

The background of the slide is a light gray color with a pattern of faint, semi-transparent chemical structures. These structures consist of various interconnected rings and lines, representing molecular frameworks, scattered across the entire page.

Casual Friday Series

# **Functional Blood Chemistry Series**

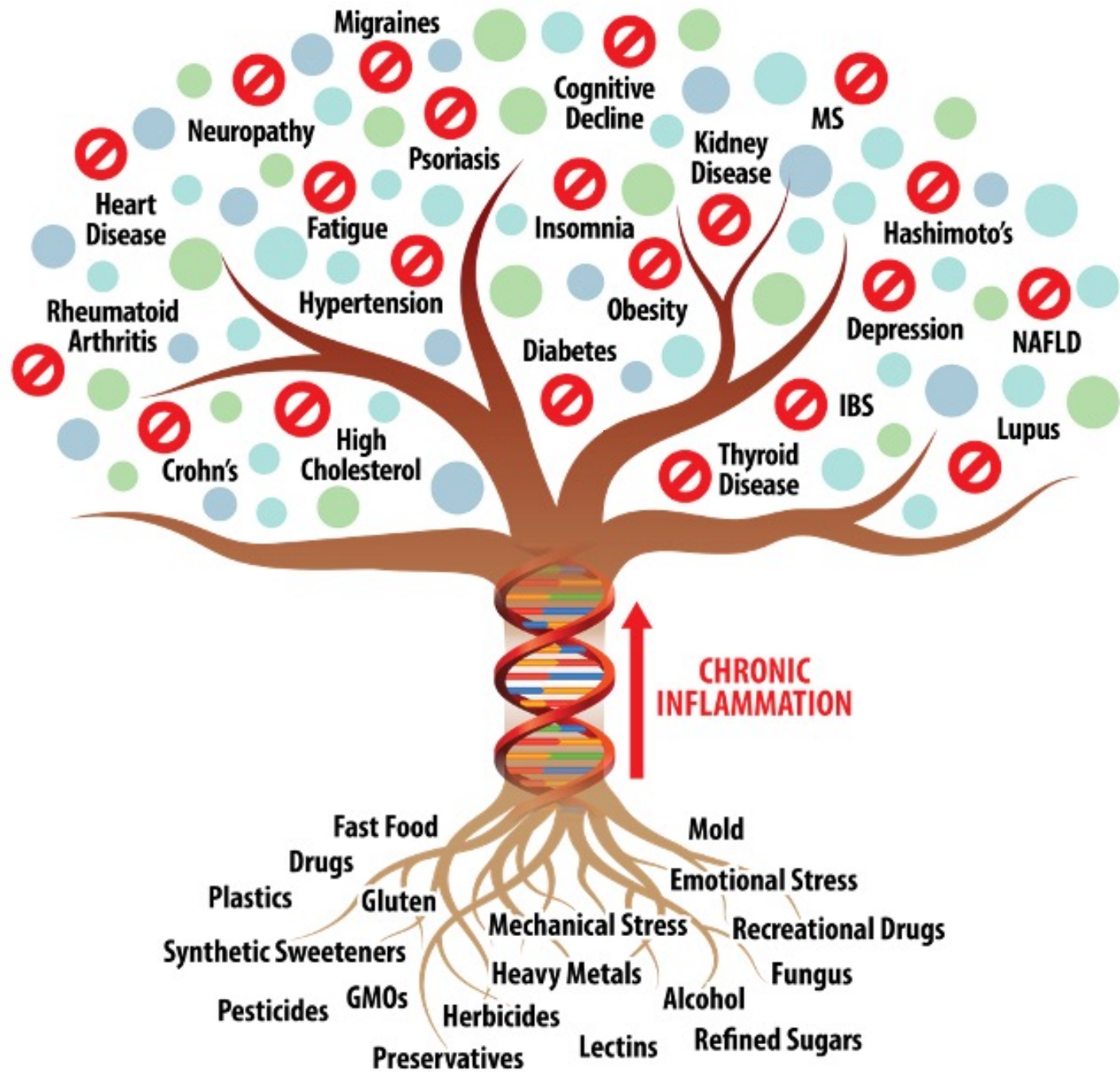
## **Pt. 18: Thyroid (II)**

A Biogenetix Clinical Presentation

[BIOGENETIX.COM](http://BIOGENETIX.COM)

