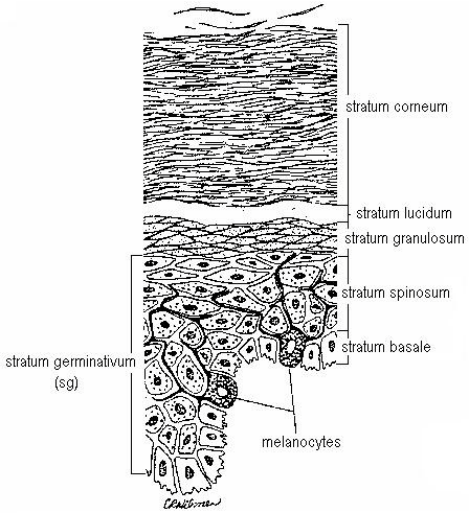
V.C. DISORDERS OF THE SKIN

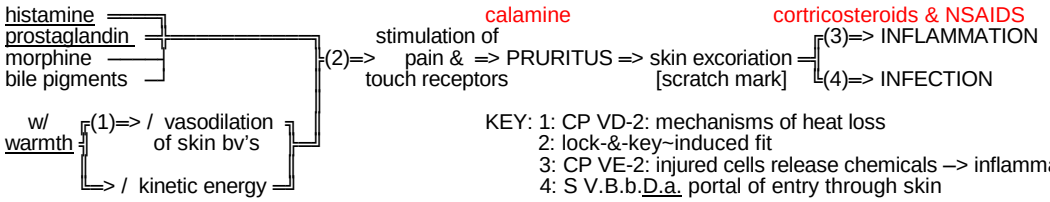
C.a. STRUCTURE OF THE EPIDERMIS



TP VC-1
Figure VC-1. Structure of the Epidermis (32, p 190)

C.b. MANIFESTATIONS OF SKIN DISORDERS

C.b.A. PRURITUS ({L itch} [itching]



KEY: 1: CP VD-2: mechanisms of heat loss
2: lock-&-key~induced fit
3: CP VE-2: injured cells release chemicals –> inflammation
4: S V.B.b.D.a. portal of entry through skin

Path VC-1. Pathophysiology of Pruritus HWA

C.b.B. LESIONS & RASHES <skin chart & book>

C.b.B.a. LESION [S III.A.b.]

C.b.B.b. RASH [in general, a single eruption is called a lesion, many eruptions together are called a rash]

C.b.B.c. PRIMARY LESIONS [arising from normal skin] TP VC-2

1) flat~nonpalpable change in skin color, diameter: macule (spot)< 1 cm < patch
(eryth\ema {G red\G a thing laid down} = red macule, usually due to inflammation)

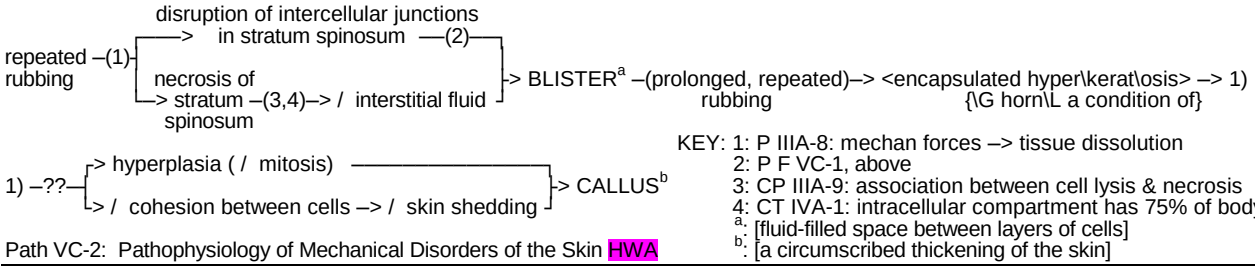
2) elevated, palpable, solid mass, diameter: papule < 0.5 cm < nodule~urtica~wheal~pomphus < 2 cm < tumor

3) elevated, palpable, fluid-filled mass, diameter: vesicle < 1 cm < bulla (blister) also pustule: filled w/ pus

C.b.B.d. SECONDARY LESIONS [arising from primary lesions]

C.c. DISORDERS OF THE SKIN [disorders originating in the skin, (subcutaneous, dermis & epidermis)]

