
Campylobacter Enterocolitis

Bacterial enterocolitis: pathogenesis, morphology, and
clinicopathologic correlation

Dr Abu Suraih Sakhri

MBBS, MD (Pathology)

www.abusuraihsakhri.com

Learning outcomes

- Identify epidemiologic reservoirs and transmission pathways for *C. Jejuni*.
- Explain motility, adherence, toxin effects, and invasion as pathogenic properties.
- Interpret acute colitis morphology and its limited etiologic specificity.
- Link enteric infection to delayed immune-mediated complications.
- Differentiate *Campylobacter* from selected bacterial enterocolitides.

Where *Campylobacter* fits in enterocolitis

- Infectious enterocolitis may produce diarrhea, abdominal pain, urgency, and bleeding.
- *C. Jejuni* is emphasized as a major bacterial enteric pathogen in high-resource settings.
- Its presentation spans watery diarrhea and inflammatory, sometimes bloody, colitis.

The dominant histologic pattern is acute, often self-limited colitis.

Enterocolitis is a clinical syndrome

- Common manifestations include diarrhea and abdominal pain.
- Urgency, perianal discomfort, and incontinence may accompany colitis.
- Hemorrhage is possible, but stool phenotype alone does not identify the organism.

An etiologic diagnosis needs context beyond symptoms.

Diarrhea: Mechanism is more useful than appearance alone

Mechanism	Interpretive concept
Secretory	Net luminal fluid secretion predominates.
Osmotic	Unabsorbed solutes retain water in the lumen.
Malabsorptive	Reduced nutrient absorption contributes to diarrhea.
Exudative	Inflamed or damaged mucosa allows inflammatory fluid and blood loss.

Diarrhea by mechanism; This table is contextual, not a claim that all mechanisms are caused by Campylobacter.

C. Jejuni: The central disease profile

- A leading bacterial cause of food poisoning and travelers' diarrhea.
- Reservoirs are predominantly animal; Poultry is a key exposure.
- Virulence properties produce epithelial injury and an acute neutrophilic response.
- Some patients develop delayed extraintestinal complications.

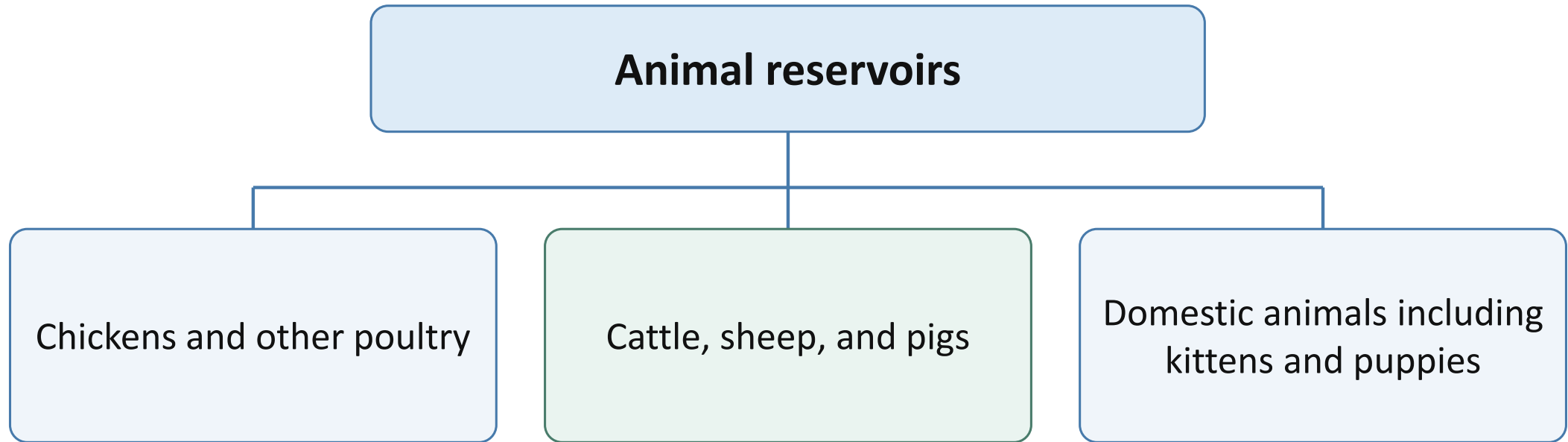
Acute intestinal disease and later immune sequelae require separate causal models.

Epidemiologic significance

- C. Jejuni as the most common bacterial enteric pathogen in high-resource countries.
- The textbook quotes a CDC estimate exceeding 1.5 million US cases annually.
- Both sporadic disease and food-associated outbreaks are important contexts.

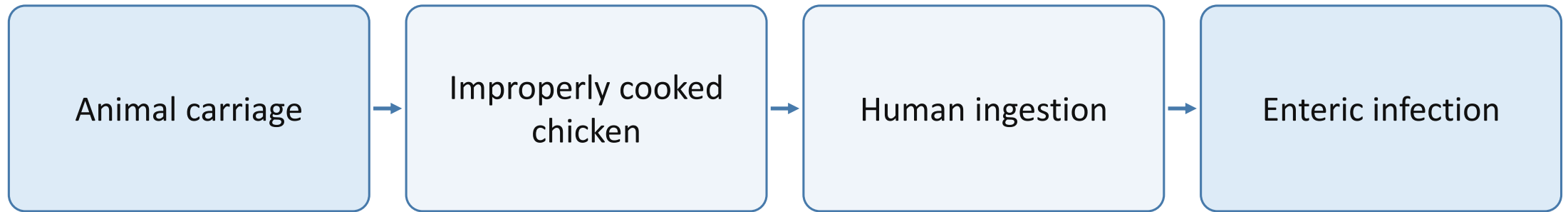
The US number is a textbook-quoted estimate, not an independently updated surveillance figure.

Animal reservoirs precede human exposure



The branches describe alternatives or related categories, not an invariant sequence.

Poultry-related transmission



Poultry exposure is especially prominent's discussion.

Food and water routes

Potential contaminated exposures

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graph TD; A[Potential contaminated exposures] --> B[Unpasteurized milk]; A --> C[Contaminated drinking water]; A --> D[Other foods associated with animal reservoirs];
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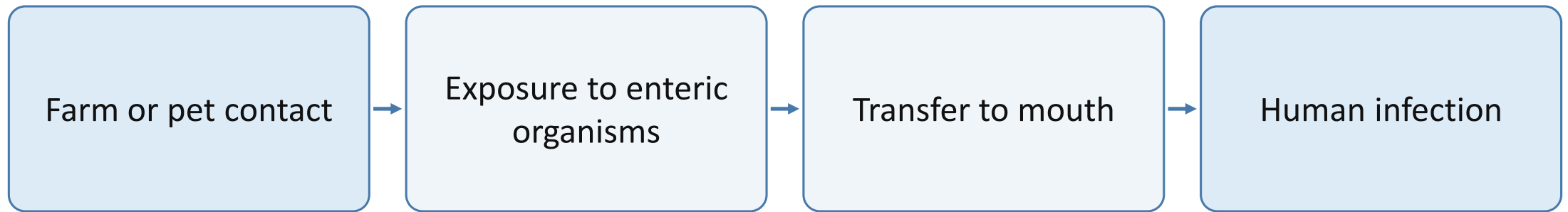
Unpasteurized milk

Contaminated drinking
water

Other foods associated
with animal reservoirs

The branches describe alternatives or related categories, not an invariant sequence.

Direct animal contact is another route



Farm animals and young companion animals are specified sources.

Transmission routes are not equally important

Route	Campylobacter interpretation
Foodborne	Major route: Especially inadequately cooked poultry.
Milk and water	Documented routes: Unpasteurized milk; Contaminated water.
Animal contact	Exposure to farm animals and some pets.
Person to person	Uncommon compared with other enteric infections.

