Casual Friday Series

# Endocrine Expertise: Hashimoto’s Mechanics

A Biogenetix Clinical Presentation

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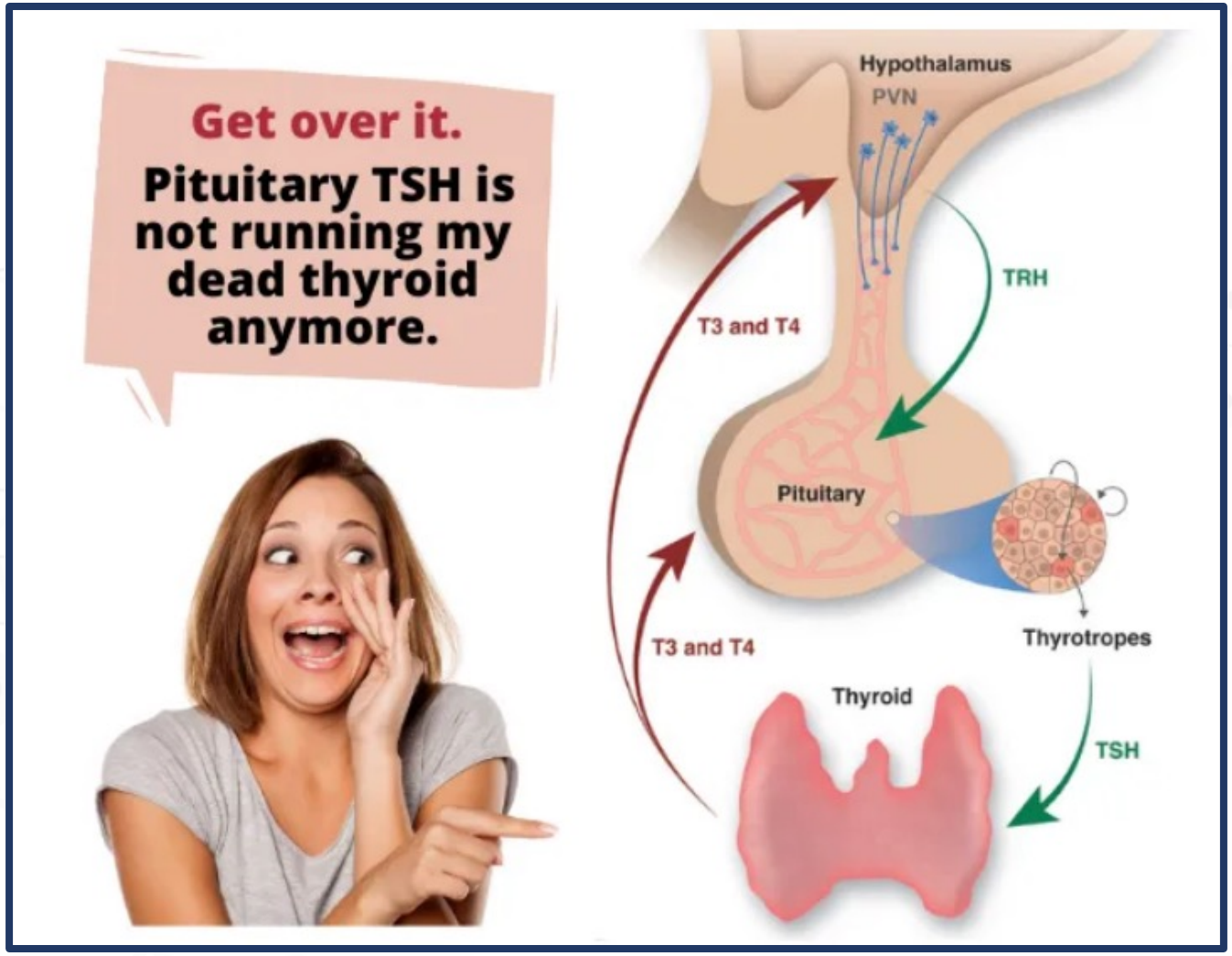
- *Information in this presentation is not intended, in itself, to diagnose, treat, reverse, cure, or prevent any disease. While this presentation is based on medical literature, findings, and text, The following statements have not been evaluated by the FDA.*
- *The information provided in this presentation 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.*

Lifestyle + Genetics = Chronic Health IMPROVEMENT




https://thyroidpatients.ca/2020/07/14/visual-bias-inflated-pituitary-hpt/

Curr Genomics. 2011 Dec; 12(8): 576–588.
Published online 2011 Dec. doi: 10.2174/138920211798120763
PMCID: PMC3271310
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# Hashimoto's Thyroiditis: From Genes to the Disease

Katja Zaletel and Simona Gaberšček*

The clinical disease may present with a variety of different manifestations ranging from a simple TAbs presence in patients with normal thyroid function to the development of severe thyroid dysfunction. Some patients present with short periods of mild thyrotoxicosis which usually cease spontaneously. Most often, euthyroid phase is followed by a gradual development of subclinical hypothyroidism which progresses slowly to overt hypothyroidism with the estimated annual risk of 4% in females [6]. According to large epidemiological surveys, HT is the most frequent cause of hypothyroidism recorded in 4% to 9.5% of the adult population [7-10]. The prevalence of HT is high which was also confirmed by the largest National Health and Nutrition Examination Survey (NHANES) III study. The results show that 18% of the population without previously known thyroid disease regardless age or gender presented with elevated TAbs; TPOAbs were positive in 11.3% and TgAbs in 10.4%. The prevalence of TAbs in females was twice as high as in males. It increased with age and was significantly higher in whites or Japanese than in blacks or Mexican Americans [8, 11]. Thus, approximately 20% of females older than 60 years were TAbs positive [8].

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TAbs serve as a useful marker for the diagnosis of thyroid autoimmunity. In HT, TPOAbs are present in nearly all (>90 %) patients, while TgAbs can be detected in approximately 80% [1, 3].

