



Virology for 2nd Year M.D. Students



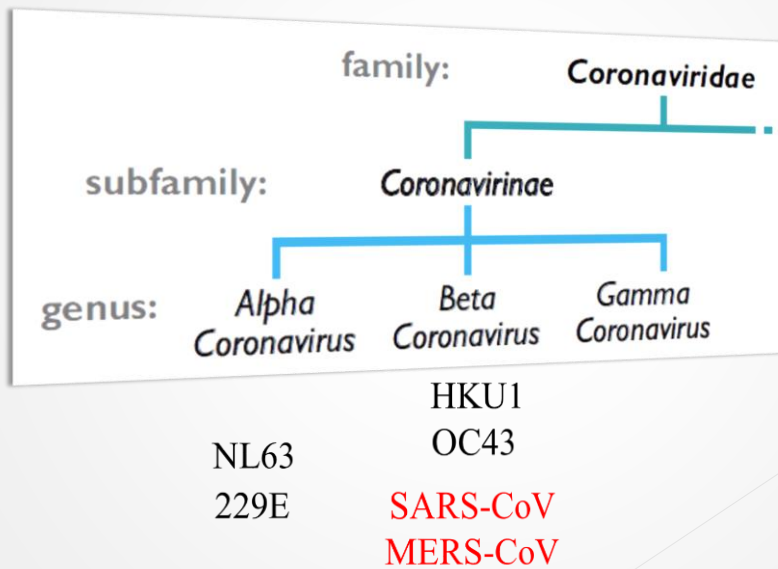
The rest of RNA viruses and Prions #14

University of Jordan
School of Medicine

Malik Sallam, MD, PhD

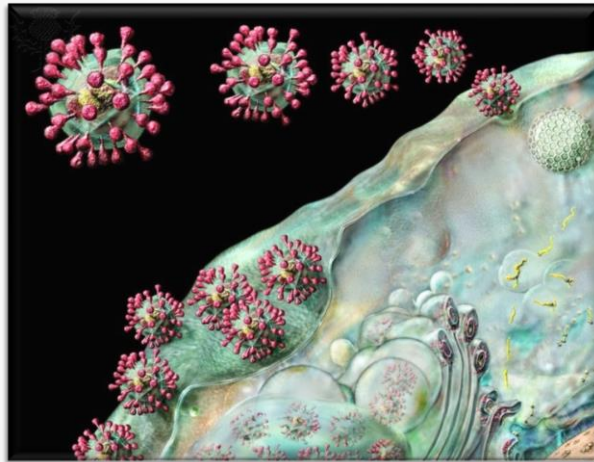


Coronaviridae Classification





Coronavirus Structure





Coronavirus Receptors

ACE2
SARS-CoV
 NL63

Aminopeptidase N
 (APN; CD13)
229E

Dipeptidyl peptidase
 4 (DPP4;CD26)
MERS-CoV

N-acetyl-9-O-
 acetylneuraminic
 acid
 OC43

O-acetylated sialic
 acid
 HKU1



Coronavirus Transmission & Clinical Features





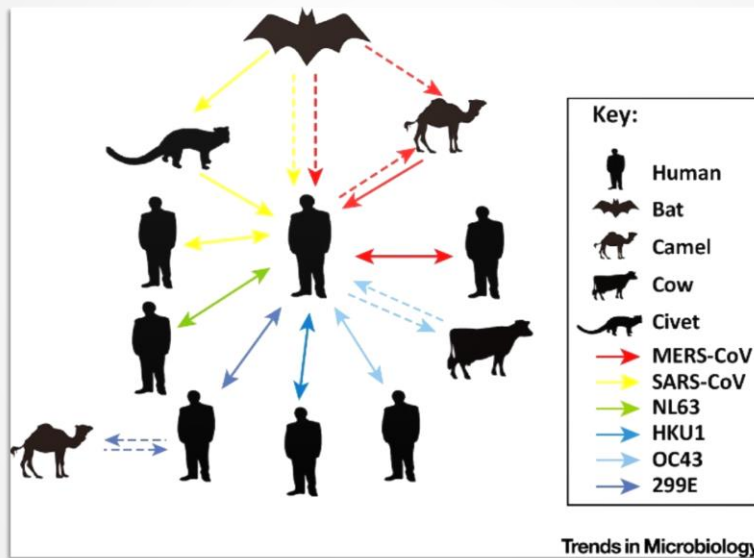
Clinical Features of Coronavirus Infections



