

Ascaris lumbricoides

Pathology of ascariasis

Life cycle · Tissue response · Clinicopathologic consequences · Diagnosis

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Learning objectives

- Trace the intestinal–pulmonary–intestinal life cycle and explain its diagnostic consequences.
- Connect larval migration with eosinophilic pneumonitis and adult worms with luminal obstruction.
- Recognize the major intestinal, appendiceal and hepatobiliary complications.
- Distinguish ascariasis from other intestinal nematode infections using pathogenesis and tests.

Parasitic enterocolitis: The relevant context

Enteric parasite groups

- Nematodes: Ascaris, Strongyloides, hookworms, pinworms
- Cestodes and trematodes also affect the bowel
- Protozoa constitute another major group

Ascariasis focus

- Large intestinal nematode infection
- Distinctive obligatory larval lung migration
- Disease from migrating larvae and adult worm burden

Ascariasis is a nematode infection

Ascaris species colonize the human intestinal lumen as adults.

A. Lumbricoides causes most human ascariasis.

A. Suum also causes a smaller proportion of cases.

The larval phase traverses tissues before intestinal maturation.

Human and pig-associated Ascaris

A. Lumbricoides

- Eggs passed by infected humans
- Human fecal contamination seeds exposure
- Predominant named cause

A. Suum

- Eggs passed by pigs
- An additional zoonotic source of infection
- Less common human infection

Global distribution and burden

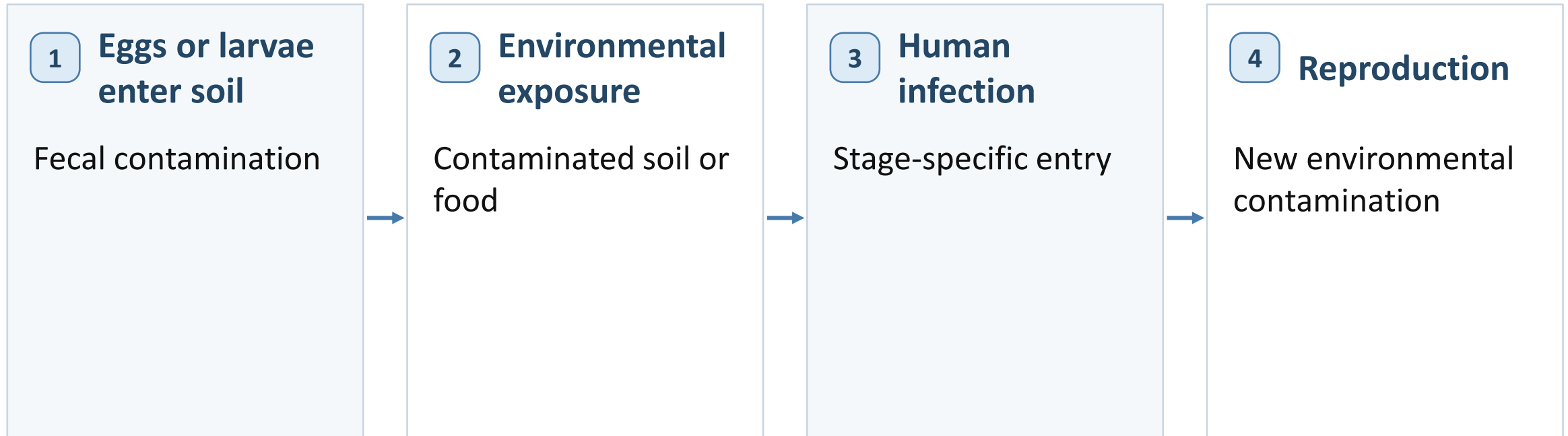
Approximately one billion infections worldwide.

Most infections occur in Asia, Africa and South America.

The estimate describes disease burden in the source; It is not a live surveillance statistic.

Unequal environmental sanitation sustains exposure.

Soil-transmitted helminths share an exposure ecology



Entry route distinguishes nematode groups

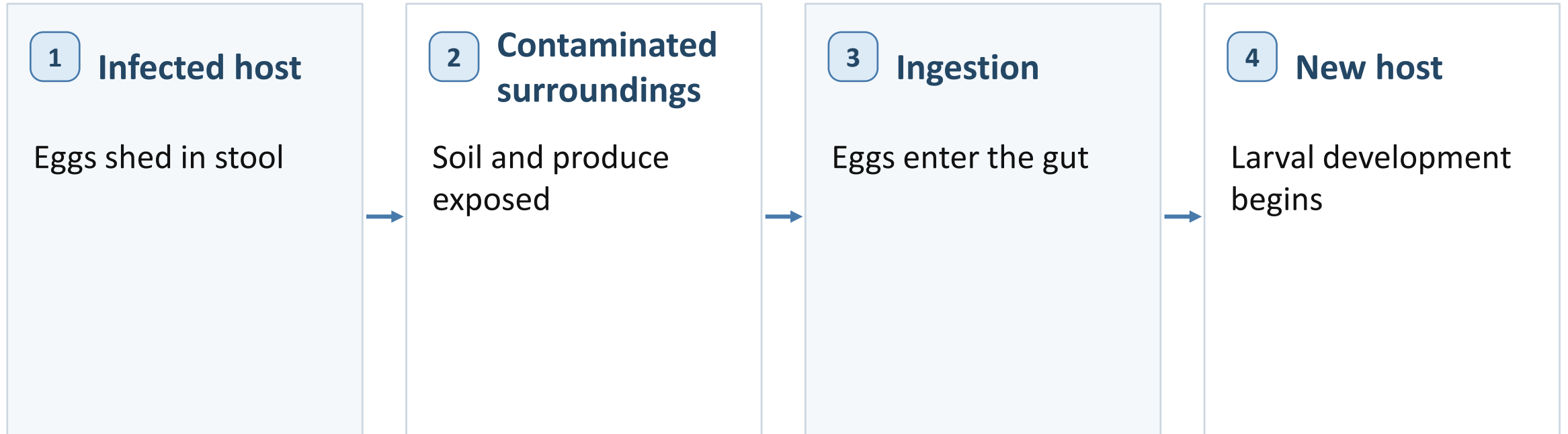
Ingestion of eggs

- Ascaris: Swallowed eggs
- Trichuris: Soil-transmitted whipworm
- Enterobius: Egg ingestion, especially from contaminated hands

Larval skin penetration

- Strongyloides: Infective larvae penetrate intact skin
- Hookworms: Entry begins through skin
- All do not share the same intestinal behavior

