



Bibliometric analysis and description of research trends on T cells in psoriasis over the past two decades (2003–2022)

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ABSTRACT

Background: It is now understood that T cells play a key role in the occurrence and development of psoriasis. Herein, a bibliometric analysis was conducted to summarize the content and trends of T cell-related research in psoriasis.

Methods: A bibliometric analysis was conducted on publications pertaining to T cells in psoriasis between 2003 and 2022 retrieved from the Web of Science Core Collection (WoSCC) database using tools such as CiteSpace, the Bibliometrix R package, and VOSviewer.

Results: The study included a total of 3595 articles authored by 14,188 individuals, including all coauthors in article bylines. The Laboratory for Investigative Dermatology at Rockefeller University, led by James G Krueger, has made significant contributions to this field through focusing on the pathogenesis of psoriasis and exploring the potential of using biological agents to treat psoriasis. Furthermore, targeted inhibitors have significantly impacted the treatment of psoriasis, with researchers focusing on small-molecule targeted drugs as a new area of research that could potentially replace biological agents.

Conclusions: Research has established the efficacy and long-term safety of targeted inhibition of T cell-related targets. Deucravacitinib, a psoriasis treatment drug targeting TYK2 as an allosteric inhibitor, has attracted significant attention and raised high expectations.

1. Introduction

Current evidence suggests that approximately 125 million people worldwide have psoriasis [1]. Psoriasis is a chronic, systemic, and inflammatory skin disease that results from the interplay between genetic and environmental factors and imposes a huge physical and psychological burden on patients [2]. Although the pathogenesis of psoriasis is not yet fully understood, many studies have shown that T cells play a key role in psoriasis [3]. In the past few years, extensive studies on the role of T cells in the pathogenesis and progression of psoriasis have led to the development of several potent inhibitors that target T cell-related targets, which have been successfully applied in clinical settings [4] and resulted into a paradigm shift in the management of this patient population [5].

Bibliometrics is a quantitative and qualitative analytical tool that enables processing of a vast amount of literature to identify the main research topics and future research hotspots [6,7]. Despite its limitations, bibliometrics has demonstrated an indispensable role

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in numerous scientific fields [8]. Previous bibliometric studies on the subdivision of psoriasis have mainly focused on psoriatic arthritis [9], psoriasis comorbidities [10], and research differences between different countries [11]. To our knowledge, no bibliometric study has assessed T cells in psoriasis. Additionally, we found that bibliometric analyzes of psoriasis articles from different periods concluded that biologic treatments were the focus of research. This can be attributed to the dual critical roles that T cells play in psoriasis, serving as both contributors to the disease's pathogenesis and potential targets for therapeutic intervention [12–14]. Herein, a bibliometric analysis was conducted to assess research on T cells in psoriasis; we systematically analyzed research content, development trends, and noteworthy achievements regarding T cells in psoriasis over the past 20 years and identified potential directions for future research.

2. Methods

2.1. Data sources and filtering strategies

To avoid errors caused by database updates, the Web of Science Core Collection (WoSCC) database was retrieved on March 7, 2023, using the following search terms: TS=(Psoriasis) AND TS=(T cell OR T cells OR T lymphocyte OR Thymocyte). We limited the search to articles published between January 1, 2003, and December 31, 2022, and restricted the publication type to original articles and reviews in English. A total of 4804 articles were retrieved. By reading the titles and abstracts, we excluded the articles whose research topics were not psoriasis and T cells and the retracted and duplicated articles. Finally, 3595 articles were included in the bibliometric analysis.

2.2. Bibliometric analysis

We used CiteSpace [15] and the Bibliometrix R package [16] to analyze the research articles, including details on the author, institutions, countries, periodicals, references and the analysis of keywords. VOSviewer [17] was used for co-occurrence, cluster analysis and visualization of the results.

3. Results

3.1. General characteristics

A total of 4804 publications were retrieved from the WOS database, and 3595 were screened out based on our exclusion criteria. As shown in Fig. 1A, the annual number of articles published was relatively stable between 2003 and 2015, with no more than 200 research articles on T cells in psoriasis published yearly. From 2016 to 2022, more than 200 articles were published annually, showing rapid development. Fig. 1B displays the top 10 countries and regions that have published the most articles in this field. Despite ranking

