

Acne scarring

Last reviewed: August 2023

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What is acne scarring?

Scarring is a widely recognised sequelae of [acne](#), an inflammatory dermatological disorder that frequently affects younger adults and can persist for years. Acne favours the face and upper back but can also develop in other sites with well-developed sebaceous glands.

Scarring refers to the fibrous process in which new collagen is laid down to heal a full-thickness injury, such as cutaneous inflammation in acne.



Ice pick scarring on the cheeks



Mild atrophic acne scarring on the back – there is also some perifollicular elastolysis



Rolling scars over the cheek



Post inflammatory pigmentation after facial acne in skin of colour



True keloids on the shoulders post acne in skin of colour



Severe atrophic scarring from back nodulocystic acne

[View more images](#)

Who gets acne scarring?

Epidemiological data on the prevalence and incidence of acne scarring varies. The duration and severity of acne often correlates to the degree of resultant scarring.

Scarring is more common in patients with moderate to severe [acne vulgaris](#), or in acne subtypes such as [nodulocystic acne](#), [acne conglobata](#), and [acne fulminans](#). Scarring can also be exacerbated in [acne excorie](#) due to picking or squeezing of primary lesions.

Some patients may have a predilection to scarring compared to others. For example, research suggests that smoking increases the risk of more severe acne scarring.

What causes acne scarring?

The formation of scars is a normal reaction of the skin in response to inflammation or injury. Further injury such as 'picking' at active acne lesions can also increase the likelihood of developing acne scars.

The majority of acne scars result from an overall inflammatory response causing net destruction of collagen fibres in the dermis in an atrophic scar. Underlying fibrosis can also occur. The consequence of a net gain of aberrant production of collagen in turn causes hypertrophic or keloid-type acne scars.

What are the clinical features of acne scarring?

The majority of acne scars are atrophic. Atrophic scars can be classified by three main types: ice-pick scars, rolling scars, and box-car scars.

Ice-pick scars: the most common type of atrophic acne scars (60–70%). These are narrow, deeper than they are wide, and V-shaped with a sharp edge extending downwards into the deep dermis or subcutaneous tissue.

Boxcar scars: the next most common type of atrophic scars; wider in size, round or oval shaped depressions with distinct edges.

Rolling scars: wide, with a sloping edge that can be smoothed out if stretched.

Perifollicular elastolysis: 2–4 mm hypo pigmented atrophic scars centred around hair follicles; most common on the back and chest

A secondary [anetoderma](#) can sometimes form after acne. This refers to a depression in the skin due to the elastic tissue within the dermis that is lost.

Less commonly, acne scarring can result in a [keloid or hypertrophic scar](#). A hypertrophic scar is the same size as the acne lesion that caused it, while a keloid scar is an excessive scar formation often larger than the causative lesion. They occur more frequently along the jawline, chest, and upper back.

'Hybrid scars' exhibit multiple physical characteristics, such as pigmentation or erythema, alongside being atrophic or hypertrophic.

Other associated skin changes

Postinflammatory skin colour changes, a common side effect of acne lesions, may arise prior to or in conjunction with acne scar formation. These can include erythema, hyperpigmentation, and hypopigmentation.

Postinflammatory erythema (more common in lighter skin) is thought to relate to microvascular changes and epidermal thinning of the skin during wound healing.

[Postinflammatory hyperpigmentation](#) (more common in darker skin types) occurs due to the deposition of melanin within the keratinocytes of skin that had recent inflammatory damage.

Colour changes associated with acne often improve with time, however this may take months or longer.

[View images of acne scarring](#)

How do clinical features vary in differing types of skin?

[Postinflammatory hyperpigmentation](#) and keloid scarring is more common in skin of colour following acne lesions. Keloid scars can continue to evolve over time.

What are the complications of acne scarring?

Significant [psychological effects](#).

Potential side-effects from attempted treatments eg, postinflammatory erythema or [hyperpigmentation](#), [infection](#), or further scarring.

Recurrence of [hypertrophic](#) or [keloid scars](#).

How is acne scarring diagnosed?

Acne scarring is diagnosed clinically based on a history of acne and scar appearances. If there is diagnostic uncertainty (eg, an evolving hypertrophic or keloid scar), a [biopsy](#) may be considered.

What is the differential diagnosis for acne scarring?

Other causes of scarring.

Skin lesions can sometimes mimic [keloid](#) and [hypertrophic scars](#), such as some skin tumours, [cutaneous squamous cell carcinoma](#), [cutaneous pseudolymphoma](#), [lobomycosis](#), and [morphoea](#).

What is the treatment for acne scarring?

[Makeup](#) may be helpful for disguising acne scars, particularly those on the face.

Treatments may also improve the appearance of acne scarring. Active acne should be [treated](#) prior to commencing scar management.

An individualised approach to treatment is required based on the size, depth, nature, and location of acne scarring; the patient's baseline skin type; and their concerns, goals, and budget. More than one type of treatment can be used as part of a multi-modal approach to correct scar colour, texture, and volume.

Resurfacing treatments

Resurfacing procedures aim to blend acne scars into the surrounding skin by removing epidermis to smooth out scar edges, stimulating growth of new cells, and promoting skin remodelling. These may be best used for more superficial acne scars. Multiple treatments may be required for best results.

[Chemical peels](#) – superficial, medium, or deep peels with various chemicals.

[TCA CROSS](#) (trichloroacetic acid chemical reconstruction of skin scars) – a specific chemical peel technique involving the application of high strength (65–100%) TCA.

[Dermabrasion](#) – physical resurfacing of the skin with a use of tools.

[Laser resurfacing](#) – ablative and non-ablative fractionated lasers.

[Skin needling](#) – involves thousands of micro-punctures to the level of the mid-dermis to achieve uniform pinpoint bleeding of the skin and promote scar tissue modulation and remodelling. This is also known as 'collagen induction therapy' or 'needle dermabrasion'.

Lifting procedures

Lifting procedures are used to improve the appearance of atrophic scars with volumetric filling of underlying soft tissue loss.

[Dermal fillers](#) – volumising; injected directly under depressed atrophic scars (eg, [hyaluronic acid implant](#), [collagen replacement therapy](#), [fat grafting](#)).

[Subcision](#) – a surgical technique in which the fibrous scar band under the targeted scar is divided, allowing the skin to return to its normal position.

Excision

[Excisional techniques](#) allow for complete scar removal and might be useful for prominent, deep, fibrotic, or hypopigmented scars. These include:

Punch or elliptical excision

Punch grafting.

Treatment of hypertrophic or keloid acne scarring

Hypertrophic or keloid scars are particularly prone to recur even after apparently successful treatment. Options may include:

Potent [topical steroids](#) applied under occlusion to the scar for several weeks

[Intralesional targeted steroid](#) or [bleomycin](#) injections into the body of the scar

[Silicone gel dressings](#) or sheeting applied for 24 hours a day for some months

Skin needling

[Pulsed dye laser \(PDL\)](#)

[Cryotherapy](#)

Surgical revision/excision.

How do you prevent acne scarring?

More inflamed acne lesions are more likely to scar. Timely appropriate [treatment for acne](#) during the active phase can reduce the incidence and severity of acne scarring and its associated [psychosocial impacts](#).

What is the outlook for acne scarring?

Acne scars are usually permanent, although may improve spontaneously over time or with treatment.

[View images of acne scarring](#)

Acne

Author: Dr Amanda Oakley, Dermatologist, Hamilton, New Zealand, July 2014. Minor amendment by Ian Coulson, Dermatologist. July 2024.

What is acne?

Acne is a common chronic disorder affecting the hair follicle and sebaceous gland, in which there is expansion and blockage of the follicle and inflammation. There are several variants.

Who gets acne?

Acne affects males and females of all races and ethnicities. It is prevalent in adolescents and young adults, with 85% of 16 to 18 year-olds affected. However, it may sometimes occur in children and adults of all ages.

What causes acne?

Acne is due to a combination of factors. The exact mechanisms are not fully understood.

- Familial tendency
- Endogenous and exogenous [androgenic hormones](#)
- Acne [bacteria](#)
- Innate immune activation with inflammatory mediators
- Distension and occlusion of the hair follicles

Flares of acne can be provoked by:

- [Polycystic ovarian disease](#)
- Drugs: [steroids](#), hormones, anticonvulsants, [epidermal growth factor receptor inhibitors](#) and others
- Application of occlusive cosmetics
- High environmental humidity
- Diet high in dairy products and high glycaemic foods.

What are the clinical features of acne?

Acne is often confined to the face but it may involve the neck, chest, and back.

It is characterised by:

- Open and closed uninflamed [comedones](#) (blackheads and whiteheads)
- [Inflamed papules and pustules](#)
- In severe acne, [nodules and pseudocysts](#)
- Post-inflammatory erythematous or pigmented macules and [scars](#)
- Adverse social and [psychological effects](#).

Severity is classified as mild, moderate, or severe.

Mild acne: total lesion count <30

Moderate acne: total lesion count 30–125

Severe acne: total lesion count >125

What tests are necessary for acne?

In most cases, tests are unnecessary. If features are atypical consider:

Skin swabs for microscopy and culture

[Hormonal tests](#) in females.



Comedonal acne



Acne vulgaris



Nodulocystic acne

See more images of acne:

[Acne affecting the back images](#)

[Facial acne images](#)

[Steroid acne images](#).

What is the treatment for acne?

Mild acne

Topical anti-acne agents, such as [benzoyl peroxide](#), [azelaic acid](#), and [tretinoin](#) or [adapalene](#) gel and some antibiotics (clindamycin). New bioactive proteins may also prove successful.

Newer topical agents such as clascoterone

Low-dose combined oral contraceptive

Antiseptic or keratolytic washes containing [salicylic acid](#)

Moderate acne

As for mild acne plus a [tetracycline](#) such as doxycycline 50–200 mg daily for 6 months or so

[Erythromycin](#) or trimethoprim if doxycycline intolerant

[Antiandrogen therapy](#) with long-term cyproterone acetate + ethinylestradiol or spironolactone may be considered in women not responding to low-dose combined oral contraceptive, particularly for women with [polycystic ovaries](#)

[Isotretinoin](#) is often used if acne is persistent or treatment-resistant

Intralesional steroid injections can be useful for acute larger acne lesions

Severe acne

Referral to a dermatologist

If fever, arthralgia, bone pain, ulcerated or extensive skin lesions, blood count should be arranged and referral is urgent

Oral [antibiotics](#) are often used in higher doses than normal

Oral [isotretinoin](#) is usually recommended in suitable patients

What is the outlook for acne?

Acne tends to improve after the age of 25 years but may persist, especially in females.

Treatment with [isotretinoin](#) can lead to long-term remission in many patients.

Albinism

Author: Vanessa Ngan, Staff Writer, 2002. Updated by Dr Harriet Cheng, June 2014.

What is albinism?

Albinism is a condition in which people have little or no melanin pigment (compound that creates colour) in their eyes, skin or hair. Because of this people with albinism look a little different from other members of their family without albinism. They have very fair skin, which is prone to sunburn, their hair is white or a very light colour, and they may squint a lot as their eyes are sensitive to sunlight.

Classification of albinism

There are two main categories of albinism: oculocutaneous and ocular albinism.

Oculocutaneous

Involves dilution of the colour of the hair, skin and eyes

Most common form of albinism

Ocular

Melanin pigment mainly missing from the eyes while the skin and hair appear normal or only slightly lighter

Accounts for 10–15% of all albinism cases

Oculocutaneous albinism (OCA) make up a group of different types of albinism based on the specific albinism gene involved. Oculocutaneous albinism type 1 and type 2 are the most common types of oculocutaneous albinism.

OCA 1A

Absent tyrosinase activity

Eyes: blue grey, reduced visual acuity

Hair: white at birth, may become yellow over time

Skin: white, moles are non-pigmented

OCA 1B

Reduced tyrosinase activity

Variable dilution of skin and hair pigment

Temperature-sensitive subtype

OCA 2/OCA 2 mutation (previously P gene)

Variable dilution of skin and hair pigment

Over time, develop solar lentigines on sun-exposed skin

Prader-Willi and [Angelman](#)-associated (chromosome 15)

OCA 3TYRP 1 mutation

Reduced eumelanin synthesis
Rufous/red and brown subtypes

OCA 4SLC45A2 mutation (previously MAPT)

Similar to OCA 2
Most common type in Japan, China and India

Prenatal testing for OCA is available in some centres.

Other less common types of albinism include:

[Hermansky-Pudlak syndrome](#)
[Cross syndrome](#)
[Chediak-Higashi syndrome](#)
[Griscelli syndrome](#).

How do you get albinism?

Albinism is mostly a recessively inherited disease, which means two albinism genes are inherited (one from each parent). If the patient's parents are only carriers of albinism (each having one albinism gene and one normal gene), they will have enough genetic information to make normal pigment and will not show any signs of albinism.

Who is at risk of albinism?

Albinism occurs worldwide and affects people of all races. Males and females alike can have the condition although ocular albinism occurs primarily in males.

About 1 in 70 people have a gene for albinism. Couples whom are each carriers of the recessive albinism gene have a 1 in 4 chance of producing a child with albinism.

What are the problems associated with albinism?

The main problems of albinism are caused by the inability of the body to produce melanin pigment (whose major role in the skin is to absorb UV light from the sun so skin is not sun-damaged). It also has a role in the development of normal vision of the eye. Having white or light coloured hair due to lack of melanin is no cause for concern, however, lack of melanin in the skin and eyes can cause the following problems:

Skin problems

Easily sunburned
Increased chance of getting [skin cancer](#), mainly [cutaneous squamous cell carcinoma](#) and [basal cell carcinoma](#)

Eye problems

Impaired vision: although not blind, vision is impaired and may not be fully corrected with glasses.
Varying degrees of near-sightedness or far-sightedness exist.

Photophobia: sensitivity to light or glare

Nystagmus: involuntary movement of the eyes back and forth

Strabismus: eyes do not fixate and track together

Retinal involvement: this is an important area of the eye as it is responsible for sending signals to the brain. Impaired transmission of signals causes various vision disorders

Other less common types of albinism may also involve problems with blood clotting, immune deficiency or problems with hearing.

One concern that should not be overlooked is the risk of isolation in people with albinism. People with albinism, especially children need to be treated normally and included in all activities. They develop normally and have normal intelligence, it is a myth that people with albinism are mentally impaired or intellectually-challenged.

What treatment or precautions can be taken?

It is important for people with albinism to protect themselves from UV exposure and thus prevent the damaging effects it can have on the skin.

Sun avoidance methods

Wear [protective clothing](#) (long sleeves and pants, shirts with collars, tightly woven fabrics that don't let light through), hats (wide-brimmed) and eyewear (specifically made to protect from UV rays)

Use broad-spectrum [sunscreens](#) with SPF of 50 or greater: apply to all exposed areas

Undergo frequent skin examinations by someone who has been taught to recognise signs of [skin cancer](#).

Because the patient has no, or little, pigmentation, skin cancers will often have no or little pigmentation. Patients with albinism should promptly report suspicious spots or growths to a doctor.

Specialist eye doctors cannot cure eye problems but can help with various optical aids to improve vision for people with albinism.

Alopecia areata

Author(s): Hon A/Prof Amanda Oakley, Dermatologist, 1997; Updated: Dr Harriet Bell, Medical Registrar, New Zealand, May 2022. Minor update by Ian Coulson, Dermatologist. Copy edited by Gus Mitchell. July 2024

What is alopecia areata?

Alopecia areata is an autoimmune condition affecting hair follicles causing hair loss. It typically presents with discrete bald patches on the scalp but can cause hair loss from all hair bearing areas.

Alopecia is a Latin term meaning hair loss, and areata refers to the patchy nature of the hair loss. The term alopecia areata is considered an umbrella term, which encompasses a number of variants including alopecia areata totalis or universalis, ophiasis, ophiasis inversus, and [diffuse alopecia areata](#).



A single patch of alopecia areata



Extensive patchy alopecia areata



Alopecia totalis



Ophiasic pattern alopecia areata



Extensive alopecia areata with retention of grey hairs



Dermoscopic image of alopecia areata presenting with exclamation mark hairs

[Click here for more images](#)

Who gets alopecia areata?

The lifetime risk of alopecia areata is approximately 2%. It affects children and adults of all skin and hair colours. Peak incidence occurs in the second and third decades and most patients experience onset before the fourth decade. Alopecia areata does not carry significant sex or ethnic predominance.

The following may increase the risk of alopecia areata:

- Chromosomal disorders such as [Down syndrome](#)
- Polyglandular autoimmune syndrome type 1
- Other autoimmune conditions such as [vitiligo](#) and [thyroid disease](#)
- A family history of alopecia areata
- Certain susceptibility genes (see below).

What causes alopecia areata?

A normal hair follicle cycles through multiple phases:

- Anagen is the active growth phase lasting one to eight years
- Catagen is a short involution phase lasting several weeks
- Telogen is the resting phase lasting several months
- Exogen is the shedding of the hair.

The exact mechanism responsible for hair loss in alopecia areata remains unclear. It is hypothesised that loss of immune privilege in anagen hair follicles plays a key role in the pathogenesis, and genetic susceptibility is also thought to contribute.

Immune privilege hypothesis

Normal anagen hair follicles are thought to exhibit immune privilege, rendering them exempt from immune surveillance and protected against autoimmune attack.

Protective immune privilege may be lost in alopecia areata, allowing hair follicle autoantigens to be presented to autoreactive CD8+ T cells.

Subsequent autoimmune attack of the anagen follicle causes premature transition of the follicle into the telogen phase with ultimate loss of the hair.

This hypothesis is supported by the observation of a dense perifollicular infiltrate of T cells on histopathological examination of anagen follicles in alopecia areata; an area that is normally sparse of immune cells.

Genetic factors

Alopecia areata has a strong hereditary component.

At least 16 genetic risk loci have been detected.

Include numerous human leukocyte antigen (HLA) class I and II alleles, and several alleles of genes involved in immune pathways, hair pigmentation, and response to oxidative stress.

Mode of inheritance appears to be complex, with environmental influences also at play.

What are the clinical features of alopecia areata?

Acute onset of hair loss may manifest in a number of patterns.

Patchy alopecia areata is the most common pattern, producing:

Focal hair loss

Well-demarcated single or several round/oval patches of normal-appearing skin.

The scalp is most commonly affected, but may also affect the:

Beard

Eyebrows

Eyelashes

Any other hair-bearing areas.

Less common patterns include:

Alopecia totalis – complete loss of scalp hair

Affects up to 5% of patients with autoimmune hair loss

Alopecia universalis – complete loss of body hair

Affects less than 1%

Ophiasis – bandlike hair loss on the occipital and temporal scalp margins

Sisaipho (ophiasis inversus) – hair loss on the frontal, temporal, and parietal scalp which may mimic male pattern hair loss

Diffuse alopecia areata (alopecia areata incognita) – rapid and widespread hair loss.

Sudden greying – loss of pigmented hairs, resulting in the unmasking of existing grey hairs ("white overnight").

Other features

Characteristic "**exclamation point hairs**" may be observed, particularly at the periphery of bald patches. An exclamation point hair is a broken strand with a relatively thick distal portion and thin proximal portion as it enters the scalp. They are the result of anagen arrest and cessation of hair shaft formation, with weakening and tapering of the shaft.

Some patients may experience localised tingling or itching preceding hair loss (trichodynia).

Upon regrowth, hairs may initially lack pigment and so grow back as white or blonde.

Nail changes can be seen in an estimated 10–40% of patients and tend to be associated with more severe disease.

Nail pitting and ridging are most common.

Other nail features include [koilonychia](#), [trachyonychia](#), Beau's lines, [onychorrhesis](#), [onychomadesis](#), [onycholysis](#), and red spots in the lunula.

[Click here for images](#)

How do clinical features vary in differing types of skin?

There are no differences in clinical features of alopecia in patients with skin of colour.

What are the complications of alopecia areata?

Poor health-related quality of life due to distress.

Higher rates of depression and generalised anxiety disorder; adjustment disorders are also common.

Other autoimmune conditions may be more commonly observed in patients with alopecia areata e.g. [thyroid disease](#), [vitiligo](#), [psoriasis](#), [diabetes mellitus](#), and atopy.

How is alopecia areata diagnosed?

Alopecia areata is typically diagnosed on clinical features, however additional tests may aid diagnosis.

[Trichoscopy](#):

Examination of the hair follicle, hair shaft, and scalp with a dermatoscope

Features of active disease include exclamation point hairs, broken or dystrophic hairs, yellow dots and black dots.

[Hair pull test](#):

Can help confirm hair loss and is often positive in alopecia areata

Involves grasping 40–60 closely grouped hairs and applying gentle traction

Positive when more than 10% of hairs are easily pulled out.

[Skin biopsy](#):

May be required if diagnosis uncertain

In acute alopecia areata, histopathology reveals a "bee-swarm pattern" of dense lymphocytic infiltrates surrounding anagen hair follicles

Increase in catagen and telogen relative to anagen follicles, and follicle miniaturisation with progression of disease.

How is the severity of alopecia areata assessed?

Scalp alopecia areata is assessed on the basis of the percentage of the scalp that is affected – this is the SALT score.

SALT 0 means there is not hair loss due to alopecia areata.

SALT 50 means 50% of the scalp is affected by alopecia areata. SALT 40 is regarded by sufferers and dermatologists as severe alopecia areata.

Lash and brow alopecia can be difficult to disguise and adds to the burden of the condition.

The ASAMI score (Alopecia Areata Severity and Morbidity Index) has recently been devised to take into account not only sites and extent of alopecia, but sufferer centred symptoms such as difficulty in camouflage, affects on work recreation, relationships, and mental wellbeing.

What is the differential diagnosis for alopecia areata?

Trichotillomania
Temporal triangular alopecia
Central centrifugal cicatricial alopecia
Discoid lupus erythematosus affecting the scalp
Tinea capitis
Telogen effluvium (may mimic [diffuse alopecia areata](#))
Androgenetic alopecia (may mimic sisaihpo pattern)
Secondary [syphilis](#) (syphilitic alopecia)
Lichen planopilaris
Frontal fibrosing alopecia.

What is the treatment for alopecia areata?

There is no cure for alopecia areata. The hair loss in alopecia areata is associated with minimal harmful physical effects and spontaneous resolution may occur.

However, the psychological impact can be significant, therefore warranting treatment. Numerous therapies have been used with variable response and high quality evidence is lacking.

Treatments for mild alopecia areata (less than 50% scalp involvement)

Intralesional corticosteroid injections

Injections of triamcinolone into areas of patchy alopecia of the scalp, beard, or eyebrow have an immunosuppressant effect and may speed up hair regrowth.
Repeated four to six weekly and stopped once regrowth is complete.

Topical treatments

[Potent corticosteroid](#) solutions, creams, or ointments
Most beneficial if used with occlusive dressings
[Minoxidil](#) solution
Ideally in combination with other therapies
[Anthralin](#) (dithranol) cream or ointment
Limited use in fair-haired individuals due to brown staining of skin and hair.

Treatments for extensive alopecia areata (greater than 50% scalp involvement, [alopecia totalis](#), or [universalis](#))

Topical immunotherapy

Chemicals such as [diphenylcyclopropenone](#) are applied to affected areas to induce an [allergic contact dermatitis](#), which may provoke hair regrowth.

T-cells are theorised to be "distracted" from attacking hair follicles due to antigenic competition.

[Severe dermatitis, urticaria, lymphadenopathy](#), and depigmentation are potential side effects and may limit use.

Systemic corticosteroids

Generally reserved for short-term use in refractory or severe cases.

Limited by well-known adverse effects.

Janus kinase (JAK) inhibitors

In June 2022, The FDA approved [baricitinib](#) use in severe alopecia areata.

Clinical trials are ongoing, but preliminary results also show promise for other JAK inhibitors.

JAK inhibitors block the T-cell-mediated inflammatory response that is thought to be responsible for damage to the hair follicle.

Some oral JAK inhibitors are more effective than topical preparations, and significantly more effective than placebos used in large clinical trials.

Baricitinib has received FDA approval for use in the USA.

Ritlecitinib has received FDA and NICE approval – after 24 weeks almost a quarter of patients achieved a SALT score of 20 or less. The response rate increased to over 40% after one year on therapy.

Deuruxolitinib has received FDA approval for severe alopecia areata in adults.

Brepocitinib may be slightly more effective than ritlecitinib, but in one trial it produced a serious inflammatory condition of skeletal muscles in two recipients.

Long term follow up data, outcomes after drug discontinuation, and long term safety remain to be answered. They may be more effective in those who have not had the disease for several years, and in those who do not have alopecia totalis or universalis. Brow and lash regrowth has been documented. They are expensive medications.

Others

Improvement following numerous other less common and less studied treatments have been reported.

Systemic examples include:

[Dupilumab](#)

[Methotrexate](#)

Ezetimibe-simvastatin

Antidepressants.

Localised examples include:

[Topical retinoids](#)

[Platelet-rich plasma](#)

[Micro-needling](#).

Education and counselling

Patients should be informed that there is no cure and response to treatments are variable.

Alopecia areata may spontaneously resolve, persist, relapse, and/or progress. Explaining the various possible disease courses can help manage expectations.

Some patients may benefit from professional counselling to adjust to the appearance altering aspect of the disorder and regain self-confidence.

Consider patient support groups.

Camouflage

Camouflaging hair loss can be helpful for patients who decide against pharmacological treatment or in those who have incomplete response.

A prosthesis can be used to disguise scalp hair loss.

Options include a full wig, hairpiece (clipped or glued to existing hair), or mesh integration system (custom made unit with hair extensions in the areas of alopecia).

Styling products such as gels, mousses, powders, and sprays help to keep hair in place, achieve scalp coverage, and add volume.

False eyelashes or artificial eyebrows

Eyebrow tattooing or microblading can be helpful.

Waterproof eyebrow pencil or eyeliner is a less permanent option.

How do you prevent alopecia areata?

We do not yet know how to prevent alopecia areata.

What is the outcome for alopecia areata?

Alopecia areata follows an unpredictable course. Spontaneous hair regrowth and recovery may occur and is common in some reports. Relapse is also common, however, and patients may have several phases of hair loss and subsequent regrowth. The risk of progression to alopecia totalis or alopecia universalis is approximately 5–10%, from which recovery is unlikely.

Response to treatment is highly variable and hair loss may recur when therapy is stopped.

Poor prognostic factors include:

Younger age at onset

More extensive disease

Hair loss of greater than one-year duration

Nail dystrophy

Ophiasis pattern

Family history of alopecia areata

Presence of atopy or other autoimmune diseases.

[Click here for images](#)

Athlete's foot

Author: Hon A/Prof Amanda Oakley, Dermatologist, Hamilton, New Zealand, 2003.

What is athlete's foot?

Athlete's foot is a term often used to describe a [fungal infection](#) (or dermatophytosis) of the foot ([tinea pedis](#)).

It most often results in peeling skin and fissuring between the toes (the toe clefts). The cleft between the fourth and fifth toes is the most frequently affected.

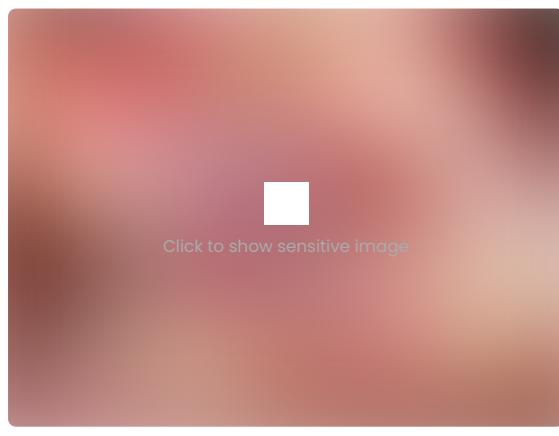
Fungal infection is not the only reason for peeling and fissuring between the toes, and the term athlete's foot is sometimes used to refer to any condition where the toe clefts are inflamed.



Athlete foot



Athlete foot



Athlete foot

What causes athlete's foot?

Athlete's foot is generally due to proliferation of dermatophyte fungi of the genera *Trichophyton rubrum*, *Trichophyton interdigitale*, and *Epidemophyton floccosum*.

Predisposing factors

Athlete's foot is more common in those who participate in sports because:

- They may wear occlusive footwear
- They sweat heavily
- They may fail to dry their feet carefully after showering
- They are exposed to fungal spores on the surfaces of communal areas.

What are the clinical features of athlete's foot?

The clinical features of athlete's foot may include:

- Moist, peeling skin between the toes
- White, yellow, or greenish discolouration
- Sometimes, thickened skin
- Painful fissures
- Unpleasant smell.

Athlete's foot is generally mild; very inflamed athlete's foot is generally due to [secondary bacterial infection](#).

How is athlete's foot diagnosed?

The diagnosis is usually clinical, as athlete's foot has a characteristic appearance. If resistant to treatment, investigations are undertaken to identify a specific infection.

- Scrapings for [fungal microscopy and culture](#)
- Swabs for [bacterial microscopy and culture](#)
- [Wood's light](#) (ultraviolet) examination looking for coral-red fluorescence, characteristic of erythrasma

What is the differential diagnosis of athlete's foot?

Athlete's foot is a localised interdigital form of [intertrigo](#).

Apart from tinea pedis, peeling and fissuring between the toes can be due to:

- [Yeast infection \(*candida*\)](#)
- [Bacterial infection \(erythrasma, pseudomonas, staphylococci and streptococci\)](#)
- [Mould infection](#)
- [Soft corn](#) (build-up of thick skin due the repetitive trauma of the toes pressing against each other)
- Injury, for example over-vigorous removal of peeling skin
- A skin condition, such as [psoriasis](#) (scaly patches in scalp, on elbows and knees or rashes in body folds), [foot eczema](#) or [keratolysis exfoliativa](#).

Non-fungal causes of athlete's foot



Pseudomonas infection



Psoriasis



Eczema

What is the treatment of athlete's foot?

Treatment for athlete's foot should begin with general measures.

Dry carefully between the toes.

Use a dusting powder to keep the affected area dry.

Keep toes apart using a cotton or foam wedge.

Wear shoes that are loose around the toes or go bare foot.

Apply a [topical antifungal](#) agent. These may also control many of the bacteria that live in the moist skin between the toes.

Whitfield ointment (3% salicylic acid, 6% benzoic acid in petrolatum) is particularly useful, as it removes the surface layer of moist peeling skin (ie, it is keratolytic), and it eliminates bacteria and fungi.

Make sure that other sites of fungal infection are also treated effectively.



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Basal Cell Carcinoma Overview

The Most Common Skin Cancer

Basal cell carcinoma (BCC) is the most common form of skin cancer and the most frequently occurring form of all cancers. In the U.S. alone, an estimated 3.6 million cases are diagnosed each year. BCCs arise from abnormal, uncontrolled growth of basal cells.

Because BCCs grow slowly, most are curable and cause minimal damage when caught and treated early. Understanding BCC causes, risk factors and warning signs can help you detect them early, when they are easiest to treat and cure.

- [What is a basal cell?](#)
- [What does BCC look like?](#)
- [How dangerous is BCC?](#)
- [How widespread is BCC?](#)
- [What is advanced BCC?](#)
- [Skin Cancer Awareness Toolkit](#)

FACT:

BCC

is serious and should be addressed as soon as possible.





Warning Signs



Treatment



What is a basal cell?

One of three main types of cells in the top layer of the skin, basal cells shed as new ones form. BCC most often occurs when DNA damage from exposure to ultraviolet (UV) radiation from the sun or indoor tanning triggers changes in basal cells in the outermost layer of skin (epidermis), resulting in uncontrolled growth.

What does BCC look like?

BCCs can look like open sores, red patches, pink growths, shiny bumps, scars or growths with slightly elevated, rolled edges and/or a central indentation. At times, BCCs may ooze, crust, itch or bleed. The lesions commonly arise in sun-exposed areas of the body. In patients with darker skin, about half of BCCs are pigmented (meaning tan, black or brown in color) and can be mistaken for a normal mole.

It's important to note that BCCs can look quite different from one person to another. Visit our [BCC Warning Signs](#) page for more images and information on BCC signs, symptoms and early detection strategies.

Please note: Since not all BCCs have the same appearance, these photos serve as a general reference to what they can look like. If you see something new, changing or unusual on your skin, schedule an appointment with your dermatologist.



An open sore that does not heal



A shiny bump or nodule



A reddish patch or irritated area



A scar-like area that is flat white, yellow or waxy in color



A small pink growth with a slightly raised, rolled edge and a crusted indentation in the center



A basal cell carcinoma may be pigmented on skin of color. Photo: Andrew Alexis, MD, MPH

How dangerous is BCC?

BCCs rarely spread beyond the original tumor site. But these lesions can grow and become disfiguring and dangerous. Untreated BCCs can become locally invasive, grow wide and deep into the skin and destroy skin, tissue and bone. The longer you wait to get treatment, the more likely it is that the BCC will recur, sometimes repeatedly.

There are some highly unusual, aggressive cases when BCC spreads to other parts of the body. In even rarer instances, this type of BCC can become life-threatening.

How widespread is BCC?

Basal cell carcinoma is quite common. The number of reported cases in the U.S. has steadily increased.

- An estimated 3.6 million Americans are diagnosed with BCC each year.
- More than one out of every three new cancers are skin cancers, and the vast majority are BCCs.
- The diagnosis and treatment of nonmelanoma skin cancers, including BCC and squamous cell carcinoma (SCC), increased up to 77 percent between 1994 and 2014.

May Is Skin Cancer Awareness Month

Ronald L. Moy, MD

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Sam Champion's Skin Cancer Journey: A Wake-Up Call for Regular Skin Exams

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Melissa's Battle Against Chronic Skin Cancer: "It Keeps Coming Back."

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Celebrities and Skin Cancer – They're Just Like Us

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Last updated: January 2025

May Is Skin Cancer Awareness Month



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Pressure ulcer

March 2023

Author(s): Dr Tristen Ng, Royal Perth Hospital, Western Australia (2023)

Previous contributors: Vanessa Ngan, Staff Writer (2003)

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Edited by the DermNet content department

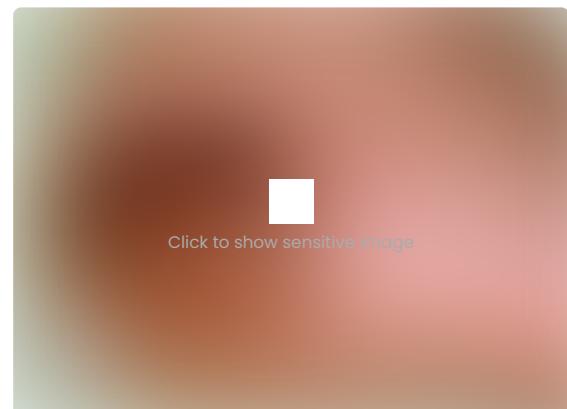
What are pressure ulcers?

Pressure ulcers are skin and soft tissue injuries sustained from prolonged pressure. Specifically, they involve a breakdown of the skin, subcutaneous tissues and sometimes even deeper structures (tendons, muscle, bone) caused by cumulative pressure and are often related to pre-existing health conditions or injuries.

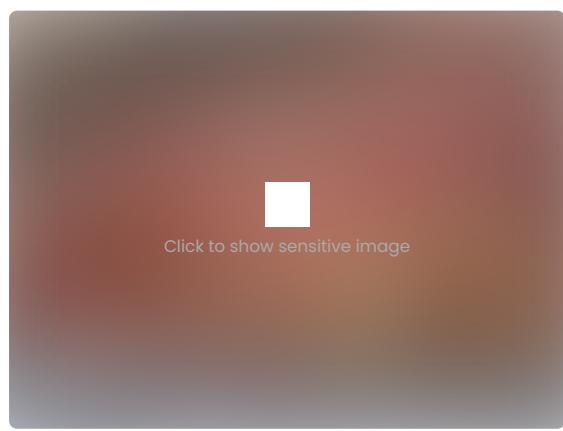
Pressure ulcers are also known as pressure sores, decubitus ulcers, or bed sores. They are often found on bony areas of the body with a thin soft tissue covering.



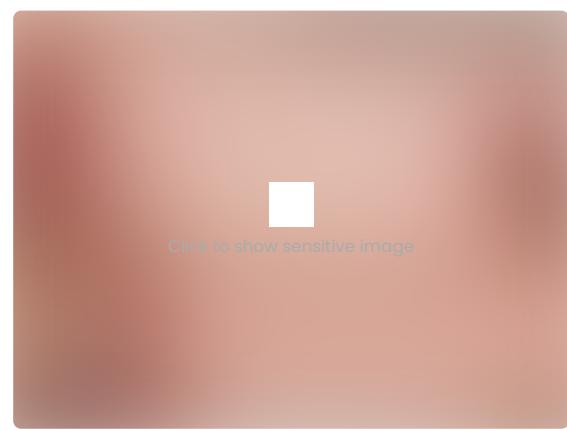
Incipient pressure ulcer on the heel - patient was paraplegic after a spinal injury



Pressure sore



Chronic pressure ulceration due to immobility and anaesthesia due to spina bifida



Pressure ulcers on gluteal fold and popliteal fossa in a wheelchair user with spina bifida



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A deep and recalcitrant sacral pressure ulcer



Click to show sensitive image

Chronic pressure ulcer

[View more images](#)

Who gets pressure ulcers?

Although pressure ulcers can affect anyone, they are most often seen in the elderly and critically ill who are immobile for long periods.

High-risk groups include palliative care patients, [comatose](#) patients, [quadriplegics](#), patients with spinal cord injuries, elderly people with orthopaedic fractures, and children with neurological dysfunction (eg, spina bifida, cerebral palsy, or spinal cord injury).

Pressure ulcers carry a high economic and psychological burden, due to hospital admissions for treatment of pressure ulcer complications and reduced quality of life for affected patients.

What causes pressure ulcers?

The most important cause of pressure ulcers is external pressure at a skin site for prolonged periods, although the exact mechanism is complex and poorly understood.

After the initial external pressure is gone, patients have a delay, or reperfusion time which increases the risk of [ulcer](#) formation.

A 'prolonged inflammatory phase' hypothesis has also been proposed, whereby pressure ulcers do not follow the normal trajectory of inflammation, remodelling, and maturation, but are instead arrested in the 'inflammatory' phase of [wound](#) healing.

Identifying external and internal risk factors is important to prevent or minimise pressure ulcers.

External factors include:

Trauma to the skin (eg, skin tears from dressings, lacerations)

[Amputations](#)

Physical restraints or chemical sedation, worsening immobility

Inadequate changes in the positioning of immobile patients

Ill-fitted medical devices (eg, Jewett braces, orogastric and nasogastric tubes, tracheostomy tubes, prosthetics, casts, and splints).

Internal factors include:

Neurological diseases (both motor and sensory)

Metabolic syndromes (eg, [obesity](#), [diabetes mellitus](#))

Cardiovascular disease

Peripheral arterial occlusive disease

[Malnutrition](#)

Advanced age.

What are the clinical features of pressure ulcers?

The clinical features of pressure ulcers range from inflamed-looking, to severely ulcerated skin exposing muscle, tendon, and even bone. Commonly affected sites include the skin overlying the coccyx, vertebral column, heels, ankles, and elbows.

For patients who spend prolonged periods lying on their side, the iliac crest, the trochanters, and the ear helix may be affected. During the prolonged prone nursing of patients with severe COVID-induced respiratory disease, facial pressure ulcers were frequent.

The revised National Pressure Ulcer Advisory Panel's (NPUAP) Pressure Injury Staging System is widely used in the staging and severity assessment of pressure ulcers based on their clinical features.

Other scoring systems, such as the Braden scale, are also used in some healthcare institutions.

Stage 1 pressure ulcers

Intact skin with various degrees of erythema that does not blanch (turn white) when compressed.

Skin may be tender, itchy, or painful.

Stage 2 pressure ulcers

Skin is red, swollen, and painful.

Partial-thickness skin loss involving a break in the dermis.

Ulcers appear shiny or dry with a red-pink wound bed with serum-filled blisters.

Upper layers of skin begin to die.

Adipose tissue, granulation tissue, slough, and eschar are absent.

Stage 3 pressure ulcers

Full-thickness skin loss involving the hypodermis.

Crater-like ulceration is present.

Adipose tissue may be seen but not muscle, tendon, ligament, cartilage, or bone.

The depth of tissue damage varies by anatomical location (eg, may appear shallow in low adiposity areas such as occiput and malleolus, while high adiposity areas like the gluteal area may appear deep).

Wounds are prone to [infection](#).

Stage 4 pressure ulcers

Full thickness with exposed muscle, tendon, ligament, cartilage, joint, or bone.

Blackened dead tissue called eschar may be seen in deep open wounds.

The risk of osteomyelitis is extremely high.

[View images](#)

How are pressure ulcers diagnosed?

Pressure ulcers remain a clinical diagnosis. The patient's skin should be examined thoroughly from scalp to toe. Special attention must be given to skin in common pressure sites, under medical devices, and skin folds in patients with larger body habitus.

The mnemonic 'BEST SHOT' is used by the NHS Stop the Pressure campaign as a checklist for common pressure ulcer sites:

- Buttocks
- Elbows and ears
- Sacral area
- Trochanters
- Spine and shoulders
- Heels
- Occiput
- Toes.

Temperature sensing technologies such as infrared thermography (IRT) have been developed to aid early prediction and early diagnosis of pressure ulcers. In a blinded prospective study of 70 patients in an ICU, IRT was found to detect skin changes 5–18 days before the visible appearance of pressure ulcers.

How do clinical features vary in differing types of skin?

Stage 1 pressure ulcers may be missed in darker [skin types](#) (eg, Fitzpatrick type 4–6) due to the absence of visible blanching or erythema.

Other parameters such as altered skin sensation, warmth, and skin firmness should be assessed in patients with darker skin types.

What are the complications of pressure ulcers?

- Infection ([cellulitis](#), [abscess](#), osteomyelitis, septic arthritis, [necrotising fasciitis](#), sepsis)
- Malignant transformation
- Ulcer recurrence

What is the differential diagnosis for pressure ulcers?

- Superficial [friction injuries](#) or excoriations
- [Thermal burns](#)
- [Cellulitis](#)
- [Marjolin ulcer](#)
- [Martorell ulcer](#)
- Vasculitides eg, [cutaneous vasculitis](#)
- Radiation injuries eg, [radiation dermatitis](#)
- [Delayed pressure urticaria](#)
- [Diabetic foot ulcers](#)
- Leg ulcers eg, [stasis ulcer](#), [arterial ulcer](#)
- [Pyoderma gangrenosum](#).

What is the treatment for pressure ulcers?

General measures

Alleviate external factors eg, cleanse and dry skin after [incontinence](#), and use [barrier creams](#) and [emollients](#).

[Special dressings](#) and [honey preparations](#) may be used to help the healing process.

Dead tissue may be removed with a scalpel (debridement).

Improve internal factors eg, patient nutrition.

Optimise the wound bed for maximal healing.

Minimise pressure on the affected area by turning and pressure relieving devices (cushions, mattresses).

Specific measures

Occlusive wound dressings to maintain a moist wound environment.

Regular patient and [wound care](#) reviews by a multidisciplinary team (may involve endocrinologists, neurologists, geriatricians, wound care nurses, infectious disease specialists, podiatrists, dietitians, and occupational therapists).

[Negative pressure dressings](#) for severe pressure ulcers.

Sometimes [maggot debridement therapy](#) is used to remove necrotic material and eschar.

Hydrotherapy debridement, using saline solution in a syringe or a water pressure jet.

[Antibiotics](#) if required for infection.

Healthy skin may be [grafted](#) onto the damaged area.

[Bioengineered skin](#) is also an emerging alternative therapy for skin grafting.

In severe or life-threatening situations, amputation of a limb may be necessary.

How do you prevent pressure ulcers?

Prevention of pressure ulcers can be classified into 3 domains: promoting movement, pressure reduction, and pressure distribution.

Prevention strategies include:

Meticulous skin care eg, [emollients](#), gentle [cleansers](#), and avoiding friction and shearing forces

Optimising patient nutrition and movement

Alternating pressure (active) air beds and mattresses (commonly used in healthcare settings to reduce and distribute pressure in hospitalised patients)

Corrugated viscoelastic foam surfaces

Special heel elevators and Prevalon boots

Individual patient positioning plans and mattress selection in community settings such as residential aged care facilities.

What is the outcome of pressure ulcers?

Prevention and early detection are crucial, as stage 3–4 ulcers take weeks to months to heal and can be resource-intensive and expensive to manage.

Surgery is only indicated in patients whose wounds are refractory to non-invasive management; these patients also need to be fit for surgery.

[Infection](#) and sepsis can be severe and life-threatening; systemic [antibiotic therapy](#) may be required.

Vacuum pumps are not routinely used as they require input from experienced wound care nurses; improper use can lead to further exacerbation of pressure ulcers.

[View images](#)

Boil

Author: Dr Amanda Oakley Dermatolo^g
 Latest update by Dr Jannet Gomez, Po
 January 2016.

n, Staff Writer; June 2014.
 n, United Kingdom,

What is a boil?

A boil (also called a furuncle) is a deep form of **bacterial folliculitis** (infection of a hair follicle).

What are the clinical features of a boil?

Boils present as one or more tender red spots, lumps or pustules. Careful inspection reveals that the boil is centred on a hair follicle. A boil is a deep form of bacterial **folliculitis**; superficial folliculitis is sometimes present at the same time. *Staphylococcus aureus* can be cultured from the skin lesions.

If there are multiple heads, the lesion is called a carbuncle. Large boils form **abscesses**, defined as an accumulation of pus within a cavity. **Cellulitis** may also occur, ie, infection of the surrounding tissues, and this may cause fever and illness.



Boil



Boil in axilla



Surrounding cellulitis



Click to show sensitive image

Carbuncle



Why do boils occur?

Most people with boils are otherwise healthy and have good personal hygiene. They do however carry *Staphylococcus aureus* on the surface of their skin (staphylococcal carrier state). Why this occurs is usually not known, but it is estimated that 10–20% of the population are staphylococcal carriers.

Staphylococcus aureus is most commonly carried in the nostrils, armpits, between the legs and in the cleft between the buttocks. It may be transferred to other sites from the nostrils via the finger nails.

Tiny nicks or grazes or something rubbing against the skin can inoculate the bacteria into the wall of a hair follicle which is a weak point in the skin's defences. Once inoculated, the bacteria cause a boil which goes on to run its usual course of about 10 days.

Although most people with boils are otherwise healthy, boils are sometimes related to immune deficiency, anaemia, diabetes, smoking or [iron deficiency](#).

What is the treatment for a boil?

Medical treatment of boils

Treatment of boils depends on their severity. Your doctor may give you specific advice and medical treatment, some are listed below:

Antiseptic or [antibacterial soap](#) in your daily bath or shower for a week then twice weekly for several weeks. The cleanser may cause a little dryness.

Use a hand sanitiser regularly to reduce the chance of reinfecting yourself or others with contaminated hands.

Antiseptic or antibiotic ointment or gel to apply to the inside of the nostrils.

Wipe the entire skin surface daily for a week with 70% isopropyl alcohol in water (this will make the skin dry).

Apply a topical antiseptic such as povidone [iodine](#) or chlorhexidine cream to the boils and cover with a square of gauze.

Your doctor may prescribe an oral [antibiotic](#) (usually the [penicillin](#) antibiotic flucloxacillin), sometimes for several weeks.

Other members of the family with boils should also follow a skin cleansing regime. Your doctor may also advise the family to apply topical antibiotic to their nostrils in case they are *Staphylococcus aureus* carriers as well.

If the boils fail to clear up, a swab should be taken for microbiological culture, in case of [methicillin \(meticillin\) resistant staphylococci](#).

Sometimes, special antibiotics may be prescribed on the recommendation of a specialist, including [fusidic acid](#), [clindamycin](#), [rifampicin](#) and cephalosporins.

General measures to prevent boils

Consult your doctor about your general health.

If you are overweight, try to reduce your weight; take regular exercise.

Follow a balanced healthy diet with meat, plenty of fruit and vegetables.

Avoid smoking.

Wash your whole body once a day with [soap or cleanser](#) and water. Wash your hands several times daily or use antiseptic [hand rubs](#).

Don't share your flannel or towel with other family members.

Maintain a clean handkerchief and don't pick your nose!

Change your underclothes and night attire regularly.

Consider modifying leisure activities that cause sweating and friction from clothing, such as squash and jogging.

If you are [iron deficient](#), a course of iron tablets may help reduce infection.

1000 mg of vitamin C each day has also been advocated to improve deficient neutrophil function.

Cellulitis

Author: Dr Amy Stanway, Department of Dermatology, Waikato Hospital, Hamilton, New Zealand, 2001. Reviewed and updated by Dr Jannet Gomez, Postgraduate Student in Clinical Dermatology, Queen Mary University London, United Kingdom; Chief Editor, Hon A/Prof Amanda Oakley, Dermatologist, Hamilton, New Zealand, July 2016.

What is cellulitis?

Cellulitis is a common bacterial skin infection of the lower dermis and subcutaneous tissue. It results in a localised area of red, painful, swollen skin, and systemic symptoms. Left untreated, cellulitis can be life-threatening.

Similar symptoms are experienced with the more superficial infection, [erysipelas](#), so cellulitis and erysipelas are often considered together.



Cellulitis of the left leg



Who gets cellulitis?

Cellulitis affects people of all ages and races.

Risk factors for cellulitis include:

Previous episode(s) of cellulitis

Fissuring of toes or heels, eg due to [athlete's foot](#), [tinea pedis](#) or [cracked heels](#)

Venous disease, eg [gravitational eczema](#), [leg ulceration](#), and/or [lymphoedema](#)

Current or prior injury, eg trauma, surgical [wounds](#), [radiotherapy](#), [IV drug use](#)

Immunodeficiency, eg [human immunodeficiency virus infection \(HIV\)](#)

Immune suppressive medications

[Diabetes](#)

Chronic kidney disease

Chronic liver disease

[Obesity](#)

[Pregnancy](#)

[Alcoholism](#)

Many people falsely attribute an episode of cellulitis to an unseen [spider bite](#). Documented spider bites have not led to cellulitis.

What causes cellulitis?

The most common bacteria causing cellulitis are [*Streptococcus pyogenes*](#) (two-thirds of cases) and [*Staphylococcus aureus*](#) (one third). Rare causes of cellulitis include:

Pseudomonas aeruginosa, usually in a puncture wound of foot or hand

Haemophilus influenzae, in children with facial cellulitis

Anaerobes, *Eikenella*, *Streptococcus viridans*, due to human bite

Pasteurella multocida, due to cat or dog bite

Vibrio vulnificus, due to saltwater exposure, eg coral injury

Aeromonas hydrophila from fresh or saltwater exposure, eg following leech bites

Erysipelothrix (erysipeloid), in butchers.

Other forms of skin injury that may increase bacterial exposure and cellulitis infection include surgical wounds and insect bites.

Cellulitis is generally not contagious as it affects the deeper layers of the skin.

What are the clinical features of cellulitis?

Cellulitis can affect any site, but most often affects the limbs

It is usually unilateral; a bilateral disease is more often due to another condition

It can occur by itself or complicate an underlying skin condition or wound.

The first sign of the illness is often feeling unwell, with fever, chills and shakes (rigors). This is due to bacteria in the bloodstream (bacteraemia). Systemic symptoms are soon followed by the development of a localised area of painful, red, swollen skin. Other signs include:

Dimpled skin (*peau d'orange*)

Warmth

Blistering

Erosions and ulceration

[Abscess formation](#)

[Purpura](#): petechiae, ecchymoses, or haemorrhagic bullae

Cellulitis may be associated with lymphangitis and lymphadenitis, which are due to bacteria within lymph vessels and local lymph glands. A red line tracks from the site of infection to nearby tender, swollen lymph glands.

After successful treatment, the skin may flake or peel off as it heals. This can be itchy.

What are the complications of cellulitis?

Severe or rapidly progressive cellulitis may lead to complications that require prompt treatment:

Necrotising fasciitis (a more serious soft tissue infection recognised by severe pain, skin pallor, loss of sensation, purpura, ulceration and necrosis)

Gas gangrene

Severe sepsis (blood poisoning)

Infection of other organs, eg pneumonia, osteomyelitis, meningitis

Endocarditis (heart valve infection).

Sepsis is recognised by fever, malaise, loss of appetite, nausea, lethargy, headache, aching muscles and joints. The serious infection leads to hypotension (low blood pressure, collapse), reduced capillary circulation, heart failure, diarrhoea, gastrointestinal bleeding, renal failure and loss of consciousness.

How is the diagnosis of cellulitis made?

The diagnosis of cellulitis is primarily based on clinical features including a physical exam.

Investigations may reveal:

Leukocytosis (raised white cell count).

Elevated C-reactive protein (CRP)

The causative organism, on the culture of blood or of pustules, crusts, erosions or wound.

Imaging may be performed. For example:

Chest X-ray in case of heart failure or pneumonia

Doppler ultrasound to look for blood clots (deep vein thrombosis)

MRI in case of necrotising fasciitis.

What is the differential diagnosis of cellulitis?

Cellulitis is often diagnosed when another inflammatory skin disease is actually responsible for redness and swelling. Conditions causing 'pseudocellulitis' include:

Eczema/dermatitis due to **stasis, contact** factors

Fungal infection eg **tinea corporis, tinea pedis**

Deep vein thrombosis

Drug eruption

Psoriasis

Lipodermatosclerosis

Thrombophlebitis

Insect bites and stings

Radiation damage following **radiotherapy**

Inflammatory breast cancer (**carcinoma erysipeloides**).

What is the treatment for cellulitis?

Cellulitis is potentially serious. The patient should rest and elevate the affected limb. The edge of the involved area of swelling should be marked to monitor progression/regression of the infection.

Knowledge of local organisms and resistance patterns is essential in selecting appropriate [antibiotics](#). The management of cellulitis is becoming more complicated due to rising rates of [methicillin-resistant *Staphylococcus aureus* \(MRSA\)](#) and macrolide- or [erythromycin-resistant *Streptococcus pyogenes*](#).

Treatment of uncomplicated cellulitis

If there are no signs of systemic illness or extensive infection, patients with mild cellulitis can be treated with oral [antibiotics](#) at home, for a minimum of 5–10 days. In some cases, antibiotics are continued until all signs of infection have cleared (redness, pain and swelling), sometimes for several months. Treatment should also include:

- Analgesia to reduce pain
- Adequate water/fluid intake
- Management of co-existing skin conditions like [venous eczema](#) or [tinea pedis](#)

Treatment of cellulitis with systemic illness

More severe cellulitis and systemic symptoms should be treated with fluids, intravenous antibiotics and oxygen. The choice of antibiotics depends on local protocols based on prevalent organisms and their resistance patterns and may be altered according to culture/susceptibility reports.

- [Penicillin](#)-based antibiotics are often chosen (eg penicillin G or flucloxacillin)
- Amoxicillin and clavulanic acid provide broad-spectrum cover if unusual bacteria are suspected
- Cephalosporins are also commonly used (eg ceftriaxone, cefotaxime or cefazolin)
- [Clindamycin](#), sulfamethoxazole/trimethoprim, [doxycycline](#) and vancomycin are used in patients with penicillin or cephalosporin allergy, or where infection with [methicillin-resistant *Staphylococcus aureus*](#) is suspected
- Broad-spectrum antibiotics may also include linezolid, ceftaroline, or daptomycin

Sometimes oral probenecid is added to maintain antibiotic levels in the blood.

Treatment may be switched to oral antibiotics when the fever has settled, cellulitis has regressed, and CRP is reducing.

Multidisciplinary care

- An internal medicine physician is consulted to assess and manage sepsis.
- The infectious diseases service can advise on microbiology and choice of antibiotic.
- A surgeon is called to drain an abscess, debride necrotic tissue, and relieve compression symptoms, eg compartment syndrome.
- An ophthalmologist should be involved in the case of [orbital cellulitis](#).
- A dermatologist may be called to confirm the diagnosis of cellulitis or suggest alternative diagnoses.
- Specialist nurses may advise on dressings and bandaging.

What is the management of recurrent cellulitis?

Patients with recurrent cellulitis should:

Avoid trauma, wear long sleeves and pants in high-risk activities, such as gardening
Keep skin clean and well moisturised, with nails well tended
Avoid having blood tests taken from the affected limb
Treat [fungal infections](#) of hands and feet early
Keep swollen limbs elevated during rest periods to aid lymphatic circulation. Those with chronic [lymphoedema](#) may benefit from [compression](#) garments.

Patients with 2 or more episodes of cellulitis may benefit from chronic suppressive antibiotic treatment with low-dose [penicillin](#) V or [erythromycin](#), for one to two years.

Chickenpox

Author: Vanessa Ngan, Staff Writer, 2002. Updated by Dr Jannet Gomez, October 2016; Dr Adam Dedat, SHO and Dr Ian Coulson, Dermatologist, United Kingdom, July 2022.

What is chickenpox?

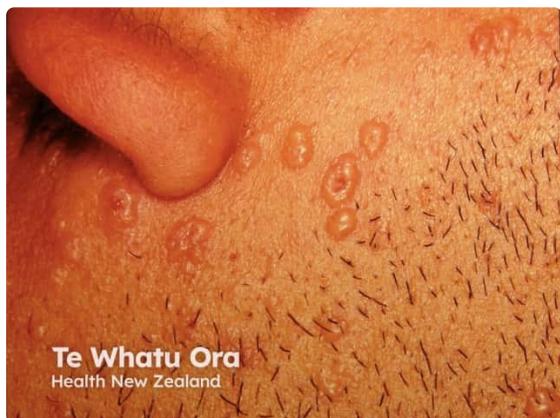
Chickenpox is a highly contagious viral infection that causes an acute fever and blistered rash, mainly in children. Chickenpox is also known as varicella.

The name may be derived from the French term for chickpea, chiche pois. Another theory is that the word 'chicken' was derived from a slang term for 'child'.

Skin rash of chickenpox



Chickenpox on back



Chickenpox



Chickenpox

Who is at risk of chickenpox?

Chickenpox occurs worldwide, affecting persons of all races, sex and age. Most cases occur in children before they are ten years of age.

Once a person has had the chickenpox infection, it is unlikely he or she will get it again, as it confers lifelong immunity.

Immunocompromised individuals are susceptible to the virus at all times and should take measures to prevent or modify the course of the disease if there has been exposure to the virus.

What is the cause of chickenpox?

Chickenpox is caused by primary infection with the varicella-zoster virus, of the *Herpesviridae* family. This virus is sometimes called herpesvirus type 3.

Chickenpox is highly contagious and is easily spread from person to person by breathing in airborne respiratory droplets from an infected person's coughing or sneezing or through direct contact with the fluid from the open sores.

A person who is not immune to the virus has a 70–80% chance of being infected with the virus if exposed to someone in the early stages of the disease.

What are the clinical features of chickenpox?

In children, chickenpox usually begins as itchy red papules progressing to vesicles on the stomach, back and face, and then spreading to other parts of the body. Blisters can also arise inside the mouth

The spread pattern can vary from child to child. There may be only a scattering of vesicles, or the entire body may be covered with up to 500 vesicles. The vesicles tend to be very itchy and uncomfortable.

Some children may also experience additional symptoms such as high fever, headache, cold-like symptoms, vomiting and diarrhoea.

Chickenpox is usually more severe in adults and can be life-threatening in complicated cases. Most adults who get chickenpox experience prodromal symptoms for up to 48 hours before breaking out in the rash. These include fever, malaise, headache, loss of appetite and abdominal pain. Chickenpox is usually more severe in adults and can be life-threatening in complicated cases.

The blisters clear up within one to three weeks but may leave a few scars. These are most often depressed ([anetoderma](#)), but they may be thickened ([hypertrophic scars](#)). Scarring is prominent when the lesions get infected with bacteria.

Cutaneous features of chickenpox



Chickenpox on face



Chickenpox

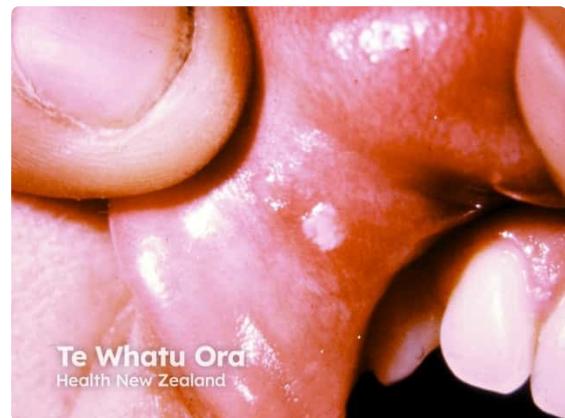


Chickenpox

Oral mucosal lesions of chickenpox



Chickenpox



Chickenpox

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How is chickenpox diagnosed?

Diagnosis of chickenpox is usually made on the presence of its characteristic rash and the presence of different stages of lesions simultaneously. A clue to the diagnosis is in knowing that the patient has been exposed to an infected contact within the 10–21 day incubation period. Patients may also have prodromal signs and symptoms. See also [chickenpox pathology](#).

Laboratory tests are often undertaken to confirm the diagnosis.

PCR detects the varicella virus in skin lesions and is the most accurate method for diagnosis.

The culture of blister fluid is time-consuming and is less frequently performed.

Serology (IgM and IgG) is most useful in pregnant women, or before prescribing immune suppression medication to determine the need for pre-treatment immunisation.

What are the complications from chickenpox?

In healthy children, chickenpox infection is usually an uncomplicated, self-limiting disease.

Complications may include:

Secondary bacterial infection of skin lesions caused by scratching

Infection may lead to abscess, cellulitis, necrotising fasciitis and gangrene

Dehydration from vomiting and diarrhoea

Exacerbation of asthma

Viral pneumonia

Chickenpox lesions may heal with scarring.

Some complications are more commonly seen in immunocompromised and adult patients with chickenpox.

Disseminated primary varicella infection; this carries high morbidity

Central nervous system complications such as Reye syndrome, Guillain–Barré syndrome and encephalitis

Thrombocytopenia and [purpura](#)

Varicella in pregnancy

Non-immune pregnant women should take care to avoid contact with people who have chickenpox and to wash hands frequently when handling food, animals, and children. Exposure to varicella virus in pregnancy may cause viral pneumonia, premature labour and delivery and rarely maternal death.

Approximately 25% of fetuses of mothers with chickenpox become infected. It is harmless to most of them. Offspring may remain asymptomatic, or develop herpes zoster at a young age without a previous history of primary chickenpox infection. They may also develop congenital varicella syndrome, one of the [TORCH](#) infections.

Congenital varicella syndrome occurs in up to 2% of fetuses exposed to varicella in the first 20 weeks of gestation. It can result in spontaneous abortion, fetal chorioretinitis, cataracts, limb atrophy, cerebral cortical atrophy and microcephaly, cutaneous scars, and neurological disability.

Mortality in newborns infected with varicella is up to 30%.

Perinatal varicella

If a mother develops chickenpox just before delivery or during the 28 days after delivery, her baby is at risk of severe infection.

Shingles (herpes zoster)

The varicella-zoster virus remains dormant in sensory ganglia after infection.

It may reactivate after many years as shingles. Shingles presents with grouped vesicular lesions, which usually affect a single dermatome.

Other infections occurring as a result of reactivation of virus include post-herpetic neuralgia, vasculopathy, myelopathy, retinal necrosis, cerebellitis and zoster sine herpete.

Complications of chickenpox

Scars from chickenpox

Herpes zoster

What is the treatment for chickenpox?

For most healthy patients with chickenpox symptomatic therapy is usually all that is required.

Trim children's fingernails to minimise scratching.

Take a warm bath and apply moisturising cream.

Paracetamol can reduce fever and pain

Avoid NSAID use outside of hospital settings due to the increased risk of severe cutaneous complications such as invasive [group A streptococcal superinfections](#).

Do not use aspirin in children as this is associated with Reye syndrome.

Calamine lotion and oral antihistamines may relieve itching.

Consider oral [aciclovir](#) (antiviral agent) in people older than 12 years, which reduces the number of days with a fever.

Immunocompromised patients with chickenpox need intravenous treatment with the antiviral [aciclovir](#).

In cases of inadvertent exposure to the virus, varicella-zoster immune globulin if given within 96 hours of initial contact can reduce the severity of the disease though not prevent it. This is used where there is no previous history of chickenpox (or the patient has no antibodies to the varicella-zoster virus on blood testing) in pregnancy, in the first 28 days after delivery, and in [immune deficient](#) or [immune-suppressed](#) patients.

How to prevent the spread of chickenpox

A person with chickenpox is contagious 1–2 days before the rash appears and until all the blisters have formed scabs. This may take 5–10 days. Children should stay away from school or childcare facilities throughout this contagious period. Adults with chickenpox who work among children should also remain home.

It can take 10–21 days after contact with an infected person for someone to develop chickenpox. This is how long it takes for the virus to replicate and come out in the characteristic rash in the new host.

As chickenpox may cause complications in immunocompromised individuals and pregnant women, these people should avoid visiting friends or family when there is a known case of chickenpox. In cases of inadvertent contact, see your doctor who may prescribe special preventive treatment.

Vaccination against chickenpox

Vaccination is available for chickenpox and is highly recommended.

Chickenpox is highly preventable by vaccination with live attenuated varicella vaccine. The vaccine is subsidised ("scheduled") for infants aged 15 months in New Zealand as well as non-immune individuals who are [immunosuppressed](#) or are in other special groups. If in New Zealand, refer to the [Immunisation Advisory Centre](#) for up-to-date information.

Cholinergic urticaria

Author: Dr Lisa Murphy, Beaumont Hospital, Ireland. Copy edited by Gus Mitchell. July 2022

What is cholinergic urticaria?

Cholinergic urticaria is a common [chronic inducible urticaria](#) that is characterised by the presence of short-lived transient [hives](#) (itchy bumps) due to stimuli that induce sweating. It typically presents with small, raised 1–4 mm wheals which last for 15–30 minutes.

It is also sometimes referred to as cholinergic angioedema or heat bumps.

[Click here for images](#)

Who gets cholinergic urticaria?

The prevalence of this physical urticaria is higher in persons with [chronic spontaneous urticaria](#). While the disorder occurs in both sexes, it occurs more frequently in males. It typically first develops in people aged 10–30 years.

What causes cholinergic urticaria?

Any stimulus that can cause [excessive sweating](#) can precipitate this inducible urticaria.

This includes:

- Exercise (most common trigger)
- High temperature exposure, eg, hot water baths
- Spicy food ingestion
- Emotional stress.

There are several theories regarding the pathogenesis including:

- Affected patients may have an increased number of muscarinic receptors on cutaneous mast cells in areas that demonstrate hives, therefore the cholinergic nervous system is the cause.
- The urticaria occurs as a result of a rise in core body temperature, associated with sweating.
- Other authorities postulate that rather than shifts in core body temperature, IgE-mediated allergy to a component of human sweat is the immediate trigger.

What are the clinical features of cholinergic urticaria?

Heat bumps typically present with:

- A number of small (1–4 mm) punctate wheals, often surrounded by a larger erythematous flare
- Wheals are [itchy](#) or burn
- They may coalesce to form larger swellings.

Lesions generally first appear on the trunk and neck and spread distally to the limbs and face. They may also appear elsewhere.

Lesions are transient, starting within minutes of the trigger stimulus and resolving within 90 minutes after stopping the initiating activity.

It almost never occurs on the palms, soles, or axillae.

It is commonly associated with both chronic spontaneous urticaria and other inducible urticarias.

Rarely it can be associated with acquired generalised anhidrosis.



Transient wheals induced by exercise and heat typical of cholinergic urticaria



The transient 5 mm wheals of cholinergic urticaria triggered by exercise



Cholinergic urticaria

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What are the complications of cholinergic urticaria?

Rarely, heat bumps may be associated with a systemic response including:

- Hypotension
- Bronchospasm
- [Angioedema](#)
- Headaches
- Wheezing / shortness of breath
- Abdominal cramps and diarrhoea.

Hepatocellular injury, asthma, [anaphylactoid reactions](#), and [anaphylactic reactions](#) have also been reported.

How is cholinergic urticaria diagnosed?

The presentation of typical lesions and symptoms in the context of a typical trigger is often sufficient evidence to form a clinical diagnosis and no further testing is required. Asking the patient to exercise or take a hot bath can allow inspection of the induced lesions.

Traditionally, intradermal methacholine has been used to produce a localised area of hives diagnostic of cholinergic urticaria. However, only one-third of patients demonstrate a positive test, and as a result, the test cannot be used to exclude a diagnosis.

What is the differential diagnosis for cholinergic urticaria?

- [Acute urticaria](#)
- [Chronic urticaria](#)
- [Contact urticaria syndrome](#)
- [Urticular vasculitis](#)
- [Dermographism urticaria](#)
- [Pressure urticaria](#)
- [Solar urticaria](#)

What is the treatment for cholinergic urticaria?

General measures

Identifying and avoiding (where possible) the triggering stimulus is the main treatment consideration in patients with cholinergic urticaria. Rapid cooling of the skin may abort an attack.

Specific measures

Twice the usual dose may be needed.

If cetirizine at twice the normal dose is not effective, a first-generation antihistamine can also be trialled noting that sedation may occur.

For patients with both [cold urticaria](#) and cholinergic urticaria, ketotifen may be helpful; this has sedative properties.

Danazol, an anabolic steroid, can also be beneficial. This is generally reserved for refractory cases due to its side-effect profile.

[Beta-blockers](#) such as propranolol have also been reported as useful pharmacological adjuncts.

[UVB phototherapy](#) may help those with recalcitrant symptoms.

[Omalizumab](#), an anti-IgE monoclonal antibody, is effective for refractory disease given via subcutaneous injection.

How do you prevent cholinergic urticaria?

Diet – avoidance of spicy foods, hot beverages, and alcohol may help reduce flares.

Avoidance of precipitating stimuli that may induce sweating, such as exercise, hot environments, saunas, and immersion in hot water.

What is the outcome for cholinergic urticaria?

Patients with cholinergic urticaria generally have favourable outcomes, with the vast majority of patients reporting alleviation of symptoms after a few years.

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Contact dermatitis

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Overview



Contact dermatitis

[Enlarge image](#)

Contact dermatitis is an itchy rash caused by direct contact with a substance or an allergic reaction to it. The rash isn't contagious, but it can be very uncomfortable.

Many substances can cause this reaction, such as cosmetics, fragrances, jewelry and plants. The rash often shows up within days of exposure.

To treat contact dermatitis successfully, you need to identify and avoid the cause of your reaction. If you avoid the substance causing the reaction, the rash often clears up in 2 to 4 weeks. You can try soothing your skin with a cool, wet cloth and other self-care steps.

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Symptoms



Contact dermatitis on the face

[Enlarge image](#)



Poison ivy blisters

[Enlarge image](#)

Contact dermatitis shows up on skin that has been directly exposed to the substance causing the reaction. For example, the rash may show up along a leg that brushed against poison ivy. The rash can develop within minutes to hours of exposure, and it can last 2 to 4 weeks.

Signs and symptoms of contact dermatitis vary widely and may include:

- An itchy rash
- Leathery patches that are darker than usual (hyperpigmented), typically on brown or Black skin
- Dry, cracked, scaly skin, typically on white skin
- Bumps and blisters, sometimes with oozing and crusting
- Swelling, burning or tenderness

When to see a doctor

See your health care provider if:

- The rash is so itchy that you can't sleep or go about your day
- The rash is severe or widespread
- You're worried about how your rash looks
- The rash doesn't get better within three weeks

- The rash involves the eyes, mouth, face or genitals

Seek immediate medical care in the following situations:

- You think your skin is infected. Clues include fever and pus oozing from blisters.
- It's hard to breathe after inhaling burning weeds.
- Your eyes or nasal passages hurt after inhaling smoke from burning poison ivy.
- You think an ingested substance has damaged the lining of your mouth or digestive tract.

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Causes

Contact dermatitis is caused by exposure to a substance that irritates your skin or triggers an allergic reaction. The substance could be one of thousands.

Feedback

of known allergens and irritants. Often people have irritant and allergic reactions at the same time.

Irritant contact dermatitis is the most common type. This nonallergic skin reaction occurs when an irritant damages your skin's outer protective layer.

Some people react to strong irritants after a single exposure. Others may develop a rash after repeated exposures to even mild irritants, such as soap and water. And some people develop a tolerance to the substance over time.

Common irritants include:

- Solvents
- Rubber gloves
- Bleach and detergents
- Hair products
- Soap
- Airborne substances
- Plants
- Fertilizers and pesticides

Allergic contact dermatitis occurs when a substance to which you're sensitive (allergen) triggers an immune reaction in your skin. It often affects only the area that came into contact with the allergen. But it may be triggered by something that enters your body through foods, flavorings, medicine, or medical or dental procedures (systemic contact dermatitis).

People often become sensitized to allergens after many contacts with it over years. Once you develop an allergy to a substance, even a small amount of it can cause a reaction.

Common allergens include:

- Nickel, which is used in jewelry, buckles and many other items

- Medications, such as antibiotic creams
- Balsam of Peru, which is used in many products, such as perfumes, toothpastes, mouth rinses and flavorings
- Formaldehyde, which is in preservatives, cosmetics and other products
- Personal care products, such as body washes, hair dyes and cosmetics
- Plants such as poison ivy and mango, which contain a highly allergenic substance called urushiol
- Airborne allergens, such as ragweed pollen and spray insecticides
- Products that cause a reaction when you're in the sun (photoallergic contact dermatitis), such as some sunscreens and cosmetics

Children develop allergic contact dermatitis from the usual offenders and also from exposure to diapers, baby wipes, jewelry used in ear piercing, clothing with snaps or dyes, and so on.

Risk factors

The risk of contact dermatitis may be higher in people who have certain jobs and hobbies. Examples include:

- Agricultural workers
- Cleaners
- Construction workers
- Cooks and others who work with food
- Florists
- Hair stylists and cosmetologists
- Health care workers, including dental workers
- Machinists
- Mechanics

- Scuba divers or swimmers, due to the rubber in face masks or goggles
-

Complications

Contact dermatitis can lead to an infection if you repeatedly scratch the affected area, causing it to become wet and oozing. This creates a good place for bacteria or fungi to grow and may cause an infection.

Prevention

You can take the following steps to help prevent contact dermatitis:

- **Avoid irritants and allergens.** Try to identify and avoid the cause of your rash. For ear and body piercings, use jewelry made of hypoallergenic material, such as surgical steel or gold.
- **Wash your skin.** For poison ivy, poison oak or poison sumac, you might be able to remove most of the rash-causing substance if you wash your skin right away after coming into contact with it. Use a mild, fragrance-free soap and warm water. Rinse completely. Also wash any clothing or other items that may have come into contact with a plant allergen, such as poison ivy.
- **Wear protective clothing or gloves.** Face masks, goggles, gloves and other protective items can shield you from irritating substances, including household cleansers.
- **Apply an iron-on patch to cover metal fasteners next to your skin.** This can help you avoid a reaction to jean snaps, for example.
- **Apply a barrier cream or gel.** These products can provide a protective layer for your skin. For example, a nonprescription skin cream containing bentoquatam (Ivy Block) may prevent or lessen your skin's reaction to poison ivy.
- **Use moisturizer.** Regularly applying moisturizing lotions can help restore your skin's outermost layer and keep your skin supple.

- **Take care around pets.** Allergens from plants, such as poison ivy, can cling to pets and then be spread to people. Bathe your pet if you think it got into poison ivy or something similar.

Video: Allergy or irritant: The truth about your rash



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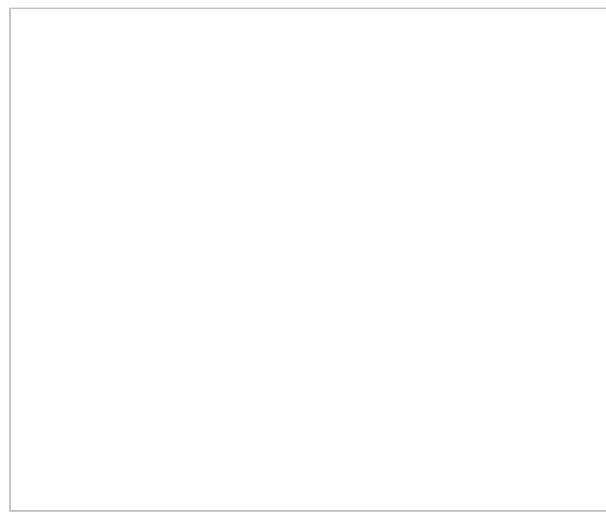
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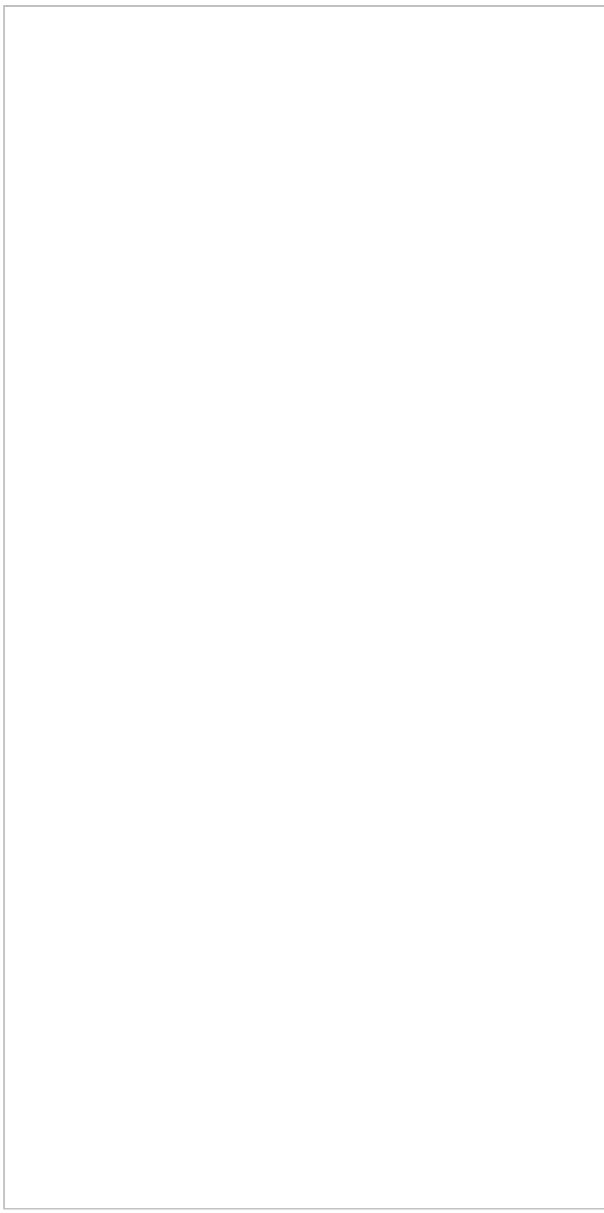
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Contact dermatitis

Author: Dr Amanda Oakley, Dermatologist, Hamilton, New Zealand, 2012.

What is contact dermatitis?

Contact dermatitis (also called contact eczema) refers to a group of skin disorders in which the skin reaction is due to direct contact with the causative agent. The term **dermatitis** implies that the outside layers of skin are affected. It can be acute (a single episode) or chronic (persistent). Dermatitis is nearly always itchy.

Contact dermatitis includes several entities.

[Chemical burns](#)

[Irritant contact dermatitis](#)

[Allergic contact dermatitis](#)

[Contact stomatitis](#) and [contact cheilitis](#)

[Protein contact dermatitis](#)

[Systemic contact dermatitis](#)

Contact dermatitis is sometimes mixed in origin, particularly when it is resulting in [hand dermatitis](#). Contact dermatitis is the most common cause of occupational skin disease, and is particularly common in [cleaners](#), healthcare workers, [food handlers and caterers](#), and [hairdressers](#). It can even occur in people using computers ([computer mouse dermatitis](#)).



Allergic contact dermatitis to rubber



Allergic contact dermatitis to rubber



What does contact dermatitis look like?

The appearance of contact dermatitis is highly variable. It may affect any area of the body and may be any shape (linear, round, polygonal, irregular). Affected skin may have any of the following features.

- Redness (erythema)
- Blisters that are small (vesicles) or large (bullae)
- Swelling (oedema)
- Dryness or scaling
- Cracks (fissuring)
- Lichenification (thickened, lined skin)
- Pigmentation increased (hyperpigmentation) or reduced (hypopigmentation).

Secondary changes may include:

- Scratch marks (excoriation)
- Crusting (due to ooze)
- Pustules (due to bacterial infection).

How is the diagnosis of contact dermatitis made?

Contact dermatitis is usually identified after taking a careful history.

- The rash is eczematous (surface skin changes such as blistering, dryness, peeling and redness)
- The rash has occurred in an area in contact with the putative cause
- The rash has not occurred in sites that are not in contact with this agent
- It may or may not affect all areas of skin in contact with the agent
- Contact dermatitis is usually asymmetrical in distribution, e.g. one hand more severely affected than the other

The various forms of contact dermatitis may appear similar to each other. They may be distinguished by the following features.

Chemical burn occurs after a single exposure to a toxic agent such as a strong acid or alkali. Only the skin in contact with the chemical is affected.

Irritant dermatitis may occur after a single exposure but more commonly follows repetitive exposure to an irritant such as frictional injury, soaps and detergents, excessive immersion in water, mild acids and alkalis, solvents. At least at first, only the skin in contact with the irritant is affected and generalised spread of dermatitis is uncommon. Irritant dermatitis is more common in atopics (often people with a previous history of [atopic eczema](#)), [sensitive skin](#) and other conditions in which skin barrier function is compromised.

Allergic dermatitis usually occurs unexpectedly after the allergen was previously tolerated. Afterwards, contact with even minute amounts of the allergen cause dermatitis on the exposed sites. The dermatitis may spread beyond the borders of direct contact with the allergen, and may spread more widely ([autoeczematisation](#)).

Contact stomatitis and **contact cheilitis** affect the inside of the mouth and lips respectively.

Protein contact dermatitis results from contact with foods (such as meat or potatoes). Immediate [contact urticaria](#) is followed by acute and sometimes chronic dermatitis in the same site.

Systemic contact dermatitis follows ingestion of a substance that has previously caused allergic contact dermatitis. It results in a symmetrical rash, often affecting flexures (e.g. [baboon syndrome](#)), and may generalise. Systemic contact dermatitis is rare.

[Patch tests](#) are important to identify contact allergens in any severe or persistent case of contact dermatitis. An [Open application test](#) may also be recommended.

How is contact dermatitis treated?

Once the causes of contact dermatitis are identified, it is important to avoid direct contact with them. But whatever the cause of the dermatitis, the barrier function of the skin has been damaged and further dermatitis may occur if exposed to irritants.

Avoid [soap](#) – use a pH-balanced cleanser suitable for sensitive skin

For treating and preventing [hand dermatitis](#), wear appropriate [gloves](#) to protect against friction, detergents, soil, plants, paints, diesel etc.

Dry skin carefully after washing

The rash can be treated with a short course of [topical corticosteroid creams](#). Apply [emollients](#) frequently while the rash is active and for some weeks afterwards as the normal skin barrier function is restored.

Severe contact dermatitis may be treated with a short course of [systemic corticosteroids](#), e.g. oral prednisone. Occasionally, for chronic contact dermatitis, [phototherapy](#) may be tried, or immunosuppressive agents such as [methotrexate](#), [cyclosporin](#) or [azathioprine](#) may be prescribed.

Cutaneous dysaesthesia

Author: Dr Eileen J McManus, Advanced Neurology Trainee, Waikato Hospital, Hamilton, New Zealand. Technical Editor: Elaine Mary Luther, Medical Student, Ross University School of Medicine, Barbados. DermNet Editor in Chief: Adjunct A/Prof. Amanda Oakley, Dermatologist, Hamilton, New Zealand. Copy edited by Gus Mitchell. February 2020.

