



THYROID MASTERCLASS WITH DR GHAZALA AZIZ-SCOTT



WELCOME



DR GHAZALA AZIZ-SCOTT

BHRT Expert & Functional Medicine Doctor

LEARNING OBJECTIVES

- Understand the anatomy and function of the thyroid gland
- Explain the function of all thyroid hormones
- Describe the conditions which lead to abnormal thyroid production
- Understand the synthesis of thyroid hormones and nutritional requirements for their production
- Demonstrate knowledge of all different thyroid medications available, both synthetic and desiccated
- Confidently evaluate thyroid disorders and treat/refer appropriately



COPYRIGHT

This presentation is Copyright © 2022 Marion Gluck Training Academy (MGTA)
This presentation was prepared by the Marion Gluck Training Academy in collaboration with the presenter.

RECORDING DISCLAIMER

By participating in this live webinar, please note that your name and/ or email address may appear in the livestream.

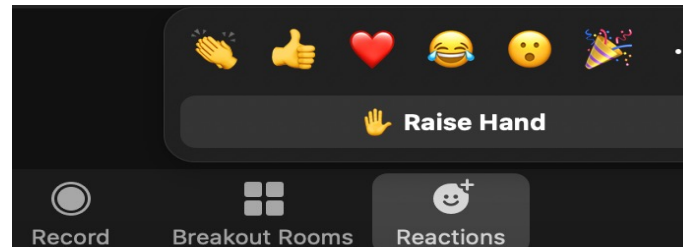
EDUCATIONAL DISCLAIMER

The information contained in this course represents the views and opinions of the presenter and do not necessarily reflect the views of the MGTA. The mere appearance of Video Content on the Site does not constitute an endorsement by MGTA or its affiliates of such Video Content. The Video Content has been made available for informational and educational purposes only. MGTA does not make any representation or warranties with respect to the accuracy, applicability, fitness, or completeness of the Video Content. MGTA does not warrant the performance, effectiveness or applicability of any sites listed or linked to in any Video Content. The Video Content is not intended to be a substitute for professional medical advice, diagnosis, or treatment. Always seek the advice of your physician or other qualified health provider with any questions you may have regarding a medical condition. Never disregard professional medical advice or delay in seeking it because of something you have read or seen on the Site. MGTA hereby disclaims any and all liability to any party for any direct, indirect, implied, punitive, special, incidental or other consequential damages arising directly or indirectly from any use of the Video Content, which is provided as is, and without warranties.



ZOOM ETIQUETTE

- Please keep yourself on **mute** throughout.
- Please submit questions through the **chat function** throughout the course. These will be responded to at the end of each section.
- There will be a Q&A section at the end of the course. Please '**raise your hand**' if you would like to ask a question



- You will then be taken off mute and can speak to the group

AGENDA



10:00 Start

- Introduction
- Thyroid anatomy and function
- Negative feedback and the HPT axis
- Thyroid hormone actions and metabolism
- Nutritional factors affecting thyroid conversion and thyroid hormone synthesis

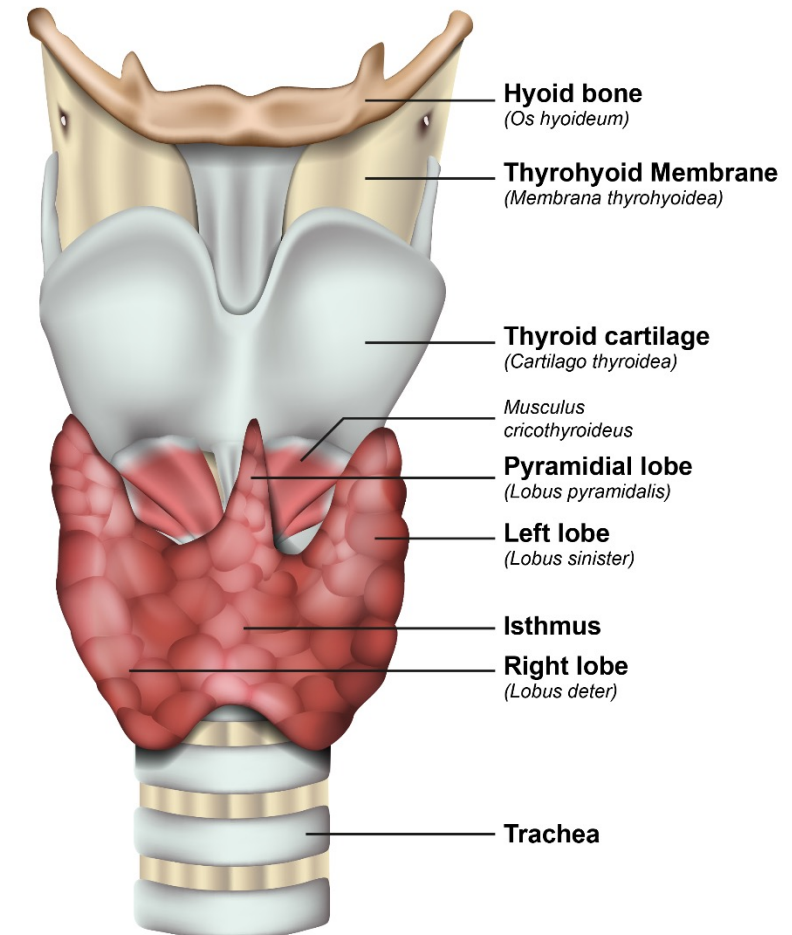
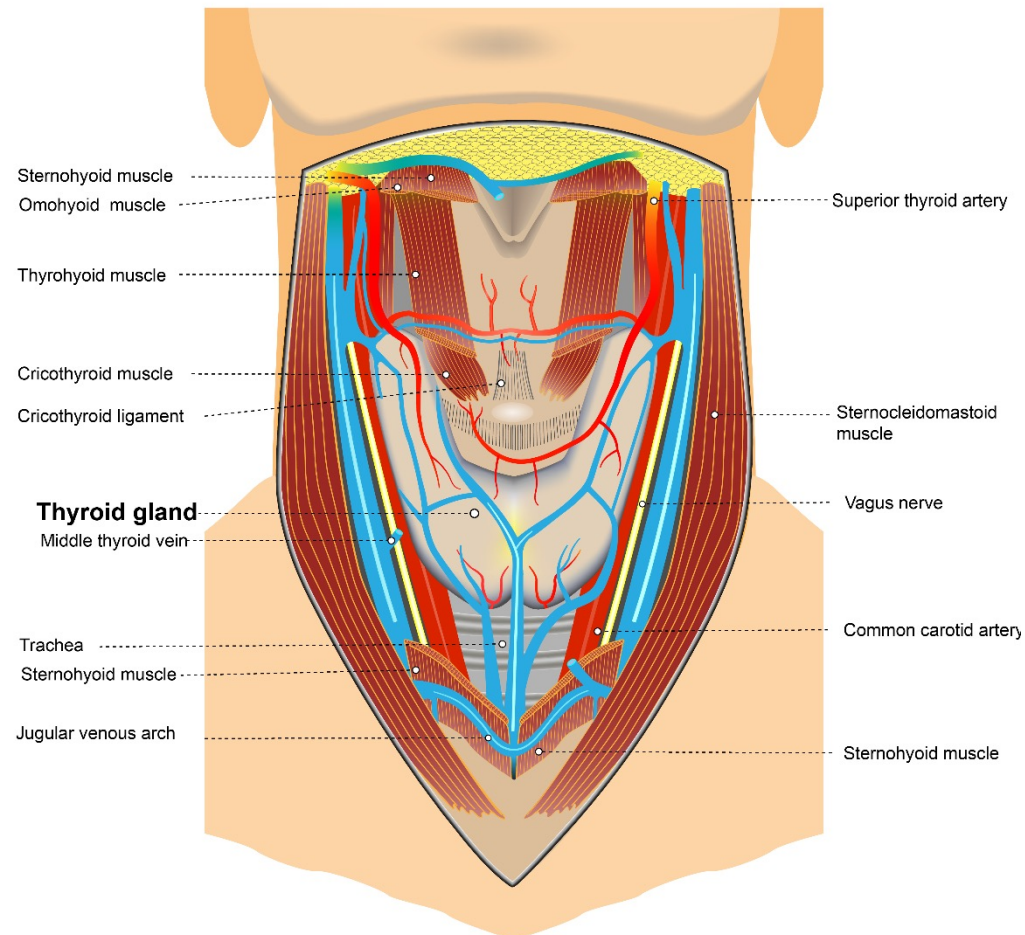
Break (10 mins each hour)

- Hormones and thyroid dysfunction
- BHRT thyroid consultation
- Blood testing
- Prescribing protocols using NDT
- Controversies surrounding NDT
- A look at conventional thyroid treatment guidelines
- A functional medicine approach to thyroid disorders
- Case studies
- Overview and take-home points
- Q&A

13:30 Finish

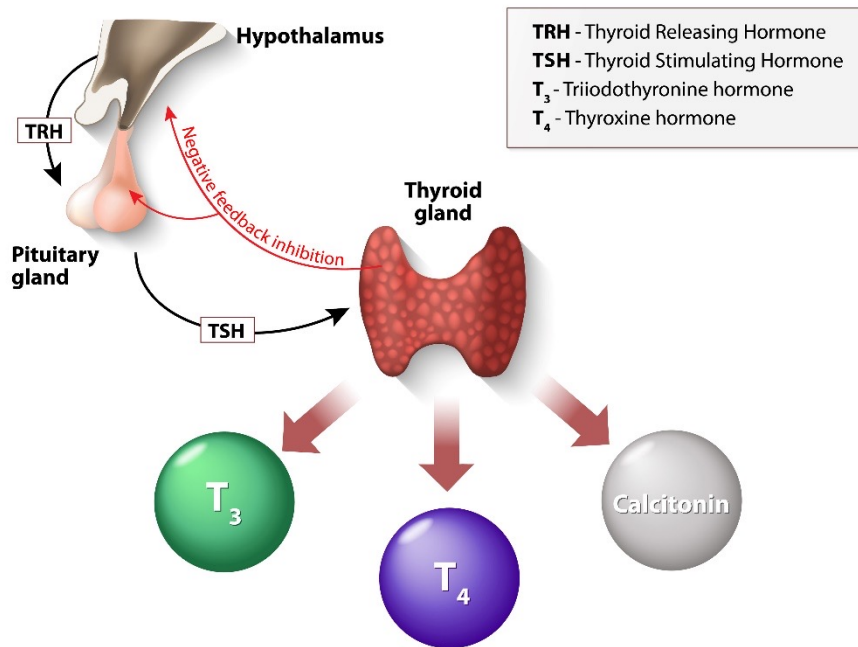
THYROID ANATOMY

The thyroid gland is one of the largest endocrine organs in the body. Its primary role is to **secrete thyroid hormones** which play a role in regulating the metabolism.



THYROID HORMONES

The production and release of thyroid hormones, T₄ and T₃ is controlled by a **feedback loop** involving the **hypothalamus**, **pituitary gland** and **thyroid gland**.

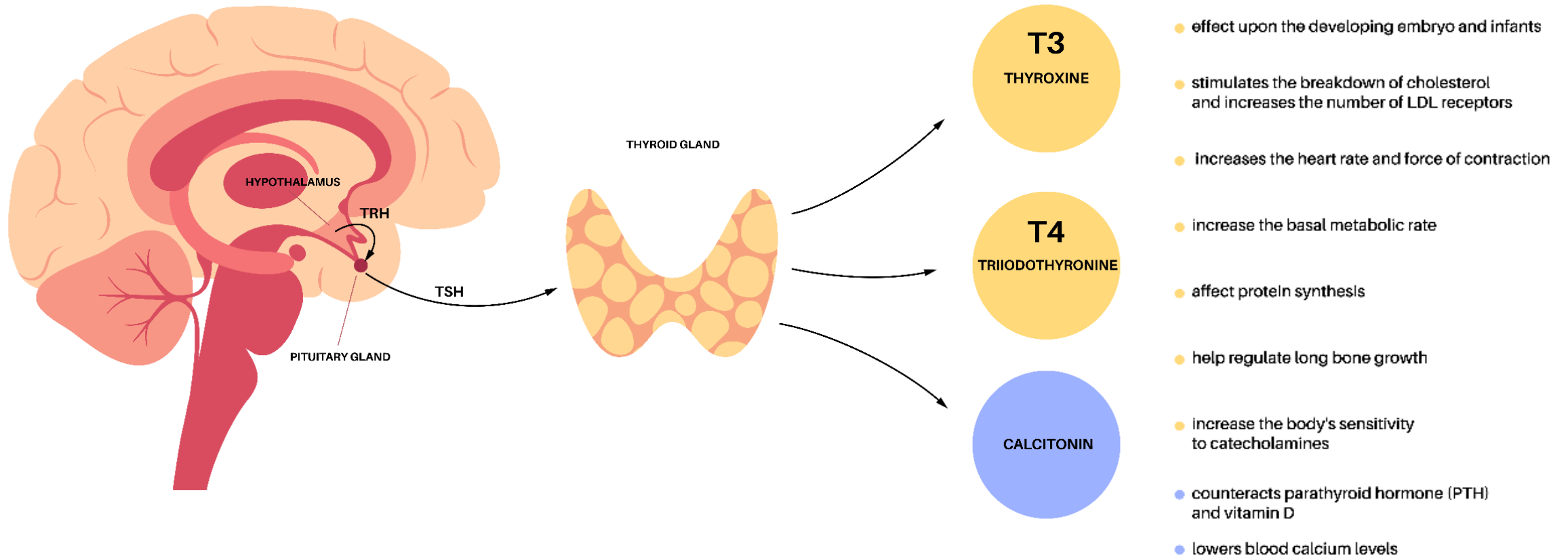


- When levels of T₄ and T₃ rise, they prevent the release of TRH from the hypothalamus and TSH from the pituitary gland.
- This allows a constant level of thyroid hormones within the body and defining the individual's set point.
- T₄ is metabolised in extra-thyroidal tissue to T₃ (active), reverse T₃ (inactive).

Activation of thyroid hormones is then controlled in body tissues such as the liver, brain and kidneys by enzymes called deiodinases which convert thyroxine into the active form **triiodothyronine**.

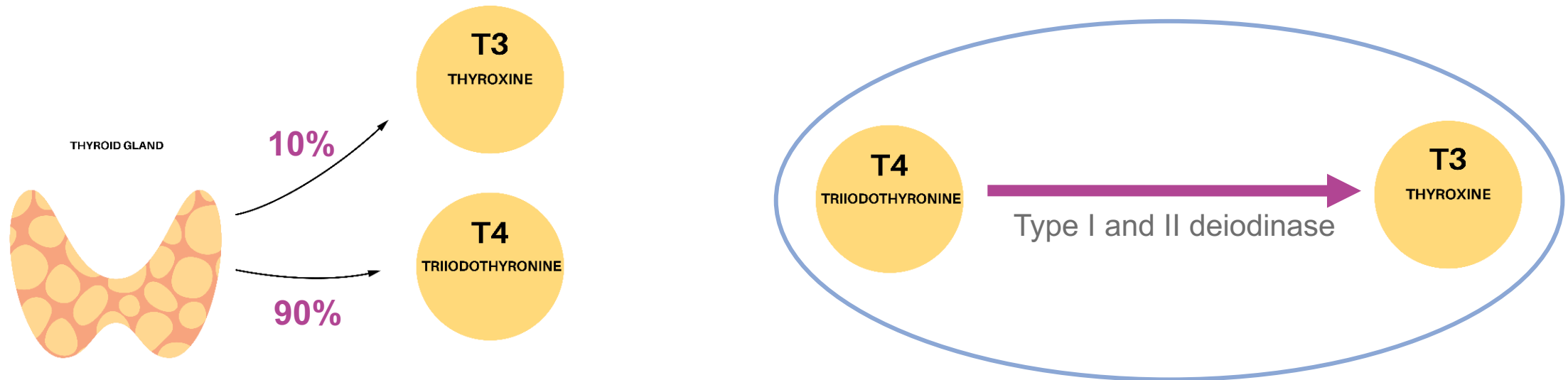
NEGATIVE FEEDBACK

Negative feedback is needed to maintain normal thyroid hormone levels.



THYROID HORMONE FUNCTION

- TSH from the pituitary stimulates the thyroid gland to secrete thyroid hormone into the blood.



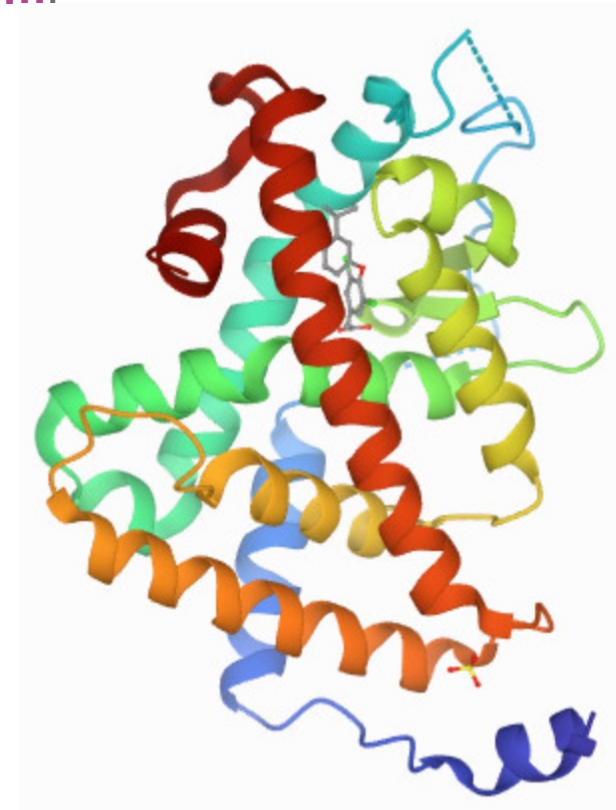
- T3 can signal at the thyroid hormone receptor present in the nuclei.
- One of the primary actions of thyroid hormone is the **control of growth**, particularly in childhood.
- In adults, the primary action is **control of metabolism**.

THYROID RECEPTORS

Thyroid hormone receptors (TRs) are types of **nuclear receptors** which act as transcription factors, affecting gene transcription and translation. They exert their effects on all cells within the body and regulate many physiological processes such as **growth, development** and **metabolism**.

All three type of receptors can bind with thyroid hormones:

- **TR- α 1** (widely expressed, esp. in cardiac and skeletal muscles)
- **TR- β 1** (mainly expressed in brain, liver and kidneys)
- **TR- β 2** (primarily expressed in hypothalamus and pituitary).



