




## The role of diet in the management of psoriasis: a scoping review

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### Abstract

Psoriasis is a chronic, systemic, immune-mediated, inflammatory skin disease associated with significant comorbidities. Globally, there are an estimated 60 million people living with psoriasis (PLwP). There is a growing body of evidence on the role of diet in psoriasis management, and demand for dietary advice is high. However, there are no specific, evidence-based dietary guidelines. This scoping review summarises the literature on use and effectiveness of diet in the management of psoriasis to improve understanding of the evidence and assist PLwP and healthcare professionals (HCPs) to discuss diet. The findings were categorised into three themes: (1) dietary intakes of PLwP, (2) the perceived role of diet in psoriasis management and (3) dietary approaches to manage psoriasis symptoms. In cross-sectional studies PLwP were reported to have higher fat and lower fibre intakes compared with controls, and lower psoriasis severity was associated with higher fibre intake. However, research is limited. PLwP perceive diet to have an impact on symptoms and make dietary modifications which are often restrictive. Systematic reviews and RCTs found certain dietary approaches improved symptoms, but only in specific populations (e.g. PLwP with obesity and PLwP with coeliac disease), and evidence for supplement use is inconclusive. The grey literature provides limited guidance to PLwP; focusing on weight loss and associated comorbidities. Larger, controlled trials are required to determine dietary approaches for psoriasis management, especially in PLwP without obesity and non-coeliac PLwP. Further understanding of diet modification, information acquisition and experiences among PLwP will enhance holistic care for psoriasis management.

**Key words:** Nutrition: Psoriasis: Inflammatory skin disease: Nutritional dermatology: Psoriasis severity: Psoriasis management

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### Introduction

Psoriasis is a chronic, systemic, immune-mediated, inflammatory skin disease<sup>(1)</sup> which can have a substantial impact on quality of life (QoL) through both physical and psychological effects<sup>(2)</sup>. It typically presents as raised, scaly plaques on the skin<sup>(2)</sup> which can cause painful and debilitating symptoms<sup>(1)</sup> and is associated with significant arthritic, cardiovascular, metabolic and psychological comorbidities<sup>(1,3,4)</sup>. Globally, there are an estimated 60 million people living with psoriasis (PLwP)<sup>(5)</sup>.

Psoriasis affects males and females equally and is more common in adults compared with children<sup>(4,6)</sup>. The reported prevalence of psoriasis among adults varies globally, from 0.09%<sup>(7)</sup> to 11.43%<sup>(8)</sup>, and is more common in high-income countries and in regions with older populations<sup>(1,4)</sup>. The highest prevalence of psoriasis is seen in Australasia (1.99%), western Europe (1.92%), central Europe (1.83%) and North America (1.50%)<sup>(4)</sup>. However, only 19% of countries have epidemiological data on psoriasis<sup>(4,5)</sup>. In the United States psoriasis is one of the most common immune-mediated diseases, affecting 3% of adults<sup>(9)</sup>, and in the United Kingdom psoriasis affects an estimated 2% of the population, approximately 1.1 million people<sup>(5)</sup>.

There is no cure for psoriasis, and treatment is focused on symptom control. Studies show that PLwP who experience

improvements in disease severity commonly experience improvements in QoL<sup>(10,11)</sup>. However, satisfaction and adherence to some treatments are suboptimal due to side effects and dissatisfaction with the time taken and degree of improvement<sup>(1,12,13)</sup>. Long-term efficacy of psoriasis treatments has also been highlighted as a concern<sup>(14)</sup>. Psoriasis imposes a significant economic burden, which increases with the number and onset of psoriasis-related comorbidities<sup>(15,16)</sup>.

### Comorbidities

Psoriatic arthritis (PsA) is the most prevalent comorbidity of psoriasis, affecting approximately 30% of people living with the disease<sup>(17)</sup> and is more prevalent in those with severe psoriasis and those who have had the disease for a longer duration<sup>(18)</sup>. Compared with the general population, PLwP have an increased risk of cardiovascular disease (CVD)<sup>(3)</sup> and people with more severe psoriasis have increased odds of developing CVD, compared with those with mild-to-moderate psoriasis<sup>(19)</sup>. It has been suggested that psoriasis may be an independent risk factor for CVD<sup>(3)</sup>. Multiple cardiovascular risk factors are also associated with psoriasis including, type 2 diabetes<sup>(20)</sup>, obesity<sup>(21)</sup>, metabolic syndrome<sup>(22)</sup>, dyslipidaemia<sup>(23)</sup> and hypertension<sup>(22)</sup>. Furthermore,

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meta-analyses have also associated psoriasis with non-alcoholic fatty liver disease<sup>(24)</sup>, certain cancers<sup>(25)</sup> and inflammatory bowel disease<sup>(26)</sup>.

Psoriasis also has a substantial psychological impact. PLwP are 1.5 times more likely to have symptoms of clinical depression compared with healthy controls<sup>(27)</sup>. Living with a chronic condition, social stigmatisation and low self-esteem play a significant role in the development of depression in PLwP<sup>(1)</sup>, and emerging evidence suggests that systemic inflammation could also be playing a role in this relationship<sup>(28)</sup>.

### Aetiology and pathophysiology

The onset of psoriasis is multifactorial and is theorised to occur due to a combination of genetic and environmental factors which trigger a dysregulated immune response, which activates and sustains a cycle of inflammation<sup>(29,30)</sup>. Multiple components of the adaptive and innate immune systems are involved in this process<sup>(2,30)</sup>. The inflammatory cascade in psoriasis starts when plasmacytoid dendritic cells are activated, which promotes myeloid dendritic cell maturation through production of interferon (IFN)  $\alpha$ , IFN- $\gamma$ , tumour necrosis factor (TNF)- $\alpha$  and interleukin (IL)- $1\beta$ <sup>(31)</sup>. This leads to the activation and production of multiple cytokines, chemokines and antimicrobial peptides that promote an ongoing proinflammatory response. These include TNF- $\alpha$ , IL-6, IL-12 and IL-23, which activate T helper (Th)1, Th17 and Th22 cells<sup>(32)</sup>, which help to sustain the self-driving cycle of inflammation by producing TNF- $\alpha$ , IFN- $\gamma$ , IL-17 and IL-22<sup>(29-31,33,34)</sup>. This response leads to epidermal keratinocyte hyperproliferation and maintains a continual cycle of inflammation<sup>(29,30,33,35)</sup>. The key role that the IL-17/IL-23 axis plays in psoriasis, as well as specific cytokines such as TNF- $\alpha$ , is demonstrated by the efficacy of biological medications which target these specific cytokines and pathways<sup>(2)</sup> (Fig. 1).

Compared with healthy controls, PLwP have increased serum levels of proinflammatory cytokines<sup>(36,37)</sup>, continual elevated levels of which lead to chronic subclinical systemic inflammation<sup>(35)</sup>, hence why psoriasis is now seen as a systemic disease rather than solely dermatological<sup>(35)</sup>. The systemic inflammation seen in psoriasis is theorised to contribute to the pathogenesis of many of the associated comorbidities<sup>(2,35,38,39)</sup>.

### Lifestyle management for psoriasis

People living with psoriasis often look to lifestyle changes to manage their symptoms. The James Lind Alliance Priority Setting Partnership on psoriasis identified the top research priority for the disease as 'Do lifestyle factors such as diet, dietary supplements, alcohol, smoking, weight loss and exercise play a part in treating psoriasis?' in 2018<sup>(40)</sup>. Lifestyle factors such as smoking, alcohol intake and stress have been shown to affect disease severity<sup>(1)</sup>, but there is limited knowledge on the role of diet in managing psoriasis. Evidence suggests that diet can modulate immunological and inflammatory responses<sup>(41)</sup> and certain nutrients or dietary patterns could potentially worsen or

alleviate psoriasis symptoms<sup>(42)</sup>. However, there are no specific dietary guidelines for psoriasis.

There is a growing body of scientific literature regarding the role of diet in the management of psoriasis, alongside an increasing amount of 'popular' dietary advice<sup>(43-45)</sup>. Studies have shown that in PLwP dietary modification is common, and that many are self-initiating dietary changes<sup>(43-45)</sup>. It is therefore important for HCPs to familiarise themselves with the current literature on diet and psoriasis<sup>(43)</sup>. By doing so, they will be able to provide informed support, combat misinformation and discuss the role of diet in managing psoriasis with PLwP<sup>(43,46)</sup>. This is particularly important considering the associated comorbidities<sup>(1)</sup>.

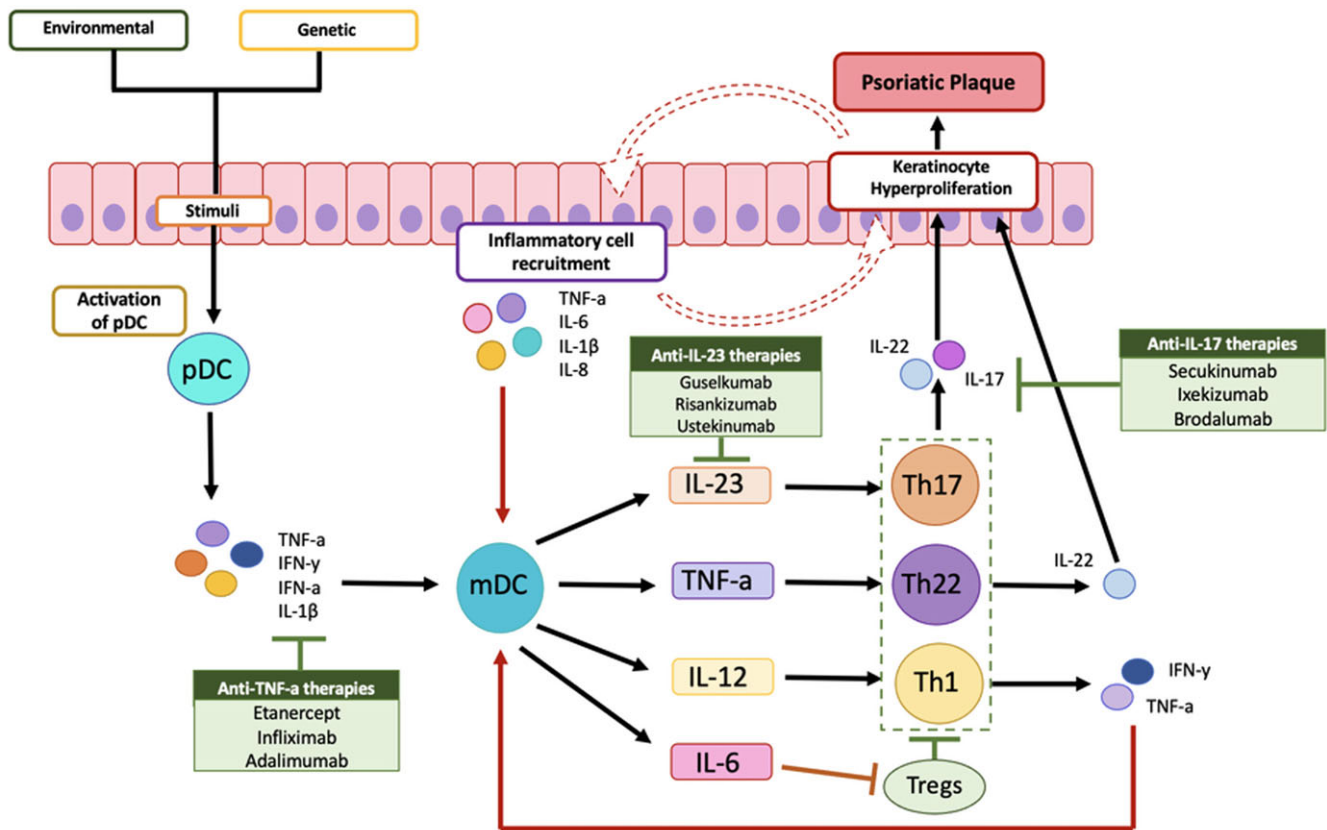
### Objectives of this review

The aim of this scoping review is to provide a comprehensive overview of the available evidence on the role of diet in the management of psoriasis. It will summarise the literature on dietary intake, the perceived role of diet in psoriasis management and evidence from dietary intervention studies on the impact of psoriasis symptoms. Additionally, this review will consider relevant grey literature on the role of diet in the management of psoriasis. A scoping review was determined as the most appropriate method given the broad study objective that will explore a range of sources, study designs and outcome measures.

### Methodology

This scoping review was conducted according to the updated methodological guidance for the conduct of scoping reviews of the Joanna Briggs Institute<sup>(47)</sup>. The search was conducted by:

- (1) Searching PubMed and SCOPUS using relevant key words and phrases. The key words used were: Psoriasis AND diet\* OR nutrition\* OR eat OR 'dietary patterns' OR 'dietary intake' OR 'dietary behaviours' OR 'dietary habits'.
- (2) Searching appropriate grey literature. Grey literature is defined as 'information produced on all levels of government, academia, business and industry in electronic and print formats not controlled by commercial publishing i.e., where publishing is not the primary activity of the producing body'<sup>(48)</sup>. For this scoping review we included grey literature produced by psoriasis organisations, nutritional societies and health authorities, and reports and guidelines on psoriasis management. The grey literature search strategy used was developed using methods from Godin *et al.*<sup>(49)</sup> for applying systematic search strategies to identify grey literature. Targeted searching of the identified resources, using appropriate search terms, was then undertaken.
- (3) Screening reference lists of relevant papers, reports and guidelines.
- (4) Searching for specific dietary modifications found to have been followed by people living with psoriasis (PLwP) individually as they emerged from the studies included in the review. The terms searched for on PubMed and SCOPUS



**Fig. 1.** Diagrammatic overview of the immune response, keratinocyte hyperproliferation and self-sustaining cycle of inflammation in psoriasis. pDC, plasmacytoid dendritic cells; mDC, myeloid dendritic cells; IL, interleukin; TNF, tumour necrosis factor; IFN, interferon; Th, T-helper cells; Tregs, regulatory T cells.

were Psoriasis AND the following: dairy-free, vegan, vegetarian, paleolithic, Pagano, ketogenic diet, low carbohydrate–high protein, red meat and nightshades.

Findings are reported according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses checklist for scoping reviews (PRISMA-ScR)<sup>(50)</sup> (see Table 1 for checklist). PRISMA diagram details the search and selection process applied during this scoping review. Studies were identified via database searches of PubMed and SCOPUS, and other methods. The grey literature was identified solely via other methods as detailed in the diagram (Fig. 2).

### Inclusion and exclusion criteria

Papers assessed for inclusion in this review were selected on the basis of relevance by title and abstract initially, and then full paper review. The review considered all methodologies of relevant studies; however, only those written in English, focused solely on psoriasis (all types of psoriasis were included), involving dietary approaches alone, and conducted in or addressing humans over 18 years old were included. The database search included papers published during the last 20 years, from 2002 until October 2022. The grey literature search was conducted between April and November 2022.

### Presentation of findings

The literature varied widely in methodology and type. As a result, this scoping review provides an overview of the current evidence according to three main themes: (1) dietary intakes of people living with psoriasis (PLWP), (2) the perceived role of diet in the management of psoriasis and (3) dietary approaches to manage psoriasis symptoms.

#### *Theme 1: dietary intakes of people living with psoriasis (PLWP)*

This theme reviews studies that explored the dietary intakes and habitual supplement use of people living with psoriasis (PLWP) (Table 2). The search identified nine studies that explored the dietary intakes of PLWP<sup>(45,51–58)</sup>. Among these, seven performed studies comparing the dietary intakes of PLWP with healthy controls<sup>(45,52–57)</sup>, one compared the dietary intake of PLWP with adults with other chronic inflammatory conditions and recommended national dietary guidelines<sup>(58)</sup> and six included studies compared the dietary intakes of PLWP depending on levels of psoriasis severity<sup>(51–54,56,57)</sup>. The search also identified two studies that investigated the habitual supplement use of PLWP compared with controls<sup>(59,60)</sup>. All studies were cross-sectional, seven used food frequency questionnaires (FFQs) to assess dietary intake<sup>(45,51–56)</sup>, one used 3 × 24-h dietary recall<sup>(58)</sup> and one study used a 7-d food recall<sup>(57)</sup>.

**Table 1.** The Preferred Reporting Items for Systematic reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) Checklist<sup>(50)</sup>

SECTION	ITEM	PRISMA-ScR CHECKLIST ITEM	REPORTED ON PAGE #
<b>TITLE</b>			
Title	1	Identify the report as a scoping review.	1
<b>ABSTRACT</b>			
Structured summary	2	Provide a structured summary that includes (as applicable): background, objectives, eligibility criteria, sources of evidence, charting methods, results, and conclusions that relate to the review questions and objectives.	1
<b>INTRODUCTION</b>			
Rationale	3	Describe the rationale for the review in the context of what is already known. Explain why the review questions/objectives lend themselves to a scoping review approach.	1–2
Objectives	4	Provide an explicit statement of the questions and objectives being addressed with reference to their key elements (e.g., population or participants, concepts, and context) or other relevant key elements used to conceptualize the review questions and/or objectives.	2
<b>METHODS</b>			
Protocol and registration	5	Indicate whether a review protocol exists; state if and where it can be accessed (e.g., a Web address); and if available, provide registration information, including the registration number.	2
Eligibility criteria	6	Specify characteristics of the sources of evidence used as eligibility criteria (e.g., years considered, language, and publication status), and provide a rationale.	2–3
Information sources*	7	Describe all information sources in the search (e.g., databases with dates of coverage and contact with authors to identify additional sources), as well as the date the most recent search was executed.	2–3
Search	8	Present the full electronic search strategy for at least 1 database, including any limits used, such that it could be repeated.	2–3
Selection of sources of evidence†	9	State the process for selecting sources of evidence (i.e., screening and eligibility) included in the scoping review.	2–3
Data charting process‡	10	Describe the methods of charting data from the included sources of evidence (e.g., calibrated forms or forms that have been tested by the team before their use, and whether data charting was done independently or in duplicate) and any processes for obtaining and confirming data from investigators.	2–3
Data items	11	List and define all variables for which data were sought and any assumptions and simplifications made.	2–3
Critical appraisal of individual sources of evidence§	12	If done, provide a rationale for conducting a critical appraisal of included sources of evidence; describe the methods used and how this information was used in any data synthesis (if appropriate).	N/A
Synthesis of results	13	Describe the methods of handling and summarizing the data that were charted.	3
<b>RESULTS</b>			
Selection of sources of evidence	14	Give numbers of sources of evidence screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally using a flow diagram.	5
Characteristics of sources of evidence	15	For each source of evidence, present characteristics for which data were charted and provide the citations.	6–7, 9, 11–22
Critical appraisal within sources of evidence	16	If done, present data on critical appraisal of included sources of evidence (see item 12).	N/A
Results of individual sources of evidence	17	For each included source of evidence, present the relevant data that were charted that relate to the review questions and objectives.	6–7, 9, 11–22
Synthesis of results	18	Summarize and/or present the charting results as they relate to the review questions and objectives.	3–25
<b>DISCUSSION</b>			
Summary of evidence	19	Summarize the main results (including an overview of concepts, themes, and types of evidence available), link to the review questions and objectives, and consider the relevance to key groups.	25–29
Limitations	20	Discuss the limitations of the scoping review process.	28
Conclusions	21	Provide a general interpretation of the results with respect to the review questions and objectives, as well as potential implications and/or next steps.	29
<b>FUNDING</b>			
Funding	22	Describe sources of funding for the included sources of evidence, as well as sources of funding for the scoping review. Describe the role of the funders of the scoping review.	29

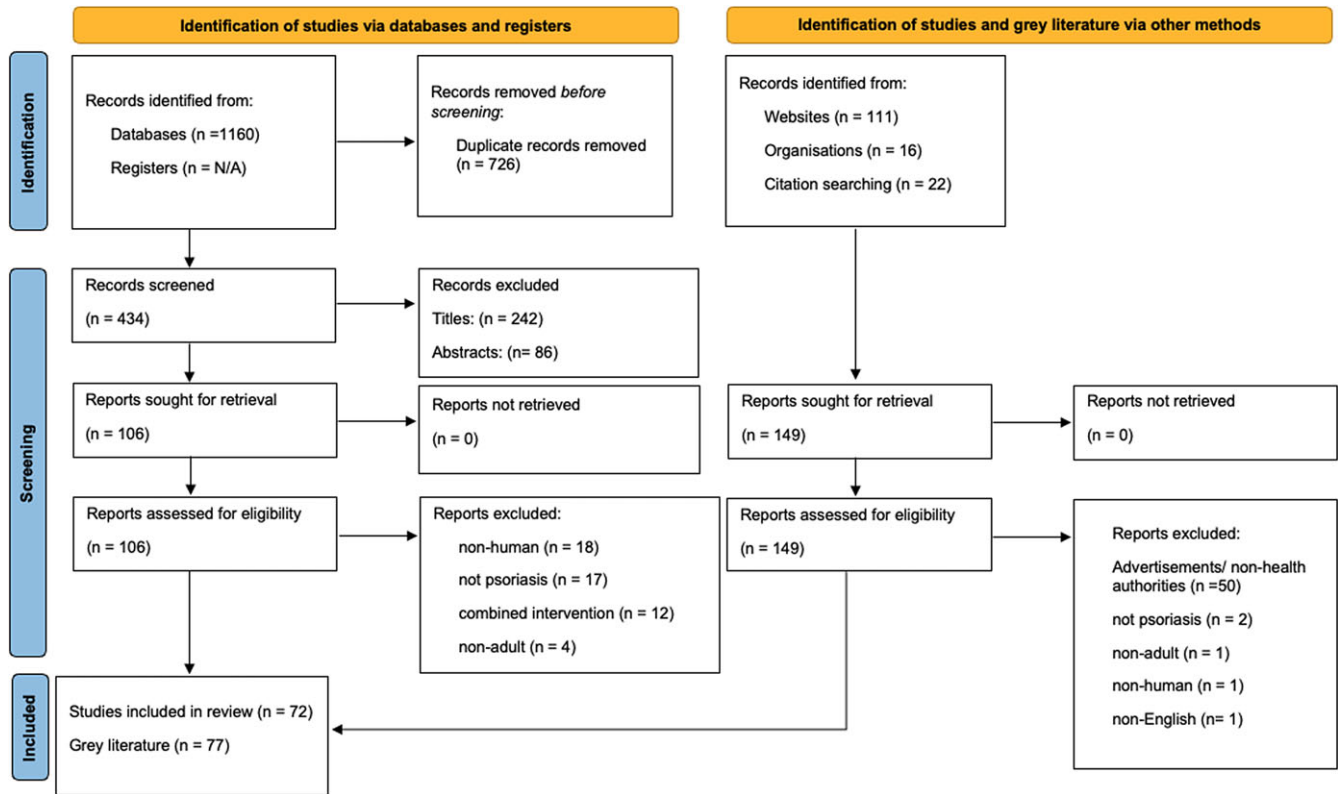


Fig. 2. PRISMA 2020 flow diagram for new systematic reviews which included searches of databases and other sources<sup>(167)</sup>.

