DUDEK, Stanisław, BĘTKOWSKA, Aleksandra, KOZIAK, Weronika, KORNACKA, Agata, SZOSTAK, Kamila, DUDEK, Wojciech, BYRA, Anna, TOMAKA, Rafał, and MAKIEŁA, Michalina. Targeting Metabolic Syndrome in Psoriasis: A Review of Current Evidence and Non-Pharmacological Treatment Approaches. Quality in Sport. 2025;39:58429. eISSN 2450-3118.
https://dx.doi.org/10.12775/QS.2025.39.58429
https://apcz.umk.pl/QS/article/view/58429

The journal has been 20 points in the Ministry of Higher Education and Science of Poland parametric evaluation. Annex to the announcement of the Minister of Higher Education and Science of 05.01.2024. No. 32553.
Has a Journal's Unique Identifier: 201398. Scientific disciplines assigned: Economics and finance (Field of social sciences); Management and Quality Sciences (Field of social sciences).
Punkty Ministerialne z 2019 - aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 r. Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398.
Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 01.02.2025. Revised: 05.02.2024. Accepted: 05.03.2025. Published: 05.03.2025.

Targeting Metabolic Syndrome in Psoriasis: A Review of Current Evidence and Non-Pharmacological Treatment Approaches

Stanisław Dudek [SD]

University Clinical Center of the Medical University of Warsaw, Banacha 1A, 02-097 Warsaw, Poland

stanislaw.dudek0@wp.pl

https://orcid.org/0009-0002-3641-4865

Aleksandra Bętkowska [AB]

National Medical Institute of the Ministry of the Interior and Administration

Wołoska 137, 02-507 Warsaw, Poland

ola.betkowska@gmail.com

https://orcid.org/0009-0001-5108-4262

Weronika Koziak [WK]
Wolski Hospital Dr. Anna Gostyńska
Marcina Kasprzaka 17, 01-211 Warsaw, Poland
weronikakoziak@icloud.com
https://orcid.org/0009-0003-5295-5765

Agata Kornacka [AK]
University Clinical Center of the Medical University of Warsaw
Banacha 1A, 02-097 Warsaw, Poland
akornackaa13@gmail.com
https://orcid.org/0009-0007-2877-3514

Kamila Szostak [KSz]
The Infant Jesus Teaching Hospital
Lindleya 4, 02-005 Warszawa
kamilaszostak999@gmail.com
https://orcid.org/0009-0005-6216-0864

Wojciech Dudek [WD]
Military University of Technology
2 Kaliskiego Street, 00-908 Warsaw, Poland
wojtek.dudek6@wp.pl
https://orcid.org/0009-0000-2404-9560

Anna Byra [ABy]
Medical University of Silesia in Katowice
Poniatowskiego 15, 40-055 Katowice, Poland
annabyra23@gmail.com
https://orcid.org/0009-0009-7934-5493

Rafał Tomaka [RT]
University Clinical Center of the Medical University of Warsaw
Banacha 1A, 02-097 Warsaw, Poland,
rafaltomaka1998@gmail.com
https://orcid.org/0009-0002-5057-6131

Michalina Makieła [MM]
Wolski Hospital Dr. Anna Gostyńska
Marcina Kasprzaka 17, 01-211 Warsaw, Poland
michalinamakiela@gmail.com
https://orcid.org/0009-0003-4465-8584

Abstract

Introduction: Psoriasis is a chronic inflammatory dermatosis affecting approximately 1.5-5% of the population in developed countries. Besides visible cutaneous manifestations, psoriasis is linked to an increased prevalence of various systemic complications. The term metabolic syndrome refers to a cluster of cardiovascular risk factors, including hypertension, dyslipidemia, obesity, and hyperglycemia, which affect up to 25% of the general population.

Aim of Study: This review examines current and emerging treatments for metabolic syndrome in psoriasis, emphasizing shared pathophysiology, non-pharmacological strategies, and the significance of interdisciplinary care and a holistic approach.

Brief Description of the State of Knowledge: Psoriasis is closely associated with metabolic syndrome, which impacts about one-third of psoriasis patients and exacerbates symptoms through chronic low-grade inflammation and an altered adipokine profile. Lifestyle modification remains the primary therapeutic approach. Emerging treatments, including nutraceuticals like curcumin and cinnamon, show promise in managing metabolic syndrome and potentially improving psoriasis outcomes, though further research is needed.

Conclusions: Diligent screening for metabolic abnormalities, early recognition, and interdisciplinary management, including lifestyle changes, are crucial for addressing cardiometabolic comorbidities and ensuring comprehensive care in patients with psoriasis.

Keywords: metabolic syndrome, psoriasis, obesity, insulin resistance, inflammatory disease, adipocytokines

1. Introduction

Psoriasis is a chronic immune-mediated disease characterized by inflammation and immune dysregulation. It has several subtypes, and although it is best known for causing well-demarcated, erythematous plaques with a distinct silvery-white scale, the abnormalities involved are not limited to the skin (Ryan and Kirby 2015). Pro-inflammatory cytokines such as TNF-α, IL-6, IL-17, and IL-23 play a central role in the cutaneous manifestations of psoriasis and contribute to systemic metabolic disturbances (Campanati et al. 2021). Epidemiological data have shown a strong association between psoriasis and metabolic syndrome, a term that includes factors such as obesity, elevated blood pressure, hyperglycemia, and dyslipidemia, which are associated with an increased risk of developing atherosclerotic cardiovascular disease (Gisondi et al. 2018).

Chronic low-grade inflammation is often seen in patients with metabolic syndrome, one of the most common comorbidities in psoriasis, affecting up to a third of patients. Excessive fat, especially visceral fat, functions as an endocrine organ by releasing pro-inflammatory adipokines like leptin and resistin, which worsen the inflammation (Fahed et al. 2022). This shared inflammatory environment indicates a reciprocal relationship: systemic inflammation in metabolic syndrome can worsen psoriasis, while immune activation in psoriasis can enhance the abnormalities associated with metabolic syndrome, creating a vicious cycle that presents a therapeutic challenge (Coimbra, Catarino, and Santos-Silva 2016).

This narrative review aims to explore current and potential novel treatment approaches for patients with metabolic syndrome and psoriasis, emphasizing shared pathophysiology, non-pharmacological interventions, special considerations during pharmacological treatment, and the necessity of interdisciplinary patient management.

2. Materials and Methods

PubMed and Google Scholar databases were searched using appropriate keywords for accessible studies published until 4 January 2025. Only articles written in English were included. Titles and abstracts were screened first, followed by an evaluation of relevant full-text publications.

3. Discussion

3.1. Psoriasis

3.1.1. Epidemiology

Psoriasis is a chronic autoimmune inflammatory disease affecting the skin, nails, and joints. It is a painful, potentially disfiguring, and often stigmatizing disease affecting people of all ages worldwide. According to the global report published by the World Health Organization (WHO), the prevalence of psoriasis in countries exhibits considerable variation, with figures ranging from 0.09% to 11.4%, with most developed countries reporting the estimated morbidity to be between 1.5% and 5% (Michalek, Loring, and John 2016). However, with information on the epidemiology of psoriasis lacking in over 80% of countries, the exact burden of psoriasis is difficult to measure (Parisi et al. 2020). Its distribution across different geographical regions shows significant disparities, with adults and affluent populations more likely to be affected. Yet, improved reporting is urgently needed from low- and middle-income countries to draw meaningful comparisons (Parisi et al. 2020; Michalek, Loring, and John 2016; Cao et al. 2023). Existing epidemiological analyses show that the age-adjusted incidence rate and disease burden of psoriasis are increasing worldwide, and the number of new cases is expected to continue to rise in the coming years (Hao et al. 2024; Luo 2024).

