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DISCORDANT THYROID FUNCTION TESTS: A NARRATIVE LITERATURE REVIEW

Youssef El Marraki [1, *], Ikhlass Lakssir [1], Inass Chaari [1], Ayoub Idrissi [1], Hamza El Jadi [1, 2] and Azzelarab Meftah [1, 2]

[1] Endocrinology, Diabetes and Metabolism, Avicenne Military Hospital, Marrakech, Morocco.
[2] Biosciences and Health Laboratory, Faculty of Medicine and Pharmacy, Cadi Ayyad University, Marrakech, Morocco.
* Corresponding Author

ORCID Details
Y. EL MARRAKI: https://orcid.org/0009-0002-5312-4114

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ABSTRACT

Discordant thyroid function tests can arise when laboratory findings conflict with the clinical presentation, when thyroid hormone and thyrotropin concentrations appear physiologically incongruent, or when measurements differ between analytical platforms. These situations require explanation before a biochemical abnormality becomes the basis for treatment. This narrative review examines a predefined reference collection, integrating physiological and analytical guidance with a retrospective diagnostic referral series, a real-world platform-change study, and an autoantibody interference case report. The evidence supports a staged assessment beginning with clinical context, treatment history, sampling conditions, and the suitability of reference intervals. Non-thyroidal illness, pregnancy, levothyroxine administration, and other medications can produce apparently contradictory results through different mechanisms. Laboratory investigation should then address method-dependent interference, including heterophile antibodies, macro-TSH, thyroid hormone autoantibodies, and abnormal hormone binding. Persistent, genuinely elevated thyroid hormone concentrations with non-suppressed thyrotropin warrant specialist evaluation for resistance to thyroid hormone beta and thyrotropin-secreting pituitary adenoma. Pituitary imaging and dynamic tests require interpretation together, because incidental lesions, initially negative imaging, and overlapping responses can mislead. The available evidence favours close clinical–laboratory collaboration, while its dependence on selected observational data limits universal diagnostic thresholds. A structured approach can help distinguish treatment-related and analytical explanations from uncommon endocrine disorders without assuming that every discordant result represents disease.

Keywords: Thyroid function tests; Thyrotropin; Free thyroxine; Assay interference; Resistance to thyroid hormone; Thyrotropinoma

1 INTRODUCTION

Discordance is a reason to reassess thyroid function tests, rather than a diagnosis in itself. It may describe disagreement between biochemical findings and the clinical examination, an unexpected relationship between thyroid-stimulating

hormone (TSH) and free thyroxine (FT4) or free triiodothyronine (FT3), or inconsistent measurements across laboratory methods. These forms overlap but need not share an explanation. For example, elevated FT4 with non-suppressed TSH may reflect treatment, interference, altered hormone binding, or a disorder of thyroid hormone action or TSH secretion. A result becomes interpretable only when its physiological and analytical context is established. [1, 2, 3]

The consequences of premature interpretation are illustrated by two complementary clinical studies. Giovanella and colleagues investigated unexpectedly high FT4 results after a change of testing platform and found that medication use explained many cases with an identifiable cause. Campi and colleagues described referral patients in whom assay interference was mistaken for rare endocrine disease, while genuine disease was sometimes dismissed as artefact. Together, these observations support a sequence that checks common contextual explanations and measurement validity while preserving the possibility of an uncommon diagnosis. This review examines that sequence and the limits of the evidence supporting it. [4, 5]

2 SCOPE AND APPROACH

This narrative review was based on a predefined reference collection. Sixteen PDFs represented fourteen distinct publications after duplicate copies of two publications were consolidated. One paper addressing discordant thyroid nodule biopsy guidelines was excluded because it concerned structural evaluation rather than biochemical thyroid function. The remaining publications were assessed for relevance to interpretation, analytical interference, diagnostic differentiation, and testing practice. The synthesis gives particular attention to directly reported clinical investigations and uses reviews and textbook chapters for explanatory context. No external literature search or quantitative pooling was performed. Findings cited within a reviewed source were treated as secondary evidence; their original publications were not assumed to have been independently verified. The diagnostic sequence presented below is a clinical synthesis, not a newly validated prediction rule or a systematic review.

