NORTHEAST
BUSINESS GROUP ON HEALTH

HP2030 HEALTHY PEOPLE 2030 CHAMPION

MONDAYS WITH DR MARK & DR MICHAEL

Monday, December 15, 2025 | 1:00 – 2:00PM

TOPIC #58

Invisible but Impactful: The Employer Imperative in Crohn's & Colitis Care

Featured Speakers:

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MONDAYS WITH DR MARK & DR MICHAEL

NORTHEAST BUSINESS GROUP ON HEALTH

Jefferson
Philadelphia University + Thomas Jefferson University

Inflammatory Bowel Disease

December 2025

Northeast Business Group on Health

Priya Sehgal MD MS MPH

Cuckoo Choudhary MD

IBD: An Overview

- Excited to Share an overview of IBD, a complex group of digestive diseases

- I will walk you through the major forms of IBD: Ulcerative colitis, Crohn's disease, and Microscopic colitis, and touch on what happens inside the body at a biological level, as well as the roles that genetics and family history play in these diseases

- **Prevalence and Impact:**
  IBD affects millions globally—approximately 1.6 million people in the U.S.
- 15 – 35 yrs.
- Not only a medical issue, but a social and emotional one, with significant impacts on quality of life

What is Inflammatory Bowel Disease (IBD)?

IBD = Chronic inflammation of the digestive tract, caused by an overactive immune system.

This inflammation can lead to a variety of symptoms, including diarrhea, abdominal pain, and fatigue. Can cause chronic damage

Two main types:

- **Crohn's Disease** – can affect *any* part of the GI tract
- **Ulcerative Colitis** – affects *only the colon*
- IBD is **not caused by stress**, **not contagious**, and **not a food allergy**.
- And then there is a third kind: Microscopic colitis

- Not to be confused with irritable Bowel Syndrome (IBS) which may have some similar symptoms but is different.

Why does Inflammatory Bowel Disease (IBD) happen?

IBD develops when four factors collide:

- Genetics
- Immune System Overactivation
- Microbiome Imbalance
- Environmental Triggers

Genetic predisposition

- IBD is not a single-gene disease. Over 250 genes have been linked to increasing the risk of developing IBD.
  - NOD2 – influences how the body recognizes bacteria in the gut (strongly associated with Crohn's disease)
  - IL23R – regulates inflammation pathways
  - ATG16L1 – affects autophagy, the "clean-up" system of immune cells
- Genes involved in barrier function, innate immunity, adaptive immunity, and microbial recognition
- These genes do not guarantee disease; they simply make the intestinal immune system more *sensitive*, more *reactive*, or less able to regulate inflammation.

Family History risk of IBD

- If one parent has IBD → child's lifetime risk ≈ 5–10%
- If both parents have IBD → risk rises to 25–35%
- If a sibling has IBD → risk ≈ 7–10%
- Identical twins:
  - Crohn's concordance up to ~50%
  - Ulcerative colitis ~10–20%
- This shows genetics matter, but environmental factors matter just as much or more.

Pathophysiology of IBD

The Role of the Immune System:

- Normally, our immune system protects us from harmful invaders like bacteria and viruses. But in IBD, the immune system mistakenly attacks the body's own tissues, causing inflammation in the gut. This abnormal immune response leads to damage in the digestive tract.

Mucosal Immunity and Chronic Inflammation:

- **Chronicity and Damage:**
  In all forms of IBD, the chronic inflammation can lead to long-term damage to the gut, including scarring, narrowing (or strictures), and sometimes the formation of fistulas, which are abnormal connections between parts of the intestines or other organs.

Other Key Players in the causation of IBD

- **The Microbiome: A Key Player**
- The gut has trillions of microbes that normally support health.

In IBD:

- Microbial diversity is reduced
- Protective bacteria are low
- Inflammatory species become high
- This imbalance (dysbiosis) amplifies immune activation and sustains inflammation.

Other Key Players in the causation of IBD

- Environmental and Lifestyle Triggers
- Even with genetic risk, people usually develop IBD only after an environmental trigger “tips the balance.”
- Key influences:
- ? Antibiotics in early life
- Smoking (major risk for Crohn’s; protective for UC)
- Diet high in emulsifiers, processed foods
- Stress, infections, and changes in gut permeability
- Urban living (increased incidence)
- NSAIDs causing mucosal injury
- IBD is highest in the U.S., Canada, UK, Northern Europe, and rising rapidly in India, China, Middle East—suggesting environmental change is a huge driver.

