DERMATOLOGY MODULE

Basal cell layer (Stratum Germinativum) – lowest, dividing cells
- tonofilaments made here, hemidesmosomes, 19 days
Spinous cell layer (Stratum Spinosum) – cells no longer dividing, making subcellular constituents
- tonofilaments compacted, desmosomes
- keratinosomes (membrane coating granules, lamellar bodies) – put lipids into intercellular spaces to act as barrier
Granular cell layer (Stratum Granulosum) – transitional area, keratohyalin granules
(keratin filaments embedded in other proteins)
- keratinosomes – fuse w/ plasma membranes, releases lipids
- 24-42 days thru spinosum and granulosum
Stratum Corneum (horny layer) – anucleate flattened cells
- 15-20 layers, cells w/ no organelles, dense keratohyalin, deposit sulfur-rich proteins so plasma membrane thickened, enzymes dissolve cell connections so shedding of cells, transit time is 14 days
Total epidermal renewal time = 59-79 days

Melanocytes – melanin transported thru dendrites and engulfed by spinous cells (keratinocytes)
Melanoblasts – from neuroectoderm (neural crest), migrate to skin, eyes, ears, leptomeninges, and GI tract
- CNS (mental retardation, cerebellar ataxia), Ears (sensorineural deafness – Wolff’s syndrome), GI tract (aganglionic megacolon)
- grey hair = decreased melanocyte replication
Tyrosinase forms melanin – eumelanin (no cysteine, brown-black) or pheomelanin (cysteine and sulfur, yellow-red)
- Melanosomes – formed in ER, holds tyrosinase, four stages:
  I – no melanin, tyrosinase active
  II – fully formed melanosome, no melanin
  III – partially melanized
  IV – fully melanized
Dark pigmented people – many stage III and IV melanosomes, large melanosomes not readily digested
Lightly pigmented – few stage III and IV melanosomes, small aggregated melanosomes (easily digested)
Rate of melanin synthesis controlled by MSH, ACTH (low in Addison’s), UV light, inflammation; Hydrocortisone and hydroquinone decrease formation

Langerhan’s Cells – in basal, spinous, and granular layers, made in bone marrow then migrate, involved in Allergic Contact Dermatitis, UV-induced Skin Cancer, Viral and Fungal Infections; decreased in Psoriasis, Sarcoidosis, UV exposed skin

DERMIS
Fibrous connective tissue – mainly collagen (85% type I), in triple helix, some elastic
Non-fibrous ground substance – mucopolysaccharides, hyaluronic acid, chondroitin, etc.

**Recessive dystrophic epidermolysis bullosa** – increased collagenase, collagen degeneration and sub-basement membrane cleavage

**Cerebriform CT nevus** – decreased collagenase, excessive accumulated collagen

**Pseudoxanthoma elasticum** – generalized elastorrhexis, abnormal dermis, BVs, Bruch’s Membrane of eye

**Mucopolysaccharide genetically determined diseases** – enzyme defect, storage disease

Basement membrane – lamina densa and lucida, subbasal lamina = anchoring fibrils (collagen), oxytalan and elaunin (elastic)

**Recessive dystrophic epidermolysis bullosa** – decreased anchoring fibrils, blistering

**Junctional epidermolysis bullosa** – abnormal hemidesmosomes

Eccrine sweat unit – **Hyperhidrosis** and **Anhydrosis**

Apocrine sweat unit – odiferous and thermoregulatory, opens into hair follicle

**Apocrine Bromhidrosis** – body odor from bacterial decomp.

**Hidradenitis Suppurativa** – pore occlusion + bacterial infection

Hair Growth Cycle – Anagen Phase (active, 80-90% hairs, 3-4 years), Catagen Phase (transitional, short), Telogen Phase (rest, 2 months)

**Telogen Effluvium** – stress shifts many hairs to telogen phase, 2-3 months later shedding

**Alopecia Areata** autoimmune, patch hair loss, white hairs usually spared

Sebaceous Glands – produce protective oil, attached to hair follicles, holocrine secretion

**Acne**

Meissner corpuscles – touch, dermal papillae on hands and feet, capsule

Vater pacini corpuscles – pressure, concentric lamellae

Merkel cells – touch, above basement membrane

**Sturge Weber syndrome** – port wine stain (lateral nevus flammeus), ass’d BV abnormalities in eye and leptomeninges (glaucoma and seizures)

