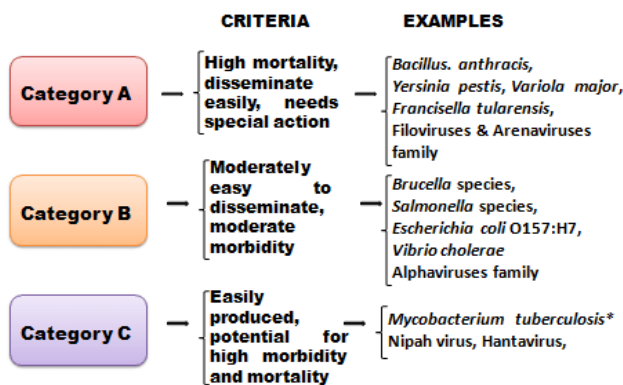




MICROBIOLOGY BINGE

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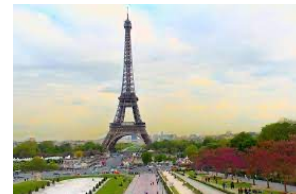
Category	A	B	C
Definition	Pose the highest risk to national security because they <ul style="list-style-type: none"> Can be easily disseminated or transmitted from person to person Result in high mortality rates Have potential to cause public panic and social disruption Require special preparedness actions 	Pose the second highest risk because they <ul style="list-style-type: none"> Are moderately easy to disseminate Result in low mortality rates Require enhancement of diagnostic and surveillance capability 	Emerging pathogens that could be engineered for mass dissemination because they: <ul style="list-style-type: none"> Are available Are easily produced and disseminated Have potential for high mortality rates
Examples	<ul style="list-style-type: none"> Anthrax Botulism Plague Smallpox Tularemia Viral hemorrhagic fevers (e.g. Ebola, Marburg) 	<ul style="list-style-type: none"> West Nile Virus Caliciviruses Hepatitis A Ricin toxin Salmonella Diarrheagenic <i>E. coli</i> 	<ul style="list-style-type: none"> Influenza SARS Rabies Multi-drug resistant tuberculosis Yellow fever Tickborne hemorrhagic fever

Category	Definition of Category	Disease	Organism(s)/Agent(s)
A	High-priority agents include organisms that pose a risk to national security because they: Can be easily disseminated or transmitted from person to person Result in high mortality and have the potential for major public health impact Might cause public panic and social disruption Require special action for public health preparedness	Anthrax Botulism Plague Smallpox Tularemia Viral hemorrhagic fevers	<i>Bacillus anthracis</i> <i>Clostridium botulinum</i> toxin <i>Yersinia pestis</i> <i>Variola major</i> <i>Francisella tularensis</i> Filoviruses (eg, Ebola, Marburg) Arenaviruses (eg, Lassa, Machupo)
B	Second highest priority agents include those that: Are moderately easy to disseminate Result in moderate morbidity rates and low mortality Require specific enhancements of laboratory diagnostic capacity and enhanced disease surveillance	Brucellosis Epsilon toxin Food safety threats Glanders Melioidosis Psittacosis Q fever Ricin toxin Staphylococcal enterotoxin B Typhus fever Viral encephalitis Water safety threats	<i>Brucella</i> species <i>Clostridium perfringens</i> <i>Salmonella</i> species, <i>Escherichia coli</i> O157:H7, <i>Shigella</i> , and so forth <i>Burkholderia pseudomallei</i> <i>Chlamydia psittaci</i> <i>Coxiella burnetii</i> <i>Ricin</i> (<i>Ricinus communis</i> (castor beans)) <i>Staphylococcus aureus</i> <i>Rickettsia prowazekii</i> Alphaviruses (eg, Venezuelan equine encephalitis, eastern equine encephalitis, western equine encephalitis) <i>Vibrio cholerae</i> , <i>Cryptosporidium parvum</i> and so forth
C	Third highest priority agents include emerging pathogens that could be engineered for mass dissemination in the future because of: Availability Ease of production and dissemination Potential for high morbidity and mortality and major health impact	Emerging infectious diseases	Nipah virus Hantavirus Tick-borne hemorrhagic fever viruses Tick-borne encephalitis viruses Yellow fever Multidrug-resistant tuberculosis

Category	Organism/disease
A	Smallpox Anthrax Tularemia Plague Botulism Viral hemorrhagic fevers
B	Brucellosis Glanders Ricin toxin Typhus fever Q fever Staphylococcal enterotoxin B Viral encephalitis (alphavirus: VEE, EEE, WEE) Water safety threats (e.g., <i>Vibrio cholerae</i> , <i>Cryptosporidium parvum</i>)
C	Emerging infectious diseases such as Nipah virus and Hantavirus



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B C D E F G M P Q R S T V W

C NIHA MAM

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Figure 1: Biosafety Levels

Biosafety Level (BSL)	Characteristics	Pathogens/Disease
BSL-4	Infectious aerosol transmission that may cause serious or lethal infections with no treatment available	Ebola virus, Variola virus (smallpox), Marburg virus
BSL-3	Infectious aerosol transmission that may cause serious or lethal infections	Coronavirus, Mycobacterium tuberculosis, Yersinia pestis (plague), malaria
BSL-2	Infectious agents of moderate risk with ingestion or mucous membrane transmission	Influenza, Lyme disease, salmonella, measles, mumps
BSL-1	Low-risk agents that are not known to cause human disease	E. coli



World Health Organization designates laboratory facilities under four level

Relation of risk groups to biosafety levels, practice and equipment	BIOSAFETY LEVELS	LABORATORY TYPE	LABORATORY PRACTICES	SAFETY EQUIPMENT
1	Basic biosafety Level 1	Basic teaching research	GMT	None; open bench work
2	Basic biosafety Level 2	Primary health services; diagnostic service, research	GMT plus protective clothing, biohazard sign	Open bench plus BSC for potential aerosols
3	Containment biosafety Level 3	Special diagnostic services, research	As level 2 plus special clothing, controlled access, directional airflow	BSC and/or other primary devices for all activities
4	Maximum containment biosafety Level 4	Dangerous pathogen units	As level 3 plus airlock entry, shower exit, special waste disposal	Class III BSC, or positive pressure suits in conjunction with class II BSCs, double ended autoclave (through the wall), filtered air

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Group 1	biological agents unlikely to cause human disease	No pathogenic organisms	-	-
Group 2	Biological agents that can cause human disease and may be hazardous to workers but are unlikely to spread to the community; effective treatment or prophylaxis is usually available.	<ul style="list-style-type: none"> Bacillus (except anthrax) Streptococci Staphylococci E. coli Clostridium species Corynebacterium diphtheria Salmonella typhi 	<ul style="list-style-type: none"> Adenovirus Calicivirus Coronavirus Herpesvirus Influenza virus 	<ul style="list-style-type: none"> Cryptococcus Candida Dermatophytes Aspergillus
Group 3	Biological agents that can cause severe human disease and are a serious hazard to workers; they may spread to the community; but effective treatment or prophylaxis is usually available.	<ul style="list-style-type: none"> B. Anthracis Brucella Coxiella burnetii Tularemia M. tuberculosis 	<ul style="list-style-type: none"> Lymphocytic choriomeningitis Haanta virus St. Louis encephalitis virus Japanese encephalitis virus West Nile encephalitis virus 	
Group 4	Same as group 3 expect that effective treatment or prophylaxis is usually not available		<ul style="list-style-type: none"> Lassa Ebola Marburg Herpes simiae Crimean-Congo haemorrhagic fever (CCHF) 	



