

Insights into vitamin D targeting gut microbiota to affect health: A bibliometric and visual analysis (1997–2024)

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ABSTRACT

This study employed bibliometric analysis to explore the relationship between vitamin D and gut microbiota, focusing on how vitamin D impacts health through gut microbiota modulation. Relevant articles from 1997 to 2024 retrieved from the Web of Science were analysed using CiteSpace and VOSviewer, with additional visualisations created in Microsoft Excel and Scimago Graphica. 710 papers were retrieved. The number of articles has steadily increased over the past 27 years. The journals *Nutrients* and *Poultry Science* published the most articles. Keyword co-occurrence analysis revealed four key areas: diseases (primarily inflammatory and metabolic), human gut microbiota, microbial components (notably short-chain fatty acids), and mechanisms (immunity, inflammation, etc.). This study highlights the rapid growth of research in this field since 1997, underscoring the potential of vitamin D/VDR signalling to target gut microbiota as a promising strategy for mitigating disease progression, warranting further investigation and attention.

KEYWORDS

bibliometric analysis, vitamin D, gut microbiota, intestinal inflammation

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1. INTRODUCTION

The human gut microbiome represents a complex ecosystem that provides essential functions to its host and can mediate physiologically important chemical transformations (Bäckhed et al., 2005). There are 10–100 trillion microorganisms in the human gut, including 50 bacterial phyla and about 100–1,000 bacterial species, collectively called the gut microbiota (Li et al., 2016; Adak and Khan, 2019). The gut microbiota is 10 times richer than somatic and germ cells (Bäckhed et al., 2005). In a human body, 150–170 bacterial species predominate, which benefit from the warm, nutrient-rich environment of the gut and perform protective, metabolic, and structural functions (Adak and Khan, 2019). The complex microbiota of the human gastrointestinal tract is becoming a key factor in human health and disease. Gut microbes produce metabolites and antioxidants, modulate the immune system, synthesise vitamins, strengthen the intestinal barrier, and regulate neurotransmitters and hormones (Hooper et al., 2012; Kamada et al., 2013; LeBlanc et al., 2013; Sommer and Bäckhed, 2013; Heintz-Buschart and Wilmes, 2018). However, homeostasis or population imbalance in the microbiota can lead to colonisation and infestation in the gut by opportunistic pathogens, which increases the risk of host immune response and negatively impacts human health (Abreu, 2010; Hou et al., 2022).

Vitamin D is a fat-soluble vitamin obtained from diet or skin exposure to ultraviolet light from sunlight (Dusso et al., 2005). The vitamin D endocrine system plays a crucial role in calcium homeostasis and bone metabolism (Dusso et al., 2005). Studies over the past 2 decades have revealed multiple biological roles of vitamin D, including induction of cell differentiation, inhibition of cell growth, immunomodulation, and control of other hormonal systems (Dusso et al., 2005). Several large epidemiological studies have demonstrated that vitamin D deficiency is associated with an increased risk of multiple adverse health outcomes, including osteoporosis, obesity, inflammatory bowel disease, type 2 diabetes (T2DM), cardiovascular disease, cancer, and autoimmune diseases (Holick, 2017). In recent years, there has been an increasing interest in the potential impact of vitamin D on the gut microbiological composition. Vitamin D is an immunomodulatory factor that influences intestinal homeostasis. Alterations in the gut microbiota have been shown to be one of the mechanisms linking vitamin D to some of its extra-skeletal clinical outcomes, e.g., vitamin D stabilises junctions between epithelial cells and promotes an appropriate balance between the gut microbiota and intestinal immunity (Fakhoury et al., 2020). Evidence on the effects of vitamin D supplementation on the human gut microbiome is relatively limited, with several RCTs reporting inconsistent results (Kanhare et al., 2018; Naderpoor et al., 2019; Singh et al., 2020; Shieh et al., 2021; Aparicio et al., 2023; Pham et al., 2023).

Bibliometric analyses are a series of mathematical and statistical analyses used to evaluate and quantify publication data to explore the dynamics of a particular specialty. Since the potential effects of vitamin D on gut microbiota were found, there has been a significant increase in the literature in this area in recent years. However, no attempt has been made to conduct an analytical bibliometric analysis. We retrieved relevant publications up to the end of this year. We analysed the journals, references and keywords of these publications to reveal global trends and research hotspots to help researchers identify current research concerns and better understand the dynamics of research in the field, which can then guide the direction of future research.

2. DATA AND METHODS

2.1. Data search strategy

Data for bibliometric analysis were downloaded from the Web of Science Core Collection (WoSCC) database (<https://www.webofknowledge.com>). Our search formula was as follows: #1 topic = vitamin D OR 25 Hydroxyvitamin D₂ OR 25 Hydroxycalciferol OR 25 Hydroxyergocalciferol OR Ercalcidiol OR 25 Hydroxycholecalciferol OR 25-Hydroxyvitamin D₃ OR Calcidiol, #2 Topic = Gastrointestinal Microbiome* OR Gut Microbiome* OR Gut Microflora OR Gut Microbiota* OR Gastrointestinal Flora OR Gut Flora OR Gastrointestinal Microbiota* OR Gastrointestinal Microbial Communit* OR Gastric Microbiome* OR Intestinal Microbiome* OR Intestinal Microbiota*, * as a wildcard character that can be substituted for zero or more characters. Final = #1 AND #2. We limited the literature type to ARTICLE and the language to ENGLISH. The publication years ranged from 1 January 1997 to 31 December 2024 (Fig. 1).

