Editied Dermatology Past Papers

> "**The road goes ever on and on**" J.R.R. Tolkien's

Let's begin.

All questions from 017 and previous years

Normal skin:

Page 2 (Test Bank - Normal skin)x

Question 1:
Not true about normal skin:
a. Stratum corneum is devoided from nuclei
b. Pacinian corpuscles are for touch
c. Sweat glands controlled by neurons
Answer: B

High-Yield Context (from Dermatology Summary, pp. 3-4):

Normal skin

- **The skin is the largest organ of the body**, forming the outermost surrounding layer; separates the body from the surrounding environment

Types of skin

- **Glabrous skin:**
  - Present on the palms and soles
  - Has thick keratin layer
  - Dermatoglyphics “skin markings or patterns”
  - Specific nerve organs
  - Lack of hair follicles or sebaceous glands
- **Hairy skin:**
  - Present all over the body except palms and soles
  - Wide variations according to anatomic site:
    - Scalp: large sebaceous glands and hair follicles
    - Axilla : large apocrine sweat glands

Embryology

- Epidermis → ectoderm
- Dermis & SQ → mesoderm
- Melanocytes → neural crest

Functions

- Protection, Heat regulation, Perception of sensation
- Secretion of sebum, sweat, Pro-vitamin D synthesis
- Mirror of the body because the skin act an organ of expression:
  - Anxiety - sweating.
  - Fear - pallor.
  - Anger - redness.

Basic structure → 3 components

-

Epidermis – stratified squamous epithelium, consists of mainly **keratinocytes**
○
Dermis– forms the structural foundation of the skin, supporting its superficial + deep layers
■ The Dermis determines the thickness of skin, the epidermis thickness is the same in all body
○
Subcutaneous-adipose layer; acts as a fat + heat store

Epidermis → 4 major types of cells
● Keratinocytes
● Melanocytes
● Langerhans cells
● Merkel cell

 # Melanocytes → neural crest \ Langerhans cells ,Merkel cell → bone marrow

Keratinocytes → 4 layers

1. **Basal cell layer**
   a. 1st row of cells, columnar
   b. Mitotic activity restricted to these cells, and form the cells of other layers
   c. Melanocytes are found between the basal cells
2. **Prickle cell layer (spinous, squamous):**
   a. Polygonal cells
   b. Intercellular cement (
   desmosomes) and intercellular cement (**tonofilaments**)
3. **Granular cell layer:**
   a. Contain
   keratohyalin granules
4. **keratin layer (S. corneum, horny layer):**
   a. Results from keratinization →
   no nucleus in cells
   b. Function as a normal barrier

The Epidermis
● Constituted by keratinizing, stratified squamous epithelium
● Keratinocytes form 95% of the epidermal cells; the other 5% form the melanocytes, Langerhans cells + Merkel cells
● The basement membrane has upward projections into the epidermis formed of the dermis called dermal papillae.
● The downward parts of the epidermis are called rete ridges

Dermis

1. Connective tissue fibers: collagen, elastin, reticulin
2. Ground substance of mucopolysaccharides
3. Cellular: fibroblasts, fibrocytes, mast cells
4. **Nerves:**
   - **Sensory: Messner, Pacinian**, Muco-cutaneous n. organs, Free nerve endings
   - Autonomic
     ●
     Meissner corpuscle
     ●
     Mediates touch
     ● In the papillary dermis
     ● More at the tips of fingers
     ●
     Pacinian corpuscle
     ● Deep in dermis
     ●
     Mediates pressure and vibration

- Onion shaped
- Palms, soles, areola and genitalia

5. blood vessels: superficial and deep
6. Muscles → voluntary (skeletal) & involuntary (smooth)
7. Lymphatics

Epidermal appendages

- These are structures originating from the epidermis but present in the dermis:
- Keratinous structures: hair/ nail
- Glandular structures:
  - Apocrine sweat glands
  -

Eccrine sweat glands

  - Sebaceous sweat glands
  -

Eccrine sweat glands

- Are the major sweat glands in the body
- Almost present everywhere on the human body
- Produce clear, odorless fluid containing mainly water and electrolytes →

Merocrine secretion

- Eccrine glands are composed:
  - intraepidermal spiral duct, the "acrosyringium";
  - dermal duct, comprising a straight and coiled portion
  - secretory tubule, coiled deep in the dermis or hypodermis.
-

Eccrine glands are innervated by the sympathetic nervous system, primarily by cholinergic fibers

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Question 2:

Which of the following is false about normal skin:

The Meissner’s corpuscles is responsible for pressure sensations

(No options provided in OCR for this question, but the statement itself is a true/false type of question. Based on the summary, Meissner's corpuscles are for touch, and Pacinian are for pressure/vibration. So, the statement "Meissner’s corpuscles is responsible for pressure sensations" is indeed false.)

High-Yield Context (from Dermatology Summary, p. 4):

Dermis

4)

Nerves:

-

Sensory: Messner, Pacinian, Muco-cutaneous n. organs, Free nerve endings

- Autonomic
-

Meissner corpuscle

-

Mediates touch

- In the papillary dermis
- More at the tips of fingers
-

Pacinian corpuscle

- Deep in dermis
-

Mediates pressure and vibration

- Onion shaped
- Palms, soles, areola and genitalia

Question 3:

Which of the following is false about normal skin:

a. Sebaceous glands originate from ectoderm

b. Merkel cells are dendritic cells that are present near nerve endings
c. Mitotic cells only seen at the basal layer
d. Melanocytes connect to 36 surrounding keratinocytes
e. Melanocytes appear clear and big relative to surrounding cells under the microscope
Answer: B
(The OCR indicates B, however, Merkel cells are dendritic-like and near nerve endings related to touch. Sebaceous glands originate from ectoderm as part of epidermal appendages. Mitotic cells are restricted to basal layer. Melanocytes do form epidermal melanin units. The appearance of melanocytes as clear cells is also a known histological feature. Let's review the summary for clarity).

Upon reviewing the summary:- Epidermis (including appendages like sebaceous glands) is from ectoderm.- Merkel cell: "Bell-like shaped cells, at the basal layer", "Free nerve endings below it, related to touch". The summary doesn't explicitly call them dendritic but mentions their shape and touch relation.- Basal cell layer: "Mitotic activity restricted to these cells".- Melanocytes: "Dendritic cells that form and secrets melanin by the mean of dendrites to the surrounding keratinocytes". The exact number 36 is not in this summary.

Given the options and typical knowledge, statement 'b' that Merkel cells are dendritic and present near nerve endings is generally considered true. Sebaceous glands do originate from ectoderm. Mitotic cells are in basal layer. Melanocytes do form epidermal melanin units (the 36 keratinocytes figure is a common textbook value). Melanocytes can appear as clear cells. If answer B is indeed correct, there might be a nuance not captured by the summary or a misunderstanding of "dendritic cells" in this context (Langerhans are the primary APC dendritic cells). However, the provided summary states: "# Melanocytes → neural crest \ Langerhans cells ,Merkel cell → bone marrow" and for Langerhans cells: "Dendritic Antigen presenting cells", for Merkel cells: "Bell-like shaped cells...related to touch". The summary does not call Merkel cells "dendritic cells".

High-Yield Context (from Dermatology Summary, pp. 3-4):

● **Embryology**
○
Epidermis → ectoderm (This implies epidermal appendages like sebaceous glands also originate from ectoderm)
○ Dermis & SQ → mesoderm
○ Melanocytes → neural crest
●
Epidermis → 4 major types of cells
●
Keratinocytes
1.
Basal cell layer
a. 1st row of cells, columnar
b.
Mitotic activity restricted to these cells, and form the cells of other layers
●
Melanocytes
○
Dendritic cells that form and secrets melanin by the mean of dendrites to the surrounding keratinocytes
●
Langerhans cells
○
Dendritic Antigen presenting cells
○ Supra-basal layer
●
Merkel cell
○ Bell-like shaped cells, at the basal layer
○ Free nerve endings below it,
related to touch# *Melanocytes → neural crest \ Langerhans cells ,Merkel cell → bone marrow*

Epidermal appendages
● These are structures originating from the epidermis but present in the dermis:
● Keratinous structures: hair/ nail
● Glandular structures:
■ Apocrine sweat glands

■ Eccrine sweat glands
■
Sebaceous sweat glands (Mistake in summary, should be **Sebaceous glands**)
● Related to hair follicles, and their ducts open in it.
● Present all over the body except the palms and soles.
●
Holocrine secretion.
● Secretion controlled by
androgens mainly.

Question 4:
Sebaceous gland are normally not found in:
a. Face
b. Vermilion of lip
c. Upper back
d. Buccal mucosa
e. Areola of nipple
Answer: D
High-Yield Context (from Dermatology Summary, p. 4):

Sebaceous glands
● Related to hair follicles, and their ducts open in it.
●
Present all over the body except the palms and soles.
● Holocrine secretion.
● Secretion controlled by androgens mainly.

(The summary states sebaceous glands are present all over the body except palms and soles. Areas like face, upper back, and areola of nipple are known to have them. Vermilion of the lip has some specialized sebaceous glands (Fordyce spots, though not typical). Buccal mucosa is an internal mucous membrane and typically lacks hair follicles and associated sebaceous glands).

Question 5:
Glabrous skin characterized by all of the following except:
a. Dermatoglyphics
b. Thick epidermis
c. Presence of encapsulated organs
d. Presence of sebaceous gland
Answer: D
High-Yield Context (from Dermatology Summary, p. 3):
● **Types of skin**
●
Glabrous skin:
○ Present on the
palms and soles
○ Has
thick keratin layer (implying thick epidermis)
○
Dermatoglyphics “skin markings or patterns”
○
Specific nerve organs (encapsulated organs like Meissner's, Pacinian)
○
Lack of hair follicles or sebaceous glands

Question 6:
Sensation of pressure in skin is mediated by:
a. Autonomic n.
b. Mucocutaneous end organs

c. Vater pacini corpuscles
d. Meissner corpuscles
Answer: C

High-Yield Context (from Dermatology Summary, p. 4):

Dermis
4)
Nerves:
-
Sensory: Messner, Pacinian, Muco-cutaneous n. organs, Free nerve endings
- Autonomic
●
Meissner corpuscle
●
Mediates touch
●
Pacinian corpuscle (Vater-Pacini)
● Deep in dermis
●
Mediates pressure and vibration

Question 7:
Mitotic division in epidermis is limited to: *
a. Basal cells
b. Melanocyte
c. Granular cells
d. Prickle cells
Answer: A

High-Yield Context (from Dermatology Summary, p. 3):

Keratinocytes → 4 layers

1. **Basal cell layer**
   a. 1st row of cells, columnar
   b.
   Mitotic activity restricted to these cells, and form the cells of other layers
   c. Melanocytes are found between the basal cells

Question 8:
Melanin of Caucasians & Negros differs in all except:
a. Size of melanosomes
b. No. of melanosomes in melanocytes
c. Degree of despereal of melanosomes
d. No. of melanin producing cells
e. No. of melanosomes in keratocytes
Answer: D
(The number of melanocytes (melanin producing cells) is generally similar across races; the difference lies in melanosome size, number within keratinocytes, and degradation rate.)

High-Yield Context (from Dermatology Summary, p. 3):

● **Epidermis → 4 major types of cells**
●
Melanocytes
○ Dendritic cells that form and secrets melanin by the mean of dendrites to the surrounding keratinocytes

 # Melanocytes → neural crest(The summary is brief on melanocytes and doesn't detail racial differences in melanin/melanosomes. This question tests knowledge beyond the provided summary details on this specific point, but the general concept of melanocytes producing melanin is there.)

Question 9:
Different color of people caused by differences in all except:
a. Size of melanosomes
b. Number of melanosomes
c. Concentration of melanocytes
d. Rate of melanocyte consumption

(OCR ends here for this question, but likely an 'e' option and an answer would follow. The number/concentration of melanocytes is generally similar between races. Differences arise from melanosome size, activity, distribution, and degradation rate.)

High-Yield Context (from Dermatology Summary, p. 3):

● **Epidermis → 4 major types of cells**
●
Melanocytes
○ Dendritic cells that form and secrets melanin by the mean of dendrites to the surrounding keratinocytes

 # Melanocytes → neural crest(Similar to the previous question, the summary's detail on this is limited. The context covers the existence and function of melanocytes.)

Page 3 (Test Bank - Normal skin cont.)

Question 10 (continued from Page 2, question 9):
Different color of people caused by differences in all except:
a. Size of melanosomes
b. Number of melanosomes
c. Concentration of melanocytes
d. Rate of melanocyte consumption
e. Number of melanocytes
Answer: E
(The summary doesn't go into this level of detail about skin color differences. However, general dermatological knowledge suggests that the number of melanocytes is roughly the same across different skin types; it's the activity of melanocytes, and the size, number, and distribution of melanosomes that vary. So, 'Number of melanocytes' (or 'Concentration of melanocytes' if they mean the same thing) is a plausible 'except' option. If the answer is E, it implies that 'Number of melanocytes' is a factor, which contradicts the general understanding that the number is similar. Let's assume 'concentration' and 'number' of melanocytes are being used to mean density in the skin, which is generally similar).

High-Yield Context (from Dermatology Summary, p. 3):

● **Epidermis → 4 major types of cells**
●
Melanocytes
○ Dendritic cells that form and secrets melanin by the mean of dendrites to the surrounding keratinocytes

 # Melanocytes → neural crest(The provided summary is very brief on the determinants of skin color and doesn't detail the differences between races/individuals. It only states what melanocytes are and their origin.)

Question 11:
One of the following cells is dendritic:
a. Langerhans
b. Histiocytes
c. T cells
d. B cells
Answer: A

High-Yield Context (from Dermatology Summary, p. 3):

Epidermis → 4 major types of cells
● Keratinocytes
● Melanocytes
○
Dendritic cells that form and secrets melanin...

●
Langerhans cells
○
Dendritic Antigen presenting cells
○ Supra-basal layer
● Merkel cell

*# Melanocytes → neural crest \ **Langerhans cells** ,Merkel cell → bone marrow(The summary explicitly calls both Melanocytes and Langerhans cells "dendritic". However, Langerhans cells are primarily known for their antigen-presenting dendritic role in the epidermis.)*

Question 12:
Meibomian gland is an:
a. Eccrine gland
b. Apocrine gland
c. Sebaceous gland
d. Holocrine gland
e. Lacrimal gland
Answer: C
(Meibomian glands are modified sebaceous glands. Sebaceous glands are holocrine glands).

High-Yield Context (from Dermatology Summary, p. 4):

Epidermal appendages
● Glandular structures:
■ Apocrine sweat glands
■ Eccrine sweat glands
■
Sebaceous sweat glands (this is a typo in the summary, it should be **Sebaceous glands**)
●
Sebaceous glands
● Related to hair follicles, and their ducts open in it.
● Present all over the body except the palms and soles.
●
Holocrine secretion.
● Secretion controlled by androgens mainly.

(The summary classifies sebaceous glands as having holocrine secretion. While it doesn't mention Meibomian glands specifically, it provides the characteristics of sebaceous glands.)

Question 13:
Wrong statement:
Sweat glands are controlled by hormones

High-Yield Context (from Dermatology Summary, p. 4):

Eccrine sweat glands
● Eccrine glands are innervated by the
sympathetic nervous system, primarily by cholinergic fibersApocrine sweat glands*(The summary does not explicitly state hormonal control for apocrine sweat glands, but mentions for sebaceous glands: "Secretion controlled by androgens mainly." Eccrine sweat glands are neurally controlled. Apocrine sweat glands are also influenced by adrenergic nerve fibers and circulating hormones, particularly androgens, play a role in their development and activity. So the general statement "Sweat glands are controlled by hormones" might be considered partially true for apocrine but less so or not directly for eccrine sweat glands in terms of primary control of secretion.)*

Based on the summary:- Eccrine glands: sympathetic nervous system (cholinergic fibers).- Apocrine glands: The summary mentions their secretion method ("Decapitation secretion") and location but not their control mechanism explicitly.- Sebaceous glands: "Secretion controlled by androgens mainly." (These are not sweat glands).

The most accurate statement from the summary is that eccrine sweat glands are neurally controlled. If "sweat glands" encompasses both, and apocrine glands are influenced by hormones, the statement isn't entirely "wrong". However, if the primary control mechanism is considered, neural control is key for eccrine sweat secretion.

Question 14:
Wrong statement:
a. Apocrine sweat glands are characterized by decapitation secretion.
b. Eccrine sweat glands have cholinergic innervation.
c. Sebaceous glands are controlled by androgens.
d. None of the above
Answer: D

High-Yield Context (from Dermatology Summary, p. 4):

Apocrine sweat glands
●
Decapitation secretion: the apical portion of the secretory cell of the gland pinches off and enters the lumen.

Eccrine sweat glands
● Eccrine glands are innervated by the
sympathetic nervous system, primarily by cholinergic fibers

Sebaceous glands
● Secretion controlled by
androgens mainly.

(All statements a, b, and c are explicitly supported by the summary. Therefore, "None of the above" being wrong implies that one of the statements a, b, or c is wrong, which contradicts the summary. Assuming the answer key D means that "None of the above" is the correct choice (i.e., statements a, b, and c are all true), then the context supports this.)

Question 15:
Sense of touch mediated by:
a. Free nerve endings
b. Meissner corpuscles
c. Pacini corpuscles
d. Muco-cutaneous endings
e. Superficial nerve plexus
Answer: B

High-Yield Context (from Dermatology Summary, p. 4):

Dermis
4)
Nerves:
-
Sensory: Messner, Pacinian, Muco-cutaneous n. organs, Free nerve endings
- Autonomic
●
Meissner corpuscle
●
Mediates touch
● In the papillary dermis
● More at the tips of fingers
●
Pacinian corpuscle
● Deep in dermis
●
Mediates pressure and vibration*(The summary lists Free nerve endings, Meissner's, and Pacinian corpuscles as sensory nerve structures. Meissner's corpuscles are specifically stated to mediate touch.)*

Question 16:
Function of Meissner corpuscle:
a. Sense of touch
b. Erector pili
c. Pressure
d. Innervates smooth muscles of vessels
answer: A

High-Yield Context (from Dermatology Summary, p. 4):

Dermis
4)
Nerves:
-
Sensory: Messner, Pacinian, Muco-cutaneous n. organs, Free nerve endings
- Autonomic
●
Meissner corpuscle
●
Mediates touch
● In the papillary dermis
● More at the tips of fingers

(Erector pili muscles are innervated by autonomic nerves. Pressure is mediated by Pacinian corpuscles. Smooth muscles of vessels are also innervated by autonomic nerves.)

Psoriasis

Question 17:
Parakeratosis is a specific feature of:
a. Lichen planus
b. Psoriasis
c. Acute eczema
d. Ichthyosis vulgaris
answer: B

High-Yield Context (from Dermatology Summary - Psoriasis, p. 5 and from general knowledge about Psoriasis histology in the summary on pp. 142-143 of the "Dermatology Lecture Slides - Psoriasis"):

From Dermatology Summary - Psoriasis (p. 5):
●
Pathophysiology
○ Genetic predisposition + Precipitating factors “Stress, infection, local trauma Alcohol, drugs,childbirth.”
○
Hyperkeratosis “increased thickness + Thick keratin scale ”, **parakeratosis** ”keratocytes retain their nuclei”, poorly adherent and easily scraped off keratocytes (‘Auspitz sign’)
○ Vasodilation, angiogenesis → Inflammation → swelling (oedema) and erythema
○ sterile pustules “accumulation of inflammatory cells” → in palmo-plantar pustulosis

From Dermatology Lecture Slides - Psoriasis (p. 142):*Slide describing the pathophysiology of Psoriasis shows images and text indicating:*
• Keratinocytes grow from the basal layer and slowly migrate to the surface.
• In normal skin, cell turnover takes about 23 days; however, psoriasis cell turnover is rapidly accelerated, taking only 3–5 days for cells to reach the surface and accumulate in large numbers (hyperkeratosis).
• Keratinocytes normally lose their nuclei as they move to the skin surface; however, in psoriasis they move so quickly that the cells retain their nuclei throughout the epidermis, seen as
parakeratosis histologically.

(Parakeratosis (retention of nuclei in the stratum corneum) is a key histological feature of psoriasis, mentioned in both the summary and explicitly in the lecture slides referenced by the summary structure.)

Question 18:
Commonest manifestation of psoriasis in nails is:
a. Onycholysis
b. Pitting
c. Subungual hyperkeratosis
d. Discoloration
e. None of these
Answer: B

High-Yield Context (from Dermatology Summary - Psoriasis, p. 5 and Nail changes section in general, p. 31-32; Psoriatic Arthritis lecture slides, p. 147):

From Dermatology Summary - Psoriasis (p. 5):

●

Psoriatic nail dystrophy

○

onycholysis, subungual hyperkeratosis, pitting, Beau's lines (transverse lines, groove), splinter hemorrhages (longitudinal black lines)

From Dermatology Summary - Diseases of the Nails (Common dermatoses and the nail unit, p. 32):

●

Psoriasis → common, 80% have nail involvement, **pitting**, transverse ridges, onycholysis, oily spots, subungual hyperkeratosis

From Dermatology Lecture Slides - Psoriatic Arthritis (Nail changes, p. 147):

1. **Onycholysis** (lifting of the nail plate off the nail bed) due to abnormal cell adhesion; this usually manifests as a white or salmon patch on the nail plate.
2. **Subungual hyperkeratosis** (accumulation of chalky-looking material under the nail) due to excessive proliferation of the nail bed that can ultimately lead to onycholysis.
3. **Pitting** (very small depressions in the nail plate) which results from parakeratotic (nucleated) cells being lost from the nail surface.

(Pitting is listed as one of the common nail changes in psoriasis.)

Question 19:
All are of psoriasis histopathological changes EXCEPT:
a. Hyperkeratosis
b. Parakeratosis
c. Munro's abscesses
d. Epidermal atrophy
e. suprapapillary plates thinning
Answer: D
(Psoriasis is characterized by epidermal acanthosis (thickening), not atrophy. Suprapapillary plates are thinned.)

High-Yield Context (from Dermatology Summary - Psoriasis, p. 5, and general Psoriasis histology from Lecture Slides pp. 142-143):

From Dermatology Summary - Psoriasis (p. 5):

●

Pathophysiology

○

Hyperkeratosis "increased thickness + Thick keratin scale ", **parakeratosis** "keratocytes retain their nuclei"
○ Vasodilation, angiogenesis → Inflammation → swelling (oedema) and erythema
○ sterile pustules "accumulation of inflammatory cells" → in palmo-plantar pustulosis

(Munro's microabscesses - collections of neutrophils in stratum corneum - are classic. Suprapapillary plate thinning is also classic due to elongation of dermal papillae. Epidermal hyperplasia/acanthosis is characteristic, not atrophy.)

From Dermatology Lecture Slides - Psoriasis (p. 142, implicitly, and general knowledge):
• Keratinocytes ... accumulate in large numbers (
hyperkeratosis).
• ...cells retain their nuclei throughout the epidermis, seen as
parakeratosis histologically.

(The summary itself on p.5 focuses on hyperkeratosis and parakeratosis. Munro's abscesses and suprapapillary plate thinning are key histological features taught with psoriasis, even if not explicitly in the short summary section on p.5, they are part of the broader understanding referenced by the lecture context for psoriasis which the summary points to.)

Question 20:
Why does nail pitting occur in patients with psoriasis?
a. Leakage of blood of dilated capillaries

b. Abnormal cell adhesion
c. Intermittent inflammation of the nail bed
d. Loss of parakeratotic cells from the nail surface
e. Excessive proliferation of the nail bed
Answer: D

High-Yield Context (from Dermatology Lecture Slides - Psoriatic Arthritis, Nail changes, p. 147):

1. **Pitting** (very small depressions in the nail plate) **which results from parakeratotic (nucleated) cells being lost from the nail surface.**

(The summary on p.5 just lists pitting as a feature. The lecture slides context for nails (p.147) explicitly states the mechanism for pitting.)

Question 21:
Psoriasis type caused by streptococcal infection:
a. Flexural
b. Nodular
c. Guttate
Answer: C

High-Yield Context (from Dermatology Summary - Psoriasis, p. 6):

● **Types**
○ plaque psoriasis.
○
Guttate → widespread small plaques scattered on the trunk and limbs, preceded by sore throat with GBS (Group B Strep, though often Group A Strep is implicated)
○ Inverse.
○ Palmo-plantar pustular psoriasis (PPPP) → yellow then brown
○ Acute generalised pustular psoriasis (Von zumbusch) → severe and unstable, precipitated by systemic steroids
○ Acropustulosis → around the nails and the fingertips
○ Flexural psoriasis → axilla, groin, natal cleft, beneath the breasts and in skin folds. No scaling
○ Napkin psoriasis → exudative
○ Erythrodermic → all skin, no scaling, triggers → steroid withdrawal, infection, alcohol, antimalarials, lithium and low calcium

(The summary explicitly links Guttate psoriasis with preceding sore throat (streptococcal infection).)

Question 22:
Psoriatic erythroderma complications EXCEPT:
a. Temperature dysregulation
b. Dehydration
c. Sepsis
d. None of the above
Answer: C
(The OCR says C. However, sepsis IS a complication. Perhaps the question or answer key is flawed. Erythroderma leads to heat loss (temperature dysregulation), fluid loss (dehydration), protein loss, and increased risk of infection (sepsis) and high-output cardiac failure.)

The provided answer C for "Sepsis" as the EXCEPTION is highly unusual, as sepsis is a known and serious complication of erythroderma. Let's check the summary. The summary notes complications of erythrodermic psoriasis as "heart failure, hypothermia, dehydration". Hypothermia is a form of temperature dysregulation. Sepsis isn't directly listed but is a general risk with extensive skin barrier breakdown.

High-Yield Context (from Dermatology Summary - Psoriasis, p. 6):

● **Types**
○
Erythrodermic → all skin, no scaling, triggers → steroid withdrawal, infection, alcohol, antimalarials, lithium and low calcium
■
Complications → heart failure, hypothermia, dehydration

(The summary lists heart failure, hypothermia (temperature dysregulation), and dehydration. Sepsis is a very common complication of erythroderma due to skin barrier failure, even if not explicitly in this short list. If the answer given (C) is correct, it implies sepsis is NOT a complication, which is medically inaccurate for generalized erythroderma. This question might be flawed or testing a very specific nuance not covered by the summary.)

Question 23:
Wrong about psoriasis: *
Doesn’t affect children

High-Yield Context (from Dermatology Summary - Psoriasis, p. 5, and lecture slides p.141, p.145):

From Dermatology Lecture Slides - Psoriasis (Introduction, p. 141):
• Can occur at any age (median age is 28), with 2 peaks of onset:
Early (16-22) and Late (57-60).

(The "Early (16-22)" onset implies it can affect older children/adolescents. Guttate psoriasis is also common in children/young adults.)

From Dermatology Summary - Psoriasis (Types - Guttate, p. 6):
○
Guttate → widespread small plaques scattered on the trunk and limbs, preceded by sore throat with GBS *(often affects children and young adults)***From Dermatology Lecture Slides - Guttate Psoriasis (p. 145):**
•
Adolescents are mostly affected...

(Psoriasis certainly can affect children. Guttate psoriasis is common in children and adolescents. Napkin psoriasis is a specific form in infants. Therefore, the statement "Doesn’t affect children" is wrong.)

Question 24:
All will exacerbate psoriasis EXCEPT:
a. Hypocalcemia
b. Anti-malarial
c. Infections
d. Hormonal changes
e. Macrolides
Answer: E

High-Yield Context (from Dermatology Summary - Psoriasis, p. 5 & 6, and lecture slides p.142, p.149):

From Dermatology Summary - Psoriasis (Pathophysiology, p. 5):
○ Genetic predisposition +
Precipitating factors “Stress, infection, local trauma Alcohol, drugs, childbirth.”

From Dermatology Summary - Psoriasis (Types - Erythrodermic, p. 6):
○ Erythrodermic → ... triggers → steroid withdrawal,
infection, alcohol, **antimalarials**, lithium and **low calcium**

From Dermatology Lecture Slides - Psoriasis (Environmental triggers, p. 142):

- Infections (streptococcal, HIV)
- Skin trauma
- Stress
- **Drugs e.g. Beta-blockers, anti-malarial, lithium**, systemic steroid withdrawal
- Smoking
- Obesity
- Hormonal factors (pregnancy)
- **Hypocalcemia.**

From Dermatology Lecture Slides - Psoriasis (Management ladder, Systemic medications, p.149):*(This section lists medications used to treat psoriasis, not exacerbate it. Macrolides are antibiotics, and while infections can exacerbate psoriasis, macrolides themselves are not typically listed as exacerbating factors for psoriasis. Some antibiotics like tetracyclines are actually used in some inflammatory conditions, though not first-line for psoriasis typically.)*

(Hypocalcemia, antimalarials, infections are known exacerbating factors. Hormonal changes (e.g., pregnancy, childbirth) can also influence psoriasis. Macrolides are not typically listed as major exacerbating drugs for psoriasis; in fact, some infections they treat could be triggers, so treating the infection might improve psoriasis.)

Question 25:
All are exacerbations of psoriasis except:
Hypercalcemia

High-Yield Context (from Dermatology Summary - Psoriasis, p. 6, and lecture slides p.142):

From Dermatology Summary - Psoriasis (Types - Erythrodermic, p. 6):
○ Erythrodermic → ... triggers → steroid withdrawal, infection, alcohol, antimalarials, lithium and
low calcium

From Dermatology Lecture Slides - Psoriasis (Environmental triggers, p. 142):

- **Hypocalcemia.**

(Hypocalcemia is listed as an exacerbating factor. Hypercalcemia is not; in fact, vitamin D analogues which affect calcium metabolism are used to treat psoriasis.)

Question 26:
Wrong about psoriasis:
Oral steroids are usually used to manage flare ups

High-Yield Context (from Dermatology Summary - Psoriasis, p. 6, and lecture slides p.142, p.146, p.150):

From Dermatology Summary - Psoriasis (Types - Erythrodermic, p. 6):
○ Erythrodermic → ... triggers →
steroid withdrawal...

From Dermatology Summary - Psoriasis (Management, p. 6):
●
Systemic corticosteroids should not be used to treat psoriasis

From Dermatology Lecture Slides - Psoriasis (Environmental triggers, p. 142):

- Drugs e.g. Beta-blockers, anti-malarial, lithium, **systemic steroid withdrawal**

From Dermatology Lecture Slides - Pustular Psoriasis (Generalized form, p.146):
• It may be precipitated by the patient taking
systemic steroids, or using potent topical steroids.

**From Dermatology Lecture Slides - Topical Treatment (p.150):\\ Systemic corticosteroids should not be used to treat psoriasis **

(Systemic steroids are generally contraindicated in psoriasis due to the risk of rebound pustular psoriasis or erythroderma upon withdrawal. While they might be used in very specific, severe, short-term situations under expert care, the statement "usually used to manage flare ups" is wrong. They can actually cause severe flare-ups (e.g., Von Zumbusch pustular psoriasis or erythroderma) upon withdrawal.)

Page 5 (Test Bank - Psoriasis cont.)

Question 27:
Wrong about psoriasis? *
Usually inherited as autosomal recessive

High-Yield Context (from Dermatology Summary - Psoriasis, p. 5, and lecture slides p.141):

From Dermatology Summary - Psoriasis (Pathophysiology, p. 5):
○
Genetic predisposition + Precipitating factors...

From Dermatology Lecture Slides - Psoriasis (Pathophysiology - Genetic predisposition, p. 141):

1. **Genetic predisposition (PSORS1 loci, specific HLAs);**
    - Family history of psoriasis. 16% of the children will have psoriasis if a single parent is affected and 50% if both parents are affected.

- In monozygotic twins, if one twin has the disease, the other one has a 70% chance of being affected. In dizygotic twins, there is only a 20% chance.

  (Psoriasis has a strong genetic component, but it's multifactorial and polygenic, not simple Mendelian autosomal recessive. The inheritance pattern is complex.)

Question 28:
A 30-year-old patient presents with itchy vesicles on extensor areas. Which of the following diseases most likely fits with the description of the eruption?
a. Shingles
b. Psoriasis
c. Dermatitis herpatiformes
d. Chicken pox
e. Bullous pemphigoid
Answer: B
(The answer key indicates B for Psoriasis. However, "itchy vesicles on extensor areas" is the classic description for Dermatitis Herpetiformis. Psoriasis typically presents as plaques with scale, not primarily vesicles, though it can be itchy and on extensors. Shingles is dermatomal and vesicular but typically painful. Chicken pox is generalized vesicles. Bullous pemphigoid is tense bullae. Given the options, C is a much better fit than B for "itchy vesicles on extensor areas". This might be an error in the question or answer key for the Test Bank).

*Let's check the Psoriasis summary for vesicular presentations:***From Dermatology Summary - Psoriasis (Types, p. 6):**
(Pustular psoriasis involves pustules, not typically vesicles. No direct mention of primary vesicular psoriasis in this summary. Dermatitis Herpetiformis is discussed later in the lecture context as intensely itchy vesicles on extensors.)

Given the provided answer is B (Psoriasis), it is possible the question is misleading or refers to a very atypical presentation or a secondary change like excoriation leading to vesiculation, which is not primary. For the sake of this exercise, I will assume the Test Bank answer is what we work with, but flag it as problematic based on classic presentations.

High-Yield Context (from Dermatology Summary - Psoriasis, p. 5-6):
●
Clinical appearance
○ Plaques → well-defined raised areas...
○ Scaling
○ Erythema or redness
○
Pustules → esp. In palmo-plantar pustulosis
●
Clinical presentation
○ Lesions improve in the sun, and are
mildly itchy
○ Distribution →
elbows, knees and scalp (extensor areas)

(While psoriasis is on extensors and can be itchy, primary vesicles are not characteristic. Pustules are a feature of pustular psoriasis.)

Question 29:
All about psoriasis ttt is true except:
a. Imuran
b. Cyclosporine
c. PUVA
d. Methotrexate
e. Systemic retinoids
Answer: A
(Imuran (Azathioprine) is listed as a 2nd line systemic agent in the summary).(The arrow points from Imuran to "Imuran = azathioprine which is considered a second line agent in treatment of psoriasis". This suggests the statement "a. Imuran" is true, and thus the question "All about psoriasis ttt is true except:" with answer A implies that the statement about Imuran as a treatment is the "except" i.e., false, or less true than others. Cyclosporine, PUVA, Methotrexate, Systemic retinoids (Acitretin) are all well-established treatments.)

High-Yield Context (from Dermatology Summary - Psoriasis, Management, p. 6 and Lecture Slides pp. 149-153):

From Dermatology Summary - Psoriasis (Systemic treatment, p. 7):
○ first-line systemic →
Acitretin (systemic retinoid), **Cyclosporin A, Methotrexate**
○ 2nd line → Mycophenolate mofetil (MMF), hydroxyurea,
azathioprine (Imuran)

From Dermatology Summary - Psoriasis (Ultraviolet treatment, p. 6):
○ Types
■ photochemotherapy “long wavelength”→
ultraviolet A plus psoralen (PUVA)

(All listed options are treatments for psoriasis according to the summary and lecture context. Azathioprine (Imuran) is a second-line systemic agent. Cyclosporine, Methotrexate, and Systemic Retinoids (Acitretin) are first-line systemic. PUVA is a form of photochemotherapy. Perhaps "true" here relates to being a first-line or very common treatment, making a second-line option the "except". This is speculative based on the provided answer.)

Question 30:
Not used in the systemic treatment of psoriasis:
a. Methotrexate
b. Isotretinoin
c. Fumaric acid
d. Cyclosporine
Answer: B
(Isotretinoin is a retinoid primarily used for acne. Acitretin is the systemic retinoid used for psoriasis.)

High-Yield Context (from Dermatology Summary - Psoriasis, Management, p. 7, and Lecture Slides pp. 152-153):

From Dermatology Summary - Psoriasis (Systemic treatment, p. 7):
○ first-line systemic →
Acitretin (vitamin A derivative - systemic retinoid), **Cyclosporin A, Methotrexate***(Fumaric acid esters are used for psoriasis in some regions, though not explicitly in this summary's first/second line list. Isotretinoin is generally for acne, while Acitretin is the systemic retinoid for psoriasis.)*

Question 31:
All of the following are considered systemic treatment options for psoriasis except:
a. TNF-a blockers
b. Acitretin
c. Cyclosporin
d. Methotrexate
e. Vitamin D analogue
Answer: E
(Vitamin D analogues are topical treatments for psoriasis, not systemic.)

High-Yield Context (from Dermatology Summary - Psoriasis, Management, pp. 6-7, and Lecture Slides pp. 150, 152-154):

From Dermatology Summary - Psoriasis (Topical treatment, p. 6):
○
Calcipotriol and tacalcitol, vitamin D analogues, are calmodulin inhibitors → for mild or moderate plaque psoriasis.

From Dermatology Summary - Psoriasis (Systemic treatment, p. 7):
○ first-line systemic →
Acitretin, **Cyclosporin A, Methotrexate**
○ Biological therapy
■ Agents
●
anti-TNF→ Etanercept, Infliximab, Adalimumab

(Vitamin D analogues are clearly listed under topical treatment in the summary.)

Question 32:
Not used in treatment of psoriasis:

a. Antimalarial
b. Cyclosporine A
c. Methotrexate
d. Systemic retinoid
e. Fumaric acid esters
Answer: A
(Antimalarials are known to exacerbate psoriasis.)

High-Yield Context (from Dermatology Summary - Psoriasis, p. 6, and Lecture Slides p.142):

From Dermatology Summary - Psoriasis (Types - Erythrodermic, p. 6):
○ Erythrodermic → ... triggers → steroid withdrawal, infection, alcohol,
antimalarials, lithium and low calcium

From Dermatology Lecture Slides - Psoriasis (Environmental triggers, p. 142):

- Drugs e.g. Beta-blockers, **anti-malarial**, lithium, systemic steroid withdrawal

(Antimalarials are listed as triggers/exacerbating factors for psoriasis, not treatments.)

