Casual Friday Series

GERD: Functional Approaches

A BIOGENETIX CLINICAL PRESENTATION

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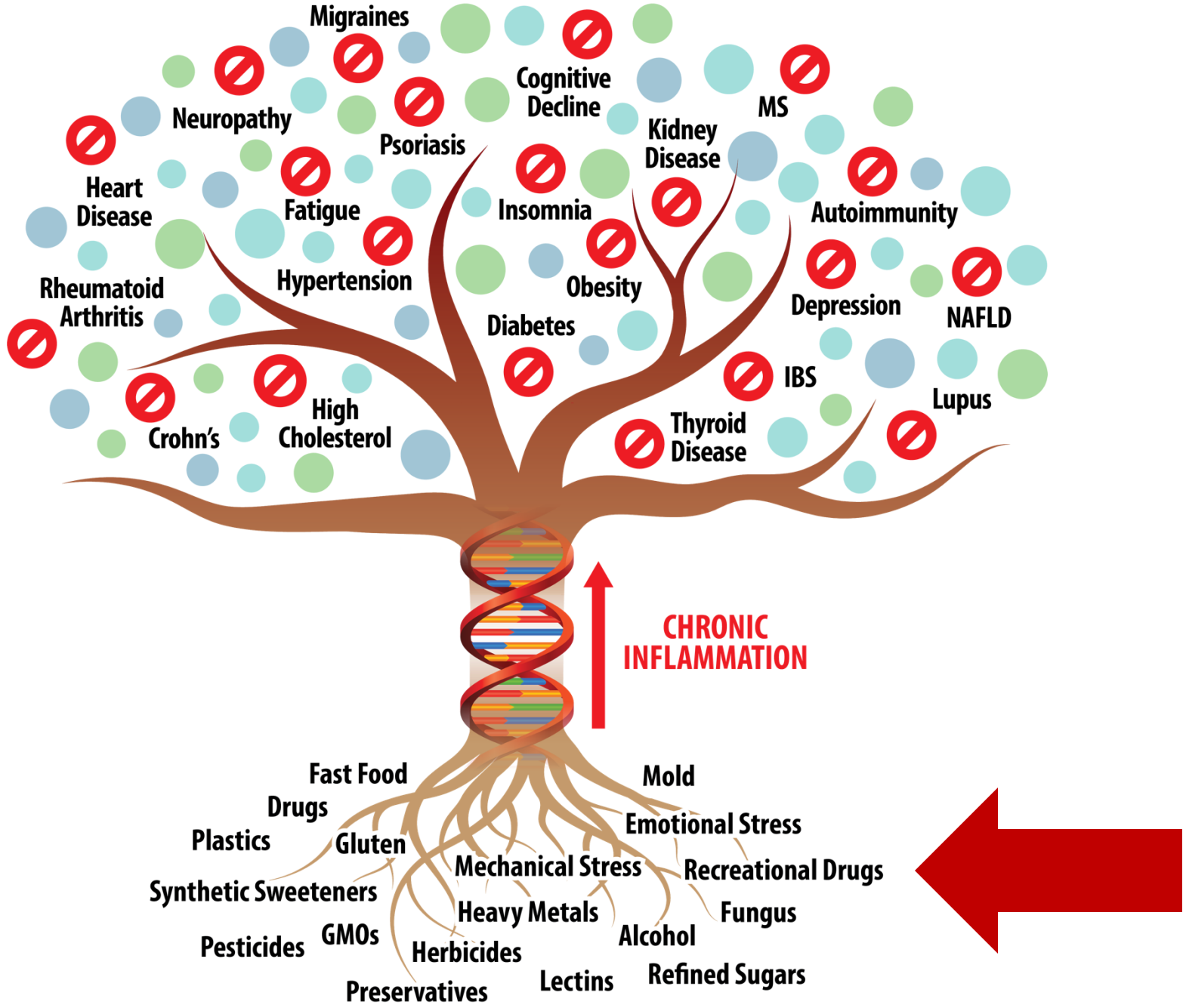
Migraines
Neuropathy
Psoriasis
Cognitive Decline
MS
Kidney Disease
Heart Disease
Fatigue
Insomnia
Autoimmunity
Rheumatoid Arthritis
Hypertension
Obesity
Depression
NAFLD
Diabetes
IBS
Lupus
Crohn's
High Cholesterol
Thyroid Disease
CHRONIC INFLAMMATION
Fast Food
Drugs
Plastics
Gluten
Synthetic Sweeteners
Pesticides
GMOs
Herbicides
Preservatives
Lectins
Refined Sugars
Alcohol
Heavy Metals
Mechanical Stress
Mold
Emotional Stress
Recreational Drugs
Fungus

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Gastroesophageal Reflux Disease

Gastroesophageal reflux disease is a condition where the retrograde flow of the stomach contents into the esophagus or beyond into other regions such as oral cavity, larynx, or the lungs results in inflammation of the esophageal mucosa. This condition is considered one of the most common diseases encountered by gastroenterologists and primary care clinicians. Risk factors for gastroesophageal reflux disease include being older than 50, having a body mass index >30, smoking, anxiety, depression, and decreased physical activity. Pharmacology that modulates the lower esophageal sphincter pressure, including nitrates, calcium channel blocker agents, and anticholinergics, can also contribute to developing gastroesophageal reflux disease. Esophageal reflux may result in several complications, including esophagitis, upper gastrointestinal bleeding, anemia, peptic ulcer, peptic stricture, dysphagia, gastric cardia cancer, and Barrett esophagus. Patients with severe gastroesophageal reflux disease who do not respond to initial strategies may require invasive procedures.