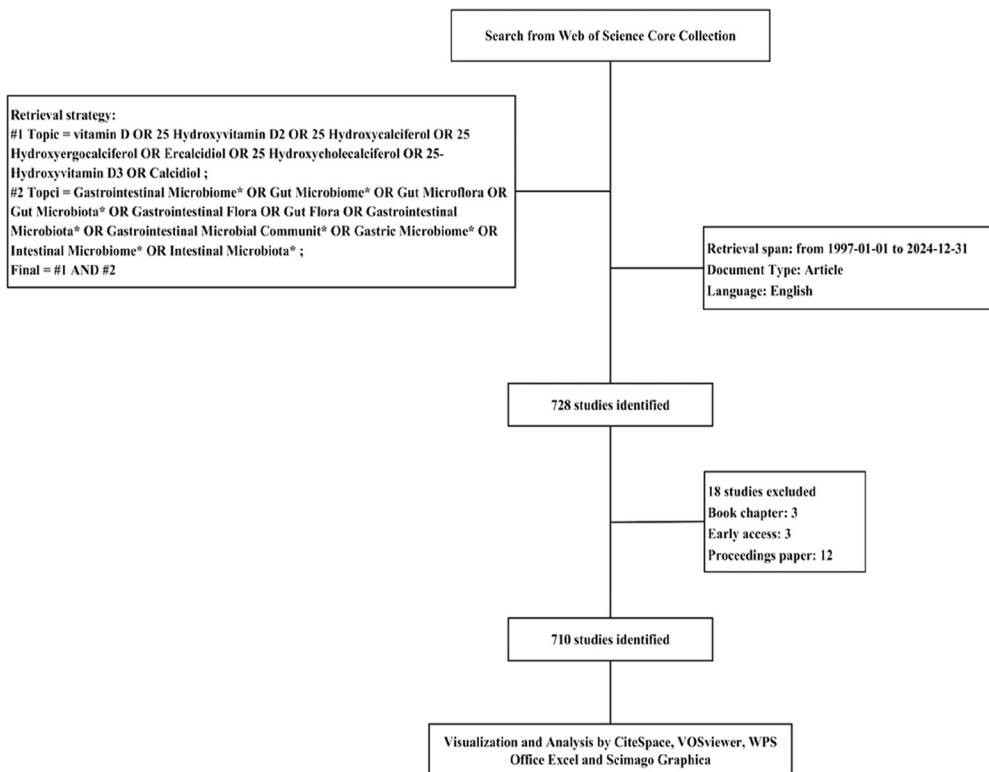


Fig. 1. Flowchart of the literature screening process for research on vitamin D and gut microbiota. Data were retrieved from the Web of Science Core Collection (1997–2024)

2.2. Data analysis

CiteSpace 6.3.R1 (Chen Chaomei, Drexel University, Philadelphia, USA) was used to construct dual-map overlay of journals, capture references with strong citation bursts, and analyse keywords co-occurrence. The retrieved literature was exported in TXT format and then imported into CiteSpace 6.3.R1 for further data processing and analysis, adjusting the parameter settings on the CiteSpace homepage: time slicing (January 1997 to December 2024), term source (title, abstract, author keywords, and keywords plus), and node filtering method (g-index, $k = 11$). The overlay visualisation map of journal co-citation showed the citation relationship between journals through coloured paths, and the topics covered by the journals. Impact Factors (IF, 2024) and JCR quartiles were obtained from the 2025 Journal Citation Reports (JCR, 2024) by Clarivate Analytics. References with strong citation bursts were used to identify the top-cited literature in the field at different times. CiteSpace provided modularity Q and weighted mean silhouette score based on network structure and cluster clarity. The closer the value of S was to 1, the more homogeneous the network was, and the clusters were convincing when S was greater than 0.7. Keywords were standardised and merged according to the Medical Subject Headings (MeSH) database to ensure terminological consistency. For instance, synonymous or hierarchical terms were consolidated, such as merging “gut microflora” into “gut microbiota”, “25-hydroxyvitamin D” into “vitamin D”, and “colon cancer” into “colorectal cancer”. A sensitivity analysis was conducted by expanding the document types to include “Reviews” and “Editorial Materials”.

VOSviewer v1.6.20 (Leiden University, Leiden, The Netherlands) was used to construct the overlay visualisation map of journals, the network visualisation map of journal co-citation analysis, and the co-citation network of cited references using the full counting method. The retrieved literature was exported in TXT format and then imported into VOSviewer v1.6.20 for further data processing and analysis. The association strength was applied as the default normalisation method to determine the proximity of nodes. The node size represents the number of articles published by the journals or the citation frequency of co-cited journals and references. The node colour represents the classification of these items or the time trend. The thickness of the lines between the nodes indicates the cooperative relationship between these items. Specific thresholds were set to ensure the clarity and representativeness of the networks: journals were included if they had published at least 5 papers; co-cited journals were analysed based on the top 50 most cited journals; and co-cited references were included with a minimum of 20 citations.

Microsoft Excel (version 6.9.0) was also applied to data drawing.

3. RESULTS

3.1. Publication outputs and trends

According to our search formula, 710 articles on vitamin D and gut microbiota have been retrieved from the publication of the first relevant article (1997) to 2024. As shown in Fig. 2, the entire period can be divided into two parts regarding the growth in the number of publications released each year: phase 1 (1997–2012) and phase 2 (2013–2024). The number of publications in the first phase was steady at a single-digit level at the beginning stage. The number of publications from 2013 has shown a noticeable year-on-year increase and has

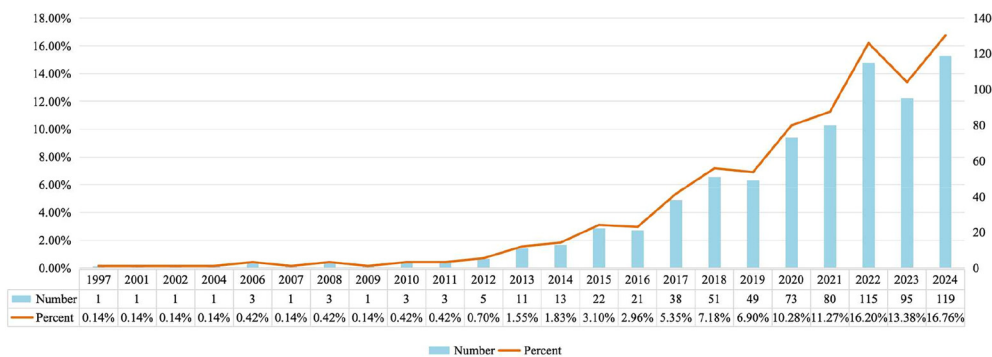


Fig. 2. Annual output and growth trend of research publications concerning vitamin D and gut microbiota (1997–2024). The bar chart illustrates the number of articles published each year, while the line graph represents the corresponding percentage of the total output. Data were sourced from the Web of Science Core Collection

increased significantly compared with the first phase. While there is a small drop in 2023, it grows again in 2024.

3.2. Analysis of journals and co-cited journals

In total, 345 journals published relative papers. The top 10 journals have published 147 articles, accounting for one-fifth of the total 710 articles (20.7%). As shown in Table 1, the journal with the most publications was Nutrients, with 37 related publications (5.2%), followed by Poultry Science ($n = 24$, 3.4%) and Frontiers in Nutrition ($n = 16$, 2.3%). The IF (2024) of the top 10 publishing journals ranged from 2.6 to 11.0, with an average IF of 5.1. The journal with the highest IF (2024) was Gut Microbes (IF = 11.0, Q1), followed by Frontiers in Immunology (IF = 5.9, Q1) and International Journal of Molecular Sciences (IF = 4.9, Q1). Besides, Fig. 3A shows the citation relationship between the journals. For example, Nutrients has a citation relationship with Gut Microbes, Scientific Reports, and Frontiers in Immunology.

