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Thyro Complex by Biotex Life Solutions: An Evidence-Based Narrative Review of Myo-Inositol, Botanical Extracts and Micronutrients Relevant to Thyroid-Related Physiology

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Abstract:

Background. Thyroid hormone synthesis, redox regulation and peripheral hormone metabolism depend on coordinated processes involving iodine transport, thyroid peroxidase, thyroglobulin, selenium-dependent enzymes, deiodinases and intracellular signalling. Multicomponent nutraceuticals combine nutrients and botanicals intended to support these pathways, but ingredient-level plausibility cannot establish the efficacy of a finished formulation.

Objective. To critically evaluate the declared Thyro Complex formulation developed by Biotex Life Solutions, with emphasis on clinical relevance, dose translation, botanical standardisation, safety, medication interactions, claim substantiation and product-specific research requirements.

Methods. A structured narrative review was developed from literature published primarily between 2016 and August 2026. Priority was given to systematic reviews, meta-analyses, randomised or prospective clinical studies, major clinical reviews, safety reports and authoritative guidelines. DOI and PMID records were checked for key references. Older evidence was retained only when indispensable for mechanism or clinically relevant interaction safety. Because complete subscription-database exports, dual-reviewer screening and a verified PRISMA log were unavailable, the article does not claim systematic-review status.

Results. The strongest direct thyroid-related human evidence concerns myo-inositol combined with selenium, although the evidence base remains small and heterogeneous. Selenium may influence thyroid-stimulating hormone (TSH), thyroid autoantibodies and oxidative-stress markers in selected populations, but patient-centred benefit and universal supplementation remain unproven. One small randomised trial of ashwagandha reported changes in thyroid indices, while case reports describe thyroiditis or thyrotoxicosis. Iodine, zinc and vitamin B12 are primarily relevant in relation to physiological requirements or deficiency. Evidence for Bauhinia variegata, Commiphora wightii, Achyranthes aspera, Glycyrrhiza glabra, Coleus forskohlii, Capsicum annuum, chromium, magnesium, copper and L-tyrosine is indirect, mechanistic, traditional or non-thyroid-specific. The declared amounts of myo-inositol, selenium and

ashwagandha are lower than doses used in principal clinical studies.

Conclusion. Thyro Complex has an ingredient-level nutritional and mechanistic rationale but no established finished-product clinical efficacy. It should not be represented as a treatment for hypothyroidism, autoimmune thyroid disease, thyroid nodules or weight gain, or as a substitute for levothyroxine or antithyroid therapy. Product-specific claims require verified formulation specifications, stability and safety data, interaction controls and a randomised controlled trial of the final commercial formulation.

Keywords: thyroid nutrition; myo-inositol; selenium; iodine; ashwagandha; nutraceutical; thyroid hormone synthesis; Hashimoto thyroiditis; levothyroxine interaction; botanical standardisation.

Abbreviations

BP, blood pressure; cAMP, cyclic adenosine monophosphate; DNA, deoxyribonucleic acid; DOI, digital object identifier; DUOX2, dual oxidase 2; fT3, free triiodothyronine; fT4, free thyroxine; GI, gastrointestinal; GRADE, Grading of Recommendations Assessment, Development and Evaluation; HPLC, high-performance liquid chromatography; PMID, PubMed identifier; PRISMA, Preferred Reporting Items for Systematic Reviews and Meta-Analyses; RCT, randomised controlled trial; T3, triiodothyronine; T4, thyroxine; TgAb, thyroglobulin antibodies; TPO, thyroid peroxidase; TPOAb, thyroid peroxidase antibodies; TSH, thyroid-stimulating hormone; UPLC, ultra-performance liquid chromatography.

1. Introduction

Thyroid hormones influence basal metabolic activity, thermogenesis, cardiovascular function, gastrointestinal motility, skeletal muscle performance, reproductive physiology and nervous-system function. Symptoms commonly associated with thyroid dysfunction—including fatigue, weight change, impaired concentration, constipation, cold intolerance and reduced exercise tolerance—are clinically important but nonspecific. They may also arise from anaemia, vitamin B12 deficiency, sleep disorders, mood disorders, infection, medication effects, undernutrition and other endocrine or systemic conditions. A thyroid-oriented supplement must therefore be distinguished from diagnostic evaluation and disease treatment [1–6].

Thyroid nutrition is biologically complex. Iodine is incorporated into thyroglobulin during T4 and T3 synthesis; selenium is incorporated into deiodinases and several antioxidant enzymes; zinc participates in thyroid-related enzyme activity, receptor biology and transcriptional regulation; and intracellular phosphoinositol signalling is relevant to thyrotropin action. These physiological relationships provide a rationale for investigating carefully formulated nutritional interventions. They do not demonstrate that supplementation benefits nutrient-replete individuals or that a multicomponent product reproduces the effects of a single ingredient studied at another dose [1,2,7–9].

Thyro Complex is described as a multicomponent nutraceutical developed by Biotex Life Solutions. The declared formula contains myo-inositol, iodine, selenium, zinc, copper, magnesium, chromium, vitamin B12, L-tyrosine and seven botanical preparations. The present review evaluates the declared composition rather than an unspecified product concept. No randomised controlled trial of the complete finished formulation was identified in the evidence set reviewed for this revision.

The objectives are to: (i) explain the thyroid-related physiological pathways relevant to the formula; (ii) examine human evidence for the principal ingredients; (iii) distinguish clinical evidence from mechanism, tradition and extrapolation; (iv) evaluate dose comparability and safety; (v) identify medication interactions and high-risk populations; and (vi) define a product-development pathway capable of supporting scientifically proportionate claims.

2. Product identity, declared composition and verification requirements

The composition below is reproduced from the author-supplied formulation. Before journal submission, the company should verify every entry against the current approved product label, certificate of analysis and

manufacturing specification. The legal manufacturer, marketer, dosage form, serving size, unit count and maximum daily intake should be harmonised across the manuscript, product label and commercial website.

The current commercial presentation should be documented visually to link the reviewed formulation to the specific product version evaluated. A dedicated product figure is therefore reserved below.

Fig 1: Thyro Complex product presentation.

Table 1. Declared composition of Thyro Complex and information required for scientifically interpretable dose and safety assessment.

Ingredient	Declared form	Amount per supplied serving
Myo-inositol	Myo-inositol	300 mg
Bauhinia variegata	Bark extract	55 mg
Commiphora wightii	Oleoresin extract	50 mg
Withania somnifera	Ashwagandha extract	50 mg
Achyranthes aspera	Plant extract	50 mg
Glycyrrhiza glabra	Root extract	15 mg
Copper	Copper lysine complex	1.1 mg
Zinc	Zinc oxide	4 mg
Chromium	Chromium picolinate	300 µg
Selenium	Selenomethionine	30 µg
Magnesium	Magnesium carbonate	75 mg
Iodine	Potassium iodide	75 µg
Vitamin B12	Cyanocobalamin	1.8 µg
Coleus forskohlii	Root extract	30 mg
Capsicum annuum	Fruit extract	10 mg
L-tyrosine	Free amino acid	75 mg

The formulation was evaluated according to the declared ingredient forms and quantities shown above. Comparisons with published studies were interpreted cautiously because botanical extracts and nutrient preparations may differ in chemical composition and standardisation across studies.

3. Review approach and evidence translation

3.1 Review design

This article is an evidence-based narrative review rather than a systematic review. Literature published primarily from January 2016 to August 2026 was prioritised. The working evidence library contained PubMed-indexed and DOI-traceable records relevant to thyroid nutrition, myo-inositol, selenium, iodine, zinc, vitamin B12, ashwagandha, the declared botanicals, safety and levothyroxine interactions. Key references were cross-checked using PubMed, DOI or publisher metadata.

