Factors contributing to scale formation

- Increased keratinocyte proliferation
- Disorders of keratinization
- Disorders affecting lipid bilayers (stratum corneum)
- Disorders affecting cell cohesion

Classification of Keratinization Disorders

- Primary = Hereditary
  - Signs develop early in life
  - Majority are autosomal recessive (parents phenotypically normal)
  - Genetic testing available for some disorders
- Secondary = acquired
  - Increased scaling is associated with almost every disorder involving the skin
Seborrhea Oleosa of Persians and Himalyans

- Severe seborrhea may be present within first 2-3 days of life
- Autosomal recessive
- Mild cases may be managed with shaving and bathing
- Severely affected often euthanized

Feline Primary Seborrhea

- Other breeds may also be affected
  - Exotic shorthair
- Affected cats have “dirty appearance”
Canine Ichthyosis

- Clinical signs
  - Abundant scales
  - Keratinous projections
  - Erythroderma
- Two forms in dogs
  - Epidermolytic
  - Non-epidermolytic

Epidermolytic Canine Ichthyosis

- Norfolk Terriers
  - Defect in keratin 10 formation
- Rhodesian ridgeback
- Labrador retriever
- Norfolk terrier
- Cavalier King Charles spaniels

Defects in keratin formation

- Blistering diseases (McLean et al. 1995)
  - K1 + K10
  - K1 + K10
  - K5 + K14
  - Blistering of skin and mucosa
  - Tegopokratin (tissue-derived epidermal growth factor)
Cavalier King Charles Spaniel
Congenital Keratoconjunctivitis Sicca and Ichthyosiform Dermatosis (CKCSID)

- KCS & frizzy coat
- Dorsal scaling, deformed claws
- 8% of UK CKC carriers
- Autosomal recessive
- FAM83H gene (mutation Exon 5 c.1016delC)
- Antagene (France) genetic test


“Bricks and Mortar”

Epidermolytic forms of ichthyosis affect corneocytes
Non-epidermolytic forms of ichthyosis affect the lipid lamellae
Other diseases affect corneodesmosomes (hereditary & acquired)

Non-Epidermolytic Canine Ichthyosis

- Defect in intercellular lipids, cornified envelop, or desmosomes

From Vet Pathol 45:2, 2008
Non-Epidermolytic Canine Ichthyosis

Breeds affected
- Jack Russell terrier (Parson Russell)
  - Low transglutaminase
- Australian terrier
- Cairn terrier
- Norfolk terrier
- American bulldog
- Golden retriever

American Bulldog Ichthyosis
**Golden Retriever Ichthyosis**

- Puppies may exhibit rough haircoat with hyperpigmentation
- Truncal lesions may not be obvious until adults or older
- Autosomal recessive inheritance
- ICT-A = Ichthyosis genetic test (Antagene Laboratory, France)
  blood + cheek swabs

**Golden Retriever Ichthyosis**

**Conditions associated with abnormalities in lipid lamellae**

- Some forms of ichthyosis
  -- Autosomal recessive congenital ichthyosis
    - Defects in 12R-lipoxygenase, eLOX-3, transglutaminase 1
  -- Harlequin ichthyosis
    - Defect in ABCA12 gene (transporter protein for epidermal lipids)
- EFA deficiency (dietary or maldigestion/malabsorption)
- Atopic Dermatitis (humans, dogs, horses)

http://www.antagene.com/en
EM of normal lamellae


EM of abnormal lamellae in atopy

One goal of therapy is restoration of epidermal lipids/barrier function.

Disease Targeting Sebaceous Glands: Sebaceous Adenitis

- **Breeds**
  - Standard poodle (autosomal recessive)
  - Akita
  - Vizsla
  - Samoyed
  - other
- **Age**
  - young to middle-aged

Sebaceous Adenitis

- Long-coated dogs
  - symmetrical alopecia, scaling, dry coat on dorsum, nose, tail, pinnae, truck
  - later follicular casts and matting
- Short-coated dogs
  - "moth-eaten"alopecia
  - secondary pyoderma
Sebaceous adenitis: treatment

- Follicle flushing shampoos (Ethyl lactate)
- Moisturizers (“intensive oil treatment”)
- Isotretinoin (1 mg/kg)
- Vitamin A (1000 IU/kg)
- Tetracycline + Niacinamide
- Cyclosporine 5 mg/kg
- Treat secondary infections
- Client Education: goal is to minimize symptoms, no cure, is hereditary