# Disclaimer

- *Information in this presentation is not intended to diagnose, treat, reverse, cure, or prevent any disease. While this presentation is based on medical literature, findings, and text, The following statements have not been evaluated by the FDA.*
- *The information provided in this presentation is for your consideration only as a practicing health care provider. Ultimately you are responsible for exercising professional judgment in the care of your own patients.*





# Contributing Factors

## (GENETIC FACTOR)

HLA-DR  
HLA-DQ & DQ8  
CTLA-4  
PTPN-22  
TSHR  
SCGB3A2  
COPY NUMBER VARIANTS  
TWIN STUDIES  
FCRL3  
IL2RA  
FOXP3  
IGF-1 RECEPTOR  
TG  
CD-40

## (ENDOGENOUS FACTORS)

HIGH LEPTIN LEVELS  
VITAMIN DEFICIENCIES  
FEMALE SEX  
PUBERTY  
PREGNANCY  
RAPID GROWTH  
AGEING

## (ENVIRONMENTAL FACTOR)

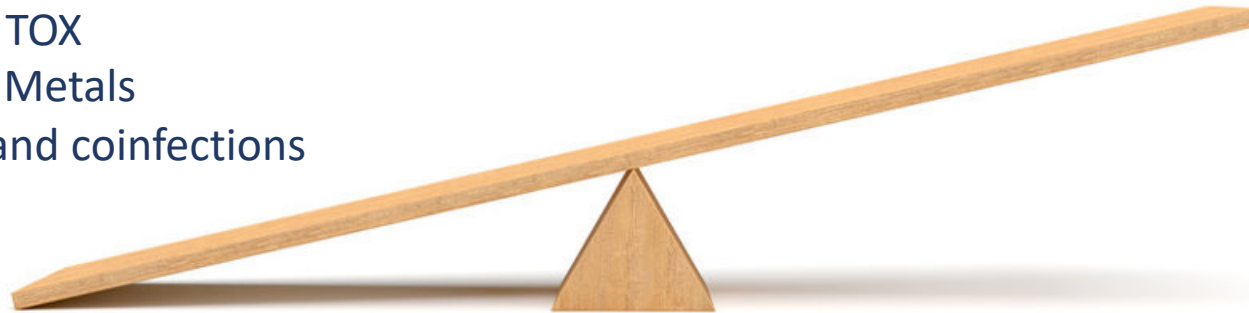
SELENIUM  
STRESS  
IODINE  
POLLUTION/ TOXINS  
INFECTIONS  
GLUTEN  
DRUGS  
ALCOHOL  
VITAMIN-D  
THYROID DAMAGE



# Contributing Factors

Blood  
Adrenal Hormone  
Sex Hormone  
Stool  
Mycotoxins  
Enviro TOX  
Heavy Metals  
Lyme and coinfections

TSH  
Free Frac Hormone



# Low Thyroid Symptoms

- Cold extremities
- Difficulty losing weight
- Depression; slow mental function
- Dry skin, hair and brittle nails
- Hair loss
- Constipation
- Poor/slow wound healing



# Thyroid Markers

TSH	• 1.5-2.0 (1.5-3.0)
Total Thyroxine (TT4)	• 6.0-12.0
Free Thyroxine (FT4)	• 1.0-1.5
Free Thyroxine Index (FTI)	• Less accurate (calculation)
Resin T3 Uptake (T3U)	• 28-38% (Men: 32-38; Women: 28-34)
Total Triiodothyronine (TT3)	• 100-180
Free Triiodothyronine (FT3)	• 3.0-4.0
Reverse T3 (rT3)	• Lab range
Thyroid Antibodies (TGB, TPO)	• If positive, likely Hashimoto's

5 basic patterns to be aware of...





