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Canine Scaling Disorders

P Profile

Definition

Scale is an accumulation of corneocyte fragments (desquamated) from the stratum corneum. Normal fragments are not visible to the naked eye; abnormal fragments accumulate and become visible without magnification. Degradation of intercellular lipids or corneodesmosomes or proliferation of basal keratinocytes will create scale. Scales may be loose or adherent; white, tan, yellow or brown in color; and fine or quite large.

Scales can be classified as primary or secondary. Primary lesions are formed as a direct result of underlying disease (eg, vitamin A-responsive dermatosis, Schnauzer comedone syndrome, ichthyosis); secondary lesions are changes in the skin due to primary lesions or self trauma, infection, or medication (eg, allergic skin disease, atopy, bacterial pyoderma).

Systems. Cutaneous, unless the scale is the result of endocrinopathies or neoplasia.

Genetic Implications.

- **Allergic skin disease:** Any breed
- **Color dilution alopecia (CDA):** Any breed with blue or fawn coat color. Most commonly seen in Doberman pinschers, Great Danes, and Yorkshire terriers
- **Endocrinopathies:** Hypothyroidism
- **Ichthyosis:** Most commonly identified in golden retrievers; also seen in Norfolk terriers, West Highland white terriers, soft-coated wheaten terriers, Cavalier



Color dilution alopecia. Note that the alopecia affects only breeds with blue- or fawn-colored coats.



Another example of CDA. Note the fine scale associated with the alopecic area.

- King Charles spaniels, and Jack Russell terriers
- **Nutritional-responsive:**
 - Zinc-responsive (Alaskan malamutes, Siberian huskies, bull terriers)
 - Vitamin A-responsive (cocker spaniels)
- **Primary seborrhea:** Extremely uncommon but overdiagnosed. This is usually a secondary seborrhea; when identified it is

most common in buff-colored cocker spaniels, Irish setters, West Highland white terriers, springer spaniels, and Doberman pinschers.

- **Sebaceous adenitis:** Standard poodles, Akitas, German shepherd dogs, Samoyeds, Vizslas
- **Schnauzer comedone syndrome (SCS):** Schnauzers

Incidence/Prevalence. Scaling is a common finding in dogs as a result of allergic skin disease and/or environmental dryness. Depending on the local gene pool, inherited forms seem to have some regional differences throughout the country.

Signalment

Species. Unusual in cats but common in dogs

Breed Predislection. For primary disease, see **Genetic Implications** under **Profile**. As a secondary disease, any breed is at risk.

Age and Range.

- **Allergic skin disease:** Young adult dogs
- **CDA:** Dogs less than 1 year of age
- **Endocrinopathies:** Hypothyroidism— young adults; hyperadrenocorticism— middle-aged and geriatric dogs
- **Ichthyosis:** Dogs less than 1 year of age. Please note that ichthyosis may not be apparent at birth—it becomes apparent as the dog matures (especially true in golden retrievers).
- **Nutritional-responsive:** Juvenile to young adult dogs
- **Primary seborrhea:** Begins in dogs at

CDA = color dilution alopecia; SCS = Schnauzer comedone syndrome

continues



Scale from a dog with cutaneous lymphoma. Note the size of the scale (large).



Scale forming an epidermal collarette. Note the circular pattern formed by the lesion.



Waxy scale due to seborrhea, resulting in matted hair

- less than 1 year of age
- **SCS:** Young adult dogs
- **Sebaceous adenitis:** Young adult to middle-aged dogs

Gender. Equal incidences

Causes

Causes of scale can be divided into congenital and acquired. Since scaling is a reaction pattern, it is important to understand that anything that affects proliferation, differentiation, or desquamation of the epidermis may produce scale.

- **Acquired:** Due to inflammation, endocrinopathies, nutritional factors, and environmental factors
- **Congenital:** Due to CDA, primary seborrhea, sebaceous adenitis, SCS, and ichthyosis

Risk Factors

- **Acquired:** Low fat diet, frequent bathing, low environmental humidity, allergic skin disease, and endocrinopathies
- **Congenital:** Poor genetics/gene pool

Pathophysiology

Acquired:

- **Endocrinopathies** (hypothyroidism, hyperadrenocorticism)
 - Cause decrease in protein production (enzymes responsible for normal desquamation), changes in cutaneous fatty acid concentrations (increase in

oleic acid; oleic acid is not effective in preventing transepidermal water loss [TEWL], the quantity of water that passes through the epidermis and evaporates into the environment), and decrease in sebaceous gland secretion due to atrophy

