



# DERMATOLOGY DRUGS FOR INFLAMMATORY CONDITIONS:

MECHANICS, INDICATIONS, AND SAFETY

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Did you know that up to **20% of dermatology prescriptions are misused** due to a lack of understanding about their mechanisms of action or side effects?

Many prescribing errors result from a limited understanding of mechanisms of action, indications, or potential side effects.

Primary care clinicians are key to bridging this knowledge gap and ensuring safe, effective treatments.

# Understanding Dermatology Pharmacology

## Learning Objectives:

-  Understand common dermatology drug classes (MOA, indications, side effects)
-  Learn to select appropriate therapies based on patient factors
-  Enhance confidence in managing common dermatologic conditions in primary care

# Dermatology Medications Simplified

## Key Drug Categories:

- Corticosteroids
- Antibiotics
- Immunomodulators
- Biologics

## Significance in Dermatology:

- Reduce inflammation
- Improve patient outcomes
- Target specific immune pathways

# Corticosteroids:

## Key Anti-Inflammatory Agents in Dermatology

**Primary Action:** Potent anti-inflammatory effects that reduce redness, swelling, and irritation in skin conditions.

### Potency Levels:

- **Class I:** Super potent (e.g., clobetasol propionate)
- **Class VII:** Least potent (e.g., hydrocortisone)

**Clinical Relevance:** Tailor potency based on location, severity, and duration to minimize side effects such as skin atrophy or systemic absorption.

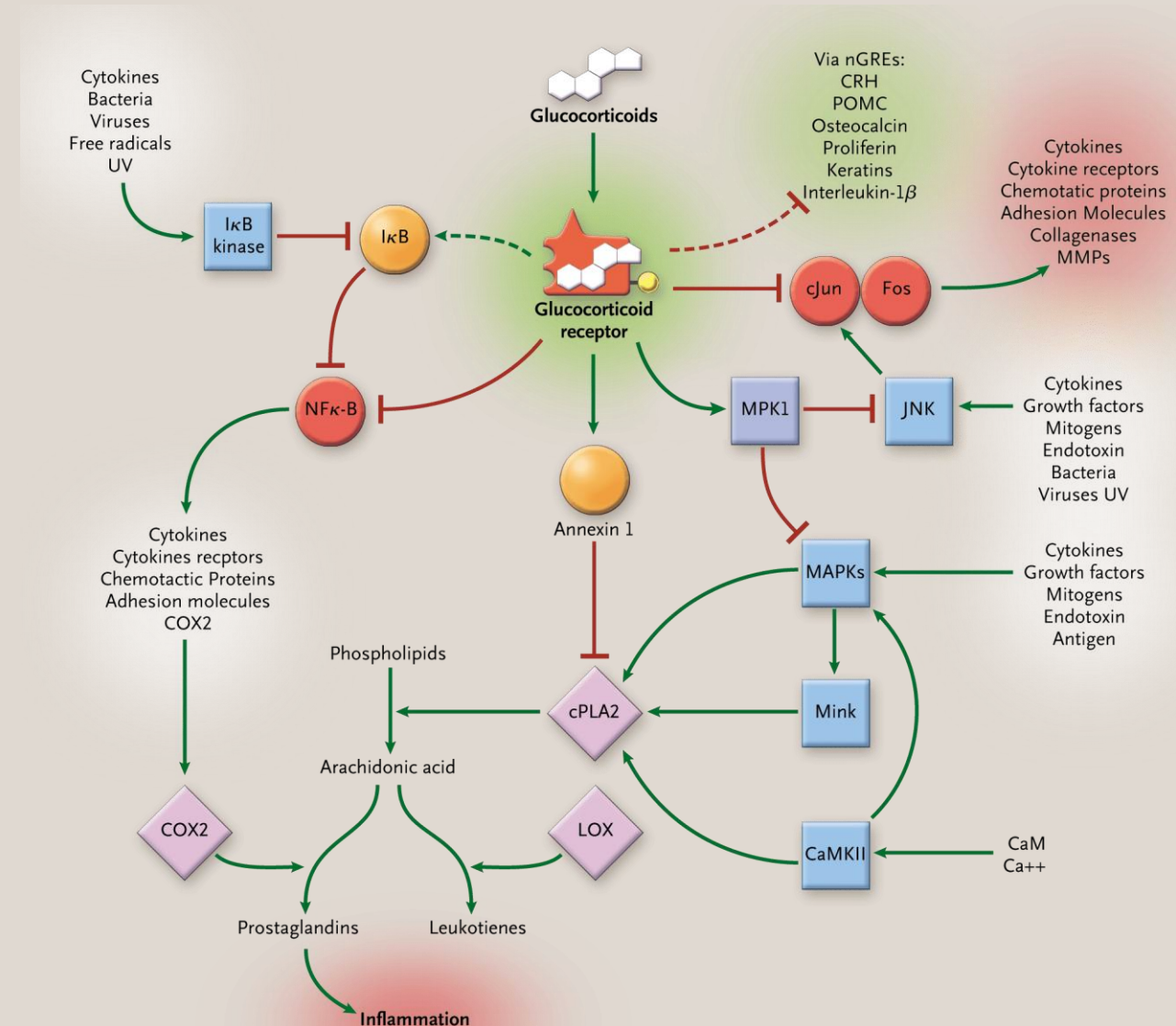
# How Corticosteroids Work

**Primary Action:** Corticosteroids bind to glucocorticoid receptors, entering the cell nucleus to modulate gene expression.

## Effect of Cytokines:

- Inhibits pro-inflammatory cytokines such as IL-1, IL-6, and TNF-alpha.
- Upregulates anti-inflammatory proteins like annexin-1, reducing inflammatory cell infiltration.

**Result:** Suppresses redness, swelling, itching, and other inflammatory responses in skin conditions.



# Indications for Corticosteroids: Managing Common Skin Conditions

## Eczema (Atopic and Contact Dermatitis):

- § Suppresses inflammation
- § Promotes skin barrier recovery
- § Used in mild to severe cases

## Psoriasis:

- § Decreases keratinocyte proliferation
- § Reduces plaque thickness and scaling
- § Adjunctive therapy for severe cases

## Contact Dermatitis:

- § Controls inflammation for both irritant and allergic types
- § Provides rapid relief of localized symptoms
- § High-potency corticosteroids may be required for thickened skin



# Understanding the Adverse Effects of Corticosteroids

**Skin Thinning (Atrophy):** Prolonged use, especially of high-potency corticosteroids, can weaken the skin, making it more susceptible to injury.

**Striae (Stretch Marks):** Reduced collagen synthesis can lead to the formation of stretch marks, particularly on areas like the thighs, abdomen, and axillae.

**Systemic Absorption:** In rare cases, prolonged use of potent corticosteroids or application over large surface areas can lead to systemic side effects, including adrenal suppression, hyperglycemia, and Cushing's syndrome, especially in children and individuals with thin skin.

## STORY TIME



# Safety Profile – Corticosteroids

## Understanding Risks and Dispelling Myths

### Short-Term Use:

- Effective for eczema, psoriasis, and dermatitis
- Minimal systemic absorption
- **Mild side effects:** skin thinning (rare with low-potency), burning, or irritation

