Allergies
Atopy

Basics

DEFINITION
Predisposition to become allergic to normally innocuous substances, such as pollens (grasses, weeds, and trees), molds, house dust mites, epithelial allergens, and other environmental allergens

PATHOPHYSIOLOGY
• Susceptible animals become sensitized to environmental allergens by producing allergen-specific IgE, which binds to receptor sites on cutaneous mast cells; further allergen exposure (inhalation, percutaneous absorption) causes mast cell degranulation, which is a type I immediate hypersensitivity reaction, and results in the release of histamine, proteolytic enzymes, cytokines, chemokines, and many other chemical mediators.
• Non-IgE antibodies (IgGd) and a late-phase reaction (8–12 hr) may also be involved.

SYSTEMS AFFECTED
• Skin/Exocrine—pruritus, xerosis, or generalized dryness of the skin; recurrent superficial pyoderma and yeast infections; recurrent bilateral otitis externa
• Ophthalmic—recurrent bilateral conjunctivitis
• Respiratory, Reproductive, and Gastrointestinal—reported, but not well documented

GENETICS
• Canine—although there is an inherited predisposition, the mode of inheritance is unknown and other factors may also be important.
• Feline—unclear

INCIDENCE/PREVALENCE
• Canine—true incidence unknown; estimated at 3%–15% of the canine population; reported to be the second most common allergic skin disease
• Feline—unknown; generally believed to be much lower than that for dogs

GEOGRAPHIC DISTRIBUTION
Canine—recognized worldwide; local environmental factors (temperature, humidity, and flora) influence the seasonality, severity, and duration of signs.

SIGNALMENT
Species
Dogs and cats

Breed Predilections
• Canine—any breed, including mongrels; because of genetic predisposition, it may be recognized more frequently in certain breeds or families, which can vary geographically.
• In the United States (canine)—Boston terriers, Cairn terriers, dalmatians, English bulldogs, English setters, Irish setters, Lhasa apsos, miniature schnauzers, pugs, Sealyham terriers, Scottish terriers, West Highland white terriers, wire-haired fox terriers, and golden retrievers
• Feline—none reported
Mean Age and Range
Canine—mean age at onset 1–3 years; range 3 months–6 years; signs may be so mild the first year that they are not noted but are usually progressive and clinically apparent before 3 years of age.

Predominant Sex
• Both sexes are probably affected equally.

SIGNS
General
• Hallmark sign—pruritus (itching, scratching, rubbing, licking)
• Primary lesions may occur, but most cutaneous changes are believed to be produced by self-induced trauma.

Historical Findings
• Facial, pedal, or axillary pruritus
• Early onset
• Family history of atopy
• May be seasonal
• Recurring skin or ear infections
• Temporary response to glucocorticoids
• Symptoms progressively worsen with time

Physical Examination Findings
• Areas most commonly affected—interdigital spaces, carpal and tarsal areas, muzzle, periocular region, axillae, groin, and pinnae
• Lesions—vary from none to broken hairs or salivary discoloration to erythema, papular reactions, crusts, alopecia, hyperpigmentation, lichenification, excessively oily or dry seborrhea, and hyperhidrosis (apocrine sweating)
• Secondary bacterial and yeast skin infections (common)
• Chronic relapsing otitis externa
• Conjunctivitis may occur.

CAUSES
• Airborne pollens (grasses, weeds, and trees)
• Mold spores (indoor and outdoor)
• House dust mite
• Animal danders
• Insects (controversial)

RISK FACTORS
• Temperate environments with long allergy seasons and high pollen and mold spore levels
• Concurrent pruritic dermatoses, such as flea allergy dermatitis and food hypersensitivity (summation effect)

Diagnosis
DIFFERENTIAL DIAGNOSIS

• Food hypersensitivity—may cause identical lesion distribution and physical examination findings but should be nonseasonal; may occur concurrently with atopy; differentiation is made by noting response to hypoallergenic diet.