- **Environmental factors**
 - Without adequate water content, enzymes necessary for separation of corneocytes (normal desquamation) will not be produced, leading to scale.
- **Inflammation**
 - Cytokines are produced when the epidermis is damaged. Cytokines and inflammatory eicosanoids stimulate epidermal proliferation in an effort to remove the noxious insult. Epidermal hyperproliferation leads to defective differentiation of the keratinocytes.
 - Causes of inflammation include:
 - **Allergic skin disease:** Atopy, cutaneous adverse food reaction, flea allergy dermatitis
 - **Infectious:** Bacterial, fungal (eg, dermatophytosis or *Malassezia*), ectoparasites (eg, *Demodex* or *Cheyletiella* mites, fleas)
 - **Neoplastic:** Epitheliotropic lymphoma
- **Nutritional factors**
 - Deficiencies in a variety of vitamins, minerals, proteins, or essential fatty acids may cause scaling.
 - Vitamin A-responsive, zinc-responsive, or fatty acid-responsive dermatoses are

not deficiencies, but respond to supplementation.

Congenital:

- **CDA:** Clumping of melanin with the hair shaft results in fragile hairs that fracture easily.
- **Ichthyosis:** Defects in structure or function of intracellular keratinocyte organelles (lamellar granules), enzymes (transglutaminase), and cytoskeleton of the keratinocyte. The end result is increase in cohesiveness of stratum corneum cells or in cellular proliferation.
- **Primary seborrhea:** Cellular defect leading to a hyperproliferative epidermis (decrease in epidermal turnover time). Please note that diagnosis of primary seborrhea is a diagnosis of exclusion.
- **Sebaceous adenitis:** Due to destruction of sebaceous glands
- **SCS:** Due to keratin plugging of sebaceous glands

Signs

History. May point you in the direction of the underlying disease.

- **Allergic skin disease/ectoparasites:** Pruritus
- **CDA:** Nonpruritic alopecia in a young dog
- **Endocrinopathies:** Hypothyroidism—lethargy, heat seeking, weight gain; hyperadrenocorticism—polyuria/polydipsia/polyphagia, panting, muscle weakness. Both of these endocrinopathies are

CDA = color dilution alopecia; SCS = Schnauzer comedone syndrome; TEWL = transepidermal water loss



An adult dog with generalized demodicosis. Demodicosis with scaling is called the squamous form.



A dog with generalized dermatophytosis. Note that crusting and erythema are also present.



A dog with allergic contact dermatitis. Note the erythema associated with the scale.

nonpruritic unless there is a secondary bacterial or *Malassezia* infection.

- **Ichthyosis:** Scaling in a young dog

Physical Examination. May also point you in the direction of the underlying disease.

- **Allergic skin disease/ectoparasites:** Evidence of pruritus (alopecia, crusts, erosions, lichenification, excoriations), presence of parasites
- **CDA:** Alopecia in dilute hair areas, presence of comedones, secondary pyoderma (papules and pustules)
- **Endocrinopathies:**
 - Hypothyroidism: Weight gain, bilateral symmetrical alopecia, bradycardia, hyperpigmentation, myxedema, superficial bacterial folliculitis, *Malassezia* infection
 - Hyperadrenocorticism: Panting, muscle weakness, bilateral symmetrical alopecia, hyperpigmentation, pendulous abdomen, hepatomegaly, superficial bacterial folliculitis, *Malassezia* dermatitis
- **Sebaceous adenitis:**
 - Standard poodle form: Seen in standard poodles, Akitas, German shepherd dogs. Signs include adherent white scaling, follicular waxy casts (matted hair from the scale), varying degrees of hypotrichosis (including alopecia), dull appearance to the hair coat, pruritus if a secondary pyoderma (papules and pustules) or *Malassezia* is present, and loss of curls in standard poodles.
 - Short-coated form: Seen in Vizslas, dachshunds. Dogs present with annular

areas of scaling and alopecia most commonly affecting the trunk. At this stage it is not uncommon to mistake this disease for a superficial bacterial folliculitis. However, failure to respond to appropriate antibiotics (eg, cephalosporins, potentiated amoxicillin, potentiated sulfas, etc) rules out superficial bacterial folliculitis.