Several common significant differences in dietary intakes of food groups were observed between controls and PLwP. Three studies found that fat intake was significantly higher in PLwP compared with controls<sup>(52,54,57)</sup>. A further study compared dietary intakes of PLwP with the recommended dietary guidelines in Poland and found that the mean dietary intakes of fat in PLwP were 148% of the recommended dietary intakes<sup>(58)</sup>. However, when compared with adults with other chronic inflammatory diseases, no significant difference in fat intake was observed. The absence of a healthy control group in this study meant that the findings are impossible to compare<sup>(58)</sup>.

Carbohydrate intake was also found to be significantly higher in PLwP compared with controls in two studies<sup>(52,57)</sup>. However, intake differences depended on the type of carbohydrate. Yazdanpanah *et al.*<sup>(52)</sup> found that total carbohydrate intake was significantly higher in PLwP compared with controls. Barrea *et al.* found that that total and simple carbohydrate intakes were significantly higher in PLwP compared with controls, whereas complex carbohydrate intake was significantly lower in PLwP when compared with controls<sup>(57)</sup>. This was the only study to assess carbohydrate intake dependent on type and was conducted in all white males ( $n = 82$ ), using a 7-d food recall, which makes the results difficult to compare<sup>(57)</sup>.

Fibre intake was found to be significantly lower in PLwP compared with controls in three studies<sup>(45,54,57)</sup>. A further study found that fibre intake of PLwP was only 53.3% in females ( $n = 17$ ) and 65% in males ( $n = 22$ ) of Polish recommended dietary guidelines (30 g/d). However, no significant difference was observed when compared with the dietary intakes of adults

with other chronic inflammatory conditions, and no healthy controls were included in this study<sup>(58)</sup>.

Findings on the dairy and sugar intakes of PLwP compared with controls were contrasting. Two studies found that sugar intake was significantly lower in PLwP in the United States<sup>(45,55)</sup>, whereas one study conducted in Japan found that PLwP consumed significantly more sugar than controls<sup>(56)</sup>. Regarding dairy intake, a large study in the United States found that PLwP consumed significantly less dairy compared with controls from the 2009 to 2010 National Health and Nutrition Examination Survey (NHANES)<sup>(45)</sup>. However, dairy intake was found to be significantly higher in PLwP compared with controls in a study conducted in Thailand<sup>(53)</sup>. Only one study found that PLwP consumed significantly less protein than controls<sup>(57)</sup>. However, this study was conducted in all white males ( $n = 82$ ), using 7-d food recall, which makes the results difficult to compare.

Regarding the intake of specific foods, several differences were observed between PLwP and controls. One study reported that PLwP had significantly higher intake of pulses compared with controls<sup>(56)</sup>, and significantly higher intakes of legumes were also reported in PLwP compared with controls<sup>(45)</sup>. Single studies reported that fruit and vegetables intakes were significantly higher in PLwP<sup>(45)</sup>, as well as coconut milk and soft drinks<sup>(53)</sup> compared with controls, whereas olive oil, eggs, berry fruits, brown rice/Riceberry, pickled foods and tree nuts<sup>(53)</sup>, and meat<sup>(56)</sup> intake were reported to be significantly lower in PLwP compared with controls. Differences in fish and seafood intakes between PLwP and controls were found in two studies; however, results were contrasting<sup>(53,56)</sup>.



**Table 2.** Summary of included studies under Theme 1: dietary intakes of people living with psoriasis

Authors, year, reference	Study region	Study design	Population characteristics	Control group	Significant findings
Polo <i>et al.</i> (2020) <sup>(51)</sup>	Brazil	Cross-sectional FFQ used for dietary intake assessment, PASI used to assess psoriasis severity	PLWP: <i>n</i> = 94, 57% female, mean age 54.9 years, mean PASI 5.3	N/A	2 dietary patterns were identified: Pattern 1 – processed and Pattern 2 – fresh food. In people with psoriasis, women ( <i>p</i> = 0.006) and those with higher income ( <i>p</i> = 0.003) were more likely to follow dietary pattern 2 – fresh food. Inverse association with adherence to Pattern 2 and cutaneous activity.
Yazdanpanah <i>et al.</i> (2021) <sup>(52)</sup>	Iran	Cross-sectional FFQ used for dietary intake assessment, PASI used to assess psoriasis severity	PLWP <i>n</i> = 45, 16 male, 29 female, mean age 40.4 years, mean BMI 26.92 kg/m <sup>2</sup> , mild-to-severe psoriasis severity	Non-psoriasis <i>n</i> = 43, sex- and age-matched	Compared with controls, PLWP had higher intakes of carbohydrates, fats, fibre, energy, vitamin E and folate ( <i>p</i> < 0.05). In the psoriasis group, higher dietary intake of fibre and vitamin E was significantly associated with lower disease severity ( <i>p</i> < 0.05).
Ingkapairoj <i>et al.</i> (2022) <sup>(53)</sup>	Thailand	Cross-sectional FFQ used for dietary intake assessment, PASI used to assess psoriasis severity	PLWP <i>n</i> = 100, 47 males, 53 females; mean age 45.87 years, mild-to-severe psoriasis severity	Non-psoriasis <i>n</i> = 100, sex- and age-matched	PLWP consumed significantly less olive oil ( <i>p</i> = 0.017), berry fruits ( <i>p</i> = 0.018), eggs (0.013), seafood ( <i>p</i> = 0.0001), fish ( <i>p</i> = 0.012), tree nuts ( <i>p</i> = 0.0001), rice/Riceberry ( <i>p</i> = 0.019) and pickled foods (0.0001) than controls. PLWP consumed significantly more dairy products ( <i>p</i> = 0.017), coconut milk ( <i>p</i> = 0.14) and soft drinks than controls ( <i>p</i> = 0.004). Those with lower psoriasis severities consumed significantly more vegetables. A higher consumption of red meat, belly meat and instant noodles was associated with greater psoriasis severity.
Affi <i>et al.</i> (2017) <sup>(45)</sup>	United States	Cross-sectional NHANES 2009–2010 dietary screening questionnaire (FFQ) used for dietary intake assessment	PLWP <i>n</i> = 1206 psoriasis, 73.3% female, mean age 50.4 years, psoriasis severity: 20.9% mild, 42.2% moderate, 36.9% severe	Non-psoriasis <i>n</i> = 2847, age- and sex-matched. From NHANES 2009–2010 dietary screening questionnaire.	PLWP consumed significantly less sugar, whole grain fibre, dairy and calcium ( <i>p</i> < 0.001), and consumed significantly more fruits, vegetables and legumes ( <i>p</i> < 0.01).
Kashani <i>et al.</i> (2021) <sup>(54)</sup>	Iran	Self-reported psoriasis severity Cross-sectional FFQ used for dietary intake assessment, ED-II used to assess dietary inflammation score, PASI used to assess psoriasis severity	PLWP <i>n</i> = 75, mean age 56.96 years, 48 males, 27 females, mean BMI 25.8 kg/m <sup>2</sup>	Non-psoriasis <i>n</i> = 74, age-, sex- and BMI-matched	PLWP consumed significantly more fat ( <i>p</i> ≤ 0.001), MUFA ( <i>p</i> ≤ 0.001), PUFA ( <i>p</i> = 0.0013), linoleic acids ( <i>p</i> = 0.007), linolenic acids ( <i>p</i> = 0.004), vitamin A ( <i>p</i> = 0.001), calcium ( <i>p</i> < 0.001) and iron ( <i>p</i> = 0.001) compared with controls. A high E-DII score (pro-inflammatory diet score) was associated with increased severity of psoriasis as measured by the PASI.



Table 2. (Continued)

Authors, year, reference	Study region	Study design	Population characteristics	Control group	Significant findings
Johnson <i>et al.</i> (2014) <sup>(55)</sup>	United States	Cross-sectional NHANES 2003–2006 dietary screening questionnaire (FFQ) used to assess dietary intake	PLwP <i>n</i> = 156	Non-psoriasis <i>n</i> = 6104 from the NHANES 2003–2006 dietary screening questionnaire	PLwP consumed significantly less sugar compared with controls ( <i>p</i> = 0.04).
Yamashita <i>et al.</i> (2019) <sup>(56)</sup>	Japan	Cross-sectional Self-administered diet history questionnaire (BDHQ), based on Japanese diet used to assess dietary intake (FFQ), PASI used to assess psoriasis severity	PLwP <i>n</i> = 70, 46 males, 24 females	Non-psoriasis <i>n</i> = 70, age- and sex-matched	Compared with controls, PLwP had significantly higher intake of fish/shellfish, pulses, sugar/sweeteners, vitamin B12 and vitamin D, and lower intake of meat. In the psoriasis group, those with a higher psoriasis severity (PASI) consumed a significantly higher amount of confection ( <i>p</i> = 0.03).
Barrea <i>et al.</i> (2015) <sup>(57)</sup>	Italy	Cross-sectional 7-d 24-h dietary recall used to assess dietary intake, PASI used to assess psoriasis severity	PLwP <i>n</i> = 41, plaque psoriasis, 100% male, treatment-naive	Non-psoriasis <i>n</i> = 41, age-, sex- and BMI-matched	Compared with controls PLwP had higher consumption of total and simple carbohydrates, total fat, PUFA, <i>n</i> -6: <i>n</i> -3 PUFA ratio and cholesterol; lower consumption of protein, complex carbohydrates, MUFA, <i>n</i> -3 PUFA and fibre ( <i>p</i> < 0.034). In PLwP higher MUFA consumption was associated with lower psoriasis severity ( <i>p</i> < 0.001); lower psoriasis severity was associated with lower total energy intake, saturated fatty acids, total PUFA, <i>n</i> -6 PUFA, <i>n</i> -6: <i>n</i> -3 PUFA ratio and simple carbohydrates and with higher <i>n</i> -3 PUFA, MUFA, fibre and complex carbohydrates ( <i>p</i> < 0.007).
Wasiluk <i>et al.</i> (2012) <sup>(58)</sup>	Poland	Cross-sectional 3 × 24-h dietary recall used to assess dietary intake, PASI used to assess psoriasis severity	PLwP <i>n</i> = 39, 22 males and 17 females	People with other chronic inflammatory skin disorders: <i>n</i> = 18, 8 males, 10 females	In PLwP fat intake exceeded the recommended daily intake and fibre intake was far lower than the recommended daily intake. Males and females with psoriasis consumed more monounsaturated fatty acids than controls.
Wilson, 2014 <sup>(60)</sup>	United States	Supplement use of people living with psoriasis		Non-psoriasis	No significant difference in dietary supplement use between controls and those with psoriasis over the past 30 d ( <i>p</i> = 0.416)
		Cross-sectional NHANES 2009–2010 dietary screening questionnaire used to assess supplement use	PLwP <i>n</i> = 184	Non-psoriasis <i>n</i> = 6027 from NHANES 2009 to 2010 dietary screening questionnaire	
Yousefzadeh <i>et al.</i> (2017) <sup>(59)</sup>	Iran	Cross-sectional Survey on supplement use over last 30 d, PASI was used to assess psoriasis severity	PLwP <i>n</i> = 138, plaque psoriasis, age 20–91 years	Non-psoriasis <i>n</i> = 138, age >20 years	Compared with controls, a significantly higher proportion of PLwP used supplements over the 30 d (72.5% versus 25.4%; <i>p</i> = 0.01). In psoriasis participants no significant difference was reported between supplement use and psoriasis severity.

FFQ, food frequency questionnaire; PASI, psoriasis area and severity index; PLwP, people living with psoriasis; BMI, body mass index; E-DII, energy-adjusted dietary inflammatory index; NHANES, US National Health and Nutrition Examination Survey; BDHQ, Japanese diet history questionnaire based on diets in Japan.

Differences in dietary intakes of specific nutrients between PLwP and controls were also reported in several studies. Polyunsaturated fatty acid (PUFA) intake was reported to be significantly higher in PLwP compared with controls in two studies<sup>(54,57)</sup>. Barrea *et al.* also found that *n*-6:*n*-3 PUFA ratio intake was significantly higher whereas *n*-3 PUFA intake was significantly lower in PLwP compared with controls<sup>(57)</sup>. However, Kashani *et al.* reported that both linoleic acid and linolenic acid intakes were higher in PLwP compared with controls<sup>(54)</sup>. Regarding monounsaturated fatty acids (MUFA) intake, the two studies that found significant differences in dietary intakes between PLwP and controls reported contrasting results<sup>(54,57)</sup>. A further study found that females with psoriasis consumed significantly more MUFA compared with females with other chronic inflammatory diseases<sup>(58)</sup>. However, no significant difference was seen in MUFA consumption in the male group, and there was no healthy control group to compare intakes with. Contrasting findings on vitamin A<sup>(54,55)</sup> and calcium intake<sup>(45,54)</sup> of PLwP and controls were also reported. Single studies reported that PLwP consumed significantly higher amounts of cholesterol<sup>(57)</sup>, vitamin B12, vitamin D<sup>(56)</sup> and iron<sup>(54)</sup>, and significantly lower amounts of vitamin E and folate<sup>(52)</sup> compared with controls.

One study explored the inflammatory potential of diets consumed by PLwP (*n* = 75) compared to age-, sex- and BMI-matched controls (*n* = 74) using FFQs and an energy-adjusted dietary inflammatory index (E-DII) as a predictive tool for inflammation potential of diets. They found that PLwP had a significantly higher energy-adjusted dietary inflammatory index with a median score of 0.10 (−1.59 to 0.83), a more pro-inflammatory diet, compared with controls where the median score was −2.14 (−2.96 to 1.00)<sup>(54)</sup>.

Differences in dietary intake between those with lower psoriasis severity and those with more severe psoriasis were also reported in several studies. Those with lower psoriasis severity had significantly higher intakes of fibre<sup>(52,57)</sup>, complex carbohydrates<sup>(57)</sup>, vegetables<sup>(53)</sup>, MUFA, *n*-3 PUFA<sup>(57)</sup> and vitamin E<sup>(52)</sup> compared with those with higher psoriasis severity. Furthermore, those with higher psoriasis severity had significantly higher intakes of total energy, saturated fatty acids, total PUFA, *n*-6 PUFA, *n*-6:*n*-3 PUFA ratio, simple carbohydrates<sup>(57)</sup>, confectionery<sup>(56)</sup> and red meat<sup>(53)</sup> compared with those with lower psoriasis severity. A single study reported that a high energy-adjusted dietary inflammatory index (E-DII) score was associated with increased severity of psoriasis<sup>(54)</sup>. Polo *et al.* found an inverse association with adherence to a 'fresh diet', characterised by predominantly fresh foods and a high consumption of fruits and vegetables, and cutaneous activity<sup>(51)</sup>. However, no definition of cutaneous activity was given, and PASI was recorded separately in this study.

Of note was that the definitions and methods for determining psoriasis severity varied between studies, and several studies did not include any definition of what constituted as lower or higher severity. Therefore, it is difficult to compare or understand the effects of these dietary intakes on psoriasis symptom severity.

**Habitual supplement use.** Two studies explored supplement use of PLwP compared with controls, over a 30-d period. These had mixed results. In the United States no significant difference in

supplement use was found between PLwP (*n* = 184) and matched controls (*n* = 6027)<sup>(60)</sup>, whereas in Iran, a significantly higher proportion of PLwP (*n* = 138) used supplements over the previous 30-d period, compared with controls (*n* = 138). However, no difference was reported between supplement use and psoriasis severity<sup>(59)</sup>.

### *Theme 2: the perceived role of diet in the management of psoriasis*

This theme comprises studies which explored the perceived role and use of diet in the management of psoriasis. Five studies were identified under this theme<sup>(44,45,61–63)</sup>, all of which were cross-sectional surveys and focused solely on the perceptions and experiences of people living with psoriasis (PLwP). No studies exploring the perceptions of healthcare professionals (HCPs) were identified in this review (Table 3).

Diet was perceived by the majority of PLwP to have an impact on their psoriasis symptoms in several studies. A survey on perceptions of dietary approaches to manage psoriasis of PLwP (*n* = 200) found that 62% of respondents perceived that following a specific diet could improve psoriasis, and 38% perceived that consuming specific foods could improve psoriasis<sup>(63)</sup>. A further study exploring dietary modifications and perceived effects on psoriasis symptoms over the past 2 years in PLwP (*n* = 43) found that 88.37% of respondents reported an improvement of psoriasis symptoms following a change in eating habits<sup>(61)</sup>. Although Afifi *et al.* found that in PLwP (*n* = 1206) 43.2% of respondents were not sure how diet affected their skin, 17.4% felt diet was slightly helping their skin, 16.7% felt diet was significantly helping their skin, and 2.2% reported that their skin condition was completely controlled by diet<sup>(45)</sup>.

People living with psoriasis commonly reported that they had made changes to their diet, the majority of which were self-prescribed. Afifi *et al.* found that in PLwP (*n* = 1206) most respondents (86%) reported using a dietary modification of some kind; of these, 40% reported following a specific diet to help their psoriasis, but only 30.7% of those that had changed their diet had discussed diet with a dermatologist<sup>(45)</sup>. A further study of 269 PLwP found that over half (52.2%) of participants had attempted between one and four dietary interventions, with 5.9% having tried more than five different dietary interventions<sup>(62)</sup>. This study also found that participants with two or more subtypes of psoriasis had tried following more diets or taking more supplements than those with only one. Additionally, an online survey exploring the dietary perceptions of PLwP (*n* = 50) found that most respondents (85%) reported that they had not received any advice from HCPs on diet. Overall, 20% had changed their diet to help psoriasis; of these, the majority (80%) had followed self-prescribed diets<sup>(44)</sup>.

The dietary changes made by PLwP were often restrictive, either following elimination diets or removing specific foods from diets. Afifi *et al.* found that a higher number of PLwP reported removing foods from their diets than those that reported trialling dietary additions<sup>(45)</sup>. Only three studies reported on specific dietary modifications trialled by PLwP and perceived symptom response.





**Table 3.** Summary of included studies under Theme 2: the perceived role of diet in the management of psoriasis

Authors, year, reference	Study region	Study design	Population characteristics	Findings
Festugato <i>et al.</i> (2011) <sup>(61)</sup>	Brazil	Cross-sectional	PLWP: <i>n</i> = 43	88.37% reported and improvement of psoriasis symptoms following a change in eating habits (eating habits were not specified). The positive aspects reported were reduction of erythema and scaling, milder outbreaks, delay in the onset of lesions, and improved quality of life.
Pham <i>et al.</i> (2021) <sup>(44)</sup>	France	Survey Cross-sectional	PLWP: <i>n</i> = 50	85% reported to have received no advice from HCP on diet; 20% had changed their diet to help psoriasis, and of these the majority (80%) had followed self-prescribed diets.
Afifi <i>et al.</i> (2017) <sup>(45)</sup>	United States	Online survey Cross-sectional	PLWP	86% reported use of a dietary modification, but only 30% had discussed diet with their doctor; 40% of participants had tried a specific diet for their psoriasis. The percentage of participants reporting skin improvement was greatest after reducing alcohol (53.8%), gluten (53.4%), nightshades (52.1%) and junk foods (50.4%) and after adding fish oil/omega-3 (44.6%), vegetables (42.5%) and oral vitamin D (41%). In diets tried, a favourable skin response was reported following the Pagano (72.2%), vegan (70%) and palaeolithic (68.9%).
		Survey	<i>n</i> = 1206, mean age 50.4 years, psoriasis severity: 20.9% mild, 42.2% moderate, 36.9% severe	
Dhinsa <i>et al.</i> (2021) <sup>(62)</sup>	United States	Psoriasis severity was self-reported Cross-sectional	PLWP	52.2% of participants had attempted between 1 and 4 dietary interventions, 5.9% had tried >5; participants with 2 or more subtypes of psoriasis tried more diets or supplements than those with 1 type.
		Survey	<i>n</i> = 269, mild-severe psoriasis	The percentage of participants reporting skin improvement was greatest after following ketogenic, (9 of 18 participants; 50%), Mediterranean (6 of 13; 46%), vegetarian (6 of 15; 40%) and gluten-free (9 of 25; 36%) diets. The most commonly tried supplements were oral vitamin D, fish oil and probiotics. Probiotics reported the most positive skin response, as well as vitamin D and fish oil.
Del Giglio <i>et al.</i> (2012) <sup>(63)</sup>	Italy	Cross-sectional	PLWP	62% of respondents perceived that following a specific diet regimen could improve psoriasis; 38% of respondents perceived that certain foods could improve psoriasis, with the majority identifying fruits and vegetables (60%) as having a beneficial effect on psoriasis and fish (10%); 46% perceived that foods could worsen psoriasis, with specific foods identified being sausages (20%), dairy products (8%), tomatoes (8%), spicy food (7%), chocolate (7%) and fried food (5%).
		Survey	<i>n</i> = 200, mean age 53 years, plaque psoriasis, moderate to severe psoriasis (PASI >5)	

PLWP, people living with psoriasis; HCP, healthcare professional; PASI, psoriasis area and severity index.

