

Strongyloidiasis

Etiology • autoinfection • intestinal morphology • hyperinfection

Pathology lecture

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Learning outcomes

- Reconstruct skin-to-lung-to-intestine transmission and the two larval fates.
- Distinguish limited infection from hyperinfection on clinical and tissue findings.
- Explain why immunosuppression amplifies autoinfection and may produce enteric sepsis.
- Interpret a simplified intestinal biopsy and select relevant specimen types.

Strongyloidiasis is a nematode infection

Agent

- *Strongyloides stercoralis* is the named organism.
- A parasitic metazoan (helminth).

Anatomic focus

- Female worms occupy small-intestinal mucosa.
- Larvae migrate through lung and gut.

Key distinction

- Larvae may become infective inside the host.
- Autoinfection permits persistent disease.

Metazoal infections involve multicellular parasites

- Metazoa are multicellular eukaryotes with organ systems.
- Parasitic metazoa may enter by ingestion, skin invasion, or arthropod transmission.
- Their tissue distribution may extend beyond the gastrointestinal tract.
- Microscopic recovery of larvae or ova is a central diagnostic principle.

Endemic and sporadic strongyloidiasis

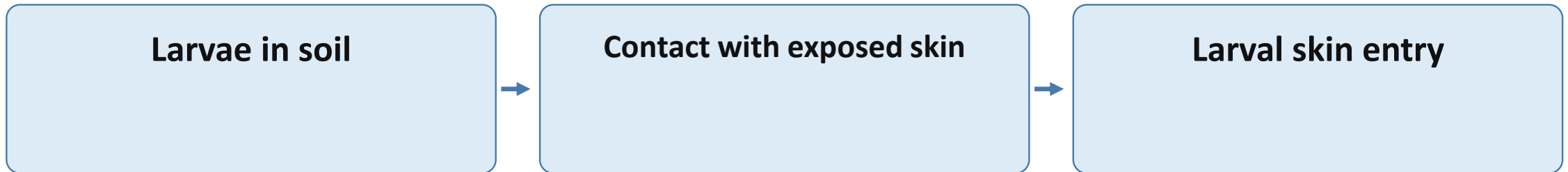
Geographic pattern	Distribution
Tropical / subtropical	Endemic in parts of South America, sub-Saharan Africa, and Southeast Asia.
United States	Sporadic infections are reported, including in Appalachia.
Interpretation	Exposure history is relevant beyond a single geographic region.

Global burden requires edition-specific context

- An estimated 30–100 million people are infected worldwide.
- This estimate describes worldwide infection burden—not prevalence within any individual region.
- No patient-level risk can be inferred from the worldwide estimate alone.

The range is a global estimate—not a region-specific rate.

Fecally contaminated soil is an exposure setting



- The parasite is environmentally associated with soil.
- The intestinal-stage offspring leave the host in stool and can contaminate soil.
- Soil contaminated by human feces sustains environmental exposure.

Infective larvae penetrate intact skin

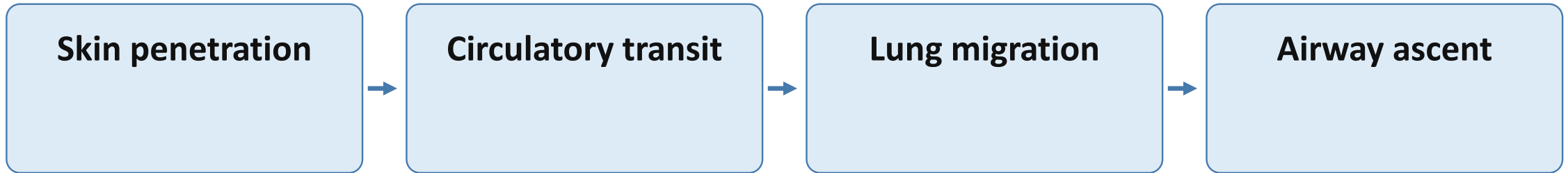
Entry route

- Exposure is through skin penetration by larvae.
- Skin penetration does not require an obvious break in the skin.

Clinical implication

- A skin route distinguishes infection from ingestion of fully developed intestinal adult worms.
- Cutaneous entry precedes systemic migration.

The early host route is a migration sequence

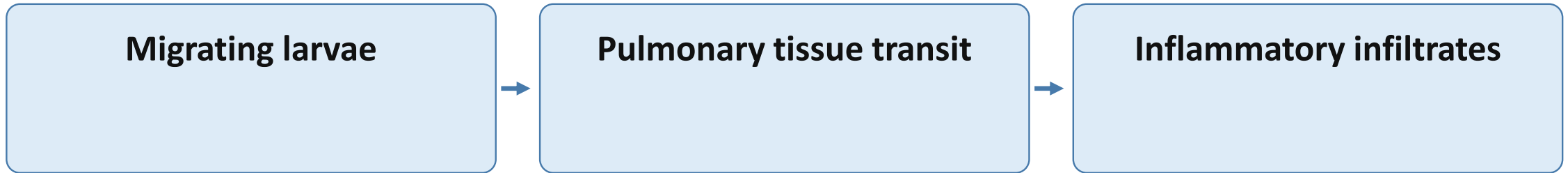


- Larvae enter through skin and travel in the circulation.
- They reach the lungs, then ascend the trachea to be swallowed.

Blood-borne movement precedes lung arrival

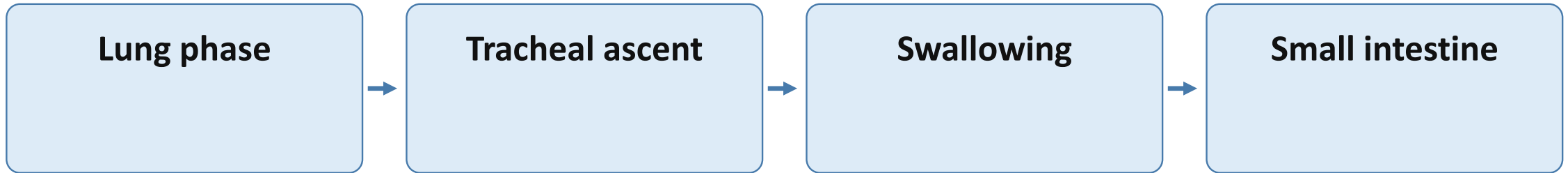
- Larvae move from skin into the circulation and onward to the lungs.
- The circulation is a migration route, not the usual final habitat of adult females.
- This sequence links a peripheral skin exposure to pulmonary findings.