HCoV	Clinical Symptoms	Case Fatality Rate	Incubation Period	Median Time to Death
229E	Coryza	N/A	2-5 days	-
OC43	Coryza	N/A	2-5 days	-
SARS-CoV	Fever, myalgia, headache, chills, cough, dyspnea, respiratory distress, diarrhea	9%	2-11 days	23 days
NL63	Coryza, Croup	N/A	2-4 days	-
HKU1	Coryza	N/A	2-4 days	-
MERS-CoV	Fever, myalgia, headache, chills, cough, dyspnea, pneumonia, vomiting, diarrhea	36%	2-13 days	14 days



Coronavirus Origin & Epidemiology



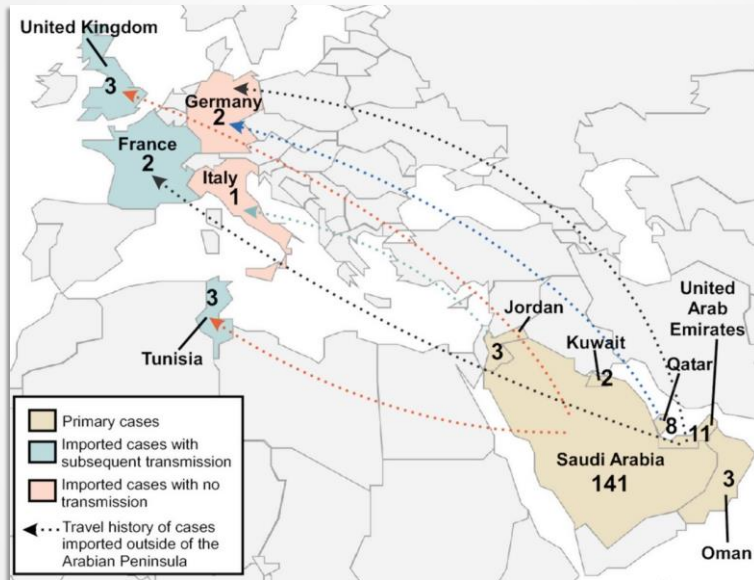


Coronavirus Origin & Epidemiology



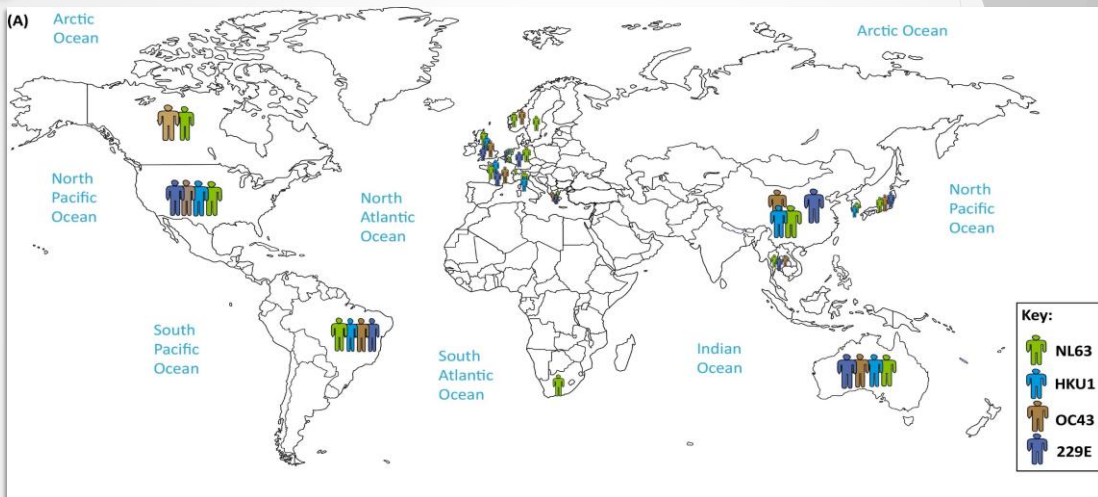


Coronavirus Origin & Epidemiology



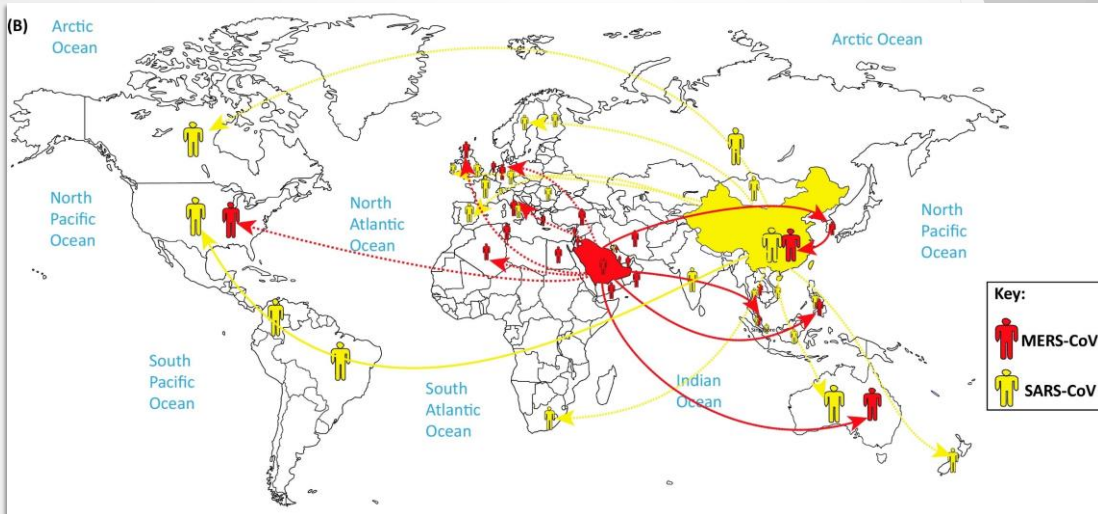


Coronavirus Origin & Epidemiology





Coronavirus Origin & Epidemiology





Coronavirus Infection Management and Prevention



COVID-19
CORONAVIRUS DISEASE

STOP THE SPREAD OF GERMS

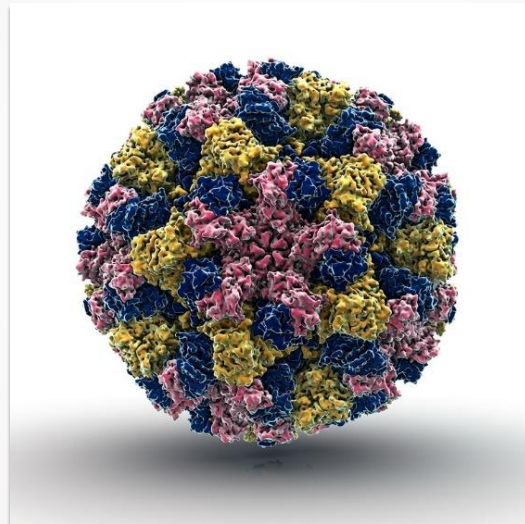
Help prevent the spread of respiratory diseases like COVID-19

- Avoid close contact with people who are sick.
- Cover your cough or sneeze with a tissue, then throw the tissue in the trash.
- Avoid touching your eyes, nose, and mouth.
- Clean and disinfect frequently touched objects and surfaces.
- Wash your hands often with soap and water for at least 20 seconds.
- Stay home when you are sick. Do not go out or public when you are sick.
- If you have the symptoms, use a face mask to avoid infecting others.
- Avoid crowds and unnecessary travel.