The most common dietary modification tried by PLwP reported across studies was reducing gluten or following a gluten-free diet<sup>(45,62)</sup>. Further diets trialled by PLwP were vegetarian, palaeolithic, ketogenic<sup>(62)</sup>, Mediterranean, low-carbohydrate-high-protein and the Pagano diet alongside reducing or removing dairy<sup>(45)</sup>. Common dietary components excluded were nightshades, alcohol and junk food<sup>(45)</sup>. Dietary additions reported to have been trialled by PLwP were increased consumption of fruit, vegetables and fish as well as vitamin D, omega-3/fish oil and probiotic supplements<sup>(45,62)</sup>.

The dietary modifications perceived to have a beneficial effect on symptoms were dairy free, vegan<sup>(45)</sup>, vegetarian, palaeolithic, the Pagano diet<sup>(45)</sup>, the ketogenic diet, the Mediterranean diet (MD) and a gluten-free diet (GFD)<sup>(45,62)</sup>. Reducing red meat, gluten, nightshades, alcohol and junk foods were also perceived to improve psoriasis skin symptoms by PLwP.<sup>(45,62)</sup> Respondents also reported improvement in skin symptoms after adding or increasing certain foods to their diet; fish, fruit and vegetables and supplements, specifically, omega-3, vitamin D and probiotics<sup>(45,62)</sup>. A further study in PLwP ( $n=43$ ) found that the majority (88.37%) of respondents reported an improvement of psoriasis symptoms following a dietary change<sup>(61)</sup>. However, the study did not specify which dietary changes were perceived to make a difference. The positive aspects reported after changing diet were reduction of erythema and scaling, milder outbreaks, delay in the onset of lesions, and improved quality of life<sup>(61)</sup>.

Dietary components were also perceived to be able to negatively affect psoriasis symptoms. In a study on those with moderate-to-severe psoriasis with a psoriasis area and severity index (PASI)  $>5$  ( $n=200$ ), 46% perceived that foods could worsen psoriasis. Specific foods identified by participants were sausages, dairy products, tomatoes, spicy food, chocolate and fried food<sup>(63)</sup>. However, Afifi *et al.* reported that 37% of respondents reported that they did not recognise any dietary triggers which may worsen their psoriasis<sup>(45)</sup>.

**Popular literature.** To be comprehensive, this review searched all diets and dietary modifications reported to have been tried by PLwP that were identified in the literature under theme 2 that had not been identified in the initial searches. PubMed and SCOPUS were searched using Psoriasis AND each of the diets or dietary modifications tried, using the same inclusion and exclusion criteria as described in the methods. No additional relevant results were found on PubMed or SCOPUS. This indicates that most of the diets that PLwP try, as reported in theme 2, have not been substantiated with any scientific evidence in relation to psoriasis management.

### Theme 3: dietary approaches for managing psoriasis symptoms

This theme included studies that explored specific dietary approaches and their impact on psoriasis symptoms. Dietary approaches were defined as specific dietary modifications followed to try to alleviate psoriasis symptoms through peer-reviewed investigations (Table 4).

The findings are presented under each relevant subtheme.

1. Specific diets
2. Dietary supplementation
3. Alternative dietary approaches

#### 1. Specific diets in the management of psoriasis

This review found that a handful of specific diets had been studied regarding the management of psoriasis: low-calorie diets (LCDs), very low-calorie ketogenic diets (VLCKD), intermittent fasting (IF), the Mediterranean diet (MD) and gluten-free diet (GFD).

**Low-calorie diets (LCDs).** Low-calorie diets (LCDs) are dietary interventions that restrict energy intake with the goal of weight loss. All LCD studies identified were conducted in people living with psoriasis (PLwP) who were living with obesity or overweight, defined as a BMI  $\geq 25$  kg/m<sup>2</sup>. Diets prescribed ranged from 500 kcal/d to 1600 kcal/d. This review did not include studies on the impact of medication, exercise or surgery for weight loss on psoriasis severity.

The beneficial effect of LCD on psoriasis severity in subjects with obesity is supported in recent systematic reviews<sup>(46,64)</sup>. A Cochrane review of lifestyle changes in the treatment of psoriasis identified six randomised control trials (RCTs) that evaluated the effects of a low-calorie diet in 499 participants with obesity<sup>(65)</sup>. The review found that low-calorie diets may lead to an improvement  $\geq 75\%$  from baseline psoriasis area and severity index in PLwP with obesity, compared with usual care. However, more RCTs with larger sample sizes are needed. The Cochrane review meta-analysis also found that known risk factors of the associated comorbidities of psoriasis were significantly reduced in the LCD group compared with the control groups at week 16<sup>(66-69)</sup>.

Several RCTs have found that LCDs significantly improve psoriasis severity in subjects who are living with overweight or obesity compared with controls<sup>(66,67)</sup>. Improvement in severity was also seen in an observational study at 12 weeks<sup>(70)</sup>. Only one study explored the long-term impact of an LCD on psoriasis severity and found that, after 48 weeks, weight loss in patients with psoriasis continued to have positive effects on symptom severity<sup>(71)</sup>.

However, an LCD followed by participants with obesity (BMI  $\geq 30$  kg/m<sup>2</sup>) for 24 weeks found no significant difference in PASI scores between the LCD group and the control<sup>(63)</sup>. However, baseline BMI was higher than in other studies, and the intervention LCD group may not have lost enough weight to produce the beneficial effect. In another study, although the LCD and control groups did not show a statistically significant difference in severity, the trend was towards reduced severity<sup>(68)</sup>.

**Very low-calorie ketogenic diet (VLCKD).** The main requirement to be defined as a ketogenic diet is carbohydrate restriction. In the studies identified in this review, the ketogenic diets also had a very low energy content (300–500 kcal/d) and were conducted only in PLwP with obesity or overweight. No systematic reviews on VLCKD and psoriasis symptoms were



**Table 4.** Summary of included studies under Theme 3: dietary approaches in the management of psoriasis symptoms

Authors, year, reference	Study design	Population characteristics	Control group	Findings	
Low-calorie diets (LCDs) Ko <i>et al.</i> (2019) <sup>(65)</sup>	Cochrane Systematic Review and Meta-Analysis	N/A	N/A	6 RCTs examined the effects of LCDs in 499 subjects with obesity. Compared with usual care, dietary intervention (strict caloric restriction) may lead to 75% or greater improvement from baseline in the psoriasis area and severity index (PASI 75). Dietary intervention may reduce the severity of psoriasis (low-quality evidence) and probably improves quality of life and reduces BMI (moderate-quality evidence) in participants with obesity when compared with usual care.	
Debbaneh <i>et al.</i> (2014) <sup>(46)</sup>	Review on the impact of weight loss interventions on psoriasis	N/A	N/A	Weight loss reduced BMI and led to improved PASI and DQLI for participants with obesity or overweight with psoriasis.	
Gisondi <i>et al.</i> (2008) <sup>(67)</sup>	RCT	PLwP	PLwP	Weight loss improved the response of patients with obesity and moderate-to-severe chronic plaque psoriasis to low-dose cyclosporine therapy: a reduction of 75% of PASI response was achieved by 66.7% of participants following a low-calorie diet and by 29.0% of patients treated with cyclosporine alone ( $p < 0.001$ ).	
Jensen <i>et al.</i> (2013) <sup>(68)</sup>	RCT	PLwP treated with cyclosporine alongside a LCD versus PLwP treated with cyclosporine alone for 24 weeks	$n = 30$ , 15 females, 15 males, moderate-to-severe psoriasis PLwP	$n = 31$ , 15 males, 16 females, moderate-to-severe psoriasis PLwP	The LCD group lost significantly more weight than the routine diet group ( $p < 0.001$ ). LCD group achieved a greater reduction in PASI ( $p = 0.06$ ) and greater improvement in DQLI ( $p = 0.02$ ) compared with control group.
Jensen <i>et al.</i> (2016) <sup>(71)</sup>	Follow-up study of RCT (Jensen <i>et al.</i> 2013)	The intervention group received an LCD (800–1000 kcal/d) for 8 weeks to induce weight loss, followed by 8 weeks of reintroduction of normal food intake, reaching 1200 kcal/d. Control group followed routine diet for 16 weeks	$n = 30$ , subjects with obesity BMI $>27$ kg/m <sup>2</sup> , moderate psoriasis PLwP	$n = 30$ , subjects with obesity BMI $>27$ kg/m <sup>2</sup> , moderate psoriasis PLwP	Changes in the severity of psoriasis (PASI and DLQI) were maintained after 48 weeks.
Del Giglio <i>et al.</i> (2012) <sup>(63)</sup>	RCT	2 periods: the LCD period (16 weeks) followed by weight-loss maintenance period (48 weeks)	$n = 30$ , subjects with obesity BMI $>27$ kg/m <sup>2</sup> , moderate psoriasis PLwP	$n = 30$ , subjects with obesity BMI $>27$ kg/m <sup>2</sup> , moderate psoriasis PLwP	No significant difference in PASI scores between the LCD group and the control group.
Guida <i>et al.</i> (2014) <sup>(69)</sup>	RCT	LCD or free diet (control) for 24 weeks, followed up for an additional 12 weeks	$n = 22$ , subjects with obesity BMI $\geq 30$ kg/m <sup>2</sup> , moderate-to-severe psoriasis PLwP	$n = 20$ , subjects with obesity BMI $\geq 29$ kg/m <sup>2</sup> , moderate-to-severe psoriasis PLwP	At 3 and 6 months, PASI was significantly reduced in patients in the LCD high $n$ -3 PUFA group compared with controls ( $p < 0.05$ ), as well as itch scores ( $p < 0.05$ ).
		The intervention group followed an LCD of 20 kcal/kg/d that was $n$ -3 polyunsaturated fatty acids rich	$n = 22$ , subjects with obesity BMI $\geq 30$ kg/m <sup>2</sup> , mild-to-severe psoriasis	$n = 20$ , subjects with obesity BMI $\geq 30$ kg/m <sup>2</sup> , mild-to-severe psoriasis	

**Table 4.** (Continued)

Authors, year, reference	Study design	Population characteristics	Control group	Findings
Al-Mutairi <i>et al.</i> (2014) <sup>(66)</sup>	RCT	PLwP	PLwP	At week 24, mean weight loss was significantly higher in the LCD group compared with the control group. A reduction in PASI of 75% was achieved by a significantly higher percentage of the LCD (85.9%) compared with the control group (59.3%) ( $p < 0.001$ ).
	LCD ( $\leq 1000$ kcal/d) for 8 weeks	$n = 131$ , subjects with obesity, receiving biologic therapy	$n = 131$ , subjects with obesity, receiving biologic therapy	
	PASI was used to assess severity, scores were assessed at baseline and every 4 weeks up to 24 weeks			
Roongpisuthipong <i>et al.</i> (2013) <sup>(70)</sup>	Single-arm trial	PLwP	N/A	At week 12 number of subjects achieving PASI50 was 50%. Mean improvement in Dermatology Life Quality Index was 62.5%.
	LCD alongside topical treatment was compared with baseline weight and PASI in patients with obesity with chronic stable plaque-type psoriasis at weeks 12 and 24	$n = 10$ , BMI $\geq 30$ kg/m <sup>2</sup> , plaque psoriasis		
<i>Very low-calorie ketogenic diet (VLCKD)</i>				
Castaldo <i>et al.</i> (2021) <sup>(73)</sup>	Open-label single-arm study	PLwP	N/A	At 4 weeks, no significant difference in weight loss compared with baseline. A significant improvement in PASI, VAS itch severity and DLQI was reported ( $p \leq 0.05$ ) compared with baseline.
	4-Week very-low-calorie (<500 kcal/d) protein-based diet providing 10–20 g of carbohydrates (from vegetables, 400–500 g/d), 20–30 g of lipids and 1.4 g/kg of ideal body weight of protein per day	$n = 30$ , 11 males and 19 females, subjects with obesity or overweight $>25$ kg/m <sup>2</sup> BMI, mean BMI 30.82 kg/m <sup>2</sup> , mean age 42.8 years, mean PASI 8.69, plaque psoriasis		
	PASI was used to assess psoriasis severity, VAS was used to assess itch severity, and DLQI was taken to assess quality of life			
Castaldo <i>et al.</i> (2020) <sup>(73)</sup>	Open-label single-arm study	PLwP	N/A	At week 10: a significant reduction in body weight, PASI score, and itch severity and DLQI ( $p \leq 0.001$ ) compared with baseline.
	10-Week 2 phase weight-loss programme: 4-week protein-sparing, <500 kcal/d, followed by 6-week balanced hypocaloric, low glycaemic index, Mediterranean-like diet (25–30 kcal/kg of ideal body weight)	$n = 37$ , subjects with overweight or obesity $>25$ kg/m <sup>2</sup> BMI, mean BMI 31.7 kg/m <sup>2</sup> , drug naive, mean PASI 13.8, chronic moderate-to-severe plaque psoriasis		
	PASI was used to assess psoriasis severity, VAS was used to assess itch severity, and DQLI was taken			
Castaldo <i>et al.</i> (2016) <sup>(72)</sup>	Case study	PLwP	N/A	At 3 months a complete remission and improved response to biologics following diet regimen. Compared with baseline the participant experienced a significant weight loss (92 kg versus 67.4 kg), improvement in PASI (15 versus 0.3) and DLQI score (12 versus 1).



Table 4. (Continued)

Authors, year, reference	Study design	Population characteristics	Control group	Findings
Mediterranean diet (MD) Phan <i>et al.</i> (2018) <sup>(75)</sup>	First stage: 4 weeks VLCKD (~300 kcal/d). Second stage: hypocaloric Mediterranean-like diet at low glycaemic index for 6 weeks	$n = 1$ , female, 40 years old, subjects with obesity BMI 35 kg/m <sup>2</sup> , severe plaque psoriasis		
	Prospective, web-based questionnaire study of respondents from the French NutriNet-Santé cohort	PLwP	Non-psoriasis	Patients with severe psoriasis displayed low levels of adherence to the Mediterranean diet. Patients with severe psoriasis had a higher risk of having a MEDI-LITE score of 0–7 (low adherence to the Mediterranean diet) compared with patients with non-severe psoriasis and patients without psoriasis.
Barrea <i>et al.</i> (2015) <sup>(77)</sup>	A Mediterranean diet adherence score (MEDI-LITE) was calculated for each participant using the average of 3–15 24-h dietary records gathered during the first 2 years after inclusion	Severe psoriasis: $n = 746$ , 76.9% females, mean age 46.8 years Non-severe psoriasis $n = 2308$ , 72.1% female, mean age 48.3 years	$n = 27\ 828$ , 76.6% females, mean age 47.8 years	
	Cross-sectional	PLwP	Non-psoriasis	A higher percentage of PLwP had a low or average adherence compared with the control group (30.6% versus 4.8%, $p < 0.001$ and 51.7% versus 77.5%, $p = 0.004$ , respectively). No significant differences in those with a high PREDIMED score (17.7% versus 17.7%)
Molina-Leyva <i>et al.</i> (2019) <sup>(76)</sup>	PREDIMED 14-item questionnaire, was used to assess adherence to the Mediterranean diet, PASI used to assess psoriasis severity	$n = 61$ patients, 49 males and 13 females, mean age: 50.2 years, mild-to-severe psoriasis.	$n = 61$ age-, sex- and BMI-matched.	Individual MD components: EVOO ( $p < 0.001$ ) and fish consumption ( $p = 0.005$ ) had an independent predictive value for PASI score. PREDIMED score was a major predictor of PASI ( $p = 0.007$ ).
	Cross-sectional	PLwP	N/A	Psoriasis severity was lower in participants with greater adherence to the Mediterranean diet for all measurements, PASI – $p = 0.007$ , BSA – $p = 0.009$ , PGA – $p = 0.01$ , subjective – $p = 0.004$ .
Korovesi <i>et al.</i> (2019) <sup>(76)</sup>	PREDIMED 14-item questionnaire was used to assess adherence to the Mediterranean diet, PASI, BSA, PGA and subjective responses were used to assess psoriasis severity	$n = 89$ , mild-to-severe psoriasis		
	Cross-sectional	PLwP	Non-psoriasis	Compared with PLwP, controls presented a higher adherence to the Mediterranean diet ( $p = 0.01$ ) with a higher MedDietScore ( $p < 0.001$ ). MedDietScore correlated negatively with PASI ( $p = 0.001$ ) and DLQI ( $p < 0.001$ ). MedDietScore was a significant negative predictor of PASI ( $p = 0.02$ ) and DLQI ( $p = 0.06$ of borderline significance) adjusting for age, gender, BMI and hsCRP.
	MedDietScore was used to assess adherence to Mediterranean diet. PASI and DLQI was used to assess severity of psoriasis	$n = 69$ , 35 men, 34 females, treatment naive, mean age 47.7 years, mean BMI of 28.9 kg/m <sup>2</sup> , moderate-to-severe psoriasis, mean DLQI of 9.5	$n = 69$ , age-, sex-, BMI-matched	Specific items of the MedDietScore were inversely associated with psoriasis severity; legumes, fish and EVOO ( $p < 0.05$ ). PASI positively correlated with dairy products ( $p = 0.002$ )



**Table 4.** (Continued)

Authors, year, reference	Study design	Population characteristics	Control group	Findings
<i>Gluten-free diet (GFD)</i> Bhatia <i>et al.</i> (2014) <sup>(79)</sup>	Review and meta-analysis	N/A	N/A	A gluten-free diet may potentially be beneficial in patients with coeliac antibody-positive psoriasis, but additional studies are needed to confirm this.
Michaëlsson <i>et al.</i> (2003) <sup>(81)</sup>	RCT	PLwP	N/A	At 3 months 73% of those with psoriasis with IgA and/or IgG AGA had a lower PASI score. Participants with elevated IgA AGA and/or IgG AGA showed a significant decrease in their mean PASI score after 3 months on a GFD. Of the six patients without AGA, there was no change in two and a pronounced deterioration in four after following a GFD for 3 months.
Addolorato <i>et al.</i> (2003) <sup>(84)</sup>	Case study	GFD followed 3-month period. Blood samples were drawn at baseline and at 3 and 6 months for assays of serum levels of IgA AGA and IgG AGA. PASI was used to assess psoriasis severity n = 37, 31 with IgA and/or IgG AGA, 6 without IgA and/or IgG antibodies to gliadin (IgA AGA and/or IgG AGA), 15 females, 22 males, mean age 45-13 years PLwP n = 1, coeliac disease not responding to usual treatment	N/A	Rapid regression of psoriasis after gluten-free diet
Kolchak <i>et al.</i> (2018) <sup>(82)</sup>	Observational	PLwP	N/A	Improvement of psoriatic lesions was observed in all patients with positive gliadin IgA antibodies. PLwP who had high levels of gluten-specific antibodies (n = 8) (IgA against gliadin peptides) saw a 36% improvement in PASI score following a GFD for 1 year. Those with higher levels of gluten-specific antibodies (n = 5) saw an even greater improvement, 56% reduction in PASI, following a GFD for 1 year.
De Bastiani <i>et al.</i> (2015) <sup>(83)</sup>	Observational	GFD for 1 year in PLwP with positive gliadin IgA antibodies; PASI was used to assess severity n = 13, 27–56 years old, PASI >2.4 PLwP	N/A	At 6 months GFD was associated with a significant improvement of skin lesions in 7 out of 8 participants with psoriasis.
Zamani <i>et al.</i> (2010) <sup>(86)</sup>	Case study	GFD for 6 months n = 8, positive for anti-tissue transglutaminase antibodies diagnosis of CD was confirmed histologically PLwP	N/A	No improvement in psoriasis severity after 6 months of following a GFD in 3 patients with coeliac disease or gluten-specific antibodies.
<i>Intermittent fasting (IF)</i> Almutairi & Shaaban, 2022 <sup>(87)</sup>	Observational	GFD for 6 months n = 3; 1 female, 2 males, mean age 28.3 years, 2 with increased IgA-tissue-transglutaminase antibodies, 1 with confirmed CD PLwP	N/A	Mean PASI was significantly reduced compared to baseline (p = 0.001). No significant difference in weight change in 102 (84.30%) patients, 14 (11.57%) gained 1 kg and 5 (4.13%) gained 2 kg. However, no patient recorded loss of weight.



Table 4. (Continued)

Authors, year, reference	Study design	Population characteristics	Control group	Findings
Damiani <i>et al.</i> (2019) <sup>(68)</sup>	Ramadan fasting for 1 month (2 main meals/d, one before sunrise and one after, refrain from eating or drinking during daylight hours, smoking and sex), PASI and BSA scores were used to assess psoriasis severity Observational	<i>n</i> = 121, stable chronic plaque psoriasis, mean PASI score of 4.36 PLwP	N/A	Significant reduction in mean PASI score after 1 month of Ramadan fasting compared with baseline ( <i>p</i> = 0.0001).
<i>Omega-3 Polyunsaturated Fatty Acids (PUFA)</i> Yang <i>et al.</i> (2019) <sup>(93)</sup>	Ramadan fasting for 1 month (2 main meals/d, one before sunrise and one after, refrain from eating or drinking during daylight hours, smoking and sex), PASI was used to assess psoriasis severity Meta-analysis of randomised controlled trials	<i>n</i> = 108, 62 males, 46 females, mean age 42.84 ± 13.61 years, moderate-to-severe plaque psoriasis N/A	N/A	13 RCTs with 625 participants were identified, 3 RCTs involving 337 participants provided usable data for meta-analysis. Fish oil supplement did not significantly reduce the severity of psoriasis when assessed by psoriasis area and severity index score compared with control groups. The current evidence does not support the use of fish oil supplement in treating psoriasis.
Upala <i>et al.</i> (2017) <sup>(94)</sup>	Systematic review	N/A	N/A	12 studies were included, findings are inconclusive on whether use of <i>n</i> -3 PUFA in patients with psoriasis is associated with improvements in severity of symptoms.
Chen <i>et al.</i> (2020) <sup>(95)</sup>	Systematic review	N/A	N/A	18 RCTs found monotherapy with fish oil <i>n</i> -3 PUFA had no effect on PASI score. Fish oil <i>n</i> -3 PUFA combined with conventional treatments resulted in a decreased PASI score.
Clark <i>et al.</i> (2019) <sup>(96)</sup>	Meta-analysis of randomised controlled trials	N/A	N/A	10 studies involving 560 participants were included in the meta-analysis. The meta-analysis indicated a significant reduction in PASI score in favour of <i>n</i> -3 PUFA group. The random effects model showed a statistically significant beneficial effect of <i>n</i> -3 PUFA supplementation on reducing erythema and scaling. Significant improvements in erythema, itching and scale were observed in the trials which used the higher dosage of <i>n</i> -3 supplementation. Larger controlled and randomised studies are needed to confirm the findings.
Tveit <i>et al.</i> (2020) <sup>(97)</sup>	RCT  Double-blind, placebo-controlled clinical study	PLwP  <i>n</i> = 32, 15 females, 17 males, mean age 47 years, mean PASI 6, BMI 29.54 kg/m <sup>2</sup>	PLwP  <i>n</i> = 32, age-, sex-, PASI-, BMI-matched	A statistically significant improvement in the mean PASI score in HRO supplementation group compared with placebo group at 26 weeks. No significant differences were observed at earlier visits (weeks 6, 12 and 18).

