

Chapter 9: Inflammatory Bowel Disease: Ulcerative Colitis and Crohn's

Summary

Inflammatory bowel disease (IBD) is a group of long-lasting conditions in which the immune system keeps attacking the gut wall. Its two main forms, ulcerative colitis and Crohn's disease, differ in where they strike and how deep the damage goes. This chapter follows Riya from her first symptoms to remission: how doctors find the inflammation with a stool test and a colonoscopy, how the treatment toolbox works (from steroids to biologic medicines), and why the modern goal is a healed gut lining, not just fewer symptoms. It also explores how gut inflammation reaches the brain, why anxiety and low mood are common during flares, and why stress and diet have not been shown to cause IBD, even though both can matter for how people feel.

Concepts Covered

1. Immune-Mediated Disease
2. Inflammatory Bowel Disease
3. Ulcerative Colitis
4. Crohn's Disease
5. Transmural Inflammation
6. Disease Flare
7. Remission
8. Extraintestinal Manifestation
9. IBD Risk Factors
10. Pediatric IBD
11. Colonoscopy
12. Fecal Calprotectin
13. Aminosalicylate
14. Corticosteroid
15. Immunomodulator
16. Biologic Therapy
17. Anti-TNF Therapy

- 18. JAK Inhibitor
- 19. Mucosal Healing
- 20. IBD Surgery

Prerequisites

- [Chapter 3: Meet the Microbiome](#) (gut microbes and dysbiosis)
- [Chapter 4: The Immune System as a Sense Organ](#) (inflammation, cytokines, and how they reach the brain)
- [Chapter 7: Stress and the Gut](#) (the HPA axis and how stress changes gut function)
- [Chapter 8: Pain, Sensitivity, and the Gut](#) (visceral pain and sensitization)

When the gut's defenses won't stand down



This is Riya's chapter. About 100,000 American kids and teens live with IBD, so there's a good chance someone in your school does, even if you'd never know it. Knowing what's actually happening inside their gut, and inside their head during a flare, makes it much easier to be a good friend to them. Let's check what the gut is really telling the brain!

9.1 A Word Riya Had Never Heard

Riya was 13 when the stomachaches started. At first they came and went, and her family blamed spicy food or a stomach bug. By the spring of eighth grade she was running to the bathroom several times a day, often with cramping pain low on the right side of her belly. She stopped finishing the dal and rice her grandmother made, because eating seemed to make the pain worse. Her jeans got loose. She fell asleep over homework, and her violin teacher asked if she was all right. (Riya is a fictional student who appears throughout this book.)

After months of this, her pediatrician ordered a stool test, then sent her to a pediatric gastroenterologist, Dr. Alvarez. A few weeks later Riya was lying in a recovery room after her first colonoscopy, still groggy, when Dr. Alvarez sat down with her parents and said a word Riya had never heard: "Crohn's." Riya was 14. Her first question was, "Did I do something to cause this?" Her second was, "Will it go away?" This chapter answers both questions honestly. The short answers are "no" and "not completely, but it can be controlled."

9.2 A Security System That Won't Stand Down

Before any technical names, picture a building with a very good security system. Normally, when a real intruder shows up, the alarm sounds, guards rush in, the intruder is handled, and the guards go back to their posts. Now imagine the system starts treating the building's own friendly visitors as intruders. The alarm keeps ringing, the guards keep arriving, and in the scuffle they break windows and damage walls. Nobody broke in. The damage comes from the defenders themselves.

That is roughly what happens in inflammatory bowel disease. Your gut lining is a border between your body and trillions of microbes (Chapter 3), and your gut immune system patrols that border constantly (Chapter 4). In IBD, the patrol overreacts and won't calm down.

An **immune-mediated disease** is a condition in which the body's own immune response, rather than an outside germ or a toxin, causes most of the damage. IBD belongs to this family, along with conditions such as rheumatoid arthritis and psoriasis.

Inflammatory bowel disease (IBD) is a group of chronic conditions in which abnormal immune reactions cause long-lasting, recurring inflammation of the digestive tract. "Chronic" means long-lasting, often lifelong. [The National Institute of Diabetes and Digestive and Kidney Diseases \(NIDDK\)](#) describes both main forms of IBD as chronic diseases in which abnormal reactions of the immune system cause inflammation in the gut. A [2021 review](#) in *Lancet Gastroenterology & Hepatology* calls IBD a lifelong condition with no cure.

"No cure" can sound scary. The other half of the story is that there are many treatments that can calm the inflammation, let the gut heal, and keep people well for long stretches.

Inflammation is the real target



I want you to hold one idea through this whole chapter: in IBD, the symptoms come from inflammation you can actually see and measure in the gut wall. That's what makes IBD different from IBS in Chapter 10, where the gut looks normal but the gut-brain conversation is off.

9.3 Two Main Types: Ulcerative Colitis and Crohn's Disease

IBD has two main forms. (A third, called microscopic colitis, mostly affects older adults and isn't covered here. And some people, especially young children, have colitis that doesn't clearly fit either main form at first; doctors call this IBD-unclassified, according to the [revised Porto criteria](#) for diagnosing IBD in children.) They share a lot, but they differ in two big ways: **where** the inflammation happens and **how deep** it goes.

Ulcerative colitis (UC) is a form of IBD in which inflammation affects only the large intestine (colon) and rectum, starting at the rectum and spreading upward in one continuous stretch, and stays in the inner layers of the gut wall. Recall the four gut wall layers from Chapter 1: mucosa, submucosa, muscularis, and serosa. In UC, the inflammation stays in the inner layers, the mucosa and submucosa, according to the [StatPearls review of ulcerative colitis](#). Most of it is in the innermost lining, the mucosa, which is how the [FDA](#) describes UC. The inflamed lining develops sores called ulcers, which bleed. A 2023 review in *The Lancet* by [Le Berre and colleagues](#) says people with UC usually first see a doctor because of bloody diarrhea. Other common symptoms, according to NIDDK, are belly cramps, mucus in the stool, an urgent need to go, and a feeling of needing to go even when the bowel is empty.

Crohn's disease is a form of IBD that can affect any part of the digestive tract, from the mouth to the anus, most often the end of the small intestine (the ileum) and the beginning of the colon, often in patches with

healthy sections in between. Doctors call those healthy gaps "skip lesions." Riya's Crohn's affects the end of her small intestine and part of her colon, the most common pattern. A 2024 *Lancet* review by [Dolinger, Torres, and Vermeire](#) describes Crohn's inflammation as often patchy and segmental, commonly in the terminal ileum and nearby colon. NIDDK lists diarrhea, belly cramps and pain, and weight loss as the most common symptoms.

The second big difference is depth. **Transmural inflammation** is inflammation that extends through the entire thickness of the gut wall, from the inner lining all the way to the outer layer. ("Trans" means across, and "mural" means wall.) Crohn's disease is transmural; ulcerative colitis usually is not.

Why does depth matter? Picture a garden hose. If irritation eats through every layer, the hose can scar and narrow, or spring a hole that tunnels into something nearby. Deep inflammation in Crohn's can cause:

- **Strictures:** scarred, narrowed sections of bowel that can block the flow of food and stool.
- **Fistulas:** abnormal tunnels from the bowel to another organ or to the skin.
- **Abscesses:** pockets of pus from infection.

According to the [StatPearls review of Crohn's disease](#), about half of patients develop one of these complications within 20 years of diagnosis. This is one reason doctors now treat Crohn's early and aim to heal the lining before damage builds up (Section 9.9).

