PARASITOLOGY: INTRODUCTION TO PARASITOLOGY

Definition of Parasitism

- A symbiotic relationship where the parasite depends on the host for survival at the host's expense.
- Possible Outcomes:
  - Parasite fails to establish
  - Host eliminates the infection
  - Host damaged by its own overactive immune response
  - Host death
- **Parasitology**: Study of organisms that depend on other living organisms.
- **Medical Parasitology**: Focuses on human parasites and their medical significance.
- **Tropical Medicine:** Branch dealing with diseases endemic in tropical regions (mostly parasitic).

A. Anatomic Physiology of Parasitic Infection

- Major Areas Affected:
  - Gastrointestinal and Urogenital Tracts
  - Blood and Tissue
  - Liver, Lung, and other major organs
  - Miscellaneous: CSF, eye, skin, extremities

A1. Host-Parasite Relationship (Adaptation)

- Locomotory and digestive organs
  - Cilia, microvilli, spine
  - Hooks and suckers
- *Streamlining*: Parasite cannot synthesize certain components; must obtain from host.
- Specialized Mechanisms:
  - *E. histolytica* → cysteine proteinases (digest cellular materials, degrade epithelial basement membrane)
  - *Schistosoma* cercariae → penetration glands
  - Cestode embryos → hooklets

A2. Effects of the Parasite on the Host

Effect	Description	Example
Interference with Vital Processes	Secretory/excretory products (enzymes) interfere with host functions	*E. histolytica* cysteine proteinases facilitate tissue invasion
Invasion & Destruction of Tissue	Direct damage by entering and rupturing cells	*Plasmodium* spp. ruptures RBCs; *S. japonicum* eggs cause granuloma → portal hypertension
Mechanical Injury & Obstruction	Physical presence causes trauma	Hookworms destroy intestinal villi; *Ascaris* masses cause obstruction
Nutrient Deprivation	Compete for essential substances	Hookworms → iron deficiency anemia; *D. latum* → Vitamin B12 deficiency → megaloblastic anemia

A3. Effects of the Host on the Parasite

Factor	Effect	Example
Genetic Makeup	Determines susceptibility	**Sickle cell trait** → protects against *P. falciparum* **Duffy Blood Factor** → susceptibility to *P. vivax*
Nutritional Status	Diet affects parasite environment	**High protein** → unsuitable for intestinal protozoa **Low protein** → amebiasis **High carbs** → tapeworms
Immune Response	Host defense mechanisms	

B. Immunology of Parasitic Infections

Parasite successfully establishes in the host
Parasite fails to established in the host become
Parasite becomes established, and the host eliminates the infection
Parasite becomes established, and the host: • overcomes but fails in trying to eliminate the organism. • the host becomes damaged itself
Parasite kills the host

B1. Host-Parasite Interactions (Natural Barriers)

- **Physical Barriers:** Skin, mucous membranes, tight junctions (*Schistosoma*)
- **Chemical Barriers:**
  - Low pH of vaginal secretions (*T. vaginalis*)
  - Gastric juices (HCl), intestinal secretions (*G. lamblia*)

B2. Protection of Parasites

- Thick egg shell (*Ascaris, Trichuris, Taenia*)
- Cystic wall of protozoa (*Entamoeba, Giardia*)

B3. Protection of the Host

- **Chemical Contents of Body Fluids:**
  - Lipase in breast milk (acts against *G. lamblia* in vitro)
  - Lysozyme in tears/saliva + IgA
- **Physiologic Functions:**
  - Peristalsis, motion of cilia
  - Coughing (expels *A. lumbricoides, P. westermani*)
  - Flushing action of urine (reduces *T. vaginalis*)

B4. Second Host Defense

- Sensing parasites via **Pathogen-Associated Molecular Patterns (PAMPs)** or **Pattern Recognition Receptors (PRRs)**

B5. Host Immune Response

- **Innate:** Phagocytosis by macrophages and dendritic cells
- **Acquired:** Adaptive immunity

B6. Parasite Evasion Mechanisms

Mechanism	Description	Examples
Resistance	Naturally resistant to innate immunity	*T. brucei* resists APOL1

Immune Suppression	Reduces effectiveness of immune cells	*Plasmodium* → lowers phagocytosis; *T. brucei* glycoproteins impair B/T cells; *E. histolytica* suppresses macrophage respiratory burst
Antigenic Variation	Changes surface proteins to evade antibodies	*T. brucei* (VSG); *P. falciparum* (MSA, RESA)
Mimicry	Masks itself with host molecules	*E. granulosus* carries P blood group antigens; *Schistosoma* acquires host antigens
Intracellular Sequestration	Hides in cells inaccessible to immune system	*T. cruzi, Leishmania, T. gondii*; *P. falciparum* attaches to endothelial cells via "knobs," avoiding splenic filtration

B7. Adverse Effects of Immune Response (Hypersensitivity)

- **Type I:** Immediate-type
- **Type II:** Immune complex formation
- **Type III:** Antibody-mediated cytotoxic reactions
- **Type IV:** Delayed-type

C. Signs and Symptoms

- Diarrhea, Fever, Chills, Abdominal pain
- Anemia, Elephantiasis, Blindness, etc.

D. Treatment

- Antiparasitic medications
- Dietary changes, Vitamin supplements
- Fluid replacement, Blood transfusion
- Bed rest

E. Vaccination

- **Prophylactic (Anti-Infection) – MOST DESIRABLE:** Prevents transmission and disease
- **Transmission-Blocking:** Targets stages infective to vector
- **Anti-Pathology:** Kills stages responsible for disease

PARASITOLOGY: INTRODUCTION TO MEDICAL PARASITOLOGY

1. BIOLOGICAL RELATIONSHIPS

Relationship	Definition	Example
Commensalism	One benefits, other unaffected	*Entamoeba coli* in human gut
Mutualism	Both benefit	Termites & flagellates (cellulase production)
Parasitism	One benefits at host's expense	*Entamoeba histolytica* (causes dysentery)

2. CLASSIFICATION OF PARASITES

Type	Definition
Endoparasite	Lives inside the host (infection)
Ectoparasite	Lives outside the host (infestation)
Obligate Parasite	Cannot live without host (tapeworms)
Facultative Parasite	Can be free-living or parasitic when needed
Accidental/Incidental	Found in host where it doesn't normally live
Erratic	Found in unusual organ
Permanent	Entire life cycle on/in host
Temporary	Only short contact with host
Spurious	Free-living organism passing through gut (no infection)

3. CLASSIFICATION OF HOSTS

Host Type	Definition	Example
Definitive/Final	Parasite reaches sexual maturity	Humans in taeniasis
Intermediate	Harbors asexual/larval stage	Pigs/cattle for *Taenia*; snails for *Schistosoma*
Paratenic	Parasite lives but doesn't develop; transfers infection	Wild boar for *Paragonimus*
Reservoir	Other animals harboring parasite; source of human infection	Pigs (*Balantidium*), rats (*Paragonimus*), cats (*Brugia*)

4. Vectors

- **Biological Vector:** Parasite develops within vector before transmission (e.g., *Aedes* mosquito for filariasis).
- **Mechanical/Phoretic Vector:** Simply transports parasite (e.g., flies, cockroaches).

5. Sources of Infection

- **Contaminated soil & water:** *Ascaris*, *Trichuris*, hookworms, *Schistosoma*
- **Food:** Undercooked fish (flukes), crabs (*Paragonimus*), snails
- **Arthropods:** Mosquitoes (malaria), Triatoma bugs (Chagas), sand flies (Leishmania)
- **Animals:** Cats (*Toxoplasma*), rats (*Hymenolepis*)
- **Other humans:** Food handlers, carriers
- **Self:** Autoinfection

6. Terms

Term	Definition
Carrier	Harbors pathogen without symptoms
Exposure	Contact with infective agent
Infection	Establishment of agent in host
Incubation Period	Infection to symptom onset
Pre-patent Period	Infection to detectable evidence
Autoinfection	Self-reinfection (e.g., *Enterobius*, *Capillaria*, *Strongyloides*)
Superinfection/Hyperinfection	Massive reinfection with same species

7. Modes of Transmission

Route	Examples
Oral (most common)	Food/water with cysts, eggs, larvae
Skin penetration	Hookworms, *Strongyloides*, *Schistosoma*
Arthropod bites	Malaria, filariasis, leishmaniasis
Congenital	*Toxoplasma* (crosses placenta)

Transmammary	*Ancylostoma*, *Strongyloides* (breast milk)
Inhalation	*Enterobius* eggs

8. Nomenclature

- **Family: Ends in -idae**
- **Genus: Capitalized, italicized**
- **Species: Lowercase, italicized**
- ***Example: Entamoeba histolytic***

9. Life Cycle

- **Parasites adapt to ensure transmission.**
- **More complex life cycle = lower survival chance.**
- **Produce numerous progeny to overcome attrition.**

10. EPIDEMIOLOGY

Term	Definition
Incidence	New cases in given period
Prevalence	% infected at given time
Cumulative Prevalence	% infected with at least one parasite
Intensity/Worm Burden	Number of worms per person
Morbidity	Clinical consequences affecting well-being

11. Treatments

Term	Definition
Deworming	Use of anthelmintic drugs
Cure Rate	% previously positive now egg-negative
Egg Reduction Rate (ERR)	% fall in egg counts after treatment
Selective Treatment	Based on diagnosis/assessment
Targeted Treatment	Risk group treated (no diagnosis)
Universal Treatment	Entire community treated
Preventive Chemotherapy	Regular large-scale drug administration
Coverage	% of target population reached
Efficacy	Drug effect in ideal conditions
Effectiveness	Drug effect in real-world conditions
Drug Resistance	Genetic loss of susceptibility

12. Prevention & Control

- **Morbidity Control:** Periodic deworming of at-risk groups
- **IEC(Information, Education, and Communication.):** Health education for healthy practices
- **Environmental Management:** Modify environment to reduce vectors
- **Environmental Sanitation:** Safe excreta disposal, clean water, hygiene

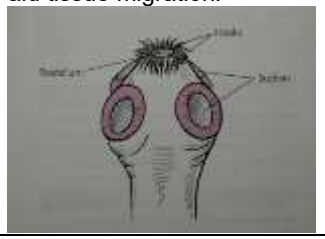
13. Eradication vs. Elimination

Term	Definition	Continued Measures?
Eradication	**Zero worldwide incidence (permanent)**	**No**
Elimination	**Zero incidence in defined area**	**Yes (surveillance needed)**

HOST-PARASITE RELATIONSHIP

The relationship between host and parasite has evolved over time, causing changes in the parasite's biology, biochemistry, immunology, and structure to ensure survival.

