Parasitic Infection

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Outline:

- Introduction
- Characteristic of parasitic infection
- Cell and tissue alterations from parasitic infection
- Summary / Q&A
Objectives

1. Understand the biology and importance of parasite infectious diseases

2. Explain the cell and tissue defects from parasitic infection

3. Explain the host immune response to parasitic infection
Pathogens

- Bacteria
- Fungus
- Virus
- Parasite
- Prion
How can microorganisms cause disease?

* Infective agents establish infection and damage tissues in several ways

- Contact or enter host cells/tissues and directly cause cell injury/death (interfere cellular mechanism)
- Release enzymes or toxins and damage host cells/tissues
- Over-induce of host cellular responses and cause additional tissue damage
Parasitic infection

“condition that parasites successfully invade the hosts for their food and residence”

The infection usually causes the host defects/ parasitic diseases.

“pathogenic parasites”

Intracellular & Extracellular parasites

Cell/Tissue destruction & Inflammation/Hypersensitivity
Classification of parasites

- **Protozoa**
  - flagellates
  - amoeba
  - sporozoa
  - ciliates

- **Helminths**
  - nematodes
  - trematodes
  - cestodes

- **Arthropods** (e.g. ticks, mites, lice, flea)

**Major infective organs**

1. Intestinal
2. Blood and tissue
3. Sexually transmitted
Ways to get infections

- **animals** (zoonotic) – Cryptosporidium, Trichinella, ...

- **insect** – Plasmodium*, Trypanosoma*, Leishmania*, Babesia*, ...

- **food** – Giardia, Entamoeba, Cyclospora, Toxoplasma*, Cryptosporidium, Trichinella, Taenia, ...

- **water** – Entamoeba, Giardia, Cryptosporidium, Schistosoma, Dracunculus, ...

*blood-borne diseases*
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<th>Type of damage</th>
<th>Mechanism</th>
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<td>Morphological</td>
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<td>Tissue</td>
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<td>Cytokine dysregulation</td>
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<td>Psychosis</td>
<td>Rheumatic fever</td>
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<td>Organism</td>
<td>Behavioural</td>
<td>Loss of predator fear, hydrophobia</td>
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EBV, Epstein-Barr virus; Ig, immunoglobulin.
Pathology of parasitic infection

1. cell & tissue damage directly from invasion, migration, or secretion
2. host immune responses / inflammation
3. site of infection
4. symptomatic / asymptomatic infection

Factors:
- complicate life cycle and developmental stages
- parasite load / co-infection
- host/parasite co-evolution (natural selection)
Pathogenesis

Direct & Indirect

Factors

- Many infections are asymptomatic
- Site of infection
- Worm load
- Immune response
Directly damaged by helminths

Cysts of the tapeworm *Echinococcus* develop in internal organs, grow to large size, and cause damage to tissues by mechanical pressure (hydatid disease).

Physical blockage of the intestine by large nematodes (*Ascaris*) or tapeworms (*Taenia* or *Dipylidium*).
Migratory tract

Masson’s Trichrome
Damaged by immune responses

**Origin**
- Adult schistosomes in blood vessels around small intestine

**Stimulus**
- Eggs laid by female
- Eggs carried in blood vessels and trapped in liver

**Response**
- Hypersensitivity to antigens of larva inside egg cause formation of granuloma.
- Liver sinusoids become blocked, impeding blood flow

**Pathology**
- Fibrosis of liver
  - Raised portal pressure
  - Perihepatic shunting of blood
  - Hepatomegaly
  - Spleenomegaly
  - Formation of varices
Eosinophilia, cardinal sign of parasitic infection
Granuloma
Infectivity and Virulence

**PROTOZOA**
- Several ways of infection
- Multiply quickly, rapid onset of symptoms
- Intracellular or latent infections, immune evasion

**HELMINTH**
- Mostly acquired ingestion of egg or larva
- Disease and symptoms are associated with worm burden
- Larva migrans or zoonotic infections
Parasites & Infected organs
Intestinal protozoan infections