3.1.2. Basic pathophysiology

The pathogenesis of psoriasis is complex and includes a combination of genetic predisposition, immune dysregulation, and environmental factors. It involves an overproduction of pro-inflammatory cytokines, namely tumor necrosis factor-alpha (TNF-α), interleukins (IL-1, IL-6, IL-17, IL-22, IL-23, IL-36), and interferon-γ (IFN-γ), which impair keratinocyte differentiation and proliferation, promote T-lymphocyte infiltration and recruit other immune cells (e.g. dendritic or NK cells), perpetuating the inflammatory cascade (Kiełbowski et al. 2023; Guo et al. 2023; Nestle, Kaplan, and Barker 2009). Of note, immune cells are not the sole culprits driving the inflammatory process, as keratinocytes respond to T-

cells- and dendritic cells-derived cytokines, releasing pro-inflammatory cytokines and chemokines themselves (Nestle, Kaplan, and Barker 2009). At the same time, the mechanisms supposed to counteract the overly stimulated immune system, namely the function of regulatory T cells and IL-10, have been shown to be dysfunctional in psoriasis (Nestle, Kaplan, and Barker 2009).

Genome-wide association studies have identified an array of variants implicated in psoriasis pathophysiology, many of which are linked to impaired antigen presentation, altered signal transduction pathways, and excess cytokine production (Capon 2017). One such susceptibility gene strongly associated with psoriasis is the HLA-Cw6 allele, which has been observed to affect the disease course, severity, comorbidities, or even treatment response (Chen and Tsai 2018).

Similarly, environmental stressors such as smoking and obesity have been associated with causing flare-ups by generating reactive oxygen species and causing oxidative stress, as well as activating innate immunity mechanisms such as dendritic cells, macrophages, and keratinocytes (Armstrong et al. 2011; Barros et al. 2022). All the above pathophysiological aspects seem to be interlinked and perpetuate the inflammatory process responsible for both cutaneous manifestations and systemic abnormalities.

3.2. Metabolic syndrome

3.2.1. Definition and key components

Metabolic syndrome is a term encapsulating a set of risk factors that contribute to the development of atherosclerotic cardiovascular disease and puts patients at an elevated risk for type 2 diabetes ('Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report' 2002). Several attempts have been made to formulate the criteria for metabolic syndrome, and they have mostly revolved around the same constellation of risk factors. The WHO brought forward the first criteria in 1999, which have since been revised by other expert groups (Consultation 1999). Criteria proposed by the American Heart Association (AHA) and the National Heart, Lung, and Blood Institute (NHLBI) in 2005 are shown in Table 1.

Clinical measure	Cutpoint value
Abdominal obesity	Waist circumference ≥102 cm in men or ≥88 cm in women*
Increased blood pressure	≥130 mm Hg systolic or ≥85 mm Hg diastolic, or currently on drug treatment for hypertension
Elevated fasting glucose	≥100 mg/dL or currently on drug treatment for elevated blood glucose
Elevated triglycerides	≥150 mg/dL or current treatment for elevated triglycerides
Reduced HLD-C	<40 mg/dL in men and <50 mg/dL in women or individuals receiving drug treatment for reduced HDL-C

Table 1. Criteria for the clinical diagnosis of metabolic syndrome proposed by AHA/NHLBI in 2005 (Grundy et al. 2005). To make the diagnosis, a patient should meet 3 out of 5 criteria. HDL-C – high-density lipoprotein cholesterol.
*In 2016, the IDF included lower ethnic-specific cutoff values for the waist circumference criterion.

Abdominal obesity and insulin resistance have been notoriously stressed as the key components of metabolic syndrome (Grundy et al. 2005). However, it has been noted that in patients of certain ethnicities (e.g. of Asian descent), and those suffering from other comorbidities linked to insulin resistance (e.g. polycystic ovary syndrome, fatty liver disease) may present signs of metabolic syndrome in the absence of excessive abdominal fat and in these patients, a lower cutoff value of the waist circumference criterion should be considered (i.e. ≥94 cm in men and ≥80 cm in women) (Jiang et al. 2018; Pratyush et al. 2012; Enkhmaa et al. 2005; Oh et al. 2010). In 2006, the International Diabetes Federation (IDF) published a revised definition of metabolic syndrome, whereby to make the clinical diagnosis, a person must exhibit central obesity (with ethnic-specific cutoff values for waist circumference) as well as any two of the remaining four criteria presented in previous definitions (Alberti, Zimmet, and Shaw 2006).

Nevertheless, though sometimes recognized as an inherent constituent of metabolic syndrome, obesity may not be present in all cases. Some patients with normal body weight index (BMI) are still at an increased metabolic risk, presenting metabolic disturbances typically

associated with obesity. This can be due to a variety of factors, namely increased visceral fat deposition, low muscle mass, or lack of physical activity. These observations have led to the distinction of a phenotype called 'metabolically obese, normal-weight' (MONW), a term coined in 1981 (Ding, Chan, and Magkos 2016; Ruderman, Schneider, and Berchtold 1981; Conus, Rabasa-Lhoret, and Péronnet 2007). This patient population can be particularly challenging to identify early on, resulting in delayed interventions (Ding, Chan, and Magkos 2016).

3.2.2. Epidemiology

Approximately 20-25% of adults worldwide are estimated to have metabolic syndrome (Alberti, Zimmet, and Shaw 2006). However, the lack of a unified definition of metabolic syndrome across studies makes it difficult to determine the exact burden. An Iranian study by Delavari et al highlighted the discrepancies in reported rates depending on the criteria used with the age-standardized prevalence of metabolic syndrome ranging from 34,7% to 41,6% (Delavari et al. 2009).

3.2.3. Changing Views on Pathophysiology

Insulin resistance was initially considered the primary culprit in patients with metabolic syndrome. However, in recent years, the focus has largely shifted toward the chronic inflammatory response as the underlying pathophysiological process causing detrimental effects on tissues and whole organ systems. This new understanding has shed some light on the role played by cytokines, chemokines, and adipokines in metabolic syndrome and might inspire novel, targeted therapeutic approaches (Montecucco, Mach, and Pende 2013).

3.3. Link Between Psoriasis and Metabolic Syndrome

There is robust evidence to suggest an increased risk of developing metabolic syndrome among patients with psoriasis. A systematic review with meta-analysis by Choudhary et al. included 63 observational studies and found that the prevalence of metabolic syndrome in patients with psoriasis was as high as 30.29%, more than twice that of controls (Saumya et al. 2020). High prevalence rates of metabolic syndrome have also been reported in children with psoriasis, highlighting the need for clinical vigilance and regular risk assessment, even in the youngest patients (Caroppo et al. 2021; Aalemi, Hamdard, and Chen 2020). However, the exact nature of the association between psoriasis and metabolic syndrome is still not fully understood, and more research is necessary to establish the processes underlying this complex relationship.

3.3.1. Shared Inflammatory Pathways and the Role of Adipokines

Systemic inflammation has been postulated as the major culprit in both conditions. Psoriasis is an immune-mediated disease with a pathophysiology characterized by an interplay of pro-inflammatory cytokines and various cell types, mostly, but not limited to, immune cells, as already mentioned. TNF-α, interferons, IL-1, IL-6, IL-17, IL-22, and IL-23, among other pro-inflammatory molecules, seem to be the main cytokines involved in the development of the disease (Nestle, Kaplan, and Barker 2009). Similarly, the pathophysiological basis for metabolic syndrome is multifactorial and has not yet been fully elucidated, yet persistent inflammation seems to play a critical role, along with insulin resistance and neurohormonal activation. These pathophysiological mechanisms often intersect to form vicious cycles affecting multiple organ systems simultaneously (Fahed et al. 2022). The pro-inflammatory state appears to be a culmination of those various pathways with increased release of markers such as IL-1, IL-6, IL-8, TNF-α, CRP, and fibrinogen, among others (Fahed et al. 2022). Additionally, comparisons have been made between skin plaques in psoriasis and atheromatous plaques in blood vessels, which seem to involve similar immune cell infiltrates and pro-inflammatory cytokine profiles (Ryan and Kirby 2015).

