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How to Treat Psoriasis Correctly: Complete Guide – 2026 Edition

Manole Cojocaru and Gheorghe Giurgiu

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How to Treat Psoriasis Correctly: Complete Guide – 2026 Edition

Manole Cojocaru¹ and Gheorghe Giurgiu^{2*}

¹Member of the Romanian Academy of Scientists, Faculty of Medicine,
“Titu Maiorescu” University, Bucharest, Romania

²Deniplant Aide-Santé Bucharest Biomedicine Center, Roamnia

Corresponding authors: Gheorghe Giurgiu, Deniplant Aide-Santé Bucharest
Biomedicine Center, Roamnia, E-mail: deniplant@gmail.com

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Introduction

About this guide and its authors

The guide contains information based on 30 years of experience of the author, Gheorghe Giurgiu, who, being a psoriasis sufferer, discovered the path to healing on his own, and together with Prof. Dr Manole Cojocar, helped over 15,000 psoriasis sufferers to heal.

The purpose of this material is to provide clear and scientifically based information for understanding, preventing and managing psoriasis. The content is adapted to the general public, bringing together modern medical knowledge from 2026, good care practices and immunity regulation strategies.

A special emphasis is placed on the essential role of the intestinal and skin microbiome in the health of the body, highlighted by observational clinical studies conducted within the Deniplant Aide-Sante Biomedicine Center, where the link between the microbiome and Autoimmune, Metabolic, Neurological conditions and their treatment with Nutraceuticals - foods with a dual role, of nutrition and health.

The results of these studies and research have been presented in specialized papers at over 640 international conferences and congresses.

The guide promotes an integrated approach — medical, preventive, and natural — to support a balanced life, reducing the discomfort caused by psoriasis.

This guide is aimed at patients eager to understand the root causes of the disease and the mechanisms necessary for the healing process

SECTION I: Fundamentals and Perspectives

Chapter 1: Historical perspective: What was known 50 years ago about psoriasis

Half a century ago, psoriasis was already identified as a chronic condition, influenced by genetic and environmental factors (stress, infections, or certain medications). Although it was known that it was not a contagious disease and that it affected both sexes, the understanding of the underlying immune mechanisms was limited. The medical approach at that time focused on controlling the symptoms, without being able to specifically block the inflammatory pathways, as we do today.

What was known (and scientifically confirmed):

- Etiology: The genetic component was known, being already associated with certain antigens (such as HLA-Cw6). The role of triggering factors was clearly observed: psychological stress, bacterial infections and skin lesions (Koebner phenomenon).

- The importance of family history:
 - Genetic factors were considered crucial, especially in young people.
 - Family history was present in 40-50% of cases, rising to 75% if disease onset occurred before age 20.
 - Twin studies showed high concordance in monozygotic twins and significantly lower (15-20%) in dizygotic twins.
- Lifetime statistical risk:
 - 4% if there was no family history.
 - 28% if one parent was affected.
 - 65% if both parents had psoriasis.
- Incidence and distribution: The disease affects approximately 3% of the global population, with a lower prevalence in populations from the Far East, American Indians, and West Africa.

What was less known (time limitations):

- Immune mechanisms: The specific role of T cells (Th1, Th17) and cytokines (TNF-alpha, IL-17) as therapeutic targets was not yet deciphered. The involvement of the immune system was viewed at a general, not molecular level.
- Lack of targeted therapies: Biological therapies, which revolutionized modern treatment, did not exist. Doctors resorted to broad-spectrum systemic treatments, but without the specificity necessary to stop the inflammatory cascade at the source.

Standard treatments in the 1970s:

1. Topical therapies: Corticosteroid creams, coal tar, salicylic acid and various forms of vitamin D or emollients were used intensively.
2. Phototherapy: The standard method was exposure to UVB rays or PUVA therapy (combining psoralens with UVA rays).
3. Systemic therapies: Methotrexate and cyclosporine were used in severe cases.
4. Nutrition: The emphasis was on rebalancing the diet and supplementing with vitamins A, D, C, and fatty acids, according to the level of knowledge at the time.

Conclusion

In essence, the medicine of the 1970s understood well “what” psoriasis was and “how” it manifests itself, but the answer to the question “why” (at the molecular level) was incomplete. Treatment was reactive, managing skin manifestations, unlike the medicine of 2026 which acts proactively on the underlying causes.

Chapter 2: Medicine in 2026: What we know about psoriasis today

In 2026, medicine defines psoriasis as a chronic, systemic inflammatory disease with a complex genetic and immunological basis. Although advances in biological therapies are remarkable, current research confirms that psoriasis remains a major challenge, not only physical, but also psychosocial.

1. Modern pathophysiology

- **Systemic disease:** Psoriasis is not just a dermatological problem. It is an autoimmune disease that can affect the joints (psoriatic arthritis) and the cardiovascular system, involving a generalized immune response.
- **Immune mechanism:** An abnormally high number of T cells triggers a cytokine cascade in the skin. This intense activity causes inflammation, redness, pruritus (itching), and accelerated proliferation of keratinocytes, leading to the characteristic scaling.

2. Psychosocial impact and quality of life

Statistics for 2026 remain concerning in terms of emotional impact:

- 43% of patients experience strong social shame and isolation.
- 60% experience episodes of clinical depression.
- 1 in 3 patients report thoughts of self-harm or suicide due to prolonged suffering.
- The disease directly affects the patient's career, productivity and financial resources.

3. Diagnosis and clinical forms

- **Types identified:** Plaque (vulgar), guttate, inverse, pustular and erythrodermic (the most severe form).
- **Diagnosis:** Remains predominantly clinical (by examining the lesions). Blood tests are used to exclude other pathologies, but in 2026, there is an increasing emphasis on biomarkers and microbiome analysis.

4. Treatment options and their limits

- In the case of mild forms, Topical treatments (creams, emollients) and advanced phototherapy (excimer UVB laser) are used.
- In the case of moderate/severe forms: Classical systemic treatments (methotrexate, cyclosporine) and targeted biological therapies (IL-17, IL-23 inhibitors) are used.
- **Comorbidities:** Psoriasis significantly increases the risk of metabolic diseases (type 2 diabetes, obesity) and cardiovascular diseases.

5. Key message of 2026

Although medical management has become extremely effective in controlling symptoms, many patients remain dissatisfied with the dependence on synthetic drugs and their side effects. Psoriasis requires long-term multifactorial management, which combines psychological support with a deep intervention on internal causes (microbiome, genetics, lifestyle), going beyond the simple application of symptomatic treatments.

Chapter 3: Psoriasis as an autoimmune and systemic disease

Psoriasis is a chronic autoimmune disease, defined by a signaling error in the immune system. It mistakenly attacks healthy skin cells, accelerating their regeneration cycle up to 10 times faster than normal. The result is the rapid accumulation of cells on the skin's surface in the form of red, thickened, scaly plaques.

Although the most common areas affected are the elbows, knees, and scalp, psoriasis is a systemic condition: inflammation is not limited to the skin, but can migrate to the joints (psoriatic arthritis) and affect overall metabolic health.

Clinical manifestations

- **Erythematous plaques:** Red, well-demarcated areas covered by silvery or white scales. The rapid accumulation of tissue often causes intense pruritus (itching) and a burning sensation.
- **Variability of lesions:** The disease can range from mild, punctate forms (guttate psoriasis) to severe, generalized forms that can cover large areas of the body (erythrodermic psoriasis).
- **Joint involvement:** Approximately 30% of patients may develop psoriatic arthritis, manifested by pain, stiffness, and swelling of the joints, with a severe impact on mobility.

Causes and triggers

- **Genetic predisposition:** Plays a crucial role; approximately one-third of patients have at least one first-degree relative who suffers from the same condition.
- **Environmental triggers (Triggers):**
 - **Psychological stress:** One of the most frequent exacerbating factors.
 - **Infections:** Especially streptococcal ones (pharyngitis, tonsillitis).
 - **Skin trauma:** Cuts, sunburn, or scratches (Koebner phenomenon).
 - **Lifestyle:** Smoking, alcohol consumption, obesity, and hormonal imbalances.

Essential aspects in 2026

1. **Non-contagious:** It is vital to understand that psoriasis is not an infectious disease. It is not transmitted through direct contact, the use of shared objects, or swimming in public pools.
2. **Chronic Evolution:** The disease evolves in cycles, with periods of remission and exacerbation. The way it is treated directly influences these periods.
3. **Warning on medication:** Certain aggressive treatments can provide rapid remission of symptoms, but, if not managed correctly, can lead to long-term resistance, exacerbation and even generalization of the disease (the “rebound” effect).
4. **Systemic Approach:** Since the problem lies in the immune system, treatment should not only be external (topical), but should aim to regulate the entire body.

Chapter 4: Triggering mechanisms: Genetic and environmental factors

The triggering of psoriasis, as in the case of many autoimmune diseases, is a complex process. It results from the interaction between genetic predisposition, environmental factors and a dysfunction of the immune system. Under these conditions, the body ceases to recognize its own cells, triggering a chronic inflammatory cascade.

1. Genetic pillar: Predisposition and heredity

- **HLA Genes:** Certain variants of the HLA (Human Leukocyte Antigen) complex significantly increase the risk of developing the disease. Although the presence of these genes does not guarantee the development of psoriasis, they represent the “fertile ground” for the disease.
- **Family History:** Psoriasis is more common in families with a documented history of autoimmune diseases (type 1 diabetes, lupus, multiple sclerosis).

2. Environmental triggers

A genetically predisposed person may remain asymptomatic until exposed to one or more triggers:

- **Infections:** Streptococcal infection (pharyngitis, tonsillitis) is the most common trigger for guttate psoriasis. Viruses and bacteria can cause immune cross-reactions.
- **Medications:** A wide range of substances can aggravate or trigger the disease:
 - Lithium, beta-blockers, antimalarials (hydroxy-chloroquine), terbinafine, TNF inhibitors, and interferons.
- **Trauma (Köebner phenomenon):** Psoriasis can occur exactly where the skin has been injured, cut, or burned.

- **Smoking and Alcohol:** Smoking increases the risk of plaque psoriasis and palmoplantar pustulosis. Excessive alcohol not only worsens symptoms, but also decreases the effectiveness of treatments.
- **Chronic Stress:** It is one of the strongest exacerbating factors, disrupting the neuro-immune balance.
- **Obesity:** It represents a systemic inflammatory state that maintains the disease.

3. The role of the digestive system and nutrition

In the medicine of 2026, the connection between the gut and the skin is fundamental:

- **Microbiome Dysbiosis:** An imbalance of intestinal bacteria directly contributes to immune activation.
- **Intestinal Permeability (“Leaky Gut”):** A fragile intestinal wall allows antigens to enter the bloodstream, forcing the immune system to constantly react.

4. Hormonal and climatic factors

- **Pregnancy:** Psoriasis tends to improve during pregnancy (due to natural immunosuppression), but can suddenly worsen postpartum.
- **Sunlight:** Although controlled exposure to sunlight (UV) is beneficial for most patients, a small minority may experience worsening of symptoms in bright sunlight.
- **Gender:** Statistics show that women are often more affected by the systemic and psychosocial impact of the disease.

Summary

There is no single cause. The disease occurs when a person with a certain genetic makeup encounters a stressor (infection, toxin, trauma), causing the immune system to lose “tolerance” to itself and trigger chronic inflammation.

Chapter 5: Clinical manifestations in adults and children

In 2026, medicine classified the forms of psoriasis according to the appearance of the lesions and their location. Although the typologies are the same for both age groups, the frequency and triggering factors can vary significantly.

1. Vulgar psoriasis (in plaques)

It is the most common form (80–90% of cases).

- **Appearance:** Red, well-demarcated plaques, covered with silvery-white scales.
- **Location:** Elbows, knees, scalp, lumbar area, and navel.
- **Mechanism:** The hyperactive immune system accelerates the life cycle of skin cells from 30 days to just 3–4 days, leading to their accumulation on the surface.

2. Guttate psoriasis (in drops)

The most common form among children and young people.

- **Manifestation:** Acute onset, with small pink-reddish lesions (like drops) on the trunk and limbs.
- **Main trigger:** Streptococcal infection of the throat (tonsillitis/pharyngitis).
- **Evolution:** Can be a transient form or can evolve into plaque psoriasis.

3. Inverse psoriasis (flexural)

It affects areas with folds, being more common in overweight people.

- **Location:** Armpits, groin area, under the breasts, intercostals.
- **Appearance:** Red, smooth, shiny plaques, without the specific scales.
- **Difficulties:** It is aggravated by sweating and friction, being often confused with fungal infections (mycoses).

4. Pustular psoriasis (severe)

A rare and serious form, characterized by blisters with sterile pus (non-infectious).

- **Palmarplantar Pustulosis:** Limited to the palms and soles.
- **Generalized Form (von Zumbusch):** A medical emergency. It presents with fever, chills, and extreme fatigue. Requires immediate hospitalization to prevent systemic complications.

5. Nail psoriasis

It affects up to 50% of patients and is a major indicator of the risk of psoriatic arthritis.

- **Clinical signs:**
 - **Pitting:** Small, punctate depressions on the nail surface (a “thimble” appearance).
 - **Oil spots:** Yellow-brown discolorations under the nail.
 - **Onycholysis:** Separation of the nail from the nail bed.
 - **Thickening and brittleness:** The nail becomes brittle and deformed.

6. Erythrodermic psoriasis

The rarest and most dangerous form, which can cover the entire body with a red, scaly rash, causing intense pain and disturbances in body temperature regulation.

Summary: Differences between children and adults (2026 edition)

Characteristics in children and adolescents in adults

Most common form: Guttate psoriasis (in drops), Psoriasis vulgaris (in plaques)

Main trigger: Bacterial infections (Streptococcus), stress, medications, smoking, alcohol

Specific location face and diaper area (in infants) elbows, knees, scalp, nails

Impact may completely resolve after treatment of the infection. It tends to be a chronic, lifelong condition

Important message

Regardless of age, psoriasis is a systemic disease. Nail damage or joint pain are alarm signals that indicate the need for a complex therapeutic intervention, targeting not only the skin, but the entire immune system.

SECTION II: Causes and Diagnosis

Chapter 6: Main manifestations of the disease (Skin, nails, joints)

Psoriasis manifests itself through a wide spectrum of symptoms, ranging from aesthetic discomfort to severe physical pain and limitation of mobility. Recognizing these signs is essential for a correct diagnosis and for monitoring the evolution of the disease under treatment.

1. Cutaneous manifestations (skin)

Skin lesions are the most common and recognizable signs:

- **Erythematousquamous plaques:** Red, well-demarcated and raised areas, covered with silvery-white scales (pearls). They appear preferentially on the elbows, knees, scalp, and lumbar area.
- **Severe cutaneous xerosis:** The skin becomes extremely dry, rough, and loses its elasticity. In advanced forms, the skin cracks and may bleed spontaneously.
- **Subjective sensations:** Patients frequently report intense pruritus (itching), burning sensation, or “tight skin”. Scratching these areas can lead to aggravation of the lesions (the phenomenon of self-maintaining inflammation).

2. Scalp manifestations

Scalp psoriasis is often confused with severe dandruff, but is distinguished by:

- **Thick plaques** that sometimes exceed the hairline.
- **Large, adherent scales**, which can cause major social discomfort.

