JHAs 2026

IBS – IBD

Irritable bowel syndrome & inflammatory bowel disease

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Vendredi, 24 avril 2026, 15:30 – 16:30

Intesto

Gastroenterologische Praxis & Crohn-Colitis-Zentrum Bern
Centre Fribourgeois de Gastroentérologie

GASTRO CENTRE GASTROENTÉROLOGIE BULLE

"The results are a bit surprising...
You are healthier than you look..."

Intestin irritable, IBS

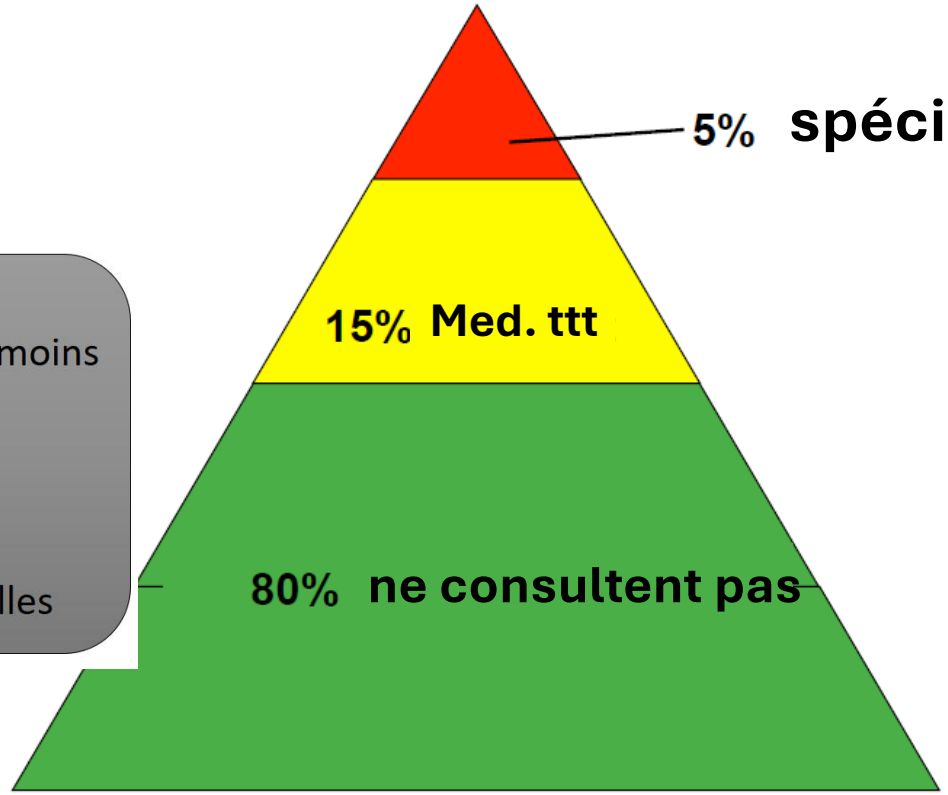
Intestin Irritable Epidemiologie

Prévalence selon les critères Rome IV

5.7% de la population générale

Douleur abdominal recurrente, en moyenne, au moins
1 jour par semaine durant les **3 derniers mois** , associé à au moins 2 des critères suivants:

1. Lié à la **defecation**
2. Associé à un changement de la **frequence des selles**
3. Associé à un changement de la **forme** (apparence) des selles



25-50 % des demandes d'avis gastro-entérologiques

Lacy BE et al. Gastroenterology 2016; 150 (6) : 1393–1407

Syndrome intestin irritable SII- D

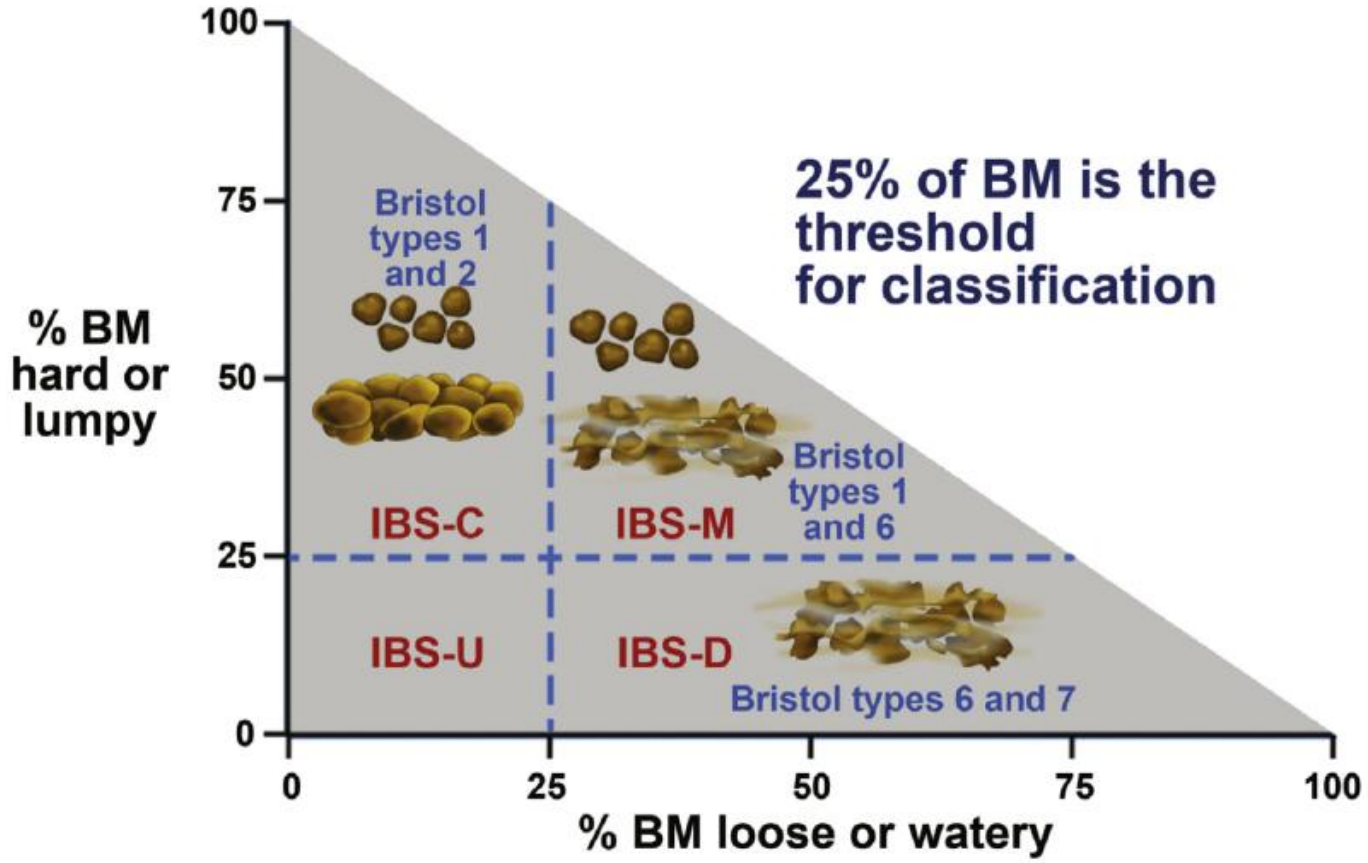
- Très frèquent, surtout ♀ jeune, symptomatologie chronique
- Criteres de Rome III et Manning
- Apparition post infectieuse fréquente
- Syndrome digestif fonctionnel le + fréquent
- Pathogenèse complexe

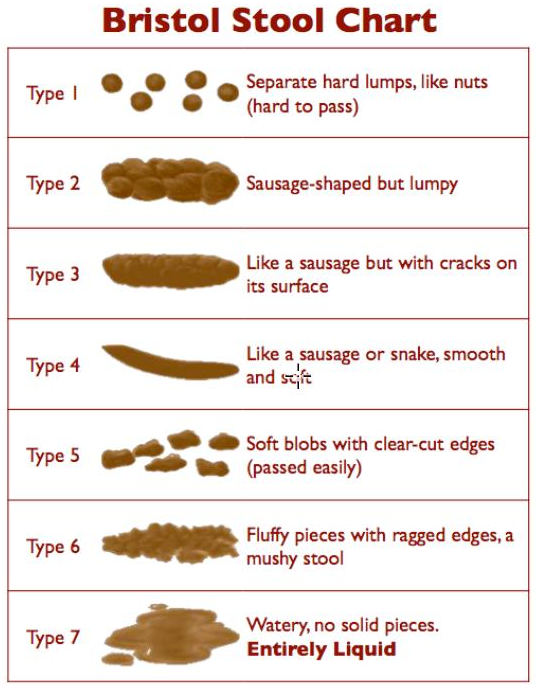
Tableau 1. Critères de Rome III (1)

Douleur abdominale ou inconfort digestif (sensation abdominale désagréable non douloureuse) survenant au moins 3 jours par mois durant les 3 derniers mois associée avec au moins 2 des critères suivants :
- Amélioration par la défécation ;
- Survenue associée à une modification de la fréquence des selles ;
- Survenue associée à une modification de la consistance des selles.

Les sous-groupes se définissent en fonction de la consistance des selles selon l'échelle de Bristol (voir figure 1).
SII constipation prédominante (C-SII): Bristol 1-2 ≥ 25 % du temps, Bristol 6-7 ≤ 25 % du temps.
SII diarrhée prédominante (D- SII) : Bristol 6-7 ≥ 25 % du temps, Bristol 1-2 ≤ 25 % du temps.
SII avec alternance diarrhée-constipation (M-SII) : Bristol 1-2 ≥ 25 % du temps et Bristol 6-7 ≥ 25 % du temps.
SII non spécifié : absence de critères suffisants pour répondre aux critères du C-SII, D-SII ou M-SII.

Sous-types d'intestins irritables





Lewis & Keaton. Scand J Gastroenterol 1997; 32:920-4

Lacy et al. Gastroenterology 2016;150:1393–1407

Syndrome intestin irritable SII - D

- But PeC: rassurer, diminuer douleurs, accompagner malade chronique

→ *Education thérapeutique importante !*

- Traitements: **loperamide (6-8x/j)**, antispasmodiques et **Ondansetron (Zofran ®), max. 3x 4mg/j.**

  Garsed K., Chernova J. et al. A randomised trial of ondansetron for the treatment of irritable bowel syndrome with diarrhea. Gut 2014; 63(10): 1617-25.
  Zheng Y, YU T et al. Efficacy and safety of 5-hydroxytryptamine 3 receptor antagonists in irritable bowel syndrome: a systematic review and meta-analysis of randomized controlled trials. PLoS One 2017 ; 12 (3), e0172846

- Modulation perception douleur: **anti-depresseurs tricycliques (Amitryptilin - Saroten®) 25mg – 150mg/j, SSRI – Venlafaxine (Effexor®) 75mg – 150mg.** psychologue, hypnose, relaxation.

- TTT empirique *(SIBO):* antibiotiques (e.g. rifaximine) / cholestyramine

Nee et al, Current and emerging drug options in the treatment of diarrhea predominant irritable bowel syndrome, 2015

Antispasmodiques / Spasmolytiques

- Action par relaxation de la musculature lisse

- Trimebutine (Debridat®,agoniste enképhalinergique)
- Mebeverine (Duspalatin®, Spasmopriv®, antimuscarinique)
- Pinaverium (Dicetel®, antagoniste recepteur Ca)
- Phloroglucinol (Spasfon®, antagoniste recept. Ca)

Inhibiteurs calciques action fibres lisses non vasculaires

- **Huile essentielle menthe** (antagoniste recept Ca) → **CARMENTHIN**

Pittler et al, Peppermint oil for irritable bowel syndrome: a critical review and metaanalysis, 1998

Alam et al, Efficacy of Peppermint oil in diarrhea predominant IBS - a double blind randomized placebo - controlled study, Mymensingh Med J 2013.

- Scopolamine hyoscyamine (**Buscopan®**, antimuscarinique anticholinergique)
- Simethicone (Andursil®, Disflatyl®, **Flatulex®** action uniquement physique)

Ruepert et al, Bulking agents, antispasmodics and antidepressants for the treatment of irritable bowel syndrome, Cochrane Database 2011

Syndrome intestin irritable SII - C

- But PeC: rassurer, diminuer douleurs, accompagner malade chronique

→ *Education thérapeutique importante !*

- Traitements: **Linaclotide (Constella® 290 mg /j) – CAVE IBD- C**, pas de prise en charge pour la constipation fonctionnelles.
- **Prucalopride (Resolor® 1mg – augm. à 2 mg)**

- Modulation perception douleur / El Diarrhée : **anti-depresseurs tricycliques (Fluoxetine – ISRS) 20mg – 80mg/j, Sertraline (Zoloft®) 50mg – 200mg/j.** psychologue, hypnose, relaxation.

- TTT empirique *(SIBO):* antibiotiques (e.g. rifaximine) / cholestyramine

Nee et al, Current and emerging drug options in the treatment of diarrhea predominant irritable bowel syndrome, 2015

Syndrome intestin irritable SII - D

- Regime FODMAPS

La démarche du régime pauvre en FODMAP en pratique

Le patient consulte son médecin généraliste pour des troubles gastro-intestinaux fonctionnels supposés

↓

Investigation et identification appropriées des symptômes potentiels

↓

Diagnostic positif de troubles gastro-intestinaux fonctionnels, comme une colopathie fonctionnelle

↓

Consultation d'un diététicien formé aux FODMAP

Phase 1

Élimination des FODMAP pendant 8 semaines

Le diététicien réalise un historique complet du patient et propose conseils, soutien et documentation pertinente sur l'alimentation pauvre en FODMAP

↓

Phase 2

Phase de réintroduction

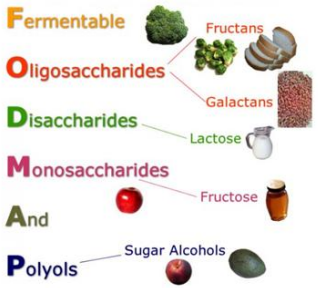
Le diététicien étudie les symptômes et le journal alimentaire de la période afin d'identifier les déclencheurs, puis conseille sur l'ordre de réintroduction des FODMAP

↓

Phase 3

Autogestion à long terme des symptômes

Le patient continue à inclure les FODMAP jusqu'à leur seuil de tolérance



LES FODMAP: LE NOUVEL ENNEMI NUMÉRO UN?

Le gluten ne serait pas le responsable des troubles intestinaux chez les personnes qui en présentent les symptômes. Il s'agirait en réalité des Fodmap: cinq catégories de sucres que l'on retrouve dans divers aliments et auxquels l'on peut être plus ou moins sensible.

Fructose en excès

Fruits:
Pommes, mangues, pastèques, fruits en conserve, jus de fruits, fruits séchés

Edulcorants:
Fructose, sirop de maïs

Miel...

Lactose

Lait:
Laits (de vache, de chèvre, de brebis), crèmes glacées, yogourts, desserts à base de lait

Fromages:
Fromages à pâte molle non affinés, ricotta, mascarpone, cottage cheese...

Fructanes

Légumes:
Artichauts, asperges, betteraves, brocolis, choux de Bruxelles, choux, aubergines, fenouil, ail, poireaux, toutes les variétés d'oignons

Céréales:
Blé ou seigle en grande quantité

Fruits:
Pommes, pastèques, kakis...

Galactanes

Légumineuses:
Pois chiches, haricots rouges, mungos, lentilles, fèves de soja...

Polyols

Fruits:
Pommes, abricots, avocats, mûres, cerises, litchis, nectarines, pêches, poires, prunes, pruneaux, pastèques

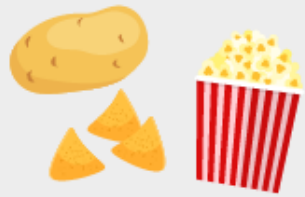
Légumes:
Choux-fleurs, poivrons verts, champignons, maïs sucré, pois mange-tout

Edulcorants:
Sorbitol, mannitol, isomalt, xylitol

Autres:
Gommes, menthe...

Eswaran et al, A Randomized Controlled Trial Comparing the Low FODMAP Diet vs. Modified NICE Guidelines in US Adults with IBS-D, 2016.
Nanayakkara et al, Efficacy of the low FODMAP diet for treating irritable bowel syndrome: the evidence to date, Clin Ex Gastroenterol 2016

LOW FODMAP DIET

FOOD	VEGETABLES	FRUITS	PROTEINS	FATS	STARCHES, CEREALS & GRAINS
EAT	 lettuce, carrot, cucumber	 strawberries, pineapples, grapes	 chicken, eggs, tofu	 oils, butter, peanuts	 potatoes, tortilla chips, popcorn
AVOID	 garlic, beans, onion	 blackberries, watermelon, peaches	 sausage, battered fish, breaded meats	 almonds, avocado, pistachio	 beans, gluten-based bread, muffins

Probiotiques

- But PeC: rassurer, diminuer douleurs, accompagner malade chronique

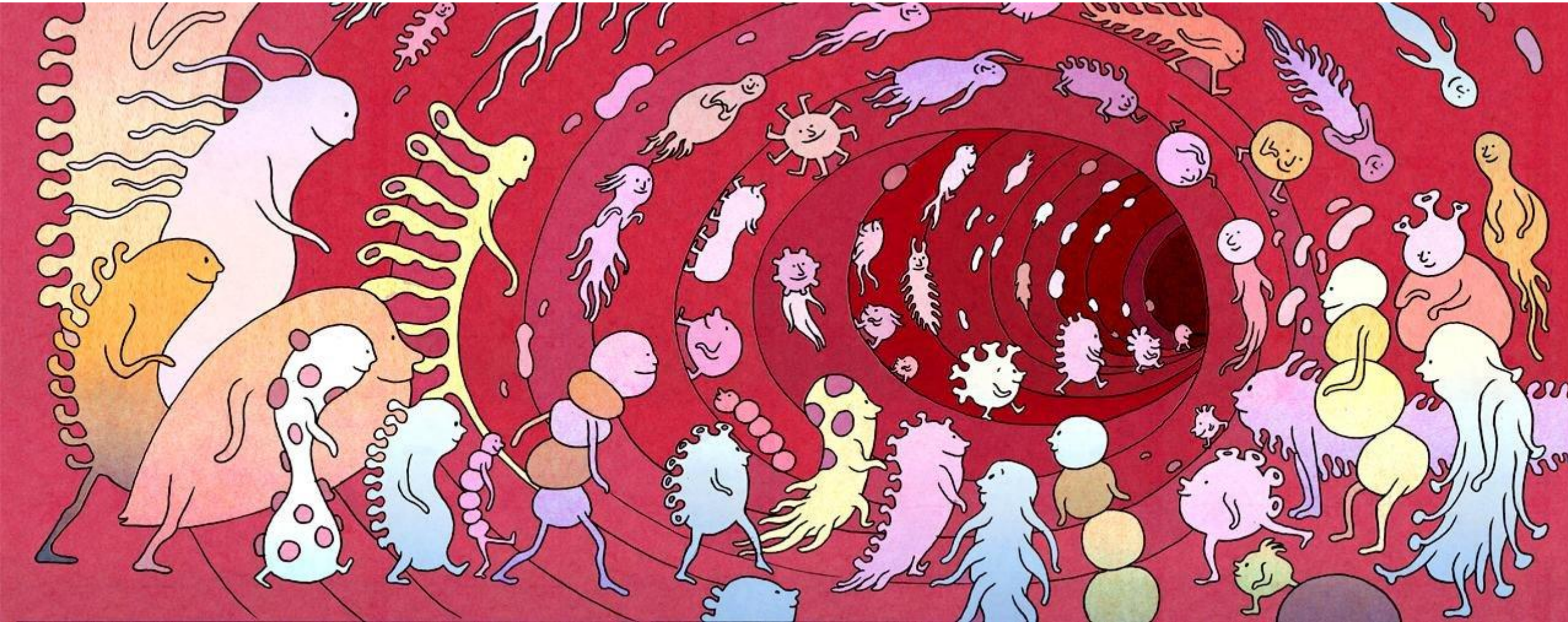
→ *Education thérapeutique importante !*

- Traitements: **Lactibiane:**

- **Alflorex ® , Bioflorin ®**

Nee et al, Current and emerging drug options in the treatment of diarrhea predominant irritable bowel syndrome, 2015

Maladies inflammatoires chroniques de l‘intestin



Source: MGH Crohn’s and colitis center

Diarrhée / MICIs

OUI ! → **> 250g selles** / j , ↑ frequence (>3), ↓ consistance

AIGUË (<2 sem.) **vs.** **CHRONIQUE** (> 3 sem.)

+/- sang

(**Dysenterie** vs. RCUH)

Cas particuliers :

- **Hôte : Immunosuppression, VIH**

  Age (*Rotavirus*), antibiotiques (*C. difficile*)

- **environnement : voyage, séjour hospitalier, tabac / alcool.**

NEJM 2014. April 17; 370 (16): 1532-40; IDSA Guidelines CID 2010.

AIGUË, infectieuse vs. chronique, inflammatoire (Morbus Crohn, colitis ulcerosa)

Diarrhée aiguë	**Diarrhée chronique (> 3 Wo)**
Douleurs abdominales, ev. sang	
+ Nausée,, vomissement	(Sub)ileus (comme complication)
Perte d'appetit → aigu (déshydratation), ensuite stable	perte pondérale … avec carence :
Rarement une anémie (déshydr.) pas de carence vitaminique	**Anémie** (fer, inflammatoire) **Malabsorption** (Vit.A, D, E, K, B12, ac. folique)
Fièvre	
Rares complications systémiques.	Megacolon toxique, perforation Fistule, Abcès, Sténose

TOXINES

Non-inflamm. , non invasive

- **1-24 heures**

(max. 72 St.)

- *Bactéries :*

S. Aureus,

B. Cereus,

C. perfringens,

E. coli (ETEC)

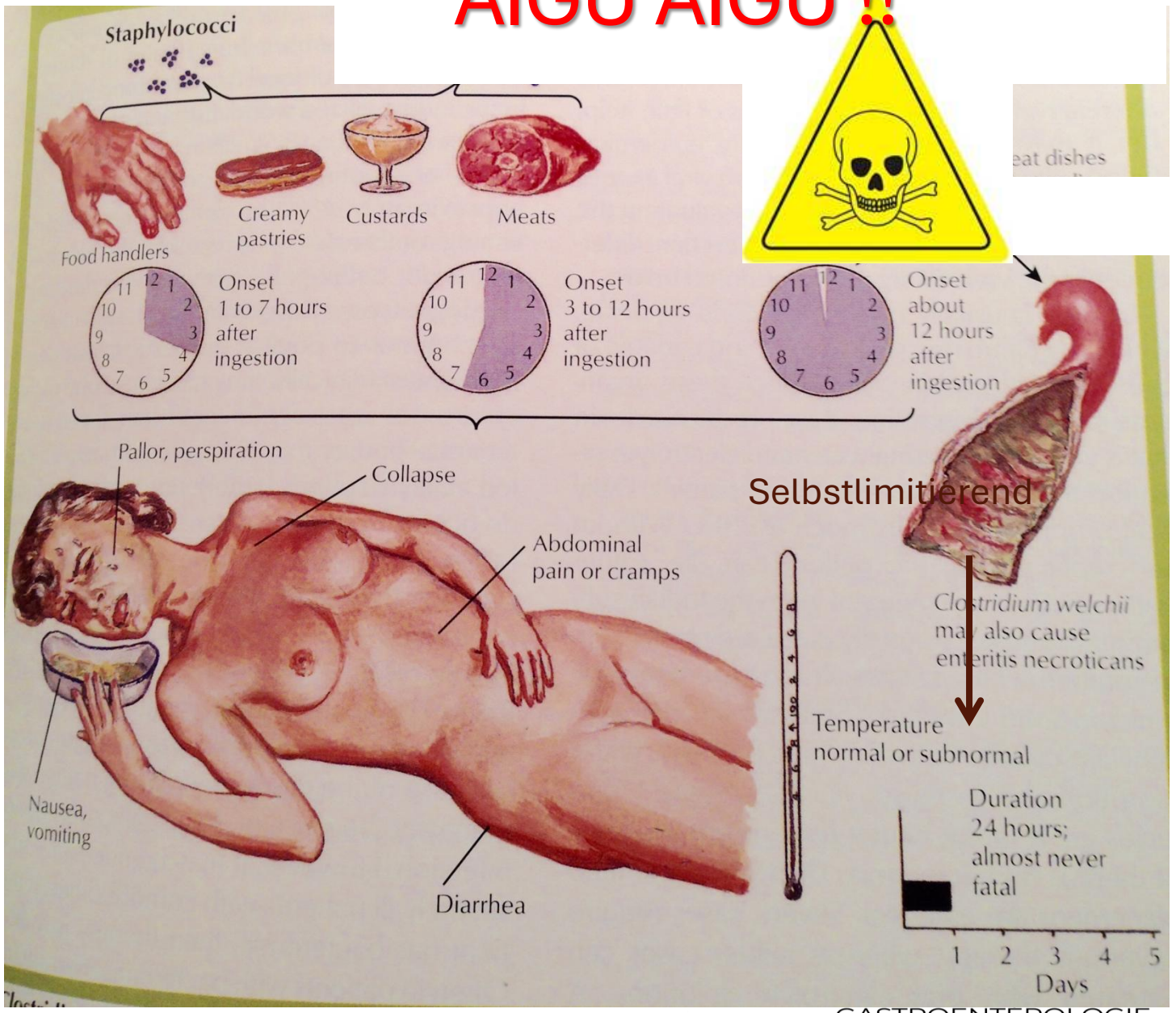
(>14-24 heures)

- *Virus:*

V. *cholerae,*
Rotavirus, Norovirus

(1-7 heures)

AIGU AIGU !!



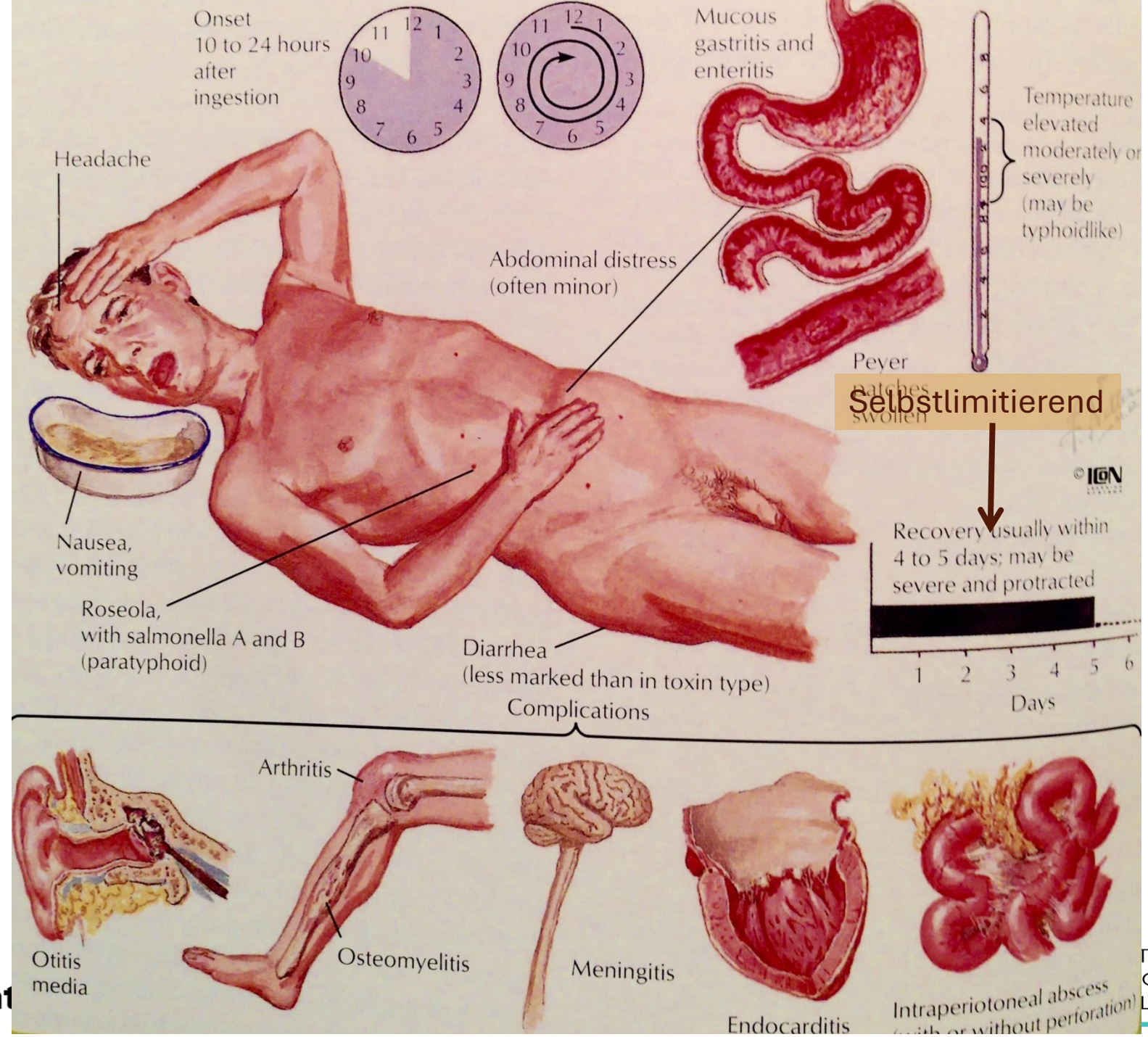
Forme adhérente :

AIGU
24 - 48 h.

