REVIEW ARTICLE

Non-Pharmacological Interventions for Treating Psoriatic Arthritis

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ABSTRACT

Background and Objective • In this review, we discuss evidence concerning the management of psoriatic arthritis (PsA) patients with non-pharmacological interventions and additionally develop physical training protocols that could be prescribed to these patients.

Methods • We selected 110 articles, published on PubMed and Google Scholar databases from 1972 to date, investigating the effects of generic hygienic-dietary recommendations and training programs in PsA or psoriasis (PSO) individuals.

Results • Although data in support are limited, aerobic, endurance, and strength exercises as well as complementary techniques may all be useful in preserving or improving residual functional capacity, joint flexibility, and muscle strength. Exercise may reduce systemic inflammation, pain, and fatigue and additionally control PsA comorbidities, like dysmetabolism or obesity.

Conclusions • The polyhedral clinical expression of PsA underlines the need for a multidisciplinary approach combining the synergistic effects of pharmacological and non-pharmacological treatments. The latter range from preventive measures, like dietary modifications, weight loss, and cigarette smoking cessation, to personalized training protocols according to disease activity and phenotype, comorbidities, and individual tolerability. In these patients, we strongly encourage the regular practice of motor activity at progressively increasing intensity with combined supervised aerobic, strength, endurance, and stretching exercises. (*Altern Ther Health Med.* 2024;30(3):36-43).

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INTRODUCTION

Psoriatic arthritis (PsA) is a chronic rheumatic disease belonging to the group of seronegative spondyloarthritis (SpA) and characterized by a variety of musculoskeletal manifestations associated with current or previous psoriasis (PSO) or family history of PSO. The disease is extremely heterogeneous in terms of clinical manifestations and severity. In 1973 Wright and Moll classified PsA into five clinical forms based on the type and number of joints affected.[1] This classification is still valid and encompasses asymmetrical oligoarthritis, symmetrical polyarthritis, spondylitis, distal interphalangeal predominant arthritis, and arthritis mutilans, each of which displays different demographic, clinical, therapeutic, and prognostic features. Besides PSO, PsA patients may also suffer from other extra-articular manifestations of the disease, like conjunctivitis, iridocyclitis, and inflammatory bowel disease, or comorbidities, such as dysmetabolic disorders, fatty liver, osteoporosis, and fibromyalgia.[2]

The etiopathogenesis of PsA and PSO is multifactorial. It is assumed that in a genetically predisposed individual, exposure to environmental factors would trigger a chronic immune response resulting in both autoimmunity and autoinflammation in the skin, joints, and entheses. In these sites, antigen-presenting cells (APCs), T lymphocytes, endothelial cells, synovial cells, and keratinocytes may induce and perpetuate the inflammatory cascade by producing key-

cytokines, like tumor necrosis factor-alpha (TNF-α) and interleukin 17 (IL-17). In addition, other cells belonging to the innate immune system may contribute to disease pathogenesis, such as innate lymphoid cells (ILC), natural killer (NK) cells, and mucosal-associated invariant T (MAIT) cells. Polymorphisms in genes coding for human leukocyte antigen (HLA) molecules as well as microorganisms, biomechanical stress, and obesity represent risk factors and may altogether participate in the onset and maintenance of the disease.[3,4]

In recent years, a cumulative amount of data has confirmed the role of dysbiosis in inducing autoimmune diseases, like PSO or PsA.[5] Studies on animal models have shown that the overgrowth of intestinal pathogens may activate APCs through the release of Pathogen-Associated-Molecular-Patterns (PAMPs), with the following differentiation of T helper (Th)1, Th17, Th9, and Th22 lymphocytes and the repression of T regulatory (Treg) cells.[6] Once activated, Th lymphocytes may migrate from the intestinal mucosa to the skin or joints, giving rise to local inflammation.