The prevalence of HT confirmed by cytology was 13.4% in consecutive patients who underwent fine-needle aspiration biopsy of thyroid nodules and was similar to that of type 2 diabetes [4]. Beside lymphoid follicles, changes in epithelial cells, formation of connective tissue, and diffuse round cell infiltration, in his report Hashimoto described also some cracking spaces close to lymphoid follicles. It is now known that these cracking spaces are mainly lymphatic vessels, localized within the interlobular septa. Their number increases within the thyroid parenchyma near the lymphoid follicles [5].

AMERICAN THYROID ASSOCIATION®

www.thyroid.org

# Hashimoto's Thyroiditis

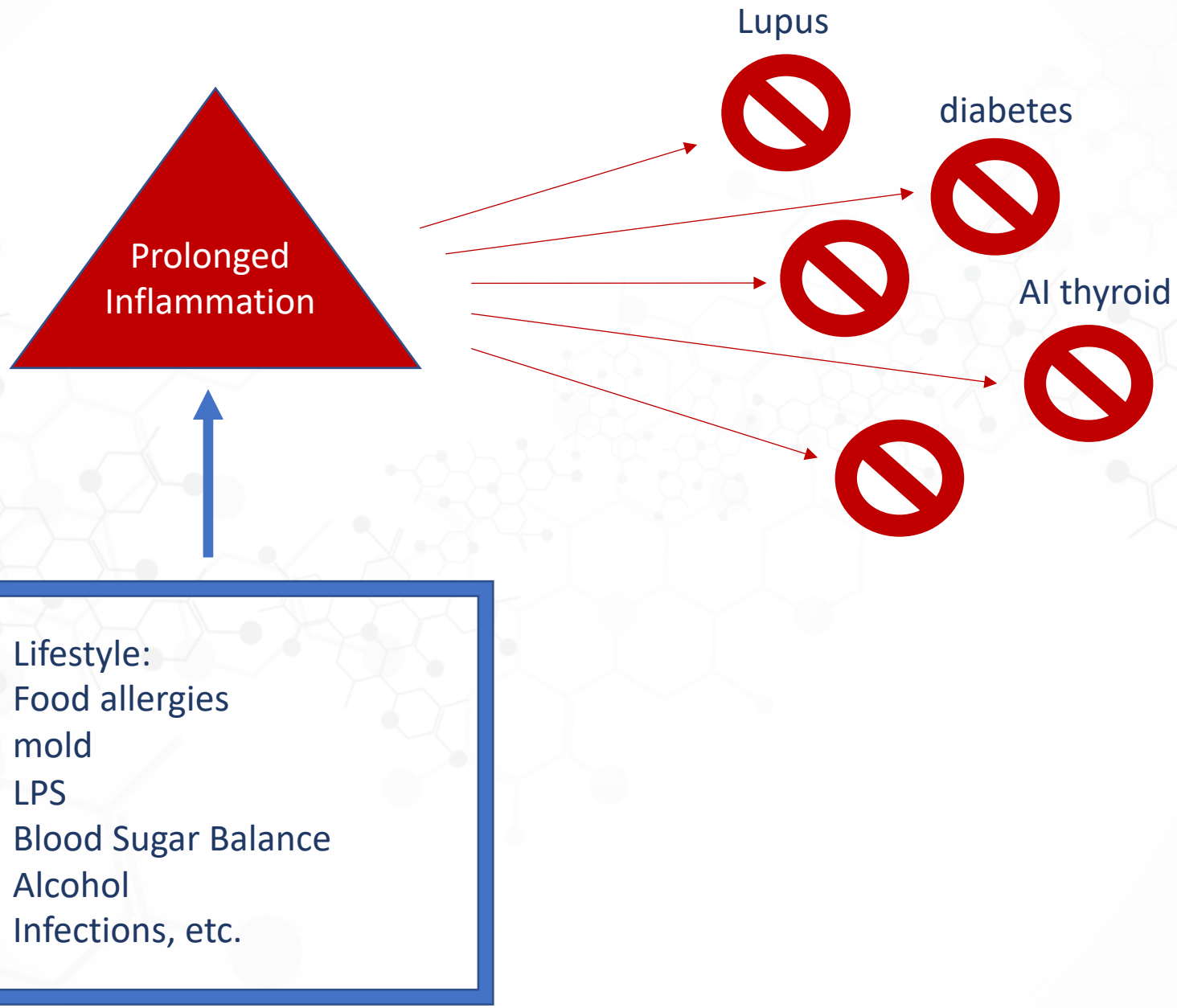
***(Lymphocytic Thyroiditis)***

## HOW IS HASHIMOTO THYROIDITIS TREATED?

Patients with elevated TPO antibodies but normal thyroid function tests (TSH and Free T4) do not require treatment. Patient with only a slightly elevated TSH (mild hypothyroidism) may not require medication and should have repeat testing after 3-6 months if this has not already been done. For patients with overt hypothyroidism (elevated TSH and low thyroid hormone levels) treatment consists of thyroid hormone replacement (see *Thyroid Hormone Treatment brochure*). Synthetic levothyroxine taken orally at an appropriate dose, is inexpensive, very effective in restoring normal thyroid hormone levels, and results in an improvement of symptoms of hypothyroidism. Most patients with Hashimoto's thyroiditis will require lifelong treatment with levothyroxine. Finding the appropriate dose, particularly at the beginning, may require testing with TSH every 6-8 weeks after any dose adjustment, until the correct dose is determined. After that, monitoring of TSH once a year is generally sufficient.

When levothyroxine is taken in the appropriate dose, it has no side effects. However, when an insufficient dose is taken, serum TSH remains elevated and patients may have persistent symptoms of hypothyroidism (see *Hypothyroidism brochure*). If the dose is excessive, serum TSH will become suppressed and patients may develop symptoms of hyperthyroidism or have other side effects (see *Hyperthyroidism brochure*).

https://www.thyroid.org/hashimotos-thyroiditis/


Lupus
diabetes
AI thyroid
Prolonged Inflammation
Lifestyle:
Food allergies
mold
LPS
Blood Sugar Balance
Alcohol
Infections, etc.