Question 33:
Characteristic of nail in psoriasis except:
Clubbing

High-Yield Context (from Dermatology Summary - Psoriasis, p. 5, and Lecture Slides p.147):

From Dermatology Summary - Psoriasis (Psoriatic nail dystrophy, p. 5):
○
onycholysis, subungual hyperkeratosis, pitting, Beau’s lines (transverse lines, groove), splinter hemorrhages
(longitudinal black lines)

From Dermatology Lecture Slides - Psoriatic Arthritis (Nail changes, p. 147):
Lists: Onycholysis, Subungual hyperkeratosis, Pitting, Beau's lines, Splinter hemorrhages.

(Clubbing is not a feature of psoriatic nail disease. It's associated with other conditions like lung disease, cardiac disease, IBD etc.)

Eczema (dermatitis)

Question 34:
Histology of spongiosis & parakeratosis with normal granular layer suggests:
a. eczema
b. psoriasis
c. lichen planus
Answer: A

High-Yield Context (from Dermatology Summary - Eczema (Dermatitis) (Pathology, p. 155 of lecture slides), Psoriasis (p. 5, and lecture slides p. 142)):

From Dermatology Lecture Slides - Eczema (Pathology, p. 155):
• There is edema in the epidermis leading to
spongiosis (separation of keratinocytes) and vesicle formation.
• The epidermis is hyperkeratotic (thickened) with dilated blood vessels and an inflammatory (Lymphocytes & eosinophil) cell infiltrate in the dermis.

(While the main summary on eczema (p.8) doesn't detail histology, the lecture context page on pathology (p.155) for eczema mentions spongiosis. Parakeratosis can be seen in chronic eczema, but a normal granular layer with spongiosis is more characteristic of acute/subacute eczema. Psoriasis has parakeratosis but typically acanthosis and reduced or absent granular layer in areas of parakeratosis. Lichen planus has hyperkeratosis, hypergranulosis, and liquefaction degeneration of basal layer.)

From Dermatology Summary - Psoriasis (Pathophysiology, p. 5):
○ Hyperkeratosis ...,
parakeratosis ”keratocytes retain their nuclei”...

(Psoriasis typically has parakeratosis and often a reduced/absent granular layer beneath it.)

(Spongiosis is the hallmark of acute eczema/dermatitis. Parakeratosis can occur in chronic eczema. A "normal granular layer" would be more typical of acute/subacute eczema rather than chronic where it might be hyperplastic or altered. Psoriasis characteristically has parakeratosis but often a thinned or absent granular layer directly under the parakeratotic scale.)

Question 35:
Adult atopic dermatitis may be associated with the following except:
a. Pruritus ani
b. Pruritus vulvae photodermatitis
c. Asthma
d. Hair fall
Answer: D

High-Yield Context (from Dermatology Summary - Eczema (Dermatitis), Atopic dermatitis, pp. 8, 157-158 of lecture slides):

From Dermatology Lecture Slides - Atopic Dermatitis (p. 157):
• Often associated with:
- An elevated serum Immunoglobulin E (IgE) level.
- Personal or family history of atopy; A genetically mediated predisposition to an excessive (IgE) reaction encompassing a triad of
Eczema, Asthma & Allergic rhinitis.

(Asthma is a well-known association (part of the atopic triad). Pruritus ani/vulvae can be manifestations of eczema in those areas. Photodermatitis can occur, though "pruritus vulvae photodermatitis" is very specific. Hair fall (alopecia) is not a primary direct association of adult AD, though severe scalp eczema could potentially affect hair, or alopecia areata (another autoimmune condition) can co-exist.)

Question 36:
One false about infantile atopic dermatitis:
a. Increase incidence of contact eczema
b. Present at birth
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Atopic Dermatitis, p. 158):
• Typically presents in
infancy or early childhood, initially with facial and subsequently flexural limb involvement.

(While it presents in infancy, it's not usually "present at birth" (congenital). It typically develops in the first few months of life. Increased susceptibility to contact eczema can occur due to impaired skin barrier.)

Question 4:
True about atopic dermatitis:
a. T helper cells have the major role in pathophysiology
b. Most common site in children is extensor areas
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Atopic Dermatitis, pp. 157-158):

From p. 157 (Pathophysiology):
• Imbalance between
T helper 2 (Two high in aTopic) vs Th1 and Th 17 (high in psoriasis).

(This implies a major role for T helper cells.)

From p. 158 (Distribution by Age):
•
Infant (birth-2 years): Face (cheeks), scalp, body, arms, legs; extensors
• Childhood (2 years-puberty): Face (cheeks); Flexural extremities

(Extensor areas are indeed common sites in infants/young children.)(The question asks for "True". Statement A about T

helper cells is true based on the pathophysiology. Statement B about extensor areas in children is also true for infants. If "children" here encompasses infants and young children, then B is also true. There might be a more specific "most common site" difference if "children" excludes infants. However, given the options, A is a fundamental pathophysiological truth.)

Question 37:
Wrong about atopic dermatitis: *
a. Begin before 2 months
b. Itching
c. Steroids
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Atopic Dermatitis, p. 158):
• Typically presents in
infancy or early childhood...

(Infancy is generally considered the first year of life. AD often starts between 2-6 months. "Begin before 2 months" is plausible for many cases. Itching is the hallmark. Steroids are a mainstay of treatment.)If the answer is A, it implies that it is wrong to say it begins before 2 months, meaning it typically begins after 2 months. This is generally true, often cited as 2-6 months of age for onset.

Question 38:
Seborrheic dermatitis, all are true except:
a. Occurs in children and adults
b. Most common in the extensors
c. Occurs in age less than 3 months
d. Scalp cradle cap in babies
e. Post-auricular and nasolabial folds are common sites
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Seborrheic dermatitis, p. 160-161):

From p. 160:
• Chronic, relapsing, and usually mild form of dermatitis that occurs in
infants and in adults.
• Biphasic incidence with a male predominance in adults:
1- Infants between the ages of
2 weeks and 12 months
2- Adolescence and adulthood

From p. 161 (Clinical manifestations In infants):
•
Scalp (vertex and frontal areas; the **'cradle-cap' area**)
•
Face (forehead, eyebrows, eyelids, **nasolabial folds**, temple)
• Diaper area
•
Retroauricular folds
• Neck
• Axillae

From p. 161 (Clinical manifestations in adults):
•
Scalp: The earliest sign is dandruff...
•
Face: Scaling & erythema of forehead, medial portion of eyebrows, eyelids, **nasolabial folds**, lateral part of the nose, and **retro-auricular** region.
•
Trunk: Papules, greasy scales.
•
Flexural areas: Erythema, greasy scaling, and secondary infection.

(Seborrheic dermatitis classically affects sebaceous gland-rich areas like scalp, face (nasolabial folds, eyebrows, retroauricular), and flexures, not typically extensors.)

Question 39:
Atopic dermatitis vs seborrhea dermatitis all true except:
a. Seborrheic start at earlier age
b. Seborrheic has worse prognosis
c. Atopic severe pruritis
d. Atopic is associated with +ve family Hx
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Atopic Dermatitis pp. 157-158, Seborrheic Dermatitis pp. 160-161):

- **Age of Onset:**
  - AD: Often 2-6 months. (p. 158)
  - SD (infantile): 2 weeks - 12 months. (p. 160) So SD can start earlier. (Statement a = true)
- **Prognosis:**
  - AD: 90% remit by puberty, but can be chronic, especially with poor prognostic factors. (p. 158)
  - SD: Infantile SD usually resolves. Adult SD is chronic and relapsing but generally mild. (p. 160) "Worse prognosis" for SD is debatable, especially infantile SD. Adult SD is chronic, but AD can also be chronic and more debilitating. (Statement b = likely false or at least questionable).
- **Pruritus:**
  - AD: "intensely itchy". (p. 158) (Statement c = true)
- **Family Hx:**
  - AD: "Personal or family history of atopy". (p. 157) (Statement d = true)

(Seborrheic dermatitis in infants often resolves, while atopic dermatitis can be more chronic. Saying SD has a worse prognosis is generally not true, especially for infantile SD. Adult SD is chronic but often manageable.)

Question 40:
All about seborrheic dermatitis are true except:
a. May occur earlier than atopic dermatitis
b. Self-limiting
c. Itching is mild
d. Chronic
e. Prognosis poorer than atopic dermatitis
Answer: E

High-Yield Context (from Dermatology Lecture Slides - Seborrheic dermatitis, pp. 160-161):

- **Age of Onset:** Infantile SD: 2 weeks - 12 months. (p. 160) Can be earlier than typical AD onset. (Statement a = true)
- **Course:**
  - Infantile SD is often **self-limiting.** (General knowledge, not explicit in summary but fits the usual description) (Statement b = true for infantile)
  - Adult SD is **chronic, relapsing**. (p. 160) (Statement d = true for adult)
- **Itching:** Scalp SD "accompanied later by itching". Face SD "Scaling & erythema". Generally considered mildly itchy compared to AD. (Statement c = generally true)
- **Prognosis:** (Statement e = same as above, likely false)

(Infantile seborrheic dermatitis is often self-limiting. Adult seborrheic dermatitis is chronic. Prognosis poorer than AD is generally false; AD can be more severe and persistent with significant QoL impact.)

Question 41:
Contact dermatitis is clinical manifestation of:
a. Cytotoxic reaction
b. Arthus reaction

c. Cell mediated reaction
d. Anaphylactic reaction
answer: C

High-Yield Context (from Dermatology Lecture Slides - Eczema (Exogenous eczema - Contact dermatitis), p. 164, and Drug Rashes classification p. 26):

From Dermatology Lecture Slides - Allergic contact dermatitis (p. 164):
• This is due to the development of
delayed hypersensitivity (type 4 allergy) to a specific chemical (sensitizer or allergen).

(Type IV hypersensitivity is a cell-mediated reaction.)

From Dermatology Lecture Slides - Drug Rashes Pathogenetic Classification (p. 26):
•
Type IV reactions: (m.c)
Delayed hypersensitivity reactions, cause generalized exanthems, phototoxic rashes, and severe drug reactions such as toxic epidermal necrolysis (TEN).

(This reinforces that Type IV reactions are delayed and common in drug-induced skin issues, and contact dermatitis is a prime example of Type IV reaction.)

Page 42 (Test Bank - Eczema (dermatitis) cont.)

Question 1:
Pathognomonic test used in Dx of contact dermatitis:
a. intradermal tests
b. patch test
c. prick test
d. skin Bx
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Eczema (Contact Dermatitis), p. 168):

Patch testing:
•
Patch testing is used to determine the substances that cause allergic contact dermatitis.
- The concentration used is critical to ensure a low false negative/positive rate.
- The optimum concentration and best vehicle have been ascertained for the most common allergens.
- The standard series contains a group of tests that encompasses the most common allergens encountered.
- Done by dermatologists in outpatient settings.
- The test patches are usually placed on the upper back (sites marked) and left in place for

48 h, then removed and any positive reactions are noted.
- A further examination is carried out at

96 h to detect any late reactions.
- Patients need to visit the unit

three times in 1week, be off systemic immunosuppressants and have an area of clear skin (usually the upper back) on which to perform the tests.

(Patch testing is specifically designed to identify allergens causing allergic contact dermatitis, a Type IV hypersensitivity.)

Question 43:
A case about a 5-year-old with features of atopic dermatitis, what do you do?
a. CBC
b. chest x-ray
c. renal function
d. lgG
e. lgE
Answer: E

High-Yield Context (from Dermatology Lecture Slides - Eczema (Atopic Dermatitis), p. 157, Investigations p. 163):

From Dermatology Lecture Slides - Atopic Dermatitis (p. 157):
- Often associated with:

- An elevated serum
Immunoglobulin E (IgE) level.
- Personal or family history of atopy...

From Dermatology Lecture Slides - Investigations of eczema (p. 163):
4- Routine blood tests are not necessary; however, an eosinophilia and raised
immunoglobulin E (IgE) level may be seen.
5- RAST (radioallergosorbent testing) looks for specific IgE levels against suspected allergens...
6- Skin prick testing may also be used to determine any specific allergies to aeroallergens or foods.

(Elevated IgE is a common finding and association in Atopic Dermatitis, reflecting its atopic nature.)

Question 44:
Lymphocytes from pt with atopic dermatitis bear greater than normal amounts of:
a. IgG
b. IgA
c. IgE
d. IgM
e. IgD
Answer: C

High-Yield Context (from Dermatology Lecture Slides - Eczema (Atopic Dermatitis), p. 157):

- Often associated with:
  - An elevated serum
  Immunoglobulin E (IgE) level.
  - Personal or family history of atopy; A genetically mediated predisposition to an excessive
  (IgE) reaction encompassing a triad of Eczema, Asthma & Allergic rhinitis.
  • Atopy is type 1 hypersensitivity reaction. (Primarily IgE mediated)
  • Imbalance between
  T helper 2 (Two high in aTopic) vs Th1 and Th 17 (high in psoriasis).

  (While the question asks about IgE on lymphocytes, the key association is elevated serum IgE and the overall IgE-mediated (Type 1) hypersensitivity component in atopy. T-helper 2 lymphocytes promote IgE production by B-cells/plasma cells.)

Question 45:
Eruption of an erythematous lesion on the face particularly the nasolabial folds, and eyebrows, scalp, what's the diagnosis?
a. Seborrheic dermatitis
b. Eczema
c. Atopic dermatitis
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Seborrheic dermatitis, p. 161):

Clinical manifestations in adults:

•
Scalp: The earliest sign is dandruff, accompanied later by itching & inflammation, and retro-auricular fissuring.
•
Face: Scaling & erythema of forehead, medial portion of **eyebrows**, eyelids, **nasolabial folds**, lateral part of the nose, and retro-auricular region.
- Trunk: Papules, greasy scales.
- Flexural areas: Erythema, greasy scaling, and secondary infection.

Clinical manifestations In infants:

•
Scalp (vertex and frontal areas; the 'cradle-cap' area)
•
Face (forehead, **eyebrows**, eyelids, **nasolabial folds**, temple)

(These are classic sites for Seborrheic Dermatitis due to high sebaceous gland activity.)

Question 46:
All of the following statements are true except:

a. Infant’s atopic eczema mostly affect the flexural sites as popliteal fossa and the wrists
b. Juvenile plantar dermatosis is caused mainly due to the socks and shoes that are impermeable
c. Lichenification may be seen in chronic eczema
d. Seborrheic eczema is linked to Malassezia
e. Eczema may be included by both external and internal factors
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Atopic Dermatitis pp. 158-159, Seborrheic Dermatitis p. 160, Eczema Clinical Features p. 156, General classification p. 155):

- **a. Infant’s atopic eczema mostly affect the flexural sites...**
  - **From p. 158 (Distribution of AD by Age):** Infant (birth-2 years): Face (cheeks), scalp, body, arms, legs; **extensors**. Childhood (2 years-puberty): Face (cheeks); **Flexural extremities**.
  - *(Statement A is FALSE. In infants, AD typically affects extensors and the face, flexures are more common in older children/adults.)*
- **b. Juvenile plantar dermatosis...**
  - **From p. 159 (Atopic Dermatitis associated features):** Juvenile plantar dermatosis: A variant of atopic eczema in which there is dry cracked skin on the forefoot in children. *(Cause due to socks/shoes impermeability is classic, though not explicit in this brief mention.)* (Statement B is generally considered true).
- **c. Lichenification may be seen in chronic eczema**
  - **From p. 156 (Eczema secondary changes):** Eczema is characteristically itchy and subsequent scratching may also modify the clinical appearance leading to: ... **Lichenification**. (Statement C is TRUE).
- **d. Seborrheic eczema is linked to Malassezia**
  - **From p. 160 (Seborrheic dermatitis - Pathogenesis):** May involve the lipid-dependent fungus **Malassezia furfur** which thrives on sebum thus causing dermatitis. (Statement D is TRUE).
- **e. Eczema may be included by both external and internal factors**
  - **From p. 155 (Classification of eczema - According to the cause):** 1. Endogenous eczema: An internal cause... 2. Exogenous eczema: An external cause... 3. Mixed eczema: Both endogenous & exogenous processes... (Statement E is TRUE).

(Statement A is definitively false based on the provided summary's distribution of AD by age.)

Question 47:
Which one cause allergic contact dermatitis: ****
a. Cobalt
b. Nickel
c. Cement
d. Rubber
e. Silver
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Contact dermatitis, common allergens list, p. 165):

Box 4.1 Common contact allergens

•
Nickel/cobalt (jewellery, clothing, wristwatch, scissors and cooking utensils).
• Potassium dichromate (chemical used to tan leather; Figure 4.17), chrome, myroxylon pereirae (balsam of Peru, fragrance; Figure 4.20).
• Formaldehyde, paraben, quaternium, methylchloroisothia zolinone, methylisothiazolinone (MCI/MI) (preservative), hair dyes, temporary tattoos and textiles; Figure 4.22).
• Ethylenediamine (adhesives and medications).
• Thiurams (cement and leather).
• Mercaptobenzothiazole, carbamates (rubber gloves and shoes).
• Neomycin, benzocaine (medications; Figure 4.21).
• Lanolin (wool alcohol, emollients and medicated ointments).

(Nickel is a very common cause of allergic contact dermatitis, often from jewelry.)

Question 48:
Commonest site of contact dermatitis produced by nail varnish is:
a. Neck
b. Nail folds
c. Nail
d. Back of hands
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Contact dermatitis clinical features, ectopic spread, p. 166):

(The summary doesn't specifically mention nail varnish dermatitis sites. However, nail varnish allergen can be transferred by fingers to other sites, particularly eyelids and neck – an "ectopic" pattern of dermatitis.)General knowledge: Nail varnish often causes dermatitis on the eyelids, face, and neck due to transfer from the fingers, rather than directly on the nails/nail folds themselves for the allergic component.

Question 49:
Commonest site of contact dermatitis produced by clothes:
a. Body flexures
b. Scalp
c. Arms
d. Legs
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Contact dermatitis clinical features, p. 166):

*(The summary page for common allergens (p.165) lists "hair dyes, temporary tattoos and **textiles**" under Formaldehyde, paraben, quaternium section as potential allergens. Body flexures (axillae, groin) are common sites for textile dermatitis due to friction, occlusion, and sweating concentrating allergens from dyes, finishes, or resins in clothing.)*

Question 50:
Unilateral hand eczema, best next step:
a. Scrap and do KOH
b. Potent topical steroids

(OCR for options ends here. The likely answer would be 'a. Scrap and do KOH' to rule out tinea manuum, especially if it's unilateral, before committing to steroids which can worsen fungal infections (tinea incognito).)

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections - Investigating, p. 105, and Tinea pedis/manuum, p.108):

From Fungal Infections - Investigating fungal infections (p. 105):

- Taking

skin scrapings, nail clippings, scalp brushings, and skin biopsies for mycological analyses.

From Fungal Infections - Tinea pedis or athlete's foot (p.108):

- The

hands may be similarly affected (tinea manuum).

- The condition needs to be differentiated from psoriasis and eczema ... and therefore

scrapings for mycology can be helpful.

(Unilateral hand eczema ("one hand, two feet" syndrome or tinea manuum) should always raise suspicion for a fungal infection, making KOH scrapings essential.)

Page 8 (Test Bank - Eczema (dermatitis) cont.)

Question 51 (continued from Page 7, question 9):
Unilateral hand eczema, best next step:
a. Scrap and do KOH
b. Potent topical steroids
c. Make him wear gloves
d. Give emollients
Answer: A

High-Yield Context (already covered under Page 7, Question 9).*(Scraping for KOH is crucial to rule out tinea manuum in unilateral hand dermatitis before considering potent topical steroids.)*

Question 52:
Which one commonly cause severe even bullous contact dermatitis:
a. Cement
b. Primula
c. Cobalt
d. Leather
e. Rubber
Answer: B
(Primula obconica is a plant well-known to cause severe, often bullous, allergic contact dermatitis.)

High-Yield Context (from Dermatology Lecture Slides - Contact dermatitis, common allergens, p.165):

(The summary lists common allergens but doesn't grade them by severity or bullous potential. However, specific plant allergens like those in Primula or Poison Ivy (not listed but analogous) are notorious for severe reactions. Cement can cause irritant and allergic (chromium) dermatitis. Cobalt is an allergen. Leather (tanning agents like dichromate) and rubber (accelerators) are also common allergens.)This question tests knowledge beyond the direct text of the summary provided.

Question 53:
Which one of the following agents cause pigmented contact dermatitis:
a. Lipsticks
b. Nail varnish
c. Deodorants
d. Perfumes
e. Hair dyes
Answer: D
(Some fragrances/perfumes, especially those containing certain plant extracts or essential oils (like bergamot oil causing Berloque dermatitis - a phototoxic reaction leading to pigmentation), can cause pigmented contact dermatitis or photocontact dermatitis with subsequent pigmentation.)

High-Yield Context (from Dermatology Lecture Slides - Contact dermatitis, common allergens, p.165, and Drug Rashes - Pigmentation, p.11):

(The summary lists fragrances (myroxylon pereirae (balsam of Peru)) as common contact allergens. Hair dyes (PPD) are also listed. While not explicitly "pigmented contact dermatitis", prolonged inflammation from any contact dermatitis can lead to post-inflammatory hyperpigmentation. Certain specific agents in perfumes can cause phototoxic reactions leading to prominent pigmentation (e.g., Berloque dermatitis).)

Question 54:
Wrong about eczema?
Contact dermatitis develops 12 hours from exposure

High-Yield Context (from Dermatology Lecture Slides - Eczema (Allergic Contact Dermatitis), p. 165, and Irritant Contact Dermatitis, p.164):

From Allergic contact dermatitis (p. 165):

-

48–96 h between contact and the development of changes in the skin.From Irritant contact dermatitis (p. 164):

- Dermatitis occurs

soon after exposure and the severity varies with the quantity, concentration, and length of exposure to the substance concerned. *(Soon could be minutes to hours for strong irritants).*

(Allergic contact dermatitis typically develops 48-96 hours after exposure in a sensitized individual. Strong irritant contact dermatitis can develop much faster, within hours. So "12 hours" is plausible for some irritant reactions but too quick for a typical allergic contact dermatitis primary reaction. If it refers to allergic, then it's wrong. If it refers to any contact dermatitis, it could be true for strong irritants.)The question is general "contact dermatitis". If it's referring to allergic contact dermatitis, then 12 hours is too soon. If it's irritant, it could be plausible. Given the options in typical MCQs, this statement is likely considered wrong because allergic contact dermatitis (the more specific immunological reaction) has a longer delay.

Question 55:
Wrong statement:
Pityriasis alba appears depigmented on wood's light

High-Yield Context (from Dermatology Lecture Slides - Atopic Dermatitis associated features, p. 159, and Wood's Lamp principles, p. 20):

From Atopic Dermatitis associated features (p. 159):

•

Pityriasis alba: variant of atopic eczema in which pale patches of **hypopigmentation** develop on the face of children

From Wood's Lamp Examination (p. 20 - Chart):
Disorder/infection/colonization: Pigmentary disorders
Vitiligo: Chalk-white to dull bluish-white (Fluorescence of dermal collagen and elastin from decreased or absence of melanin within the epidermis)
Ash leaf spots: Enhancement of hypopigmentation
Hyperpigmentation due to an increase in:

1. epidermal melanin: Enhancement of brown color
2. dermal melanin: Difference in color of lesional vs nonlesional skin becomes less obvious

(Pityriasis alba is characterized by hypopigmentation, not depigmentation. Depigmentation (complete loss of melanin, as in vitiligo) fluoresces brightly under Wood's light. Hypopigmentation (reduced melanin) may be accentuated but doesn't typically show the same intense fluorescence as true depigmentation. The statement says "appears depigmented", which is incorrect for pityriasis alba – it is hypopigmented.)

Question 56:
Thickening and hardening of the skin, with exaggeration of its normal markings:
a. Lichenification
b. Spongiosis
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Eczema secondary changes, p. 156):

Eczema is characteristically itchy and subsequent scratching may also modify the clinical appearance leading to:

- Excoriation marks
- Loss of skin surface
- Secondary infection
- Exudates
-

Lichenification. (hyperkeratosis and acanthosis from chronic rubbing, leading to thickened skin with accentuated skin lines)

From Clinical features - Chronic Eczema (p. 156):

- **Lichenification** (hyperpigmentation and **thickening of the skin where surface markings become more prominent**) i.e. the skin looks rather like the bark of a tree.

(This is the definition of lichenification.)

Question 57:
All can cause blisters except:
a. Chronic eczema
b. Impetigo
c. Pemphigoid
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Eczema clinical features p.156, Bullous Diseases intro p.13, Bacterial Infections (Impetigo) p.116):

- **Chronic eczema (p. 156):** Leads to increased scaling, xerosis, lichenification. Acute eczema has vesicles/bullae. Chronic eczema is typically dry and lichenified, not primarily blistering.
- **Impetigo (p. 116):** "develops rapidly into clusters of pustules and **vesicles** which break down into the classic golden crusts." Bullous impetigo is also a form. So impetigo *can* cause blisters/vesicles.
- **Pemphigoid (p. 13, and pp. 70-72):** Bullous pemphigoid is a classic autoimmune blistering disease characterized by tense bullae.

(Chronic eczema is characterized by dryness, scaling, and lichenification. Acute eczema has vesicles. Therefore, chronic eczema is the least likely to be the primary cause of blisters among the options.)

Pruritus / Scabies

Question 58:
The pruritus of biliary obstruction can probably be most directly related to:
a. Whole (crude) bile
b. Bile salts
c. Bile acids
d. Conjugated bilirubin
e. Unconjugated bilirubin
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Pruritus with normal skin (Metabolic), p. 9, and The Skin and Systemic Disease (Liver disease), p.19):

From Pruritus with normal skin (Metabolic, p. 9):
● Metabolic – hepatic failure,
biliary obstruction and chronic renal failure.

From The Skin and Systemic Disease (Liver disease and the skin, p. 19):
○
Obstructive → Jaundice, Pruritus. deposition of bile salts "Tx; cholestyramine"

(The summary specifically links pruritus in biliary obstruction to the deposition of bile salts.)

Question 59:
After initial exposure to & infestation with sarcoptes scabies hominis the pruritis follows:
a. Immediately
b. In 1-2 days
c. In about one week
d. Ill about 2-4 week
(Likely typo for "In about 2-4 week")
e. In about 3 months
Answer: D

High-Yield Context (from Dermatology Lecture Slides - Scabies and head lice (Infestations by parasites - Scabies), p. 28 and p.88):

From Lecture Slides - Scabies (Transmission, p.88):
• The first symptoms of itching occur
2 weeks later when the immune system reacts to the proteins in the mites, eggs, and feces in the skin.

From Summary - Scabies and head lice (Infestations by parasites - Scabies, p.28):
○ Sx after
2 weeks (immune Rx to proteins in the mites)...

(The delay is due to the time taken to develop sensitization to the mite antigens. 2-4 weeks is a typical timeframe for primary infestation.)

Question 60:
In humans scabies the best yield of positive scrapings is form:
a. Papules
b. Vesicles
c. Burrows
d. Excuriation
e. Crusts
Answer: C

High-Yield Context (from Dermatology Lecture Slides - Scabies (Diagnosis), p. 89):

Physical Examination:

1. **Burrows:**
    - Clinically, burrows can be seen, especially in the finger-web spaces and on the genitals

- Burrows are linear palpable ridges on the skin with a black speck indicating the position of the mite, which can be teased out of its burrow using a sterile needle and mounted onto a microscope slide.

(Burrows are where the female mite lives and lays eggs, making them the highest yield site for finding mites or their products.)

Question 61:
Wrong about scabies of infants:
a. Treated with permethrin 5%
b. May occur in back and face
c. No family history of itching
d. Involves palms and soles
e. Caused by scapii sarcoptes hominis
answer: C

High-Yield Context (from Dermatology Lecture Slides - Scabies (Scabies in children/infant), p.89, and Management p.91):

From Scabies in children (p.89):
- Babies and young children infested with scabies characteristically present with erythematous cutaneous papules and nodules in the
axillae and on the soles of the feet.
- Classic burrows are rarely seen in this age group.

(Distribution in infants often includes the face, scalp, palms, and soles, which are usually spared in adults.)

From Management (p.91):

1. **Permethrin cream 5%:**
    - First-line treatment
    - ...adults apply from the neck downwards; **babies/infants apply to all the skin.**

From Scabies - general (p.88):
- Transmission occurs because of
close personal contact... *(Implying family members are often affected if one has it.)*

(Scabies is highly contagious, so a family history of itching is very common if an infant has scabies. Therefore, "No family history of itching" is likely wrong.)

Question 62:
Scabies of infants, all true except:
a. Symptoms at night
b. Sparing face and back
c. Permethrin 5% cream is the first treatment of choice
d. In children it manifests as acral pustules
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Scabies (General features, Scabies in children p.89, Management p.91)):

- **a. Symptoms at night:** Scabies causes "intense itching that characteristically keeps those affected awake at night." (p.88) (True)
- **b. Sparing face and back:** In adults, face and back are often spared. However, in infants, the distribution can be more generalized and **may involve the face, scalp, palms, and soles.** (p.89, general knowledge) (False for infants)
- **c. Permethrin 5% cream is the first treatment of choice:** (p.91) (True)
- **d. In children it manifests as acral pustules:** "Babies and young children ... present with erythematous cutaneous **papules and nodules** in the axillae and on the soles of the feet. Lesions might **blister**." (p.89) Pustules can occur, especially if secondarily infected or as part of the inflammatory reaction, particularly acrally. (Plausibly true, though papules/nodules/blisters are more emphasized)

(Statement B is incorrect for infants, as the face and back can be involved.)

Question 63:
Wrong about scabies:
a. Contagious
b. More at night
c. Affect the back
Answer: C

High-Yield Context (from Dermatology Lecture Slides - Scabies (General features, Distribution p.88)):

- **a. Contagious:** "Transmission occurs because of close personal contact..." (p.88) (True)
- **b. More at night:** "Causes intense itching that characteristically keeps those affected awake at night." (p.88) (True)
- **c. Affect the back:** The typical adult distribution **spares the back** (and face). (From "Sparing face (in adults) and back" diagram on p.88) (False for typical adult scabies)

(The back is typically spared in adult scabies.)

Question 64:
Wrong about scabies:
a. Benzoyl peroxide used as systemic treatment
b. Caused by Sarcoptes Scabiei
Answer: A (benzoyl benzoate not peroxide, which is for acne)

High-Yield Context (from Dermatology Lecture Slides - Scabies (Management p.91, Cause p.88)):

From Management (p.91):

1. Permethrin cream 5%
2. Malathion lotion 0.5%
3. Ivermectin (oral - systemic)
   • If permethrin and malathion are not available, then >> 10% sulphur in yellow soft paraffin is effective and safe.
   • 25%
   benzyl benzoate emulsion may also be used.

   (Benzoyl peroxide is for acne. Benzyl benzoate is a treatment for scabies. "Systemic treatment" usually refers to oral medication like Ivermectin. Topical agents aren't "systemic treatment".)

From Title (p.88):
Scabies (Sarcoptes scabiei) (Statement b is true)

(Benzoyl peroxide is not used for scabies, and certainly not as a systemic treatment.)

Question 65:
A 50-year-old man is suspected of having scabies, which of the following statement regarding scabies is false: **
a. The genitalia is a commonly affected site
b. All members in the same household should be treated at the same time
c. It can spread by simple handshake
d. Children are often affected by scabies
e. Itching can persist for weeks even after successful treatment
Answer: C

High-Yield Context (from Dermatology Lecture Slides - Scabies (Distribution p.88, Transmission p.88, Management p.91)):

- **a. The genitalia is a commonly affected site:** Distribution includes "wrists, nipples, abdomen, **genitalia**, buttocks and ankles." (p.88 - from Box 17.3 image) (True)
- **b. All members in the same household should be treated at the same time:** "Patients should be told that they and **all their close personal contacts need to be treated at the same time**. (to prevent reinfestation)" (p.91) (True)
- **c. It can spread by simple handshake:** "Transmission occurs because of **close personal contact (at least 15 min of skin-to-skin contact)** with an infected individual." (p.88) A simple, brief handshake is usually insufficient. (False)
- **d. Children are often affected by scabies:** (General knowledge, and implied by specific descriptions of scabies in children p.89) (True)

- **e. Itching can persist for weeks even after successful treatment:** "Patients should be warned that the skin itching will take **6–8 weeks to subside**. ... Persistent itching often leads patients to conclude that the mites are still active and they subsequently treat themselves repeatedly, leading to an irritant dermatitis (Post scabies dermatitis)" (p.91) (True)

Acne and Rosacea

Question 66:
In acne vulgaris the precursor of large inflammatory lesions is:
a. Black head
b. White head
c. Papules
d. Pustules
e. None of the above
Answer: C
(Inflammatory lesions (papules, pustules, nodules, cysts) develop from pre-existing comedones (microcomedones, whiteheads, blackheads) when P. acnes proliferates and inflammation occurs. A papule is itself an early inflammatory lesion. If the question means what non-inflammatory lesion precedes a large inflammatory one, then whitehead/blackhead would be better. But a papule is already an inflammatory lesion and can progress to larger ones.)

High-Yield Context (from Dermatology Lecture Slides - Acne (Pathogenesis, p. 96, Clinical features p.98)):

From Pathogenesis (p.96):

1. Thickening of the keratin lining and subsequent obstruction of the sebaceous duct resulting in closed comedones (‘whiteheads’) or open comedones (‘blackheads’)...
2. An increase in sebum secretion.
3. An increase in *Propionibacterium acnes* bacteria within the duct.
4. Inflammation around the sebaceous gland.

   (This diagram shows the progression from microcomedo → comedo → inflammatory papule/pustule → nodule.)

From Clinical features (p.98):
- Non-inflammatory: Open vs closed comedones
- Inflammatory acne:

Papules, pustules, nodules, pseudocystes...

(Comedones (whiteheads/blackheads) are non-inflammatory precursors. Papules are the earliest inflammatory lesions. Large inflammatory lesions like nodules/cysts evolve from these.)The question is somewhat ambiguous. If "precursor" means the immediate preceding lesion type, a papule can become a pustule or nodule. If it means the very first step, it's a microcomedo, then comedo.

Question 67:
A neighbor asks your advice about oral isotretinoin for her severe acne. One of the following is incorrect: **
a. Increased triglycerides is a common side effect
b. The cumulative therapeutic dose varies from one person to another usually depending on their weight
c. Blood test must be done prior to initialization of treatment
d. All patients will experience some degree of lip dryness
e. She should not get pregnant for one year after treatment as it is teratogenic
Answer: E

High-Yield Context (from Dermatology Lecture Slides - Acne (Oral retinoids - Isotretinoin, p. 101, p.102)):

From Oral retinoids (p.101):

•

Side effects:

-

Teratogenesis (90% risk of birth defects), Female patients of childbearing age will need to use a robust form of contraception while taking isotretinoin **and for 1 month following its cessation**. Pregnancy testing is usually undertaken before the release of prescriptions for females.

-

Rise in liver enzymes and lipids, Blood testing before initiation is essential and during therapy as indicated clinically.
- All patients will experience some
drying of the lips and skin.
- Mood change and depression (patient should be told).

From Oral retinoids (p.102):
- The
cumulative target dosage for isotretinoin is **120–150mg/kg.***(Pregnancy must be avoided during treatment and for one month after stopping isotretinoin, not one year. The cumulative dose is usually calculated per kg body weight.)*

Question 68:
Not side effect of retinoids:
a. Paronychia
b. Distal lamellar splitting
c. Nail thinning
Answer: A
(The image has 'a. Paronychia' highlighted, suggesting it IS a side effect, so the question "Not side effect" with answer A would mean Paronychia is NOT a side effect. However, retinoids can cause paronychia and other nail changes. Distal lamellar splitting (onychoschizia) and nail thinning/brittleness are also known side effects of retinoids.)This question seems problematic with the provided answer. Retinoids (especially systemic like isotretinoin or acitretin) are known to cause mucocutaneous dryness, which can affect the nails and periungual skin, leading to paronychia, nail brittleness, thinning, and onychoschizia (splitting).

High-Yield Context (from Dermatology Lecture Slides - Acne (Oral retinoids), p.101, and general knowledge of retinoid side effects):
• Side effects:
- All patients will experience some
drying of the lips and skin.*(The summary on p.101 focuses on major side effects like teratogenicity, lipid/LFT changes, and dryness. Nail-specific side effects are not detailed in this particular summary but are well-documented for systemic retinoids.)*

Question 69:
One is not side effect of Isotretinoin:
a. Increase lipids
b. Scaring alopecia
c. Dryness of mouth
d. Increase liver enzyme
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Acne (Oral retinoids - Isotretinoin), p.101):

- **Side effects:**
  - Teratogenesis...
  -
  Rise in liver enzymes and lipids, Blood testing before initiation is essential...
  - All patients will experience some
  drying of the lips and skin. (Dryness of mouth is part of general mucocutaneous dryness)
  - Mood change and depression...

  (Scarring alopecia is not a typical side effect of isotretinoin; in fact, isotretinoin is used to treat severe acne which causes scarring. While some diffuse, temporary hair thinning (telogen effluvium) can occur, scarring alopecia is not characteristic.)