# Low TSH and Low T4

(Pituitary Suppression Pattern)

- Could be poor hypothalamic function
  - Serotonin, dopamine, heavy metals, inflammation
- Could be pituitary suppression
  - Cortisol, inflammation, dysbiosis (lipopolysaccharides), heavy metals



# High TSH, Low T4

(Primary Hypothyroidism)

- Autoimmune (Hashimoto's)
  - Run thyroid antibodies
    - Anti-thyroglobulin antibodies
    - Thyroid peroxidase antibodies
- Iodine deficiency
  - Iodine intake
  - Goitrogen
    - Fluoride, bromide
    - Arsenic
    - Brassica family (cabbage, broccoli, kale, etc)
- Tyrosine deficiency
  - Not likely. Must be a significant amino acid deficiency to cause this.



# Low T3 Uptake, Normal T4/T3, Low free T4/T3

- Likely excess thyroid binding globulin
  - Elevated estrogen is most common reason for excess thyroid binding globulin



# High T3 Uptake, Normal T4/T3, high free T4/T3

- Likely due to elevated testosterone
  - In women, consider blood sugar dysregulation and Polycystic Ovary Syndrome
  - High correlation between Hashimoto's and PCOS



# Normal T4, Low T3

(Thyroid Underconversion Pattern)

- Underconversion of T4 to T3
  - May also be overconverting to rT3
  - Selenium deficiency
  - Inflammation, cortisol, heavy metals, arsenic



# Chemicals and the Thyroid

- Iodine uptake – phthalates, perchlorate
- Thyroid enzymes – pesticides and fungicides
- Deactivating thyroid hormones – lead, chromium, methoxychlor (pesticide)
- PBDEs – inhibit thyroid hormone binding to transthyretin
- PCBs – binds to thyroid binding globulin
- Pentachlorophenol – inhibits thyroid hormone production
- Cadmium, lead, methyl-mercury, Bisphenol A, dioxin, pesticides
- Isoflavones, catechin?



# Impacts on TSH

## STIMULATORY

Thyrotropin-releasing hormone (TRH)

Prostaglandins

Opioids (humans)

Vasopressin (AVP) (long term)

Glucagon-like peptide 1 (GLP-1)

Leptin

Glucocorticoids (in vitro)

## INHIBITORY

Thyroid hormones and analogues

Dopamine

Gastrin

Opioids (rat)

Glucocorticoids (in vivo)

Serotonin

Cholecystokinin (CCK)

Gastrin-releasing peptide (GRP)

Vasopressin (AVP) (short-term)

Neuropeptide Y (NPY)

Interleukin 1  $\beta$  and 6

Tumor necrosis factor  $\alpha$



# Thyroid Interactions

## Interactions with other hormones

- Cortisol
  - Increased – decreases TSH and TBG (high dose), decreases T4/T3 ratio (increases T4/rT3 ratio)
  - Decreased – increased TSH
  - Increases in thyroid hormone, increase cortisol clearance in liver, and thus leads to more ACTH production
- Estrogen
  - Increased TBG, TSH (postmenopausal women), increased T4 requirement in hypothyroid patients
- Testosterone
  - Decreased TBG, decreased T4 turnover in women, reduced T4 requirement in hypothyroid patients
- Growth hormone
  - Decreased D3 activity





# Goiter



# Goiter



# Goiter



Source: Usatine RP, Smith MA, Mayeaux EJ, Chumley HS: *The Color Atlas of Family Medicine, Second Edition*: [www.accessmedicine.com](http://www.accessmedicine.com)  
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# Goiter

- A goiter can occur in a gland that is producing too much hormone (hyperthyroidism), too little hormone (hypothyroidism), or the correct amount of hormone (euthyroidism). A goiter indicates there is a condition present which is causing the thyroid to grow abnormally.