INFLAMMATOIRE

dure
3 - 4 jours
(max. 2 sem.)

e.g. *S. enteridis*



Forme pénétrante:

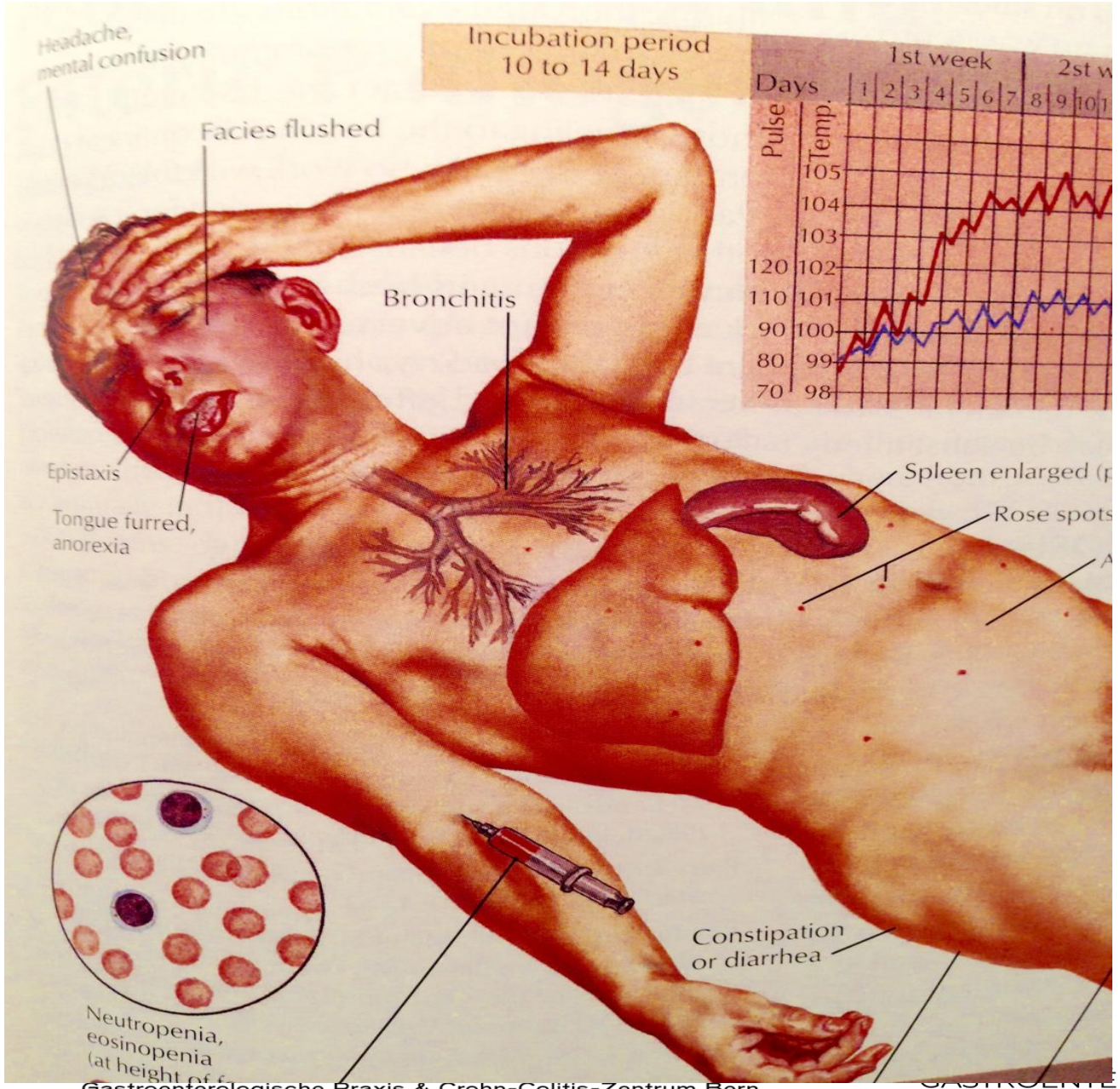
- **Persistant**

> 1 - 2 sem

+ SANG (=Dysenterie)

→ maladie systemique

e.g. *Salmonella typhi*



Therapie :

Non-inflamm.

- **EntéroToxine**

«diarrhée sécrétoire »

- *V. cholerae*,
- *E. coli* (ETEC),
- *C. perfringens*,
- *S. aureus*,
- *G. lamblia*,
- **Rotavirus**,
- **Norovirus**,
- Cryptosporidia,
- Microsporidia
- *Cyclospora*.

Hydratation:

- électrolytes

(ev. perfusion)

Inflammatoire

- *Bactéries adhérantes, Parasites*

→ *«invasion» superficielle*

- Shigella,
- E. coli (EIEC,EHEC),
- Salmonella enteritidis,
- V. parahaemolyticus,
- C. *difficile*,
- *Campylobacter jejuni*,

Pénétrante

- Invasif – pénétration
- Infection systémique

- *Salmonella typhi*,
- ***Yersinia. enterocolitica.***
- *Entamoeba histolytica*

à 3-5 jours ET Fièvre

→ ***Culture* Bactéries / Parasiten (si longue durée)**

Antibiotique : ORAL - empririque

- Fluoroquinolone :

(ciprofloxacine 500mg / norfloxacine 400mg: 2x,5-7 Tage)

- Alternative (allergie / résistance) :

azithromycine 500 mg/j pour 2-3 jours

C. diff. : metronidazol / Vancomycin

Amibes : Metronidazol

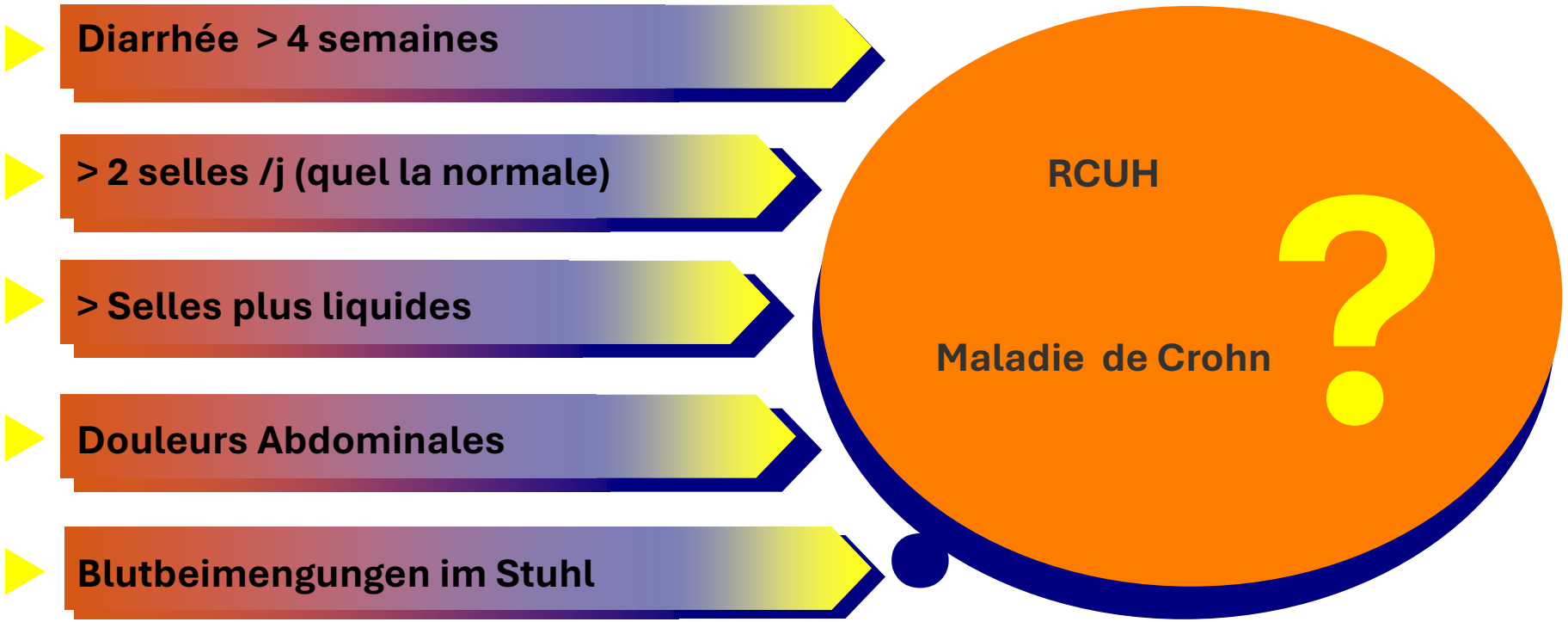
< 2 jours

> 1 semaine

Durée d'incubation

Durée + Sang

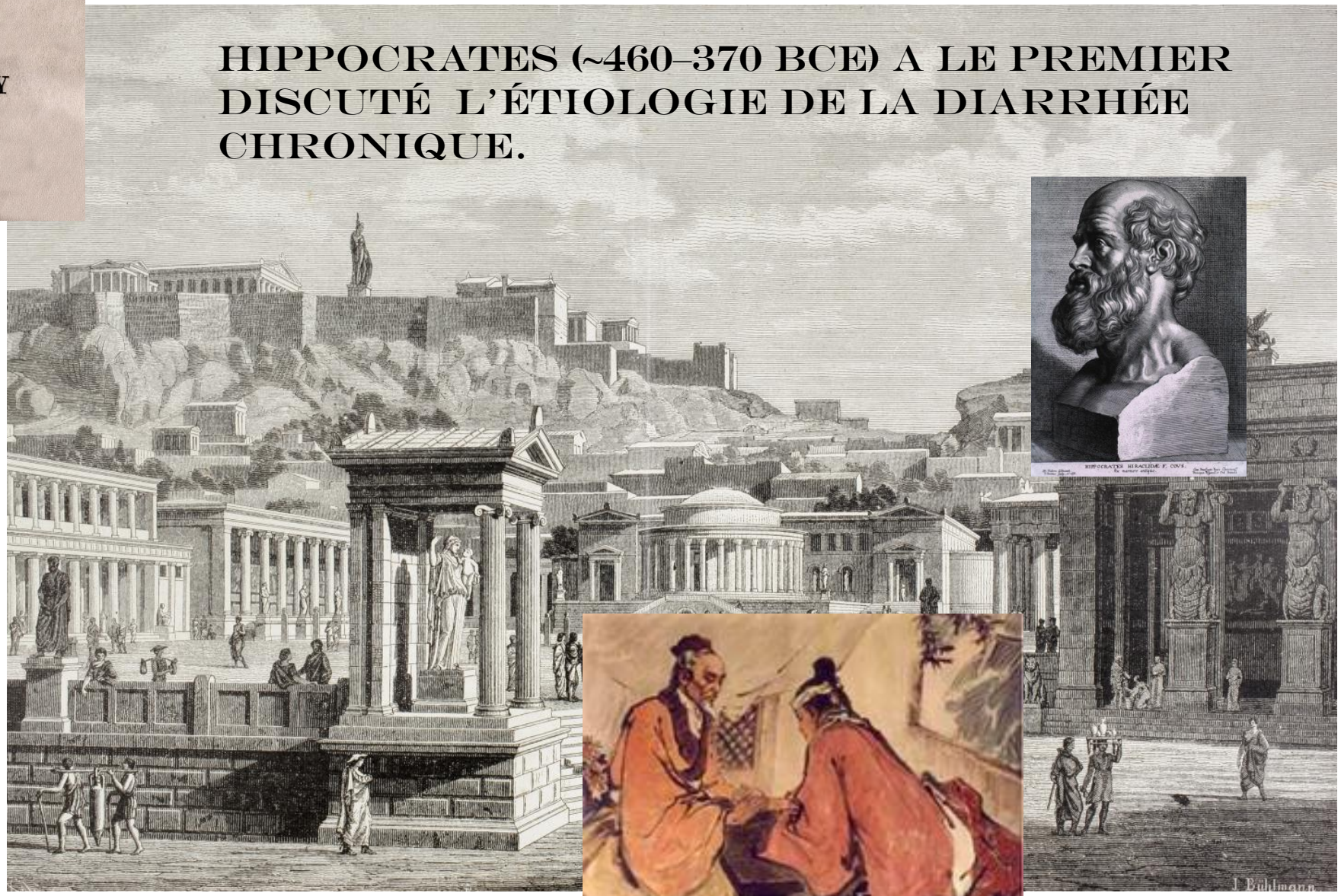
Maladies inflammatoires intestinales



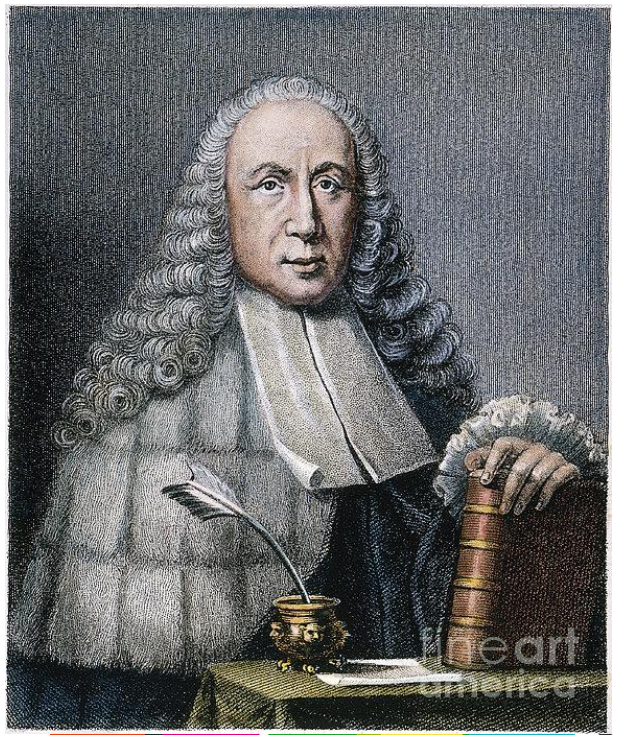
A penser chez les jeunes : 20-30 ans !

HISTORY

HIPPOCRATES (~460–370 BCE) A LE PREMIER DISCUTÉ L'ÉTIOLOGIE DE LA DIARRHÉE CHRONIQUE.



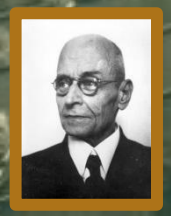
Le Médecin italien Giovanni Battista Morgagni (1682-1771) en 1769, décrit un jeune homme avec une maladie chronique, avec douleurs abdominales et diarrhée.



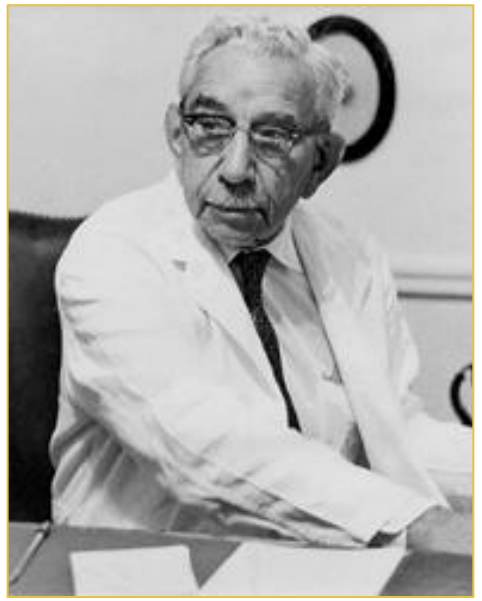
Puis d'autres cas décrits :

- 1898 par John Berg
- 1904 par le chirurgien polonais Antoni Lesniowski
- 1913, le médecine écossais T. Kennedy Dalziel,

→ 9 cas de patients souffrants d'obstruction intestinals décrits au meeting de la British Medical Association.



Morbus Crohn / Maladie de Crohn



Burrill B. Crohn Mount Sinai Hospital New York City

1932: première description : 14 patients

important in making the intestinal incision for any ureteral transplantation. The intestine should be held by four traction loops, which, when possible, should include any visible vessel in the intestinal wall that may cross the proposed line of incision. With a very sharp lance pointed knife, the peritoneum and part of the muscular coat are cut. The knife is now turned flat-
oint of the knife the remaining
ed through with gentle strokes,
scle ends to separate without
nucosal vessels or membrane.
knife, the muscle coat is pushed
outer surface of the intestinal
This part of the operation must
y.
of its simplicity, seems destined
nt rôle. Based on experimental
nd the very limited experience
s safe to say, even now, that it is

REGIONAL ILEITIS

A PATHOLOGIC AND CLINICAL ENTITY

BURRILL B. CROHN, M.D.
LEON GINZBURG, M.D.
AND
GORDON D. OPPENHEIMER, M.D.
NEW YORK

We propose to describe, in its pathologic and clinical details, a disease of the terminal ileum, affecting mainly young adults, characterized by a subacute or chronic necrotizing and cicatrizing inflammation. The ulceration of the mucosa is accompanied by a disproportionate connective tissue reaction of the remaining walls of the involved intestine, a process which frequently leads to stenosis of the lumen of the intestine, associated with the formation of multiple fistulas.

Questions

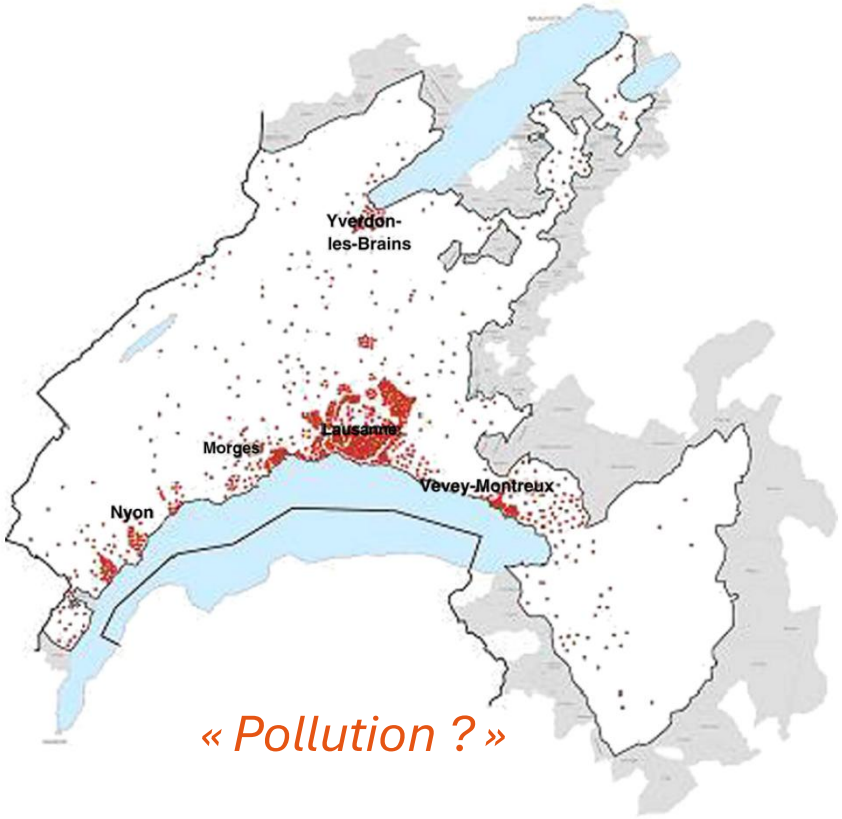
Gradient Ville → Campagne

50% - 60% plus de risque dans les villes

Est-ce fréquent ?

Quels patients ?

P. Juillerat, *et al.* Journal of Crohn's and Colitis (2008) 2, 131–141



« Pollution ? »

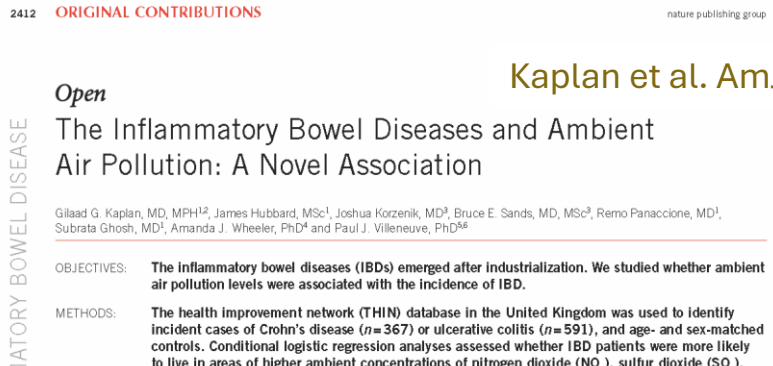
Prévalence :

1 Personne / 500.

12’000 Patients

in der Schweiz

Kaplan et al. AmJ Gastro 2010:



2412 ORIGINAL CONTRIBUTIONS nature publishing group

Open

The Inflammatory Bowel Diseases and Ambient Air Pollution: A Novel Association

Gilaad G. Kaplan, MD, MPH[1,2], James Hubbard, MSc[1], Joshua Korzenik, MD[3], Bruce E. Sands, MD, MSc[3], Remo Panaccione, MD[1], Subrata Ghosh, MD[1], Amanda J. Wheeler, PhD[4] and Paul J. Villeneuve, PhD[5,6]

OBJECTIVES: **The inflammatory bowel diseases (IBDs) emerged after industrialization. We studied whether ambient air pollution levels were associated with the incidence of IBD.**

METHODS: **The health improvement network (THIN) database in the United Kingdom was used to identify incident cases of Crohn's disease ($n=367$) or ulcerative colitis ($n=591$), and age- and sex-matched controls. Conditional logistic regression analyses assessed whether IBD patients were more likely to live in areas of higher ambient concentrations of nitrogen dioxide (NO_2), sulfur dioxide (SO_2),**

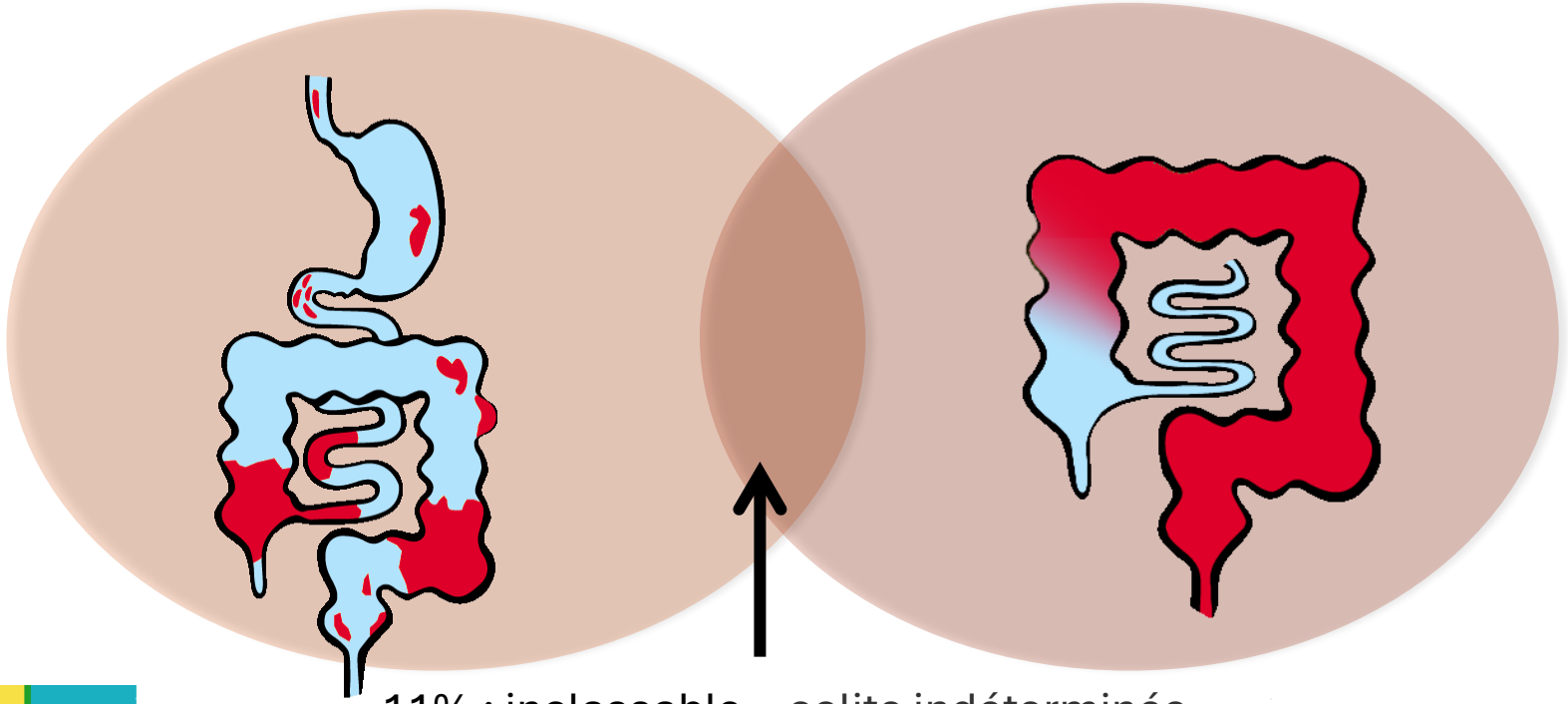
Intesto

Gastroenterologische Praxis & Crohn-
Centre Fribourgeois de Gastroentérolo

Connaissons- nous encore d'**autres personnes** atteintes par ces maladies ?