Ulcerative Colitis

Ulcerative Colitis primarily affects the colon and rectum, causing inflammation and ulceration a continuous pattern along the inner lining of the bowel. The inflammation typically starts at the rectum and may extend upward.

- **Symptoms**:
  bloody diarrhea, cramping, urgency, and fatigue. The severity can vary, with some patients experiencing frequent flare-ups and others achieving long periods of remission.

- **Treatment**:
  Treatment usually involves medications to control inflammation and suppress the immune system, such as corticosteroids, immunosuppressants, and biologic agents. In severe cases, surgery to remove the colon may be required.

Crohn’s Disease

Crohn’s Disease can affect any part of the gastrointestinal tract, from the mouth to the anus, and the inflammation tends to occur in patches. The disease can affect deeper layers of the bowel wall, leading to complications like strictures (narrowings in the intestines) and fistulas (abnormal connection from one part of the intestines to another or even from the intestines to the skin).

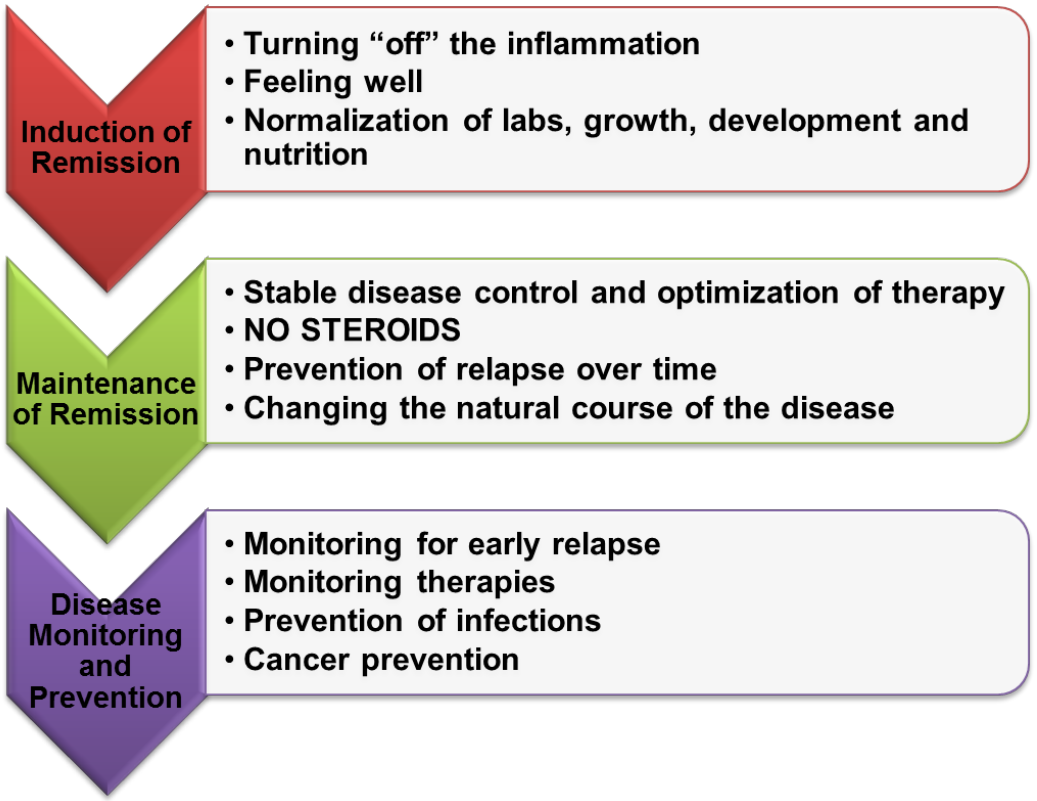
- **Symptoms**:
  Symptoms include chronic diarrhea (often without blood), abdominal pain, weight loss, and malnutrition. Unlike UC, which is confined to the colon, Crohn’s disease can lead to more varied and complex complications.