Diseases affecting desmosomes

<table>
<thead>
<tr>
<th>Component</th>
<th>Molecular Target</th>
<th>Congenital Disease</th>
<th>Acquired Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intermediate filaments</td>
<td>Keratin</td>
<td>Epidermolysis bullosa simplex</td>
<td>Ocular cicatricial pemphigoid</td>
</tr>
<tr>
<td>Desmosomes</td>
<td>Desmoglein 1, desmoglein 3</td>
<td></td>
<td>Pemphigus vulgaris, pemphigus foliaceus</td>
</tr>
<tr>
<td>Desmosomes</td>
<td>Desmoplakin, plakoglobin</td>
<td>Ectodermal dysplasia, skin fragility</td>
<td>Pemphigus foliaceus</td>
</tr>
</tbody>
</table>

Immune-mediated diseases with scaling affecting desmosomes

Pemphigus foliaceus
Immune-mediated diseases with scaling affecting desmosomes

Pemphigus foliaceous
### Diseases affecting BMZ proteins

<table>
<thead>
<tr>
<th>Component</th>
<th>Molecular Target</th>
<th>Congenital Disease</th>
<th>Acquired Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nethedinsnocoe</td>
<td>α, β, δ, ε, γ, ξ</td>
<td>Junctional epidermolysis bullosa</td>
<td>Bullous pemphigoid, mucous membrane pemphigoid</td>
</tr>
<tr>
<td>Nethedinsnocoe</td>
<td>BP230, BP180, BP1, BP58, BP59, BP57</td>
<td>Epidermolysis bullosa (humans), collagen III</td>
<td>Bullous pemphigoid, mucous membrane pemphigoid, linear IgA disease</td>
</tr>
<tr>
<td>Lamina densa (BMZ)</td>
<td>Lamininα3, γ1</td>
<td>Junctional epidermolysis bullosa</td>
<td>Mucous membrane pemphigoid, acquired mucosal immunobullous disease</td>
</tr>
<tr>
<td>Anchoring fibrils (BMZ)</td>
<td>Type VII collagen</td>
<td>Dystrophic epidermolysis bullosa</td>
<td>Epidermolysis bullosa acquisite, type I bullous systemic lupus erythematosus</td>
</tr>
</tbody>
</table>

### Epidermal-Dermal Cleft

![Epidermal-Dermal Cleft Image]

### Immune-mediated diseases with scaling affecting BMZ

- Systemic lupus erythematosus
Immune-mediated diseases with scaling affecting BMZ

Systemic lupus erythematosus

Exfoliative Cutaneous Lupus Erythematosus
German Short-Haired Pointers

- Autosomal recessive
  - Single nucleotide polymorphism on Chromosome 18
- Lesions start 6 mo-3 yrs
- Scaling on face, ears, dorsum→generalized
- Painful & pruritic
- May wax & wane, usually progressive with lameness, hunched back & lethargy

Exfoliative Cutaneous Lupus Erythematosus
German Short-Haired Pointers

- Peripheral lymphadenopathy
  - Reactive lymphoid hyperplasia
- Secondary pyoderma
- Oligospermia
- Skin biopsy: interface dermatitis
  - Superficial dermal infiltrate
  - Basal keratinocyte necrosis
  - DEJ clefs
  - Pigmentary incontinence
Exfoliative Cutaneous Lupus Erythematosus
German Short-Haired Pointers

• Antiseborrheic therapy
• Tx 2° pyoderma
• Hydrochloroquine 5-10 mg/kg once daily may slow clinical progression in some
• Cyclosporine decreases erythema and arthralgia but did not slow overall progression

Exfoliative Cutaneous Lupus Erythematosus
German Short-Haired Pointers

• Prognosis is guarded
• Bathe q 4-7 days with moisturizers
• Omega 3 fatty acids
• Carprofen 2.2 mg/kg q 12 hr OR deracoxib 1-2 mg/kg q 24 hr for control of pain
• Most are euthanized
Cell Proliferation

Epidermal Dysplasia

- **Breed**
  - West Highland White Terrier
- **Lesions**
  - by one year erythema and pruritus
  - “amardillo skin”
- Skin colonized with *Malassezia*—the dysplasia maybe reaction to yeast

Epidermal Dysplasia
Epidermal dysplasia

• Rule out
  – allergies
  – parasites
  – Malassezia
• Biopsy
  – parakeratotic hyperkeratosis
  – Basal cell “crowding”/“buds”
Epidermal Dysplasia