What is cutaneous dysaesthesia?

Cutaneous dysaesthesia is a condition defined as an unpleasant and abnormal sensation in the skin. In the mouth, a similar sensation is called [oral dysaesthesia](#).

It can be classified as generalised or localised. There are multiple variants of localised cutaneous dysaesthesia, which differ in location, duration, and symptom severity.

Who gets cutaneous dysaesthesia?

Anyone can get cutaneous dysaesthesia. There is a possible female predominance in some variants, such as scalp dysaesthesia and [notalgia paraesthetica](#) [1].

What causes cutaneous dysaesthesia?

Generalised cutaneous dysaesthesia is associated with neurological diseases, including:

- Autoimmune disorders, such as acute inflammatory demyelinating polyneuropathy and multiple sclerosis
- Peripheral neuropathies, which may be hereditary, metabolic, or induced by infection or toxin
- Thalamic infarcts
- Alcohol or drug withdrawal
- Endocrine diseases, most commonly [diabetes](#).

Localised dysaesthesia often follows nerve trauma, impingement, or irritation. This can be intracranial (in [trigeminal trophic syndrome](#)), spinal, or peripheral.

Trauma might be iatrogenic (including surgery eg, [autonomic denervation dermatitis](#)) or spontaneous (due osteoarthritis, hyperostosis, a fracture, tumour, arterial ischaemia, or tight clothing) [2].

Infections can also cause nerve injury including [herpes zoster](#), [herpes simplex](#), and [leprosy](#) [1].

Dysaesthesia associated with syringomyelia may be due to impairment of supra-spinal pathways, disinhibition of sympathetic neurons, and aberrant spreading of nociceptive afferent nerves (the fibres that send information to the brain) [3].

Other reported causative associations with cutaneous dysaesthesia are described below.

[Notalgia paraesthetica](#) has been linked with multiple endocrine neoplasia type 2A (MEN2A) caused by RET gene mutations [4].

[Brachioradial pruritus](#) can be exacerbated by sun exposure [5].

[Meralgia paraesthetica](#) has been associated with type 2 [diabetes](#) and obesity [6].

Substance P, a neuropeptide, is postulated to play a key role in trichodynia by promoting mast cell degranulation and neurogenic inflammation in the hair follicle [7].

Psychological stress and generalised anxiety disorder can aggravate symptoms or result in somatisation [8]. See [Psychosocial factors in dermatology](#).

What are the clinical features of cutaneous dysaesthesia?

Dysaesthesia or paraesthesia describes positive cutaneous symptoms such as [pruritus](#), burning, crawling, stinging, hyperaesthesia, allodynia, and pain; and negative symptoms such as anaesthesia or cold sensation. One or more of these symptoms may be present.

The examination of the affected area may be normal or there may be signs of the underlying disease if any, or secondary to rubbing and scratching (if itchy).

Generalised cutaneous dysaesthesia

Generalised cutaneous dysaesthesia presents with dysaesthesia affecting most or all of the skin surface. Symptoms can be exacerbated by temperature change, heat, or the touch of clothing.

Localised cutaneous dysaesthesia

Localised cutaneous dysaesthesia presents with symptoms confined to one area.

Symptoms can be unilateral or bilateral.

The skin may appear normal.

Secondary dermatological changes associated with [pruritus](#) may include excoriations, bruising, hyperpigmentation, and lichenification.

If nerve impingement involves sympathetic pathways as well as sensory nerves, localised dysaesthesia may be accompanied by [hyperhidrosis](#) [3].

Signs of cutaneous dysaesthesia



Lichenification from scratching



Notalgia paraesthetica



Meralgia paraesthesia

How is localised cutaneous dysaesthesia classified?

Localised cutaneous dysaesthesia has been classified in the following conditions.

Brachioradial pruritus affects the skin overlying the brachioradialis muscle of the forearm, that is, on the dorsolateral aspect of the arm around the elbow [5].

Glossodynia or burning mouth syndrome is confined to the oral mucocutaneous membrane and is a form of **oral dysaesthesia** [8].

Genital dysaesthesia may affect the vulva (**dysaesthetic vulvodynia**) or scrotum (**male genital dysaesthesia** and **scrotodynia**) [8]. Perineal dysaesthesia may be accompanied by erythema. See also **Pudendal nerve entrapment syndrome**.

Meralgia paraesthesia affects the anterolateral thigh, the distribution of the lateral femoral cutaneous nerve.

Notalgia paraesthesia affects the skin between the scapula and vertebrae (T2–T6). Forward flexion or extension of the arms may worsen symptoms [9].

Scalp dysaesthesia affects the skin overlying the occipitofrontalis muscle and scalp aponeurosis (C5–C6) [1]. This is sometimes referred to as trichodynia (literally, painful hair) when associated with hair loss or an inflammatory condition of the scalp.

Hand-foot syndrome is a form of cutaneous dysaesthesia affecting hands and feet during chemotherapy

The **trigeminal trophic syndrome** usually affects the ala of the nose (V2 branch of the trigeminal nerve) with subsequent rubbing and picking causing ulceration. The trigeminal trophic syndrome can also involve the buccal mucosa, the tongue, or eye. The tip of the nose (innervated by a branch of VI) is often spared [1].

What are the complications of cutaneous dysaesthesia?

The main complication of cutaneous dysaesthesia is decreased quality of life and impact on mood and mental health.

How is cutaneous dysaesthesia diagnosed?

Generalised cutaneous dysaesthesia

Generalised cutaneous dysaesthesia is a clinical diagnosis after a detailed history and examination have excluded a primary dermatological disease.

Serological tests may include testing for the following:

Glycosylated haemoglobin (HbA1c)

Complement (C3, C4)

- Antinuclear antibody (ANA)
- Antineutrophil cytoplasmic antibodies (ANCA)
- Antibodies to *Borrelia burgdorferi* (found in [Lyme disease](#))
- [Human immunodeficiency virus \(HIV\)](#) and [viral hepatitis](#)
- C-reactive protein (CRP)
- Iron studies
- Folate
- Vitamin B12
- Vitamin E
- Heavy metal levels
- Angiotensin-converting enzyme (ACE).

Other tests may include:

- Nerve conduction studies to look for demyelinating or axonal neuropathy
- Cerebrospinal fluid (CSF) analysis for oligoclonal bands if demyelination is suspected
- Magnetic resonance imaging (MRI) of the brain and cervical spine if demyelination or ischaemia is suspected.

Localised cutaneous dysaesthesia

The diagnosis of localised cutaneous dysaesthesia is based on clinical suspicion. A comprehensive history and examination are needed to identify any underlying cause. For instance, hyperreflexia, weakness, or autonomic dysfunction can indicate a spinal cord pathology.

Other diagnostic tests may include:

- Serology: antinuclear antibody (ANA), antineutrophil cytoplasmic antibodies (ANCA), C-reactive protein (CRP)
- [Skin biopsy](#)
- Imaging: plain X-rays, or MRI of the cervical/thoracic spine for osteophytes, cervical ribs, disc herniation, spinal lesions, or fractures.

What is the differential diagnosis for cutaneous dysaesthesia?

The differential diagnosis of cutaneous dysaesthesia should be broadened if the skin is normal or abnormal (eg, lichenification might be due to [eczema](#), nasal ulceration might be due to [skin cancer](#)).

Somatisation is sometimes implicated with cutaneous sensory disorders such as [compulsive skin picking](#).

What is the treatment for cutaneous dysaesthesia?

Cutaneous dysaesthesia is difficult to treat effectively. Management depends on the cause, body site, and severity of symptoms.

Symptomatic therapy may include:

- [Capsaicin cream](#)
- [Local anaesthetic patches](#)
- Low-dose [tricyclic](#) [1] or another antidepressant [7]
- Antiepileptics including [gabapentin](#), pregabalin, and carbamazepine [10]

Topical amitriptyline 1% with ketamine 0.5% for brachioradial pruritus [10]

Antipsychotic medications such as venlafaxine and pimozide

Physiotherapy.

Additional treatments may include:

Propranolol [11] and possibly [cannabinoids](#) [7] (reported as useful for trichodynia)

Physical barriers to reduce manipulation and ulceration of tissue in trigeminal trophic syndrome, such as [gloves](#), nocturnal thermoplastic facemask, and night-time arm splinting

Transcutaneous electrical muscle stimulation, [narrow-band ultraviolet radiation](#), and [botulinum-A](#) injections for [notalgia paraesthetica](#) [10]

Intralesional steroid injections for [meralgia paraesthesia](#).

What is the outcome for cutaneous dysaesthesia?

Prognosis depends on aetiology, symptom severity, and treatment response. There is no effect on life expectancy.

Dark circles under the eyes

Author: Brian Wu PhD. MD Candidate, Keck School of Medicine, Los Angeles, USA. DermNet Editor in Chief: Dr Amanda Oakley, Dermatologist, Hamilton, New Zealand. July 2015.

What are dark circles under the eyes?

Dark circles under the eyes describe a common appearance of the lower eyelids that has various causes. The dark appearance can be due to:

- [Increased pigmentation](#) (melanin)
- Loss of fatty tissue in the eyelid or around the eye
- Bulging fat and muscle loss
- Puffy eyelids
- Thin, translucent skin
- Shadowing due to anatomic shape of the orbit

The appearance can be challenging to treat.

Who gets dark circles under the eyes?

Those prone to dark circles under the eyes include:

- The elderly (but they are also a common complaint in adolescents)
- People of non-white ethnic background
- People with a genetic predisposition to dark circles under the eyes.

What causes dark circles under the eyes?

Pigmentation under the eyes is associated with dermal deposition of melanin. Dermal melanin deposition is often due to [post-inflammatory pigmentation](#), which may follow:

- Sun exposure
- [Atopic dermatitis](#)
- [Contact dermatitis](#)
- Rubbing or scratching the eyes.

Loss of fatty tissue in the eyelid or around the eye (tear trough) is associated with:

- Ageing
- Genetic factors
- [Smoking](#).

Bulging or puffy eyelids may be due to systemic conditions, particularly:

- [Thyroid disease](#)
- [Dermatitis](#)

Hay fever (allergy).

Thin translucent skin is commonly observed with:

Age

Genetic factors.

Shadowing is more noticeable at times, due to:

Fatigue or lack of sleep

Periorbital oedema (puffy eyelids)

Dehydration (sunken eyes).

Superficially located blood vessels and blood stasis may contribute to the darkened appearance.

How are dark circles under the eyes diagnosed?

Correct diagnosis of dark circles under the eyes can be difficult. It involves:

Personal, medical and family history

Physical examination

[Wood lamp evaluation](#), which allows the clinician to assess the depth of pigmentation.

How are dark circles under the eyes treated?

Treatment of dark circles under the eyes depends on its nature. General measures include:

Adequate sleep

Smoking cessation

Sleep with extra pillows to elevate the head and reduce eyelid swelling

Massage temporary swelling while applying a cold compress

Cold compresses also minimise the appearance of prominent blood vessels

[Cosmetic camouflage](#)

Light-reflecting concealers (these are often yellow or gold in colour) covered by translucent face powder. These should be applied in the shadows, not on the puffy skin.

Unfortunately, many of the remedies on the market lack evidence of efficacy.

Medical treatments to reduce pigmentation can include:

[Protection from sun exposure using sunglasses](#)

Topical agents; however dermal pigmentation responds poorly, and eyelids are sensitive so the stronger products may irritate (see [melasma](#))

[Chemical peels](#) to reduce fine lines and surface pigmentation

[Laser or intense pulsed light \(IPL\)](#) treatments.

[Fillers \(dermal implants\)](#) eg [hyaluronic acid injections](#) or [fat grafts](#)

Surgery to remove excess fat, muscle and skin (surgical blepharoplasty or [laser eye-lifting procedure](#)).

Considerable training and experience are required to optimise results. Improvement may be partial. An incorrect technique may make the dark circles look more prominent than before the procedure.

Dermatitis

Authors: Dr Ian Coulson, Consultant Dermatologist, Burnley, Lancashire, UK. April 2022. Previous author: Dr Amanda Oakley, Dermatologist, New Zealand, 1997. Copy edited by Gus Mitchell.

What is dermatitis?

Dermatitis refers to a group of itchy inflammatory conditions characterised by epidermal changes.

Dermatitis can be classified in a variety of ways. It may be classified by:

Cause eg, [allergic contact dermatitis](#), [photosensitive dermatitis](#)

Clinical appearance eg, [discoid dermatitis](#), [hyperkeratotic dermatitis](#), and [pompholyx](#)

Site of predilection eg, [hand dermatitis](#), [eyelid dermatitis](#), or [lower leg dermatitis](#).

In many cases, various factors may all act as underlying triggers together (allergic, irritant, and endogenous factors, especially in [hand dermatitis](#)).

The terms dermatitis and eczema are often used interchangeably. All eczema is a dermatitis, but not all dermatitis is eczema.

Dermatitis, strictly speaking, includes any cause of skin inflammation affecting the epidermis.

Eczema is derived from the Greek word for “to boil or bubble over”, which pathologically manifests as oedema within the epidermis (called spongiosis).

The term dermatitis is sometimes mistakenly attributed to mean an eczema induced by an occupational factor; this is erroneous.



Acute weepy contact dermatitis due to sticking plaster allergy



Allergic contact dermatitis due to nickel in the sides of her spectacle frame



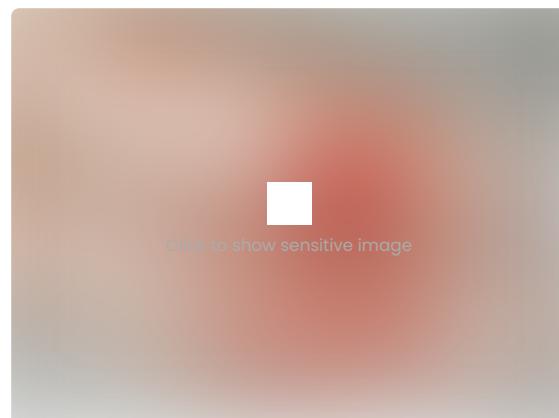
Gravitational eczema compounded by a contact allergy to a bandage constituent



Excoriated acute eczema on the extensor aspects of the knees (reverse pattern)



Fissuring over the knuckles in atopic dorsal hand eczema



Weepy lesions of discoid eczema on the legs



Weepy and impetiginised discoid eczema on the foot



Fissuring, hyperkeratosis and vesiculation in chronic fingertip dermatitis



Discoid pattern of eczema on the dorsal hands



Hand eczema on the back of the hand – mixture of irritant factors in this atopic person



Hypopigmentation and fine scale on the cheeks in pityriasis alba



Cheek and nasolabial fold redness and scaling in seborrhoeic dermatitis

Who gets dermatitis?

Dermatitis is common, affecting about one in every five persons at some stage in their life.

Different types of dermatitis are more frequent at different stages of life, for example:

[Atopic dermatitis](#) and [pityriasis alba](#) are more common in children

[Hand eczema](#) is more common in young and middle-aged adults

[Venous or gravitational eczema](#), [asteatotic dermatitis](#), and [nummular eczema](#) are more common in middle and older age groups.

There are no consistent racial factors influencing disease frequency.

What are the clinical features of dermatitis?

Dermatitis may be either acute or chronic, and although the mechanism by which the dermatitis develops may be the same, the appearances may be starkly different.

Acute dermatitis will show redness or swelling, papulation, vesiculation, oozing and weeping, and even blistering.

Chronic eczema will show skin thickening with accentuation of the skin creases, hyperkeratosis, scaling, fissuring, excoriation, and hyperpigmentation.

Subacute dermatitis will show features of both.

How do the clinical features vary in different racial groups?

Redness may be more difficult to appreciate in darker skin types.

[Post-inflammatory hypo- and hyperpigmentation](#) are more frequent in darker skin types.

What are the types of dermatitis?

Exogenous dermatitis is the result of an external factor or insult that induces skin inflammation.

Common causes include:

Allergic contact dermatitis – due to immune sensitisation of an individual to an allergen, often at even low concentration, such as nickel, hair dye, rubber, or perfumes; identified by patch testing.

Irritant contact dermatitis – will occur in anyone exposed to an irritant at sufficient concentration for long enough; irritants include soaps, detergents, organic solvents, degreasing agents, abrasives, desiccants, dust, urine, and even water

Photosensitive dermatitis – triggered by light or UV radiation

Post-traumatic dermatitis – due to physical injuries such as abrasions, burns, or surgery (eg, **autonomic denervation dermatitis**)

Dermatitis induced by local skin infections such as bacterial, fungal, and viral e.g. molluscum contagiosum and HTLV-1 disease

Drug-induced dermatitis.

Endogenous dermatitis occurs because of often ill-understood internal factors. Common types include:

Atopic dermatitis – a common form of dermatitis occurring in children and adults, and often occurring in families with a background of asthma and hay fever

Seborrhoeic dermatitis – common chronic eczema affecting the face, scalp, ears and major flexures, due to a reaction to yeasts that colonise the skin

Discoid (nummular) dermatitis – coin-shaped patches of dermatitis usually affecting the limbs

Lichen simplex – chronic dermatitis that thickens due to perpetual scratching

Pityriasis alba – pale patches of dermatitis affecting the cheeks

Hand dermatitis – internal, external irritants and allergic factors may all play a part even in a single individual

Eyelid dermatitis – again, often of mixed cause

Otitis externa – dermatitis affecting the ear canal and the pinna

Venous or gravitational dermatitis – dermatitis due to malfunction of the lower leg vein valves

Juvenile plantar dermatitis – a glazed and fissured forefoot eczema occurring in children

Metabolic dermatitis – seen in some nutritional and endocrine disorders

Chronic superficial scaly dermatitis – finger-shaped patches of eczema occurring on the trunk

Asteatotic dermatitis – crazy-paving shaped dermatitis due to degreasing of the skin from excessive bathing and soap use on the legs in the elderly

Halo dermatitis or Meyerson naevus – this can surround a benign mole

Erythrodermic dermatitis – severe dermatitis when more than 80% of the skin is affected.

What is the differential diagnosis of dermatitis?

Bacterial infections – **impetigo**, **erythrasma**

Fungal infections

Connective tissue diseases – **lupus erythematosus** and **dermatomyositis**

Rosacea

Blistering diseases – **bullous pemphigoid**

Skin tumours – **Bowen disease**, **superficial basal cell carcinomas**

Cutaneous T-cell lymphoma.

How is dermatitis investigated?

A detailed history and examination may be all that is required to make an accurate diagnosis.

The following investigations may sometimes be needed:

Skin scraping to exclude a fungal infection mimicking a dermatitis

Skin swab looking for bacterial or viral superadded infection

Patch testing to identify contact allergens

Light testing if a photosensitive dermatitis is considered

Skin biopsy to exclude mimics of dermatitis

Blood tests – IgE (usually elevated in atopic dermatitis), thyroid function (in some hand dermatitis and asteatotic dermatitis).

How is dermatitis treated?

General principles are covered here. Specific management of specific types of dermatitis are detailed on the relevant pages.

Potential allergen identification and avoidance – made on the basis of history e.g. hobbies, products used, and occupation. A patch test will confirm.

Potential irritant identification and avoidance – avoid soaps, shower gels, dust, organic solvents, and drying/desiccating agents.

Protect the skin with [personal protective equipment](#) – especially hand dermatitis, by the use of cotton gloves for dry work, and cotton with an occlusive glove appropriate to the suspected allergen or irritant.

Topical therapies

[Emollients](#) – both in place of soap, after bathing or washing, and at any time if the skin feels dry.

[Potassium permanganate soaks](#) – useful for drying up weepy exudative or blistering acute eczema.

[Paste bandages](#) – useful to help topical steroids penetrate the skin, soothe, and reduce skin trauma from scratching.

[Topical steroids](#) – generally use an ointment if the skin is dry, and a cream if it is wet and weepy.

Most work just as well if applied only once daily.

Help reduce skin inflammation that causes the eczema, and should be applied where the skin is inflamed (red and itchy).

Potent products are often used for 7–14 days, then the frequency of application is reduced to alternate days, then twice weekly, and the potency of the steroid reduced.

Twice weekly steroid treatment is often recommended to prevent disease relapse, and prevent flare-ups for extended periods.

Topical anti-inflammatory agents

Calcineurin inhibitors such as [pimecrolimus](#) and [tacrolimus](#) suppress eczema and do not have the long-term side effects of potent steroids, particularly for the face.

Newer small molecules such as [JAK inhibitors \(ruxolitinib\)](#) are either approved or being developed for the treatment of dermatitis.

Physical therapies

[Ultraviolet B and Psoralens UVA \(PUVA\)](#) may be valuable for recalcitrant atopic and discoid dermatitis.

Systemic agents

[Antihistamines](#) – to suppress the itch of eczema, a sedating antihistamine, rather than a non-sedating agent is generally needed.

Antibiotics and antivirals – should be considered if the eczema is super-infected with bacteria (*Staphylococcus*) and [herpes simplex](#).

Immunosuppressive therapies – less than 2% of chronic eczema sufferers will fail to be adequately controlled with the above therapies. Agents that reduce the overactive immune response seen in dermatitis may help. [Methotrexate](#), [azathioprine](#), and [cyclosporin](#) are the agents usually considered.

[Biological therapies](#) – antibody treatments that specifically block the key mediators of inflammation in dermatitis (cytokines) are in use and in active development for severe dermatitis. These injection treatments include [dupilumab](#), [tralokinumab](#), [lebrikizumab](#), and [nemolizumab](#).

[Oral small molecules](#) – [baricitinib](#), [upadacitinib](#), and [abrocitinib](#) either are licensed or are being considered for licence and use in moderate/severe atopic dermatitis in many countries. These

agents block the JAK/STAT pathways that in turn regulate cytokine production.

Eczema: A Complete Overview



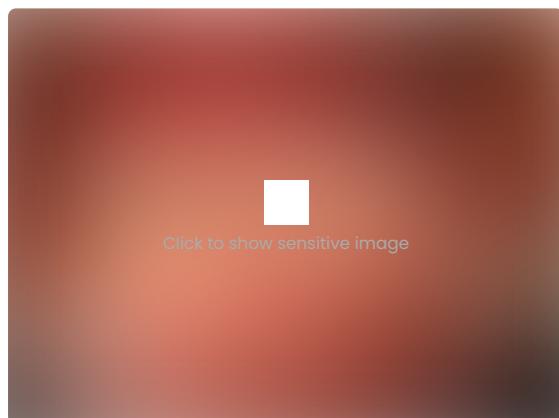
Skin problems associated with diabetes mellitus

Author: Hon A/Prof Amanda Oakley, Dermatologist, Hamilton, New Zealand, 1998. Updated by Dr Ebtisam Elghblawi, Dermatologist, Tripoli, Libya. DermNet Editor in Chief: A/Prof Amanda Oakley, Dermatologist, Hamilton, New Zealand. August 2019.

Introduction

It is estimated that 30% of patients with diabetes mellitus will experience a skin problem at some stage throughout the course of their disease. Several skin disorders are more common in diabetic patients, particularly those due to infection such as [candida](#) and [impetigo](#). Patients with type 2 diabetes also have twice the risk of developing the common scaly disease, [psoriasis](#), as non-diabetics.

Skin conditions associated with diabetes mellitus



Impetigo



Candida intertrigo



Psoriasis

Specific skin conditions associated with diabetes mellitus are described below.

[Diabetic dermopathy](#)

[Diabetic bullae](#)

[Diabetic stiff skin](#)

What is diabetes mellitus?

Diabetes mellitus constitutes a collection of diverse disorders associated with an increase in blood glucose concentration.

Diabetes is associated with impaired carbohydrate, protein, and fat metabolism due to insufficient secretion of insulin or target-tissue [insulin resistance](#). Complications of diabetes mellitus comprise both macrovascular (cardiovascular) and microvascular (retinopathy, nephropathy, or neuropathy) sequelae.

Type 1 diabetes mellitus is characterised by absolute insulin absence and is due to autoimmune beta-cell destruction. It typically presents with acute symptoms or ketoacidosis in childhood or adolescence, and lifelong insulin therapy is mandatory.

Type 2 diabetes mellitus is a common disorder categorised by [insulin resistance](#) and relative insulin deficiency. Patients are often asymptomatic and are diagnosed through screening. Strong risk factors include older age, obesity, physical inactivity, prior gestational diabetes, pre-diabetes, non-white ancestry, family history of diabetes, and [polycystic ovary syndrome](#). Modification of cardiovascular risk factors (eg, hypertension and dyslipidaemia) are an important part of treatment, along with glycaemic control to prevent microvascular complications.

Type 2 diabetes is the main cause of type 2 diabetes in children, who are usually over 10 years of age. [Acanthosis nigricans](#) accompanies childhood diabetes in 90–95%.

Gestational diabetes is diagnosed if glucose intolerance is first recognised during pregnancy at 24–28 weeks of gestation. Strong risk factors include advanced maternal age (more than 40 years), obesity, personal history of gestational diabetes or macrosomia affecting a previous child, [polycystic ovary syndrome](#), non-white ancestry, and a family history of diabetes mellitus.

Other presentations of diabetes mellitus include diabetic ketoacidosis, hyperosmolar hyperglycaemic state, diabetic cardiovascular disease, diabetic kidney disease, diabetic neuropathy, [diabetic foot](#), diabetic retinopathy, and [metabolic syndrome](#).

The increasing prevalence of diabetes requires targeted screening for detecting diabetes and prediabetes in risk groups to prevent and mitigate the progression of the disease.

Diabetic dermopathy

Diabetic dermopathy is a skin condition characterised by light brown or reddish, oval or round, slightly indented scaly patches most often appearing on the shins. Although these lesions may appear in anyone, particularly after an injury or trauma to the area, they are one of the most common skin problems found in patients with diabetes mellitus. Diabetic dermopathy has been found to occur in up to 30% of patients with diabetes.

Diabetic dermopathy is sometimes also referred to as shin spots and pigmented pretibial patches. They resemble [solar lentigines](#).



Diabetic dermopathy



Diabetic dermopathy



Diabetic dermopathy

What causes diabetic dermopathy?

The exact cause of diabetic dermopathy is unknown but may be associated with diabetic neuropathic and vascular complications, as studies have shown the condition to occur more frequently in diabetic patients with retinopathy, neuropathy and nephropathy.

Diabetic dermopathy tends to occur in older patients or those who have had diabetes for at least 10–20 years. It also appears to be closely linked to increased glycosylated haemoglobin, an indicator of poor control of blood glucose levels.

Because lesions often occur over bony parts of the body such as the shins, it is thought that diabetic dermopathy may also be a magnified response to injury or trauma to these areas. Studies have shown that shin spots have appeared in response to trauma with heat, cold or blunt objects in patients with diabetes.

What are the signs and symptoms?

Diabetic dermopathy lesions appear most frequently on the shins. Less commonly lesions can be found on the front of the thighs, forearm, side of the foot, scalp and trunk. Features of lesions are:

- Round or oval-shaped
- Reddish-brown colour
- Initially scaly but then flattens out and becomes indented
- Commonly occur on both shins.

The presence of four or more lesions is almost always limited to patients with diabetes. People presenting with shin spots not already diagnosed with diabetes should undergo a further investigation to rule out the possibility of early diabetes.

What is the treatment for diabetic dermopathy?

Diabetic dermopathy lesions or shin spots are harmless. They usually do not require any treatment and tend to go away after a few years, particularly following improved blood glucose control.

Diabetic bullae

Diabetic bullae, also known as bullosis diabetorum, are blister-like lesions that occur spontaneously on the feet and hands of diabetic patients. Although rare, diabetic bullae are a distinct marker for diabetes.

Diabetic bullae are more common in men than women

They are prevalent between the ages of 17 and 84 years.

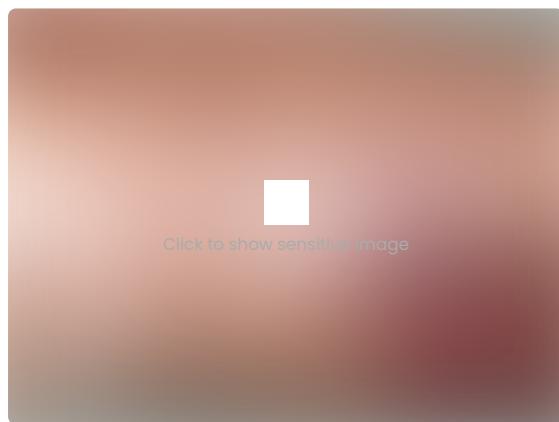
They are also more common in patients who have long-standing diabetes or multiple diabetic complications, particularly neuropathy.

The blisters are painless and can be from 0.5–17 centimetres in size. They often have an irregular shape. Two types of diabetic bullae have been defined.

Intraepidermal bullae – these are blisters filled with clear, sterile viscous fluid and normally heal spontaneously within 2–5 weeks without scarring and atrophy.

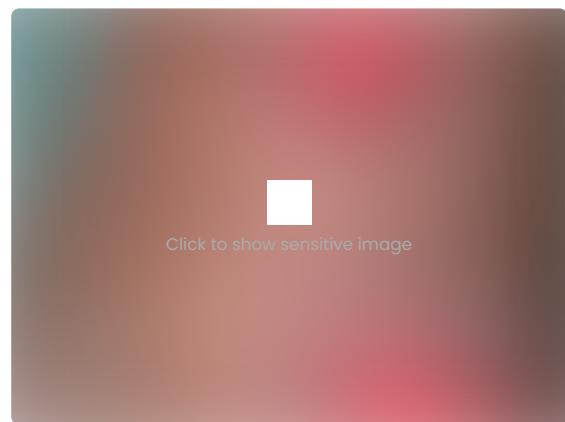
Subepidermal bullae – these are less common and may be filled with blood. Healed blisters may show scarring and atrophy.

In most cases, diabetic bullae heal spontaneously without treatment. Patients should make sure the blister remains unbroken to avoid secondary infection.



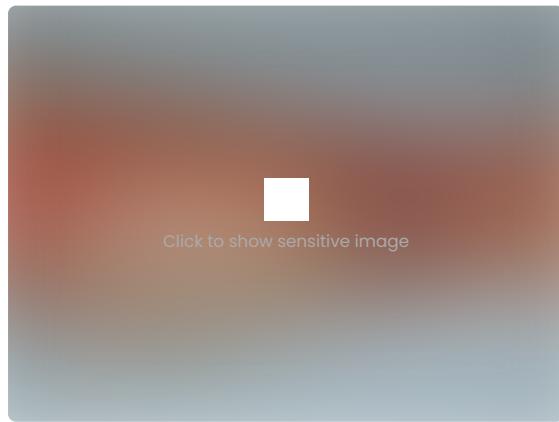
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Diabetic bullae



Click to show sensitive image

Diabetic bullae



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Diabetic bullae

Diabetic stiff skin

Many patients with longstanding type 1 diabetes develop diabetic cheiroarthropathy or diabetic stiff skin (digital sclerosis). This results in restricted mobility of the joints of their hands and stiff, waxy, thickened and yellowed skin. This is thought to be due to the reaction of glucose with proteins in the skin and increased glycation end products. These patients may also suffer from [Dupuytren contracture](#) (tendon tightening, which bends the fingers).



Diabetic stiff skin



Diabetic stiff skin



Diabetic stiff skin

Other dermatological conditions associated with diabetes

Other common conditions in diabetics are foot ulcers and [necrobiosis lipoidica](#).

Diabetics with renal failure are also prone to [reactive perforating collagenosis](#) and [Kyrle disease](#).

[Scleroedema](#) – a rare complication of type 2 diabetes causing skin thickening of the neck and upper back

Disseminated [granuloma annulare](#)

Eruptive [xanthoma](#) on the hands, arms, feet, legs, and buttocks associated with high levels of cholesterol and triglycerides



Scleredema



Generalised granuloma annulare



Eruptive xanthoma

Xanthelasma – multiple yellowish scaly patches on and around the eyelids

[Skin tags](#)

[Vitiligo](#) – an autoimmune skin problem sometimes associated with type 1 diabetes



Xanthelasma



Skin tag



Vitiligo

Te Whatu Ora

Health New Zealand

Acanthosis nigricans — darkening and thickening of skin folds, thought to be due to insulin resistance

Necrobiosis lipoidica — yellow, waxy plaques on the shin

Pruritus — this can have many causes, such as a yeast infection, dry skin, neuropathy or poor blood flow

Cutaneous dysaesthesia — due to small fibre neuropathy



Acanthosis nigricans



Necrobiosis lipoidica



Excoriations due to pruritus

Bacterial skin infections — including **stye**, **boil**, **abscess**, **paronychia**, **cellulitis**

Fungal infections — particularly *Candida albicans*



Boil



Cellulitis



Candida intertrigo

Dry skin

Author: Hon A/Prof Amanda Oakley, Dermatologist, Hamilton, New Zealand, 1997. Update: March 2022.

What is dry skin?

Dry skin refers to skin that feels dry to touch. This occurs when the skin is lacking moisture in the outer horny cell layer (stratum corneum) and this results in cracks in the skin surface.

Dry skin is also called xerosis, xeroderma or asterosis (lack of fat).

Who gets dry skin?

Both males and females of all ages can be affected. There is some racial variability in water and lipid content of the skin.

Dry skin that starts in early childhood may be one of about 20 types of [ichthyosis](#) (fish-scale skin). There is often a family history of dry skin.

Dryness is commonly seen in people with [atopic dermatitis](#).

Nearly everyone > 60 years has dry skin.

Dry skin that begins later may be seen in people with certain diseases and conditions, such as:

[Postmenopausal females](#)

[Hypothyroidism](#)

Chronic renal disease

[Malnutrition](#) and weight loss

Subclinical [dermatitis](#)

Treatment with certain drugs such as [oral retinoids](#), diuretics and [epidermal growth factor receptor inhibitors](#).

People exposed to a dry environment may experience dry skin, for example:

Low humidity: in desert climates or cool, windy conditions

Excessive air conditioning

Direct heat from a fire or fan heater

Excessive bathing

Contact with [soap](#), laundry detergents and solvents

Inappropriate topical agents such as [alcohol](#)

Frictional irritation from rough clothing or abrasives.

What causes dry skin?

Dry skin is due to abnormalities in the integrity of the [barrier function](#) of the stratum corneum, which is made up of corneocytes.

There is an overall reduction in the lipids in the stratum corneum.

The ratio of ceramides, cholesterol and free fatty acids may be normal or altered.

There may be a reduction in the proliferation of keratinocytes.

Keratinocyte subtypes change in affected skin with a decrease in keratins K1, K10 and increase in K5, K14.

Involucrin (a protein) may be expressed early, increasing cell stiffness.

The result is the retention of corneocytes and reduced water-holding capacity.

The inherited forms of [ichthyosis](#) are due to loss of function mutations in various genes (listed in parentheses below):

[Ichthyosis vulgaris](#) (FLG)

[Recessive X-linked ichthyosis](#) (STS)

Autosomal recessive congenital ichthyosis (ABCA12, TGMI, ALOXE3)

Keratinopathic ichthyoses (KRT1, KRT10, KRT2).

Acquired ichthyosis may be due to:

Metabolic factors: [thyroid](#) deficiency

Illness: lymphoma, internal malignancy, [sarcoidosis](#), [HIV infection](#)

Drugs: nicotinic acid, [kava](#), protein kinase inhibitors (eg [EGFR inhibitors](#)), [hydroxyurea](#).

What are the clinical features of dry skin?

Dry skin has a dull surface with a rough, scaly quality. The skin is less pliable and cracked. When dryness is severe, the skin may become inflamed and fissured.

Although any site can be dry, affected skin tends to involve the shins more than any other site.

The clinical features of [ichthyosis](#) depend on the specific type of ichthyosis.



Ichthyosis



Close-up of ichthyosis



Dermatitis from dry skin

[See more images of dry skin](#)

Complications of dry skin

Dry areas of skin may become itchy, indicating a form of [eczema/dermatitis](#) has developed, such as:

[Atopic eczema](#) – especially in people with [ichthyosis vulgaris](#)

[Eczema craquelé](#) – especially in older people. Also called asteatotic eczema

A dry form of [nummular dermatitis/discoid eczema](#) – especially in people that wash their skin excessively.

When the skin of an older person is dry and itchy without a visible rash, it is sometimes called [winter itch](#), 7th age itch, senile pruritus or chronic pruritus of the elderly.

Other complications may include:

[Flaky skin or itchy skin \(pruritus\)](#)

[Skin infection](#) when bacteria or viruses penetrate a break in the skin surface

Overheating, especially in some forms of [ichthyosis](#)

[Food allergy](#), eg, to peanuts, has been associated with filaggrin mutations

[Contact allergy](#), eg, to [nickel](#), has also been correlated with [barrier function](#) defects.

How is the type of dry skin diagnosed?

The type of dry skin is diagnosed by careful history and examination.

In children:

Family history

Age of onset

Appearance at birth, if known

Distribution of dry skin

Other features, eg eczema, abnormal nails, hair, dentition, sight, hearing.

In adults:

Medical history

Medications and topical preparations

Bathing frequency and use of soap

Evaluation of environmental factors that may contribute to dry skin.

Sometimes a [skin biopsy](#) may be requested. There may be additional tests requested to diagnose some types of [ichthyosis](#).

What is the treatment for dry skin?

The mainstay of treatment of dry skin and [ichthyosis](#) is [moisturisers/emollients](#). They should be applied liberally and often enough to:

Reduce [itch](#)

Improve the [barrier function](#)

Prevent entry of irritants, bacteria

Reduce transepidermal water loss.

When considering which emollient is most suitable, consider:

- Severity of the dryness
- Tolerance
- Personal preference
- Cost and availability.

Emollients generally work best if applied to damp skin, if pH is below 7 (acidic), and if containing humectants such as urea or propylene glycol.

Additional treatments include:

- [Topical steroid](#) if itchy or there is dermatitis – choose an emollient base
- [Topical calcineurin inhibitors](#) if topical steroids are unsuitable.

How can dry skin be prevented?

- Eliminate aggravating factors.
- Reduce the frequency of bathing.
- A humidifier in winter and air conditioner in summer.
- Compare having a short shower with a prolonged soak in a hot bath.
- Ensure good hydration by drinking plenty of water.
- Use lukewarm water, not hot water.
- Replace standard soap with a substitute such as a synthetic detergent [cleanser](#), water-miscible emollient, bath oil, anti-pruritic tar oil, colloidal [oatmeal](#) etc.
- Apply an [emollient](#) liberally and often, particularly shortly after bathing, and when itchy. The drier the skin, the thicker this should be, especially on the hands.

What is the outlook for dry skin?

A tendency to dry skin may persist life-long, or it may improve once contributing factors are controlled.

[See more images of dry skin](#)

Eczema herpeticum

Author: Dr Chin-Yun Lin, Dermatology Registrar, Auckland Hospital, New Zealand, 2010. DermNet update May 2023.

What is eczema herpeticum?

Eczema herpeticum is a disseminated viral infection characterised by fever and clusters of itchy blisters or punched-out erosions. It is most often seen as a complication of [atopic dermatitis/eczema](#).

Eczema herpeticum is also known as Kaposi varicelliform eruption because it was initially described by Kaposi in 1887, who thought it resembled [chickenpox/varicella](#).

What is the cause of eczema herpeticum?

Most cases of eczema herpeticum are due to *Herpes simplex* type 1 or 2.

Eczema herpeticum usually arises during a first episode of infection with [Herpes simplex](#) (primary herpes). Signs appear 5–12 days after contact with an infected individual, who may or may not have visible cold sores.

Eczema herpeticum may also complicate recurrent herpes. However, repeated episodes of eczema herpeticum are unusual.

Eczema herpeticum can affect males and females of all ages but is more commonly seen in infants and children with [atopic dermatitis](#). Patients with atopic dermatitis appear to have reduced immunity to herpes infection. Their underlying dermatitis can be mild to severe, active or inactive.

Eczema herpeticum is better called Kaposi varicelliform eruption when a breakdown of the skin barrier is not due to eczema. Examples of non-eczematous conditions prone to severe localised herpes infections are:

- [Thermal burns](#)
- [Pemphigus vulgaris](#)
- [Darier disease](#)
- [Benign familial pemphigus](#)
- [Cutaneous T-cell lymphoma/mycosis fungoides](#)
- [Ichthyosis](#).

Other viruses may occasionally be responsible for a similar eruption, such as [eczema coxsackium](#) due to coxsackievirus A16 (the cause of [hand foot and mouth disease](#)).

As [smallpox](#) has been eliminated, disseminated vaccinia as a consequence of smallpox vaccination is now very rare. It was reported to be very severe, with mortality of up to 50%.

What are the clinical features of eczema herpeticum?

Eczema herpeticum starts with clusters of itchy and painful blisters. It may affect any site but is most often seen on face and neck. Blisters can occur in normal skin or sites actively or previously affected by atopic dermatitis or another skin disease. New patches form and spread over 7–10 days and may rarely be widely disseminated throughout the body.

The patient is unwell, with fever and swollen local lymph nodes.

The blisters are monomorphic, that is, they all appear similar to each other.

They may be filled with clear yellow fluid or thick purulent material.

They are often blood-stained i.e., red, purple or black.

New blisters have central dimples (umbilication).

They may weep or bleed.

Older blisters crust over and form sores (erosions)

Lesions heal over 2–6 weeks.

In severe cases where the skin has been destroyed by infection, small white scars may persist long term.

Secondary bacterial infection with **staphylococci** or **streptococci** may lead to **impetigo** and **cellulitis**.

Severe eczema herpeticum may affect multiple organs, including the eyes, brain, lung, and liver. It can rarely be fatal.



Eczema herpeticum



Eczema herpeticum



Eczema herpeticum

[See more images of eczema herpeticum ...](#)

How is eczema herpeticum diagnosed?

Eczema herpeticum can be diagnosed clinically when a patient with known atopic dermatitis presents with an acute eruption of painful, monomorphic clustered vesicles associated with fever and malaise. Viral infection can be confirmed by viral swabs taken by scraping the base of a fresh blister. Several [laboratory tests](#) are available.

Viral culture

Direct fluorescent antibody stain

PCR (Polymerase Chain Reaction) sequencing

[Tzank smear](#) showing epithelial multinucleated giant cells and acantholysis (cell separation)

Bacterial swabs should also be taken for microscopy and culture as eczema herpeticum may resemble [impetigo](#) and it can be complicated by [secondary bacterial infection](#).

[Skin biopsy](#) reveals distinctive [pathological changes](#).

What is the treatment of eczema herpeticum?

Eczema herpeticum is considered as one of the few dermatological emergencies. Prompt treatment with antiviral medication should eliminate the need for hospital admission.

Oral [aciclovir](#) 400–800 mg 5 times daily, or, if available, valaciclovir 1 g twice daily, for 10–14 days or until lesions heal. Intravenous aciclovir is prescribed if the patient is too sick to take tablets, or if the infection is deteriorating despite treatment.

Secondary bacterial skin infection is treated with systemic [antibiotics](#).

[Topical steroids](#) previously have not been recommended but recent evidence suggests that they are safe to use, and may be necessary to treat active atopic dermatitis.

Consult an ophthalmologist when eyelid or eye involvement is seen or suspected.



Public / Diseases & conditions / Eczema / Types & treatments / **Atopic dermatitis**

ECZEMA TYPES: ATOPIC DERMATITIS OVERVIEW

Overview Symptoms Causes Treatment Skin care Can anything relieve severe atopic dermatitis?



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The American Academy of Dermatology gratefully acknowledges the support from Sanofi and Regeneron.

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What is atopic dermatitis?

Atopic dermatitis

Also called eczema, this is a common condition that causes itchy, dry, and inflamed skin. It usually begins in childhood but can start at any age. Board-certified dermatologists have expertise in helping their patients reduce flare-ups and feel more comfortable.

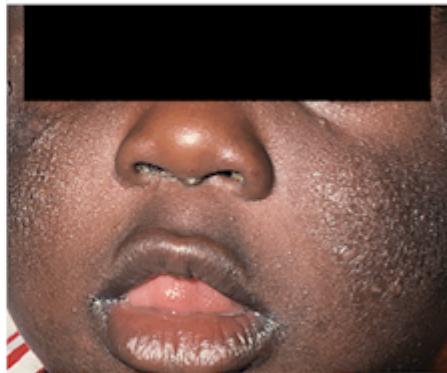
Is atopic dermatitis contagious? No. This condition cannot spread from person to person.

Babies often develop atopic dermatitis on their face

If your baby has a lighter skin tone, you may see a pink or red rash with bumps and swollen skin that may ooze or crust (A). Babies with darker skin tones are more likely to develop small bumps, extremely dry skin, and dark brown, gray, or purple skin where they have atopic dermatitis (B).



A



B

Atopic dermatitis, also known as eczema, usually begins early in life. It often appears between 2 months and 5 years of age. While most people develop atopic dermatitis by 5 years of age, this condition can also start during puberty or later.

What's the difference between atopic dermatitis and eczema?

Eczema refers to a group of conditions that cause inflamed skin. Signs of inflamed skin include a rash, itchiness, and excessive dryness. There are several types of eczema. Atopic dermatitis is the most common type. Other types of eczema include [contact dermatitis](#) and [stasis dermatitis](#).

When talking about atopic dermatitis, many people say “eczema” or “atopic eczema.” They usually call other types of eczema by their specific name. If you’re uncertain when you hear the word “eczema” in a health care setting, ask the person what type of eczema they are talking about.

How long does atopic dermatitis last?

It's a chronic disease, which means it can last a long time. For many children, the condition goes away by their teenage years. However, some people have the condition for life.

There is no way to know whether atopic dermatitis will go away or become a lifelong condition. However, you can prevent it from getting worse by seeing a board-certified dermatologist as soon as you notice signs and symptoms. An early diagnosis and proper treatment can prevent the condition from worsening. The more severe the condition becomes, the more:

- Difficult it can be to treat
- Likely a person will have atopic dermatitis as an adult

Even if atopic dermatitis goes away, the skin can still be easily irritated. For example, many people who had atopic dermatitis as a child may have problems doing wet work, which requires you to have wet hands frequently throughout the day. Jobs that involve wet work include working as a nurse, florist, hairstylist, or bricklayer.

When people who had (or have) atopic dermatitis do wet work, they often develop raw, cracked, and irritated skin that may bleed. Your dermatologist may be able to help you manage atopic dermatitis so that you can continue to do a job or hobby that involves wet work. However, sometimes, the best way to manage atopic dermatitis is to stop doing wet work.

Does atopic dermatitis worsen with age?

This condition can worsen. That's why getting proper treatment soon after you notice signs and symptoms is so important. A treatment plan that's customized to meet your needs can help prevent atopic dermatitis from worsening.

To benefit from this treatment plan, it's essential that you stick to it. Follow the recommended skin care, use medication as instructed, and find out what triggers your flare-ups so that you can avoid known triggers.

What can prevent a child from getting atopic dermatitis?

Dermatologists and other researchers are trying to answer this question. It's an important part of atopic dermatitis research because so many children develop this condition.

So far, nothing is guaranteed to stop this condition from developing. However, the results from one study are promising. All the babies in this study had a high risk of developing atopic dermatitis. During the study, one group of parents followed an eczema friendly skin care routine, which included applying moisturizer to their newborn's skin every day.

The results from this study showed that the babies who received eczema friendly skin care, which included applying an eczema friendly moisturizer, were less likely to develop atopic dermatitis than the babies who didn't receive this care.

While more research is needed, the results from this study are encouraging.

Is there a cure for atopic dermatitis?

At this time, atopic dermatitis cannot be cured. However, treatment can ease symptoms and lead to clearer — if not completely clear — skin.

Today, newer medications are easing symptoms for patients who haven't been helped by previous treatments, and researchers continue to look for better ways to treat this condition. Some of these researchers are dermatologists. The goal of this research is to develop increasingly safe and effective treatments.

Should people with atopic dermatitis keep their skin dry or moist?

You want to keep skin moisturized. Atopic dermatitis causes excessively dry skin. Keeping the skin hydrated with an eczema friendly cream or ointment can help relieve discomfort and reduce flare-ups. That's why dermatologists recommend that everyone with this condition keep their skin moisturized with a mild, fragrance-free cream or ointment.

To find out how to keep skin moisturized, follow the three steps from dermatologists that tell you [How to reduce eczema flares with moisturizer](#).

Where does atopic dermatitis appear on the body?

It can occur anywhere on the skin or scalp. However, there are certain places where this condition is more likely to develop. To find out where and see pictures of atopic dermatitis, go to [Atopic dermatitis: Symptoms](#).

Images

- Image 1: Getty Images
- Image 2: Used with permission of [DermNet NZ](#).

References

- Feldman SR, Cox LS, *et al*. "The challenge of managing atopic dermatitis in the United States." *Am Health Drug Benefits*. 2019 Apr;12(2):83-93.
- Lee HH, Patel KR, *et al*. "A systematic review and meta-analysis of the prevalence and phenotype of adult-onset atopic dermatitis." *J Am Acad Dermatol*. 2019 Jun;80(6):1526-1532.e7.
- Simpson EL, Chalmers JR, *et al*. "Emollient enhancement of the skin barrier from birth offers effective atopic dermatitis prevention." *J Allergy Clin Immunol*. 2014 Oct;134(4):818-23.
- Simpson EL, Leung DYM, *et al*. "Atopic dermatitis." In: Kang S, *et al*. *Fitzpatrick's Dermatology*. (ninth edition) McGraw Hill Education, United States of America, 2019:363-84.
- Sidbury R, Tom WL, *et al*. "Guidelines of care for the management of atopic dermatitis: Section 4. Prevention of disease flares and use of adjunctive therapies and approaches." *J Am Acad Dermatol*. 2014 Dec;71(6):1218-33.
- Ständer S. "Atopic dermatitis." *N Engl J Med*. 2021 Mar 25;384(12):1136-43.

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Last updated: 10/10/23

Female pattern hair loss

Author: Dr Leona Yip, Research Fellow, St Vincent's Hospital, Melbourne, Australia, 2007. Updated by Prof Rod Sinclair, Melbourne, Australia, July 2015.

Images courtesy of R. Sinclair, FACD

What is female pattern hair loss?

Female pattern hair loss (FPHL) is a distinctive form of [diffuse hair loss](#) that occurs in women with androgenetic alopecia. Many women are affected by FPHL. Around 40% of women by age 50 show signs of hair loss and less than 45% of women reach the age of 80 with a full head of hair.

In FPHL, there is diffuse thinning of hair on the scalp due to increased hair shedding or a reduction in hair volume, or both. It is normal to lose up to 50–100 hairs a day. Another condition called chronic [telogen effluvium](#) also presents with increased hair shedding and is often confused with FPHL. It is important to differentiate between these conditions as management for both conditions differ.

FPHL presents quite differently from the more easily recognisable [male pattern baldness](#), which usually begins with a receding frontal hairline that progresses to a bald patch on top of the head. It is very uncommon for women to bald following the male pattern unless there is excessive production of androgens in the body.





Grade 5

What causes female pattern hair loss?

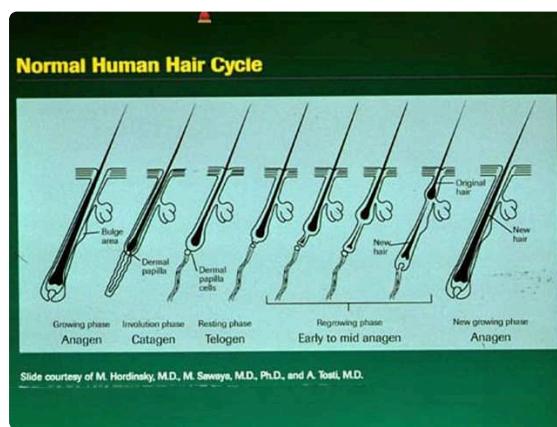
FPHL has a strong genetic predisposition. The mode of inheritance is polygenic, indicating that there are many genes that contribute to FPHL, and these genes could be inherited from either parent or both. Genetic testing to assess the risk of balding is currently not recommended, as it is unreliable.

Currently, it is not clear if androgens (male sex hormones) play a role in FPHL, although androgens have a clear role in male pattern baldness. The majority of women with FPHL have normal levels of androgens in their bloodstream. Due to this uncertain relationship, the term FPHL is preferred to 'female androgenetic alopecia'.

The role of oestrogen is uncertain. FPHL is more common after the menopause suggesting oestrogens may be stimulatory for hair growth. But laboratory experiments have also suggested oestrogens may suppress hair growth.

What is the normal hair growth cycle?

Everyone is born with a fixed number of hair follicles on the scalp that produce hairs throughout life. Hair grows from the base of the follicle at a rate of about one centimetre a month for about three years. This growth phase is called anagen. After anagen, the hair dies (catagen hair) and no longer grows. It sits dormant in the follicle for a three-month phase called telogen. After telogen, the hair follicle undergoes another anagen phase to produce new hair that grows out of the same follicle. As it grows, the old telogen hair is dislodged or pushed out. The hair cycle continues throughout life.



Hair cycle

Image © 1998 Merck Sharpe and Dohme (with permission)

Hair shedding

Increased hair shedding or telogen effluvium is a feature to FPHL. Women can use the hair shedding guide below to define whether hair shedding is normal or excessive,

To assess hair shedding, women should choose which of the six photographs of hair bundles best represents how much hair they shed on an average day.

Doctors can use the hair shedding scale to score hair loss at each patient visit to assess response to treatment. It can also be used in clinical trials to assess new treatments for excess hair shedding.

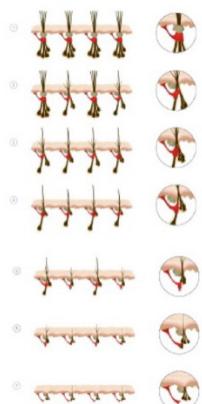
Hair shedding guide

Baseline shedding scores	Example images
Normal (1 to 3)	
Borderline (4)	
Excessive (5 - 6)	

Images courtesy of R. Sinclair, FACD

Hair miniaturisation

Unlike other areas of the body, hairs on the scalp tend to grow in tufts of 3–4. In androgenetic alopecia, the tufts progressively lose hairs. Eventually, when all the hairs in the tuft are gone, bald scalp appears between the hairs.



How long does it take for FPHL to progress?

FPHL can affect women in any age group, but it occurs more commonly after menopause. The hair loss process is not constant and usually occurs in fits and bursts. It is not uncommon to have accelerated phases of hair loss for 3–6 months, followed by periods of stability lasting 6–18 months. Without medication, it tends to progress in severity over the next few decades of life.

What are the effects of female pattern hair loss?

Many studies have shown that hair loss is not merely a cosmetic issue, but it also causes significant **psychological distress**. Compared to unaffected women, those affected have a more negative body image and are less able to cope with daily functioning. Hair loss can be associated with low self-esteem, depression, introversion, and feelings of unattractiveness. It is especially hard to live in a society that places great value on youthful appearance and attractiveness.

Should I have any hormone tests done?

Blood tests include female and male sex hormone levels as well as thyroid function, as part of the diagnostic workup.

The majority of women affected by FPHL do not have underlying hormonal abnormalities. However, a few women with FPHL are found to have excessive levels of androgens. These women also tend to suffer from acne, irregular menses and excessive facial and body hair. These symptoms are characteristic of the **polycystic ovarian syndrome** (PCOS) although the majority of women with PCOS do not experience hair loss. Less often, **congenital adrenal hyperplasia** may be responsible.

What treatments are available?

Treatments are available for FPHL although there is no cure. It is important to manage expectations when seeking treatment, as the aim is to slow or stop the progression of hair loss rather than to promote hair regrowth. However, some women do experience hair regrowth with treatment. Results are variable, and it is not possible to predict who may or may not benefit from treatment.

A Cochrane systematic review published in 2012 concluded that **minoxidil** solution was effective for FPHL. Minoxidil is available as 2% and 5% solutions; the stronger preparation is more likely to irritate and may cause undesirable hair growth unintentionally on areas other than the scalp.

Hormonal treatment, i.e. oral medications that block the effects of androgens (e.g. spironolactone, cyproterone, finasteride and flutamide) is also often tried.

A combination of low dose oral minoxidil (eg, 2.5 mg daily) and spironolactone (25 mg daily) has been shown to significantly improve hair growth, reduce shedding and improve hair density.

Once started, treatment needs to continue for at least six months before the benefits can be assessed, and it is important not to stop treatment without discussing it with your doctor first. Long term treatment is usually necessary to sustain the benefits.

Cosmetic camouflages include coloured hair sprays to cover thinning areas on the scalp, hair bulking fibre powder, and **hair wigs**. **Hair transplantation** for FPHL is becoming more popular although not everyone is suitable for this procedure.

[Low-level laser therapy](#) is of unproven benefit in pattern balding, but one device has been approved by the FDA for marketing. [Platelet-rich plasma](#) injections are also under investigation. Further studies are required to determine the magnitude of the benefit if any.

Where do I go to seek help?

Your first stop would be to see your general practitioner (GP) who can perform a medical workup to exclude other reasons for hair loss. Your GP can refer you to a dermatologist for further management of FPHL. Sometimes, it may be necessary for your doctor to perform a scalp [biopsy](#) to confirm this diagnosis.

It is important to seek reliable information and advice from authoritative sources as there are many bogus treatments that are expensive and do not work.

Folliculitis

Author: Hon A/Prof Amanda Oakley, Dermatologist, Hamilton, New Zealand. Updated by Dr Oakley and Clare Morrison, Copy Editor. April 2014.

What is folliculitis?

Folliculitis means an inflamed hair follicle due to any cause. The result is a tender red spot, often with a surface pustule.

Folliculitis may be superficial or deep. It can affect anywhere there are hairs, including chest, back, buttocks, arms, and legs. [Acne](#) and its variants are also types of folliculitis.



Superficial bacterial folliculitis





[See more images of folliculitis.](#)

What causes folliculitis?

Folliculitis can be due to infection, occlusion (blockage), irritation and various skin diseases.

Folliculitis due to infection

Swabs should be taken from the pustules for cytology and culture in the laboratory to determine if folliculitis is due to an infection.

Bacteria

Bacterial folliculitis is commonly due to *Staphylococcus aureus*. If the infection involves the deep part of the follicle, it results in a painful **boil**. Recommended treatment includes careful hygiene, [antiseptic cleanser or cream](#), [antibiotic ointment](#), or [oral antibiotics](#).

Spa pool folliculitis is due to infection with *Pseudomonas aeruginosa*, which thrives in warm water.

Gram-negative folliculitis is a pustular facial eruption also due to infection with *Pseudomonas aeruginosa* or other similar organisms. When it appears, it usually follows tetracycline treatment of acne but is quite rare.

Yeasts

The most common yeast to cause a folliculitis is *Pityrosporum ovale*, also known as **Malassezia**.

Malassezia folliculitis (*pityrosporum folliculitis*) is an itchy acne-like condition usually affecting the upper trunk of a young adult. Treatment includes avoiding moisturisers, stopping any antibiotics and using [topical antifungal](#) or [oral antifungal](#) medication for several weeks.

Candida albicans can also provoke a folliculitis in skin folds ([intertrigo](#)) or the beard area. It is treated with appropriate [topical](#) or [oral antifungal agents](#).

Fungi

Ringworm of the scalp ([tinea capitis](#)) usually results in scaling and hair loss, but sometimes results in folliculitis. In New Zealand, cat ringworm (*Microsporum canis*) is the commonest organism causing scalp fungal infection. Other [fungi](#) such as *Trichophyton tonsurans* are increasingly reported. Treatment is with an oral antifungal agent for several months.

Viral infections

Folliculitis may be caused by the [herpes simplex](#) virus. This tends to be tender and resolves without treatment in around ten days. Severe recurrent attacks may be treated with [aciclovir](#) and other antiviral agents.

[Herpes zoster](#) (the cause of shingles) may present as folliculitis with painful pustules and crusted spots within a dermatome (an area of skin supplied by a single nerve). It is treated with high-dose [aciclovir](#).

[Molluscum contagiosum](#), common in young children, can present with follicular umbilicated papules, usually clustered in and around a body fold. Molluscum may provoke [dermatitis](#).

Parasitic infection

Folliculitis on the face or scalp of older or immunosuppressed adults may be due to colonisation by hair follicle mites (*demodex*). This is known as [demodicosis](#).

The human infestation, [scabies](#), often provokes folliculitis, as well as non-follicular papules, vesicles and pustules.

Folliculitis due to irritation from regrowing hairs

Folliculitis may arise as hairs regrow after [shaving](#), [waxing](#), [electrolysis](#), or plucking. Swabs taken from the pustules are sterile – there is no growth of bacteria or other organisms. In the beard area irritant folliculitis is known as [pseudofolliculitis barbae](#) (or [folliculitis barbae](#) if associated with an infection).

Irritant folliculitis is also common on the lower legs of women ([shaving](#) rash). It is frequently very itchy. Treatment is to stop hair removal, and not begin again for about three months after the folliculitis has settled. To prevent reoccurring irritant folliculitis, use a gentle hair removal method, such as a lady's electric razor. Avoid soap and apply plenty of shaving gel, if using a blade shaver.

Folliculitis due to contact reactions

Occlusion

Paraffin-based ointments, [moisturisers](#), and adhesive plasters may all result in a sterile folliculitis. If a moisturiser is needed, choose an oil-free product, as it is less likely to cause occlusion.

Chemicals

[Coal tar](#), cutting oils and other chemicals may cause an irritant folliculitis. Avoid contact with the causative product.

Topical steroids

Overuse of [topical steroids](#) may produce a folliculitis. [Perioral dermatitis](#) is a facial folliculitis provoked by moisturisers and topical steroids. Perioral dermatitis is treated with [tetracycline](#) antibiotics for six weeks or so.

Folliculitis due to immunosuppression

[Eosinophilic folliculitis](#) is a specific type of folliculitis that may arise in some immune-suppressed individuals such as those infected by [human immunodeficiency virus](#) (HIV) or those who have cancer.

Folliculitis due to drugs

Folliculitis may be due to drugs, particularly corticosteroids ([steroid acne](#)), androgens (male hormones), adrenocorticotropic hormone (ACTH), lithium, isoniazid (INH), phenytoin and B-complex vitamins. Treatment with protein kinase inhibitors ([epidermal growth factor receptor inhibitors](#)) and targeted therapy for metastatic melanoma ([vemurafenib, dabrafenib](#)) nearly always results in folliculitis.

Folliculitis due to inflammatory skin diseases

Certain uncommon inflammatory skin diseases may cause permanent hair loss and scarring because of deep-seated sterile folliculitis. These include:

- [Lichen planus](#)
- [Discoid lupus erythematosus](#)
- [Folliculitis decalvans](#)
- [Folliculitis keloidal](#).

Treatment depends on the underlying condition and its severity. A [skin biopsy](#) is often necessary to establish the diagnosis.

Acne variants

Acne and acne-like (acneform) disorders are also forms of folliculitis. These include:

- [Acne vulgaris](#)
- [Nodulocystic acne](#)
- [Rosacea](#)
- [Scalp folliculitis](#)
- [Chloracne](#).

The [follicular occlusion syndrome](#) refers to:

- [Hidradenitis suppurativa](#) (acne inversa)
- [Acne conglobata](#) (a severe form of nodulocystic acne)
- [Dissecting cellulitis](#) (perifolliculitis capitis abscedens et suffodiens)
- [Pilonidal sinus](#).

Treatment of the acne variants may include [topical therapy](#) as well as long courses of [tetracycline](#) antibiotics, [isotretinoin](#) (vitamin-A derivative) and, in women, [antiandrogenic therapy](#).

Buttock folliculitis

Folliculitis affecting the buttocks is quite common in males and females.

Acute buttock folliculitis is usually bacterial in origin (like [boils](#)), resulting in red painful papules and pustules. It clears with antibiotics.

Chronic buttock folliculitis does not often cause significant symptoms but it can be very persistent. Although [antiseptics](#), [topical acne treatments](#), peeling agents such as alpha-hydroxy acids, long courses of oral [antibiotics](#) and [isotretinoin](#) can help buttock folliculitis, they are not always effective. [Hair removal](#) might be worth trying if the affected area is hairy. As regrowth of hair can make it worse, permanent hair reduction by [laser](#) or [intense pulsed light](#) (IPL) is best.

Fungal nail infections

October 2022

Author: Dr Adeline Hillan, Royal Perth Hospital, Australia (2022)
Previous contributors: A/Prof Amanda Oakley, Dermatologist (2003)
Reviewing dermatologist: Dr Ian Coulson
Edited by the DermNet content department

What are fungal nail infections?

Fungal infection of the nail, also known as onychomycosis, compromises over 50% of all nail disease with an estimated prevalence of 5.5%. They can affect the toenails, fingernails, or both.

Fungal nail infections are also known as tinea unguium in the case of dermatophyte infections.



Total fingernail onychomycosis in type 6 skin



Onychomycosis



White discolouration of the distal nail plate in superficial white onychomycosis



Proximal onychomycosis



Tinea unguis, toenails



Total onychomycosis due to Fusarium species

[Click here for more images](#)

Who gets fungal nail infections?

Onychomycosis is common in older aged adults (over 65 years), diabetics, immunocompromised patients (especially those with [HIV disease](#)), and athletes. Onychomycosis may be present amongst family members due to autosomal inheritance (HLA-DR8) or environmental factors. It rarely occurs in children.

Other associated comorbidities include: [tinea pedis](#), [tinea manuum](#), [psoriasis](#), peripheral vascular disease, [venous insufficiency](#), hallux valgus, smoking, asymmetric gait nail unit syndrome, [Down syndrome](#), and obesity.

Predisposing patient factors include: chronic [paronychia](#), [hyperhidrosis](#) (eg, with occlusive footwear), nail trauma, and using communal bathing or changing facilities.

What causes a fungal nail infection?

Onychomycosis can be due to infection with [dermatophytes](#) or [non-dermatophytes](#) such as moulds and yeasts.

Dermatophytes (over 75% of cases): *Trichophyton rubrum*, *Epidermophyton floccosum*, *Microsporum* species, *Trichophyton verrucosum*, *Trichophyton tonsurans*, *Trichophyton violaceum*, *Trichophyton soudanense*, *Trichophyton krajdenii*, *Trichophyton equinum*, and *Arthoderma* species.

Non-dermatophyte

Moulds (10% of cases): *Aspergillus* species, *Scopulariopsis* species, *Fusarium* species, *Acremonium* species, *Syncephalastrum* species, *Scytalidium* species, *Paecilomyces* species, *Neoscytalidium* species, *Chaetomium* species, *Onchoccola* species, and *Alternaria* species.

Yeasts (uncommon): *Candida albicans*, and rarely [non-albicans candida yeasts](#) (eg, *tropicalis*, or *parapsilosis*)

For more information, see [non-dermatophyte mould onychomycosis](#).

Emerging evidence of the role of biofilm in fungal nail disease may account for [antifungal drug resistance](#) and increased virulence.

What are the clinical features of onychomycosis?

Onychomycosis may affect a single nail or multiple, commonly affecting the first toenail. It may also affect the surrounding skin, however, very rarely causes systemic involvement.

Clinical types include:

Distal and lateral subungual onychomycosis – the distal end and sides of the nail lift or become discoloured and crumble.

Superficial white onychomycosis – flaky, white patches and pits appear on the top of the nail plate.

Proximal subungual onychomycosis – the proximal nail plate close to the lunula becomes discoloured and thickened. This pattern of disease is often related to underlying [HIV infection](#).

Endonyx onychomycosis – milky white discolouration of the nail plate develops without subungual hyperkeratosis or onycholysis.

[Onychauxis](#).

Features that are observed include:

[Subungual hyperkeratosis](#) – scaling occurs under the nail

Jagged and crumbling of the free end of the nail plate

Discolouration of the nail, eg, yellow, white, grey, or green discolouration

Ridging, crumbling, and sometimes eventual complete nail plate destruction

Scaling on the plantar skin and web spaces due to associated [tinea pedis](#)

Onychoma or dermatophytoma – a thick localised area of infection in the nail plate.

Onychomycosis may complicate other nail pathology such as trauma or [psoriasis](#).

Candida infection of the nail plate generally results from [paronychia](#) and starts near the nail fold (the cuticle). The nail fold is swollen and red, lifted off the nail plate. White, yellow, green, or black marks appear on the nearby nail and spread. The nail may lift off its bed and is tender if you press on it.

[Mould](#) infections are similar in appearance to [tinea unguis](#).

[Click here for images](#)

What are the complications of fungal nail infections?

Fungal nail infections are often regarded as a trivial cosmetic problem. However, the effect it may have on one's quality of life is undervalued as it can cause significant pain affecting full mobility and activities, and social stigma.

How is onychomycosis diagnosed?

Physical examination of all nails, and a combination of tools, such as those listed below, may be used to improve speed and accuracy of diagnosis.

Dermoscopy

[Dermoscopy](#) may be able to differentiate between onychomycosis, traumatic [onycholysis](#), and [melanonychia](#). Common dermatoscopic findings include linear bands which round proximally and taper distally, discolouration, non-longitudinal homogenous or reverse triangular patterns, subungual keratosis, white/yellow streaks, and nail plate scales.

The pattern of fungal invasion is further divided into:

Superficial onychomycosis – white patches affecting distal nail

Proximal subungual onychomycosis – involvement of the proximal nail fold under surface to distal

Endonyx onychomycosis – infection of the nail plate but not the nail bed

Mixed pattern onychomycosis.

Mycology specimens

Clippings should be taken from the crumbling free edge of the affected nail.

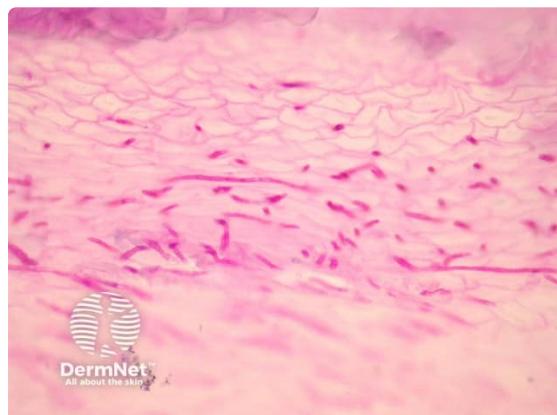
The most proximal areas of the dystrophic nail give the best yield on microscopy and culture; they can conveniently be obtained by scraping as proximally as possible under the nail with the hooked end of a clean nail file.

Lesions of superficial white onychomycosis can be scraped using a No. 15 scalpel blade.

Proximal subungual onychomycosis can be sampled by paring the overlying nail plate.

Microscopic examination reviewed under [light microscopy](#) using potassium hydroxide (to dissolve keratinocyte material) is a quick test to assess for the presence of fungal hyphae, although it lacks sensitivity and specificity.

Histopathological assessment of nail clippings using haematoxylin-eosin, periodic acid-Schiff, or Grocott methenamine silver staining to visualise fungal hyphae is easy and sensitive. For more information, see [histology stains](#) and [laboratory tests for fungal infection](#).



Onychomycosis pathology

Fungal culture testing

Fungal cultures can identify the causative organism and is the standard diagnostic test, however, results can take weeks and a large specimen collection may be required. This technique requires the nail to be cleaned with 70% isopropyl alcohol and soapy water prior to specimen collection.

Samples should be taken prior to starting any treatment.



Trichophyton rubrum culture on agar slope



The dermatophyte *T.rubrum* growing on Sabouraud fungal culture medium

Polymerase chain reaction testing (PCR)

PCR testing quickly identifies the offending organism and is highly sensitive and specific. It is becoming more commonly available, however is more costly than microscopy or fungal culture testing.

Other

Other techniques such as [confocal microscopy](#), [optical coherence tomography](#), infrared thermography, flow cytometry, immunochromatography, and mass spectrometry are currently being explored and rarely used.

A nail biopsy may also reveal [characteristic histopathological features of onychomycosis](#).

What is the differential diagnosis of onychomycosis?

Many other nail diseases may mimic the clinical signs of onychomycosis, hence the importance of diagnostic confirmation to ensure malignant conditions are not missed.

Benign conditions include: bacterial infection such as [pseudomonas argeuniosa \(CAP\)](#), [psoriasis](#), [lichen planus](#), subungual and periungual verruca, [paronychia](#), [subungual exostosis](#), [onychomatricoma](#), [yellow nail syndrome](#), and [idiopathic/traumatic onycholysis](#).

Malignant conditions include: subungual [squamous cell carcinoma](#) and [subungual melanoma](#).

What is the treatment for fungal nail infections?

Treatment aims to eliminate the offending organism and restore the nail to health and a normal appearance. Fingernail infections are usually cured more quickly and effectively than toenail infections.

Mild infections affecting less than 50% of one or two nails may respond to [topical antifungal medication](#), but cure usually requires an [oral antifungal medication](#) for several months.

Topical antifungal treatment options

[Ciclopirox 8% lacquer](#)
[Tavaborole 5% solution](#)
[Efinaconazole 10% solution](#)
[Amorolfine 5% lacquer](#).

These are often favoured before systemic treatments, if drug interactions are a concern.

With total nail involvement, [medical nail avulsion](#) using [urea](#) paste may enhance systemic antifungal efficacy.

Oral antifungal treatment options

[Terbinafine](#)
[Fluconazole](#)
[Itraconazole](#)
[Posaconazole](#)

These are widely used due to accessibility, low cost (some of them), and high efficacy; however, they may require an extended course especially for toenail involvement (3–4 months).

Non-pharmacological treatment options

Infrared laser therapy

Photodynamic therapy

Iontophoresis

Ultrasound.

Infrared radiation emission by laser can eradicate nail fungi in 1–3 sessions, however the effectiveness of laser compared to systemic therapy is lacking. Photodynamic therapy, plasma therapy, iontophoresis or ultrasound are adjuncts to topical antifungal therapy thought to enhance the absorption of drug to nail.

Treatment should be individualised, and the patient should be counselled for the estimated time to cure.

How do you prevent fungal nail infections?

Strategies to prevent recurrence include:

Keeping feet cool and dry; avoid using occlusive footwear and excessive sweating

Using thongs/flip-flops in public gyms and swimming pools

Discarding or treating infected footwear and socks

Avoiding nail trauma by trimming nails short

Avoiding unhygienic cosmetic nail practices

Using prophylactic antifungals in feet and webs

Patient counselling for optimal onychomycosis therapy to improve adherence

Addressing poorly controlled diabetes.

What is the outcome of fungal nail infections?

Approximately 20–25% of treated onychomycosis unfortunately relapse due to patient or pathogen factors such as poor circulation, advancing age, diabetes, immunosuppression, severe fungal nail clinical findings, mixed infections, and incomplete treatment. The Onychomycosis Severity Index can be used to predict response to therapy.

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FROM THE ACADEMY · Volume 74, Issue 5, P945-973.E33, May 2016

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Guidelines of care for the management of acne vulgaris

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Correction:

Errata

[Correction](#)

February 10, 2020

Abstract

Acne is one of the most common disorders treated by dermatologists and other health care providers. While it most often affects adolescents, it is not uncommon in adults and can also be seen in children. This evidence-based guideline addresses important clinical questions that arise in its



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management. Issues from grading of acne to the topical and systemic management of the disease are reviewed. Suggestions on use are provided based on available evidence.

Key words

acne · acne management · acne vulgaris · amoxicillin · antiandrogens · azithromycin · benzoyl peroxide · clindamycin · contraceptive agents · diet and acne · doxycycline · erythromycin · grading and classification of acne · guidelines · hormonal therapy · isotretinoin · light therapies · microbiological and endocrine testing · oral corticosteroids · *Propionibacterium acnes* · retinoids · salicylic · spironolactone · systemic therapies · tetracyclines · topical antibiotics · trimethoprim

Disclaimer

Adherence to these guidelines will not ensure successful treatment in every situation. Furthermore, these guidelines should not be interpreted as setting a standard of care, or be deemed inclusive of all proper methods of care, nor exclusive of other methods of care reasonably directed to obtaining the same results. The ultimate judgment regarding the propriety of any specific therapy or technique must be made by the physician and the patient in light of all the circumstances presented by the individual patient, and the known variability and biologic behavior of the disease. This guideline reflects the best available data at the time the guideline was prepared. The results of future studies may require revisions to the recommendations in this guideline to reflect new data.

Scope

This guideline addresses the management of adolescent and adult patients who present with acne vulgaris (AV). This document will discuss various acne treatments, including topical therapies, systemic agents, and physical modalities, including lasers and photodynamic therapy. In addition, grading/classification system, microbiology and endocrinology testing, complementary/alternative therapies, and the role of diet will be reviewed. This guideline does not examine the treatment of acne sequelae (eg, scarring or postinflammatory dyschromia).

Methods

A work group of 17 recognized acne experts, 1 general practitioner, 1 pediatrician, and 1 patient was convened to determine the scope of the guideline and identify clinical questions (**Table I**) in the diagnosis and management of AV. Work group members completed a disclosure of interests, which was periodically updated and reviewed throughout guideline development. If a potential conflict was noted, the work group member recused him or herself from discussion and drafting of recommendations pertinent to the topic area of the disclosed interest.



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What systems are most commonly used for the grading and classification of adult acne and acne vulgaris in adolescents (11-21 years of age) to adults?

What is the role of microbiologic and endocrine testing in evaluating patients with adult acne and acne vulgaris in adolescents to adults?

What is the effectiveness and what are the potential side effects of topical agents in the treatment of adult acne and acne vulgaris in adolescents to adults, including:

- Retinoids and retinoid-like drugs
- Benzoyl peroxide
- Topical antibiotics
- Salicylic/azelaic acids
- Sulfur and resorcinol
- Aluminum chloride
- Zinc
- Combinations of topical agents

What is the effectiveness and what are the potential side effects of the following systemic antibacterial agents in the treatment of adult acne and acne vulgaris in adolescents to adults, including:

Table I

Clinical questions used to structure the evidence review

* Indicates a new clinical question for this guideline.

[Open table in a new tab](#)

An evidence-based model was used and evidence was obtained for the clinical questions ([Table I](#)) using a systematic search of PubMed and the Cochrane Library database from May 2006 through September 2014 for clinical questions addressed in the previous version of this guideline published in 2007, and 1964 to 2014 for all newly identified clinical questions. Searches were prospectively limited to publications in the English language. MeSH terms and strings used in various combinations in the literature search included: acne or acne vulgaris combined with treatment, therapy, prevention, prophylaxis, grading, classification, scoring, microbiology, endocrinology, hormone, topical, retinoid, benzoyl peroxide (BP), antibiotic, doxycycline, minocycline, tetracycline, macrolide, erythromycin, azithromycin, trimethoprim (with or without sulfamethoxazole), oral contraceptives, antiandrogen, corticosteroid, isotretinoin, peel, complementary, alternative, herbal, diet, glycemic index, milk, antioxidants, probiotics, and fish oil. Additional studies were identified by hand-searching bibliographies of publications, including reviews and metaanalyses.

A total of 1145 abstracts were initially assessed for possible inclusion; 242 were retained for final review based on relevancy and the highest level of available evidence for the outlined clinical questions. Evidence tables were generated for these studies and used by the work group in developing recommendations. In addition, the evidence tables generated for the Academy's previous acne

guideline were also used by the work group. The Academy's previous published guidelines on acne were also evaluated, as were other current published guidelines on acne.^{1,2} Relevant references published after September 2014 are provided solely as supplemental supporting text information for recommendations as derived from the systematic search, and to address comments received during the guideline review and approval process.

The available evidence was evaluated using a unified system called the Strength of Recommendation Taxonomy (SORT) developed by editors of the US family medicine and primary care journals (ie, *American Family Physician*, *Family Medicine*, *Journal of Family Practice*, and *BMJ USA*).³ Evidence was graded using a 3-point scale based on the quality of methodology (eg, randomized control trial, case control, prospective/retrospective cohort, case series, etc) and the overall focus of the study (ie, diagnosis, treatment/prevention/screening, or prognosis) as follows:

- I.** Good-quality patient-oriented evidence (ie, evidence measuring outcomes that matter to patients: morbidity, mortality, symptom improvement, cost reduction, and quality of life).
- II.** Limited-quality patient-oriented evidence.
- III.** Other evidence, including consensus guidelines, opinion, case studies, or disease-oriented evidence (ie, evidence measuring intermediate, physiologic, or surrogate end points that may or may not reflect improvements in patient outcomes).

Clinical recommendations were developed on the best available evidence tabled in the guideline. The strength of recommendation was ranked as follows:

- A.** Recommendation based on consistent and good-quality patient-oriented evidence.
- B.** Recommendation based on inconsistent or limited-quality patient-oriented evidence.
- C.** Recommendation based on consensus, opinion, case studies, or disease-oriented evidence.

In those situations where documented evidence-based data were not available or were showing inconsistent or limited conclusions, expert opinion and medical consensus was used to generate clinical recommendations.

This guideline has been developed in accordance with the American Academy of Dermatology/American Academy of Dermatology Association "Administrative Regulations for Evidence-Based Clinical Practice Guidelines" (version approved August 2012), which include the opportunity for review and comment by the entire AAD membership and final review and approval by the AAD Board of Directors.⁴ This guideline will be considered current for a period of 5 years from the date of publication, unless reaffirmed, updated, or retired at or before that time.

Definition

AV is a chronic inflammatory dermatosis notable for open or closed comedones (blackheads and whiteheads) and inflammatory lesions, including papules, pustules, or nodules (also known as cysts).

Introduction



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Acne is a common skin disease, especially in adolescents and young adults. Approximately 50 million people in the United States have AV.⁵ Acne affects approximately 85% of teenagers, but can occur in most age groups⁶ and can persist into adulthood. The prevalence of acne in adult women is about 12%.⁷ There is no mortality associated with acne, but there is often significant physical and psychological morbidity, such as permanent scarring, poor self-image, depression, and anxiety. The direct cost of the disease is estimated to exceed \$3 billion per year.⁶

Acne is a multifactorial inflammatory disease affecting the pilosebaceous follicles of the skin. The current understanding of acne pathogenesis is continuously evolving. Key pathogenic factors that play an important role in the development of acne are follicular hyperkeratinization, microbial colonization with *Propionibacterium acnes*, sebum production, and complex inflammatory mechanisms involving both innate and acquired immunity. In addition, studies have suggested that neuroendocrine regulatory mechanisms, diet, and genetic and nongenetic factors all may contribute to the multifactorial process of acne pathogenesis. An algorithm for the treatment and management of acne in adolescents and young adults is shown in Fig 1.

	Mild	Moderate	Severe
1st Line Treatment	Benzoyl Peroxide (BP) or Topical Retinoid -or- Topical Combination Therapy** BP + Antibiotic or Retinoid + BP or Retinoid + BP + Antibiotic	Topical Combination Therapy** BP + Antibiotic or Retinoid + BP or Retinoid + BP + Antibiotic -or- Oral Antibiotic + Topical Retinoid + BP -or- Oral Antibiotic + Topical Retinoid + BP + Topical Antibiotic	Oral Antibiotic + Topical Combination Therapy** BP + Antibiotic or Retinoid + BP or Retinoid + BP + Antibiotic -or- Oral Isotretinoin
Alternative Treatment	Add Topical Retinoid or BP (if not on already) -or- Consider Alternate Retinoid -or- Consider Topical Dapsone	Consider Alternate Combination Therapy -or- Consider Change in Oral Antibiotic -or- Add Combined Oral Contraceptive or Oral Spironolactone (Females) -or- Consider Oral Isotretinoin	Consider Change in Oral Antibiotic -or- Add Combined Oral Contraceptive or Oral Spironolactone (Females) -or- Consider Oral Isotretinoin



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Fig 1 Treatment algorithm for the management of acne vulgaris in adolescents and young adults. The double asterisks (**) indicate that the drug may be prescribed as a fixed combination product or as separate component. BP, Benzoyl peroxide.