Table 1. Top 10 journals for research of vitamin D and gut microbiota

Rank	Journal	Publications	IF	JCR
1	Nutrients	37	5.0	Q1
2	Poultry Science	24	4.2	Q1
3	Frontiers in Nutrition	16	5.1	Q1
4	International Journal of Molecular Sciences	11	4.9	Q1
5	Frontiers in Immunology	10	5.9	Q1
6	Gut Microbes	10	11.0	Q1
7	Journal of Nutrition	10	3.8	Q2
8	PLOS One	10	2.6	Q2
9	Scientific Reports	10	3.9	Q1
10	Frontiers in Microbiology	9	4.5	Q1

Table 2. Top 10 co-cited journals for research of vitamin D and gut microbiota

Rank	Co-cited journal	Citations	IF	JCR
1	Nutrients	1,048	5.0	Q1
2	PLOS One	1,039	2.6	Q2
3	Nature	917	48.5	Q1
4	Gut	712	26.2	Q1
5	PNAS	689	9.1	Q1
6	Gastroenterology	687	25.9	Q1
7	Science	636	45.8	Q1
8	American Journal of Clinical Nutrition	629	6.9	Q1
9	Scientific Reports	554	3.9	Q1
10	Journal of Nutrition	502	3.8	Q2

genetics and health/nursing/medicine journals were cited predominantly in medicine/medical/clinical journals.

3.3. Analysis of co-cited references and reference burst

There were 42922 co-cited references in this field. As shown in Table 3, The top 10 co-cited references were all cited more than 35 times, with three of them greater than 50 times. Figure 4 shows the co-citation relationship among the co-cited references, such as “Wu SP, 2015, Gut” and “Wang J, 2016, Nat Genet”, “Caporaso JG, 2010, Nat Methods” and “Wang Q, 2007, Appl Environ Mic”, “Waterhouse M, 2019, Eur J Nutr” and “Singh P, 2020, Sci Rep-UK”, etc.

In addition, as shown in Table 4, the top 20 references with the strongest citation bursts were identified through CiteSpace. The strongest burst (Ooi et al., 2013) (strength = 12) is a 2013 paper by Jot Hui Ooi published in the Journal of Nutrition, demonstrating that vitamin D modulates the gut microbiome and that 1,25(OH)₂D₃ or VDR deficiency causes dysbiosis, increasing susceptibility to gut injury. In the recent three years (2022–2024), the reference with the strongest citation burst (Singh et al., 2020) (strength = 10.01) was a clinical trial published by Parul Singh in Scientific Reports in 2020. The article, which supplemented 80 healthy vitamin D-deficient women with vitamin D, found a positive effect of vitamin D supplementation on the gut microbiota and the potential for microbial gut characteristics to influence vitamin D responses.

3.4. Analysis of keywords

As shown in Table 5 to Table 8, the keywords occurred at least three times were categorised into four areas: diseases, human gut microbiota, microbial metabolites, and mechanisms.

Table 5 divided diseases into four categories: inflammatory diseases, metabolic diseases, immune diseases, and other diseases. Half of the top 10 disease-related keywords were inflammatory diseases, with inflammatory bowel disease (*n* = 69) gaining the most attention, followed by a metabolic disease, obesity (*n* = 62), and ulcerative colitis (*n* = 46). Of the 10 keywords related to the human gut microbiota (Table 6), seven were categorised as beneficial taxa and three as pathogenic taxa. Within the beneficial taxa category, *Akkermansia muciniphila* (*n* = 10) showed the highest keyword frequency, followed by *Lactobacillus* (*n* = 9), *Bacteroides* (*n* = 6),

Table 3. Top 10 co-cited references on the research of vitamin D and gut microbiota

Rank	Co-cited reference	Information	Citations
1	QIIME allows analysis of high-throughput community sequencing data	Caporaso JG (2010), Nat Methods	62
2	Vitamin D regulates the gut microbiome and protects mice from dextran sodium sulfate-induced colitis	Ooi JH (2013), J Nutr	59
3	Novel role of the vitamin D receptor in maintaining the integrity of the intestinal mucosal barrier	Kong J (2008), Am J Physiol-Gastr L	58
4	Intestinal epithelial vitamin D receptor deletion leads to defective autophagy in colitis	Wu SP (2015), Gut	49
5	Effects of high doses of vitamin D ₃ on mucosa-associated gut microbiome vary between regions of the human gastrointestinal tract	Bashir M (2016), Eur J Nutr	43
6	Lack of vitamin D receptor causes dysbiosis and changes the functions of the murine intestinal microbiome	Jin DP (2015), Clin Ther	42
7	Genome-wide association analysis identifies variation in vitamin D receptor and other host factors influencing the gut microbiota	Wang J (2016), Nat Genet	41
8	Vitamin D and the gut microbiome: a systematic review of in vivo studies	Waterhouse M (2019), Eur J Nutr	39
9	Metagenomic biomarker discovery and explanation	Segata N (2011), Genome Biol	38
10	Linking long-term dietary patterns with gut microbial enterotypes	Wu GD (2011), Science	35

and *Bifidobacterium* ($n = 6$). At the species level, *Lactobacillus plantarum* ($n = 4$) and *Faecalibacterium prausnitzii* ($n = 3$) were also identified among the reported beneficial taxa. The pathogenic taxa included *Escherichia coli* ($n = 16$), *Helicobacter pylori* ($n = 3$), and sulphate-reducing bacteria ($n = 3$). In Table 7, the microbial metabolites studied so far involve short-chain fatty acids ($n = 43$), bile acids ($n = 33$), butyrate ($n = 16$), etc. Short-chain fatty acids and bile acids accounted for 40.9%, suggesting that they are the most studied vitamin D-related microbial metabolites in the human gut. Finally, mechanism-related keywords (Table 8) were mainly categorised as inflammatory, immune, metabolic, and others. The most focused category was immune-related keywords (mentioned 189 times), followed by metabolism-related keywords (mentioned 73 times) and inflammation-related keywords (mentioned 53 times). In terms of specific keywords, the most mentioned keyword was oxidative stress ($n = 43$), followed by insulin resistance ($n = 25$), T cells ($n = 24$), and antioxidant activity ($n = 20$).