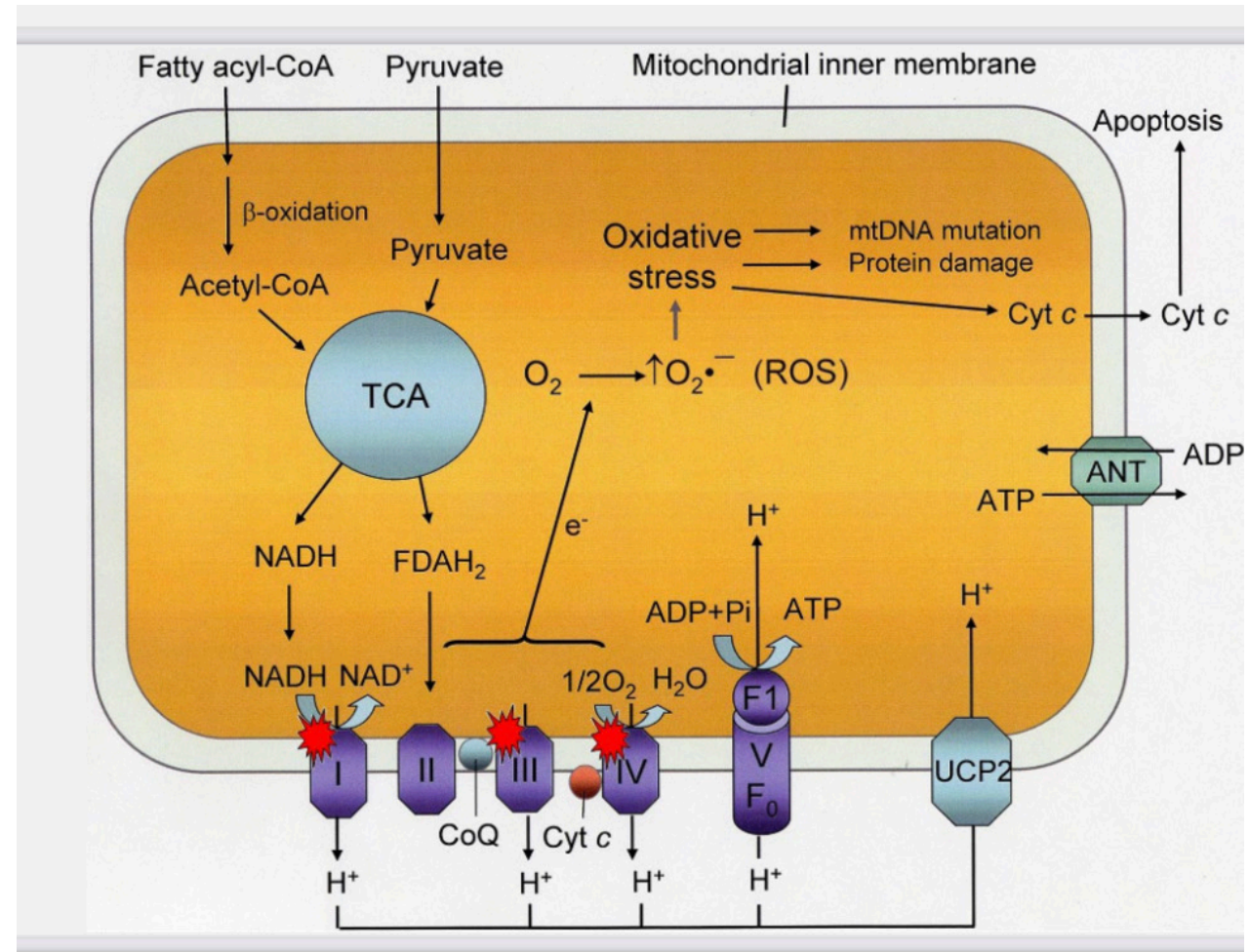
PDB ID: 1NAV

DOI Citation: Ye, L. et al (2003) Thyroid receptor ligands. 1. Agonist ligands selective for the thyroid receptor beta1

doi: [10.1021/jm021080f](https://doi.org/10.1021/jm021080f)

THYROID HORMONE FUNCTION

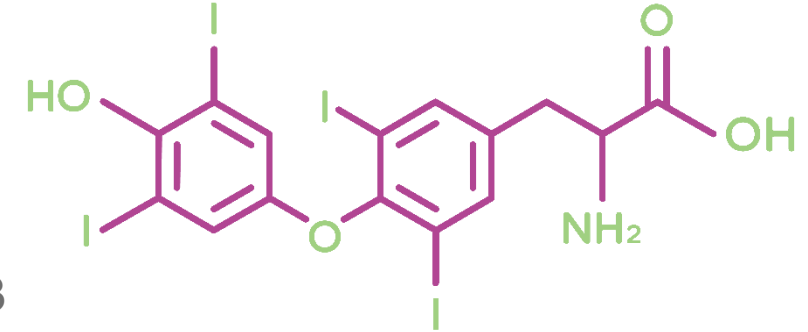
- T3 is a major regulator of **mitochondrial activity**
- **Mitochondria are present in the nuclei of all our cells**
- T3 increases mitochondrial activity by either increasing mitochondrial fuel delivery via transcriptions of genes like CPT1A and PDK4 and by increasing mitochondrial biogenesis via PPARGC1A
- T3 **increases oxidative phosphorylation** which generates reactive oxygen species that damage mitochondria, causing an induction of mitophagy. This mitophagy leads to autophagosome lysosomal degradation.





THYROXINE (T4)

- Requires **iodine** for synthesis (comprises 65% of T4's molecular weight)
- Found in 2 forms:
 - **Free T4:** travels to target tissues where it's converted to active T3
 - **Bound T4:** Accounts for >99%.
- T4 plays a crucial role in heart & digestive function, metabolism, bone health and brain development



Thyrotoxicosis

- May be caused by overactive thyroid gland, inflammation of the thyroid, or benign tumour.
- May be accompanied by goitre.

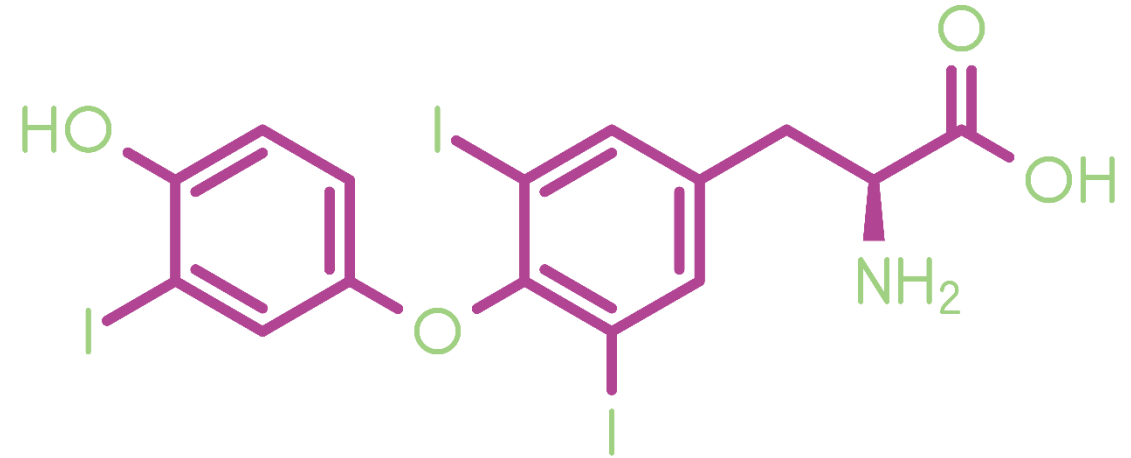


Hypothyroidism

- May be caused by autoimmune disease, poor iodine intake or medications.
- Causes reduced metabolism.

TRIIODOTHYRONINE (T3)

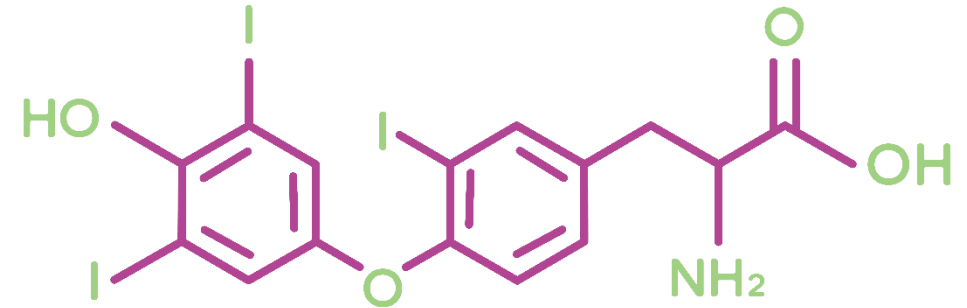
- **Active form** of thyroxine
- Requires **iodine** for synthesis (comprises 58% of T3's molecular weight)
- **90%** of TH that binds with receptors are T3
 - 10-20-fold greater affinity to thyroid receptors compared to T4
- Availability depends on **deiodinase activity**
- Major regulator of mitochondrial activity
- T3 helps to maintain muscle control, brain function and development, heart and digestive functions.





rT3

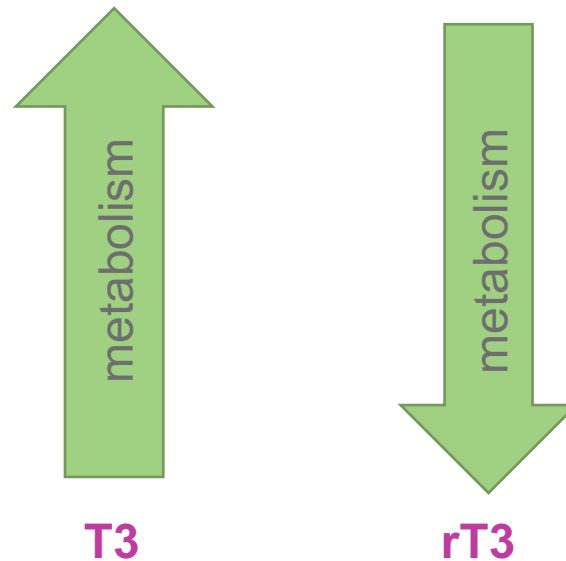
- **Metabolically inactive form** of thyroid hormone, generated from T4 via the type 3 5'-deiodinase enzyme.
- Serum concentration of rT3 is typically **10% of that of T3** but in some situations it may exceed that of T3.
 - The most common reason is in response to carbohydrate deprivation during severe illness or starvation.
- When this happens, rT3 increases due to the **inhibition of DIO1** which reduces the conversion of T4 to T3 and the deiodination of rT3. This will result in **low serum T3** and **high serum rT3**.
- Availability depends on **deiodinase activity**





HOW DO T3 AND rT3 DIFFER?

- **T3** binds to and activates the receptor, increasing metabolism
- **rT3** binds to but does not activate the receptor, decreasing metabolism as T3 access is limited



- rT3 levels are affected by genetic conditions that affect **iodothyronine deiodinases**, **thyroid transporters** and **transport proteins**.



THYROID HORMONE SYNTHESIS

Iodine from the blood is transported into the cell via the sodium iodine importer, then imported into the colloid.

Thyroid peroxidase (TPO) oxidises the iodine and then catalyses it binding to thyroglobulin.

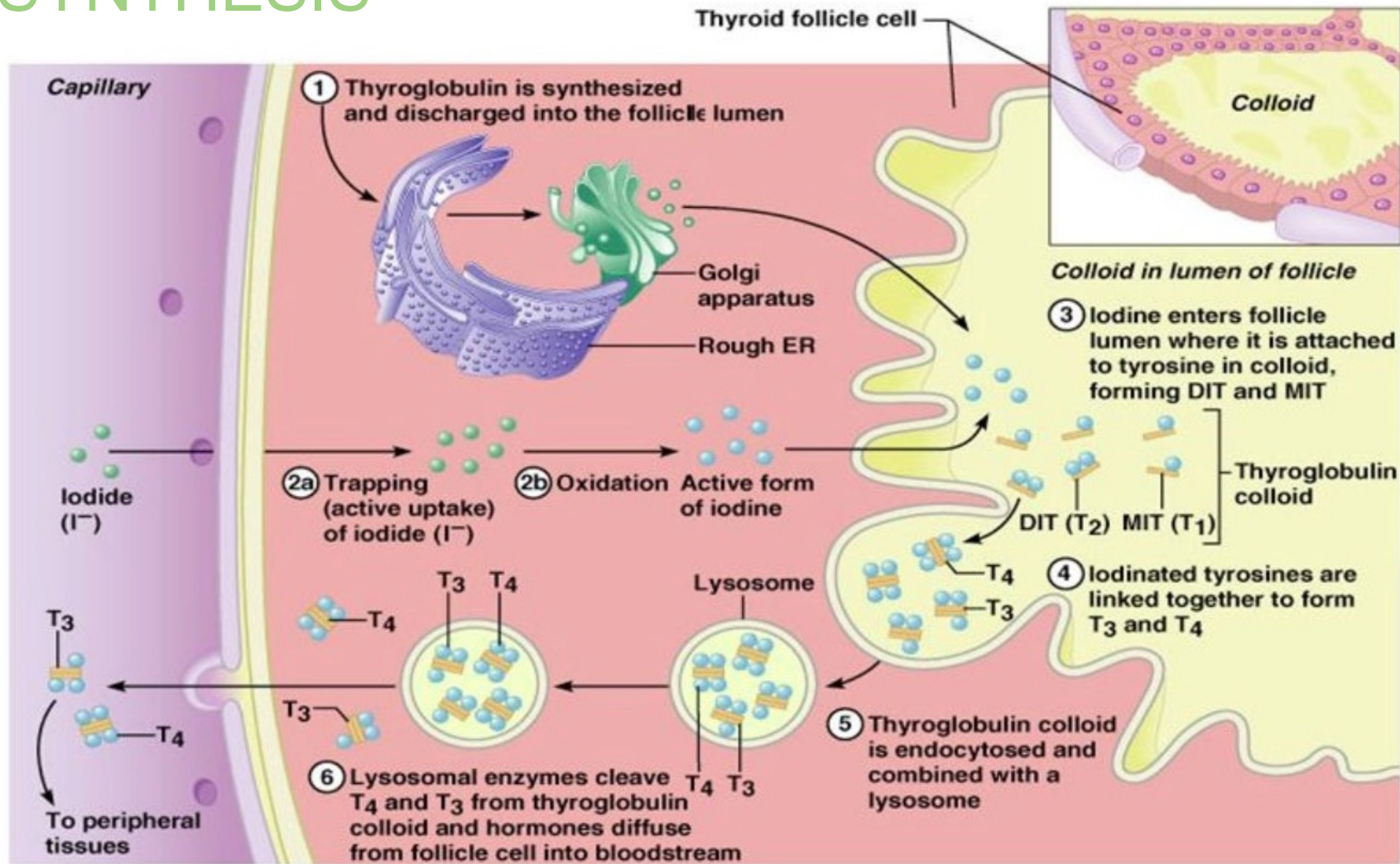
Iodine forms TH precursors, DIT and MIT, which couple together to form T4 and T3.

T3 and T4 are taken up by the cell, dissociated from the thyroglobulin molecule and released into the blood to travel to the target tissue.

T4 is converted to the active form, T3.



SYNTHESIS



Synthesis of Thyroid Hormone



MECHANISM

- The thyroid gland is responsible for the production of iodothyronines: T4, T3, rT3
- Iodothyronines are comprised of tyrosine and **iodine**
 - Thyroglobulin is formed from amino acids within the thyroid cells and is a large glycoprotein containing 140 molecules of the amino acid tyrosine
 - It is secreted into the follicular lumen where it is enzymatically combined with iodine
- Endosomes containing iodinated thyroglobulin fuse with lysosomes which enzymatically release the thyroglobulin from the resultant thyroid hormone.
- The thyroid hormones are released from the cell while the remaining **thyroglobulin is deiodinated** and recycled for further use.



THYROID BINDING GLOBULIN (TBG)

- TBG is synthesized in the **liver** and is a major transport protein that reversibly binds T4 and T3 and transports them into the bloodstream.
- Its primary function is to maintain **homeostasis** of thyroid hormones in blood serum.
- An increase in TBG may result in an increase in total T4 and T3 without an increase in hormone activity on the body.
- Increased TBG levels can be due to hypothyroidism, liver disease and pregnancy.
- A decrease in TBG causes an increase in FT4 and negatively inhibits TSH, reducing total T4 production.
- Decreased TBG levels can have many causes such as hyperthyroidism, kidney disease, liver disease, medication.
- All thyroid function tests should therefore be considered along with patient's symptoms
- The majority of T4 and T3 are **bound** to TBG
- Estrogen dominance increases the concentration of TBG by **2-3-fold**.

THYROID HORMONE METABOLISM

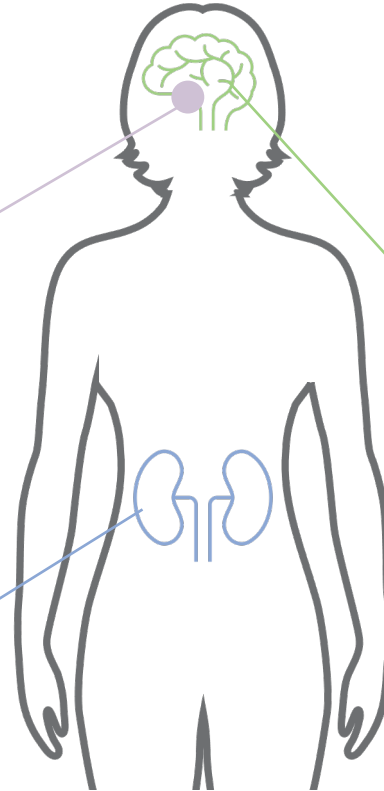
- Conversion from inactive T4 to active T3 requires 5-deiodinase production. There are 3 types of deiodinases:

Type II

- Produced in glial cells and found in the pituitary and hypothalamus.
- Activity enhances with lower T4 levels.
- Ensures there are always adequate T3 levels in the brain.

Type I

- Located in thyroid, liver and kidneys.
- Converts T4 in periphery to active T3



Type III

- Present in brain and placenta.
- Inactivates T4 by converting to rT3.
- Activity is enhanced where there are high levels of thyroid hormone.
- Protects brain and placenta from hyperthyroidism.

