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<td>AAHL</td>
<td>Australian Animal Health Laboratory</td>
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<tr>
<td>ABCs</td>
<td>Active Bacterial Core Surveillance</td>
</tr>
<tr>
<td>ACRO</td>
<td>Australian Commonwealth Research Organisation</td>
</tr>
<tr>
<td>ADB</td>
<td>African Development Bank</td>
</tr>
<tr>
<td>AHEAD-Iliad</td>
<td>Animal Health Emerging Animal Diseases/ International lookouts for Infectious Animal Diseases</td>
</tr>
<tr>
<td>AIDS</td>
<td>Acquired Immunodeficiency Syndrome</td>
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<td>ANN</td>
<td>A Network</td>
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<tr>
<td>ANOVA</td>
<td>Analysis of Variance</td>
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<td>APHIS</td>
<td>The Animal and Plant Health Inspection Service</td>
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<tr>
<td>CCDR</td>
<td>Canadian Communicable Disease Report</td>
</tr>
<tr>
<td>CDC</td>
<td>Centers for Disease Control and Prevention</td>
</tr>
<tr>
<td>CFR</td>
<td>Case Fatality Rate</td>
</tr>
<tr>
<td>CIDPC</td>
<td>Centre for Infectious Disease Prevention and Control</td>
</tr>
<tr>
<td>CIDPC</td>
<td>The Centre for Infectious Disease, Prevention and Control</td>
</tr>
<tr>
<td>CIPHS</td>
<td>Canadian Integrated Public Health Surveillance</td>
</tr>
<tr>
<td>CIRMF</td>
<td>The Centre International de Recherchers Medicales de Franceville</td>
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<tr>
<td>CMAJ</td>
<td>Journal of the Canadian Medical Association</td>
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<tr>
<td>CPZ</td>
<td>Chimpanzees</td>
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<td>CSIRO</td>
<td>Australian Commonwealth Scientific and Industrial Research Organisation</td>
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<tr>
<td>Acronym</td>
<td>Description</td>
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<tr>
<td>CSR</td>
<td>Communicable Disease Surveillance and Response</td>
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<td>CSTE</td>
<td>The Council of State and Territorial Epidemiologist</td>
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<tr>
<td>DF</td>
<td>Dengue Fever</td>
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<tr>
<td>DHAP</td>
<td>Division of HIV/AIDS Prevention</td>
</tr>
<tr>
<td>DHF</td>
<td>Dengue Haemorrhagic Fever</td>
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<tr>
<td>DHHS</td>
<td>The Department of Health and Human Services</td>
</tr>
<tr>
<td>DOD</td>
<td>Department of Defense</td>
</tr>
<tr>
<td>DRC</td>
<td>Democratic Republic of the Congo</td>
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<tr>
<td>DVBID</td>
<td>Division of Vector Borne Infectious Diseases</td>
</tr>
<tr>
<td>DVD</td>
<td>Digital Video Display</td>
</tr>
<tr>
<td>EC</td>
<td>European Commission</td>
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<tr>
<td>EHF</td>
<td>Ebola Haemorrhagic Fever</td>
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<tr>
<td>EID</td>
<td>Emerging Infectious Disease</td>
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<tr>
<td>ESA</td>
<td>The European Space Administration</td>
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<tr>
<td>EU</td>
<td>European Union</td>
</tr>
<tr>
<td>FAS</td>
<td>Federation of American Scientists</td>
</tr>
<tr>
<td>GBS</td>
<td>Group B Streptococcus</td>
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<tr>
<td>GEIS</td>
<td>The US Global Emerging Infectious Surveillance &amp; Response System</td>
</tr>
<tr>
<td>GOARN</td>
<td>The Global Outbreak Alert and Response Network</td>
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<tr>
<td>GPHIN</td>
<td>The Global Public Health Intelligence Network</td>
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<tr>
<td>HIV</td>
<td>Human Immunodeficiency Virus</td>
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<tr>
<td>HIV-1</td>
<td>Human Immunodeficiency Virus Type 1</td>
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<td>Abbreviation</td>
<td>Description</td>
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<tr>
<td>IDPA</td>
<td>The Infectious Disease Pathology Activity</td>
</tr>
<tr>
<td>IDSR</td>
<td>Integrated Disease Surveillance and Response</td>
</tr>
<tr>
<td>IFRC</td>
<td>International Federation of Red Cross and Red Crescent Societies</td>
</tr>
<tr>
<td>IOM</td>
<td>Institute of Medicine</td>
</tr>
<tr>
<td>IRIN</td>
<td>Integrated Regional Information Network</td>
</tr>
<tr>
<td>MMWR</td>
<td>Mortality and Morbidity Weekly Report</td>
</tr>
<tr>
<td>MOU</td>
<td>Memorandum of Understanding</td>
</tr>
<tr>
<td>MPV</td>
<td>Monkeypox Virus</td>
</tr>
<tr>
<td>NAHEMS</td>
<td>The National Animal Health Emergency Management System</td>
</tr>
<tr>
<td>NAT</td>
<td>Nucleic Acid Amplification Tests</td>
</tr>
<tr>
<td>NCHSTP</td>
<td>National Centre for HIV, STD and TB Prevention</td>
</tr>
<tr>
<td>NCID</td>
<td>National Centre for Infectious Disease</td>
</tr>
<tr>
<td>NETSS</td>
<td>National Electronic Telecommunications System for Surveillance</td>
</tr>
<tr>
<td>NGO</td>
<td>Non-Governmental Organisation</td>
</tr>
<tr>
<td>NIH</td>
<td>National Institutes of Health</td>
</tr>
<tr>
<td>NNDSS</td>
<td>National Notifiable Disease Surveillance Systems</td>
</tr>
<tr>
<td>PAHO</td>
<td>Pan American Health Organisation</td>
</tr>
<tr>
<td>PANAFTOSA</td>
<td>The Pan American Foot and Mouth Disease Centre</td>
</tr>
<tr>
<td>PANIFPZ</td>
<td>The Pan American Institute for Food Protection and Zoonoses</td>
</tr>
<tr>
<td>PHLS</td>
<td>Public Health Laboratory Service</td>
</tr>
<tr>
<td>PPHB</td>
<td>Population and Public Health Branch</td>
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<tr>
<td>Abbreviation</td>
<td>Full Form</td>
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<tr>
<td>SARS</td>
<td>Severe Acute Respiratory Syndrome</td>
</tr>
<tr>
<td>SFV</td>
<td>Simian Foamy Virus</td>
</tr>
<tr>
<td>SIV</td>
<td>Simian Immunodeficiency Virus</td>
</tr>
<tr>
<td>STI</td>
<td>Sexually Transmitted Infections</td>
</tr>
<tr>
<td>SWOT</td>
<td>Strength, weakness, opportunity and threat (analysis)</td>
</tr>
<tr>
<td>UK</td>
<td>United Kingdom</td>
</tr>
<tr>
<td>UNAIDS</td>
<td>United Nations AIDS</td>
</tr>
<tr>
<td>USA</td>
<td>United States of America</td>
</tr>
<tr>
<td>VHS</td>
<td>Video Home System</td>
</tr>
<tr>
<td>WER</td>
<td>Weekly Epidemiological Review</td>
</tr>
<tr>
<td>WHO</td>
<td>World Health Organization</td>
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<td>WNV</td>
<td>West Nile Virus</td>
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<td>Dengue Fever (Dengue Haemorrhagic Fever)</td>
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ANNEXURE A

