

Fast Facts



Fast Facts: Psoriasis

**Alan Menter, Catherine Smith and
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Third edition



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Declaration of Independence

This book is as balanced and as practical as we can make it.
Ideas for improvement are always welcome: feedback@fastfacts.com



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Glossary of abbreviations

APC: antigen-presenting cell

COX inhibitor: cyclooxygenase inhibitor

CRP: C-reactive protein

ESR: erythrocyte sedimentation rate

HIV: human immunodeficiency virus

HLA: human leukocyte antigen

ICAM1: intercellular adhesion molecule 1

Ig: immunoglobulin

IL: interleukin

LFA1: lymphocyte function-associated antigen 1

MED: minimal erythema dose; amount of radiation required to produce faint, but definite, erythema

NSAID: non-steroidal anti-inflammatory drug

PASI: Psoriasis Area and Severity Index

PSORS1: psoriasis susceptibility locus

PUVA: photochemotherapy; combination of methoxsalen (psoralen), a photosensitizing drug, and long-wave UVA radiation

Th cell: T helper cell

TPMT: thiopurine methyltransferase

TNF α : tumor necrosis factor- α

Introduction

Psoriasis is a common, chronic, and potentially disfiguring, skin disease. It has recently received a lot of scientific and medical attention, in great part because of major advances in our understanding of the disease and the consequent development of new treatments that are dramatically effective in many patients. Key areas of science in which advances have been made are genetics and immunology, and new findings are highlighted in this edition of the book.

Increasingly, it is being recognized that psoriasis is more than skin deep. That is to say, psoriasis is a systemic disease that may result in other associated medical conditions. Cardiovascular disease is one such condition that is now more readily recognized as a comorbidity of psoriasis, as are the consequences that this may have for therapeutic intervention. Cardiovascular disease, along with other comorbid conditions, is discussed in Chapter 2; in addition, Chapter 9 on psoriatic arthritis, written by a guest author prominent in the field of rheumatology, has been expanded and updated to cover the classification, differential diagnosis and management of this often underdiagnosed and undertreated condition.

There are many different clinical forms of psoriasis. Since publication of the last edition of this book, attempts have been made to subdivide chronic plaque psoriasis into different clinical variants and the clinical presentation of these variants is addressed in Chapter 2.

Since the second edition, there has been an explosion of interest in systemic therapy for psoriasis. Guidelines on optimizing conventional therapies have now been issued, and some older drugs are receiving wider attention and usage, particularly in Europe. Fumaric acid esters are one such example and are discussed in Chapter 7. However, the main area of therapeutic advance has been the receptor-targeted (biological) therapies. The chapter on these therapies has been expanded and updated to give an outline of the very latest developments in this important area. The advent of receptor-targeted treatments has had a dramatic beneficial effect on many patients with moderate and severe disease. These treatments also have a key role in managing individuals

with comorbidities such as arthritis and inflammatory bowel disease, and, looking forward, may have a role in switching off the systemic inflammation associated with the disease that may in turn cause other comorbidities.

This third edition of *Fast Facts: Psoriasis* is a well-illustrated, easy-to-read yet comprehensive synopsis of the state of the art in psoriasis, its pathogenesis and its management. We believe it is a valuable resource for all healthcare professionals involved in the care and treatment of individuals with this problematic but increasingly treatable disease.

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Epidemiology

Psoriasis is a common chronic, disfiguring inflammatory skin disease that affects up to 3% of the population. Several clinical variants exist (see Chapter 2) of which chronic plaque psoriasis (psoriasis vulgaris) is the most common form (85–90% of all cases).

Effects of ethnicity. Although all races are affected, there is considerable interracial variation. For example, psoriasis is relatively common in white people but appears to be very uncommon in native American Indians and in Japanese people. Prevalence appears to be highest in Scandinavian countries and northern Europe.

Effects of gender and age. Men and women are affected equally. The usual age of onset is 20–35 years, with 75% of all cases occurring for the first time before 40 years of age. However, psoriasis can occur at any age, including childhood (often signifying a more severe clinical course) and old age.

Types of psoriasis. Two types of chronic plaque psoriasis have been described, based on age of onset, association with human leukocyte antigen (HLA) and disease course.