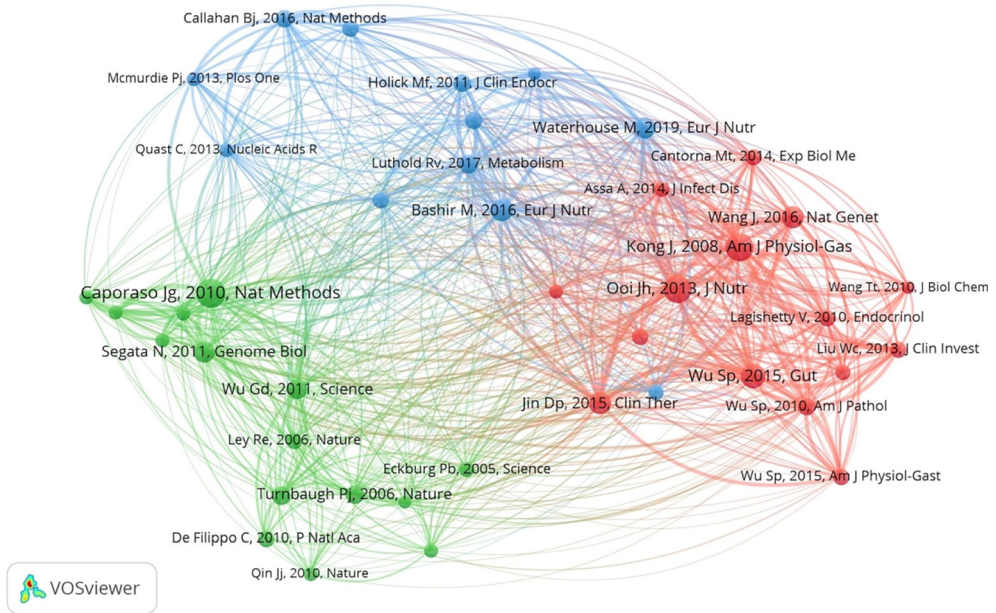


Fig. 4. The co-citation network of cited references concerning vitamin D and gut microbiota (1997–2024). This map was generated using VOSviewer (version 1.6.20) based on data from the Web of Science Core Collection. Each node represents a specific cited reference; the node size reflects the citation frequency, and the links between nodes indicate co-citation relationships. Different colours represent distinct research clusters identified through the clustering algorithm

3.5. Sensitive analysis

To evaluate the robustness of the identified research hotspots and address potential selection bias, a sensitivity analysis was performed by expanding the document types to include “Articles”, “Reviews”, and “Editorial Materials” (1997–2024). The results demonstrated a high degree of consistency between the “Article-only” dataset and the “Article + Review + Editorial” dataset (Table 9). Within the Diseases category, the top three keywords were IBD, obesity, and Crohn’s disease. In the Human Gut Microbiota category, the leading terms were *E. coli*, *H. pylori*, and *Lactobacillus*. Regarding Metabolites, the top three positions were held by short-chain fatty acids, bile acids, and fatty acids. Finally, in the Mechanisms category, oxidative stress, insulin resistance, and regulatory T cells emerged as the three most frequent keywords. The top three keywords within each category were consistent with the original findings, showing only marginal shifts in their rankings.

4. DISCUSSION

This article is the first to combine bibliometric and visualisation methods to analyse the relationship between vitamin D and gut microbiota and how vitamin D targets gut microbiota to affect

Table 4. Top 20 references with the strongest citation bursts

References	Year	Strength	Begin	End
Vitamin D regulates the gut microbiome and protects mice from dextran sodium sulfate-induced colitis	2013	12.42	2014	2018
Vitamin D deficiency promotes epithelial barrier dysfunction and intestinal inflammation	2014	6.35	2015	2019
Diet rapidly and reproducibly alters the human gut microbiome	2014	5.34	2016	2019
Lack of vitamin D receptor causes dysbiosis and changes the functions of the murine intestinal microbiome	2015	8.77	2017	2020
Intestinal epithelial vitamin D receptor deletion leads to defective autophagy in colitis	2015	6.21	2017	2020
Genome-wide association analysis identifies variation in vitamin D receptor and other host factors influencing the gut microbiota	2016	4.89	2017	2021
Effects of high doses of vitamin D ₃ on mucosa-associated gut microbiome vary between regions of the human gastrointestinal tract	2016	10.48	2018	2021
Vitamin D, immune regulation, the microbiota, and inflammatory bowel disease	2014	5.87	2018	2019
Gut microbiota interactions with the immunomodulatory role of vitamin D in normal individuals	2017	5.22	2018	2019
Effect of prenatal supplementation with vitamin D on asthma or recurrent wheezing in offspring by age 3 years: the VDAART Randomized Clinical Trial	2016	4.68	2018	2019
Bolus weekly vitamin D ₃ supplementation impacts gut and airway microbiota in adults with cystic fibrosis: a double-blind, randomized, placebo-controlled clinical trial	2018	4.93	2019	2022
Vitamin D signaling through induction of Paneth cell defensins maintains gut microbiota and improves metabolic disorders and hepatic steatosis in animal models	2016	5.62	2020	2021

(continued)

Table 4. Continued

References	Year	Strength	Begin	End
The endocrine vitamin D system in the gut	2017	5.1	2020	2021
Reproducible, interactive, scalable and extensible microbiome data science using QIIME 2	2019	6.68	2021	2024
The potential role of vitamin D supplementation as a gut microbiota modifier in healthy individuals	2020	10.01	2022	2024
Vitamin D metabolites and the gut microbiome in older men	2020	8.9	2022	2024
Relationships between vitamin D, gut microbiome, and systemic autoimmunity	2020	5.98	2022	2024
Vitamin D modulates intestinal microbiota in inflammatory bowel diseases	2021	6.27	2023	2024
The association between vitamin D and gut microbiota: a systematic review of human studies	2021	5.36	2023	2024
Vitamin D and intestinal homeostasis: Barrier, microbiota, and immune modulation	2020	4.91	2023	2024

health. The article explored the current research status and hotspot derivation in this field through journal, reference, and keyword analyses as the focus of this article, reflecting systematic understanding and perspectives over the past 27 years. Besides, to better present the research in the field to date, the article limited the document type to ARTICLE and did not include REVIEW. The primary research trends, core mechanisms, and emerging hotspots identified in this study are synthesised in Fig. 5.