Evidence was interpreted using a translation hierarchy that separates evidence quality from applicability to the finished product. Product-specific randomised controlled trials are the strongest direct evidence for Thyro Complex. Human studies of the same or highly comparable formulation are strongly informative but remain partially transferable when dose, chemical form, excipients or co-ingredients differ. Ingredient-level randomised trials and systematic reviews support ingredient-level conclusions, observational human studies support associations rather than causation, and preclinical, mechanistic, traditional and expert evidence are hypothesis-generating rather than proof of clinical efficacy[10–12].

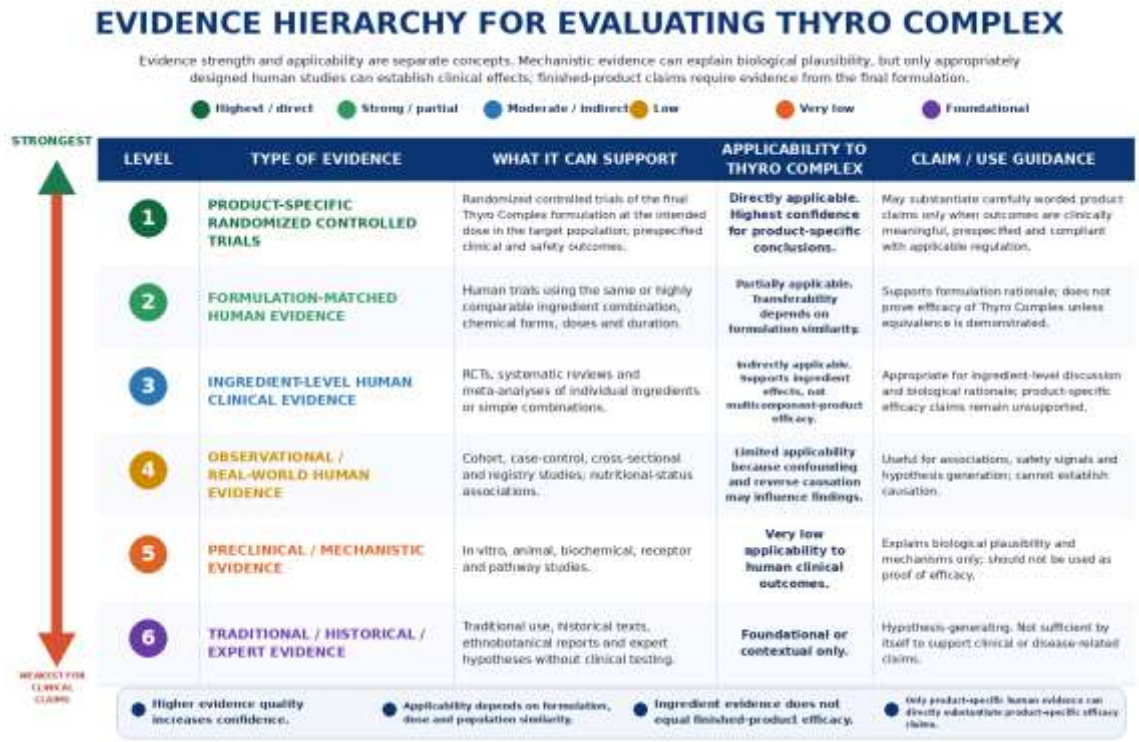

Figure 2. Evidence hierarchy for evaluating Thyro Complex. Evidence strength and applicability to the finished product are considered separately. Product-specific randomised controlled trials provide the strongest direct evidence, whereas formulation-matched and ingredient-level human studies are progressively less transferable. Observational evidence supports associations; preclinical, mechanistic and traditional evidence support biological plausibility or hypothesis generation but do not establish clinical efficacy.

3.2 Inclusion emphasis and limitations

Priority was given to systematic reviews, meta-analyses, randomised or prospective clinical studies, authoritative clinical reviews and safety evidence. Botanical reviews were retained when they clarified authentication, chemical variability or the absence of clinical thyroid evidence. Animal and in-vitro studies were not treated as evidence of clinical efficacy. Because full search exports, duplicate counts, dual-reviewer screening and formal study-level risk-of-bias judgements were not available, the manuscript does not present a PRISMA diagram, pooled effect estimate or GRADE certainty rating as if these procedures had been completed.

4. Thyroid physiology relevant to the formulation

4.1 Thyroid hormone synthesis

Dietary iodide is absorbed from the gastrointestinal tract and transported in blood to the thyroid. The sodium–iodide symporter transports iodide into the thyroid follicular cell, after which apical transport delivers iodide toward the follicular lumen. Thyroid peroxidase uses hydrogen peroxide, generated physiologically at the apical membrane largely through the DUOX2 system, to oxidise iodide and catalyse iodination of tyrosyl residues on thyroglobulin. Coupling of monoiodotyrosine and diiodotyrosine residues generates T3 and T4. Iodinated thyroglobulin is subsequently endocytosed and proteolysed, releasing thyroid hormones into the circulation. T4 is the predominant hormone secreted by the thyroid, while a substantial proportion of circulating T3 is generated subsequently by peripheral deiodination of T4[1,2,7,8].

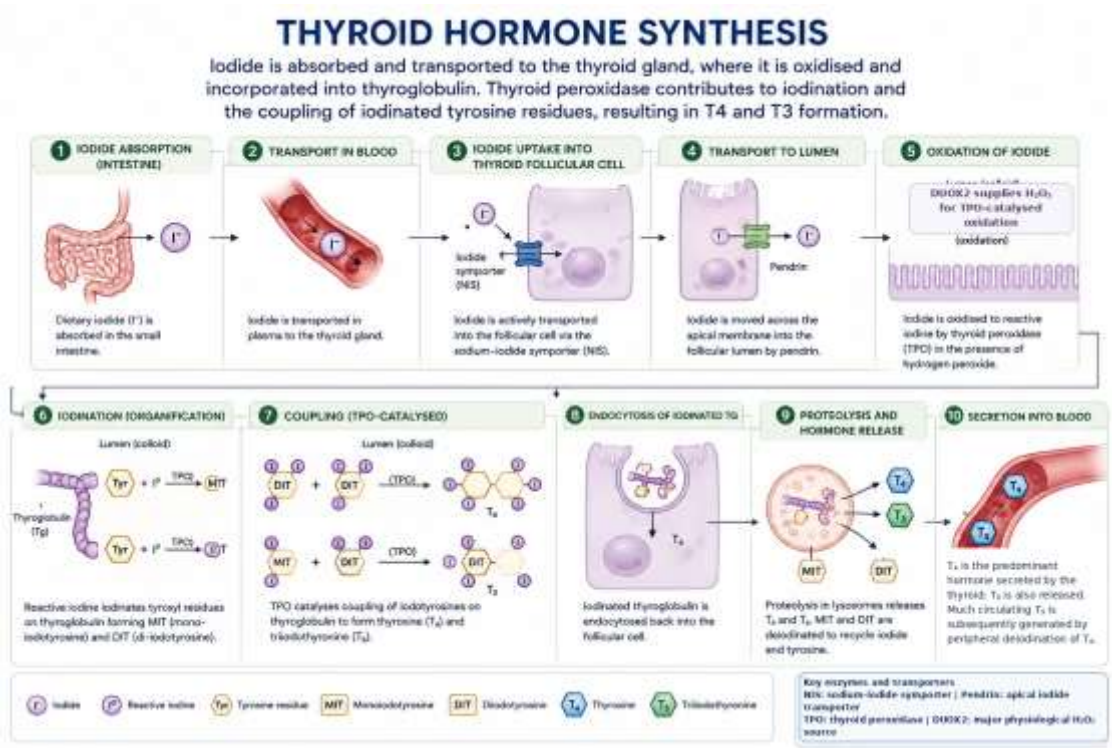

Figure 3. Thyroid hormone synthesis. Iodide is absorbed, transported into the thyroid follicular cell and delivered toward the colloid. DUOX2-dependent hydrogen peroxide generation supports thyroid-peroxidase-mediated oxidation, organification and coupling reactions that produce T4 and T3 within thyroglobulin. Following endocytosis and proteolysis, T4 and T3 are released; T4 is the predominant thyroid secretory product, and much circulating T3 is generated by peripheral deiodination.

