From Plaques to Progress: The Latest Biologic Therapies for Psoriasis

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Learning Objectives

- Describe the key pathophysiological processes contributing to the development of psoriasis
- Summarize the current treatment guidelines for psoriasis
- Discuss newly approved psoriasis treatments and promising therapies in development

Presentation and Diagnosis of Plaque Psoriasis

- Skin exam
  - Large oval/circle plaques
  - Common plaque locations – torso, scalp, elbows, knees
  - Special sites: face, scalp, hands, feet, genitals/buttocks, skin folds
  - BSA
- Symptoms
  - Itching, flaking, thick scales, bleeding, pain
  - +/- Joint symptoms
- Biopsy
  - Not necessary, but may be done in more challenging cases[1]

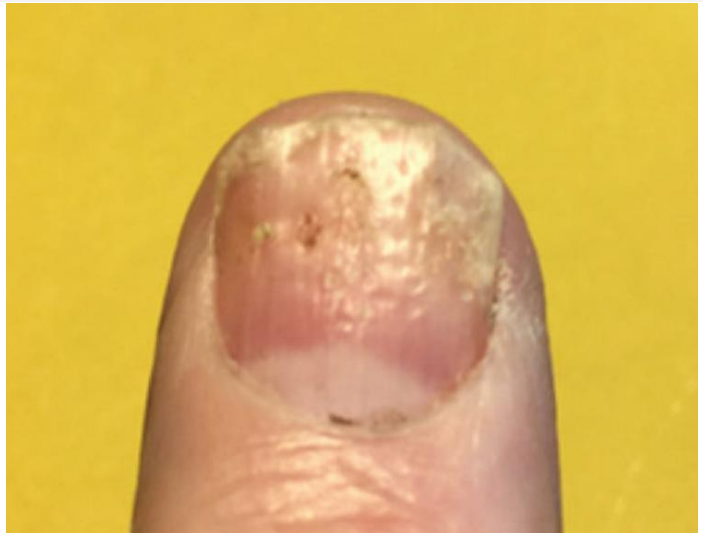


Image of nail psoriasis (above)[2]

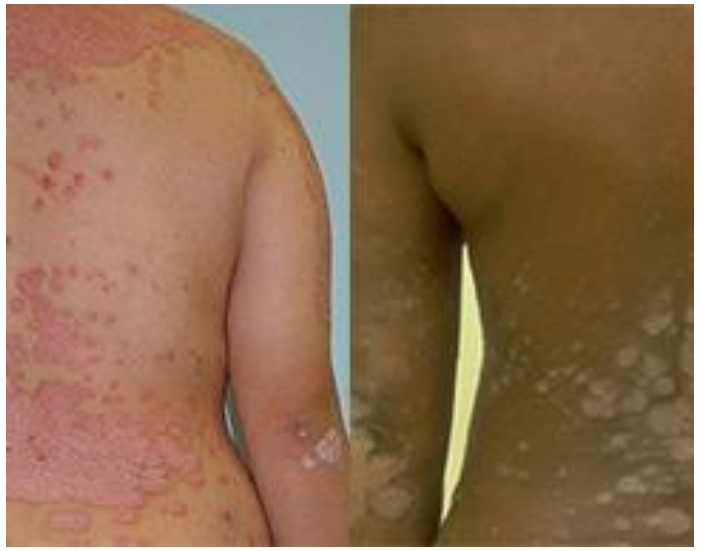


Image showing plaque psoriasis on two different skin types (above)[3]

Etiology

Genetics - >80 different genes have been identified

- HLA-C*06:02 gene at the PSORS1 loci along with , IL12B, IL23R, and LCE3B/3C[4]

Stress

- Pregnancy, life stressors, surgery, trauma, infections

Alcohol, smoking

Obesity[5]

Medications

- Beta blockers, lithium, antimalarials, interferons, imiquimod, and terbinafine[6]

Epidemiology

- >8 million people in the US and >125 million people worldwide have psoriasis
- 2-3% of the world population
- More common in Caucasians
- Equally affects males and females
- Have two different peak ages
  - 20-30 years old
  - 50-60 years old[7,8]