Systems for the grading and classification of acne

Acne grading systems may be useful in patient care. Such systems can assist in more specific classification of disease, help determine appropriate treatment options, and monitor improvement during the treatment course. Recommendations for grading and classifying acne are shown in [Table II](#), and the strength of recommendations for grading and classifying acne is shown in [Table III](#).

Clinicians may find it helpful to use a consistent grading/classification scale (encompassing the numbers and types of acne lesions as well as disease severity, anatomic sites, and scarring) to facilitate therapeutic decisions and assess response to treatment.

Currently, no universal acne grading/classifying system can be recommended.

Table II

Recommendations for grading and classification of acne

[Open table in a new tab](#)

Recommendation	Strength of recommendation	Level of evidence	References
Grading/classification system	B	II, III	8-39
Microbiologic testing	B	II, III	40-48
Endocrinologic testing	B	I, II	49-56
Topical therapies			
Benzoyl peroxide	A	I, II	57-59
Topical antibiotics (eg, clindamycin and erythromycin)	A	I, II	60-66



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Table III

Strength of recommendations for the management and treatment of acne vulgaris

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Numerous acne assessment tools have been described, taking into account various factors, such as type of acne, severity of acne, number of acne lesions, anatomic location/extent of acne,⁸ quality of life and other psychosocial metrics,⁹⁻¹³ and scarring,¹⁴ among other measures.¹⁵⁻²⁴ Recently, 18 of these grading scales were ranked based on a variety of characteristics.²⁵ To date, there is no universally agreed-upon grading system, and systems can differ greatly between studies. In addition, interobserver reliability of these scales varies, but has been poor in some studies.^{17,26,27} Methods such as photographic standards have been used to improve reproducibility.

Improvements in digital technology, photographic equipment, and teledermatology may allow for accurate, remote assessment of acne in the near future.²⁸⁻³⁰ Scientific measures, such as ultraviolet-induced red fluorescence,³¹⁻³³ casual sebum level,^{34,35} skin capacitance imaging,³⁶ skin surface pH,^{37,38} and transepidermal water loss³⁹ may also help to more objectively classify and rate acne in the future. Reproducibility, as well as ease of use and acceptance by dermatologists, will be essential for the success of any grading system.

Microbiologic testing

P acnes, a Gram-positive anaerobic rod, is the primary bacterium implicated in acne.⁴⁰⁻⁴² It has specific, nonstandard culture requirements that prohibit routine culture. Currently, microbiologic testing of acne lesions is largely unnecessary because it does not affect management, and successful antibiotic treatment may not result from a reduction of bacterial numbers.⁴⁰ The antibiotics typically used in the management of acne, tetracyclines, have additional antiinflammatory actions independent of microbial killing. As additional information is learned about *P acnes* from a molecular and genetic perspective, and its role in inciting inflammation in acne,⁴³⁻⁴⁸ more targeted therapeutic interventions in the future may result. Recommendations for microbiologic testing of acne are shown in Table IV and the strength of recommendations for microbiologic testing is shown in Table III.

Routine microbiologic testing is not recommended in the evaluation and management of patients with acne

Those who exhibit acne-like lesions suggestive of Gram-negative folliculitis may benefit from microbiologic testing

Routine endocrinologic evaluation (eg, for androgen excess) is not recommended for the majority of patients with acne



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Laboratory evaluation is recommended for patients who have acne and additional signs of androgen excess

Table IV

Recommendations for microbiologic and endocrinologic testing

[Open table in a new tab](#)

The prime situation where microbiologic testing is useful in patients with acne is in evaluating for Gram-negative folliculitis. This uncommon disorder presents as uniform and eruptive pustules, with rare nodules, in the perioral and perinasal regions, typically in the setting of prolonged tetracycline use. It is caused by various bacteria, such as *Klebsiella* and *Serratia*, and is unresponsive to many conventional acne treatments. Gram-negative folliculitis is typically diagnosed via culture of the lesions, and is generally treated with isotretinoin or an antibiotic to which the bacteria are sensitive. In cases of acne unresponsive to typical treatments—particularly with prominent truncal involvement or monomorphic appearance—*pityrosporum* folliculitis should be considered. *Staphylococcus aureus* cutaneous infections may appear similar to acne, and should be considered in the differential, particularly in cases of acute eruptions; a swab culture may be helpful in these cases.

Endocrinologic testing

While the role of androgens in acne pathogenesis is well known, endocrinologic evaluation is only warranted in certain cases, because most acne patients will have normal hormone levels. Testing is primarily indicated for patients with clinical features or a history of hyperandrogenism. In prepubertal children, these features include: acne, early-onset body odor, axillary or pubic hair, accelerated growth, advanced bone age, and genital maturation. Growth charts and a hand film for bone age are good screening tools before specific hormonal testing.^{1,49} In postpubertal females, clinical signs, such as infrequent menses, hirsutism, androgenetic alopecia, infertility, polycystic ovaries, clitoromegaly, and truncal obesity warrant further hormonal testing.^{1,2,50-52,165} Recalcitrant acne caused by androgen excess can also be seen in both men and women with nonclassical congenital adrenal hyperplasia (eg, 21-hydroxylase deficiency).^{166,167} Recommendations for endocrinologic testing of acne are shown in **Table IV**, and the strength of recommendations for endocrinologic testing is shown in **Table III**.

The most common cause of elevated androgens of ovarian origin is polycystic ovarian syndrome (PCOS).⁵³ It has recently been proposed that diagnosis of PCOS in adult females requires 2 of the 3 following criteria: androgen excess (clinical or biochemical), ovulatory dysfunction (oligo- or anovulation), or polycystic ovaries (based on ultrasonographic findings). In adolescent females, the diagnosis of PCOS can be made based on hyperandrogenism (clinical or biochemical) in the presence of persistent oligomenorrhea.¹⁶⁸ The differential diagnosis of PCOS includes thyroid disease, prolactin excess, and nonclassical congenital adrenal hyperplasia, among others.¹⁶⁸

Hormonal testing and interpretation of testing is complex. A typical hormone-screening panel includes free and total testosterone, dehydroepiandrosterone sulfate (DHEA-S), androstenedione,

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luteinizing hormone, and follicle-stimulating hormone.^{49,51-54,165} Growth hormone, insulin-like growth factor, lipid levels, insulin, sex hormone–binding globulin, free 17-β-hydroxysteroids, free androgen index, prolactin, estrogen, and progesterone may also be abnormal in those with severe acne.^{52,54-56,165,169} Insulin resistance may also represent a risk factor for acne in certain patients.¹⁷⁰ Patients with abnormal test results, or in whom there is a persistent concern for a hormonal disorder, should be further evaluated by an endocrinologist.

Topical therapies

The topical therapy of AV includes the usage of agents that are available over the counter or via prescription. Therapy choice may be influenced by age of the patient, site of involvement, extent and severity of disease, and patient preference. Topical therapies may be used as monotherapy, in combination with other topical agents or in combination with oral agents in both initial control and maintenance. Recommendations for use of topical therapies are shown in **Table V**, and the strength of recommendations for treatment of acne with topical therapies is shown in **Table III**. Prescribing information for all topical therapies is located in **Supplementary Tables I-XIII**. (Please note all Supplemental Tables can be found at www.jaad.org.)

Benzoyl peroxide or combinations with erythromycin or clindamycin are effective acne treatments and are recommended as monotherapy for mild acne, or in conjunction with a topical retinoid, or systemic antibiotic therapy for moderate to severe acne

Benzoyl peroxide is effective in the prevention of bacterial resistance and is recommended for patients on topical or systemic antibiotic therapy

Topical antibiotics (eg, erythromycin and clindamycin) are effective acne treatments, but are not recommended as monotherapy because of the risk of bacterial resistance

Topical retinoids are important in addressing the development and maintenance of acne and are recommended as monotherapy in primarily comedonal acne, or in combination with topical or oral antimicrobials in patients with mixed or primarily inflammatory acne lesions

Using multiple topical agents that affect different aspects of acne pathogenesis can be useful. Combination therapy should be used in the majority of patients with acne

Topical adapalene, tretinoin, and benzoyl peroxide can be safely used in the management of preadolescent

Table V

Recommendations for topical therapies

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Commonly used topical acne therapies include BP, salicylic acid, antibiotics, combination antibiotics with BP, retinoids, retinoid with BP, retinoid with antibiotic, azelaic acid, and sulfone agents. Although most physicians have anecdotal regimens they find beneficial, agents reviewed here are limited to those approved by the US Food and Drug Administration (FDA) for use in the United States, and for which peer-reviewed literature has been published.

BP is an antibacterial agent that kills *P acnes* through the release of free oxygen radicals and is also mildly comedolytic.^{171,172} No resistance to this agent has been reported, and the addition of BP to regimens of antibiotic therapy enhances results and may reduce resistance development. BP is available as topical washes, foams, creams, or gels, and can be used as leave-on or wash-off agents. Strengths available for acne therapy range from 2.5% to 10%. BP therapy is limited by concentration-dependent irritation, staining and bleaching of fabric, and uncommon contact allergy. Total skin contact time and formulation can also affect efficacy. Lower concentrations (eg, 2.5-5%), water-based, and wash-off agents may be better tolerated in patients with more sensitive skin.^{57,58} Results can be noted in as soon as 5 days.⁵⁹

Topical antibiotics for acne accumulate in the follicle and have been postulated to work through antiinflammatory mechanisms and via antibacterial effects.⁶⁰ These agents are best used in combination with BP (wash-off or leave-on), which increases efficacy and decreases the development of resistant bacterial strains. Monotherapy with topical antibiotics in the management of acne is not recommended because of the development of antibiotic resistance. Clindamycin 1% solution or gel is currently the preferred topical antibiotic for acne therapy.¹⁷³ Topical erythromycin in 2% concentration is available as a cream, gel, lotion, or pecten,^{61,62} but has reduced efficacy in comparison with clindamycin because of resistance of cutaneous Staphylococci and *P acnes*.^{60,63-66} Stable, fixed-combination agents are available with erythromycin 3%/BP 5%, clindamycin 1%/BP 5%, and clindamycin 1%/BP 3.75%.^{67-69,174} Combination agents may enhance compliance with treatment regimens. Rare reports of diarrhea or *Clostridium difficile*-related colitis with clindamycin topically have appeared in the literature, but the risk appears low.⁶⁶ Tolerance of these agents is excellent; clindamycin alone is pregnancy category B.

Topical retinoids are vitamin A derivatives that are prescription agents with randomized, double-blind, placebo-controlled trials supporting their use for acne treatment.^{70-72,175} Three active agents are available: tretinoin (0.025-0.1% in cream, gel, or microsphere gel vehicles), adapalene (0.1%, 0.3% cream, or 0.1% lotion^{73,74}), and tazarotene (0.05%, 0.1% cream, gel or foam). Each retinoid binds to a different set of retinoic acid receptors: tretinoin to alpha, beta, and gamma, and tazarotene and adapalene, selectively, to beta and gamma—thereby conferring slight differences in activity, tolerability, and efficacy. Retinoids are the core of topical therapy for acne because they are comedolytic, resolve the precursor microcomedone lesion, and are antiinflammatory.

These agents enhance any topical acne regimen and allow for maintenance of clearance after discontinuation of oral therapy. Retinoids are ideal for comedonal acne and, when used in combination with other agents, for all acne variants. Three topical agents are available that contain retinoids in combination with other products: adapalene 0.1%/BP 2.5%, approved for use in patients ≥9 years of age, and 2 agents with fixed combination clindamycin phosphate 1.2%/tretinoin 0.025% gel, approved for patients ≥12 years of age.^{75,76,176}



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Retinoid use may be limited by side effects, including dryness, peeling, erythema, and irritation, which can be mitigated by reduced frequency of application.¹⁷ Given any single agent, higher concentrations may be more efficacious, but with greater side effects.^{70,74,77} Some formulations of tretinoin (primarily generic products) are not photostable and should be applied in the evening. Tretinoin also may be oxidized and inactivated by the coadministration of BP. It is recommended that the 2 agents be applied at different times. Tretinoin microsphere formulation, adapalene, and tazarotene do not have similar restrictions. Topical retinoids have been associated with an increased risk of photosensitivity; concurrent daily sunscreen can be used to reduce the risk of sunburn.

There are several head-to-head studies with retinoid products. Some support greater efficacy of tazarotene over adapalene and tretinoin, and adapalene over tretinoin, but the concentrations and formulations used were varied.^{73,78-80} Data suggest that adapalene is better tolerated than multiple concentrations of tretinoin, but this is based on older formulations.⁸¹ Overall, the limitations of the existing studies prohibit direct efficacy comparisons of topical retinoids.

Tretinoin and adapalene are pregnancy category C, while tazarotene is category X; therefore, patients should be counseled on these pregnancy risks when starting a retinoid or if a woman patient desires pregnancy.

The therapy of acne in children <12 years of age with products approved by the FDA has expanded. Fixed combination BP 2.5%/adapalene 1% gel is approved for patients ≥9 years of age, and tretinoin 0.05% micronized tretinoin gel for patients ≥10 years of age. All other retinoids are approved by the FDA for patients ≥12 years of age. Current data show that retinoids in younger patients are effective and are not associated with increased irritation or risk.

Azelaic acid 20% is mildly effective as a comedolytic, antibacterial, and antiinflammatory agent. The agent has use in patients with sensitive skin or of Fitzpatrick skin types IV or greater because of the lightening effect of the product on dyspigmentation.^{82,83,178} Azelaic acid is category B in pregnancy.

The sulfone agent, dapson 5% gel, is available as a twice-daily agent for the therapy of AV. In clinical trials, topical dapson showed modest to moderate efficacy, primarily in the reduction of inflammatory lesions.^{84,85} Combination with topical retinoids may be indicated if comedonal components are present. The mechanism of action is poorly understood, and its ability to kill *P acnes* has been poorly studied. It is generally thought to work as an antiinflammatory agent. The benefit in women seems to exceed the benefit in male and adolescent patients.^{86,179} Topical dapson may be oxidized by the coapplication of BP, causing orange-brown coloration of the skin which can be brushed or washed off. Topical dapson 5% gel is pregnancy category C and has efficacy and safety data down to patients 12 years of age. Glucose-6-phosphate dehydrogenase testing is not required before starting topical dapson.

Salicylic acid is a comedolytic agent that is available over the counter in 0.5% to 2% strengths for the therapy of AV. Both wash-off and leave-on preparations are well tolerated. Clinical trials demonstrating the efficacy of salicylic acid in acne are limited.^{87,180}

Although sulfur and resorcinol have been used for many years in the treatment of acne, evidence from peer-reviewed literature supporting their efficacy is lacking.¹⁸¹ Aluminum chloride possesses

antibacterial activity and, therefore, has been investigated in the treatment of acne. Of 2 peer-reviewed studies, 1 found benefit¹⁸² and 1 did not.¹⁸³ Topical zinc alone is ineffective.¹⁸⁴⁻¹⁸⁶ There is some evidence to suggest the efficacy of sodium sulfacetamide.¹⁸⁷⁻¹⁸⁹ Topical niacinamide (nicotinamide) 2% to 4% gel is available over the counter. The limited studies available compare its efficacy to topical clindamycin 1% gel.^{190,191}

Systemic antibiotics

Systemic antibiotics have been a mainstay of acne treatment for years. They are indicated for use in moderate to severe inflammatory acne and should be used in combination with a topical retinoid and BP.^{95,192,193} Evidence supports the efficacy of tetracycline, doxycycline, minocycline, trimethoprim/sulfamethoxazole (TMP/SMX), trimethoprim, erythromycin, azithromycin, amoxicillin, and cephalexin. Recommendations for systemic antibiotics are shown in **Table VI**, and the strength of recommendations for treatment of acne with systemic antibiotics is shown in **Table III**. Prescribing information for systemic antibiotics is located in the **Supplementary Tables XIV-XXII**.

Systemic antibiotics are recommended in the management of moderate and severe acne and forms of inflammatory acne that are resistant to topical treatments

Doxycycline and minocycline are more effective than tetracycline, but neither is superior to each other

Although oral erythromycin and azithromycin can be effective in treating acne, their use should be limited to those who cannot use the tetracyclines (ie, pregnant women or children <8 years of age). Erythromycin use should be restricted because of its increased risk of bacterial resistance

Use of systemic antibiotics, other than the tetracyclines and macrolides, is discouraged because there are limited data for their use in acne. Trimethoprim-sulfamethoxazole and trimethoprim use should be restricted to patients who are unable to tolerate tetracyclines or in treatment-resistant patients

Systemic antibiotic use should be limited to the shortest possible duration. Re-evaluate at 3-4 months to minimize the development of bacterial resistance. Monotherapy with systemic antibiotics is not recommended

Concomitant topical therapy with benzoyl peroxide or a retinoid should be used with systemic antibiotics and

Table VI

Recommendations for systemic antibiotics

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The tetracycline class of antibiotics  should be considered first-line therapy in moderate to severe acne, except when contraindicated because of other circumstances (ie, pregnancy, ≤8 years of age, or Download PDF)

allergy). The antibiotics of the tetracycline class work by inhibiting protein synthesis by binding the 30S subunit of the bacterial ribosome. This class also has notable antiinflammatory effects, including inhibiting chemotaxis and metalloproteinase activity. Previous guidelines recommended minocycline as superior to doxycycline in reducing *P acnes*.¹ However, a recent Cochrane review of clinical trials found minocycline effective but not superior to other antibiotics in the treatment of acne.⁸⁸ There are few studies addressing dosing of the tetracycline class. Minocycline in an extended release form appears safest (at 1 mg/kg), but no dose response was found for efficacy.¹⁹⁴ Doxycycline appears effective in the 1.7 to 2.4 mg/kg dose range.⁸⁹ Subantimicrobial dosing of doxycycline (ie, 20 mg twice daily to 40 mg daily) has also shown efficacy in patients with moderate inflammatory acne.^{195,196}

Erythromycin and azithromycin have also been used in the treatment of acne. The mechanism of action for the macrolide class of antibiotics is to bind the 50S subunit of the bacterial ribosome. Again, there are some antiinflammatory properties for these medications, but the mechanisms are not well understood. Azithromycin has been primarily studied in the treatment of acne in open label studies with different pulse dosing regimens ranging from 3 times a week to 4 days a month, with azithromycin being an effective treatment in the time span evaluated—usually 2 to 3 months.^{92,197-204} A recent randomized controlled trial comparing 3 days per month of azithromycin to daily doxycycline did show superiority of doxycycline.²⁰⁵ Macrolides as the penicillin class represent an alternative when traditional antibiotics cannot be used.

TMP/SMX and trimethoprim have also been used for the treatment of acne. Sulfamethoxazole is bacteriostatic by blocking bacterial synthesis of folic acid, which is necessary for cell division. Trimethoprim is a folic acid analog that inhibits the enzyme dihydrofolate reductase. The 2 agents work together to block nucleotide and amino acid synthesis in the bacteria. Outside of case reports, there is 1 small, double-blind study showing that TMP/SMX is as effective as oxytetracycline.⁹³

Although data supporting their use are limited, penicillins and cephalosporins are sometimes used in the treatment of acne and can be used as an alternative treatment when circumstances dictate. In particular, these medications represent a useful option in patients who may be pregnant or who have allergies to the other classes of antibiotics. These antibiotics work by binding the penicillin-binding proteins in the bacterial cell membrane and inhibiting bacterial cell wall synthesis. There are few references to support the use of these medications in the treatment of acne outside of case reports. However, there is a small retrospective chart review with cephalexin where the majority of patients showed some clinical improvement on this medication.⁹⁴

Adverse events of systemic therapy are often a concern to patients and practitioners. However, severe adverse effects of systemic antibiotics in the treatment of acne are rare. Vaginal candidiasis and drug eruptions can occur with any antibiotic.

Adverse events with the tetracycline class vary with each medication. Photosensitivity can be seen with the tetracycline class, doxycycline being more photosensitizing than minocycline. Doxycycline is more frequently associated with gastrointestinal disturbances, and higher doses are more likely to cause symptoms.⁸⁹ Minocycline has been associated with tinnitus, dizziness, and pigment deposition of the skin, mucous membranes, and teeth. Minocycline pigmentation is more common in patients taking higher doses for longer periods of time. Doxycycline is primarily metabolized by the liver, and

can be used safely in most patients with renal impairment. When minocycline is compared to other tetracyclines, more serious adverse events are reported (8.8 cases per 100,000 patient years).^{88,90} The rare serious events associated with minocycline include autoimmune disorders, such as drug reaction with eosinophilia and systemic symptoms (DRESS), drug-induced lupus, and other hypersensitivity reactions.^{91,206-209} Finally, pseudotumor cerebri is a rare phenomenon associated with the tetracycline class of antibiotics.

The adverse events of TMP/SMX include gastrointestinal upset, photosensitivity, and drug eruptions. Multiple cutaneous reactions have been observed with patients on this medication, the most severe eruptions being Stevens–Johnson syndrome and toxic epidermal necrolysis.^{210,211} Such severe eruptions are more common in patients with HIV. The relative risk for such a severe reaction varies, but is still a rare event, with studies citing the crude relative risk at 172.²¹¹ Disorders of the hematopoietic system can also occur with TMP/SMX and can include serious blood dyscrasias, such as neutropenia, agranulocytosis, aplastic anemia, and thrombocytopenia. Although these are rare adverse events, patients on long-term therapy with this medication should be periodically monitored with a complete blood cell count. Cases of fulminant hepatitis necrosis have also occurred in patients taking this medication, as has respiratory hypersensitivity. The concurrent use of TMP/SMX and methotrexate (MTX) can be associated with severe toxicity ([Supplementary Table XVII](#)).

The macrolide class of antibiotics is most commonly associated with gastrointestinal disturbances. Erythromycin is associated with a higher incidence of diarrhea, nausea, and abdominal discomfort than azithromycin. Macrolides have been reported to cause cardiac conduction abnormalities, and rarely hepatotoxicity has been reported. Macrolide antibiotics can also decrease metabolism of cyclosporine. Azithromycin has been associated with cutaneous hypersensitivity reactions.

Penicillins and cephalosporins are most associated with the adverse events of hypersensitivity reactions ranging from mild drug eruptions to anaphylaxis. Gastrointestinal disturbances are also common and include nausea, diarrhea, and abdominal distention and discomfort.

When prescribing systemic antibiotics, the issue of bacterial resistance remains a major concern. The Centers for Disease Control and Prevention (CDC) has stressed antibiotic stewardship. This is an initiative to promote the appropriate use of antibiotics where patients receive the right dose of the right antibiotic at the right time for the right duration. Limiting antibiotic use to the shortest possible duration, ideally 3-4 months, can be accomplished with the concomitant use of a retinoid or retinoid/BP.^{212,213} While limiting the use of systemic antibiotics is necessary, the work group's consensus agrees there are a subset of patients for whom alternative therapies are inappropriate and who may require a longer course of antibiotics even while taking topical medications. In such patients, consistent follow-up and reevaluation should be used to use the antibiotic for the shortest time necessary. Monotherapy with oral antibiotics is strongly discouraged. The use of topical maintenance regimens cannot be overemphasized. Topical therapies can accomplish continued efficacy months after the discontinuation of systemic antibiotics.^{95,96,212,214} The work group's consensus agrees that such maintenance is paramount to reducing antibiotic resistance.²¹⁵ Other attempts to limit antibiotic use revolve around different dosing recommendations, such as pulse dosing and submicrobial dosing. No alternate dosing routines consistently appear superior to standard dosing.



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Finally, limiting systemic antibiotic use is urged because of the reported associations of inflammatory bowel disease,⁹⁷ pharyngitis,²¹⁶ *C difficile* infection,^{217,218} and the induction of *Candida* vulvovaginitis.

Hormonal agents

Combination oral contraceptive pills (COCs) contain both an estrogen and a progestin component. COCs were first approved by the FDA for contraception in the United States in 1960. They prevent ovulation and pregnancy by inhibiting gonadotropin-releasing hormone and, subsequently, follicle-stimulating and luteinizing hormones. These hormones are needed to begin follicular maturation and for ovulation; in their absence, ovulation does not occur. Recommendations for hormonal agents are shown in **Table VII**, and the strength of recommendations for the treatment of acne with hormonal agents is shown in **Table III**. World Health Organization (WHO) recommendations for COC usage eligibility are listed in **Table VIII**. Prescribing information for hormonal therapies is located in **Supplementary Tables XXIII-XXVIII**.

Estrogen-containing combined oral contraceptives are effective and recommended in the treatment of inflammatory acne in females

Spironolactone is useful in the treatment of acne in select females

Oral corticosteroid therapy can be of temporary benefit in patients who have severe inflammatory acne while starting standard acne treatment

In patients who have well documented adrenal hyperandrogenism, low-dose oral corticosteroids are recommended in treatment of acne

Table VII

Recommendations for hormonal agents

[Open table in a new tab](#)

COC use not recommended	Caution or special monitoring
Pregnancy	Breastfeeding (6 weeks-6 months postpartum)
Current breast cancer	Postpartum (<21 days)
Breastfeeding <6 weeks postpartum	Age ≥35 years and light smoker (<15 cigarettes per day)



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COC use not recommended	Caution or special monitoring
Age ≥35 years and heavy smoker (≥15 cigarettes per day)	History of hypertension (including pregnancy) or if monitoring is not feasible
Hypertension: systolic, ≥160 mm Hg; diastolic, ≥100 mm Hg	Hypertension: systolic, 140-159 mm Hg; diastolic, 90-99 mm Hg; or controlled and monitored

Table VIII

World Health Organization recommendations for combined oral contraceptive usage eligibility*

COC, Combined oral contraceptive.

* Data taken from Arrington et al.²¹⁹

[Open table in a new tab](#)

COCs have evolved since 1960. Ethynodiol levels have gradually decreased from around 50 to 150 µg per pill to as low as 10 µg. A variety of different progestational moieties have been used, beginning with the first-generation progestins, the estranes (ie, norethindrone and ethynodiol diacetate). Second-generation progestins include levonorgestrel and norgestimate; these progestins are referred to as the gonanes. Third-generation progestins include less androgenic gonane progestins, such as desogestrel and gestodene. First-, second-, and third-generation progestins are derived from testosterone and alone have androgenic potential. Fourth-generation progestins are not derived from testosterone and include the antiandrogenic progestin drospirenone. While progestins vary in their androgenic potential, evidence suggests that when combined with ethynodiol, the net effect of all COCs is antiandrogenic.^{219,220}

There are currently 4 COCs approved by the FDA for the treatment of acne. They are ethynodiol/norgestimate, ethynodiol/norethindrone acetate/ferrous fumarate, ethynodiol/drospirenone, and ethynodiol/drospirenone/levomefolate. The mechanism of action of COCs in the treatment of acne is based on their antiandrogenic properties. These pills decrease androgen production at the level of the ovary and also increase sex hormone-binding globulin, binding free circulating testosterone and rendering it unavailable to bind and activate the androgen receptor. In addition, COCs reduce 5-alfa-reductase activity and block the androgen receptor.^{219,221-223}

Numerous randomized controlled clinical trials have assessed the efficacy of COCs in the management of acne.^{98-101,221,224-226} It is evident from these trials that COCs reduce acne—both inflammatory and comedonal lesion counts. It is more difficult to determine which, if any, COC is consistently superior in the treatment of acne. A 2012 Cochrane metaanalysis assessed the effect of birth control pills on acne in women and included 31 trials with a total of 12,579 women. Nine trials compared a COC to placebo, and all of these COCs worked well to reduce acne. The progestins included in these 9 trials were levonorgestrel, norethindrone acetate, norgestimate, drospirenone,

dienogest, and chlormadinone acetate. Seventeen trials compared 2 COCs, but no consistent differences in acne reduction were appreciated based on formulation or dosage of the COC. Only 1 small study compared a COC to an oral antibiotic; no significant difference in self-assessed acne improvement was identified.²²¹

A recent publication evaluated the effectiveness of drospirenone 3 mg/ethinyl estradiol 20 µg in the treatment of moderate truncal AV. The COC showed significant reductions in inflammatory, noninflammatory, and total acne lesions compared to placebo.²²⁷

The risks of COCs must be weighed against the risks of the condition that they are treating or preventing. When COCs are used for contraception, their risks must be compared to the risks of pregnancy. If COCs are used exclusively for acne, their risks must be compared to the risks of acne. It is important to remember that FDA approval of all COCs for acne specifies that they are approved for the treatment of acne in women who also desire contraception.

COC use is associated with cardiovascular risks. Venous thromboembolic events (VTEs) have been the center of an ongoing debate regarding COCs. Traditionally, higher doses of ethinyl estradiol have been linked to increased risks of VTE. However, in recent years, some progestins have been implicated as risk factors for VTE. A recent Cochrane metaanalysis evaluated 25 publications reporting on 26 studies focused on oral contraceptives and venous thrombosis. The analysis concluded that all COC use increases the risk of VTE compared to nonusers. The relative risk of venous thrombosis for COCs with 30 to 35 µg of ethinyl estradiol and gestodene, desogestrel, cyproterone acetate, or drospirenone was similar and about 50% to 80% higher than for COCs with levonorgestrel.²²⁸ To put this increased risk into perspective, it is important to note that the baseline risk of VTE in nonpregnant, nonusers of COCs is 1 to 5 per 10,000 woman-years. Users of COCs have a VTE risk of 3 to 9 per 10,000 woman-years. Users of drospirenone-containing COCs have a VTE risk of about 10 per 10,000 woman-years. Pregnant women have a VTE risk between 5 and 20 per 10,000 woman-years, and women within 12 weeks postpartum have a VTE risk of between 40 and 65 per 10,000 woman-years.^{229,230}

Myocardial infarction (MI) risks are also increased in COC users. This risk is strongly associated with cigarette smoking and other risk factors, such as diabetes mellitus and hypertension. The WHO reports that COCs are not associated with an increased risk of MI in healthy, normotensive, nondiabetic, nonsmokers at any age.²³¹ There is also an increased risk of both ischemic and hemorrhagic stroke in COC users. Cigarette smoking and hypertension contribute to this increased risk, as do higher doses of ethinyl estradiol and age >35 years. While these are serious potential adverse events, these cardiovascular events are uncommon in women of reproductive age. An increased relative risk still translates to an overall low absolute risk.^{219,222,232}

COC use may be associated with an increased risk of breast cancer in some women. A large metaanalysis including data from 53,297 women with breast cancer and 100,239 controls showed an increased risk of breast cancer in current users of COCs. The relative risk of breast cancer in current COC users was 1.24 (95% confidence interval [CI], 1.15-1.33). This increased risk disappeared 10 years after COC discontinuation. Age at first use of a COC was the only factor that was associated with an overall increased risk. Risk did not appear to correlate with the duration of use of the COC or family history of breast cancer.²³³ A more recent systematic review of cancer risks associated with oral



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contraceptive use also showed an increased relative risk (1.08; 95% CI, 1.00-1.17) of breast cancer in COC users, and higher risk was associated with more recent use of a COC.²³⁴ Notably, this increased risk of breast cancer is greatest in women <34 years of age, when the overall incidence of breast cancer is at its lowest.²³³

The risk of cervical cancer may be increased in women who use COCs. An analysis of 24 observational studies found that the risk of cervical cancer increases with an increased duration of COC use. The risk declines after the COC is discontinued and the increase in risk disappears after 10 years of nonuse.²³⁵ Another systematic review found no significant increase in the risk of cervical cancer among ever-users of COCs and never-users from 9 pooled studies. This study did show an increased risk of cervical cancer in women with >5 years of COC use compared with never-users, but the difference was not statistically significant.²³⁴

There is additional concern regarding COC use in younger adolescent populations given the adverse effects of low estrogen on bone mass. Peak bone mass development occurs during adolescence and young adulthood. The addition of low-dose estrogen COCs early in the teen years may undermine the accrual of bone mass.²³⁶ Osteopenia or decreased bone mineral density with COC use has not been shown.^{237,238} However, definitive conclusions are yet to be made. In general, the use of COC for acne should be avoided within 2 years of first starting menses or in patients who are <14 years of age unless it is clinically warranted. The FDA has approved COC use for females 14 years (eg, drospirenone and desloratadine/levomefolate) or 15 years (eg, norgestimate and norethindrone/ferrous fumarate) and older (and desiring use of a COC as mentioned above).

There are many noncontraceptive benefits of COCs in addition to the improvement of acne. These include regulation of the menstrual cycle, lessening of menorrhagia and associated anemia, and a reduction in the formation of benign ovarian tumors. Decreased risks of colorectal, ovarian, and endometrial cancers have been shown in COC users.^{222,239}

Oral contraceptives may improve acne for many women. They may be used alone or in combination with other acne treatments. While some women present with signs or symptoms suggestive of a hormonally induced worsening of acne (ie, premenstrual flares or hirsutism), the use of COCs is not limited to these individuals. Any woman with signs or symptoms of hyperandrogenism should be evaluated appropriately for an underlying cause. However, COCs may be beneficial to women with clinical and laboratory findings of hyperandrogenism and in women without these findings.

COCs may be included as part of a comprehensive acne treatment regimen. Women who desire contraception or who suffer from menorrhagia may choose to begin a COC early in their acne treatment. In other women, COCs may be added to a treatment regimen when results with other agents have been limited. COCs may be used in combination with other oral acne medications, including the tetracycline class of antibiotics and spironolactone. There is much misunderstanding regarding the concomitant use of oral antibiotics and COCs and putative contraceptive failure. Rifampin and griseofulvin are the only antiinfectives that interact with COCs, lessening their effectiveness.²⁴⁰ The tetracycline class of antibiotics has not been shown to reduce the effectiveness of COCs when taken concomitantly.^{222,241,242}

Because the progestin drospirenone is an analog of spironolactone, there has been some concern that using a drospirenone-containing COC and spironolactone together might increase the risk of hyperkalemia. In 1 study, 27 women with acne were treated with a COC containing drospirenone 3 mg and ethynodiol diacetate 30 µg and spironolactone 100 mg each day. There were no significant elevations of serum potassium and there were no additional side effects significant enough to discontinue treatment.²⁴³

Acne reduction with COC use takes time. Randomized controlled trials consistently show a statistically significant improvement in acne with COCs compared to placebo by the end of cycle 3.^{98-101,224,225} Those treated with COCs for acne should be educated that acne reduction may not be appreciated for the first few months of treatment. Therefore, combining COCs with other acne medications early in treatment may be appropriate.

A Papanicolaou smear and a bimanual pelvic examination are no longer deemed mandatory before initiating the use of a COC. While these screening examinations may offer valuable information, they do not identify women who should not take a COC and should not be required before initiating treatment with a COC. Obtaining a thorough medical history and a blood pressure measurement are important before prescribing a COC.²⁴⁴ Proper patient selection is imperative to minimize risks associated with COC use. The WHO has published contraindications for the use of COCs.²⁴⁵

Spironolactone is an aldosterone receptor antagonist that exhibits potent antiandrogen activity by decreasing testosterone production and by competitively inhibiting binding of testosterone and dihydrotestosterone to androgen receptors in the skin.²⁴⁶⁻²⁴⁹ It may also inhibit 5-alfa-reductase and increase steroid hormone-binding globulin.^{250,251} Its use as an antiandrogen is not approved by the FDA for the treatment of acne. Two small, placebo-controlled prospective studies showed statistically significant improvement in acne severity and sebum production at doses ranging from 50 to 200 mg daily.^{252,253} A retrospective chart review of 85 patients treated with spironolactone 50 to 100 mg daily, either as monotherapy or as adjunctive therapy, revealed that 66% of women were clear or markedly improved with favorable tolerability at these lower doses.¹⁰² More recently, a Japanese study investigated the efficacy of spironolactone in Asian patients. One hundred thirty-nine Japanese patients (116 women and 23 men) were treated with spironolactone 200 mg daily for 8 weeks, followed by a taper of 50 mg every 4 weeks over a total of 20 weeks. All 64 women who completed the study had clinical improvement ranging from good to excellent. The study was discontinued prematurely in the male patients because of the development of gynecomastia.¹⁰³ Given the small number and size of available studies, a recent Cochrane database review concluded that there are insufficient data to support the efficacy of spironolactone in the treatment of acne.²⁵⁴ Despite the lack of published data, relying on available evidence, experience, and expert opinion, the work group supports the use of spironolactone in the management of acne in select women.

Spironolactone is well tolerated overall, and its side effects are dose-related. Common side effects include diuresis (29%), menstrual irregularities (22%), breast tenderness (17%), breast enlargement, fatigue, headache, and dizziness.²⁵⁵ Spironolactone is also pregnancy category C; animal studies have shown feminization of a male fetus early in gestation. Therefore, concomitant use of a COC is often recommended to both regulate menses and prevent pregnancy in many patients. Hyperkalemia is a potentially serious side effect that, fortunately, is rare in young healthy individuals with normal



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hepatic, adrenal, and renal function. Non-clinically relevant elevations may occur in about 13.7% of patients.²²⁸ A recent retrospective database review identifying 967 women between 18 and 45 years of age taking spironolactone 50 to 200 mg daily for acne found that only 0.75% of the 1723 associated potassium measurements exceeded 5.0 mmol/L. Six of the 13 abnormal tests were normal upon repeat testing. Patients with renal or cardiovascular disease and those taking angiotensin-converting enzyme inhibitors and angiotensin receptor blockers were excluded. Based on these findings, the authors concluded that testing for potassium in young healthy women taking spironolactone for acne is unnecessary.²⁵⁶ Serum potassium testing is therefore not required, but should be considered in older patients and in patients who are also taking angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, nonsteroidal antiinflammatory drugs, and digoxin. Measurements should be performed at baseline, during therapy, and after dose increases in these patients. Patients should also be educated about avoiding foods that are high in dietary potassium, such as low-sodium processed foods and coconut water.²⁵⁷ Spironolactone may also be used safely with drospirenone-containing COCs. No elevations in serum potassium were identified in a series of 27 patients treated with spironolactone 100 mg daily in combination with ethinyl estradiol 30 µg/drospirenone 3 mg.²⁴³

Animal studies using up to 150 times human doses of spironolactone or its metabolite found the development of thyroid, hepatic, testicular, and breast adenomas, as well as thyroid carcinoma and myelocytic leukemia. These findings contributed to a black box warning stating that the off-label and unnecessary use of spironolactone should be avoided. To date, there has been only 1 human report suggesting carcinogenicity in which the authors identified 5 hospitalized patients with breast cancer who were taking spironolactone among other medications²⁵⁸; however, subsequent longitudinal and retrospective studies found no association.^{255,259,260} In addition, a recent large retrospective matched cohort study of 1.29 million women >55 years of age found no association between spironolactone use and breast cancer with 8.4 million patient-years of use, further disproving any causal relationship.²⁶¹ These findings were supported by another large retrospective cohort study of 2.3 million women representing 28.8 million person-years that showed no association between spironolactone use and the development of breast, uterine, cervical, or ovarian cancers.²⁶²

Flutamide is a nonsteroidal selective androgen receptor blocker used in the treatment of prostate cancer. It is not approved by the FDA for use in acne. Doses ranging from 250 mg twice daily to as little as 62.5 mg daily have shown efficacy in the treatment of acne in small prospective trials.^{104,263-267} Flutamide 250 mg twice daily combined with a triphasic COC reduced acne by 80% compared with spironolactone 50 mg twice daily/COC, which reduced acne by only 50% after 3 months of therapy.²⁴⁵

Common side effects associated with flutamide include gastrointestinal distress, breast tenderness, hot flashes, headache, xerosis, and decreased libido.^{228,267} High rates of side effects among users may decrease compliance with use.¹⁰⁵ In 1 prospective randomized trial of 131 women, side effects at a dose of 125 mg daily were comparable to placebo.²⁶⁷ Importantly, flutamide use has been associated with idiosyncratic fatal hepatotoxicity, which appears to be dose- and age-related.^{268,269} Therefore, liver function tests need careful monitoring, and the risk of this serious adverse effect must be considered. Use of flutamide in the treatment of acne is discouraged except where benefit warrants the risk.



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Low-dose prednisone in doses ranging from 5 to 15 mg daily, administered alone or with high-estrogen containing COCs, has shown efficacy in the treatment of acne and seborrhea.^{106,270,271} However, long-term adverse effects of corticosteroids prohibit use as a primary therapy for acne. Prednisone in doses of 0.5 to 1 mg/kg/day is indicated for treatment of the systemic and cutaneous manifestations of acne fulminans and for treatment and prevention of isotretinoin-induced acne fulminans-like eruptions. A slow taper over several months is recommended while transitioning to isotretinoin or oral antibiotics in order to minimize relapses.^{272,273}

Isotretinoin

Oral isotretinoin, an isomer of retinoic acid, has been used in the United States for the treatment of acne for >30 years and is approved by the FDA for the treatment of severe recalcitrant AV. Its use has proven successful for most patients with severe acne, resulting in decreased sebum production, acne lesions, and acne scarring, along with a decrease in symptoms of anxiety and depression.^{107-117,274-277} It has also been effectively used in the treatment of moderate acne that is either treatment-resistant or that relapses quickly after the discontinuation of oral antibiotic therapy.^{32,134-138} It is the consensus of the current working group that the presence of moderate acne that is either treatment-resistant, or that produces physical scarring or significant psychosocial distress, is an indication for treatment with oral isotretinoin. Recommendations for isotretinoin are shown in [Table IX](#), and the strength of recommendations for treatment of acne with isotretinoin therapy is shown in [Table III](#). Prescribing information for the treatment of acne with isotretinoin is listed in [Supplementary Table XXIX](#).

Oral isotretinoin is recommended for the treatment of severe nodular acne

Oral isotretinoin is appropriate for the treatment of moderate acne that is treatment-resistant or for the management of acne that is producing physical scarring or psychosocial distress

Low-dose isotretinoin can be used to effectively treat acne and reduce the frequency and severity of medication-related side effects. Intermittent dosing of isotretinoin is not recommended

Routine monitoring of liver function tests, serum cholesterol, and triglycerides at baseline and again until response to treatment is established is recommended. Routine monitoring of complete blood count is not recommended

All patients treated with isotretinoin must adhere to the iPLEDGE risk management program

Females of child-bearing potential taking isotretinoin should be counseled regarding various contraceptive methods including user-independent forms

Table IX

Recommendations for isotretinoin

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When used for severe AV, isotretinoin is commonly initiated at a starting dose 0.5 mg/kg/day for the first month, then increased to 1.0 mg/kg/day thereafter as tolerated by the patient.¹¹⁸ In extremely severe cases, even lower starting doses, with or without the concomitant use of oral steroids, may be needed. In earlier studies of optimal dosing of isotretinoin in patients with severe AV, doses ranging from 0.1 mg/kg/day to 1.0 mg/kg/day were most commonly used. Some efficacy was generally seen at all doses, along with a dose-dependent decrease in sebum production.¹⁰⁹ While there was not a significant difference in the improvement of acne by the end of the treatment course between doses of 0.5 and 1.0 mg/kg/day in most of the studies, there was a significant difference in relapse rates and the need for retreatment; patients treated with approximately 1.0 mg/kg/day had a significantly lower relapse rate and a lower rate of retreatment with isotretinoin than those treated with 0.5 mg/kg/day.^{110,112,116} Similarly, a lower relapse rate was seen for those treated with a cumulative dose of >120 mg/kg compared to those treated with <120 mg/kg.^{110,119} It has been suggested that this dose-dependent therapeutic benefit plateaus beyond 150 mg/kg.¹¹⁹ Therefore, in patients with severe AV, the work group supports initiation of isotretinoin at 0.5 mg/kg/day when appropriate, subsequently increasing to a full dose of 1 mg/kg/day after the first month as tolerated, with a goal cumulative dose between 120 and 150 mg/kg. One recent study of 116 patients found that a cumulative dose of 220 mg/kg or more may result in lower relapse rates, but confirmation will require study in larger populations.²⁷⁸

Isotretinoin treatment has been studied in patients with treatment-resistant or quick-relapsing, moderate AV. In this patient population, multiple studies have found that low-dose isotretinoin (0.25–0.4 mg/kg/day) is effective in the treatment of acne, and that this efficacy is comparable to the use of more conventional dosing.^{32,107,134,135,279} This may also be true for a low cumulative dose regimen.¹³⁶ In addition, low-dose regimens are associated with a decreased rate of medication-related adverse effects, thereby leading to improved tolerability and increased patient satisfaction.^{107,134,135,279,280} Unlike in patients with severe acne, relapse rates in patients with moderate acne treated with low-dose isotretinoin are equal to relapse rates in those treated with conventional dosing.^{32,279} Intermittent dosing, however, is not as effective and is associated with higher relapse rates; therefore, it is not recommended.^{32,134,135,276}

Isotretinoin is highly lipophilic and is best absorbed when taken with food.^{115,120,121} Patients should be instructed to take isotretinoin with meals. One formulation, isotretinoin with lidose, uses lipid agents to encase the medication, bypassing the need for food, and can be taken on an empty stomach.¹²¹

Common adverse effects associated with the intake of isotretinoin have been well documented and reviewed in previous guidelines.¹ The most prevalent side effects involve the mucocutaneous, musculoskeletal, and ophthalmic systems, generally mimicking symptoms of hypervitaminosis A. With standard courses, these side effects are temporary and resolve without sequelae after discontinuation of the drug. Other real and speculative adverse effects of interest include inflammatory bowel disease, depression/anxiety/mood changes, cardiovascular risk factors, bone mineralization, concerns regarding scarring, and *S aureus* colonization.

Inflammatory bowel disease (IBD) consists of ulcerative colitis (UC) and Crohn disease (CD). Several retrospective analyses have been performed to determine whether there is an association between isotretinoin intake and IBD.^{122-126,281,282} While 2 studies^{123,282} have shown a potential relationship, more recent analyses^{122,125,283} suggest no association between IBD and isotretinoin ingestion. The most convincing article suggesting an association between isotretinoin and UC¹²⁴ was directly refuted by a later analysis of the same database.¹²⁵ Therefore, the work group agrees with the position statement of the American Academy of Dermatology that the “current evidence is insufficient to prove either an association or causal relationship between isotretinoin use and IBD.”²⁸⁴

Changes in mood, including depression, suicidal ideation, and suicide have been reported sporadically in patients who are taking isotretinoin.^{127,285} To date, no studies to suggest an evidence-based link between isotretinoin and depression, anxiety, mood changes, or suicidal ideation/suicide exist. Multiple studies have shown no evidence of depression from isotretinoin on a population basis.^{128-133,137,285} On the contrary, most studies have shown isotretinoin to improve or have no negative effects on mood, memory, attention, or executive functions.^{113,128-133,137,285-288} However, given the prevalence of depression, anxiety, and suicidal ideation/suicide in the general population, and especially the adolescent population who may be candidates for isotretinoin therapy, the prescribing physician should continue to monitor for these symptoms and make therapeutic decisions within the context of each individual patient.

Physicians prescribing isotretinoin need to be aware of guidelines for evidence-based monitoring of side effects. Interest in bone demineralization and premature epiphyseal closure observed with long-term oral retinoid intake led to early concerns about these issues for patients taking isotretinoin for acne. While premature epiphyseal closure has been reported in 2 isolated patients who were taking short-term isotretinoin for acne,^{289,290} these effects have not been reported in any other studies of patients taking short-term isotretinoin therapy for AV.^{119,139} It remains the opinion of this work group that routine screening for these issues is not required in patients who are taking short-term isotretinoin therapy. Serum cholesterol and triglycerides, as well as transaminases, have been known to rise in some patients taking oral isotretinoin.¹⁴⁰⁻¹⁴² While there is no proof of long-term cardiovascular risk from short-term elevation of triglycerides and cholesterol during short-term isotretinoin therapy,¹⁴⁰ the routine monitoring of serum lipid profiles and liver function studies should continue.^{139,142,291,292} This work group could find no evidence-based reason that routine monitoring of complete blood cell counts is warranted.

Several early case series described delayed wound healing or keloid formation in patients who were taking or had recently taken isotretinoin, leading to the current recommendation to delay procedures such as dermabrasion or laser resurfacing until 6 to 12 months after discontinuing isotretinoin.^{293,294} Recent prospective small interventional studies did not find atypical scarring with chemical peels or manual dermabrasion in any patient currently or recently on isotretinoin.^{291,294} There are also retrospective studies and case reports demonstrating safety with laser hair removal, pulsed dye laser, and CO₂ laser.²⁹⁵⁻²⁹⁹ While elective procedures should be delayed for 6 to 12 months when possible, careful consideration may be given on a case by case basis.

Higher rates of colonization with *S aureus* have been seen in patients taking systemic isotretinoin,
 Download PDF leading to increased rates of minor skin infections, such as folliculitis and furunculosis.^{300,301} On rare

occasions, the combination of cheilitis and *S aureus* colonization can cause lip or perioral abscesses, a serious complication requiring prompt attention.³⁰⁰

The teratogenic effects of isotretinoin and the risk for retinoic acid embryopathy are well known. After introduction of isotretinoin in the United States in 1982, there were hundreds of reports of isotretinoin-exposed pregnancies within just several years, resulting in a high rate of congenital malformations.³⁰² Because of this, the first risk management program was implemented. iPLEDGE is now the third risk management program that has been put in place in an effort to prevent isotretinoin exposure during pregnancy. As mandated by the FDA, all patients receiving isotretinoin—both men and women—are required to enroll in and adhere to the iPLEDGE risk management program. Despite this, fetal exposure has not significantly decreased since the implementation of iPLEDGE, and approximately 150 isotretinoin-exposed pregnancies still occur in the United States each year^{143,144} because of noncompliance with the iPLEDGE contraceptive requirements to abstain from sex or to use 2 contraceptive methods. Nearly one-third of all women of childbearing potential in a recent US study admitted noncompliance with iPLEDGE pregnancy prevention requirements; of those that were sexually active, 29% did not comply with the use of condoms that they had agreed to use as 1 of their methods, and 39% missed ≥1 contraceptive pills in the previous month.¹⁴⁴ Therefore, every woman of child-bearing potential taking isotretinoin should be carefully counseled regarding various contraceptive methods that are available and the specific requirements of the iPLEDGE system at each clinic visit. Patient-independent forms of birth control, including long-acting reversible contraceptives, should be considered whenever appropriate.

Miscellaneous therapies/physical modalities

There is limited evidence published in the peer-reviewed medical literature that addresses the efficacy of comedo removal for the treatment of acne despite its long-standing clinical use. It is, however, the opinion of the work group that comedo removal is often helpful in the management of comedones that are resistant to other therapies. Recommendations for miscellaneous therapies and physical modalities are listed in [Table X](#), and the strength of recommendations for treatment of acne using miscellaneous therapies and physical modalities is shown in [Table III](#). Prescribing information for miscellaneous therapies and physical modalities is located in [Supplementary Tables XXX-XXXIII](#).

There is limited evidence to recommend the use and benefit of physical modalities for the routine treatment of acne, including pulsed dye laser, glycolic acid peels, and salicylic acid peels

Intralesional corticosteroid injections are effective in the treatment of individual acne nodules

Table X

Recommendations for miscellaneous therapies and physical modalities

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Studies exist suggesting that chemical peels may improve acne. However, large, multicenter, double-blinded control trials comparing peels to placebo and comparing different peels are lacking. Glycolic acid and salicylic acid chemical peels may be helpful for noninflammatory (comedonal) lesions.^{145-147,303,304} However, multiple treatments are needed and the results are not long-lasting. In the opinion of the work group, chemical peels may result in mild improvement in comedonal acne.

Some laser and light devices may be beneficial for acne, but additional studies are needed. Studies exist evaluating the use of many lasers, including pulsed dye laser, potassium titanyl phosphate (KTP) laser, fractionated and nonfractionated infrared lasers, and the fractionated CO₂ laser. Light devices aside from lasers have also been investigated, including radiofrequency, intense pulsed light, photopneumatic therapy, and photodynamic therapy (PDT).

Of all laser and light devices, the most evidence exists for PDT in treating acne.³⁰⁵⁻³⁰⁸ With PDT, a photosensitizer, such as aminolevulinic acid, is first applied to the affected skin for a period of time (varying from 15 minutes to 3 hours). The photosensitizer is then absorbed into the pilosebaceous units and is preferentially taken up by sebocytes. A laser or light device is then used to activate the photosensitizer, generating singlet oxygen species, and thereby damaging the sebaceous glands and reducing *P acnes*. This treatment shows great promise, but additional studies are needed to determine the optimal photosensitizer, incubation time, and light source.

Intralesional injection of triamcinolone acetonide is a commonly used technique for the management of larger, nodular lesions in patients with acne.^{148,149} Rapid improvement and decreased pain are noted. Local atrophy, systemic absorption of steroids, and possible adrenal suppression may occur.³⁰⁹ Decreasing the concentration and the volume of steroid used will minimize these complications.

Complementary/alternative therapies

Two clinical trials have shown that topical tea tree oil is effective for the treatment of acne.^{150,151} In 1 study, it was comparable to BP but better tolerated. Other herbal agents, such as topical and oral ayurvedic compounds, oral barberry extract, and gluconolactone solution have been reported to have value in the treatment of acne.¹⁵²⁻¹⁵⁵

The psychological effects of acne may be profound, and it is the opinion of the expert work group that effective acne treatment can improve the emotional outlook of patients. There is weak evidence of the possible benefit of biofeedback-assisted relaxation and cognitive imagery.^{156,310}

The recommendation for using complementary and alternative therapies is listed in **Table XI**, and the strength of recommendation for treatment of acne using complementary and alternative therapies is shown in **Table III**.

Herbal and alternative therapies have been used to treat acne. Although most of these products appear to be well tolerated, limited data exist regarding the safety and efficacy of these agents to recommend their use in acne



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Table XI

Recommendation for complementary/alternative therapies

[Open table in a new tab](#)

Role of diet in acne

Emerging evidence suggests that high glycemic index diets may be associated with acne. In 2007, a randomized controlled trial with 23 Australian males 15 to 25 years of age examined the impact of a low glycemic diet on acne. Those randomized to follow the low glycemic load (LGL) diet had significant improvement in acne severity, a significant reduction in weight and body mass index (BMI), a significant decrease in free androgen index, and improved insulin sensitivity at the end of 12 weeks.¹⁵⁷ The study was limited by its small sample size and the fact that both groups lost weight. In 2012, a 10-week randomized controlled trial was conducted in 32 Korean subjects (24 men and 8 women) 20 to 27 years of age. Those randomized to the LGL diet had a statistically significant improvement in acne severity and no change in weight and BMI. Histologic analyses were conducted, and the authors found that the size of the sebaceous glands were significantly reduced in the LGL group, whereas hematoxylin–eosin stains revealed a decrease in inflammatory cells and additional stains showed a decrease in inflammatory cytokines.¹⁵⁸ Although these 2 studies are the most rigorous to date analyzing the effect of glycemic index diets on acne, a small number of studies further support this association.^{159–161,311}

While no randomized controlled trials have been conducted to examine the role of dairy consumption and acne, several observational studies suggest that certain dairy products, especially skim milk, may aggravate acne. In 2005, a retrospective study analyzed data from 47,355 adult women who were asked to recall their high school diet. They were also asked to recall if they had “physician-diagnosed acne.” In this study, acne was positively associated with the reported quantity of milk ingestion. The strongest association was noted with skim milk. Specifically, women who consumed ≥2 glasses of skim milk a day had a 44% increased risk of reporting acne.³¹² This study was heavily criticized for its retrospective design, so the same research group conducted 2 follow-up, prospective studies. The first was conducted on a cohort of girls, and found that acne was associated with total milk intake, whole milk, low-fat milk, and skim milk.¹⁶² The second study focused on boys only, and found that acne was associated with the intake of skim milk only.¹⁶³ More recently, a case control study involving 88 Malaysian subjects 18 to 30 years of age found that the frequency of milk and ice cream consumption was significantly higher in patients with acne compared to controls.¹⁶¹ Dermatologist-assessed subjects who consumed milk or ice cream ≥1 time per week had a 4-fold increased risk of having acne. No association was found with cheese or yogurt. Also in 2012, another case control study involving 563 Italian subjects 10 to 24 years of age found that the risk of acne was also increased with milk consumption.¹⁶⁴ The association was more marked with skim milk, and again no association was seen with cheese or yogurt.

Although some small preliminary studies have examined the role of antioxidants (including oral zinc³¹³), probiotics,³¹⁴ and fish oil³¹⁵ on acne, the existing evidence is not strong enough to support any



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recommendations regarding these dietary factors at this time. Recommendations for the role of diet in acne are listed in [Table XII](#), and the strength of recommendations for the role of diet in acne is shown in [Table III](#).

Given the current data, no specific dietary changes are recommended in the management of acne

Emerging data suggest that high glycemic index diets may be associated with acne

Limited evidence suggests that some dairy, particularly skim milk, may influence acne

Table XII

Recommendations for the role of diet in acne

[Open table in a new tab](#)

Gaps in research/knowledge

We have described above the significant progress that has been made in understanding the pathogenesis and treatment of acne, but there are still large gaps in our knowledge base. In [Table XIII](#), we address some of the most important current gaps in research.

Topics	Identified research gaps
General	Treatment of acne in persons of color Treatment of acne in pregnant women
Pathogenesis	Molecular and cellular mechanisms underlying acne Molecular description of postinflammatory hyperpigmentation Pathophysiology of acne scar, both atrophic and hypertrophic types Immunopathogenesis of acne
Grading and classification	Develop assessment tools that better help characterize acne in the office Develop and validate patient-reported outcome measures for assessing acne treatment in office/clinic
Topical therapies	Efficacy, safety, and side effect profile of topical therapies in children 8-12 years of age <small>Data on aspects of care that promote compliance in selected populations using topical therapy</small>

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Table XIII

Research and knowledge gaps in acne

COC, Combined oral contraceptive; PCOS, polycystic ovarian syndrome.

[Open table in a new tab](#)

Acknowledgments

We are grateful to the AAD Board of Directors, the Council on Science and Research, and the Clinical Research Committee members for reviewing the manuscripts and providing excellent suggestions. We thank Yevgeniy Balagula, MD, Cecilia Larocca, MD, Candrice R. Heat, MD, Mary-Margaret Kober, MD, Robyn Marszalek, MD, Tiffany Mayo, MD, Jean McGee, MD, Joanne Smucker, MD, and Erin Wei for their assistance in developing the evidence tables. We also thank Tammi Matillano, Mary Bodach, MLIS, Darlene Jones, and Charniel McDaniels, MS, for their technical assistance in preparing the manuscript.

The AAD strives to produce clinical guidelines that reflect the best available evidence supplemented with the judgment of expert clinicians. Significant efforts are taken to minimize the potential for conflicts of interest to influence guideline content. Funding of guideline production by medical or pharmaceutical entities is prohibited, full disclosure is obtained and evaluated for all guideline contributors throughout the guideline development process, and recusal is used to manage identified relationships. The management of conflict of interest for this guideline complies with the Council of Medical Specialty Societies' Code of Interactions with Companies. The AAD conflict of interest policy summary may be viewed at www.aad.org.

Hilary E. Baldwin, MD, served on the Advisory Board of Allergan Inc, receiving honoraria. Dr Baldwin also served as a speaker for Galderma Laboratories, GlaxoSmithKline, Ranbaxy Laboratories Ltd, and Valeant Pharmaceutical International, receiving honoraria. Diane S. Berson, MD, served on the Advisory Board of Galderma Laboratories, La-Roche-Posay Laboratoire Pharmaceutique, and Medicis Pharmaceutical Corporation, receiving honoraria. Dr Berson also served as a consultant for Procter & Gamble, receiving honoraria. Whitney Bowe, MD, served on the Advisory Board of Allergan, Inc, Galderma Laboratories, and Johnson & Johnson Consumer Products Company, receiving honoraria. Dr Bowe also served as a speaker for Bayer and consultant for Galderma Laboratories, Johnson & Johnson Consumer Products Company, L'Oréal USA Inc, Onset Therapeutics, and Procter & Gamble, receiving honoraria. Dr Bowe also received honoraria from Energizer Holdings, Inc. Julie C. Harper, MD, served as speaker for Allergan, Inc, Coria Laboratories, Galderma USA, La-Roche-Posay Laboratoire Pharmaceutique, Promius Pharma, LLC, and Valeant Pharmaceutical North America, receiving honoraria. Dr Harper served on the Advisory Board for Stiefel, receiving honoraria. Dr Harper served as consultant to Galderma Laboratories and Stiefel, receiving honoraria. Dr Harper also received other honoraria from Bayer Pharmaceuticals. Sewon Kang, MD, served on the Advisory Board for Dermira, receiving stock options, and the Advisory Board for Galderma Laboratories, Pfizer, Inc, and Unilever Home & Personal Care USA, receiving honoraria. Jonette E. Keri, MD, PhD, served on the Advisory Board for Suneva Medical, Inc, receiving honoraria. Dr Keri also served as consultant for



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F. Hoffmann-La Roche AG, receiving honoraria. James J. Leyden, MD, served as consultant for Allergan, Inc, Anacor Pharmaceuticals Inc, Cipher Pharmaceuticals, Combe Inc, Galderma Laboratories, Medicis Pharmaceutical Corporation, Obagi Medical Products, and Unilever Home & Personal Care USA, receiving honoraria. Rachel V. Reynolds, MD, served as consultant for Biosense Webster and Medtronics. Nanette Silverberg, MD, served on the Advisory Board for Leo Pharma, Inc, receiving honoraria. Dr Silverberg also served as consultant for Johnson & Johnson Consumer Products Company, receiving honoraria. Linda F. Stein Gold, MD, served on the Advisory Board for AbbVie, Galderma Laboratories, LEO Pharma, US, Lilly ICOS LLC, Medicis Pharmaceutical Corporation, Pfizer Inc, Stiefel, Taro Pharm, Valeant Pharmaceuticals International, and Warner Chilcott, receiving honoraria. Dr Stein Gold also served as speaker for Actavis and Warner Chilcott and consultant for Ferndale Laboratories, receiving honoraria. Dr Stein Gold also received other honoraria from Roche Laboratories. Jonathan S. Weiss, MD, served on the Advisory Board for Galderma Laboratories and Valeant Pharmaceuticals International, receiving honoraria. Dr Weiss also served as consultant for Abbott Laboratories, Celgene Corporation, LEO Pharma, and Sebcaia, Inc, receiving honoraria. Andrea L. Zaenglein, MD, served on the Advisory Board for Anacor Pharmaceuticals, Galderma Laboratories, Promius Pharmaceuticals, and Valeant Pharmaceuticals International, receiving honoraria. Dr Zaenglein also served as consultant for Ranbaxy Laboratories Limited, receiving honoraria. Arun L. Pathy, MD, Ali Alikhan, MD, Emmy M. Gruber, MD, Bethanee J. Schlosser, MD, PhD, Megha M. Tollefson, MD, Nancy Dolan, MD, Andy Sagan, MD, Mackenzie Stern, Kevin M. Boyer, MPH, and Reva Bhushan, MA, PhD, have no relevant relationships to disclose.

Appendix

Indication	Topical treatment of mild to moderate acne vulgaris
Dosing	2.5%, 5%, or 10% in gel, wash, or cream
Duration of dosing	Continuing use of the drug is normally required to maintain a satisfactory clinical response
Contraindications	Should not be used in patients who have shown hypersensitivity to benzoyl peroxide or to any of the other ingredients in the products
Efficacy	Clinically visible improvements will normally occur by the third week of therapy. Maximum lesion reduction may be expected after approximately 8 to 12 weeks of drug use
Adverse effects/toxicities	Hypersensitivity reactions, contact sensitization reactions, excessive erythema, and peeling



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Supplemental Table I

Prescribing information for benzoyl peroxide

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Indication	Salicylic acid is used alone or in combination with other drugs for the symptomatic treatment of acne
Dosing	Apply topically using appropriate preparations containing salicylic acid 0.5-2%
Duration of dosing	Apply appropriate 0.5-2% salicylic acid preparation 1-3 times daily. Initially, apply once daily then gradually increase to 2 or 3 times daily, if necessary. If dryness or peeling occurs, reduce application to once daily or every other day
Contraindications	Known sensitivity to salicylic acid or any other ingredient in the formulation
Adverse effects/toxicities	Hypersensitivity reactions, salicylate toxicity, excessive erythema, and scaling
Interactions	Acidifying agents, anticoagulants, antidiabetic agents, aspirin, corticosteroids, diuretics, methotrexate, pyrazinamide, sulfur, and uricosuric agents

Supplemental Table II

Prescribing information for salicylic acid

[Open table in a new tab](#)

Indication	Topical treatment of acne vulgaris
Dosing	Apply 2% solution, ointment, pledge, or gel as a thin film to affected area once or twice daily
Duration of dosing	Maintenance therapy needed to prevent recurrence
Contraindications	Known hypersensitivity to erythromycin or any ingredient in the formulation

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Efficacy	Generally effective for the treatment of mild to moderate inflammatory acne. Main action is prevention of new lesions
Other results	May induce bacterial resistance when used as monotherapy; resistance associated with decreased clinical efficacy

Supplemental Table III

Prescribing information for erythromycin (topical)

[Open table in a new tab](#)

Indication	Topical treatment of acne vulgaris
Dosing	Applied twice daily, morning and evening, after the skin is thoroughly washed, rinsed with warm water, and gently patted dry
Contraindications	Individuals who have shown hypersensitivity to any components of formulation. Concomitant topical acne therapy should be used with caution because a possible cumulative irritancy effect may occur, especially with the use of peeling, desquamating, or abrasive agents
Efficacy	In 2 controlled clinical studies, the combination of erythromycin and benzoyl peroxide applied twice daily for 8 weeks was significantly more effective than vehicle
Adverse effects/toxicities	Pseudomembranous colitis, dryness, urticarial reaction, peeling, itching, burning sensation, erythema, inflammation of the face/eyes/nose, skin discoloration, oiliness, and tenderness of skin
Other issues	Cumulative irritant or drying effect: use with caution

Supplemental Table IV

Prescribing information for combination erythromycin and benzoyl peroxide

[Open table in a new tab](#)

Indication	Topical application in the treatment of acne vulgaris
Dosing	Apply a thin film of clindamycin once daily to the skin where acne lesions appear. Use enough  to cover the entire affected area lightly

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Contraindications	History of hypersensitivity to preparations containing clindamycin or lincomycin, a history of regional enteritis or ulcerative colitis, or a history of antibiotic associated colitis
Efficacy	In a 12-week controlled clinical trial, 1% topical clindamycin gel applied once daily was more effective than the vehicle applied once daily
Adverse effects/toxicities	Severe colitis, dermatitis, folliculitis, photosensitivity reaction, pruritus, erythema, dry skin, and peeling
Interactions	Clindamycin has been shown to have neuromuscular blocking properties that may

Supplemental Table V

Prescribing information for clindamycin

[Open table in a new tab](#)

Indication	Topical treatment of inflammatory acne vulgaris
Dosing	Apply a thin layer to the face once daily, in the evening
Contraindications	Patients who have had hypersensitivity (eg, anaphylaxis) to clindamycin, benzoyl peroxide, any components of the formulation, or lincomycin. Patients with a history of regional enteritis, ulcerative colitis, or antibiotic-associated colitis (including pseudomembranous colitis)
Efficacy	Combined clindamycin plus benzoyl peroxide topically applied once daily for 11 weeks was significantly more effective than vehicle, benzoyl peroxide, and clindamycin in the treatment of inflammatory lesions of moderate to moderately severe facial acne vulgaris in 3 of 5 trials
Other results	Has not been shown to have any additional benefit when compared with benzoyl peroxide alone in the same vehicle when used for the treatment of noninflammatory acne

Supplemental Table VI

Prescribing information for combination clindamycin + benzoyl peroxide

[Open table in a new tab](#)



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Indication	Topical treatment of acne vulgaris
Dosing	Apply a thin layer of tretinoin once daily, before bedtime, to skin where lesions occur. Keep away from eyes, mouth, nasal creases, and mucous membranes
Contraindications	Known hypersensitivity to tretinoin or any ingredient in the formulation
Efficacy	In controlled trials, 21-23% of patients using topical tretinoin had successful treatment (using 6-point global severity score)
Adverse effects/toxicities	Dry skin, peeling, scaling, flaking, burning sensation, erythema, pruritus, pain of skin, sunburn, and hyper-/hypopigmentation
Interactions	Keratolytic agents and photosensitizing agents
Other issues	Ultraviolet light and environmental exposures (eg, wind and cold) can cause irritation and should be avoided; cautions should be used in patients with fish

Supplemental Table VII

Prescribing information for tretinoin

[Open table in a new tab](#)

Indication	Topical treatment of acne vulgaris in patients ≥ 12 years of age
Dosing	Apply a pea-sized amount to the entire face once daily at bedtime.
Contraindications	Patients with regional enteritis, ulcerative colitis, or history of antibiotic-associated colitis.
Efficacy	In clinical trials, 21-41% of patients using combined clindamycin and tretinoin topically demonstrated successful treatment (using Evaluator's Global Severity score)
Adverse effects/toxicities	Erythema, scaling, itching, burning, stinging, nasopharyngitis, pharyngolaryngeal pain, dry skin, cough, and sinusitis
Interactions	Concomitant use of topical medications with a strong drying effect can increase skin irritation.  Should not be used in combination with erythromycin-containing products. Should not be used in combination with neuromuscular blocking agents

Supplemental Table VIII

Prescribing information for combination clindamycin and tretinoin

[Open table in a new tab](#)

Indication	Topical treatment of acne vulgaris in patients ≥ 12 years of age
Dosing	Apply a thin film of adapalene to the entire face and any other affected areas of the skin once daily in the evening, after washing gently with a nonmedicated soap
Contraindications	Should not be administered to individuals who are hypersensitive to adapalene or any of the components in the vehicle
Efficacy	Clinical studies show that 16% of patients applying 0.1% topical adapalene and 21% of patients applying 0.3% topical adapalene had successful treatments after 12 weeks (using Investigator's Global Assessment)
Adverse effects/toxicities	Erythema, scaling, dry skin, burning/stinging, skin discomfort, pruritus, desquamation, sunburn, allergic/hypersensitivity reactions, face/eyelid edema, lip swelling, and angioedema
	Has the potential to induce local irritation in some patients, concomitant use of other potentially irritating topical products should be approached with caution. Use

Supplemental Table IX

Prescribing information for adapalene

[Open table in a new tab](#)

Indication	Topical treatment of acne vulgaris in patients ≥ 9 years of age
Dosing	Apply a thin film to affected areas of the face or trunk once daily after washing. Use a pea-sized amount for each area of the face (eg, forehead, chin, and each cheek)
Contraindications	Known hypersensitivity to adapalene or any ingredient in the formulation
Efficacy	In clinical trials, 21-47% of patients had successful treatment (using Investigator's Global Assessment)

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Adverse effects/toxicities	Erythema, scaling, dryness, stinging/burning, contact dermatitis, skin irritation, eyelid edema, sunburn, blister, pain of skin, swelling face, conjunctivitis, skin discoloration, rash, eczema, throat tightness, and allergic contact dermatitis
Interactions	Keratolytic agents and photosensitizing agents

Supplemental Table X

Prescribing information for combination adapalene and benzoyl peroxide

[Open table in a new tab](#)

Indication	Topical treatment of acne vulgaris
Dosing	Apply a thin layer of tazarotene only to the affected area once daily in the evening
Contraindications	Pregnancy and hypersensitivity
Efficacy	Tazarotene was significantly more effective than vehicle in the treatment of facial acne vulgaris
Adverse effects/toxicities	Pruritus, burning, skin redness, peeling, desquamation, dry skin, and erythema
Interactions	Photosensitizing agents
Other issues	Avoid exposure to sunlight, sunlamps, and weather extremes

Supplemental Table XI

Prescribing information for tazarotene

[Open table in a new tab](#)

Indication	Topical treatment of mild to moderate inflammatory acne vulgaris
Dosing	A thin film should be gently but thoroughly massaged into the affected areas twice daily in the morning and evening

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Contraindications	Known hypersensitivity to azelaic acid or any of its components
Adverse effects/toxicities	Pruritus, burning, stinging, tingling, erythema, dryness, rash, peeling, irritation, dermatitis, and contact dermatitis
Pregnancy category	B
Nursing	Minimally distributed into milk after topical application. Caution if used in nursing women

Safety and effectiveness in pediatric patients ≤ 12 years of age have not been

Supplemental Table XII

Prescribing information for azelaic acid

[Open table in a new tab](#)

Indication	Topical treatment of acne vulgaris
Dosing	Apply approximately a pea-sized amount, in a thin layer to the acne affected area, twice daily
Duration of dosing	If there is no improvement after 12 weeks, treatment should be reassessed
Contraindications	None
Efficacy	In clinical trials, 35-42% of patients using topical dapson were successfully treated (using the Global Acne Assessment Score)
Adverse effects/toxicities	Oiliness, peeling, dryness, erythema, burning, pruritus, pyrexia, nasopharyngitis, upper respiratory infection, sinusitis, influenza, pharyngitis, cough, joint sprain, headache, suicide attempt, depression, psychosis, tonic clonic movements, abdominal pain, severe vomiting, and pancreatitis

Supplemental Table XIII

Prescribing information for dapson

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Indication	Adjunctive treatment in moderate to severe inflammatory acne
Dosing	Children >8 years of age: 25-50 mg/kg daily in 4 divided doses Adults: 1 g daily given in divided doses; when improvement occurs in 1-2 weeks, decrease slowly to a maintenance dosage of 125-500 mg daily
Duration of dosing	Adults: continue maintenance dosage until clinical improvement allows discontinuation of the drug.
Contraindications	Hypersensitivity to any of the tetracyclines
Adverse	Gastrointestinal: anorexia, nausea, epigastric distress, vomiting, diarrhea, glossitis, black hairy tongue, dysphagia, enterocolitis, inflammatory lesions (with Candidal overgrowth) in the anogenital region, esophagitis, or esophageal ulceration Teeth: permanent discoloration during tooth development, enamel hypoplasia Skin: maculopapular and erythematous rashes, exfoliative dermatitis, onycholysis, nail discoloration, or photosensitivity Renal: rise in blood urea nitrogen (dose-related)

Supplemental Table XIV

Prescribing information for tetracycline

[Open table in a new tab](#)

Indication	Adjunctive treatment of moderate to severe inflammatory acne
Dosing	Children >8 years of age: 4 mg/kg initially followed by 2 mg/kg every 12 hours Adults: 50 mg 1-3 times daily
Contraindications	Hypersensitivity to minocycline, any tetracycline, or any component in the preparation
	Body as a whole: fever and discoloration of secretions Gastrointestinal: anorexia, nausea, vomiting, diarrhea, dyspepsia, stomatitis, glossitis, dysphagia, enamel hypoplasia, enterocolitis, pseudomembranous colitis, pancreatitis, inflammatory lesions (with monilial overgrowth) in the oral and anogenital regions, esophagitis, and esophageal ulcerations Genitourinary: vulvovaginitis Hepatic toxicity: hyperbilirubinemia, hepatic cholestasis, increases in liver enzymes, fatal hepatic failure, and jaundice. Hepatitis, including autoimmune hepatitis, and liver failure Skin: alopecia, erythema nodosum, hyperpigmentation of nails, pruritus, toxic epidermal necrolysis, vasculitis, maculopapular and erythematous rashes, exfoliative



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Supplemental Table XV

Prescribing information for minocycline

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Indication	Adjunctive treatment in severe acne
Dosing	Children >8 years of age and <100 pounds: 2 mg/lb of body weight divided into 2 doses on the first day of treatment, followed by 1 mg/lb of body weight given as a single daily dose or divided into 2 doses, on subsequent days Adults and children >100 pounds: 200 mg on the first day of treatment (administered 100 mg every 12 hours) followed by a maintenance dose of 100 mg/day
Contraindications	Hypersensitivity to any of the tetracyclines
Adverse effects/toxicities	Gastrointestinal: anorexia, nausea, vomiting, diarrhea, glossitis, dysphagia, enterocolitis, inflammatory lesions (with monilial overgrowth) in the anogenital region, hepatotoxicity, esophagitis, or esophageal ulcerations Skin: toxic epidermal necrolysis, Stevens–Johnson syndrome, erythema multiforme, maculopapular and erythematous rashes, exfoliative dermatitis, or photosensitivity Renal: rise in blood urea nitrogen (dose-related) Hypersensitivity reactions: urticaria, angioneurotic edema, anaphylaxis, anaphylactoid purpura, serum sickness, pericarditis, or exacerbation of systemic lupus erythematosus

Supplemental Table XVI

Prescribing information for doxycycline

[Open table in a new tab](#)

Indication	Not approved by the US Food and Drug Administration for treatment of acne, use is off-label
Contraindications	Known hypersensitivity to trimethoprim or sulfonamides, history of drug-induced immune thrombocytopenia with use of trimethoprim or sulfonamides, patients with documented megaloblastic anemia caused by folate deficiency, pregnant patients and nursing mothers, pediatric patients <2 months of age, and patients with marked hepatic damage or with severe renal insufficiency when renal function status cannot be monitored

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Fatalities: Stevens–Johnson syndrome, toxic epidermal necrolysis, fulminant hepatic necrosis, agranulocytosis, aplastic anemia, and other blood dyscrasias
Hematologic: agranulocytosis, aplastic anemia, thrombocytopenia, leukopenia, neutropenia, hemolytic anemia, megaloblastic anemia, hypoprothrombinemia, methemoglobinemia, eosinophilia, thrombotic thrombocytopenia purpura, or idiopathic thrombocytopenic purpura

Supplemental Table XVII

Prescribing information for trimethoprim sulfamethoxazole

[Open table in a new tab](#)

Indication	Not approved by the US Food and Drug Administration for treatment of acne, use is off-label
Contraindications	Known hypersensitivity to trimethoprim, documented megaloblastic anemia caused by folate deficiency
Adverse effects/toxicities	Dermatologic: rash, pruritus, or phototoxic skin eruptions Hypersensitivity: exfoliative dermatitis, erythema multiforme, Stevens–Johnson syndrome, toxic epidermal necrolysis (Lyell syndrome), or anaphylaxis Gastrointestinal: epigastric distress, nausea, vomiting, glossitis, elevation of serum transaminase and bilirubin, or cholestatic jaundice Hematologic: thrombocytopenia, leukopenia, neutropenia, megaloblastic anemia, or methemoglobinemia. Metabolic: hyperkalemia, hyponatremia Neurologic: aseptic meningitis Miscellaneous: fever, increases in blood urea nitrogen and serum creatinine levels
Interactions	Dapsone, phenytoin, tests for creatinine, test for methotrexate

Supplemental Table XVIII

Prescribing information for trimethoprim

[Open table in a new tab](#)

Indication	Not approved by the US Food and Drug Administration for treatment of acne, use is off-label
Contraindications	Hypersensitivity to erythromycins, patients taking terfenadine, astemizole, pimozide, or cisapride Download PDF

Adverse effects/toxicities	Gastrointestinal: pseudomembranous colitis, nausea, vomiting, abdominal pain, diarrhea, or anorexia Liver: hepatitis, hepatic dysfunction, or abnormal liver function results Cardiovascular: QT prolongation, ventricular tachycardia, or torsades de pointes Allergic reaction: urticaria to anaphylaxis Skin reaction: mild eruptions to erythema multiforme, Stevens–Johnson syndrome, or toxic epidermal necrolysis Other: pancreatitis, convulsion, or reversible hearing loss
Interactions	Antiarrhythmic agents, oral anticoagulants, azole antifungals, benzodiazepines, calcium-channel blocking agents, carbamazepine, chloramphenicol, cisapride, clindamycin/lincomycin, ergot alkaloids, 3-hydroxy-3-methylglutaryl-coenzyme A reductase inhibitors, cyclosporine, nimozide, sildenafil, and theophylline

Supplemental Table XIX

Prescribing information for erythromycin (systemic)

[Open table in a new tab](#)

Indication	Not approved by the US Food and Drug Administration for the treatment of acne, use is off-label
Contraindications	Hypersensitivity to azithromycin, erythromycin, any macrolide, or any ketolide; history of cholestatic jaundice/hepatic dysfunction associated with previous use of azithromycin
Adverse effects/toxicities	Cardiovascular: palpitations, chest pain, arrhythmias, QT prolongation, or torsade de pointes Gastrointestinal: dyspepsia, flatulence, diarrhea, loose stools, nausea, vomiting, abdominal pain, melena, cholestatic jaundice, anorexia, constipation, pseudomembranous colitis, pancreatitis, oral candidiasis, pyloric stenosis, or tongue discoloration Genitourinary: monilial, vaginitis, nephritis, or acute renal failure Nervous system: dizziness, headache, vertigo, somnolence, convulsions, hyperactivity, nervousness, agitation, or syncope Liver/biliary: hepatic dysfunction Skin/appendages: pruritus, erythema multiforme, Stevens–Johnson syndrome, or toxic epidermal necrolysis General: fatigue, asthenia, paresthesia, malaise, anaphylaxis, hearing loss, deafness

Supplemental Table XX

Prescribing information for azithromycin

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Indication	Adjunctive treatment in acne, especially during pregnancy
Dosing	Children: mild to moderate skin infections: >3 months and <40 kg, 25 mg/kg/day orally every 2 hours OR 20 mg/kg/day every 8 hours; >3 months and >40 kg 500 mg orally every 12 hours or 250 mg orally every 8 hours Adults: 250 mg twice a day up to 500 mg 3 times a day
Contraindications	Known hypersensitivity to penicillins, including serious hypersensitivity reactions, such as anaphylaxis and Stevens–Johnson syndrome to penicillins and cephalosporins
Adverse effects/toxicities	Skin: acute generalized exanthematous pustulosis, erythematous maculopapular rash, urticaria, erythema multiforme, Stevens–Johnson syndrome, or toxic epidermal necrolysis Gastrointestinal: diarrhea, nausea, or vomiting Neurologic: headache, agitation, anxiety, behavior changes, dizziness, insomnia, or seizure Immunologic: anaphylaxis, hypersensitivity reaction, or serum sickness Blood: agranulocytosis, anemia, eosinophilia, hemolytic anemia, leucopenia,

Supplemental Table XXI

Prescribing information for amoxicillin

[Open table in a new tab](#)

Indication	Adjunctive treatment in acne
Dosing	Children: 25-50 mg/kg/day every 6-8 hours Adults: 500 mg twice a day
Contraindications	Hypersensitivity to cephalosporins
Adverse effects/toxicities	Central nervous system: agitation, confusion, dizziness, fatigue, or headache Skin: erythema multiforme, genital pruritus, Stevens–Johnson syndrome, toxic epidermal necrolysis, or urticaria Gastrointestinal: abdominal pain, diarrhea, dyspepsia, gastritis, nausea, pseudomembranous colitis, or vomiting Genitourinary: genital candidiasis, vaginal discharge, or vaginitis Blood: eosinophilia, hemolytic anemia, neutropenia, or thrombocytopenia Hepatic: cholestatic jaundice, hepatitis, or increased aspartate transaminase and alanine transaminase Immunologic: anaphylaxis, angioedema, or hypersensitivity reaction Skeletal: arthralgia, arthritis Renal: interstitial nephritis

Supplemental Table XXII

Prescribing information for cephalexin

[Open table in a new tab](#)

Indication	Acne vulgaris
Dosing	1 tablet orally daily at the same time Children: after menarche, 1 tablet daily at the same time
Contraindications	Blood pressure: systolic >160 mm Hg, diastolic >100 mm Hg, or severe hypertension Carcinoma of the breast Carcinoma of the endometrium Cerebral vascular or coronary artery disease Cholestatic jaundice of pregnancy or jaundice with previous pill use Deep vein thrombosis or thromboembolic disorders Diabetes with vascular involvement Genital bleeding, undiagnosed Headaches with focal neurologic symptoms Hepatic adenomas or carcinomas Hepatocellular disease with abnormal liver function Hypersensitivity Valvular heart disease with complications Surgery with prolonged immobilization

Supplemental Table XXIII

Prescribing information for ethinyl estradiol/norgestimate

[Open table in a new tab](#)

Indication	Adjuvant therapy for acne
Dosing	Teens ≥ 15 years of age and adults: 1 pill a day every day at the same time for 21 days followed by 1 week of no tablets
Contraindications	Anaphylactic reaction or angioedema Active or history of arterial thromboembolic disease (stroke or myocardial infarction) Breast cancer Carcinoma of the endometrium Cerebral vascular or coronary artery disease Cholestatic jaundice of pregnancy or jaundice with previous pill use Deep vein thrombosis or thromboembolic disease, pulmonary embolism

Undiagnosed genital bleeding
Hepatic adenomas or carcinomas
Hepatic disease
Pregnancy

Central nervous system: headache, depression, or nervousness

Supplemental Table XIV

Prescribing information for ethinyl estradiol/norethindrone acetate/ferrous fumarate

[Open table in a new tab](#)

Indication	Acne vulgaris, hormonal therapy
Dosing	Women: 1 tablet daily at the same time every day
Contraindications	<p>Renal dysfunction, adrenal insufficiency Breast cancer or other estrogen- or progestin-sensitive cancer Cerebrovascular disease, coronary artery disease Current or history of deep vein thrombosis or pulmonary embolism Headaches with focal neurologic symptoms or migraine headaches with or without aura >35 years of age Hepatic dysfunction, hepatic tumors benign or malignant Hypercoagulopathies Hypertension, uncontrolled Pregnancy Smoking if >35 years of age Undiagnosed uterine bleeding Thrombogenic valvular or thrombogenic rhythm diseases</p>
	Cardiovascular: edema, varicose vein aggravation, increase risk of arterial

Supplemental Table XXV

Prescribing information for ethinyl estradiol/drospirenone

[Open table in a new tab](#)

Indication	Acne vulgaris, hormonal therapy
Dosing	Women after the beginning of menses: 1 pink tablet orally every day for 24 consecutive days followed by 1 orange tablet daily for 4 days Begin therapy either on the first day of menstrual period or the first Sunday after the

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	onset of menstruation
	May be initiated 4 weeks postpartum in nonlactating mothers
	Adrenal insufficiency
	Breast cancer or other estrogen- or progestin-sensitive cancer
	Cerebrovascular disease
	Coronary artery disease
	Current or history of deep vein thrombosis or pulmonary embolism
	Diabetes with vascular disease
	Headaches with focal neurologic symptoms or migraine headaches with or without aura if >35 years of age
Contraindications	Hepatic tumors, benign or malignant
	Hepatic disease
	Hypercoagulopathies, inherited or acquired
	Uncontrolled hypertension

Supplemental Table XXVI

Prescribing information for ethinyl estradiol/drospirenone/levomefolate

[Open table in a new tab](#)

Indication	Off-label use for acne vulgaris in females
Dosing	Adult: 50-200 mg orally daily
Duration of dosing	10 months
Contraindications	Acute renal failure, Addison disease, hyperkalemia, anuria, concomitant eplerenone or triamterene use, and significant renal impairment
Adverse effects/toxicities	<p>Endocrine: gynecomastia, electrolyte disturbances, hyperkalemia, metabolic acidosis, or potential feminization male fetus if taken during pregnancy</p> <p>Gastrointestinal: diarrhea, nausea, vomiting, gastric hemorrhage, or gastritis</p> <p>Skin: erythematous maculopapular rash, Stevens–Johnson syndrome, or toxic epidermal necrolysis</p> <p>Neurologic: somnolence, confusion, or headache</p> <p>Blood: agranulocytosis</p> <p>Immunologic: drug hypersensitivity syndrome, systemic lupus erythematosus</p> <p>Reproductive: amenorrhea, irregular menses, postmenopausal bleeding, or erectile dysfunction</p>

Supplemental Table XXVII

Prescribing information for spironolactone

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Indication	Acne, antiandrogen effect
Dosing	250-500 mg orally daily
Contraindications	Hypersensitivity to flutamide Severe hepatic impairment
Adverse effects/toxicities	Skin: rash, ecchymosis, or pruritus Endocrine: hot sweats, galactorrhea, or decreased libido Gastrointestinal: diarrhea, nausea, anorexia, constipation, or dyspepsia Genitourinary: impotence, cystitis, or breast tenderness Blood: anemia, leukopenia, or thrombocytopenia Hepatic: hepatotoxicity, liver failure Central nervous system: anxiety, confusion, depression, dizziness, headache, or insomnia
Baseline monitoring	Liver function tests

Supplemental Table XXVIII

Prescribing information for flutamide

[Open table in a new tab](#)

Indication	Recalcitrant nodulocystic acne
Dosing	Severe: ≥ 12 years of age: 0.5-1 mg/kg/day orally in 2 divided doses with food Moderate: ≥ 12 years of age: 0.3-0.5 mg/kg/day Adults: 0.5-1 mg/kg/day
Duration of dosing	15-20 weeks
Contraindications	Hypersensitivity to isotretinoin or any of its components Hypersensitivity to vitamin A Pregnancy
	Cardiovascular: chest pain, edema, flushing, palpitation, stroke, syncope, or thrombosis Central nervous system: aggressive behavior, depression, emotional instability, fatigue, headache, psychosis, suicidal ideation/attempts, violent behavior, stroke, pseudotumor cerebri, or seizure Skin: alopecia, cheilitis, cutaneous allergic reaction, dry nose, dry skin, eruptive

Supplemental Table XXIX

Prescribing information for isotretinoin

[Open table in a new tab](#)

Indication	Inflammatory nodulocystic acne and acne keloidalis
Dosing	<p>Nodular acne: triamcinolone acetonide in 10 mg/mL. May be diluted with sterile normal saline to 5 or 3.3 mg/mL</p> <p>Acne keloidalis: triamcinolone acetonide -10 into inflammatory follicular lesions</p> <p>Triamcinolone acetonide -40 into hypertrophic scars and keloids</p>
Contraindications	<p>Should not be injected at the site of active infections, such as impetigo or herpes</p> <p>Should not be used if previous hypersensitivity to triamcinolone</p> <p>Large injections should be avoided in those with active tuberculosis or systemic fungal infection</p> <p>Extensive plaque psoriasis, pustular psoriasis, or erythrodermic psoriasis</p> <p>Active peptic ulcer disease</p> <p>Uncontrolled diabetes, heart failure, or severe hypertension</p> <p>Severe depression or psychosis</p>
Short-term	<p>Flatten most acne nodules in 18 to 24 hours</p>

Supplemental Table XXX

Prescribing information for intralesional corticosteroid (triamcinolone acetonide)

[Open table in a new tab](#)

Indication	Acne vulgaris and acne scars
Dosing	<p>Available as free acids, partially neutralized (higher pH), buffered, or esterified solutions</p> <p>Available concentrations range from 20-70%</p> <p>Very superficial: 30-50% glycolic acid applied for 1-2 min</p> <p>Superficial: 50-70% applied for 2-5 min</p> <p>Medium depth: 70% applied for 3-15 min</p>
Duration of dosing	Once every 15 days for 4-6 months

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Lack of psychological stability and mental preparedness
Unrealistic expectations
Poor general health and nutritional status
Isotretinoin therapy within the last 6 mos
Active infection or open wounds (eg, herpes simplex, excoriations, or open acne cysts)

Supplemental Table XXXI

Prescribing information for glycolic acid peels

[Open table in a new tab](#)

Indication	Comedonal acne
Dosing	<p>Concentrations of 20-30% are available Very superficial: 20% salicylic acid Superficial: 30% salicylic acid Applied for 2-4 minutes depending on intensity of clinical response</p>
Contraindications	<p>Lack of psychological stability and mental preparedness Unrealistic expectations Poor general health and nutritional status Isotretinoin therapy within the last 6 months Active infection or open wounds (eg, herpes simplex, excoriations, or open acne cysts) Relative contraindications History of abnormal scar formation or delayed wound healing History of therapeutic radiation exposure History of rosacea, seborrheic dermatitis, atopic dermatitis, psoriasis, vitiligo, or active retinoid dermatitis For medium and deep peels: medium-depth or deep resurfacing procedure <i>within the last 2-12 months</i></p>

Supplemental Table XXXII

Prescribing information for salicylic acid peels

[Open table in a new tab](#)

Indication	Acne
Dosing	<p>Cream, cloth, foam, or liquid cleansers 2%: use to clean face once or twice a day Gel 0.5% or  2%: apply small amount to face twice a day Pads 0.5% or 2%: use pad to cover affected area 1-3 times a day</p>

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Patch 2%: use at bedtime, after washing face and allowing face to dry at least 5 min. Apply patch directly over pimple being treated. Remove in the morning

Contraindications	Hypersensitivity to salicylic acid
Adverse effects/toxicities	Central nervous system: dizziness, headache, and mental confusion Local: burning and irritation, peeling, and scaling Otic: tinnitus Respiratory: hyperventilation

Supplemental Table XXXIII

Prescribing information for combination resorcinol and salicylic acid

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References

1. Strauss, J.S. · Krowchuk, D.P. · Leyden, J.J. ...

Guidelines of care for acne vulgaris management

J Am Acad Dermatol. 2007; **56**:651-663

[Full Text](#)



[Full Text \(PDF\)](#)

[Scopus \(447\)](#)

[PubMed](#)

[Google Scholar](#)

2. Eichenfield, L.F. · Krakowski, A.C. · Piggott, C. ...

Evidence-based recommendations for the diagnosis and treatment of pediatric acne

Pediatrics. 2013; **131**:S163-S186

[Crossref](#)

[Scopus \(197\)](#)

[PubMed](#)

[Google Scholar](#)

3. Ebell, M.H. · Siwek, J. · Weiss, B.D. ...

Simplifying the language of evidence to improve patient care: Strength of recommendation taxonomy (SORT): a patient-centered approach to grading evidence in medical literature

J Fam Pract. 2004; **53**:111-120

[PubMed](#)

[Google Scholar](#)

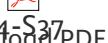
4. American Academy of Dermatology website. Guideline development process. Available at: <http://www.aad.org/practice-tools/quality-care/clinical-guidelines/guideline-development-process>. Accessed January 4, 2016.

