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Thyroid Hormones in Prostate Cancer: A Systematic Review and Bibliometric Study

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Abstract: Prostate cancer (PCa) is a prevalent malignancy in men, traditionally linked to androgen receptor signaling. Emerging evidence suggests thyroid hormones (THs, particularly T3/T4) play a complex role in PCa biology. THs regulate gene transcription via nuclear receptors TRα/β, modulating proliferation, apoptosis, and AR signaling, while non-genomic pathways through integrin αvβ3 activate MAPK/PI3K–Akt signaling, driving metabolic reprogramming, migration, and angiogenesis. Local DIO enzymes fine-tune T3/T4 levels, with DIO2 enhancing proliferation and DIO3 creating a low-TH microenvironment to facilitate immune evasion. Epidemiological studies associate hyperthyroidism or low TSH with elevated PCa risk, whereas experimental models show inconsistent effects, reflecting regulation by hormone levels, receptor distribution, and tumor molecular features. Bibliometric analyses reveal a shift from epidemiological studies to molecular, immune, and metabolic mechanistic research, though clinical translation remains limited. This review synthesizes current knowledge on THs in PCa, highlighting mechanistic insights, evidence gaps, and future directions, aiming to inform early detection, stratification, and therapeutic strategies.

Keywords: Prostate cancer; Thyroid hormones; Bibliometrics; CiteSpace; VOSviewer

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1. Introduction

Prostate cancer (PCa) is one of the most common malignant tumors in men, with increasing incidence and mortality rates [1]. Current research on PCa mainly focuses on the androgen signaling pathway, in which the interaction between androgens and the androgen receptor (AR) is pivotal to tumor initiation and progression [2]. However, recent studies have increasingly suggested that thyroid hormones (THs), particularly triiodothyronine (T3) and thyroxine (T4), may exert complex and incompletely understood effects in the initiation and progression of PCa as well as other malignancies [3].

THs are primarily synthesized and secreted by the thyroid gland. T4, as a prohormone, is converted in peripheral tissues into the biologically more active T3 by type 1 and type 2 deiodinases (DIO1, DIO2). T3 then

binds to thyroid hormone receptors (TRα/β), regulating downstream gene transcription and thereby influencing cell proliferation, differentiation, and metabolic processes [4].

In oncology, studies have revealed that alterations in physiological TH levels are closely associated with the development of multiple cancers. Evidence indicates that their effects in solid tumors, including breast, liver, and gastric cancers, are heterogeneous and depend on tumor type, hormonal milieu, and microenvironmental interactions [3]. For instance, in breast cancer, T3 induces the mRNA expression of growth factors TGFα and TGFβ in ER-positive cells [5], while T4 activates the ERK1/2 signaling pathway to upregulate PD-L1 expression, enabling immune evasion [6]. In gastric and lung cancers, reduced TH levels are often linked to tumor progression, metastasis, and poor prognosis [7,8]. These findings suggest that the role of THs in cancer may be bidirectional: in some cases, T3/T4 promote tumor progression, whereas in others they exert inhibitory effects, depending on molecular characteristics and hormone levels.

In PCa, however, the role of THs remains controversial. Epidemiological studies have reported that hyperthyroidism or reduced thyroid-stimulating hormone (TSH) levels may be associated with an increased risk of PCa [9], whereas hypothyroidism may be associated with a reduced risk [10]. In animal models, an elevated T3/T4 ratio or administration of high-dose T3 was shown to suppress prostate tumor growth [11,12]. These observations indicate that the relationship between THs and PCa is not a straightforward causal relationship but is likely influenced by hormonal background, cancer molecular features, and the tumor microenvironment. Current evidence remains inconclusive. Therefore, this review aims to provide a systematic overview, integrating bibliometric analyses, of the research progress on THs, particularly T3 and T4, in PCa, exploring their mechanisms of action and potential clinical implications, with the goal of offering a theoretical basis for future research and therapeutic strategies.

