
Diversion Colitis

Mechanism · morphology · differential diagnosis · clinical interpretation

A clinicopathologic lecture

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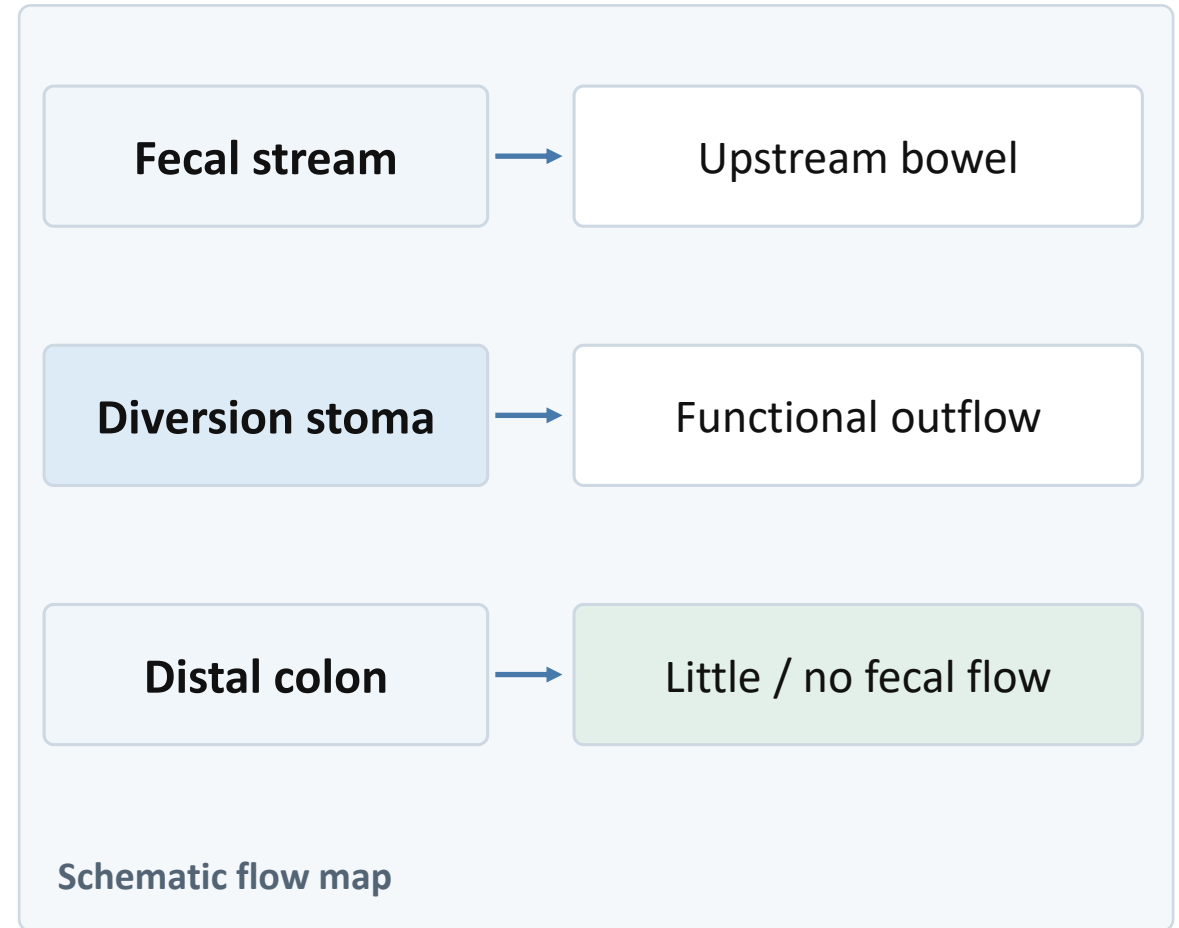
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Learning objectives

- Explain how fecal diversion creates an excluded colonic segment.
- Recognize the characteristic lymphoid follicles and the wider histologic spectrum.
- Differentiate diversion-related injury from primary inflammatory bowel disease.
- Relate proposed pathogenesis to restoration of bowel continuity.

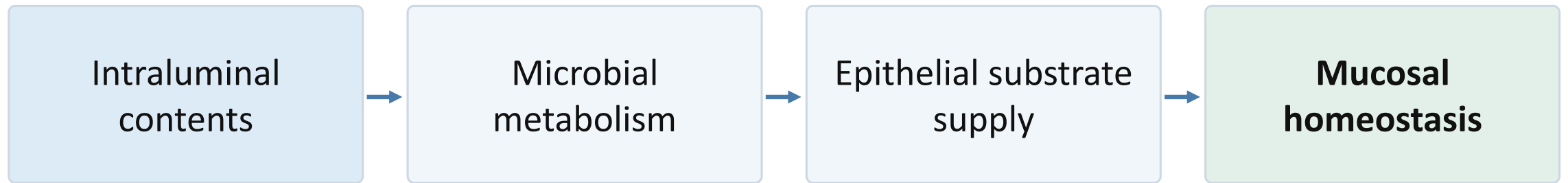
Definition: Inflammation of excluded colon

- Fecal flow is surgically rerouted away from a distal colonic segment.
- Inflammation can develop in the defunctioned mucosa.
- An anatomical exposure history is integral to the diagnosis.



The site of diversion is more informative than any isolated microscopic feature.

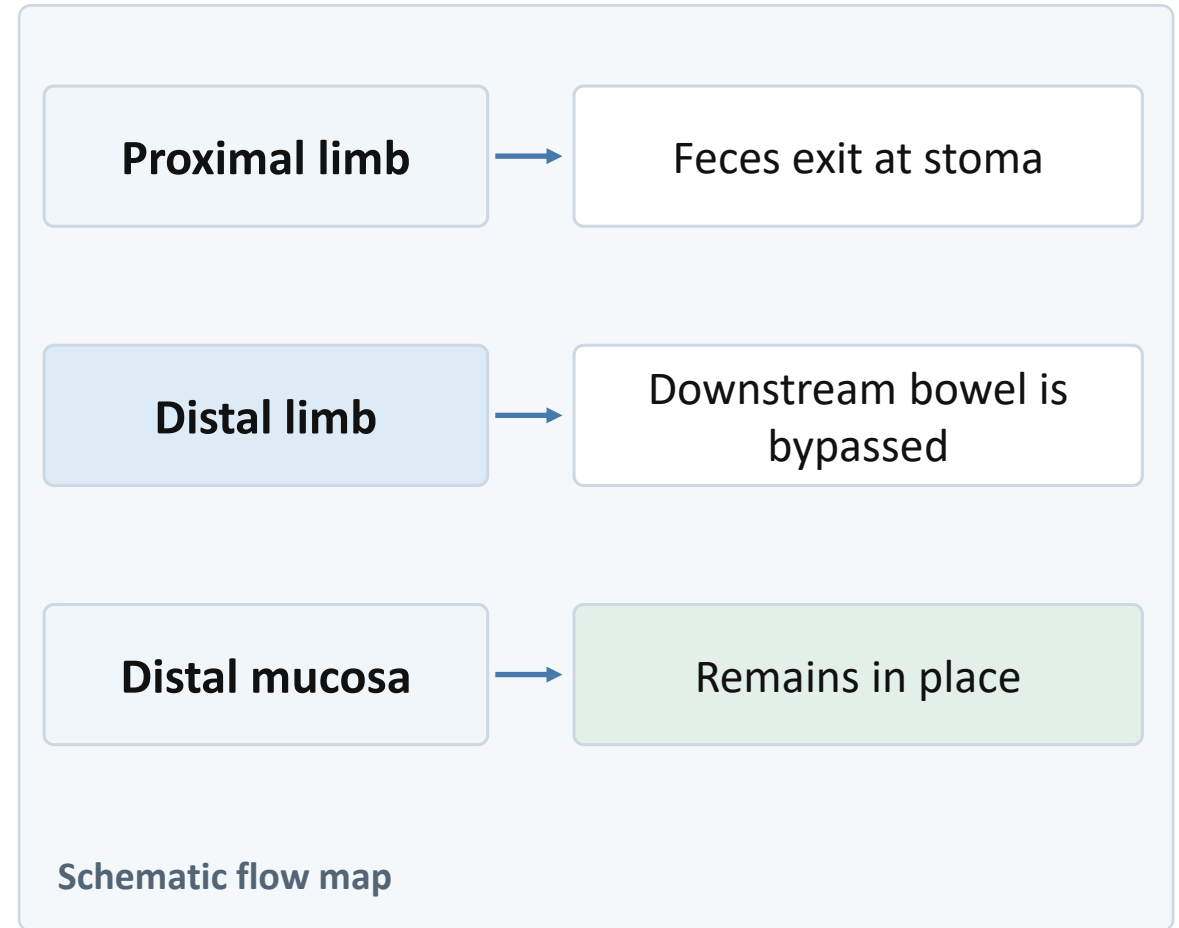
The normal fecal stream exposes colonic mucosa



- Bacterial short-chain fatty acids to epithelial signaling and energy provision.
- Diversion perturbs the normal luminal environment.

Diverting ostomy: Anatomy determines exposure

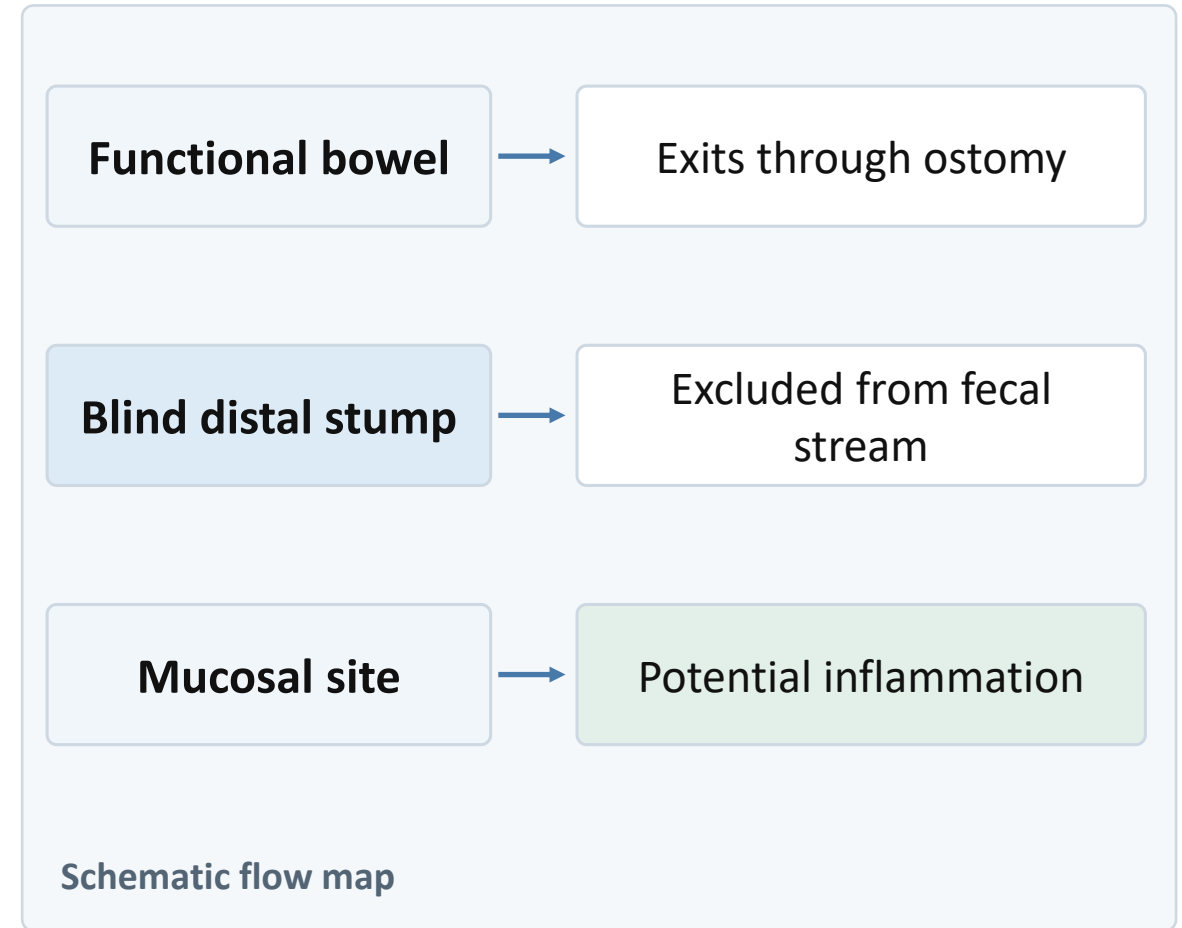
- A temporary or permanent ostomy redirects fecal transit.
- Upstream bowel remains in the functional fecal stream.
- The bypassed distal colon is the site at risk for diversion colitis.



