



Review Research Hotspots in Psoriasis: A Bibliometric Study of the Top 100 Most Cited Articles

Oana Mirela Tiucă ^{1,2,3}, Silviu Horia Morariu ^{2,3,*}, Claudia Raluca Mariean ^{1,4}, Robert Aurelian Tiucă ^{1,5,6}, Alin Codruț Nicolescu ⁷ and Ovidiu Simion Cotoi ^{4,8}

- ¹ Doctoral School of Medicine and Pharmacy, University of Medicine, Pharmacy, Science, and Technology George Emil Palade of Targu Mures, 540142 Targu Mures, Romania
- ² Dermatology Department, University of Medicine, Pharmacy, Science, and Technology George Emil Palade of Targu Mures, 540142 Targu Mures, Romania
- ³ Dermatology Clinic, Mures Clinical County Hospital, 540342 Targu Mures, Romania
- ⁴ Pathophysiology Department, University of Medicine, Pharmacy, Science, and Technology George Emil Palade of Targu Mures, 540142 Targu Mures, Romania
- ⁵ Endocrinology Department, University of Medicine, Pharmacy, Science, and Technology George Emil Palade of Targu Mures, 540142 Targu Mures, Romania
- ⁶ Endocrinology Department, Mures Clinical County Hospital, 540139 Targu Mures, Romania
- ⁷ Agrippa Ionescu Emergency Clinical Hospital, 011773 Bucharest, Romania
- ⁸ Pathology Department, Mures Clinical County Hospital, 540011 Targu Mures, Romania
- * Correspondence: silviu.morariu@umfst.ro

Abstract: (1) Introduction: Psoriasis is a chronic, immune-mediated disease that negatively impacts patients' quality of life and predisposes them to cardiovascular or metabolic diseases. This paper aims to summarize the knowledge structure and future directions in psoriasis research by means of bibliometrics. (2) Material and methods: The Thomson Reuters Web of Science database was interrogated using preestablished keywords. A list of the top 100 most cited articles focusing solely on psoriasis was compiled and analyzed. VOSviewer software was used to assess and visualize collaboration networks, citation, co-citation and co-wording analysis, and bibliographic coupling. (3) Results: The articles were written by 902 authors from 20 countries and were published in 31 journals. The United States was at the forefront of this field. Griffiths, CEM had the most citations, while the most prolific institution was Rockefeller University, New York City. Pathogenesis, especially key-pathogenic factors, immune pathways, and epidemiology were the most discussed topics. Work published in the last decade focused on the use of biologics. Keywords such as "quality of life", "efficacy", and "necrosis-factor alpha" have been widely used. (4) Conclusion: Research interest regarding psoriasis is high, leading to the rapid development of this field. Treatment modalities, especially novel-targeted therapies, immune pathways, and an integrative approach to such cases are receiving great interest and represent research hotspots in the future.

Keywords: psoriasis; therapy; immunopathogenesis; bibliometry; citation impact

1. Introduction

Psoriasis is a chronic, immune-mediated disease that negatively impacts patients' quality of life (QoL). In the last published global report [1], the World Health Organization reported an increasing prevalence of psoriasis, ranging between 1.5% and 5% in developed countries [2]. Its etiopathogenesis is complex, with genetic predisposition, an altered immune response, and various triggering factors concurring in the development of this disease [3,4]. Clinically defined by cutaneous erythema, scaling, and induration, and in some cases by joint and nail involvement, this disease seems to predispose patients to a higher risk of developing cardiovascular disease, diabetes, dyslipidemia, and metabolic syndrome [5,6].



Citation: Tiucă, O.M.; Morariu, S.H.; Mariean, C.R.; Tiucă, R.A.; Nicolescu, A.C.; Cotoi, O.S. Research Hotspots in Psoriasis: A Bibliometric Study of the Top 100 Most Cited Articles. *Healthcare* 2023, *11*, 1849. https:// doi.org/10.3390/healthcare11131849

Academic Editors: Oriana Simonetti and Wolfgang J. C. Uter

Received: 7 April 2023 Revised: 21 June 2023 Accepted: 21 June 2023 Published: 26 June 2023



Copyright: © 2023 by the authors. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (https:// creativecommons.org/licenses/by/ 4.0/). A continuous stream of research is being conducted in relation to psoriasis, aiming to shed light on the pathogenesis, management, and therapeutic outcomes of this disease. A thorough study of scientific advances in a specific field may lead to improvements in the diagnosis and treatment of various diseases. In order to evaluate progress in psoriasis research and its future directions, a bibliometric analysis is of great use.

This concept was proposed by Pritchard [7] and uses statistical parameters to identify emerging trends and collaboration patterns between research constituents. Citations are the most forthright measure of a paper's impact [8]. Additionally, performance analysis, which illustrates the contributions of research constituents, and science mapping, which depicts the relationships between them, provide additional insight into the academic significance of research papers.

Nevertheless, the use of bibliometry in medical research is relatively new. It has been sparsely utilized, especially regarding cancers [9–13]. However, to the best of our knowledge, this is the first paper to address overall research directions in psoriasis, taking into account bibliometric algorithms based on the top 100 most cited articles referring to this disease.

2. Material and Methods

2.1. Search Strategy and Data Collection

The search was conducted on the Thomson Reuters Web of Science (WoS) database on 15 January 2023. The following keywords were used: "psoriasis", "plaque psoriasis", "guttate psoriasis", "erythrodermic psoriasis", and "pustular psoriasis", separated by the Boolean OR. Articles from all fields were searched across the entire database without regard to article type or study design. Citations recorded in the indexing database are as follows: Science Citation Index Expanded, Social Sciences Citation Index, Conference Proceeding Citation Index-Social Science and Humanities, Conference Proceedings Citation Index-Science, and Emerging Sources Citation Index.

The search returned 56,731 results, that were afterward screened. Abstracts and letters were excluded. Only full-length English articles were considered. The returned articles were sorted by citation count using Paladugu's method [14]. Articles focusing on psoriatic arthritis or other inflammatory or autoimmune skin disorders were excluded. Papers referring only to psoriasis-specific topics, such as pathogenesis, treatment modalities, or outcomes, were reviewed. Search and screening of the results were made by two independent researchers to ensure relevance to the selected topic. Disagreements were resolved by discussion between the two involved researchers. The Prisma diagram (Figure 1) exemplifies the workflow. A list of the top 100 most cited articles was compiled and analyzed for various parameters over the next four weeks [15–114].

2.2. Bibliometric Analysis

Information regarding the journal, authorship, institution, publication year, and study design was extracted for the selected articles. Clarivate Journal Citation Reports was used for each journal's 2021 and 5-year impact factors. Full data regarding the selected articles were generated from the WoS database as an Excel spreadsheet and as a plain text file.

Publication-related metrics were analyzed with Microsoft Excel software. Science mapping and data visualization were performed with VOSviewer software (Version 1.6.19-2023). VOSviewer is a graphical user interface-based free software first developed by van Eck and Waltman in 2010 [115], with the latest version launched on 23 January 2023. It allows the analysis and visualization of different collaboration patterns between research constituents.

The following performance analysis parameters were evaluated: publication-related metrics (total publications, solo-authored publications, co-authored publications) and citation-related metrics (total citations and citation rate). The citation rate was calculated by dividing the total number of citations by the number of years since publication. The bibliometric and knowledge structure of the research field is evaluated in this paper by using the following science mapping techniques: citation, co-word, co-authorship

analysis, and bibliographical coupling. The counting method was set at full counting. To limit spelling differences in authors' or institutions' names an additional thesaurus file, that gives consistent labels to the same word spelled differently, was generated, and used when appropriate.

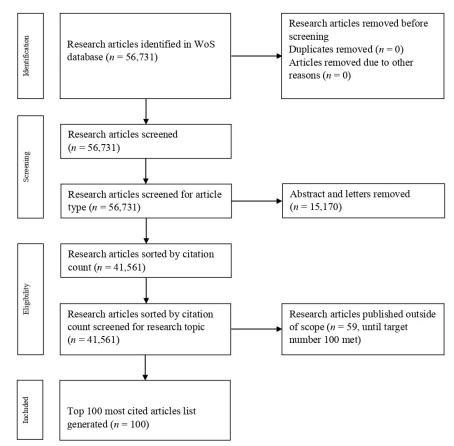


Figure 1. Prisma diagram of workflow.

3. Results

3.1. Citation Analysis

The total citation count for the analyzed articles was 68,691, with a median of 553.5 and a mean of 686.91. Eighty-four were original articles, while sixteen were reviews. Pathogenesis and epidemiology were the topics most discussed (n = 66), followed by management (n = 45) and genetics (n = 9). Twenty-six articles focused on the use and effectiveness of novel targeted immune therapies, such as biologics, in the management of moderate-to-severe plaque psoriasis. Pustular psoriasis was addressed in two articles [45,63].

Within the top 100, the citation count ranged between 405 for "Clinical response to adalimumab treatment in patients with moderate to severe psoriasis: Double-blind, randomized controlled trial and open-label extension study" by Gordon, K et al. [28] and 2145 for "Severe psoriasis—oral therapy with a new retinoid" by Fredriksson, T et al. [106].

The articles were published between 1969 and 2020. The oldest article was "Generalized pustular psoriasis—a clinical and epidemiological study of 104 cases" by Baker, H et al. [45], while the newest one was "Pathophysiology, clinical presentation, and treatment of psoriasis: a review" by Armstrong, AW et al. [73]. They had 421 and 420 citations, respectively. These two articles also have the lowest and highest citation rates, 8 and 211, respectively. Table 1 shows the top 100 articles and their respective citation rates. Figure 2 illustrates the distribution of the articles by decade.

