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Review Article

Natural Thyrotropic Agents in Hypothyroidism: An Evidence-Based Review of Ayurvedic and Traditional Medicinal Plants

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ABSTRACT

Hypothyroidism is one of the most common endocrine conditions in the world, levothyroxine is the primary drug used for treatment, but a significant number of biochemically euthyroid patients still experience symptoms fatigue, low mood, and cognitive complaints. There are many medicinal plants in Ayurveda and allied traditional medicine system that have been used traditionally for diseases similar to thyroid problems include galaganda. In this review ,we address the mechanism of action, wider therapeutic potential and thyroid hormone status of nine such plants which include Ashwagandha (Withania somnifera), Kalonji (Nigella sativa), Haridra (curcuma longa), Varuna (crataeva nurvala), Costus pictus, Kanchanara (bauhinia variegata), Guggul (Commiphora Mukul), Apamarga(achyranthes aspera) and Shigura(moringa oleifera),fenugreek (Trigonella foenum graecum L.), Tulsi (Ocimum tenuiflorum L.), Amla (Emblica officinalis gaertn.)with specific focus on their effects on thyroid hormone status. The evidence suggests that most of these plants increase the serum levels of T3 and T4 and decrease TSH in chemically induced hypothyroid animal models, presumably by stimulating thyroid peroxidase activity, by antioxidant protection of thyroid follicular cells and by modulating the hypothalamic-pituitary-thyroid axis. However , the data on clinical trials are available only for a few plants such as Withania somnifera and Nigella sativa.

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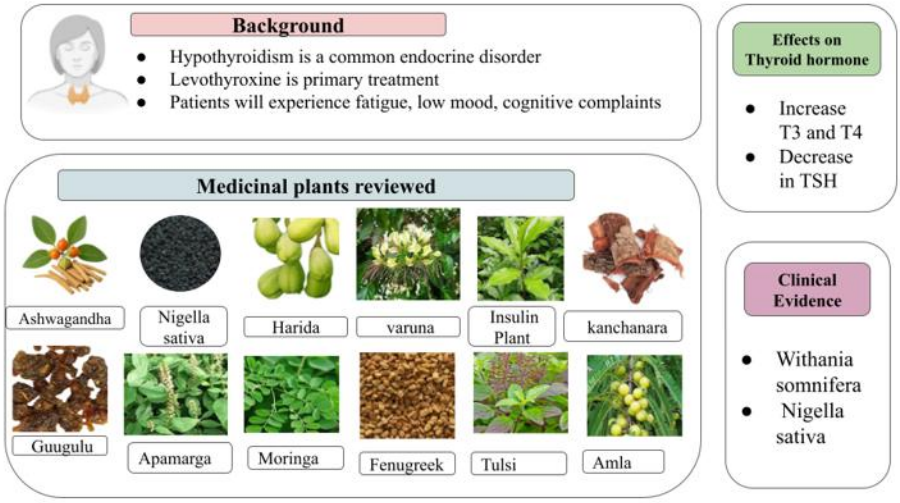

Figure 1: Graphical Overview of Ayurvedic and Traditional Medicinal Plant for the Management of Hypothyroidism and Clinical Evidence

INTRODUCTION

Hypothyroidism is caused by a lack of production or action of thyroid hormones (T3 and T4), causes weight gain, cold intolerance, cognitive slowing, constipation and fatigue. Hypothyroidism and its autoimmune etiology are among the most prevalent endocrine disorders in the world, and prevalence and treatment patterns differ depending on the iodine status, age, sex and region of the world [1]. Recent studies linking presence of the symptoms to serumT3 levels in biochemically women treated with levothyroxine support this lack of efficacy [2] and in particular mood disturbance have reported to persist in a significant number of treated women even if serum TSH is normalized [3]. There is no direct mention of thyroid gland in classical ayurvedic texts but galaganda(swelling disorder) mentioned by charaka and sushurta can be correlated with goiter and thyroid dysfunction [4]. A great amount of research (most of which was carried out in the chemically induced hypothyroid models of rats – propylthiouracil (PTU) has investigated the thyroid stimulating and thyroprotective properties of many Ayurvedic plants over the last 20 year [5] and special narratives reviews are now starting to synthesize this Ayurvedic research base for clinical readers [6]. In addition to these botanical investigation, researchers have increasingly turned to correlating the profiles of symptoms associated with hypothyroidism with the ayurvedic concept which classifies metabolic disharmony as either Mandagni and Dhatvagni Vishama[7]. These working models help to create a more comprehensive picture and understanding of patient care, which integrates the correction of hormonal parameters with the reconciling of metabolic homeostasis and balancing of kapha and Vata dosha [8]. The interest in these traditional interventions is increased, but clinical data is still limited and there is currently no strong experimental or pharmacological support to fully validate the efficacy of these traditional interventions in human being [9]. This change in perspective toward an integrative model recognize the levothyroxine supports the hormonal imbalance of the tissues and accumulation of Ama that accompany the disease state [10].