Do not confuse a functioning proximal segment with the excluded segment.

End ostomy and blind distal bowel

- An ostomy may leave a blind distal colonic segment.
- Excluded mucosa remains viable but lacks normal fecal flow.
- Biopsy site and the surgical reconstruction must be documented.



Clinical situations requiring diversion

Ulcerative colitis

Surgical treatment can create a diverted segment; Baseline IBD complicates interpretation.

Hirschsprung disease

A congenital motility disorder for which staged bowel diversion may be used.

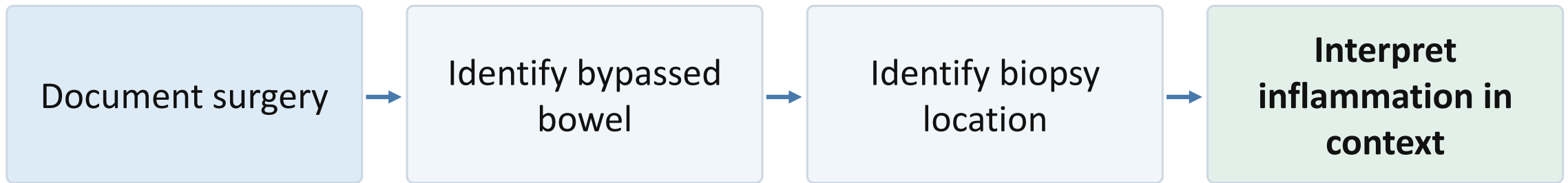
Colon cancer

Selected oncologic operations may include temporary or permanent fecal diversion.

Other disorders

The mechanism depends on diversion itself, not a single underlying diagnosis.

Anatomical localization is the first clue



- A pathology report without the ostomy history risks overdiagnosis of primary colitis.

Three clinically distinct bowel environments

Location	Exposure	Interpretive significance
Proximal functioning colon	Continuous fecal flow	Not the typical site of diversion colitis
Diverted distal colon	Excluded from fecal stream	Characteristic anatomical setting
Reconnected segment	Fecal transit restored	Diversion-related inflammation can resolve

Why diversion causes colitis remains uncertain

Proposed microbiota change

Altered microbial composition or function after fecal-stream interruption.

Proposed nutritional deprivation

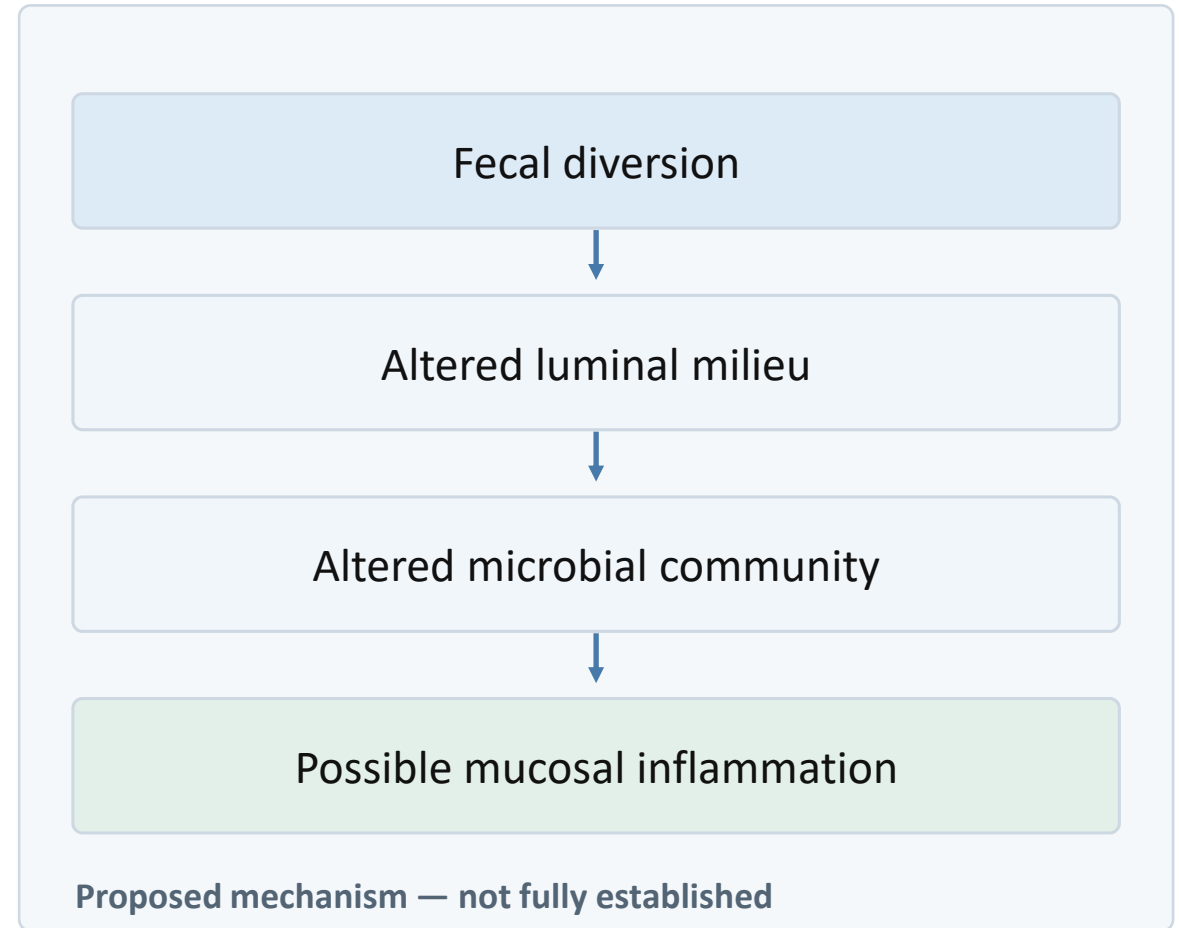
Reduced delivery of bacterial metabolites needed by colonocytes.

Observed reversibility

Improvement after flow restoration supports an exposure-dependent process.

The microbiota hypothesis

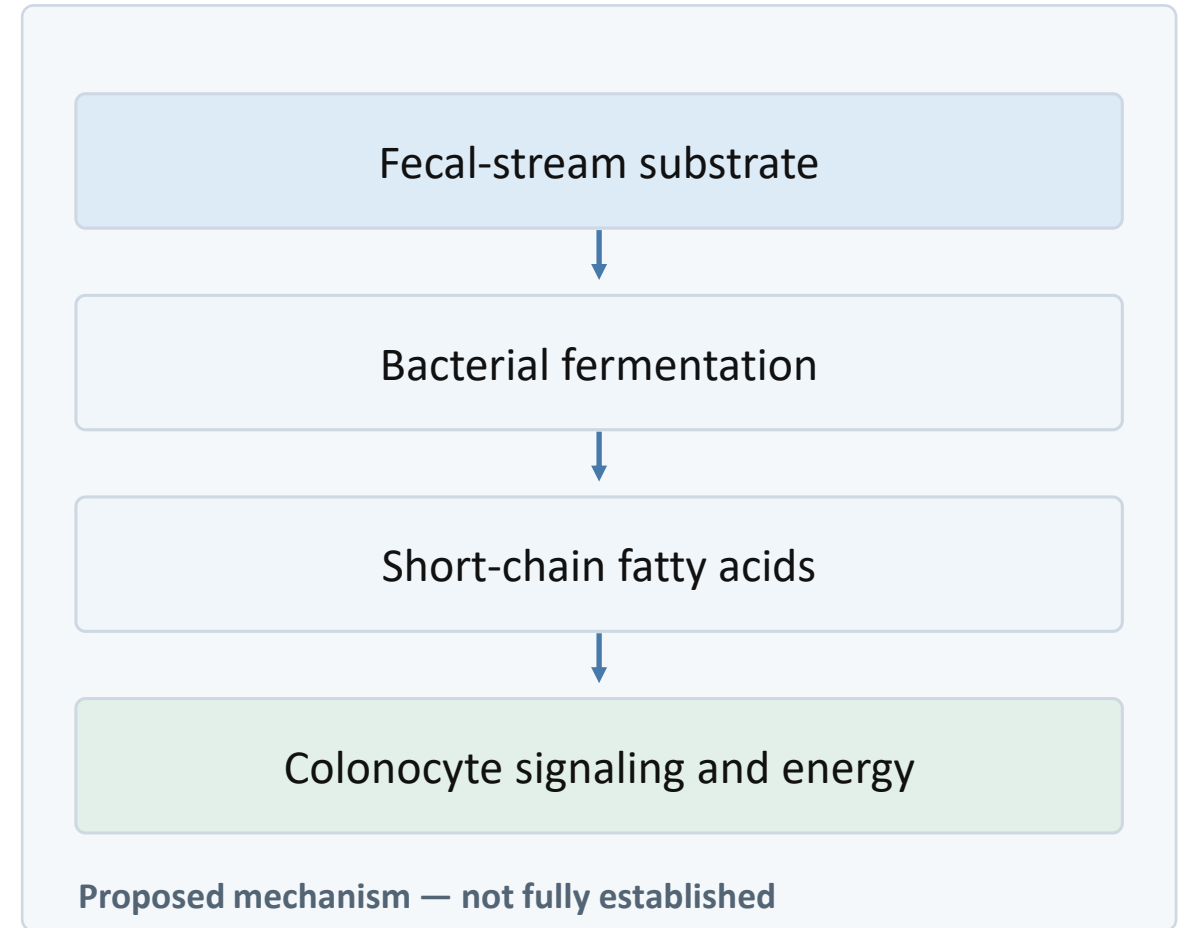
- Fecal diversion changes the luminal ecosystem.
- Changes in microbiota are a proposed contributor to mucosal inflammation.
- The exact organism-level and immune pathways are not resolved.