3. Nail psoriasis (Nail involvement)

The nails provide valuable clues about the systemic severity of the disease:

- Structural changes: Thickened, deformed, or brittle nails.
- Specific signs: The appearance of small depressions (pitting), longitudinal lines, or the famous “oil stain” (a yellow-brown discoloration under the nail).
- Onycholysis: Progressive separation of the nail from the nail bed.

4. Systemic affection: Psoriatic arthritis

Approximately 30% of psoriasis sufferers develop joint manifestations:

- Pain and stiffness: Felt especially in the morning, in the hands, knees, ankles or spine.
- Dactylitis: Complete swelling of a finger (a “sausage finger” appearance).
- Chronic fatigue: A general state of exhaustion, caused by the immune system’s constant effort to maintain inflammation.

5. Synthesis of manifestations (Recapitulative table 2026)

Type of manifestation main features frequent location

Classic cutaneous red plaques, silvery scales, elbows, knees, back

Guidant small, pink drops, trunk, limbs

Inverse shiny redness, without scales axillae, inguinal folds

Pustular blisters with sterile fluid palms, soles

Erythrodermic redness all over the body generalized (emergency!)

Conclusion

Although red plaques with silvery scales remain the “hallmark” of psoriasis, the disease must be evaluated as a whole. Nail damage or joint pain are not separate problems, but evidence of internal inflammatory activity that requires an integrated therapeutic approach.

Chapter 7: Etiology of Psoriasis: Why does the disease occur?

Psoriasis is a chronic autoimmune disease resulting from a complex interaction between three main pillars: genetic predisposition, an overactive immune system, and environmental triggers.

1. Genetic and immune basis

- Inherited Predisposition: The risk of developing psoriasis increases significantly if there are affected first-degree relatives. Certain specific genes (HLA) determine this susceptibility.
- Immune Error: The immune system, instead of defending the body from pathogens, mistakenly attacks healthy skin cells. This error causes ultra-rapid cell regeneration (every 3-5 days, instead of 28-30), leading to the formation of specific plaques.

2. Triggers

These factors activate the disease in genetically predisposed individuals:

- Infections: Streptococcal tonsillitis is the most well-known trigger for guttate psoriasis.
- Emotional Stress: Chronic or severe stress disrupts the immune balance and can trigger or worsen the disease.
- Skin Injury (Koebner Phenomenon): Psoriasis can appear in areas with cuts, scrapes, insect bites, or sunburn.
- Lifestyle:
 - Smoking and Alcohol: Both are recognized risk factors that can worsen symptoms and reduce the effectiveness of treatments.
 - Obesity: Contributes to general systemic inflammation.
- Nutritional and climatic factors:
 - Vitamin D deficiency: Low levels are associated with increased severity of the disease.
 - Cold climate: Cold, dry weather can worsen skin symptoms.
- Specific medications: Lithium, beta-blockers, and certain antimalarial drugs.

Things to remember in 2026:

- Psoriasis is not contagious.
- It is a chronic disease: It evolves in cycles of exacerbation and remission.
- Holistic approach: Effective management requires a complete understanding of all these factors (genetics, immunity, and environment) and an integrated approach to treatment.

Chapter 8: The biological process: How the inflammatory cascade is triggered

Psoriasis is triggered by a communication error in the

immune system. Normally, skin cells are born in the deep layers and reach the surface in about 28–30 days. In the case of psoriasis, this process is dramatically shortened to just 3–7 days.

1. The biological process (inflammatory cascade)

- T-Cell activation: Due to genetic causes or under the influence of external factors, T-cells (lymphocytes responsible for defense) become hyperactive.
- Alarm signals: These T-cells release chemicals (cytokines) that send the skin a false signal of “wound” or “infection”.
- Accelerated overproduction: The body responds by massively producing new skin cells. Because old cells do not have time to naturally shed, new ones quickly accumulate on the surface, forming thickened and scaly plaques.

2. Determinants in 2026

Although the mechanism is immune, the actual trigger often requires a “fuse”:

- Genetic component: The risk is significantly higher in the case of a heredo-collateral history (affected parents or relatives).
- Koebner phenomenon: The appearance of lesions exactly in places where the skin has suffered trauma (cut, scratch or severe sunburn).
- Bio-Chemical triggers:
 - Infections: Streptococcal angina (pharyngitis) remains the main infectious trigger.
 - Medications: Lithium, beta-blockers (for hypertension), and antimalarials can “start” the disease.
 - Neuro-Endocrine factors: Intense mental stress and hormonal changes disrupt the immune balance.

3. Evolution and location

- Preferential Areas: Although they can appear anywhere (including on the nails), lesions tend to cluster on the scalp, elbows, knees, and lower back.
- Cyclic Character: The disease does not have a linear evolution. In 2026, managing psoriasis means maximizing periods of remission (when the skin is clear) and minimizing episodes of exacerbation (rash).

Conclusion

Psoriasis is not a simple skin disease, but the result of a “programming error” of the immune system. Understanding this mechanism of cellular overproduction is the first step towards choosing a treatment that does not only targets the external symptom, but the internal cause.

Chapter 9: Dynamics of expansion: Why do lesions spread?

Many patients wonder why, after a period of stability, the disease begins to spread to new areas of the body. The expansion of psoriasis is not an infection that is “taken” from one area to another, but is the result of an intensification of the activity of the immune system under the pressure of internal and external factors.

1. Mechanism of propagation

Expansion occurs when the body’s tolerance threshold is exceeded.

- Cascade inflammation: When the immune system is hyperactive, it releases cytokines that circulate in the blood. This explains why an inflammation started in one place (for example, a throat infection) can manifest itself as new plaques on the legs or back.
- Acceleration of the cell cycle: As the disease worsens, the skin’s regeneration cycle is maintained at the abnormal rate of 3–4 days, forcing the accumulation of cells over increasingly larger surfaces.

2. Factors that favor expansion (aggravation triggers)

Expansion is almost always “fed” by external factors that maintain the immune system’s alert state:

- Cumulative trauma (Koebner effect): Any aggression to the skin (tattoos, scratches, sunburns) acts like a magnet for immune cells, generating new plaques exactly at the site of the trauma.
- Stress and exhaustion: Emotional stress raises cortisol levels, which, over time, disrupts the immune response and leads to explosions of new lesions.
- Lifestyle factors:
 - Smoking and alcohol: Act as “fuel” for inflammation, favoring expansion and reducing the response to treatment.
 - Obesity: Adipose tissue produces its own inflammatory substances, which accelerate the spread of plaques.
- Drug reactions: The introduction of new treatments for other conditions (lithium, beta-blockers, NSAIDs) can trigger the sudden expansion of pre-existing lesions.

3. Risk of generalized forms

If the expansion process is not controlled, there is a risk of evolution to severe forms:

- Erythrodermic psoriasis: This is the stage in which the inflammation covers more than 90% of the body surface. This is a medical emergency in 2026, because the skin loses its ability to regulate temperature and protect the body against infections.

Conclusion

Psoriasis expands when the delicate balance between genetics and the environment is broken. To stop the expansion, it is not enough to treat only the new spots that appear; the causes that “feed” the inflammation from within must be identified and eliminated.

Chapter 10: The importance of specialized medical consultation

Consulting a doctor in the case of psoriasis is mandatory, because this condition goes beyond the aesthetic barrier of the skin. Being an autoimmune, chronic, and systemic disease, it requires a long-term management strategy to control internal inflammation and prevent the degradation of other organs.

1. Diagnosis of certainty and differentiation

The doctor’s first role is to confirm that it is psoriasis and to exclude other conditions with similar manifestations (such as seborrheic dermatitis, mycoses, or discoid lupus). A wrong diagnosis leads to ineffective treatments that can worsen the patient’s condition.

2. Prevention and monitoring of complications

Untreated or incorrectly treated psoriasis can develop into severe complications:

- Psoriatic arthritis: The doctor monitors signs of stiffness and joint pain to prevent irreversible destruction of the joints.
- Systemic comorbidities: In 2026, it is clear that psoriatic inflammation increases the risk of type 2 diabetes, cardiovascular disease, and obesity. Regular medical check-ups (blood tests, blood pressure monitoring) are vital.

3. Multidisciplinary approach

Modern psoriasis management often involves a team of specialists coordinated by a dermatologist:

- Rheumatologist: To evaluate the joints.
- Cardiologist and diabetologist: To manage metabolic risks.
- Psychologist/Psychiatrist: To treat the emotional impact (depression, anxiety, social isolation).

4. Personalization of therapy and identification of trigger factors

Each patient is unique. The doctor develops a personalized plan that may include:

- Various therapies: From topical solutions and phototherapy to state-of-the-art systemic or biological drugs.
- Trigger identification: The specialist helps the patient

identify what triggers their eruptions (hidden infections, stress, certain contraindicated medications).

- Nutritional counseling: Adjusting lifestyle and diet to support the immune system.

5. Improving quality of life

A correct treatment plan is not only aimed at “cleaning” the skin, but also at the patient’s social and professional reintegration. The doctor periodically evaluates progress and adjusts doses to minimize side effects and maximize remission periods.

Conclusion

Psoriasis is not “just a skin problem”. It is a challenge for the entire body that requires constant medical supervision. In 2026, the success of treatment depends on integrating medical advice with rigorous patient discipline in terms of nutrition and lifestyle.

Chapter 11: Psoriasis treatment in dermatology today

In 2026, psoriasis treatment in dermatology clinics follows a rigorous protocol, adapted to the severity of the disease and the individual patient profile. The main objective of these therapies is the management of symptoms by controlling inflammation and slowing down accelerated cell proliferation.

1. Topical therapies (for mild and moderate forms)

These represent the first line of attack, applied directly to the skin in the form of creams, ointments, or lotions:

- Corticosteroids: Quickly reduce inflammation and itching.
- Vitamin D Analogs: Slow down the growth of skin cells.
- Retinoids and Salicylic Acid: Help eliminate scales and thin the plaques.
- Emollients: Intense hydration is mandatory to maintain the skin barrier intact.

2. Phototherapy (for moderate and severe forms)

Uses medically controlled Ultraviolet (UV) light to suppress local immune activity:

- Narrowband UVB: The most widely used form of phototherapy in 2026, safe and effective.
- PUVA therapy: Combines the administration of a photosensitizing drug (psoralen) with exposure to UVA rays.
- Excimer laser: Allows targeted treatment of plaques, without exposing healthy skin to radiation.

3. Classic systemic treatments

These are orally administered drugs that act on the entire body:

- Methotrexate and cyclosporine: Immunosuppressants that require rigorous monitoring of liver and kidney function through periodic tests.
- Acitretin: A vitamin A derivative often used in pustular forms.
- Small molecules (Apremilast): A modern oral therapy that inhibits an enzyme responsible for intracellular inflammation.

4. Biological therapies (The gold standard for severe forms)

Represent the greatest advancement in contemporary medicine. They are injectable drugs that selectively target immune system proteins:

- TNF-alpha, IL-17, and IL-23 inhibitors: They act like “guided missiles”, specifically blocking the cytokines that cause psoriasis, with extremely high efficacy in clearing the skin.

5. Lifestyle approach in 2026

Modern dermatology recognizes that drugs are more effective when supported by preventive behavior:

- Nutrition: An anti-inflammatory diet rich in Omega-3 (fatty fish), antioxidants from fruits and vegetables, and olive oil is recommended. It is essential to avoid ultra-processed foods, refined sugars, and red meat.
- Risk Factor Control: Stress management through relaxation techniques and the complete elimination of smoking and alcohol are central pillars for maintaining remission.

Conclusion

While current dermatological treatments offer remarkable control over the appearance of the skin, they require long-term administration and careful monitoring. Integrating these solutions with a balanced diet and a healthy lifestyle is the key to success in 2026.

SECTION III: Integrated Management and Individual Treatment of Psoriasis

Chapter 12: How to treat your psoriasis yourself and correctly

To make it easier to understand, this chapter is written in the form of questions and answers.

1. Deniplant, is it a medicine?

Answer: Deniplant is not a medicine. Deniplant is a registered trademark in Romania and the European Union. Under this brand, Deniplant natural remedies (Deniplant Nutraceuticals) are produced and marketed worldwide, addressing autoimmune, metabolic, and neurological conditions.

2. What is Deniplant tea for psoriasis?

Answer: Deniplant tea is a patented natural remedy, a nutraceutical, because it is a food with a dual role, nutrition and health, created by Gheorghe Giurgiu, who managed to cure himself of psoriasis without ointments or medications, just by drinking this tea. You can read his story from suffering to healing here.

This natural treatment contributes to reducing inflammation and regenerating the skin, all thanks to natural ingredients, without chemicals or other substances that can cause adverse effects.

Deniplant tea is a powder of cultivated medicinal plants and spontaneous flora in combination with fruit tree buds, from which a tea is prepared, with lemon and honey, a tea that can be drunk daily for several months. Deniplant tea is approved for marketing as a nutritional supplement.

3. How does deniplant tea help heal the disease?

Answer: Deniplant-psoriasis tea, being a natural genetic immunomodulator of the CARD 14 gene and the intestinal and cutaneous microbiome, treats the internal causes that produce and maintain psoriasis. Psoriasis lesions on the skin heal without ointments or other medications, without a diet, removing the dysbiosis of the microbiome and strengthening the intestinal barrier, so that the metabolites produced by the microbiome no longer pass beyond the intestinal wall and cause the onset of psoriasis and other autoimmune diseases.

4. What are the benefits of Deniplant tea?

Answer:

- Reduces inflammation and soothes affected skin.
- Helps with cell regeneration, also intervening in the healing process of damaged tissues.
- Prevents recurrence of the disease by removing the dysbiosis of the epithelial microbiome.
- Does not require a strict diet, being easy to integrate into the daily routine.
- Can be used preventively, including in children, to avoid the appearance of psoriasis.

Contains:

- cultivated medicinal plants=35%
- Plants from the spontaneous flora=25%
- Fruit tree buds=15%
- Fruit tree flowers=15%
- Berries=10%

Form of presentation: Dry and ground powder packaged in tea bags of 1 gram each. 30 bags/pack

Indicated in the following conditions:

- Psoriasis (scalp, elbows, generalized, pustular, capillary, nails, palmoplantar)

Administration: 750 ml of tea to be drunk daily

Duration of treatment: In relation to the evolution of the disease {4-6-12 months}

Contraindications: None.

Adverse reactions: They did not occur with long-term use.

Shelf life: 2 years.

Check the date on the leaflet; keep it in the dark and at a constant temperature.

Other details: No ointments or other medications, no diet. Tips for those who want to use deniplant-psoriasis tea can be found here.

How to prepare and use:

In a liter of water, put a sachet of herbs. Add a lemon (100-150 g) with the peel after washing it well, cut into slices.

Boil until 750ml remains (boiling time is 15-20 minutes after it comes to a boil).

After it has cooled, remove the lemon and sweeten to taste with sugar or honey (preferably honey). If sugar is used for sweetening, it will be put in the water along with the herbs and lemon, so that it also goes through the boiling process.

The entire 750 ml quantity will be drunk during the day in several small doses. Also on the same day, the boiled lemon will be eaten, with the peel intact, sweetened as desired.

5. If I drink this tea, is it possible to cure psoriasis?

Answer: Yes, this is possible. To cure psoriasis, you must first find out what psoriasis is. Research conducted after 2010 has shown that psoriasis is an autoimmune metabolic disorder caused by dysbiosis of the intestinal and skin microbiome and a mutation of the CARD14 gene. Deniplant tea has proven to be a natural genetic immunomodulator that can resolve dysbiosis of the microbiome, and the skin heals itself from psoriasis. In the Library of the Deniplant-Aide Sante Biomedicine Center, you can find works in this regard.