A parallel pathogenetic hypothesis, which does not exclude the role of dysbiosis, instead focuses on reiterated biomechanical stress.[7] Mechanical stress at cutaneous and entheseal sites may favor the release of Damage-Associated-Molecular-Patterns (DAMPs), locally activating the immune system in a similar way to PAMPs.[8] Subclinical enthesitis can be ultrasonographically revealed in 32-62% of subjects with PSO and evolution towards PsA has been reported in 23% of these patients.[9] On the other hand, cutaneous mechanical stress could also serve as the basis of the Koebner phenomenon, characterized by the onset of psoriatic plaques in chronically traumatized skin sites.[10]

Finally, PSO and PsA patients often suffer from metabolic disorders, like dyslipidemia, type 2 diabetes mellitus or hyperinsulinism, high blood pressure, and hyperuricemia. In addition, they are often obese and the incidence of PsA proportionally arises with the increase in body mass index (BMI).[11] Visceral adipose tissue can contribute to systemic inflammation by synthesizing pro-inflammatory mediators, known as adipokines, when stimulated by a *milieu* of cytokines, such as TNF-α and IL-6. Adipokines may mediate endothelial dysfunction, oxidative stress, thrombocytosis, and myointimal hyperplasia, finally leading to atherosclerosis and cardiovascular disease.[12,13]

Many risk factors involved in the etiopathogenesis of PSO or PsA may be corrected or prevented with simple hygienic-dietary interventions, like changes in lifestyle and dietary habits or the practice of physical activity. This review aims at extensively reporting scientific evidence in support of the efficacy and safety of non-pharmacological interventions for PsA and PSO individuals. Based on the results of the studies, we also developed an algorithm to manage these patients with hygienic-dietary measures and physical exercise.

METHODS

A search of the PubMed and Google Scholar databases was performed using the keywords "psoriasis" or "psoriatic arthritis" AND "non-pharmacological therapy". We selected 110 articles relevant to the aim of our review, including clinical trials, randomized controlled trials (RCTs), metanalyses, and guidelines, written in English and published from 1972 to the present.

International recommendations for the non-pharmacological management of PsA patients

Although international guidelines are starting to incorporate non-pharmacological interventions among the therapeutic options for PsA patients,[14-16] most recommendations still display a low level of evidence, due to the paucity of supportive RCTs. According to the 2018 American College of Rheumatology (ACR) guidelines,[15] any type or combination of physical activity, occupational therapy, massage therapy, and acupuncture should be recommended over no interventions in PsA individuals if well tolerated and not contraindicated. Low-impact motor activity, such as swimming or practicing oriental disciplines, should be preferred over high-impact activities, although the latter could be considered in case of no contraindications and patients' preference. Complementary therapies, such as massage therapy or acupuncture, may be indicated on a case-by-case basis after careful discussion and evaluation of the benefit/cost profile.

In the same year, the European League Against Rheumatism (EULAR) also drew up a set of recommendations for the physical management of patients with inflammatory arthropathies.[17] Though remaining non-specific in the intervention programs and not electively focusing on PsA patients, these guidelines underline the importance of physical therapy in patients with SpA. Physical training protocols should be individualized based on patients' cardiorespiratory fitness, muscle strength, flexibility, and neuromotor performance, as well as preference, safety, and feasibility.

Change in lifestyle and dietary habits

Hygienic-dietary measures aiming at the control or elimination of modifiable risk factors, such as dysmetabolism, being overweight, cigarette smoking, dysbiosis, and sedentary lifestyle, may optimize the quality of life and response to pharmacological therapies in PSO or PsA patients.[11,18] Evidence in favor of each intervention is detailed below.

Diet and weight loss. Several epidemiological studies have revealed that patients with PSO often have unbalanced diets with a higher intake of total fats and simple carbohydrates at the expense of proteins, complex carbohydrates, monounsaturated fatty acids, omega-3 polyunsaturated fatty acids (3-PUFAs), vegetables, and dietary fiber compared to healthy controls.[19,20] An impressive amount of data sustains that saturated fatty acids (SFAs), simple sugars, red meat or alcohol can exacerbate PSO, while other nutrients, such as vitamin D, vitamin B12, 3-PUFAs, dietary fiber, genistein, selenium, or probiotics could improve it (Table 1).[21-40] SFAs, simple sugars, red meat, and alcohol may favor intestinal