https://www.ncbi.nlm.nih.gov/books/NBK554462/

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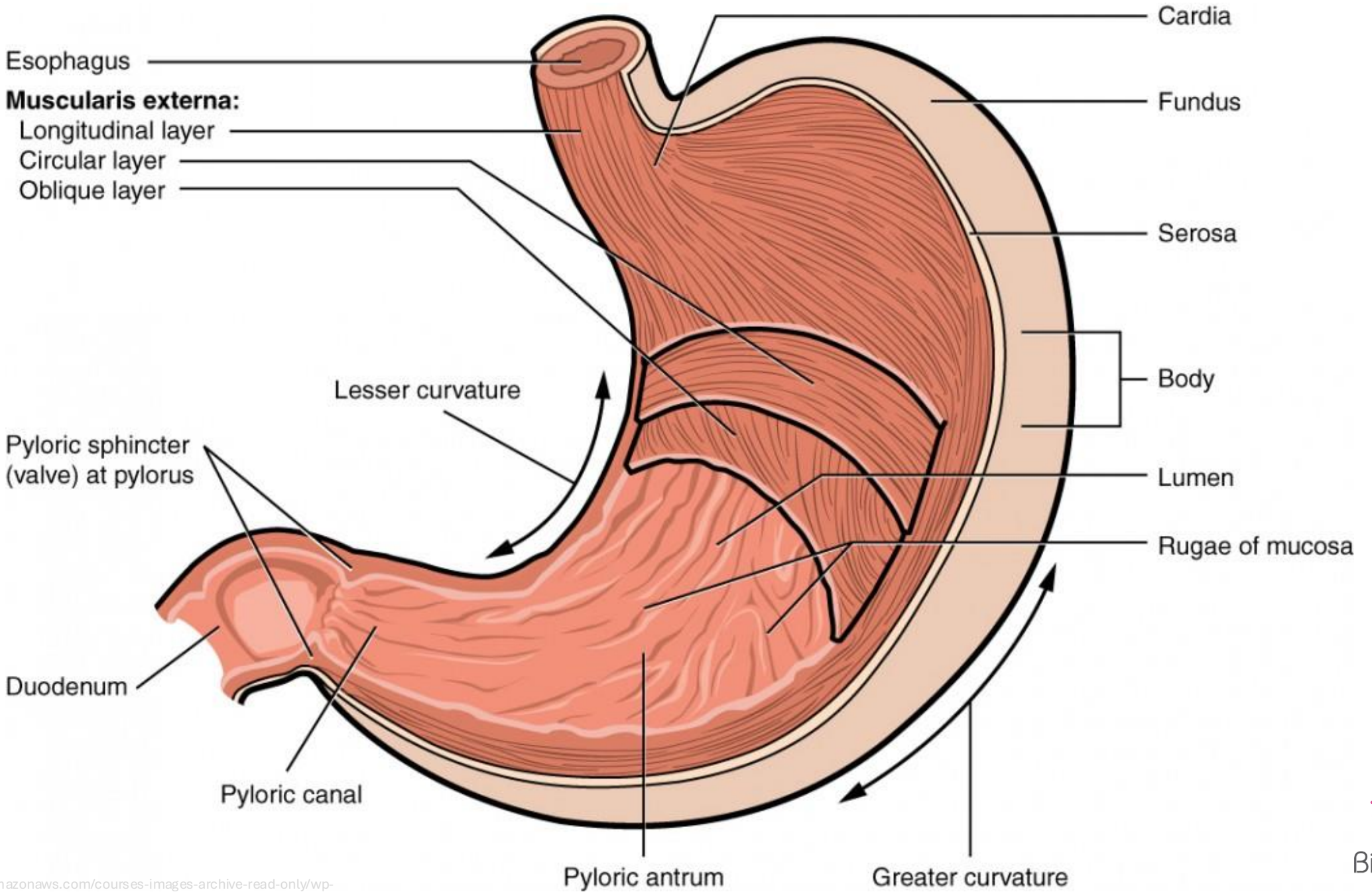

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Anatomical structures regulate esophageal function and minimize gastroesophageal reflux. A complex valvular mechanism at the esophagogastric junction antagonizes positive abdominal pressure and negative thoracic pressure. This anatomical mechanism comprises the lower esophageal sphincter, the diaphragm, the intra-abdominal portion of the esophagus, the angle of His, and the phrenoesophageal membrane.

- Lower esophageal sphincter: This physiological sphincter measures 3 to 5 cm in length. The high resting tone of the smooth muscle in the lower esophageal sphincter prevents regurgitation of gastric contents into the esophagus.
- Diaphragm: The esophagus enters the abdominal cavity through the diaphragmatic hiatus. The diaphragm provides extrinsic support to the lower esophageal sphincter.
- Abdominal portion of the esophagus: This esophageal segment is exposed to positive intra-abdominal pressure and collapses without a bolus. This collapse provides further support to the lower esophageal sphincter.
- Angle of His: This is the acute angle between the esophagus and the gastric fundus, which enhances the function of the lower esophageal sphincter.
- Phrenoesophageal membrane: This is a fibroelastic ligament that continues the transversalis fascia, which leaves the diaphragm and surrounds the esophagus.[6]