Fecal–oral acquisition of Ascaris



Contaminated produce provides an exposure route

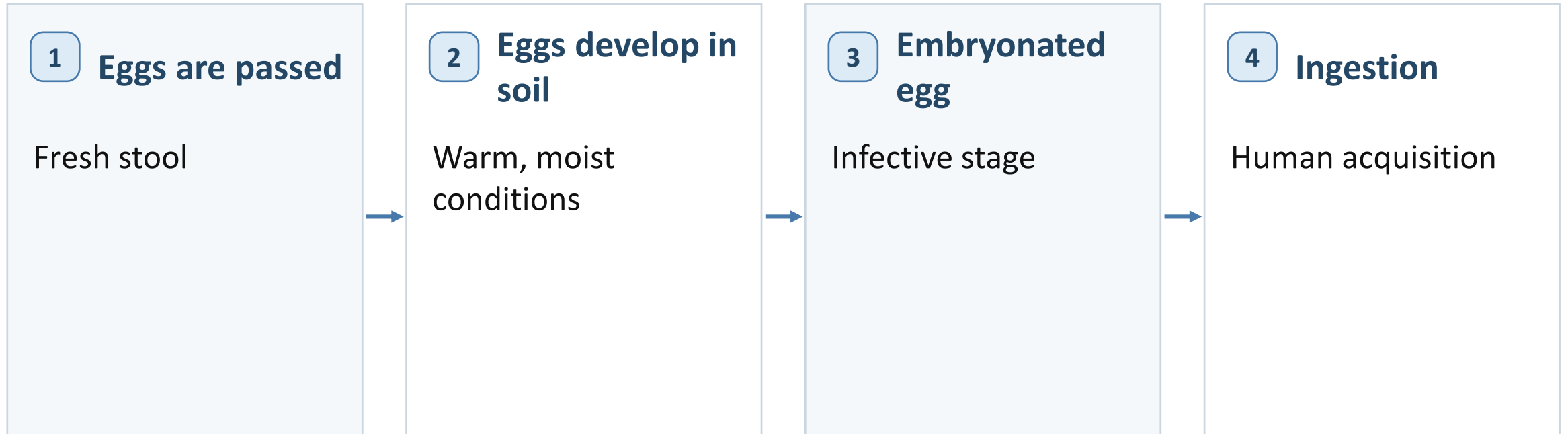
Poorly cleaned or peeled produce may carry eggs from contaminated soil.

The parasite is not acquired from eating adult worms.

The exposure route links sanitation, soil and ingestion.

Environmental contamination precedes infection in a new host.

Infective stage develops outside the host



Eggs passed $\neq$ immediate infectiousness

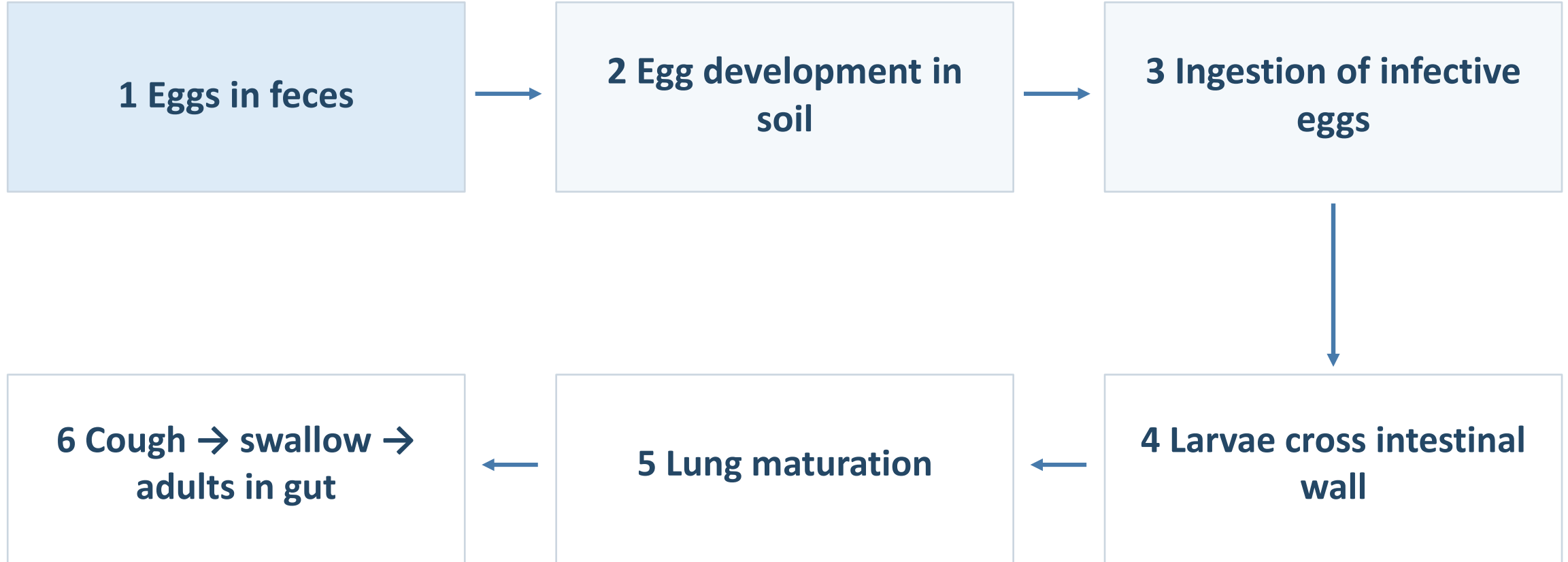
At excretion

- Eggs leave the intestine with feces
- Developmental maturation remains necessary
- Direct handling of fresh stool is not the canonical infective stage

After environmental development

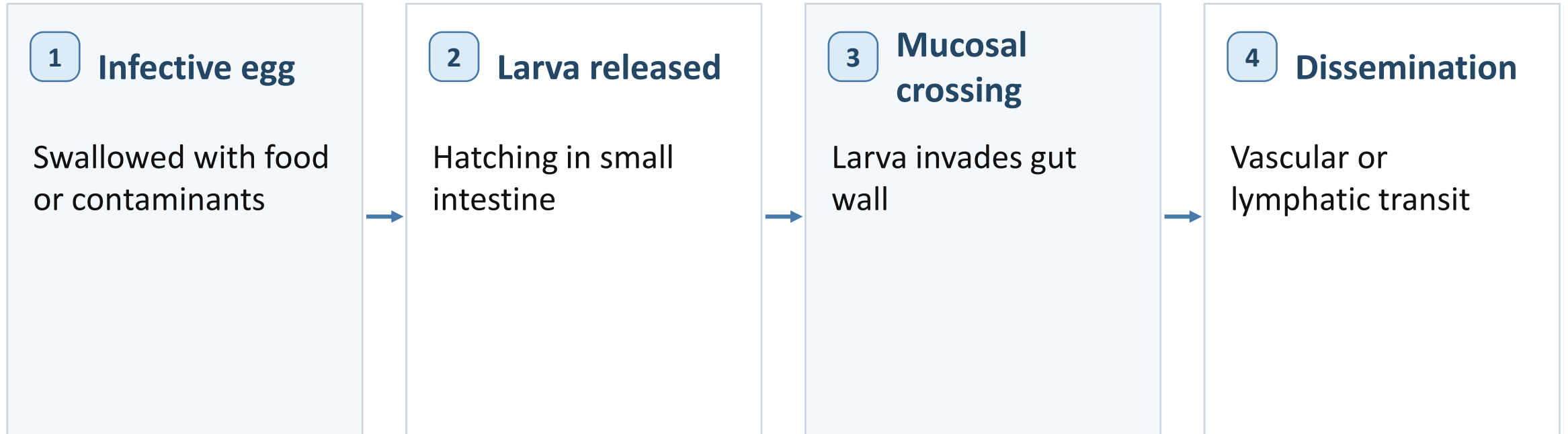
- Fertilized eggs become embryonated
- Ingestion of infective eggs starts a new cycle
- Environmental sanitation interrupts maturation and exposure

