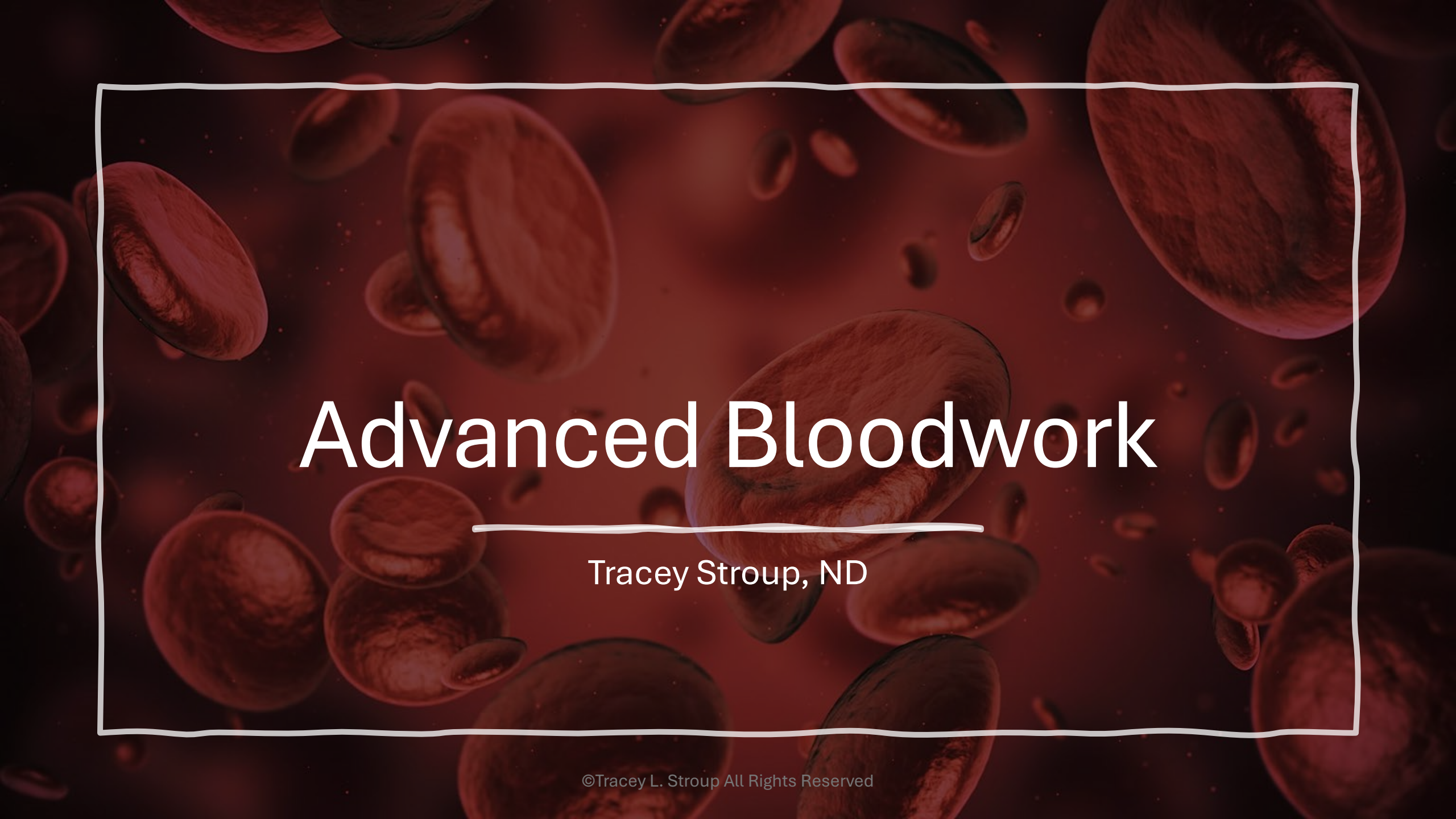


Advanced Bloodwork

Tracey Stroup, ND

Disclaimer

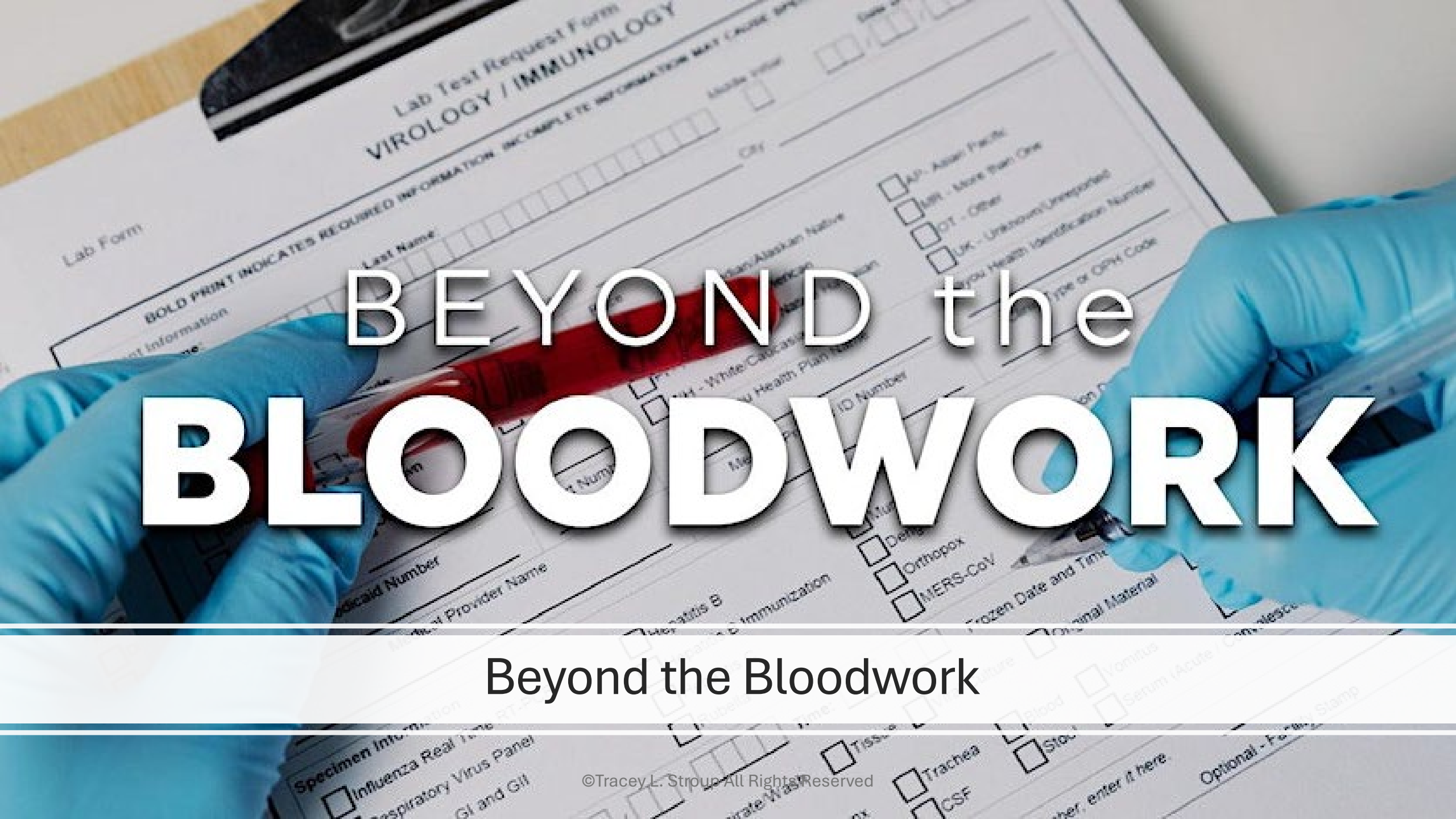
The information presented here is for informational purposes only.

About me

Dr Tracey Stroup

Naturopathic Doctor, B.S.
in Exercise and Sport Science, Certified
Natural Health Professional, a Level 2 Digestive Specialist,
Master Herbalist, and an Aromatherapist

Dr Tracey has been in the health and wellness industry for over 25 years. She is a sought out clinician, speaker and presenter for medical freedom and educator for integrative and complementary care for medical professionals. Tracey specializes in Post Covid/Jab Recovery protocols, chronic health issues, MTHFR mutation, peptide protocols and biblical health. Most importantly, Tracey is a wife, mom, and a
lover of Jesus Christ.



Beyond the Bloodwork

Why Advanced Bloodwork or Testing?

- Beneficial when the Baseline does not unveil the mystery
- Deeper assessment of the issue
- Give us a clearer picture
- Most traditional docs won’t run these tests as they are outside their “scope” or ”specialty”
- Allows for a whole person look

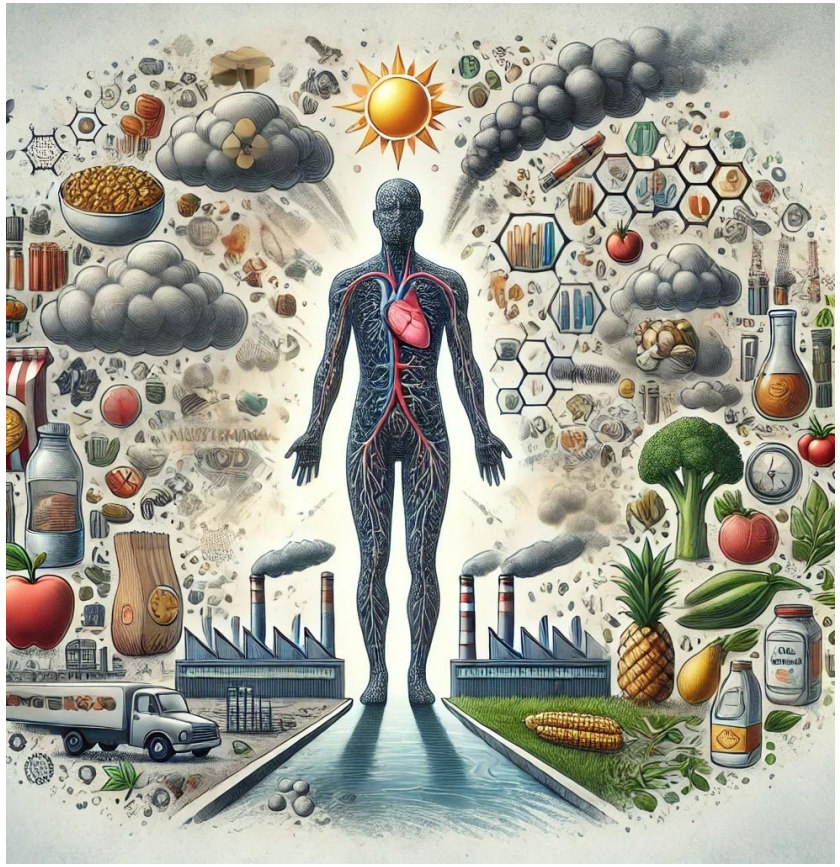
Setting the tone

Our society is in the midst of a profound metabolic crisis, driven by a toxic convergence of environmental pollutants, nutrient deficiencies, and lifestyle-induced imbalances.

Decades of industrialized farming have saturated our environment with chemicals, while modern diets are stripped of essential nutrients, leaving us deficient and vulnerable.

This toxic overload and deficiency contribute to chronic, systemic inflammation, which is at the heart of the modern epidemic of neurological disorders, obesity, cardiovascular diseases, and cancer.

The rise of these conditions reflects not just individual choices but a broken system that prioritizes convenience and profit over health, resulting in widespread metabolic dysfunction.



Advanced Bloodwork

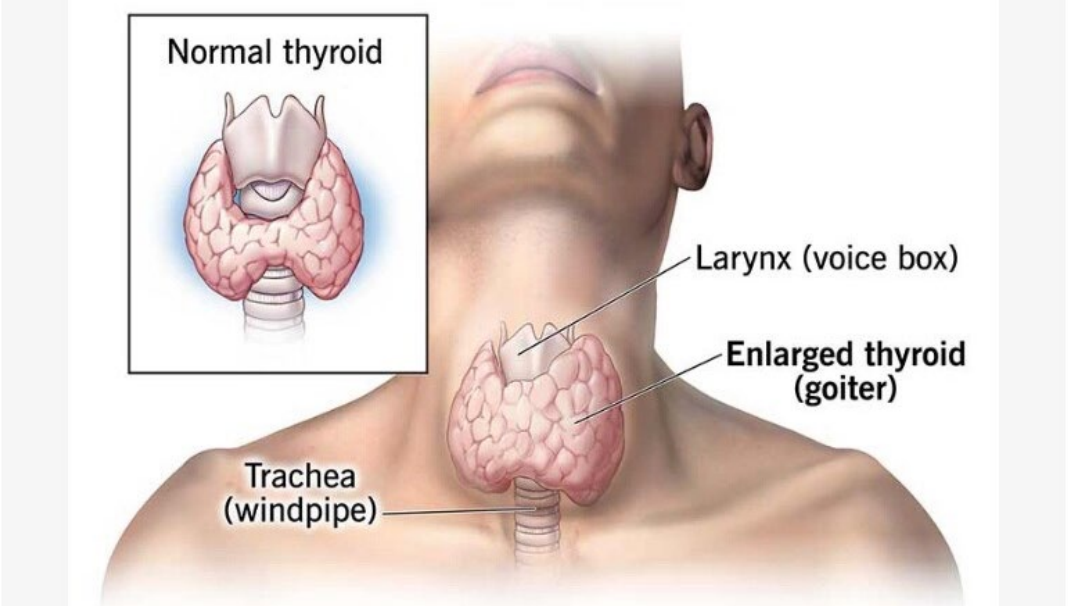
- Thyroid
- EBV/Strep
- MCAS
- Metabolic

Thyroid Disorders – Understanding Function

The thyroid is a small, butterfly-shaped gland located in the front of the neck, just below your Adam's apple. It produces two hormones that help regulate many functions in the body.