Microbiota alteration is a hypothesis, not a diagnostic laboratory test.

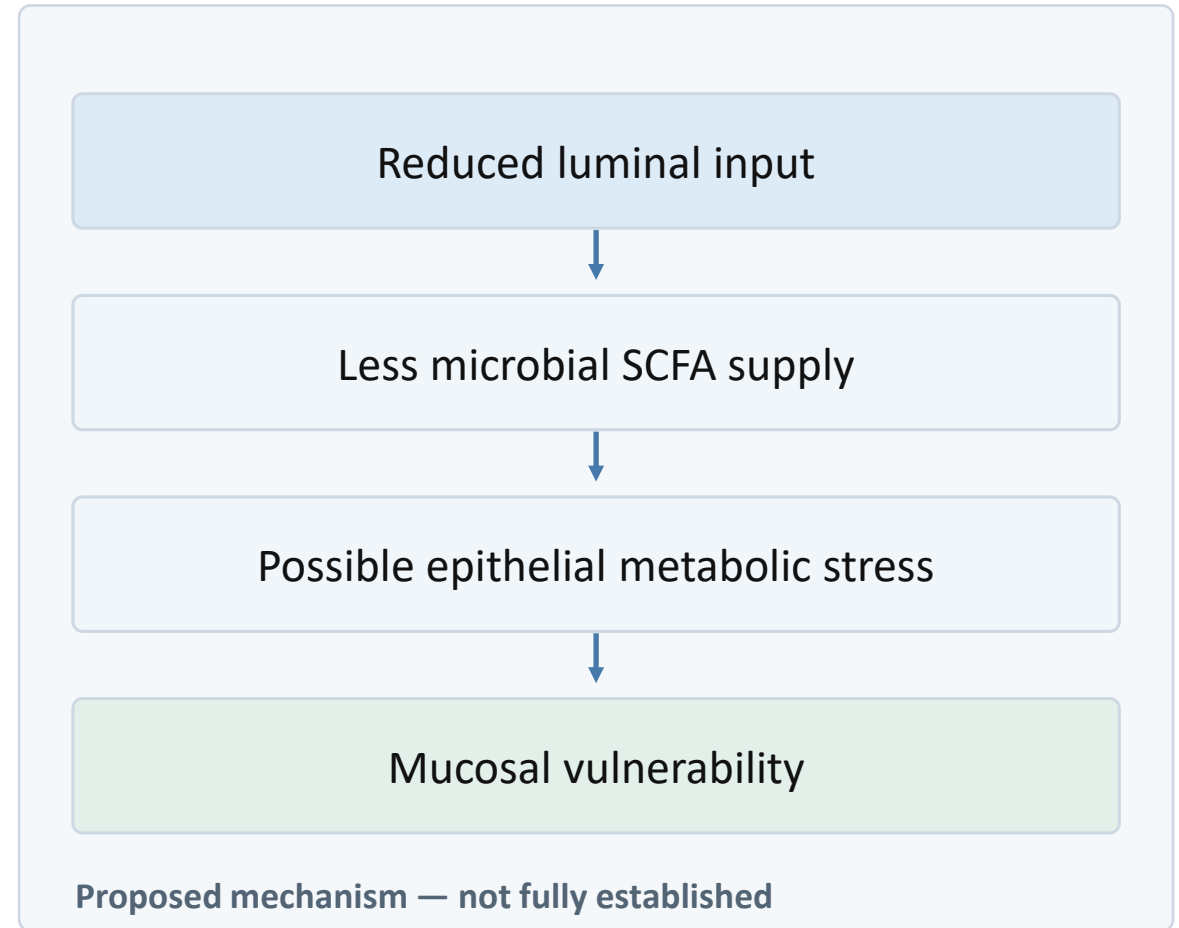
Short-chain fatty acids and the epithelium

- Luminal bacteria produce short-chain fatty acids.
- These metabolites provide an energy source for colonic epithelial cells.
- They also participate in epithelial signaling.



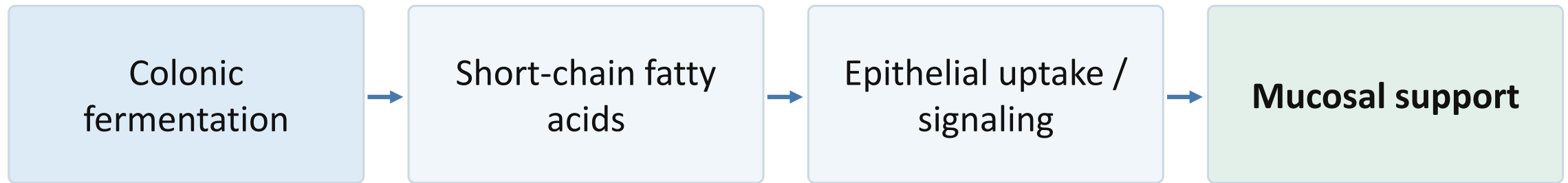
Luminal nutrient deprivation: Proposed sequence

- Fecal-stream exclusion may limit epithelial access to bacterial metabolites.
- This could reduce local metabolic support.
- The link to the complete inflammatory phenotype remains incompletely defined.



Describe an explanatory model without converting it into proven causality.

Butyrate is an illustrative short-chain fatty acid



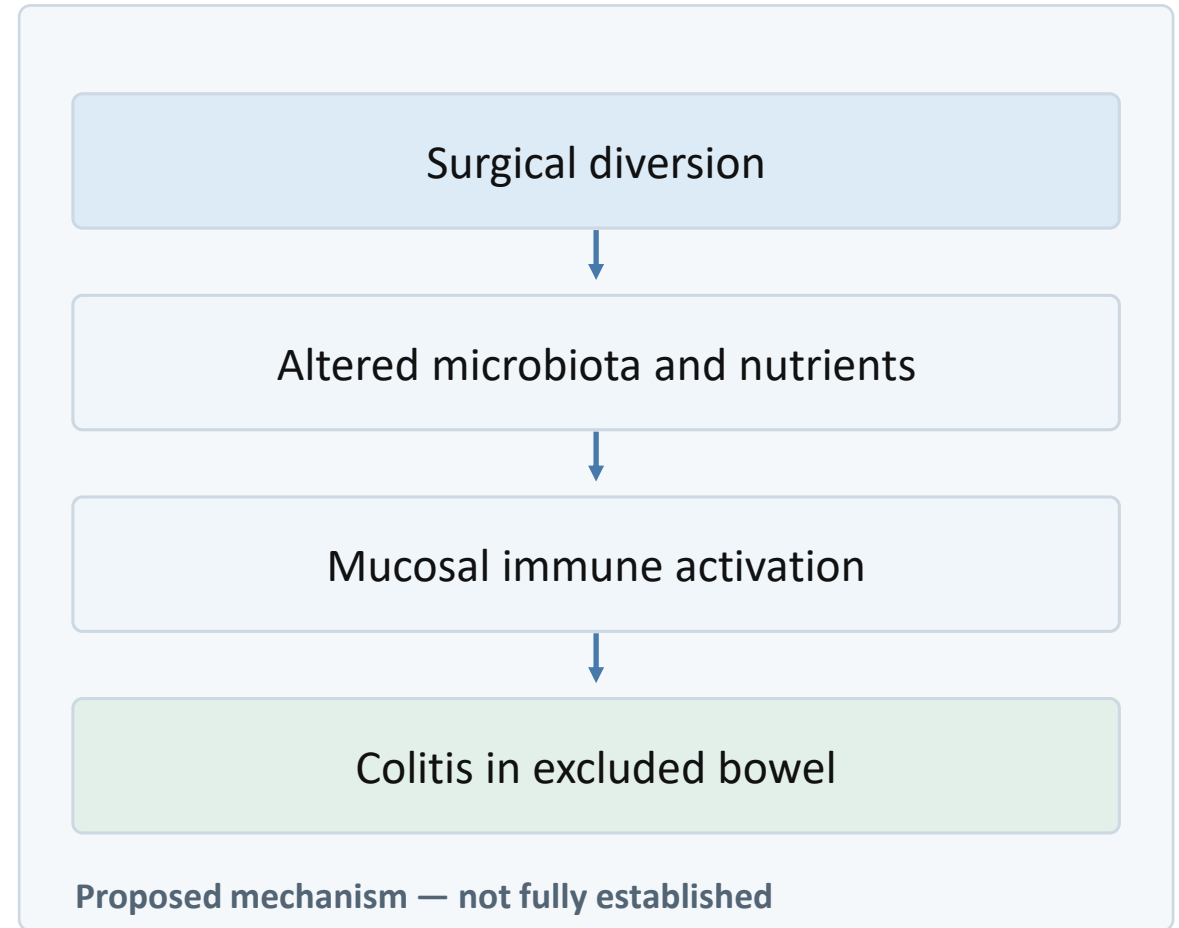
- Butyrate is one relevant short-chain fatty acid; The broader class.
- A single-metabolite model cannot explain every histologic finding.

Mechanism: Facts versus proposals

Observation	Status	Appropriate interpretation
Inflammation in defunctioned colon	Established	Typical anatomic setting
Prominent lymphoid follicles	Established	Important morphological clue
Altered luminal microbiota	Proposed	Plausible pathogenesis
Loss of SCFA supply	Proposed	Mechanistic support from topical response
Recovery with restored transit	Established	Strong clinical correlation

A unified pathogenesis model

- Surgical diversion changes luminal exposure.
- Microbiota and nutrient hypotheses may act together.
- Mucosal inflammation can become histologically conspicuous.



Endoscopy: Mucosal erythema

- The diverted mucosa may appear erythematous.
- Redness signals mucosal inflammation but is nonspecific.
- The anatomical context provides diagnostic specificity.

Endoscopic observations

Erythema

Friability

Nodularity may reflect follicles

Endoscopy: Mucosal friability

- Friability accompanies mucosal inflammation.
- A fragile surface is prone to contact-related bleeding.
- The finding overlaps with other inflammatory colitides.

Endoscopic observations

Erythema

Friability

Nodularity may reflect follicles

Endoscopic spectrum in a diverted segment

Robbins core

Erythema and friability are explicitly described.

