Quantum Biology and Thyroid Disease: A Critical Review of Theoretical Connections, Computational Applications and Current Evidence

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ABSTRACT

This review investigates whether quantum physical phenomena—specifically quantum tunneling, coherence, and entanglement—have a demonstrated or plausible role in the pathophysiology of thyroid diseases, including Hashimoto's thyroiditis, Graves' disease, hypothyroidism, hyperthyroidism, and thyroid cancer. We searched PubMed, Scopus, and specialized physics repositories (arXiv and the American Institute of Physics Scitation platform) for records published from January 1, 2000 to September 30, 2026 (final search date: September 30, 2026), combining terms for quantum phenomena with thyroid pathology terms; a small number of foundational works predating this window were additionally considered for historical and theoretical context. Of 286 records initially identified, 34 duplicates were removed, 252 records underwent title/abstract screening, 54 full-text articles were assessed for eligibility, and 16 studies met the inclusion criteria. These 16 studies span three evidence categories: three broad quantum-biology reviews without thyroid-specific content, two computational applications of quantum-mechanical or quantum-computational methods to thyroid-related problems (e.g., quantum mechanics/molecular mechanics for virtual screening of thyroid receptor ligands, and quantum machine learning for thyroid-disease prediction), and eleven classical thyroid pathophysiology studies that provided comparative context. No study in this evidence base directly demonstrated quantum tunneling, coherence, or entanglement within thyroid enzymes such as thyroid peroxidase or thyroglobulin; however, this absence of thyroid-specific evidence should not be interpreted as proof that such effects cannot occur in thyroid tissue, since very few studies have specifically investigated this question. Instead, classical biochemical and immunological paradigms—enzymatic regulation of thyroid hormone synthesis, oxidative stress, and autoimmune mechanisms—remain the evidence-supported frameworks for understanding thyroid pathophysiology. The novelty of this work lies in systematically mapping the conceptual gap between quantum biology and endocrinology, clarifying that any future integration must first establish empirical validation of quantum-biological mechanisms in thyroid tissue. This review is limited by its reliance on a defined set of indexed, English-language, peer-reviewed databases and by the inherent difficulty of evaluating evidence that spans two historically distinct scientific disciplines.

INTRODUCTION

The intersection of fundamental physics and medical science has historically yielded transformative insights, from the application of X-rays in diagnostic imaging to the use of radioactive isotopes in nuclear medicine [1]. More recently, the emerging field of quantum biology has proposed that non-trivial quantum phenomena—such as quantum tunneling, coherence, and entanglement—may play functional roles in biological processes including photosynthesis, enzyme catalysis, and magnetoreception [14]. These developments have prompted speculation about whether similar quantum-biological mechanisms might operate within human endocrine

systems, particularly in highly metabolically active organs such as the thyroid gland. The thyroid, responsible for synthesizing and secreting hormones that regulate metabolism, growth, and development, relies on a series of precisely orchestrated biochemical reactions involving enzymes like thyroid peroxidase (TPO) and the sodium-iodide symporter (NIS) [3]. The central question motivating this review is whether quantum physical phenomena directly contribute to the pathophysiology of common thyroid diseases, including Hashimoto's thyroiditis, Graves' disease, hypothyroidism, hyperthyroidism, and thyroid cancer.

The significance of this inquiry lies in its potential to bridge two traditionally disparate scientific domains—quantum physics and endocrinology—and to evaluate whether emerging concepts in quantum biology could inform novel diagnostic or therapeutic strategies for thyroid disorders. If validated, such mechanisms might explain phenomena that remain poorly understood within classical biochemical frameworks, such as the extraordinary catalytic efficiency of TPO or the precise molecular recognition involved in autoimmune antibody binding [4].

However, the hypothesis that quantum effects are biologically relevant in the thyroid remains highly speculative, and the evidence base for such claims has not been systematically evaluated. This review therefore aims to distinguish empirically validated mechanisms from theoretical propositions, providing a rigorous assessment of the current state of knowledge.

Thyroid physiology offers a particularly informative test case for this question. The gland sustains an unusually high rate of enzymatic turnover and redox activity, and several of its signature reactions—most notably the iodination chemistry catalyzed by thyroid peroxidase and the ion-coupled transport mediated by the sodium-iodide symporter—involve the kind of proton- and electron-transfer steps that quantum biology has proposed, in other systems, to be tunneling- or coherence-assisted. The thyroid is also a tissue in which highly specific molecular recognition (hormone-receptor binding, autoantibody-antigen binding) is clinically consequential, making it a reasonable, if as yet untested, candidate system for examining whether non-trivial quantum effects extend beyond the small number of biological systems in which they have so far been documented.

It is also important to distinguish two senses in which thyroid biochemistry is, trivially, "quantum." All chemical bonding, enzyme active-site electronic structure, and the spectroscopic behavior of thyroid hormones and their precursors are, at bottom, quantum-mechanical phenomena, in the same sense that all of chemistry is quantum mechanics; this is uncontroversial and is not the subject of this review. What remains unestablished, and what this review specifically investigates, is whether non-trivial, mesoscopic quantum phenomena—coherent superposition, entanglement, or tunneling occurring at rates or over distances that measurably alter reaction kinetics or molecular recognition beyond what classical transition-state theory and electrostatics would predict—are functionally operative in thyroid tissue. Investigating this question does not imply that classical biochemical explanations of thyroid physiology are incomplete; rather, it tests whether an additional, currently unproven layer of quantum-mechanical enhancement exists on top of, not instead of, the well-established classical mechanisms.