### Long-Term Use:

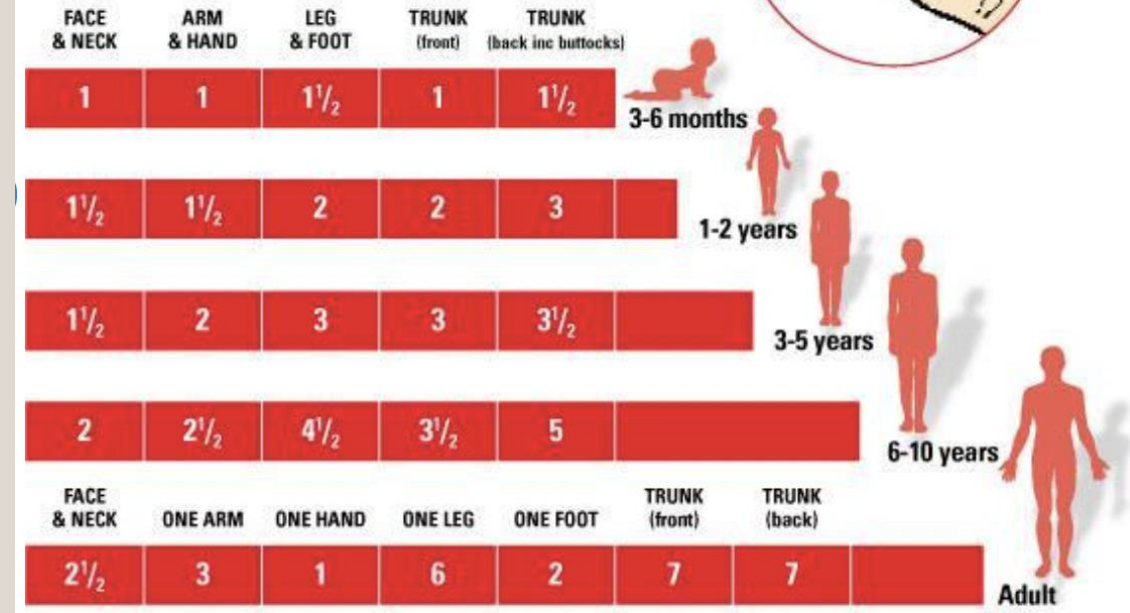
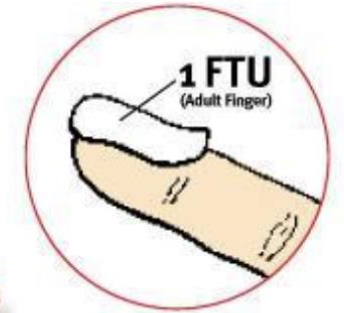
- **Risks:** Skin atrophy, stretch marks, telangiectasias, or rebound flares if overused
- **Mitigation:** Rotate low- to medium-potency steroids and follow treatment duration guidelines

### The fingertip unit method\*

FTU = Fingertip unit(adult)

1 FTU = 1/2 g of cream or ointment.

Measurement based on 5mm nozzle.



**Key Message:** Topical corticosteroids are safe when used as directed.

# Myths vs. Facts on Topical Steroid Withdrawal (TSW)

## Misinformation:

- Myth: TSW is common with all topical steroids
- Reality: TSW is **rare** and usually linked to long-term, high-potency misuse

## Statistics:

- TSW occurrence: <5% in appropriate use
- Key risk factors: High-potency steroid misuse over months/years

## Best Practices:

- Use steroids sparingly on the face or thin skin areas
- Educate patients on tapering and moisturization during breaks

**Takeaway:** Patient education and adherence are critical for safety.

# Antibiotics in Dermatology

## Treating Bacterial Skin Infections, Acne, and Rosacea

### Bacterial Skin Infections:

- **Impetigo:** Caused by *Staphylococcus aureus* or *Streptococcus pyogenes*
- **Folliculitis:** Infection of hair follicles, commonly caused by *S. aureus*

**Acne:** Targets *Cutibacterium acnes* and reduces inflammation

§ Common antibiotics: Doxycycline, Minocycline, Clindamycin (topical)

**Rosacea:** Reduces inflammatory lesions and erythema

- Common antibiotics: Metronidazole (topical), Doxycycline (oral)

### Commonly Used Antibiotics:

- **Topical:** Mupirocin, Clindamycin, Metronidazole, Minocycline
- **Oral:** Cephalexin, Dicloxacillin, Doxycycline, Minocycline, Trimethoprim/Sulfamethoxazole

### Key Points:

- Use topical antibiotics for localized skin infections or mild acne/rosacea
- Oral antibiotics are reserved for widespread or inflammatory conditions
- Limit long-term use to prevent antibiotic resistance



# Mechanism of Action – Antibiotics

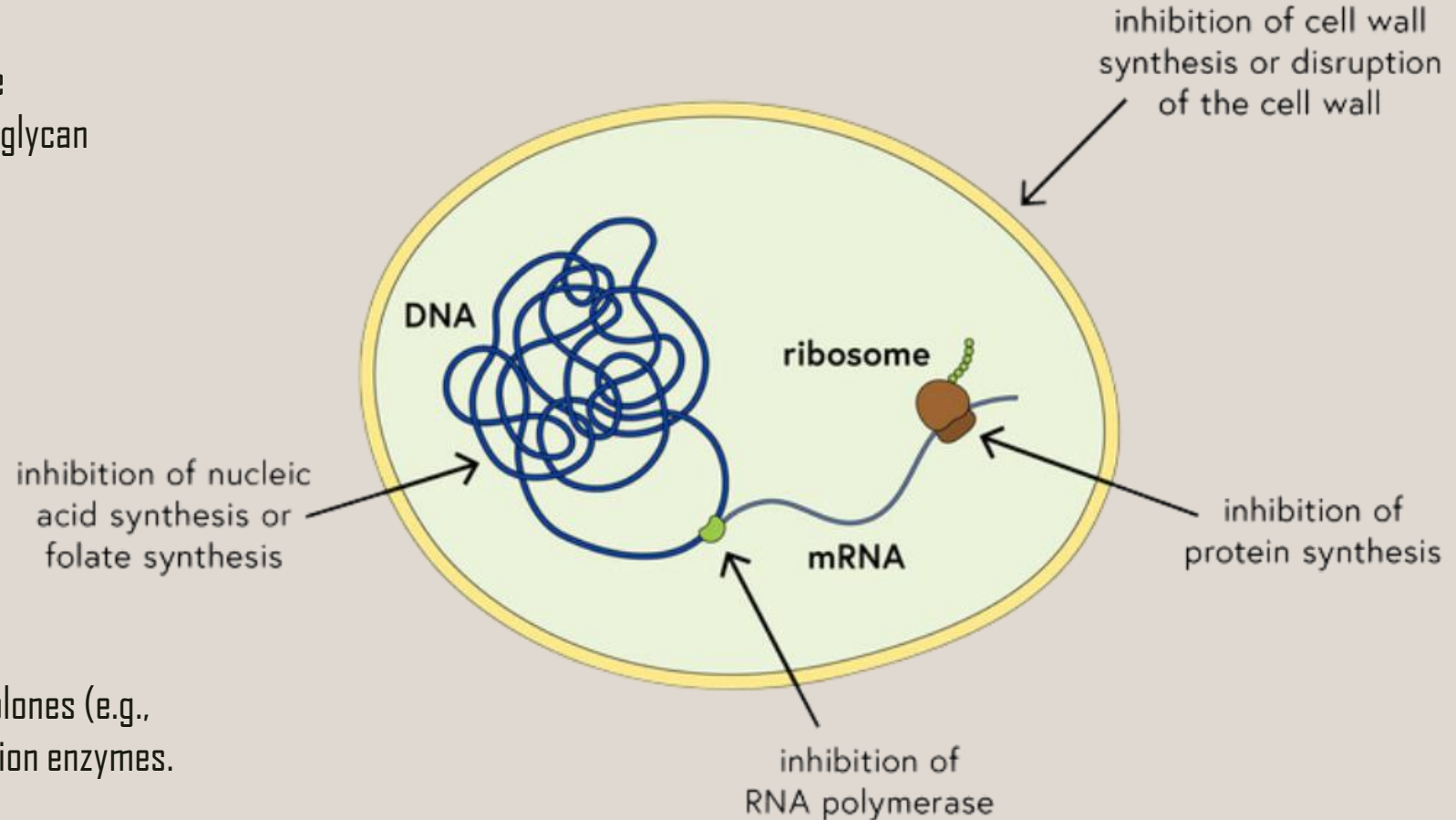
## How Antibiotics Inhibit Bacterial Growth

- **Inhibiting Cell Wall Synthesis:** Antibiotics like penicillins and cephalosporins block peptidoglycan formation, leading to bacterial lysis.

- § **Disrupting Protein Synthesis:** Macrolides (e.g., erythromycin), tetracyclines, and aminoglycosides bind to ribosomes, halting bacterial protein production.

- § **Interfering with DNA Synthesis:** Fluoroquinolones (e.g., ciprofloxacin) inhibit bacterial DNA replication enzymes.

- **Blocking Folic Acid Synthesis:** Sulfonamides prevent bacteria from synthesizing essential folic acid for growth.