• Flea bite hypersensitivity—most common cause of seasonal pruritus in many geographical regions; may occur concurrently with atopy; differentiation is made by noting lesion distribution, response to flea control, and results of intradermal skin testing.

• Sarcoptic mange—often occurs in young or recently stray dogs; usually causes severe pruritus of the ventral chest, lateral elbows, lateral hocks, and pinnal margins; multiple skin scrapings and/or complete response to a trial of miticidal therapy are indicated to rule out sarcoptic mange.

• Secondary pyoderma—usually caused by *Staphylococcus intermedius*; characterized by follicular papules, pustules, crusts, and epidermal collarettes

• Secondary yeast infections—usually caused by *Malassezia pachydermatis*; characterized by erythematous, scaly, crusty, greasy, and very malodorous body folds and intertriginous areas; demonstration of numerous budding yeast organisms by skin cytology and obtaining a favorable response to antifungal therapy are diagnostic.

• Contact dermatitis (allergic or irritant)—may cause severe erythema and pruritus of the feet and thinly haired areas of the ventral abdomen; history of exposure to a known contact sensitizer or irritant, response to a change of environment, and patch testing may be diagnostic; thought to be rare in dogs and cats.

CBC/BIOCHEMISTRY/URINALYSIS

Eosinophilia—rare in dogs without concurrent flea infections; common in cats

OTHER LABORATORY TESTS

*Serologic Allergy Tests*

• Tests to measure the amount of allergen-specific IgE antibody in the patient's serum are commercially available.

• Advantages over IDST—availability; large areas of hair do not have to be shaved

• Disadvantages—frequent false-positive reactions; limited number of allergens tested; inconsistent assay validation and quality control (may vary with the laboratory used)

• Reliability in cats is unknown.

IMAGING

N/A

DIAGNOSTIC PROCEDURES

*IDST*

• Small amounts of test allergens are injected intradermally and wheal formation is measured.

• Most accurate method of identifying offending allergens for possible avoidance or inclusion in an immunotherapy prescription.

• Results are sometimes difficult to interpret in cats owing to the relatively small wheals produced.

PATHOLOGIC FINDINGS

• Gross lesions—see Physical Examination Findings

• Skin biopsy—may help rule out other differential diagnoses; results are usually not pathognomonic.
• Dermatohistopathologic changes—acanthosis, mixed mononuclear superficial perivascular dermatitis, sebaceous gland metaplasia, and secondary superficial pyoderma

**Treatment**

**APPROPRIATE HEALTH CARE**
Outpatient

**NURSING CARE**
N/A

**ACTIVITY**
Avoid offending allergens when possible.

**DIET**
Essential fatty acid supplementation may be beneficial in some cases.

**CLIENT EDUCATION**

• Explain the progressive nature of the condition.
• Inform client that it rarely goes into remission and cannot be cured.
• Inform client that some form of therapy may be necessary for life.

**SURGICAL CONSIDERATIONS**
N/A

**Medications**

**DRUGS OF CHOICE**

*Immunotherapy (Hyposensitization)*

• Administration (usually SC injections) of gradually increasing doses of the causative allergens to affected patients in an attempt to reduce their sensitivity
• Allergens selected—based on allergy test results, patient history, and knowledge of local flora
• Indicated when it is desirable to avoid or reduce the amount of corticosteroids required to control signs, when signs last longer than 4–6 months per year, or when nonsteroidal forms of therapy are ineffective
• Successfully reduces pruritus in 60–80% of dogs and cats
• The response is usually slow, often requiring 3–6 months and up to 1 year.

*Corticosteroids*

• May be given for short-term relief and to break the itch–scratch cycle
• Should be tapered to the lowest dosage that adequately controls pruritus
• Best choices—prednisone suspension (0.5 to 1.0 mg/kg SC or IM); prednisone or methylprednisolone tablets (0.2 to 0.5 mg/kg PO q48h)
• Repository injectable corticosteroids should be avoided in dogs.
• Cats may require methylprednisolone acetate treatment (4 mg/kg SC or IM).

