SYNERGETIC

The Thyroid Detective 🔍

Finding the real culprit: a root-cause framework, from cellular mechanisms to clinical protocols, for common thyroid presentations in practice

Synergetic Botanicals | synergeticbotanicals.co.uk | synergetic.med

Meet Your Presenters



Sonia O'Donnell

Sonia is a homeopath and naturopath with over twenty years of experience. Her work blends naturopathic diagnostics with herbal medicine, a combined approach that has supported thousands of people with ME, CFS, chronic Lyme and complex childhood needs. Through her experience, she has built a clinic model enabling her to see hundreds of patients without burning out, and developed a successful product range of herbal formulas called Synergetic Botanicals and teaches widely on using naturopathic medicines for everyday health.



Izzy Seadon

Izzy is a nutritional therapist, herbalist, naturopath, and kinesiologist specialising in women's health and complex hormonal presentations. Her work focuses on identifying and addressing deeper drivers of hormonal imbalance, integrating nutrition, herbs and other naturopathic principles and therapeutics. Izzy is known for her layered, systems-based approach that prioritises treating the individual, helping practitioners and clients through a root cause framework grounded in science and aligned with traditional healing principles.



What We Will Cover Today

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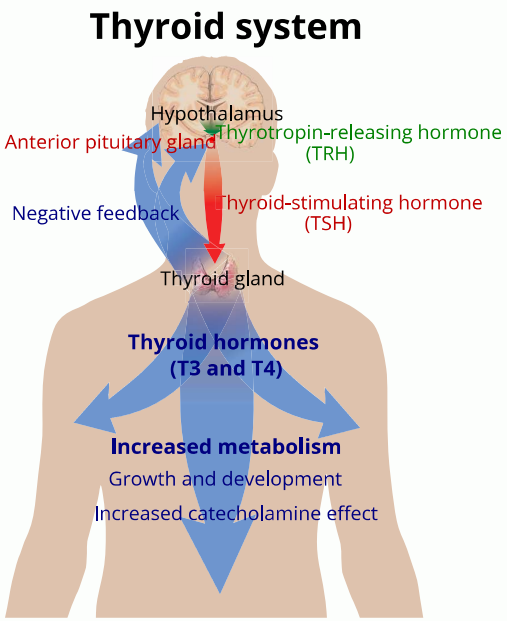
Thyroid Physiology 101



What Is It?

A butterfly-shaped gland located at the front of the neck. It acts as the body's **master thermostat**, regulating energy, metabolism, growth, and reproductive function based on constant feedback from the brain.

Regulated via the hypothalamic-pituitary- thyroid axis.

What Does It Control?

- **Metabolism** — sets the body's resting metabolic rate and heat production
- **Heart** — influences heart rate and how strongly it contracts
- **Breathing** — drives respiratory rate
- **Brain & nervous system** — affects mood, cognition, and memory
- **Reproductive health** — regulates the menstrual cycle and fertility
- **Metabolism of nutrients** — governs how the body builds and breaks down protein, carbs, and fat
- **Stress response** — sensitises the body to adrenaline and noradrenaline

What Can Go Wrong?

Hypothyroidism

Underactive thyroid - hormone output falls short of what the body needs.

Hyperthyroidism

Overactive thyroid - excess hormone production drives the metabolism too fast.

Where the Problem Originates

- **Primary** - the thyroid gland itself is the source, whether under- or overactive
- **Secondary** - the pituitary fails to signal the thyroid correctly via TSH
- **Tertiary** - the hypothalamus disrupts the signal further upstream, via TRH.

Where the Picture Gets Complicated

- Thyrotoxicosis - hormone excess without true gland overactivity
- Non-thyroidal illness syndrome - lab changes driven by systemic illness, not primary thyroid disease [1]
- Thyroid hormone resistance - normal or high hormone levels, but reduced tissue response. [2]

Hashimoto's

Autoimmune hypothyroidism - the immune system gradually attacks and reduces thyroid tissue.

Grave's Disease

Autoimmune hyperthyroidism - antibodies overstimulate the thyroid. Can transition to Hashimoto's. [3,4,5]

[1] Ganesan et al. StatPearls. 2026. [2] Rivas & Lado-Abeal. Proc Bayl Univ Med Cent. 2016. [3] Tamai et al. J Clin Endocrinol Metab. 1980. [4] Kraiem et al. Clin Endocrinol. 1992. [5] Bishay & Chen. Case Rep Endocrinol. 2016.





The Hypothyroid Epidemic

3rd

Most Prescribed

Levothyroxine is the 3rd most commonly prescribed medication in the UK. [6,7]

1 in 5-10

Post-Menopausal Women

Have elevated TSH levels, indicating underactive thyroid.[8,9]

80%

More Likely for Women to be Affected

Of those with Hashimoto's — the most common cause of low thyroid function. [10]

[6] HRA. OPAL Study. **hra.nhs.uk**. [7] Society for Endocrinology. Endocrine Abstracts. 2025. [8] Jain et al. Bioinformation. 2025. [9] Kim et al. Thyroid. 2022. [10]Hu et al. Front Public Health. 2022.

The Clues in Clinic

Hypothyroidism

- Fatigue and sluggishness
- Cold intolerance, low body temperature
- Muscle cramps, weakness, joint pain
- Constipation, slow digestion
- Depression, irritability, memory loss
- Abnormal menstruation, decreased libido
- Weight gain
- Pale/dry skin
- Coarse, thinning hair, loss of the outer third of eyebrows
- Puffy face, hoarse voice, enlarged tongue
- High cholesterol
- Periorbital oedema

Hyperthyroidism

- Heart palpitations
- Heat intolerance
- Nervousness, insomnia
- Breathlessness
- Increased bowel movements
- Light/absent periods
- Fatigue, trembling hands
- Weight loss
- Muscle weakness
- Warm/moist skin, hair loss
- Staring gaze
- Burning mouth syndrome



The Clues in the Labs

Core Markers

- **Thyroid Stimulating Hormone (TSH)** — the pituitary hormone that signals the thyroid to produce more hormone
- **Total T4 & Free T4** — bound and unbound thyroid hormone
- **Total T3 & Free T3** — the active hormone your cells actually use
- **Reverse T3** — the inactive metabolite that rises under stress and illness