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Biosafety level	Lab practices	Safety Equipment	Facility
BSL 1	Work on open lab benches or tables. Good microbiological techniques followed	PPE (lab coats, gloves, eye protection) are worn as needed.	Sink for hand washing. Separate doors from the rest of the facility.
BSL 2	Access restricted to the laboratory	Appropriate PPE (lab coats, gloves, eye protection, and face shields). Biological safety cabinets (BSC) to prevent potential aerosols. Autoclave or alternative method for decontamination	Sink and eyewash easily available. Self-closing doors.
BSL 3	Access restricted and controlled at all times. Medical surveillance of laboratory workers.	Appropriate PPE must be worn, and respirators might be required. BSC for all activities.	Hands-free sink and eyewash. Directional airflow from clean areas towards potentially contaminated areas. Entrance to the lab is through 2 self-closing and interlocked doors.
BSL 4	Change clothing before entering and shower upon exiting. Decontaminate all materials before exiting.	BSC and full-body, air-supplied, positive pressure suit. Double-ended autoclave (through the wall).	Laboratory in a separate building or isolated in a restricted zone. Dedicated supply and exhaust air, as well as vacuum lines and decontamination systems.

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Disease	Incubation Period	Disease	Incubation period (days)*	Disease	Range
Chickenpox	14–16 days	Influenza	1–2	COVID-19	3–14 days
Cholera	1–3 days	Common cold	1–3	Cholera	0.5–4.5 days
Diphtheria	2–6 days	Bronchiolitis, croup	3–5	Common cold	1–3 days
Gas gangrene	1–5 days	Acute respiratory disease (adenoviruses)	5–7	Ebola	1–21 days
Gonorrhoea	3–5 days	Dengue	5–8	HIV	2–3 weeks to months or longer
Hepatitis A	2 weeks–2 months	Herpes simplex	5–8	Influenza	1–3 days
Hepatitis B	6 weeks–6 months	Enterovirus disease	6–12	MERS	2–14 days
Hepatitis C	2 weeks–6 months	Poliomyelitis	5–20	SARS	1–10 days
Herpes simplex	4 days	Measles	9–12		
Influenza	1–3 days	Smallpox	12–14		
Leprosy	3 months–20+ years	Chickenpox	13–17		
Measles	10–12 days	Mumps	16–20		
Meningitis (bacterial)	1–7 days	Rubella	17–20		
Mumps	2–3 weeks	Mononucleosis	30–50		
Pertussis	5 days–3 weeks	Hepatitis A	15–40		
Pinworm	2–6 weeks	Hepatitis B and C	50–150		
Plague	2–6 days	Rabies	30–100		
Polio	7–14 days	Papilloma (warts)	50–150		
Rabies	2 weeks–2 months (up to 1 year)	AIDS	1–10 yr		
<i>Salmonella</i> food poisoning	12–72 hours				
<i>Staphylococcus</i> food poisoning	1–8 hours				
Syphilis	10 days–3 months				
Tetanus	3 days–5 weeks				
Tuberculosis	2–10 weeks				

*Until first appearance of prodromal symptoms.

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✿ *Pseudomonas* / *Stenotrophomonas maltophilia*
Meropenem resistant, cotrimox sensitive
vs *P. cepacia* → sensitive to both
meropenem and cotrimox.



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★ Syphilis :

- gets better in subsequent pregnancies
- Test for Rx response : VDRL t.
- First Ab: FTA - Abs

Kassowitz law

★ Dory Flop sign

- snail track ulcer
- moth eaten non scarring alopecia
- C. lata
- rhagades
- Pseudo-chancres
 - redux → 3^o
 - CVS, CNS = 3^o

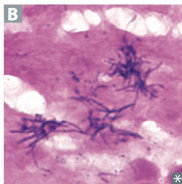
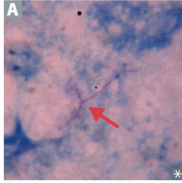


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Nocardia vs Actinomyces



Both are gram \oplus and form long, branching filaments resembling fungi.

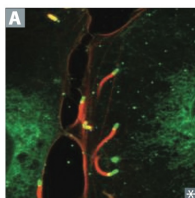
<i>Nocardia</i>	<i>Actinomyces</i>
Aerobe	Anaerobe
Acid fast (weak) A	Not acid fast B
Found in soil	Normal oral, reproductive, and GI microbiota
Causes pulmonary infections in immunocompromised (can mimic TB but with \ominus PPD); cutaneous infections after trauma in immunocompetent; can spread to CNS \rightarrow cerebral abscess	Causes oral/facial abscesses that drain through sinus tracts; often associated with dental caries/extraction and other maxillofacial trauma; forms yellow "sulfur granules"; can also cause PID with IUDs
Treat with sulfonamides (TMP-SMX)	Treat with penicillin

Treatment is a **SNAP**: Sulfonamides—*Nocardia*; *Actinomyces*—Penicillin

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Listeria monocytogenes



Gram \oplus , facultative intracellular rod; acquired by ingestion of unpasteurized dairy products and cold deli meats, transplacental transmission, by vaginal transmission during birth. Grows well at refrigeration temperatures ("cold enrichment").

Forms "rocket tails" (red in **A**) via actin polymerization that allow intracellular movement and cell-to-cell spread across cell membranes, thereby avoiding antibody. Listeriolysin generates pores in phagosomes, allowing its escape into cytoplasm. Characteristic tumbling motility in broth.

Can cause amnionitis, septicemia, and spontaneous abortion in pregnant patients; granulomatous infantisepsis; meningitis in immunocompromised patients, neonates, and older adults; mild, self-limited gastroenteritis in healthy individuals.

Treatment: ampicillin.