For more information: www.cdc.gov/COVID19
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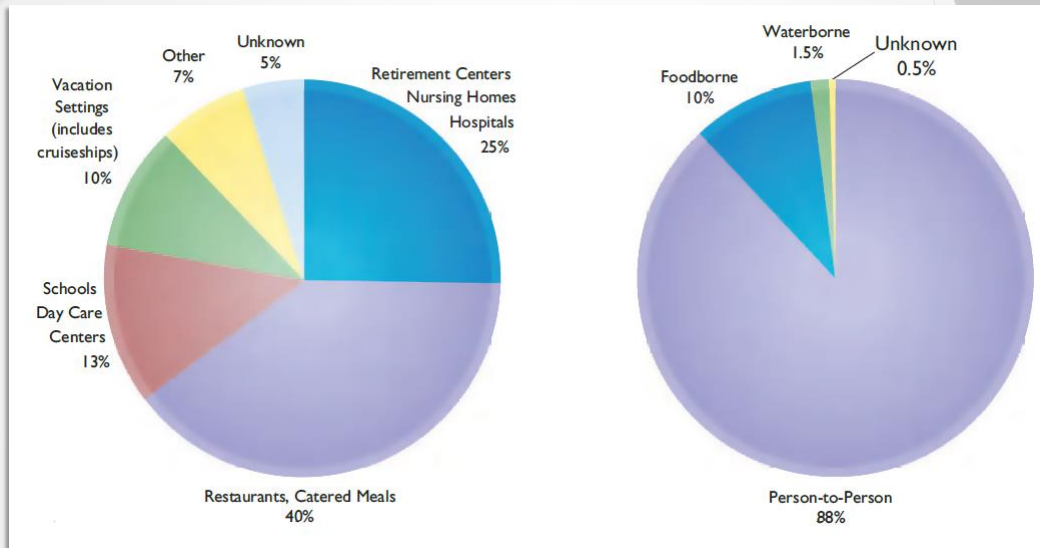