# Goiter

- Hashimoto's thyroiditis is a more common cause of goiter formation in the US. This is an autoimmune condition in which there is destruction of the thyroid gland by one's own immune system. As the gland becomes more damaged, it is less able to make adequate supplies of thyroid hormone. The pituitary gland senses a low thyroid hormone level and secretes more TSH to stimulate the thyroid. This stimulation causes the thyroid to grow, which may produce a goiter.



# Goiter

- Another common cause of goiter is Graves' disease. In this case, one's immune system produces a protein, called thyroid stimulating immunoglobulin (TSI). As with TSH, TSI stimulates the thyroid gland to enlarge producing a goiter. However, TSI also stimulates the thyroid to make too much thyroid hormone (causes hyperthyroidism). Since the pituitary senses too much thyroid hormone, it stops secreting TSH. In spite of this the thyroid gland continues to grow and make thyroid hormone. Therefore, Graves' disease produces a goiter and hyperthyroidism.

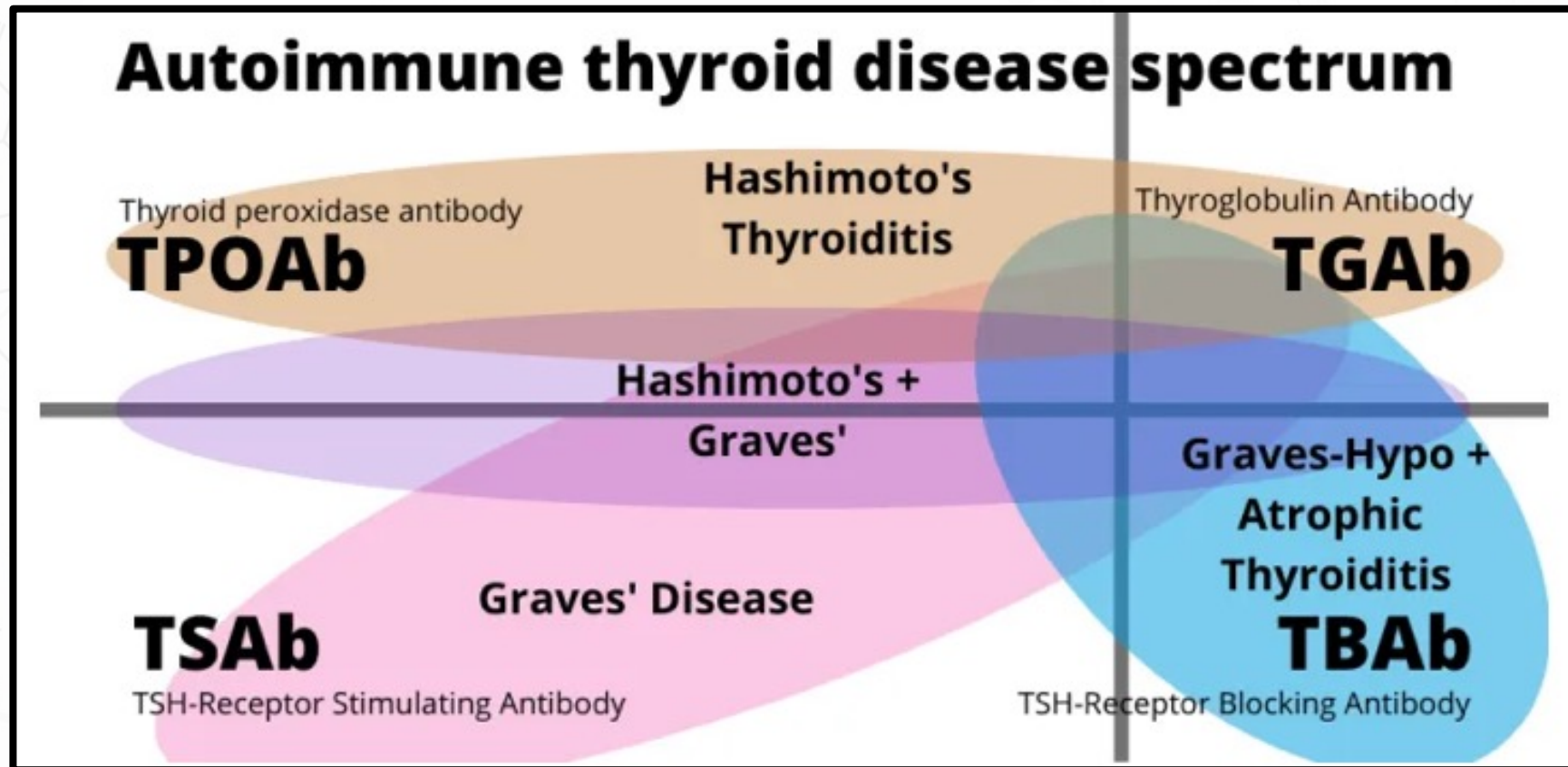


# Goiter

- Multinodular goiters are another common cause of goiters. Individuals with this disorder have one or more nodules within the gland which cause thyroid enlargement. This is often detected as a nodular feeling gland on physical exam. Patients can present with a single large nodule or with multiple smaller nodules in the gland when first detected.



# Autoimmunity





## The role of the immune system and cytokines involved in the pathogenesis of autoimmune thyroid disease (AITD)

Hanna Mikoś, Marcin Mikoś, Monika Obara-Moszyńska, Marek Niedziela <sup>1</sup>

Autoimmune thyroid disease (AITD) is the most common organ-specific autoimmune disorder. AITD development occurs due to loss of immune tolerance and reactivity to thyroid autoantigens: thyroid peroxidase (TPO), thyroglobulin (TG) and thyroid stimulating hormone receptor (TSHR). This leads to infiltration of the gland by T cells and B cells that produce