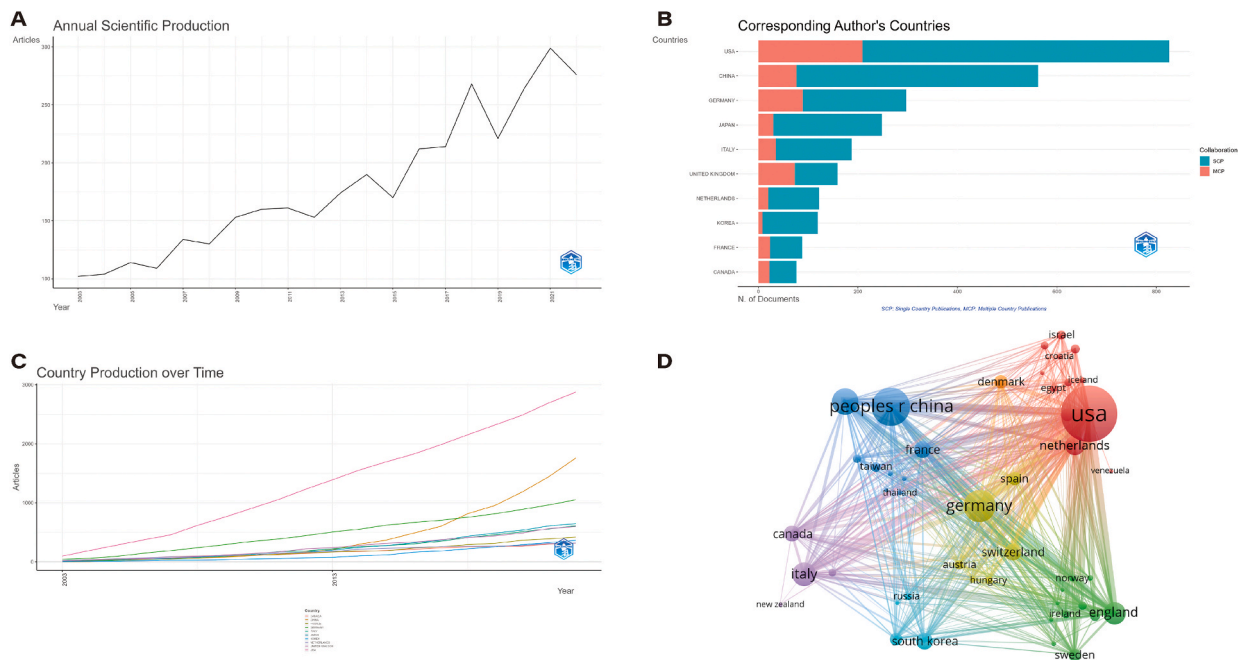


Fig. 1. General characteristics (A) Annual number of articles published; (B) Types of published articles by country; (C) Trends in the number of articles published by country; (D) Cluster analysis of cooperative relations among countries.

second in the total number of published articles, China has more Single Country Publications (SCP) than Germany. However, the number of Multiple Country Publications (MCPS) from China was lower than in Germany, while the UK had the highest proportion of MCPS. According to Fig. 1C, the US has consistently ranked first in the number of publications in this field over the past 20 years. China overtook Germany in the total number of published articles in 2018 and ranked second. The cluster analysis of cooperative relationships among countries in Fig. 1D shows that the US, China and Germany were the main leaders in international cooperation.

3.2. Author and institution

Out of the 3595 articles analyzed, there were 14,188 authors from 3048 institutions worldwide, resulting in an average of 3.95 authors per article. The top 10 authors in this field are presented in Table 1, with six being from the USA, including four from Rockefeller University. The number of publications by the top 10 authors in different years is presented in Fig. 2A, with James G Krueger, Johann E Gudjonsson, and Peter C M van de Kerkhof being the most consistent authors in terms of publishing over 20 years. Fig. 2B shows that Rockefeller University maintained its top position regarding the number of publications, while the University of Michigan and the University of California San Francisco frequently exchanged the second and third places in the total number of publications. Fig. 2C and D shows James G Krueger and his institution, Rockefeller University, as the leading authors and institutions in global cooperation.

3.3. Journal analysis

A total of 3595 articles were published in 687 journals. Table 2 shows the top 10 journals with an average impact factor of 7.56, including JCR Q1 (n = 8) and Q2 (n = 2) subjournals. The cited references were from 7941 journals. Table 3 shows the top 10 most cited journals, with an average impact factor of 39.70, including JCR Q1 (n = 9) and Q2 (n = 1) journals. Fig. 3 shows the citation paths between journals, with the citing journals on the left and the cited journals on the right. These results showed that journals focusing on Molecular/Biology/Genetics were cited by journals on Molecular/Biology/Immunology, Medicine/Medical/Clinical and Dentistry/Dermatology/Surgery.