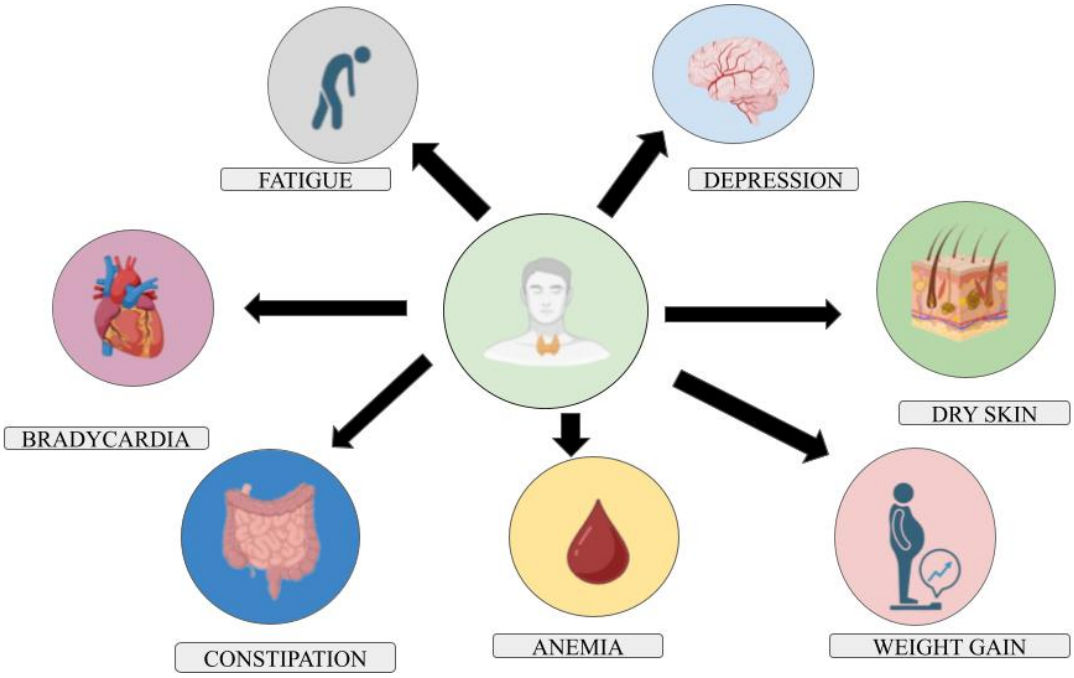

Figure 2: clinical outcome of hypothyroidism

1.1 Methodology

The 4 data base searches(PubMed,Scopus, Springer LINL and Google Scholar) resulted in 654 records. Of these,145 records were removed pior to screening(110 due to duplicate record, 20 due to automation tool, 15 due to other reaons) and 500 records were subject to screening.

Of these 220 were excluded and 280 were requested for retrieval, 15 were not retrived, 256 were retrived and assessed for eligibility for full text. Of all the reports, 158 were discarded due to variety of reasons.