4.1. General information

710 relevant articles were retrieved from WoSCC to capture the overall direction and research hotspots in the field. Before 2013, the number of annual publications in the field was less than 10, indicating that research in the field was in its infancy and the research base was relatively weak. Between 2013 and 2024, the number of annual publications began to increase significantly, with an average of 57.25 articles per year. In 2022, the annual publication number has exceeded 100, indicating that the research in this field is in a period of explosive growth, and related research has received more and more attention from scholars. Although the number fell below 100 in 2023, it rose again in 2024 and surpassed 2022.

Table 5. Diseases involved in studies of vitamin D and gut microbiota

Rank	Inflammatory Diseases	Count	Metabolic Diseases	Count	Immune Diseases	Count	Others	Count
1	Inflammatory bowel disease	69	Obesity	62	Allergy	19	Colorectal cancer	44
2	Ulcerative colitis	46	Osteoporosis	26	Multiple sclerosis	16	Alzheimer's disease	15
3	Crohn's disease	34	Metabolic syndrome	21	Irritable bowel syndrome	16	Schizophrenia	6
4	Asthma	21	Fatty liver	14	Systemic Lupus Erythematosus	4	Breast cancer	6
5	Colitis	16	Cardiovascular disease	10			Autism	4
6	Atopic dermatitis	13	Non-alcoholic steatohepatitis	9			Dementia	4
7	Experimental autoimmune encephalomyelitis	6	Atherosclerosis	8			Aberrant crypt foci	3
8	Eczema	5	Sarcopenia	7			Antibiotic-Associated Diarrhoea	3
9	Arthritis	4	Overweight	6			Anxiety	3
10	Myalgic encephalomyelitis	3	Hepatic steatosis	5			Parkinson's disease	3
11	Periodontal disease	3	Hypertension	5			Polycystic ovary syndrome	3
12	Rheumatoid arthritis	3	Non-alcoholic fatty liver disease	4			Sepsis	3
13			Type 1 Diabetes	4				
14			Cirrhosis	3				
15			Type 2 Diabetes	3				

Table 6. Human gut bacteria associated with vitamin D

Rank	Probiotics	Count	Pathogenic bacteria	Count
1	<i>Akkermansia muciniphila</i>	10	<i>Escherichia coli</i>	16
2	<i>Lactobacillus</i>	9	<i>Helicobacter pylori</i>	3
3	<i>Bacteroides</i>	6	<i>Sulphate-reducing bacteria</i>	3
4	<i>Bifidobacteria</i>	6		
5	<i>Butyrate-producing bacteria</i>	6		
6	<i>Lactobacillus plantarum</i>	4		
7	<i>Faecalibacterium prausnitzii</i>	3		

Table 7. Microbial metabolites associated with vitamin D

Rank	Metabolites	Count	Rank	Metabolites	Count
1	Short-chain fatty acids	43	11	Phenolic-compounds	4
2	Bile acids	33	12	Trans-retinoic acid	4
3	Fatty acids	18	13	Amino acid	3
4	Butyrate	16	14	Flavonoids	3
5	Polyunsaturated fatty-acids	13	15	Soy isoflavones	3
6	Polyphenols	11	16	Succinate	3
7	Retinoic acid	9	17	Trimethylamine n-oxide	3
8	Lipopolysaccharide	9			
9	Lithocholic acid	6			
10	Ursodeoxycholic acid	5			

4.2. Knowledge base

Among the top 10 journals in terms of publication number, in terms of the IF (2023), only one journal was IF > 10 (Gut Microbes, IF = 11.0), and three journals were 5≤IF < 10 (Frontiers in Immunology, IF = 5.9; Frontiers in Nutrition, IF = 5.1; and Nutrients, IF = 5.0). In terms of JCR partitioning, eight journals were Q1, and two journals were Q2, indicating that several relevant articles have been published in high-quality journals, but it is still challenging to publish relevant papers in journals with high IF. Among the top 10 most cited journals were highly specialised journals such as Nature and Science, which can influence the direction and foundation of scholarship in this field. The dual-map overlay of journals reflects that the main research in this field involved multiple levels, including molecular and genetics, as well as physiological mechanisms such as immunity. This suggests that the research in this area was more focused on the molecular level and basic research in the life sciences. Meanwhile, public health and clinical applications were noted.

A co-cited reference, cited together in several other publications, can be considered the research basis in an area (Wu et al., 2022). The most cited paper (Caporaso et al., 2010) (n = 62) was an introduction to QIIME (Quantitative Insights Into Microbial Ecology), an open-source bioinformatics tool for analysing and interpreting microbiome data originally released by Rob Knight’s team in 2010. QIIME and its updated version, QIIME 2, have played

Table 8. Mechanisms involved in studies of vitamin D and gut microbiota

Rank	Immunity	Count	Inflammation	Count	Metabolism	Count	Others	Count
1	T cells	24	Nuclear factor Kappa-b	17	Insulin resistance	25	Oxidative stress	43
2	Intestinal barrier	18	TNF- α	12	Lipid metabolism	15	Antioxidant activity	20
3	Barrier function	17	C-reactive protein	9	Farnesoid X receptor	8	Gut-brain axis	8
4	Nuclear factor Kappa-b	17	TGF- β	5	Calcium absorption	5	Carcinogenesis	6
5	Dendritic cells	15	TGR5	4	TGF- β	5	Antimicrobial activity	4
6	Regulatory T cells	15	Inflammatory responses	3	Tgr5	4		
7	Intestinal permeability	12	Toll-like receptors	3	Glucagon- like peptide-1 Nuclear receptors	4		
8	TNF- α	12			Bone metabolism	4		
9	Innate immunity	12				3		
11	Autophagy	7						
12	Intestinal epithelial cells	7						
13	Macrophages	6						
14	Paneth cells	5						
15	TGF- β	5						
16	TGR5	4						
17	Nuclear receptors	4						
18	Intestinal mucosal barrier	3						
19	Tight junction proteins	3						
20	Toll-like receptors	3						

an important role in microbiome research, providing reproducible and transparent data science that has greatly contributed to the rapid growth of the field (Bolyen et al., 2019). The second most cited paper (Ooi et al., 2013) ($n = 59$) was a mouse study published in 2013 by Jot Hui Ooi. This article presented that vitamin D regulates the gut microbiota and that lower expression of E-cadherin on gut epithelial and immune cells and fewer tolerogenic dendritic cells in VDR

Table 9. Top 10 most frequent keywords within each category in sensitivity analysis