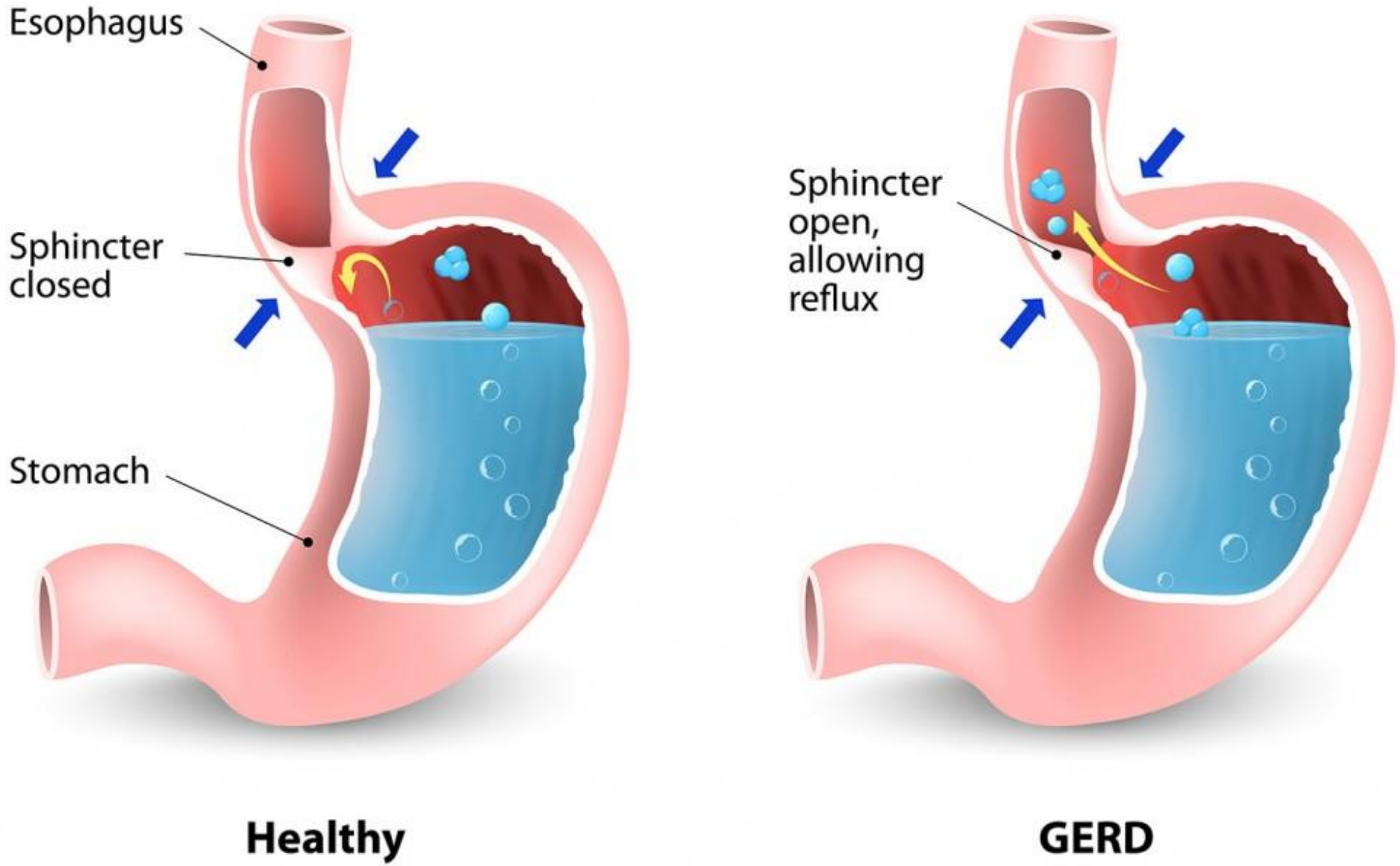
Physiologic mechanisms also protect against gastroesophageal reflux. These mechanisms include but are not limited to, esophageal peristalsis, saliva production, and inherent esophageal mucosal protection.

- Esophageal motility: Esophageal peristalsis promotes the return of regurgitated acid to the stomach.
- Saliva production: Swallowed saliva contains bicarbonate and is slightly alkaline; salivary mucins also act as lubricants.
- Esophageal epithelial protection: Esophageal submucosal glands also secrete bicarbonate and mucin to protect distal esophageal mucosa from acidic stomach contents.[7][8]

The reflux of gastric contents into the esophagus in healthy individuals is limited, and the refluxed contents are cleared through esophageal peristalsis. However, patients with GERD cannot clear these refluxed contents or produce protective physiological mechanisms. The underlying etiologies of GERD include but are not limited to:

- Transient relaxation of the lower esophageal sphincter or a low resting lower esophageal sphincter pressure
- Hiatal hernia
- Extrinsically increased intra-abdominal pressure, as in obesity
- Intrinsically increased intra-abdominal pressure, as observed during pregnancy or in patients with high-volume ascites
- Impaired esophageal motility
- Impaired saliva production
- Impaired esophageal mucosal defense mechanisms [9][10][11][12][13]

Reflux esophagitis occurs in patients with GERD when toxic substances such as gastric acid, pepsin, and bile salts come into contact with the esophageal mucosa, resulting in damage to the distal esophageal mucosa and mucosal breaks that can be detected through endoscopy in 30% to 40% of patients. The histology of GERD is not specific, as the histological changes may also be present in other pathological states, such as adjacent mucosa in esophageal cancer.[37] The histological changes associated with reflux esophagitis secondary to GERD include the following.



Healthy **GERD**

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https://gastrohealth.com/-/media/news/patient-care-photos/gastroesophageal-reflux-disease-1024x676.ashx

Mechanics

G Cells (antrum):

- Gastrin (stim HCL, motility, GB and pancreatic contractions)

Parietal Cells:

- Hydrochloric acid (via proton pump [H+])
- Intrinsic factor

ECL:

- Histamine (stim HCL)