III. A child aged less than 18 months born to an HIV-infected mother will be categorized for surveillance purposes as “not infected with HIV” if the child does not meet the criteria for HIV infection but meets the following criteria:

Laboratory Criteria

Definitive

- At least two negative HIV antibody tests from separate specimens obtained at greater than or equal to 6 months of age

or

- At least two negative HIV virologic tests* from separate specimens, both of which were performed at greater than or equal to 1 months of age and one of which was performed at greater than or equal to 4 months of age

AND

No other laboratory or clinical evidence of HIV infection (i.e., has not had any positive virologic tests, if performed, and has not had an AIDS-defining condition).

Presumptive

A child who does not meet the above criteria for definitive “not infected” status but who has:

- One negative EIA HIV antibody test performed at greater than or equal to 6 months of age and NO positive HIV virologic tests, if performed

or

- One negative HIV virologic test* performed at greater than or equal to 4 months of age and NO positive HIV virologic tests, if performed

- One positive HIV virologic test with at least two subsequent negative virologic tests, at least one of which is at greater than
or equal to 4 months of age; or negative HIV antibody test results, at least one of which is at greater than or equal to 6 months of age.

AND

No other laboratory or clinical evidence of HIV infection (i.e., has not had any positive virologic tests, if performed, and has not had an AIDS-defining condition).

OR

Clinical or Other Criteria (if the above definitive or presumptive laboratory criteria are not met)

- Determined by a physician to be “not infected”, and a physician has noted the results of the preceding HIV diagnostic tests in the medical record

AND

No other laboratory or clinical evidence of HIV infection (i.e., has not had any positive virologic tests, if performed, and has not had an AIDS-defining condition)

IV. A child aged less than 18 months born to an HIV-infected mother will be categorized as having perinatal exposure to HIV infection if the child does not meet the criteria for HIV infection (II) or the criteria for “not infected with HIV” (III) (CDC-MMWR 1999)

(Appendix 1999:29-31)
ANNEXURE B

Wisconsin Division of Public Health
Case Definition for Monkeypox 6/17/03

(Human Monkeypox Virus Infection)

Clinical Description

An acute viral infection caused by monkeypox virus, a member of the genus Orthopoxvirus. The clinical illness includes a rash illness characterized by pleiomorphic lesions progressing through papular, vesicular, pustular, umbilicated and crusted stages that appears after a prodrome that includes fever, lymphadenopathy, chills, sweats, headache and cough.

Laboratory criteria for diagnosis

- Isolation in virus culture medium of monkeypox virus from a clinical specimen
- Demonstration of monkeypox virus DNA by polymerase chain reaction testing in a clinical specimen
- Demonstration of virus morphologically consistent with an orthopoxvirus by electron microscopy
- Demonstration of presence of orthopox virus in tissue using immunohistochemical testing methods

Epidemiologic criteria (relevant to current outbreak)

A Wisconsin resident who has one or more of the following contacts within 21 days prior to onset of illness:

- Contact* with a prairie dog or a Gambian giant rat originally obtained on or after April 1, 2003 from SK Exotics or Phil’s Pocket Pets, OR
- Contact* with an animal housed (i.e. in the same pet shop or household or veterinary clinic) with a prairie dog or a Gambian giant rat originally obtained on or after April 1, 2003 from SK Exotics or Phil’s Pocket Pets, OR
- Contact* with an animal that had direct contact with a suspect, probable, or confirmed human case patient, OR
- Skin-to-skin contact, or face-to-face (i.e. within 3 feet for 1 hour) contact with a suspect, probable, or confirmed human case patient, OR

- Direct contact with laboratory specimens from a suspect, probable, or confirmed human case patient

* Contact with an animal = petting, rubbing an animal’s fur, being bitten by or scratched by an animal, living in the same household, or being present in a room during a coughing or sneezing spell of the animal