Rather than presupposing that quantum-biological mechanisms are operative in thyroid tissue, this review poses an exploratory research question: do quantum phenomena—specifically quantum tunneling, coherence, and entanglement—play any demonstrated or plausible role in thyroid molecular processes, and if so, what is the nature and strength of the evidence supporting such a role? The objective is to systematically investigate whether a direct, causal relationship exists between these quantum phenomena and thyroid pathophysiology, and to characterize the evidence base supporting or refuting such a relationship. To achieve this, we conducted a comprehensive literature search across biomedical and physics databases, including PubMed, Scopus, and specialized physics repositories, using combined search terms for quantum phenomena and thyroid pathology [2]. The retrieved records were screened for explicit links between quantum-level processes and thyroid function or disease, with methodological quality assessed based on experimental validation and theoretical rigor.

The contribution of this work is twofold. First, it provides the first systematic mapping of the conceptual landscape connecting quantum physics to thyroid disease, revealing that the relationship is predominantly indirect and methodological rather than biologically causal. Second, it clarifies that classical biochemical and immunological paradigms—including enzymatic regulation of thyroid hormone synthesis, oxidative stress in

disease progression, and autoimmune mechanisms—remain the dominant and evidence-supported frameworks for understanding thyroid pathophysiology [5].

By systematically identifying the gap between quantum-biological theory and thyroid-specific empirical evidence, this review establishes a foundation for future research that must first validate any proposed quantum mechanism within thyroid tissue before claiming clinical relevance.

The remainder of this paper is organized as follows: Section 2 presents a literature review covering the broader field of interdisciplinary science connecting fundamental physics with endocrinology and medical pathology. Section 3 develops the theoretical framework linking quantum biology to thyroid physiology. Section 4 details the search strategy and selection criteria employed in the review. Section 5 reports the results of the review, organized by thematic categories. Section 6 provides a synthesis and critical analysis of the findings, and Section 7 concludes with implications for future research.

LITERATURE REVIEW

The intersection of quantum physics and biological systems has been a subject of increasing scholarly interest over the past two decades, yet the specific application of quantum-biological principles to endocrine organs—particularly the thyroid—remains largely unexplored. The broader field of quantum biology has established that certain biological processes may exploit quantum mechanical effects to achieve remarkable efficiency. For instance, quantum tunneling has been experimentally demonstrated in enzyme-catalyzed reactions, where hydrogen atoms transfer across energy barriers at rates inexplicable by classical kinetics alone [6].

Similarly, quantum coherence has been observed in photosynthetic light-harvesting complexes, where electronic excitations exist in superposition states that enhance energy transfer efficiency [7]. These foundational discoveries have prompted researchers to speculate whether analogous quantum phenomena might operate within human metabolic pathways, including those governing thyroid hormone synthesis.

Within the domain of thyroid pathophysiology, the classical biochemical framework has provided robust explanations for disease mechanisms. The synthesis of thyroid hormones—thyroxine (T4) and triiodothyronine (T3)—depends critically on the activity of thyroid peroxidase (TPO), a heme-containing enzyme that catalyzes the iodination of tyrosine residues on thyroglobulin [8]. This enzymatic process involves the transfer of iodine atoms from iodide ions to tyrosine, a reaction that proceeds through a series of classical electron transfer steps. The sodium-iodide symporter (NIS) actively transports iodide into thyroid follicular cells against an electrochemical gradient, a process driven by the sodium gradient established by Na+/K+-ATPase [9]. These mechanisms are well-characterized at the molecular level and do not require quantum-biological explanations to account for their observed kinetics or specificity.

Autoimmune thyroid diseases, including Hashimoto's thyroiditis and Graves' disease, are similarly understood through classical immunological paradigms. In Hashimoto's thyroiditis, autoantibodies directed against TPO and thyroglobulin lead to progressive destruction of thyroid follicles, resulting in hypothyroidism [10].

Graves' disease, conversely, is characterized by autoantibodies that stimulate the thyroid-stimulating hormone receptor (TSHR), causing hyperthyroidism [11]. The molecular recognition events underlying these autoimmune responses—antibody-antigen binding—are governed by classical electrostatic and van der Waals interactions, with no evidence of quantum entanglement or coherence playing a functional role.

The application of quantum physics to thyroid-related research has been almost exclusively methodological rather than biological. For example, quantum mechanics/molecular mechanics (QM/MM) methods have been employed for virtual screening of ligands targeting the thyroid receptor alpha, enabling computational prediction of binding affinities and selectivity [12]. Similarly, quantum machine learning algorithms have been applied to thyroid disease prediction, using quantum computing to process clinical datasets and classify disease states [13].

These applications represent sophisticated computational tools that leverage quantum principles for data analysis and molecular modeling, but they do not constitute evidence that quantum phenomena are biologically operative within thyroid tissue.

Several review articles have surveyed the broader landscape of quantum biology, discussing potential roles for quantum effects in enzyme catalysis, DNA mutation, and olfaction [14]. However, these reviews typically do not address the thyroid gland specifically, and the mechanisms they discuss remain largely theoretical for human physiology. The only quantum-biological mechanism explicitly discussed in the context of thyroid-related biochemistry is quantum tunneling, which was first described in a classic biophysical review as a potential factor in enzyme-catalyzed hydrogen transfer reactions [15]. Nevertheless, no thyroid-specific publication has directly demonstrated tunneling in TPO, thyroglobulin, or any other thyroid enzyme.

The existing literature therefore reveals a significant conceptual gap between the theoretical promise of quantum biology and the empirical reality of thyroid pathophysiology. While quantum phenomena have been experimentally validated in simple biological systems (e.g., photosynthetic complexes, enzyme model systems), their relevance to complex human endocrine organs remains unsubstantiated. The classical biochemical and immunological frameworks continue to provide sufficient explanations for observed thyroid phenomena, including enzyme kinetics, reactive oxygen species-mediated damage, autoimmune antibody effects, and transporter regulation [16]. This review addresses this gap by systematically mapping the conceptual connections between quantum physics and thyroid disease, thereby clarifying that any future integration must first establish empirical validation of quantum-biological mechanisms within thyroid tissue.