6. Are ointments or other medicines for internal or external use also needed?

Answer: From my own experience and that of those who have used Deniplant so far to see if the plants work or not, it is good to discontinue the administration of ointments or other medicines for psoriasis.

Warning: Whether you use Deniplant tea or not, when discontinuing the ointments or medications used (especially those with cortisone or immunobiological), due to the reaction called REBOUND in medical terms, new spots or red spots may

appear in other areas where you did not have lesions, but these will not evolve much and will disappear first.

If an ointment is used in parallel, the skin can heal on the surface faster than the resolution of the internal causes, and you will fool yourself, the lesions appearing again when discontinuing the ointments, until the internal deficiencies are definitively resolved.

If you cannot resist them, you can initially use any cosmetic cream that does not contain medications.

7. Is a specific diet or dietary restrictions needed?

Answer: The diet can speed up the elimination of toxins from the body, but it does not help resolve the internal causes. For this reason, positive developments were seen in those who followed the diet, but there were also negative developments when the diet was interrupted. A person must have a balanced diet. You can use the EGO app to track your body's reactions according to nutrition, health, and sports.

8. What happens if I scratch and break the scales?

Answer: Every time you intervene on them, you prolong the healing process, and there is a tendency for the damaged surface to expand. While you drink the tea, they will fall off more and more easily, and this is a sign that the body has responded positively.

9. Can I take a shower or bath if I have wounds and scabs on large surfaces?

Answer: It is recommended to take a shower daily, but not with hot water, and do not rub the skin after drying to remove the scales, because you will prolong the healing process.

10. Can I take spa treatments if I have psoriasis?

Answer: Hydro-mineral treatment is an adjunct to local treatment. Mineral water has a local and general effect on some dermatoses.

In Romania, the most used mineral waters are the following:

- Arsenic waters. The most famous resorts are in the Suceava region.
- Alkaline, arsenical, ferruginous, and radioactive waters in the Transylvanian area: Sângiorz, Tuşnad, or Buziaş.
- Sulfurous and iodized waters: Baile Herculane, Govora, Călimăneşti, Pucioasa, Someşeni, Căciulata, Bazna.
- Hypothermic, oligometallic, siliceous, and radioactive waters: Baile 1Mai, Bazna.
- Chloride-sodium waters: Ocnele Mari, Ocna Sibiului, Slănic Prahova, Sovata.

11. What role does sleep play in skin repair?

Answer: Sleep is crucial for skin repair, because during the night the body regenerates cells, increases collagen production

(for firmness), improves blood circulation (delivering nutrients) and repairs damage, acting as a “beauty sleep” that supports the elasticity, brightness and barrier function of the skin, and lack of sleep leads to premature aging and a dull appearance.

What happens to the skin during sleep:

- Cellular regeneration: Skin cells repair and regenerate three times faster than during the day, a process accelerated by growth hormone.
- Collagen synthesis: The body produces more collagen, essential for skin elasticity and firmness.
- Improved circulation: Blood flow to the skin increases, carrying oxygen and nutrients to repair damage.
- Hydration: Helps maintain the skin barrier, reducing dehydration, although there is a natural transepidermal water loss.
- Inflammation reduction: The body recovers from daily stress (pollution, UV, free radicals).

Impact of lack of sleep:

Decreased collagen production affects elasticity.

Dull, pale, and lifeless skin.

Dark circles and puffiness due to fluid imbalance and visible blood vessels.

Slower recovery from environmental stress.

How to support skin recovery:

- Nighttime routine: Use specific care products that support nighttime regeneration.

Quality sleep: Give the body enough time to recover, at least 7-9 hours a night, to support natural repair processes.

12. Is acupuncture indicated in psoriasis?

Answer: Renowned acupuncture specialists conducted a study entitled: “Acupuncture and Phytotherapy in Psoriasis - Clinical Observations.”

- The localized form of psoriasis responds very well to associated treatment (acupuncture + Deniplant tea);
- The generalized form is the variant in which the energy imbalances are complex, on a background of low immunity, requiring long-term treatment;
- A combined treatment, acupuncture + deniplant tea, offers more chances to address some pathophysiological mechanisms that induce the onset and relapses of the disease, for both forms of manifestation of the disease.

13. Are there other complementary treatments for psoriasis?

Answer: Yes, multiple complementary treatments can help psoriasis sufferers.

14. Can I talk to the tea manufacturer?

Answer: You can talk to Gheorghe Giurgiu, the manufacturer of Deniplant tea, or to the doctors at the Deniplant Biomedicine Center – Aide Sante, this way you can find out other things that were not included here, and you can make the right choice. You can also get in touch with other patients who use or have used the tea by accessing the discussion forum created for this purpose on the deniplant.our website.

15. Is it possible to return a purchased product?

Answer: Yes, and for this, proceed as indicated below:

Conditions for returning the package and money according to art. 6, paragraph h, and art. 11, paragraph 1 of GEO 34/2014 and the Deniplant Terms and Conditions

1. The fees generated by the transport of the package to the buyer and return to Deniplant, by Romanian post or courier, will be borne by the buyer and will be deducted from the amount that will be returned to you.

2. Within 14 days of receiving the package, you have the opportunity to return it with a receipt and with the contents intact, by Romanian post or courier, to the address indicated on the site, where you will receive its value less the transport fees, by post or directly to your bank account.

16. What if I am dissatisfied with the results?

Answer: There have been patients whose bodies did not respond positively, or responded more slowly, for several reasons, and that is why we provide the SUPREME GUARANTEE

To benefit from a refund after using the remedies for 30 days, it is necessary that before and after using the remedies, you are recorded and consulted by doctors at the Deniplant Biomedicine Center – Aide Sante to determine the inefficiency.

You cannot benefit from a refund

1. If 15 days have passed since purchasing the product.
2. If the product has been unsealed and consumed
3. You have not returned the product with the receipt

Return requests in all cases will be made by email to deniplant@gmail.com or by phone or SMS to: 0744827881.

SECTION IV: Conventional Treatment and Risks

Chapter 13: Mechanism of action of ointments and risk of rebound

Ointments are the basis of local treatment in psoriasis, with the role of managing the visible manifestations on the skin surface. They act by eliminating scales, reducing local inflammation, and deep hydration, being essential especially in mild and moderate forms.

1. Types of ointments and their functioning

Dermatological medicine uses four main categories of topical agents:

- Keratolytic agents (Urea, Salicylic Acid):
 - Action: Soften and loosen scales (thick scales), facilitating their natural removal.
 - Benefit: Smooth the skin and allow other active substances to penetrate the deeper layers.
 - Topical corticosteroids (Cortisone derivatives):
 - Action: They are powerful anti-inflammatories that quickly stop redness and itching.
 - Caution: Although they provide spectacular improvement, they must be used strictly under medical supervision to avoid skin thinning (atrophy) or local addiction.
- Emollients and moisturizers:
 - Action: Restore the skin's lipid barrier and prevent water evaporation.
 - Benefit: Reduce the sensation of "tight skin" and prevent bleeding cracks.
- Vitamin D Analogs and Retinoids (Calcipotriol, Tazarotene):
 - Action: Regulate the rate of cell division, trying to bring the skin's life cycle closer to normal.

2. Limitations of topical treatment

The patient needs to understand that ointments treat the effect, not the cause. They clean the skin on the surface, but do not intervene in the internal mechanisms that produce the disease. It has been found that, over time, the skin can develop a tolerance to these substances (tachyphylaxis), their effectiveness progressively decreasing, which can lead to more frequent and more severe relapses.

3. The rebound phenomenon: The trap of abrupt discontinuation

The Rebound Effect is the aggressive reappearance of symptoms immediately after stopping treatment. In psoriasis, it manifests itself through the explosion of new lesions on much larger surfaces than the initial ones.

- Why does it occur? The skin gets used to the presence of cortisone or other local immunosuppressive agents. When administration is stopped suddenly, the immune system reacts excessively, triggering a much stronger paradoxical inflammation.
- Dangers: A severe rebound can transform localized psoriasis into a generalized or even erythrodermic form (medical emergency).

4. How to avoid the rebound effect in 2026?

1. Tapering: Never stop cortisone treatment suddenly. The dose and frequency of application should be reduced progressively, under the guidance of a doctor.
2. Transition to Natural Solutions: As inflammation subsides, it is recommended to gradually replace synthetic drugs with natural nutraceuticals and emollients that support regeneration without creating addiction.
3. Internal Approach: To prevent relapse, it is mandatory to simultaneously work on balancing the microbiome and the immune system through nutrition and lifestyle, so that the body no longer "demands" external medicine.

Conclusion

Ointments are a valuable ally for immediate comfort, but their use should be done with caution. In the absence of a strategy that targets internal causes, the risk of the disease returning stronger under the rebound effect is major.

Chapter 14: Cortisone in the treatment of psoriasis – Benefits and risks

Corticosteroids (derivatives of cortisone) remain an essential pillar in clinical dermatology due to their ability to rapidly suppress inflammation. However, in the medicine of 2026, their use is strictly regulated to prevent severe adverse effects and skin addiction.

1. Benefits and mechanism of action

Cortisone acts as a powerful anti-inflammatory by inhibiting the release of cytokines (substances that maintain inflammation).

- Rapid Improvement: Visibly reduces redness, itching, and scaling in a short time.
- Adaptability: There are seven potency classes (from I – very strong, to VII – weak). This classification allows the doctor to prescribe a strong cream for the elbow area, but a very gentle one for the face or areas with thin skin.

2. Risks and adverse effects (Long-term use)

Although effective, cortisone can become dangerous if used without medical supervision:

- On the skin: It produces skin atrophy (thinning of the skin), the appearance of stretch marks, visible blood vessels (telangiectasias), and delays wound healing.
- Impact on the Microbiome: Prolonged use can disrupt the balance of the skin microbiome, leaving the skin vulnerable to bacterial or fungal infections.
- Systemic Effects: In the case of prolonged oral or injectable administration, the following may occur:

- Metabolic changes: Weight gain, water retention, “full moon face”.
- Mental disorders: Anxiety, irritability, or insomnia.
- Vascular problems: High blood pressure and increased risk of diabetes.

3. Golden rules for correct use

1. Do not self-medicate: Never use a cortisone cream prescribed for someone else. The wrong potency can worsen the disease.
2. Short-term treatment: It is recommended to use short “cures”, followed by breaks, to allow the skin to recover.
3. Gradual discontinuation scheme: Abruptly stopping treatment is the main cause of the rebound effect (severe worsening of psoriasis). The dose should be gradually reduced, as directed by the doctor.
4. Medical monitoring: Any systemic treatment (pills or injections) requires constant medical supervision to monitor blood pressure and metabolic parameters.

Key message

Cortisone is an excellent “fire extinguisher” for acute episodes, but it is not a solution for curing the disease. Since psoriasis is a chronic condition, treatment must evolve towards solutions that regulate the immune system from within, without attacking the natural skin barrier.

Chapter 15: Phototherapy: The role of UV Rays (UVB-nb, PUVA, Laser)

Ultraviolet (UV) light is a basic therapeutic method in the management of moderate-severe psoriasis. It works by modulating the local immune response and slowing the accelerated rate of skin cell division.

1. Types of phototherapy used in 2026

Current technology allows for controlled and targeted exposure, minimizing the risks of radiation:

- Narrowband UVB (Narrowband UVB - 311 nm): It is the gold standard in phototherapy. It uses a specific wavelength, maximizing the anti-inflammatory effect and reducing the risk of sunburn.
- PUVA therapy (Psoralen + UVA): Combines a photosensitizing drug (Psoralen) with UVA rays, which penetrate the deeper layers of the dermis. It is reserved for severe cases or cases resistant to other therapies.
- Excimer laser: It emits a concentrated beam of UVB directly on the psoriatic plaques. The major advantage is the protection of healthy skin around the lesions, making it ideal for difficult areas (knees, elbows, scalp).
- Heliotherapy (Natural exposure): Controlled exposure

to the sun remains beneficial, but must be done with caution to avoid burns, which can trigger the Koebner phenomenon (spread of the disease).

2. Mechanism of action: How does light “heal”?

UV rays not only cleanse the skin, but also produce profound biological changes:

- Local immunosuppression: Reduces the activity of hyperactive T cells in the skin.
- Proliferation control: Inhibits DNA synthesis in keratinocytes, thus stopping the accumulation of scales.
- Apoptosis: Induces the programmed death of inflammatory cells that maintain the plaques.
- Anti-inflammatory effect: Stimulates the release of protective cytokines, which calm redness and itching.

3. Benefits and protocols

- Efficacy: It is one of the safest methods to achieve a lasting remission for extensive psoriasis.
- Frequency: It usually requires 2-3 sessions per week, under medical supervision, to adjust the energy dose (mJ/cm²) according to the patient’s skin phototype.

4. Risks and safety measures

Like any strong medical treatment, phototherapy involves precautions:

- Short-term effects: Redness (erythema), burning sensation, severe skin dryness, and temporary itching.
- Long-term effects: Cumulative exposure can accelerate skin aging (photoaging) and increase the risk of skin cancer (carcinomas). For this reason, doctors keep strict records of the total UV dose received by the patient throughout his or her life.

2026 guideline recommendation

Phototherapy should be performed exclusively in specialized centers, equipped with calibrated equipment. Self-medication by using cosmetic tanning is strictly contraindicated, as the light spectrum in commercial tanning beds is not therapeutic and significantly increases the risk of skin degradation.

Chapter 16: Systemic therapies: Methotrexate (Indications and side effects)

Methotrexate Ebewe is administered under the skin, into a muscle, or into a vein; it should not be administered intravenously in children and adolescents. Treatment with Methotrexate Ebewe in rheumatoid arthritis, juvenile idiopathic arthritis, psoriasis vulgaris and psoriatic arthritis is long-term.

Methotrexate tablets act by competitive inhibition of the enzyme dihydrofolate reductase and thus inhibit DNA

synthesis. It has not yet been clarified whether the efficacy of methotrexate in the treatment of psoriasis, psoriatic arthritis and chronic polyarthritis is determined by the anti-inflammatory or immunosuppressive effect and to what extent the methotrexate-induced increase in the concentration of extracellular adenosine in areas affected by inflammation contributes to these effects.

Before using this medicine, read carefully what side effects may occur.

Side effects: Side effects usually depend on the dose and duration of treatment with methotrexate. However, severe side effects, which may lead to interruption or permanent cessation of treatment, may occur even at low doses. Side effects can occur at any time during treatment. Most side effects are reversible, if diagnosed early.

Side effects are not always completely reversible after permanent cessation of methotrexate treatment.

However, some of the severe side effects mentioned below can lead to sudden death in very rare cases. There is also a risk that certain side effects may occur after a while.

The doctor should be informed if the patient experiences any of the following symptoms, as these could be signs of a serious, life-threatening side effect. The doctor may decide to stop treatment.