Reported extensions

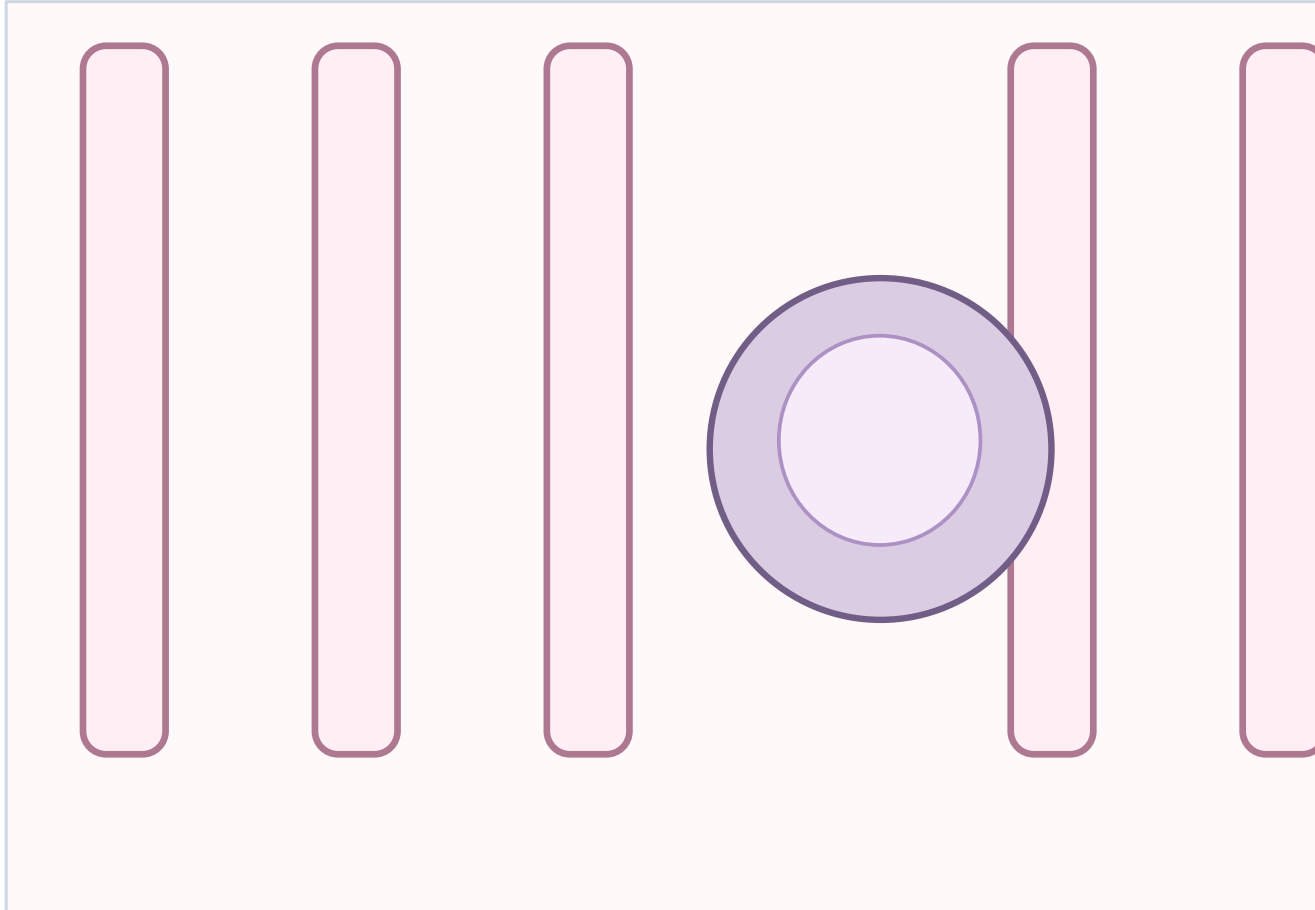
Granularity, edema, erosion, ulcers and nodularity occur in published series.

Interpretive rule

Neither endoscopic appearance nor severity alone identifies etiology.

Histologic hallmark: Lymphoid follicles

Schematic — not a histologic specimen

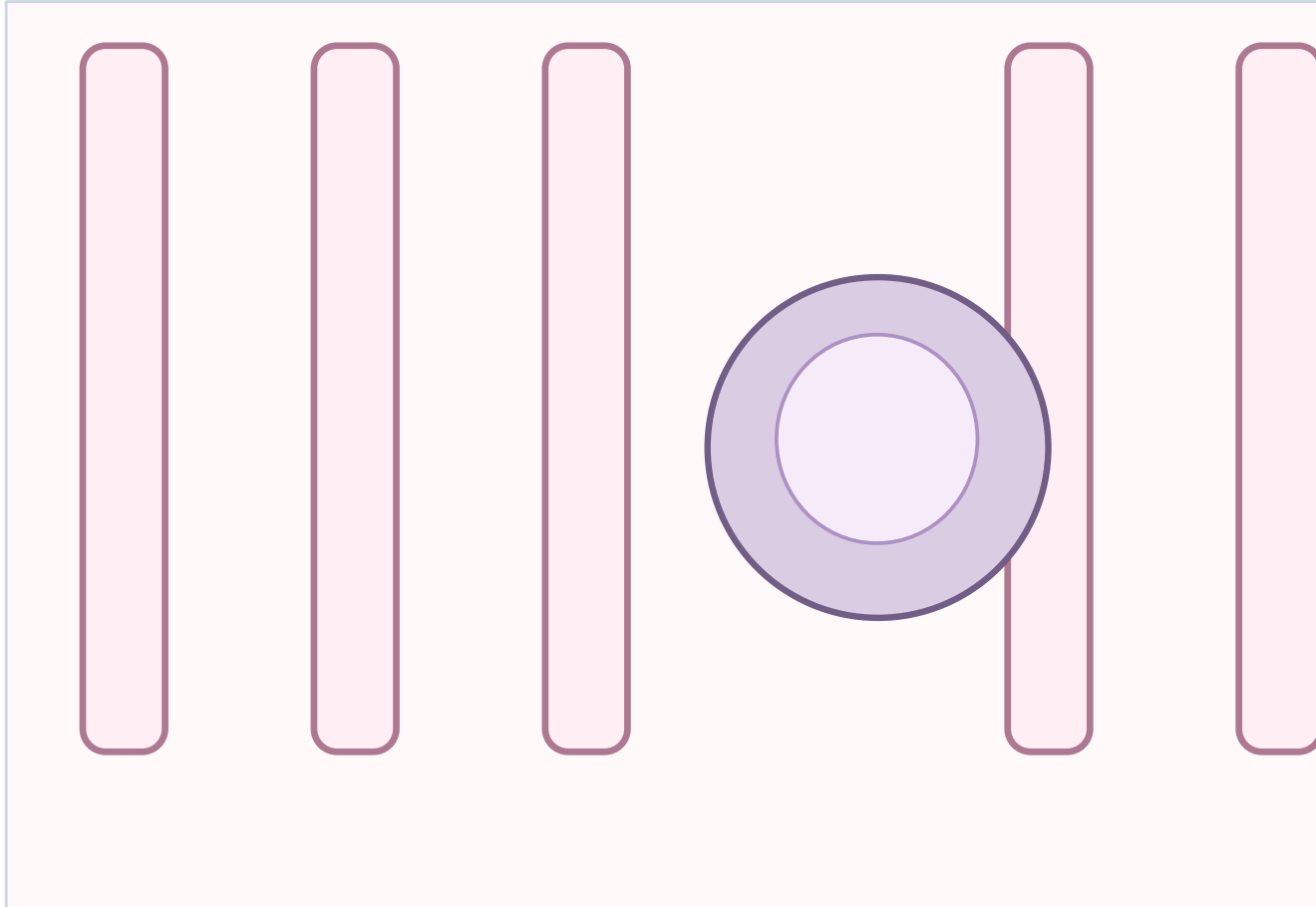


Expanded lymphoid follicle

- Numerous mucosal lymphoid follicles are the most striking Robbins feature.
- Enlarged follicular structures create a recognizable low-power pattern.
- This clue gains meaning when the biopsy is from diverted colon.

Lymphoid follicle architecture

Schematic — not a histologic specimen

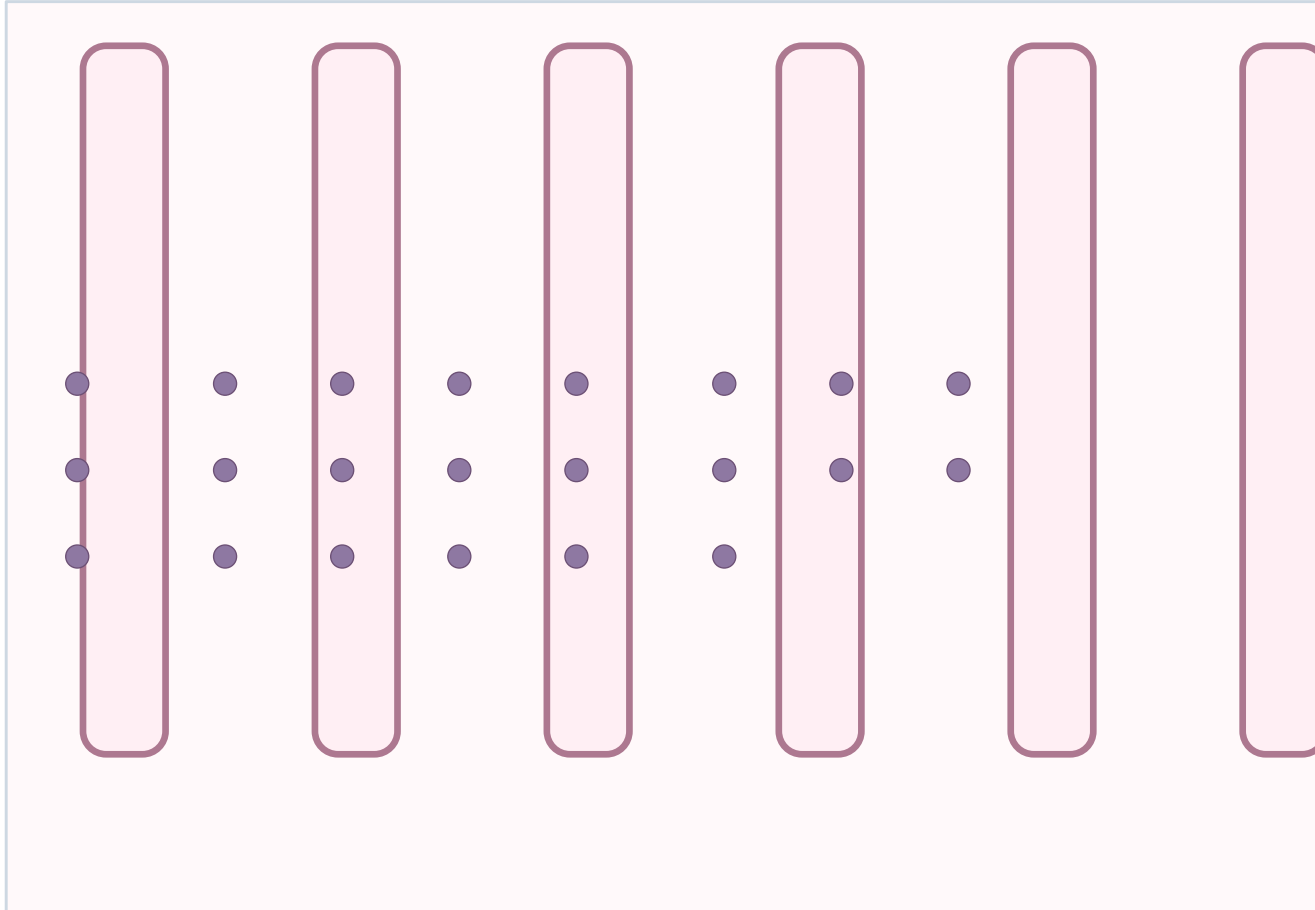


Expanded lymphoid follicle

- A reactive follicle may contain a germinal center.
- The lymphoid structure is distinct from epithelial crypts.
- Assess follicles together with surface and crypt injury.