Question 70:
Not a side effect of retinoic acid: *
a. Thrombocytopenia
b. Elevating liver enzymes
c. Dryness of mucosal membranes
d. Diffuse hair loss
e. Increased intracranial pressure
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Acne (Oral retinoids - Isotretinoin), p.101, noting "retinoic acid" is often used interchangeably with systemic retinoids like isotretinoin in such questions):

- **Side effects:**
  -
  Teratogenesis...
  -
  Rise in liver enzymes and lipids...
  - All patients will experience some
  drying of the lips and skin (mucosal membranes).
  -
  Mood change and depression...
  -
  (Increased intracranial pressure (pseudotumor cerebri) is a rare but serious side effect, especially with concomitant tetracycline use.)
  -
  (Diffuse hair loss (telogen effluvium) can occur.)(Thrombocytopenia is not a commonly listed major side effect of isotretinoin, though changes in other blood counts can occasionally be seen. The others are known side effects.)

Question 71:
Side effects of retinoic acid EXCEPT:
a. Renal failure
b. Fall of hair
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Acne (Oral retinoids - Isotretinoin), p.101):

(As above, diffuse hair fall (telogen effluvium) can be a side effect. Significant renal failure is not a characteristic direct side effect of isotretinoin, though pre-existing renal impairment might require dose adjustment for some drugs, isotretinoin is primarily metabolized by the liver.)

Question 72:
All are side effects of isotretinoin except:
a. Teratogenicity
b. Hair loss
c. Elevated liver enzymes
d. Infertility
Answer: D

High-Yield Context (from Dermatology Lecture Slides - Acne (Oral retinoids - Isotretinoin), p.101):

- **Side effects:**
  -
  Teratogenicity (90% risk of birth defects)...
  -
  Rise in liver enzymes and lipids...
  - All patients will experience some drying of the lips and skin.
  - Mood change and depression...
  -
  (Diffuse hair loss (telogen effluvium) can occur.)(Isotretinoin is a potent teratogen causing birth defects, but it does not cause infertility. Patients must use contraception because of teratogenicity, not due to effects on fertility itself.)

Question 73:
Patient with moderate acne not responding to tetracycline since 6 months, you give:
a. Isotretinoin
b. Antiandrogen
c. Benzyl peroxide
d. Salicylic acid
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Acne (Systemic treatments - Oral retinoids), p.101):

- **Oral retinoids → Iso-Tretinoin**
  • For
  resistant cases
  • SE → teratogenesis, drying of the lips & skin, elevated liver enzymes and lipids...

  (Moderate acne unresponsive to prolonged conventional therapy (like oral antibiotics - tetracycline) is an indication for considering oral isotretinoin.)

Question 74:
All true about acne vulgaris except:
a. Isotretinoin is very effective in cystic form
b. Patients with acne usually have much higher titers to staph. albus than normal adults
Answer: B
(The primary bacterium implicated in acne is Propionibacterium acnes (now Cutibacterium acnes), not Staphylococcus albus (now Staphylococcus epidermidis). While S. epidermidis is part of normal skin flora, higher titers are not a defining characteristic of acne vulgaris pathophysiology focused on P. acnes.)

High-Yield Context (from Dermatology Lecture Slides - Acne (Pathogenesis p.96, Oral retinoids p.101)):

From Pathogenesis (p.96):
3. An increase in
Propionibacterium acnes bacteria within the duct.

From Oral retinoids (p.101):
•
Isotretinoin is usually reserved for **resistant diseases** unresponsive to other oral therapies. *(Cystic acne is a severe, resistant form where isotretinoin is highly effective.)*

(Statement a is true; isotretinoin is very effective for severe cystic acne. Statement b is false; the focus is on P. acnes, not Staph albus.)

Page 11 (Test Bank - Acne and Rosacea cont.)

Question 75 (continued from Page 10):
All true about acne vulgaris except:
a. Isotretinoin is very effective in cystic form
b. Patients with acne usually have much higher titers to staph. albus than normal adults
c. Application of CO2 slush is useful in reducing acne pit scars on the face
d. Greasy cosmetics may cause acne
e. Comedones predominate the picture in chlor-acne
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Acne (Pathogenesis p.96, Oral retinoids p.101, External factors p.97), and general dermatological knowledge):

- **a. Isotretinoin is very effective in cystic form:** (True, see context for Page 10, Q9)
- **b. Patients with acne usually have much higher titers to staph. albus...:** (False, primary bacterium is *P. acnes*, see context for Page 10, Q9)
- **c. Application of CO2 slush...:** (CO2 slush (cryoslush) is an older treatment that causes superficial peeling and was used for acne and superficial scarring. Plausible as "useful", though less common now.)
- **d. Greasy cosmetics may cause acne:**
  - **From Underlying factors - External Factors (p.97):**
    - Oil e.g., cooking, engineering
    - Coal tar
    - **Cosmetics: also comedogenic.**
    - ((Pomade Acne: acne on the hair line as a result of the use of rich oils in the scalp))
  - (True, comedogenic/greasy cosmetics can exacerbate acne.)

- **e. Comedones predominate the picture in chlor-acne:** (Chloracne is a specific type of acne caused by exposure to halogenated aromatic hydrocarbons, classically presenting with numerous comedones. True.)

(Answer B remains the most definitively incorrect statement based on primary acne pathophysiology focusing on P. acnes.)

Question 78:
First lesion of acne
a. Comedones
b. Papule
c. Pustule
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Acne (Pathogenesis diagram p.96, Clinical features p.98)):

From Pathogenesis diagram (p.96): The diagram shows progression from a "Normal follicle" to "Early comedo (microcomedo)", then "Closed comedo (whitehead)" or "Open comedo (blackhead)". These are then followed by "Inflammatory papule/pustule".

From Clinical features (p.98):

•
Non-inflammatory: Open vs closed comedones
- Closed comedones (white heads) small papules, no follicular opening, no erythema
- Open comedones (blackheads) dilated follicular opening with core of shed keratin (color due to melanin and lipid oxidation).

(The earliest clinically visible lesions are comedones (whiteheads and blackheads), which develop from microcomedones. Papules and pustules are inflammatory lesions that develop from comedones.)

Question 79:
All occur in acne vulgaris except:
a. Pustules
b. Nodules
c. Comedones
d. Papules
e. Vesicles
Answer: E

High-Yield Context (from Dermatology Lecture Slides - Acne (Clinical features p.98)):

From Clinical features (p.98):

•
Non-inflammatory: Open vs closed comedones
•
Inflammatory acne: Papules, pustules, nodules, pseudocystes (filled with pus and serosanguinus fluid) may combine to sinus tracts.

(Acne vulgaris is characterized by comedones, papules, pustules, nodules, and cysts. Vesicles (small fluid-filled blisters) are not typical primary lesions of acne vulgaris. Acneiform eruptions from other causes might sometimes have vesicles, but not classic acne vulgaris.)

Question 80:
Wrong about acne vulgaris:
a. Epidermal edema
b. Increase in sebum production
c. Stagnation of ...
d. Proliferation of Propionibacterium
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Acne (Pathogenesis p.96)):

The main changes of acne are (p.96):

1. Thickening of the keratin lining and subsequent obstruction of the sebaceous duct...

2. An **increase in sebum secretion.**
3. An increase in **Propionibacterium acnes** bacteria within the duct.
4. Inflammation around the sebaceous gland.

   (Increased sebum production and proliferation of P. acnes are key. Stagnation of sebum due to ductal obstruction is also part of it. Epidermal edema (spongiosis) is characteristic of eczema, not primarily acne, though inflammation in acne involves dermal edema and inflammatory infiltrate.)

Question 81:
Which is wrong about acne treatment:
Metronidazole is commonly used in systemic treatment of acne

High-Yield Context (from Dermatology Lecture Slides - Acne (Treatment overview pp.100-102) and Rosacea (Treatment p.21, p.104)):

From Acne Treatment (pp.100-102):

- Topical treatments listed: Benzoyl peroxide, Salicylic acid, Azelaic acid, Topical retinoids, Topical antibiotics (erythromycin, clindamycin).
- Systemic treatments listed: Oral antibiotics (Tetracyclines, doxycycline, erythromycin, trimethoprim), Hormone therapies (OCPs, antiandrogens), Oral retinoids (Isotretinoin).

  (Metronidazole is not listed as a standard systemic treatment for acne.)

From Rosacea Treatment (p.21 summary text, p.104):

- **Topical metronidazole** or azelaic acid → mild cases, for papules and pustules
- Oral antibiotics → tetracycline, doxycycline, erythromycin

  (Metronidazole, typically topical, is a mainstay for Rosacea, not systemically for acne vulgaris.)

Question 82:
All of the following result in flare up of acne except: **
a. Estrogen
b. Steroid
c. Antimalarial drugs
d. Vitamin b12
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Acne (Underlying causes - Hormones, Medications p.97)):

From Underlying causes - Hormones (p.97):

- **Androgenic hormones**, Virilising tumours, PCOS, CAH, Cushing's syndrome (**Steroids**)

From Underlying causes - Medications (p.97):

- **Steroids**, OCP, **Phenytoin**(antiepileptics), Isoniazid, Lithium.

(Androgens flare acne. Steroids (corticosteroids) can cause steroid acne. Some OCPs containing certain progestins can flare acne, but OCPs with higher estrogen and anti-androgenic progestins are used to treat acne. Therefore, estrogen itself, especially in the context of combined OCPs, tends to improve acne rather than flare it. Vitamin B12 and antimalarials are not classically listed as major acne flaring agents in this summary, though some case reports exist for B12.)Given the options, estrogen is the most likely "except" as it's often used (in COCPs) to treat acne.

Question 83:
Acne, all true except:
a. Propionibacterium acne is incriminated
b. Isotretinoin is group d in pregnancy
c. Clindamycin is not given to children
d. Follicular plugging is the first step in pathogenesis

Answer: B
(Isotretinoin is Pregnancy Category X, meaning definite human teratogenicity, contraindicating its use if pregnant. Group D implies potential risk, but benefits may outweigh risk in serious situations – this is not the case for isotretinoin during pregnancy as the risk is too high.)

High-Yield Context (from Dermatology Lecture Slides - Acne (Pathogenesis p.96, Oral Retinoids p.101, Topical Antibiotics p.100)):

- **a. Propionibacterium acne is incriminated:**
  - From Pathogenesis (p.96): "An increase in **Propionibacterium acnes** bacteria within the duct." (True)
- **b. Isotretinoin is group d in pregnancy:**
  - From Oral retinoids (p.101): "**Teratogenesis (90% risk of birth defects)**, Female patients of childbearing age will need to use a robust form of contraception..." (This indicates it's far more dangerous than Category D; it's Category X). (False)
- **c. Clindamycin is not given to children:**
  - From Topical treatments - Topical antibiotics (p.100): "...erythromycin and **clindamycin** either alone or in combination..." (Topical clindamycin is widely used. Oral clindamycin has more restrictions, but the statement is broad. Systemic antibiotics like tetracyclines are avoided in young children. There isn't a blanket "not given to children" for clindamycin, especially topical.) (Likely false as a general statement, but depends on context - topical vs systemic and age of child).
- **d. Follicular plugging is the first step in pathogenesis:**
  - From Pathogenesis (p.96): "Thickening of the keratin lining and **subsequent obstruction of the sebaceous duct** resulting in closed comedones...". The diagram also shows the "Early comedo (microcomedo)" as the first pathological step. (True)

(Isotretinoin is Pregnancy Category X, not D. This is a critical safety point.)

Question 84:
One false about acne rosacea:
a. Occur in teenagers
b. Associated with telangiectasia
c. Associated with rhinophyma
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Rosacea (Definition p.21/p.102, Clinical types p.103)):

From Definition (p.21/p.102):
- Characterized by key components (Facial flushing, persistent facial erythema, **telangiectasia**, and inflammatory papules/pustules and edema).
- (NO comedones)

From Clinical types (p.103):

1. Erythematotelangiectatic: flushing, facial erythema +- **telangiectasia**
2. papulopustular: centrofacial eruption multiple small papules
3. **Phymatous**: thickened hypertrophic nodular skin, prominent pores, can affect **nose (rhinophyma)**, chin, forehead, ears, eyelids

   (Rosacea typically affects adults, usually 30-50 years old, not primarily teenagers. Acne vulgaris is common in teenagers. Telangiectasia and rhinophyma are characteristic features of rosacea.)

Question 85:
Difference between acne vulgaris and rosacea:
a. Comedones
b. Pustules
c. Papules
d. Telangiectasia

e. Erythema
Answer: A (found in vulgaris not rosacea) + D (found in rosacea not vulgaris)

High-Yield Context (from Dermatology Lecture Slides - Acne (Clinical features p.98) and Rosacea (Definition p.21/p.102)):

Acne Vulgaris (p.98):
- Non-inflammatory: Open vs closed

comedones
- Inflammatory acne:

Papules, pustules, nodules, pseudocystes.

(Erythema is present around inflammatory acne lesions. Telangiectasias are not a primary feature of acne vulgaris.)

Rosacea (p.21/p.102):
- Characterized by key components (Facial flushing, persistent facial

erythema, **telangiectasia**, and inflammatory **papules/pustules** and edema).
-

(NO comedones)*(The presence of comedones is characteristic of acne vulgaris and their absence is characteristic of rosacea. Telangiectasia is characteristic of rosacea but not acne vulgaris. Both can have papules, pustules, and erythema.)*

Urticaria and angioedema

Question 86:
Edematous erythematous lesion that blanches with pressure:
Wheal

High-Yield Context (from Dermatology Lecture Slides - Urticaria and Angioedema (Introduction p.10, p.80)):

From Introduction (p.10 summary, p.80 lecture):
- Urticaria describes transient pruritic swellings of the skin often referred to as

wheals.
- Results from edema in the superficial layers of the skin causing

well-demarcated erythematous lesions.
- Primary lesion →

wheal surrounded by flare

(This is the definition of a wheal/hive, the primary lesion of urticaria. They are edematous, erythematous, and blanchable.)

Question 87:
False about urticaria:
a. Leaves hypopigmented scar
b. 90% of chronic cases the cause is unknown
c. Wheal is the primary lesion
d. Very itchy
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Urticaria and Angioedema (Introduction p.10/p.80, Classification p.82)):

From Introduction (p.10 summary, p.80 lecture):
- Urticaria describes

transient pruritic swellings... **wheals**...
- Resolve with

no marks on the skin. (This implies no scarring or pigmentation changes typically).
- Common, self limiting, controlled with antihistamine.
- Onset → Minutes to hours, lasting minutes or hours (usually less than 24 h)
- Primary lesion →

wheal surrounded by flare.
- Urticaria is very

itchy...

From Classification (p.82):

- Another approach is to classify urticaria according to the underlying cause, but in

50% of cases no cause is identified (idiopathic).*(Wheals are transient and resolve without scarring or typically pigmentation changes. Chronic idiopathic urticaria is common, though 50% not 90% is cited in the lecture slide. Wheal is the primary lesion. It is very itchy.)Given the options, statement A is definitively false. While the 90% figure for idiopathic chronic urticaria might be high compared to the summary's 50%, the lack of scarring is a key feature.*

Question 88:

ttt of choice for acute urticaria: *

a. Antihistamine

b. Systemic steroids

c. Local steroids

d. Adrenaline

e. Kallikrein

Answer: A

High-Yield Context (from Dermatology Lecture Slides - Urticaria and Angioedema (General management p.10/p.86)):

From General management (p.10 summary, p.86 lecture):

-

Oral antihistamines are the mainstay of treatment/prevention of urticaria and angioedema. (2nd generation H1 blockers regular dose, up to 4 tabs daily, not PRN)

- In severe cases: synergistic combinations of H1-receptor blockers plus H2 blockade...and leukotriene receptor antagonists...
- Oral corticosteroids may be indicated in very severe eruptions, particularly those associated with urticarial vasculitis. (not for long duration)
- Patients who are known to be at risk of severe life-threatening urticaria/ angioedema with respiratory distress may be asked to carry with them a pre-assembled syringe and needle (EpiPen®, or Anapen®) to inject

adrenaline intramuscularly

(Antihistamines are the first-line treatment for acute urticaria. Systemic steroids are for severe/resistant cases. Adrenaline is for anaphylaxis/severe angioedema with respiratory compromise.)

Question 89:

Urticaria, which is wrong: **

a. Oral steroids are first line treatment

b. Sedating and non-sedating antihistamine are used

Answer: A

High-Yield Context (from Dermatology Lecture Slides - Urticaria and Angioedema (General management p.10/p.86)):

From General management (p.10 summary, p.86 lecture):

-

Oral antihistamines are the mainstay of treatment/prevention... (2nd generation H1 blockers regular dose... e.g. Loratidine, Cetrizine, Desloratidine. Also implies use of older sedating ones if needed, or for nighttime.)

-

Oral corticosteroids may be indicated in very severe eruptions... (not for long duration)

(Antihistamines are first-line. Oral steroids are reserved for severe or refractory cases, not first-line.)

Question 90:

Cold urticaria:

a. Sometimes familial

b. Usually acquired

c. May be ..... transferable in serum

d. May result in unconscious .....

e. All of the above

Answer: E

High-Yield Context (from Dermatology Lecture Slides - Urticaria and Angioedema (Cholinergic/Cold Urticaria p.83), general knowledge):

From Cholinergic urticaria (p.83):
• A rarer form of cholinergic urticaria can result
from exposure to the cold:
- Patients report urticaria on exposed skin during cold weather... more generalized reaction following swimming in an outdoor pool.
- Affected individuals should avoid swimming in cold water and ingestion of ice-cold drinks as **anaphylaxis and death have been reported.** (implies potential for unconsciousness/severe reaction).

(The summary talks about cold-induced cholinergic urticaria. Classic cold contact urticaria can be acquired (most common) or rarely familial. Some forms of acquired cold urticaria can be associated with cryoglobulins, which are transferable in serum. Severe reactions can lead to anaphylaxis and unconsciousness, especially with large area exposure like swimming in cold water.)

Question 91:
The main cells involved in urticaria are:
a. Neutrophils
b. Mast cells
c. Eosinophils
d. Histamine
e. Lymphocytes
Answer: B
(Histamine is a mediator, not a cell. Mast cell degranulation releases histamine and other mediators.)

High-Yield Context (from Dermatology Lecture Slides - Urticaria and Angioedema (Pathophysiology p.10/p.80)):

From Pathophysiology (p.10 summary, p.80 lecture):
•
Mast cell degranulation due to a trigger will lead to the release of **histamine**, bradykinin, and other pro-inflammatory mediators
- Urticaria may be IgE mediated... or
mast cells may be directly stimulated...
- In patients with chronic urticaria,
histamine can be released spontaneously or in response to non-specific stimuli...

(Mast cells are central to the pathogenesis of urticaria through the release of histamine and other mediators.)

Question 92:
Most reliable Dx test of cholinergic urticaria:
a. Intradermal methacholine
b. Intradermal nicotinic acid
c. Intradermal sciine
d. Biopsy
e. Exercise & heat
Answer: A
(The answer key indicates A. Provocation with exercise and heat (option e) is the common clinical way to diagnose cholinergic urticaria. Intradermal methacholine can induce whealing in some patients with cholinergic urticaria because their mast cells are hyperreactive to cholinergic stimuli, but exercise/heat provocation is more standard and physiological. The question asks for "most reliable Dx test". The summary (p.82) lists "Cholinergic urticaria: typically urticaria following a warm shower/bath, or after exercise.")

*Let's check the lecture slides (p.82) for diagnostic tests for cholinergic urticaria.***From Cholinergic urticaria (p.82):**
- Typically urticaria
following a warm shower/bath, or after exercise.
- The lesions consist of pinhead-sized wheals with a red flare around them.

(The summary does not list specific diagnostic tests beyond clinical provocation by heat/exercise. Intradermal methacholine is a known test, but its reliability compared to physiological provocation is debatable. If the test bank answer is A, we'll assume it's considered highly specific in that context.)

Question 93:
All these drugs used for ttt of urticaria except:

a. Systemic steroid
b. Dimetane tab
c. Allerfrin tab
d. Antihistamine ointment
Answer: D

High-Yield Context (from Dermatology Lecture Slides - Urticaria and Angioedema (General management p.10/p.86)):

From General management (p.10 summary, p.86 lecture):

- **Oral antihistamines are the mainstay**... (Dimetane and Allerfrin are brand names for antihistamines).
- **Oral corticosteroids may be indicated in very severe eruptions**...

(Topical antihistamines (ointments) are generally not effective for urticaria as the process is systemic and involves deeper mast cell degranulation. Oral administration is required for effective systemic antihistamine levels.)

Question 94:
Best antihistamine for day-time use:
a. Ethanolamines
b. Piperidines
c. Phenothiazines
d. Ethvlenediamines
e. Alkylamines (Page 13)
Answer: D {not sure } (Answer from page 13 is D. Piperidines (b) often refers to second-generation, non-sedating antihistamines like loratadine, fexofenadine, which are ideal for daytime. Ethanolamines, some Alkylamines, and Phenothiazines are older, sedating antihistamines. Ethylenediamines are also a class of first-generation antihistamines, some with sedation.)*

The provided answer D {not sure} on page 13 is for Ethylenediamines. Generally, for daytime use, second-generation antihistamines (many of which are piperidine derivatives like loratadine, fexofenadine, or piperazine derivatives like cetirizine) are preferred due to their non-sedating properties. The classes listed (Ethanolamines, Phenothiazines, Ethylenediamines, Alkylamines) are mostly first-generation. Among first-gen, some alkylamines are considered less sedating than others, but second-gen are superior for daytime.)

High-Yield Context (from Dermatology Lecture Slides - Urticaria and Angioedema (General management p.10/p.86)):

- Oral antihistamines are the mainstay...

(2nd generation H1 blockers regular dose... e.g. Loratidine, Cetrizine, Desloratidine.

(The summary emphasizes 2nd generation H1 blockers which are non-sedating and suitable for daytime use. These often fall under piperidine or piperazine derivatives.)

Page 13 (Test Bank - Urticaria and angioedema cont. & general question)

Question 95 (continued from page 12, question 9e):
Best antihistamine for day-time use:
e. Alkylamines
Answer: D {not sure }
(As discussed above, the answer key D refers to option d from page 12 (Ethylenediamines). Second-generation non-sedating antihistamines are best for daytime.)

High-Yield Context: (Already covered under Page 12, Question 9).

Question 96:
Degree of level of contact sensitivity to an allergen is influenced by:
a. Amount of allergen to which the subjects exposed
b. Frequency of exposure to the allergen
c. The route of exposure
d. All of the above
answer: D

High-Yield Context (from Dermatology Lecture Slides - Allergic Contact Dermatitis, general principles, p.164-165):

From Allergic contact dermatitis (p.164):

- This is due to the development of delayed hypersensitivity (type 4 allergy) to a specific chemical (**sensitizer or allergen**).
- Such allergens will not cause eczema even in high concentration in a normal person, but severe eczema may be provoked by

brief exposure to a very low concentration in a sensitized person.*(This implies concentration (amount) and previous sensitization (related to frequency/route) are important. The "degree of level" of sensitivity itself is established during the sensitization phase and then elicited upon re-exposure. The factors listed (amount, frequency, route) all play a role in both initial sensitization and the elicitation/severity of reaction upon re-exposure in a sensitized individual.)General immunological principles support that amount, frequency, and route of exposure influence sensitization and the severity of subsequent allergic reactions.*

Drug rashes

Question 97:

Acne medicinosa by all of following except:

a. Phenytoin

b. B12

c. Azelaic acid

d. Steroid

Answer: C

High-Yield Context (from Dermatology Lecture Slides - Drug Rashes that alter normal skin function (Acne), p.12; Acne (Underlying causes - Medications), p.97):

From Drug Rashes (Drugs which exacerbate pre-existing dermatoses - Acne, p.12):

- Acne → OCP, particularly progesterone-only pills,

Corticosteroids, cyclosporine and anti-epileptics such as **phenytoin**

From Acne - Underlying causes - Medications (p.97):

-

Steroids, OCP, **Phenytoin**(antiepileptics), Isoniazid, Lithium.

(Steroids and phenytoin are listed as causing/exacerbating acne (acne medicamentosa or drug-induced acne). Vitamin B12 in high doses has been anecdotally linked to acneiform eruptions. Azelaic acid is a treatment for acne and rosacea, not a cause.)

From Acne - Topical treatments (p.100):

-

Azelaic acid→ anti keratinising and antibacterial effects

Question 98:

Wrong about TEN:

a. Most common cause is infection

b. Requires intensive care most of the time

c. Highly fatal

Answer: A

High-Yield Context (from Dermatology Lecture Slides - Drug Rashes (Severe drug reactions - SJS/TEN), p.13, p.31):

From Severe drug reactions in the skin - Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) (p.13 summary, p.31 lecture):

-

life-threatening drug-induced hypersensitivity reactions in the skin and mucous membranes

- Tx → stop the drug and

supportive care and intravenous immunoglobulin or corticosteroids

From SJS/TEN (p.31 in lecture slides):

- Mortality from TEN may be as high as 90% (score >5) and is estimated using the SCORTEN tool

(TEN is almost always drug-induced. Infections can cause SJS-like illnesses (e.g. Mycoplasma-induced rash and

mucositis), but classic TEN is overwhelmingly drug-related. It requires intensive care due to extensive skin loss and is highly fatal.)

From Drug Rashes - Common drug-induced rashes table (p.12):
SJS/TEN:
Antibiotics, anticonvulsants, NSAIDs, anti-retrovirals, allopurinol, barbiturates, ramipril, diltiazem. *(All drugs)*

Question 99:
The tissue mainly involved in Steven Johnson syndrome is:
a. skin
b. lungs
c. mucocutaneous membrane
d. urinary bladder
e. liver
Answer: C

High-Yield Context (from Dermatology Lecture Slides - Drug Rashes (Severe drug reactions - SJS/TEN), p.13, p.31, p.32):

From Severe drug reactions in the skin - Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) (p.13 summary, p.31/32 lecture):
- life-threatening drug-induced hypersensitivity reactions in the

skin and mucous membranes
- (p.32) Characterized by: Widespread, painful areas of epidermal detachment;

Erosions of the mucous membranes, including eyes, mouth, genitalia and respiratory tract.

(SJS/TEN characteristically involves severe mucocutaneous blistering and erosions.)

Question 100:
Wrong:
Steven Johnson Syndrome involves oral mucosa and skin of more than 30% of body surface area

High-Yield Context (from Dermatology Lecture Slides - Drug Rashes (Severe drug reactions - SJS/TEN), p.13, p.31):

From Severe drug reactions in the skin - Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) (p.13 summary, p.31 lecture):
- The terms SJS and TEN represent points along a spectrum of severity,
-

SJS → <10% BSA detachment,
- TEN → >30% BSA,
- 'SJS–TEN overlap' →between 10% and 30% loss.

(SJS is defined as <10% BSA detachment. Involvement of >30% BSA is classified as TEN. So the statement is wrong.)

Question 101:
A 30-year-old epileptic patient is admitted to hospital with a suspected acute drug eruption. Through systematic history taking and physical examination you suspect toxic epidermal necrolysis. Which of the following would not fit with your diagnosis?
a. Commencing a new anti-epileptic medication 2 months ago
b. A prodrome of fever and malaise
c. Painful skin
d. 40% skin detached
e. The absence of oral erosions
Answer: E

High-Yield Context (from Dermatology Lecture Slides - Drug Rashes (Severe drug reactions - SJS/TEN), p.13, p.31, p.32):

From SJS/TEN (p.31/32 lecture):
- (p.32) Characterized by:

Widespread, painful areas of epidermal detachment; **Erosions of the mucous membranes, including eyes, mouth, genitalia** and respiratory tract.
- (p.32) The appearance of the eruption may be preceded by a

prodrome of fever, malaise and coryzal symptoms, and **skin pain** is often the first cutaneous manifestation, prior to the

appearance of the rash.
- (p.31) Medications: Anticonvulsants...

(often implicated). Latency periods for drug reactions can be weeks to months.
- (p.31) TEN →

>30% BSA*(Oral erosions are a hallmark of SJS/TEN. Absence of oral erosions would be atypical for TEN. 40% skin detachment fits TEN. Prodrome and painful skin are typical. Antiepileptics are common culprits, and a 2-month latency is plausible.)*

Bullous disease

Question 102:
Bullous pemphigoid:
a. Linear IgG & C3
b. Granular IgG & C3
c. Linear IgA & C3
d. Granular IgA & C3
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Bullous Diseases (Investigation - Direct Immunofluorescence for Bullous Pemphigoid), p.14, and Pemphigoid, p.70, p.71, p.76):

From Table 8.1 Skin biopsy findings in immunobullous diseases (p.14 summary / p.71 lecture):
Bullous pemphigoid:
Histology features: Subepidermal blister containing mainly eosinophils
Immunofluorescence features:
Linear band of IgG at the basement membrane zone. Linear band of C3 at the basement membrane zone

From Pemphigoid (p.70 lecture):
- Autoimmune sub-epidermal blistering diseases with circulating IgG and

basement membrane zone–bound IgG antibodies and C3.
- By DIF,

linear deposits of IgG, C3, or IgA at the dermo–epidermal junction.

From Bullous Pemphigoid - Direct immunofluorescence (p.76 lecture):

- Usually demonstrates IgG and C3 deposition in a **linear band** at the dermal-epidermal junction

(Bullous Pemphigoid is characterized by linear deposits of IgG and C3 along the basement membrane zone on direct immunofluorescence.)

Question 103:
Antibodies are directed in bullous pemphigoid towards:
a. Hemidesmosomes
b. Desmosomes
c. dermal papilla
d. Granular cell layer
e. None of the above
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Bullous Diseases (Pathophysiology - Bullous Pemphigoid vs Pemphigus), p.13, p.69, p.70):

From Pathophysiology (p.13 summary):
○
Bullous pemphigoid → IgG autoantibodies that target the basement membrane cells (hemidesmosome proteins)
- ○ Pemphigus Vulgaris → autoantibodies directed against desmosomal cadherin desmoglein 3 (Dsg3)
- ○ Pemphigus foliaceous (PF) → desmoglein 1 (Dsg1)

From Pathophysiology (lecture slide p.70):
Bullous pemphigoid: Results from autoantibodies that target the basement membrane cells (
hemidesmosome proteins BP180 and BP230).

(Antibodies in Bullous Pemphigoid target components of the hemidesmosome, specifically BPAG1 (BP230) and BPAG2 (BP180).)

Question 104:
Which of the following diagnostic aids is the most valuable in differentiating bullous pemphigoid from erythema multiforme:
a. Histology
b. Tzanck test
c. Immunofluorescence
d. Electron microscopy
e. Clinical features
Answer: C

High-Yield Context (from Dermatology Lecture Slides - Bullous Diseases (Investigation), p.14/p.71):

From Investigation (p.14 summary / p.71 lecture):

- **direct immunofluorescent analysis of perilesional skin. → gold standard**
- The level and pattern of immunoglobulin staining on direct immunofluorescence is diagnostic.

(Direct immunofluorescence (DIF) showing linear IgG/C3 at the BMZ is diagnostic for Bullous Pemphigoid. Erythema Multiforme is an interface dermatitis, and while it can have bullae, its DIF findings are different (often granular C3/IgM in vessel walls or negative).)

Question 105:
Wrong about pemphigus and pemphigoid:
a. Pemphigoid is associated with more morbidity and mortality
b. Abs against desmogleins in pemphigus and collagen 17 in pemphigoid
c. Intraepidermal blisters in pemphigus
d. Subepidermal blisters in pemphigoid
Answer: A
(Pemphigus vulgaris, especially before steroids, was often fatal and associated with high morbidity. Bullous pemphigoid, while serious, generally has a better prognosis, though it can also have significant morbidity in the elderly.)

High-Yield Context (from Dermatology Lecture Slides - Bullous Diseases (Intro, Pathophysiology, Specific diseases), pp.13-15, pp.69-78):

- **a. Pemphigoid morbidity/mortality:**
  - Pemphigus Vulgaris (p.72): Historically high mortality. Still significant.
  - Bullous Pemphigoid (p.75): Affects elderly, can have morbidity.
  - *(Generally, Pemphigus Vulgaris is considered to have higher morbidity and mortality than Bullous Pemphigoid, especially if untreated.)* (Statement A is likely false as Pemphigus is often worse).
- **b. Abs against desmogleins in pemphigus and collagen 17 in pemphigoid:**
  - Pemphigus (p.70): Abs against **desmoglein 3 (P. vulgaris) or desmoglein 1 (P. foliaceus)**.
  - Pemphigoid (p.70): Abs against hemidesmosome proteins **BP180 (Collagen XVII)** and BP230. (True)
- **c. Intraepidermal blisters in pemphigus:**
  - Pemphigus (p.70): **Intraepidermal** split leads to flaccid bullae formation. (True)
- **d. Subepidermal blisters in pemphigoid:**
  - Pemphigoid (p.70): **Subepidermal** split leads to tense bullae formation. (True)

(Statement A is the most likely "wrong" statement, as Pemphigus Vulgaris is generally considered more severe with higher mortality than Bullous Pemphigoid.)

Question 106:
Pemphigus vulgaris characterized by all except:
a. More in elderly people
b. Rare disease
c. More in jews
d. Fatal if untreated
e. Presence of acantholysis
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Pemphigus Vulgaris, pp. 72-74):

From Pemphigus Vulgaris (p.72):

- Affects

middle-aged people (40-60).

•

(Rare disease is generally true.)

•

(Increased incidence in Ashkenazi Jews is well-documented, so "More in jews" is true.)

•

(Historically fatal if untreated due to fluid/protein loss and infection. True.)

•

(Acantholysis - loss of keratinocyte adhesion - is the hallmark histological feature causing intraepidermal blisters. True.)

(Pemphigus vulgaris typically affects middle-aged individuals (40-60 years), not primarily "elderly people", although it can occur in older individuals. Bullous Pemphigoid is more characteristic of the elderly.)

Question 107:

Which of the following disease showing racial prevalence:
a. Bullous pemphigoid
b. Pemphigus vulgaris
c. Chronic cicatricial pemphigoid
d. Juvenile pemphigoid
e. Dermatitis herpetiformis
answer: B

High-Yield Context (from Dermatology Lecture Slides - Pemphigus Vulgaris, general knowledge):

(As mentioned above, Pemphigus vulgaris has a higher incidence in individuals of Ashkenazi Jewish descent and those from the Mediterranean basin. Other bullous diseases listed don't have such a distinct racial/ethnic predilection highlighted in standard texts or this summary.)

Question 108:

Wrong about pemphigus vulgaris:
a. Middle age
b. Bad general condition
c. Tense bulla
Answer: C

High-Yield Context (from Dermatology Lecture Slides - Pemphigus Vulgaris, p.72, p.70):

From Pemphigus Vulgaris (p.72):

- Affects

middle-aged people (40-60). (Statement a = True)

- Characterized by painful

flaccid blisters and erosions arising on normal skin.

- Patient is

ill. (Implies bad general condition) (Statement b = True)

From Pathophysiology - Pemphigus vulgaris (p.70):

- Intraepidermal split leads to

flaccid bullae formation.

(Pemphigus vulgaris blisters are characteristically flaccid due to the superficial, intraepidermal split. Tense bullae are characteristic of subepidermal blistering diseases like Bullous Pemphigoid.)

Question 109:

Patient presented with painful mouth ulcers and flaccid bullae, which of the following is the most likely diagnosis:
Pemphigus vulgaris

High-Yield Context (from Dermatology Lecture Slides - Pemphigus Vulgaris, p.72):

From Pemphigus Vulgaris (p.72):

- Seventy percent of patients develop

painful oral erosions which usually precede the onset of skin blisters by weeks or months.

- Characterized by painful

flaccid blisters and erosions arising on normal skin.

- The bullae are easily broken and even rubbing normal skin causes the superficial epidermis to slough off (Positive Nikolsky's sign).

(This is the classic presentation of Pemphigus Vulgaris.)

Question 110:
The bullae of pemphigus vulgaris are:
a. Subcorneal
b. Supradermal
c. Dermal
d. None of the above (Added based on typical MCQs, as Supradermal is vague, Subcorneal is for P. Foliaceus, Dermal implies subepidermal)
Answer: D
(The OCR for the question ends here, the answer implies options were given. The split is intraepidermal, specifically suprabasal for P. vulgaris. "Supradermal" is not a standard histological term for level of split. "Subcorneal" is for P. foliaceus. "Dermal" would mean subepidermal. So, if the options were restrictive, "None of the above" might be correct if "intraepidermal, suprabasal" was not an option.)

High-Yield Context (from Dermatology Lecture Slides - Pemphigus Vulgaris - Diagnosis, p.74, Pathophysiology p.70):

From Diagnosis of PV - Histopathology (p.74):

- **Intraepidermal cleft**
- **Supra-basal** epidermal cells separate from the basal cells to form clefts and blisters.

From Pathophysiology - Pemphigus vulgaris (p.70):

- **Intraepidermal split** leads to flaccid bullae formation.

(The bullae are intraepidermal, specifically suprabasal.)

Page 16 (Test Bank - Bullous diseases cont.)

Question 111 (continuation of page 15, question 9d):
The bullae of pemphigus vulgaris are:
d. None of the above
Answer: D
(Assuming the options were Subcorneal, Supradermal, Dermal, then None is appropriate as it's Intraepidermal, Suprabasal)

High-Yield Context: (Already covered under Page 15, Question 9).

Question 112:
Pemphigus vulgaris is a: *
a. viral dis.
b. Autoimmune dis
c. Bacterial dis.
d. Hormonal dis.
e. Unknown
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Pemphigus, p.72, and Autoimmune Blistering Diseases intro p.70):

From Pemphigus (p.72):

- A rare group of **autoimmune**, intra-epidermal blistering diseases involving the skin and mucous membranes.

From Autoimmune Blistering diseases (p.70):

Impaired adhesion of epidermal cells to each other or impaired adhesion of the epidermis to the epidermal basement membrane mediated by our own immune system;
auto-immune.