C.c.A. THOSE CAUSED BY MECHANICAL PROCESSES



KEY: 1: P IIIA-8: mechan forces –> tissue dissolution
2: P F VC-1, above
3: CP IIIA-9: association between cell lysis & necrosis
4: CT IVA-1: intracellular compartment has 75% of body water
[a]: [fluid-filled space between layers of cells]
[b]: [a circumscribed thickening of the skin]

Path VC-2: Pathophysiology of Mechanical Disorders of the Skin HWA

C.c.B. THOSE CAUSED BY INFECTION
C.c.B.a. NORMAL COURSE OF EVENTS

(infectious) etiologic agent encounters { mechanical barrier, skin microflora, sebum, immune response =(1)=> prevention of infection / death of the etiologic agent

Path VC-3. General Pathophysiology of Defense Mechanisms in the Skin Key: 1: S V.G: Immunity

C.c.B.b. DISEASE STATES
C.c.B.b.(A.) VIRAL INFECTIONS
C.c.B.b.(A.a.) VERRUCAE [warts] <caused by DNA-containing papovaviruses known as Human Papilloma Viruses (**HPV**)>

HPV invade keratinocytes –(1,2)–> { thickening of stratum corneum; irregular proliferation of stratum spinosum } => VERRUCAE

Path VC-4. General Pathophysiology of Verrucae

KEY: 1: P IIIC-2: P VB-1: viruses insert their **DNA** into our genome
2: P IIIC-2: carcinogen & proto-oncogenes & antioncogenes –> / growth

C.c.B.b.(A.b.) HERPES SIMPLEX VIRUS (**HSV**) (cold sore~fever blister)
C.c.B.b.(A.b.A.) **HSV** TYPE I above waist

primary infection w/ **HSV** I –(1,2)–> { SORE THROAT, FEVER, PAINFUL VESICLES, ULCERS OF THE MOUTH AREA } –(3)–> **IR** –> partial immunity ——> **HSV** I: latency in cell bodies of sensory neurons in the trigeminal nerve ——(T)–> 1)

1) { stress, sunlight, exposure, menses, trauma } –> activation of latent **HSV** I –(4)–> replication –> viruses carried down neurofibrils of afferent fiber –> secondary infection (more localized & less severe than 1°)

TP VC-2a

Path VC-5. Pathophysiology of Primary & Secondary Infections w/ **HSV**

KEY: 1: P VB-1: viral infection –> cell lysis
2: CP VE-1: cell lysis –> SHARP
3: S V.G: immune response
4: P VB-1: viral infection –(latent period)–> cell lysis

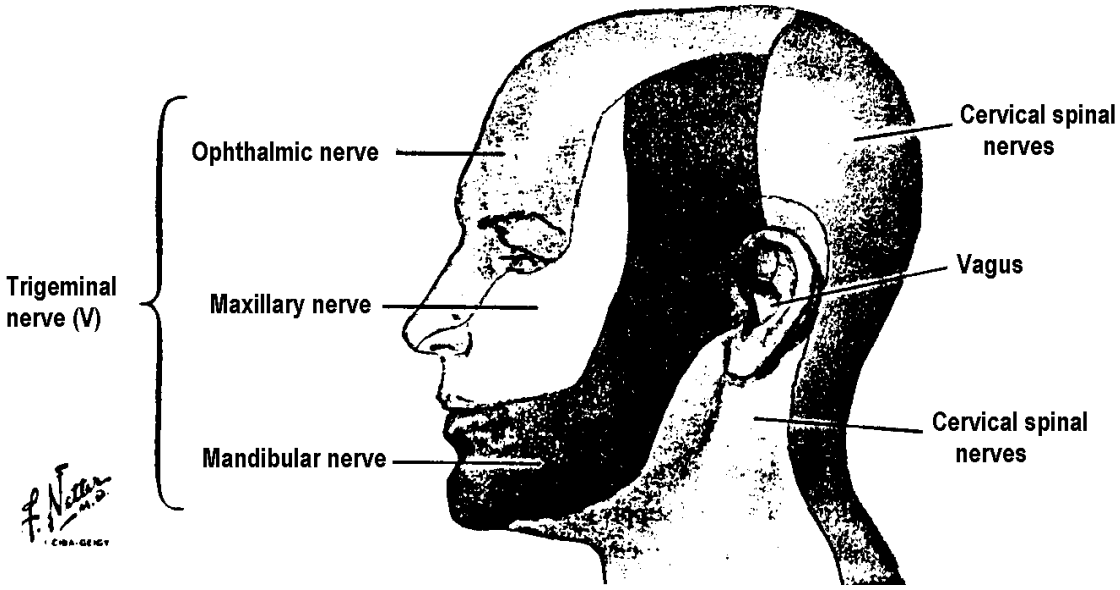


Figure VC-2. Dermatomes of the Branches of the Trigeminal Nerve (21, p 18)