Pituitary

Immune System

TSH

Lifestyle:
Mercury
Mold
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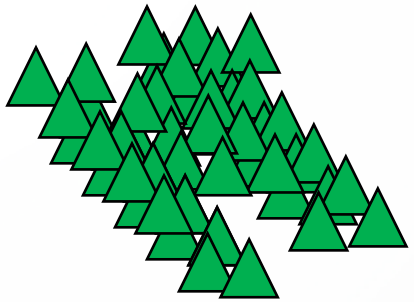
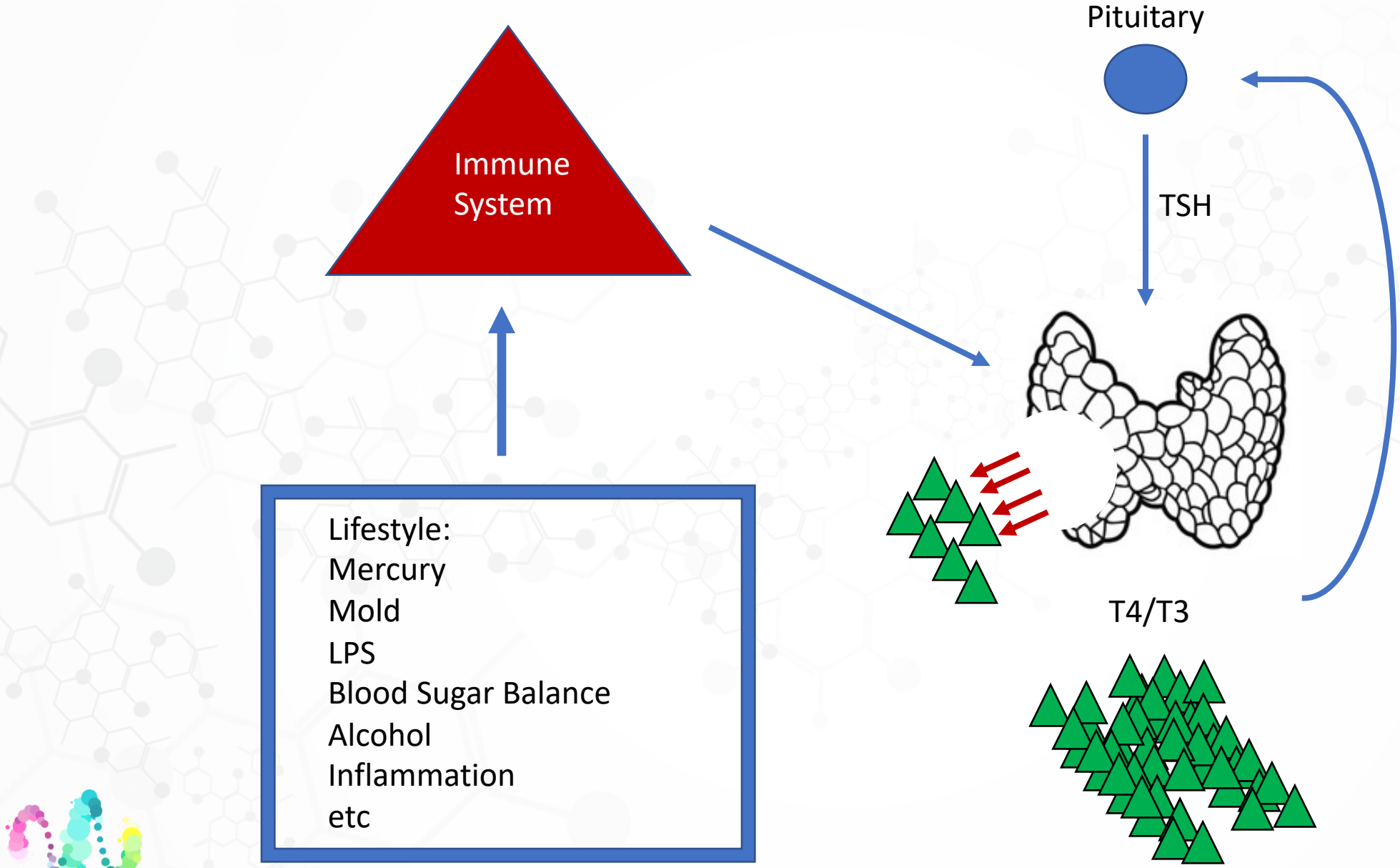
Alcohol
Inflammation
etc