Question 113:
Not a cause of generalized blistering?
Pemphigus gestationis

High-Yield Context (from Dermatology Lecture Slides - Pemphigoid Gestationis, p.77):

From Pemphigoid Gestationis (p.77):
- Acute-onset intensely pruritic papules, plaques, and blisters spread

from the periumbilical area outwards. (umbilical involvement)

(While it can spread, it often starts localized around the umbilicus and then may become more generalized, but it's not inherently a primary generalized blistering disease in the same way some others might present initially. It is a blistering disease, however. The question is "Not a cause of generalized blistering?". PG can become generalized.)This question is tricky. Pemphigoid gestationis is a blistering disease and can become generalized. Perhaps it's considered less "generalized" from onset compared to widespread Bullous Pemphigoid or severe Pemphigus Vulgaris presentations. Without other options, it's hard to judge its "incorrectness" definitively from the summary alone.

Question 114:
One doesn’t cause epidermal bullous:
a. Impetigo
b. Dermatitis herpatiformes
c. Eczema
d. Pemphigoid vulgaris
Answer: C *(Dermatitis Herpetiformis (b) and Pemphigoid Vulgaris (d - should be Bullous Pemphigoid or Pemphigus Vulgaris for context) cause
subepidermal and *intraepidermal* bullae respectively. Eczema (c) causes *intraepidermal* vesicles/bullae due to spongiosis. Impetigo (a) can cause superficial intraepidermal (subcorneal) blisters in its bullous form. So the question might be asking which one *doesn't* cause bullae that are primarily defined by an "epidermal" (i.e. within the epidermis or at its base) defect leading to the bulla. All listed conditions cause blisters related to epidermal integrity or the dermo-epidermal junction.
"Epidermal bullous" is broad.
If "epidermal bullous" means the split is
within the epidermis:

- Impetigo (bullous form): subcorneal (intraepidermal)
- Dermatitis herpetiformis: subepidermal
- Eczema: intraepidermal (spongiotic vesicles)
- Pemphigoid (Bullous Pemphigoid): subepidermal
- Pemphigus Vulgaris: intraepidermal (suprabasal)

Given the answer C (Eczema): Eczema causes intraepidermal vesicles due to spongiosis. Perhaps the term "bullous" is key, and eczema vesicles are usually smaller, though bullous eczema exists. Dermatitis herpetiformis and Pemphigoid cause subepidermal bullae. Impetigo can be bullous. This answer is confusing based on the summary.
If the question meant "One doesn't cause
autoimmune epidermal bullous disease", then Eczema would fit. But the question is general.
The lecture summary describes Eczema (p.155) with "vesicle formation" from spongiosis, and Pemphigoid (p.70) as "sub-epidermal blistering".)*

High-Yield Context (from various summary sections):

- **Impetigo (p.116):** Bullous lesions can occur. Histologically, bullous impetigo shows subcorneal cleavage.
- **Dermatitis Herpetiformis (p.78):** Autoimmune blistering disorder. Histology: small vesicles containing neutrophils (micro-abscesses) in the upper dermis. DIF: granular IgA in upper dermis. This leads to a *subepidermal* blister.
- **Eczema (p.156):** Acute Eczema: Vesicular/bullous lesions and edema. Histology (p.155): edema in the epidermis leading to spongiosis and vesicle formation (intraepidermal).

- **Pemphigoid (Bullous Pemphigoid) (p.75):** Autoimmune blistering skin disease. Characterized by tense blisters (subepidermal).
- **Pemphigus Vulgaris (p.72):** Flaccid blisters (intraepidermal).

If the answer is C (Eczema), it's problematic as acute eczema does cause epidermal vesicles/bullae. Perhaps the distinction is between "bullous disease" (a primary blistering disorder) vs. vesicles as a secondary feature of inflammation as in eczema. Dermatitis Herpetiformis and Pemphigoid are primary bullous diseases.

Question 115:
Mucous membranes are extensively involved in epidermolysis bullosa:
a. Simplex
b. Hyperplastica
c. Polydysplastica
d. Cockayne
Answer: {not sure }
(The Test Bank marks this as "not sure". Epidermolysis Bullosa (EB) is a group of inherited blistering disorders. Mucous membrane involvement varies greatly depending on the type and subtype of EB. Some forms, particularly Junctional EB and Dystrophic EB, can have severe and extensive mucous membrane involvement. EB Simplex often has milder or no mucosal involvement. "Hyperplastica" and "Polydysplastica" are not standard major classifications of EB, though some subtypes might have names including these terms or reflecting these features. Cockayne syndrome is a separate progeroid disorder.)

High-Yield Context (from general knowledge, as EB is not detailed in the provided summary):*Epidermolysis Bullosa is a group of inherited mechanobullous disorders. Mucosal involvement is common and can be severe in Junctional EB and Dystrophic EB. EB Simplex typically has less severe mucosal involvement.*

Question 116:
All arc healing diseases without scaring except:
a. Epidermolysis bullosa simplex
b. Rash of secondary syphilis
c. Impetigo
d. Dystrophic epidermolysis bullosa
e. Herpes zoster
Answer: D

High-Yield Context (from various summary sections and general knowledge):

- **a. Epidermolysis bullosa simplex:** Blisters in EB Simplex are typically intraepidermal (within basal layer or above) and generally heal *without* scarring, though milia and nail dystrophy can occur.
- **b. Rash of secondary syphilis (p.40):** Usually macular or papulosquamous, resolves without scarring.
- **c. Impetigo (p.116):** Superficial bacterial infection, usually heals without scarring unless deeply excoriated or ecthyma develops.
- **d. Dystrophic epidermolysis bullosa:** Blisters form beneath the lamina densa in the upper dermis. Healing is characterized by significant **scarring**, milia formation, and mitten deformities.
- **e. Herpes zoster (p.126):** Vesicular eruption, usually heals well but can leave post-inflammatory pigmentation changes or, if severe or secondarily infected, scarring, especially in older or immunocompromised individuals. Generally, uncomplicated zoster heals without significant scarring.

(Dystrophic EB is defined by its scarring potential due to the deep level of blistering.)

Connective tissue disease / Cutaneous manifestations of systemic disease

Question 117:
Which of the following result in hypertrophy and distal proliferation of nail circle on nail fold:
Lichen planus

High-Yield Context (from Dermatology Lecture Slides - Connective Tissue Disease, Vasculitis (Lichen planus - Nails), p.16, and Diseases of the Nails (Lichen planus), p.32):

From Diseases of the Nails - Lichen planus (p.32):
● Lichen planus →
atrophy of the nail plate, pterygium “cuticle may be thickened and grow over the nail plate”

(Lichen planus of the nails can cause various changes including thinning, ridging, and pterygium formation (scarring where the nail fold fuses to the nail bed, often with loss of the nail plate). "Hypertrophy and distal proliferation of nail circle on nail fold" is a bit vague, but pterygium involves the nail fold and can lead to nail destruction and an altered appearance of the nail matrix/fold area.)

Question 118:
Wrong about lichen planus:
a. If it involves the mucosa, gingivae is the most common location
b. Itchy
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Connective Tissue Disease, Vasculitis (Lichen planus), p.16):

From Lichen planus (LP) (p.16):
●
itchy eruption, shiny purple-coloured flat-topped papules, on the wrists and ankles.
● White lines called Wickham’s striae may appear on the surface of the lesions
● May affect the
mouth, labia minora, nails”linear ridges” or scalp “scarring alopecia”
● Variants → hypertrophic,
bullous, oral LP*(Lichen planus is characteristically itchy (one of the "P"s - Pruritic). Oral LP is common, and while gingivae can be affected, the buccal mucosa is a very common, if not the most common, site for oral LP, often showing lacy white Wickham's striae.)Statement A: While gingival LP occurs, buccal mucosa is classically considered the most common site for oral LP.*

Question 119:
Not in lichen planus nail:
a. Thinning
b. Dystrophy
c. Pterygium
d. Longitudinal riding
e. Paronychia
Answer: E

High-Yield Context (from Dermatology Lecture Slides - Connective Tissue Disease, Vasculitis (Lichen planus - Nails), p.16, and Diseases of the Nails (Lichen planus), p.32):

From Lichen planus (LP) (p.16):
● May affect the ...
nails”linear ridges” ...

From Diseases of the Nails - Lichen planus (p.32):
● Lichen planus →
atrophy (thinning) of the nail plate, pterygium “cuticle may be thickened and grow over the nail plate”

(Nail LP can cause thinning, atrophy (leading to dystrophy), pterygium, and longitudinal ridging/fissuring. Paronychia (inflammation of the nail folds) is not a primary characteristic feature of nail lichen planus itself, though severe inflammation or secondary infection could theoretically lead to it, it's not a defining feature.)

Question 120:
All of the following are common changes of the nail in lichen planus except:
a. Pitting
b. Pterygium
c. Thinning
d. Longitudinal ridging
e. Onycholysis
Answer: A

(Pitting is very characteristic of psoriasis, less so for lichen planus. Onycholysis can occur in LP, but pitting is not a primary feature.)

High-Yield Context (from Dermatology Lecture Slides - Diseases of the Nails (Lichen planus vs Psoriasis), p.32):

From Lichen planus (p.32):

- **atrophy (thinning) of the nail plate, pterygium**, cuticle may be thickened and grow over the nail plate. (Longitudinal ridging is also common). Onycholysis (separation of nail from bed) can also occur.

From Psoriasis (p.32):

- **pitting**, transverse ridges, onycholysis, oily spots, subungual hyperkeratosis

(Pitting is a hallmark of psoriatic nails. While LP nails have many changes, pitting is not typically listed as a common or characteristic one.)

Question 121:
The histological sign pathognomonic for lichen planus is:
a. Hypergranulosis
b. Hyperkeratosis
c. Papillomatosis
d. Parakeratosis
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Lichen Planus (Histological features), p.8 (from the "Connective tissue disease, Vasculitis, and Related Disorders" separate slide deck), and general LP histology):

From Lecture Slides - Connective Tissue Disease (Lichen Planus Histology, p.8):
Characterized by a band of lymphocytes attacking the basal keratinocytes which result in edema, subepidermal clefts, and death of some keratinocytes.

(Classic histology of LP also includes: Hyperkeratosis, irregular acanthosis (often "saw-tooth" rete ridges), ***hypergranulosis*** *(wedge-shaped), liquefaction degeneration of the basal cell layer, and a band-like lymphocytic infiltrate in the upper dermis. Civatte bodies (apoptotic keratinocytes) may be seen. Hypergranulosis is a very characteristic feature.)While not explicitly "pathognomonic" as the entire picture is needed, hypergranulosis is a key and prominent feature. Papillomatosis is more for warts. Parakeratosis is for psoriasis.*

Question 122:
All about lichen planus are true except:
a. Self limiting
b. 50% of cases clear within 18 months
c. Chronicity due to presence of meous lesion & hypertrophic lesions
(typo for "mucous" or "mucosal" lesions)
d. Presence of Wickham’s striate
e. Presence of hypogranulosis
Answer: E

High-Yield Context (from Dermatology Lecture Slides - Connective Tissue Disease, Vasculitis (Lichen planus), p.16, and Histology p.8 (separate slide deck)):

From Lichen planus (LP) (p.16):

- itchy eruption, shiny purple-coloured flat-topped papules...
- **White lines called Wickham’s striae** may appear on the surface of the lesions
- May affect the mouth, labia minora, nails or scalp
- Variants →

hypertrophic, bullous, oral LPFrom Lecture Slides - Connective Tissue Disease (Lichen Planus Histology, p.8 and general LP features):*(LP is often self-limiting, many cases resolve within 1-2 years. Mucosal and hypertrophic lesions tend to be more chronic. Wickham's striae are characteristic. Histologically, LP shows* ***hypergranulosis****, not hypogranulosis.)*

Question 123:
Not in lichen planus?

Nail thickening

High-Yield Context (from Dermatology Lecture Slides - Diseases of the Nails (Lichen planus), p.32):

From Lichen planus (p.32):

- **atrophy (thinning) of the nail plate**, pterygium...

(Lichen planus of the nails more commonly causes thinning and atrophy, not thickening. Thickening is more typical of onychomycosis or psoriasis (subungual hyperkeratosis).)

Question 124:
Itching is characteristic feature in:
a. Pityriasis rosea
b. Psoriasis
c. Lichen planus
d. Pityriasis versicolor
Answer: C

High-Yield Context (from various summary sections):

- **Pityriasis rosea (p.127 lecture slides):** herald patch, Christmas tree pattern, can be mildly itchy.
- **Psoriasis (p.5 summary, p.143 lecture slides):** "Psoriasis is usually only mildly itchy."
- **Lichen planus (p.16 summary):** "**itchy** eruption..." (one of the key "P"s: Pruritic).
- **Pityriasis versicolor (p.27 summary, p.110 lecture slides):** Often asymptomatic or mildly itchy.

(Lichen planus is classically very itchy.)

Question 125:
All of the following diseases are associated with macules as their primary lesions, except:
a. Lichen planus
b. Lentigo
c. Vitiligo
d. Erythema
e. Post inflammatory hyperpigmentation
Answer: A

High-Yield Context (from various summary sections):

- **Lichen planus (p.16 summary):** "shiny purple-coloured flat-topped **papules**" (primary lesion is a papule).
- **Lentigo (p.34 summary - freckles):** "small **macular** well-demarcated pigmented lesions"
- **Vitiligo (p.19 summary - hypopigmentation):** "sharply demarcated, symmetrical macular lesions"
- **Erythema (general term):** Can be macular (e.g., toxic erythema is a macular rash).
- **Post inflammatory hyperpigmentation (p.9 summary - eczema complication, p.18 - skin changes):** Results in macular changes of color.

(Lichen planus presents with papules as its primary lesion.)

Question 126:
Wrong statement about DLE (discoid lupus erythematosus)?
No scarring

High-Yield Context (from Dermatology Lecture Slides - Connective Tissue Disease, Vasculitis (Discoid lupus erythematosus), p.9 of separate slide deck, p.17 summary):

From Discoid lupus erythematosus (DLE) (p.9 of CTD slides, p.17 summary):

- photosensitive disorder, well-defined coin shaped erythematous lesions with **atrophy, scaling and scarring** occur on the face, scalp (alopecia, follicular plugging)
- Tx → potent and super-potent topical steroids to **limit scarring**

(DLE is well known to cause scarring, especially on the scalp (scarring alopecia) and face.)

Page 18 (Test Bank - Connective tissue disease / Cutaneous manifestations of systemic diseases cont.)

Question 127:
False about connective tissue disease: *
a. Subacute lupus causes cutaneous scarring
b. Discoid lupus will become systemic Lupus in <5%
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Connective Tissue Disease, Vasculitis (SCLE, DLE), p.9 of separate slide deck, p.17 summary):

From Subacute cutaneous lupus erythematosus (SCLE) (p.9 of CTD slides, p.17 summary):
- skin lesions that are scaly and evolve as polycyclic annular lesions or plaques, in sun-exposed areas.

*(SCLE is generally considered non-scarring, though it can leave dyspigmentation.)***From Discoid lupus erythematosus (DLE) (p.9 of CTD slides, p.17 summary):**
- photosensitive disorder, well-defined coin shaped erythematous lesions with

atrophy, scaling and scarring ...
- Risk of developing SLE→ 5%

(DLE causes scarring. SCLE is typically non-scarring. The statement that DLE progresses to SLE in <5% (or around 5%) is generally considered true.)

Question 128:
Not in lupus? *
Neonatal lupus develops into SLE in 20% of cases

High-Yield Context (from Dermatology Lecture Slides - Connective Tissue Disease, Vasculitis (Neonatal lupus erythematosus), p.9 of separate slide deck, p.17 summary):

From Neonatal lupus erythematosus (NLE) (p.9 of CTD slides, p.17 summary):
- transplacental passage of anti Ro/La
- Annular scaly lesions on the face/scalp
-

Risk congenital heart block*(NLE is caused by maternal autoantibodies. The skin rash is transient and resolves as maternal antibodies clear. The main serious risk is congenital heart block, which is permanent. NLE itself in the infant does not "develop into SLE". The mother may have SLE or another CTD, or be asymptomatic but positive for Ro/La antibodies.)*

Question 129:
Which of the following causes patchy scarring alopecia:
a. SLE
b. Discoid lupus
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Connective Tissue Disease, Vasculitis (DLE), p.9 of separate slide deck, p.17 summary):

From Discoid lupus erythematosus (DLE) (p.9 of CTD slides, p.17 summary):
- well-defined coin shaped erythematous lesions with atrophy, scaling and scarring occur on the face,

scalp (alopecia, follicular plugging)*(DLE is a classic cause of scarring alopecia.)*

Question 130:
Destruction of basal cell layer occurs in:
a. Discoid lupus erythromatosis
b. Morphea
c. Dermatomyositis
d. Psoriasis
e. Pityriasis rosea
Answer: A

High-Yield Context (from general histopathology of these conditions, as not explicitly detailed in the summary's short text for DLE):

- **Discoid lupus erythematosus:** Histology shows interface dermatitis with liquefaction degeneration (destruction/vacuolar alteration) of the basal cell layer, follicular plugging, and perivascular/periappendageal

lymphocytic infiltrates.

- **Morphea:** Sclerosis (thickening) of collagen in the dermis, epidermal atrophy may be present later.
- **Dermatomyositis:** Can also have an interface dermatitis similar to lupus, with basal cell damage.
- **Psoriasis:** Acanthosis, parakeratosis, neutrophils.
- **Pityriasis rosea:** Mild acanthosis, spongiosis, parakeratosis, superficial perivascular infiltrate.

(Both DLE and Dermatomyositis can feature basal cell layer damage (interface dermatitis). Given DLE is listed as 'a', it's a strong candidate. Dermatomyositis also fits.)The summary for DLE (p.17) doesn't detail histology, but the lecture slides it references (p.9 of CTD deck) would cover this. Liquefaction degeneration of the basal layer is key for DLE.

Question 131:
Commonest cutaneous eruption in SLE:
a. Erythema of light exposed areas
b. Butterfly rash
c. Discoid lesion
d. Erythema of palms
e. Diffuse multiform erythema
Answer: A
(The butterfly (malar) rash is very characteristic, but generalized photosensitivity (erythema of light exposed areas) is also extremely common and might be considered the "commonest eruption" in a broader sense including non-specific photosensitive rashes.)

High-Yield Context (from Dermatology Lecture Slides - Connective Tissue Disease, Vasculitis (Lupus erythematosus), p.8 of CTD deck, p.16 summary):

From Lupus erythematosus (LE) (p.8 of CTD deck, p.16 summary):
● 75% → skin involvement, most commonly an erythematous
‘butterfly’ distribution rash on the face
● SLE Dx (four of the following)
○
malar rash
○ discoid plaques
○
photosensitivity*(Malar rash is classic. Photosensitivity leading to erythema in light-exposed areas is also a diagnostic criterion and very common.)*

Question 132:
Which of the following is associated with muscular atrophy? *
a. Linear morphea
b. Pustular morphea
c. Diffuse morphea
d. Disseminated morphea
e. None of the above
Answer: E
(The OCR gives E. Morphea (localized scleroderma) involves sclerosis of the skin and subcutaneous tissue. If deep, it can involve underlying muscle and fascia, leading to muscle atrophy and joint contractures, especially in linear morphea (particularly "en coup de sabre" or affecting limbs). So, linear morphea (a) can be associated with muscular atrophy. "Pustular", "Diffuse", "Disseminated" are descriptors for morphea patterns, and deep involvement in any of these could theoretically affect muscle.)If the answer E ("None of the above") is correct, it implies none of the listed morphea types are associated, which is incorrect for deep/linear forms. This question may be problematic or based on a specific nuance not in the general summary.

High-Yield Context (from Dermatology Lecture Slides - Connective Tissue Disease, Vasculitis (Morphea), p.8 of CTD deck, p.16 summary):

From Morphea (p.8 of CTD deck, p.16 summary):
● benign localised systemic sclerosis, discoloured firm skin. typically in abdomen, chest or back
● If in head “frontoparietal area → (‘en coup de sabre’), alopecia and groove

(The summary is very brief. Deeper forms of morphea, especially linear morphea or pansclerotic morphea, can involve muscle and lead to atrophy.)

Question 133:
A girl with photosensitivity and ANA titer of 1:32, next step?
Repeat ANA in 3 months if sunscreen wasn't effective for the rash (?)

High-Yield Context (from Dermatology Lecture Slides - Connective Tissue Disease, Vasculitis (Lupus erythematosus), p.8 of CTD deck, p.16 summary and investigations p.15):

From Investigations (p.15):
2.1.
Antinuclear antibodies (ANAs)
2.2. Extractable nuclear antibodies (ENA), (Ro, La)

(A low positive ANA (1:32 is very low positive or borderline) with photosensitivity is non-specific. It could be early/mild CTD, drug-induced, or even seen in healthy individuals. Management would involve sun protection, symptomatic treatment for rash, and further specific autoantibody testing (ENA panel like anti-Ro, La, Sm, RNP; dsDNA) if CTD is suspected clinically. Simply repeating ANA in 3 months without further investigation if rash persists despite sunscreen might not be the most complete next step, but monitoring is part of it.)

Question 134:
Tissue involved in morphea may include:
a. Epidermis
b. Subcutaneous tissue
c. Muscles
d. Bones
e. All of the above
Answer: B
(The OCR shows B. Morphea primarily involves the dermis, but can extend to subcutaneous tissue. Deeper forms can involve fascia, muscle, and even bone. The epidermis is often secondarily affected (atrophy or hyperpigmentation). So, subcutaneous tissue (B) is definitely involved. If the answer is strictly B, it means the question considers muscle/bone involvement less typical or defines morphea as primarily dermal/subcutaneous.)

High-Yield Context (from general knowledge of morphea, as summary is brief):*Morphea is sclerosis of the skin. It starts in the dermis and can extend into the subcutaneous fat. Deep morphea (morphea profunda, pansclerotic morphea, linear morphea) can involve underlying fascia, muscle, and bone.Given the options, "All of the above" (e) would be true for some severe/deep forms. If the answer is B (Subcutaneous tissue), it's because that's a common extent beyond the dermis.*

Question 135:
Which is false about morphea:
a. It is localized form of scleroderma
b. It improves with time
c. Not caused by UV light exposure
d. Presents with as hairy well-defined patches
Answer: D

High-Yield Context (from Dermatology Lecture Slides - Connective Tissue Disease, Vasculitis (Morphea), p.8 of CTD deck, p.16 summary):

From Morphea (p.8 of CTD deck, p.16 summary):
●
benign localised systemic sclerosis (should be "localised scleroderma"), discoloured firm skin.
● If in head "frontoparietal area → ('en coup de sabre'),
alopecia and groove

(Morphea is localized scleroderma (a=true). Some forms can improve or "burn out" over time, but others are chronic or progressive (b=can be false). UV light (UVA1 phototherapy) is actually used as a treatment for some forms of morphea, suggesting UV itself is not a primary cause (c=true). Morphea lesions are typically characterized by induration and pigment changes, and can cause alopecia (hair loss) within the sclerotic plaques, not present as "hairy" patches (d=false).)

Question 136:
Childhood dermatomyositis is frequently ass. With:
a. CA
b. DM
c. Mental retardation
d. Calcinosis
Answer: D

High-Yield Context (from Dermatology Lecture Slides - Connective Tissue Disease, Vasculitis (Dermatomyositis), p.9 of separate slide deck, p.17 summary, and p.19 of general lecture slides):

From Dermatomyositis (p.9 of CTD deck, p.17 summary):
- skin, muscle “discomfort and weakness in proximal limbs” and blood vessels.
- Signs: Heliotrope rash, shawl sign & v sign, Gottron’s papules, Ragged cuticles and dilated nailfold

From Wrong statement (about calcinosis in adults, p.19 of general lecture slides):
"In dermatomyositis there is a risk of calcinosis in adults"
(Implying calcinosis is a feature. Calcinosis cutis is particularly common in juvenile dermatomyositis.)(Childhood (juvenile) dermatomyositis is frequently associated with calcinosis cutis. While adult dermatomyositis is associated with malignancy (CA), this is rare in childhood DM. DM is Diabetes Mellitus. Mental retardation is not a primary association.)

Page 19 (Test Bank - Connective tissue disease / Cutaneous manifestations of systemic diseases cont.)

Question 137:
All true about dermatomyositis except:
a. Frequently associated with underlying malignancy in adults
b. Affects children & adults
c. More common in males
d. Heliotrope rash is pathognomonic
Answer: C

High-Yield Context (from Dermatology Lecture Slides - Dermatomyositis, p.9 of CTD deck, p.17 summary):

From Dermatomyositis (p.9 of CTD deck, p.17 summary):
- skin, muscle “discomfort and weakness in proximal limbs” and blood vessels.
- Signs:

Heliotrope rash → purple hue on the upper & lower eyelids, cheeks and forehead.

(Dermatomyositis (DM) is associated with underlying malignancy in a significant proportion of adult cases (a=true). It affects both children (juvenile DM) and adults (b=true). Heliotrope rash and Gottron's papules are pathognomonic cutaneous features (d=true). DM has a female predominance, not more common in males (c=false).)

Question 138:
All of the following are seen in dermatomyositis except:
a. Gottron’s sign
b. Proximal muscle weakness
c. Ragged cuticle
d. Heliotrope sign
e. ... plaques
Answer: E
(The ellipsis indicates missing information for "plaques". Gottron's sign/papules, heliotrope rash, proximal muscle weakness, and ragged cuticles/dilated nailfold capillaries are all characteristic. "Plaques" is too vague; if it refers to Gottron's plaques (confluent papules), it's true.)

High-Yield Context (from Dermatology Lecture Slides - Dermatomyositis, p.9 of CTD deck, p.17 summary):

From Dermatomyositis - Signs (p.9 of CTD deck, p.17 summary):
-

Heliotrope rash → purple hue on the upper & lower eyelids, cheeks and forehead.
- shawl sign & v sign→ anterior ‘V’ and posterior “shawl “aspect of the neck
-

Gottron’s papules → dorsal surface of the fingers (esp. joints) may be affected by the erythematous eruption and purplish papules

- **Ragged cuticles and dilated nailfold**
- muscle “discomfort and **weakness in proximal limbs**”

Question 139:
One affect nail cuticle:
Dermatomyositis

High-Yield Context (from Dermatology Lecture Slides - Dermatomyositis, p.9 of CTD deck, p.17 summary):

From Dermatomyositis - Signs (p.9 of CTD deck, p.17 summary):

- **Ragged cuticles and dilated nailfold***(Periungual changes including ragged cuticles and dilated nailfold capillaries are characteristic of dermatomyositis.)*

Question 140:
Wrong statement:
In dermatomyositis there is a risk of calcinosis in adults

High-Yield Context (from previous question context and general knowledge):*Calcinosis cutis is a well-recognized complication of dermatomyositis, occurring in both adults and, more commonly, in children (juvenile DM). So, the statement "In dermatomyositis there is a risk of calcinosis in adults" is TRUE. The question asks for the "Wrong statement", so this provided statement itself (which is true) is part of a larger set of options in the original exam which is not fully captured here.*

Question 141:
Which one is a documented cause of erythema multiforme minor: *
a. Drugs
b. Pregnancy
c. DM
d. Herpes simplex labialis
e. Internal malignancy
Answer: D

High-Yield Context (from Dermatology Lecture Slides - The Skin and Systemic Disease (Erythema Multiforme), p.9 of separate slide deck, p.18 summary):

From EM (p.9 of Systemic Disease slides, p.18 summary):

- Raised erythematous macules, ‘target lesions’
- acral sites (palms, soles, digits, elbows, knees and face)
- immunologically mediated hypersensitivity reaction
- EM Major. → mucous membranes + cutaneous EM rash (<10% of body surface area)
- **Most common infectious trigger is herpes simplex virus (HSV 1 or 2) or cold sore**
  - Or Mycoplasma pneumonia or haemolytic Streptococcus
- **Adverse reactions to medications are also a common trigger for EM***(Herpes simplex virus is the most common cause of recurrent erythema multiforme minor. Drugs are also a common cause.)*

Question 142:
Which of following is the most common cause of Erythema multiforme: *
a. Herpes simplex virus
b. Mycoplasma
c. Pregnancy
Answer: A

High-Yield Context (from Dermatology Lecture Slides - The Skin and Systemic Disease (Erythema Multiforme), p.9 of separate slide deck, p.18 summary):

From EM (p.9 of Systemic Disease slides, p.18 summary):

- **Most common infectious trigger is herpes simplex virus (HSV 1 or 2) or cold sore**

Question 143:
Not a cause of erythema nodosum:
a. Pregnancy
b. Herpes simplex
Answer: B

High-Yield Context (from Dermatology Lecture Slides - The Skin and Systemic Disease (Erythema Nodosum), p.9 of separate slide deck, p.18 summary):

From Erythema Nodosum (EN) (p.9 of Systemic Disease slides, p.18 summary):
- tender/painful subcutaneous erythematous nodules on the shins
- inflammation in the adipose tissue
- Causes
  - Infectious causes of EN include

Streptococcus, Mycoplasma pneumoniae, TB
  - non-infectious triggers include

medications, inflammatory bowel disease, sarcoidosis, pregnancy, Behcet and Hodgkin disease.*(Herpes simplex is the most common cause of Erythema Multiforme, but it is not typically listed as a common cause of Erythema Nodosum. Pregnancy is a known cause of EN.)*

Question 144:
Which one mostly associated with underlying malignancy: *
a. Erythema nodosum
b. Erythema multiforme
c. Biological erythema
d. Erythema gyratum repens
e. Chemical erythema
Answer: A
(The OCR says A. The note says "please remember that erythema nodosum can also be caused by malignancies (Hodgkin and Non Hodgkin lymphoma, leukemia, renal cell carcinoma)". Erythema Gyratum Repens (d) is a classic paraneoplastic dermatosis, often described as "wood grain" pattern, strongly associated with underlying malignancy, especially lung cancer. Erythema nodosum can be associated with malignancy (e.g., lymphoma, leukemia) but also many other non-malignant causes. If EGR was an option, it would be a stronger association. Given the options and the OCR answer, EN is being considered.)

High-Yield Context (from Dermatology Lecture Slides - The Skin and Systemic Disease (Erythema Nodosum, Paraneoplastic syndromes), p.9 of separate slide deck, p.18 summary, and general knowledge):

From Erythema Nodosum (EN) (p.9 of Systemic Disease slides, p.18 summary):
- Causes: ...non-infectious triggers include ...

Hodgkin disease.From Skin manifestations of underlying malignancy (p.20 lecture slides):
Lists Acanthosis nigricans, Figurate erythemas, Pruritus, Dermatomyositis, Acquired ichthyosis.

(While EN can be paraneoplastic, Erythema Gyratum Repens is a more specific and stronger paraneoplastic marker if it were an option and clearly described.)

Question 145:
All condition may be precipitated by streptococcal throat infection except:
a. Erythema gyratum repens
b. Erythema marginatum
c. Erythema nodosum
d. Erythema multiforme
Answer: A

High-Yield Context (from various disease sections):

- **Erythema gyratum repens:** Classic paraneoplastic dermatosis. Not typically linked to strep.
- **Erythema marginatum:** Major Jones criterion for Rheumatic Fever, which is a post-streptococcal syndrome.
- **Erythema nodosum (p.18 summary):** Infectious causes include Streptococcus.
- **Erythema multiforme (p.18 summary):** Infectious triggers include Mycoplasma pneumonia or haemolytic Streptococcus (though HSV is most common).

(Erythema Gyratum Repens is primarily associated with malignancy.)

Question 146:
Which of the following where ulceration can occur (E=erythema):
a. E. nodosum
b. E. multiforme
c. E. repens
d. E. nodosum leprosum
e. E. annulare
Answer: D

High-Yield Context (from various disease sections):

- **E. nodosum (p.18 summary):** tender/painful subcutaneous erythematous **nodules**. Typically do not ulcerate, though can rarely.
- **E. multiforme (p.18 summary):** 'target lesions', can have central blistering or necrosis which can lead to erosions/ulceration in severe forms (EM Major, SJS).
- **E. repens (Erythema Gyratum Repens):** Annular, migratory erythematous eruption, not typically ulcerative.
- **E. nodosum leprosum (ENL):** Immune complex reaction in lepromatous leprosy, presents as tender erythematous nodules that **can ulcerate.**
- **E. annulare (Erythema Annulare Centrifugum) (p.18 summary):** single/ multiple erythematous expanding rings, asymptomatic. Not typically ulcerative.

(Erythema Nodosum Leprosum is well-known for its potential to ulcerate.)

Page 20 (Test Bank - Connective tissue disease / Cutaneous manifestations of systemic diseases cont.)

Question 147:
All about dermatitis herpetiformis except:
a. Chronic dis.
b. May occur at any age
c. Frequently associated with enteropathy
d. Autoimmune dis.
e. Prickle cell layer is the abnormal layer
Answer: E

High-Yield Context (from Dermatology Lecture Slides - Bullous Diseases (Dermatitis Herpetiformis), p.14 of summary, p.78 of lecture slides):

From Dermatitis Herpetiformis (DH) (p.14 summary, p.78 lecture):

- Intensely pruritic

autoimmune blistering disorder

- Affects

young/middle ages adults

- Associated with an underlying

gluten-sensitive enteropathy i.e. celiac disease

- IgA antibodies develop against tissue transglutaminase … and these cross-react with epidermal transglutaminase
- DH is a

chronic condition and therefore lifelong management is needed.

(Dermatitis Herpetiformis is an autoimmune disease associated with gluten-sensitive enteropathy (celiac disease). It's chronic. It typically affects young to middle-aged adults, less common at extremes of age. The abnormality is subepidermal blister formation due to IgA deposition at the dermo-epidermal junction targeting transglutaminases, not primarily a defect in the prickle cell layer, though inflammation affects the epidermis.)

Question 148:
Bullae of dermatitis herpetiformis are preceded histopathologically by:
a. Subepidermal microvacuoles
b. Neutrophilic & eosinophilic micro abscesses
c. Acantholysis of basal cell layer

d. Hydropic degeneration of basal layer
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Bullous Diseases (Dermatitis Herpetiformis - Histology), p.78 of lecture slides):

From Dermatitis Herpetiformis (DH) (p.78 lecture):

- **Histological finding: small vesicles containing neutrophils(micro-abscesses) in the upper dermis.** (specifically at the tips of dermal papillae)
- DIF: granular deposition of IgA in the upper dermis.

(Neutrophilic microabscesses in the dermal papillae are the characteristic early histological feature before overt blister formation.)

Question 149:
Celiac dis. May be ass. With:
a. Dermatitis herpetiformis
b. Pemphigus vulgaris
c. Bullous pemphigoid
d. Erythema multiforme
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Bullous Diseases (Dermatitis Herpetiformis), p.14 of summary, p.78 of lecture slides):

From Dermatitis Herpetiformis (DH) (p.14 summary, p.78 lecture):

- Associated with an underlying
gluten-sensitive enteropathy i.e. celiac disease

Question 150:
Treatment of dermatitis herpatiformis:
a. Diamino-diphenyl sulfone (DDS)
b. Systemic steroids
c. PUVA
d. Retinoic acid
Answer: A (=dapsone)

High-Yield Context (from Dermatology Lecture Slides - Bullous Diseases (Dermatitis Herpetiformis - Management), p.14 of summary, p.78 of lecture slides):

From Dermatitis Herpetiformis (DH) - Management (p.14 summary, p.78 lecture):

1. Strict gluten-free diet:
2. **Dapsone** or sulphapyridine
3. DH is a chronic condition and therefore lifelong management is needed.

(Dapsone (diamino-diphenyl sulfone) is the mainstay of pharmacological treatment for DH, providing rapid relief of itching and lesions. A gluten-free diet is essential for long-term management and to address the underlying celiac disease.)

Question 151:
Occurs in vitiligo:
a. Destruction of melanocytes
b. Abnormal melanin synthesis
c. Abnormal tyrosinase enzyme
d. All of the above
e. None of the above
Answer: A

High-Yield Context (from Dermatology Lecture Slides - The Skin and Systemic Disease (Hypopigmentation - Vitiligo), p.10 of Systemic Disease slides, p.19 summary/p.44 lecture):

From Hypopigmentation - Vitiligo (p.10 Systemic Disease slides, p.19 summary/p.44 lecture):

- Localised

depigmentation, sharply demarcated, symmetrical, acquired, **destruction of melanocytes.***(Vitiligo is characterized by the autoimmune destruction of melanocytes, leading to a loss of melanin production in affected areas.)*

Question 152:

Which is false about vitiligo:

a. If affects males and females equally

b. Onset usually in 20s and 30s

c. It results in HYPOpigmented patches

d. Result from destruction of melanocytes

Answer: C

High-Yield Context (from Dermatology Lecture Slides - The Skin and Systemic Disease (Hypopigmentation - Vitiligo), p.10 of Systemic Disease slides, p.19 summary/p.44 lecture):

From Hypopigmentation - Vitiligo (p.10 Systemic Disease slides, p.19 summary/p.44 lecture):

- Localised

depigmentation (complete loss of pigment), sharply demarcated, symmetrical, acquired, destruction of melanocytes.

(Vitiligo results in depigmented (white) patches due to complete loss of melanocytes, not hypopigmented (lighter than normal but still some pigment). It affects males and females equally. Onset can be at any age, but often in the 20s and 30s. It is due to destruction of melanocytes.)

Question 153:

False about vitiligo: *

a. Male : female (1:1)

b. Associated with thyroiditis

c. Peak age of incidence is 20-30s

d. It is a disease of abnormal melanisation

Answer: D

High-Yield Context (from Dermatology Lecture Slides - The Skin and Systemic Disease (Hypopigmentation - Vitiligo), p.10 of Systemic Disease slides, p.19 summary/p.44 lecture):

From Hypopigmentation - Vitiligo (p.10 Systemic Disease slides, p.19 summary/p.44 lecture):

- Localised depigmentation...

destruction of melanocytes.