Rank	Diseases	Count	Human gut bacteria	Count	Metabolites	Count	Mechanisms	Count
1	Inflammatory bowel disease	179	<i>Escherichia coli</i>	25	Short-chain fatty acids	125	Oxidative stress	108
2	Obesity	155	<i>Helicobacter pylori</i>	19	Bile acids	86	Insulin resistance	80
3	Crohn's disease	141	<i>Lactobacillus</i>	16	Fatty acids	60	Regulatory T cells	63
4	Ulcerative colitis	126	<i>Lactic acid bacteria</i>	13	Polyunsaturated fatty-acids	57	Nuclear factor kappa-b	57
5	Colorectal cancer	64	<i>Butyrate-producing bacteria</i>	13	Butyrate	23	Farnesoid X receptor	47
6	Multiple sclerosis	54	<i>Akkermansia muciniphila</i>	9	Retinoic acid	23	Tnf-α	41
7	Osteoporosis	50	<i>Bacteroides</i>	6	Polyphenols	17	Dendritic Cells	41
8	Asthma	50	<i>Sulphate-reducing bacteria</i>	6	Lithocholic acid	19	Lipid Metabolism	31
9	Metabolic syndrome	49	<i>Bifidobacteria</i>	6	Ursodeoxycholic acid	17	Intestinal permeability	29
10	Irritable bowel syndrome	37	<i>Lactobacillus plantarum</i>	6	Lipopolysaccharide	7	C reactive protein	22

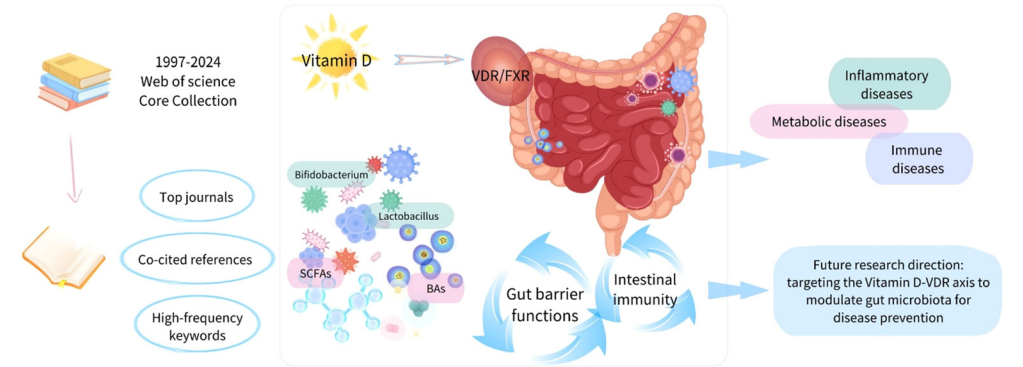


Fig. 5. A schematic framework of research trends and biological mechanisms in the field of vitamin D and gut microbiota (1997–2024)

and Cyp27b1 knockout mice result in dysbiosis and more severe intestinal inflammation. The third most cited paper (Kong et al., 2008) ($n = 58$), published in 2008 by Juan Kong, demonstrated through animal studies that vitamin D deficiency may compromise the mucosal

barrier, leading to an increased susceptibility to mucosal injury and an increased risk of developing inflammatory bowel disease (IBD).

4.3. Hotspots and frontiers

References with the strongest citation bursts represent emerging topics in the field of study at a given time, as these references exploded in the number of times they were cited by researchers during that period (Wu et al., 2022). Based on the content of the references with citation bursts in our study, it can be seen that the research was centred on the following topics: the role of vitamin D in the regulation of the intestinal flora and the mechanisms of the interactions, the relationship between vitamin D deficiency and intestinal disorders, the role of VDR, the role of vitamin D in the regulation of the immune system, and the clinical trials of vitamin D supplementation.

Keywords are the author's highly condensed and summarised content of the article. Through the co-occurrence analysis of keywords, we could quickly capture research hotspots in a certain field (Wu et al., 2022). According to our previous analysis, it can be found that vitamin D insufficiency/VDR gene deletion affects the homeostasis of human gut microbiota, alters the species and number of microbiota and microbial metabolites, and influences the course of various diseases, especially inflammatory and metabolic diseases through multiple mechanisms, such as immunity, inflammation, and metabolism.

4.3.1. Vitamin D affects disease progression. Several large epidemiological studies have shown that vitamin D deficiency increases the risk of several adverse health outcomes. Of the keywords that occurred at least three times, 44 were in the disease category, 12 were inflammatory and 15 were metabolic diseases. In terms of inflammatory diseases, half of them were related to intestinal inflammation. The most frequent occurrence is IBD, a chronic inflammatory disease of the gastrointestinal tract that includes Crohn's disease (CD) and ulcerative colitis (UC) (Kaplan, 2015). Both diseases are associated with an impaired intestinal barrier and dysbiosis of the gut microbiota (Ryan et al., 2020). Abnormal immune responses to the gut microbiota are responsible for the disease in genetically susceptible individuals. It has been observed that populations living in northern latitudes receive less UVB exposure, resulting in vitamin D deficiency, which in turn increases the prevalence of IBD (Shivananda et al., 1996; Ananthakrishnan et al., 2012; Schultz and Butt, 2012). A retrospective cohort study found that vitamin D deficiency was associated with parameters of IBD complexity and that increasing 25(OH)D concentrations over time were associated with decreased CD activity (Janssen et al., 2019). Vitamin D supplementation to IBD patients improves the short IBD questionnaire score, an indicator of disease specific health related quality of life (Hlavaty et al., 2014), and reduces the likelihood of disease recurrence (Gubatan et al., 2017).

In terms of metabolic diseases, multiple studies have found an inverse correlation between vitamin D levels and pro-inflammatory markers, insulin resistance, glucose intolerance, metabolic syndrome, obesity, and cardiovascular disease (Argano et al., 2023). The mechanism of vitamin D amelioration in metabolic diseases may be related to gut microbiota. Many metabolic diseases have similarly proposed a pathogenic role for gut microbial dysbiosis and increased intestinal permeability (Selber-Hnatiw et al., 2020). In turn, gut dysbiosis leads to more opportunistic pathogens than beneficial bacteria in the gut, which is thought to be associated with low-grade metabolic inflammation, providing the basis for pathological processes such as

obesity and insulin resistance (León-Pedroza et al., 2015). Among the metabolic diseases, our analysis showed that obesity has been studied the most. A meta-analysis enrolling 24,600 children and adolescents showed a relative risk of 1.41 (95% CI: 1.26–1.59) between obesity and vitamin D deficiency (Fiamenghi and Mello, 2021). In high-fat diet (HFD)-induced obesity mice, vitamin D₃ supplementation improved intestinal barrier function, endotoxemia, and some obesity parameters, including weight, blood lipid level, and inflammatory factor (Xiang et al., 2024). In a clinical trial study, vitamin D supplementation for six weeks in overweight or obese women resulted in significant reductions in body weight, waist circumference, and BMI compared with the control group (Khosravi et al., 2018). An umbrella meta-analysis of the anti-obesity properties of vitamin D found that the results of 14 meta-analyses showed that vitamin D supplementation significantly improved levels of obesity indices (e.g., BMI and waist circumference) compared with controls (Musazadeh et al., 2022).