3.4. Reference analysis

A total of 100,773 references were cited in 3595 publications. Fig. 4A illustrates the top 10 co-cited references and the detailed information is shown in Table 4, including 5 reviews and 5 articles. Specifically, there were reviews on the overall understanding of psoriasis published in 2009 [18] and 2015 [19], as well as reviews on the immunology of psoriasis published in 2007 [20], 2009 [21], and 2014 [22]. In 2007, ZHENG Y et al. found that IL-22 produced by Th17 cells mediates IL-23-induced acanthosis and skin inflammation by activating STAT3 as a key mechanism in psoriasis [23]. In 2008, LOWES MA et al. discovered psoriasis exhibits a mixed Th1 and Th17 inflammatory milieu [24]. In 2011, a study by CAI YH et al. revealed that dermal dendritic cells (DC) and macrophages (M ϕ) are responsible for producing IL-23, and IL-23 activated gamma delta T cells are the main producers of IL-17, which leads to the progression of psoriasis [25]. In 2012, LEONARDI C and PAPP KA reported the efficacy of Ixekizumab, an IL-17 monoclonal antibody, and Brodalumab, an IL-17 receptor antibody, in treating psoriasis. Fig. 4B shows the top 25 publications with the highest burst citation intensity. The most recent article with a sudden burst in references is Hawkes JE's review, which summarizes the crucial role of IL-23/IL-17 and T cells in psoriasis, leading to a significant shift in the disease pathogenesis model [26]. Fig. 4C shows the cluster analysis results of references, including 6 key clusters, which are: "Chemokine Receptors", "Guselkumab", "Single-cell RNA Sequencing", "IL-22", "Efalizumab", and "IL-20". Fig. 4D shows the timeline relationships of key clusters, with significant emphasis on "Single-cell RNA Sequencing".

Table 1
Core author list.

Author	No. of Publications	No. of Local Citations	H_index	Country	Affiliation	Affiliation Ranking by No. of Publications
KRUEGER JG	111	5275	64	USA	ROCKEFELLER UNIV	1
GUDJONSSON JE	42	1397	28	USA	UNIV MICHIGAN	2
WANG Y	40	313	15	China	CAPITAL MED UNIV	61
SUAREZ-FARINAS M	38	1496	31	USA	ROCKEFELLER UNIV	1
LOWES MA	37	2782	34	USA	ROCKEFELLER UNIV	1
MENTER A	34	984	27	USA	BAYLOR UNIV	69
GOTTLIEB AB	33	550	25	USA	ROCKEFELLER UNIV	1
VAN DE KERKHOFF PCM	33	299	17	The Netherlands	RADBOUD UNIV NIJMEGEN	7
NESTLE FO	32	2477	27	United Kingdom	KINGS COLL LONDON	4
LI J	31	360	13	China	CENT S UNIV	46

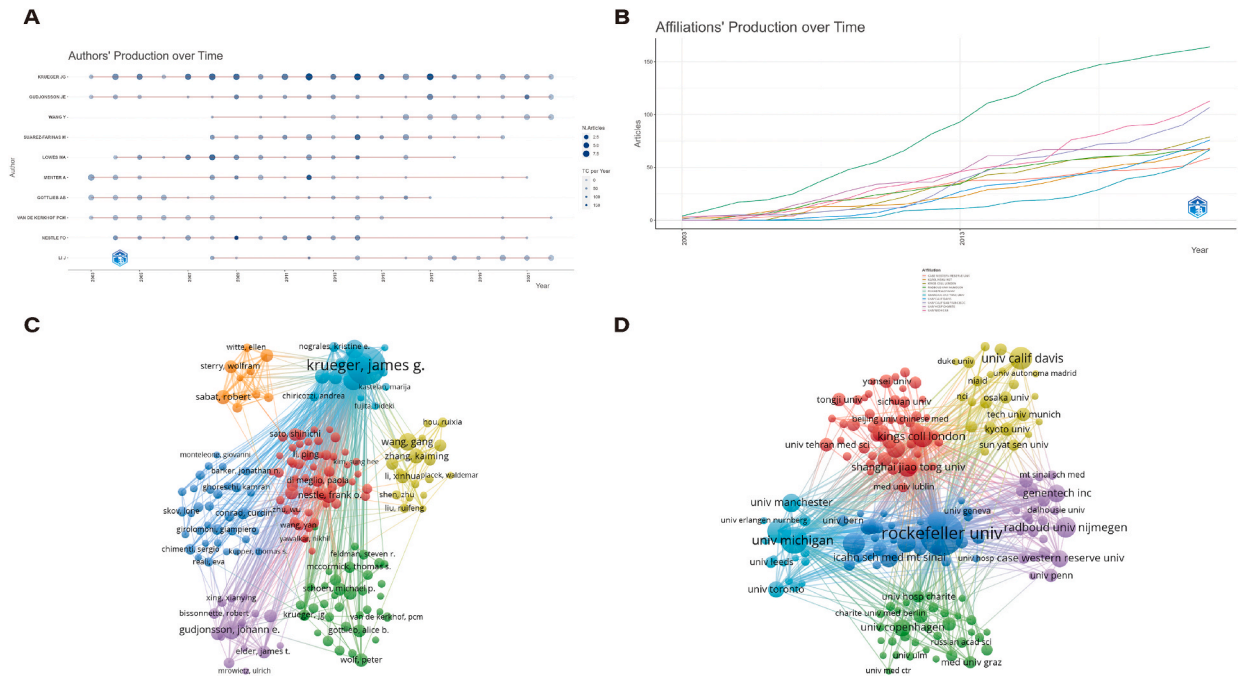


Fig. 2. Author and institution (A) Author's annual contribution; (B) Trends in the number of articles published by institutions; (C) Cluster analysis of collaborative relationships among authors; (D) Cluster analysis of cooperative relations among institutions.