2. Relationship between thyroid hormones and the prostate

In recent years, studies have increasingly recognized the prostate as an important target tissue of THs [13]. THs are involved in prostate growth, development, metabolic regulation, and potential processes of tumorigenesis through multiple mechanisms, including hormone transport, local activation, receptor-mediated signaling, and interactions with other endocrine factors.

2.1. Uptake and transport of thyroid hormones in the prostate

Studies have found that the entry of T3 and T4 into prostate cells mainly depends on specific transport proteins, including monocarboxylate transporters MCT8 (SLC16A2), MCT10 (SLC16A10), organic anion transporting polypeptides (OATPs), and members of the L-type amino acid transporter family. Among these, MCT8 is considered the most efficient and specific transporter of THs [14]. Expression of MCT8 has been detected in PCa cell lines such as LNCaP, DU145, and PC-3, suggesting that prostate cells possess the capacity for active THs uptake [14]. In addition, the high-affinity T3-binding protein μ-crystallin (CRYM) has been identified in both normal and cancerous prostate tissues. Within cells, CRYM binds T3, regulates its cytoplasmic accumulation and nuclear translocation, and simultaneously reduces T3 binding to TRs, thereby exhibiting potential antitumor activity [15].

2.2. Local activation and inactivation of thyroid hormones in the prostate

The activity of THs in the prostate is tightly regulated by the deiodinase system. The prostate expresses DIO1, which converts T4 into the more bioactive T3. However, its activity gradually declines with aging or androgen

deprivation, while sexual activity can enhance DIO1 activity through sympathetic nervous mechanisms, thereby maintaining local T3 levels in the prostate [16]. Conversely, type 3 deiodinase (DIO3) is upregulated under malignant or stress conditions, mediating the inactivation of T3 to produce reverse triiodothyronine (rT3). This creates a state of local hypothyroidism, which promotes tumor cell proliferation and immune evasion [17]. In animal models supplemented with T3, increased DIO3 expression accompanied by decreased DIO1 and DIO2 expression has also been observed, suggesting that THs in the prostate are subject to dynamic feedback regulation [12].

2.3. Expression of thyroid hormone receptors and signaling mechanisms in the prostate

Existing studies have shown that prostate epithelial cells express thyroid hormone nuclear receptors TRα1, TRα2, and TRβ [18]. Using immunoblotting and mRNA analysis, TRβ protein expression has been detected in multiple human PCa cell lines, including LNCaP, PC-3, and DU145 [19]. In addition to nuclear receptor–mediated mechanisms, THs can also activate non-genomic signaling pathways, such as integrin αvβ3–mediated signaling, which rapidly activates MAPK and PI3K/Akt pathways. These pathways contribute to metabolic reprogramming, proliferation, migration, and angiogenesis in prostate cells [20]. This non-classical mechanism appears to be enhanced in PCa, indicating its potential tumor-promoting role.

3. Association between thyroid hormones and prostate cancer

3.1. Epidemiological associations between thyroid function and PCa

Epidemiological studies provide important evidence for the potential association between thyroid hormones (THs) and prostate cancer (PCa) risk. Some epidemiological surveys have suggested that subclinical hypothyroidism is not significantly associated with PCa risk [21], although other researchers have proposed that untreated hypothyroidism may exert a protective effect against PCa [10]. For instance, in a prospective cohort study involving 3,649 patients with 20 years of follow-up, 7.8% of men were diagnosed with PCa. Serum TSH and free T4 were measured, and analysis revealed that higher TSH levels were associated with a reduced PCa risk (adjusted HR: 0.7 per 1 mIU/L increase), whereas higher free T4 was associated with an increased PCa risk (adjusted HR: 1.11 per 1 pmol/L) [22]. In addition, higher free T3 levels have also been closely associated with an increased risk of PCa [23]. Therefore, current findings remain somewhat controversial.