- **Treatment**:
  Like UC, treatment typically includes anti-inflammatory medications, immunosuppressants, and biologics. However, because of its ability to affect deeper layers of the intestine, Crohn’s often requires more aggressive management and, in some cases, surgery.

Microscopic Colitis

- Microscopic Colitis is characterized by chronic, non bloody, watery diarrhea and inflammation in the colon that is only visible under a microscope. Unlike UC and CD, MC doesn't cause major pain or cramping.
- **Diagnosis**: It is diagnosed through taking multiple biopsies of the colon, which almost always looks normal when the doctor does the colonoscopy.
- And that is why microscopic colitis can often be overlooked or misdiagnosed. The doctor has to be thinking about it in their differential to take biopsies of the normal/almost normal looking colon. If biopsies are not taken, it can be confused with IBS.
- Treatment includes anti-inflammatory drugs and sometimes immune-modulating therapies.

Goals of Treatment for IBD Patients



- Clinical remission (i.e. IBD symptoms have resolved)
- Mucosal improvement (i.e. overall decrease in mucosal inflammation)
- On a steroid-free treatment regimen

The medication that works best is the one your patient will take consistently.

Selecting Therapeutic Targets in Inflammatory Bowel Disease (STRIDE) Initiative of IO-IBD

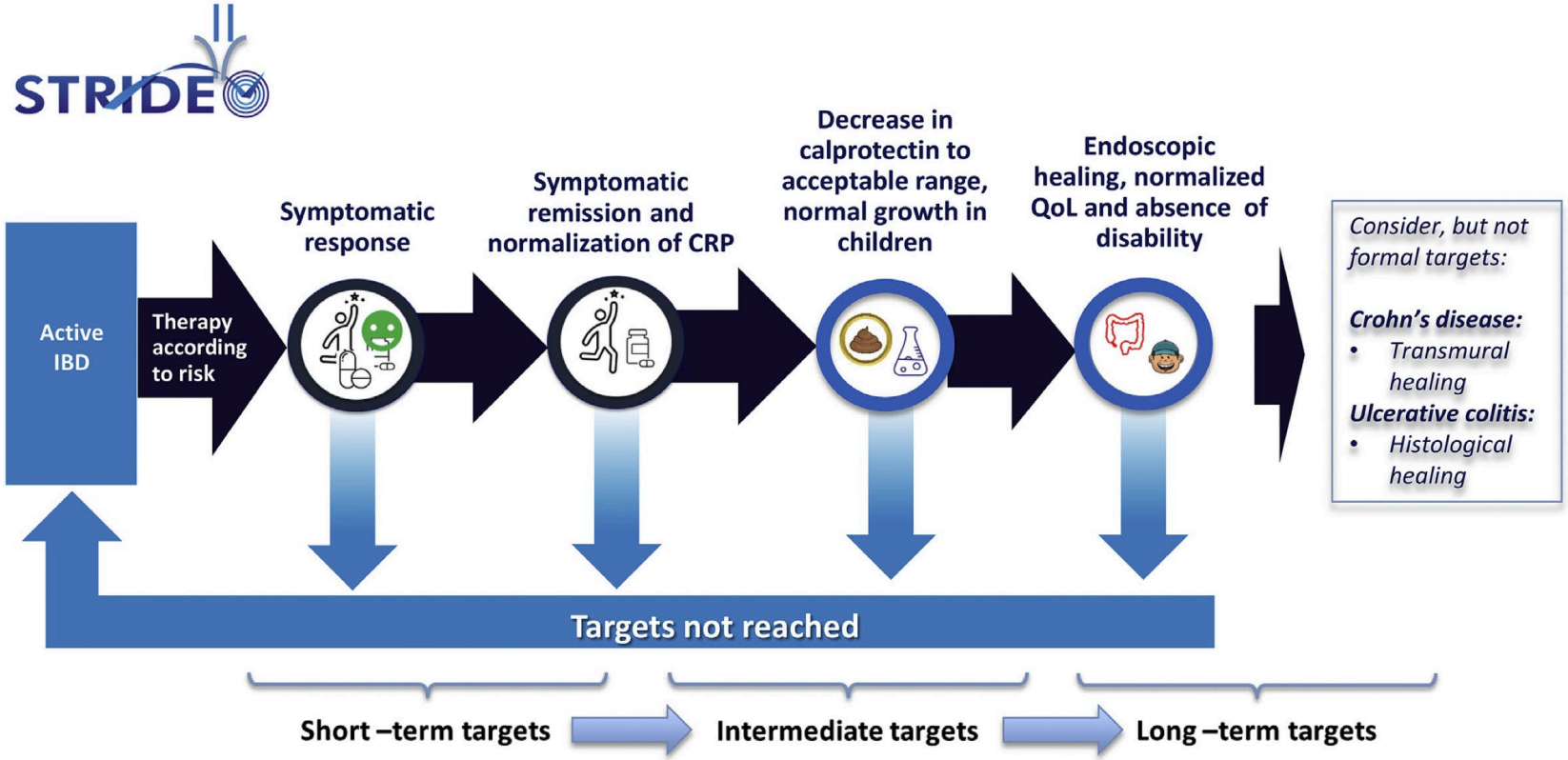


Figure 2. Treatment targets in CD and UC.

Dan Turner et al. STRIDE II consensus. Gastroenterology 2021;160:1570–1583

Risk Stratification Principles

We want to identify patients
before they have severe disease

We want to identify patients
at risk for severe disease

Dubinsky, M AIBD 2024

Risk Stratification of IBD patients

ULCERATIVE COLITIS

Risk factors for colectomy

- Under age 40
- Extensive colitis
- Steroid-requiring disease
- Deep ulcers
- History of hospitalization
- High CRP and ESR
- C. difficile infection
- Cytomegalovirus infection

CROHN'S DISEASE

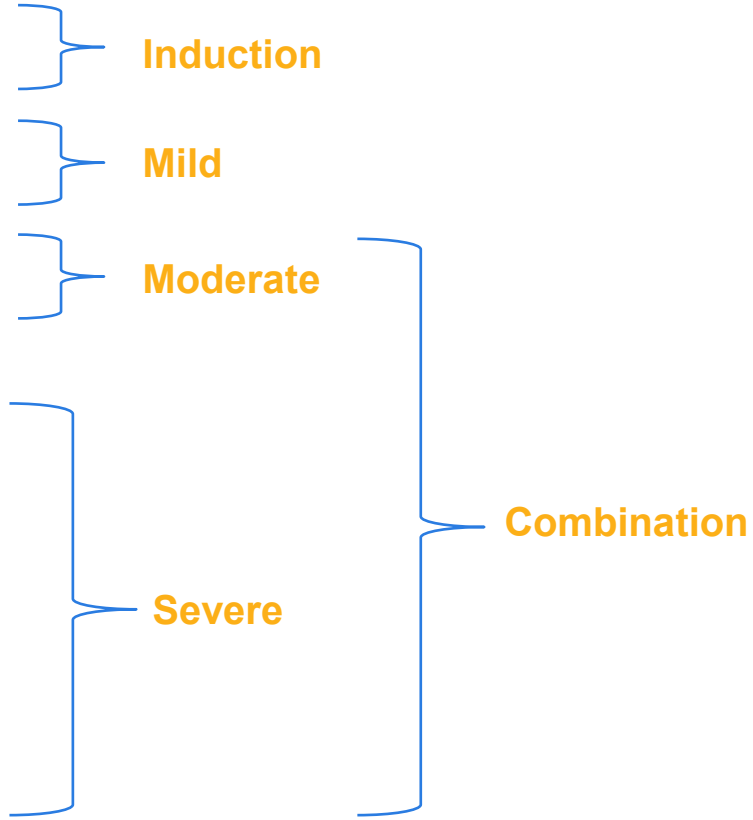
Risk factors for rapid disease progression