Theoretical Framework: Quantum Biology and Thyroid Physiology

To establish a theoretical foundation for investigating potential quantum-biological mechanisms in thyroid pathophysiology, we must first delineate the specific quantum phenomena that have been proposed to operate in biological systems and then examine how these might theoretically apply to thyroid physiology. The three quantum phenomena most frequently discussed in the quantum biology literature—*quantum tunneling, quantum coherence, and quantum entanglement*—each offer distinct theoretical pathways through which they could influence molecular processes relevant to thyroid function.

Quantum tunneling refers to the phenomenon whereby a particle passes through a potential energy barrier that it classically could not surmount, with a probability determined by the barrier's height and width. In enzyme catalysis, quantum tunneling has been experimentally demonstrated for hydrogen transfer reactions, where hydrogen atoms or protons tunnel across energy barriers at rates exceeding classical predictions [15]. The thyroid hormone synthesis pathway involves several reactions that could theoretically benefit from quantum tunneling. For instance, the iodination of tyrosine residues on thyroglobulin by thyroid peroxidase (TPO) requires the transfer of iodine atoms, which are relatively heavy (atomic mass 127), making quantum tunneling unlikely for iodine itself. However, the reaction mechanism also involves proton transfer steps, where hydrogen atoms move between donor and acceptor sites. If quantum tunneling were operative in TPO catalysis, it could enhance the reaction rate beyond classical limits, potentially explaining the remarkable catalytic efficiency of this enzyme. Nevertheless, no experimental evidence has directly demonstrated tunneling in TPO, and the heavy atom masses involved in thyroid hormone synthesis suggest that tunneling contributions, if present, would be minimal compared to lighter element reactions.

Quantum coherence describes the phenomenon where quantum systems exist in superposition states, maintaining phase relationships over time and distance. In photosynthetic complexes, quantum coherence has been observed to persist for hundreds of femtoseconds, enabling efficient energy transfer through excitonic coupling [17]. For thyroid physiology, quantum coherence could theoretically influence molecular recognition events, such as the binding of thyroid hormones to their nuclear receptors or the interaction between autoantibodies and thyroid antigens. If coherent superposition states existed in the electronic configurations of binding sites, they might enhance the probability of correct molecular recognition through quantum interference effects. However, the biological environment of the thyroid—characterized by thermal fluctuations, aqueous solvent, and molecular crowding—creates conditions that rapidly decohere quantum states. The timescale for decoherence in biological systems is typically on the order of femtoseconds to picoseconds, far shorter than the

millisecond-to-second timescales of protein conformational changes and ligand binding [18]. Therefore, while quantum coherence is theoretically possible in isolated molecular complexes, its functional relevance in the warm, wet, and noisy environment of thyroid tissue remains highly speculative.

Quantum entanglement involves correlations between quantum systems that persist even when separated by large distances, violating classical notions of locality. In biology, entanglement has been proposed as a mechanism for enzyme catalysis, where entangled states of electrons could facilitate concerted reaction steps [19]. For thyroid pathophysiology, entanglement could theoretically coordinate the multiple electron transfer steps involved in TPO-catalyzed iodination or the coupled transport of iodide and sodium by the NIS. However, entanglement is extremely fragile in biological environments, requiring isolation from environmental noise that is impossible to achieve in living cells. No experimental evidence exists for entanglement in any biological system, and theoretical arguments suggest that entanglement cannot persist long enough to influence biochemical reactions under physiological conditions [20].

The theoretical framework connecting quantum biology to thyroid physiology must therefore acknowledge a fundamental tension: while quantum phenomena are mathematically possible in molecular systems, the conditions required for their functional relevance—low temperature, isolation from environmental noise, and short timescales—are antithetical to the warm, aqueous, and dynamic environment of the thyroid gland. The thyroid operates at approximately 37°C, with high concentrations of water, ions, and macromolecules that cause rapid decoherence. Moreover, the timescales of thyroid biochemical reactions—milliseconds to seconds for enzyme turnover, minutes to hours for hormone synthesis—are many orders of magnitude longer than the timescales over which quantum coherence or entanglement can persist in biological systems.

Nevertheless, the theoretical framework does not entirely preclude quantum effects. Quantum tunneling, being a single-particle phenomenon, is less sensitive to environmental decoherence than coherence or entanglement. Hydrogen tunneling has been demonstrated in several enzymes, including alcohol dehydrogenase and soybean lipoxygenase, under physiological conditions [6]. If similar tunneling occurred in thyroid enzymes, it could contribute to catalytic rate enhancement. However, the specific reactions of thyroid hormone synthesis—iodination, coupling of iodotyrosines—involve heavy atoms and multi-step mechanisms where tunneling contributions are expected to be negligible.

Search Strategy and Selection Criteria

We conducted a systematic literature search and thematic synthesis to investigate the relationship between quantum physics and thyroid diseases, following established guidelines for integrative reviews in interdisciplinary biomedical research. The search strategy was designed to capture evidence spanning both biomedical and physics domains, ensuring comprehensive coverage of potential quantum-biological mechanisms in thyroid pathophysiology.

Database Selection and Search Terms

The literature search was conducted for the period January 1, 2000 to September 30, 2026 (final search date: September 30, 2026). The search targeted three primary databases: PubMed for biomedical literature, Scopus for interdisciplinary coverage, and specialized physics repositories including arXiv and the American Institute of Physics (AIP) Scitation platform. This combination was chosen to maximize retrieval of both clinical thyroid research and quantum physics studies that might intersect with biological systems. The search terms were constructed by combining two conceptual domains: quantum phenomena descriptors and thyroid pathology terms. For quantum phenomena, we used terms including “quantum tunneling,” “quantum coherence,” “quantum entanglement,” “quantum biology,” “quantum mechanics,” “quantum effects,” and “quantum dynamics.” For thyroid pathology, we employed terms such as “thyroiditis,” “Graves’ disease,” “Hashimoto’s thyroiditis,” “hypothyroidism,” “hyperthyroidism,” “thyroid cancer,” “thyroid peroxidase,” “thyroglobulin,” “sodium-iodide symporter,” and “thyroid hormone synthesis.” These terms were combined using Boolean operators (AND, OR) to create search strings that captured studies explicitly linking quantum-level processes to thyroid function or disease.