4.2 Redox control and deiodination

Thyroid hormone synthesis requires controlled hydrogen peroxide generation. This makes the thyroid highly dependent on redox regulation. Selenium-containing glutathione peroxidases and thioredoxin reductases contribute to antioxidant defence, while selenium-containing deiodinases participate in activation and inactivation of thyroid hormones. Type 1 and type 2 deiodinases contribute to T3 production; type 3 deiodinase contributes to hormone inactivation. Selenium deficiency can disrupt these systems, but supplementation beyond adequacy does not necessarily improve clinical outcomes [8,13–16].

4.3 Thyroid hormones, energy and symptom specificity

Thyroid hormones influence mitochondrial activity, oxygen consumption, lipid and carbohydrate metabolism, protein turnover and thermogenesis. These functions explain associations between thyroid dysfunction, fatigue and weight change. They do not justify using fatigue, weight gain or impaired concentration as self-diagnostic indicators of thyroid disease. Any product claim concerning fatigue, cognition or weight requires direct measurement with validated outcomes in an appropriately defined population.

5. Myo-inositol and selenium

5.1 Biological rationale

Myo-inositol participates in phosphoinositide signalling and has been proposed as a second-messenger component of TSH-related pathways. Selenium contributes to deiodinases and redox enzymes. Their combination is therefore biologically complementary: myo-inositol may influence signal transduction, while selenium may influence deiodination and antioxidant defence. This rationale is mechanistic and should not be presented as proof that any dose or combination normalises thyroid function [17,18].

5.2 Human evidence

Clinical investigations of myo-inositol combined with selenium have generally enrolled patients with autoimmune thyroiditis and mild or subclinical hypothyroidism. Studies commonly used approximately 600 mg/day myo-inositol with about 83 µg/day selenium for six months. Several reports described reductions in TSH or thyroid autoantibodies and, in some cohorts, changes in quality-of-life or thyroid-hormone measures. However, the evidence base is small, includes overlapping research groups and formulations, and is limited by heterogeneous designs, selected populations and incomplete long-term outcomes [18–23].

Recent meta-analyses found that combination supplementation may reduce TSH more than selenium alone and may influence thyroglobulin antibodies, but effects on TPO antibodies, T3 and T4 were not consistently significant. The 2026 updated analysis still included only a small number of eligible comparative studies and explicitly called for larger randomised trials. Accordingly, the evidence supports a clinical-development hypothesis rather than routine treatment or a product efficacy claim[10–12,24,62].

Table 2. Principal human evidence for myo-inositol–selenium combinations relevant to the formulation.

Study	Population	Intervention	Principal finding	Translation limitation
Nordio et al., 2017	Autoimmune thyroiditis with subclinical hypothyroidism	Myo-inositol 600 mg + selenium 83 µg/day; 6 months	Reported improvement in TSH and selected thyroid measures	Small study; formulation-specific; not the Thyro Complex dose [20].
Payer et al., 2022	Women with or at risk for subclinical hypothyroidism	Myo-inositol 600 mg + selenium 83 µg/day; 6 months	Improvement in selected clinical and biochemical measures	Prospective multicentre design; not definitive product evidence [19].
Pace et al., 2020	Hashimoto thyroiditis	Selenium with or without myo-inositol	Evaluated progression and thyroid-related biomarkers	Requires cautious interpretation and dose matching[22].
Yavari et al., 2024	Thyroid disorders	Systematic review/meta-analysis	TSH reduction and small T4 effect reported; T3/TPOAb effects inconsistent	Few studies and heterogeneity[11].
Zuhair et al., 2024	Autoimmune thyroiditis	Systematic review/meta-analysis	Combination favoured for TSH and TgAb; other outcomes not consistently significant	Approximately three comparative studies[24].
Stanchev et al., 2026	Hashimoto thyroiditis with subclinical hypothyroidism	Updated meta-analysis and trial sequential analysis	Greater TSH reduction versus selenium alone	Limited study count; further RCTs required[10].

5.3 Dose translation to Thyro Complex

The declared Thyro Complex serving provides myo-inositol 300 mg and selenium 30 µg. These quantities are approximately one-half and one-third, respectively, of the amounts used in the principal combination studies. If the labelled directions permit two units daily, the maximum daily exposure may be closer to the

investigated myo-inositol amount but would also double every other ingredient. The manuscript must therefore state the exact recommended serving and maximum daily intake. It is not scientifically appropriate to cite trials of 600 mg myo-inositol plus 83 µg selenium as direct evidence for one unit of the finished product.

6. Selenium as an individual ingredient

Selenium is essential to deiodinase activity and antioxidant defence. Modern syntheses provide a more nuanced conclusion than either "selenium is ineffective" or "selenium treats Hashimoto thyroiditis." A 2024 meta-analysis of 35 unique studies reported modest reductions in TSH among participants not receiving thyroid-hormone replacement and reductions in TPO antibodies and malondialdehyde, with variable certainty across outcomes. Earlier meta-analyses reached conflicting conclusions because of heterogeneous populations, selenium forms, baseline status, doses, durations and endpoints[13–16,25–27].

Antibody reduction is a surrogate outcome. It does not necessarily indicate symptom improvement, improved quality of life, reduced medication requirement or prevention of overt hypothyroidism. A recent randomised trial also found that selenium and placebo were similarly effective for quality-of-life improvement in hypothyroidism, reinforcing the importance of placebo effects and patient-centred outcomes[13,28].

Safety depends on total exposure from diet and other supplements. Selenomethionine has high bioavailability, but long-term excess can cause adverse effects. The product dossier should specify elemental selenium per unit and per maximum daily serving, chemical form, duration of use, overlap warnings and the rationale for the selected dose[29,30].

7. Iodine and nutritional adequacy

Iodine is an indispensable substrate for T4 and T3 synthesis. Deficiency can compromise hormone production, but indiscriminate iodine supplementation is not universally beneficial. Excess exposure can precipitate or aggravate thyroid dysfunction in susceptible individuals, particularly those with autoimmune thyroid disease or nodular thyroid tissue. Pregnancy, lactation, geographic iodine status, iodised-salt use, seafood or seaweed intake and concurrent supplements alter the interpretation of a 75 µg product dose[2,7,31,32].

The most defensible product wording is nutritional: "provides iodine, a nutrient involved in normal thyroid hormone synthesis." The formula should not be described as correcting iodine-deficiency disease unless deficiency is documented and treatment is clinically supervised.

8. Zinc, copper, magnesium, vitamin B12 and L-tyrosine

8.1 Zinc

Zinc is relevant to deiodinase regulation, hypothalamic–pituitary signalling, thyroid-hormone receptor structure and transcriptional processes. Systematic reviews suggest lower zinc concentrations in some hypothyroid populations, but the clinical supplementation literature remains limited and inconsistent. Benefit is most plausible in people with inadequate zinc status rather than in universally supplemented populations[33–35].

8.2 Copper and magnesium

Copper and magnesium participate in broad enzymatic, redox and energy-related functions. Meta-analytic evidence has not consistently shown clinically meaningful differences in copper or magnesium status across all hypothyroid populations. Their inclusion therefore supports general nutritional formulation logic, not a thyroid-treatment claim. Elemental quantities must be reported because the declared complex or salt weight is not equivalent to the elemental nutrient dose[33,36].

8.3 Vitamin B12

A 2023 systematic review and meta-analysis found lower vitamin B12 levels in overt hypothyroidism than in healthy controls, while associations were not consistent across all thyroid disorders. Correcting B12 deficiency can improve deficiency-related anaemia, neuropathy, fatigue or cognitive symptoms, but

vitamin B12 supplementation is not established as a thyroid-modifying treatment. The declared 1.8 µg is a nutritional amount and should not be represented as a treatment dose for established deficiency[37].

8.4 L-tyrosine

Tyrosine residues within thyroglobulin are iodinated during hormone synthesis. This biochemical role does not demonstrate that oral L-tyrosine supplementation increases T4 or T3, particularly because dietary protein normally supplies substantially greater amounts than the 75 mg declared in the formula. L-tyrosine should be discussed as a substrate rationale only.