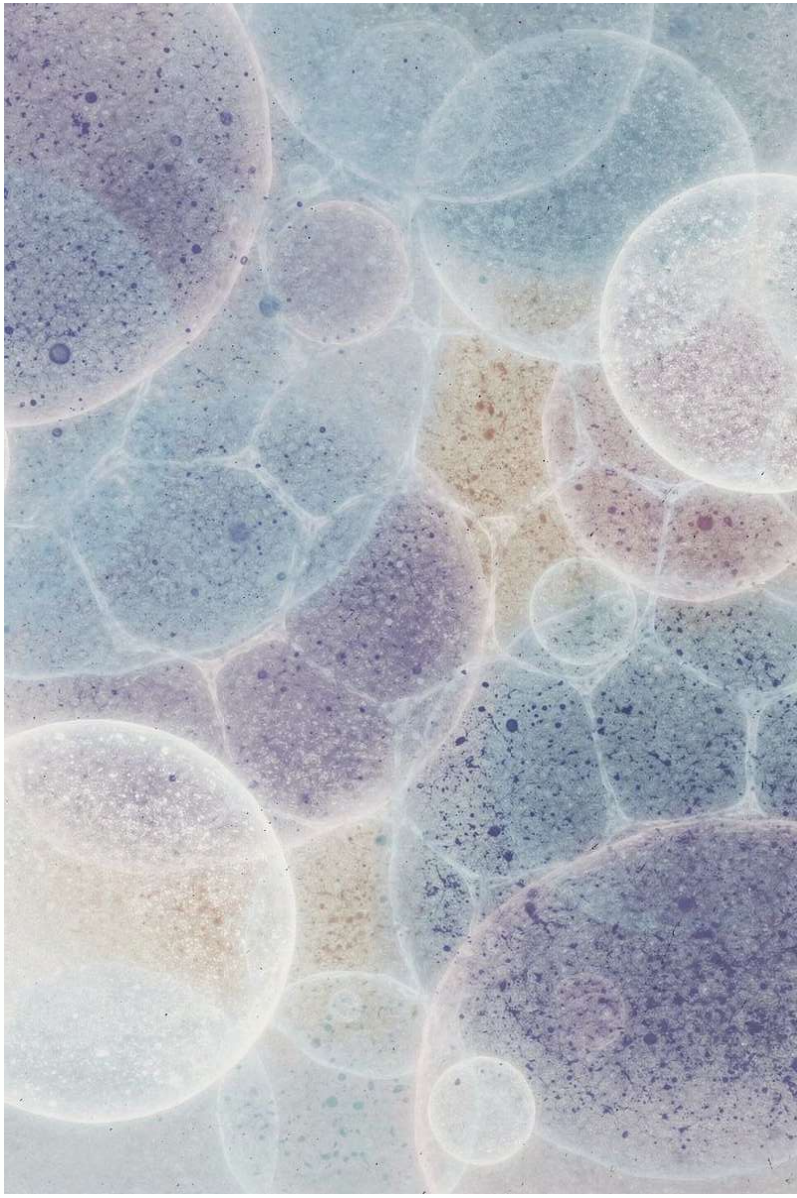
Refining the Picture

- **Thyroid Binding Globulin (TBG)** — a protein made in the liver that carries thyroid hormones through the bloodstream
- **TRH / TRH stimulation test** — differentiates secondary (pituitary) from tertiary (hypothalamic) hypothyroidism
- **T3 Uptake** — indirect measure of binding capacity, not a T3 level itself; used with Total T4 to estimate free hormone availability
- **TPO Ab, TRAb & TGB Ab** — detect autoimmune thyroid disease



The Real Culprit: Is It The Cell or the Gland?

Understanding functional tissue hypothyroidism when blood tests look normal.



T4 - T3 Conversion

T4 is largely inactive, a prohormone that is converted into active T3 at the tissue where it's needed. Conversion is carried out by three **deiodinase enzymes (D1, D2, D3)**, each doing a different job. [11]

D1 — Systemic Activator

Location: Liver & Kidney

Converts **T4 → T3** and releases it into the bloodstream for systemic use throughout the body.

D2 — Local Activator

Location: Brain, Pituitary & Muscle

Converts **T4 → T3** locally, *inside the cell* – fueling tissue-specific thyroid action without raising blood T3.

D3 — The Off Switch

Location: Widespread

Converts **T4 / T3 → Inactive rT3 & T2**, shunting thyroid hormone away from active use when needed.

Two Supply Routes

Tissues either use **circulating T3** (e.g., heart) or **make their own locally** (e.g., brain).

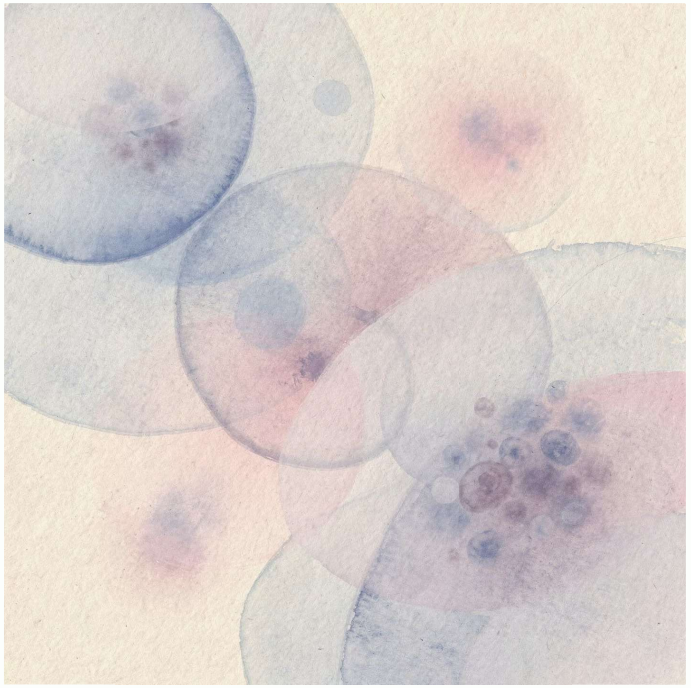
Each tissue can have a different D1:D2:D3 balance simultaneously. Therefore**, normal blood T4/T3 can coexist with tissue-level hypothyroidism** if local D3 activity is high.

[11] Bianco & Kim. J Clin Invest. 2006



Finding the Real Culprit





The Pituitary Looks Fine...

TSH primarily reflects the pituitary's local conversion, not what's happening in every other tissue. The pituitary can look "satisfied" while peripheral tissue is deprived of active hormone without changing TSH levels at all. [12, 13]

...But the Tissues Are Starved

Stress, infection, toxin load, and gut/liver dysfunction can drive the shift from homeostasis to allostasis by altering deiodinase activity (↓ D1, ↑ D3) as the body's attempt to *protect* itself under strain. [12,13]

ⓘ Many "low thyroid" patients in clinic can have functional tissue hypothyroidism with normal bloods because the root cause is acting downstream of where TSH looks [12,14,15]

[12] Chatzitomaris et al. Front Endocrinol. 2017. [13] Hoermann et al. Front Endocrinol. 2017. [14] Kalra & Khandelwal. Indian J Endocrinol Metab. 2011. [15] Abdalla & Bianco. Clin Endocrinol. 2014.

How Does it Work: Dr Balcavage's 5-Phase Model of Thyroid Dysfunction

Cellular Stress

Shift from thyroid **homeostasis** to **allostasis**; cellular hypothyroidism begins as the body adapts to strain.

Check root causes: hypoxia • nutrient depletion • heavy metal toxicity • micro-organisms • inflammation • oxidative stress • organic chemicals • toxins • medications

Cell Danger Response (CDR)

Cells **down-regulate metabolism**, including thyroid hormone transport and action, as a protective survival measure.

Autoimmune Activation

Release of **PAMPs/DAMPs** can trigger an immune attack in cases with an autoimmune component.

Subclinical Hypothyroidism

TSH begins rising as gland destruction becomes significant enough for the pituitary to detect insufficient T4.

Glandular Exhaustion

Overt hypothyroidism; **labs finally "catch up"** to what tissues have experienced for some time.