A. Adaptation of Parasites

Feature	Adaptation
Locomotory Organs	Apicomplexa have none; free-living flatworms have cilia, parasitic ones do not.
Digestive Organs	Cestodes/trematodes absorb nutrients through **tegument with microvilli**.
Attachment Organs	**Hooks and suckers** anchor parasite and aid tissue migration. 
Size & Shape	*Ascaris* uses constant movement to maintain position; thickened integument resists host enzymes and juices. 
Protective Coverings	Spines (intestinal flukes) prevent abrasion; egg/larvae/cyst coverings resist digestion and environment.
Reproductive System	Highly developed; most flatworms are **hermaphroditic** (except *Schistosoma*); flukes undergo asexual reproduction in intermediate hosts.
Biochemical (Streamlining)	Loss of metabolic pathways; parasite cannot synthesize certain components and must obtain from host (e.g., hemoflagellates, helminths).

B. Specialized Mechanisms for Entry

Parasite	Mechanism
Entamoeba histolytica	**Cysteine proteinases –** penetrate mucosa, degrade basement membrane
***Schistosoma* cercariae**	**Penetration glands** – enzymes digest skin for entry
Cestode embryos	**Six hooklets** – aid tissue penetration before encysting

C. Effects of the Parasite on the Host

Mechanism	Example	Effect
Interference with Vital Processes	*E. histolytica* cysteine proteinases	Digest cellular materials, degrade epithelial basement membrane → tissue invasion

Invasion & Destruction	*Plasmodium* in RBCs	Cell rupture → merozoite release
	S. japonicum eggs in liver	Granuloma → fibrosis → portal hypertension
	Hookworm cutting plates	Destroy intestinal villi
	Ascaris masses	Intestinal obstruction; may invade appendix/bile ducts
Nutrient Deprivation	Hookworm (heavy infection)	Chronic blood loss → iron deficiency anemia
	D. latum	Competes for Vitamin B12 → megaloblastic anemia

D. Effects of the Host on the Parasite

Factor	Example	Effect on Infection
Genetic Makeup	Sickle cell trait	Protects against *P. falciparum*
	Duffy blood factor	Increases susceptibility to *P. vivax*
Nutritional Status	High-protein diet	Unsuitable for intestinal protozoans
	Low-protein diet	Favors amebiasis symptoms & complications
	High-carbohydrate diet	Favors tapeworm development
Immune Response	Acquired immunity	Rarely absolute; modifies disease severity in endemic areas

IMMUNOLOGY OF PARASITICE INFECTIONS

1. Function of the Immune System

- Protects the body from pathogens through a **tightly-controlled balancing act**.
- **Dysfunction can lead to:** Unchecked infection OR harmful overactivation.
- Immunity to parasites is complicated because:
  - Eukaryotic parasites are **similar to host cells** in makeup.
  - Parasites have **evolved evasion strategies** over millions of years.

2. Possible Outcomes of Host-Parasite Interaction

1. Parasite **fails to establish**
2. Parasite establishes → **host eliminates infection**
3. Parasite establishes → host **partially overcomes** but not totally
4. Parasite establishes → host **damages itself** trying to eliminate it
5. Parasite establishes → **kills the host**

3. HOST-PARASITE INTERACTIONS (First Line Defense)

Barrier Type	Mechanism	Examples / Evasion by Parasites
Skin	Physical barrier	Hookworms/Strongyloides secrete proteins; Schistosoma cercariae use lytic enzymes
Mucous Membranes	Tight junctions block entry	
Chemical Barriers	Low pH of vaginal secretions/gastric juices	T. vaginalis can't survive low pH; G. lamblia motility reduced by intestinal secretions
		Evasion: Thick egg shells (Ascaris, Trichuris, Taenia); cystic walls (Entamoeba, Giardia) resist acid
Body Fluids	Lipase in breast milk (toxic to G. lamblia)	
	Lysozyme in tears/saliva + IgA	Destroys microorganisms
Physiologic Functions	Peristalsis, cilia motion, coughing, flushing of urine	Expels parasites; urine reduces T. vaginalis

3.2 SECOND HOST DEFENSE

- When physical barriers are overcome, the body senses invading parasites via:
  - **Pathogen-Associated Molecular Patterns (PAMPs)**
  - **Pattern Recognition Receptors (PRRs)**

4. HOST IMMUNE RESPONSE

A. Innate Immune Response

- **Non-specific, immediate** mechanical, chemical, and cytokine-mediated actions.
- **Phagocytosis** by macrophages and dendritic cells → oxidative killing + toxic peptides.
- Some parasites **survive inside macrophages** (Leishmania, T. gondii, T. cruzi) → require cell-mediated immunity (NK cells, T-lymphocytes).
- **Toll-like Receptors (TLRs):** Earliest recognition mechanism.
  - 10 TLRs identified; each activated by specific components (LPS, lipoproteins, flagellin, viral RNA).
  - TLR activation → cytokine production (interferon gamma, IL-1) → activates NK cells, macrophages → inflammatory response.

B. Acquired Immune Response

- Stimulated by **parasite antigens** (surface, secretions/excretions, somatic tissues).
- **Immunologic priming:** First contact → subsequent exposures yield faster, stronger response (immunologic memory).
- **Types:** Antibody-dependent OR Cell-mediated.
- **Genetic control:** MHC gene products regulate T-lymphocyte activity; HLA is also a factor.

5. ACQUIRED IMMUNE RESPONSE MECHANISMS

5. 1 T-Helper Lymphocytes (CD4)

Subset	Produces	Activates	Response Type
Th1	Gamma interferon, IL-2	Cytotoxic lymphocytes (CD8), macrophages	**Cell-mediated immunity**
Th2	IL-4, IL-5, IL-6	B-lymphocytes → plasma cells → immunoglobulins	**Humoral immunity**

5.2 Cell-Mediated Immunity Examples:

- Direct cytotoxicity on parasite OR indirect via NK cells/antibody-producing B-cells.
- Migrating larvae of *Toxocara canis* killed through cell-mediated activity.

5.3 Humoral Immunity (Antibodies):

- Classes: **IgE, IgG, IgM, IgA**
- **In helminthic infections:** Eosinophilia + elevated serum IgE are common.
- **IgE + mucosal mast cells/eosinophils/goblet cells** → expulsion of adult GI helminths.
- **Antibody-Dependent Cell-Mediated Cytotoxicity (ADCC):**
  - IgE + eosinophils → destroy parasites (Schistosoma, microfilariae in tropical pulmonary eosinophilia).
  - Neutrophils and platelets also participate.
  - IgM + secretory IgA mediate ADCC in *G. lamblia*.

5.4 Other Antibody Actions:

- **IgG + IgM:** Prevent erythrocyte penetration by *Plasmodium* and *Babesia*; with complement → lyse *T. cruzi* trypomastigotes.
- **Secretory IgA:** Protects against metacestode and GI infections.
- **IgA + IgG + cell-mediated immunity:** Clears *Cryptosporidium* in immunocompetent hosts.

6. PARASITE EVASION MECHANISMS

Mechanism	Description	Examples
Resistance to Immune Response	Survive/replicate despite innate immunity; resistant to complement, macrophages	*T. brucei* resists APOL1; helminth cuticle resists neutrophils/macrophages
Immune Suppression	Reduce immune function	*Plasmodium* → lowers phagocytosis, defective antigen processing; *T. brucei* glycoproteins impair B/T cells; *E. histolytica* suppresses macrophage respiratory burst; *Fasciola* downregulates Th1; *Wuchereria* → polyclonal hypergammaglobulinemia
Antigenic Variation	Change surface proteins to evade antibodies	*T. brucei* (VSG); *P. falciparum* (MSA, RESA repeat variation); *G. lamblia*
Host Mimicry	Mask self with host molecules	*E. granulosus* carries P blood group antigens; *Schistosoma* acquires host antigens
Intracellular Sequestration	Hide inside cells	*T. cruzi, Leishmania, T. gondii* in macrophages; *P. falciparum* attaches to endothelial cells via "knobs" → avoids splenic filtration and antibodies

7. Specific Examples of Immune-Mediated Pathology:

- **Cerebral malaria:** Caused by sequestration of *P. falciparum*-infected RBCs in deep vasculature.
- **Schistosomiasis:** Pathology from immune response to eggs trapped in organs.
- **T. cruzi:** Antibodies may cross-react with adrenergic/muscarinic receptors → autonomic dysfunction, arrhythmias.

8. PRACTICAL APPLICATIONS

Understanding host immune response to parasites is useful for:

- **Immunodiagnosis**
- **Predicting pathology**
- **Immunoregulation and immunomodulation** studies
- **Vaccine development**
- **Development of novel anti-parasitic drugs**

GROUPS OF PARASITES WITH MEDICAL AND PUBLIC HEALTH IMPORTANCE

1. CLASSIFICATION OF PARASITES

Taxonomic Hierarchy (Linnaean)

Kingdom → Subkingdom → Phylum → Class → Order → Family → Genus → Species

- **Traditional basis:** Morphological characterization of different life stages.
- **Modern tools:** DNA sequencing, proteomics, RNA interference, PCR – useful for identifying **cryptic species**.
- **Current approach:** Integrate **molecular and morphological** methods.

2. PROTOZOA (Unicellular Eukaryotes)

General Characteristics

- Nucleus, cytoplasm, outer membrane, organelles.
- **Locomotory organelles:** Cilia, flagella, pseudopodia.
- **Apical complex:** Aids in penetration of target cells (in some).
- Require **wet environment** for feeding, locomotion, reproduction.
- **Trophozoite:** Vegetative, motile, feeding stage.
- **Cyst:** Infective, resistant stage (to environment, acid, etc.).
- Can multiply within host and/or vector.