Ectoderm = epidermis, sebaceous glands, eccrine glands, appocrine glands, nails, hair

Neuroectoderm = melanocytes and nerves

Mesoderm = collagen, elastic, BV, fat and muscle

Epidermis – 6 weeks

Basal germinative, stratum intermedium and periderm – 9 weeks

Melanocytes – 8-10 weeks

Epidermal appendages – 12-14 weeks

Keratinization – 14-16 weeks

Like adult by 17 weeks

**Biopsy** – select early lesions in vesicular, bullous, and pustular lesions

Select well developed lesions for all others

**Cytology** – for P. vulgaris and other vesiculo-bullous diseases
**Immunofluorescence** – for vesiculo-bullous diseases, lupus, and leukocytoclastic vasculitis (cutaneous acute vasculitis, palpable purpura, neuts. And fibrin around dermal venules)

**Polariscopic exam** – for deposits, foreign bodies, gout and amyloid

Flat primary lesions – macule, patch
Raised primary lesions – papule, nodule, plaque (flat raised area)
Fluid filled primary lesions – vesicle and bullae (clear fluid), pustule (white/yellow cloudy fluid)
Secondary lesions – erosion, ulceration, excoriation, fissure, scar, hyper or hypopigment

**Macule** – flat, non-palpable (i.e. Vitiligo)
**Papule** – solid, elevated (i.e. Molluskum contagiosum)
**Plaque** – circumscribed, thickened, multiple papules that coalesce (i.e. Psoriasis, Lichen Planus)
**Urticaria** – pinkish papules that blanch, transient edema (i.e. allergic contact dermatitis)
**Nodule** – dome-shaped solid lesion (i.e. Melanoma)
**Papilloma** – elevated lesion w/ finger-like projections (i.e. skin tags)
**Cyst** – epithelium-lined cavity (i.e. Epidermal inclusion cyst)
**Vesicle and Bulla** – fluid-filled circumscribed elevated lesions (i.e. Bullous Pemphigoid)
**Pustule** – pus (i.e. Impetigo)
**Purpura, Petechiae, Ecchymosis** – bleeding into skin
**Scale** – cornified cells, accelerated growth (i.e. Psoriasis, Ichthiosis orgalis)
**Crust** – dried exudate (from bacteria) composed of serum and cells (i.e. Impetigo)
**Erosion** – superficial, no scar w/ healing (i.e. Pemphigous vulgaris)
**Ulcer** – deeper, scar, complete loss of epidermis and part of dermis (i.e. Stasis dermatitis, Leishmaniasis)
**Fissure** – linear defect into dermis (i.e. Psoriasis)
**Scar** – fibrosis (i.e. trauma, infection)
**Atrophy** – decreased skin substance
**Lichenification** – popular thickening, accentuation of normal skin markings, hyperpigmentation, sometimes scales (i.e. wearing ring) (callous is thicker than this)
**Orthokeratosis** – hyperkeratosis without nuclei
**Parakeratosis** – hyperkeratosis with nuclei (i.e. Psoriasis, Squamous cell carcinoma)
**Acanthosis** – thickened stratum spinosum
**Spongiosis** – INTER-cellular edema
**Ballooning** – INTRA-cellular edema (i.e. viral inclusions)
**Acantholysis** – loss of cohesion between epidermal cells (i.e. Pemphigus vulgaris)
**Vacuolar alteration** – at dermal-epidermal junction, form clefts or vesicles
**Fibrosis** – increased collagen, *increased* fibroblasts (i.e. scar)
**Sclerosis** – increased collagen, *decreased* fibroblasts (i.e. Scleroderma)

**Xeroderma pigmentosum** – genetic, can’t repair UV damage (pyrimidine dimmers), premature aging of skin with huge increase in skin cancers
**Basal Cell Carcinoma** – most common, no precursor, 80% head and neck, bleeds and scabs, many subtypes (macronodular, micronodular, superficial spreading, morpheiform – aggressive, basosquamous), rarely metastasize, watch for “band-aid” or “headband sign”, raised pearly border, basalioma cell, absence of intercellular bridges, blue balls on micro, palisading of peripheral cells, stromal and more pleomorphic type = cut out, many small islands = BAD b/c can infiltrate and cause loss of function

**Nevus sebaceous** – present at birth, can get basal cell carcinoma in this, remove around puberty

**Actinic Keratosis** – precursor to SCC (1-30% go on), superficial scaling lesions (feel it), sun damage (on head, neck and arms)