C.c.B.b.(A.b.B.) **HSV** TYPE 2 below waist (**STD**s~venereal diseases) (S IX.C)

C.c.B.b.(A.c.) HERPES ZOSTER (shingles)

HWA

viral latency in sensory cell bodies
infection w varicella-zoster –> chicken pox –(1)–> partial immunity –> (dorsal root ganglia) of cranial & spinal nerves –(T)–> 1)

idiopathic ef
1) —> activates latent –(2)–> dermatome (trigeminal & spinal nerves) –> ERYTHEMATOUS PAPULES –> VESICLES) –> 1)
varicella-zoster (F VC-2) (F 23.8) TP VC-3
1) –(3)–> PUSTULES

most common: T5-T7
digestive processes also shut down
Path VC-6. Pathophysiology of Shingles

KEY: 1: S V.G: immune response
2: P VC-5: 1° infection w virus –> movement of virus down neurofibrils of afferent fiber
3: CP VE-3: role of leukocytes in inflammation

C.c.B.b.(B.) BACTERIAL INFECTIONS

C.c.B.b.(B.a.) IMPET\IGO {L attack\?} [superficial bacterial infection of skin]

infection w *Staphylococcus* or *β-hemolytic Streptococcus*
(1) —> pus => ERYTHEMATOUS PUSTULES
(2) —> liquifaction necrosis w bacteria —> ERYTHEMATOUS VESICLES
—> ERYTHEMATOUS BULLAE
—> eruption of HONEY-COLORED SEROUS LIQUID that dries to HONEY-COLORED CRUST
—> tissue damage =(3)=> prostaglandin release =(4)=> PRURITUS =(4)=> SPREAD

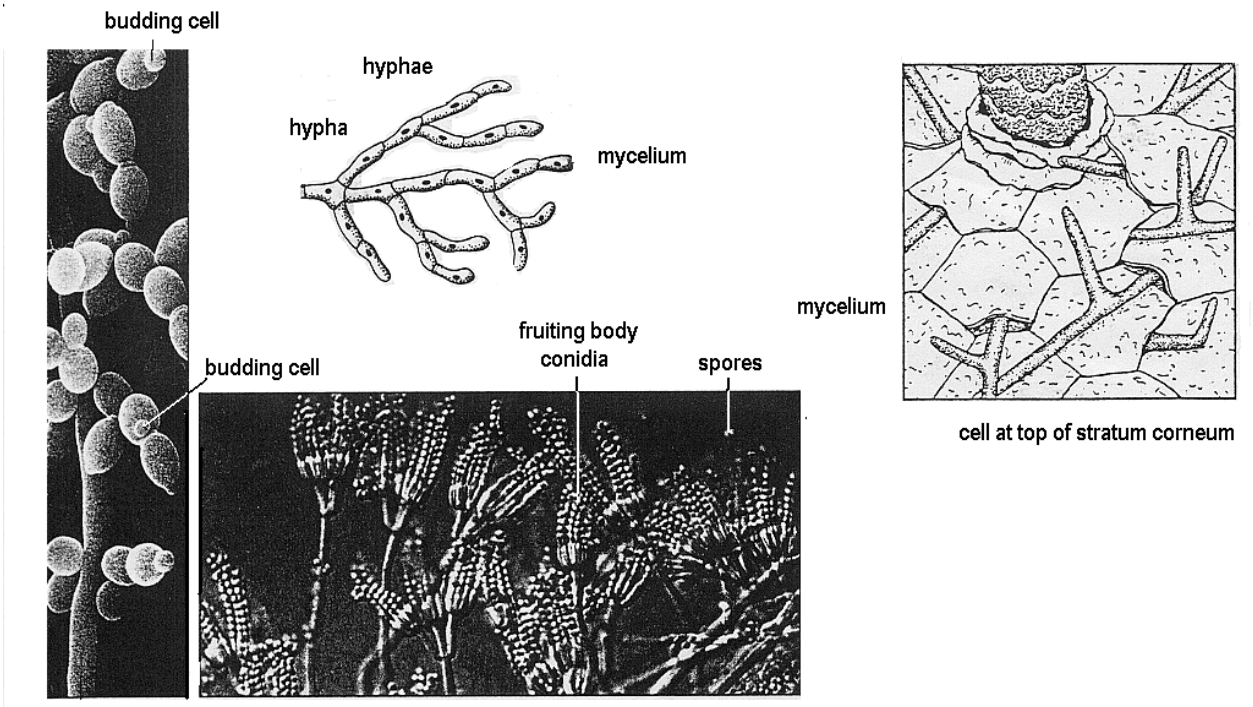
KEY: 1: CP VE-3: role of leukocytes in inflammation
2: S B.b.I.a: role of bacterially-produced toxins
3: CP VE-1: tissue damage & prostaglandin release
4: CP VC-1: pathophysiology of pruritus