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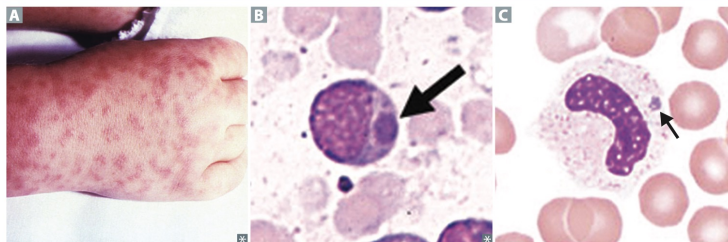


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RASH RARE

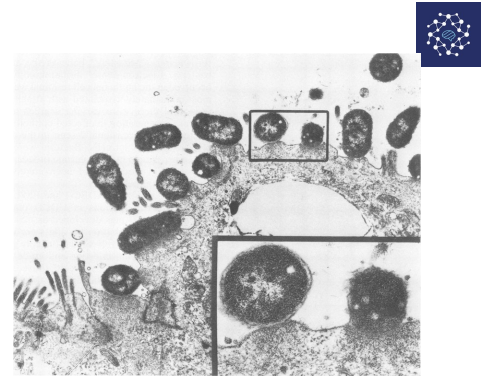
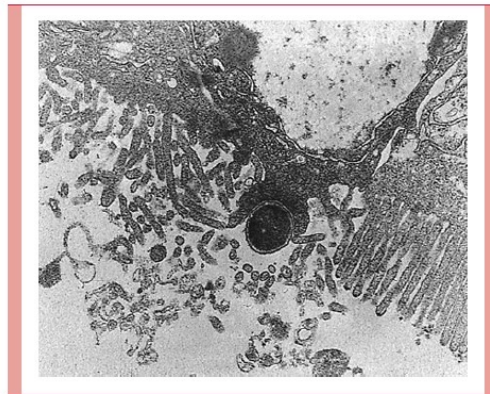
Ehrlichiosis	<i>Ehrlichia</i> , vector is tick. M onocytes with morulae B (mulberrylike inclusions) in cytoplasm.	MEGA: M onocytes = Ehrlichiosis G ranulocytes = Anaplasmosis
Anaplasmosis	<i>Anaplasma</i> , vector is tick. G ranulocytes with morulae C in cytoplasm.	
Q fever	<i>Coxiella burnetii</i> , no arthropod vector. Bacterium inhaled as aerosols from cattle/sheep amniotic fluid. Presents with headache, cough, flulike symptoms, pneumonia, possibly in combination with hepatitis. Common cause of culture \ominus endocarditis.	Q fever is caused by a Q uite C omplicated b ug because it has no rash or vector and its causative organism can survive outside in its endospore form. Not in the <i>Rickettsia</i> genus, but closely related.



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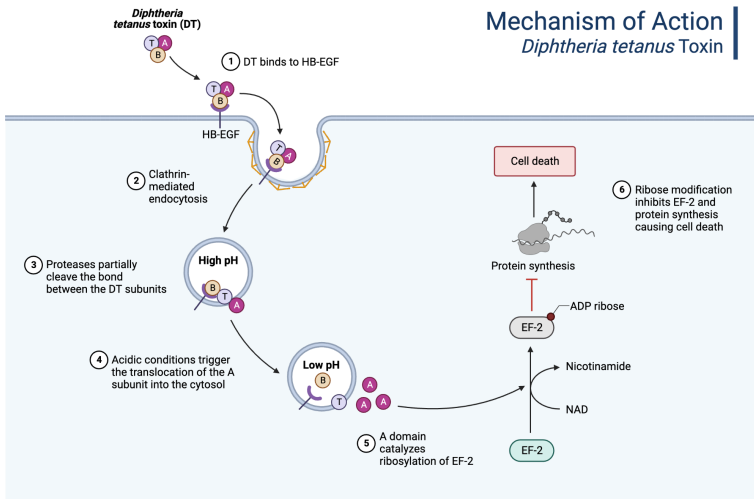
FIGURE 21-4
Enteropathogenic *Escherichia coli* (EPEC) attachment to epithelial cells. The EPEC are attaching to and effacing the microvilli on the epithelial cell surface. The cell's filamentous actin is rearranged at the attachment point



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Mechanism of Action
Diphtheria tetanus Toxin



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Endotoxin

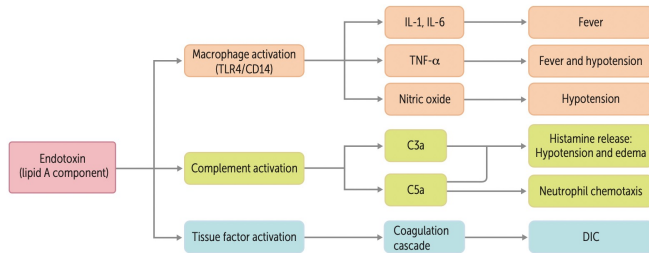
LPS found in outer membrane of gram \ominus bacteria (both cocci and rods). Composed of O-antigen + core polysaccharide + lipid A (the toxic component). *Neisseria* have lipooligosaccharide.

Released upon cell lysis or by living cells by blebs detaching from outer surface membrane (vs exotoxin, which is actively secreted).

Three main effects: macrophage activation (TLR4/CD14), complement activation, and tissue factor activation.

ENDOTOXINS:

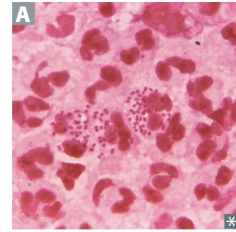
- Edema
- Nitric oxide
- DIC/Death
- Outer membrane
- TNF- α
- O-antigen + core polysaccharide + lipid A
- eXtremely heat stable
- IL-1 and IL-6
- Neutrophil chemotaxis
- Shock



Gram \ominus diplococci. Metabolize glucose and produce IgA proteases. Contain lipooligosaccharides (LOS) with strong endotoxin activity.

N gonorrhoeae is often intracellular (within neutrophils) **A**.

Acid production: meningococci—maltose and glucose; gonococci—glucose.



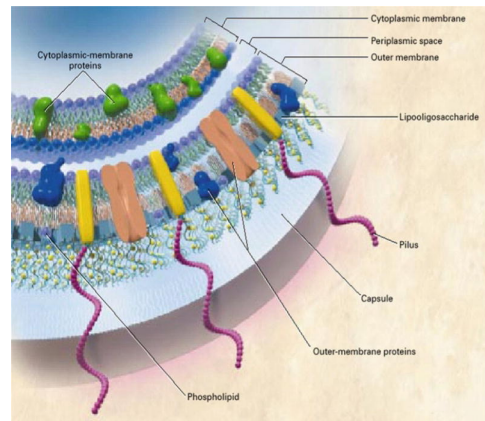
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TABLE 26-4

Virulence factors of *Neisseria meningitidis*

Virulence factors	Biological functions
Capsule	Prevents phagocytosis
LOS endotoxin	Causes damage of the blood vessels associated with meningococcal infections
IgA protease	Destroys IgA immunoglobulin, thereby helps gonococci to attach to the epithelial cells of the upper respiratory tract
Lipooligosaccharides	Stimulates release of TNF- α , which results in host cell damage



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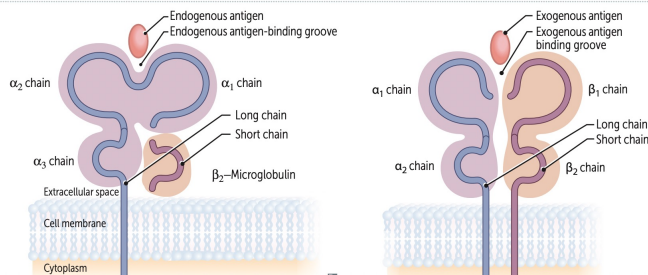