Lung migration can provoke inflammation



- Migrating larvae can induce inflammatory infiltrates in the lungs.
- The pulmonary phase occurs before establishment of adult intestinal infection.

Airway ascent returns larvae to the gut



- Larvae ascend the trachea and are then swallowed.
- The airway route provides the connection between lung migration and intestinal residence.

Female worms reside in small-intestinal mucosa

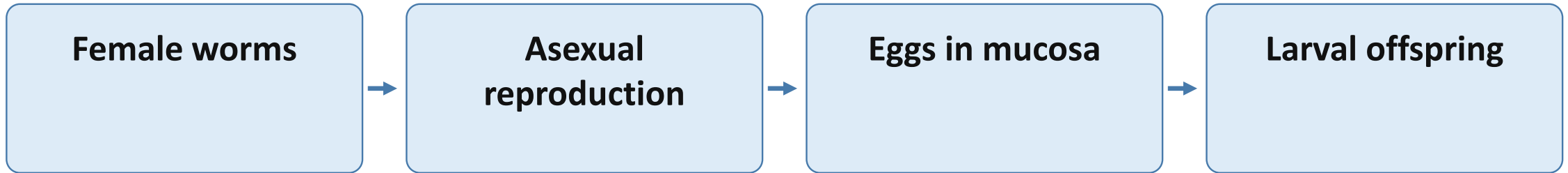
Habitat

- Female worms occupy the small-intestinal mucosa.
- In a biopsy, attention is directed to duodenal crypts.

Pathologic importance

- Intestinal residence allows egg production and generation of larvae.
- The bowel is also the site of host-tissue interaction.

Parthenogenesis is an intestinal reproductive mechanism



- Female Strongyloides produce eggs by parthenogenesis.
- The intestinal offspring may follow different routes rather than necessarily leaving the host.

Larval development creates two possible fates

Environmental branch

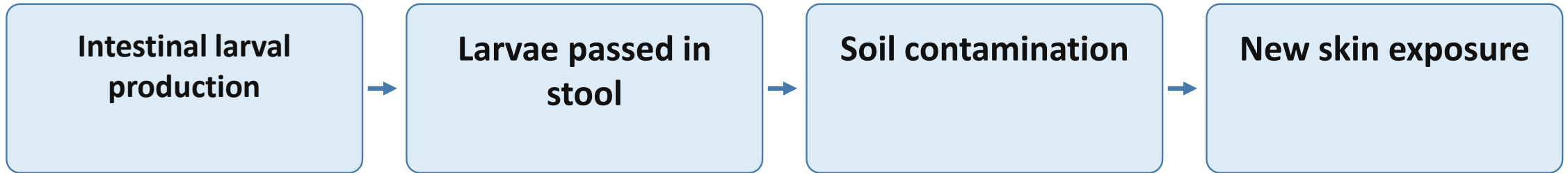
- Most larvae are passed in stool.
- Larvae may contaminate soil.
- Further exposure starts another infection.

Internal branch

- Some larvae develop into an infective form within the host.
- Internal infective stages drive autoinfection.
- This mechanism can repeat without a new soil exposure.

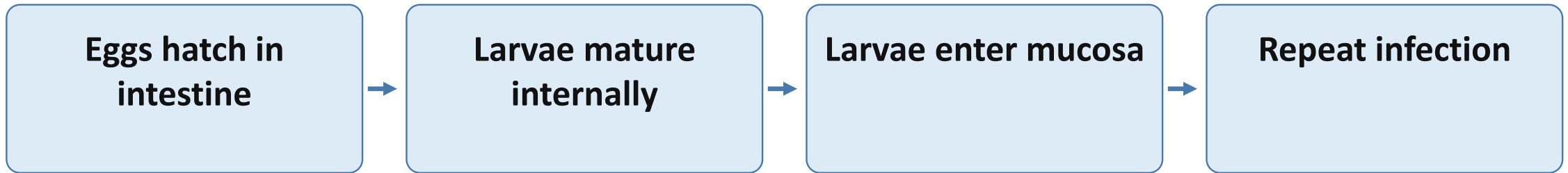
The fork in larval fate is the central pathogenetic feature.

Stool excretion completes the environmental branch



- Most intestinal larvae are passed in stool.
- A further environmental exposure can continue the infection cycle.

Autoinfection is development inside the host



- Strongyloides eggs can hatch within the intestine.
- The resulting larvae may penetrate intestinal mucosa without an obligatory external stage.

Autoinfection makes long-term persistence possible

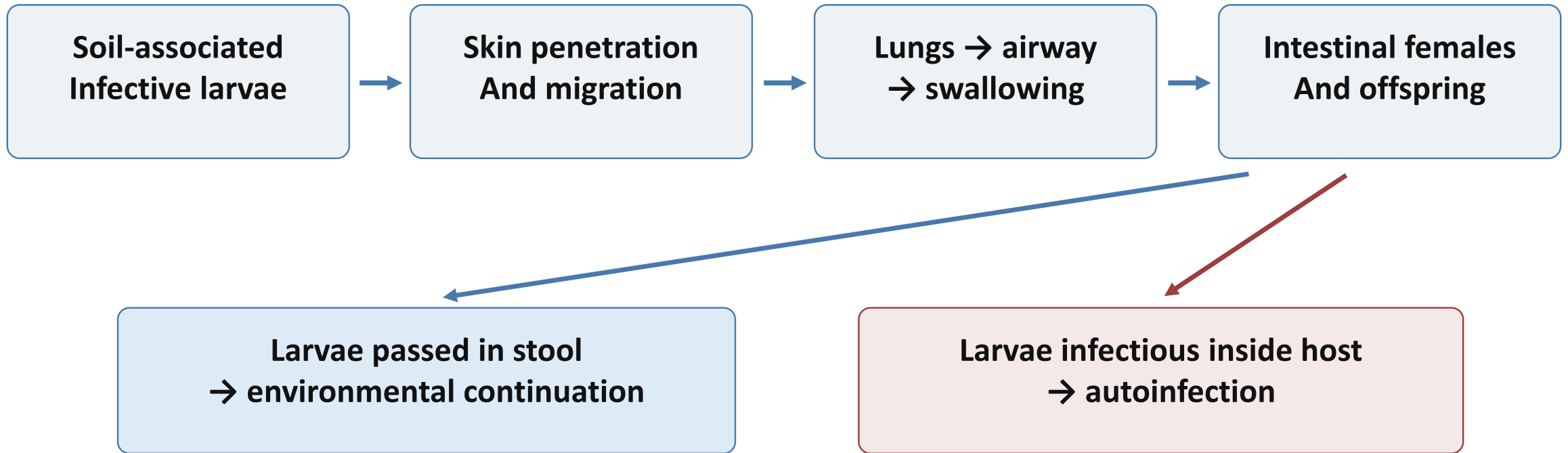
Persistence

- Internal recycling can maintain infection.
- Infection can persist for the lifetime of the host.