- **SCS:** Comedone formation on the dorsum

Dx Diagnosis

Definitive Diagnosis (See Aids & Resources for diagnostics used to identify various conditions)

- **History**
 - Age of onset
 - Environment
 - Topical therapies
 - Degree of pruritus: If present, consider ectoparasites, allergic skin disease, bacterial pyoderma, or *Malassezia* dermatitis
 - Presence of constitutional signs (lethargy, polyuria/polydypsia, excessive panting)
- **Dermatologic exam**
 - Appearance of hair coat
 - Texture and density (may be dull, dry, thinning, or normal)
 - Hypotrichosis or alopecia (if present, may be symmetrical, focal, or multifocal)
 - Presence of ectoparasites
 - Primary lesions in addition to scale
 - Alopecia: Posttraumatic, consider hypersensitivities; spontaneous,

consider endocrinopathies, follicular dysplasia (eg, CDA), or drugs/medications (eg, glucocorticoids, chemotherapy)

- Comedones: Bacterial, fungal, parasitic (*Demodex*), endocrinopathy (hyperadrenocorticism), primary cornification defects (eg, SCS, acne) or medications/drugs (eg, glucocorticoids)
- Crusts: Bacterial, fungal (dermatophytes, *Malassezia*), autoimmune (pemphigus, vasculitis), ectoparasites
- Follicular casts: Vitamin A-responsive dermatosis, primary seborrhea, sebaceous adenitis, or demodicosis
- Hyperpigmentation: Endocrinopathies
- Macules/patches: Epitheliotropic lymphoma, hypersensitivities or irritants
- Nodules/tumors: Epitheliotropic lymphoma, mast cell tumor
- Papules/plaques: Bacterial, fungal, neoplastic (epitheliotropic lymphoma); if not follicularly oriented, consider ectoparasites

continues



Scale due to sebaceous adenitis. Note that the scale is tightly adhered to the hair—this is known as a follicular cast (**arrows**).



Standard poodle with sebaceous adenitis. Note the scale, the loss of curls, and the partial alopecia of the trunk.

- Pustules: Bacterial, fungal (dermatophytes, *Malassezia*), autoimmune (pemphigus foliaceus), parasitic (*Demodex*)
- **Laboratory findings**
 - Skin scrapings, impression smears, flea combing to identify ectoparasites
 - Fungal or bacterial culture
 - Impression smears to identify *Malassezia* and bacteria
 - CBC, chemistry profile, and urinalysis may reveal changes that occur with hypothyroidism (elevated cholesterol, mild nonregenerative anemia) or hyperadrenocorticism (mature neutrophilia, eosinopenia, lymphopenia, elevated alkaline phosphatase, hyposthenuria, proteinuria, lower urinary tract infection)
 - If hypothyroidism or hyperadrenocorticism suspected: Thyroid testing or adrenal function testing (LDDS or ACTH-stimulation test)
 - Biopsy to diagnose:
 - CDA
 - Ichthyosis
 - SCS
 - Sebaceous adenitis
 - Biopsy will identify:
 - Allergic skin disease, but not the etiology
 - Endocrinopathy, but not the etiology
 - Seborrhea, but not differentiate primary versus secondary
 - Nutritional-responsive dermatosis,

but biopsy may not always differentiate these dermatoses from allergic skin disease

Differential Diagnosis

Depends on age of onset, breed, coat color, presence of pruritus or constitutional signs. See **Causes** under **Profile**.

Tx Treatment

Inpatient or Outpatient

Outpatient treatment is normally recommended.

Client Education

Most causes of scaling can be treated but not cured. However, scaling caused by nutritional-responsive or environmental causes, cutaneous adverse food reaction, or ectoparasites can be cured while atopic dermatitis can only be controlled. Please note that any dog affected with diseases that have a genetic basis should be neutered.

Medications

- Treat the underlying disease (See **Table** on next page).
- Therapy for clinical signs—may include:
 - Shampoos \$ (See **Aids & Resources** for product suggestions)
 - Humectants (applied as a final rinse after a bath or misted directly onto the skin from the bottle) \$ (See **Aids & Resources** for product suggestions)

- Calcitriol \$\$\$-\$\$\$\$
 - 10 ng/kg Q 24 H
 - Weekly measurement of electrolytes and parathyroid hormone is recommended when oral formulation is used.
- Synthetic retinoids \$\$\$\$
 - Isotretinoin (1–3 mg/kg PO Q 12–24 H)
 - Acitretin (0.5–1 mg/kg Q 24 H)
- Natural vitamin A \$\$\$\$\$
 - 625–800 IU/kg PO Q 24 H
- Oral supplementation with linoleic acid may be beneficial in cases of scaling. Linoleic acid is the most important fatty acid in regard to preventing TEWL and maintaining barrier function. \$

Cost Key

\$ = < \$100	\$\$\$\$ = \$500-1000
\$\$ = \$100-250	\$\$\$\$\$ = > \$1000
\$\$\$ = \$250-500	

Follow-Up

Patient Monitoring

Since the underlying disease causing scaling is frequently not curable (eg, allergies, CDA, sebaceous adenitis) it is important to examine the patient anytime clinical signs (such as pruritus) recur or new lesions appear. If the dog is on synthetic retinoids (which may cause hypercholesterolemia, hypertriglycerolemia, hepatopathies, and decreased tear production), a baseline CBC, serum chemistry profile, and Schirmer's tear test should be performed. These tests should be repeated 30 days after therapy begins and then every 6 months. If the lipid profile of a patient becomes elevated, changing to a low fat diet will frequently resolve it. ■

See **Aids & Resources**, back page, for references, contacts, and appendices.