Maladie de Crohn

Colite ulcéreuse



11% : inclassable - colite indéterminée

- ***Shannen Doherty***

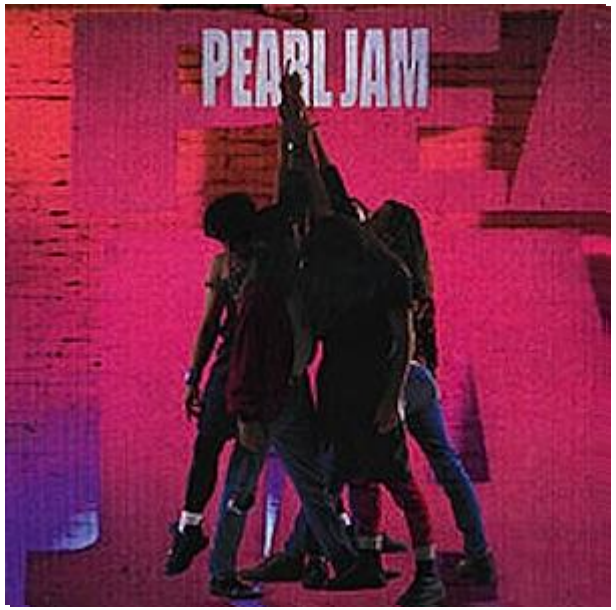
M. Crohn

« Ike »

- ***Dwight Eisenhower***

34th U.S. President (1953-1961)

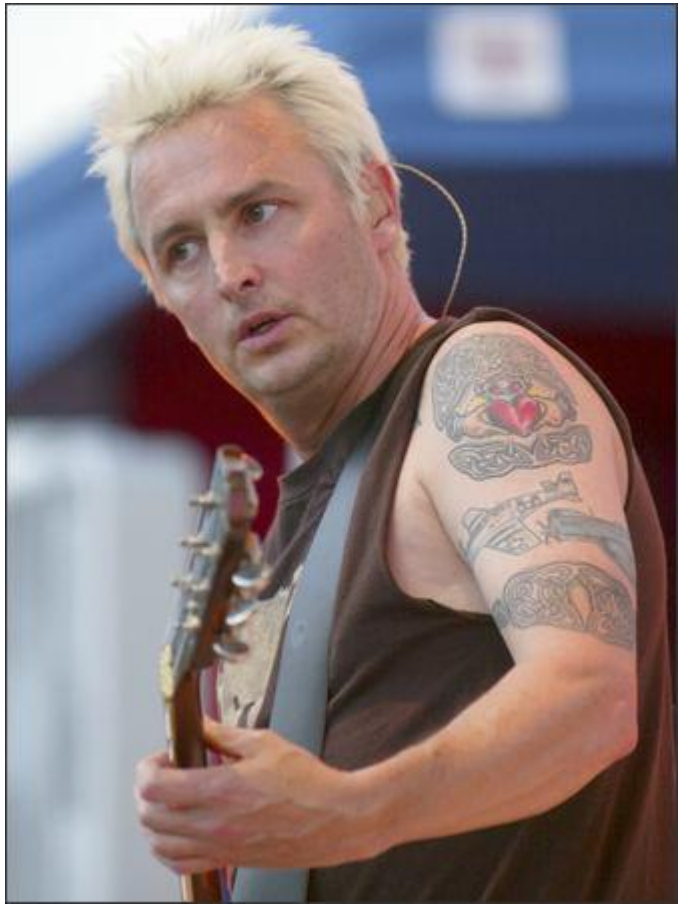
M. Crohn, 1956



- ***Mike Mc Cready***

Lead Guitar

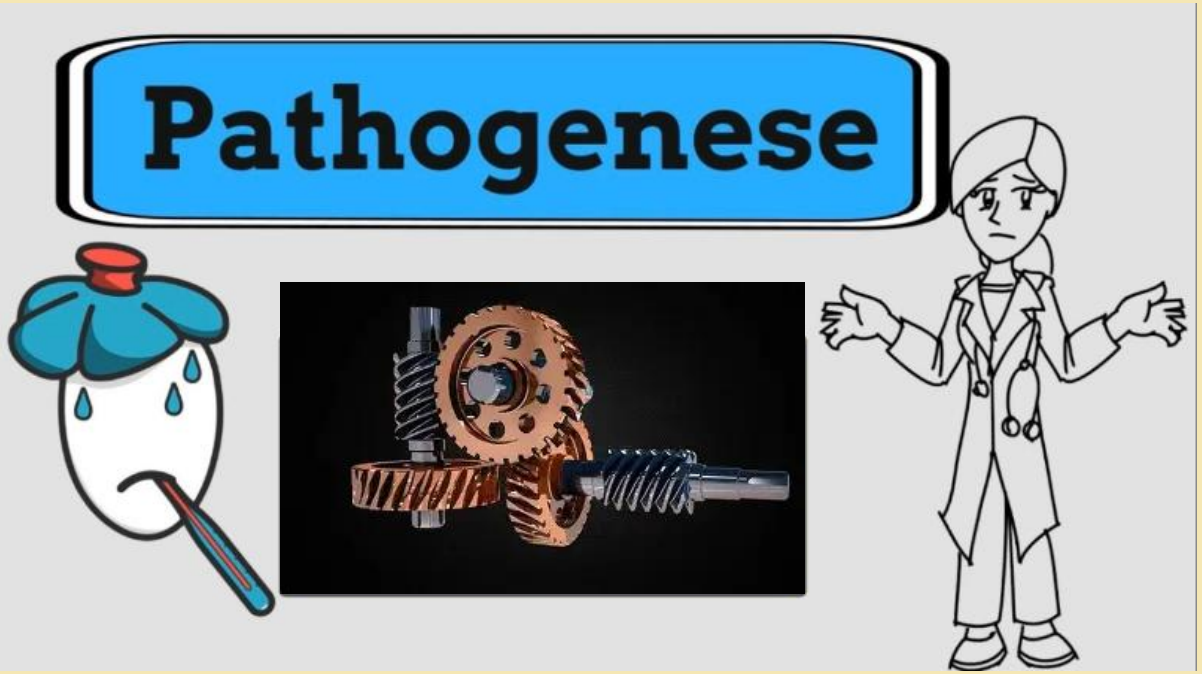
PEARL JAM

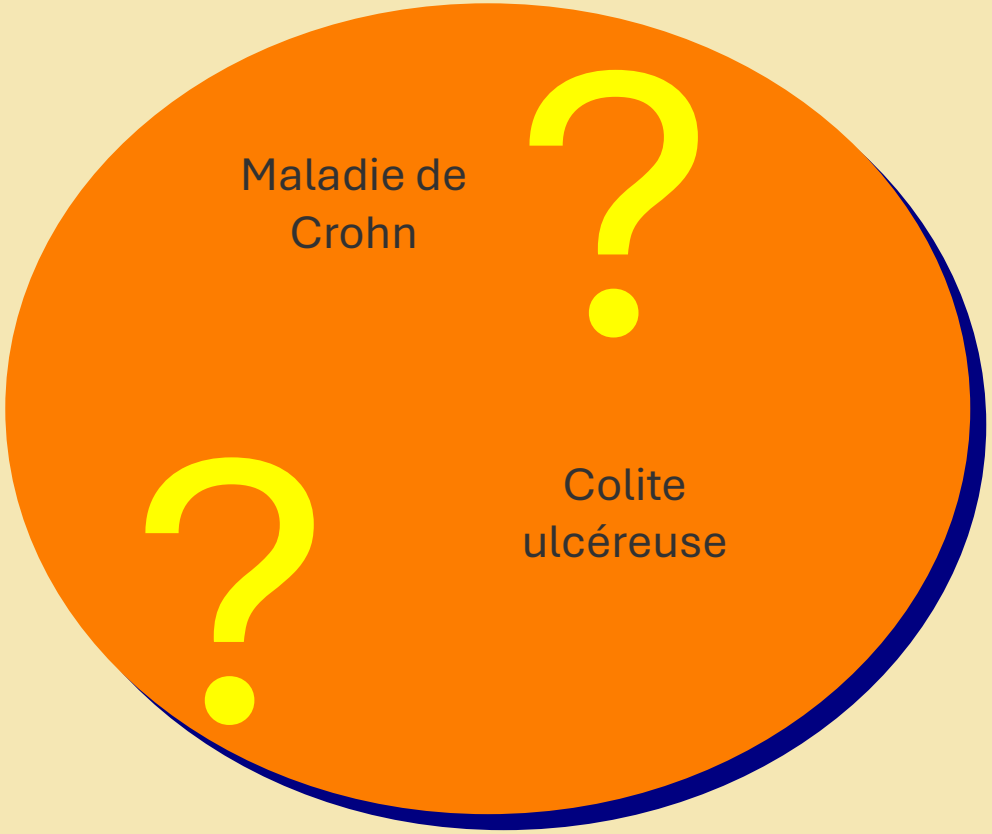


M. Crohn, 1988

- ***Anastacia***

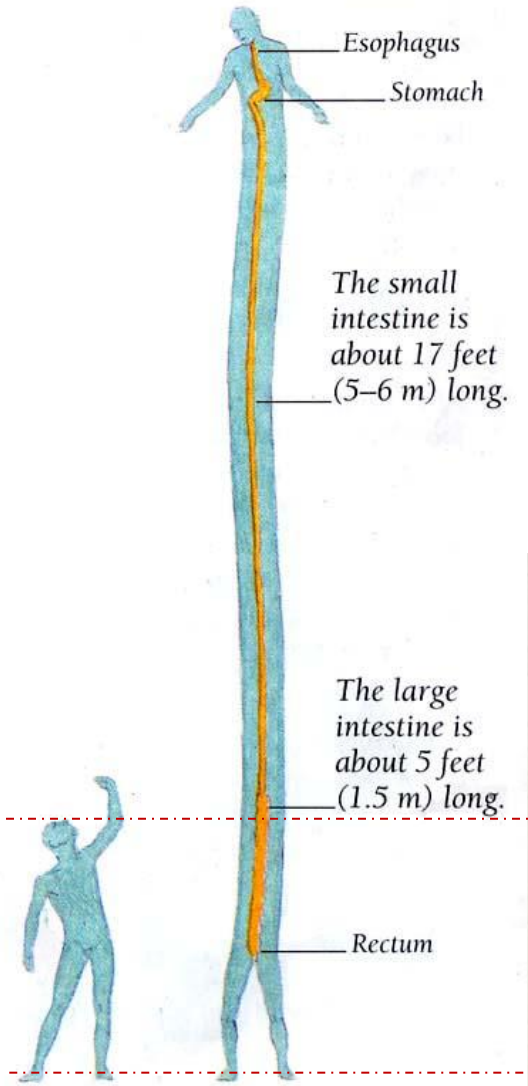
M. Crohn, 1981
(13 ans)

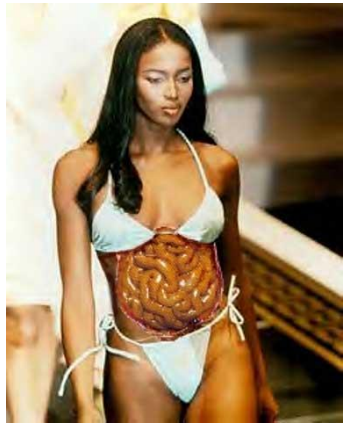


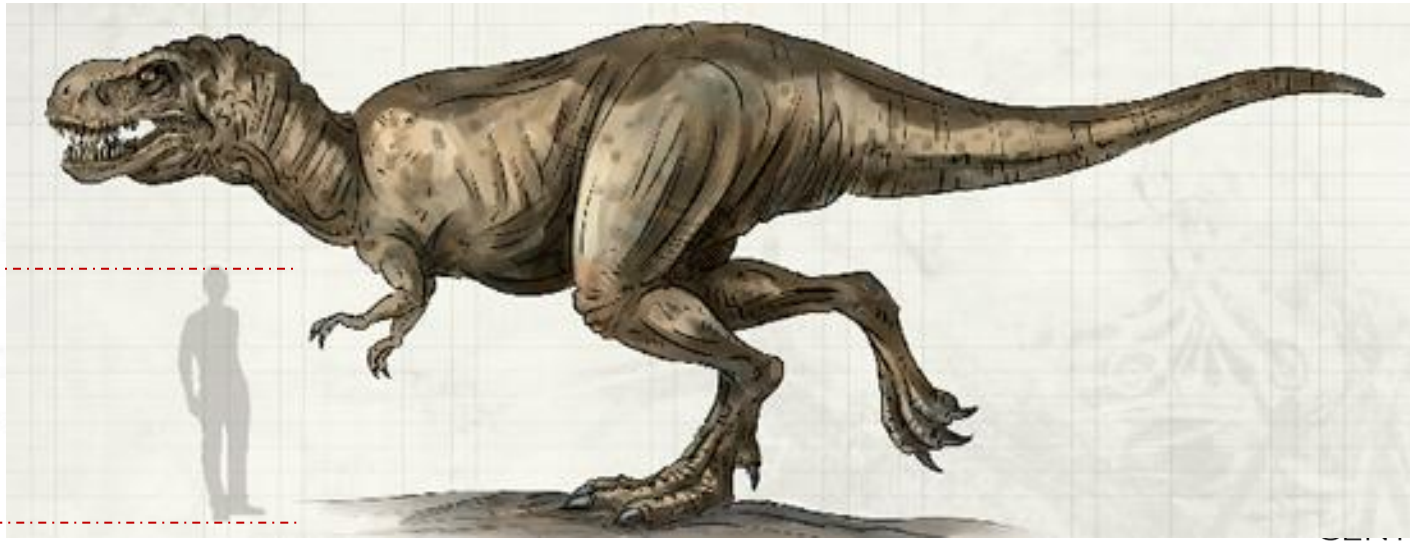


POURQUOI

Concernant les MICIs: le problème… notre intestin est très long!



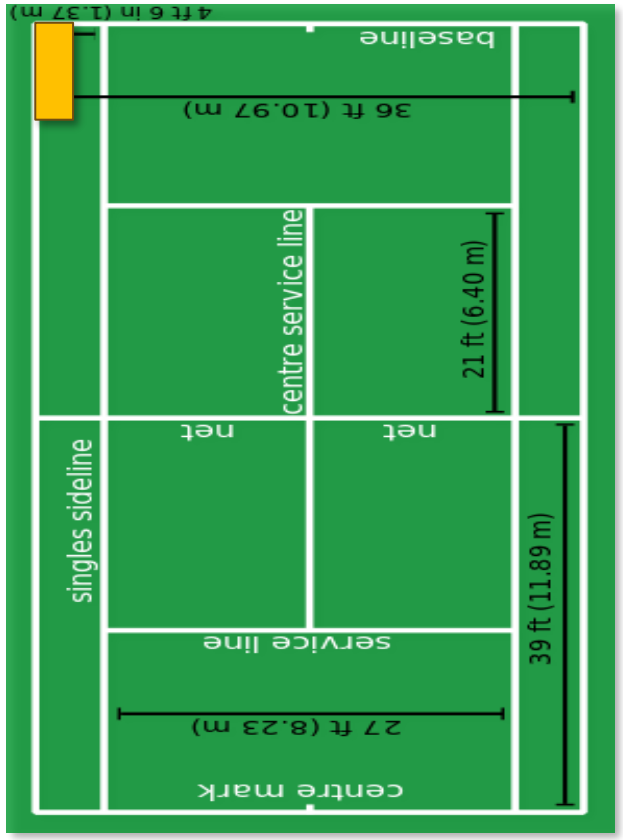


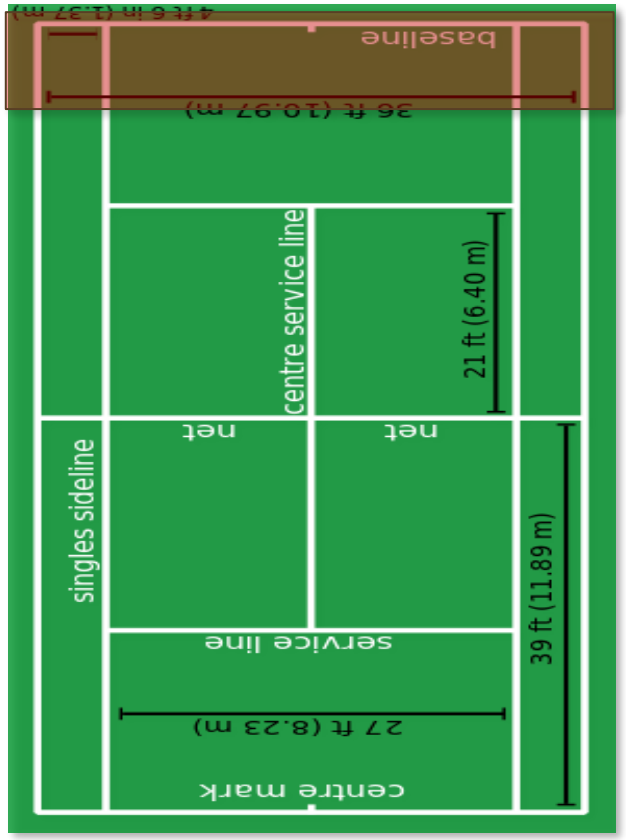


et représente un énorme surface !

peau 1-2 m^2

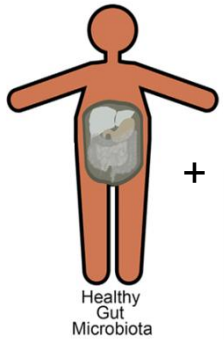
poumons 100 m^2





Intestin 300 - 500m^2 !!

Microbiote

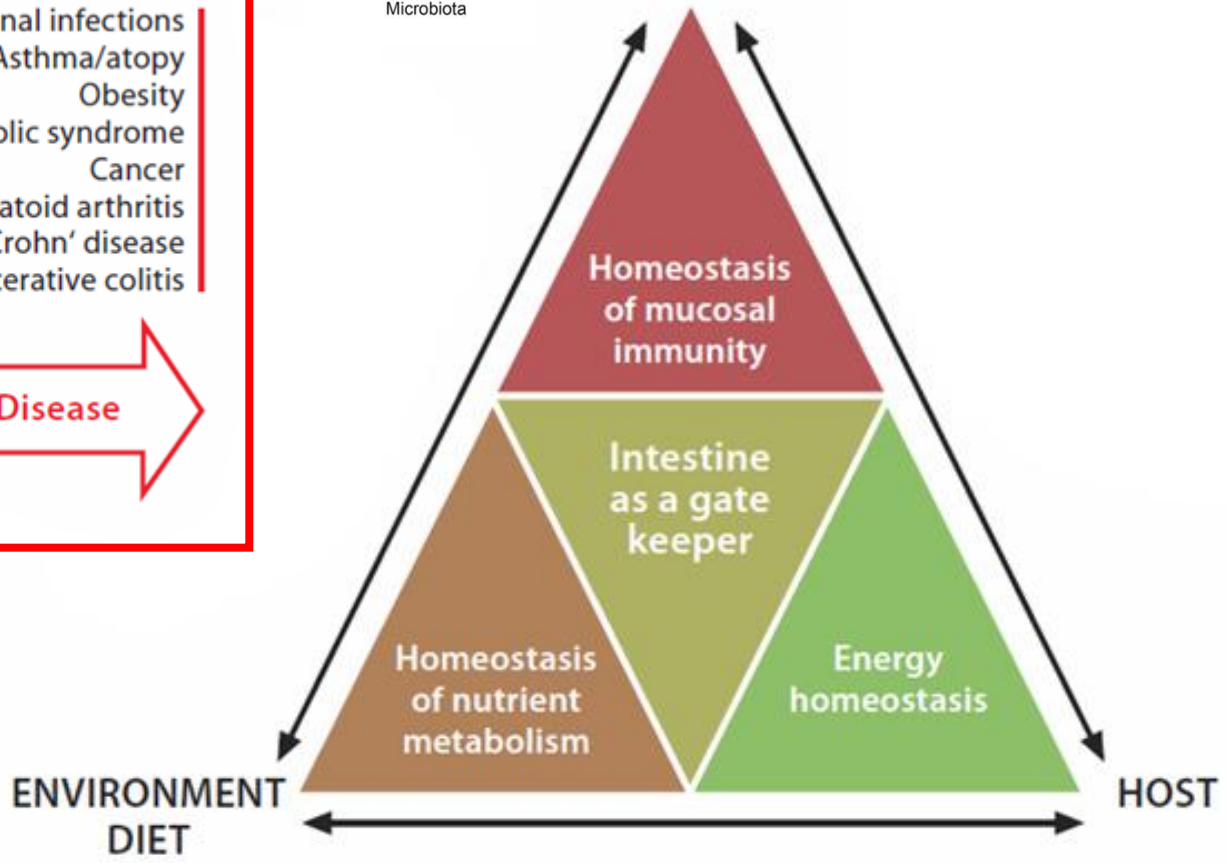


+

MICROBIOTA = *considéré comme un nouvel ORGANE !*

Gastrointestinal infections
Asthma/atopy
Obesity
Metabolic syndrome
Cancer
Rheumatoid arthritis
Crohn' disease
Ulcerative colitis

Disease

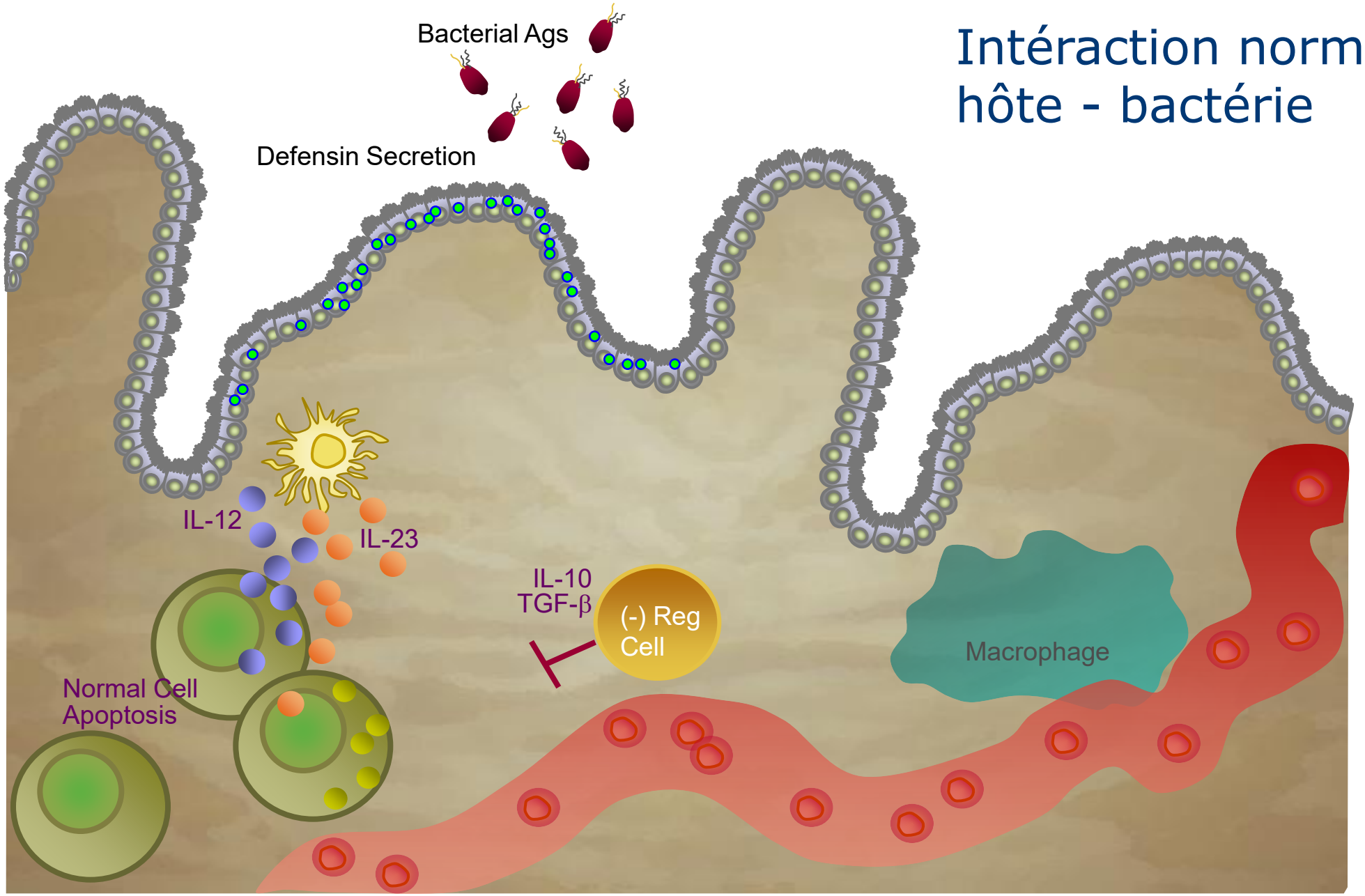


Resistance to infections
No intestinal disorders
Healthy ageing

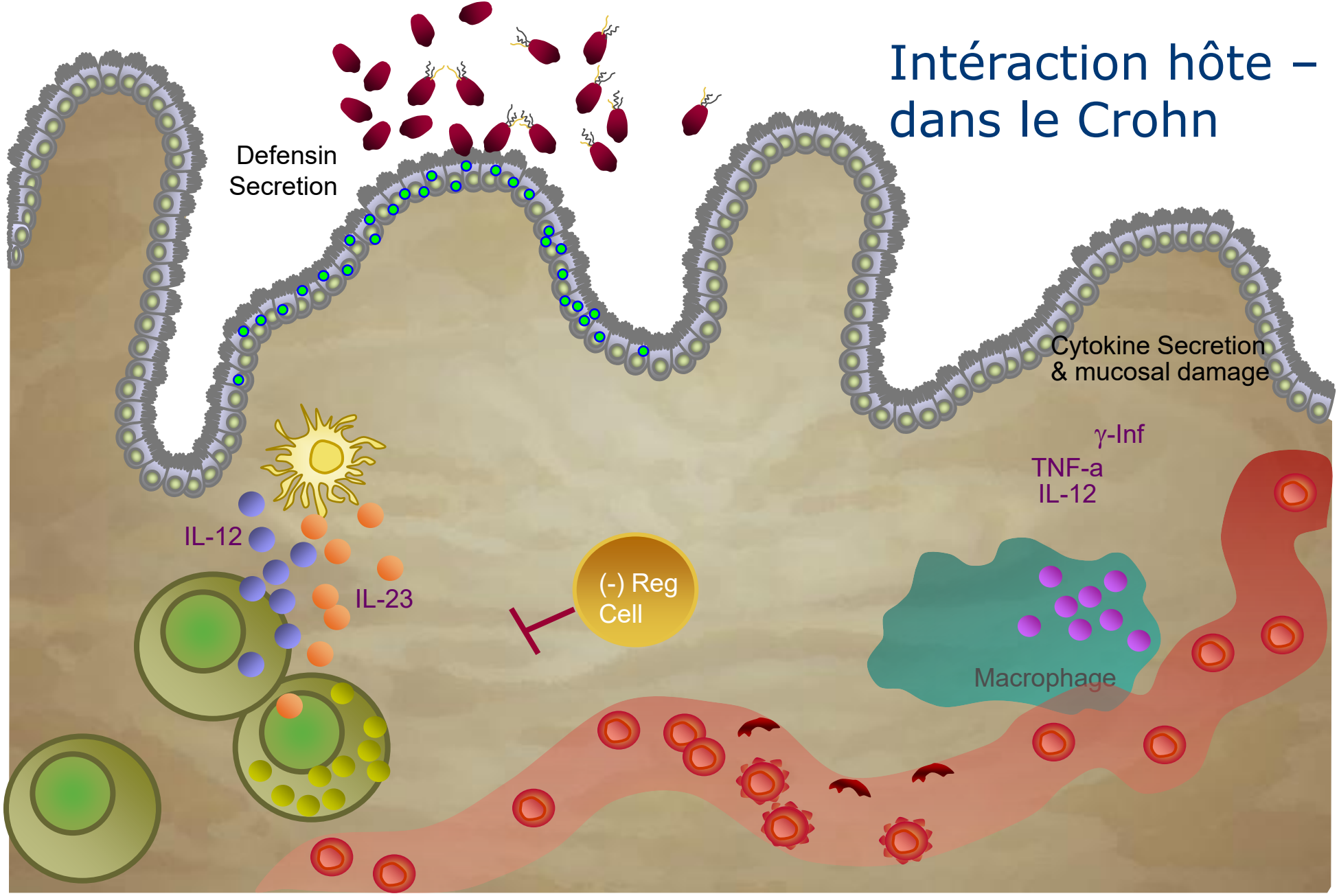
Health

Adapté de Wouters Dig Dis 2012;30(suppl 3):33–39 and Martins dos Santos Current Opinion in Biotechnology 2010, 21:539–550

