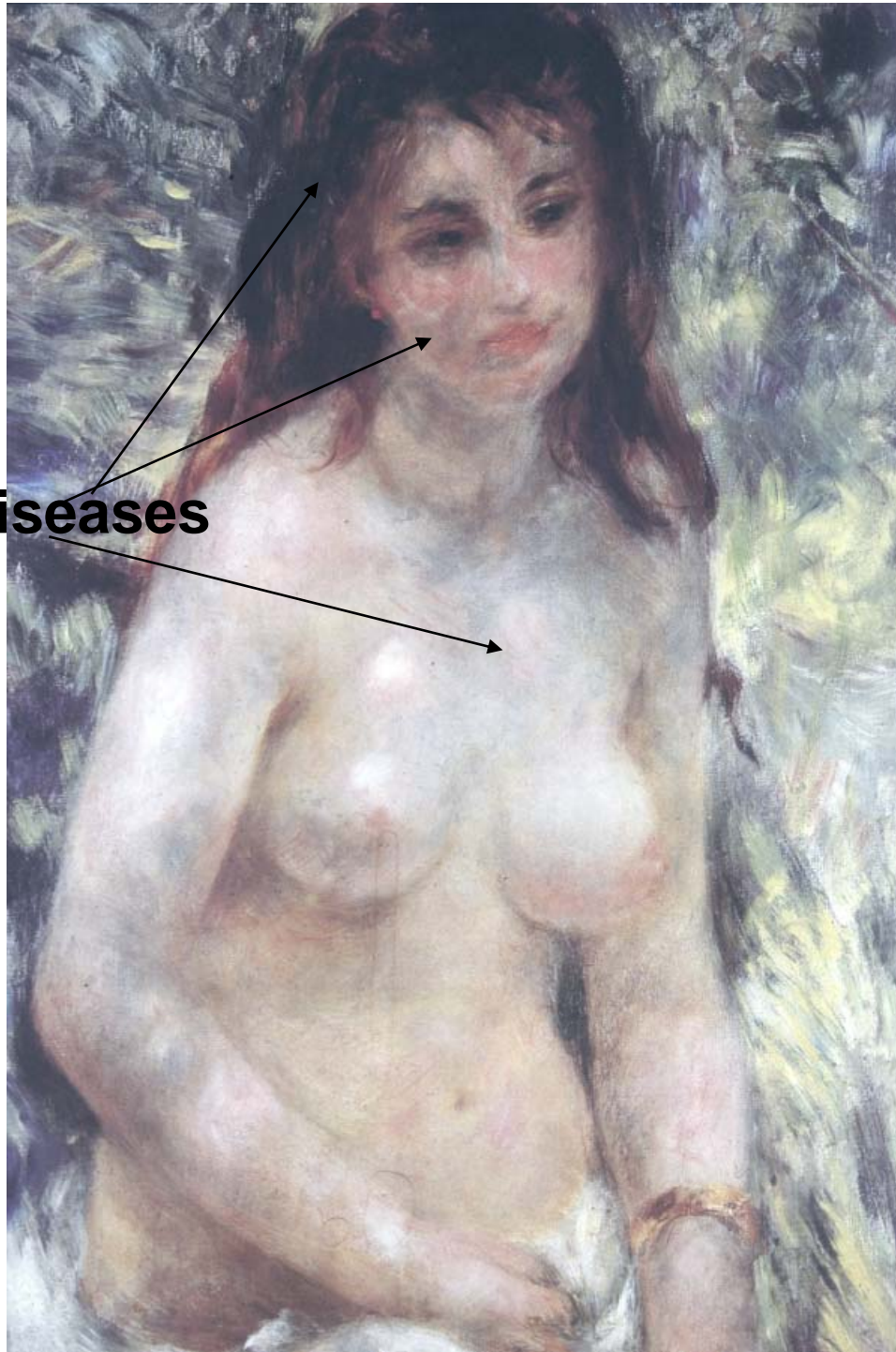


Seborrhoeic diseases

Acne vulgaris

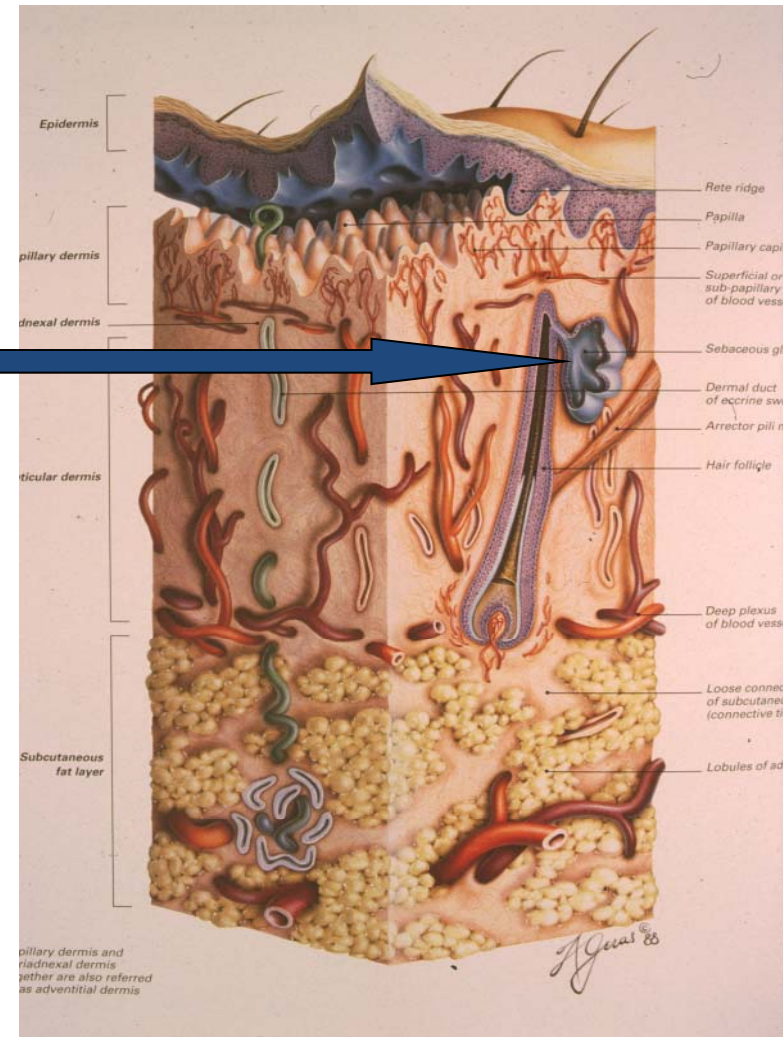
Márta Marschalkó

Seborrheic diseases

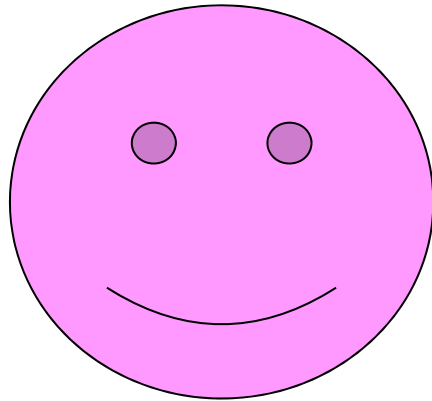


Seborrhoea

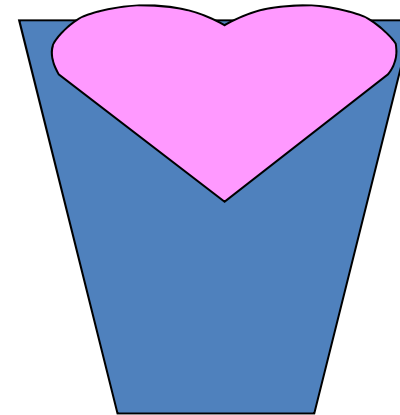
- Sebaceous gland
- Hair follicle
- Sebum lipids
- Seborrhoea: sebum-orrhoea