Caliciviridae



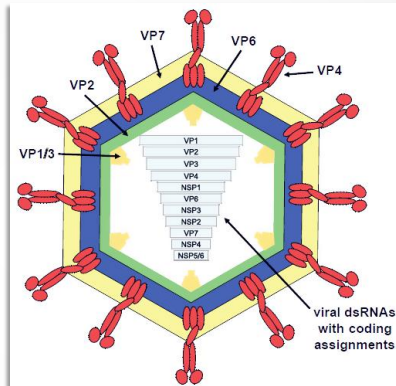


Norovirus Transmission and Clinical Features



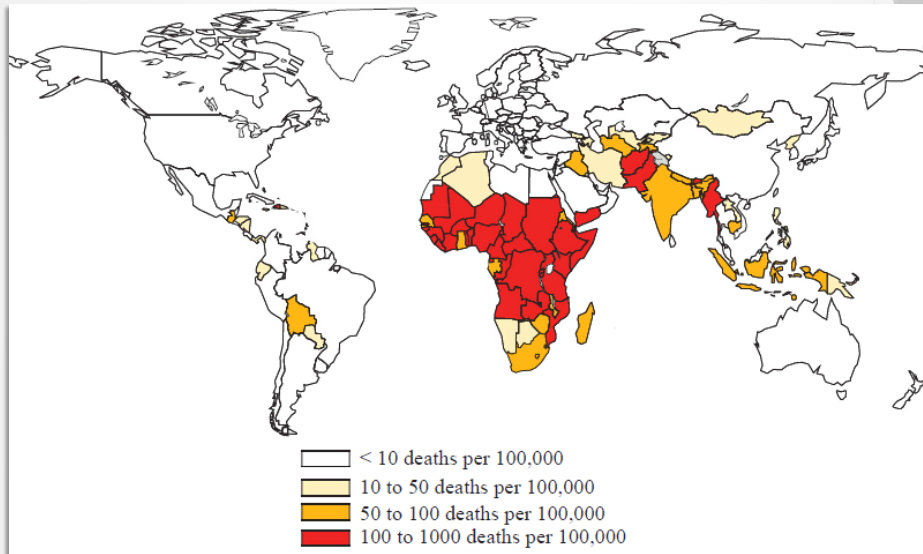


Reoviridae



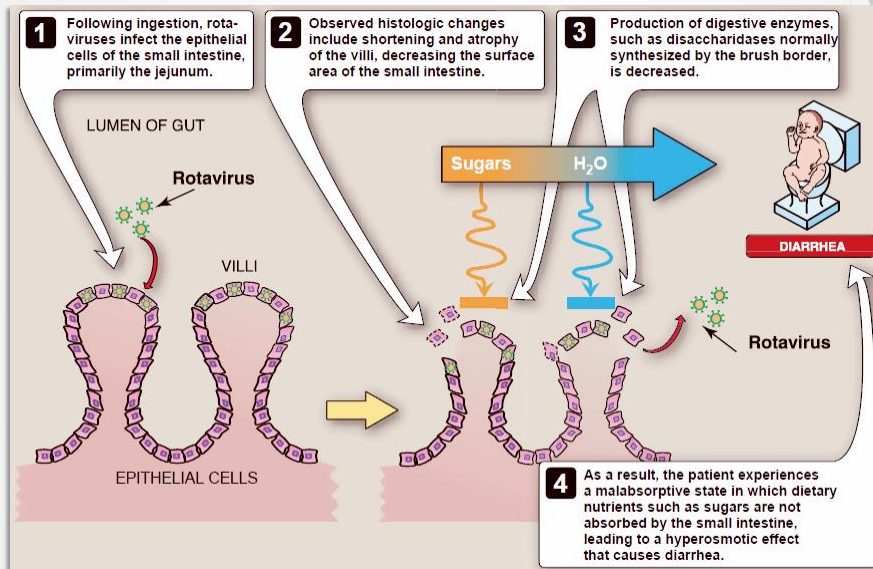


Rotavirus Gastroenteritis





Rotavirus Gastroenteritis





Rotavirus Dx, Rx and Prevention



Ready to use/ No reconstitution require

Oral suspension in pre-filled oral applicator