**Giardia lamblia** (*G. duodenalis* or *G. intestinalis*; intestinal flagellate)

**Site:** small intestine

**Mechanism:** ingest cysts contaminated in water or food

**Parasite:** common pathogen found in duodenum or jejunum of human as trophozoite (heart-shape + flagella) that can attach to the villi or the cyst forms (two or four nuclei) that can pass through the colon and stool

**Pathology:** “Giardiasis”, usually weak pathogenesis / irritation and low-grade inflammation of duodenal/jejunal mucosa / acute or chronic diarrhea associated with crypt hypertrophy, villous atrophy or flattening and epithelial cell damage / stools may be watery, semisolid, greasy, and foul smelling / weakness, weight loss, abdominal clamp, distention, and flatulence for long periods

**Diagnosis:** stool exam., immunological techniques
Duodenum
crypt hyperplasia, villous atrophy
Entamoeba histolytica
(intestinal and tissue amoeba)

**Site:** only in lumen of colon or other tissues e.g. liver,…

**Mechanism:** ingest cysts contaminated in water or food - oral/anal

**Parasite:** cyst contain a glycogen vacuole and chromatoid bodies found in colon and mushy feces / amoeboid trophozoite is the form that present in tissues

**Pathology:** “Amebiasis”, worldwide with 100,000 deaths/year /
trophozoite can invade epithelium and form discrete ulcers with a pinhead-sized center and raised edges → flask-shaped ulcer / mucus, necrotic cells, and amoeba / trophozoite can penetrate muscle layers and serosa and perforate into peritoneal cavity / inflammation, granulomatous tumor-like mass formed on intestinal wall / diarrhea, nausea, vomiting, cramp, loss of appetite, weight loss

**Diagnosis:** stool exam., immunological techniques
Flask shaped ulcers - Base in submucosa and small opening on the mucosal surface
**Cryptosporidium** (intestinal sporozoa)

**Site:** small intestine and tissues e.g. lung

**Mechanism:** ingest oocysts contaminated in water or food

**Parasite:** *C. hominis* infect the immunocompromised person

**Pathology:** “Cryptosporidiosis”, attach to the surface of villi of lower small bowel / mild gastroenteritis / watery diarrhea / severe, intractable diarrhea in AIDS

**Diagnosis:** stool exam., acid-fast staining, immunological techniques

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**Cyclospora** (intestinal sporozoa)

**Parasite:** oocysts take days or weeks to become infectious

**Pathology:** “Cyclosporiasis”, shortening of villi / infiltration of inflammatory cells / diarrhea / anorexia / fatigue / weight loss / often prolonged but ultimately self-limiting
Meronts containing crescent-shaped merozoites

Probable unicellular gametocytes
Blood and tissue protozoan infections

*Trypanosoma brucei* (blood flagellate)

**Site:** blood, lymph

**Mechanism:** tsetse fly bite and release trypomastigotes

**Parasite:** trypomastigotes found in bloodstream with elongated bodies supporting a longitudinal undulating membrane

**Pathology:** “Sleeping sickness”, swelling at the site of inoculation, spread to lymph node, bloodstream, and CNS / lassitude, inability to eat, tissue wasting, unconsciousness, death

**Diagnosis:** blood smear, CSF, lymph node aspirate, serology test
**Trypanosoma cruzi** (blood flagellate)

**Site:** intracellular amastigote in tissue e.g. heart muscle, liver, brain (parasympathetic ganglia)

**Mechanism:** kissing bug feces rubbed into bite or eyes / transfusion / transplacenta

**Parasite:** trypomastigotes found in bloodstream / amastigote as rounded intracellular stage

**Pathology:** “Chagas disease”, subcutaneous inflammation nodule or chagoma, unilateral swelling of eyelids / fever, acute regional lymphadenitis / interstitial myocarditis / chronic infection destroy nerve plexuses in alimentary tract walls leads to megaesophagus, megacolon (esp. in Brazilian, but not in Colombian and Central american)