Entire life cycle at a glance



Schematic: Sequence is conceptual, not a time-scaled or anatomic diagram

Hatching follows ingestion



Intestinal entry is a transient larval event

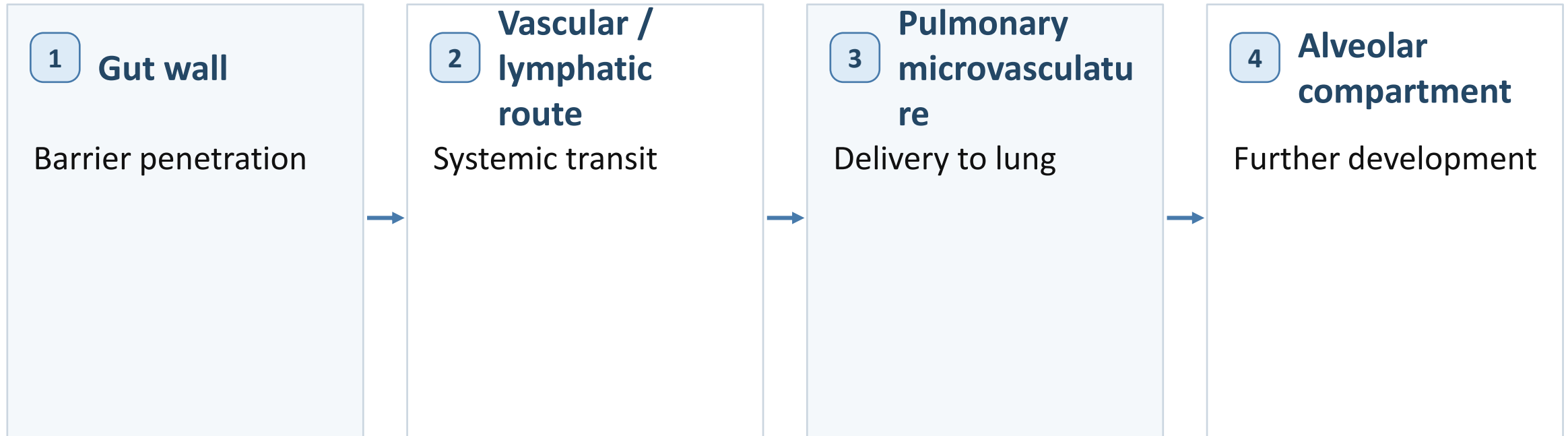
Larvae cross the intestinal mucosa before entering the circulation.

Chapter 8 specifically describes proximal colon and cecum penetration.

Chapter 17 summarizes the event as intestinal mucosal penetration.

The early tissue phase is not the same as adult intestinal residence.

Larval migration connects gut to lung



The larval and adult compartments differ

Early larva

Mucosa → circulation → alveoli



Adult worm

Intestinal lumen (predominantly jejunum)

Pulmonary maturation precedes return to the gut

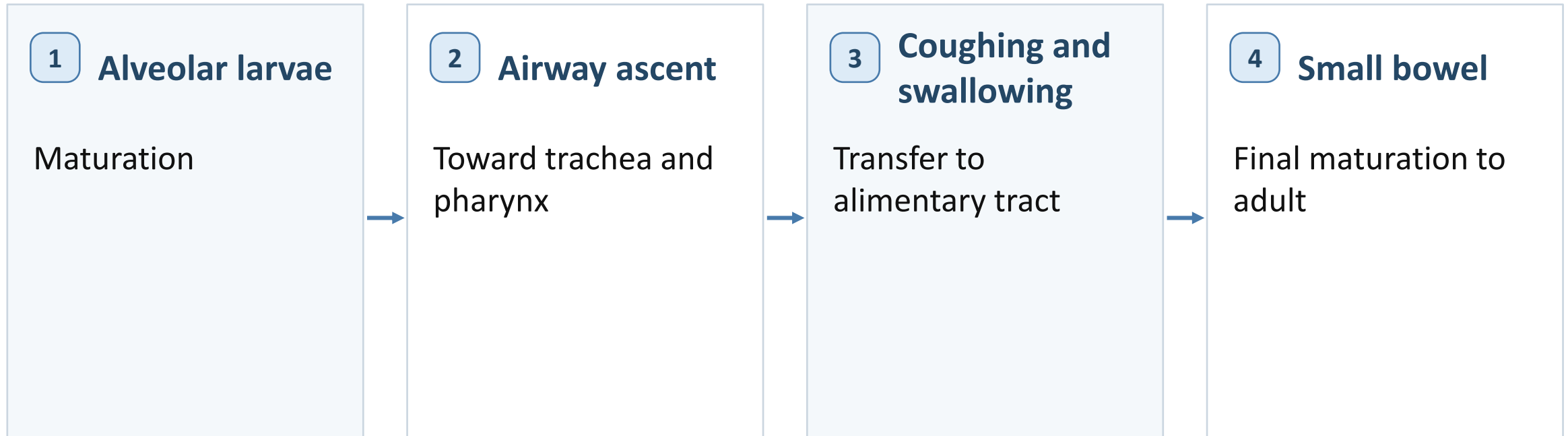
Larvae develop in the lungs for approximately 10–14 days.

They reach the alveolar compartment.

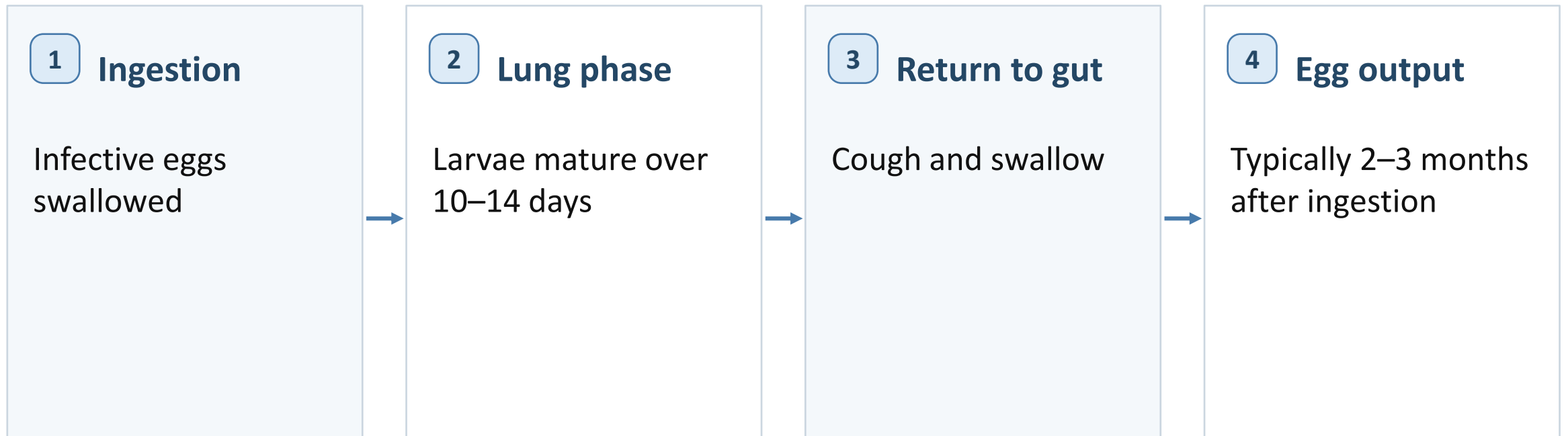
The pulmonary phase can induce inflammatory symptoms.

They subsequently ascend the airway before being swallowed.

Exit from lung depends on airway transit



Time course of development



Adult worms inhabit the intestine

Adults are found throughout the bowel, mainly the jejunum.

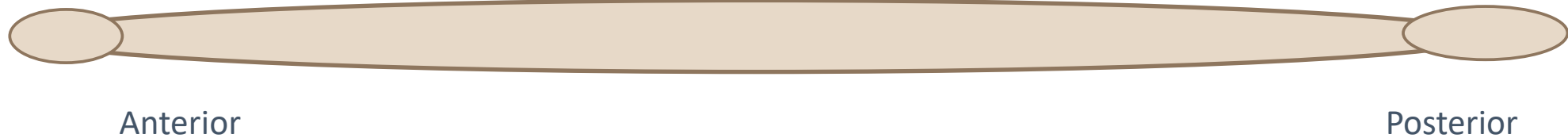
Worms return to the gut after pulmonary development.