Rank	Authors	Journal	Publication Year	Total Citations	Citation Rate
1	Fredriksson et al. [106]	Dermatologica	1978	2145	49
2	Nestle et al. [66]	New England Journal of Medicine	2009	2041	157
3	Parisi et al. [48]	Journal of Investigative Dermatology	2013	1507	167
4 5	Zheng et al. [62]	Nature	2007 2015	1450	97 191
5 6	Boehncke et al. [81] Langley et al. [103]	Lancet New England Journal of Medicine	2015 2014	1338 1337	191 167
7	Gelfand et al. [102]	JAMA—Journal of The American Medical Association	2014	1337	84
8	Van der Fits et al. [54]	Journal of Immunology	2009	1290	99
9	Lowes et al. [72]	Nature	2007	1291	86
10	Griffiths et al. [85]	Lancet	2007	1244	83
11	Leonardi et al. [41]	Lancet	2008	1237	88
12	Parrish et al. [77]	New England Journal of Medicine	1974	1296	27
13	Papp et al. [40]	Lancet	2008	1105	79
14	Rapp et al. [89]	Journal of The American Academy of Dermatology	1999	1078	47
15	Nair et al. [47]	Nature Genetics	2009	1010	78
16 17	Lowes et al. [56]	Annual Review of Immunology, Vol 3	2014 2003	918 934	115 49
17	Leonardi et al. [44] Schon et al. [67]	New England Journal of Medicine New England Journal of Medicine	2003	934 877	49 52
10 19	Reich et al. [60]	Lancet	2005	847	50
20	Cargill et al. [17]	American Journal of Human Genetics	2003	843	56
21	Nestle et al. [78]	Journal of Experimental Medicine	2005	812	48
22	Tyring et al. [42]	Lancet	2006	825	52
23	Lowes et al. [96]	Journal of Investigative Dermatology	2008	776	55
24	Papp et al. [25]	New England Journal of Medicine	2012	743	74
25	Krueger et al. [110]	Archives of Dermatology	2001	786	37
26	Leonardi et al. [22]	New England Journal of Medicine	2012	733	73
27	Di Cesare et al. [109]	Journal of Investigative Dermatology	2009	780	60
28	Wolk et al. [52]	European Journal of Immunology	2006	727	45
29 30	Neimann et al. [80] Strange et al. [15]	Journal of The American Academy of Dermatology Nature Genetics	2006 2010	739 747	46 62
31	Lee et al. [57]	Journal of Experimental Medicine	2010	689	38
32	Chaudhari et al. [39]	Lancet	2001	724	34
33	Menter et al. [21]	Journal of The American Academy of Dermatology	2008	677	48
34	Krueger et al. [18]	New England Journal of Medicine	2007	617	41
35	Tsoi et al. [49]	Nature Genetics	2012	661	66
36	Henseler et al. [92]	Journal of The American Academy of Dermatology	1985	660	18
37	Christophers [86]	Clinical And Experimental Dermatology	2001	623	30
38	Griffiths et al. [30]	New England Journal of Medicine	2010	634	53
39 40	Lin et al. [65]	Journal of Immunology	2011	627	57 55
$\begin{array}{c} 40\\ 41 \end{array}$	Marrakchi et al. [63] Nograles et al. [108]	New England Journal of Medicine British Journal of Dermatology	2011 2008	610 611	55 44
42	Langley et al. [98]	Annals of The Rheumatic Diseases	2005	607	36
43	Arican et al. [105]	Mediators of Inflammation	2005	594	35
44	Sonkoly et al. [68]	Plos One	2007	577	38
45	Detmar et al. [71]	Journal of Experimental Medicine	1994	584	21
46	Griffiths et al. [29]	Lancet	2015	592	85
47	Sano et al. [107]	Nature Medicine	2005	544	32
48	Papp et al. [16]	British Journal of Dermatology	2005	573	34
49	Mcinnes et al. [38]	Lancet	2013	561	62
50	Hammarstrom et al. [55]	Proceedings of The National Academ	1975	581	12
51 52	Mrowietz et al. [33]	Archives of Dermatological Research	2011	550	50
52 53	Stern et al. [91] Saurat et al. [43]	Journal of Investigative Dermatology British Journal of Dermatology	2004 2008	558 550	31 39
55 54	Lebwohl et al. [75]	New England Journal of Medicine	2003	539	77
55	Rendon et al. [94]	International Journal of Molecular Sciences	2019	520	173
56	Chan et al. [53]	Journal of Experimental Medicine	2006	510	32
57	Stern et al. [101]	New England Journal of Medicine	1979	544	13
58	Rachakonda et al. [95]	Journal of The American Academy of Dermatology	2014	535	67
59	Lebwohl [84]	Lancet	2003	498	26
60	Gordon et al. [76]	New England Journal of Medicine	2016	517	86
61	Gottlieb et al. [100]	Nature Medicine	1995	507	19
62	Hollox et al. [90]	Nature Genetics	2008	508	36
63	Michalek et al. [20]	Journal of The European Academy of Dermatology and Venereology	2017	481	96 20
64 65	Pathirana et al. [46] Henseler et al. [34]	Journal of The European Academy of Dermatology and Venereology Journal of The American Academy of Dermatology	2009 1995	507 500	39 19
66	Wada et al. [24]	Plos One	2012	300 492	49
	, add et an [=1]		2012	1/4	

Table 1. Top 100 articles ranked by citation count [15–114].

Rank	Authors	Journal	Publication Year	Total Citations	Citation Rate
67	Ellis et al. [32]	JAMA—Journal of The American Medical Association	1986	503	14
68	Kagami et al. [27]	Journal of Investigative Dermatology	2010	462	39
69	Blauvelt et al. [36]	Journal of The American Academy of	2017	493	99
70	Takeshita et al. [87]	Journal of The American Academy of	2017	472	94
71	Nickoloff et al. [99]	Journal of Clinical Investigation	2004	415	23
72	Davidovici et al. [88]	Journal of Investigative Dermatology	2010	477	40
73	Sommer et al. [58]	Archives of Dermatological Research	2006	477	30
74	Abrams et al. [31]	Journal of Clinical Investigation	1999	469	20
75	Sugiyama et al. [35]	Journal of Immunology	2005	444	26
76	Ma et al. [51]	Journal of Clinical Investigation	2008	451	32
77	Stern et al. [64]	New England Journal of Medicine	1997	487	19
78	Mehta et al. [74]	European Heart Journal	2010	488	41
79	Ellis et al. [113]	New England Journal of Medicine	2001	461	22
80	Melski et al. [70]	Journal of Investigative Dermatology	1977	483	11
81	Gisondi et al. [82]	British Journal of Dermatology	2007	468	31
82	Kurd et al. [112]	Archives of Dermatology	2010	474	40
83	Zenz et al. [97]	Nature	2005	442	26
84	Hawkes et al. [93]	Journal of Allergy And Clinical Immunology	2017	441	88
85	Gelfand et al. [79]	Archives of Dermatology	2005	446	26
86	Krueger et al. [111]	Journal of The American Academy of Dermatology	2002	431	22
87	Lebwohl et al. [19]	New England Journal of Medicine	2003	449	24
88	Naldi et al. [26]	Journal of Investigative Dermatology	2005	457	27
89	Gottlieb et al. [59]	Journal of The American Academy of Dermatology	2004	439	24
90	Farber et al. [69]	Dermatologica	1974	457	10
91	Homey et al. [114]	Journal of Immunology	2000	421	19
92	Chiricozzi et al. [61]	Journal of Investigative Dermatology	2011	437	40
93	Greb et al. [83]	Nature Reviews Disease Primers	2016	424	71
94	Armstrong et al. [73]	JAMA-Journal of The American Medical Association	2020	421	211
95	Reich et al. [37]	Journal of The American Academy of Dermatology	2017	420	84
96	Nair et al. [104]	American Journal of Human Genetics	2006	418	26
97	Trembath et al. [50]	Human Molecular Genetics	1997	410	16
98	Gordon et al. [28]	Journal of The American Academy of Dermatology	2006	405	25
99	Baker et al. [45]	British Journal of Dermatology	1968	420	8
100	Nickoloff et al. [23]	American Journal of Pathology	1991	406	13



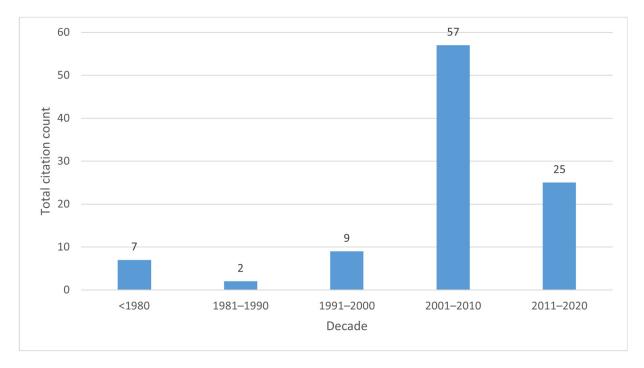


Figure 2. Articles distribution by decade.

Based on bibliometric algorithms that consider citation patterns, such as total citation number and the topic addressed in these papers, the analyzed articles were grouped into eight clusters. Cluster 1 is defined by 24 articles, clusters 2 and 3 by 16 articles each, cluster 4 by 15 articles, cluster 5 by 14 articles, cluster 6 by 9 articles, cluster 7 by 5 articles, and cluster 8 by a single article. The previously mentioned clusters are shown in Figure 3 as map-based connections. Each color represents a thematic cluster, whereas each node represents an author. The size of each individual node and font size is proportional to the number of citations, both related to the completed data set and to each individual cluster.

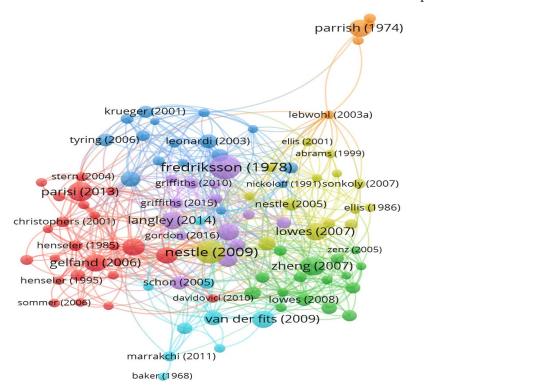


Figure 3. The bibliometric map of the selected articles based on citation patterns. (Cluster colors are as follows: cluster 1—red, cluster 2—green, cluster 3—blue, cluster 4—yellow, cluster 5—purple, cluster 6—turquoise, cluster 7—orange, cluster 8—pink) [15–114].

Griffiths, CEM, Krueger, JG, Papp, K, Krueger, GG, and Menter, A contributed to the greatest number of articles and received a total of 9646, 7669, 8944, 7492, and 6945 citations, respectively. Table 2 highlights the top 10 most cited authors.

Table 2. Most cited authors.