Histology: Lamina propria inflammation

Schematic — not a histologic specimen



Lamina propria inflammatory cells

- The lamina propria may contain abundant inflammatory cells.
- Lymphocytes, monocytes, macrophages and plasma cells.
- The mixture may be conspicuous even without severe crypt injury.

Cell populations and their location

Lymphocytes

Chronic mucosal immune component; Prominent in follicles and lamina propria.

Plasma cells

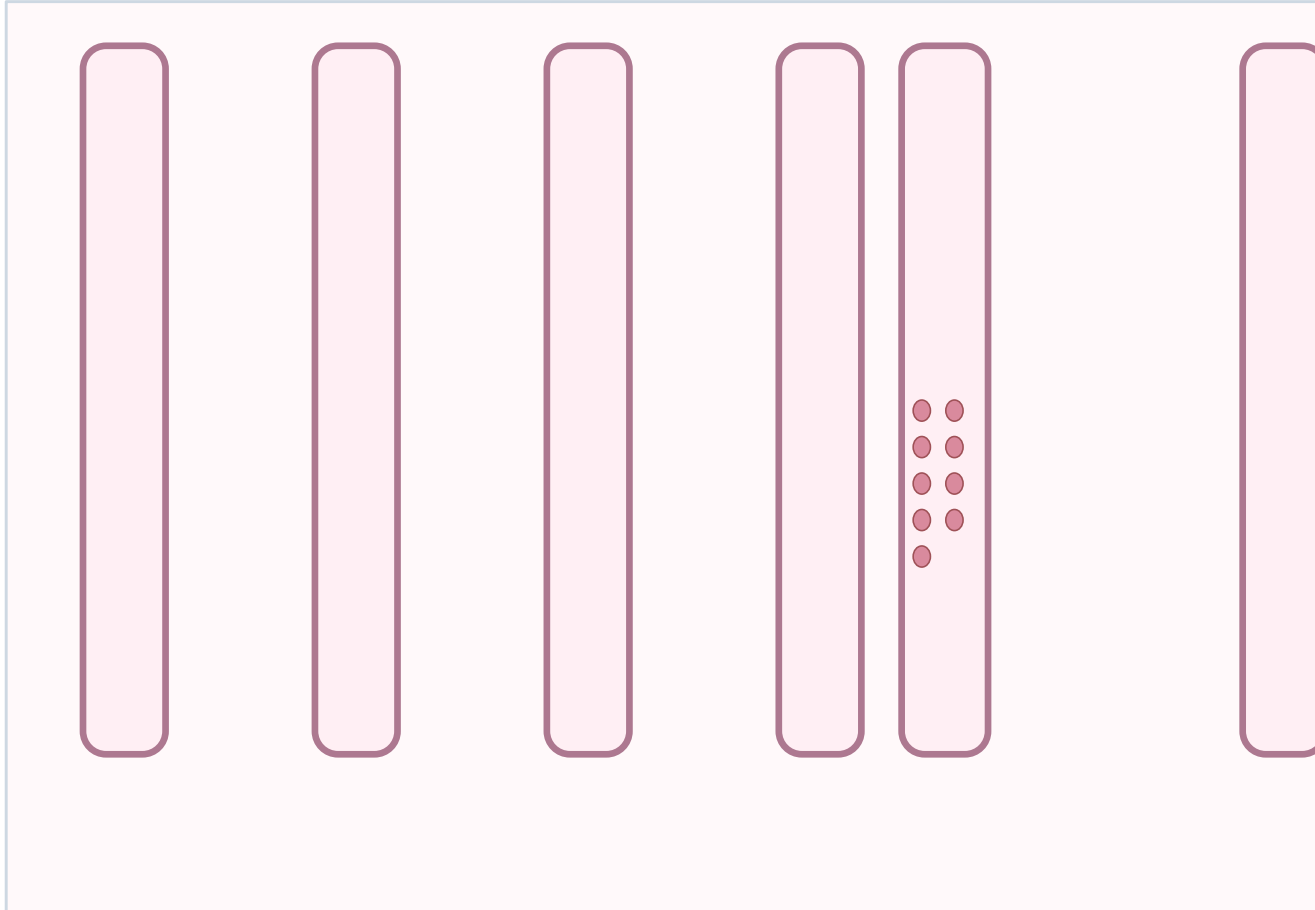
May contribute to a dense chronic lamina propria infiltrate.

Monocytes / macrophages

Additional mononuclear inflammatory populations.

Histology: Active crypt injury

Schematic — not a histologic specimen

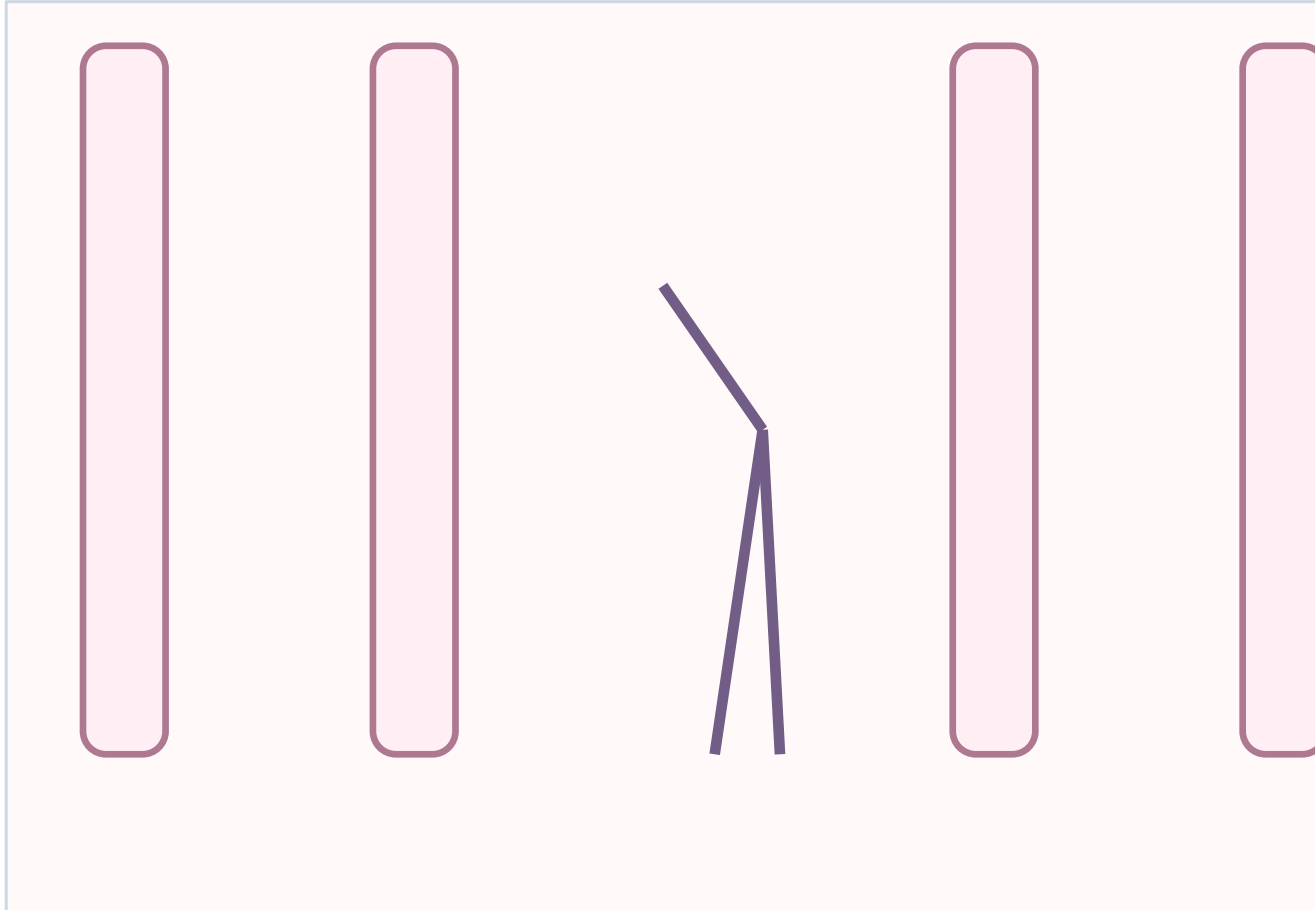


Neutrophils within a crypt

- Severe disease can include crypt abscesses.
- Crypt abscess denotes neutrophils collected inside a crypt lumen.
- This pattern is also seen in inflammatory bowel disease.

Histology: Altered crypt architecture

Schematic — not a histologic specimen



Branching / altered crypt orientation

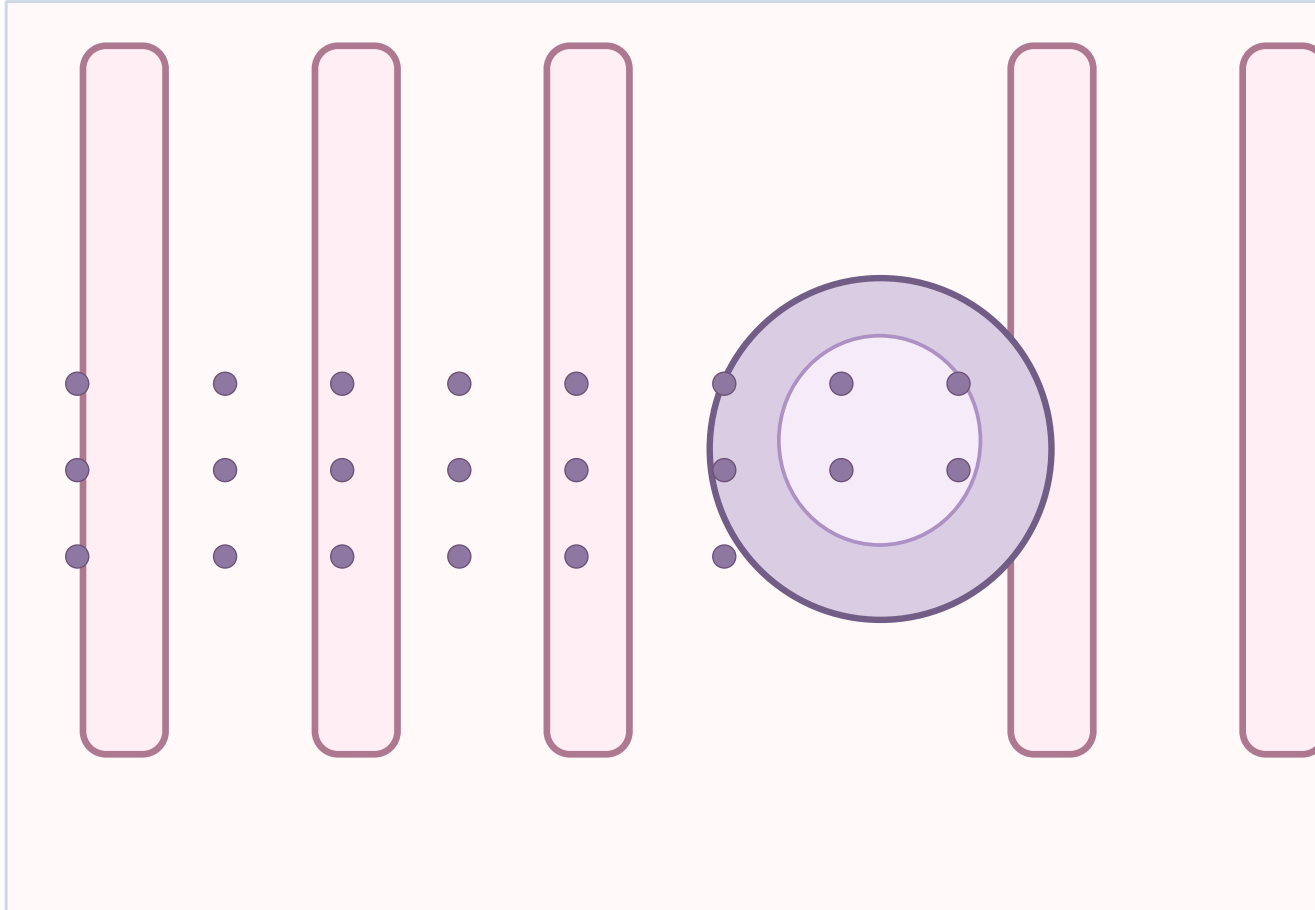
- Architectural distortion may accompany severe diversion colitis.
- Abnormal branching and orientation indicate mucosal injury and repair.
- Distortion alone must not establish a new diagnosis of chronic IBD.