Table 2
Journal publications.

Sources	Articles	IF (2021)	JCR
J INVEST DERMATOL	235	7.59	Q1
BRIT J DERMATOL	140	11.11	Q1
EXP DERMATOL	121	4.51	Q1
FRONT IMMUNOL	119	8.78	Q1
J IMMUNOL	94	5.43	Q2
INT J MOL SCI	83	6.20	Q1
J DERMATOL SCI	81	5.40	Q1
J ALLERGY CLIN IMMUN	71	14.29	Q1
J EUR ACAD DERMATOL	61	9.22	Q1
ARCH DERMATOL RES	57	3.03	Q2

Table 3
Citations of journals.

Sources	Articles	IF (2021)	JCR
J IMMUNOL	13398	5.43	Q2
J INVEST DERMATOL	13152	7.59	Q1
BRIT J DERMATOL	7432	11.11	Q1
J EXP MED	6856	17.57	Q1
J AM ACAD DERMATOL	5434	15.48	Q1
IMMUNITY	4499	43.47	Q1
NATURE	4386	69.50	Q1
NAT IMMUNOL	4161	31.25	Q1
NEW ENGL J MED	3926	176.08	Q1
J CLIN INVEST	3750	19.47	Q1

3.5. Analysis of research hotspots

A total of 4716 keywords were assigned to 3595 publications, later expanded to 5971 Keywords Plus. 439 keywords that appeared more than 10 times were screened (Fig. 5A). Fig. 5B depicts the trend of major keywords, revealing a gradual increase in attention towards T cell research over time. Fig. 5C displays the trend of research topics over time. The progression of research topics can be

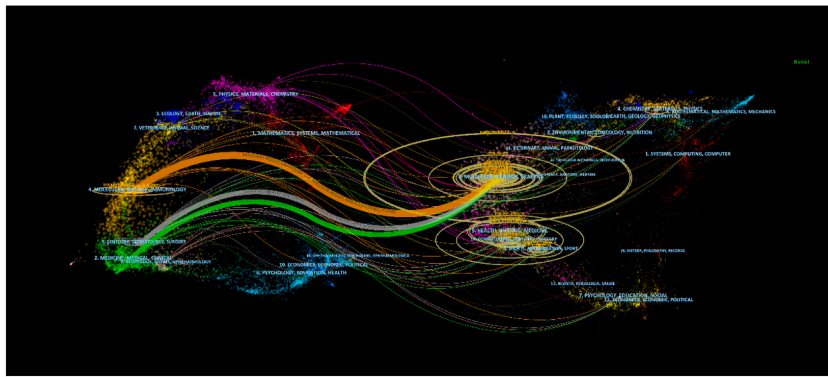


Fig. 3. Dual-map overlay of journals related to T cells in psoriasis. This figure shows the citation paths between journals, with the citing journals on the left and the cited journals on the right.