„Seborrhoeic” regions



Face



Upper part of the chest, back

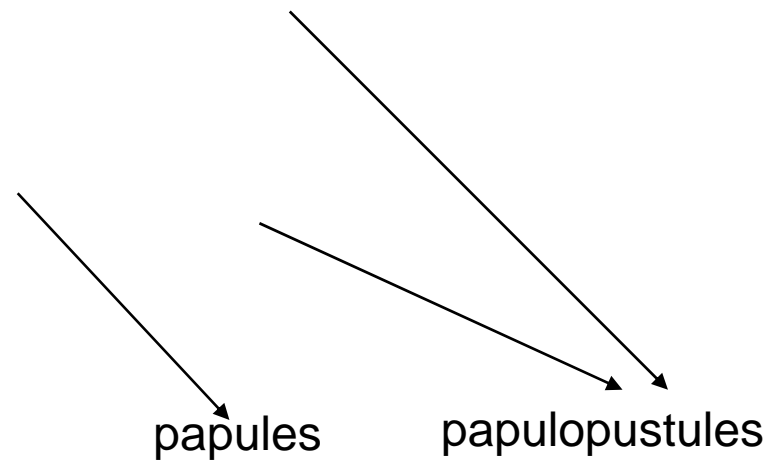
HUMAN SEBUM

- **Contains: squalene, cholesterol, cholesterol esters, wax esters, and triglycerides**
- **bacterial enzymes hydrolyze triglycerides**
- **lipid mixture** reaching the skin surface: **free fatty acids, mono-and diglycerides**