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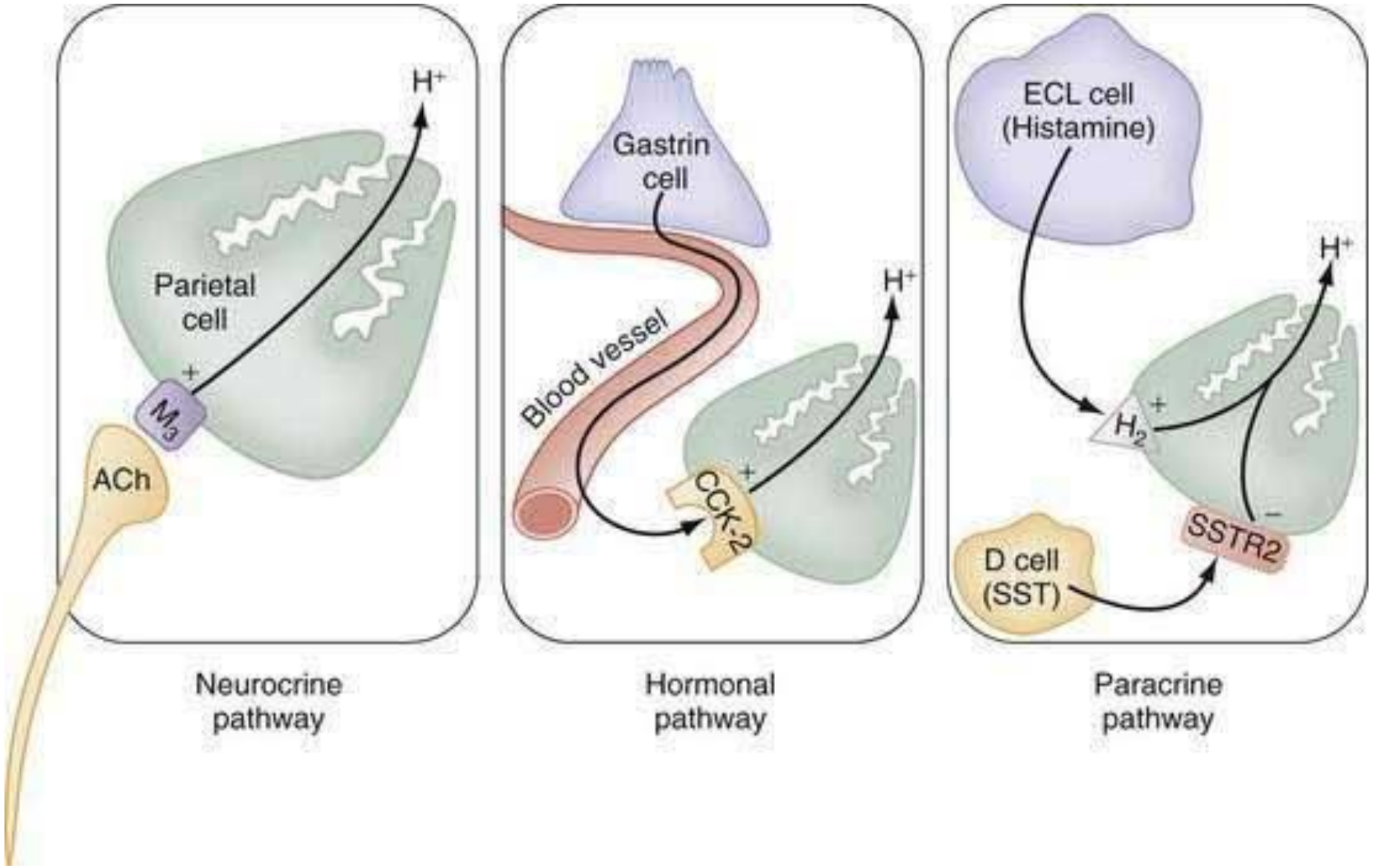
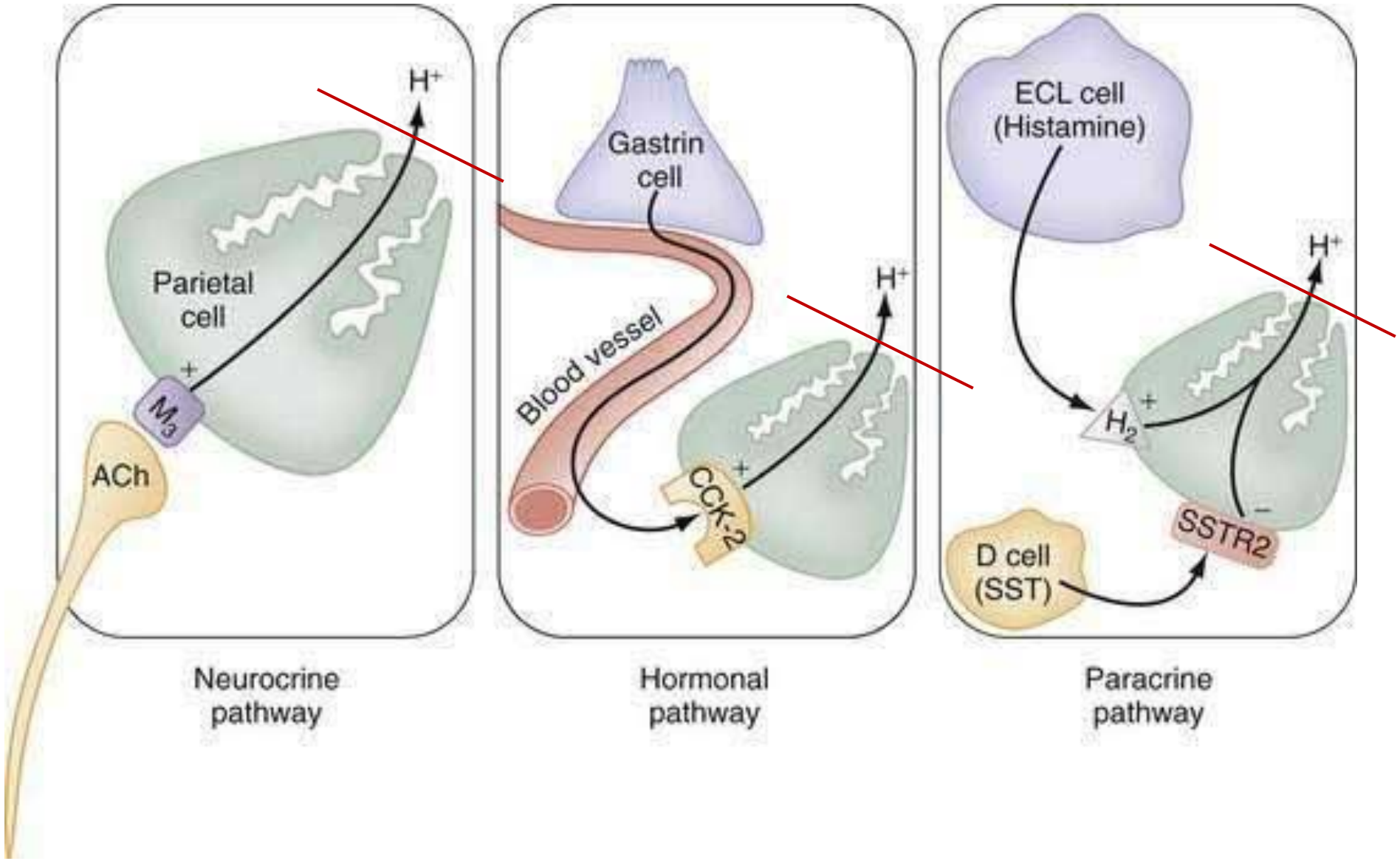