antibodies specific for clinical manifestations of hyperthyroidism in Graves' disease (GD) and chronic autoimmune thyroiditis (cAIT). In addition, T cells in Hashimoto's thyroiditis induce apoptosis in thyroid follicular cells, leading ultimately to the destruction of the gland. Cytokines are involved in the pathogenesis of thyroid diseases working in both the immune system and directly targeting the thyroid follicular cells. They are involved in the induction and effector phase of the immune response and



# Autoimmunity

In GD, thyroid stimulating immunoglobulins (TSI) bind to the TSH receptor (TSHR) and mimic TSH stimulation of the thyroid gland. Because TSI induced thyroid hormone secretion is not controlled by negative feedback, such stimulation causes uncontrolled hyperthyroidism.<sup>8</sup>

TSI are IgG antibodies that can cross the placental barrier and cause neonatal thyrotoxicosis in newborns delivered by mothers with GD.<sup>9,10</sup>

The TSH receptor contains a large extracellular domain that presents epitopes for a variety of autoantibodies, including TSI and Thyroid Blocking Immunoglobulins TBI.<sup>11-13</sup> In contrast to TSI, TBI bind to the TSH receptor and inhibit TSH stimulation of thyroid cells, leading to hypothyroidism. Commonly used Thyrotropin Receptor Autoantibody (TRAb) assays do not distinguish between TSI and TBI.



# Autoimmunity

“Hashimoto's thyroiditis, or inflammation of the thyroid gland, is an autoimmune disorder. That means it is caused by a malfunction in your immune system. Instead of protecting your thyroid tissue, your immune cells attack it. These immune cells can cause hypothyroidism(underactive thyroid), a goiter (enlarged thyroid), or both. Eventually, the thyroiditis process can even destroy your entire thyroid, if left undetected or untreated.”

“Doctors aren't entirely sure why the immune system, which is supposed to defend the body from harmful viruses and bacteria, sometimes turns against the body's healthy tissues.”