[16] Balcavage & Halderman. The Thyroid Debacle. 2022.

Same Mechanism, Different Entry Points

Chronic Stress & HPA-Thyroid Link

Cortisol dysregulation suppresses T4→T3 conversion and HPT output.

The Cell Danger Response

Cellular energy status affects thyroid hormone transport and conversion.

Immune Disruption & Molecular Mimicry

Stealth infections can trigger inflammation and cause AIT.

Liver & Gut Function

The liver drives ~80% of T4→T3 conversion; dysbiosis and gut permeability disrupt it.

Hormonal Interplay

Oestrogen influences thyroid hormone binding globulin.

Toxin Overload

Heavy metals, PCBs and halogens block conversion and limit iodine uptake.

Root cause work means finding which door the patient walked through.

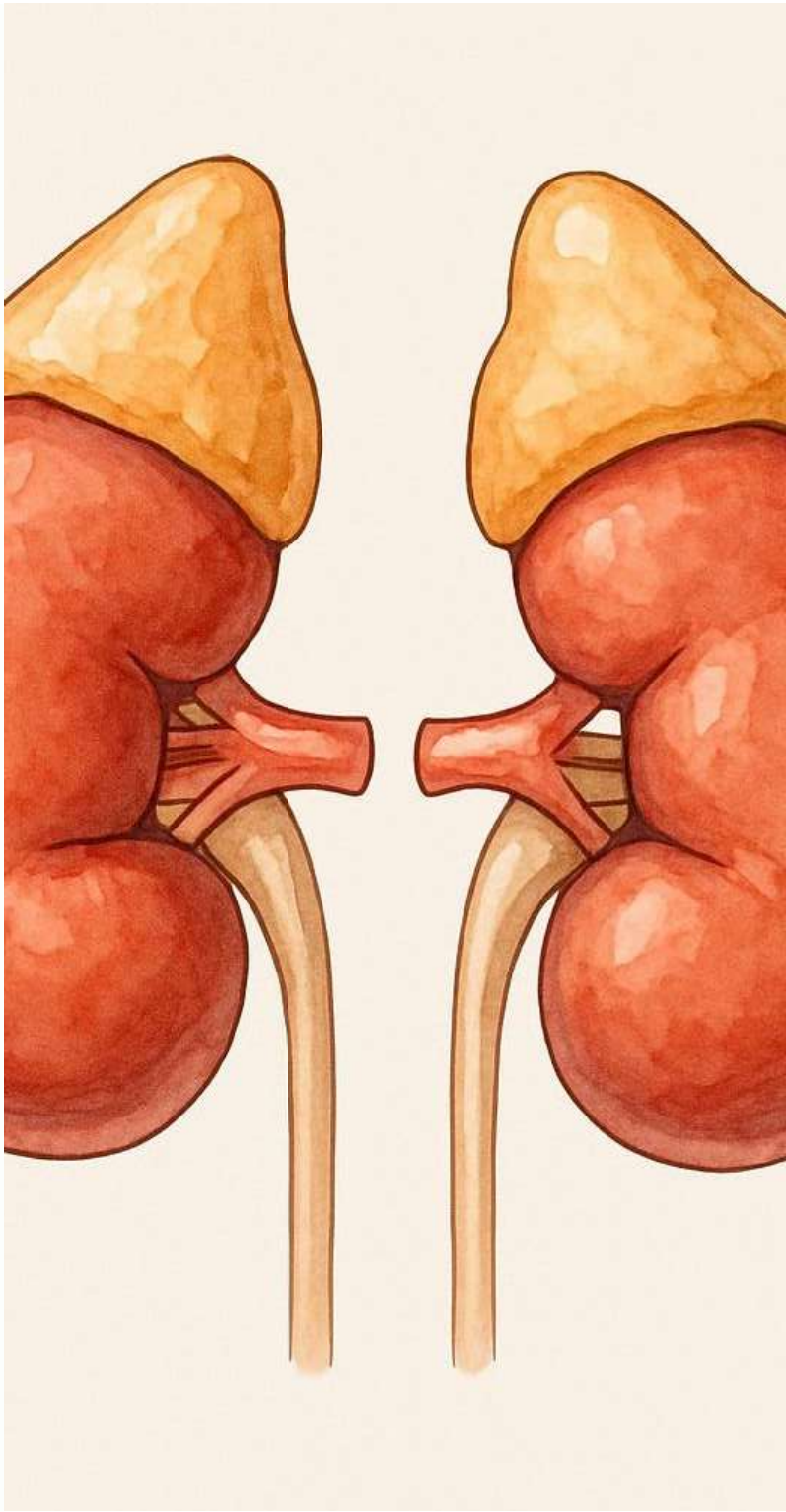


Chronic Stress & the HPA-Thyroid Link

- **Cortisol pulls the body's deiodinase balance in two different directions at once:**
  - In peripheral tissue, it reduces active T3 and raises reverse T3.
  - In the pituitary, it can initially boost local T3 production. The pituitary reports a normal TSH, even while the rest of the body is starved of active thyroid hormone. [17, 12]
- **Chronic stress erodes the system over time:**
  - Sustained HPA activation depletes glutathione, a cofactor the deiodinase enzymes need to function.
  - Oxidative stress directly blunts D2 activity, eventually even in the pituitary. [18]
  - Chronic stress drives insulin resistance, and if selenium is also deficient, more T4 gets pushed toward inactive reverse T3 rather than active T3. [19]

ⓘ A dysregulated nervous system undermines everything downstream. It will always be a priority to address first.

[17] Gierach & Junik. Endokrynol Pol. 2026. [12] Chatzitomaris et al. Front Endocrinol. 2017. [18] Mendoza & Hollenberg. Pharmacol Ther. 2017. [19] Gorini & Vassalle. Antioxidants. 2022.