9. Ashwagandha

Ashwagandha is the only declared botanical with a small randomised placebo-controlled trial directly examining subclinical hypothyroidism. The trial administered 600 mg/day of standardised root extract for eight weeks and reported changes in TSH, T3 and T4. The sample was small, treatment was short and independent replication is lacking. The Thyro Complex declaration of 50 mg is approximately one-twelfth of the investigated daily extract dose, and equivalence cannot be assessed without plant-part, extraction and withanolide specifications[38].

Safety signals are clinically relevant. Case reports describe thyrotoxicosis with supraventricular tachycardia and painless thyroiditis associated temporally with ashwagandha use, with improvement after discontinuation. Case reports cannot establish incidence, but they demonstrate that a botanical promoted for stress may influence thyroid physiology in susceptible individuals. The label and manuscript should recommend professional advice for diagnosed thyroid disease, hyperthyroidism, thyroid medication, pregnancy, autoimmune disease, liver disease or symptoms of excessive thyroid activity[39,40].

10. Other botanical ingredients

Botanical constituents can influence thyroid-related pathways through diverse biochemical mechanisms, but mechanistic interference or traditional use does not establish therapeutic benefit. Interpretation requires attention to botanical identity, plant part, extract composition, dose and exposure[59].

10.1 Bauhinia variegata

Recent reviews describe the phytochemistry and broad pharmacology of Bauhinia species and B. variegata, including traditional use and diverse preclinical activities. No adequately controlled 2016–2026 human thyroid trial was identified. The bark extract can therefore be presented only as a traditional and phytochemical research component. Authentication, extract ratio, solvent and marker fingerprinting are prerequisites for meaningful evaluation[41,42].

10.2 Commiphora wightii

Guggul and guggulsterones have longstanding traditional and preclinical associations with lipid metabolism and thyroid-related mechanisms. Contemporary literature is dominated by reviews, non-thyroid pharmacology and chemical-standardisation work rather than convincing human thyroid trials. Chemotype variability is important, and the formula should report total guggulsterones and the E-/Z-isomer profile[43,44].

10.3 Achyranthes aspera

Modern reviews summarise the phytochemistry and broad pharmacology of Achyranthes aspera. Thyroid-stimulatory claims are largely based on older animal work rather than contemporary human evidence. The declared “plant extract” should be replaced by an exact plant part and extract specification[45].

10.4 Coleus forskohlii

Forskolin activates adenylate cyclase and has mechanistic relevance to cyclic-AMP signalling. Modern Coleus forskohlii literature primarily addresses biotechnology, forskolin production and non-thyroid metabolic applications. The formula’s 30 mg root extract cannot be interpreted without a forskolin percentage. Mechanistic cAMP activity does not establish clinical thyroid efficacy[46].

10.5 Glycyrrhiza glabra

Liquorice contains pharmacologically active constituents, particularly glycyrrhizin and glycyrrhetinic acid. No credible direct clinical evidence establishes thyroid benefit. Conversely, safety evidence links glycyrrhizin exposure with sodium retention, potassium loss, hypertension, edema and arrhythmia susceptibility. The product specification must report glycyrrhizin content and intended duration, and warnings should address hypertension, renal or cardiovascular disease, diuretics, corticosteroids, digoxin and pregnancy[47–49].

10.6 Capsicum annuum

Capsicum and capsaicin have been investigated for thermogenesis, body weight and metabolic-syndrome outcomes. Meta-analyses report small changes in selected anthropometric or metabolic measures, but these data do not demonstrate increased thyroid-hormone synthesis or treatment of thyroid-related weight gain. Capsaicinoid standardisation and gastrointestinal tolerability should be reported[50–52].

Table 3: Evidence limitations and standardisation priorities for the botanical ingredients.

Botanical	Current thyroid-relevant evidence	Essential product specification
Bauhinia variegata	Traditional/preclinical; no adequate modern human thyroid trial	Bark identity, extract ratio, solvent and chromatographic markers
Commiphora wightii	Historical animal/mechanistic thyroid rationale; modern human evidence inadequate	Total guggulsterones, E-/Z-isomers, source sustainability and interactions
Achyranthes aspera	Older animal evidence and modern general pharmacology reviews	Exact plant part, extraction and validated chemical markers
Glycyrrhiza glabra	No thyroid efficacy; clinically relevant BP and potassium safety concerns	Glycyrrhizin/glycyrrhizic-acid content and duration limits
Coleus forskohlii	cAMP mechanism and non-thyroid applications	Forskolin percentage and extract ratio
Capsicum annuum	Indirect metabolic evidence; not thyroid efficacy	Total capsaicinoids and GI tolerability
Ashwagandha	One small thyroid RCT plus thyroid-related case reports	Root/leaf identity, withanolide content and thyroid safety plan

11. Chromium picolinate and metabolic positioning

Chromium picolinate has been investigated mainly in glucose metabolism, insulin resistance and polycystic ovary syndrome rather than thyroid disease. These findings cannot support a thyroid claim. A small pharmacokinetic study predating the principal review window found reduced levothyroxine exposure when chromium picolinate was coadministered; a later systematic review of levothyroxine interactions summarised an approximately 17% reduction in bioavailability in that experiment. This older evidence is retained because it is directly relevant to the likely user population[53,54].

People taking levothyroxine should therefore obtain professional advice regarding dose separation and thyroid-function monitoring. The product should not make a universal timing instruction unless that interval has been assessed by the manufacturer and is consistent with clinical guidance.

12. Dose relevance and evidence map

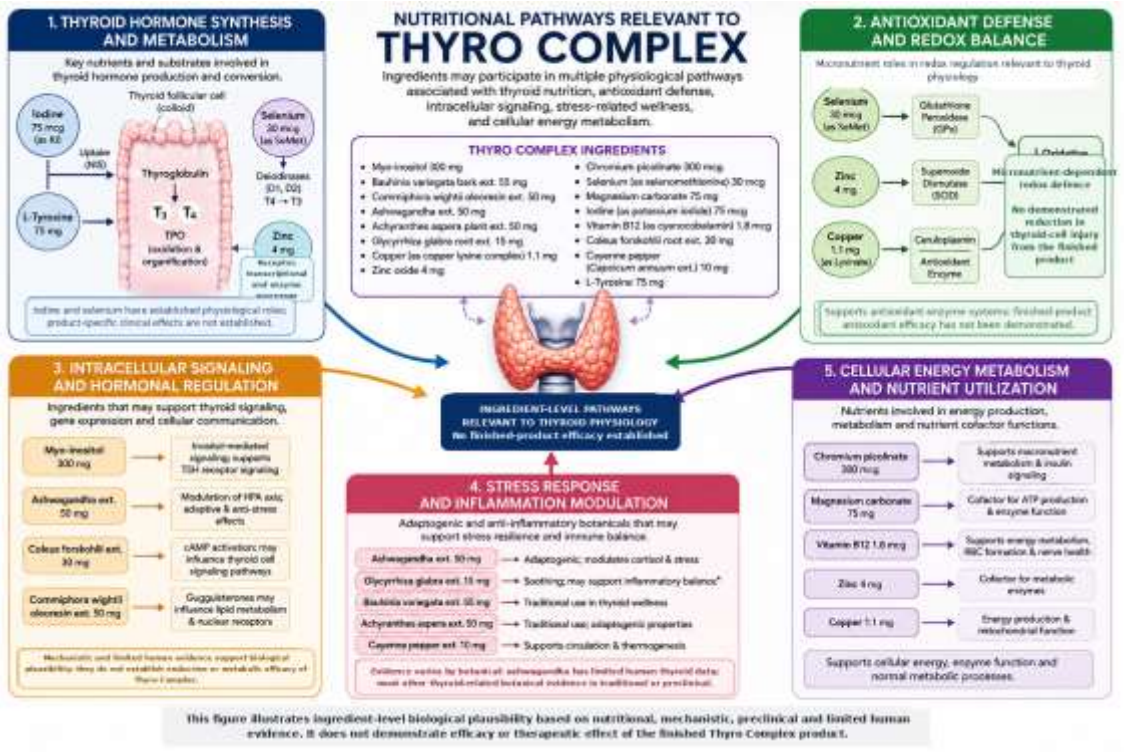

Figure 4. Nutritional pathways relevant to Thyro Complex. The figure maps ingredient-level physiological and mechanistic pathways related to thyroid nutrition, redox regulation, intracellular signalling, stress-related physiology and cellular energy metabolism. Iodine and selenium have established physiological roles, whereas evidence for several botanicals is limited, preclinical or traditional. Zinc is depicted in thyroid-related receptor, transcriptional and enzyme processes rather than as a direct thyroid-peroxidase cofactor. The figure represents biological plausibility and does not demonstrate efficacy of the finished product.