# Autoimmunity

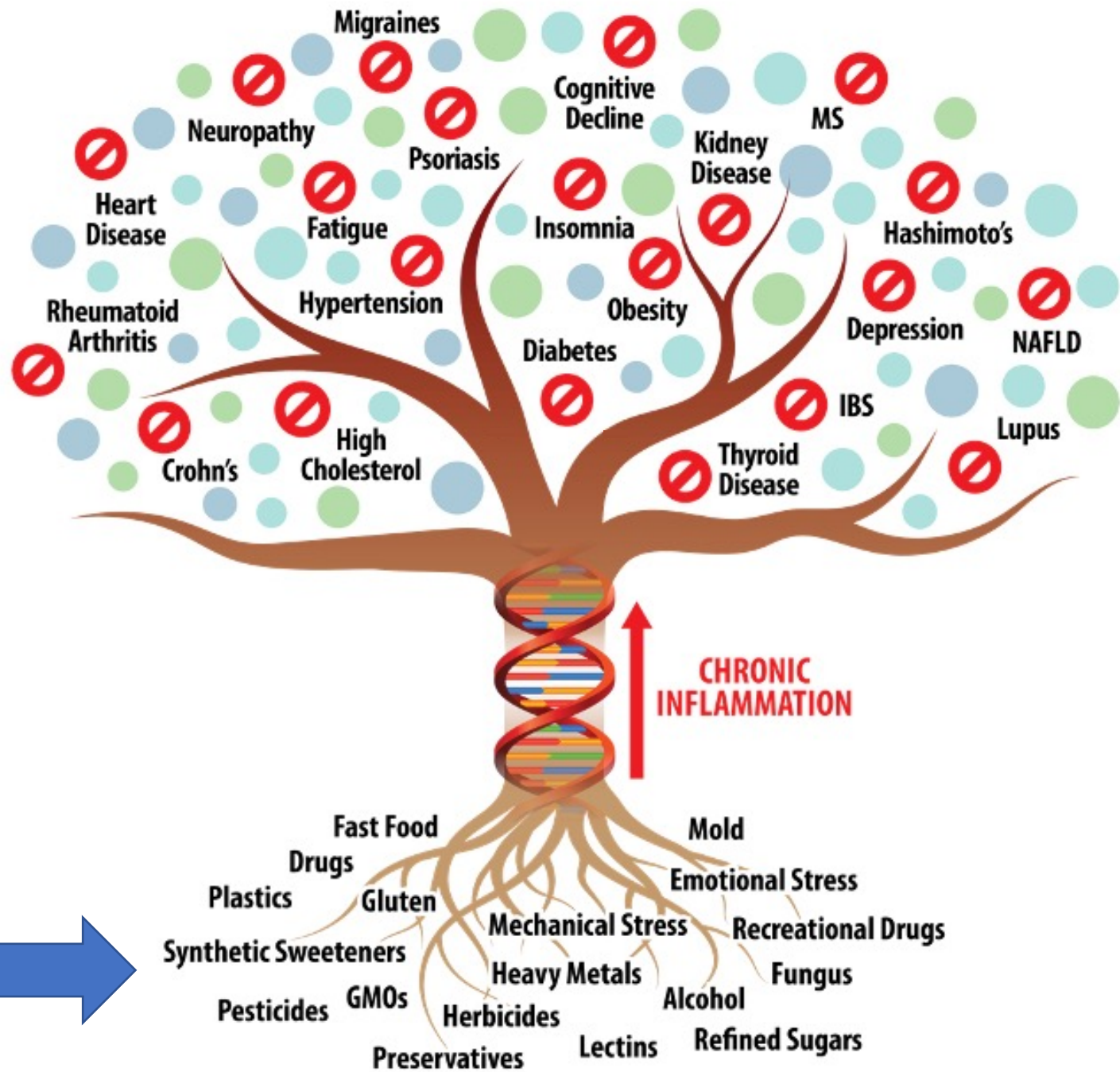
In Hashimoto's thyroiditis, large amounts of damaged immune cells invade the thyroid. These immune cells are called lymphocytes; this is where Hashimoto's other name—chronic lymphocytic thyroiditis—is derived from.