Based on this multi-stage screening process 107 studies were found that meet the inclusion criteria, thus being included inqualitative synthesis. The selection process was done in accordance with PRISMA 2020 guidelines to guarantee transparency and reproducibility

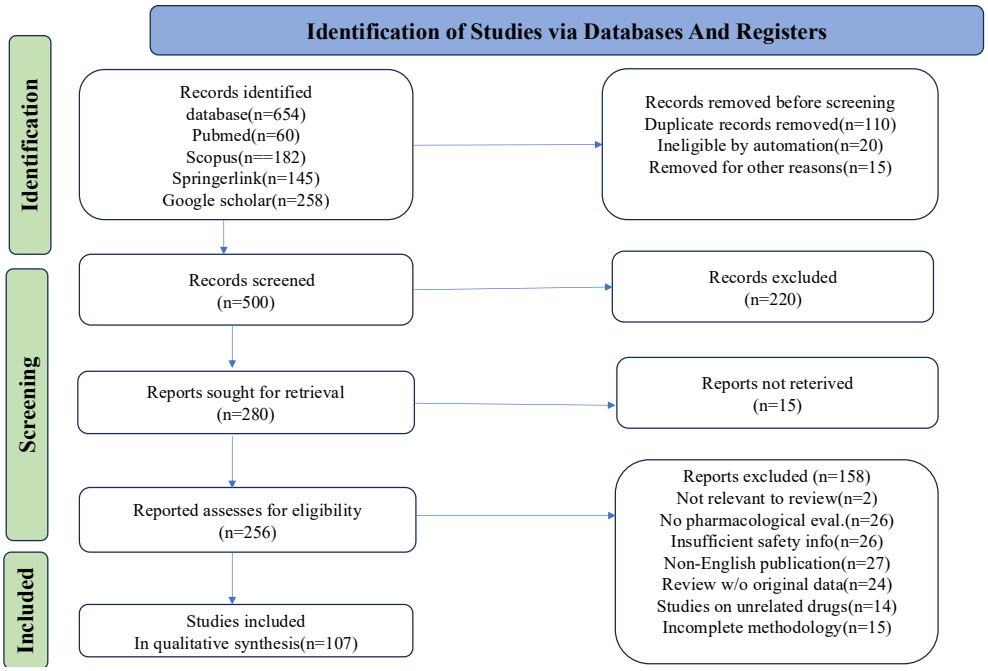

Figure 3: Flow chart of PRISMA analysis

1.2 Classification of hypothyroidism

Depending on the site of the defect in the hormone axis, hypothyroidism can be categorized as:

1. Primary Hypothyroidism: It is the most common type, caused by thyroid gland itself dysfunction, and occurs in about 99%, the reason for this type dysfunction within the thyroid gland itself [11].
2. Secondary Hypothyroidism: This is when no overactive thyroid gland is caused by deficiency of thyroid-stimulating hormone (TSH) [11].
3. Tertiary Hypothyroidism: It is a form that comes from an area of the brain called the hypothalamus which is unable to make enough of a hormone called thyrotropin-releasing-hormone (TRH) [11].