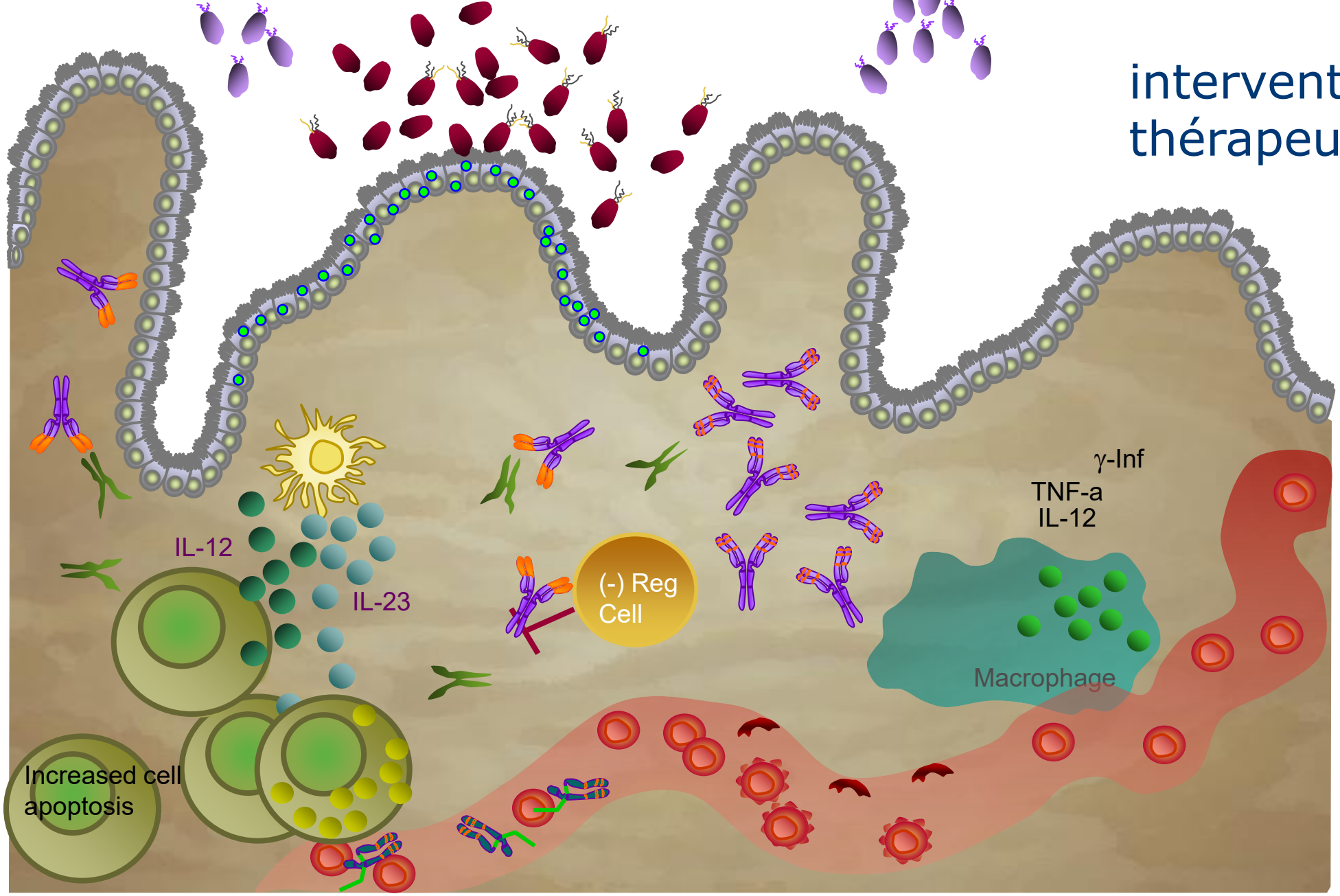
Intéraction normale : hôte - bactérie



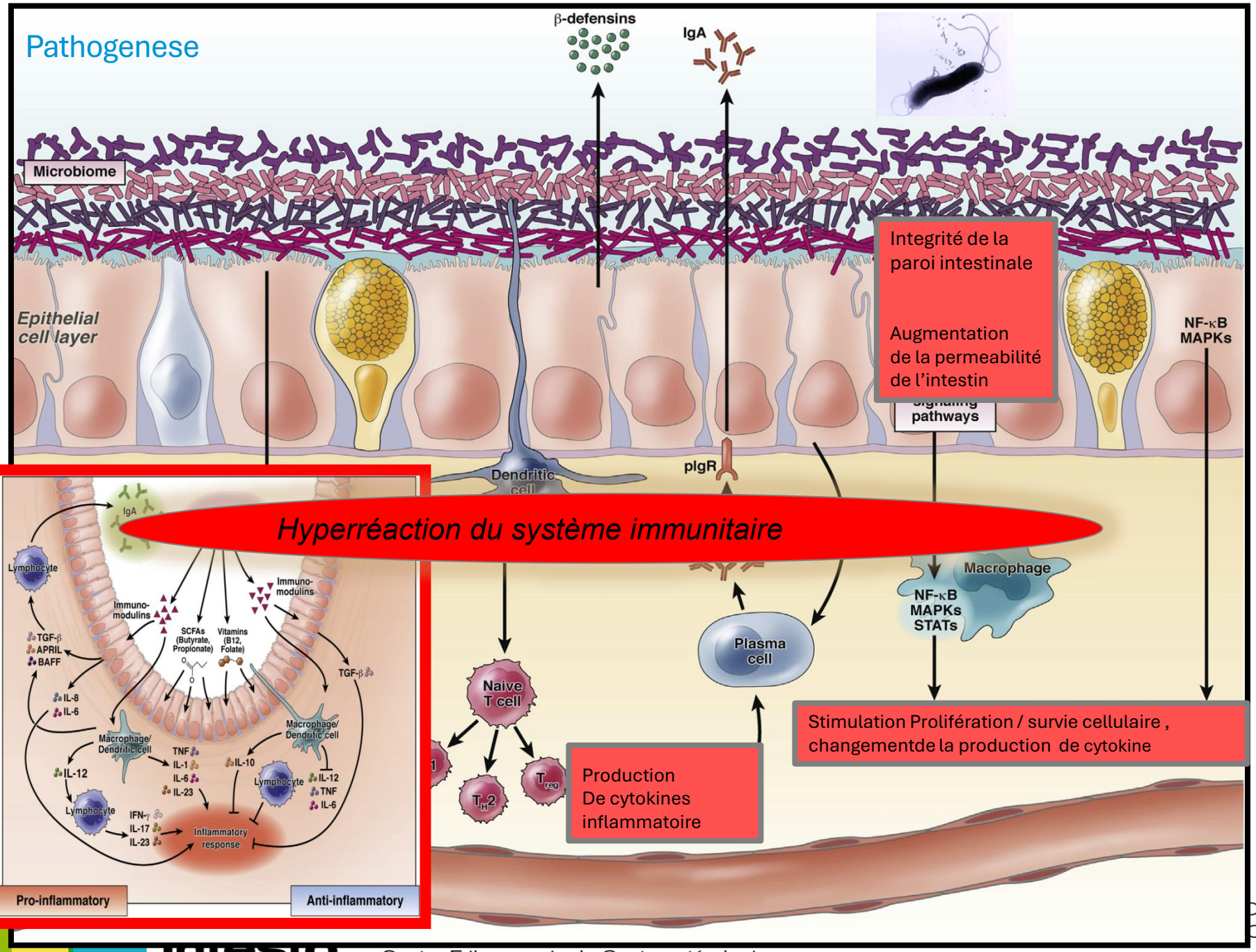
Intéraction hôte – bactérie dans le Crohn



interventions thérapeutiques



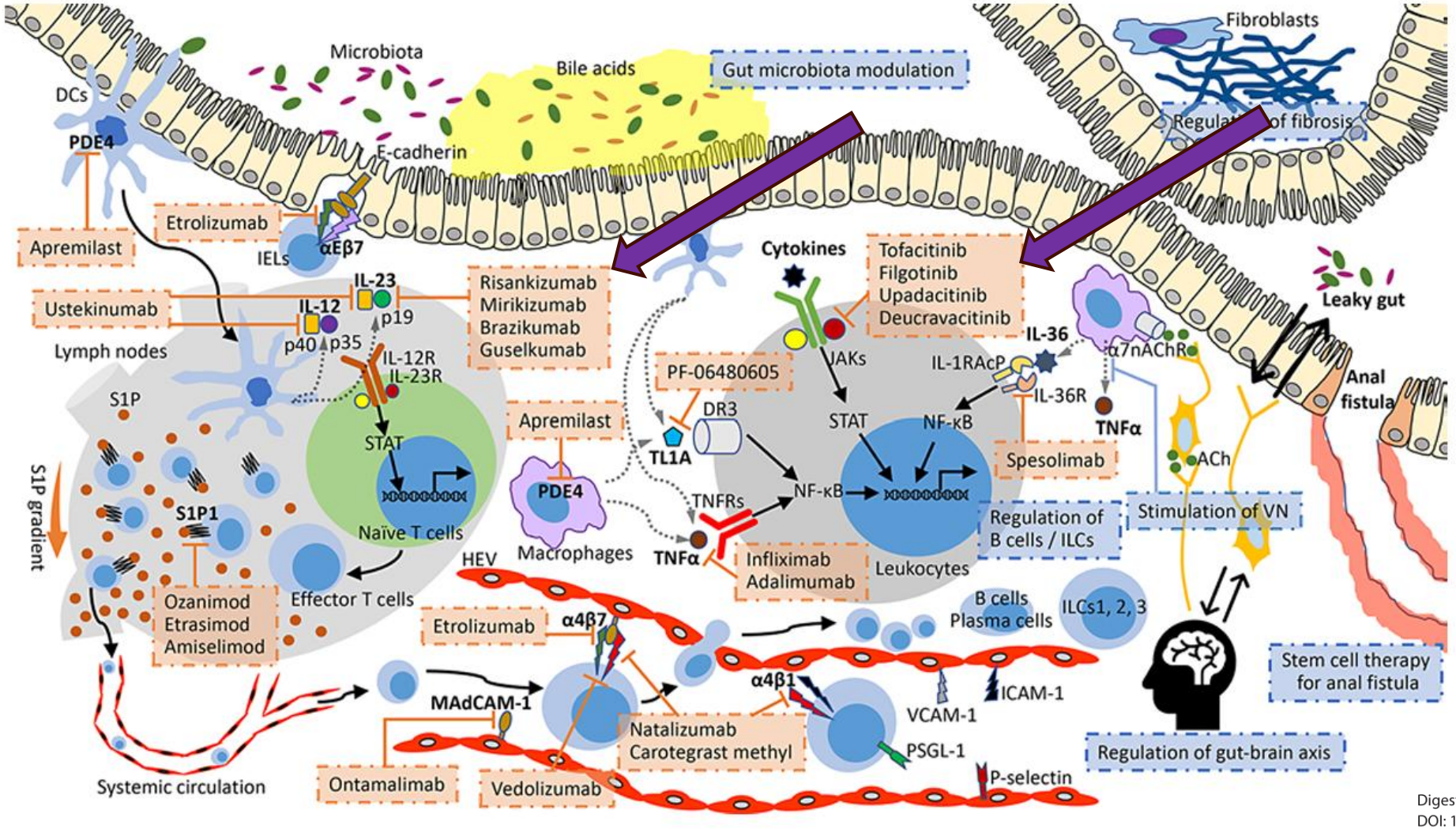
Pathogenese



Erickson AR, et al. Integrated metagenomics/metaproteomics reveals human host-microbiota signatures of Crohn's disease. PLoS One 2012;7:e49138

Hollister et al. Gastroenterology 2014;146:1449–1458: Compositional and Functional Features of the Gastrointestinal microbiome and Their Effects on Human Health

C‘est même plus compliqué …



Digestion 2023;104:74–81
DOI: 10.1159/000527422

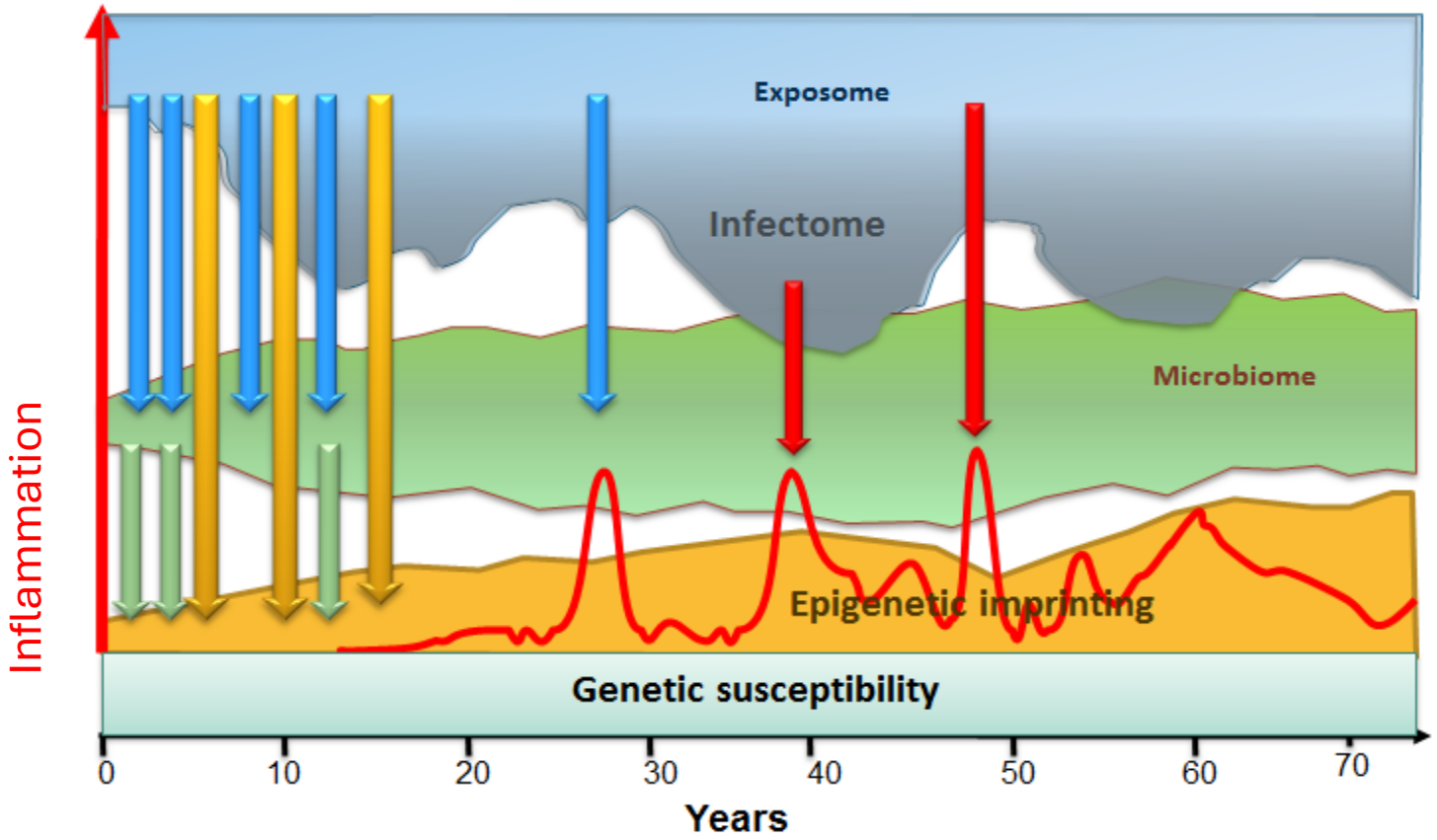
FACTEURS DE RISQUE

Colite ulcereuse ?

Maladie de Crohn

Comment influencer ?

«Exposome»



From Rogler G, Vavricka S. Exposome in IBD: Recent Insights in Environmental Factors that Influence the Onset and Course of IBD.Inflamm Bowel Dis. 2014

Darmflora

sozialer Status

Medikamenten

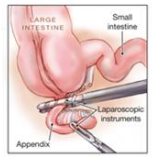
Stress

Diät

Umweltfaktoren

Rauchen

Appendektomie



Genen

Immunsystem

M. Crohn/ Colitis ulcerosa

CAVEAT : Diese Umweltfaktoren können eine gewisse Rolle spielen, aber (ausser für Rauchen und NSAIDS) gibt es keine Empfehlungen die zu vermeiden.

Das Immunsystem und die Prädisposition (Genetik) sind auch wichtig.

→ *Der Einfluss auf CED ist MULTIFAKTORIEL, nicht universell.*

Tabac / Nicotine

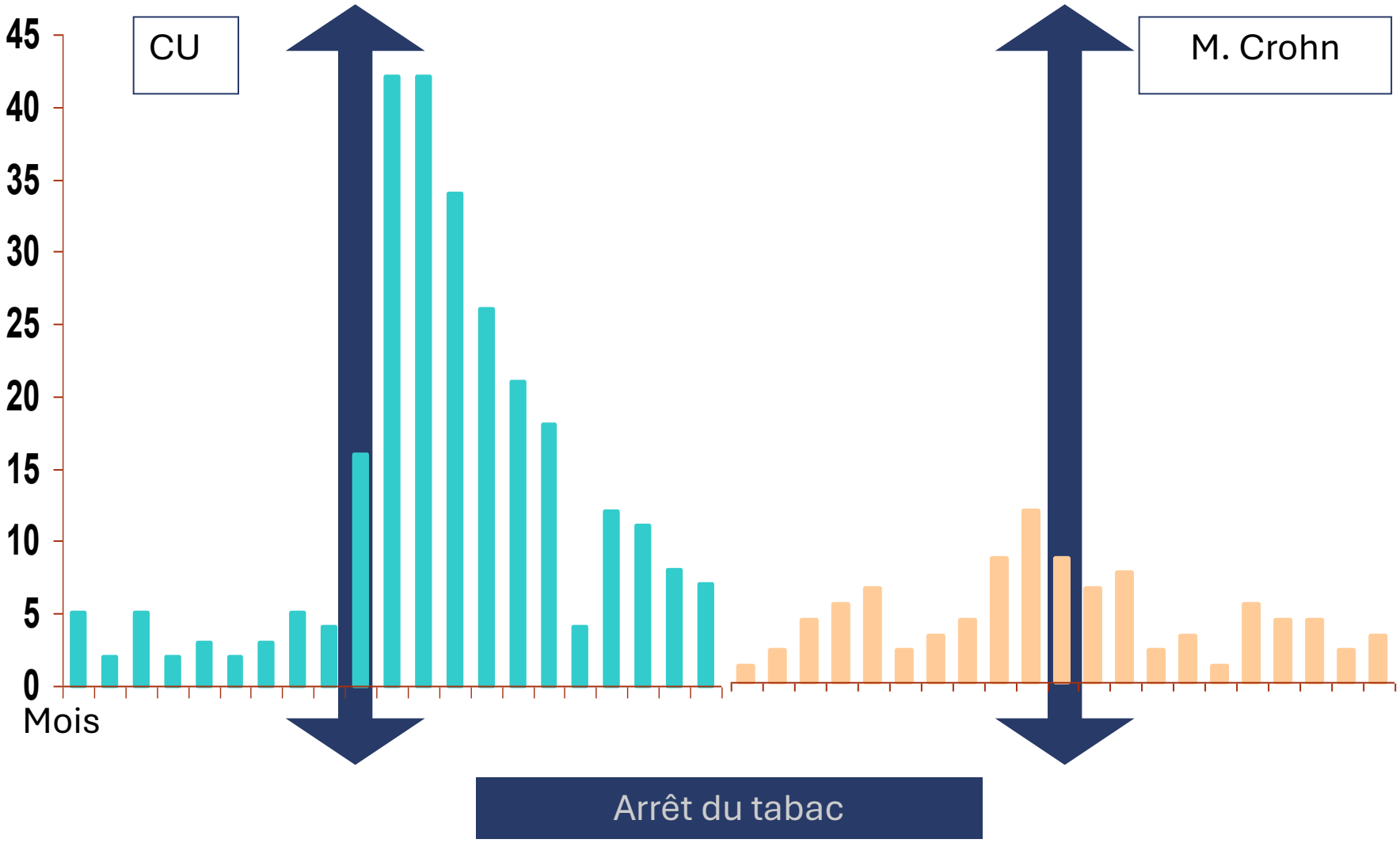
Protecteur pour la colite ulcereuse !! (0R 0.5)

MAIS, pas une raison pour fumer!

Et. par contre negativ pour le Crohn (OR 2.0)

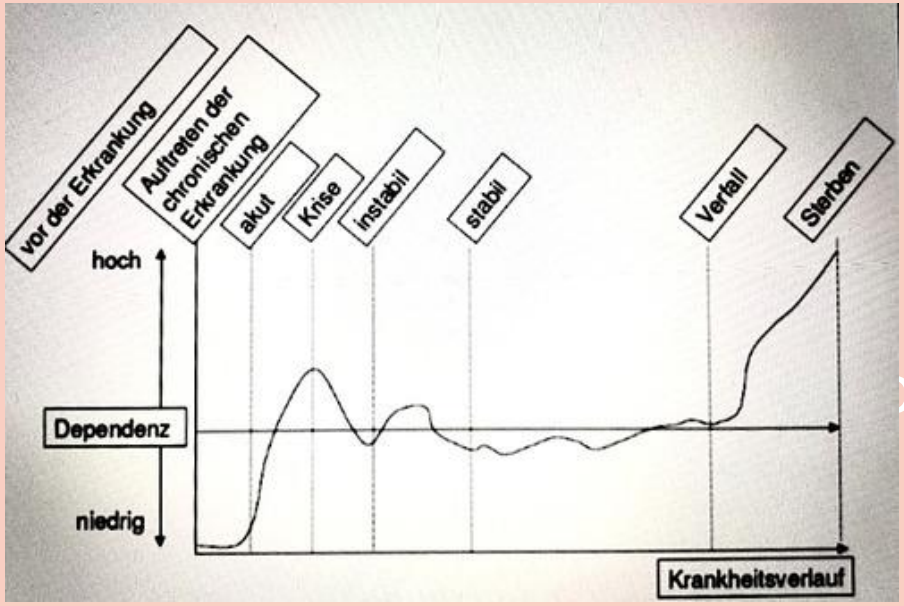
Cosnes J et al. *Best Pract Res Clin Gastroenterol*. 2004;18:481-496.
Beaugerie L et al. *Am J Gastroenterol*. 2001
Seksik P et al. *Inflamm Bowel Dis*. 2009
Cosnes J, et al. Gastroenterology 2001;120:1093–9.
Rubin DT, Hanauer SB. Eur J Gastroenterol Hepatol 2000;12:855– 62.

Arrêt du tabac et developpement d'une MICI

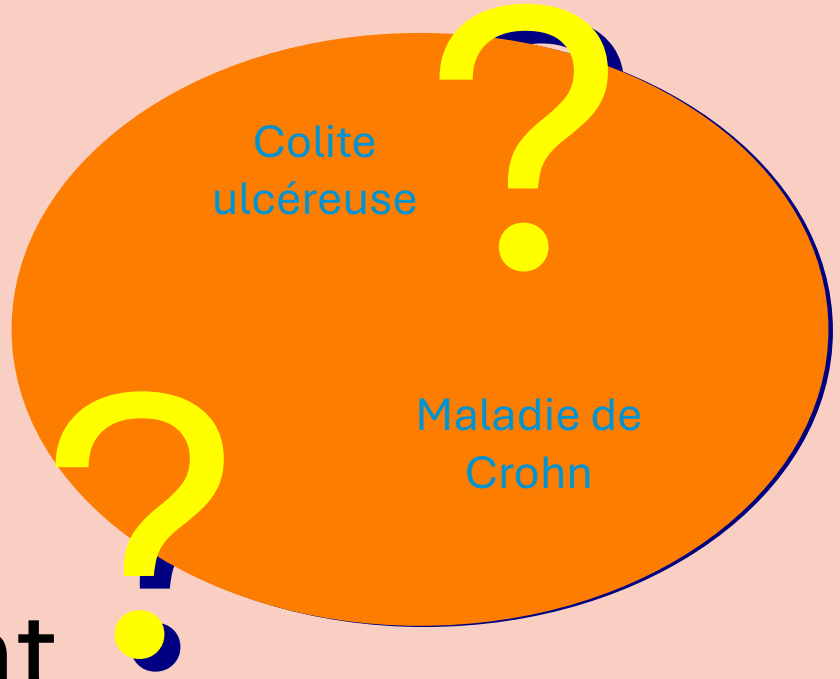


Cosnes J et al. *Clin Gastroenterol Hepatol* 2004.

Courtesy of Prof. Cosnes



onen



ATTEINTE / Comportement

Maladie de Crohn

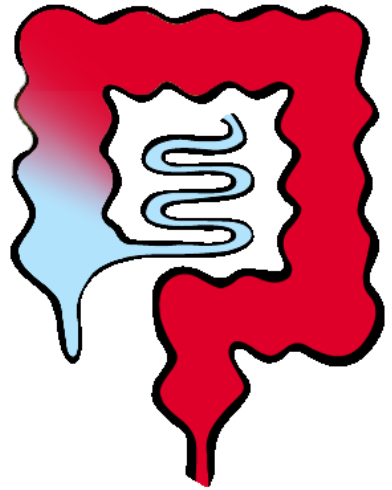
Colite ulcéreuse

Segmentaire ↔ continue

Transmurale ↔ superficielle

Tous le tube digestif ↔ le colon

Diarrhée ↔ saignements rectaux
Douleurs abdominales ténesmes
Carences (malabsorption)

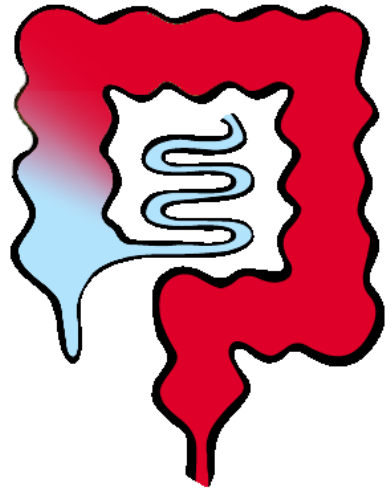


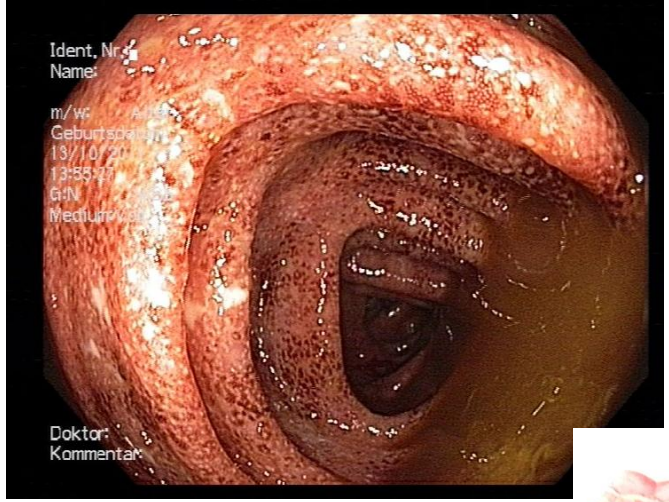
Fistule, abcès, sténose

megacolon toxique, perforation

Maladie de Crohn

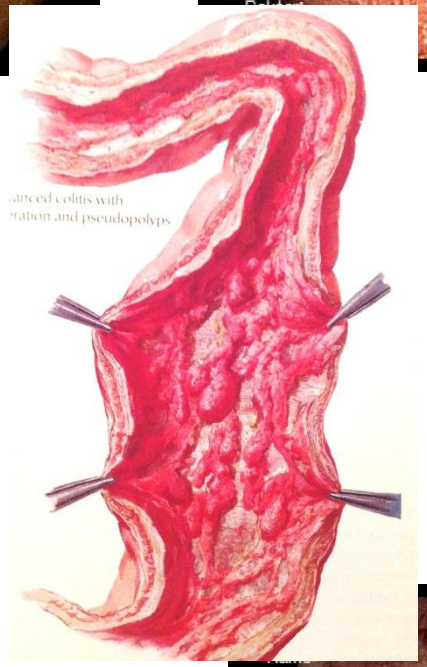
Colite ulcéreuse



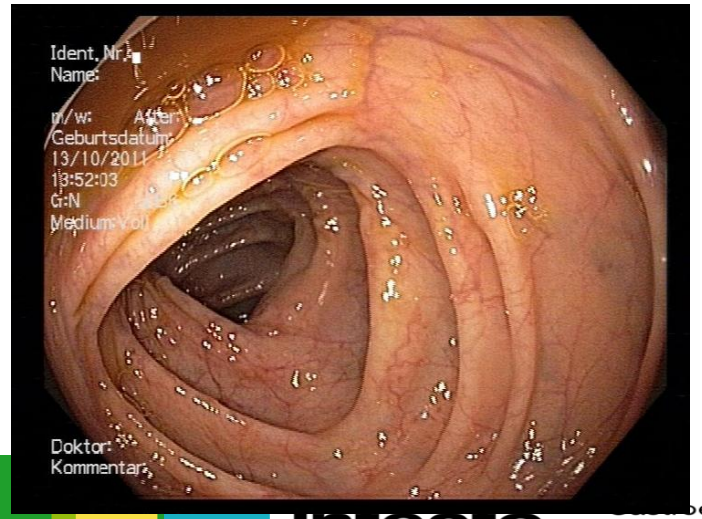


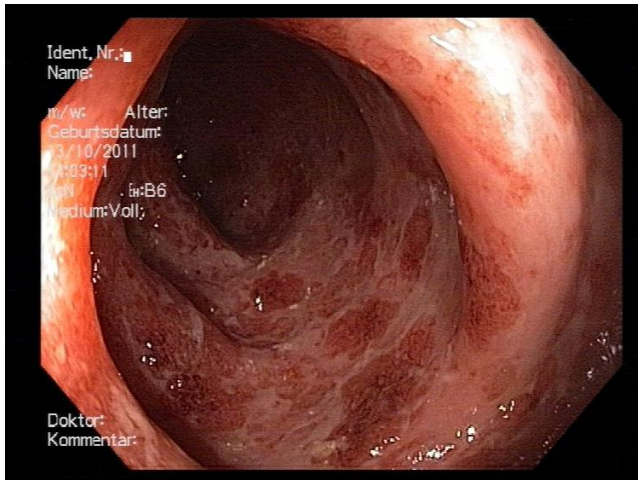
Endoscopie

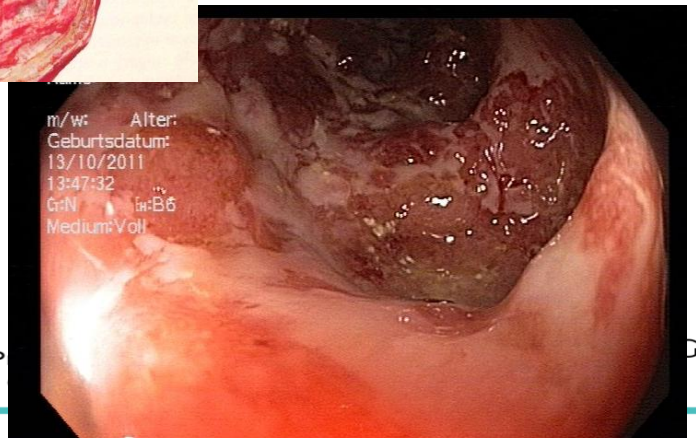
Colite ulcéreuse



ASPECT NORMAL



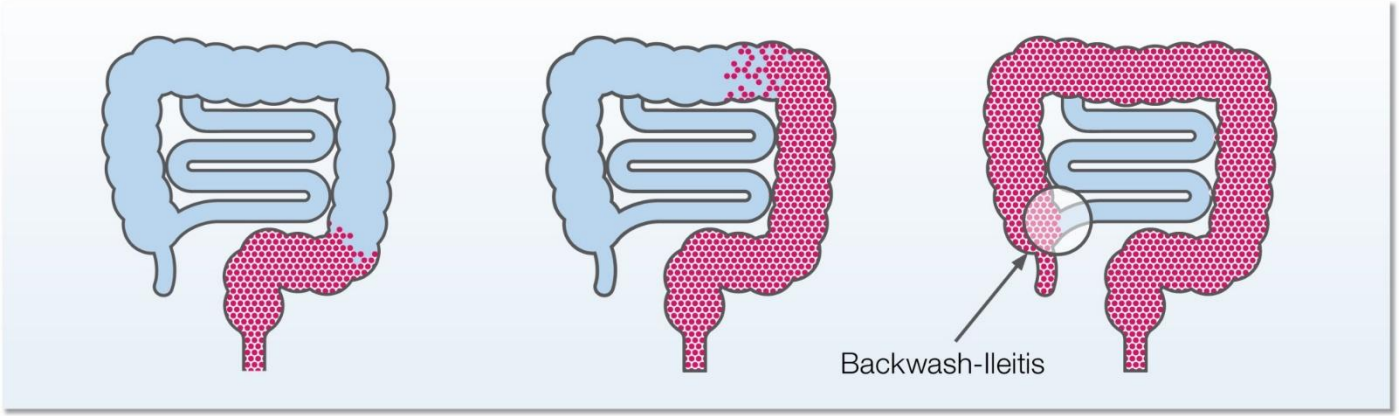




Colitis ulcereuse – atteinte

Classification de Montréal de l'extension de la colite ulcéreuse

E1 **E2** **E3**



•Proctite (40–50 %)