Consequences

- A remote exposure may remain biologically relevant.
- Host immune status may later change the course of infection.

The life cycle branches to external exit or autoinfection



Two branches arise from intestinal larval development

Strongyloides differs from many intestinal worms

Feature	Strongyloides	Other intestinal helminths
Infective development	May occur within the host	Other intestinal worms often require an outside stage
Chronicity	Autoinfection permits lifelong persistence	Persistent internal cycling is not the general model
Clinical hazard	Uncontrolled autoinfection in immunosuppression	This particular hazard is emphasized for Strongyloides

Contrast: Acquisition differs from ascariasis

Strongyloides stercoralis

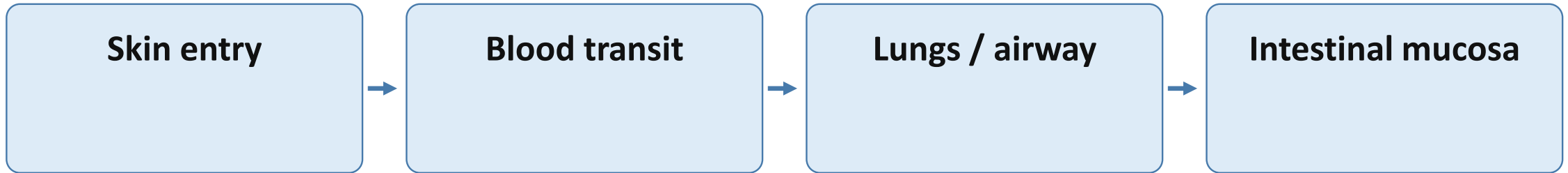
- Larvae penetrate the skin.
- Autoinfection can follow internal larval maturation.
- Biopsy may reveal larvae and adult females.

Ascaris lumbricoides

- Infection follows ingestion of eggs.
- Eggs from stool contaminate the environment.
- Intestinal infection may be established by identifying eggs in stool.

Entry stage and autoinfection distinguish these two nematodes.

Anatomy connects entry, migration, and disease



- Different anatomic sites reflect different stages of one infection.
- Mucosal residency establishes a site for chronicity; Deeper invasion characterizes severe disease.

Many immunocompetent infections are silent

- In an immunocompetent host, infection is frequently asymptomatic.
- Clinical silence does not demonstrate absence of intestinal parasites.
- The parasite may nevertheless persist by autoinfection.

Clinical severity and infection persistence are separate dimensions.

Uncomplicated disease may cause gastrointestinal symptoms

Diarrhea

- A listed intestinal symptom.
- Interpret in the context of potential helminth exposure.

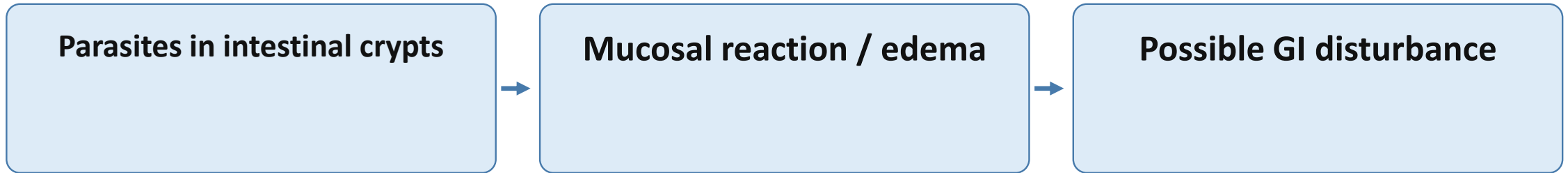
Bloating

- Another possible manifestation.
- Not specific by itself.

Malabsorption

- Malabsorption occurs occasionally.
- Can indicate intestinal functional involvement.

Mucosal disease explains gastrointestinal involvement



- Intestinal infection combines crypt parasites, lamina-propria inflammation, and edema.
- Linking structural changes to symptoms is a clinicopathologic interpretation, not a measured causal estimate.

Pulmonary manifestations track the larval phase

- Larval passage through the lungs can induce inflammatory infiltrates.
- The lung is part of the ordinary migratory trajectory.
- Pulmonary parasites during severe infection may also reflect a high organism burden.

Eosinophils signal a tissue response

Intestinal tissue

- Mild infection features eosinophil-rich lamina-propria inflammation.

Peripheral blood

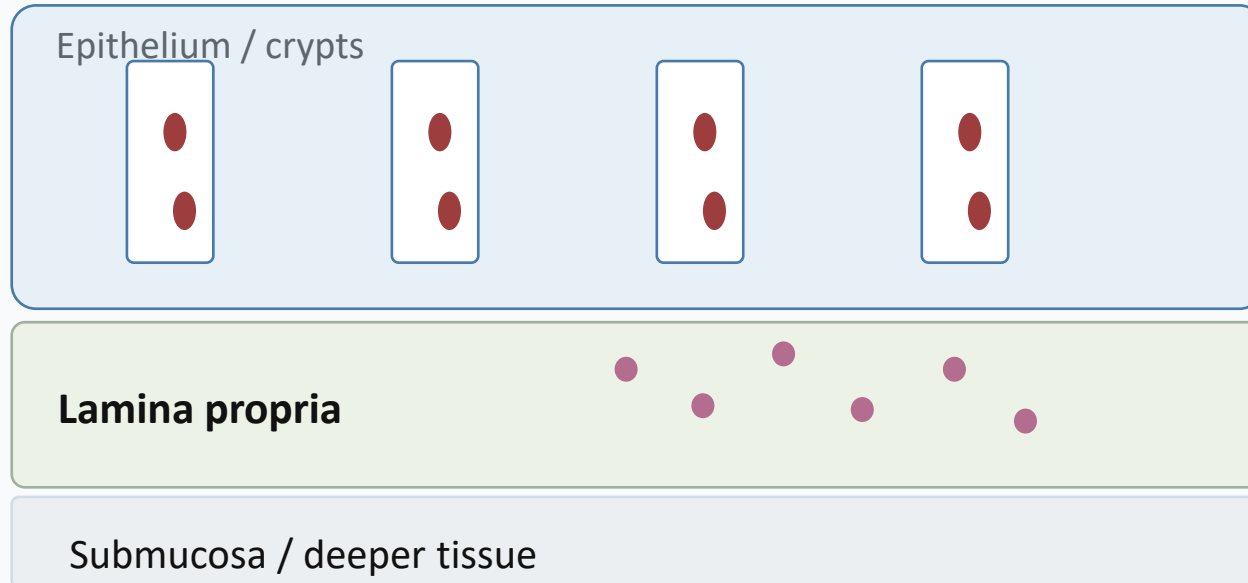
- Peripheral eosinophilia may occur.