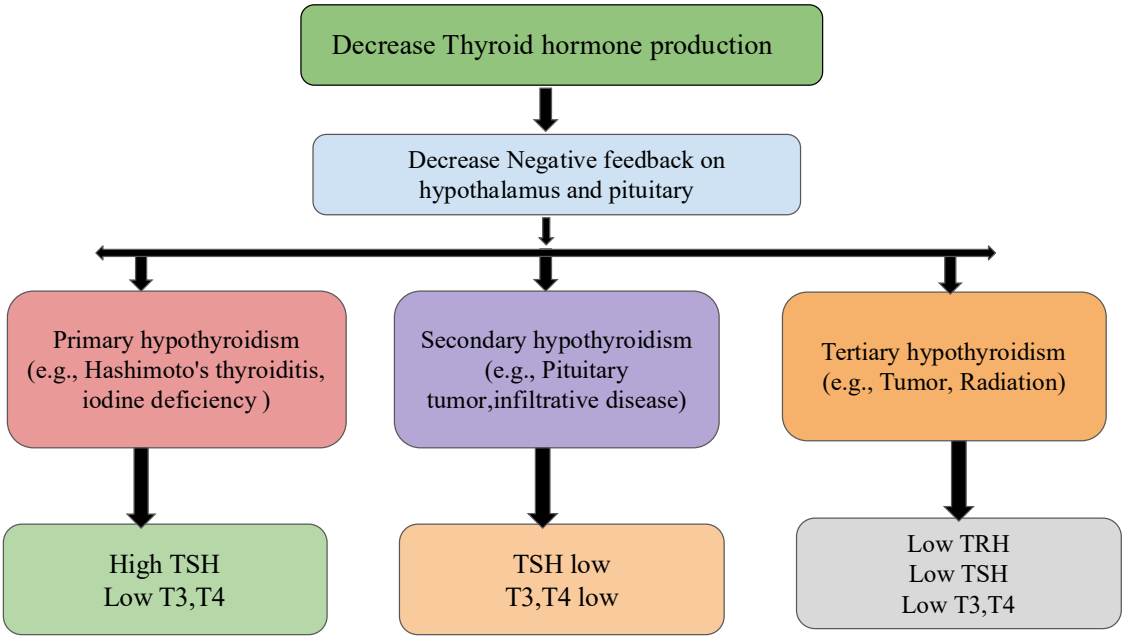

Figure 4: Classification of Hypothyroidism based on the site of dysfunction and associated hormonal profiles

1.3 Epidemiology

Hypothyroidism is one of the most prevalent endocrine disorders globally, but the incidence of the disease varies widely between different geographical regions and the different diagnostic criteria. In the European population, overt hypothyroidism occurs in 0.2-0.5% and in the USA, it occurs in 0.3-3.7% of the general population, the range is due to variability in the definition and population studied. The incidence of spontaneous hypothyroidism is approximately 3.5-5.0 cases per 1000 women per year, and is heavily skewed to women. From the survey data of US NHANES, the overall prevalence of the hypothyroidism was found to be 4.6% in the sampled population [12].

This burden seems to be much heavier in developing countries, especially in the South Asian region. A study done in India revealed and the prevalence of hypothyroidism was high, with about 10% of all adults affected, and was significantly associated with female gender and age [13].