**Cutaneous Horn** – squamous cells w/ keratinized material overlying, sticks out far, hyperplasia growth, seen in hypertrophic AK, warts and SCC

**Bowden’s Disease** – superficial, in situ, full thickness atypia, slowly enlarging single red scaling plaque, not necessarily in sun exposed areas

**Erythroplasia of Queyrat** – squamous cell in situ on penis in uncircumcised

**Keratoacanthoma** – keratin filled nodule on sun exposed skin, grow rapidly, spontaneously involute, mets in 10%

**Squamous cell carcinoma** – 2nd most common skin cancer, 80% on head, neck and arms (lips = men), neoplastic growth of keratinocytes, locally invasive and can mets, increased risk of mets if in scar tissue, irradiated skin, infection or arsenical keratosis,  risk = sun, burns, scars, radiation, inflammation, pink and frothy on micro, loss of polarity, much cytoplasm (large cells), keratinocytic bridges, mitoses, sometimes necrosis and hemorrhage

Broder’s Classification – based on degree of differentiation (↑ keratinization = ↑ grade)

KIN system – keratinocytic neoplasia, helps decide how aggressively to treat, better

**Malignant Melanoma** – lifetime risk = 1/39, 50% develop before age 40, m.c.c. in women age 25-29, risk = genetics, blistering sunburns, # of moles, atypical moles, skin type, large congenital nevi and family history, ABCDE rule (asymmetry, boarders – irregular, color - many, diameter – bigger than pencil eraser, enlarging), regression – BAD, arise in congenital nevus in 25%, ↑ nevi = ↑ rate of melanoma, large pink frothy cells throughout epidermis, big malignant melanocytes, much pigment, nests of melanocytes (may see a congenital nevus), deeper melanoma = ↑ rate of mets

**Lentigo Maligna** (Hutchinson’Freckle) – melanoma in situ, over 50 y.o., slow growing pigmented macule, 5% become invasive, limited to epidermis, great cause of medical-legal suits, cut out 1 cm beyond what actually seen, elderly on head and neck, looks like sun or aging spot, single cells of atypical melanocytes in line near surface

**Lentigo Maligna Melanoma** – on sun exposed skin of elderly

**Superficial Spreading Malignant Melanoma** – 70% of all melanomas, ABCD rule

**Acral Lentiginous Melanoma** – on palms, soles, nail beds, mucous membranes, in blacks and Asians, sunlight not a factor

**Nodular Melanoma** – 15-30% of melanomas, grows rapidly, NO ABCD rule, backs of men, legs of women
Clark’s Method – defined by levels based on extent of skin involvement, not used anymore

- I. epidermis
- II. Papillary dermis
- III. papillary/dermis interface
- IV. Reticular dermis
- V. fat

Breslow’s method – depth from granular layer to deepest level of melanocyte involvement

- I. Low Risk - <0.76mm, level II or III
- II. Moderate Risk – 0.75-1.5mm (23% mortality)
- III. High Risk - >1.5mm and level IV or V (37% mortality)

Must do sentinel node biopsy if deeper than 1.0mm
Once metastasizes, incurable and survival is 6 months

Congenital Nevi – melanoma risk is size dependent (esp. greater than 20 cm)

Dysplastic Nevus Syndrome – familial = 100% risk of melanoma by age 70
Aquired = 3-14% risk, prophylactic removal NOT protective
nevus w/ architectural disorder w/ or w/out cytological atypia (not necessarily increased risk of melanoma), do mole mapping to detect changes

Halo Nevi – melanocytic surrounded by depigmentation, in kids and teenagers, NO risk of malignancy

Psoriasis – chronic inflam, prototype of scaly rash, well-demarcated pink plaques covered by loose scales (can pull off in sheets – see punctuate bleeding), on extensor surfaces (elbows, knees, lower back, butt), no stratum granulosum, parakeratosis (have nuclei), accelerated rate of regeneration, neutrophils in epidermis (form pustules), elongated dermal papillae, decreased Langerhans cells, 10% have arthritis, shows Koebner Phenomenon (lesions in trauma areas, also w/ Lichen Planus and Warts), etiology = genetic (HLA markers), keratinocyte proliferative activity, and immune system (activated T cells), hyperproliferation (shortened cell cycle), Tx = immunosuppressive (↓ T cells and cytokines), UV light, methotrexate, tars, Vit. D3 scale = parakeratosis
punctuate bleeding w/ scale removal = thin epidermis w/ tortuous capillaries in dermis
plaque = acanthosis (thickened stratum spinosum)
erythema = perivascular inflammation