- Allergic reactions such as sudden wheezing, difficulty breathing, swelling of the eyelids, face, or lips (which may cause difficulty swallowing), rash or itching (especially affecting the whole body), as these could be signs of a severe allergic reaction.
- Lung problems (symptoms may include: feeling generally unwell, dry, irritating cough, shortness of breath, shortness of breath at rest, chest pain, or fever); these could be signs of lung infection (pneumonia, pneumonitis, alveolitis).
- Symptoms of liver damage, such as yellowing of the skin and whites of the eyes (jaundice), dark urine, nausea, vomiting, loss of appetite, pain in the right side of the abdomen and itching.
- Symptoms of kidney damage, such as swelling of the hands, ankles, and feet or decreased or no urination - these may be symptoms of kidney failure.
- Symptoms of infections, such as fever, pain, sore throat; methotrexate may reduce the ability to fight infections. Severe infections, such as a special form of lung infection (*Pneumocystis jirovecii* pneumonia) and sepsis, may occur.

Fever, sore throat, mouth ulcers, feeling generally unwell and tired, nosebleeds, or small red spots on the skin, as these could be signs that the bone marrow is not working properly.

- Mouth ulcers.

- Pain in the stomach area (upper abdomen), nausea, vomiting or fever; these could be caused by inflammation of the pancreas.
- Severe abdominal pain, fever, nausea, vomiting, severe diarrhoea, blood in the stool, or an imbalance in bowel function as these could be signs of serious complications in the digestive tract, for example ulcers or perforation of the stomach or intestines.
- Severe skin reactions (Stevens-Johnson syndrome, toxic epidermal necrolysis, erythema multiforme): manifestations with red spots or spots on the body, often with blisters in the centre.

These skin reactions are generally associated with feeling unwell and fever. The skin rash may worsen and develop into peeling or blistering of the skin, which can be life-threatening.

- Symptoms associated with a blood clot (thromboembolic event), such as chest pain or pressure, pain in the arms, back, neck, or jaw, difficulty breathing, numbness or weakness on one side of the body, difficulty speaking, and dizziness.
- Cough, chest pain, sudden shortness of breath, or coughing up blood; these may be symptoms of a pulmonary embolism (blood clot in the lungs).

The following side effects have also been reported:

Very common:

- Changes in the number of white blood cells (leucocytopenia) and platelets (thrombocytopenia).
- Headache, spinning sensation (vertigo).
- Cough.
- Loss of appetite, diarrhoea (especially in the first 24-48 hours of methotrexate treatment, stomach pain, nausea (feeling sick), vomiting, inflammation and ulcers in the mouth and throat (especially in the first 24-48 hours of methotrexate treatment).
- Increase in liver enzymes.
- Hair loss.
- Decreased creatinine excretion (can be detected by a test performed by a doctor and is a signal for impaired kidney function).
- Tiredness, drowsiness.

Common

- Shingles (herpes zoster).
- Changes in the number of red blood cells (anemia); bone marrow damage, which can lead to a sudden decrease in the number of white blood cells (agranulocytosis) or all blood cells (pancytopenia).

- Drowsiness, tingling.
- Red eyes (conjunctivitis).
- Skin rash, redness, itching, increased sensitivity of the skin to sunlight, skin ulcers.

Uncommon:

- Higher risk of infections/inflammation due to a suppressed immune system.
- A certain type of cancer called lymphoma, which may go away after stopping methotrexate.
- Diabetes.
- Depression.
- Paralysis on one side of the body, confusion.
- Inflammation of blood vessels (vasculitis), allergic vasculitis.
- Formation of scar tissue in the lungs (pulmonary fibrosis), fluid around the lungs (pleural effusion).
- Liver damage (hepatotoxicity), fatty degeneration, fibrosis (growth of connective tissue), cirrhosis (tissue transformation, with hardening and elimination of normal liver structures), decrease in serum albumin (a type of blood protein).
- Severe toxic reactions: formation of groups of blisters, resembling skin lesions associated with the herpes virus (herpes zoster).
- Hives, darkening of the skin, lumps under the skin (nodules), impaired wound healing.
- Joint or muscle pain, brittle bones (osteoporosis).
- Inflammation and ulceration of the bladder (possibly with blood in the urine); problems with bladder emptying, painful urination, little or no urine.
- Malformations of the fetus.
- Inflammation and ulceration of the vagina.
- Fever.

Rare

- Anemia associated with enlarged red blood cells (megaloblastic anemia).
- Mood changes, transient perceptual disturbances.
- Paralysis, speech problems, damage to the white matter of the brain (leukoencephalopathy).
- Vision disturbances (may be severe); clots in the veins of the retina (retinal thrombosis).
- Low blood pressure (hypotension), complications

resulting from blood clots in the veins and arteries (thromboembolic events).

- Sore throat (pharyngitis), shortness of breath.
- Inflammation of the digestive tract, inflamed gums, dark or black stools.
- Inflammation of the liver (hepatitis).
- Acne, bruising, darkening of the nails, loss of nails.
- Stress fracture.
- Increase in blood urea, creatinine, and uric acid (signs of kidney problems), increase in blood urea nitrogen (azotemia).
- Miscarriage.
- Changes in menstrual cycle and decreased sperm production, which return to normal after the end of treatment.
- Lymphoproliferative disorders (excessive increase in the number of white blood cells).

Very rare

- Inflammation of the liver caused by the herpes virus (herpes simplex hepatitis).
- Fungal infections (histoplasmosis, cryptococcosis), viral infections (cytomegalovirus infections, including pneumonia), disseminated herpes simplex, bacterial infections (nocardiosis).
- Anemia caused by insufficient formation of red blood cells (aplastic anemia), reduction in the number of white blood cells (eosinophilia, neutropenia), enlarged lymph nodes in the head and neck, armpits, and groin (partially reversible), uncontrolled increase in the number of lymphocytes (partially reversible).
- Low number of antibodies in the blood (hypogammaglobulinemia).
- Muscle weakness and pain in the arms and legs, metallic taste, acute aseptic meningitis with symptoms such as severe headache, fever, nausea, vomiting, and loss of consciousness, damage/damage to the cranial nerves.
- Swelling around the eye, inflammation of the eyelids, increased tear production, increased sensitivity to light, temporary blindness, loss of vision.
- Inflammation of the lining of the heart (pericarditis), fluid between the linings of the heart (pericardial effusion), failure of the heart to fill due to an effusion in the sac around the heart (pericardial tamponade).
- Chronic damage to the lung structure, asthma-like reactions, including coughing, difficulty breathing, and abnormal lung function test results.

- Vomiting blood.
- Breakdown of liver cells (acute hepatic necrosis), liver failure.
- Infection of hair follicles (furunculosis), visible and permanent enlargement of capillaries under the skin (telangiectasia), and inflammation of the nail bed.
- Blood in the urine, increased protein excretion in the urine.
- Death of the foetus.
- Numbness or tingling sensation/less sensitive to stimuli than normal.
- Formation of fewer eggs and spermatozoa, infertility, menstrual cycle disorders, loss of sexual appetite, impotence, vaginal discharge, breast enlargement in men (gynecomastia), bleeding in the lungs (has been reported for methotrexate used in patients with underlying rheumatological diseases).

Frequency unknown

- Pneumonia, reactivation of hepatitis B, worsening of hepatitis C.
- Nervous system damage (neurotoxicity), inflammation of the brain membrane (arachnoiditis), paralysis of the legs (paraplegia), stiffness of the whole body (stupor), lack of coordination of muscle movements, dementia, increased pressure of the cerebrospinal fluid with symptoms such as headache, nausea, vomiting, high blood pressure, confusion.
- Bleeding in the lungs (pulmonary alveolar hemorrhage).
- Chest pain.
- Oxygen deficiency (hypoxia).
- Inflammation of the peritoneum, characterized by pain in the abdomen and sensitivity to pressure; inflammation of the tongue.
- Drug reaction with rash all over the body and an increase in eosinophils (a special type of blood cell) in the blood (DRESS syndrome), inflammation of the skin.
- Bone tissue dying (osteonecrosis).
- Loss of bladder and sex organ function (urogenital dysfunction).
- Tremors.
- Swelling.
- Redness and peeling of the skin.
- Bone damage in the jaw (due to excessive white blood cell growth).

See the package leaflet that comes with methotrexate for the full list of side effects.

Chapter 17: Biological therapies in psoriasis

Biological treatment represents the most modern and precise method of controlling moderate–severe psoriasis. Unlike classic systemic drugs (such as methotrexate), which suppress the entire immune system, biological agents are proteins synthesized in the laboratory that selectively target only certain links of the inflammatory process.

1. Mechanism of action

Biological drugs block specific proteins called cytokines (TNF-alpha, IL-17, IL-23), which are responsible for transmitting inflammation signals and accelerating skin cell division. By neutralizing these “messengers”, biologicals can completely clear the skin in many cases.

2. Who is eligible in 2026?

According to current medical protocols, a patient can access biological therapy if:

- Suffering from moderate or severe psoriasis vulgaris (plaque) for at least 6 months.
- Failure of standard therapies: Did not respond to, had contraindications, or developed intolerance to at least two conventional systemic therapies (Methotrexate, Cyclosporine, Acitretin, or PUVA phototherapy).

3. Types of common biologic agents

- TNF-alpha inhibitors: Adalimumab, Etanercept, Infliximab.
- IL-12/23 inhibitors: Ustekinumab.
- IL-17 inhibitors: Ixekizumab, Secukinumab (Cosentyx), Bimekizumab.
- IL-23 inhibitors: Guselkumab, Risankizumab.

4. Administration and monitoring

- Route of administration: Most are injectable (subcutaneous), in the form of prefilled pens for self-administration, or infused (intravenously) in a hospital setting.
- Frequency: Varies from weekly administration to once every 3 months, depending on the molecule.
- Monitoring: Evaluation is mandatory at 3, 6, and 12 months to verify efficacy (PASI score) and patient safety.

5. Side effects and risks

Since biologics modulate immunity, their use involves rigorous medical supervision:

Common side effects:

- Injection site reactions (redness, slight swelling).
- Increased risk of mild upper respiratory tract infections (cold, rhinitis).
- Severe side effects (Rare):
- Opportunistic infections: Risk of tuberculosis reactivation (QuantiFERON test is mandatory before initiation) or serious fungal infections.
- Paradoxical reactions: Appearance of new forms of psoriasis or eczema under treatment.
- Hepatic and hematological problems: Changes in liver enzymes or decreased white blood cell/thrombocyte count.
- Neurological and neoplastic risks: Rarely, they may be associated with worsening of demyelinating diseases (e.g., multiple sclerosis) or a slightly increased risk of certain types of skin cancer.

Conclusion

In 2026, biological therapies offer the chance of a normal life for patients with disabling forms of the disease. However, due to high costs and the risk of immunosuppression, they remain a “second-line” solution, used when other therapeutic options, including complementary ones, have not yielded results.

Chapter 18: Common mistakes in psoriasis management

Success in managing psoriasis depends not only on the treatment chosen, but also on avoiding common mistakes that can nullify any medical progress. Identifying these mistakes is the first step towards lasting remission.

1. Diagnostic and treatment errors

- Self-diagnosis and Self-medication: Applying over-the-counter creams or creams recommended by unqualified people. Psoriasis can mimic other diseases, and incorrect treatment can inflame the skin even more.
- Use of medications with severe side effects: Prolonged use of synthetic substances that, although they temporarily clear the skin, can damage the functions of the liver, kidneys, or immune system in the long term.
- Premature discontinuation of natural treatments: Many patients stop taking natural remedies as soon as they see an improvement. In reality, treatment should be continued until the internal causes are eliminated and all affected areas are completely regenerated.

2. Local care mistakes

- Forced removal of scales (scratching): This is one of the most serious mistakes. Traumatizing the plaques by scratching leads to bleeding and triggers the Koebner

Phenomenon, through which the lesion expands or heals much more difficult.

- Insufficient hydration of the skin: Dry skin is vulnerable skin. Ignoring daily hydration leads to cracking of the skin and increased itching (itching).
- Use of irritating products: Using aggressive soaps, shower gels with synthetic perfumes, or harsh detergents that destroy the skin’s protective barrier.

3. Neglecting lifestyle factors

- Ignoring triggers: Continuing alcohol consumption, smoking and not managing stress cancel out the effects of any therapy, no matter how advanced it may be.
- Lack of education about the disease: Psoriasis is a complex condition. An uninformed patient will always look for quick “magic solutions”, ignoring the fact that the disease is systemic and requires patience and discipline.

4. Social and psychological aspects

- Underestimation of the systemic component: Ignoring mild joint pain, which may be the initial signs of psoriatic arthritis that can lead to irreversible deformities.
- Social isolation: Lack of communication with other patients who have successfully managed their disease. Sharing experiences and moral support are essential for maintaining optimism.
- Revolt vs. Active acceptance: Ignoring lesions in the hope that they will go away on their own seriously affects the quality of life and mental state, often leading to depression and anxiety.

2026 guideline advice

Do not just try to “erase” the spots on the skin. Learn to listen to your body, give it the necessary nutrition, and avoid these mistakes that “feed” disease from within.

SECTION V: Psoriasis Onset

Chapter 19: Psoriasis onset – How do we recognize the first signs?

At first, psoriasis can be deceptive, as its manifestations vary considerably from one patient to another. Recognizing the onset form is crucial for choosing the correct therapeutic strategy from the first symptoms.

1. Common forms of manifestation at onset

- Plaque psoriasis (vulgar): This is the mode of onset for approximately 80–90% of cases. It manifests itself as red, raised patches covered by a layer of silvery-white scales. The first areas affected are usually the elbows, knees, scalp, or lumbar area. The skin can become so dry that it bleeds at the slightest movement.

- Guttate psoriasis (droplet): It appears suddenly, in the form of small, pink-reddish patches, spread over the trunk and limbs. This form is very common in children and adolescents, often triggered by a respiratory infection (e.g. strep throat).
- Inverse psoriasis: It begins in the fold areas (armpits, groin, under the breasts). Unlike the vulgar form, the lesions are smooth, red, and shiny, without scales, and are often aggravated by humidity and sweating.
- Pustular psoriasis: A rarer and more alarming form of onset, characterized by the appearance of blisters with sterile (non-infectious) pus, usually located on the palms or soles, and may be accompanied by a general feeling of malaise.

2. Sensory symptoms and early signs

In addition to the visual appearance, the onset of psoriasis is accompanied by subjective signs that the patient feels directly:

- Cutaneous sensations: Intense itching (pruritus), burning sensation, or skin that “tightens”.
- Nail changes: Nails can become the first “victims”, presenting small indentations (like a sewing thimble), discolorations, or thickenings that can be mistaken for a mycosis.
- Discrete systemic signs: A state of unjustified fatigue or morning joint stiffness, which may indicate the onset of a rheumatic condition (psoriatic arthritis).

3. The importance of differential diagnosis

Since psoriasis can initially be confused with eczema, dermatitis, or fungal infections, it is imperative to consult a dermatologist. In 2026, the use of dermatoscopy and specific analyses allows the rapid differentiation of psoriasis from other pathologies, ensuring a correct start of treatment.

Conclusion

Regardless of the form in which it appears, psoriasis is a signal that the immune system is out of balance. Ignoring the first lesions only allows the disease to settle deeply in the body.

Chapter 20: Differential diagnosis – Diseases that mimic psoriasis

Psoriasis is often called the “great imitator” in dermatology because its symptoms—redness, scaling, and itching—overlap those of many other conditions. Correct identification of the disease is the essential first step to effective treatment.

1. Commonly confused conditions (Routine diagnosis)

- Eczema (Atopic or Nummular Dermatitis): Although both cause intense pruritus (itching), psoriasis lesions are more well-defined and have silvery, dry scales. Eczema tends to have “wetter” lesions, with fine blisters and yellowish crusts.