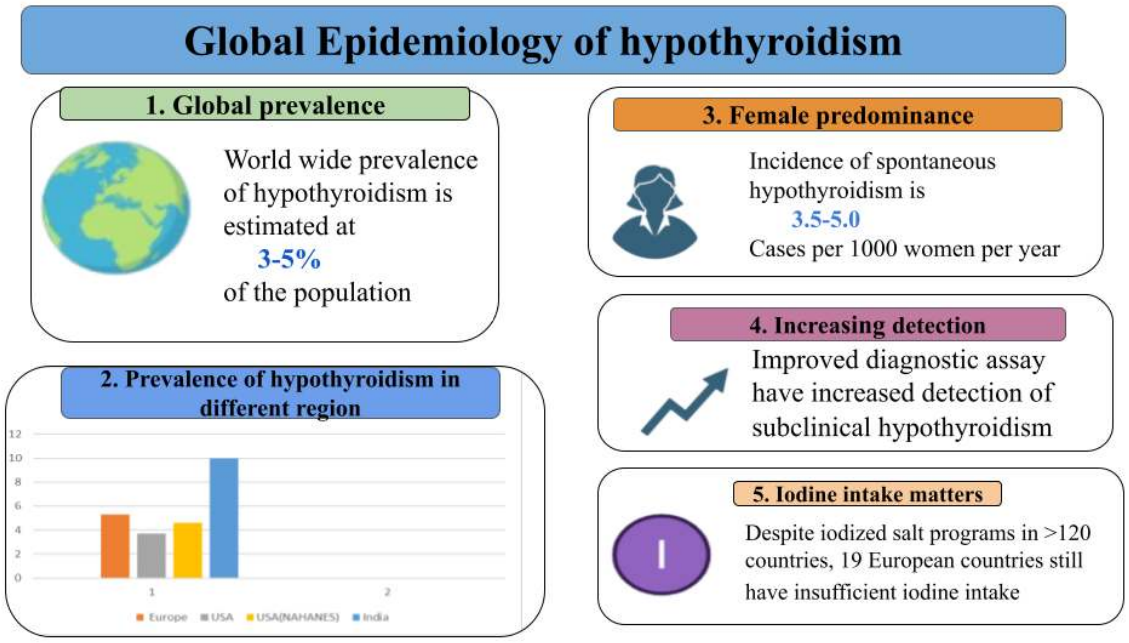

Figure 5: Global epidemiology of Hypothyroidism

1.4 Pathophysiology and the Hypothalamic-Pituitary-Thyroid Axis

Thyroid hormone synthesis requires uptake of iodide by the sodium-iodide symporter (NIS), oxidation and organification of iodide by thyroid peroxidase (TPO) and coupling of iodothyronines to form T4 and T3 [14]. This process is controlled by the hypothalamic-pituitary-thyroid (HPT) axis, the hypothalamus releases thyrotropin-releasing hormone (TSH), TSH acts on the thyroid to stimulate hormone production, and T3 and T4 exert negative feedback at the hypothalamus and pituitary [15]. Primary hypothyroidism is diagnosed when TSH is elevated and free T4 is low or low-normal, while subclinical hypothyroidism is a milder from disturbance of this feedback loop in which TSH is elevated but free T4 is still within normal range [16]]. The most prevalent etiology in iodine-sufficient-region is chronic autoimmune thyroiditis, which is characterized by infiltration of the gland by lymphocytes, meanwhile the causative antibodies, thyroglobulin (TgAb) and thyroid peroxidase (TPOAb), are diagnostic markers are also thought to cause progressive glandular destruction [17]. Given its high prevalence and significant impact on systemic metabolic homeostasis, thud endocrine disorder necessitates precise clinical management to prevent long-term complications associated with chronic thyroid hormone deficiency. Thyroid hormones have systemic because they regulate body temperature and basal metabolic rate [18]. This regulation is disputed in these areas, in part by autoantibodies, and that is why it is important to understand the molecular mechanisms that trigger the injury of the thyrocytes [19]. The inability to make adequate amounts of hormone may then be the result of the progressive destruction of glandular tissue by autoantibodies [20]. Clinical signs of this deficiency include lethargy and bradycardia as well as impairment of thermogenesis, which demonstrates the important physiological role of adequate levels of circulating thyroid hormones [21]. Moreover, the clinical difference between subclinical overt hypothyroidism is crucial in determining treatment and can help identify a higher risk for

cardiovascular problems like atrial fibrillation and coronary artery disease by people with hypothyroidism who hadn't received any treatment [22]. Epidemiological studies have shown that hypothyroidism, in its overt from at least, is a significant public heath problem with a prevalence of around 5% in western population, and is far more common in women than in men, with some studies indicating a ten-fold difference in prevalence rate [23,24].

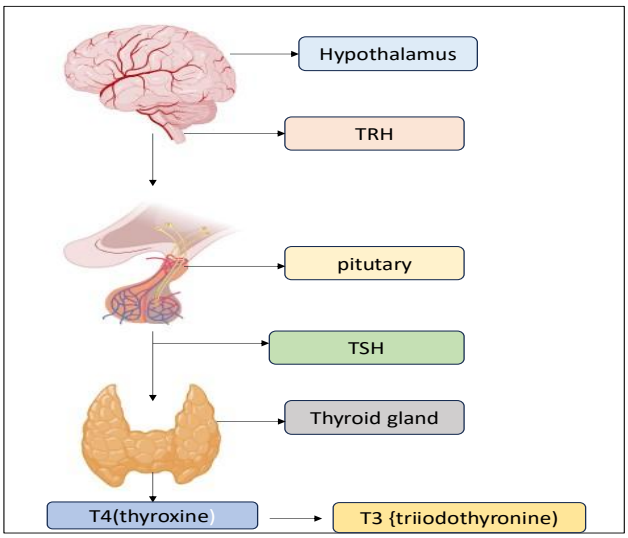

Figure 6: Hypothalamic-Pituitary-Thyroid (HPT) Axis Regulating Thyroid Hormone Secretion

1.5 Conventional Management and its Limitation

For decades the standard treatment for hypothyroidism has been levothyroxine, a synthetic T4, because it has oral bioavailability, a long half-life (allowing for once daily), and produces satisfactory result in most people with hypothyroidism, by lowering TSH and easing the symptoms. At the population level, however, problems with adherence, absorption of levothyroxine, and interacting comorbidities and drugs and issues with dose titration all lead to a population of 35-60% of levothyroxine users who are outside the target range of TSH at any given time [25]. Levothyroxine treated women also report more anxiety and depressive symptoms compared to euthyroid women, indicating a discrepancy between the biochemistry and the women's perception of quality of life [26]. Such continuing worries about post-thyroidectomy effects have promoted research on other treatments, such as T4/T3 combination or the administration of desiccated thyroid extract [27]. Although the early studies found the combination therapies to be not superior to monotherapy, the studies have been criticized for methodological flaws, including enrolment of patients without a clearly defined T3 deficiency and the use of suboptimal tools to evaluate patient-reported outcomes [28]. Moreover, approximately 10-15% of the patients still experience a persistent neurocognitive dysfunction despite normalization of the serum TSH level, indicating that a "one-size-fits-all" approach to serum TSH normalization may not meet the complex physiological needs of all patients. There is evidence that about one-fifth of patients treated with levothyroxine alone may not reach physiologic serum levels pf T3, which raises the question of whether levothyroxine is adequate to