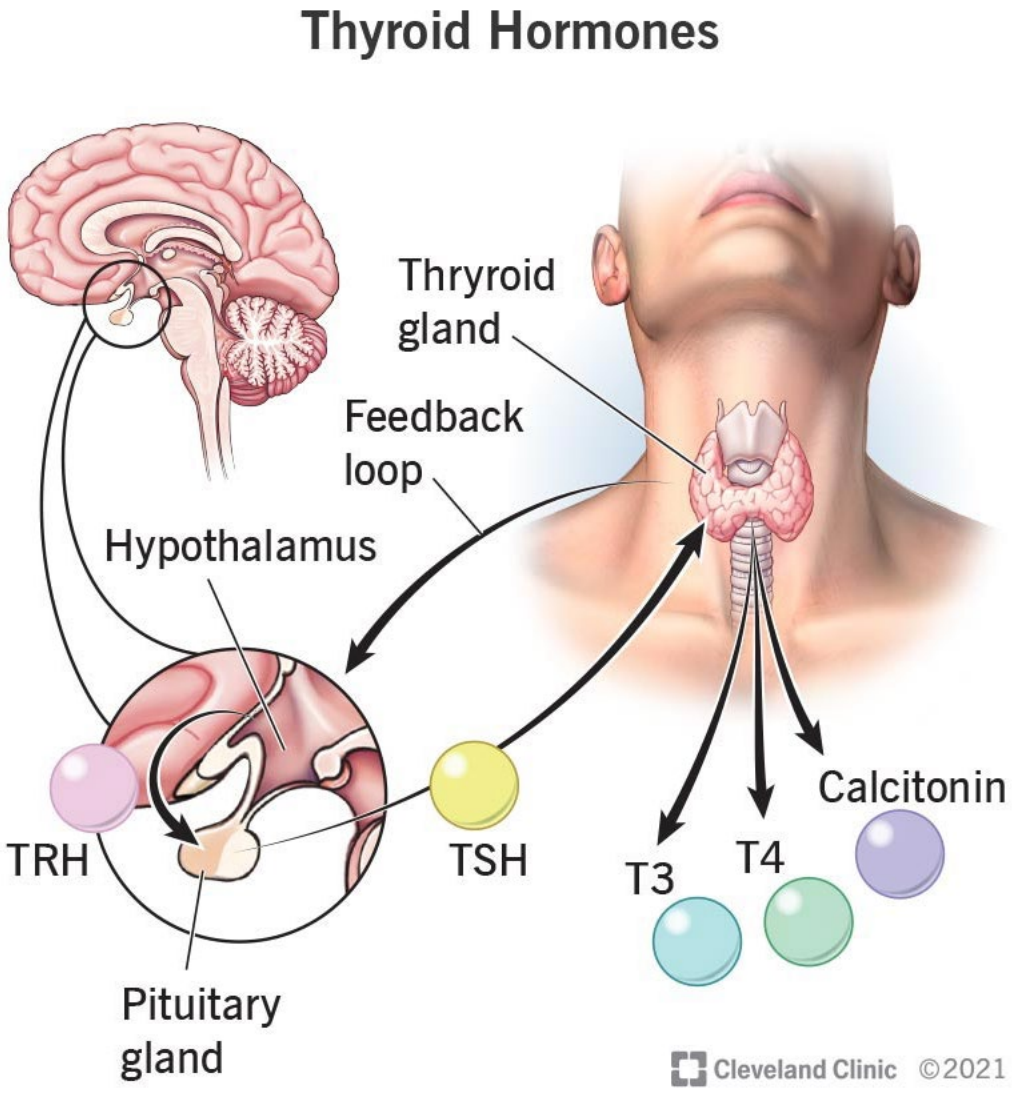
These hormones are triiodothyronine (T3) and thyroxine (T4). Most thyroid disorders stem from issues with T3 conversion or T3 resistance.

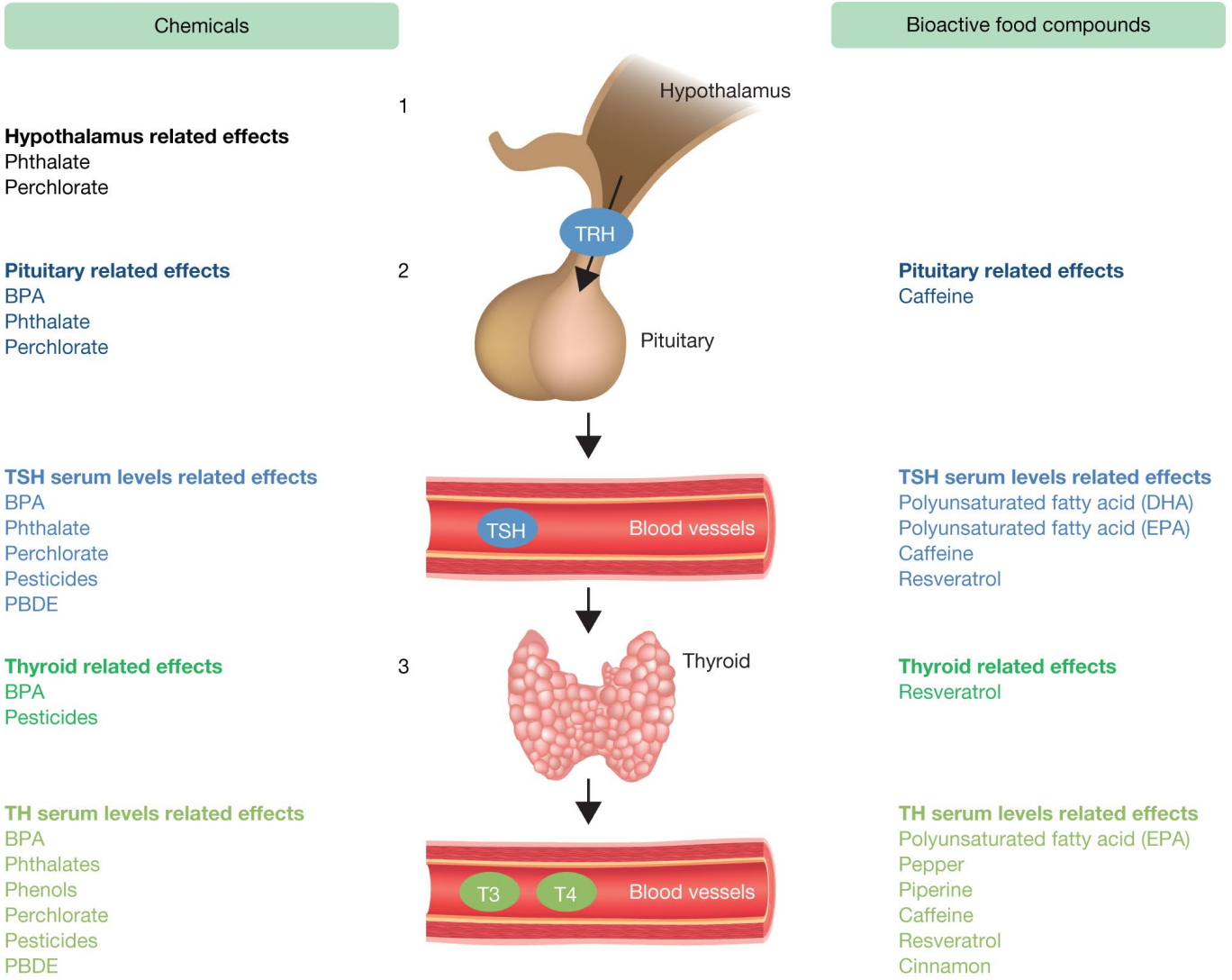
The thyroid produces these hormones by taking iodine from the food you eat, combining it with the amino acid tyrosine, and then converting it into the hormones. These hormones travel through your bloodstream to tissues and organs throughout your body.



Feedback loop

- Heavily affected by stress
- Nutrients or depletion
- Heavy metals
- Inflammation

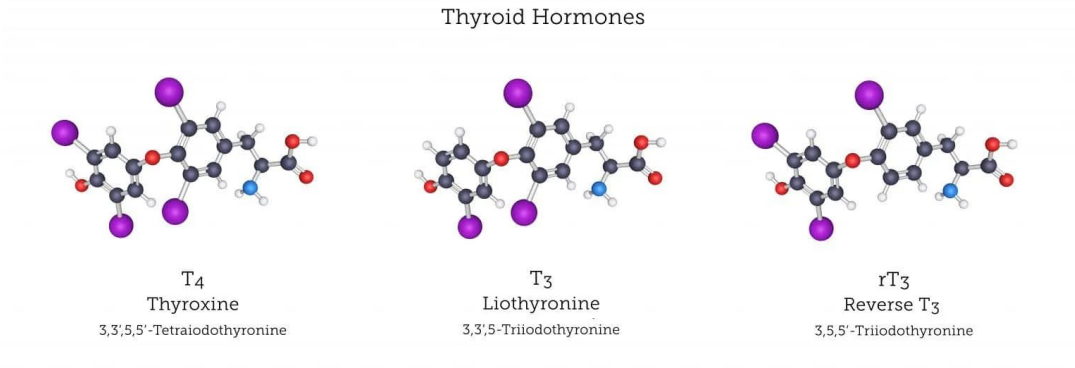




Simple scheme of the hypothalamus–pituitary–thyroid axis. The hypothalamus produces thyrotropin-releasing hormone (TRH), which stimulates the pituitary to release thyrotropin (thyroid-stimulating hormone, TSH). TSH is released in circulation and stimulates the thyroid gland to produce thyroid hormones (THs) (Ortiga-Carvalho et al. 2014). The thyroid produces the main THs, thyroxine (T4) and triiodothyronine (T3). TSH stimulates all the steps of TH biosynthesis and release, as well as the expression and activity of several proteins: the solute carrier family 5A (also known as sodium-iodide symporter (NIS)), pendrin (PDS), dual oxidase type 2 (DUOX), thyroid peroxidase (TPO), thyroglobulin (Agic et al. 2007) and deiodinases type 1 (D1, DIO1), 2 (D2, DIO2) and 3 (D3, DIO3). In physiologic balance, T4 and T3 (3) regulate their own concentrations in the blood by negative feedback acting at the hypothalamic (1) and pituitary (2) levels (Ortiga-Carvalho et al. 2016). BPA, bisphenol A; DHA, docosahexaenoic acid; EPA, eicosapentaenoic acid; PBDE, polybrominated diphenyl ether.

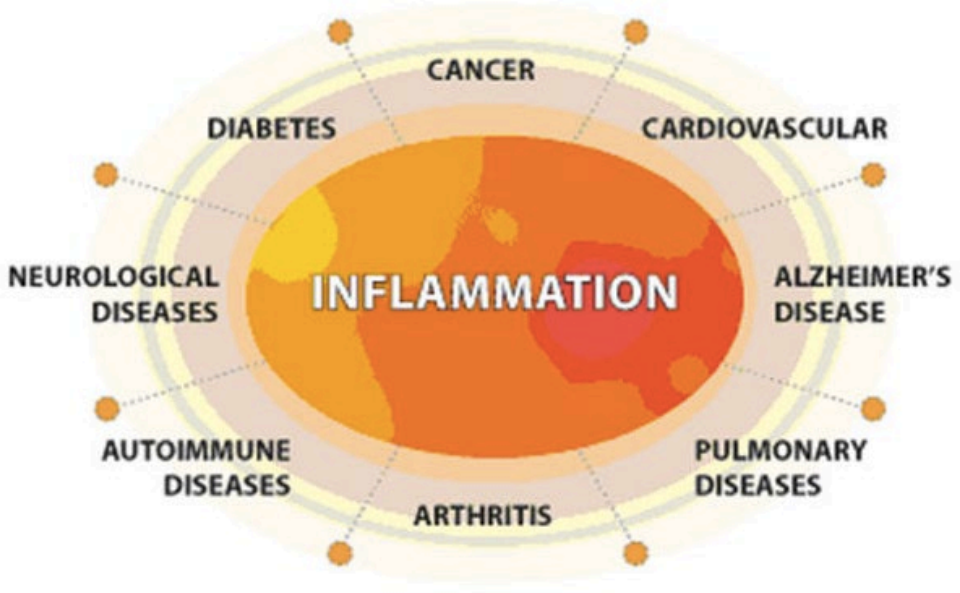
Thyroid Disorders – Problem 1- Conversion

- If T4 is not converting into T3, it is unable to relay its message to the cells no matter how much T4 is circulating through the blood.
- Unfortunately, the typical treatment option is to administer synthetic T4 like thyroxine or Synthroid to push TSH levels into the normal range, but this does nothing to address the actual problem.