- Douleur à la défécation(Tenesmus)
- Incontinence

Colite gauche (30–40 %)

- Selles sanguinolentes
- constipation

Pancolite (20 %)

- Perte pondérales
- fièvre
- Saignements importants
- Douleurs abdominales

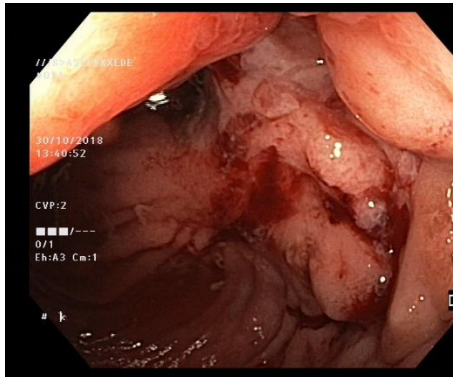
Backwash-Ileitis (10–20 % des Patients avec une Pancolite)

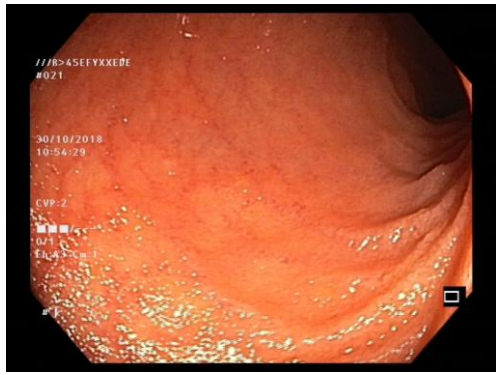
Harrisons Gastroenterologie und Hepatologie, 1. Auflage; Martin Zeitz, Hartmut H.-J. Schmidt, Christian Bojarski (Hrsg.); *ABW Wissenschaftsverlag*, 2011.

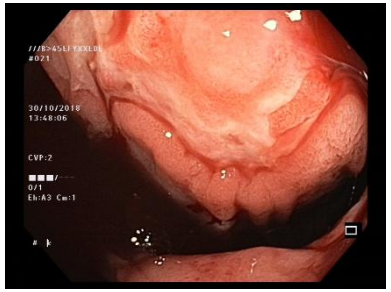
Maladie de Crohn

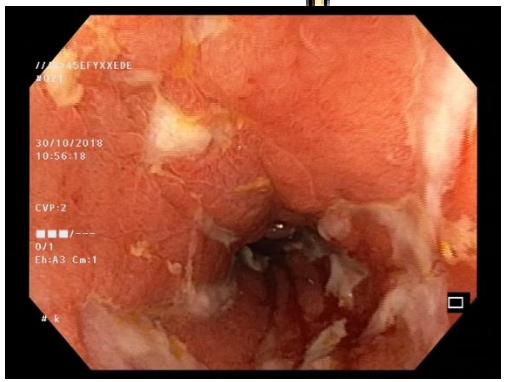
Colite ulcéreuse

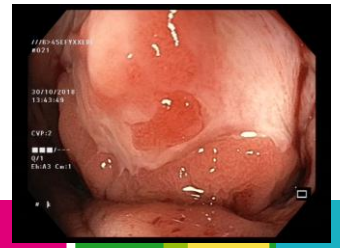
Maladie de Crohn







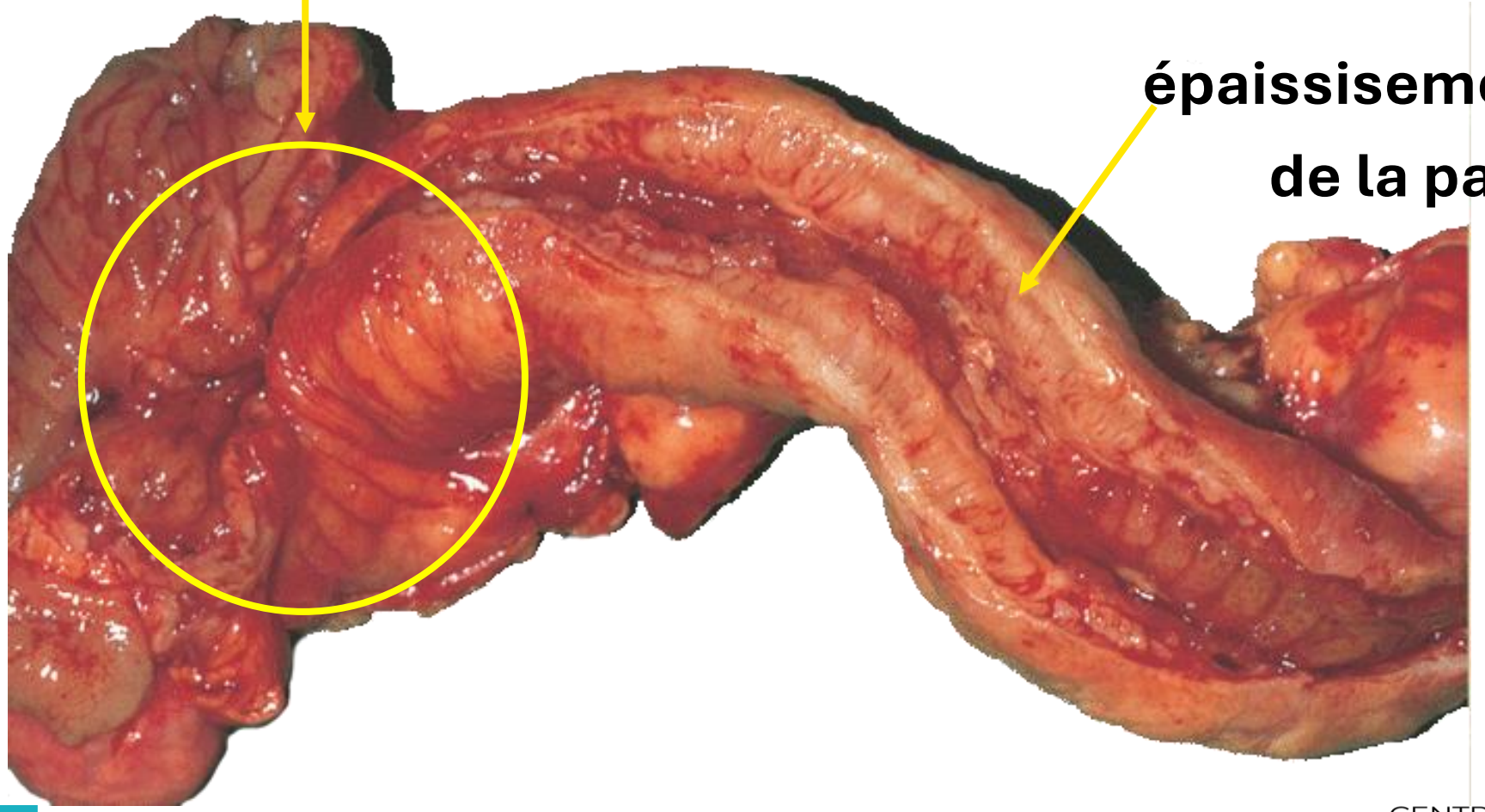




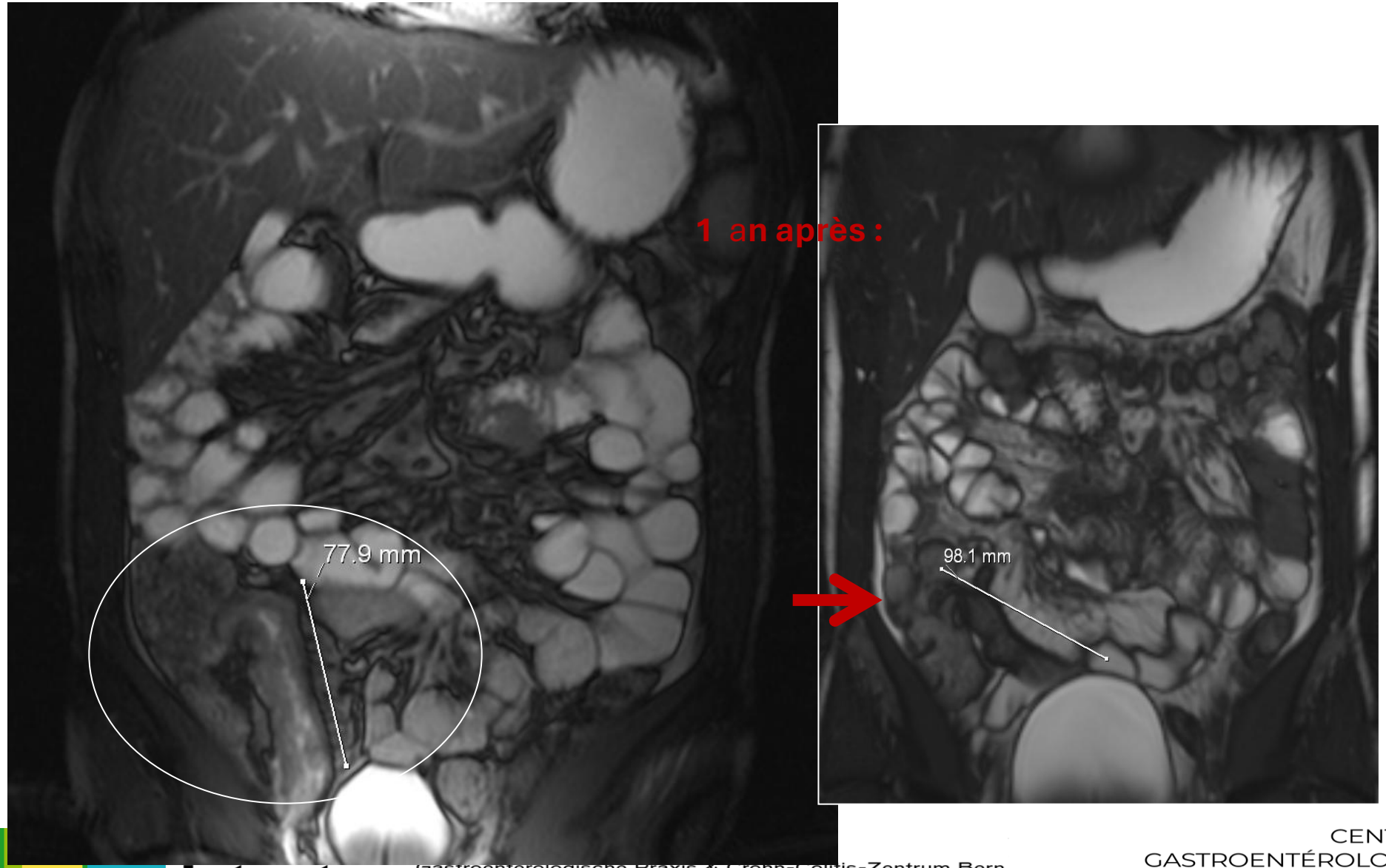
Aspect en «pavés»
„Pflastersteinrelief“

Maladie de Crohn: opération de résection de l‘intestin

Rétrecissement (sténose)

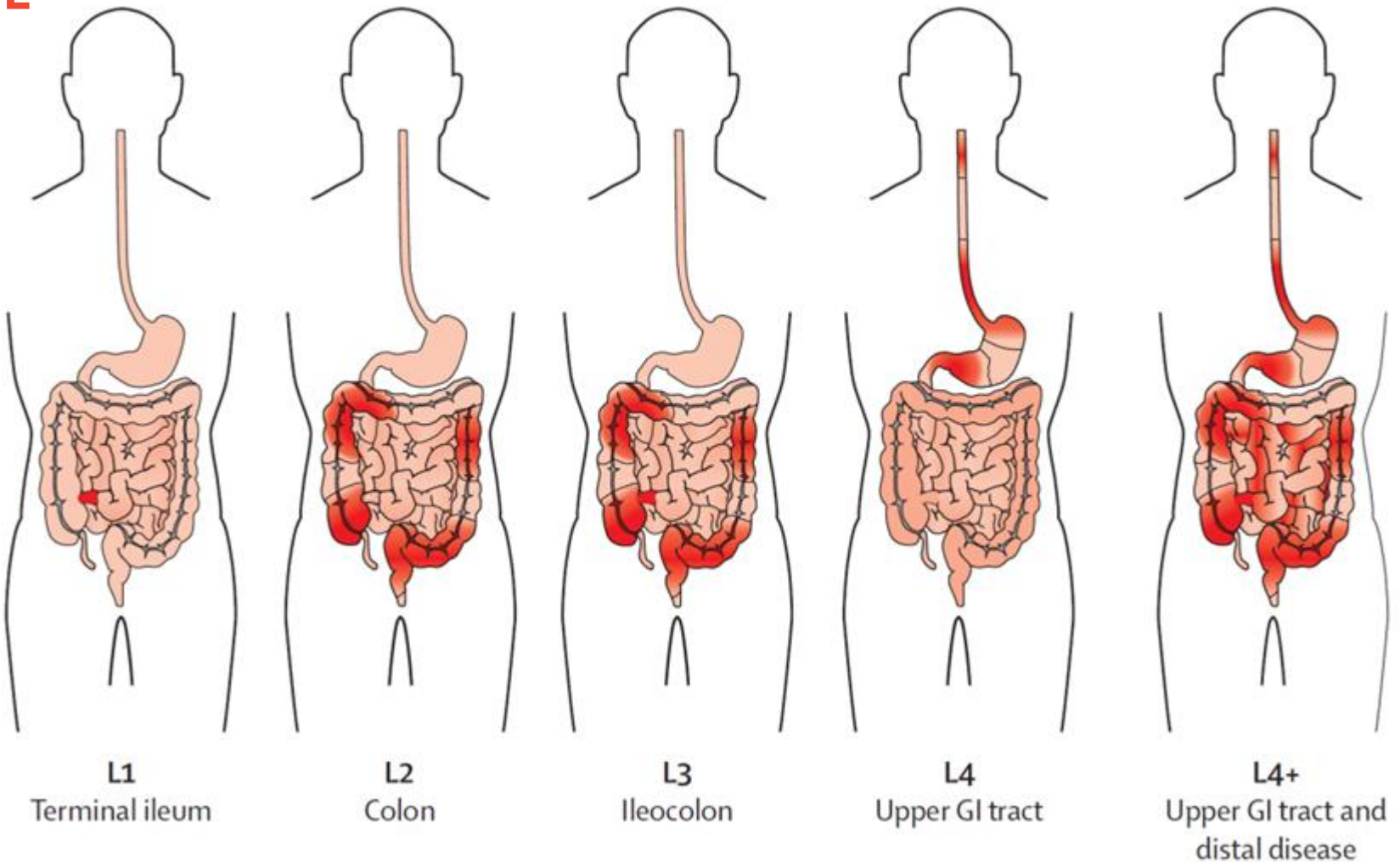


Exemple d‘IRM - Lésion inflammatoire -



La classification de Montréal de l‘extension de la maladie de Crohn

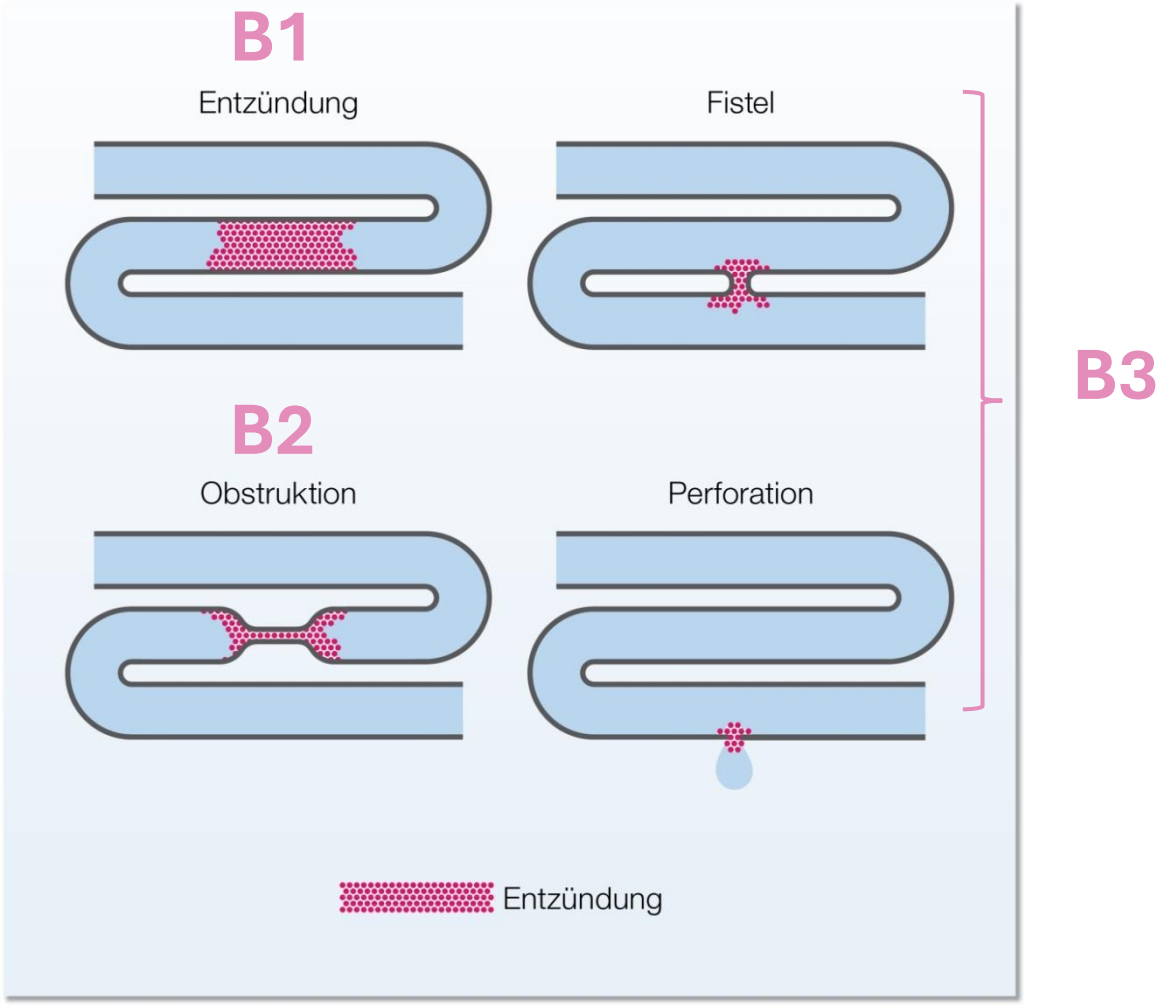
Localisation L



Silverberg MS, et al. Can J Gastroenterol 2005; 19:5–36.
Source : Baumgart *et al.* Lancet 2012

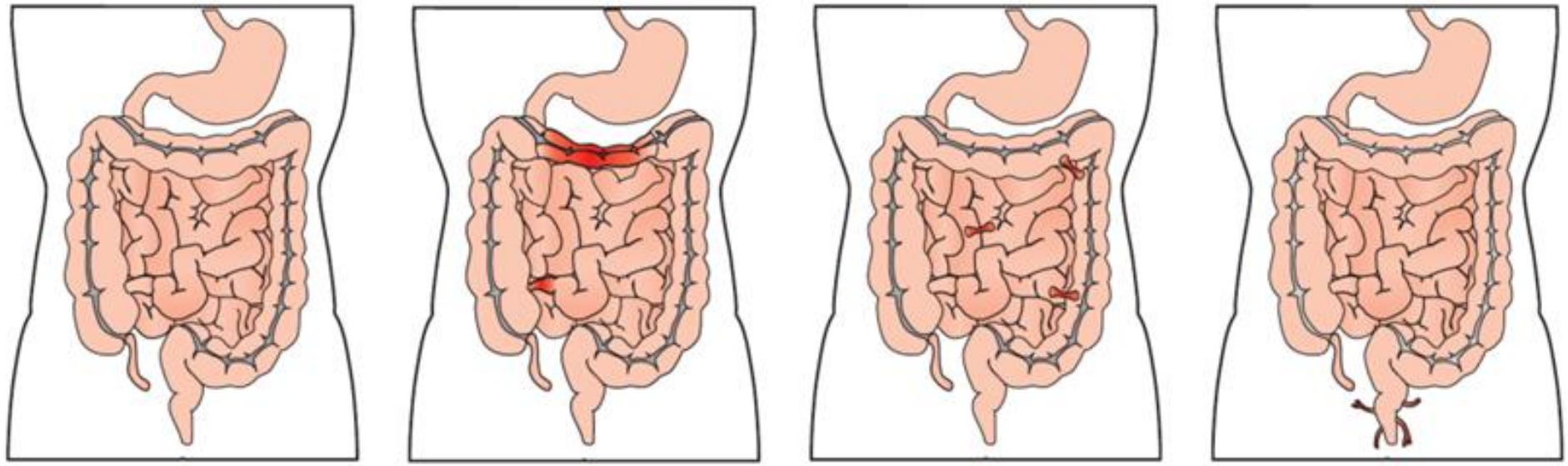
Maladie de Crohn – comportement / Behavior B

- Inflammatoire B1
- Sténosant B2
- Pénétrant B3 avec fistule interne et/ou abcès abdominal (conglomérat „tumoral“ – accolement des anses intestinales)



Harrisons Gastroenterologie und Hepatologie, 1. Auflage; Martin Zeitz, Hartmut H.-J. Schmidt, Christian Bojarski (Hrsg.); *ABW Wissenschaftsverlag*, 2011.