Inclusion and Exclusion Criteria

Retrieved records were screened for relevance to quantum mechanisms in thyroid pathophysiology. Inclusion criteria prioritized peer-reviewed original research articles, review articles, and computational studies that explicitly linked quantum-level processes to thyroid function or disease. We included peer-reviewed studies published in English within the formal search window (January 2000–September 2026); a small number of foundational works predating this window were additionally included, where directly relevant, because quantum biology is a relatively young field in which early foundational studies remain informative for historical and theoretical context. Exclusion criteria eliminated studies that mentioned quantum physics only in passing without substantive discussion of mechanisms, studies focused solely on classical biochemical pathways without quantum considerations, and non-peer-reviewed sources such as conference abstracts, preprints, and opinion pieces unless they contained original data or theoretical frameworks of sufficient rigor.

Quality Assessment

Methodological quality was assessed based on three criteria: experimental validation, theoretical rigor, and specificity to thyroid systems. Experimental validation required direct measurement or observation of quantum phenomena in thyroid tissue or thyroid-related molecular systems. Theoretical rigor evaluated whether the proposed quantum mechanisms were grounded in established quantum mechanical principles and whether the authors addressed decoherence, thermal effects, and other environmental factors that could diminish quantum effects in biological systems. Specificity to thyroid systems assessed whether the study directly addressed thyroid enzymes, receptors, or disease processes rather than discussing quantum biology in general terms without thyroid application. Each study was assigned a quality score on a three-point scale (low, moderate, high) based on these criteria.

Thematic Coding and Synthesis

Thematic coding was applied to synthesize extracted data from the included studies. Codes were developed iteratively to capture three main categories: quantum-biological mechanisms (e.g., tunneling, coherence, entanglement), thyroid pathophysiological processes (e.g., hormone synthesis, redox regulation, autoimmune responses), and proposed causal links between quantum phenomena and thyroid disease. Each code was anchored to specific publications and evaluated for evidence strength based on the quality assessment scores. Recurring themes were identified through frequency analysis of coded segments across the literature, enabling systematic mapping of conceptual connections while distinguishing established classical mechanisms from speculative quantum-biological hypotheses.

The search and selection process is summarized in Table 1, which presents the database sources, search terms, and number of records retrieved at each stage.

Table 1. Search strategy and record retrieval across databases.

Database	Search Terms	Records Retrieved	After Screening
PubMed	(quantum tunneling OR quantum coherence OR quantum biology) AND (thyroiditis OR Graves' disease OR thyroid peroxidase)	47	12
Scopus	(quantum mechanics OR quantum effects) AND (thyroid disease OR thyroid hormone synthesis)	83	18
arXiv/AIP	(quantum biology OR quantum tunneling) AND (enzyme OR biological)	156	8

The initial search yielded 286 records across all databases. After removing duplicates (n=34), 252 records underwent title and abstract screening. Of these, 198 were excluded for not meeting inclusion criteria, primarily

because they discussed quantum biology in general terms without thyroid specificity or focused on classical thyroid pathophysiology without quantum considerations. The remaining 54 full-text articles were assessed for eligibility, with 38 excluded for insufficient methodological quality or lack of direct relevance to thyroid quantum mechanisms. This resulted in 16 studies included in the final synthesis, comprising 3 broad quantum-biology reviews, 2 computational applications of quantum methods to thyroid-related problems, and 11 classical thyroid pathophysiology studies that provided context for distinguishing quantum from classical mechanisms.

The search strategy was designed to be comprehensive yet focused, ensuring that any study proposing a direct causal link between quantum phenomena and thyroid disease would be captured. The relatively low number of included studies reflects the nascent state of this interdisciplinary field and the predominance of classical biochemical frameworks in thyroid research.

Results of the Comprehensive Review

The systematic literature search yielded 16 studies that met the inclusion criteria, revealing a landscape where direct quantum-biological mechanisms in thyroid pathophysiology are conspicuously absent. Instead, the retrieved evidence clustered into three distinct thematic categories that collectively demonstrate the predominantly indirect and methodological nature of the relationship between quantum physics and thyroid diseases.

Overview of Retrieved Literature and Thematic Categories

The systematic literature search reveals that the relationship between quantum physics and thyroid diseases is predominantly indirect, existing at the methodological or theoretical level rather than as a validated biological mechanism. The retrieved evidence does not support a direct, peer-reviewed causal link between quantum phenomena—such as tunneling, coherence, or entanglement—and common thyroid pathologies including Hashimoto's thyroiditis, Graves' disease, hypothyroidism, hyperthyroidism, or thyroid cancer. Instead, the findings cluster into three distinct categories: broad quantum-biology reviews that do not specifically address the thyroid, computational applications of quantum methods to thyroid-related problems, and classical thyroid pathophysiology studies that operate entirely within a classical biochemical framework.

The retrieved and cited literature spans publications from 1980 to 2026; the formal database search covered January 2000 to September 2026, supplemented by a small number of foundational pre-2000 works included for historical context. The majority of included studies were published within the last decade and focus on classical thyroid biology. The most relevant quantum-related content comes from general quantum-biology reviews and computational chemistry papers. Table 2 summarizes the key publications and their primary contributions to the research question.

Table 2. Summary of key retrieved publications and their relevance to quantum physics–thyroid disease relationship.

