Basic Science and Common Inflammatory Dermatoses Tutorial

Tuesday 20th January 2026

Session Instructions

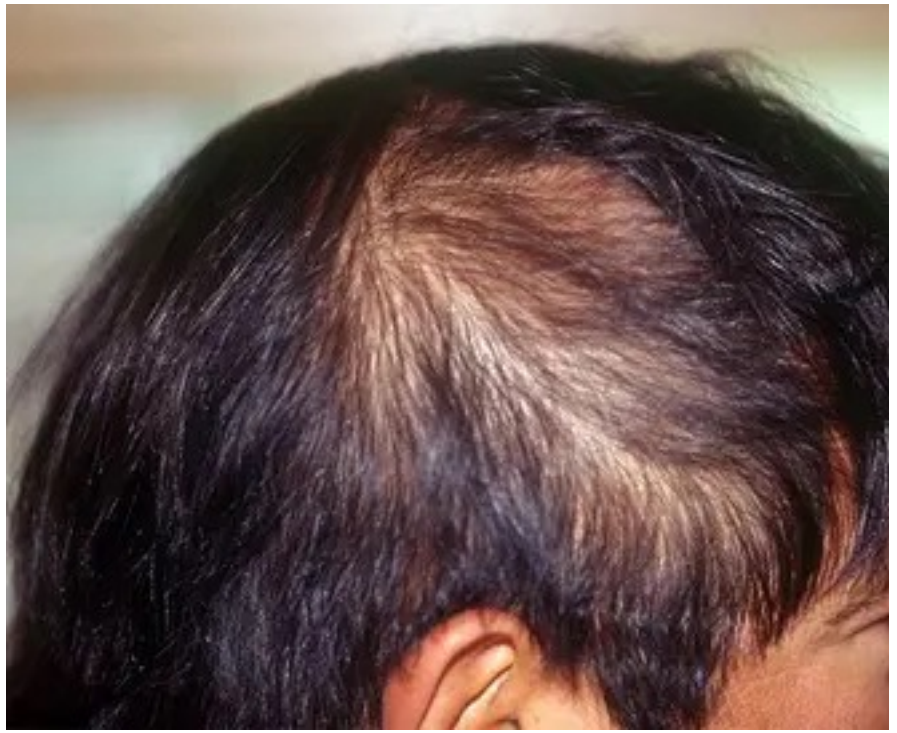
- You will have 15 minutes to work through the following 3 clinical cases. You will be prompted to move onto the next case after 5 minutes by your tutor.

- Please work in small groups of approx. 5 people and nominate a spokesperson to feedback your answers to the group.

- After 15 minutes, we will then begin the group debrief led by your session tutor.

Case 1

- A 24-year-old male presents to his GP with concerns about increased hair shedding over the few weeks.
- He reports noticing large amounts of hair on his pillow and in the shower drain. He denies scalp pain, pruritus, or patchy hair loss.
- Three months ago, he experienced a severe febrile illness (Influenza A). He has no significant past medical history and takes no regular medications.
- Physical examination reveals diffuse thinning of scalp hair without scarring, erythema, or focal patches of hair loss.
- A hair pull test is positive, with several hairs easily extracted



www.medicinenet.com

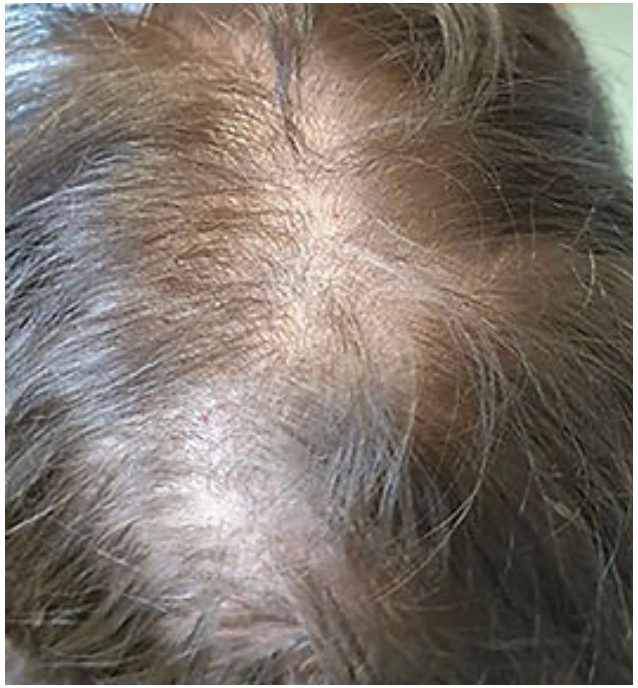
Qu 1: What is the most likely diagnosis?