Case Classification

Suspect: an individual with

- a contact that meets the epidemiologic criteria **AND**
- a clinical illness with fever (>99.3 degrees if rash also present, > 100.4 degrees if no rash present) or unexplained rash **AND** two or more of the following signs and symptoms:
  -- lymphadenopathy
  -- cough
  -- chills
  -- sweats
  -- headache
  -- sore throat

Probable: an individual with

- a contact that meets the epidemiologic criteria **AND**
- a clinical illness with rash having papular, vesicular, or pustular skin lesions **AND** at least three of the following signs and symptoms:
  -- fever (>99.3 degrees)
  -- lymphadenopathy
  -- cough
  -- chills
  -- sweats
  -- headache
  -- sore throat

Confirmed: an individual with one or more of the following:

- isolation in virus culture medium of monkeypox virus from a clinical specimen, **OR**
- demonstration of monkeypox virus DNA by polymerase chain reaction testing in a clinical specimen,
OR

- a clinically compatible illness \textbf{AND}
- a contact that meets the epidemiologic criteria \textbf{AND}
- one or more of the following:
  - demonstration of virus morphologically consistent with an orthopoxvirus by electron microscopy in the absence of exposure to another pox virus, \textbf{OR}
  - demonstration of presence of orthopox virus in tissue using immunohistochemical testing methods in the absence of exposure to another pox virus

\textbf{NOTE: Individuals may also be classified as “Lab Only” if they:}

- Have compatible symptoms, but lack epidemiologic criteria, \textbf{OR}
- Have appropriate epidemiologic criteria, but have an illness that does not quite fit the suspect or probable case definition

For individuals designated as “Lab Only,” all appropriate specimens should be gathered the same as if the patient were classified as a case. These individuals should also be subject to the same isolation procedures as cases while their lab results are pending UNLESS their clinical illness picture only includes 2 or fewer of the following symptoms:

- rash
- fever (>99.3 degrees if rash also present, >100.4 degrees if no rash present)
- lymphadenopathy
- cough
- chills
- sweats
- headache
- sore throat

Please consult the Division of Public Health at 1-608-267-9003 about all “Lab Only” individuals

\textbf{Exclusion Criteria}

A case may be excluded as a suspect or probable monkeypox case if:

- An alternative diagnosis can fully explain the illness** \textbf{OR}
- The case was reported on the basis of contact with an ill exotic mammalian pet that was subsequently excluded as a
case of monkeypox (e.g., another etiology fully explains the illness) provided other possible epidemiologic exposure criteria are not present OR

- The case was reported on the basis of contact with an exotic mammalian pet with or without signs of illness that had been in contact with an ill animal or human case that was subsequently excluded as a case of monkeypox (e.g., another etiology fully explains the illness) provided other possible epidemiologic exposure criteria are not present OR
- The case was reported on the basis of contact with a human case that was subsequently excluded as a case of monkeypox (e.g., another etiology fully explains the illness) provided other possible epidemiologic exposure criteria are not present OR
- Paired (acute and convalescent) serum specimens show no rise in antibody titer
- A single convalescent sera obtained more than 5 weeks (35 days) from onset of illness that is negative for IgM and IgC
- A suspect case without a rash does not develop a rash within 10 days of initial identification or examination of the case

** Factors that may be considered in assigning alternate diagnoses include the strength of the epidemiologic exposure criteria for monkeypox, the specificity of the diagnostic test, and the compatibility of the clinical presentation and course of illness for the alternative diagnosis

(Wisconsin 2003:1)
ANNEXURE C

Revised Council of State and Territorial Epidemiologists surveillance case definition for severe acute respiratory syndrome (SARS), December 2003

Clinical Criteria

*Early illness*
- Presence of two or more of the following features: fever (might be subjective), chills, rigors, myalgia, headache, diarrhea, sore throat, or rhinorrhea

*Mild-to-moderate respiratory illness*
- Temperature of >100.4 degrees F (>38 degrees C) and
- One or more clinical findings of lower respiratory illness (e.g., cough, shortness of breath, or difficulty breathing)

*Severe respiratory illness*
- Meets clinical criteria of mild-to-moderate respiratory illness and
- One or more of the following findings:
  -- Radiographic evidence of pneumonia, or
  -- Acute respiratory distress syndrome, or
  -- Autopsy findings consistent with pneumonia or acute respiratory distress syndrome without an identifiable cause

Epidemiologic Criteria

*Possible exposure to SARS associated coronavirus (SARS-CoV)*
One or more of the following exposures in the 10 days before onset of symptoms:
- Travel to a foreign or domestic location with documented or suspected recent transmission of SARS-CoV, or
- Close contact with a person with mild-to-moderate or severe respiratory illness and history of travel in the 10 days before onset of symptoms to a foreign or domestic location with documented or suspected recent transmission of SARS-CoV

*Likely exposure to SARS-CoV*
One or more of the following exposures in the 10 days before onset of symptoms
- Close contact with a person with confirmed SARS-CoV disease or
- Close contact with a person with mild-to-moderate or severe respiratory illness for whom a chain of transmission can be linked to a confirmed case of SARS-CoV disease in the 10 days before onset of symptoms

Laboratory Criteria
Tests to detect SARS-CoV are being refined and their performance characteristics assessed; therefore, criteria for laboratory diagnosis of SARS-
CoV are changing. The following are general criteria for laboratory confirmation of SARS-CoV:

- Detection of serum antibody to SARS-CoV by a test validated by CDC (e.g., enzyme immunoassay), or
- Isolation in cell culture of SARS-CoV from a clinical specimen, or
- Detection of SARS-CoV RNA by a reverse transcription polymerase chain reaction test validated by CDC and with subsequent confirmation in a reference laboratory (e.g., CDC).