Diet in the management of psoriasis

**Table 4.** (Continued)

Authors, year, reference	Study design	Population characteristics	Control group	Findings
	Dietary supplement containing HRO (3:1 DHA-to-EPA ratio) 292 mg PUFA, total daily does was 2.6 g EPA/DHA and 5.9 g lipid or placebo for 26 weeks. Participants were instructed to stop supplements of cod liver oil, <i>n</i> -3 PUFA and choline for 4 weeks prior to study. PASI was used to assess psoriasis severity			
<i>Vitamin D</i> Theodoridis <i>et al.</i> (2021) <sup>(104)</sup>	Systematic review and meta-analysis of efficacy of oral vitamin D supplementation in lessening disease severity of patients with psoriasis	N/A	N/A	4 studies were included in the analysis. A favourable effect of oral vitamin D supplementation in patients with psoriasis could not be verified. More randomised controlled trials with larger sample sizes are needed to produce robust results.
Stanescu <i>et al.</i> (2022) <sup>(103)</sup>	Review oral vitamin D therapy in patients with psoriasis	N/A	N/A	Findings suggest that more large-scale studies are needed to determine the efficacy, optimal dose and adverse effects of vitamin D administration in patients with psoriasis.
Finamor <i>et al.</i> (2013) <sup>(107)</sup>	Open-label intervention study Vitamin D3 35 000 IU/d for 6 months alongside a low-calcium diet (avoiding dairy products and calcium-enriched foods such as oat, rice or soya 'milk') and hydration (minimum 2.5 litres daily)	PLwP <i>n</i> = 9, all patients presented low vitamin D status (serum 25(OH) D3 ≤ 30 ng/mL) at baseline	N/A	At 6 months 25(OH)D3 levels significantly increased. PASI score significantly improved in all 9 participants.
Al-Sultany <i>et al.</i> (2020) <sup>(108)</sup>	Comparative therapeutic study	PLwP	PLwP	At 3 months a significant increase in serum vitamin D levels and significant improvement in PASI score was seen in participants in the vitamin D group compared with control group ( <i>p</i> = 0.033).
	Oral vitamin D supplement of 50 000 IU/week for 3 months alongside topical potent corticosteroid (clobetasol propionate) compared with control of just topical potent corticosteroid (clobetasol propionate) PASI was used to assess psoriasis severity and vitamin D serum level	<i>n</i> = 38, 13 females, 25 males, mean age 34.63 years old, mean BMI 26.82 ± 5.4 kg/m <sup>2</sup> , moderate-to-severe plaque-psoriasis (PASI). Oral vitamin D (50 000 IU weekly dose for 3 months)	<i>n</i> = 38, age-, BMI-, PASI score-, baseline vitamin D level-matched. Usual treatment of topical potent corticosteroid (clobetasol propionate)	
Disphanurat <i>et al.</i> (2019) <sup>(106)</sup>	RCT	PLwP	PLwP	At 3 months, the oral vitamin D2 group had significantly higher PASI improvement than the placebo group ( <i>p</i> = 0.034). The mean serum 25(OH)D level was significantly higher in the oral vitamin D group than in the placebo group ( <i>p</i> = 0.029). At 6 months serum 25(OH)D concentrations were significantly inversely correlated with PASI scores.
	A double-blind, placebo-controlled study  Oral vitamin D2 supplementation 60 000 IU/2 weeks or placebo for 6 months	<i>n</i> = 23, 13 females, 11 males, mean age 52.39 years, mean PASI 4.68, mean BMI 26.3 kg/m <sup>2</sup>	<i>n</i> = 22, age-, sex-, PASI-, BMI-matched	



**Table 4.** (Continued)

Authors, year, reference	Study design	Population characteristics	Control group	Findings
Jarret <i>et al.</i> (2017) <sup>(109)</sup>	RCT	PLwP	PLwP	At 12 months, no significant difference in psoriasis severity (PASI) was observed between vitamin D group and control group.
	Double-blind, placebo-controlled, vitamin D3 supplementation of 100 000 IU/month for 12 months	<i>n</i> = 23, 50–84 years	<i>n</i> = 42, age-, PASI-, serum vitamin D level-matched	
Ingram <i>et al.</i> (2018) <sup>(110)</sup>	RCT	PLwP	PLwP	PASI did not differ between groups at any time. However, 25(OH)D increased in both groups, rendering these findings inconclusive
	Double-blind, placebo-controlled, vitamin D3 supplementation of 100 000 IU/month for 12 months	<i>n</i> = 67	<i>n</i> = 34, age-, PASI-, serum vitamin D level-matched	
Mahtani <i>et al.</i> (2022) <sup>(111)</sup>	Case series	PLwP	N/A	Complete control of psoriasis in each participant over a period of 2–6 months.
	Oral vitamin D3 supplements 30 000 IU/d for 2–6 months. Those with severe vitamin D deficiency were given a one-time loading dose of 600 000 IU vitamin D followed by 30 000 IU vitamin D3/d. PASI was used to assess psoriasis severity	<i>n</i> = 6, 5 females, 1 male, aged 37–63 years, severe psoriasis		
<i>B vitamins</i>				
Unpublished				
<i>Selenium</i>				
Serwin <i>et al.</i> (2003) <sup>(116)</sup>	Placebo-controlled trial	PLwP	PLwP	No significant difference in PASI at 4 weeks.
	200 µg of selenium daily as selenomethionine or placebo alongside topical treatment with 5% salicylic acid ointment, 0.1–0.3% dithranol ointment for 4 weeks	<i>n</i> = 11	<i>n</i> = 12	
Serwin <i>et al.</i> (2006) <sup>(117)</sup>	Placebo-controlled trial	PLwP	PLwP	No significant difference in PASI at any timepoint.
	200 µg of selenium daily as selenomethionine or placebo alongside narrowband ultraviolet B therapy and for 4 weeks. Assessment at baseline, 2 weeks, 4 weeks and 4 weeks post-study	<i>n</i> = 19	<i>n</i> = 18	
Kharaeva <i>et al.</i> (2009) <sup>(118)</sup>	A double-blind placebo-controlled clinical study	PLwP	PLwP	Supplementation resulted in significant improvement of PASI score in supplementation group compared with control.
	Oral supplementation with coenzyme Q <sub>10</sub> (ubiquinone acetate, 50 mg/d), vitamin E (natural α-tocopherol, 50 mg/d) and selenium (aspartate salt, 48 µg/d) dissolved in soy lecithin or a placebo of soy lecithin for 30–35 d	<i>n</i> = 14, 6 females, 8 males, mean age 36.2 years, severe erythrodermic psoriasis	<i>n</i> = 14, sex-, age-matched, severe erythrodermic psoriasis	
<i>Probiotics</i>				
Zeng <i>et al.</i> (2021) <sup>(123)</sup>	A systematic review and meta-analysis of randomised controlled trials and pre-clinical trials	N/A	N/A	2 studies found. Probiotics can improve PASI, but more studies are required.

**Table 4.** (Continued)

Authors, year, reference	Study design	Population characteristics	Control group	Findings
Atabati <i>et al.</i> (2020) <sup>(126)</sup>	Review	N/A	N/A	1 study on probiotic supplementation and psoriasis severity identified. Probiotics may have ameliorating effects on psoriatic skin. Larger controlled studies are needed.
Lin <i>et al.</i> (2021) <sup>(127)</sup>	Single-arm, open-label preliminary clinical trial 12-Week supplementation of oral probiotics <i>Bacteroides fragilis</i> BF839, compared with baseline at 4 and 12 weeks	PLwP  <i>n</i> = 27, 18 males and 9 females, aged between 22 and 67 years, mean PASI 9.1 ± 5.9, mild-to-severe psoriasis vulgaris	N/A	Mean PASI at 12 weeks significantly lower ( <i>p</i> < 0.01) compared with baseline.
Navarro-Lopez <i>et al.</i> (2019) <sup>(124)</sup>	RCT  Double-blind, placebo-controlled trial 12-Week supplementation of 3 probiotic strains in 1:1:1 ratio or placebo freeze-dried powder with maltodextrin. PASI and PGA were evaluated at baseline, 2 weeks, 6 weeks and 12 weeks	PLwP  <i>n</i> = 46, aged 18–70 years, plaque psoriasis, mild-to-moderate psoriasis	PLwP  <i>n</i> = 44, plaque psoriasis, mild-to-moderate psoriasis	At 12 weeks, 66.7% of patients in the probiotic group and 41.9% in the placebo group showed a reduction in psoriasis area and severity index of up to 75% ( <i>p</i> < 0.05). At 6 months, a lower risk of psoriasis relapse after the intake of the probiotic mixture was seen compared with control.
Moludi <i>et al.</i> (2021) <sup>(125)</sup>	RCT  Double-blind, placebo-controlled trial 2 × probiotic oral capsule 3 <i>Lactobacillus</i> strains; multi- <i>Lactobacillus acidophilus</i> , <i>Bifidobacterium bifidum</i> , <i>Bifidobacterium lactis</i> and <i>Bifidobacterium langum</i> with 1.8109 colony-forming units (CFU) a day for 8 weeks or placebo (maltodextrin) for 8 weeks. PASI, PSS and DQLI were measured at baseline	PLwP  <i>n</i> = 25, 18–50 years old, 60% female, mean PASI 10.65	PLwP  <i>n</i> = 25, age-, sex-, PASI-matched	At 8 weeks the probiotic group had a significantly reduced mean PASI score compared with the control group (10.65 ± 5.12 to 5.39 ± 2.73) ( <i>p</i> = 0.049) and a reduced mean DLQI ( <i>p</i> = 0.045) and PSS ( <i>p</i> = 0.047) score.
Vijayashankar & Raghunath (2012) <sup>(129)</sup>	Case study  <i>Lactobacillus sporogenes</i> with biotin 10 mg 3× per day	PLwP  1 female, 47 years, severe pustular psoriasis	N/A	After 2 weeks of supplementation, psoriasis improved. At 6-month follow-up (following same treatment) the patient is free of psoriatic lesions.





Table 4. (Continued)

Authors, year, reference	Study design	Population characteristics	Control group	Findings
Moludi <i>et al.</i> (2022) <sup>(128)</sup>	RCT  Double-blind placebo-controlled clinical trial  8 weeks of probiotic oral capsules containing multi-strain ( <i>Lactobacillus acidophilus</i> , <i>Bifidobacterium bifidum</i> , <i>Bifidobacterium lactis</i> and <i>Bifidobacterium langum</i> ) 1.6× 10 <sup>9</sup> CFU/g bacteria or placebo. PASI used to assess severity	PLwP  <i>n</i> = 23, mild-to-severe psoriasis	PLwP  <i>N</i> = 23, mild-to-severe psoriasis, age-, sex-, BMI- and PASI-matched	At 8 weeks the probiotics group had significantly improved PASI score and quality of life.
<i>Alternative dietary approaches</i> Gamret <i>et al.</i> (2018) <sup>(132)</sup>	Systematic review	N/A	N/A	Treatment with oral curcumin examined in 3 studies conferred statistically and clinically significant improvements in psoriasis plaques. Larger controlled studies are needed to confirm these findings.
Antiga <i>et al.</i> (2015) <sup>(131)</sup>	RCT  Double-blind  Meriva, a lecithin-based delivery system of curcumin at 2 g/d with usual topical steroid treatment, or with topical steroids alone for 12 weeks	PLwP  <i>n</i> = 31, 17 females, 14 males, aged 19–62 years, mild-to-moderate psoriasis vulgaris (PASI <10)	PLwP  <i>n</i> = 32, sex-, age- and PASI-matched	A significant decrease in PASI participants treated with both topical steroids and oral curcumin compared with participants treated only with topical steroids.
Ahmed <i>et al.</i> (2014) <sup>(134)</sup>	Open-label trial  Supplementation of crude NS powder (500 mg capsule three times per day) for 12 weeks compared with baseline	PLwP  <i>n</i> = 20	N/A	At 12 weeks significant improvement in mean PASI score compared with baseline.
Greenberger <i>et al.</i> (2012) <sup>(135)</sup>	Prospective, randomised, double-blinded pilot study  Oral alga <i>Dunaliella bardawil</i> or placebo of starch powder capsules taken daily for 12 weeks. PASI was used to assess psoriasis severity	PLwP  <i>n</i> = 17, 6 females, 11 males, mean age 52 years, mean BMI 27 kg/m <sup>2</sup>	PLwP  <i>n</i> = 11, 3 females, 8 males, age-, BMI-matched	At 6 weeks the reduction in the mean PASI score was significantly higher in the alga <i>Dunaliella bardawil</i> group than in the placebo group (61.3% versus 34%, respectively, <i>p</i> = 0.002).
Barrea <i>et al.</i> (2018) <sup>(136)</sup>	Cross-sectional case–control observational study  Coffee consumption was collected using a 7-d food diary record; PASI was used to assess psoriasis severity	PLwP  <i>n</i> = 221, treatment-naive	N/A	Coffee consumers have a lower PASI score versus non-consumers ( <i>p</i> < 0.001). The lowest PASI score were seen in participants consuming 3 cups of coffee per day ( <i>p</i> < 0.001), which was also the most common daily serving (34.8%), whereas the highest PASI score was found among those drinking ≥4 cups per day.

Table 4. (Continued)

Authors, year, reference	Study design	Population characteristics	Control group	Findings
Kurd <i>et al.</i> (2008) <sup>(139)</sup>	Single-arm, non-controlled, open-label clinical trial	PLWP	N/A	At 12 weeks a significant decrease in PASI from baseline ( $p = 0.04$ )
Kim <i>et al.</i> (2013) <sup>(130)</sup>	Oral curcumin 4.5 g/d Cross-sectional	$n = 12$ PLWP	N/A	17.5% used health supplements as CAM to help psoriasis. Health supplements were reported by 21.2% to be helpful for psoriasis; the most popular health supplements taken were: Aloe 15 (17%), Chlorella 12 (13.6%) and green tea (13.6%).
	Questionnaire on complementary and alternative supplementation used in PLWP	$n = 189$ , 70 females, 119 males, mean age 42 years		

PLWP, people living with psoriasis; RCT, randomised control trial; BMI, body mass index; PASI, psoriasis area and severity index; DLQI, dermatology life quality index; LCD, low calorie diet; n-3 PUFA, omega-3 polyunsaturated fatty acid; VLCKD, very low-calorie ketogenic diet; MD, Mediterranean diet; EVOO, extra-virgin olive oil; BSA, body surface area; PGA, physician global assessment; GFD, gluten-free diet; IgA, immunoglobulin A; IgG, immunoglobulin G; anti-glucan antibodies; IF, intermittent fasting; HRO, herring roe oil; DHA, docosahexaenoic acid; EPA, eicosapentaenoic acid; IU, international units; PSS, psoriasis severity scale; CAM, complementary and alternate methods; NS, *Nigella sativa*.

identified in this review. Three studies were identified that had explored the effects of a VLCKD on psoriasis severity<sup>(72–74)</sup>.

A single-arm open-label trial ( $n = 37$ ) found that weight loss following a VLCKD (<500 kcal/d; 1.2 g of protein/kg of ideal body weight/d) for 4 weeks followed by a balanced LCD (25–30 kcal/kg of ideal body weight per day) for 6 weeks significantly improved in psoriasis area and severity index (PASI) and itch severity<sup>(73)</sup> in drug-naive adults with an overweight BMI and stable plaque psoriasis. Castaldo *et al.* (2021) explored the effect of a 4-week VLCKD of <500 kcal/d, providing 10–20 g of carbohydrates (from vegetables, 400–500 g/d), 20–30 g of lipids, and 1.4 g per kg of ideal body weight of protein per day, on the psoriasis severity of participants ( $n = 30$ ) with overweight or obesity<sup>(74)</sup>. After 4 weeks there was a significant improvement in PASI, itch severity and dermatology life quality index (DLQI) ( $p \leq 0.05$ ). However, no significant difference in weight loss compared with baseline was reported at 4 weeks<sup>(74)</sup>. One case study of a female with severe psoriasis and obesity following a VLCKD<sup>(72)</sup> was also identified. Following a psoriasis relapse after treatment, the patient was put on a VLCKD of a protein-based enteral nutrition liquid of approximately 300 kcal/d, containing a protein content of 1.2 g/kg of ideal body weight, for 4 weeks. Compared with baseline, the patient lost 11 kg, and a significant reduction in psoriasis severity was observed (>80% PASI) after 4 weeks.

**Mediterranean diet (MD).** The Mediterranean diet (MD) is typically high in fruits and vegetables, legumes, whole grains, fish, nuts and monounsaturated fatty acids (MUFA) such as extra-virgin olive oil (EVOO), with a moderate intake of meat, dairy and alcohol<sup>(75)</sup>. Four studies explored MD in the management of psoriasis<sup>(75–78)</sup>. These were all cross-sectional studies that assessed the association between a score reflecting adherence to the MD and psoriasis severity. The higher the score, the higher the adherence to a MD. No randomised control trials (RCTs) were found to have been conducted on MD and psoriasis severity. Three of the studies also compared MD adherence of PLWP compared with controls<sup>(57,75,76)</sup>.

Controls presented a significantly higher adherence to a Mediterranean diet compared with PLWP in all three case-control studies identified<sup>(75–77)</sup>. Barrea *et al.*<sup>(77)</sup> found that psoriasis participants exhibited statistically significant differences compared with controls, in the consumption of certain individual MD dietary components. Controls consumed significantly more EVOO, fruit, fish and nuts and significantly less red meat than those with psoriasis<sup>(77)</sup>.

Regarding psoriasis severity and MD adherence, those with less severe psoriasis had a higher adherence to a Mediterranean diet in all four studies. Barrea *et al.*<sup>(77)</sup> used PREDIMED score to assess MD adherence in people with mild to severe psoriasis ( $n = 62$ ). The study concluded that that PREDIMED score was a major predictor of psoriasis severity determined by PASI ( $p = 0.007$ ). Individual MD components were also shown to have an independent predictive value for PASI score, higher consumptions of EVOO ( $p < 0.001$ ) and fish ( $p = 0.005$ ) were significantly associated with lower psoriasis severity scores<sup>(77)</sup>. A summary of individual foods associated with higher or lower psoriasis severity is presented in Table 5.



**Table 5.** Summary of individual foods included in the studies identified that were associated with lower or higher psoriasis severity.