Table 1. Nutrients Ameliorating or Exacerbating PsA and PSO

Nutrients ameliorating PsA and PSO	Mechanism of Mechanism of action/evidence	Nutrient exacerbating PsA and PSO	Mechanism of action
Vitamin D (cod liver oil, fish, mushrooms, bovine liver, eggs, and cheese)	• Prevention of the release of TNF-α, IL-1β, IL-6, and IL-8 from monocytes/macrophages; inhibition of antigenic presentation and activation of autoreactive lymphocytes;[22] • Reduced serum vitamin D levels in patients with PSO or PsA compared to controls;[22] • Vitamin D sequestration in the adipose tissue of obese/overweight patients;[22] • Reduced vitamin D intake or intestinal absorption in patients with concomitant inflammatory colitis.[22]	Saturated fatty acids (foods of animal origin, including butter and red meat)	• Inflammation promotion through the activation of the NLRP3 inflammasome platform and the IL-23/IL-17 cytokine pathway;[21] • Promotion of intestinal dysbiosis by dietary heme in animal models, with subsequent unbalance in *Enterobacteriaceae*/Lactobacilli ratio, butyrate synthesis, and T lymphocyte differentiation;[33] • Risk factors for obesity, dyslipidemia, and cardiovascular disease.[21]
Vitamin B12 (fish, shellfish, oysters, clams or salmon roe, cattle liver, pork, or chicken)	• Clearance of reactive oxygen species and antioxidant role;[23] • Conversion of homocysteine into methionine, through the transfer of a methyl group in turn released by 5-methyl-tetrahydrofolate, whose deficit may lead to hyperhomocysteinemia, recognized as an independent risk factor for the development of cardiovascular diseases;[23] • Higher serum homocysteinemia level in PSO individuals compared to controls.[24]	Alcohol	• Epidermal barrier damage resulting in an increased permeability to exogenous pathogens, which may in turn stimulate the proliferation of keratinocytes and the release of pro-inflammatory cytokines;[32,33] • Chronic alcohol abuse associated with susceptibility to skin infections caused by Gram+ and Gram- bacteria;[31,33] • Chronic alcohol consumption associated with intestinal dysbiosis;[33] • Direct induction of keratinocyte proliferation and hyper-expression of genes associated with proliferative patterns by ethanol and acetone;[31,34] • PSO onset or exacerbation triggered by the hepatic synthesis of acetaldehyde that occurs during the metabolism of ethanol through the release of reactive oxygen species and the activation of MAPK/AP-1, NF-κB, and JAK/STAT cascades;[31,35] • Activation of lymphocytes, monocytes, and macrophages with subsequent proliferation and production of pro-inflammatory cytokines.[31,33]
Genistein (soy)	• Strong anti-inflammatory activity;[25] • Improvement of psoriasiform dermatitis in animal models treated with genistein topical application, with a reduction in clinical severity and the cutaneous expression of pro-inflammatory cytokines.[26]		
Selenium (fish and crustaceans, eggs, poultry, or cereals)	• Antioxidant and immunoregulatory properties, thanks to selenoproteins that include glutathione peroxidase and thioreductase;[27,28] • Control of the production of eicosanoids involved in the inflammatory response;[27] • Lower serum selenium levels in PSO patients compared to controls.[29]		
Probiotics (*Lactobacillus, Bifidobacterium,* and *Saccharomyces boulardii*), found in foods (yogurt and fermented foods) and food supplements	• Remodulation in the composition of the gastrointestinal flora;[38,39,41] • Synergistic effect with medications in animal models of arthritis;[39] • Conflicting data concerning improvements in PsA disease activity.[40]		

Abbreviations:: TNF-α, tumor necrosis factor-alpha; IL, interleukin; PsA, psoriatic arthritis; PSO, psoriasis; NLRP3, NLR Family Pyrin Domain Containing 3; MAPK, mitogen-activated protein kinase; AP-1, activator-protein-1; NFκB, nuclear factor-κB; JAK/STAT, Janus kinase/signal transducers and activators of transcription.