**Diagnosis:** blood smear (extracellular), tissue biopsy (intracellular)
**Leishmania** *(blood flagellates)*

**Site:** skin; rolled edge ulceration

**Mechanism:** sandfly injects promastigotes; phagocytosis of amastigote by macrophage or monocyte

**Parasite:** parasite multiply in cytoplasm of the cell → cell burst → release parasites to phagocytose again

**Pathology:** “Leishmaniasis”, 1) **cutaneous** – ulcer / blisters / hypersensitivity / granulomatous scarring reaction  
2) **mucocutaneous or nasopharyngeal** – nasal septum damage / blockage of trachea / respiratory infection 3) **visceral** – spleen hyperplasia

**Diagnosis:** skin biopsy, PCR, intradermal lieshmanin skin test
Histology of cutaneous leishmaniasis (H&E stain): a) Diffuse chronic inflammatory cell infiltrate in dermis with multiple non-caseating granulomata (×40); b) Tuberculoid-type granuloma with central histiocytes and peripheral inflammatory cells (×100); c) Leishman-Donovan bodies (arrows) within cytoplasm of epithelioid histiocytes (×600); d) Langhans type giant cells (arrows) within a granuloma (×600).
**Acanthamoeba castellanii**

**Site:** brain, spinal cord, eye

**Mechanism:** parasites from fresh water resource penetrate through skin or eye / contaminated contact-lens

**Parasite:** parasites enter the body invade into the brain

**Pathology:** granulomatous amebic encephalitis (GAE) / keratitis

**Diagnosis:** CSF

**Naegleria fowleri**

**Mechanism:** nasal membrane $\rightarrow$ cribriform plate of ethmoid bone $\rightarrow$ brain tissue

**Pathology:** cerebrum/cerebellum basilar hemorrhage and damage / primary amebic meningoencephalitis (PAM) or
**Plasmodium** (blood sporozoa)

**Site:** intracellular RBC, liver cells

**Mechanism:** female Anopheles mosquito bite and release sporozoites from mosq. salivary gland

**Parasite:** *P. falciparum, P. vivax, P. ovale, P. malariae, P. knowlesi*; sporozoites enter liver cells → merozoites → enter blood stream and invade RBC

**Pathology:** incubation period between 9-30 days; fever to death; chill from rupture of iRBC; Pf adhere to endothelial lining of blood vessel → cerebral malaria; Pv dormancy stage - hypnozoite

**Diagnosis:** blood smear
**Babesia microti** (blood sporozoa)

**Site:** intracellular RBC

**Mechanism:** tick bite, blood transfusion

**Parasite:** morphology is similar to Plasmodium

**Pathology:** incubation period between 7-10 days; asymptomatic, malaise, anorexia, nausea, fatigue, fever, sweats, myalgia, arthralgia, depression; more severe in senile, splenectomized, AIDS persons

**Diagnosis:** blood smear
**Toxoplasma gondii** (tissue sporozoa)

**Site:** intracellular in CNS, bone marrow

**Mechanism:** ingest the parasites in undercooked meat, oocysts from cat feces, blood transfusion

**Parasite:** normal final host is cat, human is intermediate host; parasites develop asexual cycle in various cell types especially macrophage; acute stage disease-tachyzoites (rapid multiplying cells); chronic stage disease-bradyzoites (slow multiplying cells)

**Pathology:** most infections are asymptomatic, fatal infection may develop in AIDS persons; retinitis or chorioretinitis, encephalitis, pneumonitis can occur in immunosuppressive individuals

**Diagnosis:** serology test
Sexually transmitted protozoan infections

*Trichomonas vaginalis* (genito-urinary flagellate)

**Site:** vagina

**Mechanism:** person to person through sexual intercourse

**Parasite:** exists only as a trophozoite (4 free flagella that arise from single stalk and a single fifth flagellum that form undulating membrane), no cyst stage

**Pathology:** most infections are asymptomatic or mild / female infection normally limited to vulva, vagina, and cervix, does not usually extend to uterus / mucosa inflammation, erosion and cover with a frothy yellow or cream-colored discharge / male may be infected to prostate, seminal vesicles, and urethra