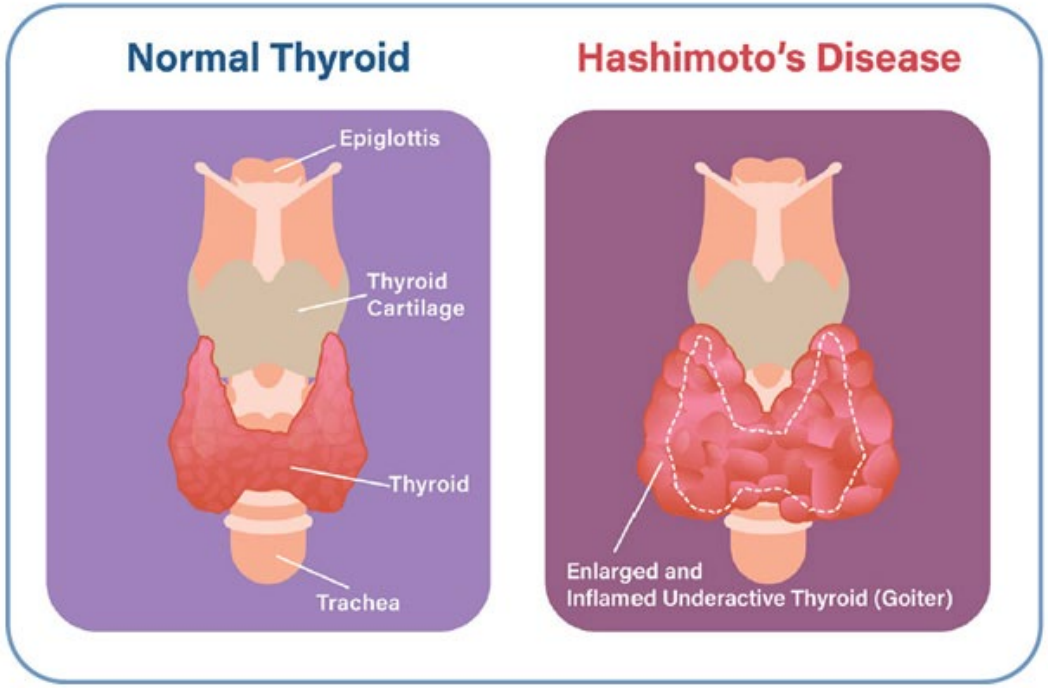
Thyroid Disorders Problem 2 - Inflammation

- Inflammation reduces the amount of active T3 available. For instance, high levels of inflammatory markers like interleukin-6, tumor necrosis factor, C-reactive protein, and interferon-alpha have all been associated with low levels of circulating T3.[7 8 9 10]
- Inflammation also affects thyroid receptors on the cellular membrane, making it harder or even impossible for them to dock and relay their message into the cell. In this way, thyroid hormone resistance is similar to insulin resistance, as the T3 is functional and circulating, but isn't able to communicate with the cells.



Thyroid Disorders Problem 3 – Hashimoto’s

Hashimoto's thyroiditis, is an autoimmune disorder in which the immune system attacks the thyroid gland, leading to inflammation and impaired thyroid function



Viruses lay latent until something tips the immune system

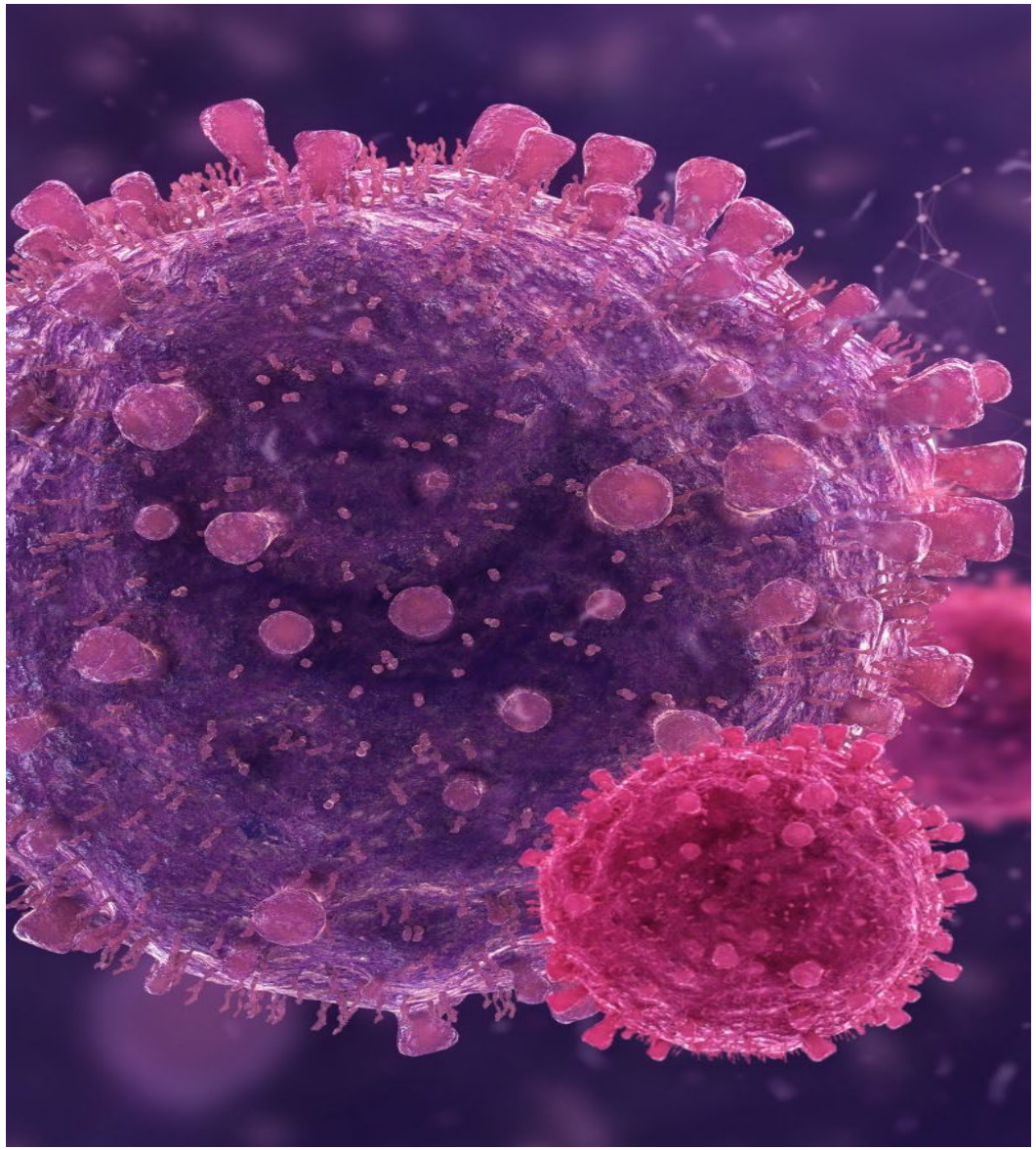
Hashimoto's is a great example of a latent virus destroying a function over time. Epstein-Barr (EBV) is a virus that causes mononucleosis (also known as "mono" or "glandular fever" in the UK).

A 2015 Polish study found the Epstein-Barr virus in the thyroid cells of 80 percent of people with Hashimoto's and 62.5 percent of people with Graves'.

Specific immune cells known as CD8+T cells are needed to fight off the Epstein-Barr virus. However, some individuals may have a low baseline level of these types of immune cells.CD8+T cells decrease with age, are lower in women, and are also decreased when vitamin D intake is low.

When levels of these fighter cells are insufficient, the Epstein-Barr virus may take up residence in the thyroid and essentially hijack it to help the virus hide and multiply. Add poor Lifestyle, SAD diet, deficiencies and toxicity. It's the perfect storm.

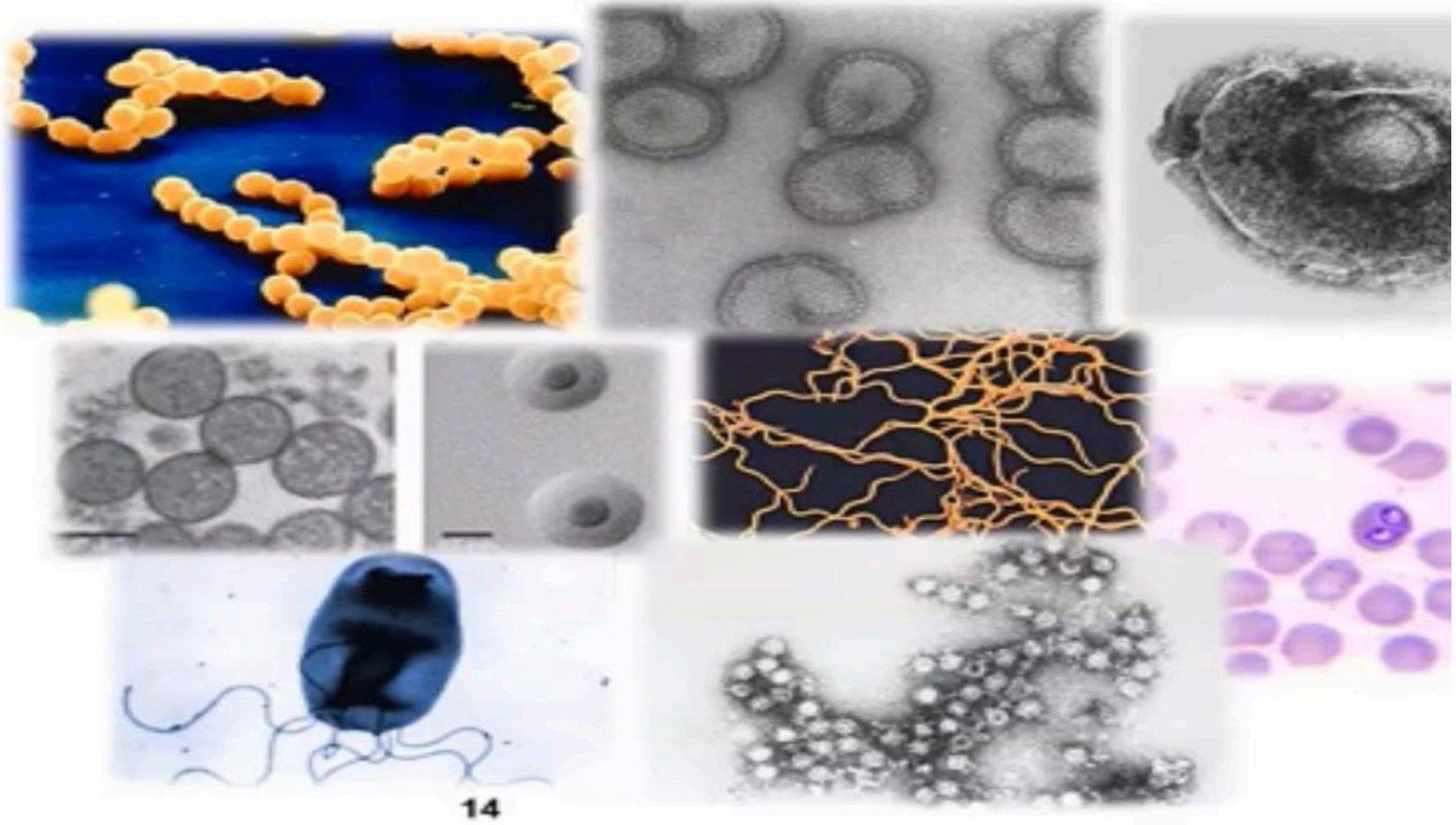
Begin PAMP & DAMP!



Probiotics can enhance the antitumor immune response of $CD8^+$T cells. It can play a synergistic antitumor role. On the one hand, its mechanism is through regulating intestinal flora, and on the other hand, through regulating the antitumor immune function of $CD8^+$T cells.

Certain Infections Are More Frequently Associated with Autoimmune Encephalopathies and Neuropsychiatric Symptoms

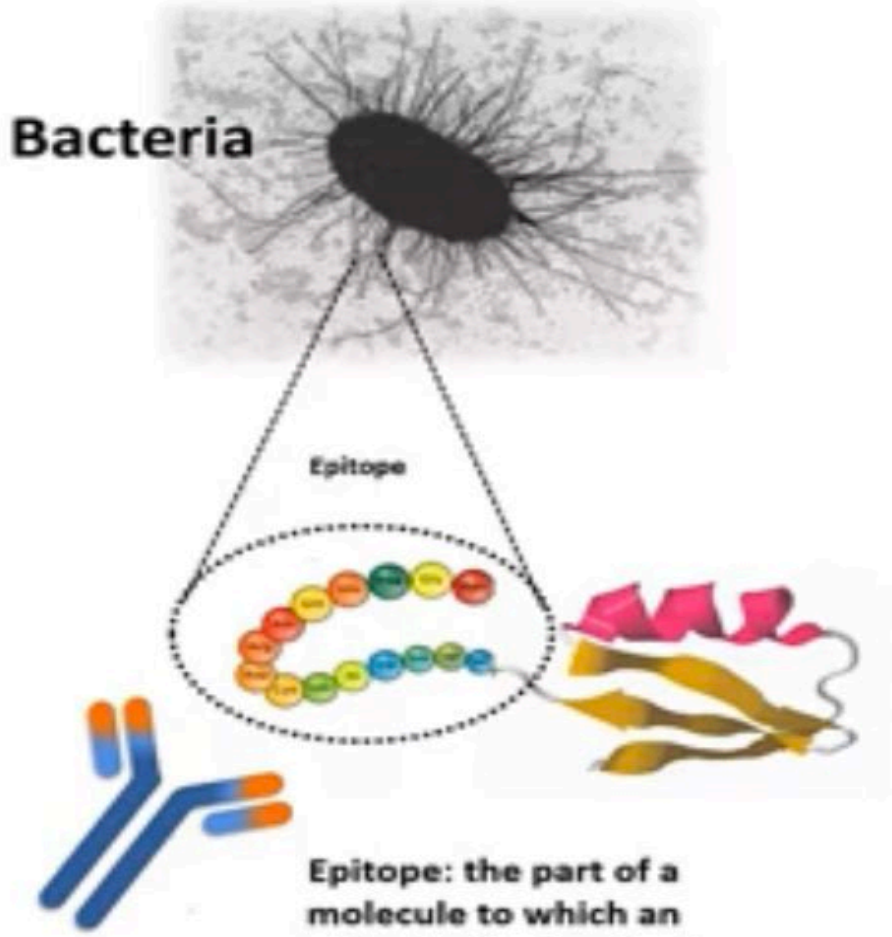
- Group A streptococci
- Influenza A
- Varicella (chickenpox)
- Mycoplasma
- Lyme disease
- Babesia
- Bartonella
- Coxsackie virus
- Others