[Google Scholar](#)

5. White, G.M.

Recent findings in the epidemiologic evidence, classification, and subtypes of acne vulgaris

J Am Acad Dermatol. 1998; **39**:S34-S37



[Download PDF](#)

[Full Text](#)[Full Text \(PDF\)](#)[PubMed](#)[Google Scholar](#)

-
6. Bhate, K. · Williams, H.C.

Epidemiology of acne vulgaris

Br J Dermatol. 2013; **168**:474-485

[Crossref](#)[Scopus \(210\)](#)[PubMed](#)[Google Scholar](#)

-
7. Goulden, V. · Stables, G.I. · Cunliffe, W.J.

Prevalence of facial acne in adults

J Am Acad Dermatol. 1999; **41**:577-580

[Abstract](#)[Full Text \(PDF\)](#)[PubMed](#)[Google Scholar](#)

-
8. Tan, J.K. · Tang, J. · Fung, K. ...

Development and validation of a comprehensive acne severity scale

J Cutan Med Surg. 2007; **11**:211-216

[Crossref](#)[Scopus \(111\)](#)[PubMed](#)[Google Scholar](#)

-
9. Mallon, E. · Newton, J.N. · Klassen, A. ...

The quality of life in acne: a comparison with general medical conditions using generic questionnaires

Br J Dermatol. 1999; **140**:672-676

[Crossref](#)[Scopus \(471\)](#)[PubMed](#)[Google Scholar](#)

-
10. Gupta, M.A. · Johnson, A.M. · Gupta, A.K.

The development of an Acne Quality of Life scale: reliability, validity, and relation to subjective acne severity in mild to moderate acne vulgaris

Acta Derm Venereol. 1998; **78**:451-456

[Crossref](#)[Scopus \(139\)](#)[PubMed](#)[Google Scholar](#)

-
11. Lasek, R.J. · Chren, M.M.

Acne vulgaris and the quality of life of adult dermatology patients

Arch Dermatol. 1998; **134**:454-458

[Crossref](#)[Scopus \(280\)](#)[PubMed](#)[Google Scholar](#)

-
12. Martin, A.R. · Lookingbill, D.P. · Botek, A. ...

Health-related quality of life among patients with facial acne—assessment of a new acne-specific questionnaire

Clin Exp Dermatol. 2001; **26**:380-385

[Crossref](#)[Scopus \(125\)](#)[PubMed](#)[Google Scholar](#)

-
13. Rapp, S.R. · Feldman, S.R. · Graham, G. ...

[Download PDF](#)

The Acne Quality of Life Index (Acne-QOLI): development and validation of a brief instrument

Am J Clin Dermatol. 2006; **7**:185-192

[Crossref](#) [Scopus \(o\)](#) [PubMed](#) [Google Scholar](#)

-
14. Dreno, B. · Khammari, A. · Orain, N. ...

ECCA grading scale: an original validated acne scar grading scale for clinical practice in dermatology

Dermatology. 2007; **214**:46-51

[Crossref](#) [Scopus \(155\)](#) [PubMed](#) [Google Scholar](#)

-
15. Pochi, P.E. · Shalita, A.R. · Strauss, J.S. ...

Report of the Consensus Conference on Acne Classification. Washington, D.C., March 24 and 25, 1990

J Am Acad Dermatol. 1991; **24**:495-500

 [Full Text \(PDF\)](#) [PubMed](#) [Google Scholar](#)

-
16. Doshi, A. · Zaheer, A. · Stiller, M.J.

A comparison of current acne grading systems and proposal of a novel system

Int J Dermatol. 1997; **36**:416-418

[Crossref](#) [PubMed](#) [Google Scholar](#)

-
17. Lucky, A.W. · Barber, B.L. · Girman, C.J. ...

A multirater validation study to assess the reliability of acne lesion counting

J Am Acad Dermatol. 1996; **35**:559-565

[Abstract](#)  [Full Text \(PDF\)](#) [Scopus \(113\)](#) [PubMed](#) [Google Scholar](#)

-
18. Cook, C.H. · Centner, R.L. · Michaels, S.E.

An acne grading method using photographic standards

Arch Dermatol. 1979; **115**:571-575

[Crossref](#) [PubMed](#) [Google Scholar](#)

-
19. Burke, B.M. · Cunliffe, W.J.

The assessment of acne vulgaris—the Leeds technique

Br J Dermatol. 1984; **111**:83-92

[Crossref](#) [PubMed](#) [Google Scholar](#)

-
20. Allen, B.S. · Smith, Jr., J.G.

Various parameters for grading acne vulgaris

Arch Dermatol. 1982; **118**:23-25

[Crossref](#) [PubMed](#) 

Download PDF

21. Dreno, B. · Poli, F. · Pawin, H. ...
Development and evaluation of a Global Acne Severity Scale (GEA Scale) suitable for France and Europe
J Eur Acad Dermatol Venereol. 2011; **25**:43-48
Crossref Scopus (147) PubMed Google Scholar
-
22. Hayashi, N. · Akamatsu, H. · Kawashima, M.
Acne Study Group. Establishment of grading criteria for acne severity
J Dermatol. 2008; **35**:255-260
PubMed Google Scholar
-
23. Hayashi, N. · Suh, D.H. · Akamatsu, H. ..., Acne Study Group
Evaluation of the newly established acne severity classification among Japanese and Korean dermatologists
J Dermatol. 2008; **35**:261-263
Crossref Scopus (12) PubMed Google Scholar
-
24. Tan, J. · Wolfe, B. · Weiss, J. ...
Acne severity grading: determining essential clinical components and features using a Delphi consensus
J Am Acad Dermatol. 2012; **67**:187-193
Full Text  Full Text (PDF) Scopus (0) PubMed Google Scholar
-
25. Tan, J.K. · Jones, E. · Allen, E. ...
Evaluation of essential clinical components and features of current acne global grading scales
J Am Acad Dermatol. 2013; **69**:754-761
Full Text  Full Text (PDF) PubMed Google Scholar
-
26. Beylot, C. · Chivot, M. · Faure, M. ...
Inter-observer agreement on acne severity based on facial photographs
J Eur Acad Dermatol Venereol. 2010; **24**:196-198
Crossref Scopus (5) Google Scholar
-
27. Tan, J.K. · Fung, K. · Bulger, L.
Reliability of dermatologists in acne lesion counts and global assessments
J Cutan Med Surg. 2006; **10**:160-165
Crossref Scopus (32) PubMed Google Scholar
-
28. Bergman, H. · Tsai, K.Y. · Seo, S.J. ...
Remote assessment of acne: the use of acne grading tools to evaluate digital skin images
Telemed J E Health. 2009; **15**:426-430  PDF

[Crossref](#)[Scopus \(8\)](#)[PubMed](#)[Google Scholar](#)

-
29. Min, S. · Kong, H.J. · Yoon, C. ...
Development and evaluation of an automatic acne lesion detection program using digital image processing
Skin Res Technol. 2013; **19**:e423-e432
[Crossref](#) [Google Scholar](#)
30. Qureshi, A.A. · Brandling-Bennett, H.A. · Giberti, S. ...
Evaluation of digital skin images submitted by patients who received practical training or an online tutorial
J Telemed Telecare. 2006; **12**:79-82
[Crossref](#) [Scopus \(18\)](#) [PubMed](#) [Google Scholar](#)
31. Choi, C.W. · Choi, J.W. · Park, K.C. ...
Ultraviolet-induced red fluorescence of patients with acne reflects regional casual sebum level and acne lesion distribution: qualitative and quantitative analyses of facial fluorescence
Br J Dermatol. 2012; **166**:59-66
[Crossref](#) [Scopus \(4\)](#) [Google Scholar](#)
32. Choi, C.W. · Lee, D.H. · Kim, H.S. ...
The clinical features of late onset acne compared with early onset acne in women
J Eur Acad Dermatol Venereol. 2011; **25**:454-461
[Crossref](#) [Scopus \(12\)](#) [Google Scholar](#)
33. Dobrev, H.
Fluorescence diagnostic imaging in patients with acne
Photodermat Photoimmunol Photomed. 2010; **26**:285-289
[Crossref](#) [Scopus \(2\)](#) [Google Scholar](#)
34. Choi, C.W. · Choi, J.W. · Youn, S.W.
Subjective facial skin type, based on the sebum related symptoms, can reflect the objective casual sebum level in acne patients
Skin Res Technol. 2013; **19**:176-182
[Crossref](#) [Scopus \(2\)](#) [Google Scholar](#)
35. Kim, M.K. · Choi, S.Y. · Byun, H.J. ...
Comparison of sebum secretion, skin type, pH in humans with and without acne
Arch Dermatol Res. 2006; **298**:113-119
[Crossref](#) [Scopus \(64\)](#) [!\[\]\(eefb0f7373c006973a817812df480659_img.jpg\)](#) [Google Scholar](#)

[Download PDF](#)

36. Xhaulaire-Uhoda, E. · Pierard, G.E.
Skin capacitance imaging of acne lesions
Skin Res Technol. 2007; **13**:9-12
[Crossref](#) [Scopus \(10\)](#) [Google Scholar](#)
37. Youn, S.H. · Choi, C.W. · Choi, J.W. ...
The skin surface pH and its different influence on the development of acne lesion according to gender and age
Skin Res Technol. 2013; **19**:131-136
[Crossref](#) [Scopus \(28\)](#) [PubMed](#) [Google Scholar](#)
38. Youn, S.W. · Kim, J.H. · Lee, J.E. ...
The facial red fluorescence of ultraviolet photography: is this color due to Propionibacterium acnes or the unknown content of secreted sebum?
Skin Res Technol. 2009; **15**:230-236
[Crossref](#) [Scopus \(18\)](#) [Google Scholar](#)
39. Zane, C. · Capezzera, R. · Pedretti, A. ...
Non-invasive diagnostic evaluation of phototherapeutic effects of red light phototherapy of acne vulgaris
Photodermat Photoimmunol Photomed. 2008; **24**:244-248
[Crossref](#) [Scopus \(11\)](#) [Google Scholar](#)
40. Cove, J.H. · Cunliffe, W.J. · Holland, K.T.
Acne vulgaris: is the bacterial population size significant?
Br J Dermatol. 1980; **102**:277-280
[Crossref](#) [PubMed](#) [Google Scholar](#)
41. Mourelatos, K. · Eady, E.A. · Cunliffe, W.J. ...
Temporal changes in sebum excretion and propionibacterial colonization in preadolescent children with and without acne
Br J Dermatol. 2007; **156**:22-31
[Crossref](#) [Scopus \(72\)](#) [PubMed](#) [Google Scholar](#)
42. Shaheen, B. · Gonzalez, M.
A microbial aetiology of acne: what is the evidence?
Br J Dermatol. 2011; **165**:474-485
[Crossref](#) [Scopus \(35\)](#) [Google Scholar](#)
43. Fitz-Gibbon, S. · Tomida, S. · Chiu, B.H. ...
Propionibacterium acnes strain populations in the human skin microbiome associated with acne
[Download PDF](#)



-
44. Holland, C. · Mak, T.N. · Zimny-Arndt, U. ...

Proteomic identification of secreted proteins of *Propionibacterium acnes*

BMC Microbiol. 2010; 10:230

[Crossref](#)

[Scopus \(74\)](#)

[PubMed](#)

[Google Scholar](#)

-
45. Lomholt, H.B. · Kilian, M.

Population genetic analysis of *Propionibacterium acnes* identifies a subpopulation and epidemic clones associated with acne

PLoS One. 2010; 5:e12277

[Crossref](#)

[Scopus \(209\)](#)

[PubMed](#)

[Google Scholar](#)

-
46. Miura, Y. · Ishige, I. · Soejima, N. ...

Quantitative PCR of *Propionibacterium acnes* DNA in samples aspirated from sebaceous follicles on the normal skin of subjects with or without acne

J Med Dent Sci. 2010; 57:65-74

[Google Scholar](#)

-
47. Tochio, T. · Tanaka, H. · Nakata, S. ...

Accumulation of lipid peroxide in the content of comedones may be involved in the progression of comedogenesis and inflammatory changes in comedones

J Cosmet Dermatol. 2009; 8:152-158

[Crossref](#)

[Scopus \(20\)](#)

[Google Scholar](#)

-
48. Tomida, S. · Nguyen, L. · Chiu, B.H. ...

Pan-genome and comparative genome analyses of *propionibacterium acnes* reveal its genomic diversity in the healthy and diseased human skin microbiome

MBio. 2013; 4:e00003-e00013

[Crossref](#)

[Scopus \(151\)](#)

[Google Scholar](#)

-
49. Lucky, A.W. · Biro, F.M. · Simbartl, L.A. ...

Predictors of severity of acne vulgaris in young adolescent girls: results of a five-year longitudinal study

J Pediatr. 1997; 130:30-39

[Full Text](#)



[Full Text \(PDF\)](#)

[Scopus \(153\)](#)

[PubMed](#)

[Google Scholar](#)

-
50. Bunker, C.B. · Newton, J.A. · Kilborn, J. ...

Most women with acne have polycystic ovaries

Br J Dermatol. 1989; 121:675-680

[Download PDF](#)

-
51. Lawrence, D.M. · Katz, M. · Robinson, T.W. ...

Reduced sex hormone binding globulin and derived free testosterone levels in women with severe acne

Clin Endocrinol. 1981; **75**:87-91

-
52. Timpatanapong, P. · Rojanasakul, A.

Hormonal profiles and prevalence of polycystic ovary syndrome in women with acne

J Dermatol. 1997; **24**:223-229

-
53. Lucky, A.W.

Endocrine aspects of acne

Pediatr Clin North Am. 1983; **30**:495-499

-
54. Lucky, A.W. · McGuire, J. · Rosenfield, R.L. ...

Plasma androgens in women with acne vulgaris

J Invest Dermatol. 1983; **81**:70-74



-
55. Abulnaja, K.O.

Changes in the hormone and lipid profile of obese adolescent Saudi females with acne vulgaris

Braz J Med Biol Res. 2009; **42**:501-505

-
56. Arora, M.K. · Seth, S. · Dayal, S.

The relationship of lipid profile and menstrual cycle with acne vulgaris

Clin Biochem. 2010; **43**:1415-1420

-
57. Fyrand, O. · Jakobsen, H.B.

Water-based versus alcohol-based benzoyl peroxide preparations in the treatment of acne vulgaris

Dermatologica. 1986; **172**:263-267

-
58. Mills, Jr., O.H. · Kligman, A.M. · Chi, P. ...

Comparing 2.5%, 5%, and 10% benzoyl peroxide on inflammatory acne vulgaris

59. Schutte, H. · Cunliffe, W.J. · Forster, R.A.

The short-term effects of benzoyl peroxide lotion on the resolution of inflamed acne lesions

Br J Dermatol. 1982; **106**:91-94

60. Mills, Jr., O. · Thornberry, C. · Cardin, C.W. ...

Bacterial resistance and therapeutic outcome following three months of topical acne therapy with 2% erythromycin gel versus its vehicle

Acta Derm Venereol. 2002; **82**:260-265

61. Bernstein, J.E. · Shalita, A.R.

Topically applied erythromycin in inflammatory acne vulgaris

J Am Acad Dermatol. 1980; **2**:318-321

62. Jones, E.L. · Crumley, A.F.

Topical erythromycin vs blank vehicle in a multiclinic acne study

Arch Dermatol. 1981; **117**:551-553

63. Shalita, A.R. · Smith, E.B. · Bauer, E.

Topical erythromycin v clindamycin therapy for acne. A multicenter, double-blind comparison

Arch Dermatol. 1984; **120**:351-355

64. Leyden, J.J. · Shalita, A.R. · Saatjian, G.D. ...

Erythromycin 2% gel in comparison with clindamycin phosphate 1% solution in acne vulgaris

J Am Acad Dermatol. 1987; **16**:822-827

65. Kuhlman, D.S. · Callen, J.P.

A comparison of clindamycin phosphate 1 percent topical lotion and placebo in the treatment of acne vulgaris

Cutis. 1986; **38**:203-206



66. Becker, L.E. · Bergstresser, B.R. · Whiting, D.A. ...

Topical clindamycin therapy for acne vulgaris. A cooperative clinical study

Arch Dermatol. 1981; **117**:482-485

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

-
67. Leyden, J.J. · Hickman, J.G. · Jarratt, M.T. ...

The efficacy and safety of a combination benzoyl peroxide/clindamycin topical gel compared with benzoyl peroxide alone and a benzoyl peroxide/erythromycin combination product

J Cutan Med Surg. 2001; **5**:37-42

[PubMed](#)

[Google Scholar](#)

-
68. Lookingbill, D.P. · Chalker, D.K. · Lindholm, J.S. ...

Treatment of acne with a combination clindamycin/benzoyl peroxide gel compared with clindamycin gel, benzoyl peroxide gel and vehicle gel: combined results of two double-blind investigations

J Am Acad Dermatol. 1997; **37**:590-595

[Full Text](#)



[Full Text \(PDF\)](#)

[Scopus \(189\)](#)

[PubMed](#)

[Google Scholar](#)

-
69. Tschen, E.H. · Katz, H.I. · Jones, T.M. ...

A combination benzoyl peroxide and clindamycin topical gel compared with benzoyl peroxide, clindamycin phosphate, and vehicle in the treatment of acne vulgaris

Cutis. 2001; **67**:165-169

[PubMed](#)

[Google Scholar](#)

-
70. Krishnan, G.

Comparison of two concentrations of tretinoin solution in the topical treatment of acne vulgaris

Practitioner. 1976; **216**:106-109

[PubMed](#)

[Google Scholar](#)

-
71. Bradford, L.G. · Montes, L.F.

Topical application of vitamin A acid in acne vulgaris

South Med J. 1974; **67**:683-687

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

-
72. Shalita, A.R. · Chalker, D.K. · Griffith, R.F. ...

Tazarotene gel is safe and effective in the treatment of acne vulgaris: a multicenter, double-blind, vehicle-controlled study

Cutis. 1999; **63**:349-354

[PubMed](#)

[Google Scholar](#)

-
73. Shalita, A. · Weiss, J.S. · Chalker, D.K. ...

A comparison of the efficacy and safety of adapalene gel 0.1% and tretinoin gel 0.025% in the treatment of acne vulgaris: a multicenter trial



[Download PDF](#)



74. Cunliffe, W.J. · Caputo, R. · Dreno, B. ...

Clinical efficacy and safety comparison of adapalene gel and tretinoin gel in the treatment of acne vulgaris: Europe and U.S. multicenter trials

J Am Acad Dermatol. 1997; 36:S126-S134



75. Richter, J.R. · Bousema, M.T. · De Boulle, K.L.V. ...

Efficacy of a fixed clindamycin phosphate 1.2%, tretinoin 0.025% gel formulation (Velac) in the topical control of facial acne lesions

J Dermatolog Treat. 1998; 9:81-90

76. Zouboulis, C.C. · Derumeaux, L. · Decroix, J. ...

A multicentre, single-blind, randomized comparison of a fixed clindamycin phosphate/tretinoin gel formulation (Velac) applied once daily and a clindamycin lotion formulation (Dalcin T) applied twice daily in the topical treatment of acne vulgaris

Br J Dermatol. 2000; 143:498-505

77. Christiansen, J.V. · Gadborg, E. · Ludvigsen, K. ...

Topical tretinoin, vitamin A acid (Airol) in acne vulgaris. A controlled clinical trial

Dermatologica. 1974; 148:82-89

78. Dunlap, F.E. · Mills, O.H. · Tuley, M.R. ...

Adapalene 0.1% gel for the treatment of acne vulgaris: its superiority compared to tretinoin 0.025% cream in skin tolerance and patient preference

Br J Dermatol. 1998; 139:17-22

79. Kakita, L.

Tazarotene versus tretinoin or adapalene in the treatment of acne vulgaris

J Am Acad Dermatol. 2000; 43:S51-S54



80. Webster, G.F. · Berson, D. · Stein, L.F. ...

Efficacy and tolerability of once-daily tazarotene 0.1% gel versus once-daily tretinoin 0.025% gel in the treatment of facial acne vulgaris: a randomized trial

Cutis. 2001; 67:4-9

-
81. Galvin, S.A. · Gilbert, R. · Baker, M. ...
Comparative tolerance of adapalene 0.1% gel and six different tretinoin formulations
Br J Dermatol. 1998; **139**:34-40
[Crossref](#) [PubMed](#) [Google Scholar](#)
-
82. Cunliffe, W.J. · Holland, K.T.
Clinical and laboratory studies on treatment with 20% azelaic acid cream for acne
Acta Derm Venereol Suppl (Stockh). 1989; **143**:31-34
[PubMed](#) [Google Scholar](#)
-
83. Katsambas, A. · Graupe, K. · Stratigos, J.
Clinical studies of 20% azelaic acid cream in the treatment of acne vulgaris. Comparison with vehicle and topical tretinoin
Acta Derm Venereol Suppl (Stockh). 1989; **143**:35-39
[PubMed](#) [Google Scholar](#)
-
84. Draelos, Z.D. · Carter, E. · Maloney, J.M. ...
Two randomized studies demonstrate the efficacy and safety of dapson gel, 5% for the treatment of acne vulgaris
J Am Acad Dermatol. 2007; **56**:439.e1-439.e10
[Full Text](#)  [Full Text \(PDF\)](#) [Scopus \(134\)](#) [Google Scholar](#)
-
85. Lucky, A.W. · Maloney, J.M. · Roberts, J. ...
Dapsone gel 5% for the treatment of acne vulgaris: safety and efficacy of long-term (1 year) treatment
J Drugs Dermatol. 2007; **6**:981-987
[Google Scholar](#)
-
86. Tanghetti, E. · Harper, J.C. · Oefelein, M.G.
The efficacy and tolerability of dapson 5% gel in female vs male patients with facial acne vulgaris: gender as a clinically relevant outcome variable
J Drugs Dermatol. 2012; **11**:1417-1421
[PubMed](#) [Google Scholar](#)
-
87. Shalita, A.R.
Treatment of mild and moderate acne vulgaris with salicylic acid in an alcohol-detergent vehicle
Cutis. 1981; **28**:556-558 561
[PubMed](#) [Google Scholar](#) 
[Download PDF](#)

88. Garner, S.E. · Eady, A. · Bennett, C. ...
Minocycline for acne vulgaris: efficacy and safety
Cochrane Database Syst Rev. 2012; CD002086
[Google Scholar](#)
-
89. Leyden, J.J. · Bruce, S. · Lee, C.S. ...
A randomized, phase 2, dose-ranging study in the treatment of moderate to severe inflammatory facial acne vulgaris with doxycycline calcium
J Drugs Dermatol. 2013; **12**:658-663
[Google Scholar](#)
-
90. Lebrun-Vignes, B. · Kreft-Jais, C. · Castot, A. ..., French Network of Regional Centers of Pharmacovigilance
Comparative analysis of adverse drug reactions to tetracyclines: results of a French national survey and review of the literature
Br J Dermatol. 2012; **166**:1333-1341
[Crossref](#) [Scopus \(52\)](#) [PubMed](#) [Google Scholar](#)
-
91. Kermani, T.A. · Ham, E.K. · Camilleri, M.J. ...
Polyarteritis nodosa-like vasculitis in association with minocycline use: a single-center case series
Semin Arthritis Rheum. 2012; **42**:213-221
[Crossref](#) [Scopus \(51\)](#) [PubMed](#) [Google Scholar](#)
-
92. Rafiei, R. · Yaghoobi, R.
Azithromycin versus tetracycline in the treatment of acne vulgaris
J Dermatolog Treat. 2006; **17**:217-221
[Crossref](#) [Scopus \(21\)](#) [Google Scholar](#)
-
93. Jen, I.
A comparison of low dosage trimethoprim/sulfamethoxazole with oxytetracycline in acne vulgaris
Cutis. 1980; **26**:106-108
[PubMed](#) [Google Scholar](#)
-
94. Fenner, J.A. · Wiss, K. · Levin, N.A.
Oral cephalixin for acne vulgaris: clinical experience with 93 patients
Pediatr Dermatol. 2008; **25**:179-183
[Crossref](#) [Scopus \(8\)](#) [Google Scholar](#)
-
95. Gold, L.S. · Cruz, A. · Eichenfield, L. ...
[!\[\]\(6ec328c6983ef27b22b240b72c0c4b9d_img.jpg\) Download PDF](#)

Effective and safe combination therapy for severe acne vulgaris: a randomized, vehicle-controlled, double-blind study of adapalene 0.1%-benzoyl peroxide 2.5% fixed-dose combination gel with doxycycline hyclate 100 mg

Cutis. 2010; **85**:94-104

[PubMed](#) [Google Scholar](#)

96. Leyden, J. · Thiboutot, D.M. · Shalita, A.R. ...

Comparison of tazarotene and minocycline maintenance therapies in acne vulgaris: a multicenter, double-blind, randomized, parallel-group study

Arch Dermatol. 2006; **142**:605-612

[Crossref](#) [Scopus \(71\)](#) [PubMed](#) [Google Scholar](#)

97. Margolis, D.J. · Fanelli, M. · Hoffstad, O. ...

Potential association between the oral tetracycline class of antimicrobials used to treat acne and inflammatory bowel disease

Am J Gastroenterol. 2010; **105**:2610-2616

[Crossref](#) [Scopus \(147\)](#) [PubMed](#) [Google Scholar](#)

98. Lucky, A.W. · Koltun, W. · Thiboutot, D. ...

A combined oral contraceptive containing 3-mg drospirenone/20-microg ethinyl estradiol in the treatment of acne vulgaris: a randomized, double-blind, placebo-controlled study evaluating lesion counts and participant self-assessment

Cutis. 2008; **82**:143-150

[PubMed](#) [Google Scholar](#)

99. Maloney, J.M. · Dietze, Jr., P. · Watson, D. ...

Treatment of acne using a 3-milligram drospirenone/20-microgram ethinyl estradiol oral contraceptive administered in a 24/4 regimen: a randomized controlled trial

Obstet Gynecol. 2008; **112**:773-781

[Crossref](#) [Scopus \(63\)](#) [PubMed](#) [Google Scholar](#)

100. Maloney, J.M. · Dietze, Jr., P. · Watson, D. ...

A randomized controlled trial of a low-dose combined oral contraceptive containing 3 mg drospirenone plus 20 microg ethinylestradiol in the treatment of acne vulgaris: lesion counts, investigator ratings and subject self-assessment

J Drugs Dermatol. 2009; **8**:837-844

[PubMed](#) [Google Scholar](#)

101. Plewig, G. · Cunliffe, W.J. · Binder, N. ...

Efficacy of an oral contraceptive containing EE 0.03 mg and CMA 2 mg (Belara) in moderate acne resolution: a randomized, double-blind, placebo-controlled phase III trial

Contraception. 2009; **80**:25-32



[Download PDF](#)

[Full Text](#)[Full Text \(PDF\)](#)[Scopus \(37\)](#)[PubMed](#)[Google Scholar](#)

102. Shaw, J.C.

Low-dose adjunctive spironolactone in the treatment of acne in women: a retrospective analysis of 85 consecutively treated patients

J Am Acad Dermatol. 2000; **43**:498-502

[Full Text](#)[Full Text \(PDF\)](#)[PubMed](#)[Google Scholar](#)

103. Sato, K. · Matsumoto, D. · Iizuka, F. ...

Anti-androgenic therapy using oral spironolactone for acne vulgaris in Asians

Aesthetic Plast Surg. 2006; **30**:689-694

[Crossref](#)[Scopus \(o\)](#)[PubMed](#)[Google Scholar](#)

104. Wang, H.S. · Wang, T.H. · Soong, Y.K.

Low dose flutamide in the treatment of acne vulgaris in women with or without oligomenorrhea or amenorrhea

Changgeng Yi Xue Za Zhi. 1999; **22**:423-432

[PubMed](#)[Google Scholar](#)

105. Castelo-Branco, C. · Moyano, D. · Gomez, O. ...

Long-term safety and tolerability of flutamide for the treatment of hirsutism

Fertil Steril. 2009; **91**:1183-1188

[Full Text](#)[Full Text \(PDF\)](#)[Scopus \(o\)](#)[PubMed](#)[Google Scholar](#)

106. Nader, S. · Rodriguez-Rigau, L.J. · Smith, K.D. ...

Acne and hyperandrogenism: impact of lowering androgen levels with glucocorticoid treatment

J Am Acad Dermatol. 1984; **11**:256-259

[Abstract](#)[Full Text \(PDF\)](#)[PubMed](#)[Google Scholar](#)

107. Amichai, B. · Shemer, A. · Grunwald, M.H.

Low-dose isotretinoin in the treatment of acne vulgaris

J Am Acad Dermatol. 2006; **54**:644-646

[Full Text](#)[Full Text \(PDF\)](#)[Scopus \(126\)](#)[PubMed](#)[Google Scholar](#)

108. Goldstein, J.A. · Socha-Szott, A. · Thomsen, R.J. ...

Comparative effect of isotretinoin and etretinate on acne and sebaceous gland secretion

J Am Acad Dermatol. 1982; **6**:760-765

[Abstract](#)[Full Text \(PDF\)](#)[Scopus \(87\)](#)[PubMed](#)[Google Scholar](#)

109. Jones, D.H. · King, K. · Miller, A.J.

A dose-response study of Isotretinoin acid in acne vulgaris

[110.](#) Layton, A.M. · Knaggs, H. · Taylor, J. ...

Isotretinoin for acne vulgaris—10 years later: a safe and successful treatment

Br J Dermatol. 1993; **129**:292-296

[111.](#) Lehucher-Ceyrac, D. · Weber-Buisset, M.J. ...

Isotretinoin and acne in practice: a prospective analysis of 188 cases over 9 years

Dermatology. 1993; **186**:123-128

[112.](#) Peck, G.L. · Olsen, T.G. · Butkus, D. ...

Isotretinoin versus placebo in the treatment of cystic acne. A randomized double-blind study

J Am Acad Dermatol. 1982; **6**:735-745

[113.](#) Rubinow, D.R. · Peck, G.L. · Squillace, K.M. ...

Reduced anxiety and depression in cystic acne patients after successful treatment with oral isotretinoin

J Am Acad Dermatol. 1987; **17**:25-32

[114.](#) Stainforth, J.M. · Layton, A.M. · Taylor, J.P. ...

Isotretinoin for the treatment of acne vulgaris: which factors may predict the need for more than one course?

Br J Dermatol. 1993; **129**:297-301

[115.](#) Strauss, J.S. · Leyden, J.J. · Lucky, A.W. ...

A randomized trial of the efficacy of a new micronized formulation versus a standard formulation of isotretinoin in patients with severe recalcitrant nodular acne

J Am Acad Dermatol. 2001; **45**:187-195

[116.](#) Strauss, J.S. · Rapini, R.P. · Shalita, A.R. ...

Isotretinoin therapy for acne: results of a multicenter dose-response study

J Am Acad Dermatol. 1984; **10**:490-496

- [117.](#) Strauss, J.S. · Stranieri, A.M.
Changes in long-term sebum production from isotretinoin therapy
J Am Acad Dermatol. 1982; **6**:751-756
Abstract  [Full Text \(PDF\)](#) [PubMed](#) [Google Scholar](#)
- [118.](#) Goldsmith, L.A. · Bologna, J.L. · Callen, J.P. ...
American Academy of Dermatology Consensus Conference on the safe and optimal use of isotretinoin: summary and recommendations
J Am Acad Dermatol. 2004; **50**:900-906
[Full Text](#)  [Full Text \(PDF\)](#) [Scopus \(99\)](#) [PubMed](#) [Google Scholar](#)
- [119.](#) Lehucher-Ceyrac, D. · de La Salmoniere, P. · Chastang, C. ...
Predictive factors for failure of isotretinoin treatment in acne patients: results from a cohort of 237 patients
Dermatology. 1999; **198**:278-283
[Crossref](#) [Scopus \(63\)](#) [PubMed](#) [Google Scholar](#)
- [120.](#) Strauss, J.S. · Leyden, J.J. · Lucky, A.W. ...
Safety of a new micronized formulation of isotretinoin in patients with severe recalcitrant nodular acne: a randomized trial comparing micronized isotretinoin with standard isotretinoin
J Am Acad Dermatol. 2001; **45**:196-207
[Full Text](#)  [Full Text \(PDF\)](#) [Scopus \(55\)](#) [PubMed](#) [Google Scholar](#)
- [121.](#) Webster, G.F. · Leyden, J.J. · Gross, J.A.
Comparative pharmacokinetic profiles of a novel isotretinoin formulation (isotretinoin-Lidose) and the innovator isotretinoin formulation: a randomized, 4-treatment, crossover study
J Am Acad Dermatol. 2013; **69**:762-767
[Full Text](#)  [Full Text \(PDF\)](#) [PubMed](#) [Google Scholar](#)
- [122.](#) Alhusayen, R.O. · Juurlink, D.N. · Mamdani, M.M. ...
Isotretinoin use and the risk of inflammatory bowel disease: a population-based cohort study
J Invest Dermatol. 2013; **133**:907-912
[Full Text](#)  [Full Text \(PDF\)](#) [Scopus \(70\)](#) [PubMed](#) [Google Scholar](#)
- [123.](#) Crockett, S.D. · Gulati, A. · Sandler, R.S. ...
A causal association between isotretinoin and inflammatory bowel disease has yet to be established
Am J Gastroenterol. 2009; **104**:2387-2393
[Crossref](#) [Scopus \(51\)](#) [PubMed](#) [Google Scholar](#)
- [124.](#) Crockett, S.D. · Porter, C.Q. · Martin, C.F. ...
 [Download PDF](#)
Isotretinoin use and the risk of inflammatory bowel disease: a case-control study

[125.](#) Etminan, M. · Bird, S.T. · Delaney, J.A. ...

Isotretinoin and risk for inflammatory bowel disease: a nested case-control study and meta-analysis of published and unpublished data

JAMA Dermatol. 2013; **149**:216-220

[Crossref](#)

[Scopus \(73\)](#)

[PubMed](#)

[Google Scholar](#)

[126.](#) Reddy, D. · Siegel, C.A. · Sands, B.E. ...

Possible association between isotretinoin and inflammatory bowel disease

Am J Gastroenterol. 2006; **101**:1569-1573

[Crossref](#)

[Scopus \(94\)](#)

[PubMed](#)

[Google Scholar](#)

[127.](#) Sundstrom, A. · Alfredsson, L. · Sjolin-Forsberg, G. ...

Association of suicide attempts with acne and treatment with isotretinoin: retrospective Swedish cohort study

BMJ. 2010; **341**:c5812

[Crossref](#)

[Scopus \(136\)](#)

[PubMed](#)

[Google Scholar](#)

[128.](#) Bozdag, K.E. · Gulseren, S. · Guven, F. ...

Evaluation of depressive symptoms in acne patients treated with isotretinoin

J Dermatolog Treat. 2009; **20**:293-296

[Crossref](#)

[Scopus \(28\)](#)

[PubMed](#)

[Google Scholar](#)

[129.](#) Chia, C.Y. · Lane, W. · Chibnall, J. ...

Isotretinoin therapy and mood changes in adolescents with moderate to severe acne: a cohort study

Arch Dermatol. 2005; **141**:557-560

[Crossref](#)

[Scopus \(124\)](#)

[PubMed](#)

[Google Scholar](#)

[130.](#) Cohen, J. · Adams, S. · Patten, S.

No association found between patients receiving isotretinoin for acne and the development of depression in a Canadian prospective cohort

Can J Clin Pharmacol. 2007; **14**:e227-e233

[PubMed](#)

[Google Scholar](#)

[131.](#) Jick, S.S. · Kremers, H.M. · Vasilakis-Scaramozza, C.

Isotretinoin use and risk of depression, psychotic symptoms, suicide, and attempted suicide

Arch Dermatol. 2000; **136**:1231-1236

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

Download PDF

132. Nevoralova, Z. · Dvorakova, D.
Mood changes, depression and suicide risk during isotretinoin treatment: a prospective study
Int J Dermatol. 2013; **52**:163-168
Crossref Scopus (10) Google Scholar
133. Rehn, L.M. · Meririnne, E. · Hook-Nikanne, J. ...
Depressive symptoms and suicidal ideation during isotretinoin treatment: a 12-week follow-up study of male Finnish military conscripts
J Eur Acad Dermatol Venereol. 2009; **23**:1294-1297
Crossref Scopus (48) PubMed Google Scholar
134. Agarwal, U.S. · Besarwal, R.K. · Bhola, K.
Oral isotretinoin in different dose regimens for acne vulgaris: a randomized comparative trial
Indian J Dermatol Venereol Leprol. 2011; **77**:688-694
Crossref Scopus (17) Google Scholar
135. Akman, A. · Durusoy, C. · Senturk, M. ...
Treatment of acne with intermittent and conventional isotretinoin: a randomized, controlled multicenter study
Arch Dermatol Res. 2007; **299**:467-473
Crossref Scopus (86) PubMed Google Scholar
136. Borghi, A. · Mantovani, L. · Minghetti, S. ...
Low-cumulative dose isotretinoin treatment in mild-to-moderate acne: efficacy in achieving stable remission
J Eur Acad Dermatol Venereol. 2011; **25**:1094-1098
Crossref Scopus (0) PubMed Google Scholar
137. Kaymak, Y. · Ilter, N.
The effectiveness of intermittent isotretinoin treatment in mild or moderate acne
J Eur Acad Dermatol Venereol. 2006; **20**:1256-1260
Crossref Scopus (43) PubMed Google Scholar
138. Lee, J.W. · Yoo, K.H. · Park, K.Y. ...
Effectiveness of conventional, low-dose and intermittent oral isotretinoin in the treatment of acne: a randomized, controlled comparative study
Br J Dermatol. 2011; **164**:1369-1375
Crossref Scopus (29) Google Scholar
139. Leachman, S.A. · Insogna, K.L. · Katz, L. ...
Bone densities in patients receiving isotretinoin for cystic acne
Download PDF

[140.](#) Bershad, S. · Rubinstein, A. · Paterniti, J.R. ...

Changes in plasma lipids and lipoproteins during isotretinoin therapy for acne

N Engl J Med. 1985; **313**:981-985

[141.](#) De Marchi, M.A. · Maranhao, R.C. · Brandizzi, L.I. ...

Effects of isotretinoin on the metabolism of triglyceride-rich lipoproteins and on the lipid profile in patients with acne

Arch Dermatol Res. 2006; **297**:403-408

[142.](#) Zech, L.A. · Gross, E.G. · Peck, G.L. ...

Changes in plasma cholesterol and triglyceride levels after treatment with oral isotretinoin. A prospective study

Arch Dermatol. 1983; **119**:987-993

[143.](#) Shin, J. · Cheetham, T.C. · Wong, L. ...

The impact of the iPLEDGE program on isotretinoin fetal exposure in an integrated health care system

J Am Acad Dermatol. 2011; **65**:1117-1125

[144.](#) Collins, M.K. · Moreau, J.F. · Opel, D. ...

Compliance with pregnancy prevention measures during isotretinoin therapy

J Am Acad Dermatol. 2014; **70**:55-59

[145.](#) Grover, C. · Reddu, B.S.

The therapeutic value of glycolic acid peels in dermatology

Indian J Dermatol Venereol Leprol. 2003; **69**:148-150

[146.](#) Dreno, B. · Fischer, T.C. · Perosino, E. ...

Expert opinion: efficacy of superficial chemical peels in active acne management—what can we learn from the literature today? Evidence-based recommendations

J Eur Acad Dermatol Venereol. 2011; **25**:695-704

147. Ilknur, T. · Demirtasoglu, M. · Bicak, M.U. ...

Glycolic acid peels versus amino fruit acid peels for acne

J Cosmet Laser Ther. 2010; **12**:242-245

[Crossref](#)

[Google Scholar](#)

148. Levine, R.M. · Rasmussen, J.E.

Intralesional corticosteroids in the treatment of nodulocystic acne

Arch Dermatol. 1983; **119**:480-481

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

149. Potter, R.A.

Intralesional triamcinolone and adrenal suppression in acne vulgaris

J Invest Dermatol. 1971; **57**:364-370

[Abstract](#)



[Full Text \(PDF\)](#)

[PubMed](#)

[Google Scholar](#)

150. Bassett, I.B. · Pannowitz, D.L. · Barnetson, R.S.

A comparative study of tea-tree oil versus benzoylperoxide in the treatment of acne

Med J Aust. 1990; **153**:455-458

[PubMed](#)

[Google Scholar](#)

151. Enshaieh, S. · Jooya, A. · Siadat, A.H. ...

The efficacy of 5% topical tea tree oil gel in mild to moderate acne vulgaris: a randomized, double-blind placebo-controlled study

Indian J Dermatol Venereol Leprol. 2007; **73**:22-25

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

152. Fouladi, R.F.

Aqueous extract of dried fruit of *Berberis vulgaris L.* in acne vulgaris, a clinical trial

J Diet Suppl. 2012; **9**:253-261

[Crossref](#)

[Scopus \(10\)](#)

[Google Scholar](#)

153. Hunt, M.J. · Barnetson, R.S.

A comparative study of gluconolactone versus benzoyl peroxide in the treatment of acne

Australas J Dermatol. 1992; **33**:131-134

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

154. Lalla, J.K. · Nandedkar, S.Y. · Paranjape, M.H. ...

Clinical trials of ayurvedic formulations in the treatment of acne vulgaris

J Ethnopharmacol. 2001; **78**:99-102

[Crossref](#)

[Scopus \(14\)](#)

[PubMed](#)

[Google Scholar](#)

Download PDF

155. Paranjpe, P. · Kulkarni, P.H.
Comparative efficacy of four Ayurvedic formulations in the treatment of acne vulgaris: a double-blind randomised placebo-controlled clinical evaluation
J Ethnopharmacol. 1995; **49**:127-132
Crossref Scopus (17) PubMed Google Scholar
-
156. Hughes, H. · Brown, B.W. · Lawlis, G.F. ...
Treatment of acne vulgaris by biofeedback relaxation and cognitive imagery
J Psychosom Res. 1983; **27**:185-191
Crossref Scopus (57) PubMed Google Scholar
-
157. Smith, R.N. · Mann, N.J. · Braue, A. ...
The effect of a high-protein, low glycemic-load diet versus a conventional, high glycemic-load diet on biochemical parameters associated with acne vulgaris: a randomized, investigator-masked, controlled trial
J Am Acad Dermatol. 2007; **57**:247-256
Full Text  Full Text (PDF) Scopus (222) PubMed Google Scholar
-
158. Kwon, H.H. · Yoon, J.Y. · Hong, J.S. ...
Clinical and histological effect of a low glycaemic load diet in treatment of acne vulgaris in Korean patients: a randomized, controlled trial
Acta Derm Venereol. 2012; **92**:241-246
Crossref Scopus (122) PubMed Google Scholar
-
159. Smith, R. · Mann, N. · Makelainen, H. ...
A pilot study to determine the short-term effects of a low glycemic load diet on hormonal markers of acne: a nonrandomized, parallel, controlled feeding trial
Mol Nutr Food Res. 2008; **52**:718-726
Crossref Scopus (86) PubMed Google Scholar
-
160. Preneau, S. · Dessinioti, C. · Nguyen, J.M. ...
Predictive markers of response to isotretinoin in female acne
Eur J Dermatol. 2013; **23**:478-486
Google Scholar
-
161. Ismail, N.H. · Manaf, Z.A. · Azizan, N.Z.
High glycemic load diet, milk and ice cream consumption are related to acne vulgaris in Malaysian young adults: a case control study
BMC Dermatol. 2012; **12**:13
Crossref Scopus (87)  PubMed Google Scholar
-
162. Adebamowo, C.A. · Spiegelman, D. · Berkey, C.S. ...
Download PDF

Milk consumption and acne in adolescent girls

Dermatol Online J. 2006; **12**:1

[PubMed](#) [Google Scholar](#)

-
163. Adebamowo, C.A. · Spiegelman, D. · Berkey, C.S. ...

Milk consumption and acne in teenaged boys

J Am Acad Dermatol. 2008; **58**:787-793

[Full Text](#)  [Full Text \(PDF\)](#) [Scopus \(216\)](#) [PubMed](#) [Google Scholar](#)

-
164. Di Landro, A. · Cazzaniga, S. · Parazzini, F. ...

Family history, body mass index, selected dietary factors, menstrual history, and risk of moderate to severe acne in adolescents and young adults

J Am Acad Dermatol. 2012; **67**:1129-1135

[Full Text](#)  [Full Text \(PDF\)](#) [Scopus \(41\)](#) [PubMed](#) [Google Scholar](#)

-
165. Seirafi, H. · Farnaghi, F. · Vasheghani-Farahani, A. ...

Assessment of androgens in women with adult-onset acne

Int J Dermatol. 2007; **46**:1188-1191

[Crossref](#) [Scopus \(48\)](#) [PubMed](#) [Google Scholar](#)

-
166. Degitz, K. · Placzek, M. · Arnold, B. ...

Congenital adrenal hyperplasia and acne in male patients

Br J Dermatol. 2003; **148**:1263-1266

[Crossref](#) [Scopus \(41\)](#) [PubMed](#) [Google Scholar](#)

-
167. Trapp, C.M. · Oberfield, S.E.

Recommendations for treatment of nonclassic congenital adrenal hyperplasia (NCCAH): an update

Steroids. 2012; **77**:342-346

[Crossref](#) [Scopus \(40\)](#) [PubMed](#) [Google Scholar](#)

-
168. Legro, R.S. · Arslanian, S.A. · Ehrmann, D.A. ...

Diagnosis and treatment of polycystic ovary syndrome: an Endocrine Society clinical practice guideline

J Clin Endocrinol Metab. 2013; **98**:4565-4592

[Crossref](#) [Scopus \(1345\)](#) [PubMed](#) [Google Scholar](#)

-
169. Saleh, B.O.

Role of growth hormone and insulin-like growth factor-I in hyperandrogenism and the severity of acne vulgaris in young males

Saudi Med J. 2012; **33**:1196-1200

 [Download PDF](#)

[Google Scholar](#)

-
170. Del Prete, M. · Mauriello, M.C. · Faggiano, A. ...
Insulin resistance and acne: a new risk factor for men?
Endocrine. 2012; **42**:555-560
[Crossref](#) [Scopus \(74\)](#) [PubMed](#) [Google Scholar](#)
-
171. Cunliffe, W.J. · Dodman, B. · Ead, R.
Benzoyl peroxide in acne
Practitioner. 1978; **220**:479-482
[PubMed](#) [Google Scholar](#)
-
172. Fulton, Jr., J.E. · Farzad-Bakshandeh, A. · Bradley, S.
Studies on the mechanism of action to topical benzoyl peroxide and vitamin A acid in acne vulgaris
J Cutan Pathol. 1974; **1**:191-200
[Crossref](#) [PubMed](#) [Google Scholar](#)
-
173. Padilla, R.S. · McCabe, J.M. · Becker, L.E.
Topical tetracycline hydrochloride vs. topical clindamycin phosphate in the treatment of acne: a comparative study
Int J Dermatol. 1981; **20**:445-448
[Crossref](#) [PubMed](#) [Google Scholar](#)
-
174. Pariser, D.M. · Rich, P. · Cook-Bolden, F.E. ...
An aqueous gel fixed combination of clindamycin phosphate 1.2% and benzoyl peroxide 3.75% for the once-daily treatment of moderate to severe acne vulgaris
J Drugs Dermatol. 2014; **13**:1083-1089
[Google Scholar](#)
-
175. Lucky, A.W. · Cullen, S.I. · Funicella, T. ...
Double-blind, vehicle-controlled, multicenter comparison of two 0.025% tretinoin creams in patients with acne vulgaris
J Am Acad Dermatol. 1998; **38**:S24-S30
[Full Text](#)  [Full Text \(PDF\)](#) [Scopus \(53\)](#) [PubMed](#) [Google Scholar](#)
-
176. Dreno, B. · Bettoli, V. · Ochsendorf, F. ...
Efficacy and safety of clindamycin phosphate 1.2%/tretinoin 0.025% formulation for the treatment of acne vulgaris: pooled analysis of data from three randomised, double-blind, parallel-group, phase III studies
Eur J Dermatol. 2014; **24**:201-209
 [Download PDF](#)

[Google Scholar](#)

-
177. Pedace, F.J. · Stoughton, R.
Topical retinoic acid in acne vulgaris
Br J Dermatol. 1971; **84**:465-469
[Crossref](#) [PubMed](#) [Google Scholar](#)
-
178. Kircik, L.H.
Efficacy and safety of azelaic acid (AzA) gel 15% in the treatment of post-inflammatory hyperpigmentation and acne: a 16-week, baseline-controlled study
J Drugs Dermatol. 2011; **10**:586-590
[PubMed](#) [Google Scholar](#)
-
179. Del Rosso, J.Q. · Kircik, L. · Gallagher, C.J.
Comparative efficacy and tolerability of dapson 5% gel in adult versus adolescent females with acne vulgaris
J Clin Aesthet Dermatol. 2015; **8**:31-37
[Google Scholar](#)
-
180. Shalita, A.R.
Comparison of a salicylic acid cleanser and a benzoyl peroxide wash in the treatment of acne vulgaris
Clin Ther. 1989; **11**:264-267
[PubMed](#) [Google Scholar](#)
-
181. Elstein, W.
Topical deodorized polysulfides. Broadscope acne therapy
Cutis. 1981; **28**:468-472
[PubMed](#) [Google Scholar](#)
-
182. Hurley, H.J. · Shelley, W.B.
Special topical approach to the treatment of acne. Suppression of sweating with aluminum chloride in an anhydrous formulation
Cutis. 1978; **22**:696-703
[PubMed](#) [Google Scholar](#)
-
183. Hjorth, N. · Storm, D. · Dela, K.
Topical anhydrous aluminum chloride formulation in the treatment of acne vulgaris: a double-blind study
Cutis. 1985; **35**:499-500
[PubMed](#) [Google Scholar](#)  [Download PDF](#)

184. Bojar, R.A. · Eady, E.A. · Jones, C.E. ...

Inhibition of erythromycin-resistant propionibacteria on the skin of acne patients by topical erythromycin with and without zinc

Br J Dermatol. 1994; **130**:329-336

[Crossref](#)

[Scopus \(82\)](#)

[PubMed](#)

[Google Scholar](#)

185. Cochran, R.J. · Tucker, S.B. · Flannigan, S.A.

Topical zinc therapy for acne vulgaris

Int J Dermatol. 1985; **24**:188-190

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

186. Stainforth, J. · MacDonald-Hull, S. · Papworth-Smith, J.W. ...

A single-blind comparison of topical erythromycin/zinc lotion and oral minocycline in the treatment of acne vulgaris

J Dermatolog Treat. 1993; **4**:119-122

[Crossref](#)

[Google Scholar](#)

187. Lebrun, C.M.

Rosac cream with sunscreens (sodium sulfacetamide 10% and sulfur 5%)

Skinmed. 2004; **3**:92

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

188. Tarimci, N. · Sener, S. · Kilinc, T.

Topical sodium sulfacetamide/sulfur lotion

J Clin Pharm Ther. 1997; **22**:301

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

189. Thiboutot, D.

New treatments and therapeutic strategies for acne

Arch Fam Med. 2000; **9**:179-187

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

190. Shalita, A.R. · Smith, J.G. · Parish, L.C. ...

Topical nicotinamide compared with clindamycin gel in the treatment of inflammatory acne vulgaris

Int J Dermatol. 1995; **34**:434-437

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

191. Khodaeiani, E. · Fouladi, R.F. · Amirnia, M. ...

Topical 4% nicotinamide vs. 1%  clindamycin in moderate inflammatory acne vulgaris

Int J Dermatol. 2013; **52**:999-1004

[Download PDF](#)

192. Tan, J. · Humphrey, S. · Vender, R. ...

A treatment for severe nodular acne: a randomized investigator-blinded, controlled, noninferiority trial comparing fixed-dose adapalene/benzoyl peroxide plus doxycycline vs. oral isotretinoin

Br J Dermatol. 2014; **171**:1508-1516

193. Zaenglein, A.L. · Shamban, A. · Webster, G. ...

A phase IV, open-label study evaluating the use of triple-combination therapy with minocycline HCl extended-release tablets, a topical antibiotic/retinoid preparation and benzoyl peroxide in patients with moderate to severe acne vulgaris

J Drugs Dermatol. 2013; **12**:619-625

194. Fleischer, Jr., A.B. · Dinehart, S. · Stough, D. ...

Safety and efficacy of a new extended-release formulation of minocycline

Cutis. 2006; **78**:21-31

195. Toossi, P. · Farshchian, M. · Malekzad, F. ...

Subantimicrobial-dose doxycycline in the treatment of moderate facial acne

J Drugs Dermatol. 2008; **7**:1149-1152

196. Moore, A. · Ling, M. · Bucko, A. ...

Efficacy and safety of subantimicrobial dose, modified-release doxycycline 40 mg versus doxycycline 100 mg versus placebo for the treatment of inflammatory lesions in moderate and severe acne: a randomized, double-blinded, controlled study

J Drugs Dermatol. 2015; **14**:581-586

197. Maleszka, R. · Turek-Urasinska, K. · Oremus, M. ...

Pulsed azithromycin treatment is as effective and safe as 2-week-longer daily doxycycline treatment of acne vulgaris: a randomized, double-blind, noninferiority study

Skinmed. 2011; **9**:86-94

198. Antonio, J.R. · Pegas, J.R. · Cestari, T.F. ...

Azithromycin pulses in the treatment of inflammatory and pustular acne: efficacy, tolerability and safety



Download PDF

199. Innocenzi, D. · Skroza, N. · Ruggiero, A. ...

Moderate acne vulgaris: efficacy, tolerance and compliance of oral azithromycin thrice weekly for

Acta Dermatovenerol Croat. 2008; 16:13-18

[Google Scholar](#)

200. Bardazzi, F. · Savoia, F. · Parente, G. ...

Azithromycin: a new therapeutical strategy for acne in adolescents

Dermatol Online J. 2007; 13:4

[Google Scholar](#)

201. Basta-Juzbasic, A. · Lipozencic, J. · Oremovic, L. ...

A dose-finding study of azithromycin in the treatment of acne vulgaris

Acta Dermatovenerol Croat. 2007; 15:141-147

[Google Scholar](#)

202. Kus, S. · Yucelten, D. · Aytug, A.

Comparison of efficacy of azithromycin vs. doxycycline in the treatment of acne vulgaris

Clin Exp Dermatol. 2005; 30:215-220

[Crossref](#)

[Scopus \(65\)](#)

[PubMed](#)

[Google Scholar](#)

203. Parsad, D. · Pandhi, R. · Nagpal, R. ...

Azithromycin monthly pulse vs daily doxycycline in the treatment of acne vulgaris

J Dermatol. 2001; 28:1-4

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

204. Gruber, F. · Grubisic-Greblo, H. · Kastelan, M. ...

Azithromycin compared with minocycline in the treatment of acne comedonica and papulo-pustulosa

J Chemother. 1998; 10:469-473

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

205. Ullah, G. · Noor, S.M. · Bhatti, Z. ...

Comparison of oral azithromycin with oral doxycycline in the treatment of acne vulgaris

J Ayub Med Coll Abbottabad. 2014; 26:64-67

[Google Scholar](#)



206. Shaughnessy, K.K. · Bouchard, S.M. · Mohr, M.R. ...

[Download PDF](#)

Minocycline-induced drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome with persistent myocarditis

J Am Acad Dermatol. 2010; **62**:315-318

[Full Text](#)



[Full Text \(PDF\)](#)

[Scopus \(73\)](#)

[PubMed](#)

[Google Scholar](#)

-
207. Smith, K. · Leyden, J.J.

Safety of doxycycline and minocycline: a systematic review

Clin Ther. 2005; **27**:1329-1342

[Abstract](#)



[Full Text \(PDF\)](#)

[Scopus \(293\)](#)

[PubMed](#)

[Google Scholar](#)

-
208. Tripathi, S.V. · Gustafson, C.J. · Huang, K.E. ...

Side effects of common acne treatments

Exp Opin Drug Saf. 2013; **12**:39-51

[Crossref](#)

[Scopus \(0\)](#)

[PubMed](#)

[Google Scholar](#)

-
209. Weinstein, M. · Laxer, R. · Debosz, J. ...

Doxycycline-induced cutaneous inflammation with systemic symptoms in a patient with acne vulgaris

J Cutan Med Surg. 2013; **17**:283-286

[Crossref](#)

[Google Scholar](#)

-
210. Firoz, B.F. · Henning, J.S. · Zarzabal, L.A. ...

Toxic epidermal necrolysis: five years of treatment experience from a burn unit

J Am Acad Dermatol. 2012; **67**:630-635

[Full Text](#)



[Full Text \(PDF\)](#)

[Scopus \(78\)](#)

[PubMed](#)

[Google Scholar](#)

-
211. Roujeau, J.C. · Kelly, J.P. · Naldi, L. ...

Medication use and the risk of Stevens-Johnson syndrome or toxic epidermal necrolysis

N Engl J Med. 1995; **333**:1600-1607

[Crossref](#)

[Scopus \(1285\)](#)

[PubMed](#)

[Google Scholar](#)

-
212. Thiboutot, D.M. · Shalita, A.R. · Yamauchi, P.S. ...

Adapalene gel, 0.1%, as maintenance therapy for acne vulgaris: a randomized, controlled, investigator-blind follow-up of a recent combination study

Arch Dermatol. 2006; **142**:597-602

[Crossref](#)

[Scopus \(98\)](#)

[PubMed](#)

[Google Scholar](#)

-
213. Poulin, Y. · Sanchez, N.P. · Bucko, A. ...

A 6-month maintenance therapy with adapalene-benzoyl peroxide gel prevents relapse and continuously improves efficacy among patients with severe acne vulgaris: results of a randomized controlled trial



[Download PDF](#)

Br J Dermatol. 2011; **164**:1376-1382

[Crossref](#)[Scopus \(71\)](#)[PubMed](#)[Google Scholar](#)

214. Tan, J. · Stein Gold, L. · Schlessinger, J. ...

Short-term combination therapy and long-term relapse prevention in the treatment of severe acne vulgaris

J Drugs Dermatol. 2012; **11**:174-180

[PubMed](#) [Google Scholar](#)

215. Moon, S.H. · Roh, H.S. · Kim, Y.H. ...

Antibiotic resistance of microbial strains isolated from Korean acne patients

J Dermatol. 2012; **39**:833-837

[Crossref](#) [Scopus \(62\)](#) [PubMed](#) [Google Scholar](#)

216. Margolis, D.J. · Fanelli, M. · Kupperman, E. ...

Association of pharyngitis with oral antibiotic use for the treatment of acne: a cross-sectional and prospective cohort study

Arch Dermatol. 2012; **148**:326-332

[Crossref](#) [Scopus \(42\)](#) [PubMed](#) [Google Scholar](#)

217. Bartlett, J.G. · Chang, T.W. · Gurwith, M. ...

Antibiotic-associated pseudomembranous colitis due to toxin-producing clostridia

N Engl J Med. 1978; **298**:531-534

[Crossref](#) [PubMed](#) [Google Scholar](#)

218. Carroll, K.C. · Bartlett, J.G.

Biology of *Clostridium difficile*: implications for epidemiology and diagnosis

Annu Rev Microbiol. 2011; **65**:501-521

[Crossref](#) [Scopus \(196\)](#) [PubMed](#) [Google Scholar](#)

219. Arrington, E.A. · Patel, N.S. · Gerancher, K. ...

Combined oral contraceptives for the treatment of acne: a practical guide

Cutis. 2012; **90**:83-90

[Google Scholar](#)

220. Davtyan, C.

Four generations of progestins in oral contraceptives

Proceedings of UCLA Healthcare. 2012; **16** Available at:

www.med.ucla.edu/modules/xfsection/download.php?fileid=638

Accessed January 5, 2016

[Google Scholar](#)

Download PDF

- 221.** Awojolu, A.O. · Gallo, M.F. · Lopez, L.M. ...
Combined oral contraceptive pills for treatment of acne
Cochrane Database Syst Rev. 2012; CD004425
[Google Scholar](#)
- 222.** Harper, J.C.
Should dermatologists prescribe hormonal contraceptives for acne?
Dermatol Ther. 2009; **22**:452-457
[Crossref](#) [Scopus \(26\)](#) [PubMed](#) [Google Scholar](#)
- 223.** Rabe, T. · Kowald, A. · Ortmann, J. ...
Inhibition of skin 5 alpha-reductase by oral contraceptive progestins in vitro
Gynecol Endocrinol. 2000; **14**:223-230
[Crossref](#) [PubMed](#) [Google Scholar](#)
- 224.** Koltun, W. · Lucky, A.W. · Thiboutot, D. ...
Efficacy and safety of 3 mg drospirenone/20 mcg ethinylestradiol oral contraceptive administered in 24/4 regimen in the treatment of acne vulgaris: a randomized, double-blind, placebo-controlled trial
Contraception. 2008; **77**:249-256
[Full Text](#)  [Full Text \(PDF\)](#) [Scopus \(79\)](#) [PubMed](#) [Google Scholar](#)
- 225.** Koltun, W. · Maloney, J.M. · Marr, J. ...
Treatment of moderate acne vulgaris using a combined oral contraceptive containing ethinylestradiol 20 mug plus drospirenone 3 mg administered in a 24/4 regimen: a pooled analysis
Eur J Obstet Gynecol Reprod Biol. 2011; **155**:171-175
[Full Text](#)  [Full Text \(PDF\)](#) [Scopus \(38\)](#) [PubMed](#) [Google Scholar](#)
- 226.** Jaisamrarn, U. · Chaovisitsaree, S. · Angsuwathana, S. ...
A comparison of multiphasic oral contraceptives containing norgestimate or desogestrel in acne treatment: a randomized trial
Contraception. 2014; **90**:535-541
[Full Text](#)  [Full Text \(PDF\)](#) [Google Scholar](#)
- 227.** Palli, M.B. · Reyes-Habito, C.M. · Lima, X.T. ...
A single-center, randomized double-blind, parallel-group study to examine the safety and efficacy of 3mg drospirenone/0.02 mg ethinyl estradiol compared with placebo in the treatment of moderate truncal acne vulgaris
J Drugs Dermatol. 2013; **12**:633-637
[Google Scholar](#) 

228. George, R. · Clarke, S. · Thiboutot, D.

Hormonal therapy for acne

Semin Cutan Med Surg. 2008; **27**:188-196

[Crossref](#)

[Scopus \(90\)](#)

[PubMed](#)

[Google Scholar](#)

229. The American College of Obstetricians and Gynecologists website. Committee opinion 540. Risk of venous thromboembolism among users of drospirenone-containing oral contraceptive pills.

Available at: <http://www.acog.org/Resources-And-Publications/Committee-Opinions/Committee-on-Gynecologic-Practice/Risk-of-Venous-Thromboembolism>. Accessed January 6, 2016.

[Google Scholar](#)

230. US Food and Drug Administration website. Combined hormonal contraceptives (CHCs) and the risk of cardiovascular disease endpoints. Available at: <http://www.fda.gov/downloads/Drugs/Dru gSafety/UCM277384.pdf>. Accessed January 6, 2016.

[Google Scholar](#)

231. Acute myocardial infarction and combined oral contraceptives: results of an international multicentre case-control study. WHO Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception

Lancet. 1997; **349**:1202-1209

[Full Text](#)



[Full Text \(PDF\)](#)

[PubMed](#)

[Google Scholar](#)

232. Katsambas, A.D. · Dessinioti, C.

Hormonal therapy for acne: why not as first line therapy? facts and controversies

Clin Dermatol. 2010; **28**:17-23

[Full Text](#)



[Full Text \(PDF\)](#)

[Scopus \(45\)](#)

[PubMed](#)

[Google Scholar](#)

233. Breast cancer and hormone replacement therapy: collaborative reanalysis of data from 51 epidemiological studies of 52,705 women with breast cancer and 108,411 women without breast cancer. Collaborative Group on Hormonal Factors in Breast Cancer

Lancet. 1997; **350**:1047-1059

[Full Text](#)



[Full Text \(PDF\)](#)

[Scopus \(2526\)](#)

[PubMed](#)

[Google Scholar](#)

234. Gierisch, J.M. · Coeytaux, R.R. · Urrutia, R.P. ...

Oral contraceptive use and risk of breast, cervical, colorectal, and endometrial cancers: a systematic review

Cancer Epidemiol Biomarkers Prev. 2013; **22**:1931-1943

[Crossref](#)

[Scopus \(274\)](#)

[PubMed](#)

[Google Scholar](#)

235. International Collaboration of Epidemiological Studies of Cervical Cancer · Appleby, P. · Beral, V.

...



Cervical cancer and hormonal contraceptives: collaborative reanalysis of individual data for 16,573 women with cervical cancer and 35,509 women without cervical cancer from 24

[Download PDF](#)

epidemiological studies

Lancet. 2007; **370**:1609-1621

[Full Text](#)



[Full Text \(PDF\)](#)

[Scopus \(411\)](#)

[PubMed](#)

[Google Scholar](#)

-
236. Lloyd, T. · Rollings, N. · Andon, M.B. ...

Determinants of bone density in young women. I. Relationships among pubertal development, total body bone mass, and total body bone density in premenarchal females

J Clin Endocrinol Metab. 1992; **75**:383-387

[Crossref](#)

[Scopus \(84\)](#)

[PubMed](#)

[Google Scholar](#)

-
237. Cromer, B.A. · Bonny, A.E. · Stager, M. ...

Bone mineral density in adolescent females using injectable or oral contraceptives: a 24-month prospective study

Fertil Steril. 2008; **90**:2060-2067

[Full Text](#)



[Full Text \(PDF\)](#)

[Scopus \(101\)](#)

[PubMed](#)

[Google Scholar](#)

-
238. Lloyd, T. · Petit, M.A. · Lin, H.M. ...

Lifestyle factors and the development of bone mass and bone strength in young women

J Pediatr. 2004; **144**:776-782

[Full Text](#)



[Full Text \(PDF\)](#)

[Scopus \(80\)](#)

[PubMed](#)

[Google Scholar](#)

-
239. Maguire, K. · Westhoff, C.

The state of hormonal contraception today: established and emerging noncontraceptive health benefits

Am J Obstet Gynecol. 2011; **205**:S4-S8

[Full Text](#)



[Full Text \(PDF\)](#)

[Scopus \(92\)](#)

[PubMed](#)

[Google Scholar](#)

-
240. ACOG Committee on Practice Bulletins-Gynecology

ACOG practice bulletin. No. 73: Use of hormonal contraception in women with coexisting medical conditions

Obstet Gynecol. 2006; **107**:1453-1472

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

-
241. Helms, S.E. · Bredle, D.L. · Zajic, J. ...

Oral contraceptive failure rates and oral antibiotics

J Am Acad Dermatol. 1997; **36**:705-710

[Abstract](#)



[Full Text \(PDF\)](#)

[Scopus \(86\)](#)

[PubMed](#)

[Google Scholar](#)

-
242. London, B.M. · Lookingbill, D.P.