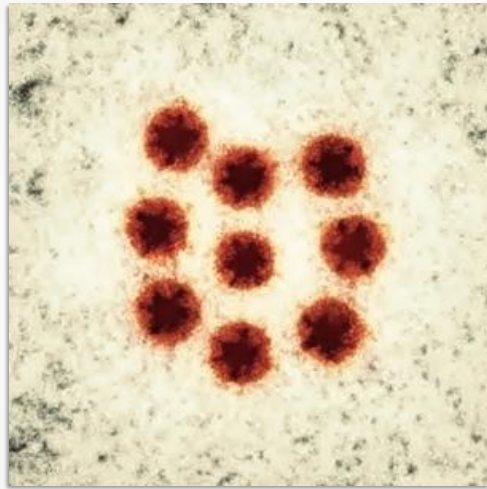
Rotarix®

Oral suspension in pre-filled oral applicator
Rotavirus vaccine, live
Oral use
Do not inject!

GlaxoSmithKlin



Astrovirus





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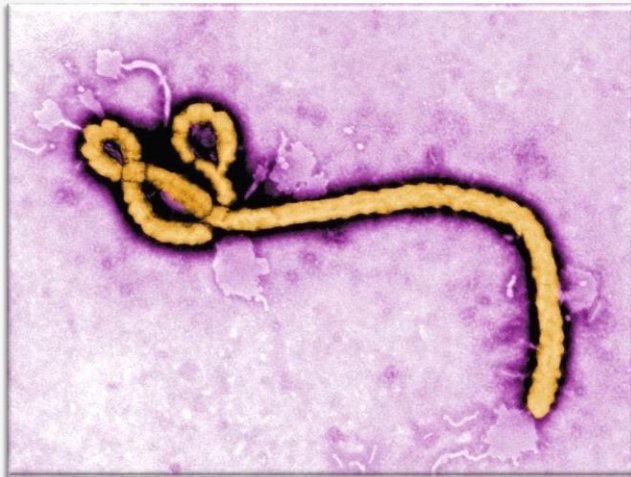
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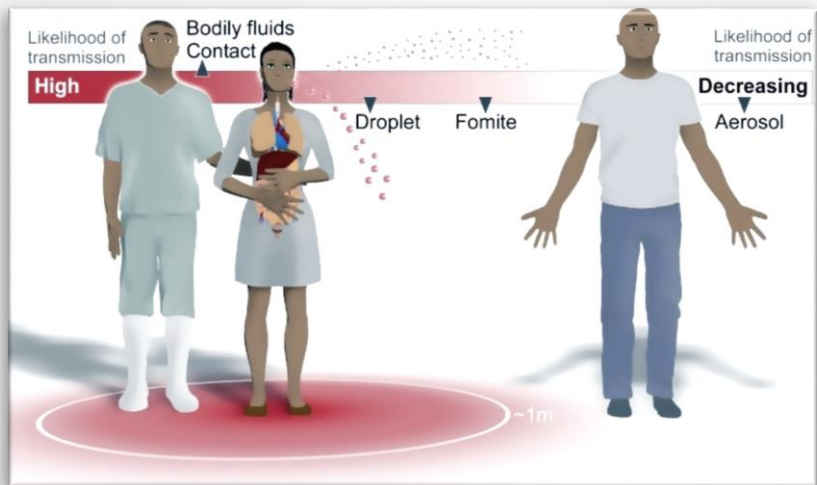