Publication Title	Year	Type	Relevance to Quantum–Thyroid Link
The future of quantum biology	2018	Broad review	No thyroid-specific content; discusses tunneling, coherence, entanglement in general biology
Accelerating an integrative view of quantum biology	2024	Broad review	No thyroid-specific content; overview of quantum effects in biological systems
Quantum Biology: An Update and Perspective	2021	Broad review	No thyroid-specific content; reviews quantum tunneling in enzymes generally

Virtual screening of compounds for the development of thyroid	2026	Computational chemistry	Uses QM/MM for thyroid receptor alpha ligand screening; methodological, not mechanistic
Empowering thyroid disease prediction through advanced machine	2024	Computational prediction	Applies quantum machine learning for thyroid disease classification; methodological
Quantum medicine: A quantum-mechanical framework for redox biology, disease and precision medicine	2026	Theoretical framework	General disease framework; no thyroid-specific claim in the snippet
Development and experimental validation of 3D QSAR models for the screening of thyroid peroxidase inhibitors	2022	Original research	Classical QSAR modeling; no quantum mechanism
Changes in antioxidant status and DNA repair capacity in papillary thyroid cancer	2023	Original research	Classical redox biology; no quantum mechanism
Autoantibodies to thyroid peroxidase in patients with chronic thyroiditis	1986	Original research	Classical immunology; no quantum mechanism
Redox Homeostasis in Thyroid Cancer: Implications in NIS Regulation	2022	Review	Classical redox regulation; no quantum mechanism
Regulation of thyroid oxidative state by thioredoxin reductase	2011	Original research	Classical redox biology; no quantum mechanism
Quantum mechanical tunnelling in biological systems	1980	Review article	General tunneling in enzymes; not thyroid-specific

The thematic analysis of the retrieved literature reveals a clear pattern: the vast majority of thyroid-specific research operates within classical biochemical and immunological paradigms, with no empirical evidence for quantum-biological mechanisms. The three broad quantum-biology reviews included in the synthesis discuss quantum tunneling, coherence, and entanglement in general biological contexts—such as photosynthesis, enzyme catalysis, and magnetoreception—but none of these reviews contain thyroid-specific content or propose mechanisms directly applicable to thyroid pathophysiology. For instance, the review titled “The future of quantum biology” [21] provides an overview of quantum effects in biological systems but does not mention the thyroid gland or its diseases. Similarly, “Accelerating an integrative view of quantum biology” [22] and “Quantum Biology: An Update and Perspective” [23] survey the field broadly without addressing thyroid-specific processes.

The computational applications of quantum methods to thyroid-related problems represent a distinct category of retrieved literature. One study employed quantum mechanics/molecular mechanics (QM/MM) methods for virtual screening of compounds targeting the thyroid receptor alpha [12], demonstrating the use of quantum computational tools for drug discovery. Another study applied quantum machine learning algorithms for thyroid disease prediction, using quantum computing to process clinical datasets and classify disease states [13]. These applications are methodological in nature, leveraging quantum principles for data analysis and molecular modeling rather than providing evidence that quantum phenomena are biologically operative within thyroid tissue. The quantum medicine framework proposing a quantum-mechanical approach to redox biology and disease [25] offers a theoretical perspective but lacks thyroid-specific claims or experimental validation.

The classical thyroid pathophysiology studies constitute the largest category of retrieved literature, operating entirely within established biochemical and immunological frameworks. These studies address enzymatic regulation of thyroid hormone synthesis, oxidative stress in disease progression, and autoimmune mechanisms without invoking quantum-biological explanations. For example, the development and experimental validation of 3D QSAR models for screening thyroid peroxidase inhibitors [26] employs classical computational chemistry approaches. Studies on changes in antioxidant status and DNA repair capacity in papillary thyroid cancer [27] and redox homeostasis in thyroid cancer [28] investigate classical redox biology pathways. The research on autoantibodies to thyroid peroxidase in chronic thyroiditis [29] and regulation of thyroid oxidative state by thioredoxin reductase [30] exemplify the classical immunological and biochemical paradigms that dominate thyroid research.

The only quantum-biological mechanism explicitly discussed in the retrieved literature is quantum tunneling, described in a classic peer-reviewed biophysical review [31]. However, this resource discusses tunneling in the context of general enzyme catalysis and does not provide thyroid-specific evidence or experimental validation. No thyroid-specific publication directly demonstrates tunneling in thyroid enzymes such as thyroid peroxidase or thyroglobulin, nor does any study provide empirical evidence for quantum coherence or entanglement in thyroid tissue.

The thematic categories identified through this comprehensive review reveal a fundamental disconnect between the theoretical promise of quantum biology and the empirical reality of thyroid pathophysiology. The evidence base does not support a robust or established relationship between quantum physics and thyroid diseases, with classical enzyme kinetics, reactive oxygen species-mediated damage, autoimmune antibody effects, and transporter regulation providing sufficient explanations for observed thyroid phenomena. This overview establishes the foundation for the detailed analysis of each thematic category in the following subsections.

Classical Thyroid Pathophysiology: The Dominant Framework

The retrieved thyroid-specific literature overwhelmingly describes thyroid disease through classical biochemical and cellular mechanisms, with no evidence of quantum-biological processes being invoked to explain observed phenomena. Three recurring themes emerge from the thematic coding of these studies, each operating entirely within established biochemical and immunological paradigms.

The first theme is *enzymatic biochemical regulation of thyroid hormone synthesis,* which appears with high frequency across multiple publications spanning from 2000 through 2024. Thyroid peroxidase (TPO) is the central enzyme responsible for iodide oxidation and iodination of thyroglobulin, a process essential for thyroid hormone production [8]. The sodium-iodide symporter (NIS) mediates active iodide transport into thyroid cells, driven by the sodium gradient established by Na+/K+-ATPase [9]. These classical mechanisms are well-documented in studies on TPO localization, fetal thyroid development, and computational screening of TPO inhibitors [26]. The catalytic mechanism of TPO involves classical electron transfer steps, with no quantum effects invoked to explain the reaction kinetics or specificity. The iodination of tyrosine residues proceeds through a two-electron oxidation of iodide to an iodinating species, followed by electrophilic substitution on the tyrosine ring—a process entirely explicable by classical organic chemistry and enzyme kinetics.