Seborrhoeic diseases: clinical forms

- **Acne vulgaris**
- **Seborrhoeic dermatitis / on the scalp:
dandruff/**
- **Rosacea**
- **Perioral dermatitis**

Acne vulgaris



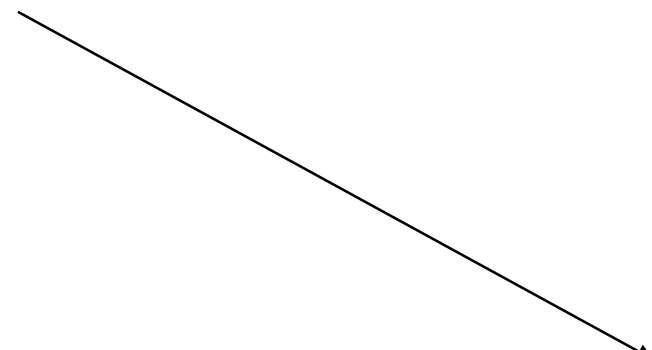
Acne vulgaris

Acne infantum

Seborrhoeic diseases: clinical forms

- Acne vulgaris
- Seborrhoeic dermatitis / on the scalp:
dandruff/
- Rosacea
- Perioral dermatitis

**Seborrhoeic
dermatitis**



Superficial, scaly
plaques

Clinical forms:

1. dry, scaling dermatitis (dandruff)
2. greasy, crusted form
3. moist form

Aetiology: *Pityrosporon ovale*
(yeast)

(on the base of the altered
skin lipids)

Seborrhoeic dermatitis

Skin surface lipids

- Intracellular lipids of corneal layer (sterol esters, cholesterol)
- Fats from sebaceous glands: (triglycerids, wax esters, squalens)

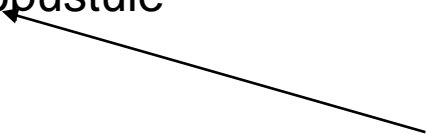
Seborrhoeic dermatitis



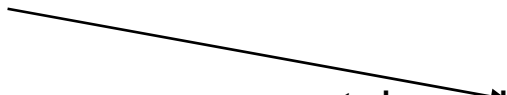
Seborrhoeic diseases: clinical forms

- Acne vulgaris
- Seborrhoeic dermatitis / on the scalp:
dandruff/
- **Rosacea**
- Perioral dermatitis

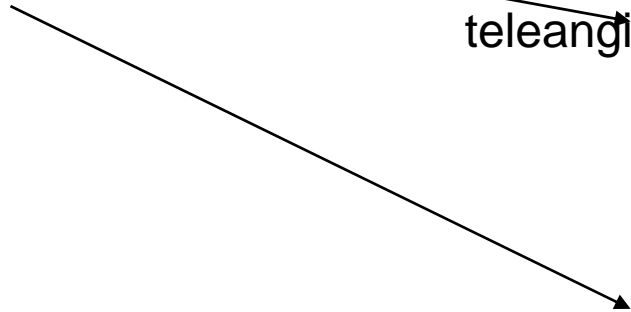
papulopustule



teleangiectasia



papule



Rosacea

Rosacea

Rhinophyma

Seborrhoeic diseases: clinical forms

- **Acne vulgaris**
- **Seborrhoeic dermatitis / on the scalp:
dandruff/**
- **Rosacea**
- **Perioral dermatitis**