support peripheral tissue deiodinase activity [29]. Levothyroxine monotherapy-treated patients often have increased free T4 and decreased serum T3 levels than healthy euthyroid subjects, which may reflect unphysiological T4 to T3 secretion ratio [30]. However, this shift in T3/T4 ratio could lead to suboptimal thyroid status in periphery because there may be differences between the levels of TSH and the level of thyroid activity in specific peripheral tissues [31,32].

1.6 Rationale for Phyto Therapeutic Approaches

Botanical with a long history of use in ethnomedicine, particularly Ayurveda and Unani medicine have been used for beneficial effects on thyroid function, physiological mechanisms by which some of these plants may act on the HPT axis have been demonstrated by pharmacological screening, and include direct stimulation of thyroidal iodine uptake and hormone secretion, modulation of peripheral deiodinase activity and antioxidant protection of thyrocytes against oxidative injury, which has been implicated in autoimmune thyroiditis. [33]. Other botanical remedies, however, like Melissa officinals or Lycopus europaeus are used for their peripheral hormone modulating properties and are found to have the capacity to lower serum TSH and circulating thyroid hormones, similar to the effects of exogenous thyroxine, against conditions of excessive metabolic demand [34]. The different pharmacological properties are often related to specific secondary metabolites such as flavonoids and polyphenols, which may interact selectively with thyroid hormone receptors, or affect the rate of conversion of thyroxine to triiodothyronine [35]. Besides, recent studies on polyherbal formulations, which include a combination of various bioactive plant extracts, seek to fill these treatment gaps by trying to restore homeostasis by multi-faceted approach, with a synergistic effect in hormone regulation [36]. These natural adjuvants show potential multi-target activity but it is important that they are used as supporting agents in addition to currently available clinical treatments in severe pathologies [37]. In clinical studies going forward, attention should be given to strict standardization and safety profiles of these traditional remedies to support their use as effective and safe ingredients in endocrine treatment [38].

However, comprehensive analytical frameworks such as high throughput screening and controlled human trials remain important to define the exact pharmacodynamic of these extracts and to tackle current regulatory issues related to consistency and quality control [39]. Also, the limited number of large-scale, quality clinical trails require transition towards evidence-based validation to reconcile the traditional ethnomedical information with contemporary endocrine requirements [40].

2. Scope and Approach of this Review

The present narrative-integrative review encompasses the peer-reviewed preclinical (animal and vitro/in vivo) studies, and systemic review/meta-analyses dealing with the medicinal plant, botanical extract and different categories of related nutraceuticals (e.g., selenium) and thyroid hormone status. Plants were selected based on (a) mechanistic pr pharmacological data were specifically related to thyroid hormone synthesis, secretion, and peripheral metabolism of thyroid hormone (b) availability of at least preliminary human data or substantial preclinical literature on the subject and (c) significance of the plant in ethnomedical and nutraceutical literature for thyroid support. It is not a comprehensive systemic review to give the clinical, the researcher and the informed patient a comprehensive picture of what is known and unknown about each of the

botanicals, but is intended to give an evidence-graded overview of this. More specific, the difference between mechanistic plausibility, animal efficacy and proven clinical benefit in human beings is discussed.