dysbiosis, and thus promote the aberrant activation of autoinflammatory and autoimmune pathways. Conversely, 3-PUFAs, vitamin D, vitamin B12, short-chain fatty acids (SCFAs), selenium, genistein, dietary fiber, and probiotics could improve PSO through the inhibition of inflammatory pathways and the induction of immunotolerance.[33,41] Patients with PsA very often suffer from subclinical or clinical colitis or duodenitis that may be the result of gut dysbiosis.[42-44] In these subjects, the use of probiotics may be recommended.[45] Probiotic strains, such as *Lactobacillus, Bifidobacterium,* or *Saccharomyces boulardii,* administered in food or supplements, may restore dysbiosis and intestinal mucosal health,[45,46] although their effect on PsA disease is conflicting.[40,47] Additionally, PSO and PsA patients should be encouraged to adopt a Mediterranean diet. A study conducted on 62 patients affected by PSO with mild to severe disease activity and 62 matched controls revealed that PSO patients have a lower consumption of extra-virgin olive oil, fruit, fish, and nuts and a greater intake of red meat than controls.[20] Furthermore, adherence to the Mediterranean diet was inversely associated with PSO extension and severity, measured through the Psoriasis Area Severity Index (PASI), and systemic inflammation, evaluated through serum C-reactive protein (CRP) levels. Similarly, an Italian cross-sectional study on PsA patients reported an inverse correlation between adherence to the Mediterranean diet and disease activity measured through the disease activity index for PsA (DAPSA) and a positive association between DAPSA and BMI.[48] The Mediterranean diet may prevent obesity, which represents an important risk factor for PSO and PsA development or scarce therapeutic response.[49] Results of a recent meta-analysis showed that in PSO obese patients dietary interventions can reduce disease severity (low level of evidence), improve quality of life, and decrease BMI (moderate-to-low level of evidence) compared to usual care, while a combined intervention (diet plus exercise) could reduce disease severity and BMI compared to educational programs (moderate level of evidence).[50] Obesity, together with folic acid and vitamin B12 deficiency, cigarette smoking, and alcohol consumption, are altogether risk factors for hyperhomocysteinemia, which in turn, is associated with cardiovascular disease.[51] Above-average alcohol intake, particularly common among patients with PSO,[52] may also worsen skin lesions by altering the cutaneous hydrolipidic film, which consequently increases the permeability to exogenous pathogens and local activation of pro-inflammatory pathways.[53]

Finally, vitamin D supplementation should be always considered, given its anti-inflammatory role and relative deficiency in PSO and PsA subjects, especially if obese or with malabsorption disorders.[22] In addition, vitamin D may prevent osteopenia or osteoporosis, both of which represent common PsA comorbidities.

Cigarette smoking cessation. Cigarette smoking is a known risk factor triggering or worsening PSO.[54] The risk of PSO increases with the number of cigarettes smoked per day and it is higher in women than in men, while a gradual reduction is observed after smoking cessation.[55,56] According to a Norwegian study on 18 747 participants, current smoking was associated with a PSO odds ratio (OR) of 1.49 for males and 1.48 for females compared to non-smokers.[57] Smoking more than 20 cigarettes per day was associated with

significantly higher OR of PSO development in males. The most plausible underneath mechanism is oxidative stress which may drive endothelial dysfunction, and thus contribute to increased cardiovascular risk in these patients.[58-60] Surprisingly, in a large United Kingdom (UK) population-based study, cigarette smoking was associated with an increased PsA risk in the general population and a decreased PsA risk in PSO patients.[61] This paradox may however be attributed to methodological bias in study population selection and group allocation.