**Diagnosis:** microscopic exam of discharge, urine, tissue scraping
Trichomonas vaginalis
Intestinal helminthic infections

Enterobius vermicularis (pinworm – intestinal nematode)

Site:  lumen of cecum and colon

Mechanism:  ingest the eggs; anal-oral behavior

Parasite:  found worldwide, common in temperate than tropical climates; most common helminthic infection in mostly children in US

Pathology:  perianal pruritus - hypersensitivity reaction to the eggs that laid around the perianal region; irritability and fatigue from loss of sleep

Diagnosis:  Scotch Tape test, stool exam
**Trichuris trichiura**  
(whipworm – intestinal nematode)

**Site:** cecum and colon

**Mechanism:** ingest the eggs from fecally contaminated food

**Parasite:** the eggs require 3 weeks of incubation to become infective, larva hatch in small intestine then mature and migrate to colon

**Pathology:** anterior end of worm lodges in the mucosa → hemorrhage, mucosal cell destruction, infiltration of eosinophils and plasma cells; asymptomatic to abdominal pain, distension, diarrhea, cramps, rectal prolapse

**Diagnosis:** stool exam
Ascaris lumbricoides
(roundworm – intestinal nematode)

**Site:** small intestine; larva through lung

**Mechanism:** ingest the eggs from fecally contaminated food

**Parasite:** larva hatches in duodenum, penetrate through mucosa, circulatory system, lodge in the lung capillaries, penetrate the alveoli, migrate to trachea and pharynx, larvae are swallowed and return to intestine and become adults

**Pathology:** high number of parasites cause obstruction of bowel, bile and pancreatic duct; inflammation in lung; reinfeciton causes bronchial spasm, mucus production, cough, eosinophilia

**Diagnosis:** stool exam
**Ancylostoma duodenale and Necator americanus**  
(hookworm – intestinal nematode)

**Site:** small intestine; larva through skin, lung

**Mechanism:** larvae in soil penetrate skin

**Parasite:** migration is similar to *Ascaris*; adult worms attach to intestinal villi with buccal teeth

**Pathology:** worms feed on blood and tissue; severe anemia, iron deficiency, diarrhea

**Diagnosis:** stool exam
Ancylostoma in intestine
*Strongyloides stercoralis*

(threadworm – intestinal & tissue nematode)

**Site:** small intestine; larva through skin, lung

**Mechanism:** larvae in soil penetrate skin

**Parasite:** worms lay eggs within the intestine, larvae hatch from eggs and pass through the feces; “autoreinfection”; parasites penetrate the intestine, migrate through circulatory system, enter lung, heart

**Pathology:** severe diarrhea, abdominal pain, gastrointestinal bleeding, coughing, wheezing, hemoptysis

**Diagnosis:** stool exam, broncheal lavage
Figure 3: High power view showing details of intramucosal eggs and larvae (H and E x 400)
Figure 4: Section of adult worm in gastric biopsy (H and E x 400)
Trichinella spiralis (intestinal & tissue nematode)

Site: adult in small intestine (1-4 months); larva encystes in muscular tissue

Mechanism: eat undercooked meats

Parasite: adult worms in small intestine mate and produce larvae, then larvae penetrate intestine, circulate in the blood, encyst in muscle

Pathology: fever, cough, eosinophilia, calcification, myalgia, weakness

Diagnosis: serology test, muscle biopsy
**Fasciolopsis buski** (giant intestinal trematode)

**Site:** small intestine

**Mechanism:** eat the encysted metacercariae on aquatic vegetation

**Parasite:** found in east and south asia

**Pathology:** asymptomatic to ulceration, intestinal wall abscess, diarrhea, abdominal pain, intestinal obstruction

**Diagnosis:** stool exam
**Taenia saginata and T. solium**
(tapeworm – intestinal and tissue cestode)

**Site:** small intestine

**Mechanism:** eat cysticerci encysted in undercooked beef or pork

**Parasite:** adult worms can reach lengths of several meters

**Pathology:** asymptomatic to mild e.g. diarrhea, abdominal pain; medical significant is that human can be the intermediate host