Interpretation

- Eosinophils are a response to infection—not a stand-alone parasite identification.

Mild infection has a crypt-centered pattern

Simplified intestinal wall



No tissue-invasive larvae shown in mild infection

- Larvae predominate among visible worms.
- Parasites are located in duodenal crypts.
- The underlying tissue does not show invasion in mild infection.

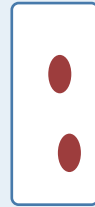
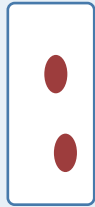
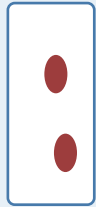
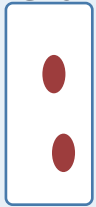
Duodenal crypts are the first histologic search zone

- The typical mild-infection location is within the crypt compartment.
- A crypt-limited organism distribution is different from stromal or vascular invasion.
- Correlate any candidate organism with the accompanying mucosal response.

Lamina propria shows eosinophil-rich inflammation

Simplified intestinal wall

Epithelium / crypts



Lamina propria



Submucosa / deeper tissue

No tissue-invasive larvae shown in mild infection

- An eosinophil-rich cellular infiltrate is described in mild infection.
- The infiltrate involves the lamina propria.
- Organism localization and inflammatory pattern should be documented separately.

Mucosal edema accompanies mild intestinal disease

- Mucosal edema may accompany crypt-centered intestinal infection.
- It accompanies the lamina-propria eosinophil-rich reaction.
- Edema is a tissue response; It is not itself proof of strongyloidiasis.

A negative deep-tissue finding has meaning

Mild infection

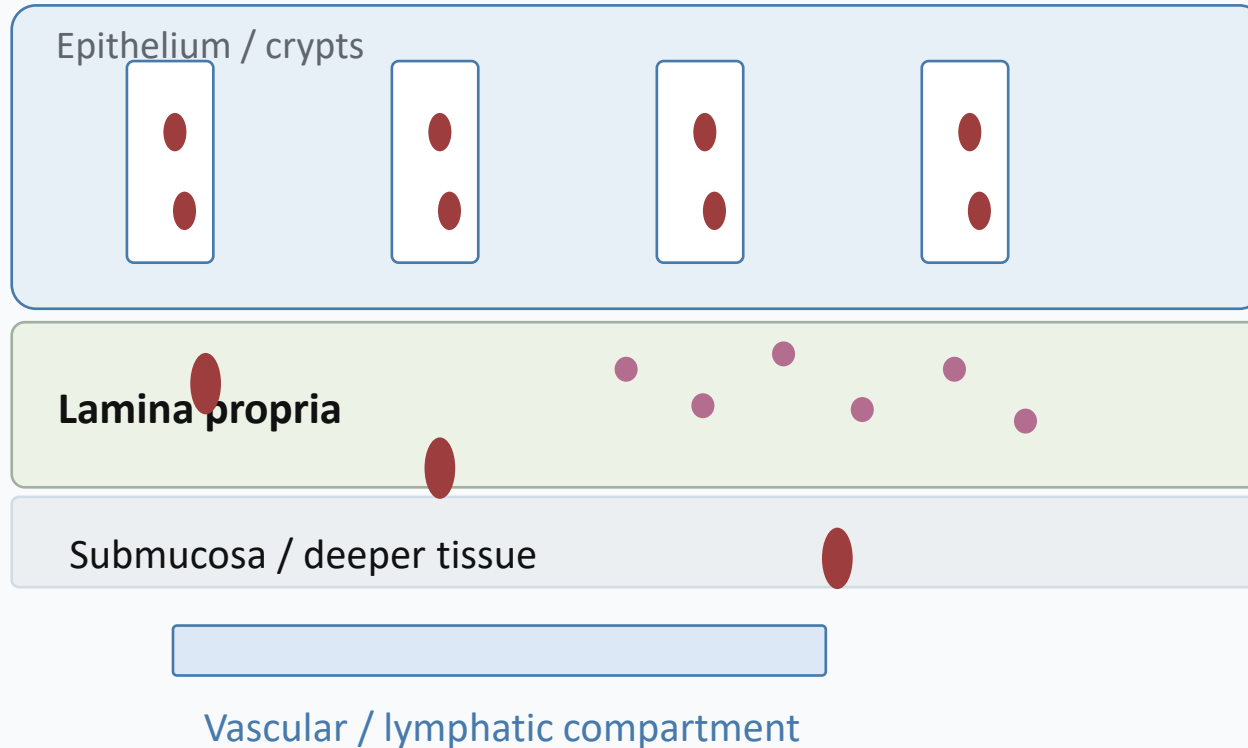
- Worms are in crypts.
- Underlying tissue is not invaded in the described mild pattern.
- Eosinophil-rich mucosa and edema may be present.

Hyperinfection

- Larvae enter submucosa.
- Lymphatics and vessels can contain invading parasites.
- Invasion is accompanied by a mononuclear infiltrate.

Morphologic interpretation requires compartment mapping

Simplified intestinal wall

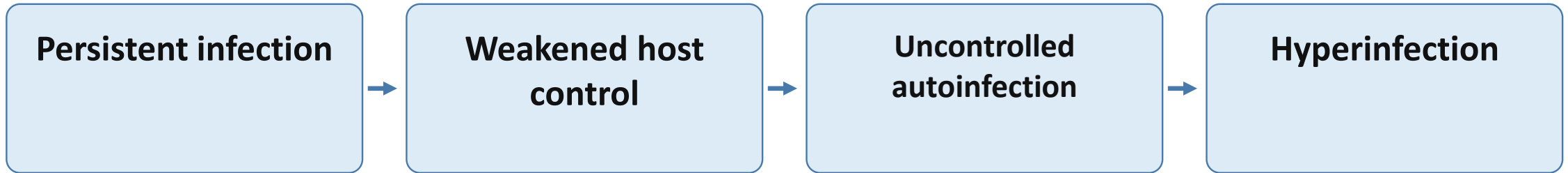


- First define crypt, lamina propria, and submucosal compartments.
- Then ask whether larvae remain luminal/crypt-associated or enter deeper tissues.
- Vessel involvement supports the invasive hyperinfection pattern.