Comportement (behavior) de la maladie de Crohn, selon la classification de Montréal



B1
Without stricture formation non-penetrating

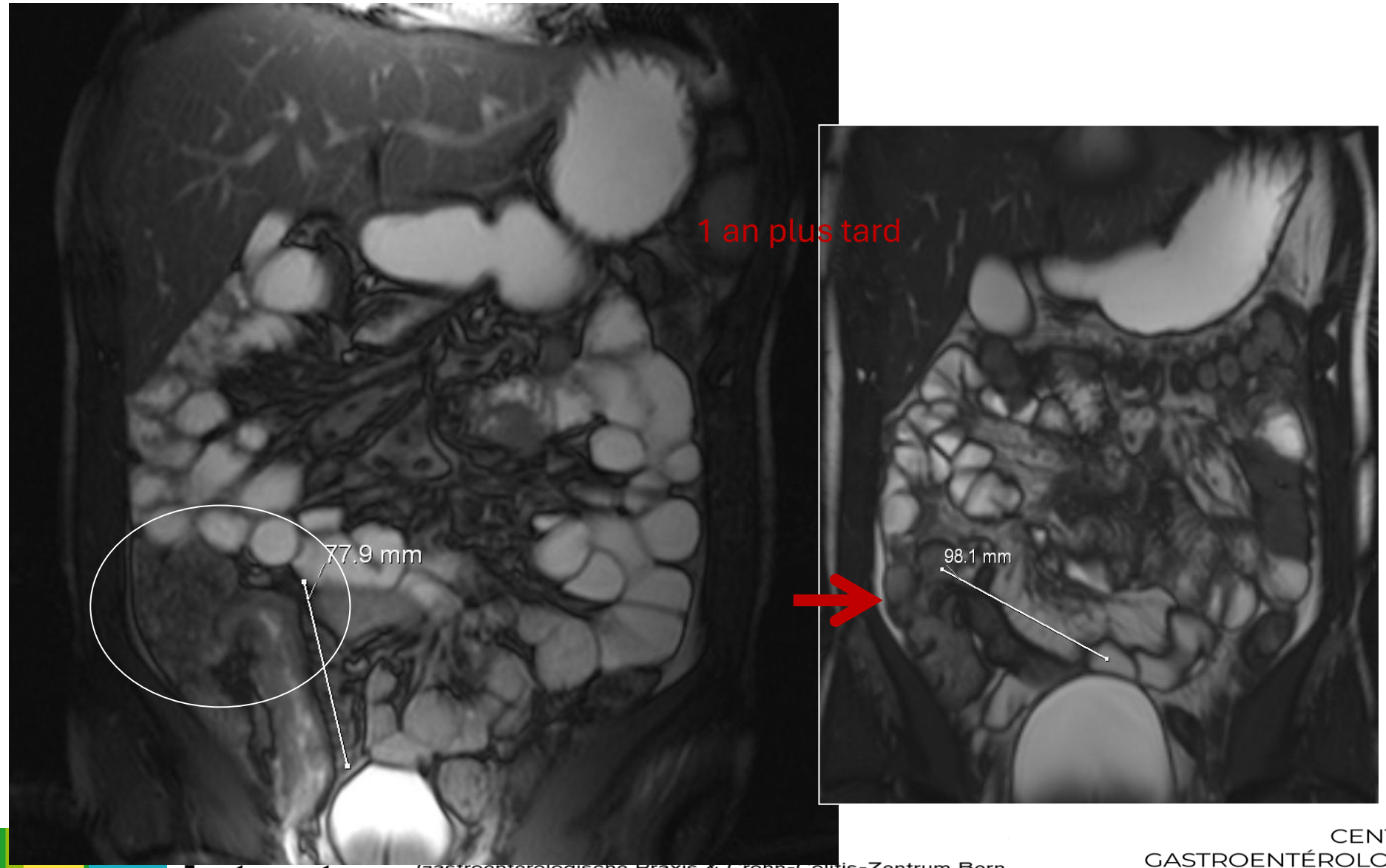
B2
With stricture formation

B3
Internally penetrating

B3p
Perianally penetrating

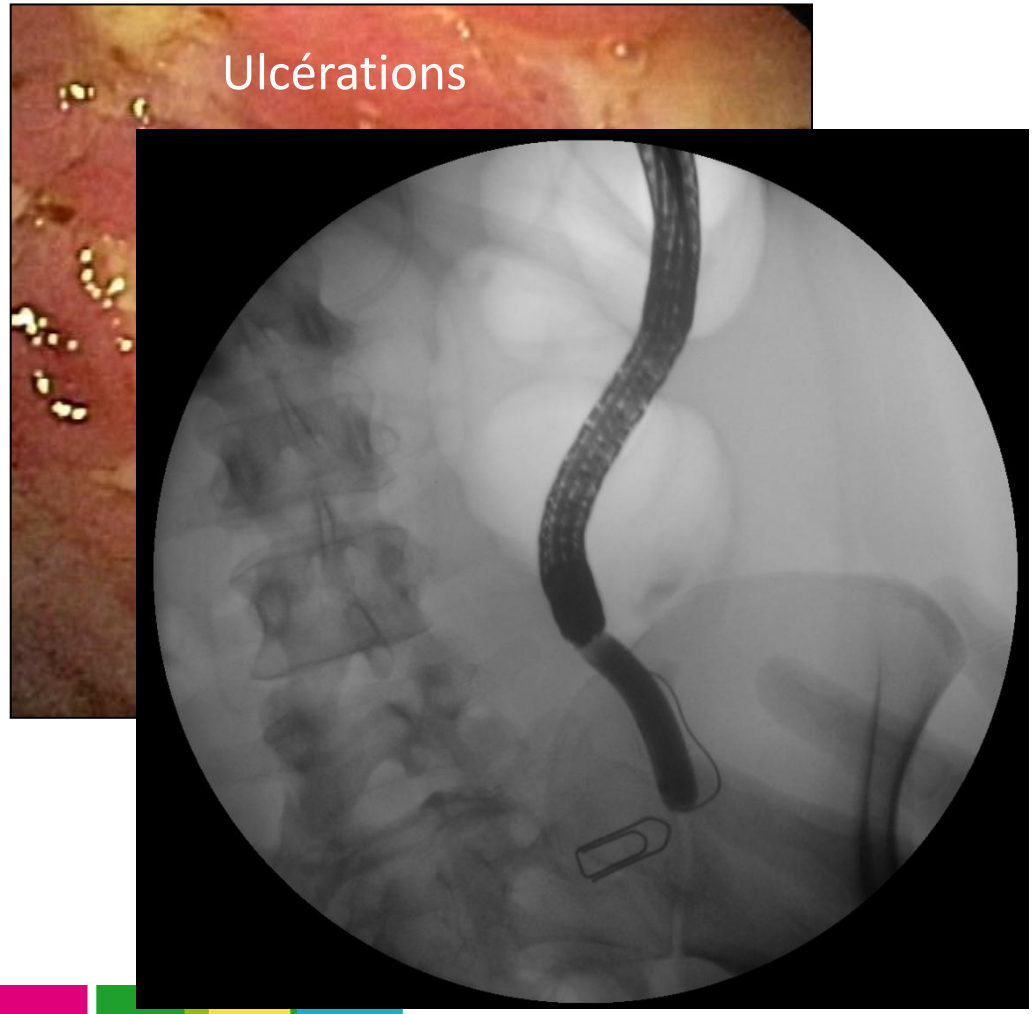
Silverberg MS, et al. Can J Gastroenterol 2005; 19:5–36.
Quelle: Baumgart *et al.* Lancet 2012

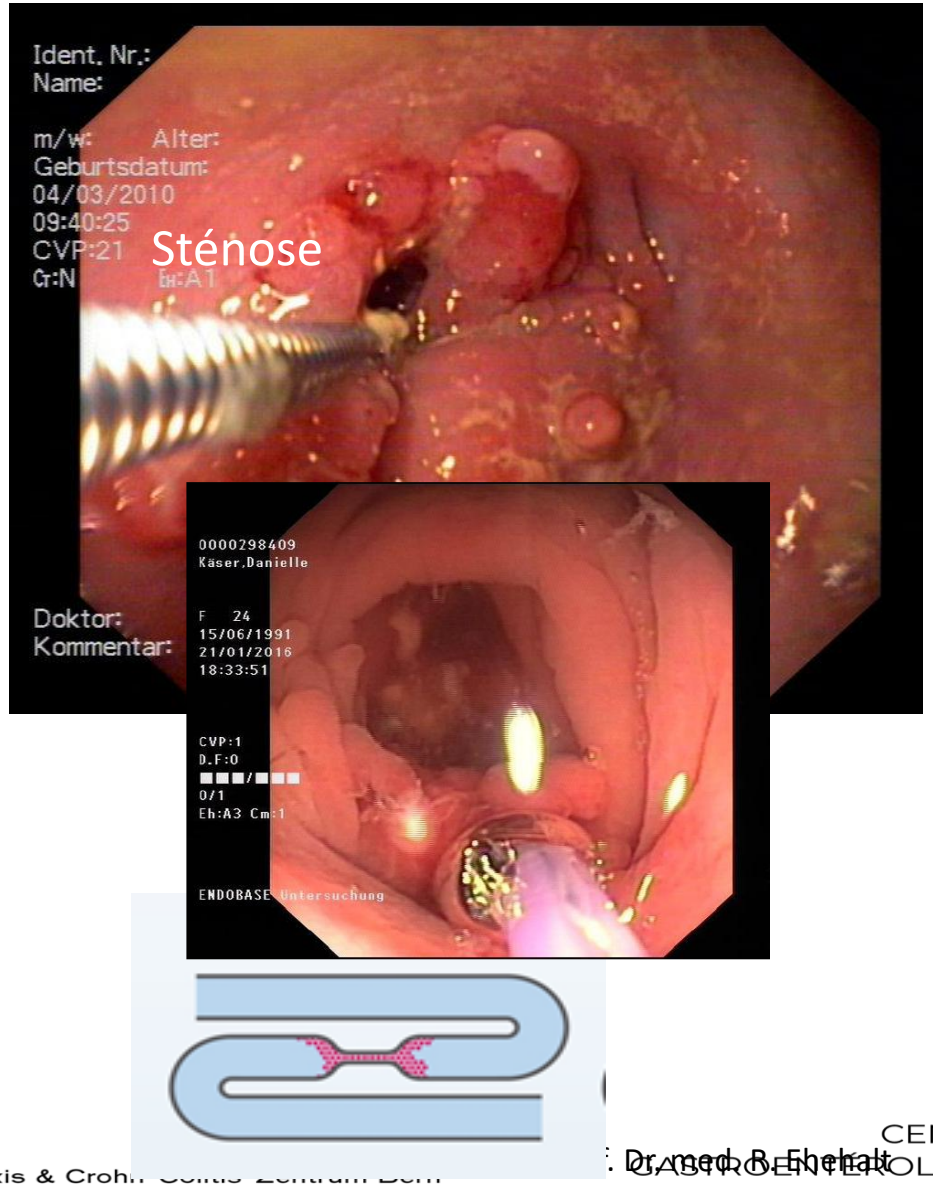
MRI-exemple - Type B1 -



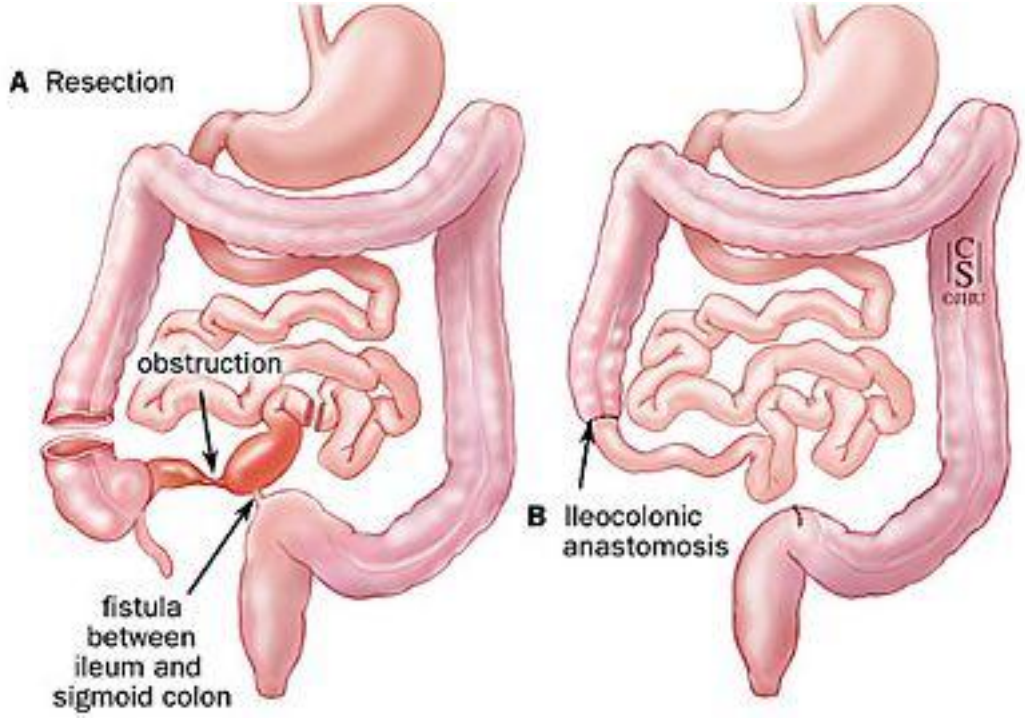
Complication → Stenose

–Type B2 - sténosant



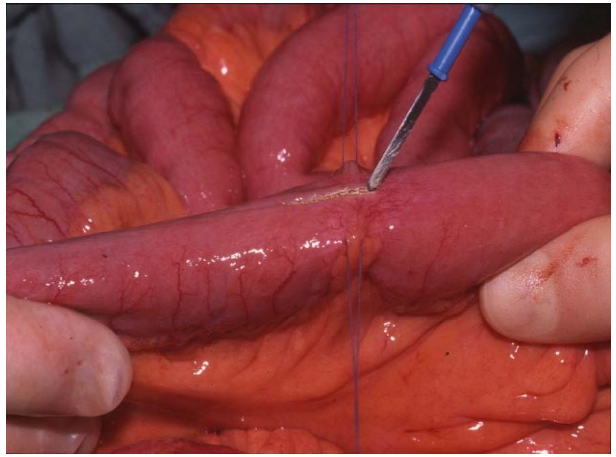


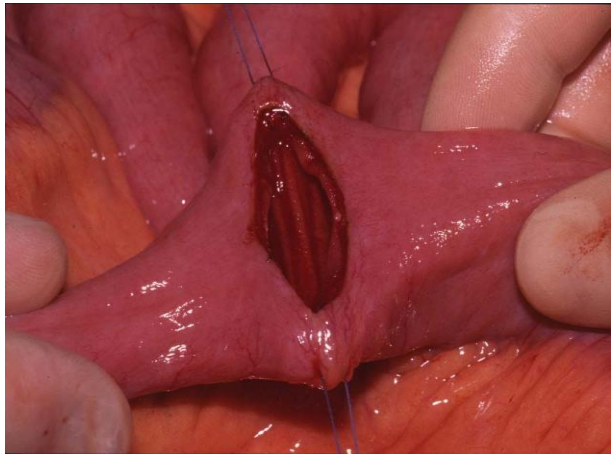
Résection Ileo-colique

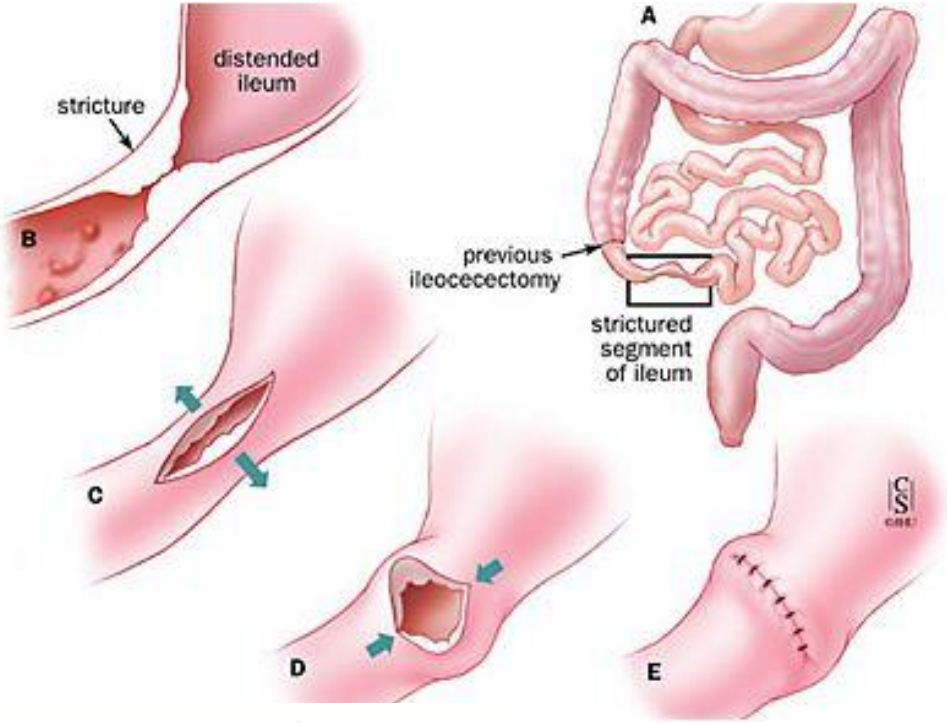


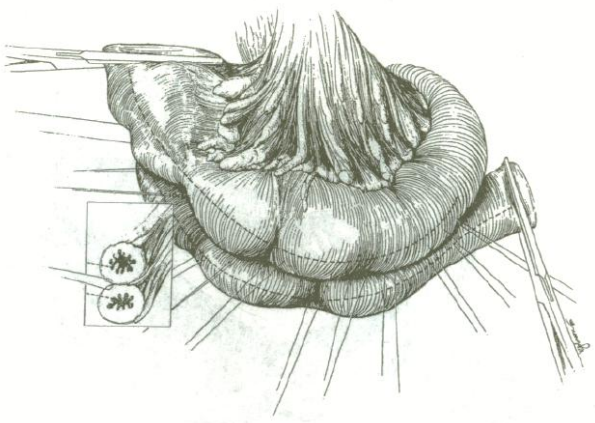
L'approch laparoscopique est "standard of care" !

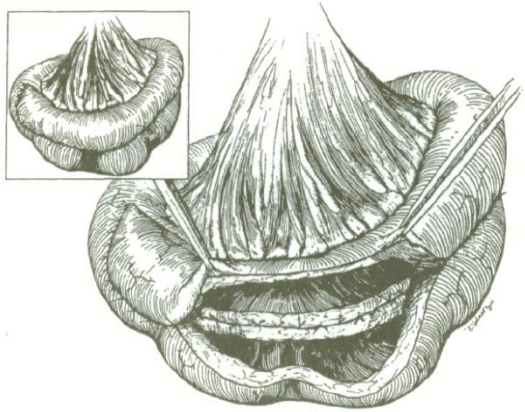
stricturoplasties





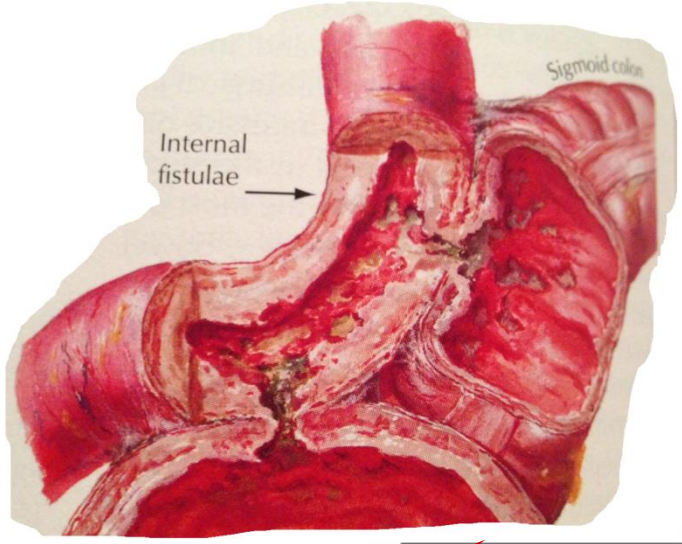


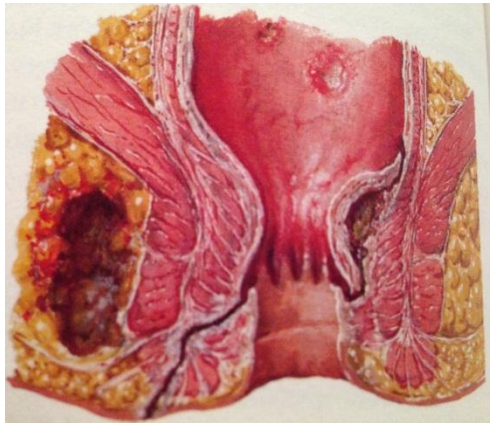


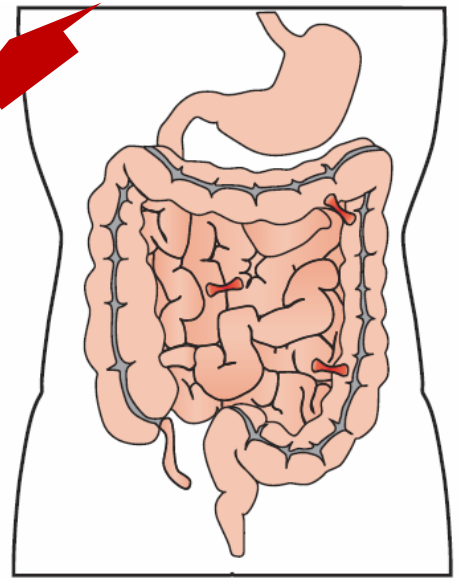


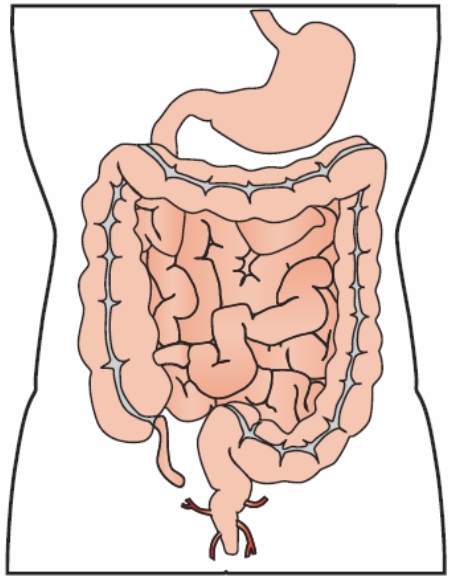
Maladie pénétrante Maladie de Crohn
avec Fistule

- B3 Typ -
- + P Typ -









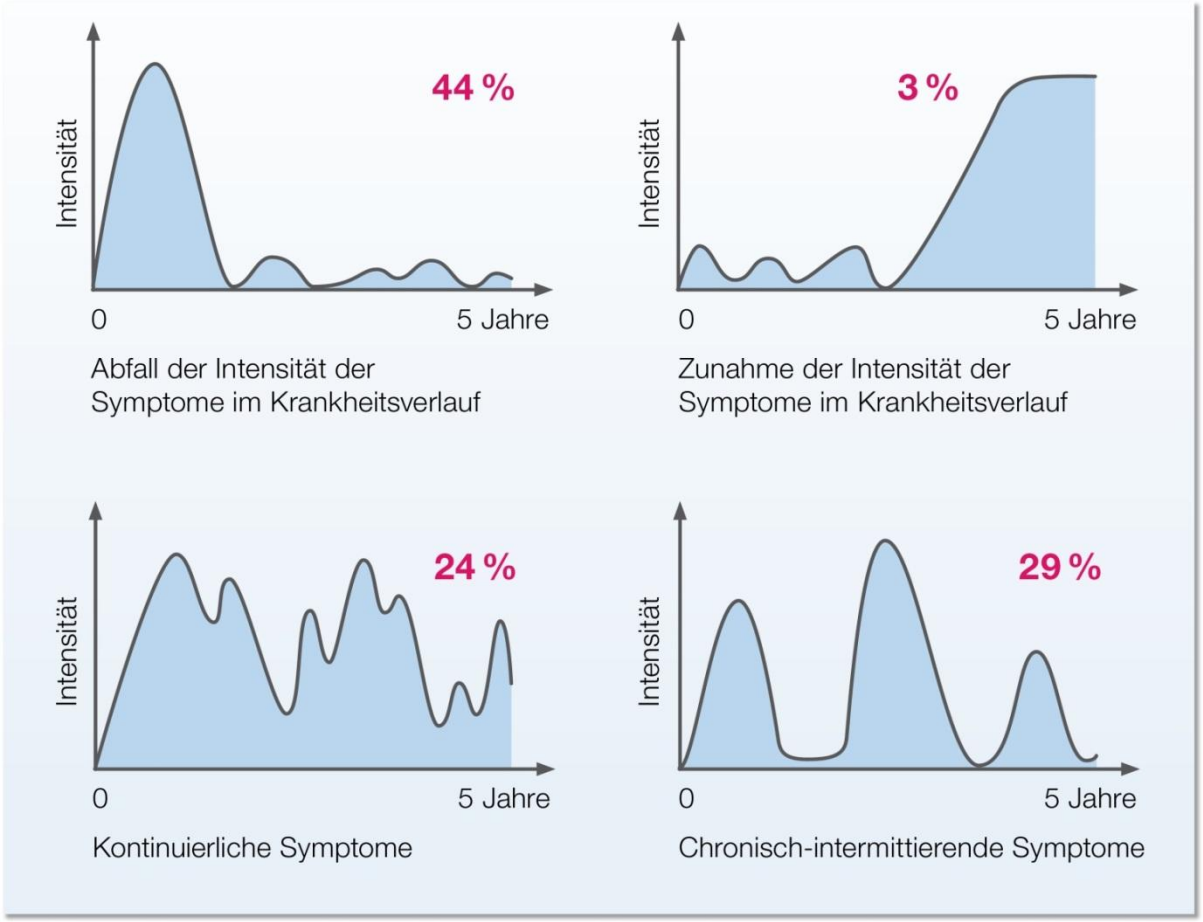
B3
Internally penetrating

B3p
Perianally penetrating

Zentrum Bern

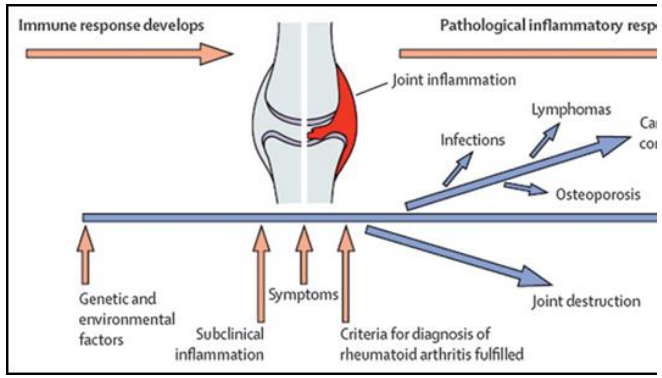
CENTRE
GASTROENTÉROLOGIE
BULLE

Maladie de Crohn – les formes d‘évolution typiques

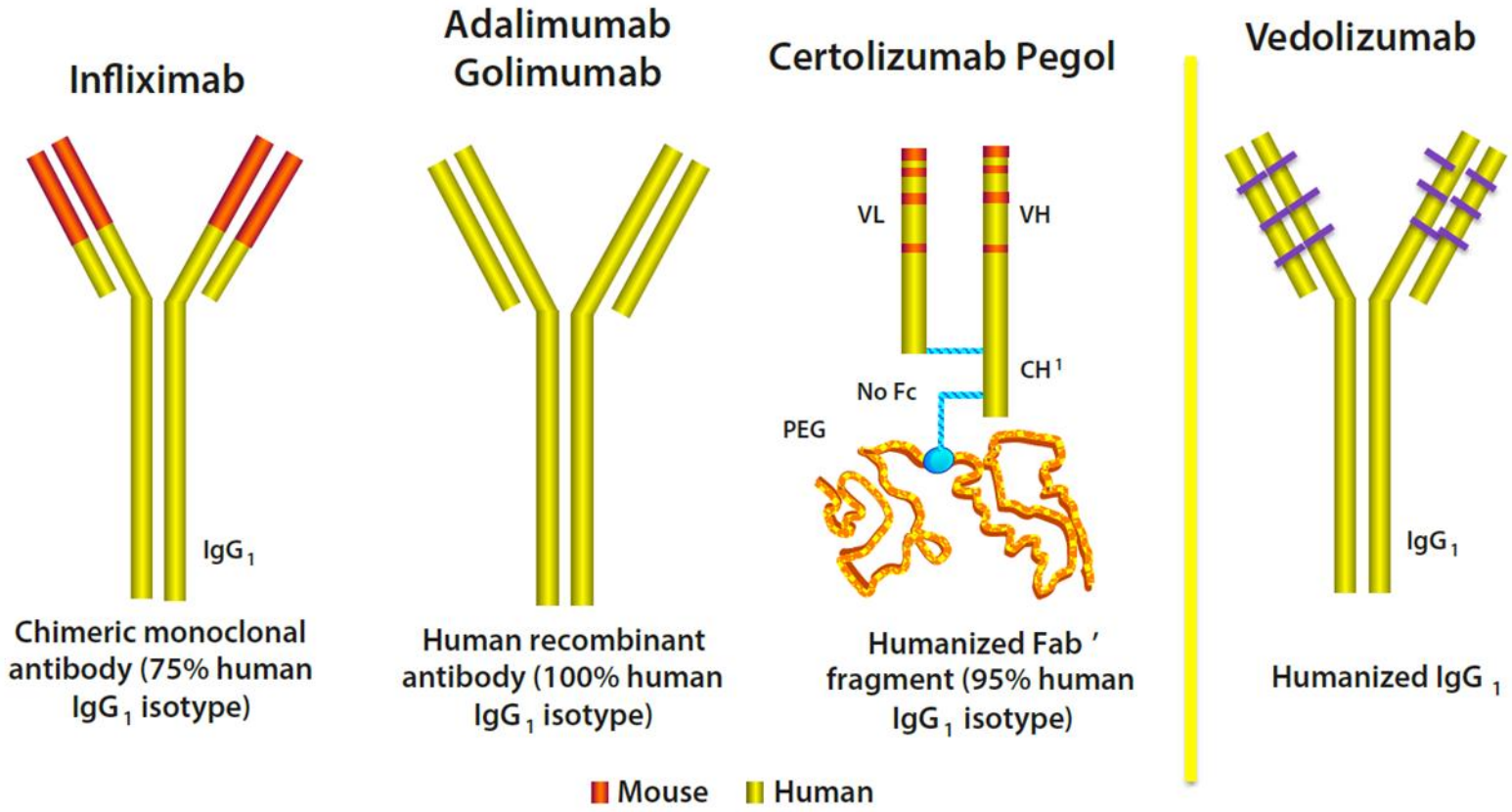


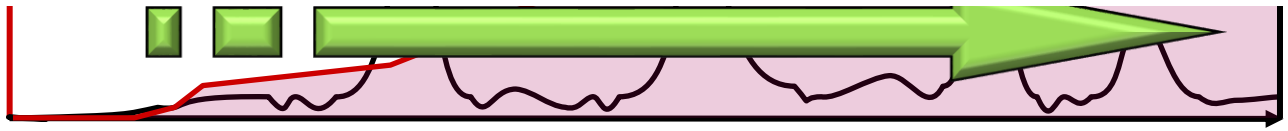
Henriksen M, *et al. Scand J Gastroenterol* 2007; 42: 602–610.