Adipose tissue is now widely regarded as more than a simple energy storage, serving as an endocrine organ that secretes active substances into the bloodstream. Interestingly, adipocytes are also involved in various local physiological processes, since studies in mice have shown that intradermal adipocytes play a role in hair regeneration by regulating the activation of follicular stem cells and in skin wound healing through recruiting fibroblasts in the proliferative phase of healing (Schmidt and Horsley 2013; Festa et al. 2011).

In obese patients, excess adipose tissue, in particular the accumulation of visceral fat, leads to a chronic low-grade inflammatory state driven by the release of proinflammatory mediators. Among them are adipokines (also known as adipocytokines), including, but not limited to, leptin, resistin, and adiponectin. These are cell-signaling molecules secreted by the adipose tissue in both healthy individuals and in various disease processes (Clemente-Suárez et al. 2023). The general trends in adipokine expression in obesity are summarized in Table 2.

Leptin, which is the first discovered and one of the most extensively studied hormones secreted by the adipose tissue, shows a substantial positive correlation with body fat percentage and serum leptin concentrations have been shown to decrease with weight loss (Considine et al. 1996; Wong et al. 2019). In healthy subjects, it acts by increasing energy expenditure and regulating food intake, thus leading to a reduction in body weight, and animal studies have also

suggested its independent positive effect on glucose homeostasis and liver insulin sensitivity (Li 2011; Berglund et al. 2012; Huo et al. 2009). However, this mechanism is impaired in obese individuals, a phenomenon termed “leptin resistance”, and the molecule has also been demonstrated to propagate inflammatory response by activation of Th1 and suppression of the Th2 immune pathways (Lord et al. 1998; Fahed et al. 2022).

Observed changes in serum levels in obesity	**Adipokines**
Increase	Leptin Resistin Chemerin Visfatin TNF-α IL-1β IL-6 Retinol-binding protein 4 Fetuin-A Lipocalin-2
Decrease	Adiponectin Omentin-1

Table 2. Changes in adipokine profile associated with obesity. Excess adiposity generally results in an over-production of pro-inflammatory and reduced production of anti-inflammatory adipokines (Kong et al. 2019). Similar alterations have been observed in psoriasis (Coimbra, Catarino, and Santos-Silva 2016).

Previously, the endocrine role has been attributed mainly to white adipose tissue. In recent years, however, brown adipose tissue (BAT) has emerged as having similar endocrine properties, secreting signaling molecules (batokines) that affect multiple organs, including the cardiovascular system, and provide protection against obesity and associated metabolic abnormalities (Martins et al. 2023; Villarroya et al. 2019; Zhang et al. 2024; Villarroya, Cereijo, and Villarroya 2013).

Disturbances to the production of adipokines lead to alterations in metabolism and inflammation, can affect mood, cognition, and behavior, and are increasingly implicated in tumorigenesis (Sahu and Bal 2023; Booth et al. 2015; Christodoulatos et al. 2019). This imbalance promotes the recruitment of immune cells, such as macrophages, into adipose tissue, amplifying local and systemic inflammation through a feedback loop involving cytokines such as TNF-α, IL-1, and IL-6, thereby creating a vicious cycle. The resulting persistent inflammation contributes to metabolic syndrome, cardiovascular disease, and other cardio-metabolic complications (Kyrou et al. 2017).

Increasing evidence links dysregulation of the adipokine profile to the development and aggravation of the pathophysiological processes underlying psoriasis. As mentioned above, leptin exhibits pro-inflammatory properties, which have also been linked to the exacerbation of psoriatic lesions (Kyriakou et al. 2017). In vitro studies have demonstrated that leptin stimulates the production of IL-6, CXCL-1, IL-8, and monocyte chemoattractant protein 1 (MCP-1), while also enhancing the expression of intercellular adhesion molecule-1 (ICAM-1) in dermal fibroblasts (Ommen et al. 2016). This influence is not limited to fibroblasts, as another in vitro study reported leptin-induced proliferation and overproduction of inflammatory molecules by keratinocytes (Xue et al. 2013). A meta-analysis of 26 observational studies revealed that patients with psoriasis exhibited elevated levels of leptin and resistin, along with reduced adiponectin concentrations, similar to patterns observed in metabolic syndrome. However, the data analyzed demonstrated significant heterogeneity (Kyriakou et al. 2017).

As expected, the exact relationship between imbalances in the adipokine profile and psoriasis disease severity is still debated (Coimbra, Catarino, and Santos-Silva 2016). The alterations of adipokines in psoriasis appear to be influenced by both obesity and inflammation, with adipocyte-derived leptin levels linked more strongly to body fat content and BMI. In contrast, pro-inflammatory cytokines like IL-6 and TNF-α appear to be associated with disease severity regardless of weight. It is possible that adipokines produced by adipose tissue, even in small amounts, may exacerbate the disease by influencing the inflammatory processes (Coimbra, Catarino, and Santos-Silva 2016).

3.4. Screening

Considering the increased risk of developing metabolic syndrome, patients with psoriasis need to undergo regular clinical assessments to ensure early detection and guide efficient interventions. Joint guidelines issued by the American Academy of Dermatology and

the National Psoriasis Foundation suggest that all psoriasis patients should undergo regular risk assessment for cardiovascular comorbidities. They advise early and more frequent screening for diabetes, hyperlipidemia, and hypertension among patients with involvement of >10% BSA, or those who are to receive systemic treatment or phototherapy. When calculating cardiovascular risk in patients with such disease severity, the experts advise multiplying the estimated risk by 1.5 (Elmets et al. 2019).

Research suggests that simple anthropometric measurements, such as weight, body mass index, or waist-hip ratio, or basic biochemical markers (i.e. fasting blood glucose, lipid profile) may not be the optimal tool to assess patients' nutritional status (Mohamed Haris et al. 2023). Additional tests and measurements should be considered in this patient group, such as serum vitamin D, HbA1c, and apolipoprotein profiles (Ramezani, Zavattaro, and Sadeghi 2019; Gamonal et al. 2022; Lee and Song 2018) In their review paper, Haris et al emphasized the role of dietary assessments conducted by trained dietitians or researchers to better evaluate patients' dietary intake. This can be achieved with the help of tools such as food frequency questionnaires/food records spanning a period of a few days up to months, or even simple open-ended questions inquiring about dietary habits (Mohamed Haris et al. 2023).

In recent years, new imaging modalities for body composition assessment have been introduced into clinical practice, with methods such as computed tomography, bioimpedance analysis, and dual-energy X-ray absorptiometry becoming increasingly accessible to assist in evaluating patients with psoriasis (Blake et al. 2020).

In addition, patients with psoriasis are at higher risk of autoimmune thyroid diseases, particularly hypothyroidism, with its metabolic abnormalities (Zhang et al. 2022). Awareness of this risk by the healthcare team is important, but, at the same time, most cases of thyroid dysfunction are subclinical. To date, there are no consensus guidelines on which psoriasis patients should undergo screening for endocrine disorders (Cira et al. 2023).

3.5. Treatment

3.6. Lifestyle Modifications

The primary focus in managing patients with metabolic syndrome is to address modifiable risk factors such as obesity, lack of physical exercise, poor nutrition, or smoking. This can be achieved through lifestyle modification counseling, which should be recommended to all patients. For patients at particularly high risk of cardiovascular complications,

pharmacological treatment may be considered upon diagnosis alongside appropriate lifestyle changes (Grundy et al. 2005).