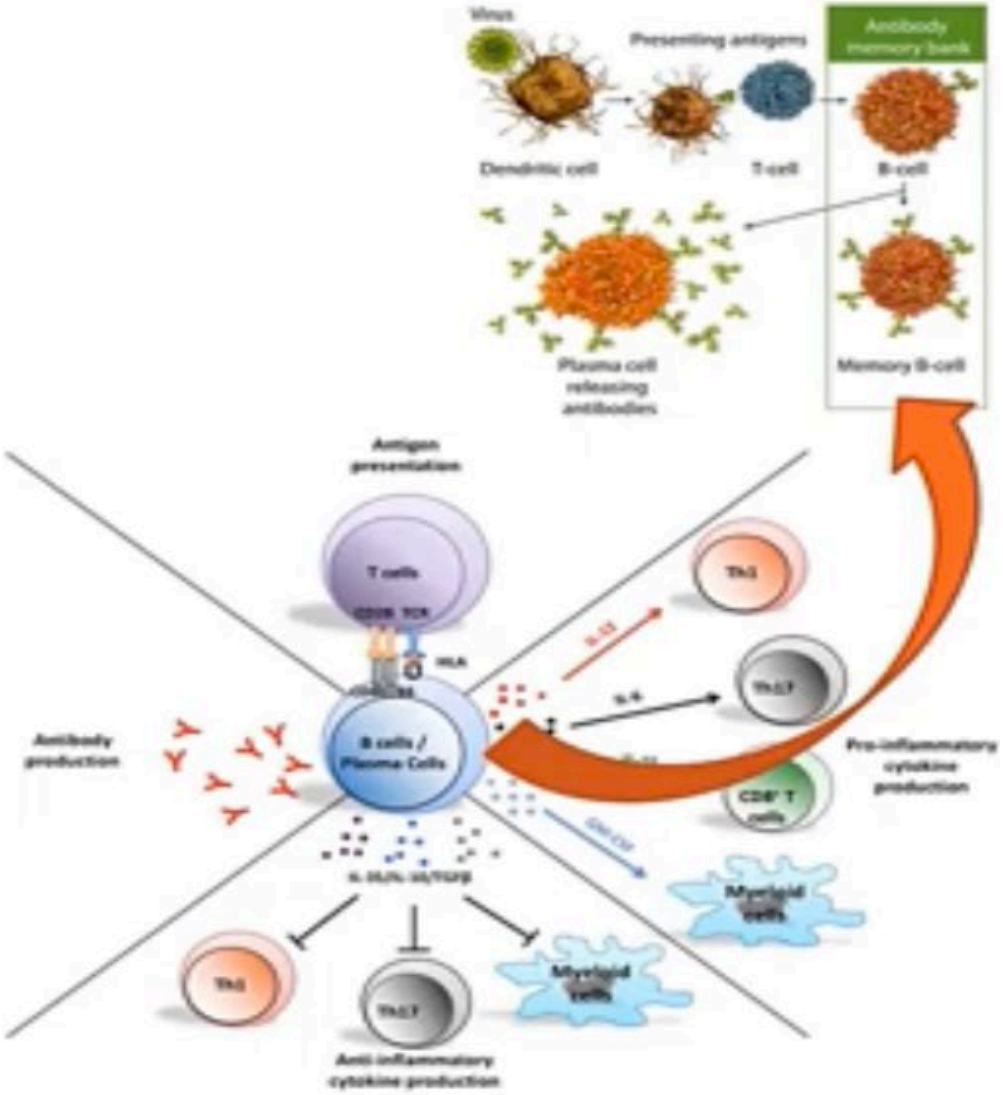


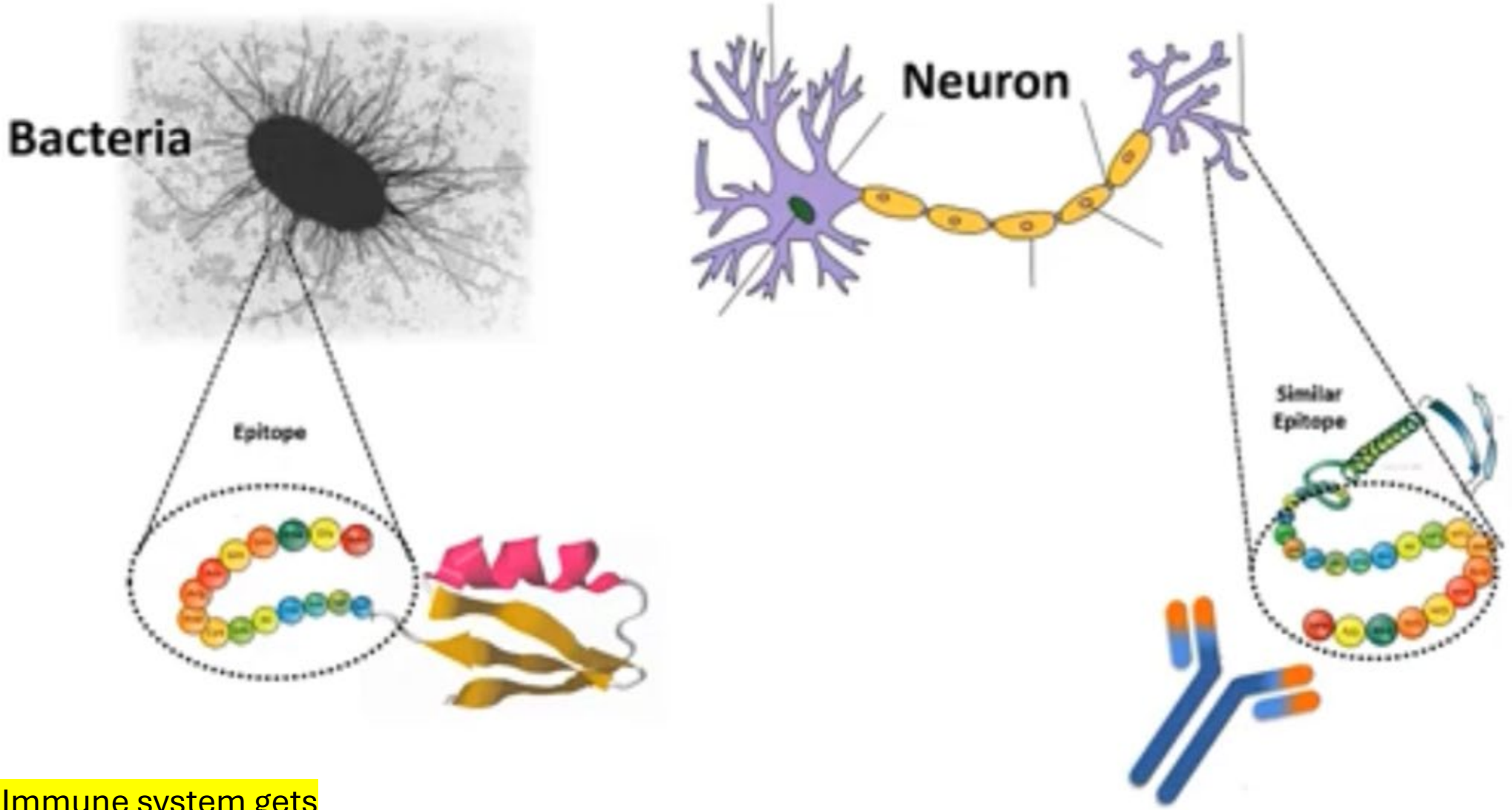
PAMP & DAMP

- Antibiotics unsupported
- Not enough “other” nutrition to overcome
- “Other” Toxicity in the way
- Environmental Influences – Childhood vaccine schedule

Antibodies Recognize "Epitopes" on Infectious Agents



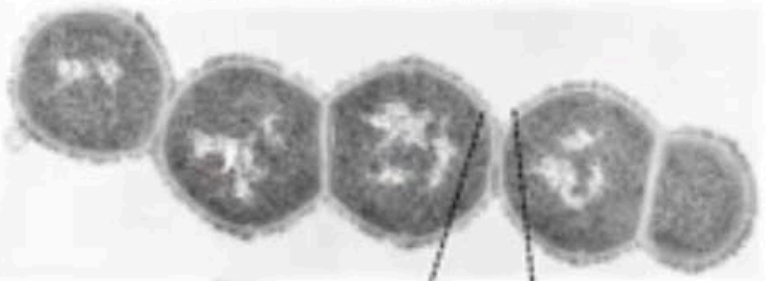


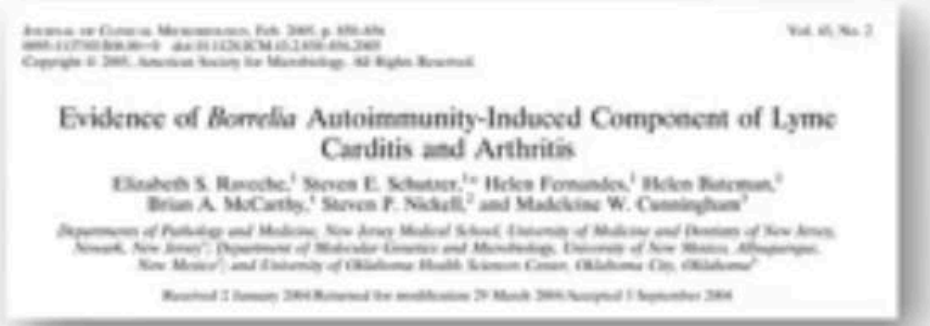


The Immune system gets Confused

Autoimmune response through "Molecular Mimicry"

Streptococcus pyogenes





Evidence of *Borrelia* Autoimmunity-Induced Component of Lyme Carditis and Arthritis

"Sequence similarity between the *B. burgdorferi* protein ***OspA*** and the *S. pyogenes* ***M5 protein***"

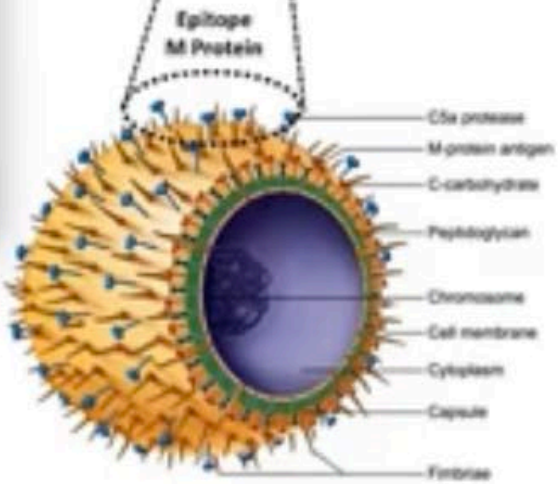


Image from Talaro & Talaro (2002)

"The IgM anti-B. burgdorferi … cross-reacted with S. pyogenes M and myosin, both of which share sequence homology with B. burgdorferi OspA, ***suggesting a role for molecular mimicry in the generation of these Ab reactivities***

Thyroid Ranges and Patterns

Thyroid Levels Chart

Test	Standard Reference Range	Optimal Reference Range
TSH	0.45 - 4.12 mIU/L	0.5 - 2.5 mIU/L
T3	0.8 - 2 ng/ml	0.8 - 2 ng/ml
T4	4.5 - 9.8 µg/dl	4.5 - 9.8 µg/dl
FREE T4	0.93 - 1.7 ng/dl	1 - 1.7 mIU/L
FREE T3	2.8 - 4.0 pg/ml	3 - 4.0 mIU/L
REVERSE T3	8 - 25 ng/dl	Less than 15 ng/dl
Anti-TPO	Below 35 IU/mL	Below 35 IU/mL

	TPO antibodies (IU/mL)	TSH (mIU/L)	Free T4 (ng/dL)
Hashimoto's	Detectable: 35+ At-Risk: 500+		
Sluggish Thyroid		4.5 to 7	Normal (0.8 to 1.8)
At-Risk Hashimoto's and Sluggish Thyroid	500+	4.5 to 7	Normal (0.8 to 1.8)
Hypothyroid		4.5+	<0.8
At-Risk Hashimoto's and Hypothyroid	500+	4.5+	<0.8