Frequency of pregnancy in acne patients taking oral antibiotics and oral contraceptives

Arch Dermatol. 1994; **130**:392-393

[Download PDF](#)

[Crossref](#)[PubMed](#)[Google Scholar](#)

243. Krunic, A. · Ciurea, A. · Scheman, A.

Efficacy and tolerance of acne treatment using both spironolactone and a combined contraceptive containing drospirenone

J Am Acad Dermatol. 2008; **58**:60-62

[Full Text](#)[Full Text \(PDF\)](#)[Scopus \(64\)](#)[PubMed](#)[Google Scholar](#)

244. Stewart, F.H. · Harper, C.C. · Ellertson, C.E. ...

Clinical breast and pelvic examination requirements for hormonal contraception: current practice vs evidence

JAMA. 2001; **285**:2232-2239

[Crossref](#)[PubMed](#)[Google Scholar](#)

245. Cusan, L. · Dupont, A. · Gomez, J.L. ...

Comparison of flutamide and spironolactone in the treatment of hirsutism: a randomized controlled trial

Fertil Steril. 1994; **61**:281-287

[Abstract](#)[Full Text \(PDF\)](#)[PubMed](#)[Google Scholar](#)

246. Boisselle, A. · Dionne, F.T. · Tremblay, R.R.

Interaction of spironolactone with rat skin androgen receptor

Can J Biochem. 1979; **57**:1042-1046

[Crossref](#)[PubMed](#)[Google Scholar](#)

247. Menard, R.H. · Martin, H.F. · Stripp, B. ...

Spironolactone and cytochrome P-450: impairment of steroid hydroxylation in the adrenal cortex

Life Sci. 1974; **15**:1639-1648

[Crossref](#)[PubMed](#)[Google Scholar](#)

248. Menard, R.H. · Stripp, B. · Gillette, J.R.

Spironolactone and testicular cytochrome P-450: decreased testosterone formation in several species and changes in hepatic drug metabolism

Endocrinology. 1974; **94**:1628-1636

[Crossref](#)[PubMed](#)[Google Scholar](#)

249. Rifka, S.M. · Pita, J.C. · Vigersky, R.A. ...

Interaction of digitalis and spironolactone with human sex steroid receptors

J Clin Endocrinol Metab. 1978; **46**:338-344

[Crossref](#)[PubMed](#)[Google Scholar](#)[Download PDF](#)

250. Zouboulis, C.C. · Akamatsu, H. · Stephanek, K. ...

Androgens affect the activity of human sebocytes in culture in a manner dependent on the localization of the sebaceous glands and their effect is antagonized by spironolactone

Skin Pharmacol. 1994; **7**:33-40

[Crossref](#) [PubMed](#) [Google Scholar](#)

251. Serafini, P.C. · Catalino, J. · Lobo, R.A.

The effect of spironolactone on genital skin 5 alpha-reductase activity

J Steroid Biochem. 1985; **23**:191-194

[Crossref](#) [Scopus \(50\)](#) [PubMed](#) [Google Scholar](#)

252. Muhlemann, M.F. · Carter, G.D. · Cream, J.J. ...

Oral spironolactone: an effective treatment for acne vulgaris in women

Br J Dermatol. 1986; **115**:227-232

[Crossref](#) [PubMed](#) [Google Scholar](#)

253. Goodfellow, A. · Alaghband-Zadeh, J. · Carter, G. ...

Oral spironolactone improves acne vulgaris and reduces sebum excretion

Br J Dermatol. 1984; **111**:209-214

[Crossref](#) [PubMed](#) [Google Scholar](#)

254. Brown, J. · Farquhar, C. · Lee, O. ...

Spironolactone versus placebo or in combination with steroids for hirsutism and/or acne

Cochrane Database Syst Rev. 2009; CD000194

[PubMed](#) [Google Scholar](#)

255. Shaw, J.C. · White, L.E.

Long-term safety of spironolactone in acne: results of an 8-year followup study

J Cutan Med Surg. 2002; **6**:541-545

[Crossref](#) [Scopus \(76\)](#) [PubMed](#) [Google Scholar](#)

256. Plovanich, M. · Weng, Q.Y. · Mostaghimi, A.

Low usefulness of potassium monitoring among healthy young women taking spironolactone for acne

JAMA Dermatol. 2015; **151**:941-944

[Crossref](#) [Scopus \(0\)](#) [Google Scholar](#)

257. Zeichner, J.A.

Evaluating and treating the adult female patient with acne

J Drugs Dermatol. 2013; **12**:1416-1427

[PubMed](#) [Google Scholar](#) [Download PDF](#)

258. Loube, S.D. · Quirk, R.A.

Letter: breast cancer associated with administration of spironolactone

Lancet. 1975; **1**:1428-1429

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

259. Danielson, D.A. · Jick, H. · Hunter, J.R. ...

Nonestrogenic drugs and breast cancer

Am J Epidemiol. 1982; **116**:329-332

[PubMed](#)

[Google Scholar](#)

260. Friedman, G.D. · Ury, H.K.

Initial screening for carcinogenicity of commonly used drugs

J Natl Cancer Inst. 1980; **65**:723-733

[PubMed](#)

[Google Scholar](#)

261. Mackenzie, I.S. · Macdonald, T.M. · Thompson, A. ...

Spironolactone and risk of incident breast cancer in women older than 55 years: retrospective, matched cohort study

BMJ. 2012; **345**:e4447

[Crossref](#)

[Scopus \(58\)](#)

[PubMed](#)

[Google Scholar](#)

262. Biggar, R.J. · Andersen, E.W. · Wohlfahrt, J. ...

Spironolactone use and the risk of breast and gynecologic cancers

Cancer Epidemiol. 2013; **37**:870-875

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

263. Cusan, L. · Dupont, A. · Belanger, A. ...

Treatment of hirsutism with the pure antiandrogen flutamide

J Am Acad Dermatol. 1990; **23**:462-469

[Abstract](#)



[Full Text \(PDF\)](#)

[PubMed](#)

[Google Scholar](#)

264. Muderris, II · Bayram, F. · Guven, M.

Treatment of hirsutism with lowest-dose flutamide (62.5 mg/day)

Gynecol Endocrinol. 2000; **14**:38-41

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

265. Carmina, E. · Lobo, R.A.

A comparison of the relative efficacy of antiandrogens for the treatment of acne in hyperandrogenic women

Clin Endocrinol. 2002; **57**:231-234



[Crossref](#)

[Scopus \(65\)](#)

[Download](#)

[PubMed](#)

[Google Scholar](#)

- 266.** Adalatkhah, H. · Pourfarzi, F. · Sadeghi-Bazargani, H.
Flutamide versus a cyproterone acetate-ethinyl estradiol combination in moderate acne: a pilot randomized clinical trial
Clin Cosmet Investig Dermatol. 2011; **4**:117-121
[Crossref](#) [Google Scholar](#)
- 267.** Calaf, J. · Lopez, E. · Millet, A. ...
Long-term efficacy and tolerability of flutamide combined with oral contraception in moderate to severe hirsutism: a 12-month, double-blind, parallel clinical trial
J Clin Endocrinol Metab. 2007; **92**:3446-3452
[Crossref](#) [Scopus \(68\)](#) [PubMed](#) [Google Scholar](#)
- 268.** Wysowski, D.K. · Freiman, J.P. · Tourtelot, J.B. ...
Fatal and nonfatal hepatotoxicity associated with flutamide
Ann Intern Med. 1993; **118**:860-864
[Crossref](#) [PubMed](#) [Google Scholar](#)
- 269.** Garcia Cortes, M. · Andrade, R.J. · Lucena, M.I. ...
Flutamide-induced hepatotoxicity: report of a case series
Rev Esp Enferm Dig. 2001; **93**:423-432
[PubMed](#) [Google Scholar](#)
- 270.** Saihan, E.M. · Burton, J.L.
Sebaceous gland suppression in female acne patients by combined glucocorticoid-oestrogen therapy
Br J Dermatol. 1980; **103**:139-142
[Crossref](#) [PubMed](#) [Google Scholar](#)
- 271.** Darley, C.R. · Moore, J.W. · Besser, G.M. ...
Low dose prednisolone or oestrogen in the treatment of women with late onset or persistent acne vulgaris
Br J Dermatol. 1982; **108**:345-353
[Crossref](#) [Scopus \(5\)](#) [Google Scholar](#)
- 272.** Jansen, T. · Plewig, G.
Acne fulminans
Int J Dermatol. 1998; **37**:254-257
[Crossref](#) [Scopus \(31\)](#) [PubMed](#) [Google Scholar](#)
- 273.** Karvonen, S.L.
Acne fulminans: report of clinical findings and treatment of twenty-four patients
[Download PDF](#)



274. Chivot, M. · Midoun, H.

Isotretinoin and acne—a study of relapses

Dermatologica. 1990; **180**:240-243

Crossref

PubMed

Google Scholar

275. Goulden, V. · Clark, S.M. · McGeown, C. ...

Treatment of acne with intermittent isotretinoin

Br J Dermatol. 1997; **137**:106-108

Crossref

PubMed

Google Scholar

276. King, K. · Jones, D.H. · Daltrey, D.C. ...

A double-blind study of the effects of 13-cis-retinoic acid on acne, sebum excretion rate and microbial population

Br J Dermatol. 1982; **107**:583-590

Crossref

PubMed

Google Scholar

277. Lester, R.S. · Schachter, G.D. · Light, M.J.

Isotretinoin and tetracycline in the management of severe nodulocystic acne

Int J Dermatol. 1985; **24**:252-257

Crossref

PubMed

Google Scholar

278. Blasiak, R.C. · Stamey, C.R. · Burkhart, C.N. ...

High-dose isotretinoin treatment and the rate of relapse, relapse, and adverse effects in patients with acne vulgaris

JAMA Dermatol. 2013; **149**:1392-1398

Crossref

Scopus (98)

PubMed

Google Scholar

279. De, D. · Kanwar, A.J.

Combination of low-dose isotretinoin and pulsed oral azithromycin in the management of moderate to severe acne: a preliminary open-label, prospective, non-comparative, single-centre study

Clin Drug Investig. 2011; **31**:599-604

Crossref

Scopus (3)

Google Scholar

280. Lee, J.J. · Feng, L. · Reshef, D.S. ...

Mortality in the randomized, controlled lung intergroup trial of isotretinoin

Cancer Prev Res (Phila). 2010; **3**:728-744

Crossref

Scopus (0)

PubMed

Download PDF

Google Scholar

281. Bernstein, C.N. · Nugent, Z. · Longobardi, T. ...
Isotretinoin is not associated with inflammatory bowel disease: a population-based case-control study
Am J Gastroenterol. 2009; **104**:2774-2778
[Crossref](#) [Scopus \(102\)](#) [PubMed](#) [Google Scholar](#)
-
282. Dubeau, M.F. · Iacucci, M. · Beck, P.L. ...
Drug-induced inflammatory bowel disease and IBD-like conditions
Inflamm Bowel Dis. 2013; **19**:445-456
[Crossref](#) [Scopus \(7\)](#) [Google Scholar](#)
-
283. Rashtak, S. · Khaleghi, S. · Pittelkow, M.R. ...
Isotretinoin exposure and risk of inflammatory bowel disease
JAMA Dermatol. 2014; **150**:1322-1326
[Crossref](#) [Scopus \(2\)](#) [Google Scholar](#)
-
284. American Academy of Dermatology website. Position statement on isotretinoin. Available at: <http://www.aad.org/Forms/Policies/Uploads/PS/PS-Isotretinoin.pdf>. Accessed January 6, 2016.
[Google Scholar](#)
-
285. Marqueling, A.L. · Zane, L.T.
Depression and suicidal behavior in acne patients treated with isotretinoin: a systematic review
Semin Cutan Med Surg. 2005; **24**:92-102
[Crossref](#) [Scopus \(78\)](#) [PubMed](#) [Google Scholar](#)
-
286. Hull, S.M. · Cunliffe, W.J. · Hughes, B.R.
Treatment of the depressed and dysmorphophobic acne patient
Clin Exp Dermatol. 1991; **16**:210-211
[Crossref](#) [PubMed](#) [Google Scholar](#)
-
287. Myhill, J.E. · Leichtman, S.R. · Burnett, J.W.
Self-esteem and social assertiveness in patients receiving isotretinoin treatment for cystic acne
Cutis. 1988; **41**:171-173
[Google Scholar](#)
-
288. Ormerod, A.D. · Thind, C.K. · Rice, S.A. ...
Influence of isotretinoin on hippocampal-based learning in human subjects
Psychopharmacology. 2012; **221**:667-674
[Crossref](#) [Scopus \(8\)](#) [Google Scholar](#)
-
289. Luthi, F. · Egel, Y. · Theumer, N.
[Download PDF](#) 

Premature epiphyseal closure in an adolescent treated by retinoids for acne: an unusual cause of anterior knee pain

Joint Bone Spine. 2012; **79**:314-316

[Crossref](#)

[Scopus \(2\)](#)

[Google Scholar](#)

290. Steele, R.G. · Lugg, P. · Richardson, M.

Premature epiphyseal closure secondary to single-course vitamin A therapy

Aust N Z J Surg. 1999; **69**:825-827

[Crossref](#)

[Google Scholar](#)

291. Lammer, E.J. · Chen, D.T. · Hoar, R.M. ...

Retinoic acid embryopathy

N Engl J Med. 1985; **313**:837-841

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

292. McElwee, N.E. · Schumacher, M.C. · Johnson, S.C. ...

An observational study of isotretinoin recipients treated for acne in a health maintenance organization

Arch Dermatol. 1991; **127**:341-346

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

293. Rubenstein, R. · Roenigk, Jr., H.H. · Stegman, S.J. ...

Atypical keloids after dermabrasion of patients taking isotretinoin

J Am Acad Dermatol. 1986; **15**:280-285

[Abstract](#)



[Full Text \(PDF\)](#)

[PubMed](#)

[Google Scholar](#)

294. Zachariae, H.

Delayed wound healing and keloid formation following argon laser treatment or dermabrasion during isotretinoin treatment

Br J Dermatol. 1988; **118**:703-706

[Crossref](#)

[PubMed](#)

[Google Scholar](#)

295. Bagatin, E. · Parada, M.O. · Miot, H.A. ...

A randomized and controlled trial about the use of oral isotretinoin for photoaging

Int J Dermatol. 2010; **49**:207-214

[Crossref](#)

[Scopus \(0\)](#)

[PubMed](#)

[Google Scholar](#)

296. Picosse, F.R. · Yarak, S. · Cabral, N.C. ...

Early chemabrasion for acne scars after treatment with oral isotretinoin

Dermatol Surg. 2012; **38**:1521-1526



[Crossref](#)

[Scopus \(7\)](#)

[Download PDF](#)



[Google Scholar](#)

- 297.** Chandrashekhar, B.S. · Varsha, D.V. · Vasanth, V. ...
Safety of performing invasive acne scar treatment and laser hair removal in patients on oral isotretinoin: a retrospective study of 110 patients
Int J Dermatol. 2014; **53**:1281-1285
[Crossref](#) [Google Scholar](#)
-
- 298.** Kim, H.W. · Chang, S.E. · Kim, J.E. ...
The safe delivery of fractional ablative carbon dioxide laser treatment for acne scars in Asian patients receiving oral isotretinoin
Dermatol Surg. 2014; **40**:1361-1366
[Crossref](#) [Scopus \(46\)](#) [PubMed](#) [Google Scholar](#)
-
- 299.** Yoon, J.H. · Park, E.J. · Kwon, I.H. ...
Concomitant use of an infrared fractional laser with low-dose isotretinoin for the treatment of acne and acne scars
J Dermatolog Treat. 2014; **25**:142-146
[Crossref](#) [Scopus \(6\)](#) [Google Scholar](#)
-
- 300.** Basak, P.Y. · Cetin, E.S. · Gurses, I. ...
The effects of systemic isotretinoin and antibiotic therapy on the microbial floras in patients with acne vulgaris
J Eur Acad Dermatol Venereol. 2013; **27**:332-336
[Crossref](#) [Scopus \(3\)](#) [Google Scholar](#)
-
- 301.** Williams, R.E. · Doherty, V.R. · Perkins, W. ...
Staphylococcus aureus and intra-nasal mupirocin in patients receiving isotretinoin for acne
Br J Dermatol. 1992; **126**:362-366
[Crossref](#) [PubMed](#) [Google Scholar](#)
-
- 302.** Dai, W.S. · LaBraico, J.M. · Stern, R.S.
Epidemiology of isotretinoin exposure during pregnancy
J Am Acad Dermatol. 1992; **26**:599-606
[Abstract](#)  [Full Text \(PDF\)](#) [PubMed](#) [Google Scholar](#)
-
- 303.** Atzori, L. · Brundu, M.A. · Orru, A. ...
Glycolic acid peeling in the treatment of acne
J Eur Acad Dermatol Venereol. 1999; **12**:119-122
[Crossref](#) [PubMed](#) [Google Scholar](#)
-
- 304.** Levesque, A. · Hamzavi, I. · Seite, S. ...
Randomized trial comparing a chemical peel containing a lipophilic hydroxy acid derivative of salicylic acid with a salicylic acid peel in subjects with comedonal acne
 [Download PDF](#)

[305.](#) Pollock, B. · Turner, D. · Stringer, M.R. ...

Topical aminolevulinic acid-photodynamic therapy for the treatment of acne vulgaris: a study of clinical efficacy and mechanism of action

Br J Dermatol. 2004; **151**:616-622

[Crossref](#)

[Scopus \(237\)](#)

[PubMed](#)

[Google Scholar](#)

[306.](#) Gold, M.H. · Bradshaw, V.L. · Boring, M.M. ...

The use of a novel intense pulsed light and heat source and ALA-PDT in the treatment of moderate to severe inflammatory acne vulgaris

J Drugs Dermatol. 2004; **3**:S15-S19

[PubMed](#)

[Google Scholar](#)

[307.](#) Wang, X.L. · Wang, H.W. · Zhang, L.L. ...

Topical ALA PDT for the treatment of severe acne vulgaris

Photodiagnosis Photodyn Ther. 2010; **7**:33-38

[Crossref](#)

[Scopus \(28\)](#)

[PubMed](#)

[Google Scholar](#)

[308.](#) Ma, L. · Xiang, L.H. · Yu, B. ...

Low-dose topical 5-aminolevulinic acid photodynamic therapy in the treatment of different severity of acne vulgaris

Photodiagnosis Photodyn Ther. 2013; **10**:583-590

[Crossref](#)

[Scopus \(0\)](#)

[PubMed](#)

[Google Scholar](#)

[309.](#) Lee, S.J. · Hyun, M.Y. · Park, K.Y. ...

A tip for performing intralesional triamcinolone acetonide injections in acne patients

J Am Acad Dermatol. 2014; **71**:e127-e128

[Full Text](#)



[Full Text \(PDF\)](#)

[PubMed](#)

[Google Scholar](#)

[310.](#) Kim, K.S. · Kim, Y.B.

Anti-inflammatory effect of Keigai-rengyo-to extract and acupuncture in male patients with acne vulgaris: a randomized controlled pilot trial

J Altern Complement Med. 2012; **18**:501-508

[Crossref](#)

[Scopus \(5\)](#)

[Google Scholar](#)

[311.](#) Burris, J. · Rietkerk, W. · Woolf, K.

Relationships of self-reported dietary factors and perceived acne severity in a cohort of New York young adults

J Acad Nutr Diet. 2014; **114**:384-392

[Download PDF](#)

[Full Text](#)[Full Text \(PDF\)](#)[Scopus \(66\)](#)[PubMed](#)[Google Scholar](#)

312. Adebamowo, C.A. · Spiegelman, D. · Danby, F.W. ...

High school dietary dairy intake and teenage acne

J Am Acad Dermatol. 2005; **52**:207-214

[Full Text](#)[Full Text \(PDF\)](#)[PubMed](#)[Google Scholar](#)

313. Sardana, K. · Garg, V.K.

An observational study of methionine-bound zinc with antioxidants for mild to moderate acne vulgaris

Derm Ther. 2010; **23**:411-418

[Crossref](#)[Scopus \(7\)](#)[Google Scholar](#)

314. Jung, G.W. · Tse, J.E. · Guiha, I. ...

Prospective, randomized, open-label trial comparing the safety, efficacy, and tolerability of an acne treatment regimen with and without a probiotic supplement and minocycline in subjects with mild to moderate acne

J Cutan Med Surg. 2013; **17**:114-122

[Crossref](#)[Scopus \(111\)](#)[PubMed](#)[Google Scholar](#)

315. Khayef, G. · Young, J. · Burns-Whitmore, B. ...

Effects of fish oil supplementation on inflammatory acne

Lipids Health Dis. 2012; **11**:165

[Crossref](#)[Scopus \(3\)](#)[Google Scholar](#)

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Hair loss

Author: Honorary Associate Professor Amanda Oakley, Dermatologist, Hamilton, New Zealand, 1998. Further updates: December 2015 and May 2023.

What is hair loss?

The medical term for hair loss is alopecia. There may be associated scalp disease or scarring.

Alopecia may be localised or **diffuse**.

It can affect the scalp or other parts of the body.

It may be due to hair shedding, poor quality hair, or hair thinning.

There may be areas of skin that are completely bald.

There may be associated skin disease or scarring.

Unfortunately, hair loss may not be easy to remedy.

Who gets hair loss?

As all our hair follicles are formed during fetal growth, it is inevitable that we will notice hair loss of some kind in later life.

Hair loss occurs in:

Males and females

Children and adults

People with any colour or type of hair.

Hair loss can be an isolated problem or associated with another disease or condition. It can be temporary or permanent, depending on the cause.

How does hair grow?

Hair grows on most parts of the skin surface, except palms, soles, lips and eyelids. Hair thickness and length varies according to the site.

Vellus hair is fine, light in colour, and short in length

Terminal or androgenic hair is thicker, darker and longer

A hair shaft grows within a follicle at a rate of about 1 cm per month. It is due to cell division within the hair bulb at the base of the follicle. The cells produce the three layers of the hair shaft (medulla, cortex, cuticle), which are mainly made of the protein keratin (which is also the main structure of skin and nails).

Hair growth follows a cycle. However, these phases are not synchronised, and any hair may be at a particular phase at random.

The three main phases of the hair cycle are:

1. Anagen: actively growing hair, most of them
2. Catagen: in-between phase of 2–3 weeks when growth stops and the follicle shrinks, 1–3% of hairs
3. Telogen: resting phase for 1–4 months, up to 10% of hairs in a normal scalp.

Hair length depends on the duration of anagen. Short hairs (eyelashes, eyebrows, hair on arms and legs) have a short anagen phase of around one month. Anagen lasts up to 6 years or longer in scalp hair.

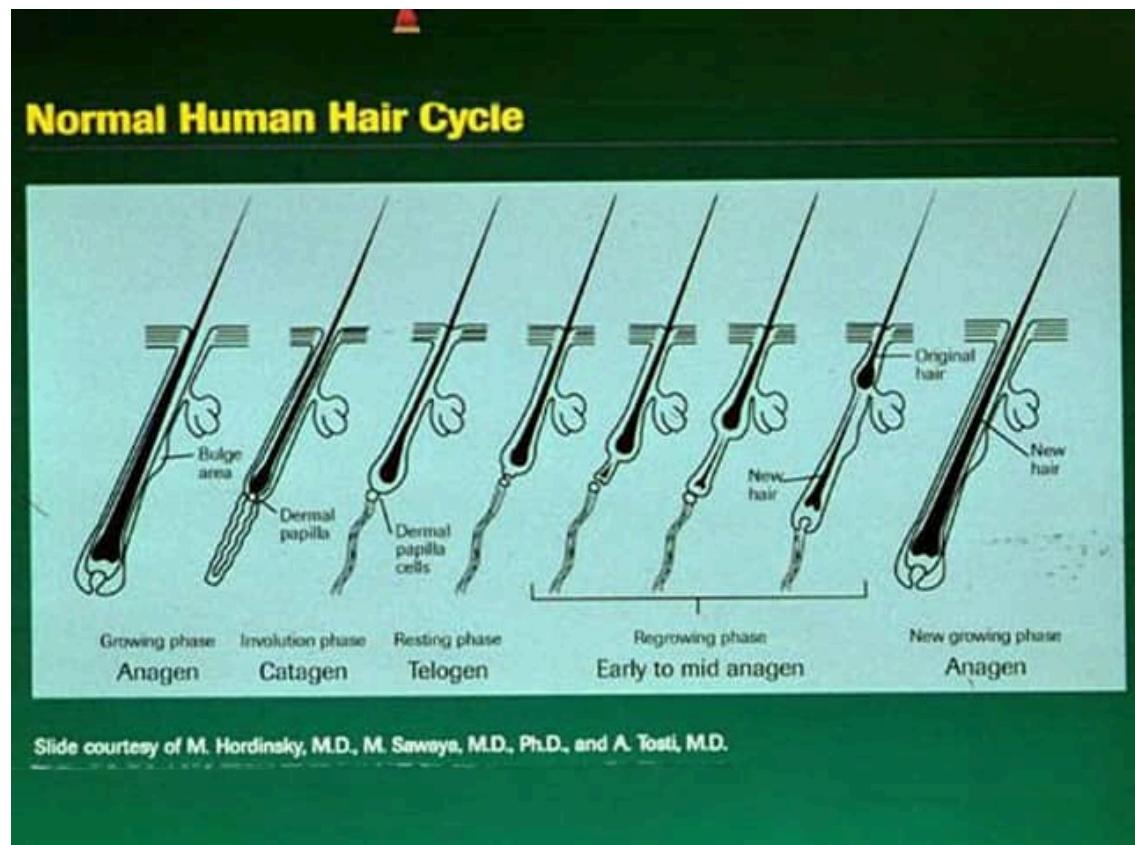


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What causes hair loss?

Hair loss can be due to:

- The decreased growth of the hair: [anagen hair loss](#)
- Increased shedding of the hair: [telogen hair loss](#)
- Conversion of thick terminal hairs to thin vellus hairs; [male](#) and [female pattern hair loss](#)
- Congenital or acquired [hair shaft abnormalities](#)
- An inflammatory skin disease that damages or destroys the hair bulb.

What are the clinical features of hair loss?

The features of hair loss depend on the cause. Actual symptoms such as itch and soreness are generally absent unless caused by accompanying inflammatory skin disease. However, a burning, prickly discomfort known as trichodynia may accompany hair shedding.

Anagen hair loss

Anagen hair is tapered or broken-off. Anagen is variable in duration. Children with otherwise normal hair but that cannot grow their hair long may have [short anagen syndrome](#).

Anagen shedding is known as [anagen effluvium](#) and has a sudden onset.

Anagen effluvium is caused by:

- [Autoimmune disease](#), including severe diffuse [alopecia areata](#)
- [Medications](#), especially [cytotoxic/chemotherapy drugs](#)
- An inherited or congenital condition, such as [loose anagen syndrome](#)

Short broken hairs and empty follicles may be observed. If caused by a drug or toxin, hair growth can return to normal within 3–6 months of its withdrawal.

Anagen hair loss



Hair shedding during chemotherapy



Hair lost through chemotherapy



Alopecia areata

Telogen hair loss

Telogen hair has a bulb at the end (club hair). Excessive shedding is known as [telogen effluvium](#). It occurs 2–6 months after an event that stops active hair growth.

Telogen effluvium is caused by:

- Child-bearing
- Fever
- Weight loss
- Haemorrhage
- A surgical operation, illness or psychological stress
- [Medications](#), including [contraceptives](#), anticoagulants, [anticonvulsants](#).

Sometimes there appears to be no recognisable cause for telogen effluvium, and shedding can continue for years (chronic telogen effluvium). Scalp hair continues to grow but has a shorter natural length than normal.



Telogen effluvium



Telogen effluvium

Pattern hair loss (androgenetic alopecia)

Pattern hair loss is due to genetic programming or hormonal influences. It is also called androgenetic alopecia because it is influenced by androgens.

Pattern alopecia is apparent in about 50% of individuals by the age of 50 years.

[Male pattern alopecia](#) affects vertex and temporal scalp.

[Female pattern alopecia](#) is less pronounced and affects the anterior scalp.



Male pattern balding



Female pattern balding



Severe female pattern balding

Hair shaft abnormalities

[Hair shaft defects](#) can be inherited and congenital, or acquired due to disease or injury (eg, excessive brushing, hair pulling [trichotillomania], hairdryer heat, relaxing chemicals, bleach). See [African hair practices](#).

Hair shaft abnormalities are diagnosed by [dermatoscopy](#) or microscopic examination of the hair, and sometimes by scanning electron microscopy. They include:

Fractures: trichorrhexis nodosa, trichoschisis, trichoclasis ([trichothiodystrophy](#))

Irregularities: trichorrhexis invaginata (seen with ichthyosis in [Netherton syndrome](#)), Marie-Unna hypotrichosis (uncombable hair), pili bifurcati, pili annulati, pseudopili annulati, monilethrix (beaded hair), pseudomonilethrix

Coiling and twisting: pili torti (twisted hair), woolly hair, trichonodosis (knotted hair).

Dermatological disease

Conditions resulting in reversible patchy hair thinning, poor hair quality and bald patches include:

Localised [alopecia areata](#)

A localised infection, such as [tinea capitis](#)

Severe local skin disease, such as [psoriasis](#), [seborrhoeic dermatitis](#), [atopic dermatitis](#), pityriasis rubra pilaris, [cutaneous lupus erythematosus](#), [cutaneous T-cell lymphoma](#)

Generalised skin disease ([erythroderma](#)).

Hair loss due to scalp conditions



Tinea capitis



Psoriasis © R Suhonen



Seborrhoeic dermatitis

Systemic disease

Systemic diseases resulting in reversible patchy hair thinning, poor hair quality and bald patches include:

Iron deficiency
Thyroid hormone deficiency
Systemic lupus erythematosus
Syphilis
Severe acute or chronic illness.

Destructive inflammatory skin diseases

Inflammation in the dermis or subcutaneous tissue may injure the hair follicle resulting in localised bald patches in which there are no visible follicles; this is called scarring alopecia or cicatricial alopecia.

Traumatic causes of scarring alopecia may be due to:

Injury
Surgery
Radiation
Traction (tight curls)
Central centrifugal cicatricial alopecia.

Traumatic forms of alopecia



Trichotillomania



Trichotillomania



Traction alopecia

Infections causing scarring alopecia include:

Bacterial infection: boils and abscesses (*Staphylococcus aureus*)
Fungal infection: kerion (inflammatory *tinea capitis*)
Viral infection: shingles (*herpes zoster*).

Inflammatory skin diseases causing scarring alopecia include:

- Folliculitis decalvans
- Dissecting cellulitis
- Lichen planopilaris
- Frontal fibrosing alopecia
- Alopecia mucinosa
- Discoid lupus erythematosus
- Localised scleroderma.

Pseudopelade of Brocq is a condition in which there are localised areas of the scalp in which hair follicles have disappeared without visible inflammation.

Scarring alopecia



Discoid lupus erythematosus



Folliculitis decalvans



Lichen planopilaris

Complications of hair loss

Whatever the type of hair loss, it may be extremely distressing and embarrassing, reducing the quality of life and causing **psychosocial problems**. Loss of normal scalp hair increases the risk of:

- Sunburn
- Injury.

How is hair loss diagnosed?

A careful history and full skin examination can generally result in the correct diagnosis. Additional tests may include:

Hair pull test to determine the relative proportion of anagen and telogen hairs

[Wood lamp examination](#)

Swabs of pustules for [bacterial](#) and [viral culture](#)

Skin scrapings and hair clippings for [mycology](#)

Blood tests for haematology, thyroid function, serology.

What is the treatment for hair loss?

Treatment depends on the diagnosis.

Infections should be treated.

Deficiencies should be remedied.

Causative drugs may be discontinued.

Inflammation can be suppressed.

Treatment may be available for specific conditions.

A systematic review of 30 studies suggests a discussion on nutritional supplementation should be considered between patient and physician.

How can hair loss be prevented?

Most types of hair loss cannot be actively prevented. However, it is prudent to avoid injury to the hair shaft.

Dry hair naturally or with a hairdryer on a cool setting

Minimise chemical treatments or use them infrequently

Use loose hairstyles to avoid traction injury

What is the outlook for hair loss?

The outlook for hair loss depends on the diagnosis. Scarring alopecia is permanent.

[Anagen](#) and [telogen](#) shedding generally stops in time

Early treatment of [pattern](#) alopecia may reduce the speed of thinning

Treatment of any inflammatory disease may be successful

Hair loss can be disguised or covered

Hair replacement techniques include [wigs](#), [hairpieces](#) and [surgery](#).

Hand, foot, and mouth disease

August 2022

Author: Dr Stanley Leong, Paediatric Registrar; Dr Caroline Mahon, Dermatologist, Christchurch Hospital, New Zealand

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What is hand, foot, and mouth disease?

Hand, foot, and mouth disease (HFMD or HFM) is a common, self-limiting, viral infection that causes blisters on the hands, feet, and inside or around the mouth. It mainly affects children under the age of 5 years.

HFMD, also called enteroviral vesicular stomatitis, occurs sporadically worldwide. Epidemics are most common during warm weather, usually in the late summer or early autumn.

It is important to note that HFMD is NOT related to foot and mouth disease of animals.

[Click here for images](#)

Who gets hand, foot, and mouth disease?

HFMD mostly occurs in children under 10 years of age with 95% of the cases occurring in toddlers aged under 5 years. However, it can also affect older children/adolescents. Adults, especially those who are immunocompromised, may also be affected. However, HFMD only rarely affects healthy adults.

Hand, foot, and mouth disease is very common. The average annual incidence of HFMD has been reported as 90–2400 cases per 100,000 people in some countries.

What causes hand, foot, and mouth disease?

Enteroviral vesicular stomatitis (HFMD) is usually caused by the Coxsackie virus, most commonly the A16 subtype. It may also be caused by other viruses such as:

Coxsackie A virus (5, 6, 7, 9, 10)

Coxsackie B virus (2, 5)

Enterovirus 71

Echoviruses.

Enterovirus 71 infection is associated with more severe infections that may involve the heart, lungs, and can also cause inflammation of the lining of the brain (meningitis).

Transmission occurs via direct contact with blister fluid or droplets spread from the mouth. It can spread very rapidly among family members or within a school. The virus can be shed in faeces and saliva for several weeks.

What are the clinical features of hand, foot, and mouth disease?

The illness usually begins with one or all of the following: fever, sore throat, loss of appetite, and lethargy. However, many children remain well in themselves despite the rash. The blisters usually appear 1–2 days following the fever.

The incubation period is typically 3–6 days and children remain infectious until the blisters have ruptured and healed (usually 7–10 days).

Skin findings typically include:

Blisters on the tops of the hands, feet and/or palms, and soles. Lesions usually:

Feel tender

Evolve over time from flat pink macules to small, elongated, red-greyish blisters

Are often oval rather than round

Peel off within a week, without leaving a scar.

Small blisters (vesicles) and [ulcers](#) may develop in and/or around the lips and mouth and the back of the throat. These can sometimes be very painful. Oral intake may be significantly impacted, especially in infants and younger children.

In children with [eczema](#), or past eczema, blisters, flat red macules and papules may develop over other areas of the skin, especially the buttocks and sometimes on the arms, legs, and genital skin.

Atypical HFMD can result in a more widespread rash and blistering. Features may include:

Red, crusted macules and papules without blistering

Large blisters (bulla)

Targetoid (bulls-eye, or target-shaped) lesions

Nail shedding

Involvement of atypical or unusual sites such as the ears.



Oval vesicles on the sole in hand, foot and mouth disease

Nail changes noted 6 weeks after hand and mouth blister resolution



Oral hand, foot, and mouth disease

[Click here for more images](#)

How do clinical features vary in differing types of skin?

In children with pre-existing eczema ([atopic dermatitis](#)), HFMD lesions may be localised in eczematous areas ([eczema coxsackium](#)).

What are the complications of hand, foot, and mouth disease?

Severe complications are very uncommon in people that are otherwise healthy. They include:

Dehydration due to inadequate fluid intake. This can cause significant problems in younger children.

Fingernail and toenail changes are often noted about two months after HFMD infections due to coxsackie A6 infection.

Transverse lines in the nail plate that slowly move outwards

Onychomadesis (nail shedding) may occur about 2 months after the illness, however, eventually the nails return to normal.

Serious enteroviral infection can lead to:

Widespread blistering

Enteritis

Myocarditis

Inflammation of the brain and or the lining of the brain (meningoencephalitis)

Loss of nerve function in a limb (acute flaccid paralysis)

Pulmonary oedema and pneumonia

Haemorrhagic conjunctivitis

In pregnancy, viruses that cause HFMD can cause first trimester spontaneous miscarriage or intrauterine growth restriction

Meningoencephalitis, thrombocytopenia, disseminated intravascular coagulopathy, cardiomyopathy and hepatitis in the newborn have rarely been described.

How is hand, foot, and mouth disease diagnosed?

HFMD is usually diagnosed clinically. Cutaneous lesions are typically distributed symmetrically over common sites of the skin such as the hands, feet, and in and around the mouth in a child.

Other diagnostic tools include:

Polymerase chain reaction (PCR) testing

Viral DNA may be detected from nasopharyngeal, (throat or nose), swabs and stool specimens.

Analysis of blood, cerebrospinal fluid (CSF), and faeces samples can confirm the diagnosis, but are rarely needed except in atypical or severe cases.

[Skin biopsy](#) of a blister

Very rarely indicated.

Shows acral skin with lymphocytic infiltrates at the epidermis.

The infiltrate is associated with keratinocyte apoptosis in early lesions.

See [hand, foot, and mouth disease pathology](#) for more information.

What is the differential diagnosis for hand, foot, and mouth disease?

Bacterial infections: such as Group A *Streptococcus* and *Staphylococcus aureus*, may cause similar blistering skin lesions, eg, [bullous impetigo](#).

Other [viral infections](#) such as human parechoviruses, [herpes simplex virus](#), adenoviruses, varicella zoster virus, Epstein–Barr virus, and [human herpesvirus](#) 6 and 7.

Bullous [insect bite reactions](#) may also present on the hands and feet in children.

[Pompholyx](#) eczema.

What is the treatment for hand, foot, and mouth disease?

Specific treatment is not usually required for HFMD, and the focus is symptomatic care. HFMD rarely causes serious complications. Antibiotics do not work and should not be given to children with HFMD.

No vaccines or specific antiviral medications are available.

General measures

Pain relief

Simple analgesia such as paracetamol or ibuprofen as needed.

Antiseptic [mouthwashes](#) or topical soothing agents (eg, lignocaine) can be used in children with painful oral/palatal ulcers.

Aspirin should not be used routinely due to the risk of Reye syndrome.

Hydration

Constantly offer the child sips of water/juice to prevent dehydration.

If oral intake is poor, nasogastric or intravenous fluids may be indicated.

Blister care

Leave blisters to dry naturally.

Do not pierce/rupture the blisters to reduce contagion.

Keep the blisters clean and apply non-adherent dressings to erosions.

Should children with hand, foot, and mouth disease stay home from school?

In the vast majority of cases, HFMD is a mild illness and there is no need to keep children from school once they are well enough to attend.

The blisters remain infective until they have dried, which is usually within a few days. However, the virus sheds through faecal stools and these remain infective for up to a month after the illness. Therefore, it is impractical to keep children who are well away from school.

General preventative measures include:

Thorough hand hygiene especially after coming into contact with one's bodily fluid.

This includes touching their blisters, helping them to blow their nose and changing nappies or helping with toileting.

Minimise sharing personal items such as cutlery, drinking cups, towels, toothbrushes, and clothing.

What is the outcome for hand, foot, and mouth disease?

HFMD infection is usually mild and complete resolution is seen within 7–10 days. Infection often results in long-term immunity to the specific virus, however a second episode can occur following infection with a different member of the enterovirus group.

[Click here for images](#)

Miliaria

Author: Dr Amanda Oakley, Dermatologist, Hamilton, New Zealand, 1997. Updated by Dr Anita Eshraghi, Dermatologist, Sweden. February 2018. Revised September 2020

What is miliaria?

Miliaria is a common skin disease caused by blockage and/or inflammation of eccrine sweat ducts.

Miliaria is frequently seen in hot humid environmental climates in patients in hospital and in the neonatal period.

Based on the level of the sweat duct obstruction, miliaria is divided into three types:

Miliaria crystallina (sudamina) is due to obstruction of the sweat ducts close to the surface of the skin in the stratum corneum of the epidermis

Miliaria rubra follows obstruction of the sweat ducts in the deeper layers of the skin (mid-epidermis)

Miliaria profunda (tropical anhidrosis) is the result of sweat leaking from the sweat glands into the middle layer of skin (blockage at or below the dermoepidermal junction) following repeated episodes of miliaria rubra.



Miliaria



Miliaria



Who gets miliaria?

Miliaria crystallina affects up to 9% of neonates, with the mean age of 1 week. It can also occur in adults with fever.

Miliaria rubra is the most common type of heat rash. It is seen in children and in up to 30% of adults who move to a tropical environment or are unexpectedly exposed to heat and humidity. Although miliaria may develop within days of arriving in the tropics, it often takes several months to peak.

Miliaria profunda is rare and presents in adult males.

Heat rash can affect all age groups and racial groups.

What causes miliaria?

Prickly heat results from sweating. The main contributing causes and risk factors are:

- Immature sweat ducts in a newborn child
- A hot and humid environment
- Intense exercise or physical activity
- Fever
- Occlusion of the skin with non-porous dressings or synthetic clothing against the skin
- Hospitalised or bedridden patients lying on waterproofed mattresses or mattress-protectors.

Other diseases and treatments that have been associated with miliaria are:

- [Drug-induced hyperhidrosis](#)
- [Adverse reaction to medication](#) such as induction chemotherapy
- [Stevens-Johnson syndrome / toxic epidermal necrolysis](#)
- Genetic disease (Morvan syndrome and pseudohypoaldosteronism type I)
- Radiotherapy.

What are the clinical features of miliaria?

Symptoms of heat rash vary depending on type:

Miliaria crystallina presents as 1–2 mm superficial clear blisters that easily break. The blisters can look like beads of sweat. There is no inflammation. The blisters are usually seen widely spread on the head, neck, and upper trunk. The vesicles break easily to leave a bran-like scale.

Miliaria rubra, the most common form of heat rash, results in red, 2–4 mm, non-follicular papules and papulovesicles. They are very itchy. Background erythema is often present. In children, miliaria involves the trunk and the skin folds of the neck, axilla or groin. In adults, miliaria often affects the upper trunk, scalp, neck and flexures, particularly in areas of friction with clothing.

Miliaria pustulosa is a variant of miliaria rubra with pustules.

Miliaria profunda presents as asymptomatic deep papules. The flesh-coloured, 1–3 mm diameter papules develop on the trunk and extremities.

What are the complications of miliaria?

Complications of miliaria include:

- [Secondary bacterial infection](#), commonly caused by [staphylococci](#)
- Impaired thermoregulation
- [Hyperhidrosis](#) in non-affected areas.

How is miliaria diagnosed?

Heat rash is diagnosed on its typical clinical presentation.

In severe cases or repeated episodes, [punch biopsy](#) can be useful. Miliaria crystallina shows vesicles associated with the sweat ducts within or just under the stratum corneum of the epidermis. Histology of miliaria rubra shows spongiosis and spongiotic vesicles.

Tzanck smear taken from vesicles will distinguish miliaria from herpes simplex or toxic erythema of the newborn.

What is the differential diagnosis for miliaria?

Skin disorders presenting with papules, vesicles, or pustules that look similar to miliaria include:

- [Herpes simplex](#)
- [Fungal infections](#)
- [Bacterial folliculitis](#)
- [Acne](#)
- [Acute generalised exanthematous pustulosis \(AGEP\)](#)
- [Toxic erythema of newborn](#)
- [Grover disease.](#)

What is the treatment for miliaria?

Resolution of miliaria requires minimising heat and humidity to reduce sweating and the avoidance of irritation to the skin. Strategies to avoid sweating, keeping the skin cool, and reducing irritation can help treat and prevent heat rash:

- Work in an air-conditioned office for at least a few hours a day
- Sleep in a ventilated, cool bedroom
- Move away from a tropical climate, avoiding humid weather
- Avoid excessive clothing and tight clothing
- Avoid excessive soap and irritants
- Wear shirts and blouses made of breathable synthetic fabrics or cotton
- Remove wet clothing
- Cool water compresses and taking a cool bath
- Patients should be educated on symptoms of heat exhaustion.

Heat rash may require medical care which can involve:

- Calamine lotion to relieve discomfort; because calamine lotion is drying, an [emollient](#) may be required.
- Treatment of fever with antipyretic such as paracetamol (American terminology acetoaminophen)
- Mild [topical steroids](#)
- [Antiseptics](#) and anti-staphylococcal [antibiotics](#) for secondary infection.

What is the outcome for miliaria?

Most cases of heat rash resolve within a day or two after changing to a cooler environment without any treatment or complications.

Herpes simplex

Author: Hon A/Prof Amanda Oakley, Dermatologist, Hamilton, New Zealand, 1997. Updated October 2015.

What is herpes simplex?

Herpes simplex is a common viral infection that presents with localised blistering. It affects most people on one or more occasions during their lives.

Herpes simplex is commonly referred to as cold sores or fever blisters, as recurrences are often triggered by a febrile illness, such as a cold.



Herpes labialis



Herpes simplex on the cheek



Herpetic whitlow

What causes herpes simplex?

Herpes simplex is caused by one of two types of herpes simplex virus (HSV), members of the [Herpesvirales](#) family of double-stranded DNA viruses.

Type 1 HSV is mainly associated with oral and facial infections

Type 2 HSV is mainly associated with genital and rectal infections ([anogenital herpes](#))

However, either virus can affect almost any area of skin or mucous membrane.

After the primary episode of infection, HSV resides in a latent state in spinal dorsal root nerves that supply sensation to the skin. During a recurrence, the virus follows the nerves onto the skin or mucous membranes, where it multiplies, causing the clinical lesion. After each attack and lifelong, it enters the resting state.

During an attack, the virus can be inoculated into new sites of skin, which can then develop blisters as well as the original site of infection.

Who gets herpes simplex?

Primary attacks of Type 1 HSV infections occur mainly in infants and young children. In crowded, underdeveloped areas of the world, nearly all children have been infected by the age of 5. In less crowded places, the incidence is lower; for example, less than half of university entrants in Britain have been infected. Type 2 HSV infections occur mainly after puberty and are often transmitted sexually.

HSV is transmitted by direct or indirect contact with someone with active herpes simplex, which is infectious for 7–12 days. Asymptomatic shedding of the virus in saliva or genital secretions can also lead to transmission of HSV, but this is infrequent, as the amount shed from inactive lesions is 100 to 1000 times less than when it is active. The incubation period is 2–12 days.

Minor injury helps inoculate HSV into the skin. For example:

A thumb sucker may transmit the virus from their mouth to their thumb.

A health-care worker may develop [herpetic whitlow](#)

A rugby player may get a cluster of blisters on one cheek ('scrumpox').

What are the clinical features of herpes simplex?

Primary herpes simplex

Primary infection with HSV can be mild or subclinical, but symptomatic infection tends to be more severe than recurrences. Type 2 HSV is more often symptomatic than Type 1 HSV.

Primary Type 1 HSV most often presents as gingivostomatitis, in children between 1 and 5 years of age. Symptoms include fever, which may be high, restlessness and excessive dribbling. Drinking and eating are painful, and the breath is foul. The gums are swollen and red and bleed easily. Whitish vesicles evolve to yellowish ulcers on the tongue, throat, palate and inside the cheeks. Local lymph glands are enlarged and tender.

The fever subsides after 3–5 days and recovery is usually complete within 2 weeks.

Primary Type 2 HSV usually presents as genital herpes after the onset of sexual activity. Painful vesicles, ulcers, redness and swelling last for 2 to 3 weeks, if untreated, and are often accompanied by fever and tender inguinal lymphadenopathy.

In males, herpes most often affects the glans, foreskin and shaft of the penis. Anal herpes is more common in males who have sex with men than with heterosexual partners.

In females, herpes most often arises on the vulva and in the vagina. It is often painful or difficult to pass urine. Infection of the cervix may progress to severe ulceration.

Recurrent herpes simplex

After the initial infection, whether symptomatic or not, there may be no further clinical manifestations throughout life. Where viral immunity is insufficient, recurrent infections are common, particularly with Type 2 genital herpes.

Recurrences can be triggered by:

- Minor trauma, surgery or procedures to the affected area
- Upper respiratory tract infections
- Sun exposure
- Hormonal factors (in women, flares are not uncommon prior to menstruation)
- Emotional stress

In many cases, no reason for the eruption is evident.

The vesicles tend to be smaller and more closely grouped in recurrent herpes, compared to primary herpes. They usually return to roughly the same site as the primary infection.

Recurrent Type 1 HSV can occur on any site, most frequently the face, particularly the lips (herpes simplex labialis).

Recurrent Type 2 HSV may also occur on any site, but most often affects the genitals or buttocks.

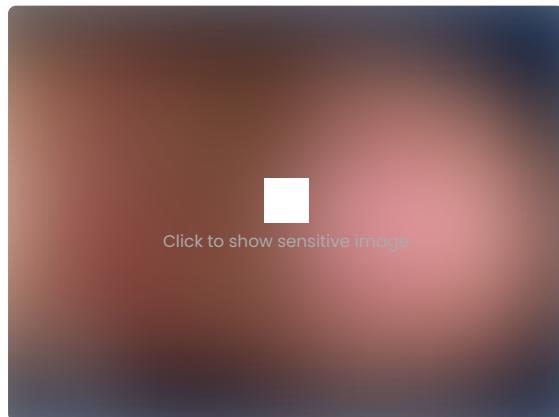
Itching or burning is followed an hour or two later by an irregular cluster of small, closely grouped, often umbilicated vesicles on a red base. They normally heal in 7–10 days without scarring. The affected person may feel well or suffer from fever, pain and have enlarged local lymph nodes.

Herpetic vesicles are sometimes arranged in a line rather like [shingles](#) and are said to have a **zosteriform distribution**, particularly when affecting the lower chest or lumbar region.

White patches or scars may occur at the site of recurrent HSV attacks and are more evident in those with the skin of colour.



Pustule due to herpes simplex



Click to show sensitive image

Clustered vesicles, due to HSV, have broken down into erosions on the proximal nail fold

[See more images of herpes simplex.](#)

How is herpes simplex diagnosed?

If there is clinical doubt, HSV can be confirmed by culture or PCR of a viral swab taken from fresh vesicles. HSV serology is not very informative, as it's positive in most individuals and thus not specific for the lesion with which they present.

What are the complications of herpes simplex?

Eye infection

Herpes simplex may cause swollen [eyelids](#) and conjunctivitis with opacity and superficial ulceration of the cornea (dendritic ulcer, best seen after fluorescein staining of the cornea).

Throat infection

Throat infections may be very painful and interfere with swallowing.

Eczema herpeticum

In patients with a history of [atopic dermatitis](#) or [Darier disease](#), HSV may result in severe and widespread infection, known as eczema herpeticum. The skin disease can be active or historical. Numerous blisters erupt on the face or elsewhere, associated with swollen lymph glands and fever.

Erythema multiforme

A single episode or recurrent [erythema multiforme](#) is an uncommon reaction to herpes simplex. The rash of erythema multiforme appears as symmetrical plaques on hands, forearms, feet and lower legs. It is characterised by [target lesions](#), which sometimes have central blisters. Mucosal lesions may be observed.

Nervous system

Cranial/facial nerves may be infected by HSV, producing temporary paralysis of the affected muscles. Rarely, neuralgic pain may precede each recurrence of herpes by 1 or 2 days (Maurice syndrome). Meningitis is rare.

Widespread infection

Disseminated infection and/or persistent ulceration due to HSV can be serious in debilitated or immune deficient patients, for example in people with human immunodeficiency virus (HIV) infection.



Dendritic ulcer



Eczema herpeticum



Erythema multiforme

What is the treatment for herpes simplex?

Mild, uncomplicated eruptions of herpes simplex require no treatment. Blisters may be covered if desired, for example with a hydrocolloid patch. Severe infection may require treatment with an antiviral agent.

Antiviral drugs used for herpes simplex and their usual doses are:

Aciclovir – 200 mg 5 times daily for five days

Valaciclovir – 500 mg twice daily for five days

Famciclovir – as a single dose of 3 x 500 mg

In New Zealand, famciclovir is not currently funded by PHARMAC (April 2019).

Higher doses and/or longer courses of antiviral drugs may be used for immunocompromised patients, eczema herpeticum, or for disseminated herpes simplex.

Topical aciclovir or penciclovir may shorten attacks of recurrent herpes simplex, provided the cream is started early enough.

Can herpes simplex be prevented?

As sun exposure often triggers facial herpes simplex, [sun protection](#) using high protection factor [sunscreens](#) and other measures are important.

Antiviral drugs will stop HSV multiplying once it reaches the skin or mucous membranes but cannot eradicate the virus from its resting stage within the nerve cells. They can, therefore, shorten and prevent attacks but a single course cannot prevent future attacks. Repeated courses may be prescribed, or the medication may be taken continuously to prevent frequent attacks.

Herpes zoster

Author: Hon A/Prof Amanda Oakley, Dermatologist, Hamilton, New Zealand, 1997. Updated October 2015.

What is herpes zoster?

Herpes zoster is a localised, blistering and painful rash caused by reactivation of varicella-zoster virus (VZV). Herpes zoster is also called shingles.

VZV is also called herpesvirus 3 and is a member of the Herpesvirales order of double-stranded DNA viruses.



Herpes zoster



Herpes zoster



Herpes zoster

Who gets herpes zoster?

Anyone who has had varicella ([chickenpox](#)) may subsequently develop herpes zoster. Zoster can occur in childhood but is much more common in adults, especially older people. People with various kinds of cancer have a 40% increased risk of developing zoster. People who have had zoster rarely get it again; the chance of getting a second episode is about 1%.

Herpes zoster often affects people with weak immunity.

What causes herpes zoster?

After primary infection—varicella—VZV remains dormant in dorsal root ganglia nerve cells in the spine for years before it is reactivated and migrates down sensory nerves to the skin to cause herpes zoster.

It is not clear why herpes zoster affects a particular nerve fibre. Triggering factors are sometimes recognised, such as:

- Pressure on the nerve roots
- Radiotherapy at the level of the affected nerve root
- Spinal surgery
- An infection
- An injury (not necessarily to the spine)
- Contact with someone with varicella or herpes zoster

What are the clinical features of herpes zoster?

Herpes zoster is characterised by [dermatomal distribution](#), that is the blisters are confined to the cutaneous distribution of one or two adjacent sensory nerves. This is usually unilateral, with a sharp cut-off at the anterior and posterior midlines.

The clinical presentation of herpes zoster depends on the age and health of the patient and which [dermatome](#) is affected.

The first sign of herpes zoster is usually localised pain without tenderness or any visible skin change. It may be severe, relating to one or more sensory nerves. The pain may be just in one spot, or it may spread out. The patient may feel quite unwell with fever and headache. The lymph nodes draining the affected area are often enlarged and tender.

Within one to three days of the onset of pain, a blistering rash appears in the painful area of skin. It starts as a crop of red papules. New lesions continue to erupt for several days within the distribution of the affected nerve, each blistering or becoming pustular then crusting over.

The chest (thoracic), neck (cervical), forehead (ophthalmic) and lumbar/sacral sensory nerve supply regions are most commonly affected at all ages. The frequency of ophthalmic herpes zoster increases with age. Herpes zoster occasionally causes blisters inside the mouth or ears, and can also affect the genital area. Sometimes there is pain without rash—herpes zoster "sine eruptione"—or rash without pain, most often in children.

Pain and general symptoms subside gradually as the eruption disappears. In uncomplicated cases, recovery is complete within 2–3 weeks in children and young adults, and within 3–4 weeks in older patients.



Herpes zoster



Herpes zoster



[See more images of herpes zoster.](#)

What are the complications of herpes zoster?

- Involvement of several [dermatomes](#), or sometimes, bilateral eruptions in unique dermatomes
- Eye complications when the ophthalmic division of the fifth cranial nerve is involved
- Deep blisters that take weeks to heal followed by scarring
- Muscle weakness in about one in 20 patients. Facial nerve palsy is the most common result (see [Ramsay Hunt syndrome](#)). There is a 50% chance of complete recovery, but some improvement can be expected in nearly all cases
- Infection of internal organs, including the gastrointestinal tract, lungs, and brain (encephalitis)

Herpes zoster is infectious to people who have not previously had chickenpox.

Herpes zoster in the early months of pregnancy can harm the fetus, but luckily this is rare. Shingles in late pregnancy can cause chickenpox in the fetus or newborn. Herpes zoster may then develop as an infant.

Post-herpetic neuralgia

[Post-herpetic neuralgia](#) is defined as persistence or recurrence of pain in the same area, more than a month after the onset of herpes zoster. It becomes increasingly common with age, affecting about a third of patients over 40. It is particularly likely if there is facial infection. Post-herpetic neuralgia may be a continuous burning sensation with increased sensitivity in the affected areas or spasmodic shooting pain. The overlying skin is often numb or exquisitely sensitive to touch. Sometimes, instead of pain, the neuralgia results in a persistent itch (neuropathic [pruritus](#)).

What is the treatment of herpes zoster?

Prevention of herpes zoster

Because the risk of serious complications from herpes zoster is more likely in older people, those aged over 60 years might consider the zoster vaccine, which can reduce the incidence of herpes zoster by half. In people who do get herpes zoster despite being vaccinated, the symptoms are usually less severe, and post-herpetic neuralgia is less likely to develop. In New Zealand, the zoster vaccine will be funded from 1 April 2018 for people aged between 66 and 80 years old.

Herpes zoster vaccination is contraindicated in immunosuppressed patients due to the risk of it causing disseminated herpes zoster infection.

General measures

Rest and pain relief

Protective ointment applied to the rash, such as petroleum jelly.

Oral antibiotics for secondary infection

Specific measures

Antiviral treatment can reduce pain and the duration of symptoms if started within one to three days after the onset of herpes zoster. Aciclovir 800 mg 5 times daily for seven days is most often prescribed. Valaciclovir and famciclovir are also useful. The efficacy of prescribing systemic steroids is unproven.

Post-herpetic neuralgia

Early use of antiviral medication

[Local anaesthetic](#) applications

[Topical capsaicin](#)

Tricyclic antidepressant medications such as [amitriptyline](#)

Anti-epileptic medications [gabapentin](#) and pregabalin

Transcutaneous electrical nerve stimulation or acupuncture

[Botulinum toxin](#) into the affected area

Nonsteroidal anti-inflammatories and opioids are generally unhelpful.



HIVES: FAQS

[FAQs](#) [Symptoms](#) [Causes](#) [Treatment](#) [At-home relief](#) [Chronic spontaneous urticaria](#) [Relief from chronic hives](#)
[Cold temperature hives](#)



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The American Academy of Dermatology gratefully acknowledges the support from Sanofi and Regeneron.

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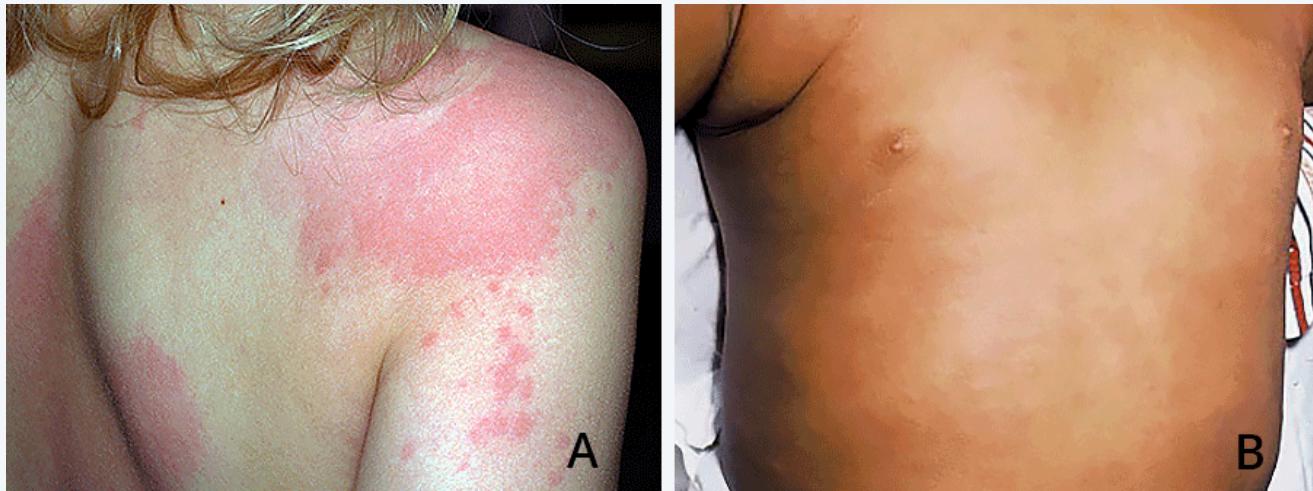
What are hives?

Hives

Hives are a skin reaction that causes bumps, raised patches, or both to suddenly appear. These bumps and patches often itch intensely. Most cases of hives are temporary and go away without treatment. When hives linger, a board-certified dermatologist can help prevent flare-ups.

Are hives contagious? No, you cannot catch hives.

Sometimes, it may seem like you can catch hives because some people develop hives when they get a contagious disease like strep throat, COVID-19, or a common cold. What's really happening is this. First, the person catches a contagious disease. Next, their immune system reacts to the infection by making histamine. It's histamine that causes hives.



Hives on children's skin

Hives usually cause a rash of intensely itchy bumps and patches on the skin. On lighter skin tones (A), you may see a pink or red rash. People with darker skin tones (B) often have hives that closely match their skin tones, so hives can be more difficult to spot.

What does urticaria mean?

Urticaria (ur-tih-KAR-e-uh) is the medical word for hives. It refers to the often-itchy bumps and patches that suddenly appear on the skin.

Some people who have hives also develop swelling deep in their skin. The medical word for this swelling is **angioedema** (an-jee-oh-uh-DEE-mah).

How long do hives usually last?

An individual hive tends to go away within 24 hours, but new hives can appear. For most people, a case of hives usually lasts a few days to a few weeks.

A case of hives can also last longer than a few weeks. If you continue to get hives for 6 weeks or longer, you have **chronic hives**. A board-certified dermatologist can successfully treat chronic hives.

Are hives dangerous?

Most cases of hives are harmless and go away on their own.

Sometimes, hives require immediate medical care. Go to urgent care or the nearest emergency room if you have any of the following:

- Swelling on your face, inside your mouth, or in your throat
- Problems swallowing or breathing
- Light-headed or faint feeling
- Racing heart

If hives cause swelling elsewhere on your body (aside from your face, mouth, or throat), it's usually harmless. Keep in mind that swelling can come back in a different area. Any time swelling develops on your face or inside your mouth or throat, get immediate medical care.

If the swelling continues to come and go, getting [treatment for hives](#) can prevent swelling.

Are hives curable?

No, hives cannot be cured, but this condition is treatable.

If you have hives that last longer than 6 weeks, a dermatologist can develop a treatment plan that provides relief. To find out how dermatologists treat patients with long-lasting hives, see what two dermatologists say about treating long-lasting hives at [Chronic hives: How dermatologists help people get relief](#).

Do hives spread?

Hives cannot spread from one person to another.

Hives can spread on your body. If you have a rash, hives can get bigger or join together. More hives can appear. Some people develop widespread hives that cover much of their body.

Why do people break out in hives for no apparent reason?

Hives appear suddenly, so it can feel as if they develop for no reason. Keep in mind that hives develop when your skin is reacting to something. This reaction may be due to one of the following:

- Allergy (e.g., food, insect bite, medication)
- Infection (e.g., common cold, strep throat, or a urinary tract infection)
- Something physical touching your skin (e.g., sunlight, pressure, or cold)

The medical term for hives caused by something physical touching your skin is called **inducible hives**. It means you have a flare-up whenever something specific touches your skin, such as pressure or sunlight.

The most common type of inducible hives occurs when skin is scratched, rubbed, or stroked. The medical name for this is **dermatographism** (der-maa-toe-GRA-fi-ism), which means “writing on the skin.” It causes

swelling and itchy skin on the area rubbed, stroked, or scratched. The hives usually disappear within minutes after the rubbing, scratching, or stroking is stopped.

For more information about inducible hives, go to [10 ways to get relief from chronic hives](#).

Although hives are due to a skin reaction, it's important to know that many people never find out what's causing their hives. When you don't know the cause, you have **spontaneous hives**. Even when you don't know the cause, hives can be effectively treated.

Regardless of what triggers hives, bumps and patches can appear on the skin in many ways. To see different ways that hives can appear and find out about symptoms, go to [Hives: Signs and symptoms](#).

Images

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References

Antia C, Baquerizo K, et al. "Urticaria: A comprehensive review: Epidemiology, diagnosis, and work-up." *J Am Acad Dermatol*. 2018 Oct;79(4):599-614.

Grattan CEH, Saini S. "Urticaria and angioedema." In: Bolognia JL, et al. *Dermatology*. (fourth edition). Elsevier, China, 2018:308-9.

Hide M, Takahagi S, et al. "Urticaria and angioedema." In: Kang S, Amagai M, et al. *Fitzpatrick's Dermatology* (ninth edition). McGraw Hill Education, New York, 2019: 684-8.

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Last updated: 5/30/24

Hyperthyroidism

Author: Dr Shendy Engelina, Core Medical Trainee, Northampton General Hospital, UK. Chief Editor: Dr Amanda Oakley, Dermatologist, Hamilton, New Zealand, April 2016.

Introduction

Abnormal levels of circulating thyroid hormone (thyroxine) and underlying diseases may lead to alterations in the appearance of skin, hair and nails. The thyroid gland can be overactive, resulting in hyperthyroidism, discussed here, or underactive, resulting in [hypothyroidism](#).

What is hyperthyroidism?

Hyperthyroidism is due to excessive thyroxine. It is also known as thyrotoxicosis. It can lead to a variety of signs and symptoms.

Who gets hyperthyroidism?

Hyperthyroidism:

Can occur at any age, but usually affects older adults aged 40–60 years.

It is predominantly seen in females (5:1 female-to-male ratio).

Hyperthyroidism is more common among individuals with a personal or family history of autoimmune disorders, such as type 1 [diabetes](#), pernicious anaemia, [vitiligo](#), [lichen sclerosus](#) and [Addison disease](#).

Other risk factors include [smoking](#), high intake of dietary or supplemental [iodine](#), and trauma to the thyroid gland (eg, surgery).

What causes hyperthyroidism?

There are several causes for hyperthyroidism.

Graves disease

Graves disease is the most common cause of hyperthyroidism (80%).

It is an [autoimmune disorder](#) that involves antibodies activating the thyroid-stimulating hormone (TSH) receptor in thyroid cells.

The thyroid follicular cells proliferate abnormally, resulting in goitre (a swelling in the neck) and excess thyroxine.

There are a genetic predisposition and association with PTPN22, CTLA-4 and human leukocyte antigen (HLA) genotype.

Neonatal hyperthyroidism can rarely occur in babies born to mothers with Graves disease.



Goitre



Goitre

Thyroid nodule or nodules

A thyroid nodule is a benign growth within the thyroid gland.

A solitary nodule can manifest as toxic adenoma.

Multiple nodules can manifest as toxic multi-nodular goitre.

Active nodules interfere with the regulation of normal control of thyroxine production.

Medications

Hyperthyroidism arises from:

Excessive intake of levothyroxine used to treat hypothyroidism. This is called factitious or iatrogenic hyperthyroidism

Amiodarone, an iodinated drug used in heart disease, which can induce both hyperthyroidism and hypothyroidism

Lithium, often used for mental illness and eating disorders. It can also induce hyperthyroidism and hypothyroidism

Subacute thyroiditis

Subacute thyroiditis is also known as De Quervain thyroiditis.

Thyroiditis is inflammation of the thyroid gland.

Subacute thyroiditis follows a viral infection or pregnancy.

The gland is painful and tender for several months.

Thyroiditis is usually self-limiting and resolves spontaneously without treatment.

It initially causes temporary over-production of thyroid hormone (hyperthyroid phase), which is then followed by under-production ([hypothyroid phase](#)) before thyroid function returns to normal.

Rare causes of hyperthyroidism

Rare causes of hyperthyroidism include:

Thyroid cancer, a malignant proliferation of thyroid tissue.

Some forms of pituitary adenoma, a benign growth within the pituitary gland in the brain. Pituitary adenoma causes secondary hyperthyroidism.

What are the clinical features of hyperthyroidism?

Hyperthyroidism results in an increase in the body's metabolic rate, which characterised by:

Flushing of the face and hands

Smooth, moist and warm skin

Fine, soft and thinned scalp hair

Distorted and overgrown nails (thyroid acropachy) that may lift off the nail bed (**onycholysis**)

Generalised itching (**pruritus**)

Urticaria



Thyroid acropachy



Thyroid acropachy



Thyroid acropachy

Other common systemic features include palpitations, tremor, weight loss, heat intolerance, anxiety and menstrual disturbance (irregular or light period).

Graves disease may also cause thyroid dermopathy resulting in pretibial myxoedema and exophthalmos, which are associated with the presence of thyroid antibodies.



Thyroid ophthalmopathy



Thyroid ophthalmopathy



Thyroid ophthalmopathy

Pretibial myxoedema is due to **mucinosis**, a generalised excess of glycosaminoglycans in the dermis.

It affects a small number of patients with Graves disease (0.5–4.3%) and commonly occurs 1–2 years after the diagnosis.

Reddish, tender, swelling, nodules and plaques occur on the shins, calves and feet.

There may be violaceous or yellow-brown discolouration of the overlying skin with prominent hair follicles giving an orange-peel appearance. It may also look warty.



Pretibial myxoedema



Pretibial myxoedema



Pretibial myxoedema

What are the complications of hyperthyroidism?

Untreated hyperthyroidism can cause serious systemic complications including:

- Atrial fibrillation (irregular heart rhythm)
- Cardiomyopathy (weak heart)
- Heart failure
- Stroke

Osteoporosis and bone fracture

Medical emergencies associated with hyperthyroidism include thyroid storm and thyrotoxic periodic paralysis.

Thyroid storm

Thyroid storm is also called thyroid crisis.

It is characterised by features of a hypermetabolic state, including confusion, agitation, psychosis, hyperthermia and tachycardia.

It is rare (1%) but life-threatening and usually occurs in patients with undiagnosed or ineffectively treated hyperthyroidism.

Thyroid storm is commonly precipitated by infection, trauma, surgery, radioiodine therapy or withdrawal of an anti-thyroid drug.

Thyrotoxic periodic paralysis

Thyrotoxic periodic paralysis is characterised by acute onset of hypokalaemia.

Muscle paralysis is secondary to an intracellular shift of potassium, induced by thyroxine.

It is rare but potentially fatal.

Compared to other ethnicities, thyrotoxic periodic paralysis is more common in Asians, including Filipinos, Malaysians, Thais, Vietnamese and Koreans, in which it affects 2% of the hyperthyroid population.

How is hyperthyroidism diagnosed?

Hyperthyroidism is diagnosed with thyroid function tests (TFTs).

Serum TSH is usually low.

If there is a pituitary adenoma (rare), TSH can be high.

Serum T4 (thyroxine) and T3 (triiodothyronine) levels are usually high.

In subclinical hyperthyroidism, TSH is low in the presence of normal T4 and T3 level.

Interpretation of
thyroid function
tests

	TSH	Free T4 (thyroxine)	Free T3 (triiodothyronine)
(Primary) hyperthyroidism	Low	High	High
Secondary hyperthyroidism	High	High	High
Subclinical hyperthyroidism	Low	Normal	Normal

Interpretation of
thyroid function
tests

Primary hypothyroidism	High	Low	Low or normal
Secondary hypothyroidism	Low or normal	Low	Low or normal
Subclinical hypothyroidism	Borderline high	Normal	Normal
Sick euthyroid syndrome	Low	Low	Low

Serum autoantibodies are markers of autoimmune disease. They should include:

- Anti-thyroid peroxidase (TPO) antibodies. These are present in 75% of patients with Graves disease and are usually absent in multinodular goitre
- Anti-thyroglobulin antibodies
- TSH receptor antibodies

Full blood count and inflammatory markers, such as C-reactive protein, are routinely included to screen for anaemia (commonly associated with hyperthyroidism) and systemic infection causing thyroiditis.

Imaging should include:

- Ultrasound of the thyroid gland to detect a nodule.
- Thyroid uptake scan to detect cold areas (no activity), and overactive hot spots.

What is the treatment for hyperthyroidism?

Hyperthyroidism is either treated with medication, by radioactive iodine, surgery or a combination of these.

Anti-thyroid medication

Carbimazole is the treatment of choice to reduce the production of thyroid hormone. It is a pro-drug of methimazole, which is available in some countries as an alternative to carbimazole.

Dose varies from 10–40 mg daily, depending on the T4 level. It usually takes 4–8 weeks to be fully effective.

Common side effects include nausea, rash and pruritus. It can also rarely cause bone marrow suppression (< 0.5%). Patients must be warned to seek urgent medical review and have blood tests, should they develop fever, mouth ulcers and sore throat.

Carbimazole may be stopped when remission is achieved (usually after 18–24 months of starting treatment).

Propylthiouracil is recommended in pregnant women and for those intolerant of the side effects of carbimazole.

Beta blockers, such as propranolol, are also commonly used to control tremor and palpitations. Calcium channel blockers, such as verapamil, can be given as an alternative.

Radioactive iodine

Radioactive iodine is usually indicated for relapses of Graves disease and toxic nodular goitre. However, it has increasingly been used as first-line treatment for hyperthyroidism in teenagers.

The radioactive substance is ingested and destroys some of the thyroid cells leading to a reduction in thyroxine production.

It usually takes 3–4 months for treatment to show the full effect.

Radio-iodine is contraindicated in pregnancy and breastfeeding. Women are strongly advised to not become pregnant for at least six months following radio-iodine treatment. Patients should limit prolonged contact with others, especially children and pregnant women, for at least 2–4 weeks post-treatment.

Surgery

Partial or total thyroidectomy is surgical removal of part or the whole of the thyroid gland respectively.

It may be indicated for large goitre causing compressive symptoms such as shortness of breath and in those resistant to medical treatments.

What is the outcome for hyperthyroidism?

Treatment of hyperthyroidism is usually effective. Regular thyroid function tests are recommended following successful treatment, as some individuals develop further relapses, with annual blood tests long term.

Impetigo

Author: Dr Kate Quirke, Senior House Officer, Rotorua Hospital, New Zealand. Copy edited by Gus Mitchell. March 2022. Previous author: Hon A/Prof Amanda Oakley, Dermatologist, Waikato Hospital, New Zealand.

What is impetigo?

Impetigo is a common, superficial, highly contagious **bacterial skin infection** characterised by pustules and honey-coloured crusted erosions.

It affects the superficial layers of the epidermis and is typically caused by *Staphylococcus aureus* and *Streptococcus pyogenes* (Group A beta – haemolytic streptococci (GABHS)). It can be classified into non-bullous (also known as ‘school sores’) and bullous impetigo. **Ecthyma** is a deep form of impetigo causing deeper erosions of the skin into the dermis.

Secondary infection of wounds or other skin lesions with the same pathogens is called ‘impetiginisation’.



Honey-coloured crusts on the chin in impetigo



‘Kissing lesions’ on both sides of the axilla in impetigo



Perioral honey-coloured crusts in impetigo



Widespread bullous impetigo over the back



An intact blister and erosions in bullous impetigo



Honey-coloured crusted lesions in facial impetigo

For more images of impetigo, [click here](#).

Who gets impetigo?

Impetigo is most common in young children but can occur at any age. It is usually transmitted through direct contact.

Risk factors which may predispose an individual to impetigo include:

Skin conditions: [atopic dermatitis](#), [contact dermatitis](#), [scabies](#), [chickenpox](#)

Skin trauma: lacerations, [insect bites](#), [thermal burns](#), abrasions

Immunosuppression

Warm, humid climate

Poor hygiene

Crowded environments.

What causes impetigo?

Impetigo is caused by *Staphylococcus aureus*, and less commonly *Streptococcus pyogenes*.

Non-bullous impetigo

Caused by either *Staphylococcus aureus*, *Streptococcus pyogenes*, or both bacteria conjointly.

Intact skin is usually resistant to colonisation from bacteria. Disruption in skin integrity allows for invasion of bacteria via the interrupted surface.

Bullous impetigo

Due to *Staphylococcus aureus* which produces exfoliative toxins (exfoliatins A and B).

Exfoliative toxins target intracellular adhesion molecules (desmoglein - 1) present in the epidermal granular layer.

Results in dissociation of epidermal cells which causes blister formation.

Can occur on areas of intact skin.

What are the clinical features of impetigo?

Non-bullous impetigo

Most commonly found on the face or extremities but skin on any part of the body can be involved.
Begins with a single erythematous macule which evolves into a pustule or vesicle.
Pustule or vesicle ruptures releasing serous contents which dries leaving a typical honey-coloured crust.
Minimal or no surrounding erythema.
Can spread rapidly with satellite lesions due to autoinoculation.
"Kissing lesions" arise where two skin surfaces are in contact.
Patients are typically otherwise well; they may experience some [itching](#) and regional [lymphadenopathy](#).

Bullous impetigo

Usually found on the face, trunk, extremities, buttocks, and perineal regions.
Can spread distally due to autoinoculation.
Present as quickly appearing superficial, small or large thin roofed bullae which tend to spontaneously rupture and ooze yellow fluid leaving a scaley rim (collarette).
More likely to have systemic symptoms of malaise, fever, and lymphadenopathy.

Crusted weepy lesions in facial impetigo

Impetigo on the leg

Facial impetigo

How do clinical features vary in differing types of skin?

The initial erythematous macule in non-bullous impetigo may be more difficult to see on darker skin tones.

What are the complications of impetigo?

Wider spread infection: [cellulitis](#), lymphangitis, and bacteraemia.
[Staphylococcal scalded skin syndrome](#).

Scarlet fever.

Post-streptococcal glomerulonephritis: a rare, acute renal condition following infection with *Streptococcus pyogenes* (group A streptococcus). This is due to a [type III hypersensitivity reaction](#) and presents 2–6 weeks post-skin infection.

Streptococcal toxic shock syndrome: a rare complication causing diffuse erythematous rash, hypotension, and pyrexia.

Postinflammatory pigmentation.

Scarring, particularly with [ecthyma](#).

How is impetigo diagnosed?

Impetigo is usually a clinical diagnosis based on the features described above.

A skin swab for culture and sensitivity may be beneficial if the impetigo is recurrent, widespread or there is concern of [MRSA infection](#).

Nasal swabs should be carried out in recurrent infection as they can identify staphylococcal nasal carriage which requires specific management.

Rarely a [biopsy](#) may be indicated if the diagnosis is unclear (in particular for bullous impetigo) or if it is refractory to treatment.

Histological features are characteristic.

What is the differential diagnosis for impetigo?

[Pemphigus foliaceus](#)

[Pemphigus vulgaris](#)

[Folliculitis \(pustular\)](#)

[Herpes simplex virus](#)

What is the treatment and prevention of impetigo?

General measures

Regular gentle cleansing; removal of honey-coloured crusts.

Practice good hand hygiene and keep fingernails cut short.

Cover the affected areas with watertight dressing to prevent spread.

Specific measures

Topical antibiotics

For localised non-bullous impetigo, application of [antiseptic](#) 2–3 times per day for 5–7 days is recommended (e.g. hydrogen peroxide 1% cream or povidone – iodine 10% ointment).

Topical antibiotics such as [fusidic acid](#) or [mupirocin](#) are effective in treating non-bullous impetigo, however, their use may not be recommended in some countries due to bacterial resistance.

Topical antibiotics can be considered when antiseptic treatment has not worked or is not appropriate (e.g. impetigo around the eyes).

Fusidic acid is first-line.

Mupirocin use is often reserved for possible MRSA infection.

Oral antibiotics

Recommended in bullous impetigo, widespread non-bullous impetigo (>3 lesions), when topical treatment fails, a person is at high risk of complications, or when a person is systemically unwell.

Oral [flucloxacillin](#) is often the first line antibiotic of choice.

Alternatives may include trimethoprim + sulfamethoxazole or erythromycin (eg, if penicillin allergic or for MRSA infection).

Preventative measures

Avoid touching affected areas.

Practice good hand hygiene; wash hands before and after applying creams.

Use a clean cloth each time to wash and dry affected areas.

Do not share towels or face cloths.

Clothing and bedding should be changed daily; wash using hot temperatures.

Avoid close contact with others – school/nursery children should stay home until lesions have crusted over, or they have received at least 24 hours of treatment.

What is the outcome for impetigo?

Impetigo is usually self-limiting without serious complications. Without treatment, impetigo usually heals in 2–3 weeks; with treatment lesions resolve within 10 days.

Postinflammatory hypopigmentation or hyperpigmentation may occur but scarring is uncommon.

Infantile acne

Author: Dr Amanda Oakley, Dermatologist, Hamilton, New Zealand, 1999. Reviewed by Vanessa Ngan, Staff Writer, February 2014.

What is infantile acne?

True infantile acne generally affects the cheeks, and sometimes the forehead and chin, of children aged six weeks to one year. It is more common in boys and is usually mild to moderate in severity. In most children it settles down within a few months.

The acne may include [comedones](#) (whiteheads and blackheads), inflamed papules and pustules, nodules, and cysts. It may result in [scarring](#).



Infantile acne



Infantile acne



Infantile acne

[See more images of acne in young children.](#)

What is the cause of infantile acne?

The cause of infantile acne is unknown. It is thought to be genetic in origin. It is not usually due to excessive testosterone or other androgenic hormones and children with infantile acne are usually otherwise quite normal in appearance.

Acne is rare in older prepubertal children aged 2 to 6. It is associated with higher levels of androgens than is expected for the age of the child. These may result in virilisation. Signs of virilisation are:

- Excessive body hair
- Abnormal growth
- Genital and breast development
- Body odour.

Hormone abnormalities in children with acne may be associated with the following conditions:

- Congenital adrenal hyperplasia
- Cushing syndrome
- 21-Hydroxylase deficiency
- Precocious [puberty](#)
- Androgen-secreting tumours
- Medications
- Premature adrenarche ([early puberty](#)).

Should any tests be done?

In most babies with acne, no investigations are necessary. In older children, or if there are other signs of virilisation, the following screening tests may be useful.

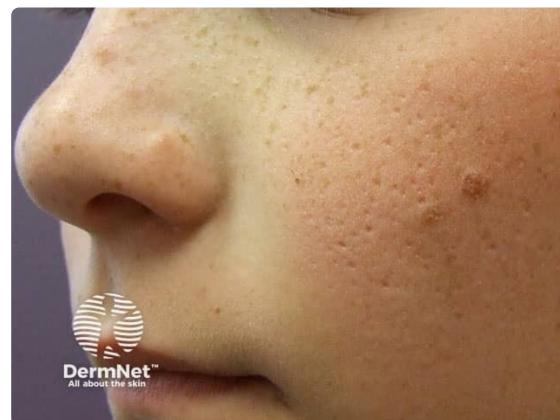
- Blood tests: DHEAS, testosterone, 17-hydroxyprogesterone, LH, FSH, prolactin
- X-ray: bone age measurement

What is the result of infantile acne?

Severe infantile acne may result in permanent [scarring](#). Individuals with severe infantile acne tend to develop troublesome [acne at puberty](#).



Scarring from infantile acne



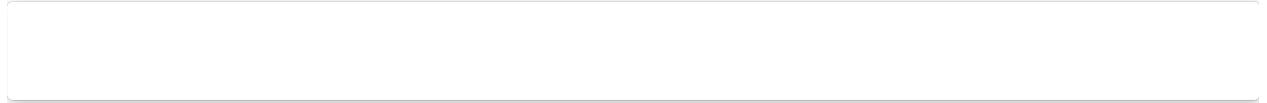
Scarring from infantile acne

What is the treatment for infantile acne?

Treatment of infantile acne is usually with topical agents such as [benzoyl peroxide](#) or [erythromycin](#) gel.

In severe cases, oral [antibiotics](#) such as [erythromycin](#) and trimethoprim, or [isotretinoin](#) may be required.

NOTE: [tetracycline](#) antibiotics should not be used in young children because they may cause yellow staining of the developing permanent teeth.



Iron deficiency

Author: Vanessa Ngan, Staff Writer, 2005. Updated by Dr Sara de Menezes, Basic Physician Trainee, Alfred Health, Melbourne, Australia; Chief Editor, Dr Amanda Oakley, Hamilton, New Zealand, July 2016.

What is anaemia?

Anaemia (American spelling, anemia) is a deficiency of red blood cells. It can occur either through the reduced production or an increased loss of red blood cells.

Three essential elements must be present to produce red blood cells: iron, vitamin B12 and folic acid. The most common cause of anaemia is iron deficiency, affecting more than 2 billion people worldwide.

What is iron-deficiency anaemia?

The estimated prevalence of iron deficiency worldwide is double that of iron deficiency anaemia. Iron deficiency anaemia occurs when there is insufficient iron to create red blood cells

Who gets iron deficiency?

The main groups at risk of iron deficiency and iron-deficiency anaemia are pre-school children, adolescents, pregnant and young women, which are times of increased physiological need for iron.

What causes iron deficiency?

In people living in developing countries, iron deficiency tends to be due to insufficient dietary iron intake or to blood loss from intestinal worm colonisation. In high-income countries, iron deficiency may result from a vegetarian diet, chronic blood loss, or malabsorption.

Diet-related iron deficiency

Malnutrition – poverty, premature babies (milk is a poor source of iron), young children who are picky eaters

Strict vegetarian and vegan diets

Cereal-based diets – decreases iron bioavailability, as phytates in grains reduce iron absorption

Blood loss

Heavy menstruation (periods)

Gastrointestinal bleeding – from peptic ulcer, polyps or cancer, may occur over a long period

Excessive blood donation

Gastrointestinal iron deficiency

Malabsorption

Crohn disease

Helicobacter infection or atrophic gastritis, which may also lead to B12 deficiency

Intestinal parasitic infections, such as **hookworm** or tapeworm

Medication-related iron-deficiency

Aspirin and [non-steroidal anti-inflammatory drugs](#) – cause gastritis

Proton pump inhibitors – may impair iron absorption

Other conditions

Pregnancy

Bleeding disorders, such as von Willebrand disease

End-stage renal failure – a combination of blood loss from dialysis and low erythropoietin levels (a hormone that stimulates red blood cell production)

Congestive cardiac failure – possibly due to subclinical inflammation and impaired iron absorption

Myelodysplasia – bone marrow disease which can present with anaemia

Intravascular haemolysis (rare) as in paroxysmal nocturnal haemoglobinuria

What are the clinical features of iron deficiency?

The signs and symptoms of an iron deficiency depend on whether the patient is anaemic, and if so, how fast the anaemia develops. In cases where anaemia develops slowly, the patient can often tolerate extremely low concentrations of red blood cells (< 100 g/L) for some weeks before developing any symptoms. The first symptoms to appear are due to low delivery of oxygen to tissues, and may include:

Lethargy

Weakness

Poor concentration

Shortness of breath

Palpitations.

Skin signs of iron deficiency anaemia

Skin signs of anaemia are often subtle and may include:

Paleness of skin, palm creases and conjunctiva

[Angular cheilitis](#), painful cracks at the corners of the mouth

Atrophic [glossitis](#), loss of tongue papillae (smooth, shiny tongue)

[Pruritus](#) and [dry skin](#)

[Nail disorders](#), including koilonychia

Dry and brittle hair

Increased hair shedding ([telogen effluvium](#)) resulting in [diffuse alopecia](#).



Angular cheilitis



Koilonychia

Systemic symptoms of iron deficiency anaemia

Other characteristic manifestations of iron deficiency anaemia may include:

Pica – an appetite for clay, dirt, paper or starch

Pagophagia – a pica for ice, considered quite specific for iron deficiency. Responds rapidly to iron replacement.

Beeturia – excretion of red urine with the consumption of beets. In people with normal iron levels, ferric ions decolourise betalain (the red pigment in beets). In iron-deficient states, there are inadequate amounts of iron to decolourise this pigment.

Restless legs syndrome – marked discomfort in the legs occurring at rest that is relieved by movement.

Iron deficiency may also predispose to [bacterial](#) and [fungal infections](#) such as [impetigo](#), [boils](#) and [candidiasis](#).

What tests should be done?

Full blood count

A full, or complete, blood count (FBC, CBC) is essential to detect anaemia. Iron deficiency can be present when blood count indices are normal.

If anaemia is due to iron deficiency, the cells are smaller and contain less haemoglobin resulting in lowered red blood cell count or haematocrit, mean corpuscular volume (MCV) and mean cell haemoglobin concentration (MCH). Reticulocyte haemoglobin content (Ret-Hb), which tends to be low in iron deficiency anaemia, can be used to monitor response to iron replacement. Red cell distribution width (RDW) can reveal mixed iron and vitamin B12 deficiency as this results in red cells of variable size.

Ferritin

Ferritin is a measure of iron stores and is the most sensitive and specific test for iron deficiency. Low levels of ferritin less than 15 µg/ml are diagnostic of iron deficiency. Levels higher than 40 µg/ml in a healthy person are considered optimal.

Normal or high levels of ferritin do not exclude iron deficiency, because ferritin acts as an acute phase reactant. Levels are higher in the presence of chronic inflammation (eg, [rheumatoid arthritis](#)) when erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP) are elevated. In the context of inflammation, significantly higher cut-off values for ferritin are used (eg, 100 µg/ml) and are more predictive of iron deficiency. Ferritin is also more elevated in patients with chronic kidney disease and heart failure.

Other iron tests

In iron deficiency:

Serum iron is reduced – be aware that serum iron can be very variable, fluctuating through the day, and serum iron is not useful in assessing iron stores

Iron binding capacity is increased – a measure of the capacity of iron to bind with transferrin (an iron transporter)

Transferrin saturation is reduced

Soluble transferrin receptor (sTfR) is reduced – this reflects total body stores, except if there is a disease of the bone marrow. sTfR is an expensive test. It is useful at discriminating iron deficiency in difficult cases, for instance, in patients with chronic renal failure or chronic inflammation like rheumatoid arthritis. It is unchanged in anaemia of chronic disease.

Retest iron status after three months of iron supplementation.

Older patients sometimes have unexplained iron deficiency anaemia. If bowel investigation is negative, bone marrow examination may be considered in undifferentiated cases.

What is the treatment for iron deficiency?