• Management
  – Treat secondary infections
  – Vitamin A 1000 IU/kg
  – Retinoids
    • Retin-A topical
    • Acitretin
  – Antiseborrheic shampoos
Inflammation → Epidermal Hyperplasia

- Ectoparasites
- Allergies
- Infections
- Environmental

Parasites as causes of scaling

- Cheyletiella = “walking dandruff”

Cheyletiella – Clinical Signs

- Dorsal distribution
- Scale
- Variable pruritus (possible hypersensitivity in severely pruritic animals)
- Exfoliative erythema
- Miliary dermatitis or “fur mowing” in cats
Cheyletiella - Diagnosis

- Visualization of parasite:
  - Larger than sarcoptes
  - Prominent mouth hooks
- Visualization of eggs:
  - Small and attached to hair by fine strands (vs large louse nits firmly cemented to hairs)

Cheyletiella

Cheyletiella - Diagnosis

- Methods of parasite visualization:
  - Direct examination of “walking dandruff” (naked eye or hand lens)
  - Superficial skin scrape
  - Acetate tape preparation
  - Fecal floatation
  - Flea combing
  - Vacuum test
Cheyletiella

- Treatment
  - All in-contact animals
  - Minimum of 6 weeks (ideally 2-4 weeks past clinical cure)
  - Treat environment (pyrethrin spray)
  - Keratolytic shampoo

Treatment

- Lime Sulfur:
  - Dip weekly for 6 weeks
- Fipronil:
  - 0.25% spray: 1-2 pumps/lb q 2 weeks for 3-4 treatments
  - 10% spot on: apply 1-2 times/month for 3 treatments
- Selamectin:
  - Apply once monthly for 3 treatments

Parasites as causes of scaling

- *Lynxacarus radovsky* = cat fur mite
- *Sarcoptes scabiei var canis*
- *Notoedres cati*
- *Felicola subrostratus*
Parasites as causes of scaling

- *Demodex cati*
- *Demodex gatoi*

**Demodex gatoi**

- Short bodied
- Found in the stratum corneum
- Contagious
- Pruritic
- May find mites on skin scrapings or fecal flotation

**Demodex gatoi**

- Distribution
  - Head, neck, elbows
  - Ventral abdomen
- Clinical signs
  - Alopecia
  - Scale, erythema
  - Excessive grooming
**Demodex gatoi**

**Therapy**
- Stop any glucocorticoid or progesterone tx
- Treat all in-contact animals
- Lime sulfur is treatment of choice
  - improve in 3 weeks
  - treat for 4-6 weeks minimum
- Ivermectin: 300 mcg/kg once weekly, variable response
- Advantage Multi (q 7-14 days, variable response)
- Amitraz: 125 ppm (1/2 normal strength)

---

**Demodex cati**

**Localized follicular demodicosis**
- Rare condition
- Eyelids, periocular, head, neck
- Differential for feline acne
- Usually responsive to lime sulfur or other mild parasiticides
- Often self-limiting

---

**Demodex cati**

**Generalized follicular demodicosis**
- Very rare
- Siamese and Burmese at risk
- Usually have an underlying condition
  - FIV, FeLV, Diabetes Mellitus, Bowen’s disease
Demodex cati

Treatment: generally much easier than K9
– Manage pyoderma
– Lime sulfur dip weekly
– Ivermectin 300 mcg/kg wkly + Lime sulfur dips
– Amitraz at 125 ppm (1/2 strength)

Parasites as causes of scaling
• Demodex canis
• Demodex injai

Parasites as causes of scaling
• Lynxacarus radovsky = cat fur mite
• Sarcoptes scabiei var canis
• Notoedres cati
• Trichodectes canis
• Felicola subrostratus
• Linognathus setosus
Parasites as causes of scaling

Allergies as cause of scaling

Atopic dermatitis

Allergies as cause of scaling
Allergies as cause of scaling

- Atopic dermatitis
- Food allergy
- Flea allergy
Allergies as cause of scaling

- Food allergy
- Flea allergy

Allergies in Cats

- Common manifestations include
  - Pruritus +/- crusts/scales
  - Feline Miliary Dermatitis
  - Eosinophilic Granuloma Complex
  - Feline Symmetrical Alopecia

Allergies

- Most common clinical sign is "Overgrooming"
**Allergies in Cats**

- Atopic Dermatitis—Diagnosis
  - R/O ectoparasites
  - R/O food allergies
  - R/O infections
  - Investigate for “offending” allergens
    - Serum IgE testing
    - Intradermal testing