Rare finding: Granulomas

- Granulomas can occur, but rarely.
- Granulomas are not sufficient to identify Crohn disease in a diverted segment.
- Their interpretation depends on morphology, background disease and clinical context.
- Do not treat rarity as exclusion or proof of a specific mechanism.

Spectrum of mucosal damage

Schematic — not a histologic specimen



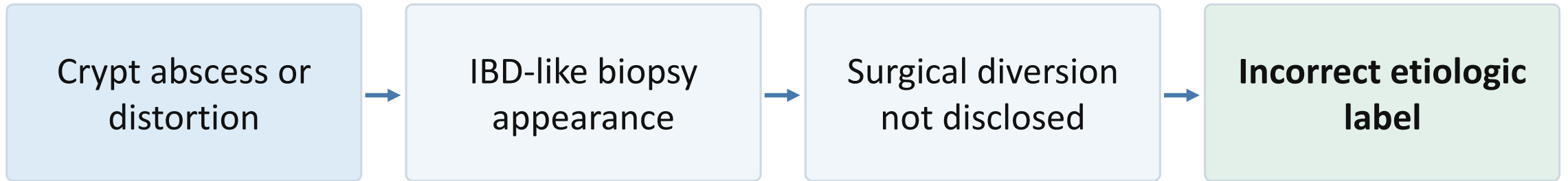
Expanded lymphoid follicle

- Follicular hyperplasia may dominate the biopsy.
- Inflammatory infiltrates and crypt activity can coexist.
- Severe examples may resemble primary IBD.

Hallmark versus nonspecific findings

Finding	Role in diversion colitis	Important caution
Prominent follicles	Characteristic / high-yield	Not absolutely specific
Lamina propria infiltrate	Common accompanying feature	Many causes possible
Crypt abscesses	Severe activity pattern	IBD and infections may show this
Crypt distortion	Severe / chronic injury	Cannot establish IBD alone
Granulomas	Rare possible feature	Do not automatically diagnose Crohn disease

Why histology alone may mislead



- Histologic resemblance does not establish the same disease mechanism.
- The surgical history must be reconciled with the tissue findings.

No single slide feature is diagnostic

Morphology

Follicles and inflammation strongly suggest a possibility.

Anatomy

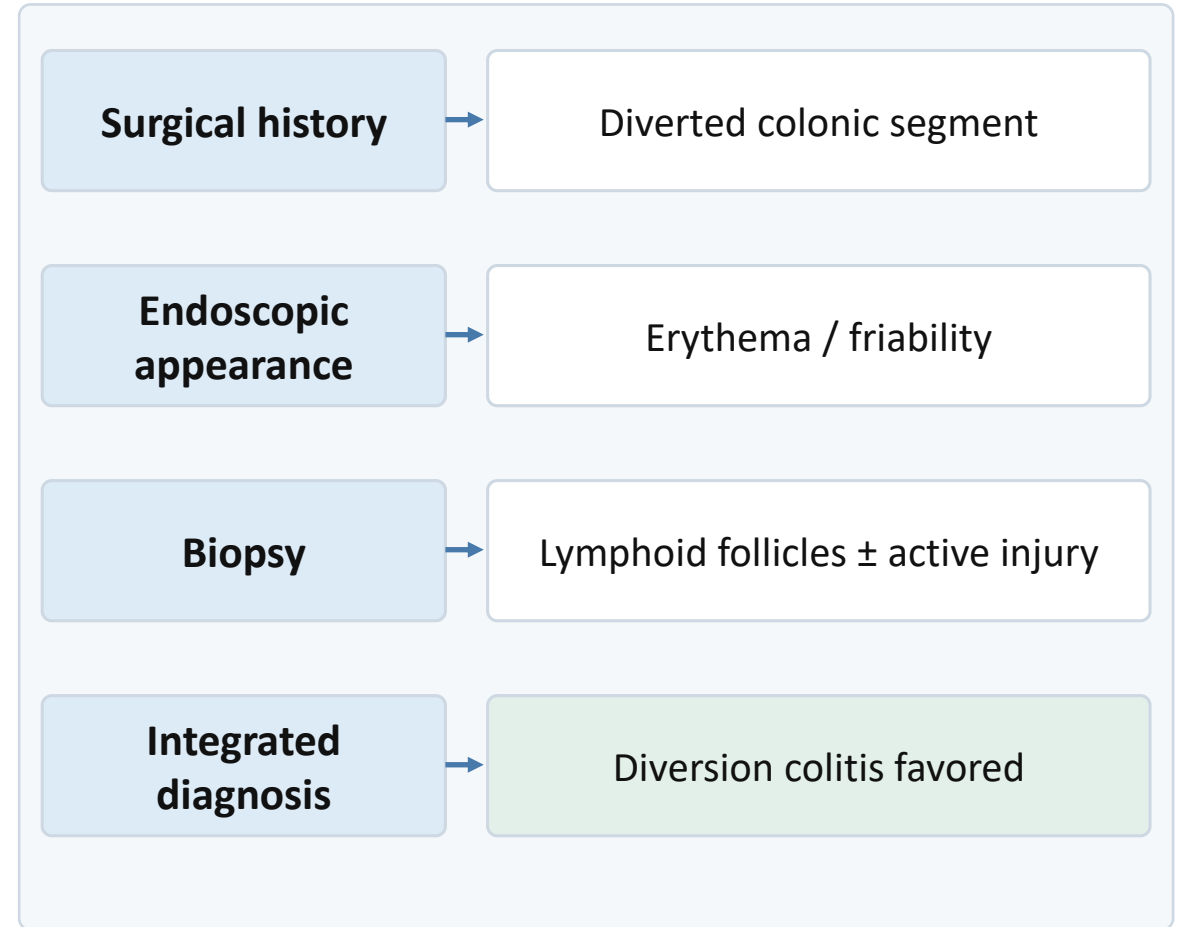
A bypassed distal colonic segment makes the diagnosis plausible.

Clinical setting

Pre-existing disease and timing alter the differential.

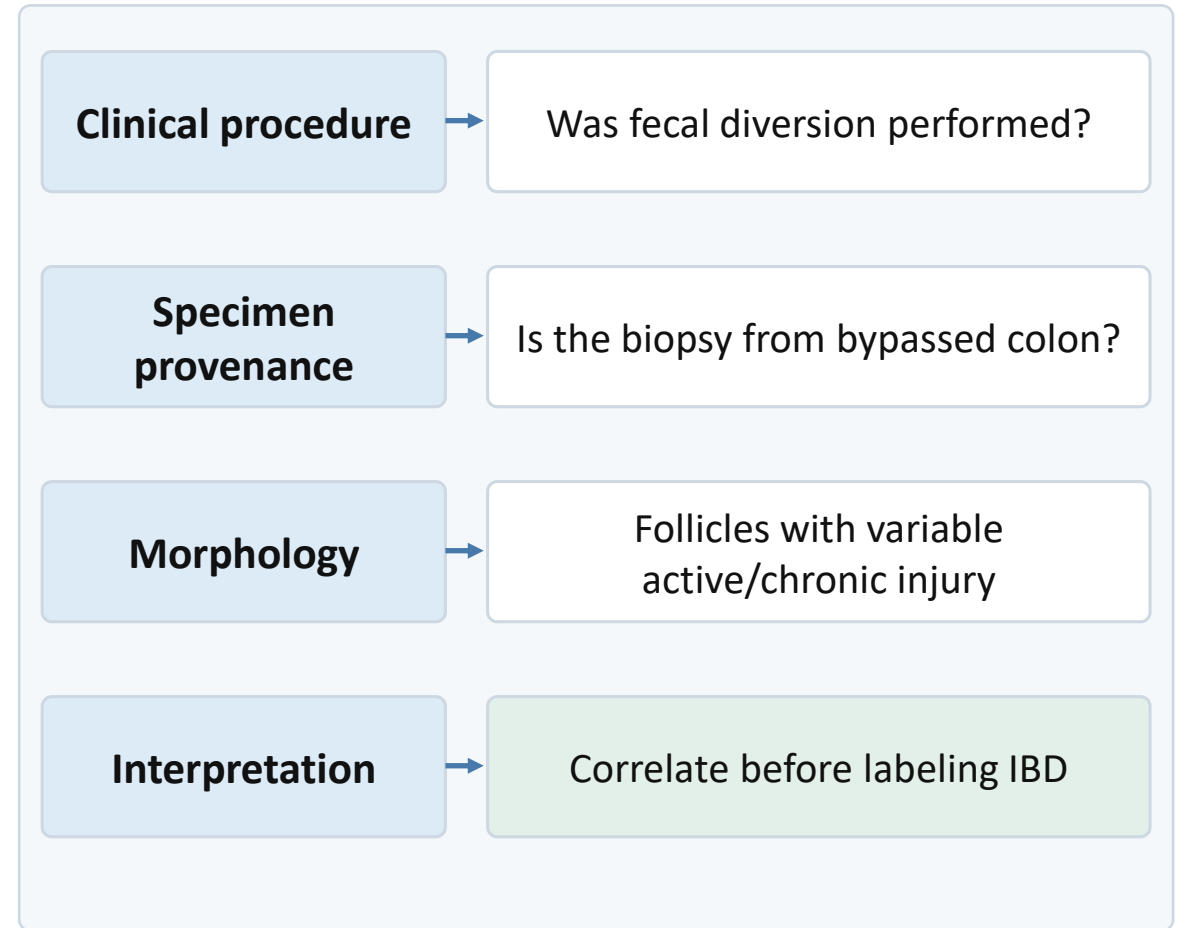
Diagnostic triangulation

- Surgical history establishes a diverted segment.
- Endoscopy documents mucosal inflammation.
- Histology identifies the pattern and its severity.
- A combined assessment supports diversion colitis.



What the requisition should communicate

- Type and approximate date of ostomy.
- Site of each biopsy relative to the fecal stream.
- Underlying disease: Ulcerative colitis, cancer, motility disorder, etc.
- Current symptoms and endoscopic findings, when known.