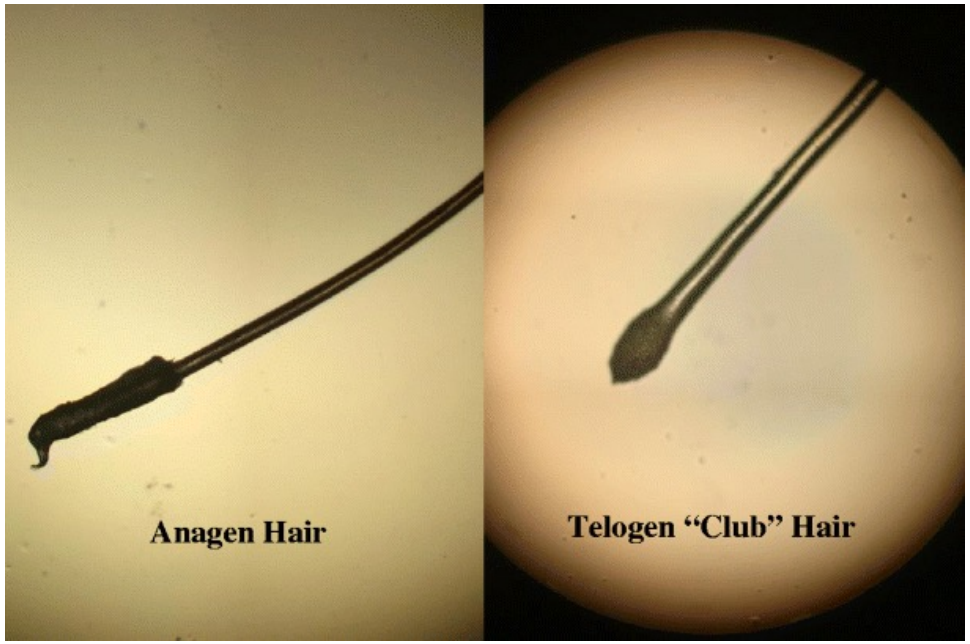
Telogen Effluvium

- Non-scarring form of diffuse hair loss with no clinical or histological evidence of inflammation
- Temporary hair loss due to a large increase in the number of hairs in the shedding phase (telogen)
- It is normal to lose up to about 100 hairs a day as a result of the normal scalp hair cycle.
- If there is some shock to the system (e.g. febrile illness in this case), as many as 70% of the anagen hairs can be precipitated into telogen, thus reversing the usual ratio.
- The resting scalp club hairs remain firmly attached to the hair follicles at first. New hairs coming up through the scalp then push out the resting club hairs and increased hair fall is noticed 2 to 4 months after the triggering event.

Telogen Effluvium

- Telogen effluvium is usually diagnosed by its clinical features
- Hair thinning involves the entire scalp +/- loss of other body hair
- Examination shows diffuse thinning without focal areas of total alopecia or scarring, and short hairs of normal thickness
- A hair pull test reveals an increased number of extracted hairs; most are in telogen with a typical epithelial sac (club hair)





www.dynamicregenmedicine.co.uk, https://doi.org/10.1007/s13671-017-0169-6

Qu 2: Which of the following best explains the underlying pathophysiological mechanism of this condition?

A. Autoimmune destruction of hair follicles during the anagen phase
B. Premature transition of hair follicles from anagen to telogen phase
C. Irreversible fibrosis of hair follicles
D. Androgen-mediated miniaturization of hair follicles
E. Fungal infection of hair shafts

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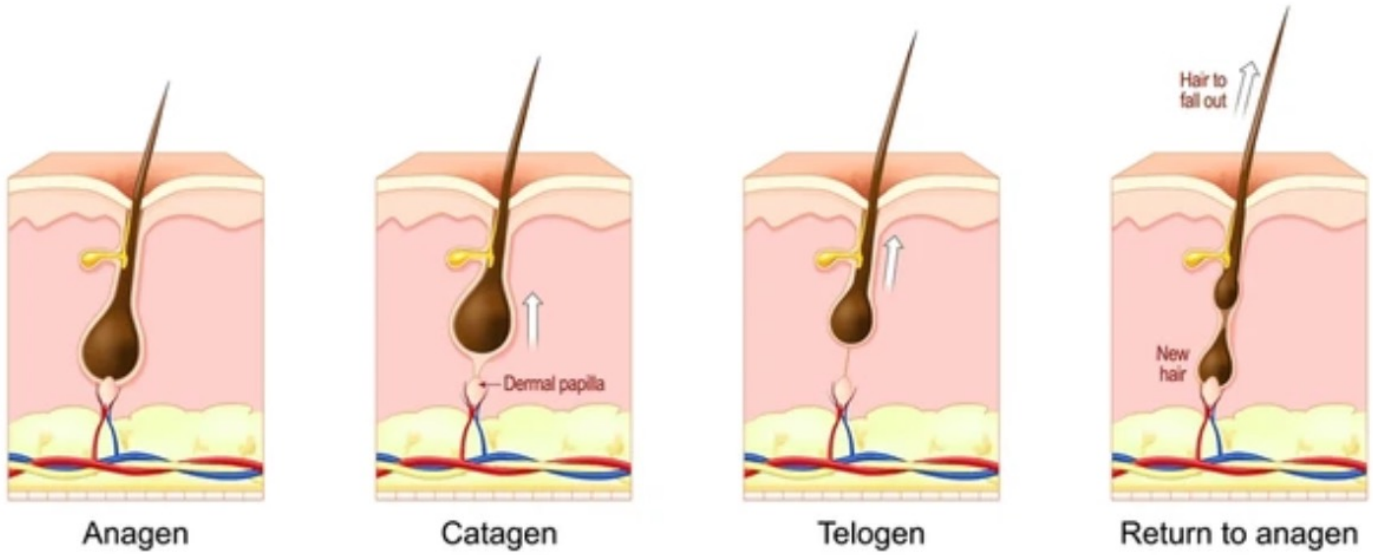


What causes telogen effluvium?