T4/T3

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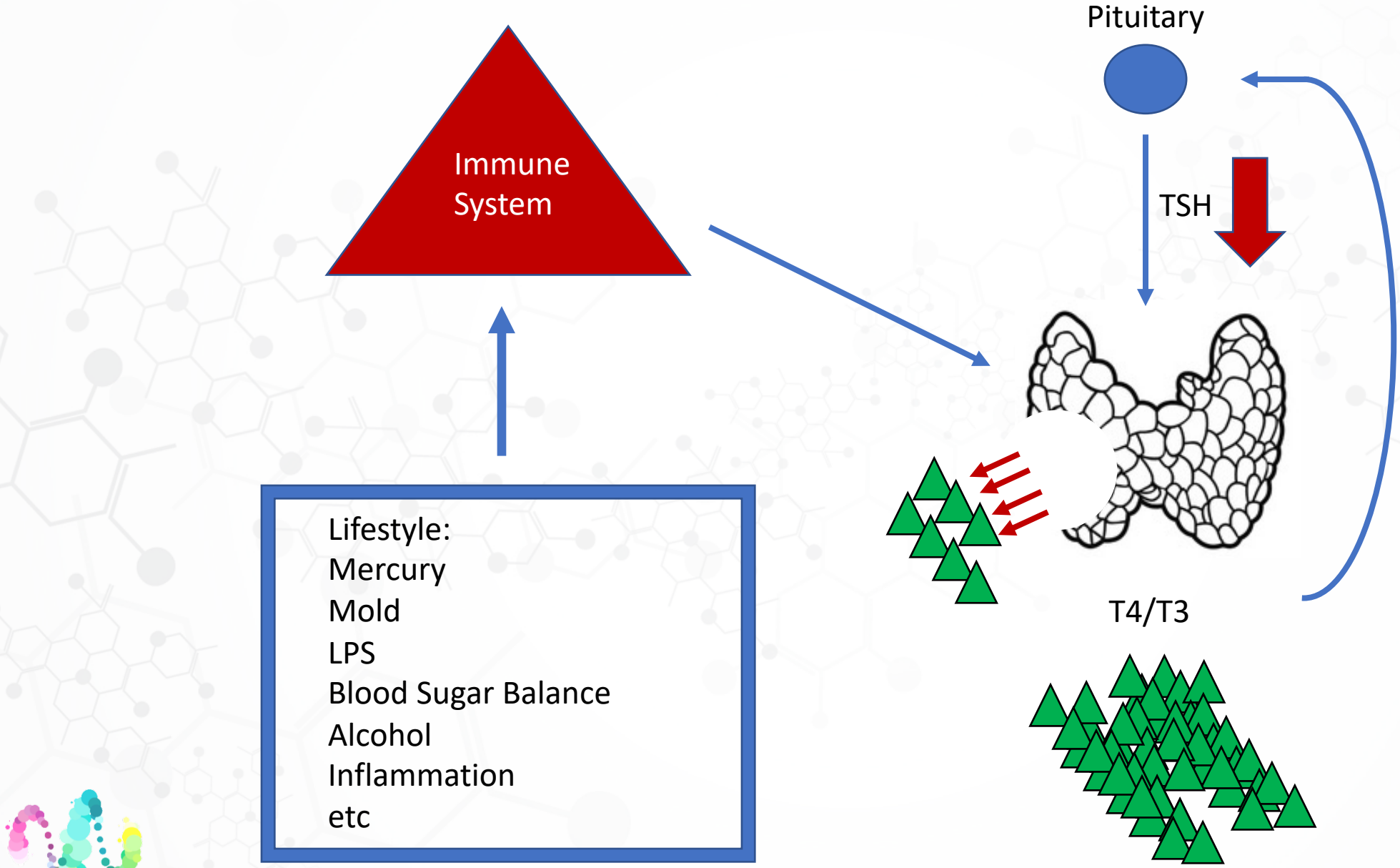
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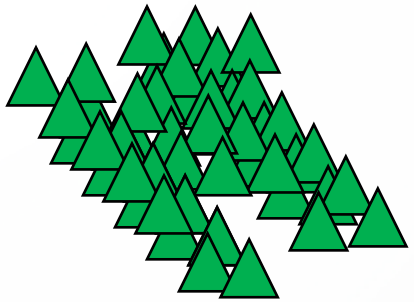