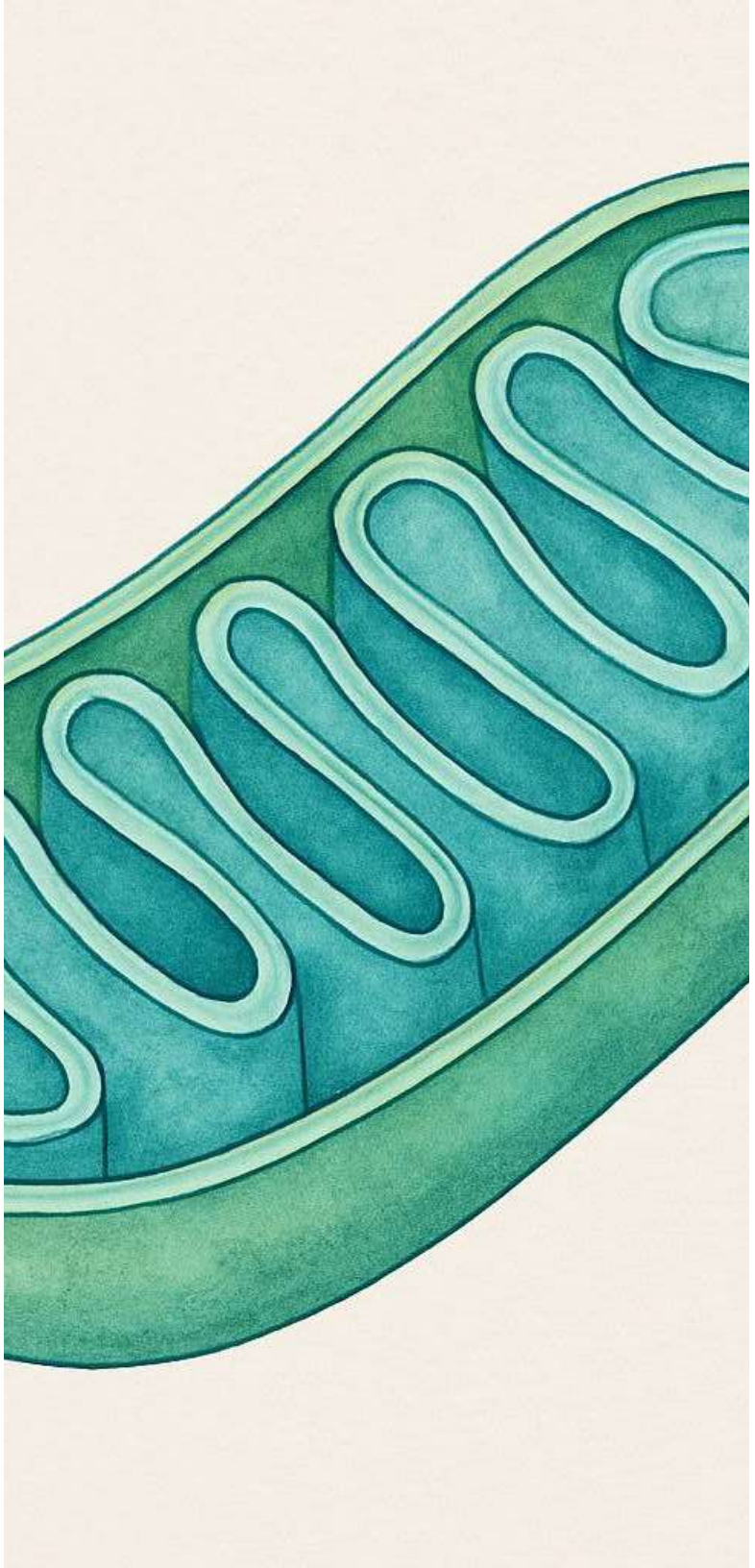


Low Energy, High Alert: The Cell Danger Response

- **Making thyroid hormone costs energy that can be disrupted by cellular strain:**
  - Iodide uptake into the thyroid is active, ATP-dependent transport.
  - Making thyroid hormone (organification) generates hydrogen peroxide, which has to be cleared by glutathione.
  - If glutathione is already depleted from chronic stress, hormone synthesis is affected. [20, 18]
  - **It's a two-way street:** T3 itself boosts mitochondrial ATP production so poor thyroid status and poor cellular energy feed each other. [21]
- **The Cell Danger Response is the umbrella mechanism:**
  - Under any threat — infection, toxins, hypoxia, heavy metals, inflammation, nutrient depletion — cells down-regulate metabolism as protection, including how thyroid hormone gets into cells (via dedicated transport) and how it's converted once there (deiodinase activity). [22, 23]

> ⓘ Low cellular energy = low thyroid function. The key is finding the driving cause.

[20] Szanto et al. Int J Mol Sci. 2019. [18] Mendoza & Hollenberg. Pharmacol Ther. 2017. [21] Short et al. Am J Physiol Endocrinol Metab. 2001. [22] Naviaux. Mitochondrion. 2014. [23] Wittmann et al. Fluids Barriers CNS. 2015.



Immune Disruption & Molecular Mimicry

Infections can trigger autoimmune thyroid attack through overlapping mechanisms: molecular mimicry (a pathogen's proteins resemble thyroid tissue), bystander activation (collateral immune damage while fighting infection), and cryptic antigen exposure (tissue damage reveals thyroid proteins the immune system has never "seen" before). [24]

Viral:

- **EBV** — higher IgG+ titres in children with autoimmune thyroiditis. [25]
- **HSV II** — linked to new-onset Hashimoto's. [26]
- **HCV** — increases AIT risk, treated and untreated. [27]

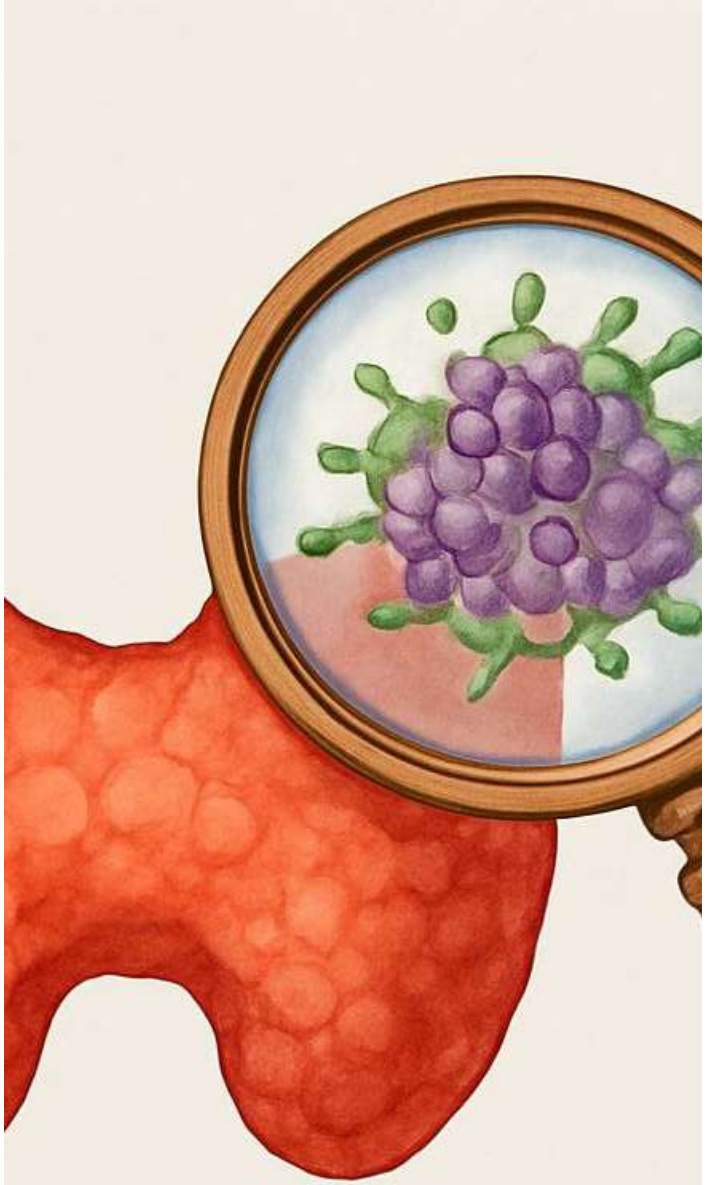
Bacterial:

- **Yersinia enterocolitica** — shares amino acid sequences with thyroid tissue (direct molecular mimicry); meta-analysis shows a significant association with AITD overall, strongest for Graves'. [28,29,30]
- **H. pylori** — found in 86% of one AIT cohort vs. 40–45% of controls. [31]

Clinical observation: Lyme disease and co-infections are associated with hypothyroidism in practice, though needs further research.