3.2. Potential mechanisms

3.2.1. Receptor-mediated gene transcription

THs regulate the expression of specific genes in PCa cells by binding to TRα and TRβ. The binding of T3 to TR activates target gene transcription, thereby regulating cell proliferation, differentiation, and apoptosis [21]. In LNCaP cells, T3 markedly upregulates genes such as Cyclin D1 and MMP-2, which are associated with tumor proliferation and invasion [11]. Moreover, T3 has been shown to enhance AR expression by modulating NCOA4, an AR-associated protein, thereby increasing the androgen responsiveness of PCa cells [24]. These findings suggest that THs may influence tumor growth by upregulating AR and its co-activators, thereby modulating androgen responsiveness in PCa cells.

3.2.2. Non-genomic signaling pathways

In addition to the classical nuclear receptor pathway, THs have been found to activate intracellular signaling

cascades through membrane receptors such as integrin αvβ3, including the MAPK/ERK and PI3K/Akt pathways, thereby regulating cell proliferation, migration, and invasion [25]. Specifically, the binding of T3 to integrin αvβ3 promotes MAPK pathway activation, which further enhances the invasive potential of PCa cells.

3.2.3. Role of the DIO enzyme family in PCa

The DIO enzyme family plays a crucial role in TH metabolism within PCa. DIO1 and DIO2 catalyze the conversion of T4 to T3, whereas DIO3 facilitates T3 inactivation. Studies have shown that DIO2 expression is upregulated in PCa cells, which may enhance tumor cell proliferation and invasion by increasing local T3 concentrations [26]. Therefore, DIO2 activity may represent a key regulatory factor mediating the effects of THs in PCa.

4. Bibliometric analysis of studies on thyroid hormones and prostate cancer

4.1. Data sources and search strategy

We selected Web of Science Core Collection (WoSCC) as the data source [27]. The literature was restricted to the period from January 1, 2015, to December 31, 2024. The search strategy was: TS = ("thyroid function" OR "thyroid hormone*" OR "Triiodothyronine" OR "Free T3" OR "Thyroxine" OR "Free T4" OR "Thyroid Stimulating Hormone" OR "TSH" OR "Thyrotropin" OR "Thyrotropin-Releasing Hormone" OR "TRH" OR "Thyroid Antibodies" OR "TPOAb" OR "TgAb" OR "TRAb" OR "TSHR-Ab") AND ("prostate cancer" OR "prostate carcinoma" OR "prostate neoplasm*" OR "prostatic malignancy" OR "prostate tumor*" OR "prostate adenocarcinoma"). The document types were limited to original articles and reviews. A total of 141 publications were retrieved, including 95 original articles and 46 reviews. All publications were in English and focused on studies related to THs and PCa.

4.2. Analysis methods

The study extracted raw data from WoSCC, including publication count, citation frequency, h-index, publication year, country/region, institutions, authors, journals, references, and keywords. VOSviewer (version 1.6.19) was employed to visualize keywords and institutional collaborations, and to construct co-citation, co-occurrence, and collaboration networks in this research field [28]. CiteSpace 6.2.R6 (64-bit) Basic was used to map the intellectual evolution and temporal distribution of document clusters, thereby revealing research progress and trends in the field of THs and PCa [29]. To analyze annual research trends, R Studio 4.3.1 and the ggplot2 package [30] were applied to perform statistical analysis and visualization of publication volume, further demonstrating the academic attention and developmental trends in this area.

4.3. Trends and changes in research output

Based on data from WoSCC, the number of publications on THs and PCa has exhibited fluctuations (**Figure 1**). From 2015 to 2017, the number of relevant publications remained relatively stable, at 15, 18, and 12 articles, respectively, indicating initial attention to this field. Between 2018 and 2020, the number of studies increased markedly, reaching 19 in 2020, reflecting strong academic interest in the relationship between THs and PCa. However, from 2021 onwards, the number of publications declined annually, with 11 articles in both 2021 and 2022, and an estimated 10 in 2024, suggesting a waning research momentum, possibly due to a plateau in the

field or a shift in focus to other areas. Overall, this field remains promising and valuable, and future progress may emerge through novel research approaches or interdisciplinary collaborations.