Transmission ranking follows the source discussion; Not a quantified attributable-fraction analysis.

Exposure reconstruction: An initial history

A young adult develops diarrhea after a shared meal involving chicken; A companion reports no symptoms.

Question

Which exposure should be prioritized, and does the unaffected companion exclude infection?

Improperly cooked poultry is a major possible route. Not every exposed person needs to develop symptomatic illness.

A relatively small inoculum can initiate disease

- The source reports illness after ingestion of as few as 500 viable organisms.
- A relatively low infective dose can make transmission efficient.
- An infective dose is not the same as a certainty of infection after every exposure.

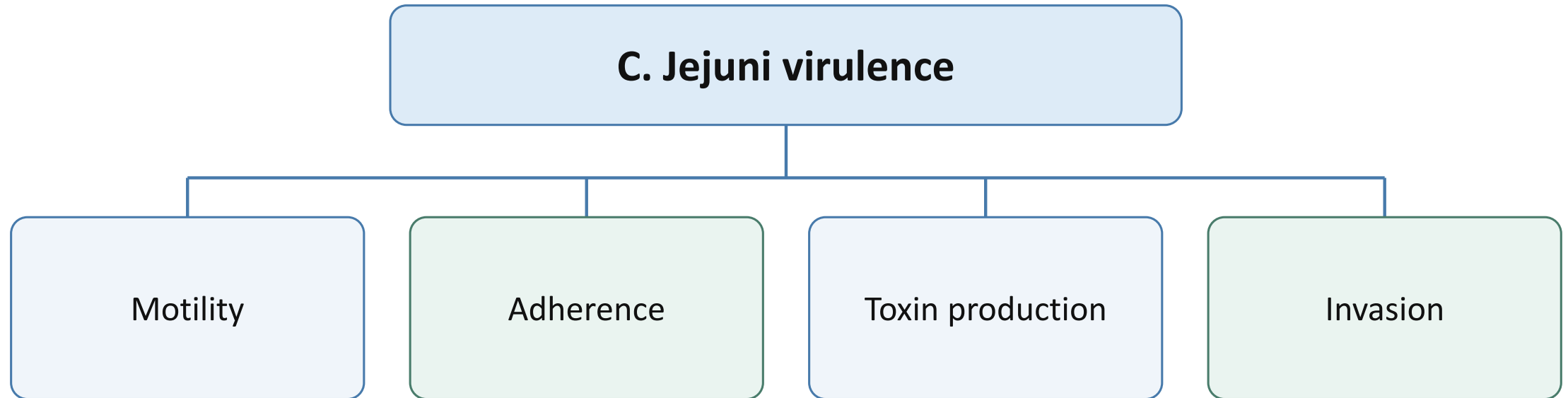
As few as 500 is a source-described possibility, not a universal threshold.

Microbial morphology matters

- Campylobacter organisms are curved or comma-shaped Gram-negative bacteria.
- They are flagellated and motile.
- Flagellar motility facilitates subsequent adherence and colonization.

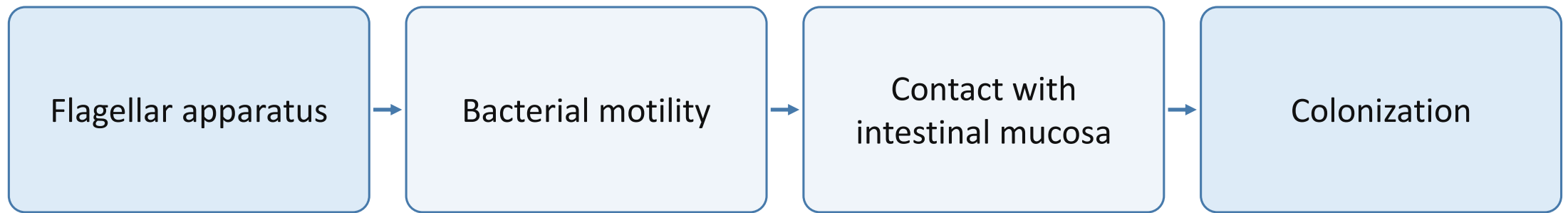
Morphology describes microbial form; It does not identify the organism in a tissue biopsy.

Four properties explain virulence



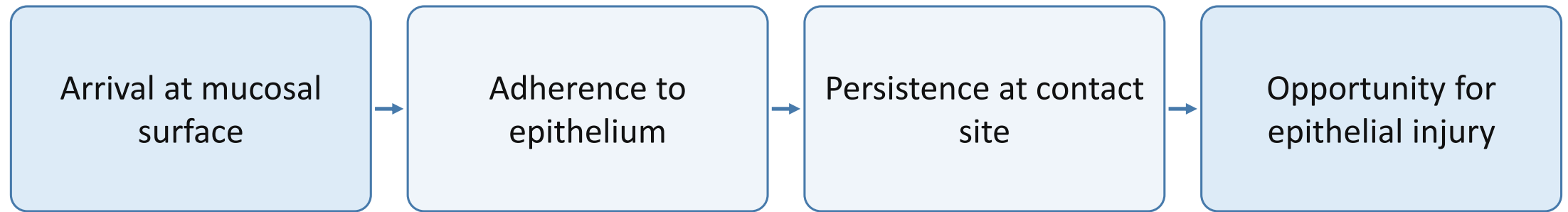
These are related but distinct mechanistic or clinical categories.

Motility helps establish mucosal colonization



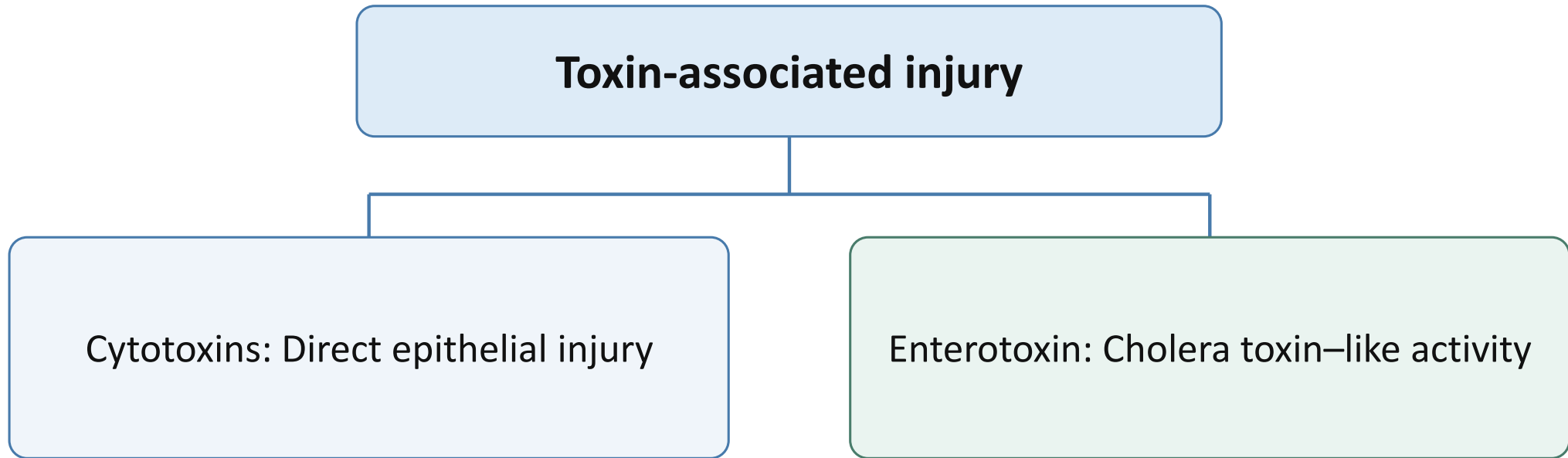
Motility supports the initial interaction with the intestinal surface.