The mature parasites occupy the intestinal lumen.

Heavy parasite burdens are linked to obstructive complications.

Adult morphology: Large intestinal roundworm

Intestinal luminal adult — schematic longitudinal outline



Adult worms measuring approximately 15–35 cm.

CDC differentiates generally larger female and smaller male worms.

Recognition of an expelled adult can establish infection.

High egg output perpetuates transmission

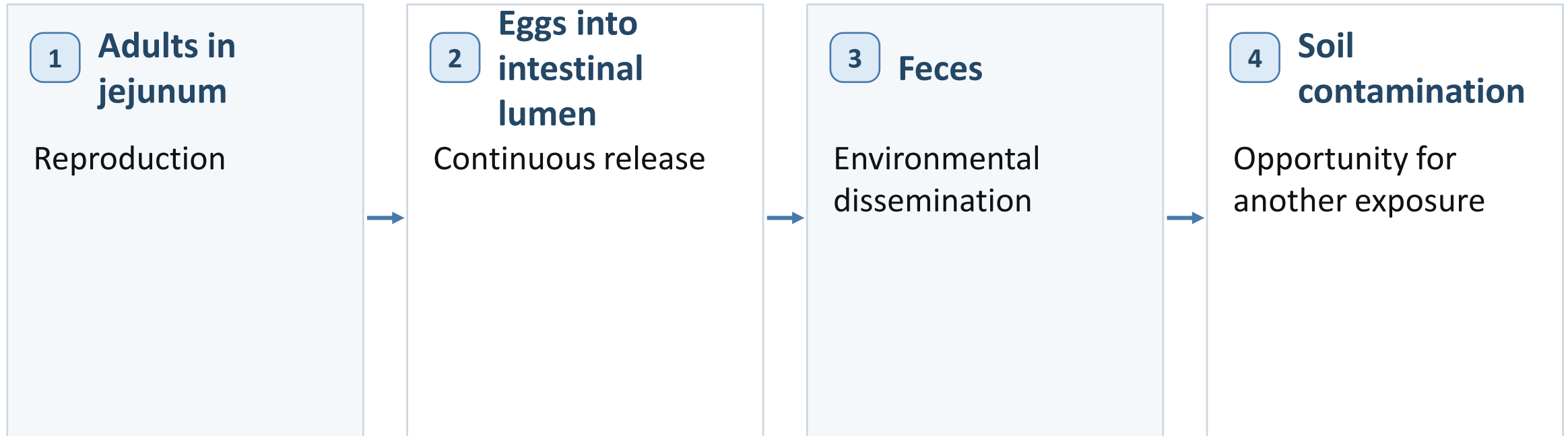
A female *Ascaris* may release approximately 200,000 eggs per day.

Eggs are shed in feces, contaminating the environment.

Production occurs once intestinal sexual maturity is reached.

The number is a biological source estimate, not an infection-intensity cut-off.

Egg shedding completes the environmental cycle



Stage determines symptoms and investigations

Larval migration

- Transient pulmonary inflammatory phase
- Dry cough, dyspnea and fever may occur
- Stool eggs may still be absent early

Adult intestinal phase

- Frequently clinically silent
- Pain or bowel obstruction with heavy burdens
- Stool egg detection usually establishes diagnosis

Clinical manifestations cluster by parasite stage

Stage	Dominant process	Clinical consequences
Migrating larva	Passage through lung	Eosinophilic pneumonitis
Luminal adult	Intestinal worm burden	Abdominal symptoms, obstruction
Migrating adult	Entry into ducts / appendix	Cholangitis, pancreatitis, appendiceal obstruction

Most infected individuals have no symptoms

Ascariasis is often asymptomatic.

Clinical severity varies with stage and worm burden.

Absence of abdominal complaints does not exclude infection.

Complicated cases arise disproportionately with heavy burdens or aberrant migration.

Worm burden predicts the intestinal phenotype

Light infection

- Often asymptomatic
- Nonspecific symptoms may be absent
- Detected only when eggs or worms are observed

Heavy infection

- Greater likelihood of pain, nausea and vomiting
- Malnutrition may occur
- Mechanical intestinal obstruction is an important complication

Eosinophilic inflammation is a key tissue response

Parasite exposure

Tissue antigens and larval passage



Host response

Eosinophil-rich inflammatory infiltrates

Eosinophilic response is not species-specific

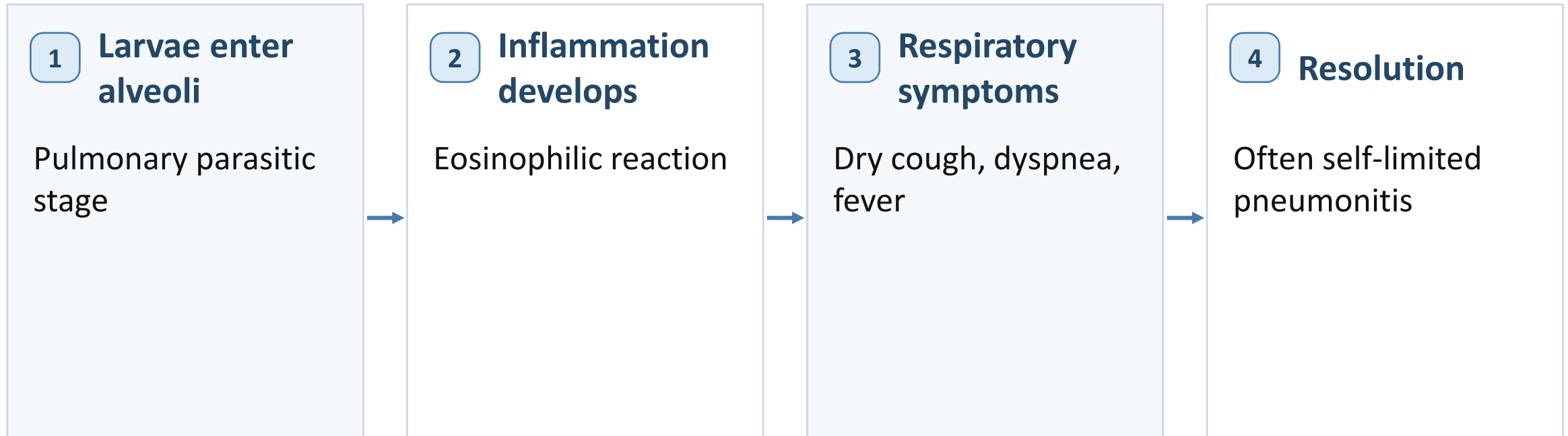
**Eosinophil-rich tissue inflammation supports helminthic injury,
not *Ascaris* identification.**

Robbins Figure 17.34B demonstrates eosinophilic tissue reaction.

Strongyloides may produce a similar reaction.

Identify the parasite through morphology or appropriate specimen findings.

Lung migration can cause Loeffler syndrome



Loeffler syndrome: Clinical pattern

Transient eosinophilic pneumonitis during pulmonary larval migration.

Symptoms include dry cough, dyspnea and fever.

It may precede evidence of mature intestinal worms.

The initial pulmonary illness as self-limited.