Table 4. Dose translation between the declared product and the available evidence.

Ingredient	Thyro Complex declaration	Research/clinical context	Interpretation
Myo-inositol	300 mg	Commonly 600 mg/day with selenium	Lower than principal trials; no finished-product equivalence
Selenium	30 µg	Often approximately 83 µg/day in combination trials; variable in selenium-only studies	Lower than principal combination studies; baseline status matters
Ashwagandha extract	50 mg	600 mg/day in the small subclinical-hypothyroidism RCT	Substantially lower; standardisation unknown
Iodine	75 µg	Nutritional requirements vary by life stage and total intake	Physiological dose; benefit depends on baseline iodine status

Zinc oxide	4 mg declared	Trials and nutritional studies generally report elemental zinc	Elemental zinc must be clarified
Magnesium carbonate	75 mg declared	No established thyroid-treatment dose	Elemental magnesium must be clarified
L-tyrosine	75 mg	No established thyroid-treatment dose	Small relative to dietary amino-acid intake
Botanical extracts	10–55 mg	Published extracts vary widely in ratios and marker content	Milligram comparison is invalid without extract standardisation

13. Safety, referral and medication interactions

13.1 High-risk groups

Professional advice should be obtained before use by individuals with diagnosed thyroid disease; thyroid nodules or goitre; levothyroxine, liothyronine or antithyroid medication; pregnancy or breastfeeding; cardiovascular, hepatic or renal disease; autoimmune disease; multiple medicines; or unexplained palpitations, tremor, sweating or weight loss. Children and adolescents should not use the product without explicit professional direction because age-specific safety and efficacy have not been established. These precautions are consistent with the broader clinical need to recognise thyroid overactivity and medication-related risk, and with evidence that herbal products can produce clinically relevant adverse events[60,61].

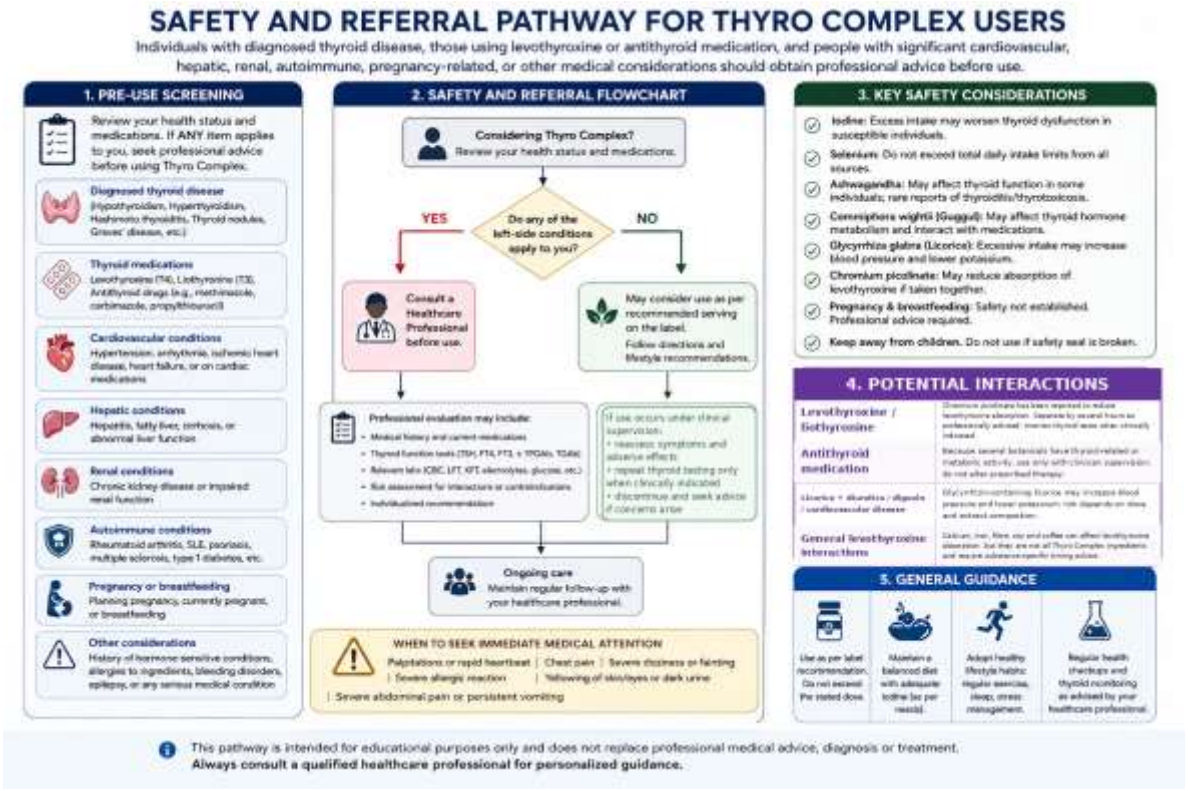

Fig 5: Safety and referral pathway for Thyro Complex users. Individuals with diagnosed thyroid disease, those using levothyroxine, liothyronine or antithyroid medication, and people with significant cardiovascular, hepatic, renal, autoimmune, pregnancy-related or other medical considerations should obtain professional advice before use. Interaction management is substance-specific: chromium picolinate has direct evidence of reduced levothyroxine absorption when coadministered, whereas common levothyroxine interactions with calcium, iron, fibre, soy and coffee should not be misrepresented as Thyro Complex ingredients.

13.2 Levothyroxine and laboratory considerations

Food, coffee, soy, fibre, calcium, iron, gastric-acid suppression and gastrointestinal disease can reduce or destabilise levothyroxine absorption. Chromium picolinate is specifically relevant to this formulation and has been reported to reduce levothyroxine exposure when coadministered; separation by several hours should therefore be considered under professional guidance rather than applying one universal timing rule to every ingredient. Abnormal thyroid tests in a supplement user may reflect adherence, administration timing, malabsorption, interacting products or assay interference rather than progression of thyroid disease[54–58].

Table 5: Major safety and interaction considerations.

Context	Concern	Recommended manuscript/label position
Levothyroxine/liothyronine	Chromium picolinate has been reported to reduce levothyroxine absorption; common food/mineral/GI interactions also affect levothyroxine but are not all Thyro Complex ingredients.	Separate chromium by several hours as professionally advised; use substance-specific timing guidance and monitor TSH/fT4 when clinically indicated.
Antithyroid medicines	Ingredients that influence thyroid physiology may complicate control	Use only under clinician supervision
Diuretics/corticosteroids/digoxin	Glycyrrhizin-containing liquorice may promote sodium retention and potassium loss; risk depends on exposure and extract standardisation.	Avoid or monitor according to glycyrrhizin exposure, cardiovascular/renal status and concomitant medicines.
Antidiabetic therapy	Chromium and other ingredients may affect glucose-related outcomes	Monitor glucose and medication response where appropriate
Anticoagulant/antiplatelet therapy	Botanical interactions cannot be excluded	Medication review before use
Thyroid laboratory testing	Biotin is not declared, but users may take separate products; illness and medication timing affect tests	Record all supplements and medicines before testing
Pregnancy/breastfeeding	Iodine requirements differ; botanical safety is not established	Professional advice required; avoid unsupported routine use

14. Evidence-to-claim translation

Claims must be proportionate to the evidence and specific to the final product. Mechanistic evidence can support a rationale section in an academic paper, but it cannot support language implying demonstrated clinical benefit. Ingredient-level human evidence may justify discussing a research hypothesis, yet differences in dose, chemical form, botanical standardisation, excipients and co-ingredients limit direct extrapolation.