Authors, year, reference	Study design	Population characteristics	Findings
<b>Associated with lower psoriasis severity</b>			
<i>Extra-Virgin Olive Oil (EVOO)</i>			
Barrea <i>et al.</i> (2015) <sup>(57)</sup>	Cross-sectional	PLwP	Individual MD component: higher consumption of EVOO was associated with lower psoriasis severity ( $p < 0.001$ ).
	PREDIMED 14-item questionnaire, was used to assess adherence to the Mediterranean diet, PASI used to assess psoriasis severity	$n = 61$ patients, 49 males and 13 females, mean age: 50.2 years, mild-to-severe psoriasis	
Korovesi <i>et al.</i> (2019) <sup>(76)</sup>	Cross-sectional	PLwP	Individual MD component: EVOO was inversely associated with psoriasis severity ( $p < 0.05$ ).
	MedDietScore was used to assess adherence to Mediterranean diet. PASI and DLQI was used to assess severity of psoriasis	$n = 69$ , 35 men, 34 females, treatment naive, mean age 47.7 years, mean BMI of 28.9 kg/m <sup>2</sup> , moderate-to-severe psoriasis, mean DLQI of 9.5	
<i>Fish (non-specific)</i>			
Barrea <i>et al.</i> (2015) <sup>(57)</sup>	Cross-sectional	PLwP	Individual MD component: higher fish consumption was associated with lower psoriasis severity ( $p = 0.005$ ).
	PREDIMED 14-item questionnaire, was used to assess adherence to the Mediterranean diet, PASI used to assess psoriasis severity	$n = 61$ patients, 49 males and 13 females, mean age: 50.2 years, mild-to-severe psoriasis	
Korovesi <i>et al.</i> (2019) <sup>(76)</sup>	Cross-sectional	PLwP	Individual MD component: fish was inversely associated with psoriasis severity ( $p < 0.05$ ).
	MedDietScore was used to assess adherence to Mediterranean diet. PASI and DLQI was used to assess severity of psoriasis	$n = 69$ , 35 men, 34 females, treatment naive, mean age 47.7 years, mean BMI of 28.9 kg/m <sup>2</sup> , moderate-to-severe psoriasis, mean DLQI of 9.5	
<i>Legumes (non-specific)</i>			
Korovesi <i>et al.</i> (2019) <sup>(76)</sup>	Cross-sectional	PLwP	Individual MD component: legume consumption was associated with reduced psoriasis severity ( $p < 0.05$ ).
	MedDietScore was used to assess adherence to Mediterranean diet. PASI and DLQI was used to assess severity of psoriasis	$n = 69$ , 35 men, 34 females, treatment naive, mean age 47.7 years, mean BMI of 28.9 kg/m <sup>2</sup> , moderate-to-severe psoriasis, mean DLQI of 9.5	
<i>Vegetables (non-specific)</i>			
Barrea <i>et al.</i> (2015) <sup>(77)</sup>	Cross-sectional	PLwP	Individual MD component: vegetables $\geq 2$ servings per day negatively correlated with PASI ( $p < 0.001$ ).
	PREDIMED 14-item questionnaire, was used to assess adherence to the Mediterranean diet; PASI used to assess psoriasis severity	$n = 61$ patients, 49 males and 13 females, mean age: 50.2 years, mild-to-severe psoriasis	
Ingkapiroj <i>et al.</i> (2022) <sup>(53)</sup>	Cross-sectional	PLwP	Frequently consuming vegetables ( $\geq 3$ times per week) was associated with lower psoriasis severity ( $p = 0.02$ ).
	FFQ used for dietary intake assessment; PASI used to assess psoriasis severity	$n = 100$ , 47 males, 53 females; mean age 45.87 years, mild-to-severe psoriasis severity	

**Table 5.** (Continued)

Authors, year, reference	Study design	Population characteristics	Findings
<i>Fruits (non-specific)</i> Barrea <i>et al.</i> (2015) <sup>(77)</sup>	Cross-sectional  PREDIMED 14-item questionnaire was used to assess adherence to the Mediterranean diet; PASI used to assess psoriasis severity	PLwP  <i>n</i> = 61 patients, 49 males and 13 females, mean age: 50.2 years, mild-to-severe psoriasis	Individual MD component: Fruits $\geq 3$ servings per day negatively correlated with PASI ( $p < 0.001$ ).
Associated with higher psoriasis severity			
<i>Red meat</i> Ingkapiroj <i>et al.</i> (2022) <sup>(53)</sup>	Cross-sectional  FFQ used for dietary intake assessment; PASI used to assess psoriasis severity	PLwP  <i>n</i> = 100, 47 males, 53 females; mean age 45.87 years, mild-to-severe psoriasis severity	Frequently consuming red meat ( $\geq 3$ times per week) was associated with higher psoriasis severity ( $p = 0.01$ ).
<i>Dairy (non-specific)</i> Korovesi <i>et al.</i> (2019) <sup>(76)</sup>	Cross-sectional  MedDietScore was used to assess adherence to Mediterranean diet. PASI and DLQI was used to assess severity of psoriasis	PLwP  <i>n</i> = 69, 35 men, 34 females, treatment naive, mean age 47.7 years, mean BMI of 28.9 kg/m <sup>2</sup> , moderate-to-severe psoriasis, mean DLQI of 9.5	PASI positively correlated with dairy products ( $P = 0.002$ ).
<i>Confection (sugary sweet foods)</i> Yamashita <i>et al.</i> (2019) <sup>(56)</sup>	Cross-sectional  Self-administered diet history questionnaire (BDHQ), based on Japanese diet used to assess dietary intake (FFQ), PASI used to assess psoriasis severity	PLwP  <i>n</i> = 70, 46 males, 24 females	In the psoriasis group, those with a higher psoriasis severity (PASI) consumed a significantly higher amount of confection ( $p = 0.03$ ).

PLwP, people living with psoriasis; BMI, body mass index, PASI, psoriasis area and severity index; DLQI, dermatology life quality index; VAS, visual analogue scale; MD, Mediterranean diet; EVOO, extra-virgin olive oil; BSA, body surface area; FFQ, food frequency questionnaire.

A large national-cross sectional study in PLwP in France ( $n = 3557$ ) found that a higher percentage of participants with severe psoriasis had a MEDI-LITE score of 0–7 (low adherence to the Mediterranean diet) compared with those without severe psoriasis. Mediterranean diet score was also found to be negatively correlated with PASI ( $p = 0.001$ )<sup>(75)</sup>. In a smaller study ( $n = 69$ ) using MedDietScore to assess MD adherence in PLwP, MedDietScore was a significant negative predictor of PASI ( $p = 0.02$ ) adjusting for age, gender and BMI. Higher consumption of legumes, fish and EVOO ( $p < 0.05$ ) were found to be associated with lower PASI scores, whereas higher dairy product consumption was positively correlated with psoriasis severity ( $p = 0.002$ )<sup>(76)</sup>. The severity of psoriasis was lower in participants with greater adherence to the Mediterranean diet assessed using PASI ( $p = 0.007$ ), body surface area (BSA) ( $p = 0.009$ ) and practitioner global assessment (PGA) ( $p = 0.01$ ) in a further cross-sectional study on PLwP using PREDIMED questionnaire to assess adherence to the Mediterranean diet<sup>(78)</sup>.

**Gluten-free diet (GFD).** A gluten-free diet (GFD) eliminates gluten, a protein found in wheat, barley and rye. Psoriasis is associated with an increased risk of coeliac disease, compared with the general population<sup>(79,80)</sup>. Coeliac disease is a chronic condition affecting the small intestine, which is activated by the consumption of gluten. Studies suggest that psoriasis and coeliac disease share common genetic and inflammatory pathways<sup>(79)</sup>. Gluten-specific serum antibody levels followed by a biopsy is used to diagnose coeliac disease. In those without a coeliac disease diagnosis, gluten-specific antibodies are higher in PLwP compared with controls. However, whether there is an association between higher antibody levels and greater psoriasis severity is unclear<sup>(79)</sup>.

Two systematic reviews on diet and psoriasis found that a GFD may be beneficial in reducing psoriasis severity in those with coeliac disease or gluten-specific antibodies<sup>(64,79)</sup>. From the findings of their review, Bhatia *et al.* recommended that healthcare professionals (HCPs) screen patients with psoriasis for symptoms of gluten sensitivity, followed by gluten-specific antibody tests<sup>(79)</sup>. Those with positive antibody tests should then be advised to trial a GFD for symptom management<sup>(79)</sup>. However, there was no suggestion on the length of GFD trial.

Several studies have shown the beneficial impact of following a GFD on psoriasis severity in participants with coeliac disease or gluten-specific antibodies. A study on psoriasis patients ( $n = 39$ ) with elevated gluten-specific antibodies showed a significant decrease in mean PASI score after 3 months on a GFD compared with a control group<sup>(81)</sup>. Those with moderate-to-severe psoriasis showed an even greater PASI reduction than those with mild psoriasis. The control group consisted of PLwP but without coeliac disease or gluten-specific antibodies, who also followed a GFD. In this group there was no change in disease severity, and in two participants there was a substantial worsening of psoriasis severity.

A further study by Ref. <sup>(82)</sup>, found that PLwP who had high levels of gluten-specific antibodies ( $n = 8$ ) (IgA against gliadin peptides) saw a 36% improvement in PASI score following a GFD for 1 year. Those with higher levels of gluten-specific antibodies ( $n = 5$ ) saw an even greater improvement, 56%

reduction in PASI, following a GFD for 1 year. A GFD also significantly improved psoriasis symptoms in nine patients with coeliac disease compared with baseline at 3 months and was maintained at 6 months<sup>(83)</sup>. Complete clearance of psoriatic skin symptoms following a GFD for 1 month has also been reported in individual case studies<sup>(84,85)</sup>. However, these data are based on small, uncontrolled studies.

One study found no improvement in psoriasis severity after 6 months of following a GFD in three patients with coeliac disease or gluten-specific antibodies<sup>(86)</sup>. However, this was another small uncontrolled study.

**Intermittent fasting (IF).** More recently intermittent fasting has been studied in the management of psoriasis. Two studies have explored this dietary approach, using fasting during Ramadan to explore the effects on psoriasis<sup>(87,88)</sup>.

Almutairi and Shaaban (2022) assessed the effects of Ramadan fasting on psoriasis severity of 121 people with stable chronic plaque psoriasis in Kuwait<sup>(87)</sup>. Participants followed traditional Ramadan fasting for 1 month, which consists of refraining from eating, drinking or smoking during daylight hours. Participants consumed two main meals a day, one before sunrise and one after. At 1 month, no participant recorded any weight loss, but mean PASI was significantly reduced compared with baseline<sup>(87)</sup>. A further study<sup>(88)</sup> also investigated the impact of Ramadan fasting on psoriasis severity in participants with moderate-to-severe psoriasis ( $n = 108$ ). Following the month of fasting, a significant reduction in mean PASI score was observed compared with baseline<sup>(88)</sup>.

One pilot study exploring the effects of modified intermittent fasting, the 5:2 diet (consuming normally for 5 d and restricting calorie intake on 2 non-consecutive days) on psoriasis severity was also found<sup>(89)</sup>. Preliminary study findings presented at the European Academy of Dermatology and Venerology Spring Symposium, show a significant reduction in scaling and thickness in patients with mild psoriasis after following a 5:2 diet<sup>(89)</sup>.

**Other diets.** Vegetarian, vegan and plant-based diets have been discussed in the literature as diets with potential to help alleviate psoriasis symptoms<sup>(90,91)</sup>. However, this is based on the assumptions that following these diets would result in increased consumption of fruits, vegetables and antioxidants and the reduced consumption of saturated fats<sup>(90)</sup>. Whilst cross-sectional studies have shown that following a MD, which is characterised by high fruit and vegetable consumption and low saturated fat intake, could help lessen psoriasis severity<sup>(75,77)</sup>, this is not the same as following a vegan or vegetarian diet. Following a vegan, vegetarian and plant-based diet does not always result in increased fruit and vegetable consumption. So far, no studies have been undertaken explicitly exploring vegetarian, vegan or plant-based diets in the management of psoriasis.

## 2. Supplementation in the management of psoriasis

Several supplements have been studied in the management of psoriasis: omega-3 polyunsaturated fatty acids (PUFA), vitamin D, selenium, B vitamins and probiotics.



**Omega-3 polyunsaturated fatty acids (PUFA).** Several recent systematic reviews and meta-analysis have been conducted to evaluate the effects of omega-3 PUFA supplementation on psoriasis severity, with conflicting results. Most studies gave omega-3 PUFA as fish oil supplements.

A systematic review based on thirteen randomised control trials (RCTs) and a meta-analysis of three RCTs found that fish oil supplementation did not significantly reduce the severity of psoriasis assessed by psoriasis area and severity index (PASI) compared with controls, concluding that the current evidence does not support the use of fish oil supplement in treating psoriasis<sup>(92)</sup>. This was in line with a previous systematic review<sup>(93)</sup>.

Another systematic review found that supplementation with fish oil omega-3 PUFA alone had no effect on PASI score. However, when combined with traditional psoriasis treatments, a significant reduction in PASI score was observed compared with controls<sup>(94)</sup>.

In contrast, a recent 2019 meta-analysis found that supplementation of omega-3 PUFA did significantly reduce PASI score. Significant improvements in specific psoriasis skin symptoms, erythema, itching and scale, were observed in trials which used higher doses of omega-3 PUFA supplementation (>1800 mg/d)<sup>(95)</sup>. The positive effects of high doses of omega-3 on psoriasis symptoms were in line with a recent study on the effect of herring roe oil (HRO) on psoriasis severity<sup>(96)</sup>. A significant improvement in mean PASI score with HRO supplementation of 2600 mg eicosapentaenoic (EPA)/docosahexaenoic (DHA) per day, was observed compared with placebo treatment, at week 26. The authors of this study theorised that the beneficial effects of HRO were due to its EPA and DHA acids ratio of 3:1, compared with omega-3 PUFA from fish oils, which is typically 1:1.

Omega-3 PUFA supplementation has been shown to have beneficial effects on the co-morbidities associated with psoriasis<sup>(97)</sup>.

**Vitamin D.** Topical vitamin D is a widely used treatment for plaque psoriasis<sup>(2)</sup>. Lower levels of serum vitamin D have been reported in psoriatic patients compared with controls<sup>(98,99)</sup>. A small, but significant, inverse correlation between serum 25(OH) D and the severity of psoriasis has also been reported<sup>(100,101)</sup>, hence the interest in oral vitamin D and psoriasis management.

So far, studies have shown mixed results on the effectiveness of oral vitamin D supplementation in the management of psoriasis. Systematic reviews have found no clear evidence to support vitamin D supplementation in the management of psoriasis symptoms<sup>(43,64,102)</sup>. A recent meta-analysis found that a favourable effect of oral vitamin D supplementation in patients with psoriasis could not be verified<sup>(103)</sup>. However, more RCTs are required to confirm these conclusions. There is evidence indicating that vitamin D supplements for the treatment of psoriasis should not be prescribed in participants with normal serum levels of vitamin D<sup>(104)</sup>. It is unclear from the literature whether those with deficient or insufficient vitamin D levels have an improved skin response compared with those with optimal levels.

An RCT assessing the effect of oral vitamin D2 on psoriasis severity found that D2 supplementation significantly increased the serum vitamin D level and significantly improved PASI scores

in patients with psoriasis compared with the placebo group at 3 months. There was no significant difference in baseline serum 25(OH)D vitamin D between groups, and some vitamin D insufficiency was seen in both groups<sup>(105)</sup>. In a study that gave high doses of oral vitamin D3 (35 000 IU/d) to PLwP ( $n = 9$ ) and low vitamin D status ( $\leq 30$  ng/mL), significant improvements in psoriasis severity were observed at 6 months compared with baseline<sup>(106)</sup>. However, this was a small, uncontrolled study and participants were also required to follow a low-calcium diet (excluding dairy) over the course of the study. A further study also found a significant improvement in PASI score in participants given oral vitamin D supplement of 50 000 IU per week for 3 months alongside usual treatment compared with the control group who just received usual treatment<sup>(107)</sup>.

However, several RCTs have shown no beneficial effect of oral vitamin D supplementation on psoriasis severity. No significant difference was found in people with mild psoriasis over 12 months of vitamin D3 supplementation, or in those with plaque or moderate-to-severe psoriasis over 3 months compared with controls<sup>(108,109)</sup>.

A recent series of case studies showed complete control of psoriasis with a high daily dose of 30 000 IU of vitamin D3 over a period of 2–6 months. Only two participants presented with severe vitamin D deficiency and were given a one-off loading dose of 600 000 IU vitamin D, all others had optimal levels<sup>(110)</sup>. Other uncontrolled studies have also indicated that oral vitamin D supplementation for  $\geq 6$  months can significantly improve PASI score<sup>(103)</sup>.

Epidemiological studies have demonstrated a strong association between vitamin D insufficiency and risk of several psoriasis-associated comorbidities, including cardiovascular disease (CVD) and metabolic syndrome<sup>(111)</sup>.

**B vitamins.** Vitamin B12 deficiency has been associated with psoriasis<sup>(112)</sup>. However, studies so far have focused on intramuscular doses of vitamin B12 and have been shown to be ineffective. Two systematic reviews on dietary approaches to psoriasis did not recommend vitamin B12 supplementation in the management of psoriasis due to the lack of studies<sup>(43,64)</sup>. Vitamin B12 is an important cofactor in the metabolism of homocysteine, elevated levels of which have been associated with increased risk of CVD<sup>(113)</sup>. This review also found one ongoing RCT on the effect of high doses of vitamin B2 (riboflavin) on psoriasis severity that is yet to be published<sup>(114)</sup>.

**Selenium.** Reviews have not found any significant improvement in psoriasis severity with selenium supplementation<sup>(43,64,115,116)</sup>. A small number of studies have evaluated the effect of selenium supplementation on psoriasis severity<sup>(115–117)</sup>. One study found a significant beneficial effect of selenium on PASI score compared with controls. However, the supplement was combined with coenzyme Q<sub>10</sub> and vitamin E<sup>(117)</sup>.

**Probiotics.** Recent studies have drawn attention to the role that the gut microbiome plays in the pathogenesis of dermatological conditions, including psoriasis<sup>(118)</sup>. Psoriasis is associated with inflammatory bowel disease (IBD), and studies have shown that the gut microbiome is altered in psoriasis

compared with controls<sup>(119,120)</sup>. It has also been reported that patients with moderate-to-severe psoriasis have a lower gut microbial diversity than patients with mild disease<sup>(121)</sup>. As a result, probiotic supplementation has become a recent research focus in the management of psoriasis.

A systematic review on the effectiveness of probiotic supplements in psoriasis found that probiotics significantly reduced PASI scores in psoriasis compared with controls after 12 weeks of supplementation and may be an effective treatment for alleviating psoriasis symptoms. However, these findings were based on only two RCT studies that explored probiotic supplementation and PASI, and larger-scale RCTs are needed to confirm this<sup>(122)</sup>.

Several studies have shown probiotic supplementation to have a beneficial effect on psoriasis severity<sup>(123–127)</sup>. A recent RCT found that consuming a probiotic drink containing *Lactobacillus* strains for 8 weeks significantly reduced PASI and psoriasis symptom scale (PSS) scores compared with the placebo group<sup>(124)</sup>. A further double-blind placebo-controlled trial ( $n = 46$ ) found that after 8 weeks of multi-strain probiotic oral supplementation PASI and quality of life scores had significantly improved compared with the placebo group<sup>(127)</sup>. Additionally, single-arm trial ( $n = 27$ ) reported significant reduction in PASI compared with baseline at 12 weeks of probiotic supplementation<sup>(126)</sup>. One case study also reported that supplementation of a probiotic containing *Lactobacillus* strains had a strong alleviating effect on skin symptoms in a patient with pustular psoriasis after 15 d. The patient continued with probiotic supplementation, and after 6 months psoriasis severity had reduced further<sup>(128)</sup>.

### 3. Alternative dietary approaches in the management of psoriasis

This review identified several studies that explored alternative dietary approaches in the management of psoriasis. These were defined as non-traditional dietary approaches, the majority of which were small studies. One cross-sectional questionnaire was identified that explored the use of complementary alternative methods used by people living with psoriasis (PLwP). This study found that health supplements were reported by 21.2% to be helpful for psoriasis; the most popular health supplements taken were aloe (17%), chlorella (13.6%) and green tea (13.6%)<sup>(129)</sup>.

Several small studies have been conducted on oral curcumin, a phytochemical found in the spice turmeric, and psoriasis severity, with mixed results<sup>(130–132)</sup>. A review of the RCTs suggested that more studies are needed on the effects of oral curcumin on psoriasis severity before any conclusions can be made<sup>(131)</sup>.

The impact of oral *Nigella sativa* (NS) on psoriasis severity in sixty participants with mild-to-moderate psoriasis was investigated in an RCT. Participants were given an oral dose of NS (500 mg three times daily) for 12 weeks; at 12 weeks psoriasis area and severity index (PASI) score had decreased from baseline<sup>(133)</sup>. However, whether this was significant or not was not clear. One RCT investigated oral capsules containing alga *Dunaliella bardawil*, a natural source of the retinoid precursor 9-*cis*  $\beta$ -carotene, in participants with mild plaque psoriasis ( $n = 34$ )<sup>(134)</sup>. Participants received capsules of the alga or a placebo, and at 6 weeks, the reduction in the mean PASI score

was significantly higher in the alga group compared with the placebo group ( $p = 0.002$ ).

The association between coffee consumption and severity of psoriasis was evaluated in a cross-sectional study of treatment naive PLwP ( $n = 221$ ). Coffee consumers were found to have a significantly lower PASI score compared with non-consumers ( $p < 0.001$ ), with the lowest PASI score seen in those consuming three cups of coffee per day, and the highest PASI score was found among those drinking four or more cups per day<sup>(135)</sup>.

### Grey literature

The relevant grey literature sources identified and included in this review included reports, guidelines and other materials produced by a range of stakeholders in psoriasis management. Most of the grey literature identified, regarding psoriasis management, provided no dietary guidance for people living with psoriasis (PLwP), and most did not mention the word diet or nutrition at all. In those that did mention diet, the vast majority focused on weight loss and dietary approaches for comorbidities associated with psoriasis. For example, the National Institute for Health and Clinical excellence (NICE) guidelines for psoriasis assessment and management mention reducing alcohol intake and losing weight, but only as modifiable risk factors for associated comorbidities<sup>(136)</sup>. Several sources did provide further information regarding diet specific to psoriasis symptom management. The National Psoriasis Foundation (NPF) in the United States conducted a systematic review on the dietary recommendations for adults with psoriasis in 2018<sup>(64)</sup>. Based on this, they recommend weight loss in PLwP with obesity or overweight as the only evidence-based dietary approach for psoriasis management on their website and suggested that a gluten-free diet (GFD) could provide relief in those with coeliac or gluten sensitivity. Several grey literature resources provided warnings about diets that claim to 'cure' psoriasis and misinformation that can be found online and included evidence-based dietary advice. However, overall, there was a lack of advice on who to go to for dietary support, the health impacts and the risks associated with following restrictive diets that claim to help psoriasis, and guidance for following a restrictive diet.

### Discussion

In this study, we reviewed the current evidence on the role of diet in the management of psoriasis. We included all types of study designs that met the inclusion criteria, as well as relevant grey literature. This has enabled us to provide a comprehensive overview of the current evidence and a unique insight into the role of diet in the management of psoriasis regarding dietary intake of people living with psoriasis (PLwP), the use and perceived effectiveness of diet of PLwP, and dietary approaches for psoriasis management. We reviewed seventy-two peer-reviewed studies as well as seventy-seven relevant grey literature resources. The principal findings suggest that diet could play a role in psoriasis management; however, most evidence comes from small heterogeneous studies. Therefore, specific psoriasis dietary guidelines and recommendations

cannot be made. The breadth of this scoping review also enabled us to map the research gaps and highlight areas for future research, to be able to better understand the role that diet plays in psoriasis management and improve dietary support for PLwP. The results of this scoping review were organised into three themes, alongside the grey literature. The discussions for each theme are presented below.