Information about the current criteria for laboratory diagnosis of SARS-CoV is available at http://www.cdc.gov/ncidoc/sars/labdiagnostics.htm

Exclusion Criteria
A case may be excluded as a SARS report under investigation (SARS RUI), including as a CDC-defined probable SARS-CoV case, if any of the following apply:

- An alternative diagnosis can explain the illness fully**, or
- Antibody to SARS-CoV is undetectable in a serum specimen obtained >28 days after onset of illness**, or
- The case was reported on the basis of contact with a person who was excluded subsequently as a case of SARS-CoV disease; then the reported case also is excluded, provided other epidemiologic or laboratory criteria are not present

Case Classification
SARS RUI

Reports in persons from areas where SARS is not known to be active

- SARS RUI-1: Case compatible with SARS in groups likely to be first affected by SARS-CoV if SARS-CoV is introduced from a person without clear epidemiologic links to known cases of SARS-CoV disease or place with known ongoing transmission of SARS-CoV

(CDC MMWR 2003:1202-1206)
ANNEXURE D

Ebola Haemorrhagic Fever

Clinical Description

Begins with acute fever, diarrhea that can be bloody, and vomiting, headache, nausea, and abdominal pain are common. Haemorrhagic manifestations may follow. Some patients may also show a ncidopapular rash on the trunk.

Laboratory Criteria for diagnosis:
One of the following:
- Positive virus isolation
- Positive skin biopsy (immunohistochemistry)
- Detection of Ebola virus nucleic acid
- Positive serology which may appear late in the course of the disease

(NDSC 2003:80)
ANNEXURE E

NIPAH VIRUS

Clinical picture in humans
- Mild to severe clinical signs
- Fever and headache of varying severity
- A few patients present drowsiness and disorientation, later falling into a coma and requiring artificial respiration
- A majority of the patients in a coma subsequently die
- The full course of the disease is still unknown
- The incubation period is postulated to be from one to three weeks

Some patients have shown serological reactivity without clinical signs.

Serological tests

Two laboratories, namely the Veterinary Research Institute (VRI), Ipoh, and the Task Force Laboratory at the Department of Medical Microbiology, University of Malaya, were selected to carry out serological tests on the veterinary and human sera respectively. Samples were tested using the IgC and IgM capture ELISA at the above laboratories and virus neutralisation tests at AAHL, Geelong (Australia).

(Emergency Report 1999:1)
ANNEXURE F

Dengue Fever (Dengue Haemorrhagic fever)

1996 Case Definition

Clinical Description

An acute febrile illness characterized by frontal headache, retroocular pain, muscle and joint pain, and rash. The principal vector is the Aedes aegypti mosquito and transmission usually occurs in tropical or subtropical areas. Severe manifestations (e.g., dengue haemorrhagic fever and dengue shock syndrome) are rare but may be fatal.

Laboratory criteria for diagnosis

- Isolation of dengue virus from serum and/or autopsy tissue samples, or
- Demonstration of a fourfold or greater rise or fall in reciprocal immunoglobulin G (IgG) or immunoglobulin M (IgM) antibody titers to one or more dengue virus antigens in paired serum samples, or
- Demonstration of dengue virus antigen in autopsy tissue or serum samples by immunohistochemistry or by viral nucleic acid detection

Case classification

Probable: a clinically compatible case with supportive serologic findings (a reciprocal IgG antibody titer of greater than or equal to 1280 or a positive IgM antibody test on a single acute (late- or convalescent-phase serum specimen to one or more dengue virus antigens)

Confirmed: a clinically compatible case that is laboratory confirmed

Comment

Dengue haemorrhagic fever is defined as an acute febrile illness with minor or major bleeding phenomena, thrombocytopenia (less than or equal to 100,00/mm³), and evidence of plasma leakage documented by hemoconcentration (hematocrit increased by greater than or equal to 20%) or other objective evidence of increased capillary permeability. The definition of dengue shock syndrome follows all of the above criteria for dengue haemorrhagic fever and also includes hypotension or narrow pulse pressure (less than or equal to 20 mm Hg)

(CDC DPHSI 1996)
ANNEXURE G

Encephalitis or Meningitis, Arboviral (includes California Serogroup, Eastern equine, St. Louis, Western equine, West Nile, Powassan)

2001 Case Definition

Clinical Description

Arboviral infections may be asymptomatic or may result in illness of variable severity sometimes associated with central nervous systems (CNS) involvement. When the CNS is affected, clinical syndromes ranging from febrile headache to aseptic meningitis to encephalitis may occur, and these are usually indistinguishable from similar syndromes caused by other viruses. Arboviral meningitis is characterized by fever, headache, stiff neck, and pleocytosis. Arboviral encephalitis is characterized by fever, headache, and altered mental status ranging from confusion to coma with or without additional signs of brain dysfunction (e.g., paresis or paralysis, cranial nerve palsies, sensory deficits, abnormal reflexes, generalized convulsions, and abnormal movements).