The table sums up the differences.

Feature	Ulcerative colitis	Crohn's disease
Where	Colon and rectum only	Anywhere from mouth to anus; most often end of small intestine and start of colon
Pattern	Continuous, starting at the rectum	Patchy, with healthy "skip" areas
Depth	Inner layers (mostly the mucosa)	Transmural (through the whole wall)
Typical first complaint	Bloody diarrhea	Diarrhea, belly pain, weight loss
Possible complications	Severe bleeding, a dangerously swollen colon	Strictures, fistulas, abscesses
Can surgery remove the disease?	Removing the colon and rectum removes the disease from the gut	No; it can come back in another part of the gut

A memory hook for the two types



Ulcerative **colitis** has "colon" right in the name, so it stays in the colon and rectum. **Crohn's** can "roam": any part of the gut, in patches, through every layer.

9.4 How Common Is IBD, and Who Gets It?

IBD isn't rare, though counting it is tricky. One method uses insurance claims, the billing records that show who was actually diagnosed and treated. Another is a survey asking adults whether a doctor ever told them they had the disease. They measure different things, so they give different numbers.

Using claims data, a [2023 study](#) in *Gastroenterology* led by James Lewis estimated that IBD is diagnosed in about 721 of every 100,000 Americans, roughly 0.7% of the population, or about 2.39 million people. A 2025 study by [Kappelman and colleagues](#), using the same kind of data, found about 122 cases per 100,000 people under age 20, or roughly 100,000 young people.

National surveys give a higher number: in 2023 to 2024, 1.6% of US adults (about 4.1 million) said a doctor had told them they had Crohn's disease or ulcerative colitis, according to a [2026 report from the National Center for Health Statistics](#). Surveys count anyone who was ever told they had the disease, which can include old or mistaken diagnoses, so the claims figure is probably the better estimate of how many people are living with diagnosed IBD. (Claims data have gaps too: they can't count people who had no insurance record in the years studied.)

IBD can start at any age, but it most often begins in young adulthood. The Lewis study found that new diagnoses peak in people's twenties. NIDDK says UC most often begins between ages 15 and 30, and Crohn's is most likely to begin between ages 20 and 29. The Lewis study also found that IBD is diagnosed less often in Black, Asian, and Hispanic Americans than in white Americans, although a 2023 *Lancet* review notes that IBD is becoming more common around the world.

Pediatric IBD

Pediatric IBD is inflammatory bowel disease that begins in childhood or the teen years, before adulthood. Riya, diagnosed at 14, is a pediatric case. Kids and teens face extra challenges because their bodies are still growing. NIDDK lists slowed growth, lower weight gain, and delayed puberty among possible complications of IBD in children, and long courses of steroids can also slow growth and weaken bones.

That is one reason pediatric care has changed. A 2026 study of 787 commercially insured US children newly diagnosed with Crohn's disease ([Gonzalez and colleagues](#), *American Journal of Gastroenterology*) found that among children diagnosed in 2019 to 2023, 75% had started an advanced medicine, such as a biologic, within a year of diagnosis, a big increase over earlier years. This observational study shows a change in how doctors treat children, not proof that the change improves long-term outcomes.

IBD risk factors

So what causes IBD? Nobody knows exactly. **IBD risk factors** are the inherited and environmental factors that raise a person's chance of developing IBD without, on their own, causing it. Researchers think IBD develops when a person with a genetic tendency meets certain environmental exposures, and the immune system's relationship with gut microbes goes off track. Le Berre's 2023 review summarizes it this way for UC: genetic predisposition plus environmental exposures, with barrier defects, microbes, and a dysregulated immune response all strongly involved.

Here are the main risk factors with good human evidence:

- **Genes and family history.** IBD runs in families. NIDDK notes that having a parent or sibling with IBD raises a person's chance of developing it. In 2001, a team led by Jean-Pierre Hugot reported in *Nature* that variants in a gene called NOD2 were linked to Crohn's disease. NOD2 makes a protein that helps immune cells recognize bacterial parts, a clue that the problem involves how the immune system reads gut microbes. Many more gene regions have been found since, but each one raises risk only a little. Most people who carry a risk variant never get IBD.
- **Smoking.** NIDDK says research has shown smoking may double the chance of developing Crohn's disease.
- **The microbiome.** Studies find differences between the gut microbes of people with and without IBD (NIDDK). This is a correlation: inflammation itself and its treatments also change the microbiome, so scientists are still sorting out which comes first.

Notice what is *not* on that list as a proven cause: stress or "bad diet." Large studies have found some links worth investigating. For example, a 2021 study that followed more than 116,000 adults in 21 countries for about 10 years ([Narula and colleagues, BMJ](#)) found that people who ate more ultra-processed foods were more likely to be diagnosed with IBD later. That is a correlation, not proof of cause, and it says nothing about any one person. Stress and diet come up constantly online, and both deserve a Hype Meter check. (Need a refresher on how the grades work? See [the Hype Meter](#).)

Hype Meter: "IBD is caused by stress"

Strong

Moderate

Preliminary

Hype

Verdict: Hype. No good evidence shows that stress causes IBD to start; the causes involve genes, the immune system, gut microbes, and the environment. One large study of more than 150,000 US nurses found that women with recent depression symptoms were more likely to be diagnosed with Crohn's disease later (but not ulcerative colitis), an association that doesn't show cause ([Ananthakrishnan and colleagues, 2013](#)). A 2009 review by Singh, Graff, and Bernstein found strong evidence for an *association* between perceived stress and flares in people who already have IBD, and a weaker link with stressful life events. That is correlation, not cause: a flare can raise stress just as easily as stress might nudge a flare.

What would change the grade: More large, long-term studies following healthy people before diagnosis, showing that stress consistently predicts who develops IBD after accounting for genes, smoking, and other factors, plus evidence of how it would work. Even then, "stress is one risk factor" would be very different from "stress causes IBD."

9.5 Flares and Remission

IBD rarely stays the same for long. It tends to come in waves.

A **disease flare** is a period when IBD becomes active, with increased inflammation and symptoms such as diarrhea, pain, bleeding, and tiredness. **Remission** is a period when IBD is quiet: symptoms fade or disappear, and ideally the inflammation settles too. NIDDK says periods of remission can last for weeks or years, and that the goal of treatment is to keep people in remission long term.

Think of it like the seasons in a dry forest. A flare is like a wildfire season, and remission is the quiet season in between. The quiet season is real and can last a long time, but the underlying conditions haven't vanished. Firefighters don't sell their trucks when the fires stop. In the same way, people with IBD usually keep taking maintenance medicine during remission, because it is the medicine that keeps the quiet season going. NIDDK says people with ulcerative colitis typically need lifelong treatment with medicines unless they have surgery to remove the colon and rectum.

Doctors distinguish clinical remission (the person feels well) from endoscopic healing (the gut lining looks healed on a camera exam). These don't always match: a person can feel fine while inflammation still simmers, which is why doctors keep checking (Section 9.7).

Flares can be set off by several things. Stopping or missing maintenance medicine is one. Gut infections can trigger symptoms, and the [Singh review](#) notes that some pain relievers called NSAIDs (such as ibuprofen) have been linked to flares, which is why NIDDK advises people with IBD to talk with their doctor before taking over-the-counter medicines. Often a flare has no obvious trigger at all, which is frustrating, and is nobody's fault.