**Pitfalls which Limit Usefulness of Serum IgE testing**

- Poor reproducibility
- Poor specificity for IgE
- Many false positives
  - non-specific binding
- Little distinction between positive tests in normal and allergic cats
- Great seasonal variability
  - half-life of serum IgE = 2.5 days
- Not all reactions are IgE mediated

**Intradermal allergy testing**

- Evaluates antigen-specific IgE and/or IgGd bound to mast cells in skin but not a perfect test (still have false + and false -)
- Cat reactions can be more difficult to read
Treatment Options for Feline Atopy

- Control secondary infections
- Eliminate ectoparasites
- Allergen-specific immunotherapy
  - Subcutaneous
  - Oral
- Skin barrier repair
- Omega 3 fatty acids
- Antihistamines
- Corticosteroids
- Cyclosporine
- Oclacitinib?

Diagnosis of Food Allergy

- Neither in vitro nor intradermal testing for food ingredients are accurate predictors of food allergies
  - Differences in antigens of test proteins versus by-products in pet foods
  - Changes in proteins during digestion
  - Many adverse food reactions are not IgE mediated

Definitive Diagnosis of Food Allergy

- Symptoms resolve with dietary change
- Symptoms recur with provocation
Infections → epidermal hyperplasia and/or epidermolysis

Dermatophytes produce extracellular proteinases that digest keratin

Dermatophytosis: Pathogenesis

- hyphae proliferate on the hair surface
- produce keratolytic enzymes → penetration of the hair cuticle
- migrate down the hairshaft until they reach the keratogenous zone (Adamson’s fringe)
Dermatophytosis: Cause

- *Microsporum canis*
  - zoophilic *CATS*
  - most common cause of dermatophytosis in cats and dogs

- *Microsporum gypseum*
  - geophilic - normally found in the soil

- *Trichophyton mentagrophytes*
  - zoophilic *RODENTS*

Dermatophytosis: Feline

- Localized infections
  - One or more annular areas of alopecia
  - May mimic chin acne or “stud tail”
  - Onychomycosis

- Dermatophyte kerion

- Dermatophyte pseudomycetoma
Dermatophytosis: Diagnosis

• Wood’s Lamp
  – warm up a few minutes before use
  – infected hairs fluoresce yellow-green
  – noninfected scales and crusts may appear a brighter white color - this is NOT a positive reaction
  – only some strains of dermatophytes glow:
    • M. canis, M. audouini, M. distortum, T. schoenleinii

• Only about 50% of M. canis will fluoresce.

Wood’s Lamp

Plucked Hairs

• Plucking hairs with hemostats
• Take hairs from periphery of lesions
• Obtain approx 10-20 hairs
Dermatophytosis: Diagnosis

- Microscopic examination of hair
  - Clear the keratin:
    - 10% - 20% KOH
    - chlorphenolac
  - Mineral oil can also be used
- fragmented hairs; larger than normal hair diameter; loss of definition between the hair cuticle, cortex and medulla

Diagnosis of Dermatophytes

- M. canis
- M. gypseum
- T. mentagrophytes

Dermatophytosis: Diagnosis

- Fungal culture
  - MOST REALIABLE test
  - pluck hairs from the periphery of a lesion
  - sterile tooth brush for generalized cases

- DTM = Dermatophyte test medium
  - Dermatophytes utilize the protein in the media first, leading to alkaline metabolites that cause the media to turn red within 10-14 days
  - Saprophytes use carbohydrates first, creating acidic metabolites – then use protein, usually after 10 - 14 days
Dermatophytosis: Diagnosis

- DTM
  - DO NOT RELY ON COLOR CHANGES
  - MUST identify the organism through morphologic and microscopic characteristics

- Rapid Sporulating Media (RSM)
  - encourages rapid sporulation, and organism identification

MacKenzie Collection Technique

- Useful for identifying asymptomatic carriers (cats) and for determining if animal is no longer contagious
- Be sure to brush around face and ears
- Collect hairs from bristles or cut bristles and place them onto media

Monitoring treatment

- Continue treatment until 3 negative cultures
- Toothbrush cultures (also use to screen healthy appearing cats for carrier status)
- Monitor environment through culturing hairs or pads used to wipe floors/walls
**Topical Therapy**

- Decreases contagion—can kill ectothrix spores
- Little effect on endothrix spores or hyphae within hairs
- Repeat applications q 5-7 days
  - Lime sulfur
  - Miconazole/chlorhexidine
  - Enilconazole

---

**Topical Therapy—Lime Sulfur**

- Contains calcium polysulfides → pentathionic acid and hydrogen sulfide after application → fungicidal & bactericidal, also kills many mites and lice
- 1:16 to 1:32 dilutions applied q 4-7 days, do not rinse

---

**Shaving?**

- **Pro:**
  - remove infected hairs
- **Con:**
  - microtrauma to skin likely to spread infection
  - Infected hairs may be dispersed into environment
- **Consider for**
  - Generalized infection in long-haired cat
Bathing?