Phylum Classification of Medically Important Protozoa

Phylum	Subphylum	Locomotory Organ	Genera
Sarcomastigophora	**Mastigophora**	Flagella (whip-like)	*Giardia, Chilomastix, Trichomonas, Dientamoeba, Trypanosoma, Leishmania*
	Sarcodina	Pseudopodia (foot-like)	*Entamoeba, Endolimax, Iodamoeba, Acanthamoeba, Naegleria*
Ciliophora		Cilia (hair-like)	*Balantidium coli* (only one of medical importance)
Apicomplexa		Apical complex (polar rings, conoid, rhoptries, micronemes) for penetration	*Plasmodium, Babesia, Toxoplasma, Cystoisospora, Cryptosporidium, Cyclospora*
Microspora		Polar filament/tube for injecting infective material into host cell	*Enterocytozoon, Encephalitozoon* (opportunistic in AIDS)

3. METAZOA (Multicellular)

A. NEMATODES (Roundworms)

General Characteristics:

- Elongated, cylindrical, bilateral symmetry.
- **Complete digestive tract** with **triradiate muscular pharynx**.
- **Separate sexes** (some parthenogenetic).
- **Sensory organs:** Amphids (anterior), Phasmids (posterior) – used for classification.

A1. Classification by Phasmids:

Group	Phasmids	General
Phasmid nematodes (Secernentia)	Present	*Ascaris, Parastrongylus, Hookworms, Strongyloides, Enterobius, Wuchereria, Brugia*
Aphasmid nematodes (Adenophorea)	Absent	*Trichuris, Trichinella, Capillaria*

A2. Classification by Habitat:

Habitat	Genera
Small Intestine	*Ascaris, Hookworms, Strongyloides, Capillaria*
Large Intestine (Colon)	*Trichuris, Enterobius*
Extraintestinal	*Wuchereria, Brugia* (lymphatics); *Parastrongylus* (eyes/meninges); *Trichinella* (muscles)

A3. Modes of Transmission:

Mode	Examples
Ingestion of embryonated eggs	*Ascaris, Trichuris, Enterobius*
Skin penetration (filariform larvae)	Hookworms, *Strongyloides*
Mosquito bite	*Wuchereria, Brugia*
Ingestion of infective larvae (in food)	*Capillaria* (fish), *Trichinella* (pork), *Parastrongylus* (snails)
Autoinfection	*Capillaria, Strongyloides, Enterobius*
Inhalation	*Enterobius, Ascaris* (possible)

B. CESTODES (Tapeworms)

B1. General Characteristics:

- **Flatworms (Platyhelminthes):** Dorso-ventrally flattened, segmented, ribbon-like.
- **No digestive tract**, no circulatory system.
- **Hermaphroditic.**
- **Structure:** Scolex (attachment) → Neck (growth) → Strobila (segments/proglottids: immature → mature → gravid).

B2. Comparison of Medically Important Orders:

Feature	Pseudophyllidea	Cyclophyllidea
Scolex	Spatulate with sucking grooves (bothria)	Globular with 4 muscular suckers
Genital Pore	Present	Present
Uterine Pore	Present (eggs released continuously)	Absent (apolysis: gravid segments detach)
Eggs in Diagnosis	Eggs found in stool	Eggs and segments found in stool
Egg Type	Operculated, immature	Non-operculated, contain hexacanth embryo
Intermediate Hosts	Two	One
Larval Type	Procercoid → Plerocercoid	Cysticercus, Cysticercoid, Hydatid
Examples	*Diphyllobothrium* (definitive host: human); *Spirometra* (intermediate host: human)	*Taenia* (cysticercus), *Hymenolepis, Dipylidium,* *Raillietina* (cysticercoid), *Echinococcus* (hydatid)

B3. Modes of Infection:

→ **Adult tapeworm:** Ingestion of infected intermediate host (undercooked meat/fish).

→ **Larval infection:** Ingestion of eggs (e.g., *Taenia solium* → cysticercosis; *Echinococcus* → hydatid cyst).

C. TREMATODES (Flukes)

C1. General Characteristics:

- **Flatworms:** Leaf-like, unsegmented.
- **Incomplete digestive tract**, no circulatory system.
- **Hermaphroditic** (except *Schistosoma* which has separate sexes).
- **Suckers:** Oral sucker + Ventral sucker (acetabulum); some have genital sucker (gonotyl).
- **Life Cycle:** Require **two intermediate hosts**.
  - **First intermediate host:** Always a snail.
  - **Second intermediate host:** Fish, crustacean, snail, or aquatic plants.
- **Eggs:** Operculated (except schistosomes? – actually schistosomes are non-operculated but have a spine).
- **Infective stage for host: Metacercaria** (encysted larva) – **EXCEPT** *Schistosoma* (infective stage = **cercaria**).

C2. Classification by Habitat:

Habitat	Genera
Blood (mesenteric veins)	*Schistosoma* spp. (blood flukes)
Lungs	*Paragonimus westermani* (lung fluke)
Liver & Bile Passages	*Fasciola, Clonorchis, Opisthorchis*
Intestines	*Fasciolopsis, Echinostoma, Heterophyids*

C3. Egg Development:

D. ARTHROPODS

D1. General Characteristics:

Egg Type at Passage	Examples
Mature (contains miracidium)	*Schistosoma, Clonorchis, Opistorchis, Heterophyids*
Immature (requires aquatic development)	*Paragonimus, Fasciola, Fasciolopsis, Echinostoma*

- Bilateral symmetry, segmented body, **jointed appendages**.
- **Chitinous exoskeleton**.
- Includes: Insects, mites, ticks, spiders, scorpions, centipedes, millipedes, crustaceans, pentastomids.

D2. Effects on Human Health:

Effect	Examples
Envenomization	Bites (spiders, flies, bugs, mites, ticks); Stings (scorpions, ants, wasps, bees)
Allergens	Arthropod parts/secretions
Biological Vectors	Mosquitoes, flies (transmit *Plasmodium*, filaria, trypanosomes, *Babesia*, *Leishmania*)
Mechanical Vectors	Flies, cockroaches (carry pathogens from unsanitary environments)

Dermatologic Conditions	Prolonged contact with fleas, lice
Myiasis	Infestation by fly larvae invading tissues

NEMATODES (Hookworms)

1. Necator americanus	*2. Ancylostoma duodenale*
	

1. General Overview

- **Soil-transmitted helminths** (nematodes).
- **Blood-sucking** parasites attached to **small intestinal mucosa**.
- Common in **tropical/subtropical** areas as single or mixed infections.

2. PARASITE BIOLOGY

Adult Morphology

Feature	*Necator americanus*	*Ancylostoma duodenale*
Size (Female)	9-11 mm x 0.35 mm	Slightly larger
Size (Male)	5-9 mm x 0.30 mm	Slightly larger
Head Curvature	Curved opposite to body (hook-like)	Same direction as body curvature
Buccal Capsule	Ventral pair of **semilunar cutting plates**	Two pairs of **curved ventral teeth**
Male Posterior	Broad caudal bursa with rays (for copulation)	Same

3. Muscle Type: Meromyarian (2-5 cells per dorsal/ventral half).

Larval Stages

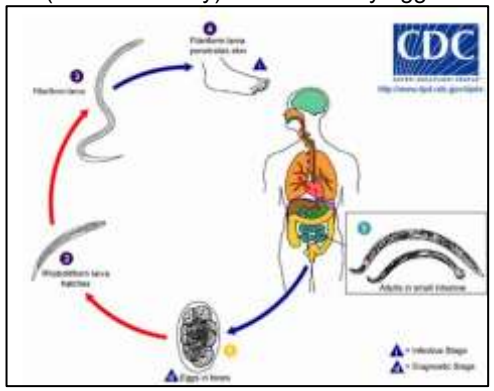
Stage	Key Features
Rhabditiform Larva	Indistinguishable between species; larger than *Strongyloides*, longer buccal cavity, smaller genital primordium
Filariform Larva (L3 - Infective) 	- *N. americanus*: Buccal spears conspicuous, parallel; transverse striations on tail sheath - *A. duodenale*: Buccal spears inconspicuous; transverse striations on tail sheath

4. Egg

- **Shape:** Bluntly rounded ends.
- **Shell:** Single thin, transparent hyaline shell.
- **Contents at oviposition:** Unsegmented → 2-8 cell stage in fresh feces.
- **Differentiation:** Difficult to distinguish between species.

5. LIFE CYCLE

1. **Adults** in small intestine → copulation → females lay eggs → passed in feces.
2. **In soil:** Eggs embryonate → hatch in 1-2 days → **rhabditiform larva**.
3. After 7-10 days, molts twice → **filariform larva (L3)** – **infective stage**.
4. **Penetrates skin** → enters venules → heart → lungs → alveoli.
5. Ascends trachea → swallowed → small intestine.
6. Matures to adults (sexual maturity) → females lay eggs.



Alternative route for *A. duodenale*: Oral (ingestion), transmammary, or dormant in muscles.

PATHOGENESIS & CLINICAL MANIFESTATIONS

Site	Pathology	Symptoms
Skin (entry site)	Maculopapular lesions, erythema	**"Ground itch"** or **"dew itch"** (severe itching, edema, papulovesicular eruptions for up to 2 weeks)
Lungs (migration)	Minute hemorrhages, eosinophilic/leukocytic infiltration	Bronchitis/pneumonitis (rare in tropics)
Small Intestine (adult attachment)	Blood-sucking, tissue damage	Abdominal pain, steatorrhea, diarrhea (with blood/mucus), eosinophilia

Chronic Effects:

- **Iron deficiency anemia** (microcytic, hypochromic) – due to continuous blood loss.

- *A. duodenale* causes greater blood loss/worm than *N. americanus*.
- **Hypoalbuminemia** – loss of blood, lymph, protein.
- Symptoms: Exertional dyspnea, weakness, dizziness, lassitude, rapid pulse, edema, albuminuria.

Prognosis: Generally good; complications mild compared to ascariasis.
Immunity: No perpetual immunity; Th2 response (eosinophils, mast cells); polyvalent IgE may provide some protection.

DIAGNOSIS

- **Definitive:** Identification of eggs in feces.
- **Methods:**
  - **Direct fecal smear:** Only for heavy infections.
  - **Kato-Katz:** Quantitative (eggs/gram); eggs clear rapidly with glycerine (30-60 min).
  - **Concentration methods:** Zinc sulfate flotation, formalin-ether/ethyl acetate – more sensitive.
  - **FLOTAC:** Higher sensitivity for STH.
  - **Culture (Harada-Mori):** For species identification (hatches larvae).
  - **Molecular:** PCR (DNA in feces), ELISA (coproantigens).