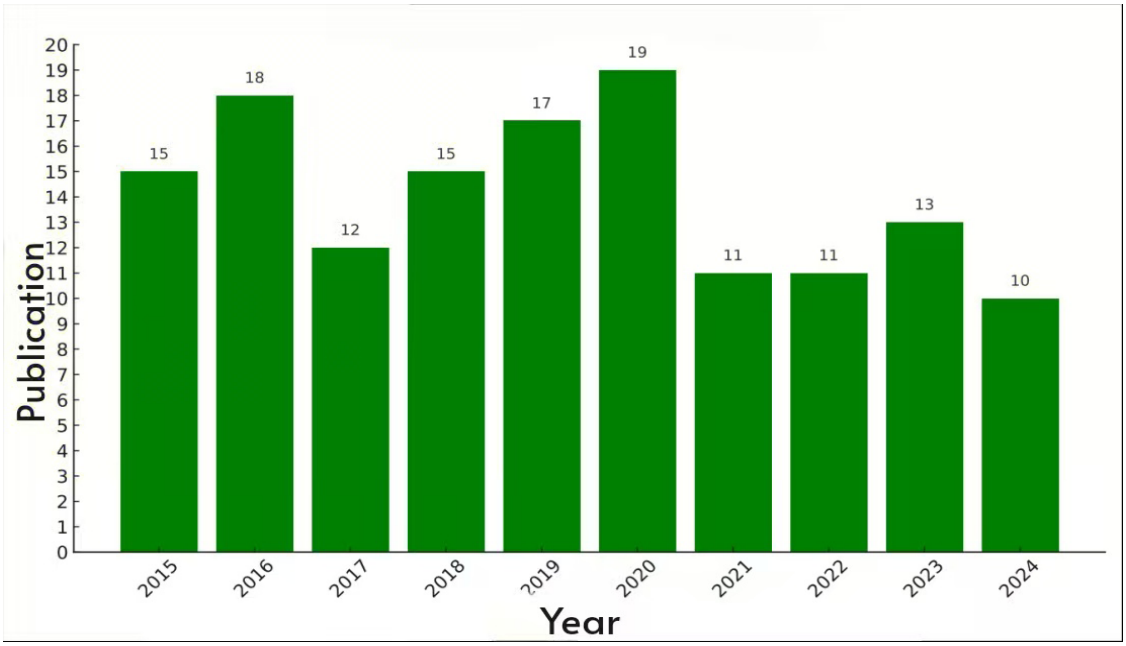

Figure 1. Trends in publications on THs and PCa research.

4.4. Dual-map overlay analysis of journals

The dual-map overlay generated by CiteSpace reveals the interdisciplinary knowledge flow characteristics in the field of THs and PCa research. The core paths are represented by two prominent knowledge transfer directions: the first is Molecular/Biology/Immunology → Molecular/Biology/Genetics (yellow path, z = 5.367, f = 9810), indicating that findings in immunology exert a profound influence on the field of genetics. For example, the interaction between thyroid antibodies (e.g., TPOAb/TgAb) and the PCa immune microenvironment (e.g., PD-1/PD-L1 signaling) [31] has promoted research on epigenetic regulatory mechanisms in genetics, such as methylation of the THRB gene promoter [32]. The second path is Medical/Clinical/Healthcare → Molecular/Biology/Genetics (green path, z = 2.151, f = 4300), indicating a relatively weak feedback from clinical medicine to basic research. This may result from barriers in translating clinical observational data (e.g., the correlation between TSH levels and PCa staging) into molecular mechanisms. On one hand, heterogeneity in thyroid function assessment standards (e.g., TSH cut-off values) complicates mechanistic studies; on the other hand, multi-omics integration techniques for clinical samples (e.g., single-cell sequencing) are not yet widespread, limiting in-depth analysis of molecular pathways. For instance, clinical observations of thyroid dysfunction in patients with castration-resistant prostate cancer (CRPC) [33] have stimulated functional studies on DIO2/DIO3 metabolic enzymes, yet relevant translational outcomes remain limited [34]. The dual-map overlay analysis (**Figure 2**) suggests that research on THs and PCa follows a "basic-to-clinical" model, with deep integration of immunology and genetics emerging as the dominant trend, while clinical translation remains constrained by data heterogeneity and technical barriers.