Once iron deficiency has been established, the underlying cause should be investigated and managed (correct/control GI bleeding or menstrual blood loss, eg, with the levonorgestrel-releasing intrauterine device or [tranexamic acid](#) for a woman with heavy periods). Most people with iron deficiency anaemia will need iron replacement therapy to correct the anaemia and replenish iron stores. The benefit of treating iron deficiency without anaemia is still uncertain. Specific groups of patients like those with cardiovascular disease (with heart failure or angina) should receive red blood cell transfusions which will correct both hypoxia (low oxygen) and the iron deficiency.

Increase dietary iron

Red meat contains haem iron, which is readily absorbed. Non-haem iron sources may need the help of vitamin C in the form of fresh fruit or tablets.

Many manufactured foods contain iron, so it is essential to read the labels.

Calcium (in milk products) and tannin in tea, coffee and red wine, reduce the absorption of non-haem iron, so these should be taken several hours before a meal. Conversely, vitamin C (ascorbic acid) enhances the absorption of iron when they are taken together.

Oral iron

Iron supplementation is safe in pregnancy, infants, children and adults. It can be used in iron deficiency anaemia and anaemia of chronic disease.

Iron preparations come in the form of tablets, oral liquids and injection. Oral preparations are most commonly used.

Oral iron preparations from reputable sources include:

- Ferrous fumarate 33% elemental iron
- Ferrous sulfate 20% elemental iron
- Ferrous gluconate 12% elemental iron

Enteric-coated and slow-release formulations are less well absorbed, but better tolerated. Taking iron with vitamin C (ascorbic acid) may increase its absorption and help replenish iron stores more quickly. Lower dose preparations are less effective.

In anaemic patients, once haemoglobin levels are corrected to within the normal range, iron replacement should be continued for a further three months to replenish iron stores. Aim for serum ferritin levels over 50 µg/ml.

Iron absorption is reduced in the presence of gastrointestinal disease (atrophic gastritis, infection with *Helicobacter pylori*, coeliac disease, inflammatory bowel disease), chronic kidney disease and inflammatory conditions.

Interactions with iron

Iron may interfere with the absorption of some medications, including:

Doxycycline
Fluoroquinolones
[Mycophenolate mofetil](#)
Penicillamine
Thyroid hormones.

Iron absorption is decreased by calcium, tannins (in tea and red wine) and plant phytates (in cereals). Iron should be taken at a different time of day.

Iron infusions

Intravenous infusions are used in patients that cannot tolerate oral supplementation, or where iron losses exceed the daily amount that can be absorbed orally. Intravenous iron is also essential in the management of anaemia in patients with chronic kidney disease that are receiving dialysis and treatment with erythropoiesis-stimulating agents (agents to stimulate red blood cell production). Parenteral iron in patients with heart failure has led to improvements in physical performance, symptoms and quality of life.

The most commonly used intravenous preparation is iron polymaltose, which is infused over several hours. Other intravenous preparations include low molecular weight iron dextran, iron carboxymaltose, iron sucrose and ferric gluconate complex.

Side effects of iron replacement

Adherence to recommended oral iron replacement therapy may be poor with some patients as iron preparations are associated with a high incidence of side effects. These include nausea, constipation, diarrhoea and black stools. To reduce this:

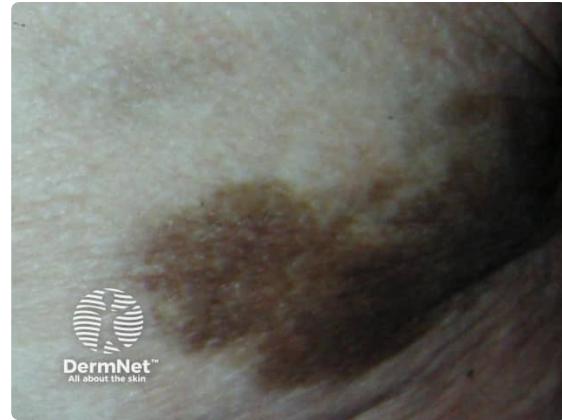
Take the iron preparation after meals — but iron absorption is reduced
Wait 30 minutes before lying down
Divide the dose and take it twice daily
Take it alternate days, which is better tolerated
If treatment is not urgent, start with one tablet twice weekly and gradually increase the dose as tolerated
Start with doses containing under 30 mg of elemental iron.

Intravenous iron polymaltose may cause infusion reactions such as headache, nausea and muscle pains. Severe allergic reactions including [anaphylaxis](#) have been reported. Delayed reactions include fever and joint pain. Extravasation is rare but may lead to persistent brown discolouration of affected skin.

Intramuscular injections of iron are now rarely used. They may result in long-lasting brown staining (siderosis), pain, haematoma and sterile abscesses. Improvement in iron staining has been reported following treatment with Q-switched [ruby](#) and [Nd:YAG](#) laser.



Intramuscular



Intramuscular, close-up



Intravenous extravasation

What is the outcome for iron deficiency anaemia?

Most patients with uncomplicated iron deficiency anaemia should experience:

- Rapid resolution of pagophagia
- Improved feeling of well-being within the first few days of treatment
- Increase in reticulocyte count (red blood cell precursors) and haemoglobin concentration within a week
- Slow recovery of tongue papillae, skin, nails and hair.

In those who do not respond to treatment, alternative diagnoses need to be considered, for example, B12 or folate deficiencies, myelodysplastic syndrome (bone marrow abnormalities) and inherited anaemias.

Measles

Author: Dr Amanda Oakley, Dermatologist, Hamilton, New Zealand, 2002. Updated by Dr Jannet Gomez, October 2016. Updated October 2020

What is measles?

Do you have confirmed measles? Send DermNet your pictures.

Measles, also known as English measles, rubeola or morbilli, is a highly contagious viral infection causing fever and a rash.

Measles is a notifiable disease.



Koplik spots Day 1



Rash Day 3

[See more images of measles.](#)

What is the cause of measles?

Measles is caused by the measles virus, which belongs to the morbillivirus family.

How common is measles?

Before widespread immunisation against measles in industrialised countries, measles was a very common childhood disease that carried a high death rate. Nowadays in countries where measles is part of an immunisation programme, the risk of exposure and incidence of actual disease cases is low. A recent trend by some parents not to immunise their children has led to an increase in the number of cases of measles, and its complications.

In developing countries, measles still occurs frequently and is associated with a high rate of complications and death. It remains a common disease even in some developed countries of Europe and Asia.

WHO reported a surge in cases worldwide in 2018, with nearly 10 million cases, and more than 140,000 deaths mostly in children under 5 years of age. The highest incidence rates were in the Ukraine, Somalia, Democratic Republic of Congo, Liberia and Madagascar. The US reported its highest rate of infection in 25 years, and four countries in Europe lost their 'measles elimination' status.

How do you get measles?

Measles is highly contagious and is easily spread from person to person by breathing in airborne respiratory droplets from an infected person's coughing or sneezing.

An infected person is contagious 2 days before any symptoms show, and remains infectious for at least 5 days after the onset of rash.

An acute infection of measles almost always gives lifelong immunity.

Who is at risk of measles?

Individuals at particular risk of measles infection include:

Infants who have lost their passive immunity from their mothers (acquired from their mother through the transfer of antibody across the placenta) and before their first immunisation

Unvaccinated travellers to areas where measles is endemic

Individuals with [immunodeficiency](#) (eg, due to infection with [HIV/AIDS](#), leukaemia, cancer, [corticosteroid therapy](#)), regardless of their immunisation status

Migrants and refugees.

The greatest risk for severe measles and its complications is seen in:

Malnourished individuals (particularly children who are deficient in vitamin A)

Those with an underlying immune deficiency

Pregnant women.

What are the signs and symptoms of measles?

Measles develops through distinct clinical stages.

Incubation period

Ranges from 7–14 days (average 10–11 days).

The patient usually has no symptoms.

Some may experience symptoms of primary viral spread (fever, spotty rash, and respiratory symptoms due to virus in the bloodstream) within 2–3 days of exposure.

Prodrome

Generally begins 10–12 days after exposure.

Presents as fever, malaise, and loss of appetite, followed by conjunctivitis (red eyes), cough, and coryza (blocked or runny nose).

2–3 days into the prodromal phase, Koplik spots appear. These are blue-white spots on the inside of the mouth opposite the molars, and occur 24–48 hours before the **exanthem** (rash) stage.

Prodromal symptoms usually last for 2–5 days but in some cases may persist for as long as 7–10 days.

Exanthem (rash)

Flat red spots ranging from 0.1–1.0cm in diameter appear on the 4th or 5th day following the start of symptoms.

This non-itchy rash begins on the face and behind the ears. Within 24–36 hours it spreads over the entire trunk and extremities (palms and soles rarely involved).

The spots may join together, especially in areas of the face.

The onset of the rash usually coincides with a high fever of at least 40C.

The rash begins to fade 3–4 days after it first appears. It fades first to a purplish hue and then to brown/coppery coloured lesions with fine scales.

Recovery

A cough may persist for 1–3 weeks.

Measles-associated complications may be the cause of persisting fever beyond the 3rd day of the rash.

Measles exanthem



How is measles diagnosed?

Diagnosis of measles is based on the characteristic history and physical examination. Because the disease is now so rarely seen in developed countries, any suspected cases require laboratory confirmation. This is particularly useful in the following situations:

- Sporadic cases
- Atypical cases
- Confusion with other diseases.

Acute measles is usually confirmed on a viral nasopharyngeal or throat swab analysed by polymerase chain reaction (PCR). Blood and urine samples can also be used. This should be done within 5 days of onset of rash, however, positive results are sometimes obtained up to 10–14 days after the rash has resolved.

Blood is also taken for measles IgM and IgG antibodies (serology). Levels of specific IgM become elevated during the active infection phase and IgG antibody appears during the recovery phase.

Viral culture of the throat and nasopharyngeal swabs is preferred in immunocompromised patients where serological evidence might be absent due to decreased immune response. An immunofluorescence test for measles antigen can also be considered in patients with poor immunity.

What is the treatment of measles?

There is no specific treatment for measles, which is why immunisation is so important. Treatment for mild cases of measles is supportive. Bed rest is vital, as it prevents complications and prevents the spread of the virus.

Give paracetamol for fever. Aspirin should not be given to a child with a viral illness, as it is associated with the development of Reyes syndrome.

Vitamin A for children in developing countries or that are malnourished. WHO recommends a dose of 200,000 international units (IU) of vitamin A for two days, for reducing measles and its complications. A Cochrane Review found this reduced mortality and pneumonia-related mortality in children under the age of 2 years.

Maintain adequate fluid intake to prevent dehydration.

Use a humidifier to provide relief from cough/sore throat.

Provide nutritional support as necessary.

Observe high-risk individuals carefully to prevent complications.

Patients with drowsiness, dehydration, or discomfort breathing require hospitalisation for supportive care.

Antibiotics are only needed to treat secondary bacterial infections such as otitis media, infectious diarrhoea, pneumonia, and sepsis.

Ribavirin (antiviral) is used to treat measles infection in immunocompromised patients and in those affected with subacute sclerosing panencephalitis.

What are the complications from measles?

Approximately 30% of reported measles cases have one or more complications.

Gastrointestinal: diarrhoea that may be fatal if dehydration occurs, [mouth ulceration](#), appendicitis, hepatitis, mesenteric adenitis, and pancreatitis.

Ears: otitis media (almost exclusively in children) may lead to deafness.

Respiratory tract: laryngobronchitis, measles croup, and pneumonia (either primary viral or secondary bacterial) – the most common cause of death from measles.

Heart: myocarditis and pericarditis.

Haematological system: thrombocytopenia, causing bleeding and [disseminated intravascular coagulation \(DIC\)](#).

Eyes: conjunctivitis and/or corneal ulceration leading to blindness (especially if vitamin A deficient), and squint.

Kidneys: acute glomerulonephritis (inflammation of kidneys) and renal failure.

Nervous system: febrile seizures and encephalitis.

[Malnutrition](#) (especially if from a poor community).

Measles infection during pregnancy increases the risk of premature labour and delivery, fetal loss and maternal death.

Rarely, subacute sclerosing panencephalitis—a fatal condition—develops decades after a measles infection due to persistence of the measles virus in the central nervous system.

How can measles be prevented?

Measles can be prevented by vaccination with live attenuated measles vaccine. It is available as a single antigen preparation or combined with live attenuated mumps and/or rubella vaccines.

Combined measles, mumps and rubella (MMR) vaccine is currently part of routine immunisation programmes in most industrialised countries, including New Zealand.

Measles vaccine induces long-term (probably life-long) immunity in most individuals. Vaccination schedules recommend a two-dose immunisation strategy; the first dose at 12–15 months of age, followed by a second dose at 4–6 years.

Measles vaccine should not be given during pregnancy. Women not previously immunised against measles should avoid pregnancy for one month (28 days) after receiving the MMR vaccine.

Immune globulin does not prevent measles, but it is helpful in decreasing the severity of illness in those exposed to the virus. It is recommended for:

Pregnant women

People with immune deficiency

Infants.

Individuals vaccinated prior to 1968 may require revaccination, as vaccines used before this time may not have conferred life-long immunity.

Melanoma

October 2022

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What is melanoma?

Melanoma, also referred to as malignant melanoma, is a potentially very serious skin cancer in which there is an uncontrolled growth of melanocytes (pigment cells).

Normal melanocytes are found in the basal layer of the epidermis (outer layer of skin). Melanocytes produce a protein called melanin, which protects skin cells by absorbing [ultraviolet \(UV\) radiation](#).

Non-cancerous growth of melanocytes results in [moles \(benign melanocytic naevi\)](#) and freckles (ephelides and lentigines). In contrast, the cancerous growth of melanocytes results in melanoma.

Melanoma is described as:

[In situ](#), if a tumour is confined to the epidermis

[Invasive](#), if a tumour has spread into the dermis

[Metastatic](#), if a tumour has spread to other tissues.

Management of melanoma is rapidly evolving. For up-to-date recommendations for the diagnosis and management of melanoma, refer to local guidelines such as the [Australian Cancer Council clinical practice guidelines](#).



Superficial spreading malignant melanoma – irregular border, variable pigmentation, and areas of clinical regression



Superficial spreading malignant melanoma – an enlarging pigmented lesion with variable pigmentation, irregular edge, and asymmetry



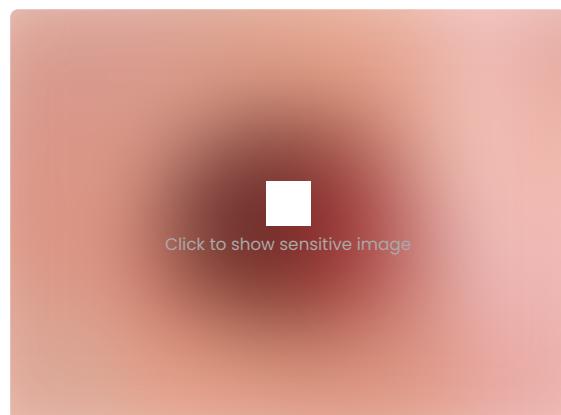
Acral lentiginous malignant melanoma - irregular edge, with variable pigmentation, asymmetry and areas of regression on the heel



Nodular malignant melanoma in a vertical growth phase - rapidly enlarging scaly pigmented nodule



Irregular pigmented longitudinal bands in melanoma of the nail unit



Amelanotic melanoma arising within pigmented melanoma



Amelanotic subungual melanoma - a red lesion arising from the nail fold that has produced destruction of the nail plate



Multiple blue nodules of cutaneous metastatic malignant melanoma



A superficial malignant melanoma - irregular and notched margin, variable and irregular pigmentation in an itchy and enlarging pigmented lesion

For more images, please see links at the bottom of the page.

Who gets melanoma?

Australia and New Zealand have the highest reported incidence and mortality of melanoma globally. About 1 in 15–18 white-skinned Australians and New Zealanders are expected to develop melanoma in their lifetime. Melanoma is the third most common cancer in Australia and New Zealand.

In 2019, there were 2,727 melanoma registrations in New Zealand. There were 362 deaths from melanoma in 2016.

Melanoma can occur in adults of any age but is very rare in children. According to the Australian Institute of Health and Welfare, in 2021:

7.9% of people diagnosed with melanoma were aged under 40 years
10.7% were aged 40–49
17.8% were aged 50–59
23.9% were aged 60–69
23.7% were aged 70–79
16% were aged 80 or older.

The mean age for melanoma diagnosis is 65.7 years among men and 62.4 years among women. Melanoma is the most common cancer diagnosed in young Australians aged 15–29 years, accounting for 15% of all cancers in this age group.

The main risk factors for developing the more common type of melanoma (eg, [superficial spreading melanoma](#)) include:

Increasing age
Previous invasive melanoma or [melanoma in situ](#)
Previous [basal cell](#) or [squamous cell carcinoma](#)
Many melanocytic naevi ([moles](#))
Multiple (>5) [atypical naevi](#) (large or histologically dysplastic moles)
A strong family history of melanoma with two or more first-degree relatives affected
White/fair skin
Parkinson disease
UV exposure
History of [sunburn](#)
Weakened immune system or cancer-prone syndromes.

These risk factors are not as relevant to rarer types of melanoma.

Although the presence of a large number of common nevi is a strong risk factor for cutaneous melanoma, the majority of melanomas arise de novo. A 2017 meta-analysis of 38 studies including over 20,000 melanomas found that only 29 percent were nevus-associated, with the rest arising de novo.

What causes melanoma?

Melanoma is thought to begin as an uncontrolled proliferation of melanin-producing cells (melanocytic stem cells) that have undergone a genetic transformation.

The cause of these cell mutations can be acquired or inherited.

Acquired 'sporadic' mutations are the most common cause of cancer. They occur from damage to a cell during a person's life. The most common cause of melanoma is overexposure to UV radiation, eg, sun exposure, sunbed use.

Germline 'inherited' mutations are passed down from the parent. Inherited (familial) melanoma is far less common (<10%). CDKN2A (also called p16INK4A or MTS1) is the gene primarily linked in up to 20–40% of familial melanomas. However, in recent years, a growing number of other genes have been implicated, including CDK4, MC1R, MITF, TERT, ACD, BAP1, POT1, and TERF2IP.

Cutaneous melanoma can arise from otherwise normal-appearing skin (in about 75% of melanomas) or from within a pre-existing mole or freckle, which starts to grow larger and change in appearance.

Precursor lesions include:

[Benign melanocytic naevus](#) (normal mole)

[Atypical or dysplastic naevus](#) (unusual-looking mole)

Atypical [lentiginous junctional naevus](#) (flat naevus in heavily sun-damaged skin) or atypical [solar lentigo](#)

Large or giant-sized [congenital melanocytic naevus](#) (brown birthmark).

Melanomas have two growth phases, radial and vertical. Most melanomas arise as superficial tumours confined to the epidermis (ie, they have a horizontal growth phase; *in situ*). These generally grow slowly, but at any time, further genetic changes may cause the tumour to progress to the vertical growth phase, in which the malignant cells breach the basement membrane, invading deeper tissues, resulting in invasive melanoma.

Once the melanoma cells have reached the dermis, they may spread to other tissues via the lymphatic system to the local lymph nodes or via the bloodstream to other organs such as the lungs or brain. This is known as metastatic disease or secondary spread. The chance of this happening mainly depends on how deep the cells have penetrated the skin.

Melanoma classification

Subtypes of melanoma

In 2018, the World Health Organization (WHO) revised the classification of melanoma, distinguishing them by their cumulative solar damage (CSD), anatomic site, epidemiology and mutation signatures. These now include low and high CSD melanomas, [desmoplastic melanoma](#), [Spitz melanomas](#), [acral melanomas](#), [mucosal melanomas](#), melanomas in [congenital nevus](#), melanomas in [blue nevus](#), and [uveal melanomas](#).

Conventional classification

The more traditional morphologic classification by histopathology of melanoma, includes the subtypes:

[Superficial spreading melanoma](#) (~55–60% of all melanomas)

[Nodular melanoma](#) (~10–15% of all melanomas)

[Lentigo maligna melanoma](#) and [lentiginous melanoma](#) (in sun-damaged sites) (~10–15% of all melanomas)

[Acral lentiginous melanoma](#) (on soles of feet, palms of hands or nails; <5% of all melanomas).

Less common variants include:

[Desmoplastic melanoma](#) (~1–2% of all melanomas)

[Amelanotic melanoma](#)

[Spitzoid melanoma](#)

[Mucosal melanoma](#)
[Spindle cell melanoma](#)
[Ocular \(eye\) melanoma.](#)

Combinations may also occur, for example, nodular melanoma arising within a superficial spreading melanoma.

Melanoma is usually epithelial in origin ie, starting in the skin or, less often, mucous membranes. However, very rarely, melanoma can start in an internal tissue such as the brain (primary CNS melanoma) or the back of the eye.

Classification by age

Melanoma can also be classified according to its relationship with age.

Childhood melanomas (< 10 years of age)

Extremely rare (<4%)
Risk factors are genetic, environmental and iatrogenic or acquired immunosuppression
Infrequently associated with excessive sun exposure
Compared to melanoma in adults, they are more often amelanotic (flesh coloured, pink or red), nodular, bleeding and ulcerated.
May arise within giant congenital melanocytic naevi > 40 cm diameter
Often spitzoid melanoma type lacking conventional ABCD features (see below)

Early-onset melanomas

More common in women than men
The most common clinical subtype is [superficial spreading](#)
Associated with many melanocytic naevi
Tend to be seen on the lower extremity
Tend to have a BRAF V600E genetic mutation
Associated with intermittent sun exposure

Late-onset melanomas

More common in men than in women
The most common clinical subtype is [lentigo maligna](#)
Often occur on the head and neck
Associated with accumulated lifelong sun exposure

What are the clinical features of melanoma?

Melanomas can occur anywhere on the body, not only in areas that get a lot of sun. In New Zealand, the most common site in men is the back (around 40% of melanomas in men), and the most common site in women is the leg (around 35% of melanomas in women).

Although melanoma usually starts as a skin lesion, it can also grow on mucous membranes ([mucosal melanoma](#)), such as the lips or genitals. Occasionally it occurs in other parts of the body such as the eye, brain, mouth or vagina.

The first sign of a melanoma is usually an unusual looking [freckle](#) or [mole](#) and may itch or bleed. Melanomas may grow across the skin (known as the radial growth phase) or grow in depth (known as the vertical growth phase).

Melanoma may be detected at an early stage when it is only a few millimetres in diameter, but it may grow to several centimetres in diameter before it is diagnosed.

A melanoma may have a variety of colours including:

Tan, dark brown, black, blue, red and, occasionally, light grey.

Melanomas that are lacking pigment are called amelanotic melanoma.

There may be areas of regression that are the colour of normal skin, or white and scarred.

During its horizontal phase of growth, a melanoma is normally flat. As the vertical phase develops, the melanoma becomes thickened, raised, and palpable.

Some melanomas are itchy or tender. More advanced lesions may bleed easily or crust over.

Most melanomas have characteristics described by the ABCDE+EFG melanoma criteria or the Glasgow 7-point checklist.

The ABCDEG of Melanoma

For [superficial melanomas](#) — ABCDE signs

- A: Asymmetry of shape and colour
- B: Border irregularity, including smudgy or ill-defined margin
- C: Colour variation and change
- D: Different (formerly diameter)
- E: Evolving (enlarging, changing)

Melanomas may not conform to the 'ABCD' rule alone. For nodular melanomas, also consider the EFG signs

- E: Elevated
- F: Firm to touch
- G: Growing

See [ABCDE+EFG criteria](#).

Taking a thorough history is important, and any lesion that changes in size, shape, colour, or elevation for more than one month should be reviewed by a dermatologist or biopsied.

Glasgow 7-point checklist

Major features

- Change in size
- Irregular shape
- Irregular colour

Minor features

- Diameter >7 mm
- Inflammation
- Oozing

How do clinical features vary in differing types of skin?

White or pale skin colour is an independent but significant risk factor for melanoma across diverse ethnic groups. However, people of all skin colours with a family history of melanoma are at increased risk of developing melanoma due to a genetic predisposition.

In skin of colour, it can be harder to identify melanomas, their growth phase, and their pattern as the surrounding skin may mask or match the colour of the melanoma.

People with skin of colour tend to have:

Thicker melanomas at diagnosis and higher mortality rates

Significantly higher rates of melanomas in areas not exposed to the sun, including the subungual, palmar, and plantar surfaces (eg, [acral lentiginous melanoma](#) in Pacific Islanders, blacks, and Asians)

Non-cutaneous melanomas (eg, [mucosal melanoma](#), [ocular melanoma](#)).

This topic is further discussed on the [melanoma of skin colour](#) page.

What are the complications of melanoma?

[Metastasis](#) and related systemic effects

Side effects from systemic or radiation therapy

Death

Complications due to surgery:

Wound infection

Inability to close

Dehiscence

Skin necrosis

Incomplete resection

Seromas, lymphoedema, and lymphoceles with node dissection.

How is melanoma diagnosed?

Melanoma may be suspected because of a lesion's clinical features or a history of change.

A thorough history and skin examination will be performed using the "[ugly duckling](#)" sign, ABCDE rule, and the Glasgow revised seven-point checklist (described above). This may be supported by [dermoscopy](#), [confocal microscopy](#), [total body photography \(mole mapping\)](#), and [adhesive patch genomic analysis](#), among other methods.

Dermoscopy — Some melanomas are extremely difficult to recognise clinically. The dermatoscopic appearance is helpful in the diagnosis of melanoma. Melanoma-specific criteria often include an atypical pigment network; brown-black dots/globules; multiple (5–6) colours asymmetrically distributed; blue-white veil; depigmentation; and irregular vascular pattern. Dermoscopy monitoring should only be considered for flat or slightly raised lesions, whereas any suspicious or nodular lesions should be excised.

Confocal Microscopy — Where available, reflectance confocal microscopy (RCM) may be useful for clinical and dermoscopic examination of suspected melanoma lesions. RCM is a live imaging technology that allows for instant viewing of the epidermis and papillary dermis with nearly histologic resolution. Areas of higher melanin concentration appear as bright areas on a confocal

image. This technology is especially useful for identifying amelanotic or hypomelanotic melanomas, mapping surgical margins pre-operatively, and monitoring lesions over time.

Photographic skin surveillance – Where available, total body photography (TBP), eg, MoleMap, should be considered for high-risk individuals, particularly those with high naevus counts and dysplastic naevi. TBP provides a baseline for monitoring new lesions and changes in pre-existing naevi. While not all changed lesions need to be excised, if there is clinical or dermoscopic evidence for melanoma at any point, excision is recommended.

Radiographic investigations – Imaging, such as CT or PET, may be appropriate for staging or surveillance of melanomas that are at significant risk for distant metastases.

Adhesive patch genomic analysis – skin surface tape stripping is a non-invasive test that can be used on pigmented lesions to obtain their genomic signature. It can be used to assist in the diagnosis of melanoma.

Biopsy

Histopathology is required for the definitive diagnosis of melanoma.

Where possible, suspicious lesions should be surgically removed by **diagnostic excision** with a 2 mm clinical margin and upper subcutis for pathological examination. A partial (incisional) biopsy is best avoided but may be considered where complete excision is not feasible, eg, large lesions. **Nail biopsy** for suspected **melanoma of the nail** is discussed elsewhere.

The **pathological diagnosis of melanoma** can be very difficult. **Immunohistochemical stains** may help confirm melanoma and include S-100, SOX10, MART-1, HMB-45, MITF, tyrosinase and PRAME. Also, molecular techniques such as gene expression profiling and fluorescence in situ hybridisation (FISH), may aid in diagnosis.

If there is invasive melanoma, the pathologist will comment on:

Diagnosis of primary melanoma

Breslow thickness—the Breslow thickness is reported for invasive melanomas to the nearest 0.1 mm. It is measured vertically in millimetres from the top of the granular layer (or base of superficial ulceration) to the deepest point of tumour involvement. It is a strong predictor of outcome; the thicker the melanoma, the more likely it is to metastasise (spread).

Clark level of invasion—indicates the anatomic plane of invasion. Deeper Clark levels have a greater risk of metastasis. It is useful in predicting the outcome of thin tumours. It is less useful than Breslow thickness for thick tumours.

Level 1: In situ melanoma

Level 2: Melanoma has invaded the papillary dermis

Level 3: Melanoma has filled the papillary dermis

Level 4: Melanoma has invaded the reticular dermis

Level 5: Melanoma has invaded the subcutaneous tissue

Margins of excision—the normal tissue around a tumour

Mitotic rate—a measure of how fast the cells are proliferating

Presence of ulceration.

The report may also include comments about the cell type, growth pattern, invasion of blood vessels or nerves, inflammatory response, regression, associated in-situ disease and any associated naevus (original mole).

Staging

Melanoma staging means finding out if the melanoma has spread from its original site in the skin. Most melanoma specialists refer to the American Joint Committee on Cancer (AJCC) cutaneous melanoma staging guidelines (8th edition, 2018). In summary, the stages are:

Stage	Characteristics
Stage 0	In situ melanoma
Stage 1	Thin melanoma 2 mm in thickness
Stage 2	Thick melanoma > 2 mm in thickness, or > 1mm thickness with ulceration
Stage 3	Melanoma spread to involve local lymph nodes
Stage 4	Distant metastases have been detected

What is the differential diagnosis for melanoma?

Benign melanocytic naevi (moles)

Pigmented [basal cell carcinoma](#) (the most common skin cancer in whites, Asians, and Hispanics)

[Squamous cell carcinoma](#) (the most common skin cancer in blacks and Indians)

Pigmented [actinic keratosis](#)

[Seborrheic keratosis](#)

[Dermatofibroma](#)

[Pyogenic granuloma](#)

[Postinflammatory hyperpigmentation](#)

[Keloid scars](#)

[Cherry haemangioma](#)

What is the treatment for melanoma?

Overview

A dermatologist and oncologist may both be involved to provide a recommended treatment option based on the melanoma stage and other factors such as age and general health.

[Surgery](#) is the most common treatment for early-stage (Stage 0, I or II) melanoma. If caught early, 90% of melanomas can be cured with simple surgery alone.

More advanced melanomas (Stage III or IV) may require a combination of treatments, including surgery, drug therapy, and [radiation therapy](#). All melanoma patients with distant metastases should be reviewed by a multidisciplinary team to ensure an optimal treatment combination.

Melanoma therapy is rapidly evolving with the frequent availability of new and more effective treatments. Access to these new treatments is dependent on the results of clinical trials and approval by government bodies. *For an up-to-date summary of therapies available, please refer to your local guideline such as the Australian Cancer Council clinical practice guidelines for the diagnosis and management of melanoma.*

Specific measures

Wide local excision

Following confirmation of the diagnosis, [wide local excision](#) is carried out at the primary melanoma site. The extent of surgery depends on the thickness of the melanoma and its site.

Margins recommended in New Zealand (2013):

- Melanoma in situ: 5–10 mm
- Melanoma < 1 mm: 10 mm
- Melanoma 1–2 mm: 10–20 mm
- Melanoma > 2 mm: 20 mm

Australian Clinical Practice Guidelines for the diagnosis and management of melanoma (updated 2018), recommend, where possible:

- Melanoma in situ: 5 mm, and wider margins if appropriate
- Melanoma < 1 mm: 10 mm
- Melanoma 1–2 mm: 10 – 20 mm
- Melanoma 2–4 mm: 10 – 20 mm
- Melanoma > 4 mm: 20 mm

Systemic therapy

If the melanoma is widespread, surgical treatment is not always successful in eradicating cancer. Some patients may be offered new or experimental treatments. Currently, there are two main types of drug therapy used to treat melanoma:

Targeted therapy

- BRAF inhibitors: [dabrafenib](#) and [vemurafenib](#)
- MEK inhibitors: [trametinib](#)
- Combination BRAF and MEK inhibitors: [dabrafenib](#)
- C-KIT inhibitors: [imatinib](#), [nilotinib](#)
- Immunotherapy/immune-modulating therapy
 - PD-1 antagonist: [pembrolizumab](#) and [nivolumab](#)
 - PD-L1: [atezolizumab](#)
 - CTLA-4 antagonist: [ipilimumab](#)
 - LAG-3 inhibitor: [relatlimab](#)
 - Interleukin-2 (IL-2)**
 - Interferon alfa 2b**
 - Chimeric antigen receptor T cell therapy (CAR-T)
 - Imiquimod**
 - Oncolytic virus - [Talimogene laherparepvec \(Imlygic\)](#)

For a more detailed summary of the immunotherapy agents currently available, see the [Cancer Research Institute](#) website.

Chemotherapy is rarely used in Australia and New Zealand for melanoma management.

Radiation therapy

[Radiation therapy](#) is a localised treatment that uses high-energy radiation to kill cancer cells. It may be given on its own (when the patient has a melanoma too widespread for surgery or is unfit for surgery), after surgery (particularly when there is a high risk of recurrence) or as a combination therapy.

Radiation therapy should be considered in patients with a single or small number of brain metastases, painful bone metastases, problematic skin, soft tissue or nodal metastases that have not responded to systemic therapy. This may help relieve symptoms from the metastases.

Should the lymph nodes be removed?

In Australia and New Zealand, it is recommended that lymphatic mapping with a [sentinel node biopsy](#) is performed for melanomas thicker than 1 mm or where the melanoma is greater than 0.75 mm with other high-risk pathological features. However, while the biopsy may help in staging cancer and directing the use of adjuvant therapies, it does not offer any survival advantage.

When the sentinel node is removed it is assessed for the presence of malignant cells, which may indicate melanoma spread to other body parts. In the past, a positive sentinel node biopsy required a [lymph node dissection](#) to remove all the lymph nodes in the area. However, this is no longer the preferred treatment. Patients may choose to have the procedure to reduce the risk of lymph node field relapse.

What happens at follow-up after a melanoma diagnosis?

Follow-up after a melanoma diagnosis is required to:

Detect recurrence and metastasis ([metastatic melanoma](#)) early

Diagnose a new primary melanoma at the first possible opportunity. A second invasive melanoma occurs in 5–10% of melanoma patients, and a new [melanoma in situ](#) is diagnosed in more than 20% of melanoma patients.

The current Cancer Council Australia and Melanoma Institute Australia (2022) Clinical Guidelines make the following recommendations for follow-up for patients with invasive melanoma:

Skin [self-examination](#) and [sun-smart behaviour](#) education

Routine skin checks by the patient's preferred health professional

Follow-up intervals:

Stage I: follow-up annually for 10 years

Stage IIA: every 6 months for 2 years, then annually for 8 years

Stage IIB and IIC: every 3 to 4 months for 2 years, every 6 months during year 3, then annually for 5 years.

Stage IIIA-C: every 3 months for 2 years, every 6 months during year 3, then annually for 5 years.

Individual patient needs should be considered before an appropriate follow-up is offered

Provide education and support to help the patient adjust to their illness.

The follow-up appointments may be undertaken by the patient's general practitioner and specialist.

Follow-up appointments may include:

Feel for the regional lymph nodes

General skin examination

Check of the scar where the primary melanoma was removed – including visual inspection and palpation

Full physical examination

Sequential TBP and dermoscopy monitoring of concerning melanocytic lesions in those with many melanocytic naevi or atypical melanocytic naevi.

In those with more advanced primary disease, follow-up may include:

Blood tests: such as LDH, S100B

Imaging: ultrasound, X-ray, CT, MRI, and FDG-PET scans.

Most tests are not worthwhile for patients with stage 1 or 2 melanoma unless there are signs or symptoms of disease recurrence or metastasis. No tests are necessary for healthy patients who have remained well for five years or longer after the removal of their melanoma.

How do you prevent melanoma?

Preventative measures involve addressing risk factors such as exposure to UV radiation, eg, wearing [protective clothing](#), using [sunscreen](#) (SPF 50), and avoiding [tanning beds](#). For more information, see [skin cancer](#).

What is the outcome of melanoma?

Melanoma in situ is cured by excision because it has no potential to spread around the body.

The risk of spread and ultimate death from invasive melanoma depends on several factors, including anatomic location, pathologic factors, and mutation status. However, the main factor is the Breslow thickness of the melanoma at the time it was surgically removed.

Metastases are rare for melanomas < 0.75 mm in size, and the risk associated with tumours 0.75–1 mm thick is about 5%. The risk steadily increases with thickness so that melanomas > 4 mm have a risk of metastasis of about 40%.

For images, see the following links:

[Superficial spreading melanoma images](#)

[Nodular melanoma images](#)

[Amelanotic melanoma images](#)

[Lentigo maligna melanoma images](#)

[Desmoplastic melanoma images](#)

[Melanoma in situ images](#)

[Metastatic melanoma images](#)



PREVENTION	SKIN CANCER INFORMATION	RISK FACTORS	EARLY DETECTION	TREATMENT & RESOURCES	BLOG	GET INVOLVED	ABOUT US
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Melanoma Overview

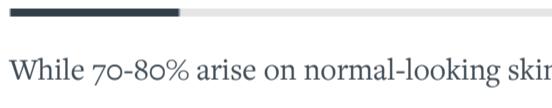
A Dangerous Skin Cancer

Malignant melanoma is a serious form of skin cancer that begins in cells known as melanocytes. It is less common than basal cell carcinoma (BCC) and squamous cell carcinoma (SCC), but it is more dangerous because of its ability to spread to other organs more rapidly if it is not treated at an early stage.

Learn more about melanoma types, risk factors, causes, warning signs and treatment.

MELANOMA FACT

Only 20-30% of melanomas are found in existing moles.



While 70-80% arise on normal-looking skin.



- [What is a melanocyte?](#)
- [What does melanoma look like?](#)
- [How dangerous is it?](#)
- [How widespread is it?](#)
- [What are the four main types of malignant melanoma?](#)
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Stages



Treatment



What is a melanocyte?

Melanocytes are skin cells found in the upper layer of skin. They produce a pigment known as melanin, which gives skin its color. There are two types of melanin: eumelanin and pheomelanin. When skin is exposed to ultraviolet (UV) radiation from the sun or tanning beds, it causes skin damage that triggers the melanocytes to produce more melanin, but only the eumelanin pigment attempts to protect the skin by causing the skin to darken or tan. Melanoma occurs when DNA damage from sunburns or tanning due to UV radiation triggers changes (mutations) in the melanocytes, resulting in uncontrolled cellular growth.

About Melanin

Naturally darker-skinned people have more eumelanin and naturally fair-skinned people have more pheomelanin. While eumelanin has the ability to protect the skin from sun damage, pheomelanin does not. That's why people with darker skin are at lower risk for developing skin cancer than fair-skinned people who, due to lack of eumelanin, are more susceptible to sun damage, burning and skin cancer. But, skin cancer can happen to anyone, regardless of skin tone.

What does melanoma look like?

Melanomas present in many different shapes, sizes and colors. That's why it's tricky to provide a comprehensive set of warning signs. Since detecting it early is so vital, please learn the common signs, symptoms and early detection strategies on our Warning Signs page. View more images on our Skin Cancer Pictures page.

How dangerous is melanoma?

Melanoma is usually curable when detected and treated early. Once it has spread deeper into the skin or other parts of the body, it becomes more difficult to treat and can be deadly.

- The estimated five-year survival rate for U.S. patients whose melanoma is detected early is about 99 percent.
- An estimated 8,430 people (5,470 men and 2,960 women) will die of melanoma in the U.S. in 2025.

How widespread is melanoma?

- An estimated 212,200 cases of melanoma will be diagnosed in the U.S. in 2025.
- 107,240 cases will be *in situ* (noninvasive), confined to the epidermis (the top layer of skin), and 104,960 cases will be invasive, penetrating the epidermis into the skin's second layer (the dermis).
- Of the invasive cases, 60,550 will occur in men and 44,410 will occur in women.

What are the four main types of melanoma of the skin?

Superficial spreading melanoma

What you should know: This is the most common type of melanoma.

How and where it grows: It can arise in an existing mole or appear as a new lesion. When it begins in a mole that is already on the skin, it tends to grow on the surface of the skin for some time before penetrating more deeply. While it can be found nearly anywhere on the body, it is most likely to appear on the torso in men, the legs in women and the upper back in both.

What it looks like: It may appear as a flat or slightly raised and discolored, asymmetrical patch with uneven borders. Colors include shades of tan, brown, black, red/pink, blue or white. It can also lack pigment and appear as a pink or skin-tone lesion (amelanotic).

Lentigo maligna

What you should know: This type often develops in older people. When this cancer becomes invasive or spreads beyond the original site, the disease is known as lentigo maligna melanoma.

How and where it grows: This is similar to the superficial spreading type, growing close to the skin surface at first. The tumor typically arises on sun-damaged skin on the face, ears, arms or upper torso.

What it looks like: It may look like a flat or slightly raised, blotchy patch with uneven borders. Color is usually blue-black, but can vary from tan to brown or dark brown.

Acral lentiginous melanoma

What you should know: This is the most common type of melanoma found in people of color, including individuals of African ancestry.

How and where it grows: It often appears in hard-to-spot places including under the nails (subungual) and on the soles of the feet or palms of the hands.

What it looks like: It may appear as a black or brown area on the skin.

Musician Bob Marley was diagnosed with acral lentiginous melanoma, which ultimately claimed his life at age 36. When a dark spot appeared under his toenail, Marley attributed it to a soccer injury. Eventually he was diagnosed with the disease but refused the recommended treatment, surgical removal of the toe. His melanoma spread to other areas of his body and tragically cut his life short.

Nodular melanoma

What you should know: This is the most aggressive type of melanoma. It accounts for 10 to 15 percent of all cases.

How and where it grows: The tumor grows deeper into the skin more rapidly than other types and is most frequently found on the torso, legs and arms, as well as the scalp in older men. It is usually invasive at the time it is first diagnosed.

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Milium

Author: Dr Monisha Gupta, Dermatologist, Sydney, Australia; Amanda Oakley FRACP, Dermatologist, Hamilton, New Zealand, 2009.

What is a milium?

A milium is a small [cyst](#) containing keratin (the skin protein); they are usually multiple and are then known as milia. These harmless cysts present as tiny pearly-white bumps just under the surface of the skin.

What are the clinical features of milia?

Milia are common in all ages and both sexes. They most often arise on the face and are particularly prominent on the [eyelids](#) and cheeks, but they may occur elsewhere.

There are various kinds of milia.

Neonatal milia

Affect 40–50% of newborn babies
Few to numerous lesions
Often seen on the nose, but may also arise inside the mouth on the gum margins (Bohn nodules) or palate (Epstein pearls) or more widely on the scalp, face and upper trunk
Heal spontaneously within a few weeks of birth.

Primary milia in children and adults

Found around [eyelids](#), cheeks, forehead and genitalia.
In young children, a row of milia may appear along the nasal crease.
May clear in a few weeks or persist for months or longer.

Juvenile milia

Associated with Rombo syndrome, [basal cell naevus syndrome](#), [Bazex-Dupre-Christol syndrome](#), [pachyonychia congenita](#), [Gardner syndrome](#) and other genetic disorders
May be congenital (present at birth) or appear later in life.

Milia in childhood



Neonatal centrofacial milia



Periocular milia



Milia associated with epidermolysis bullosa

Milia en plaque

Multiple milia appear on within an inflamed plaque up to several centimetres in diameter.

Usually found on an [eyelid](#), behind the ear, on a cheek or jaw.

Affect children and adults, especially middle-aged women.

Sometimes associated with another skin disease including [pseudoxanthoma elasticum](#), [discoid lupus erythematosus](#), [lichen planus](#).

Multiple eruptive milia

Crops of numerous milia appear over a few weeks to months.

Lesions may be asymptomatic or itchy.

Most often affect the face, upper arms and upper trunk.

Traumatic milia

Occur at the site of injury as the skin heals.

Arise from eccrine sweat ducts.

Examples include [thermal burns](#), [dermabrasion](#), [blistering rashes](#) such as [bullosum pemphigoid](#). Often seen on the back of hands and fingers in [porphyria cutanea tarda](#), and on the vulva in [lichen sclerosus](#) (see [Vulval cysts](#)).

A milia-like calcified nodule may develop after neonatal heel stick blood test.

Milia associated with drugs

May rarely follow the use of topical medication, such as phenols, [hydroquinone](#), [5-fluorouracil cream](#), and a [corticosteroid](#).



Milia-en-plaque



Periocular milia



Milia after pemphigoid blisters

[See more images of milia.](#)

How are milia diagnosed?

Milia have a characteristic appearance. However, on occasion, a [skin biopsy](#) may be performed. This shows a small epidermoid cyst coming from a vellus hair follicle.

Milia should be distinguished from other types of [cyst](#), [comedones](#), [xanthelasma](#) and [syringomas](#). Colloid milia are golden coloured bumps on cheeks and temples associated with excessive exposure to sunlight.

They should also be distinguished from milia-like cysts noted on [dermoscopy](#) in [seborrhoeic keratoses](#), [papillomatous moles](#) and some [basal cell carcinomas](#).

What is the treatment of milia?

Milia do not need to be treated unless they are a cause for concern for the patient. They often clear up by themselves within a few months. Where possible, further trauma should be minimised to reduce the development of new lesions.

The lesion may be de-roofed using a sterile needle or blade and the contents squeezed or pricked out.

They may be destroyed using diathermy and [curettage](#), or [cryotherapy](#).

For widespread lesions, [topical retinoids](#) may be helpful.

[Chemical peels](#), [dermabrasion](#) and [laser ablation](#) have been reported to be effective when used for very extensive milia.

Milia en plaque may improve with minocycline (a [tetracycline](#) antibiotic).

Melanoc

Author: Dr Amanda Oakley, Dermatolo

Credit: Many images have been

What is a melanocytic naevus?

A melanocytic naevus (American spelling 'nevus'), or mole, is a common benign skin lesion due to a

A melanocytic naevus can be present at birth (a [congenital melanocytic naevus](#)) or appear later (an acquired naevus). There are various kinds of congenital and acquired melanocytic naevi (American spelling 'nevi').

Who gets melanocytic naevi?

Almost everyone has at least one melanocytic naevus.

About 1% of individuals are born with one or more congenital melanocytic naevi. This is usually sporadic, with rare instances of familial congenital naevi.

Fair-skinned people tend to have more melanocytic naevi than darker skinned people.

Melanocytic naevi that appear during childhood (aged 2 to 10 years) tend to be the most prominent and persistent throughout life.

Melanocytic naevi that are acquired later in childhood or adult life often follow sun exposure and may fade away or involute later.

Most white-skinned New Zealanders have 20–50 melanocytic naevi.

What causes melanocytic naevi?

Although the exact reason for the local proliferation of naevus cells is unknown, it is clear that the number of melanocytic naevi a person has depends on genetic factors, on sun exposure, and on immune status.

People with many melanocytic naevi tend to have family members that also have many similar lesions.

Somatic mutations in RAS genes are associated with [congenital melanocytic naevi](#).

New melanocytic naevi may erupt following the use of BRAF inhibitor drugs ([vemurafenib](#), [dabrafenib](#)).

People living in Australia and New Zealand have many more naevi than their relatives residing in Northern Europe.

[Immunosuppressive treatment](#) leads to an increase in the numbers of naevi.

What are the clinical features of melanocytic naevi?

Melanocytic naevi vary widely in clinical, dermatoscopic and histological appearance.

They may arise on any part of the body.

They differ in appearance depending on the body site.

They may be flat or protruding.

They vary in colour from pink or flesh tones to dark brown, steel blue, or black.

Light-skinned individuals tend to have light-coloured naevi and dark-skinned individuals tend to have dark brown or black naevi.

Although mostly round or oval in shape, moles are sometimes unusual shapes.

They range in size from a couple of millimetres to several centimetres in diameter.

Classification of melanocytic naevi

Congenital melanocytic naevus

[Congenital melanocytic naevi](#) are classified according to their actual or predicted adult size in maximum dimension and on specific characteristics.

Small congenital naevus	Medium congenital naevus	Giant naevus	Hairy congenital naevus
Small congenital naevus is 1.5 cm diameter.	Medium congenital naevi are 1.5–19.9 cm diameter.	A large or giant congenital melanocytic naevus is ≥ 20 cm	Hairy congenital naevi grow thick long hairs.
			

Café au lait macule	Speckled lentiginous naevus	Naevus of Ota	Mongolian spot
Café au lait macule is a flat brown patch.	Speckled lentiginous naevus is a flat brown patch with darker spots.	Naevus of Ota is a bluish brown mark around forehead, eye and cheek.	Mongolian spot is a large bluish mark most often seen on buttocks of a newborn.
			

Café au lait macule

Speckled
lentiginous naevus

Naevus of Ota

Mongolian spot

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The pathological classification of melanocytic naevi relates to where naevus cells are found in the skin.

Junctional naevus

Dermal naevus

Compound naevus

Combined naevus

A junctional naevus has groups or nests of naevus cells at the junction of the epidermis and the dermis. A flat mole.	A dermal or intradermal naevus has naevus cell nests in the dermis. A papule, plaque or nodule with a pedunculated, papillomatous (Unna naevus) or smooth surface (Miescher naevus).	A compound naevus has nests of naevus cells at the epidermal-dermal junction as well as within the dermis. A central raised area surrounded by a flat patch.	A combined naevus has two distinct types of mole within the same lesion – usually blue naevus and compound naevus.
			

[See more images of moles.](#)

[Dermatoscopy](#) has given rise to a new classification based on the pigment patterns of melanocytic naevi. Examples include:

Reticular naevus

Globular naevus

Blue naevus

Starburst naevus

Reticular naevus reveals a lattice of intersecting brown lines.	Globular naevus characteristically shows aggregated brown oval structures.	The blue naevus is a uniform structureless lesion, steel blue in colour.	Starburst naevus reveals radial lines around the periphery of the lesion.
			

Site-related naevus: facial	Site-related naevus: acral	Naevus with special features	Unclassifiable naevus
Facial naevi reveal pseudonetwork around hair follicles	Acral naevi (these are on palms and soles) tend to be made up of parallel lines.	Naevi with special features include eczematised naevus (illustrated), irritated naevi and halo naevi .	The unclassifiable naevus doesn't have any of the other patterns.






Acquired melanocytic naevus

Ordinary moles that appear after birth may be referred to as acquired naevi. Acquired melanocytic naevi are given a variety of names and there is considerable overlap of descriptions.

Signature naevi are the predominant group of naevi in an individual with multiple moles.

Solid brown naevus	Solid pink naevus	Eclipse naevus	Cockade naevus
Solid brown naevi have uniform brown pigmentation.	Solid pink naevi are seen in fair-skinned individuals and lack melanin pigmentation.	Eclipse naevus has a ring, or segment of a ring, of darker pigment around a tan or pink centre. Often found in the scalp.	Cockade, or naevus en cocarde/cockade, has a central dark naevus surrounded by concentric circles of light and dark pigmentation like a rosette.






Naevus with perifollicular hypopigmentation	Fried-egg naevus	Lentiginous naevus	Naevus with eccentric pigmentation
<p>Naevi with perifollicular hypopigmentation have white spots around each hair. Easier to see by dermoscopy.</p>    	<p>Fried-egg naevus is a compound naevus with a flat rim of pigment around a bumpy central portion – the bump can be lighter or darker than the pigmented rim.</p>	<p>Lentiginous naevi are small, dark brown or black, flat lesions, often with a slightly paler rim – people with multiple lentiginous naevi have been said to have cheetah phenotype.</p>	<p>The Bologna sign refers to a harmless, small area of darker colour on one side of the naevus.</p>

Uncommon types of melanocytic naevi include:

Spitz naevus or epithelioid cell naevus: a pink (classic Spitz) or brown (pigmented Spitz) dome-shaped mole that arises in children and young adults.

Reed naevus: darkly pigmented type of Spitz naevus with starburst dermatoscopic pattern

Agminated naevi: a cluster of similar moles

Kissing naevus: adjacent melanocytic naevi on upper and lower eyelids, due to naevus formation prior to separation of eyelids *in utero*.

The term **atypical naevus** may be used in several ways.

A benign lesion that has some clinical or histopathological characteristics of **melanoma**

A melanocytic naevus with specific characteristics: large (> 5 mm); ill-defined or irregular borders; varying shades of colour; with flat and bumpy components.

Or, any funny-looking naevus; large, or different from the patient's other naevi.

Atypical naevi usually occur in fair-skinned individuals and are due to sun exposure. They may be solitary or numerous. Pathology is reported as dysplastic junctional or compound naevus and has specific histological features (the Clark naevus).

Common naevus	Naevus in dark skin	Atypical naevus	Dysplastic naevus
<p>A common naevus is a flat mole with a single uniform colour.</p>	<p>In dark skin, naevi are often black in colour.</p>	<p>People with multiple atypical naevi are at increased risk of melanoma (cancerous mole).</p>	<p>Dysplastic naevus describes an atypical mole that has specific histological criteria.</p>

Common naevus

Naevus in dark skin

Atypical naevus

Dysplastic naevus

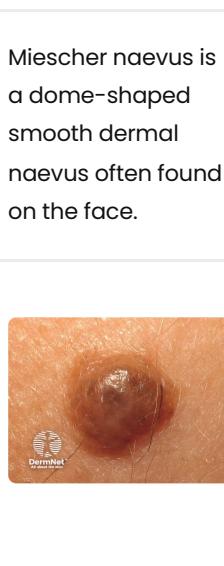
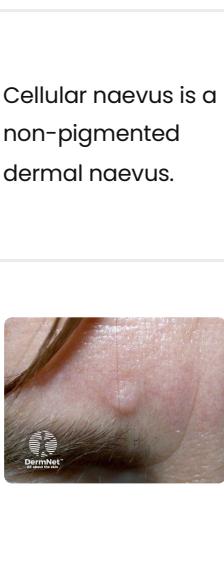
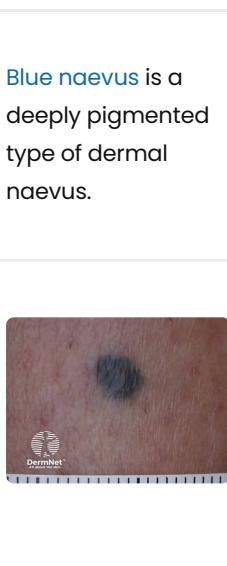


Blue naevus

Cellular naevus

Miescher naevus

Unna naevus

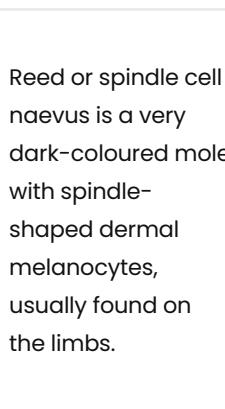
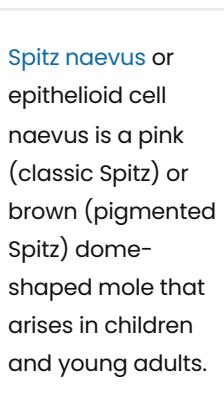
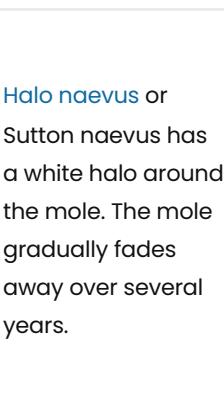


Meyerson naevus

Halo naevus

Spitz naevus

Reed naevus



Recurrent naevus	Agminated naevus	Acral naevus	Nail unit naevus
Recurrent naevus refers to the reappearance of pigment in a scar following surgical removal of a mole – this may have an odd shape.	An agminated naevus is a cluster of similar moles or freckles.	Acral naevus refers to one on the palm or sole.	Nail unit naevus causes a uniform longitudinal band of pigment on a nail.
			

[See more images of halo naevi](#)

[See more images of atypical naevi.](#)

What are the complications of melanocytic naevi?

People worry about their moles because they have heard about [melanoma](#), a malignant proliferation of melanocytes that is the most common reason for death from [skin cancer](#).

At first, melanoma may look similar to a harmless melanocytic naevus, but in time it becomes more disordered in structure and tends to enlarge.

People with a greater number of naevi have a higher risk of developing melanoma than those with few naevi, especially if they have over 100 of them.

Melanocytic naevi sometimes change for other reasons than melanoma, for example following sun exposure or during pregnancy. They can enlarge, regress or involute (disappear).

A [Meyerson naevus](#) is itchy and dry because it is surrounded by eczema.

A Sutton or [halo naevus](#) is surrounded by a white patch and fades away over several years

A recurrent naevus is one that appears in a scar following surgical removal of a melanocytic naevus – this may have an odd shape.

How is a melanocytic naevus diagnosed?

Melanocytic naevi are usually diagnosed clinically by their typical appearance. If there is any doubt about the diagnosis, an expert may be consulted in person or with the help of clinical and dermatoscopic images. This is especially important if:

A naevus changes size, shape, structure or colour

A new naevus develops in adult life (> 40 years)

It appears different from the person's other naevi (a so-called ugly duckling)

It has ABCD characteristics (Asymmetry, Border irregularity, Colour variation, Diameter > 6 mm)

It is bleeding, crusted or itchy.

Most skin lesions with these characteristics are actually harmless when evaluated by an expert using [dermatoscopy](#). Short-term digital dermatoscopic imaging may be used in equivocal flat lesions to check for change over time.

Naevi that remain suspicious for [melanoma](#) are excised for histopathology (diagnostic biopsy). A partial [biopsy](#) is not recommended, as it may miss an area of cancerous change.

What is the treatment for melanocytic naevus?

Most melanocytic naevi are harmless and can be safely left alone. They may be removed in the following circumstances:

To exclude cancer

If a naevus is a nuisance: perhaps irritated by clothing, comb or razor

Cosmetic reasons: the mole is unsightly.

Surgical techniques include:

[Excision biopsy](#) of a flat or suspicious melanocytic naevus

[Shave biopsy](#) of a protruding melanocytic naevus

[Electrosurgical](#) destruction

[Laser](#) to lessen pigment or remove coarse hair.

Can melanocytic naevi be prevented?

The number of melanocytic naevi can be minimised by strict protection from the sun, starting from birth. [Sunscreen](#) alone is not sufficient to prevent new naevi from appearing.

At any age, [sun protection](#) is important to reduce [skin ageing](#) and the risk of [skin cancer](#).

In New Zealand, the [SunSmart Sun Protection Alert](#) advises when protection is required.

[Cover up](#). Wear a hat, long sleeves and a long skirt or trousers. Choose fabrics designed for the sun (UPF 40+) when outdoors.

Apply [sunscreen](#) to areas you can't cover. Choose broad-spectrum high protection (SPF 50+) sunscreens, applied frequently to exposed areas.

What is the outlook for melanocytic naevi?

Most melanocytic naevi that appear in childhood remain forever. Teenagers and young adults tend to have the greatest number of naevi. There are fewer in later life because some of them slowly fade away.

To increase the chance of spotting [melanoma](#) early, recommend:

A [self-skin examination](#) monthly

A patient noticing a significant change in a mole or a new lesion should show this to their doctor or dermatologist

Regular skin examinations in patients with many naevi, [atypical naevi](#), or who have had a previous skin cancer

Total body photography and digital dermatoscopic imaging ([mole mapping](#)) for patients at high risk of melanoma, especially if they have many melanocytic naevi.

Nail psoriasis

Authors: Vanessa Ngan, Staff Writer, 2003; A/Prof Amanda Oakley, Dermatologist, Hamilton, New Zealand, February 2016; DermNet Update August 2021. Copy edited by Gus Mitchell

What is nail psoriasis?

Nail psoriasis, also known as psoriatic nail dystrophy, is due to [psoriasis](#) involving the nail matrix or nail bed, resulting in specific and non-specific clinical changes in the nail.



Nail psoriasis



Nail psoriasis



Nail psoriasis

Who gets nail psoriasis?

Nail psoriasis affects 90% of patients with [chronic plaque psoriasis](#) at some time in their life. It is more common in adults with a prevalence of up to 80%, compared to children in whom it has been reported in 7–13%. In the absence of skin or joint disease, psoriatic nail disease has been described in 5–10% of adults.

Psoriatic nail disease may be a risk factor for the development of [psoriatic arthritis](#) and is often associated with prolonged severe cutaneous psoriasis.

Nail psoriasis can affect all races and age groups, and both sexes, although a male predominance has been reported in one large case series.

What causes nail psoriasis?

Psoriasis is a multifactorial systemic disease including inflammation and epidermal hyperproliferation.

Nail psoriasis can involve the nail bed, nail matrix, hyponychium, and nail folds.

Theories include:

Activation of the antimicrobial peptide LL-37 by *Candida* and the cytokine overflow theory

Increased expression of interleukin(IL)-10 in the affected nail bed compared to downregulation of IL-10 in psoriatic skin lesions

Koebnerisation of psoriasis in [onychomycosis](#) or nail trauma.

What are the clinical features of nail psoriasis?

Fingernails and toenails can be affected by nail psoriasis.

Psoriatic nail dystrophy can cause tenderness and pain, altered sense of fine touch, and difficulty picking up or manipulating objects such as shoelaces or buttons.

Clinical signs of nail matrix involvement

Pitting

[Leukonychia](#)

Red spots in lunule

Onychorrhexis (longitudinal nail ridge, split, or fissure)

Beau lines (transverse lines and ridges)

Nail crumbling

Signs of psoriatic nail matrix involvement



Pitting and onychorrhexis



Leukonychia and Beau lines



Click to show sensitive image

Crumbling nails

Clinical signs of nail bed involvement

Oil-drop sign and salmon patch

Onycholysis – typically with a pink zone proximally

Subungual hyperkeratosis

Splinter haemorrhages under the distal third of the nail plate



Onycholysis and salmon patch



Subungual hyperkeratosis



Splinter haemorrhages

Other clinical signs of psoriatic nails

Paronychia due to periungual psoriasis

Acrodermatitis continua of Hallopeau

Twenty-nail dystrophy

Psoriatic nail dystrophy



Onycholysis and red dot in lunule



Periumgual psoriasis and paronychia



Trachyonychia

[see also [Nail psoriasis images](#)]

What are the complications of nail psoriasis?

Secondary onychomycosis in the damaged nail plate

Psychosocial effects impacting social relationships and work-related activities

Association with psoriatic arthritis and metabolic syndrome

How is nail psoriasis diagnosed?

Nail psoriasis is usually diagnosed clinically in a patient with psoriatic arthritis and/or cutaneous psoriasis.

The severity of nail psoriasis can be estimated using the Nail Psoriasis Severity Index (NAPSI) in which each nail is divided into quadrants and scored for clinical signs to come up with a numerical score.

Nail clippings for [fungal microscopy and culture](#) should be taken as onychomycosis may precede or complicate psoriatic nail dystrophy, and immunosuppressive medications may be used in treatment.

A proximal [nail matrix biopsy](#) is occasionally needed to confirm the diagnosis of nail psoriasis, particularly in the absence of signs of psoriasis elsewhere or where only a single nail is affected and a tumour cannot be excluded by other means. Biopsy can lead to permanent nail deformity.

What is the differential diagnosis for nail psoriasis?

[Onychomycosis](#)
[Nail trauma](#)
[Lichen planus of the nail](#)
[Parakeratosis pustulosa](#)

What is the treatment for nail psoriasis?

General measures

Minimise nail trauma, keep affected nails short
Treat associated onychomycosis first for at least three months

Specific measures

Topical treatments

[Topical steroids](#) – betamethasone dipropionate, clobetasol propionate

[Topical calcipotriol](#)

[Topical calcineurin inhibitors](#)

Systemic treatments

[Methotrexate](#)

[Acitretin](#)

[Biological agents for psoriasis](#) – infliximab, adalimumab, etanercept, ustekinumab

Novel small molecules – apremilast, tofacitinib [see Janus kinase inhibitors]

Nonpharmacological treatments

[Phototherapy](#)

[Lasers](#)

[Photodynamic therapy](#)

What is the outcome for nail psoriasis?

Nail psoriasis has a variable response to treatment. The visible response may take weeks or months due to slow growth of the nail plate, and relapses are common.

Psoriatic nail disease can fluctuate in severity over time and can resolve spontaneously.

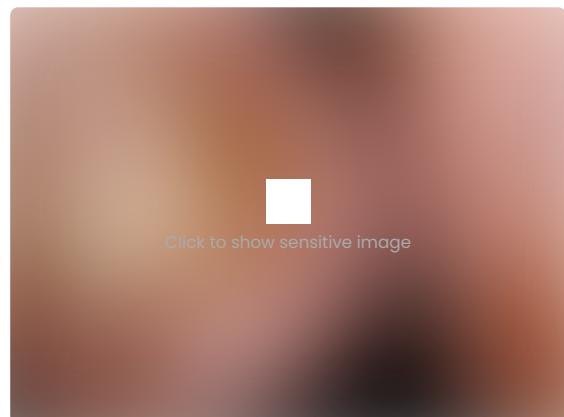
Paronychia

Author: Hon A/Prof Amanda Oakley, Dermatologist, Hamilton, New Zealand, 1997. Updated by Dr Jannet Gomez, August 2017.

What is paronychia?

Paronychia is inflammation of the skin around a finger or toenail. It can be acute (< 6 weeks) or chronic (persisting > 6 weeks).

Paronychia is also called whitlow. It may be associated with felon.



Who gets paronychia?

Acute paronychia can affect anyone. However, it is more likely to follow a break in the skin, especially between the proximal nail fold/cuticle and the nail plate. For example:

If the nail is bitten ([onychophagia](#)) or the nail-fold is habitually picked (eg, [habit-tic nail deformity](#))

In infants that suck their fingers or thumbs

Following manicuring

Ingrown toenails ([onychocryptosis](#))

On the application of sculptured or artificial fingernails

Treatment with **oral retinoid** that dries the skin (**acitretin, isotretinoin**)

Other drugs, including **epidermal growth factor receptor** and **BRAF** inhibitors (**vemurafenib, dabrafenib**)

Chronic paronychia mainly occurs in people with **hand dermatitis**, or who have constantly cold and wet hands, such as:

- Dairy farmers
- Fishermen
- Bartenders
- Cleaners
- Housewives
- People with poor circulation

Acute and chronic skin infections tend to be more frequent and aggressive in patients with **diabetes** or chronic debility, or that are **immune suppressed** by drugs or disease.

What causes paronychia?

Acute paronychia is usually due to bacterial infection with ***Staphylococcus aureus*** (which may be **multiresistant**), ***Streptococcus pyogenes***, **Pseudomonas**, or other bacterial pathogens. It can also be due to the cold sore virus, **Herpes simplex** (**herpetic whitlow**), and the yeast, ***Candida albicans***.

The cause or causes of chronic paronychia are not fully understood. In many cases, it is due to **dermatitis** of the nail fold. Often several different micro-organisms can be cultured, particularly ***Candida albicans*** and the Gram-negative bacilli, **pseudomonas**.

What are the clinical features of paronychia?

Acute paronychia

Acute paronychia develops rapidly over a few hours, and usually affects a single nail fold. Symptoms are pain, redness and swelling.

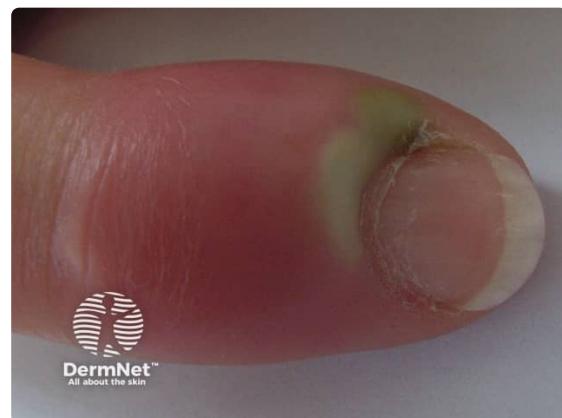
If herpes simplex is the cause (**herpetic whitlow**), multiple tender vesicles may be observed.

Sometimes yellow pus appears under the cuticle and can evolve to **abscess**. The nail plate may lift up (**onycholysis**). Acute paronychia due to ***S. pyogenes*** may be accompanied by fever, lymphangitis and tender lymphadenopathy.

Acute **candida** more commonly infects the proximal nail fold.



Paronychia and ingrown toenail in an athlete



Acute staphylococcal paronychia



Acute herpetic paronychia

Chronic paronychia

Chronic paronychia is a gradual process. It may start in one nail fold, particularly the proximal nail fold, but often spreads laterally and to several other fingers. Each affected nail fold is swollen and lifted off the nail plate. This allows the entry of organisms and irritants. The affected skin may be red and tender from time to time, and sometimes a little pus (white, yellow or green) can be expressed from under the cuticle.

The nail plate thickens and is distorted, often with transverse ridges.



Nailfold swelling



Nail dystrophy



Eczema

[See more images of paronychia ...](#)

What are the complications of paronychia?

Acute paronychia can spread to cause a serious hand infection ([cellulitis](#)) and may involve underlying tendons ([infectious tendonitis](#)).

The main complication of chronic paronychia is [nail dystrophy](#). It is often associated with distorted, ridged nail plates. They may become yellow or green/black and brittle. After recovery, it takes up to a year for the nails to grow back to normal.

How is paronychia diagnosed?

Paronychia is a clinical diagnosis, often supported by laboratory evidence of infection.

Gram stain microscopy may reveal bacteria

Potassium hydroxide microscopy may reveal fungi

Bacterial culture

Viral swabs

Tzanck smears

Nail clippings for culture ([mycology](#)).

What is the treatment for paronychia?

Acute paronychia

Soak affected digit in warm water, several times daily.

Topical [antiseptic](#) may be prescribed for a localised, minor infection.

Oral [antibiotics](#) may be necessary for severe or prolonged bacterial infection; often a [tetracycline](#), such as doxycycline, is prescribed.

Consider early treatment with [aciclovir](#) in case of severe [herpes simplex](#) infection ([herpetic whitlow](#)).

Surgical incision and drainage may be required for [abscess](#) followed by irrigation and packing with gauze.

Rarely, the nail must be removed to allow pus to drain.

Chronic paronychia

Attend to predisposing factors.

Keep the hands dry and warm.

Avoid wet work, or use totally waterproof gloves that are lined with cotton.

Keep fingernails scrupulously clean.

Wash after dirty work with soap and water, rinse off and dry carefully.

Apply [emollient](#) hand cream frequently – dimethicone barrier creams may help.

Treatment should focus on [dermatitis](#) and any microbes grown on culture [1].

[Topical corticosteroid](#) ointment is applied for 2–4 weeks and repeated for flares.

[Tacrolimus](#) ointment is an alternative when dermatitis is not responding to routine management [2].

[Intralesional steroid injections](#) are sometimes used in resistant cases.

[Antiseptics](#) or [antifungal lotions or solutions](#) may be applied for several months.

[Oral antifungal agent](#) ([itraconazole](#) or [fluconazole](#)), if [C. albicans](#) is confirmed.

Other management

Patients with diabetes and vascular disease with toenail paronychia infections should be examined for signs of [cellulitis](#).

Surgical excision of the proximal nail fold may be necessary.

Eponychial marsupialisation involves surgical removal of a narrow strip of skin next to the nail, to reduce the risk of infection [3].

Swiss roll technique has the advantage of retaining the nail plate and quicker recovery [4].

What is the outlook for paronychia?

Acute paronychia usually clears completely in a few days, and rarely recurs in healthy individuals.

Chronic paronychia may persist for months or longer and can recur in predisposed individuals.

Pseudofolliculitis barbae

Author(s): Hana Numan, Senior Medical Writer, DermNet Staff. Dr Jannet Gomez, PG Student in Clinical Dermatology, United Kingdom, 2016; A/Prof Amanda Oakley, Dermatologist, New Zealand, 1998. Copy edited by Gus Mitchell. February 2022

What is pseudofolliculitis barbae?

Pseudofolliculitis barbae (PFB) is a common inflammatory reaction of the hair follicle, most often on the face as a result of [shaving](#). Also known as “razor bumps” or “shaving bumps”, it can also occur on any site where hair is shaved or plucked, including the axilla, pubic area, and legs.

[Folliculitis barbae](#) presents similarly, but is due to infection. Folliculitis barbae and pseudofolliculitis barbae can coexist.



Pseudofolliculitis barbae



Pseudofolliculitis barbae



Ingrowing beard hair

Who gets pseudofolliculitis barbae?

Although PFB can occur in men of all races, it predominantly affects men of African ancestry (approximately 45–80%). This is likely due to a greater prevalence of tightly curled, coarse hair in the African population. A study demonstrated that the presence of a single nucleotide substitution in the hair follicle companion layer-specific keratin (K6hf) is an additional risk factor. It can also affect women of all races, especially occurring in the groin.

It is associated with improper shaving technique and is more common with blade razor users compared to electric shavers.

What causes pseudofolliculitis barbae?

Razor bumps are thought to be caused by intrafollicular or transfollicular penetration of tight curly hair, often of coarse nature. They may also occur in skin folds or scar tissue, allowing straight hair to re-enter.

This often occurs due to close shaving, as cut hair results in a sharp pointed end and may re-enter by either:

Piercing the skin surface (transfollicular)

Retract beneath the skin surface and pierce the follicular epithelium (intrafollicular).

This subsequently leads to a foreign body inflammatory reaction. The injured follicles are highly susceptible to infection, causing [folliculitis barbae](#).

What are the clinical features of pseudofolliculitis barbae?

An acne-like eruption presenting as ingrown hairs associated with flesh-coloured or red follicular papules.

Most often on the face and neck of men after shaving.

Under the jawline is typical, a site where the hair follicles grow in various directions.

PFB may also occur in any site where hair is shaved or plucked, including the axilla, pubic area, and legs.

May be itchy or tender.

Lesions may bleed when shaved.

May coexist with, and be aggravated by [eczema/dermatitis](#).

Once healed, [postinflammatory hyperpigmentation](#) and scarring may occur.

How do clinical features vary in differing types of skin?

Pseudofolliculitis barbae is more common in those with darkly pigmented skin. It is also more likely to occur in those with curly and coarse hair.

What are the complications of pseudofolliculitis barbae?

[Folliculitis barbae](#)

[Abscess](#)

[Postinflammatory hyperpigmentation](#)

[Scarring including keloid scarring](#)



Pseudofolliculitis barbae



Pseudofolliculitis



Rounded pigmented papules in the beard area in pseudofolliculitis barbae

How is pseudofolliculitis barbae diagnosed?

Pseudofolliculitis barbae is a clinical diagnosis. Dermoscopy may be used as an aid to visualise ingrown hairs and exclude differential diagnoses.

What is the differential diagnosis for pseudofolliculitis barbae?

- [Folliculitis barbae](#)
- [Tinea barbae](#)
- [Acne vulgaris](#)
- [Folliculitis keloidalis \(keloidalis nuchae\)](#)
- [Cutaneous sarcoidosis](#)

What is the treatment and prevention for pseudofolliculitis barbae?

The safest and most definitive cure is to discontinue shaving activity, thus patients should have the cause of PFB clearly explained to them. This may be unsuitable for many (eg, work, cultural, or personal reasons). If shaving is discontinued, it may take several weeks for the inflammatory response to cease (whilst hairs regrow).

General measures

Ensure proper [shaving technique](#) or trialling different techniques to find which suits best. It may also be beneficial to:

- Shave in the direction of the follicle, not against it
- Do not stretch the skin
- Use short strokes
- Use sharp blades
- Avoid shaving in the same area twice (leave approx. 1 mm of stubble)
- Shave with the non-shaving hand behind the back (to reduce the temptation to make the skin taught, and thereby producing an extra close shave).
- Trial switching from a blade razor to an electric shaver.
- Reduce shaving activity e.g. only every other day.
- Consider alternative [hair removal techniques](#) eg, [laser therapy](#).
- Ensure skin is well [moisturised](#) — preparations containing [glycolic acid](#) can exfoliate the skin surface and reduce the risk of new inflamed spots.

Specific measures

A [topical steroid cream](#) and/or topical antimicrobials (e.g., [clindamycin](#), [erythromycin](#)) can reduce mild inflammation and itching.

Topical acne treatments such as [benzoyl peroxide](#) and [tretinoin](#) are used to suppress follicular hyperkeratosis.

Oral antibiotics such as [tetracyclines](#) may reduce inflammation.

10 minute compresses of warm water, saline, or burrows solution (aqueous solution of aluminum acetate) can help soothe the area and remove crusts.

Chemical depilatories can help prevent PFB such as barium sulfide paste and calcium thioglycolate; these can be irritating.

[Photodynamic therapy](#).

[Laser epilation \(Nd YAG\)](#).

What is the outcome for pseudofolliculitis barbae?

Pseudofolliculitis barbae subsides approximately 4–6 weeks following cessation of the causative hair removal technique (most often shaving). The likelihood of experiencing further razor bumps can be reduced by trialling a different hair removal technique.

If no change is made, the condition will likely persist. Complications such as infection or scarring may arise and further treatment may be required.



Menu



Search



Symptoms often start between ages 15 and 25 but can start at any age. Men, women, and children of all skin colors can get psoriasis.

Common Psoriasis Locations

- [Elbows](#)
- [Face](#)
- [Feet](#)
- [Hands](#)
- [Knees](#)
- [Nails](#)
- [Scalp](#)
- [Skin folds \(including genitals\)](#)

Psoriasis can be anywhere on the body. Plaques can be a few small patches or can affect large areas. It's possible to have psoriasis plaques and scales in more than one location on the body at a time. Psoriasis on certain locations, called [high-impact sites](#), can have an increased negative impact on quality of life, regardless of the total area affected by psoriasis.

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Psoriasis

Last reviewed: June 2023

Author(s): Dr Chelsea Jones, Newcastle, Australia; Dr Monisha Gupta, Dermatologist, Australia (2020); updated by Dr Salim Uddin, Medical Registrar, New Zealand; A/Prof Amanda Oakley, Dermatologist, Hamilton, New Zealand (2023).

Reviewing dermatologist: Dr Ian Coulson

Edited by the DermNet content department

What is psoriasis?

Psoriasis is a chronic inflammatory skin condition characterised by clearly defined, red and scaly plaques. It is classified into several types.

Who gets psoriasis?

Psoriasis affects 2–4% of males and females. It can start at any age including [childhood](#), with onset peaks at 15–25 years and 50–60 years. It tends to persist lifelong, fluctuating in extent and severity. It is particularly common in Caucasians but may affect people of any ethnicity. About one-third of patients with psoriasis have family members with psoriasis.

What causes psoriasis?

Psoriasis is multifactorial. It is classified as an immune-mediated genetic skin disease.

This involves a complex interaction between the innate and adaptive immune systems.

Genome-wide association studies report that the histocompatibility complex HLA-C*06:02 (previously known as HLA-Cw6) is associated with early-onset psoriasis and [guttate psoriasis](#). This major histocompatibility complex is not associated with [psoriatic arthritis](#), [nail psoriasis](#), or late-onset psoriasis.

Immune factors and inflammatory cytokines (messenger proteins) such as IL1 β and TNF α , IL-23, and IL-17 are responsible for the clinical features of psoriasis. These have therefore become targets for biological drugs and have led to success in drug management.

What are the clinical features of psoriasis?

Psoriasis usually presents with symmetrically distributed, red, scaly plaques with well-defined edges. The scale is typically silvery white, except in skin folds where the plaques often appear shiny with a moist peeling surface. The most common sites are the [scalp](#), elbows, and knees, but any part of the skin can be involved. The plaques are usually very persistent without treatment.

[Itch](#) is mostly mild but may be severe in some patients, leading to scratching and lichenification characterised by thickened leathery skin and increased skin markings. Painful skin cracks or fissures may occur, particularly on the palms and soles.

Psoriasis can demonstrate the [Koebner phenomenon](#). This involves the generation of new lesions on the skin that has been damaged or irritated such as by injury, burns etc.

When psoriatic plaques clear up, they may leave brown or pale marks (postinflammatory hypo- or [hyperpigmentation](#)) that can be expected to fade over several months.

Auspitz sign refers to pinpoint bleeding upon removal of the scaly layer in plaque psoriasis. It is related to the dilated dermal capillaries involved in the histological pathogenesis of chronic psoriasis.

How is psoriasis classified?

Certain features of psoriasis can be categorised to help determine appropriate investigations and treatment pathways - overlap may occur. These include:

- Early age of onset <35 years (75%) vs late age of onset >50 years
- Acute eg [guttate psoriasis](#) vs [chronic plaque psoriasis](#)
- Localised eg, [scalp](#), [palmoplantar](#) psoriasis vs generalised psoriasis
- Small plaques (<3 cm) vs large plaques (>3 cm)
- Thin plaques vs thick plaques
- [Nail involvement](#) vs no nail involvement

Types of psoriasis

Guttate psoriasis

Post-streptococcal [acute guttate psoriasis](#)

- Widespread small plaques
- Often resolves after several months



Small plaques of psoriasis appear almost like rain drops splashed over the skin



Close up of guttate psoriasis



Post-inflammatory hypopigmentation as guttate psoriasis is resolving

Small plaque psoriasis

Often late age of onset

Plaques <3 cm

Chronic plaque psoriasis

Persistent and treatment-resistant

Plaques >3 cm

Most often affects elbows, knees, and lower back

Ranges from mild to very extensive



Well demarcated plaques with silvery scale in chronic plaque psoriasis



Thick silvery scale over the elbow



Close up of well demarcated plaques with silvery scale in chronic plaque psoriasis

Unstable plaque psoriasis

The rapid extension of existing or new plaques

Koebner phenomenon: new plaques at sites of skin injury

Induced by infection, **stress**, drugs, or drug withdrawal

Flexural psoriasis (inverse psoriasis)

Affects body folds and **genitals**

Smooth, well-defined patches

Colonised by **candida** yeasts



Well demarcated salmon pink erythema which extends to the apex of the skin fold due to flexural psoriasis



Well demarcated salmon pink erythema which extends to the apex of the skin fold and into the natal cleft due to flexural psoriasis



Axillary psoriasis

Scalp psoriasis

Often the first or only site of psoriasis



Well demarcated plaques of scalp psoriasis



Scalp psoriasis often extends beyond the hair line



Adherent scale in scalp psoriasis

Sebopsoriasis

Overlap of [seborrhoeic dermatitis](#) and psoriasis

Affects the scalp, face, ears and chest

Colonised by [malassezia](#)



Sebopsoriasis of ear



Sebopsoriasis of chest



Sebopsoriasis affecting the retroauricular skin

Palmoplantar psoriasis

Affecting the palms and/or soles

Keratoderma

Painful fissuring



Hyperkeratosis of the soles and heels due to psoriasis – painful fissuring is often a problem



Palmar plaque psoriasis



Hyperkeratosis of the soles and heels due to psoriasis

Nail psoriasis

Pitting, [onycholysis](#), yellowing, and ridging

Associated with inflammatory arthritis



Irregular pits and onycholysis of a finger nail – note the yellow band proximal to the onycholysis



Onycholysis, proximal yellow band and nail plate splitting due to nail unit psoriasis



Onycholysis (separation of the plate from the bed) due to psoriasis

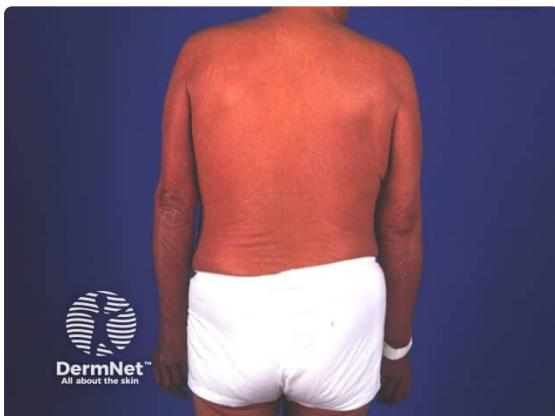
Erythrodermic psoriasis

Erythrodermic psoriasis is rare.

May or may not be preceded by another form of psoriasis

Acute and chronic forms

May result in systemic illness with temperature dysregulation, electrolyte imbalance, or cardiac failure



Confluent skin redness and scaling – the patient was shivery and feels unwell



Confluent redness affecting more than 80% of the body – erythrodermic psoriasis



Close up of the confluent redness and scale in erythrodermic psoriasis

There is some controversy as to whether [generalised pustulosis](#) and [localised palmoplantar pustulosis](#) are classified as being within the psoriasis spectrum.