- Insufficient contact time to kill dermatophytes
- May spread infection
- A few studies have shown benefit with use of ketoconazole/miconazole/chlorhexidine baths in combination with systemic treatment

Dermatophytosis: Treatment

- Systemic therapy (hasten resolution)
  - all animals with multifocal lesions
  - all long haired animals
  - cats!
  - animals not responding to 2 - 4 weeks of topicals
  - Difficult for topicals to penetrate into hair follicles
Systemic Antifungal Therapy

- Highly recommended for all dermatophyte-infected animals.
- Continue treatment until three negative cultures have been obtained (weekly intervals).

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mode of Action</th>
<th>Dose</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Griseofulvin</td>
<td>Inhibits cell wall synthesis, nucleic acid synthesis and mitosis; disrupts mitotic spindle formation;</td>
<td>Micronized: 25‐60 mg/kg PO q 12 hr</td>
<td>Ultramicronized: 2.5‐15 mg/kg PO q 12 hr; Give with fatty meal; monitor CBC for bone marrow suppression; do not use if FIV or FeLV +.</td>
</tr>
<tr>
<td>Ketoconazole</td>
<td>Inhibits p450 enzymes, blocks 14α‐demethylase preventing ergosterol synthesis + other actions</td>
<td>5‐10 mg/kg PO q 24 hr</td>
<td>Give with food; not recommended in cats; monitor for hepatotoxicity (rare); be aware of drug interactions.</td>
</tr>
<tr>
<td>Itraconazole</td>
<td>Inhibits p450 enzymes, blocks 14α‐demethylase + other actions</td>
<td>5‐10 mg/kg PO q 24 hr</td>
<td>May use every other week; monitor liver enzymes.</td>
</tr>
<tr>
<td>Fluconazole</td>
<td>Inhibits p450 enzymes, blocks 14α‐demethylase + other actions</td>
<td>10 mg/kg PO q 24 hr</td>
<td>Penetrates into CNS and eyes; monitor liver enzymes; at 10 mg/kg, some dogs develop vasculitis.</td>
</tr>
<tr>
<td>Lufenuron</td>
<td>Inhibits chitin synthesis</td>
<td>80‐100 mg/kg PO q 48 hr</td>
<td>Insufficient proof of efficacy to recommend use in treatment of dermatophytosis.</td>
</tr>
</tbody>
</table>

Infections as causes of scaling

- FIV
- FeLV exfoliative dermatitis
- Feline poxivirus
Infections as causes of scaling

Malassezia dermatitis

Leishmaniasis

Environmental causes of scaling

Feline solar dermatitis:
Actinic dermatitis
Feline Solar Dermatitis

- Lesions
  - Erythema
  - Alopecia
  - Ulcers
  - Crusts
- May progress to squamous cell CA

Feline Solar Dermatitis

- Treatment
  - Keep out of sun
  - Sunscreens
  - Amputate ear tips
Environmental causes of scaling

- Fireplace/heater/heating pad
  - Dx: history and season of year
  - Tx: moisturizers

Endocrinopathies with scaling

- hypercortisolism

Endocrinopathies with scaling

- hypercortisolism
Endocrinopathies with scaling

Cell Differentiation

- Regulated by growth factors
  - Epidermal growth factor
  - Transforming growth factor α and TGF β
- Retinoids (promote)
- Calcium (promote)
- Zinc (promotes)
- Extracellular matrix
- Mesenchymal tissue
Zinc Deficiency $\rightarrow$ incomplete differentiation of keratinocytes

Orthokeratotic hyperkeratosis  Parakeratotic hyperkeratosis

Lethal Acrodermatitis in Bull Terriers

- Growth retardation
- Progressive acrodermatitis
- Chronic pyoderma
- Chronic paronychia
- Diarrhea
- Pneumonia
- Abnormal behavior
- Median survival ~ 7 months