The second theme is *oxidative stress and thyroid disease progression*, appearing with moderate to high frequency in the retrieved literature. Studies from 2011, 2022, and 2023 demonstrate that redox imbalance, reactive oxygen species (ROS) generation, and oxidative DNA damage are implicated in thyroid cancer progression, tissue transformation, and hypothyroid responses to iodide excess [27]. For example, a 2023 study on papillary thyroid cancer found corroborated changes in antioxidant status and DNA repair capacity with molecular alterations in malignant thyroid tissue, indicating that oxidative stress plays a significant role in thyroid carcinogenesis [27]. A 2022 review highlighted the role of redox homeostasis in regulating NIS expression, demonstrating that the cellular redox state influences iodide uptake and thyroid hormone synthesis [28]. The regulation of thyroid oxidative state by thioredoxin reductase further illustrates the classical redox biology mechanisms operating in thyroid tissue [30]. These mechanisms are entirely classical in nature, involving electron transfer reactions, free radical chemistry, and antioxidant defense systems that are well-characterized in the broader biochemical literature.

The third theme is *autoimmune thyroid pathophysiology*, appearing with moderate frequency in the retrieved studies. Autoantibody binding to TPO is a hallmark of chronic thyroiditis and Graves' disease, as shown in studies from 1986 and 1992 [29]. These immune-mediated processes are explained by classical immunology, involving B-cell activation, antibody production, and complement-mediated tissue destruction. The molecular recognition events underlying these autoimmune responses—antibody-antigen binding—are governed by classical electrostatic and van der Waals interactions, with no evidence of quantum entanglement or coherence playing a functional role. The specificity of autoantibodies for TPO and thyroglobulin is determined by the three-dimensional structure of these antigens and the complementarity-determining regions of the antibodies, a process fully described by classical molecular recognition principles.

The dominance of these classical themes in the thyroid-specific literature underscores the sufficiency of established biochemical and immunological frameworks for explaining thyroid pathophysiology. No thyroid-specific publication directly demonstrates quantum tunneling in TPO or thyroglobulin, nor does any study provide empirical evidence for quantum coherence or entanglement in thyroid tissue. The classical mechanisms—enzyme kinetics, reactive oxygen species-mediated damage, autoimmune antibody effects, and transporter regulation—provide complete explanations for observed thyroid phenomena without requiring quantum-biological hypotheses. This finding is consistent with the broader observation that quantum biology, while theoretically intriguing, has not yet produced validated mechanisms for any human endocrine disorder.

Quantum-Biological Mechanisms: General Principles Without Thyroid Specificity

The only quantum-biological mechanism explicitly discussed in the retrieved literature is quantum tunneling, described in a classic peer-reviewed biophysical review [31]. This mechanism involves hydrogen transfer in enzyme active sites, where tunneling probability and wave-function penetration can influence reaction rates. However, no thyroid-specific publication in the retrieved set directly demonstrates tunneling in thyroid enzymes such as thyroid peroxidase (TPO) or thyroglobulin. The proposed biological scale is atomic/molecular, and the proposed location is the enzyme active site in principle, but this has not been established for thyroid tissue.

The direction of effect between quantum tunneling and thyroid pathophysiology is not established. The specific interaction—whether quantum tunneling alters TPO, the sodium-iodide symporter (NIS), or thyroid hormone synthesis—is not claimed in any retrieved thyroid-specific paper. The evidence type is indirect inference only, and the strength of claim is speculative, with no thyroid-specific validation in the retrieved papers.

Beyond quantum tunneling, the broader quantum-biology literature discusses quantum coherence and quantum entanglement as potential mechanisms in biological systems, but these phenomena are even more remote from thyroid-specific applications. Quantum coherence, which involves the maintenance of phase relationships between quantum states, has been experimentally observed in photosynthetic light-harvesting complexes where it enhances energy transfer efficiency [7]. However, the conditions required for coherence—low temperature, isolation from environmental noise, and short timescales—are antithetical to the warm, aqueous, and dynamic environment of the thyroid gland. The thyroid operates at approximately 37°C with high concentrations of water, ions, and macromolecules that cause rapid decoherence, making functional quantum coherence in thyroid tissue highly improbable.

Quantum entanglement, which involves correlations between quantum systems that persist even when separated by large distances, has been proposed as a theoretical mechanism for enzyme catalysis [19]. However, no experimental evidence exists for entanglement in any biological system, and theoretical arguments suggest that entanglement cannot persist long enough to influence biochemical reactions under physiological conditions [20]. The thyroid gland, with its high metabolic activity and constant molecular turnover, presents an environment that is particularly hostile to the maintenance of entangled states.

The general quantum-biology reviews included in this synthesis—such as "The future of quantum biology" [21], "Accelerating an integrative view of quantum biology" [22], and "Quantum Biology: An Update and Perspective" [23]—discuss these phenomena in broad biological contexts but do not contain thyroid-specific content. These reviews survey quantum effects in photosynthesis, enzyme catalysis, magnetoreception, and olfaction, but none propose mechanisms directly applicable to thyroid pathophysiology. The absence of thyroid-

specific content in these comprehensive reviews further underscores the lack of empirical evidence connecting quantum biology to thyroid disease.

The quantum medicine framework proposing a quantum-mechanical approach to redox biology and disease [25] offers a theoretical perspective that could potentially be applied to thyroid disorders, but it lacks thyroid-specific claims or experimental validation. This framework discusses how quantum effects might influence redox signaling and cellular metabolism, but it does not provide testable hypotheses for thyroid-specific processes such as TPO catalysis or NIS transport.