Remission is not a reason to stop medicine on your own



Feeling great in remission is exactly what the medicine is supposed to do, not proof that you don't need it anymore. Any change to an IBD treatment plan is a decision to make with your care team, never on your own.

9.6 More Than the Gut: Extraintestinal Manifestations

IBD lives in the gut, but its effects don't stay there. An **extraintestinal manifestation** is a sign of IBD that shows up outside the intestines. ("Extra" means outside, and "intestinal" means the intestines.) NIDDK lists several:

- **Joints:** certain types of arthritis, with sore, swollen joints.
- **Skin:** rashes, including red, tender bumps under the skin.
- **Eyes:** redness, pain, or irritation.
- **Liver and bile ducts:** including a condition called primary sclerosing cholangitis (PSC).
- **Blood and bones:** anemia (too few red blood cells, which causes tiredness) and thinning bones, which can come from the disease and from long-term steroid use.
- **Growth:** slowed growth and delayed puberty in children.

An international expert group (the [EXTRA consensus](#), published in 2022) noted that these manifestations happen frequently in IBD. They make sense once you remember that IBD is an immune disease. Immune cells and messenger molecules called cytokines (Chapter 4) travel in the blood, so inflammation that starts in the gut can affect tissues elsewhere.

The same idea explains effects on the brain, which we'll reach in Section 9.10.

9.7 Finding the Inflammation: Fecal Calprotectin and Colonoscopy

Belly pain and diarrhea can come from many conditions, including infections, IBS, and celiac disease. NIDDK describes the usual path to an IBD diagnosis: a medical and family history, a physical exam, and then blood tests, stool tests, endoscopy, and imaging.

The stool test: a smoke detector for gut inflammation

Riya's first clue came from a stool test. **Fecal calprotectin** is a protein released mainly by white blood cells called neutrophils that can be measured in stool; high levels signal inflammation in the intestines. When the gut wall is inflamed, neutrophils pour into it and some spill into the stool, carrying calprotectin with them. A 2019 review by [Ricciuto and Griffiths](#) explains that calprotectin levels rise in proportion to the degree of inflammation, which is why the test is useful for both screening and monitoring.

Think of fecal calprotectin as a smoke detector: it can tell you there is probably a fire somewhere in the intestines, but not where it is or what started it. A 2023 meta-analysis ([Dajti and colleagues](#); 17 studies, 1,956 adults) found that the test correctly flagged about 86% of people who had IBD and correctly cleared about 92% of people who had IBS instead. All the included studies had high or unclear risk of bias, so the exact numbers are uncertain. A high result leads to the next step.

Colonoscopy: looking directly

Colonoscopy is a procedure in which a doctor passes a long, thin, flexible tube with a light and a tiny camera through the anus to look at the lining of the rectum and entire colon, and often the end of the small intestine, usually while the patient is sedated. NIDDK says endoscopy tests, which include colonoscopy, are the most accurate way to diagnose Crohn's disease. During the colonoscopy, the doctor takes biopsies: tiny pieces of the lining, painless to remove, that a pathologist examines under a microscope.

The doctor looks for the pattern of inflammation. Continuous inflammation starting at the rectum points to ulcerative colitis. Patchy inflammation with skip areas, especially at the end of the small intestine, points to Crohn's. Because Crohn's can affect parts of the small intestine a colonoscope can't reach, doctors may add imaging such as MRI, or a capsule endoscopy, where the patient swallows a pill-sized camera (NIDDK). For children and teens, the revised Porto criteria recommend both an upper endoscopy (a camera passed through the mouth to view the esophagus, stomach, and first part of the small intestine) and a colonoscopy for everyone suspected of having IBD, plus imaging of the small intestine unless the picture is clearly typical UC.

Here is how the two main tests compare.

	Fecal calprotectin	Colonoscopy with biopsy
What it is	Stool sample sent to a lab	Camera exam of the colon and end of the small intestine
What it tells you	Whether there is likely inflammation	Where the inflammation is, how severe, what type
Invasive?	No	Yes (bowel prep, sedation)
Main use	Screening and repeat monitoring	Diagnosis and checking for healing

Colonoscopy also has a long-term job. People who have had IBD in a large part of the colon for many years are more likely to develop colorectal cancer. NIDDK says people with long-standing ulcerative colitis that involves a third or more of the colon need closer screening, and that people with Crohn's disease in the large intestine may be advised to have screening colonoscopies to look for cancer or precancerous cells (called dysplasia). For Crohn's, NIDDK adds that the risk is higher the longer someone has had the disease, and in people who

also have **PSC** or a family history of colorectal cancer. These screening checks are a routine part of long-term IBD care.

9.8 The Treatment Toolbox

Treatment for IBD has two jobs: **induction** (calming an active flare) and **maintenance** (keeping remission going). NIDDK says medicines do not cure Crohn's disease, but they can reduce inflammation and bring on and maintain remission. Doctors choose based on the type of IBD, where it is, how severe it is, and the person's age.

So many drug names



The drug names in this section are a mouthful, and you don't need to memorize them. Focus on the *job* each class does, and which part of the immune alarm system it turns down.

Here are the main classes, from oldest to newest.

Aminosalicylate. An **aminosalicylate** (also called a 5-ASA drug) is an anti-inflammatory medicine, such as mesalamine or sulfasalazine, that works on the lining of the gut and is used mainly for mild to moderate ulcerative colitis. NIDDK says doctors prescribe them to treat mild or moderate UC or to help people stay in remission. They are long-established older medicines. They are not a standard treatment for Crohn's disease.

Corticosteroid. A **corticosteroid** (often just called a steroid) is a powerful, fast-acting medicine related to the body's stress hormone cortisol that broadly calms the immune system. Examples include prednisone and budesonide; they are lab-made, much stronger relatives of the cortisol you met in Chapter 7. A 2021 *JAMA* review by [Cushing and Higgins](#) describes steroids giving rapid symptom relief while longer-term therapy gets started, which is why Riya got a course at diagnosis. NIDDK warns that steroids should be used only short term, because long-term use can cause serious side effects, including bone thinning. Steroids are a bridge, not a destination.

Immunomodulator. An **immunomodulator** is a medicine that dampens the immune system's activity more slowly and steadily, used mainly to maintain remission or to help other medicines work better. Examples include azathioprine, 6-mercaptopurine (together called thiopurines), and methotrexate. They are long-established and take weeks to months to work fully.

Biologic therapy. **Biologic therapy** is treatment with a medicine made by living cells, usually a lab-made antibody, that blocks one specific immune signal or cell pathway involved in inflammation. Instead of turning down the whole immune system, a biologic is like cutting one specific wire in the alarm system. Biologics are given by injection or by infusion into a vein; Riya takes one by injection. Different biologics block different targets:

- **Anti-TNF therapy** is a type of biologic treatment that blocks tumor necrosis factor (TNF), a cytokine that drives inflammation in IBD. Infliximab and adalimumab are examples. Anti-TNF medicines were the first biologics approved for IBD.
- **Anti-integrin therapy** (vedolizumab) blocks a molecule that immune cells use to enter the gut, so fewer inflammatory cells reach the gut wall.
- **Anti-interleukin therapies** block other cytokines called interleukins. Ustekinumab blocks interleukins 12 and 23; risankizumab, mirikizumab, and guselkumab block interleukin 23.