*Antihistamines*

• Less effective than are corticosteroids
• Efficacy as a sole treatment is probably in the 10%–20% range.
• May act synergistically with essential fatty acid supplements
• Corticosteroid therapy can often be avoided or given at a reduced dosage when used concurrently.
• Dogs—hydroxyzine (1–2 mg/kg PO q8h), chlorpheniramine (0.2–0.4 mg/kg PO q12h), diphenhydramine (2.2 mg/kg PO q8h), and clemastine (0.04–0.10 mg/kg PO q12h)
• Cats—chlorpheniramine (0.5 mg/kg PO q12h); efficacy estimated at 10%–50%

CONTRAINDICATIONS
N/A

PRECAUTIONS
• Corticosteroids—use judiciously in dogs to avoid iatrogenic hyperglucocorticism and associated problems, aggravation of pyoderma, and induction of demodicosis.
• Antihistamines—can produce drowsiness, anorexia, vomiting, diarrhea, and even increased pruritus; use with caution in patients with cardiac arrhythmias.

POSSIBLE INTERACTIONS
The antihistamine astemizole have been associated with life-threatening cardiac arrhythmias in humans when administered concomitantly with imidazole antifungal drugs.

ALTERNATIVE DRUGS
• Frequent bathing in cool water with antipruritic shampoos can be beneficial.
• Supplementation with ω-3 and ω-6 fatty acids helps some pruritic patients; some studies have indicated that ω-3 (eicosapentaenoic acid 66 mg/kg/day) may be more effective than ω-6 (linoleic acid 130 mg/kg/day); other studies suggest that a 5:1 ratio of ω-6:ω-3 in the diet is indicated.
• Tricyclic antidepressants (doxepin 1.0–2.0 mg/kg PO q12h; or amitriptyline 1.0–2.0 mg/kg PO q12h) have been given to dogs as antipruritics but their overall effectiveness and mode of action is unclear; not extensively studied in the cat

Follow-Up

PATIENT MONITORING
• Examine patient every 2–8 weeks when a new course of therapy is started
• Monitor pruritus, self-trauma, pyoderma, and possible adverse drug reactions
• Once an acceptable level of control is achieved, examine patient every 3–12 months
• CBC, serum chemistry profile, and urinalysis—recommended every 6–12 months for patients on chronic corticosteroid therapy

PREVENTION/AVOIDANCE
• If the offending allergens have been identified through allergy testing, the owner should undertake to reduce the animal’s exposure as much as possible.
• Minimizing other sources of pruritus (e.g., fleas, food hypersensitivity, and secondary skin infections) may reduce the level of pruritus enough to be tolerated by the animal.

POSSIBLE COMPLICATIONS
Secondary pyoderma and concurrent flea allergy dermatitis

EXPECTED COURSE AND PROGNOSIS
• Not life-threatening unless intractable pruritus results in euthanasia
• If left untreated, the degree of pruritus worsens and the duration of signs last longer each year of the animal's life.
• Only rare cases spontaneously resolve.

Miscellaneous

ASSOCIATED CONDITIONS
• Flea allergy dermatitis
• Food hypersensitivity
• Pyoderma
• Otitis externa

AGE-RELATED FACTORS
Severity worsens with age.

ZOONOTIC POTENTIAL
None

PREGNANCY
• Corticosteroids—contraindicated during pregnancy
• Antihistamines—safety during pregnancy has not been established.