- There needs to be a balance between **active T3** and **inactive rT3**. This is maintained by deiodinases.
- T4 metabolism favors rT3 under certain stressors (pregnancy, caloric restriction, emotional stress and chronic illness)

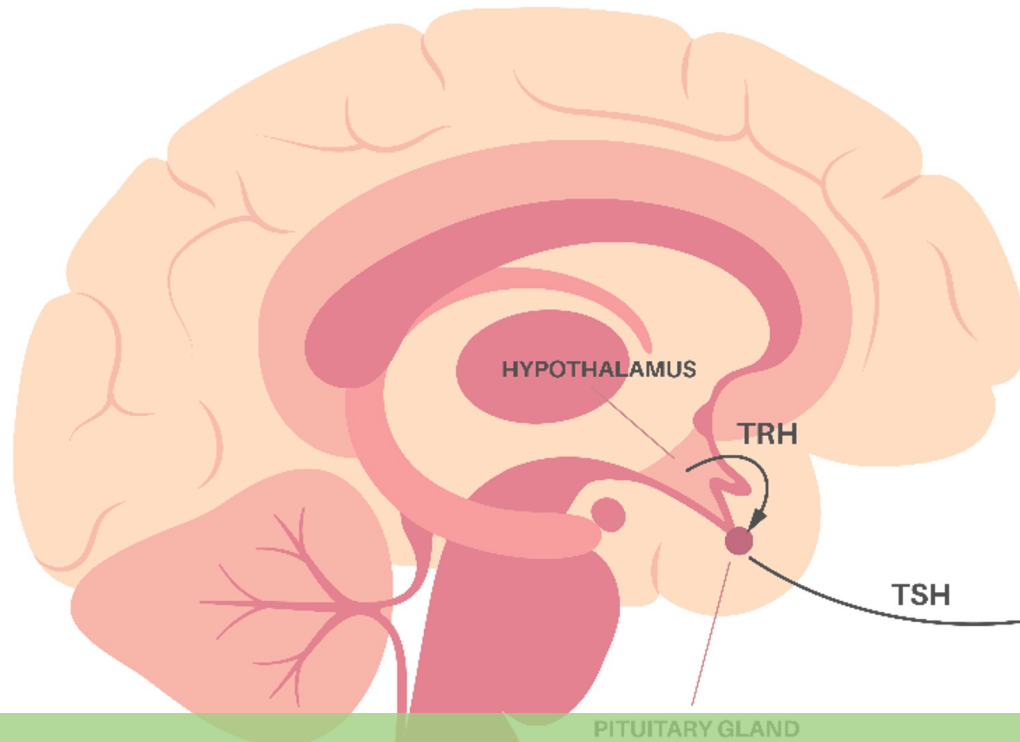
FACTORS THAT AFFECT 5-DEIODINASE PRODUCTION

- Selenium deficiency
- Stress
- Cadmium, mercury or lead
- Inadequate protein intake

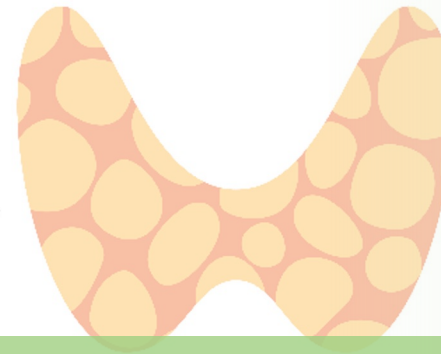


- High carbohydrate diet
- Elevated cortisol
- Chronic illness
- Decreased kidney or liver function

THYROID HORMONES



THYROID GLAND



WHAT INHIBITS T4 TO T3 CONVERSION?

WHAT COFACTORS ARE IMPORTANT FOR THYROID HORMONE SYNTHESIS?



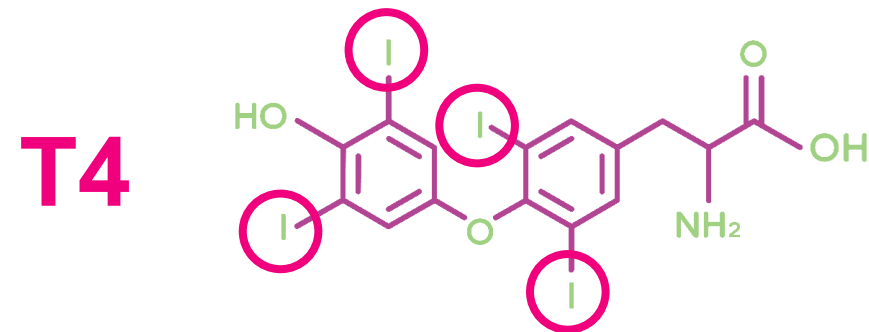
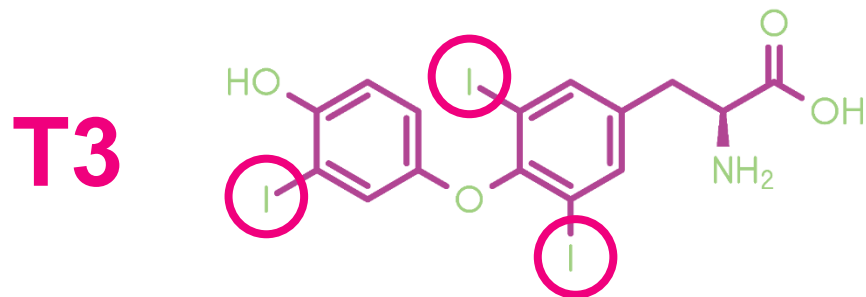
NUTRIENT DEFICIENCIES

- The first therapeutic intervention should be to **address the diet** and look at adding appropriate supplements, especially if the patient has borderline thyroid issues.
- Many co-factors for healthy thyroid production are missing in our **modern diet**
 - Without these building blocks thyroid dysfunction will occur.



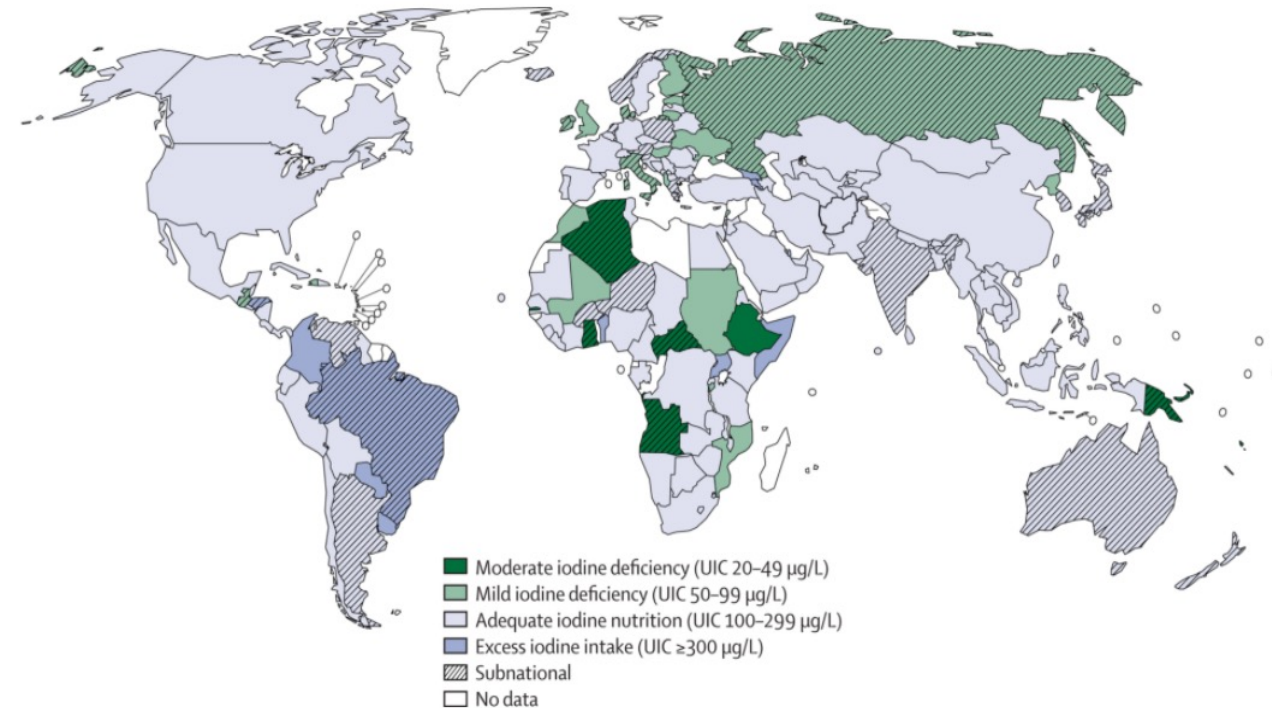
NUTRIENT DEFICIENCIES: IODINE

- Iodine is a **halogen** and has many important roles in the body
 - It competes with other toxic halogens for receptors and binding sites (bromine, chlorine, fluorine)
- It regulates the **activity of the thyroid gland**
- It is stored in particularly high concentrations in the thyroid gland
 - Large amounts are also stored in the salivary glands, cerebrospinal fluid, the brain, breasts and ovaries
- Both T3 and T4 require iodine for their production



NUTRIENT DEFICIENCIES: IODINE

- Iodine is an **essential** trace element found in the soil and water, found in several chemical forms. It is reduced to iodide in the gut and is nearly completely absorbed in the stomach and duodenum.
- The **thyroid** and **kidneys** primarily remove iodine from the circulation.
 - half life of approx. 10 hours (shortened in iodine deficiency or hyperthyroidism).
- The most common cause of hypothyroidism in world's population is **iodine deficiency**.
- Conversely, **excess iodine** can also cause hypothyroidism.



[https://www.thelancet.com/journals/landia/article/PIIS2213-8587\(14\)70225-6/fulltext#relatedClinic](https://www.thelancet.com/journals/landia/article/PIIS2213-8587(14)70225-6/fulltext#relatedClinic)



NUTRIENT DEFICIENCIES: IODINE

- Adults (above 12 years) need **150mcg** of iodine per day and pregnant and lactating women need **250mcg**
- A study of the iodine status of 14-15-year-old schoolgirls showed that the UK, along with Australia and the USA, is now **iodine deficient**.¹
 - Nearly 70% of the samples were below 100ug/L
 - 18% of samples showed very low iodine levels below 50µg/L.
- Mild iodine deficiency **impairs cognition** in children and severe iodine deficiency **reduces IQ** by 10 to 15 points.
- One explanation for this may be to do with the fact that we are not drinking so much milk.
 - One glass of milk contains approximately half the RDA of iodine.



1. Vanderpump M Lazarus J Smyth P Burns R Eggo M Han, T et al. Assessment of the UK iodine status: a national survey. Endocrine Abstracts. Presented at the Society for Endocrinology BES 2011: 11 April 2011-14 April 2011



NUTRIENT DEFICIENCIES: IODINE

“Young women of child-bearing age are the most susceptible to the adverse effects of iodine-deficiency and even mild deficiency may have an impact on the developing brain of fetuses and young children. It can also cause a goitre. According to the World Health Organisation, iodine-deficient communities have IQs up to 13.5 points lower than similar but iodine-sufficient communities.”

- The Society for Endocrinology



NUTRIENT DEFICIENCIES: IRON

- Normal thyroid metabolism depends on adequate supplies of iron, together with iodine, selenium and zinc.¹
- Low iron levels may result in **thyroid peroxidase becoming less active**, thus reducing the production of thyroid hormones.²
 - Thyroid metabolism, conversion and binding may also be modified, and the circulating of TSH may be increased.²
 - Ferritin deficiency decreases deiodinase activity; slows down conversion of T4 to T3, causing relative T3 deficiency at tissue level
- **Anaemia in hypothyroidism**
 - The low thyroid hormone levels of hypothyroidism **suppress the activity of bone marrow**, thereby lowering the production of red blood cells and triggering anemia.
 - Research shows as many as **43%** of people with symptomatic hypothyroidism have IDA. That's compared to 29% in the general population. **Persistent fatigue** is a main feature of the condition.³



IRON DEFICIENCY ALONE

- Symptoms of **anaemia** mimic those of **hypothyroidism**
 - For example, hair loss is a sign of hypothyroidism but it is also caused by low iron levels.¹
- All hypothyroid patients should be tested for iron and ferritin levels.
 - Ferritin levels should be between **50-70** for optimal T4 to T3 conversion
- Ferritin deficiency decreases deiodinase activity (slows down the conversion of T4 to T3)
- There is an association between iron deficiency and low T4 and low T3 levels in the general adult population, without any changes in TSH



1. Moeinvaziri M, Mansoori P, Holakooee K, Safaee Naraghi Z, Abbasi A. Iron status in diffuse telogen hair loss among women. Acta Dermatovenerol Croat. 2009;17(4):279-84. PMID: 20021982.

FERRITIN

Symptoms of low ferritin

- Hypothyroid like symptoms (low energy, hair loss, fatigue etc.)
- Increased hair loss or lack of hair growth
- Inability to tolerate exercise and reduced overall activity level
- Decreased immune function
- Symptoms of GI related issues (gas/ bloating, other nutrient deficiencies, low stomach acid etc.)

Causes of low ferritin

- Low iron diet
- Heavy periods
- Achlorhydria leading to malabsorption
- Prolonged PPI use





NUTRIENT DEFICIENCIES: SELENIUM

- Thyroid gland has the highest concentration of selenium in the body.
- Selenium is protective of thyroid and immune health and balances iodine levels
- Used as a building block for **selenoproteins**
 - **Deiodinases** are a subgroup of selenoproteins which convert T4 to T3
 - Low selenium levels cause low T3 production. TSH rises subsequently and the thyroid gland produces more T4
- Selenium can **reduce thyroid antibodies** by 40%
 - It is one of the most common deficiencies in people with **Hashimoto's** and widely recognized as a trigger of autoimmune thyroid conditions.
 - There is an association between the presence of an **anxiety or mood disorder** and the presence of TPO antibodies.¹

1. Carta MG, Loviselli A, Hardoy MC, et al. The link between thyroid autoimmunity (antithyroid peroxidase autoantibodies) with anxiety and mood disorders in the community: a field of interest for public health in the future. BMC Psychiatry. 2004;4:25.



NUTRIENT DEFICIENCIES: SELENIUM

- Taking 200µg of **sodium selenite** reduces TPO antibody level by 40% after 90 days and 55% after six months.
 - If selenium is stopped, antibody levels will go back up.
- Good sources of selenium include meat products, brazil nuts, fish, pasta, rice, bread, cereals and yeast.
- Populations at risk for selenium deficiency include:
 - Hashimoto's
 - Grain free, gluten free diets
 - Celiac disease, gluten sensitivities
 - Grave's disease
 - Pregnancy or post-partum thyroiditis
 - Thyroid eye disease



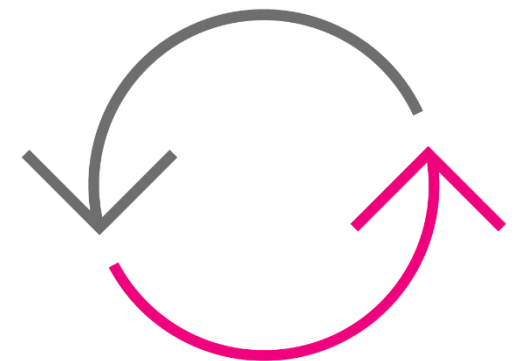
Adequate levels of both iodine and selenium are required for optimal thyroid hormone metabolism.

55 mcg of selenium daily RDA



NUTRIENT DEFICIENCIES: ZINC

- Low zinc levels **prevent the conversion** of T4 to T3.
 - Zinc is an essential cofactor of the enzyme type 1 **deiodinase**
- The **T3 receptor is thought to require zinc** to adopt its biologically active conformation.
 - Some of the effects of zinc deficiency, therefore, may be due to loss of zinc from the T3 receptor and impairment of T3 action.
- Zinc deficiency may result in **decreased thyroid hormone levels and resting metabolic rate (RMR)**.
- Zinc is needed to produce **TSH**
 - a deficiency can lead to hypothyroidism and hypothyroidism can lead to a zinc deficiency, as thyroid hormones are required for the absorption of zinc.
- Low zinc levels are associated with increased levels of **autoimmunity**.





FACTORS AFFECTING ZINC LEVELS



VITAMIN DEFICIENCIES

Deficiency in vitamins A, B2, B6 and B12



MEDICATIONS

Beta blockers, birth control, oestrogen, lithium, phenytoin, theophylline, chemotherapy



DIET

Too many cruciferous vegetables, low protein, low fat, low carbohydrate, excessive alcohol, soy



OTHER

Ageing, excess alpha-lipoic acid, diabetes, fluoride, lead, mercury, pesticides, radiation, stress, copper excess, calcium excess, dioxins, PCB, inadequate DHEA or cortisol production, phthalates



NUTRIENT DEFICIENCIES: COPPER

- Stimulates the production of T4 and prevents overabsorption of T4 in the blood cells, by controlling the body's calcium levels.
- Stimulates the production of phospholipids needed for TSH stimulation
- Blood copper levels are shown to be positively correlated with FT4
- In a study, hypothyroid patients were found to have significantly decreased levels of zinc and copper, which indicates abnormal metabolism of zinc and copper in hypothyroidism and may exaggerate the complications of hypothyroidism



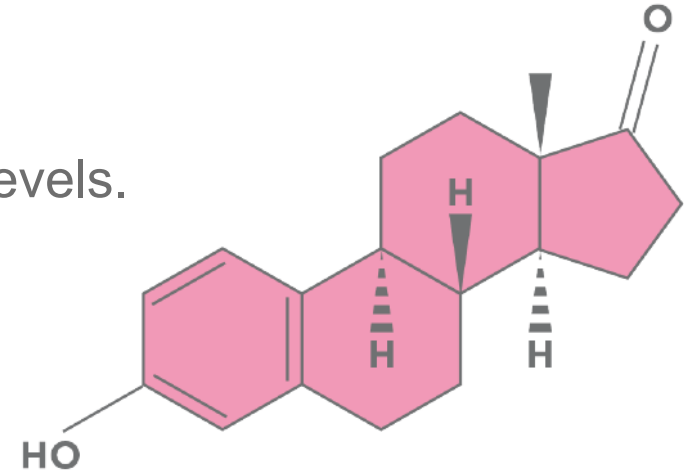
THE SYNERGY OF THYROID HORMONES WITH OTHER ENDOCRINE SYSTEMS

HO



OESTROGEN

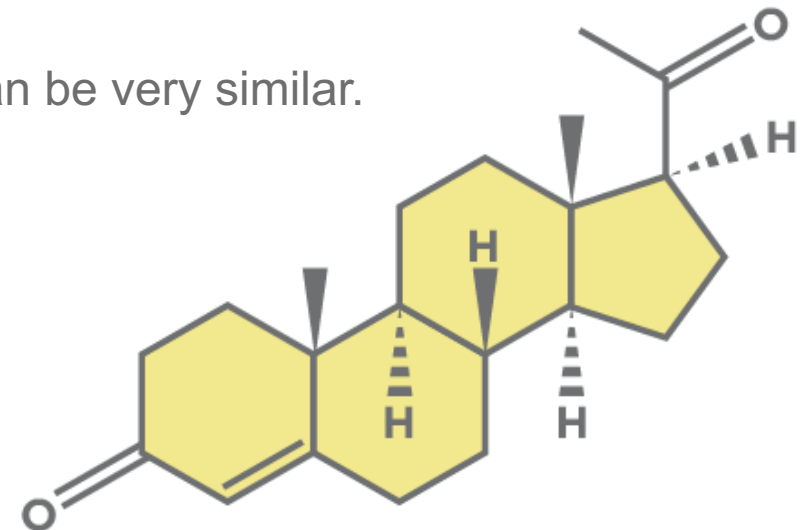
- Thyroid function is sensitive to fluctuations in sex hormone levels.
- Oestrogen stimulates the **production of thyroglobulin** and **increases thyroid binding globulin**.
- Changes in oestrogen levels (e.g. during perimenopause) means thyroid hormones will fluctuate.
 - This can affect metabolism, muscle strength, energy production, heart function and temperature regulation.
- **Thyroid diseases are more prevalent in women** particularly between puberty and menopause and women are more susceptible to the goitrogenic effect of iodine deficiency.
- Carcinomas of the thyroid are **3x more frequent in women** than in men, and the peak rates occur earlier in women.