Seborrhoeic diseases-elementary lesions

- superficial: scaly dermatitis

- Papules
- Papulopustules
- Nodules
- Cysts

- scars

Acne keloid

Acne vulgaris

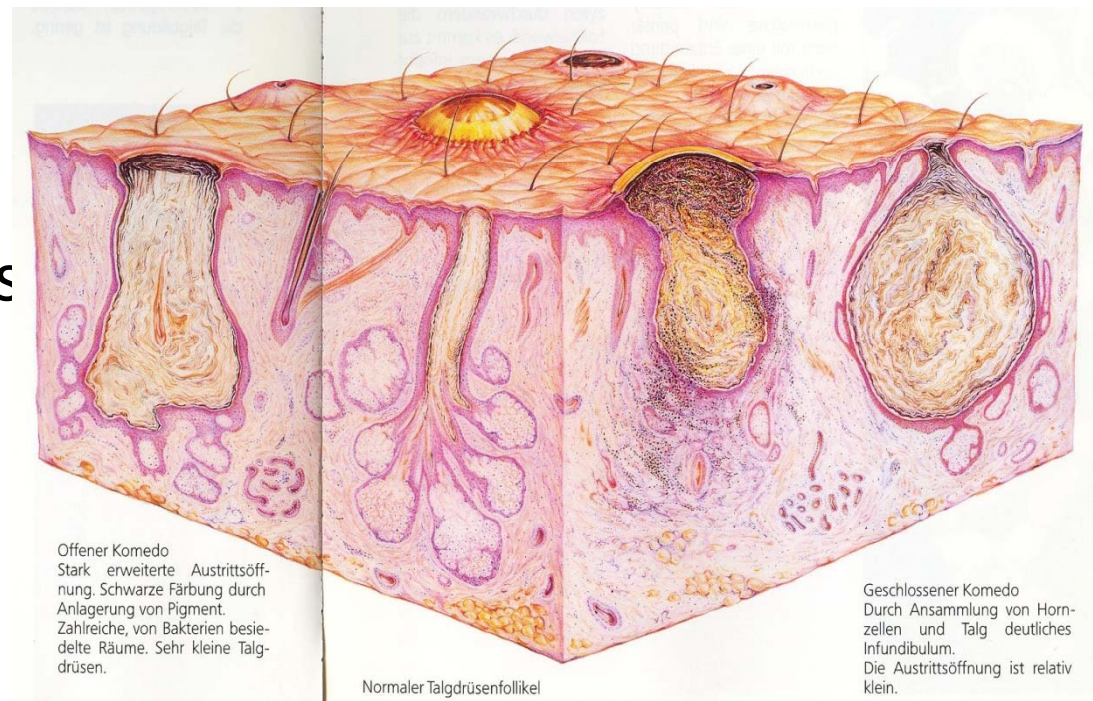
- a sebaceous gland disease
- a process that involves the pilosebaceous unit

Acne vulgaris

- a self-limited disease, seen primarily in adolescents
- lesions consist of **comedones, papules, pustules, nodules,**
- sequelae to active lesions: pitted or hypertrophic **scars**

Acne

- **Clinical findings**
- **Comedo** (open, closed)
- papulopustule
- nodule
- Cyst
- scar (hypertrophic, keloid)



Acne

- Comedo (open, closed)
 - Papulopustule
 - Nodule
 - Cyst
 - Scar (hypertrophic, keloid)
- Acne comedonica
 - Acne vulgaris
 - Nodular acne
 - Cystic acne
 - Acne keloid

Acne comedonica

- comedo

Papulopustular acne- acne vulgaris

Papulopustules

Acne vulgaris

Nodular acne

Acne vulgaris

- a sebaceous gland disease
- a process that involves the **pilosebaceous unit.**

Acne vulgaris-clinical features

- Acne is often an early manifestation of **puberty**; in the young patient the predominant lesions are **comedones**
- The greatest number of cases is seen during the middle-to-late teenage period;
- Subsequently the incidence decreases
- Particularly in women, acne may persist through the third decade or even later

Acne vulgaris-clinical features

- early manifestation of **puberty**; predominant lesions: **comedones**
- greatest number of cases: during the **middle-to-late teenage period**

- **Subsequently the incidence decreases**

Acne vulgaris-pathomechanism

- 1. Follicular hyperkeratosis
- 2. Sebum production
- 3. P. acnes
- 4. Androgens