Progression des lés



Atteinte intestinal:
„Lémann score“





Début de la Maladie — Diagnostic — phase primaire de la maladie

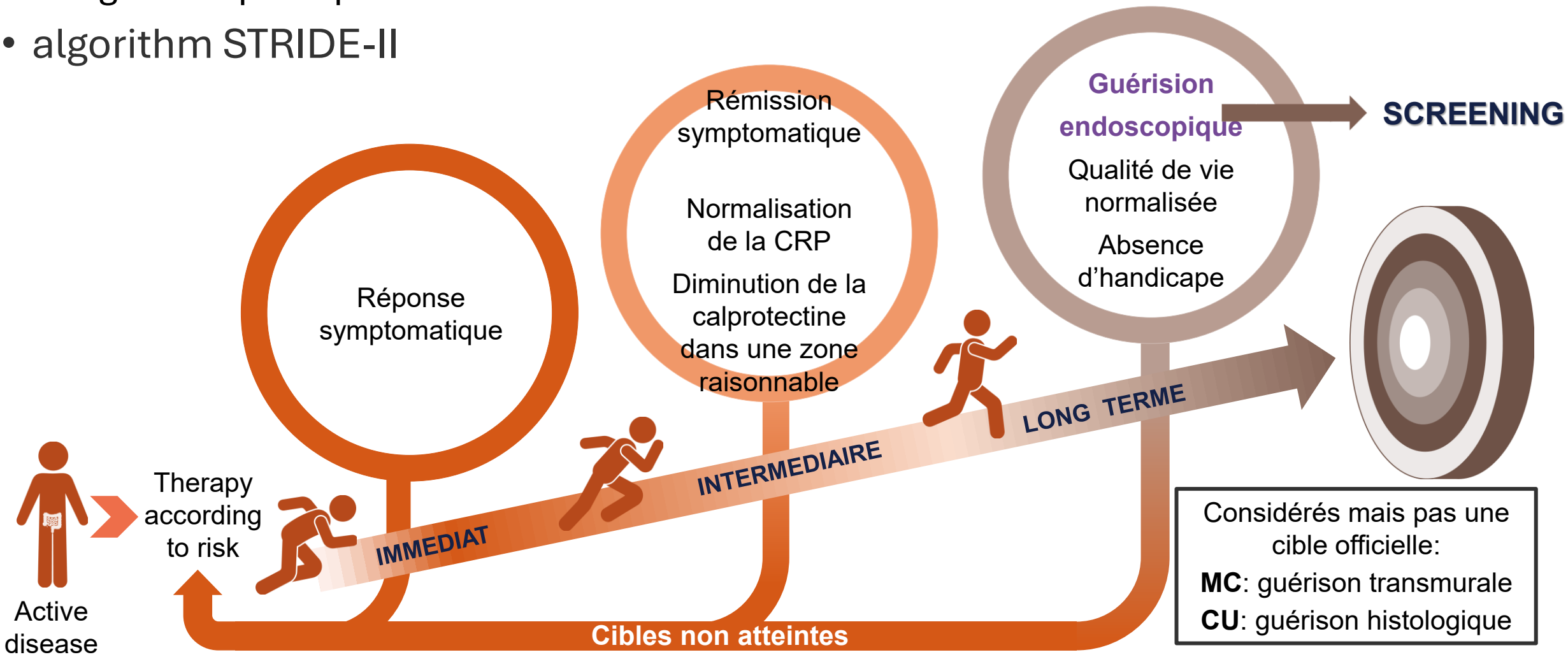
Préclinique — clinique

Pariente B, *et al. Inflamm Bowel* 2011;17:1415–1422.

CDAI: Crohn's Disease Activity Index; CDEIS: Crohn's Disease Endoscopic Index of Severity; CRP: C-reaktives Protein

Stratégie thérapeutiques

- algorithm STRIDE-II



CRP, C-reactive protein; FC, faecal calprotectin; QoL, quality of life; STRIDE, Selecting Therapeutic Targets in Inflammatory Bowel Disease. Turner D, et al. *Gastroenterology*. 2021;160:1570–83.

A PROPOS des **complications** – comment les prévenir !! -

- **Bilan martial et vitaminique**

* 40% de carence vitaminique dans la maladie de Crohn

	Bilan martial	Vitamines*	Calprotectine
M. Crohn - en Rémission - active	6 -12 MO 3 MO	6 -12 MO 3 - 6 MO	6 MO 2 - 3 MO
Colite ulcéreuse - en rémission - active	6 - 12 MO 3 MO	12 MO	4 MO 1 - 2 MO

ORIGINAL RESEARCH CLINIC

The Relevance of Vitamin and Iron Deficiency in Patients with Inflammatory Bowel Diseases in Patients of the Swiss IBD Cohort

Matiar Madanchi, Stefania Fagagnini, MD,*,† Nicolas Fournier, PhD,‡ Luc Biedermann, MD,* Jonas Zeitz, MD,* Edouard Battegay, MD,† Lukas Zimmerli, MD,§ Stephan R. Vavricka, MD,*,¶ Gerhard Rogler, MD, PhD,*,‖ and Michael Scharl, MD*,‖ on behalf of the Swiss IBD Cohort Study Group***

enterologische Praxis & Crohn-Colitis·
Fribourgeois de Gastroentérologie

CONCLUSIONS

In summary, our data support that folate, vitamin B12, and iron levels in patients suffering from IBD regardless of the subtype should be monitored routinely. In the case of vitamin deficiency, a substitution therapy needs to be established, thus preventing a complicated disease course.

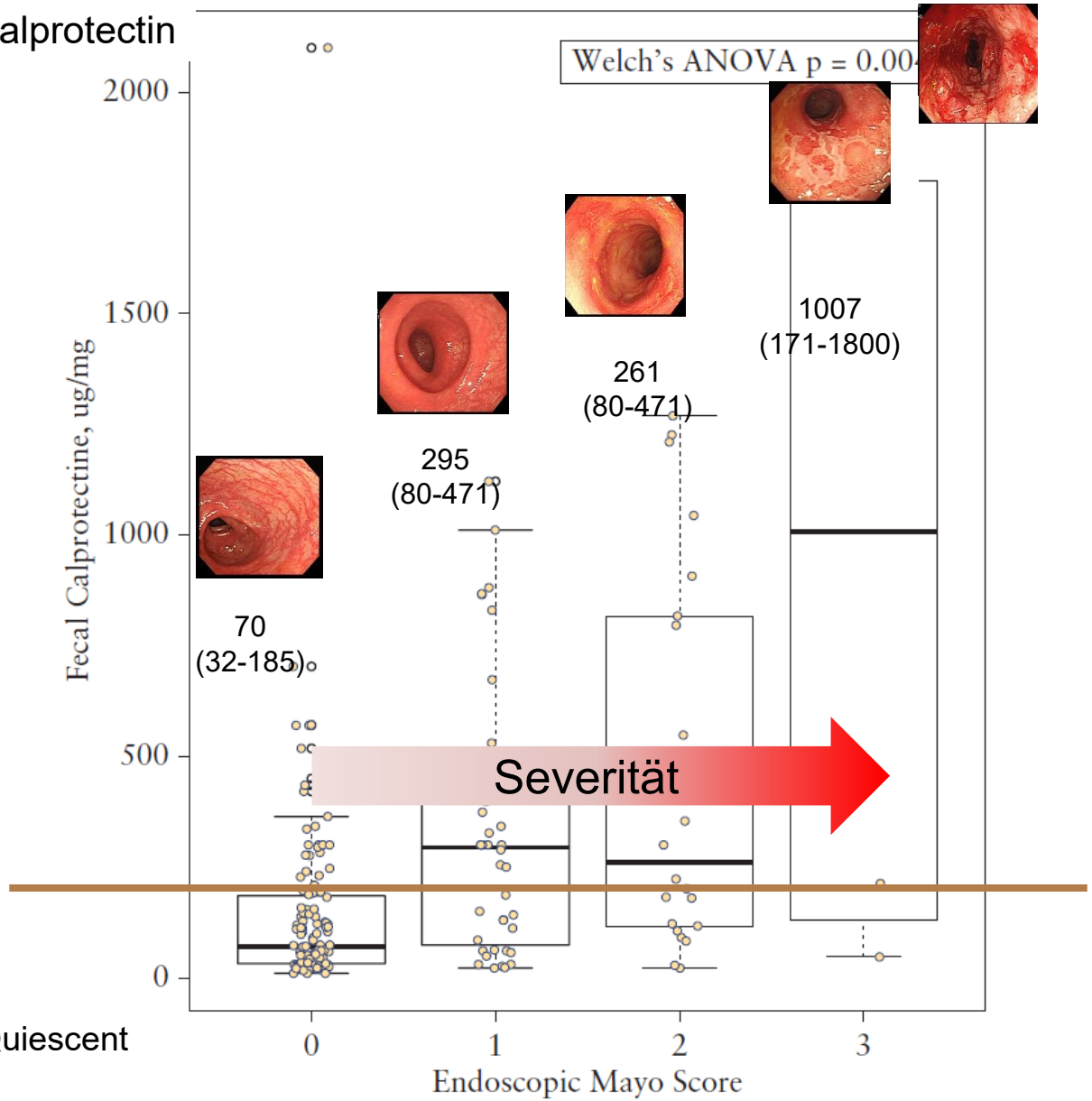
Correlation calprotectine - Endoscopie

- Cutoffs

(1) Normal vs. MICIs > 50 mg/Kg

(2) Activité : > 250 mg/Kg

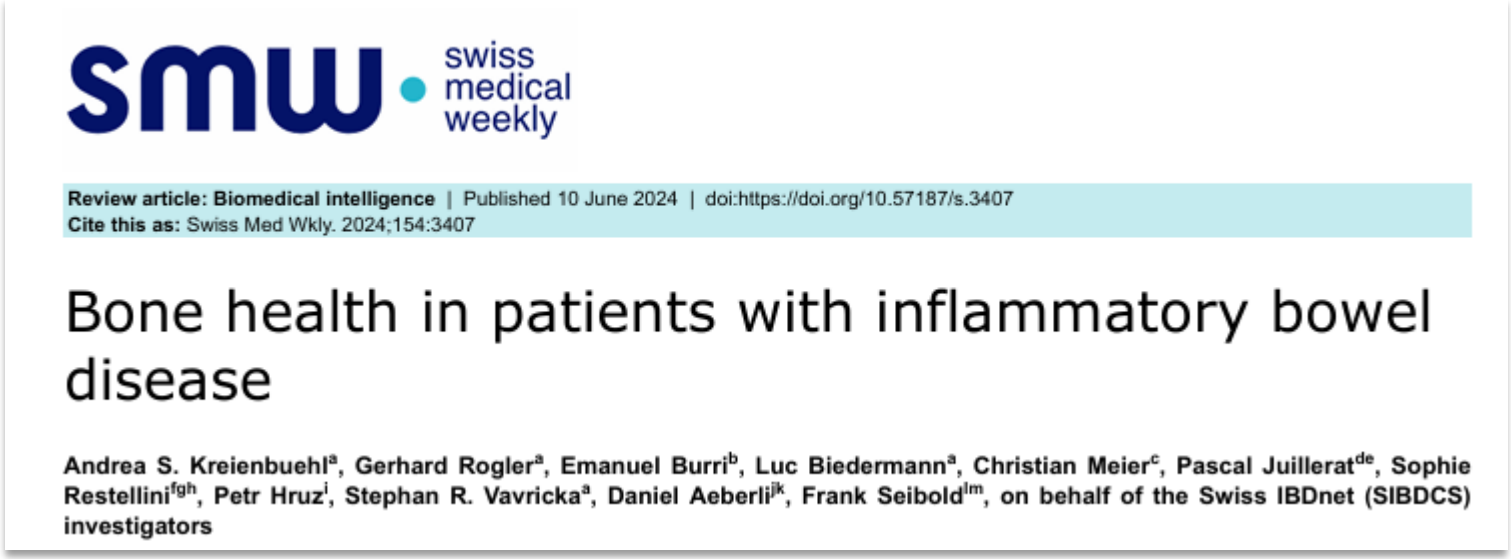
(3) **Rémission (endo + Histo) < 150**



N= 185 CU Patienten bei Mc Gill University, Montréal

Faecal Calprotectin Predicts Endoscopic and Histological Activity in Clinically Quiescent Ulcerative Colitis Lara Hart, *et al. Journal of Crohn's and Colitis*, 2020, 46–52

VITAMINE D / OSTEOPOROSE



smw swiss medical weekly

Review article: Biomedical intelligence | Published 10 June 2024 | doi:https://doi.org/10.57187/s.3407
Cite this as: Swiss Med Wkly. 2024;154:3407

Bone health in patients with inflammatory bowel disease

Andrea S. Kreienbuehl[a], Gerhard Rogler[a], Emanuel Burri[b], Luc Biedermann[a], Christian Meier[c], Pascal Juillerat[de], Sophie Restellini[fgh], Petr Hruz[i], Stephan R. Vavricka[a], Daniel Aeberli[jk], Frank Seibold[lm], on behalf of the Swiss IBDnet (SIBDCS) investigators

Table 1 :
The World Health Organization's definition of osteoporosis based on DXA measurement [51].

Interpretation	T-score
Normal	–1.0 and higher
Osteopenia	–1.0 to –2.5
Osteoporosis	–2.5 and lower
Severe osteoporosis	–2.5 and lower with one or more fragility fractures

Osteopenia and osteoporosis are often underestimated, particularly in patients with IBD. In the Swiss IBD Cohort Study (SIBDCS), 19% of patients had low bone mineral density [1]

Other studies : Osteopenia 35-40 % and Osteoporosis 15%

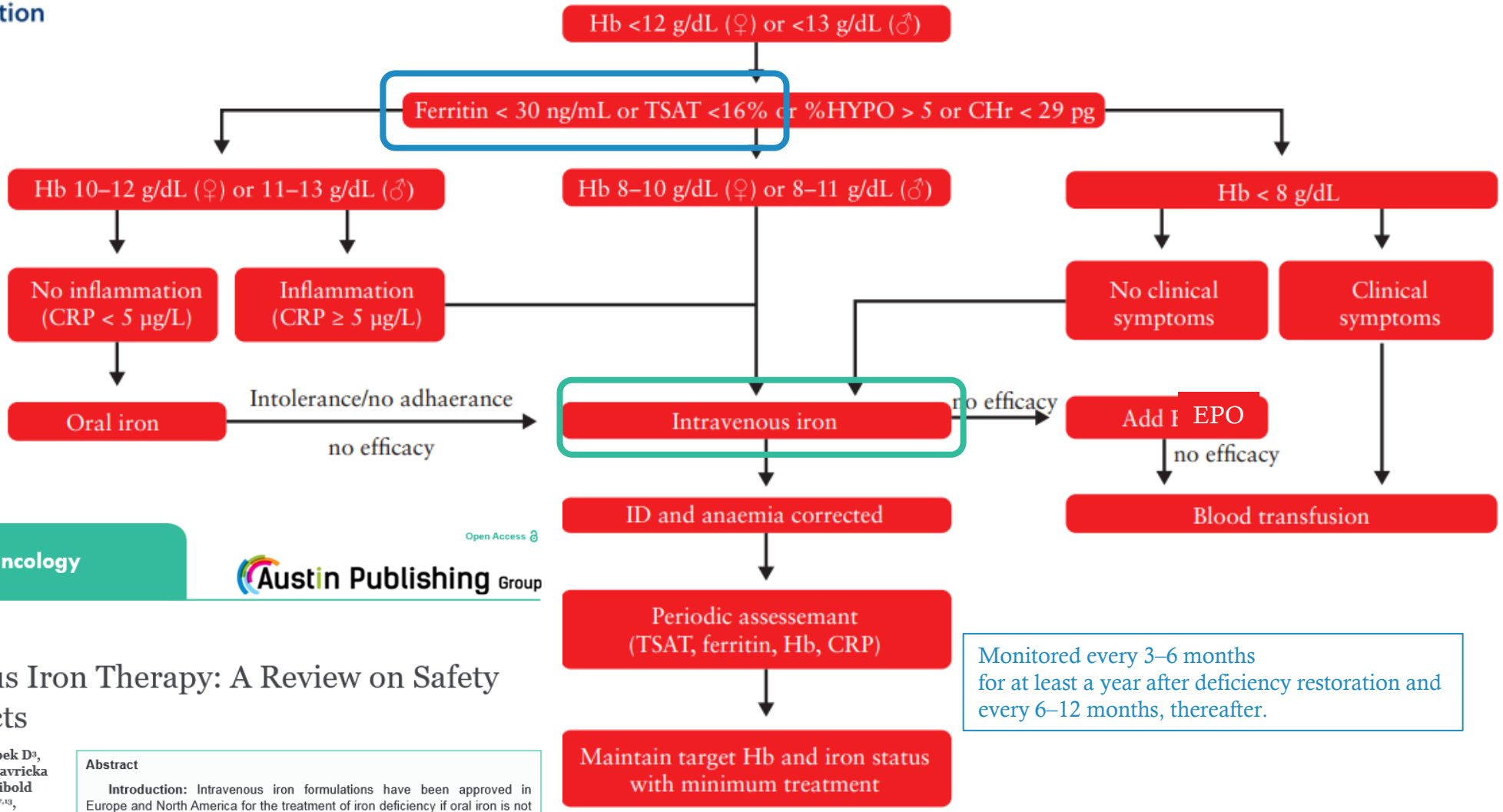
1 Schüle S, Rossel JB, Frey D, Biedermann L, Scharl M, Zeitz J, et al.; Swiss IBD cohort study. Prediction of low bone mineral density in patients with inflammatory bowel diseases. United European Gastroenterol J. 2016 Oct;4(5):669–76.

Bernstein M, Irwin S, Greenberg GR. Maintenance infliximab treatment is associated with improved bone mineral density in Crohn's disease. The American journal of gastroenterology 2005;100:2031-5

Reinshagen M. Osteoporosis in inflammatory bowel disease. Journal of Crohn's & colitis 2008;2:202-7.

Anemia management in IBD patients

European Crohn's and Colitis Organisation



Monitored every 3–6 months
for at least a year after deficiency restoration and every 6–12 months, thereafter.

Annals of Hematology & Oncology

Open Access

Austin Publishing Group

Review Article

Modern Intravenous Iron Therapy: A Review on Safety and Practical Aspects

Juillerat P[1,13*], Angelillo-Scherrer A[2], Surbek D[3], Restellini S[4], Biedermann L[5], Rogler G[5], Vavricka SR[5,6], Schoepfer A[7], Burri E[8], Degen L[9], Seibold F[10,11], Mottet C[12], Maillard MH[7], Michetti P[7,13], Battegay E[14#], and Stein J[15,16#]

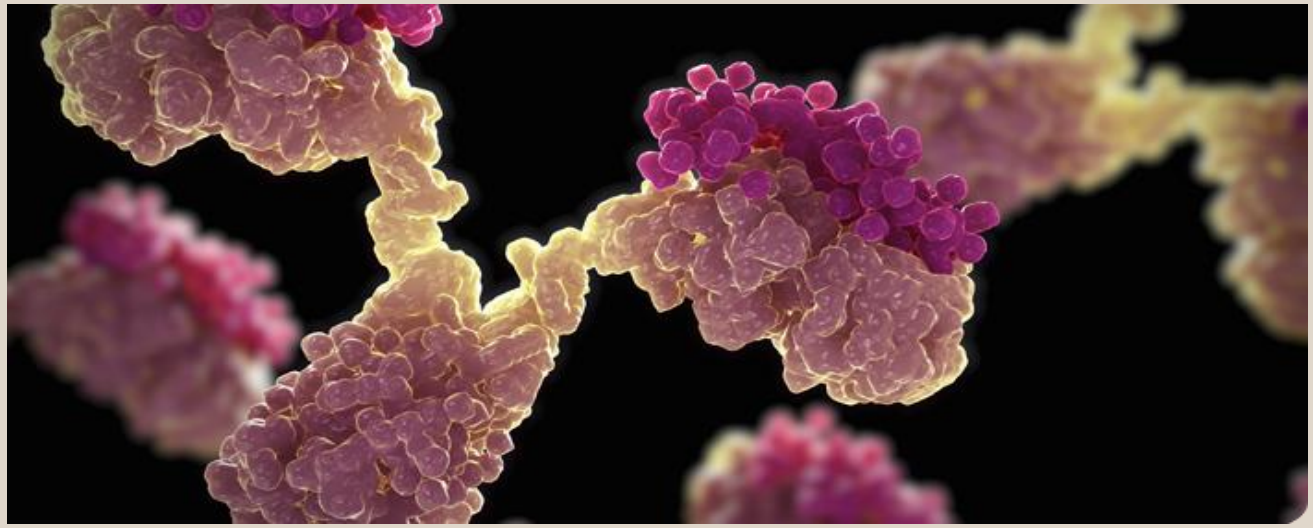
Abstract

Introduction: Intravenous iron formulations have been approved in Europe and North America for the treatment of iron deficiency if oral iron is not tolerated or not efficacious. Recently-developed intravenous iron formulations

Figure 2. Workup for the management of iron deficiency anaemia in patients with IBD [adapted from Martin *et al.* 2017[83]]. CRP, C-reactive protein; ESA,

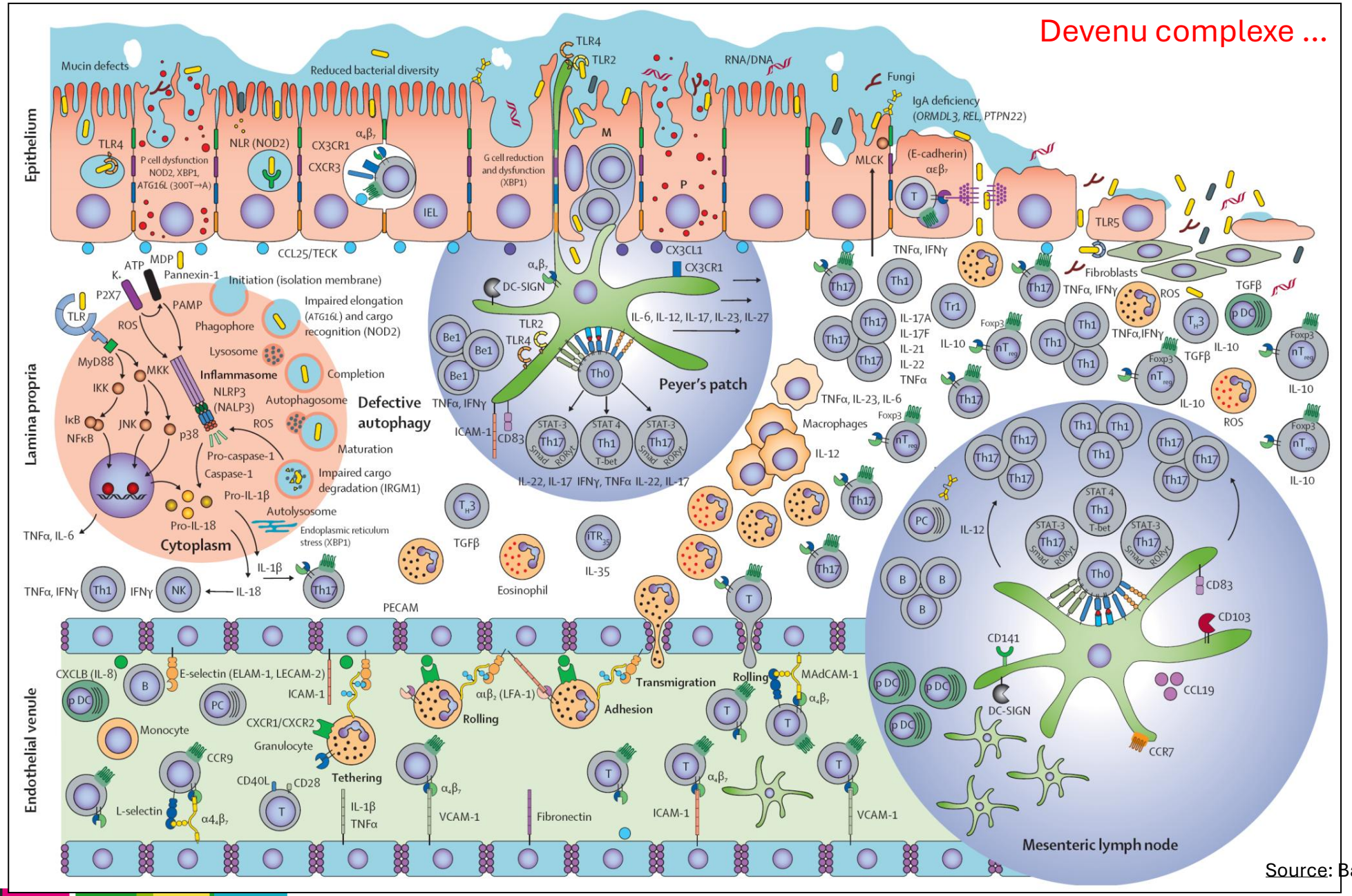
Hannah Gordon, …, Pascal Juillerat, …, Torsten Kucharzik, ECCO Guidelines on Extraintestinal Manifestations in Inflammatory Bowel Disease, Journal of Crohn's and Colitis, Volume 18, Issue 1, January 2024, Pages 1–37, https://doi.org/10.1093/ecco-jcc/jjad108

THERAPIE



Comment traiter

Devenu complexe ...



Source: Baumgart *et al.* Lancet 2012

Interventions thérapeutiques dans les MICIs à travers le temps

1950s
Steroide

1970s
Thiopurines

1990s
Methotrexate

2000s
Anti-TNFs

2015
Anti-Integrines

2016

2017
Ustekinumab

2019
Tofacitinib

2019

2022
Ozanimod

2023
Risankizumab

USTE

TOFA

OZA

RISA

1998

IFX

2006

2007

ADA

2012

***2024* :**
JAK -1- inhibitors
Add. IL-23 inhib.
S1P modulators
TL1A inhibitors

Mulder DJ, et al. J Crohns Colitis. 2014;8:341-8; Park SC, et al. Gut Liver. 2015;9:18-27.

Intesto

Gastroenterologische Praxis & Crohn-Colitis-Zentrum Bern
Centre Fribourgeois de Gastroentérologie

Morbus Crohn

Thérapies de MICIs, durant les 5 dernières années.

2020
Ustekinumab

2021
LIR!C trial

2023
Risankizumab

2024
Upadacitinib

2025
Etrasimod

2025
Guselkumab

2025
Mirikizumab

- Obefazimod
- Tulisokibart
- Anti-IL1
- TLP2 inhib.
- IL-36 inhib
- TYK 2 .....