Pulmonary disease and stool tests may be discordant

Clinical finding

- Cough and dyspnea during early infection
- Eosinophilic pulmonary inflammation
- History of relevant exposure

Laboratory limitation

- Intestinal sexual maturity has not occurred
- Eggs may not yet be passed in stool
- Early negative stool examination does not exclude larval ascariasis

Uncommon larval complication: Hepatic abscess

Migrating larvae can also produce hepatic abscesses.

This is described in the gastrointestinal chapter.

It represents extraintestinal larval-associated tissue injury.

Avoid conflating it with biliary obstruction by mature worms.

Intestinal symptoms may be nonspecific

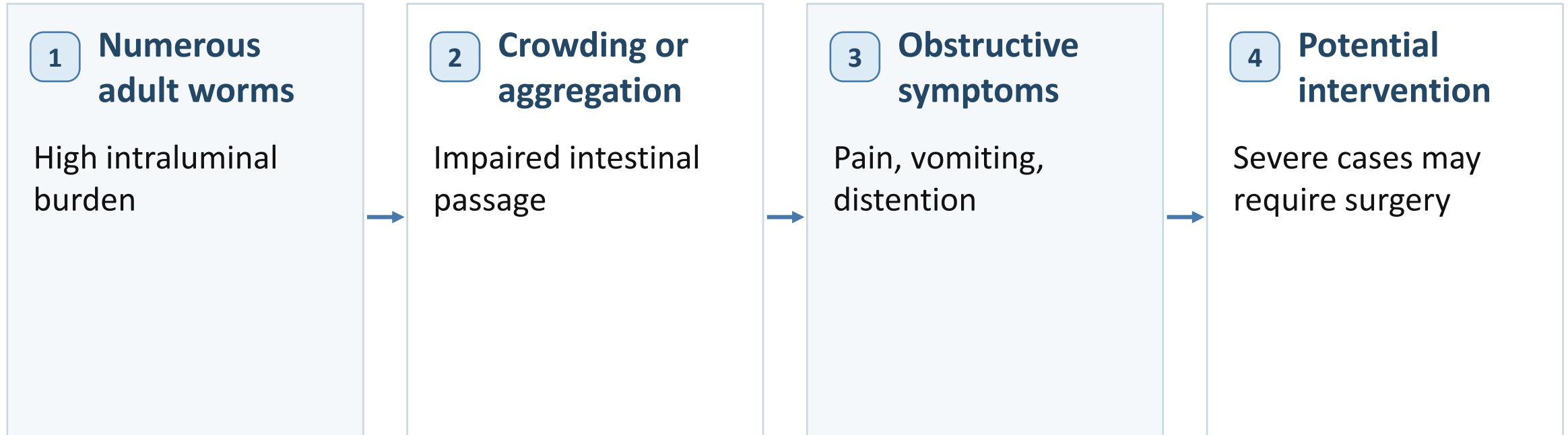
Common gastrointestinal complaints

- Abdominal discomfort or pain
- Loss of appetite, nausea, vomiting
- Diarrhea

Interpretive limitation

- Symptoms overlap with many other enteric disorders
- Clinical pattern alone does not establish the parasite
- Find eggs or adult organisms for confirmation when possible

Mechanical obstruction: The luminal mechanism



Intestinal obstruction is a major severe outcome

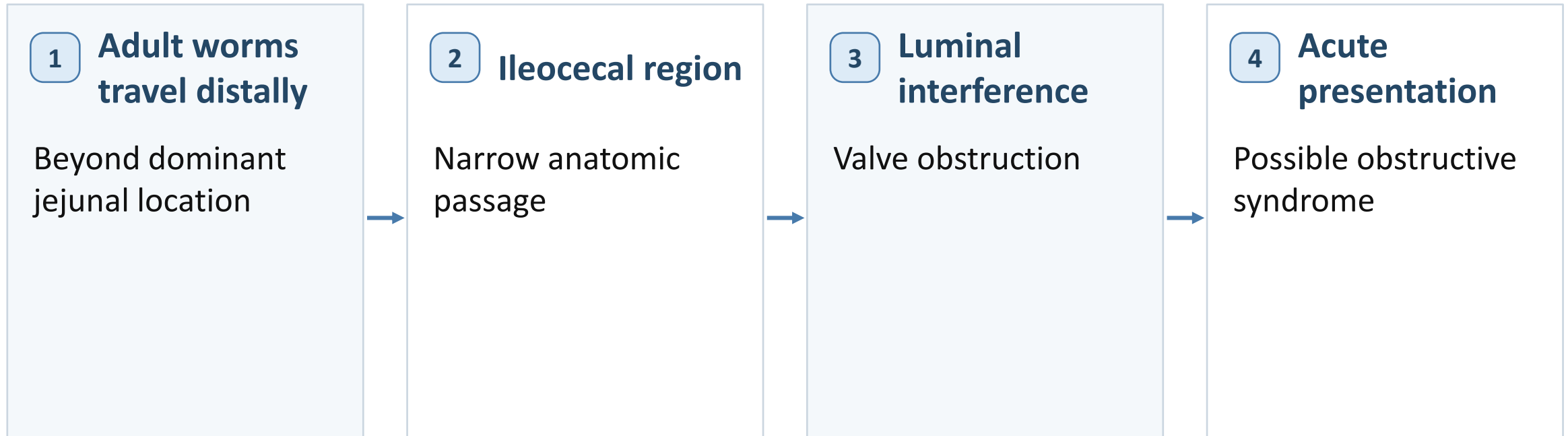
Heavy worm burden can obstruct the bowel.

The process is primarily mechanical rather than invasive mucosal destruction.

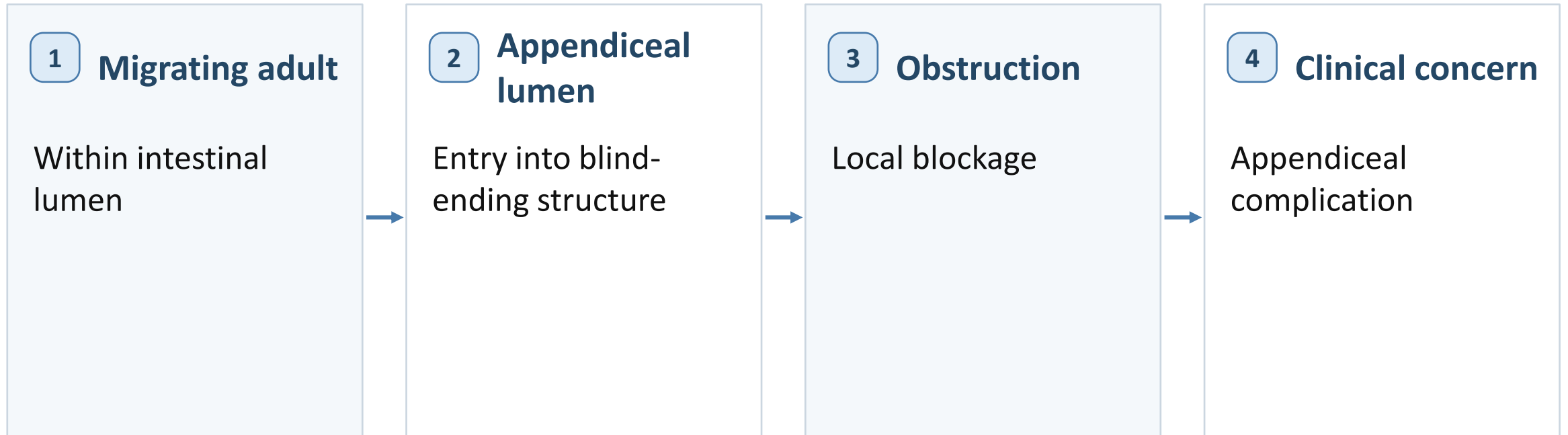
The small intestine is the principal adult habitat.