Advanced Bloodwork

EBV/Strep

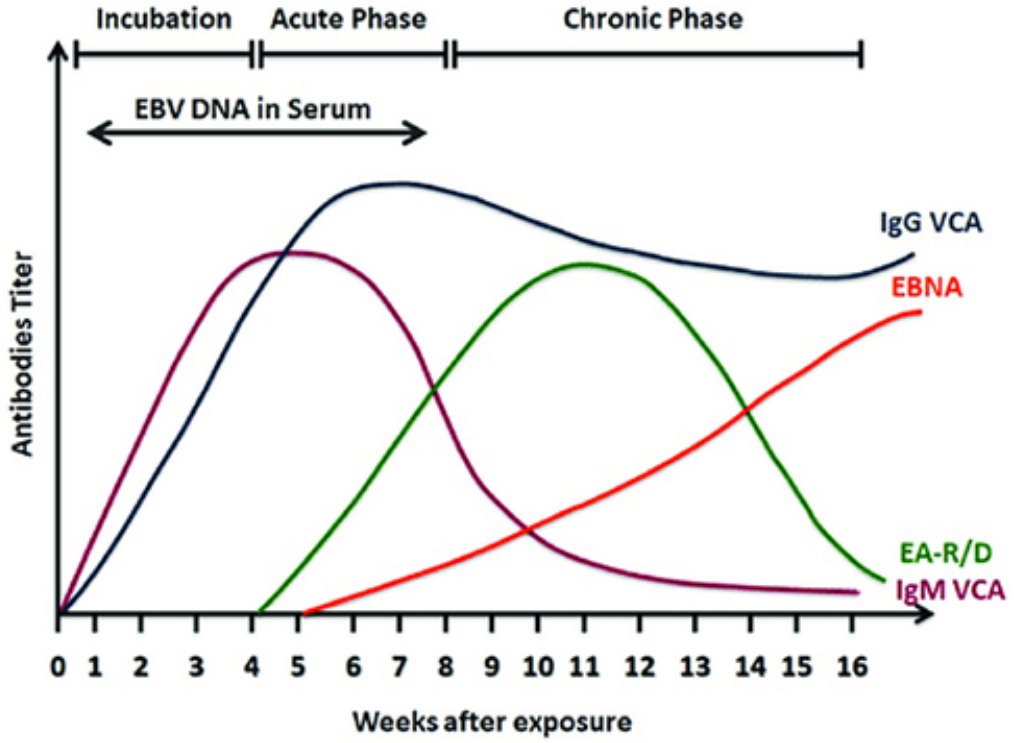
EBV - STREP

Understanding the tests - VCA IgG

- Since this is the antibody that most of the population on the globe carries, positive VCA IgG alone does not mean much. It just means you have had exposure to EBV, and potentially, it can affect your health in the future. You may be perfectly healthy too. VCA IgG can stay positive all throughout your life. However, what if your VCA IgG is triple digits or “>600”? In that case, it is likely that you have chronic active EBV, but it is not reactivated at this time. Some people take very good care to maintain a healthy stress level, eat a balanced diet, get good restorative sleep, and live a healthy lifestyle in general, and they do not reactivate for decades.
- *NOTE ON HOW LABS RECORD >600

Understanding the tests - EA- D IgG

- EA-D IgG is the most important marker to look for to distinguish between past and current infection; EA-D IgG can mean that this is your initial infection OR that this is your current reinfection. This is the antibody that is most often left out of the EBV lab panel by doctors, so it is really important that you make sure your doctor does not skip it, whether this is your first or subsequent test.

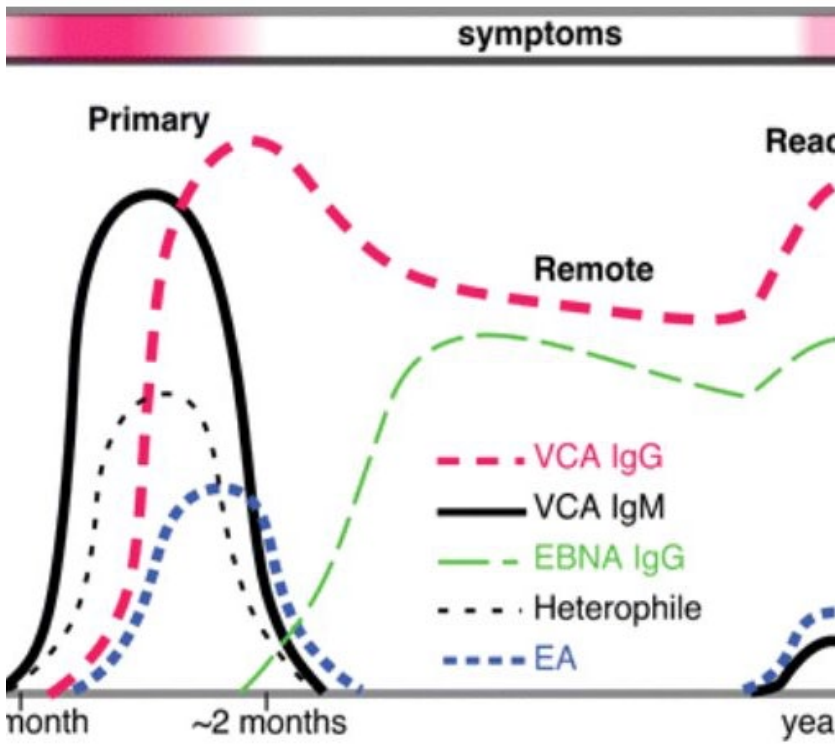


Patterns to Remember

- As you see, **VCA IgM** is flared up in the very initial (primary) infection, within the third week I would say, and then it can show up again years after. But it is really rare or too low to be positive at recurrence.

- On the other hand, **VCA IgG** starts showing after the first month or so of initial infection and then stays elevated for life. Again, this is the antibody most of the global population has elevated. This alone does not mean you have a chronic condition related to EBV. But as I mentioned, if it is very high, then it is a red flag, and you want to dig a little deeper.

- **EBNA IgG** flares up only around/after the two first months of initial infection—this antibody also tends to stay elevated for the rest of your life. As I mentioned, positive EBNA IgG along with positive VCA IgG is not enough to suggest you have an active infection now unless you add EA-D IgG. You also have to consider your timeline, medical history, mono history, and symptoms. And this can simply mean that you are in between reactivations (this can last for years and decades).



From the Newfoundland and Labrador Public Health Laboratory.

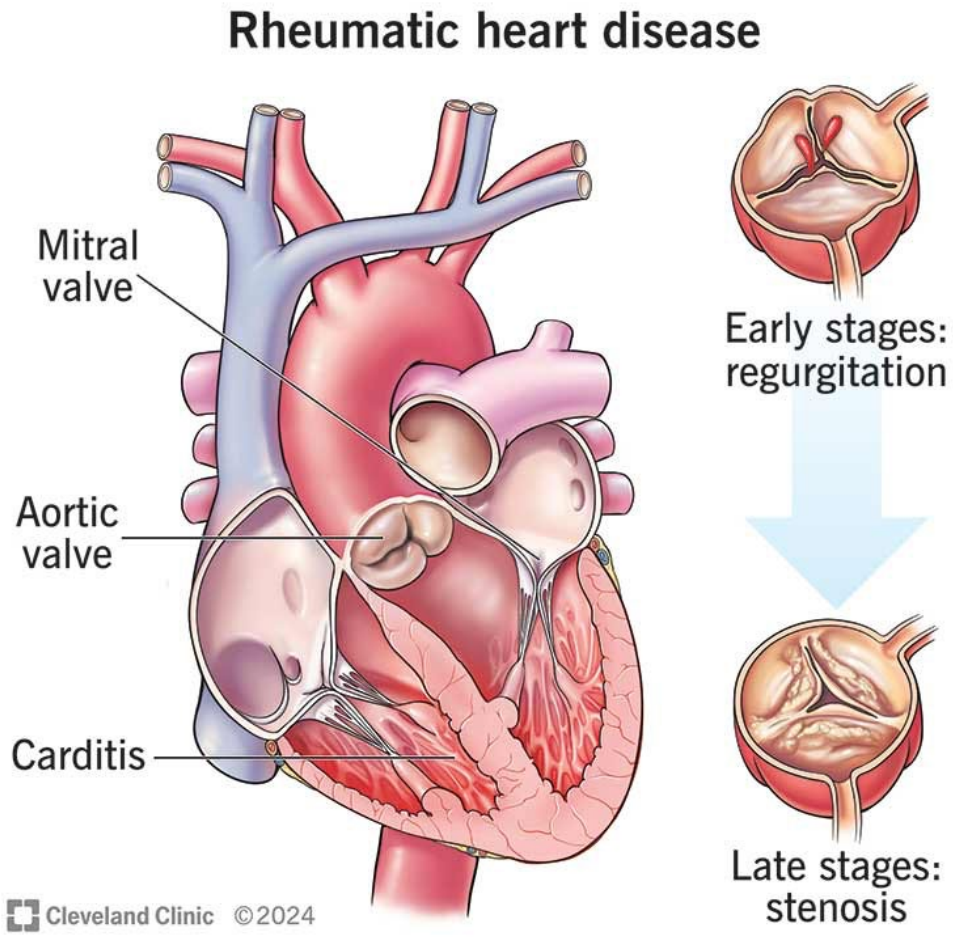
Patterns to Remember

Test results most likely indicate the following:

VCA-IgM	VCA-IgG	EA-D, IgG	EBNA, IgG	Possible Interpretation
Negative	Negative	Negative	Negative	No infection, symptoms due to another cause, susceptible to EBV infection
Positive	Positive	Negative	Negative	Early, primary infection
Negative or positive	Positive	Positive	Negative	Active infection, though EA-D IgG may persist for life in about 20% of people
Negative	Positive	Negative	Positive	Past infection
Negative	Positive	Positive	Positive	May indicate reactivation of virus

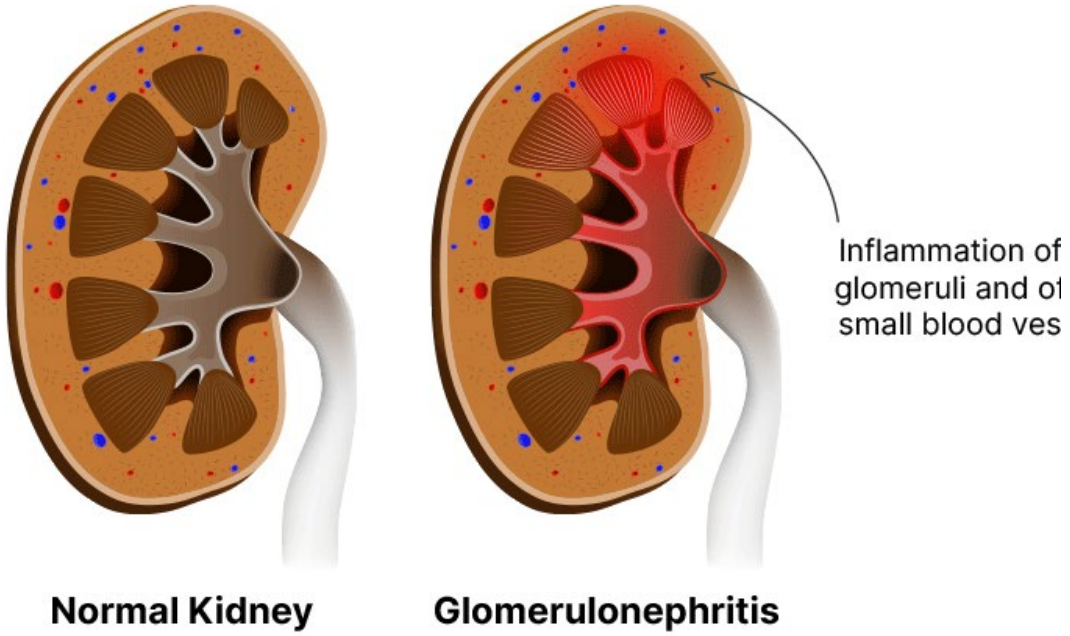
Strep and Rheumatic Fever

- One of the most well-known examples of molecular mimicry related to Strep is **rheumatic fever**, a serious complication that can follow untreated or poorly treated strep throat.
- The immune system's response to Streptococcus pyogenes (GAS) results in the production of antibodies that, through molecular mimicry, attack the heart (especially the **heart valves**), joints, brain, and skin.
- In this case, **M proteins** on the Streptococcus bacteria resemble proteins in human heart tissues, leading to inflammation and scarring of the heart valves (rheumatic heart disease).



Post-streptococcal glomerulonephriti

- Another condition linked to strep and molecular mimicry is **post-streptococcal glomerulonephritis**, where the immune system attacks the kidneys following a strep infection.
- The immune complexes formed against the bacterial antigens get trapped in the kidneys, leading to inflammation and kidney damage.



Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infections (PANDAS)

- In some children, a strep infection can trigger **PANDAS**, a condition in which the immune response cross-reacts with neurons in the brain, specifically the **basal ganglia**, a region involved in movement and behavior regulation.
- This can result in sudden-onset **obsessive-compulsive disorder (OCD), tics, and other neuropsychiatric symptoms**. The theory of molecular mimicry here involves antibodies created against strep bacteria that mistakenly target neurons.