Adherence makes colonization possible



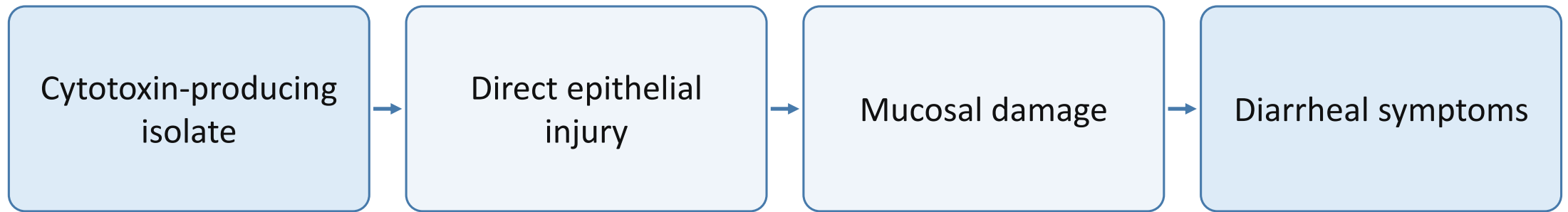
Adherence is a distinct property from invasion or toxin production.

Toxin production is heterogeneous



The branches describe alternatives or related categories, not an invariant sequence.

Cytotoxins can damage epithelial cells



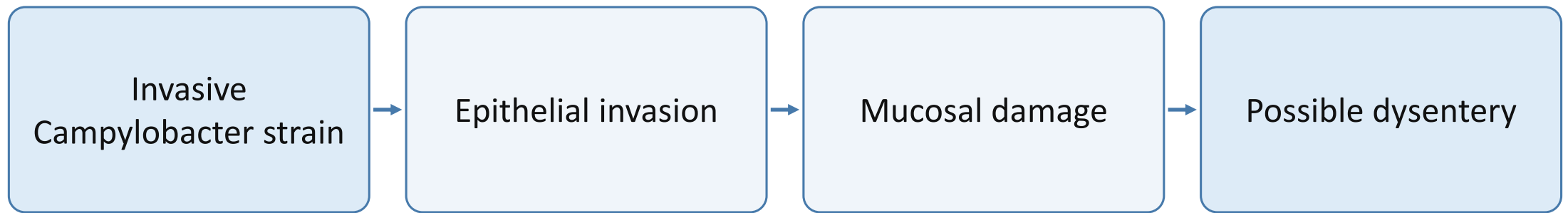
The text supports direct epithelial injury; No specific toxin gene is required for this slide.

Cholera-like does not mean identical to cholera

Property	Interpretive caution
Campylobacter	Some isolates release a cholera toxin–like enterotoxin.
Cholera	The source separately details cAMP–CFTR signaling for V. Cholerae.
Do not infer	The identical molecular signaling pathway for every C. Jejuni strain.

Prevents incorrect transfer of a separately described cholera mechanism.

Invasion links bacteria with inflammatory disease



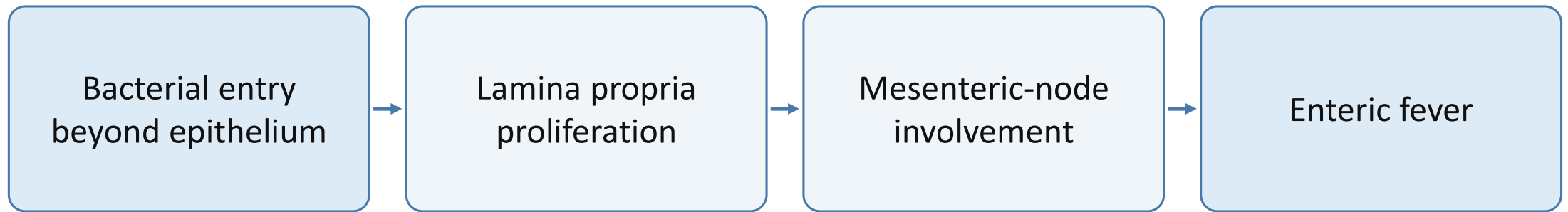
The source relates bloody diarrhea particularly to invasion.

Why watery and bloody phenotypes coexist

- Watery diarrhea is described as the primary symptom.
- Bloody diarrhea can occur, especially with invasive-associated damage.
- Only a minority of strains are described as invasive in the source.
- Clinical phenotype should not be equated with a directly demonstrated strain genotype.

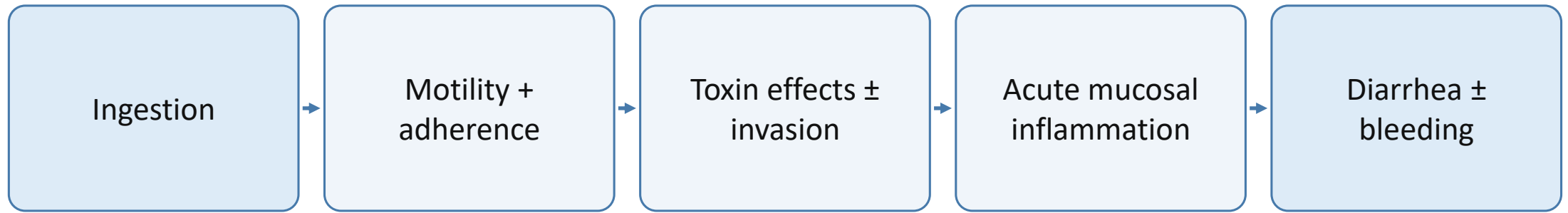
Clinical expression and specific virulence traits should not be conflated.

Lamina propria involvement can cause enteric fever



A deeper tissue response is distinct from uncomplicated mucosal diarrhea.

Integrated mechanism of enterocolitis



Schematic synthesis; The steps represent possible contributors, not a mandatory sequence in every case.

Inflammatory response explains tissue injury

- The pathologic hallmark is neutrophilic infiltration of mucosa and epithelium.
- Neutrophils in crypt epithelium constitute cryptitis.
- Neutrophils collected inside crypt lumens form crypt abscesses.

The inflammatory pattern is informative but not organism-specific.

The dominant intestinal site is the colon

Feature	Textbook characterization
Major affected site	Colon, in the bacterial-enterocolitis comparison table.
Symptom profile	Watery or bloody diarrhea.
Mucosal response	Acute neutrophilic colitis.
Important warning	Biopsy pattern alone cannot establish C. Jejuni.