Major histocompatibility complex I and II

	MHC encoded by HLA genes. Present antigen fragments to T cells and bind T-cell receptors (TCRs).	
	MHC I	MHC II
LOCI	HLA-A, HLA-B, HLA-C MHC I loci have 1 letter	HLA-DP, HLA-DQ, HLA-DR MHC II loci have 2 letters
BINDING	TCR and CD8	TCR and CD4
STRUCTURE	1 long chain, 1 short chain	2 equal-length chains (2 α , 2 β)
EXPRESSION	All nucleated cells, APCs, platelets (except RBCs)	APCs
FUNCTION	Present endogenous antigens (eg, viral or cytosolic proteins) to CD8+ cytotoxic T cells	Present exogenous antigens (eg, bacterial proteins) to CD4+ helper T cells
ANTIGEN LOADING	Antigen peptides loaded onto MHC I in RER after delivery via TAP (transporter associated with antigen processing)	Antigen loaded following release of invariant chain in an acidified endosome
ASSOCIATED PROTEINS	β_2 -microglobulin	Invariant chain

STRUCTURE



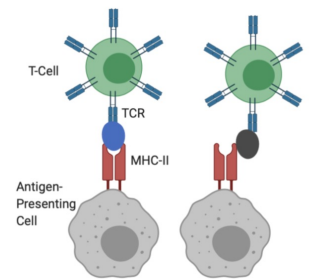
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Superantigens causing shock

Staphylococcus aureus	Toxic shock syndrome toxin (TSST-1)	Cross-links β region of TCR to MHC class II on APCs outside of the antigen binding site → overwhelming release of IL-1, IL-2, IFN- γ , and TNF- α → shock	Toxic shock syndrome: fever, rash, shock; other toxins cause scalded skin syndrome (exfoliative toxin) and food poisoning (heat-stable enterotoxin)
Streptococcus pyogenes	Erythrogenic exotoxin A		Toxic shock-like syndrome: fever, rash, shock; scarlet fever

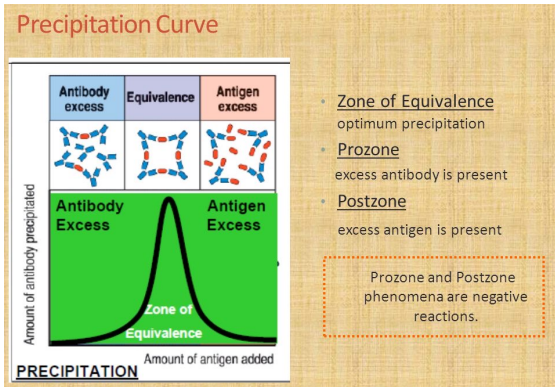
TSST-1 is a superantigen that binds to MHC II and T-cell receptor, resulting in polyclonal T-cell activation and cytokine release. Staphylococcal toxic shock syndrome (TSS)—fever, vomiting, diarrhea, rash, desquamation, shock, end-organ failure. TSS results in \uparrow AST, \uparrow ALT, \uparrow bilirubin. Associated with prolonged use of vaginal tampons or nasal packing. Compare with *Streptococcus pyogenes* TSS (a toxic shock-like syndrome associated with painful skin infection).



- = Presented peptide recognized by TCR
- = Superantigen binding non-specifically to MHC and recognized by TCR

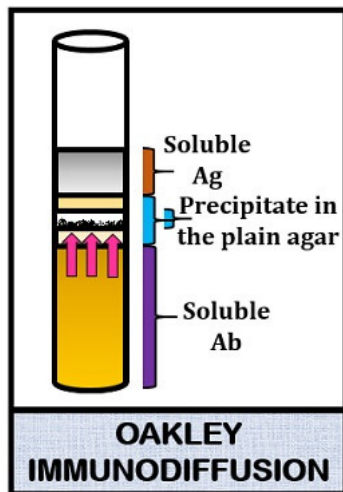
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Negative in 1:20 , 1:40
 Positive in 1:80, 1:160
 = Antibody excess (prozone phenomenon)

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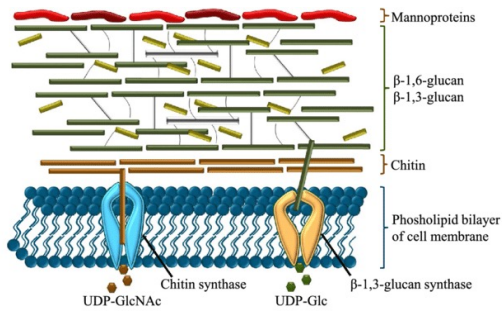




Features	Primary immune response	Secondary immune response
1. Lag period	4-10 days	1-3 days
2. Peak response time	7-10 days	3-5 days
3. Antibody response	Depends on the antigen	More potent than the primary immune response
4. Isotype generate	IgM	IgG
5. Antigens	Both thymus-dependent and independent	Depends only on the thymus
6. Antibody affinity	Lower	Higher

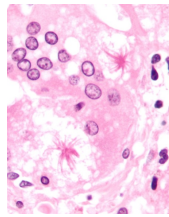
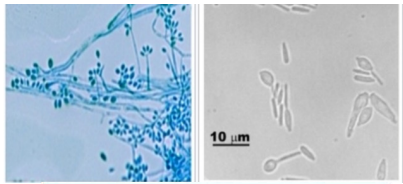
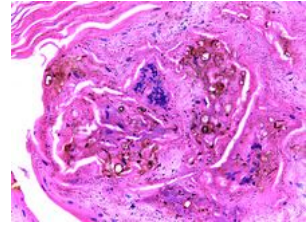
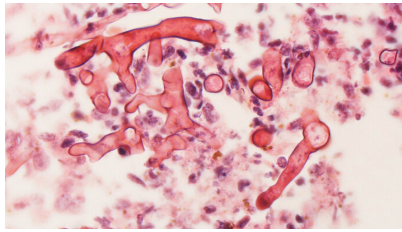
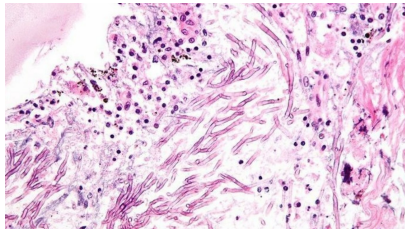
IgG avidity assay helps in differentiating past from primary infection – increases with past infection

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Causes of Sporotrichoid Lesions

Nodular lymphangitis



Organism	Exposure
<i>Sporothrix schenckii</i>	Gardening, soil, splinters, animal bites/scratches
<i>Nocardia brasiliensis</i>	Gardening, soil, splinters
<i>Mycobacterium marinum</i>	Aquarium, fish handling, water exposure
<i>Leishmania brasiliensis</i>	Living/traveling in endemic regions