Ebola





Ebola Transmission





Ebola



- Characterized by fever, headache, sore throat, and myalgia followed by abdominal pain, vomiting, diarrhea, and rash, with both internal and external bleeding, often leading to shock and death.
- Ebola virus has a tropism for cells of the macrophage system, dendritic cells, interstitial fibroblasts, and endothelial cells.



Ebola



- Very high titers of virus are present in many tissues, including the liver, spleen, lungs, and kidneys, and in blood and other fluids.
- Filoviruses have the highest mortality rates of all the viral hemorrhagic fevers (syndromes with severe multi-organ involvement).



Rabies Virus Description



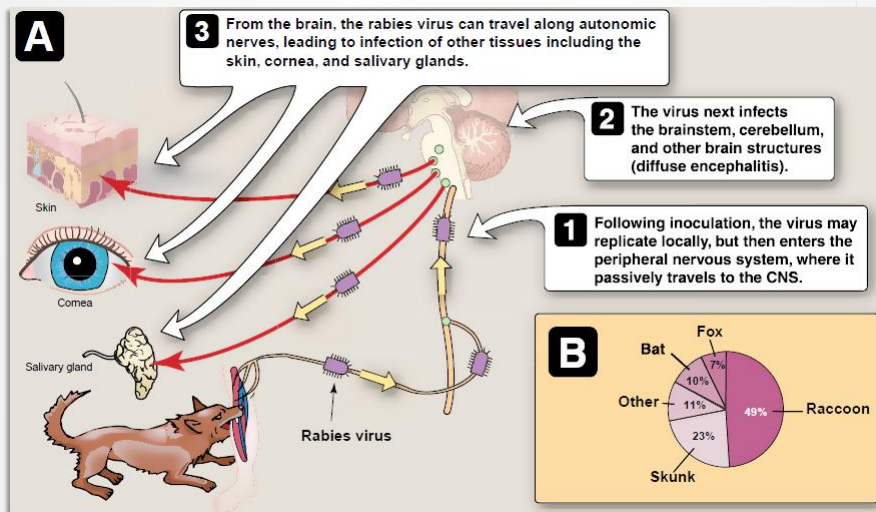


Animal Susceptibility





Rabies Pathogenesis and Pathology





Rabies Pathogenesis and Pathology



- Rabies virus multiplies in muscle at the site of inoculation and then enters peripheral nerves at NMJs and spreads up the nerves to the CNS.
- The virus multiplies in the CNS and progressive **encephalitis** develops.
- The virus then spreads through peripheral nerves to the salivary glands and other tissues.
- **The organs with the highest titers of virus are the salivary gland.** Other organs where rabies virus has been found include pancreas, kidney, heart, retina, and cornea.