SYNONYMS
• Canine allergic inhalant dermatitis
• Canine atopic dermatitis
• Canine atopic disease

SEE ALSO
• Fleas and Flea Control
• Food Reactions (dermatologic)
• Otitis Externa and Media
• Pyoderma

ABBREVIATION
IDST = intradermal skin test

Fleas and Flea Control

Basics

DEFINITION
• Flea allergy dermatitis—hypersensitivity reaction to antigens in flea saliva with or without evidence of fleas and flea dirt
• Flea infestation—large number of fleas and a large amount of flea dirt with or without a flea allergy dermatitis

PATHOPHYSIOLOGY
• Flea bite hypersensitivity (FBH)—caused by a low molecular weight hapten and two high molecular weight allergens that help initiate the allergic reaction
• High molecular weight allergens—increased binding to dermal collagen; when bound, form a complete antigen necessary for eliciting FBH
• Flea saliva—contains histamine-like compounds that irritate skin
• Intermittent exposure favors FBH; continuous exposure is less likely to result in hypersensitivity.
• Both IgE and IgG antiflea antibodies have been noted.
• Immediate and delayed hypersensitivity reactions have been noted.
• Late-phase IgE-mediated response—part of FBH reaction; occurs 3–6 hr after exposure
• Cutaneous basophil hypersensitivity—part of FBH reaction; an infiltration of basophils into the dermis; mediated either by IgE or IgG; subsequent exposures cause the basophils to degranulate; manifests as immediate and delayed hypersensitivity

SYSTEMS AFFECTED
Skin/Exocrine

GENETICS
FBH—unknown inheritance pattern; more common in atopic breeds

INCIDENCE/PREVALENCE
Varies with climatic conditions and flea population

GEOGRAPHIC DISTRIBUTION
• FBH—may occur anywhere; nonseasonal only in climates that are warm and humid year round and in animals housed indoors

SIGNALMENT
Species
Dogs and cats

Breed Predilection
FBH—any breed; most common in atopic breeds

Mean Age and Range
FBH—rare < 6 months of age; average age range, 3–6 years, but may be seen at any age

Predominant Sex
N/A

SIGNS
Historical Findings
• Compulsive biting
• Chewing (corncob nibbling)
• Licking, primarily in the back half of the body but may include the antebrachial regions
• Cats—scratching around the head and neck
• Signs of fleas and flea dirt

Physical Examination Findings
• Depends somewhat on the severity of the reaction and the degree of exposure to fleas (i.e., seasonal vs. year-round)
• Finding fleas and flea dirt is beneficial, although not essential, for the diagnosis of FBH; sensitive animals require a low exposure and tend to overgroom, making identification of the parasites difficult.
• Dogs—lesions concentrated in a triangular area of the caudal-dorsal-lumbosacral region; caudal aspect of the thighs, lower abdomen, inguinal region, and cranial forearms usually involved; primary lesions are papules; secondary lesions (e.g., hyperpigmentation, lichenification, alopecia, and scaling) common in uncontrolled FBH; secondary folliculitis and furunculosis may be seen.

• Cats—several patterns are seen; most common is a miliary crusting dermatitis in a wedge-shaped pattern over the caudal dorsal lumbosacral region and often around the head and neck; other presentations are alopecia of the inguinal region with or without inflammation or eosinophilic plaques and other forms of eosinophilic granuloma complex.

• Exposure to other animals and previous flea treatment should be ascertained.

CAUSES
See Pathophysiology

RISK FACTORS
FBH—intermittent exposure to fleas increases the likelihood of development; commonly seen in conjunction with atopy

Diagnosis

DIFFERENTIAL DIAGNOSIS
• Food allergy
• Atopy
• Sarcoptic mange
• Cheyletiellosis
• Primary keratinization defects
• Diagnosis is best based on the history and laboratory tests.