Head to synergetic.med for An Integrative Approach to Chronic Fatigue- Viral Patterns in CFS.

[24] Benvenga & Guarneri. Rev Endocr Metab Disord. 2016. [25] Thomas et al. Endocrine. 2008. [26] Di Crescenzo et al. Infez Med. 2013. [27] Ferri et al. Front Endocrinol. 2017. [28] Benvenga et al. Thyroid. 2006. [29] Guarneri et al. Thyroid. 2011. [30] Zangiabadian et al. Endocr Metab Immune Disord Drug Targets. 2021. [31] de Luis et al. J Clin Gastroenterol. 1998.

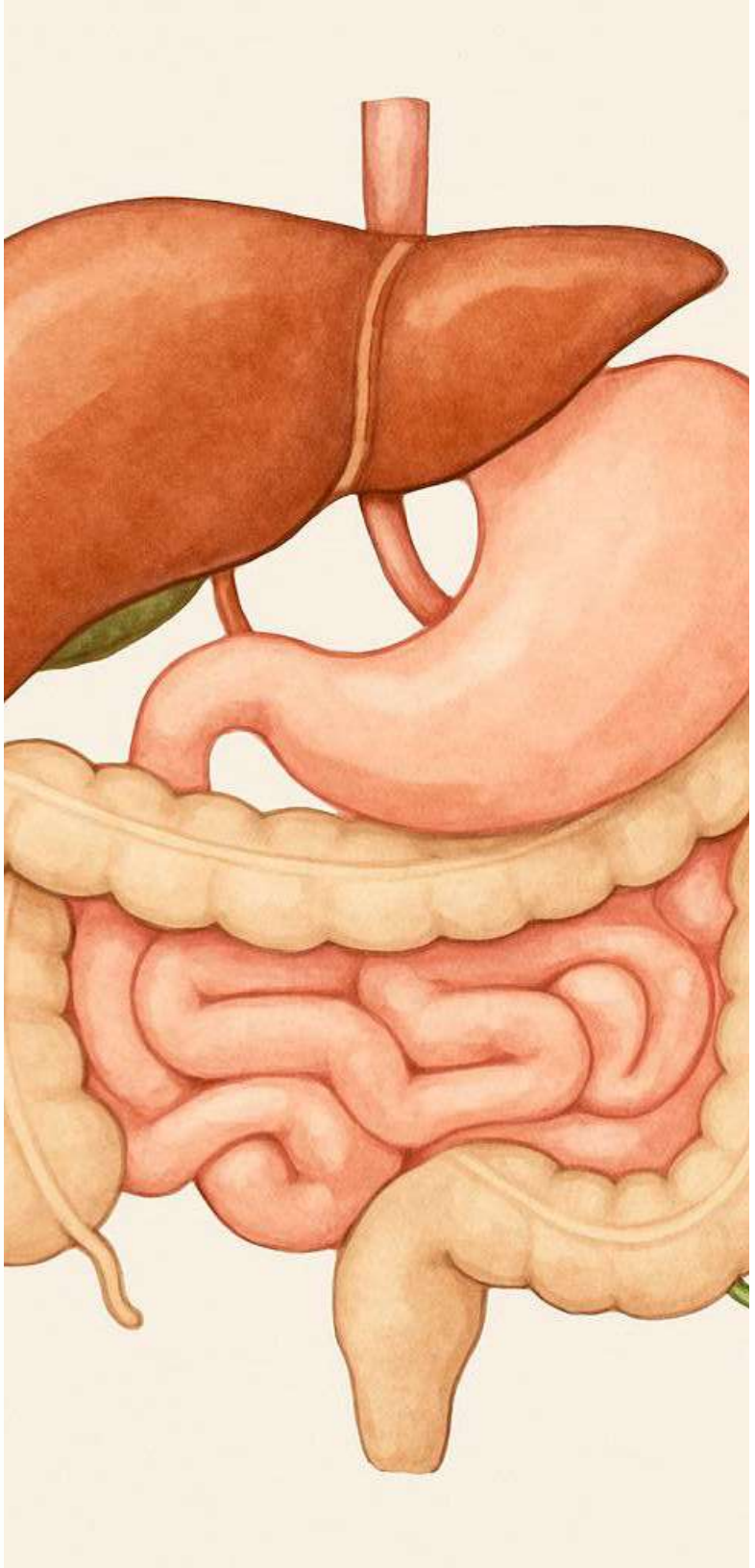


Liver & Gut: The Conversion Engine

- **The liver drives most circulating T3:**
  - ~80% of **blood-level** T4→T3 conversion happens via the liver (D1). Liver congestion, fatty liver, or impaired detox capacity can all reduce this conversion capacity, so a struggling liver can present as a thyroid problem even when the gland itself is fine.[32, 33]
- **A vicious cycle:**
  - The remaining ~20% relies on gut bacterial activity. But low thyroid function slows gut motility impairing T4→T3 conversion. [34,35]
- **Deeper insights on dysbiosis:**
  - Some beneficial bacteria contribute to tyrosine biosynthesis, a shared building block for both thyroid hormone and adrenal catecholamines so dysbiosis can affect thyroid and adrenal function together. [36]
  - Gut bacteria also control thyroid hormone recycling meaning dysbiosis can make thyroid hormone availability, and even levothyroxine absorption, unpredictable.[37]

ⓘ What presents as a thyroid problem could be secondary to gut dysbiosis or yeast overgrowth.

[32] Bruinstroop et al. Eur Thyroid J. 2023. [33] Marschner et al. Cells. 2023. [34] Yaylali et al. Gastroenterol Res Pract. 2009.[35] Xu et al. Front Physiol. 2024.[36] Liu et al. Front Endocrinol. 2023. [37] Virili & Centanni. Mol Cell Endocrinol. 2017.

