disease that causes death and disability in many parts of the world.
a. Etiologic agent--Mycobacterium tuberculosis.
b. Mode of transmission--Exposure to airborne droplet nuclei containing bacilli from infected person.
c. Disease process.
(1) Primary tuberculosis.
(a) Bacteria are contained within tubercles causing granuloma formation.
(b) Tubercles become necrotic and form masses of cheesy debris (caseous material).
(2) Disseminated miliary tuberculosis (about 5 percent).
(a) Primarily in immuno compromised patients.
(b) Dissemination (kidneys, brain, spleen, and liver).

d. Incubation period--4 to 12 weeks from infection to demonstrable primary lesions or significant tuberculin reaction.

e. Epidemiology--occurs worldwide.

f. Prevention and control.

(1) Public education in mode of spread and methods of control of tuberculosis.

(2) Improve living conditions such as reduce overcrowding.
### Other Microbiological Diseases

<table>
<thead>
<tr>
<th>Agent</th>
<th>Mode of Transmission</th>
<th>Clinical Manifestations</th>
<th>Laboratory Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yersinia pestis—causes plague, the &quot;Black Death&quot;</td>
<td>bites by infected fleas, direct contact, and airborne</td>
<td>bubonic plague, septicemic, pneumonic</td>
<td>culture ID, recovery of agent from blood, bubo, sputum, or tissue</td>
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<tr>
<td>Hepatitis A virus</td>
<td>fecal to oral; contaminated water or food</td>
<td>asymptomatic and subclinical, jaundice</td>
<td>detection of antibodies</td>
</tr>
<tr>
<td>Hepatitis B virus</td>
<td>human blood, contaminated needles</td>
<td>asymptomatic, jaundice, palpable liver</td>
<td>immunodiagnosis -- detection of antigens (HBsAg) and antibodies</td>
</tr>
<tr>
<td>Chlamydia trachomatis</td>
<td>person to person contact</td>
<td>trachoma -- eye disease, STD, LGV, PID, urethritis</td>
<td>cell culture, EIA, FA</td>
</tr>
<tr>
<td>Chlamydia psittaci</td>
<td>exposure to infected birds</td>
<td>pneumonia</td>
<td>cell culture, EIA, FA</td>
</tr>
<tr>
<td>Chlamydia pneumoniae</td>
<td>human to human via respiratory droplets</td>
<td>pneumonia, pharyngitis, bronchitis</td>
<td>cell culture, EIA, FA</td>
</tr>
<tr>
<td>Rabies--Rhabdovirus</td>
<td>bite of infected animal</td>
<td>headache, fever, malaise, delirium, convulsions, respiratory paralysis</td>
<td>FA of brain tissue, virus isolation in cell culture</td>
</tr>
<tr>
<td>Mycobacterium tuberculosis</td>
<td>respiratory droplets</td>
<td>pulmonary disease, fatigue, fever, weight loss, bloody sputum</td>
<td>positive PPD skin test, AFB stain of sputum, culture on special media</td>
</tr>
</tbody>
</table>
g. Laboratory Diagnosis.

(1) Positive tuberculin skin test.

(2) Acid-fast bacilli in stained smears from sputum or other body fluids.

(3) Abnormal chest radiographs indicative of pulmonary infiltration, cavitation, and fibrosis.

(4) Isolation of tubercula bacilli on culture.

F. Biological Warfare Agents and Detection.

NOTE: Do not get involved in a political or ethical discussion, immediately change subject/discussion.

1. The use of biological agents as offensive weapons in warfare has not been confirmed by modern microbiological methods. There is strong circumstantial historical evidence of such use.

   Wiener, Stanley L., Military Medicine, Volume 152. 1:25, pg. 25, January 1987.

2. Purpose.
   a. Antipersonnel--incapacitate.
   b. Antianimal--livestock (affects economy and morale).
   c. Anticrop--affects economy and morale.

3. Advantages.
   a. Widely available.
   b. Easily obtained.
   c. Silent.
   d. Invisible.

   JAMA, Volume 262, No. 5. pg. 677, Medicine in Defense Against Biological Warfare, August 9, 1987.