Normal host defenses limit autoinfection

- Immunocompetent hosts frequently have limited or asymptomatic disease; Immunosuppression permits severe infection.
- Suppression of eosinophils and other responding immune cells weakens host control.
- A change in host defenses can convert persistent infection into high-burden disease.

Immunosuppression changes the clinical phenotype



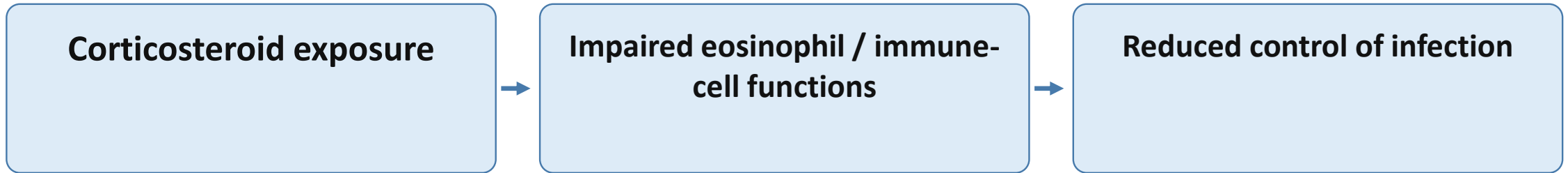
- Uncontrolled internal reinfection underlies extreme organism burdens.
- Severe hyperinfection can be fatal.

Prolonged corticosteroid therapy is a major risk

- Prolonged corticosteroid treatment is a prominent hyperinfection risk.
- Corticosteroids inhibit functions of eosinophils and other host immune cells.
- Corticosteroids also stimulate increased infective larval production by female worms.

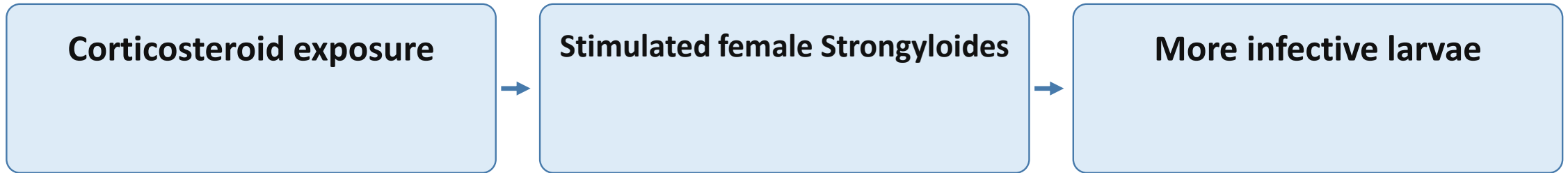
Corticosteroids affect host defense and infective larval production.

Corticosteroids suppress responding immune cells



- Eosinophils accumulate in tissues in response to Strongyloides.
- Suppression of immune-cell function is one mechanism linked to hyperinfection.

Corticosteroids may increase infective larval production



- Female worms produce more infective larvae in response to corticosteroids.
- This parasite-directed effect complements suppression of host immunity.

HTLV-1 infection is associated with severe infection

- Human T-lymphotropic virus type I is named as a risk context.
- The association reflects perturbed immune control mechanisms.
- The HTLV-1 association does not specify individual risk or exact mechanism.

Transplantation and lymphoma are additional risks

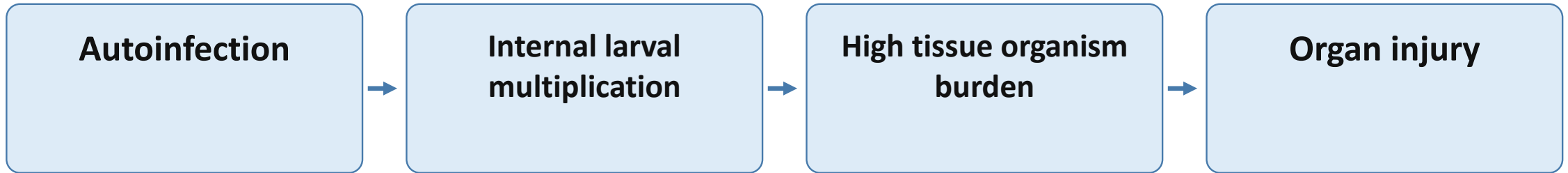
Organ transplantation

- A listed condition associated with increased hyperinfection risk.
- Immune control can be perturbed.

Lymphoma

- Also named among associated disease states.
- Risk should be interpreted in the clinical context.