HWA
Critical Path VC-7. Pathophysiology of Impetigo

C.c.B.b.(B.b.) ECTHYMA {G spirited, eager} [deeper, ulcerative form of impetigo]

C.c.B.b.(C.) FUNGAL INFECTIONS

fungi reproduce sexually by spores –> hyphae –> mycelia [tangled hyphae] –> fruiting body –> spores
or
fungi reproduce asexually by budding cells



TP VC-4
Figure VC-3. Important Fungal Anatomy (20, pp 374 & 381) & an Example of a Fungal Infection (19, p 100)

C.c.B.b.(C.a.) TINEA [any fungal infection of the skin: e.g., ringworm, athlete's foot]

Microsporum
Trichophyton
Epidermophyton
(dermat\ophytes) fungi were once thought to be plants
{G skin\G plants} } mycelia in stratum corneum } =secrete enzymes=> digest keratin => SUPERFICIAL SKIN SCALING
=> diffuse to dermis =(1)=> INFLAMMATION (erythema) => <tinea>

KEY: 1: S. E.b.B.: inflammatory mediators

Path VC-8. Pathophysiology of Tinea HWA

C.c.C. NON-INFECTIOUS INFLAMMATORY SKIN DISORDERS
[inflammation caused predominantly by irritation rather than infection]

C.c.C.a. ACNE [a lesion caused by inflammation of the pilo\sebace\ous {L hair\L grease\? Pertaining to} unit

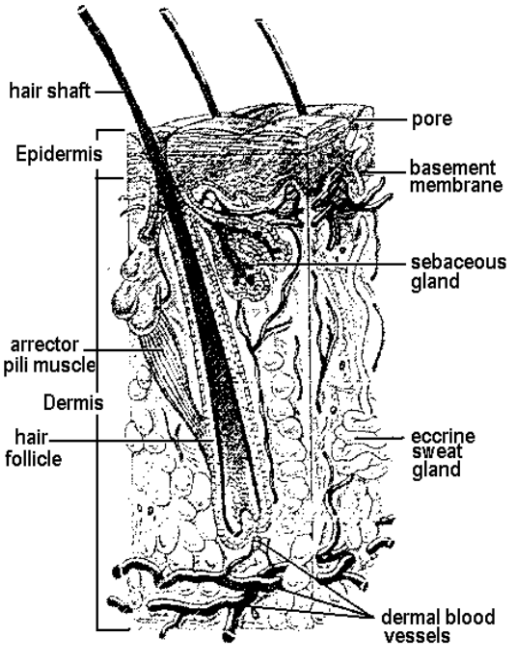


Figure VC-4. Pilosebaceous Unit (47, p 171) TP VC-5

onset of puberty => / androgens => / secretion of sebaceous glands into hair follicle: { fatty acids; tri-di- or monoglycerides; squalene; sterol esters; wax esters } white (sebum) => 1)

androgens => / adjacent epidermal melanocytes => / melanin black

1) => accumulation in open pilosebaceous duct => blackhead (comedone, pl comedones) com/é/dōne
1) => accumulation in closed pilosebaceous duct => whitehead (comedone) com/é/dōne/eez non-inflammatory

KEY: [a]*Proprionibacterium acnes*
1: CP VE-1: arachidonic acid –> inflammation

=> accumulation in surrounding dermis => / bacteria[a] => lipases (if present) => free fatty acids =(1)=> INFLAMMATORY PAPULES & PUSTULES