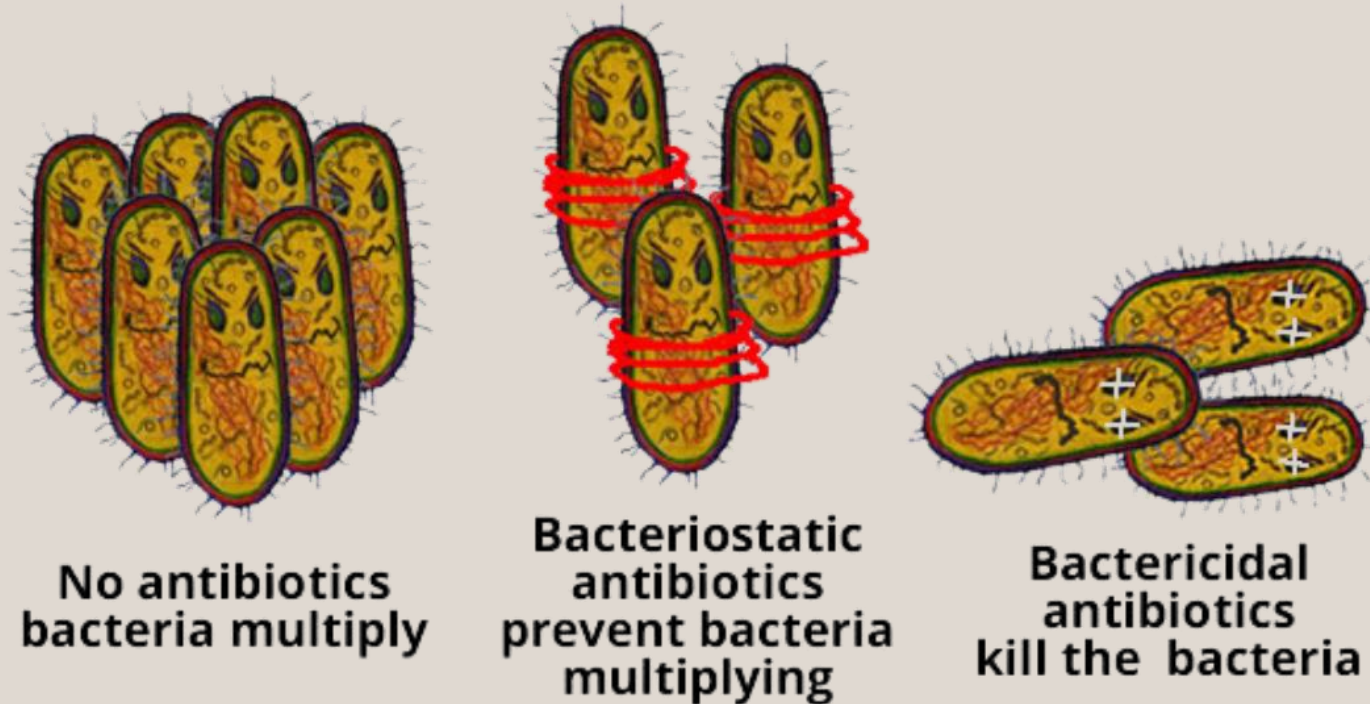


# Mechanism of Action – Antibiotics

## How Antibiotics Inhibit Bacterial Growth

### Bactericidal vs. Bacteriostatic:

- **Bactericidal:** Kills bacteria (e.g., penicillins, aminoglycosides)
- **Bacteriostatic:** Prevents bacterial growth (e.g., tetracyclines, macrolides)



**Key Point:** Antibiotics are selective for bacterial targets, minimizing harm to human cells.

# Indications for Antibiotics

## Common Uses in Dermatology

**Acne:** Target *Cutibacterium acnes* and inflammation

- **Topical:** Clindamycin, Erythromycin, Minocycline
- **Oral:** Doxycycline, Minocycline (subantimicrobial dosing reduces resistance risks)

**Rosacea:** Reduces inflammation and erythema

- **Topical:** Metronidazole, Azelaic Acid
- **Oral:** Doxycycline (low-dose, subantimicrobial for anti-inflammatory effect)

**Cellulitis:** Treats deeper bacterial infections (often *Streptococcus* or *Staphylococcus*)

- **Oral** Cephalexin, Dicloxacillin, or Clindamycin

**Impetigo:** Treats superficial infections caused by *S. aureus* or *S. pyogenes*

- **Topical** Mupirocin
- **Oral** Cephalexin or Amoxicillin-Clavulanate for widespread lesions

**Key Point:** Antibiotics must be tailored to the condition, severity, and resistance risks.

# Adverse Effects of Antibiotics

## Risks and Mitigation in Dermatologic Use

### Antibiotic Resistance:

- § Prolonged use increases resistance risk, especially with topical agents
- § **Clinical Pearl:** Use antibiotics in combination with benzoyl peroxide for acne to minimize resistance

### Gastrointestinal (GI) Upset:

- § Common with oral antibiotics like Doxycycline and Minocycline
- § **Mitigation:** Take with food (if allowed) and avoid irritating agents like NSAIDs concurrently
- § Usually not seen with subantimicrobial dosing

### Photosensitivity:

- Associated with tetracyclines (e.g., Doxycycline)
- **Clinical Pearl:** Counsel patients to use broad-spectrum sunscreen daily and avoid peak sun hours

### Clinical Pearls:

- Limit the duration of antibiotic use (e.g., 3–6 months for acne)
- Transition patients to maintenance therapies like retinoids or azelaic acid once improvement is noted

# Safety Profile – Antibiotics

## Risks to Consider with Antibiotic Use

### Antibiotic Resistance:

- § Prolonged or unnecessary use promotes resistant bacteria (e.g., MRSA)
- § **Clinical Pearl:** Avoid monotherapy with topical antibiotics; combine with benzoyl peroxide for acne to mitigate resistance risk

### *C. difficile* Infections:

- Antibiotic use can disrupt normal gut flora, allowing *Clostridioides difficile* to proliferate
- Common culprits: Broad-spectrum antibiotics (e.g., Clindamycin, Fluoroquinolones)
- **Clinical Pearl:** Emphasize probiotics or prebiotics during treatment to support gut health when appropriate



### Clinical Pearls:

- § Limit duration of therapy to essential periods (e.g., 3–6 months for acne)
- § Monitor for symptoms of *C. difficile* (e.g., persistent diarrhea) after antibiotic use, particularly in immunocompromised or elderly patients



# Immunomodulators

## Modulating Immune Responses

**Eczema (Atopic Dermatitis):** Suppresses inflammatory pathways to reduce itching and flare-ups

- **Common agents:** Tacrolimus (Protopic), Pimecrolimus (Elidel)

**Psoriasis:** Reduces hyperactive immune response contributing to plaque formation

- Topical and systemic options: Calcitriol cream (Dovonex), Apremilast (Otezla)

### Mechanism of Action:

- Modulate specific immune pathways, targeting T-cells or cytokines (e.g., IL-4, IL-13)
- Prevents excessive inflammation without widespread immunosuppression

### Advantages:

- **Non-steroidal:** Safe for long-term use in sensitive areas (e.g., face, eyelids)
- Fewer side effects compared to corticosteroids

### Clinical Pearls:

- **Eczema:** Use a thin layer; combine with moisturizers for enhanced skin barrier repair
- **Psoriasis:** Tailor treatment based on severity—topical for mild, systemic for moderate-to-severe

# Novel Immunomodulator: Vtama (Tapinarof)

## Mechanism:

- Aryl Hydrocarbon Receptor (AhR) Agonist: Activates the AhR pathway to downregulate inflammation and restore skin barrier homeostasis

## Approved Indications:

- Plaque Psoriasis: Approved for adults with mild-to-moderate disease
- Eczema (Atopic Dermatitis): Newly approved as a steroid-free option for managing inflammation and itching

## Immunomodulation:

- Modulates key inflammatory pathways like IL-17A, crucial in psoriasis and eczema pathogenesis



**Clinical Pearl:** Vtama can be used for long-term maintenance therapy without the risks of topical corticosteroids, especially in sensitive areas like the face or intertriginous zones.