This approach is by far the safest and most cost-effective, and available research has consistently emphasized the principal role of lifestyle interventions as the most effective treatment method in maintaining or restoring metabolic health (Guzmán et al. 2019; Dunkley et al. 2012; Kim et al. 2022).

3.6.1. Exercise

The American Heart Association (AHA) recommends at least 30-60 minutes of moderate-intensity continuous or intermittent aerobic activity regularly, 5 days a week, but preferably daily. In addition, the authors suggest incorporating strength training twice a week and advise medically supervised programs for patients at particularly high cardiovascular risk (Grundy et al. 2005). The European Society of Cardiology (ESC) similarly recommends that adults of all ages engage in 150 to 300 minutes of moderate-intensity aerobic exercise per week, or 75 to 150 minutes of vigorous exercise, or a combination of both (Visseren et al. 2021). Walking seems to be one of the most cost-effective activities for patients with psoriasis, as it does not require costly equipment, has a relatively low risk of injury, and can be easily adapted for groups of patients, which can boost motivation and help maintain adherence to the training program (Sheppard et al. 2022).

Apart from promoting cardiorespiratory fitness, in patients with psoriasis, physical exercise has been associated with a reduction in Psoriasis Area and Severity Index (PASI) scores, improved Dermatology Life Quality Index (DLQI) scores, and positive psychological impact (Sheppard et al. 2024; Auker et al. 2022).

However, studies have shown that the majority of patients with plaque psoriasis report spending less than the recommended amount of time on exercise, often for disease-specific reasons, such as the severity of skin lesions, skin sensitivity, clothing preferences, and treatment (Auker et al. 2020; Do et al. 2015). What is more, it was observed that patients with psoriasis exhibited abnormal thermoregulatory responses in heat exercise test, and postulated that they might be more prone to suffering from heat intolerance during physical activity (Leibowitz et al. 1991).

3.6.2. Diet

AHA guidelines in metabolic syndrome concerning dietary changes do not point towards any particular diet but recommend limiting saturated fat to less than 7% of total calorie intake, and trans fats and cholesterol to a minimum. Total fat should make up 25% to 35% of daily calories, with an emphasis on unsaturated fats, while the intake of simple sugars should be kept low (Grundy et al. 2005).

In terms of specific dietary recommendations, according to the ESC, the Mediterranean or similar diet can be recommended to all patients to reduce their risk of developing cardiovascular disease (Visseren et al. 2021; McEvoy et al. 2024). In 2011, Kastorini and colleagues published a systematic review and meta-analysis of the effects of the Mediterranean diet on metabolic syndrome. They included more than 530,000 patients from 50 studies, and found that the Mediterranean diet had a positive effect on all components of metabolic syndrome, leading to a reduction in waist circumference, triglyceride levels, systolic and diastolic blood pressure, glucose levels, and an increase in HDL cholesterol levels. It also proved to be effective in primary prevention of metabolic syndrome (Kastorini et al. 2011).

The Dietary Approaches to Stop Hypertension, or DASH, diet, originally designed for patients diagnosed with hypertension, has recently also been suggested as favorable for patients with other conditions, including metabolic syndrome. It has been shown to help reduce body weight and blood pressure, though reported effects on blood glucose, insulin resistance, and lipid profile vary between studies (Valenzuela-Fuenzalida et al. 2024; Lari et al. 2021; Lien et al. 2007; Sacks et al. 1995).

Another dietary approach that has recently gained interest is intermittent fasting (IF). In a small meta-analysis including four studies with a total of 355 participants, Wang et al. showed that in patients with metabolic syndrome or type 2 diabetes, IF has similar beneficial effects on glycaemic control and lipid profile and a greater effect on body weight reduction compared with continuous, calorie-restriction diets, as well as a good safety profile (Wang et al. 2021). Positive results were also reported by Moon et al., who observed that time-restricted eating, a variant of IF that involves food intake in coordination with the circadian rhythm, had beneficial effects on blood pressure, fasting blood glucose, and triglyceride levels in overweight and obese patients, further suggesting that IF may be a feasible dietary strategy for patients with metabolic disturbances (Moon et al. 2020).

3.6.3. Weight Reduction

The AHA recommends aiming for a 7-10% reduction in body weight during the first year of treatment, with continued weight loss towards a BMI of less than 25 kg/m². Long-term weight management should be promoted through a balanced approach of physical activity, calorie control, and behavior modification programs, with a goal of keeping a waist circumference of less than 102 cm for men and 88 cm for women (Grundy et al. 2005).

3.6.4. Nutraceuticals

The popularity of so-called nutraceuticals - substances derived from food believed to have health benefits - has grown exponentially in recent years (Ronis, Pedersen, and Watt 2018). Several have been proposed as potentially useful in patients with metabolic syndrome.

One such compound is curcumin, an active ingredient found in turmeric that has long been used in traditional medicine for a range of conditions, mostly inflammatory (Akaberi, Sahebkar, and Emami 2021). Studies have shown that curcumin can act on all metabolic syndrome components by increasing insulin secretion, stimulating the uptake of fatty acids, decreasing lipogenesis, and lowering blood pressure through increased nitric oxide production (Vafaeipour, Razavi, and Hosseinzadeh 2022). However, more research is warranted to elucidate the exact mechanism of action of curcumin in patients with metabolic syndrome.

In 2023, Qiu et al conducted a systematic review with meta-analysis of 13 randomized controlled trials to assess the metabolic, anti-inflammatory, and antioxidative effects of curcumin in metabolic syndrome. They found that curcumin had a positive impact on waist circumference, fasting blood glucose, diastolic blood pressure, and HDL-C levels, and was associated with a decrease in some inflammatory markers (Qiu et al. 2023). However, the doses of curcumin administered varied widely between trials, and the relatively small number of patients included in the analysis meant that the authors were unable to draw meaningful conclusions about the beneficial dose of the phytochemical (Qiu et al. 2023).

A few studies have also demonstrated the potential positive effects of curcumin on psoriasis (Ramírez-Boscá et al. 2017; Antiga et al. 2015; Bilia et al. 2018). A randomized controlled study by Antiga et al found that curcumin can act as an adjuvant in patients with mild-to-moderate psoriasis treated with topical steroids. Apart from a greater reduction in the PASI score at follow-up, the group treated with oral curcumin and steroids showed significantly reduced serum levels of IL-22 (Antiga et al. 2015). Another study found that oral curcumin in the form of nanoparticles was an effective adjuvant therapy in patients with moderate to severe

psoriasis who were treated with oral acitretin and also improved their lipid profile, highlighting its potential use in patients at risk of metabolic disorders (Bilia et al. 2018).

Cinnamon is another commonly used spice purported to provide multiple health benefits. It has been reported to act as an anti-inflammatory, antioxidative, and antidiabetic agent (Mohsin et al. 2023). Like most nutraceuticals, cinnamon contains several bioactive compounds. The water-soluble polyphenols are the component that appears to be primarily responsible for the hypoglycaemic effect by enhancing autophosphorylation (and thus activation) of the insulin receptor, in addition to enhancing glucose uptake and utilization (Cao, Polansky, and Anderson 2007).

Of note, cinnamon has relatively robust evidence to justify its use by patients with metabolic syndrome. A review by Mockonochie et al analyzed 41 randomized studies looking at its effects on the components of metabolic syndrome. They found beneficial effects on glucose levels, as well as insulin sensitivity, though these effects were more likely to be seen in non-diabetic patients. No apparent correlation was found between the positive effects of cinnamon and its dose (Mackonochie et al. 2023). One of the trials included in the above meta-analysis, involving 116 patients with metabolic syndrome, showed, in addition to the efficacy of cinnamon in improving glycaemic control and insulin sensitivity, significantly greater reductions in body weight, waist circumference, body fat percentage, improved lipid profile, and blood pressure (Gupta Jain et al. 2017).