4. Disadvantages.
   a. Endangers their creators and users.
   b. Negative long-term ecological consequences.


5. Likely Agents. ³ ⁴ ⁵

a. Anthrax--*Bacillus anthracis*.
b. Brucellosis--*Brucella abortis*.
c. Tularemia--*Francisella tularensis*.
d. Plague--*Yersinia pestis*.
e. Botulism toxins--*Clostridium botulinum*.
f. Q fever--*Coxiella burnetii*.
g. Venezuelan Equine Encephalitis.
h. Rift Valley fever.
i. Crimean-Congo hemorrhagic fever.
j. Trichothecene Mycotoxins (from Fusaria mould-- allegedly found in "yellow rain").
k. Cholera--*Vibrio cholerae*.
l. Melioidosis--*Pseudomonas pseudomallei*.
m. *Clostridium perfringens* toxins.

n. Ricin.
o. Smallpox virus.
p. Staphylococcal enterotoxin B.


a. Vaccine production that could be converted to offensive agent production.


b. While the potential also exists for a threat nation to develop "superagents", it is generally assumed that microbiological warfare agents will be the same agents we see in everyday clinical medicine. They may also be "exotic" or tropical agents not usually encountered in the United States, but most likely identifiable.


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c. Genetically engineered microorganisms, that is, genetic code for cobra venom incorporated into influenza virus.

7. Delivery mechanisms.
   a. Aerosol.
   b. Vapor--carried or controlled by wind and weather.
   c. Missiles.
   d. Artillery.
   e. Mines.
   f. Fighter--bombers.
   g. Cruise missiles.

8. Detection.
   a. Extremely difficult.
   b. Prior air samplers (mx-2) and bioagent detectors (mx-19) are not effective and it is unlikely that prior warning of a bioweapon attack will be achieved before the first group of patients with sublethal or lethal exposures are received.

Military Medicine, Volume 152. 1:25, pg. 25, Strategies of Biowarfare Defense.

9. Protection.
   a. Light-weight protective clothing.
   b. Masks with HEPA filters.
   c. Use of broad spectrum antibacterial, antiviral drugs or agents that enhance the immune system.
   d. Immunizations.

10. Defense--Three basic questions that require answers in order to implement some form of defensive response.
    a. How do we know the outbreak of disease is a biowarfare attack and not a natural epidemic?
       (1) Large number of ill and dying, especially if clinical manifestations resemble a threat agent.
       (2) Spectrum of disease skewed toward severe cases.

---

(3) Unusual or impossible agents for a given geographic region (requires clinical and laboratory diagnosis of specific agents).

(4) Multiple, simultaneous outbreaks with endemic agents or new agents not usually found in the attack region.

(5) Epidemic caused by multi-resistant pathogen not previously isolated from clinical cases.

(6) Dead animals of many types.

(7) Identification of delivery vehicles.

(8) Claims by aggressors.

(9) Prior intelligence.

b. What is the agent or agents being used by hostile forces? (Requires clinical and/or laboratory diagnosis.)

c. What countermeasures can be taken?

(1) Immunizations and chemoprophylaxis.

(2) Medical intelligence--warning of agent or impending attack.

(3) Protective equipment--gloves, boots, suit, respirator, and hardened shelter.

(4) Decontamination.

G. Reference for current situations: See the Disease and Environmental Alert Report (DEARS) from Armed Forces Medical Intelligence Center (AFMIC) at Fort Detrick, MD.

H. Detection of Biological Warfare Agents and Emerging Technology.

1. Biological Integrated Detection System (BIDS)--Chemical School.

   a. Vehicle mounted with self-contained power unit and long-range communications.

   b. Aerosol sampling.

   c. Bioluminometer (uses ATP to detect biological material).

   d. Flow cytometer--detect and classify bacterial cells vs. bacterial spores.

   e. Threshold system (EIA ?)--identify specific agents. (anthrax, staphylococcal enterotoxin B [SEB], botulism toxin, plague)

   f. Smart Ticket--single agent detection. (membrane-based antigen capture assay)

2. Initiatives of Biological Defense Research Program.
a. Dipstick--membrane linked enzyme immunoassays.

b. Fiber optic biosensor--sandwich. fluoroimmunoassay on optical fiber

c. Flow-Through Assay--membrane based antigen capture using colloidal gold or dyed latex.

d. Chromatographic assay--membrane migration assay using colloidal gold or dyed latex.

e. Rapid Flow-through Hand-held device--membrane based antigen capture using colloidal gold.

For additional and detailed information regarding the diseases presented, refer to the chart below.


<table>
<thead>
<tr>
<th>DISEASE</th>
<th>REF 1 page</th>
<th>REF 2 page</th>
<th>REF 3 page</th>
<th>REF 4 page</th>
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<tbody>
<tr>
<td>Campylobacteriosis</td>
<td>381</td>
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<td>Cholera</td>
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<td>Infectious Diseases Associated with Escherichia coli</td>
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