JAK inhibitor. A **JAK inhibitor** is a pill, a small molecule rather than an antibody, that blocks Janus kinase (JAK) enzymes inside immune cells, which relay signals from many cytokines at once. Tofacitinib and upadacitinib are examples. Another class of pills, called S1P receptor modulators (ozanimod and etrasimod), keeps certain immune cells trapped in lymph nodes so fewer reach the gut. The S1P modulators are approved for UC but not for Crohn's disease.

The table below shows when the US Food and Drug Administration (FDA) first approved some of these medicines specifically for ulcerative colitis or Crohn's disease in adults. Many were approved earlier for other diseases; the years here are for IBD.

Medicine (class)	First FDA approval for UC	First FDA approval for Crohn's
Infliximab (anti-TNF)	2005	1998
Adalimumab (anti-TNF)	2012	2007
Vedolizumab (anti-integrin)	2014	2014
Ustekinumab (anti-IL-12/23)	2019	2016
Tofacitinib (JAK inhibitor)	2018	Not approved for Crohn's
Ozanimod (S1P modulator)	2021	Not approved for Crohn's
Upadacitinib (JAK inhibitor)	2022	2023
Risankizumab (anti-IL-23)	2024	2022
Mirikizumab (anti-IL-23)	2023	2025
Guselkumab (anti-IL-23)	2024	2025

New medicines keep arriving, including for children. In April 2026, [the FDA approved ustekinumab](#) for moderately to severely active Crohn's disease in children aged 2 and older, and later in 2026 for ulcerative colitis in the same age group. Other drugs are still experimental. For example, an oral drug called obefazimod is being tested for UC, but as of October 2026 it is **not** FDA approved; its maker said in July 2026 that it planned to submit an application by the end of the year.

Food as treatment: exclusive enteral nutrition

For children with Crohn's disease, a liquid formula can be a real medical treatment. In **exclusive enteral nutrition** (EEN), a child drinks a nutritionally complete formula, and usually nothing else, for a set number of weeks under a care team's supervision. The [newest guideline on pediatric Crohn's disease](#) from two European expert groups (ECCO and ESPGHAN), published in 2026, names two preferred ways to induce remission in children with lower-risk Crohn's disease in the bowel: EEN, or the Crohn's Disease Exclusion Diet combined with partial enteral nutrition (a structured list of allowed foods plus formula for part of each day). For children at high risk of a complicated course, the same guideline recommends starting an anti-TNF biologic as the first treatment. Both nutrition therapies are supervised medical treatments with a defined plan, not lifestyle diets, and long-term care still aims for a healed lining and normal growth.

Hype Meter: "Diet alone can cure Crohn's disease"

Strong

Moderate

Preliminary

Hype

Verdict: Hype. No diet has been shown to cure Crohn's disease. The real finding is narrower: the 2026 ECCO-ESPGHAN guideline update prefers exclusive enteral nutrition (a supervised liquid-formula treatment), or a structured exclusion diet combined with partial formula, to induce remission in children with lower-risk Crohn's. Inducing remission is not curing, and a nutrition therapy planned and monitored by a care team is not a diet plan sold online. **What would change the grade:** Long-term randomized trials showing that a specific diet keeps people in lasting remission with a healed lining, without medicine, would make a narrower claim worth grading. "Cure" would require the disease to stay gone for good.

IBD surgery

IBD surgery is an operation to remove a severely diseased section of bowel or to treat a complication of IBD, such as a stricture, fistula, abscess, severe bleeding, or precancerous changes. The two types of IBD lead to different kinds of surgery:

- **In ulcerative colitis**, surgeons usually remove the colon and rectum. Because UC lives only there, this removes the disease from the gut. NIDDK describes two common options: an internal pouch made from the end of the small intestine and connected to the anus (so stool still leaves the usual way), or an ileostomy, an opening on the belly called a stoma where stool collects in a pouch. The Le Berre review says 10% to 20% of people with UC still need this surgery despite modern medicines.

- **In Crohn's disease**, surgeons remove only the damaged section and reconnect the healthy ends (a resection). NIDDK notes surgery won't cure Crohn's, because it can come back elsewhere. The Cushing and Higgins review says more than half of people with Crohn's eventually develop complications that need surgery.

Surgery is not a failure. For many people, it is the step that gives them their life back.

9.9 Treat to Target: Mucosal Healing

For a long time, the goal of IBD treatment was simple: make the symptoms go away. Doctors now aim higher.

Mucosal healing is the healing of the gut's inner lining (the mucosa), so that ulcers and visible inflammation are gone when doctors look with an endoscope. It is also called endoscopic healing. Why does it matter if you already feel fine? Because inflammation that keeps simmering under the surface can slowly damage the bowel, leading to strictures, fistulas, and surgery, especially in Crohn's.

An international group of IBD experts, the International Organization for the Study of IBD, published targets called **STRIDE-II** in 2021. Based on a review of 435 studies and voting by experts, STRIDE-II set short-term targets of symptom relief and normal blood and stool markers, such as calprotectin. Its long-term targets are clinical remission and endoscopic healing, plus the absence of disability, a restored quality of life, and normal growth in children. This approach is called "treat to target." If the stool test or colonoscopy shows inflammation is still there, the care team may adjust treatment even if the person feels well.

European pediatric guidelines make the same point for children. The 2020 ECCO-ESPGHAN guideline said repeated calprotectin tests or bowel imaging are more reliable ways to track treatment response than symptom scores alone, and its 2026 update lists endoscopic remission, normal growth, and a good quality of life as long-term goals.

9.10 IBD and the Brain

Now we reach the gut-brain part of the story. People with IBD often describe low energy, worry, or sadness that comes with a flare. Is that the stress of being sick, or does gut inflammation change the brain? Probably both, with arrows in both directions.

How common are anxiety and depression in IBD?

In 2021, **Barberio and colleagues** combined data from 77 studies of 30,118 adults with IBD. They found that about 32.1% had symptoms of anxiety and about 25.2% had symptoms of depression, roughly 1 in 3 and 1 in 4. During active disease, the numbers were higher: anxiety symptoms in 57.6% (compared with 38.1% in inactive disease) and depression symptoms in 38.9% (compared with 24.2%). These were scores on screening questionnaires, not clinical diagnoses.

How to read a gut-brain claim: the 2021 IBD anxiety and depression meta-analysis

Question	This study
Mouse or human?	Human. Adults with IBD (studies had to include at least 100 adult patients).
Correlation or cause?	Correlation. It pooled observational studies that measured symptoms at one time. It can't show whether IBD causes anxiety, anxiety worsens IBD, or both.
How many?	Very large: 77 studies with 30,118 patients in total. But the studies differed a lot from each other, so the pooled numbers are averages across very different groups.
How long?	Mostly snapshots. Each study measured symptoms at one point, so it can't show how mood changes over time in one person.
Who paid for it?	The paper states that the study received no specific funding.
Is something being sold?	No. The authors' message was that gut doctors should screen for and treat anxiety and depression.

The conclusion: anxiety and depression symptoms are common in IBD, especially during flares. What the study can't tell us is why. The jump in depression symptoms from inactive to active disease fits the idea that inflammation affects mood, but it also fits the idea that feeling sick and missing school lower mood. Both are likely true at once.