- Type I, the commonest form, occurs in young adults with a high probability of a positive family history. Affected individuals tend to have more severe disease that runs a more irregular course.
- Type II has a peak incidence between 50 and 60 years of age. In these individuals, a positive family history is very uncommon and the disease tends to be mild and localized.

Socioeconomic burden. Psoriasis severely affects a patient's quality of life in terms of both psychological and physical well-being. Studies comparing psoriasis with other important chronic diseases have shown that the impact of psoriasis on the patient's quality of life is at least as

great as that of ischemic heart disease, diabetes and chronic obstructive pulmonary disease. Psoriasis is therefore a disease of major socioeconomic importance; in the USA alone, the annual cost to society has been estimated at US\$3 billion. Furthermore, there is increasing evidence of an association between psoriasis and important comorbidities including cardiovascular and psychiatric disease; evidence suggests that patients with moderate and severe disease have a high mortality due to cardiovascular events (see Chapter 2).

In the UK, although most patients with psoriasis have relatively mild disease, current evidence suggests that 30% require second-line treatment (phototherapy or systemic medication) that involves referral to a dermatologist and that perhaps 40% of all dermatology inpatients have psoriasis. In the USA, where inpatient dermatology care is rarely available, and at many institutions in the UK, specialized psoriasis day centers have evolved for patients with moderate-to-severe disease.

Pathogenesis and pathophysiology

The pathogenesis of psoriasis can be described in terms of three inter-related phases (Figure 1.1):

- the interplay between environmental and genetic factors
- the interaction between innate/acquired immunity and key inflammatory and epidermal cells
- changes in the epidermis and dermis, such as growth and blood vessel formation, that lead to the development of the clinical psoriatic plaque.

Genetics. In 1963, Gunnar Lomholt, a pioneer in the epidemiology of psoriasis, stated in his classic thesis that the disease ‘is capricious and refuses to part with its innermost secret’, but also wrote ‘that psoriasis is genetically conditioned is beyond doubt’. The validity of this statement has been borne out by population and family studies and research in twins, all of which suggest an important genetic component to the disease. For example, in terms of types I and II psoriasis (see above), approximately 80% of patients with type I psoriasis are positive for the *HLA-Cw6* gene, compared with only 20% of those

3 Differential diagnosis

Although diagnosis is relatively straightforward, a number of other dermatological entities may be confused with psoriasis (Table 3.1). A careful medical and family history and physical examination, together with laboratory findings, will usually reveal the correct diagnosis.

Infection

Candidiasis. In flexural areas, peripheral pustules are characteristic of *Candida* infection. The presence of yeast and pseudohyphae in Gram-stained microscopy specimens will confirm infection.

Tinea, or ringworm, is an infection caused by a dermatophyte fungus and may sometimes be mistaken for psoriasis. Where diagnostic doubt exists, appropriate mycological specimens (skin, hair and/or nail) should be taken.

Tinea capitis is ringworm of the head. Although a minor degree of hair thinning is not uncommon in established cases of psoriasis, well-

TABLE 3.1

Differential diagnosis of psoriasis

- Candidiasis
- Tinea infection
- Syphilis (secondary)
- Eczema (atopic or nummular)
- Contact dermatitis
- Drug-induced rashes
- Lichen planus
- Superficial basal cell carcinoma and Bowen's disease
- Cutaneous T-cell lymphoma (mycosis fungoides)
- Pityriasis rosea



Figure 3.1 A scalp infected with *Trichophyton tonsurans*, which is a common cause of tinea capitis. Clinical signs vary from mild scaling to, as here, marked alopecia and suppurative inflammation (kerion formation).

demarcated areas of hair loss (Figure 3.1) and signs such as the ‘black dots’ or fractured hair shafts characteristic of tinea infection are highly unusual in psoriasis. In addition, tinea capitis seldom shows the classic silvery scale of psoriasis.

Tinea corporis affects the body (Figure 3.2). Lack of symmetrical lesions, presence of peripheral scale and central clearing are all more characteristic of tinea than of psoriasis.



Figure 3.2 A patch of tinea corporis due to *Microsporum canis*. Note the typical peripheral scale and relative central clearing.