Physical exercise. It is well-known that the regular practice of physical activity in combination with a balanced diet can reduce cardiovascular risk, prevent endothelial dysfunction, and lower systemic inflammation.[62] Furthermore, physical exercise may improve joint flexibility, preserve residual function, ameliorate aerobic capacity, reinforce bones and muscles, and normalize BMI in rheumatic patients.[63] Several studies have also underlined many repercussions on the immune system, in terms of peripheral lymphocyte count, cytokine secretion, antibody synthesis, and NK cell activation.[64-66] In addition, exercise may induce an increase in plasma endorphin levels, with beneficial effects on nociception and mental distress.[63,64] The latter is quite common among PSO or PsA patients and is associated with poor sleep quality, fatigue, depression, and reduced work productivity.[67] Psychological stress also represents one of the main triggers of PSO, favoring its onset or re-exacerbation.[68-70]

The American College of Sports Medicine and the American Heart Association (ACSM/AHA) recommend at least 30 minutes per day of moderate-intensity physical activity to improve health and fitness in the general population.[71] However, pain, fatigue, stiffness, disabilities, and lack of knowledge about long-term exercise benefits represent barriers to physical activity in rheumatic patients.[63] Psychological distress associated with PSO onset may result in the reduction, or even abandonment, of social and recreational activities. A survey conducted in Finland aimed at evaluating the impact of PSO on leisure activities in a sample of 262 individuals showed that more than half of the interviewed subjects had abandoned or reduced their free time activities and more than 30% of them had given up sport after being diagnosed with the disease.[72] These results are in line with those reported in an Italian study conducted on 416 young sportive subjects compared with 489 controls and 400 patients with PSO.[73] This study also underlines the preventive role played by physical activity on stress-induced PSO.

Although physical activity confers numerous advantages in terms of disease activity, pain, fatigue, quality of life, muscle strength, and cardiovascular comorbidities in PsA subjects,[74] reiterated cutaneous and musculoskeletal traumatisms may precipitate PSO or enthesitis.[7,8,10] In a cross-sectional study, conducted on 84 patients with PsA and 25 healthy volunteers, a significant association was found between high modified MAdrid Sonographic Enthesis Index (MASEI) scores and the practice of physical activity in PsA individuals.[75] Though in need of being confirmed in longitudinal studies, these data suggest that PsA enthesitis may be accentuated by exercise, therefore requiring careful evaluation of the risk/benefit profile of prescribed interventions. These will be individually detailed in the sections below.

Aerobic and endurance exercise. Aerobic exercise, also called "cardio", consists of a motor practice with modest joint impact and is therefore recommendable to patients with articular diseases or musculoskeletal pain. Aerobic exercise can improve the range of motion (ROM) and relieve stiffness while decreasing inflammation.[15] In addition, the regular practice of aerobic activity can positively influence mood and nociception. Examples of low to medium-impact aerobic exercise include walking, swimming, and cycling. To further reduce joint or entheseal stress, aerobic exercise can be performed in water. The optimal strategy should be decided after a careful evaluation of patients' residual capabilities and physical performance. While PSO subjects seem not to differ from healthy individuals in terms of basal metabolic rate, maximum oxygen consumption (VO_2max), and respiratory parameters,[76] PsA patients may have a higher degree of cardio-pulmonary involvement and a major pharmacological burden than PSO individuals that may negatively influence health status and aerobic capacity. In these subjects, motor activity should be therefore tailored and carried out at progressively increasing intensity (e.g., initial 10-minute training sessions to be repeated 3 times during the day). Conversely, patients with PSO alone or trained PsA patients without complications may also be prescribed high-intensity training. Results from an RCT analyzing the impact of an 11-week high-intensity interval training (HIIT) on disease activity and disease perception in 67 patients with PsA with stable disease and no comorbidities revealed that HIIT is safe and well-tolerated and has a positive effect on fatigue.[77] According to a study, practicing vigorous physical activity may also prevent PSO development over a 14-year follow-up period with superior effects to walking, cycling, jogging, or running.[78] Although walking represents a paradigmatic example of low-impact aerobic activity recommendable to either PsA or PSO patients, it could potentially increase the risk of plantar fascia and Achilles tendon enthesitis and, according to some authors, even trigger PsA in genetically predisposed subjects.[79] Home-based aerobic programs may also be useful for promoting an active lifestyle, especially in patients who cannot afford classes due to economic or logistical reasons. According to a study conducted on 30 PsA patients with minimal disease activity,[80] compliance of participants to home-based aerobic exercises can reach up to 76.6%, with patients experiencing significant improvements in both the physical and mental domains of the 36-item Short Form Health Survey (SF-36) and Health Assessment Questionnaire for the Spondyloarthropathies (HAQ-S) scores.