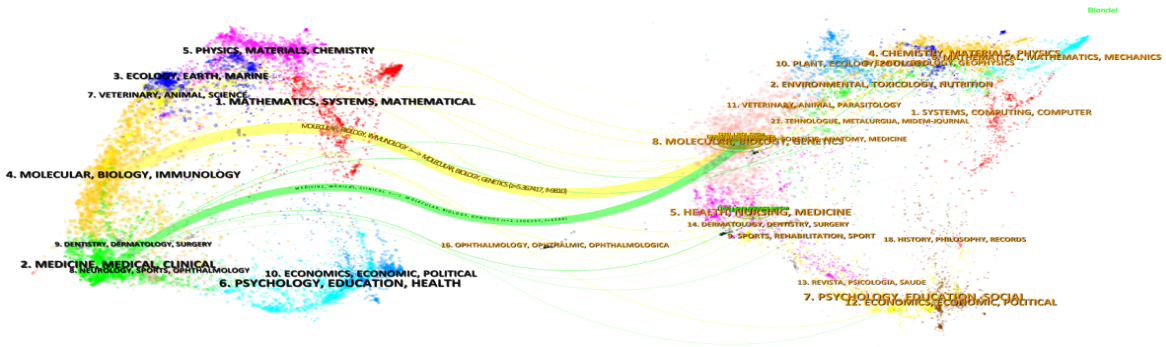

Figure 2. Dual-map overlay showing interdisciplinary knowledge flows in THs and PCa research.

4.5. Analysis of co-cited publications

Based on a co-citation network analysis of 141 publications (**Figure 3**), the field of THs and PCa research has formed three core knowledge clusters, exhibiting notable temporal evolution and translational potential. These clusters correspond to three major research themes:

(1) THs Signaling Pathways and Tumor Proliferation: This cluster focuses on how THs promote tumor proliferation via classical nuclear receptors (e.g., THRA, THRB) and non-classical signaling pathways (e.g., PI3K/AKT, integrin αvβ3).

(2) Thyroid Dysfunction and Clinical Prognosis: Research in this cluster reveals a close relationship between thyroid dysfunction and clinical outcomes in PCa.

(3) THs and Metabolic Reprogramming: Studies indicate that THs drive glycolysis via the AMPK/mTOR pathway [35], while thyroid hormone disruptors (e.g., bisphenol A) can induce lipid metabolism abnormalities and promote dedifferentiated phenotypes [36]. This suggests that THs not only contribute to tumor growth and metastasis but also play a critical role in the metabolic reprogramming of tumor cells.