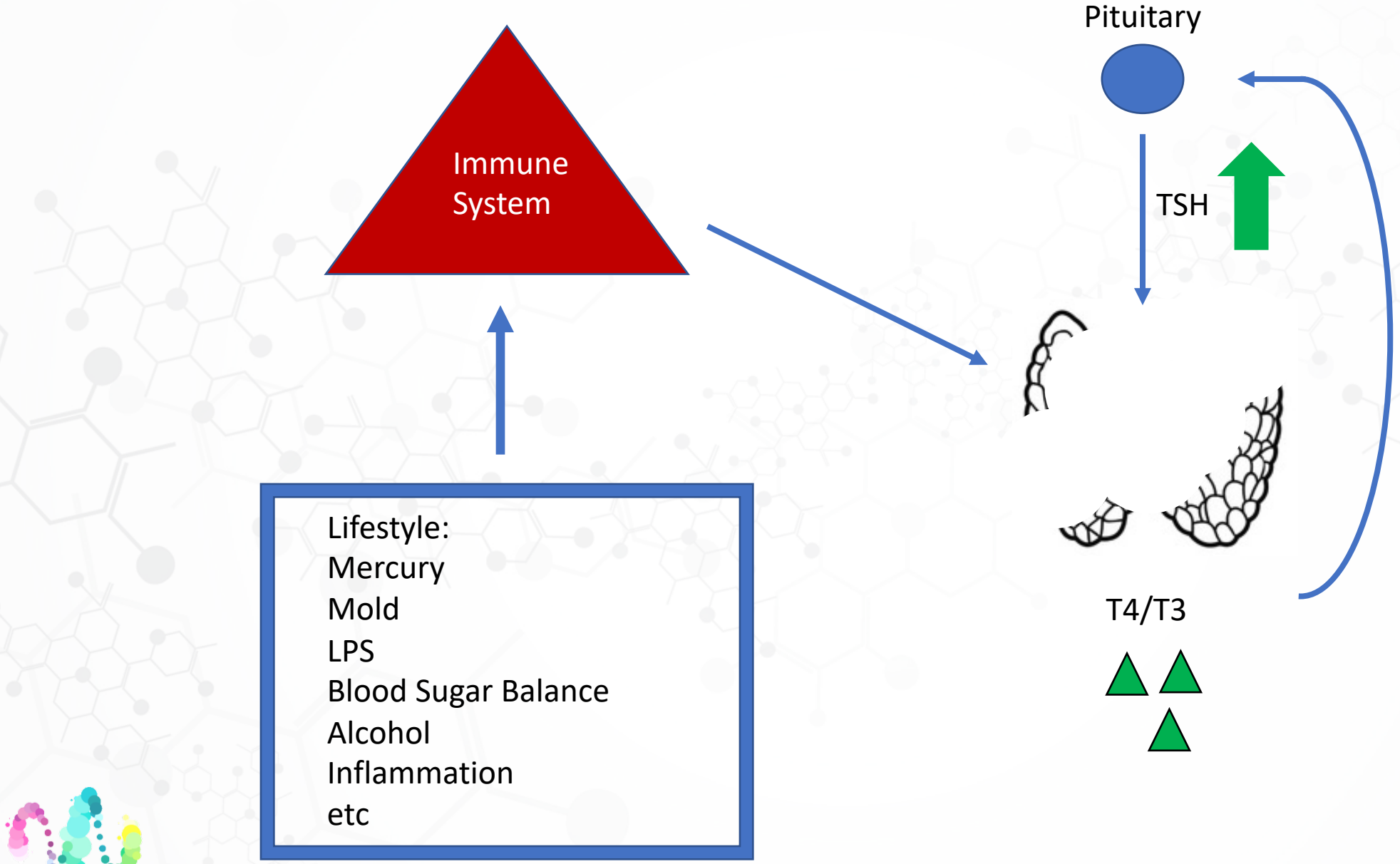
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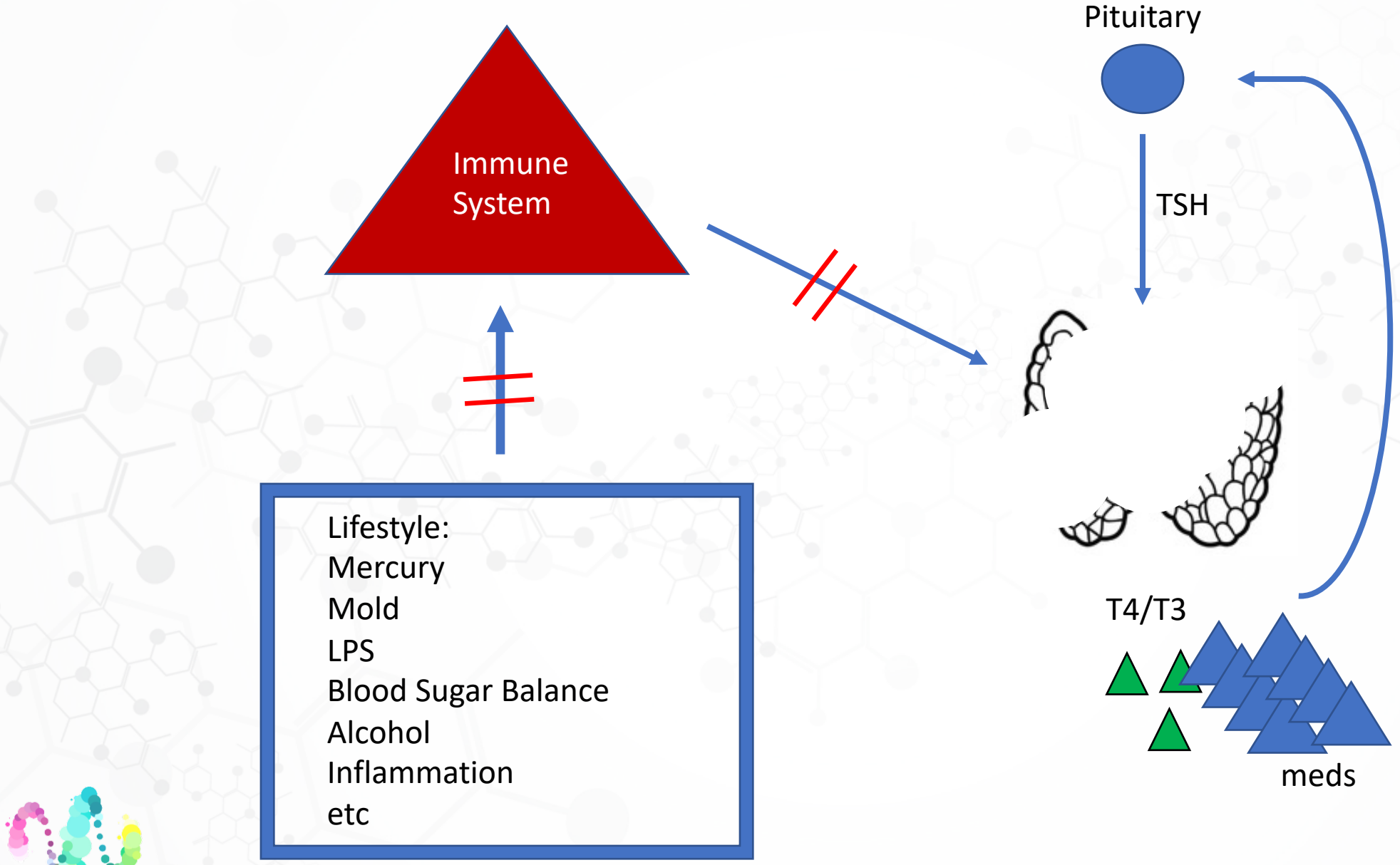
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