Lethal Acrodermatitis in Bull Terriers

- Usually smallest puppies in litter
- Weak nursing but even more difficulty eating hard foods
- High arched hard palate—food impacts in the roof of the mouth
- Splayed toes
- Nails grow fast but distorted
Lethal Acrodermatitis

Lethal Acrodermatitis in Bull Terriers

- Autosomal recessive trait
- Clinical pathology
  - Neutrophilia
  - Hypercholesterolemia
  - Low SAP, ALP
  - Low plasma zinc
- Immunological evaluation:
  - Depressed lymphocyte blastogenesis responses
- Antagene (France) 2-4 ml blood EDTA—genetic test being developed

Lethal Acrodermatitis in Bull Terriers

- Management (poor prognosis)
  - Treat skin infections (cephalexin, clavulanic acid-amoxicillin; ketoconazole if yeast)
  - Zinc + fatty acid supplements
  - Aloe vera cream
  - Liquid diet often best
  - Probiotics or yogurt
**Zinc Deficiency: Type I**

**Zinc Deficiency: Type II**

**Zinc Responsive Dermatoses**

- **Diagnosis**
  - Signalment
  - Clinical signs
  - Biopsy: parakeratosis
  - Serum Zinc levels
  - Response to Tx

- **Treatment**
  - Zinc sulfate 10 mg/kg
  - Zinc methionine 1.7 mg/kg
  - Good diet (avoid excess calcium)
  - Essential fatty acids
Vitamin A-Responsive Dermatosis

Vitamin A-Responsive Follicular Hyperkeratosis

Follicular hyperkeratosis

May respond to Vitamin A or retinoid therapy
Vitamin A-Responsive Follicular Hyperkeratosis

- Diagnosis
  - Biopsy
- Management
  - Vitamin A 1000 IU/kg/day
  - Antiseborrheic shampoos

Metabolic/Nutritional Disorders with scaling

Main nutrition, malnutrition, malabsorption

Metabolic/Nutritional Disorders with scaling

Superficial necrolytic dermatitis
Metabolic/Nutritional Disorders with scaling

Infections/Inflammation → epidermal hyperplasia
Infections → epidermal hyperplasia and/or epidermolysis

Exfoliating toxins ETA and ETB → “Staph scalded skin syndrome”

Dermatophytes produce extracellular proteinases that digest keratin

Infectious/Infiltrative diseases causing scaling

Leishmaniasis
Infiltrative diseases causing scaling

CTCL

Neoplastic/paraneoplastic disorders with scaling

CTCL
Neoplastic/paraneoplastic disorders with scaling

CTCL
Neoplastic/paraneoplastic disorders with scaling

Feline paraneoplastic alopecia

Neoplastic/paraneoplastic disorders with scaling

Exfoliative dermatitis associated with thymoma

Feline Thymoma

- Reported cases have had generalized erythematous dermatitis
- Skin is thickened with cracks and fissures
- Hair coat is scurfy and scaly
- Skin biopsies show lymphocytic interface dermatitis
- Thoracic radiographs consistent with thymoma
- Surgical removal usually curative

New Zealand Veterinary Journal 51(5), 244-247, 2003
Review pathomechanisms of scaling

- Increased keratinocyte proliferation
  - Hereditary or response to inflammation

- Disorders of keratinization
  - Hereditary, infectious, nutritional or neoplastic

- Disorders affecting lipid bilayers (stratum corneum)
  - Hereditary, nutritional, metabolic or environmental

---

Review pathomechanisms of scaling

- Disorders affecting desquamation
  - Hereditary, infectious, metabolic or environmental

- Disorders affecting cell cohesion
  - Hereditary, infectious, immune-mediated

- Infiltrative disorders of epidermis
  - Infectious, inflammatory, neoplastic

---

Key Considerations

- Breed and age
- Littermates or parents affected?
- Predominant symptoms
- Is inflammation present?
  - What is cause?
  - What are contributing factors?
- Evaluate for parasites, infections, allergies, nutritional deficiencies, endocrine/metabolic diseases, neoplastic/paraneoplastic disorders
- Biopsies helpful in many cases
Review of major points

• Cutaneous scaling/crusts are non-specific
• Signalment, history, PE findings, skin scrapings, skin cytology, laboratory findings are helping in prioritizing DDX
• CE is essential
• Symptomatic treatment may help improve skin barrier function, HOWEVER
• Key is identifying and controlling underlying factors