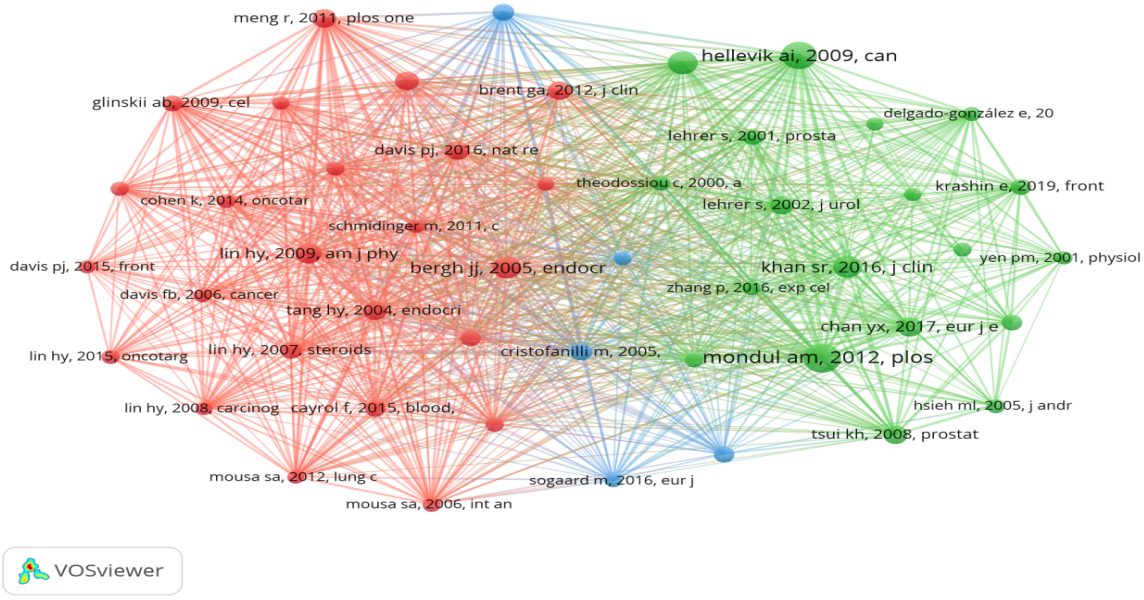

Figure 3. Visualization of the co-citation network of references.

4.6. Analysis of research hotspots and keywords

4.6.1. Co-occurrence analysis of keywords

Based on co-occurrence analysis of keywords from the selected publications using VOSviewer (**Figure 4**), the study identified five major research clusters, each representing distinct research directions and themes within the field. Each cluster reflects the specific focus of different disciplines on the relationship between THs and PCa.

(1) Red cluster: Focuses on the molecular mechanisms of PCa development, with core keywords including "prostate cancer," "thyroid hormone," "expression," "cell proliferation," and "biomarker," highlighting researchers' strong interest in the role of THs in regulating gene expression and cell proliferation during PCa pathogenesis.

(2) Yellow cluster: Primarily pertains to studies on thyroid hormone receptors, encompassing keywords such as "androgen receptor," "nuclear receptor," "estrogen receptor," and "gene expression." This cluster indicates that researchers are particularly interested in the potential regulatory roles of THs through interactions between nuclear receptors and sex hormone receptors within PCa cells, representing a significant current research direction.

(3) Green cluster: Emphasizes the effects of THs on tumor cells, with core keywords including "in vitro," "gene," "proliferation," "hypothyroidism," and "angiogenesis." This cluster reflects laboratory-based investigations into the effects of THs on PCa cell growth, differentiation, and tumor angiogenesis.

(4) Blue cluster: Relates to epidemiological studies, with keywords such as "prostate cancer," "cancer," "breast cancer," "mortality," and "meta-analysis," highlighting the role of THs across different cancer types, particularly in male populations and postmenopausal women.

(5) Purple cluster: Focuses on the association between THs levels and cancer risk, with keywords including "triiodothyronine," "thyroxine," "serum triiodothyronine," and "carcinoma." This cluster reflects the ongoing academic interest in the relationship between THs levels and PCa risk.

Through co-occurrence analysis, the diversity and complexity of research in the field of THs and PCa are clearly observed, with clusters interweaving, indicating a shift toward multidisciplinary and integrative research directions.