- Autoimmune associations →

Thyroid disease, Myasthenia gravis, Pernicious anemia, Alopecia areata, Hypoparathyroidism, Addison’s disease, DM

(Vitiligo has equal M:F incidence (a=true). It is associated with autoimmune thyroiditis (b=true). Peak onset is often 20s-30s (c=true). It is a disease of loss of melanocytes leading to lack of melanisation, not primarily "abnormal melanisation" by existing melanocytes (d=false).)

Question 154:

No. of melanocytes in vitiligo is:

a. Decrease

b. Increase

c. Normal

d. All of the above

Answer: A

High-Yield Context (from Dermatology Lecture Slides - The Skin and Systemic Disease (Hypopigmentation - Vitiligo), p.10 of Systemic Disease slides, p.19 summary/p.44 lecture):

From Hypopigmentation - Vitiligo (p.10 Systemic Disease slides, p.19 summary/p.44 lecture):

- Localised depigmentation...

destruction of melanocytes.*(In vitiliginous patches, melanocytes are absent or markedly reduced due to their destruction.)*

Question 155:

Vitiligo is significantly ass. With:

a. Hypopituitary
b. Hyperthyroidism
c. Hypothyroidism

(OCR ends, answer not visible but likely C or B as thyroid autoimmunity is common.)

High-Yield Context (from Dermatology Lecture Slides - The Skin and Systemic Disease (Hypopigmentation - Vitiligo), p.10 of Systemic Disease slides, p.19 summary/p.44 lecture):

From Hypopigmentation - Vitiligo (p.10 Systemic Disease slides, p.19 summary/p.44 lecture):

- Autoimmune associations →

Thyroid disease (both hyper- e.g., Graves', and hypo- e.g., Hashimoto's thyroiditis), Myasthenia gravis, Pernicious anemia, Alopecia areata, **Hypoparathyroidism**, Addison's disease, DM.

(Both hyperthyroidism and hypothyroidism due to autoimmune thyroid disease are associated with vitiligo.)

Page 21 (Test Bank - Connective tissue disease / Cutaneous manifestations of systemic diseases cont.)

Question 156 (continued from Page 20, question 9d):
Vitiligo is significantly ass. With:
d. Hypoparathyroidism
Answer: C
(The question as presented ends with c. Hypothyroidism. From the context above, vitiligo is associated with autoimmune thyroid disease (hypo or hyper), pernicious anemia, Addison's, alopecia areata, DM, and hypoparathyroidism. If C (Hypothyroidism) was the chosen answer, it is a correct association.)

High-Yield Context: (Already covered under Page 20, Question 9).

Question 157:
Vitiligo may be associated with all except: *
a. Thyrotoxicosis
b. Pernicious anemia
c. Addison's dis.
d. Gastric dis.
e. Reticulosis
Answer: E

High-Yield Context (from Dermatology Lecture Slides - The Skin and Systemic Disease (Hypopigmentation - Vitiligo), p.10 of Systemic Disease slides, p.19 summary/p.44 lecture):

From Hypopigmentation - Vitiligo (p.10 Systemic Disease slides, p.19 summary/p.44 lecture):

- Autoimmune associations →

Thyroid disease (Thyrotoxicosis e.g. Graves'), **Pernicious anemia**, Alopecia areata, Hypoparathyroidism, **Addison's disease**, DM.

(Reticulosis refers to proliferative disorders of the reticuloendothelial system, like lymphomas. While some lymphomas can have cutaneous manifestations or be associated with paraneoplastic syndromes, vitiligo is primarily linked to other autoimmune conditions, not directly to reticulosis as a common association.)(Gastric disease like pernicious anemia (autoimmune gastritis) is an association.)

Question 158:
Piebaldism:
Autosomal dominant

High-Yield Context (from general knowledge, as not detailed in the hypopigmentation section of the summary which focuses on vitiligo and post-inflammatory. Piebaldism is mentioned in the lecture slide on p.44 under "Hypopigmentation"):

From Lecture Slides - Hypopigmentation (p.44):Piebaldism:

-

AD, triangular hypopigmented patches at the forehead and white forelock, disorder of melanocyte development.

(Piebaldism is an autosomal dominant condition characterized by congenital patches of depigmented skin and hair, most classically a white forelock, due to absent melanocytes in affected areas resulting from mutations in the KIT gene.)

Question 159:
Causes of post inflammatory hypopigmentation:
a. Psoriasis
b. Lichen planus
c. All of the above
Answer: C

High-Yield Context (from Dermatology Lecture Slides - The Skin and Systemic Disease (Hypopigmentation), p.10 of Systemic Disease slides, p.19 summary/p.44 lecture):

From Hypopigmentation - Post Inflammatory (p.10 Systemic Disease slides, p.19 summary/p.44 lecture):
- Post Inflammatory conditions such as

psoriasis, eczema, lichen planus and lupus erythematosus; infections, tuberous sclerosis (‘ash leaf’ macules).

(Both psoriasis and lichen planus are listed as causes of post-inflammatory hypopigmentation.)

Question 160:
ttt of choice for chloasma:
a. Salicylic acid skin ointment
b. Phenol lotion
c. Eldoquin ointment
d. 5-PU 0.1%
Answer: C
(Eldoquin is a brand name for hydroquinone, a common topical skin-lightening agent used for melasma/chloasma.)

High-Yield Context (from general knowledge on melasma treatment, as specific treatments for chloasma/melasma are not detailed in the provided summary sections on hyperpigmentation which are very brief p.18/p.45):*Standard treatments for melasma (chloasma) include sun protection, topical hydroquinone, topical retinoids, azelaic acid, and sometimes chemical peels or lasers. Phenol peels are very strong and carry risks. 5-PU (psoralen) is used for PUVA photochemotherapy, which would worsen hyperpigmentation. Salicylic acid is a keratolytic.*

Question 161:
Commonest cutaneous lesions of Hodgkin’s disease is: *
a. Tumors
b. Secondary to pruritus
c. Exfoliative erythroderma
d. Ichthyosis
e. Ulcers
Answer: B

High-Yield Context (from Dermatology Lecture Slides - The Skin and Systemic Disease (Skin manifestations of underlying malignancy), p.10 of Systemic Disease slides, p.20 summary):

From Skin manifestations of underlying malignancy (p.10 Systemic Disease slides, p.20 summary):
- Non-specific skin changes associated with malignant disease
  - …
  -

Generalized pruritus (particularly lymphoma)
  - Figurate erythema
  - Superficial thrombophlebitis.
- Acquired ichthyosis→

Hodgkin’s disease, sarcoma, lymphoma

(Generalized pruritus is a very common non-specific cutaneous manifestation of Hodgkin's disease. The skin changes are often secondary to scratching (excoriations, lichenification). Acquired ichthyosis can also occur. Specific tumor infiltrates ("tumors") are less common than these reactive changes.)

Bacterial infections

Question 162:
Which of the following is the most superficial infection of the skin: *

a. Ecthyma
b. Impetigo
c. Cellulites
d. Furuncles
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Bacterial skin infections (Superficial vs Deeper), p.23 summary, p.115-116 lecture):

From Bacterial Skin Infections - Classification (p.23 summary, p.115 lecture):

•
Superficial Bacterial Infections:
-
Impetigo (staph, strep)
- Bacterial folliculitis (staph)
- Boils (Abscesses) (staph) (
Note: Boils/Furuncles involve the hair follicle and extend deeper than just the very superficial epidermis, so their classification as purely "superficial" can be debated compared to non-follicular impetigo.)
- Pseudofolliculitis
- Erythrasma (corynobacterium)

•
Deeper Bacterial Infections:
- Erysipelas (strep)
-
Cellulitis (strep)
- Necrotizing Fasciitis (Mixed aerobic and anaerobic)
- Staphylococcus Scalded Skin Syndrome (staph)
-
Ecthyma (strep) (*Ecthyma is a deeper form of impetigo, an ulcerative pyoderma that extends into the dermis.*)

(Impetigo (non-bullous) is a very superficial infection of the epidermis, often subcorneal. Ecthyma is deeper, ulcerating into the dermis. Cellulitis is infection of the dermis and subcutaneous tissue. Furuncles are deep follicular infections.)

Question 163:
Which one cause impetigo contagiosum:
Staph & strep

High-Yield Context (from Dermatology Lecture Slides - Bacterial skin infections (Impetigo), p.23 summary, p.116 lecture):

From Superficial infections - Impetigo (p.23 summary, p.116 lecture):
- Is usually caused by

S. aureus or **Streptococcus pyogenes.**

Question 164:
Impetigo may occur in: *
a. Elderly
b. Infants
c. Adult
d. Young adults
Answer: B
(Impetigo is common in children, especially infants and young children, due to close contact and minor skin trauma, though it can occur at any age.)

High-Yield Context (from general knowledge of impetigo epidemiology. The summary does not specify age groups but describes it as common and contagious.):*Impetigo is most prevalent in children aged 2-5 years but can affect any age group, particularly in conditions of poor hygiene, crowding, or warm climates.*

Question 165:
Coral red in wood’s light:
Erythrasma

High-Yield Context (from Dermatology Lecture Slides - Bacterial skin infections (Erythrasma), p.23 summary, p.118 lecture):

From Superficial infections - Erythrasma (p.23 summary / p.118 lecture):

- flexural skin sites, Corynebacterium minutissimum,
-

Wood's ultraviolet light → fluoresces pink. (Classically coral-pink or coral-red).

Question 166:
Patient presented with WELL defined erythematous plaque on the calf of her lower limb. U/S was done to assess for blood flow, result was normal, what is the most likely diagnosis:
a. Erysipelas
b. Cellulitis
c. DVT
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Bacterial skin infections (Erysipelas vs Cellulitis), p.23 summary, p.118-119 lecture):

From Deeper Infections - Erysipelas (p.23 summary / p.118 lecture):

- Caused by a Streptococcus infection
- Over approximately 48 h the inflammation spreads across the skin with a characteristic red, shiny, raised, spreading plaque with a

well-demarcated edge.

- The face and lower legs are most frequently affected

From Deeper Infections - Cellulitis (p.23 summary / p.119 lecture):

- Develops more slowly than erysipelas and has a

poorly defined margin and marked regional lymphadenopathy.

(A well-defined, raised, erythematous plaque is characteristic of erysipelas. Cellulitis has more diffuse, poorly demarcated borders. Normal U/S for blood flow helps rule out DVT.)

Question 167:
Erysipelas, all true except:
a. Well-defined
b. Can be with fever
c. Penicillin is the drug of choice
d. Caused by staph.
e. Mostly on L.L
Answer: D

High-Yield Context (from Dermatology Lecture Slides - Bacterial skin infections (Erysipelas), p.23 summary, p.118-119 lecture):

From Deeper Infections - Erysipelas (p.23 summary / p.118-119 lecture):

- Caused by a

Streptococcus infection

- ...characteristic red, shiny, raised, spreading plaque with a

well-demarcated edge.

- Occasionally, blistering may occur at the active edge; patients may have

fever and malaise.

- The face and

lower legs are most frequently affected

- Treatment (p.119): If the infection is severe, treat with: Intravenous benzylpenicillin; Orally with amoxicillin...

(Erysipelas is primarily caused by Streptococcus pyogenes (Group A Strep), not Staphylococcus, although staph can cause cellulitis or co-infect.)

Question 168:
Not a superficial skin infection?
Ecthyma

High-Yield Context (from Dermatology Lecture Slides - Bacterial skin infections (Classification), p.23 summary, p.115 lecture):

From Bacterial Skin Infections - Classification (p.23 summary, p.115 lecture):

- Superficial Bacterial Infections:
- Impetigo ...
-

Deeper Bacterial Infections:

- Erysipelas (strep)
- Cellulitis (strep)
- ...
-

Ecthyma (strep)

(Ecthyma is an ulcerative form of pyoderma that extends into the dermis, hence considered a deeper infection than superficial impetigo.)

Question 169:
Wrong about ecthyma:
a. Superficial infection
b. Causes generalized dryness
c. Strep is the most common cause
d. Increased in immunocompromised
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Bacterial skin infections (Ecthyma), p.23 summary, p.120 lecture):

From Deeper Infections - Ecthyma (p.23 summary / p.120 lecture):

- Is often referred to as a

deeper form of impetigo as the group Aβ-hemolytic **Streptococci (S. pyogenes)** invade the dermis leading to **superficial ulcers.**

- Lesions start as small pustules that have adherent crust and underlying ulceration
- Most commonly occur on the lower legs of children and elderly people who live in humid climates.
- Lesions usually heal slowly with scarring

(Ecthyma is a deeper infection (ulcerative), not superficial. It's typically caused by Strep, can be more common or severe in immunocompromised individuals or with poor hygiene. "Generalized dryness" is not a primary feature of ecthyma itself, which is a localized ulcerative lesion.)

Viral infections

Question 172: Commonest rash of secondary syphilis:
a. Vesicular
b. Maculo-papular
c. Papular
d. Pustular bullous
Answer: B

High-Yield Context (from Dermatology Lecture Slides - STDs (Syphilis - Secondary), p.40 of lecture slides):

From Secondary syphilis (p.40 lecture):

- Early sign of secondary syphilis is Rose colored

macules usually on shoulders and flanks

- Then

papulosquamous lesions appear.

- palmoplantar manifestation (Copper- pin spots, bronze colored, surrounded by collarette scales) this stage is highly contagious.

(Secondary syphilis classically presents with a widespread maculopapular or papulosquamous rash.)

Question 171:
Rash of secondary syphilis is:
a. Scaly
b. Itchy
c. Vesicular
d. None of the above
Answer: A

High-Yield Context (from Dermatology Lecture Slides - STDs (Syphilis - Secondary), p.40 of lecture slides):

From Secondary syphilis (p.40 lecture):
- widespread eruption of red-brown

scaly patches and macules that affects the trunk and limbs (particularly palms and soles)
- Then

papulosquamous lesions appear.

(The rash of secondary syphilis is typically non-itchy (though some pruritus can occur) and is often scaly, especially in its papulosquamous form. It is not primarily vesicular.)

Question 172:
Main local source of staph. pyogenes contaminating the skin:
a. Nose

(OCR for options ends, answer implies A. The question likely means Staphylococcus aureus, as Streptococcus pyogenes is a different bacterium. The anterior nares are the primary reservoir for S. aureus.)

High-Yield Context (from Dermatology Lecture Slides - Bacterial skin infections (Investigations - Nasal swabs), p.23 summary, p.114 lecture):

From Investigations - Nasal swabs (p.23 summary / p.114 lecture):
- May identify

Staphylococcus aureus carriers (MRSA) who can suffer from recurrent infections because of bacterial shedding from the **nose.***(The anterior nares are the main carriage site for Staphylococcus aureus.)*

Page 23 (Test Bank - Bacterial infections cont.)

Question 173 (continued from Page 22, question 11):
Main local source of staph. pyogenes contaminating the skin:
a. Nose
b. Scalp
c. Axillae
d. Perineum
e. Mouth
Answer: A
(Assuming the question meant Staphylococcus aureus, the nose (anterior nares) is the main reservoir. Streptococcus pyogenes is often carried in the throat or on the skin.)

High-Yield Context: (Already covered under Page 22, Question 11, with the clarification that the question likely refers to *S. aureus* whose primary reservoir is the anterior nares.)

Page 24 (Test Bank - Viral infections)

Question 174:
Commonest form of recurrent herpes simplex is:
a. Herpes labialis
b. Herpetic whitlow
c. Herpetic conjunctivitis
d. Eczema herpeticum
e. Marginal keratitis
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Herpes Simplex), p.24 summary, p.124-125 lecture):

From Herpes Simplex 1 (p.124 lecture):

- Usually occurs in or

around the mouth/nose, with variable involvement of the face.

- Lesions consist of small vesicles which crust over and are associated with regional lymphadenopathy.

From Herpes Simplex (p.125 lecture):

- Episodes of reactivation of HSV may be triggered by the

cold ('cold sore')...

- Recurrent HSV-1 infections of the

lips and perioral area are estimated to occur in 20 to 40 percent of the infected population.

(Herpes labialis (cold sores/fever blisters) is the most common manifestation of recurrent HSV-1 infection.)

Question 175:

False about herpes genitalia:

Patient should be symptomatic to be contagious

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Herpes infection - Latent infection & Reactivation), p.39 of STD lecture slides, p.124 general Herpes Virus):

From Herpes infection (Latent infection & Reactivation) (p.39 of STD lecture slides):

2nd or reactivation infection

A-

Asymptomatic (virus in shedding phase, most of transmission happens during this stage)

B- Symptomatic.

(Asymptomatic viral shedding is common with HSV, meaning individuals can be contagious even without visible lesions or symptoms.)

Question 176:

In herpes zoster, all are true except:

a. Pain may precede the appearance of rash

b. The rash is vesicular

c. Commonly bilateral

d. Frequently associated with underlying malignancy

e. More serious in elderly pt

Answer: C

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Varicella Zoster Virus - Shingles), p.24 summary, p.126-127 lecture):

From Varicella Zoster Virus - Shingles (p.24 summary, p.126-127 lecture):

- In shingles,

pain, fever, and malaise may precede the rash (a=true)

- ...characterized usually by its

dermatomal distribution... (implies unilateral) (c=false)

- Erythematous papules usually precede

vesicles which develop over several days... (b=true)

- In the context of HIV, shingles lesions may be multi-dermatomal, extensive and hemorrhagic. (d=true in immunocompromised, malignancy is a risk factor for zoster)
- Occasionally, peripheral motor neuropathy can result and a proportion of patients develop severe chronic postherpetic neuralgia.

(More common and severe in elderly) (e=true)

(Herpes zoster is characteristically unilateral, following a dermatome. Bilateral zoster is very rare and usually suggests significant immunosuppression.)

Question 177:

Wrong about shingles? *

Treated with topical acyclovir

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Varicella Zoster Virus Management), p.24 summary, p.127 lecture):

From Varicella Zoster Virus Management (p.24 summary, p.127 lecture):

- Patients ideally should receive

high-dose aciclovir for 7 days within 72 hrs of the onset of the eruption.

- If the eye is affected or there is nerve compression, then

intravenous aciclovir should be considered...

(Systemic antiviral therapy (oral or IV acyclovir, valacyclovir, famciclovir) is the standard treatment for shingles to reduce severity, duration, and risk of complications like postherpetic neuralgia. Topical acyclovir is used for herpes simplex labialis but is not adequate for shingles.)

Question 178:

Shingles, all true except:

a. Oral and topical steroids are frequently used

b. Postherpetic neuralgia can last for months

c. Its reactivation of varicella zoster virus

Answer: A

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Varicella Zoster Virus - Shingles, Management), p.24 summary, p.126-127 lecture):

From Varicella Zoster Virus - Shingles & Management (p.24 summary, p.126-127 lecture):

- Subsequently

shingles (reactivation) may occur as the virus remains latent in the sensory nerve ganglia. (c=true)

- Occasionally, peripheral motor neuropathy can result and a proportion of patients develop severe chronic

postherpetic neuralgia. (can last for months to years) (b=true)

- Patients ideally should receive high-dose

aciclovir... If the eye is affected...patients may require **systemic steroids** to prevent nerve paralysis in severe cases.

(Systemic steroids are sometimes used in specific situations (e.g., Ramsay Hunt syndrome, ophthalmic zoster) to reduce inflammation and pain, but not "frequently used" as a general rule for all shingles cases, and their benefit for preventing PHN is debated. Topical steroids are generally not indicated for acute zoster lesions unless there's a significant eczematous component, and even then, with caution.)

Question 179:

Wrong about systemic ttt of H. zoster:

a. Immunodeficiency

b. > 50-year-old

c. Peripheral N involvement

d. More than one dermatome

answer: C

(Systemic treatment is indicated for peripheral nerve involvement to reduce nerve damage and risk/severity of PHN. Options a, b, and d are all indications/risk factors for more severe disease or complications, warranting systemic treatment.)

High-Yield Context (from general guidelines for shingles treatment, as specific indications for systemic treatment beyond "high-dose aciclovir" are not detailed in this summary beyond ophthalmic/nerve compression cases):*Indications for systemic antiviral treatment in herpes zoster include: age >50 years, moderate to severe pain, moderate to severe rash, involvement of head or neck (including ophthalmic zoster, Ramsay Hunt), immunocompromised status, and involvement of more than one dermatome (disseminated zoster). Peripheral nerve involvement (e.g. motor neuropathy, severe neuralgia) is a reason for aggressive treatment, including systemic antivirals and sometimes steroids, not a reason against it.*

Question 180:

Ask about family history in all except:

a. Pityriasis rosea

b. Scabies

c. Psoriasis

d. Atopic dermatitis

e. Vitiligo
Answer: A

High-Yield Context (from various summary sections):

- **Pityriasis rosea (p.127 lecture):** Thought to be triggered by viral infection (HHV-6/7). Not typically familial.
- **Scabies (p.88 lecture):** Highly contagious, often affects multiple family members/close contacts. Family history is very relevant.
- **Psoriasis (p.141 lecture):** Strong genetic predisposition, family history is common.
- **Atopic dermatitis (p.157 lecture):** Strong genetic component, personal or family history of atopy is key.
- **Vitiligo (p.44 lecture):** Autoimmune, can have familial clustering and association with other autoimmune diseases in family members.

(Pityriasis rosea is generally considered a sporadic, likely viral-triggered exanthem and not primarily a familial/genetic condition for which family history is a key diagnostic component.)

Question 181:
A rash that is not primarily macular?
Pityriasis rosea

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Pityriasis Rosea), p.24 summary, p.127 lecture):

From Pityriasis rosea (PR) (p.24 summary, p.127 lecture):
- PR classically presents with an initial single annular erythematous

patch with a collarette of scale – the **herald patch.**
- The rest of the rash consists of multiple smaller scaly

patches on the trunk, upper arms, and thighs

(A patch is a large macule (>1cm). The lesions of PR are typically patches or plaques (slightly elevated patches), often oval, with fine scale. So it is primarily composed of patches/plaques, which are either large macules or slightly raised, not just simple macules like freckles.)The question is "not primarily macular". While patches are large macules, PR lesions often have some degree of scale and can be slightly palpable/plaque-like.

Question 182:
One is false about pityriasis rosea: *
a. Cause herald patches
b. Very itchy rash
c. Self-limiting
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Pityriasis Rosea), p.24 summary, p.127-128 lecture):

From Pityriasis rosea (PR) (p.24 summary, p.127-128 lecture):
- PR classically presents with an initial single annular erythematous patch with a collarette of scale – the

herald patch. (a=true)
- The rash

settles spontaneously over about 4–6 weeks, but a mild topical steroid and emollient can be given if the rash is **pruritic or inflammatory.** (c=true)

(Pityriasis rosea can be itchy, but often it's mild to moderate. "Very itchy rash" may be an exaggeration or less typical than seen in, for example, severe eczema or scabies. Some cases are not very itchy at all.)

Question 183:
Patient presented with mild itchy erythematous patches on the trunk, neck and upper limb with collaret scales, what is the diagnosis?
Pityriasis rosea

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Pityriasis Rosea), p.24 summary, p.127 lecture):

From Pityriasis rosea (PR) (p.24 summary, p.127 lecture):
- PR classically presents with an initial single annular erythematous

patch with a **collarette of scale** – the herald patch.

- The rest of the rash consists of multiple smaller scaly

patches on the **trunk, upper arms, and thighs** (old-fashioned bathing suit distribution).

- On the back, the lesions may follow the angle of the ribs in a

'Christmas tree pattern'.*(This is a classic description of Pityriasis Rosea.)*

Question 184:

Pityriasis rosea wrong:

a. Mild non itchy usually

b. Rash before herald lesion

c. Self-limiting

Answer: B

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Pityriasis Rosea), p.24 summary, p.127-128 lecture):

From Pityriasis rosea (PR) (p.24 summary, p.127-128 lecture):

- PR classically presents with an

initial single annular erythematous patch with a collarette of scale – the **herald patch.** (The herald patch *precedes* the generalized rash.) (b=false)

- The rash settles spontaneously over about 4–6 weeks... if the rash is

pruritic or inflammatory. (Implies it can be non-itchy or mildly itchy) (a=true)

- The rash

settles spontaneously over about 4–6 weeks... (c=true)

(The herald patch appears first, followed by the generalized rash. So "Rash before herald lesion" is wrong.)

Page 25 (Test Bank - Viral infections cont.)

Question 185:

The cause of pityriasis rosea: *

a. HSV 6

b. HSV7

c. HSV1,2

d. a + b

e. a + b + c

Answer: D

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Pityriasis Rosea), p.24 summary, p.127 lecture):

From Pityriasis rosea (PR) (p.24 summary, p.127 lecture):

- Pityriasis rosea (PR) has been thought recently to be triggered by an upper respiratory tract infection with

human herpes virus type 6 or 7.

Question 186:

Associated with HSV 6&7:

a. Pityriasis rosea

b. Lichen planus

Answer: A

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Pityriasis Rosea), p.24 summary, p.127 lecture):

From Pityriasis rosea (PR) (p.24 summary, p.127 lecture):

- Pityriasis rosea (PR) has been thought recently to be triggered by an upper respiratory tract infection with

human herpes virus type 6 or 7.

Question 187:

Herald patch is a specific lesion for: *

a. Pityriasis alba

b. P. versicolor

c. P. rosea

d. P. capitis

e. P. rubra pilaeis

Answer: C

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Pityriasis Rosea), p.24 summary, p.127 lecture):

From Pityriasis rosea (PR) (p.24 summary, p.127 lecture):

- PR classically presents with an initial single annular erythematous patch with a collarette of scale – the **herald patch.**

Question 188:
Which is the causative of molluscum contagiosum: *
Pox virus

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Poxviruses - Molluscum Contagiosum), p.24 summary, p.128 lecture):

From Poxviruses (p.24 summary, p.128 lecture):

- Pox viruses are large DNA viruses, with a predilection for the epidermis.
-

Molluscum Contagiosum and Orf are also **pox viruses.From Molluscum Contagiosum (p.24 summary, p.128 lecture):**

- Caused by

Molluscum Contagiosum virus. (which is a type of poxvirus)

Question 189:
Wrong about plain warts? *
Should always be treated because they don't resolve by themselves

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Wart viruses - Treatment), p.26 summary, p.131 lecture):

From Treatment (of warts) (p.26 summary, p.131 lecture):

- Warts commonly occur in childhood and

usually resolve spontaneously.

- There are however numerous treatment options available.

(Many warts, especially in children, resolve spontaneously without treatment over months to years.)

Question 190:
Warts most commonly affect body flexures:
a. Plane warts
b. Common warts
c. Digitate warts
d. Filiform warts
e. Seborrheic warts
Answer: D
(The answer key says D, Filiform warts. Filiform warts often occur on the face, neck, and eyelids. Body flexures are not their most common site. Common warts are frequent on hands and feet. Plane warts on face, hands, shins. Plantar warts on soles. This is a tricky question and depends on what is meant by "most commonly affect". Filiform warts are common on the face/neck; perhaps the "flexures" of the neck are implied for D to be correct. Seborrheic warts (keratoses) are not true viral warts.)

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Wart viruses - Types), p.25-26 summary, p.130 lecture):

From Wart viruses (p.26 summary, p.130 lecture, image shows types):

- Periungual (around nails)
-

Filiform (often face, neck)

- Common (verruca vulgaris, often hands)
- Plantar (soles)
- Flat (plane) (often face, hands)

(The summary doesn't detail specific site predilections for each type beyond general areas. Filiform warts are typically on the face, eyelids, neck, lips. Flexures are not their prime location.)

Question 191:
Not a treatment for viral warts:

a. Cryotherapy
b. 5FU
c. Salicylic acid
d. Topical steroids
Answer: D

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Wart viruses - Treatment), p.26 summary, p.131 lecture):

From Treatment (of warts) (p.26 summary, p.131 lecture):

•
Salicylic and lactic acids in various formulations...
- Gels/ointments/paints/lotions should be applied daily after paring down the wart surface.
- Duct tape...
- For large/painful/recalcitrant warts other measures may be considered.

*
Liquid nitrogen (cryotherapy)
* Carbon dioxide 'snow'
* Diathermy loop cautery
* Podophyllin (for perianal warts)
* Immune response modifier Imiquimod® (for genital warts)

(5-FU (Fluorouracil) is used off-label for some warts, especially recalcitrant ones. Cryotherapy and salicylic acid are standard treatments. Topical steroids are not used to treat warts and can potentially worsen them by suppressing local immunity.)

Question 192:
Wrong about common wart:
a. If not treated majority will turn to skin cancer
b. Caused by dsDNA
c. Common in children
d. Majority will resolve spontaneously
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Wart viruses), p.25-26 summary, p.129-131 lecture):

From Wart viruses (p.25 summary, p.129-130 lecture):
- (p.129) More than 100 different subtypes of

HPV have currently been identified. (HPV is a DNA virus - dsDNA) (b=true)
- (p.129) HPV subtypes 6 and 11 are responsible for the majority of genital warts and subtypes 16/18 with the development of cervical/anal/vulval/vaginal/oral carcinomas.

*(This implies some HPV types are oncogenic, but common cutaneous warts caused by non-oncogenic types very rarely turn into skin cancer in immunocompetent individuals.)***From Treatment (p.26 summary, p.131 lecture):**
- Warts commonly occur in

childhood (c=true) and usually **resolve spontaneously.** (d=true)

(Malignant transformation of common cutaneous warts into skin cancer is extremely rare in immunocompetent individuals. Certain HPV types in specific locations (e.g., genital, or in immunosuppressed patients like epidermodysplasia verruciformis) have oncogenic potential, but not common warts typically.)

Question 193:
All of the following are true about plane warts except:
a. Occurs most commonly in the face
b. Spiky top
c. Different colors
d. Children and adolescents
e. Koebner's phenomena
Answer: B

High-Yield Context (from general knowledge of plane warts, as specific morphology not detailed beyond "Flat (plane)" in the summary image on p.130):*Plane warts (verruca plana) are characteristically flat-topped or slightly elevated,*

smooth papules, often skin-colored or light brown. They are common on the face, hands, and shins, especially in children and adolescents. They can exhibit Koebner phenomenon (spread along lines of trauma). "Spiky top" is more characteristic of filiform warts or some common warts, not plane warts.

Question 194:
Plantar warts, all true except:
a. Most common in children
b. Smooth surface
c. The most common type of warts
d. Fleshy, pink and greyish
Answer: C
(Common warts (verruca vulgaris) on hands are generally considered the most common type overall. Plantar warts are common, but perhaps not the most common type globally compared to hand warts.)

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Wart viruses - Plantar warts), p.26 summary, p.130 lecture):

From Plantar warts (p.26 summary, p.130 lecture image caption):

- Plantar warts (verruca) form painful plaques (mosaic) containing black ‘dots’ that represent thrombosed capillaries.

(Plantar warts occur on the soles. They are common in children and young adults. Due to pressure, they grow inwards and can be covered by a callus, so the surface may appear somewhat smooth or hyperkeratotic, not necessarily "spiky" but not always perfectly smooth either. They can be fleshy. "The most common type" is debatable.)If answer C is correct, it implies plantar warts are not the most common. Smooth surface (b) can be true if covered by callus.

Page 26 (Test Bank - Viral infections cont.)

Question 195:
Elevated papules with a smooth surface, flesh lesions, colored brownish grayish of pinkish:
a. Plane warts
b. Common warts
c. Filiform warts
Answer: A

High-Yield Context (from general knowledge, and summary image p.130):*Plane warts are typically small, flat-topped (smooth surface) or slightly elevated papules, often skin-colored, pinkish, or light brown. This description fits plane warts well.*

Question 196:
Papule with rough, dry, hyperkeratotic surface represent which type of warts:
a. Common warts
b. Plantar warts
Answer: A (didn’t mention on the foot)

High-Yield Context (from general knowledge, and summary image p.130):*Common warts (verruca vulgaris) are characteristically rough, hyperkeratotic papules or nodules. Plantar warts are on the foot and are also hyperkeratotic, but often grow inwards due to pressure.*

Question 197:
ttt of choice for genital warts:
a. Salicylic acid 10%
b. Topical 5-FU
c. Surgery
d. Cryotherapy
e. Podophyllotoxin
Answer: E

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Wart viruses - Treatment of genital warts), p.26 summary, p.131 lecture, and STD lecture slides p.39):

From Treatment (general warts, p.131):

- Immune response modifier

Imiquimod®

- 5% cream is licensed for the treatment of

genital warts.From STD lecture slides (Tx of HPV, p.39):

- Destruction
  - Physical destruction by

cryotherapy (very painful)

  - Cytotoxic agent →

Podophyllotoxin, imiquimod 5%

  - Green tea catechins
  - Cidofovir gel
  - IFN- alpha (painful)
- Systemic “oral” → if extensive or failed topical Tx
  - Isotretinoin
  - Cidofovir

(Podophyllotoxin and Imiquimod are common patient-applied treatments. Cryotherapy, surgical excision, TCA are provider-administered. Salicylic acid is more for common cutaneous warts. 5-FU is used for some resistant warts but not typically first-line for genital warts due to irritation.)

Question 198:

ttt of choice of condolymata acuminata is:
a. Trichloroacetic acid
b. monochloroacetic acid
c. cantharidin
d. podophyllin
Answer: D

High-Yield Context (from general knowledge, and similar to above):*Condylomata acuminata are genital warts. Podophyllin (a resin from which podophyllotoxin is derived) is a provider-applied treatment. Trichloroacetic acid (TCA) is also provider-applied. Cantharidin is for non-genital warts.*

Question 199:

True about warts:
a. HPV is a double stranded DNA virus
b. Caused by HHV-6
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Wart viruses), p.25 summary, p.129 lecture):

From Wart viruses (p.25 summary, p.129 lecture):

- More than 100 different subtypes of

HPV have currently been identified.

(HPV (Human Papillomavirus) is a double-stranded DNA virus. HHV-6 is a herpesvirus, associated with roseola infantum and pityriasis rosea.)

Question 200:

All of the following skin eruptions are caused by viral infections except: **
a. Scarlet fever eruption
b. Pityriasis rosea
c. Roseola infuntum eruption
d. Slapped check syndrome of erythema infectiosum
e. Rubella eruption
Answer: A

High-Yield Context (from various summary sections on viral exanthems, and bacterial infections):

- **Scarlet fever:** Caused by exotoxins from *Streptococcus pyogenes* (a bacterium).
- **Pityriasis rosea (p.127 lecture):** Associated with HHV-6 or HHV-7 (viral).
- **Roseola infantum (p.134 lecture):** Caused by HHV-6 (viral).
- **Slapped cheek syndrome (Erythema infectiosum) (p.133 lecture):** Caused by Parvovirus B19 (viral).
- **Rubella (p.133 lecture):** Caused by Rubella virus (viral).

Fungal infections

Question 201:
Regarding cutaneous fungal infections one of the following statements is incorrect: *
a. Tinea capitis is usually treated with systemic antifungals
b. Seborrheic dermatitis is a differential diagnosis for psoriasis
c. Tinea pedis is usually treated topically
d. Chronic paronychia is usually caused by mixed yeast and bacterial infection
e. Pityriasis versicolor rarely recurs
Answer: E

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections, various sections pp.27-28 summary, pp.106-112 lecture):

- **a. Tinea capitis is usually treated with systemic antifungals (p.27 summary/p.106, p.108 lecture):** True. "Oral griseofulvin or terbinafine" for tinea capitis. "Topical antifungals are unable to penetrate the hair shaft..."
- **b. Seborrheic dermatitis is a differential diagnosis for psoriasis (p.27 summary/p.106 lecture - Tinea capitis DDx includes scalp eczema/psoriasis, seborrheic dermatitis):** True.
- **c. Tinea pedis is usually treated topically (p.27 summary/p.108 lecture):** True. "Tx→ topical antifungal (Terbinafine)"
- **d. Chronic paronychia is usually caused by mixed yeast and bacterial infection (p.28 summary/p.111 lecture):** True. (Often Candida and bacteria in wet-work individuals).
- **e. Pityriasis versicolor rarely recurs (p.28 summary/p.110 lecture - Treatment):** "Treatment: Topical selenium sulfide and topical ketoconazole 2% cream applied once daily for 2 weeks reportedly cures between 70 and 80% of patients, but **one-third relapse**." (False, recurrence/relapse is common).

Question 202:
Fungal responsible for epidemics of tinea capitis: *
a. T. Canis
b. T. verrucosum
c. T. mentagrophyte
d. M. audouinii
Answer: D

High-Yield Context (from general knowledge of tinea capitis epidemiology, as not explicitly detailed to this level in the summary, which focuses on T. tonsurans as most common in urban settings):*Historically, Microsporum audouinii was a common cause of epidemic tinea capitis in children (anthropophilic). Trichophyton tonsurans is now more common in many urban areas. T. canis (should be Microsporum canis) is zoophilic from cats/dogs. T. verrucosum is zoophilic from cattle. T. mentagrophytes can be anthropophilic or zoophilic.*

Question 203:
Doesn't cause tinea capitis:
a. Microsporum audouinii
b. Trichophyton schoenleinii
c. Trichophyton tonsurans
d. Trichophyton verrucosum
Answer: {all the above are causes of tinea capitis}

High-Yield Context (from general knowledge of dermatophytes causing tinea capitis):*All listed fungi are well-known causes of tinea capitis. T. schoenleinii causes favus, a severe form of tinea capitis.*

Question 204:
1st line ttt of T. capitis:
a. Griseofulvin
b. Topical miconazole
c. Steroids
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections (Tinea capitis - Treatment), p.27 summary/p.106, p.108 lecture):

From Tinea capitis (p.106 lecture):

- T. tonsurans must be treated with

systemic antifungal therapy to clear the endothrix infection, **1st line is Griseofulvin.From Tinea Capitis Treatment (p.108 lecture):**

-

Oral griseofulvin (10mg/kg if over 1 month of age; occasionally 20mg/kg is required) is the current **FDA-approved treatment.**

Question 205:

All about Griseofulvin are true except:

a. Has better absorption after meal

b. Commonest side effect is headache

c. Contraindicated in pregnancy

d. Phenobarbitone may neutralize its effect

e. May used in ttt of s.barbae

Answer: E

(Griseofulvin is active against dermatophytes. S. barbae usually refers to bacterial folliculitis of the beard (sycosis barbae) or tinea barbae (fungal). Griseofulvin is used for tinea barbae. Perhaps "s.barbae" is a typo for something else, or it refers to bacterial sycosis barbae for which griseofulvin would be ineffective. If it means tinea barbae, then griseofulvin is a treatment.)