NEXT:

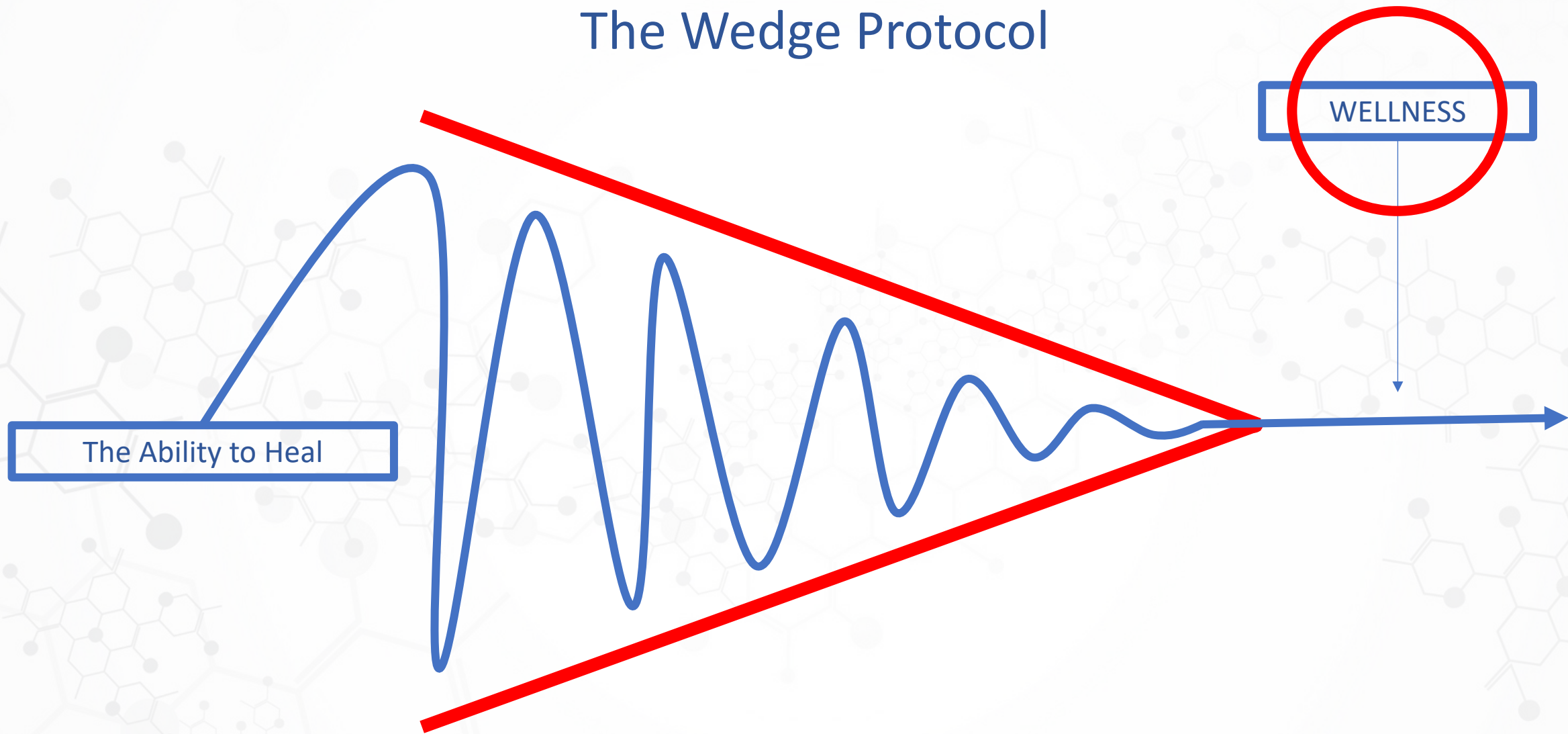


Mechanisms, Drivers, Assessments





# The Wedge Protocol



The Ability to Heal

WELLNESS