Possible pathways from gut to brain

Here are the main routes scientists are studying, building on earlier chapters:

1. **Cytokines.** Inflammatory cytokines can signal the brain and trigger "sickness behavior" such as tiredness, poor sleep, and low mood (Chapter 4).
2. **The vagus nerve.** Vee's home nerve carries sensory information from the gut to the brainstem (Chapter 2). It also carries signals back down. A 2016 paper by [Bruno Bonaz and colleagues](#) describes the vagus nerve as having anti-inflammatory effects, both through the HPA axis and through a pathway that calms immune cells in the gut.
3. **Pain and sensitization.** Repeated pain during flares can make the nervous system more sensitive (Chapter 8).

4. **Life stress.** Living with an unpredictable illness is stressful, and stress changes how the gut feels and functions (Chapter 7). NIDDK notes that stress may change the gut microbiome in ways that can worsen Crohn's symptoms.

Because the vagus nerve can calm inflammation, some researchers have asked whether stimulating it with an implanted device could treat Crohn's. This idea gets shared online more confidently than the evidence allows.

Hype Meter: "Vagus nerve stimulation can treat Crohn's disease"

Strong

Moderate

Preliminary

Hype

Verdict: Preliminary. Bonaz and colleagues had shown that vagus nerve stimulation improved colitis in rats. In a 2016 pilot study, they implanted stimulators in 7 people with active Crohn's disease and followed them for 6 months. Two were removed from the study at 3 months because they got worse; five reached remission. With only 7 people and no comparison group, the study shows the approach is feasible, not that it works. Implanted vagus nerve stimulators are FDA approved for some other conditions, but **not** for Crohn's disease. **What would change the grade:** Larger randomized trials comparing real stimulation with sham (fake) stimulation in people with Crohn's, measuring both symptoms and healing of the gut lining.

Care for the whole person

Because mood and IBD are so tangled, the Barberio authors recommended that gut doctors screen for and treat anxiety and depression, and the Dolinger review includes a psychosocial assessment (a check on mood, stress, and daily life) in a complete evaluation. Treating anxiety or depression won't replace IBD medicine, but it can make living with IBD more manageable. Chapter 11 goes deeper.

If you're going through this

If you have IBD and feel anxious before flares or low during them, you're not weak and you're not imagining it. Mood changes are common in IBD, and they're worth bringing up with your care team just like a new symptom. Many teens find it helps to have a school plan with restroom access, arranged through the nurse or counselor, and one friend who knows. You can also ask your doctor about talking with a psychologist who works with people with IBD.

For families

Parents often wonder whether something they did, fed, or allowed caused their child's IBD. It didn't: IBD comes from a mix of genes, the immune system, gut microbes, and environment, and no family causes it. Helpful things to say sound like "That sounds really hard" or "What would help today?" Less helpful are "Are you sure you need the bathroom again?" or "Have you tried cutting out sugar?" If your child's medicine is hard to afford or keep up with, tell the care team, because there are often ways to help, and missed doses can lead to flares.

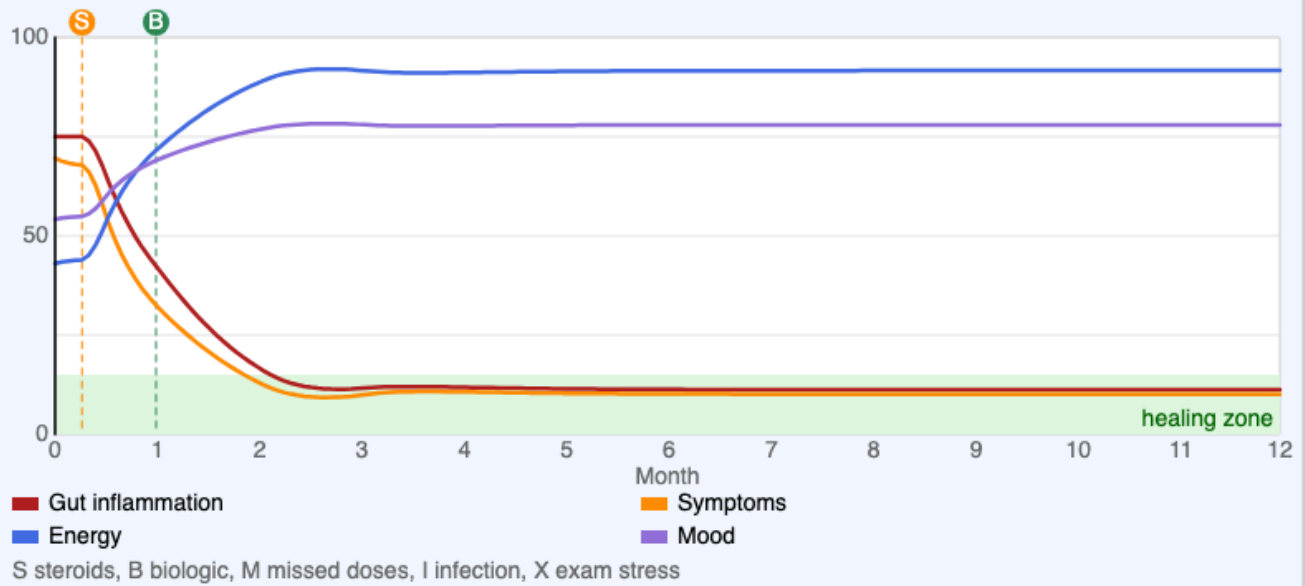
9.11 Seeing the Year at Once: The IBD Flare Timeline

You now know the pieces: inflammation, symptoms, flares and remission, medicines that work on different time scales, and the gut-brain links to energy and mood. The MicroSim below puts them together on one chart. Each curve runs from 0 to 100 in made-up units. "Inflammation" stands for the immune attack in the gut wall, "symptoms" for pain, diarrhea, and urgency, and the green "healing zone" for inflammation low enough that the lining can heal.

Diagram: IBD Flare Timeline

IBD Flare Timeline

Add events, then press Start. Illustrative model, not a prediction.



Month 0.0: inflammation 75, symptoms 70, energy 43, mood 54
Paused at month 0. Press Start to move through the year, or add an event.

Start

Reset

Clear events

Event:

Missed biologic doses

Add event

Month: 6



[Back to IBD Flare Timeline Lesson Plan](#)

Interactive simulation: <https://aaryavvatts-lab.github.io/gut-brain-textbook/sims/ch09-ibd-flare-timeline/main.html>

Open the IBD Flare Timeline fullscreen



MicroSim specification: IBD Flare Timeline



Type: microsim **sim-id:** ch09-ibd-flare-timeline

Library: p5.js

Status: Implemented

Bloom Level: Analyze

Bloom Verb: relate, analyze

Learning Objective: The learner will relate gut inflammation, symptoms, energy, and mood over 12 months in Crohn's disease and analyze how treatment events (steroids, a biologic, missed doses) and other events (an infection, a stressful exam week) change each curve on a different time scale.

Canvas: 510 px drawing area plus 130 px controls (iframe 642 px); responsive to about 380 px wide.

Controls: Start/Pause (moves a playhead through the year), Reset, Clear events; Event select (Start steroids, Start biologic, Missed biologic doses, Gut infection, Stressful exam week) with an Add event button; Month slider (0 to 12, step 0.5).

Rules: Starts with active disease. Steroids act within about a week and taper off by 12 weeks, lowering symptoms faster than inflammation. A biologic builds up over weeks and holds inflammation low; missed doses (45 days) let its effect fade, and inflammation returns over weeks. An infection adds a short spike. Exam stress raises symptoms and lowers mood for about two weeks but does not change inflammation.