TREATMENT

Drug	Dose	Mechanism	Notes
Albendazole (drug of choice)	400 mg single dose (adults & children >2 yrs)	Benzimidazole; blocks glucose uptake; larvicidal & ovicidal	Chewable/suspension available
Mebendazole	500 mg single dose	Same	

Adverse Effects: Mild/transient – epigastric pain, diarrhea, headache, dizziness.
Supportive: Iron supplementation, adequate diet (for anemia & hypoproteinemia).
Drug Resistance: Reported in countries with regular deworming; monitoring needed (cure rate, egg reduction rate, reinfection rate).

EPIDEMIOLOGY

- **Global:** 576–740 million infected; >50,000 deaths/year from anemia.
- **Distribution:** Once distinct (*A. duodenale* – Europe/SW Asia; *N. americanus* – Africa/Americas), now both widespread in tropics/subtropics.

Philippines:

- **Species:** 97% *N. americanus*, 1% *A. duodenale*, 2% mixed.
- **High-risk groups:** Farmers, pregnant women (5.5%), adolescents (2.8%), military (46.9%), indigenous communities (13.6%), food handlers (22.7% Manila, 14.8% Cebu).
- **Transmission factors:**
  - Environment: Damp, sandy/friable soil, 24-32°C, decaying vegetation.
  - Fecal pollution (open defecation, night soil fertilizer).
  - Skin contact with infected soil.
- **Routes:**
  - *N. americanus*: Percutaneous only.
  - *A. duodenale*: Percutaneous, oral (raw vegetables), transmammary, dormant in muscles.

Animal Hookworms (Humans - Cutaneous Larva Migrans):

- *Ancylostoma braziliense* (cat), *A. caninum* (dog) → cause **creeping eruption**.
- *Ancylostoma ceylanicum*: First human case in Philippines (1968, Ilocos Norte).

PREVENTION & CONTROL

1. **Mass Drug Administration (MDA):** WHO recommends annual treatment for school-age children if STH prevalence ≥20%.
2. **WASHED Approach:**
   - **W**ater access
   - **A**ccess to sanitation
   - **S**anitation promotion
   - **H**ygiene education
   - **E**nvironmental management
   - **D**eworming
3. **Community-Led Total Sanitation (CLTS):** Discourage open defecation; sanitary disposal of feces.
4. **Personal Protection:** Wear shoes, slippers, boots.
5. **Vaccine Development:** Human Hookworm Vaccine Initiative (Sabin Institute); Phase I trial completed for secretory antigen vaccine.

Strongyloides stercoralis

1. General Overview

- **Nematode** with **free-living** (rhabditiform) and **parasitic** (filariform) stages.
- Only species of the genus naturally **pathogenic to humans**.
- Also known as **threadworm**.

2. PARASITE BIOLOGY

Adult Morphology

Form	Size	Key Features
Parasitic Female	2.2 mm x 0.04 mm	Colorless, semi-transparent, finely striated cuticle; slender anterior, conical pointed tail; short buccal cavity with 4 indistinct lips; long esophagus (anterior ¼); vulva posterior ⅓; uteri contain 8-12 eggs
Free-living Female	1 mm x 0.06 mm	Smaller; muscular double-bulbed esophagus; straight cylindrical intestine
Free-living Male	0.7 mm x 0.04 mm	Ventrally curved tail; 2 copulatory spicules, gubernaculum; no caudal alae
Parasitic Male		Not reliably identified

3. Larval Stages

Stage	Size	Key Features
Rhabditiform Larva	225 µm x 16 µm	Elongated esophagus with pyriform posterior bulb; shorter buccal capsule than hookworm; larger genital primordium
Filariform Larva (Infective)	~550 µm	Non-feeding, slender; smaller than hookworm; **distinct cleft at tip of tail**

4. Egg

- **Size:** 50-58 µm x 30-34 µm.
- **Shell:** Clear, thin.
- Similar to hookworm eggs but **smaller**.

5. LIFE CYCLE

5.1 Free-Living Cycle (Soil)

1. Free-living female lays **embryonated eggs** in soil.
2. Hatch into **rhabditiform larvae** (feed on organic matter).
3. Develop into **free-living adults** (continue cycle).
4. When conditions unfavorable → rhabditiform larvae → **filariform larvae (L3)** – infective to humans.

5.2 Parasitic Cycle (Human)

1. **Filariform larvae penetrate skin** → enter circulation → heart → lungs → alveoli.
2. Migrate to **larynx** → swallowed → **small intestine (duodenum)** .
3. Develop into **parthogenetic females** in ~1 month.
4. Females invade intestinal mucosa → lay eggs (hatch into **rhabditiform larvae**).
5. Rhabditiform larvae migrate to lumen → passed in feces.

5.3 Autoinfection

- Rhabditiform larvae in large intestine → develop into **filariform larvae**.
- Penetrate intestinal mucosa or perianal skin → enter circulation → restart cycle **without leaving host**.

6. PATHOGENESIS & CLINICAL MANIFESTATIONS

Three Phases of Acute Infection

Phase	Pathology	Symptoms
1. Skin Invasion	Filariform larvae penetrate	Erythema, pruritic hemorrhagic papules
2. Larval Migration	Lung destruction	Lobar pneumonia with hemorrhage, cough, tracheal irritation (bronchitis-like)
3. Intestinal Penetration	Adult females in mucosa (duodenum > jejunum)	Variable symptoms

7. Severity of Infection

Infection Level	Symptoms
Light	Asymptomatic
Moderate	Diarrhea alternating with constipation
Heavy	**Cochin China diarrhea** – intractable, painless, intermittent watery/bloody stools
Hyperinfection	Accelerated autoinfection (usually immunocompromised); GI + pulmonary symptoms; ↑ larvae in stool/sputum

8. Chronic Strongyloidiasis

- Often asymptomatic.
- Possible: Intermittent vomiting, diarrhea, constipation, borborygmi.
- Common: **Anal pruritus, urticaria, larva currens rashes**.
- Reported: Recurrent asthma, nephritic syndrome.

9. Complications

- Edema, emaciation, anorexia, anemia, lobar pneumonia, ileus, intestinal obstruction, GI bleeding, malabsorption → cachexia.

10. Prognosis

- **Light infections:** Good.
- **Moderate/heavy:** High mortality (massive tissue invasion).
- **Disseminated:** In cancer, malnutrition, HIV/AIDS, HTLV-1, immunosuppressive drugs (post-transplant) – often fatal.

11. DIAGNOSIS

Method	Notes
Eosinophilia	Clue (unexplained)
Stool Concentration (Baermann funnel gauze)	Repeated exams increase detection
Harada-Mori Culture	One of most successful for identification
Nutrient Agar Plates	Recommended method
Other	Beale's string test, duodenal aspiration, small bowel biopsy
Sputum/Urine	Larvae found in disseminated disease
Serology	Cross-reacts with filarial antigens (limited use in endemic areas)

12. TREATMENT

Drug	Dose/Notes
Ivermectin (drug of choice)	Best for chronic uncomplicated; higher doses/longer for h
Albendazole	Previously used; ovicidal + larvicidal
Thiabendazole	Previously used

13. Adverse Effects:

- **Thiabendazole:** Dizziness, GI irritation, drowsiness, pruritus, headache (hours-long).
- **Albendazole:** Transient GI discomfort, headache.

Contraindications: Pregnancy, hypersensitivity.

Monitoring:

- **Egg reduction rate:** Cannot be determined (eggs not passed in feces).
- **Reinfection rate:** Difficult to calculate (autoinfection).

14. EPIDEMIOLOGY

- **Global:** 50–100 million infected; distribution similar to hookworm (tropics, subtropics, Europe, USA).
- **Soil-transmitted helminth.**

Philippines:

- **Prevalence:** Relatively rare.
  - 1.2% (50/4,208) by Harada-Mori.
  - 0.05% (148/294,176) overall.
  - Fluctuates 0–2.3% by area.
- **Demographics:** More common in **male children 7–14 years old**.

Transmission Factors:

- Poor sanitation, indiscriminate fecal disposal.

- **Autoinfection:** Explains infections lasting >30 years (e.g., US veterans from Korea/Vietnam).

15. PREVENTION & CONTROL

- Same as **hookworm** (soil-based transmission).
- **Key Measures:**
  - Improve environmental sanitation.
  - Stop indiscriminate defecation.
  - Wear footwear.
  - Health education (personal, family, community hygiene).
  - Treat infected individuals.
- **Special Precautions:**
  - Clear *Strongyloides* infection in:
    - Cancer patients
    - Debilitating diseases (e.g., pulmonary TB)
    - Malnourished individuals
    - Pre-organ transplant patients
  - Prevents **disseminated strongyloidiasis** (almost always fatal).

Enterobius vermicularis	

General Overview

- Causes **enterobiasis** or **oxyuriasis**.
- **Hallmark symptom:** Perianal itching (**pruritus ani**).
- Low morbidity/mortality but can cause **ectopic complications** if worms migrate.
- **Most common helminth** in **temperate regions** with good sanitation.
- **Muscle type:** Meromyarian (2-5 cells per dorsal/ventral half).

1. PARASITE BIOLOGY

Adult Morphology

Feature	Female	Male
Size	8-13 mm x 0.4 mm	2-5 mm x 0.1-0.2 mm
Key Features	Long pointed tail; uteri distended with eggs	Curved tail; single spicule; dies after copulation
Both Sexes	Cuticular alar expansions at anterior end; prominent posterior esophageal bulb	

Larva (Rhabditiform)

- **Size:** 140-150 µm x 10 µm.
- Has esophageal bulb but **no cuticular expansions**.

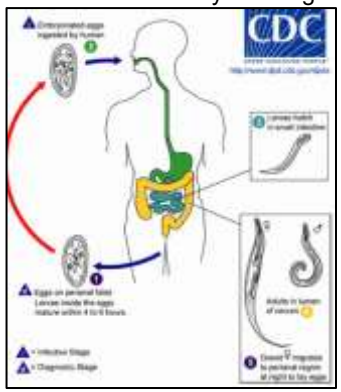
Egg

- **Shape:** Asymmetrical, **D-shaped** (one side flat, one convex).
- **Size:** 50-60 µm x 20-30 µm (avg 55 x 36 µm).
- **Shell:** Outer triple albuminous (mechanical protection) + inner lipoidal membrane (chemical protection).
- **Contents:** Tadpole-like embryo → fully mature outside host in **4-6 hours**.

LIFE CYCLE

1. **Adults** reside in **cecum** and adjacent intestines.
2. **Gravid female migrates** down intestinal tract → exits anus (usually **evening**) → deposits eggs on **perianal skin**.
   - One female lays **~11,105 eggs/day** (range 4,672–16,888).
   - Female dies after egg deposition.
3. Eggs become **embryonated** within **6 hours**.
4. **Infection route:** Ingestion of embryonated eggs (or inhalation).
5. Eggs hatch in **duodenum** → larvae pass to cecum → develop into adults.