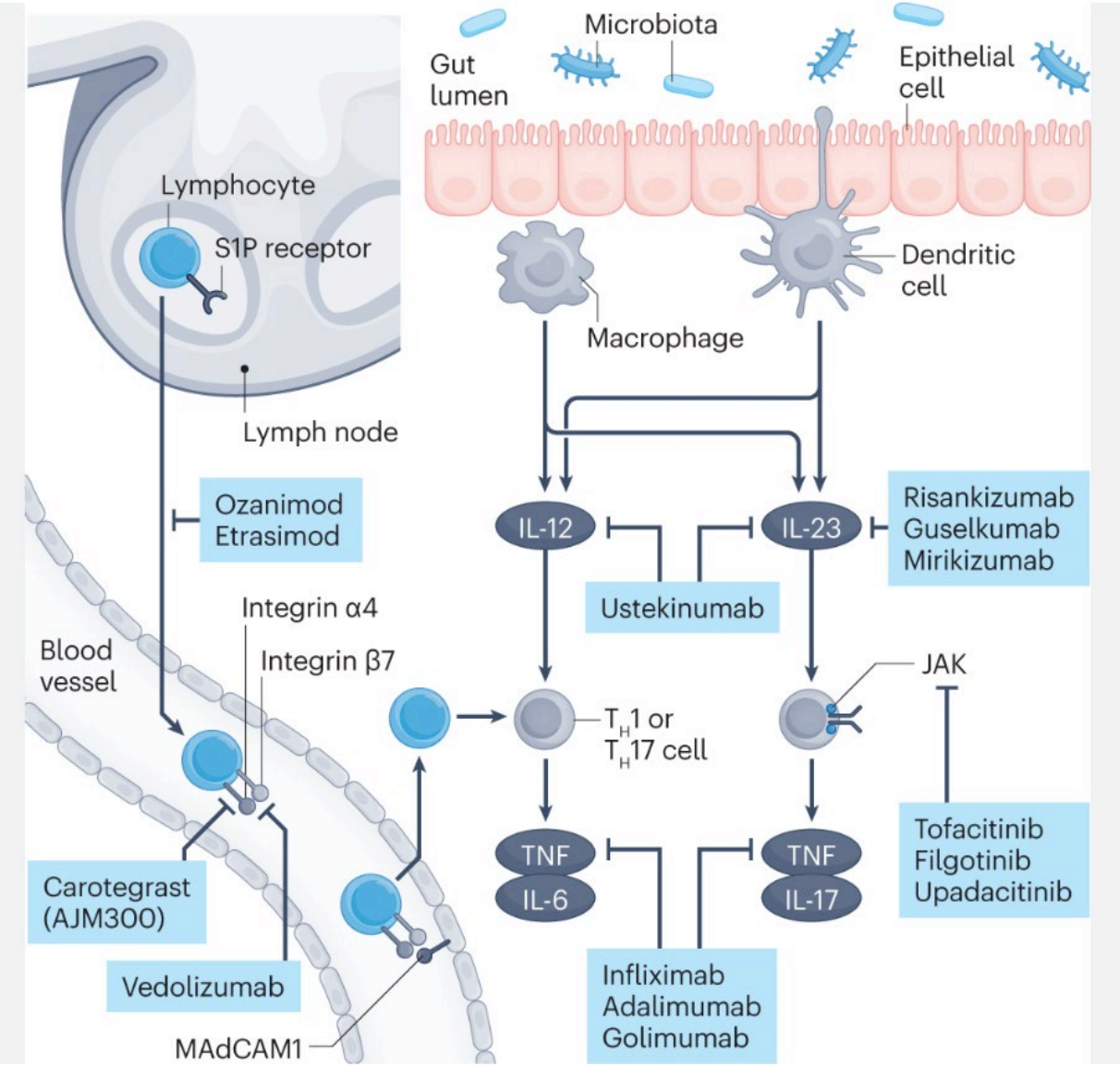
- Under age 30 at diagnosis
- Extensive anatomic involvement
- Perianal disease
- Severe rectal disease
- Deep ulcers
- Previous surgical resection
- Stricturing behavior
- Penetrating behavior

AGA CARE PATHWAY
https://ibd.care/provider/aga-care-pathways/stratify-according-to-risk