Rank	Author	Articles	Total Citations
1	Griffiths, CEM	12	9646
2	Krueger, JG	12	7669
3	Papp, K	11	8944
4	Krueger, GG	10	7492
5	Menter, A	10	6945
6	Langley, RG	8	5747
7	Lebwohl, M	8	6318
8	Nestle, F	8	6779
9	Gottlieb, A	7	4080
10	Reich, K	7	5026

The top 100 articles were published in 31 journals, which published between one and sixteen articles. *New England Journal of Medicine* (n = 16) published the greatest number of articles within the top 100 and had the highest number of citations (12,817). *Lancet* had

hammarstrom (1975)

the highest impact factor (202.73), published the third-highest number of articles (n = 10), and received the third-highest number of total citations (8966). *Dermatologica* had the highest average citation per publication (1301), having published 2 papers with a total of 2602 citations. Table 3 displays articles, citation count, and various journal metrics.

Table 3. Jo	urnal metrics	;.
-------------	---------------	----

Rank	Journal	Total Articles	Total Citations	2021 Impact Factor	5-Year Impact Factor	Average Citations/Publication
1	New England Journal of Medicine	16	12,817	176.08	125.16	801.06
2	Journal of The American Academy of Dermatology	12	6847	15.48	12.07	570.58
3	Lancet	10	8966	202.73	130.84	896.6
4	Journal of Investigative Dermatology	8	5377	7.59	8.38	672.13
5	British Journal of Dermatology	5	2623	11.11	9.29	524.6
6	Nature Genetics	4	2926	41.37	39.32	731.5
7	Journal of Immunology	4	2782	5.43	6.17	695.5
8	Journal of Experimental Medicine	4	2595	17.57	16.42	648.75
9	Nature	3	3183	69.5	63.58	1061
10	JAMA—Journal of The American Medical Association	3	2257	157.37	101.12	752.33
11	Archives of Dermatology	3	1706	4.78	4.45	568.67
12	Journal of Clinical Investigation	3	1335	19.47	19.23	445
13	Dermatologica	2	2602	N/A	N/A	1301
14	American Journal of Human Genetics	2	1260	11.04	12.87	630
15	Plos One	2	1069	3.75	4.06	534.5
16	Nature Medicine	2	1051	87.24	68.31	525.5
17	Archives of Dermatological Research	2	1025	3.03	3.19	512.5
18	Journal of The European Academy of Dermatology and Venereology	2	987	9.22	7.72	493.5
19	Annual Review of Immunology, Vol 32	1	917	32.48	35.19	917
20	European Journal of Immunology	1	726	6.68	6.09	726
21	Clinical and Experimental Dermatology	1	623	4.48	3.19	623
22	Annals of The Rheumatic Diseases	1	607	28	20.69	607
23	Mediators of Inflammation	1	594	4.52	5.6	594
24	Proceedings of The National Academy of Sciences of The United States of	of 1	581	12.77	13.45	581
25	Journal of Investigative Dermatology Symposium Proceedings	1	557	3.73	2.48	557
26	International Journal of Molecular Sciences	1	518	6.2	6.62	518
27	European Heart Journal	1	488	35.85	33.03	488
28	Journal of Allergy and Clinical Immunology	1	440	14.29	13.76	440
29	Nature Reviews Disease Primers	1	424	65.03	83.06	424
30	Human Molecular Genetics	1	410	5.12	5.99	410
31	American Journal of Pathology	1	406	5.77	5.48	406

3.2. Co-Authorship Analysis

The 100 analyzed articles summed 902 authors, out of which 167 contributed to more than 2 papers, while 18 authored more than 5 articles. Three papers were solo-authored [84,86,111]; the highest number of contributing authors for a paper was 136 [49]. Leonardi and Papp first authored the highest number of papers (n = 3). Griffiths, CEM (n = 12), Krueger, JG (n = 12), Papp, K (n = 11), Krueger, GG (n = 10), and Menter, A (n = 10) contributed to the greatest number of articles. The authors who contributed to more than five papers are shown in Figure 4 as map-based connections scored by average citations.

The authors contributing to the 100 articles originated from 322 institutions and 20 countries. The United States had the most citations (48,556), as well as the highest number of papers (n = 73). Germany ranked second, with 18,722 citations from 28 articles. The top five institutions that contributed to the papers were Rockefeller University (n = 13), the University of Manchester (n = 12), Probity Medical Research (n = 11), the University of Michigan, and the University of Utah (n = 10, each). Table 4 and Figure 5 depict the top 10 institutions, respectively, countries, that contributed to the top 100 most cited articles.

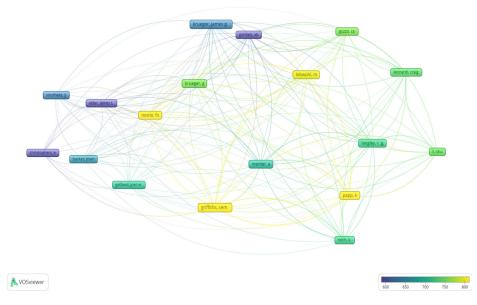


Figure 4. Map-based representation of authors contributing to more than 5 papers scored by average citations. (Color legend: the authors that had the most citations are depicted using yellow frames, while the least cited with purple frames) [15–114].

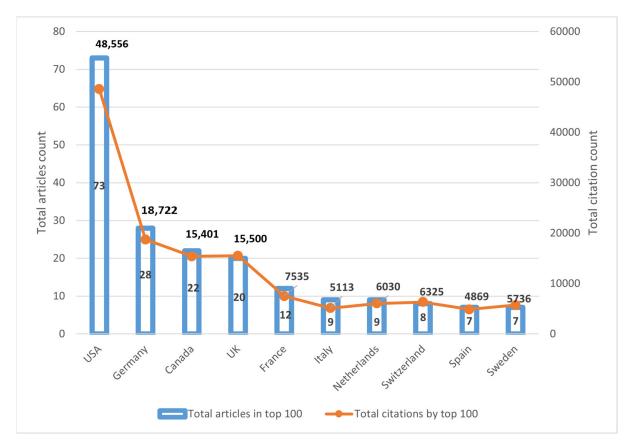


Figure 5. Top countries by total citation count and number of articles.

Rank	Institution	Country	Articles	Citations	Total Link Strength
1	Rockefeller University	USA	13	8201	7
2	University of Manchester	UK	12	9788	11
3	Probity Medical Research	Canada	11	8223	11
4	University of Michigan	USA	10	5384	7
5	University of Utah	USA	10	7097	10
6	Dalhousie University	Canada	9	6354	9
7	Harvard University	USA	9	6605	8
8	Penn University	USA	9	5473	4
9	Saint Louis University	USA	8	6282	8
10	Baylor University	USA	7	4474	7

Table 4. Institutions with the most articles.

3.3. Co-Word Analysis

A total of 471 unique keywords were identified from all articles. After removing keywords such as "psoriasis", "plaque psoriasis", "chronic plaque psoriasis", "severe plaque psoriasis", "vulgaris", "to-severe psoriasis", "psoriasis vulgaris", and "vulgaris lesions" that could affect the analysis, a minimum threshold of two occurrences was set for each keyword and the bibliometric map was generated. Based on occurrence, they were divided into 7 clusters, as follows: cluster 1 = 37 items, cluster 2 = 28 items, cluster 3 = 23 items, cluster 4 = 19 items, cluster 5 = 18 items, cluster 6 = 12 items, and cluster 7 with 8 items. Figure 6 displays them as map-based connections, while Figure 7 displays keywords occurrence density in the selected articles. A set of the 10 most used keywords was generated and presented in Table 5.

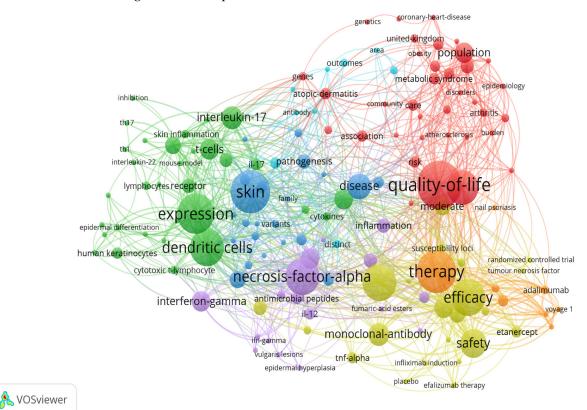


Figure 6. The scientometric map of the keywords from the analyzed articles. (Cluster colors are as follows: cluster 1—red, cluster 2—green, cluster 3—blue, cluster 4—yellow, cluster 5—purple, cluster 6—turquoise, cluster 7—orange) [15–114].

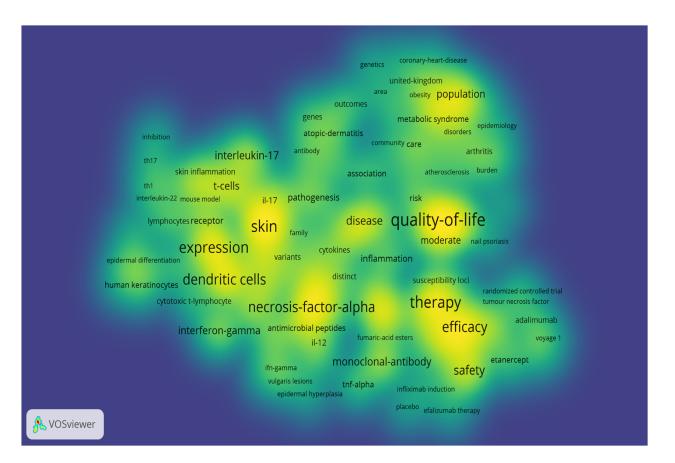


Figure 7. Research tendency based on the density of the keywords used in the 100 articles. (Color legend: the intensity of the yellow and green colors symbolize the frequency of respective keywords).

Rank	Keyword	No. of Occurrences	Cluster
1	Quality of life	17	1
2	Skin	15	3
3	Therapy	15	7
4	Expression	14	2
5	Dendritic cells	13	2
6	Double-blind	13	4
7	Efficacy	13	4
8	Necrosis factor alpha	13	5
9	Rheumatoid arthritis	12	1
10	Safety	10	4

Table 5. Top 10 most used keywords.