Typical triggers include:

- Childbirth: postpartum hair loss
- Severe trauma or illness (especially if a high fever occurs)
- Stressful or major life events
- Surgical procedures
- Marked weight loss, extreme dieting, or nutritional deficiency (e.g. iron deficiency)
- Severe skin problems affecting the scalp
- Starting new medications or stopping a hormone treatment (such as coming off the contraceptive pill)
- Endocrine disorders (e.g. hypothyroidism, hyperthyroidism)

Qu 3: What investigations would you consider doing?

- Blood tests can be carried out to exclude other possible causes of hair loss – FBC, ferritin, zinc, vitamin D and thyroid function

- Examination of the hair with light microscopy would demonstrate club telogen hairs

- Very rarely a skin biopsy may be required where diagnosis is uncertain, but this is typically not required

Management

- Telogen effluvium is self-resolving

- The shedding phase in the hair cycle lasts between 3 to 6 months. After this, new hair starts to grow. It may take many months for hair to return to normal volume and thickness.

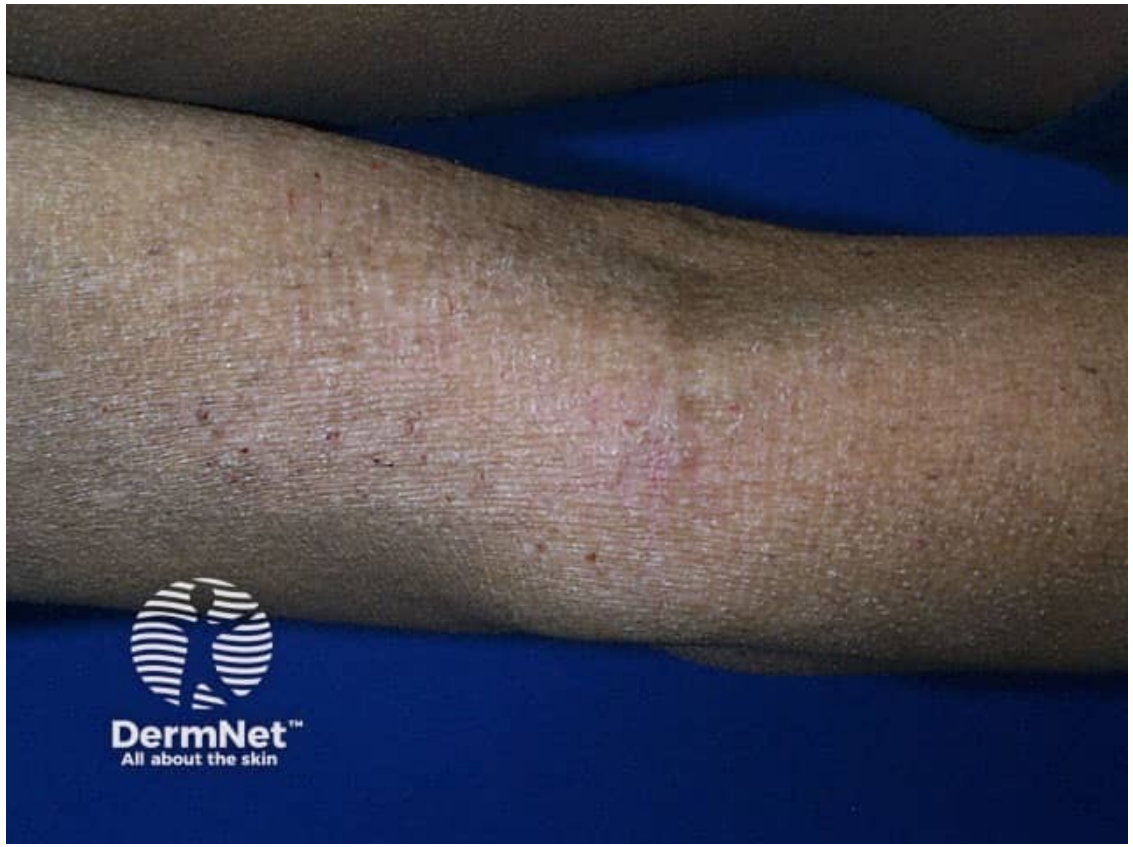
- Recommendations include:
  - Gentle handling of the hair, avoiding over-vigorous combing, brushing and any type of scalp massage
  - Ensure a nutritious diet, with plenty of protein, fruit and vegetables
  - Correct any abnormalities in thyroid function, iron, vitamin D or zinc

Case 2

- A 28-year-old female with a past medical history of asthma and allergic rhinitis presents with a history of intermittently itchy skin over many years, which has worsened over the recent winter months.

- Examination reveals poorly defined, erythematous plaques with excoriations and lichenification of the skin. You note a background of generalised dry skin.

- This predominantly affects flexural areas (antecubital and popliteal fossae) as well as her face and hands.



Qu 1: What is the most likely diagnosis?