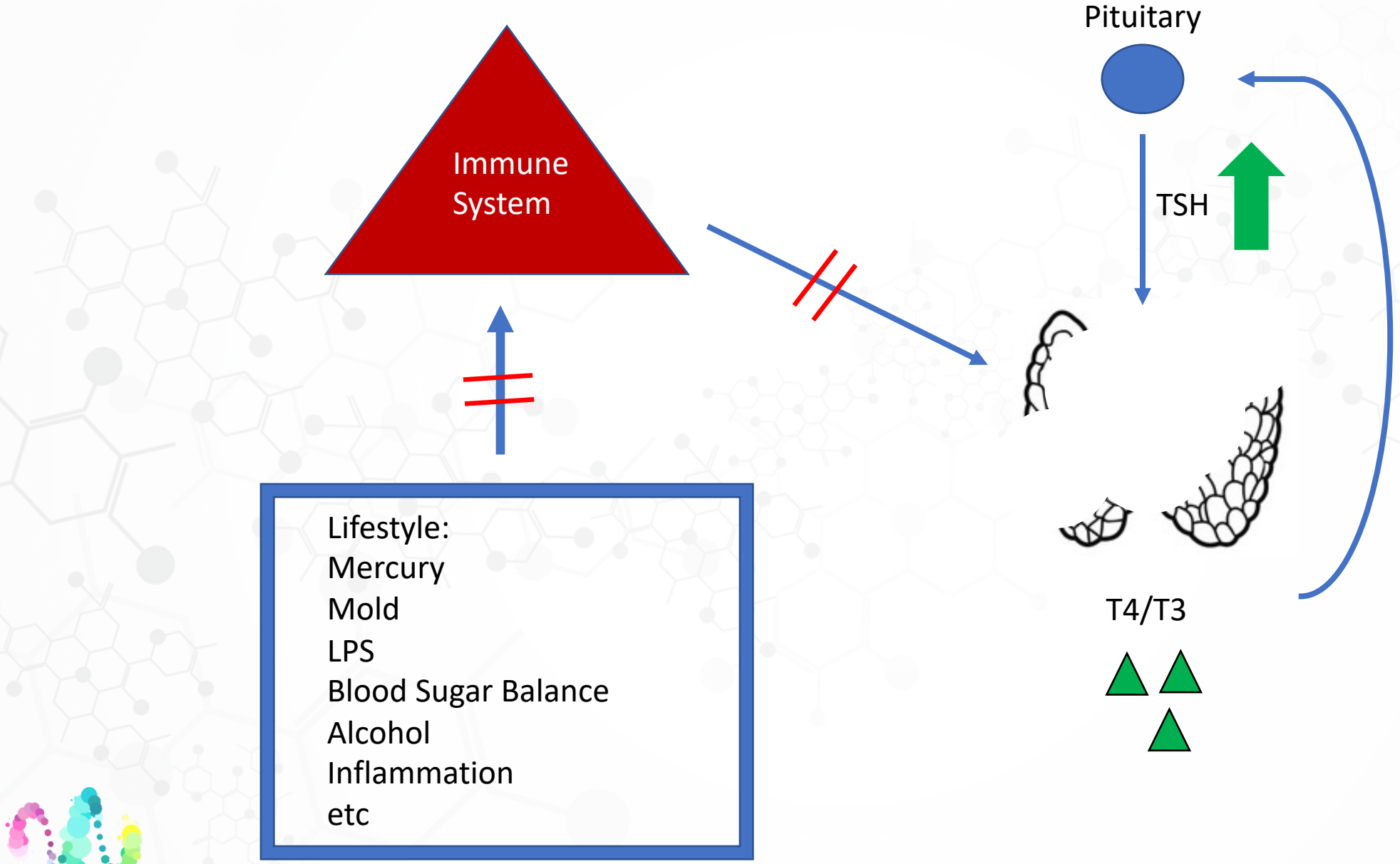
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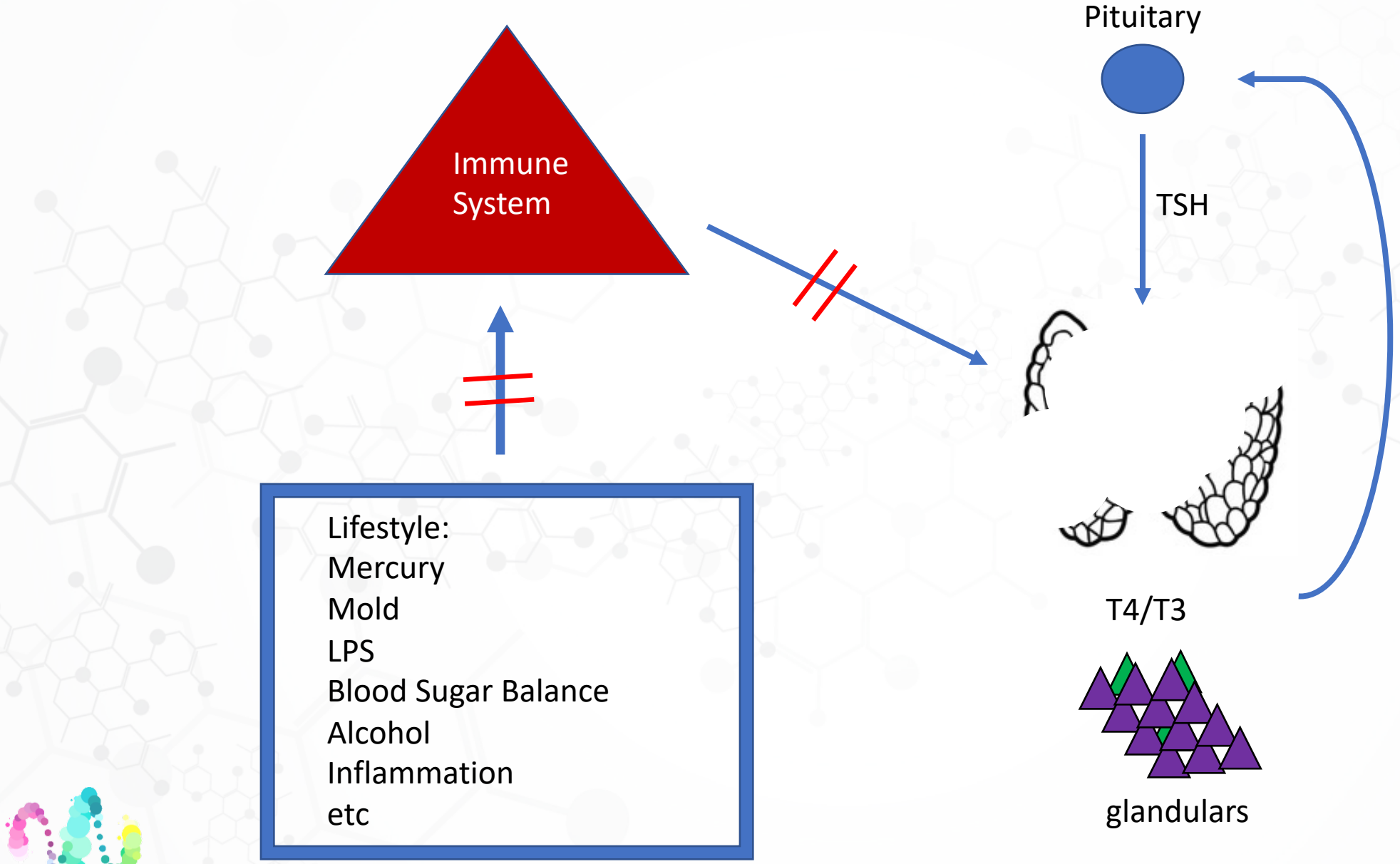
