



## **Rosacea and Acneiform eruptions**

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- I have no financial conflicts of interest
- I have no financial affiliation with the companies that manufacture medications or products presented in this presentation



Kollias, Helen. "Research Review: Research, big food, and science." Precision Nutrition, n.p. n.d. 27 Aug 2014

# Objectives

- Objectives:
- Review the spectrum of acneiform eruptions
- Identify the major forms of rosacea
- Become familiar with common treatments for rosacea

# Acneiform eruptions

- Skin eruptions that resemble acne vulgaris.
- Most commonly:

**Acne vulgaris / Rosacea / Seborrheic Dermatitis**

**Drug reactions**

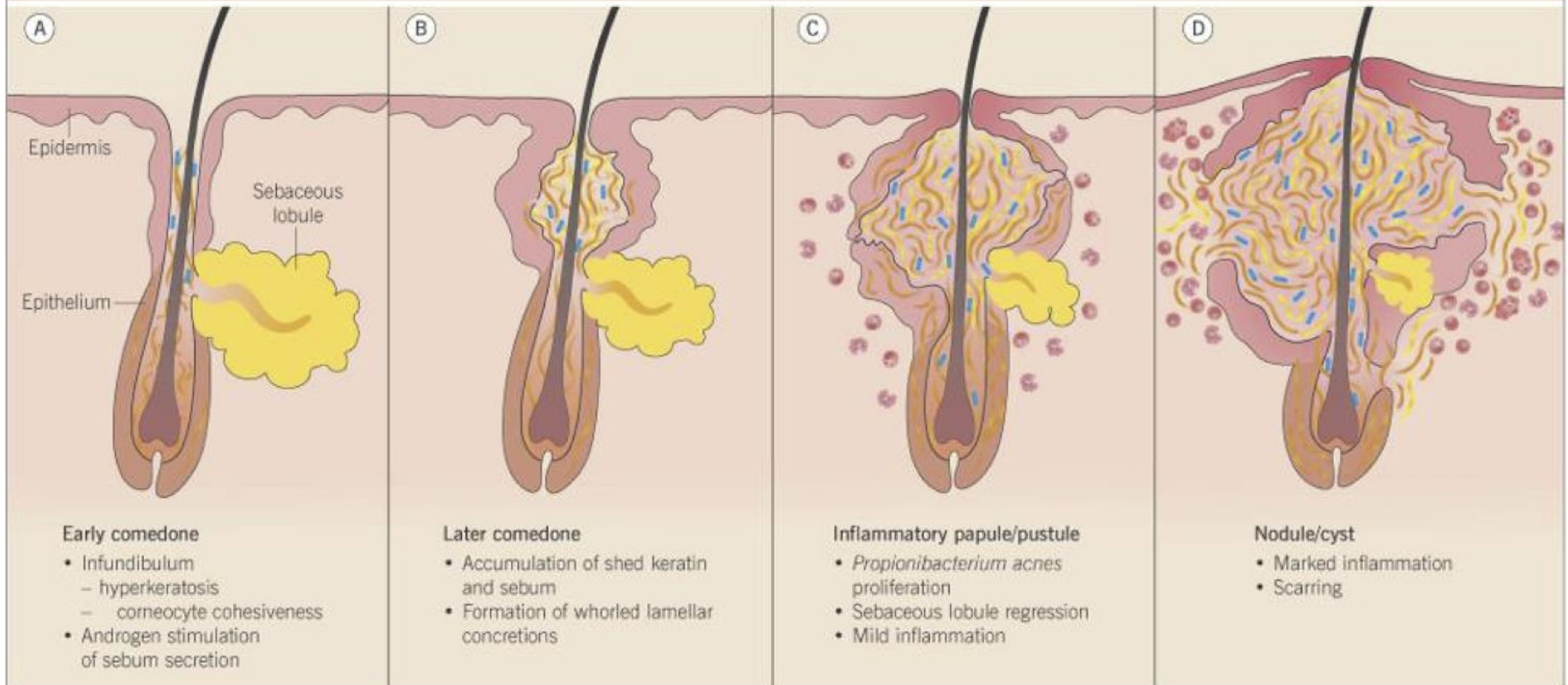
**Infections**

**Genetic or other metabolic disorders**

# Acne vulgaris

- Acne vulgaris
- Characterized by:
  - Comedones (black heads)
  - Inflammatory papules (often in various stages of healing)
  - Pustules

## PATHOGENESIS OF ACNE



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# Rosacea

- Rosacea
- Characterized by:
  - History of facial flushing
  - Inflammatory papules (often monomorphic)
  - Fibrous changes of the nose
  - Perioral or periorificial involvement