Retroinfection: Hatched larvae enter anus directly and migrate back into large intestine.



Egg Survival:

- Resistant to disinfectants.
- Succumb to **dry air** within 1 day.
- **Moist conditions:** Viable up to 13 days.
- Survive in dust → airborne infection possible.

PATHOGENESIS & CLINICAL MANIFESTATIONS

Site	Pathology	Symptoms
Intestinal Mucosa	Mild catarrhal inflammation from attachment; possible deeper inflammation (secondary bacterial invasion)	Abdominal pain
Perianal Region	Mechanical irritation from egg-laying females	**Intense pruritus ani** (worse at night) → scratching → secondary bacterial infection
Systemic (Children)		Insomnia, poor appetite, weight loss, irritability, teeth grinding

Complications (Aberrant Migration)

Site	Condition
Appendix	Appendicitis
Female Reproductive Tract	Vaginitis, endometritis, salpingitis
Peritoneal Cavity	Granuloma around eggs/worms
Ectopic Sites (rare)	Liver, lung

Prognosis: Good, but extremely contagious (**familial/group disease**).

DIAGNOSIS

Method	Details
Clinical Suspicion	Perianal itching (relieved by scratching)
Adult Worms	May be seen in feces or perianal area
Eggs in Feces	Only **~5%** of cases
Graham's Scotch Tape Swab (GOLD STANDARD)	Perianal tape swab; highest sensitivity; low-cost, easy, specific

TREATMENT

Drug	Dose	Notes
Mebendazole (drug of choice)	100 mg PO single dose	Cure rate >90%
Albendazole (drug of choice)	400 mg PO single dose	Same
Pyrantel Pamoate (secondary)	11 mg/kg base PO single dose (max 1g)	

Important Considerations:

- Treat **entire household** (familial disease).
- **Cure confirmed** after **7 consecutive negative perianal smears**.
- **Egg reduction rate:** Difficult to determine (eggs not in feces for Kato-Katz).
- **Adverse effects:** Mild, transient GI disturbance, headache.
- **Contraindications:** Hypersensitivity.

EPIDEMIOLOGY

- **Global:** 208.8 million infected; temperate AND tropical regions; common in developed & developing countries.
- **USA/Canada:** 18 million infected; prevalence 12-41% (Washington, D.C.).

Philippines:

- **Schoolchildren:** 29% (private schools), 56% (public schools).
- **Sex:** Higher in females (16%) than males (9%).
- Eggs found in **nail clippings**.

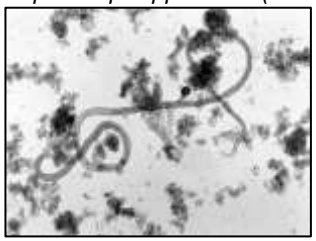
Transmission:

- **NOT controlled by sanitation alone** (eggs deposited perianally, not in feces).
- **Routes:** Oral, inhalation (dust), retroinfection.
- **Risk factors:** Overcrowding, thumb-sucking, nail-biting, lack of parental knowledge.

PREVENTION & CONTROL

Measure	Details
Personal Hygiene	Cut nails short; handwashing after toilet, before/after meals
Bathing	Use showers (not bathtubs)
Sleeping	Infected persons sleep alone until treated
Laundry	Wash underwear, night clothes, bedding in **hot soapy water**
Environment	Vacuum clean beds and contaminated areas
Treatment	Treat entire family (chemotherapy)
Mass Drug Administration (MDA)	Expected to impact prevalence
Health Education	In schools (teachers & children) on control/prevention

Capillaria philippinensis (intestinal capillariasis)



General Overview

- One of four *Capillaria* species infecting humans.
- First reported in **1963** (Northern Luzon, Philippines) by Chitwood et al.
- **Zoonotic disease** – natural hosts are **fish-eating birds**; humans are **incidental hosts**.
- **Intestinal capillariasis** characterized by: Abdominal pain, chronic diarrhea, gurgling stomach.
- Can lead to **protein-losing enteropathy**, electrolyte imbalance, malabsorption → **death** if untreated.

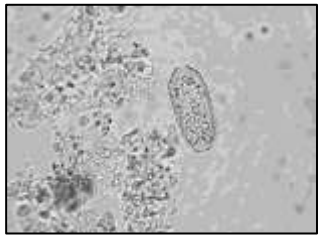
1. PARASITE BIOLOGY

Classification

- **Superfamily:** Trichinelloidea (same as *Trichuris* and *Trichinella*).
- **Characteristic shape:** Thin filamentous anterior + thicker, shorter posterior.

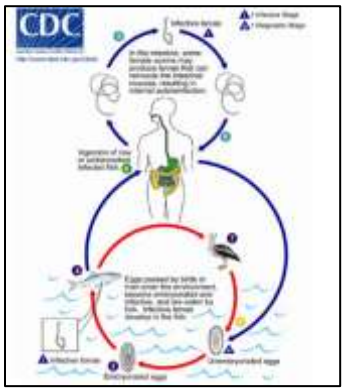
Adult Morphology

Feature	Male	Female
Length	1.5–3.9 mm	2.3–5.3 mm
Key Features	Spicule 230–300 µm (unspined sheath)	Vulva at junction of anterior/middle thirds
Both Sexes	Esophagus has **stichocytes** (rows of secretory cells) → structure called **stichosome**; anus subterminal	



Egg

- **Shape:** Peanut-shaped.
- **Shell:** Striated with **flattened bipolar plugs**.
- **Size:** 36–45 µm x 20 µm.
- Passed in feces → embryonate in **soil or water** (must reach water for ingestion by fish).



2. LIFE CYCLE

1. **Eggs** in water ingested by **small freshwater/brackish water fish**.
2. Eggs hatch in fish intestines → develop into **infective larvae**.
3. Humans eat **uncooked/undercooked fish** → larvae escape fish intestines → develop into adults in **human intestines**.
4. **First-generation females** produce **larvae** (to build population).
5. **Subsequent generations** produce **eggs** (some produce both larvae and eggs, or larvae only).
6. **Autoinfection:** Some larvae retained in gut → develop into adults → **hyperinfection** (massive worm burden; up to **200,000 worms/liter of bowel fluid** in autopsy).

3. PATHOGENESIS & CLINICAL MANIFESTATIONS

Symptoms (Progressive)

Stage	Symptoms
Early	Abdominal pain, borborygmi, intermittent diarrhea
Progressive	8–10 voluminous stools/day; weight loss, malaise, anorexia, vomiting, edema
Severe (if untreated)	Protein-losing enteropathy, hypoalbuminemia, malabsorption (fats, sugars), ↓ xylose, ↓ $K^+/Na^+/Ca^{2+}$, ↑ IgE → potentially **fatal**

Pathology

- **No tissue invasion** but causes:
  - Micro-ulcers in epithelium
  - Compressive degeneration of cells
  - Flattened/denuded villi (histology)
  - Dilated mucosal glands
  - Lamina propria infiltrated with plasma cells, lymphocytes, macrophages, neutrophils
- Endoscopy: Segmental erythematous inflammation with superficial erosions.

4. DIAGNOSIS

Method	Findings
Stool Exam (Direct smear/wet mount, concentration)	Characteristic eggs; may also see larvae + adult worms
Duodenal Aspiration	Parasites recovered from small intestine
ELISA (coproantigen)	High specificity (no cross-reaction with *Fasciola*/*Schistosoma*)
Immunoblot/ELISA with *T. spiralis* antigen	Cross-reaction; 100% sensitivity & specificity in studies

TREATMENT

Drug	Dose
Mebendazole (drug of choice)	200 mg twice daily x 20 days
Albendazole (alternative)	400 mg once daily x 10 days

Supportive Care:

- Electrolyte replacement (K^+, Na^+, Ca^{2+}).
- High-protein diet.

Warning: Relapses occur if treatment is **not completed**.

5. EPIDEMIOLOGY

Global Distribution

Country/Region	Notes
Philippines	First recorded (Northern Luzon); 1966 epidemic (Ilocos Sur) → 1,000+ cases, 77 deaths. Nearly 2,000 cases (1967–1990). Also in Zambales, Southern Leyte, Compostela Valley, Zamboanga del Norte (>70 deaths), Zamboanga del Sur, Agusan del Sur, Misamis Occidental.
Other Countries	Thailand, Iran, Japan, Indonesia, UAE, South Korea, India, Taiwan (30 cases, 1983–2003), Egypt, Lao PDR

Transmission

- Eating **uncooked small freshwater/brackish water fish** (e.g., *bagsit* in Ilocos).
- Outbreak in Monkayo (1998) misdiagnosed as "mystery disease" → deaths.

6. PREVENTION & CONTROL

Measure	Details
Sanitation	Prevent indiscriminate disposal of human waste (1967 epidemic linked to washing contaminated bed sheets in lagoons)
Health Education	Discourage eating raw fish; improve health-seeking behavior
Capacity Building	Train health personnel (including lab staff) for early/accurate diagnosis and treatment

WUCHERERIA BANCROFTI (Bancroftian Filariasis)

Feature	Description
Disease	**Bancroftian filariasis**
Periodicity	**Nocturnal (12 MN – 2 AM)**
Vector	**Mosquitoes: *Aedes, Culex, Anopheles***
Microfilaria	**- 270–290 µm, sheath longer than body - Snake-like with graceful curves - Dark-staining nuclei in central axis - No terminal nuclei**

BRUGIA MALAYI (Malayan Filariasis)

Feature	Description
Disease	**Malayan filariasis**
Periodicity	**Subperiodic (mostly nocturnal)**
Vector	**Mosquitoes: *Mansonia***
Adult Worms	**- Male: 13–23 mm - Female: 43–55 mm (indistinguishable from *W. bancrofti* female)**
Microfilaria	**- 177–230 µm, sheathed - Angular curvatures with secondary kinks - 2 terminal nuclei at tip of tail**

COMMON FEATURES (Both Species)

Aspect	Details
Habitat	**Adults in lymphatic vessels; microfilariae in peripheral blood**
Infective Stage	**L3 filariform larva (transmitted by mosquito bite)**
Diagnostic Stage	**Sheathed microfilariae in peripheral blood (nocturnal collection)**
Pathogenesis	**- Lymphatic dilation & obstruction - Lymphedema → elephantiasis - Hydrocele (common in *W. bancrofti*)**

Epidemiology (Global)	**- >1 billion at risk (80+ countries)** **- >120 million infected** **- 43 million seriously affected**
Epidemiology (Philippines)	**- Camarines Norte, Camarines Sur, Albay, Sorsogon** **- Mindoro, Palawan, Romblon** **- Mountain Province** **- All provinces of Mindanao**

Trichinella spiralis

Overview

- **Viviparous/Larviparous** (females produce larvae, not eggs).
- Common parasite of **pigs**; humans are **accidental hosts**.
- Disease: **Trichinellosis** (zoonotic infection).