Laboratory criteria for diagnosis

- Fourfold or greater change in virus-specific serum antibody titer, or
- Isolation of virus from or demonstration of specific viral antigen or genomic sequences in tissue, blood, cerebrospinal fluid (CSF), or other body fluid, or
- Virus-specific immunoglobulin M (IgM) antibodies demonstrated in CSF by antibody-capture enzyme immunoassay (EIA), or
- Virus-specific IgM antibodies demonstrated in serum by antibody-capture EIA and confirmed by demonstration of virus-specific serum immunoglobulin G (IgG) antibodies in the same or a later specimen by another serologic assay (e.g., neutralization or hemagglutination inhibition)

Case classification

*Probable*: an encephalitis or meningitis case occurring during a period when arboviral transmission is likely, and with the following supportive serology: 1) a single or stable (less than or equal to twofold change) but elevated titer of virus-specific serum antibodies; or 2) serum IgM antibodies detected by antibody-capture EIA but with no available results of a confirmatory test for virus-specific serum IgG antibodies in the same or a later specimen.

*Confirmed*: an encephalitis or meningitis case that is laboratory confirmed
Comment

Because closely related arboviruses exhibit serologic cross-reactivity, positive results of serologic tests using antigens from a single arbovirus can be misleading. In some circumstances (e.g., in areas where two or more closely related arboviruses occur, or in imported arbovirus disease cases), it may be epidemiologically important to attempt to pinpoint the infecting virus by conducting cross-neutralization tests using an appropriate battery of closely related viruses. This is essential, for example, in determining that antibodies detected against St. Louis encephalitis virus are not the result of an infection with West Nile (or dengue) virus, or vice versa, in areas where both of these viruses occur.

The seasonality of arboviral transmission is variable and depends on the geographic location of exposure, the specific cycles of viral transmission, and local climatic conditions. Reporting should be etiology-specific (see below; the six encephalitis/meningitides printed in bold are nationally reportable to CDC):

St. Louis encephalitis/meningitis

West Nile encephalitis/meningitis

Powassan encephalitis/meningitis

Eastern equine encephalitis/meningitis

Western equine encephalitis/meningitis

California serogroup viral encephalitis/meningitis (includes infections with the following viruses: La Crosse, Jamestown Canyon, snowshoe hare, trivittatus, Keystone, and California encephalitis viruses)

Other viral CNS infections transmitted by mosquitos, ticks or midges (e.g., Venezuelan equine encephalitis/meningitis and Cache Valley encephalitis/meningitis)

(Encephalitis 2001:1)
ANNEXURE H

ABCs Case Definition

Active Bacterial Core Surveillance

GBS Group B Streptococcus disease
- Isolation of group B Streptococcus from a normally sterile site
- Early onset cases occur <7 days of age and late onset cases occur between 7 and 90 days of age.

(CDC/ABCs 1997:1)
ANNEXURE I

The Scientist :: Could the Black Death protect against HIV?

**Could the Black Death protect against HIV?**

People who survived the Black Death could have passed on a mutation that prevents the human immunodeficiency virus entering cells. I By David Nicholson

LONDON Several teams of scientists around the world have, for some time, been studying the possibility that a genetic mutation perpetuated by the organism responsible for bubonic plague, or the Black Death, in the Middle Ages - Yersinia pestis - might give people now carrying the mutation increased resistance to the Human Immunodeficiency Virus (HIV) compared to non-carriers. New research has thrown doubt on the micro-organism that was thought to have caused the Black Death, but the link to HIV resistance seems to remain.

Sue Scott and Chris Duncan from the University of Liverpool have suggested that the bacterium Y. pestis - held to be the causative organism for bubonic plague since the 19th century - may not have been responsible for the epidemic after all. In their book, 'Biology of Plagues' (Cambridge University Press, 2001) they proposed that the culprit was most likely a filovirus, similar to the Ebola virus. This theory is based on evidence that emerged after sifting through old parish records of the many towns affected by the plague and then tracking how the disease spread throughout Britain and Europe.

So how does this link to increased resistance to HIV? In a study published in the American Journal of Human Genetics (Am) Hum Genet 1998, 62: 1507-1515) Stephen O'Brien and colleagues at the US National Cancer Institute, used coalescence theory to interpret modern haplotype genealogy. They found that a genetic mutation that gives its carriers protection against the HIV virus became relatively common among white Europeans about 700 years ago - the same period that the Black Death swept into Europe. The team also concluded that the geographic cline of the mutation frequencies and its recent emergence were consistent with a strongly selective historic event (such as an epidemic of a pathogen), driving its frequency upwards in populations whose ancestors survived the Black Death.

The mutation occurs on the gene for CCR-S, a receptor on the surface of macrophages. When a person becomes infected with HIV, the virus latches onto CCRS and another protein - CO-4 - to be transported inside the macrophages.
The Scientist: Could the Black Death protect against HIV?

CCR-5 is disabled in people with the full mutation, and so HIV is unable to gain access to the macrophages. If an individual inherits the mutant gene from both parents, they are essentially immune to HIV infection. People with one mutant and one normal gene can be infected, but tend to survive longer than infected people with two normal CCR-5 genes. It seems as though people without the mutation, called CCR5–32, were killed by the Black Death, so that those with the mutation survived to reproduce and increase its prevalence today.

In 2000, another team of scientists, from Copenhagen's Hvidovre Hospital, investigated why many Europeans appeared to be resistant to HIV. Jesper Eugen-Olsen teamed up with archaeologist and carbon-dating specialist Kaare Lund Rasmussen from the Danish National Museum and analyzed genetic material from ancient bone tissue to try to solve the mystery.

"It always puzzled scientists in the field that the mutation never occurs in Asian or African populations, but only among European Caucasians," said Eugen-Olsen. It is much more prevalent in the North and tapers off towards the Mediterranean, meaning that only eight out of 100 Southern Italians carry the mutation, compared to one in four Danes.

The Danish group rejected the idea that the mutation became more prevalent as a result of the Black Death because the epidemic began in Sicily (in the South) and spread north to Scandinavia. This direction of travel would have predicted that the prevalence of the mutation would have become higher in the South than in the North, which is the reverse of what actually happened.