**Seborrheic keratosis** – superficial, verrucous, pigmented greasy lesion w/ prolif. Of epidermal cells resembling basal cells, enclosing horny cysts, after 3rd decade

**Lichen Plantus** – chronic inflam, itchy, self-limited, violaceous (=has lymphocytes), flat-topped papules that form plaques, colloid (Civatte) bodies (apoptotic cells), CMI response (helper T’s in LP, cytokines cause epidermal cell damage), volar aspects of forearms and around ankles, lichen stria if on tongue, orthohyperkeratosis (no nuclei), wedge hypergranulosis (b/c sparing of follicle), epidermal acanthosis, lichenoid mononuclear inflam (destruction of basal keratinocytes – colloid cleft, obscure D/E junction, melanophages), prominent granulosus layer (vs. Psoriasis), Tx = corticosteroids, PUVA

5 P’s = purplish (violaceous), planar (flat topped), polygonal, pruritic, papules

Hyperplastic Lichen Planus – parakeratosis, extensive acanthosis, dermal fibroplasias and pigmentation

Pink flat papules w/ reticulate gray lines (Wickham’s Striae) = orthohyperkeratosis

violaceous = inflammation and macrophages

Differential Diagnosis:

1. Discoid Lupus Erythematosus- lichenoid pattern, more scarring, damage to basal layer w/ lymphocytes, damage at D/E junction, atrophic hairless hypopigmented slightly scaling plaque, inflam tissue w/ follicular involvement, perivascular and perifollicular inflam infiltrate, immunofluorescence positive for IgM and IgG and C3 along D/E junction, autoimmune

2. Fixed Drug Eruption – eosinophilic, many meds cause this, usually at one spot (topical drug), D/E junction w/ lymphs and eosinophils

3. Contact Dermatitis

**Allergic contact dermatitis** – wheal, hive, urticaria (think transient edema, blanching)

**Bullous Pemphigoid** – subepidermal nonacantholytic blister, elderly, resistant to rupture, on flexor surfaces, thighs, axillae, groin, lower abdomen, and oral, erythema and tense bullae, eosinophils, deposit of IgG and D3 along D/E junction (basement membrane zone), autoimmune

**Eczema/Dermatitis** – vesicobullous phase or papulosquamous changes, cutaneous response to noxious stimuli, erythema and swelling of skin, oozing and/or vesiculation (like poison ivy), crust and scaling, thickening and evidence of repeated excoriation, hyperpigmentation, scratch papule formation and/or lichenification (thickening of skin w/ increased lines, can feel it), can be localized in flexural points

Contact Dermatitis: primary irritant (harsh chemicals) or allergic (T-cell mediated inflam)
Atopic Dermatitis = pruritis, young age (facial and extensor involvement), if adults (flexural lichenification), chronic, personal/family history of atopy (asthma, allergies, atopic dermatitis)
Acute Dermatitis = parakeratosis w/ inflam crust, spongiosis and exocytosis, vesicles, acanthosis, perivascular dermal inflam (neuts and eos) and edema, basal layer intact scale crust = parakeratosis w/ inflam
papule/plaque = spongiosis, acanthosis
erythema = dermal inflam irritant = neutrophils allergic = eosinophils
Chronic Dermatitis = hyperkeratosis, hypergranulosis, psoriasiform acanthosis, thickened collagen w/in papillary dermis, mild perivascular inflam plague/nodule = hyperkeratosis, acanthosis, dermal fibrosis excoriatio = superficial ulcer
Photoallergic dermatitis – i.e. hydrochlorothiazide, pruritic rash on face, neck and arms, spongiotic inflam tissue, eosinophils, necrotic epidermal cells, immunologic, requires allergen and UV light, rash may persist after medication withdrawn
Seborrheic dermatitis – scaly macular eruption, mostly of face, scalp (dandruff), and other areas of sebaceous gland secretion (sternum, upper back, ears, groin or axillae), midline disease, abnormal response to yeast (Pityrosporum), more common in adults, eczematous patches, parakeratotic crust adjacent to follicular ostia, spongiosis, exocytosis, inflam (perivascular and perifollicular), edema and dilated blood vessels (Do PAS and KOH to rule out fungus)
Tx (also for eczema) = corticosteroids, immunomodulators, antihistamines, emollients, antibiotics, ketoconazole
Mycosis Fungoides – primary cutaneous T-cell lymphoma, t-lymphs are neoplastic, peppered thru dermis, could eventually ulcerate travel and lead to death, hard to diagnose, resembles eczema or psoriasis