High-Yield Context (from general pharmacology of Griseofulvin):

- a. Absorption is enhanced with a fatty meal (True).
- b. Headache is a common side effect (True).
- c. Teratogenic, contraindicated in pregnancy (True).
- d. Phenobarbital (an enzyme inducer) can decrease griseofulvin levels/efficacy (True).
- e. Griseofulvin *is* used for dermatophyte infections including tinea barbae (fungal infection of the beard area). If "s.barbae" refers to *Staphylococcal* sycosis barbae (bacterial), then griseofulvin would not be used. This option is ambiguous.*

Question 206:

Apple green fluorescence is seen in: **

a. Tinea capitis

b. Tinea curuis

Answer: A

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections (Principles of diagnosis - Wood's light), p.27 summary, p.106 lecture):

From Principles of diagnosis (p.27 summary / p.106 lecture):

-

Wood’s light (ultraviolet light) can reveal **Microsporum infections of hair**, as they produce a **green-blue fluorescence.** *(Some species of Microsporum (e.g., M. canis, M. audouinii) causing tinea capitis fluoresce greenish/bluish-green under Wood's light. Tinea cruris is not hair-bearing and the causative fungi don't typically fluoresce.)*

Question 207:

One of the following fungi induce inflammatory tinea capitis:

a. T. violaceum

b. T. sulphureum

c. T. tonsurans

d. T. verrucosum

e. T. mentagrophyte

Answer: D

High-Yield Context (from general knowledge; zoophilic fungi tend to cause more inflammation):*Zoophilic dermatophytes (acquired from animals) typically elicit a more vigorous inflammatory response in humans than anthropophilic ones. Trichophyton verrucosum (from cattle) and Microsporum canis (from cats/dogs) are common zoophilic fungi that cause inflammatory tinea, including kerion formation. T. mentagrophytes also has zoophilic strains that are inflammatory. T. tonsurans and T. violaceum are anthropophilic and often cause less inflammatory, "black dot"*

tinea capitis or chronic infections.T. verrucosum is a well-known cause of inflammatory tinea capitis, often leading to kerion.

Question 208:
Commonest fungal infection in adults:
Tinea pedis

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections (Feet and Hands - Tinea pedis), p.27 summary, p.108 lecture):

From Tinea pedis or athlete's foot (p.27 summary, p.108 lecture):
- A common disease mainly affecting

adults.*(Tinea pedis is very common in adults.)*

Question 209:
Wrong about tinea pedis:
a. Most common adult fungal infection
b. Can be caused by E.floccosum
c. Zoophilic
d. Caused by T.violaceum
Answer: D

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections (Tinea pedis), p.27 summary, p.108 lecture, and general mycology):

- **a. Most common adult fungal infection:** Tinea pedis is very common in adults (True).
- **b. Can be caused by E.floccosum:** *Epidermophyton floccosum* is a common cause of tinea pedis (and tinea cruris) (True).
- **c. Zoophilic:** The common causative agents of tinea pedis (*Trichophyton rubrum, Trichophyton interdigitale (formerly T. mentagrophytes var. interdigitale), Epidermophyton floccosum*) are primarily anthropophilic (human-to-human spread). Zoophilic fungi can occasionally cause tinea pedis but it's less typical for the common forms. (Likely False as a general statement for typical tinea pedis).
- **d. Caused by T.violaceum:** *Trichophyton violaceum* is an anthropophilic dermatophyte that primarily causes tinea capitis, especially in endemic regions. It's not a common cause of tinea pedis. (False).

(The question asks for "Wrong about tinea pedis". Statement D is definitively wrong as T. violaceum is not a common cause of tinea pedis. Statement C "Zoophilic" is also generally incorrect for the most common causative agents of tinea pedis. If the answer is D, it is clearly wrong.)

Page 28 (Test Bank - Fungal infections cont.)

Question 210:
T. corporis lesion:
a. Multiple vesicles
b. Annular
c. Wheal
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections (General features of fungi in the skin), p.27 summary, p.106 lecture):

From General features of fungi in the skin (p.27 summary / p.106 lecture):
- Some lesions have a prominent scaling margin with apparent clearing in the center, leading to

annular or ring-shaped lesions – hence the term 'ringworm'.

(Tinea corporis (ringworm of the body) classically presents as annular or arcuate lesions with a raised, erythematous, scaling border and central clearing.)

Question 211:
Slightly elevated scaling margins and halo central clearing:
T. corporis

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections (General features of fungi in the skin), p.27 summary, p.106 lecture):

From General features of fungi in the skin (p.27 summary / p.106 lecture):

- Some lesions have a prominent

scaling margin with apparent **clearing in the center**, leading to annular or ring-shaped lesions – hence the term 'ringworm'.

(This is a classic description of tinea corporis.)

Question 212:
All true about tinea versicolor except:
a. Apple green color on wood's lamp
b. Hypo or hyper pigmentation
c. Patches or plaques
d. Scaling
e. Most common causative agent is Malassezia
answer: A

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections (Pityriasis versicolor), p.27 summary, p.110 lecture):

From Pityriasis versicolor (p.27 summary, p.110 lecture):

- M. Furfur (Malassezia furfur)
- Well-defined macular lesions of variable colour ('versicolor') →

hyper or hypo pigmented → darker brown to pale tan

- Diagnosis by: … Wood's Light exam:

Golden yellow color

- Well-defined

macular lesions … fine **scale** appear.

(Pityriasis versicolor fluoresces a golden-yellow or coppery-orange under Wood's light, not apple green. Apple green fluorescence is characteristic of some Microsporum species in tinea capitis.)

Question 213:
Wrong about tinea versicolor:
Cherry red fluorescence under wood's lamp

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections (Pityriasis versicolor), p.27 summary, p.110 lecture):

From Pityriasis versicolor (p.27 summary, p.110 lecture):

- Diagnosis by: … Wood's Light exam:

Golden yellow color*(Cherry red fluorescence under Wood's light is characteristic of erythrasma (Corynebacterium minutissimum). Pityriasis versicolor is golden-yellow.)*

Question 214:
Treatment of toenail onychomycosis (three yellow nails)? *
Oral antifungal

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections (Onychomycosis - Treatment), p.28 summary, p.111 lecture):

From Nails Onychomycosis Treatment (p.28 summary, p.111 lecture):

-

Systemic therapy with terbinafine 250mg daily for 16 weeks (toenails) or 8 weeks (fingernails) is usually considered the **first line.**

- Topical treatment: should be considered for a single nail or very mild distal nail-plate onychomycosis.

(For multiple affected toenails (like "three yellow nails"), systemic oral antifungal therapy is generally required for effective treatment as topical agents have poor penetration.)

Question 215:
Tinea ungium is the infection of:
a. Lateral nail folds
b. Posterior nail folds

c. Nail plate
d. Nail bed
e. Nail matrix
Answer: C

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections (Onychomycosis), p.27 summary, p.110 lecture):

From Nails - Onychomycosis (p.27 summary, p.110 lecture):

- Mostly adult toenails
-

Nail plates → thickened, brittle and white to yellow/brown, from distal to proximal

(Tinea unguium refers to dermatophyte infection of the nail unit, primarily affecting the nail plate and nail bed. The visible changes are in the nail plate.)

Question 216:
Which's wrong about tinea ungium:
a. Change in nail color
b. Onycholysis
c. Prolonged treatment
d. Caused by T. verrucosum
Answer: D

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections (Onychomycosis), p.27-28 summary, p.110-111 lecture):

- **a. Change in nail color:** Nail plates → thickened, brittle and **white to yellow/brown** (True).
- **b. Onycholysis:** (Separation of nail from bed) is a common feature (True).
- **c. Prolonged treatment:** Systemic therapy for toenails is 16 weeks (4 months) (True).
- **d. Caused by T. verrucosum:** Common causes of onychomycosis are *Trichophyton rubrum* and *Trichophyton interdigitale*. *T. verrucosum* is a zoophilic fungus primarily causing inflammatory tinea in cattle and humans (e.g., tinea capitis, tinea barbae, tinea corporis). While it *can* rarely cause onychomycosis, it's not a typical or common cause compared to others. (Likely False as a common cause).

Question 217:
Which of the following causes erythema with satellite pustules over body flexors:
Candida cutaneous infection

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections (Yeast infections - Candida intertrigo), p.28 summary, p.106, p.112 lecture):

From Tinea cruris - DDx (p.27 summary / p.106 lecture):

■
Candida ⇒ intense erythema and satellite lesionsFrom Yeast infections - Candida Intertrigo (p.28 summary / p.112 lecture):

- Candida infection may occur in the

flexures...

- Candida intertrigo is symmetrical and

'satellite' pustules or papules outside the outer rim of the rash are typical.

(Erythema with satellite pustules in flexural areas is characteristic of Candida intertrigo.)

Question 218:
Not associated with candidal infection: *
a. Occurs between the 2nd and 3rd fingers
b. Affects proximal lamella
c. Corner of the mouth
d. Tongue and oral mucosa
e. Genital area
Answer: A

(While interdigital candidiasis can occur, especially in toe webs (erosio interdigitalis blastomycetica), it's less classically defining for "fingers" compared to other manifestations. Proximal nail fold involvement (chronic paronychia often with Candida), angular cheilitis (corners of mouth), oral thrush (tongue/mucosa), and vulvovaginal/balanitic candidiasis (genital area) are all well-known sites. Candidiasis between fingers is less common than between toes or in other listed sites.)

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections (Candida infection), p.28 summary, p.112 lecture):

From Candida infection (p.28 summary, p.112 lecture):

- Mucous membranes and flexures...
- C. Albicans →

mouth"white plaques or erythema" (tongue and oral mucosa), **vagina "thrush" (genital area)**

-

Chronic paronychia (p.28 summary / p.111 lecture): Around the nails ... Erythema and swelling of the nail fold... (often Candida involvement, affecting proximal/lateral nail folds, which are related to proximal lamella of nail).

(Angular cheilitis (corners of mouth) is also commonly caused by Candida.)Given the options, A is the least typical or prominent site compared to the others for primary candidal infection sites emphasized.

Question 219:

Which one is not commonly colonize healthy skin: *

a. Staph. albus

b. Staph. aureus

c. Candida albicans

Answer: C

High-Yield Context (from Dermatology Lecture Slides - Bacterial skin infections (Normal skin flora) p.22 summary/p.114 lecture, and Fungal infections (Candida) p.28 summary/p.112 lecture):

From Bacterial skin infections - Normal skin flora (p.22 summary, p.114 lecture):

-

Coagulase-negative Staphylococcus (e.g., Staph. epidermidis, formerly often grouped as Staph. albus), Corynebacterium, Diphtheroids, and α-haemolytic Streptococci in the epidermis.

- Propioni- bacterium in the pilosebaceous unit.

*(Staphylococcus aureus can be carried on healthy skin or in nares (carrier state), but is also a pathogen.)***From Yeast infections - Candida (p.28 summary, p.112 lecture):**

- Yeast, including

C. albicans, may be found in the mouth and vagina of healthy individuals.*(Candida albicans is part of the normal flora of mucous membranes (mouth, GI tract, vagina) in many healthy individuals. It is less commonly considered a prominent colonizer of healthy, intact skin in large numbers, though it can be found transiently or in intertriginous areas. Staph epidermidis (albus) and even S. aureus (in carrier state) are more typical skin colonizers.)*

Question 220:

Candidiasis may affect all of the following except:

a. Skin

b. Mucous membranes

c. Nail

d. Hair

Answer: D

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections (Candida infection), p.28 summary, p.112 lecture, and Onychomycosis p.27 summary/p.110 lecture):

From Candida infection (p.28 summary, p.112 lecture):

-

Mucous membranes and flexures...

- C. Albicans →

mouth, **vagina**

- Candida intertrigo (skin folds)

From Onychomycosis (p.27 summary, p.110 lecture):
○ May be caused by
Candida albicans (affecting nails)

(Candida commonly infects skin (intertrigo, paronychia), mucous membranes (oral thrush, vulvovaginitis), and nails (candidal onychomycosis/paronychia). It is not a typical pathogen of the hair shaft itself (like dermatophytes in tinea capitis). Scalp candidiasis can occur on the skin of the scalp, but not primarily infecting the hair fiber.)

Question 221:
Kerion is caused by
T. verrucosum & T. mentagrophytes

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections (Tinea capitis - Kerion), p.27 summary/p.107 lecture, and general knowledge):

From Tinea capitis (p.27 summary / p.107 lecture):
•
Kerion formation → inflamed, boggy, pustular lesion on the scalp

(Kerion is a severe, inflammatory form of tinea capitis. It is typically caused by zoophilic dermatophytes, which elicit a strong host immune response. Trichophyton verrucosum (from cattle) and zoophilic strains of Trichophyton mentagrophytes are classic causes of kerion. Microsporum canis (from cats/dogs) can also cause kerion.)

Page 29 (Test Bank - Fungal infections cont.)

Question 222:
One of the following fungi is ectothrix:
a. T. schoenleinii
b. T. violaceum
c. T. mentagrophytes
d. T. tonsurans
e. T. sulphureum
Answer: C

High-Yield Context (from general mycology knowledge as ectothrix/endothrix classification is not explicitly detailed in the provided summary, though implied by T. tonsurans being endothrix on p.106 lecture):*Hair invasion by dermatophytes is classified as ectothrix (arthroconidia on the outside of the hair shaft) or endothrix (arthroconidia inside the hair shaft). Favus (caused by T. schoenleinii) is a distinct type with scutula.*

- *T. schoenleinii:* Causes favic type invasion.
- *T. violaceum:* Typically endothrix.
- ***T. mentagrophytes:*** **Typically ectothrix.**
- *T. tonsurans:* Typically endothrix (stated on p.106 lecture slides: "it penetrates the hair shaft (endothrix fungus)").
- *T. sulphureum:* Can be endothrix.

(T. mentagrophytes is a classic example of an ectothrix invader.)

Question 223:
Fungi doesn’t fluoresce under wood's light:
a. M. audouinii
b. M. canis
c. M. gypsum
d. T. mentagrophytes
e. T. schoenleinii
Answer: D

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections (Wood's light), p.27 summary, p.106 lecture, and general knowledge):

From Principles of diagnosis (p.27 summary / p.106 lecture):
- Wood’s light (ultraviolet light) can reveal

Microsporum infections of hair, as they produce a **green-blue fluorescence.***(Generally, Microsporum species (like M. audouinii, M. canis, M. gypseum) that cause tinea capitis often fluoresce. Trichophyton species usually do not fluoresce, with some exceptions (e.g., T. schoenleinii causing favus can have a pale greenish fluorescence). T. mentagrophytes typically does not fluoresce.)*

Question 224:
All dermatophytes fluoresce under wood's light except:
a. Microsporum canis
b. Microsporum audouinii
c. T. schoenleinii
d. T. rubrum
Answer: D

High-Yield Context (from context above and general knowledge):*As noted, Microsporum species often fluoresce. T. schoenleinii (favus) may show a pale greenish fluorescence. Trichophyton rubrum, a very common dermatophyte causing tinea pedis, corporis, cruris, and onychomycosis, does NOT fluoresce under Wood's light.*

Question 225:
Which dermatophyte is likely to be acquired from cattle:
a. Trichophyton rubrum
b. Trichophyton schoenleinii
c. Trichophyton verrucosum
d. Trichophyton tonsurans
e. Microsporum gypsum
Answer: C

High-Yield Context (from general mycology knowledge, as specific animal sources are not detailed in the summary):

- *T. rubrum:* Anthropophilic.
- *T. schoenleinii:* Anthropophilic.
- ***T. verrucosum:*** **Zoophilic, primarily from cattle.**
- *T. tonsurans:* Anthropophilic.
- *M. gypseum:* Geophilic (from soil), can infect animals and humans.

(T. verrucosum is the classic dermatophyte associated with cattle.)

Hair and scalp & diseases of the nails

Question 226:
Which’s wrong about hair:
a. Male hair grows faster
b. Growth rate = 1cm/month
c. Hair spends growing 3-4 years before falling
d. All hair characteristics are genetically determined
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Hair and Scalp (Hair cycle, Hair Types), p.29 summary, p.63-64 lecture):

From Hair cycle (p.29 summary / p.64 lecture):
●
Anagen → active growth phase, 1000 days (approx. 3 years), 85% of hair
● Catagen → short growth arrest phase,10 days
● Telogen → resting phase,100 days. 15% of hair

(Average hair growth rate is about 0.3-0.4 mm/day, which is roughly 1 cm per month (b=true). Anagen phase duration (growth phase) is genetically determined and averages 2-7 years, so 3-4 years is within range (c=true). Hair characteristics like color, texture, and density are genetically determined (d=true). Whether male hair grows faster than

female hair is debatable and can vary by site and individual; there isn't a consistent, significant difference making 'male hair grows faster' a universal truth to be considered correct here, so A being "wrong" is plausible.)

Question 227:
% of hair follicles in scalp present in anagen phase is:
a. 50%
b. 60%
c. 70%
d. 85%
Answer: D

High-Yield Context (from Dermatology Lecture Slides - Hair and Scalp (Hair cycle), p.29 summary, p.64 lecture):

From Hair cycle (p.29 summary / p.64 lecture):
-
Anagen → active growth phase, 1000 days, 85% of hair
- Catagen → short growth arrest phase,10 days
- Telogen → resting phase,100 days. 15% of hair

Question 228:
Which of the following is the resting stage of hair:
Telogen

High-Yield Context (from Dermatology Lecture Slides - Hair and Scalp (Hair cycle), p.29 summary, p.64 lecture):

From Hair cycle (p.29 summary / p.64 lecture):
- Anagen → active growth phase...
- Catagen → short growth arrest phase...
-
Telogen → resting phase,100 days. 15% of hair

Question 229:
All are causes of traumatic alopecia except:
a. Traction
b. Pressure
c. Marginal
d. Trichotillomania
e. ....
Answer: E
(Assuming "e" is something not traumatic. Traction alopecia (from tight hairstyles), pressure alopecia (prolonged pressure), and trichotillomania (hair pulling) are all forms of traumatic alopecia. "Marginal" might refer to marginal traction alopecia.)

High-Yield Context (from general knowledge, as "traumatic alopecia" is not specifically detailed as a category in the provided summary sections on hair loss, which focus on androgenetic, areata, and telogen effluvium):*Traumatic alopecia results from physical damage to the hair or follicles. Traction, friction, pressure, and compulsive pulling (trichotillomania) are all causes.*

Question 230:
Wrong about alopecia areata?
a. Fluorescent on woods lamp
b. Causes non-scarring alopecia
c. Can occur in children
d. Recurring in nature
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Hair and Scalp (Alopecia areata), p.29 summary, p.64 lecture):

From Alopecia areata “AA” (p.29 summary / p.64 lecture):
- AA → smooth round or oval patches of
non-scarring hair loss on the scalp. (b=true)
- Exclamation mark hair ... is diagnostic for AA
- Poor Px →
childhood onset, atopic, ophiasis, nail dystrophy, family history (c=true)

- Associated autoimmune→ vitiligo, thyroiditis

(Alopecia areata is an autoimmune, non-scarring alopecia. It is recurrent. Wood's lamp is used for fungal infections (some fluoresce) or pigmentary disorders, not typically for diagnosing alopecia areata, and AA lesions do not fluoresce.)

Question 231:
Telogen effluvium, what's wrong:
a. Can be caused by drugs
b. Wood's lamp helps in diagnosis
c. Can happen few months after childbirth
d. Presents as diffuse thinning of hair
e. Non-scarring alopecia
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Hair and Scalp (Telogen effluvium), p.29 summary, p.64 lecture):

From Telogen effluvium (p.29 summary / p.64 lecture):
-
Diffuse synchronous shedding approximately **2 months after the triggering event** (d=true, c=true for post-partum TE)
- Causes
■ Hormonal (e.g. pregnancy and
post-partum)
■ Nutritional (e.g. iron deficiency,hypo-proteinaemia),Acute weight changes
■
Drugs (e.g. β -blockers, anticoagulants, retinoids, immunisation) (a=true)
■ Systemic disease (e.g. chronic inflammatory diseases,IBD, malignancy)
■ Stress (e.g. pyrexia, major surgery or trauma)
- Investigations → TFT, ferritin, vitamin B12, folate and zinc

(Telogen effluvium is a diffuse, non-scarring hair loss (e=true). Wood's lamp is not used in its diagnosis.)

Question 232:
Which of the following results in anagen effluvium:
Cytotoxic drugs

High-Yield Context (from general knowledge, as anagen effluvium is not explicitly detailed in the summary which focuses on telogen effluvium):*Anagen effluvium is an abrupt loss of anagen (growing) hairs, most classically caused by chemotherapy (cytotoxic drugs) or radiation therapy, which affects rapidly dividing cells including those in the hair matrix.*

Question 233:
Not in telogen effluvium:
a. Post-partum
b. Post-surgical
c. Crush diet
d. Post febrile
e. Cytotoxic drugs
answer: E

High-Yield Context (from Dermatology Lecture Slides - Hair and Scalp (Telogen effluvium - Causes), p.29 summary, p.64 lecture):

From Telogen effluvium - Causes (p.29 summary / p.64 lecture):
■ Hormonal (e.g. pregnancy and
post-partum)
■ Nutritional (e.g. iron deficiency,hypo-proteinaemia),Acute weight changes (
Crush diet)
■ Drugs (e.g. β -blockers, anticoagulants, retinoids, immunisation)
■ Systemic disease (e.g. chronic inflammatory diseases,IBD, malignancy)
■ Stress (e.g.
pyrexia (post febrile), major **surgery (post-surgical)** or trauma)

(Cytotoxic drugs cause anagen effluvium, not telogen effluvium.)

Question 234:
Which of the following doesn't cause diffuse non-scarring alopecia:
a. Trichotillomania
b. Heparin
c. Surgery shock
d. Telogen effluvium
e. Cytotoxic drugs
Answer: A

High-Yield Context (from various hair loss sections):

- **Trichotillomania:** Compulsive hair pulling, causes *patchy* alopecia with broken hairs of different lengths, not typically diffuse. It is non-scarring unless there's severe secondary infection/inflammation from chronic pulling.
- **Heparin (an anticoagulant):** Can cause telogen effluvium (diffuse, non-scarring).
- **Surgery shock:** A major stressor, can cause telogen effluvium (diffuse, non-scarring).
- **Telogen effluvium:** By definition, a diffuse non-scarring alopecia.
- **Cytotoxic drugs:** Cause anagen effluvium, which is diffuse and non-scarring.

(Trichotillomania typically causes patchy, irregular hair loss, not diffuse loss.)

Page 31 (Test Bank - Hair and scalp & diseases of the nails cont.)

Question 235
All may cause non-cicatricial alopecia except:
a. Surgical shock
b. Morphea
c. Heparin
d. Protein malnutrition
Answer: B

High-Yield Context (from various hair loss sections and Morphea section):

- **Surgical shock (p.64 lecture):** Stressor, can cause telogen effluvium (non-cicatricial).
- **Morphea (localized scleroderma) (p.16 summary/p.8 CTD lecture):** Sclerosis of the skin. If it affects the scalp ("en coup de sabre"), it causes **scarring (cicatricial) alopecia** within the sclerotic plaque.
- **Heparin (p.64 lecture - drug causes of TE):** Can cause telogen effluvium (non-cicatricial).
- **Protein malnutrition (p.64 lecture - nutritional causes of TE):** Can cause telogen effluvium (non-cicatricial).

(Morphea on the scalp causes scarring alopecia.)(The lecture slide p.31 itself has a useful summary box about types of alopecia not included in the main book summary).
SUMMARY (from slide p.31):
Diffuse non-scarring alopecia:
1- telogen effluvium
2- anagen effluvium
3- endocrine dysfunction
4- nutritional
5- anhidrotic ectodermal dysplasia
6- hereditary hair shaft abnormalities
7- diffuse alopecia areata
Diffuse scarring alopecia:
1- erosive pustular dermatosis (physical agents (burns))

Question 236:
All of the following cause diffuse non-scaring alopecia except:
a. Heparin
b. 2ry syphilis
c. Anhydrotic ectodermal dysplasia

d. Cachexia
Answer: C
(The OCR gives C. The summary box on p.31 of lecture notes lists "anhidrotic ectodermal dysplasia" under diffuse non-scarring alopecia. Secondary syphilis causes a "moth-eaten" patchy alopecia, which is non-scarring, not typically diffuse. Heparin causes TE (diffuse, non-scarring). Cachexia (severe malnutrition) causes TE (diffuse, non-scarring). The note "2ry syphilis causes patchy non-scarring alopecia" on the slide highlights this. So if the question asks for "diffuse", 2ry syphilis (b) would be a better "except". If the answer is C, it contradicts the summary box on the same page.)Let's re-evaluate based on the provided answer C. If Anhydrotic ectodermal dysplasia is the "except", it means it doesn't cause diffuse non-scarring alopecia, or it causes scarring alopecia, or patchy. However, it's known for sparse, fine hair (hypotrichosis), which is diffuse and non-scarring. This seems like a conflict with the provided answer and the slide's own summary box.

High-Yield Context (from summary box on lecture slide p.31 and general knowledge):

Diffuse non-scarring alopecia (from slide p.31 box):
1- telogen effluvium (caused by heparin, cachexia)
2- anagen effluvium
3- endocrine dysfunction
4- nutritional
5-
anhidrotic ectodermal dysplasia
6- hereditary hair shaft abnormalities
7- diffuse alopecia areata
Patchy non-scarring alopecia (from slide p.31 box):
1- alopecia areata
2- tinea capitis
3-
secondary syphilis (moth eaten)
4- hereditary female pattern alopecia
5- traction alopecia

(Secondary syphilis causes patchy (moth-eaten) non-scarring alopecia, not diffuse. Anhydrotic ectodermal dysplasia causes diffuse sparse hair, which is non-scarring. So B should be the answer if "diffuse" is key.)

Question 237:
Non-scarring alopecia all except:
a. Male pattern baldness
b. Alopecia areata
c. 2ry syphilis
d. Sarcoidosis
Answer: D
High-Yield Context (from summary box on lecture slide p.31 and general knowledge):

- **Male pattern baldness (Androgenetic alopecia):** Non-scarring.
- **Alopecia areata (p.64 lecture):** Non-scarring. (Also in patchynon-scarring box p.31)
- **2ry syphilis (p.31 lecture summary box):** Patchy non-scarring (moth-eaten).
- **Sarcoidosis:** Can cause specific granulomatous infiltration of the scalp leading to **scarring alopecia.**

Question 238:
All are causes of diffuse non-scarring alopecia except:
a. Telogen effluvium
b. Anagen effluvium
c. Hypothyroidism
d. Hair shaft abnormalities
e. Male pattern of hair loss
Answer: D
(The provided answer is D, but the image highlights E. Male pattern hair loss (androgenetic alopecia) is typically patterned (vertex, frontal), not truly "diffuse" in the same way as TE/AE, though it leads to diffuse thinning within the affected pattern. Hair shaft abnormalities often lead to hair breakage and can appear as diffuse thinning/poor hair quality, and are

non-scarring. Hypothyroidism causes TE (diffuse non-scarring). The summary box on p.31 lists "hereditary hair shaft abnormalities" as a cause of diffuse non-scarring alopecia.)If the answer is E (Male pattern of hair loss): Androgenetic alopecia causes patterned loss, which can be extensive and appear "diffuse" within that pattern, but it's not the same as a generalized TE/AE. This is a plausible "except" for "diffuse".The note on the slide "male pattern of hair loss causes patchy non-scarring alopecia" is potentially misleading; it's patterned, not typically described as "patchy" like alopecia areata.

High-Yield Context (from summary box on lecture slide p.31):

Diffuse non-scarring alopecia:
1-
telogen effluvium (caused by hypothyroidism)
2-
anagen effluvium
3- endocrine dysfunction (e.g.,
hypothyroidism)
4- nutritional
5- anhidrotic ectodermal dysplasia
6-
hereditary hair shaft abnormalities
7- diffuse alopecia areata

(Male pattern hair loss is not in this list of diffuse non-scarring; it's patterned.)

Question 239:
Cuticle of nail is formed by:
a. Ventral laver of posterior fold
b. Dorsal layer of posterior fold
c. Lateral fold
d. Matrix
Answer: A

High-Yield Context (from general nail anatomy, as the provided summary/slides (p.30-32/p.64-65) focus on nail plate formation by matrix and nail disorders, not specific origin of cuticle layers):*The nail cuticle (eponychium) is an extension of the stratum corneum of the proximal nail fold. The proximal nail fold has a dorsal and a ventral surface. The cuticle originates from the ventral aspect of the proximal nail fold.*

Question 240:
Not a result of disordered keratinization of nail matrix:
a. Change in nail shape
b. Long ridging
c. Thick nails
d. .....
Answer: D
(Missing option makes it hard to analyze. Disordered keratinization of the nail matrix can lead to changes in nail plate thickness (thick or thin nails), surface (ridging, pitting), and overall shape/texture.)

High-Yield Context (from Diseases of the Nails section, p.31-32 lecture slides):*(The nail matrix produces the nail plate. Any disorder affecting matrix keratinization will result in nail plate abnormalities like pitting, ridges, Beau's lines, changes in thickness or texture.)*

Question 241:
Which of the following is the characteristic hair lesion seen in Netherton’s syndrome:
Trichorrhexis invaginatum

High-Yield Context (from general knowledge, as Netherton's syndrome is not detailed in the provided summary):*Netherton's syndrome is a rare autosomal recessive disorder characterized by ichthyosis linearis circumflexa, atopic diathesis, and hair shaft abnormalities. The pathognomonic hair shaft defect is* ***trichorrhexis invaginata*** *("bamboo hair"), where the distal part of the hair shaft telescopes into the proximal part. Trichorrhexis nodosa can also be seen.*

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skin tumors

Question 242:
Benign tumor of the epidermis: *
a. Actinic keratosis
b. Seborrheic keratosis
answer: B

High-Yield Context (from Dermatology Lecture Slides - Benign Skin Tumours (Seborrheic keratoses), p.34 summary/p.49 lecture, and Premalignant Skin Tumours (Actinic keratoses), p.36 summary/p.36 lecture):

From Benign Skin Tumours - Seborrhoeic keratoses (p.34 summary, p.49 lecture):

- **Benign growths of immature keratinocytesFrom Premalignant and Malignant skin lesions - Actinic keratoses (p.36 summary, p.36 lecture):**
- Distribution → over sun exposed skin... patch of thick, scaly, or crusty skin.
- Px →

20% risk of progression to squamous cell carcinoma if untreated*(Seborrheic keratosis is a benign epidermal tumor. Actinic keratosis is a premalignant lesion of the epidermis.)*

Question 243:
Which one of the following is an eccrine sweat gland tumor:
a. Trichoepithelioma
b. Syringoma
c. Pilomatrixoma
d. Trichofolliculoma
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Benign Skin Tumours (Benign tumour papules), p.35 summary, p.54 lecture):

From Benign tumour papules (p.35 summary, p.54 lecture):

●
Syringomas
○
eccrine glands, multiple, slow-growing, small and flesh coloured
○ on the face around the eye around puberty
●
Trichoepitheliomas
○
hair follicle, multiple, slow-growing, small and flesh coloured
○ On face and scalp
●
Pilomatrixoma (p.35 summary for nodules):
○
Hair matrix, very hard slow-growing lump,on the head/neck of a child
●
Trichofolliculoma (not explicitly in this papule/nodule section, but is a benign follicular tumor)

(Syringoma is a benign tumor of eccrine sweat ducts.)

Question 244:
Wrong about nevus:
a. Result from abnormal proliferation of melanocytes
b. Developmental disorder
c. Common in infants
d. Increase after ACTH injection
e. Flair up during adolescence
Answer: C

High-Yield Context (from Dermatology Lecture Slides - Benign Skin Tumours (Melanocytic naevi), p.34 summary, p.50 lecture):

From Melanocytic naevi (p.34 summary, p.50 lecture):

○

Congenital melanocytic nevi ('birthmark')
■ Small < 2 cm, medium (2-20cm), giant > 20cm ... grow in proportion to the growth of the child...
○
Acquired melanocytic naevi
■ RF → solar radiation and a genetic susceptibility.
(These appear later in childhood/adulthood).

(Congenital nevi are present at birth or appear in infancy. Acquired nevi develop throughout childhood and adolescence, often peaking in young adulthood. So nevi are common in infants (congenital) or develop during childhood/adolescence (acquired). Statement c "Common in infants" refers to congenital nevi, which is true. Acquired nevi increase in number during childhood and adolescence (e=true). ACTH can stimulate melanocytes (d=true). They are developmental proliferations of melanocytes (a,b=true). Perhaps "common in infants" is less true than their appearance/increase during later childhood/adolescence for acquired nevi, which are more numerous overall.)If C is the answer, it implies that while congenital nevi exist, the bulk of nevi (acquired) appear later, making the statement "Common in infants" less accurate overall for all types of nevi.

Question 245:
Tumor may show malignant degeneration:
a. Compound nevus
b. Junctional nevus
c. Dermal nevus
d. Epidermal nevus
Answer:
(Answer is not provided. Any melanocytic nevus (junctional, compound, dermal) has a potential, albeit usually very small, for malignant degeneration into melanoma. Junctional nevi are sometimes considered to have a slightly higher theoretical risk due to the location of melanocytes at the DEJ, but any can transform. Epidermal nevus is different (hamartoma of epidermis) and can rarely develop BCC/SCC within it, not melanoma.)

High-Yield Context (from Dermatology Lecture Slides - Benign Skin Tumours (Melanocytic naevi), p.34 summary, p.50 lecture, and Malignant Skin Tumours (Melanoma), p.37 summary):From Melanocytic naevi (p.34 summary):
○ Congenital melanocytic nevi ('birthmark') ... Giant lesions → most likely to undergo malignant change (~5%).

From Melanoma (p.37 summary):
○ RF ⇒ ... Pre-existing moles

(Giant congenital nevi have the highest risk. Acquired nevi, especially dysplastic ones, also carry risk. The type (junctional, compound, dermal) indicates the location of nevus cells, all are melanocytic.)

Question 246:
Commonest site of squamous cell epithelioma:
a. Upper lip
b. Lower lip
c. Face
d. Hands
Answer: C
(If referring to SCC of the skin, sun-exposed areas like the face, ears, scalp, dorsal hands are common. The lower lip is a very common site for SCC due to sun exposure, more so than the upper lip. Overall "Face" encompasses many high-risk sites including lips, nose, ears, cheeks.)

High-Yield Context (from Dermatology Lecture Slides - Malignant Skin Tumours (Squamous cell carcinoma), p.37 summary, p.59 lecture):

From Squamous cell carcinoma (SCC) (p.37 summary, p.59 lecture):
○ Distribution:
■ Mostly in
head and neck (70%)
■ On
sun-exposed areas (dorsal hands, scalp, lip, and superior surface of pinna)*(Face is a major area. Lower lip is particularly prone to SCC compared to upper lip.)*

Question 247:
Which of the following is premalignant: *
a. Lentigo maligna
b. Bowen’s disease
c. Erythroplasia of Queyrat
d. Actinic keratosis
Answer: D

High-Yield Context (from Dermatology Lecture Slides - Premalignant and Malignant skin lesions (Actinic keratoses, Bowen's disease), p.36-37 summary, p.36-37 lecture):

From Premalignant skin tumours - Actinic keratoses (p.36 summary, p.36 lecture):
- Px →

20% risk of progression to squamous cell carcinoma if untreatedFrom Premalignant skin tumours - Bowen’s disease “SCC in situ” (p.37 summary, p.37 lecture):
- If in the glans penis or prepuce →

Erythroplasia of QueyratFrom Malignant skin tumours - Melanoma - Types (p.37 summary, p.60-61 lecture):
■
Lentigo maligna melanoma → on face,elderly
● single or multiple solar lentigos → irregular ,larger pigmented macule (
lentigo maligna,tx;imiquimod) → if darker\nodule → Lentigo maligna melanoma

(Actinic keratosis is premalignant for SCC. Bowen's disease and Erythroplasia of Queyrat are SCC in situ. Lentigo maligna is melanoma in situ.)The question asks for "premalignant". AK is premalignant. Bowen's, Erythroplasia, and Lentigo Maligna are all forms of carcinoma in situ, which is a step beyond premalignant, it is non-invasive cancer. However, in many classifications, "premalignant" is used more broadly to include in situ lesions. If D is the answer, it's because AK is clearly premalignant and not yet in situ carcinoma.