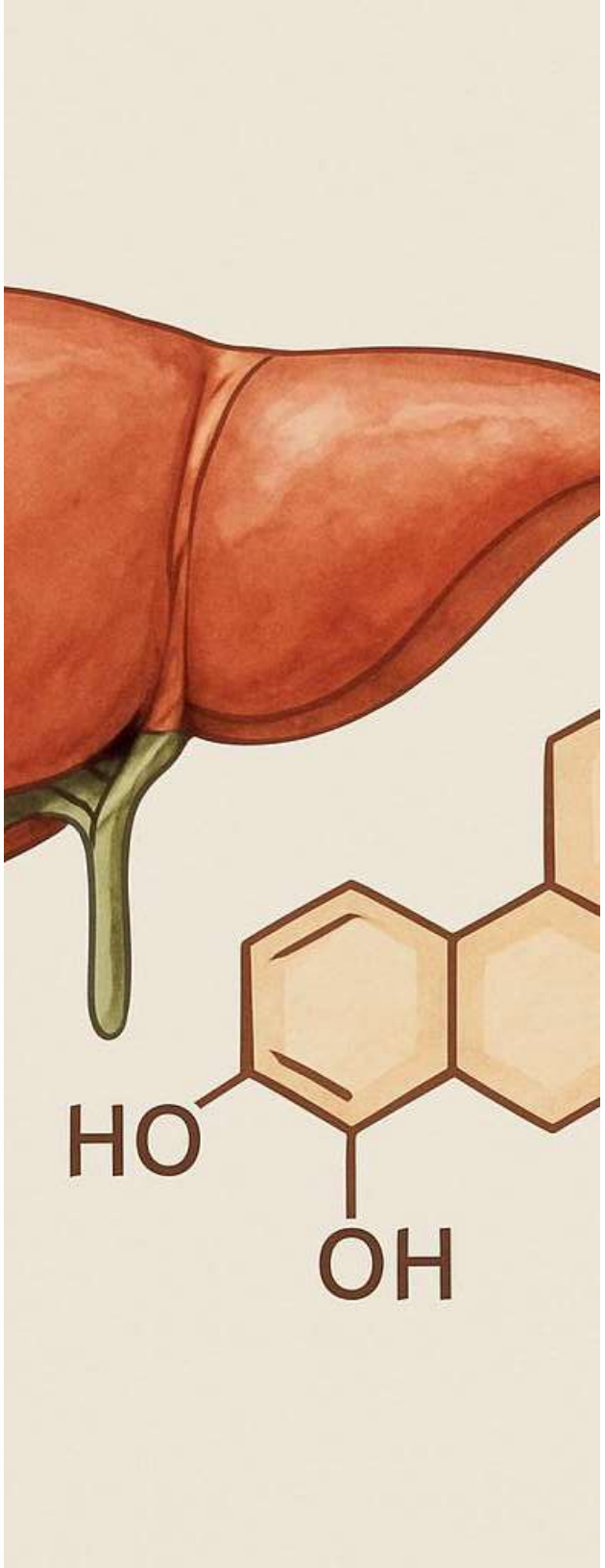


Oestrogen Dominance & TBG Dysregulation

- **The basics:**
  - 99% of thyroid hormone travels through the blood attached to a carrier protein, thyroid-binding globulin (TBG). Only the small unattached "free" portion is actually active in the body
- **Why this matters:**
  - More TBG means more hormone gets locked up and unavailable → hypothyroid symptoms. Less TBG means more hormone is free and active → hyperthyroid symptoms
- **Oestrogen is a major driver of this:**
  - It slows the clearance of TBG from the blood, raising circulating levels. Women on HRT or with oestrogen dominance can have hypothyroid symptoms even when their total T4 looks completely normal. [38]
- **Testing Implications:**
  - TBG is made in the liver, so an elevated result isn't always hormonal. Check liver function before assuming oestrogen excess.
  - Always check **Free** T4/T3 alongside **Total** T4/T3. Total alone can look reassuring while the patient is functionally hypothyroid.
- **An additional consideration?**
  - In cell studies excess oestrogen decreased sodium-iodide symporter expression reducing the thyroid's ability to take up iodide. [39]

Head to synergetic.med for free course on Hidden Patterns of Hormonal Issues

[38] Ain, Mori & Refetoff. J Clin Endocrinol Metab. 1987. [39] Furlanetto et al. Endocrinology. 1999.



Environmental Toxins & Thyroid Disruption

- **Environmental compounds compete with iodine**
  - Several common exposures, perchlorate (found in some water supplies and fertilizers), nitrate, and fluoride, compete with iodine for the same thyroid uptake pathway. [40]
- **Heavy metals disrupt thyroid function at multiple points:**
  - Mercury, lead, and cadmium accumulate in thyroid tissue and raise antibody levels. [41, 42, 43]
  - Cadmium specifically blocks D1 (5′-deiodinase), the enzyme needed to convert T4 into active T3. [44]
- **PCBs mimic the hormone itself**
  - Their structure is similar enough to thyroid hormone that they can bind directly to thyroid receptors, sometimes activating them, sometimes blocking them. [45]
- **Radiation hits the uptake mechanism:**
  - Radiation disrupts the sodium-iodide symporter directly, reducing iodide uptake. [46] Separately, occupational exposure to ionising radiation is linked to autoimmune thyroid disease. [47]
- **Pesticides disrupt thyroid function through multiple mechanisms**
  - Including blocking iodine uptake at the sodium-iodine symporter, inhibiting the enzymes needed for hormone synthesis and conversion, and interfering with hormone transport and receptor binding. Organochlorine exposure specifically is linked to increased hypothyroidism risk. [48, 49, 50]

Head to synergetic.med for An Integrative Approach to Chronic Fatigue- Detoxification

[40] Tonacchera et al. Thyroid. 2004. [41] Pamphlett et al. PLoS ONE. 2021. [42] Abdelgawwad El-Sehrawy et al. J Trace Elem Med Biol. 2025. [43] Nie et al. Environ Pollut. 2017. [44] Gupta & Kar. Comp Biochem Physiol. 1999. [45] Wang et al. Emerging Contaminants. 2025. [46] Nordén et al. Cancer Res. 2007. [47] Völzke et al. J Clin Endocrinol Metab. 2005. [48] Brent. Thyroid. 2010. [49] Leemans et al. Front Endocrinol. 2019. [50] Sirikul & Sapbamrer. BMC Public Health. 2023.