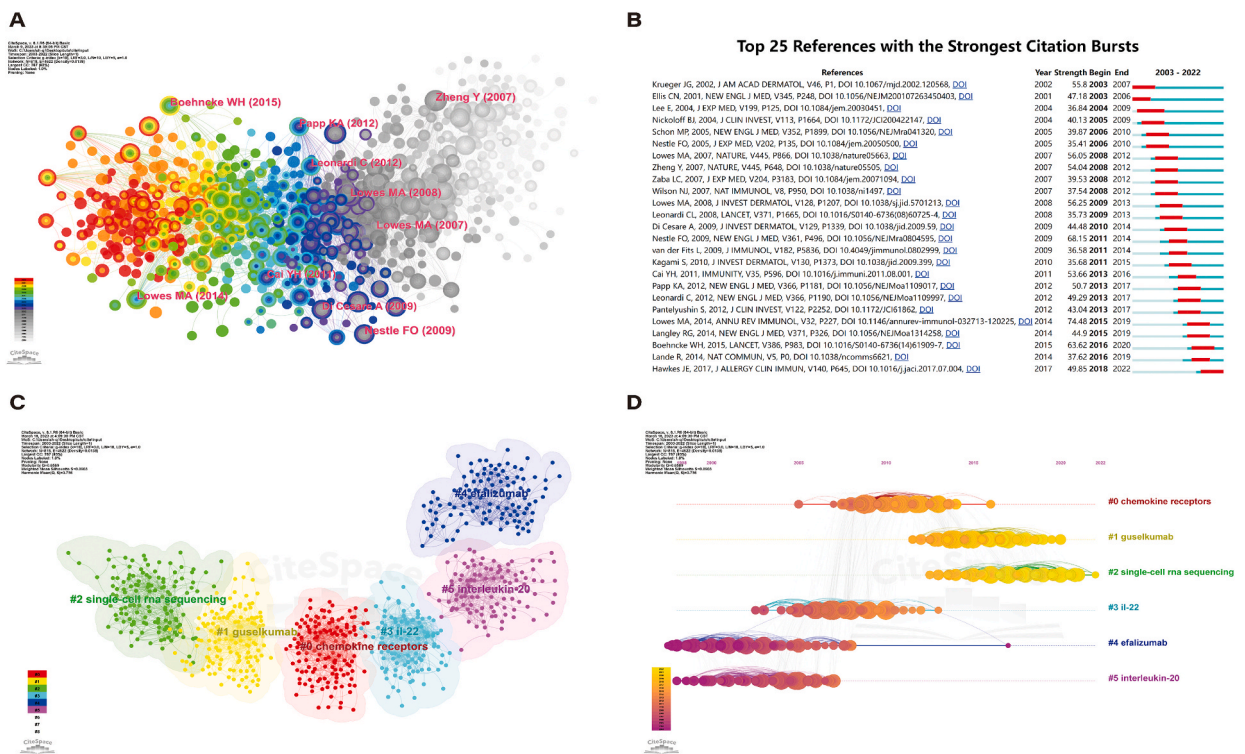


Fig. 4. Reference analysis (A) Co-occurrence of references; (B) Outbreak citing references; (C) Clustering of references; (D) Reference timeline diagram.

observed, from the earliest focus on Acitretin to the current interest in Tofacitinib (anti-JAK), which encompasses six therapeutic drugs. In chronological order, Acitretin, Alefacept (anti-CD2), Infliximab (anti-TNF- α), Interleukin-12/23 monoclonal antibody, Secukinumab (anti-IL-17A) and Tofacitinib. Fig. 5D shows the cluster analysis results of keywords, including 6 key clusters, which are: "Expression", "Interferon Gamma", "Efficacy", "ROR Gamma T", "Susceptibility Loci", "Cutaneous T-cell Lymphoma". Fig. 5E shows the timeline relationships of key clusters; the main focus is "Interferon Gamma".

4. Discussion

The past few years have witnessed unprecedented scientific progress, especially in research on T cells in psoriasis. Among the 10 countries with the largest number of published articles, only China is a developing country, indicating that developed countries mainly dominate this field. A higher total number of published articles and proportion of international cooperation was observed among the 7 European countries compared to the 3 Asian countries, which could be linked to racial and ethnic disparities in psoriasis [27,28]. Major

Table 4
Top 10 most cited references.

Title	Journal	Count	Centrality	DOI	Type	Author	Year
Psoriasis	NEW ENGL J MED	175	0	10.1056/NEJMRA0804595	Review	NESTLE FO	2009
Immunology of psoriasis	ANNU REV IMMUNOL	160	0.02	10.1146/ANNUREV-IMMUNOL-032713-120225	Review	LOWES MA	2014
Psoriasis vulgaris lesions contain discrete populations of Th1 and Th17 T cells	J INVEST DERMATOL	160	0.04	10.1038/SJ.JID.5701213	Article	LOWES MA	2008
Pathogenesis and therapy of psoriasis	NATURE	157	0.01	10.1038/NATURE05663	Review	LOWES MA	2007
Interleukin-22, a T(H)17 cytokine, mediates IL-23-induced dermal inflammation and acanthosis	NATURE	152	0.05	10.1038/NATURE05505	Article	ZHENG Y	2007
Psoriasis	LANCET	140	0.01	10.1016/S0140-6736(14)61909-7	Review	BOEHNCKE WH	2015
Brodalumab, an anti-interleukin-17-receptor antibody for psoriasis	NEW ENGL J MED	125	0.11	10.1056/NEJMoa1109017	Article	PAPP KA	2012
Anti-interleukin-17 monoclonal antibody ixekizumab in chronic plaque psoriasis	NEW ENGL J MED	125	0.13	10.1056/NEJMoa1109997	Article	LEONARDI C	2012
Pivotal role of dermal IL-17-producing $\gamma\delta$ T cells in skin inflammation	IMMUNITY	123	0.02	10.1016/J.IMMUNI.2011.08.001	Article	CAI YH	2011
The IL-23/Th17 axis in the immunopathogenesis of psoriasis	J INVEST DERMATOL	117	0.01	10.1038/JID.2009.59	Review	DI CESARE A	2009