PPI intervention:



https://clinicalgate.com/wp-content/uploads/2015/05/B9781416061892000494_f12.jpg

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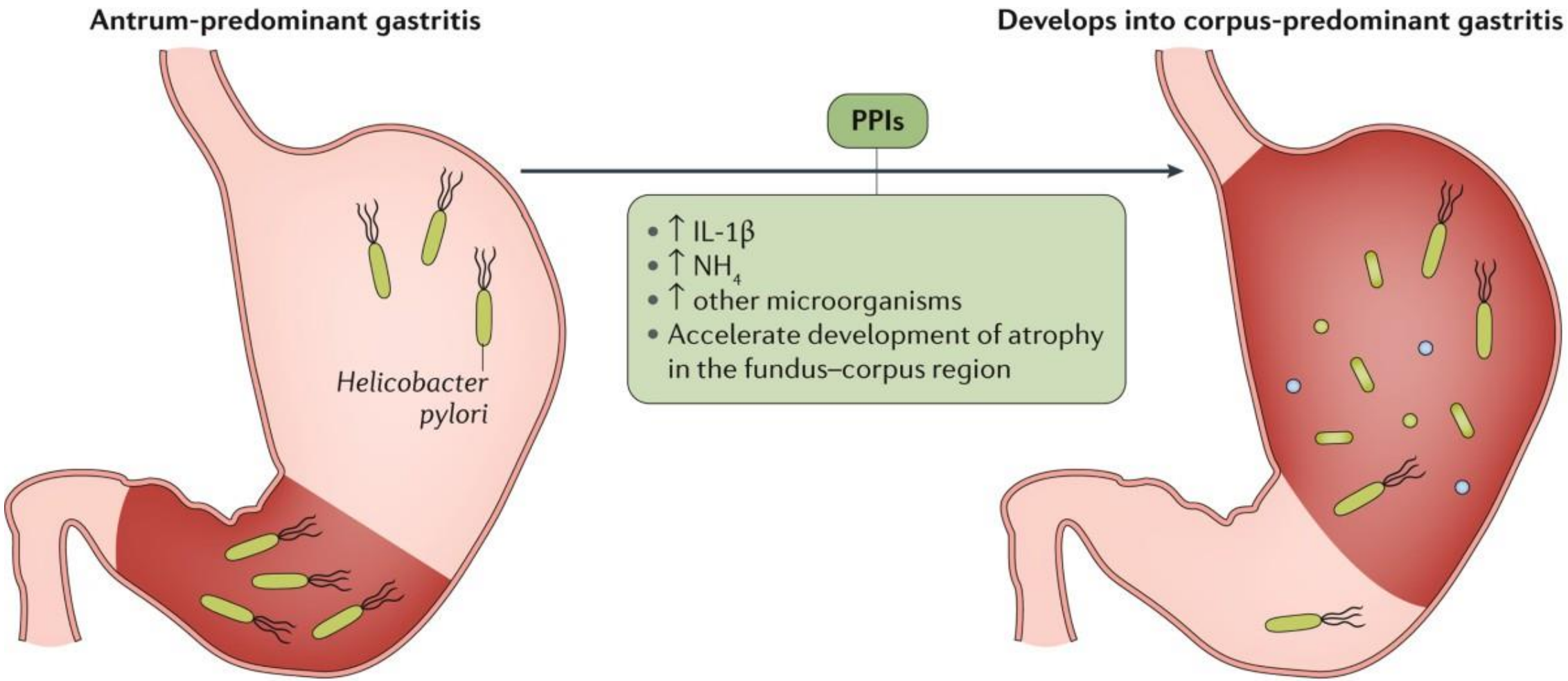
Cureus. 2023 Dec 3;15(12):e49883. doi: 10.7759/cureus.49883

Impact of Proton Pump Inhibitors on Kidney Function and Chronic Kidney Disease Progression: A Systematic Review

Mihirkumar P Parmar [1,✉], Safa Kaleem [2], Periyaiyadever Samuganathan [3], Lyluma Ishfaq [4], Tejawi Anne [5], Yashaswi Patel [6], Sashank Bollu [7], Roopeessh Vempati [8,9]

PubMed, PubMed Central (PMC), and Google Scholar articles from the last 10 years, from 2013 to 2023, and looked for links between PPI use and a number of kidney-related outcomes. These included acute kidney injury, a drop in the estimated glomerular filtration rate (eGFR), and new cases of CKD. The findings of this systematic review highlight the need for a thorough evaluation of the benefits and risks associated with PPI use, particularly in patients with pre-existing kidney conditions, in order to inform clinical decision-making and improve were taken out and looked at to see if there were any links between PPI use and different kidney-related events, such as acute kidney injury, a drop in the estimated eGFR, and the development of CKD. The review also explores potential mechanisms underlying PPI-induced nephrotoxicity. The findings of this systematic review highlight the need for a thorough evaluation of the benefits and risks associated with PPI use, particularly in patients with pre-existing kidney conditions, in order to inform clinical decision-making and improve patient care. Further research is warranted to better understand the complex interplay between PPIs, kidney function, and CKD progression.


Antrum-predominant gastritis
Develops into corpus-predominant gastritis
PPIs
• ↑ IL-1β
• ↑ NH_4
• ↑ other microorganisms
• Accelerate development of atrophy in the fundus–corpus region
Helicobacter pylori