2.1 Interpreting the biochemical pattern

TSH is useful in primary thyroid disease because relatively small changes in circulating thyroid hormones can produce much larger changes in pituitary output. However, this relationship presupposes an interpretable feedback system and an appropriate temporal context. Pituitary disease, recent treatment, intercurrent illness, and medications can invalidate a simple reading of TSH as an independent measure of thyroid status. Population reference intervals also encompass wider variation than an individual's usual hormone concentrations. A normal TSH therefore cannot resolve every disagreement between symptoms and hormone measurements, and an isolated marginal abnormality does not identify its cause. [1, 2, 3]

Table 1: Biochemical patterns and initial interpretive priorities

Pattern	Explanations to consider	Initial priority
Elevated FT4 with non-suppressed TSH	Levothyroxine exposure, amiodarone, binding or assay effects; persistent genuine elevation may indicate RTHβ or TSHoma	Review medication and sampling history, then investigate measurement validity before specialist differentiation. [1, 4]
Low FT4 with normal or low TSH	Central hypothyroidism, non-thyroidal illness, TSH-suppressing drugs, or method-dependent error	Assess pituitary and illness context; a normal TSH does not exclude central disease. [3]
Elevated TSH with normal FT4	Subclinical hypothyroidism, recovery from illness, levothyroxine-related disequilibrium, or TSH interference	Establish persistence and context; discuss interference when the result remains unexplained. [1, 2]
Low TSH with normal FT4	Subclinical hyperthyroidism, T3 excess, treatment-related TSH suppression, or drug/illness effects	Measure T3 when indicated and establish the temporal and treatment context. [1, 3]
Abnormal total hormones with less disturbed free hormones and TSH	Altered binding-protein concentration or affinity	Assess binding and method effects rather than interpreting total hormone concentration alone. [1, 3]

Abbreviations: RTHβ, resistance to thyroid hormone beta; TSHoma, TSH-secreting pituitary adenoma. The listed explanations are not exhaustive and the patterns do not establish diagnoses.

Total and free hormone concentrations answer different questions. Changes in thyroxine-binding globulin (TBG) can alter total T4 and T3 without corresponding thyroid dysfunction. Free hormone assays aim to account for protein

binding, but their estimates remain vulnerable to altered binding equilibria, circulating competitors, and assay design. The fact that a result is labelled “free” does not make it independent of binding-related interference. Comparing total and free measurements can consequently be useful, provided neither is treated as an infallible reference. [1, 3]

The pattern should narrow the investigation without prematurely fixing the diagnosis. Low FT4 with an inappropriately normal or low TSH requires consideration of central hypothyroidism, although illness, medications, and method-dependent error can produce similar findings. Elevated TSH with normal FT4 may represent subclinical primary hypothyroidism, recovery from illness, treatment-related disequilibrium, or TSH assay interference. These are competing explanations to be assessed using persistence, history, and laboratory investigation. Table 1 summarises the principal interpretive problems. [1, 3]

3 CLINICAL AND TREATMENT CONTEXT

3.1 Temporal changes and intercurrent illness

The relationship between TSH and thyroid hormones is less reliable during transitions than during stable thyroid function. TSH can remain suppressed after circulating hormones have normalised during treatment of hyperthyroidism; conversely, TSH elevation may persist after correction of prolonged hypothyroidism. Interpreting either situation as treatment failure risks inappropriate dose adjustment. Previous results, treatment changes, and the direction of change are therefore integral to interpretation. Repeat sampling should answer a defined question about persistence or recovery rather than merely reproduce an unexplained abnormality. [1, 3]

Non-thyroidal illness introduces both physiological and analytical uncertainty. Reduced T3, variable FT4, and low or normal TSH can occur, while recovery may be accompanied by transient TSH elevation. Different free hormone methods may yield divergent results in the same ill patient. Premawardhana's review also emphasises that these changes are not confined to intensive care. Acute illness therefore weakens the specificity of an abnormal result for intrinsic thyroid disease, but it does not exclude coexisting thyroid pathology. The indication for testing and the plan for reassessment remain important. [1, 3, 6]