Chronic Ear Infections in Kids

When a baby is young, they have a lot of Naïve T cells…

However, reoccurring infections begin to create senescent cells…

Oxidative stress, inflammation, immune dysregulation

Most cases I have seen begin with underlying strep

KILL MENTALITY

Antibiotics…antibiotics…antibiotics…

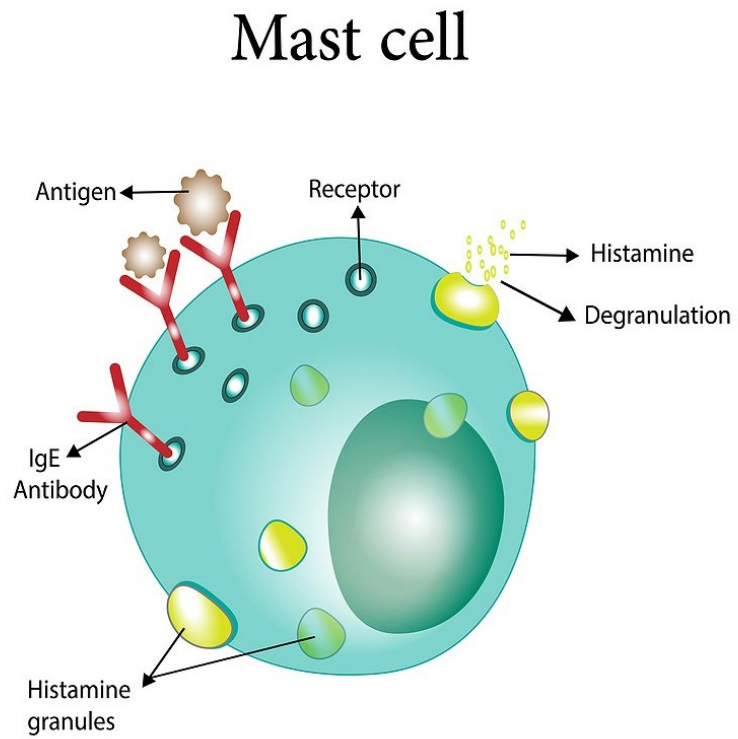
532	$5.00	Complete Blood Count (CBC) With Differential (005009)
513	$6.00	Antistreptolysin O (ASO) Antibodies (006031)
1054	$36.00	Anti-DNase B (Streptococcal) Antibodies (096289)

Advanced Bloodwork

MCAS

MCAS Mast Cell Activation Syndrome

- Mast cells are a type of white blood cell, specifically part of the immune system and are predominantly found in connective tissue. They are involved in the body's response to allergens and pathogens. When triggered by an allergic reaction or an immune response, mast cells release substances like histamine, cytokines, and leukotrienes, which can cause inflammation, itching, and other allergic symptoms. These cells play a crucial role in defending the body against parasites and in wound healing, but their overactivation can lead to allergic diseases like asthma, hay fever, and eczema.
- **MC have over 200 different types of receptors**. This is how they can respond to everything that's happening outside and inside you.



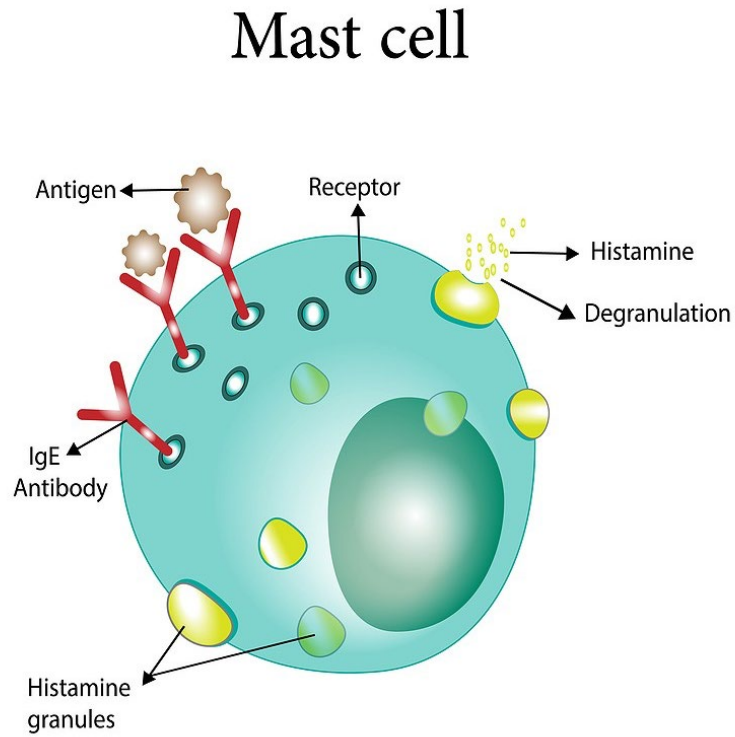
MCAS Mast Cell Activation Syndrome

When the body is exposed to something, mast cells release chemicals called mast cell mediators.

- Example of mast cell mediators: Histamine

Mast cells normally launch a response and then stabilize again.

Mast cells have little granules that store these mediators. When they are released, it's called **degranulation**

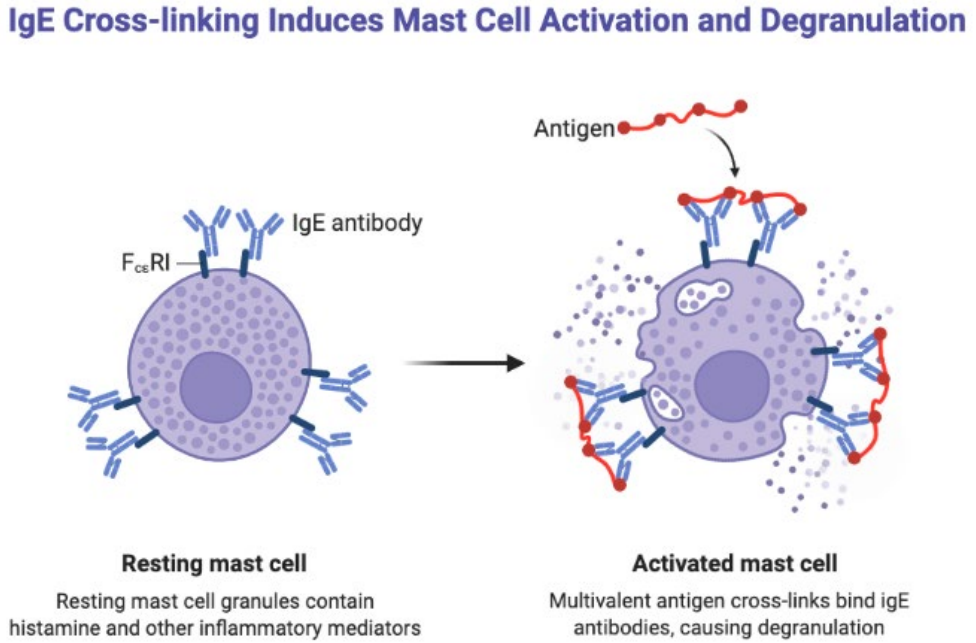


MCAS - **Degranulation**

Here are just a few of the other types of mediators released in degranulation and what they do:

- **cytokines** – assist in inflammation increase, inflammation reduction, cell signaling
- **leukotrienes** – play a role in blood vessel dilation or constriction
- **prostaglandins** – has a role in blood vessel dilation or constriction
- **tryptase** – play a role in allergic responses
- **neurotransmitters** and **neuropeptides** – has a role in nervous system signaling
- **platelet-activating factor** – play a role in immune defense, inflammation, anaphylaxis

- All of these can work to keep you healthy.



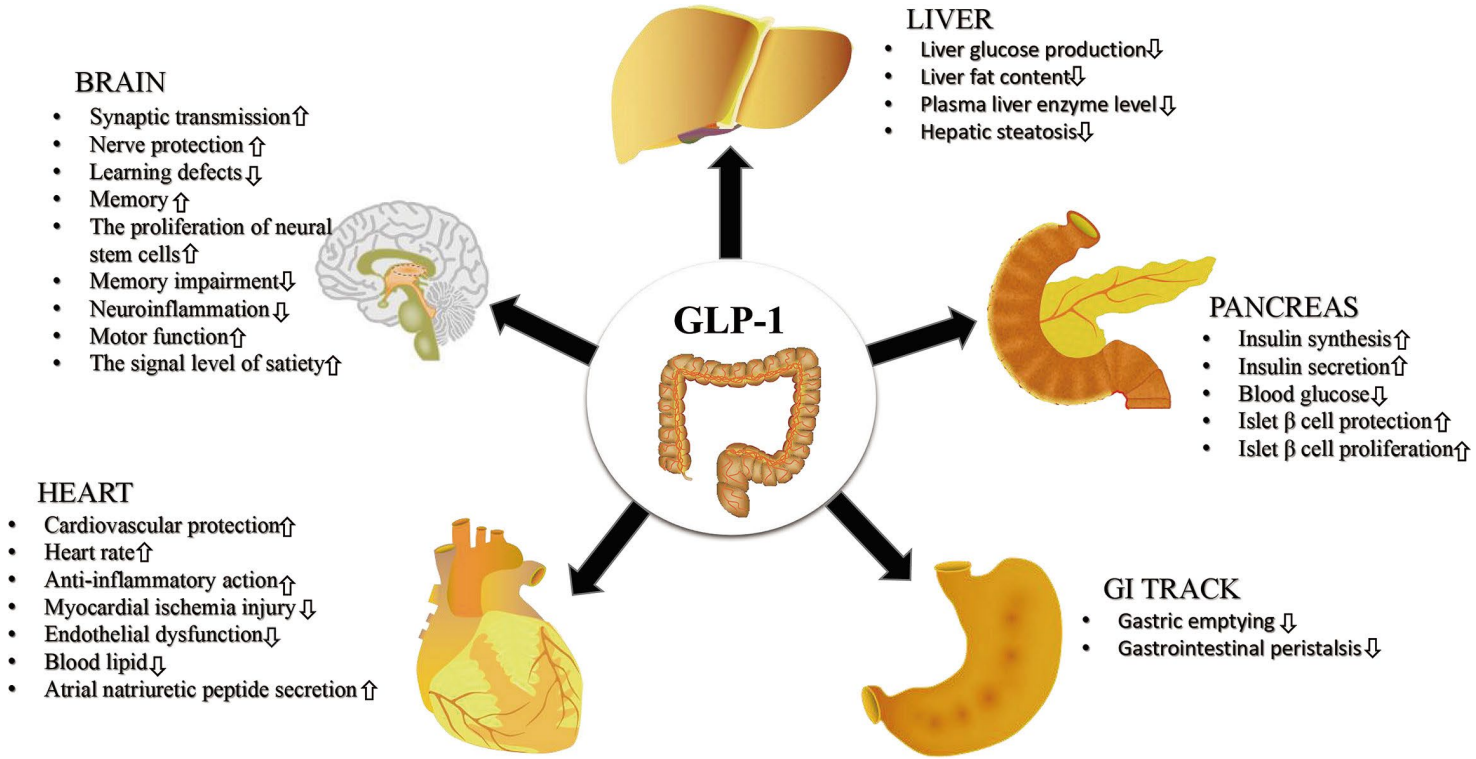
MCAS - **Degranulation**

Over time, constant triggers can cause your mast cell receptors to become overly sensitive and hypervigilant. And because of this, your mast cell mediators can be released at the wrong times or at very high levels.

Advanced Bloodwork

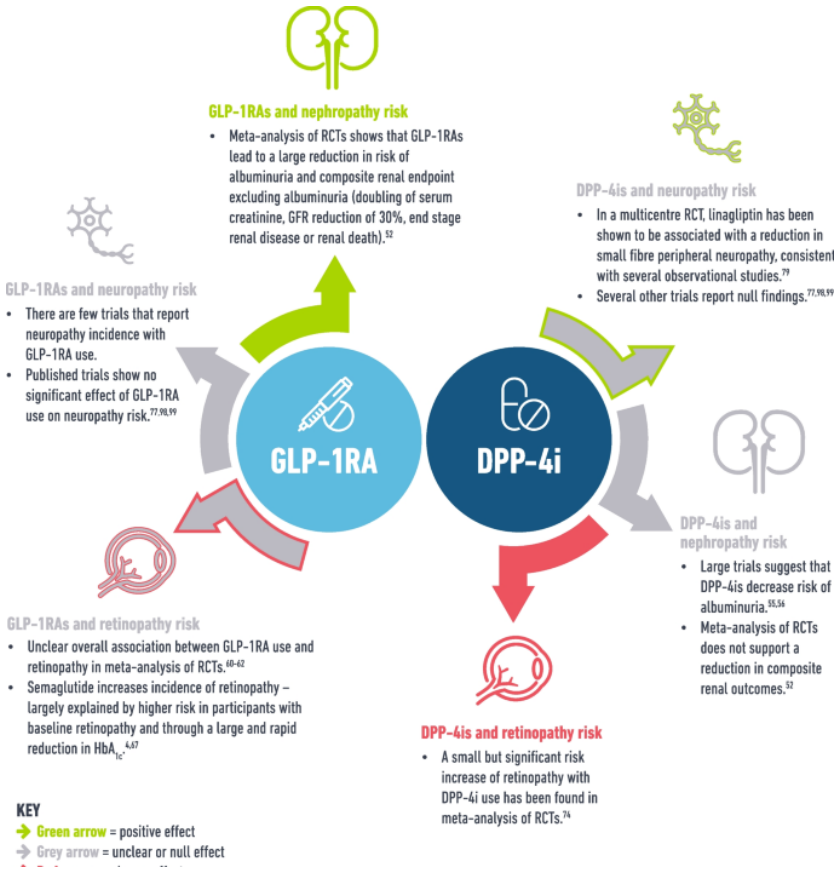
Metabolic

Semaglutide
GLP-1 Agonist (activate)



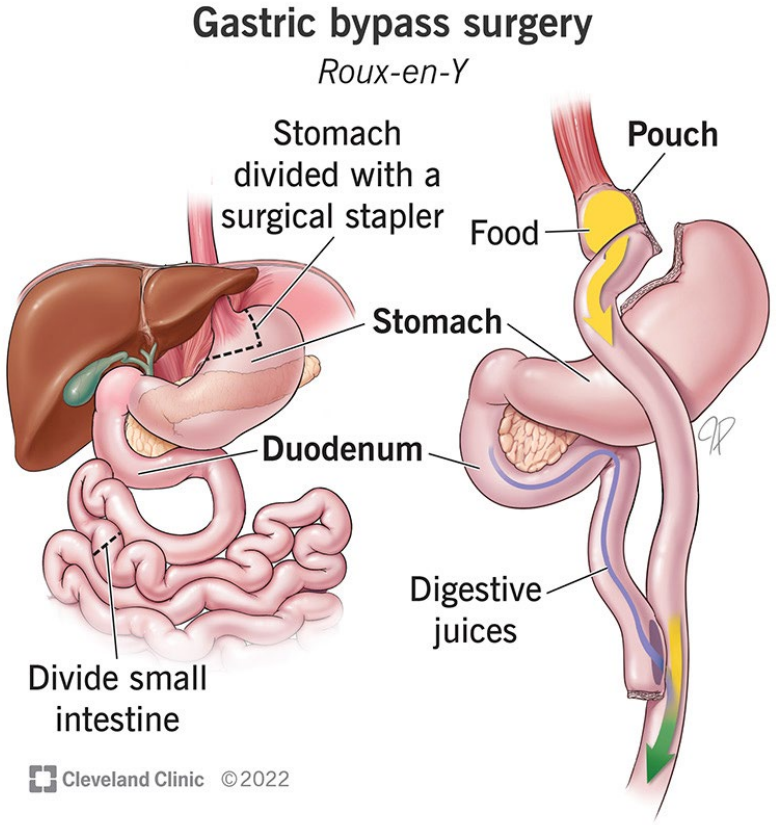
What are Incretins?