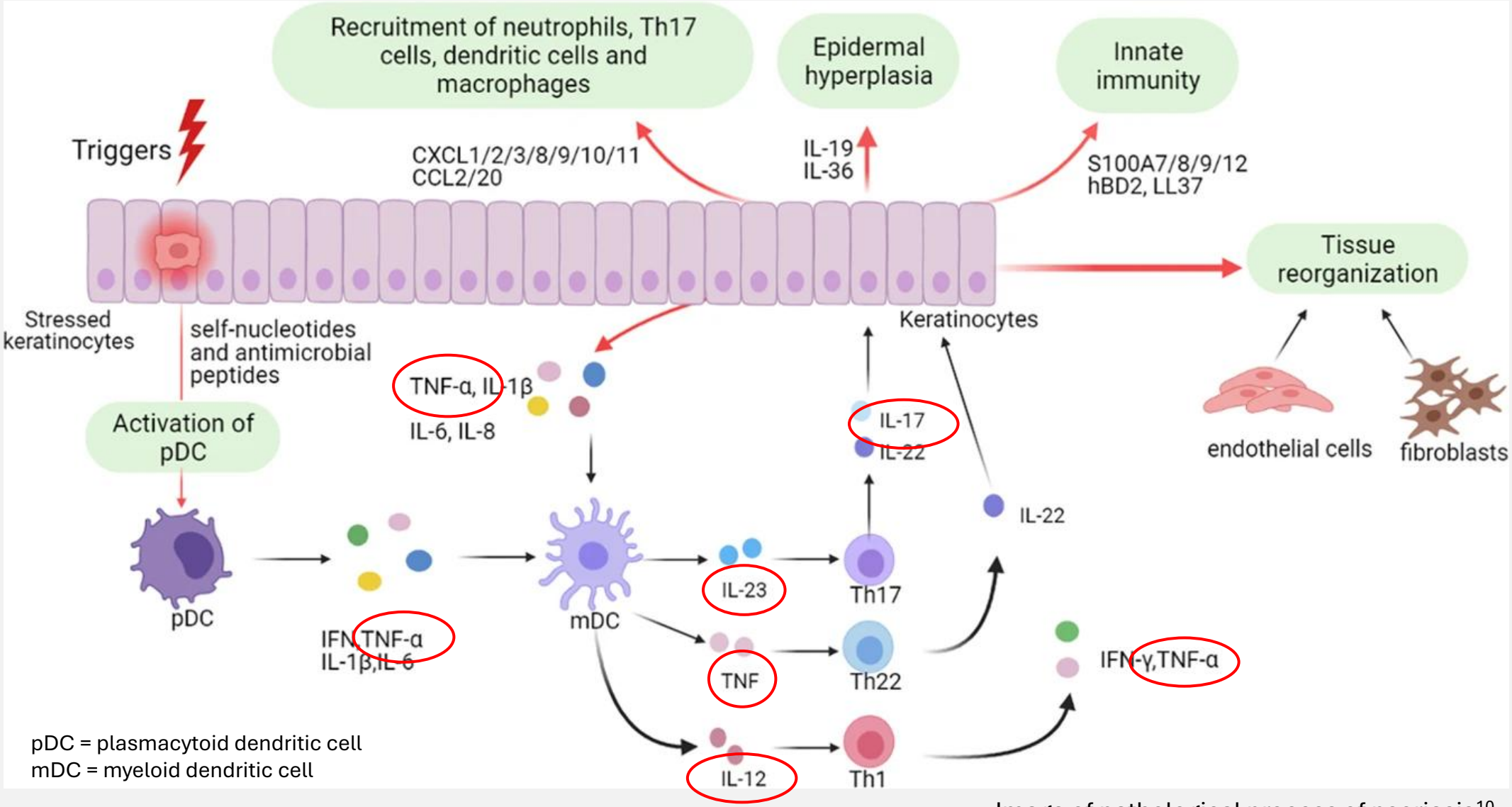
Pathophysiology

- Psoriasis vulgaris is caused by dysregulation of the immune system – including hyperactivation of T cells and increased pro-inflammatory cytokine release leading to hyperproliferation of the keratinocytes in the epidermis along with an increase in epidermal cell turnover[9]

Key:

- T cells: normally fight infection (in this case they misidentify our skin cells as something that needs attacked)
- Pro-inflammatory cytokines: cause inflammation and can stimulate the synthesis of other cytokines to the area (TNF alpha, IL17, IL23, IL12/23)
- Keratinocytes: major cell type found in the epidermis
- Epidermis: outermost layer of skin

Pathophysiology



pDC = plasmacytoid dendritic cell
mDC = myeloid dendritic cell

Image of pathological process of psoriasis[10]

Checkpoint 1

True or false: Psoriasis vulgaris is caused by dysregulation of the immune system –including hyperactivation of T cells and increased pro-inflammatory cytokine release leading to hyperproliferation of the keratinocytes in the epidermis along with an increase in epidermal cell turnover

Atopic Dermatitis vs Plaque Psoriasis

Plaque Psoriasis

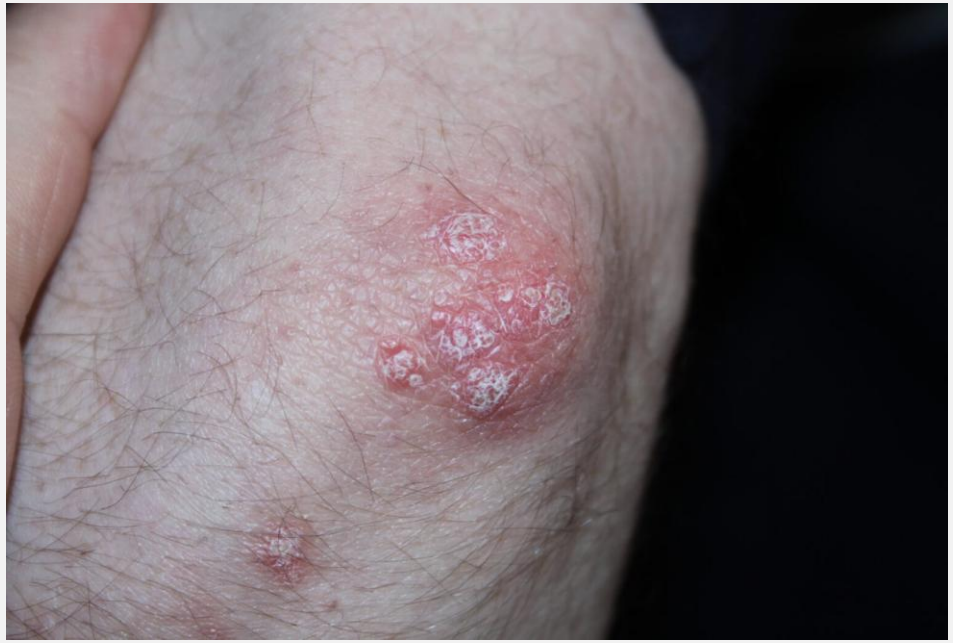
- Distinct border, raised silvery-white or red scales
- Favor the elbows and knees
- Burn, sting, mild itch
- Autoimmune process acts more like an infectious response
- Treated with TNF alpha, IL 17, IL23, IL23/23 inhibitors
- Females=Males
- Adulthood

Both

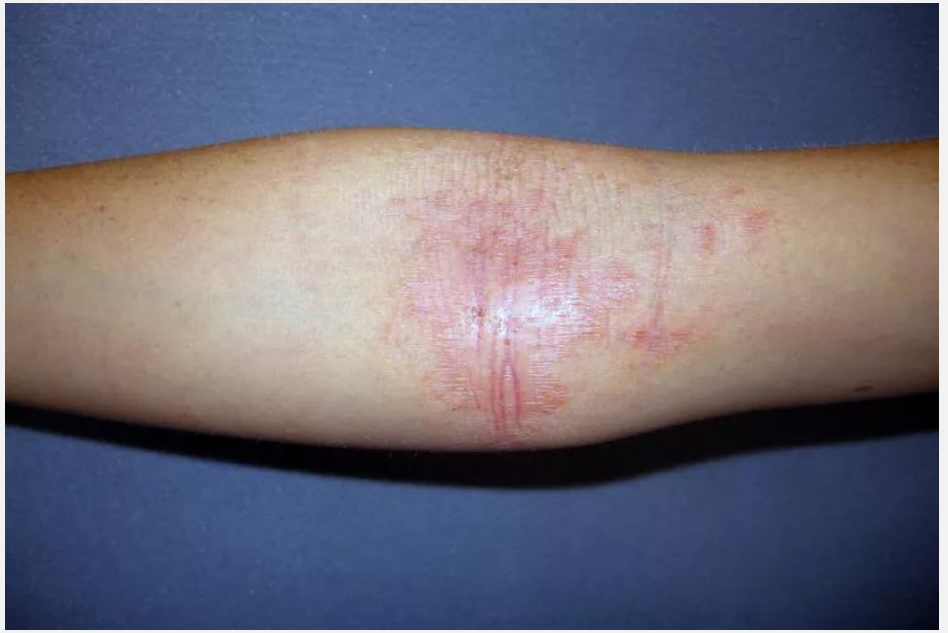
- Occur anywhere on the body, dry looking
- Itch
- Seem to have genetic + environmental triggers
- Both can be treated with topical corticosteroids, phototherapy, and biologics