Testing during acute illness is most informative when prompted by relevant clinical findings, previous thyroid disease, medication exposure, or unexplained tachydysrhythmia, rather than included indiscriminately in an admission panel. When immediate clarification is unnecessary, reassessment after the acute disturbance has resolved can help distinguish transient changes from persistent dysfunction. This approach should remain conditional on clinical assessment; an illness-related explanation should not become a reason to disregard convincing evidence of thyroid or pituitary disease. [3, 6]

3.2 Pregnancy and reference intervals

Pregnancy changes both the concentrations being measured and their interpretation. Increased TBG alters total hormone concentrations, while human chorionic gonadotropin influences thyroid stimulation and TSH, particularly early in gestation. Free hormone estimates also show method-dependent differences. Applying non-pregnant reference intervals can therefore create apparent discordance. The reviewed sources support interpretation using pregnancy-appropriate intervals, with attention to the analytical method for FT4, rather than transferring a single numerical threshold between laboratories. [1, 2, 3]

3.3 Levothyroxine and other medications

Levothyroxine exposure deserves early attention when FT4 is elevated but TSH is not suppressed. The interval between tablet ingestion and sampling affects measured T4, and intermittent ingestion may produce normal or elevated hormone concentrations despite persistently raised TSH. Impaired absorption and interactions with food or medication can create further instability. The history should establish the prescribed regimen, actual administration, recent changes, and concomitant substances before the pattern is attributed to resistance or pituitary disease. [1, 3]

In Giovanella and colleagues' exploratory study, 306 patients with available clinical information had elevated FT4 with normal TSH; an explanation was identified in 263. Thyroxine therapy accounted for 191 of those 263 cases (72.6%), amiodarone for 38 (14.4%), other drugs for 20 (7.6%), and analytical interference for 14 (5.3%). These percentages describe the subgroup with an identified explanation, not all discordant patients or the general testing population. The observational attribution supports careful medication review but does not establish a universally applicable distribution of causes. [4]

Medication effects should be separated mechanistically. Amiodarone can alter hormone metabolism and produce a higher T4 relative to T3, as well as cause thyroid disease. Dopamine and glucocorticoids can suppress TSH secretion.

Heparin can instead generate an apparent free hormone elevation through lipase-mediated production of non-esterified fatty acids during sample storage or incubation, displacing hormone from binding proteins. This effect can occur with low-molecular-weight heparin and may also affect equilibrium dialysis. Recognising these distinctions directs the laboratory investigation more effectively than treating every drug-associated result as immunoassay interference. [1, 3]

4 ESTABLISHING MEASUREMENT VALIDITY

4.1 TSH assay interference

In a two-site TSH immunoassay, endogenous antibodies can bridge assay antibodies and produce an exaggerated signal, or impede binding and produce a falsely low result. Macro-TSH, an immunoglobulin-associated form of TSH, can also cause disproportionate measured TSH elevation. The biochemical pattern may resemble genuine thyroid dysfunction, so the clinical mismatch is a prompt for laboratory discussion rather than proof of interference. Conversely, apparently plausible results do not establish that interference is absent. [1, 2]

Laboratory approaches include comparison with a different assay, dilution studies for TSH, and antibody-removal techniques such as polyethylene glycol precipitation or protein G/A treatment. Garg and colleagues also describe gel filtration chromatography in investigating macro-TSH. These procedures provide different kinds of evidence and should be selected and interpreted by the laboratory. A change after treatment or a non-linear dilution response can support interference; identifying the responsible mechanism requires interpretation alongside the assay characteristics and clinical findings. [1, 7]

Supplement exposure and antibodies directed against assay components broaden this differential. Razvi and colleagues and the platform-change study identify biotin, anti-streptavidin antibodies, and anti-ruthenium antibodies among potential sources of interference. In the Campi series, biotin use was identified in a patient who had undergone investigation and treatment for suspected TSHoma before normal results were obtained with a different assay design. The case supports asking explicitly about non-prescription products and discussing the analytical method with the laboratory. It does not establish that every discordant result in a person taking biotin is caused by that supplement. [2, 4, 5]