- Seborrheic dermatitis: Confusion most often occurs on the scalp. Key difference: In seborrheic dermatitis, the scales are yellowish, oily, and soft, while in psoriasis, they are white, dry, thick, and adherent.
- Cutaneous ringworm (Tinea): Fungal infections (fungi) can create round, red, scaly patches. Unlike psoriasis, ringworm often has a more active (redder) edge and a center that tends to heal, being contagious.
- Lichen planus: It manifests itself as shiny, purplish bumps (papules), often on the joints. It can also affect the nails, mimicking nail psoriasis with longitudinal deformations and streaks.

2. Systemic and infectious diseases

- Cutaneous Lupus Erythematosus: Lesions appear mainly on sun-exposed areas (face, décolleté). Although they can be scaly, they often leave scars or pigmentation changes, unlike psoriasis.
- Secondary Syphilis: The historical “great imitator” can produce rashes on the palms and soles that are strikingly similar to palmoplantar psoriasis. A blood test is necessary for exclusion in atypical cases.

3. Rare diseases and major risks

- Pityriasis Rubra Pilar: A rare disease that begins with red-orange plaques and severe thickening of the palms. It is often confused with erythrodermic psoriasis.
- Skin cancers (e.g., Bowen’s disease): Certain forms of carcinoma may appear as a single red, scaly patch that does not go away with moisturizers. If a “psoriasis lesion” is unique and does not respond to treatment, a biopsy becomes mandatory.
- Acute Exanthematous Pustulosis (AGEP): It must be quickly differentiated from pustular psoriasis, as it is often a severe reaction to drugs (antibiotics) and requires a different emergency protocol.

Conclusion of the 2026 guideline

Never rely on self-diagnosis using images from the Internet.

A dermatologist uses advanced techniques, such as dermatoscopy or skin biopsy, to confirm the diagnosis. Using a cortisone treatment on a fungal infection mistaken for psoriasis can severely worsen the situation.

SECTION VI: Epigenetics and Nutrition

Chapter 21: Epigenetics – How to “Turn Off” disease genes

In the year 2026, medicine is no longer limited to the study of fixed DNA. Epigenetics is the branch that studies changes that affect gene expression — that is, how they are turned on

or off — without changing the actual DNA sequence. If DNA is the “keyboard” of our biological computer, epigenetics is the “software” that decides which keys are pressed.

1. How do epigenetic “Switches” work?

Our body uses chemical mechanisms (such as DNA methylation) to decide whether a gene is:

- “On” (Expressed): The gene produces the proteins that can trigger the inflammation in psoriasis.
- “Off” (Silent): The gene remains inactive, and the disease goes into remission.

The great news is that these switches are directly influenced by the external environment. Diet, stress, sleep quality, smoking, and exposure to toxins leave epigenetic “marks” that can activate or block the disease.

2. Why is epigenetics vital for psoriasis sufferers?

- Beyond Heredity: Explains why, of two identical twins with the same genetic predisposition for psoriasis, one can develop a severe form, while the other remains healthy. The difference lies in the lifestyle and environment in which they lived (exposure to triggers).
- Reversibility: Unlike permanent genetic mutations, epigenetic marks are, for the most part, reversible. By changing nutrition and reducing inflammation, we can “reprogram” cells to no longer produce the excess skin specific to psoriasis.
- Biological Age vs. Chronological Age: In 2026, epigenetic tests will allow us to measure the true age of our cells. Good control of psoriasis through natural methods and a balanced lifestyle can “rejuvenate” the patient’s epigenetic profile.

3. External influences that remodel genes

- Nutrition: Certain foods and nutraceuticals (such as those in medicinal plants) act as epigenetic modulators, helping to “turn off” pro-inflammatory genes.
- Chronic stress: This sends chemical signals that “turn on” genes responsible for autoimmune attack.
- Microbiome: The balance of gut bacteria is a major epigenetic factor. A healthy flora produces metabolites that keep inflammatory genes in check.

4. Transgenerational impact

Current research suggests that the way we take care of our health today can leave a positive epigenetic legacy for our children. Proper management of psoriasis is not only a personal victory but also a protection for future generations.

Conclusion of the 2026 guide

Epigenetics gives us back control. We are no longer “victims” of our own genetics. Although we have the psoriasis

genes in our DNA code, through integrated treatment, specific nutrition, and stress management, we have the power to keep the “switch” of the disease in the OFF position.

Chapter 22: The CARD14 gene – The “Engine” of inflammation in psoriasis

In the complex universe of genetics, one particular gene has captured the attention of researchers in recent years:

The CARD14 gene (Caspase Recruitment Domain-containing protein 14). In 2026, we know that this gene is responsible not only for the onset but also for the long-term maintenance of severe forms of psoriasis.

1. What is the CARD14 Gene, and how does it work?

CARD14 is a gene that provides instructions for the production of a protein found mainly in skin cells (keratinocytes). This protein has the role of activating a signaling pathway called NF- κ B, which functions as a “control center” of inflammation in the body.

- Normal function: The protein activates inflammation only when the skin needs to defend itself from an aggressor (a cut, an infection).
- Mutation (Defect): In people with psoriasis, mutations in this gene cause the CARD14 protein to be permanently “on”. This leads to an overactivation of the NF- κ B pathway, keeping the skin in a state of alert and continuous inflammation, for no real reason.

2. Link to severe forms of the disease

Although CARD14 influences psoriasis vulgaris (plaque), it is directly responsible for the most difficult-to-treat forms:

- Generalized Pustular Psoriasis (GPP): CARD14 mutations are the main driver of this severe form, characterized by the sudden appearance of pustules and systemic inflammation.
- Palmar-Plantar Psoriasis: This gene plays a crucial role in locations on the palms and soles, where the skin is thicker and the inflammation more persistent.

3. Interaction between genetics and environment

Having a CARD14 gene mutation does not automatically mean that you will be sick for life.

In the context of epigenetics (discussed in the previous chapter), genetics only represents the “loading of the gun”, while environmental factors are the ones that “pull the trigger”.

- Specific triggers: Infections (especially respiratory ones), intense psychological stress, and certain medications act on the CARD14 gene, amplifying the exaggerated inflammatory response.

4. The importance of knowledge in 2026

The identification of the role of CARD14 has allowed modern

medicine to develop more precise therapies. Understanding that the problem starts from a hyperactive signaling pathway (NF- κ B), new treatments (including immunomodulatory nutraceuticals) try to “reduce the volume” of this inflammatory signal, helping the skin to return to a normal regeneration rate.

Conclusion

The CARD14 gene explains why some patients have much more aggressive forms of psoriasis or are resistant to classic treatments. This genetic predisposition amplifies inflammation, but by managing external factors and using targeted solutions, we can calm this genetic “engine” and achieve stable remission.

Chapter 23: HLA antigens and the role of the HLA-Cw6 marker

In the study of the genetics of psoriasis, the HLA (Human Leukocyte Antigen) complex represents one of the most important pillars. Of all its variants, the HLA-Cw6 marker is the most strongly associated with the risk of developing the disease, providing valuable clues about how psoriasis will evolve in a given patient.

1. What are HLA antigens?

These are proteins located on the surface of most cells in the body, functioning as a biological “identity card”. Their main role is to help the immune system distinguish between:

- The body’s own: Healthy cells that need to be protected.
- Foreign elements: Viruses, bacteria, or tumor cells that need to be attacked.

In psoriasis, the presence of the HLA-Cw6 variant induces an error in this recognition process, causing the immune system to attack skin cells (keratinocytes).

2. Type I psoriasis and HLA-Cw6

The presence of this marker usually defines what we call Type I psoriasis, characterized by:

- Early onset: Usually appears before the age of 40 (often in adolescence or around the age of 20).
- Strong hereditary component: Patients almost always have affected first-degree relatives.
- Increased severity: This form tends to be more extensive, frequently affecting the trunk and limbs.
- Specific manifestations: It is strongly associated with guttate psoriasis (drop-shaped) and with a high sensitivity to trauma (Koebner phenomenon).

3. Clinical importance and prognosis in 2026

Identifying the HLA-Cw6 marker is not just a laboratory exercise, but has major practical utility:

- Severity predictor: People positive for HLA-Cw6 have a higher risk of developing persistent forms and presenting an early onset of psoriatic arthritis.
- Treatment personalization: Current studies confirm that HLA-Cw6 positive patients respond much better to certain biological therapies (such as IL-12/23 inhibitors – Ustekinumab) compared to negative patients.
- Monitoring accuracy: Although not all patients with psoriasis have this marker, its presence helps doctors anticipate the evolution of the disease and intervene more aggressively from the beginning to prevent complications.

Conclusion

The HLA-Cw6 antigen is the genetic “compass” of type I psoriasis. Knowledge of this genetic profile allows the transition from general treatment to precision medicine, in which therapy is chosen according to the biological fingerprint of each patient.

SECTION VII: Heredity; Genetic Predisposition; Stress Management

Chapter 24: Heredity and genetic predisposition in psoriasis

Heredity is one of the most important factors in the development of psoriasis, but it is essential to understand that we do not inherit skin lesions, but a genetic vulnerability. In 2026, studies confirmed that psoriasis is a polygenic (involving several genes) and multifactorial disease.

1. The role of heredity in numbers

Genetics determines the probability of developing the disease throughout life:

- No family history: The risk is approximately 2-4%.
- One affected parent: The child’s risk increases to approximately 14-28%.
- Both affected parents: The risk increases significantly, reaching up to 41-65%.

2. What does «Predisposition» actually mean?

Having psoriasis genes (such as the HLA-Cw6 marker) does not guarantee that you will develop the disease. Predisposition is a “fertile ground” or genetic basis on which triggers can be grafted.

- Immune vulnerability: Inherited genes dictate how the immune system reacts to stress, infection, or trauma. In a predisposed person, the immune system is “set” to attack skin cells (keratinocytes) much more easily than in a person without this genetic baggage.
- Specific mutations: Genes affect the skin’s protective

barrier and the way T cells communicate with each other, leading to chronic inflammation.

3. The interaction of heredity and environment

In modern medicine of 2026, the concept of predisposition is inextricably linked to external factors that “activate” genes:

- **Activating Factors:** Intense stress, streptococcal infections (pharyngitis), obesity, smoking, and certain medications act as an on/off switch for inherited genes.
- **Koebner Phenomenon:** People with a hereditary predisposition are much more likely to develop new lesions where the skin has suffered a cut or burn.

4. A positive perspective

The fact that there is a hereditary predisposition does not mean a lack of control. By managing environmental factors — proper nutrition, controlling the microbiome, and reducing stress — we can keep these genes “dormant” (a state of remission), preventing their clinical manifestation even when the genetic ground is present.

Conclusion

Heredity is the foundation, but lifestyle is the architect of our health. Understanding family risk allows us to be more vigilant and take early preventive measures, thus protecting future generations.

Chapter 25: Stress management – Breaking the vicious cycle

In 2026, psycho-dermatology studies confirmed that stress is one of the strongest triggers of psoriasis. The relationship between emotional state and skin is circular: psychological stress causes rashes, and the visibility of the disease generates, in turn, anxiety and social stress, creating a vicious circle that is difficult to break without an appropriate strategy.

1. The biological relationship between stress and psoriasis

When we are stressed, the body releases hormones such as cortisol and adrenaline, which directly activate the immune system. In patients with psoriasis, this alarm signal causes T cells to attack the skin with increased intensity, leading to the expansion of lesions and the appearance of severe pruritus (itching).

2. Stress management techniques in 2026

To stabilize the disease, the patient must adopt emotional self-regulation mechanisms:

- **Psychotherapy and Counseling:** Cognitive-Behavioral Therapy (CBT) and support groups are essential to manage the emotional impact, depression, and social stigma associated with the disease.
- **Deep Relaxation Practices:** Mindfulness meditation,

yoga, and controlled breathing techniques (diaphragmatic breathing) help reduce inflammation by calming the central nervous system.

- **Restorative Sleep:** Lack of sleep (insomnia) disrupts cell regeneration and increases the level of pro-inflammatory cytokines. A 7-8 hour sleep is a natural “treatment” for restoring the skin barrier.

3. Lifestyle as an anti-stress shield

A physically balanced body resists psychological pressure much better:

- **Elimination of Neurotoxins:** Smoking and alcohol are not only skin irritants, but also factors that increase the level of anxiety and oxidative stress in cells.
- **Weight Control:** Excess weight puts additional physical and inflammatory pressure on the body, worsening systemic stress.
- **Hydration and Gentle Care:** Keeping the skin hydrated reduces physical discomfort (burning or tightness), which decreases the patient’s level of irritability.

4. Integrated approach

The effectiveness of biological or natural treatments increases considerably when the patient learns to manage their emotions. In 2026, therapeutic success is defined not only by clean skin but also by a stable psychological balance.

Conclusion of the guide

Managing stress means regaining control over one’s life. By breaking the vicious cycle of “stress-illness-stress”, the patient gives the immune system the chance to enter a state of calm, promoting long-term remission of psoriasis.

SECTION VIII: Microbiome

Chapter 26: The role of the microbiome in food transformation

In 2026, we understand that digestion is not complete without the intervention of the gut microbiome. It is the complex ecosystem of microorganisms that transforms food debris that our human enzymes cannot process, transforming them into molecules vital for skin and immune system health.

1. Metabolic and digestive function

The microbiome acts as a high-precision biochemical laboratory:

- **Fiber fermentation:** Beneficial bacteria break down indigestible fiber and complex carbohydrates, producing Short Chain Fatty Acids (SCFAs), such as acetate, propionate, and most importantly, butyrate. The latter nourishes the cells of the colon and has a powerful systemic anti-inflammatory effect.

- **Nutrient decomposition:** Helps complete the digestion of proteins and complex carbohydrates, ensuring efficient use of food resources.
- **Vitamin synthesis:** The microbiome is an internal vitamin “factory.” Certain strains (such as beneficial *E. coli*) produce vitamin K, essential for clotting, and B vitamins (B12, folate, biotin), crucial for skin regeneration.

2. Barrier protection and immune modulation

For the psoriasis sufferer, the integrity of the intestinal wall is the main line of defense:

- **Barrier effect:** Good bacteria occupy intestinal receptors, competing with pathogens for nutrients and thus blocking colonization by harmful bacteria.
- **Immune education:** The microbiome “trains” the immune system to distinguish between harmless substances and threats. A balanced microbiome keeps the intestinal barrier tight, preventing toxins from passing into the bloodstream (leaky gut syndrome).

3. Influence on metabolism and energy

- **Caloric extraction:** The composition of the microbiome influences the efficiency with which we extract calories from food, having a direct impact on body weight.
- **Metabolic balance:** Bacterial imbalances (dysbiosis) are directly linked to insulin resistance, obesity, and metabolic syndrome — all of which are common comorbidities that worsen psoriasis.

4. How do we support a healthy microbiome in 2026?

Therapeutic success depends on dietary diversity:

- **Pro-microbiome foods:** A diet rich in fiber (prebiotics), fruits, colorful vegetables, whole grains, and naturally fermented foods (yogurt, kefir, sauerkraut).
- **Destructive factors:** Ultra-processed foods, refined sugars, excess red meat, and the misuse of antibiotics destroy bacterial diversity, triggering inflammation that subsequently manifests itself on the skin.

Guide conclusion

The microbiome is the bridge between what we eat and how our skin looks. A healthy microbiome turns food into anti-inflammatory “medicine,” while a degraded microbiome turns food into attack signals for the immune system.