PROGESTERONE

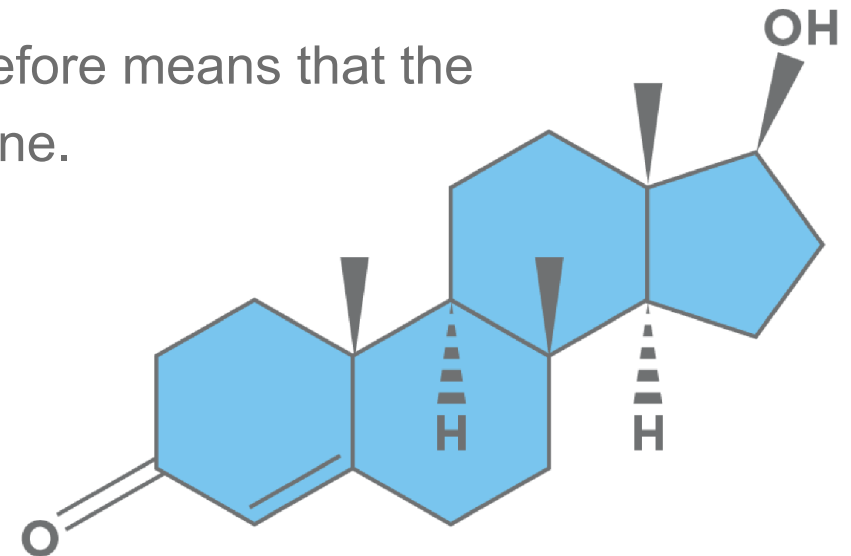
- Progesterone **decreases thyroid binding globulin** and **increases the activity of thyroid hormones**.
- Thyroid hormones, like progesterone, **accelerate metabolism** and **utilise fat** for energy production.
- *The Journal of Endocrinology* featured a study showing the thyroid hormone **T3 significantly stimulated the release of progesterone** from luteal cells.
- Research published in *Clinical Endocrinology* concluded that progesterone therapy increases T4.
- Symptoms of **low progesterone** and **reduced thyroid** activity can be very similar.





TESTOSTERONE

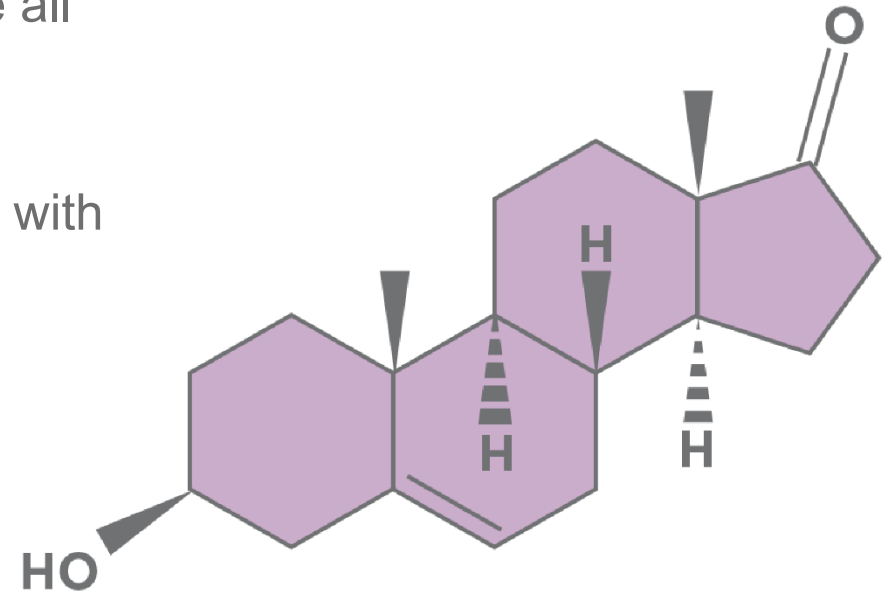
- Adequate production of thyroid hormone stimulates the hypothalamus, resulting in production of **GnRH (gonadotropin-releasing hormone)**.
- GnRH then signals the pituitary gland to produce **LH (luteinising hormone)**.
- LH signals the ovaries/testes to produce & release testosterone.
- Poor production of thyroid hormone (hypothyroidism) therefore means that the gonads will not be signalled to produce enough testosterone.





DHEA

- It is common for people with **Addison's disease** to have hypothyroidism and because DHEA is produced by the adrenals, problems with the adrenals can affect its production and the levels present in the blood.
- Thyroid conditions can impact other hormone levels in the body, which can in turn also affect DHEA levels.
- One study showed that **DHEA, DHEA-S and PREG-S** were all significantly lower in patients with hypothyroidism.
- Interestingly, although DHEA levels were normal in patients with hyperthyroidism, their DHEA-S and PREG-S levels were **significantly higher**.¹

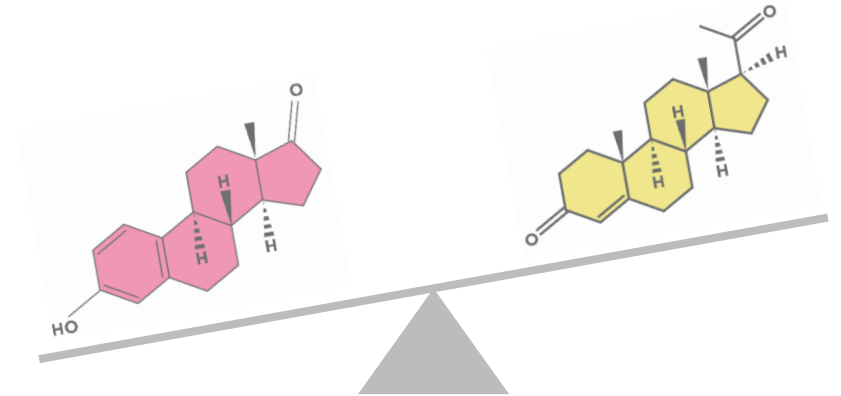


1. Serum Dehydroepiandrosterone, Dehydroepiandrosterone Sulfate, and Pregnenolone Sulfate Concentrations in Patients with Hyperthyroidism and Hypothyroidism Noriko Tagawa et al American Association for Clinical Chemistry <http://clinchem.aaccjnl.org/content/46/4/523>

OESTROGEN DOMINANCE AND HYPOTHYROIDISM

Oestrogen dominance impacts on thyroid health by impacting **conversion of T4 to T3**.

- When oestrogen is high and progesterone is low the liver **increases production of TBG**.
- This is released into the blood and **binds free T3 and T4** before it can reach the cells and exert its effects.
- **Thyroid panels may appear normal**, but the hormones are unavailable for the body to use, eventually causing symptoms of hypothyroidism.



These symptoms often manifest during **perimenopause** when progesterone levels are significantly decreased but oestrogen is still present leading to relative oestrogen dominance.



TREATING OESTROGEN DOMINANCE

- Many practitioners suggest **supplemental progesterone** for women with oestrogen dominance
 - Postmenopausal women who supplemented with 300 mg/day of micronised progesterone had increased levels of free T4.¹
 - Simply adding one hormone to balance another doesn't usually address the root cause of the imbalance.
- Herbs such as **cohosh**, **ashwaganda** and **chasteberry** can help to re-balance oestrogen, testosterone and progesterone
- **DIM** supports healthy oestrogen levels by promoting its conversion to 2-hydroxy oestrogens and reducing the unwanted 16-hydroxy and 4-hydroxy oestrogens.
- **Calcium D-glucarate** supports the glucuronidation pathway to excrete oestrogen through the bile/ urine.
- Together with **high-quality multivitamin** and **lifestyle changes**, the production of TBG is decreased
 - Allows the thyroid hormones to enter cells, and thyroid symptoms often subside.



1. Sathi P, Kalyan S, Hitchcock CL, Pudek M, Prior JC. Progesterone therapy increases free thyroxine levels--data from a randomized placebo-controlled 12-week hot flush trial. Clin Endocrinol (Oxf). 2013 Aug;79(2):282-7

HOW TO INCREASE CONVERSION OF T4 TO T3

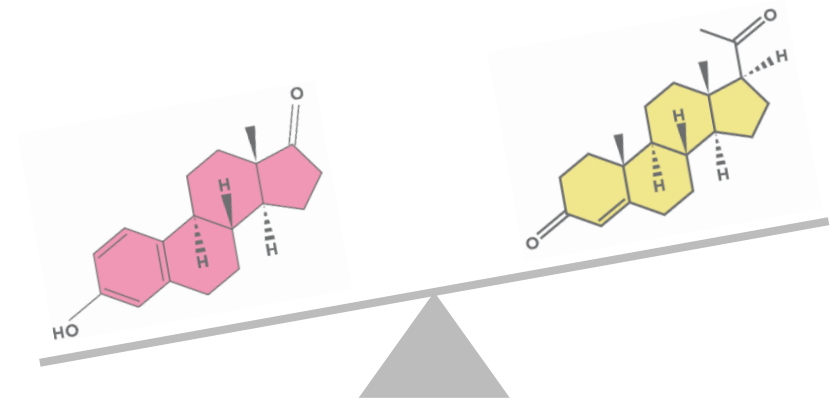
- Increase **selenium** in diet
- Supplement with 200mcg selenium daily for 3 months, then retest level
- Increase **potassium** in diet
- **Iron**; correct deficiency
- **Zinc** (chelated), 10 mg daily (body can only absorb 7 -10mg of zinc at any given time)
- High **protein** diet (1.2- 1.6 g per kg)
- **Ashwagandha**- 600mg daily
- Vitamins **A, B2 & E**



Maares, M., & Haase, H. (2020). A Guide to Human Zinc Absorption: General Overview and Recent Advances of In Vitro Intestinal Models. *Nutrients*, 12(3), 762. <https://doi.org/10.3390/nu12030762>

HOW TO INCREASE CONVERSION OF T4 TO T3

- Restore sex hormone balance
- Adequate tyrosine levels (can be low in vegans, vegetarians & body builders) as this is an essential amino acid in thyroglobulin synthesis
- Nutri Advanced T convert (contains gum guggal)- guggulsterones in this herb stimulate T3 production as well as other essential cofactors
- Selenium, iron, copper, zinc, magnesium, B vitamins are essential cofactors and should be included in the diet and/or supplemented



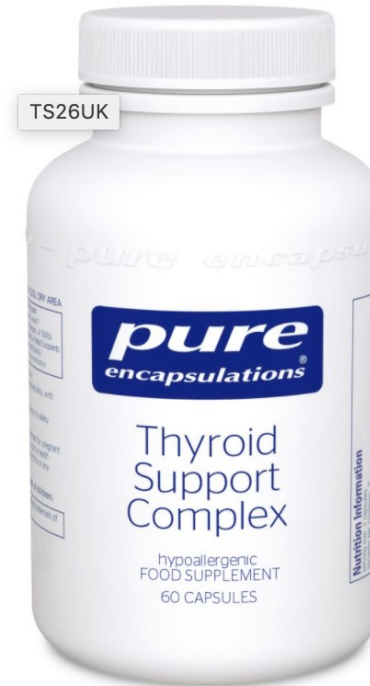
RECOMMENDED THYROID SUPPLEMENTS



Ingredients:

Magnesium 25mg, 7%*
Vitamin C 250mg, 313%
L-Tyrosine 250mg -
Calcium 40mg 5%
Vitamin E 67mg 558%
Iodine 75µg, 50%
Zinc 4mg, 40%
Vit B6 12.5mg 893%
Selenium 25µg -
Liquorice root 10mg -
Vit B3 12.5mg, 78%
Vit B1 5mg, 455%
Vit B2 5mg, 357%
Copper 0.5mg, 37.5%
Vit A 750µg, 94%
Folate 75µg, 37.5%

*% NRV



Ingredients:

Vitamin A 750 µg, 94%*
Vitamin C 150mg, 188%
Vitamin D3 5µg, 100%
Iodine 150µg, 100%
Zinc 20mg, 200%
Selenium 200µg 364%
L-Tyrosine 500mg
Ashwagandha 400mg
Forskohlii 100mg
Guggul 50mg
Turmeric 100mg

* % NRV



THYROID SUPPORT CONTINUED

T-Convert	Synergistic blend of mukul myrrh and minerals to support the health of the thyroid gland. Gum guggul, selenium, magnesium, zinc, copper and magnesium have all been shown to support the conversion of T4 to T3.
L-tyrosine	L-tyrosine L-tyrosine is an amino acid that can be converted in the brain and in the adrenal glands to dopamine, norepinephrine, and epinephrine. These hormones may be depleted by stress, overwork and certain drugs. By replenishing norepinephrine in the brain, mental energy levels may be improved. Iodine and tyrosine are required for thyroid hormone production.
Vitamin B12	Hypothyroid patients are often vitamin B12 deficient. It is not clear what the link between B12 deficiency and low thyroid function is, nor if thyroid function will improve with B12 supplementation, but, since low B12 can causes serious neurologic damage, all hypothyroid patients should be tested.
Nutrition and Lifestyle	Some foods contain goitrogenic substances that reduce the utilisation of iodine. Raw goitrogenic foods (such as canola oil and vegetables from the Brassica family) and soy foods that have not undergone fermentation and/ or food processing should be consumed in moderation and discontinued if symptoms should appear. Isoflavone molecules in soy can inhibit an enzyme involved in thyroid hormone synthesis so consumption of soy should be reduced.



THYROID DYSFUNCTION



DECREASED THYROID ACTIVITY

HYPOTHYROIDISM

Clinical condition characterized by a reduction in T4 and T3 levels

- **Primary failure** – failure of production within the thyroid gland
- **Secondary failure** – caused by other influences on the thyroid

Causes:

- Hashimoto's disease
- Iodine/ tyrosine/ iron/ selenium deficiency
- Oestrogen dominance
- Genetics - DAO snp + many others
- Post partum, thyroiditis
- Radiation to neck area
- Radioactive iodine treatment
- Medications – e.g. interferon alpha. Lithium, amiodarone
- Thyroid surgery/parathyroid surgery
- Hypopituitarism
- Disorders of hypothalamus
- Ageing

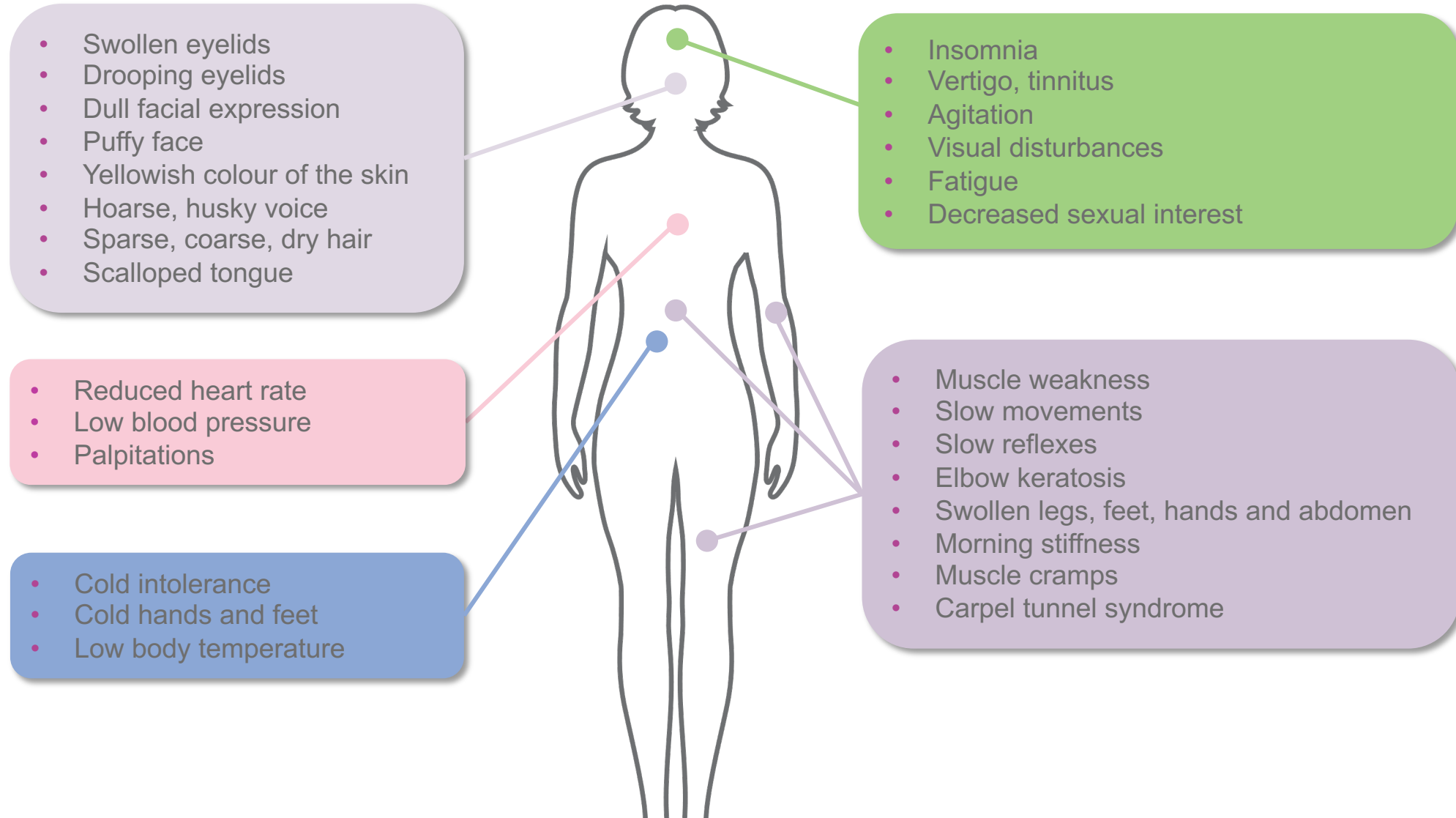
HYPOTHYROIDISM

Before and after
thyroid treatment..