How do clinical features vary in differing types of skin?

Plaque psoriasis is the most common type of psoriasis in all racial groups. Non-Caucasians tend to have more extensive skin involvement than Caucasians. Asian populations are reported to have the highest percentage of body surface area involvement.

In skin of colour, the plaques are typically thicker with a more pronounced silver scale and itch. The pinkness of early patches may be more difficult to appreciate.

Thick plaques may appear violet or dark in colour. Plaque psoriasis is more likely to result in **postinflammatory hyperpigmentation** or hypopigmentation in the skin of colour, which further impacts the quality of life even after disease clearance.

Other types of psoriasis show variable rates in different skin types. **Palmoplantar psoriasis** is reported to be more common in the Indian population. Non-Caucasians are more likely to present with pustular and **erythrodermic psoriasis** than Caucasians, whereas **flexural psoriasis** is said to occur at a lower rate in skin of colour.



Chronic plaque psoriasis in skin of colour



Chronic plaque psoriasis in skin of colour



Small plaque psoriasis with prominent scale in skin of colour

Factors that aggravate psoriasis

Streptococcal tonsillitis ('Strep throat') and other infections

Injuries such as cuts, abrasions, or sunburn (**koebnerised psoriasis**)

Sun exposure in ~10% of psoriasis patients (sun exposure is more often beneficial)

Dry skin

Obesity, smoking, or excessive alcohol

Medications such as **lithium, beta-blockers, antimalarials, nonsteroidal anti-inflammatories, terbinafine, immunotherapy**, and others

Stopping **oral steroids** or strong **topical corticosteroids**, often referred to as steroid withdrawal rebound

Other environmental factors such as a **stressful event**.

Health conditions associated with psoriasis

Patients with psoriasis are more likely than others to have associated health conditions such as are listed here.

Inflammatory “[psoriatic arthritis](#)” (an autoimmune disease) and [spondyloarthropathy](#) (seen in up to 40% of patients with early-onset [chronic plaque psoriasis](#)).

[Inflammatory bowel disease](#) ([Crohn disease](#) and ulcerative colitis).

[Uveitis](#) (a form of inflammation of the eye).

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[Localised palmoplantar pustulosis](#), [generalised pustulosis](#), and [acute generalised exanthematous pustulosis](#).

Non-alcoholic fatty liver disease (see [Liver problems and psoriasis](#)).

Psoriasis and metabolic syndrome

Metabolic syndrome refers to the combination of obesity, hypertension, dyslipidaemia, and insulin resistance.

It is present in many patients with psoriasis, especially those with severe [chronic plaque psoriasis](#).

The link between psoriasis, obesity, and type 2 [diabetes mellitus](#) is independent of age, sex, and smoking history.

The link is thought to be due to genetic factors and the presence of chronic inflammation.

The interleukin (IL)-23/Th 17 axis drives both psoriatic skin inflammation and atherosclerosis; psoriasis is an independent risk factor for cardiovascular disease.

Lifestyle modifications such as weight loss, lipid profile reduction, and tight control of glucose levels reduce the mortality from cardiovascular events in patients with psoriasis.

How is psoriasis diagnosed?

Psoriasis is diagnosed by its clinical features. If necessary, diagnosis is supported by typical [skin biopsy](#) findings.

Assessment of psoriasis

Medical assessment entails a careful history, examination, questioning about the effect of psoriasis on daily life, and evaluation of comorbid factors.

Validated tools used to evaluate psoriasis include:

[Psoriasis Area and Severity Index \(PASI\)](#)

[Self-Administered Psoriasis Area and Severity Index \(SAPASI\)](#)

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[SKINDEX-16](#)

The severity of psoriasis is classified as mild in 60% of patients, moderate in 30% and severe in 10%.

Evaluation of comorbidities may include:

Psoriatic Arthritis Screening Evaluation (PASE) or Psoriasis Epidemiology Screening Tool (PEST)
Body mass index (BMI) and waist circumference
Blood pressure
Electrocardiogram (ECG)
Blood sugar/glucose level and glycated haemoglobin (HbA1c)
Lipid profile, liver function, and uric acid level.

Treatment of psoriasis

General advice

Patients with psoriasis should be well-informed about their skin condition and its treatment.
Recommendations include:

[Smoking cessation](#)
[Safe limits for alcohol consumption](#)
[Maintaining optimal weight.](#)

Topical therapy

Mild psoriasis is generally treated with topical agents alone. The selected treatment depends on the body site and the extent and severity of psoriasis, and may include:

[Emollients and moisturisers](#)
[Coal tar preparations](#)
[Dithranol](#)
[Salicylic acid](#)
[Vitamin D analogue \(eg, calcipotriol\)](#)
[Topical corticosteroids](#)
[Combination calcipotriol/betamethasone dipropionate ointment/gel or foam](#)
[Calcineurin inhibitors \(tacrolimus, pimecrolimus\)](#)

Phototherapy

Most psoriasis centres offer [phototherapy](#) (light therapy) with [ultraviolet \(UV\) radiation](#), often in combination with topical or systemic agents, including:

[UVB phototherapy](#)
[PUVA \(photochemotherapy\)](#)
[Targeted phototherapy](#)
[Excimer laser.](#)

Systemic therapy

Moderate to severe psoriasis warrants treatment with a systemic agent and/or [phototherapy](#). The most common treatments are:

[Methotrexate](#)
[Ciclosporin](#)
[Acitretin.](#)

Other medicines occasionally used for psoriasis include:

Apremilast
Hydroxyurea
Dimethyl fumarate.

Systemic corticosteroids are best avoided due to the risk of severe withdrawal flare of psoriasis and adverse effects.

Biologic therapy

Biologic or **biological therapy** is reserved for severe psoriasis and **psoriatic arthritis** that have failed to respond to conventional systemic therapy. The use of biologics has increased with the development of novel therapies targeting key inflammatory pathways such as TNF-alpha and IL-17.

Biologics are costly and may result in side effects. They should be initiated by specialists familiar with their use.

TNF-alpha inhibitors:

Adalimumab
Etanercept
Infliximab
Certolizumab pegol.

Interleukin 17 (IL-17) inhibitors:

Ixekizumab
Secukinumab
Brodalumab
Bimekizumab

Interleukin 23 (IL-23) inhibitors:

Ustekinumab
Guselkumab
Tildrakizumab
Risankizumab

Adalimumab, etanercept, and ustekinumab have been used to treat severe **psoriasis in children**.

Newer agents:

Tapinarof 1% cream is an aryl hydrocarbon receptor agonist that downregulates TH2 cells, IL-17 activation, and disease severity. Tapinarof is undergoing Phase III trials for treatment in the paediatric population.

Small molecules: **Deucravacitinib**, brexpocitinib, and rapsacitinib are tyrosine kinase 2 (TYK2) inhibitors (see **Janus kinase inhibitors**).

Navigating Psoriasis: Clinical Perspectives on Features, Ma...



Psoriasis

Last reviewed: June 2023

Author(s): Dr Chelsea Jones, Newcastle, Australia; Dr Monisha Gupta, Dermatologist, Australia (2020); updated by Dr Salim Uddin, Medical Registrar, New Zealand; A/Prof Amanda Oakley, Dermatologist, Hamilton, New Zealand (2023).

Reviewing dermatologist: Dr Ian Coulson

Edited by the DermNet content department

What is psoriasis?

Psoriasis is a chronic inflammatory skin condition characterised by clearly defined, red and scaly plaques. It is classified into several types.

Who gets psoriasis?

Psoriasis affects 2–4% of males and females. It can start at any age including [childhood](#), with onset peaks at 15–25 years and 50–60 years. It tends to persist lifelong, fluctuating in extent and severity. It is particularly common in Caucasians but may affect people of any ethnicity. About one-third of patients with psoriasis have family members with psoriasis.

What causes psoriasis?

Psoriasis is multifactorial. It is classified as an immune-mediated genetic skin disease.

This involves a complex interaction between the innate and adaptive immune systems.

Genome-wide association studies report that the histocompatibility complex HLA-C*06:02 (previously known as HLA-Cw6) is associated with early-onset psoriasis and [guttate psoriasis](#). This major histocompatibility complex is not associated with [psoriatic arthritis](#), [nail psoriasis](#), or late-onset psoriasis.

Immune factors and inflammatory cytokines (messenger proteins) such as IL1 β and TNF α , IL-23, and IL-17 are responsible for the clinical features of psoriasis. These have therefore become targets for biological drugs and have led to success in drug management.

What are the clinical features of psoriasis?

Psoriasis usually presents with symmetrically distributed, red, scaly plaques with well-defined edges. The scale is typically silvery white, except in skin folds where the plaques often appear shiny with a moist peeling surface. The most common sites are the [scalp](#), elbows, and knees, but any part of the skin can be involved. The plaques are usually very persistent without treatment.

[Itch](#) is mostly mild but may be severe in some patients, leading to scratching and lichenification characterised by thickened leathery skin and increased skin markings. Painful skin cracks or fissures may occur, particularly on the palms and soles.

Psoriasis can demonstrate the [Koebner phenomenon](#). This involves the generation of new lesions on the skin that has been damaged or irritated such as by injury, burns etc.

When psoriatic plaques clear up, they may leave brown or pale marks (postinflammatory hypo- or [hyperpigmentation](#)) that can be expected to fade over several months.

Auspitz sign refers to pinpoint bleeding upon removal of the scaly layer in plaque psoriasis. It is related to the dilated dermal capillaries involved in the histological pathogenesis of chronic psoriasis.

How is psoriasis classified?

Certain features of psoriasis can be categorised to help determine appropriate investigations and treatment pathways - overlap may occur. These include:

- Early age of onset <35 years (75%) vs late age of onset >50 years
- Acute eg [guttate psoriasis](#) vs [chronic plaque psoriasis](#)
- Localised eg, [scalp](#), [palmoplantar](#) psoriasis vs generalised psoriasis
- Small plaques (<3 cm) vs large plaques (>3 cm)
- Thin plaques vs thick plaques
- [Nail involvement](#) vs no nail involvement

Types of psoriasis

Guttate psoriasis

Post-streptococcal [acute guttate psoriasis](#)

- Widespread small plaques
- Often resolves after several months



Small plaques of psoriasis appear almost like rain drops splashed over the skin



Close up of guttate psoriasis



Post-inflammatory hypopigmentation as guttate psoriasis is resolving

Small plaque psoriasis

Often late age of onset

Plaques <3 cm

Chronic plaque psoriasis

Persistent and treatment-resistant

Plaques >3 cm

Most often affects elbows, knees, and lower back

Ranges from mild to very extensive



Well demarcated plaques with silvery scale in chronic plaque psoriasis



Thick silvery scale over the elbow



Close up of well demarcated plaques with silvery scale in chronic plaque psoriasis

Unstable plaque psoriasis

The rapid extension of existing or new plaques

Koebner phenomenon: new plaques at sites of skin injury

Induced by infection, **stress**, drugs, or drug withdrawal

Flexural psoriasis (inverse psoriasis)

Affects body folds and **genitals**

Smooth, well-defined patches

Colonised by **candida** yeasts



© Dr Ph Abimelec

Well demarcated salmon pink erythema which extends to the apex of the skin fold due to flexural psoriasis



DermNet™
All about the skin

Well demarcated salmon pink erythema which extends to the apex of the skin fold and into the natal cleft due to flexural psoriasis



Axillary psoriasis

Scalp psoriasis

Often the first or only site of psoriasis



Well demarcated plaques of scalp psoriasis



Scalp psoriasis often extends beyond the hair line



Adherent scale in scalp psoriasis

Sebopsoriasis

Overlap of [seborrhoeic dermatitis](#) and psoriasis

Affects the scalp, face, ears and chest

Colonised by [malassezia](#)



Sebopsoriasis of ear



Sebopsoriasis of chest



Sebopsoriasis affecting the retroauricular skin

Palmoplantar psoriasis

Affecting the palms and/or soles

Keratoderma

Painful fissuring



Hyperkeratosis of the soles and heels due to psoriasis – painful fissuring is often a problem



Palmar plaque psoriasis



Hyperkeratosis of the soles and heels due to psoriasis

Nail psoriasis

Pitting, [onycholysis](#), yellowing, and ridging

Associated with inflammatory arthritis



Irregular pits and onycholysis of a finger nail – note the yellow band proximal to the onycholysis



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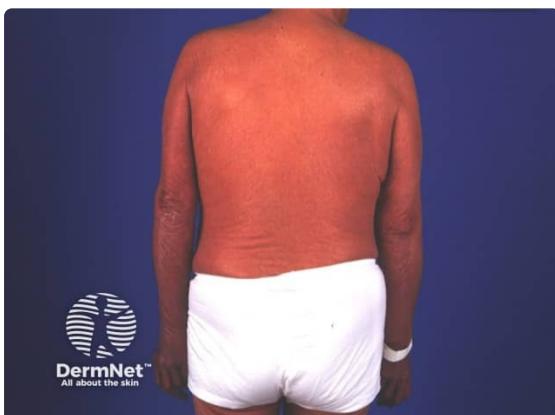
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Rheumatic fever

Author: Marie Hartley, Staff Writer, 2010. Latest update by Dr Jannet Gomez, Postgraduate Student in Clinical Dermatology, Queen Mary University London, United Kingdom, July 2016.

What is acute rheumatic fever?

Acute rheumatic fever (ARF) is caused by a reaction to a [bacterial infection](#) with particular strains of group A [streptococcus](#). It has long been thought that ARF only follows streptococcal pharyngitis (sore throat), however recent studies from Aboriginal populations in Australia have suggested streptococcal skin infection may precede some cases of ARF. Those who have experienced one episode of ARF are more likely to suffer recurrent attacks with subsequent group A streptococcal infections.

The skin sign of acute rheumatic fever is erythema marginatum.

Who gets rheumatic fever?

ARF usually affects children aged 5–15 years. Most cases of ARF currently occur in developing countries. Worldwide there is an estimated 470 000 new cases of ARF annually (60% of whom eventually develop rheumatic heart disease). In most developed countries ARF is now rare, with a few notable exceptions; the highest documented rates of ARF in the world are in Maori and Pacific people in New Zealand, Aboriginal Australians, and those in Pacific Island nations.

Rheumatic fever is associated with poverty, overcrowding and poor sanitation facilities. It is suspected that there are genes that make some families more susceptible to the disease.

What are the clinical features of acute rheumatic fever?

Symptoms of ARF generally develop several weeks after an episode of streptococcal pharyngitis. However, many patients do not recall having a sore throat. Non-specific symptoms include:

- Fever
- Abdominal pain
- Muscle aches

ARF causes a variety of more specific clinical features:

- Polyarthritis (multiple inflamed joints) – Most often ankles, knees, elbows, wrists. Arthritis can migrate from one joint to another.
- Carditis (inflammation of the heart) – This involves the heart valves, heart muscle, and membrane surrounding the heart.
- Sydenham chorea – This is a disorder of the nervous system characterised by personality changes, muscle weakness, and involuntary movements.

Skin signs in ARF may include:

Erythema marginatum rheumaticum – This is a characteristic type of [annular erythema](#) that occurs in about 10% of first attacks of ARF in children; it is very rare in adults. The rash can be difficult to detect in dark-skinned people. When present, it is found on the trunk and upper arms and legs, but almost never on the face, palms or soles. The rash appears as pink or red macules (flat spots) or papules (small lumps), which spread outwards in a circular shape. As the lesions advance, the edges become raised and red, and the centre clears. The lesions are not itchy or painful, and sometimes go unnoticed by the patient. The lesions can fade and reappear within hours, reappearing in hot conditions. Erythema marginatum may persist intermittently for weeks to months, even after successful treatment of ARF.

Subcutaneous nodules (small lumps under the skin) – These are uncommon, occurring in less than 2% of patients with ARF. The painless nodules are found over joints (such as the elbows, knees, ankles, and knuckles), the back of the scalp, and the vertebrae (backbone). The nodules are firm, round, mobile, and range from 0.5–2 cm in size. The nodules are usually only found when severe carditis is present. They usually resolve within one month but may persist for longer.



Erythema marginatum



Erythema marginatum



Erythema marginatum

What are the complications of acute rheumatic fever?

The most severe complication of recurrent ARF is permanent damage to heart valves, known as rheumatic heart disease. The disease can result in permanent damage to:

Heart valves, particularly the mitral valve and aortic valve, which can lead to valvular stenosis and/or regurgitation.

Heart muscles, reducing pumping action ie causing heart failure.

The membrane around the heart, causing pericarditis.

Irregular heart rhythms, such as atrial fibrillation.

How is acute rheumatic fever diagnosed?

The diagnosis of ARF is challenging, as there are no clinical features or diagnostic tests available to confirm or rule out this condition. Instead, the diagnosis is made using the Jones criteria. These criteria require evidence of a preceding group A streptococcus infection, and various combinations of the characteristic features above and other non-specific clinical features. A detailed explanation of these criteria (and modifications for the New Zealand environment) can be found on the [National Heart Foundation of New Zealand website](#).

Tests to confirm evidence of a group A streptococcal infection include:

Blood tests to look for elevated or rising antibodies to group A streptococcus. The most commonly used tests are the plasma antistreptolysin O and the antideoxyribonuclease B titres.

Culture of throat swabs and rapid antigen tests for group A streptococcus are less accurate.

Other tests used in the assessment of a patient with suspected ARF include:

Blood tests – markers of inflammation, such as ESR or CRP, may be raised

Electrocardiogram and echocardiogram to identify heart involvement

Doppler and colour flow mapping to detect minor valvular defects not evident clinically

What is the treatment for rheumatic fever?

Following a diagnosis of rheumatic fever, it is standard practice to treat the group A streptococcal infection that led to the disease with oral [penicillin](#) (although this practice has not been proven to alter long-term outcomes).

Following the initial attack, patients are treated with continuous penicillin to prevent further streptococcal colonisation or infection and additional damage to the heart. Continuous penicillin is also recommended for people with established rheumatic heart disease. Continuous penicillin is generally given by injection every four weeks for a minimum of 10 years. Some patients, such as those with severe carditis, may require lifelong treatment.

Aspirin or naproxen are added to reduce inflammation, fever and pain. In extreme cases, a corticosteroid such as prednisone may be added.

Patients with rheumatic heart disease may occasionally require heart surgery to repair or replace damaged heart valves.

Can rheumatic fever be prevented?

ARF can be prevented with timely treatment of group A streptococcal pharyngitis, particularly in people aged 5–15 years. People with sore throat and fever should see the doctor for advice. Vaccines are currently in development and are eagerly awaited.

Rosacea

Author: Dr Olivia Kuo, Addenbrookes Hospital, Cambridge, United Kingdom, March 2022; minor update September 2024.

Previous contributors: A/Prof Amanda Oakley, Dermatologist; Vanessa Ngan, Staff Writer, New Zealand, 2014. Copy edited by Gus Mitchell.

What is rosacea?

Rosacea is a chronic inflammatory skin condition predominantly affecting the central face and most often starts between the age of 30–60 years.

Rosacea is common and is characterised by persistent facial redness. It typically has a relapsing and remitting course, with symptoms controlled by lifestyle measures, general skin care, medications, and procedural interventions.



Papulopustular rosacea on the cheeks



Erythematotelangiectatic and papulopustular rosacea on the cheeks



Papular rosacea on the cheeks



Telangiectatic vessels in a rhinophyma shown on dermoscopy



Rhinophyma and papular rosacea on the chin



Papulopustular and ocular rosacea

[Click here for more images of rosacea](#)

Who gets rosacea?

Rosacea is estimated to affect around 5% of adults worldwide. Although rosacea is often thought to affect women more than men, studies have revealed an approximately equal gender distribution.

Rosacea typically presents after the age of 30 and becomes more prevalent with age. However, it can occur at any age and occasionally presents in children. Although rosacea can affect anyone, it is more common in those with fair skin, blue eyes, and those of Celtic or North European descent. It may be more difficult and under-recognised in patients with skin of colour.

Rosacea has been associated with depression, hypertension, cardiovascular diseases, anxiety disorder, dyslipidemia, diabetes mellitus, migraine, rheumatoid arthritis, *Helicobacter pylori* infection, ulcerative colitis, and dementia.

What causes rosacea?

The pathogenesis of rosacea is thought to be multifactorial and includes:

Genetic susceptibility

Association with single nucleotide polymorphisms related to the class II major histocompatibility complex.

Altered microbiome of the skin and gut

Bacterial overgrowth of the small intestine, *Helicobacter pylori* infection, and increased density of *Demodex folliculorum* and *Staphylococcus epidermidis* on the skin may play a role in skin inflammation.

Dysregulation of the immune response may lead to excessive inflammation, vasodilation, lymphatic dilatation, and angiogenesis.

Neurocutaneous mechanisms

Triggers include ultraviolet (UV) radiation, temperature change, exercise, spicy foods, alcohol, psychological stress, air pollution, and tobacco smoking. Calcitonin gene-related peptide (CGRP) may play a role in flushing and erythema.

Impaired skin barrier

Affected skin displays features indicating skin barrier impairment, allowing bacterial colonisation and inflammation.

Innate immunity

In the skin of patients with rosacea, there is increased expression and activity of toll-like receptor 2, cathelicidins, kallikrein 5, and mast cells.

Furthermore, cathelicidin LL-37 increases sensitivity of the skin to the sun.

The result is an exaggerated innate immune reaction to the initial trigger.

Adaptive immunity

Dominant T-helper (Th)1/Th17 gene expression in all features of rosacea.

Increased Th17 expression can increase levels of cathelicidin LL-37 in keratinocytes and drive further inflammation.

The most significant environmental trigger is UV radiation; affected skin is more sensitive to exposure. UV radiation can damage the dermis and increase skin inflammation.

What are the clinical features of rosacea?

Cutaneous features include:

Transient recurrent erythema, ie, **flushing**

Persistent facial erythema

Telangiectasia

Facial skin other than in the nasal alar region

Eyelid margin telangiectasia

Together often termed erythematotelangiectatic rosacea

Inflammatory papules and pustules (papulopustular)

Phymatous changes

Thickening of the skin due to **hyperplasia/fibrosis of the sebaceous glands** of the face

Most common area affected is the nose (termed **rhinophyma**)

More commonly present in men.

Rhinophyma

Rhinophyma showing swelling and sebaceous gland openings

Steroid induced papulopustular rosacea

Steroid induced rosacea on the forehead

Steroid induced facial rosacea

Steroid induced papular rosacea

Swelling and papulopustules in steroid induced rosacea

Occasionally rosacea induces facial lymphoedema (Morbihan disease), producing redness, and swelling of the face and lids.

Facial tenderness and burning pain accompanied by redness and flushing (neurogenic rosacea) is a rare variant of rosacea.

Lid lymphoedema and glabellar swelling in Morbihan disease

Violaceous swelling of the lower lids in Morbihan disease

Non-cutaneous ocular features (affects over 50% of patients with rosacea):

- Dryness
- Foreign-body sensation
- Photophobia
- Conjunctivitis
- Blepharitis
- Keratitis – can lead to long-term eyesight impairment.

[Click here for images of rosacea](#)

How do clinical features vary in differing types of skin?

Rosacea is diagnosed more frequently in fair-skinned patients of Celtic and Northern European descent.

It may be harder to identify key features of rosacea in patients with skin of colour. These features are likely under-recognised and rosacea may be underdiagnosed in these patients.

What are the complications of rosacea?

Complications of rosacea include:

Phymatous rosacea

Inflammatory eye complications, eg, blepharokeratoconjunctivitis, sclerokeratitis

Physical discomfort, eg, from ocular symptoms

Negative psychosocial effects such as increased anxiety, depression, low self-esteem, and social isolation

Trigger avoidance leading to lifestyle limitations.

How is rosacea diagnosed?

Rosacea is diagnosed clinically in the majority of cases. Diagnosis is made according to diagnostic and major criteria recommended by the 2017 global ROSacea COnsensus (ROSCO) panel. This requires one diagnostic criterion or two major criteria to be fulfilled.

In patients with darker phototypes where erythema and telangiectasia (visible blood vessels) is more difficult to visualise, greater emphasis may be placed on other major and minor features.

Diagnostic criteria

Persistent centrofacial erythema associated with periodic intensification by potential trigger factors

Phymatous changes.

Major criteria (must occur in centrofacial distribution)

Flushing/transient centrofacial erythema

Inflammatory papules and pustules

Telangiectasia – visible blood vessels (excluding nasal alar telangiectases, which are common in adults)

[Ocular rosacea](#) (lid margin telangiectasia, blepharitis, keratitis/conjunctivitis/sclerokeratitis/anterior uveitis).

Minor features

Burning sensation of the skin

Stinging sensation of the skin

Oedema

Dry sensation of the skin.

In cases where there is diagnostic uncertainty, [skin biopsy](#) may be considered.

What is the differential diagnosis for rosacea?

Other conditions that could present with similar cutaneous features include:

- Acne vulgaris
- Carcinoid
- Demodicosis (demodex folliculitis)
- Dermatomyositis
- Drug reaction
- Eczema
- Idiopathic facial aseptic granuloma
- Periorificial dermatitis or periocular dermatitis
- Photo-damaged skin
- Pyoderma faciale
- Seborrhoeic dermatitis
- Steroid-induced acne
- Steroid-induced rosacea
- Systemic lupus erythematosus.

What is the treatment for rosacea?

Although there is no cure for rosacea, symptoms can be managed with the following lifestyle measures, medical, and procedural interventions.

General measures

All patients with rosacea should receive education on general skincare and lifestyle measures.

Lifestyle advice

Encourage patients to record a symptom diary to aid the identification of triggers:

Common triggers include spicy food, hot/cold temperatures (hot baths), exercise, sun exposure, cosmetic products, medications (those that cause vasodilation), alcohol, fruits and vegetables, dairy, marinated meat products

Avoid the triggers identified.

General skincare advice

Moisturise frequently

Use gentle over-the-counter cleansers

Mild, synthetic detergent-based cleansers rather than traditional soaps due to risk of irritation

Use physical sunscreens (ie, zinc oxide/titanium oxide) with SPF ≥ 30

Provides broad-spectrum UV radiation and visible light protection

May be better tolerated than chemical sunscreens

Avoid exfoliants

Avoid alcohol-based topical products

Avoid use of topical steroids as they may aggravate the condition

Cosmetics with a green tint are useful to minimise the appearance of redness.

Psychosocial considerations

Assess the patient's psychosocial burden of disease and consider referral for psychological support where necessary.

Specific measures

Existing treatments for rosacea can be very effective — however, they often target only one feature. This means that a combination of therapies are required where patients present with multiple features and in severe rosacea.

Many of the following treatments are first-line therapies recommended by the 2019 ROSCO panel:

Transient erythema (flushing)

Alpha-adrenergic agonists (topical [brimonidine](#), topical oxymetazoline) — they are often used infrequently for special occasions only, as persistent use may result in rebound flushing on discontinuation

[Oral beta-blockers \(carvedilol\)](#)

Oral clonidine may reduce flushing

CGRP antagonists (erenumab)

Persistent erythema

Alpha-adrenergic agonists (topical brimonidine, topical oxymetazoline, as above)

[Intense pulsed light therapy](#)

[Vascular laser](#)

Inflammatory papules/pustules

Topical [azelaic acid](#) (for mild/moderate only)

Topical [ivermectin](#)

Topical [metronidazole](#) (for mild/moderate only)

Topical [erythromycin](#)

[Oral tetracyclines](#) (oxytetracycline, lymecycline, doxycycline)

Oral macrolides ([erythromycin](#), azithromycin)

Oral [metronidazole](#)

Oral [isotretinoin](#) often at low dose (for refractory disease only)

Telangiectasia

[Electrodesiccation](#)

[Intense pulsed light therapy](#)

[Vascular laser](#)

Neurogenic rosacea

Gabapentin

Amitriptyline

Oral beta-adrenergic blockers

Consideration of endoscopic sympathectomy

Phyma

If clinically inflamed: doxycycline, isotretinoin

If clinically non-inflamed: physical modalities to remove excess tissue and reshape the structures (eg, **ablative CO₂ laser, erbium laser**, radiofrequency, surgical debulking).

Ocular rosacea

General management

Increase dietary intake of omega-3 fatty acids

Warm compresses

Gentle eyelash/eyelid cleansing to express sebum trapped in the meibomian glands

First-line medical management

If mild-moderate: topical azithromycin/**topical calcineurin inhibitors**

If severe: azithromycin, doxycycline.

For more information, see [Ocular rosacea](#).

What is the outcome for rosacea?

Although rosacea is not a life-threatening condition, it is a chronic disease that requires long-term management of relapsing and remitting symptoms. Complete resolution of clinical features has been shown to prolong time to symptom relapse and have greater positive impact on quality of life compared with incomplete resolution.

[Click here for images of rosacea](#)

Scabies

Author: Dr Jessica Rachel Maguire, IMT,
Dermatologist, United Kingdom, April 2

Dr Ian Coulson,

What is scabies?

Scabies is a transmissible skin disease caused by the ectoparasitic mite *Scabies var. hominis*. This variant infests humans only; scabies cannot be caught or transferred to other animals.

Scabies is a highly contagious infestation of the human epidermis. Scabies was described by Aristotle who likened the disease to 'lice of the flesh'. Scabies presents as a rash with intense itching; it may have a characteristic appearance and distribution.

[Click here for images](#)

Who gets scabies?

Scabies is a global disease, and anyone can be affected. It is estimated that about 204 million people are affected by scabies worldwide, with an annual incidence of 455 million cases. Scabies is more common in the elderly, children, and adolescents.

The burden of disease is higher in low-income areas, in the tropics, and in poorly resourced communities and countries.

Risk factors for scabies, particularly in developing countries, include:

- Crowded conditions
- Poor hygiene
- Poverty
- Malnutrition
- Homelessness
- Immunodeficiency.

In developed countries, scabies occurs in family outbreaks and children may acquire it from close play contact in school or sleepovers. In situations where there may be close physical contact for the delivery of personal care, such as in [care homes](#), large outbreaks are not uncommon. In young adults, sexual spread is frequent.

What causes scabies?

Scabies infection is usually transmitted through close bodily skin contact such as holding hands for prolonged periods; spread amongst sexual partners is common. A brief handshake or hug does not usually allow for transmission unless the patient has crusted scabies.

Spread via fomites (clothing, towels, etc.) is very uncommon as the mite perishes within hours of leaving the host; it is relevant in [crusted scabies](#).

Scabies cycle

The smaller male mite fertilises the female on the surface of the skin and dies shortly after mating.

The female mite burrows into the stratum corneum of the host where they lay 2 to 3 eggs per day.

The female continues to burrow at a rate of 0.5 to 5mm per day for the duration of its 4 to 8-week lifespan.

Eggs are deposited in the burrow and hatch into larvae after approximately 2 to 5 days.

Hatched larvae mature into adult scabies mites in about 3 weeks. They migrate to the skin surface, and the cycle continues.

The itchy rash is the result of a TH1-mediated hypersensitivity reaction to various mite-related antigens and is thus delayed.

What are the clinical features of scabies?

Infection typically presents with a classical itchy rash. Lesions are symmetrical, and mainly affect the hands, wrists, axillae, thighs, buttocks, waist, soles of the feet, areola and vulva in females and penis and scrotum in males. The neck and above are usually spared, except in cases of crusted scabies and in infections occurring in infants, the elderly, and the immunocompromised.

However, the rash may be generalised and [eczematous features](#) may dominate and mask the classical signs. Clinical suspicion is paramount.

Itch

Generalised

Occurs in 4 to 6 weeks following initial infection (during which time the host is infectious)

Occurs within hours of subsequent re-infection

Worse at night-time

May persist for several weeks after completion of treatment.

Rash

Erythematous papules

Excoriations

Linear scratch marks

[Dermatitis](#)

Nodules which may be skin coloured, red-brown, or violaceous

Crusting (hyperkeratosis as seen in crusted scabies)

Vesicles which may also be secondary to a superimposed bacterial infection.

Pathognomonic features which can assist in diagnosis

Burrows

Curvilinear or serpiginous thread-like tracks measuring around 5–10 mm – these can be subtle

Typically identified in the web spaces, palms, soles, fingers, toes, inner wrists, elbows, umbilicus, and beltline.

Nodules

Varying in size between 3 and 15 mm

May develop on the penis and scrotum in men
On the upper thighs and gluteal folds
Around the areolae in women
In the axillae



A scabies burrow on the toe



A burrow on the palm - the mite can just be seen adjacent to the vesicle



Numerous palmar scabies burrows in an elderly lady



Scabies burrows on the palm



Scabies burrows on a babies foot



Interdigital scaling in the first web space of the hand

Crusted scabies

This variant occurs in the elderly and immunosuppressed – the palms and soles become covered in crusted plaques. There is massive infestation with mites, numbering in the thousands; it is highly infectious and there may be heavy infestation of bedding and upholstery.



Thick crusted plaques on the thumb and palm due to crusted scabies in an elderly woman

[Click here for more images](#)

Secondary eruption

This can be a widespread nonspecific eczematous eruption on the torso and limbs and may partially obliterate the primary pathognomonic features.

How do clinical features vary in differing types of skin?

Scabies may present as granulomatous nodules in an infected person with a darker skin type.

Inflammatory changes appear as redness in white skin and greyness in black skin.

What are the complications of scabies?

Crusted scabies

Previously known as Norwegian Scabies.

Associated with dense hyperkeratosis of the skin.

Caused by an altered host immune response.

Seen particularly in patients with:

Reduced T-cell immunity e.g., [HIV](#)

Reduced peripheral sensation e.g., [leprosy](#)

Patients unable to debride mites mechanically e.g., [Down syndrome](#) and subungual hyperkeratosis.

Results in uncontrolled proliferation of mites (thousands to millions of mites, compared to 10–15 seen in classical scabies).

Bullous scabies

Usually seen in elderly males.

Bullae may be tense or flaccid.

Thought to be due to either secondary infection of a scabetic lesion with *Staphylococcus aureus* or due to an autoantibody response.

Nodular scabies

May be due to a local hypersensitivity reaction to dead mites or may be secondary to persistent infection.

Commonly affects male genitalia (may be a widespread dominant feature in children) and breasts.

Can be seen around the axillae or on the buttocks.

May persist for months despite otherwise successful scabies treatment.

Other complications of classical scabies include:

Scabies incognito

Modification of the clinical picture secondary to inappropriate use of steroids after misdiagnosis.

Results in delayed diagnosis, sometimes for months or even years.

Secondary bacterial infection

Staphylococcus aureus or *Streptococcus pyogenes* bacteraemia.

Impetigo.

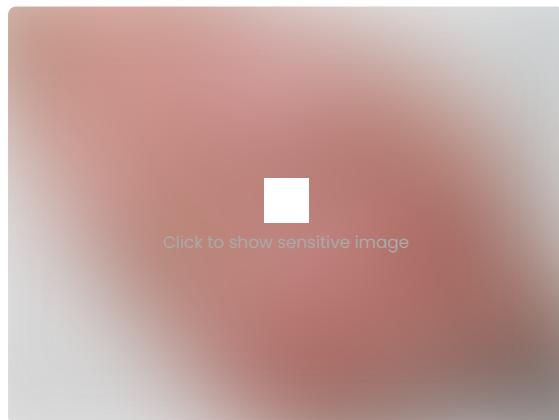
Pustular lesions on the palms, soles, fingers, and toes.

Toxin-mediated diseases such as [toxic shock syndrome](#), glomerulonephritis, and acute rheumatic fever.

Psychological distress

Scabies often afflicts young adults living away from home for the first time and the development of a contagious rash can be embarrassing. Stigma can lead to an inability to ensure household or sexual contacts are treated.

Sleep disturbance and an unsightly rash compound the emotional impact of the illness, especially where diagnosis and comprehensive treatment is delayed, when the symptoms can persist for many months.



Click to show sensitive image

Scabies with superadded streptococcal infection



Scabies nodules in the axilla



Axillary scabies nodules in an infant

How is scabies diagnosed?

A high index of suspicion should be used when assessing a patient with a new widespread itchy rash, especially one who reports itchy close contacts. The diagnosis is typically clinical and most easily confirmed using [dermoscopy](#).

Several invasive and non-invasive tests exist to aid in confirming a diagnosis of scabies:

Dermoscopy may reveal the 'delta wing jet' or 'mini triangle' sign. In crusted scabies, dermoscopy may show hyperkeratosis.

For information on trichoscopic findings in scabies of the scalp, see [Trichoscopy of scalp infestations](#).

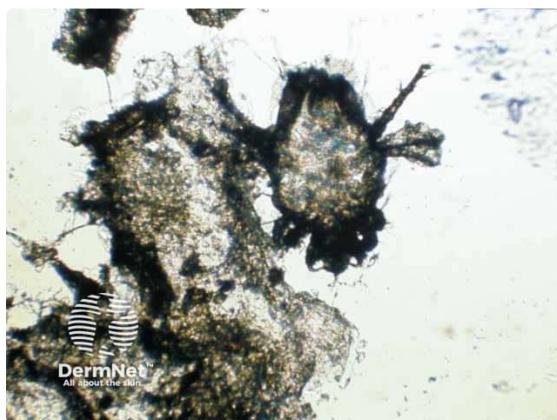
Skin scrapings using either a blade or needle allow direct visualisation of mites eggs or mite faeces on microscopy. Visualisation of a live mite by the patient galvanises treatment concordance.

A burrow ink test may reveal a classical zig-zag line where the ink has tracked into a burrow.

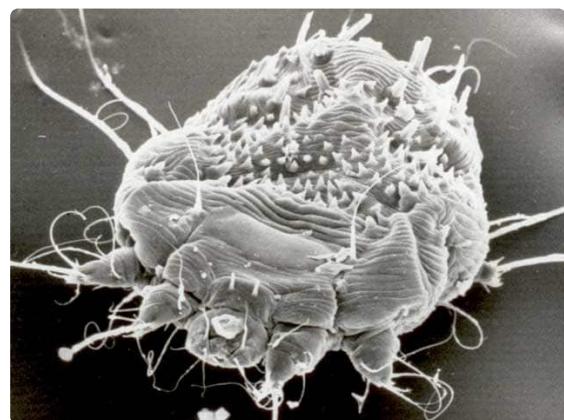
Adhesive tape test is used to transfer a sample from a suspicious lesion directly to a microscope slide.

[Skin biopsy](#), although rarely necessary, will show eggs, larvae, faeces, and mites.

It is useful to examine close contacts in suspicious cases as pathognomonic signs are often seen in those with minimal symptoms.



A scabies mite on microscopy after extraction from a burrow



Scanning electron microscopy of Sarcoptes scabiei

The International Alliance for the Control of Scabies (IACS) updated consensus criteria for the diagnosis of scabies in 2020, copied below:

A: Confirmed scabies is diagnosed if there is at least one of:

A1: Mites, eggs or faeces on light microscopy of skin samples

A2: Mites, eggs or faeces visualized on an individual using a high-powered imaging device

A3: Mite visualised on an individual using dermoscopy.

B: Clinical scabies is diagnosed if there is at least one of:

B1: Scabies burrows

B2: Typical lesions affecting male genitalia

B3: Typical lesions in a typical distribution and two history features.

C: Suspected scabies is diagnosed if there is one of:

C1: Typical lesions in a typical distribution and one history feature

C2: Atypical lesions or atypical distribution and two history features.

History features are:

H1: Itch

H2: Positive contact history with an individual who has an itch or typical lesions in a typical distribution.

Notes

1. These criteria should be used in conjunction with the full explanatory notes and definitions (in preparation by IACS).
2. Diagnosis can be made at one of the three levels (A, B or C).
3. A diagnosis of clinical and suspected scabies should only be made if other differential diagnoses (such as eczema and impetigo) are considered less likely than scabies.

What is the differential diagnosis for scabies?

[Bullous skin disorders](#)

[Dermatitis](#)

[Folliculitis](#)

[Papular urticaria](#)

[Prurigo \(subacute\)](#)

[Psoriasis](#)

[Infantile acropustulosis](#)

What is the treatment for scabies?

General measures

Launder sheets, towels, and clothes worn recently. Outer garments, duvets, and blankets should be aired for 72 hours.

Vacuum soft furnishings where possible, although this may be of limited benefit.

Clip nails and clean any subungual debris.

Specific measures

It is important to note that all close contacts of a confirmed case of scabies should complete eradication therapy, whether they are symptomatic or not. Contacts may be infected but asymptomatic for several weeks, therefore they may continue to infect others and even reinfect the index case.

Topical therapies

Topical 5% [permethrin](#) (acaricidal and ovicidal) cream or lotion remains first-line therapy.

Applied from the jawline downwards, and left overnight for 8–12 hours. Reapplication is needed if hands are washed during the treatment period. Infants, the elderly, and the immunosuppressed should also treat the face and scalp.

Application should include under the nails and between the toes.

Treatment should be repeated after 7–10 days.

If the case is permethrin-resistant, or permethrin is not available, alternative [insecticides](#), include 10–25% benzyl benzoate or 0.5% malathion aqueous lotion.

Spinosad is a topical horticultural insecticide and is effective against scabies where it is available.

Oral therapies

Oral [ivermectin](#) is indicated in cases of topical failure, inability to comply with topical therapy, non-adherence to topical therapy, institutional outbreaks, mass treatment of populations, and crusted scabies.

Repeat dose after 2 weeks.

Latest data suggests oral ivermectin is equally as effective as topical 5% permethrin cream or lotion, however may be slower-acting.

According to French dermatology guidance (2024), ivermectin may be an option for children over the age of 2 months. It can also be used in breastfeeding women and during pregnancy from the second trimester onwards, if topical therapies have proved ineffective.

Oral moxidectin is as yet unlicensed, but an effective agent that has good skin retention – it may be effective as a single treatment and prevent re-infestation.

Practical advice to patients

Provide written instruction.

Explain the necessity for complete, contemporaneous compliance with topical therapy.

Household members should elect an early, convenient evening on which to be treated, assemble bedding and clothing for laundry, and apply the topical treatment before dressing and remaking beds.

Itching may last for several weeks, and this does not represent persistent infection or treatment failure.

Post-scabetic itch

May persist for weeks

Moderate potency [topical steroids](#) may be used and required for 3–4 weeks, particularly if the patient has a widespread eczematous reaction

[Antihistamines](#) are helpful in some cases

Emollient use and soap avoidance should be advised

Persistent nodules may need potent [topical](#) or [intralesional](#) steroids.

Scabies and pregnancy

5% permethrin cream or lotion is considered safe in pregnancy and while lactating.

Alternative topical preparations include sulphur 5–10% cream, ointment, or lotion.

Oral ivermectin is not advised during pregnancy.

What is the outcome for scabies?

Classical scabies is associated with a good prognosis provided compliance is satisfactory and all close contacts (symptomatic or not) are simultaneously treated. Crusted scabies may need prolonged and repeated treatment and patients may have significant underlying conditions that will influence the prognosis. Pyococcal infection of scabies is one of the most common causes of acute glomerulonephritis globally.

Scabies reinfection is common and is usually secondary to spread from untreated close contacts. Repetitive use of disinfectants can lead to [irritant dermatitis](#), and in the mistaken belief of continued scabies infestation, yet more disinfectant is used.

[Click here for images](#)

Scalp psoriasis

November 2022

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Edited by the DermNet content department

What is scalp psoriasis?

Scalp psoriasis is a skin condition characterised by red, thickened, well-demarcated patches or plaques with overlying silvery-white scales, affecting part or all of the scalp.

It can be contained within the hairline, though frequently affects the back of the head, or extends onto the forehead ([facial psoriasis](#)), ears, or neck. While often camouflaged by the hair, scalp psoriasis can be a source of embarrassment and distress due to itching and dandruff-like flaking. It may occur in isolation, or with any other form of [psoriasis](#), and is typically a chronic, relapsing-remitting condition.



Scalp psoriasis extending beyond the anterior hairline



Confluent psoriasis in the scalp extending anteriorly from the hairline



Marked thick scaling in psoriasis of the scalp



Extensive scaling over the parietal scalp due to psoriasis.
There is psoriasis also in the ears and around the hair line



Marked scaling in psoriasis of the scalp



Severe scalp psoriasis with areas of pityriasis amiantacea

[Click here for more images](#)

Who gets scalp psoriasis?

Psoriasis is estimated to affect 2% of the population worldwide and can occur at any age. Among those affected by psoriasis, approximately 80% experience scalp involvement.

What causes scalp psoriasis?

In [psoriasis](#), skin cells tend to form quickly (hyperproliferation), within days rather than weeks, due to faulty immune system signals. As a result, excess cells pile up on the skin surface, causing patches and plaques.

Psoriasis, including scalp psoriasis, is thought to be caused and affected by a combination of genetic, immune, hormonal, and environmental factors, such as:

- [Bacterial infections, such as streptococcal sore throats](#)
- [Human immunodeficiency virus \(HIV\) infection](#) and acquired immunodeficiency syndrome (AIDS)
- Hormonal changes (psoriasis often intensifies around [puberty](#), the postpartum period, and menopause; and often improves during [pregnancy](#))
- Sunlight/[ultraviolet radiation](#) — usually helps but occasionally exacerbates psoriasis
- Use of harsh chemical products or [soaps](#)
- [Stress](#)
- [Obesity](#)
- Trauma or injury to the skin ([Koebner phenomenon](#))
- [Cigarette smoking](#)
- [Alcohol consumption](#)
- [Medication](#) — this may induce or exacerbate psoriasis.

What are the clinical features of scalp psoriasis?

Scalp [psoriasis](#) is characterised by well-defined, red, thickened patches or plaques on the scalp with overlying silvery-white scales.

Scales can flake off causing 'dandruff'.

It can be localised to parts of the scalp and often affects the back of the head, or can involve the entire scalp.

It can be very [itchy](#)

Scaling may build up and produce an appearance similar to overlapping Mediterranean roof tiles ([Pityriasis amiantacea](#))

In severe cases, scalp psoriasis can be associated with temporary localised hair loss ([alopecia](#)).

[Click here for images](#)

How do clinical features vary in differing types of skin?

According to Gelfand et al (2005), the prevalence of all psoriasis was 1.3% in black patients compared to 2.5% in white patients, which is probably linked to genetics.

However, the diagnosis of psoriasis may also be delayed or missed in darker [skin phototypes](#) where the presentation of psoriasis may vary from purple to dark brown patches with grey or silver scales.

When managing scalp psoriasis in patients with different skin and hair types, it is important to formulate a topical treatment regimen that is compatible with each patient's hair care practices and cultural preferences. For example, hair texture that requires a reduced frequency of hair washing thereby renders daily medicated shampoos unsuitable. Once weekly washing in conjunction with daily application of a topical corticosteroid in a vehicle compatible with preferred hair styling practices may be more acceptable.

What are the complications of scalp psoriasis?

Dry itchy scalp.

Sleep disturbance secondary to itching.

[Skin infections](#) due to scratching and impaired skin barrier.

Anxiety and reduced self-esteem due to scalp appearance and dandruff-like flaking.

Reversible hair loss ([alopecia](#)).

Rarely, scarring alopecia can develop due to chronic, relapsing scalp psoriasis.

Scalp psoriasis may be associated with [psoriatic arthritis](#) (PsA).

How is scalp psoriasis diagnosed?

Scalp psoriasis is generally diagnosed clinically. Key trichoscopic findings include red dots, hairpin vessels, and red globular rings (for more information, see [trichoscopy of inflammatory conditions](#)). A [skin biopsy](#) may be performed in some cases to confirm the diagnosis.

Additionally, assessing psoriasis severity – for example, using the Psoriasis Scalp Severity Index (PSSI) – and its impact on quality-of-life is important.

What is the differential diagnosis for scalp psoriasis?

[Seborrhoeic dermatitis](#) – can co-exist with scalp psoriasis (called [sebopsoriasis](#)).

Fungal scalp infection ([tinea capitis](#)).

Pityriasis amiantacea

Pityriasis rubra pilaris.

What is the treatment for scalp psoriasis?

Scalp psoriasis can be difficult to treat due to the delivery of therapy in and around the hair, which complicates the application of many topical products. Cosmetic considerations also affect treatment adherence.

Usually, lotions, solutions, or gels are more suitable for the scalp than heavier products such as ointments. In recent years, a number of new formulations have been developed (eg, foams, shampoos, and sprays) that enhance cosmetic acceptability and adherence. Most treatments will need to be used regularly for several weeks before a benefit is seen, and may have to be applied regularly to keep the scalp clear.

See [Treatment of psoriasis](#) for more information.

General measures

Regular use of [emollients and moisturisers](#) such as scalp oils.

Avoiding known triggers where possible.

Modifiable lifestyle factors such as maintaining a healthy weight, limiting alcohol intake, and smoking cessation.

Avoiding picking or scratching the scalp, which can cause further damage.

Some people find cutting their hair short helps control scalp psoriasis, likely as it makes treatments easier to apply.

Specific measures

Topical medications are recommended as first-line treatment of mild-to-moderate scalp psoriasis, and can also be used concomitantly with phototherapy and/or systemic therapies in moderate-to-severe cases.

Topical agents

[Topical corticosteroid](#) is recommended for short-term treatment (eg, clobetasol propionate 0.05% shampoo, steroid foams, water-based gels, scalp applications); there is limited data on long-term monotherapy for the scalp.

Topical Vitamin D analogues eg, [calcipotriol](#) — unlike steroids, these do not cause skin atrophy; however, they are found to be less effective than corticosteroids for scalp psoriasis (also not recommended for use on the face).

Combined corticosteroid and Vitamin D therapies eg, [calcipotriol 0.005% and betamethasone dipropionate 0.05% gel](#) has been found to be superior to its individual ingredients, with a fast onset of action and no reports of skin atrophy, striae, purpura, or significant effects on serum calcium.

[Keratolytic \(anti-scaling\) agents](#), eg, [salicylic acid](#), [urea](#), or a dimethicone-based topical keratolytic spray (eg, Loyon®)

Salicylic acid shampoos can enhance the penetration of other topical treatments including corticosteroids, and have been recommended by the US National Psoriasis Foundation.

[Coal tar](#) shampoos (2–10%) — coal tar can be very effective for body psoriasis although less evidence for use in scalp psoriasis, and less cosmetically acceptable as can stain the scalp and hair.

Coconut oil compound ointment (eg, Coco-Scalp®) — a combination of coal tar, salicylic acid, and sulphur that can be left on for at least an hour (or even overnight) before being shampooed off. Compliance can be aided by watching an instructional video on its application and use (see [Treating scalp psoriasis](#) linked below).

[Ketoconazole](#), ciclopirox, zinc pyrithione, and other [antifungal shampoos](#) — effective for dandruff and seborrhoeic dermatitis; varying effect on sebopsoriasis and psoriasis.

Less commonly used topical therapies

[Intralesional corticosteroids](#) — have been applied in practice and remain a second-line treatment option, although specific studies evaluating this treatment regimen for scalp psoriasis

are lacking.

Dithranol — a ‘short contact’ cream. Less commonly used now but can produce long remissions. Can cause irritation and staining; avoid in those with grey or blonde hair.

Phototherapy

Targeted phototherapy with a laser or non-laser light source can help improve symptoms.

Can be challenging to target the scalp in the presence of hair.

Direct treatment may be helped by a handheld **ultraviolet B (UVB)** comb device.

Systemic agents

Consider first-line for patients with scalp psoriasis and accompanying moderate-to-severe whole-body psoriasis; and second-line in other cases where there has been an inadequate response to topical therapy.

Immunosuppressants such as **methotrexate** and **ciclosporin**.

Vitamin A derivative **acitretin**.

Phosphodiesterase-4 (PDE4) inhibitor **apremilast**.

Biologic agents such as anti-TNF, IL-17 and IL-12,-23 inhibitors (eg, **etanercept**, **adalimumab**, **infliximab**, **brodalumab**, **secukinumab**, **ixekizumab**, **ustekinumab**, **guselkumab**, **risankizumab**, and others.

Support groups can also be helpful for those living with psoriasis.

How do you prevent scalp psoriasis?

Scalp psoriasis tends to be a chronic problem. However, regular scalp care, maintenance treatment, lifestyle factors, and avoiding triggers or exacerbating factors can help prevent or reduce the severity of flares.

What is the outcome of scalp psoriasis?

While scalp psoriasis is generally a chronic, relapsing-remitting condition, there are many available treatment options. The mainstay of treatment is topical therapy, although this can be challenging for scalp conditions given their location and the presence of hair.

Newer topical formulations, such as foams and sprays, can help to improve treatment tolerability and outcomes. In patients with moderate-to-severe disease who do not respond to topical treatments, there are a number of systemic therapies including **immunomodulatory agents** and **biological treatments**.

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Scalp tumours and cysts

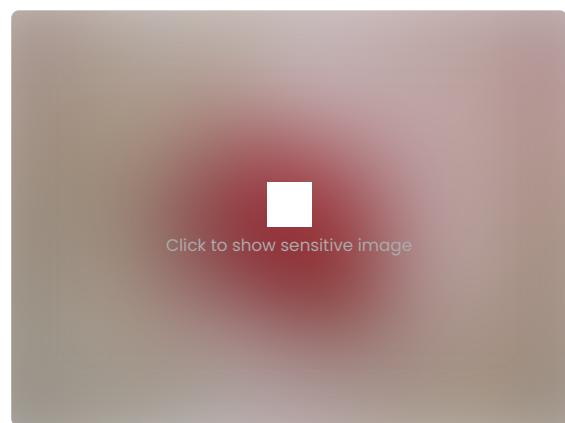
Authors: Rajan Ramji, Clinical Medical Education Fellow, Faculty of Medical and Health Sciences, The University of Auckland, Auckland, New Zealand; Jenny Chung, Dermatology Registrar, Middlemore Hospital, Auckland, New Zealand. Copy edited by Gus Mitchell. February 2022

What are scalp tumours?

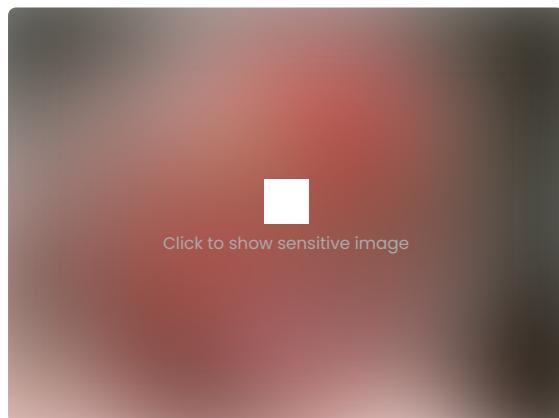
The scalp comprises the area from the back of the head (beginning at the superior nuchal lines) to the eyebrows (supraorbital margin). Scalp tumours are benign or malignant cutaneous lesions which arise on the scalp.



Pilar scalp cysts



Click to show sensitive image



Giant neglected basal cell carcinoma ulcerated down to the skull



Superficial spreading malignant melanoma on the scalp with recent development of nodular component

Angiosarcoma

Who gets scalp tumours?

Scalp tumours occur worldwide. Most scalp tumours (93–99%) are benign as opposed to malignant.

Approximately 40–50% of benign scalp tumours are cysts with an estimated 20% incidence in Western populations. [Trichilemmal \(or pilar\) cysts](#) are especially common and it is estimated 80% of these cysts occur on the scalp. The remaining proportion of benign scalp tumours primarily comprises [lipomas](#) (~30%) and [melanocytic naevi](#) (28%). [Seborrhoeic keratoses](#) and [actinic keratoses](#) are increasingly common with age and the latter develop particularly as the hair thins.

Although only 1–2% of scalp tumours are malignant, they comprise approximately 13% of malignant cutaneous tumours. The most common (in decreasing order of commonality) malignant scalp tumours include [basal cell carcinoma](#) (~41%), [squamous cell carcinoma](#) (~17%), [cutaneous metastases](#), [adnexal tumours](#), [angiosarcomas](#), and [lymphomas](#).

What causes scalp tumours?

The causes of both benign and malignant scalp tumours are varied and can depend on the underlying tissue of origin and associated co-morbidities. Scalp tumours can arise from cells in both the skin (epidermis and dermis) and deeper tissue layers. It may also originate from other cells in the body due to metastases.

What are the clinical features of scalp tumours?

Both benign and malignant scalp tumours can occur elsewhere on the body but may have different physical features. The exact features displayed are dependent on the originating site and cells of the tumour, summarised in Table 1 and 2.

Table 1. Benign scalp tumours

Epidermoid cyst	Keratinocytes	Firm, flesh-coloured or yellow papules/nodules which may have a central punctum that exudes foul smelling debris.
Dermoid cyst	Keratinocytes	Firm dough-like lumps consisting of epidermal/dermal tissue components.
Seborrhoeic keratosis	Keratinocytes	Flat or raised lesions with a stuck-on appearance, variable coloration and diameter. Commonly seen in adults over 60 years of age.
Melanocytic naevus	Melanocytes	Flat or raised localized proliferation of melanocytes. Higher propensity for scalp variants to display dysplastic histological features.

Blue naevus	Melanocytes	Flat or raised localized proliferation of spindle shaped or ovoid naevus cells in the dermis.
Trichilemmal cyst	Hair follicles	Keratin filled nodules derived from the outer hair root sheath and lacking a central punctum. Rarely develops into benign proliferating trichilemmal tumours although this occurs more commonly on the scalp – especially in older women.
Pilomatrixoma	Hair follicles	Skin coloured or purplish irregular papules derived from hair matrix cells which commonly become hard and bony due to calcification.
Sebaceoma	Sebaceous glands	Sebaceous cell proliferations that manifest as skin coloured or yellow nodules originating from deeper in the skin than sebaceous adenomas.
Sebaceous adenoma	Sebaceous glands	A more superficially located form of sebaceomas manifesting as skin coloured or yellow papules or nodule
Hidrocystoma	Apocrine/eccrine glands	Apocrine or eccrine derived skin coloured or blue cysts which may arise as solitary multiple lesions. Typically seen on the scalp or face – particularly the eyelid margins (Moll gland cysts).
Syringoma	Apocrine/eccrine glands	Firm skin coloured or yellow papules millimetres in diameter and commonly occurring in clusters.
Eccrine poroma	Apocrine/eccrine glands	Papules, plaques, or nodules derived from the epithelial terminal duct which histologically differentiate into poroid (glandular duct) cells.
Lipoma	Adipocytes	Smooth round collection of subcutaneous fat with a rubbery texture to palpation.
Infantile haemangioma	Vascular	Bright red, blue or flesh-coloured, non-tender and non-pulsatile papules/plaques representing a vascular malformation in the dermis or subcutaneous tissue.

Cavernous haemangioma	Vascular	Infantile haemangiomas representing vascular malformations in the lower dermis or subcutaneous tissue.
Venous malformation	Vascular	Skin coloured, blue or purple swellings of variable size that represent malformed veins and are a form of vascular naevi.
Lymphangioma	Lymphatic	Malformed lymph ducts of variable size that are a form of vascular naevi and most prominent in infancy or childhood
Leiomyoma	Myocytes	Proliferations of myocytes which can develop from both smooth and skeletal muscle. Typically present as firm, smooth, and tender hyperpigmented or red-brown nodules.
Dermatofibroma	Collagen(fibrous)/ histiocytes	Solitary, firm papules or nodules of variable coloration that may dimple on pinching.
Hypertrophic scar	Collagen(fibrous)/ histiocytes	A growth of fibrous tissue that develops as part of wound healing processes. Typically begins as red and prominent before becoming flat and pale.
Keloid scar	Collagen(fibrous)/ histiocytes	Firm smooth fibrous tissue that characteristically extends beyond the site of the precipitating injury.
Infantile myofibromatosis	Collagen(fibrous)/ histiocytes	Firm or rubbery circular nodules formed from the proliferation of myofibroblasts in the dermis or subcutaneous tissue.
Neurofibroma	Neural	Well circumscribed soft or firm growths derived from Schwann cells, fibroblasts, mast cells, and vascular components of underlying nerves. Occurs in association with Café-au-lait macules in Neurofibromatosis 1 (NFI).
Schwannoma	Neural	Smooth, soft, and solitary skin-coloured or yellow papules or nodules originating in the dermis or subcutaneous tissue and derived from Schwann cells forming the myelin sheath of nerves.
Langerhans cell histiocytosis	Haematologic	Overactive accumulation of Langerhans cells in the epidermis with a spectrum of clinical manifestations typically seen in childhood or adolescence.

		May appear as pink or reddish-brown papules, pustules, vesicles, or blisters with crusting, scale, or impetiginisation.
Rosai-Dorfman disease	Haematologic	Unprovoked histiocyte proliferation disorder characterized by massive cervical lymphadenopathy. Less than 10% of cases may manifest with multiple macules, papules, nodules, and plaques of red, red-brown or yellow coloration.
Juvenile xanthogranuloma	Haematologic	A non-Langerhans cell histiocytosis that typically manifests as domed red-brown or yellow papules or nodules in children or adolescents. Typically manifests in skin but may also develop in eyes or internal organs.

Table 2. Malignant scalp tumours

Squamous cell carcinoma	Keratinocytes	A malignant proliferation of keratin producing cells extending beyond the epidermis. Scalp variants more frequently present with ulceration and have a higher propensity for recurrence.
Intraepidermal squamous cell carcinoma	Keratinocytes	Also known as Bowen's disease. A malignant proliferation of keratin producing cells localized within the epidermis.
Basal cell carcinoma	Keratinocytes	A malignant proliferation of keratin producing cells extending beyond the epidermis. Scalp variants more frequently present with ulceration and have a higher propensity for recurrence. Pigmented or nodular subtypes are also more commonly seen and are more likely to demonstrate a melanocytic pattern on dermoscopy.
Keratoacanthoma	Keratinocytes	Rapidly growing firm circular nodule with a keratin core. A variant of squamous cell carcinoma.
Malignant melanoma	Melanocytes	Indistinct presentation of variously pigmented or nonpigmented lesions derived from a malignant proliferation of melanocytes. Scalp melanomas are more commonly seen in older men and are associated with alopecia.

		Compared to other body areas, there may be a higher propensity to present as recurrent desmoplastic or amelanotic melanoma. Lesions are also more likely to develop ulceration and have a greater Breslow thickness.
Malignant proliferating trichilemmal tumour	Hair follicles	Keratinized nodules or cysts derived from outer hair root sheath cells with low potential for metastasis. May develop from trichilemmal cysts.
Pilomatrix carcinoma	Hair follicles	A low grade adnexal carcinoma derived from hair matrix cells that may manifest as skin coloured or purplish irregular papules.
Sebaceous carcinoma	Sebaceous glands	A form of adnexal carcinoma where the cells demonstrate a differentiation into sebaceous cells. Lesions lack distinguishing features but may appear as yellow nodules or plaques with ulceration or crusting.
Apocrine carcinoma	Sweat glands	An adenocarcinoma derived from apocrine glands. Lesions may manifest as ulcerating or bleeding nodules but otherwise have an indistinct presentation and are usually diagnosed histologically.
Porocarcinoma	Sweat glands	An adenocarcinoma derived from eccrine (sweat) glands. As with apocrine carcinomas, lesions have a nondescript appearance but may appear as ulcerating or bleeding nodules that are diagnosed histologically.
Atypical lipomatous tumour	Adipocytes	Malignant proliferation of adipocytes that rarely develops in the skin but can resemble an enlarging lipoma. Differentiated from liposarcoma on the basis of histology findings.
Liposarcoma	Adipocytes	Malignant proliferation of adipocytes that can present identically to an atypical lipomatous tumour or an enlarging lipoma.
Angiosarcoma	Vascular	Aggressive tumours that uncommonly develop from endothelial cells in blood (haemangiosarcoma) or lymphatic (lymphangiosarcoma) vessels

		<p>May manifest as painful rapidly growing bruises, blue-black nodules, or persistent ulcers.</p>
Leiomyosarcoma	Myocytes	<p>Malignant proliferation of smooth muscle cells that may also develop in the dermis or subcutaneous tissue.</p>
Dermatofibrosarcoma protuberans	Collagen(fibrous)/ histiocytes	<p>Slow growing tumours derived from collagen that develop in the dermis. Typically manifests as red-brown to skin coloured painless lichenified plaques or fixed, firm nodules.</p>
Fibrosarcoma	Collagen(fibrous)/ histiocytes	<p>Malignant proliferation of spindled fibroblasts or myofibroblasts which are typically firm and spherical but otherwise nondescript in appearance. Generally carries a poor prognosis.</p>
Merkel cell carcinoma	Neural	<p>Aggressive tumours with high metastatic potential, thought to be derived from pressure receptors (Merkel cells) in the skin. Approximately 80% of cases are found to have concomitant Merkel cell polyomavirus. The most common site of development is the head and neck region. Scalp lesions are typically larger and have an even higher risk of metastasis.</p>
Malignant peripheral nerve sheath tumour	Neural	<p>Proliferations of one or more types of cells that compose peripheral nerve sheaths – typically originate from perineural or endoneurial fibroblasts. Associated with plexiform neurofibromas.</p>
Lymphoma	Haematologic	<p>A malignant proliferation of lymphocytes. Multiple cutaneous and non-cutaneous variants exist. The most commonly occurring variants on the scalp include primary cutaneous follicle centre and/or marginal zone lymphomas.</p>
Metastases	Various	<p>Secondary malignant proliferations that develop from the spread of a primary malignancy beyond its site of origin. The scalp can both develop malignancies which metastasize and act as a site for metastases from other body sites. Metastases commonly associated with the scalp include:</p>

Melanoma
Papillary thyroid carcinoma
Breast cancer
Pulmonary adenocarcinoma
Colorectal adenocarcinoma
Hepatocellular carcinoma
Cholangiocarcinoma
Renal cell carcinoma
Langerhans cell histiocytosis

How are scalp tumours diagnosed?

Some scalp tumours may be diagnosed through a clinical examination alone. A [biopsy](#) or radiologic workup may be necessary to confirm the diagnosis.

What are the treatments for scalp tumours?

Treatment options are dependent on the nature of the tumour, anatomical location, and underlying diagnosis. Given that malignant scalp tumours carry a worse prognosis than lesions in other anatomic areas, radical surgical excision is more likely to be recommended. [Excision](#) can be complicated by the relative lack of scalp skin mobility. [Mohs micrographic surgery](#) may be a preferable option in these cases.

Other treatments may include:

[Cryotherapy](#)
[Electrodessication and curettage](#)
[Photodynamic therapy](#)
Topical therapy (i.e. 5% [imiquimod](#))
[Radiotherapy](#).

What is the outcome for scalp tumours?

Malignant scalp tumours tend to carry a worse prognosis than equivalent tumours elsewhere on the body. The exact prognosis is dependent on the specific tumour involved and the degree of invasion beyond the skin. Scalp metastases (either originating from scalp tumours or elsewhere) typically carry a poor prognosis.

Seborrhoeic dermatitis

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What is seborrhoeic dermatitis?

Seborrhoeic dermatitis is a common, chronic, or relapsing form of [eczema/dermatitis](#) that mainly affects the sebaceous gland-rich regions of the scalp, face, and trunk.

There are infantile and adult forms of seborrhoeic dermatitis. This benign inflammatory condition is sometimes associated with [psoriasis](#) and is known as [sebopsoriasis](#). Seborrhoeic dermatitis is also known as seborrhoeic eczema ("seborrheic" in American English).

Dandruff (also called ' pityriasis capitis') is an uninflamed form of seborrhoeic dermatitis on the scalp. Dandruff presents as diffuse bran-like scaly patches within hair-bearing areas of the scalp without underlying erythema. Dandruff may be asymptomatic or mildly [pruritic](#).



Seborrhoeic blepharitis and dermatitis on the cheeks



Scale and erythema due to seborrhoeic dermatitis on the glabella and brows



Flexural seborrhoeic dermatitis in the axilla



Confluent erythema and scale due to scalp seborrhoeic dermatitis



Presternal seborrhoeic dermatitis and pityrosporum folliculitis



Pigmented paranasal seborrhoeic dermatitis in skin of colour

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Who gets seborrhoeic dermatitis?

Seborrhoeic dermatitis is a common skin condition affecting 3% to 12% of the population.

It has a biphasic incidence occurring in infants and in adolescents and adults.

Infantile seborrhoeic dermatitis affects babies under the age of 3 months and usually resolves by 6-12 months of age.

Adult seborrhoeic dermatitis tends to begin in late adolescence. Prevalence is greatest in young adults and in older people. It is more common in males than in females.

Seborrhoeic dermatitis often occurs in otherwise healthy patients. However, the following factors are sometimes associated with severe adult seborrhoeic dermatitis:

Oily skin (seborrhea)

Familial tendency to seborrhoeic dermatitis or a family history of psoriasis

Immunosuppression: organ transplant recipients, human immunodeficiency virus ([HIV](#)) infection, and patients with lymphoma

Neurological and psychiatric diseases: Parkinson's disease, tardive dyskinesia, depression, epilepsy, facial nerve palsy, spinal cord injury, and congenital disorders such as [Down syndrome](#)

Use of neuroleptic medications

Treatment for psoriasis with [psoralen and ultraviolet A \(PUVA\) therapy](#)

Lack of sleep, and stressful events.

What causes seborrhoeic dermatitis?

The aetiology is not completely understood.

Several factors are associated with the condition e.g. hormone levels, fungal infections, nutritional deficits, neurogenic factors. Proliferation of *Malassezia* yeast genus is believed to play a role. The lipases and phospholipases produced by *Malassezia*, a saprophyte of normal skin, cleave free fatty acids from triglycerides present in sebum. This may induce inflammation. Differences in skin barrier lipid content and function may account for individual presentations.

What are the clinical features of seborrhoeic dermatitis?

Infantile seborrhoeic dermatitis

Infantile seborrhoeic dermatitis causes cradle cap (diffuse, greasy scaling on the scalp). The rash may spread to affect armpit and groin folds (a type of **napkin dermatitis**).

There are salmon-pink patches that may flake or peel.

It is not especially itchy, so the baby often appears undisturbed by the rash, even when generalised.



Infantile seborrhoeic dermatitis – note eczema in the napkin area and axillae and cradle cap



Thick yellow scale in cradle cap



Inflammatory infantile seborrhoeic dermatitis – note lesions in the body folds

Adult seborrhoeic dermatitis

Seborrheic dermatitis commonly affects areas of the skin with high **sebum** production, such as the scalp, nasolabial folds, glabella, eyebrows, beard, ears, retroauricular skin, sternum, and other skin folds.

Typical features include:

Winter flares, improving in summer following sun exposure

Minimal **itch** most of the time

Combination oily and dry mid-facial skin

Ill-defined localised scaly patches or diffuse scale in the scalp

Blepharitis: scaly red eyelid margins

Salmon-pink, thin, scaly, and ill-defined plaques in skin folds on both sides of the face

Petal or ring-shaped flaky patches on the hairline and on anterior chest

Rash in the armpits, under the breasts, in the groin folds, and genital creases

Malassezia folliculitis (inflamed hair follicles) on the cheeks and upper trunk.

Extensive seborrheic dermatitis affecting the scalp, neck, and trunk is sometimes called **pityriasisiform seborrhoeide**.

How do clinical features vary in differing types of skin?

Seborrheic dermatitis is very common among patients of darker skin types. Studies have shown that it is among the five most common diagnoses observed in black patients.

People of darker skin may present with scaly hypopigmented macules and patches in typical areas of involvement. Arcuate or petal-like patches may be seen, termed petaloid seborrhoeic dermatitis.

Children of colour often do not experience the classic cradle cap appearance of seborrheic dermatitis, but instead have [erythema](#), flaking, and [hypopigmentation](#) of the affected areas and folds of skin.



Seborrhoeic dermatitis around the hair line and forehead in skin of colour

What are the complications of seborrhoeic dermatitis?

Secondary [bacterial](#) or [fungal](#) infection

Skin thinning, dilated blood vessels, and [steroid-induced telangiectasia](#)

Psychosocial impact due to appearance of skin

How is seborrhoeic dermatitis diagnosed?

The diagnosis of seborrhoeic dermatitis is a clinical diagnosis based on the location, appearance, and behaviour of the lesions.

If the diagnosis is uncertain, a [biopsy](#) can be undertaken. This would typically show parakeratosis in the epidermis, plugged follicular ostia, and spongiosis in the case of seborrhoeic dermatitis. The dermis typically has a sparse, perivascular, lymphohistiocytic inflammatory infiltrate.

As *Malassezia* are a normal component of skin flora, their presence on microscopy of skin scrapings is not diagnostic.

For information on trichoscopic (dermoscopy) findings in seborrhoeic dermatitis, see [trichoscopy of inflammatory conditions](#).

What is the differential diagnosis for seborrhoeic dermatitis?

[Atopic dermatitis/eczema](#)

[Candidiasis](#)

[Contact dermatitis/eczema](#)

[Erythrasma](#)

[Impetigo](#)

[Lichen simplex chronicus](#)

Nummular dermatitis/eczema
Pityriasis rosea
Psoriasis
Rosacea
Scalp psoriasis
Secondary syphilis
Systemic lupus erythematosus
Tinea capitis, corporis.

What is the treatment for seborrhoeic dermatitis?

General measures

Educating the patient about the skin condition and appropriate skincare routine.
Identifying modifiable lifestyle factors e.g. a high fruit intake is associated with less seborrhoeic dermatitis whereas stress may precipitate flare-ups.

Specific measures

Treatment of seborrhoeic dermatitis often involves several of the following options.

Keratolytics: used to remove scale when necessary, e.g. [salicylic acid](#), lactic acid, [urea](#), propylene glycol.

Topical antifungal agents: applied to reduce *Malassezia* e.g. [ketoconazole](#), or ciclopirox shampoo and/or cream. Note, some strains of *Malassezia* are resistant to azole antifungals. Try zinc pyrithione or [selenium sulphide](#).

Mild [topical corticosteroids](#): for 1–3 weeks to reduce the inflammation of an acute flare.

Topical [calcineurin inhibitors](#): ([pimecrolimus cream](#), [tacrolimus ointment](#)) are indicated if topical corticosteroids are needing to be used frequently, as they have fewer adverse effects on facial skin with long term use.

In resistant cases in adults, [oral itraconazole](#), [tetracycline antibiotics](#), or [phototherapy](#) may be recommended. Low-dose [oral isotretinoin](#) has also been shown to be effective for severe or moderate disease.

[Roflumilast](#) 0.3% foam has had recent FDA approval for the use of seborrhoeic dermatitis in patients aged 9 years and older.

Scalp treatment

Medicated shampoos containing ketoconazole, ciclopirox, selenium sulfide, zinc pyrithione, [coal tar](#), and salicylic acid, used twice weekly for at least a month and if necessary, indefinitely.

[Steroid scalp applications](#) reduce itching and should be applied daily for a few days every so often.

[Calcineurin inhibitors](#) such as tacrolimus can be used as steroid alternatives.

[Coal tar](#) cream can be applied to scaling areas and removed several hours later by shampooing. Combination therapy is often advisable.

Alternative treatments, such as tea tree oil shampoo, may be used.

Face, ears, chest, and back

Cleanse the affected skin thoroughly once or twice each day using a non-soap cleanser.

Apply ketoconazole or ciclopirox cream once daily for 2 to 4 weeks, repeated as necessary.

Hydrocortisone cream can also be used, applied up to twice daily for 1 or 2 weeks. Occasionally a more potent topical steroid may be prescribed.

Topical calcineurin inhibitors such as pimecrolimus cream or tacrolimus ointment may be used instead of topical steroids.

A variety of herbal remedies are commonly used, but their efficacy is uncertain.

Management in infants

Regular washing of the scalp with baby shampoo or aqueous cream is followed by gentle brushing to clear the scales.

White petrolatum may be useful.

[Topical antifungal agents](#) are often prescribed, depending on the extent of the rash.

For more information, see [infantile seborrhoeic dermatitis](#).

What is the outcome for seborrhoeic dermatitis?

While seborrhoeic dermatitis may be self-limiting, it may take a long period of time to resolve. Cradle cap in infants usually takes a few weeks or months to disappear. In adults, the condition is frequently chronic and long-term maintenance treatment is often necessary.

[Click here for more images of seborrhoeic dermatitis](#)

[Diseases & Conditions](#)

Shingles

[Request an appointment](#)[Symptoms & causes](#)[Diagnosis & treatment](#)

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Overview

Shingles is a viral infection that causes a painful rash. Shingles can occur anywhere on your body. It typically looks like a single stripe of blisters that wraps around the left side or the right side of your torso.

Shingles is caused by the varicella-zoster virus — the same virus that causes chickenpox. After you've had chickenpox, the virus stays in your body for the rest of your life. Years later, the virus may reactivate as shingles.

Shingles isn't life-threatening. But it can be very painful. Vaccines can help lower the risk of shingles. Early treatment may shorten a shingles infection and lessen the chance of complications. The most common complication is postherpetic neuralgia. This is a painful condition that causes shingles pain for a long time after your blisters have cleared.

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Symptoms

Shingles symptoms usually affect only a small section on one side of your body. These symptoms may include:

- Pain, burning or tingling
- Sensitivity to touch
- A red rash that begins a few days after the pain
- Fluid-filled blisters that break open and crust over
- Itching

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- Fever
- Headache
- Sensitivity to light
- Fatigue

Pain is usually the first symptom of shingles. For some people, the pain can be intense. Depending on the location of the pain, it can sometimes be mistaken for problems with the heart, lungs or kidneys. Some people experience shingles pain without ever developing the rash.

Most commonly, the shingles rash develops as a stripe of blisters that wraps around either the left or right side of the torso. Sometimes the shingles rash occurs around one eye or on one side of the neck or face.



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Shingles

Shingles is characterized by pain or a tingling sensation in a limited area on one side of the face or torso, followed by a red rash with small, fluid-filled blisters.

When to see a doctor

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- The pain and rash occur near an eye. If left untreated, this infection may lead to permanent eye damage.
- You're 50 or older. Age increases your risk of complications.
- You or someone in your family has a weakened immune system. This may be due to cancer, medications or chronic illness.
- The rash is widespread and painful.

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Causes

Shingles is caused by the varicella-zoster virus — the same virus that causes chickenpox. Anyone who's had chickenpox may develop shingles. After you recover from chickenpox, the virus enters your nervous system and stays inactive for years.

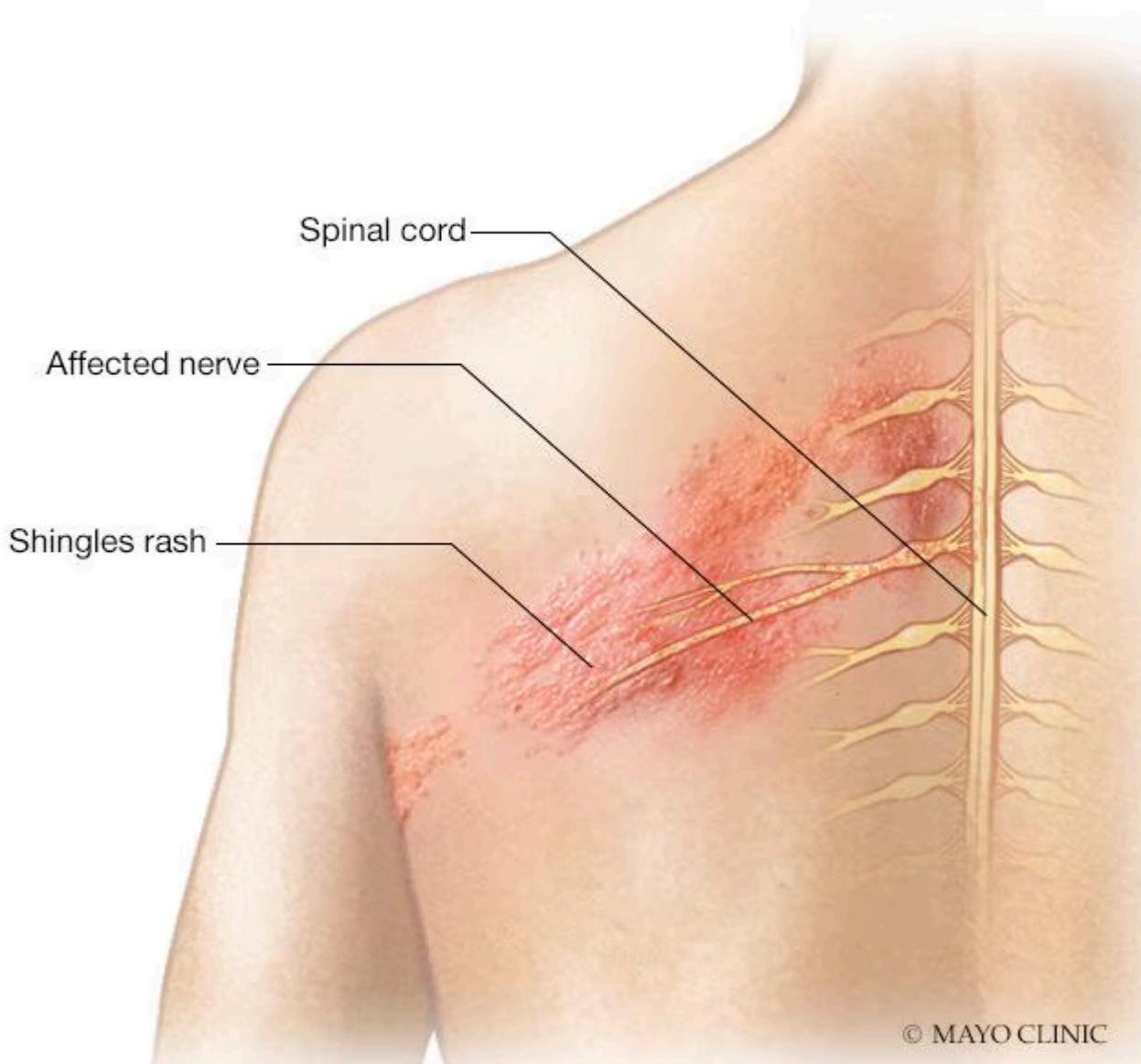
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The reason for shingles is unclear. It may be due to lowered immunity to infections as people get older. Shingles is more common in older adults and in people who have weakened immune systems.

Varicella-zoster is part of a group of viruses called herpes viruses. This is the same group that includes the viruses that cause cold sores and genital herpes. As a result, shingles is also known as herpes zoster. But the virus that causes chickenpox and shingles isn't the same virus that causes cold sores or genital herpes, which is a sexually transmitted infection.



Shingles affects the nerves

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Are you contagious?

A person with shingles can pass the varicella-zoster virus to anyone who isn't immune to chickenpox. This usually occurs through direct contact with the open sores of the shingles rash. Once infected, though, the person will develop chickenpox rather than shingles.

Chickenpox can be dangerous for some people. Until your shingles blisters scab over, you are contagious. Avoid physical contact with anyone who hasn't yet had chickenpox or the chickenpox vaccine. That includes people with weakened immune systems, pregnant women and newborns.

Risk factors

Anyone who has ever had chickenpox can develop shingles. Most adults in the United States had chickenpox when they were children. That was before the availability of the routine childhood vaccination that now protects against chickenpox.

Factors that may increase your risk of developing shingles include:

- **Age.** The risk of developing shingles increases with age. Shingles typically occurs in people older than 50. And people over the age of 60 are more likely to experience more-severe complications.
- **Some diseases.** Diseases that weaken your immune system, such as HIV/AIDS and cancer, can increase your risk of shingles.
- **Cancer treatments.** Radiation or chemotherapy can lower your resistance to diseases and may trigger shingles.
- **Some medications.** Drugs that prevent rejection of transplanted organs can increase your risk of shingles. Long-term use of steroids, such as prednisone, may also increase your risk of developing shingles.