Atopic Dermatitis

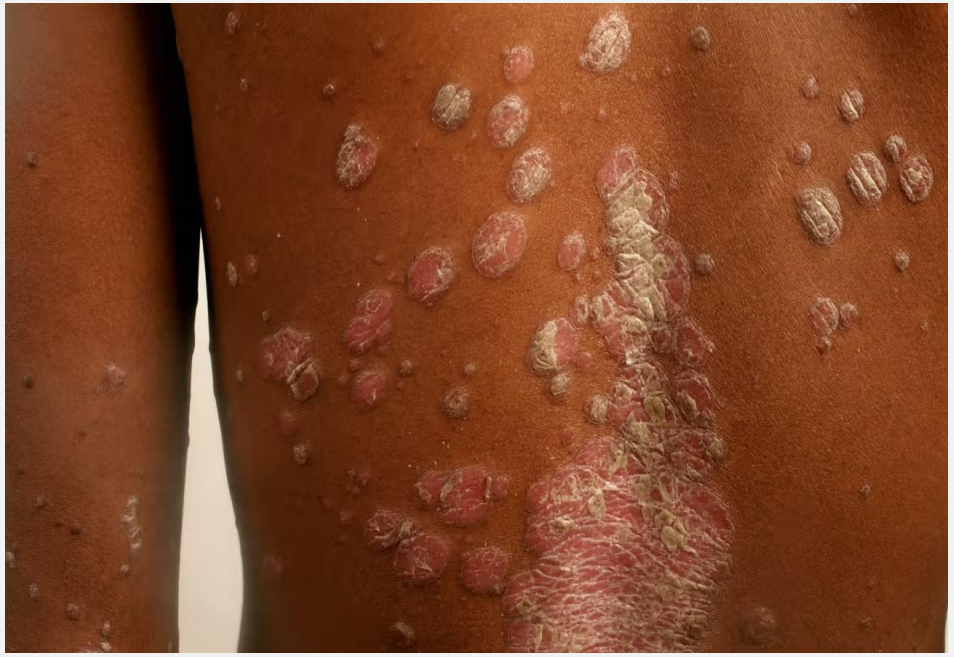
- No distinct borders, red bumps/blisters
- Favors front of the elbows and behind the knees (areas that bend)
- Very itchy
- Mutation in the filaggrin gene, plays a role in the skin barrier – skin more likely to be dry
- Autoimmune response more allergic pathway
- Treated with IL4, IL13, IL31, and JAK inhibitors
- Females>males
- Childhood[11]

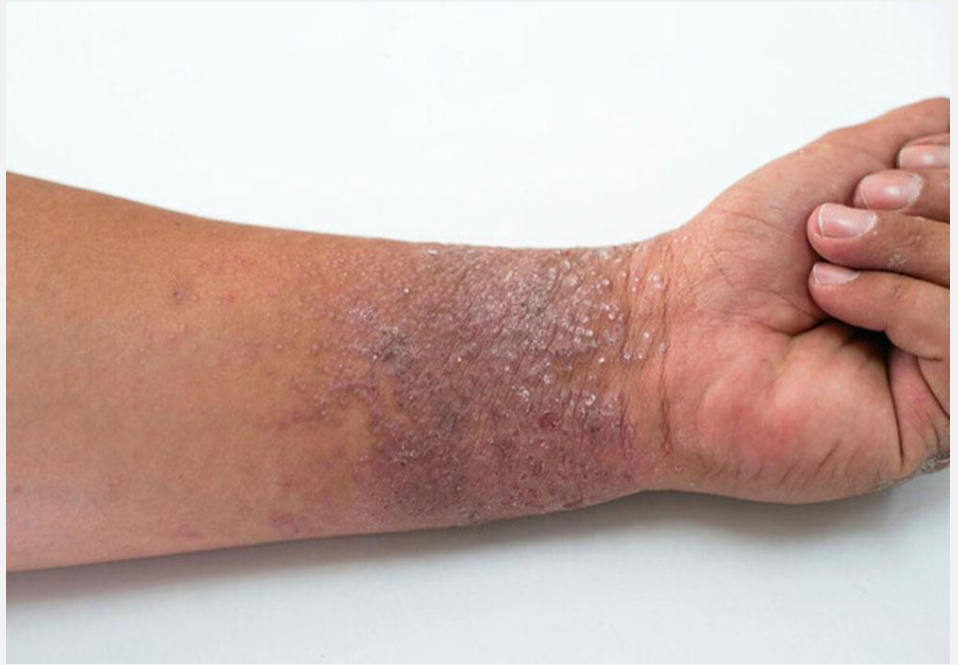


Plaque Psoriasis – above[12] and below[13]



Atopic Dermatitis – above[14] and below[15]





Treatment Guideline for Mild-Moderate Psoriasis

- Mild = BSA <3%, Moderate = 3-10% BSA
  - Topicals corticosteroids
    - 1st line: medium to high potency for at least 4 weeks before adding on a steroid-sparing topical
      - Examples of medium potency: triamcinolone 0.1% cream
      - Examples of high potency: betamethasone dipropionate 0.05% cream
    - Low potency steroids for face and intertriginous areas
      - Examples of low potency: desonide 0.05% cream, fluocinolone 0.01% oil, hydrocortisone 1% cream
      - Super-high potency for thicker plaques
      - Examples of super-high potency: betamethasone dipropionate 0.05% ointment, clobetasol 0.05% cream, halobetasol 0.05% cream
    - Mechanism: anti-inflammatory, antiproliferative, immunosuppressive, and vasoconstrictive effects
    - Risks: steroid atrophy, striae, folliculitis, telangiectasia, purpura, worsening of psoriasis after stopping[16]

Treatment Guideline for Mild-Moderate Psoriasis

(continued)

- Vitamin D Analogs
  - Example: calcipotriene 0.005% cream
- Mechanism: binding to vitamin D receptors, which inhibit keratinocyte proliferation and enhance keratinocyte differentiation
- Can be used as a steroid-sparing option
- Can use long term[16]

Guidelines for Non-biologic Systemic Therapies

- Methotrexate (off-label)
  - Mechanism: competitive inhibitor of dihydrofolate reductase, decreasing folate cofactors required for the synthesis of nucleic acids
  - Dosing: oral/IM/subcutaneously once **weekly** (7.5-25 mg) + folic acid on days that MTX is not being administered
  - Monitoring: acute hepatitis panel, TB test, LFTs and CBC at baseline, then CBC and LFTs every 3-6 months
  - Risks: hepatotoxicity, immunosuppression, myelosuppression, GI/pulmonary /dermatologic toxicities
  - BBW: adverse reactions (listed above), pregnancy (teratogenic), and hypersensitivity
- Acitretin
  - Oral retinoid, vitamin A derivative
  - Mechanism: not fully understood, possible modulation of epidermal differentiation and proliferation along with has anti-inflammatory and immunomodulatory effects
  - Dosing: 10-50 mg daily
  - Not immunosuppressive
  - Monitoring: Lipid profile and LFTs (baseline and at 4-week intervals for the first 3 months, then every 3 months), CBC and renal function tests (baseline, then every 12 weeks)
  - BBW: pregnancy (teratogenic), hepatotoxicity[17]