Fig 7.3. Luiz, H.V., Manita, I., Portugal, J. (2016). Hypothyroidism. In: Imam, S., Ahmad, S. (eds) Thyroid Disorders. Springer, Cham. https://doi.org/10.1007/978-3-319-25871-3_7

SYMPTOMS AND SIGNS OF LOW THYROID PRODUCTION



RISK FACTORS

- Being female
- Age 60 or above
- Family history of thyroid disease
- Family history of autoimmune disease or having an autoimmune disease
- Caucasian or Asian ethnicity



PREVELANCE:

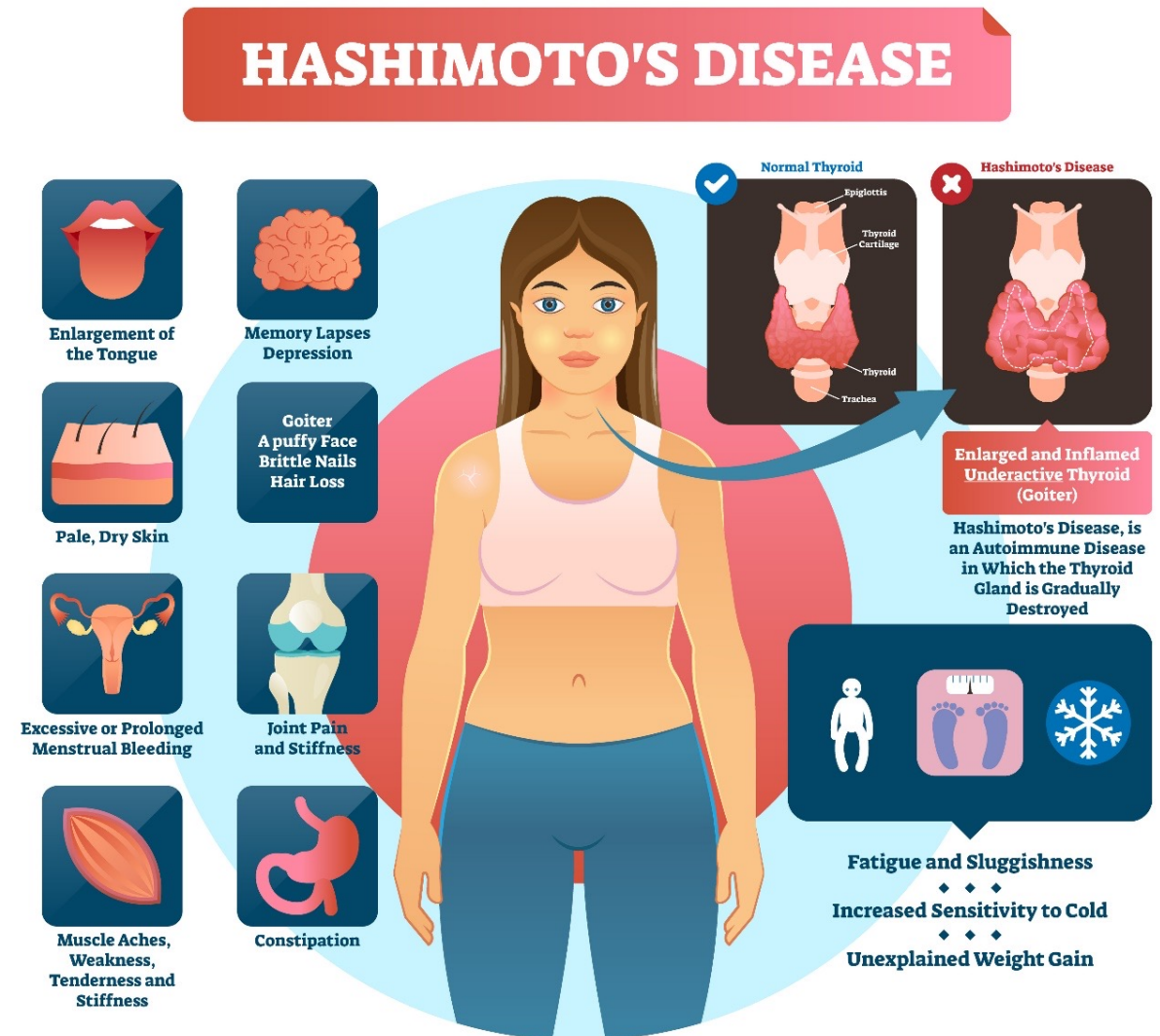
- In UK, official figures stand at **2% of the population** and in **more than 5% of those over 60**.
- 5 - 10 x more common in women

LONG TERM CONSEQUENCES:

- Cardiovascular disease
- Obesity
- Joint pain
- Infertility

HASHIMOTOS

- This occurs when patients develop antibodies to thyroid antigens, most commonly **anti-TPO**
 - Many also form anti-Tg and TSH receptor-blocking antibodies
- These antibodies attack the thyroid tissue, leading to **inadequate production of thyroid hormones** and the **gradual loss of function** of the thyroid gland.
- **Women** more commonly affected (10:1) and tend to be diagnosed between **30-50** years of age.
- Early symptoms include **constipation**, **fatigue**, **dry skin** and **weight gain**.
- In the early stages, condition can relapse and remit





HASHIMOTOS THYROIDITIS

The thyroid gland usually enlarges but not always. It may also start with an over-active phase before becoming underactive.

Thyroid peroxidase antibodies (TPOAb) is an important biomarker in diagnosis of Hashimoto's. Lymphocytes produce antibodies against TPOAb which elevates the levels.

WHAT'S THE DIFFERENCE BETWEEN HYPOTHYROIDISM & HASHIMOTO'S DISEASE?

Hypothyroidism is a clinical state of low levels of thyroid hormone in the body, which can occur as a result of a variety of different reasons, such as iodine deficiency or surgical removal of the thyroid.

Hashimoto's disease causes the immune system to form antibodies against the thyroid gland, resulting in the immune system attacking the thyroid gland, eventually leading to hypothyroidism, and is the cause of 90% of cases of hypothyroidism in the US.

HYPERTHYROIDISM

- Hyperthyroidism occurs when the thyroid gland produces too much thyroid hormone.
- Thyrotoxicosis, especially hyperthyroidism, may be recognized by a **goitre**.
- About 20x more common in women than men.

CAUSES

- Main causes of primary hyperthyroidism are Graves' disease, toxic multinodular goitre and toxic adenoma



SYMPTOMS & SIGNS OF HYPERTHYROIDISM

- Other symptoms include:
 - Heat intolerance
 - Weight loss
 - Increased appetite
 - Increased bowel movements
 - Irregular menstrual cycle
 - Rapid/irregular heartbeat
 - Palpitations
 - Tiredness
 - Irritability
 - Tremor
 - Hair thinning/loss
 - Retraction of the eyelids

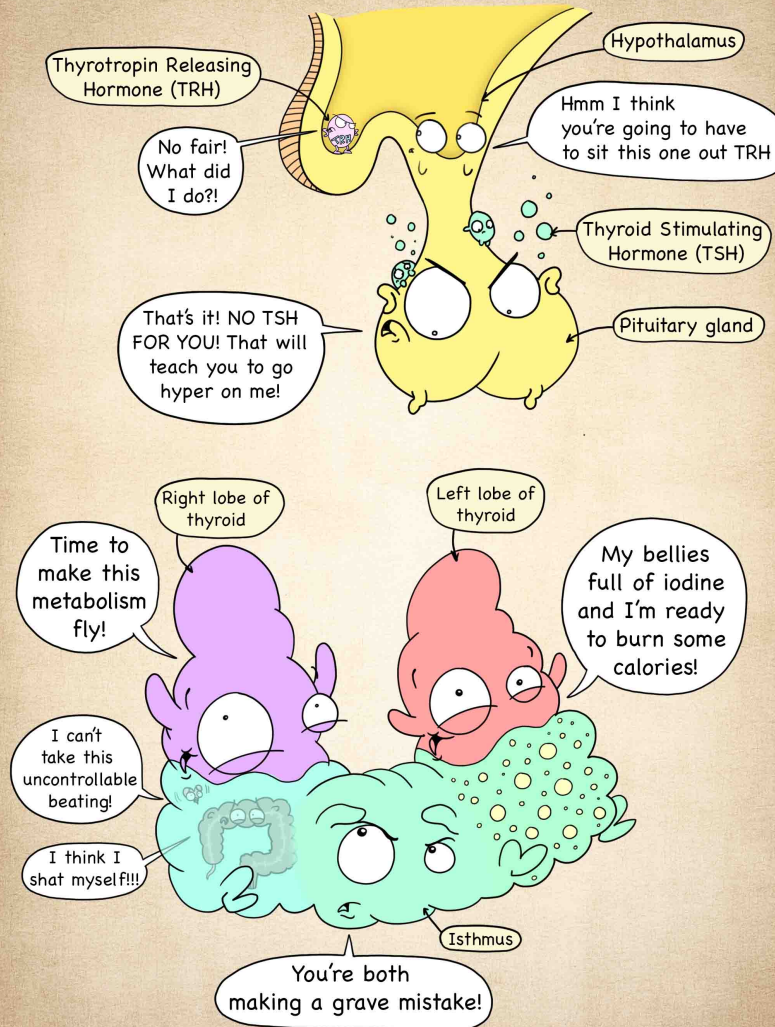




RISK FACTORS

- Smoking increases risk of both Graves' disease and toxic nodular goitre.
- In areas of **high** iodine intake, Graves' disease is the major cause. In areas of **low** iodine intake, the major cause is nodular goitre.
- **PREVELANCE:**
- In UK official figures stand at 2% in UK women and 0.2% in men.
- Autoimmune hyperthyroidism (Graves' disease) is the most common form in the UK, accounting for 60 to 80% of cases.
- 5- 10 x more common in women
- **LONG TERM CONSEQUENCES:**
- Atrial fibrillation, leading to increased risk of stroke, congestive heart failure, osteoporosis

HYPERTHYROIDISM



(c) The Comical Anatomist 2019

- ☐ Insulin
- ☐ Parathyroid
- ☒ T3
- ☒ T4

DIAGNOSTICS

- ☒ Free T3
- ☒ Free T4



THYROID TESTING



- Most clinicians rely on serum (TSH) and serum-free T4.
- TSH is responsible for stimulating the thyroid to produce more iodothyronines.
 - Levels inversely correlate with active thyroid hormone concentrations; as T3 increases, TSH decreases.
- Free levels of thyroxine are measured in the serum rather than total T4 levels, which would include protein-bound T4.
- Free T4 can be a proxy for serum T3 levels.
- Most often, thyroxine levels are the last to become abnormal in thyroid disorders as the upstream products, TSH and T4, maintain available T3 at their own expense.¹

¹ Wang D et al. Reference intervals for thyroid-stimulating hormone, free thyroxine, and free triiodothyronine in elderly Chinese persons. Clin Chem Lab Med. 2019 Jun 26;57(7):1044-1052.



SUBCLINICAL HYPOTHYROIDISM: THE CONTROVERSY AND TESTING CHALLENGES

- Experts continue to make differing recommendations for the treatment of subclinical hypothyroidism
- In subclinical hypothyroidism, **TSH levels are elevated**, while **circulating thyroid hormone levels are normal**.
- Some reports indicate that 90% of patients with subclinical hypothyroidism have TSH levels between **4 and 10 mIU/L**
 - Other reports define the thyroid condition based on a TSH screening level over **4.5 mIU/L**.
- The upper limit of the TSH range for diagnosis and levothyroxine treatment is debated, as well as the effectiveness of conventional thyroid hormone therapy in mild hypothyroidism. In functional medicine, we aim to keep the TSH < 2
- Treat the patient and the symptoms- aim for quality of life



BLOOD TEST RANGES

Test	From	To	Units	Notes
TSH	0.4	4.0	mU/l	<ul style="list-style-type: none"> • If not on thyroid treatment, TSH should be below 2 • Check levels as often as needed (usually every 6 weeks) and keep adjusting doses as needed. Try to keep TSH between 1 and 2 • Important to monitor symptoms not just blood test results. Some people feel better with a higher or lower TSH
Free T4	9.0	25.0	pmol/l	<ul style="list-style-type: none"> • High TSH and low FT4 and low FT3 suggests an underactive thyroid (hypothyroidism) • Low TSH and high FT4 and high FT3 suggests an overactive thyroid (hyperthyroidism) • If the TSH level is slightly raised but the FT4 and FT3 level is still within the normal reference range this is called subclinical hypothyroidism or mild thyroid failure. • A low TSH with a low FT4 may be a result of a failure of the pituitary gland (secondary hypothyroidism caused by hypopituitarism) or a response to any significant illness.
Free T3	3.5	7.8		<ul style="list-style-type: none"> • As above

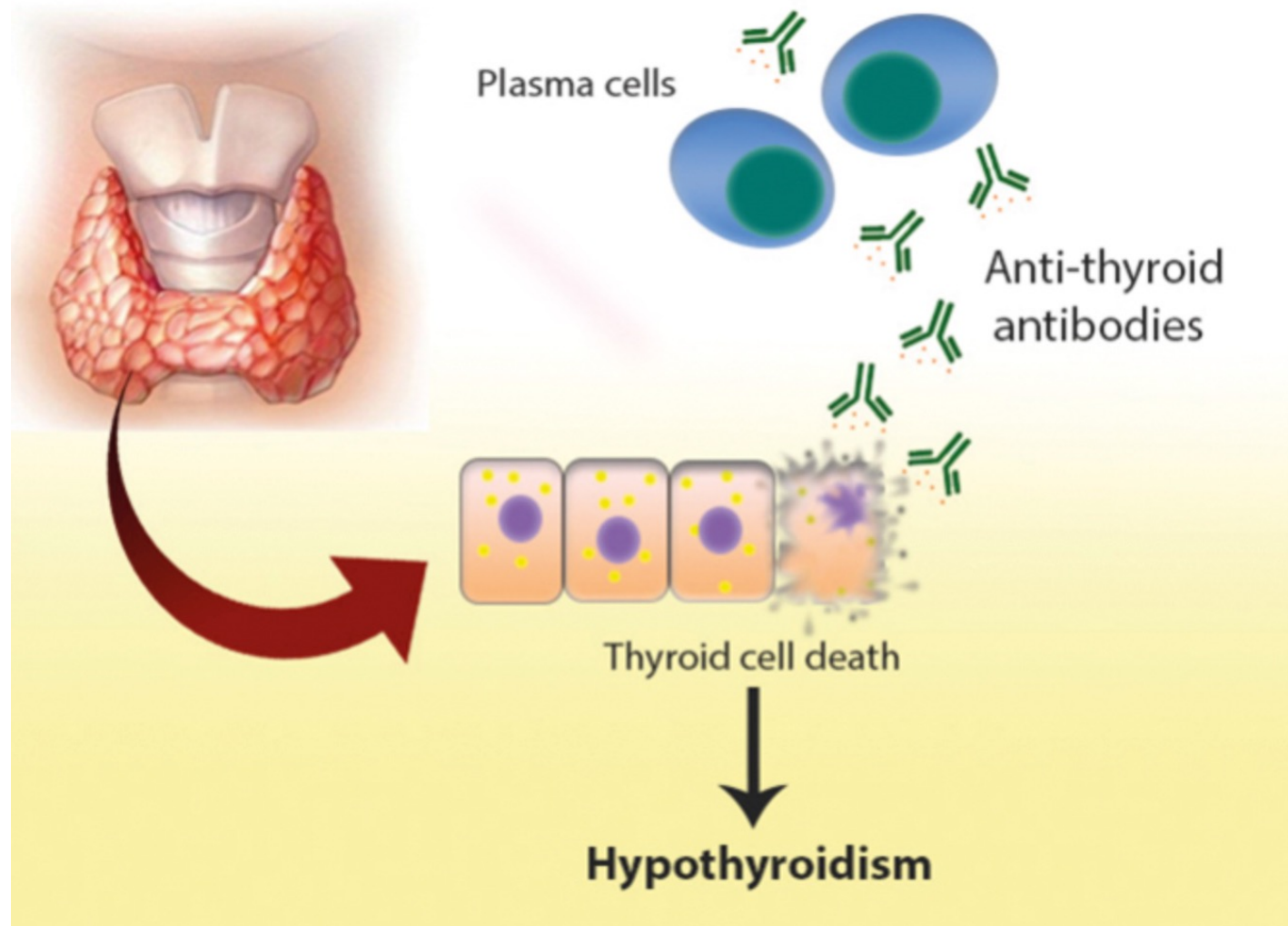


THYROID ANTIBODIES

- Antibody tests are used to **confirm the diagnosis of autoimmune thyroid diseases**.
 - Some people will test positive for more than one type of thyroid antibody.
- In people with **subclinical thyroid disease** the presence of antibodies can indicate that a person may go on to develop full-blown thyroid disease in the future, but that treatment is not yet required.
- **Positive antibodies** can also be present in people without thyroid disease (anemia, RA or diabetes)

Antibodies	What do they indicate?
Thyroid peroxidase antibodies (TPOAb)	Raised in Hashimoto's thyroiditis (or autoimmune thyroiditis) and sometimes raised in Grave's disease.
Thyroglobulin antibodies (Tg Ab)	Sometimes raised in Hashimoto's thyroiditis
Thyroid stimulating hormone receptor antibodies (TSHR Ab, also known as TRAb)	Raised in Grave's disease

THYROID ANTIBODIES



<https://www.entandaudiologynews.com/features/audiology-features/post/thyroid-dysfunction-and-meniere-s-disease-is-there-a-relationship>



REVERSE rT3

- Elevated rT3 is indicative of reduced thyroid transport, due to **mitochondrial dysfunction**.
 - Any disease associated with mitochondrial dysfunction may display elevated rT3.
- Conditions associated with elevated T3:
 - Insulin resistance, diabetes mellitus, obesity, chronic and acute dieting, anxiety and depression, ageing, CFS and fibromyalgia, cardiovascular disease, inflammation and chronic illness, hypercholesterolemia and hypertriglyceridemia
- Excess rT3 will **further inhibit conversion** from T4 to T3.
- FT3 and rT3 **occupy the same receptor sites**.
 - T3 will activate the receptor, rT3 will not.
- If rT3 is high, the patient will have **symptoms of hypothyroidism**, even if labs are normal.