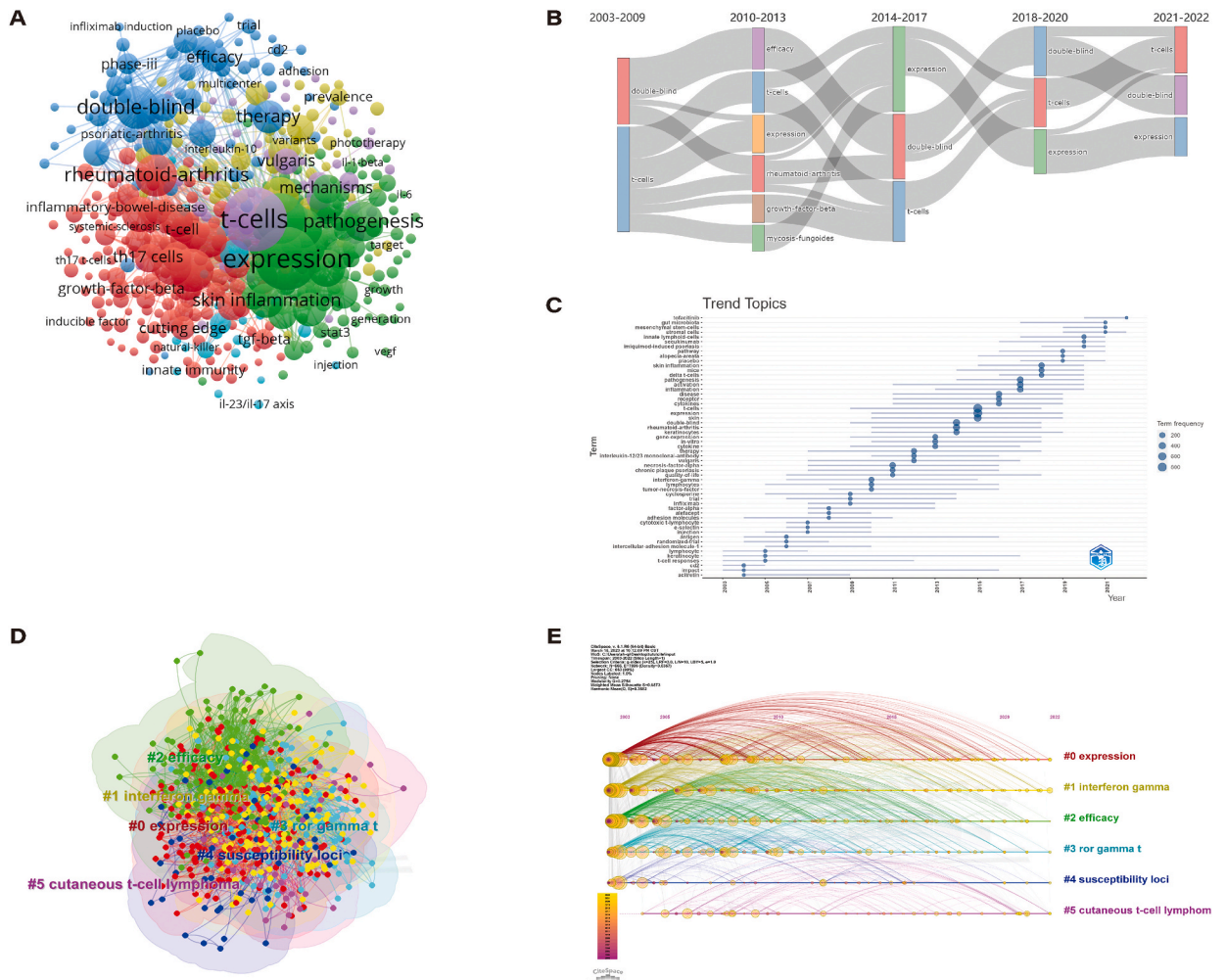


Fig. 5. Analysis of research hotspots (A) Keyword co-occurrence; (B) Change trend of keywords; (C) Trend topics; (D) Keyword clustering; (E) Keyword timeline diagram.