2025
Risankizumab

2021
Tofacitinib

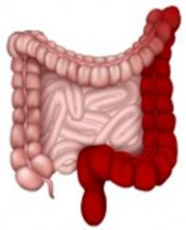
2022
Ozanimod

2024
Upadacitinib

2024
Mirikizumab

2024
Etrasimod

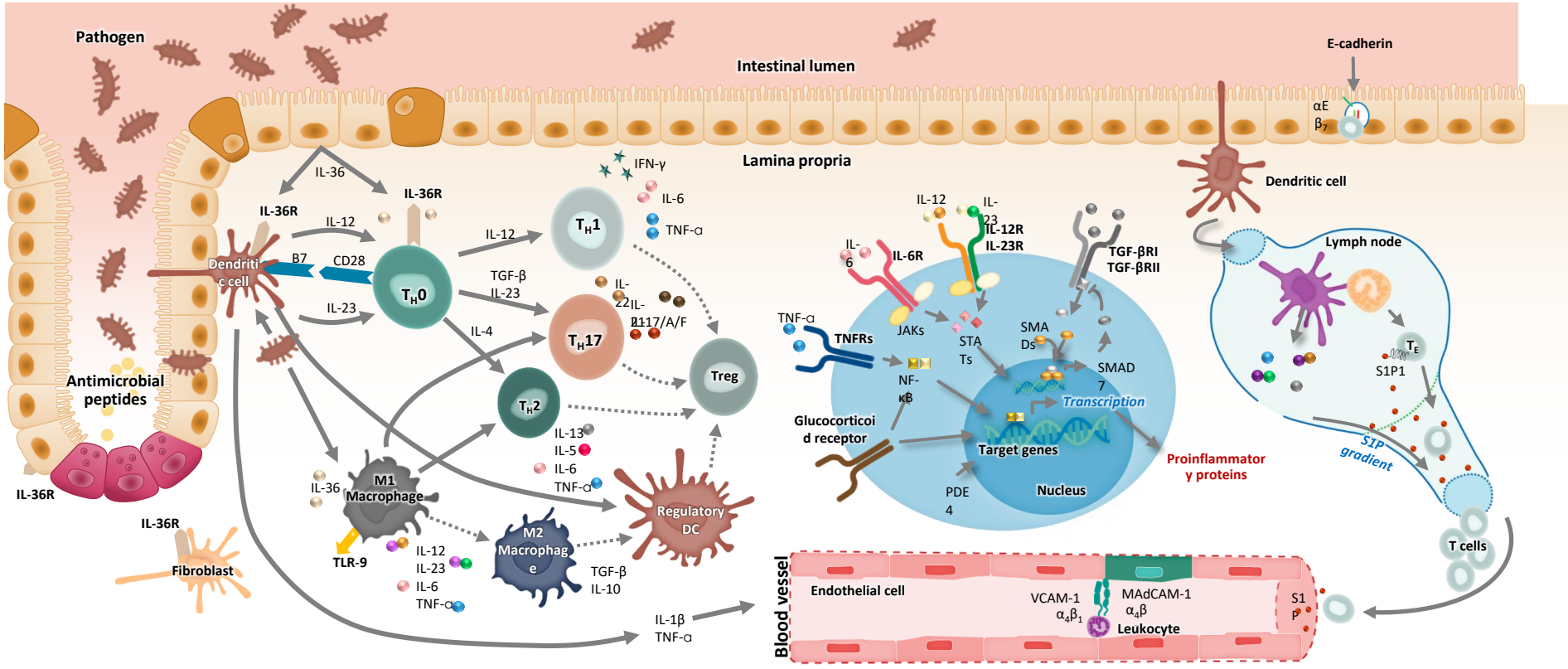
2025
Guselkumab



Colitis ulcerosa

Adaptiert vom Mulder DJ, et al. J Crohns Colitis. 2014;8:341-8; Park SC, et al. Gut Liver. 2015;9:18-27.

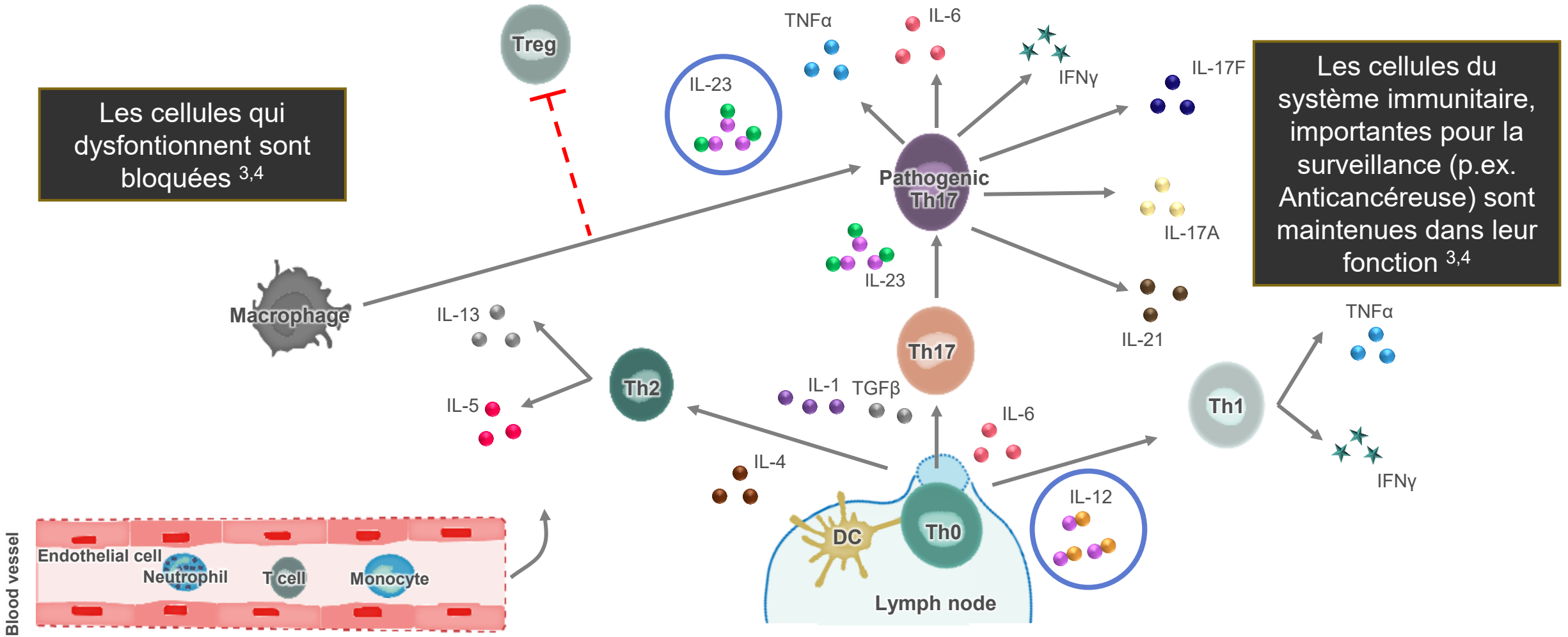
De nombreuses options pour “bloquer” l’inflammation dans les MICIs



Adapted from AbbVie slide set

Les nouvelles molécules - sont devenues plus spécifiques (e.g. IL-23 inhib.)

Les cellules qui dysfontionnent sont bloquées [3,4]



Les cellules du système immunitaire, importantes pour la surveillance (p.ex. Anticancéreuse) sont maintenues dans leur fonction [3,4]

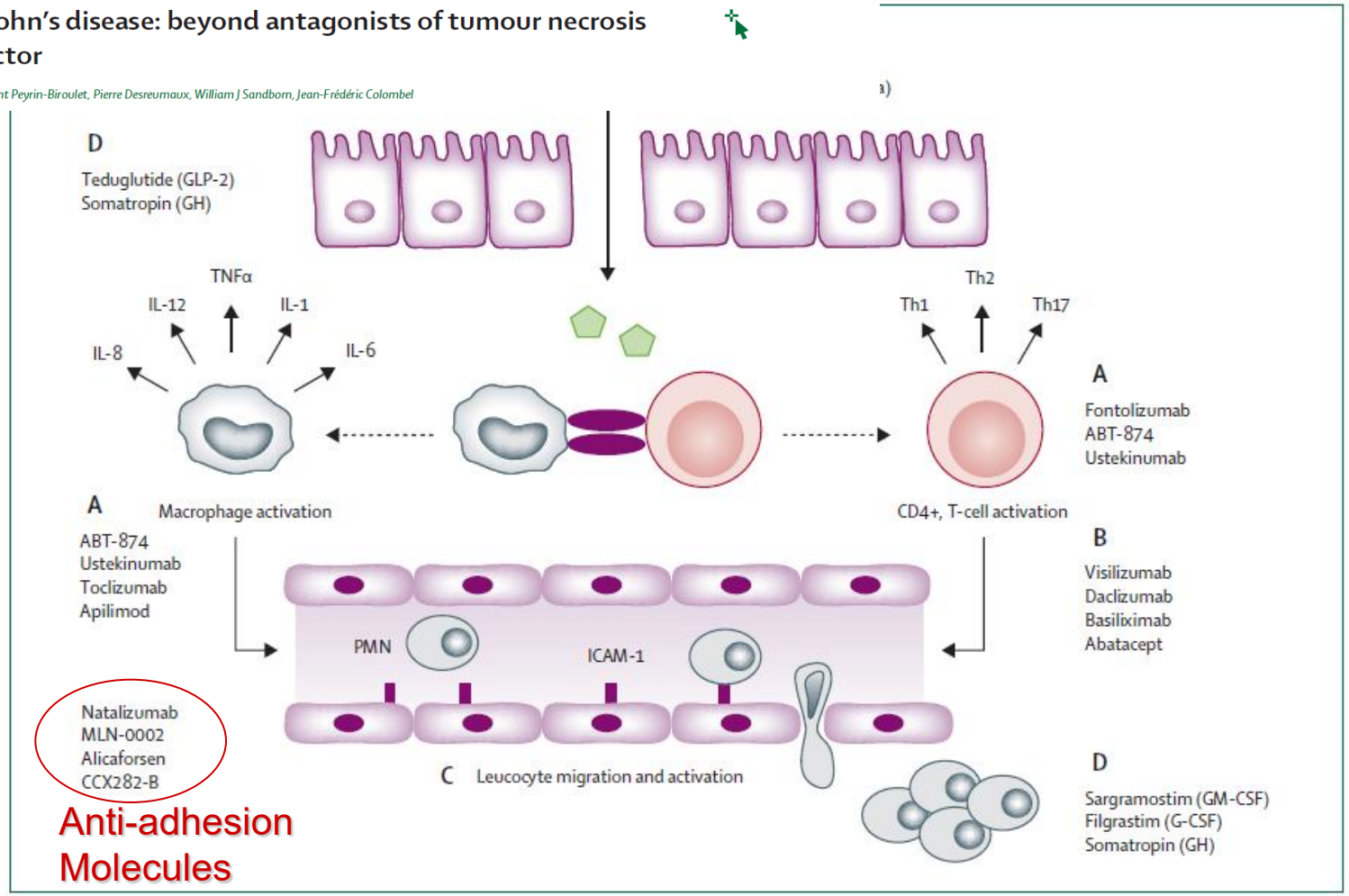
DC, dendritic cell; IFN, interferon; IBD, inflammatory bowel disease; IL, interleukin; TGF, transforming growth factor; Th, T helper cell; TNF, tumor necrosis factor; Treg, regulatory T cell.

1. Friedrich M, et al. *Immunity.* 2019;50:992–1006; 2. Hue S, et al. *J Exp Med.* 2006;203:2473–83; 3. Zhu J and Paul WE. *Immunol Rev.* 2010;238:247–62; 4. Haabeth OAW, et al. *Nat Commun.* 2011;2:240.

Différents mécanismes d'action

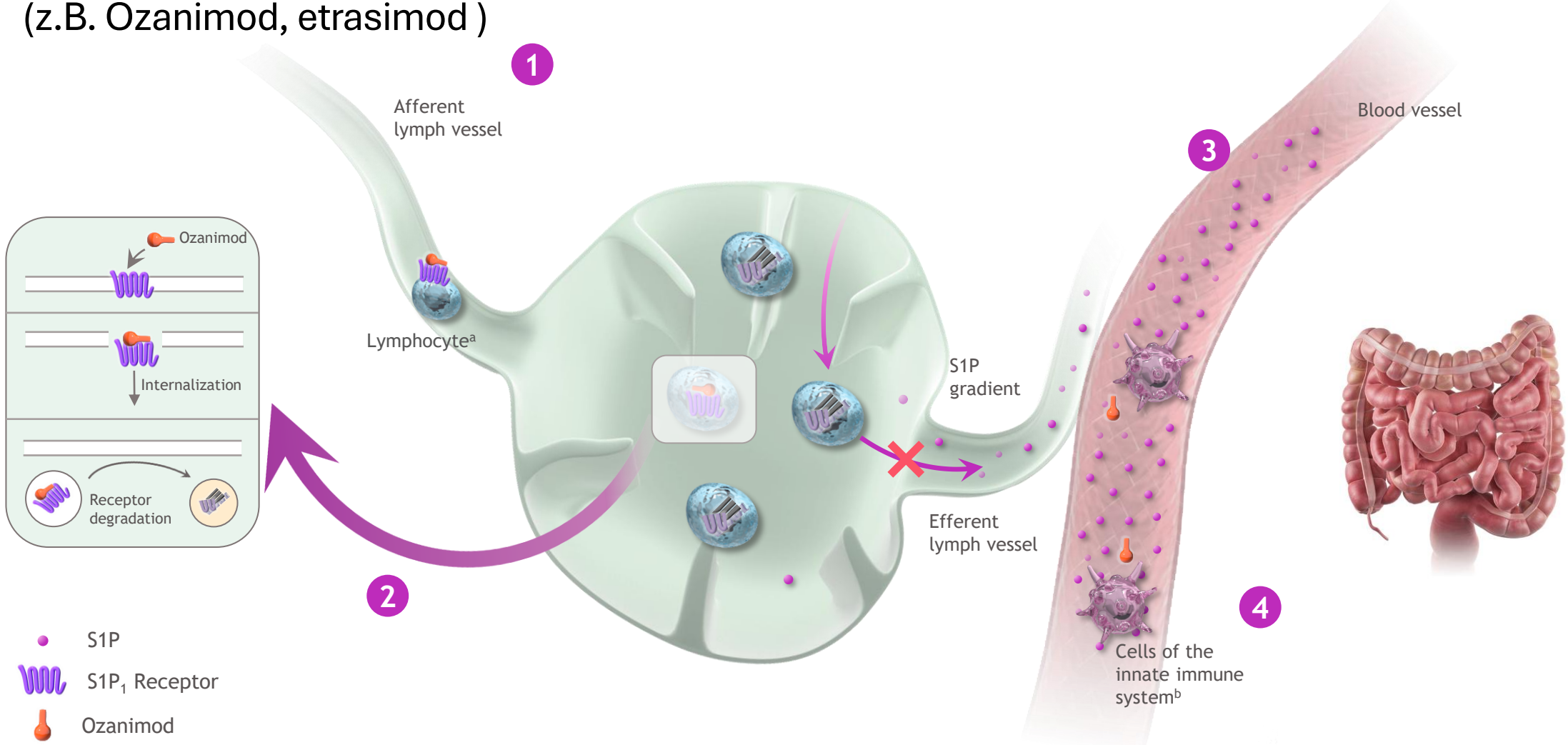
Crohn's disease: beyond antagonists of tumour necrosis factor

Laurent Peyrin-Biroulet, Pierre Desreumaux, William J Sandborn, Jean-Frédéric Colombel



Anti-adhesion Molecules

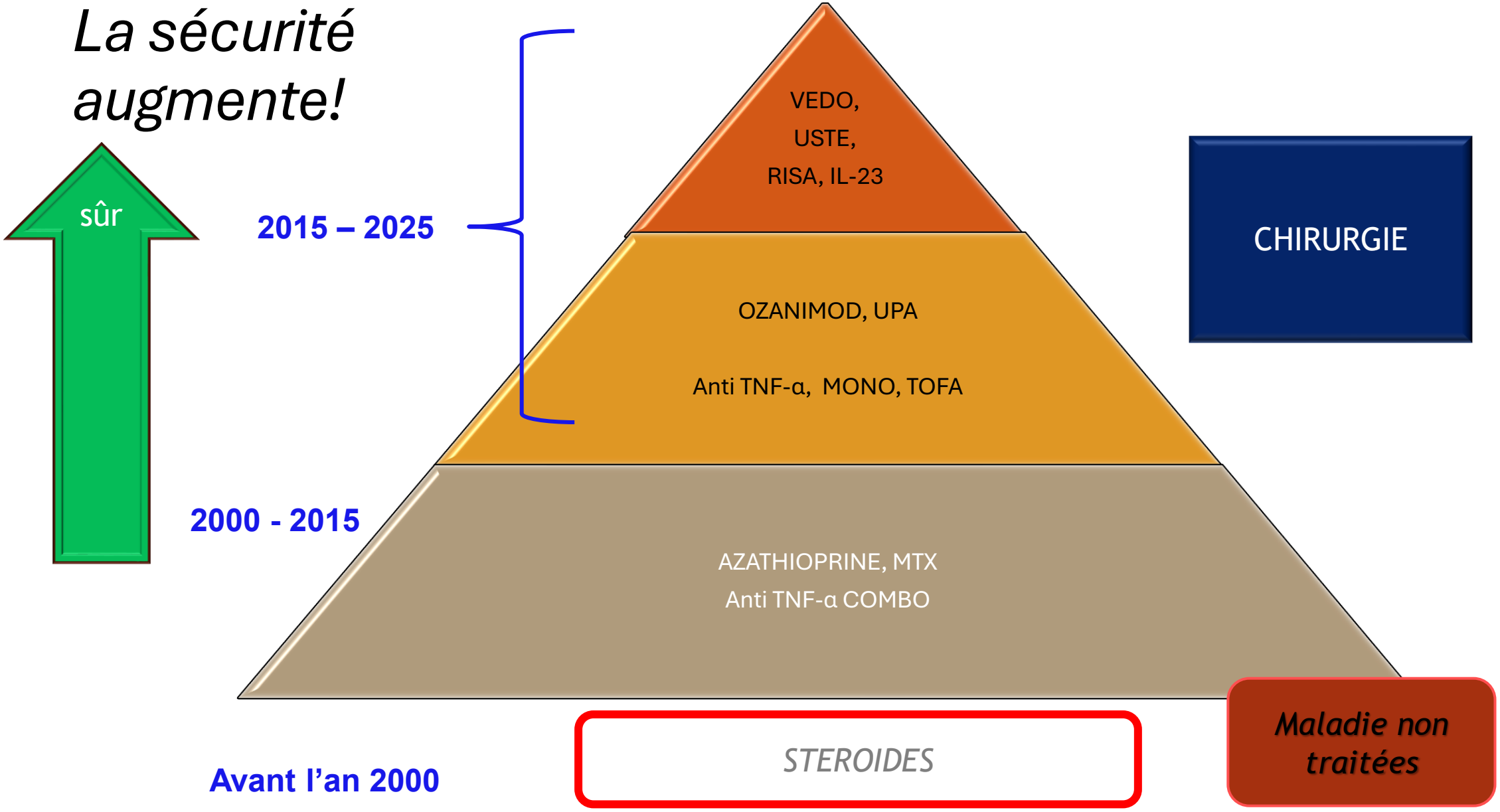
Certains medicaments conserve même les lymphocytes actives dans les ganglions lymphatiques (z.B. Ozanimod, etrasimod)



[a]Including T cells and B cells. [b]Innate immune cells include macrophages, monocytes, and natural killer cells, among others.
S1P, sphingosine 1-phosphate; $S1P_1$, sphingosine 1-phosphate receptor subtype 1.

1. Scott FL et al. *Br J Pharmacol*. 2016;173:1778-1792. Ozanimod (RPC1063) is a potent sphingosine-1-phosphate receptor-1 (S1P1) and receptor-5 (S1P5) agonist with autoimmune disease-modifying activity
2. Supplement to Scott FL et al. *Br J Pharmacol*. 2016;173:1778-1792.

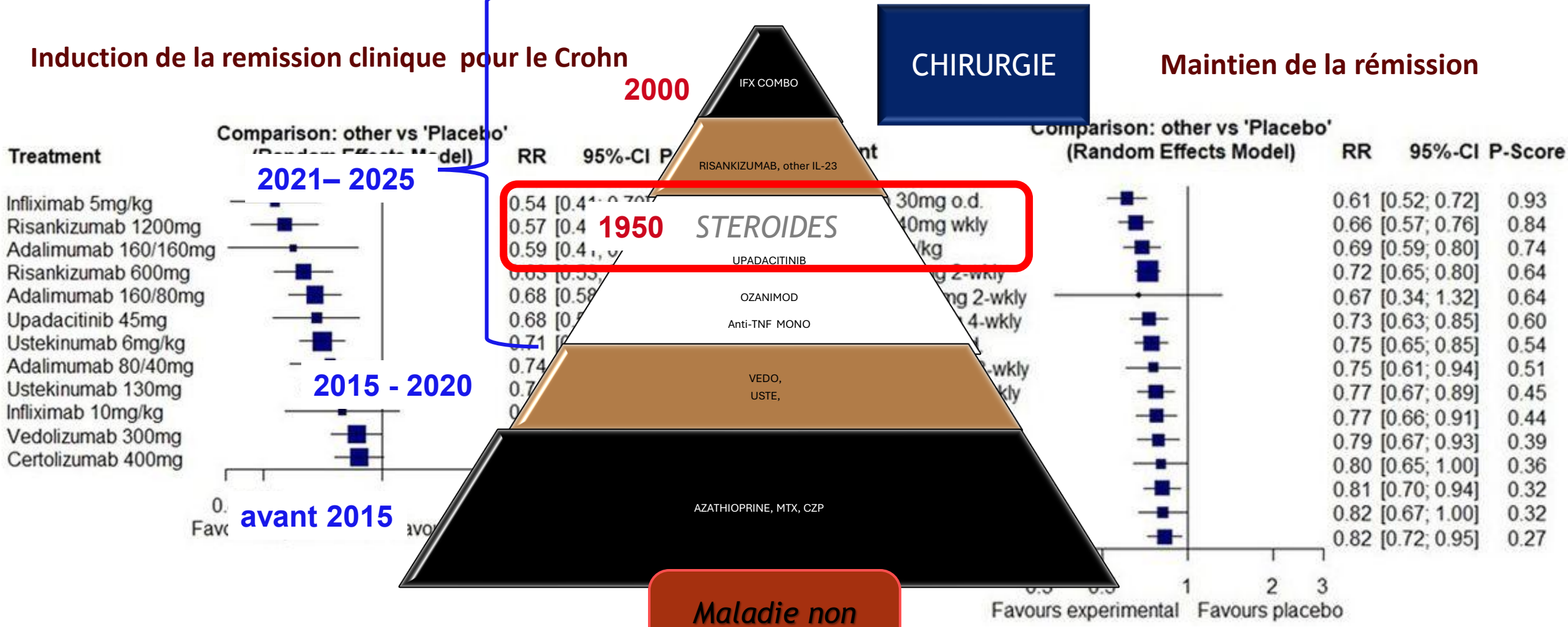
La sécurité augmente!



L'efficacité aussi !

Induction de la remission clinique pour le Crohn

Maintien de la rémission



Brigida Barberio et al. Efficacy of biological therapies and small molecules in induction and … nal Crohn's disease: systematic review and network meta-analysis; Gut 2023;72:264-274

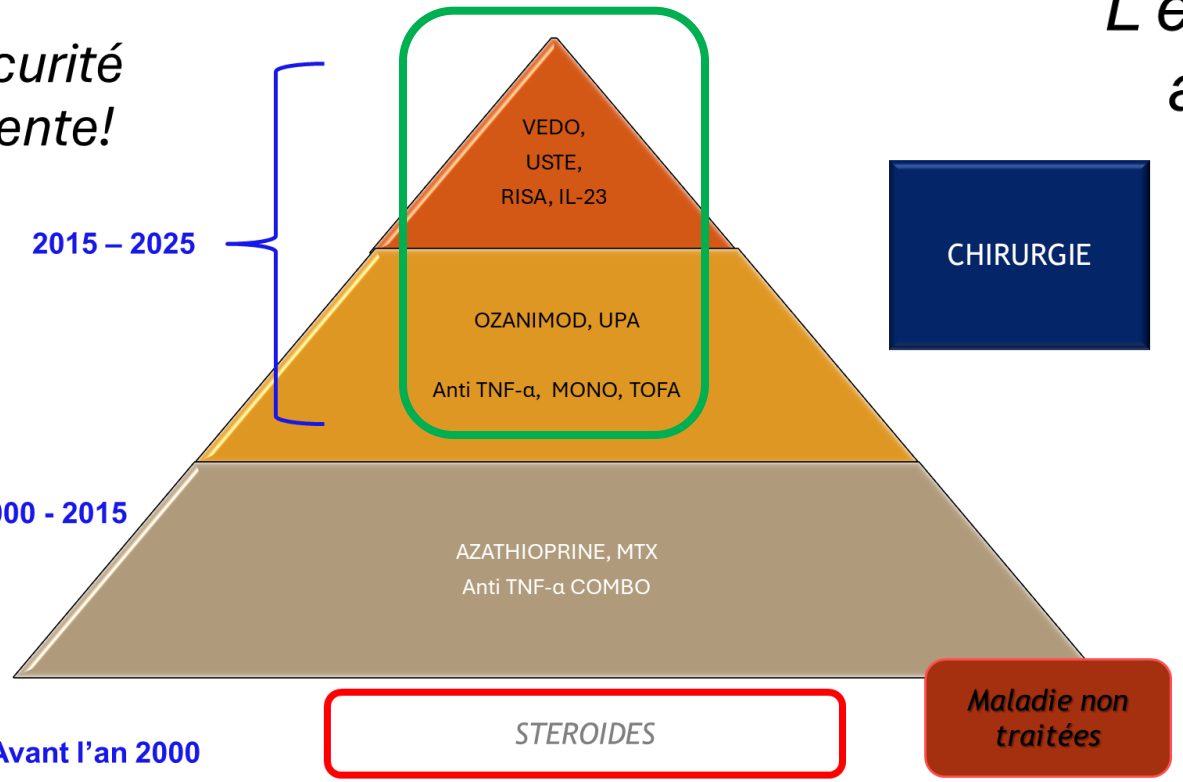
Intesto

Gastroenterologische Praxis & Crohn-Colitis-Zentrum Bern
Centre Fribourgeois de Gastroentérologie

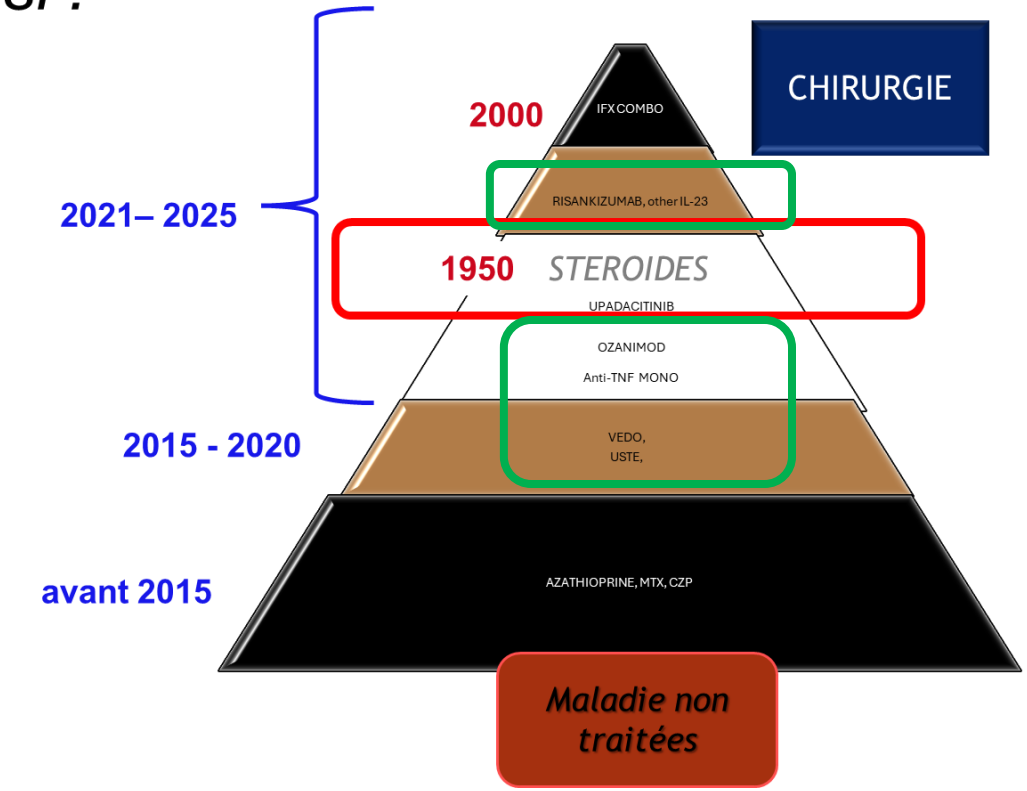
CENTRE GASTROENTÉROLOGIE BULLE

La sécurité augmente!

sûr



L'efficacité aussi !



CONCLUSIONS

- La prise en charge des maladies inflammatoires intestinales est devenue centrale en gastro-entérologie
- Variété thérapeutique grandissante, ces 20 dernières années
- Prise en charge multidisciplinaire , cruciale (spécialistes / généralistes)
- Détection des complications et effets indésirables - rôle clé du généraliste.

Merci pour votre ATTENTION !

Merci de votre attention !

@PascaJuillerat

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