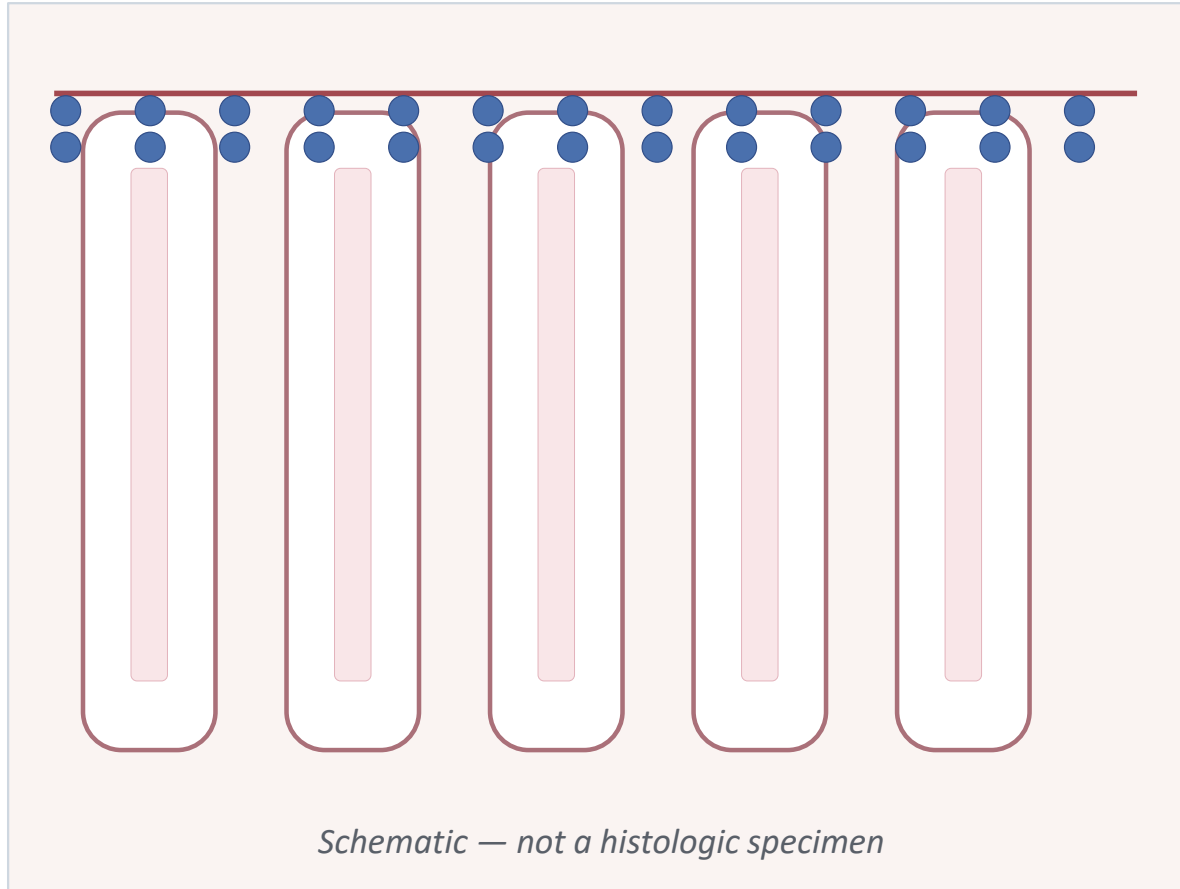
This is a general profile rather than a map of an individual patient.

Acute self-limited colitis is the morphologic pattern

- A conspicuous acute neutrophilic response.
- Superficial mucosal and intraepithelial neutrophils may be prominent.
- Cryptitis and crypt abscesses may be present.
- Overall crypt architecture is typically preserved.

The source figure associates *Campylobacter* with an acute self-limited colitis pattern.

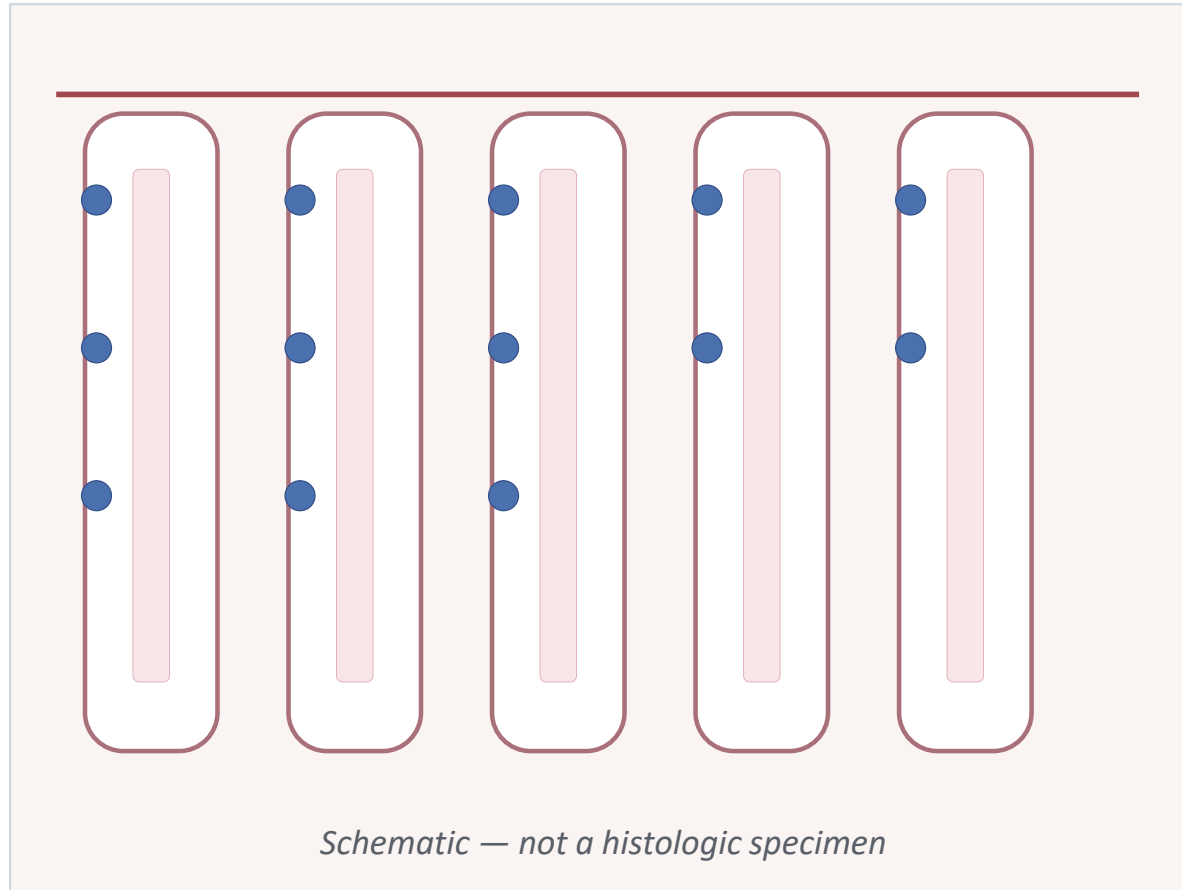
Superficial mucosal inflammation



What to recognize

- Neutrophils accumulate especially in superficial mucosa.
- Look for surface epithelial inflammatory injury.
- Location and inflammatory cell type matter more than an isolated generic label.