LIFECODE GX- NUTRIGENOMICS- how nutrients affect epigenetic expression of our genes

- The Thyroid Balance report analyses the genes involved in the thyroid hormone lifecycle: synthesis - centrally (in the thyroid) and in activation in peripheral tissues, transport and metabolism, processing of cofactors (vitamins D and A) and inhibitors (stress and toxins).

It also examines genes that confer susceptibility to inflammation and autoimmunity.

Individuals have variations on genes called SNPs or SINGLE NUCLEOTIDE POLYMORPHISMS which affect the TRANSCRIPTION of the genes coding for various enzymes involved in the thyroid hormone cycle



LIFECODE GX



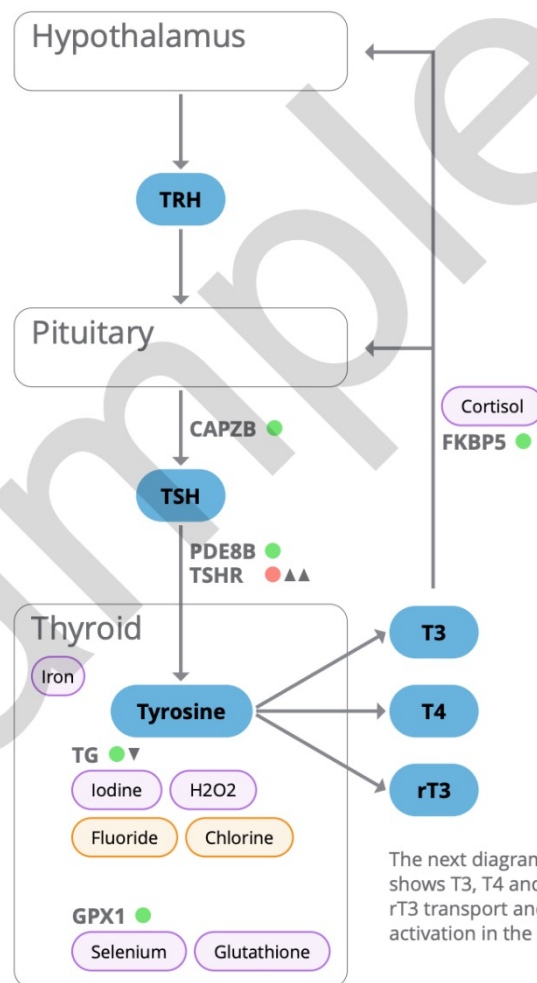
HPT Axis Diagram

Autoimmune

CTLA4 ●●●
 FOXE1 ●▲▲
 PTPN22 ●▼▼
 HLA-DQA1 ●
 HLA-DQB1 ●

Inflammation

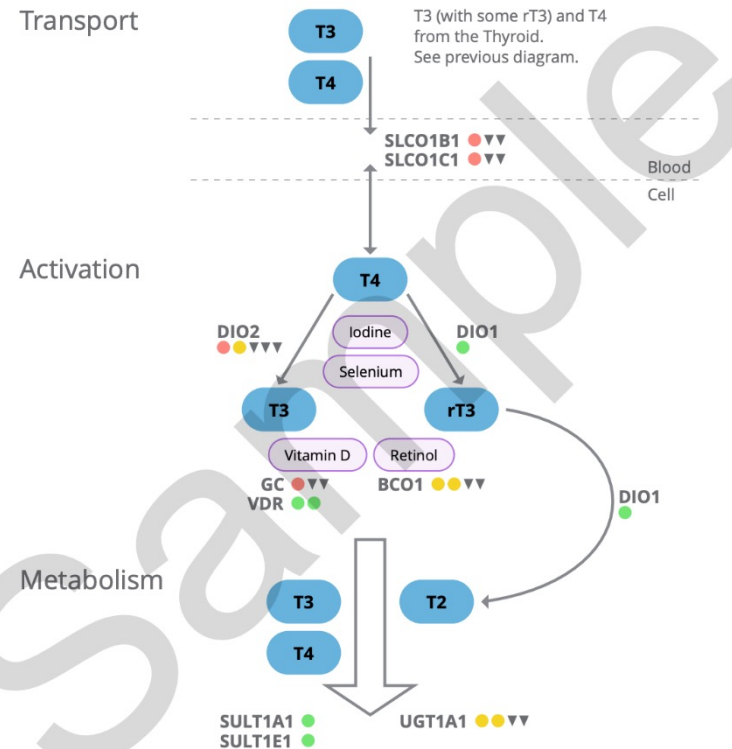
IL6 ●▼▼
 TNF ●
 GC ●▼▼
 VDR ●●
 CD40 ●▲▲
 FCRL3 ●



LIFECODE GX



Transport and Activation Diagram





NORDIC LABS

Thyroid Plus -hormone test that provides a thorough analysis of thyroid hormone metabolism. It includes central thyroid gland regulation and activity, thyroid production and secretion, peripheral thyroid conversion, and thyroid autoimmunity

Indications include

Anxiety/panic attacks

Coldness

Constipation

Decreased memory and concentration

Dysmenorrhea

Fluid retention

Headaches

PMS

Poor skin

Weight gain

PATIENT: Sample Report
 TEST NUMBER: TN123456
 PATIENT NUMBER: PN123456
 GENDER: Male
 AGE: XX
 DATE OF BIRTH: dd-mm-yyyy

COLLECTED: dd-mm-yyyy
 RECEIVED: dd-mm-yyyy
 TESTED: dd-mm-yyyy

TEST REF: TST-XXXXXX
 PRACTITIONER: Nordic Laboratories
 ADDRESS:

TEST NAME: Thyroid Plus

Thyroid Plus

Sample Type - Serum	Result	Reference Range	Units
Central Thyroid Regulation & Activity			
Total Thyroxine (T4)	<13 L	<dl	58-161 nmol/L
Thyroid Stimulating Hormone (TSH)	5.32 H	5.32	0.40-4.00 microIU/mL
Free Thyroxine (FT4)	10.3 L	10.3	11.5-22.7 pmol/L
Peripheral Thyroid Function			
Free T3	5.8	5.8	2.8-6.5 pmol/L
FT4 : FT3 Ratio	1.8		
Reverse T3 (rT3)	0.30	0.30	0.14-0.54 nmol/L
Thyroid Auto Immunity			
Thyroglobulin (TG)	<32	<dl	<= 40 IU/mL
Peroxidase (TPO)	87 H	87	<= 34 IU/mL
Key Guide			
<div>Within Reference Range</div> <div>Outside Reference Range</div>			



THYROID IMAGING

- Ultrasound scan can be used to evaluate **lumps** or **nodules** and whether they are attached to the mass is connected to the thyroid gland or another part of the body.
- It **cannot** be used to detect cancerous lumps or to diagnose underactive or overactive thyroid disorders.
- Takes about 30 mins to complete and is non-invasive.
- Cost approx. £135





SOME IMPORTANT POINTS

- **Blood tests** are currently the most accurate way to diagnose and manage thyroid disorders.
- **Symptoms and how the patient feels** are an important part of the diagnosis.
- It is important that the **TSH level** is within the optimum reference range (below 2)
- For those taking medication for a thyroid disorder, there is scope to **fine-tune** treatment. Consider doing a more comprehensive panel. Look for a root cause as it may be reversible.
- Those with a diagnosed thyroid disorder or have had previous treatment for an overactive thyroid should have a blood test **every 12 months or as appropriate if treatment has been changed.**
- Those with a thyroid disorder should have a blood test in **early pregnancy** or if they are **planning a pregnancy.**
- Patients should not alter medication doses without consulting a doctor.
- Thyroid disease is often hereditary and it is important to be aware of family history of thyroid disorders



TREATMENT/ PRESCRIBING

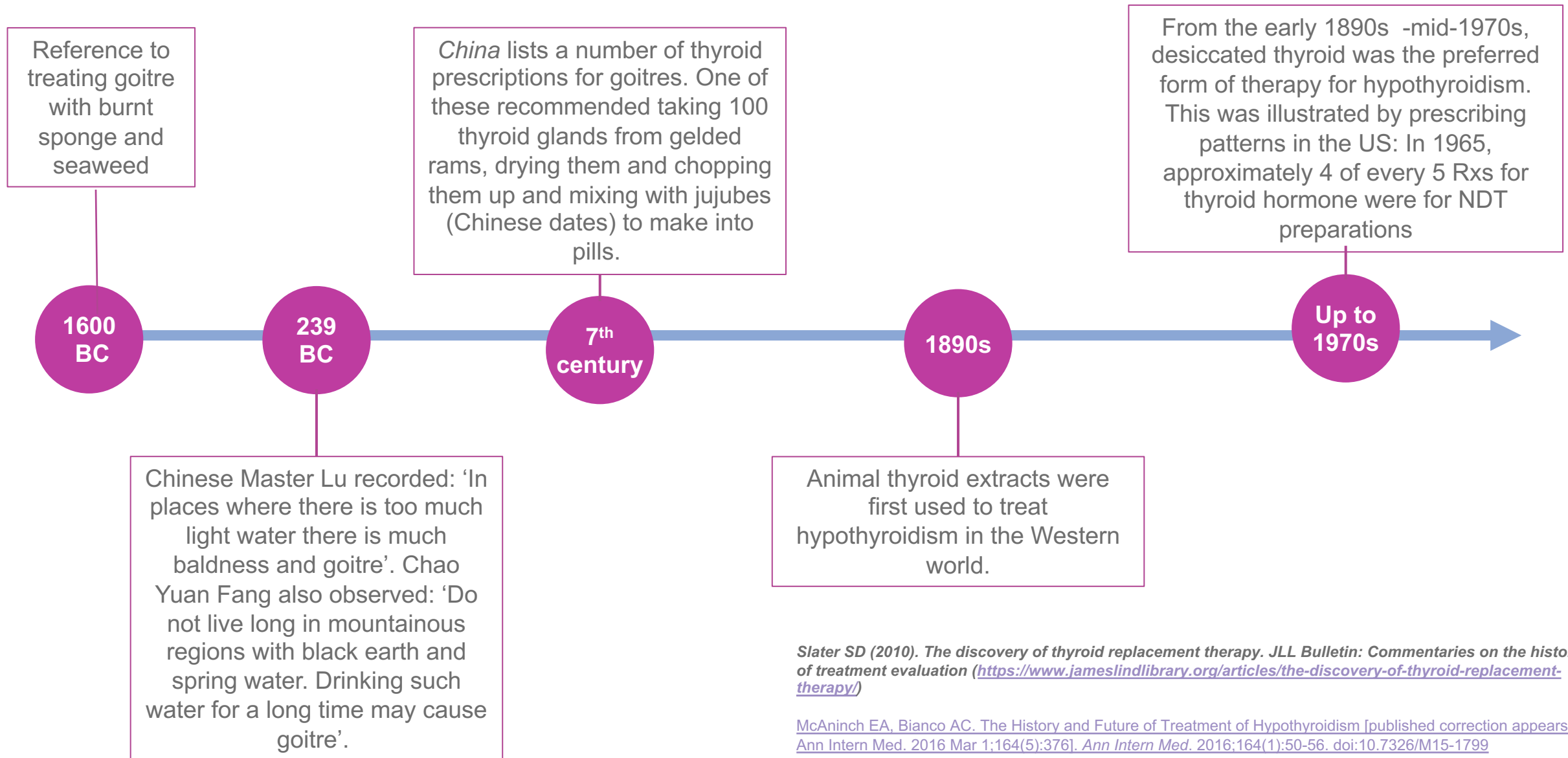


HISTORY OF THYROID MEDS

- The thyroid gland derives its name from the **Greek, θυρεός, shield**, because of the shield-like shape of the tracheal cartilage.
- Its long history is inseparable from that of goitre – **Latin guttur**, showing there must always have been people with goitrous, swollen necks.
- There are many past references worldwide to endemic goitre and cretinism, notably prevalent in mountain valley regions but absent in coastal regions.



HISTORY OF THYROID MEDS



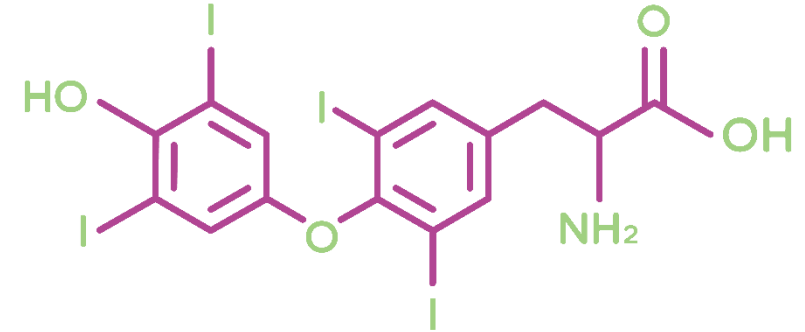


LEVOTHYROXINE (T4)

- The most commonly prescribed thyroid medication for hypothyroidism
- Important to remember that it's **inactive** and needs to be converted!
- Usually compounded in immediate release as it has such a long half-life:

6-7 days in euthyroid patients
9-10 days in hypothyroid patients
3-4 days in hyperthyroid patients

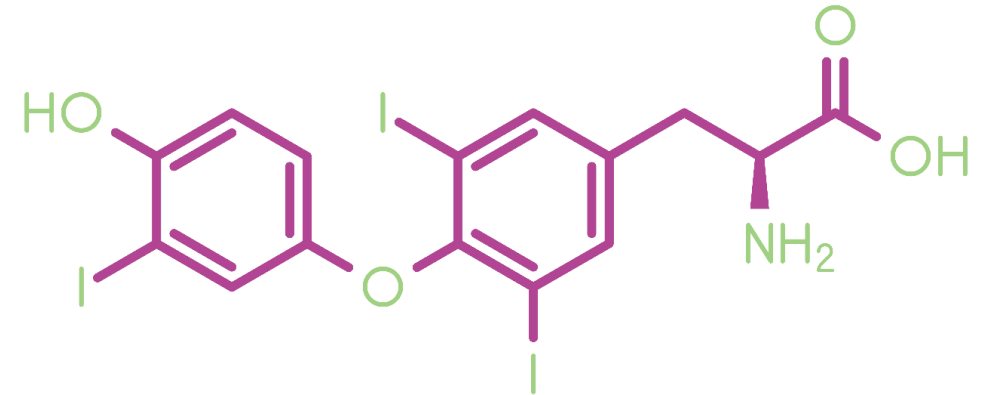
- You may want to **avoid slow-release** T4 due to the long half -life and patient absorption issues.
- Compounded T4 contains XX compared with commercially available T4 which contains many fillers such as corn starch, sugar, talc and lactose, which patients can respond negatively to.
- Take on an empty stomach first thing in the morning. Wait **60 mins** before drinking caffeinated beverages or any food.





LIOTHYRONINE (T3)

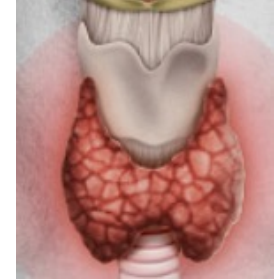
- For patients who have not responded positively to T4 treatment, a trial of T3 or combined T4 & T3 can be given.
- Elimination half-life of 2 days or less, so immediate or slow-release capsules can be used.
- Take on an empty stomach first thing in the morning. Wait 30 mins before drinking caffeinated beverages or any food.
- Consistency is key- take medication at the same time every day





THYROID USP

- Desiccated porcine thyroid
- Not completely bioidentical- differs by 4 amino acid chains, so if treating someone with autoimmune issues, they may make antibodies against this.
- Standardised to T4 38mcg and T3 9mcg per 65mg (1 grain)
- Do a lab evaluation in 4-6 weeks; which symptoms have improved or not improved?



Thyroid USP also contains small amounts of T1 and T2, so if converting from a desiccated tablet to a synthetic T4 & T3, the patient may not do so well due to the lack of T1 & T2. The small amount of activity can improve symptoms in some patients.

- Need to keep a close eye on patients with autoimmune issues
- Stress, winter months (lack of vitamin D) can mean doses need adjusting.



Conversion Guide

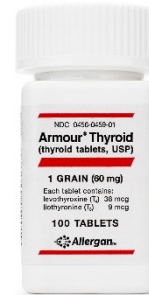
Armour Thyroid	NP-Thyroid	Nature Throid	Levothyroxine (synthetic T4)	Liothyronine (synthetic T3)
¼ grain (15 mg)	¼ grain (15 mg)	¼ grain (16.25mg)	9.5mcg	2.25mcg
½ grain (30mg)	½ grain (30mg)	½ grain (32.5mg)	19mcg	4.5mcg
		¾ grain (48.75mg)	28.5mcg	6.75mcg
1 grain (60mg)	1 grain (60mg)	1 grain (65mg)	38mcg	9mcg
		1 ¼ grain	47.5mcg	11.25
1.5 grain (90mg)	1.5 grain (90mg)	1 ½ grain	57mcg	13.5mcg
		1 ¾ grain	66.5mcg	15.75mcg
2 grain (120mcg)	2 grain (120mcg)	2 grain	76mcg	18mcg
		2 ¼ grain	85.5mcg	20.25mcg
		2 ½ grain	95mcg	22.5mcg
3 grain (180mg)		3 grain	114mcg	27mcg
4 grain (240mg)		4 grain	152mcg	36mcg
5 grain (300mg)		5 grain	190mcg	45mcg



TYPES OF COMBINED THYROID MEDICATION



Specialist Pharmacy
Compounded T3 & T4
& porcine derived NDT



Armour Thyroid
Porcine derived NDT



Novothyral
Synthetic



Synthroid
Synthetic



ERFA Thyroid
Porcine derived NDT



Nature Thyroid
Porcine derived NDT



BENEFITS OF COMPOUNDED THYROID MEDS

- Dose can be made to **individualised** strengths and patients can be started on less than the commercially available 25µg
- Made without **lactose** and **unnecessary fillers**= easier to absorb
- Made without **colouring agents**
- Made without **gluten** (gluten looks like a thyroid molecule)
- Improve patient **compliance** by **combining** T4 and T3
- Cheaper than using T3 and T4 separately
- Support pharmacists can offer
- Adjunct therapies such as hair tonics to help with symptoms
- Compounding pharmacies play a critical role during medicine shortages, which is a prevalent concern for thyroid meds such as Armour. Compounded ensures a stable supply.