Atopic Dermatitis (atopic eczema)

- Atopic dermatitis, also called atopic eczema, the most common inflammatory skin disease worldwide, presents as generalised skin dryness, itch, and rash.

- It typically affects people with an ‘atopic tendency’ clustering with hay fever, asthma, and food allergies.

- Atopic dermatitis results from a complex interplay between environmental and genetic factors.

- The clinical phenotype of atopic dermatitis can vary greatly, but is characterised by remission and relapse with acute flares on a background of chronic dermatitis.

- Acute dermatitis can be red (erythematous), weeping/crusted (exudative) and may have blisters (vesicles). Over time the dermatitis becomes chronic and the skin becomes less red but thickened (lichenified) and scaly. Cracking of the skin (fissures) can occur.

Qu 2: Which of the following best explains the underlying pathophysiological mechanism of this condition?

A. Immune complex deposition in small dermal blood vessels

B. Impaired epidermal barrier function due to filaggrin gene mutation

C. Autoantibody formation against basement membrane proteins

D. Overproduction of sebum by hyperactive sebaceous glands

E. Keratinocyte hyperproliferation driven by a Th17-mediated inflammatory response

Qu 2: Which of the following best explains the underlying pathophysiological mechanism of this condition?

A. Immune complex deposition in small dermal blood vessels = vasculitis

B. Impaired epidermal barrier function due to filaggrin gene mutation = eczema

C. Autoantibody formation against basement membrane proteins = immunobullous disease (e.g. bullous pemphigoid)

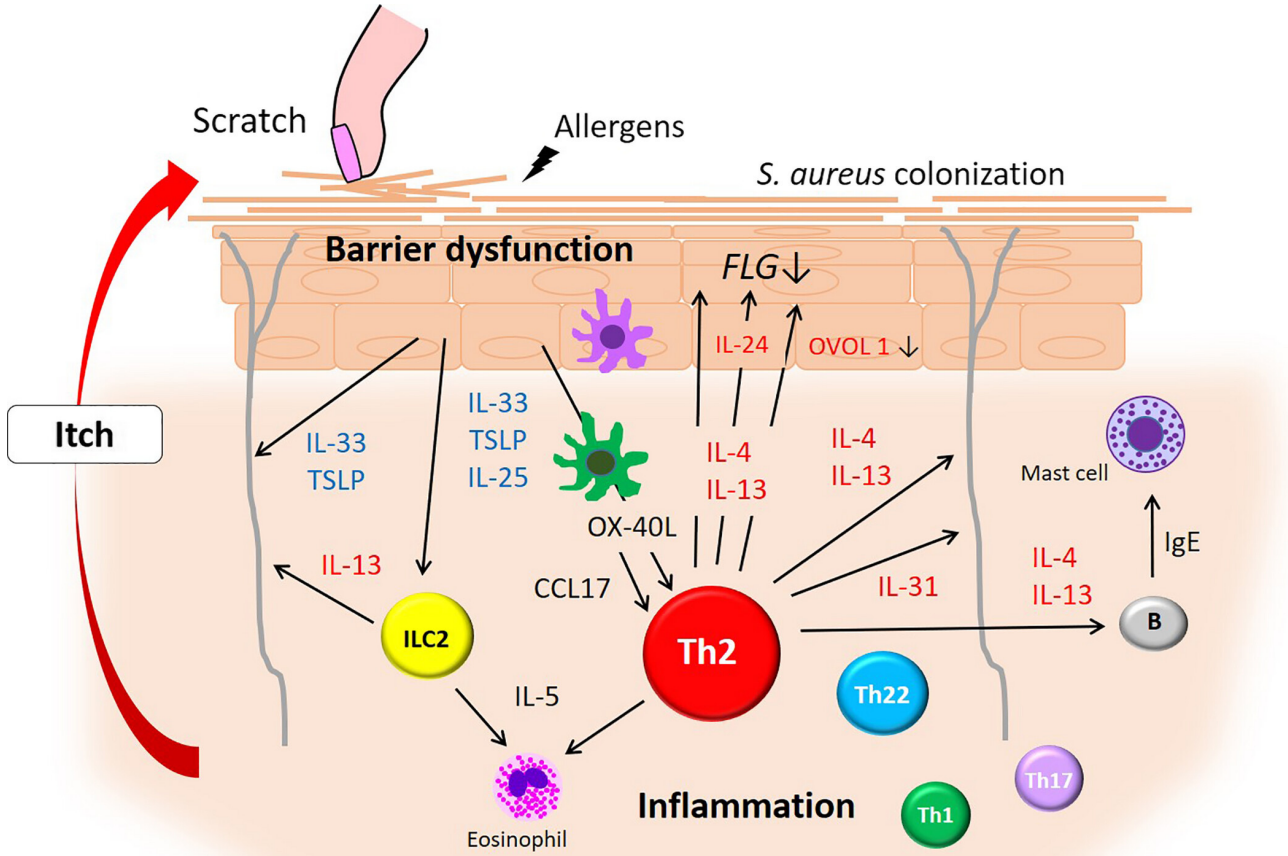
D. Overproduction of sebum by hyperactive sebaceous glands = acne

E. Keratinocyte hyperproliferation driven by a Th17-mediated inflammatory response = psoriasis

Atopic Dermatitis Pathogenesis

- Atopic dermatitis has multiple causes, involving interactions between the immune system, genetic skin barrier defects, and environmental factors

- Immune system imbalance is central, particularly an overactive **Th2 response** with cytokines such as **IL-4, IL-13, and IL-31**, which drive inflammation and weaken the skin barrier.