4.2 Thyroid hormone autoantibodies and binding abnormalities

Thyroid hormone autoantibodies can interfere with free or total hormone measurements by interacting with reagents used in competitive assays. Srichomkwun and colleagues investigated a 21-year-old woman with autoimmune thyroid disease and elevated iodothyronine measurements that initially raised concern for RTHβ. Results differed across platforms, FT4 measured by equilibrium dialysis was within its reference interval, and binding of labelled hormones to an immunoglobulin fraction supported the presence of thyroid hormone autoantibodies. This is direct mechanistic evidence from a single patient, not an estimate of the frequency of interference among patients with thyroid autoimmunity. [8]

The case also challenges a simple classification of assays as susceptible or resistant to interference. Although two-step methods can reduce contact between patient serum components and the labelled tracer, one two-step platform still returned elevated results in this investigation. Assay architecture, antibody properties, and the particular interaction matter. Agreement between two methods is therefore less reassuring when both share a relevant vulnerability, and a two-step result should be regarded as additional evidence rather than an absolute guarantee of validity. [1, 8]

Abnormal hormone binding provides another explanation for a euthyroid clinical state with unusual measurements. Quantitative changes in binding proteins primarily affect total hormone concentrations, whereas variant proteins, including albumin in familial dysalbuminaemic hyperthyroxinaemia, can also distort free hormone estimates. A family history and evaluation of relatives may be useful, but an inherited pattern is not specific to hormone resistance. Binding-protein disorders and disorders of hormone action must remain separate diagnostic possibilities. [1, 3]

4.3 Choosing corroborative measurements

When interference remains plausible, laboratory evaluation may combine a fresh sample, a method with a different analytical design, total hormone measurements, and a direct separation method for free hormone assessment. Equilibrium dialysis and ultrafiltration separate free from protein-bound hormone, whereas routine immunoassays estimate free hormone under assay-dependent conditions. Direct separation can resolve some immunoassay problems, but displacement effects, particularly those related to heparin, remain a limitation. No single procedure should be applied without considering the mechanism under investigation. [1, 3, 9]

Reporting errors also warrant attention. The preanalytical and postanalytical framework described by Garg and colleagues includes patient identification, transcription, units, and the suitability of reference data. These checks are especially relevant when comparing results from different institutions. Before attributing a striking numerical difference to a biological event, the clinician and laboratory should establish that the measurements, units, and reference intervals are comparable. [7]

4.4 Platform changes and reference intervals

A platform change can expose a mismatch between previous expectations and a new assay's characteristics. In the Giovanella study, 4,648 of 5,111 FT4 measurements from patients with normal TSH were within the manufacturer's interval (90.9%). Local reference limits were explored using both direct and indirect approaches, and the authors considered the manufacturer's intervals appropriate for their population overall. The study therefore does not show that a change of platform necessarily creates erroneous results or requires automatic replacement of the manufacturer's interval. [4]

Its broader contribution is methodological: a laboratory should verify the suitability of an interval when introducing a method and explain relevant changes to clinicians. The study's direct approach used routine patients selected by normal TSH, while the indirect algorithm modelled a mixed routine dataset; neither eliminates every source of selection or classification bias. Verification of an interval and investigation of patient-specific interference are related quality tasks, but one cannot substitute for the other. [4, 8]

5 PERSISTENT HORMONE ELEVATION WITHOUT TSH SUPPRESSION

5.1 Distinguishing RTHβ from TSHoma

Once contextual explanations and measurement problems have been investigated, persistent genuine elevation of thyroid hormones with non-suppressed TSH warrants specialist assessment. RTHβ and TSHoma are central considerations, but clinical features and basal hormone concentrations overlap. Neither the absence of obvious thyrotoxicosis nor the magnitude of FT4 elevation reliably separates them. Coexisting primary thyroid disease can further modify the presentation. The distinction should therefore rest on converging clinical, familial, biochemical, genetic, and imaging evidence. [1, 5]