3.Overview of plants reviewed

Table 1: Botanical Profile of Medicinal Plants Reviewed for the Management of Hypothyroidism

Common name	Botanical name	Family	Part used
Ashwagandha	Withania somnifera(L.)	Solanaceae	Root
Kalonji	Nigella sativa L.	ranunculaceae	Seed(oil)
Harida	Curcuma longa L.	zingiberaceae	Rhizome
Varuna	Crateva nurvala buch. Ham	capparidaceae	Leaf
Insulin plant	Costus pictus D. Don	Costaceae	Leaf
kanchanara	Bauhimina variegata L.	Fabaceae	Stem bark
Guggulu	Commiphora mukul	Burseraceae	Oleo-gum resin
Apamarga	Achyranthes aspera L.	Amarnthacea	Leaf
Moringa	Moringa oleifera Lam.	Moringaece	Leave, seed, bark
Fenugreek	Trigonella foenum-graecum L.	fabaceae	Seeds
Tulsi	Ocimum tenuiflorum L.	lamiaceae	Leaves
Amla	Emblica officinalis gaertn.	euphorbiaceae	Fruit

4. Overview of the Pharmacological Mechanism and Thyroid-Modulating Effects of Selected Ayurvedic and Traditional Medicinal Plants

Table 2: Summary of the principle mechanism of action, reported pharmacological activities, Thyroid specific effects for selected Ayurvedic and traditional medicinal plants evaluated for the management of Hypothyroidism.

Sr no.	Name	Main mechanism of action	Reported therapeutic effects	Action in hypothyroidism	Reference
1.	Withania somnifera(L.)	Modulate the HPT axis, reduce cortisol, and support thyroid hormone synthesis.	Anti-stress, anxiolytic, antioxidant, reduce anxiety, morning cortisol	Increased T4 in rodents, improved TSH in subclinical hypothyroidism and restored thyroid hormones in diabetic hypothyroid models.	[7],[8],[9],[10], [41],[42],[43], [44],[45]

2.	Nigella sativa	Reduce oxidative stress and inflammation, regulate COX-2 and improve thyroid hormone metabolism.	Used for hyperlipidemia, hypertension, type 2 diabetes, antioxidant, anti-inflammatory	Reduced TSH and anti-TPO antibodies, increased T3, improves metabolic markers, and prevent cognitive impairment in hypothyroid rats.	[46],[47],[48],[49], [50],[51],[52], [53],[54]
3.	Curcuma longa L.	Proposed TRH receptor agonist with antioxidant, anti-inflammatory	antioxidant, anti-inflammatory, anti-cancer, neuroprotective, reproductive hormone axes	Restored antioxidant enzyme, reduced lipid peroxidation, showed potential thyroid receptor modulation in computational studies	[55],[56],[57],[58], [59]
4.	Crataeva nurvala buch. Ham.	Enhances T4-T3 conversion via deiodinase activity	Used for prostatic disorder, also antihyper-cholesterolemic, anti-diabetic	Dose-dependently increased free T4, lowered TSH, maintained euthyroid status	[60],[61],[62],[63], [64],
5.	Costus pictus D. don	Restore thyroid hormones while reducing cholesterol, LDL, oxidative stress	Known as the insulin plant for anti-diabetic use, anti-microbial, antioxidant, hepatoprotective	Restored thyroid hormones, reduced TSH, improved lipid profile	[65],[66],[67],[68],
6.	Bauhinia variegata	Improves thyroid weight, radioiodine uptake	Used for glandualar swelling, lymphadenopathy, skin disorder	Improved thyroid weight, radioiodine uptake, histology and cholesterol levels.	[69] [70],[71],[72],
7.	Commiphora mukul	Stimulate thyroid activity, increase T3, and improve lipid metabolism	Classical resin for hyperlipidema, obesity, arthritis, lipid lowering	Increased T3, thyroid weight and iodine uptake, thyroid stimulatory effects may be more pronounced in females	[73], [74],[75],[76], [77],[78],[79], [80],
8.	Achyranthes aspera L.	Increase circulating T4/T3 and reduces hepatic lipid peroxidation	Diuretic, anti-inflammatory, wound-healing, antiarthritic, purgative, antimalarial	Increased T3/T4, reduced hepatic lipid peroxidation, and enhanced metabolic activity.	[81] [82],[83],[84],
9.	Moringa oleifera Lam.	Lower TSH, increase T3/T4	Antioxidant, anti-inflammatory, hypolipidemic, used as dietary supplement	Increased T3/T4 , reduced TSH, and computational studies	[85] [86],[87], [88], [89],[90], [91],