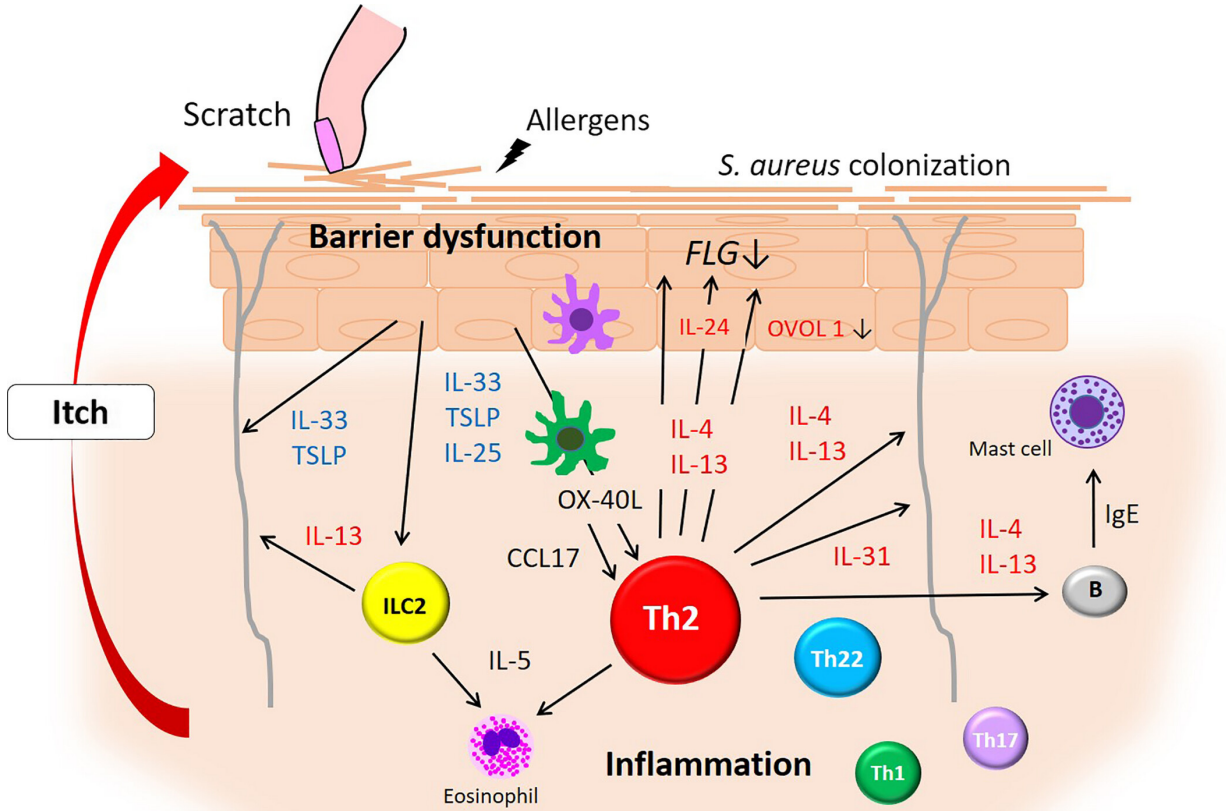
doi: 10.1111/1346-8138.15664

Atopic Dermatitis Pathogenesis

- Inherited skin barrier defects, especially filaggrin gene (FLG) mutations, lead to increased skin permeability, transepidermal water loss and reduced antimicrobial protection

- Atopy is a genetic tendency toward eczema, asthma, and hay fever. Environmental triggers (infections, irritants, allergens, foods) and can provoke flares

- The skin and gut microbiome play an important role, with eczema flares linked to overgrowth of *Staphylococcus aureus*



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Qu 3: What management options can be used to treat this condition?

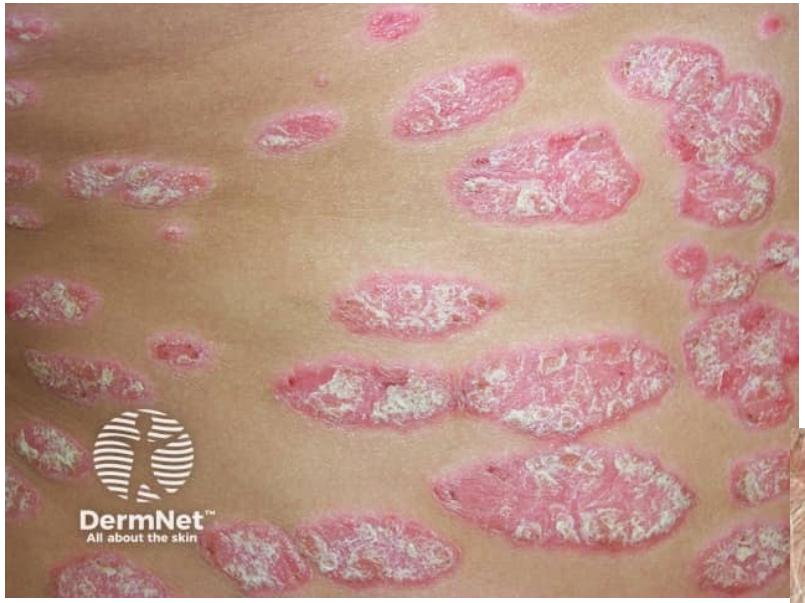
- **The basics**
  - Regular emollient (moisturiser) to rebuild the skin barrier
  - Soap substitute
- **Topical therapies**
  - Topical corticosteroids
  - Topical calcineurin inhibitors
- **Phototherapy**
  - NB-UVB or PUVA
- **Systemic immunosuppressants**
  - Methotrexate
  - Ciclosporin
  - Azathioprine (used less nowadays)
- **Advanced therapies**
  - Biologics (anti-IL4Ra/13/31Ra)
  - JAK inhibitors (anti-JAK1/2/3/TYK2)