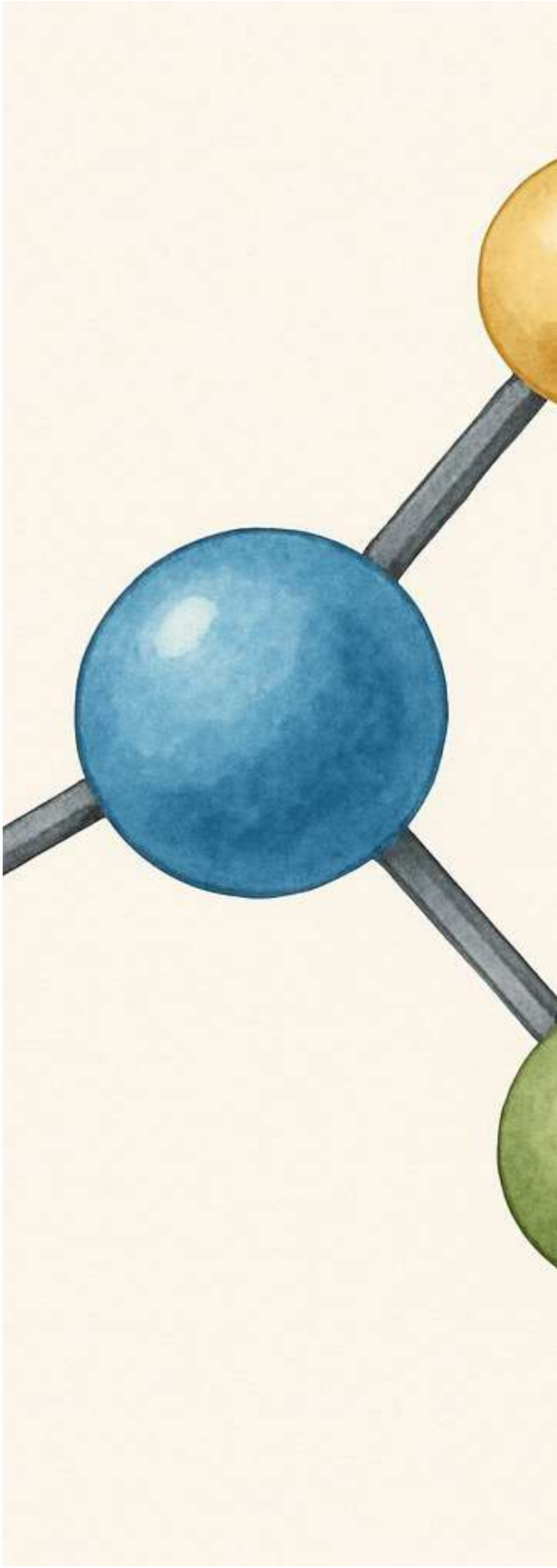


Nutrient Cofactors: Iodine, Tyrosine & Selenium

- **Iodine:**
  - 70–80% of the body's iodide is stored in the thyroid. [51] Getting it into the gland requires active, ATP-dependent transport (the sodium-iodide symporter) and building the hormone itself generates hydrogen peroxide, which needs glutathione to clear safely. [20]
  - Iodine supplementation may increase oxidative stress if glutathione reserves are already low. [20]
- **Tyrosine:**
  - The amino acid backbone for both thyroid hormone and adrenal catecholamines, a potential shared bottleneck between the two systems. [52] Needs phenylalanine, iron, biopterin, and a healthy gut to form properly. [36]
- **Selenium:**
  - Selenium plays a dual role — both a cofactor for the enzymes that convert T4 to T3, and an antioxidant (via glutathione peroxidase). [15, 19]
  - Infection can increase the body's peroxide load and deplete selenium/glutathione reserves, a documented mechanism in viral infections like COVID-19, competing with thyroid hormone synthesis for the same limited resources. [53,54]
- **The throughline:**
  - Iodine uptake runs on cellular energy, selenium reserves can be depleted by infection, and tyrosine depends on gut health to form in the first place.

Fix the foundation first: you can't out-supplement a system that isn't running well underneath

[51] Zimmermann. Endocr Rev. 2009. [20] Szanto et al. Int J Mol Sci. 2019. [52] Cooper, Bloom & Roth. Biochemical Basis of Neuropharmacology. 1986. [36] Liu et al. Front Endocrinol. 2023. [15] Abdalla & Bianco. Clin Endocrinol. 2014. [19] Gorini & Vassalle. Antioxidants. 2022. [53]Taylor & Radding. Front Nutr. 2020. [54] Karbownik-Lewińska & Kokoszko-Bilska. Thyroid Res. 2012.



Cracking the Case:
A Framework for Thyroid Support



Clinical Decision Pathway

Simplify every case by asking the right questions in the right order.



Phase 1: Stabilise First

Ask: *is the nervous system regulated enough to support deeper work?*

Sleep, anxiety, and fatigue point back to the HPA-thyroid link. Instability undermines everything downstream, supporting the nervous system helps interventions to hold.



Phase 2: Open Drainage

Ask: *are elimination pathways actually open?*

If liver or gut function is impaired, progress elsewhere will be limited. Aggressive intervention can mobilise burden faster than the body can clear it, some patients need better drainage vs. detoxification.



Phase 3: Reduce Toxic Load

Ask: *is there a specific contributor to address?*

Where clinically indicated, investigate and address environmental, microbial, or toxicological triggers now that drainage capacity is in place.



Phase 4: Target the Dominant Driver

Ask: *which is most active in this patient, right now? Stress,, immune, gut/liver issues, toxicity?*

With the terrain prepared, interventions tend to be more effective and less likely to trigger symptom flaring or relapse.



Monitor closely, throughout:

Track symptom patterns against menstrual cycle, energy, sleep, and digestive function as ongoing clinical indicators.



If Symptoms Worsen → Step Back

System overwhelmed → return to stabilise, drainage or support phase. Worsening is information, not failure.



Reassess the driver before escalating:

If a protocol isn't working, revisit which root cause is actually dominant before adding more support. Complexity is rarely the solution



Use Symptom Support Early

Incorporate tools to support the management of symptoms while deeper work is underway.

Clinical framework — Synergetic Botanicals, informed by root-cause mechanisms covered in this session.

Further Practitioner Resources

The Synergetic Method is designed to help practitioners move beyond symptom-led approaches and explore ways of incorporating naturopathic and herbal approaches into practice alongside your main modality.

We are currently building a growing practitioner education platform including:

- free live webinars for practitioners
- webinar recordings available through the **Synergetic.med** practitioner platform
- practitioner-focused treatment protocols and clinical resources
- education around integrating herbs and naturopathy into practice
- webinars exploring practical naturopathic and homeopathic approaches

Supporting practitioners to explore the underlying factors influencing health and building confidence in using an integrative clinical tool kit.



SYNERGETIC
Botanicals

Synergetic Botanicals: A Systems-Based Approach

Synergetic Botanicals formulations are designed to work with the body's regulatory systems, using herbs that support the terrain rather than targeting isolated symptoms.

Built on Clinical Principles

Multi-System Support

Formulas are designed to support regulation, processing and load, addressing the terrain rather than isolated pathways.

Modulation, Not Override

Working with physiology rather than forcing change, supporting the body's own regulatory capacity.

Simplified Protocols

Reducing the need for multiple single-ingredient supplements, each formula is designed to do more with less.

Phase-Based Application

From stabilisation to detoxification. Formulas designed to be introduced at the right stage of the protocol.

Synergetic Botanicals



Phase 1: Stabilisation

Phase 1 stabilises the HPA axis and cellular resilience to build foundation.

- **SB Adrenal**
- **SB Cell Protect**
- **SB Nerve & Gut**



Phase 2: Drainage

Phase 2 opens the gut, liver and lymphatic pathways.

- **SB Bitters**
- **SB Digest**
- **SB Lymph**



Phase 3: Detoxification

Phase 3 supports the body to eliminate what is mobilised.

- **SB Fulvic**
- **SB Digest**
- **SB Super Detox**



Phase 4: Target Driver

Phase 4 targets whichever driver is dominant for this patient.

- **SB Myco**
- **SB Para**
- **SB Defend**

Synergetic Botanicals range are available from **Your Health Basket**, **Amrita Nutrition** and **synergeticbotanicals.co.uk**

Case Files:

Real Patients & Q&A



CASE STUDY 1

Immune-Driven

Presentation & Findings

- 32 year old female
- Sharp head pains like pick-axe
- Intermittent loss of sensation in arm/leg
- Brain fog
- History of glandular fever at 15
- Noticed gluten sensitivity
- Constipation
- Prone to yeast infections
- TgAB - 158, Total T4 96, FT3 4.7, TSH 3.02

Approach

- Intermittent neuro symptoms suggest inflammation beyond the thyroid - we used SB Nerve & Gut with New Vistas Nervous System Liquescence to support this.
- Raised TgAb with EBV in history suggest immune dysregulation - we used SB Defend and New Vistas EBV to combat this
- Recurrent yeast infections give us another source of immune stimulation - we used SB Myco to address fungal balance.