Guidelines for Non-biologic Systemic Therapies

(continued)

- Cyclosporine
  - Mechanism: potent immunosuppressant that functions by binding cyclophilin, which then inhibits calcineurin and blocks proinflammatory signaling
  - Dosing: 1 to 3 mg/kg/day in 2 divided doses, titrate up to a max of 4 mg/kg/day
  - Not recommended for long term use due to serious adverse effects – nephrotoxicity, hypertension
- Apremilast (Otezla)
  - Mild to moderate psoriasis
  - ESTEEM 1 trial showed benefit for scalp and palmoplantar involvement
  - Dosing: titration pack up to 30 mg twice daily, if CrCl <30 mL/min then 30 mg daily
  - Mechanism: PDE4 inhibitor leading to an increase in intracellular cAMP levels leading to a down regulation of T helper and interferon pathways and increase in IL10 which has anti-inflammatory properties
  - Monitoring: renal function occasionally
  - Warnings/precautions: depression, dehydration, respiratory tract infections, diarrhea, nausea/vomiting, headache, weight loss
  - Drug interactions – avoid use with strong CYP450 inducers
  - Not immunosuppressive
  - 12-16 weeks to work[17]

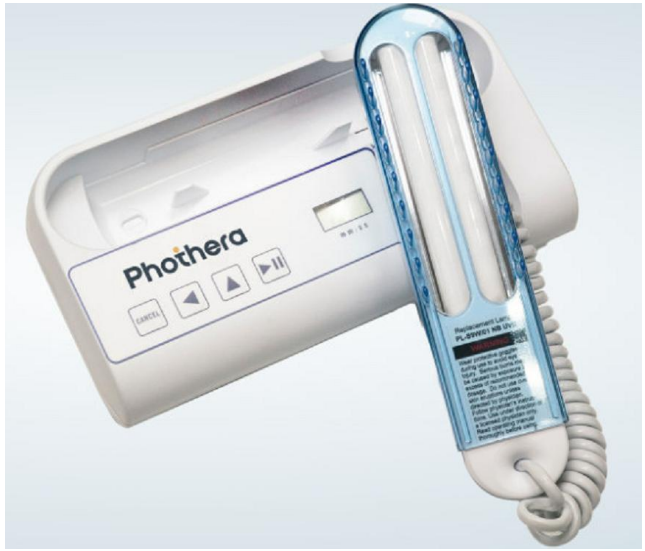
Phototherapy

- Narrowband UVB preferred over psoralen plus UVA (PUVA)
- Treatment is determined based on Fitzpatrick Skin Type
- Treatments are 2-3 times per week
- Can be done in clinic or at home
- Used as monotherapy or in combination with other treatments (topicals or oral agents[18]

Skin type	Skin color	Characteristics
I	White, very fair, red or blond hair, blue eyes, freckles	Always burns, never tans
II	White; fair; red or blond hair; blue, hazel, or green eyes	Usually burns, tans with difficulty
III	Cream-white, fair with any eye or hair color	Sometimes mild burn, gradually tans
IV	Brown, typical Mediterranean white skin	Rarely burns, tans with ease
V	Dark brown, Middle Eastern skin types	Very rarely burns, tans very easily
VI	Black	Never burns, tans very easily

Image of Fitzpatrick Skin Type[18]

Light Units



Handheld light unit[19]

Full body 3D unit image[19]

Hand/foot unit[19]

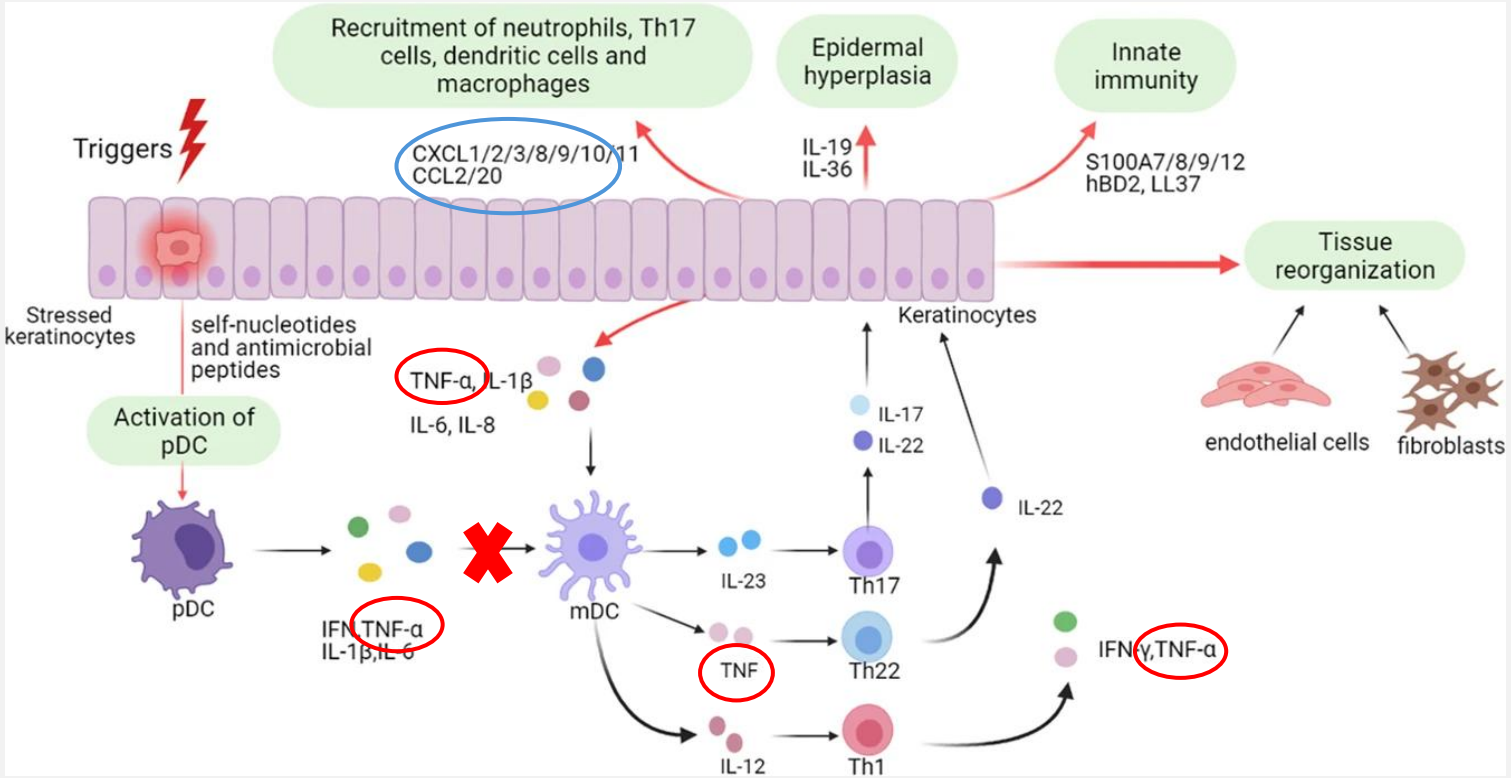
Biologic Guidelines

- Moderate to severe psoriasis
  - Moderate = 3-10% BSA, severe >10% BSA
- Biologic should be tailored to the individual
  - What other comorbidities do they have?
  - Contraindications?
  - Allergies?
  - What therapies have they tried and failed?
  - Dosing schedule?[20]