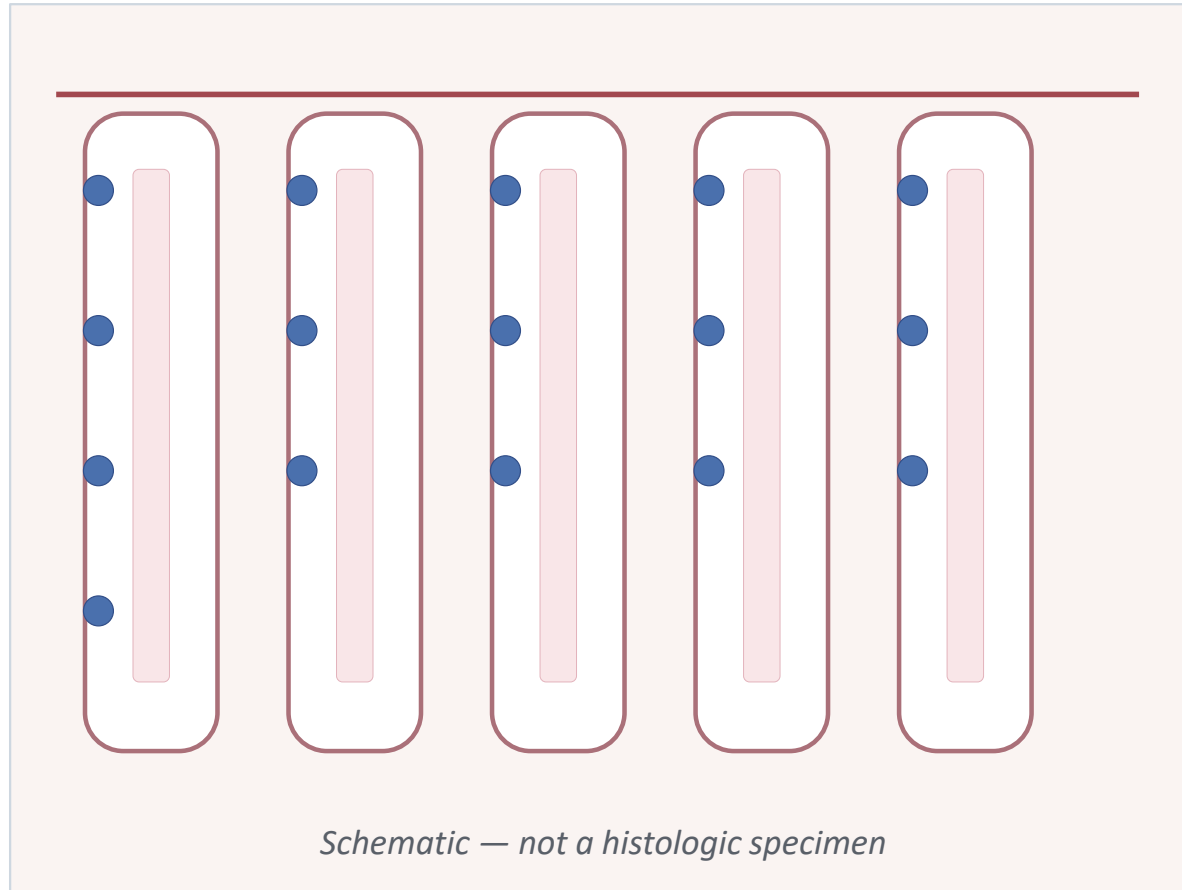
Intraepithelial neutrophils



What to recognize

- Neutrophils may appear inside the epithelial layer.
- This finding denotes acute epithelial inflammation.
- It does not identify the bacterial species by itself.

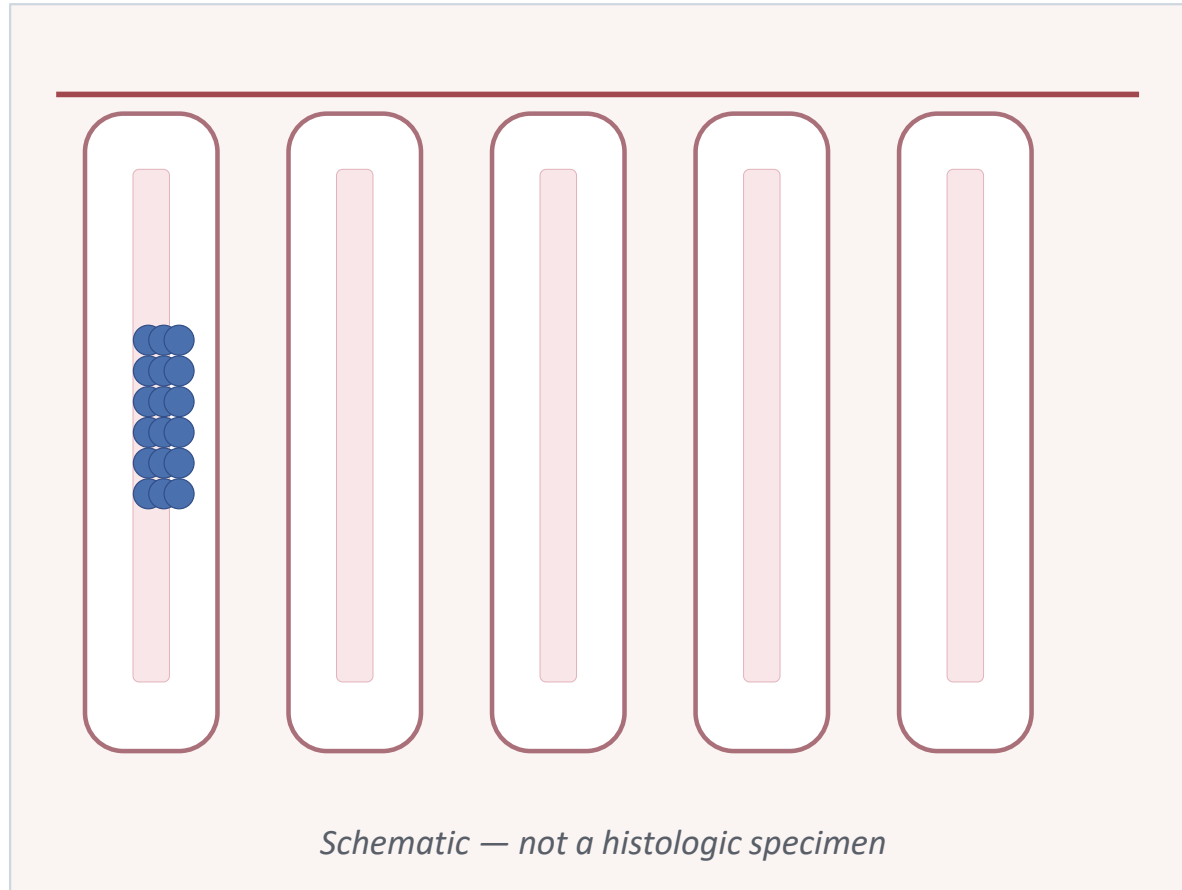
Cryptitis: Neutrophils invade crypt epithelium



What to recognize

- Cryptitis refers to neutrophils within crypt epithelium.
- It reflects active injury to glandular epithelial structures.
- The label specifies location, not the causative organism.

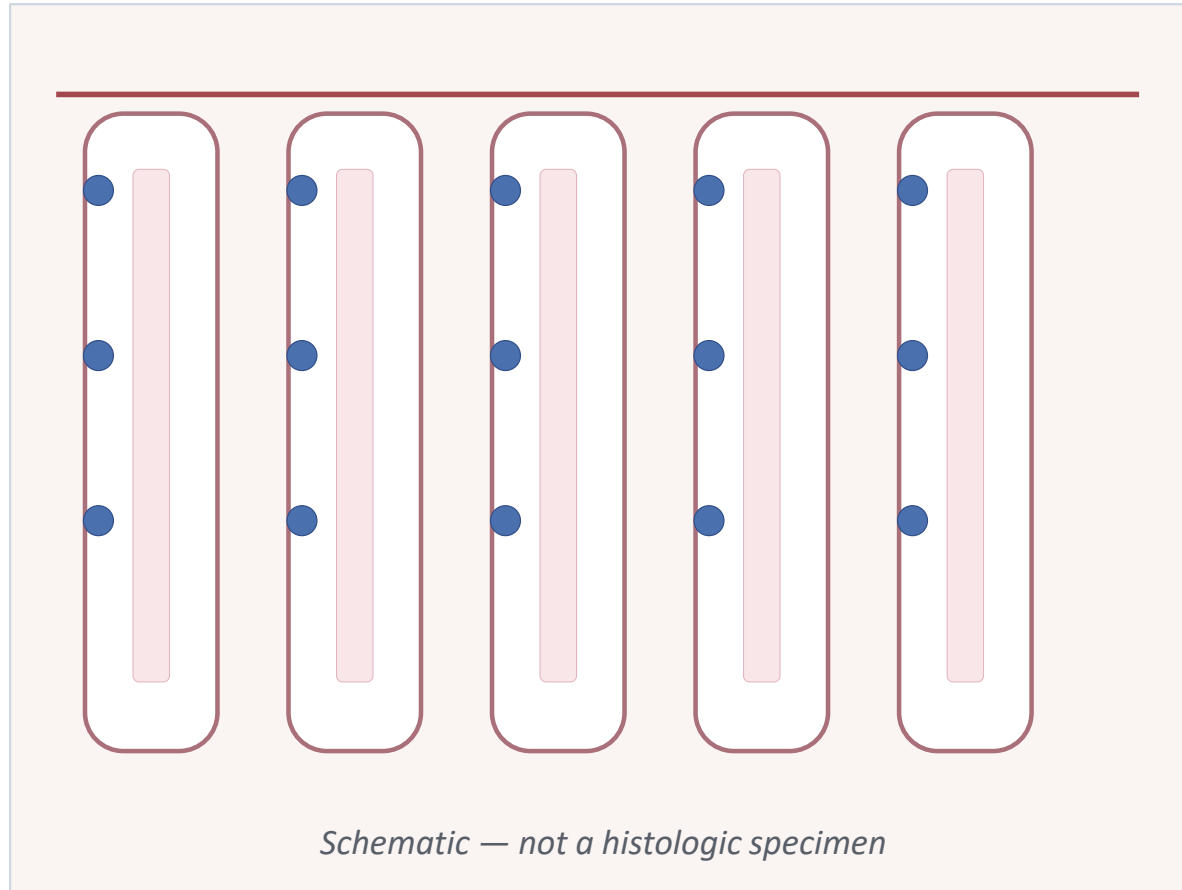
Crypt abscess: Neutrophils occupy a crypt lumen