Severe obstruction may require surgery.

Ileocecal obstruction is a separate anatomic risk



Appendiceal entry can produce local obstruction



Adult migration explains hepatobiliary complications

Small intestinal adult

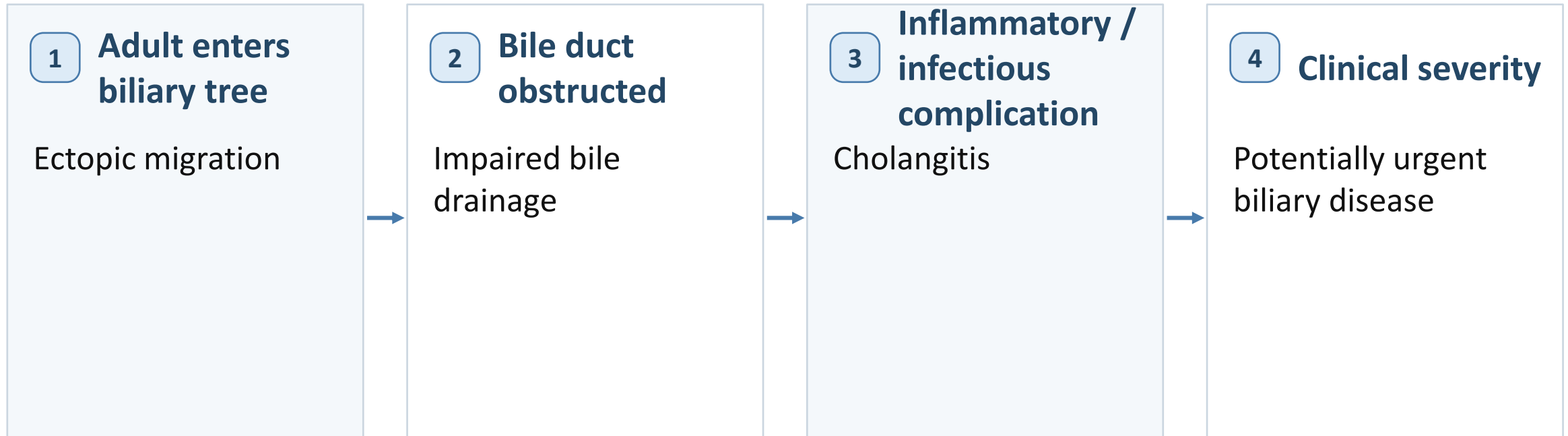
Aberrant migration into biliary tree



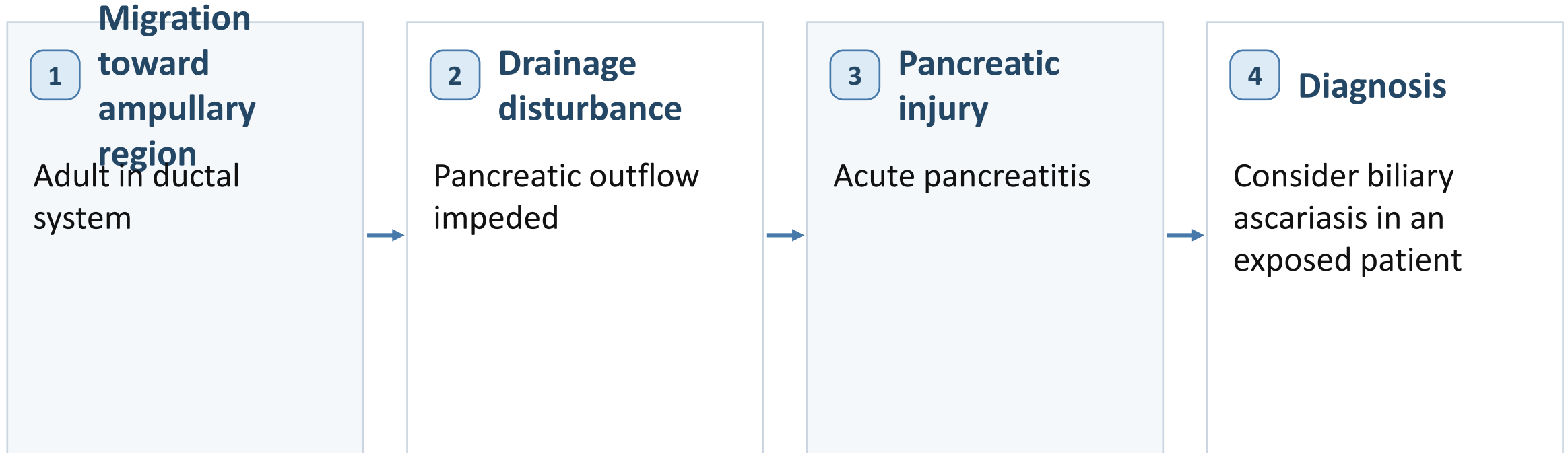
Ductal lumen

Obstruction $\pm$ secondary inflammation

Biliary ascariasis may cause cholangitis



Pancreatitis can follow worm migration



Complications by anatomic location

Compartment	Parasitic process	Recognized consequence
Lung	Larval migration	Loeffler pneumonitis
Small intestine	Large adult worm mass	Bowel obstruction
Ileocecal / appendix	Adult migration	Regional obstruction
Biliary / pancreatic ducts	Aberrant adult migration	Cholangitis, pancreatitis
Liver	Larval tissue complication	Hepatic abscess

Heavy infection can impair nutrition

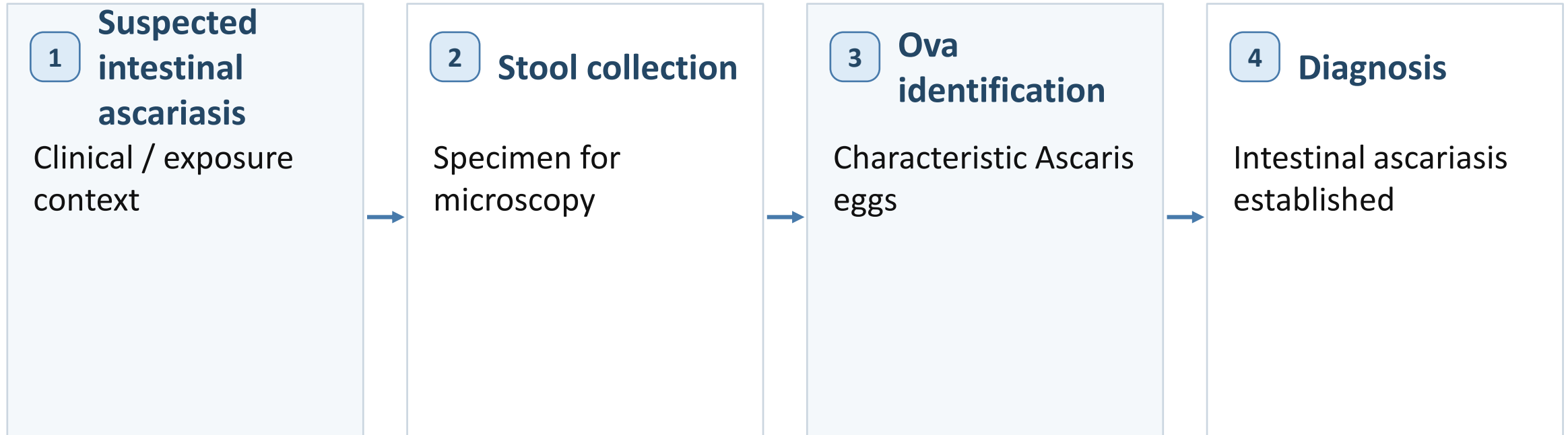
Malnutrition is a documented complication of heavy ascariasis.