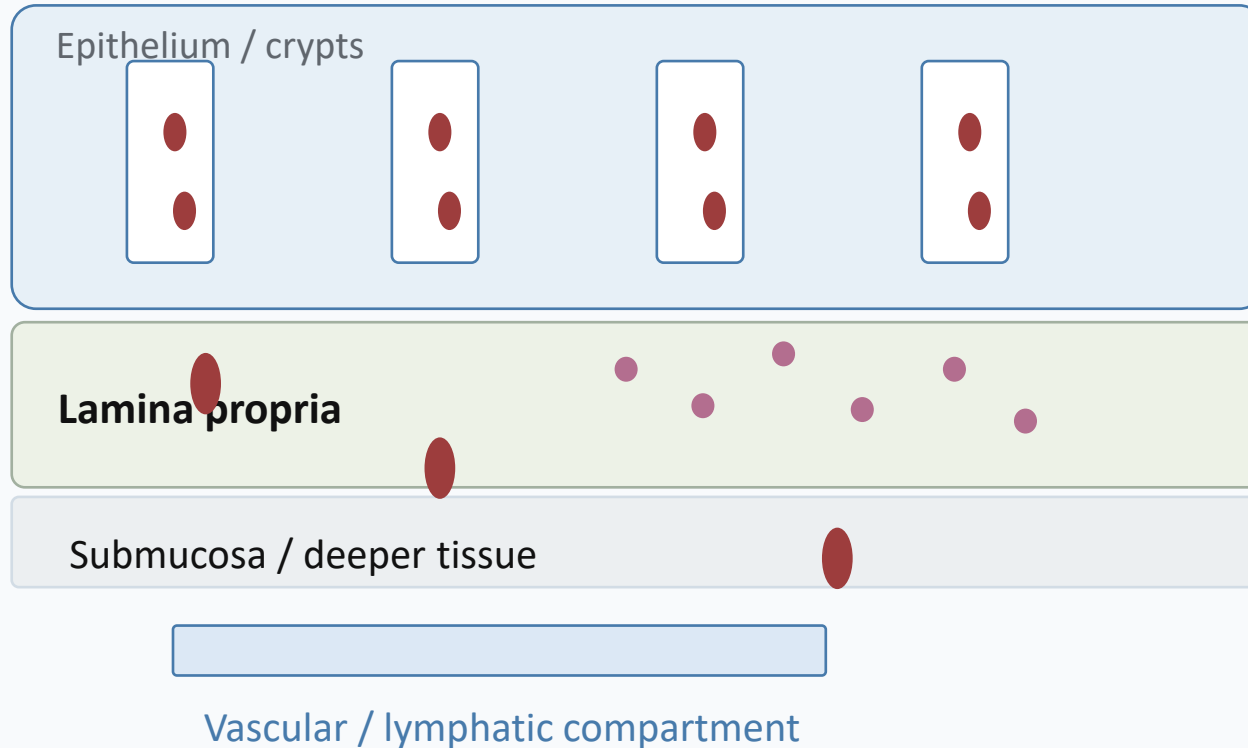
Hyperinfection amplifies a pre-existing pathway



- The cycle becomes uncontrolled in susceptible hosts.
- Severity derives from repeated internal infection and tissue invasion.

Hyperinfection yields numerous intestinal parasites

Simplified intestinal wall



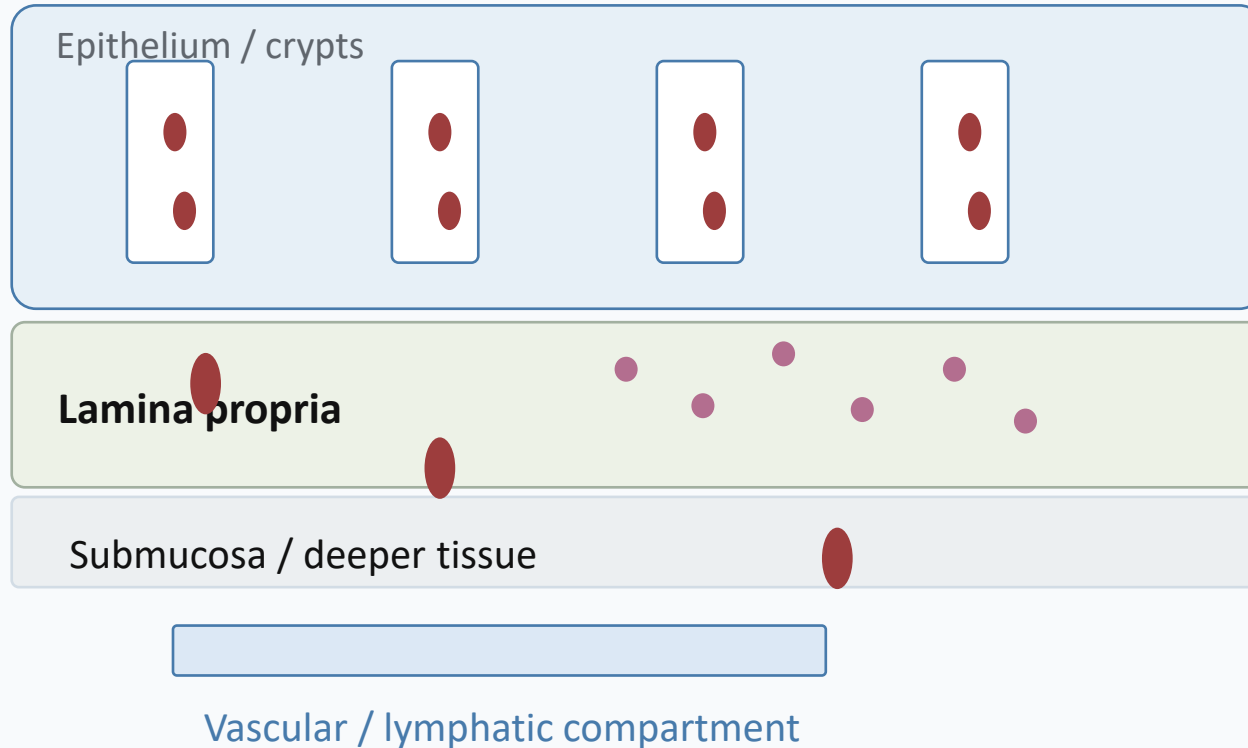
- Adults, larvae, and eggs become numerous in intestinal crypts.
- Hyperinfection can involve both duodenal and ileal crypts.
- Parasite stages should not be conflated when reading the section.

Duodenum and ileum may contain every parasite stage

- Hyperinfection increases the number of adult worms, larvae, and eggs visible in crypts.
- Both the duodenum and ileum are named in the hyperinfection description.
- This is a qualitative histopathologic distinction; No count threshold is supplied.

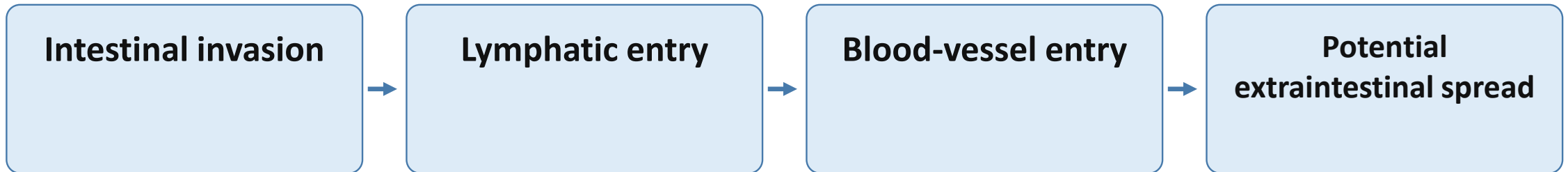
Hyperinfection invades colonic submucosa

Simplified intestinal wall



- Larvae can invade the colonic submucosa.
- Deeper location indicates departure from the mild crypt-only pattern.
- Submucosal larvae should be reported as tissue-invasive organisms.