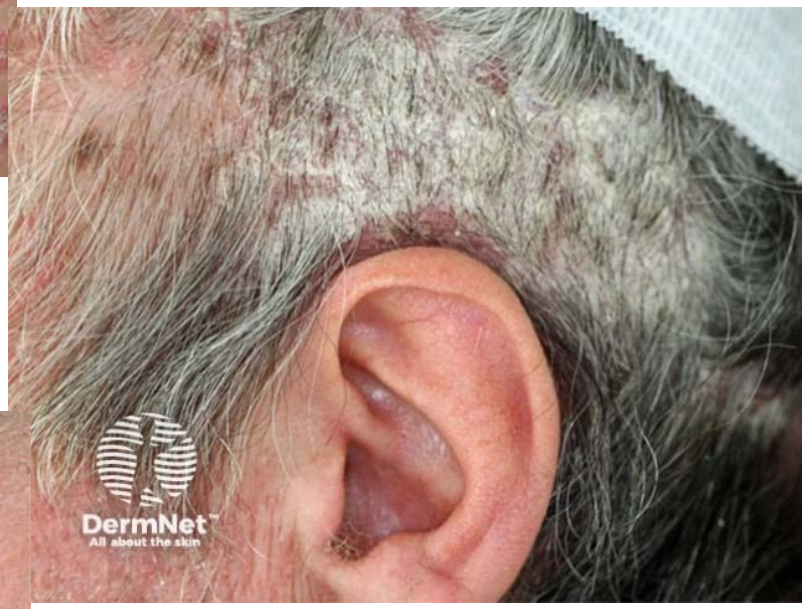
Case 3

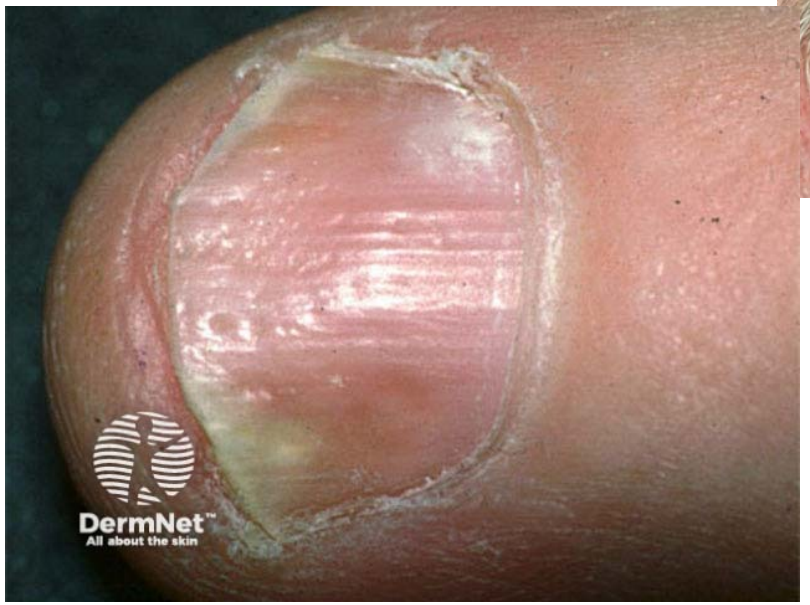
- A 55-year-old male presents with a several year history of a persistent, mildly itchy, raised skin rash, which are gradually increasing in number. He has also developed lower back pain.

- Examination reveals well-demarcated, erythematous plaques with thick silvery-white scale over the extensor surfaces of the elbows, knees, lower back and scalp.

- You also note pitting of the fingernails and lifting of the distal nail plate.