Malnutrition among intestinal complications.

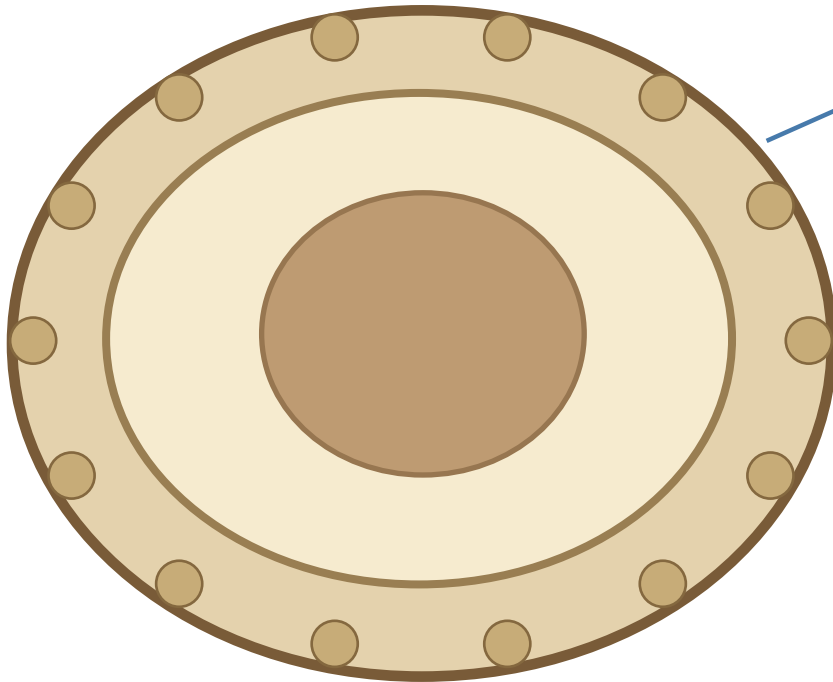
CDC notes that severe infection in children may contribute to stunted growth.

Do not infer a precise risk magnitude from these qualitative source statements.

The standard diagnostic specimen is stool



Fertilized eggs: Conceptual morphology



Schematic fertilized Ascaris egg

- Fertilized eggs are rounded and have a thick shell.
- An outer mammillated layer is often bile-stained brown.
- CDC DPDx describes a length range of 45–75 μm .

Mammillated coat (when present)

Fertile and infertile eggs differ

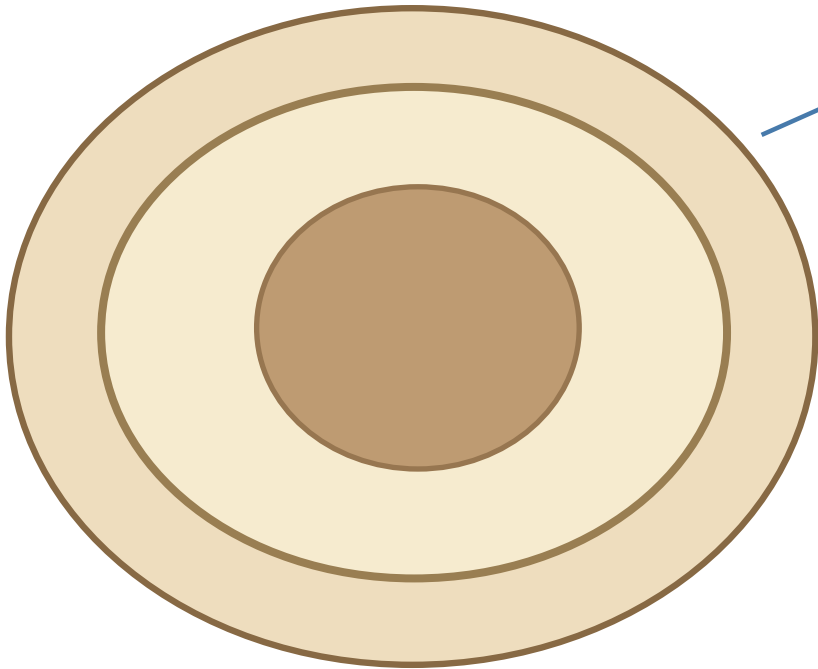
Fertilized egg

- Relatively rounded / oval
- Thicker shell
- Common mammillated outer surface
- Can develop to infectivity in soil

Unfertilized egg

- Often more elongated
- Larger and thinner-walled
- Variable mammillation; Granular contents
- Not infective on ingestion

Decorticated eggs: An important pitfall



Schematic egg without mammillated coat

- Loss of the outer mammillated coating changes egg appearance.
- Decorticated eggs can be fertilized or unfertilized.
- Missing outer bumps does not alone exclude Ascaris.

Outer coat absent

Stool microscopy workflow

Step	Rationale	CDC DPDx procedure
Collect and preserve	Maintain diagnostic morphology	Stool specimen with fixative
Concentrate	Increase recovery of ova	Formalin–ethyl acetate sedimentation
Microscopic review	Find characteristic eggs	Wet mount of concentrate
Alternative	Moderate/heavy burden	Direct wet mount if concentration unavailable

Why a single negative stool result can mislead

Early pulmonary phase

- Adult female has not matured
- Egg shedding may not have begun
- Clinically suspected infection may be stool-negative

Established intestinal phase

- Egg shedding is usually demonstrable
- Stool microscopy becomes more informative
- Adult worms may occasionally be passed

Adult worm identification is another diagnostic route

Intestinal luminal adult — schematic longitudinal outline



Large roundworm morphology may be evident after spontaneous passage.

Adult worms passed from the rectum or occasionally coughed up.

CDC DPDx describes three lips at the anterior end and sex-related tail differences.

Pulmonary-phase specimens are different

Larvae may be demonstrated in sputum during pulmonary migration.

CDC DPDx also discusses gastric aspirates in this phase.

Specimen choice should match the parasite stage.

A stool-based strategy alone is imperfect before egg production.

Routine tissue reaction is supportive, not definitive

Eosinophils indicate a host response; Morphology determines parasite identity.

An eosinophil-rich lesion in ascariasis.

Similar inflammatory patterns occur in other helminth infections.

A diagnosed parasite should not be named solely from eosinophil predominance.

Comparing intestinal nematode mechanisms

Organism	Acquisition	Distinctive mechanism
Ascaris	Egg ingestion	Lung transit; Adult luminal obstruction
Strongyloides	Skin penetration	Autoinfection possible
Hookworms	Skin penetration	Duodenal blood feeding, anemia
Trichuris	Soil-transmitted infection	Severe burden: Colitis / prolapse

Ascaris versus Strongyloides

Ascaris

- Infective egg is swallowed
- Mature worm occupies bowel lumen
- Pulmonary transit during larval phase
- High burden may obstruct intestine or biliary tract

Strongyloides

- Larvae penetrate intact skin
- Autoinfection occurs within host
- Immunosuppression may permit hyperinfection
- Larvae and adults provoke tissue response

Ascaris versus hookworm

Ascaris

- Ingestion of environmentally developed eggs
- Large intestinal luminal adults
- Mechanical obstruction is key severe bowel complication

Hookworms

- Larvae enter through skin
- Adults attach to duodenal mucosa
- Blood loss and iron deficiency anemia characterize chronic infection

Ascaris versus whipworm and pinworm

Trichuris trichiura

- Predominantly pediatric burden
- Heavy infection can cause bloody diarrhea
- Rectal prolapse is a severe complication

Enterobius vermicularis

- Perianal deposition of eggs
- Nocturnal pruritus ani
- Adhesive tape test rather than relying on routine stool eggs

Clinicopathologic localization exercise

Patient feature	Parasite phase to consider	Reason
Dry cough + eosinophilic pneumonitis	Larval lung phase	Migration through alveoli
Colicky abdominal pain + obstruction	Adult intestinal phase	Crowded luminal worms
Cholangitis + intestinal worm exposure	Migrating adult	Possible biliary duct entry
Early infection + negative stool ova	Prepatent phase	No egg output yet

Case 1 — cough before stool positivity

Clinical presentation

A patient from an endemic setting presents with dry cough, fever and dyspnea after a plausible soil/food exposure. An early stool examination is negative.