4.3.2. Vitamin D affects gut microbiota composition. Alterations in the gut microbiota are one of the extra-skeletal functions of vitamin D. A meta-analysis of 14 interventions and 11 observational studies found that vitamin D supplementation to healthy adults, pregnant women, IBD patients, prediabetes patients, etc., was associated with significant changes in microbiota composition (Bellerba et al., 2021). Oral vitamin D supplementation to healthy adults significantly increased gut microbial diversity, with an increase in probiotics (*Bifidobacterium* spp. and *A. muciniphila*) (Singh et al., 2020) and a significant decrease in the relative abundance of gamma proteobacteria (*Escherichia/Shigella* spp. or *Pseudomonas* spp.) (Bashir et al., 2016). *Bifidobacterium pseudocatenulatum* and *A. muciniphila* contribute to host immune homeostasis and intestinal barrier integrity (Ottman et al., 2017; Wang et al., 2023). According to our analysis, *A. muciniphila* was the second most popular probiotic. *A. muciniphila* has potential benefits in alleviating autoimmune diseases and chronic inflammatory diseases such as IBD, obesity, and diabetes (Rodrigues et al., 2022). The ratio of Bacteroidetes to Firmicutes is critical in maintaining intestinal homeostasis, and a key feature of intestinal dysbiosis is the down-regulation of Bacteroidetes and an increase in Firmicutes (Turnbaugh et al., 2006). An animal experiment has found that low Bacteroidetes and high Firmicutes numbers are associated with metabolic syndrome and obesity (Turnbaugh et al., 2006). Vitamin D supplementation increases the ratio of Bacteroidetes to Firmicutes, which corrects the dysbiosis of the intestinal flora in obese mice (Singh et al., 2020; Xiang et al., 2024).

Vitamin D signalling contributes significantly to intestinal homeostasis. VDR, an ancient nuclear hormone receptor, has a biological role in regulating the physiologically active metabolite of 1,25(OH)₂D₃ (Newmark et al., 2017). VDR levels in the gut are higher than in other tissues and cell types (Barbáchano et al., 2017) and play an important role in immunity, proliferation, differentiation, endocrinology, and host-microbe interactions, and are important factors in gut microbiota homeostasis. VDR may be one of the targets of vitamin D for regulating gut microbiota. In the VDR knockout mice, probiotics *Lactobacillus* species and *A. muciniphila* were depleted in the faeces, whereas *Clostridium* was enriched (Jin et al., 2015; Su et al., 2016). Our analysis showed that the members of LAB were the most popular probiotics. The members of the genus *Lactobacillus* belonging to the phylum Firmicutes are the most important probiotics in the intestinal microbiome and have been found to balance intestinal barrier integrity and mucosal barrier defences and improve host immune responses (Rastogi and Singh, 2022). However, *Clostridium difficile* is a major cause of hospital-acquired

diarrhoea. Its toxins damage the cytoskeleton of intestinal epithelial cells, increasing permeability and activating the release of inflammatory mediators, resulting in intestinal injury and inflammation (Hirota et al., 2012).

4.3.3. Vitamin D affects intestinal immunity and barrier functions. Among the keywords, mechanism-related keywords were mentioned most frequently. Keywords like T cells, dendritic cells, innate immunity, and adaptive immunity, etc. emphasise the role of vitamin D in intestinal immunity. Keywords like barrier function, intestinal barrier, and intestinal permeability all suggest the importance of the intestinal barrier to health.

According to our analyses, vitamin D modulates multiple immune cells, inflammatory cytokines, and signalling pathways to maintain intestinal tolerance. Research have showed that $1,25(\text{OH})_2\text{D}_3$ can enhance the development of regulatory T cells and induce myeloid dendritic cells toward a tolerogenic phenotype, characterised by decreased CD40/CD80/CD86, low IL-12, and enhanced IL-10 secretion (Barragan et al., 2015; Hafkamp et al., 2022). Furthermore, vitamin D supplementation inhibits Th1 and Th17 cell production while improving the ratio of anti-inflammatory (IL-4 and IL-10) to inflammatory cytokines (TNF- α , IFN- γ , IL-2, IL-6, and IL-17) (Cantorna et al., 2015; Nielsen et al., 2018; Xiang et al., 2024).

At the molecular level, vitamin D downregulates multiple inflammatory pathways, including COX-2, NF-kappaB, MAPK, and TNF- α , thereby alleviating colitis (Garcia et al., 2020). Our analysis showed that the NF-kappaB has been studied most. NF-kappaB modulates innate and adaptive immunity and regulates the production of immunoregulatory proteins, anti-inflammatory cytokines, and other tolerogenic factors in the intestine (Mukherjee et al., 2024). Dysregulation of NF-kappaB drives various inflammatory pathologies like IBD (Mukherjee et al., 2024). In ulcerative colitis biopsies, $1,25(\text{OH})_2\text{D}_3$ inhibits the MLCK-MLC pathway by blocking NF-kB activation, thus preserving the mucosal barrier permeability (Du et al., 2015). In mice with carbon tetrachloride (CCl_4)-induced liver injury, a model relevant to gut barrier disruption, $1,25(\text{OH})_2\text{D}_3$ deficiency promotes lipid peroxidase accumulation, increases NADPH oxidases expression, and induces Kupffer cell infiltration, along with LPS/toll-like receptor 4 signalling-mediated proinflammatory response (Shibamoto et al., 2023).

Beyond these pathways, antimicrobial peptides (AMPs) have a remarkable function in the gut innate immunity. In mouse small intestinal crypts, Paneth cells secrete antimicrobial components, including defensins and lysozyme, upon sensing bacterial antigens (Ayabe et al., 2000). Low VDR levels in the gut are associated with impaired Paneth cells antimicrobial activity, defective autophagy, and downregulated lysozyme and AMPs. These alternations contribute to the development of chronic mucosal inflammation (Wu et al., 2015; Sun, 2016; Lu et al., 2021).