Path VC-9. Etiology of Acne HWA

C.c.C.b. PSOR/IASIS {G itch/G presence of} [a chronic skin disease characterized by reddish patches covered by silvery scales]

increased incidence in blacks
idiopathic
etiology
skin trauma
(+ genotype)=> hyperplasia => / migration time from s. basale to s. corneum <1/3-1/2 of normal (N = 30 days)> => 1)
versus encapsultated hyperkeratosis seen earlier
(/ cell _____)

1) / keratinization => thinner epidermis => / trauma to dermal papillae
=> damage to papillary capillaries
=> clubbing of papillae
=>: REDDISH PATCHES OF VARIOUS SIZES COVERED W/ SILVERY SCALES
capillaries
thin stratum corneum

HWA
Path VC-10. Pathophysiology of Psoriasis

C.c.D. ALLERGIC SKIN RESPONSES

C.c.D.a. CONTACT DERMATITIS

EF[a]-(1) => hypersensitivity =(1)=> inflammation =(2)=> MILD ERYTHEMA w EDEMA, VESICLES or BULLAE w/ bacterial infection => 2° lesions of <contact dermatitis>

KEY: [a]wool, dyes, perfumes, detergents, nickel in watch bands, resins, solvents, poison ivy & oils to name a few
1: S V.H: alterations in the immune response: wbc's
2: S V.E: inflammation

No TP
Path VC-11. General Pathophysiology of Contact Dermatitis

C.c.D.b. ECZEMA~ATOPIC DERMATITIS (S V.H.b.A.c.)

C.c.D.c. DRUG-INDUCED SKIN ERUPTIONS (S V.H.b.C.)

C.c.E. INSECT BITES, VECTORS & ECTOPARASITES

C.c.E.a. SCABIES [infestation by the mite, *Sarcoptes scabiei*]

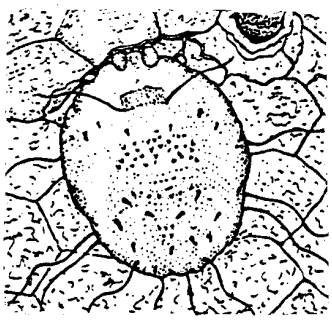


Figure VC-5. A Scabies Mite (19, p 85)

infestation w/ *Sarcoptes scabiei*
=> burrows in epidermis
=> feces in burrows
(irritants)
=> PRURITUS => SCRATCHING => EXCORIATIONS => 2° bacterial infections
=> SMALL REDDISH VESICLES IN CERTAIN AREAS

No TP
Path VC-12. Pathophysiology of Scabies Infestation

C.c.E.b. PEDICULOSIS [infestation by lice: *Pediculus* or *Phthirus*]

1) Body lice (*Pediculus* {L louse} *humanus coporis*
2) Pubic lice (*Phthirus pubis* "crabs")
3) Head lice (*Pediculus humanis capitis*)

Body Louse | Pubic Louse | Head Louse

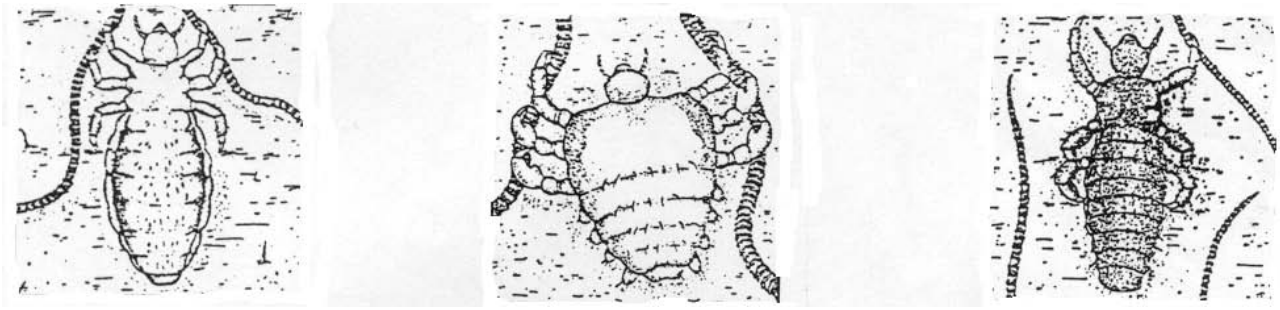


Figure VC-6. The Etiologic Agents of Pediculosis (19, p 60, 77, 73)