Acne vulgaris-Pathomechanism

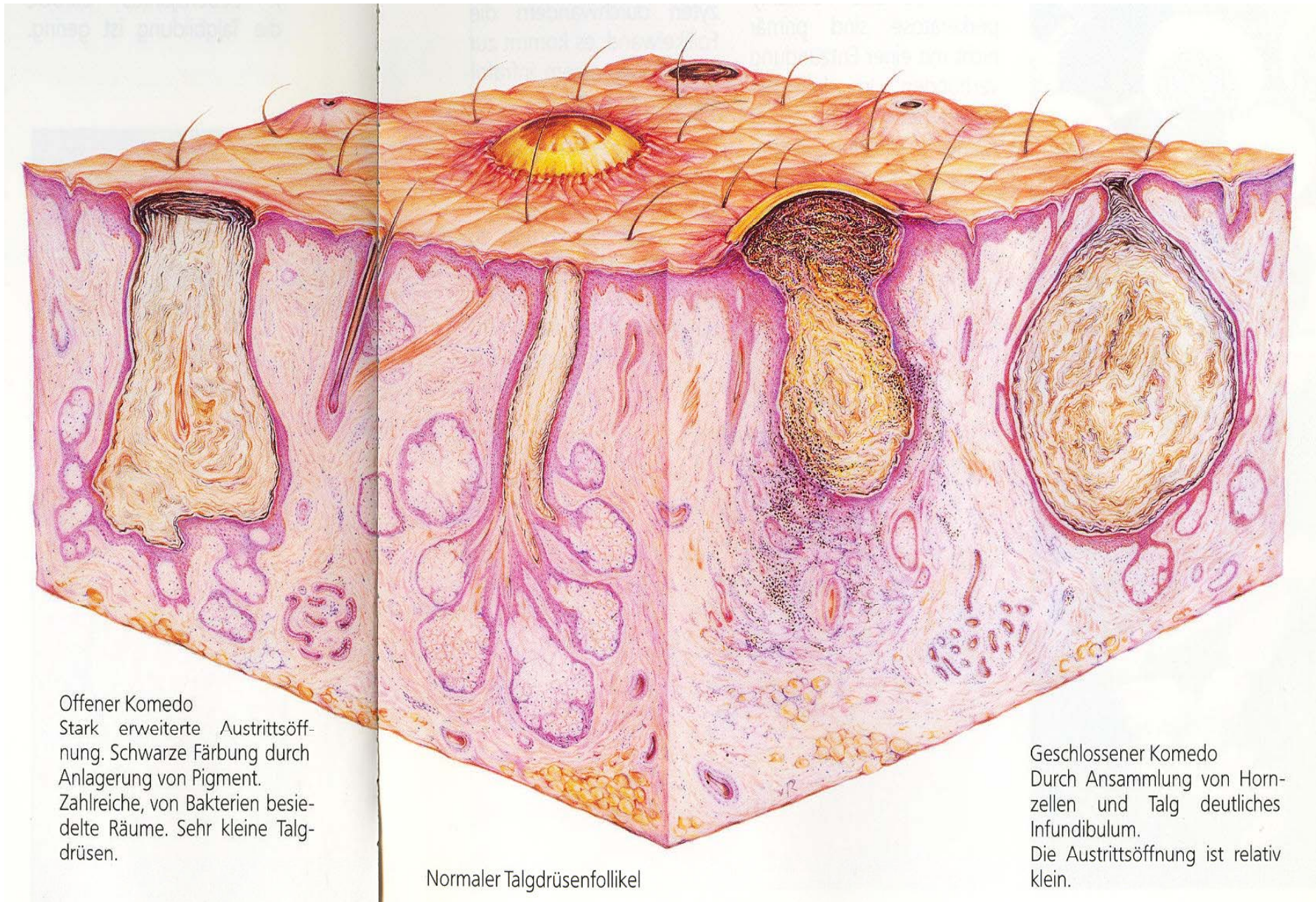
1.

- **primary change: alteration in the pattern of keratinization within the hair follicle**
- **increase in cellular turnover in keratinocytes**

Acne vulgaris-Pathomechanism

1.

- **primary change is an alteration in the pattern of keratinization within the follicle /the keratinous material becomes more dense, the lamellar granules are less numerous/**
- **there is an increase in cellular turnover in comedones.**



Offener Komedo
 Stark erweiterte Austrittsöffnung. Schwarze Färbung durch Anlagerung von Pigment. Zahlreiche, von Bakterien besiedelte Räume. Sehr kleine Talgdrüsen.

Normaler Talgdrüsenfollikel

Geschlossener Komedo
 Durch Ansammlung von Hornzellen und Talg deutliches Infundibulum. Die Austrittsöffnung ist relativ klein.

Acne vulgaris: role of sebum production 2.

- **sebum plays a role in the pathogenesis of the disease**
- **sebum is comedogenic; causes inflammation**

Acne vulgaris: sebum production

- **acne appears:**
in puberty at a time when sebaceous development occurs
- **acne can be controlled by drugs which inhibit sebaceous glands:**
antiandrogens, estrogens, oral retinoids

Suppression

- Comedo formation:
 - Benzoyl peroxid
 - Topical antibiotics
 - Topical retinoids
 - Oral isotretinoin
 - Oral tetracyclin
- Sebum production:
 - Oral isotretinoin
 - antiandrogens

Acne vulgaris: role of sebum production

- **sebum plays a role in the pathogenesis of the disease**
- **sebum is comedogenic; causes inflammation**
- **acne occurs in the neonatal period when the sebaceous glands are well developed**

Acne vulgaris: P. acnes

3.

- the predominant organism in the follicular flora is **Propionibacterium acnes**.
- P. acnes (formerly **Corynebacterium acnes** type 1) and P. granulosum (formerly C. acnes type 2).

Acne vulgaris: P.acnes

3.