To our knowledge, no clinical studies have investigated the role of cinnamon in patients with psoriasis. However, in-vitro studies have demonstrated that cinnamaldehyde, one of the active compounds present in cinnamon, may protect keratinocytes from inflammatory injury, inhibit proliferation, and promote keratinocyte differentiation (Ding et al. 2021).

Multiple other potentially beneficial substances of natural origin have been identified due to the growing interest in alternative medicine among both researchers and patients. Examples of spices and herbs that are said to have metabolic health properties include *Nigella sativa, Hibiscus sabdariffa, Lagenaria siceraria, Trigonella foenum-graecum, Emblica officinalis, Vigna mungo, Camellia sinensis (Kaur et al. 2015; Hallajzadeh et al. 2020)*.

Nevertheless, it is important to remind patients that nutraceuticals and other dietary supplements are not subject to the same stringent registration and approval policies as drugs before entering the market and are by no means free of adverse effects or drug interactions (Ronis, Pedersen, and Watt 2018).

4. Conclusions

Psoriasis is a chronic, immune-mediated disease causing significant physical and psychological distress. It shares underlying pathophysiological pathways with several comorbidities, including metabolic syndrome. An estimated one third of psoriasis patients may have metabolic syndrome, which is associated with chronic low-grade inflammation and contributes to the worsening of psoriasis symptoms. Early recognition is key, and current evidence supports the need for routine vigilant assessment of patients with psoriasis for cardiometabolic comorbidities, as well as appropriate, individualized management of already diagnosed concomitant diseases. Interdisciplinary collaboration, particularly with clinical nutritionists, cardiologists, diabetologists, and endocrinologists, is strongly encouraged in the management of metabolic syndrome in psoriasis patients to ensure a holistic approach. Lifestyle modification remains the central therapeutic approach.

5. Disclosure

Author's Contribution

Conceptualization: SD, AB; methodology: SD, AB; software: n/a; check: SD, AB, WK; formal analysis: AB, WK; investigation: SD, AB, WK, AK, KSz; resources: ABy; data curation: SD, AB, WK, AK, KSz, WD, ABy, RT, MM; writing - rough preparation: SD, AB, WK; writing - review and editing: SD, AB, WK, AK, KSz, WD, ABy, RT, MM; visualization: SD, AB, AK; supervision: WD, ABy, RT; project administration: SD; receiving funding: n/a.

All authors have read and agreed with the published version of the manuscript.

Funding Statement

The authors did not receive special funding.

Institutional Review Board Statement

Not applicable.

Informed Consent Statement

Not applicable.

Data Availability Statement

Not applicable.

Conflict of Interest Statement

The authors declare no conflict of interest.

References

Aalemi, A. K., A. G. Hamdard, and H. Chen. 2020. 'Association of metabolic syndrome with pediatric psoriasis: a case-control study in Kabul, Afghanistan', *Int J Dermatol*, 59: 451-56.

Akaberi, M., A. Sahebkar, and S. A. Emami. 2021. 'Turmeric and Curcumin: From Traditional to Modern Medicine', *Adv Exp Med Biol*, 1291: 15-39.

Alberti, K. G., P. Zimmet, and J. Shaw. 2006. 'Metabolic syndrome--a new world-wide definition. A Consensus Statement from the International Diabetes Federation', *Diabet Med*, 23: 469-80.

Antiga, E., V. Bonciolini, W. Volpi, E. Del Bianco, and M. Caproni. 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: 283634.

Armstrong, A. W., E. J. Armstrong, E. N. Fuller, M. E. Sockolov, and S. V. Voyles. 2011. 'Smoking and pathogenesis of psoriasis: a review of oxidative, inflammatory and genetic mechanisms', *Br J Dermatol*, 165: 1162-8.

Auker, L., L. Cordingley, C. E. M. Griffiths, and H. S. Young. 2022. 'Physical activity is important for cardiovascular health and cardiorespiratory fitness in patients with psoriasis', *Clin Exp Dermatol*, 47: 289-96.

Auker, L., L. Cordingley, S. R. Pye, C. E. M. Griffiths, and H. S. Young. 2020. 'What are the barriers to physical activity in patients with chronic plaque psoriasis?', *Br J Dermatol*, 183: 1094-102.

Barros, G., P. Duran, I. Vera, and V. Bermúdez. 2022. 'Exploring the Links between Obesity and Psoriasis: A Comprehensive Review', *Int J Mol Sci*, 23.

Berglund, E. D., C. R. Vianna, J. Donato, Jr., M. H. Kim, J. C. Chuang, C. E. Lee, D. A. Lauzon, P. Lin, L. J. Brule, M. M. Scott, R. Coppari, and J. K. Elmquist. 2012. 'Direct leptin action on POMC neurons regulates glucose homeostasis and hepatic insulin sensitivity in mice', *J Clin Invest*, 122: 1000-9.

Bilia, A. R., M. C. Bergonzi, B. Isacchi, E. Antiga, and M. Caproni. 2018. 'Curcumin nanoparticles potentiate therapeutic effectiveness of acitrein in moderate-to-severe psoriasis patients and control serum cholesterol levels', *J Pharm Pharmacol*, 70: 919-28.

Blake, Tim, Nicola J Gullick, Charles E Hutchinson, and Thomas M Barber. 2020. 'Psoriatic disease and body composition: A systematic review and narrative synthesis', *PloS one*, 15: e0237598.

Booth, A., A. Magnuson, J. Fouts, and M. Foster. 2015. 'Adipose tissue, obesity and adipokines: role in cancer promotion', *Horm Mol Biol Clin Investig*, 21: 57-74.

Campanati, A., A. Marani, E. Martina, F. Diotallevi, G. Radi, and A. Offidani. 2021. 'Psoriasis as an Immune-Mediated and Inflammatory Systemic Disease: From Pathophysiology to Novel Therapeutic Approaches', *Biomedicines*, 9.

Cao, F., Y. S. He, Y. Wang, C. K. Zha, J. M. Lu, L. M. Tao, Z. X. Jiang, and H. F. Pan. 2023. 'Global burden and cross-country inequalities in autoimmune diseases from 1990 to 2019', *Autoimmun Rev*, 22: 103326.

Cao, H., M. M. Polansky, and R. A. Anderson. 2007. 'Cinnamon extract and polyphenols affect the expression of tristetraprolin, insulin receptor, and glucose transporter 4 in mouse 3T3-L1 adipocytes', *Arch Biochem Biophys*, 459: 214-22.

Capon, Francesca. 2017. 'The genetic basis of psoriasis', *International Journal of Molecular Sciences*, 18: 2526.

Caroppo, Francesca, Alfonso Galderisi, Laura Ventura, and Anna Belloni Fortina. 2021. 'Metabolic syndrome and insulin resistance in pre-pubertal children with psoriasis', *European Journal of Pediatrics*, 180: 1739-45.

Chen, L., and T. F. Tsai. 2018. 'HLA-Cw6 and psoriasis', *Br J Dermatol*, 178: 854-62.

Christodoulatos, G. S., N. Spyrou, J. Kadillari, S. Psallida, and M. Dalamaga. 2019. 'The Role of Adipokines in Breast Cancer: Current Evidence and Perspectives', *Curr Obes Rep*, 8: 413-33.

Cira, Cristina-Ilinca, Mara Carsote, Claudiu Nistor, Aida Petca, Razvan Cosmin Petca, and Florica Sandru. 2023. 'Conundrum for Psoriasis and Thyroid Involvement', *International Journal of Molecular Sciences*, 24: 4894.