1. Species Infecting Humans

Species	Geographic Region
Trichinella spiralis spiralis	Temperate regions
Trichinella spiralis nativa	Arctic regions
Trichinella spiralis nelsoni	Africa

2. Adult Morphology

Feature	Female	Male
Size	3.5 mm x 0.06 mm	1.5 mm x 0.04 mm
Color	Whitish	Whitish
Reproductive Organs	Single ovary, oviduct, seminal receptacle, coiled uterus, vulva, vagina	Single testis, posterior cloaca, pair of caudal appendages, pair of papillae

3. Larva

- **Size at birth:** 80–120 µm x 5.6 mm.
- **Size in muscle fiber:** Up to 1300 µm x 35–40 µm.
- **Feature:** Spear-like burrowing anterior tip.

4. **Habitat**

- **Adults:** Small intestine.
- **Larvae:** Striated muscle (encysted).

5. Hosts (Final & Intermediate)

- **Final host:** Rats, pigs, bears, dogs, foxes, carnivores/omnivores.
- **Hosts serve as BOTH final and intermediate** (harbor adults + larvae).
- **Accidental host:** Humans.

6. Life Cycle

1. Ingestion of **raw/undercooked meat** (containing encysted larvae).
2. Larvae excyst in stomach → mature into adults in **small intestine**.
3. Females produce **larvae** (not eggs) → larvae migrate via bloodstream to **striated muscle**.
4. Encyst in muscle → remain viable for years.

7. **Clinical Manifestations**

Phase	Symptoms
1. Intestinal Phase (Adults)	Inflammation of jejunum/duodenum mucosa; malaise, diarrhea, abdominal cramps
2. Muscle Invasion Phase (Larvae)	Fever, edema (periorbital), muscle pain (myalgia), **peripheral eosinophilia**
3. Convalescent Phase	Encapsulated larvae in muscle (often asymptomatic)

8. Diagnosis

Method	Details
History of exposure	Ingestion of raw/undercooked pork or wild game
Muscle biopsy	Demonstration of encysted larvae
Biochemical tests	Elevated muscle enzymes (CPK, LDH)
Beck's xenodiagnosis	Feeding suspected meat to lab animals

9. Treatment

Severity	Management
Non-life-threatening	Rest, increased fluids, fever reducers, pain relievers
Life-threatening	Prednisone, thiabendazole, steroids

10. Prevention and Control

- **Health education** (risks of raw/undercooked meat).
- **Cook meat** to **77°C** (internal temperature).
- **Freeze meat:** -15°C for 20 days OR -30°C for 6 days.
- **Note:** Smoking, salting, drying are **NOT effective**.
- **Meat inspection** (especially pork).
- **Keep pigs in rat-free pens** (prevent cycle).

10. Algorithm for the diagnosis of the probability of acute trichinellosis in humans

GROUP	SYMPTOMS
A	Fever, eyelid and/or facial edema, myalgia
B	Diarrhea, neurological signs, cardiac signs, conjunctivitis, subungual hemorhages, cutaneous rash
C	Eosinophilia (>1,000 eosinophils/ml) and/ or increased total IgE levels, increased levels of muscular enzymes
D	Positive serology (with a highly specific test), seroconversion, positive muscular biopsy

ANISAKIS SPP.

1. Overview

- Causes **Anisakiasis**.
- **Definitive host:** Marine mammals.
- **Human host:** Accidental; larvae in **stomach/intestine**.

2. Mode of Transmission

- Consumption of **raw or undercooked fish** (containing larvae).

3. Clinical Manifestation

- **Acute abdominal pain** (often severe, mimicking appendicitis or gastric ulcer).
- **Allergic reactions** (urticaria, anaphylaxis).

4. **Diagnosis**

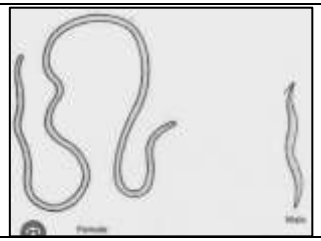
Method	Details
Gastroscopy/Endoscopy	Direct visualization and removal of larva
Surgery	For complicated cases (obstruction, perforation)
Serology	ELISA, Radioallergosorbent Test (RAST)

5. **Treatment**

- **Mechanical removal** of larva using **endoscopic forceps**.

- **Corticosteroids** (for allergic reactions).
- **Albendazole** (if removal not possible).

Dracunculus medinensis

Common name	Israeli worm, Dragon worm, Medina worm, Guinea worm, Fiery Serpent (of the serpent) worm
Disease	Drancunculiasis or Dracontiasis (accident disease)
Mode of Transmission	Ingestion of cyclops in contaminated water
Definitive Host	Man
Intermediate Host	Cyclops, Copepods, Watercress
Adult	Male – diffiucult to demonstrate as they die after fertilization • 21 by 0.4mm • Anterior end coild itself at least once Female – looks like thick twine of thread • Viviparous • 840 by 1.5mm • Prominent rounded anterior end
Morphology	
Clinical Manifestations	• Allergic reactions • Bacterial infection leading to disability to even death • Painful ulcer at the site, where gravids demale lays larvae • Female worm produce blister, which is formed at the site, which worm comes out upon contact to water
Treatment	• Metronidazole • Thiabendazole
Worm Removal	Worm Removal 1. Place affected body part on cool water. 2. Adult worm breaks through the blister and is eager to explore the outside world. 3. Clean the wound thoroughly. 4. Manual extraction by winding the worm throught the stick.
Prevention and Control	• Drink only clean water • Boil water before drinking if unsure • Do not drink the same water used in bathing • Filter the water using the filter mesh • Education

Visceral Larva Migrans

Common name	Toxocara canis – Dog Ascarid Toxocara cati – Cat Ascarid
.Naturally infect animals and accidentally parasitic to human producing disease	
known as Visceral Larval Migrans or Toxocariasis.	

CUTANEOUS LARVA

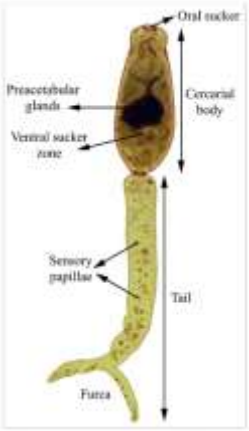
A. braziliense	Cat hookworm Possess pair of large teeth and pair of inconspicuous media teeth in the buccal cavity
A. ceylanicum	Smallest hookworm Common on cats and less frequent on dogs
Treatment	Albendazole Ivermectin

FILARIAL NEMATODES

Wuchereria - sheathed with no nucler at tip
Loa - sheathed: caudal nuclei continuous with those of trunk
Brugia - sheathed with 2 caudal nuclei
M. perstans - unsheathed: nuclei extending to tip of the blunt tail
M. ozzard - tail tapers to a thin filament containing column of 4-6 ovoid nuclei
Onchocerca - unsheathed: no nuclei (anterio and posterior)
M. streptocerca - unsheathed; posterior end is strong bent

- **Clinical Manifestations**
  - Tropica pulmonary eosinophilia (occult filariasis)
  - Elephantiasis; caused by comples immune reaction of long duration
  - Hydrocele/chylocele:accumulation due to lymphatic obstruction
  - Dermatolymohangioa denitis (DLA)L sign of active infection
- **Diagnosis**
  - Diethylcarbamazine citrate provocation test
  - Detection of CFA
  - Nucleopore filter
  - Knotts's concentration technique
  - Thick smear
- Treatment – diethylcarbamazine (DEC)

TREMATODE



•All trematodes are transmitted through **oral-fecal route EXCEPT Schistosomes**
•All trematodes are **hermaphroditic/monoecious EXCEPT Schistosomes**
•All trematodes are **operculated** EXCEPT Schistosomes
•All trematodes have **metacercaria** EXCEPT Schistosomes
•All trematodes **require two intermediate** host EXCEPT Schistosomes

1. GENERAL FEATURES

•Have integument used for absorption and secretion
•Adult flukes are flat, leaf-like, elongated and nonsegmented
•Have suckers
→ Oral: situated at the anterior end of the worm
→ Ventral or acetabulum: located on the ventral surface posterior to the oral sucker

- Incomplete digestive system has mouth but NO anal opening
- Intestine may be branched/dendritic, or simple
- Parasitic trematodes are hermaphroditic EXCEPT Schistosomes
- Eggs may be mature or immature, and consists of vitelline cells and membrane, and shell.
- Most are operculated EXCEPT Schistosomes

2. CERCARIAL SERIES

- Miracidia
  → Emerges from the egg
  → Ciliated
  → Swim in the water to seek first intermediate host
- Sporocyst
  → Saclike structure
  → Develop within intermediate host
- Rediae
  → Intermediate larval developing within the sporocyst
- Cercaria
  → Seeks second intermediate host
- Metacercariae
  → Consumed by human

3. DIAGNOSTIC STAGE

→ Embryonated/unembryonated egg

4. HABITAT

- Blood
  → *Schistosoma japonicum*
  → *Schistosoma haematobium*
  → *Schistosoma mansoni*

1. Schistosoma mansoni – Manson's blood fluke
- large, somewhat oblong, and possesses prominent large, lateral spine

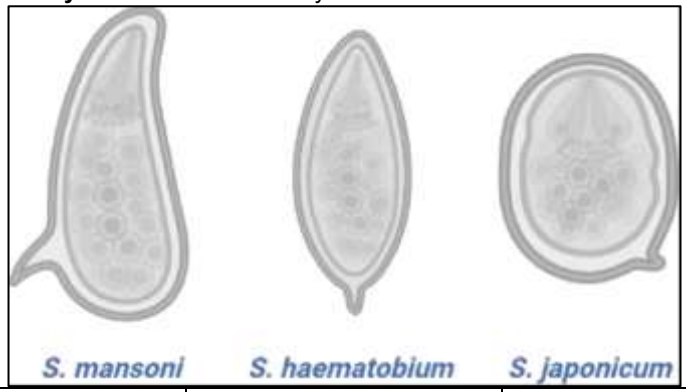
2. Schistosoma japonicum – Oriental blood fluke/East
- smallest of the Schistosoma spp.
- presence of a small lateral spine

3. Schistosoma haematobium – Versicle bladder fluke
- resembles S. mansoni in size and shape

Presence of large, prominent, terminal spine, that distinguishes it from other Scistooma spp.