C.c.E.c. BEDBUGS{L a bug} *lectal/arius* {L a place where something is kept/L bed or couch}

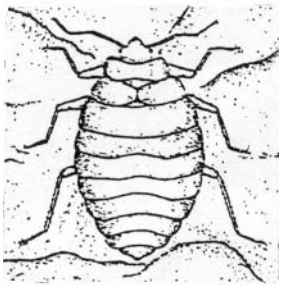


Figure VC-7. A Bedbug (10, p 50)

bedbug bite => anticoagulant in saliva =(1)=> type-I sensitivity reaction => PRURITUS
=> OVAL OR OBLONG WHEAL W HEMORRHAGIC PUNCTUM AT CENTER
=> BULLOUS LESION

No TP

Path VC-13. General Pathophysiology of a Bedbug Bite KEY: 1: P VH-2: Alterations in immune response: IgE

C.c.E.d. TICKS (Ixodidae) {G like birdlime???}
(see our web page for life cycle & other features)

problem: ticks can carry arboviruses, Rickettsial organisms, & arbobacteria

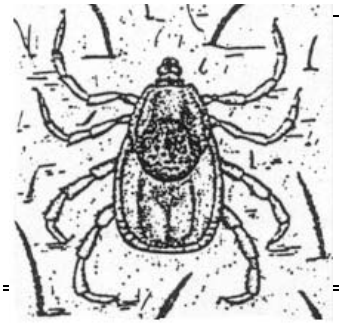


Figure VC-8. A Tick (19, p 25)

Table VC-1. Tick-borne Arboviruses, Tick-borne Rickettsias & Tick Borne Arbobacteria,

Kemorovo tick fever	Colorado tick fever	Kyasanur Forest disease
Siberian tick typhus	Russian spring encephalitis	Russian summer encephalitis
Central European encephalitis	Powasan encephalitis	Langat encephalitis
Negishiencephalitis	Omsk hemorrhagic fever	relapsing fever
Boutonneuse fever	Japanese River fever	Rocky Mountain spotted fever
royal farm virus	Rhanja virus	Ehrlichiosis
Nairobi sheep disease	Tsutsugamushi disease	Lyme disease spirochete
babesiasis	louping ill	Tularemia gram - rod

==========

C.c.E.e. FLEAS *Pulex irritans* problem: fleas can carry some of the same pathogens as ticks
Flea bite => HIGHLY PRURITIC PAPULE W CENTRAL PUNCTATUM ON COVERED PARTS OF BODY

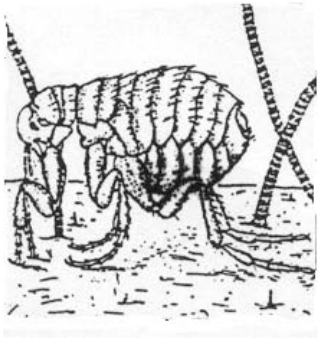


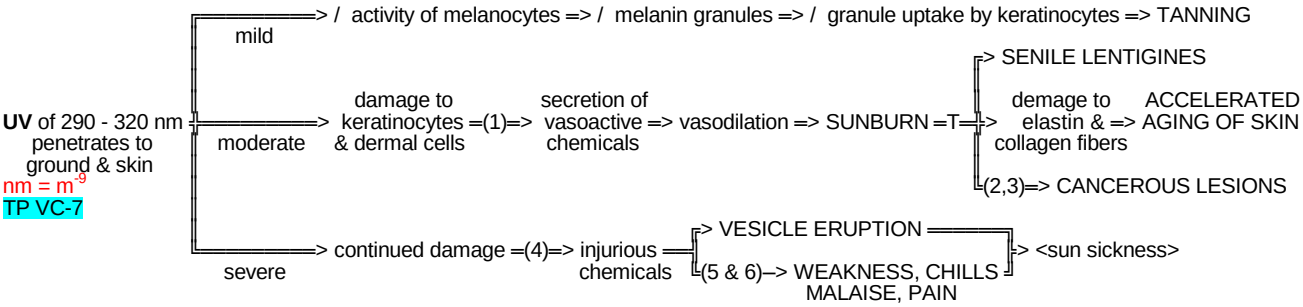
Figure VC-9. A Flea (10, p 55)