Question 248:
70-year-old male, fair skinned, presented with fine scaled erythematous plaques on the back of his hands, that was also found on his bald scalp, what is the likely diagnosis:
Actinic keratosis

High-Yield Context (from Dermatology Lecture Slides - Premalignant Skin Tumours (Actinic keratoses), p.36 summary, p.36 lecture):

From Actinic keratoses (AK) (p.36 summary, p.36 lecture):
- Distribution →

over sun exposed skin→ face (including the lip), **dorsal hands**, distal limbs and **bald scalp**
- RF →

fair/sun-damaged skin and increasing age
- Morphology →

patch of thick, scaly, or crusty skin. dark, light, tan, pink, red...
■ Classic AKs → white,
scaly macules, papules or plaques of various thickness, with surrounding **erythema***(This is a classic description of actinic keratoses.)*

Question 249:
False about actinic keratosis:
a. It is a malignant condition
b. Mostly affecting fair skinned people
c. Mostly on sun exposed areas
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Premalignant Skin Tumours (Actinic keratoses), p.36 summary, p.36 lecture):

From Actinic keratoses (AK) (p.36 summary, p.36 lecture):
- Distribution →

over sun exposed skin (c=true)
- RF →

fair/sun-damaged skin and increasing age (b=true)

- Px →

20% risk of progression to squamous cell carcinoma if untreated*(Actinic keratosis is a premalignant condition, not yet malignant, though it has the potential to become SCC.)*

Question 250:
The most frequent site for mets of BCC:
a. Skin
b. Regional LN
c. Lung
d. Brain
e. Liver
Answer: B
(Metastasis from BCC is extremely rare. When it does occur, regional lymph nodes are the most common site, followed by lung, bone, etc. However, it is important to stress its rarity.)

High-Yield Context (from general knowledge of BCC, as metastasis is not discussed in the summary which focuses on local behavior and treatment):*Basal cell carcinoma is locally invasive and destructive but has an exceedingly low metastatic potential (estimated <0.1% to 0.5%). When metastases do occur, they most commonly spread to regional lymph nodes, then lungs, bones, skin, and liver.*

Page 33 (Test Bank - skin tumors cont.)

Question 251:
Most common BCC type:
Noduloulcerative

High-Yield Context (from Dermatology Lecture Slides - Malignant Skin Tumours (Basal cell carcinoma - BCC types), p.37 summary, p.59 lecture):

From Basal cell carcinoma (BCC) - BCC types (p.37 summary, p.59 lecture):
■
Nodular → small pearly papules or nodules, rolled edge ulcer, telangiectasia. On sun-exposed areas of the head and neck *(This is the most common type. "Noduloulcerative" refers to a nodular BCC that has ulcerated, also very common).*
■ Superficial → erythematous patch, on trunk
■ Pigmented
■ Morphoeic or sclerosing → superficial atrophic scar, loss of the normal skin markings and the indistinct edge
■ Rodent ulcer→ central necrosis.

Question 252:
All about BCC is true except:
a. More common in Caucasian
b. Always associated with bad prognosis
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Malignant Skin Tumours (Basal cell carcinoma), p.37 summary, p.59 lecture):

From Basal cell carcinoma (BCC) (p.37 summary, p.59 lecture):
○ most common cancer in humans.lifetime risk of around 30%
○ RF → age,
fair skin, high-intensity UV exposure, radiation, immunosuppression, previous BCCs *(More common in Caucasians/fair skin)* (a=true)
○ Management → Depends on Histology… Mohs' surgery… excision… radiotherapy… cryotherapy… imiquimod… PDT…
(BCC is generally slow-growing and has an excellent prognosis with appropriate treatment, especially for common types in usual locations. Metastasis is extremely rare. Some high-risk subtypes or locations can recur or be locally aggressive, but "always associated with bad prognosis" is false.) (b=false)

Question 253:
Nodule on nose, glossy, with telangiectasia, diagnosis:
a. BCC
b. SCC
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Malignant Skin Tumours (Basal cell carcinoma - Morphology, BCC types), p.37 summary, p.59 lecture):

From Basal cell carcinoma (BCC) - Morphology (p.37 summary):
○
shiny,translucent pearly skin nodule or red patch or ‘rolled edge’ ulcer

From BCC types - Nodular (p.37 summary, p.59 lecture):
■ Nodular → small
pearly papules or nodules, rolled edge ulcer, telangiectasia. On sun-exposed areas of the head and neck

(This is a classic description of nodular BCC, often found on the nose.)

Question 254:
Wrong about squamous cell carcinoma?
75% of lesions are on extremities

High-Yield Context (from Dermatology Lecture Slides - Malignant Skin Tumours (Squamous cell carcinoma - Distribution), p.37 summary, p.59 lecture):

From Squamous cell carcinoma (SCC) - Distribution (p.37 summary, p.59 lecture):
•
Mostly in head and neck (70%)
• On
sun-exposed areas (dorsal hands, scalp, lip, and superior surface of pinna)*(While extremities (dorsal hands, forearms) are common sites due to sun exposure, the head and neck region accounts for the majority (around 70%). So, 75% on extremities is likely an overstatement and incorrect if head/neck is 70%.)*

Question 255:
Wrong about SCC:
a. Lower growth than BCC
b. Caused by exposure to sun
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Malignant Skin Tumours (SCC vs BCC), p.37 summary, p.59 lecture):

From Squamous cell carcinoma (SCC) (p.37 summary, p.59 lecture):
○ Symptoms →
rapidly growing,painful and hyperkeratotic
○ RF →
solar radiation,HPV and chronic scar is a RF... (b=true)

(SCC typically grows more rapidly and has a higher metastatic potential than BCC. BCC is generally slow-growing. So "Lower growth than BCC" for SCC is false.)

Question 256:
Melanoma with early metastasis:
Nodular melanoma

High-Yield Context (from Dermatology Lecture Slides - Malignant Skin Tumours (Melanoma - Types), p.37 summary, p.60 lecture):

From Types of melanoma (p.37 summary, p.60 lecture):
■ Superficial spreading melanoma → most common...
■ Lentigo maligna melanoma → on face,elderly
■
Nodular melanoma → dark nodule from the start, vertical growth (worse Px)
■ Acral melanoma→ palm and soles and near/under the nails

(Nodular melanoma is characterized by early vertical growth phase, which is associated with deeper invasion and higher risk of early metastasis compared to melanomas with a prolonged radial growth phase like superficial spreading melanoma or lentigo maligna melanoma.)

Question 257:
Worst type of MM:
Nodular

High-Yield Context: (Same as above - Nodular melanoma due to early vertical growth phase has a worse prognosis.)

Question 258:
Skin melanoma all of these are evidence-based prognostics except:
Gender

High-Yield Context (from Dermatology Lecture Slides - Malignant Skin Tumours (Melanoma - Prognosis), p.37 summary, p.61 lecture):

From Melanoma - Prognosis (p.37 summary, p.61 lecture):
○ Prognosis → depends on the
depth of invasion (assessed by Breslow) + LNs, ulcers, mets*(Key prognostic factors for melanoma include Breslow thickness (depth of invasion), ulceration, mitotic rate, lymph node involvement, and distant metastasis. While gender can be a demographic factor (e.g., females may have more leg melanomas, males more trunk), it's not considered a primary "evidence-based prognostic factor" in the same way as tumor thickness or presence of ulceration/metastasis in staging systems like AJCC.)*

Question 259:
Asymmetrical lesion, with ill-defined border, and variable shades of colors (diameter was not mentioned in the question), diagnosis:
a. Superficial spreading
b. Lentigo maligna
c. Nodular
d. Acral
e. Amelanotic
Answer: A

High-Yield Context (from general knowledge of ABCDE criteria for melanoma and melanoma subtypes; the summary describes these features for melanoma in general, p.37 lecture slides/p.60):*The ABCDE criteria for melanoma include Asymmetry, Border irregularity, Color variegation, Diameter >6mm, and Evolving. The description given (asymmetry, ill-defined border, variable colors) strongly suggests melanoma. Superficial Spreading Melanoma (SSM) is the most common type and often presents with these features in its radial growth phase.*

- **Superficial spreading melanoma (p.60 lecture):** "irregular margin, brown to black pigmentation , surrounding inflammation or pale, nodules (worse Px)"
- **Lentigo maligna melanoma (p.60 lecture):** "irregular, larger pigmented macule"

(The features described are classic for melanoma, and SSM is a very common type exhibiting these characteristics.)

Question 260:
Breslow thickness:
a. From granular layer to deepest point of invasion
b. From dermis to deepest point of invasion
c. Thickness in lymph nodes
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Malignant Skin Tumours (Melanoma - Prognosis, Breslow thickness image), p.37 summary, p.61 lecture):

From Melanoma - Prognosis (p.37 summary, p.61 lecture):
○ Prognosis → depends on the
depth of invasion (assessed by Breslow)...

(The image for Breslow Depth on slide p.61 shows the measurement from the top of the granular layer (or base of ulcer if ulcerated) to the deepest point of tumor invasion in the dermis/subcutis.)

Question 261:
The percentages of malignant melanomas are thought to arise from pigmented nevi:
a. 0-5%

b. 10-20%
c. 20-40%
d. 40-60%
e. 60-80%
Answer: C

High-Yield Context (from general epidemiology of melanoma, as specific percentage not in this summary):*A significant proportion of melanomas arise de novo (on previously normal skin), but a substantial percentage (often cited around 20-50%, varying in studies) arise from pre-existing nevi. The option c. 20-40% is a plausible range.*

Question 262:
Woman with unilateral, eczematous areolar rash, next step? *
Do skin biopsy

High-Yield Context (from Dermatology Lecture Slides - Malignant Skin Tumours (Paget's disease of the nipple), p.38 summary, p.63 lecture):

From Paget's disease of the nipple (p.38 summary, p.63 lecture):

- Presents with

unilateral non-specific erythematous changes on the areola/nipple spreading to the surrounding skin.

- The cause is an underlying adenocarcinoma of the ducts.
- It should be considered in any patient with

eczematous changes in one breast that fails to respond to simple treatment (do a biopsy)*(A unilateral, eczematous rash of the areola/nipple that doesn't respond to typical eczema treatment should raise suspicion for Paget's disease of the breast, and a skin biopsy is essential for diagnosis.)*

Question 263:
Which tumor is most frequently mets to skin:
a. Pulmonary CA
b. Renal CA
c. Prostate CA
d. Breast CA
e. Gastric CA
Answer: D

High-Yield Context (from Dermatology Lecture Slides - Malignant Skin Tumours (Cutaneous metastasis), p.38 summary, p.62 lecture):

From Cutaneous metastasis → Metastases from internal organs (p.38 summary, p.62 lecture):
○
breast, lung,GI tract, renal tract, oral pharynx, larynx and melanoma (originating from the retina and leptomeninges

(Breast cancer is one of the most common malignancies to metastasize to the skin in women. In men, lung cancer is often cited as most common.)

STDs

Question 264:
ttt of choice in all stages of syphilis is
a. Benzathine penicillin
b. Crystalline penicillin
c. Ampicillin
d. Prostaphyllin
Answer: A
(While crystalline penicillin is used for neurosyphilis, Benzathine penicillin G is the treatment of choice for primary, secondary, and early latent syphilis, as well as late latent and tertiary syphilis without neurosyphilis. For "all stages" in a general sense, penicillin is key, and benzathine penicillin covers most.)

High-Yield Context (from Dermatology Lecture Slides - STDs (Syphilis - Treatment), p.41 of lecture slides):

From Treatment → Benzathine penicillin (p.41 lecture):

- Primary + Secondary + Early latent: → 2-4 IU *1 IM

- Late latent + tertiary→ 2-4 IU *1 \ week for 3 wks
- neuro or ocular manifestations → 4 IU Q 4 hrs for 14 days . (This part implies Crystalline Penicillin G for neuro/ocular)

(Penicillin is the drug of choice. Benzathine penicillin G IM is used for most stages. Crystalline penicillin G IV is for neurosyphilis and congenital syphilis with CNS involvement.)

Question 265:
Most sensitive & specific test for early dx of syphilis is:
a. FTA
b. TFI
c. HR
d. FTA-ABS
Answer: D
(FTA-ABS (Fluorescent Treponemal Antibody Absorption) is a specific treponemal test that becomes positive early in infection and usually remains positive for life. Dark field microscopy of a chancre lesion is the most direct way to diagnose early primary syphilis if available and lesion is present, but among serological tests, treponemal tests like FTA-ABS become positive before non-treponemal tests fully react.)

High-Yield Context (from Dermatology Lecture Slides - STDs (Syphilis - Diagnosis), p.40 of lecture slides):
From Diagnosis (Primary syphilis) (p.40 lecture):
1-
Dark field microscopy (most sensitive)
2- RPR /VDRL → Rapid Plasma Reagin\ Venereal disease research laboratory test
3-
Treponemal Ab (less sensitive +ve only in 80%)

(The summary lists Dark Field as most sensitive for primary. Treponemal Ab tests like FTA-ABS or TPPA are highly specific and become positive early.)

Question 266:
Commonest serological test used in follow up at syphilitic pt:
a. FTA
b. TPI
c. WR
d. RPCF
e. FTA-ABS
Answer:
(The question seems to intend "WR" (Wassermann Reaction, an old complement fixation test) or "RPCF" (Reiter Protein Complement Fixation) as options for non-treponemal tests, as these are used for follow-up. However, the answer is E (FTA-ABS). Treponemal tests like FTA-ABS usually remain positive for life even after successful treatment and are NOT used to monitor treatment response. Non-treponemal tests (like VDRL or RPR) are used for follow-up, as their titers decrease with successful treatment. This question or answer is problematic.)If the question meant "Commonest serological test used INITIALLY at syphilitic pt for DIAGNOSIS", then a treponemal test like FTA-ABS would be common (often in conjunction with a non-treponemal). But for follow-up, non-treponemal tests are used.

High-Yield Context (from general knowledge of syphilis testing, as follow-up testing strategy is not detailed in this summary):*Non-treponemal tests (VDRL, RPR) are used to monitor treatment response because their titers usually decline after successful therapy. Treponemal tests (FTA-ABS, TPPA) typically remain positive for life and are not useful for monitoring response.*

Question 267:
If dark field examination fails to reveal spirochetes from a penile chancre then Dx may established alternatively by:
a. Darkfield examination of blood
b. Darkfield examination of blood from aspirate from regionally enlarged LN
c. TIT (treponemal immobilization test)
d. Any of the above
Answer: D
(Darkfield can be done on LN aspirates. TIT (Treponemal Immobilization Test) is an older, specific treponemal serological test. Darkfield of blood is generally not useful as spirochetemia is low.)

High-Yield Context (from Dermatology Lecture Slides - STDs (Syphilis - Diagnosis), p.40 of lecture slides):

From Diagnosis (Primary syphilis) (p.40 lecture):
1-
Dark field microscopy (most sensitive) (of chancre exudate)
2- RPR /VDRL
3-
Treponemal Ab*(If direct darkfield from chancre fails, serology (treponemal and non-treponemal tests) is the next step. Darkfield from LN aspirate is possible but less common than serology. TIT is a treponemal test.)*

Question 268:
Moth-eaten alopecia is found in:
a. Secondary syphilis
b. Primary syphilis
c. Tertiary syphilis
Answer: A

High-Yield Context (from Dermatology Lecture Slides - STDs (Syphilis - Secondary), p.40 of lecture slides):

From Secondary syphilis (p.40 lecture):
- ...
-
patchy alopecia may complicate the infection. (This is the "moth-eaten" alopecia).

Question 269:
All seen in secondary syphilis except:
a. Patchy scaring alopecia
b. Moth eaten alopecia
c. Asymmetric body rash
Answer: A

High-Yield Context (from Dermatology Lecture Slides - STDs (Syphilis - Secondary), p.40 of lecture slides):

From Secondary syphilis (p.40 lecture):
- widespread eruption of red-brown scaly

patches and macules that affects the trunk and limbs (particularly palms and soles) *(The rash is typically widespread and symmetrical, though can have variations.)*
-
patchy alopecia may complicate the infection. (This is non-scarring).

(The alopecia of secondary syphilis is non-scarring. The rash is generally symmetrical.)

Question 270:
All about 2ry syphilis true except:
a. Never itches
b. Contagious
c. STD (serologic test for syphilis) is +ve in 1OO% of cases
d. Most commonly vesicular
e. Presents with generalized rash
Answer: D

High-Yield Context (from Dermatology Lecture Slides - STDs (Syphilis - Secondary), p.40 of lecture slides):

From Secondary syphilis (p.40 lecture):
- widespread eruption of red-brown scaly

patches and macules ... (e=true)
- Condylomata lata one of the forms of secondary syphilis (moist colored skin) (b=true, lesions are highly contagious)
-
(Rash is typically non-pruritic, though some itching can occur (a=mostly true)).
-
(Serological tests (both non-treponemal and treponemal) are virtually always positive in secondary syphilis (c=true)).(The rash of secondary syphilis is typically maculopapular or papulosquamous, not primarily vesicular.)

Question 271:
All about syphilis true except:
a. Incubation period 9-90 days
b. Rx of choice is crystalline penicillin
Answer: B

High-Yield Context (from Dermatology Lecture Slides - STDs (Syphilis - Incubation, Treatment), p.39-41 of lecture slides):

From Syphilis - Incubation period (p.39 lecture):
- Incubation period of most STD is less than 7-10 days EXCEPT:
  - 1-

Syphilis up to 3 months (9-90 days) (a=true)

From Treatment (p.41 lecture):
●
Benzathine penicillin
- Primary + Secondary + Early latent: → 2-4 IU *1 IM
- Late latent + tertiary→ 2-4 IU *1 \ week for 3 wks
- neuro or ocular manifestations → 4 IU Q 4 hrs for 14 days . (implies Crystalline Penicillin G for these specific severe forms)

(Benzathine penicillin G is the treatment of choice for most stages of syphilis. Crystalline penicillin is reserved for neurosyphilis and some cases of congenital syphilis.)

Question 272:
Associated with mucous patch:
a. 2ry syphilis
b. 1ry syphilis
c. 3ry syphilis
d. Gonorrhea
Answer: A

High-Yield Context (from general knowledge of syphilis manifestations, as mucous patches are not explicitly in this summary but are a classic sign of secondary syphilis):*Mucous patches are painless, grayish-white, slightly raised erosions on mucous membranes (oral, genital) that are highly infectious and characteristic of secondary syphilis.*

Page 35 (Test Bank - STDs cont.)

Question 273:
Which about secondary syphilis is incorrect:
a. Lesions usually appear 6-16 weeks after infection
b. Lesions usually involves palms & soles
c. Most lesions contain spirochetes
d. Lymphadenopathy is usually absent
e. Lesions seldom itch
Answer: D

High-Yield Context (from Dermatology Lecture Slides - STDs (Syphilis - Secondary), p.40 of lecture slides):

From Secondary syphilis (p.40 lecture):
- widespread eruption of red-brown scaly patches and macules that affects the trunk and limbs (**particularly palms and soles**) (b=true)
- Condylomata lata one of the forms of secondary syphilis (moist colored skin)

(These lesions are highly infectious, containing spirochetes) (c=true)
●
(Generalized lymphadenopathy is a common feature of secondary syphilis.) (d=false)
●
(Lesions are typically non-pruritic, or seldom itch) (e=true)
●
(Appears weeks to a few months after the chancre, so 6-16 weeks after infection is plausible) (a=true)

(Generalized lymphadenopathy is characteristic of secondary syphilis.)

Question 274:
One is false about secondary syphilis manifestation:
a. Auditory neuritis
b. Periostitis
c. Polyhedral asymmetrical rash
d. Painful lymphadenopathy
Answer: D
(The rash of secondary syphilis is typically widespread and symmetrical, though it can be pleomorphic ("polyhedral" is unusual terminology). Lymphadenopathy is common and usually non-tender or mildly tender, not typically "painful" unless secondarily inflamed. Auditory/Ocular involvement (neuritis) and periostitis can occur in secondary syphilis.)

High-Yield Context (from general knowledge, as specific organ involvement details are not in this summary):*Secondary syphilis is a systemic disease. The rash is typically symmetrical. Lymphadenopathy is generalized and usually painless or minimally tender.*

Question 275:
Condolymata Lata in:
a. Viral warts
b. 1ry syphilis
c. 2ry syphilis
d. 3ry syphilis
Answer: C

High-Yield Context (from Dermatology Lecture Slides - STDs (Syphilis - Secondary), p.40 of lecture slides):

From Secondary syphilis (p.40 lecture):
●
Condylomata lata one of the forms of secondary syphilis (moist colored skin)

(Condylomata lata are broad, flat, moist, wart-like lesions found in intertriginous areas in secondary syphilis. Condylomata acuminata are viral warts caused by HPV.)

Question 276:
In classical syphilitic chancre all of the following statement are true except:
a. Occur at site of inoculation
b. Commonly single
c. Commonly painful
d. Considered an allergic reaction
e. Rich with treponemes
Answer: C

High-Yield Context (from Dermatology Lecture Slides - STDs (Syphilis - Primary), p.40 of lecture slides):

From Primary syphilis (p.40 lecture):
- Chancre ;

painless genital ulcer at the site of inoculation (a=true, c=false)
- Solitary, painless, firm, indurated ulcer... (b=true, commonly single)
- Dark field microscopy (most sensitive)

(implies rich with treponemes) (e=true)

(A chancre is a primary lesion due to direct infection, not an allergic reaction (d=false, but C is more classically false). The classical chancre is painless.)

Question 277:
In blood at normal refrigerator temperature (+4c) Treponema pallidum dies within:
a. 24hrs
b. 72-92hrs
c. 48hrs

d. 1 week
Answer: C

High-Yield Context (from general microbiology/transfusion medicine knowledge; T. pallidum is fragile outside the body):*Treponema pallidum is very sensitive to cold and drying. In stored blood for transfusion, it typically does not survive beyond 48-72 hours at refrigerator temperatures (1-6°C). The exact time can vary, but 48 hours is a commonly cited viability limit under these conditions.*

Question 278:
The best site to take a swab for gonorrhea is:
a. Labia minora
b. Labia majora
c. Anus
d. Endocervical swab
e. Vaginal wall
Answer: D

High-Yield Context (from general knowledge of gonorrhea diagnosis in females, as specific swabbing sites are not detailed in this summary for gonorrhea which is on p.41 of lecture slides):*In females, the endocervix is the preferred site for diagnosing gonococcal infection as it is commonly infected and columnar epithelium is susceptible. Urethral swabs can also be taken. Vaginal swabs are less sensitive as the vagina is lined by squamous epithelium, which is less susceptible to gonococcal infection.*

Question 279:
One is false about gonococcus:
a. Caused by G-ve diplococci
b. Female 50% are asymptomatic
c. Require therapeutic low level of penicillin for long time
d. Columnar epithelium is site of predilection
Answer: C

High-Yield Context (from Dermatology Lecture Slides - STDs (Gonorrhea), p.41 of lecture slides):

From Gonorrhea (p.41 lecture):

-

Gram negative diplococci, intracellular bacteria. (a=true)

- Incubation period: 2-5 days .
- -presentation :

Females
-
50% are asymptomatic (b=true)
- Urethritis ...
Endocervicitis ... Salpingitis

(Columnar epithelium (e.g., endocervix, urethra, rectum, pharynx) is the preferred site of infection for Neisseria gonorrhoeae (d=true). Penicillin resistance in gonococcus is widespread, and current treatments involve combination therapy with agents like ceftriaxone and azithromycin. High-dose penicillin was used in the past but is no longer reliable due to resistance; "low level for long time" is not the approach and not effective for resistant strains.) (c=false)

Question 280:
Majority of gonococcus strains are sensitive to penicillin concentration of (u/ml blood):
a. .002
b. 1.1
c. .03
d. .25
Answer: C
(This question refers to historical penicillin sensitivity before widespread resistance. Highly sensitive strains were inhibited by very low concentrations (e.g., ≤0.03-0.06 µg/mL or units/mL). With increasing resistance, higher concentrations were needed. The answer C (0.03) suggests a cutoff for "sensitive" strains.)

High-Yield Context (from historical microbiology of *N. gonorrhoeae* and penicillin susceptibility):*Before the era of widespread penicillin resistance, many gonococcal strains were highly susceptible to penicillin, with MICs (Minimum*

Inhibitory Concentrations) often below 0.1 µg/mL (or units/mL). An MIC of 0.03 u/mL would represent a very sensitive strain.

Question 281:
Which of the following is the causative of Chancroid:
Haemophilus ducreyi

High-Yield Context (from Dermatology Lecture Slides - STDs (Chancroid), p.41 of lecture slides):

From Chancroid (p.41 lecture):

- Causative agent is

Haemophilus ducreyi

- Incubation period 3-10 days.
- Always symptomatic. (start as multiple painful purulent ulcer with undermined edges)
- Gram stain → chaining pattern characteristic to haemophilus ducreyi
- Tx → Ceftriaxone, Azithromycin

Page 36 (Test Bank - STDs cont.)

Question 282:
Drug of choice in ttt of non-gonococcal urethritis:
a. Septrin
b. Tetracycline
c. Penicillin
d. Spectinomycin
Answer: B

High-Yield Context (from general knowledge of NGU treatment, as it's not explicitly detailed in the provided summary which focuses on syphilis, gonorrhea, chancroid, LGV, and granuloma inguinale):*Non-gonococcal urethritis (NGU) is most commonly caused by Chlamydia trachomatis, followed by Mycoplasma genitalium and other organisms. First-line treatment often includes **azithromycin (a macrolide)** as a single dose or **doxycycline (a tetracycline)** for 7 days. Penicillin is not effective against Chlamydia or Mycoplasma. Spectinomycin was used for gonorrhea but has limited efficacy against Chlamydia. Septrin (co-trimoxazole) is not a primary choice.Given the options, Tetracycline (like doxycycline) is a standard treatment for NGU.*

Ichthyosis

Question 283:
Ichthyosis vulgaris wrong:
a. usually involves extensors
b. involves flexors
answer: B

High-Yield Context (from Dermatology Lecture Slides - Ichthyosis (Ichthyosis Vulgaris), p.42 of lecture slides):

From Ichthyosis Vulgaris (p.42 lecture):

- Autosomal dominant disorder.
- Most common type of all types of ichthyosis.
- Presentation
  - starts after the 2nd or 3rd months of age , sometimes it may delay up to or after the 1st year of life.
  - You may see dandruff on the head due to excessive scales
  - Commonly associated with Keratosis pilaris.
  - Involves the

extensors WITH sparing of flexors.

  - Minimal itching.

(Ichthyosis vulgaris characteristically spares the flexural areas (axillae, antecubital and popliteal fossae, groin).)

Question 284:
Wrong in ichthyosis vulgaris:
a. Most common type
b. Usually associated with keratosis piliaris

c. Present at birth
d. Sparing flexures
answer: C

High-Yield Context (from Dermatology Lecture Slides - Ichthyosis (Ichthyosis Vulgaris), p.42 of lecture slides):

From Ichthyosis Vulgaris (p.42 lecture):

- **Most common type** of all types of ichthyosis. (a=true)
- Commonly associated with **Keratosis pilaris.** (b=true)
- Presentation
  - **starts after the 2nd or 3rd months of age**, sometimes it may delay up to or after the 1st year of life. (c=false, not present at birth)
  - Involves the extensors WITH **sparing of flexors.** (d=true)

Question 285:
Which is associated with atopy:
a. Ichthyosis simplex
b. X-linked ichthyosis
c. Lamellar ichthyosis
d. Bullous ichthyosiform hyperkeratosis
e. Ichthyosis hystrix
Answer: A
(Ichthyosis vulgaris, often referred to as ichthyosis simplex in older terminologies or due to its relatively milder nature, is frequently associated with atopy (eczema, asthma, hay fever). This association is linked to filaggrin gene mutations, which are implicated in both ichthyosis vulgaris and atopic dermatitis.)

High-Yield Context (from general knowledge, as the specific association of ichthyosis types with atopy beyond Ichthyosis Vulgaris is not detailed in the summary, though filaggrin and ichthyosis vulgaris are mentioned on p.42 lecture notes for IV):*Ichthyosis vulgaris (sometimes called ichthyosis simplex) has a strong association with atopic dermatitis. Mutations in the filaggrin gene are common to both conditions.*

Question 286:
Congenital ichthyosis associated with renal agenesis and hernia:
a. X-linked
b. Vulgaris
Answer: A

High-Yield Context (from general knowledge of syndromic ichthyoses, as specific systemic associations are not detailed for each ichthyosis type in the provided summary):*X-linked recessive ichthyosis, caused by steroid sulfatase deficiency, can sometimes be associated with other findings if it's part of a contiguous gene deletion syndrome, but renal agenesis and hernia are not its primary classic associations. However, some rare syndromic ichthyoses or genetic syndromes that include ichthyosis might have renal or other systemic involvement. This question is very specific and may rely on knowledge beyond the summary.Without further context from the summary specifically linking these, it's hard to confirm. Generally, X-linked ichthyosis is known for corneal opacities and cryptorchidism, not primarily renal agenesis/hernia.*

Question 287:
One of the following condition leads to thickening of all skin layers:
a. Ichthyosis simplex
b. Ichthyosis hystrix
c. Ichthyosis nigricans
d. Ichthyosis congenital
Answer: C
(This question is difficult to answer definitively without more context or clarification. "Thickening of all skin layers" is vague. Ichthyoses primarily involve epidermal changes (hyperkeratosis). "Ichthyosis nigricans" is not a standard term for a type of ichthyosis; it might be confused with acanthosis nigricans, which involves epidermal thickening (acanthosis) and hyperkeratosis, giving a velvety, thickened appearance, but isn't a primary ichthyosis. Ichthyosis hystrix refers to severe,

often spiky hyperkeratosis. Ichthyosis simplex (vulgaris) has fine scales. Congenital ichthyoses (like lamellar or CIE) have marked scaling and hyperkeratosis.)If "Ichthyosis nigricans" is a typo for Acanthosis Nigricans, then AN does involve epidermal thickening. However, AN is not an ichthyosis. Given the options are ichthyoses, perhaps the question implies a severe form like harlequin ichthyosis (a severe congenital ichthyosis) where the entire skin is massively thickened, but "all skin layers" (including dermis and subcutis significantly) is still an overstatement for most ichthyoses.Given the likely options, this is problematic. If the intent was Acanthosis Nigricans (misnamed), it causes epidermal thickening.

Question 288:
Keratinization process is defective in all except:
a. Lichen sclerosis atrophicans
b. Ichthyosis hystrix
c. Psoriasis
d. Epidermolytic hyperkeratosis
e. Ichthyosis lamellaris
Answer: A

High-Yield Context (from various summary sections and general knowledge):

- **Ichthyosis hystrix, Epidermolytic hyperkeratosis, Ichthyosis lamellaris:** These are all disorders of keratinization (ichthyoses).
- **Psoriasis (p.5 summary):** Characterized by hyperkeratosis and parakeratosis (abnormal keratinization).
- **Lichen Sclerosus (et Atrophicus) (p.16 summary):** Characterized by epidermal atrophy, homogenization of dermal collagen, and an inflammatory infiltrate. While there are epidermal changes, the primary defect is not usually described as one of "keratinization" in the same way as ichthyoses or psoriasis, but rather an inflammatory and sclerotic process affecting the dermis and epidermis.

(Lichen sclerosus involves epidermal atrophy and dermal sclerosis, not primarily a defect in the keratinization process itself like in ichthyoses or the altered keratinocyte proliferation/differentiation of psoriasis.)

Question 289:
Bullous ichthyosis erythroderma is inherited as:
Autosomal dominant

High-Yield Context (from Dermatology Lecture Slides - Ichthyosis (Classification), p.42 of lecture slides):

From CONGENITAL ICHTHYOSIS (p.42 lecture):
1- ichthyosis Vulgaris
2- X- linked recessive ichthyosis.
3- NBIE (Non-Bullous ichthyosis Erythroderma)
4-
Bullous ichthyosiform Erythroderma .
5- lamellar ichthyosis

(The summary lists Bullous Ichthyosiform Erythroderma (also known as Epidermolytic Hyperkeratosis) but doesn't state its inheritance pattern on this page. General knowledge: Epidermolytic Hyperkeratosis (Bullous Congenital Ichthyosiform Erythroderma) is autosomal dominant, caused by mutations in keratin 1 or 10 genes.)

Question 290:
True about Non-bullous ichthyosiform erythroderma:
Autosomal recessive inheritance

High-Yield Context (from Dermatology Lecture Slides - Ichthyosis (Classification), p.42 of lecture slides):*(The summary lists NBIE (Non-Bullous ichthyosis Erythroderma). General knowledge: Congenital Ichthyosiform Erythroderma (CIE), which is a non-bullous form, is typically autosomal recessive.)*

Miscellaneous

Question 291:
Penetration of skin by UV light is greater from:
a. UVB
b. UVC

c. UVA
d. Not related to wavelength
e. Negligible
Answer: C

High-Yield Context (from general knowledge of UV radiation, as this specific detail isn't in the provided summary):*UV radiation is divided into UVA, UVB, and UVC.*

- **UVC (200-290 nm):** Mostly absorbed by the ozone layer, does not significantly reach Earth's surface. If it does, it's very superficial.
- **UVB (290-320 nm):** Penetrates the epidermis, is the primary cause of sunburn, and contributes to skin cancer and photoaging.
- **UVA (320-400 nm):** Penetrates deeper into the dermis than UVB. Contributes to photoaging, skin cancer, and can cause tanning.

  (UVA has longer wavelengths and penetrates deeper into the skin (dermis) compared to UVB (epidermis).)

Question 292:
When is the intensity of UVA highest during the day?
a. 8am
b. 3pm
c. 12pm
d. The level of UVA is actually constant throughout the day
e. 10am
Answer: C
(or D, depending on interpretation. While UVB peaks strongly around midday, UVA levels are more constant throughout daylight hours but are still generally highest when the sun is highest, i.e., around solar noon (12pm-2pm). "Constant throughout the day" (D) is an oversimplification but reflects that UVA is present for longer periods than peak UVB. If C (12pm) is the answer, it refers to the peak intensity.)

High-Yield Context (from general knowledge of UV radiation patterns):*UVB intensity peaks significantly between 10 a.m. and 4 p.m. UVA radiation is present throughout all daylight hours and its intensity is more consistent than UVB, but it still tends to be highest around midday when the sun's rays are most direct.*

Question 293:
When choosing or advising on the use of sunscreens the following points should be taken into account, except:
a. Physical sunscreen reflect UV radiation and are highly effective
b. Chemical sunscreen absorb UV radiation and cause less opaque/grey appearance when applied to the skin
c. In reality the level of sun protection is 1/3 of that specified on the sunscreen bottle
d. The SPF (numerical sun protection factor) is a measure of UVB protection
e. The letter rating (A-E) is a measure of UVA protection
Answer: E
(Sunscreen UVA protection rating systems vary by region. In Europe, a "UVA in a circle" logo or a star rating (e.g., Boots Star Rating in UK) is common. An A-E letter rating is not a universally standard or widely recognized system for UVA protection on sunscreens. SPF measures UVB. Physical sunscreens reflect/scatter. Chemical sunscreens absorb. The 1/3 protection claim is controversial and relates to difficulties in real-world application achieving lab-tested SPF.)

High-Yield Context (from general knowledge of sunscreens):

- a. Physical (mineral) sunscreens (zinc oxide, titanium dioxide) work by reflecting and scattering UV. (True)
- b. Chemical (organic) sunscreens absorb UV radiation and convert it to heat; often more cosmetically elegant. (True)
- c. The "1/3 rule" is not a universally accepted fact about SPF effectiveness, though real-world application often yields lower protection than lab SPF.
- d. SPF primarily measures protection against UVB. (True)
- e. UVA protection is indicated by other systems (e.g., PA+, Broad Spectrum label, UVA circle, star ratings). A simple A-E letter rating is not standard. (False as a universal system).

Question 294:
Side effect of prolonged topical corticosteroid therapy include all of the following except:

a. Atrophy of skin
b. Telangiectasia
c. Pigmentation
d. Overgrowth of hair
e. Freckling
Answer: E

High-Yield Context (from general knowledge of topical steroid side effects, as this is not detailed in a specific "miscellaneous" section of the summary):
*Prolonged use of topical corticosteroids can cause:

- **Skin atrophy** (thinning of epidermis and dermis) (a=true)
- **Telangiectasia** (dilated blood vessels) (b=true)
- **Hypopigmentation** or sometimes hyperpigmentation (c=true, though hypo is more classic with atrophy)
- **Hypertrichosis** (increased hair growth in the area of application, especially with potent steroids on the face) (d=true)
- Striae, easy bruising, acneiform eruptions, perioral dermatitis, tachyphylaxis, risk of systemic absorption with potent steroids over large areas/occlusion.

Freckling (ephelides) is related to sun exposure and genetics, not a side effect of topical steroids. Steroids can cause pigment changes, but not new freckles.

Question 295:
Topical steroids can cause all of following except:
a. Hair loss
b. Hypopigmentation
c. Rosacea
d. Atrophy
e. Cataract
Answer: A

High-Yield Context (from context above and general knowledge):

- **Hypopigmentation:** Can occur, especially with atrophy. (b=true)
- **Rosacea (Steroid Rosacea):** Prolonged use of topical steroids on the face can induce or worsen rosacea-like changes. (c=true)
- **Atrophy:** A common side effect. (d=true)
- **Cataract/Glaucoma:** Systemic absorption or direct application near the eyes of potent topical steroids can increase risk of glaucoma and cataracts. (e=true)
- **Hair loss (Alopecia):** Topical steroids are used to *treat* some forms of alopecia (like alopecia areata). While skin atrophy could theoretically affect follicles, primary hair loss is not a typical side effect; hypertrichosis is more common. (a=false as a common side effect).

Question 296:
Systemic steroids are often indicated in the treatment of the following conditions, except: **
a. Systemic vasculitis
b. Severe drug eruptions
c. Urticaria with significant angioedema
d. Widespread eczema
e. Widespread chronic plaque psoriasis
Answer: E

High-Yield Context (from various summary sections on treatment, e.g., Psoriasis p.6/p.150 lecture, Urticaria p.10/p.86 lecture, Drug Rashes p.13/p.31-33 lecture, Eczema p.170 lecture):

- **Systemic vasculitis:** Often requires systemic steroids. (True)
- **Severe drug eruptions (e.g., SJS/TEN, DRESS):** Systemic steroids are often used. (True for DRESS, controversial but sometimes used in SJS/TEN).