Egg Morphology

- **Shape:** Electric bulb in shape / Flask-shaped
- **Spine:** Has a **terminal spine** (located at the end)
- **Color:** Bile stained (yellowish-brown)
- **Shell:** Operculated
- **Content:** Contains mature miracidium when oviposited
- **Hatching:** Does not hatch in water but is ingested with a molluscan host
- **Infectivity:** Infective to snails only



Species	Spine Location	Shape
S. haematobium	Terminal spine	Electric bulb/flask
S. mansoni	Lateral spine	Elongated
S. japonicum	Minute/absent spine	Oval/round

Stages

- **Egg**
  → Ovoidal
  → Rounded/pear shaped
  → Thin shell where tissues or RBCs adhere
  → Pale yellow
  → Curved hook/spine on the side near one of the polar end
  → Immature eggs do not mature in the soil
  → Hatch only in relatively clean water with sufficient oxygen
- Miracidium
  → Apical papilla

→ Epidermal plates covered with cilia
→ Permitive gut
→ Pair of cephalic unicellular penetration glands opening by a duct at the base of the apical papilla
→ Two pairs of flame cells
→ Germinal cells

DEVELOPMENTAL STAGES – disappears when penetration is complete

- First stage (mother) sporocyst
  - → Develops near the point of entry
  - → Mother sporocyst: elongated sac filled germinal cells
  - → Develop into daughter sporocyst
  - → Migrate to connective tissues to the liver
  - → Cercariae are produced
- Second stage (daughter sporocyst)
- Cercaria

→ Emerged from daughter sporocyst and escape from the snail into waters
→ Has body and forked tails
→ Has oral sucker and a small ventral sucker
→ Penetration is mediated by lytic enzymes secreted by cephalic glands

- Schistosomulum

→ After skin penetration
→ Survive in serum or physiologic saline at 37 degress Celsius
→ Found in pleural cavity on the 2nd day of infection
→ In the parenchyma on the 6th day
→ Branches of portal vein later

- Adult schistosome

→ Have separate sexes

MALE

→ Shorter but sturdier sex
→ Has gynecophoral canal where female is held
→ Testes are arranged in one row above the ventral sucker

FEMALE

→ longer sucker, ventral sucker and gonophore
→ Suckers aid in movement; enables the fluke to maintain their position inside the

- Single pyramidal ovary is located in the midline

DIAGNOSIS

SPECIMEN COLLECTION FOR SCHISTOSOMA SPECIES (TREMATODES)

- **For S. mansoni and S. japonicum:**
  - Specimen of choice: Stool
  - Method: Recovery of eggs in stool or rectal biopsy specimens.
- **For S. haematobium:**
  - Specimen of choice: Urine
  - Method: Concentrated urine specimens (preferably collected around midday).

IMMUNODIAGNOSIS FOR SCHISTOSOMA SPECIES (TREMATODES)

1. **Intradermal Test**
   - Detects immediate cutaneous hypersensitivity.
2. **Indirect Hemagglutination Test**
   - Uses adult worm and egg antigens.
3. **Circumoval Precipitin Test (COPT)**
   - Specific serologic test for schistosomiasis.

Laboratory Diagnosis

- **Specimen of choice:** Sputum
- **Method:** Recovery of eggs in sputum specimens
- **Other Methods:**
  - **Radiographs:** Aid in diagnosis (shows pulmonary lesions/cysts)
  - **Multi-dot ELISA:** Serologic test for antibody detection

EPIDEMIOLOGY

- **Global Distribution:** Has a global distribution (endemic in parts of Africa, South America, and Asia).
- **In the Philippines:**
  - Leyte
  - Samar
  - Mindoro
  - Davao
  - Sorsogon
  - Camarines

Intermediate Host

- **First Intermediate Host:** *Brotia (Antemelania asperata)* snail
- **Larval Development:** Miracidium develops into sporocyst and redial stages of development within the snail.

Second Intermediate Host

- **Crab Species:** *Sundathelphusa philippina* or *Parathelphusa grapsoides* (formerly named)
- **Role:** Harbors the metacercaria that is infective to man

Treatment

- **Drug of choice:** Praziquantel

Dosage: 25 mg/kg body weight, 3 times a day for 3 days

- **Alternative drug:** Bithionol

Dosage: 15-25 mg/kg/day on alternate days for a total of 10-15 days

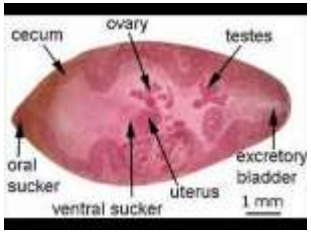
- Lungs

→ ***Paragonimus westermani***

- Oriental fluke
- Other species: *P. philippinensis, P. siamensis* (cats)
- Infective stage: Metacercariae
- Diagnostic: unembryonated egg in sputum or in feces

EGG MORPHOLOGY

- Size: 78-120 µm long; 45-60 µm wide
- Shape: Somewhat oval
- Shell: Surrounded by a thin, smooth shell
- Operculum: Prominent operculum with shoulders
- Contents: Contains undeveloped miracidium



ADULT
- 20-75 mm long, 8-20 mm wide
- Flat, leaf-shaped, blunt anterior end, undulating, tandem, dendritic testes
- Poorly-developed oral and ventral suckers, branched ovaries, vast vitelline follicles
- Can be distinguished from other fasciolas by a lack of cephalic cone or "shoulder" and the unbranched ceca

Adult Worm Morphology (*Paragonimus westermani*)
- **Reproductive System:** Hermaphroditic
- **Testes:** Lobed, deeply situated, located opposite each other
- **Ovary:** Located anterior to the testes
- **Suckers:** Ventral sucker is present
- **Cuticle:** Possesses spines (similar to other trematodes)

CLINICAL SYMPTOMS

Disease Names:
- Paragonimiasis
- Pulmonary Distomiasis
- Lung Fluke Disease
- Endemic Hemoptysis
- Parasitic Hemoptysis

Pathophysiology:
- **Cysteine Proteases** (cleave human IgG = evades immunity)

Symptoms:
- Cough
- Fever
- Chest pain
- Increased production of blood-tinged sputum

Neurologic Condition (Cerebral Paragonimiasis):
- Infection may develop to serious neurologic involvement
- Patients may experience:
  - Seizures
  - Visual difficulties
  - Decreased precision of motor skills

Other Manifestations:
- Chronic bronchitis
- Eosinophilia
- Production of fibrous tissue

- **Intestines**
  → ***Fasciolopsis buski***
  - GIANT/LARGE INTESTINAL FLUKE
  - Yellow ellipsoidal, thin, shell, operculated, filled with yolk cells
  - 130-160 mm long; 70-90 mm wide
  - **Infective Stage**: Metacercaria (to definitive host)
  - **Intermediate Host:** Snail (Segmentina, Hippeutis, or Planorbis)
  - **Reservoir Host**: Pig
  - **Mode of Transmission**: ingestion of water or raw vegetation of contaminated with the cyst
  - **Symptoms**: allergic reaction, anemia (pale skinetc.), ascites (accumulation of fluid in the peritoneal cavity), diarrhea, fever, obstruction of the bowel, stomachache, swelling of the skin, toxemia
  - **Diagnosis**: DFS, water sedimentation method
  - **Treatment for Fasciolopsias:** praziquantel, mebendazole, thiabendazole, pyrantel pamoate, oxyclozanide, nitroxinil, hexachlorophene

Adult Characteristics of *Fasciolopsis buski*
- **Habitat:** In large infestations, they inhabit most of the gastrointestinal tract (starting from the stomach)
- **Reproduction:** Mature adults are hermaphroditic (having both male and female reproductive organs)
- **Fecundity:** Produce over 25,000 eggs per day

TREATMENT
- **Praziquantel** is the drug of choice
- **Oxamniquine** fro S. mansoni only

→ ***Echinostoma ilocanum***
- **Common name:** Garrison's fluke
- Collar spines (49-51 horsehoe shape) around their oral suckers
- Oral suckerslies at the center of the circumoral disk
- FIRST and SECOND Host: snail
- Echinostomiasis
  → *Heterophyes heterphyes*
- ***Von Siebold's Fluke***
- ***Small Fish Fluke***
- ***Heterophoid fluke***
- Smallest but deadliest fluke

Egg Morphology (*Heterophyes heterophyes*)
- **Size:** 30 x 15 µm (micrometers)
- **Shape:** Short ellipse
- **Shell:** Thick with operculum
- **Color:** Yellow-brown
- **Content:** Contains mature miracidium

Adult Worm Morphology (*Heterophyes heterophyes*)
- **Size:** Measuring 1.0 to 1.7 mm by 0.3 to 0.4 mm
- **Shape:** Minute teardrop-shaped flukes
- **Habitat:** Found in the small intestines of fish-eating birds and mammals
- **Liver**
  → ***Fasciola hepatica***

Fascioliasis
- Persons infected with ***Fasciola hepatica*** experience symptoms caused by the presence and attachment of the adult worm to the biliary tract.