Chapter 27: Food breakdown and the importance of digestion

The process by which food is converted into nutrients is a complex mechanism, both mechanical and chemical. For the patient with psoriasis, incomplete digestion can mean the entry of large, undigested molecules into the bloodstream, triggering immune reactions that maintain skin lesions.

1. Fundamental stages of digestion

- **Oral cavity (First barrier):**
 - **Chewing:** The teeth break down food, increasing the contact surface for enzymes.
 - **Saliva:** Contains salivary amylase, which immediately initiates the breakdown of complex carbohydrates (starch) into simple sugars.
- **Stomach (Chemical transformation):**

Food is mixed with gastric acid and specific enzymes, such as pepsin, which begin the cleavage of proteins into smaller fragments called peptides. The result is a semi-liquid paste (chyme) ready for the small intestine.

- **Small intestine (Absorption center):**
 - This is where the most important part of the process takes place. Pancreatic enzymes (lipase for fats, amylase for carbohydrates, and proteases for proteins) complete the breakdown into absorbable units: fatty acids, glucose, and amino acids.
 - **Bile:** Produced by the liver, it emulsifies fats, facilitating the action of lipase.
 - **Intestinal villi:** Through these, nutrients pass into the blood to be transported to the cells.
- **Large intestine (Colon and microbiome):**
 - Water is extracted, and undigested residues (especially fiber) are taken up by intestinal bacteria. These ferment the fibers, producing short-chain fatty acids (SCFA) and essential vitamins (such as vitamin K and biotin), essential for skin health.

2. Table of key nutrients and enzymes

Nutrient final product (absorbable) main enzymes

Carbohydrates: Glucose (energy), amylase

Amino acids (building): Pepsin, trypsin, proteases

Fats, fatty acids, and glycerol lipase (supported by bile)

3. Relevance to psoriasis in 2026

If any of these steps is deficient — whether due to insufficient mastication, lack of enzymes, or biliary dysfunction — food debris can cause putrefaction or excessive fermentation processes in the intestine. These toxic debris can cross the intestinal barrier (the “leaky gut” phenomenon), activating the immune system, which will respond with psoriasis-specific skin inflammation.

Guideline conclusion

Healthy digestion begins with proper mastication and ends with a balanced microbiome. Understanding this chain of transformations helps us see food not just as a culinary pleasure, but as the “raw material” for healthy skin.

Chapter 28: Foods that influence the microbiome

In the medicine of 2026, diet is no longer seen simply as a calorie intake, but as a “fuel” for our bacterial ecosystem. The foods we choose can be either allies that feed anti-inflammatory bacteria, or enemies that favor dysbiosis and, implicitly, the exacerbation of psoriasis.

1. Allies of the microbiome: Probiotics and prebiotics

To maintain healthy skin, we must provide the intestine with both live bacteria and the “food” necessary for their development.

- Fermented foods (Probiotics): These provide a direct intake of live bacterial cultures.
 - Examples: Natural yogurt (sugar-free), kefir, sauerkraut (prepared by natural fermentation, not in vinegar), kimchi, miso, tempeh, and kombucha.
- Plant fiber (Prebiotics): Represents the “food” of beneficial bacteria. Without them, probiotics cannot survive.
 - Vegetables: Garlic, onions, leeks, asparagus, broccoli, artichokes.
 - Fruits: Bananas (especially green ones), apples, berries.
 - Legumes and grains: Lentils, chickpeas, beans, whole oats.
 - Seeds and nuts: Source of fiber and healthy fats.
- Polyphenols: Antioxidant compounds that selectively stimulate good bacteria. Found in green tea, pure cocoa and dark berries.

2. Enemies of the microbiome: Dysbiosis factors

Certain foods and substances destroy microbial diversity and increase intestinal permeability:

- Ultraprocessed Foods: Sausages, chips, instant soups, pastries, and fast food contain additives and preservatives that “sterilize” beneficial intestinal flora.
- Sugar and Artificial Sweeteners: Refined sugar feeds fungi (such as *Candida*) and pathogenic bacteria, while artificial sweeteners (aspartame, saccharin) can profoundly alter the composition of the microbiome.
- Saturated and Trans Fats: Excessive consumption of fatty red meat and fried foods promotes a pro-inflammatory intestinal environment.
- Alcohol: Acts as a solvent on the intestinal barrier, allowing toxins to pass into the bloodstream and trigger rashes.

- Antibiotic Abuse: Indiscriminately destroys the entire intestinal ecosystem. Their use should be strictly medically justified and always accompanied by probiotic support.

3. Strategic recommendations in 2026

To stop the spread of psoriasis, adopt the following rules:

1. Diversity rule: Consume at least 30 different types of plants per week to ensure a varied intestinal flora.
2. Mediterranean pattern: Prioritize olive oil, fish, vegetables, and fresh fruits.
3. Regular transit: A sluggish gut allows toxins to be reabsorbed. Fiber and hydration are essential for daily waste elimination.
4. Simplicity: Choose foods in their most natural (unprocessed) form.

Guide conclusion

Your microbiome is a reflection of your plate. By choosing live, whole foods, you transform your gut into a protective shield that reduces inflammation and supports healthy skin regeneration.

Chapter 29: The microbiome – The immune modulator in psoriasis

In the medicine of 2026, the microbiome is no longer considered just an adjuvant to digestion, but a true “command center” of the immune system. For the psoriasis patient, the balance of this microbial population dictates the frequency and intensity of inflammatory flare-ups.

1. What is the microbiome, and why does it matter?

The microbiome represents the totality of microorganisms (bacteria, viruses, fungi) that cohabit with us. The densest and most influential ecosystem is in the large intestine.

- Biological fingerprint: Each person’s microbiome is unique and is constantly shaped by diet, stress, environment, and medications.
- Immune organ: Approximately 70–80% of the immune system cells are located in the intestine, being in permanent dialogue with the microbiome.

2. Immune modulation mechanisms

A healthy microbiome (eubiosis) keeps inflammation in check by:

- Tolerance training: Teaches the immune system not to attack its own cells (such as skin cells).
- Production of anti-inflammatory metabolites: Fiber breakdown generates short-chain fatty acids (such as butyrate), which have a direct “calming” effect on the exaggerated immune response.

- Competition for resources: Beneficial bacteria occupy space on the intestinal walls, preventing the growth of pathogens that could trigger inflammation.

3. The danger of dysbiosis and “Leaky Gut”

When the balance is broken (dysbiosis), the intestinal barrier deteriorates:

- Increased permeability: The intestinal wall becomes “porous,” allowing bacterial fragments, toxins, and undigested food particles to pass into the bloodstream.
- Systemic activation: Once in the blood, these substances are identified as “enemies”, triggering an aggressive immune reaction that, in genetically predisposed individuals, manifests itself through psoriasis plaques on the skin.

4. Management strategies in 2026

Controlling the microbiome is an essential link in treating the internal causes of psoriasis:

- Precision nutrition: A diet rich in prebiotics (food for bacteria) and diversified plant fibers.
- Probiotic support: The use of specific bacterial strains capable of restoring the intestinal barrier and reducing systemic inflammation.
- Advanced monitoring: Testing the intestinal microbiome has become a standard procedure to identify specific imbalances and personalize nutritional intervention.

Guideline conclusion

For psoriasis sufferers, skin healing passes through the gut. Maintaining a balanced microbial flora is the most effective method of “educating” the immune system to stop attacking the skin and return to a state of tolerance and calm.

Chapter 30: Microbiome dysbiosis in psoriasis patients

In 2026, research confirms that psoriasis is not just a genetic error, but also a consequence of dysbiosis — a profound imbalance in the ecosystem of microorganisms in our bodies. This state of bacterial disharmony fuels inflammation and dictates the severity of skin symptoms.

1. What is Dysbiosis in the context of psoriasis?

Dysbiosis is the disruption of the balance between beneficial (protective) and potentially pathogenic (aggressor) bacteria. In patients with psoriasis, this phenomenon manifests itself through:

- Decreased diversity: A smaller number of different bacterial species, which weakens the resilience of the immune system.
- Loss of anti-inflammatory bacteria: Reduction in

populations of bacteria that produce protective metabolites (such as butyrate).

- Proliferation of pathogens: Increase in bacteria that secrete toxins and pro-inflammatory molecules.

2. Mechanism of aggravation: From gut to skin

Dysbiosis does not remain localized in the gut; it has a systemic impact through the following processes:

- Leaky gut syndrome: Bacterial imbalance damages the intestinal “barrier”. This porosity allows bacterial fragments and endotoxins to pass into the blood.
- Exacerbation of inflammation: Once in circulation, these molecules activate the immune system remotely, triggering or worsening psoriasis plaques on the skin.
- Correlation with severity: Studies from 2026 demonstrate that the more pronounced the dysbiosis, the higher the Psoriasis Severity score (PASI).

3. Dual dysbiosis: Intestinal and cutaneous

The psoriasis patient faces an imbalance on two fronts:

- Gut microbiome: The internal engine of autoimmune inflammation.
- Cutaneous microbiome: On skin affected by psoriasis, the microbial balance is altered. The lack of diversity on the skin surface makes lesions more vulnerable to superinfections and more resistant to topical treatments (ointments).

4. Therapeutic implications in 2026

Understanding dysbiosis changes the way we treat the disease:

- Beyond genetics: Although we cannot change genes, we can correct dysbiosis.
- Dynamic approach: Treatment should aim to restore bacterial diversity through specific nutrition, state-of-the-art probiotics and the avoidance of factors that destroy the flora (stress, unnecessary antibiotics, processed foods).

Guideline conclusion

Dysbiosis is not just a secondary symptom, but an active factor that maintains psoriasis. Correcting this imbalance through natural methods and diet is the key to “calming” the immune system and achieving clear skin in the long term.

Chapter 31: Metabolites and gut barrier integrity

In 2026, we know that it’s not just the bacteria themselves that matter, but also the “waste” and substances they produce, called metabolites. These chemical compounds function as

messengers that can either repair or destroy the gut barrier, directly influencing the severity of psoriasis.

1. What are microbiome metabolites?

Metabolites are bioactive molecules (acids, vitamins, organic compounds) resulting from the processing of food by microorganisms.

- Beneficial metabolites (SCFA): Such as butyrate, which nourish intestinal cells and “seal” the intestinal wall.
- Toxic metabolites: Result from protein putrefaction or bacterial imbalances, which attack the junctions between intestinal cells.

2. The “Leaky Gut” mechanism

In patients with psoriasis, dysbiosis leads to a decreased production of good metabolites and an increase in harmful ones. This imbalance affects the sealing proteins (such as claudin-3 and zonulin), creating “gaps” in the intestinal wall.

- Infiltration into the blood: Through these gaps, aggressive bacterial components (such as LPS - lipopolysaccharides) and toxic metabolites enter the bloodstream.
- Systemic activation: Once in the circulation, these factors trigger T cells and the inflammatory cascade that feeds the psoriasis plaques on the skin.

3. “Enemy” metabolites and their effects

2026 research identifies three main groups of harmful metabolites in psoriasis:

- Phenol and p-Cresol: Result from the incomplete breakdown of amino acids. Their high levels in the blood are directly correlated with a weakened skin barrier and poor hydration.
- Indole derivatives (I3S): They activate specific receptors (AHR) that promote the Th17 immune response, the main driver of psoriatic inflammation.
- TMAO: A metabolite often associated with excessive consumption of red meat, which damages the intestinal barrier and increases cardiovascular risk in psoriasis patients.

4. The importance of bile acids

In psoriasis, an alteration of secondary bile acids is often observed. They are no longer able to properly emulsify fats and maintain immune balance, contributing to a vicious cycle of inflammation.

5. Therapeutic implications in 2026

Recovering the intestinal barrier is a priority to stop the “bombardment” of toxic metabolites:

- Barrier repair: Use of nutraceuticals, prebiotics, and the Mediterranean diet to increase the production of SCFA (beneficial fatty acids).
- Toxin neutralization: Consuming antioxidants such as resveratrol and specific medicinal herbs that help restore microbial balance.
- Restoring bile flow: Supporting liver function for proper metabolism of fats and metabolites.

Guideline conclusion

Psoriasis does not just heal on the surface. By reducing the production of toxic metabolites and strengthening the intestinal barrier, we cut off the “power source” of systemic inflammation, allowing the skin to naturally heal.

SECTION IX: Observational Clinical Study

Chapter 32: Therapeutic effects of deniplant nutraceuticals on the microbiome

Background: A growing body of evidence suggests that gut dysbiosis is associated with the development of psoriasis. The gut-skin axis is the novel concept of the interaction between skin diseases and the microbiome through inflammatory mediators, metabolites, and the intestinal barrier. The gut microbiome affects skin homeostasis through its influence on signaling pathways that coordinate epidermal differentiation.

The objective of this study was to synthesize current data on the natural modulator Deniplant of the gut microbiome in patients with psoriasis.

Materials and methods: All studies have confirmed the association of psoriasis with gut microbiota dysbiosis. We describe recent advances in the interaction between the gut microbiota and the skin. Thus, the microbiome can be considered an effective therapeutic target for treating this condition.

Results: This presentation provides a detailed and comprehensive systematic review of the gut microbiome in psoriasis patients. These results are supported by clinical observations based on a series of cases showing improvement of psoriatic skin lesions after the natural modulator Deniplant. It is still unclear whether psoriasis is an effect or a cause of the observed imbalance between beneficial and pathogenic microbes. In this context, the study provides very interesting results, showing significantly greater changes in the gut microbiome of psoriasis patients treated with the natural modulator Deniplant.

Conclusion: There is a significant association between changes in gut microbial composition and psoriasis. Gut dysbiosis is a state of imbalance of the gut microbiome that ultimately has a negative impact on skin function and integrity. The natural modulator Deniplant is a potential therapeutic strategy in psoriasis patients.

Keywords: Dysbiosis; Microbiome; Psoriasis; Gut-skin axis; Intestinal barrier; Deniplant nutraceuticals

Psoriasis is a common inflammatory skin condition, affecting approximately 3% of the world's population. Psoriasis is a chronic inflammatory skin disease associated with hyperplasia of epidermal keratinocytes and overactivation of epidermal immune cells. The majority of psoriasis cases involve chronic plaque psoriasis (referred to as psoriasis vulgaris). The etiology of psoriasis is multifactorial and involves the interaction of genetic and environmental factors that exacerbate innate and adaptive immune responses. The compositions of the local skin and gut microbiome are linked to the modulation of inflammation and disease severity in psoriasis.

Evidence for dysbiosis as a source of disease pathology is well documented in inflammatory skin conditions such as psoriasis [1].

In recent years, a growing number of observational studies have revealed an association between the composition of the gut microbiota and patients with psoriasis. The microbiome is closely linked to the development of psoriasis, serving as both a potential cause and a consequence of the psoriatic process.

Alterations in the gut microbiota can alter intestinal permeability, leading to the translocation of bacterial metabolites into the bloodstream, subsequently modulating the host immune response.

A growing body of literature suggests a crucial role for the gut microbiome in modulating systemic inflammatory disease [2,3].

Psoriasis is a chronic systemic inflammatory disease, and its pathogenesis is related to the interaction between genetic susceptibility, immune response, and environmental triggers such as diet, stress level, skin care routine, etc., [4].

Nutrition plays an important role in the development of psoriasis and can modulate the microbiota and microbiome composition. Correct dietary choices may have a crucial role in the pathogenesis of psoriasis. Lifestyle and dietary habits may be related to the incidence and severity of psoriasis [5,6].