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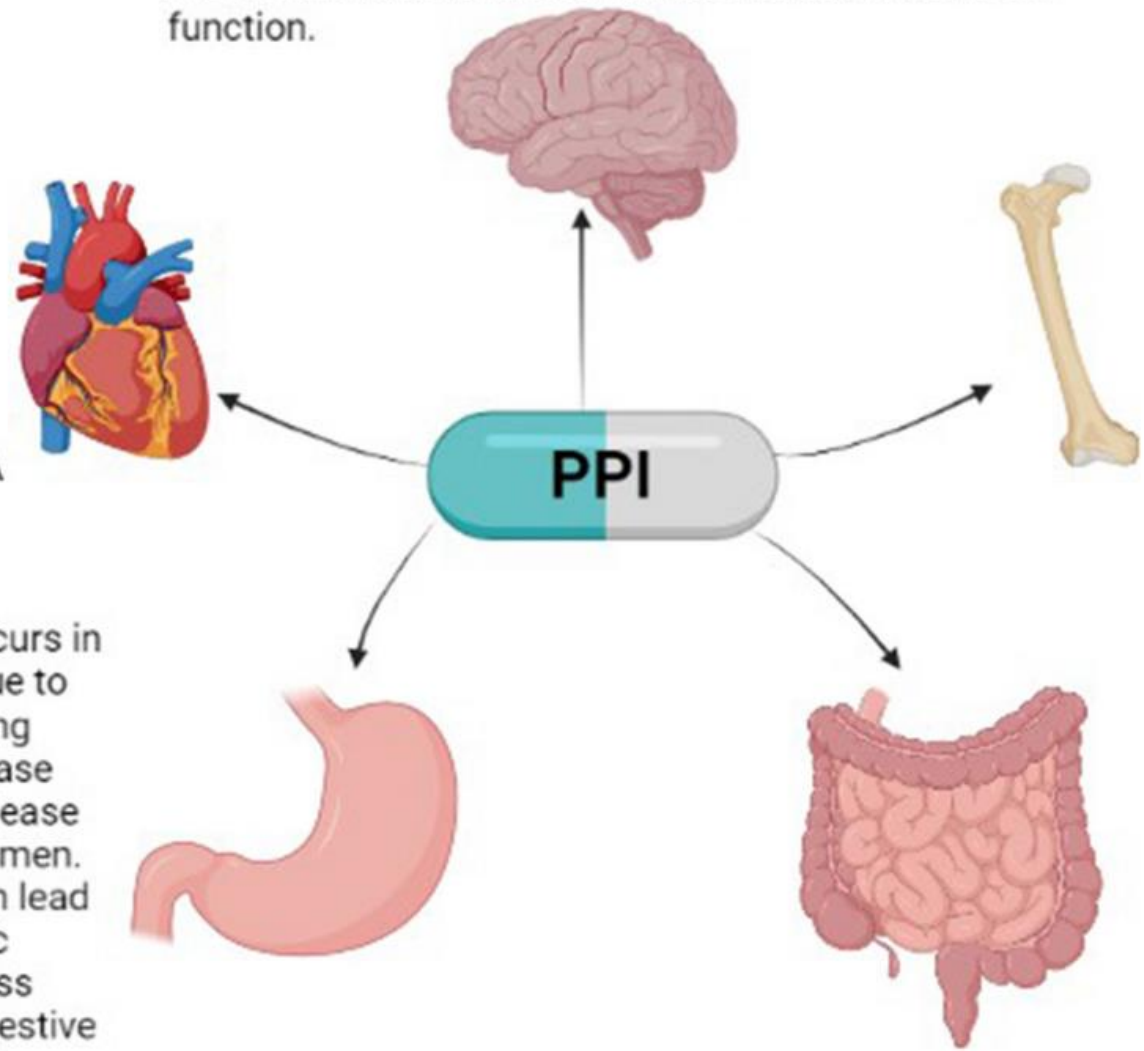
May lead to increased amyloid beta amounts via decrease in scavenger enzymes.
Also nutrient deficiencies can lead to abnormal brain function.

May impact healthy heart function via nutrient deficiencies, nitric oxide levels, alterations in cytochrome p450, and increases in chromogranin A

Bone fractures and bone growth are impacted subsequently by alterations in nutrient absorption. May also impact parathyroid, leading to an increased loss in bone mineralization.

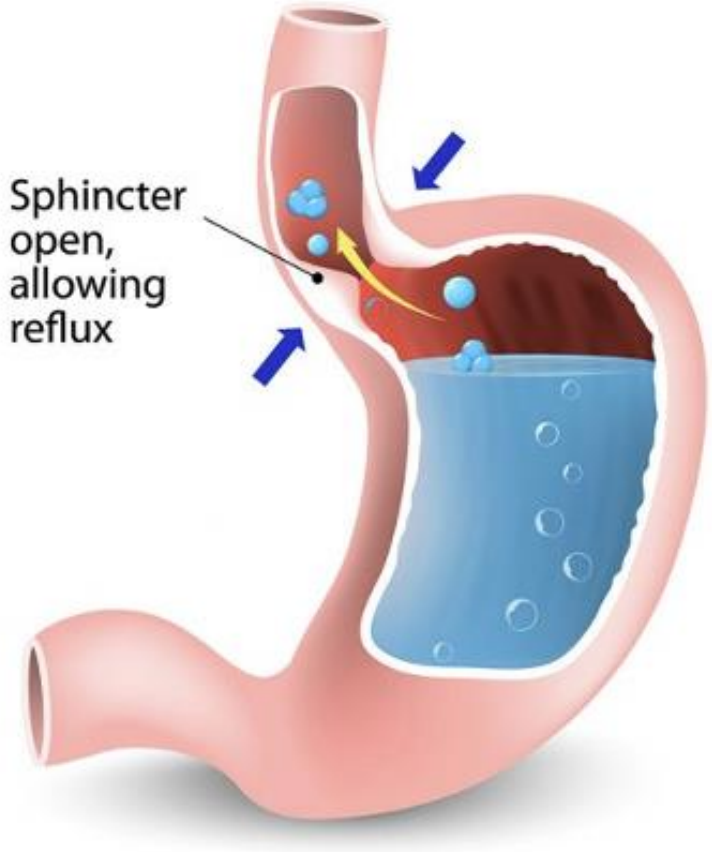


Alkalization occurs in the stomach due to covalent bonding with H^+/K^+ ATPase blocking the release of H^+ into the lumen. Alkalization can lead to opportunistic microbes to pass through the digestive tract, leading to overgrowth.

Dysbiosis occurs in the digestive tract due to subsequent changes in the gastrointestinal environment. Absorption of vitamins and other nutrients are impacted.

https://media.springernature.com/lw1200/springer-static/image/art%3A10.1186%2Fs42269-023-01107-9/MediaObjects/42269_2023_1107_Fig1_HTML.png

Functional Perspectives



GERD

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Functional Perspectives

Total Protein: 7.0-7.3

- Intake?
- High or Low
- On PPI?

AST, ALT, GGT:

- NAFLD
- Digestion/Detoxification Connection

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Functional Perspectives

Stool Testing:

- Reflection of enzyme function (Genova)
- Microbiome balance
- Inflammatory status

Food sensitivity Testing:

- IgG, IgA, IgE

Tox panel:

- What's in the food you're eating?