Table 6. Evidence-to-claim translation for Thyro Complex.

Claim category	Examples	Evidence requirement

Potentially supportable after label and regulatory review	Provides iodine and selenium involved in normal thyroid-related physiology; supports nutritional intake; contains nutrients involved in normal energy metabolism	Nutrient content must be analytically verified; wording must comply with applicable regulation
Requires a finished-product controlled trial	Reduces fatigue; improves focus or cognitive clarity; improves thyroid-related quality of life; improves TSH, fT4, fT3 or thyroid antibodies; supports hormonal balance	Use validated outcomes, prespecified endpoints and the intended commercial formulation
Should be avoided	Treats hypothyroidism or hyperthyroidism; reverses Hashimoto thyroiditis; shrinks thyroid nodules; replaces levothyroxine; prevents thyroid disease; guarantees weight loss	Disease-treatment and medication-replacement claims are unsupported and potentially unsafe

The current commercial messaging should also be harmonised. The approved label, manuscript and website should use one dosage-form term, one serving recommendation and one legal manufacturer/marketer identity. Statements such as "clinically validated formula" should not be used unless the final product itself has been evaluated in an appropriate clinical study.

15. Product quality and standardisation requirements

A product-centred review is strengthened by transparent quality documentation. Without batch-specific analytical data, the reader cannot determine whether the reviewed preparation is reproducible or comparable to published extracts. The following information should be incorporated into the technical dossier and summarised in the manuscript when available.

Table 7: Recommended quality and product-development documentation.

Domain	Minimum documentation
Identity	Botanical authority, plant part, macroscopic/microscopic authentication, DNA barcoding where appropriate, voucher specimen
Extract definition	Drug-to-extract ratio, native-extract amount, solvent, carrier and residual solvent limits
Marker assay	Withanolides, guggulsterones, forskolin, glycyrrhizin and capsaicinoids; validated HPLC/UPLC or equivalent methods
Nutrient assay	Elemental iodine, selenium, zinc, copper, chromium and magnesium per unit and maximum daily serving
Contaminants	Heavy metals, pesticides, aflatoxins, microbial limits and adulterant screening
Finished-product performance	Content uniformity, disintegration/dissolution, moisture, hardness/friability as applicable
Stability	Accelerated and real-time stability, packaging compatibility, shelf-life justification and storage conditions

Traceability	Supplier qualification, batch numbers, certificate of analysis and change-control procedure
Safety governance	Adverse-event reporting, complaint investigation, interaction review and periodic literature surveillance

16. Proposed clinical-development programme

Figure 6. Proposed clinical-development pathway for Thyro Complex. Scientifically supportable claims should be developed through ingredient authentication, quality-control testing, formulation and stability assessment, safety evaluation, a pilot safety-and-feasibility study, an exploratory randomised controlled trial and, where stronger claims are sought, confirmatory randomised controlled evaluation of the finished product. Pilot findings are hypothesis-generating and should not be treated as claim-substantiating efficacy evidence.

16.1 Pilot investigation

A pilot study should first establish feasibility, tolerability, adherence, administration timing and the suitability of the selected population. It should not combine euthyroid individuals with fatigue, untreated subclinical hypothyroidism, autoimmune thyroiditis and stable levothyroxine users without prespecified stratification. These groups have different baseline risks and different expected responses.

16.2 Exploratory randomised controlled trial

After feasibility and safety are established, an exploratory randomised controlled trial should evaluate the exact intended commercial formulation at the labelled serving against a matched placebo. The population and primary outcome must be prespecified, the trial should be prospectively registered, and participants with diagnosed thyroid disease should continue standard care under clinical supervision. Validated patient-

reported outcomes and thyroid biochemical measures should be selected only when justified by the target population and intended claim.

Table 8: Recommended specifications.

Design domain	Recommended specification
Population	Adults with one clearly defined phenotype, such as stable subclinical hypothyroidism or euthyroid adults with documented nutrient insufficiency; avoid mixing populations without stratification
Intervention	Exact commercial Thyro Complex formulation, verified batch and labelled daily serving
Comparator	Matched placebo plus unchanged standard care
Duration	Pilot: 8–12 weeks; confirmatory study: commonly 6–12 months depending on primary endpoint
Primary endpoint	One prespecified clinically meaningful endpoint: validated fatigue/quality-of-life score or a defined biochemical endpoint
Secondary endpoints	TSH, fT4, fT3, TPOAb, TgAb, sleep, stress, cognition, waist circumference and metabolic biomarkers where justified
Safety	Adverse events, blood pressure, pulse, liver and renal function, electrolytes, thyroid-overactivity symptoms and medication changes
Baseline stratification	Thyroid medication, TSH category, antibodies, selenium status, iodine status, zinc and B12 status
Analysis	Intention-to-treat analysis, multiplicity control, missing-data plan and prespecified subgroup analysis
Reporting	CONSORT, registered protocol, funding/conflict disclosure and access to a product specification dossier

16.3 Confirmatory randomised controlled trial

If the exploratory trial produces a credible product-specific signal, a larger confirmatory trial should test prespecified clinically meaningful outcomes using the final commercial formulation, an appropriate comparator and a prespecified statistical analysis plan. Multicentre recruitment, adequate duration, safety monitoring, and prespecified subgroup analyses should be considered based on the intended claim. A confirmatory trial, rather than a pilot study, provides the stronger basis for product-specific claim substantiation.

16.4 Post-market safety and real-world evaluation

Following commercial use, adverse-event surveillance, complaint review, batch-quality monitoring and appropriately designed real-world studies can refine the safety profile and assess longer-term product performance. Real-world observations are complementary to randomised evidence and should not be presented as equivalent proof of efficacy.

17. Limitations

This review is limited by the absence of a completed systematic review workflow, incomplete access to subscription database export records, and the lack of a clinical trial evaluating the finished product. Several formula botanicals have little or no contemporary human thyroid evidence. Botanical doses are difficult to interpret because marker content and extract ratios were not supplied. The evidence for myo-inositol–selenium and ashwagandha was generated using preparations and doses that differ from the declared

formulation. Safety conclusions are also constrained until the maximum daily serving, elemental nutrient content, excipients and botanical marker levels are verified.

The literature itself has limitations. Many thyroid-supplement studies are small, short, focused on surrogate biochemical outcomes and conducted in selected populations. Reductions in antibody titres do not necessarily translate into symptom improvement, prevention of overt hypothyroidism or reduced medication requirements. Observational associations between nutrient status and thyroid disease are vulnerable to reverse causation and confounding.

18. Conclusion

Thyro Complex has a plausible ingredient-level rationale involving thyroid nutrition, redox regulation, intracellular signalling and cellular energy metabolism. The most relevant human evidence concerns myo-inositol combined with selenium in selected patients with autoimmune thyroiditis and subclinical hypothyroidism. Selenium has a substantial but heterogeneous clinical literature. Ashwagandha has one small direct thyroid trial and clinically relevant safety reports. Iodine, zinc and vitamin B12 are primarily relevant to physiological adequacy or deficiency. Evidence for the remaining botanical and mineral components is indirect, mechanistic, traditional or non-thyroid-specific.

These findings do not establish that Thyro Complex treats thyroid disease or improves fatigue, cognition, weight, thyroid antibodies or hormone concentrations. The declared serving also differs materially from the doses used in principal ingredient studies. The appropriate current position is therefore a multicomponent nutraceutical requiring strict product standardisation, interaction safeguards and conservative claim language. A randomised controlled trial of the final formulation is necessary before product-specific efficacy claims can be considered.