3.4. Bibliographical Coupling

Based on patterns of citing the same references, the 100 articles were divided into 10 clusters. Cluster 1 consists of 26 items, cluster 2 of 23 items, cluster 3 of 16 items, cluster 4 of 14 items, cluster 5 of 13 items, cluster 6 of 4 items, and clusters 7, 8, 9, and 10 of 1 item each. The map-based connections between all clusters can be found in Figure 8.

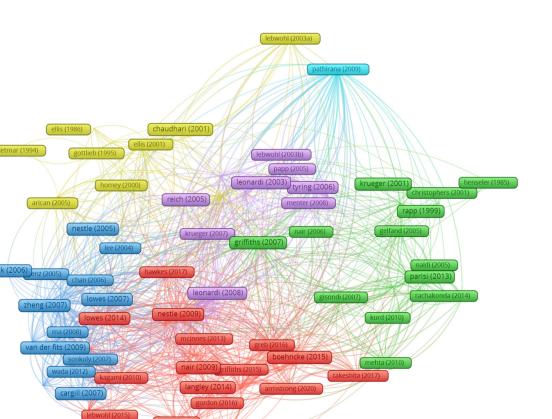




Figure 8. The scientometric map based on existing patterns of citing the same bibliography source. (Due to a high number of bibliographic clusters that led to an overview design when exporting from VOSviewer, this figure depicts the first six clusters, as follows: cluster 1—red, cluster 2—green, cluster 3—blue, cluster 4—yellow, cluster 5—purple, cluster 6—turquoise) [15–54,56–66,68–76,78–105,107–114].

3.5. Co-Citation Analysis

hollox (2008)

The analyzed articles summed 3645 references. A minimum threshold of one for each reference was set The most co-cited reference was the article of Rapp, SR et al. [89], which was co-cited by 19 other articles, with 1077 links with other articles and a total link strength of 1358, followed by Fredrickson, T et al. [106], also co-cited by 19 articles, but with lower links (636) and link strength (804), and Gelfand, JM and Leonardi, CL both co-cited 15 times. Figure 9 shows the density of co-cited references. Co-citation frequency is depicted using different intensities of yellow and green. The network of references co-cited more than five times is shown in Figure 10, scored by average citations. They were divided into 4 clusters, as follows: cluster 1 = 43 items, cluster 2 = 30 items, cluster 3 = 30 items, and cluster 4 = 23 items. References located among different clusters and with the highest link strengths are circled with black.

angg	yard e, 1965, j biol chem,
*cane stat branch, 1995, surv	
parrish ja, 1974, new engl j m	
melski jv, 1977, j invest derm elder jt, 1989, science, v243, krueger jg, 2000, j am acad de baker bs, 1984, brit j dermato ellis cn, 2001, new engl j med fredriksson t, 1978, dermatolo	
rapp sr, 1999, j am acad derma ^{burch prj, 1965, acta derm-ven} nair rp, 2006, am j hum genet, nestle fo, 2009, new engl j me naldi I, 2005, j invest dermat eckel rh, 2005, lancet, v365,	1976, brit med j. v2, p2
albert ma, 2001, Jama j am med	baden hp. 1972. biochim biophy

Figure 9. Co-citation tendency. (Color legend: the intensity of the yellow and green colors symbolizes the frequency of respective keywords) [15–114].

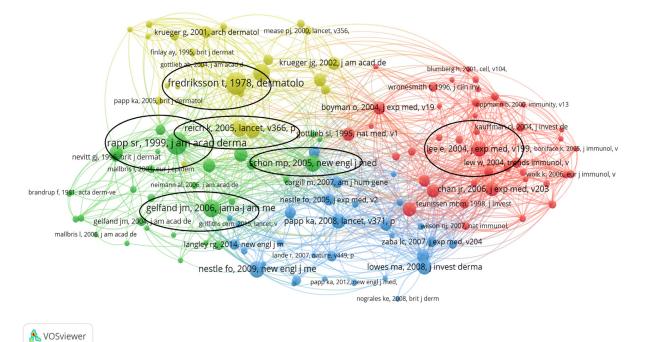


Figure 10. The scientometric map based on co-citation patterns. (Cluster colors are as follows: cluster 1—red, cluster 2—green, cluster 3—blue, cluster 4—yellow) [15–114].

4. Discussion

Psoriasis has attracted much interest in the scientific community over the years. In the face of constant evolution and novelties added to the field, it is of great use to maintain the connection to research areas of interest. Bibliometric analysis is able to handle, using quantitative methods, large amounts of literature, to avoid bias usually associated with qualitative-based systematic reviews, and to provide the knowledge structure and future trends of a research topic or field [116].

Among the top 100 articles reviewed, pathogenesis and epidemiology were the topics most often discussed, being the focus of 66 articles. Only 12 articles focused on the clinical and diagnostic aspects of the disease. As psoriasis remains mainly a clinical diagnosis, the emphasis is on better understanding and managing the disease. This points to the fact that the vast majority of the high-impact literature focused on understanding how and why psoriasis develops. This culminated in a trend to explore the role of different immune and inflammatory pathways since the beginning of the 2000s. As key contributors to the immunopathogenesis of psoriasis, keratinocytes provide antimicrobial peptides, such as S100A7 and LL-37, that bind to host DNA and form DNA-LL-37 complexes, which stimulate dendritic cells to produce IFN-alpha and activate myeloid dendritic cells. Activated dendritic cells produce mediators, including IL-12 and IL-23, that lead to T-cells differentiation into Type 1 [35] and Type 17 T-helper cells. Th17 cells play an important role in epithelial immune surveillance [109]. A special focus was set on key pathogenic factors of psoriasis, such as TNF- α , IL-6, IL-8, IL-17, IL-22, IL-23, and IFN-gamma, providing insightful information about disease mechanisms. [51,53,61,62,105]. Additionally, 10 papers have specifically addressed [15,17,47,49,50,56,63,68,104] the genetic basis of psoriasis, with extensive genetic testing that identified more than 50 psoriasis susceptibility loci [27,49,56]. The most important one, PSORS1 [88] is located within the major histocompatibility complex (MHC) on chromosome 6p21 and is directly linked to HLA-Cw6-allele. The gene variants of interest modulate immune pathways and processes that contribute to disease susceptibilities, such as antigen presentation, the IL-23/IL-17 axis, and the type I IFN pathway [49]. A distinctive and interesting approach to psoriasis genetics has been addressed by Sonkoly et al. [68] that identified a specific, dysregulated microRNA expression profile in psoriatic skin compared to healthy skin: miR-203 and miR-125b regulate keratinocyte proliferation and differentiation, while miR-21 inhibits T cell apoptosis. Consequently, research areas referring to therapeutical means have shifted from photochemotherapy and classical immunosuppressant therapies to novel targeted therapies in the last twenty years. Of the specific therapeutic options, this analysis identified the increasingly dominant trend in reporting the use of monoclonal antibodies, twenty-six out of forty-five articles referring to treatment options were focused only on the safety and effectiveness of such novel therapies. The significance of this trend is more accurately reflected by work published in the last decade because ten out of seventeen papers published in this timeframe were focused on treatment, out of which eight addressed specifically various monoclonal antibodies.

Even though only six articles focused specifically on disease comorbidities and four on QoL it is important to mention that these aspects have been uniformly addressed over time and mentioned in other papers, suggesting a constant focus of the research community on these topics. Treatment and management of psoriasis should not address only cutaneous manifestations, but also associated comorbidities and should aim to increase the QoL [117]. Biologics represent a cornerstone in the management of this disease because apart from alleviating skin lesions they seem to work up to a certain extent for associated comorbidities as well. Research focusing on biologics seems to steal the focus in the future as well, for further exploration.

The paper of Fredriksson et al. [106], published in 1978 in *Dermatologica*, was the most cited article in our analysis. It explored the effectiveness of a retinoic acid derivate in treating severe psoriasis. The study was significant at the time because apart from evaluating the effectiveness of a novel retinoid, it was the first article that introduced a currently worldwide used disease severity score, Psoriasis Area Severity Index (PASI).

PASI score is currently used in dermatology to assess disease severity and thus, allows the classification of psoriasis in mild, moderate, and severe. Further therapeutic options are selected taking into account various parameters, the PASI score being one of the strongest.

The oldest article included in this analysis, published by Baker et al. [45] focused on generalized pustular psoriasis and identified two etiologically and evolution-wise distinct subtypes of this rare form of psoriasis. The newest one, published by Armstrong et al. [73] offers a state-of-the-art review on clinical presentation, epidemiology, and therapeutic advancements. Due to the fact that the previously mentioned, most recent article included was published in 2020, ongoing research may significantly impact the top 100 articles over the next few years.

This study identified a significant difference in publication and citation patterns in the last two decades compared to before 2000, for which the articles total only 18 and 17.3% of the total citations. This can be explained by the fact that research published before the 2000s focused on pathogenesis, clinics, and conventional treatment options, thus laying the foundation for today's knowledge about psoriasis. Moreover, these last two decades represent the beginning of immunopathogenesis and biologics.

The collaboration network of authors, countries, and institutions provides an overall picture of the leading researchers in this field on different levels. The United States, Germany, Canada, and the United Kingdom are leaders in the field. Additionally, the most prolific institutions and authors originate in these countries, indicating greater research resources. Moreover, these countries possess some of the most comprehensive and better-updated National Registries, allowing a proper evaluation and follow-up of patients suffering from this disease while also serving as comprehensive research databases. Even though the University of Manchester ranked second, its total link strength is higher than that of the institution ranked first, suggesting a higher connection to other institutions analyzed. The authors that published the most papers were Griffiths, CEM (n = 12), Krueger, JG (n = 12), Papp, K (n = 11), Krueger, GG (n = 10), and Menter, A (n = 10). On the other hand, when analyzing the authors who contributed to more than five papers based on citations link strength, Nestle, FO, Griffiths, CEM, Papp, K, and Lebwohl, M are proven to be the most influential scholars in their field.