TNF-α Inhibitors as a Class

adalimumab (Humira and biosimilars), etanercept (Enbrel), infliximab (Remicade and biosimilars), certolizumab (Cimzia)

- Mechanism: decrease pro-inflammatory cytokines, reduce T cell activation, and reduce number of blood cells going to tissue
- Side effects: injection site reactions, infection risk
- All approved for psoriatic arthritis
- Warnings/precautions/counseling:
  - No live vaccines
  - May need to hold for surgery (pending risk) or during illness
  - Risk for antibody development
  - Lab monitoring: CBC/D, CMP, acute hepatitis panel, hepatitis B surface antibody, and TB test at baseline; annual TB test
  - BBW: infection, malignancy
  - Avoid: cancer within the past 5 years, heart failure, demyelinating diseases, hepatitis B[20]

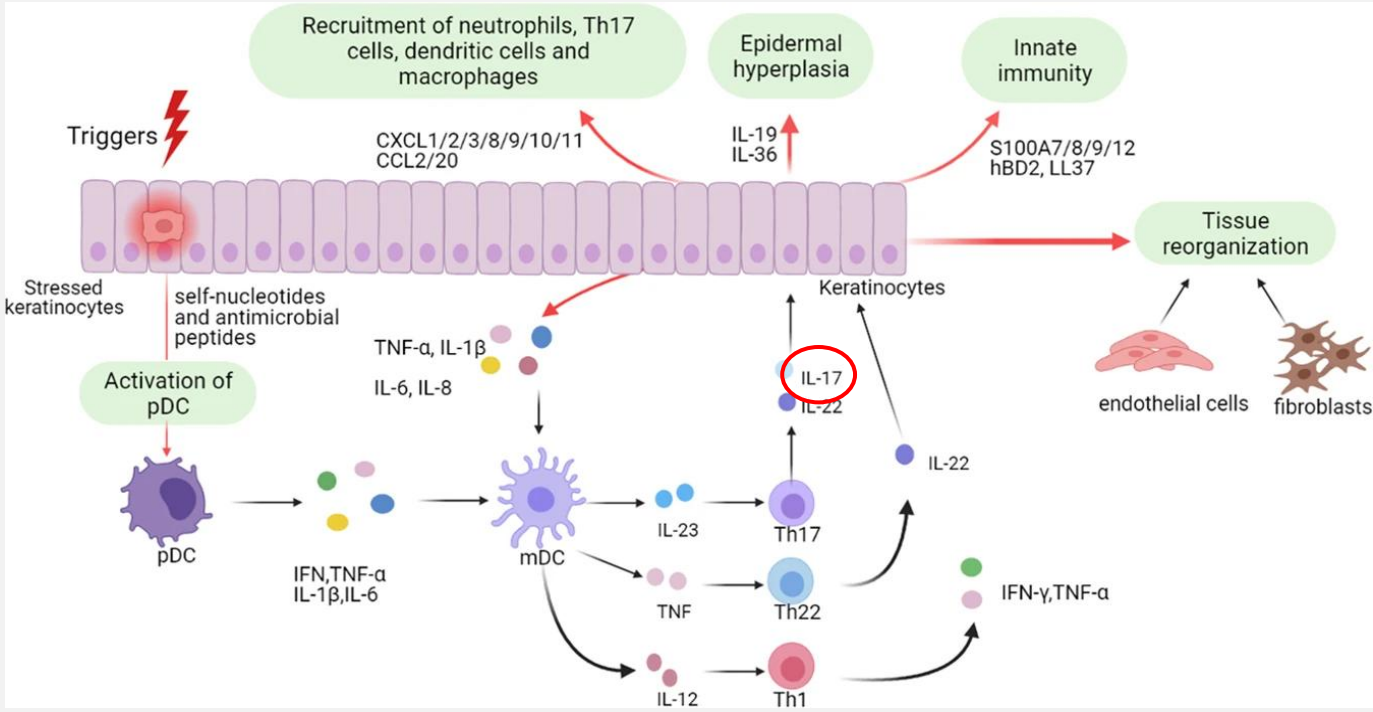


	adalimumab (Humira and biosimilars)[20]	etanercept (Enbrel)[20]	infliximab (Remicade and biosimilars)[20]	certolizumab (Cimzia)[20]
Dosing	80 mg subcutaneously x 1 dose, then 40 mg every other week starting 1 week after initial dose	50 mg subcutaneously twice weekly for three months, then 50 mg weekly	5 mg/kg IV at 0, 2, and 6 weeks, followed by 5 mg/kg every 8 weeks thereafter	400 mg subcutaneously every other week (can consider lower dose in patients ≤90 kg)
Formulation	Pen, syringe	Pen, syringe	Infusion	Syringe
Pearls	• Has biosimilars • 3-4 months to effect	• Fallen out of favor as newer agents are more effective • 3-4 months to effect	• Has biosimilars • 8-10 weeks for effect	• Preferred agent in pregnancy – pegylated and dose not cross the placenta • 3-4 months to effect

IL17 Inhibitors as a Class

secukinumab (Cosentyx), ixekizumab (Taltz), brodalumab (Siliq)

- Mechanism: all work at IL17
  - Cosentyx + Taltz: bind to IL17A to prevent it from binding the receptor and causing less inflammation and keratinocytes
  - Siliq: blocks the receptor on IL17A, more broad mechanism and more immunosuppression
- Side effects: injection site reactions, infection risk
- Warnings/precautions/counseling:
  - No live vaccines
  - May need to hold for surgery (pending risk) or illness
  - Risk for antibody development
  - Lab monitoring: CBC/D, CMP, acute hepatitis panel, hepatitis B surface antibody, and TB test at baseline; annual TB test
  - Avoid: patients with Crohn's/IBD/UC, pregnancy, cancer within the past 5 years[20]