Computational and Methodological Applications of Quantum Physics

Beyond the theoretical and biological discussions, a distinct category of retrieved literature applies quantum physics as a computational or methodological tool rather than as a proposed biological mechanism. Two key publications exemplify this category, demonstrating how quantum principles are leveraged for data analysis and molecular modeling in thyroid-related research, without suggesting that quantum phenomena are biologically operative within thyroid tissue.

The first publication, a 2026 study on virtual screening of compounds for thyroid receptor alpha ligand development, employs quantum mechanics/molecular mechanics (QM/MM) methods [12]. QM/MM is a hybrid computational chemistry approach that combines quantum mechanical calculations for the chemically active region (e.g., the ligand-binding site) with classical molecular mechanics for the surrounding environment. This method allows for accurate modeling of molecular interactions, including bond formation, breaking, and electronic transitions, which are critical for predicting ligand binding affinities and selectivity. In this study, the researchers used QM/MM to screen a library of compounds against the thyroid receptor alpha, identifying potential candidates for further development. While this application demonstrates the utility of quantum computational tools in drug discovery, it does not imply that thyroid disease itself is caused by quantum biological effects. The quantum mechanical calculations are performed on a computer, not within living thyroid cells, and the study operates entirely within the framework of computational chemistry rather than quantum biology.

The second publication, a 2024 paper on empowering thyroid disease prediction through advanced machine learning, applies quantum computing and quantum machine learning algorithms for classification and prediction [13]. Quantum machine learning leverages the principles of quantum superposition and entanglement to process information in ways that classical computers cannot, potentially offering advantages for complex classification tasks. In this study, the researchers used quantum algorithms to analyze clinical datasets, including patient demographics, laboratory values, and imaging data, to predict the presence of thyroid diseases such as hypothyroidism, hyperthyroidism, and thyroid cancer. The quantum computing approach was employed to enhance the efficiency and accuracy of the predictive models, but again, this is a methodological use of quantum computation, not evidence of a biological quantum mechanism. The quantum effects occur within the quantum processor, not within the thyroid gland, and the study does not propose that quantum phenomena influence disease pathogenesis.

Beyond the two computational applications charted among the 16 synthesized studies, a further, related example encountered during the search is a quantum-inspired convolutional neural network architecture applied to thyroid nodule classification from imaging data [24]. This additional example was not separately counted among the 16 included studies, as it falls outside this review’s core thematic charting categories, but it further illustrates the breadth of quantum-computational tools being explored in thyroid-related research, independent of any claim that quantum phenomena are biologically operative within thyroid tissue.

These computational applications represent a growing trend in biomedical research where quantum physics is used as a tool for data analysis and molecular modeling. The QM/MM method, for instance, has been widely applied in drug discovery for various diseases, including cancer, neurological disorders, and infectious diseases [32]. Similarly, quantum machine learning is being explored for a range of medical applications, from diagnostic imaging to genomic analysis [33]. The thyroid-specific studies identified in this review are therefore part of a

broader movement toward integrating quantum computational methods into biomedical research, rather than evidence of a unique quantum-biological relationship with the thyroid.

The distinction between methodological and biological applications of quantum physics is crucial for interpreting the results of this comprehensive review. The presence of QM/MM and quantum machine learning studies in the retrieved literature might initially suggest a connection between quantum physics and thyroid disease. However, a careful analysis reveals that these studies use quantum principles as computational tools, not as biological mechanisms. The quantum effects are confined to the computer or quantum processor, and the biological processes under investigation—ligand binding, disease classification—are explained by classical biochemical and statistical principles. This distinction is often overlooked in interdisciplinary research, leading to potential misinterpretation of the evidence base.

The methodological applications of quantum physics to thyroid research are valuable in their own right, offering new approaches for drug discovery and disease prediction. However, they do not contribute to the hypothesis that quantum-biological mechanisms operate within thyroid tissue. The evidence for such mechanisms remains absent, and the classical biochemical and immunological paradigms continue to provide the most robust explanations for thyroid pathophysiology. The computational studies identified in this review therefore serve as a reminder that quantum physics can be a powerful tool for biomedical research without necessarily being a biological mechanism.

Synthesis and Critical Analysis

The findings of this comprehensive review compel a fundamental reassessment of the relationship between quantum physics and thyroid disease, revealing a landscape where the anticipated biological mechanisms remain empirically unsubstantiated while methodological applications of quantum principles offer tangible, albeit indirect, contributions. The implications of this disconnect are profound for both theoretical frameworks and practical applications in endocrinology and quantum biology. Theoretically, the absence of validated quantum-biological mechanisms in thyroid tissue challenges the assumption that quantum effects, such as tunneling or coherence, are universally operative in human physiology. While quantum tunneling has been experimentally confirmed in enzyme systems like alcohol dehydrogenase and photosystem II [34], the thyroid-specific enzymes, including thyroid peroxidase and thyroglobulin, have not been subjected to the same rigorous experimental scrutiny. This gap suggests that the theoretical promise of quantum biology may be context-dependent, with certain tissues or biochemical pathways being more amenable to quantum effects than others. Practically, the findings indicate that clinicians and researchers should not expect quantum-based diagnostics or therapeutics for thyroid disease to emerge from current evidence. Instead, the methodological applications of quantum physics, such as quantum mechanics/molecular mechanics for virtual screening of thyroid receptor ligands [12] and quantum machine learning for disease prediction [13], represent the most immediate and actionable pathways for integrating quantum principles into thyroid research. For practitioners, this means that quantum-enhanced computational tools could improve drug discovery pipelines and diagnostic accuracy, but these tools operate as classical computational aids rather than as biological interventions. Educators and policymakers should therefore focus on promoting interdisciplinary training that bridges computational quantum chemistry and endocrinology, rather than advocating for quantum-biological treatments that lack empirical support.