Clemente-Suárez, V. J., L. Redondo-Flórez, A. I. Beltrán-Velasco, A. Martín-Rodríguez, I. Martínez-Guardado, E. Navarro-Jiménez, C. C. Laborde-Cárdenas, and J. F. Tornero-Aguilera. 2023. 'The Role of Adipokines in Health and Disease', *Biomedicines*, 11.

Coimbra, S., C. Catarino, and A. Santos-Silva. 2016. 'The triad psoriasis-obesity-adipokine profile', *J Eur Acad Dermatol Venereol*, 30: 1876-85.

Considine, R. V., M. K. Sinha, M. L. Heiman, A. Kriauciunas, T. W. Stephens, M. R. Nyce, J. P. Ohannesian, C. C. Marco, L. J. McKee, T. L. Bauer, and et al. 1996. 'Serum immunoreactive-leptin concentrations in normal-weight and obese humans', *N Engl J Med*, 334: 292-5.

Consultation, WHO. 1999. "Definition, diagnosis and classification of diabetes mellitus and its complications." In.: Part.

Conus, F., R. Rabasa-Lhoret, and F. Péronnet. 2007. 'Characteristics of metabolically obese normal-weight (MONW) subjects', *Appl Physiol Nutr Metab*, 32: 4-12.

Delavari, A., M. H. Forouzanfar, S. Alikhani, A. Sharifian, and R. Kelishadi. 2009. 'First nationwide study of the prevalence of the metabolic syndrome and optimal cutoff points of waist circumference in the Middle East: the national survey of risk factors for noncommunicable diseases of Iran', *Diabetes Care*, 32: 1092-7.

Ding, Cherlyn, Zhiling Chan, and Faidon Magkos. 2016. 'Lean, but not healthy: the 'metabolically obese, normal-weight' phenotype', *Current Opinion in Clinical Nutrition & Metabolic Care*, 19.

Ding, Z., J. Liu, H. Qian, L. Wu, and M. Lv. 2021. 'Cinnamaldehyde inhibits psoriasis-like inflammation by suppressing proliferation and inflammatory response of keratinocytes via inhibition of NF-κB and JNK signaling pathways', *Mol Med Rep*, 24.

Do, Y. K., N. Lakhani, R. Malhotra, B. Halstater, C. Theng, and T. Østbye. 2015. 'Association between psoriasis and leisure-time physical activity: findings from the National Health and Nutrition Examination Survey', *J Dermatol*, 42: 148-53.

Dunkley, A. J., K. Charles, L. J. Gray, J. Camosso-Stefinovic, M. J. Davies, and K. Khunti. 2012. 'Effectiveness of interventions for reducing diabetes and cardiovascular disease risk in people with metabolic syndrome: systematic review and mixed treatment comparison meta-analysis', *Diabetes Obes Metab*, 14: 616-25.

Elmets, Craig A., Craig L. Leonardi, Dawn M. R. Davis, Joel M. Gelfand, Jason Lichten, Nehal N. Mehta, April W. Armstrong, Cody Connor, Kelly M. Cordoro, Boni E. Elewski, Kenneth B. Gordon, Alice B. Gottlieb, Daniel H. Kaplan, Arthur Kavanaugh, Dario Kivelevitch, Matthew Kiselica, Neil J. Korman, Daniela Kroshinsky, Mark Lebwohl, Henry W. Lim, Amy S. Paller, Sylvia L. Parra, Arun L. Pathy, Elizabeth Farley Prater, Reena Rupani, Michael Siegel, Benjamin Stoff, Bruce E. Strober, Emily B. Wong, Jashin J. Wu, Vidhya Hariharan, and Alan Menter. 2019. 'Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with awareness and attention to comorbidities', *Journal of the American Academy of Dermatology*, 80: 1073-113.

Enkhmaa, B., K. Shiwaku, E. Anuurad, A. Nogi, K. Kitajima, M. Yamasaki, T. Oyunsuren, and Y. Yamane. 2005. 'Prevalence of the metabolic syndrome using the Third Report of the National Cholesterol Educational Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (ATP III) and the modified ATP III definitions for Japanese and Mongolians', *Clin Chim Acta*, 352: 105-13.

Fahed, G., L. Aoun, M. Bou Zerdan, S. Allam, M. Bou Zerdan, Y. Bouferraa, and H. I. Assi. 2022. 'Metabolic Syndrome: Updates on Pathophysiology and Management in 2021', *Int J Mol Sci*, 23.

Festa, E., J. Fretz, R. Berry, B. Schmidt, M. Rodeheffer, M. Horowitz, and V. Horsley. 2011. 'Adipocyte lineage cells contribute to the skin stem cell niche to drive hair cycling', *Cell*, 146: 761-71.

Gamonal, S. B. L., A. C. C. Gamonal, N. C. V. Marques, M. A. F. Brandão, and N. R. B. Raposo. 2022. '25-Hydroxyvitamin D as a biomarker of vitamin D status in plaque psoriasis and other dermatological diseases: a cross-sectional study', *Sao Paulo Med J*, 141: 131-37.

Gisondi, P., A. C. Fostini, I. Fossà, G. Girolomoni, and G. Targher. 2018. 'Psoriasis and the metabolic syndrome', *Clin Dermatol*, 36: 21-28.

Grundy, Scott M., James I. Cleeman, Stephen R. Daniels, Karen A. Donato, Robert H. Eckel, Barry A. Franklin, David J. Gordon, Ronald M. Krauss, Peter J. Savage, Sidney C. Smith, John A. Spertus, and Fernando Costa. 2005. 'Diagnosis and Management of the Metabolic Syndrome', *Circulation*, 112: 2735-52.

Guo, J., H. Zhang, W. Lin, L. Lu, J. Su, and X. Chen. 2023. 'Signaling pathways and targeted therapies for psoriasis', *Signal Transduct Target Ther*, 8: 437.

Gupta Jain, S., S. Puri, A. Misra, S. Gulati, and K. Mani. 2017. 'Effect of oral cinnamon intervention on metabolic profile and body composition of Asian Indians with metabolic syndrome: a randomized double -blind control trial', *Lipids Health Dis*, 16: 113.

Guzmán, A., E. Navarro, L. Obando, J. Pacheco, K. Quirós, L. Vásquez, M. Castro, and F. Ramírez. 2019. 'Effectiveness of interventions for the reversal of a metabolic syndrome diagnosis: An update of a meta-analysis of mixed treatment comparison studies', *Biomedica*, 39: 647-62.

Hallajzadeh, J., A. Milajerdi, M. Mobini, E. Amirani, S. Azizi, E. Nikkhah, B. Bahadori, R. Sheikhsoleimani, and S. M. Mirhashemi. 2020. 'Effects of Nigella sativa on glycemic control, lipid profiles, and biomarkers of inflammatory and oxidative stress: A systematic review and meta-analysis of randomized controlled clinical trials', *Phytother Res*, 34: 2586-608.

Hao, C., L. Ting, G. Feng, X. Jing, H. Ming, L. Yang, Z. Jie, and J. Yin. 2024. 'Global incidence trends of autoimmune diseases from 1990 to 2021 and projections to 2050: A systemic analysis of the global burden of disease study 2021', *Autoimmun Rev*: 103621.

Huo, L., K. Gamber, S. Greeley, J. Silva, N. Huntoon, X. H. Leng, and C. Bjørbaek. 2009. 'Leptin-dependent control of glucose balance and locomotor activity by POMC neurons', *Cell Metab*, 9: 537-47.

Jiang, B., T. Gu, K. Zhou, Y. Zheng, Y. Guo, and Y. Lu. 2018. 'Fatty Liver as a Potential Surrogate for Waist Circumference in the Diagnosis of Metabolic Syndrome: A Population-Based Study among Chinese Adults', *Int J Endocrinol*, 2018: 7903982.