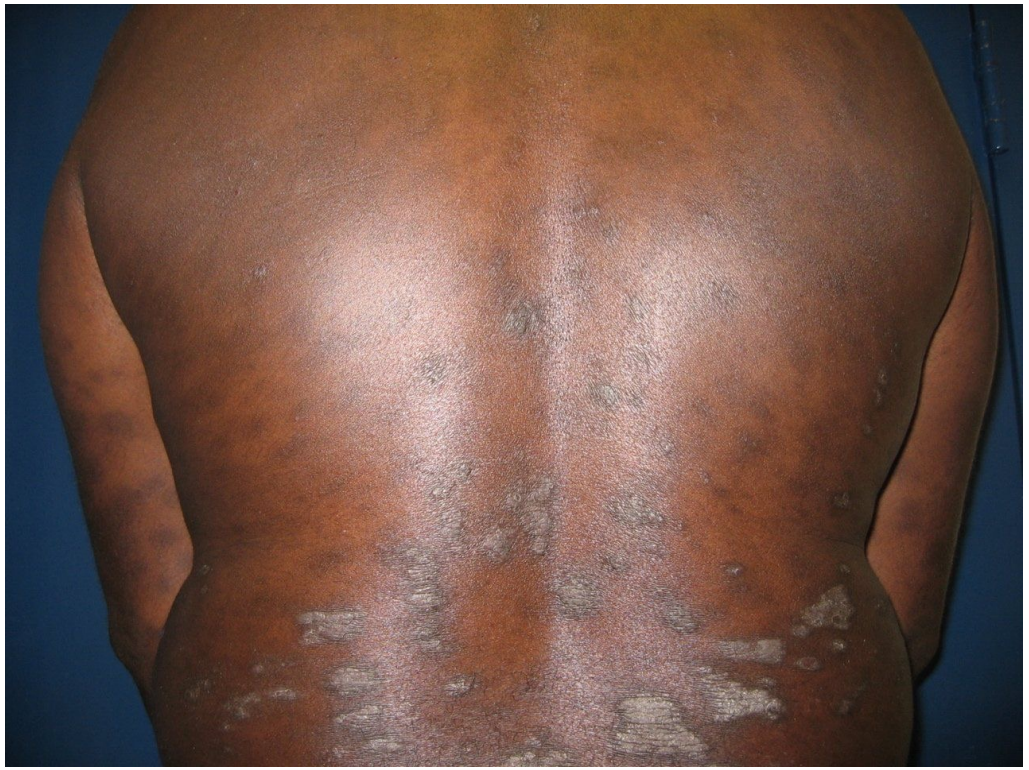




Qu 1: What is the most likely diagnosis?

Psoriasis

- Chronic inflammatory skin condition
- Classified into several types (e.g. chronic plaque, nail, guttate, inverse/flexural, pustular, erythrodermic)
- Can start at any age with peaks at 15–25 years and 50–60 years
- Well-demarcated, inflamed, scaly plaques with extensor distribution. Typically mildly itchy. Other common sites include scalp, nails and lower back/buttocks (NB not scaly in flexural variant)
- Screen for multimorbidity (arthritis, depression, metabolic syndrome)



www.dermatologytimes.com

Qu 2: The final result of cytokine signalling in psoriasis is:

A. Keratinocyte apoptosis

B. Reduced epidermal turnover

C. Loss of melanocytes

D. Suppression of T-cell activity

E. Increased keratinocyte proliferation

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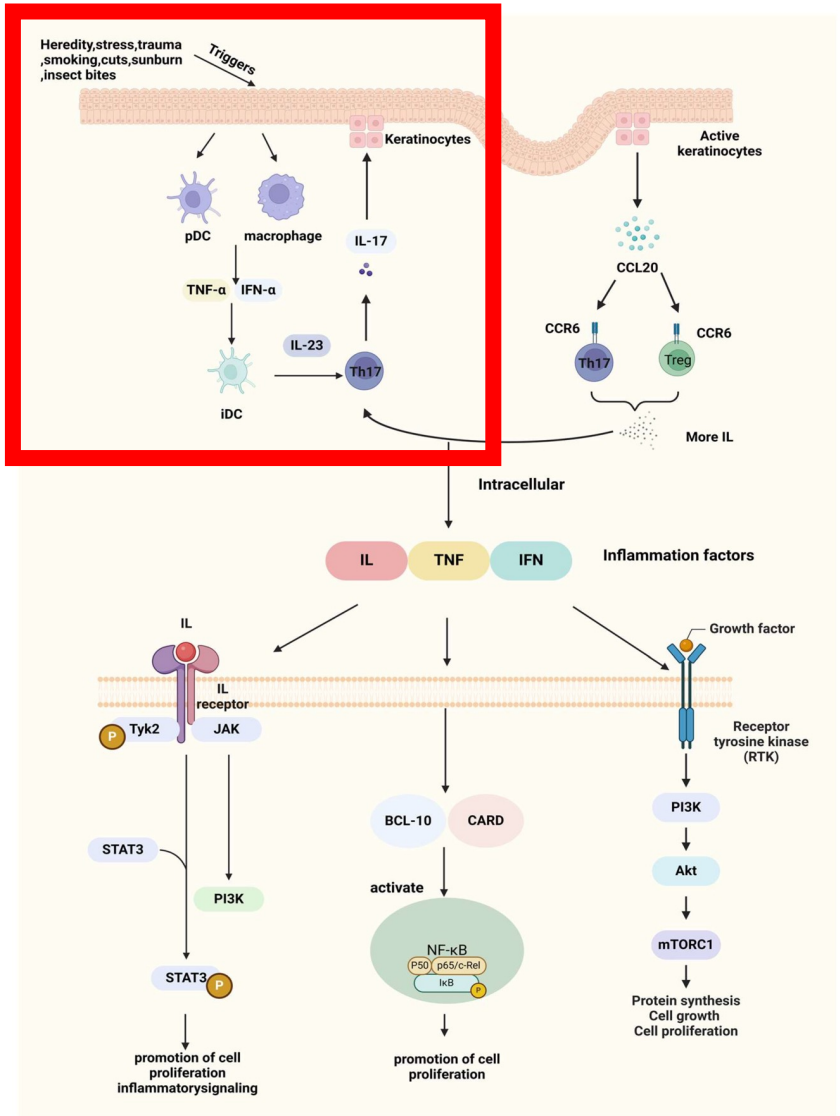
Psoriasis Pathogenesis