Interpretation: Pre-existing IBD matters

No antecedent IBD

Inflammation confined to diverted mucosa strongly supports diversion-associated injury.

Known ulcerative colitis

Residual UC and superimposed diversion changes may overlap.

Known Crohn disease

Patchy active injury or granulomas require careful correlation; Avoid single-feature inference.

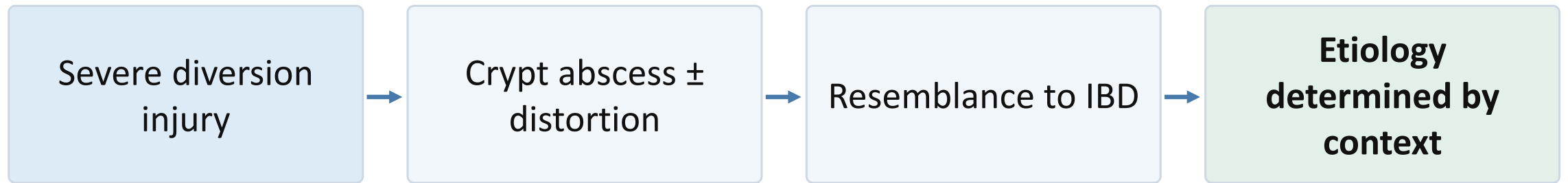
Diversion colitis versus ulcerative colitis

Feature	Diversion colitis	Ulcerative colitis
Defining setting	Diverted bowel segment	Primary chronic inflammatory disease
Anatomical distribution	Excluded segment	Continuous colonic / rectal disease
Lymphoid follicles	Often striking	May occur but not defining
Crypt abscesses	Possible if severe	Common active-disease feature
Interpretive pivot	Fecal-stream status	Overall chronic clinical course

Diversion colitis versus Crohn disease

Feature	Diversion colitis	Crohn disease
Precipitating setting	Surgical diversion	Primary immune-mediated bowel disease
Granulomas	Rarely possible	Can occur as hallmark of Crohn
Crypt changes	Possible with severe injury	Related to repeated damage / repair
Critical discriminator	Affected diverted segment	Distribution and transmural / extraintestinal features

IBD-like morphology does not equal IBD



- Robbins explicitly cautions that severe lesions can look like IBD.
- Restoration of fecal flow produces a different causal test from immunologic labeling.

Microscopic colitis: A distinct pattern

Feature	Diversion colitis	Microscopic colitis
Required context	Fecal diversion	No diversion requirement
Endoscopy	Often erythema / friability	Typically normal
Typical biopsy clue	Prominent follicles	Thick collagen band or excess intraepithelial lymphocytes
Clinical emphasis	Excluded segment	Chronic watery nonbloody diarrhea

Graft-versus-host disease: Different lesion

Feature	Diversion colitis	GI graft-versus-host disease
Setting	Excluded colon	Allogeneic stem-cell transplant
Dominant histology	Follicles + variable inflammation	Epithelial / crypt apoptosis
Lamina propria	Often cellular infiltrate	May be relatively sparse
Diagnostic key	Fecal diversion	Transplant history

Drug-related apoptotic enterocolitis

Checkpoint inhibitors

Can induce apoptotic enterocolitis in treated oncology patients.

Mycophenolate

Another association with gastrointestinal epithelial apoptosis.

Diversion distinction

Fecal-stream exclusion and follicles support a different clinicopathologic process.

Practical comparative differential

Clinical clue	Expected emphasis	Most useful discriminator
Diversion surgery	Follicular hyperplasia ± colitis	Excluded bowel anatomy
Long-standing continuous colitis	Crypt injury and chronicity	UC distribution and history
Patchy transmural phenotype	Granulomas / Crohn features	Full Crohn phenotype
Watery diarrhea; Normal scope	Collagen / IEL change	Microscopic colitis biopsy
HSCT or relevant drug	Crypt epithelial apoptosis	Exposure history

Symptoms can be absent

- Endoscopic or histologic diversion colitis may be clinically silent.
- Visible inflammation does not imply disabling symptoms.
- Clinical decisions should consider burden and indication for the ostomy.

Endoscopic observations

Erythema

Friability

Nodularity may reflect follicles

Symptoms when diversion colitis is symptomatic

Rectal discharge

Mucus and sometimes blood may pass from the distal stump.

Anorectal symptoms

Tenesmus or discomfort may occur.

Abdominal / rectal pain

Symptom severity varies across patients.

Differentiate symptoms from biopsy activity

Domain	What is observed	What cannot be inferred
Clinical	Pain, discharge, bleeding or none	Microscopic severity from symptoms alone
Endoscopic	Erythema, friability, other changes	Exact histology without biopsy
Microscopic	Follicles, infiltrate, active injury	Patient burden without clinical history

Report findings before causal attribution

Description

Prominent lymphoid follicles;
Variable chronic / active
mucosal inflammation.

Context

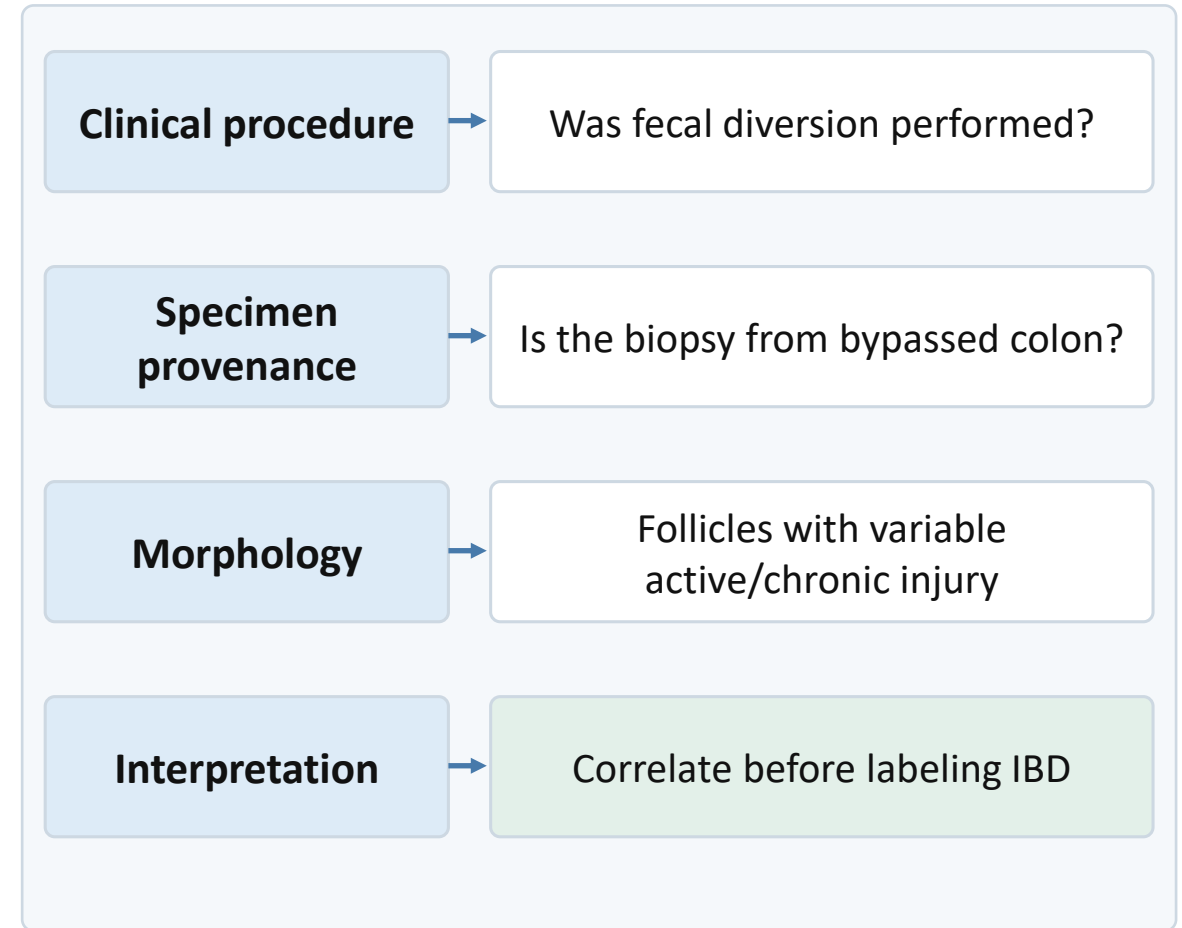
Biopsy is from a diverted distal
colonic segment.

Interpretation

Findings compatible with
diversion colitis, if clinical and
endoscopic context supports
it.

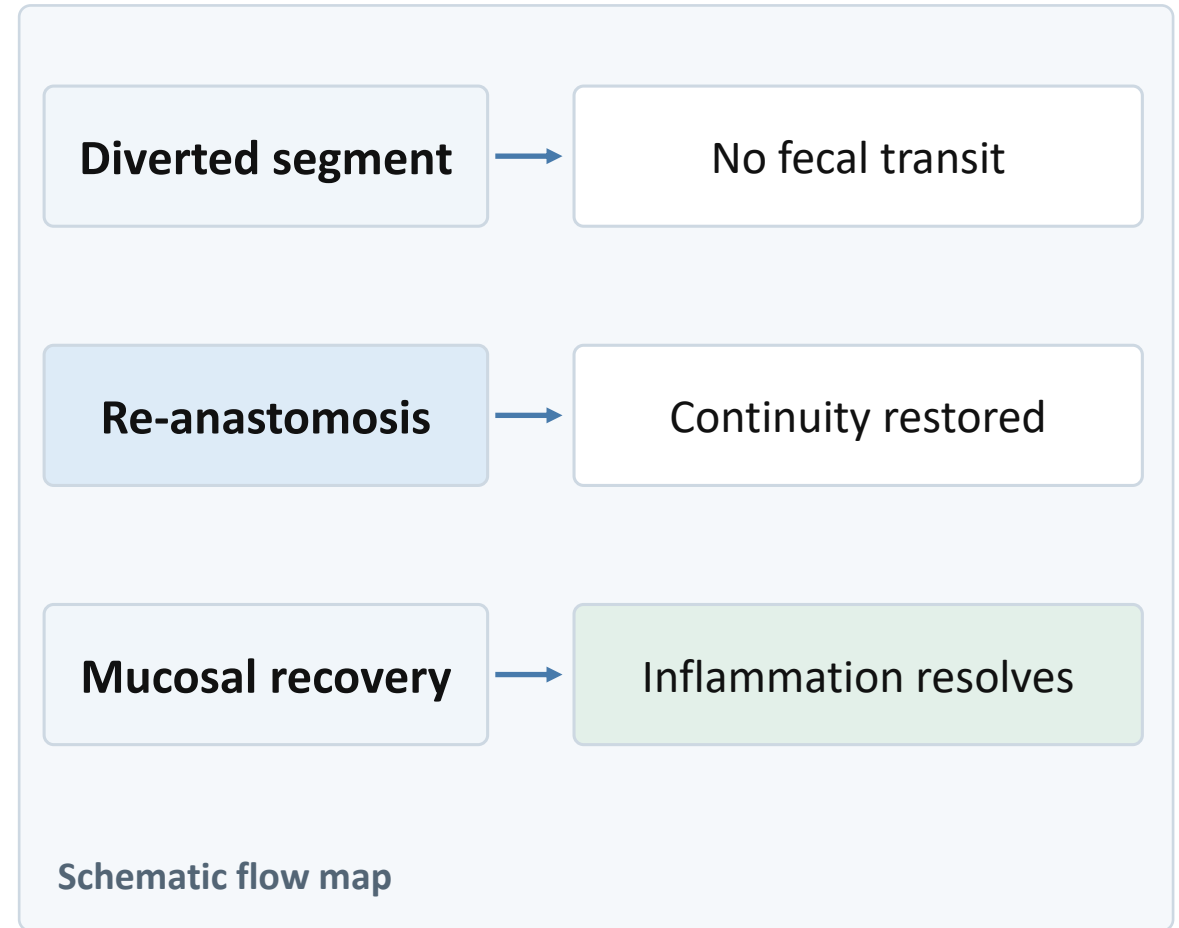
A safe reporting formulation

- “Colonic mucosa with prominent lymphoid follicular hyperplasia.”
- “Associated chronic inflammation; Active crypt injury if present.”
- “In a diverted segment, these changes are compatible with diversion colitis.”
- “Correlate with prior IBD and current anatomic distribution.”