### Theme 1: dietary intakes of people living with psoriasis

The studies included in this review suggest that the dietary intakes of PLwP differ from those of controls. The studies frequently found PLwP to have higher dietary intakes of fat<sup>(52,54,57)</sup> and lower intakes of fibre<sup>(45,52,57)</sup> compared with controls. Studies also reported differences in intakes of sugar, dairy, pulses and legumes, vegetables and polyunsaturated fatty acids (PUFA) compared with controls. Furthermore, the evidence also suggests that the dietary intakes of people with less severe psoriasis differ from those with higher psoriasis severity.

High-fat diets (HFDs) have been shown to elicit low-grade systemic inflammation through elevated production of pro-inflammatory cytokines also seen in psoriasis, including interleukin (IL)-1 $\beta$ , IL-6 and tumour necrosis factor (TNF)- $\alpha$ . HFDs also play a key role in the development and progression of multiple diseases, including cardiovascular disease (CVD), type II diabetes, atherosclerosis and some cancers<sup>(137)</sup>. Murine studies have found that HFDs exacerbate the imiquimod induced psoriasiform dermatitis in mice<sup>(138,139)</sup>, and in both mice with obesity and lean mice, those fed with HFDs developed a more severe early psoriasiform skin inflammation<sup>(140)</sup>. This suggests that increased fat consumption could play a role in psoriasis symptom severity. Information on the specific fats consumed was lacking in the studies included in this review and would provide more insight into the potential mechanisms behind these dietary intakes.

Several studies also reported that PLwP had lower intakes of fibre compared with controls, and in psoriasis populations lower intakes of fibre were seen in those with more severe psoriasis compared with those with lower psoriasis severity<sup>(52,57)</sup>. Fibre has been shown to decrease levels of plasma inflammatory markers including C-reactive protein (CRP), IL-6 and TNF- $\alpha$ <sup>(141)</sup>, which play a key role in the pathophysiology of psoriasis. Dietary fibre also has a beneficial effect on the gut microbiome, and through short-chain fatty acid production produces immune and inflammatory regulation responses<sup>(141)</sup>. However, higher intakes of pulses and legumes, which are high in fibre, were reported in PLwP than in controls<sup>(45,56)</sup>. Additionally, a gluten-free diet (GFD) is associated with reduced fibre intake. Following a gluten-free diet in people with coeliac disease has been shown to improve psoriasis symptoms, and coeliac is seen more commonly in people with psoriasis compared with the general population<sup>(79)</sup>. Following a GFD is also a common dietary modification trialled by PLwP<sup>(45,62)</sup>, which could explain the difference reported in fibre intakes between PLwP and controls. Following a GFD was also frequently perceived to improve psoriasis symptoms by PLwP. However, these studies did not include information on the coeliac status of participants, and lack of information on types and sources of fibre make it difficult to compare results and

understand the potential mechanisms of action. Furthermore, fibre intake is associated with relevant health impacts and has appetite regulating and anti-obesogenic effects, and higher intakes have been associated with lower systemic inflammation<sup>(142)</sup>. Consuming adequate amounts of dietary fibre is also associated with multiple health benefits, including reduced CVD risk<sup>(143)</sup>. Therefore, understanding the fibre intake in PLwP is also important due to the associated co-morbidities.

A significantly higher consumption of vegetables was reported in those with lower psoriasis severity compared with people with higher severity<sup>(53)</sup>. Vegetables are key sources of vitamins and polyphenolic compounds which have antioxidant and anti-inflammatory properties<sup>(41,144,145)</sup>. Flavonoids and carotenoids, polyphenolic compounds commonly found in vegetables, have been shown to enhance immune pathways and inhibit certain pro-inflammatory pathways<sup>(41)</sup>. Specifically relevant to psoriasis, they have been shown to reduce pro-inflammatory cytokines IL-6 and TNF- $\alpha$ <sup>(41)</sup>, which are involved in the pathophysiology of psoriasis<sup>(31)</sup>. Vegetables are also important sources of dietary fibre<sup>(146)</sup>. However, no studies identified specific vegetables consumed, so it is difficult to suggest potential pathways. Interestingly, Afifi *et al.* found that PLwP had a higher intake of fruits and vegetables compared with controls<sup>(45)</sup>. This could be attributable to people with psoriasis following popular psoriasis dietary recommendations, which typically suggest that fruits and vegetables can improve psoriasis symptoms. This review has shown that dietary modifications among PLwP to try to manage psoriasis are common<sup>(45,62)</sup>. Dietary changes after diagnosis or to manage symptoms may also explain the contradictory findings regarding sugar, dairy and fish intakes of PLwP compared with controls. Removing or reducing dairy and sugar as well as following a vegetarian diet are recommended as dietary approaches to manage psoriasis in popular literature.

Two studies found that total polyunsaturated fatty acid (PUFA) intake was significantly higher in PLwP compared with controls<sup>(54,57)</sup>. Although, when assessed on PUFA type, Barrea *et al.* found that *n*-3 PUFA intake was significantly lower in PLwP compared with controls, and lower intakes were associated with higher severity<sup>(57)</sup>. *n*-3 PUFA are potentially potent anti-inflammatory agents<sup>(147)</sup>. However, this was a small study conducted in treatment-naive males; therefore, generalisability and comparability are limited.

Although the studies identified suggest that dietary intake is different in PLwP and controls and between those with different severities, the evidence is limited. This review identified nine studies that have been conducted in only seven countries worldwide. All the studies were cross-sectional, and most had sample sizes under 200 people. Additionally, the methodologies varied substantially between studies, which impairs the ability to compare results between studies. Most of the studies used food frequency questionnaires (FFQs) to assess dietary intakes of participants that, although useful in these types of studies, rely on self-reported information, participant memory and perceptions of portion sizes, and foods may be missed if not presented on FFQ lists. It is also difficult to focus on the effect of one dietary component, as diet is a complex combination of different nutrients<sup>(148)</sup> and multiple other lifestyle factors can impact the development and severity of psoriasis<sup>(1)</sup>. Longitudinal



population-based studies are needed to further investigate a causal role between dietary intake and psoriasis, and effects on severity in PLWP. However, the studies identified in this review give an insight into the dietary intakes of PLWP and highlight important research gaps. Furthermore, the differences in dietary intakes could also impact general health and prompt further research in PLWP, due to the associated comorbidities that could also be exacerbated by the dietary intakes highlighted here, in particular, high fat and low fibre intakes<sup>(149)</sup>.

### *Theme 2: the perceived role of diet in the management of psoriasis*

The belief that diet impacts psoriasis symptoms is common in PLWP, and many adjust their diets accordingly. However, most PLWP do not discuss diet with their healthcare professional prior to making dietary changes. This is concerning considering that most of the dietary changes tried were restrictive. This study also searched the scientific literature for evidence on the dietary approaches trialled by PLWP reported in the studies found under Theme 2. Except for the Mediterranean diet (MD) and GFD, no studies were identified that had explored the use of any of the dietary approaches reportedly followed in the management of psoriasis. This suggests that most dietary approaches tried by PLWP are unsubstantiated, self-prescribed and taken from the popular literature. Following fad diets long-term or restrictive diets without the guidance of healthcare professionals (HCPs) can result in micronutrient deficiencies (MND)<sup>(150,151)</sup>. Micronutrient deficiencies have been reported in people with irritable bowel syndrome (IBS) who self-prescribe elimination diets without consulting HCPs<sup>(150,152)</sup>. Elimination diets could also result in further health impacts. Following a GFD was a common dietary modification trialled by PLWP<sup>(45)</sup>. A GFD has been shown to be lower in dietary fibre and some essential micronutrients which have protective properties, such as cholesterol lowering and improved glycaemic control<sup>(149)</sup>, relevant to PLWP considering the associated comorbidities. Additionally, gluten-free foods are often more expensive<sup>(153)</sup>. Furthermore, restrictive diets have also been linked to reduced quality of life (QoL), disordered eating and orthorexia<sup>(152)</sup>.

The most common dietary modifications reported to improve psoriasis were reducing dairy, gluten, nightshades, alcohol and sugar. Apart from alcohol, and gluten in those with coeliac and gluten sensitivity, the mechanisms of how reducing these specific dietary components could improve psoriasis are unclear and have not been researched. Theories suggest that the potential pro-inflammatory impact of sugar consumption could be the reason behind this effect; high amounts of dietary sugars have been shown to promote T-cell-mediated inflammation<sup>(154)</sup>. Dairy is commonly demonised in popular literature as being pro-inflammatory, most likely due to the saturated fat and lactose content of certain dairy products<sup>(155)</sup>. However, a recent systematic review found that dairy products and dairy proteins have neutral to beneficial effects on biomarkers of inflammation<sup>(155)</sup>. Nightshades are plants from the Solanaceae family, which include potatoes, tomatoes, peppers and aubergines. They contain solanine and alkaloids which have been linked with inflammation<sup>(45)</sup>. However, no association between

nightshades and inflammation is supported by scientific studies in humans. Furthermore, they are high in nutrients beneficial to health.

Overall, the evidence is limited; only five studies were identified under this theme in this review<sup>(44,45,61–63)</sup>, all of which were cross-sectional surveys which relied on self-reported information and memory. Participants may have been more likely to have an interest in diet or believe that diet helps manage their psoriasis, which may have impacted results. Additionally, sample sizes were small, with only two studies with a sample size over 200, most participants were white females, and no studies included information on other factors known to affect psoriasis severity, including stress and smoking. Despite the limitations of these studies, the findings highlight important factors to consider in psoriasis care, as well as highlighting important research gaps.

Studies exploring the perceived role of diet in the management of psoriasis have been conducted in only four countries worldwide. None has been conducted in the United Kingdom, and this represents an important research gap as over 1.1 million people in the United Kingdom are estimated to be living with psoriasis. Instagram and online forums are commonly used by people with acne to seek information on nutritional suggestions to help their skin condition<sup>(156)</sup>. However, no studies identified explored the sources of dietary information of PLWP or content of recommended dietary changes. Only three of the studies provided information on specific dietary modifications made. Further understanding the dietary recommendations suggested in the popular literature and duration of diets trialled could help HCPs understand the potential impact on nutrient status and ways to support PLWP. Following restrictive diets long term can lead to micronutrient deficiencies<sup>(150,151)</sup>. Furthermore, specific symptom responses to dietary modifications have not been investigated. It was commonly reported that most PLWP do not discuss diet with an HCP prior to making dietary changes<sup>(44,45,61)</sup>. Understanding the reasons behind this will give insight into patient support needs and enable HCP to better understand how to assist PLWP. Another notable gap in the literature are studies exploring the perceptions of HCPs involved in psoriasis management on the role of diet.

### *Theme 3: dietary approaches to manage psoriasis*

The strongest evidence for dietary methods in the management of psoriasis symptoms is for low-calorie diets (LCDs) in subjects with obesity or overweight. The link between obesity and psoriasis is well recognised<sup>(3,157)</sup>. Several studies have demonstrated a relationship between increased BMI and increased psoriasis severity<sup>(46,64,65)</sup>. Excess body weight is also associated with increased incidence of psoriasis<sup>(157,158)</sup> and reduced response to psoriasis treatments<sup>(159)</sup>. The relationship is theorised to be a result of increased pro-inflammatory cytokine release due to increased adipose tissue. Weight reduction in subjects with obesity reduces adipose tissue and, consequently, inflammation<sup>(66)</sup>. Limited research has been conducted on ketogenic diets and psoriasis. Two open-label single-arm studies<sup>(73,74)</sup> and one case study<sup>(72)</sup> were identified in this review, all of which used very low-calorie ketogenic diets, between 300 and 500 kcal/d, and were conducted



only in subjects with obesity or overweight. Although significant improvements in psoriasis severity were observed in these studies<sup>(72–74)</sup> it is currently unclear whether this was due to the very low calorie content or the specific ketogenic properties (protein-based diet with low carbohydrate intake) of the diets followed. Further RCTs are needed to fully assess the additional benefits of VLCKD versus other non-ketogenic diets with the same calorie intake. This is in line with conclusions of a narrative review on nutritional management of VLCKD in psoriasis<sup>(160)</sup>. Furthermore, no studies have been conducted on ketogenic diets in PLWP without overweight or obesity.

A gluten-free diet (GFD) in coeliac or gluten-sensitive populations of people with psoriasis also seems to have a beneficial effect on symptom severity. Overall, evidence suggests that patients with psoriasis with gluten-related antibodies may benefit from a GFD; however, larger trials are still lacking<sup>(82)</sup>. Additionally, it should be acknowledged that not all people living with psoriasis are also living with obesity or overweight, or have a sensitivity to gluten. This leaves a large proportion of patients without any evidence-based dietary advice. A recent Cochrane review highlighted the need for more studies on the effects of diets other than LCDs on psoriasis severity, dietary interventions in people without obesity, and in people with mild psoriasis<sup>(65)</sup>.

A greater adherence to a Mediterranean diet (MD) shows a positive trend in helping to manage psoriasis. This dietary pattern is anti-inflammatory and is associated with significant reductions in both IL-6 and IL-1 $\beta$  levels<sup>(161)</sup>, key pro-inflammatory cytokines in psoriasis<sup>(31)</sup>, which may explain the reason for these findings. Certain individual foods components of the MD (extra-virgin olive oil, fruit, vegetables and fish<sup>(77)</sup>) have been associated with lower psoriasis severity (Table 5). The MD is a dietary pattern typically high in fruits and vegetables, legumes, whole grains, fish, nuts and monounsaturated fatty acids (MUFA) such as extra-virgin olive oil (EVOO), with a moderate intake of meat, dairy and alcohol<sup>(75)</sup> shown to have anti-inflammatory effects<sup>(161)</sup>. Dietary patterns are complex combinations of foods and nutrients that act synergistically; they account for inter-relations of foods, represent the cumulative exposure to different diet components, and may have stronger effects on health than any single component<sup>(162,163)</sup>. Therefore, it is difficult to attribute effects to single dietary components<sup>(162–165)</sup>. It is also important to note that these findings are based on cross-sectional studies, which cannot establish a cause-and-effect relationship between adherence to the Mediterranean diet or its individual components and psoriasis severity.

Regarding supplementation, high doses of omega-3 with an increased eicosapentaenoic (EPA):docosahexaenoic (DHA) ratio of 3:1 have shown potential for alleviating psoriasis symptoms<sup>(96)</sup>. A higher ratio of EPA:DHA is associated with further reductions in C-reactive protein (CRP) compared with lower ratios<sup>(166)</sup>. CRP is a pro-inflammatory biomarker shown to be elevated in psoriasis<sup>(36)</sup>, which may explain some of the beneficial effect seen in higher ratio supplementation. Probiotic supplementation also shows promising results for alleviating psoriasis symptoms; however, the studies identified for the review were small and heterogeneous. Additionally, limited evidence is available on long-term follow-ups, specific strain, amount, dosage or duration of consumption for probiotics.

Overall, the evidence for dietary supplementation in managing psoriasis is inconclusive, and no evidence on optimal dose for any supplement is apparent. Larger controlled studies are needed to elucidate any dietary approach that is helpful, specifically in PLWP without obesity and coeliac- or gluten-sensitivity-free populations. This is in line with findings from several systematic reviews on diet and psoriasis<sup>(64,122)</sup>.

## Grey literature

Considering the limited evidence for specific dietary approaches for psoriasis management, it is understandable that there are no specific dietary guidelines for the management of psoriasis. This could also be why most of the grey literature provides very limited dietary information specific to psoriasis management and instead focuses on dietary advice for associated comorbidities. However, this review has highlighted that, despite the lack of guidelines, PLWP do modify their diet to try to manage their psoriasis symptoms, often without consulting a healthcare professional (HCP)<sup>(44,45,62)</sup>. The diets trialled are often restrictive and could have detrimental effects on health and wellbeing. Therefore, it is important for HCPs and PLWP to understand the potential harms of following restrictive diets, especially considering the associated comorbidities. Stakeholders and those responsible for providing support for PLWP should provide more specific guidance on the potential harms of popular diets, in particular highlighting the risks of following restrictive diets, dietary aspects to consider if following elimination diets and the importance of consulting an HCP regarding dietary modifications. However, this review has highlighted that there is limited research on the use of popular diets and support that PLWP would like regarding diet. This is especially true for the United Kingdom where, even though there are an estimated 1.1 million PLWP, no studies were identified that have explored the perceived role of diet in the management of psoriasis, dietary information acquisition and dietary support wanted by PLWP in the United Kingdom, which highlights an important research gap. Further understanding dietary information acquisition and advice being suggested could enable stakeholders to provide more support to PLWP by increasing awareness of the potential health impacts of following popular diets.

## Limitations of the review

Although the search strategy and inclusion criteria used in this scoping review followed PRSIMA-ScR systematic methods for scoping reviews, there are several limitations. Only literature written in English was included; this could have excluded relevant studies and guidelines conducted in other languages. The challenges of searching for grey literature could result in bias, and the reproducibility of grey literature searching is difficult. The strategy used to search grey literature in this review was based on the methodology of Godin *et al.*<sup>(49)</sup>, and pre-defined targeted search terms were used to try to reduce bias and improve reproducibility, alongside using incognito mode during google searches. However, website searching is dependent on the specific website and correct functioning. Additionally, this review did not contact experts in the field of psoriasis management



to ask for additional reports or unpublished studies they were aware of. This may have resulted in some relevant studies and grey literature being omitted. Including studies that investigated the nutritional status of PLwP may have also made this review more comprehensive.

Despite these limitations, this review provides a comprehensive summary of the current available evidence and grey literature regarding the role of diet in the management of psoriasis. This review goes beyond previous review articles on diet and psoriasis, by including grey literature, studies on the dietary perceptions of people living with psoriasis and systematically searching for common dietary modifications used by PLwP on PubMed and SCOPUS as they emerged from the literature.

## Conclusion

This scoping review provides a comprehensive overview of evidence of the role of diet in the management of psoriasis. It is the first study to review such a wide evidence base on the role of diet in managing psoriasis: exploring the dietary intakes of people living with psoriasis (PLwP), perceptions and use of diet in PLwP, the dietary approaches and the grey literature on psoriasis management.

Overall, there is limited evidence on all themes identified in this review, and the methodology and outcome measures of the studies identified vary widely. Dietary intakes of PLwP warrant homogeneous longitudinal studies to elucidate a causal relationship between diet and psoriasis status. In the absence of dietary guidelines, PLwP are self-prescribing dietary modifications suggested in the popular literature. These are often restrictive and could have detrimental effects on health and wellbeing. No studies have been conducted on sources of dietary information for people with psoriasis. There is also an absence of studies investigating the effects of popular dietary recommendations on psoriasis symptoms, patient experience and the perceptions of healthcare professionals (HCPs) on the role of diet in managing psoriasis. None has been conducted in the United Kingdom.

Some dietary methods have been shown to improve psoriasis severity, but only in specific populations: low-calorie diets in people with obesity or overweight, and gluten-free diets in those with coeliac disease or gluten sensitivity. The evidence suggests that diets with anti-inflammatory properties, particularly the Mediterranean diet (MD), may have beneficial effects on psoriasis through moderating specific inflammatory pathways in psoriasis<sup>(42,161)</sup>. However, this is based on cross-sectional studies<sup>(75–78)</sup>, and larger intervention studies are needed before any cause and effect can be ascertained regarding psoriasis or psoriasis severity. Other dietary approaches lack high-quality evidence to support their use. Larger controlled trials in PLwP without obesity or overweight, and coeliac or gluten-sensitive free populations, are necessary prior to any dietary recommendations being made for psoriasis management.

Several studies identified in this review highlighted individual foods that were associated with psoriasis severity. However, it is difficult to attribute effects to single dietary components, as dietary patterns are complex combinations of foods and

nutrients which work synergistically<sup>(162,163)</sup>. Caution should be used when singling out specific foods as 'good' or 'bad' for certain conditions, without a robust evidence base, as this is an oversimplification which may lead to unhealthy eating behaviours<sup>(164,165)</sup>. Deleterious dietary recommendations should also consider the food that will be substituted as a result of cutting out specific foods<sup>(163–165)</sup>. It is also important to note that these findings are based on cross-sectional studies, which cannot establish a cause-and-effect relationship between these individual foods and psoriasis severity.

In the absence of dietary guidelines or evidence-based dietary recommendations for psoriasis, nutritionists and healthcare professionals should provide dietary support to PLwP in other ways, beyond standard healthy eating guidance. This should include highlighting the potential negative impacts of popular restrictive diets and the importance of discussing dietary modifications with healthcare professionals with nutritional expertise.

Grey literature resources for HCPs and PLwP should provide more comprehensive advice on diet specific to psoriasis. This should include information on the risks of following restrictive or elimination diets and the importance of discussing dietary modifications with an HCP. However, to make this advice as beneficial as possible, further research is needed to understand dietary information acquisition of PLwP, commonly recommended diets in the popular literature and the perceptions of PLwP on the dietary support available. Understanding the role of diet in the management of psoriasis from HCPs' point of view would also enable advice to be more comprehensive. With the significant comorbidities associated with psoriasis, understanding dietary behaviours, perceived skin response, information acquisition and patient experience will play a key role in holistic patient care for people with psoriasis.

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## Competing interests

The authors declare no conflict of interest.

## Authorship

Poppy Hawkins: developed search strategy, performed scoping review, screening and interpretation of included literature, and drafted the manuscript. Rosalind Fallaize, Kate Earl and Athanasios Tektonidis: contributed to study design and drafting of the manuscript. All authors included reviewed and approved the final manuscript.