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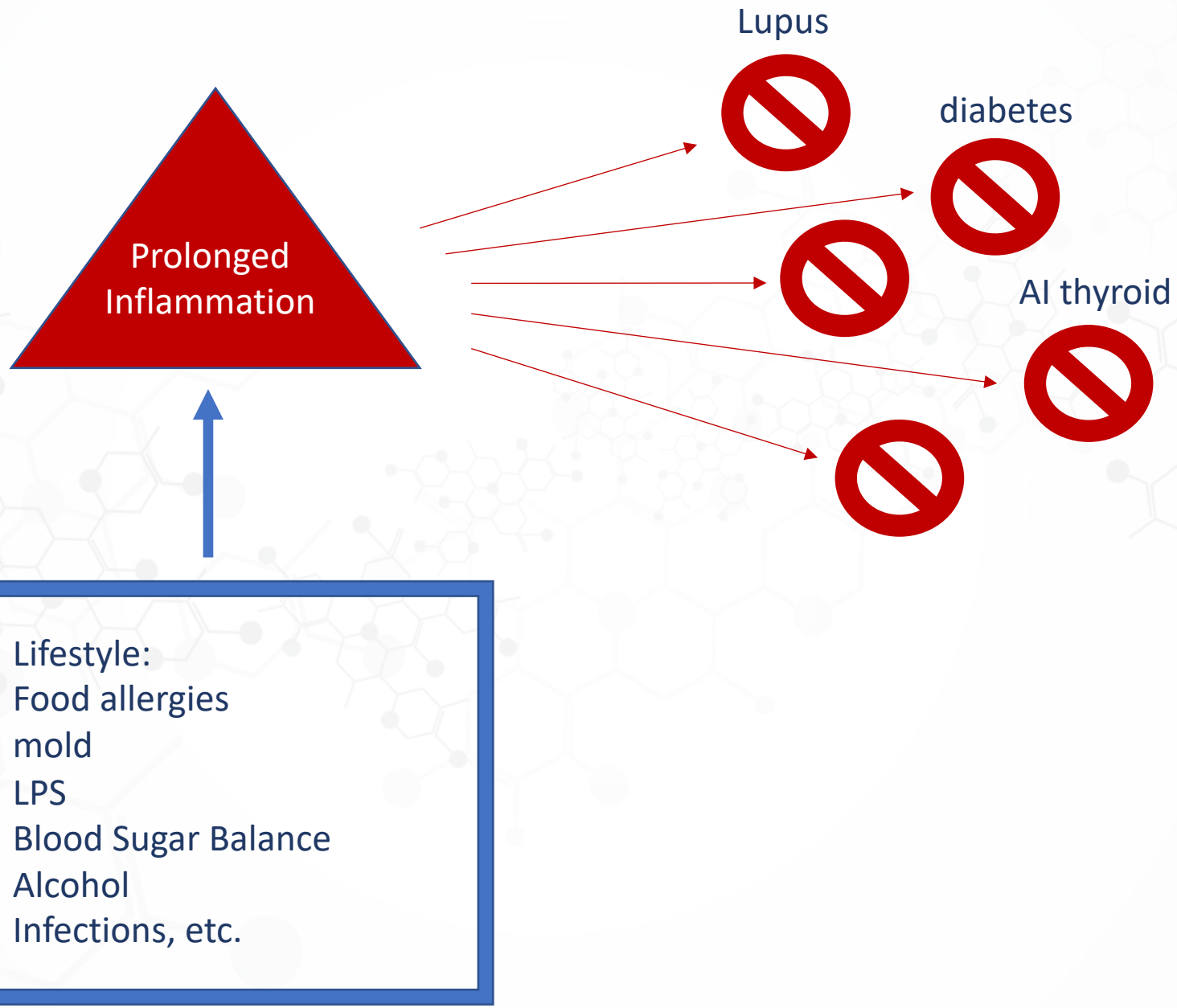
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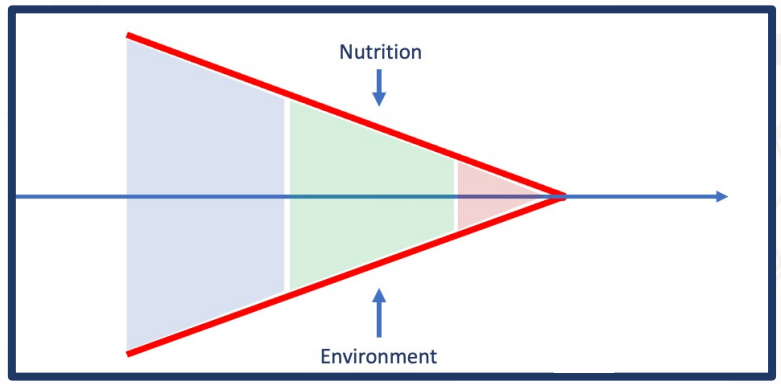