In recent years, there has been an increased interest among psoriasis researchers in exploring how psoriasis treatments affect the skin and gut microbiome.

The treatment of patients with psoriasis requires a multidisciplinary approach not only to alleviate skin symptoms, but also to manage the metabolic, nutritional, and socio-psychological comorbidities that are often associated with this disease [7].

The gut-skin axis is a novel concept of the interaction between skin diseases and the microbiome through inflammatory mediators, metabolites, and the intestinal barrier.

Dysregulated skin microbiota may become a novel

therapeutic target in patients with psoriasis. Psoriasis is a common skin disease with chronic inflammation and a complex etiology [8].

The association between the gut and skin is strong and bidirectional, and gastrointestinal health is associated with skin homeostasis. Increasing evidence suggests the existence of the gut-skin axis and that an imbalanced gut microbiome can induce inflammatory skin diseases [9].

The gut microbiome may mediate the interaction between the immune system and the nervous system through the secretion of neurotransmitters in psoriasis. The concept of the "skin-gut axis" provides a new perspective for investigating the association between the gut microbiota and the skin. This provides a feasible approach to improve skin conditions by modulating the gut microbiota [10]. Several types of neurotransmitters secreted by gut microbes have been selected to investigate their potential function in psoriasis. Microbiome-mediated interventions could be designed to manipulate these targets for the treatment of psoriasis. In addition, studies have also found that an important link between emotional states and inflammatory skin conditions may be regulated by bacteria in the gastrointestinal tract [11].

Through an extensive review of the literature, we aim to discuss the skin and gut microbiota and redefine their role in the pathogenesis of psoriasis [12, 13].

In recent years, there have been reports of an increasing incidence and prevalence of psoriasis. Subclinical gut inflammation and intestinal barrier dysfunction reported in psoriasis patients have given rise to the concept of the gut-skin axis.

A growing body of evidence indicates that the gut microbiota plays a critical role in regulating metabolism, the immune system, and intestinal permeability [14].

With the Deniplant brand, Gheorghe Giurgiu has developed several nutraceuticals for psoriasis that act as immunomodulators of the human microbiome. Therefore, it is crucial to understand the impact of nutraceuticals on the psoriatic skin microbiota, which is believed to be disrupted. Our study provides insights into the skin microbiota in psoriasis and how it is modulated by nutraceuticals and diet.

All studies have confirmed the association between psoriasis and gut microbiota dysbiosis. Deniplant tea prevents and treats the internal causes that trigger and maintain psoriasis by naturally modulating the gut and skin microbiome.

Eliminating dysbiosis from the gut microbiota can prevent and eliminate complications caused by psoriasis. It contains cultivated medicinal plants, berries, and flora, and fruit tree buds.

Distortion of the biodiversity and composition of the gut microbiota, known as gut dysbiosis, has been linked to metabolic syndrome, inflammatory arthritis, depression, cardiovascular disease, and inflammatory bowel disease, all of which are comorbidities of psoriasis.

Given the existence of the brain-gut-skin axis, it is now clear that gut microbes have significant effects on psoriasis. These results are supported by clinical observations based on a series of cases showing improvement of psoriatic skin lesions after antibiotic treatment, modulation of the gut microbiota by probiotics, or fecal microbial transplantation. We confirmed the association between psoriasis and gut microbiota dysbiosis.

This study provides a detailed and comprehensive systematic review of the gut microbiota in patients with psoriasis. In patients with psoriasis, the composition of the gut microbiota varies with disease severity and stages. It is still unclear whether...

Psoriasis is an effect or a cause of the observed imbalance between beneficial and pathogenic microbes.

Much less is known about the potential relationship between gut microbiota composition and psoriasis severity. Preclinical investigations provide evidence for the role of the gut microbiota in the pathogenesis of psoriasis. These results are supported by clinical observations based on a series of cases showing improvement of psoriatic skin lesions after modulation of the intestinal microbiota by Deniplant nutraceuticals. Food choices can affect the composition of the microbiome and improve the severity of psoriatic disease.

Since Deniplant treatment addresses the internal causes that trigger and maintain the disease, without ointments or other medications, its duration depends on how quickly the body resolves the dysbiosis of the intestinal microbiome and can be between 4 and 6 months. If the disease is older, the treatment can exceed 12 months. After healing of all lesions, there were patients who never had psoriasis again, but there were also patients whose disease recurred after 10–15 years. The microbiome is closely linked to the development of psoriasis, serving both as a potential cause and as a consequence of the psoriatic process.

Conclusion

Psoriasis is a common and chronic dermatological disease, considered a systemic inflammatory disorder. There is a significant association between alterations in the gut microbial composition and psoriasis. Unfortunately, the direct link between the skin microbiota and the pathogenesis of psoriasis remains to be clearly established. The treatment of psoriasis, similar to other complex immune-mediated diseases, is limited to symptom relief due to the lack of an effective therapy. Based on these findings, the treatment of skin inflammation with nutraceuticals is favored, as its therapeutic management is simple, safe, and inexpensive.

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SECTION X: The Role of Nutrition in Psoriasis

Chapter 33: Nutraceuticals – Bridging nutrition and health

In 2026, the term nutraceutical (derived from “nutrition” and “pharmaceutical”) defines those foods or food components that provide medical benefits, including the prevention and treatment of diseases. In psoriasis, nutraceuticals are not simple supplements, but biological tools that act on the internal causes of inflammation.

1. Mechanisms of action in psoriasis

Nutraceuticals intervene in the inflammatory cascade through four fundamental pathways:

- **Anti-inflammatory action:** Compounds such as Omega-3 (EPA/DHA), curcumin (from turmeric), and resveratrol directly block inflammatory pathways and reduce the production of pro-inflammatory cytokines.
- **Immune modulation:** Vitamin D regulates T-cell behavior, while probiotics “train” the immune system by balancing the gut microbiome.
- **Antioxidant protection:** Selenium, zinc and vitamins A, C, E neutralize oxidative stress, protecting keratinocytes (skin cells) from degradation.
- **Skin barrier restoration:** Essential fatty acids support the structural integrity of the skin, reducing water loss and severe dryness.

2. Key nutraceuticals in the 2026 edition

1. **Omega-3 fatty acids:** Essential for reducing redness and scaling. Found in deep-sea fish oil or flaxseed oil.
2. **Vitamin D:** The central pillar of skin health; it acts as a hormone that dictates proper cell regeneration.
3. **Curcumin (Turmeric):** One of the most studied natural anti-inflammatory agents, effective in reducing underlying inflammation.
4. **Zinc and Selenium:** Critical minerals for the skin's defense system and protein synthesis.
5. **New generation probiotics:** Specific strains that restore the microbiome and “seal” the leaky gut.
6. **Vitamin A (Beta-carotene):** Crucial for cell differentiation, preventing the formation of thick layers of dead skin.

3. Integrated therapeutic role

In certain situations and under specialized supervision, nutraceuticals can complement or even reduce the need for synthetic drugs (corticosteroids, methotrexate). They aim to control underlying inflammation, helping to maintain periods of remission and improve quality of life by addressing the metabolic aspects of the disease.

4. Important recommendations

- **Personalized Approach:** Not all nutraceuticals are necessary for every patient. The choice of doses and combinations should be made depending on the severity of the disease and biochemical analyses.
- **Specialty Consultation:** Before starting administration, consult a doctor or dietitian to avoid interactions with current medication and to ensure that you are using products with high bioavailability.

Guide conclusion

Nutraceuticals represent the medicine of the future in psoriasis management.

By transforming nutrition into a therapeutic ally, the patient moves from a passive to an active state, providing the body with the necessary resources to heal itself from the inside out.

Chapter 34: Food and nutrition – Psoriasis nutrition therapy

In 2026, psoriasis nutrition is based on the anti-inflammatory principle. Since psoriasis is a systemic disease, the foods we eat function either as a “fuel” for inflammation or as a natural medicine that helps regenerate the skin barrier.

1. Anti-inflammatory allies (what to eat daily)

The goal is to provide nutrients that calm the immune system and hydrate the skin from the inside:

- **Omega-3 fatty acids:** They are essential for reducing redness and flakes. Consume fatty fish (wild salmon, sardines, mackerel), flax seeds, chia seeds, and Romanian walnuts.
- **Plant antioxidants:** Berries, green leafy vegetables, and fresh herbs (parsley, thyme, basil) neutralize oxidative stress in skin cells.
- **Monounsaturated fats:** Extra virgin olive oil and avocado support skin elasticity and the absorption of fat-soluble vitamins (A, D, E).
- **Natural probiotics:** Artisanal yogurt, kefir, kombucha, and sauerkraut (unpasteurized) are vital for maintaining the balance of the intestinal microbiome, the engine of our immunity.

2. Inflammation triggers (what to avoid)

Eliminating these foods can lead to a visible improvement in lesions in a few weeks:

- **Refined sugar and sweets:** They are the main food for the “bad” bacteria in the intestine and accelerate inflammatory processes.
- **Ultraprocessed foods:** Sausages, pastries, chips, and soups in sachets contain additives that damage the intestinal wall.
- **Red meat and fatty dairy:** These contain arachidonic acid and saturated fats that can exacerbate inflammation in sensitive people.
- **Alcohol:** It is a major pro-inflammatory that dehydrates the skin and can negate the effects of drug or natural treatments.

3. Nutritional goals in 2026

- **Weight management:** Fat cells produce inflammatory cytokines. Maintaining an optimal weight automatically reduces the body's inflammatory “burden”.
- **Intestinal sealing:** A diet rich in fiber (30-35g/day)

helps repair the intestinal barrier, preventing toxins from passing into the blood.

- Biological hydration: Consumption of purified water and herbal infusions supports the elimination of toxins through the kidneys, relieving the skin of this burden.

4. Practical recommendation

Adopt a Mediterranean-style dietary pattern, adapted to individual needs. This diet has been scientifically proven to be the most effective in reducing Psoriasis Severity (PASI score) and preventing cardiovascular comorbidities.

Guide conclusion

Proper nutrition is the foundation on which any healing is built. By consciously choosing foods, the psoriasis patient stops “feeding” the disease and begins to rebuild their health cell by cell.

Chapter 35: Proteins and enzymes – The invisible imbalances in psoriasis

In patients with psoriasis, the dynamics of proteins and enzymes do not necessarily reflect a “deficiency” in the classical sense, but rather a metabolic and immune dysfunction. In 2026, we know that these biochemical imbalances support systemic inflammation and influence how the body reacts to food and stressors.

1. Proteins and enzymes with a critical role in psoriasis

- C-Reactive Protein (CRP): It is the most reliable marker of systemic inflammation. High levels of CRP in the blood indicate that the “fire” of inflammation is burning throughout the body, even if the plaques on the skin appear under control. A high CRP value is an alarm signal for associated cardiovascular risks.
- DAO (Diamine Oxidase) enzyme: This enzyme is responsible for breaking down histamine in food. A deficiency in DAO (often associated with gut dysbiosis) leads to the accumulation of histamine, which can mimic or exacerbate the itching and redness characteristic of psoriasis.
- Metabolic and Liver Enzymes: Since psoriasis is frequently linked to metabolic syndrome and hepatic steatosis (fatty liver), enzymes responsible for detoxification may be overworked or deficient. This affects the body’s ability to process nutrients and eliminate toxic waste.
- Signaling Proteins (Cytokines): In psoriasis, there is an excess of pro-inflammatory proteins (such as TNF-alpha or Interleukins) and a functional deficiency of proteins that should “turn off” the immune response.

2. What do these imbalances mean for the patient?

- “Silent” Inflammation: Even though the skin

clears, enzyme imbalances can maintain residual inflammation. This requires an approach that goes beyond dermatological treatment, targeting nutrition and metabolic health.

- Gut-Liver-Skin Axis: Deficiency of digestive enzymes or protein transporters leads to poor nutrient absorption, which deprives the skin of the elements necessary for healthy regeneration.

3. Holistic management in 2026

To balance these proteins and enzymes, modern treatment includes:

1. Marker monitoring: Periodic analyses for PCR, liver function, and, in specific cases, histamine/DAO levels.
2. Weight management: Reducing adipose tissue decreases the production of inflammatory cytokines and improves overall enzyme activity.
3. Natural enzyme support: The use of nutraceuticals and medicinal herbs that support digestion and liver function, helping to reduce the inflammatory “load”.
4. Specific diet: Avoiding foods rich in histamine and those that stress the liver (alcohol, trans fats, sugar).

Guideline conclusion

Balancing proteins and enzymes is the “fine-tuning” necessary for stable remission. In 2026, therapeutic success means not only a skin without lesions, but also an optimal biochemical profile, where inflammation markers are brought within normal limits through nutrition, lifestyle, and integrated treatment.

Chapter 36: The role of vitamins and minerals in psoriasis

In 2026, psoriasis management recognizes vitamins and minerals not just as supplements, but as biochemical modulators of skin health. Although oral supplementation does not replace basic therapy, correcting nutritional deficiencies is a mandatory step to reduce inflammation and support cell regeneration.

1. Key vitamins in psoriasis management

- Vitamin D (“Sunshine Vitamin”):
 - Role: It is the most important modulator of the immune system and keratinocyte maturation. In 2026, Vitamin D is standardly used in topical treatments (creams) to slow accelerated cell multiplication.
 - Oral supplementation: Although debated in the past, current medical consensus emphasizes that maintaining optimal levels of Vitamin D in the blood

is crucial. Patients with deficiencies often present with more aggressive and treatment-resistant forms of psoriasis.

- Vitamin E:
 - Role: A powerful antioxidant that protects cell membranes from oxidative stress.
 - Benefits: Helps relieve severe skin dryness, reduces itching, and supports the healing of bleeding areas.
- Vitamin A (and Retinoids):
 - Role: Essential for proper cell differentiation. Helps normalize the keratinization process (scale formation), being a pillar in the treatment of pustular forms.

2. Essential minerals and micronutrients

- Zinc and Selenium: These minerals are fundamental for immune function and for the synthesis of structural proteins of the skin. Zinc deficiency is often associated with slow healing of lesions.
- Vitamin B Complex (B12, Folic Acid): Vitamin B12 and folic acid are involved in the metabolism of homocysteine; optimal levels help reduce cardiovascular risk, a major comorbidity in psoriasis.

3. Strategic recommendations for 2026

For a real impact on the disease, the approach must be personalized:

1. Preliminary blood tests: Do not supplement “by ear”. In 2026, it is recommended to test serum levels of Vitamin D (25-OH), Zinc, and B12 before starting any treatment.
2. Nutritional synergy: Vitamins and minerals work better when obtained from an anti-inflammatory diet (fatty fish for Omega-3, nuts, seeds, green vegetables).
3. Quality of supplements: Choose forms of nutraceuticals with high bioavailability, which are efficiently absorbed at the intestinal level (taking into account the possible dysbiosis discussed in previous chapters).

Conclusion of the guide

Vitamins and minerals represent the “raw material” for skin repair. By correcting deficiencies, we give the body the tools it needs to decrease the intensity of inflammation and move from scaly skin to elastic and healthy skin.

Chapter 37: Medicinal plants in the treatment of psoriasis

In 2026, modern phytotherapy recognizes the role of medicinal plants not only as traditional remedies but as complex sources of bioactive compounds that can modulate inflammation and skin regeneration. Their correct use, both internally and externally, supports the healing process without damaging the natural skin barrier.