Only one double-blind RCT examined the effects of endurance training, aiming at augmenting muscle size, strength, and power in a cohort of PsA patients.[81] Results showed significant amelioration in HAQ-S and Bath

Ankylosing Spondylitis Disease Activity Index (BASDAI) scores, with no adverse events.

Aquatic exercise and hydrotherapy. Thanks to its properties, water represents an optimal environment for exercising in an atraumatic way. The encouraging data in patients with ankylosing spondylitis (AS),[82] suggest that swimming could be indicated in PsA patients with axial skeleton involvement, but evidence in support is lacking.

Hydrotherapy, on the other hand, brings numerous benefits regarding physical and cognitive function, energy, sleep, and participation in the activities of daily life. According to a qualitative study, PsA patients consider hydrotherapy as a multidimensional experience, in which situational factors (water temperature, previous training, group composition, music, and the competence of the instructor) and hydrotherapy effects (psychological implications, improvements in physical capacity, socialization, pain perception, and participation in daily life and work activities) are equally relevant.[83]

Stretching. Static or dynamic stretching consists of a practice used in the preparatory or final phase of many sports activities that aim at restoring or maximizing musculoskeletal flexibility and ROM.[84] In PsA patients, adapted stretching techniques for musculoskeletal pain could help avoid joint stiffness and enhance flexibility.[84] However, the benefits of this discipline in PsA are uncertain. Stretching is an integral part of postural rehabilitation programs for AS individuals, in whom it shows undiscussed advantages in improving spinal flexibility, joint stiffness, and quality of life.[85] Similar effects are also conceivable in patients with PsA with an axial or enthesitic form of disease.

Mind-body disciplines. Mind-body disciplines belong to the group of relaxation techniques that focus on muscle relaxation, breathing exercises, and meditation and include Yoga, Tai chi, and Pilates.

Yoga is an oriental practice based on slow movements and breathing techniques.[86] Results of a recent study showed that an 8-week supervised Yoga program significantly improved lower limb function, pain, and quality of life in a cohort of 21 children with enthesitis-related arthritis, which can be considered a prototype of juvenile PsA.[87]

Tai chi is another holistic discipline based on controlled movements, meditation, and stretching exercises combined with deep breathing.[88] Benefits of this practice are numerous and include ameliorations of body flexibility and strength, mental well-being and relaxation, and stress and chronic pain reduction.[89] No data concerning the effects of Tai chi on PsA patients are however available. In a single-blind RCT on SpA individuals, an 8-week Tai chi training led to significant improvements in disease activity and spine flexibility.[90] These data could indirectly suggest the effectiveness of this method in the treatment of PsA patients with a predominant axial involvement.

Pilates, which takes its name from its founder Joseph Hubertus Pilates, is performed with free body exercises that merge dance, Yoga, and gymnastic movements and aim to increase body awareness and ameliorate balance and harmony.[91] If constantly practiced, this discipline can ameliorate posture, joint mobility and flexibility, physical strength, pain, stress relief, and cardiopulmonary performance.[92] Although no evidence is available for PsA patients, results from a single-blind RCT on AS individuals showed significant amelioration in Bath Ankylosing Spondylitis Functional Index (BASFI), BASDAI, and Bath Ankylosing Spondylitis Metrology Index (BASMI) scores and chest expansion after a 12-week Pilates training,[93] thereby suggesting its presumable usefulness in axial PsA.