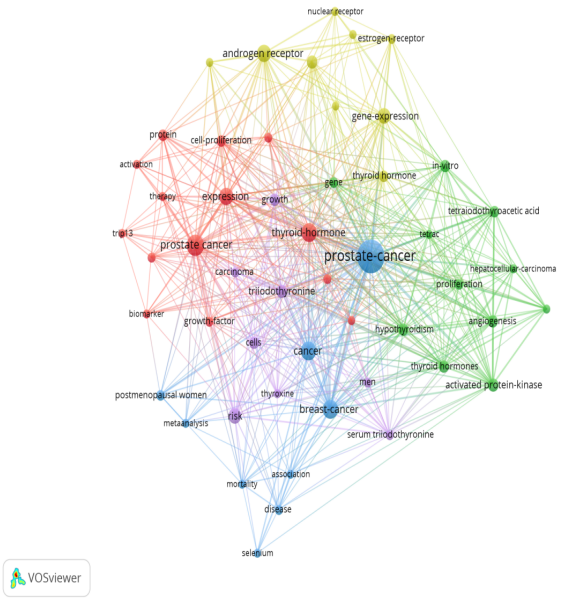

Figure 4. Co-occurrence network of keywords in THs and PCa research.

4.6.2. Burst analysis of keywords

Using CiteSpace, we conducted a burst analysis of keywords from the collected publications (**Figure 5**) and identified the 15 keywords with the most rapidly increasing citation frequencies between 2015 and 2024, revealing the research evolution and emerging hotspots in the field.

Between 2015 and 2017, research hotspots focused on the biological functions and mechanisms of THs, with keywords such as "postmenopausal women," "thyroid hormone receptor," and "cancer prevention" exhibiting strong bursts, reflecting substantial attention to the role of THs in different populations, particularly postmenopausal women, and their potential in cancer prevention [37,38,39].

During 2016–2017, the keywords "prostate cancer cells" and "carcinoma cells" emerged as bursts, indicating that research at the cellular level of PCa gradually became a focus, with attention shifting from clinical populations to cellular model studies.

After 2018, the period from 2019 to 2020 saw the most concentrated keyword bursts, particularly for "gene expression" and "thyroid hormones," with burst strengths of 1.88 and 1.83, respectively. This indicates that considerable attention was given to the mechanisms by which THs regulate gene expression, cell growth, and their roles in tumorigenesis.

From 2020 onward, the keywords "beta" and "carcinoma" exhibited significant bursts, reflecting researchers' efforts to explore the effects of THs on tumor progression, metastasis, and apoptosis. Meanwhile, the bursts of the keywords "breast" and "thyroid hormone" also signify the expansion of research in this field, with increasing focus on the roles of THs in other cancer types, such as breast cancer.

Through burst analysis, it can be observed that the research focus in this field has gradually expanded from fundamental molecular mechanisms to the clinical applications across different cancer types, particularly regarding the association between THs and cancer risk.