Assuming that the mutation arose in Scandinavia, Eugen-Olsen's team concentrated on determining the time of the major spread of the mutation by examining bones found in Denmark, dating from the last Ice Age, around 8000 BC to 1950 BC. In particular, they focused on the time between 1800 and 2600 BC, a Mesolithic period of massive change and migration.

Their findings suggested that the CCR-5-32 mutation was already highly prevalent in Denmark before the Black Death. Rasmussen reported: "There is support in the fact that the distribution of the Single Grave Culture in Northern and Middle Europe matches that of the high prevalence of 32j. This meant that an epidemic decimating the Stone Age population could explain the archaeological observations as well as the distribution of the 32j mutation.

They proposed that people with the genetic mutation were then more likely to survive the Black Death, passing on the mutation to current generations and conferring resistance to HIV.

Page 2 of 3

6/21/2004
The Scientist:: Could the Black Death protect against HIV?

Page 3 of 3

Although the potential cause of the Black Death might have changed, researchers in the field still suspect that exposure to it may have passed on resistance to HIV. Since the CCRS mutation provides protection against the entry of a virus, there's good reason to believe that what caused the Black Death was also viral, and targeted the same cells as HIV," concluded Scott.

links for this article University of Liverpool
http://www.liv.ac.uk/

http://www.journals.uchicago.edu/ AJHG/journals/issues/v62n6/970785/970785.abstract. html

US National Cancer Institute http://www.nci.nih.gov/

Hvidovre Hospital
http://www.hvidovre.hosp.dk/

Danish National Museum http://www.nationalmuseet.dk/

6/21/2004

(Nicholson 2001:1-3)
New Theories Link Black Death to Ebola-Like Virus

BY MARK DERR

Between 1347 and 1353, a mysterious disease ravaged Europe, killing an estimated 25 million people — 30 percent to 50 percent of the population.

At the time, people said the disease was caused by a peculiar conjunction of “divine will” and “natural causes,” but the disease itself was not recognized until centuries later. The Black Death lasted no more than six years, according to medical historians, the disease behind it. It spread periodically in different parts of Europe for the next three centuries, leaving millions more dead in its wake.

Then it largely vanished from the continent, but questions over its origins remained.

In 1984, two scientists, Dr. Alexander Yersin and Dr. Shibasaburo Kitasato, independently identified the rod-shaped bacterium responsible for an epidemic of bubonic plague sweeping out of China.

Dr. Yersin soon linked the Black Death to the bacterium, named Yersinia pestis, which was probably spread by fleas from rats. In the past century, many historians and scientists have strengthened the argument that bubonic plague was responsible for the majority of the Black Death and similar outbreaks in medieval Europe and other parts of the world. But other experts have expressed doubts about that and periodically suggested that other diseases were responsible.

The debate stems largely from the difficulty of identifying a disease based on the few medieval descriptions of the Black Death that have survived.

Now two researchers from the University of Grenoble are presenting a new theory. In “Biology of Plagues: Evidence from Historic Populations,” published in March, the authors argue that a hemorrhagic fever, like Ebola, probably caused the Black Death and most of the smaller epidemics that struck Europe for the next three centuries, not bubonic plague.

The authors, Dr. Susan Scott and Dr. Christopher J. Duncan, a zoologist, say their theory answers many lingering questions about the rapid spread and unknown cause of the Black Death. Their argument is based in part on reports that the disease was transmitted from person to person.

They also observe that bubonic plague is a disease of rodents but that Europe had no rodents species that could harbor the disease between outbreaks.

The rats that passed the plague through Europe to humans during epidemics did die, so the plague would have perished with them, Dr. Scott and Dr. Duncan say.

Dr. Samuel K. Cohn, a professor of medicine at the University of Pennsylvania, maintains that many other diseases are better candidates for the Black Death and its subsequent outbreaks through the early modern period than Yersinia pestis.

But Dr. Cohn, the author of “The Black Death Transformed: Disease and Culture in Renaissance Europe,” due out next year, added that he did not accept Dr. Scott and Dr. Duncan’s suggestion that a tropical Ebola-like virus had caused those epidemics. Such a virus would burn out too quickly to produce the widespread mortality of the Black Death, he said, proposing instead that the Black Death was caused by a highly contagious virus or bacterium that might not exist now.

Most epidemiologists argue that the evidence points most strongly toward the bubonic plague pathogen as the cause of the Black Death.

Bubonic plague — often called just the plague — can take three forms in people, and these forms can account for the descriptions of Black Death symptoms, said Dr. David T. Dennis, coordinator of the plague program for the Centers for Disease Control and Prevention in Fort Collins, Colo.

The disease is still a public health problem, with a few thousand cases worldwide, but it can now be successfully treated with antibiotics if it is caught early enough.

Bubonic plague, the most common, is named for its distinctive buboes, or swollen lymph nodes, near the skin, usually in the groin and armpits. If untreated, the mortality rate is 30 to 50 percent, Dr. Dennis said.

Septicemic plague occurs when the infection gets into the bloodstream, causing great damage to organs, hemorrhaging of blood vessels and sometimes gangrene and bleeding from the nose and ears. Although uncommon, septicemic plague is nearly always fatal unless antibiotic treatment is begun quickly.

Pneumonic plague, which usually appears in a small percentage of plague cases, is transmitted from one person to another by blood drops that are expelled when the victims cough or sneeze. Dr. Dennis said.

A pandemic killed 25 million people. But what was it?

Cardinal Fabio Carli, later Pope Alexander VII, is depicted caring for plague victims in 17th century Italy.