HYPOTHYROIDISM: SUGGESTED PROTOCOL

MARION GLUCK TRAINING ACADEMY 2021 ©



BHRT Consultation Protocol

- ☐ Medical history
 - ☐ Menstrual history
 - ☐ Manual/palpation exam of the thyroid to identify goitre
- Blood tests: TSH, FT4, FT3, Thyroglobulin Abs, Thyroid peroxidase

Results to support underactive thyroid diagnosis:

- Blood tests showing elevated TSH, low T3 and low T4 or a combination of
- Positive thyroid antibodies confirm Hashimoto's
- Repeat blood tests at a suitable interval if patients are symptomatic but blood test appear normal
- Consider doing more comprehensive panel that includes T3 rT3 and auto antibodies

Guidelines for explaining the diagnosis to the patient:

1. Explain results and what they mean using a diagram of the feedback mechanism of how the thyroid works.
2. Explain why TSH is elevated and T3 and T4 are low, and reassure that the treatment is safe and will be monitored
3. Even if results are normal, a patient may still have symptoms of underactive thyroid.
4. Explain that treatment is often very effective and will need regular monitoring.



HYPOTHYROIDISM: SUGGESTED PROTOCOL

- **Dosages for T3:** start 5mcg; make increments as necessary according to thyroid function test results (10-20mcg daily, increase to 60mcg od in 2-3 divided doses. Dose should be increased gradually BNF)
- **Dosages for T4:** start at 25mcg; make increments as necessary

Symptoms	Prescription (Rx)
Mild Fatigue, apathy, lethargic, thinning hair and low mood	25 mcg T4 thyroxine (Levothyroxine) OR ¼ grain of Armour Thyroid (0.015 mcg equivalent dose)
Mild to moderate Excessive fatigue, depression, lethargy, thinning hair, dry skin, loss of side of eyebrows	50 mcg T4 thyroxine (Levothyroxine) + 5 mcg T3 OR ½ grain of Armour Thyroid (0.0235 mcg equivalent dose)
Severe Excessive fatigue, depression, lethargy, hair loss, very dry skin, loss of side of eyebrows, increased infections and aches and pains	50 mcg – 150 mcg T4 thyroxine plus 10-20 mcg T3 per day. (Armour not prescribed in severe cases)

ONGOING MONITORING

- **SIDE EFFECTS**

- increased appetite
 - weight loss
 - heat sensitivity.
 - excessive sweating
 - headache
 - hyperactivity
 - nervousness
 - anxiety
- (all symptoms of *hyperthyroidism*)



- **Recommended dose titration increments**

Dosages for T3: start 5mcg; make increments depending on subsequent testing

Dosages for T4: start at 25mcg; make increments as necessary



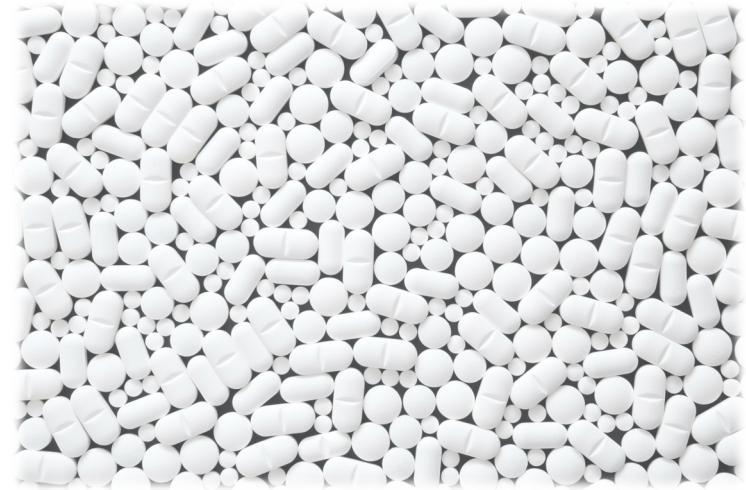
PRECAUTIONS

CONTRAINDICATIONS

- Hyperthyroidism
- Adrenal insufficiency such as Addison's disease

REMEMBER:

Thyroid medications have a narrow therapeutic index, so careful dose titration is needed. Doses that are incorrect by just a small amount can result in symptoms of over or under treatment



THYROID HORMONES, EITHER ALONE OR WITH OTHER THERAPEUTIC AGENTS, SHOULD NOT BE USED FOR THE TREATMENT OF OBESITY OR WEIGHT LOSS FOR PEOPLE WITH THYROID DISEASE. LARGER DOSES MAY PRODUCE SERIOUS OR EVEN LIFE-THREATENING MANIFESTATIONS OF TOXICITY.



HYPERTHYROIDISM

- High T4 and T3 and low or non-existent amounts of TSH indicate an **overactive thyroid**
- **This is usually dealt with in secondary care. Can discuss with an Endocrinologist and put on medications to help with symptomatic control**
- **Radioactive iodine**
- **Antithyroid drugs:** Carbimazole and propylthiouracil
- **Surgery:** thyroidectomy
- **Beta blockers**

RECOMMENDED TO REFER TO AN ENDOCRINOLOGIST



CURRENT GUIDELINES: BRITISH THYROID ASSOCIATION

Advice for healthcare professionals:

- Generic prescribing of levothyroxine remains appropriate for majority of patients and the licensing of these generic products is supported by bioequivalence testing.
 - A small proportion of patients treated with levothyroxine report symptoms, often consistent with thyroid dysfunction, when their tablets are changed to a different product.
 - If a patient reports symptoms after changing their levothyroxine product, consider testing thyroid function.
 - If a patient is persistently symptomatic after switching levothyroxine products, consider consistently prescribing a specific levothyroxine product known to be well tolerated by the patient.
 - If symptoms or poor control of thyroid function persist despite adhering to a specific product, consider prescribing levothyroxine in an oral solution formulation.
 - Report suspected adverse reactions to levothyroxine medicines, including symptoms after switching products, to the Yellow Card scheme.
-
- **Some health authorities are advising GPs to switch patients on Liothyronine (L-T3) to Levothyroxine (L-T4).**
 - The commercial imperative is to cut prescription costs - clinical needs of patient should come before financial considerations.
 - The BTA statement recommends L-T4 as the standard treatment for hypothyroidism. Treatment with combination L-T3 plus L-T4 should only be considered in exceptional cases.



CURRENT GUIDELINES: BRITISH THYROID ASSOCIATION

FAQs:

- **Should my patient be switched from L-T3 to L-T4?**
- The decision to switch from L-T3 to L-T4 should be based on clinical considerations and should be reached in conjunction with the patient after a discussion of the risks and benefits. Patients established on L-T3 who continue to derive benefit from its use should continue on L-T3. However, patients with uncertain benefits should be considered for a switch to L-T4 and advice should be sought from an endocrinologist on how this can be safely done.
- **Is there any problem switching my long-established L-T3 patients to L-T4?**
 - A change in treatment can result in some instability in thyroid status which can actually cost more to address than continuing with L-T3. The patient should be made aware of this risk to their thyroid status before switching.
- **What is the process for patients who have been advised to switch to L-T4 from combined L-T3 + L-T4 or from L-T3 only?**
 - This should be made gradually with the aim of avoiding under-replacement or overreplacement. The final L-T4 requirement is likely to be around 1.6mcg/kg. Any information about previous L-T4 dosage that achieved a normal serum TSH will be a useful guide. Gradual reduction of L-T3 starting at the same time as introducing or increasing L-T4 may be a preferable alternative. Careful monitoring of the patient by an endocrinologist during the transition period is essential.
- **Are there any particular patient types which merit extra caution?**
 - In patients over the age of 60, or of any age with known heart disease, additional care is required to avoid over-replacement and L-T4/L-T3 combination therapy or L-T3 only therapy is not recommended in pregnancy.



CURRENT GUIDELINES: NICE

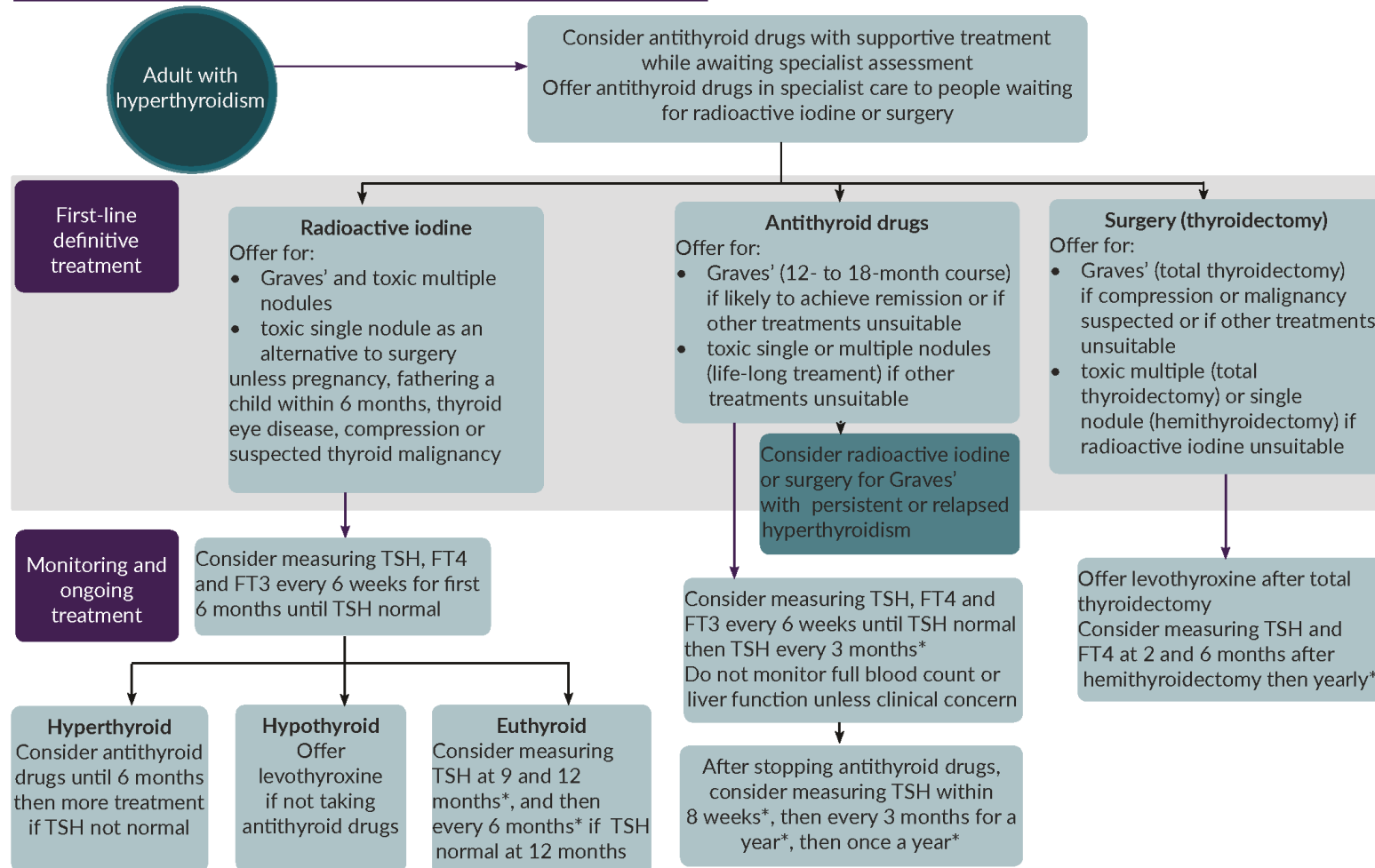
Thyroid disease: assessment and management NICE guideline [NG145] Published: 20 November 2019

- Offer levothyroxine as first-line treatment for adults, children and young people with primary hypothyroidism.
- Do not routinely offer liothyronine for primary hypothyroidism, either alone or in combination with levothyroxine, because there is not enough evidence that it offers benefits over levothyroxine monotherapy, and its long-term adverse effects are uncertain.
- Do not offer natural thyroid extract for primary hypothyroidism because there is not enough evidence that it offers benefits over levothyroxine, and its long-term adverse effects are uncertain.
- Natural thyroid extract does not have a UK marketing authorisation so its safety is uncertain.
- Consider starting levothyroxine at a dosage of 1.6 micrograms per kilogram of body weight per day (rounded to the nearest 25 micrograms) for adults under 65 with primary hypothyroidism and no history of cardiovascular disease.
- 1-Consider starting levothyroxine at a dosage of 25 to 50 micrograms per day with titration for adults aged 65 and over and adults with a history of cardiovascular disease.

CURRENT GUIDELINES: NICE

Hyperthyroidism in adults: management and monitoring

NICE National Institute for Health and Care Excellence



*With cascading - measuring FT4 in the same sample if TSH above reference range, and FT4 and FT3 in the same sample if TSH below reference range.

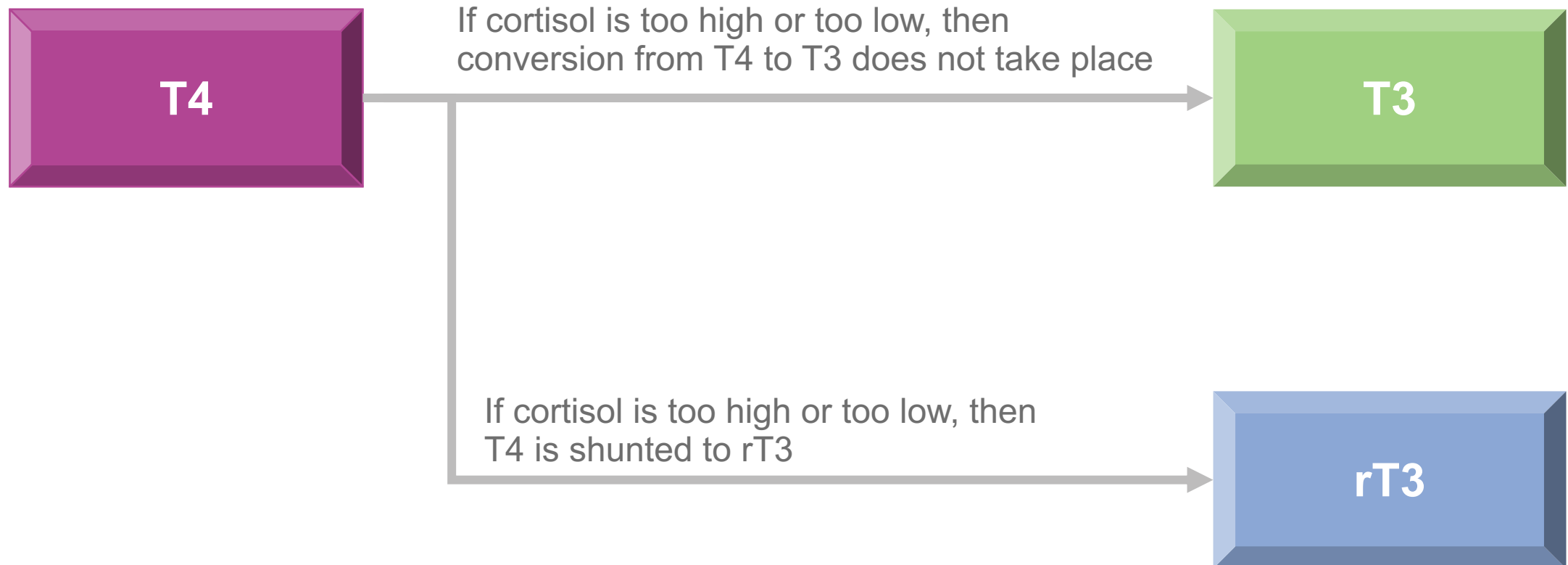


A FUNCTIONAL MEDICINE PERSPECTIVE

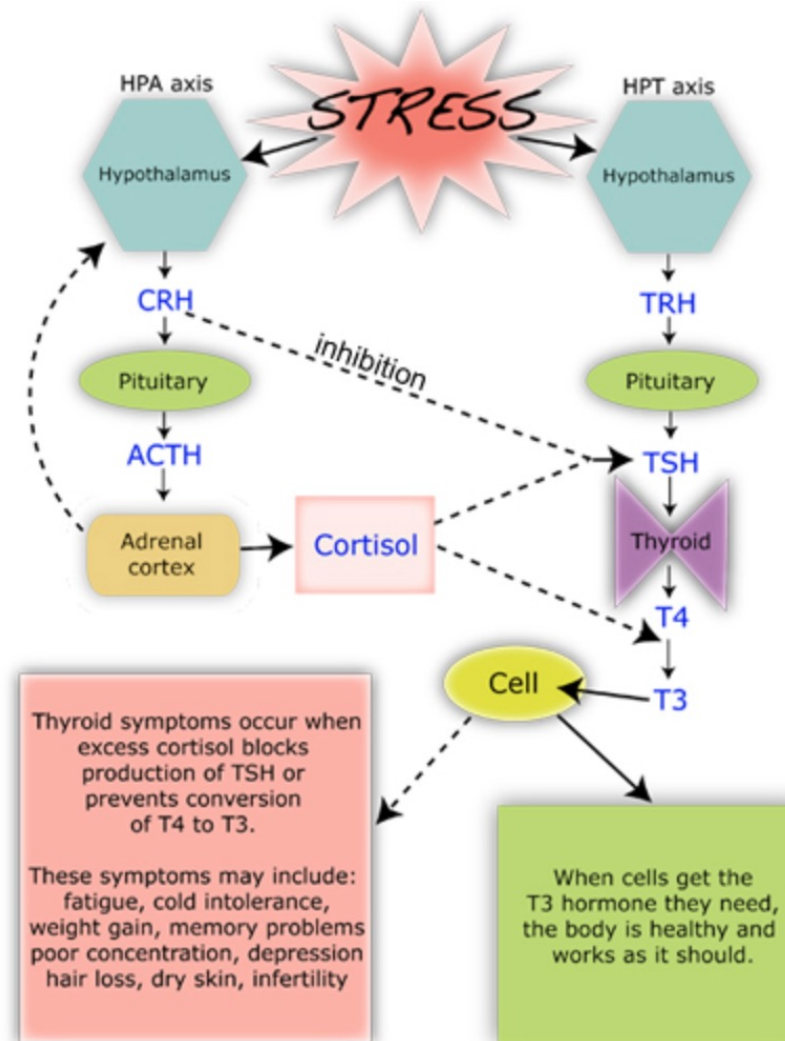


STRESS AND THE THYROID GLAND

With hypothyroidism, it is important to consider what is happening with adrenal function.