Feedback: A readout gives the month and all four values at the playhead, plus a note explaining the most recent event.

Provenance: Steroids as a short-term bridge and biologics for maintenance (NIDDK; Cushing and Higgins 2021); mood worse in active disease (Barberio 2021); stress and flares associated but cause unclear (Singh 2009). All numbers are illustrative.

Start with the default plan (steroids, then a biologic at month 1) and press Start. In the first two months, the orange symptom curve drops faster than the red inflammation curve. That gap is why someone can feel better before their gut has healed, and why doctors check calprotectin or look with a scope instead of relying on symptoms alone.

Now choose **Missed biologic doses**, set the month to 6, and add it. Inflammation doesn't jump the next day; it creeps back over weeks, and symptoms, energy, and mood follow. Then add a **Stressful exam week** at month 10. Symptoms and mood change, but the red inflammation line doesn't move. That was a deliberate choice: the evidence links stress with how people feel, but it doesn't show that stress drives gut inflammation. Finally, press **Clear events** to see a year with no treatment.

What this model leaves out

- Real IBD is far less predictable. Flares often happen with no clear trigger, and two people on the same medicine can respond very differently.
- The model has one inflammation number, but real inflammation differs by location, depth, and type (UC versus Crohn's), and complications like strictures don't heal the same way.
- Medicine timing is simplified. Real drugs differ in how fast they work, some stop working over time, and side effects aren't shown.
- Mood depends on far more than gut symptoms: sleep, friends, school, family, and mental health care all matter, and mood can affect symptoms too.
- All numbers are made up for teaching. This is not a prediction for any real person.

At the Bedside: Riya's Story, From Diagnosis to Remission



Meet the patient (fictional)

Riya is 14 and in the spring of eighth grade. For about six months she has had cramping pain in her lower right belly, diarrhea several times a day, and a tiredness that sleep doesn't fix. She has lost weight without trying and has stopped finishing meals. Her parents notice she has quit practicing violin and goes to bed early.

What the doctor checks: Dr. Alvarez asks how long the symptoms have lasted, about blood in the stool, nighttime symptoms, joint pain, rashes, and family history, and checks Riya's growth chart. Blood tests show mild anemia and signs of inflammation. A stool test rules out infection and shows a high fecal calprotectin. Next come an upper endoscopy and a colonoscopy, both with biopsies, and an MRI of the small intestine.

Connecting the signals: Months of diarrhea, weight loss, and fatigue with a high calprotectin point to real inflammation, not a disorder of gut-brain interaction like IBS. The colonoscopy shows patchy ulcers with healthy skip areas in the end of the small intestine and part of the colon, the biopsies show inflammation typical of Crohn's, and the MRI shows thickening through the bowel wall, a sign of transmural inflammation.

Diagnosis: Crohn's disease affecting the end of the small intestine and part of the colon (pediatric IBD).

Why it happens: Riya likely inherited some genetic tendency toward IBD, and her gut immune system began overreacting to her gut microbes and lining instead of settling down. Nothing she ate or felt caused it.

The mind-body side: Riya's mood had dropped well before diagnosis, partly from feeling sick and embarrassed and perhaps partly from inflammatory signals reaching her brain (Section 9.10). She also began worrying that a flare might start during an orchestra concert. Dr. Alvarez's team screens her for anxiety and depression and connects her with the clinic psychologist, who teaches coping skills for pre-flare worry. The school nurse sets up a plan for restroom access and missed work.

Treatment today: Because Riya's symptoms were moderate and she was losing weight, Dr. Alvarez talked with the family about two ways to calm the flare quickly: exclusive enteral nutrition, which European pediatric guidelines prefer for children with lower-risk disease, or a short course of steroids. They chose steroids, with a careful taper, and Dr. Alvarez planned a biologic medicine by injection for long-term maintenance. (Care teams weigh these choices for each child; neither is right for everyone.) Within weeks Riya felt better, and within a few months her calprotectin came down. A follow-up check showed her lining healing, which is the goal of "treat to target." Now 16, she is mostly in remission and keeps taking her biologic, with occasional flares that her care team manages. She is back to violin and cooking with her grandmother.

What's new in research: Pediatric care is moving toward advanced medicines earlier. A 2026 study of 787 US children with newly diagnosed Crohn's disease found that 75% of those diagnosed in 2019 to 2023 started an advanced therapy, such as a biologic, within a year (Gonzalez et al., *American Journal of Gastroenterology*, 2026). **Evidence grade: Moderate.** It is a large observational study of insurance records from commercially insured children. It shows a clear change in practice, but it couldn't confirm whether earlier treatment lowered the need for surgery, so randomized and longer studies are still needed.

 Your turn: reason through a variation

Suppose a 15-year-old named Kenji has had diarrhea with visible blood for two months, an urgent need to go, and cramping, but no weight loss. His calprotectin is high, and his colonoscopy shows continuous inflammation starting at the rectum and extending partway up the colon, limited to the inner lining, with no skip areas and a normal small intestine. Which type of IBD fits best, and how is his likely treatment different from Riya's?

Answer: The continuous pattern starting at the rectum, limited to the inner layers and to the colon, plus bloody diarrhea, fits ulcerative colitis rather than Crohn's disease. For mild to moderate UC, doctors often start with an aminosalicylate, which is not a standard treatment for Crohn's, and add steroids or advanced medicines if needed. If UC ever became severe and didn't respond to medicine, removing the colon and rectum would remove the disease from his gut, which isn't true for Crohn's.

This is a fictional teaching case. It is not medical advice. If you or someone you know has symptoms like these, talk to a doctor.

Common Misconceptions

Myth: IBD and IBS are the same thing with slightly different names.

Reality: IBD (Crohn's disease and ulcerative colitis) involves visible, measurable inflammation and damage in the gut wall, found on tests like fecal calprotectin and colonoscopy. IBS is a disorder of gut-brain interaction in which the gut looks normal on these tests. They are treated very differently (see Chapter 10).

Myth: IBD is caused by stress or a bad diet.

Reality: IBD develops from a combination of genes, an overactive immune response, gut microbes, and environmental factors such as smoking. Perceived stress is associated with flares in people who already have IBD, and some large studies link depression symptoms or eating patterns with a higher later risk, but these are

correlations. No good evidence shows that stress or diet by itself starts the disease, and nobody causes their own IBD.

Myth: IBD only affects the gut.

Reality: IBD can cause extraintestinal manifestations in the joints, skin, eyes, liver, blood, and bones, and can slow growth in children. Anxiety and depression symptoms are also common, especially during active disease.

Myth: Once you are in remission, you can stop your treatment.

Reality: Maintenance medicine is usually what keeps remission going, and NIDDK says people with ulcerative colitis typically need lifelong treatment unless their colon and rectum are removed. Any change in treatment should be decided with a doctor.

Myth: If you feel fine, your gut must be healed.

Reality: Symptoms can improve before inflammation is gone. That's why care teams track calprotectin and check the lining directly, aiming for mucosal healing, not just feeling better.

Your Gut Check

Test yourself. Try each one before you open the answer.

? 1. Claim Check: "I healed my Crohn's in 30 days with this gut-reset cleanse. Doctors just want to keep you on drugs! Link in bio." ✓

Grade: Hype. No cleanse or diet has been shown to cure Crohn's disease, and one person's story can't separate the cleanse from a natural period of remission, which can happen on its own. The real evidence for nutrition is narrower: supervised exclusive enteral nutrition can induce remission in some children with Crohn's, and it is followed by maintenance medicine. The "link in bio" means something is being sold. A careful version would say: "Some nutrition therapies, prescribed by a care team, can help induce remission in certain children with Crohn's, but they are not a cure."