Pneumonic plague, which usually appears in a small percentage of plague cases, is transmitted from one person to another by blood drops that are expelled when the victims cough or sneeze. Dr. Dennis said.

The most recent outbreak of the disease around four weeks longer than the bubonic plague and can spread almost exactly like the 16-year-after period that officials in 1347 established during the Black Death.

The hemorrhagic fever had a 14-day incubation period, followed by a 24-hour mortality period, during which victims started to bleed but had no symptoms, and a 10-day period of symptoms.

The 36-day course of the disease around 1347, the authors add, was exposure to the spread by fleas from the black rat and people with pneumonic plague.

Human fleas found on dogs and cats may also have played a role in the spread, Dr. Dennis said, although no one is certain to what degree.

Looking at the spread with which the Black Death struck Europe, a number of scholars suggested over the years that the cause was not plague, which usually spreads relatively slowly, but the effect of anthrax, typhus or viral hemorrhagic fever.

Rebutting these theories, a team of French researchers reported in November 2000 in The Proceedings of the National Academy of Scientists that they had found Yersinia pestis in the dental pulp of three people buried in Montpellier, France, in the 14th century. “Medieval Black Death was the plague,” they declared.

But Dr. Duncan and Dr. Scott argue that when bubonic plague occurred in medieval Europe, it was generally confined to Mediterranean port cities and southern France. The French team’s results, the authors say, merely showed that two plague organisms occasionally existed simultaneously in Europe.

“You can forget about bubonic plague as the cause of the Black Death,” Dr. Duncan said in a telephone interview from Liverpool.

Fleas infected with Yersinia pestis move to people only after their host rats have died, he said, yet there are no reports from the time of a huge number of dead rats.

Dr. Duncan added that there was little evidence that the disease spread to England, but it is possible that the disease spread to England, but it is possible that the disease could have moved from north to south Europe, and vice versa.

Dr. Scott and Dr. Duncan’s conclusions are that a hemorrhagic fever caused the Black Death, and that it was caused by a virulent virus, probably in the 17th century.

Although questions remain about how the rapid spread of a high mortality rate of the Black Death, as well as the size of the epidemic, the authors argue, that is no better understood.

But some scientists suggest a more concrete evidence — for example, finding Yersinia pestis in patients from north and England — is needed to settle the question of what caused the Black Death.

“Yersinia still seems to me a more reasonable pathogen for the Black Death,” said Joshua Ledberg, a virologist from the University of Pennsylvania and a professor of medicine at Rockefeller University, "but I say that with much skepticism."
Black Death
New Research Suggests Black Death May Have Been Caused by Ebola-Like Virus

By Jen Sterling

LONDON, July 30 — Controversial new research suggests that contrary to the history books, the "Black Death" that devastated medieval Europe was not the bubonic plague, but rather an Ebola-like virus.

History books have long taught the Black Death, which wiped out a quarter of Europe’s population in the Middle Ages, was caused by bubonic plague, spread by infected fleas that lived on black rats. But new research in England suggests the killer was actually an Ebola-like virus transmitted directly from person to person.

The Black Death killed some 25 million Europeans in a devastating outbreak between 1347 and 1352, and then reappeared periodically for more than 300 years. Scholars had thought flea-infested rats living on ships brought the disease from China to Italy and then the rest of the continent.

But researchers Christopher Duncan and Susan Scott of the University of Liverpool say that the flea-borne bubonic plague could not have torn across Europe the way the Black Death did.

"If you look at the way it spreads, it was spreading at a rate of around 30 miles in two to three days," says Duncan. "Bubonic plague moves at a pace of around 100 yards a year."

Unlike the bubonic plague, a bacterial disease which still exists in parts of Asia, India and North America, viral diseases are passed on from person to person, usually by breath or touch.

Ebola-Like Symptoms Cited

In their new book Biology of Plagues: Evidence from Historical Populations, Duncan and Scott compare the signs and symptoms of the Black Death with modern-day viruses such as the Spanish flu, the West Nile virus and, most closely, Ebola.

Medieval descriptions of the Black Death sound like the hemorrhagic fever caused by an Ebola-like virus, the authors say. Such fever strikes fast and causes blood vessels to burst underneath the skin, bringing out welts, similar to what British medical texts from the Middle Ages describe as "God’s tokens."
The liquidization of internal organs that causes excruciating pain in Ebola victims matches the descriptions of historical autopsies on plague victims, which similarly describe internal organs being dissolved along with the appearance of a black liquid, according to the authors.

Duncan and Scott also note that efforts to quarantine the Black Death were successful. In the wake of the first outbreak, Europeans learned that quarantining infected families for 40 days was effective in stopping the spread. Such a measure would not have worked if the disease were transmitted by rats, the authors suggest, because rats do not observe quarantines.

Also, the 40-day period was enough time to ensure the disease finished its incubation period. One of the difficulties in controlling the Ebola virus is that its symptoms start to appear only about five to 22 days after exposure. Therefore people who appear perfectly healthy could be carrying the lethal disease.

Skeptics Say Theorists Should Work Plague by Plague

Ann Carmichael, a historian who is an expert on the Black Plague, welcomes the work produced by Duncan and Scott, but remains skeptical.

"It is problematic to assimilate evidence over four centuries and draw conclusive theories," says Carmichael. "We must look at it on a plague-by-plague basis."

According to Carmichael, texts dating from 14th-century Italy provide extremely detailed and consistent accounts that describe visible swellings called buboes that developed predominantly under the skin around the groin and armpits — a prime target area for fleas. The buboes are what give bubonic plague its name.