Keywords are a hallmark of the literature, and their analysis can shed light on research and trends in a specific field. The analyzed articles summed up 471 keywords. After setting a minimum threshold of 2 occurrences for each keyword, a bibliometric map based on the 145 eligible items was created and presented in Figure 6. Seven clusters, each defining a research area, were defined. The top 10 keywords with the highest number of occurrences were "quality-of-life", "skin", "therapy", "expression", "dendritic cells", "double-blind", "efficacy", "necrosis-factor-alpha", "rheumatoid arthritis", and "safety". Figure 7 illustrates the main areas of interest based on the density of keywords in the analyzed articles, where we can observe that the research focuses on quality of life, immune pathways, and treatment safety. Reference co-citation analysis can reflect a domain's knowledge structure and indicate research hotspots. The analysis showed that the most co-cited references were the papers of Rapp, SR "Psoriasis causes as much disability as other major medical diseases" [89] and Fredrickson, T "Severe psoriasis-oral therapy with a new retinoid" [106], both co-cited 19 times and serving as an additional indicator that treatment and life-quality are main topics in the research field. The works of Rapp, SR, Fredriksson, T, Gelfand, JM, Reich, K, Schon, MP, and Lee, E which bring attention to topics such as pathogenesis and novel treatment options, have the highest link strengths and are located among different clusters, indicating that they may serve as landmarks in the field.

The limitation of this bibliometric analysis resides mainly in the fact that only fulllength English articles indexed in the WoS database have been taken into account. This has been partially addressed by not limiting article access type in any kind. Moreover, no time limit has been set when researching articles to be included in the analysis, thus a larger and more accurate overview of the research field has been obtained. To diminish the effect of time on accumulated citations, a citation rate was also calculated in order to identify articles that received a large number of citations over a short period of time. Due to the fact that bibliometric analysis covers a broad area of research, it should be taken into account that papers with the highest citations might address general topics. In order to limit this and to provide an overview of the past, present, and future of psoriasis research, we used a combination of techniques: co-citation analysis to uncover knowledge foundations, bibliographic coupling to understand the present development of themes, and co-word analysis to assess existing or future relationships among topics in psoriasis research.

5. Conclusions

Research interest in the scientific community regarding psoriasis is high, leading to the rapid and constant development of this field. This is the first bibliometric study focusing on psoriasis, providing an overview of the intellectual structure and scientific directions in the field, taking into account algorithms based on the top 100 most cited articles on the subject. The research focus is shifting from disease presentation. Treatment modalities, especially novel-targeted therapies, immune pathways, and an integrative, complex approach to such cases are receiving great interest and represent research hotspots in the future.

Author Contributions: Conceptualization: O.M.T., S.H.M. and O.S.C.; formal analysis: O.M.T.; methodology: O.M.T., C.R.M. and A.C.N.; resources: C.R.M. and R.A.T.; validation: O.S.C. and S.H.M.; visualization: O.M.T. and R.A.T.; writing—original draft: O.M.T., C.R.M. and R.A.T.; writing—revision and editing: O.M.T. and C.R.M.; supervision: O.S.C. and S.H.M. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: All data presented can be made available upon request.

Acknowledgments: This article is part of a Ph.D. thesis from the Doctoral School of Medicine and Pharmacy of the University of Medicine, Pharmacy, Science, and Technology George Emil Palade of Targu Mures, titled "The impact of systemic inflammation in modulating disease presentation in psoriasis", which will be presented by Oana Mirela Tiucă by the fall of 2024.

Conflicts of Interest: The authors declare no conflict of interest.

References

- 1. WHO Organization. Global Report on Psoriasis; World Health Organization: Geneva, Switzerland, 2016.
- Damiani, G.; Bragazzi, N.L.; Karimkhani Aksut, C.; Wu, D.; Alicandro, G.; McGonagle, D.; Guo, C.; Dellavalle, R.; Grada, A.; Wong, P.; et al. The global, regional, and national burden of psoriasis: Results and insights from the global burden of disease 2019 Study. *Front. Med.* 2021, *8*, 743180. [CrossRef]
- 3. Mahil, S.K.; Capon, F.; Barker, J.N. Genetics of psoriasis. Dermatol. Clin. 2015, 33, 1–11. [CrossRef]
- 4. Zeng, J.; Luo, S.; Huang, Y.; Lu, Q. Critical role of environmental factors in the pathogenesis of psoriasis. *J. Dermatol.* 2017, 44, 863–872. [CrossRef]
- Gisondi, P.; Fostini, A.C.; Fossà, I.; Girolomoni, G.; Targher, G. Psoriasis and the metabolic syndrome. *Clin. Dermatol.* 2018, 36, 21–28. [CrossRef]
- 6. Xiaohong, L.; Zhenting, Z.; Yunjie, Y.; Wei, C.; Xiangjin, X.; Kun, X.; Xin, L.; Lu, L.; Jun, L.; Pin, C. Activation of the sting-irf3 pathway involved in psoriasis with diabetes mellitus. *J. Cell. Mol. Med.* **2022**, *26*, 2139–2151. [CrossRef]
- 7. Pritchard, A. Statistical Bibliography or Bibliometrics? J. Doc. 1969, 25, 348–349.
- 8. Stremersch, S.; Verniers, I.; Verhoef, P.C. The quest for citations: Drivers of article impact. J. Mark. 2007, 71, 171–193. [CrossRef]
- 9. Fan, K.S.; Fan, K.H.; Tse, P.L.; Ding, H.; Su, R.; Kwok, H.T. Analysis of top-cited articles on melanoma. *Acta Dermatovenerol. Alp. Pannonica Adriat.* 2022, *31*, 127–133. [CrossRef]
- Kwok, H.T.; Van, M.; Fan, K.S.; Chan, J. Top 100 cited articles in male breast cancer: A bibliometric analysis. *Breast Dis.* 2022, 41, 15–20. [CrossRef]
- 11. Zhang, Y.; Yu, C. Bibliometric evaluation of publications (2000–2020) on the prognosis of gastric cancer. *Inquiry* **2021**, *58*, 469580211056015. [CrossRef] [PubMed]
- Joyce, C.W.; Sugrue, C.M.; Joyce, K.M.; Kelly, J.L.; Regan, P.J. 100 citation classics in the melanoma literature: A bibliometric analysis. *Dermatol. Surg.* 2014, 40, 1284–1298. [CrossRef] [PubMed]

- Cocuz, I.G.; Cocuz, M.E.; Repanovici, A.; Sabău, A.-H.; Niculescu, R.; Tinca, A.-C.; Vunvulea, V.; Budin, C.E.; Szoke, A.R.; Popelea, M.C.; et al. Scientific research directions on the histopathology and immunohistochemistry of the cutaneous squamous cell carcinoma: A scientometric study. *Medicina* 2022, *58*, 1449. [CrossRef]
- 14. Paladugu, R.; Schein, M.; Gardezi, S.; Wise, L. One hundred citation classics in general surgical journals. *World J. Surg.* 2002, *26*, 1099–1105. [CrossRef]
- Strange, A.; Capon, F.; Spencer, C.C.A.; Knight, J.; Weale, M.E.; Allen, M.H.; Barton, A.; Band, G.; Bellenguez, C.; Bergboer, J.G.M.; et al. A genome-wide association study identifies new psoriasis susceptibility loci and an interaction between HLA-C and ERAP1. *Nat. Genet* 2010, 42, 985–990.
- Papp, K.; Tyring, S.; Lahfa, M.; Prinz, J.; Griffiths, C.; Nakanishi, A.; Zitnik, R.; van de Kerkhof, P.; Grp, E.P.S. A global phase iii randomized controlled trial of etanercept in psoriasis: Safety, efficacy, and effect of dose reduction. *Br. J. Dermatol.* 2005, 152, 1304–1312. [CrossRef]
- Cargill, M.; Schrodi, S.J.; Chang, M.; Garcia, V.E.; Brandon, R.; Callis, K.P.; Matsunami, N.; Ardlie, K.G.; Civello, D.; Catanese, J.J.; et al. A large-scale genetic association study confirms il12b and leads to the identification of il23r as psoriasis-risk genes. *Am. J. Hum. Genet* 2007, *80*, 273–290. [CrossRef]
- 18. Krueger, G.G.; Langley, R.G.; Leonardi, C.; Yeilding, N.; Guzzo, C.; Wang, Y.; Dooley, L.T.; Lebwohl, M.; Grp, C. 1275 P. S. A human interleukin-12/23 monoclonal antibody for the treatment of psoriasis. *N. Engl. J. Med.* **2007**, *356*, 580–592. [CrossRef]
- 19. Lebwohl, M.; Tyring, S.; Hamilton, T.; Toth, D.; Glazer, S.; Tawfik, N.; Walicke, P.; Dummer, W.; Wang, X.; Garovoy, M.; et al. A novel targeted T-cell modulator, efalizumab, for plaque psoriasis. *N. Engl. J. Med.* **2003**, *349*, 2004–2013. [CrossRef]
- 20. Michalek, I.M.; Loring, B.; John, S.M. A systematic review of worldwide epidemiology of psoriasis. *J. Eur. Acad. Dermatol. Venereol.* 2017, *31*, 205–212. [CrossRef]
- Menter, A.; Tyring, S.K.; Gordon, K.; Kimball, A.B.; Leonardi, C.L.; Langley, R.G.; Strober, B.E.; Kaul, M.; Gu, Y.; Okun, M.; et al. Adalimumab therapy for moderate to severe psoriasis: A randomized, controlled phase iii trial. *J. Am. Acad. Dermatol.* 2008, 58, 106–115. [CrossRef]
- 22. Leonardi, C.; Matheson, R.; Zachariae, C.; Cameron, G.; Li, L.; Edson-Heredia, E.; Braun, D.; Banerjee, S. Anti-interleukin-17 monoclonal antibody ixekizumab in chronic plaque psoriasis. *N. Engl. J. Med.* **2012**, *366*, 1190–1199. [CrossRef]
- 23. Nickoloff, B.; Karabin, G.; Barker, J.; Griffiths, C.; Sarma, V.; Mitra, R.; Elder, J.; Kunkel, S.; Dixit, V. Cellular-localization of interleukin-8 and its inducer, tumor-necrosis-factor-alpha in psoriasis. *Am. J. Pathol.* **1991**, *138*, 129–140.
- Wada, Y.; Cardinale, I.; Khatcherian, A.; Chu, J.; Kantor, A.B.; Gottlieb, A.B.; Tatsuta, N.; Jacobson, E.; Barsoum, J.; Krueger, J.G. Apilimod inhibits the production of il-12 and il-23 and reduces dendritic cell infiltration in psoriasis. *PLoS ONE* 2012, 7, e35069. [CrossRef]
- 25. Papp, K.A.; Leonardi, C.; Menter, A.; Ortonne, J.-P.; Krueger, J.G.; Kricorian, G.; Aras, G.; Li, J.; Russell, C.B.; Thompson, E.H.Z.; et al. Brodalumab, an anti-interleukin-17-receptor antibody for psoriasis. *N. Engl. J. Med.* **2012**, *366*, 1181–1189. [CrossRef]
- Naldi, L.; Chatenoud, L.; Linder, D.; Fortina, A.; Peserico, A.; Virgili, A.; Bruni, P.; Ingordo, V.; Lo Scocco, G.; Solaroli, C.; et al. Cigarette smoking, body mass index, and stressful life events as risk factors for psoriasis: Results from an italian case-control study. J. Investig. Dermatol. 2005, 125, 61–67. [CrossRef]
- 27. Kagami, S.; Rizzo, H.L.; Lee, J.J.; Koguchi, Y.; Blauvelt, A. Circulating Th17, Th22, and Th1 cells are increased in psoriasis. *J. Investig. Dermatol.* **2010**, 130, 1373–1383. [CrossRef]
- Gordon, K.B.; Langley, R.G.; Leonardi, C.; Toth, D.; Menter, M.A.; Kang, S.; Heffernan, M.; Miller, B.; Hamlin, R.; Lim, L.; et al. Clinical response to adalimumab treatment in patients with moderate to severe psoriasis: Double-blind, randomized controlled trial and open-label extension study. J. Am. Acad. Dermatol. 2006, 55, 598–606. [CrossRef]
- Griffiths, C.E.M.; Reich, K.; Lebwohl, M.; van de Kerkhof, P.; Paul, C.; Menter, A.; Cameron, G.S.; Erickson, J.; Zhang, L.; Secrest, R.J.; et al. Comparison of ixekizumab with etanercept or placebo in moderate-to-severe psoriasis (UNCOVER-2 and UNCOVER-3): Results from two phase 3 randomised trials. *Lancet* 2015, *386*, 541–551. [CrossRef]
- 30. Griffiths, C.E.M.; Strober, B.E.; van de Kerkhof, P.; Ho, V.; Fidelus-Gort, R.; Yeilding, N.; Guzzo, C.; Xia, Y.; Zhou, B.; Li, S.; et al. Comparison of ustekinumab and etanercept for moderate-to-severe psoriasis. *N. Engl. J. Med.* **2010**, *362*, 118–128. [CrossRef]
- Abrams, J.; Lebwohl, M.; Guzzo, C.; Jegasothy, B.; Goldfarb, M.; Goffe, B.; Menter, A.; Lowe, N.; Krueger, G.; Brown, M.; et al. CTLA4ig-mediated blockade of T-cell costimulation in patients with psoriasis vulgaris. *J. Clin. Investig.* 1999, 103, 1243–1252. [CrossRef]
- 32. Ellis, C.; Gorsulowsky, D.; Hamilton, T.; Billings, J.; Brown, M.; Headington, J.; Cooper, K.; Baadsgaard, O.; Duell, E.; Annesley, T.; et al. Cyclosporine improves psoriasis in a double-blind-study. *JAMA-J. Am. Med. Assoc.* **1986**, 256, 3110–3116. [CrossRef]
- Mrowietz, U.; Kragballe, K.; Reich, K.; Spuls, P.; Griffiths, C.E.M.; Nast, A.; Franke, J.; Antoniou, C.; Arenberger, P.; Balieva, F.; et al. Definition of treatment goals for moderate to severe psoriasis: A european consensus. *Arch. Dermatol. Res.* 2011, 303, 1–10. [CrossRef]
- 34. Henseler, T.; Christophers, E. Disease concomitance in psoriasis. J. Am. Acad. Dermatol. 1995, 32, 982–986. [CrossRef]
- Sugiyama, H.; Gyulai, R.; Toichi, E.; Garaczi, E.; Shimada, S.; Stevens, S.; McCormick, T.; Cooper, K. Dysfunctional blood and target tissue CD4(+)CD25(high) regulatory T cells in psoriasis: Mechanism underlying unrestrained pathogenic effector t cell proliferation. *J. Immunol.* 2005, 174, 164–173. [CrossRef]