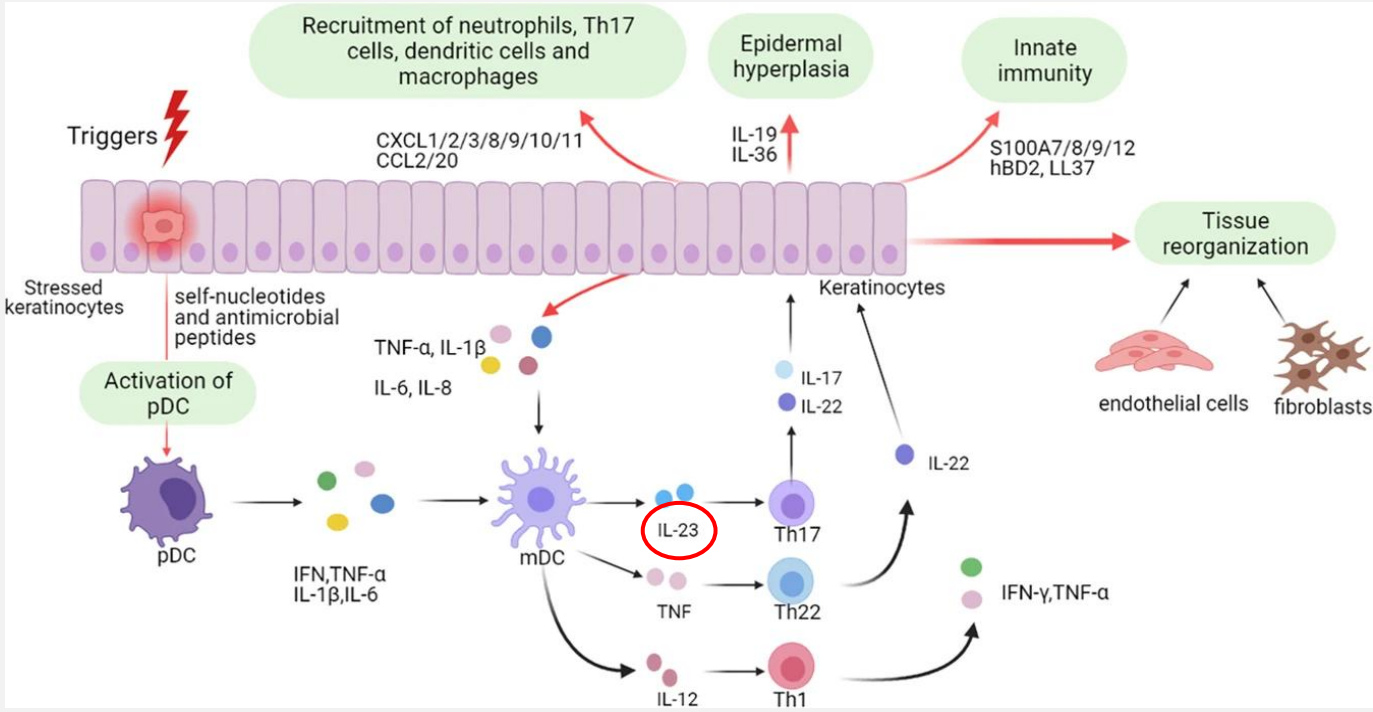


	secukinumab (Cosentyx)[20]	ixekizumab (Taltz)[20]	brodalumab (Siliq)[20]
Dosing	300 mg subcutaneously once weekly at weeks 0, 1, 2, 3, and 4 followed by 300 mg every 4 weeks	160 mg subcutaneously once, followed by 80 mg at weeks 2, 4, 6, 8, 10, and 12, and then 80 mg every 4 weeks	210 mg subcutaneously at weeks 0, 1, and 2, followed by 210 mg once every 2 weeks
Formulation	Pen, syringe	Pen, syringe	Syringe
Pearls	• Approved in psoriatic arthritis • 12 weeks to see full effect	• Approved in psoriatic arthritis • Neutropenia • Can show effectiveness as early as 1-2 weeks, but 12 weeks to see full effect	• NOT approved in psoriatic arthritis • BBW for suicidal ideation/behavior • 12 weeks to see full effect

IL23 Inhibitors as a Class

guselkumab (Tremfya), risankizumab-rzaa (Skyrizi), tildrakizumab (Ilumya)

- Mechanism: prevents interaction with IL23 receptor leading to less inflammation and keratinocyte hyperproliferation, also prevents production of downstream proinflammatory cytokines (IL17)
- Side effects: injection site reactions (least common), infection risk (lowest)
- Warnings/precautions/counseling:
  - No live vaccines
  - May need to hold for surgery (pending risk) or illness
  - Risk for antibody development
  - Lab monitoring: CBC/D, CMP, acute hepatitis panel, hepatitis B surface antibody, and TB test at baseline; annual TB test
  - Avoid: pregnancy, cancer within the past 5 years[20]