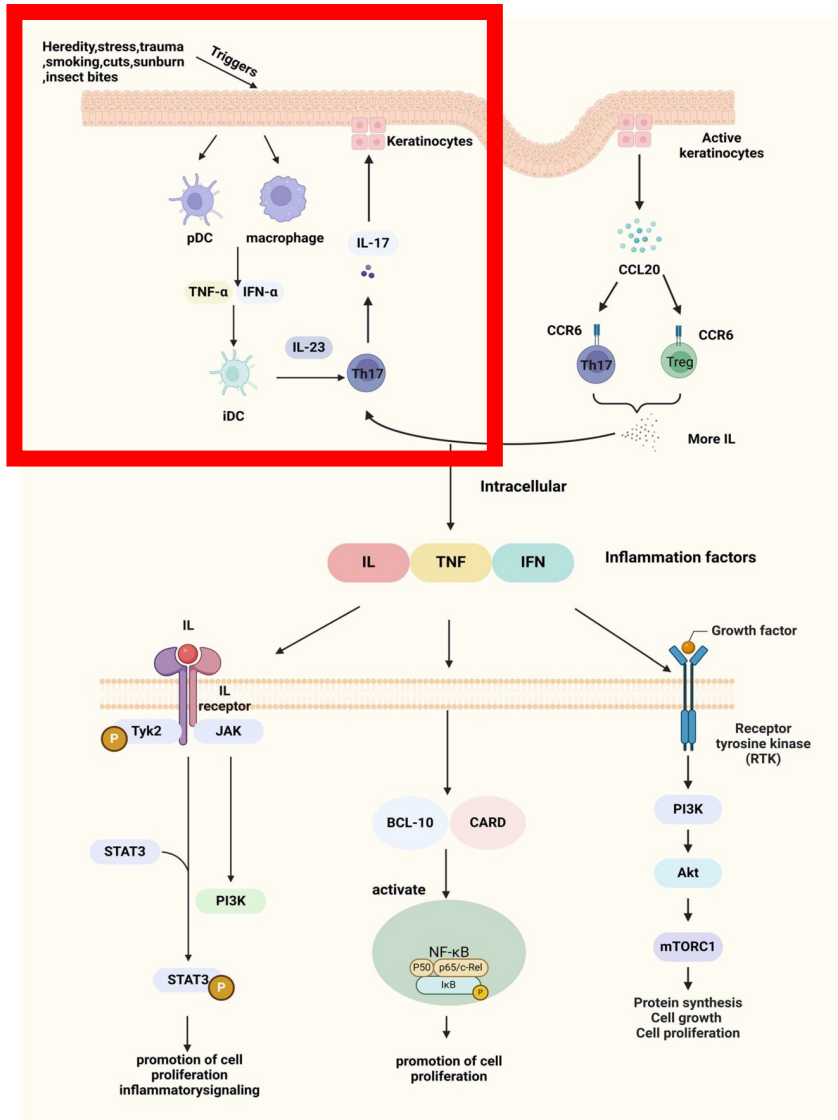
- Triggers include stress, trauma, infection and genetics
- HLA-C*06:02 is the major genetic determinant of psoriasis and associated with specific phenotype (early onset, more severe disease course)
- Triggers cause stressed keratinocytes to release self-nucleic acids and antimicrobial peptides which form complexes
- These complexes activate dermal dendritic cells (DCs) and macrophages, which produce type 1 interferons (e.g. TNF-α, IL-1, IFN-α)



doi: 10.3390/pharmaceutics17010056

Psoriasis Pathogenesis

- Type 1 INFs promotes antigen presentation by dendritic cells
- These dendritic cells migrate to lymph nodes and release IL-23, promoting **Th17** cell differentiation
- **Th17** cells produce IL-17 which recruit inflammatory cells to the skin and further stimulate keratinocytes with excess keratinocyte proliferation and psoriatic plaque formation



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Qu 3: What management options can be used to treat this condition?

- **Lifestyle factors**
  - Alcohol/ smoking cessation
  - Maintaining health weight
- **Mental Health Support**
- **Topical therapies**
  - Regular emollients
  - Topical Vit D analogues
  - Topical corticosteroids
  - Topical calcineurin inhibitors
  - Topical keratolytics
  - Topical coal tar
- **Phototherapy**
  - NB-UVB or PUVA
- **Systemic immunosuppressants**
  - Methotrexate
  - Ciclosporin
- **Other oral therapies**
  - Acitretin
  - Apremilast
  - Deucravacitinib
- **Advanced therapies**
  - Biologics (anti-TNFa, IL-12/23, IL-17 and IL-23)