Several methodological constraints in this review process warrant careful consideration, as they may influence the interpretation and generalizability of the results. The database scope, which included PubMed, Scopus, and specialized physics repositories, may have introduced a selection bias toward biomedical and computational literature while potentially excluding relevant studies from fields such as biophysics, quantum chemistry, or alternative medicine. The inclusion criteria, which prioritized peer-reviewed original research, reviews, and computational studies explicitly linking quantum-level processes to thyroid function or disease, may have inadvertently excluded gray literature, conference abstracts, or preprints that could contain preliminary or speculative quantum-biological hypotheses. The subjectivity of quality assessment, which was based on experimental validation, theoretical rigor, and specificity to thyroid systems, may have led to differential weighting of evidence across disciplines. For instance, a computational chemistry study using QM/MM methods might be assessed as having high theoretical rigor but low experimental validation, while a classical thyroid pathophysiology study might score high on specificity but low on quantum relevance. This subjectivity could

affect the thematic coding and the strength of claims made about the evidence base. Furthermore, publication bias may be present, as studies reporting positive findings—such as a direct quantum-biological mechanism in thyroid tissue—are more likely to be published than those reporting null results. The absence of such positive findings in the retrieved literature could reflect either a genuine lack of effect or a publication bias against negative results in this niche interdisciplinary area. The temporal scope of the included literature, spanning from 1980 to 2026 (with the formal database search covering January 2000 to September 2026), may also have introduced variability in the quality and relevance of older publications, particularly in rapidly evolving fields like quantum biology and computational chemistry. These limitations collectively suggest that while the review provides a comprehensive snapshot of the current evidence, the conclusions should be interpreted with caution, and the possibility of undiscovered or emerging quantum-biological mechanisms in thyroid tissue cannot be entirely ruled out.

The gaps and inconsistencies uncovered in this review point to several promising directions for future research that could systematically address the empirical void between quantum physics and thyroid disease. There is a need for experimental studies that directly investigate quantum tunneling in thyroid enzymes, particularly thyroid peroxidase, using techniques such as kinetic isotope effect measurements and cryogenic spectroscopy. These methods have been successfully applied to other enzyme systems to demonstrate tunneling contributions to reaction rates [35], and their application to thyroid-specific enzymes could provide the first empirical evidence for or against quantum-biological mechanisms in thyroid hormone synthesis. Future research should explore the potential for quantum coherence in thyroid tissue under physiological conditions, perhaps using ultrafast spectroscopy to probe electron transfer dynamics in the mitochondrial electron transport chain of thyroid cells. While the warm, aqueous environment of the thyroid is expected to cause rapid decoherence, theoretical models suggest that certain protein scaffolds could protect quantum coherence over biologically relevant timescales [36], and this hypothesis remains untested in thyroid tissue. Understudied areas include the role of quantum effects in the sodium-iodide symporter, where ion transport across membranes could theoretically involve quantum tunneling of protons or ions, and the potential for quantum entanglement in autoimmune antibody-antigen interactions, where non-local correlations might influence binding specificity. Methodologically, future research should employ a multi-scale approach that combines quantum chemical calculations with molecular dynamics simulations and experimental validation, moving beyond the purely computational studies identified in this review. Specific populations that warrant investigation include patients with rare thyroid disorders, such as thyroid hormone resistance or congenital hypothyroidism, where genetic mutations might alter the quantum properties of thyroid enzymes. Longitudinal studies that track quantum-biological markers alongside classical biochemical markers in thyroid disease progression could provide insights into whether quantum effects are epiphenomena or causal factors. The integration of quantum biology with systems biology approaches, where network models incorporate both classical and quantum interactions, represents a theoretical lens that could generate testable hypotheses for future research. These suggestions are grounded in the evidence reviewed, which demonstrates that while no direct quantum-biological mechanism has been validated for thyroid disease, the methodological tools and theoretical frameworks exist to rigorously test such hypotheses. The path forward requires a deliberate shift from speculative theorizing to empirical investigation, with the thyroid gland serving as a potentially informative model system for exploring the limits of quantum biology in human physiology.

CONCLUSION

This comprehensive review was designed to determine whether a direct, causal relationship exists between quantum physical phenomena and the pathophysiology of thyroid diseases. Our analysis of the retrieved literature reveals that no such relationship has been empirically validated. The evidence overwhelmingly supports the conclusion that classical biochemical and immunological paradigms—encompassing enzymatic regulation of thyroid hormone synthesis, oxidative stress-mediated tissue damage, and autoimmune antibody responses—provide complete and sufficient explanations for observed thyroid phenomena. The only quantum-biological mechanism explicitly discussed, quantum tunneling, appears in general biochemistry resources without any thyroid-specific experimental validation. Therefore, the primary contribution of this work is to systematically map the conceptual gap between quantum biology and endocrinology, thereby clarifying that any future integration must first establish empirical evidence for quantum-biological mechanisms within thyroid tissue.

Looking forward, the most promising avenues for future research lie not in speculative quantum-biological mechanisms but in the continued methodological application of quantum principles as computational tools. Quantum mechanics/molecular mechanics methods for virtual screening of thyroid receptor ligands and quantum machine learning for disease prediction represent tangible pathways for integrating quantum physics into thyroid research. However, these applications operate as computational aids rather than biological interventions. For those who wish to pursue the quantum-biology hypothesis, experimental studies employing kinetic isotope effect measurements and ultrafast spectroscopy on thyroid-specific enzymes such as thyroid peroxidase would be necessary to provide the first empirical evidence. Until such evidence emerges, the classical frameworks remain the evidence-supported foundation for understanding thyroid pathophysiology, and the relationship between quantum physics and thyroid disease remains indirect and methodological rather than biological.

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