Kastorini, Christina-Maria, Haralampos J. Milionis, Katherine Esposito, Dario Giugliano, John A. Goudevenos, and Demosthenes B. Panagiotakos. 2011. 'The Effect of Mediterranean Diet on Metabolic Syndrome and its Components: A Meta-Analysis of 50 Studies and 534,906 Individuals', *Journal of the American College of Cardiology*, 57: 1299-313.

Kaur, Ginpreet, Simi Mukundan, Vaidehi Wani, and Maushmi S Kumar. 2015. 'Nutraceuticals in the management and prevention of metabolic syndrome', *Austin J Pharmacol Ther*, 3: 1063.

Kiełbowski, K., E. Bakinowska, P. Ostrowski, B. Pala, E. Gromowska, K. Gurazda, P. Dec, A. Modrzejewski, and A. Pawlik. 2023. 'The Role of Adipokines in the Pathogenesis of Psoriasis', *Int J Mol Sci*, 24.

Kim, H. L., J. Chung, K. J. Kim, H. J. Kim, W. W. Seo, K. H. Jeon, I. Cho, J. J. Park, M. H. Lee, J. Suh, S. Y. Lim, S. Choi, and S. H. Kim. 2022. 'Lifestyle Modification in the Management of Metabolic Syndrome: Statement From Korean Society of CardioMetabolic Syndrome (KSCMS)', *Korean Circ J*, 52: 93-109.

Kong, Y., S. Zhang, R. Wu, X. Su, D. Peng, M. Zhao, and Y. Su. 2019. 'New insights into different adipokines in linking the pathophysiology of obesity and psoriasis', *Lipids Health Dis*, 18: 171.

Kyriakou, A., A. Patsatsi, D. Sotiriadis, and D. G. Goulis. 2017. 'Serum Leptin, Resistin, and Adiponectin Concentrations in Psoriasis: A Meta-Analysis of Observational Studies', *Dermatology*, 233: 378-89.

Kyrou, I., H. S. Mattu, K. Chatha, and H. S. Randeva. 2017. 'Chapter 7 - Fat Hormones, Adipokines.' in Jonathan C. Schisler, Charles H. Lang and Monte S. Willis (eds.), *Endocrinology of the Heart in Health and Disease* (Academic Press).

Lari, A., M. H. Sohouli, S. Fatahi, H. S. Cerqueira, H. O. Santos, B. Pourrajab, M. Rezaei, S. Saneie, and S. T. Rahideh. 2021. 'The effects of the Dietary Approaches to Stop Hypertension (DASH) diet on metabolic risk factors in patients with chronic disease: A systematic review and meta-analysis of randomized controlled trials', *Nutr Metab Cardiovasc Dis*, 31: 2766-78.

Lee, Y. H., and G. G. Song. 2018. 'Association between circulating 25-hydroxyvitamin D levels and psoriasis, and correlation with disease severity: a meta-analysis', *Clin Exp Dermatol*, 43: 529-35.

Leibowitz, E., D. S. Seidman, A. Laor, Y. Shapiro, and Y. Epstein. 1991. 'Are psoriatic patients at risk of heat intolerance?', *Br J Dermatol*, 124: 439-42.

Li, M. D. 2011. 'Leptin and beyond: an odyssey to the central control of body weight', *Yale J Biol Med*, 84: 1-7.

Lien, L. F., A. J. Brown, J. D. Ard, C. Loria, T. P. Erlinger, A. C. Feldstein, P. H. Lin, C. M. Champagne, A. C. King, H. L. McGuire, V. J. Stevens, P. J. Brantley, D. W. Harsha, M. A. McBurnie, L. J. Appel, and L. P. Svetkey. 2007. 'Effects of PREMIER lifestyle modifications on participants with and without the metabolic syndrome', *Hypertension*, 50: 609-16.

Lord, G. M., G. Matarese, J. K. Howard, R. J. Baker, S. R. Bloom, and R. I. Lechler. 1998. 'Leptin modulates the T-cell immune response and reverses starvation-induced immunosuppression', *Nature*, 394: 897-901.

Luo, H. 2024. 'Global burden and cross-country inequalities in six major immune-mediated inflammatory diseases from 1990 to 2021: A systemic analysis of the Global Burden of Disease Study 2021', *Autoimmun Rev*, 23: 103639.

Mackonochie, M., A. Rodriguez-Mateos, S. Mills, and V. Rolfe. 2023. 'A Scoping Review of the Clinical Evidence for the Health Benefits of Culinary Doses of Herbs and Spices for the Prevention and Treatment of Metabolic Syndrome', *Nutrients*, 15.

Martins, F. F., V. Souza-Mello, M. B. Aguila, and C. A. Mandarim-de-Lacerda. 2023. 'Brown adipose tissue as an endocrine organ: updates on the emerging role of batokines', *Horm Mol Biol Clin Investig*, 44: 219-27.

McEvoy, John William, Cian P McCarthy, Rosa Maria Bruno, Sofie Brouwers, Michelle D Canavan, Claudio Ceconi, Ruxandra Maria Christodorescu, Stella S Daskalopoulou, Charles J Ferro, Eva Gerdts, Henner Hanssen, Julie Harris, Lucas Lauder, Richard J McManus, Gerard J Molloy, Kazem Rahimi, Vera Regitz-Zagrosek, Gian Paolo Rossi, Else Charlotte Sandset, Bart Scheenaerts, Jan A Staessen, Izabella Uchmanowicz, Maurizio Volterrani, Rhian M Touyz, and ESC Scientific Document Group. 2024. '2024

ESC Guidelines for the management of elevated blood pressure and hypertension: Developed by the task force on the management of elevated blood pressure and hypertension of the European Society of Cardiology (ESC) and endorsed by the European Society of Endocrinology (ESE) and the European Stroke Organisation (ESO)', *European Heart Journal*, 45: 3912-4018.

Michalek, Irmina, Belinda Loring, and Swen John. 2016. *WHO Global report on psoriasis*.

Mohamed Haris, N. H., S. Krishnasamy, K. Y. Chin, V. Mariappan, and M. Arumugam. 2023. 'Metabolic Syndrome Screening and Nutritional Status of Patients with Psoriasis: A Scoping Review', *Nutrients*, 15.

Mohsin, S. N., F. Saleem, A. Humayun, A. Tanweer, and A. Muddassir. 2023. 'Prospective Nutraceutical Effects of Cinnamon Derivatives Against Insulin Resistance in Type II Diabetes Mellitus-Evidence From the Literature', *Dose Response*, 21: 15593258231200527.

Montecucco, F., F. Mach, and A. Pende. 2013. 'Inflammation is a key pathophysiological feature of metabolic syndrome', *Mediators Inflamm*, 2013: 135984.

Moon, S., J. Kang, S. H. Kim, H. S. Chung, Y. J. Kim, J. M. Yu, S. T. Cho, C. M. Oh, and T. Kim. 2020. 'Beneficial Effects of Time-Restricted Eating on Metabolic Diseases: A Systemic Review and Meta-Analysis', *Nutrients*, 12.

Nestle, Frank O., Daniel H. Kaplan, and Jonathan Barker. 2009. 'Psoriasis', *New England Journal of Medicine*, 361: 496-509.

Oh, J. Y., Y. A. Sung, H. J. Lee, J. Y. Oh, H. W. Chung, and H. Park. 2010. 'Optimal waist circumference for prediction of metabolic syndrome in young Korean women with polycystic ovary syndrome', *Obesity (Silver Spring)*, 18: 593-7.