## Hashimoto's mechanics summary:

1. Most common cause of hypothyroidism in the United States.
2. Always ends up in hypothyroidism, if given enough time.
3. Nodules are common, and rarely mean cancer.
4. Modern medicine has its head in the sand, because they're working on a fruit-based mechanism.
5. Patients that fail to control inflammation end up acquiring further autoimmune diagnoses.
6. Thyroid hormone levels often need to be addressed, retest thyroid panel. Often.
7. Medication?

47-year-old female. Prediabetes, hypothyroidism, fatigue, weight gain, depression, mild constipation.

| | | | | |
|---|---|---|---|---|
| TSH | 3.120 | | uIU/mL | 0.450-4.500 |
| Thyroxine (T4) | 9.9 | | ug/dL | 4.5-12.0 |
| T3 Uptake | 29 | | % | 24-39 |
| Free Thyroxine Index | 2.9 | | | 1.2-4.9 |
| Triiodothyronine (T3) | 105 | | ng/dL | 71-180 |
| Triiodothyronine (T3), Free | 2.6 | | pg/mL | 2.0-4.4 |
| Reverse T3, Serum [A] | 22.8 | | ng/dL | 9.2-24.1 |

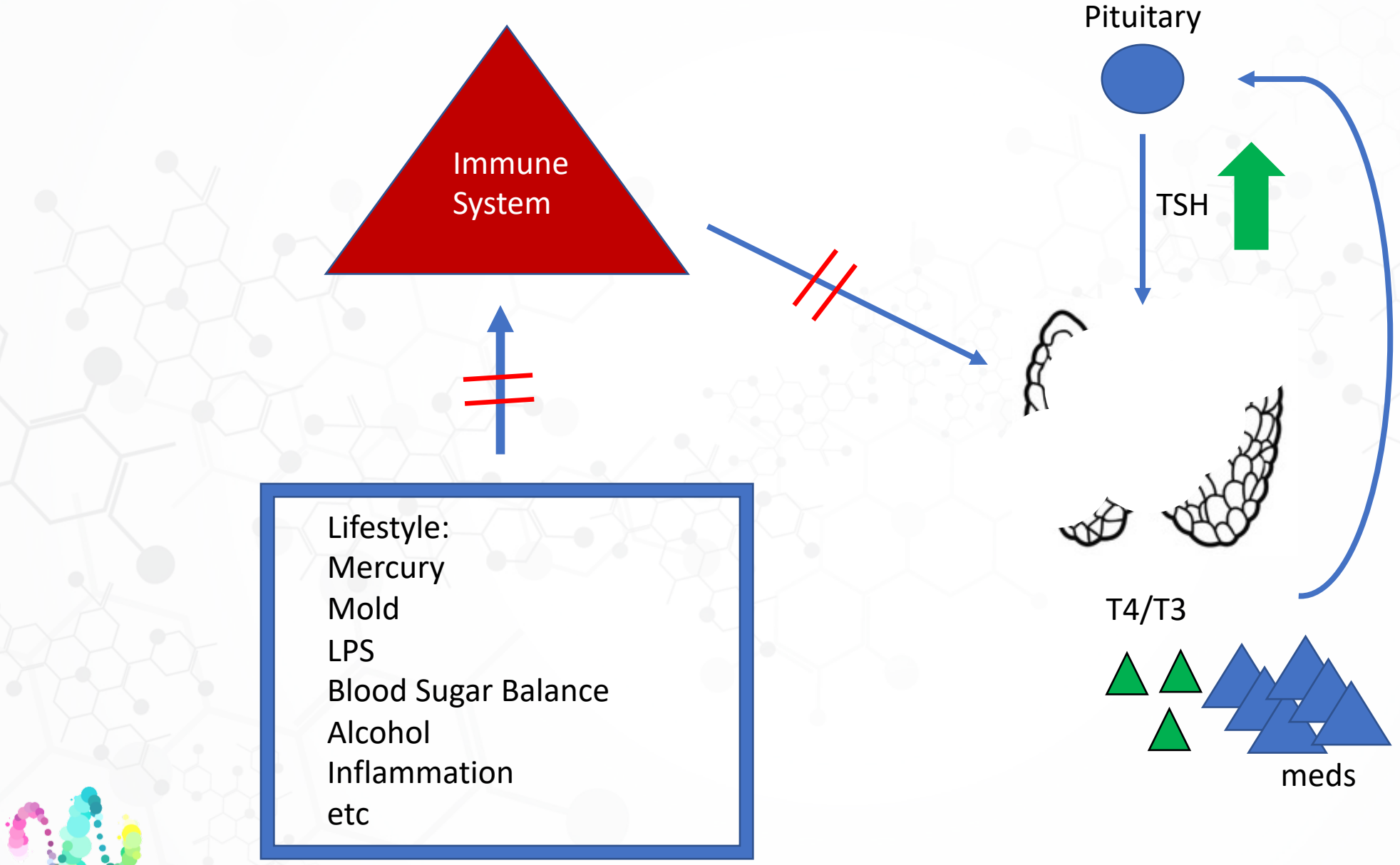
| | | | | |
|---|---|---|---|---|
| T4,Free(Direct) | 1.52 | | ng/dL | 0.82-1.77 |
| **Thyroid Peroxidase (TPO) Ab** | **278** | **High** | IU/mL | 0-34 |
| **Thyroglobulin Antibody** | **7.2** | **High** | IU/mL | 0.0-0.9 |

**Thyroglobulin Antibody measured by Beckman Coulter Methodology**

47-year-old female. Prediabetes, hypothyroidism, fatigue, weight gain, depression, mild constipation.

| | | | | |
|---|---|---|---|---|
| Homocyst(e)ine | 8.3 | | umol/L | 0.0-14.5 |
| **TSH** | **5.950** | **High** | uIU/mL | 0.450-4.500 |
| Thyroxine (T4) | 6.1 | | ug/dL | 4.5-12.0 |
| **T3 Uptake** | **23** | **Low** | % | 24-39 |
| Free Thyroxine Index | 1.4 | | | 1.2-4.9 |
| Triiodothyronine (T3) | 95 | | ng/dL | 71-180 |
| Triiodothyronine (T3), Free | 2.4 | | pg/mL | 2.0-4.4 |
| Reverse T3, Serum [A] | 12.4 | | ng/dL | 9.2-24.1 |
| T4,Free(Direct) | 0.97 | | ng/dL | 0.82-1.77 |
| **Thyroid Peroxidase (TPO) Ab** | **181** | **High** | IU/mL | 0-34 |
| **Thyroglobulin Antibody** | **20.5** | **High** | IU/mL | 0.0-0.9 |

**Thyroglobulin Antibody measured by Beckman Coulter Methodology**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# Biogenetix: 833-525-0001

zeb@biogenetix.com

kim@biogenetix.com