Therapeutic target: Restore the fecal stream

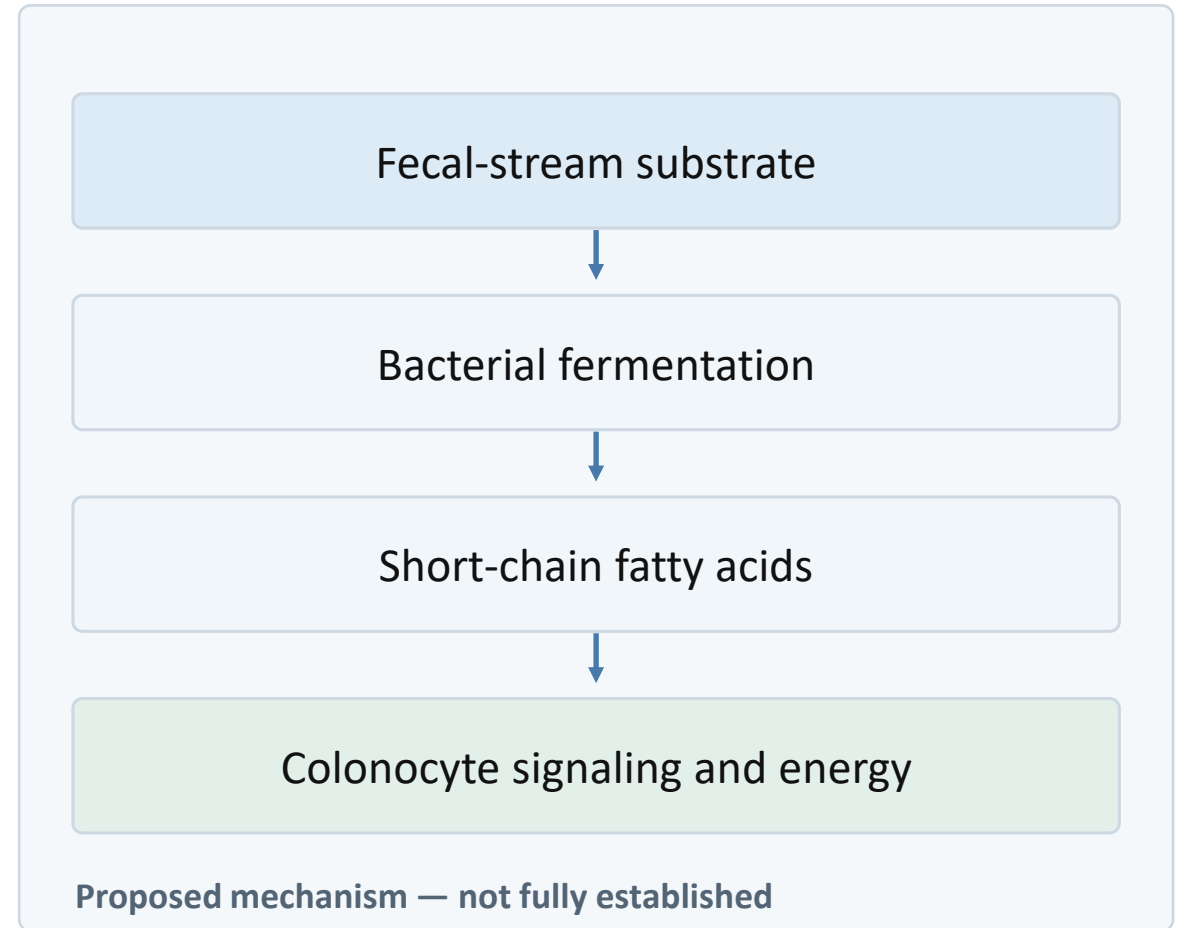
- Re-anastomosis restores bowel continuity.
- Diversion colitis resolves after restoration of flow.
- The response supports the importance of luminal exposure.



Restoration of continuity is the defining mechanistic intervention.

Short-chain fatty-acid enemas

- Deliver microbial metabolites directly to excluded mucosa.
- That these enemas can promote mucosal recovery.
- Their use is infrequent because of unpleasant odor.



How much does topical response prove?

Supported observation

Topical short-chain fatty acids can promote recovery.

Biological inference

Colonocyte substrate supply may contribute to pathogenesis.

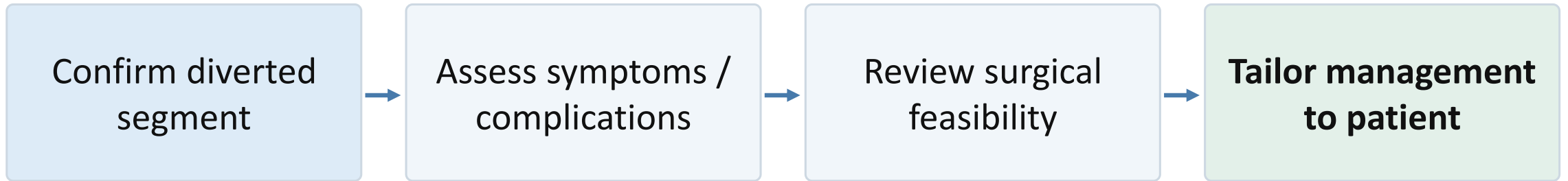
Boundary

Response does not establish one exclusive causal pathway or universal efficacy.

Additional treatments: Evidence is limited

- Published series describe topical mesalamine or corticosteroids in selected patients.
- There is no universally established severity scale or single standardized medical regimen.
- The surgical plan and feasibility of restoring continuity remain central.
- Avoid extrapolating efficacy from case reports into treatment guarantees.

Therapeutic decision: Anatomy and symptoms



- Anatomical restoration is etiologically directed when feasible.
- Topical approaches may be considered when restoration is not practical; Evidence remains variable.

Vignette 1: Excluded rectum

A patient has a prior colostomy for colon cancer. Endoscopy of the defunctioned rectal stump shows erythema and friability. Biopsy shows numerous lymphoid follicles with germinal centers.

What diagnosis is most consistent with these combined observations?

- Interpret location, clinical history and morphology together.

Vignette 1: Clinicopathologic reasoning

Surgical diversion + inflamed distal stump + follicular mucosal hyperplasia

1. Diversion colitis is favored.
2. The diagnostic anchor is the defunctioned segment, not a unique histologic cell.
3. Consider alternative or superimposed disease if the history indicates it.

Vignette 2: The Crohn disease trap

In a diverted segment, biopsy shows crypt abscesses, marked crypt distortion and a rare granuloma. The referral says only “rule out Crohn disease” and omits the ostomy history.

What information is needed before assigning a new Crohn diagnosis?

Vignette 2: Avoid a false disease label

Severe diversion colitis may resemble inflammatory bowel disease histologically.

1. Obtain surgical and pre-diversion diagnostic history.
2. Map specimens to the diverted and functioning bowel.
3. Reassess morphology in the complete distributional and clinical context.

Pitfalls that change patient care

Unreported diversion

Biopsy interpreted as de novo IBD despite excluded-bowel anatomy.

Overweighting granulomas

Rare granuloma mistaken for sufficient proof of Crohn disease.

Overweighting crypt abscesses

Active injury treated as etiologically specific.

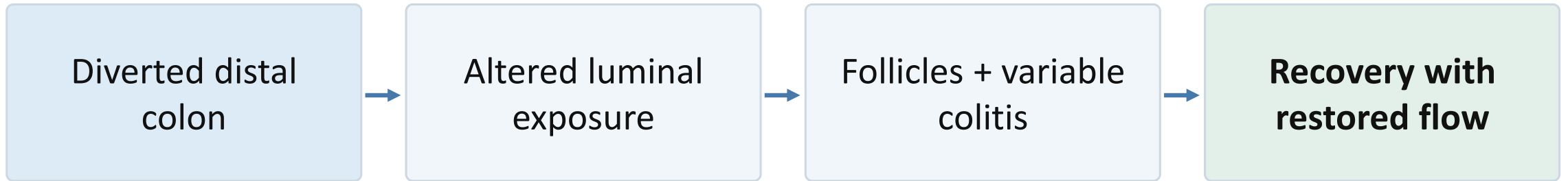
Assuming all symptoms reflect inflammation

Clinical and microscopic severity can diverge.

A practical diagnostic checklist

- Confirm the ostomy configuration and which segment was biopsied.
- Describe follicular hyperplasia and other mucosal changes precisely.
- Identify overlapping patterns of active or chronic injury.
- Compare with prior histology and established IBD, when relevant.
- Give a conditional integrated diagnosis rather than a stand-alone morphologic label.

Integrated model: Lesion to intervention



- The definitive teaching relationship is anatomical exposure → reversible mucosal injury.
- Intermediate microbial and nutrient mechanisms remain incompletely established.

Five points to retain

Setting

Fecal stream surgically diverted from distal colon.

Endoscopy

Erythematous, friable mucosa.

Histology

Prominent mucosal lymphoid follicles.

Severity

Can include crypt abscesses, distortion and rare granulomas.

Resolution

Restoration of fecal flow reverses diversion colitis.

Knowledge check: Morphology

A biopsy from a defunctioned colonic segment shows reactive lymphoid follicles, chronic inflammation, and scattered crypt abscesses.

Which finding is the distinctive clue, and what prevents overdiagnosing IBD?

Knowledge check: Interpretation

Characteristic clue: Prominent mucosal lymphoid follicles.

1. Crypt abscesses are not etiology-specific.
2. Diverted anatomy strongly supports diversion-associated inflammation.
3. Prior IBD and disease distribution must still be evaluated.

Knowledge check: Pathogenesis

Short-chain fatty-acid enemas improve mucosal inflammation in some diverted colonic segments.

What mechanistic hypothesis does this support, and what is still uncertain?

Knowledge check: Evidence boundary

The finding supports a role for microbiota-derived epithelial nutrients.

1. Short-chain fatty acids are microbial metabolites and epithelial energy sources.

2. Restoration of normal flow is associated with disease resolution.

3. The complete pathogenic pathway is not definitively established.

Selected references

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