CBC/BIOCHEMISTRY/URINALYSIS
• Usually normal
• Cats—hyper eosinophilia may be detected

OTHER LABORATORY TESTS
• Skin scrapings—negative
• Flea combings—fleas or flea dirt, but often nothing is found
• RAST and ELISA—variable accuracy; both false-positive and false-negative results reported

IMAGING
N/A

DIAGNOSTIC PROCEDURES
• Diagnosis usually based on historical information and distribution of lesions
• Fleas or flea dirt is supportive but is often quite difficult to find, especially in cats.
• Identification of *Dipylidium caninum* segments is supportive.
• Intradermal allergy testing with flea antigen—reveals positive immediate reactions in 90% of flea-allergic animals; delayed reactions (24–48 hr) may sometimes be observed in allergic animals that show no immediate reaction.
• The most accurate test may be response to appropriate treatment.
PATHOLOGIC FINDINGS

- Superficial perivascular dermatitis
- Eosinophilic intraepidermal microabscesses—strongly suggest FBH
- Eosinophils as a major cellular component of the dermis—supportive of FBH
- Histopathologic evaluation—cannot accurately differentiate FBH from atopy, food allergy, or other hypersensitivities

Treatment

APPROPRIATE HEALTH CARE
Outpatient therapy

CLIENT EDUCATION

- Inform owners that there is no cure for FBH.
- Advise owners that flea-allergic animals often become more sensitive to flea bites as they age.
- Inform owners that controlling exposure to fleas is currently the only means of therapy; hyposensitization has not worked satisfactorily.

Medications

DRUGS OF CHOICE

- Corticosteroids—anti-inflammatory dosages for symptomatic relief while the fleas are being controlled
- Antihistamines—symptomatic relief
- Fipronil (GABA antagonist)—monthly spot treatment for cats and dogs and spray treatment for dogs; activity against fleas and ticks; resistant to removal with water; excellent safety and efficacy profile
- Imidacloprid—monthly spot treatment for cats and dogs; excellent safety and efficacy profile
- Systemic treatments—limited benefit because they require a flea bite that has already initiated FBH; may help animals with flea infestation; primarily licensed for use in only dogs; lufenuron, a chitin inhibitor, available as an oral formulation for cats and dogs and as an injection for cats; permethrin available as a spot treatment and reputed to have some repellent activity; imidacloprid (flea adulticide) available as a spot treatment for cats and dogs.
- Sprays—usually contain pyrethrins and pyrethroids (synthetic pyrethrins) with an insect growth regulator or synergist; generally effective < 48–72 hr; advantages are low toxicity and repellent activity; disadvantages are frequent applications and expense.
- Indoor treatment—fogs and premises sprays; usually contain organophosphates, pyrethrins, and/or insect growth regulators; apply according to manufacturer's directions; treat all areas of the house; can be applied by the owner; advantages are weak chemicals and generally inexpensive; disadvantage is labor intensity; premises sprays concentrate the chemicals in areas that most need treatment.
• Professional exterminators—advantages are less labor-intensive; relatively few applications; sometimes guaranteed; disadvantages are strength of chemicals and cost; specific recommendations and guidelines must be followed.

• Inert substances—boric acid, diatomaceous earth, and silica aerogel; treat every 6–12 months; follow manufacturer’s recommendations; very safe and effective if applied properly

• Outdoor treatment—concentrated in shaded areas; sprays usually contain pyrethroids or organophosphates and an insect growth regulator; powders are usually organophosphates; product containing nematodes (*Steinerma carpocapsae*) is very safe and chemical-free.

**PRECAUTIONS**

• Insecticidal sprays and dips should not be used on dogs and cats ≤ 3 months, unless otherwise specified on the label.

• Pyrethrin/pyrethroid-type flea products—adverse reactions include depression, hypersalivation, muscle tremors, vomiting, ataxia, dyspnea, and anorexia.

• Organophosphates—adverse reactions include hypersalivation, lacrimation, urination, defecation, vomiting, diarrhea, miosis, fever, muscle tremors, seizures, coma, and death.

• All pesticides must be applied according to label directions.

• Toxicity—if any signs are noted, the animal should be bathed thoroughly to remove any remaining chemicals and treated appropriately.

• Rodents and fish are very sensitive to pyrethrins.

**POSSIBLE INTERACTIONS**

• Organophosphate treatments—do not use more than one form at a time.

• Topical organophosphates—avoid in cats, very young animals (< 3 months of age), and sick or debilitated animals.