# Novel Immunomodulator: Zoryve (Roflumilast)

## Mechanism:

- PDE-4 Inhibitor: Reduces inflammation by inhibiting phosphodiesterase-4 (PDE-4), leading to lower levels of pro-inflammatory cytokines (e.g., TNF- $\alpha$ , IL-13, IL-17)

## Approved Indications:

- Plaque Psoriasis: Suitable for mild-to-moderate cases
- Seborrheic Dermatitis: Effective in reducing scaling, erythema, and itching
- Eczema (Atopic Dermatitis): Provides steroid-free control of inflammation and itch in sensitive skin areas

## Immunomodulation:

- Targets localized inflammation while minimizing systemic immunosuppressive effects



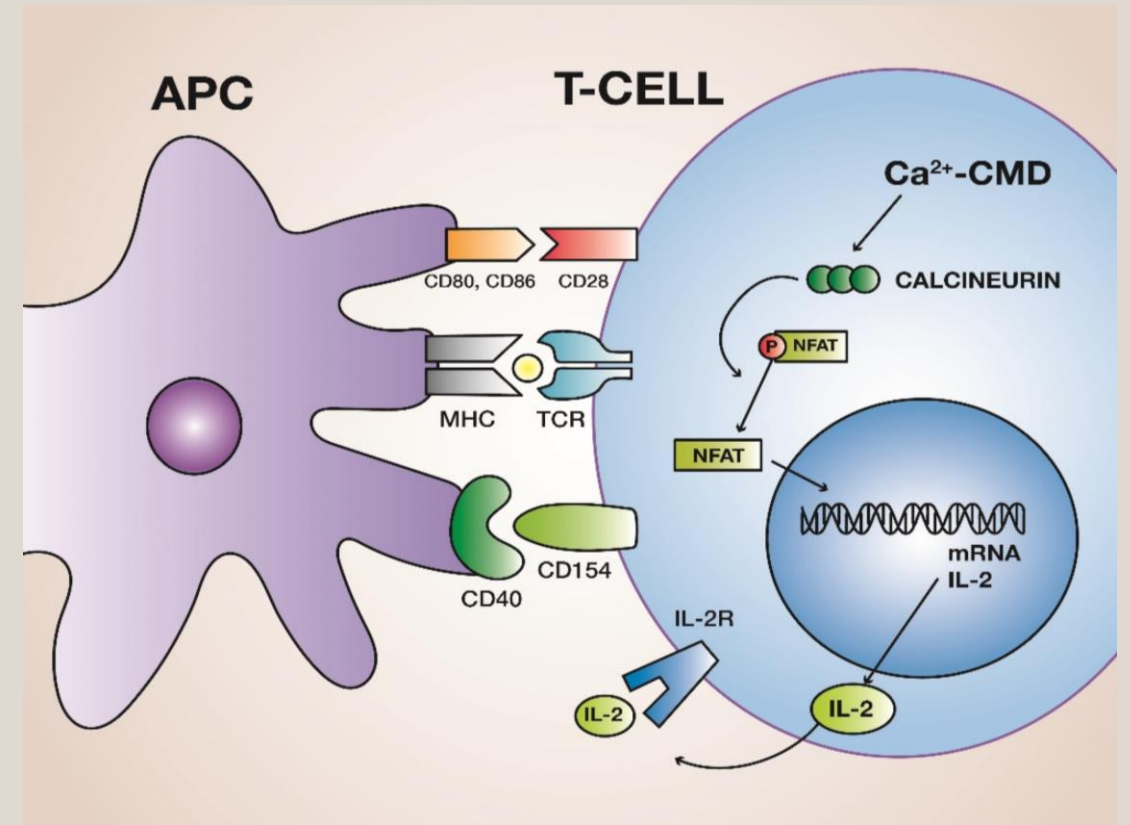
**Clinical Pearl:** Zoryve is particularly useful for areas prone to irritation, like the scalp, where seborrheic dermatitis and eczema often occur.

# Mechanism of Action: Immunomodulators

## Targeting Immune Pathways for Dermatologic Treatment

### Calcineurin Inhibitors:

- **Mechanism:** Inhibits calcineurin, preventing activation of T-cells and reducing inflammatory cytokine production (e.g., IL-2)
- **Common Agents:** Tacrolimus (Protopic), Pimecrolimus (Elidel)
- **Applications:** Effective for eczema, especially in sensitive areas (e.g., eyelids, face)



**Clinical Pearl:** Ideal for long-term use due to lack of skin atrophy seen with corticosteroids.

# Mechanism of Action: Immunomodulators

## Targeting Immune Pathways for Dermatologic Treatment

### JAK Inhibitors:

- **Mechanism:** Blocks Janus Kinase (JAK) pathways, disrupting signaling of pro-inflammatory cytokines (e.g., IL-4, IL-13, IL-31)
- **Common Agents:** Tofacitinib (systemic), Ruxolitinib (topical)
- **Applications:** Used in moderate-to-severe eczema and other inflammatory dermatoses

### Advantages of Immunomodulators:

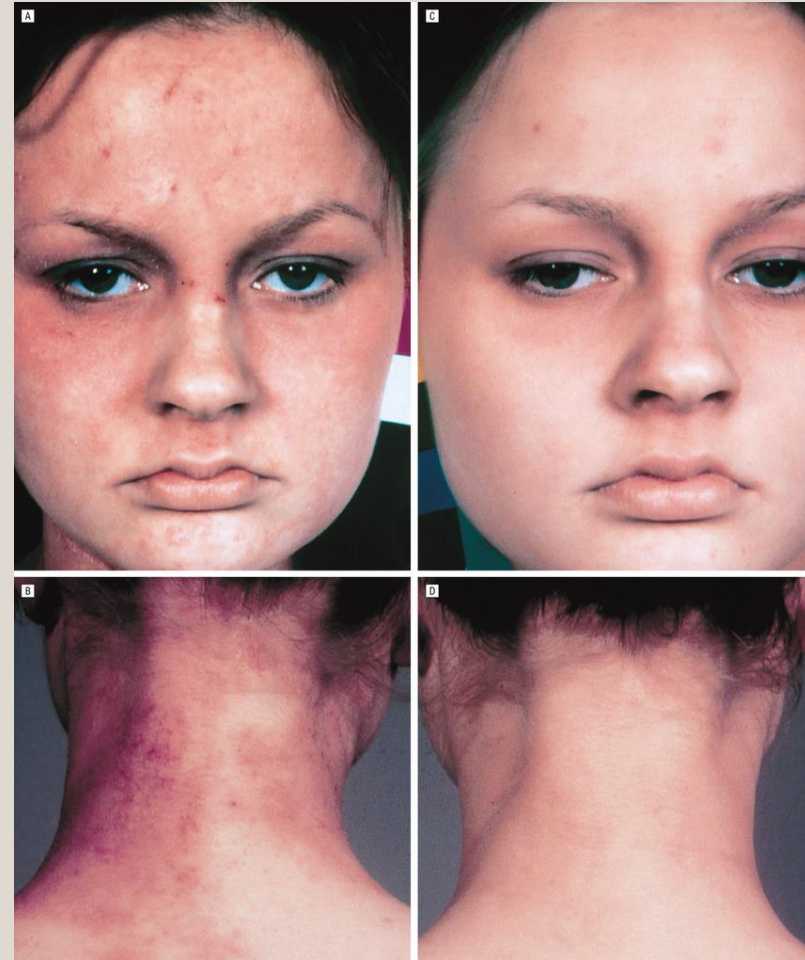
- § Targeted action minimizes widespread immune suppression
- § Safer for long-term use compared to systemic corticosteroids

**Clinical Pearl:** Monitor for systemic side effects with oral JAK inhibitors, such as increased infection risk.

# Indications for Immunomodulators

## Atopic Dermatitis:

- Modulates inflammatory pathways to reduce flare-ups and itching
- **Agents:** Tacrolimus, Pimecrolimus (topical), and Dupilumab (systemic IL-4/ IL-13 inhibitor)



**Clinical Pearl:** Ideal for facial or sensitive areas where corticosteroids may cause atrophy.

# Indications for Immunomodulators

## Vitiligo:

- Promotes repigmentation by targeting autoimmune destruction of melanocytes
- **Agents:** Tacrolimus (topical), Ruxolitinib (JAK inhibitor)