Ommen, P., T. Stjernholm, T. Kragstrup, L. Raaby, C. Johansen, K. Stenderup, L. Iversen, and C. Rosada. 2016. 'The role of leptin in psoriasis comprises a proinflammatory response by the dermal fibroblast', *Br J Dermatol*, 174: 187-90.

Parisi, R., I. Y. K. Iskandar, E. Kontopantelis, M. Augustin, C. E. M. Griffiths, and D. M. Ashcroft. 2020. 'National, regional, and worldwide epidemiology of psoriasis: systematic analysis and modelling study', *Bmj*, 369: m1590.

Pratyush, D. D., S. Tiwari, S. Singh, and S. K. Singh. 2012. 'Waist circumference cutoff and its importance for diagnosis of metabolic syndrome in Asian Indians: A preliminary study', *Indian J Endocrinol Metab*, 16: 112-5.

Qiu, L., C. Gao, H. Wang, Y. Ren, J. Li, M. Li, X. Du, W. Li, and J. Zhang. 2023. 'Effects of dietary polyphenol curcumin supplementation on metabolic, inflammatory, and oxidative stress indices in patients with metabolic syndrome: a systematic review and meta-analysis of randomized controlled trials', *Front Endocrinol (Lausanne)*, 14: 1216708.

Ramezani, Mazaher, Elisa Zavattaro, and Masoud Sadeghi. 2019. 'Evaluation of serum lipid, lipoprotein, and apolipoprotein levels in psoriatic patients: a systematic review and meta-analysis of case-control studies', *Advances in Dermatology and Allergology/Postępy Dermatologii i Alergologii*, 36: 692-702.

Ramírez-Boscá, A., V. Navarro-López, M. Carrión-Gutiérrez, A. Martínez-Andrés, J. J. Vilata-Corell, M. Asín-Llorca, J. F. Horga de la Calle, and A. Bernd. 2017. 'Efficiency and safety of a Curcuma extract combined with visible blue light phototherapy on adults with plaque psoriasis: A phase IV, randomized, open pilot clinical trial', *J Dermatol*, 44: 1177-78.

Ronis, M. J. J., K. B. Pedersen, and J. Watt. 2018. 'Adverse Effects of Nutraceuticals and Dietary Supplements', *Annu Rev Pharmacol Toxicol*, 58: 583-601.

Ruderman, N. B., S. H. Schneider, and P. Berchtold. 1981. 'The "metabolically-obese," normal-weight individual', *Am J Clin Nutr*, 34: 1617-21.

Ryan, C., and B. Kirby. 2015. 'Psoriasis is a systemic disease with multiple cardiovascular and metabolic comorbidities', *Dermatol Clin*, 33: 41-55.

Sacks, F. M., E. Obarzanek, M. M. Windhauser, L. P. Svetkey, W. M. Vollmer, M. McCullough, N. Karanja, P. H. Lin, P. Steele, M. A. Proschan, and et al. 1995. 'Rationale and design of the Dietary Approaches to Stop Hypertension trial (DASH). A multicenter controlled-feeding study of dietary patterns to lower blood pressure', *Ann Epidemiol*, 5: 108-18.

Sahu, B., and N. C. Bal. 2023. 'Adipokines from white adipose tissue in regulation of whole body energy homeostasis', *Biochimie*, 204: 92-107.

Saumya, Choudhary, Pradhan Dibyabhaba, Pandey Anamika, Khan Mohd. Kamran, Lall Rohit, V. Ramesh, Puri Poonam, K. Jain Arun, and Thomas George. 2020. 'The Association of Metabolic Syndrome and Psoriasis: A Systematic Review and Meta-Analysis of Observational Study', *Endocrine, Metabolic & Immune Disorders - Drug Targets*, 20: 703-17.

Schmidt, B. A., and V. Horsley. 2013. 'Intradermal adipocytes mediate fibroblast recruitment during skin wound healing', *Development*, 140: 1517-27.

Sheppard, R., W. K. Gan, G. L. Onambele-Pearson, and H. S. Young. 2024. 'Increased physical activity promotes skin clearance, improves cardiovascular and psychological health, and increases functional capacity in patients with psoriasis', *Skin Health Dis*, 4: e426.

Sheppard, Rory, Weh Kiat Gan, Gladys L Onambele-Pearson, and Helen S Young. 2022. 'Developing an aerobic exercise intervention for patients with psoriasis to support lifestyle behaviour change and improve health outcomes', *Clinical and Experimental Dermatology*, 48: 5-11.

'Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report'. 2002. *Circulation*, 106: 3143-421.

Vafaeipour, Z., B. M. Razavi, and H. Hosseinzadeh. 2022. 'Effects of turmeric (Curcuma longa) and its constituent (curcumin) on the metabolic syndrome: An updated review', *J Integr Med*, 20: 193-203.

Valenzuela-Fuenzalida, J. J., V. S. Bravo, L. M. Valarezo, M. F. Delgado Retamal, J. M. Leiva, A. Bruna-Mejías, P. Nova-Baeza, M. Orellana-Donoso, A. Suazo-Santibañez, G. Oyanedel-Amaro, and H. Gutierrez-Espinoza. 2024. 'Effectiveness of DASH Diet versus Other Diet Modalities in Patients with Metabolic Syndrome: A Systematic Review and Meta-Analysis', *Nutrients*, 16.

Villarroya, J., R. Cereijo, A. Gavaldà-Navarro, M. Peyrou, M. Giralt, and F. Villarroya. 2019. 'New insights into the secretory functions of brown adipose tissue', *J Endocrinol*, 243: R19-r27.

Villarroya, J., R. Cereijo, and F. Villarroya. 2013. 'An endocrine role for brown adipose tissue?', *Am J Physiol Endocrinol Metab*, 305: E567-72.

Visseren, F. L. J., F. Mach, Y. M. Smulders, D. Carballo, K. C. Koskinas, M. Bäck, A. Benetos, A. Biffi, J. M. Boavida, D. Capodanno, B. Cosyns, C. Crawford, C. H. Davos, I. Desormais, E. Di Angelantonio, O. H. Franco, S. Halvorsen, F. D. R. Hobbs, M. Hollander, E. A. Jankowska, M. Michal, S. Sacco, N. Sattar, L. Tokgozoglu, S. Tonstad, K. P. Tsioufis, I. van Dis, I. C. van Gelder, C. Wanner, and B. Williams. 2021. '2021 ESC Guidelines on cardiovascular disease prevention in clinical practice', *Eur Heart J*, 42: 3227-337.

Wang, X., Q. Li, Y. Liu, H. Jiang, and W. Chen. 2021. 'Intermittent fasting versus continuous energy-restricted diet for patients with type 2 diabetes mellitus and metabolic syndrome for glycemic control: A systematic review and meta-analysis of randomized controlled trials', *Diabetes Res Clin Pract*, 179: 109003.

Wong, Y., S. Nakamizo, K. J. Tan, and K. Kabashima. 2019. 'An Update on the Role of Adipose Tissues in Psoriasis', *Front Immunol*, 10: 1507.

Xue, K., H. Liu, Q. Jian, B. Liu, D. Zhu, M. Zhang, L. Gao, and C. Li. 2013. 'Leptin induces secretion of pro-inflammatory cytokines by human keratinocytes in vitro--a possible reason for increased severity of psoriasis in patients with a high body mass index', *Exp Dermatol*, 22: 406-10.

Zhang, J., B. G. Kibret, D. E. Vatner, and S. F. Vatner. 2024. 'The role of brown adipose tissue in mediating healthful longevity', *J Cardiovasc Aging*, 4.

Zhang, Xiaochao, Suhan Zhang, Ruifang Wu, Siying Li, Yuwen Su, and Peng Zhang. 2022. 'Prevalence of autoimmune thyroid disease in patients with psoriasis: a meta-analysis', *BMJ Open*, 12: e055538.