? 2. Predict: Riya goes to a month-long summer music camp and forgets to bring her biologic, so she misses two doses. She feels fine for the first couple of weeks. What would you predict, and why? ✓

Feeling fine at first fits how biologics work: their effect fades gradually. Over the following weeks, as the medicine level drops, the immune signal it blocked can return, so inflammation may creep up, followed by symptoms, tiredness, and lower mood, as in the IBD Flare Timeline. The right move is to contact Dr. Alvarez's team as soon as she realizes, rather than waiting for symptoms.

? 3. Explain: At a check-up, Riya says she feels great, but her fecal calprotectin is higher than last time. Explain how both can be true and why her doctor cares. ✓

Calprotectin comes from neutrophils that move into an inflamed gut wall, so a higher level suggests inflammation is increasing, even if it isn't yet causing symptoms. Symptoms often lag behind inflammation or don't match it closely. Her doctor cares because the treatment target is a healed lining, not just feeling well, and simmering inflammation in Crohn's can slowly lead to complications like strictures. The doctor might repeat the test, order imaging or a colonoscopy, or adjust treatment.

? 4. Apply: A 17-year-old with ulcerative colitis has had painful, swollen knees and red, sore eyes during his last two flares. His friend says, "That can't be related to your gut." Use this chapter to respond. ✓

Both can be extraintestinal manifestations of IBD. NIDDK lists certain types of arthritis and eye irritation among the effects of UC outside the gut. They make sense because IBD is an immune-mediated disease: inflammatory cells and cytokines travel in the blood, so inflammation that starts in the gut can affect other tissues. He should tell his care team, because these problems may need their own treatment and may also signal disease activity.

5. Read the data. Three US studies estimated how many people have IBD, using different methods:

Study	Method	Who was counted	Result
Lewis et al. 2023	Insurance claims (diagnosis and treatment records)	All ages	About 721 per 100,000 (about 0.7%), roughly 2.39 million people
Kappelman et al. 2025	Insurance claims	Under age 20	About 122 per 100,000, roughly 100,000 young people
NCHS 2026 (survey years 2023 to 2024)	National survey: "Has a doctor ever told you...?"	Adults 18 and older	1.6% of adults, about 4.1 million

? 5. What does this data show, and what can't it show? ✓

It shows that IBD is far from rare, with millions of Americans affected, including about 100,000 people under 20. It also shows that the method changes the answer: the adult survey (1.6%) is more than twice the claims estimate (about 0.7%), probably because a survey counts anyone ever told they had IBD, including mistaken diagnoses. These numbers can't show what causes IBD or whether it is increasing, since the methods and time periods differ. A helpful follow-up would track one population with the same method over many years.

Exercises

Remember

1. Name the two main types of inflammatory bowel disease and state where in the digestive tract each one can occur.



Answer



Ulcerative colitis affects only the colon and rectum. Crohn's disease can affect any part of the digestive tract, from the mouth to the anus, most often the end of the small intestine and the start of the colon.

1. What does a high fecal calprotectin result suggest, and which cells release calprotectin?



Answer



It suggests inflammation in the intestines. Calprotectin comes mainly from neutrophils, a type of white blood cell that moves into inflamed gut tissue.

Understand

1. Explain why transmural inflammation in Crohn's disease can lead to strictures and fistulas, while ulcerative colitis usually doesn't.



Answer



Transmural inflammation goes through the whole thickness of the gut wall. Deep inflammation can scar and narrow the bowel (strictures) or eat through the wall and form tunnels to other organs or the skin (fistulas). Ulcerative colitis usually stays in the inner layers, so it rarely causes these deep complications.

1. Explain the difference between how a corticosteroid and a biologic therapy calm inflammation, and why steroids are used only short term.



Answer



A corticosteroid broadly turns down much of the immune system and works quickly, so it calms a flare fast. A biologic blocks one specific immune signal, such as TNF, and is used for long-term maintenance. Steroids are used only short term because long-term use can cause serious side effects, such as bone thinning, and they aren't meant to maintain remission.

Apply

1. Use the IBD Flare Timeline. Run the default plan and record the month when symptoms first fall below 25 and the month when inflammation first enters the healing zone. Then explain what that gap means for how doctors decide whether treatment is working.
2. A friend with Crohn's disease tells you she's thinking of skipping her next injection because she has felt great for six months. Using what you learned about remission, maintenance, and mucosal healing, write two or three supportive sentences you could say that encourage her to talk with her care team, without giving medical advice yourself.

Analyze

1. Compare the claims-data estimate (about 0.7% of Americans) with the survey estimate (1.6% of adults) for IBD. Identify at least two reasons they differ, and explain which number is better for answering "How many Americans are living with diagnosed IBD?"
2. In the Barberio 2021 meta-analysis, depression symptoms were more common in active IBD (38.9%) than in inactive IBD (24.2%). Analyze at least three different explanations for this difference, including at least one gut-to-brain pathway and one that does not involve inflammation acting on the brain.

Evaluate

1. A news headline reads: "Stress triggers Crohn's flares, study finds." Using the Singh 2009 review and the Hype Meter, evaluate whether the headline is fair. Write a more accurate headline of 15 words or fewer.
2. Some experts argue that children with Crohn's should start a biologic soon after diagnosis; others prefer to start with milder treatments and move up only if needed. Using evidence from this chapter (pediatric growth, the Gonzalez 2026 study, the 2026 ECCO-ESPGHAN guideline update, and treat-to-target goals), evaluate the strengths and weaknesses of each approach. What evidence would settle the question?

Create

1. Design a one-page "IBD at school" guide for teachers that explains, in plain language, what IBD is, what a flare looks like, and three specific accommodations that could help a student like Riya. Make sure it doesn't blame the student or suggest diet or stress caused the disease.
2. Design a study to test whether a 12-week anxiety-coping program improves quality of life in teens with Crohn's disease. Describe the groups, how you would assign teens to them, what you would measure (include at least one measure of inflammation and one of mood), and how long you would follow them.

Key Takeaways

- Inflammatory bowel disease is an immune-mediated disease: the body's own immune response causes long-lasting, recurring gut inflammation. It has no cure, but treatment can keep people in remission for long periods.
- Ulcerative colitis affects only the colon and rectum, continuously and in the inner layers. Crohn's disease can affect any part of the gut, in patches, with transmural inflammation that can cause strictures, fistulas, and abscesses.
- About 2.4 million Americans, including about 100,000 people under 20, have diagnosed IBD (claims data); surveys give higher numbers because they measure something different. Risk comes from genes, the immune system, gut microbes, and environment, such as smoking. Stress and diet have not been shown to cause it.
- Fecal calprotectin acts like a smoke detector for gut inflammation, and colonoscopy with biopsy shows where the inflammation is and what type it is.
- Treatment classes include aminosalicylates, corticosteroids (a short-term bridge), immunomodulators, biologic therapies such as anti-TNF medicines, and JAK inhibitors, plus exclusive enteral nutrition in children and IBD surgery when needed. Maintenance treatment usually continues during remission.
- The modern goal is mucosal healing, a healed gut lining, not just fewer symptoms, because symptoms can improve before inflammation is gone.
- IBD affects the whole person: extraintestinal manifestations are common, and so are anxiety and depression symptoms, especially during flares.