				support possible thyroid receptor activation	
10.	Trigonella foenum-graecum	Enhance insulin sensitivity, reduce oxidative stress, protect pancreatic β cell , improve lipid metabolism	Anti-diabetic, hypolipidemic, cardioprotective, hepatoprotective	Suppress HPT axis, improve thyroid hormone secretion, low TSH	[92],[93],[94],[95],[96],[97]
11.	Ocimum tenuiflorum	Anti-oxidant, anti-inflammatory, adaptogenic effects	Anti-diabetic, hepatoprotective, radioprotective	Reduce T4 but increase T3, decrease TSH,	[98],[99],[100],[101],
12.	Emblica officinals	Hydrolysable tannins, phenolics, flavonoids provide potent anti-oxidant and free-radical scavenging activity	Anti-oxidant, anti-inflammatory, hepatoprotective, cardioprotective, antidiabetic, hypolipidemic, and immunomodulatory	Reduce T3/T4 level, alleviates oxidative stress	[102],[103],[104],[106],

5. Clinical evidence

1. **Ashwagandha** (Withania somnifera) root extract:

Trial design: prospective, randomized, double-blind, placebo-controlled pilot study. (duration:8 weeks)

Patients: 50 patients in subclinical Hypothyroidism (TSH 4.5-10μIU/L) were divided into treatment (n=25) and the placebo (n=25) groups and 4 were lost to follow up before the second visit.

Outcomes: Serum TSH ($p<0.001$), T3 ($p=0.0031$) and T4 ($p=0.0096$) got significantly better than placebo, as thyroid indices normalized and mild, temporary adverse effects were seen in 8% of those studied.

Serum TSH ($p<0.0031$), T3($P=0.0031$) and T4 ($p=0.0096$) got significantly better than placebo, as thyroid indices normalized and mild, temporary adverse effects were seen in 8% of those studied [10].

2. **Nigella sativa (black seed):**

Trial design: Randomized, double-blinded, placebo-controlled trial (duration: 8 weeks)

Patients. Forty patients (ages 22-50) with Hashimoto's thyroiditis were randomly assigned to intervention or a control group, excluding the 3 patients in the intervention group who complained of itching/nausea and 4 patients from the control group who withdrew, the data presented here are 31 patients in each group.

Outcomes: In the nigella sativa group, a significantly reduction in body weight and BMI, significant increase in serum T3, and significant decrease in serum TSH and anti-TPO and serum VECF were observed, but in the placebo group, no change in body weight, BMI, serum TSH, anti-TPO, serum VEGF were observed [47].

6. CONCLUSION

Several Ayurvedic medicinal plants were found to possess preclinical and, in a few cases, early clinical evidence of their ability to modulate thyroid hormone status by their direct thyrotropic and deiodinase activity and through their antioxidant and hypothalamic-pituitary-axis activity . There is more translational evidence for Ashwagandha and Nigella sativa (randomized controlled trials in subclinical hypothyroidism and Hashimoto's thyroiditis, respectively , and the remaining plants are backed primarily by animal pharmacology and broader (non-thyroid-specific) pharmacological review literature which shows plausible and related mechanisms. These botanicals have the potential to be used as complementary therapies to levothyroxine and other conventional treatments for hypothyroidism, especially in low-resource areas, but before any can be recommended, they must be rigorously and standardly tested in humans to provide evidence-based treatment and as with any medication, continue to be monitored for adverse reactions, including iatrogenic thyrotoxicosis.

7. future prospects

Future research priorities include well designed randomized, double-blind, placebo-controlled clinical trials of plants (such as Varuna, Costus pictus, Kanchanra, Guggulu, Apamarga, and Moringa oleifera) indicated only by animal studies, especially receptors studies and in silico docking of plants, including Ashwagandha, onto the TRβ1 and TRH receptor, to determine whether plants have a receptor-mediated, enzyme-medicated or purely antioxidant effect, clinical trails in autoimmune (Hashimoto's) hypothyroidism to assess whether Ashwagandha in combination with levothyroxine reduces the amount of the drug needed or if it improves the residual symptoms experienced by most treated patients, studies of the classical preparation (such as this will eventually bring to a close the methodological gaps and gaps in understanding between traditional botanical formulations and evidence-based integrative approaches which supplement modern endocrinological treatment. Systemic isolation and characterization of active phytochemical constituents continue to be fundamental to the discovery of multi-targeted potential of nature interventions in the current therapeutic scenario.

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