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Candida albicans

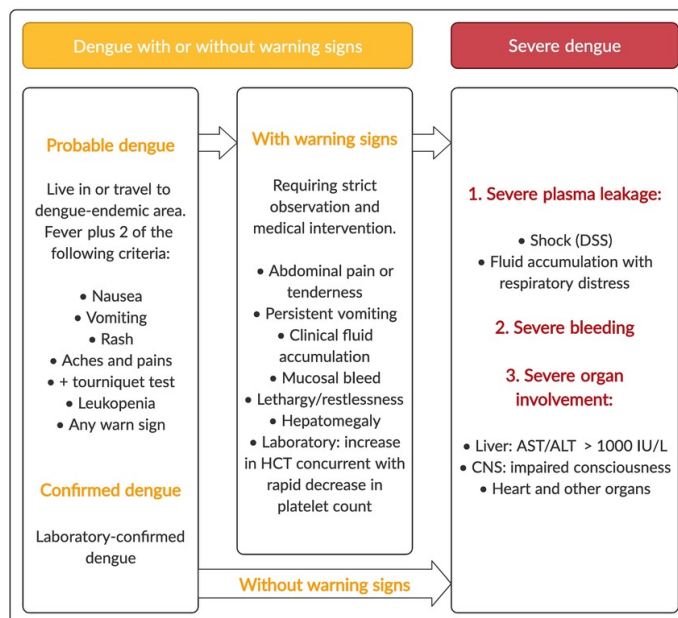
alba = white. Dimorphic; forms pseudohyphae and budding yeasts at 20°C **A**, germ tubes at 37°C **B**.

Systemic or superficial fungal infection. Causes oral **C** and esophageal thrush in immunocompromised (neonates, steroids, diabetes, AIDS), vulvovaginitis (diabetes, use of antibiotics), diaper rash, infective endocarditis (people who inject drugs), disseminated candidiasis (especially in neutropenic patients), chronic mucocutaneous candidiasis.

Treatment: oral fluconazole/topical azoles for vaginal; nystatin, azoles, or, rarely, echinocandins for oral; fluconazole, echinocandins, or amphotericin B for esophageal or systemic disease.



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DF/DHF	Grade	Symptoms	Laboratory
DF*		Fever with two or more of the following signs: headache, retro-orbital pain, myalgia, arthralgia plus positive tourniquet test	Leucopenia occasionally Thrombocytopenia, may be present, no evidence of plasma loss
DHF	I	Above signs plus positive tourniquet test	Thrombocytopenia $\leq 100,000/\mu\text{L}$, hematocrit rise $\geq 20\%$
DHF	II	Above signs plus spontaneous bleeding	Thrombocytopenia $\leq 100,000/\mu\text{L}$, hematocrit rise $\geq 20\%$
DHF	III**	Above signs plus circulatory failure (weak pulse, hypotension, restlessness)	Thrombocytopenia $\leq 100,000/\mu\text{L}$, hematocrit rise $\geq 20\%$
DHF	IV**	Profound shock with undetectable blood pressure and pulse	Thrombocytopenia $\leq 100,000/\mu\text{L}$, hematocrit rise $\geq 20\%$

DF: dengue fever; DHF: dengue hemorrhagic fever; *Modified WHO criteria; **DHF Grade III and IV are also called as dengue shock syndrome (DSS).

Fever (Acute onset of 2-7 days)	
Haemorrhagic manifestations	Evidenced by positive tourniquet test, petechiae, ecchymoses or purpura, or bleeding from mucosa, gastrointestinal tract, injection sites, or other locations.
Thrombocytopenia	Platelet count of $\leq 100,000$ cells/mm ³
Dengue Shock Syndrome (DSS)	
Criteria for DHS (As above) and signs of shock	Tachycardia, cool extremities, delayed capillary refill, weak pulse, lethargy or restlessness (a sign of reduced brain perfusion).
	Pulse pressure ≤ 20 mmHg with increased diastolic pressure, e.g. 100/80 mmHg.
	Hypotension by age, defined as systolic pressure < 80 mmHg for those aged < 5 years or 80 to 90 mmHg for older children and adults.

The first two clinical criteria, plus thrombocytopenia and haemoconcentration or a rising haematocrit, are sufficient to establish a clinical diagnosis of DHF. The presence of liver enlargement in addition to the first two clinical criteria is suggestive of DHF before the onset of plasma leakage. In cases with shock, a high haematocrit and marked thrombocytopenia support the diagnosis of DSS. A low ESR (< 10 mm/first hour) during shock differentiates DSS from septic shock.(6).

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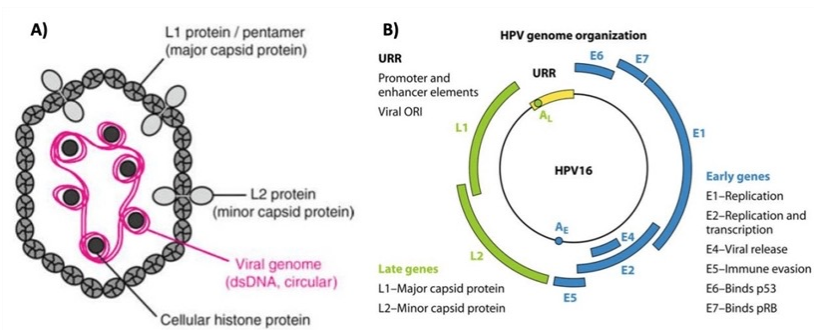


Type of symmetry	Examples
Naked icosahedral	Poliovirus
	Adenovirus
	Hepatitis A virus
Naked helical	Tobacco mosaic virus
Enveloped icosahedral	Herpes virus
	Yellow fever virus
	Rubella virus
Enveloped helical	Corona virus
	Rabies virus
	Influenza virus
	Parainfluenza virus
	Mumps virus
	Measles virus
Complex	Poxvirus

	Non-enveloped viruses	Enveloped viruses
DNA viruses	Parvovirus	Herpes virus
	Adenovirus	Hepatitis B virus
RNA viruses		Poxvirus
	Picornavirus	Hepatitis c virus
	Astrovirus	Rubella virus
	Reovirus	Chikungunya virus
	Calicivirus	Dengue virus
	Hepatitis A and E virus	Japanese encephalitis virus
	Kyasanur Forest disease virus	
	Zika virus	

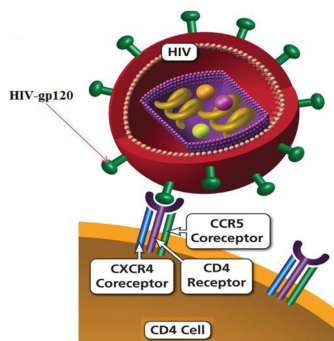
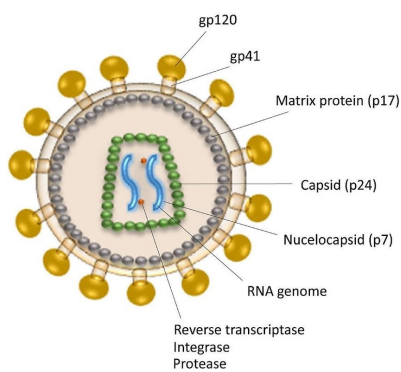
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The natural virus capsid is composed of two proteins, L1 and L2, but vaccines only contain L1.
E6,E7- cancer