THE CORTISOL CONNECTION





ENDOCRINE DISRUPTORS, STRESS, AND INFLAMMATION- IMPACT ON THYROID FUNCTION

- **Endocrine disruptors** can have a potentially major impact on the function of the thyroid.
 - Endocrine-disrupting chemicals, such as bisphenol A (BPA), phthalates, and flame retardant compounds (PCBs), can interfere with thyroid gland functioning and thyroid hormone transport through multiple mechanisms.¹
 - These are often found in food, food packaging, water and personal care products.
- Observational studies have also noted a higher prevalence of autoimmune thyroid diseases in people living in **polluted areas, near petrochemical plants**, and in areas contaminated with **organochlorine pesticides** or **PCBs**.²
- **Stress and chronic inflammation** may also impact thyroid hormone balance.





ENHANCING THYROID HEALTH

- Addressing toxicant exposures, systemic inflammation, and chronic stress are all approaches to enhancing thyroid health.
 - **biotransformation**
 - **anti-inflammatory and elimination diets**
 - **specific nutrients**
 - **optimising gut and liver health**
- Adequate intake and availability of **iodine** and **iron** are crucial for thyroid hormone synthesis, while selenium and zinc are needed for the conversion of T4 to T3
- **Ashwagandha** has been used to help address thyroid dysfunctions.
 - Study results showed an improved serum TSH and T4 after receiving 600mg of root extract per day for 8 weeks compared to placebo, with few mild and temporary adverse effects.¹
- The **thyroid-liver axis** is another illustration of the system-wide impact of thyroid health. The liver plays an important role in thyroid hormone activation, transport, and metabolism, and thyroid hormones impact hepatocyte activity and liver metabolism.

1. Sharma AK, Basu I, Singh S. Efficacy and Safety of Ashwagandha Root Extract in Subclinical Hypothyroid Patients: A Double-Blind, Randomized Placebo-Controlled Trial. J Altern Complement Med. 2018 Mar;24(3):243-248.



ENHANCING GUT HEALTH

- **Gut health and a balanced microbial landscape**
- Intestinal health is vital to many system processes in the body, including thyroid homeostasis.
 - Dysbiosis and intestinal autoimmune diseases have been reported concurrently with autoimmune thyroid diseases. Functional thyroid disorders have been associated with bacterial overgrowth and a difference in microbial composition.
- **GI inflammation affects T3.**
- Changes in peripheral deiodination of thyroxine may be the cause of decreased concentration of T3 in some patients with Crohn's disease and other inflammatory bowel conditions. Inflammation and dysbiosis can be addressed in these patients and this improves the severity of the inflammatory bowel disease and general wellbeing

Knezevic J, Starchl C, Tmava Berisha A, Amrein K. Thyroid-gut-axis: how does the microbiota influence thyroid function? *Nutrients*. 2020;12(6):1769. doi:[10.3390/nu12061769](https://doi.org/10.3390/nu12061769)
Vierhapper H, Grubeck-Loebenstien B, Ferenci P, Lochs H, Bratusch-Marrain P, Waldhäusl W. Alterations in thyroxine metabolism in Crohn's disease. *Hepatogastroenterology*. 1981 Feb;28(1):31-3. PMID: 6783498.



ENHANCING GUT HEALTH

- **Diversified diet**, limited use of processed foods, avoidance of prolonged restricted diets, and consumption of adequate dietary fibre, all promote a healthy microbiome.
- Consumption of **whole grains/ fibre** may modify the intestinal microbiota and promote the growth of *Bifidobacteria* and *Lactobacilli*. Studies have shown that seafood, including marine seaweeds and invertebrates, are rich in dietary fibres and can help maintain symbiosis in the gut.
- Recommend INVIVO: Bio.Clear Microbia GI as a herbal antimicrobial as part of a 5 R gut restoration programme. Usually patient has functional gut testing to evaluate the exact cause of dysbiosis and other gut imbalances and can be done in conjunction with a nutritional therapist.





LOW DOSE NALTREXONE

- LDN is a safe, non-toxic and inexpensive drug that helps regulate a dysfunctional immune system.
- It binds to the **endorphin receptors** for about 1 – 1/2 hours and its effects last about 4 - 6 hours.
- The effects of LDN are **analgesia and anti-inflammatory**.
 - One of the other effects is that it increases the production of your own endorphins.
- While no scientific studies look directly at the relationship between low-dose naltrexone and Hashimoto's, some anecdotal evidence from clinicians and patients suggests that **LDN may improve Hashimoto's symptoms** and may even **lower TPO antibodies**.



Suggested starting doses:

The typical starting dose for people with Hashimoto's is 1.5 mg every night at bedtime. May need titrating.



ANTI-INFLAMMATORY ELIMINATION DIET

- One of the most prevalent and common root causes of symptoms in Hashimoto's is **food sensitivities**.
- Reactive foods trigger an **inflammatory response in the GI tract**, leading to malabsorption of nutrients, and intestinal permeability (contributing to leaky gut) when they are eaten.
- **Eliminating foods** can reduce gut and brain symptoms, skin breakouts, and pain. Some people will also see a significant reduction in thyroid antibodies

Common food sensitivity reactions:



GUT REACTIONS

Acid reflux, bloating, burning, constipation, diarrhoea, gas, cramping



HEART REACTIONS

Increased pulse, increased heart rate, palpitations



BRAIN REACTIONS

Anxiety, brain fog, depression, dizziness, fatigue, headaches, insomnia



SKIN REACTIONS

Acne, eczema, rashes



MUSCLE REACTIONS

Joint aches, pain, swelling, numbness, tingling in arms or legs



LUNG REACTIONS

Asthma, congestion, coughing, postnasal drip

THE BASIC ELIMINATION DIET

Food to be eliminated:

- Alcohol
- Gluten
- Dairy
- Soy
- Eggs
- Corn
- Nuts
- Shellfish
- Preservatives



Order for reintroduction of foods:

1. Alcohol
2. Gluten
3. Dairy
4. Soy
5. Eggs
6. Corn
7. Nuts
8. Shellfish
9. Preservatives



If symptoms do not improve after the initial three-week elimination, there may be other intolerances that need to be eliminated. The Advanced Elimination Diet is a good next step.



CASE STUDIES



"It's simple. My nurse blindfolds me, I spin around a few times, and then I try to reattach your tail."



CASE STUDY 1

Name: Barbara **Age:** 51



HISTORY & SYMPTOMS

- Long term history of hypothyroidism-presented with low energy, dry skin, puffy ankles, cold intolerance and weight gain.
- Initially started on levothyroxine which was titrated up incrementally.
- Poor conversion from T4 to T3

BLOOD TEST RESULTS

- Feb 2021
- TSH **0.06** (0.27- 4.2)
- T4 **20.7** (12-22)
- T3 **5.1** (3.1- 6.8)

Rx

- Thyroxine 100mcg daily



CASE STUDY 1

Name: Barbara **Age:** 51

FOLLOW UP BLOOD TEST RESULTS

- May 2021
TSH **0.08** (0.27- 4.2)
T4 **19** (12- 22)
Still feeling low energy and fatigue
- Dec 2021-
TSH **1.13** (0.27-4.2)
T4 **16.6** (12- 22)
T3 **3.4** (3.1- 6.8)
TGA **12.5** (0-115)
TPA **26** (0-34)
First time she had autoantibodies checked when I inherited her

What Rx would you think of offering Barbara?



CASE STUDY 1

Name: Barbara **Age:** 51

Rx

Added T3 5 mcg bd

Blood test results

- Feb 2022
TSH **1.26** (0.27- 4.2)
T3 **4.08** (3.1- 6.8)
T4 **13.72** (12- 22)

Rx

Increased Thyroxine to 100mcg
alternating with 125 mcg
Continue T3 5 mcg bd

Plan

Re-check thyroid function in 3 months

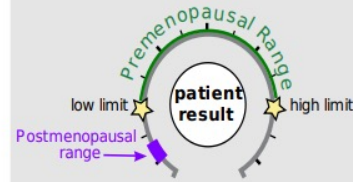


CASE 1

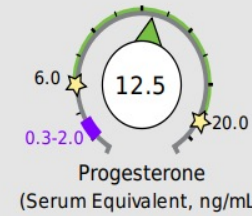
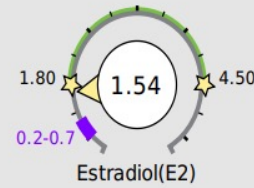
DUTCH PLUS RESULTS

Hormone Testing Summary

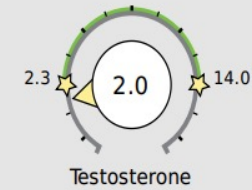
Key (how to read the results):



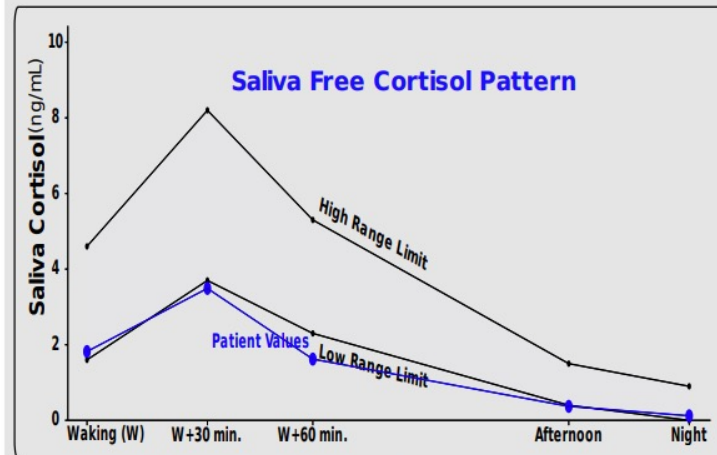
Sex Hormones See Pages 2 and 3 for a thorough breakdown of sex hormone metabolites



Progesterone Serum Equivalent is a calculated value based on urine pregnanediol.



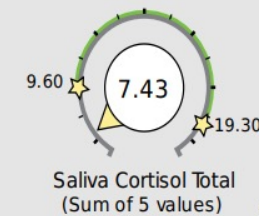
Adrenal Hormones See pages 4 and 5 for a more complete breakdown of adrenal hormones



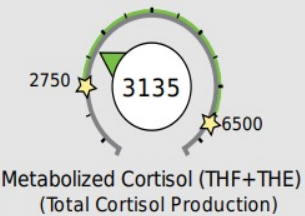
Free cortisol best reflects tissue levels. Metabolized cortisol best reflects total cortisol production.

Total DHEA Production

Age	Range
20-39	1300-3000
40-59	750-2000
>60	500-1200



cortisol
metabolism



PLEASE BE SURE TO READ BELOW FOR ANY SPECIFIC LAB COMMENTS. More detailed comments can be found on page 9.

The Cortisol Awakening Response (CAR) was 1.67ng/mL (expected range 1.5-4.0) or 91.8% (range 50-160%). See page 5 for more details.



CASE STUDY 2

Name: Marissa **Age:** 60



HISTORY & SYMPTOMS

- Long term hypothyroidism- started at time of her perimenopause. Symptoms of fatigue and weight gain.
- Monitored as part of her BHRT treatment

BLOOD TEST RESULTS

- Sept 2020-
- TSH **1.4** (0.27- 4.2)
- T4 **14.4** (12- 22)
- T3 **5.5** (3.1- 6.8)

Rx

Novothyral 100mcg daily (100mcg T4, 20mcg T3)



CASE STUDY 2

Name: Marissa **Age:** 60

FOLLOW UP BLOOD TEST RESULTS

- March 2022
- TSH **1.25** (0.27- 4.2)
- T3 **3.8** (3.1-6.8),
- T4 **12.3**(12-22)

Feeling much more tired,
dry skin, finding it hard to
shift weight.

Rx

Continue Novothyral 100 mcg and add in T4 25mcg

When would you re-check the blood tests?



CASE STUDY 2

Name: Marissa **Age:** 60

Thyroid function to be rechecked 3 months

Some of the additional T4 should convert to T3



CASE STUDY 3

Name: Amreen **Age:** 57



HISTORY & SYMPTOMS

- Subclinical hypothyroidism
- Controversy about the range of TSH

BLOOD TEST RESULTS

- May 2021- TSH **5.9** (0.27- 4.2), T3 **4.4** (3.1- 6.8) T4 **13.6** (12-22) TGA **27.6** (0-115) TPA **8.2** (0-34)
- June 2021- TSH **4.63** (0.27-4.2), T4 **12.9** (12-22), TPA normal

TREATMENT

Nutritional therapist, elimination diet, gluten free, thyroid support complex



CASE STUDY 3

Name: Amreen **Age:** 57

FOLLOW UP BLOOD TEST RESULTS

- October 2021- TSH **5.99** (0.27-4.2), T3 **3.2** (3.1- 6.8), T4 **15.6** (12- 22), TGA **19.3** (0-115), TPA **<9** (0-34)
- March 2022- TSH **8.47** (0.27-4.2). T3 **4.5** (3.1-6.8), T4 **14.6** (12-22), TGA **25.5** (0-115), TPA **10.9** (0-34)

What would you offer as a prescription?



CASE STUDY 3

Name: Amreen **Age:** 57

Rx

As she is symptomatic of hypothyroidism, we started her on 25mcg Thyroxine daily, aiming for a TSH <2



CASE STUDY 4

Name: Ralph Age: 64



HISTORY AND SYMPTOMS

- Lives in Crete where he had his bloods done
- Presented with low energy and libido plus weight gain and high blood pressure

Rx

Test 25mg od
DHEA 25mg od

BLOOD TEST RESULTS (May 2020)

Testosterone **4.75** (2.41- 8.3)
DHEA **0.580** (0.330- 2.95)



CASE STUDY 4

Name: Ralph Age: 64

BLOOD TEST RESULTS

- TSH 12.57 (0.27 to 4.2)
- T4 61 (60-165)
- T3 1.56 (0.9-3)

What do these blood test results show?



CASE STUDY 4

Name: Ralph Age: 64

SUBCLINICAL THYROIDISM

Ralph was not keen on starting any medication and decided to wait and see if anything improved

BLOOD TEST RESULTS (AUG 2020)

DHEA 9.1,
Total testosterone **12.25** (2.41- 8.3)
TSH **14.17** (0.27- 4.2)
T4 **43** (60- 165)
T3 **1.25** (0.9-3)

Major improvement in symptoms with 5 kg weight loss, lipid profile improved, LFTs normalised and BP normalised

Rx

Armour Thyroid ½ grain daily



CASE STUDY 4

Name: Ralph Age: 64

BLOOD TEST RESULTS (Nov 2020)

TSH 7.3 (0.27- 4.2)

T4 67 (60 - 165)

T3 2.05 (0.9 - 3)

WOULD YOU CHANGE HIS CURRENT Rx? (Armour ½ grain od)



CASE STUDY 4

Name: Ralph Age: 64

Rx

Armour thyroid increased to 1 grain daily

Problems with delivery to Crete due to Brexit & COVID- sourced Thyreogland from Germany but felt much better on Armour Thyroid

BLOOD TEST RESULTS (SEP 2021)

TSH **0.58** (0.27- 4.2)

T4 **79** (60- 165)

T3 **2.23** (0.9- 3)

Patient feels very well and
thyroid function is optimal!



CASE STUDY 5

Name: Helena **Age:** 53



HISTORY & SYMPTOMS

- Perimenopausal symptoms- erratic cycle, heavy bleeding, irritability, worsening PMS, terrible insomnia

BLOOD TEST RESULTS (JULY 2020)

May 2021- TSH **1.92** (0.27- 4.2), T3 **2.4** (3.1- 6.8) T4 **9.6** (12-22) FSH **3.8**
Progesterone **7**, testosterone **1.2**, oestradiol **3047**, DHEA **7.5**, vit D **92**

TREATMENT

Already taking DHEA 10mg so added Utrogestan 200mg nocte. Also added a thyroid support complex and DIM extra to reduce oestrogen dominance



CASE STUDY 5

Name: Helena **Age:** 53

FOLLOW UP BLOOD TEST RESULTS

- November 2020-TSH **1.81** (0.27-4.2), T3 **3.8** (3.1- 6.8), T4 **13.5** (12- 22), TGA **26.3** (0-115), TPA **15.7** (0-34)
- Addressing the oestrogen dominance, adding progesterone and supporting the thyroid with nutrients, improved thyroid function.
- The initial disturbance was down to the oestrogen dominance impacting thyroid function. It was useful to know there was no autoimmunity present.



TAKE HOME POINTS

Whenever T4 is administered, you are depending upon proper conversion of T4 to T3 to obtain desired metabolic effects

It may be important to replace T4 and T3 as there may be factors that inhibit this conversion.

The human body is complex- important to consider thyroid health in the context of overall health. Look for nutritional deficiencies and reversible factors. Treat the patient and symptoms, not just the numbers.

Always consider adrenal health when replacing thyroid hormones- addressing both can be of synergistic benefit to the patient



RECOMMENDED READING

- **Your Thyroid and how to Keep it Healthy**, Dr Barry Durrant Peatfield
- **Diagnosis and Management of Hypothyroidism**, Dr Gordon Skinner
- **Hypothyroidism Type 2- The Epidemic**, Dr Mark Starr
- **Hypothyroidism: The Unsuspected Illness**, Dr Broda Barnes
- **Stop the Thyroid Madness**, Janine A Bowthorpe
- **Why do I still have thyroid symptoms?** Datis Kharrazian
- **Adrenal Thyroid Revolution**, Aviva Romm
- **Hashimoto's Thyroiditis: Root Cause**, Izabella Wentz
- **Resources:** IFM

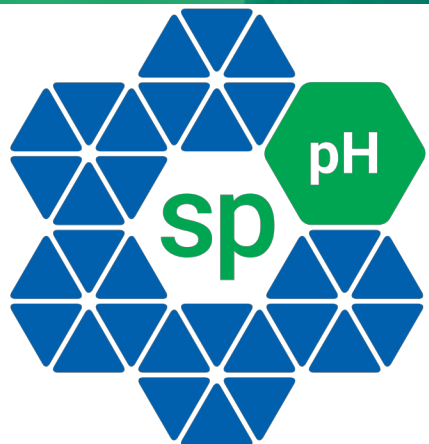


RESOURCES

- [The Institute for Functional Medicine](#)
- [Tanya Borowski-](#) Nutritional & Functional Medicine Practitioner- podcast and courses
- [The Thyroid Answers podcast](#) by Dr Eric Balcavage



Q&A



specialist pharmacy



PRESCRIBER HELPDESK

To speak with a pharmacist directly, call

+44 (0) 203 991 7799

or email

prescribersupport@specialist-pharmacy.com

- Thyroid prescriber guide available upon request
- Price list with NDT conversion table

A close-up photograph of a silver stethoscope resting on a thick stack of papers. The stethoscope's chest piece is in sharp focus, while the background papers are blurred. A solid green horizontal bar is positioned across the lower portion of the image, serving as a background for the text.

THANK YOU