Declarations

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References

1. Shulhai AM, Rotondo R, Petraroli M, et al. The role of nutrition on thyroid function. Nutrients. 2024; 16(15):2496. doi:10.3390/nu16152496.
2. Rayman MP. Multiple nutritional factors and thyroid disease, with particular reference to autoimmune thyroid disease. Proc Nutr Soc. 2019; 78(1):34–44. doi:10.1017/S0029665118001192.

3. Ragusa F, Fallahi P, Elia G, et al. Hashimotos' thyroiditis: epidemiology, pathogenesis, clinic and therapy. Best Pract Res Clin Endocrinol Metab. 2019; 33(6):101367. doi:10.1016/j.beem.2019.101367.

4. Ralli M, Angeletti D, Fiore M, et al. Hashimoto's thyroiditis: an update on pathogenic mechanisms, diagnostic protocols, therapeutic strategies, and potential malignant transformation. Autoimmun Rev. 2020; 19(10):102649. doi:10.1016/j.autrev.2020.102649.

5. Larsen D, Singh S, Brito M. Thyroid, diet, and alternative approaches. J Clin Endocrinol Metab. 2022; 107(11):2973–2981. doi:10.1210/clinem/dgac473.

6. Hu S, Rayman MP. Multiple nutritional factors and the risk of Hashimoto's thyroiditis. Thyroid. 2017; 27(5):597–610. doi:10.1089/thy.2016.0635.

7. O'Kane SM, Mulhern MS, Pourshahidi LK, Strain JJ, Yeates AJ. Micronutrients, iodine status and concentrations of thyroid hormones: a systematic review. Nutr Rev. 2018; 76(6):418–431. doi:10.1093/nutrit/nuy008.

8. Winther KH, Rayman MP, Bonnema SJ, Hegedüs L. Selenium in thyroid disorders—essential knowledge for clinicians. Nat Rev Endocrinol. 2020; 16(3):165–176. doi:10.1038/s41574-019-0311-6.

9. Duntas LH, Brenta G. A renewed focus on the association between thyroid hormones and lipid metabolism. Front Endocrinol (Lausanne). 2018; 9:511. doi:10.3389/fendo.2018.00511.

10. Stanchev P, Kraeva M, Petrov PP, et al. Myo-inositol plus selenium vs. selenium alone in Hashimoto's thyroiditis with subclinical hypothyroidism: a systematic review and updated meta-analysis with trial sequential analysis. J Clin Med. 2026; 15(9):3179. doi:10.3390/jcm15093179.

11. Yavari M, Akbari M, Ramezani Ahmadi A, Siavash Dastjerdi M, Hashemi MS. Investigating the effect of combined use of selenium and myo-inositol supplements on thyroid function and autoimmune characteristics in thyroid disorders: a systematic review and meta-analysis. Expert Rev Endocrinol Metab. 2024; 19(3):269–277. doi:10.1080/17446651.2023.2295487.

12. Zuhair V, Sheikh AT, Shafi N, et al. Role of supplementation with selenium and myo-inositol versus selenium alone in patients of autoimmune thyroiditis: a systematic review and meta-analysis. Clin Med Insights Endocrinol Diabetes. 2024; 17:11795514241300998. doi:10.1177/11795514241300998.

13. Huwiler VV, Maissen-Abgottspon S, Stanga Z, et al. Selenium supplementation in patients with Hashimoto thyroiditis: a systematic review and meta-analysis of randomized clinical trials. Thyroid. 2024; 34(3):295–313. doi:10.1089/thy.2023.0556.

14. Wichman JEM, Winther KH, Bonnema SJ, Hegedüs L. Selenium supplementation significantly reduces thyroid autoantibody levels in patients with chronic autoimmune thyroiditis: a systematic review and meta-analysis. Thyroid. 2016; 26(12):1681–1692. doi:10.1089/thy.2016.0256.

15. Winther KH, Wichman JEM, Bonnema SJ, Hegedüs L. Insufficient documentation for clinical efficacy of selenium supplementation in chronic autoimmune thyroiditis, based on a systematic review and meta-analysis. Endocrine. 2017; 55(2):376–385. doi:10.1007/s12020-016-1098-z.

16. Kong XQ, Qiu GY, Yang ZB, Tan ZX, Quan XQ. Clinical efficacy of selenium supplementation in patients with Hashimoto thyroiditis: a systematic review and meta-analysis. Medicine (Baltimore). 2023; 102(20):e33791. doi:10.1097/MD.0000000000033791.

17. Paparo SR, Ferrari SM, Patrizio A, et al. Myoinositol in autoimmune thyroiditis. Front Endocrinol (Lausanne). 2022; 13:930756. doi:10.3389/fendo.2022.930756.

18. Payer J, Jackuliak P, Kužma M, Džupon M, Vaňuga P. Supplementation with myo-inositol and selenium improves the clinical conditions and biochemical features of women with or at risk for

subclinical hypothyroidism. Front Endocrinol (Lausanne). 2022; 13:1067029. doi:10.3389/fendo.2022.1067029.

19. Nordio M, Basciani S. Treatment with myo-inositol and selenium ensures euthyroidism in patients with autoimmune thyroiditis. Int J Endocrinol. 2017; 2017:2549491. doi:10.1155/2017/2549491.
20. Nordio M, Basciani S. Myo-inositol plus selenium supplementation restores euthyroid state in Hashimoto's patients with subclinical hypothyroidism. Eur Rev Med Pharmacol Sci. 2017; 21(2 Suppl):51–59. PMID:28724185.
21. Pace C, Tumino D, Russo M, et al. Role of selenium and myo-inositol supplementation on autoimmune thyroiditis progression. Endocr J. 2020; 67(11):1093–1098. doi:10.1507/endocrj.EJ20-0062.
22. Benvenga S, Nordio M, Laganà AS, Unfer V. The role of inositol in thyroid physiology and in subclinical hypothyroidism management. Front Endocrinol (Lausanne). 2021; 12:662582. doi:10.3389/fendo.2021.662582.
23. Samuel CG, Singh P, Abdullahi H, Ibrahim I. Unlocking the therapeutic potential: selenium and myo-inositol supplementation in thyroid disorders—efficacy and future directions. Life (Basel). 2025; 15(10):1500. doi:10.3390/life15101500.
24. Peng B, Wang W, Gu Q, Wang P, Teng W, Shan Z. Effects of different supplements on Hashimoto's thyroiditis: a systematic review and network meta-analysis. Front Endocrinol (Lausanne). 2024; 15:1445878. doi:10.3389/fendo.2024.1445878.
25. Hu Y, Feng W, Chen H, et al. Effect of selenium on thyroid autoimmunity and regulatory T cells in patients with Hashimoto's thyroiditis: a prospective randomized-controlled trial. Clin Transl Sci. 2021; 14(4):1390–1402. doi:10.1111/cts.12993.
26. Tian X, Li N, Su R, Dai C, Zhang R. Selenium supplementation may decrease thyroid peroxidase antibody titer via reducing oxidative stress in euthyroid patients with autoimmune thyroiditis. Int J Endocrinol. 2020; 2020: 9210572. doi:10.1155/2020/9210572.
27. Wu Q, Wang Y, Chen P, et al. Increased incidence of Hashimoto thyroiditis in selenium deficiency: a prospective 6-year cohort study. J Clin Endocrinol Metab. 2022; 107(9):e3603–e3611. doi:10.1210/clinem/dgac410.
28. Larsen CB, Winther KH, Cramon PK, et al. Selenium supplementation and placebo are equally effective in improving quality of life in patients with hypothyroidism. Eur Thyroid J. 2024; 13(1):e230175. doi:10.1530/ETJ-23-0175.
29. EFSA Panel on Nutrition, Novel Foods and Food Allergens (NDA). Scientific opinion on the tolerable upper intake level for selenium. EFSA J. 2023; 21(1):7704. doi:10.2903/j.efsa.2023.7704.
30. Bodnar M, Szczyglowska M, Konieczka P, Namiesnik J. Methods of selenium supplementation: bioavailability and determination of selenium compounds. Crit Rev Food Sci Nutr. 2016; 56(1):36–55. doi:10.1080/10408398.2012.709550.
31. Bath SC. Thyroid function and iodine intake: global recommendations and relevant dietary trends. Nat Rev Endocrinol. 2024; 20:474–486. doi:10.1038/s41574-024-00983-z.
32. Alexander EK, Pearce EN, Brent GA, et al. 2017 Guidelines of the American Thyroid Association for the diagnosis and management of thyroid disease during pregnancy and the postpartum. Thyroid. 2017; 27(3):315–389. doi:10.1089/thy.2016.0457.
33. Talebi S, Ghaedi E, Sadeghi E, et al. Trace element status and hypothyroidism: a systematic review and meta-analysis. Biol Trace Elem Res. 2020; 197(1):1–14. doi:10.1007/s12011-019-01963-5.