What to recognize

- A crypt abscess is a collection of neutrophils within a crypt lumen.
- It may accompany cryptitis in acute infectious colitis.
- It is not equivalent to a deep tissue abscess.

Preserved crypt architecture is a central clue



What to recognize

- Crypt spacing and orientation may remain normal.
- Active neutrophilic inflammation can coexist with retained architecture.
- This supports an acute pattern but is not etiologically definitive.

Severe damage complicates architectural assessment

- Extensive mucosal injury may obscure the usual crypt spacing or alignment.
- Apparent distortion in a damaged focus should be interpreted cautiously.
- Assessment benefits from preserved regions where architecture remains interpretable.

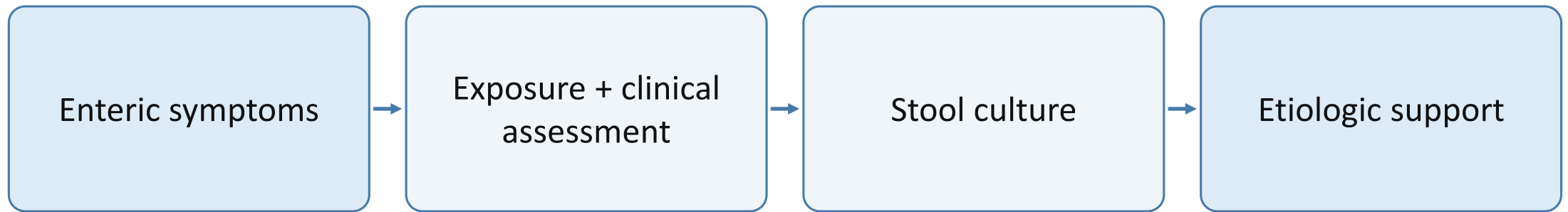
The source explicitly warns that preserved architecture may be difficult to assess in severe injury.

Inflammation is a pattern—not microbiologic proof

Finding	What it establishes	What it does not establish
Neutrophils in surface	Acute mucosal inflammation	The identity of an organism
Cryptitis or abscess	Active crypt epithelial injury	Campylobacter specifically
Preserved crypts	An acute-pattern architectural clue	Absolute exclusion of other causes

Pathology must be integrated with stool microbiology and exposure history.

Diagnostic reasoning: Why stool culture matters



Stool culture is the primary diagnostic method because biopsy findings are nonspecific.

Pathology sign-out exercise

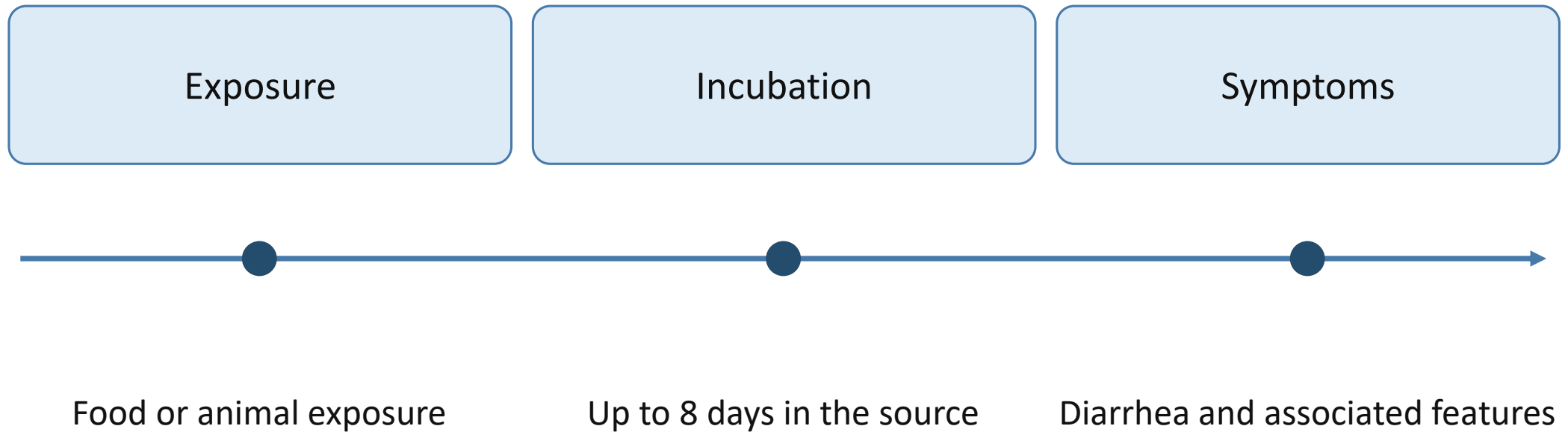
Colon biopsy: Superficial neutrophils, focal cryptitis and crypt abscesses, with largely maintained crypt spacing.

Question

What pattern can be diagnosed, and what conclusion must be avoided?

Describe active/acute colitis with preserved architecture. Do not identify C. Jejuni solely from the biopsy.

Incubation precedes gastrointestinal symptoms



Prodromal symptoms can precede diarrhea

- The text describes a possible influenza-like prodrome.
- Such a prodrome can occur before onset of diarrhea.
- A systemic prodrome does not distinguish *Campylobacter* from every competing pathogen.

Clinical context, not a pathognomonic symptom.

Watery diarrhea is the principal symptom

- The source designates watery diarrhea as the primary symptom.
- Abdominal symptoms and acute colitis can coexist.
- Bleeding may develop in a subset of patients.

The stool phenotype can evolve and is not a stand-alone pathogen test.

Dysentery varies by age group

Patient group	Source description
Children	Bloody diarrhea develops in more than half of affected children.
Adults	Bloody diarrhea is described as much less frequent than in children.
Interpretation	Do not use absence of blood to exclude Campylobacter.

Bloody diarrhea is age-associated but does not establish the bacterial cause.

Young children are a major affected group

- Campylobacter enteritis occurs commonly in young children.
- Bloody diarrhea is reported more often in children than adults.
- The inflammatory phenotype should prompt a broad enteric differential.

Age alters presentation but cannot determine etiology alone.

A second clinical peak occurs in young adulthood

- Campylobacter has a second age peak among young adults.
- This distinguishes its age distribution from a simple early-childhood infection profile.
- Exposure history is particularly relevant in an adult with acute food-associated diarrhea.