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Complications from shingles can include:

- **Postherpetic neuralgia.** For some people, shingles pain continues long after the blisters have cleared. This condition is known as postherpetic neuralgia. It occurs when damaged nerve fibers send confused and exaggerated messages of pain from your skin to your brain.
 - **Vision loss.** Shingles in or around an eye (ophthalmic shingles) can cause painful eye infections that may result in vision loss.
 - **Neurological problems.** Shingles may cause inflammation of the brain (encephalitis), facial paralysis, or problems with hearing or balance.
 - **Skin infections.** If shingles blisters aren't properly treated, bacterial skin infections may develop.
-

Prevention

A shingles vaccine may help prevent shingles. People who are eligible should get the Shingrix vaccine, which has been available in the United States since its approval by the Food and Drug Administration in 2017. The Zostavax vaccine is no longer available in the U.S., but other countries may still use it.

Shingrix is approved and recommended for people age 50 and older, whether they've had shingles or not. People who've had the Zostavax vaccine in the past or don't know whether they've had chickenpox may also receive the Shingrix vaccine.

Shingrix is also recommended for people who are 19 years of age and older who have weakened immune systems due to disease or medication.

Shingrix is a nonliving vaccine made of a virus component. It's given in two doses, with 2 to 6 months between doses. The most common side effects of the shingles vaccine are redness, pain and swelling at the injection site. Some people also experience fatigue, headache and other side effects.

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lower your risk of postherpetic neuralgia. Studies suggest that Shingrix offers protection against shingles for more than five years.

Talk to your health care provider about your vaccination options if you:

- Have had an allergic reaction to any component of the shingles vaccine
- Have a weakened immune system due to a condition or medication
- Have had a stem cell transplant
- Are pregnant or trying to become pregnant

The shingles vaccine is used only as a way to prevent shingles. It's not intended to treat people who currently have the disease.

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By Mayo Clinic Staff

Aug 20, 2022

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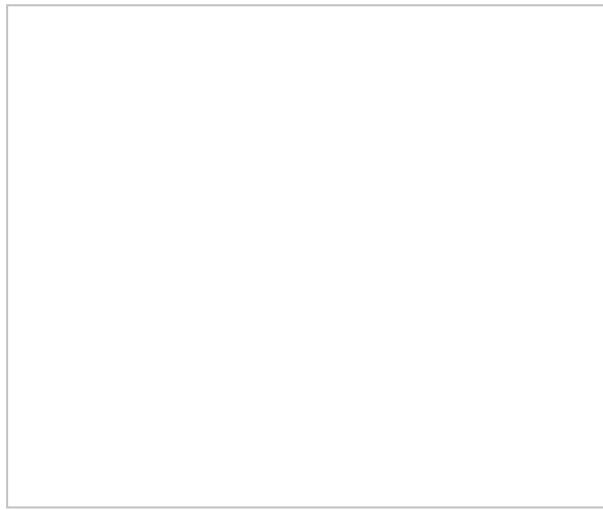
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Skin tag

Author: Dr Amanda Oakley MBChB FRACP, Dept of Dermatology Waikato Hospital, Hamilton, New Zealand, 2004.

What is a skin tag?

A skin tag is a common soft harmless lesion that appears to hang off the skin. It is also described as:

- Acrochordon
- Papilloma
- Fibroepithelial polyp
- Soft fibroma
- Pedunculated (this means it is on a stalk)
- Filiform (this means it is thread-like)

Skin tags develop in both men and women as they grow older. They are skin coloured or darker and range in size from 1mm to 5cm. They are most often found in the skin folds (neck, armpits, groin). They tend to be more numerous in obese persons and in those with type 2 diabetes mellitus.

Skin tags



Skin tags



Skin tag



Skin tag

Skin tags are made up of loosely arranged collagen fibres and blood vessels surrounded by a thickened or thinned-out epidermis.

[Seborrhoeic keratoses](#), [viral warts](#) or [molluscum contagiosum](#) may resemble skin tags.

Lesions resembling skin tags



Seborrhoeic keratosis



Seborrhoeic keratosis



Molluscum contagiosum

[See more images of skin tags.](#)

What causes skin tags?

It is not known what causes skin tags. However, the following factors may play a role:

- Chaffing and irritation from skin rubbing together
- High levels of growth factors, particularly during pregnancy or in [acromegaly](#) (gigantism)
- Insulin resistance (syndrome X)
- Human papillomavirus (wart virus)

How can they be removed?

Skin tags can be removed for cosmetic reasons by the following methods:

- [Cryotherapy](#) (freezing)
- [Surgical excision](#) (often with scissors)
- [Electrosurgery](#) (diathermy)
- Ligation (a suture is tied around the neck of the skin tag)

Sunburn

Author: Vanessa Ngan, Staff Writer, 2005.

What is sunburn?

Sunburn is erythema and oedema from excessive exposure to the sun's rays, more specifically the ultraviolet (UV) radiation emitted from the sun. Sunburn may also occur from exposure to other UV light sources such as in a [solarium](#) or tanning salon.

At a cellular level, sunburn is associated with microscopic changes in the skin. There is the formation of UV-induced sunburn cells and a reduction in Langerhans cells and mast cells, which play an essential part of the body's immune defence system.

What causes sunburn?

To better understand the causes of sunburn we need to take a look at some basic principles of the electromagnetic (light) spectrum. This spectrum is divided according to wavelength into the ultraviolet (< 400 nm), visible (400–760 nm), and infrared (> 760 nm). The ultraviolet (UV) spectrum is divided into three broad areas:

Ultraviolet A (UV-A) = 320–400 nm

Ultraviolet B (UV-B) = 290–320 nm

Ultraviolet C (UV-C) = < 290 nm.

UV-C radiation is filtered out or absorbed in the outer atmosphere so does not pose a problem to humans. UV-A and UV-B radiation are the primary causes of sunburn. The skin reacts differently to each waveband.

Reactions to UV-A and UV-B radiation

UV-A	UV-B
<p>Less potent than UV-B but is the wavelength that reaches the surface of the earth most (about 90% at midday)</p> <p>Penetrates the middle skin layer (dermis) and subcutaneous fat causing damage to the site where new skin cells are created</p> <p>Long-term exposure causes injury to the dermis resulting in ageing skin</p>	<p>Much more potent at causing erythema</p> <p>About 90% is absorbed by the surface skin layer (epidermis)</p> <p>Epidermis responds by releasing chemicals that cause the reddening and swelling characteristic of the early signs of sunburn</p> <p>Repeated exposure causes injury to the epidermis resulting in ageing skin</p>

Who is at risk of sunburn?

Skin phototyping categorises people into one of six groups based on baseline skin colour and the tendency to tan and burn when exposed to UV radiation.

Skin Phototype	Typical Features	Tanning ability	MED (mJ/cm ²)
I	Pale white skin, blue/hazel eyes, blond/red hair	Always burn do not tan	15-30
II	Fair skin, blue eyes	Burn easily, tan poorly	25-40
III	Darker white skin	Tan after the initial burn	30-50
IV	Light brown skin	Burn minimally, tan easily	40-60
V	Brown skin	Rarely burn, tan darkly easily	60-90
VI	Dark brown or black skin	Never burn always tan darkly	90-150

People with type I skin phototyping are at much greater risk of sunburn than their type VI counterparts. The amount of UV radiation, measured in energy per unit area, to produce erythema at an exposed site is called the minimal erythema dose (MED), and this is significantly lower in people with a low skin phototype grading. Fifteen minutes of midday sun exposure may cause sunburn in a white skin person, while a darker skinned person may tolerate the exposure for hours.

Other factors that increase the incidence of sunburn include:

Regions situated closer to the equator

Areas at high altitude – UV radiation increases 4% for every 300m increase in elevation

Skin exposure between 10 am, and 2 pm – 65% of UV radiation reaches the earth between these times

Clear skies: clouds and environmental pollution reduce UV radiation

Environmental reflection – UV radiation is 80% reflected by snow and ice

What are the clinical features of sunburn?

The signs and symptoms of sunburn differ according to the skin phototype and length of exposure to UV radiation.

Signs and symptoms usually occur 2-6 hours after exposure and peak at 12-24 hours; they may include:

Erythema (redness)

Oedema (swelling)

Tenderness and irritation

Skin feels hot to touch

Pain

Blistering (severe cases)

Chills and fever (severe cases)

In severe cases of sunburn, severe skin burning may result in second-degree burns, dehydration, electrolyte imbalances, secondary infection, shock or even death.



Type 2



Around 4-7 days after exposure skin may start to peel and flake off.



Peeling after sunburn



Peeling after sunburn



Peeling after sunburn

What is the treatment of sunburn?

The treatment of sunburn is to provide relief of the discomfort it can cause with the use of analgesics (pain-killers), cool baths, [aloe vera](#) lotions and moisturisers.

However, sunburn is better prevented than treated. [Sun protection](#) is your best defence against sunburn and other damaging effects of UV radiation.

Avoid sun exposure, especially between 10 am to 2 pm

Wear [protective clothing](#), including wide-brimmed hats

Apply and re-apply [sunscreen](#) with a Sun Protection Factor (SPF) of 50+

An oral food supplement containing [Polypodium leucotomas](#) may provide additional oral photoprotection and reduce sunburn.

If you are inadvertently exposed and expect to be sunburned, you may lessen the severity of the burn with the following measures:

Take two aspirin immediately and then two every four hours

Apply a topical steroid to exposed areas twice daily for two or three days

What are the long-term consequences?

It is now clear the long-term consequences of overexposure to the sun or other sources of UV radiation are significant. One blistering sunburn at least doubles the likelihood of developing skin cancer later.

Premature [skin ageing](#) and [wrinkling](#)

[Brown spots and freckles](#) ([lentigines](#))

Development of premalignant lesions ([actinic keratoses](#))

Development of [skin cancer](#) (eg, [melanoma](#), [basal cell carcinoma](#), [squamous cell carcinoma](#))

Tinea corporis

Created 2003. Updated: Dr Harriet Bell, House Officer, Auckland City Hospital, Auckland, New Zealand. Copy edited by Gus Mitchell. November 2020.

What is tinea corporis?

Tinea corporis is a superficial fungal infection of the skin that can affect any part of the body, excluding the hands and feet, scalp, face and beard, groin, and nails. It is commonly called 'ringworm' as it presents with characteristic ring-shaped lesions.

Typical annular lesions of ringworm



Tinea corporis



Who gets tinea corporis?

Tinea corporis is found in most parts of the world, but particularly in hot humid climates. It is most commonly seen in children and young adults, however all age groups can be infected including newborns.

Medical risk factors include:

Previous or concurrent tinea infection
Diabetes mellitus
Immunodeficiency
Hyperhidrosis
Xerosis
Ichthyosis.

Environmental risk factors include:

Household crowding
Infection of household members
Keeping house pets
Wearing occlusive clothing
Recreational activities involving close contact with others including shared change rooms.

A genetic predisposition has been postulated, particularly for tinea imbricata.

What causes tinea corporis?

Tinea corporis is predominantly caused by dermatophyte fungi of the genera *Trichophyton* and *Microsporum*. The anthropophilic species *T. rubrum* is the most common causative agent of tinea corporis worldwide including New Zealand. Other species that may cause tinea corporis include:

T. interdigitale
T. tonsurans – secondary to tinea capitis or skin-to-skin contact
M. canis (cats, dogs), and less commonly other zoonotic species including *T. verrucosum* (cattle),
T. equinum (horses) and *T. erinacei* (hedgehogs).

Tinea corporis is spread by the shedding of fungal spores from infected skin. Transmission is facilitated by a warm, moist environment and the sharing of fomites including bedding, towels, and clothing. Dermatophyte infection elsewhere on the skin, such as tinea pedis, can also be transferred. The incubation period is 1–3 weeks. The dermatophyte invades and spreads in the stratum corneum, but is unable to penetrate deeper layers in healthy skin.

What are the clinical features of tinea corporis?

Tinea corporis initially presents as a solitary circular red patch with a raised scaly leading edge. A lesion spreads out from the centre forming a ring-shape with central hypopigmentation and a peripheral scaly red rim (ringworm). The border can be papular or pustular. Itch is common. With time, multiple lesions can develop which may coalesce to form a polycyclic pattern. The distribution of lesions is typically asymmetrical.

Sharp red scaly margin of tinea corporis

Papules and pustules of tinea corporis

Asymmetry of tinea corporis

See also [Tinea corporis images](#)

Clinical variants of tinea corporis

Clinical variants of tinea corporis can include the following types.

Kerion – an intense pustular inflammatory reaction due to zoophilic fungi.

Tinea gladiatorum – affects participants in contact sports such as wrestling or martial arts due to skin-to-skin contact. It is usually caused by *T. tonsurans*.

Tinea imbricata – extensive concentric rings forming polycyclic plaques with thick scale due to *T. concentricum*. It is particularly itchy. This variant is common in the Pacific Islands and other equatorial tropical areas.

Tinea incognito – lacks the typical features of tinea corporis due to suppression of the inflammatory reaction following the topical application of corticosteroids or calcineurin inhibitors. Lesions tend to be widespread with poorly defined borders lacking scale and erythema.

Majocchi granuloma – a variant involving the hair follicles and subcutaneous tissue, most commonly found on the limbs after shaving. It presents as perifollicular papules or pustules. *T. rubrum* is the usual organism.

Bullous tinea corporis – a rare variant presenting with vesicles or blisters.

Clinical variants of tinea corporis

Kerion – inflammatory tinea corporis

Majocchi granuloma – invasive tinea corporis

Tinea incognito

What are the complications of tinea corporis?

Tinea corporis is contagious, spreading elsewhere on the skin and to others.

Immunosuppressed patients, such as those with [HIV/AIDS](#), can present with disseminated infection.

Chronic dermatophytosis is *T. rubrum* infection of at least four body sites following a prolonged fluctuating course and recurrence despite treatment.

[Dermatophytide reactions](#) are an allergic rash at a distant site caused by an inflammatory fungal infection.

Secondary [bacterial infection](#) with *Staph aureus* is common in children with [atopic dermatitis](#) and tinea corporis.

How is tinea corporis diagnosed?

Tinea corporis should be considered in the setting of a solitary patch or asymmetrical rash and confirmed on mycology to determine the likely source.

Examination should include the entire skin surface, including the scalp and nails, for other sites of involvement.

[Dermoscopy](#) may assist the clinical diagnosis with features of erythema, white scaling, peripheral or patchy distribution of blood vessels, tiny follicular papules, brown spots surrounded by white-yellow rings, and wavy or broken hairs.

Skin scrapings taken from the scaly lesion edge and mounted in 10–20% potassium hydroxide can be examined under a light microscope for hyphae and spores. Fungal culture of the skin scrapings provides identification of the causative organism (see [Laboratory tests for fungal infection](#)).

Tinea corporis is sometimes diagnosed on [skin biopsy](#), and characteristic histology changes are seen (see [Tinea corporis pathology](#)). Histology is typically required for the diagnosis of Majocchi granuloma.

What is the differential diagnosis for tinea corporis?

The differential diagnosis for tinea corporis can include:

[Discoid eczema](#)

[Psoriasis](#)

[Pityriasis rosea](#) herald patch

[Seborrhoeic dermatitis](#).

What is the treatment for tinea corporis?

General measures

Skin should be kept clean and dried thoroughly. Loose-fitting light clothing is recommended in hot humid climates. Avoid close contact with infected individuals and the sharing of fomites. Examination of household members and pets for the source of infection and appropriate treatment reduces the risk of re-infection.

Specific measures

Localised tinea corporis may respond to [topical antifungal medications](#) such as imidazoles and [terbinafine](#). Application needs to include an adequate margin around the lesion and a prolonged course continuing for at least 1–2 weeks after the visible rash has cleared. However, recurrence is common.

[Oral antifungal treatment](#) is usually required if tinea corporis is involving a hair-bearing site, is extensive, or has failed to clear with topical antifungals. Systemic therapy is also required for Majocchi granuloma and tinea imbricate. Recommended oral agents are [terbinafine](#) and [itraconazole](#).

What is the outcome for tinea corporis?

With appropriate treatment and good patient compliance, tinea corporis can be cured. However, recurrence or re-infection can occur if treatment has stopped too soon, or the source of infection has not been identified and treated.

Tinea pedis

Original page created in 2003. Updated by Dr Thomas Stewart, General Practitioner, Sydney, Australia. DermNet Editor in Chief: Adjunct A/Prof Amanda Oakley, Dermatologist, Hamilton, New Zealand. Copy edited by Gus Mitchell. April 2018.

What is tinea pedis?

Tinea pedis is a foot infection due to a [dermatophyte](#) fungus. It is the most common dermatophyte infection and is particularly prevalent in hot, tropical, urban environments.

Interdigital involvement is most commonly seen (this presentation is also known as [athlete's foot](#), although some people use the term for any kind of tinea pedis).

Tinea pedis may be accompanied by [tinea cruris](#), [tinea manuum](#) or [tinea unguium](#).

What causes tinea pedis?

The three most common [dermatophyte fungi](#) causing tinea pedis are:

Trichophyton (T.) rubrum

T. interdigitale, previously called *T. mentagrophytes* var. *interdigitale*

Epidermophyton floccosum



Moccasin tinea pedis



Tinea pedis



Tinea pedis



Tinea pedis



Tinea pedis



Tinea pedis

[See more images of tinea pedis ...](#)

Who gets tinea pedis?

Tinea pedis usually occurs in males and adolescents/young adults, but can also affect females, children and older people. Infection is usually acquired by direct contact with the causative organism, for example using a shared towel, or by walking barefoot in a public change room.

Other risk factors include:

- Occlusive footwear (for example, heavy industrial boots)
- Excessive sweating ([hyperhidrosis](#))
- Underlying [immunodeficiency](#) or [diabetes mellitus](#)
- [Systemic corticosteroids](#) or [immune suppressive medications](#)
- Poor peripheral circulation or [lymphoedema](#).

What are the clinical features of tinea pedis?

Tinea pedis tends to be asymmetrical, and may be unilateral. It usually presents in one of three ways:

- Itchy erosions and/or scales between the toes, especially between 4th and 5th toes
- Scale covering the sole and sides of the feet (hyperkeratotic/moccasin type, usually caused by *T. rubrum*)
- Small to medium-sized blisters, usually affecting the inner aspect of the foot (vesiculobullous type).

It can also uncommonly cause oozing and ulceration between the toes (ulcerative type), or pustules (these are more common in tinea pedis due to *T. interdigitale* than that due to *T. rubrum*).

The diagnosis of tinea pedis can be made clinically in most cases, based on the characteristic clinical features. Other typical sites, such as toenails, groin, and palms of the hands, should be examined for fungal infection, which may support a diagnosis of tinea pedis.

Diagnosis is confirmed by skin scrapings, which are sent for microscopy in potassium hydroxide (when segmented hyphae may be observed) and culture ([mycology](#)). Culture may not be necessary if typical fungal elements are observed on microscopy.

What is the differential diagnosis of tinea pedis?

The differential diagnosis of tinea pedis includes:

- Foot eczema — especially [pompholyx](#) (pedopompholyx), or [irritant contact dermatitis](#) due to persistent moisture between closely adherent toes
- [Contact allergic dermatitis](#) to a component of footwear (such as a [rubber accelerant](#), [shoe adhesive](#), [potassium dichromate](#) used as leather tanning agent, or [fabric dye](#))
- [Psoriasis](#) ([plantar psoriasis](#))
- [Plantar pustulosis](#)
- [Plantar keratoderma](#).

These inflammatory disorders are more likely to be symmetrical and bilateral. [Mycology](#) is negative.

What is the treatment for tinea pedis?

General measures should be first-line, including meticulous drying of feet, especially between the toes, avoidance of occlusive footwear, and the use of barrier protection (sandals) in communal facilities.

[Topical antifungal therapy](#) once or twice daily is usually sufficient. These include azoles, allylamines, butenafine, ciclopirox, and tolnaftate. A typical course is 2 to 4 weeks, but single dose regimes can be successful for mild infection [1,2].

For those who do not respond to topical therapy, an [oral antifungal agent](#) may be needed for a few weeks. These include:

- [Terbinafine](#)
- [Itraconazole](#)
- [Fluconazole](#)
- [Griseofulvin](#) (this may be inferior to other oral agents and may not be available in some countries) [3,4].

Patients with the hyperkeratotic variant of tinea pedis may benefit from the addition of a topical keratolytic cream containing [salicylic acid](#) or [urea](#) [5].

How can recurrence of tinea pedis be prevented?

To minimise recurrence of tinea pedis:

- Dry feet and toes meticulously after bathing
- Use desiccating foot powder once or twice daily
- Avoid wearing occlusive footwear for long periods
- Thoroughly dry shoes and boots
- Clean the shower and bathroom floors using a product containing bleach
- Treat shoes with antifungal powder.

If treatment of tinea pedis is unsuccessful, consider reinfection, coexistent untreated [fungal nail infection](#), reinfection due to untreated family member, or an alternative diagnosis.

Trachyonychia

Last reviewed: September 2023

Author(s): Szu-Wen Chen, The University of Auckland; and Honorary Associate Professor Paul Jarrett, Dermatologist, Middlemore Hospital and Department of Medicine, The University of Auckland, Auckland, New Zealand (2023)

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Edited by the DermNet content department

What is trachyonychia?

Trachyonychia is characterised by brittle nails that show diffuse longitudinal ridging and can be accompanied by pitting, loss of lustre, or a roughened nail plate.

Trachyonychia, also known as 'rough nails' or 'sandpaper nails', can involve any number of nails. Twenty-nail dystrophy refers to trachyonychia that affects all 20 nails.



Opaque trachyonychia – there is opacity and longitudinal ridging



Shiny trachyonychia – the plate is bright and shiny



Trachyonychia due to 20 nail dystrophy – all of the nails were affected

[More images](#)

Who gets trachyonychia?

Trachyonychia can occur in individuals of all ages, but is predominantly seen in children and young adults, with the peak incidence occurring between 3–12 years of age. It has been reported in both males and females, without a significant gender predilection.

Rare familial cases are described (autosomal dominant) and in identical twins.

What causes trachyonychia?

The pathogenesis of trachyonychia remains unclear. It is hypothesised that inflammation within the nail matrix is involved in the development of the disease, and the extent of inflammatory activity is believed to influence disease severity.

Lichen planus

Psoriasis

Ichthyosis vulgaris

Atopic dermatitis

Vitiligo.

What are the clinical features of trachyonychia?

Trachyonychia can be classified into two subtypes based on clinical presentation and severity.

Opaque trachyonychia:

More severe and more common

Associated with persistent and diffuse inflammation in nail matrix

Thin, brittle, and 'sandpaper-like' appearance, characterised by prominent longitudinal ridging and frequent [onychoschizia](#) (nail splitting)

Hyperkeratotic, irregular cuticles with superficial scaling.

Shiny trachyonychia:

Mild manifestation with multifocal and intermittent inflammation in the nail matrix

Nail plates maintain lustre, presenting with numerous small geometric pits arranged in a pattern that creates longitudinal ridges and reflects light.

In both opaque and shiny trachyonychia, koilonychia (spoon-shaped nails) can be observed, and fingernails are more commonly involved than toenails.

In twenty-nail dystrophy, all nails are affected.

The severity of the condition can vary between different nails within the same patient, and it is possible for both opaque and shiny varieties to coexist.

[Click here for images](#)

How do clinical features vary in differing types of skin?

There are no variations in the clinical features of trachyonychia between patients with different skin colours.

What are the complications of trachyonychia?

Psychological distress and reduced self-esteem due to nail changes.

Difficulty with daily function.

How is trachyonychia diagnosed?

Trachyonychia is primarily a clinical diagnosis based on history, full skin examination, and characteristic nail changes. However, some tests can assist in the diagnosis.

Dermoscopy — distinctive changes include scaling, longitudinal ridging, pitting, involvement of proximal nail plate, involvement of >50% of the proximal nail plate width, onychoschizia, and thickened and ragged cuticles.

Nail clippings — exclusion of [fungal nail infection](#) or associated disorders.

Nail unit biopsy — not routinely performed. Spongiosis of the nail unit is a typical histological finding in trachyonychia.

What is the differential diagnosis for trachyonychia?

[Onychomycosis](#)

[Alopecia areata](#)

[Nail psoriasis](#)

[Lichen planus](#)

What is the treatment for trachyonychia?

Treatment may not always be necessary as trachyonychia can improve or resolve spontaneously over time in many patients.

General measures

Observation, reassurance, counselling, and active non-intervention.

Emollients in opaque trachyonychia to improve the nail surface texture.

Camouflage with nail polish in shiny trachyonychia to improve appearance.

Specific measures

There is a lack of gold-standard evidence-based approaches due to limited data. Numerous treatment modalities have been trialled in small numbers of patients and responses vary.

Topical therapy:

[Corticosteroids](#)

[Calcipotriol/betamethasone dipropionate ointment](#)

[Fluocinonide/bifonazole cream](#)

[Retinoids](#) (eg, tarazotene gel)

[Keratolytic agents](#) (eg, urea cream)

[Chemotherapy](#) (eg, 5-[fluorouracil](#) 5% cream)

[PUVA](#)

Weekly nail plate dressings once a week with agents such as lactic acid, silicon dioxide, or aluminium acetylacetone.

Intralesional therapy:

[Triamcinolone](#).

Systemic therapy:

It is not usually justified to exhibit systemic therapy as most patients find this a cosmetic concern only, but if it is severe enough to have functional effects, the agents below have been advocated.

[Corticosteroids](#)

[Oral retinoids](#)

[Ciclosporin](#)

[Biotin](#)

[Hydroxychloroquine](#)

[Janus kinase \(JAK\) inhibitors \(eg, oral tofacitinib\)](#).

How do you prevent trachyonychia?

There are no specific preventive measures known for trachyonychia.

What is the outcome for trachyonychia?

Trachyonychia is a benign but chronic condition. Resolving nail abnormalities without treatment can be a gradual process, often spanning several years.

[Click here for images](#)



Types of Eczema

Eczema is the name for a group of inflammatory skin conditions. There are seven different types of eczema. Learn about the causes, symptoms and treatments for each form of eczema.

What are the different types of eczema?

Eczema is the name for a group of inflammatory skin conditions that cause skin to become dry, itchy, flaky and bumpy. There are seven different types of eczema:

- [Atopic dermatitis](#)
- [Contact dermatitis](#)
- [Dyshidrotic eczema](#)
- [Neurodermatitis](#)
- [Nummular eczema](#)
- [Seborrheic dermatitis](#)
- [Stasis dermatitis](#)

While the exact cause of eczema is unknown, researchers do know that people who develop eczema do so because of a combination of genes and environmental triggers. Some people with eczema have a mutation of the gene responsible for creating filaggrin. Filaggrin is a protein that helps our bodies maintain a healthy, protective barrier on the very top layer of the skin. Without enough filaggrin to build a strong skin barrier, moisture can escape and bacteria, viruses and more can enter.

Can you have more than one type of eczema?

It's possible to experience more than one type of eczema at the same time or at different times on multiple parts of the body. For effective diagnosis and management, it's crucial to know what type you are experiencing. Dermatologists can help identify which type or types of eczema you may have and how to treat and prevent eczema flares of all types.

What's the difference between each type of eczema?

Depending on the type of eczema, there may be different causes, symptoms and treatment. Below is an overview of each type of eczema.

Basics of atopic dermatitis

Atopic dermatitis is the most common and chronic form of eczema, often starting in infancy or early childhood, but it can persist into adulthood or develop later in life. It is characterized by dry and intensely itchy patches of skin that may crack, ooze or form crusts. Atopic dermatitis can appear as red, gray, brown or purplish depending on skin tone. Commonly affected areas include the face, hands, inside the elbows and behind the knees, although it can appear anywhere on the body.

Atopic dermatitis often occurs alongside asthma and allergic rhinitis (also known as "hay fever"), and it can run in families. Atopic dermatitis is thought to result from a combination of genetic, environmental and immune system factors. In people with atopic dermatitis, the skin barrier is weakened, making it more susceptible to dryness, irritants, allergens and microbes. Symptoms can be triggered by allergens, harsh soaps, cold weather or stress, among other

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be cured, treatment focuses on managing symptoms and minimizing flares through routine moisturizing, anti-inflammatory medications and lifestyle adjustments.

[Get more details on symptoms, diagnosis and treatment for atopic dermatitis](#)

Basics of contact dermatitis

Contact dermatitis develops when the skin comes into direct contact with a substance that causes irritation or an allergic reaction. Irritant contact dermatitis occurs when substances like soaps or chemicals cause direct damage to the skin, while allergic contact dermatitis is an immune reaction on the skin triggered by allergens like nickel or poison ivy.

Symptoms include redness, swelling, itching and sometimes blisters. The reaction is usually limited to the area of skin that comes in contact with the irritant or allergen, but it can be severe if exposure continues.

This condition can affect anyone. It can be occupational, meaning it occurs in people exposed to allergens or irritants at work. Once the trigger is identified, avoiding the substance is critical to prevent further reactions.

[Get more details on symptoms, diagnosis and treatment for contact dermatitis](#)

See Photos of Eczema

Eczema can vary in appearance for many reasons including type, level of severity, affected part of the body and, notably, skin tone. The Eczema Visual Guide is the largest publicly-available repository of images showcasing the various forms of eczema across all skin tones.

[View photos](#)

Basics of dyshidrotic eczema

Dyshidrotic eczema is a condition characterized by the sudden appearance of small, intensely itchy blisters on the sides of the fingers, toes, palms or soles of the feet. These blisters can cause significant discomfort and may be filled with fluid. Over time, the affected skin may crack, peel or thicken, especially if the condition becomes chronic. Triggers include stress, exposure to certain metals (like nickel or cobalt), seasonal allergies or excessive moisture on the hands and feet due to an improper immune response and improper sweating mechanisms occurring in people with dyshidrotic eczema.

This form of eczema is more common in older children and adults and tends to occur in cycles, with flares lasting for weeks before subsiding. The symptoms can be managed with topical steroids, antihistamines or lifestyle modifications like wearing gloves or avoiding irritants.

[Get more details on symptoms, diagnosis and treatment for dyshidrotic eczema](#)

Basics of neurodermatitis

Neurodermatitis is characterized by intense itching. It often starts with a patch of itchy skin that becomes more irritated when scratched, leading to a cycle of itching and scratching. It can result in thick, leathery patches of skin and can be triggered by stress.

[Get more details on symptoms, diagnosis and treatment for neurodermatitis](#)

7 types of eczema explained by a dermatologist

Introducing the 7 Types of Eczema (and What to do ...)



In this webinar, [Dr. Eric Simpson](#), a board-certified dermatologist and professor of Dermatology at the School of Medicine at Oregon Health & Science University, explains the difference between the seven types of eczema. He explains how to recognize them, possible triggers and how to treat them.

Basics of nummular eczema

Nummular eczema, also known as nummular dermatitis or discoid eczema, is characterized by coin-shaped spots of irritated, itchy skin. These spots can become crusty or scaly. It is often mistaken for ringworm due to their similarities in appearance.

[Get more details on symptoms, diagnosis and treatment for nummular eczema](#)

Basics of seborrheic dermatitis

Seborrheic dermatitis is a chronic form of eczema that affects oily areas of the body, such as the scalp, face (around the nose and eyebrows) and/or upper and central chest. It causes red, greasy and inflamed skin covered with white or yellowish scales. In adults, it is commonly referred to as dandruff when confined to the scalp, and in infants, it is called "cradle cap." The condition can be recurrent, with flares often triggered by stress, cold weather, hygiene practices or hormonal changes.

Treatment often includes medicated shampoos, topical antifungal or corticosteroid creams and maintaining a regular skincare routine to control symptoms. In severe cases, other prescription medications may be necessary.

[Get more details on symptoms, diagnosis and treatment for seborrheic dermatitis](#)

Basics of stasis dermatitis

Stasis dermatitis develops due to poor circulation in the lower legs, often as a result of chronic venous insufficiency, where blood pools in the veins instead of flowing back to the heart. The condition typically presents with swelling, redness and itching, with the skin becoming dry, scaly and discolored over time. In severe cases, open sores or ulcers may form, increasing the risk of infection.

This condition is most common in older adults and those with underlying vein issues, such as varicose veins or a history of blood clots. Treatment focuses on improving circulation through compression stockings, physical movements, elevating the legs and managing inflammation with topical treatments. Long-term management is crucial to prevent complications like ulcers or skin infections.

[Get more details on symptoms, diagnosis and treatment for stasis dermatitis](#)

Related Stories

[How to Tell the Difference Between Nummular Eczema and Ringworm](#)

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Vitiligo

Author: Dr Bushra Alsayaydeh, Dermatologist, Amman, Jordan, August 2022. Previously: A/Prof Amanda Oakley, Dermatologist, Hamilton, New Zealand, 1999.

What is vitiligo?

Vitiligo is an acquired, chronic, depigmenting disorder of the skin, in which pigment-producing cells (melanocytes) that determine the colour of skin, hair, and eyes are progressively lost. It appears as milky-white patches of skin ([leukoderma](#)) and can be cosmetically very disabling, particularly in people with dark skin.

It is currently widely accepted that vitiligo is the result of autoimmune destruction of melanocytes.



Vitiligo over the knuckles – koebnerisation due to trauma often localises vitiligo



Symmetrical wrist vitiligo – a common location



Vitiligo over the back and elbows



Vitiligo on the lid with poliosis of the lashes



Vitiligo on the lips



Follicular repigmentation in a patch of vitiligo



Extensive symmetrical facial vitiligo



Vitiligo around the hairline of a Samoan woman

[Click here for more images of vitiligo](#)

Who gets vitiligo?

Vitiligo affects 0.5–2% of the population.

Race: relatively consistent incidence in all races, but appears to be:

Less common in the Han Chinese population

More common in India (up to 8.8% of the population).

Sex: both men and women appear to be equally affected

Women tend to constitute a higher percentage of overall outpatient visits, due to greater concerns about cosmetic appearance.

Onset: the average age of onset is between 20–24 years, but can occur at any age. Typically, there are two peaks of onset, early (<10 years) or late (around 30 years).

41% of segmental vitiligo cases start before the age of 10.

50% of non-segmental vitiligo cases start before the age of 20.

80% of all cases present before the age of 30.

Inheritance: follows a polygenic pattern, with a 23% monozygotic twin concordance, supporting the cause of vitiligo to be multifactorial (genetic and non-genetic environmental factors).

More than 20–30% of the affected individuals report vitiligo in a first or second-degree relative.

Autoimmune disease development has been associated with generalized vitiligo, the most common type of vitiligo, especially if there is a family history of vitiligo and other autoimmune disorders.

The strongest association is with [thyroid disease](#), which can affect up to 15% of adults and 5–10% of children with vitiligo.

Other less frequently associated autoimmune disorders with vitiligo are:

[Rheumatoid arthritis](#)

[Insulin-dependent diabetes mellitus](#) (mostly adult-onset)

Pernicious anaemia (B12 deficiency)

Addison disease

Systemic lupus erythematosus

Alopecia areata

Other autoimmune dermatologic conditions, eg, [psoriasis](#) and [lichen sclerosus](#).

Vitiligo is also three times more common in recipients of allogeneic bone marrow and stem-cell transplants than in the healthy population.

What causes vitiligo?

Vitiligo is due to the loss or destruction of melanocytes (melanin-producing cells).

Genetic factors appear to contribute to 80% of vitiligo risk, whilst environmental factors account for 20%. Many genetic loci have been identified, all related to the immune system, except for TYR which encodes tyrosinase, a key enzyme in melanin production and a major autoantigen in vitiligo.

The convergence or integrated theory combines immunological, biochemical, oxidative, and environmental mechanisms that work jointly in those with a genetic susceptibility is widely accepted.

This could be explained through three phases:

Initial phase: less adhesive melanocytes are prone to internal and external oxidative stresses, leading to the production of more toxic reactive oxygen species (ROS).

Progression phase: an imbalance between ROS and antioxidants will activate the adaptive immune system bridged by the innate immune system.

CD8+ cytotoxic cells release cytokines, mainly interferon- γ (IFN- γ) that activate the JAK-STAT pathway through its receptors on keratinocytes. This will lead to the production of chemokines (CXC), predominantly by keratinocytes, but also by melanocytes themselves, leading to IFN- γ -CXCR3- CXCL9/10 axis loop feedback.

Acting together on their common CXCR3 receptor, CXCL9 drives the main bulk of CD8+ cell homing, while CXCL10 promotes their localisation to affected skin lesions and induction of melanocyte apoptosis through CXC3B activation. Of note, where both humoral (antibody) and T-cell responses appear to be implicated, antibody titres do not correlate with disease activity nor the localisation of distinct vitiligo lesions.

Maintenance phase: established lesions are maintained by resident melanocyte reactive T-cells (TRM cells), through the IL15-dependent pathway. These TRM cells may be responsible for what is called an 'autoimmune memory', in which relapses occur mostly at the same exact site of previous lesions.

Understanding the molecular pathogenesis of vitiligo serves as a promising source for the development of more targeted therapies.

Vitiligo and other conditions

Vitiligo is also a component of some rare multiorgan syndromes, such as:

[Vogt-Koyanagi-Harada syndrome](#)

[Alezzandrini syndrome](#).

These syndromes affect organs that normally house melanocytes, and are now believed to constitute one disease entity with variable clinical expression.

Other rare dermatologic syndromes that present with lesions indistinguishable from vitiligo are:

[APECED \(APS1\) syndrome](#)

Schmidt's (APS2) syndrome.

A vitiligo-like leukoderma may occur in patients with [metastatic melanoma](#).

Vitiligo can also be induced by drugs, such as immune checkpoint inhibitors ([pembrolizumab](#), [nivolumab](#)) and BRAF inhibitors ([vemurafenib](#), [dabrafenib](#)) used to treat [metastatic melanoma](#).

What are the clinical features of vitiligo?

The onset of vitiligo is usually insidious.

The most common presentation is the complete loss of pigment in single or multiple macules or patches of skin, with characteristic chalk- or milky-white colour.

Lesion characteristics

Typically asymptomatic, but rarely pruritic in active lesions.

Multiple macules are sometimes described as confetti-like.

Well-defined with convex borders; the borders may sometimes either be:

- The colour of unaffected skin

- Hyperpigmented or hypopigmented

- Inflamed and red.

Sometimes smaller patches coalesce, merging into more complex shapes.

Location

Can be found on any part of the body, although more common in sun-exposed areas or areas more prone to repetitive trauma (eg, eyelids, lips, nostrils, fingertips, and toes), body folds (armpits, groin, navel), and nipples. Interestingly, these areas are naturally more pigmented.

Vitiligo also favours sites of injury; this is called the isomorphic [Koebner phenomenon](#). Injury can be induced by either:

- Physical (cuts, abrasions, scratching)

- Mechanical (friction, chronic pressure, eg, eye rubbing, lip-licking, watches, tight-fitting clothes)

- Burns (chemical, sunburn)

- Inflammation (psoriasis, herpes zoster, dermatitis)

- Therapeutic (phototherapy, radiotherapy).

Vitiligo can occur in less pigmented areas of skin that can be often overlooked especially in light-skinned patients, eg, the palms and soles, and oral mucosa.

Precipitating factors and other features

Emotional stress, pregnancy, oral contraceptives, vitamin deficiencies, and many other factors have been described as precipitating factors for vitiligo; however, correlation is still not proven.

Vitiligo may occasionally start as multiple halo naevi.

- Found in up to 31% of cases.

- Halo naevi can precede or coincide with vitiligo.

Loss of hair colour, (leukotrichia or poliosis), may affect the scalp, eyebrows, eyelashes, and body hair in 10–60% of patients. It does not correlate with disease activity, but could be a predictor of poorer response to therapy due to the destruction of melanocyte reservoir in hair follicles.

Premature hair greying has been described but the association is still uncertain.

The retina may be affected, however, the colour of the iris does not change.

Sensory hearing impairment has been described in some patients with vitiligo, presumably due to cochlear melanocyte loss, but clear evidence is lacking.

Sunburn in vitiligo lesions may be a problem

Some evidence suggests that people with vitiligo might have a lower risk for internal malignancies and skin cancer.

Two nationwide retrospective studies from South Korea and Taiwan, have shown that vitiligo patients are at lower risk of internal malignancies, in addition to lower BCC and SCC risk in the Taiwanese study.

Another retrospective study of 1307 vitiligo patients found individuals with vitiligo are at lower risk for both melanoma and NMSC. However, some of these studies found a possible higher risk for thyroid cancer.

Severity

Severity is variable and there is no way to predict how much or how fast pigment will be lost.

Vitiligo appears more evident in patients with naturally dark skin.

Extension of vitiligo can occur over a few months, then it stabilises.

Some spontaneous repigmentation may occur from the hair follicles, and the overall size of the white patch may reduce.

At some time in the future, the vitiligo is likely to extend again.

Cycles of pigment loss followed by periods of stability may continue indefinitely.

Light-skinned people usually notice pigment loss during the summer as the contrast between the affected skin and suntanned skin becomes more distinct.

The pigment has occasionally been reported to be lost from the entire skin surface.

Active disease predictors are peripheral hypopigmentation, confetti-like depigmentation, ill-rather than well-defined borders, and the Koebner phenomenon.

For more information, see the section on severity assessment below.

How is vitiligo classified?

The Vitiligo European Task Force (VETF) came to a consensus about the classification of vitiligo in 2007. They decided on four main categories with subtypes.

Classification	Subtypes	Comments
Non-segmental vitiligo	Focal Mucosal Acrofacial Lip-tip Generalized Universal	Tends to be bilateral and symmetrical in distribution. Stable or unstable.
Segmental vitiligo	Focal Mucosal Uni-segmental, bi- or multi-segmental	Affects children Single white patch in 90% Follows dermatomal distribution (most common: trigeminal), does not cross midline, head involved in > 50% of cases Border often irregular + leukotrichia

		Rapid onset, remains stable after the first six months to two years Protracted course Cutaneous mosaicism (Blaschkoid , dermatomal, phylloid, checkerboard patterns)
Mixed vitiligo	Non-segmental combined with segmental vitiligo	Rare Bilateral segmental follows non-segmental (months-years) Predictors to transform into mixed variant: leukotrichia, halo naevi
Unclassified vitiligo	Focal at onset Multifocal asymmetrical non-segmental Unifocal mucosal Punctate (confetti or vitiligo- ponctué) Hypochromic (minor) vitiligo Follicular vitiligo	Punctate: small macules (1–2 mm). Called leukoderma punctata, if there are no other classical vitiligo patches Hypochromic (minor): in type V/ VI skin, mainly seborrheic distribution Follicular: prominent leukotrichia with absent/ limited skin involvement

More than 90% of the adult vitiligo cases are of the generalized vulgaris or acrofacial types, while in children, segmental vitiligo constitutes 15–30% of the cases.

Rare clinical subtypes of vitiligo include:

Trichrome vitiligo: describes three shades of skin colour, where there is an intermediate hypopigmented hue between the white patch and the normal skin.

Quadrachrome and pentachrome vitiligo: four and five shades of colours has been rarely described with multiple shades of tan brown, in addition to white and normal skin colour.

Red vitiligo: white patches that have raised, red, inflammatory borders.

Blue vitiligo: post-inflammatory patches with a bluish-grey hue that correlate histologically with pigment incontinence in dermal melanophages.

[Click here for images of vitiligo](#)

How is the severity of vitiligo assessed?

In most cases, the severity of vitiligo is not formally assessed. However, clinical photographs may be taken to monitor the condition.

At least two scoring systems have been devised for vitiligo and are used in clinical trials.

Vitiligo Area Scoring Index (VASI)

Vitiligo European Task Force (VETF) system

VASI

VASI is based on the [PASI scoring system](#) for psoriasis. It measures the extent and degree of depigmentation in 6 sites: hands, upper extremities, trunk, lower extremities/feet, and the head and neck.

VETF

VETF is based on [SCORAD scoring system](#) for atopic dermatitis. The VETF assesses the extent, staging, and spreading/progression in 5 sites: head/neck, trunk, arms, legs and hands/feet. It grades from 0 (normal pigmentation) to 4 (complete hair whitening). Spreading is assessed using the following scores: 0 (stable disease), -1 (regressive disease) and +1 (progressive disease).

VETF includes a clinical assessment form to record the sex, age, duration of disease, age of onset, episodes of repigmentation, the impact of vitiligo on quality of life, family history, additional medical conditions, and the [Fitzpatrick skin type](#) of the patients.

How do clinical features vary in differing types of skin?

The distribution and characteristics of vitiligo patches are similar in different skin types; however, while vitiligo can be barely noticeable in some people with lighter skin complexions, it is usually more obvious in darker skin types. This can cause significant cosmetic disability, along with its psychological consequences.

What are the complications of vitiligo?

Cosmetically disabling

Psychosocial stresses and social stigma to affected individuals, particularly in people with dark skin.

Higher risk of acquiring an associated autoimmune condition among individuals with vitiligo compared to the general population.

Vitiligo has an otherwise benign nature with most of those affected being in good health.

How is vitiligo diagnosed?

Vitiligo is usually a clinical diagnosis, based on its characteristic appearance, and no specific tests are required to make the diagnosis.

Tools that can aid in diagnosis include:

Wood's lamp

Enhances white patches, making it easier to see less conspicuous lesions.

Especially helpful in light-skinned people, patches with partial loss of pigment, or to monitor response to therapy.

The UVA is absorbed by collagen fibres in the dermis, and fluoresces back as bright white in absence of epidermal melanin (which normally absorbs UVA).

Dermoscopy

Characteristically shows a white glow, with some clues that can help in differentiating between stable and active disease [see [Dermoscopy of vitiligo](#)].

Skin biopsy

Occasionally recommended, particularly in early or inflammatory vitiligo, when a lymphocytic infiltration may be observed.

Where melanocytes are typically absent in the epidermis of established vitiligo patches, some argue that total loss of melanocytes never occurs, indicating potential functionality restoration with treatment.

Blood tests

To assess other potential autoimmune diseases or [polyglandular syndromes](#) may be arranged, especially if combined with a positive family history

Examples include thyroid function tests, ANA, and B12 levels.

What is the differential diagnosis for vitiligo?

For localised vitiligo lesions

[Halo naevus](#)

[Naevus anemicus](#)

[Naevus depigmentosus](#).

For generalised vitiligo

Inherited hypomelanosis

[Piebaldism](#).

[Waardenburg syndrome](#).

[Tuberous sclerosis](#).

[Hypomelanosis of Ito](#) (pigmentary mosaicism).

Secondary hypomelanosis

Infectious: [pityriasis versicolor](#), [leprosy](#) (tuberculoid/lepromatous), [secondary syphilis](#), [treponematoses](#) and [onchocerciasis](#) (late stages).

Post-inflammatory: [lichen sclerosus et atrophicus](#), [morphoea](#)/ [scleroderma](#), [discoid lupus erythematosus](#), [psoriasis](#), and [after-burn](#).

Paraneoplastic: cutaneous lymphoma ([mycosis fungoides](#)), cutaneous [melanoma](#) (localized/distant autoimmune reaction)

Drug/ toxin-induced:

Systemic: [imatinib](#), [chloroquine](#), fluphenazine, physostigmine, [targeted melanoma immunotherapy](#).

Topical: [imiquimod](#), [corticosteroids](#).

Occupational exposure: phenolic compounds; mainly on exposed skin (hands, face).

Idiopathic

[Progressive macular hypomelanosis](#).

[Idiopathic guttate hypomelanosis](#).

What is the treatment for vitiligo?

There is no cure for vitiligo and treatment is often unsatisfactory. The aim is to stop progression of the disease (stabilisation), and to achieve satisfactory re-pigmentation.

Treatment is most successful on the face and trunk; whereas hands, feet, and areas with white hair respond poorly. Compared to long-standing patches, new ones are more likely to respond to medical therapy.

While the hair follicle is the main source of pigment restoration, another potential reservoir can be at the borders of the white patches. When successful re-pigmentation occurs, melanocyte stem cells (DOPA-negative) in the middle and lower outer root sheath (ORS), or bulge at the base of the hair follicle are activated (become larger, with intense DOPA oxidase activity). They migrate to the skin surface to form pigment islands, appearing as perifollicular brown macules. Otherwise, re-pigmentation can occur in less common patterns such as marginal, diffuse, or combined.

Treatment response is evaluated in terms of proportion of skin that has retained pigment. In studies, a good response is usually translated as > 50% or 75%, depending on the study's design.

General measures

A cut, graze, or scratch may lead to a new patch of vitiligo

Minimise skin injury by wearing protective loose-fitting clothing.

Cosmetic camouflage can disguise vitiligo. Options include:

Make-up, dyes, and stains

Waterproof products

Dihydroxyacetone-containing products – "tan without the sun."

Micropigmentation or tattooing for stable vitiligo.

Sun protection with [clothing](#), [sunscreen](#) use, and lifestyle modification.

Depigmented skin can only burn on exposure to ultraviolet radiation (UVR); it cannot tan.

[Sunburn](#) may cause vitiligo to spread.

Tanning of normal skin makes vitiligo patches appear more visible.

Specific measures

There are several modalities that are proven to be helpful in vitiligo. Optimal therapeutic response is often seen with combination therapies.

Topical treatments

Topical corticosteroids

Can be used on the trunk and limbs for up to 3 months.

Calcineurin inhibitors ([pimecrolimus cream](#) and [tacrolimus ointment](#))

Preferred for vitiligo affecting the eyelids, face, neck, armpits, and groin, because, unlike topical steroids, they do not cause skin atrophy.

Topical vitamin D derivatives ([calcipotriol](#), tacalcitol)

Second line; effective only as combination therapy.

Ruxolitinib cream

A [Janus kinase 1 and 2 inhibitor](#) that is FDA approved for the treatment of non-segmental vitiligo in adult and paediatric patients 12 years of age and older.

Significant clinical improvement for facial vitiligo, in individuals 12 years and older with non-segmental vitiligo with 10% or less affected BSA, with sustained safety after 52 weeks has been seen in two phase 3 double-blind clinical trials. Body vitiligo showed less dramatic response.

Other controversial therapies include pseudocatalase and topical prostaglandin inhibitors

Further studies are needed to confirm efficacy.

Phototherapy

Phototherapy refers to treatment with ultraviolet (UV) radiation. Options include:

- Whole-body or localised [UVB phototherapy](#)
- [Excimer laser UVB](#) (308 nm) or [targeted UVB](#) for small areas of vitiligo
- Oral, topical, or bathwater [photochemotherapy \(PUVA\)](#)
- In-office or home phototherapy.

Phototherapy probably works in vitiligo by two mechanisms.

- Immune suppression – preventing the destruction of the melanocytes
- Stimulation of cytokines (growth factors).

Treatment is usually given twice weekly for a trial period of 3–4 months. If re-pigmentation is observed, treatment is continued until re-pigmentation is complete or for a maximum of 1–2 years.

- Phototherapy is unsuitable for very fair-skinned people.
- The treatment intensity aims for the vitiligo skin to be a light "carnation" pink.
- If re-pigmentation is observed, treatment is continued until re-pigmentation is complete or for a maximum of 1–2 years.
- It is essential to avoid burning (red, blistered, peeling, itchy or painful skin), as this could cause the vitiligo to get worse.

A meta-analysis of 35 different studies reporting outcomes after phototherapy for generalised vitiligo. A marked or clinically useful response was achieved in 36% after 12 months of narrowband UVB and in 62% after 12 months of PUVA. The face and neck responded better than the trunk, which responded better than the extremities. It was not very effective on the hands and feet.

Systemic therapy

Systemic steroids

Short pulse therapy to slow rapid progression, or as mini-pulse oral steroids to stabilize active disease, eg, dexamethasone 2.5–4 mg, for two consecutive days per week, for 3–6 months.

[Methotrexate](#)

[Ciclosporin](#)

[Mycophenolate mofetil](#)

Oral minocycline 100 mg/day, a [tetracycline](#) antibiotic with anti-inflammatory properties

Subcutaneous [afamelanotide](#)

An α -melanocyte-stimulating hormone (α -MSH) analogue. Showed superior re-pigmentation when combined with NB-UVB, but excessive tanning in unaffected surrounding skin made final cosmetic results less satisfying.

None of these treatments are based on randomised controlled trial data.

Surgical treatment of stable vitiligo

Surgical treatment for stable and segmental vitiligo requires removal of the top layer of vitiligo skin (by [shaving](#), [dermabrasion](#), [sandpapering](#), or [laser](#)) and replacement with pigmented skin removed from another site.

Techniques include:

Non-cultured melanocyte-keratinocyte cell suspension transplantation
Punch grafting (mini-grafting)
Blister grafts, formed by suction or [cryotherapy](#)
Split skin grafting
Cultured autografts of melanocytes grown in tissue culture.

Depigmentation therapy

[Depigmentation therapy](#), using 20% monobenzyl ether of hydroquinone (MBEH), may be considered in severely affected, dark-skinned individuals with vitiligo that has failed to re-pigment spontaneously or with therapy.

[Cryotherapy](#) and [laser](#) treatment (eg, 755-nm Q-switched [alexandrite](#) or 694 nm Q-switched [ruby](#)) have also been used successfully to depigment small areas of vitiligo.

Future therapies

Other novel potential therapies that are still under research include those targeting IFN- γ -JAK-STAT1 pathway (targeting CXCL9/10), anti-IL15 and anti-CD122 (targeting TRM), Wnt signalling antagonists, and others.

Psychosocial support

Vitiligo results in reduced quality of life and psychological difficulties in many patients, with problems like depression and poor self-esteem, especially in adolescents and in females. Patients should be assured that there is always something on the table to help in managing their condition, from using camouflage to available therapies with possible measurable improvement. The psychosocial impacts of vitiligo tend to be more severe in some countries, cultures, and religions than in others.

Family support, counselling, and cognitive behavioural treatment can be of benefit.

How do you prevent vitiligo?

Unfortunately, there are no proven effective measures to prevent vitiligo. Although dogma related to many theories, ayurvedics, vitamin supplements, and alternative medicine has been endorsed by many vitiligo support groups, these are not based on scientific evidence. Aggressive treatment may help in halting the progression of a rapidly progressive disease.

What is the outcome for vitiligo?

The clinical course of generalised vitiligo is highly unpredictable.

In general, vitiligo progresses slowly and gradually over months, then remains quiescent for years, and is usually difficult to control.

Spontaneous repigmentation may occur after exposure to sun, mainly in younger individuals, but this is unpredictable and is usually incomplete. This usually starts as brown spots arising around the hair follicles (perifollicular), usually darker than the normal skin colour, where the overall size of the white patch may get smaller.

Vitiligo can extend further, either by the appearance of new spots, or by peripheral enlargement of pre-existing ones.

Cycles of pigment loss followed by periods of stability may continue indefinitely.

Poor prognostic indicators include longstanding disease, leukotrichia, mucosal involvement, and Koebner phenomenon.

[Click here for images of vitiligo](#)

Viral wart

Author: Dr Amanda Oakley, Dermatologist, Hamilton, New Zealand, 1996. Updated by Dr Jannet Gomez, Postgraduate student in Clinical Dermatology, Queen Mary University London, UK, December 2016. Updated February 2021 and minor update April 2023.

What is a viral wart?

A viral wart is a very common benign lesion caused by infection with human papillomavirus (HPV). Viral warts can be classified by site as being cutaneous or mucosal as the HPV types are quite distinct [see [Anogenital wart](#) and [Sexually acquired human papillomavirus](#) for further information on mucosal HPV infection]. A cutaneous wart is also called a verruca or papilloma, and warty-looking lesions of any cause may be described as verrucous or papillomatous.

Viral warts



Common warts



Plane warts



Filiform wart

[See more images of viral warts.](#)

Who gets cutaneous viral warts?

Warts are particularly common in:

School-aged children, however they may occur at any age

[Dermatitis](#), due to a defective skin barrier

People with [drug-induced immunosuppression](#) such as with long-term [azathioprine](#) or [ciclosporin](#) use, or have [human immunodeficiency virus \(HIV\)](#) infection.

What causes cutaneous viral warts?

Warts are due to infection by the human papillomavirus (HPV), a double-stranded DNA virus. There are more than 150 known HPV types, only some of which infect the skin, giving rise to a variety of clinical presentations. Infection begins in the basal layer of the epidermis, causing proliferation of the keratinocytes (skin cells) and hyperkeratosis, and production of infectious virus particles – the wart. The most common HPV types infecting the skin are types 1, 2, 3, 4, 10, 27, 29, and 57.

HPV is spread by direct skin-to-skin contact or autoinoculation; if a wart is scratched or picked, a wart may develop under the fingernail (subungual wart) or virus may be spread to another area of skin. Autoinoculation of the virus in a scratch can result in a line of warts (pseudo-koebnerisation). The incubation period can be as long as twelve months, depending on the amount of virus inoculated.

[see [Non-sexually acquired human papillomavirus infection](#)]

Autoinoculation of warts



Pseudo-koebnerisation of warts



Subungual wart



Subungual wart

What are the clinical features of viral warts?

Cutaneous viral warts have a hard, keratinous surface. Tiny red or black dots visible in the wart are papillary capillaries.

Common wart

Common warts (*verruca vulgaris*) present as cauliflower-like papules with a rough, papillomatous and hyperkeratotic surface ranging in size from 1 mm to 1 cm or more. They may be solitary or multiple. Common warts are found most often on the knees, backs of fingers or toes, and around the nails (periungual).

Common warts (*verruca vulgaris*)



Verruca vulgaris on a thumb



Plantar wart

Plantar warts (*verruca plantaris*) include tender inwardly growing myrmecia on the sole caused by HPV 1, and clusters of superficial less painful mosaic warts due to HPV 2. Myrmecial warts are typically tender with lateral and direct pressure, are surrounded by yellow hyperkeratotic callus-like skin showing accentuated skin markings, but with discontinuation of the skin lines through the actual wart.

Plantar epidermoid cysts are associated with HPV 60 infection of the eccrine ducts.

Plantar warts, myrmecial type (*verruca plantaris*)



Plantar warts

Plantar warts, mosaic type



Plane wart

Plane warts are typically multiple small flat-topped skin-coloured papules located most commonly on the face, hands, and shins. On the shins and beard-area of the face the virus is often spread by [shaving](#) resulting in numerous warts. Plane warts are mostly caused by HPV types 3 and 10.

Plane warts (*verruca plana*)



Verruca plana in the beard area spread by shaving



Filiform wart

A filiform wart is a cluster of fine fronds emerging from a narrow pedicle base usually found on the face. They are also described as digitate (finger-like).

Filiform and digitate warts





Filiform wart

Butcher's wart

Butcher's warts are specifically caused by HPV 7 infecting the hands of butchers and others whose occupation involves chronic exposure to a cold moist environment. They clinically resemble common warts and tend to be numerous.

Epidermodysplasia verruciformis

Epidermodysplasia verruciformis is a rare autosomal recessive condition susceptible to skin infection with specific HPV types that cause flat pityriasis versicolor-like lesions and [squamous cell carcinoma](#).

What are the complications of cutaneous viral warts?

Viral warts are infectious to the patient and others.

Cutaneous warts can have significant psychosocial effects such as teasing at school, embarrassment, permission refused for swimming lessons.

Periungual warts can cause nail dystrophy and destruction.

Pain due to plantar warts (myrmecia type) interferes with walking and sporting activities, causing knee or hip pain.

In epidermodysplasia verruciformis the specific HPV types involved can cause cutaneous squamous cell carcinomas.

Periungual warts causing nail dystrophy



Ungual wart



How is a cutaneous viral wart diagnosed?

Cutaneous viral warts are usually diagnosed clinically. Clinical clues to diagnosis can include:

Pinpoint red or black dots (papillary capillaries) are revealed when the wart is pared down. Patent capillaries cause pinpoint bleeding. Plantar corns lack the papillary capillaries.

Location of a plantar wart is not restricted to pressure sites whereas a plantar callus or corn is always at a pressure site.

Tenderness is maximal with lateral pressure for a plantar wart whereas a corn or callus is more tender with direct pressure.

Dermoscopy assists visualisation of the papillary capillaries of a viral wart, and can distinguish other verrucous lesions such as a [seborrhoeic keratosis](#).

Diagnosis of viral warts



Red and black pinpoint dots of papillary capillaries



Dermoscopy: viral wart



Dermoscopy: viral wart

[Skin biopsy](#) is sometimes required when squamous cell carcinoma cannot be excluded clinically such as in an organ transplant recipient susceptible to both. [see [Verruca vulgaris pathology](#), [Verruca plana pathology](#)]

What is the differential diagnosis of a cutaneous viral wart?

Differential diagnoses for a cutaneous viral wart can include:

- Seborrhoeic keratosis
- Squamous cell carcinoma
- Plantar [corn and callus](#).

What is the treatment for viral warts?

Treatment may not be required in all cases as most warts resolve spontaneously especially in children. Indications for active treatment include:

- Immunosuppression
- Presence of complications
- Patient preference.

Treatments do not kill the virus, but work by removing virus-containing skin. Persistence with the treatment and patience is essential! Remember HPV infects the basal cell layer of the epidermis so warts recur rapidly if the virus has not been eradicated.

Topical treatment

Topical treatment includes wart paints, pastes, or patches containing [salicylic acid](#), [podophyllin](#), or similar compounds, which work by removing the surface skin cells.

Topical treatment is applied once daily to the wart. Treatment with wart paint usually makes the wart smaller and less uncomfortable; 70% of warts resolve within twelve weeks of daily applications.

- Soften the wart by soaking in a bath or bowl of hot soapy water.
- Rub the wart surface with a piece of pumice stone or emery board.
- Apply wart paint or paste accurately and include a rim of normal skin.
- Allow the paint to dry before covering with plaster or duct tape.
- Next day remove the old paint and dead surface skin layer with a pumice stone and reapply the paint or paste.

If the wart paint makes the skin sore, stop treatment until the discomfort has settled, then recommence as above.

Cryotherapy

[Cryotherapy](#) with liquid nitrogen is repeated at one to two-week intervals to cause peeling of the surface layer. It is uncomfortable and results in blistering for several days or weeks. Treatment is required frequently to prevent the wart regrowing between appointments. Success is in the order of 70% after 3–4 months of regular freezing.

A hard freeze using liquid nitrogen might leave a permanent white mark. It can also cause temporary numbness if performed over a superficial nerve such as on the side of a finger.

Treatment of warts



Cryotherapy for warts

Electrosurgery

Electrosurgery ([curettage and cautery](#)) has been used for large and resistant warts. Under local anaesthetic, the growth is pared away and the base burned. The wound heals in two weeks or longer (depending on the site); even then 20% of warts can be expected to recur within a few months. This treatment leaves a permanent scar which can be painful to walk on if located on a pressure site. Recurrent wart in a scar is very hard to treat.

Other treatments

Other treatments for recurrent, resistant or extensive warts include:

The immune modulator, [imiquimod](#) cream - is approved for treating anogenital warts but is usually ineffective for cutaneous warts

[Bleomycin injections](#)

[Pulsed dye laser](#) destruction of feeding blood vessels

[Photodynamic therapy](#)

[Laser vaporisation](#)

[Diphencyprone](#), dinitrochlorobenzene, or squaric acid to cause localised allergic contact dermatitis over the wart.

How can viral warts be prevented?

[Vaccines against human papillomavirus](#) are available to prevent [anogenital warts](#). Anecdotally, these have been reported to result in the clearance of non-genital warts in some people without definite evidence that the vaccine is the cause of remission.

What is the outcome for cutaneous viral warts?

No treatment is universally effective at eradicating viral warts.

In children, even without treatment, 50% of warts disappear within six months, and 90% are gone in 2 years.

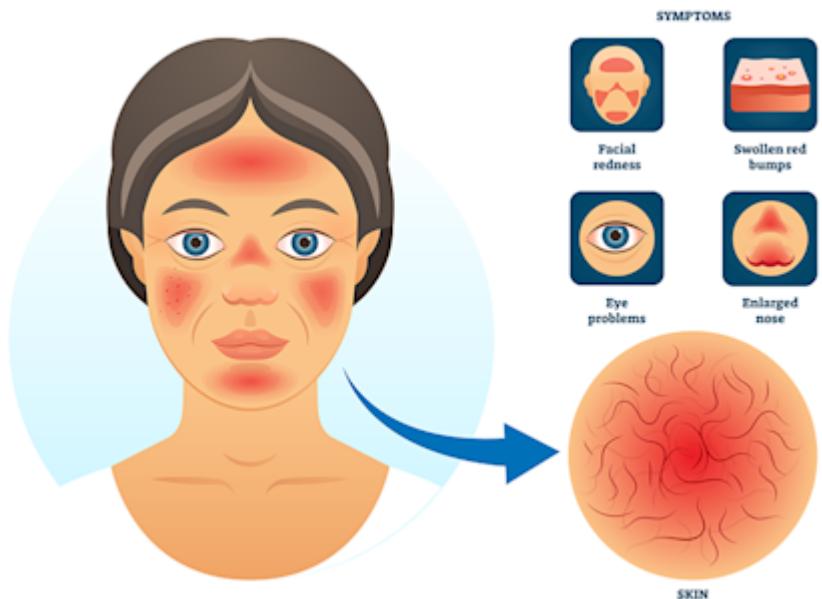
Viral warts are more persistent in adults, but they clear up eventually. They are likely to recur in patients that are [immunosuppressed](#), for example, organ transplant recipients. Recurrence is more frequent in tobacco [smokers](#).

Immunity to HPV is likely to be type-specific.



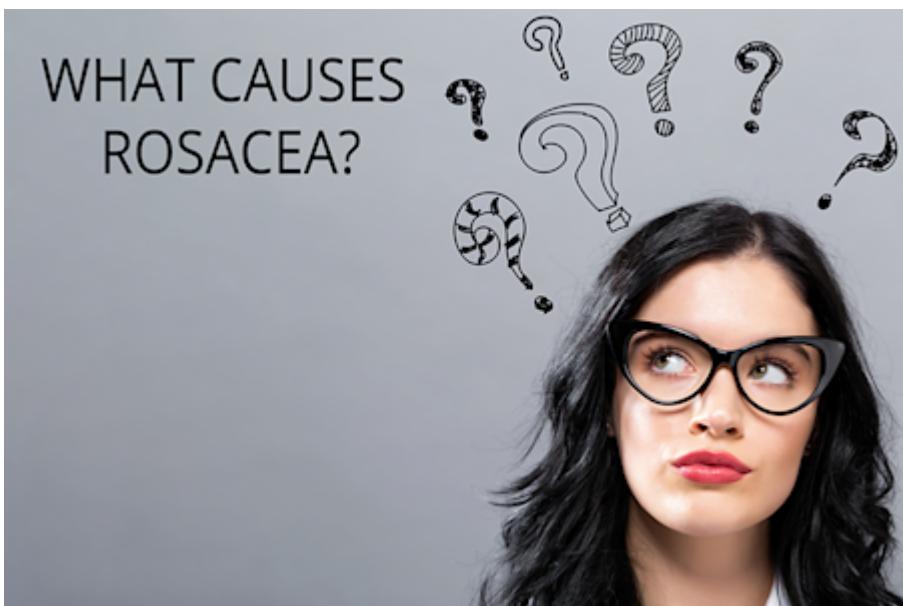
WHAT IS ROSACEA?

Rosacea is a skin condition that mainly affects the face. You may see flushing, lasting color (red, violet, or brown), acne-like breakouts, or visible blood vessels. Some people develop irritated eyes or thickening skin. Your dermatologist knows how to diagnose rosacea and create a treatment plan that can bring relief.



Rosacea: Overview

Rosacea often begins with a tendency to blush or flush easily. Here's why you want to get it diagnosed in this early stage.



Rosacea: Causes

Scientists are still working on answering this question. What they have learned is that all these factors likely play a role.



Rosacea: Symptoms

Rosacea causes so many signs and symptoms that researchers created these subtypes. One affects your eyes.



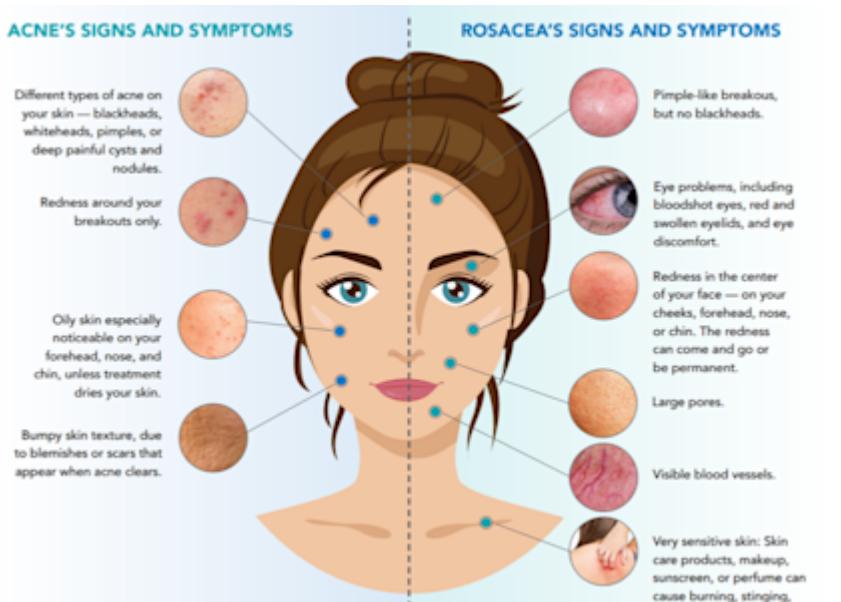
Could my child have rosacea?

Few children and teens have rosacea, but it's worth considering if you notice any of these signs or symptoms.



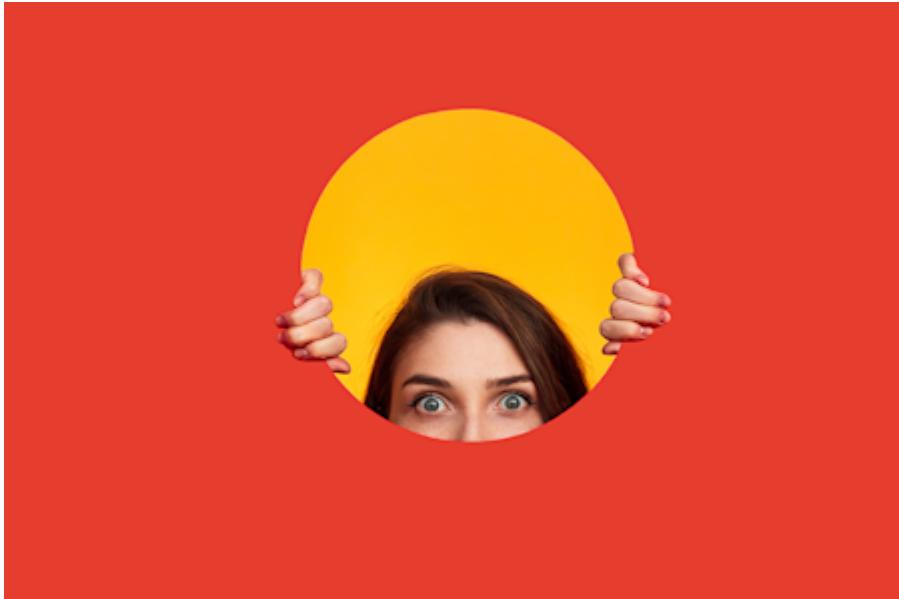
Rosacea in darker skin tones

If you have a darker skin tone, you may never see a red face. Should you notice any of these other signs, it's time to see a dermatologist to find out if you could have rosacea.



Acne or rosacea?

These two conditions can look alike. This infographic shows you how they differ.



Is rosacea causing your red face?

Rosacea can make your face red all (or most of) the time, but, it's not the only sign. Do you have any of these other signs or symptoms?

White nail

October 2022

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What are white nails?

A white nail, also known as leukonychia, is the partial or full discolouration of the nail plate on one or more fingernails or toenails.

White nails are the most common nail dyschromia. The nail will lose its general pink undertone and appear white.



Punctate leukonychia



Parallel bands of leukonychia due to cyclical chemotherapy



Congenital leukonychia



'Half and half nail': leukonychia is affecting the proximal half of the nail



Congenital leukonychia



Leukonychia due to hypoalbuminaemia

[Click here for more images](#)

How is a white nail classified?

Leukonychia can be classified by underlying pathology, its distribution, or how it develops.

Classification according to pathology

Leukonychia can be subdivided into true and apparent discolouration.

True leukonychia: discolouration due to abnormal nail plate keratinisation. The white nail will not be hidden by pressure application of the nail plate to the bed.

Apparent leukonychia: secondary to disease of the nail bed. This appearance disappears with pressure application on the nail.

Classification according to distribution

Leukonychia can be partial or total.

Total leukonychia: whitening of the entire nail plate.

Partial leukonychia: 3 subtypes are described.

Punctate

Longitudinal

Striate (see below).

Classification by development timeline

White nails can be acquired or congenital.

Congenital: familial leukonychia is more commonly inherited recessively, although dominant patterns are possible. This is due to a mutation in the phospholipase C delta-1 gene in which all nails appear milky and porcelain white.

Acquired: secondary to systemic disease. Important to note that congenital leukonychia may also be secondary to systemic disease (see below).

Who gets a white nail?

White nails can affect anyone of any gender, age or ethnicity. Its presence may warrant a work-up for systemic disease.

What causes a white nail?

Trauma

True leukonychia: partial or whole nail plate damage caused by injury to the nail plate or matrix. Keratin disruption with trapped air within the nail plate, resulting in reflection and lack of transparency.

Punctate leukonychia: occurs after nail biting, manicuring, knocks and bangs, and tight footwear use.

Striate leukonychia: also known as Mees lines or transverse leukonychia, may follow damage to the nail matrix; furrows and ridges may also appear.

Total leukonychia: can follow a more serious injury, often with detachment of the nail plate from the nail bed, and alteration to the nail contour.

Poisoning and drugs

Mees line, Lindsay nails, Muehrcke lines (see below), and punctate leukonychia may be associated with:

Heavy metal poisoning (eg lead, [arsenic](#))

[Chemotherapy](#)

Sulphonamides.

There are three distinct types of apparent leukonychia that may be associated with the systemic disease:

Muehrcke lines: A pair of observable, non-palpable, horizontal (transverse) white lines across the nail due to variable blood flow

Lindsay nails (half-and-half nails): Proximally white or pink-coloured nail with a distal darkening

Terry nails: Whitening of the majority of the nail with a thin 0.5–3.0 mm distal darkening.

Systemic illness

Terry nails have been associated with:

Liver cirrhosis

Chronic kidney disease

Heart failure

Hypoalbuminemia (due to protein malabsorption, eg, in colitis)

[Hypothyroidism \(cretinism\)](#)

Zinc deficiency

Hyperthyroidism.

Lindsay nails have been associated with:

Chronic kidney disease

[Psoriasis](#).

Muehrcke lines have been associated with:

Hypoalbuminemia.

What are the clinical features of a white nail?

True leukonychia with partial distribution:

Punctate leukonychia:

Most common form of true leukonychia

Described as small white spots on the nails

Tends to be the result of trauma and isolated to a few nails.

Longitudinal leukonychia:

Small white longitudinal bands

Presenting in individuals with [Darier disease](#) or [Hailey-Hailey disease](#).

Striate or transverse leukonychia (Mees line):

One or more white horizontal bands across the entire nail in parallel with the lunula.

Patients with multiple true leukonychia warrant a thorough history, physical examination, and medication review to exclude a toxic or systemic etiology. This is also true of leukonychia extending the full width of the nail plate.

[Click here for images](#)

How do clinical features vary in differing types of skin?

They appear similar in different skin types.

What are the complications of a white nail?

White nails are a cosmetic nuisance but may be a marker of an underlying systemic disease, but per se do not have any physical complications.

How is a white nail diagnosed?

A thorough history and physical examination may be sufficient for diagnosis. Although, when the cause is unclear, the following tests may be helpful:

Nail clippings to exclude fungal infection

[Nail biopsy](#)

Blood tests to evaluate systemic disease, particularly renal and liver function tests.

What is the differential diagnosis for a white nail?

Onychomycosis (also known as pseudoleukonychia) – the disease of the nail plate is due to external factors

Onycholysis

[Nail psoriasis](#)

Trachyonychia ([twenty-nail dystrophy](#))

[Vitiligo](#) of the nail.



White discolouration of the distal nail plate in superficial white onychomycosis



Nail psoriasis

[Click here for more images](#)

What is the treatment for a white nail?

Treatment ultimately depends on the presence of any underlying cause.

There is no treatment for trauma-related leukonychia. Punctate lesions will disappear as the nail follows its natural growth pattern (around 6 to 9 months for a fingernail).

How do you prevent a white nail?

Avoidance of trauma-induced leukonychia will help prevent development of white nails.

Avoiding contact with irritating substances, and wearing appropriate protective equipment if contact is required.

Avoiding excessive use of nail polish or excessive mechanical force with false nail application/removal.

Minimising picking and biting nails.

Wearing appropriate shoes to prevent excessive pressure on toes.

Applying [moisturisers](#) to hydrate nails.

What is the outcome for a white nail?

Leukonychia due to minor trauma or medication may completely resolve over a few months. In other cases, the white nail plate may remain permanently or demonstrate recurrence.

Winter itch

Author: Dr Varitsara Mangkorntongsakul, Junior Medical Officer, Central Coast Local Health District, Gosford/Hamlyn Terrace, NSW, Australia. DermNet Editor in Chief: Adjunct A/Prof Amanda Oakley, Dermatologist, Hamilton, New Zealand. Copy edited by Gus Mitchell/Maria McGivern. August 2018.

What is winter itch?

Winter itch, also known as pruritus hiemalis, is a type of subclinical [dermatitis](#), that affects individuals during cold weather.

Clinical signs of winter itch



Apparently normal skin



Excoriations



Dermatitis

Who gets winter itch?

Winter itch can affect the healthy population of all ages, but has a higher prevalence in older people with [dry skin](#).

It is uncommon in children and adolescents.

It affects men and women equally.

It is not influenced by socioeconomic status or personal hygiene.

What causes winter itch?

The exact cause of winter itch is unknown. Factors associated with winter itch include:

Itching associated with cold weather, often coexisting with [dry skin](#)

Itch presenting in autumn and winter, and clearing up during the summer months

The aggravation of symptoms by the wearing of certain types of [textures](#), such as flannel or woollen wear.

Similar symptoms have also been reported in patients exposed to refrigerated air conditioning during the summer months. Winter itch is not influenced by frequency of bathing or by the temperature of the bath water.

What are the clinical features of winter itch?

Winter itch does not cause a primary visible rash. The affected skin generally appears healthy, but usually slightly dry.

The itch affects most or all parts of the body, but most commonly occurs on the legs.

Typical sites are the inner surface of the thighs, above and behind the knees, on the calves, and around the ankles.

It does not affect the hands, feet, face or scalp.

The degree of irritation varies from mild to severe; when severe, it can result in great distress and discomfort, that may require medical attention.

The itchiness is sudden in onset, and is generally most noticeable at night and when removing clothing.

Secondary lesions arise as a consequence of [chronic scratching](#).

The skin becomes erythematous with noticeable scratch marks.

Scratched skin may be dry, rough and thickened ([lichenified](#)).

Intense rubbing of the skin may cause desquamation.

There may be a secondary [folliculitis](#), with torn and [broken hair shafts](#) due to constant scratching.

How is winter itch diagnosed?

Winter itch can be associated and confused with other diseases that cause itchiness, particularly [dermatitis](#) (where there is a visible rash), and [pruritus](#) associated with systemic diseases (which tends to be generalised).

Detailed history taking and careful examination are important to rule out [other potential causes of pruritus](#).

What is the treatment for winter itch?

Treatment is mainly to provide symptomatic relief and prevent scratching.

Bathe in warm water prior to sleep. Some people report a benefit from the addition of sodium bicarbonate to the water (a quarter of a cup of baking soda swished around in a full bath).

[Emollients](#) are the mainstay of treatment. Apply a moisturising cream after bathing and whenever the skin feels itchy or dry.

Wear lightweight clothing such as silk, linen, and muslin.

Avoid irritating fabrics, such as woollen clothing.

Use [topical corticosteroids](#) to treat secondary [dermatitis](#) and [lichen simplex](#).

[Capsaicin](#) cream can be useful for localised areas of persistent itch.

Prescriptions

Systemic agents are not indicated for winter itch, but oral [antihistamines](#) and [systemic corticosteroids](#) are sometimes prescribed as a clinical trial.

What is the outcome for winter itch?

Winter itch normally resolves after the winter months.

The duration of itching varies among individuals. It may last for a few days or weeks, and occasionally throughout the winter.

Winter itch may recur throughout a person's lifetime or disappear permanently at the end of the first attack.

Yellow nail syndrome

Author: Vanessa Ngan, Staff Writer, 2006.

What is yellow nail syndrome?

Yellow nail syndrome is a rare disorder of the nail, which is usually accompanied by lymphoedema (swelling of parts of the body caused by blockage or damage to the drainage of the lymphatic system). It may also be associated with recurrent pleural effusions (fluid collection in space surrounding the lungs) and less commonly bronchiectasis (chronic, abnormal dilation of the bronchi in the lungs), chronic bronchitis and sinus infections.



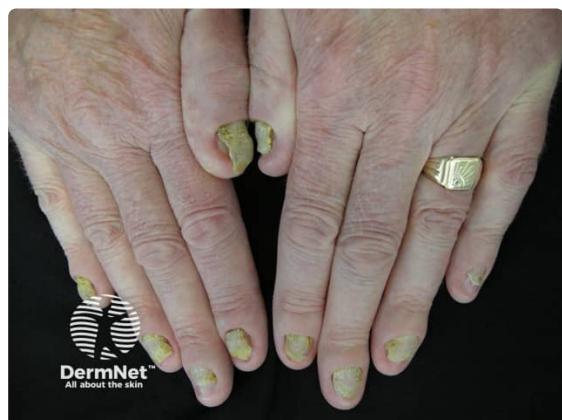
Yellow nail syndrome



Yellow nail syndrome



Yellow nail syndrome



Yellow nail syndrome



What are the signs and symptoms?

Yellow nail syndrome most often starts in middle age, although a similar condition has been described in younger children.

The three main features of yellow nail syndrome are described in the following table.

Feature	Characteristics
Nail changes	<p>All nails may be affected</p> <p>Nails are slow growing or appear to have stopped growing</p> <p>Nails become thicker and turn a pale yellow or greenish-yellow colour with edges slightly darker</p> <p>Nails mainly remain smooth but may be cross-ridging and nail humped with loss of cuticles</p> <p>Onycholysis (separation of nail from the nail bed) may affect one or more nails</p>
Lymphoedema	<p>Swelling occurs in about 80% of patients and most frequently affects the legs</p> <p>Signs of swelling usually occur after nail changes appear and may not be seen for some months later</p> <p>Swelling less often affects the hands, face or genitals</p>
Respiratory signs	<p>Pleural effusions occur in about 36% of patients</p> <p>In about 30% of patients, the initial symptom is related to pleural effusions</p> <p>Patients often give a history of recurrent attack of bronchitis, chronic sinusitis, and pneumonia</p>

What causes yellow nail syndrome?

The cause of yellow nail syndrome is unknown. However, it is seen in patients with chronic bronchiectasis or sinusitis, pleural effusions, internal malignancies, [immunodeficiency](#) syndromes, and [rheumatoid arthritis](#). In some cases the lymphatic abnormality may be congenital (occur during development) but in most it is probably related to the other associated conditions.

What treatment is available?

Patients should receive appropriate medical treatment for their respiratory symptoms and oedema.

Nail changes once established are usually permanent, although complete reversion to normal nails has occurred in some cases. Treatment of nails includes topical vitamin E solution and oral itraconazole. Some studies have shown that nutritional supplementation with vitamin E appears to be effective in controlling yellow nail syndrome, for unknown reasons. Zinc supplements have also been used but it is unclear whether they are effective.

Itraconazole and fluconazole are oral antifungal agents. It has been noted that they appear to speed up the rate of growth of nails, which may be of benefit in yellow nail syndrome even though it is not caused by fungal infection.