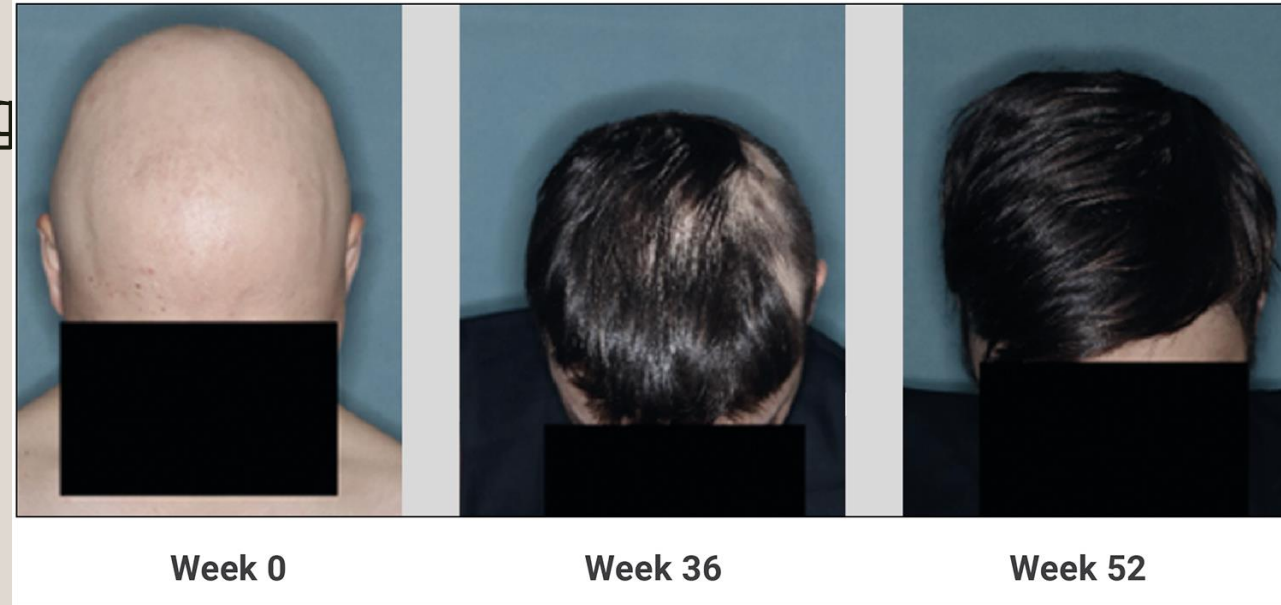


**Clinical Pearl:** Works best in combination with phototherapy for enhanced repigmentation.

# Indications for Immunomodulators

## Alopecia Areata:

- Targets immune response leading to hair follicle destruction
- **Agents:** Baricitinib, Tofacitinib (oral JAK inhibitors)
- **Clinical Pearl:** Monitor for systemic side effects



## Overall Advantages:

- Non-steroidal therapies with fewer side effects in long-term use
- Particularly effective in sensitive or resistant areas



# Common Adverse Effects of Immunomodulators

## Burning Sensation:

- Most frequent with topical calcineurin inhibitors like Tacrolimus and Pimecrolimus
- Typically, transient and resolves with continued use
- **Clinical Pearl:** Recommend application on damp skin or after a moisturizer to reduce discomfort

## Lipid Profile Changes:

- Seen with systemic JAK inhibitors (e.g., Tofacitinib, Baricitinib)
- Can elevate cholesterol and triglycerides over time
- **Clinical Pearl:** Monitor lipid panels regularly during systemic treatment and provide dietary counseling

# Common Adverse Effects of Immunomodulators

## Herpes Reactivation:

- Immunomodulation can trigger reactivation of latent herpes simplex virus
- More common with systemic agents like JAK inhibitors
- **Clinical Pearl:** Prophylactic antivirals may be considered for high-risk patients

## Other Considerations:

- Risk-benefit assessment is crucial in chronic conditions
- Educate patients on recognizing symptoms early (e.g., persistent burning or viral reactivation signs)

# Long-Term Safety Considerations

## Topical Calcineurin Inhibitors (e.g., Tacrolimus, Pimecrolimus):

- Safe for long-term use, even on sensitive areas (face, eyelids)
- Minimal risk of skin atrophy
- Real-world data supports safety and lack of increased cancer risk

## JAK Inhibitors (e.g., Tofacitinib, Baricitinib):

- Monitor lipids (cholesterol/triglycerides)
- Screen for latent infections (e.g., TB)
- Educate patients about potential for serious infections.

## Advantages of Non-Steroidal Options:

- Better long-term tolerability
- Reduced cumulative side effects compared to corticosteroids

# Biologics: Targeting Specific Pathways in Immune Response

**What are they?** Advanced therapies from living organisms, targeting specific immune system components.

**How are they different?** Traditional drugs broadly suppress the immune system, causing more side effects. Biologics offer precision medicine.

## **Key Benefits:**

- Fewer systemic side effects
- High efficacy for severe/refractory conditions
- Long-term safety

**Clinical Pearl:** Educate patients on what biologics are and how they differ from traditional treatments. Highlight that they are not "general immune suppressants" but precision tools.

# Mechanism of Action – Biologics

## Key Targeting of Inflammatory Pathways

**IL-17:** Drives inflammation in psoriasis by recruiting inflammatory cells

§ Blocked by: Secukinumab, Ixekizumab

**IL-23:** Regulates the differentiation of inflammatory T-cells

§ Blocked by: Guselkumab, Risankizumab

**IL-4 and IL-13:** Central to atopic dermatitis and asthma

§ Blocked by: Dupilumab

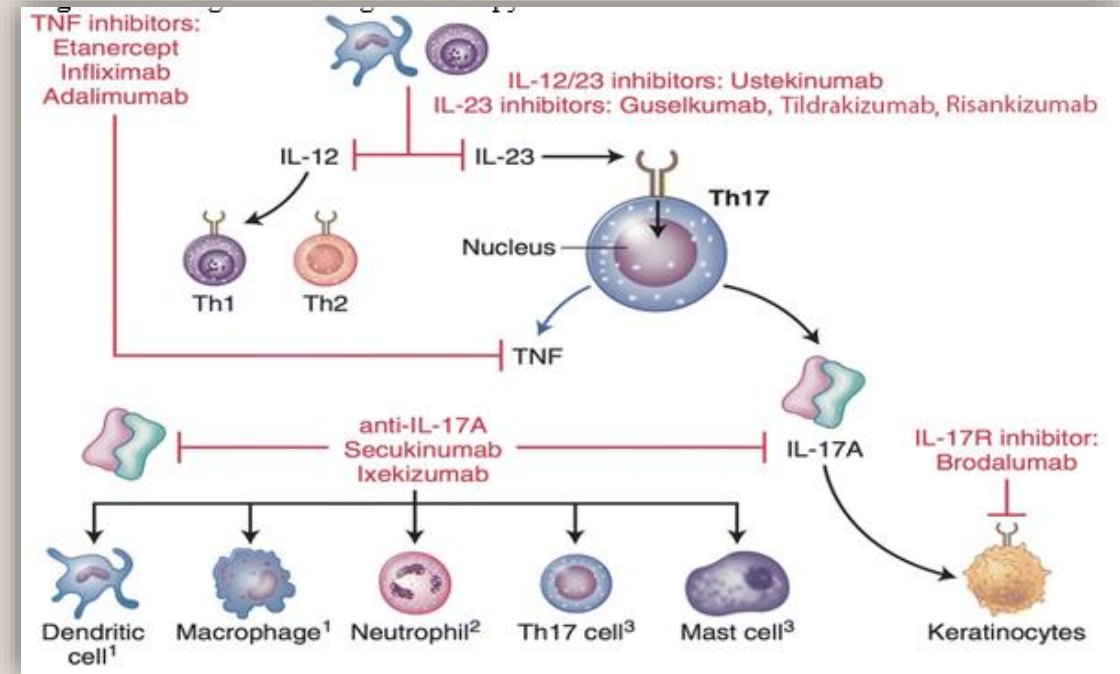
**TNF- $\alpha$ :** Promotes systemic inflammation in autoimmune diseases

■ Blocked by: Adalimumab, Etanercept

# Mechanism of Action – Biologics

## Cytokine Cascade Disruption:

- Biologics interrupt the cytokine cascade to prevent immune system overactivation.
- Block specific pathways:
  - **IL-17 and IL-23:** Psoriasis, Ankylosing Spondylitis
  - **IL-4 and IL-13:** Atopic Dermatitis, Asthma
  - **TNF- $\alpha$ :** Rheumatoid Arthritis, Psoriasis, Hidradenitis Suppurativa



**Clinical Pearl:** Biologics are most effective when inflammation is driven by specific cytokines, enabling precise control of symptoms with minimal systemic effects.