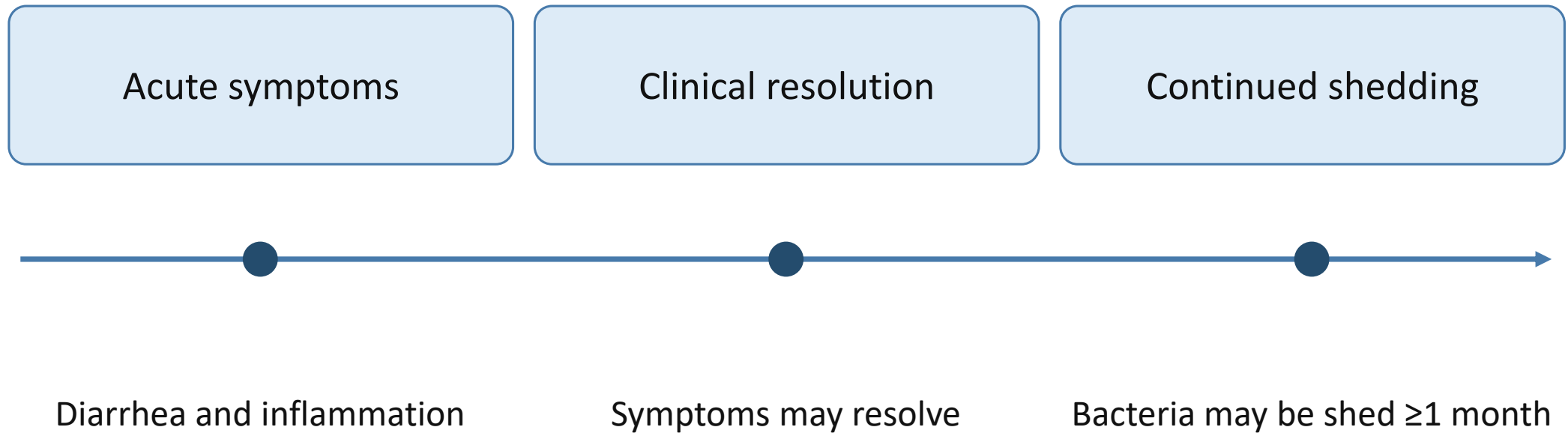
A bimodal clinical pattern does not imply a unique route in each age group.

Older adults may have deceptively mild symptoms

- Older patients report typical symptoms such as pain less often than children.
- Despite fewer reported symptoms, severe illness and hospitalization are more likely.
- Symptom intensity therefore need not track disease severity across age groups.

A caution about age-related clinical interpretation.

Shedding can outlast symptomatic illness

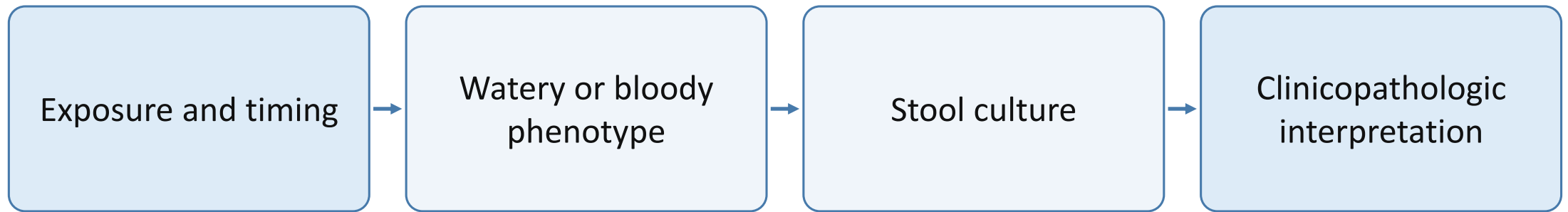


Antibiotic treatment is usually unnecessary

- Antibiotic therapy is generally not required.
- This reflects the usual self-limited clinical course.
- The excerpt does not provide drug selection, dosing, or specific exception criteria.

Do not infer a detailed treatment algorithm from this pathology excerpt.

Etiologic work-up integrates complementary evidence



Biopsy can characterize inflammation but ordinarily cannot assign species.

Reactive arthritis is a delayed sequela

- Reactive arthritis can occur after *Campylobacter* infection.
- The source emphasizes an association with HLA-B27 genotype.
- This is a postinfectious systemic manifestation, not evidence of bacteria inside the joint.

Link temporal history and immune predisposition.

HLA linkage differs across complications

Complication	HLA relationship described
Reactive arthritis	Primarily in patients with HLA-B27.
Erythema nodosum	Not described as HLA-linked.
Guillain–Barré syndrome	Not described as HLA-linked.

This distinction is explicit in the Robbins passage.

Erythema nodosum is another associated event

- The infection can precede an inflammatory extraintestinal skin manifestation.
- The source names erythema nodosum among possible complications.
- Unlike reactive arthritis, it is not characterized as HLA-linked here.

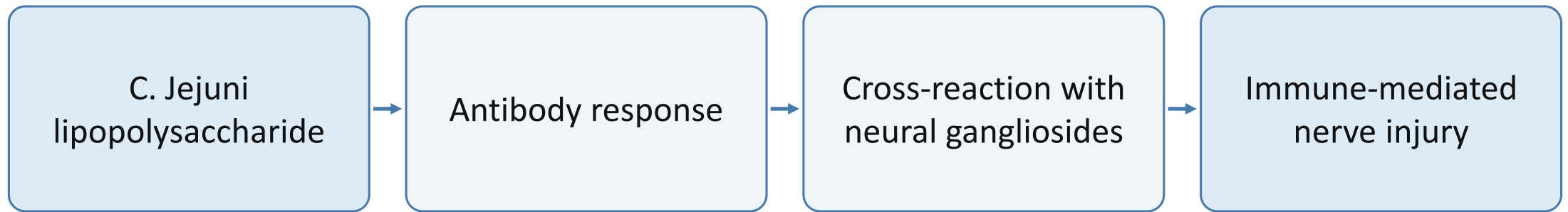
Association is supported; Frequency and dermatopathology are not specified in this source.

Guillain–Barré syndrome follows immune nerve injury

- GBS is characterized as flaccid paralysis.
- Its pathogenesis involves immune-mediated inflammation of peripheral nerves.
- A preceding *Campylobacter* infection may be relevant to the clinical history.

Temporal association requires careful denominator interpretation.

Molecular mimicry: Proposed route to GBS



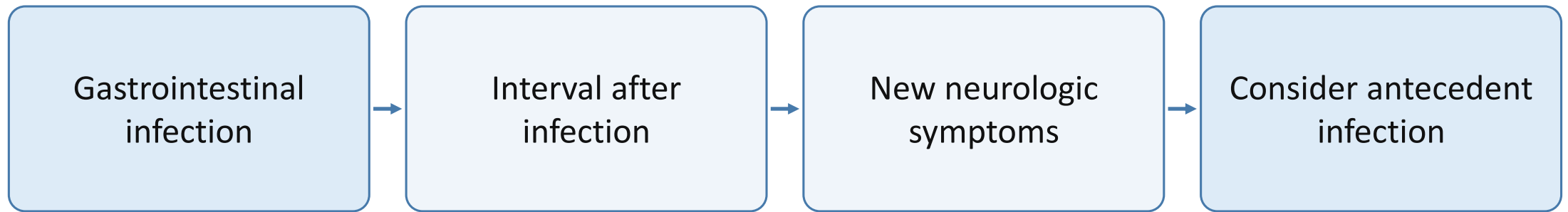
The textbook implicates antibody cross-reactivity, without claiming every GBS case shares this mechanism.

Risk among infected patients is not the reverse probability

Question	Textbook statement
Given Campylobacter infection, how often GBS?	Estimated risk <0.1%.
Given GBS, how often antecedent Campylobacter?	Up to 40% documented infection in preceding 2 weeks.
Additional positive microbiology or antibodies in GBS	Up to 50% positive stool cultures or circulating antibodies.

Different denominator groups: The figures must not be interchanged.

Interpreting the postinfectious interval



Documented *Campylobacter* infection in the 2 weeks preceding some GBS presentations.