Lymphatic and vascular invasion marks severity



- Larvae can enter lymphatics and blood vessels in hyperinfection.
- Filariform larvae can enter blood vessels and the muscularis mucosa.

The cellular reaction may become mononuclear

Limited intestinal infection

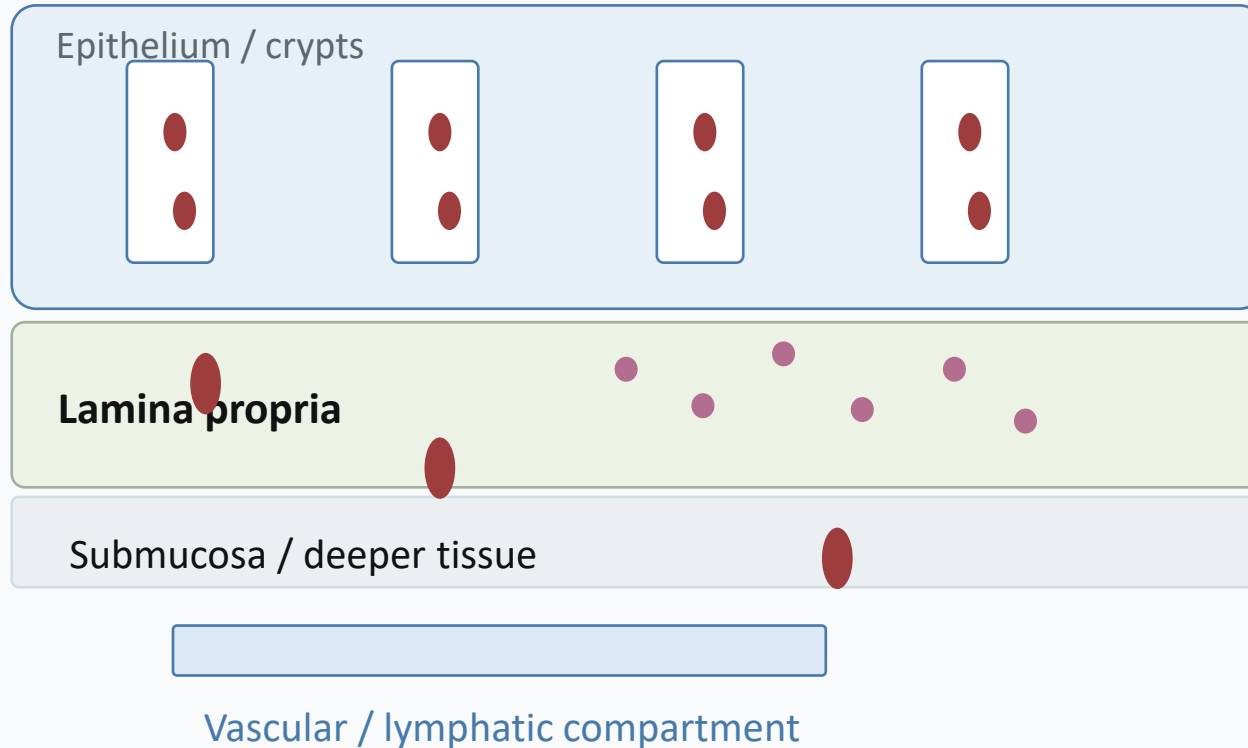
- Eosinophil-rich lamina propria.
- Mucosal edema.
- Crypt-associated larvae.

Hyperinfection

- A mononuclear infiltrate accompanies tissue invasion.
- Submucosal / vascular larvae may be seen.
- Markedly increased parasites across intestinal sites.

Crypt and vessel involvement in hyperinfection

Simplified intestinal wall



- Female worm, eggs, and rhabditoid larvae occur within duodenal crypts.
- Filariform larvae are entering vessels and muscularis mucosa.
- The described severe case occurred after high-dose cortisone exposure.

Hyperinfection can involve organs beyond bowel

Skin

- Parasites of multiple stages may be found.
- Evidence of disease outside the intestinal crypts.

Lung

- Parasites can be found in lung tissue.
- Respiratory specimens may carry many larvae.

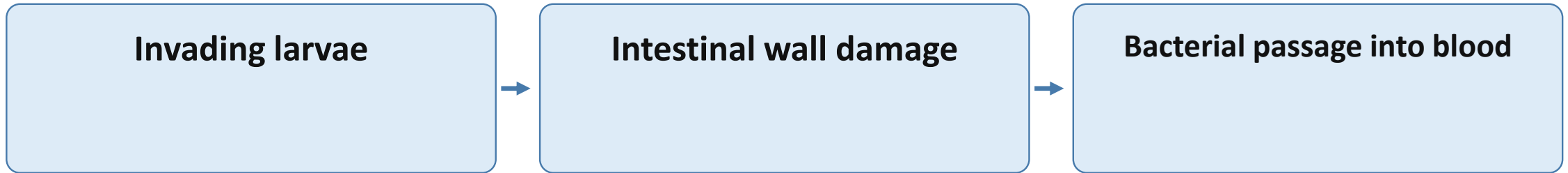
Other organs

- Different parasite stages may occur outside the gastrointestinal tract.
- No exhaustive organ list is supplied.

Large larval burdens may be visible in sputum

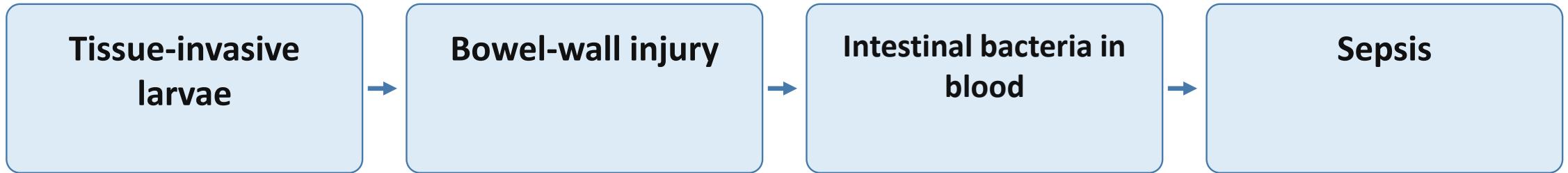
- Numerous parasites may occur in sputum during hyperinfection.
- Sputum is therefore a possible direct source of parasite detection.
- This finding illustrates extraintestinal parasite burden, not merely a nonspecific lung infiltrate.