# Indications for Biologics

## Transforming Treatment in Chronic Conditions

### Psoriasis:

- Targets pathways driving plaque formation
- Improves skin & quality of life
- Early use may prevent psoriatic arthritis

### Hidradenitis Suppurativa:

- Reduces inflammation (nodules/abscesses)
- Offers alternatives for TNF- $\alpha$  non-responders



### Prurigo Nodularis:

- § Reduces itch and lesions (best with emollients/anti-itch).

### Eczema:

- § Reduces flares, itching, improves skin barrier
- § Options for children and adults

### Key Message:

Biologics provide targeted solutions for severe dermatologic conditions, improving physical symptoms, quality of life, and long-term outcomes.

# Key Adverse Effects of Biologics

## Understanding Potential Risks

### Increased Risk of Infection:

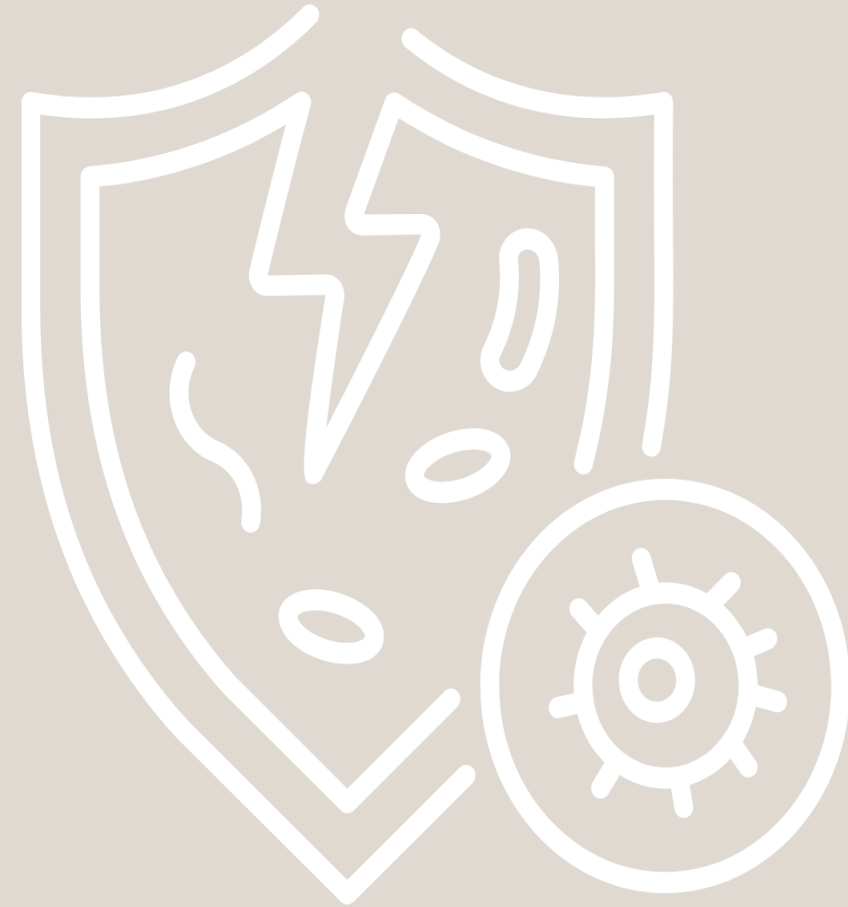
- § **Common:** URI, sinusitis
- § **Serious:** Tuberculosis (TB), herpes zoster, fungal infections
- § **Clinical Pearl:** Screening for latent TB and hepatitis B reduces risks

### Risk of Malignancy:

- § Rare but reported in real-world data
- § Evidence suggests biologics targeting IL-17 and IL-23 pathways have lower malignancy risk

### Injection Site Reactions:

- Localized redness, swelling, and pain are most common





# Real-World Data on Adverse Effects

## Infection Rates:

- Clinical trials report <5% rate of serious infections
- Real-world data confirm that screening and patient selection minimize risks

## Malignancy Risk:

- TNF- $\alpha$  inhibitors: Slightly elevated risk for lymphoma in some populations
- IL-17/IL-23 inhibitors: No significant increase in malignancy in 5-year follow-ups

**Clinical Pearl:** Regular monitoring and patient education mitigate most risks.

# Patient Monitoring Strategies

## Minimizing Risks with Biologics

### Why Monitor?

- Biologics modify the immune response, which can increase the risk of reactivating latent infections
- Pre-treatment testing is essential to minimize these risks and ensure patient safety

### Tuberculosis (TB):

- Perform a TB skin test (TST) or interferon-gamma release assay (IGRA) before initiating biologics
- Patients with a history of TB or exposure should be treated for latent TB infection before starting biologics
- Conduct regular symptom checks (e.g., persistent cough, fever) during treatment

### Hepatitis B and C:

- Test for hepatitis B surface antigen (HBsAg), core antibodies (anti-HBc), and hepatitis C antibody (anti-HCV) before starting therapy
- Be aware of the risk of reactivation, particularly with TNF- $\alpha$  inhibitors
- Consult a hepatologist if reactivation occurs; antiviral prophylaxis may be indicated

**Clinical Pearl:** Always document baseline results and educate patients on symptoms of infection to report immediately.

# Understanding JAK Inhibitors

## Breakthrough in Treating Inflammatory Dermatologic Conditions

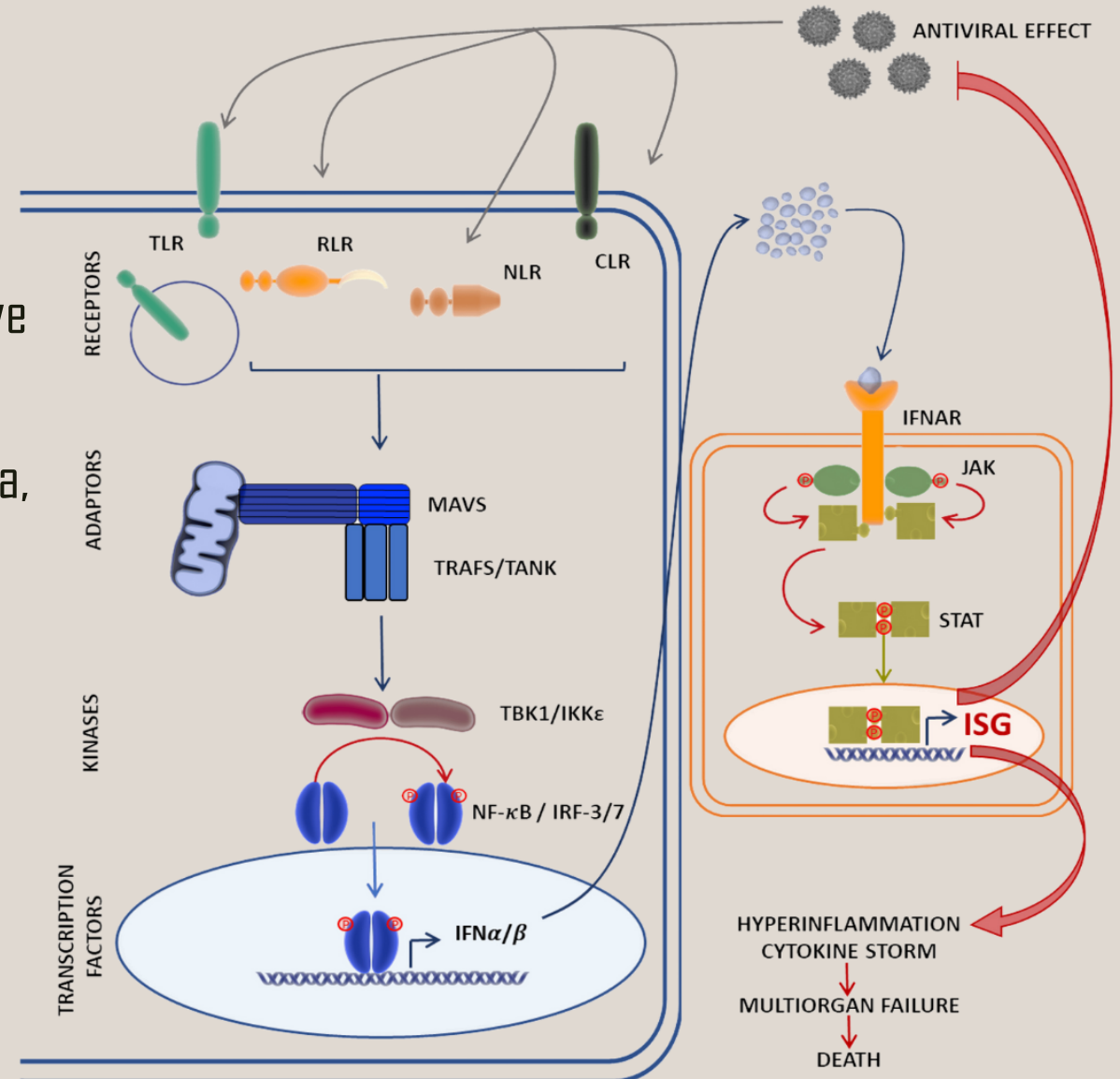
**What are they?** Small molecules that block the JAK-STAT pathway, a key player in immune activation and inflammation