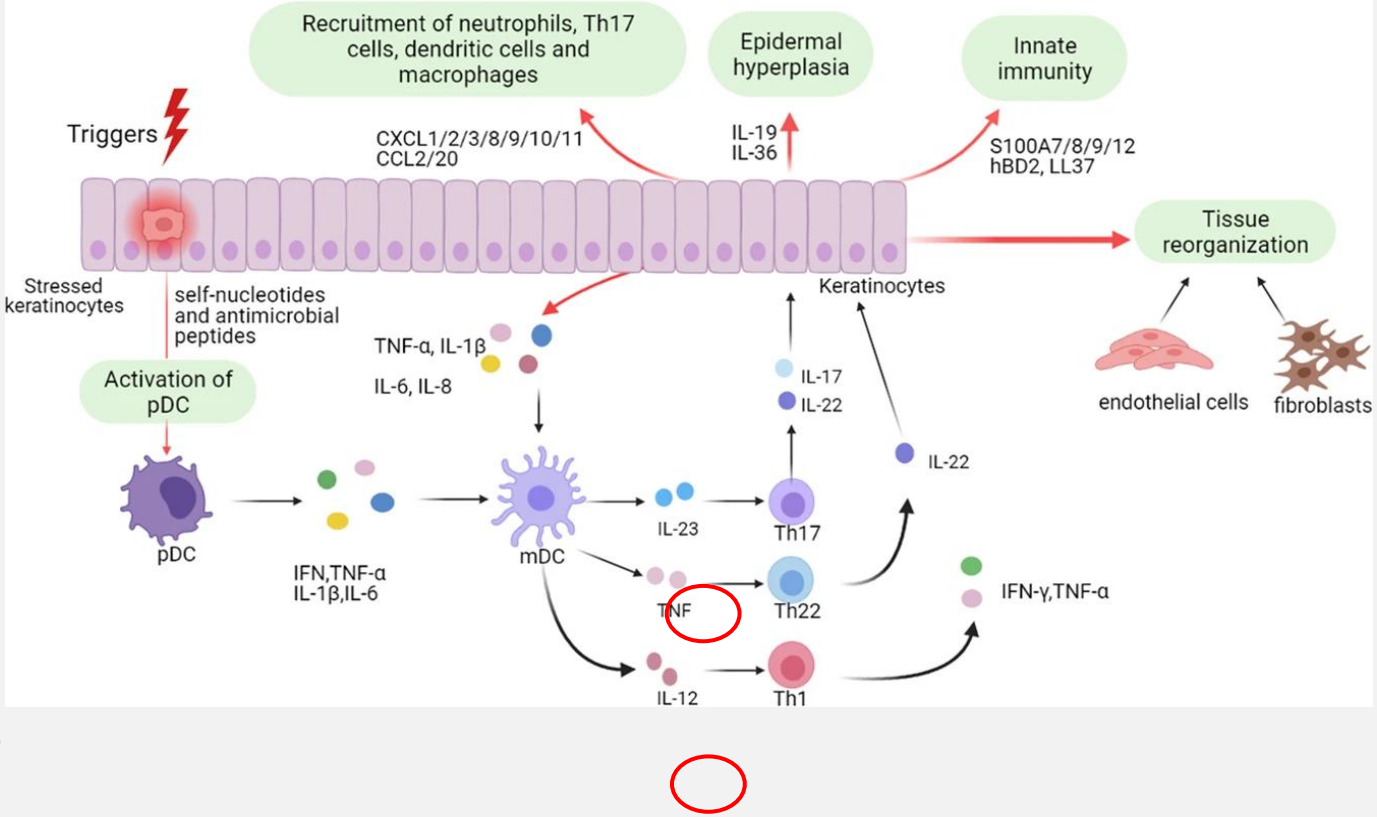


	guselkumab (Tremfya)[20]	risankizumab-rzaa (Skyrizi)[20]	tildrakizumab (Ilumya)[20]
Dosing	100 mg at weeks 0, 4, and then every 8 weeks thereafter	Prefilled syringe and auto-injector: 150 mg at weeks 0, 4, and then every 12 weeks thereafter	100 mg subcutaneously at weeks 0, 4, and then every 12 weeks thereafter
Formulation	Pen, syringe	Pen, syringe	Syringe
Pearls			• Must be administered by a healthcare provider

IL12/23 Inhibitors

Ustekinumab (Stelara and biosimilars)[20]

- Mechanism: binds with IL-12 and IL-23 to block proinflammatory cytokines like IL17
- Side effects: injection site reactions, infection risk
- Dosing (syringes only):
  - ≤100 kg: SUBQ: 45mg at 0 and 4 weeks, and then 45mg every 12 weeks thereafter
  - >100 kg: SUBQ: 90mg at 0 and 4 weeks, and then 90mg every 12 weeks thereafter.
- Warnings/precautions/counseling:
  - No live vaccines
  - May need to hold for surgery (pending risk) or illness
  - Risk for antibody development
  - Lab monitoring: CBC/D, CMP, acute hepatitis panel, hepatitis B surface antibody, and TB test at baseline; annual TB test
  - Avoid: pregnancy, cancer within the past 5 years



Biologics Approved in Pediatrics[21]

Approved	Not Approved
Apremilast (Otezla) - >6yo, weighing >20kg	Adalimumab (Humira and biosimilars)
Etanercept (Enbrel) - >4yo	Infliximab (Remicade and biosimilars)
Ixekizumab (Taltz) - >6yo	Certolizumab (Cimzia)
Secukinumab (Cosentyx) - >6yo	Brodalumab (Siliq)
Ustekinumab (Stelara and biosimilars) - >6yo	Guselkumab (Tremfya)
	Risankizumab-rzaa (Skyrizi)
	Tildrakizumab (Ilumya)

Checkpoint 2

Sue Riasis is a 70-year-old female that has a diagnosis of psoriasis vulgaris. Over the past several years she has tried/failed several high potency topical steroids, phototherapy, and calcipotriene. Her dermatologist comes into your office to ask for advice on which injectable biologic to start based on her past medical history of DM2, HTN, CAD, Crohn's disease, and stage 3 CKD. Which of the following classes of biologics would you **AVOID** and why?

a. TNF-alpha inhibitors due to stage 3 CKD
b. IL23 inhibitors due to CAD
c. IL17 inhibitors due to Crohn's disease
d. Both IL17 and IL23 inhibitors due to Crohn's disease

Newer Agents

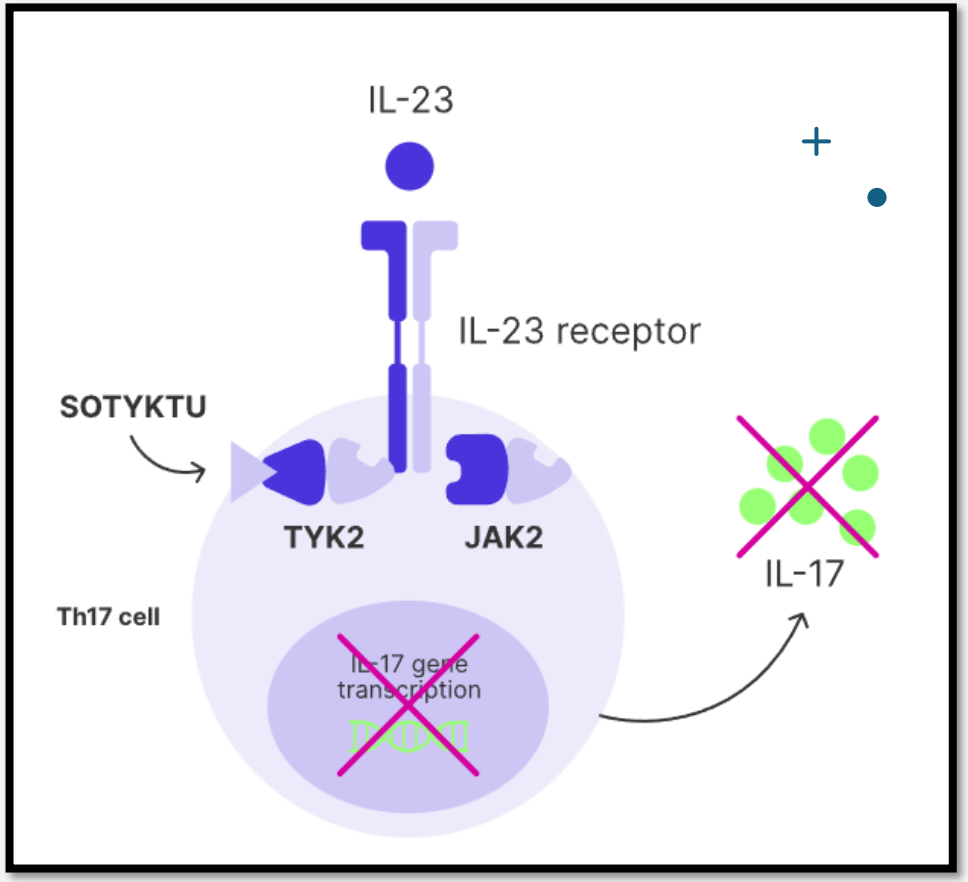
Bimekizumab (Bimzelx)