- 36. Blauvelt, A.; Papp, K.A.; Griffiths, C.E.M.; Randazzo, B.; Wasfi, Y.; Shen, Y.-K.; Li, S.; Kimball, A.B. Efficacy and safety of guselkumab, an anti-interleukin-23 monoclonal antibody, compared with adalimumab for the continuous treatment of patients with moderate to severe psoriasis: Results from the phase iii, double-blinded, placebo- and active comparatore-controlled voyage 1 trial. *J. Am. Acad. Dermatol.* 2017, *76*, 405–417.
- 37. Reich, K.; Armstrong, A.W.; Foley, P.; Song, M.; Wasfi, Y.; Randazzo, B.; Li, S.; Shen, Y.K.; Gordon, K.B. Efficacy and safety of guselkumab, an anti-interleukin-23 monoclonal antibody, compared with adalimumab for the treatment of patients with moderate to severe psoriasis with randomized withdrawal and retreatment: Results from the phase iii, double-blind, placeboand active comparatore-controlled voyage 2 trial. J. Am. Acad. Dermatol. 2017, 76, 418–431.
- McInnes, I.B.; Kavanaugh, A.; Gottlieb, A.B.; Puig, L.; Rahman, P.; Ritchlin, C.; Brodmerkel, C.; Li, S.; Wang, Y.; Mendelsohn, A.M.; et al. Efficacy and safety of ustekinumab in patients with active psoriatic arthritis: 1 year results of the phase 3, multicentre, double-blind, placebo-controlled psummit 1 trial. *Lancet* 2013, 382, 780–789. [CrossRef]
- 39. Chaudhari, U.; Romano, P.; Mulcahy, L.; Dooley, L.; Baker, D.; Gottlieb, A. Efficacy and safety of infliximab monotherapy for plaque-type psoriasis: A randomised trial. *Lancet* 2001, 357, 1842–1847. [CrossRef]
- 40. Papp, K.A.; Langley, R.G.; Lebwohl, M.; Krueger, G.G.; Szapary, P.; Yeilding, N.; Guzzo, C.; Hsu, M.-C.; Wang, Y.; Li, S.; et al. Efficacy and safety of ustekinumab, a human interleukin-12/23 monoclonal antibody, in patients with psoriasis: 52-week results from a randomised, double-blind, placebo-controlled trial (Phoenix 2). *Lancet* **2008**, *371*, 1675–1684. [CrossRef]
- Leonardi, C.L.; Kimball, A.B.; Papp, K.A.; Yeilding, N.; Guzzo, C.; Wang, Y.; Li, S.; Dooley, L.T.; Gordon, K.B.; PHOENIX 1 study investigators. Efficacy and safety of ustekinumab, a human interleukin-12/23 monoclonal antibody, in patients with psoriasis: 76-week results from a randomised, double-blind, placebo-controlled trial (phoenix 1). *Lancet* 2008, 371, 1665–1674. [CrossRef]
- Tyring, S.; Gottlieb, A.; Papp, K.; Gordon, K.; Leonardi, C.; Wang, A.; Lalla, D.; Woolley, M.; Jahreis, A.; Zitnik, R.; et al. Etanercept and clinical outcomes, fatigue, and depression in psoriasis: Double-blind placebo-controlled randomised phase III trial. *Lancet* 2006, 367, 29–35. [CrossRef]
- 43. Saurat, J.-H.; Stingl, G.; Dubertret, L.; Papp, K.; Langley, R.G.; Ortonne, J.-P.; Unnebrink, K.; Kaul, M.; Camez, A.; Invest, C.S. Efficacy and safety results from the randomized controlled comparative study of adalimumab vs. Methotrexate vs. Placebo in patients with psoriasis (champion). *Br. J. Dermatol.* **2008**, *158*, 558–566. [CrossRef]
- 44. Leonardi, C.; Powers, J.; Matheson, R.; Goffe, B.; Zitnik, R.; Wang, A.; Gottlieb, A.; Bagel, J.; Camisa, C.; Caro, I.; et al. Etanercept as monotherapy in patients with psoriasis. *N. Engl. J. Med.* **2003**, *349*, 2014–2022. [CrossRef]
- 45. Baker, H.; Ryan, T. Generalized pustular psoriasis—A clinical and epidemiological study of 104 cases. *Br. J. Dermatol.* **1968**, *80*, 771–793. [CrossRef]
- 46. Pathirana, D.; Ormerod, A.D.; Saiag, P.; Smith, C.; Spuls, P.I.; Nast, A.; Barker, J.; Bos, J.D.; Burmester, G.-R.; Chimenti, S.; et al. European S3-guidelines on the systemic treatment of psoriasis vulgaris. *J. Eur. Acad. Dermatol. Venereol.* 2009, 23, 5–70. [CrossRef]
- 47. Nair, R.P.; Duffin, K.C.; Helms, C.; Ding, J.; Stuart, P.E.; Goldgar, D.; Gudjonsson, J.E.; Li, Y.; Tejasvi, T.; Feng, B.-J.; et al. Genome-wide scan reveals association of psoriasis with IL-23 and NF-kappa *B* pathways. *Nat. Genet* **2009**, *41*, 199–204. [CrossRef]
- 48. Parisi, R.; Symmons, D.P.M.; Griffiths, C.E.M.; Ashcroft, D.M.; Management, I. Global epidemiology of psoriasis: A systematic review of incidence and prevalence. *J. Investig. Dermatol.* **2013**, *133*, 377–385. [CrossRef]
- Tsoi, L.C.; Spain, S.L.; Knight, J.; Ellinghaus, E.; Stuart, P.E.; Capon, F.; Ding, J.; Li, Y.; Tejasvi, T.; Gudjonsson, J.E.; et al. Identification of 15 new psoriasis susceptibility loci highlights the role of innate immunity. *Nat. Genet* 2012, 44, 1341–1348. [CrossRef]
- 50. Trembath, R.; Clough, R.; Rosbotham, J.; Jones, A.; Camp, R.; Frodsham, A.; Browne, J.; Barber, R.; Terwilliger, J.; Lathrop, G.; et al. Identification of a major susceptibility locus on chromosome 6p and evidence for further disease loci revealed by a two stage genome-wide search in psoriasis. *Hum. Mol. Genet* **1997**, *6*, 813–820. [CrossRef]
- Ma, H.-L.; Liang, S.; Li, J.; Napierata, L.; Brown, T.; Benoit, S.; Senices, M.; Gill, D.; Dunussi-Joannopoulos, K.; Collins, M.; et al. Il-22 is required for th17 cell-mediated pathology in a mouse model of psoriasis-like skin inflammation. *J. Clin. Investig.* 2008, 118, 597–607. [CrossRef]
- 52. Wolk, K.; Witte, E.; Wallace, E.; Doecke, W.-D.; Kunz, S.; Asadullah, K.; Volk, H.-D.; Sterry, W.; Sabat, R. Il-22 regulates the expression of genes responsible for antimicrobial defense, cellular differentiation, and mobility in keratinocytes: A potential role in psoriasis. *Eur. J. Immunol.* **2006**, *36*, 1309–1323. [CrossRef]
- Chan, J.R.; Blumenschein, W.; Murphy, E.; Diveu, C.; Wiekowski, M.; Abbondanzo, S.; Lucian, L.; Geissler, R.; Brodie, S.; Kimball, A.B.; et al. IL-23 stimulates epidermal hyperplasia via TNF and IL-20R2-dependent mechanisms with implications for psoriasis pathogenesis. J. Exp. Med. 2006, 203, 2577–2587. [CrossRef]
- 54. van der Fits, L.; Mourits, S.; Voerman, J.S.A.; Kant, M.; Boon, L.; Laman, J.D.; Cornelissen, F.; Mus, A.-M.; Florencia, E.; Prens, E.P.; et al. Imiquimod-induced psoriasis-like skin inflammation in mice is mediated via the II-23/II-17 Axis. *J. Immunol.* **2009**, *182*, 5836–5845. [CrossRef]
- 55. Hammarstrom, S.; Hamberg, M.; Samuelsson, B.; Duell, E.; Stawiski, M.; Voorhees, J. Increased concentrations of nonesterified arachidonic-acid, 12l-Hydroxy-5,8,10,14-eicosatetraenoic acid, prostaglandin-E2, and prostaglandin-F2alpha in epidermis of psoriasis. *Proc. Natl. Acad. Sci. USA* **1975**, *72*, 5130–5134. [CrossRef]
- 56. Lowes, M.A.; Suarez-Farinas, M.; Krueger, J.G. Immunology of Psoriasis. Annu. Rev. Immunol. 2014, 32, 227–255. [CrossRef]
- 57. Lee, E.; Trepicchio, W.; Oestreicher, J.; Pittman, D.; Wang, F.; Chamian, F.; Dhodapkar, M.; Krueger, J. Increased Expression of Interleukin 23 P19 and P40 in Lesional Skin of Patients with Psoriasis Vulgaris. *J. Exp. Med.* **2004**, *199*, 125–130. [CrossRef]