Campi and colleagues retrospectively reviewed 121 referral records, retaining 100 cases for analysis. Their main-text cohort included genetically established RTHβ, RTH-like cases without an identified *THRB* variant, TSHomas, and assay interference. This was an enriched diagnostic population rather than a consecutive general laboratory population. Its principal contribution is to document how errors occurred, including misclassification of interference as endocrine disease and misclassification of genuine disease as artefact. The group proportions should not be interpreted as the prevalence of these diagnoses among all patients with discordant tests. [5]

5.2 Imaging and biochemical differentiation

Pituitary imaging is particularly vulnerable to interpretation outside its biochemical context. In the main-text analysis, MRI initially failed to visualise a lesion in 6 of 26 TSHomas, while small lesions were found in 11 of 45 imaged RTHβ patients. Abnormal imaging was also reported in patients with spurious hyperthyroxinaemia. A visible lesion is consequently insufficient to establish autonomous TSH secretion, and a negative scan does not exclude a small TSHoma. The reported case of pituitary surgery followed by recognition of familial RTHβ illustrates the consequences of allowing imaging to determine the diagnosis prematurely. [5]

Peripheral markers and pituitary glycoprotein α-subunit measurements can contribute but also overlap. Sex hormone-binding globulin is affected by factors other than thyroid hormone action, including oestrogen exposure, liver function, insulin resistance, and growth hormone. In the Campi series, the α-subunit-related molar ratio was normal in some micro-TSHomas and elevated in some postmenopausal women with genetically proven RTHβ. These observations support using such measurements as corroboration rather than as decisive standalone tests. [1, 5]

Dynamic tests offer further evidence when appropriate and available. The Campi study examined thyrotropin-releasing hormone (TRH) stimulation and T3 suppression, but not every participant underwent every test. T3 suppression data were reported for 19 TSHomas, 16 genetically proven RTHβ patients, and 15 controls. Although the authors identified useful discrimination within their series, post-suppression values overlapped between five RTHβ patients and the TSHoma group. Coexisting thyroid disease could also affect TRH responses. Reported thresholds therefore require caution outside this selected cohort and cannot replace assessment of the complete diagnostic picture. [5]

T3 suppression is a specialist procedure with important constraints. The series identifies severe cardiac disorders, atrial fibrillation, untreated sinus tachycardia, and uncorrected adrenal insufficiency among contraindications, and notes that testing is usually avoided in older patients because of potential cardiac risk. Responses to prolonged somatostatin analogue exposure may assist selected difficult cases, but treatment responses also require interpretation in context. These investigations belong after measurement validity has been addressed, rather than serving as an initial response to an unexplained FT4 result. [1, 5]

5.3 Family assessment and genetics

A similarly abnormal biochemical pattern in relatives can support an inherited explanation and inform selection of genetic testing. A compatible pathogenic *THRB* finding strengthens the diagnosis of RTHβ; absence of an identified variant does not by itself establish TSHoma. Negative results must also prompt reconsideration of binding abnormalities and analytical explanations before a non-*THRB* resistance phenotype is assigned. Family testing is therefore most useful as part of the investigation, rather than as a substitute for analytical verification. [1, 3, 5]

The broader differential also depends on which hormones are abnormal. Koulouri and colleagues describe disorders of hormone transport and metabolism with patterns that differ from the combined T4 and T3 elevation often considered in RTHβ or TSHoma. These include monocarboxylate transporter 8 (MCT8)-related disease, with raised T3 and low T4, and functional deiodinase deficiency, with raised T4 and normal or low T3. Developmental and multisystem manifestations are important in recognising these uncommon disorders. This reinforces the need to interpret the complete biochemical and clinical phenotype before selecting genetic investigations. [1]

5.4 A practical diagnostic sequence

The combined evidence supports the staged sequence in Table 2. Its ordering reflects the need to establish what was measured and under which conditions before pursuing disease-specific tests. The sequence is not rigid: convincing evidence of pituitary dysfunction or a clinically significant thyroid disorder requires assessment alongside the analytical investigation. Equally, persistent discordance should not be dismissed because an initial test or scan is reassuring. The referral series demonstrates that reassessment over time can change the diagnosis. [1, 3, 5]