Outcome

- Head pains resolved
- Sensation disturbances stopped
- Brain fog and constipation significantly improved
- Yeast symptoms settled
- TgAb reduced on follow-up
- Overall thyroid function and wellbeing improved

Case study. Individual outcomes cannot be generalised

CASE STUDY 2

Toxicity Driven

Presentation & Findings

- 42 year old female
- Heavy, painful periods
- Constipation, bloating & wind
- Poor sleep, energy, low mood
- Hair loss & weight gain
- Stressful job
- High arsenic and mercury on testing (amalgam fillings)
- TSH 3.29, T4 12.5, TgAb

Approach

- Constipation and bloating suggest poor elimination may be slowing toxin removal - we used Rose Hip Plus for better clearance.
- Heavy, painful periods suggest liver-clearance burden - we supported elimination first with SB Digest and New Vistas Digestive Liquescence.
- The toxic burden may be impairing thyroid function - we used New Vistas Metex and SB Super Detox to support clearance.

Outcome

- Bloating and constipation improved
- Periods became lighter and less painful
- Sleep, mood and energy improved
- Hair shedding reduced
- Weight began to stabilise
- Heavy-metal findings reduced on retesting
- Thyroid markers moved in a more favourable direction

Case study. Individual outcomes cannot be generalised

CASE STUDY 3

Stress & Adrenal Driven

Presentation & Findings

- 36 year old female
- Shortened cycles with light bleeding (21-26 days)
- Prone to constipation, fissures & wind (managed through diet and avoiding gluten)
- Easily fatigued, poor circulation, cold extremities
- Irregular appetite, stressful job
- Thinning hair, dry skin
- Testing-
- TSH 1.42, TPO Ab 13.30, T4 15.2, TgAB antibodies - 258 iu/l

Approach

- Fatigue, cold extremities and a job suggest adrenal and stress-system involvement - we used SB Adrenal to support regulation.
- Dry skin, thinning hair and poor circulation suggest reduced cellular nourishment and fatty acid support - we used SB Cell Protect Oil to support cell membranes and inflammatory balance.
- Irregular appetite and constipation suggest poor gut unction - we used SB Fulvic to support mineral availability, and gut biome.

Outcome

- Energy and stamina improved
- Cold hands and feet became less noticeable
- Bowel function became more regular
- Hair shedding reduced and skin became less dry
- Cycles became more consistent
- She felt more resilient to stress and less easily depleted

Case study. Individual outcomes cannot be generalised

Key Clinical Takeaways

- **Normal labs don't rule out thyroid dysfunction.**
  - The gland can look fine on standard bloodwork while tissues are still starved of active hormone. The labs are the start of the case, not the end of it.
- **Same markers, different drivers, different protocols.**
  - Stress, infection, toxins, gut/liver dysfunction, hormones, and mitochondrial strain can all produce the same picture, knowing which one to address moves you beyond generic protocols to getting deeper solutions for your clients.
- **Working on foundations first helps interventions hold.**
  - Stabilise → Open Drainage → Reduce Toxic Load and then Target the Dominant Driver. Skip ahead, and even the right intervention won't stick.



Thank You & Questions

Next Steps:

- Join our free in-depth hormones course **https://synergetic.med/course/hormonebalance**
- Find Our Practitioner-Only Formulas at **synergeticbotanicals.co.uk** or Your Health Basket and Amrita Nutrition
- **Advanced Practitioner Qualification in Homotoxicology & Integrative Detoxification** on 24th & 25th October (in London and Online)

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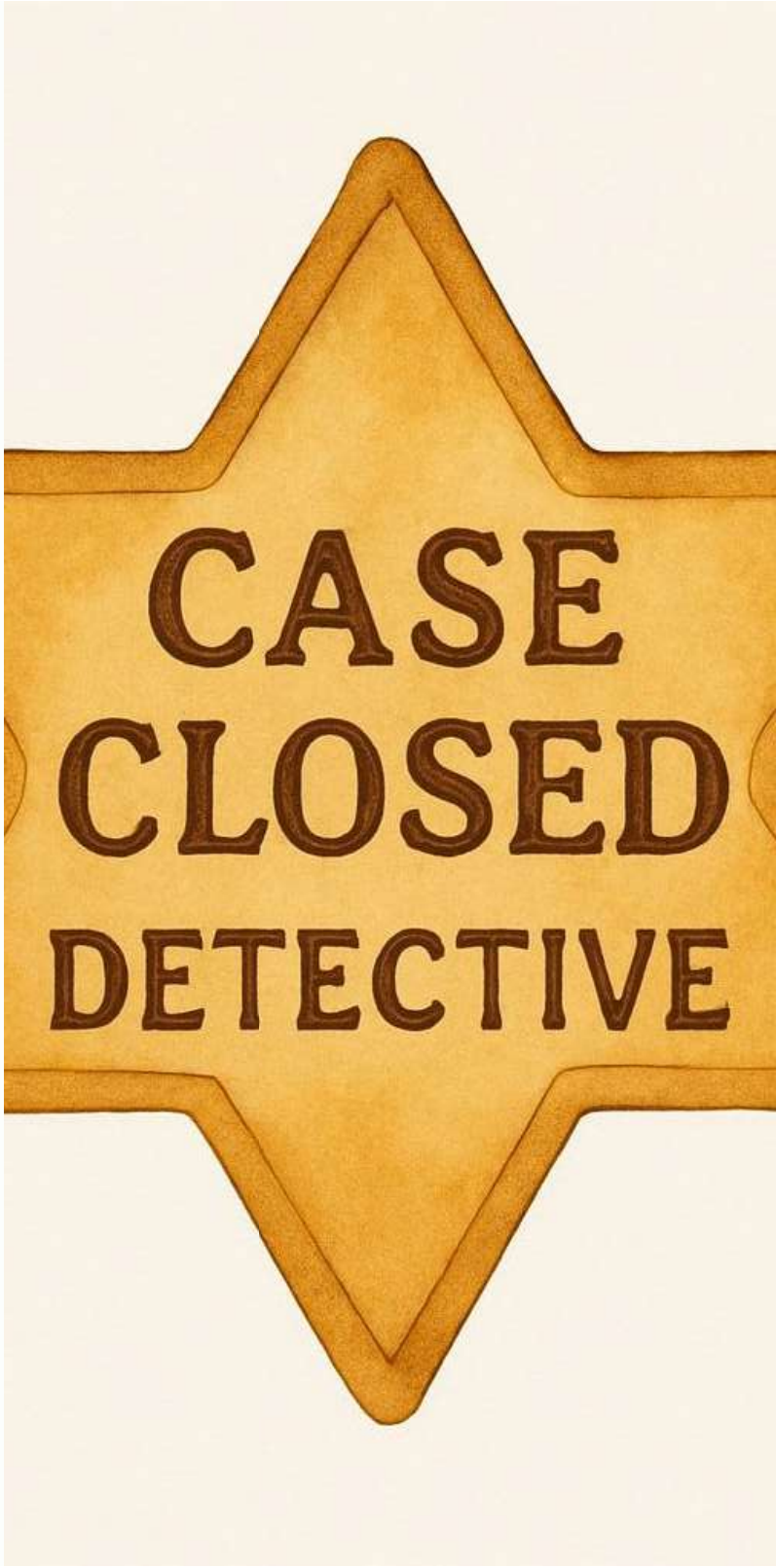

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