- 58. Sommer, D.M.; Jenisch, S.; Suchan, M.; Christophers, E.; Weichenthal, M. Increased prevalence of the metabolic syndrome in patients with moderate to severe psoriasis. *Arch. Dermatol. Res.* **2006**, *298*, 321–328. [CrossRef]
- Gottlieb, A.; Evans, R.; Li, S.; Dooley, L.; Guzzo, C.; Baker, D.; Bala, M.; Marano, C.; Menter, A. Infliximab induction therapy for patients with severe plaque-type psoriasis: A randomized, double-blind, placebo-controlled trial. *J. Am. Acad. Dermatol.* 2004, *51*, 534–542. [CrossRef]
- Reich, K.; Nestle, F.; Papp, K.; Ortonne, J.; Evans, R.; Guzzo, C.; Li, S.; Dooley, L.; Griffiths, C.; Investigators, E.S. Infliximab induction and maintenance therapy for moderate-to-severe psoriasis: A phase iii, multicentre, double-blind trial. *Lancet* 2005, 366, 1367–1374. [CrossRef]
- Chiricozzi, A.; Guttman-Yassky, E.; Suarez-Farinas, M.; Nograles, K.E.; Tian, S.; Cardinale, I.; Chimenti, S.; Krueger, J.G. Integrative responses to il-17 and tnf-alpha in human keratinocytes account for key inflammatory pathogenic circuits in psoriasis. *J. Investig. Dermatol.* 2011, 131, 677–687. [CrossRef]
- Zheng, Y.; Danilenko, D.M.; Valdez, P.; Kasman, I.; Eastham-Anderson, J.; Wu, J.; Ouyang, W. Interleukin-22, a t(h)17 cytokine, mediates il-23-induced dermal inflammation and acanthosis. *Nature* 2007, 445, 648–651. [CrossRef]
- 63. Marrakchi, S.; Guigue, P.; Renshaw, B.R.; Puel, A.; Pei, X.-Y.; Fraitag, S.; Zribi, J.; Bal, E.; Cluzeau, C.; Chrabieh, M.; et al. Interleukin-36-receptor antagonist deficiency and generalized pustular psoriasis. *N. Engl. J. Med.* **2011**, *365*, 620–628. [CrossRef]
- 64. Stern, R.; Nichols, K.; Vakeva, L. Malignant melanoma in patients treated for psoriasis with methoxsalen (psoralen) and ultraviolet a radiation (puva). *N. Engl. J. Med.* **1997**, *336*, 1041–1045. [CrossRef]
- 65. Lin, A.M.; Rubin, C.J.; Khandpur, R.; Wang, J.Y.; Riblett, M.; Yalavarthi, S.; Villanueva, E.C.; Shah, P.; Kaplan, M.J.; Bruce, A.T. Mast cells and neutrophils release il-17 through extracellular trap formation in psoriasis. *J. Immunol.* **2011**, *187*, 490–500. [CrossRef]
- 66. Nestle, F.O.; Kaplan, D.H.; Barker, J. Mechanisms of disease: Psoriasis. N. Engl. J. Med. 2009, 361, 496–509. [CrossRef]
- 67. Schon, M.; Boehncke, W. Medical Progress—Psoriasis. N. Engl. J. Med. 2005, 352, 1899–1912. [CrossRef]
- 68. Sonkoly, E.; Wei, T.; Janson, P.C.J.; Saaf, A.; Lundeberg, L.; Tengvall-Linder, M.; Norstedt, G.; Alenius, H.; Homey, B.; Scheynius, A.; et al. Micrornas: Novel regulators involved in the pathogenesis of psoriasis? *PLoS ONE* **2007**, *2*, e610. [CrossRef]
- 69. Farber, E.; Nall, M. Natural-History of Psoriasis in 5600 Patients. *Dermatologica* **1974**, *148*, 1–18. [CrossRef]
- Melski, J.; Tanenbaum, L.; Parrish, J.; Fitzpatrick, T.; Bleich, H. Oral methoxsalen photochemotherapy for treatment of psoriasis—Cooperative clinical-trial. J. Investig. Dermatol. 1977, 68, 328–335.
- Detmar, M.; Brown, L.; Claffey, K.; Yee, K.; Kocher, O.; Jackman, R.; Berse, B.; Dvorak, H. Overexpression of vascular-permeability factor vascular endothelial growth-factor and its receptors in psoriasis. *J. Exp. Med.* **1994**, *180*, 1141–1146. [CrossRef]
- 72. Lowes, M.A.; Bowcock, A.M.; Krueger, J.G. Pathogenesis and therapy of psoriasis. Nature 2007, 445, 866–873. [CrossRef]
- 73. Armstrong, A.W.; Read, C. Pathophysiology, clinical presentation, and treatment of psoriasis a review. *JAMA-J. Am. Med. Assoc.* **2020**, *323*, 1945–1960. [CrossRef]
- 74. Mehta, N.N.; Azfar, R.S.; Shin, D.B.; Neimanns, A.L.; Troxel, A.B.; Gelfand, J.M. Patients with severe psoriasis are at increased risk of cardiovascular mortality: Cohort study using the general practice research database. *Eur. Heart J.* **2010**, *31*, 1000–1006. [CrossRef]
- 75. Lebwohl, M.; Strober, B.; Menter, A.; Gordon, K.; Weglowska, J.; Puig, L.; Papp, K.; Spelman, L.; Toth, D.; Kerdel, F.; et al. Phase 3 studies comparing brodalumab with ustekinumab in psoriasis. *N. Engl. J. Med.* **2015**, *373*, 1318–1328. [CrossRef]
- 76. Gordon, K.B.; Blauvelt, A.; Papp, K.A.; Langley, R.G.; Luger, T.; Ohtsuki, M.; Reich, K.; Amato, D.; Ball, S.G.; Braun, D.K.; et al. Phase 3 trials of Ixekizumab in moderate-to-severe plaque psoriasis. *N. Engl. J. Med.* **2016**, 375, 345–356. [CrossRef]
- 77. Parrish, J.; Fitzpatrick, T.; Tanenbaum, L.; Pathak, M. Photochemotherapy of psoriasis with oral methoxsalen and longwave ultraviolet-light. *N. Engl. J. Med.* **1974**, *291*, 1207–1211. [CrossRef]
- 78. Nestle, F.; Conrad, C.; Tun-Kyi, A.; Homey, B.; Gombert, M.; Boyman, O.; Burg, G.; Liu, Y.; Gilliet, M. Plasmacytoid predendritic cells initiate psoriasis through interferon-alpha production. *J. Exp. Med.* **2005**, *202*, 135–143. [CrossRef]
- 79. Gelfand, J.; Weinstein, R.; Porter, S.; Neimann, A.; Berlin, J.; Margolis, D. Prevalence and treatment of psoriasis in the united kingdom—A population-based study. *Arch. Dermatol.* **2005**, *141*, 1537–1541. [CrossRef]
- Neimann, A.L.; Shin, D.B.; Wang, X.; Margolis, D.J.; Troxel, A.B.; Gelfand, J.M. Prevalence of cardiovascular risk factors in patients with psoriasis. J. Am. Acad. Dermatol. 2006, 55, 829–835. [CrossRef]
- 81. Boehncke, W.H.; Schoen, M.P. Psoriasis. Lancet 2015, 386, 983–994. [CrossRef]
- 82. Gisondi, P.; Tessari, G.; Conti, A.; Piaserico, S.; Schianchi, S.; Peserico, A.; Giannetti, A.; Girolomoni, G. Prevalence of metabolic syndrome in patients with psoriasis: A hospital-based case-control study. *Br. J. Dermatol.* **2007**, *157*, 68–73. [CrossRef]
- 83. Greb, J.E.; Goldminz, A.M.; Elder, J.T.; Lebwohl, M.G.; Gladman, D.D.; Wu, J.J.; Mehta, N.N.; Finlay, A.Y.; Gottlieb, A.B. Psoriasis. *Nat. Rev. Dis. Prim.* **2016**, 2, 16082. [CrossRef]
- 84. Lebwohl, M. Psoriasis. Lancet 2003, 361, 1197–1204. [CrossRef]
- 85. Griffiths, C.; Barker, J. Psoriasis 1—Pathogenesis and clinical features of Psoriasis. Lancet 2007, 370, 263–271. [CrossRef]
- 86. Christophers, E. Psoriasis—Epidemiology and clinical spectrum. Clin. Exp. Dermatol. 2001, 26, 314–320. [CrossRef]
- Takeshita, J.; Grewal, S.; Langan, S.M.; Mehta, N.N.; Ogdie, A.; Van Voorhees, A.S.; Gelfand, J.M. Psoriasis and comorbid diseases epidemiology. J. Am. Acad. Dermatol. 2017, 76, 377–390. [CrossRef]
- Davidovici, B.B.; Sattar, N.; Joerg, P.C.; Puig, L.; Emery, P.; Barker, J.N.; van de Kerkhof, P.; Stahle, M.; Nestle, F.O.; Girolomoni, G.; et al. Psoriasis and systemic inflammatory diseases: Potential mechanistic links between skin disease and co-morbid conditions. J. Investig. Dermatol. 2010, 130, 1785–1796. [CrossRef]