• Straight permethrin sprays or spot-ons—do not use in cats.

• Cythioate—contraindicated in heartworm-positive dogs and greyhounds

• Piperonyl butoxide—do not use in concentrations > 1% in cats.

**ALTERNATIVE DRUGS**

• Powders—usually contain organophosphates or carbamates; advantage is high residual effectiveness; disadvantages are dry skin and toxicity; organophosphates and carbamates should be avoided in cats.

• Dips, sprays, powders, and foams—dips usually contain organophosphates and synthetic pyrethrins and should not be used more than once per week; follow manufacturer’s instructions for safest and best results; after repeated use, these agents can be drying or irritating; newer, safer spot treatments have essentially replaced these products.

**Follow-Up**

**PATIENT MONITORING**

• Pruritus—a decrease means the FBH is being controlled.

• Fleas and flea dirt—absence is not always a reliable indicator of successful treatment in very sensitive animals.
PREVENTION/AVOIDANCE
• See Medications
• Year-round warm climates—year-round flea control
• Seasonally warm climates—begin flea control in May or June

POSSIBLE COMPLICATIONS
• Secondary bacterial infections
• Acute moist dermatitis
• Acral lick dermatitis

EXPECTED COURSE AND PROGNOSIS
Prognosis is good, if strict flea control is instituted.

Miscellaneous

ASSOCIATED CONDITIONS
Approximately 80% of atopic dogs are also allergic to flea bites.

AGE-RELATED FACTORS
Organophosphates—use with utmost caution in old animals; not recommended for use in very young animals (< 3 months).

ZOONOTIC POTENTIAL
In areas of moderate to severe flea infestation, people can be bitten by fleas; usually papular lesions are located on the wrists and ankles.

PREGNANCY
• Corticosteroids and organophosphates—do not use in pregnant bitches and queens.
• Carefully follow the label directions of each individual product to determine its safety.

SYNONYMS
• Flea bite allergy
• Flea bite hypersensitivity

ABBREVIATIONS
• ELISA = enzyme-linked immunosorbent assay
• GABA = \(\gamma\)-aminobutyric acid
• RAST = radioallergosorbent test

Food Reactions (Dermatologic)

Basics

DEFINITION
Pruritic, nonseasonal reactions associated with ingestion of one or more substances in the animal's food

PATHOPHYSIOLOGY
• Pathogenesis not completely understood
• Immediate and delayed reactions to specific ingredients—documented in the veterinary literature; immediate reactions presumed to be type I hypersensitivity reactions; delayed owing to type III or IV
• Food intolerance—nonimmunologic, idiosyncratic reaction; involves metabolic, toxic, or pharmacologic effects of offending ingredients
• Food hypersensitivity is the most common term used, because it is not easy to distinguish between immunologic and idiosyncratic reactions.

SYSTEMS AFFECTED
• Skin/Exocrine—pruritus in any location on the body; otitis externa
• Gastrointestinal—vomiting; diarrhea; more frequent bowel movements
• Nervous—very rare; seizures have been documented with food hypersensitivity/intolerance

GENETICS
N/A

INCIDENCE/PREVALENCE
• Approximately 5% of all skin diseases and 10–15% of all allergic skin diseases in dogs and cats are the result of food hypersensitivity
• Third-most-common pruritic skin disease in the dog; second-most-common in the cat
• Percentages vary greatly with clinicians and geographical location.