34. Beserra JB, Morais JBS, Severo JS, et al. Relation between zinc and thyroid hormones in humans: a systematic review. Biol Trace Elem Res. 2021; 199(11):4092–4100. doi:10.1007/s12011-020-02562-5.

35. Severo JS, Morais JBS, de Freitas TEC, et al. The role of zinc in thyroid hormones metabolism. Int J Vitam Nutr Res. 2019; 89(1-2):80–88. doi:10.1024/0300-9831/a000262.

36. Zhou Q, Xue S, Zhang L, Chen G. Trace elements and the thyroid. Front Endocrinol (Lausanne). 2022; 13: 904889. doi:10.3389/fendo.2022.904889.

37. Benites-Zapata VA, Ignacio-Cconchoy FL, Ulloque-Badaracco JR, et al. Vitamin B12 levels in thyroid disorders: a systematic review and meta-analysis. Front Endocrinol (Lausanne). 2023; 14: 1070592. doi:10.3389/fendo.2023.1070592.

38. 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; 24(3):243–248. doi:10.1089/acm.2017.0183.

39. Hayashi M, Hamada H, Azuma SI, Hayashi K. Painless thyroiditis by Withania somnifera (Ashwagandha). Cureus. 2024; 16(3):e55352. doi:10.7759/cureus.55352.

40. Kamal HI, Patel K, Brdak A, Heffernan J, Ahmad N. Ashwagandha as a unique cause of thyrotoxicosis presenting with supraventricular tachycardia. Cureus. 2022; 14(3):e23494. doi:10.7759/cureus.23494.

41. Gudavalli D, Pandey K, Ede VG, Sable D, Ghagare AS, Kate AS. Phytochemistry and pharmacological activities of five species of Bauhinia genus: a review. Fitoterapia. 2024; 174:105830. doi:10.1016/j.fitote.2024.105830.

42. Kamal Y, Khan T, Haq I, et al. Phytochemical and biological attributes of Bauhinia variegata L. (Caesalpiniaceae). Braz J Biol. 2022; 82:e257990. doi:10.1590/1519-6984.257990.

43. Kunnumakkara AB, Banik K, Bordoloi D, et al. Googling the Guggul (Commiphora and Boswellia) for prevention of chronic diseases. Front Pharmacol. 2018; 9:686. doi:10.3389/fphar.2018.00686.

44. Haque I, Mukhopadhyay K. Occurrence of guggulsterone content based chemotypes in Commiphora wightii. Curr Bot. 2024; 15:41–46. doi:10.25081/cb.2024.v15.8785.

45. He X, Wang X, Fang J, et al. The genus Achyranthes: a review on traditional uses, phytochemistry, and pharmacological activities. J Ethnopharmacol. 2017; 203:260–278. doi:10.1016/j.jep.2017.03.035.

46. Mitra M, Gantait S, Mandal N. Coleus forskohlii: advancements and prospects of in vitro biotechnology. Appl Microbiol Biotechnol. 2020; 104(6):2359–2371. doi:10.1007/s00253-020-10377-6.

47. Nazari S, Rameshrad M, Hosseinzadeh H. Toxicological effects of Glycyrrhiza glabra (licorice): a review. Phytother Res. 2017; 31(11):1635–1650. doi:10.1002/ptr.5893.

48. Wu T, Yang J, Xia J, Sun G. Effects of licorice functional components intakes on blood pressure: a systematic review with meta-analysis and network toxicology. Nutrients. 2024; 16(21):3768. doi:10.3390/nu16213768.

49. Deutch MR, Grimm D, Wehland M, Infanger M, Krüger M. Bioactive candy: effects of licorice on the cardiovascular system. Foods. 2019;8(10):495. doi:10.3390/foods8100495.

50. Jang HH, Lee J, Lee SH, Lee YM. Effects of Capsicum annuum supplementation on the components of metabolic syndrome: a systematic review and meta-analysis. Sci Rep. 2020; 10(1):20912. doi:10.1038/s41598-020-77983-2.

51. Zhang W, Zhang Q, Wang L, et al. The effects of capsaicin intake on weight loss among overweight and obese subjects: a systematic review and meta-analysis of randomised controlled trials. Br J Nutr. 2023; 130(9):1645–1656. doi:10.1017/S0007114523000697.

52. Zsiborás C, Mátics R, Hegyi P, et al. Capsaicin and capsiate could be appropriate agents for treatment of obesity: a meta-analysis of human studies. Crit Rev Food Sci Nutr. 2018; 58(9):1419–1427. doi:10.1080/10408398.2016.1262324.

53. John-Kalarickal J, Pearlman G, Carlson HE. New medications which decrease levothyroxine absorption. Thyroid. 2007; 17(8):763–765. doi:10.1089/thy.2007.0060.

54. Wiesner A, Gajewska D, Paśko P. Levothyroxine interactions with food and dietary supplements—a systematic review. Pharmaceuticals (Basel). 2021; 14(3):206. doi:10.3390/ph14030206.

55. Skelin M, Lucijanić T, Amidžić Klarić D, et al. Factors affecting gastrointestinal absorption of levothyroxine: a review. Clin Ther. 2017; 39(2):378–403. doi:10.1016/j.clinthera.2017.01.005.

56. Guzman-Prado Y, Vita R, Samson O. Concomitant use of levothyroxine and proton pump inhibitors in patients with primary hypothyroidism: a systematic review. J Gen Intern Med. 2021; 36(6):1726–1733. doi:10.1007/s11606-020-06403-y.

57. Ferrari SM, Patrizio A, Mazzi V, et al. Lactose intolerance and levothyroxine malabsorption: a review of the literature and report of a series of patients treated with liquid L-T4 without lactose. Front Endocrinol (Lausanne). 2024; 15: 1386510. doi:10.3389/fendo.2024.1386510.

58. Liu H, Lu M, Hu J, et al. Medications and food interfering with the bioavailability of levothyroxine: a systematic review. Ther Clin Risk Manag. 2023; 19:503–523. doi:10.2147/TCRM.S414460.

59. Di Dalmazi G, Giuliani C. Plant constituents and thyroid: a revision of the main phytochemicals that interfere with thyroid function. Food Chem Toxicol. 2021; 152:112158. doi:10.1016/j.fct.2021.112158.

60. Lee JY, Jun SA, Hong SS, Ahn YC, Lee DS, Son CG. Systematic review of adverse effects from herbal drugs reported in randomized controlled trials. Phytother Res. 2016; 30(9):1412–1419. doi:10.1002/ptr.5647.

61. Ross DS, Burch HB, Cooper DS, et al. 2016 American Thyroid Association guidelines for diagnosis and management of hyperthyroidism and other causes of thyrotoxicosis. Thyroid. 2016; 26(10):1343–1421. doi:10.1089/thy.2016.0229.

62. Osowiecka K, Myszkowska-Ryciak J. The influence of nutritional intervention in the treatment of Hashimoto's thyroiditis—a systematic review. Nutrients. 2023; 15(4):1041. doi:10.3390/nu15041041.