Physiokinesitherapy and complementary therapies. Physiokinesitherapy (PKT) is a branch of physiotherapy aiming at motor rehabilitation and includes, beyond physical exercise, patient education, occupational therapy, orthosis use, lymphatic drainage, and other physical approaches, like tecar therapy, laser therapy, cryotherapy, magnetotherapy, manual therapy, or acupuncture. PKT may be especially useful in PsA individuals who fail to respond to pharmacological therapies or develop permanent disabilities affecting autonomy in daily gestures, function, and quality of life.[94]

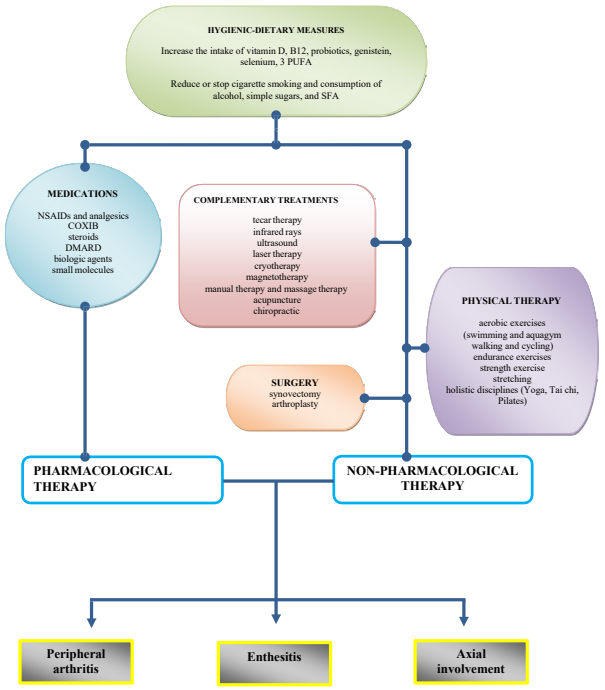
If associated with exercise, complementary therapies may play a synergistic role in symptom relief and amelioration of global health status. Tecar therapy, laser therapy, ultrasounds, magnetotherapy, cryotherapy, and therapeutic massage may be indicated in case of periarticular soft tissue inflammation, fibrosis, or calcification.[95-99] Three studies reported significant benefits with laser therapy on pain alleviation, foot function, disease activity, and plantar fascia thickness in patients with calcaneal enthesitis with/without SpA diagnosis.[100-102]

Acupuncture was also reported to be a simple, convenient, effective, and safe method with long-lasting therapeutic effects, although results concerning PSO and PsA remain controversial.[103-105] The technique involves the stimulation of specific cutaneous points (acupoints) via the insertion of thin needles to relieve pain, tension, and stress.[104] Despite benefits on pain and function, acupuncture may precipitate PSO, as a plausible expression of the Koebner phenomenon.[105]

Expert opinion and intervention proposal

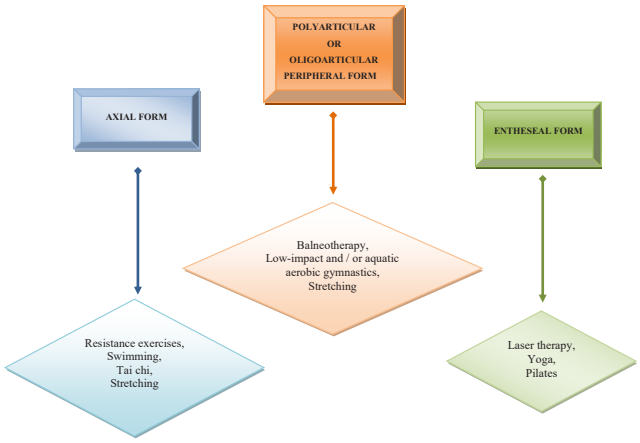
The non-pharmacological treatment of PSO and PsA patients includes several interventions that range from dietary modifications to physical exercise and complementary techniques. All these approaches can relieve symptoms and psychological stress, improve the course of the disease, prevent permanent joint damage and disability, counteract the development of comorbidities, and ameliorate the quality of life. Accordingly, PSO and PsA patients would mostly benefit from multidisciplinary management based on the collaboration between rheumatologists and other experts and require constant monitoring of disease activity and residual physical function as well as patients' wishes and expectations.[106] Despite the undisputed benefits, there is a lack of high-quality research and clear guidelines for