- **Urticaria with significant angioedema (p.86 lecture):** Oral corticosteroids may be indicated in very severe eruptions... (True)
- **Widespread eczema (p.170 lecture):** Rapidly reducing course of oral prednisolone ... to control very severe generalized acute eczema. (True for acute severe flares).
- **Widespread chronic plaque psoriasis (p.6 summary / p.150 lecture): Systemic corticosteroids should not be used to treat psoriasis.** (Due to risk of rebound/pustular psoriasis). (False)

Question 297:
Systemic steroid used in ttt of:
a. Nodular acne vulgaris
b. Ordinary acne vulgaris
c. Acne medicamenosa
d. Acne cosmotica
e. Acne fulminans
Answer: E

High-Yield Context (from Dermatology Lecture Slides - Acne (Acne conglobata/fulminans), p.21 summary, p.99 lecture):

From Acne conglobata/fulminans (p.21 summary, p.99 lecture):
- Severe, more in boys and tropical climates
- Extensive, nodulocystic acne and abscess formation
-

Fulminans → associated with systemic symptoms of malaise, fever and joint pains*(Acne fulminans is a rare, severe form of acne with abrupt onset, ulcerative lesions, and systemic symptoms like fever and arthralgia. Systemic corticosteroids are often a key part of its treatment, usually in combination with isotretinoin or oral antibiotics.)(Acne medicamentosa is caused by drugs, e.g., steroids. Ordinary acne vulgaris is not typically treated with systemic steroids unless extremely inflammatory as part of a short course, but not as primary therapy. Acne fulminans is the classic indication for systemic steroids in acne.)*

Question 298:
Phthirus pubis may be transmitted from the pubic hair to:
a. Scalp hair

(OCR ends. Other options would likely be other hairy areas like axillae, eyelashes, eyebrows.)

High-Yield Context (from Dermatology Lecture Slides - Scabies and head lice (Pubic lice), p.29 summary, p.94 lecture):

From Pubic lice "crab lice" (p.29 summary, p.94 lecture):
- pubic,

axillary and eyelash areas ⇒ check for sexually transmitted diseases
- Mgt → topical permethrin 5% to skin from the neck downwards...
- If

eyelashes involved → petrolatum only*(Phthirus pubis (crab louse) prefers coarse body hair. While primarily in the pubic region, it can also infest hair in the axillae, chest, beard, eyebrows, and eyelashes. Scalp hair is generally too fine and densely packed for P. pubis, which is adapted for coarser, more spaced hair. Head lice (Pediculus humanus capitis) infest scalp hair.)So, transmission to scalp hair is uncommon for P. pubis.*

Page 39 (Test Bank - Miscellaneous cont.)

Question 299 (continued from Page 38, question 8):
Phthirus pubis may be transmitted from the pubic hair to:
a. Scalp hair
b. Chest hair
c. Eye lashes
d. Eye brows
Answer: C
(The OCR shows C. As discussed, P. pubis infests coarse body hair. Eyelashes and eyebrows are common sites of ectopic infestation. Chest hair is also possible. Scalp hair is least likely.)

High-Yield Context: (Already covered under Page 38, Question 8).

Question 300:
All true about erythroderma except:
a. >80% of skin
b. Biopsy usually done
c. Hyperthermia and dehydration
Answer: A
(The OCR gives A. Erythroderma is defined as generalized erythema and scaling affecting >90% of the body surface area. So ">80% of skin" is true, not false. "Hyperthermia and dehydration" - dehydration is a major complication; temperature dysregulation leads to hypothermia due to heat loss, not typically hyperthermia unless there's a concurrent infection causing fever. Biopsy is often done to determine the underlying cause. So, "Hyperthermia" is likely the incorrect part of statement C. If A is the answer, it implies the >80% threshold is wrong, but >90% is standard, so >80% would still be true.)This question is problematic with the given answer A. Let's assume the standard definition of >90% BSA for erythroderma. Then ">80%" is still encompassed and true. The most incorrect part of the options given the choices (if we must pick one "except") might be "Hyperthermia" as hypothermia is more typical.

High-Yield Context (from general knowledge of erythroderma, as it's only briefly mentioned in Psoriasis section p.6):
*Erythroderma (exfoliative dermatitis) is generalized redness and scaling involving >90% of the body surface.
Complications include:

- Temperature dysregulation (usually **hypothermia** due to heat loss)
- Fluid and electrolyte imbalance (**dehydration**)
- Protein loss
- High-output cardiac failure
- Increased risk of infection/sepsis

A skin biopsy is crucial to help identify the underlying cause (e.g., psoriasis, eczema, drug reaction, lymphoma).If the answer is A, it implies >80% is not the definition. If >90% is the strict definition, then a statement of ">80%" is less precise but not strictly "false" if the actual involvement is, say, 95%. The "Hyperthermia" part of C is more definitively wrong than A.

Question 301:
Not associated with erythroderma:
a. malignancy
b. Lichen Planus
c. Psoriasis
d. Congenital ichthyosis
e. Drug induced
Answer: B

High-Yield Context (from general knowledge of causes of erythroderma):*Common causes of erythroderma include:*

- Pre-existing dermatoses: **Psoriasis**, atopic dermatitis, seborrheic dermatitis.
- **Drug reactions** (e.g., DRESS, or severe reactions to allopurinol, sulfonamides, anticonvulsants).
- **Malignancy:** Cutaneous T-cell lymphoma (Sézary syndrome), other lymphomas/leukemias.
- Idiopathic.
- Rarely, severe **congenital ichthyoses** (e.g., Congenital Ichthyosiform Erythroderma).

Lichen planus can very rarely become erythrodermic, but it's not a common or typical cause compared to psoriasis, eczema, or drug reactions.

Question 302:
One may cross normal placenta:
a. IgG
b. IgM
c. IgA
d. IgE

e. All Ig
Answer: A

High-Yield Context (from general immunology knowledge):*Immunoglobulin G (IgG) is the only class of antibody that can cross the placenta from mother to fetus, providing passive immunity to the newborn. This is facilitated by the neonatal Fc receptor (FcRn) on placental cells. IgM, IgA, IgE, and IgD do not significantly cross the placenta.(This is relevant to NLE where maternal IgG autoantibodies (anti-Ro/La) cross the placenta, p.17 summary).*

Question 303:
Dermatoscope, what is wrong:
a. used for seeing hyphae and spores
b. used for pigmented lesions
c. used for alopecia areata
d. Hand-held tool
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Diagnosis of dermatologic disease (Dermoscope), p.20 summary/p.114 lecture):

From Dermoscope (p.20 summary / p.114 lecture):
A non-invasive, diagnostic tool which visualizes
subtle clinical patterns of skin lesions and subsurface skin structures not normally visible to the unaided eye.

(Dermoscopy (dermatoscopy) is primarily used for the evaluation of pigmented skin lesions (to differentiate benign nevi from melanoma) and non-pigmented skin tumors. It can also be helpful in diagnosing other conditions like scabies (visualizing the mite/burrow), hair and scalp disorders (trichoscopy, e.g., alopecia areata - exclamation mark hairs, yellow dots), inflammatory dermatoses, and some infections. It's a hand-held tool. While it magnifies the skin surface, directly visualizing fungal hyphae and spores typically requires microscopy of a KOH preparation, not just dermoscopy, though dermoscopy might show suggestive scale patterns in tinea.)

Question 304:
Drug of choice for cutaneous leishmaniasis:
a. Tetracycline
b. Erythromycin
c. Antimonials
d. Sulfones
Answer: C

High-Yield Context (from general knowledge of Leishmaniasis treatment, as it's not covered in the provided summary):
*Treatment of cutaneous leishmaniasis (CL) depends on the species, extent and location of lesions, and host immune status.

- For simple, uncomplicated Old World CL, topical treatments or no treatment (as some heal spontaneously) may be options.
- For more complex, New World, or mucocutaneous leishmaniasis, systemic therapy is usually required.
- **Pentavalent antimonials (e.g., sodium stibogluconate, meglumine antimoniate)** have been the mainstay of systemic treatment for many years, although toxicity and resistance are concerns.
- Other options include amphotericin B, miltefosine, paromomycin (topical or systemic), and azoles (e.g., ketoconazole, fluconazole, itraconazole) for some species.

Sulfones (like dapsone) and antibiotics (tetracycline, erythromycin) are not primary treatments for leishmaniasis.

Question 305:
A child from Jordan valley developed a painless ulcer on his face on the site of a mosquito bite, mostly?
Leishmaniasis (it should be a fly not a mosquito)

High-Yield Context (from general knowledge of Leishmaniasis):*Cutaneous leishmaniasis is transmitted by the bite of infected female phlebotomine **sand flies** (not mosquitoes). It typically presents as a papule at the bite site that enlarges and often ulcerates, forming a painless ulcer with raised borders. The Jordan Valley is an endemic area for cutaneous leishmaniasis (caused by Leishmania tropica or L. major).*

Question 306:
Leishmaniasis recidivans is due to:
a. L. Donovani
b. L. tropica
c. L. brazilience
d. Allergic reaction to L. body
Answer: B

High-Yield Context (from general knowledge of Leishmaniasis recidivans):*Leishmaniasis recidivans (lupoid leishmaniasis) is a chronic, relapsing form of cutaneous leishmaniasis that typically occurs around the scar of a healed primary lesion, often years later. It is most commonly associated with* ***Leishmania tropica*** *and is thought to represent a state of persistent, low-grade infection with a strong cell-mediated immune response.L. donovani causes visceral leishmaniasis. L. braziliensis causes mucocutaneous leishmaniasis.*

Question 307:
Cutaneous leishmaniasis is an infection of the:
a. cutaneous fat
b. Str. malpighia
c. R.E. cells
d. Mast cells
e. Langerhans cells
Answer: C

High-Yield Context (from general knowledge of Leishmaniasis pathogenesis):*Leishmania parasites are obligate intracellular protozoa that infect* ***macrophages*** *(part of the reticuloendothelial system - R.E. cells) of the host. After inoculation by sand fly bite, promastigotes are phagocytosed by macrophages, where they transform into amastigotes and multiply, eventually rupturing the macrophage to infect new cells. Langerhans cells (epidermal dendritic cells/macrophages) can also be initially infected.Stratum Malpighii (stratum basale and spinosum of epidermis) is not the primary site of infection. Cutaneous fat and mast cells are not primary targets.*

Question 308:
Eye involvement may occur in all except:
a. Intermediate leprosy
b. Lepromatous leprosy
c. Rosacea
d. Sarcoid (Page 40)
e. Bechet’s syndrome (Page 40)
Answer: A
(The OCR gives A. Ocular involvement is well-documented in lepromatous leprosy (common), rosacea (ocular rosacea), sarcoidosis (uveitis, conjunctival granulomas), and Behçet's syndrome (uveitis, retinal vasculitis). Leprosy is a spectrum; while lepromatous (multibacillary) has more widespread involvement, borderline forms can also have eye issues. "Intermediate leprosy" is not a standard classification term; it might refer to borderline forms or indeterminate leprosy. Indeterminate leprosy is early and often self-healing with minimal manifestations.)

High-Yield Context (from Rosacea p.103 lecture, and general knowledge of the other conditions):

- **Rosacea (p.103 lecture - Clinical types):** 4. **Ocular**: dry gritty, eyelid edema, blepharitis, conjunctivitis.
- **Leprosy:** Ocular complications are common, especially in multibacillary (lepromatous, borderline lepromatous) forms, due to direct infiltration, nerve damage (lagophthalmos, corneal anesthesia), and immune reactions.
- **Sarcoidosis:** Ocular involvement is common (uveitis, conjunctival nodules, lacrimal gland involvement).
- **Behçet's syndrome:** Ocular inflammation (uveitis, retinal vasculitis) is a major criterion and can lead to blindness.

  (If "Intermediate leprosy" refers to early indeterminate leprosy, eye involvement would be less likely or absent compared to the others.)

Page 40 (Test Bank - Miscellaneous cont. & BEST OF LUCK)

Question 309: (continued from Page 39, question 10d,e):
Eye involvement may occur in all except:
d. Sarcoid
e. Bechet’s syndrome

Answer: A
(As discussed above, the answer A refers to "Intermediate leprosy" from page 39.)

High-Yield Context: (Already covered under Page 39, Question 10).

Question 310:
ttt of lepromatous leprosy should be continued for (yrs):
a. 2
b. 5
c. 10
d. Life long
Answer: A

High-Yield Context (from WHO guidelines for leprosy treatment, as it's not in the provided summary):
*The WHO recommends multidrug therapy (MDT) for leprosy.

- **Paucibacillary (PB) leprosy:** Rifampicin and Dapsone for 6 months.
- **Multibacillary (MB) leprosy (which includes lepromatous leprosy):** Rifampicin, Clofazimine, and Dapsone for **12 months.***Some national programs or older regimens might have used 24 months (2 years) for MB leprosy, but the standard WHO recommendation is now 12 months. "Lifelong" is incorrect. 5 or 10 years is also incorrect for standard MDT completion.Given the options, and if older guidelines were considered where 24 months was sometimes used for MB, then A (2 years) might be chosen. However, current standard is 12 months for MB.*

Question 311:
A lady presents with hyperpigmented lesion on her face that has been increasing in size, she is worried about it. most appropriate next step is:
a. Dermoscopic examination
b. Incisional biopsy
c. Excisional biopsy
d. Chemical peeling
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Diagnosis of dermatologic disease (Dermoscope) p.20 summary/p.114 lecture, and principles of evaluating pigmented lesions):

From Dermoscope (p.20 summary / p.114 lecture):
A non-invasive, diagnostic tool which visualizes
subtle clinical patterns of skin lesions and subsurface skin structures not normally visible to the unaided eye.

(For a hyperpigmented lesion on the face that is changing (increasing in size) and causing concern, the first step in evaluation by a dermatologist is often dermoscopy. Dermoscopy helps to assess features suggestive of benignity or malignancy (e.g., melanoma, pigmented BCC). Based on dermoscopic findings, a decision can be made regarding reassurance, monitoring, or biopsy (incisional or excisional). Chemical peeling is a cosmetic procedure and not a diagnostic first step for a concerning, changing lesion.)

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018 EXAM

Question 312:
Not part of the normal flora?
a. Group A Strep
b. Diphtheroid
c. Staph aureus
d. Corynebacterium diphtheriae
e. Coagulase negative Staph
Answer: C
(The OCR answer is C - Staph aureus. While S. aureus can be carried on the skin or in the nares of healthy individuals (carrier state), it's also a significant pathogen. Group A Strep (Streptococcus pyogenes) can also be carried (e.g., in the throat) but is primarily a pathogen. Diphtheroids and Coagulase-negative Staph are definitively normal flora. Corynebacterium diphtheriae is the cause of diphtheria and not normal skin flora. The question asks "Not part of the normal flora". S. aureus is often considered part of the transient or intermittent flora, and can be a carrier state, but it's

also a major pathogen. C. diphtheriae is definitely not normal skin flora. If the answer key says C, it's focusing on S. aureus's pathogenic potential over its carrier status.)

High-Yield Context (from Dermatology Lecture Slides - Bacterial skin infections (Normal skin flora) p.22 summary/p.114 lecture):

From Bacterial skin infections - Normal skin flora (p.22 summary, p.114 lecture):

- Normal skin flora consists of:

Coagulase-negative Staphylococcus, **Corynebacterium**, **Diphtheroids**, and α-haemolytic Streptococci in the epidermis.
 Propioni- bacterium in the pilosebaceous unit.

- Nasal swabs: May identify

Staphylococcus aureus carriers (MRSA)...

(This confirms Coagulase-negative Staph, Corynebacterium, and Diphtheroids are normal flora. It also mentions S. aureus carrier state. Group A Strep and C. diphtheriae are not listed as typical normal skin flora in this summary.)

Question 313:
Not seen in Tinea capitis ?
a. Exclamation mark sign
b. Scales
c. Focal alopecia
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections (Tinea capitis) p.27 summary/p.106-107 lecture, and Hair and Scalp (Alopecia Areata) p.29 summary/p.64 lecture):

From Tinea capitis (p.27 summary / p.107 lecture):

- Clinically, features are highly variable: diffuse

scaling, grey patches, black dots (broken-off hairs), multiple pustules, **patchy alopecia**, extensive alopecia with inflammation, kerion formation... (b and c are seen)

From Alopecia areata "AA" (p.29 summary / p.64 lecture):

○
Exclamation mark hair " narrower closer to the scalp" is **diagnostic for AA***(Exclamation mark hairs are characteristic of Alopecia Areata, not Tinea Capitis. Tinea capitis causes scaling and focal alopecia ("patches of alopecia").)*

Question 314:
Which of the following STDs is matched incorrectly with its causative pathogen ?
a. Syphilis – Treponema Pallidum
b. Gonorrhea – Neisseria
c. Lymphogranuloma venereum – Hemophilus ducreyi
d. Genital warts – HPV
Answer: C

High-Yield Context (from Dermatology Lecture Slides - STDs (various sections), pp.39-41 of STD lecture slides):

- **a. Syphilis – Treponema Pallidum (p.39):** -Causative agent is spirochete **Treponema Pallidum** (Correct)
- **b. Gonorrhea – Neisseria (p.41):** - Gram negative diplococci... (*Neisseria gonorrhoeae*) (Correct)
- **c. Lymphogranuloma venereum – Hemophilus ducreyi:**
  - **From Lymphogranuloma Venereum (LGV) (p.41):** - Causative agent is **Chlamydia trachomatis serotype L1-L3**
  - **From Chancroid (p.41):** - Causative agent is **Haemophilus ducreyi**
  - (Incorrectly matched)
- **d. Genital warts – HPV (p.39):** Human papillomavirus **HPV** ... Clinical presentation of genital warts ... condyloma acuminatum (Correct)

(Lymphogranuloma venereum is caused by Chlamydia trachomatis serovars L1-L3. Haemophilus ducreyi causes Chancroid.)

Question 315:
Which statement is incorrect :

a. Nickel is the most common allergen worldwide
b. Balsam of Peru is used in pigments
c. Pityriasis alba is a type of atopic dermatitis
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Contact dermatitis (Common allergens) p.165, Atopic Dermatitis (Associated features) p.159):

- **a. Nickel is the most common allergen worldwide (p.165): Nickel/cobalt** (jewellery, clothing, wristwatch, scissors and cooking utensils). *(Nickel is very widely recognized as one of the most common contact allergens.)* (Likely True)
- **b. Balsam of Peru is used in pigments:**
  - **From Common contact allergens (p.165):** Potassium dichromate (chemical used to tan leather; Figure 4.17), chrome, **myroxylon pereirae (balsam of Peru, fragrance;** Figure 4.20).
  - *(Balsam of Peru is primarily known as a fragrance allergen and is found in perfumes, cosmetics, and some topical medications. It's not typically used "in pigments" directly, though it might be in a product that also contains pigments.)* (Likely Incorrect as its primary use)
- **c. Pityriasis alba is a type of atopic dermatitis (p.159): Pityriasis alba: variant of atopic eczema** in which pale patches of hypopigmentation develop on the face of children (True)

(Balsam of Peru is primarily a fragrance component, not a pigment.)

Question 316:
Which statement is correct:
a. Necrobiosis lipoidica is commonly associated with DM
b. Lofgren syndrome has a poor prognosis
Answer: A

High-Yield Context (from Dermatology Lecture Slides - The Skin and Systemic Disease (Necrobiosis Lipoidica) p.19 summary/p.40 lecture, and Sarcoidosis (Löfgren syndrome) p.39 lecture):

- **a. Necrobiosis lipoidica is commonly associated with DM (p.19 summary / p.40 lecture):**
  - between 40% and 60% of patients with this condition may develop diabetes...
  - Associated with **DM (especially type 1)**
  - (True)
- **b. Lofgren syndrome has a poor prognosis (p.39 lecture):**
  - Löfgren syndrome: • Acute • Multisystemic • **Benign, good px, resolves alone**
  - (False, Lofgren syndrome generally has a good prognosis and often resolves spontaneously.)

Question 317:
Choose the mismatch :
Answer: Patch test – irritant contact dermatitis

High-Yield Context (from Dermatology Lecture Slides - Eczema (Contact Dermatitis - Patch testing), p.168):

From Patch testing (p.168):

- Patch testing is used to determine the substances that cause

allergic contact dermatitis.*(Patch testing is specifically for identifying Type IV hypersensitivity reactions causing allergic contact dermatitis. For irritant contact dermatitis, the diagnosis is usually clinical based on exposure history and morphology, as everyone would react to a strong enough irritant; patch testing is not the primary diagnostic tool for irritancy.)*

Question 318:
Which of these drugs is used in scabies:
a. Benzyl peroxide
b. Benzyl benzoate
c. Tetracyclines
Answer: B

High-Yield Context (from Dermatology Lecture Slides - Scabies and head lice (Scabies - Management), p.28 summary, p.91 lecture):

From Management (of scabies) (p.28 summary / p.91 lecture):

1. Permethrin cream 5%
2. Malathion lotion 0.5%
3. Ivermectin: (oral)
   • If permethrin and malathion are not available, then >> 10% sulphur in yellow soft paraffin is effective and safe.
   • 25%
   benzyl benzoate emulsion may also be used.

   (Benzyl peroxide is for acne. Tetracyclines are antibiotics. Benzyl benzoate is a recognized treatment for scabies.)

Question 319:
Choose the mismatch :
Answer: Henoch Schoenlein purpura – IgG antibodies

High-Yield Context (from Dermatology Lecture Slides - Connective Tissue Disease, Vasculitis (Henoch–Schönlein purpura), p.8 of CTD deck, p.4 lecture):

From Henoch–Schönlein purpura (p.8 CTD deck / p.4 lecture):

- Pathophysiology: Deposition of
IgA, complement and immune complexes in small vessels...
- Dx→ Skin/renal biopsy → deposition of
IgA*(Henoch-Schönlein Purpura (HSP), now often called IgA Vasculitis, is characterized by the deposition of IgA-containing immune complexes.)*

Question 320:
Correct sentence:
Answer: In neonatal lupus, the lesions subside when the antibodies drop

High-Yield Context (from Dermatology Lecture Slides - Connective Tissue Disease, Vasculitis (Neonatal lupus erythematosus), p.9 of CTD deck, p.17 summary/p.11 lecture):

From Neonatal lupus erythematosus (NLE) (p.9 CTD deck / p.17 summary):

- **transplacental passage of anti Ro/La** (maternal IgG antibodies)
- Annular scaly lesions on the face/scalp
- Risk congenital heart block

From NLE (p.11 lecture):

- Skin lesions may require topical steroids and sunscreen but
usually resolve spontaneously as the level of autoantibody depletes.*(The cutaneous lesions of neonatal lupus are caused by passively transferred maternal autoantibodies (anti-Ro, anti-La, which are IgG). These antibodies are gradually cleared from the infant's circulation over several months, and the skin lesions typically resolve accordingly.)*

Question 321:
Choose the correct statement regarding bullous pemphigoid :
a. Antibodies against hemidesmosomes
b. Positive nekolsky sign
c. The bullae are flaccid
d. Mucous membranes are commonly involved
Answer: A

High-Yield Context (from Dermatology Lecture Slides - Bullous Diseases (Bullous Pemphigoid), p.13-15 summary, pp.70-72, pp.75-77 lecture):

- **a. Antibodies against hemidesmosomes (p.13 summary / p.70 lecture):**
  - Bullous pemphigoid → IgG autoantibodies that target the basement membrane cells (**hemidesmosome proteins BP180 and BP230**). (Correct)
- **b. Positive Nikolsky sign (p.76 lecture - Bullous Pemphigoid):**
  - Nikolsky’s sign is **negative.** (Positive Nikolsky sign is seen in Pemphigus Vulgaris)
- **c. The bullae are flaccid (p.76 lecture - Bullous Pemphigoid):**

- The bullae are **tense** with good structural integrity. (Flaccid bullae are seen in Pemphigus Vulgaris)

- **d. Mucous membranes are commonly involved (p.76 lecture - Bullous Pemphigoid):**
  - Mucous membrane involvement occurs in about **20% of cases.** (Less common and less severe than in Pemphigus Vulgaris, where it's often the presenting feature and more extensive). So, "commonly" might be an overstatement compared to Pemphigus.

Question 322:
Incorrect statement :
Answer: Erythema nodusum is due to dermal inflammation

High-Yield Context (from Dermatology Lecture Slides - The Skin and Systemic Disease (Erythema Nodosum), p.9 of separate slide deck, p.18 summary):

From Erythema Nodosum (EN) (p.9 Systemic Disease slides / p.18 summary):
- tender/painful

subcutaneous erythematous nodules on the shins
- inflammation in the

adipose tissue (panniculitis)*(Erythema nodosum is a septal panniculitis, meaning inflammation of the septa within the subcutaneous adipose tissue. While the overlying dermis may show some reactive changes, the primary inflammation is in the subcutis, not the dermis.)*

Question 323:
A red violaceous lesion after trauma that bleeds easily:
Answer: Pyogenic granuloma

High-Yield Context (from Dermatology Lecture Slides - Benign Skin Tumours (Benign vascular tumours - Pyogenic granuloma), p.35 summary, p.53 lecture):

From Pyogenic granuloma (p.35 summary / p.53 lecture):
- At digits, capillary haemangioma, not infectious, does not resolve spontaneously
-

easily bleeds → profuse and recurrent
- removed surgically by curettage and cautery...
- Lesions may arise at the

site of trauma, often on the digits.
- Grows

rapidly and easily bleeds with minor trauma.

(Pyogenic granuloma (lobular capillary hemangioma) is a benign vascular proliferation that often appears as a rapidly growing, friable, red to violaceous papule or nodule, frequently at a site of minor trauma, and characteristically bleeds very easily.)

Question 324:
A postmenopausal woman with white itchy and painful vulva:
Answer: Lichen sclerosus

High-Yield Context (from Dermatology Lecture Slides - Connective Tissue Disease, Vasculitis (Lichen sclerosus), p.8 of CTD deck, p.16 summary):

From Lichen sclerosus (LS) (p.8 CTD deck / p.16 summary):
-

itchy eruption which mainly affects the **genital and perineal regions in women**
- Clinical Features: Well-demarcated atrophic patches and plaques with a distinctive

ivory-white color.
- Fibrosis of the underlying tissues with associated loss of normal genital architecture

(Lichen sclerosus is common in postmenopausal women and presents with intense itching (pruritus), soreness/pain, and characteristic white, atrophic plaques on the vulva and perianal skin. It can lead to scarring and loss of normal architecture.)

Question 325:
A question about drugs used in psoriasis :

Answer: Oral steroids (most likely answer)
(This implies the question was "Which of the following is NOT typically used / is contraindicated in psoriasis?" and "Oral steroids" was the correct choice for that.)

High-Yield Context (from Dermatology Lecture Slides - Psoriasis (Management - Systemic corticosteroids), p.6 summary, p.150 lecture):

From Psoriasis Management (p.6 summary / p.150 lecture):
●
Systemic corticosteroids should not be used to treat psoriasis. (Due to risk of rebound pustular psoriasis or erythroderma upon withdrawal).

(This is a critical point in psoriasis management.)

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019 EXAM

Question 326:

Statement: Type 4 hypersensitive for allergic contact dermatitis*(This is a TRUE statement.)*

High-Yield Context (from Dermatology Lecture Slides - Eczema (Allergic Contact Dermatitis), p. 164, and Drug Rashes classification p. 26):

From Allergic contact dermatitis (p. 164):
- This is due to the development of

delayed hypersensitivity (type 4 allergy) to a specific chemical (sensitizer or allergen).

From Drug Rashes Pathogenetic Classification (p. 26):
•
Type IV reactions: (m.c)
Delayed hypersensitivity reactions...

Question 327:

Statement: Wrong: Psoriasis is Autosomal receive*(This is a TRUE statement that "Psoriasis is Autosomal recessive" is wrong. Psoriasis has complex, polygenic inheritance.)*

High-Yield Context (from Dermatology Lecture Slides - Psoriasis (Pathophysiology - Genetic predisposition), p. 141):

1. **Genetic predisposition (PSORS1 loci, specific HLAs);**
    - Family history of psoriasis. 16% of the children will have psoriasis if a single parent is affected and 50% if both parents are affected.
    - In monozygotic twins, if one twin has the disease, the other one has a 70% chance of being affected. In dizygotic twins, there is only a 20% chance.

    (This indicates a strong genetic component but not simple autosomal recessive inheritance.)

Question 328:

Statement: Wrong: Systematic steroid given in chronic psrioasis*(This is a TRUE statement that "Systemic steroid given in chronic psoriasis" is wrong. Systemic steroids are generally contraindicated.)*

High-Yield Context (from Dermatology Lecture Slides - Psoriasis (Management - Systemic corticosteroids), p.6 summary, p.150 lecture):

From Psoriasis Management (p.6 summary / p.150 lecture):
●
Systemic corticosteroids should not be used to treat psoriasis. (Due to risk of rebound pustular psoriasis or erythroderma upon withdrawal).

Question 329:

Statement: Acanthosis nigracans is velvet plaque in folds and creases*(This is a TRUE statement describing Acanthosis Nigricans.)*

High-Yield Context (from Dermatology Lecture Slides - The Skin and Systemic Disease (Acanthosis nigricans AN), p.10 of Systemic Disease slides, p.18 summary/p.40 lecture):

From Acanthosis nigricans AN (p.10 Systemic Disease slides / p.18 summary):

- asymptomatic

velvety thickening of the skin, posterior and lateral aspects of the neck, **axillae and arm flexures** (folds and creases)

From Acanthosis Nigricans (p.40 lecture):

- Asymptomatic

velvety dark symmetrical plaques usually affecting the posterior and lateral aspects of the neck, **axillae, and arm flexures**

Question 330:

Statement: Macule is <1cm flat*(This is a TRUE statement defining a macule.)*

High-Yield Context (from Dermatology Lecture Slides - Describing a lesion in dermatology (Primary lesions - Non raised), p.44 lecture):

From Primary lesions - Non raised "without elevation or depression" (p.44 lecture):

a.

Macule – a flat area of altered colour <2cm in diameter

b. Patch – a flat area of altered colour >2cm in diameter

(Note: The Test Bank question says <1cm, the lecture slide says <2cm. Both are common definitions, with <1cm or <0.5cm sometimes used to differentiate from a patch. The "flat" part is key.)

Question 331:

Statement: Erythmya nodusm mc cause? Strep*(This is a TRUE statement. Streptococcal infection is a very common cause of Erythema Nodosum.)*

High-Yield Context (from Dermatology Lecture Slides - The Skin and Systemic Disease (Erythema Nodosum - Causes), p.9 of separate slide deck, p.18 summary):

From Erythema Nodosum (EN) - Causes (p.9 Systemic Disease slides / p.18 summary):

- Infectious causes of EN include

Streptococcus, Mycoplasma pneumoniae, TB

Question 332:

Statement: Scabies treatment is topical permethrin.*(This is a TRUE statement. Permethrin 5% cream is first-line topical treatment.)*

High-Yield Context (from Dermatology Lecture Slides - Scabies and head lice (Scabies - Management), p.28 summary, p.91 lecture):

From Management (of scabies) (p.28 summary / p.91 lecture):

1. **Permethrin cream 5%:**
   - **First-line treatment**
   - Should be left overnight, two applications 7 days apart, adults apply from the neck downwards; babies/infants apply to all the skin.

Question 333:

Statement: Onychomycosis treatment? Systemic antifungal*(This is a TRUE statement for most cases, especially toenail involvement or multiple nails.)*

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections (Onychomycosis - Treatment), p.28 summary, p.111 lecture):

From Nails Onychomycosis Treatment (p.28 summary, p.111 lecture):

-

Systemic therapy with terbinafine 250mg daily for 16 weeks (toenails) or 8 weeks (fingernails) is usually considered the **first line.**

- Topical treatment: should be considered for a single nail or very mild distal nail-plate onychomycosis.

Question 334:

Statement: BCC IS PEARLY AND TRANSLUCENT Nodule*(This is a TRUE statement describing the classic appearance of nodular BCC.)*

High-Yield Context (from Dermatology Lecture Slides - Malignant Skin Tumours (Basal cell carcinoma - Morphology), p.37 summary, p.59 lecture):

From Basal cell carcinoma (BCC) - Morphology (p.37 summary):
○
shiny,translucent pearly skin nodule or red patch or ‘rolled edge’ ulcer

From BCC types - Nodular (p.37 summary, p.59 lecture):
■ Nodular → small
pearly papules or nodules, rolled edge ulcer, telangiectasia.

Question 335:

Statement: Wrong: tinea versicolor rarely recurs*(This is a TRUE statement that "tinea versicolor rarely recurs" is wrong. Recurrence is common.)*

High-Yield Context (from Dermatology Lecture Slides - Fungal Infections (Pityriasis versicolor - Treatment), p.28 summary, p.110 lecture):

From Pityriasis versicolor - Treatment (p.28 summary / p.110 lecture):
- Treatment: Topical selenium sulfide and topical ketoconazole 2% cream applied once daily for 2 weeks reportedly cures between 70 and 80% of patients, but
one-third relapse.*(Relapse/recurrence is common for pityriasis versicolor.)*

Question 336:

Statement: Auspitz sign seen in? Psoriasis*(This is a TRUE statement.)*

High-Yield Context (from Dermatology Lecture Slides - Psoriasis (Pathophysiology), p.5 summary, p.142 lecture):

From Psoriasis - Pathophysiology (p.5 summary):
○ Hyperkeratosis … parakeratosis ”keratocytes retain their nuclei”, poorly adherent and
easily scraped off keratocytes (‘Auspitz sign’)From Psoriasis - Pathophysiology (p.142 lecture):
- The rapid turnover and failure of proper maturation results in defective keratinocytes, which are poorly adherent and
easily scraped off (‘Auspitz sign’) revealing underlying dilated blood vessels.

Question 337:

Statement: What is not seen in psoriasis? Bullous lesions*(This is generally TRUE for classic plaque psoriasis. Pustular psoriasis has pustules. While severe inflammation might rarely lead to some blistering, frank bullae are not a primary characteristic feature of typical psoriasis. Bullous lesions are features of bullous diseases like pemphigoid/pemphigus.)*

High-Yield Context (from Dermatology Lecture Slides - Psoriasis (Clinical appearance, Types), pp.5-6 summary, pp.143-146 lecture):
The summary describes plaques, scales, erythema, and
pustules (in pustular psoriasis). It does not mention bullae as a typical feature.

Question 338:

Statement: Wrong: neonatal lupus transform to SLE in 20% of cases*(This is a TRUE statement that "neonatal lupus transform to SLE in 20% of cases" is wrong. NLE in the infant does not transform into SLE.)*

High-Yield Context (from Dermatology Lecture Slides - Connective Tissue Disease, Vasculitis (Neonatal lupus erythematosus), p.9 of CTD deck, p.17 summary/p.11 lecture):

From Neonatal lupus erythematosus (NLE) (p.9 CTD deck / p.17 summary):
- transplacental passage of anti Ro/La
- Annular scaly lesions on the face/scalp
- Risk congenital heart block

(NLE is due to maternal antibodies and is transient in the infant (except for heart block). It does not mean the infant will develop SLE. The mother might have SLE or another CTD.)

Question 339:

Statement: Herald patch is seen in? Pityriasis rosea*(This is a TRUE statement.)*

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Pityriasis Rosea), p.24 summary, p.127 lecture):

From Pityriasis rosea (PR) (p.24 summary, p.127 lecture):

- PR classically presents with an initial single annular erythematous patch with a collarette of scale – the **herald patch.**

Question 340:

Statement: Urticaria, which is wrong: Oral steroids are first line treatment*(This is a TRUE statement that "Oral steroids are first line treatment" for urticaria is wrong. Antihistamines are first-line.)*

High-Yield Context (from Dermatology Lecture Slides - Urticaria and Angioedema (General management p.10/p.86)):

From General management (p.10 summary, p.86 lecture):

- **Oral antihistamines are the mainstay** of treatment/prevention of urticaria and angioedema.
- **Oral corticosteroids may be indicated in very severe eruptions**... (not for long duration)

Question 341:

Statement: Slapped cheek disease cause? Erythema infectiosum (parvovirus)*(This is a TRUE statement.)*

High-Yield Context (from Dermatology Lecture Slides - Viral Infections (Erythema infectiosum), p.25 summary, p.133 lecture):

From Erythema infectiosum (fifth disease) (p.25 summary / p.133 lecture):

- Caused by **parvovirus B19**...
- Clinical Presentation: The disease manifests as a prodrome of mild fever before the onset of a hot erythematous eruption on the cheeks – hence the **'slapped cheek syndrome'.**

Question 342:

A neighbor asks your advice about oral isotretinoin for her severe acne. One of the following is incorrect:
a. Increased triglycerides is a common side effect
b. The cumulative therapeutic dose varies from one person to another usually depending on their weight
c. Blood test must be done prior to initialization of treatment
d. All patients will experience some degree of lip dryness
e. She should not get pregnant for one year after treatment as it is teratogenic
Answer: E

High-Yield Context (from Dermatology Lecture Slides - Acne (Oral retinoids - Isotretinoin), p.101, p.102):

From Oral retinoids (p.101):

Side effects:

- **Teratogenesis (90% risk of birth defects)**, Female patients of childbearing age will need to use a robust form of contraception while taking isotretinoin **and for 1 month following its cessation**.
- **Rise in liver enzymes and lipids (triglycerides)**, **Blood testing before initiation is essential** and during therapy as indicated clinically.
- All patients will experience some **drying of the lips and skin.From Oral retinoids (p.102):**
- The **cumulative target dosage** for isotretinoin is **120–150mg/kg.** (Calculated based on weight).

*(Statement e is incorrect because pregnancy must be avoided during treatment and for **one month** after stopping isotretinoin, not one year.)*

> End