C.c.E.f. MOSQUITOES (west Nile virus, yellow fever, dengue fever)
(see my web page for life cycle-source: http://mosquitomagnet.com/help/common/mosquito_lc.htm)

C.c.E.g. OTHER BITING FLIES (black flies, midges~no-see-ums, deer flies)
(see my web page for life cycle-source: http://www.glacvcd.org/HTML%20Pages/Black%20Flies.htm

C.c.E.h. BEE STINGS Type I allergic response Path V.H.-2

C.c.F. PHOTOSENSITIVITY & SUNBURN



KEY: 1: CP VE-1: damaged cells release vasoactive chemicals
2: S IIIA.b.C.c.(A.a.): ionizing radiation & free radicals
3: S III.C.c.G: (pealing skin –> / UV_b exposure –> / neoplasms of the skin)
4: P IIIA-7: release of cellular enzymes following cell lysis
5: P VE-1: prostaglandins
6: P VD-6: early chill phase/ more later

Path VC-14. Photosensitivity & its Consequences TP VC-6

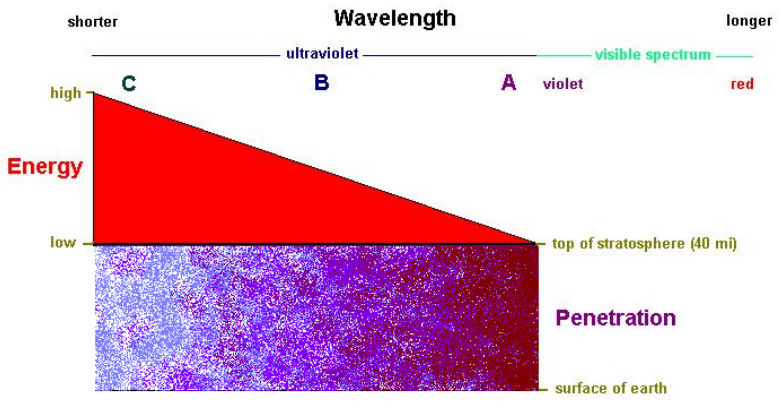


Figure VC-10. Important Properties of Ultraviolet Light TP VC-7

C.c.G. NEOPLASMS OF THE SKIN

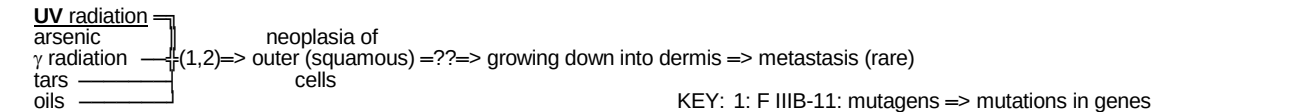
C.c.G.a. NEVI (moles) [benign, congenital or acquired tumors]

Characteristics: flat, elevated; pigmented, non-pigmented; hairy; non-hairy

C.c.G.b. BASAL CELL CARCINOMA [malignant tumor of keratinocytes in deep epidermis]

sustained exposure to sun –(Path VC-14)–> neoplasia of basal cells –??–> nonmetastasizing: **BCC**

C.c.G.c. SQUAMOUS CELL CARCINOMA [malignant tumor of keratinocytes in outer epidermis]

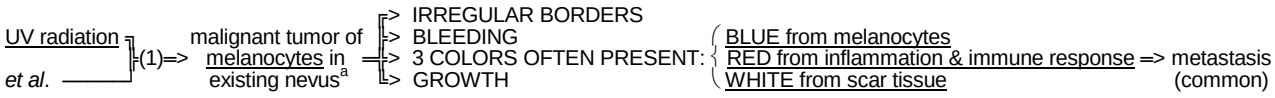


KEY: 1: F IIIB-11: mutagens => mutations in genes
2: P IIIC-2: mutations in antioncogenes –> neoplasia (44, p 67)

no TP

Path VC-15. General Pathophysiology of Squamous Cell Carcinoma

C.c.G.d. MALIGNANT MELANOMA [malignant tumor of melanocytes]



KEY: 1: P IIIC-2: proto-oncogenes & anti-oncogenes
[a]occasionally arise spontaneously

no TP

Path VC-16. General Pathophysiology of Malignant Melanoma

A
B
C
D