Article archived on www.cliniciansbrief.com

ACTH = adrenocorticotrophic hormone; CBC = complete blood count; CDA = color dilution alopecia; LDDS = low-dose dexamethasone suppression; SCS = Schnauzer comedone syndrome

Treatment of Dogs with Scaling Disorders

Disease	Treatment
Allergic	<p>Depending on the type of allergy, treatment may include:</p> <ul style="list-style-type: none"> • Hypoallergenic diet (cutaneous adverse food reaction) • Selamectin, fipronyl, imidacloprid (flea allergy dermatitis) • Antihistamines (See Aids & Resources), pulse-dose prednisone, allergen specific immunotherapy, modified cyclosporine (5 mg/kg Q 24 H) <p><i>Mild scaling:</i> A, omega-3 & -6 fatty acids <i>Moderate scaling:</i> SS, humectant, omega-6 fatty acid <i>Pyoderma:</i> BPS, humectant, omega-3 & -6 fatty acids <i>Malassezia:</i> H, humectant, omega-3 & -6 fatty acids</p>
Bacterial	<p><i>Mild scaling:</i> H, humectant, cephalixin 22–33 mg/kg Q 8–12 H for min 21 days <i>Moderate to severe scaling:</i> BPS, humectant, cephalixin 22–33 mg/kg Q 8–12 H for min 21 days</p>
Color dilution alopecia, primary seborrhea, sebaceous adenitis	<p><i>Mild scaling:</i> A, omega-6 fatty acid <i>Moderate to severe scaling:</i> SS, humectant, omega-6 fatty acid Synthetic retinoids or vitamin A 600–800 IU/kg Q 24 H. Please note that these patients are prone to secondary infections. If present, topical therapy should include antimicrobials.</p>
Dermatophytosis	<p>Azole-containing shampoo with or without H; for dogs, griseofulvin-microsized (NOT ultramicrosized) 50 mg/kg Q 24 H. Therapy should be continued until 3 fungal cultures are negative.</p>
Environmental factors	<p><i>Mild scaling:</i> A, omega-6 fatty acid <i>Moderate scaling:</i> SS, humectant, omega-6 fatty acid</p>
Hyperadrenocorticism	<p><i>Mild scaling:</i> A, omega-3 & -6 fatty acids <i>Moderate scaling:</i> SS, humectant, omega-3 & -6, fatty acids, lysodren or trilostane</p>
Hypothyroidism	<p><i>Mild scaling:</i> A, omega-6 fatty acid <i>Moderate scaling:</i> SS, humectant, omega-6 fatty acid, L-thyroxine</p>
Ichthyosis	<p>SS, humectant, oral vitamin A, omega-6 fatty acid</p>
Malassezia	<p>Azole-containing shampoo with or without H, ketoconazole 5 mg/kg Q 24 H with food or itraconazole 5 mg/kg 2 consecutive days/wk for min 21 days</p>
Neoplastic	<p><i>Mild scaling:</i> A, omega-3 & -6 fatty acids <i>Moderate scaling:</i> SS, humectant, omega-3 & -6 fatty acids. Lomustine, an alkylating agent, has been effective in dogs with cutaneous lymphoma at a dose of 50 mg/m² Q 21–30 days. Please familiarize yourself with this drug before using.</p>
Parasitic	<p><i>Mild scaling:</i> A, omega-6 fatty acid <i>Moderate to severe scaling:</i> SS, humectant, omega-6 fatty acid Ivermectin, selamectin, fipronyl, imidacloprid</p>
Schnauzer comedone syndrome	<p>BPS, vitamin A 600–800 IU/kg Q 24 H</p>
Vitamin-A responsive	<p>SS or BPS, vitamin A 600–800 IU/kg Q 24 H</p>
Zinc-responsive	<p>SS or BPS, zinc methionine 1–2 mg/kg Q 12 H or zinc gluconate 5 mg/kg Q 24 H</p>

A = hypoallergenic moisturizing shampoo; BPS = benzoyl peroxide with sulfur shampoo; H = chlorhexidene-containing shampoo; SS = sulfur salicylic acid shampoo