- P. acnes is important in the pathogenesis of acne
- **inflammation** results from the production of **free fatty acids**
- P.acnes is the main source of follicular lipases

Acne vulgaris: P. acnes

3.

- **P. acnes also produces other extracellular enzymes: proteases, hyaluronidases**
- **may produce inflammation: the organism has been shown to secrete chemotactic factors**

Suppression

- **P.acnes**

- Benzoylperoxide
- Topical antibiotics
- Oral isotretinoin
- Oral tetracycline
- spironolactone

- **Inflammation:**

- Benzoylperoxide
- Topical antibiotics
- Topical retinoids
- Oral isotretinoin
- Oral tetracycline
- Spironolactone

Acne vulgaris: role of androgens

4.

- Androgens are known to **regulate** the development of the sebaceous gland and sebum production
- they may play a role in the follicular hyperkeratinization seen in acne

Acne vulgaris: role of androgens

4.

- **Hyperandrogenism** should be considered to be a contributing factor to the development of acne in female patients: if sudden in its onset, or associated with **hirsutism** or irregular menstrual periods.
- A medical history and physical examination directed toward eliciting any symptoms or signs of hyperandrogenism should be performed.

Acne vulgaris: role of androgens

4.

- **Hyperandrogenism:**
- a contributing factor to the development of acne in female patients
- association with **hirsutism** or irregular menstrual periods
- A medical history and physical examination directed toward eliciting any symptoms or signs of hyperandrogenism should be performed.

Acne vulgaris: role of androgens

4.

- **patient should be asked about the menstrual periods**
- **hyperandrogenism can also result: deepening of the voice, increase in libido**
- **skin signs associated with hyperandrogenism: hirsutism, male pattern alopecia, truncal obesity**

Acne treatment

- **Local:**

- Benzoylperoxid
- Antibiotics
- retinoids

- **Systemic:**

- Antibiotics (tetracyclin, erythromycin)
- Retinoid: Roaccutane
- antiandrogens

Corticosteroid acne

- **monomorphic papulopustules (on the trunk)**
- **Provoked by topical and systemic steroid treatment**
- **Withdrawal of steroid regression**
- **Treatment: conventional acne therapy**

Drug induced acne

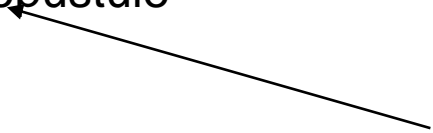
- **Antibiotics: penicillin, macrolids, doxycyclin, oflaxin, chloramphenicol, cotrimoxazole**
- **ACTH, nystatin, isoniazid, itraconazole, hydroxychloroquine, naproxen, Hg, clanzapin, litium**

Rosacea

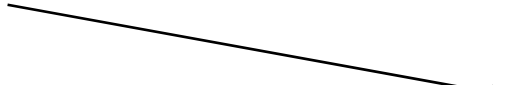
Rosacea

- Third and fourth decades
- Commoner in women
- Flushing, erythema on the face
- Persistent erythema, teleangiectasia
- Papules, pustules, papulopustules