Postinfectious irritable bowel syndrome

- Campylobacter infection is associated with subsequent irritable bowel syndrome.
- Persistent bowel symptoms do not necessarily indicate continuing bacterial invasion.
- The source states an association but does not specify the individual risk or mechanism.

Avoid inventing a rate or a detailed pathogenesis beyond the provided text.

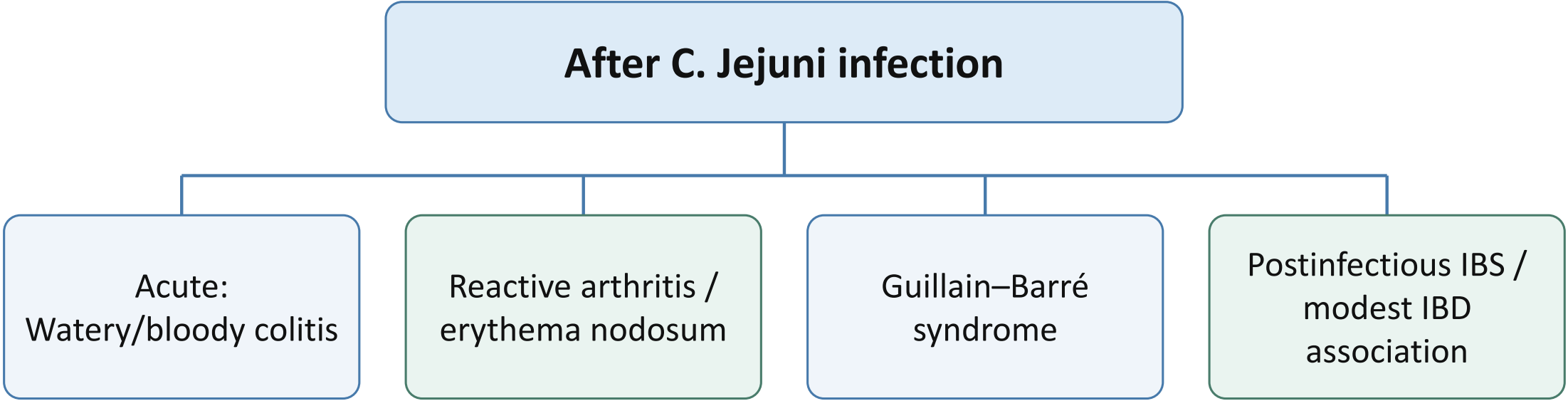
Inflammatory bowel disease: A modest association

- A modestly increased subsequent risk of inflammatory bowel disease.
- A population association does not mean that *Campylobacter* alone causes IBD.
- A later chronic inflammatory syndrome requires its own independent evaluation.

Causation, absolute risk, and subtype-specific effects are not established by this excerpt.

Intestinal and delayed manifestations diverge

After C. Jejuni infection



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graph TD; A[After C. Jejuni infection] --> B[Acute: Watery/bloody colitis]; A --> C[Reactive arthritis / erythema nodosum]; A --> D[Guillain–Barré syndrome]; A --> E[Postinfectious IBS / modest IBD association];
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The diagram is a flowchart starting with a central box 'After C. Jejuni infection'. A horizontal line below this box branches into four vertical lines, each leading to a separate box. The boxes are: 'Acute: Watery/bloody colitis' (light blue), 'Reactive arthritis / erythema nodosum' (light green), 'Guillain–Barré syndrome' (light blue), and 'Postinfectious IBS / modest IBD association' (light green).

Acute:
Watery/bloody colitis

Reactive arthritis /
erythema nodosum

Guillain–Barré
syndrome

Postinfectious IBS /
modest IBD
association

These are related but distinct mechanistic or clinical categories.

Campylobacter versus cholera

Dimension	Campylobacter	Cholera
Primary site	Colon	Small intestine
Symptoms	Watery or bloody diarrhea	Profuse watery diarrhea
Tissue response	Acute neutrophilic colitis	Minimal biopsy changes
Pathogenesis	Motility, toxins ± invasion	Noninvasive, toxin-driven secretion

Comparison drawn from adjacent Robbins sections.

Campylobacter versus Shigella

Dimension	Campylobacter	Shigella
Motility	Flagellated; Motile	Nonmotile
Reservoir	Animal reservoirs	Humans
Stool profile	Watery or bloody	Commonly bloody
Typical distribution	Colon	Left colon ± ileum
Microscopy	Acute colitis, preserved crypts	Can resemble acute colitis; Aphthous ulcers possible

Both require microbiologic identification; Biopsy overlap is substantial.

Campylobacter versus nontyphoid Salmonella

Dimension	Campylobacter	Nontyphoid Salmonella
Exposure	Poultry, milk, water, animals	Meat, poultry, eggs, milk
Sites	Predominantly colon	Colon and small intestine
Symptoms	Watery or bloody diarrhea	Watery or bloody diarrhea
Notable consequences	GBS, reactive arthritis	Sepsis or abscess; Reactive arthritis possible

Clinical symptoms overlap; Stool culture resolves etiologic ambiguity.

Campylobacter versus EHEC-associated colitis

Dimension	Campylobacter	Enterohemorrhagic E. Coli
Shared phenotype	Can cause bloody diarrhea	Bloody diarrhea
Histology	Neutrophilic acute colitis	Ischemia-like mucosal injury
Key complication	Postinfectious GBS association	Hemolytic uremic syndrome
Interpretation	Neither bleeding nor biopsy alone proves species	Target organism-specific microbiology

The EHEC pattern is based on the neighboring table/figure caption.

Four etiologic profiles at a glance

Organism	Exposure clue	Main site	Distinctive association
C. Jejuni	Poultry or animals	Colon	GBS; Reactive arthritis
V. Cholerae	Contaminated water	Small bowel	Severe fluid/electrolyte losses
Shigella	Human fecal–oral	Left colon ± ileum	Marked dysentery
Nontyphoid Salmonella	Meat, eggs, poultry	Colon ± small bowel	Sepsis or abscess

Compressed teaching comparison Chapter 17.

Case: Poultry exposure and inflammatory diarrhea

A young adult reports diarrhea after eating incompletely cooked chicken. Stool later becomes blood-streaked.

Question

Which organism is plausible, and which virulence property may help explain the blood?

C. jejuni is a plausible pathogen. Invasion-associated mucosal injury is linked to dysentery, but specific microbiologic diagnosis is needed.

Case: What can a mucosal biopsy prove?

A colonic biopsy shows intraepithelial neutrophils, crypt abscesses and mostly regular crypt spacing.

Question

Does the histology alone establish campylobacteriosis?

No. These findings indicate an acute colitis pattern. Stool culture is emphasized for specific microbiologic diagnosis.

Case: Weakness following diarrheal illness

A patient develops flaccid weakness after a recent diarrheal episode; The stool illness has subsided.

Question

What postinfectious syndrome warrants consideration, and what proposed mechanism connects it to *Campylobacter*?

Consider Guillain–Barré syndrome. Cross-reactive antibodies to bacterial LPS and neural gangliosides are implicated.

Mechanism, morphology, and clinical effect align

Exposure

Poultry, milk, water and animal contact

Mechanism

Motility + adherence; Toxin injury ± invasion

Morphology

Acute neutrophilic colitis;
Cryptitis/abscesses; Preserved crypts

Clinical

Watery or bloody diarrhea; Occasional delayed immune complications

References and source boundaries

Primary teaching source

Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th edition (2026), Chapter 17, Campylobacter Enterocolitis (pp. 718–719).

Contextual comparisons

Chapter 17: Infectious Enterocolitis and adjacent bacterial enterocolitis sections and Table 17.7 (pp. 716–721).

Note: no original textbook histology was reproduced. Crypt schematics are editable conceptual diagrams; they are not patient specimens.