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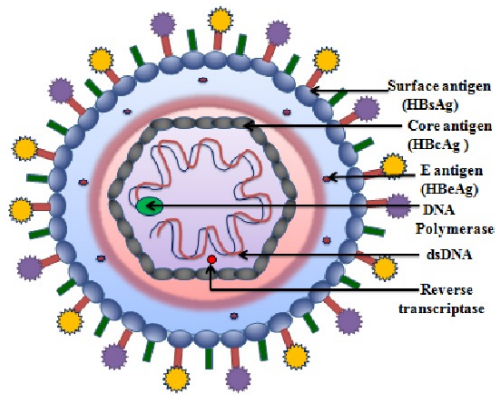


Fostemsavir is a first-in-class HIV attachment inhibitor that works by attaching directly to HIV gp120 and, as a result, blocking HIV from attaching to CD4 on the host cell.

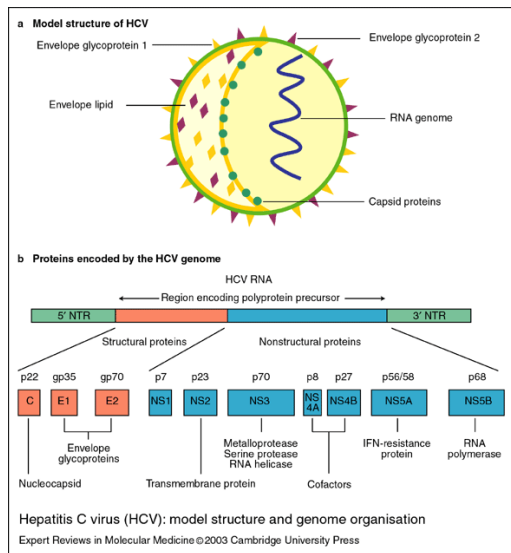
Two inhibitors interfering with the entry process have been approved for clinical use. Maraviroc prevents the interaction of gp120 with the co-receptor CCR5, and T20 (enfuvirtide, Fuzeon) binds to HR1 in gp41 and prevents the formation of the six-helix bundle (9,-11).

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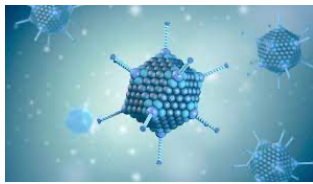
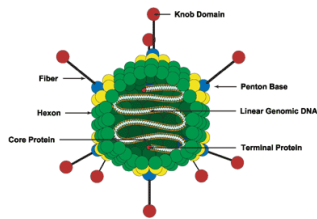
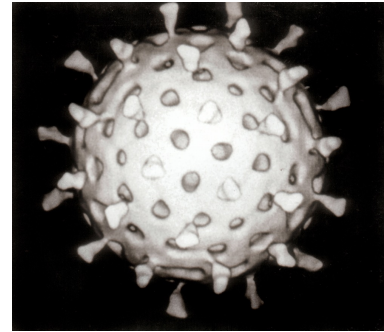
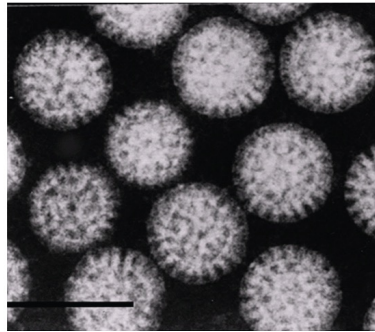
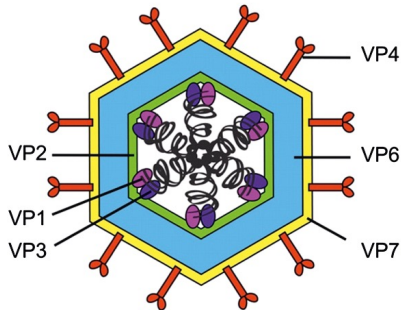


Hepatitis C therapy Chronic HCV infection treated with multidrug therapy that targets specific steps within HCV replication cycle (HCV-encoded proteins). Examples of drugs are provided.

DRUG	MECHANISM	TOXICITY
NS5A inhibitors		
Elbasvir	Inhibits NS5A, a viral phosphoprotein that plays a key role in RNA replication	Headache, diarrhea
Ledipasvir		
Pibrentasvir	Exact mechanism unknown	
Velpatasvir		
NS5B inhibitors		
Sofosbuvir	Inhibits NS5B, an RNA-dependent RNA polymerase acting as a chain terminator. Prevents viral RNA replication	Fatigue, headache
NS3/4A inhibitors		
Glecaprevir	Inhibits NS3/4A, a viral protease, preventing viral replication	Headache, fatigue
Grazoprevir		
Alternative drugs		
Ribavirin	Inhibits synthesis of guanine nucleotides by competitively inhibiting IMP dehydrogenase	Hemolytic anemia, severe teratogen

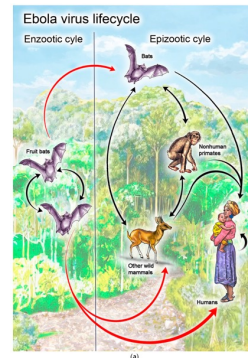
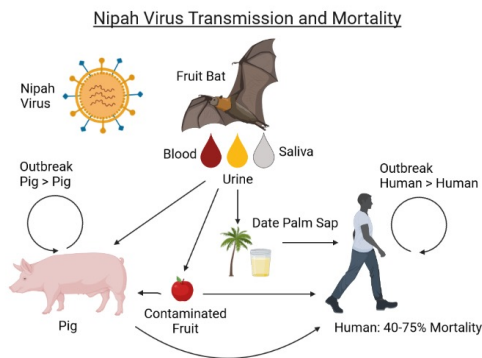
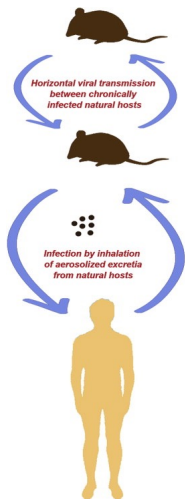
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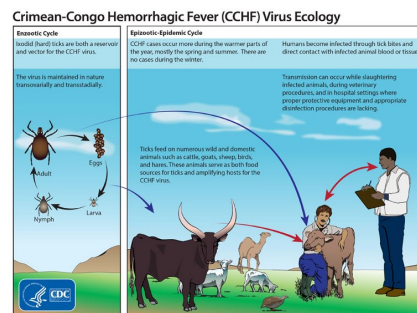
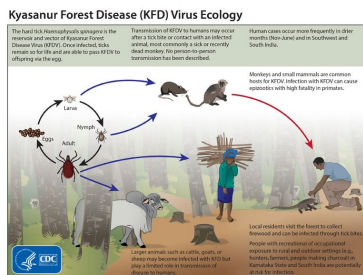
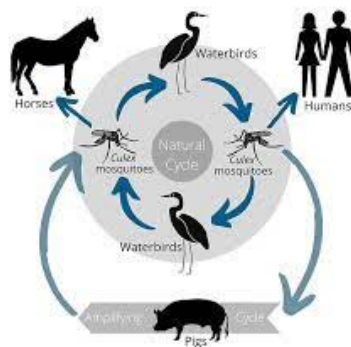
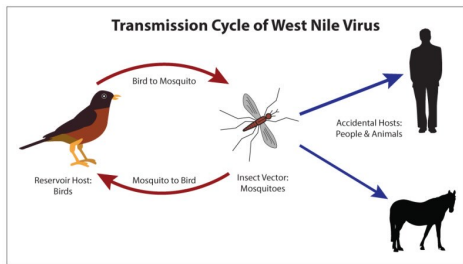
VIRUS	SHAPE
Rabies virus	Bullet shaped
Pox virus	Brick shaped
Tobacco mosaic virus	Rod shaped
Ebola virus	Filamentous
Astro virus	Star shaped
Adenovirus	Space vehicle shaped
Rotavirus	Wheel shaped