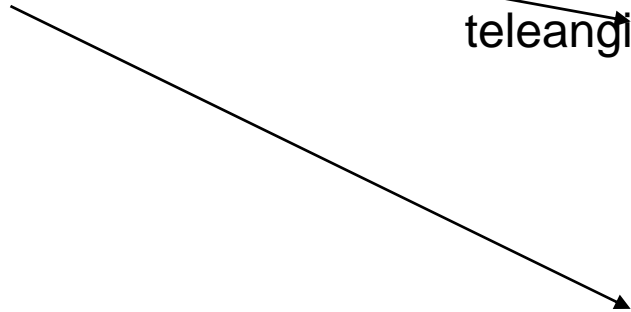
papulopustule



teleangiectasia



papule



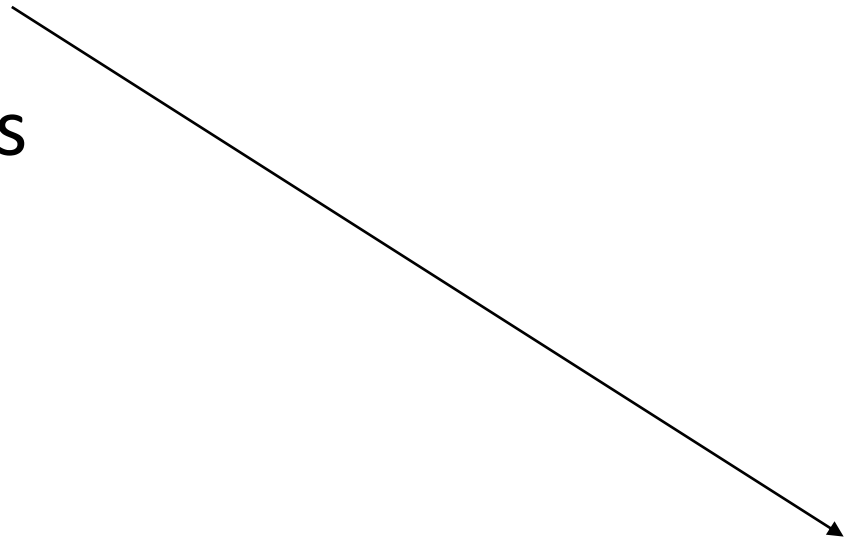
Rosacea

Rhinophyma

- Sebaceous gland hypertrophy concentrated on the nose
- Producing gross tissue overgrowth and hypertrophy

Rosacea- differential diagnosis

- Acne vulgaris
- Contact dermatitis
- Lupus erythematosus
- Light sensitivity
- Perioral dermatitis



SLE-butterfly rash

Contact dermatitis

Rosacea- treatment

- Long term antibiotics- commonly tetracycline (for months)
- Topical metronidazole

Skin symptoms due to liver diseases

- **Discoloration: jaundice**
- **Hyperpigmentation /chloasma, melasma/**
- **Liver palm, teleangiectasia, spider nevus**
- **Purpura, suffusion**
- **Gynecomastia, hypogonadism, hypotrichosis**
- **Porphyria cutanea tarda**
- **Nail abnormalities**
- **Xanthoma, xanthelasma,**
- **pruritus**