contributions in this field have come from The Laboratory for Investigative Dermatology at Rockefeller University, led by James G Krueger, focusing on the pathogenesis of psoriasis and exploring the potential of using biological agents to treat psoriasis [29]. The effect of Efalizumab (anti-CD11a) on psoriasis was reported in 2002 [30]. Subsequently, different biologics such as Alefacept [31], Fontolizumab (anti-IFN- γ) and Infliximab [32], have been evaluated to further investigate the immune mechanism of psoriasis. With the emergence of new technologies, they have also introduced gene set enrichment analysis (GSEA) [33], RNA-seq [34], and Single-cell RNA seq [35] to study the mechanisms underlying psoriasis. In 2022, they noted that the small-molecule targeted drug Janus kinase (JAK) 1–3 inhibitor was effective against moderate to severe psoriasis, but safety concerns remain, and no JAK inhibitor has received regulatory approval for psoriasis. While Tyrosine kinase (TYK) 2 regulates signalling and functional responses downstream of key IL-12, IL-23, and type I IFN receptors in the pathogenesis of psoriasis, targeting the active sites of the TYK2 regulatory domain instead of the catalytic domain avoids the toxicity associated with JAK1-3 inhibitors. Moreover, laboratory and clinical evidence showed that inhibition of the TYK2 regulatory domain could alleviate psoriasis, and it was speculated that TYK2 regulatory domain inhibitors are effective in the clinical treatment of psoriasis [36,37].

The keyword clustering results of the references showed the foundational content of T cell research in psoriasis. "Chemokine receptors" are a group of receptors on the surface of immune cells, which are mainly used to recruit immune cells to inflammatory tissues following chemokine signals and play a key role in the pathogenesis of psoriasis [38]. Studies have found that inhibition of C–C chemokine receptor type (CCR) 4, CCR6 and C-X-C Motif Chemokine Receptor (CXCR) 2 could alleviate psoriasis [39–41]. However, it has been established that inhibition of CCR10 can aggravate psoriasis [42]. The mechanism of action of the chemokine receptors in psoriasis is currently under investigation. Currently, no chemokine receptors have been approved for treating psoriasis. "Guselkumab (anti-IL-23p19)" and "Efalizumab" are both biologic agents for the treatment of psoriasis, with Efalizumab being the representative biologic agent of earlier years [43]. It was withdrawn from the market in 2009 due to its association with severe adverse reactions of progressive multifocal leukoencephalopathy [44]. Guselkumab was launched 10 years after Efalizumab and has become a representative biological agent in recent years, with considerable curative effect [45] and long-term safety [46]. However, a network meta-analysis revealed multiple psoriasis drugs, including Infliximab, Ixekizumab (anti-IL-17A), Bimekizumab (anti-IL-17A and IL-17F), and Risankizumab (anti-IL-23) are currently the most effective drugs for treatment [47]. Our findings suggest that most studies have focused on "Single-cell RNA Sequencing", studying the transcriptome of individual cells in tissues. Single-cell RNA Sequencing revealed that Th17 cells, CD8⁺ T cells, DC3 cells, macrophages, ILCs and KCs all play a key role in the occurrence and development of psoriasis [48]. Both "IL-22" and "IL-20" are important cytokines in psoriatic inflammation. IL-22 transmits phosphorylation signals downstream through Janus kinase (JAK) 1 and tyrosine kinase (TYK) 2, including the mitogen-activated protein kinase (MAPK) pathway (p38 kinase, ERK1/2, MEK1/2 and JNK), STAT3, STAT1 and STAT5 [49]. It is widely acknowledged that a moderate amount of IL-22 helps maintain immune homeostasis, while excessive levels can induce inflammation and lead to psoriasis [50]. IL-20 has also been reported to have anti-inflammatory [51] and pro-inflammatory [52] effects on psoriasis. Currently, the mechanism of IL-22 and IL-20 regulation in psoriasis is not fully understood, which has hindered the development of inhibitors targeting them for clinical applications.

The keyword analysis results of the included articles showed the hotspots of T cell research in psoriasis. The earliest and the newest topics in the hot topic trend are psoriasis drugs. The drugs in question are Acitretin (traditional drug), followed by the evolving biologics Alefacept, Infliximab, Interleukin-12/23 monoclonal antibody and Secukinumab. According to the latest findings, small molecule targeted drug Tofacitinib has demonstrated the importance of targeted inhibitors in treating psoriasis, indicating that small-molecule targeted drugs are the next frontier after biologics. It was observed that the trend in the targets of inhibitor drugs shifted from CD to TNF, IL-12/23, IL-17, and now JAK, based on the types of antagonistic targets, and the route of administration has changed from the injection of biological agents to oral small-molecule drugs. The results of keyword clustering demonstrate the main issues being addressed in the study. "Expression" and "Efficacy" are terms used to study psoriasis drug treatment. Tofacitinib, for example, reduces the expression of inflammatory genes and exerts its efficacy against psoriasis [53]. Secukinumab reduces ACE2 expression and may benefit psoriasis patients at risk of SARS-CoV-2 infection [54]. "Interferon Gamma" is the only member of type II interferon and can aggravate psoriasis by activating the Janus kinase (JAK)-signal transducer and activator of transcription (STAT) signalling pathway [55]. HuZAF (anti-IFN- γ), which directly targets IFN- γ , is ineffective against psoriasis patients [56]. Nonetheless, Tofacitinib, which targets JAKs downstream of IFN- γ , has been reported to improve symptoms in psoriasis patients with higher baseline IFN- γ levels [57]. This phenomenon has not been fully understood, and it is speculated that it may be related to the functional plasticity of Th17 cells [58]. "Retinoid-related orphan receptor gamma T (ROR γ T)" is essential for Th17 cell differentiation as well as for IL-17A, IL-17F and IL-22 production, and inhibition of ROR γ T is considered a promising strategy for psoriasis [59]. Several ROR gamma T inhibitors have been reported effective against psoriasis in recent years [60–62]. However, no ROR γ T inhibitor has been approved for clinical treatment. "Susceptibility Loci" have been identified in a multi-ethnic psoriasis population [63]. More than 70 psoriasis susceptibility loci have been discovered so far [64]. Combining the results into a genetic risk score can yield a more accurate prediction of psoriasis [65]. Cutaneous T-cell Lymphoma (CTCL) exhibits clinical manifestations similar to psoriasis, frequently leading to misdiagnosis as psoriasis during the early stages [66]. A variety of biological agents for psoriasis can aggravate the progression of CTCL. Therefore, to ensure treatment safety, clinicians should use biological agents for psoriasis patients after obtaining histopathological results [67].