INFECTIONS DISEASE AND SKIN

Acne – propronibacterium acnes (bacteria), oil (sebum), comedones (abnormal keratinizations), pustules, papules, cysts, Tx = antibiotics

Cellulitis - infected dermis and subcutaneous skin, usually Group A beta-hemolytic strep, poorly demarcated, rapidly spreading, hot, painful, erythematosus, indurated, often fever, chills and malaise

Impetigo – “honey-yellow crust”, usually kids, superficial subcorneal infection, small vesicle to crust, subcorneal pustule, usually Staph aureus (could be Group A beta-hemolytic strep), Tx = PCN to prevent acute glomerulonephritis, Mupirocin for localized infection

Follicular Abscess – normal epidermis w/ nodule of inflam, spread into rest of dermis, neutrophils, could be from Pseudomonas (hot tub) or E. Coli (didn’t wash hands) or
others, some parakeratosis, keratin plugs, inflammation (neutrophils) around follicle, look for organism (i.e. bacterial cocci, fungal spores and hyphae)

**Furuncle** – deeper infection of follicle, warm red hardened nodule, usually Staph

**Carbuncle** – cluster of furuncles, penetrate deeply, severe inflam and necrosis

**Staph Scalded Skin Syndrome** (Ritter’s Disease) – cutaneous response to 2 extracellular exfoliative toxins produced by Staph aureus of phage group II (ETA is chromosomal, ETB is plasmid), toxins act extracelllularly and cause subcorneal separation of epidermal cells, toxin antibody present in 75% of >10y.o., toxins cleave epidermis beneath granular layer by rupturing desmosomes (Biopsy looks like superficial pemphigus so clinical history is important)

**Mycobacterium Marinum** – from fishtanks and pools, scale crust, granulomas, necrosis in deep dermis, do acid fast bacillus stain or light blue Zeil-Nelson stain (?), ask if fish died, Tx = doxycycline, minocycline, clarithromycin, or TMP-SMX for at least 3 months

**Erythrasma** – Cornebacterium Minutissimum, shine black light and get bright color, Tx = antibacterials, picture shows in groin area

**Cutaneous Anthrax** – latent period is 1-12 days, mortality is 10-20% treated, hands face and neck most commonly, micro see G+ rods and spores, clinical progression – non-tender pruritic macule/papule, vesicle or bullae (1-2days), bullae enlarge and rupture (edema, satellite vesicles), ulcer forms w/ crust, eschar w/ erythematous plaque, minimal scarring

**Warts** – HPV, contagious, (must rule out veroucious carcinoma), hyperkeratosis, fingerlike projections, elongated dermal papillae, perinuclear vacuoles, altered nuclear contour (chromatin), hard to treat b/c go deep, must do cryosurgery w/ nitrogen, topical therapy of salicylic acid or topical immunomodulators (i.e. Imiquinod), cancer risk depends on HPV strain, lose dermatogliphics (vs. callous), periventricular halos, nuclear atypia, wrinkling, can have flat warts w/ linear faint papules on gross

HPV 1 – deep plantar warts, 2 and 4 – common warts, 6 and 11 – condyloma (STD), 16,18, and 31 – genital neoplasia

**Molluskum Contageosum** – spread in bathtubs and swimming pools, dome shaped papule w/ central umbilication, micro shows big dome, viral infected cells, mostly cytoplasm, some falling apart, DNA poxvirus, contagious (incubation is 4-8 weeks), in kids resolve in <2 years, Tx = destructive or chemical or immunomodulators, usually no scarring, key to diagnosis are umbilicated papule and cytoplasmic inclusions

**Small Pox** – papules, vesicles, and pustules, fulminant and highly contagious, immunity about 10 years, eradicated

**Herpes Simplex** – grouped pustules, acantholysis, intraepidermal vesicles, necrosis, perinuclear halos, inclusion bodies, multinucleated, smudged chromatin, dsDNA, very contagious, has pain, pustules and erosions, Tx = acyclovir, valacyclovir, famciclovir
**Varicella** – chicken pox, highly contagious, truncal to peripheral, pruritic small clear vesicles w/ pink halos (“dewdrops on a rose petal”), 14 day incubation, much worse in adults, lesions all at different stages, Tx = prevent w/ vaccine, antiviral if severe