- Incretins are a group of metabolic hormones that play a significant role in regulating insulin secretion and blood glucose levels. The two most well-known incretins are **Glucagon-Like Peptide-1 (GLP-1)** and **Gastric Inhibitory Polypeptide (GIP)** (also known as Glucose-Dependent Insulinotropic Polypeptide).
- Incretins are particularly important in the context of type 2 diabetes. People with type 2 diabetes often have impaired incretin responses, leading to difficulties in managing blood glucose levels. This has led to the development of medications that mimic or enhance the effects of incretins, such as GLP-1 receptor agonists (e.g., exenatide, liraglutide) and **DPP-4 inhibitors (e.g., sitagliptin), which prolong the action of incretins in the body.**



Gastric Bypass

- Gastric bypass surgery, particularly Roux-en-Y gastric bypass (RYGB), is a well-known procedure for weight loss and has significant effects on the body's metabolism, including the regulation of incretin hormones. Incretins are a group of metabolic hormones, including glucagon-like peptide-1 (GLP-1) and gastric inhibitory polypeptide (GIP), that enhance insulin secretion in response to food intake.



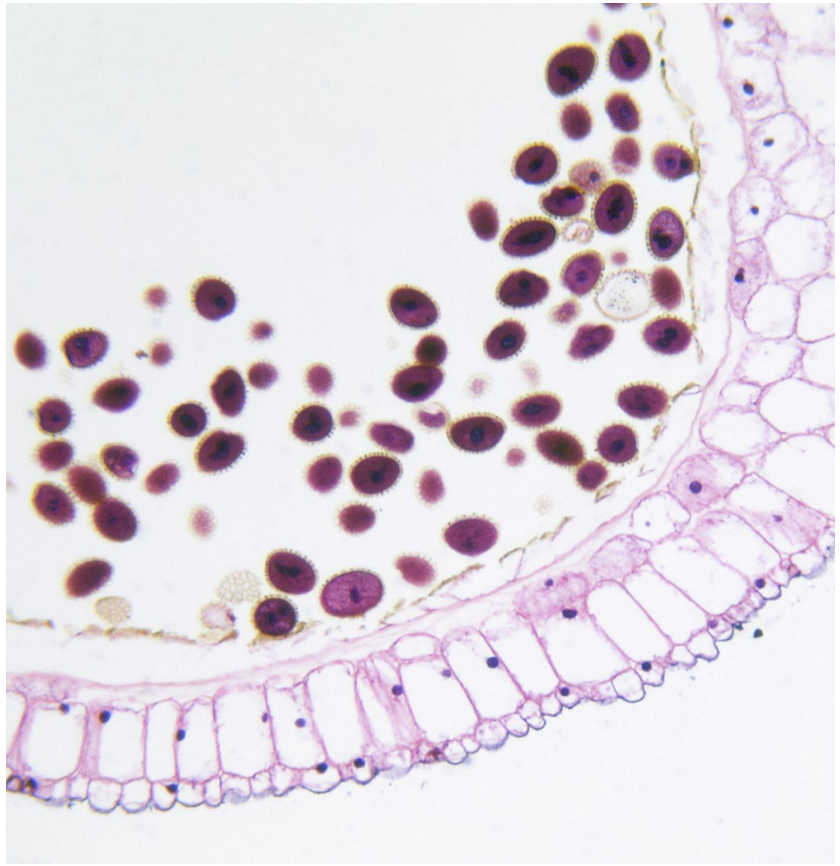
Gastric Bypass Findings

1. **Increased Incretin Response:** After gastric bypass surgery, there's a notable increase in the secretion of incretins, particularly GLP-1. This heightened incretin response plays a crucial role in improving insulin secretion and glucose homeostasis, contributing to the remission of type 2 diabetes often observed after the surgery.
2. **Rapid Glycemic Control:** The enhanced incretin effect post-surgery leads to rapid improvement in blood sugar levels. This effect is often seen before significant weight loss occurs, suggesting that the surgery has direct metabolic effects beyond just weight reduction.
3. **Altered Gut Hormone Profile:** Gastric bypass surgery changes the anatomy of the gastrointestinal tract, which alters the release of various gut hormones, including incretins. The rerouting of food away from the upper part of the small intestine, where GIP is normally secreted, results in a shift towards increased GLP-1 secretion.
4. **Sustained Metabolic Benefits:** The increase in incretin levels post-surgery contributes to long-term improvements in metabolic health. This includes sustained weight loss, improved insulin sensitivity, and ongoing glucose control, which are crucial for managing and potentially reversing type 2 diabetes.
5. **Potential Therapeutic Insights:** Understanding the incretin response to gastric bypass surgery has informed the development of incretin-based therapies, such as GLP-1 receptor agonists, which are now widely used in the treatment of type 2 diabetes and obesity.
6. All of this occurred within WEEKS of the surgery, even before weight loss occured

Gastric Bypass – Biological Mechanisms

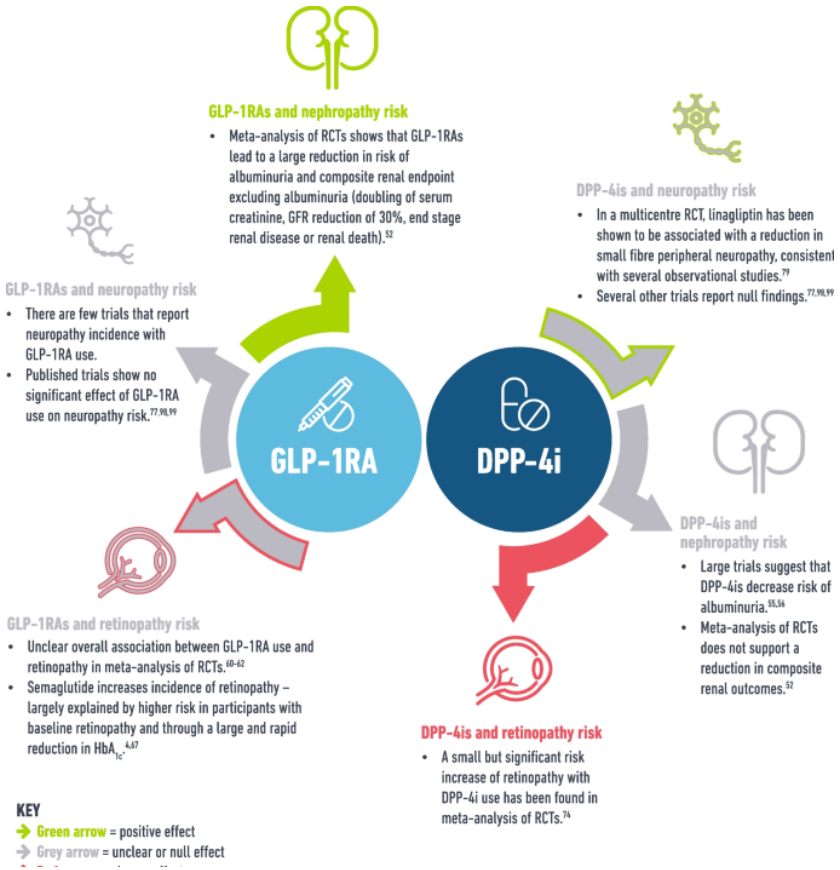
The increased secretion of incretins, particularly GLP-1, after gastric bypass surgery is primarily attributed to the rerouting of food, which results in direct and enhanced stimulation of L-cells in the distal small intestine. This is coupled with changes in gut hormone signaling, increased L-cell activity, alterations in the gut microbiota, and enhanced nutrient sensing. These combined effects lead to the pronounced increase in GLP-1 levels, contributing to the improved glucose metabolism and other metabolic benefits observed after surgery.

GUT HEALTH MATTERS!



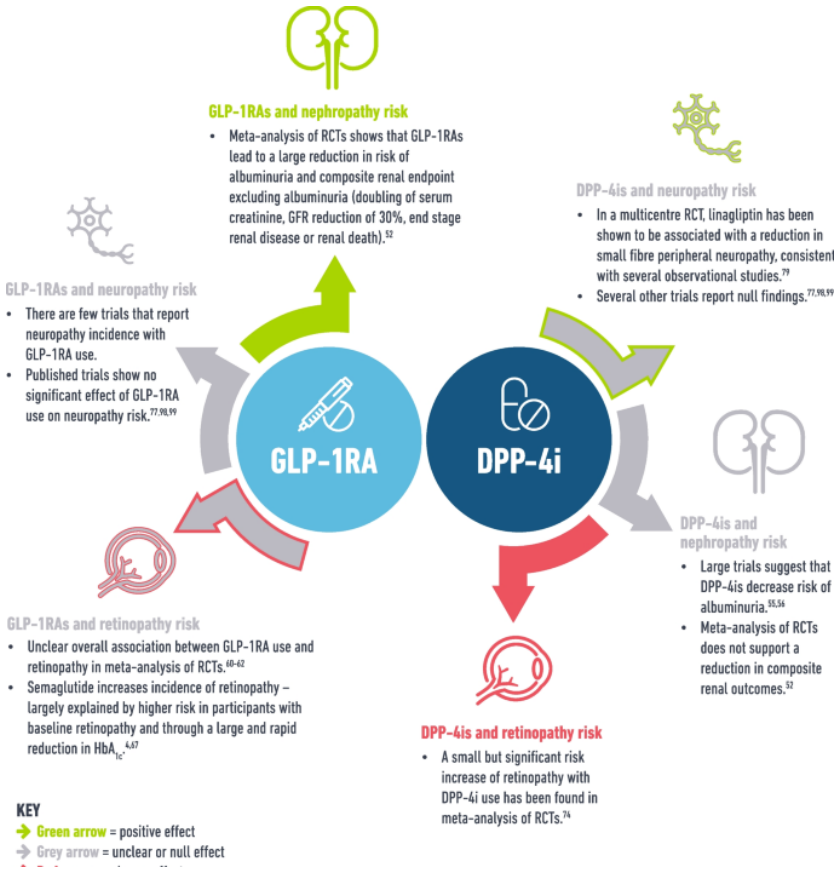
What are Incretins?