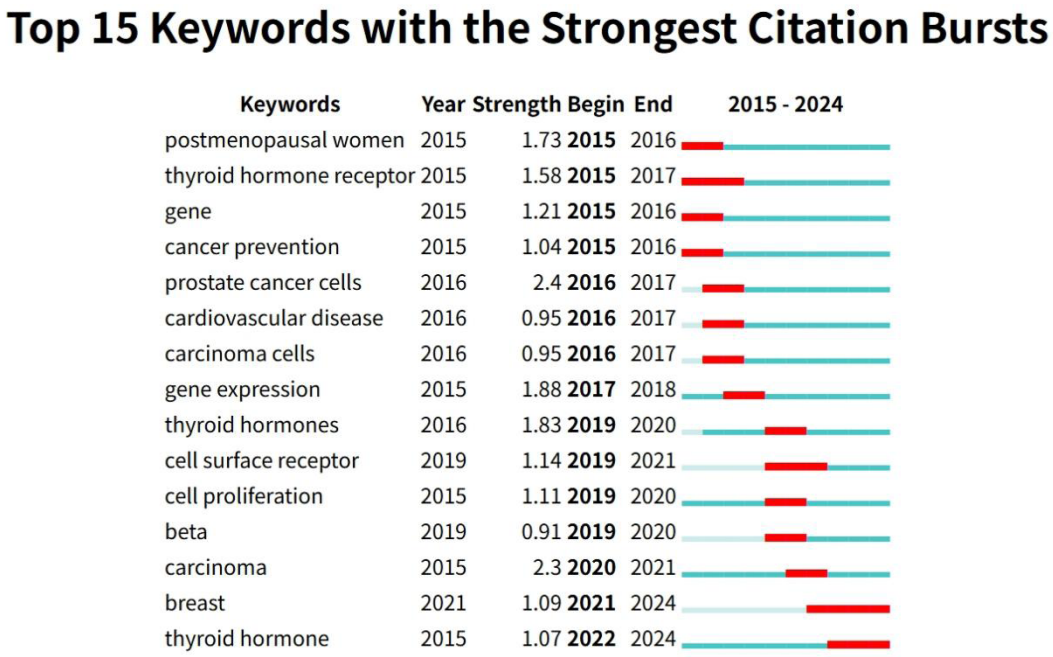

Figure 5. Burst analysis of keywords in research on THs and PCa.

5. Discussion

Existing studies consistently suggest that THs play a significant yet complex role in the initiation and progression of PCa. However, these effects are not unidirectional but rather dualistic. On one hand, epidemiological findings indicate that hyperthyroid states or elevated T3/T4 levels may increase PCa risk, whereas hypothyroidism may confer protective effects. On the other hand, certain cellular and animal studies have shown that T3 can inhibit tumor growth. This paradox suggests that multiple factors, including hormone concentration, receptor subtype distribution, local metabolic status, and tumor molecular subtype heterogeneity influence the role of THs in PCa.

Mechanistically, THs regulate the biological behaviors of PCa through multiple signaling pathways. The classical nuclear receptor pathway, mediated by TRα/β, modulates gene transcription, influencing cell cycle proteins, invasion-related factors, and AR expression, thereby indirectly promoting PCa cell proliferation and invasion. Concurrently, non-genomic mechanisms, particularly the MAPK/PI3K–Akt signaling pathway mediated by integrin αvβ3, exert rapid regulatory effects on metabolic reprogramming, cell migration, and angiogenesis in PCa. Moreover, the DIO enzyme family, key regulators of local TH metabolism, exhibits pathologically significant expression in prostate tissue: DIO2 upregulation increases local T3 levels, promoting proliferation, whereas DIO3 degrades T3 to establish a "locally hypothyroid" microenvironment, facilitating immune evasion by tumors. Collectively, these mechanisms indicate that THs may maintain a "dynamic balance" in PCa, rather than solely exerting pro- or anti-tumor effects.

Clinically, the relationship between THs and PCa holds potential translational value. Serum levels of TSH, FT3, and FT4 may serve as auxiliary biomarkers for prediction and prognosis; when combined with PSA and molecular subtyping, they could enhance the accuracy of individualized risk assessment. The influence of THs on the AR pathway and metabolic regulation suggests that thyroid functional status should be integrated into PCa stratification frameworks to optimize therapeutic decision-making. Furthermore, therapeutic strategies targeting DIO enzyme regulation or integrin αvβ3 blockade may represent novel treatment approaches for CRPC. Notably, thyroid function may also affect the efficacy and adverse effect management of androgen deprivation therapy (ADT) and novel AR pathway inhibitors, highlighting.

6. Conclusion

This study systematically reviews the research progress on the role of THs in the initiation and progression of PCa. Current evidence indicates that the effects of THs are bidirectional, potentially promoting tumor progression via nuclear receptors and non-genomic pathways, while also exerting anti-tumor effects under specific conditions. These outcomes depend on multiple factors, including hormone levels, receptor subtypes, local metabolic status, and the tumor microenvironment. Bibliometric analyses reveal that research hotspots in this field are gradually shifting from epidemiological associations toward molecular mechanisms and clinical translation. However, current studies are still limited by small sample sizes, heterogeneous stratification criteria, and lack of clinical validation. Future studies should focus on large-scale, multicenter prospective cohorts and multi-omics integrative analyses to elucidate the precise roles of THs in PCa and explore their potential as predictive biomarkers and therapeutic targets.

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Disclosure statement

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