**Herpes Zoster** – shingles, painful grouped vesicles (often purpuric) w/in a dermatome, bubbly blisters, reactivation from dorsal root ganglion, incidence increased w/ age, Tx = may speed resolution and decrease post-herpetic neuralgia, decrease contagiousness, acyclovir, valacyclovir, famciclovir

**Candida Albicans** – fungus, causes oral candidiasis (thrush), intertrigo, paronychia (nails), folliculitis, chronic mucocutaneous candidiasis (IC), disseminated candidiasis(IC), Tx = topical nystatin, imidazoles, ciclopiroxolamine

**Fungal Infections (Dermatophytoses)** – many types of tinea, Trychophyton or Microsporum species, scaly slightly red enlarging area, do KOH stain on scale, micro (PAS stain) shows spores and septated segmental hyphae (vs. yeast has NO septae, pseudohyphae and elongated), hyphae and spores w/in stratum corneum (adjacent to inflam. crust), also in hair shafts and nail plates, Tx = systemics for scalp and nails (Griseofulvin, terbinafine, itraconazole), other areas use topicals ( clotrimazole, econazole, ciclopiroxolamine)

**Tinea Versicolor** – YEAST (NOT a fungus!), superficial scaly macules and patches on trunk and proximal extremities, hypo or hyperpigmented macules, could be Malassezia furfur or Pityrosporum ovale, does NOT respond to Griseofulvin (b/c yeast), shows up w/ sun exposure, more common in humid environments, micro shows dense aggregates of spores and hyphae in stratum corneum (similar to fungus), “spaghetti and meatballs” scale = hyperkeratosis (not as much as Psoriasis)
KOH prep = organisms w/in stratum corneum
hypopigmentation = density of organisms

**Pityriasis Rosea** – “small scale”, “red”, light red spots on body, children and young adults, acute and self-limited, seasonal variation (winter highest), oval patches along lines of cleavage (Christmas tree pattern), NO involvement of palms and soles, Collarette scale (ring around lesion), herald patch (initial lesion only, slowly enlarging, epidermal acanthosis, don’t mistake for fungus), papulosquamous, can be scaly white raised papules (b/c of dermal inflammation), mounds of parakeratosis, superficial perivascular inflammation, NO fungal organisms, edema in stratum spinosum, some areas have normal epidermis, granular zone seen (vs. Psoriasis), likes trunk and proximal extremities

**Secondary Syphilus** – small papules and scaly areas, often darkened macular areas on palms and soles, cause is Treponema pallidum, appears 6-12 weeks after chancre, lasts 4-6 weeks, Diagnosis = skin findings, lymphadenopathy, constitutional signs, lab (+VDRL or RPR, FTA-ABS is confirmatory), Treatment = Benzathine PCN, Erythromycin, Doxycycline, Tetracycline
Early stage = psoriasiform lichenoid dermatitis, superficial and deep inflammation, plasma cells and histiocytes!, neutrophils in epidermis (forms pustules)
Late stage = ↑ epitheloid histiocytes forming granulomas

**Lice/Pediculosis** – body vs. pubic lice, can see along hair shaft, perethrins and permethrins, mayonnaise occlusion, kill the nits

**Scabies** – mites, get pustule, organism burrows into skin, see organisms, fecal nuggets and dense inflammation on micro, 5% permethrin and lindane, always consider this w/ itching

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**REVIEW CASES FROM WEDNESDAY, MARCH 3rd**
1. Acute allergic contact dermatitis
2. Photoallergic dermatitis
3. Tinea pedis, manuum, unguium
4. Psoriasis
5. Lichen Planus
6. Discoid Lupus Erythematosus
7. Bullous Pemphigoid
8. Basal Cell Carcinoma and Squamous Cell Carcinoma
9. Compound Melanocytic Nevus
10. Malignant Melanoma

**REVIEW CASES FROM FRIDAY, MARCH 5th**
1. Psoriasis
2. Discoid Lupus Erythematosis
3. Necrobiosis Lipoidica Diabeticorum
4. Actinic Keratosis
5. Seborrheic Keratosis
6. Granuloma Anulare
7. Acne Vulgaris
8. Sarcoid and Eczema
9. Discoid Lupus