**Why a breakthrough?** Targeted therapy for overactive immune responses

**Conditions treated:** Atopic dermatitis, Alopecia areata, Vitiligo

**Advantages:**

- Rapid symptom improvement (especially in severe cases)
- Less systemic immunosuppression than biologics



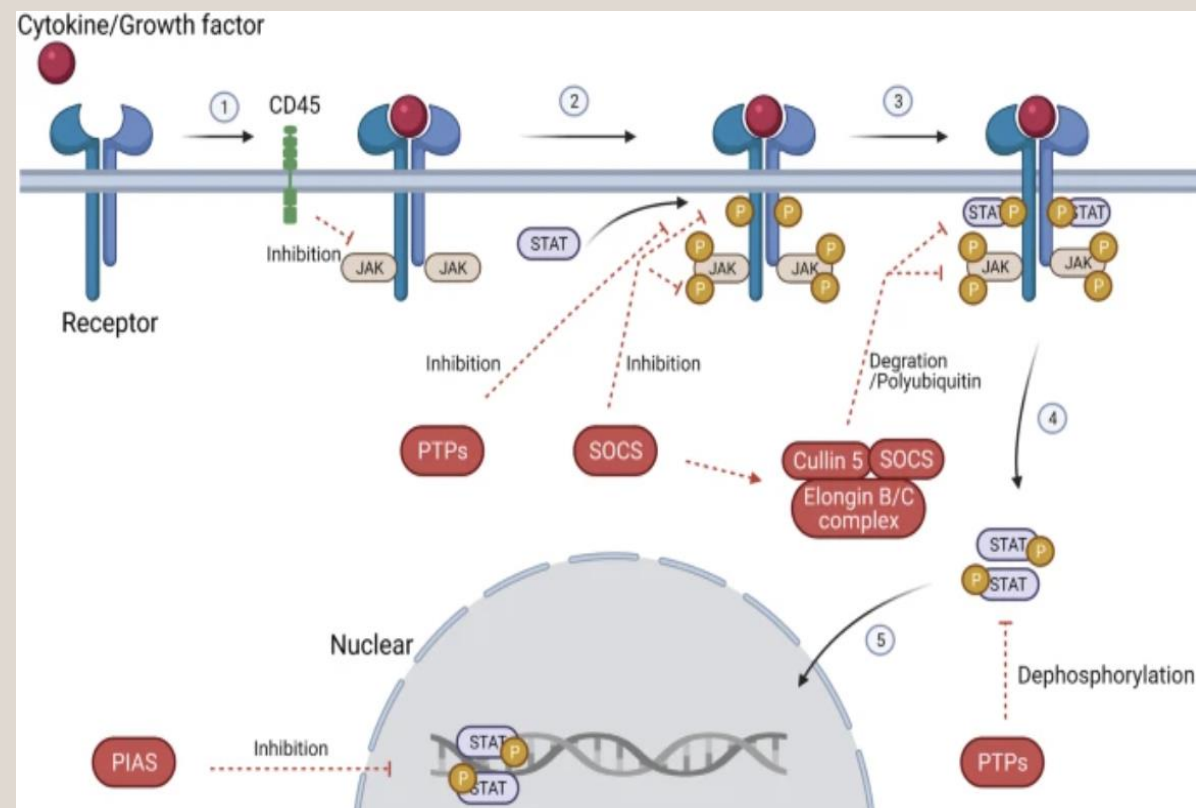
# JAK-STAT Pathway: Simplified

**Janus Kinase (JAK):** A family of enzymes (JAK1, JAK2, JAK3, TYK2) that mediate signaling for cytokines involved in inflammation and immunity

**Signal Transducer and Activator of Transcription (STAT):** Proteins activated by JAKs to promote gene expression related to inflammation

## Mechanism of JAK Inhibitors:

- **Target:** JAK inhibitors block the activation of JAK enzymes, preventing the phosphorylation of STAT proteins
- **Effect:** Reduces inflammatory cytokine signaling, including IL-4, IL-6, IL-13, and IFN- $\gamma$ , which are critical in autoimmune and inflammatory diseases

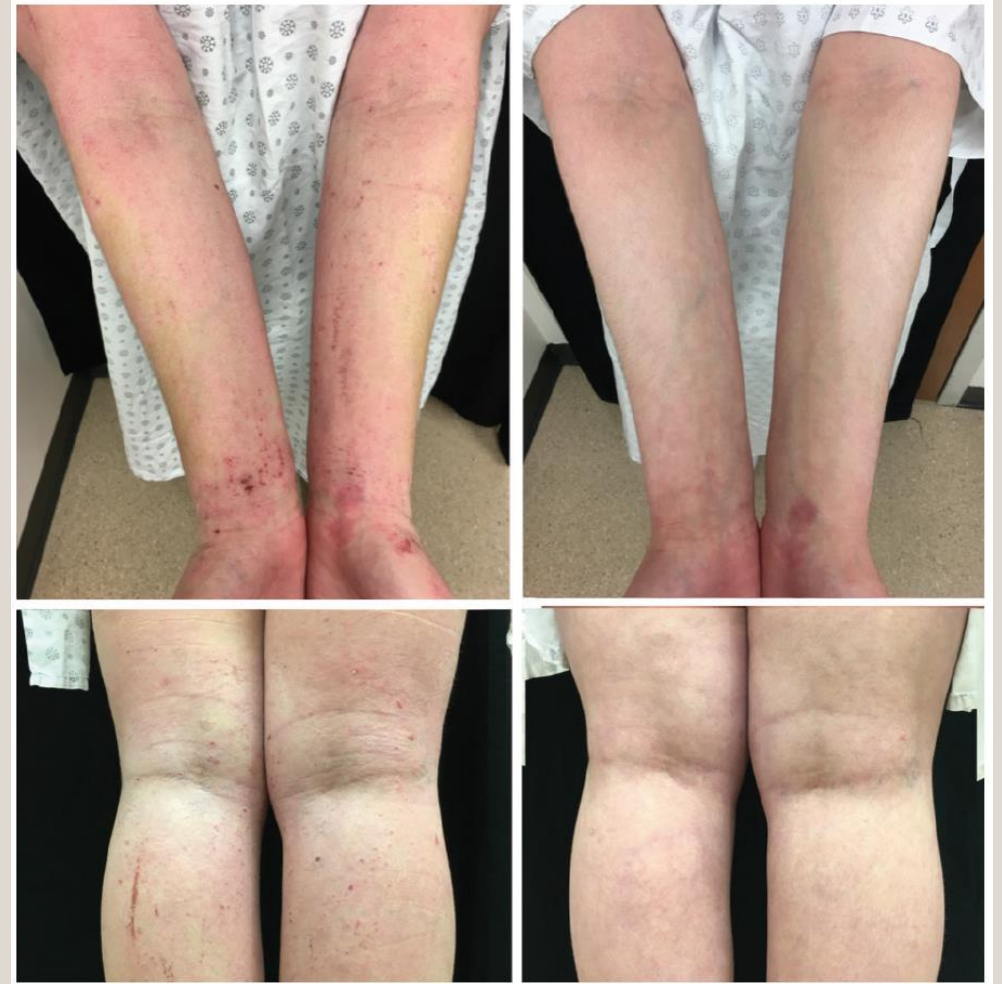


**Clinical Pearl:** JAK inhibitors are particularly effective for patients with severe disease or those who fail traditional therapies. Monitor for side effects like lipid abnormalities and infections.