**Diagnosis:** stool exam (tapeworm segments)
"Cysticercosis" (T. solium larva)

**Site:** cysticerci in skin, liver, lung, kidney, muscle, eye, brain

**Mechanism:** as intermediate host by eat eggs via human fecal-oral route

**Parasite:** similar to the pig, parasites encyst in various human tissues

**Pathology:** associated with the organ involved e.g. ophthalmocysticercosis, neurocysticercosis

**Diagnosis:** serology test, CT scans, MRI, x-rays
**Diphyllobothrium latum**
(broad fish tapeworm – intestinal cestode)

**Site:** small intestine

**Mechanism:** eat cysticerci encysted in undercooked fish

**Parasite:** worm rapidly grow, can be more than 10 meters in length, worm segment can be released > 1 million eggs per day

**Pathology:** abdominal discomfort, loss of appetite, weight loss, unusual capacity to absorb Vit. B\textsubscript{12}

**Diagnosis:** stool exam (eggs & tapeworm segments)
**Hymenolepis nana**
(dwarf tapeworm – intestinal cestode)

**Site:** small intestine

**Mechanism:** eat eggs from feces; autoreinfection via fecal-oral route

**Parasite:** one of the most common tapeworm infection worldwide, mostly in children

**Pathology:** minor intestinal disturbance

**Diagnosis:** stool exam (eggs & tapeworm segments)
Blood and tissue helminthic infections

*Wuchereria bancrofti* and *Brugia malayi*  
(lymphatic filariasis – tissue nematode)

**Site:** adult worms in lymph nodes, lymphatic ducts

**Mechanism:** mosquito bite transmits larvae

**Parasite:** found in tropical and subtropical climates, adult worms are found in lymphatic vessels and the female release the larvae, larvae called microfilariae enter the peripheral blood

**Pathology:** inflammation, fibrotic reactions, lymphangitis, fever, painful lymph node, edema,

**Diagnosis:** blood smear for microfilariae
**Onchocerca volvulus**  
(river blindness – tissue nematode)

**Site:** adult worms in skin nodules

**Mechanism:** black fly bites and transmits larvae

**Parasite:** prevalence is >17 million, among these 270K are blind / larvae develop into adults in subcutaneous tissues, encapsulated to form nodule (onchocercoma) / microfilariae can migrate within skin

**Pathology:** tissue damage from larvae releasing, migrating of microfilariae in the interstitial fluid e.g. vitreous humor caused visual loss, visual clouding, photophobia, retinal damage, incurable blindness

**Diagnosis:** skin snips
**Dracunculus medinensis**  
(guinea worm – tissue nematode)

**Site:** adult worms in subcutaneous of lower legs, ankles, feet

**Mechanism:** drink water contaminated with infected copepods

**Parasite:** after a year of systemic wandering in the body, the worms become mature and mate

**Pathology:** broad range of pathology / female adults travel to the skin usually the lower legs → blister formation / secondary bacterial infection / severe infection → gangrene, anaphylaxis

**Diagnosis:** worm in skin blister
“Larva migrans” (zoonotic larval nematode)

**Parasite:** parasites of animal / humans are dead-end hosts

**Pathology:** larvae degenerate, induce immune responses to dead or dying larvae, eosinophilia is a common feature

*cutaneous larva migrans (CLM):* dog hookworm larvae migrate in the epithelial layer of skin → red, itchy tracts on the skin / erythema, and papules at the site of entry / serpiginous tracts of red inflammation

*visceral larva migrans (VLM):* whaleworm larvae in herring, salmon, rockfish can invade gastric mucosa or intestinal tissue → extreme abdominal pain that mimics appendicitis or small bowel obstruction / eosinophilic granuloma