## References

- World Health Organization (2016) *Global Report on Psoriasis*, 2016. World Health Organization. <https://apps.who.int/iris/handle/10665/204417>. Accessed on June 2021.
- Griffiths CEM, Armstrong AW, Gudjonsson JE, *et al.* (2021) Psoriasis. *Lancet* **397**, 1301–1315. [https://doi.org/10.1016/S0140-6736\(20\)32549-6](https://doi.org/10.1016/S0140-6736(20)32549-6).
- Takeshita J, Grewal S & Langan SM (2017) Psoriasis and comorbid diseases: epidemiology. *J Am Acad Dermatol* **76**, 377–390.
- Parisi R, Rutter MK, Lunt M, *et al.* (2015) Psoriasis and the risk of major cardiovascular events: cohort study using the Clinical Practice Research Datalink. *J Invest Dermatol* **135**, 2189–2197.
- Global Psoriasis Atlas – GPA Statistics. Available at <https://www.globalpsoriasisatlas.org/en/statistics>. Accessed May 20, 2022.
- Michalek IM, Loring B & John SM (2017) A systematic review of worldwide epidemiology of psoriasis. *J Eur Acad Dermatol Venereol* **31**, 205–212. <https://doi.org/10.1111/JDV.13854>.
- Gibbs S (1996) Skin disease and socioeconomic conditions in rural Africa: Tanzania. *Int J Dermatol* **35**, 633–639. <https://doi.org/10.1111/J.1365-4362.1996.TB03687.X>.
- Danielsen K, Olsen AO, Wilsgaard T, *et al.* (2013) Is the prevalence of psoriasis increasing? A 30-year follow-up of a population-based cohort. *Br J Dermatol* **168**, 1303–1310. <https://doi.org/10.1111/BJD.12230>.
- Armstrong AW, Mehta MD, Schupp CW, *et al.* (2021) Psoriasis prevalence in adults in the United States. *JAMA Dermatol* **157**, 940–946. <https://doi.org/10.1001/JAMADERMATOL.2021.2007>.
- Mattei PL, Corey KC & Kimball AB (2014) Psoriasis Area Severity Index (PASI) and the Dermatology Life Quality Index (DLQI): the correlation between disease severity and psychological burden in patients treated with biological therapies. *J Eur Acad Dermatol Venereol* **28**, 333–337. <https://doi.org/10.1111/JDV.12106>.
- Puig L, Thom H, Mollon P, *et al.* (2017) Clear or almost clear skin improves the quality of life in patients with moderate-to-severe psoriasis: a systematic review and meta-analysis. *J Eur Acad Dermatol Venereol* **31**, 213–220. <https://doi.org/10.1111/JDV.14007>.
- Stern RS, Nijsten T, Feldman SR, *et al.* (2004) Psoriasis is common, carries a substantial burden even when not extensive, and is associated with widespread treatment dissatisfaction. *J Invest Dermatol Symp Proc* **9**, 136–139.
- Florek AG, Wang CJ & Armstrong AW. (2018) Treatment preferences and treatment satisfaction among psoriasis patients: a systematic review. *Arch Dermatol Res* **310**, 271–319. <https://doi.org/10.1007/S00403-018-1808-X>.
- Zhou X, Chen Z & Bi X (2021) An update review of biosimilars of adalimumab in psoriasis – bioequivalence and interchangeability. *Drug Des Devel Ther* **15**, 2987–2998. <https://doi.org/10.2147/DDDT.S317382>.
- Feldman SR (2015) Inflammatory diseases: Integrating biosimilars into clinical practice. *Semin Arthritis Rheum* **44**, S16–S21. <https://doi.org/10.1016/j.semarthrit.2015.04.003>.
- Vanderpuye-Orgle J, Zhao Y, Lu J, *et al.* (2015) Evaluating the economic burden of psoriasis in the United States. *J Am Acad Dermatol* **72**, 961–967.e5. <https://doi.org/10.1016/J.JAAD.2015.02.1099>.
- Mease PJ, Gladman DD, Papp KA, *et al.* (2013) Prevalence of rheumatologist-diagnosed psoriatic arthritis in patients with psoriasis in European/North American dermatology clinics. *J Am Acad Dermatol* **69**, 729–735. <https://doi.org/10.1016/J.JAAD.2013.07.023>.
- Ogdie A, Ad Langan S, Love T, *et al.* Prevalence and treatment patterns of psoriatic arthritis in the UK. <https://doi.org/10.1093/rheumatology/kes324>.
- Armstrong AW, Schupp C & Bebo B (2012) Psoriasis comorbidities: results from the National Psoriasis Foundation Surveys 2003 to 2011. *Dermatology* **225**, 121–126. <https://doi.org/10.1159/000342180>.
- Armstrong AW, Harskamp CT & Armstrong EJ (2013) Psoriasis and the risk of diabetes mellitus: a systematic review and meta-analysis. *JAMA Dermatol* **149**, 84–91. <https://doi.org/10.1001/2013.JAMADERMATOL.406>.
- Armstrong AW, Harskamp CT & Armstrong EJ. (2012) The association between psoriasis and obesity: a systematic review and meta-analysis of observational studies. *Nutr Diabetes* **2**. <https://doi.org/10.1038/NUTD.2012.26>.
- Armstrong EJ, Harskamp CT & Armstrong AW (2013) Psoriasis and major adverse cardiovascular events: a systematic review and meta-analysis of observational studies. *J Am Heart Assoc* **2**, e000062.
- Ma C, Schupp CW, Armstrong EJ, *et al.* (2014) Psoriasis and dyslipidemia: a population-based study analyzing the National Health and Nutrition Examination Survey (NHANES). *J Eur Acad Dermatol Venereol* **28**, 1109–1112. <https://doi.org/10.1111/jdv.12232>.
- Candia R, Ruiz A, Torres-Robles R, *et al.* (2015) Risk of non-alcoholic fatty liver disease in patients with psoriasis: a systematic review and meta-analysis. *J Eur Acad Dermatol Venereol* **29**, 656–662. <https://doi.org/10.1111/JDV.12847>.
- Trafford AM, Parisi R, Kontopantelis E, *et al.* (2019) Association of psoriasis with the risk of developing or dying of cancer: a systematic review and meta-analysis. *JAMA Dermatol* **155**, 1390–1403. <https://doi.org/10.1001/JAMADERMATOL.2019.3056>.
- Alinaghi F, Tekin HG, Burisch J, *et al.* (2020) Global prevalence and bidirectional association between psoriasis and inflammatory bowel disease—a systematic review and meta-analysis. *J Crohns Colitis* **14**, 351–360. <https://doi.org/10.1093/ecco-jcc/jjz152>.
- Dowlatslahi EA, Wakkee M, Arends LR, *et al.* (2014) The prevalence and odds of depressive symptoms and clinical depression in psoriasis patients: a systematic review and meta-analysis. *J Invest Dermatol* **134**, 1542–1551. <https://doi.org/10.1038/JID.2013.508>.
- Liang SE, Cohen JM & Ho RS (2019) Psoriasis and suicidality: a review of the literature. *Dermatol Ther* **32**, e12771. <https://doi.org/10.1111/DTH.12771>.
- Zeng J, Luo S, Huang Y, *et al.* (2017) Critical role of environmental factors in the pathogenesis of psoriasis. *J Dermatol* **44**, 863–872. <https://doi.org/10.1111/1346-8138.13806>.
- Campanati A, Marani A, Martina E, *et al.* (2021) Psoriasis as an immune-mediated and inflammatory systemic disease: from pathophysiology to novel therapeutic approaches. *Biomedicine* **9**, 1511. <https://doi.org/10.3390/BIMEDICINES9111511>.
- Armstrong AW & Read C (2020) Pathophysiology, clinical presentation, and treatment of psoriasis: a review. *JAMA* **323**, 1945–1960.
- Zhou X, Chen Y, Cui L, *et al.* (2022) Advances in the pathogenesis of psoriasis: from keratinocyte perspective. *Cell Death Dis* **13**, 1–13. <https://doi.org/10.1038/s41419-022-04523-3>.
- Nestle FO, Kaplan DH & Barker J (2009) Mechanisms of disease: psoriasis. *N Engl J Med* **361**, 496–509.
- Mahil SK, Capon F & Barker JN (2016) Update on psoriasis immunopathogenesis and targeted immunotherapy. *Semin*

- Immunopathol* **38**, 11–27. <https://doi.org/10.1007/S00281-015-0539-8>.
35. Korman NJ (2020) Management of psoriasis as a systemic disease: what is the evidence? *Br J Dermatol* **182**, 840–848. <https://doi.org/10.1111/BJD.18245>.
  36. Dowlatshahi EA, Van Der Voort EAM, Arends LR, *et al.* (2013) Markers of systemic inflammation in psoriasis: a systematic review and meta-analysis. *Br J Dermatol* **169**, 266–282. <https://doi.org/10.1111/bjd.12355>.
  37. Bai F, Zheng W, Dong Y, *et al.* (2017) Serum levels of adipokines and cytokines in psoriasis patients: a systematic review and meta-analysis. *Oncotarget* **9**, 1266–1278. <https://doi.org/10.18632/oncotarget.22260>.
  38. Rendon A & Schäkel K (2019) Psoriasis pathogenesis and treatment. *Int J Mol Sci* **20**. <https://doi.org/10.3390/IJMS20061475>.
  39. Hwang ST, Nijsten T & Elder JT (2017) Recent highlights in psoriasis research. *J Invest Dermatol* **137**, 550–556. <https://doi.org/10.1016/j.jid.2016.11.007>.
  40. Psoriasis Top 10 | James Lind Alliance. Available at <https://www.jla.nihr.ac.uk/priority-setting-partnerships/psoriasis/to-p-10-priorities.htm>. Accessed November 19, 2021.
  41. Katsimbri P, Korakas E, Kountouri A, *et al.* (2021) The effect of antioxidant and anti-inflammatory capacity of diet on psoriasis and psoriatic arthritis phenotype: nutrition as therapeutic tool? *Antioxidants* **10**, 1–28. <https://doi.org/10.3390/antiox10020157>.
  42. Kanda N, Hoashi T & Saeki H (2020) Nutrition and psoriasis. *Int J Mol Sci* **21**, 1–19. <https://doi.org/10.3390/ijms21155405>.
  43. Millsop JW, Bhatia BK, Debbaneh M, *et al.* (2014) Diet and psoriasis, part III: role of nutritional supplements. *J Am Acad Dermatol* **71**, 561–569. <https://doi.org/10.1016/j.jaad.2014.03.016>.
  44. Pham T, Sokol H, Halioua B, *et al.* (2021) Immune-mediated inflammatory diseases and nutrition: results from an online survey on patients' practices and perceptions. *BMC Nutr* **7**. <https://doi.org/10.1186/s40795-021-00446-y>.
  45. Afifi L, Danesh MJ, Lee KM, *et al.* (2017) Dietary behaviors in psoriasis: patient-reported outcomes from a U.S. National Survey. *Dermatol Ther (Heidelb)* **7**, 227–242. <https://doi.org/10.1007/s13555-017-0183-4>.
  46. Debbaneh M, Millsop JW, Bhatia BK, *et al.* (2014) Diet and psoriasis, part I: impact of weight loss interventions. *J Am Acad Dermatol* **71**, 133–140. <https://doi.org/10.1016/j.jaad.2014.02.012>.
  47. Peters MDJ, Marnie C, Tricco AC, *et al.* (2020) Updated methodological guidance for the conduct of scoping reviews. *JBI Evid Synth* **18**, 2119–2126. <https://doi.org/10.11124/JBIES-20-00167>.
  48. ICGL Luxembourg Definition (1997) Third International Conference on Grey Literature in 1997. Expanded in New York, 2004.
  49. Godin K, Stapleton J, Kirkpatrick SI, *et al.* (2015) Applying systematic review search methods to the grey literature: a case study examining guidelines for school-based breakfast programs in Canada. *Syst Rev* **4**. <https://doi.org/10.1186/s13643-015-0125-0>.
  50. Tricco AC, Lillie E, Zarin W, *et al.* (2018) PRISMA extension for scoping reviews (PRISMA-ScR): checklist and explanation. *Ann Intern Med* **169**, 467–473.
  51. Polo TCF, Corrente JE, Miot LDB, *et al.* (2020) Dietary patterns of patients with psoriasis at a public healthcare institution in Brazil. *An Bras Dermatol* **95**, 452–458. <https://doi.org/10.1016/j.abd.2020.02.002>.
  52. Yazdanpanah MJ, Vahabi-Amlashi S, Nematy M, *et al.* (2021) Association of serum lipid profiles and dietary intakes of vitamin E and fiber with psoriasis severity. *Caspian J Intern Med* **12**, 606–612. <https://doi.org/10.22088/cjim.12.4.606>.
  53. Ingkapiroj K, Chularojanamontri L, Chaiyabutr C, *et al.* (2022) Dietary habits and perceptions of psoriatic patients: Mediterranean versus Asian diets. *J Dermatol Treatment* **33**, 2290–2296. <https://doi.org/10.1080/09546634.2021.1959500>.
  54. Kashani A, Moludi J, Fateh HL, *et al.* (2021) Dietary inflammatory index in relation to psoriasis risk, cardiovascular risk factors, and clinical outcomes: a case-control study in psoriasis patients. *Appl Physiol Nutr Metab* **46**, 1517–1524. <https://doi.org/10.1139/apnm-2021-0217>.
  55. Johnson JA, Ma C, Kanada KN, *et al.* (2014) Diet and nutrition in psoriasis: analysis of the National Health and Nutrition Examination Survey (NHANES) in the United States. *J Eur Acad Dermatol Venereol* **28**, 327–332. <https://doi.org/10.1111/jdv.12105>.
  56. Yamashita H, Morita T, Ito M, *et al.* (2019) Dietary habits in Japanese patients with psoriasis and psoriatic arthritis: low intake of meat in psoriasis and high intake of vitamin A in psoriatic arthritis. *J Dermatol* **46**, 759–769. <https://doi.org/10.1111/1346-8138.15032>.
  57. Barrea L, Macchia PE, Tarantino G, *et al.* (2015) Nutrition: a key environmental dietary factor in clinical severity and cardio-metabolic risk in psoriatic male patients evaluated by 7-day food-frequency questionnaire. *J Transl Med* **13**. <https://doi.org/10.1186/s12967-015-0658-y>.
  58. Wasiluk D, Stefanska E, Ostrowska L, *et al.* (2012) Nutritive value of daily food rations of patients with psoriasis vulgaris: a preliminary report. *Postepy Dermatol Alergol* **29**, 348–355. <https://doi.org/10.5114/pdia.2012.31487>.
  59. Yousefzadeh H, Mahmoudi M, Banihashemi M, *et al.* (2017) Investigation of dietary supplements prevalence as complementary therapy: comparison between hospitalized psoriasis patients and non-psoriasis patients, correlation with disease severity and quality of life. *Complement Ther Med* **33**, 65–71. <https://doi.org/10.1016/j.ctim.2017.06.005>.
  60. Wilson PB (2014) Is dietary supplementation more common among adults with psoriasis? Results from the national health and nutrition examination survey. *Complement Ther Med* **22**, 159–165. <https://doi.org/10.1016/j.ctim.2013.12.007>.
  61. Festugato M (2011) Pilot study on which foods should be avoided by patients with psoriasis | Estudo piloto sobre alimentos que devem ser evitados nos portadores de psoríase. *An Bras Dermatol* **86**, 1103–1108. <https://doi.org/10.1590/S0365-05962011000600006>.
  62. Dhinsa H, Wu N, Gibbons M, *et al.* (2021) Diet and nutritional behaviors in patients with psoriasis: a cross-sectional study. *JAAD Int* **5**, 76–77. <https://doi.org/10.1016/j.jdin.2021.07.009>.
  63. Del Giglio M, Gisondi P, Tessari G, *et al.* (2012) Weight reduction alone may not be sufficient to maintain disease remission in obese patients with psoriasis: a randomized, investigator-blinded study. *Dermatology* **224**, 31–37. <https://doi.org/10.1159/000335566>.
  64. Ford AR, Siegel M, Bagel J, *et al.* (2018) Dietary recommendations for adults with psoriasis or psoriatic arthritis from the Medical Board of the National Psoriasis Foundation: a systematic review. *JAMA Dermatol* **154**, 934–950. <https://doi.org/10.1001/jamadermatol.2018.1412>.
  65. Ko S-HSH, Chi C-CCC, Yeh M-LML, *et al.* (2019) Lifestyle changes for treating psoriasis. *Cochrane Database Syst Rev* **2019**. <https://doi.org/10.1002/14651858.CD011972.pub2>.
  66. Al-Mutairi N & Nour T (2014) The effect of weight reduction on treatment outcomes in obese patients with psoriasis on biologic therapy: a randomized controlled prospective trial. *Expert Opin Biol Ther* **14**, 749–756. <https://doi.org/10.1517/14712598.2014.900541>.





67. Gisondi P, Del Giglio M, Di Francesco V, *et al.* (2008) Weight loss improves the response of obese patients with moderate-to-severe chronic plaque psoriasis to low-dose cyclosporine therapy: a randomized, controlled, investigator-blinded clinical trial. *Am J Clin Nutr* **88**, 1242–1247. <https://doi.org/10.3945/ajcn.2008.26427>.
68. Jensen P, Zachariae C, Christensen R, *et al.* (2013) Effect of weight loss on the severity of psoriasis: a randomized clinical study. *JAMA Dermatol* **149**, 795–801. <https://doi.org/10.1001/jamadermatol.2013.722>.
69. Guida B, Napoleone A, Trio R, *et al.* (2014) Energy-restricted, *n*-3 polyunsaturated fatty acids-rich diet improves the clinical response to immuno-modulating drugs in obese patients with plaque-type psoriasis: a randomized control clinical trial. *Clin Nutr* **33**, 399–405. <https://doi.org/10.1016/j.clnu.2013.09.010>.
70. Roongpisuthipong W, Pongpudpunth M, Roongpisuthipong C, *et al.* (2013) The effect of weight loss in obese patients with chronic stable plaque-type psoriasis. *Dermatol Res Pract* **2013**. <https://doi.org/10.1155/2013/795932>.
71. Jensen P, Christensen R, Zachariae C, *et al.* (2016) Long-term effects of weight reduction on the severity of psoriasis in a cohort derived from a randomized trial: a prospective observational follow-up study. *Am J Clin Nutr* **104**, 259–265. <https://doi.org/10.3945/ajcn.115.125849>.
72. Castaldo G, Galdo G, Rotondi A, *et al.* (2016) Very low-calorie ketogenic diet may allow restoring response to systemic therapy in relapsing plaque psoriasis. *Obes Res Clin Pract* **10**, 348–352. <https://doi.org/10.1016/j.orcp.2015.10.008>.
73. Castaldo G, Rastrelli L, Galdo G, *et al.* (2020) Aggressive weight-loss program with a ketogenic induction phase for the treatment of chronic plaque psoriasis: a proof-of-concept, single-arm, open-label clinical trial. *Nutrition* **74**. <https://doi.org/10.1016/j.nut.2020.110757>.
74. Castaldo G, Pagano I, Grimaldi M, *et al.* (2021) Effect of very-low-calorie ketogenic diet on psoriasis patients: a nuclear magnetic resonance-based metabolomic study. *J Proteome Res* **20**, 1509–1521. <https://doi.org/10.1021/acs.jproteome.0c00646>.
75. Phan C, Touvier M, Kesse-Guyot E, *et al.* (2018) Association between mediterranean anti-inflammatory dietary profile and severity of psoriasis: results from the NutriNet-Santé cohort. *JAMA Dermatol* **154**, 1017–1024. <https://doi.org/10.1001/jamadermatol.2018.2127>.
76. Korovesi A, Dalamaga M, Kotopouli M, *et al.* (2019) Adherence to the Mediterranean diet is independently associated with psoriasis risk, severity, and quality of life: a cross-sectional observational study. *Int J Dermatol* **58**, e164–e165. <https://doi.org/10.1111/ijd.14523>.
77. Barrea L, Balato N, Di Somma C, *et al.* (2015) Nutrition and psoriasis: is there any association between the severity of the disease and adherence to the Mediterranean diet? *J Transl Med* **13**. <https://doi.org/10.1186/s12967-014-0372-1>.
78. Molina-Leyva A, Cuenca-Barrales C, Vega-Castillo JJJ, *et al.* (2019) Adherence to Mediterranean diet in Spanish patients with psoriasis: cardiovascular benefits? *Dermatol Ther* **32**. <https://doi.org/10.1111/dth.12810>.
79. Bhatia BK, Millsop JW, Debbaneh M, *et al.* (2014) Diet and psoriasis, part II: celiac disease and role of a gluten-free diet. *J Am Acad Dermatol* **71**, 350–358. <https://doi.org/10.1016/j.jaad.2014.03.017>.
80. Ungprasert P, Wijampreecha K & Kittanamongkolchai W (2017) Psoriasis and risk of celiac disease: a systematic review and meta-analysis. *Indian J Dermatol* **62**, 41. <https://doi.org/10.4103/0019-5154.198031>.
81. Michaëlsson G, Gerdén B, Hagforsen E, *et al.* (2000) Psoriasis patients with antibodies to gliadin can be improved by a gluten-free diet. *Br J Dermatol* **142**, 44–51.
82. Kolchak NA, Tetarnikova MK, Theodoropoulou MS, *et al.* (2018) Prevalence of antigliadin IgA antibodies in psoriasis vulgaris and response of seropositive patients to a gluten-free diet. *J Multidiscip Healthc* **11**, 13–19. <https://doi.org/10.2147/JMDH.S122256>.
83. De Bastiani R, Gabrielli M, Lora L, *et al.* (2015) Association between coeliac disease and psoriasis: Italian primary care multicentre study. *Dermatology* **230**, 156–160. <https://doi.org/10.1159/000369615>.
84. Addolorato G, Parente A, De Lorenzi G, *et al.* (2003) Rapid regression of psoriasis in a coeliac patient after gluten-free diet: a case report and review of the literature. *Digestion* **68**, 9–12. <https://doi.org/10.1159/000073220>.
85. Tahiri L, Azzouzi H, Squalli G, *et al.* (2014) Celiac disease causing severe osteomalacia: An association still present in Morocco! *Pan Afr Med J* **19**. <https://doi.org/10.11604/pamj.2014.19.43.2757>.
86. Zamani F, Alizadeh S, Amiri A, *et al.* (2010) Psoriasis and coeliac disease; is there any relationship? *Acta Derm Venereol* **90**, 295–296. <https://doi.org/10.2340/00015555-0829>.
87. Almutairi N & Shaaban D. (2022) Clinical implications of intermittent Ramadan fasting on stable plaque psoriasis: A prospective observational study. *Postepy Dermatol Alergol* **39**, 368–374. <https://doi.org/10.5114/ada.2021.107098>.
88. Damiani G, Watah A, Bridgewood C, *et al.* (2019) The impact of Ramadan fasting on the reduction of PASI score, in moderate-to-severe psoriatic patients: a real-life multicenter study. *Nutrients* **11**,. <https://doi.org/10.3390/nu11020277>.
89. Grine L, Hilhorst N, Michels N, *et al.* (2022) The effects of modified intermittent fasting in psoriasis (MANGO): protocol for a two-arm pilot randomized controlled open cross-over study. *JMIR Res Protoc* **11**. <https://doi.org/10.2196/26405>.
90. Marta F (2022) Veganism in acne, atopic dermatitis, and psoriasis: benefits of a plant-based diet. *Clin Dermatol*. <https://doi.org/10.1016/j.clindermatol.2022.09.008>.
91. Rastmanesh R (2009) *Psoriasis and Vegetarian Diets: A Role for Cortisol and Potassium?* Vol **72**. United States, 368. <https://doi.org/10.1016/j.mehy.2008.09.031>.
92. Yang SJ & Chi CC (2019) Effects of fish oil supplement on psoriasis: a meta-analysis of randomized controlled trials. *BMC Complement Altern Med* **19**. <https://doi.org/10.1186/s12906-019-2777-0>.
93. Upala S, Yong WC, Theparee T, *et al.* (2017) Effect of omega-3 fatty acids on disease severity in patients with psoriasis: a systematic review. *Int J Rheum Dis* **20**, 442–450. <https://doi.org/10.1111/1756-185X.13051>.
94. Chen X, Hong S, Sun X, *et al.* (2020) Efficacy of fish oil and its components in the management of psoriasis: a systematic review of 18 randomized controlled trials. *Nutr Rev* **78**, 827–840. <https://doi.org/10.1093/NUTRIT/NUZ098>.
95. Clark CCT, Taghizadeh M, Nahavandi M, *et al.* (2019) Efficacy of  $\omega$ -3 supplementation in patients with psoriasis: a meta-analysis of randomized controlled trials. *Clin Rheumatol* **38**, 977–988. <https://doi.org/10.1007/S10067-019-04456-X>.
96. Tveit KSKS, Brokstad KAKA, Berge RKRK, *et al.* (2020) A randomized, double-blind, placebo-controlled clinical study to investigate the efficacy of herring roe oil for treatment of psoriasis. *Acta Derm Venereol* **100**, 1–6. <https://doi.org/10.2340/00015555-3507>.
97. Khan SU, Lone AN, Khan MS, *et al.* (2021) Effect of omega-3 fatty acids on cardiovascular outcomes: a systematic review and meta-analysis. *EClinicalMedicine* **38**, 100997. <https://doi.org/10.1016/j.eclinm.2021.100997>.