# Seborrheic dermatitis

- Seborrheic dermatitis
- Characterized by:
  - Patchy erythema, often with thicker greasy scale
  - Occasional erythematous papules
  - Distributed in hair bearing skin of head, central face, and upper torso



# Acneiform drug reactions

- Drug reactions
- Characterized by:
  - Sudden onset
  - Monomorphic lesions
  - Improvement/resolution after discontinuation of inciting drug
  - Corroborating history

# Acneiform drug reactions

- Acute generalized exanthematous pustulosis
- Steroids
- Kinase inhibitors
- Halogenated hydrocarbons

# Acute Generalized Exanthematous Pustulosis

- Often pustular eruption soon after initiation of drug
- Typically in the folds of the torso
- Accompanied by fever and neutrophilia
- Sometimes there is accompanying hepatitis

# Steroid acne

- Initially steroid acne is noncomedonal.
- High dose steroids over a few weeks
- Can be complicated by exacerbation of acne vulgaris

# Kinase inhibitors

- Imatinib
- Epidermal Growth Factor Receptor Inhibitors
  - Cetuximab
  - Erlotinib
  - Gefitinib
- Small molecular weight kinase inhibitorw
  - Sorafenib and vemurafinib

# Halogenated hydrocarbons

- Acne-like eruption after exposure to halogenated drugs or compounds
- Iodinated contrast dyes, I<sup>131</sup> treatment, potassium iodide treatment
- Fluoride dental gels
- Hydrocarbons alone also cause acneiform eruptions
  - Cutting oils
  - Crude petroleum

# Infectious acneiform eruptions

- Pityrosporum folliculitis
- Demodex mites
- Gram-negative folliculitis
- HIV/AIDS (eosinophilic folliculitis)
- Secondary syphilis
- Deep fungal infection (Majocchi's granuloma)

- Rosacea





- Rosacea
- Chronic inflammatory condition of the face of as yet unknown origin
- Characteristics:
- Transient, later permanent, facial flushing
- Thick and irregular skin of the face and nose may occur
- Sometimes plaque-like, edematous changes may occur



# Variants of Rosacea

- Erythematotelangiectatic
  - Facial flushing with/without telangiectatic mats
- Papulopustular
  - Erythematous papules, generally central face
  - Can become chronic plaques of edema
- Phymatous
  - Thickened and irregular nodularity of the skin
- Ocular
  - Dry eyes, foreign body sensation, lid/periocular edema

- Pathophysiology
  - Possibly genetic? Celtic and Northern European predominance
  - Dysregulation of the innate immune system in skin
    - Increased cathelicidin expression
    - Increased Toll-like receptor 2 expression
    - Matrix metalloproteinase expression increased
    - Increased kallikrein 5 expression
  - Local colonization may trigger the inflammatory pathways.
    - *Demodex* mites
    - *Staphylococcus epidermidis*
    - *Helicobacter pylori* ??

- Pathophysiology (cont'd)
  - UV radiation
    - Sun exposure triggers similar metalloproteinase activity and fibrosis
  - Neurogenic
    - Transient receptor potential (TRP) channels are more highly expressed.
  - Altered Skin Barrier Function?
    - Increased transepidermal water loss in rosacea

- Treatments
- Patient education
  - Identify potential modifiable triggers of rosacea
- Skin care
  - Moisturization and barrier protection of skin
  - Sunscreen use to block UV effects

- Treatments (cont'd)
- Topicals
  - Topical metronidazole 0.75 – 1% (lotion, cream, gel)
  - Topical sodium sulfacetamide
  - Azelaic acid
  - Permethrin
  - Ivermectin
  - Benzoyl peroxide
  - $\alpha$ -adrenergic agonists
  - Calcineurin inhibitors

- Systemic treatments
- Tetracycline-class antibiotics
  - Anti-inflammatory effects
- Beta-blockers
  - Constriction of dermal blood vessels
- Isotretinoin
  - Decreased TLR2 expression?

# Summary

- Acneiform eruptions
  - Include more than acne vulgaris, rosacea, and seborrheic dermatitis
  - Infectious etiologies
  - Exposure to hydrocarbons, especially halogenated compounds
  - Certain chemotherapeutics, especially kinase inhibitors
  - Genetic disorders



# Summary

- Rosacea
  - Likely caused by immune dysregulation
    - Inflammation
    - Vascular vasodilation
    - Presence of commensal organisms may exacerbate
  - Treatments
    - Identify triggers
    - Topical antibiotics
    - Tetracycline-class systemic antibiotics
    - Topical alpha-agonists
    - Isotretinoin

- Questions?

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