Overall, the progress of targeted inhibitors over the last 20 years has resulted in significant changes in the treatment of psoriasis, with the goal of treatment now being complete or near-complete symptom improvement. While biological agents have shown considerable efficacy and long-term safety for psoriasis, their macromolecular protein structure makes oral administration impossible, and injection can be inconvenient for patients, leading to poor compliance. Small molecule drugs based on the targeted inhibition principle that can be administered orally have become an increasingly popular research topic. The main targets of small molecule drugs that have been approved or are close to being approved for marketing are phosphodiesterase 4 (PDE4), JAK1-3, TYK2, etc. A systematic review and meta-analysis showed that Apremilast (anti-PDE4) is safe and effective in treating psoriasis [68]. According to

German guidelines, Apremilast is the first-line oral small-molecule drug [69]. Inhibitors targeting JAK1-3 have not been approved for treating psoriasis due to safety concerns, and selective JAK inhibitors may provide similar efficacy and better safety profile than non-selective JAK inhibitors [70]. Therefore, Deucravacitinib, a selective inhibitor targeting the same family of TYK2, has been approved to treat psoriasis [71]. Besides, the latest randomized double-blind clinical trial study showed that Deucravacitinib was superior to Apremilast in multiple efficacy endpoints and was well tolerated [72,73].

Our study provides a strong connection between laboratory and clinical research in the field of psoriasis. Over the past 20 years, as our understanding of the role of T cells in psoriasis has deepened, we have discovered numerous key targets for the disease. The development of psoriasis treatment focuses on selecting more effective and safer targets for precise inhibition. Biological agents have emerged as a reliable choice for therapeutic drugs due to their proven efficacy and safety. However, their delivery methods still possess certain limitations. As a result, the latest oral small molecule drugs are gaining attention and generating expectations. These findings serve as a comprehensive knowledge summary for researchers dedicated to this field. However, this article still has certain limitations. We exclusively relied on English articles included in the WOS database over the past 20 years, potentially resulting in incomplete results. The inclusion and exclusion of articles were manually conducted by the authors, introducing the possibility of subjective errors and biased outcomes. Additionally, the clustering algorithms utilized in the analysis software may inherently restrict the full display of all results. Nonetheless, this is the first bibliometric article focusing on the field of T cells in psoriasis and can lead researchers to conduct in-depth exploration of promising small molecule drugs for psoriasis.

5. Conclusions

The bibliometric analysis of T cell research in psoriasis revealed that each subtype of T cells has a significant role in the pathogenesis and progression of psoriasis. Although the pathogenesis of psoriasis has not been fully clarified, targeted inhibition of T cell-related targets has been validated to have considerable efficacy and long-term safety. Deucravacitinib, a psoriasis treatment drug targeting TYK2 as an allosteric inhibitor, has attracted significant attention and raised high expectations.

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Data availability statement

All the results found during this study are available in this article.

Ethics statement

Review and/or approval by an ethics committee was not needed for this study because this study was an analysis based on the literature and did not involve human or animal studies.

CRedit authorship contribution statement

Junchen Li: Writing – original draft, Data curation, Conceptualization. **Jianfeng Zhang:** Writing – original draft, Formal analysis. **Chenqi Guo:** Writing – original draft, Investigation. **Peng Lin:** Writing – original draft, Project administration. **Qian Shen:** Writing – original draft, Visualization. **Haiyue Lin:** Writing – original draft, Validation. **Yu Zhang:** Writing – review & editing, Supervision.

Declaration of competing interest

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