- Class: IL17 inhibitor
- Trial: BE-READY
- Mechanism: dual inhibition of IL17A and IL17F leading to decreased cytokine activity and more effective inflammatory suppression
- Dosing: 320 mg subcutaneously once every 4 weeks for the first 16 weeks (5 doses), and then every 8 weeks thereafter
    - In obesity: ≥120 kg: 320 mg subcutaneously once every 4 weeks
- Dosage forms: pen, syringe
- Monitoring: same baseline labs as the other biologics + LFTs every 3-6 months
- Side effects: antibody development, infection
- Pearls:
    - Not approved in pediatrics
    - Avoid in patients with IBD/Crohn's/UC, depression, or pre-existing liver dysfunction[22]

Newer Agents

Deucravacitinib (Sotyktu)

- Class: tyrosine kinase 2 inhibitor (part of the JAK family)
- Trial: POETYK PSO-1
- Mechanism: binds to TYK2 domain which mediates signaling of IL-23, IL-12, and Type I interferons
- Dosing: 6 mg by mouth daily
- Dosage forms: tablet
- Side effects: infection
- Monitoring: TB test, acute hepatitis panel, LFTs, and triglycertides at baselines. LFTs and triglycerides periodically thereafter
- Warnings/precautions: MACE, LFT elevations, rhabdomyolysis/CPK elevations, thrombosis, malignancy
- Pearls: only JAK inhibitor approved for psoriasis[23]



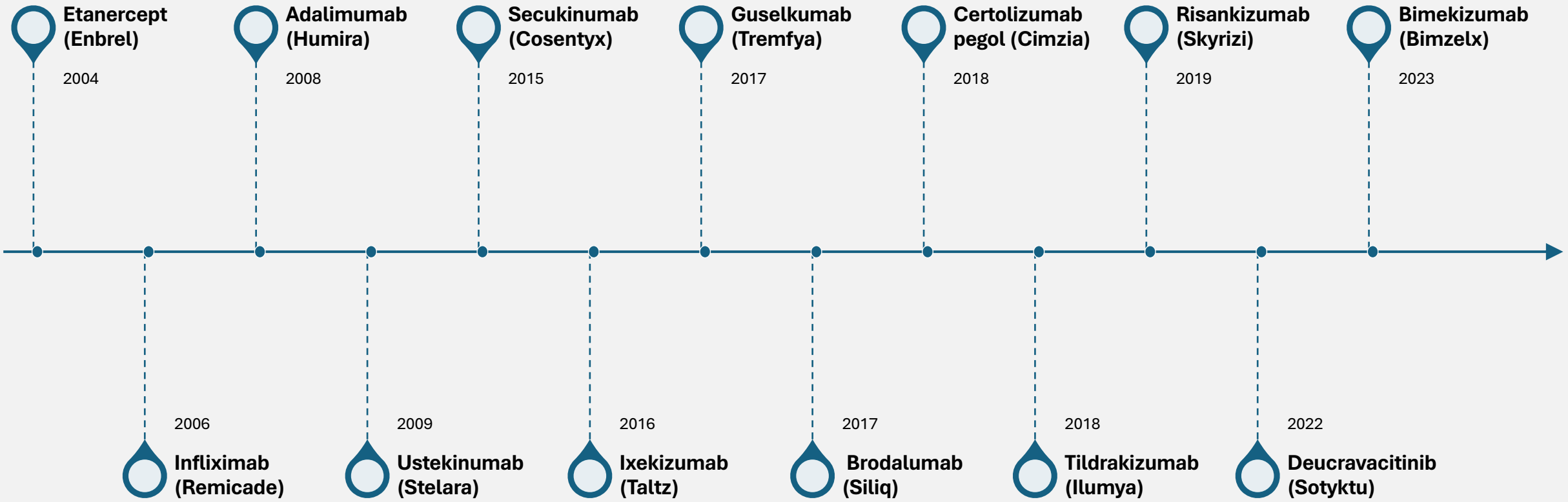
Sotyktu mechanism of action image[24]

Checkpoint 3

Which newer agent for the treatment of psoriasis has a novel pathway characterized by selectively inhibiting TYK2, a kinase involved in the IL-23 and IL-17 inflammatory pathways?

a. Deucravacitinib
b. Bimekizumab
c. Roflumilast
d. Guselkumab

Timeline


Etanercept (Enbrel)
2004
Infliximab (Remicade)
2006
Adalimumab (Humira)
2008
Ustekinumab (Stelara)
2009
Secukinumab (Cosentyx)
2015
Ixekizumab (Taltz)
2016
Guselkumab (Tremfya)
2017
Brodalumab (Siliq)
2017
Certolizumab pegol (Cimzia)
2018
Tildrakizumab (Ilumya)
2018
Risankizumab (Skyrizi)
2019
Deucravacitinib (Sotyktu)
2022
Bimekizumab (Bimzelx)
2023

Agents in the Pipeline

- Oral IL23 inhibitor (Icotrokinra)
  - Being studied in adolescents (>12yo) and adults
  - "84.1% of adolescent patients treated with once daily Icotrokinra achieved an Investigator's Global Assessment (IGA) score of 0/1) and 70.5% achieved a Psoriasis Area and Severity Index (PASI) 90 response, compared to 27.3% and 13.6% receiving placebo, respectively, at Week 16"
  - "Response rates continued to improve through Week 24 where 86.4% of adolescents achieved IGA 0/1 and 88.6% achieved PASI 90.[1] Further, at Week 24, 75% of adolescents achieved IGA 0 (completely clear skin) and 63.6% achieved PASI 100"
  - Favorable safety profile[24]
- A3 Adenosine Receptor Agonist (Piclidenoson)
  - A3 adenosine receptor agonist that inhibits production of interleukin-17 and interleukin-23 in keratinocytes
  - Study was limited due to high withdrawal rate due to COVID-19 - did not meet primary endpoint of at least 75% improvement
  - More trials needed[25]
- PDE4 inhibitors (Orismilast)
  - Differs from Otezla because of higher potency, enhanced selectivity for PDE4 subtypes, broader anti-inflammatory effects, and a modified release formulation designed to reduce GI side effects

Tying it All Together

- Pharmacist role
  - Make this process smooth
  - Assist provider in therapy choice, lab monitoring, and follow up
  - Staying up to date with current guidelines and what may be coming down the pipeline
- So how do we get our patients from "plaques to progress"?
  - Understand plaque psoriasis
  - Find a biologic that works for the patient – this isn't always achieved on the first try
  - Be honest with length of time that it may take to see results
  - Assist with affordability (copay card vs manufacturer assistance vs M3P program)
  - Counseling – side effects, injection technique, disposal
  - Follow up with patients

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