Adult Worm Morphology (*Fasciola hepatica*)

- **Shape:** Narrow, oblong, flat worm
- **Suckers:** Oral sucker is slightly larger than the ventral sucker
- **Digestive System:** Blind intestinal ceca are simple and extend to the caudal region
- **Life Span:** 20-30 years

Symptoms include:

- Headache
- Fever
- Chills
- Pains in the liver area

DIAGNOSIS

Fascioliasis

- **Specimen of choice:** Stool (for recovery of eggs)
- **Limitation:** Eggs are indistinguishable from other species (particularly *Fasciolopsis buski*)
- **Additional information required:** Patient symptoms and travel history are necessary to diagnose the causative species

TREATMENT: DIAGNOSIS

Fascioliasis

- Specimen of choice: Stool (for recovery of eggs)
- Limitation: Eggs are indistinguishable from other species (particularly *Fasciolopsis buski*)
- Additional information required: Patient symptoms and travel history are necessary to diagnose the causative species

TREATMENT: Praziquantel, bithionol, triclabendazole

EPIDEMIOLOGY

Fascioliasis

- Distribution: Has a worldwide distribution
- Economic importance: Significant in livestock-raising countries
- In the Philippines:
    - The dominant species is *Fasciola gigantica*
    - Affects cattle and water buffaloes

→ *Fasciola gigantica*

→ *Clonorcha sinensis*

- Chinese liver fluke

→ *Opistorchis felineus*

→ *Opistorchis viverrini*

- Pancreas

→ *Eurythema pancreaticum*

1. *Onchocerca volvulus*

- Blinding worms
- Causes blindinf filariasis or “river blindess”, genital elephantiasis, ochocercal dermatitis, iridoclitis
- Habitat: subcutaneous tissues
- Diagnosis: skin nips (larvae)
  Unsheathed
- No nuclei (anterior and posteriori)
- Periodicity: nonperiodic
- Vector: Black fly (simuliu)

2. ***Loa Loa***

- Common Name: African Eyeworm
- Causes fugitive swelling/calabar
- Periodicity: Diurnal (12nn)
- Vector:
- Horseflies (Tabanidae)
- Deerflies (Chrysops)
- Mango Flies

3. ***Mansonella***

Mansonella perstans
Mansonella ozzardi
Mansonella streptocerca

- Nonperiodic
- Vector: Black fly (Simulium), Midges (Calicoides)
-

SPECIMEN COLLECTION

A. EXAMINATION OF STOOL/FECAL SAMPLE

Purpose: Most common method for diagnosing intestinal parasites – demonstrates eggs, larvae, adults, trophozoites, cysts, or oocysts.

Collection:

- Container: Clean, wide-mouthed, waxed cardboard or plastic with tight lid.
- Quantity: Thumb-sized (formed) or 5-6 Tbsp (watery).
- Frequency: 3 specimens (every other day) or 3 in 10 days; amoeba: up to 6 in 14 days.
- Avoid: Urine, water, soil, toilet paper.
- PPE: Gloves, lab gown required.

Handling:

- Examine immediately (trophozoites die in 30-60 min).
- Refrigeration: OK for antigen testing only; kills trophozoites but not cysts/eggs.
- **Never freeze** or incubate.
- Drugs interfering with exam: Antacids, kaolin, mineral oil, barium/bismuth (7-10 days), antimicrobials (2-3 weeks), gallbladder dyes (3 weeks).

Labeling:

- Patient name, age, sex, date/time, physician, procedure, diagnosis, prior infections, travel history.

B. STOOL PRESERVATIVES

Preservative	Key Features
10% Formalin	All-purpose; 5% for cysts, 10% for eggs/larvae; buffered form preserves morphology
Schaudinn's Solution	For stained smears; contains toxic mercuric chloride
Polyvinyl Alcohol (PVA)	Adheres stool to slide; preserves cysts/trophozoites for staining; contains mercury
Merthiolate-Iodine-Formalin (MIF)	Fixative + stain; iodine unstable; difficulty in ID
Sodium Acetate-Acetic Acid Formalin (SAF)	Mercury-free; long shelf-life; staining less sharp than PVA
Ratio: 1 part stool : 3 parts preservative	

C. TECHNIQUES

C1. Direct Fecal Smear (DFS)

- 2 mg stool + drop of 0.85% NSS (for trophozoites) or Lugol's iodine (for cysts).
- Examine under low power.
- Micrometry: Measures cysts/ova for ID.

C2. Kato Thick Smear

- 50-60 mg stool, cellophane soaked in glycerine + malachite green.
- Glycerine = clearing agent; malachite green = pale green background.
- Best for thick-shelled eggs (*Ascaris, Trichuris*).
- Not for: Thin-shelled eggs (hookworm), watery stool, cysts/trophozoites.
- Examine within 10-20 min (hookworm eggs clear quickly).

C3. Concentration Techniques

Principle: Separate parasites based on specific gravity.

- **Sedimentation:** Parasites sink (higher SG).
- **Flotation:** Parasites float (lower SG).

Sedimentation Methods:

Method	Reagents	Best For	Notes
Acid-Ether	40% HCl + Ether	*Trichuris, Capillaria, Schistosoma* (animal stools)	May destroy cysts
Formalin-Ether/Ethyl Acetate	10% formalin + Ether/ethyl acetate	Helminth eggs + protozoan cysts	Ethyl acetate: better for cestode eggs/Giardia cysts; less efficient fat extraction

Flotation Methods:

Method	Solution	Best For	Notes
Zinc Sulfate	33% $ZnSO_4$ (SG 1.18-1.20)	General	May distort cysts/thin eggs
Brine	Saturated NaCl	Low-cost, simple	Shrinks hookworm/Schistosoma; not for operculated eggs
Sheather's Sugar	Boiled sugar + phenol	Coccidian oocysts (*Cryptosporidium, Cyclospora, Cystoisospora*)	Best with phase microscopy

C4. Stool Culture Methods

Purpose: Speciation of hookworm (eggs identical); differentiate hookworm vs. *Strongyloides*.

Method	Procedure	Notes
Copro Culture	Stool + moist soil/charcoal; Baermann harvest	Based on larval migration
Harada-Mori (Test Tube)	Stool on filter paper in tube with water; 10 days	Hookworm larvae go down; *Strongyloides* may go up; infective stage – caution!

Important: Do not refrigerate stools for culture.

C5. Egg Counting Procedures

Purpose: Assess intensity of infection (worm burden) and drug efficacy.

Method	Procedure	Notes
Kato-Katz	Sieved stool, template (20-50 mg), cellophane cover	WHO-recommended; multiply by factor; avoid overclearing (hookworm eggs disappear)
Stoll Egg Count	0.1 N NaOH diluent, displacement flask, Stoll pipette	Multiply by 100 for eggs/g

C6. Staining of Stool Specimen

- **For amebae & intestinal protozoans:** Iron-hematoxylin, trichrome, PAS, chlorazol black E.
- **For coccidian oocysts:** Kinyoun's acid-fast stain.
  - *Cryptosporidium*: 4-6 µm, spherical, pink/red.
  - *Cyclospora*: 8-10 µm, spherical, pink/red.
  - *Cystoisospora*: Ovoid, pink/red.

D. PERIANAL SWAB

Purpose: Recover eggs of *Enterobius vermicularis* and *Taenia* spp.

D1. Cellulose Tape (Scotch Tape) Method

- Sticky tape applied to perianal skin (early morning, before bath).
- Examine for eggs/adults.
- Toluene/xylene drop aids visualization.
- Repeat if negative.

Artifacts: WBCs, plant cells, fibers, Charcot-Leyden crystals, yeast, starch – may be mistaken for parasites.

Fecal Occult Blood Test: Guaiac pad + H_2O_2 → pseudoperoxidase reaction (blue color) – screens for colorectal cancer.

E-F. EXAMINATION OF BLOOD

Purpose: Detect parasites in blood (malaria, filaria, trypanosomes, *Babesia*).

F1. Finger-Prick Method

- Free-flowing blood (avoid tissue fluid dilution).

Preparation	Use
Wet/Fresh	Detect motile microfilariae/trypomastigotes (cannot speciate)
Thick Film	Screening (malaria, microfilariae); dehemoglobinized before staining
Thin Film	Species identification (malaria); methanol-fixed

Stains:

Stain	Features
Giemsa	Gold standard; RBCs pale red, nuclei purple
Wright's	Alcohol-based (no fixation needed); RBCs light red, nuclei blue
Delafield Hematoxylin	For microfilariae detail; permanent mounting

Capillary Tube Methods:

- **Buffy Coat Films:** Break tube at white cell layer; concentrate trypanosomes/Leishmania.
- **Quantitative Buffy Coat (QBC):** Acridine orange + UV; parasites fluoresce (malaria, microfilariae, trypanosomes, *Babesia*).

F2. Venous Blood Concentration

Method	Procedure	Use
Knott's Concentration	1 mL blood + 10 mL 2% formalin → centrifuge	Low microfilaremia
Membrane Filtration	Lyse blood with distilled water, filter	Traps microfilariae

G-H. EXAMINATION OF SPUTUM

Parasites Found: *Ascaris, Strongyloides*, hookworm larvae, *Paragonimus* ova, *Echinococcus*, *Entamoeba*, *Cryptosporidium*, *E. gingivalis*, *T. tenax*.

- **Best specimen:** First morning.
- **Induction:** 10% NaCl or H_2O_2 if unable to expectorate.

H1. Macroscopic Exam

- Consistency: Serous, mucoid, purulent, bloody.
- Color: Yellow (pus), greenish (*Pseudomonas*), bright red (fresh bleed), rust (hemoglobin breakdown).

H2. Microscopic Exam

- Wet mount (saline/iodine) for trophozoites.
- **Concentration:** Thick sputum + 3% NaOH → centrifuge → examine sediment.

I. EXAMINATION OF URINE

Parasites Found: *Trichomonas vaginalis*, *Wuchereria bancrofti* (chyluric samples), *Schistosoma haematobium* eggs.

- **Best specimen:** First morning (concentrated).
- **Method:** Centrifuge → examine sediment.
- *T. vaginalis*: Rounded, globular, jerky tumbling motility.

J. EXAMINATION OF TISSUE ASPIRATES

Site	Parasites
Liver	*Entamoeba* (aspirate from abscess wall, not center)
Duodenum	*Giardia, Strongyloides* (string test/Enterotest)
Skin	*Leishmania* (aspirate from ulcer bed; Giemsa stain → amastigotes)

K. EXAMINATION OF CEREBROSPINAL FLUID (CSF)

Parasites Found:

- Trypomastigotes: *T. cruzi, T. b. rhodesiense, T. b. gambiense*
- Trophozoites: *Naegleria*
- *Parastrongylus*

Important:

- Examine immediately (trypomastigotes die in 20 min; *Naegleria* motility lost).
- Centrifuge at 7000 g for 10 min → examine sediment.

L. EXAMINATION OF TISSUE BIOPSY MATERIAL

L1. Muscle Biopsy

- **Use:** *Trichinella spiralis* (encapsulated larvae), *Taenia solium* (cysticercosis), *Spirometra* (sparganosis).
- **Method:** Press small piece between slides → examine.

L2. Rectal Biopsy

- **Most common biopsy for parasites.**
- **Use:** *Schistosoma japonicum* eggs (deposited in tissue).