Figure 1. Multidisciplinary pharmacological and non-pharmacological management of PsA patients



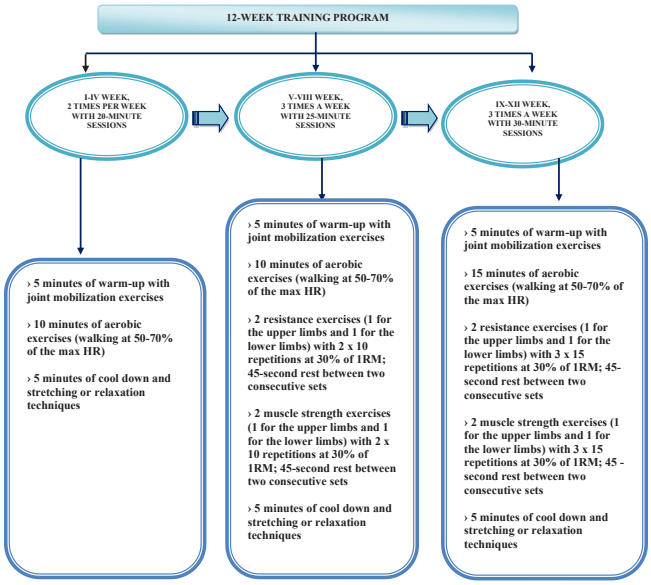
Abbreviations: 3-PUFA, omega-3 poly-unsaturated fatty acids; NSAIDs, non-steroidal anti-inflammatory drugs; COXIB, cyclooxygenase 2 inhibitors; DMARDs, disease-modifying anti-rheumatic drugs.

Figure 2. Non-Pharmacological Interventions Recommendable to PsA Patients and Individualized According to PsA Phenotype



prescribing non-pharmacologic interventions in PsA or PSO patients. The benefit-risk profile of physical interventions, how they can be prescribed, and their synergistic role with pharmacologic therapies are all items on the future research agenda. Overall, hygienic-dietary recommendations should be considered as the first step of the management of each PSO or PsA patient, while motor activity programs should be diversified according to disease activity and phenotype, comorbidities, and individual tolerability (Figure 1 and Figure 2). Patients should be prescribed a balanced diet, with an adequate supply of vitamin D and B12, probiotics,

Figure 3. Flowchart of a 12-Week Training Program that can be Prescribed to a Generic Patient with PsA



Abbreviations: HR max, maximum heart rate; RM, repetition maximum.

genistein, selenium, and 3-PUFAs, and a reduced intake of alcohol, simple sugars, and SFAs. Furthermore, each subject should be encouraged to quit smoking and undergo weight loss. Concerning physical activity, there are still no standardized protocols for this category of subjects. Whenever possible, supervised physical programs should be preferred over home-based training protocols and aerobic activities should always be part of the training. An ideal exercise protocol fitting to a generic PsA patient is depicted in Figure 3. According to this scheme, patients should undergo alternate sessions of moderate-to-low-impact aerobics/strength exercises and stretching/relaxation techniques. After 12 weeks, patients should be re-evaluated for improvements or side effects and the protocol should be adapted accordingly. This general program could be diversified according to the predominant phenotype, as proposed by other authors.[107] Like AS individuals, patients with axial disease may mostly benefit from stretching, swimming, and postural exercises, while those with peripheral arthritis may be managed with aquatic or land-based aerobic activities and joint mobilization exercises in a similar way to rheumatoid arthritis (RA) subjects.[108-110]

CONCLUSION

Although supported by limited evidence, non-pharmacological interventions represent a cornerstone in the management of PsA or PSO symptoms and comorbidities. Modifications in lifestyle and personalized training programs should always parallel pharmacological strategies in a multidisciplinary scenario. Aerobic and strength training, hydrotherapy, mind-body disciplines, and complementary therapies may be recommended to PsA patients, with diversified approaches based on disease phenotype.

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