- GLP-1 -**Glucagon-Like Peptide-1 (GLP-1)** is an incretin hormone that plays a key role in regulating blood glucose levels and overall energy balance. It has several important functions, particularly in relation to insulin secretion, appetite control, and glucose metabolism.
- Reduces glucagon (which is the hormone that tells liver to release glucose to the liver)
  - Actually, suppresses glucagon
- Delays gastric emptying
- CNS direct affect to tell the brain you're not hungry



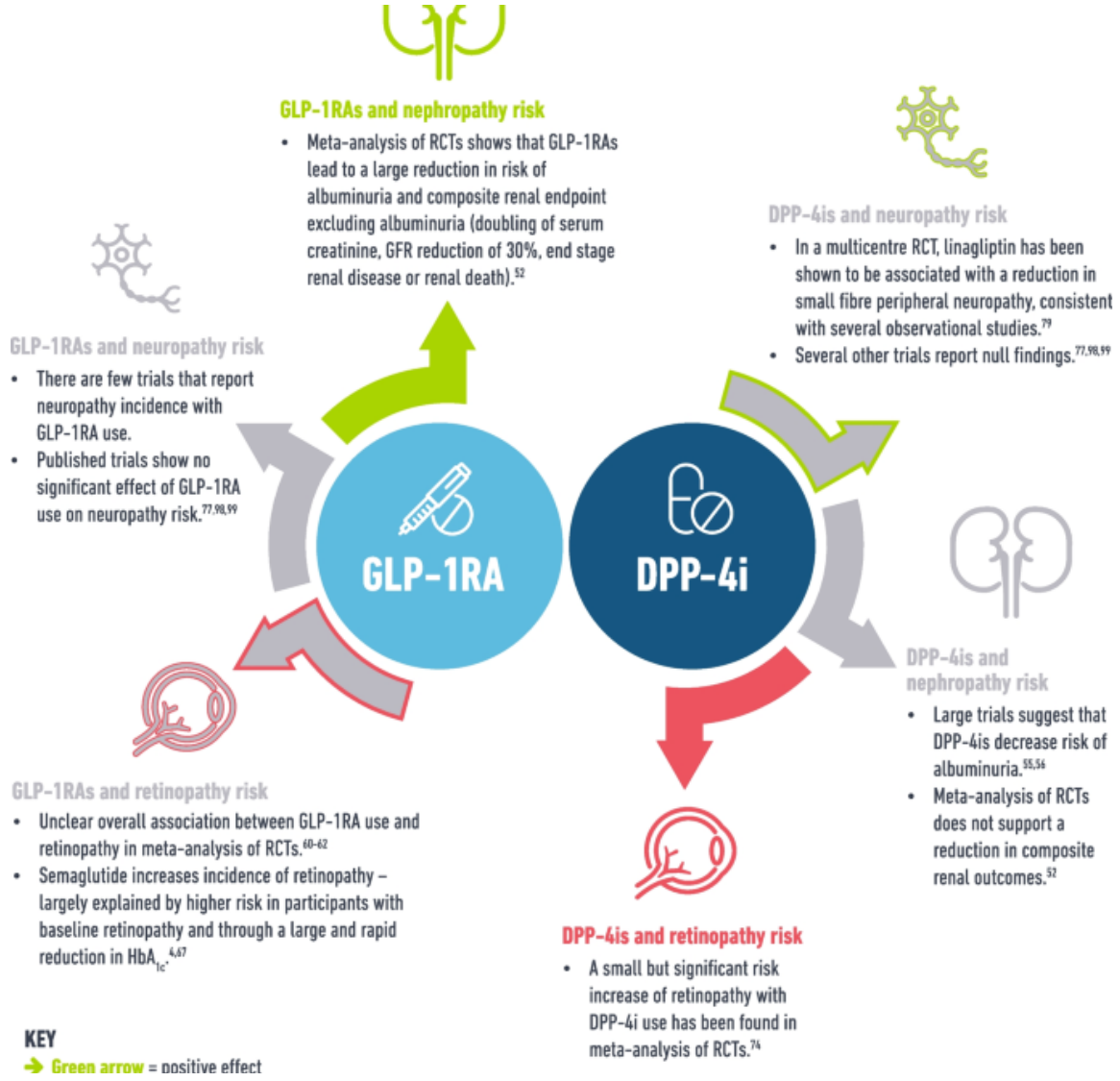
What are Incretins?

- **Stimulation of Insulin Secretion**:
  - GLP-1 enhances the secretion of insulin from the beta cells of the pancreas in a glucose-dependent manner. This means that GLP-1 stimulates insulin release only when blood glucose levels are elevated, such as after a meal. This helps lower blood glucose levels effectively without causing hypoglycemia.
- **Inhibition of Glucagon Secretion**:
  - GLP-1 suppresses the secretion of glucagon, a hormone produced by the alpha cells of the pancreas. Glucagon typically raises blood glucose levels by promoting the release of glucose from the liver. By inhibiting glucagon, GLP-1 helps reduce the production of glucose by the liver, further contributing to lower blood glucose levels.
- **Slowing of Gastric Emptying**:
  - GLP-1 slows the rate at which food moves from the stomach to the small intestine. This delayed gastric emptying can help control postprandial (after-meal) blood glucose spikes and also contributes to feelings of fullness, which can reduce overall food intake.
- **Promotion of Satiety and Reduction of Appetite**:
  - GLP-1 acts on the brain, particularly the hypothalamus, to promote feelings of satiety (fullness) and reduce appetite. This effect can lead to decreased food intake and is one reason why GLP-1-based therapies can be effective for weight management in people with obesity or type 2 diabetes.
- **Cardiovascular Effects**:
  - Some studies suggest that GLP-1 may have beneficial effects on the cardiovascular system, including improving heart function and protecting blood vessels.



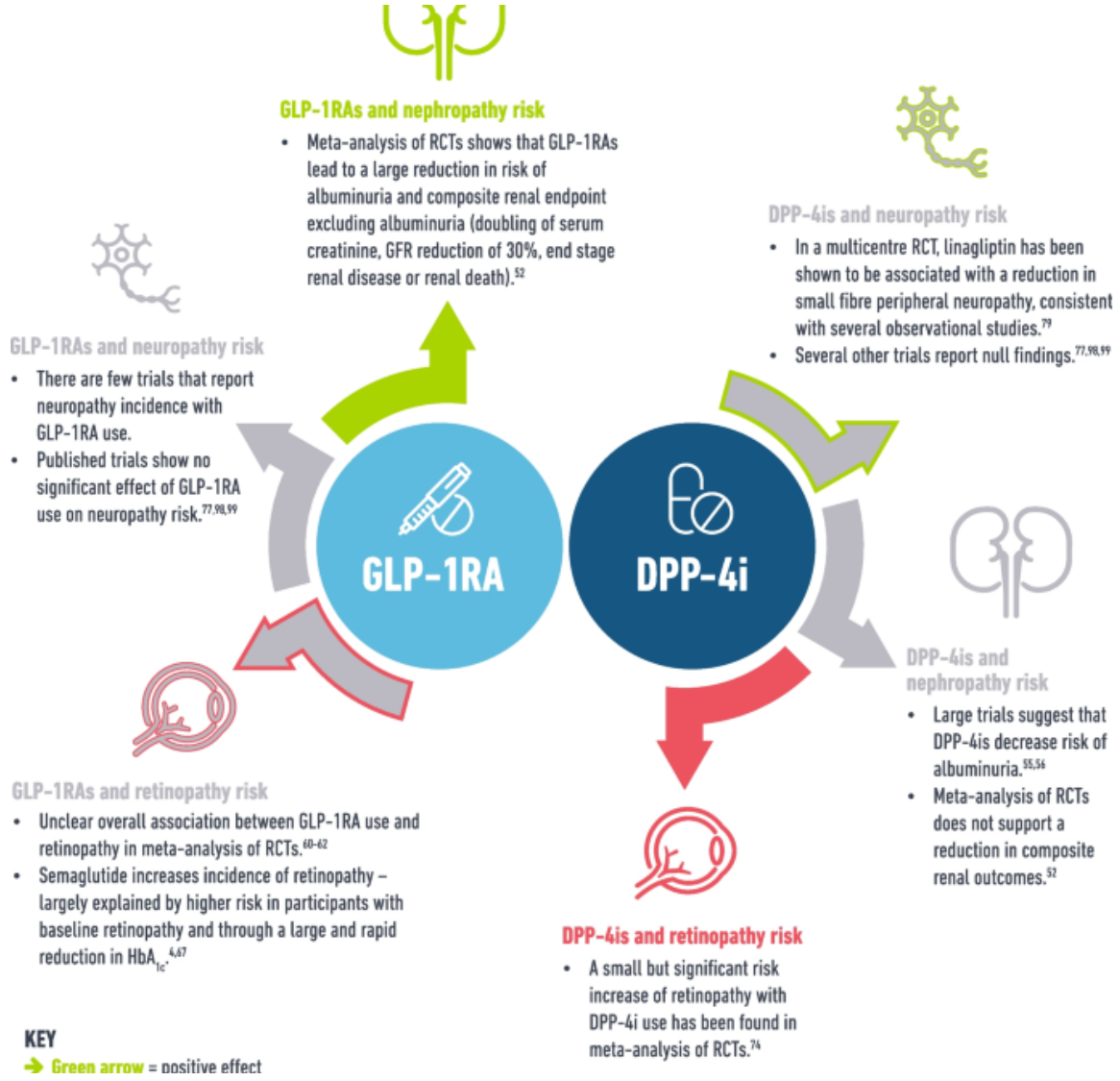
What are Incretins?

- GIP - **Gastric Inhibitory Polypeptide (GIP)**, also known as **Glucose-Dependent Insulinotropic Polypeptide**,
- One of the two main incretin hormones (the other being GLP-1). GIP plays a significant role in regulating insulin secretion and glucose metabolism.
- GIP - Insulin secretagogue - Increases the body's ability to produce more insulin to a high glucose load.
    - It enhances the secretion of insulin from the beta cells of the pancreas, but only when blood glucose levels are elevated
    - Potential role in fat metabolism



What are Incretins?

- **DPP-4** is an enzyme that breaks down incretin hormones like GLP-1 and GIP, which are important for insulin secretion and blood glucose regulation.
- DPP-4 levels and activity are determined by a combination of genetic factors, age, body weight, inflammatory states, hormonal regulation, pathological conditions, medications, and lifestyle factors.



What are Incretins?

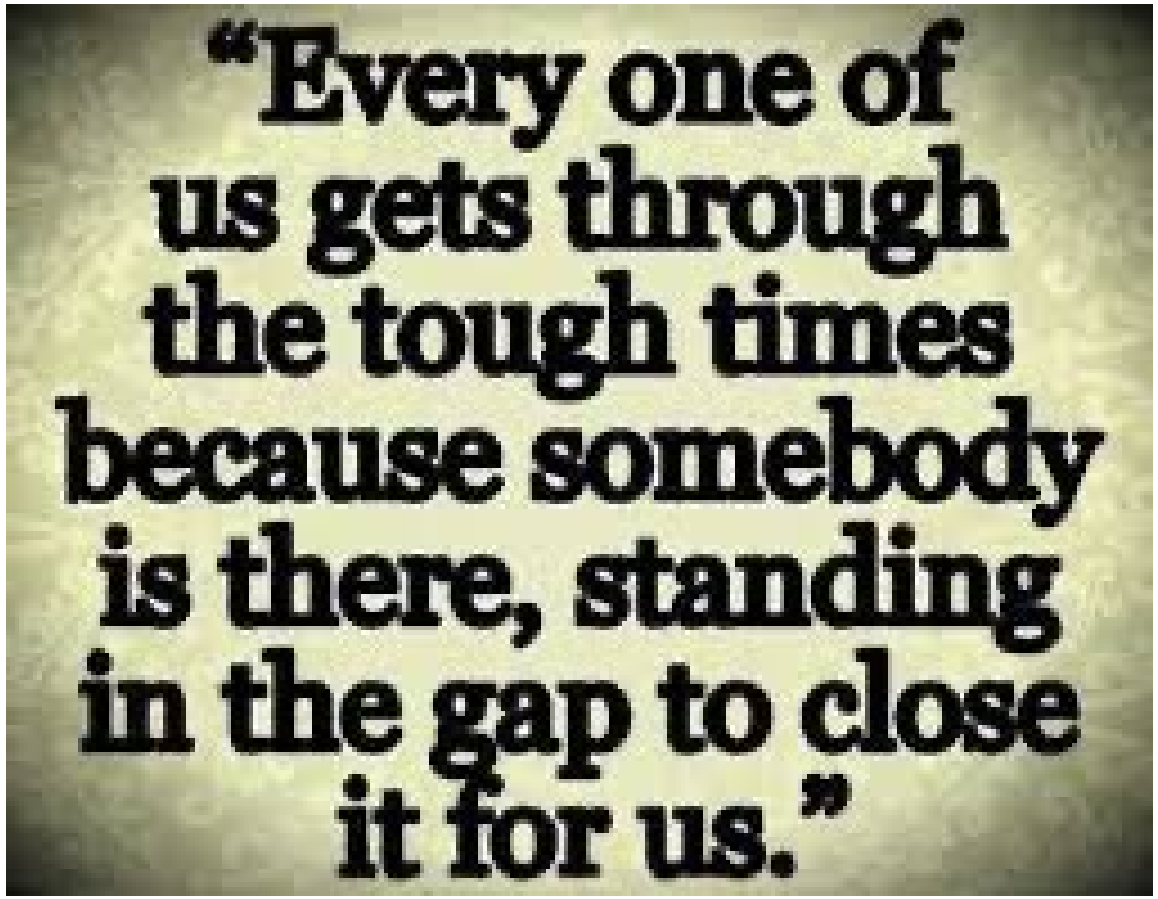
- **Cholecystokinin (CCK)**
- **Released by the Duodenum**: CCK is released by the I cells in the duodenum (the first part of the small intestine) in response to the presence of fats and proteins.
- **Stimulates Digestion**: CCK stimulates the release of digestive enzymes from the pancreas and bile from the gallbladder, which are essential for the digestion of fats and proteins.
- **Satiety Signal**: CCK also contributes to satiety by acting on the brain to reduce hunger and slow gastric emptying, similar to PYY.
    - Stimulates Gallbladder to produce bile
    - Bile also activates proteolytic enzymes to break down proteins
    - Fat helps with break down proteins to amino acids and more are seen In the blood stream

- **Peptide YY (PYY)**
- **Released Post-Meal**: PYY is secreted by the L cells in the ileum and colon in response to food intake, particularly after a meal rich in fats and proteins.
- **Satiety and Appetite Reduction**: PYY acts on receptors in the brain, especially in the hypothalamus, to reduce appetite and promote satiety. This helps limit further food intake after a meal.
- **Slows Gastric Emptying**: PYY slows down the movement of food from the stomach to the intestines, which aids in digestion and prolongs the feeling of fullness.

It’s not the drug it’s the dose

Offer a support or exit strategy

Ezekiel 22:30: "I looked for anyone to repair the wall and stand in the gap for me on behalf of the land, so I wouldn't have to destroy it. But I couldn't find anyone"



Thank you!

Q&A

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