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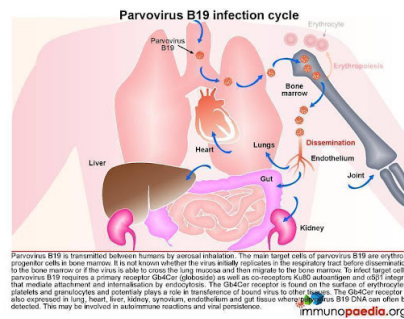
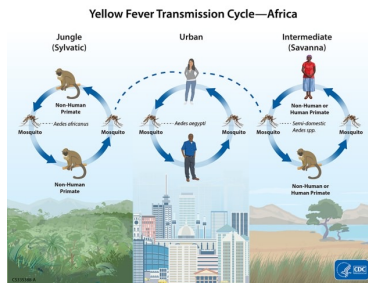
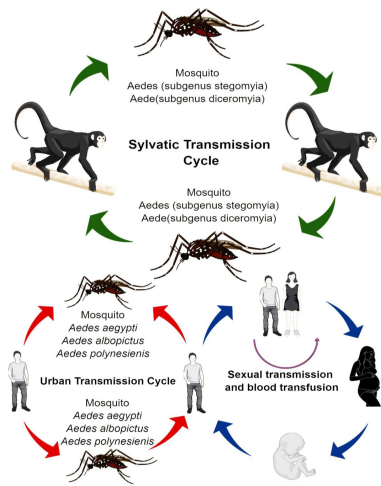


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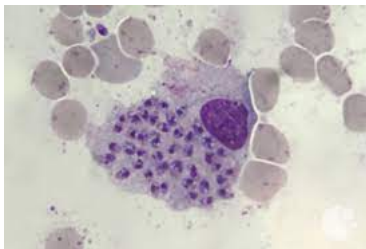


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<i>Cryptosporidium parvum</i>	<i>Cyclospora cayetanensis</i>	<i>Isospora belli</i>
Causes cryptosporidiosis	Causes cyclosporiasis	Causes isosporiasis

Geog. Distribution: worldwide


Parasite takes the shape of oocyst containing sporozoites
Man is infected by ingestion of the sporulated oocyst



4-6µ



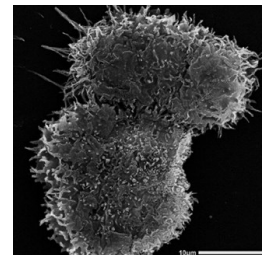
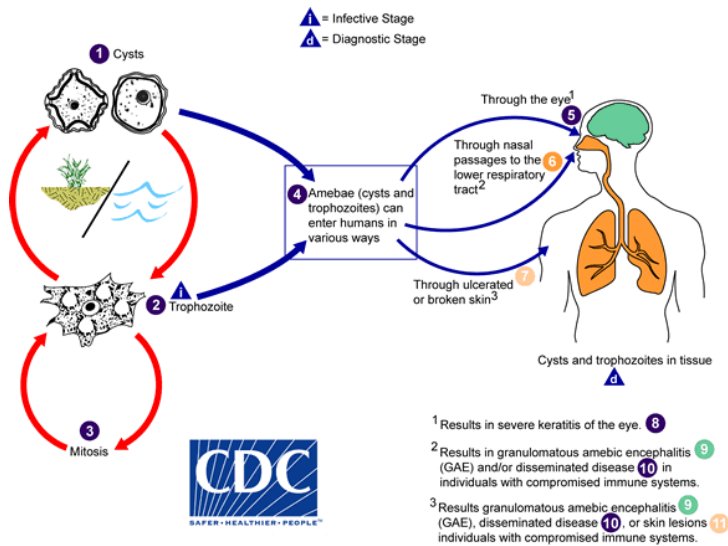
8-10µ



30X12µ

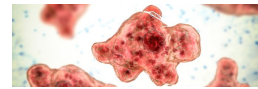
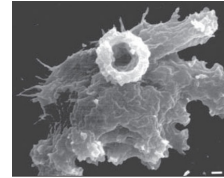
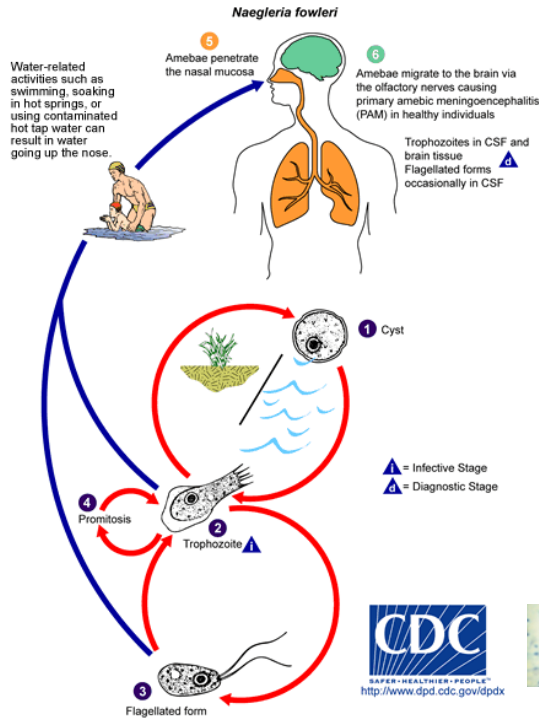
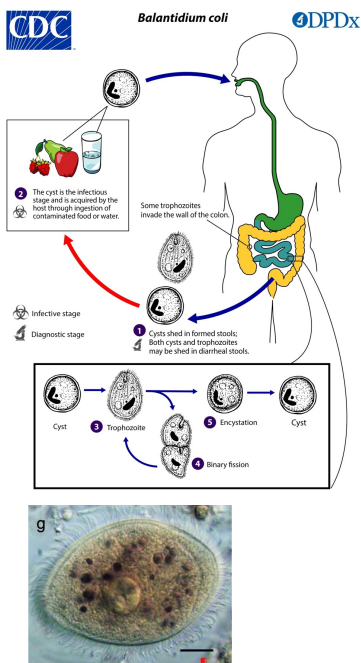