Intestinal wall invasion can compromise the barrier



- Invading larvae can damage the intestinal wall.
- This damage provides a route for enteric bacteria to enter circulation.

Enteric bacterial sepsis is a serious complication



- Sepsis originates from intestinal bacteria in the stated mechanism.
- The microbial complication is secondary to parasitic intestinal injury.

Limited infection and hyperinfection are distinct

Feature	Limited / mild infection	Hyperinfection
Parasite burden	Usually fewer; mainly crypt larvae	Many adults, larvae, eggs
Tissue site	Duodenal crypts	Crypts plus submucosa / vessels
Inflammation	Eosinophil-rich lamina propria	Mononuclear infiltrate with invasion
Beyond intestine	Often little clinical illness	Other organs and possibly sputum
Clinical concern	GI symptoms or asymptomatic	Severe disease and bacterial sepsis

Case: Eosinophils and crypt-associated larvae

CLINICAL / HISTOPATHOLOGY VIGNETTE

A duodenal biopsy shows larvae restricted to crypts, lamina-propria eosinophils, and mucosal edema. No submucosal invasion is described.

Which disease pattern best fits the supplied morphology?

Mild intestinal strongyloidiasis; The organisms are crypt-limited rather than deeply invasive.

Case: Steroid exposure and systemic larvae

CLINICAL / HISTOPATHOLOGY VIGNETTE

A person treated with prolonged corticosteroids has numerous intestinal parasite stages; Larvae are identified in vessel-like channels and sputum.

What unifies the organ distribution and predisposing factor?

Uncontrolled autoinfection with hyperinfection; Corticosteroids reduce immune control and can stimulate infective larval production.

Case: HTLV-1 and an initially silent infection

CLINICAL / HISTOPATHOLOGY VIGNETTE

An adult with a history of soil exposure and no earlier gastrointestinal complaints subsequently develops severe infection in the context of HTLV-1.

Why does an earlier asymptomatic period not exclude disease?

Strongyloides may persist through autoinfection, and HTLV-1 is a listed risk context for hyperinfection.

Direct identification remains the diagnostic principle

Excretions

- Metazoal infection can be diagnosed by larvae or ova recovered in excretions.
- For Strongyloides, larvae may pass in stool.

Tissues

- Histologic sections may show worms / larvae and a tissue response.
- Site and stage distinguish disease severity.

Serology

- The metazoal overview lists serology as a diagnostic modality.
- No Strongyloides-specific assay characteristics are supplied.

The specimen should match the disease compartment

Specimen or site	Expected finding	Interpretive role
Stool	Most larvae exit in stool	Shows environmental exit and an excretion-based detection opportunity
Duodenal tissue	Larvae; in severe disease adults and eggs	Identifies crypt-level infection and reaction
Colon submucosa / vessels	Invasive larvae	Supports tissue-invasive severe infection
Sputum	Numerous larvae in hyperinfection	Can demonstrate respiratory involvement

Identification requires more than eosinophilia

Supportive reaction

- Lamina-propria eosinophils.
- Peripheral eosinophilia may occur.
- Mucosal edema can accompany infection.

Direct parasitic evidence

- Parasites seen in crypts or tissue.
- Larvae passed in stool.
- Larvae may be recovered from sputum in severe disease.

An inflammatory pattern suggests context; An organism establishes its presence.

Do not conflate Strongyloides with Ascaris

Question	Strongyloides	Ascaris
Entry	Skin penetration by larvae	Ingestion of environmental eggs
Characteristic recycling	Host autoinfection possible	No parallel mechanism described
Reported stool stage	Larvae commonly passed	Egg detection is diagnostic
Major severe pattern in excerpts	Autoinfection hyperinfection	Obstruction / biliary complications with heavy burden

A pathologist should report depth of invasion

Organisms	Location	Host response
• What stages are visible? • Larvae only, or adults / eggs too?	• Crypts or deeper submucosa? • Lymphatic / vascular involvement?	• Eosinophil-rich edema? • Mononuclear infiltrate associated with invasion?

These are reporting prompts derived from the described morphology.

Host context changes interpretation of the biopsy

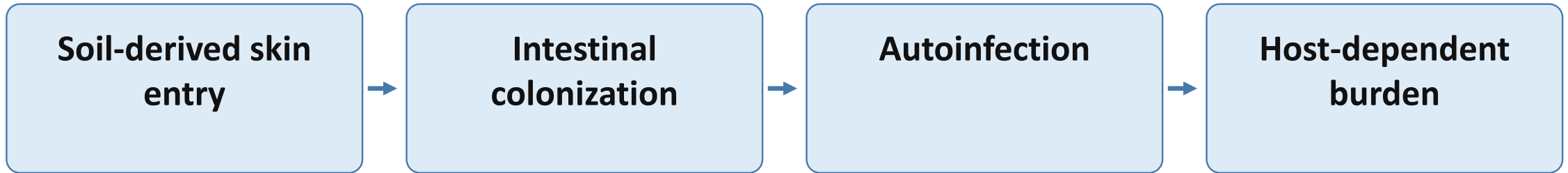
CLINICAL / HISTOPATHOLOGY VIGNETTE

Two biopsies show duodenal *Strongyloides* larvae. One patient is clinically well; The other is receiving high-dose corticosteroid therapy.

Which additional histologic and clinical findings require attention?

In the steroid-exposed patient, investigate higher worm burden, eggs/adults, deep or vascular invasion, extraintestinal parasites, and enteric sepsis.

Synthesis: The central causal pathway



- Limited infection: Crypt larvae, eosinophil-rich lamina propria, edema.
- Hyperinfection: Invasive larvae, widespread parasites, and possible bacterial sepsis.

Four histopathologic conclusions

- In mild disease, duodenal crypt parasites accompany eosinophil-rich mucosal inflammation.
- Autoinfection permits lifelong persistence without an obligatory new exposure.
- Immunosuppression—especially corticosteroids or HTLV-1—can lead to hyperinfection.
- Deep invasion, extraintestinal larvae, and enteric sepsis define major severity concerns.

Source references and figure limitations

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th edition (2026). Chapter 8, Strongyloidiasis, pp. 361–362.
- Same textbook, Chapter 17, Parasitic Enterocolitis—Strongyloides, p. 725.
- Original Fig. 8.57 was described in the supplied text, but the figure image itself was not available in the provided files.
- All diagrams in this deck are editable explanatory schematics, not original microscopy or reproduced textbook figures.