Rabies Pathogenesis and Pathology

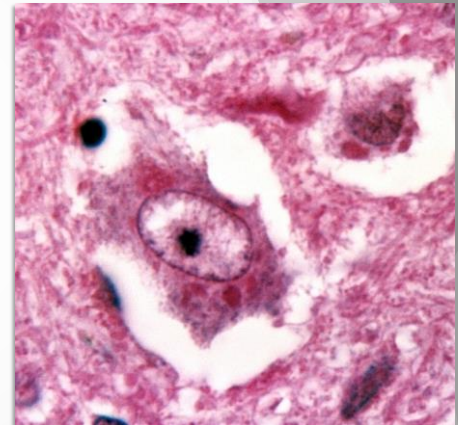
- The incubation period (typically 1–3 months but **may vary from 1 week to 1 year**) depends on the host's age, genetics, and immune status, the **viral strain**, **amount of inoculum**, the **severity of lacerations**, and the **distance the virus has to travel** from its point of entry to the CNS.
- There is a higher attack rate and shorter incubation period in persons bitten on the face or head; the lowest mortality occurs in those bitten on the legs.



Rabies Pathogenesis and Pathology



- Rabies virus produces a specific **eosinophilic cytoplasmic** inclusion, **the Negri body**, in infected nerve cells, that are filled with viral nucleocapsids.
- **The presence of such inclusions is pathognomonic of rabies** but is not observed in at least 20% of cases.





Rabies Clinical Features

- The clinical spectrum can be divided into three phases:
 - **Prodrome:** malaise, anorexia, headache, photophobia, nausea and vomiting, sore throat, and fever.
 - **Acute neurologic phase:** CNS signs including nervousness, apprehension, hallucinations, and bizarre behavior. sympathetic overactivity is observed, including lacrimation, pupillary dilatation, and increased salivation and perspiration. Hydrophobia and aerophobia are common as well.
 - **Coma and death:** The major cause of death is cardiorespiratory arrest.



Rabies Diagnosis



- Clinical through history of exposure.
- Rabies antigens or nucleic acid detection.
- Serology.
- Virus isolation.

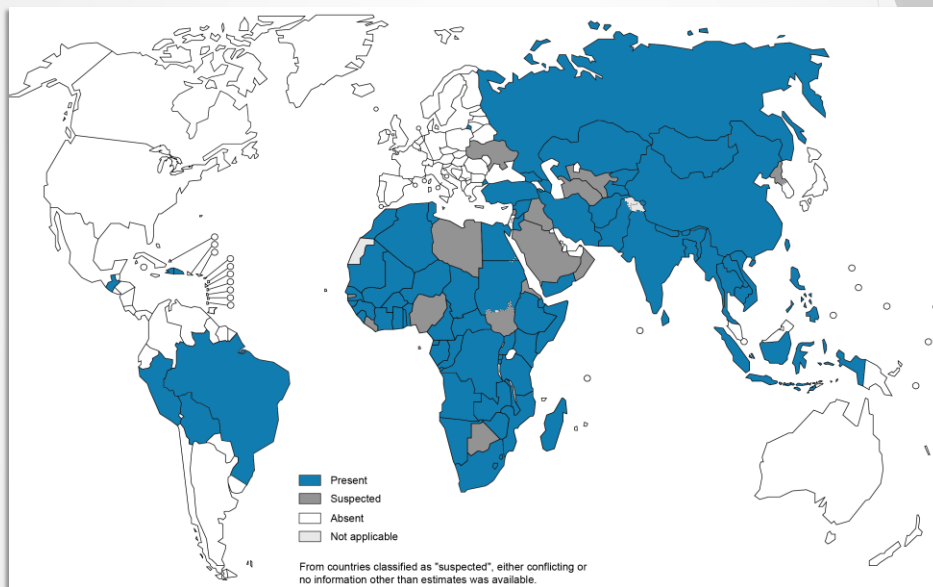


Rabies Management

- Once an individual has clinical symptoms of rabies, there is no effective treatment.
- A killed rabies virus vaccine is available for prophylaxis.
- Postexposure prophylaxis refers to treatment after an animal bite or suspected of being rabid, and consists of thorough **cleaning of the wound, passive immunization, and active immunization.**
- Prevention of initial exposure is, however, the most important mechanism for controlling human rabies.