The colonic mucus barrier is the primary defence against intestinal pathogens. Research indicates that $1,25(\text{OH})_2\text{D}_3$ deficiency thins the mucus layer, thereby increasing bacterial translocation to the mesenteric lymph nodes (Zhu et al., 2019) and promoting disease progression. Vitamin D/VDR maintains intestinal barrier integrity by regulating intestinal epithelial TJ proteins (Su et al., 2016). For instance, vitamin D supplementation upregulates ZO-1 and occludin expressions in both T2DM and HFD-induced obesity rats (Hussein et al., 2022; Xiang et al., 2024). In clinical settings, $1,25(\text{OH})_2\text{D}_3$ treatment in active UC patients decreases the claudin-1 and claudin-2 protein levels while increasing claudin-4 and claudin-7 protein levels to enhance barrier function (Stio et al., 2016; Domazetovic et al., 2020). Mechanistically, Du et al. (2015) have found that VDR signalling protects mucosal integrity by abrogating MLCK-dependent TJ

dysregulation during colonic inflammation. Furthermore, VDR is essential for mucosal homeostasis, maintaining the integrity of the junction complexes and supporting the healing capacity of the colonic epithelium (Kong et al., 2008). In addition, the vitamin D/VDR pathway stimulates specialised epithelial cells, such as Paneth cells and lamina propria B cells, to limit the systemic entry of the microbiota and their products, ultimately ameliorating intestinal inflammation (Aggeletopoulou et al., 2023).

4.3.4. Microbial metabolites affect intestinal health. Changes in gut microbiota can lead to changes in metabolites. Many microbial metabolites have been shown to be implicated in disease progression. According to our analyses, the intestinal metabolites affected by vitamin D are mainly concentrated in short-chain fatty acids (SCFAs) and bile acids (BAs).

SCFAs are the end products of dietary fibre and resistant starch after fermentation by gut microbes, such as acetate, propionate, and butyrate (Hu et al., 2018). SCFAs act as signalling molecules into the systemic circulation, thereby influencing the metabolism and function of various peripheral tissues to combat metabolic diseases such as obesity and diabetes (den Besten et al., 2015). By stimulating GLP-1 secretion through activating GPCR receptors (GPR41/43), SCFAs can improve glucose metabolism and insulin secretion (Tolhurst et al., 2012). Our analysis showed butyrate is the most frequently mentioned short-chain fatty acid. Multiple studies have found a reduced abundance of butyrate-producing bacteria in T2DM (Qin et al., 2012; Arora and Tremaroli, 2021). Men with higher levels and higher activation ratios of $1,25(\text{OH})_2\text{D}_3$ are more likely to have butyrate-producing bacteria associated with better gut microbial health (Thomas et al., 2020). In a mice model of colitis, administration of the bacterial product butyrate increases intestinal VDR expression and inhibits inflammation (Sun, 2016). Thus, butyrate-producing bacteria become a possible target for disease control.

BAs are potent metabolic and immune signalling molecules predominantly expressed in the entero-hepatic system and innate immune cells (Biagioli and Carino, 2019). BAs are amphiphilic molecules that promote lipid emulsification and solubilisation, synthesised from cholesterol in the liver and then transported to the gut where BAs carry out metabolism by gut bacteria (Cai et al., 2022). In addition to their role in nutrient absorption, BAs are ligands for the VDR, connecting gut bacteria to host VDR function and playing an important role in regulating inflammation and immune function (Makishima et al., 2002; Ishizawa et al., 2018). Lithocholic acid, a secondary bile acid formed by a small proportion of intestinal bacteria in the *Clostridium* genus, induces VDR signalling to inhibit Th1 activation to control the adaptive immune response (Pols et al., 2017). Besides, the Farnesoid X receptor (FXR), a primary bile acid-activated receptors (BARs) highly expressed in the liver and intestine, plays a critical role in regulating bile acid (Clifford et al., 2021) and lipid homeostasis (Sinal et al., 2000). Studies showed that intestinal FXR was essential to the development of diet-induced obesity, insulin resistance, and non-alcoholic fatty liver disease (Li et al., 2013; Jiang et al., 2015). Parséus et al. (2017) have found that in HFD-induced obesity mice, the gut microbiota may promote weight gain and hepatic steatosis by modulating FXR signalling; conversely, FXR may contribute to obesity by altering the microbiota composition (Parséus et al., 2017). Furthermore, some research has highlighted a significant crosstalk between VDR and FXR. Honjo et al. (2006) showed that the VDR ligand may directly target the trans-activating function of FXR. Interestingly, intestinal epithelial VDR deletion significantly increased intestinal FXR expression, an effect further amplified by a HFD (Chatterjee et al., 2020). In addition, secondary BAs produced

by gut microbiota can activate the VDR, creating a feedback loop. Consequently, the interaction between the gut microbiota, VDR, and FXR is hypothesised to be vital for maintaining intestinal health and preventing diseases.

5. LIMITATIONS

Despite the comprehensive insights provided by this bibliometric analysis, several limitations should be acknowledged. First, this study restricted the search to English-language publications, which introduces a potential “language bias.” Significant research findings published in other languages (e.g., Chinese, German, or Spanish) were not captured, potentially leading to an underrepresentation of regional scientific contributions. Second, while our sensitivity analysis demonstrated that including “Review” and “Editorial Materials” did not significantly alter the core research hotspots, this approach might still exclude emerging data or interdisciplinary perspectives found in other forms of grey literature or conference proceedings. Finally, the hotspots and indicators revealed by bibliometric analysis exhibit a certain degree of lag. High-quality papers published recently (e.g., in late 2024) may not yet reflect their true academic influence through citation-based metrics. Future studies could benefit from integrating multi-source data and incorporating non-English literature to provide a more globally balanced perspective on the vitamin D-gut microbiota axis.

6. CONCLUSIONS

This bibliometric study can help researchers discover the publication status, research hotspots, and research trends of vitamin D and gut microbiota-related studies from 1997 to 2024. The current research suggests that the study of the interaction between the vitamin D/VDR signalling pathway and gut microbiota still holds a good potential for further investigation. Moving forward, more high-quality RCTs are warranted to evaluate the efficacy of targeting specific microbial taxa or modulating key metabolites through the vitamin D/VDR signalling pathway. Investigating how these interventions restore gut barrier integrity and immune homeostasis will be crucial for developing precise therapeutic strategies to manage inflammatory, metabolic, and immune-related diseases.

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