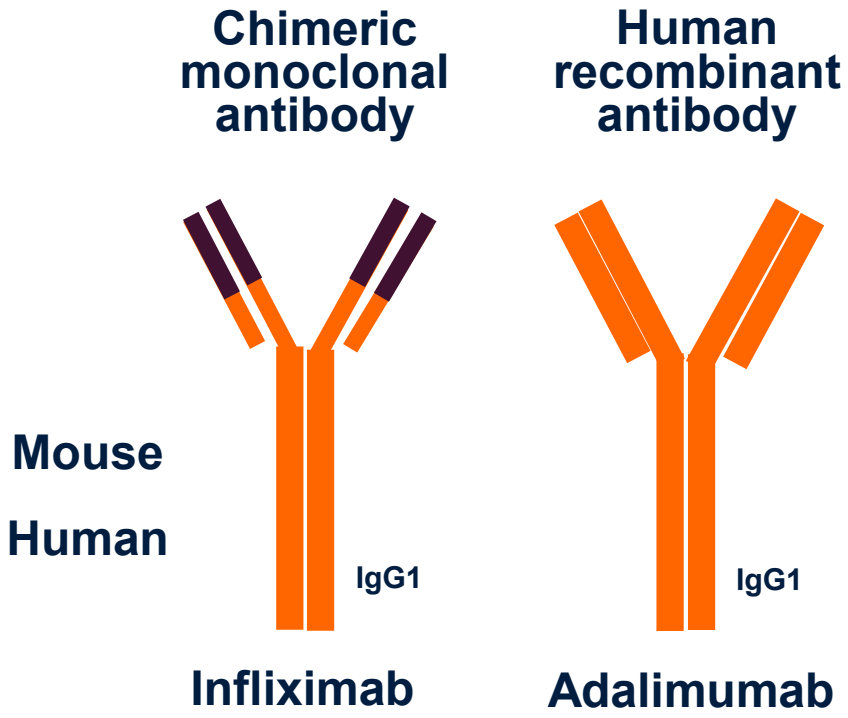
Treatment options

- **Anti-inflammatory:** Corticosteroids
- **Aminosalicylates (5ASA):** Mesalamine, Balsalazide, Sulfasalazine
- **Immunomodulators:** Azathioprine/6-mercaptopurine, Methotrexate
- **Biologic agents:**
- *Anti-tumor necrosis alpha (anti-TNF):* Infliximab, Adalimumab, Certolizumab, Golimumab
- *Anti-integrin (anti α4β7):* Vedolizumab
- *Anti interleukin (anti-IL 12/23):* UST, MIRI, GUS, RISA
- *Janus kinase (JAK) inhibitor:* Tofacitinib, Upadacitinib
- *Sphingosine-1-phosphate receptor agonist:* Ozanimod, Etrasimod
- **Miscellaneous:** Cyclosporine, Tacrolimus
- **Surgery** → Colectomy for Ulcerative colitis, resection for Crohn's disease




Gut lumen
Microbiota
Epithelial cell
Lymphocyte
S1P receptor
Lymph node
Dendritic cell
Macrophage
Ozanimod
Etrasimod
IL-12
IL-23
Risankizumab
Guselkumab
Mirikizumab
Ustekinumab
Integrin α4
Integrin β7
Blood vessel
JAK
T_H1 or T_H17 cell
TNF
IL-6
TNF
IL-17
Tofacitinib
Filgotinib
Upadacitinib
Carotegrast (AJM300)
Vedolizumab
Infliximab
Adalimumab
Golimumab
MAdCAM1