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	Test	Current Result and Flag		Previous Result and Date	Units	Reference Interval
	Glucose [01]	95			mg/dL	70-99
▲	**BUN** [01]	**55**	**High**		mg/dL	8-27
▲	**Creatinine** [01]	**2.57**	**High**		mg/dL	0.57-1.00
▼	**eGFR**	**20**	**Low**		mL/min/1.73	>59
	BUN/Creatinine Ratio	21				12-28
	Sodium [01]	136			mmol/L	134-144
	Potassium [01]	4.2			mmol/L	3.5-5.2
	Chloride [01]	96			mmol/L	96-106
	Carbon Dioxide, Total [01]	22			mmol/L	20-29
	Calcium [01]	9.5			mg/dL	8.7-10.3
	Protein, Total [01]	6.3			g/dL	6.0-8.5
	Albumin [01]	3.9			g/dL	3.9-4.9
	Globulin, Total	2.4			g/dL	1.5-4.5
	Bilirubin, Total [01]	0.8			mg/dL	0.0-1.2
▲	**Alkaline Phosphatase** [01]	**158**	**High**		IU/L	44-121
	AST (SGOT) [01]	19			IU/L	0-40
	ALT (SGPT) [01]	6			IU/L	0-32

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Homocyst(e)ine

	Test	Current Result and Flag		Previous Result and Date	Units	Reference Interval
▲	**Homocyst(e)ine** [04]	**28.0**	**High**		umol/L	0.0-17.2

Uric Acid

	Test	Current Result and Flag		Previous Result and Date	Units	Reference Interval
	Uric Acid [01]	6.3			mg/dL	3.0-7.2
				Therapeutic target for gout patients: <6.0		

Phosphorus

	Test	Current Result and Flag		Previous Result and Date	Units	Reference Interval
▲	**Phosphorus** [01]	**4.4**	**High**		mg/dL	3.0-4.3

LDH

	Test	Current Result and Flag		Previous Result and Date	Units	Reference Interval
	LDH [01]	196			IU/L	119-226

GGT

	Test	Current Result and Flag		Previous Result and Date	Units	Reference Interval
▲	**GGT** [01]	**87**	**High**		IU/L	0-60

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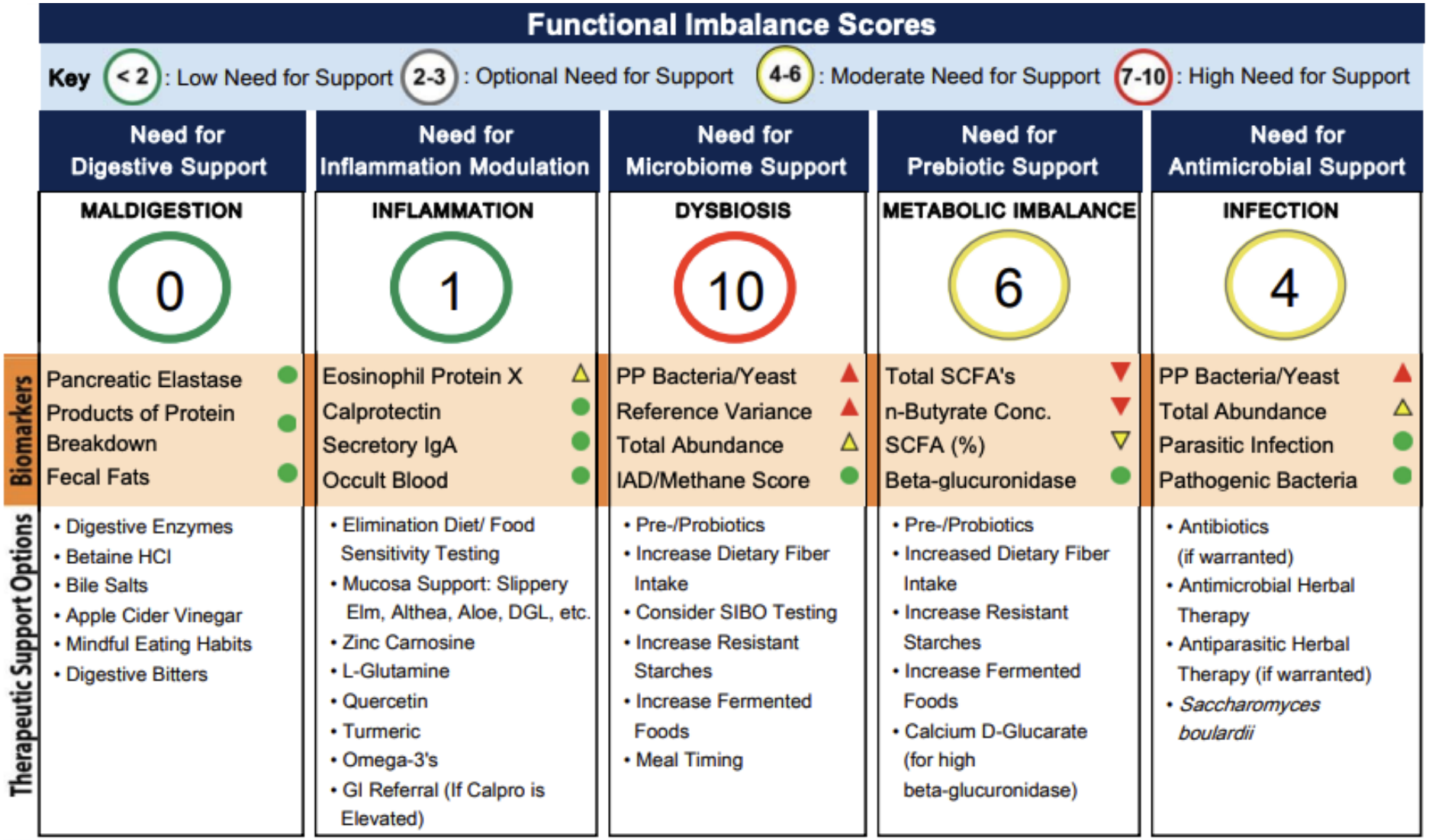
Functional Imbalance Scores
Key
< 2 : Low Need for Support
2-3 : Optional Need for Support
4-6 : Moderate Need for Support
7-10 : High Need for Support
Need for Digestive Support
Need for Inflammation Modulation
Need for Microbiome Support
Need for Prebiotic Support
Need for Antimicrobial Support
MALDIGESTION
0
INFLAMMATION
1
DYSBIOSIS
10
METABOLIC IMBALANCE
6
INFECTION
4
Biomarkers
Pancreatic Elastase
Products of Protein Breakdown
Fecal Fats
Eosinophil Protein X
Calprotectin
Secretory IgA
Occult Blood
PP Bacteria/Yeast
Reference Variance
Total Abundance
IAD/Methane Score
Total SCFA's
n-Butyrate Conc.
SCFA (%)
Beta-glucuronidase
PP Bacteria/Yeast
Total Abundance
Parasitic Infection
Pathogenic Bacteria
Therapeutic Support Options
• Digestive Enzymes
• Betaine HCl
• Bile Salts
• Apple Cider Vinegar
• Mindful Eating Habits
• Digestive Bitters
• Elimination Diet/ Food Sensitivity Testing
• Mucosa Support: Slippery Elm, Althea, Aloe, DGL, etc.
• Zinc Carnosine
• L-Glutamine
• Quercetin
• Turmeric
• Omega-3's
• GI Referral (If Calpro is Elevated)
• Pre-/Probiotics
• Increase Dietary Fiber Intake
• Consider SIBO Testing
• Increase Resistant Starches
• Increase Fermented Foods
• Meal Timing
• Pre-/Probiotics
• Increased Dietary Fiber Intake
• Increase Resistant Starches
• Increase Fermented Foods
• Calcium D-Glucarate (for high beta-glucuronidase)
• Antibiotics (if warranted)
• Antimicrobial Herbal Therapy
• Antiparasitic Herbal Therapy (if warranted)
• Saccharomyces boulardii