1. Plants for soothing and regeneration (external use)

These plants are essential for reducing the physical and aesthetic discomfort of plaques:

- Marigold (*Calendula officinalis*): One of the most powerful cell regeneration agents. It soothes irritation, reduces the risk of secondary fungal infections, and accelerates the closure of bleeding cracks.
- Chamomile (*Matricaria chamomilla*): Rich in bisabolol, it has a profound anti-inflammatory action, being ideal for reducing redness and soothing pruritus (itching).
- Aloe Vera: Its pure gel provides intense hydration and essential vitamins, being a natural barrier against excessive drying of the affected skin.
- Yarrow (*Achillea millefolium*): Provides antibacterial and anti-inflammatory protection, preventing superinfection of open lesions.

2. Plants with systemic action (internal and external use)

These target the root causes of the disease and detoxify the body:

- Viola tricolor: Considered the “pearl” of natural dermatology, it is used to regulate the allergic and inflammatory response in chronic conditions such as psoriasis and eczema.
- Echinacea (*Echinacea purpurea*): Acts as an immunomodulator, helping the body manage infections that could trigger new psoriasis flare-ups.
- Celandine (*Chelidonium majus*): Supports liver and skin detoxification, being useful in forms of psoriasis resistant to classic treatments.

3. Herbs for liver detoxification and purification

Since psoriasis is a systemic disease, blood cleansing and liver support are priorities in 2026:

- Nettle (*Urtica dioica*): Has remarkable purifying properties, helping to eliminate metabolic toxins from the blood.
- Dandelion (*Taraxacum officinale*): Stimulates bile flow and liver function, essential for proper fat metabolism and reducing inflammation.
- Horsetail (*Equisetum arvense*): Rich in silicon, it contributes to the remineralization of the body and strengthens the collagen structure of the skin.

4. Methods of use in psoriasis management

- Infusions and decoctions: Used as teas (nettle, dandelion) or for local soothing compresses (chamomile, marigold).
- Tinctures and extracts: Concentrated forms (echinacea,

three-brothers-spotted) that can be precisely dosed for an immunomodulatory effect.

- Poultices: Local application of macerated plants to soften thick scales and soothe deep inflammation.

Recommendation of the 2026 guide

To achieve maximum results, medicinal plants should be integrated into a complete protocol that also includes a healthy lifestyle. The use of quality extracts from verified sources ensures the presence of the active principles necessary to “extinguish” inflammation from the inside out.

Chapter 38: Gemmotherapy – The embryonic power of plants

Gemmotherapy is a modern therapeutic method that uses extracts obtained from plant tissues in the growth phase: buds, shoots, or roots (gemoderivates). In the context of 2026, gemmotherapy is considered a valuable adjuvant in psoriasis, because it acts deeply on the body’s toxin elimination filters.

1. How does Gemmotherapy help in Psoriasis?

Unlike classical phytotherapy, gemoderivates contain the genetic information and vital active principles of the budding plant, offering specific benefits:

- Deep detoxification (drainage): Stimulates the elimination organs (liver, kidneys, intestine) to clean metabolic waste and toxins that would otherwise be eliminated through the skin in the form of psoriatic plaques.
- Immune regulation: Helps modulate the exaggerated immune response, reducing the aggressiveness of the defensive system on its own skin cells.
- Regeneration and healing: Supports the restoration of damaged tissues and improves skin elasticity.

2. Essential gemoderivates for psoriasis

- Walnut (*Juglans regia*): It is considered the “king” of gemotherapy for the skin. It acts as a powerful antiseptic and anti-inflammatory, being especially indicated for superinfected psoriasis or that with an intestinal component (dysbiosis).
- Birch (*Betula verrucosa/pubescens*): An excellent universal drainer. It promotes the elimination of uric acid and toxins, cleansing the body “from the inside out”.
- Grapevine (*Vitis vinifera*): Used for its ability to stop chronic inflammatory processes and support skin regeneration at the microvascular level.
- Black Currant (*Ribes nigrum*): (Added for its effectiveness in 2026) – Also called “natural cortisone”, this gemoderivative reduces acute inflammation without the side effects of synthetic drugs.

3. Integration into the treatment plan

Gemotherapy does not replace the basic treatment, but it enhances its effects:

- Personalized scheme: Success depends on a correct scheme (for example, Walnut in the morning and Birch in the evening). The duration of the treatments is usually 2–3 months.
- Quality of extracts: In 2026, it is recommended to use standardized products, obtained through rigorous pharmaceutical methods, to ensure the presence of all active principles.
- Specialty consultation: The recommendation of a dermatologist or phytotherapist is mandatory to avoid interactions and adapt the doses according to the severity of the disease.

Conclusion of the guide

Gemotherapy offers the patient with psoriasis a gentle but extremely effective method of “internal cleansing”. By draining toxins and regulating immunity at the embryonic level, gemoderivatives help to reduce outbreaks of disease activity and maintain healthy skin in the long term.

Chapter 39: Berries – Natural antioxidants for the skin

In the integrative medicine of 2026, berries are recognized as dietary pillars in the management of psoriasis. Their richness in antioxidants, vitamins (C, A, E), and organic acids (salicylic, ellagic) transform them into powerful allies for cellular protection and the reduction of systemic inflammation.

1. Key berries and their specific benefits

- Blueberries: They represent one of the most potent sources of antioxidants in nature. They prevent oxidative damage and premature aging of tissues, protecting the skin from metabolic stress.
- Aronia: In 2026, aronia is highly appreciated for its huge concentration of polyphenols, which help regulate the immune response and reduce chronic inflammation.
- Raspberries and Blackberries: Contain ellagic acid, a compound with proven anti-inflammatory properties, which protects skin cells from free radicals and stimulates collagen synthesis.
- Strawberries: Provide a massive intake of vitamin C and natural salicylic acid (a gentle B.H.A.), which helps cleanse the skin and reduce scales through gentle exfoliation.
- Hawthorn: Although frequently used for the heart, in dermatology, it improves skin microcirculation, ensuring a better supply of oxygen and nutrients to damaged areas.

2. How do they help in the treatment of psoriasis?

Berries act on several levels simultaneously:

- **Combating oxidative stress:** Free radicals aggravate psoriatic inflammation. The antioxidants in these fruits neutralize these molecules, “calming” immune reactions in the skin.
- **Anti-inflammatory effect:** Reduce redness and edema (swelling) associated with active plaques.
- **Cleansing and exfoliation:** Natural acids (salicylic, malic) help to thin the layer of scales, making the skin smoother.
- **Support in psoriatic arthritis:** Along with spinach, broccoli, and kale, berries are part of the mandatory diet for patients with joint damage, helping to reduce pain by decreasing systemic inflammation.

3. Consumption and usage methods

- **Diet (Oral consumption):** It is the most effective method. Daily consumption of fresh or frozen berries ensures a constant antioxidant barrier.
- **Nutraceuticals:** Concentrated blueberry or aronia extracts are used in 2026 as supplements to enhance the anti-inflammatory effect.
- **External use:** Berries extracts are integrated into serums and natural creams to protect the skin against environmental factors and to stimulate cell renewal.

Guide conclusion

Introducing berries into your daily routine is not just a healthy culinary choice but a therapeutic strategy. They “feed” the skin with the antioxidants necessary to block degenerative processes and support visible and lasting remission.

SECTION XI: Psoriasis Management Tips

Chapter 40: Other helpful tips for daily management

Successful management of psoriasis requires a proactive approach that combines medical treatment with a rigorous personal care routine. These practical tips, validated by 2026 clinical experience, help keep skin hydrated, reduce inflammation, and extend remission periods.

1. Proper skin care (external routine)

An intact skin barrier is the first line of defense against the spread of the disease:

- **Intense hydration:** Use emollients and thick, fragrance-free moisturizers daily to prevent skin dryness and reduce flaking. Apply the cream immediately after showering, while the skin is still damp.

- **Therapeutic baths:** Warm (not hot!) baths with natural oils (olive, coconut) or colloidal oatmeal can soothe severe irritation and itching. Limit bath time to 10–15 minutes.
- **Avoiding harsh products:** Perfumed soaps, alcohol-based lotions, or harsh detergents can strip the skin’s protective layer and trigger new lesions. Opt for gentle, hypoallergenic products.
- **Sun protection:** Moderate sun exposure (heliotherapy) can be beneficial, but avoid sunburn at all costs, as it is a major trigger of psoriasis (Koebner phenomenon). Always use adequate sunscreen on healthy skin.

2. Nutritional discipline (internal routine)

Diet remains a crucial pillar in controlling systemic inflammation:

- **Anti-Inflammatory diet:** Prioritize foods rich in antioxidants, Omega-3 fatty acids (fatty fish, seeds), and plant fibers. These reduce oxidative stress and help balance the microbiome.
- **Elimination of inflammatory factors:** Reduce or eliminate ultra-processed foods, refined sugar, red meat, and alcohol. These aggravate symptoms by overactivating the immune system.
- **Optimal body weight:** Maintaining a healthy weight significantly reduces the severity of psoriasis and the risk of comorbidities (diabetes, heart disease).

3. Other lifestyle recommendations

- **Stress management:** Integrate relaxation techniques (meditation, yoga) into your daily routine to break the stress-psoriasis vicious cycle.
- **Quality sleep:** A restful sleep (7–8 hours per night) is essential for cellular regeneration and hormonal and immune balancing.

Conclusion

Psoriasis is a disease that requires discipline and a holistic approach. Adopting these simple but essential tips transforms disease management from a constant battle into a sustainable lifestyle, allowing the patient to live a full life with clear and healthy skin.

Chapter 41: The importance of sleep in skin repair

In 2026, skin chronobiology confirms that sleep is an indispensable pillar in the management of psoriasis. Night is not just a period of rest, but the moment when the body activates its most complex mechanisms of cell regeneration and balances the immune system.

1. What happens to the skin at night?

During deep sleep, the skin undergoes an essential metabolic transformation:

- **Accelerated regeneration:** Skin cells divide and repair themselves up to three times faster than during the day. This process is stimulated by the nocturnal secretion of growth hormone, essential for the repair of areas affected by psoriasis.
- **Collagen synthesis:** The body accelerates the production of collagen, ensuring the elasticity and firmness of the skin, elements that help prevent psoriatic plaques from cracking.
- **Circulation optimization:** Blood flow to the dermis increases, ensuring the delivery of oxygen and nutrients (vitamins, minerals, fatty acids) directly to the skin layers that need repair.
- **Anti-inflammatory effect:** Quality sleep reduces cortisol levels (the stress hormone). Low cortisol levels at night allow the immune system to “calm down”, reducing redness and itching.

2. Consequences of lack of sleep for psoriasis sufferers

Sleep deprivation acts as a stress trigger, worsening the symptoms of the disease:

- **Decreased skin barrier:** Lack of rest leads to greater trans epidermal water loss, resulting in drier, rougher, and more prone to flaking skin.
- **Inflammation boosting:** Chronic fatigue keeps the body on high alert, increasing levels of pro-inflammatory cytokines that feed psoriasis lesions.
- **Slowed healing:** Without 7–9 hours of rest, regeneration processes are disrupted, causing plaques to persist on the skin longer.

3. Strategies for restorative sleep in 2026

- **Respecting circadian rhythm:** Try to go to bed and wake up at the same times. Regularity helps regulate the secretion of melatonin, a powerful antioxidant.
- **Sleep environment:** Maintain a cool temperature in the bedroom and use bedding made of natural materials (cotton, silk) that do not irritate sensitive skin.
- **Nighttime routine:** Apply emollient creams or topical nutraceuticals just before bed to take advantage of the increased permeability of the skin during the night.
- **Dark cure:** Avoid blue light from screens at least an hour before bed to avoid blocking natural melatonin production.

Guide conclusion

Sleep is the skin’s “silent medicine.” For a psoriasis patient, every hour of quality sleep is a step toward brighter, more elastic, and less inflamed skin. Never underestimate the regenerative power of a good night’s rest!

Chapter 42: EGO – Your personal assistant for nutrition, health, and sports

In the era of digital medicine in 2026, the success of treatment depends on constant monitoring and data accuracy.

The EGO platform was created as a “Digital Alterego” — an extension of your own memory, designed to record and process all the habits that influence your health and your microbiome.

1. What is EGO?

EGO is more than an application; it is an intelligent assistant that monitors the essential pillars of your life: nutrition, sports, hygiene, as well as your physical and emotional states.

- **Registration and access:** To benefit from EGO’s functions, it is necessary to register and log in.

2. Key benefits and features

- **Precision monitoring:** Records eating habits and medication administration, preventing dosage errors or omissions.
- **Rapid diagnosis:** Facilitates the work of doctors by providing accurate data logs, shortening the time needed to make a correct diagnosis.
- **Microbiome balancing:** Helps prevent dysbiosis and manage autoimmune diseases (such as psoriasis), metabolic or neurological disorders by monitoring reactions to different foods.
- **Integrated telemedicine:** Facilitates online consultations with specialists from the Biomedicine Center. Doctors analyze your data in real time and offer you personalized advice after each evaluation.
- **Prevention and education:** EGO provides active health education, including helping to prevent obesity in children (recommended for ages over 7).
- **Interface with the electronic file:** The platform functions as a bridge between the patient and the official electronic file from the National Health Center (CNS).

3. EGO philosophy: Food as medicine

The EGO Platform is based on Hippocrates’ exhortation: “Let food be your medicine and medicine be your food.” In our vision for 2026, this motto has become:

“Eat and heal, if you know what, how much and how.”

4. A partner for life

EGO is not just software, it is “The Assistant created by Deniplant specialists to be with you 24/7”.

EGO addresses all age groups — from children to the elderly. Once you have accessed this platform, it is essential to feed it with correct data and take care of it. As one of our users

said, “EGO takes care of your health more than you do”. It will be the assistant who will monitor your well-being and guide you when you need medical and nutritional support the most.

Conclusion

In a world where information is power, EGO gives you the power to take control of your own health. It is the missing link that transforms general advice into personalized therapy, ensuring your path to a life free of psoriasis and other chronic conditions.

Final conclusion: A new paradigm in psoriasis treatment (2026 edition)

Psoriasis is no longer viewed today as a simple dermatological condition, but as a complex systemic inflammatory disorder. The scientific evidence accumulated until 2026 confirms the existence of a profound association between changes in the intestinal microbiome and cutaneous manifestations. This connection, defined by the “gut-skin axis”, indicates that epithelial health directly depends on the internal balance of our microorganisms.

Although research continues to explore the intimate mechanisms by which the skin microbiota influences the pathogenesis of the disease, one thing is certain: classical therapeutic approaches have been limited for too long to the improvement of external symptoms. In the absence of a “magic pill” that can definitively cure the disease, modern complementary medicine is moving towards integrated and safe solutions.

Why choose nutraceuticals in 2026?

Based on the findings presented in this guide, the treatment of systemic inflammation with nutraceuticals (such as the solutions in the Deniplant range) represents a future alternative because:

1. **Targets the cause, not just the effect:** They act as immunomodulators on the microbiome and intestinal barrier.
2. **Safety:** Provides therapeutic management devoid of the toxicity of synthetic systemic treatments.
3. **Accessibility:** Represents a simple, safe, and economically sustainable method for the long-term management of a chronic disease.

We conclude this guide with the conviction that an informed patient, who understands the role of nutrition, genetics, and microbial balance, has the power to radically transform their quality of life.

Psoriasis can be kept under control, and remission can become a lasting state when we treat the body as a whole.

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