You followed Riya from diagnosis to remission!



You can now explain what's happening inside a gut with IBD, how doctors find it and treat it, and why mood belongs in the conversation. Next, in Chapter 10, we meet Sam, whose tests all came back normal, and discover why "normal tests" doesn't mean "nothing is wrong."

Quiz: Inflammatory Bowel Disease: Ulcerative Colitis and Crohn's

Test your understanding of Chapter 9. Choose the best answer, then click **Show Answer** to check.

Question 1

Which statement correctly describes where ulcerative colitis occurs?

- A. Anywhere from the mouth to the anus, in patches
- B. Only in the colon and rectum, continuously from the rectum upward
- C. Only in the small intestine, through the whole thickness of the wall
- D. Only in the stomach and the first part of the small intestine

 **Show Answer**



The correct answer is **B**. Ulcerative colitis affects only the colon and rectum, starting at the rectum and spreading upward in one continuous stretch, in the inner layers of the wall. Option A describes Crohn's disease, which is the most common mix-up.

Concept Tested: Ulcerative Colitis **Bloom's Level:** Remember

Question 2

What does "transmural inflammation" mean?

- A. Inflammation that moves from one person to another
- B. Inflammation that affects only the inner lining of the colon
- C. Inflammation that appears outside the gut, such as in the joints
- D. Inflammation that extends through the entire thickness of the gut wall

 Show Answer



The correct answer is **D**. "Trans" means across and "mural" means wall, so transmural inflammation goes through every layer of the gut wall. It is typical of Crohn's disease. Option C describes an extraintestinal manifestation, a different idea.

Concept Tested: Transmural Inflammation **Bloom's Level:** Remember

Question 3

Fecal calprotectin is a useful test because it:

- A. Comes mainly from neutrophils that move into inflamed gut tissue, so high stool levels signal intestinal inflammation
- B. Shows exactly which part of the intestine is inflamed
- C. Measures how many bacteria live in the colon
- D. Confirms a diagnosis of Crohn's disease without any other tests

 Show Answer



The correct answer is **A**. Calprotectin is released mainly by neutrophils, and its level in stool rises with intestinal inflammation. Like a smoke detector, it signals that inflammation is probably present but can't show where it is or what type it is (options B and D), which is why colonoscopy with biopsy is still needed.

Concept Tested: Fecal Calprotectin **Bloom's Level:** Remember

Question 4

Why do doctors use corticosteroids only as a short-term treatment in IBD?

- A. They take many months to start working
- B. They work only in ulcerative colitis, not Crohn's disease
- C. They act fast to calm inflammation, but long-term use can cause serious side effects such as bone thinning
- D. They cure IBD after one course, so longer use is unnecessary

 Show Answer



The correct answer is **C**. Steroids broadly calm the immune system and relieve symptoms quickly, which makes them a good bridge while longer-term medicine starts working. Long-term use can cause serious side effects, so they are not used to maintain remission. Option D is wrong because no medicine cures IBD.

Concept Tested: Corticosteroid **Bloom's Level:** Understand

Question 5

How does a biologic therapy such as an anti-TNF medicine differ from a corticosteroid?

- A. A biologic blocks one specific immune signal, while a steroid broadly turns down much of the immune system
- B. A biologic is a pill, while a steroid must be injected
- C. A biologic is used only during flares, while a steroid is used for long-term maintenance
- D. A biologic kills gut bacteria, while a steroid repairs the gut lining

 Show Answer



The correct answer is **A**. An anti-TNF biologic is a lab-made antibody that blocks one cytokine, tumor necrosis factor, while steroids act broadly. Option C reverses their usual roles: steroids are the short-term bridge, and biologics are commonly used for maintenance.

Concept Tested: Biologic Therapy **Bloom's Level:** Understand

Question 6

Why is mucosal healing, not just symptom relief, a long-term treatment target in IBD?

- A. Because symptoms in IBD are imaginary and can't be measured
- B. Because mucosal healing means the disease is cured
- C. Because a healed lining means medicine can always be stopped
- D. Because symptoms can improve while inflammation keeps simmering and slowly damaging the bowel

 **Show Answer**



The correct answer is **D**. Symptoms and inflammation don't always match, so a person can feel well while inflammation continues, which in Crohn's can lead to strictures and fistulas. STRIDE-II therefore set endoscopic healing as a long-term target. Option B is wrong because healing is a sign of good control, not a cure.

Concept Tested: Mucosal Healing **Bloom's Level:** Understand

Question 7

A 16-year-old has had bloody diarrhea and urgency for two months. Colonoscopy shows continuous inflammation from the rectum partway up the colon, limited to the inner lining, and a normal small intestine. Which diagnosis fits best?

- A. Crohn's disease
- B. Ulcerative colitis
- C. Irritable bowel syndrome
- D. Hirschsprung disease

 **Show Answer**



The correct answer is **B**. Continuous inflammation starting at the rectum, limited to the colon and the inner layers, with bloody diarrhea, is the pattern of ulcerative colitis. Crohn's (option A) would usually show patchy, transmural inflammation, often in the end of the small intestine. IBS (option C) would not show visible inflammation.

Concept Tested: Ulcerative Colitis **Bloom's Level:** Apply

Question 8

Riya has felt well for months on her biologic. A classmate says, "You're in remission, so you can stop your shots now." Based on the chapter, what is the best response?

- A. "You're right, remission means the disease is gone."
- B. "I'll stop for a month and see what happens."
- C. "Maintenance medicine is usually what keeps remission going, so any change is a decision for my care team."
- D. "Remission only happens in ulcerative colitis, so it doesn't apply to me."

 Show Answer



The correct answer is **C**. Remission is a quiet period, not a cure, and maintenance medicine usually keeps it going. Stopping on your own (option B) can let inflammation return weeks later, as the IBD Flare Timeline shows. Remission happens in both Crohn's and UC, so option D is false.

Concept Tested: Remission **Bloom's Level:** Apply

Question 9

In a 2021 meta-analysis of 30,118 adults with IBD, depression symptoms were found in 38.9% of people with active disease and 24.2% of people with inactive disease. Which conclusion is best supported?

- A. Depression symptoms are more common during active IBD, but this study can't show which causes which
- B. Gut inflammation has been proven to cause depression
- C. Depression causes IBD flares
- D. Treating depression cures IBD

 Show Answer



The correct answer is **A**. The studies were observational snapshots using symptom questionnaires, so they show an association. Inflammation might affect mood, feeling sick might lower mood, or mood might affect symptoms. Options B and C each claim one direction of cause that this design can't establish.

Concept Tested: Disease Flare **Bloom's Level:** Analyze

Question 10

A survey found that 1.6% of US adults say a doctor told them they had IBD, while insurance claims data put IBD at about 0.7% of Americans. What best explains the difference?

- A. IBD became twice as common in the time between the two studies
- B. One of the studies must have made an arithmetic mistake
- C. Claims data count only children, so they miss most adults
- D. The survey counts anyone ever told they had IBD, which can include old or mistaken diagnoses, while claims require actual diagnosis and treatment records



Show Answer



The correct answer is **D**. The two methods measure different things: self-report of ever being told versus records of diagnosis and treatment. Option A is tempting, but different methods can't show a trend, and the claims estimate covers all ages, so option C is wrong too.

Concept Tested: Inflammatory Bowel Disease **Bloom's Level:** Analyze