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High (>95th percentile)

Mycotoxins | Heavy Metals | Environmental Toxins

TEST NAME	CURRENT RESULT	PREVIOUS RESULT	CURRENT RESULT / PREVIOUS RESULT	REFERENCE
Citrinin (CTN)	26.49		0 — 7.05 — 12.5	≤12.53 ng/g
Dihydrocitrinone	22.62		0 — 9.3 — 16.5	≤16.53 ng/g
Fumonisins B2	10.54		0 — 4.05 — 7.2	≤7.2 ng/g
Mycophenolic Acid	17.79		0 — 3.6 — 6.4	≤6.4 ng/g
Ochratoxin A (OTA)	11.27		0 — 3.83 — 6.8	≤6.8 ng/g
Zearalenone (ZEN)	1.2		0 — 0.38 — 0.67	≤0.67 ng/g
Barium*	8.94		0 — 2.33 — 5.59	≤5.59 ug/g
Uranium*	0.11		0 — 0.02 — 0.04	≤0.04 ug/g
4-Nonylphenol	3.79		0 — 0.42 — 2.06	≤2.06 ug/g
Bisphenol A (BPA)*	9.6		0 — 2.12 — 5.09	≤5.09 ug/g

* Indicates NHANES population data reference ranges.

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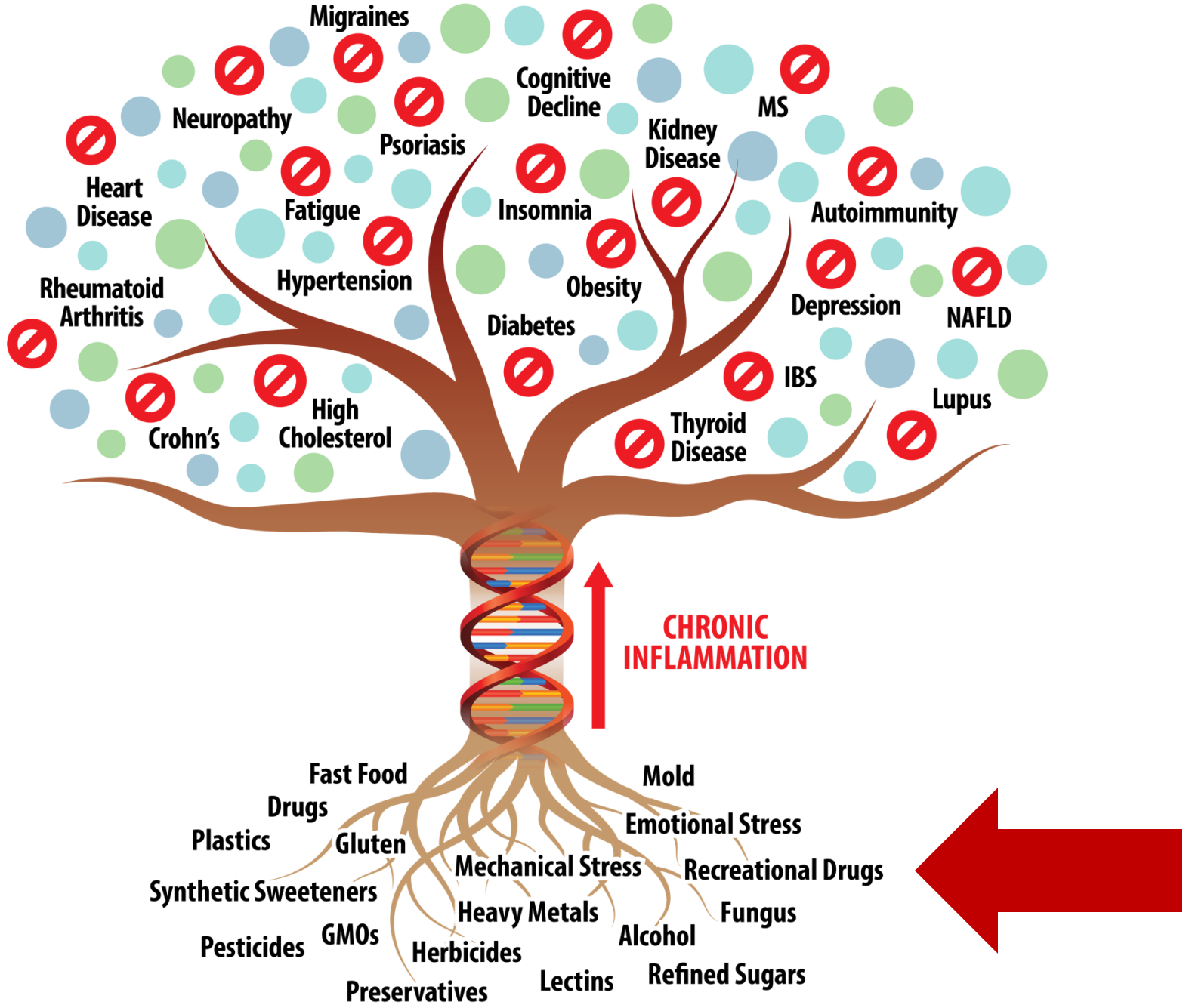
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Herbicides
Preservatives
Lectins
Refined Sugars
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Submit your case to the CC team

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