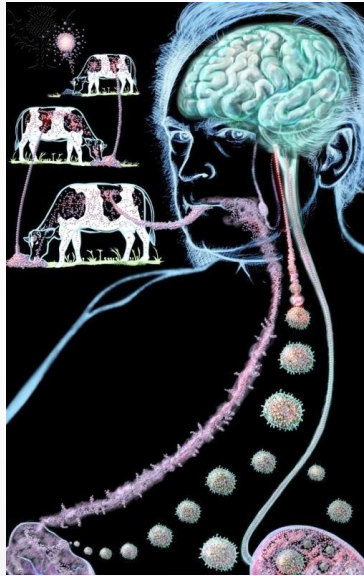


Rabies Epidemiology





Transmissible Spongiform Encephalopathies (Prion Diseases)





Transmissible Spongiform Encephalopathies (Prion Diseases)



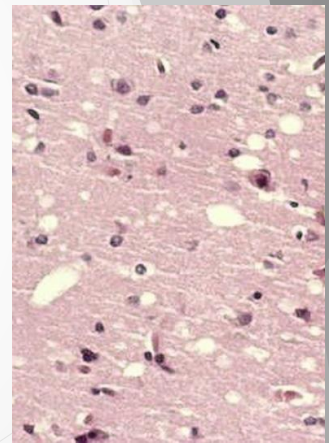
- Transmissible spongiform encephalopathies are **degenerative CNS diseases**.
- Infectivity is associated with **proteinaceous material devoid of detectable amounts of nucleic acid**.
- Prions are resistant to treatment with formaldehyde, dry heat, boiling, ethanol, proteases, and ionizing radiation.
- Prions are sensitive to phenol, household bleach, ether and autoclaving (1 hour, 121°C). Guanidine thiocyanate is highly effective in decontaminating medical supplies and instruments.



Prion Diseases



- Prion diseases are **confined to the nervous system**.
- The basic features are **neurodegeneration and spongiform changes**.
- Long incubation periods (months to decades) precede the onset of clinical illness and are followed by chronic progressive disease (weeks to years).
- The diseases are always fatal, with no known cases of remission or recovery.

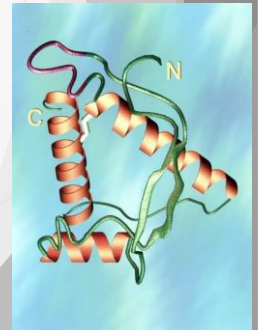




Prion Diseases



- PrP^C Protein (encoded by a chromosomal gene, designated PRNP in humans)
- The PrP gene is located on the short arm of chromosome 20 in humans.
- In contrast to viruses, **prions are nonimmunogenic**.
- The crossing of prions from one species to another is restricted by what has been called the “species barrier”.
- The prion diseases are uniformly fatal. No human or animal has ever recovered from a prion disease once neurologic dysfunction is manifest.





Transmissible Spongiform Encephalopathies (Prion Diseases)



- Prions are composed entirely of protein.
- It is proposed that PrP^{Sc} acts as a template which promotes the conversion of PrP^C to PrP^{Sc} and that this conversion involves **only conformational change**.
- In humans, the PrP prion diseases occur in three different forms: (a) sporadic, (b) inherited, and (c) infectious





Kuru and Creutzfeldt–Jakob Disease



- CJD is a progressive multifocal dementia, peak onset between 60–65.
- Kuru occurred only in the eastern highlands of New Guinea and was spread by customs surrounding **ritual cannibalism** of dead relatives. Since the practice has ceased, the disease has disappeared.
- CJD in humans develops gradually, with progressive dementia, ataxia, and myoclonus, and leads to death in 5–12 months.



BSE and vCJD



- A disease similar to scrapie, designated bovine spongiform encephalopathy (BSE), or “mad cow disease,” emerged in cattle in Great Britain in 1986.
- This outbreak was traced to the use of cattle feed that contained contaminated bone meal from scrapie-infected sheep and BSE-infected cattle carcasses.
- The use of such cattle feed was prohibited in 1988. The epidemic of “mad cow disease” peaked in Great Britain in 1993.



BSE and vCJD



- In 1996, a new variant form of CJD was recognized in the United Kingdom that occurred in younger people and had distinctive pathologic characteristics similar to those of BSE.
- It is now accepted that the new variant forms of CJD and BSE are caused by a common agent, indicating that the BSE agent had infected humans.
- Through 2006, over 150 people had been diagnosed with new variant CJD in England, and most had died.