Melasma (gravidæ

Purpura, **suffusion**

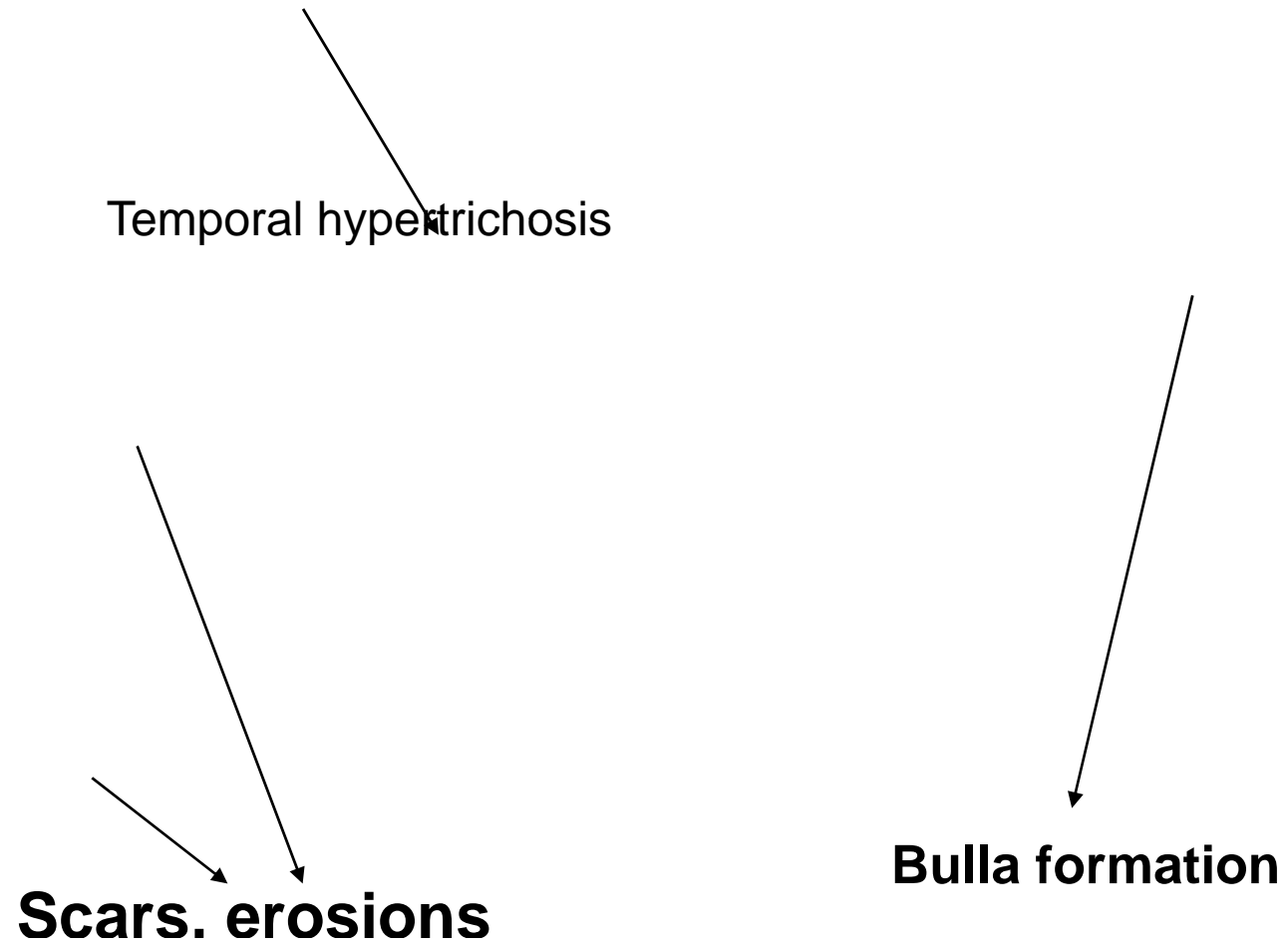
Porphyria cutanea tarda

- **Fragility of the skin**
- **Bullae –scars (on sun exposed areas)**
- **Hypertrichosis (face, temporal region, arms)**
- **Hyperpigmentation (patchy, diffuse)**

Porphyria cutanea tarda

Porphyria cutanea tarda

PCT



Porphyria cutanea tarda

- **Photosensitive disorder**
- **Abnormal haem biosynthesis- uroporphyrin decarboxylase deficiency**
- **Familial, or sporadic**
- **Precipitated by exposure to chemicals toxic to liver /alcohol, hexachlorobenzene /, or drugs metabolized in the liver /estrogens/**

PCT

- **Type I. : sporadic /alcohol, estrogens, virus, SLE /**
- **Type II.:familial /chemicals /hexachlorobenzene/**

PCT

- **Erosion, bulla formation, scars**
- **Hypertrichosis /temporal/**
- **Hyperpigmentation**

- **On sun exposed areas**

Effects of corticosteroids on the skin

- **Steroid acne**
- **Atrophia**
- **Striae distensae**
- **Hypertrichosis**
- **Cushing syndr.**
- **Infections**

● **Striae distensae**

hypertrichosis

Cushing face

Steroid rosacea, acne

Striae

Skin atrophie



Striae

Skin symptoms associated with diabetes mellitus

- Infections /bacterial, fungal /
- Xanthomatosis
- Microangiopathia
- Macroangiopathia
- Neuropathia
- Rubeosis faciei
- Bullosis diabeticorum
- Scleroedema
- Necrobiosis lipoidica
- Malum perforans pedis

Diabetic foot

Diabetic foot

Necrobiosis lipoidica

Malum perforans pedis

Skin symptoms in diabetes mellitus

- **1. Pruritus – generalised, localised**
- **2. Infections –Staphylococcus, Candida**
- **3. Metabolic disorders: xanthelasma, xanthomatosis**
- **4. Angiopathy: necrobiosis lipoidica, diabetic foot**

Microangiopathia

- **Basal membran thickening**
- **Increased permeability**
- **Due to glycosylation of basal membrane proteins / collagen type IV, laminin, heparan sulphate/**

Microangiopathy

Microvascular abnormalities include increased “leakiness” or vessel wall permeability

Decreased response of vessels to sympathetic innervation

Decreased ability to respond to thermal and hypoxemic stress

Concomittant arteriosclerosis of large vessels

Immunodeficiency

Derangements of immunoregulatory mechanisms frequently occur in DM

Hyperglycemia and ketoacidosis diminish chemotaxis, phagocytosis, and bactericidal ability of white blood cells

Lipid metabolism

Dysregulation of lipid metabolism is a result of insulin deficiency

The activity of lipoprotein lipase is directly dependent on insulin, making insulin central to the processing of triglyceride-rich chylomicrons and very-low-density lipoproteins (VLDLs)

In insulin-deficient diabetic patients, defective lipid processing may lead to massive hypertriglyceridemia

1. Generalized pruritus

- Skin dryness
- Intercellular hyperglycemia
- Osmotic changes in the epidermis
- Diabetic thick skin
- Decreased cellular immunity

2. Infectious conditions

Streptococci

Staphylococci

Yeasts

Dermatophytes

Mucormycosis

3. Eruptive xanthomatosis

**Lipoproteins, chylomicrons permeate cutaneous vessel walls
and accumulate in macrophages in the dermis**

4. Microcirculation disturbance

Necrobiosis lipoidica

Pruritus

- Uremie
- Obstruct. bile disease /biliaris cirrh./
- Endocrin disorders: thyreotoxicosis, hypothyreosis, diabetes mellitus, carcinoid
- Myeloproliferative processes: Hodgkin disease, polycythemia, lymphoma, myeloma multiplex
- Visceral ca
- Anemie / Fe /