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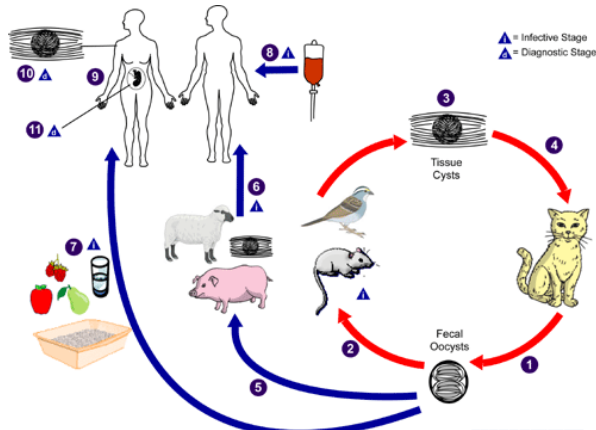
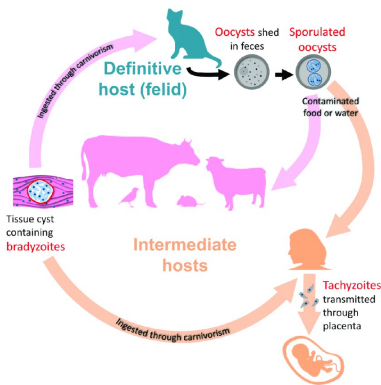


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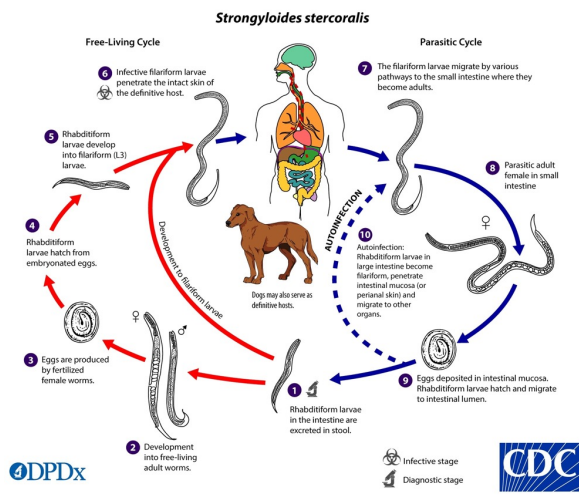




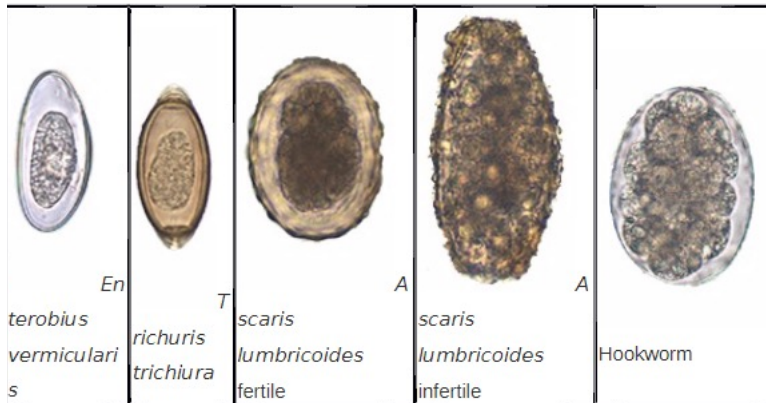
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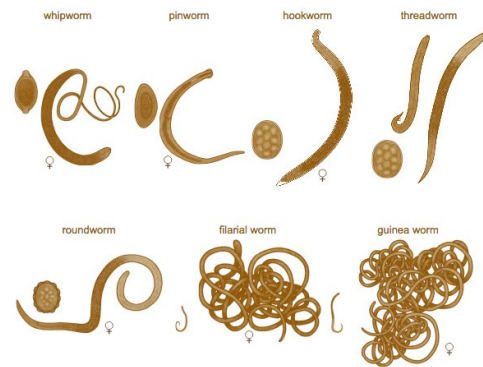
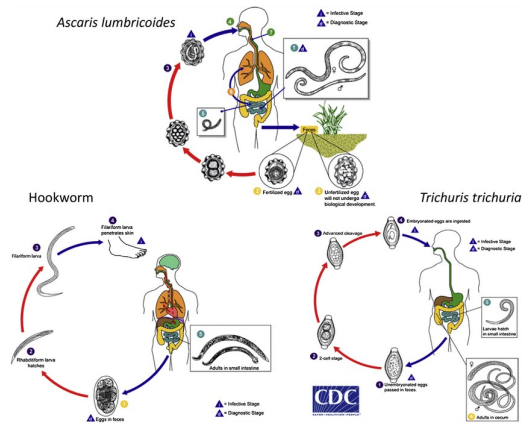
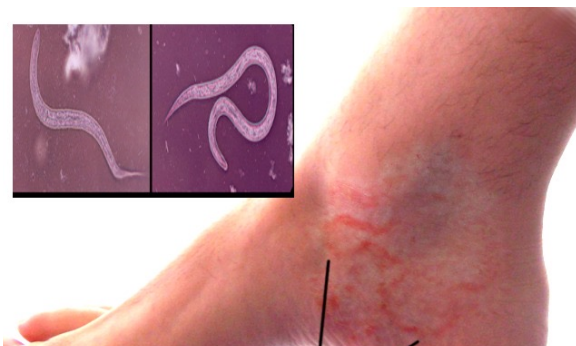
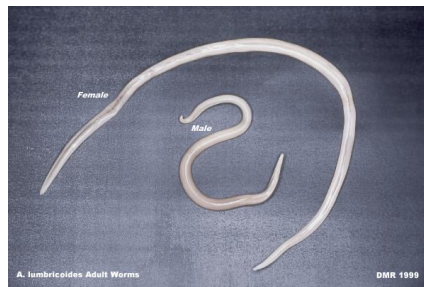


Fig. 9-12-1. *A. lumbricoides*, adult female, gross specimen.

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Disinfectant but not sanitizer	Hydrogen peroxide
Cell wall free bacteria= mollicutes	Mycoplasma, ureaplasma
Genetic reassortment similar to influenza	Rotavirus
Endemic in India	CCHF , west nile (not yellow/ ebola)
T:B cells	3:1
COVID cannot be detected by	Southern blotting.

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