- Rapp, S.; Feldman, S.; Exum, M.; Fleischer, A.; Reboussin, D. Psoriasis causes as much disability as other major medical diseases. J. Am. Acad. Dermatol. 1999, 41, 401–407. [CrossRef]
- Hollox, E.J.; Huffmeier, U.; Zeeuwen, P.L.J.M.; Palla, R.; Lascorz, J.; Rodijk-Olthuis, D.; van de Kerkhof, P.C.M.; Traupe, H.; de Jongh, G.; den Heijer, M.; et al. Psoriasis is associated with increased beta-defensin genomic copy number. *Nat. Genet* 2008, 40, 23–25. [CrossRef]
- Stern, R.; Nijsten, T.; Feldman, S.; Margolis, D.; Rolstad, T. Psoriasis is common, carries a substantial burden even when not extensive, and is associated with widespread treatment dissatisfaction. *J. Investig. Dermatol. Symp. Proc.* 2004, *9*, 136–139. [CrossRef]
- 92. Henseler, T.; Christophers, E. Psoriasis of early and late onset—Characterization of 2 types of psoriasis-vulgaris. *J. Am. Acad. Dermatol.* **1985**, *13*, 450–456. [CrossRef]
- 93. Hawkes, J.E.; Chan, T.C.; Krueger, J.G. Psoriasis pathogenesis and the development of novel targeted immune therapies. *J. Allergy Clin. Immunol.* 2017, 140, 645–653. [CrossRef]
- 94. Rendon, A.; Schaekel, K. Psoriasis pathogenesis and treatment. Int. J. Mol. Sci. 2019, 20, 1475. [CrossRef]
- Rachakonda, T.D.; Schupp, C.W.; Armstrong, A.W. Psoriasis prevalence among adults in The United States. J. Am. Acad. Dermatol. 2014, 70, 512–516. [CrossRef]
- 96. Lowes, M.A.; Kikuchi, T.; Fuentes-Duculan, J.; Cardinale, I.; Zaba, L.C.; Haider, A.S.; Bowman, E.P.; Krueger, J.G. Psoriasis vulgaris lesions contain discrete populations of Th1 and Th17 T cells. *J. Investig. Dermatol.* **2008**, *128*, 1207–1211. [CrossRef]
- Zenz, R.; Eferl, R.; Kenner, L.; Florin, L.; Hummerich, L.; Mehic, D.; Scheuch, H.; Angel, P.; Tschachler, E.; Wagner, E. Psoriasis-like skin disease and arthritis caused by inducible epidermal deletion of jun proteins. *Nature* 2005, 437, 369–375. [CrossRef]
- 98. Langley, R.; Krueger, G.; Griffiths, C. Psoriasis: Epidemiology, clinical features, and quality of life. *Ann. Rheum. Dis.* 2005, 64, 18–23. [CrossRef]
- 99. Nickoloff, B.; Nestle, F. Recent insights into the immunopathogenesis of psoriasis provide new therapeutic opportunities. J. Clin. Investig. 2004, 113, 1664–1675. [CrossRef]
- Gottlieb, S.; Gilleaudeau, P.; Johnson, R.; Estes, L.; Woodworth, T.; Gottlieb, A.; Krueger, J. Response of psoriasis to a lymphocyteselective toxin (dab(389)il-2) suggests a primary immune, but not keratinocyte, pathogenic basis. *Nat. Med.* 1995, 1, 442–447. [CrossRef]
- 101. Stern, R.; Thibodeau, L.; Kleinerman, R.; Parrish, J.; Fitzpatrick, T. Risk of cutaneous carcinoma in patients treated with oral methoxsalen photochemotherapy for Psoriasis. *N. Engl. J. Med.* **1979**, *300*, 809–813. [CrossRef]
- Gelfand, J.M.; Neimann, A.L.; Shin, D.B.; Wang, X.; Margolis, D.J.; Troxel, A.B. Risk of myocardial infarction in patients with psoriasis. JAMA-J. Am. Med. Assoc. 2006, 296, 1735–1741. [CrossRef]
- 103. Langley, R.G.; Elewski, B.E.; Lebwohl, M.; Reich, K.; Griffiths, C.E.M.; Papp, K.; Puig, L.; Nakagawa, H.; Spelman, L.; Sigurgeirsson, B.; et al. Secukinumab in plaque psoriasis—Results of two phase 3 trials. *N. Engl. J. Med.* **2014**, *371*, 326–338. [CrossRef]
- 104. Nair, R.; Stuart, P.; Nistor, I.; Hiremagalore, R.; Chia, N.; Jenisch, S.; Weichenthal, M.; Abecasis, G.; Lim, H.; Christophers, E.; et al. Sequence and haplotype analysis supports hla-c as the psoriasis susceptibility 1 gene. Am. J. Hum. Genet 2006, 78, 827–851. [CrossRef]
- 105. Arican, O.; Aral, M.; Sasmaz, S.; Ciragil, P. Serum Levels of TNF-Alpha, IFN-gamma, IL-6, IL-8, IL-12, IL-17 and IL-18 in patients with active psoriasis and correlation with disease severity. *Mediat. Inflamm.* 2005, 2005, 273–279. [CrossRef]
- 106. Fredriksson, T.; Pettersson, U. Severe psoriasis—Oral therapy with a new retinoid. *Dermatologica* 1978, 157, 238–244. [CrossRef]
- 107. Sano, S.; Chan, K.; Carbajal, S.; Clifford, J.; Peavey, M.; Kiguchi, K.; Itami, S.; Nickoloff, B.; DiGiovanni, J. STATt3 links activated keratinocytes and immunocytes required for development of psoriasis in a novel transgenic mouse model. *Nat. Med.* 2005, 11, 43–49. [CrossRef]
- Nograles, K.E.; Zaba, L.C.; Guttman-Yassky, E.; Fuentes-Duculan, J.; Suarez-Farinas, M.; Cardinale, I.; Khatcherian, A.; Gonzalez, J.; Pierson, K.C.; White, T.R.; et al. Th17 cytokines interleukin (IL)-17 and IL-22 modulate distinct inflammatory and keratinocyte-response pathways. *Br. J. Dermatol.* 2008, *159*, 1092–1102. [CrossRef]
- 109. Di Cesare, A.; Di Meglio, P.; Nestle, F.O. The IL-23/Th17 axis in the immunopathogenesis of psoriasis. *J. Investig. Dermatol.* 2009, 129, 1339–1350. [CrossRef]
- 110. Krueger, G.; Koo, J.; Lebwohl, M.; Menter, A.; Stern, R.; Rolstad, T. The impact of psoriasis on quality of life—Results of a 1998 national psoriasis foundation patient-membership survey. *Arch. Dermatol.* **2001**, *137*, 280–284.
- 111. Krueger, J. The immunologic basis for the treatment of psoriasis with new biologic agents. J. Am. Acad. Dermatol. 2002, 46, 1–26. [CrossRef]
- 112. Kurd, S.K.; Troxel, A.B.; Crits-Christoph, P.; Gelfand, J.M. The risk of depression, anxiety, and suicidality in patients with psoriasis a population-based cohort study. *Arch. Dermatol.* **2010**, *146*, 891–895.
- Ellis, C.; Krueger, G.; Grp, A.C.S. Treatment of chronic plaque psoriasis by selective targeting of memory effector T lymphocytes. N. Engl. J. Med. 2001, 345, 248–255. [CrossRef]
- 114. Homey, B.; Dieu-Nosjean, M.; Wiesenborn, A.; Massacrier, C.; Pin, J.; Oldham, E.; Catron, D.; Buchanan, M.; Muller, A.; Malefyt, R.; et al. Up-regulation of macrophage inflammatory protein-3 alpha/ccl20 and cc chemokine receptor 6 in psoriasis. *J. Immunol.* 2000, 164, 6621–6632. [CrossRef]
- 115. Van Eck, N.J.; Waltman, L. Software Survey: Vosviewer, a computer program for bibliometric mapping. *Scientometrics* **2010**, *84*, 523–538. [CrossRef]

- 116. Donthu, N.; Kumar, S.; Mukherjee, D.; Pandey, N.; Lim, W.M. How to conduct a bibliometric analysis: An overview and guidelines. *J. Bus. Res.* 2021, 133, 285–296. [CrossRef]
- 117. Reid, C.; Griffiths, C.E.M. Psoriasis and treatment: Past, present and future aspects. *Acta Dermatol. Venereol.* **2020**, *100*, Adv00032. [CrossRef]

Disclaimer/Publisher's Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.