*ocular larva migrans (OLM):* dog or racoon round worm larvae migrate to various tissues / can lead to VLM, OLM, or NLM
**Clonorchis sinensis and Fasciola sp.**
(liver flukes – tissue trematode)

**Site:** adult worms in bile duct, after migration through liver parenchyma

**Mechanism:** eat metacercariae in undercooked fish (Clonorchis) or water vegetation (Fasciola)

**Parasite:** Clonorchis-Chinese liver fluke / Fasciola-Sheep liver fluke

**Pathology:** asymptomatic, liver fibrosis, bile duct fibrosis and hyperplasia / fever, chill, epigastric pain, eosinophilia, cholangitis, portal fibrosis, jaundice, biliary obstruction, cirrhosis

**Diagnosis:** stool exam.
Opisthorchis viverrini
(asian liver flukes – tissue trematode)

**Site:** adult worms in bile duct, after migration through liver parenchyma

**Mechanism:** eat metacercariae in undercooked fish (Clonorchis)

**Parasite:** major liver fluke in north-east of Thailand

**Pathology:** asymptomatic, liver fibrosis, bile duct fibrosis and hyperplasia / fever, chill, epigastric pain, eosinophilia, cholangitis, portal fibrosis, jaundice, biliary obstruction, cirrhosis / “cholangiocarcinoma”

**Diagnosis:** stool exam.
Paragonimus westermani
(lung flukes – tissue trematode)

**Site:** adult worm in lung

**Mechanism:** eat metacercariae in raw crabs and other freshwater crustaceans

**Parasite:** larvae excyst in gut and migrate to lung, where they become encapsulated, eggs are released and move to trachea, pharynx, then swallowed and pass through the feces

**Pathology:** eggs induce inflammation and forming granulomas, adults form nodules within lung tissue / pulmonary tuberculosis-paragonimiasis / worms can be found ectopic sites (brain, liver, intestinal wall)

**Diagnosis:** stool exam
**Schistosoma mansoni, S. japonicum, S. haematobium** (blood flukes)

**Site:** adult worm in venous vessels of liver and intestine

**Mechanism:** cercariae penetrate skin

**Parasite:** 200 million people are infected worldwide / cercariae penetrate epidermis transform into schistosomules, enter the peripheral circulation and become adults in the hepatoportal system or venous plexus surround the bladder

**Pathology:** significantly related to eggs → granulomas, fibrosis, portal hypertension / *S. haematobium* → urethral pain, dysuria, hematuria, bladder obstruction, secondary bacterial infection

**Diagnosis:** stool exam / urine
**Echinococcus granulosus** (hydatid cyst)

**Site:** hydatid cysts in liver, spleen, lung, peritoneum, brain

**Mechanism:** eggs from feces, contact with canine animals

**Parasite:** three segmented tapeworm found in dog and other canines / similar to pork tapeworm, larvae penetrate gut and migrate to various tissues e.g. liver, spleen, muscle, brain / larvae develop into hydatid cyst (fluid-filled cyst)

**Pathology:** liver is the most common site / atrophy, portal hypertension, cirrhosis

**Diagnosis:** serology, CT scans, MRI, x-rays
Ectoparasites

Tick

Mite

Louse

Flea
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<td>Louse feces</td>
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<td>Brill-Zinsser disease</td>
<td><em>R. prowazekii</em></td>
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<td><em>Coxiella burnetii</em></td>
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<td>Ehrlichiosis</td>
<td><em>Ehrlichia sennetsu, E. canis</em></td>
<td>Tick bite</td>
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Summary

- intestinal protozoa
- blood and tissue protozoa
- intestinal helminth
- blood and tissue helminth
References

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   6th Edition, Raphael Rubin, David Strayer, Emanuel Rubin. Lippincott Williams & Wilkins

2. Jawetz, Melnick, & Adelberg’s Medical Microbiology (2013)

**Station 1** – Amoebic colitis

**Station 2** – Malaria

**Station 3** – Taeniasis

**Station 4** – Opisthorchiasis, Cholangiocarcinoma

protozoa

helminth