Anti-TNF alpha

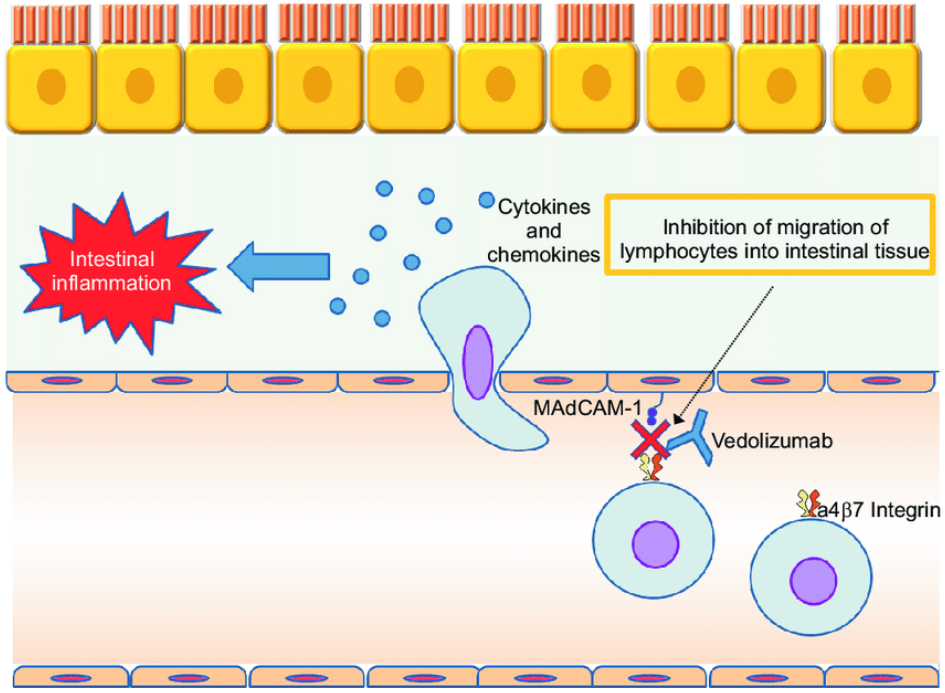


- Used for induction and maintenance for both CD and UC
  - Infliximab: 5-10 mg/kg IV at week 0, 2, 6 → q8 weeks IV
  - Adalimumab: 160 mg SQ week 0 → 80 mg SQ week 2 → 40 mg q2 SQ
  - Certolizumab: 400 mg weeks 0 and 2 → 400 mg q4 weeks *(only for CD)*
  - Golimumab: 200 mg SQ → 100 mg SQ → q4 weeks SQ *(only for UC)*

Special considerations:

- **Fistulizing disease in CD**
- **EIM: joint pain, uveitis**
- **Avoid in patients with melanoma, heart failure, active malignancy**

Anti-integrin: Vedolizumab



- Indication: Induction and maintenance of UC and CD
- Dosing: 300 mg IV weeks 0, 2, 6 then q8 weeks
- In UC: VARSITY trial (superiority to ADA)
- In CD: more effective for colonic crohn's (?)

Special considerations:

- **Favorable safety profile**
- **Good for elderly, history of malignancy**

Other biologic agents

- **Anti interleukin (anti-IL 12/23 and Il-23): Ustekinumab OR RISA, GUS, MIRI**
  - Lesser risk for infection and malignancy than anti-TNF agents
  - Loading dose infusion, maintenance sc
- **Janus kinase (JAK) inhibitor: Tofacitinib, Upadacitinib**
  - First class of oral biologic agents
  - TOFA approved only for UC, UPA approved for CD and UC
  - Small molecule, no immunogenicity
  - However strong immunosuppressant and high risk for infections, esp zoster, dyslipidemia, venous thromboembolism
- **Sphingosine-1-phosphate receptor agonist: Ozanimod, Etrasimod**
  - Approved only for UC
  - Oral small molecule, no immunogenicity
  - Many contraindications related to comorbidities, risk of infections, PML, macular edema
  - Dosing pack

Key Points

- IBD patients require support – life long chronic and debiliting illness, Crohn's and Colitis Foundation is a great resource
- The diagnosis of IBD is based on suggestive clinical symptoms, radiographic and/or endoscopic + pathologic findings
- The choice of treatment should be based on the disease activity (i.e. patient reported symptoms) and disease severity (i.e. risk factors suggestive of more complicated IBD)
  - The goals of treatment are clinical remission (i.e. absence of symptoms) and mucosal improvement (i.e. decreased intestinal inflammation) on a steroid-free treatment regimen
- We have many new therapies, mechanisms of action and routes of delivery
- Surgery may be needed (can be curative for Ulcerative colitis)

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Let’s stay in touch!

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Upcoming NEBGH events:

- **January 12 –** Mondays with Dr. Mark & Dr. Michael
- **January 21 –** The Benefits Leader's 90-Day ROI Playbook

SAVE THE DATE FOR THESE 2026 IN-PERSON EVENTS!

- **February 12 –** 31st Annual Tribute to Leadership
- **March 26 –** Cardiometabolic Health as a Business Imperative
- **June 18 –** 15th Annual Health & Wellness Benefits Conference

Remember to rate this webinar!