How can ascariasis still explain this presentation?

Reasoning prompts

- Identify the life-cycle stage.
- Explain the eosinophilic lung response.
- Explain the diagnostic timing problem.

Case 1 — clinicopathologic interpretation

- 1 Pulmonary larval migration can cause self-limited Loeffler pneumonitis.
- 2 Egg output requires intestinal adult maturation; Early stool microscopy may be negative.
- 3 Interpret the negative examination according to the temporal phase.

Case 2 — an obstructed intestine

Clinical presentation

A child with a heavy intestinal helminth burden develops worsening abdominal pain, vomiting and features of mechanical bowel obstruction.

What pathophysiologic mechanism best links *Ascaris* to this illness?

Reasoning prompts

- Localize the mature worms.
- Consider burden-dependent effects.
- Identify the potentially severe complication.

Case 2 — mechanistic interpretation

- 1 Adult worms occupy the bowel lumen, especially the jejunum.
- 2 Large parasite numbers can crowd the lumen and obstruct intestinal transit.
- 3 Severe obstruction may require surgical management.

Case 3 — pancreatobiliary symptoms

Clinical presentation

A patient with suspected intestinal ascariasis develops a clinical syndrome of acute pancreatitis or cholangitis.

Why does a predominantly intestinal nematode produce ductal disease?

Reasoning prompts

- Track where an adult worm might move.
- Consider the effect on ductal drainage.
- Distinguish adult migration from larval pneumonitis.

Case 3 — ductal migration

1

Mature worms may migrate from the intestine into the biliary tree.

2

Mechanical interference with biliary/pancreatic drainage can lead to cholangitis or pancreatitis.

3

This is a different complication pathway from pulmonary larval migration.

Transmission interruption follows life-cycle logic

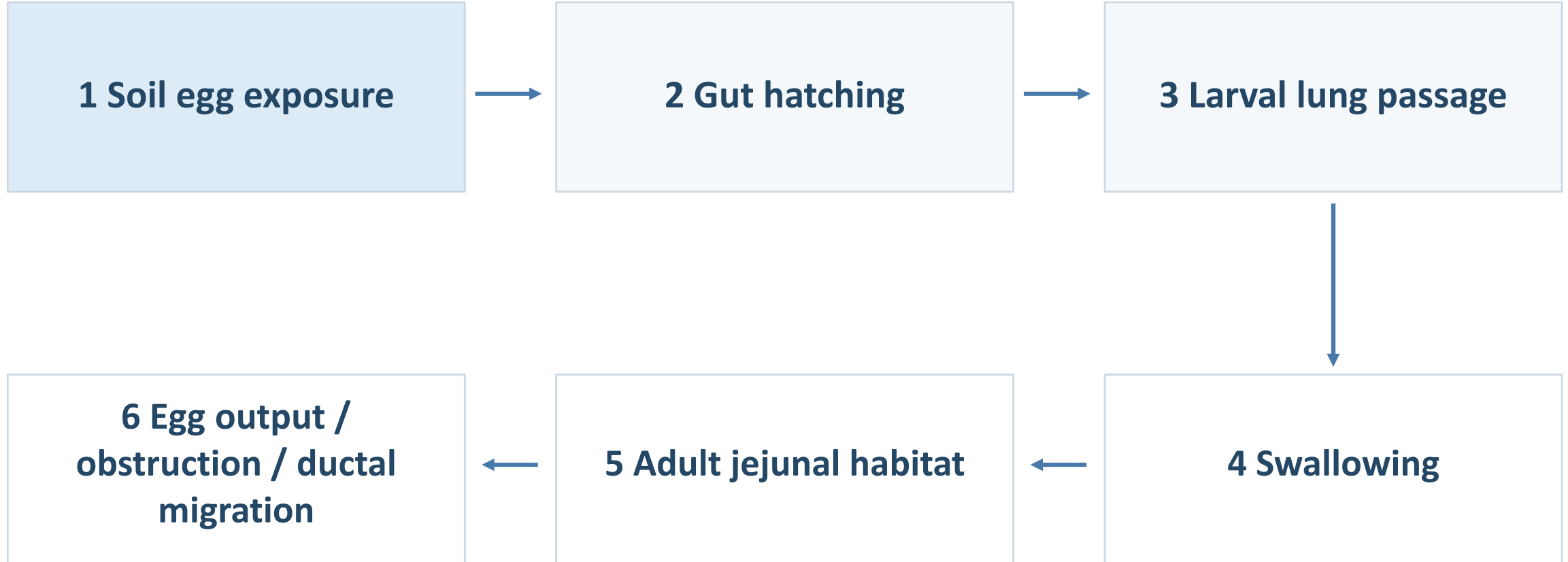
Environmental control

- Sanitation limits dispersal of fecal eggs.
- Preventing contamination reduces egg development opportunities.
- Produce hygiene reduces ingestion of environmental eggs.

Clinical identification

- Stool egg detection identifies intestinal infection.
- Recognizing early pulmonary disease prevents false reassurance.
- Detect complications by the anatomic presentation.

Integrative pathogenesis map



Schematic: Sequence is conceptual, not a time-scaled or anatomic diagram

Three mechanisms, three clinical outcomes

Biologic action	Tissue / organ consequence	Clinical result
Larvae migrate through lungs	Eosinophilic pulmonary inflammation	Loeffler pneumonitis
Adults accumulate in intestinal lumen	Mechanical interruption of transit	Intestinal obstruction
Adults enter bile ducts	Ductal obstruction / inflammation	Cholangitis or pancreatitis

High-yield synthesis

Think in stages: Swallowed egg → migrating larva → luminal adult.

Larval phase: Pulmonary inflammation, often before stool eggs appear.

Adult phase: Frequent silence, but heavy burdens obstruct the bowel.

Ectopic adult migration: Biliary and pancreatic complications.

Four errors worth avoiding

Incorrect assumption

- All soil-transmitted worms enter through the skin.
- Early stool-negative infection excludes *Ascaris*.
- Eosinophil-rich inflammation identifies species.
- Every adult worm infection is symptomatic.

Correct interpretation

- *Ascaris* infection begins by swallowing eggs.
- Prepatent lung-phase infection may be stool-negative.
- Inflammation is supportive but nonspecific.
- Many infections are asymptomatic.

References and provenance

Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th ed. 2026. Chapter 17, “The Gastrointestinal Tract,” p. 725.

Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th ed. 2026. Chapter 8, “Infectious Diseases,” p. 362.

CDC DPDx. Ascariasis. Laboratory morphology, parasite biology and diagnosis (page reviewed July 19, 2019). <https://www.cdc.gov/dpdx/ascariasis/>

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