- doi.org/10.1016/J.ECLINM.2021.100997/ATTACHMENT/079022C4-42B6-4816-A327-3B5BF0CF0017/MMC1.DOCX.
98. Gisondi P, Rossini M, Di Cesare A, *et al.* (2012) Vitamin D status in patients with chronic plaque psoriasis. *Br J Dermatol* **166**, 505–510. <https://doi.org/10.1111/j.1365-2133.2011.10699.x>.
  99. Hambly R & Kirby B (2017) The relevance of serum vitamin D in psoriasis: a review. *Arch Dermatol Res* **309**, 499–517. <https://doi.org/10.1007/S00403-017-1751-2>.
  100. Chandrashekar L, Krishna Kumari GR, Rajappa M, *et al.* (2015) 25-Hydroxy vitamin D and ischaemia-modified albumin levels in psoriasis and their association with disease severity. *Br J Biomed Sci* **72**, 56–60. <https://doi.org/10.1080/09674845.2015.11666797>.
  101. Filoni A, Vestita M, Congedo M, *et al.* (2018) Association between psoriasis and vitamin D: duration of disease correlates with decreased vitamin D serum levels: an observational case-control study. *Medicine (United States)* **97**. <https://doi.org/10.1097/MD.00000000000011185>.
  102. Stanescu AMA, Simionescu AA & Diaconu CC (2021) Oral vitamin D therapy in patients with psoriasis. *Nutrients* **13**, 1–12. <https://doi.org/10.3390/NU13010163>.
  103. Theodoridis X, Grammatikopoulou MG, Stamouli EM, *et al.* (2021) Effectiveness of oral vitamin D supplementation in lessening disease severity among patients with psoriasis: a systematic review and meta-analysis of randomized controlled trials. *Nutrition* **82**. <https://doi.org/10.1016/J.NUT.2020.111024>.
  104. Megna M, Ferrillo M, Barrea L, *et al.* (2020) Vitamin D and psoriasis: an update for dermatologists and nutritionists. *Minerva Endocrinol* **45**, 138–147. <https://doi.org/10.23736/S0391-1977.20.03190-9>.
  105. Disphanurat W, Viarasilpa W, Chakkavittumrong P, *et al.* (2019) The clinical effect of oral vitamin D2 supplementation on psoriasis: a double-blind, randomized, placebo-controlled study. *Dermatol Res Pract* **2019**. <https://doi.org/10.1155/2019/5237642>.
  106. Finamor DC, Sinigaglia-Coimbra R, Neves LCM, *et al.* (2013) A pilot study assessing the effect of prolonged administration of high daily doses of vitamin D on the clinical course of vitiligo and psoriasis. *Dermato-Endocrinology* **5**, 222–234. <https://doi.org/10.4161/derm.24808>.
  107. Al-Sultany HA (2020) Oral vitamin D therapy for chronic plaque-psoriasis among Iraqi patients, efficacy, and safety. *Int J Drug Deliv Technol* **10**, 227–231. <https://doi.org/10.25258/ijddt.10.2.7>.
  108. Jarrett P, Camargo CA, Coomarasamy C, *et al.* (2017) A randomized, double-blind, placebo-controlled trial of the effect of monthly vitamin D supplementation in mild psoriasis\*. **29**, 324–328. <https://doi.org/10.1080/09546634.2017.1373735>.
  109. Ingram MA, Jones MB, Stonehouse W, *et al.* (2018) Oral vitamin D<sub>3</sub> supplementation for chronic plaque psoriasis: a randomized, double-blind, placebo-controlled trial. *J Dermatol Treat* **29**, 648–657. <https://doi.org/10.1080/09546634.2018.1444728>.
  110. Mahtani R & Nair PMK (2022) Daily oral vitamin D<sub>3</sub> without concomitant therapy in the management of psoriasis: a case series. *Clin Immunol Commun* **2**, 17–22. <https://doi.org/10.1016/J.CLICOM.2022.01.001>.
  111. Judd SE & Tangpricha V (2009) Vitamin D deficiency and risk for cardiovascular disease. *Am J Med Sci* **338**, 40. <https://doi.org/10.1097/MAJ.0B013E3181AAEE91>.
  112. Brazzelli V, Grasso V, Fornara L, *et al.* (2010) Homocysteine, vitamin B12 and folic acid levels in psoriatic patients and correlation with disease severity. *Int J Immunopathol Pharmacol* **23**, 911–916. <https://doi.org/10.1177/039463201002300327>.
  113. Škovieřová H, Vidomanová E, Mahmood S, *et al.* (2016) The molecular and cellular effect of homocysteine metabolism imbalance on human health. *Int J Mol Sci* **17**, 1733. <https://doi.org/10.3390/IJMS17101733>.
  114. Gudjonsson J (2022) The effect of riboflavin on moderate to severe plaque type psoriasis. *Unpublished* 2022.
  115. Serwin AB, Wasowicz W, Gromadzinska J, *et al.* (2003) Selenium status in psoriasis and its relations to the duration and severity of the disease. *Nutrition* **19**, 301–304. [https://doi.org/10.1016/S0899-9007\(02\)01081-X](https://doi.org/10.1016/S0899-9007(02)01081-X).
  116. Serwin AB, Wasowicz W & Chodynicka B (2006) Selenium supplementation, soluble tumor necrosis factor- $\alpha$  receptor type 1, and C-reactive protein during psoriasis therapy with narrowband ultraviolet B. *Nutrition* **22**, 860–864. <https://doi.org/10.1016/J.NUT.2006.05.011>.
  117. Kharaeva Z, Gostova E, De Luca C, *et al.* (2009) Clinical and biochemical effects of coenzyme Q<sub>10</sub>, vitamin E, and selenium supplementation to psoriasis patients. *Nutrition* **25**, 295–302. <https://doi.org/10.1016/j.nut.2008.08.015>.
  118. Benhadou F, Mintoff D, Schnebert B, *et al.* (2018) Psoriasis and microbiota: a systematic review. *Diseases* **6**, 47. <https://doi.org/10.3390/DISEASES6020047>.
  119. Schade L, Mesa D, Faria AR, *et al.* (2022) The gut microbiota profile in psoriasis: a Brazilian case-control study. *Lett Appl Microbiol* **74**, 498–504. <https://doi.org/10.1111/LAM.13630>.
  120. Hidalgo-Cantabrana C, Gómez J, Delgado S, *et al.* (2019) Gut microbiota dysbiosis in a cohort of patients with psoriasis. *Br J Dermatol* **181**, 1287–1295. <https://doi.org/10.1111/BJD.17931>.
  121. Dei-Cas I, Giliberto F, Luce L, *et al.* (2020) Metagenomic analysis of gut microbiota in non-treated plaque psoriasis patients stratified by disease severity: development of a new Psoriasis-Microbiome Index. *Sci Rep* **10**, 1–11. <https://doi.org/10.1038/s41598-020-69537-3>.
  122. Zeng L, Yu G, Wu Y, *et al.* (2021) The effectiveness and safety of probiotic supplements for psoriasis: a systematic review and meta-analysis of randomized controlled trials and preclinical trials. *J Immunol Res* **2021**. <https://doi.org/10.1155/2021/7552546>.
  123. Navarro-López V, Martínez-Andrés A, Ramírez-Boscá A, *et al.* (2019) Efficacy and safety of oral administration of a mixture of probiotic strains in patients with psoriasis: a randomized controlled clinical trial. *Acta Derm Venereol* **99**, 1078–1084. <https://doi.org/10.2340/00015555-3305>.
  124. Moludi J, Khedmatgozar H, Saiedi S, *et al.* (2021) Probiotic supplementation improves clinical outcomes and quality of life indicators in patients with plaque psoriasis: a randomized double-blind clinical trial. *Clin Nutr ESPEN* **46**, 33–39. <https://doi.org/10.1016/j.clnesp.2021.09.004>.
  125. Atabati H, Esmaili SA, Saburi E, *et al.* (2020) Probiotics with ameliorating effects on the severity of skin inflammation in psoriasis: evidence from experimental and clinical studies. *J Cell Physiol* **235**, 8925–8937. <https://doi.org/10.1002/JCP.29737>.
  126. Lin C, Zeng T, Deng Y, *et al.* (2021) Treatment of psoriasis vulgaris using *Bacteroides fragilis* BF839: a single-arm, open preliminary clinical study. *Shengwu Gongcheng Xuebao/Chin J Biotechnol* **37**, 3828–3835. <https://doi.org/10.13345/J.CJB.210198>.
  127. Moludi J, Fathollahi P, Khedmatgozar H, *et al.* (2022) Probiotics supplementation improves quality of life, clinical symptoms, and inflammatory status in patients with psoriasis. *J Drugs Dermatol* **21**, 637–644. <https://doi.org/10.36849/JDD.6237>.

128. Vijayashankar M & Raghunath N (2012) Pustular psoriasis responding to probiotics – a new insight. *Our Dermatol Online* **3**, 326–329. <https://doi.org/10.7241/OURD.20124.71>.
129. Kim GW, Park JM, Chin HW, *et al.* (2013) Comparative analysis of the use of complementary and alternative medicine by Korean patients with androgenetic alopecia, atopic dermatitis and psoriasis. *J Eur Acad Dermatol Venereol* **27**, 827–835. <https://doi.org/10.1111/J.1468-3083.2012.04583.X>.
130. Antiga E, Bonciolini V, Volpi W, *et al.* (2015) Oral curcumin (Meriva) is effective as an adjuvant treatment and is able to reduce IL-22 serum levels in patients with psoriasis vulgaris. *Biomed Res Int* **2015**. <https://doi.org/10.1155/2015/283634>.
131. Gamret ACC, Price A, Fertig RMRM, *et al.* (2018) Complementary and alternative medicine therapies for psoriasis: a systematic review. *JAMA Dermatol* **154**, 1330–1337.
132. Kurd SK, Smith N, VanVoorhees A, *et al.* (2008) Oral curcumin in the treatment of moderate to severe psoriasis vulgaris: a prospective clinical trial. *J Am Acad Dermatol* **58**, 625–631. <https://doi.org/10.1016/J.JAAD.2007.12.035>.
133. Ahmed Jawad H, Ibraheem Azhar Y & Al-Hamdi KI (2014) Evaluation of efficacy, safety and antioxidant effect of *Nigella sativa* in patients with psoriasis: a randomized clinical trial. *J Clin Exp Invest* **5**, 186–193.
134. Greenberger S, Harats D, Salameh F, *et al.* (2012) 9-*cis*-rich  $\beta$ -carotene powder of the alga *Dunaliella* reduces the severity of chronic plaque psoriasis: a randomized, double-blind, placebo-controlled clinical trial. *J Am Coll Nutr* **31**, 320–326. <https://doi.org/10.1080/07315724.2012.10720430>.
135. Barrea L, Muscogiuri G, Di Somma C, *et al.* (2018) Coffee consumption, metabolic syndrome and clinical severity of psoriasis: good or bad stuff? *Arch Toxicol* **92**, 1831–1845. <https://doi.org/10.1007/S00204-018-2193-0>.
136. Psoriasis: Assessment and Management Clinical Guideline (2012). London: Royal College of Physicians (UK). (NICE Clinical Guidelines, No. 153). Available from: <https://www.ncbi.nlm.nih.gov/books/NBK247829/>.
137. Duan Y, Zeng L, Zheng C, *et al.* (2018) Inflammatory links between high fat diets and diseases. *Front Immunol* **9**, 2649. <https://doi.org/10.3389/FIMMU.2018.02649>.
138. Honda T & Kabashima K (2019) Current understanding of the role of dietary lipids in the pathophysiology of psoriasis. *J Dermatol Sci* **94**, 314–320. <https://doi.org/10.1016/j.jde.2019.05.003>.
139. Higashi Y, Yamakuchi M, Fukushige T, *et al.* (2018) High-fat diet exacerbates imiquimod-induced psoriasis-like dermatitis in mice. *Exp Dermatol* **27**, 178–184. <https://doi.org/10.1111/EXD.13484>.
140. Herbert D, Franz S, Popkova Y, *et al.* (2018) High-fat diet exacerbates early psoriatic skin inflammation independent of obesity: saturated fatty acids as key players. *J Invest Dermatol* **138**, 1999–2009. <https://doi.org/10.1016/j.jid.2018.03.1522>.
141. Kuo SM (2013) The interplay between fiber and the intestinal microbiome in the inflammatory response. *Adv Nutr* **4**, 16–28. <https://doi.org/10.3945/AN.112.003046>.
142. Krishnamurthy VMR, Wei G, Baird BC, *et al.* (2012) High dietary fiber intake is associated with decreased inflammation and all-cause mortality in patients with chronic kidney disease. *Kidney Int* **81**, 300–306. <https://doi.org/10.1038/KI.2011.355>.
143. Lattimer JM & Haub MD (2010) Effects of dietary fiber and its components on metabolic health. *Nutrients* **2**, 1266–1289. <https://doi.org/10.3390/nu2121266>.
144. Block G, Jensen CD, Dalvi TB, *et al.* Vitamin C treatment reduces elevated C-reactive protein. <https://doi.org/10.1016/j.freeradbiomed.2008.09.030>.
145. Salinthon S, Kerns AR, Tsang V, *et al.* (2013)  $\alpha$ -Tocopherol (vitamin E) stimulates cyclic AMP production in human peripheral mononuclear cells and alters immune function. *Mol Immunol* **53**, 173–178. <https://doi.org/10.1016/J.MOLIMM.2012.08.005>.
146. Dhingra D, Michael M, Rajput H, *et al.* Dietary fibre in foods: a review. <https://doi.org/10.1007/s13197-011-0365-5>.
147. Calder PC (2006) n-3 Polyunsaturated Fatty Acids, Inflammation, and Inflammatory Diseases. *Am J Clin Nutr* **83**, 1505S–1519S. <https://doi.org/10.1093/ajcn/83.6.1505S>.
148. Hu FB (2002) Dietary pattern analysis: a new direction in nutritional epidemiology. *Curr Opin Lipidol* **13**, 3–9. <https://doi.org/10.1097/00041433-200202000-00002>.
149. McKeown NM, Fahey GC, Slavin J, *et al.* (2022) Fibre intake for optimal health: how can healthcare professionals support people to reach dietary recommendations? *BMJ* **378**. <https://doi.org/10.1136/BMJ-2020-054370>.
150. Nazarenkov N, Seeger K, Beeken L, *et al.* (2019) Implementing dietary modifications and assessing nutritional adequacy of diets for inflammatory bowel disease. *Gastroenterol Hepatol (N Y)* **15**, 133.
151. Malik N, Tonstad S, Paalani M, *et al.* (2020) Are long-term FAD diets restricting micronutrient intake? A randomized controlled trial. *Food Sci Nutr* **8**, 6047. <https://doi.org/10.1002/FSN3.1895>.
152. Simons M, Taft TH, Doerfler B, *et al.* (2021) Narrative review: risk of eating disorders and nutritional deficiencies with dietary therapies for irritable bowel syndrome. *Neurogastroenterol Motil* **2021**, e14188. <https://doi.org/10.1111/NMO.14188>.
153. Aljada B, Zohni A & El-Matary W (2021) The gluten-free diet for celiac disease and beyond. *Nutrients* **13**. <https://doi.org/10.3390/NU13113993>.
154. Ma X, Nan F, Liang H, *et al.* (2022) Excessive intake of sugar: an accomplice of inflammation. *Front Immunol* **13**. <https://doi.org/10.3389/fimmu.2022.988481>.
155. Nieman KM, Anderson BD & Cifelli CJ (2021) The effects of dairy product and dairy protein intake on inflammation: a systematic review of the literature. *J Am Coll Nutr* **40**, 571–582. <https://doi.org/10.1080/07315724.2020.1800532>.
156. Smollich M & Tischner L (2022) Corrigendum: patient perceptions about acne, nutrition, and a dietary information gap. *Front Commun (Lausanne)* **7**. <https://doi.org/10.3389/FCOMM.2022.983839>.
157. Kumar S, Han J, Li T, *et al.* (2013) Obesity, waist circumference, weight change and the risk of psoriasis in US women. *J Eur Acad Dermatol Venereol* **27**, 1293–1298. <https://doi.org/10.1111/JDV.12001>.
158. Wolk K, Mallbris L, Larsson P, *et al.* (2009) Excessive body weight and smoking associates with a high risk of onset of plaque psoriasis. *Acta Derm Venereol* **89**, 492–497. <https://doi.org/10.2340/00015555-0711>.
159. Bardazzi F, Balestri R, Baldi E, *et al.* (2010) Correlation between BMI and PASI in patients affected by moderate to severe psoriasis undergoing biological therapy. *Dermatol Ther* **23**. <https://doi.org/10.1111/j.1529-8019.2009.01281.x>.
160. Barrea L, Caprio M, Camajani E, *et al.* (2022) Clinical and nutritional management of very-low-calorie ketogenic diet (VLCKD) in patients with psoriasis and obesity: a practical guide for the nutritionist. *Crit Rev Food Sci Nutr*. <https://doi.org/10.1080/10408398.2022.2083070>.
161. Koelman L, Egea Rodrigues C & Aleksandrova K (2022) Effects of dietary patterns on biomarkers of inflammation and immune responses: a systematic review and meta-analysis





- of randomized controlled trials. *Adv Nutr* **13**, 101–115. <https://doi.org/10.1093/ADVANCES/NMAB086>.
162. Schulze MB & Hoffmann K (2006) Methodological approaches to study dietary patterns in relation to risk of coronary heart disease and stroke. *Br J Nutr* **95**, 860–869. <https://doi.org/10.1079/BJN20061731>.
163. Schulze MB, Martínez-González MA, Fung TT, *et al.* (2018) Food based dietary patterns and chronic disease prevention. *BMJ* **361**. <https://doi.org/10.1136/BMJ.K2396>.
164. Tapsell LC, Neale EP, Satija A, *et al.* Foods, Nutrients, and Dietary Patterns: Interconnections and Implications for Dietary Guidelines 1,2. <https://doi.org/10.3945/an.115.011718>.
165. Jacobs DR, Tapsell LC & Temple NJ (2011) Food synergy: the key to balancing the nutrition research effort. *Public Health Rev* **33**, 507–529. <https://doi.org/10.1007/BF03391648>.
166. AbuMweis S, Abu Omran D, Al-Shami I, *et al.* (2021) The ratio of eicosapentaenoic acid to docosahexaenoic acid as a modulator for the cardio-metabolic effects of omega-3 supplements: a meta-regression of randomized clinical trials. *Complement Ther Med* **57**. <https://doi.org/10.1016/J.CTIM.2021.102662>.
167. Page MJ, Moher D, Bossuyt PM, *et al.* (2021) PRISMA 2020 explanation and elaboration: updated guidance and exemplars for reporting systematic reviews. *BMJ* **372**. <https://doi.org/10.1136/BMJ.N160>.