Table 2: Staged assessment of discordant thyroid function tests

Stage	Main question	Practical assessment
Define the discrepancy	Are results inconsistent with the patient, each other, or another method?	Review clinical findings, earlier results, units, reference intervals, and the reason for testing. [1, 7]
Establish context	Could physiology, illness, treatment, or sampling explain it?	Review pregnancy, recent illness, thyroid therapy and dose timing, medications, and supplements; assess persistence when appropriate. [1, 3]
Investigate measurement	Is there evidence of assay or binding-related distortion?	Discuss alternative assay design, TSH dilution or antibody-removal studies, total hormones, and specialist free hormone measurement with the laboratory. [1, 8, 9]
Assess genuine persistent abnormalities	Does the pattern support pituitary disease, hormone resistance, or another endocrine explanation?	Integrate pituitary assessment, family studies, targeted genetics, and selected dynamic tests; interpret MRI with biochemical evidence. [1, 3, 5]
Reconcile and follow up	Has the explanation remained consistent over time?	Reassess unresolved results, document the method and relevant interference, and maintain a follow-up plan. [3, 5]

This sequence is a synthesis of the cited sources and has not been prospectively validated as a unified pathway.

5.5 Testing practice and evidence limitations

Appropriate test selection is part of preventing avoidable discordance. The 2019 National Guideline Centre evidence review identified no relevant comparative clinical studies or economic evaluations for its testing-strategy question. Its cascade approach—TSH first for most adults, followed by FT4 or FT4 and FT3 according to TSH—was based on committee consensus, with simultaneous TSH and FT4 considered when secondary disease was suspected. This distinction matters: a pragmatic testing recommendation is not equivalent to demonstrated superiority for patient outcomes. [10]

The 2026 systematic review by Pioch and colleagues included 47 studies evaluating 54 interventions, with five randomised trials. It found predominantly favourable effects on testing-related outcomes and highlighted clinical decision support as a promising approach. However, much of the evidence was non-randomised, and the review rated non-randomised evidence as very low certainty. The findings support efforts to improve ordering practice, while reductions in testing volume alone cannot establish fewer diagnostic errors or better patient outcomes. Testing policies must also retain the exceptions needed to recognise central disease and investigate genuinely discordant findings. [3, 10, 11]

This review has several limitations. Its reference collection was supplied rather than systematically assembled, so completeness and freedom from selection bias cannot be assumed. Much of the explanatory literature is secondary and spans different assay generations. The principal diagnostic series is retrospective, selected, and incompletely tested; its abstract and main text also report different diagnostic group counts. Numerical comparisons here therefore use the main-text tables and their stated denominators. The platform-change study concerns one clinical setting, uses routine data to estimate reference intervals, and reports manufacturer sponsorship and author relationships with the manufacturer. The autoantibody investigation establishes a mechanism in one patient, without defining its population frequency. These constraints limit generalisation of a single diagnostic yield, prevalence estimate, or threshold. [4, 5, 8]

The sources leave a practical research question unresolved: whether a prospectively implemented sequence of clinical review, targeted analytical investigation, and specialist assessment improves outcomes compared with existing practice. Evaluations should distinguish accurate diagnostic resolution, avoidance of inappropriate treatment, timeliness, and patient outcomes from test counts alone. Better characterisation of platform-specific interference and reference intervals remains relevant, but analytical advances do not remove the need for clinical correlation. These priorities follow from the limitations of the diagnostic and testing-strategy evidence rather than from a demonstrated benefit of any single new pathway. [1, 3, 4, 5, 10, 11]

6 CONCLUSION

Discordant thyroid function tests require an explanation that is coherent with the patient, the treatment history, and the measurement method. The evidence reviewed supports clinical reassessment and targeted laboratory investigation before rare disorders are diagnosed, while warning that genuine disease can also be overlooked. Persistent hormone elevation with non-suppressed TSH warrants specialist differentiation using complementary evidence; imaging or a single dynamic response is insufficient in isolation. The most defensible approach is structured, collaborative, and open to reassessment, with the strength of each conclusion matched to the evidence available.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

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Disclosure of conflict of interest

The authors declare no conflicts of interest regarding this manuscript.

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