# Indications for JAK Inhibitors

## Atopic Dermatitis:

- Effectively reduces itching, redness, and inflammation by targeting cytokines like IL-4 and IL-13.
- **Example Agents:** *Baricitinib, Upadacitinib, Abrocitinib.*



**Clinical Pearl:** Ideal for moderate-to-severe cases unresponsive to topical treatments or biologics.

# Indications for JAK Inhibitors

## Vitiligo:

- Promotes repigmentation by blocking cytokines (e.g., IFN- $\gamma$ ) that impair melanocyte regeneration
- *Example Agent: Ruxolitinib (topical)*



**Clinical Pearl:** Best results are observed with early-stage vitiligo or in combination with phototherapy.



# Indications for JAK Inhibitors

## Alopecia Areata:

- Stimulates hair regrowth by halting the immune attack on hair follicles
- **Example Agents:**  
Tofacitinib, Baricitinib



**Clinical Pearl:** Long-term results depend on consistent use and early intervention.

# Adverse Effects of JAK Inhibitors

## Mild Side Effects:

- Nausea
- Headache
- Infections (e.g., Upper Respiratory Tract Infections [URTI])

***COMMON BUT  
MANAGEABLE***



## Severe Side Effects:

- Risk of Blood Clots (e.g., deep vein thrombosis, pulmonary embolism)
- Malignancies (e.g., lymphoma, skin cancers in long-term use)
- Serious Infections (e.g., tuberculosis, opportunistic infections)

***RARE BUT  
SIGNIFICANT***



## Clinical Pearl:

- § Patients with high cardiovascular or malignancy risk may need alternative therapies.
- § Regular monitoring and pre-treatment screening (e.g., TB test) are critical.



# Ensuring Safety: Monitoring and Screening for JAK Inhibitors

## Pre-Treatment Screening:

- **Tuberculosis (TB):** Perform TB skin test (TST) or interferon-gamma release assay (IGRA). Treat latent TB before initiating JAK inhibitors
- **Hepatitis Screening:** Check for hepatitis B and C to prevent viral reactivation

## Infection Monitoring:

- High risk of infections, including: Herpes zoster & opportunistic infections
- Educate on recognizing early signs of infection (e.g., fever, persistent cough)

## Routine Lab Tests:

- **Lipid Profile:** Monitor for increases in cholesterol and triglycerides
- **Complete Blood Count (CBC):** Assess for anemia, neutropenia, or lymphopenia
- **Liver Function Tests (LFTs):** Monitor for hepatotoxicity

# How to Choose the Best Treatment

## Safety:

- § Screen for comorbidities (e.g., cardiovascular disease, TB, hepatitis)
- § Consider long-term risks (e.g., infections, malignancy, blood clots)

## Efficacy:

- § Evaluate the medication's success in clinical trials and real-world settings
- § *Match drug mechanisms (e.g., JAK inhibition, IL-targeting) to the disease pathway*

## Patient-Specific Considerations:

- Disease severity (mild, moderate, severe)
- Preference for oral vs. topical treatments
- Lifestyle factors (e.g., ability to adhere to monitoring or dosing schedules)

**Clinical Pearl:** Always consider a stepwise approach - start with the least invasive, escalating to systemic therapies for refractory cases.

# Weighing Drug Classes: JAK Inhibitors and Beyond

## JAK Inhibitors (e.g., Baricitinib, Ruxolitinib):

- **Efficacy:** Rapid symptom relief, particularly in moderate-to-severe conditions
- **Safety:** Monitor for infections, lipid changes, and thrombosis risk
- **Indications:** Alopecia areata, vitiligo, atopic dermatitis

## Biologics (e.g., Dupilumab, Secukinumab):

- **Efficacy:** Highly targeted therapy for specific cytokines (e.g., IL-4, IL-17)
- **Safety:** Lower infection risk compared to JAK inhibitors but requires monitoring for injection site reactions
- **Indications:** Psoriasis, eczema, hidradenitis suppurativa

## Topical PDE-4 Inhibitors (e.g., Zoryve):

- **Efficacy:** Best for mild-to-moderate localized disease
- **Safety:** Minimal systemic side effects
- **Indications:** Psoriasis, seborrheic dermatitis, eczema

# A Stepwise Approach to Treatment Selection

## Step 1: Assess Disease Severity

- Mild → Topicals (e.g., PDE-4 inhibitors or AhR Agonists)
- Moderate → Biologics or systemic JAK inhibitors
- Severe → Combine systemic therapy with adjunctive topicals

## Step 2: Screen for Safety

- Pre-treatment screening for TB, hepatitis, and cardiovascular risks
- Monitor for lipid and liver abnormalities during treatment

## Step 3: Patient Factors

- Prioritize patient convenience (e.g., frequency of dosing, route of administration)
- Consider insurance coverage and affordability

## Step 4: Monitor and Reassess

- Adjust therapy based on response and side effects
- Switch or escalate treatment if no improvement in 3–6 months

# Empowering Patients Through Education

## Effective Communication on Medication Use

### Understanding the Medication:

- § Explain the purpose: "This medication targets inflammation to reduce your symptoms"
- § Demonstrate how to use topical or oral medications correctly"

### Discussing Side Effects:

- Be transparent: "Mild nausea or headache might occur but should improve with time."
- Address severe risks: "Rarely, this can increase infection risk—notify us if you develop a fever."

### Providing Practical Advice:

- Set realistic expectations for improvement: "You may notice results in 2-4 weeks."
- Offer lifestyle tips to support treatment: "Using moisturizers regularly can enhance results."

**Clinical Pearl:** Use teach-back techniques - ask patients to repeat instructions to ensure understanding.

# Tools to Enhance Patient Understanding

## Resources for Better Patient Engagement

**Medication Guides:** Provide easy-to-read handouts explaining medication use and safety tips.

**Digital Tools:** Offer access to reputable apps or websites for medication tracking (e.g., MyMedications). Refer patients to pharmaceutical company patient education resources.

**Visual Aids:** Use charts or diagrams to explain treatment pathways (e.g., how inflammation is reduced).

### **In-Person Strategies:**

- Keep explanations concise and relatable: "Think of this medication as blocking the 'alarm' causing your symptoms."
- Schedule follow-up visits to review progress and address concerns

**Clinical Pearl:** Personalize education materials based on patient preferences (e.g., visual learners may benefit from infographics).

# Empowering Clinicians: Safety and Efficacy of Dermatology Medications

## 1. The Reality of Safety Concerns

- Address common patient fears:
  - **Corticosteroids:** Skin thinning and systemic absorption
  - **Biologics:** Infection risks, malignancies
  - **JAK Inhibitors:** Thrombosis and cardiovascular concerns
- Highlight the actual risk is low when appropriately prescribed.

## 2. Efficacy Backed by Data

- **Corticosteroids:** Rapid control of inflammation; effective for flares.
- **Biologics:** Significant improvement in moderate-to-severe psoriasis and eczema (>70% PASI or EASI reduction in clinical trials).
- **JAK Inhibitors:** Fast onset of action for refractory atopic dermatitis and alopecia areata.

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## 3. Strategies to Reassure Patients

- **Share** comparative risk data with other therapies.
- **Educate** on the importance of adherence for long-term benefits.
- **Emphasize** routine monitoring to minimize risks.

## 4. Key Takeaway

- Primary care clinicians are **pivotal in bridging the gap** between patient fears and evidence-based decision-making.
- **Empower** your patients with facts, not fears.