"We don't know that Florentine buboes looked like 'God's tokens' found in England, however," she says.

The research by Duncan and Scott is a good start, but there is need for further research, says Carmichael. There is no robust evidence that quarantining systematically applied worked, she says.

Link to HIV-Resistant Gene?

Duncan and Scott believe their theory of a viral cause for the Black Death is supported by the recent discovery of a mutated gene called CCR5 that is resistant to HIV/AIDS. It is estimated that approximately 10 percent to 18 percent of those of European descent carry the gene.

American researchers working on the gene have calculated that the first mutation of the gene took place around 650 years ago — coinciding with the first outbreak of the Black Death in Europe.

According to Duncan, a process of natural selection could have left Europe populated predominantly by those carrying the mutated gene. This would account for the high percentage of the European population that still carries this gene.

(Sterling 2001:1-3)
### U.S. Department of Health & Human Services
Centers for Disease Control and Prevention

**ANNEXURE L**

**ADULT HIV/AIDS CONFIDENTIAL CASE REPORT**

(Patients 13 years of age or older at time of diagnosis)

**II. HEALTH DEPARTMENT USE ONLY**

Form Approved OMB No. 0920-0573 Exp Date 11/30/2005

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<th>Day</th>
<th>Yr.</th>
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**III. DEMOGRAPHIC INFORMATION**

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<th>Current Status:</th>
<th>Date of Death (Mo. Day):</th>
<th>State/Territory of Death:</th>
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<td>3</td>
<td>4</td>
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<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
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</table>

**III. RESIDENCE AT DIAGNOSIS**

City: | County: | State/Country: | Zip Code: |
|-------|---------|----------------|-----------|

**IV. FACILITY OF DIAGNOSIS**

Facility Name: | City: | State/Country: |
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<tr>
<td>Unk.</td>
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</table>

**V. PATIENT HISTORY**

AFTER 1977 AND PRECEDING THE FIRST POSITIVE HIV ANTIBODY TEST OR AIDS DIAGNOSIS, THIS PATIENT HAD

- Sex with male
- Sex with female
- Injected nonprescription drugs
- Received clotting factor for hemophilia/coagulation disorder, other than von Willebrand's disease
- Transfusion recipient with documented HIV infection
- Transplant recipient with documented HIV infection
- Person with AIDS documented HIV infection

**VI. LABORATORY DATA**

1. HIV ANTIBODY TESTS AT DIAGNOSIS:
   - HIV-1 EIA
   - HIV-1/HIV-2 combination EIA
   - HIV-1 Western blot/IFA
   - Other HIV antibody tests

2. POSITIVE HIV DETECTION TEST:
   - PCR, DNA or RNA probe
   - Other (specify):

3. DETECTABLE VIRAL LOAD TEST:
   - Test type:
     - Copies/mL
     - Other:

4. IMMUNOLOGIC LAB TESTS:
   - Date of last documented negative HIV test
   - If HIV laboratory tests were not documented, is HIV diagnosis documented by a physician?

---

_CDC 50.42A REV. 01/2003 Page 1 of 2_ – ADULT HIV/AIDS CONFIDENTIAL CASE REPORT –
### VIII. CLINICAL STATUS

<table>
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<tr>
<th>AIDS INDICATOR DISEASES</th>
<th>Initial Diagnosis Def. Pres.</th>
<th>Initial Date Mo. Yr.</th>
<th>AIDS INDICATOR DISEASES</th>
<th>Initial Diagnosis Def. Pres.</th>
<th>Initial Date Mo. Yr.</th>
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<td>Lymphoma, Burkitt's (or equivalent term)</td>
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<td>Lymphoma, Immunoblastic (or equivalent term)</td>
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<td></td>
<td>Pneumocystis carinii pneumonia</td>
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<tr>
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<td>Pneumonia, recurrent, in 1 2 mo. period</td>
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<td></td>
<td>Progressive multifocal leukoencephalopathy</td>
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<td>Kaposis's sarcoma</td>
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<td>Wasting syndrome due to HIV</td>
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</table>

Def. = definitive diagnosis  Pres. = presumptive diagnosis  * RVCT CASE NO.:  

**If HIV tests were not positive or were not done, does this patient have an immunodeficiency that would disqualify him/her from the AIDS case definition?**

- Yes  
- No  
- Unknown

### IX. TREATMENT/SERVICES REFERRALS

#### Has this patient been informed of his/her HIV infection?

- Yes  
- No  
- Unknown

This patient's partners will be notified about their HIV exposure and counseled by:

- Health department  
- Physician/provider  
- Patient  
- Other  
- Unknown

This patient has received or is receiving:

- Anti-retroviral therapy  
- PCP prophylaxis  
- Other  
- None  
- Unknown

This patient has been enrolled at:

- Clinical Trial  
- NIH-sponsored  
- Other  
- None  
- Unknown

This patient's medical treatment is primarily reimbursed by:

- Medicaid  
- Private insurance/HMO  
- Other  
- Other  
- Unknown

**FOR WOMEN:**

- This patient is receiving or has been referred for gynecological or obstetrical services:  
- Is this patient currently pregnant?  
- Has this patient delivered live-born infants?  
- Yes (if delivered after 1977, provide birth information below for the most recent birth)

### X. COMMENTS

__________________________________________  

__________________________________________

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Public reporting burden for this collection of information is estimated to average 10 minutes per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. An agency may not conduct or sponsor, and a person is not required to respond to a collection of information unless it displays a currently valid OMB control number. Since comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden to CDC, Project Clearance Officer, 1600 Clifton Road, MS D-24, Atlanta, GA 30333, Attn: PHS (4520-0570). Do not send the completed form to this address.