GEOGRAPHIC DISTRIBUTION
N/A

SIGNALMENT
Species
Dogs and cats

Breed Predilections
None

Mean Age and Range
Any age

Predominant Sex
None

SIGNS
General Comments
A wide range of signs that can mimic any of the other hypersensitivity reactions

Historical Findings
• Nonseasonal pruritus of any body location
• Poor response to anti-inflammatory doses of glucocorticoids suggests a food hypersensitivity.
• Vomiting
• Diarrhea
• Excessive borborygmus, flatulence, and frequent bowel movements

Physical Examination Findings
• *Malassezia* dermatitis, pyoderma, and otitis externa
• Plaques
• Pustules
• Erythema
• Crusts
• Scale
• Self-induced alopecia
• Excoriation
• Lichenification
• Hyperpigmentation
• Urticaria
• Angioedema
• Pyotraumatic dermatitis

CAUSES
• Immune-mediated reactions—result of the ingestion and subsequent presentation of one or more glycoproteins (allergens) either before or after digestion; sensitization may occur at the gastrointestinal mucosa, after the substance is absorbed, or both.
• Nonimmune (food intolerance) reactions—result of ingestion of foods with high levels of histamine or substances that induce histamine either directly or through histamine-releasing factors

RISK FACTORS
• Unknown
• It is speculated that in juvenile animals intestinal parasites or intestinal infections may cause damage to the intestinal mucosa, resulting in the abnormal absorption of allergens and subsequent sensitization.

Diagnosis

DIFFERENTIAL DIAGNOSIS
• Flea bite hypersensitivity—usually confined to the caudal half of the body; often seasonal
• Atopy—associated with pruritus on the face, ventrum, and feet; often seasonal; if pruritus first occurs at 1; 6 months or > 6 years of age, then food hypersensitivity may be more likely than inhalant allergy.
• Drug reactions—history of drug administration before the development of signs and improvement after withdrawal of the suspected drug confirms the diagnosis.
• Scabies—pruritus often very specific in the location (ears, elbows and hocks); mites in skin scrapings and response to specific therapy confirm the diagnosis.

CBC/BIOCHEMISTRY/URINALYSIS
No important changes

OTHER LABORATORY TESTS
N/A

IMAGING
N/A
DIAGNOSTIC PROCEDURES

Food Elimination Diet

- Definitive test for food hypersensitivity
- Tailored to the individual patient
- The diet must be restricted to one protein and one carbohydrate to which the animal has had limited or no previous exposure.
- It may take up to 13 weeks for maximum improvement of the clinical signs.
- If the patient is sensitive to one or more foods, noticeable improvement will be seen by the 4th week of the diet.

Challenge and Provocation Diet Trials

- Used if the patient improves on the elimination diet
- Challenge—feed the patient with the original diet; a return of the signs confirms that something in the diet is causing the signs; the challenge period should last until the signs return but no longer than 10 days.
- Provoke (provocation diet trial)—if the challenge confirmed the presence of a food hypersensitivity, add single ingredients to the elimination diet; test ingredients include a full range of meats (beef, chicken, fish, pork, lamb), a full range of grains (corn, wheat, soybean, rice), eggs, and dairy products; the provocation period for each ingredient should last up to 10 days or less if signs develop sooner (dogs usually develop signs within 1–2 days); results guide the selection of commercial foods that do not contain the offending substance(s).

PATHOLOGIC FINDINGS

- Skin biopsies—not diagnostic; help confirm other differentials
- Histopathologic findings—variable; common findings suggest hypersensitivity; a secondary pyoderma or Malassezia infection may be seen.

Treatment

APPROPRIATE HEALTH CARE
Outpatient management

NURSING CARE
N/A

ACTIVITY
No change

DIET
Avoid any food substances that caused the clinical signs to return during the provocation phase of the diagnosis.

CLIENT EDUCATION

- Make sure the client understands the principles involved in each phase of the diagnostic test diets.
- Inform client to eliminate treats, chewable toys, vitamins, and other chewable medications (e.g., heartworm preventative), which may contain ingredients from the patient’s previous diet.
- Outdoor pets must be confined to prevent foraging and hunting, which might alter the test diet.
• Provide handouts for clients to take home.
• Advise client that all family members must be aware of the test protocol and must help keep the test diet clean and free of any other food sources.

**SURGICAL CONSIDERATIONS**
N/A

**Medications**

**DRUGS OF CHOICE**
• Systemic antipruritic drugs—may be useful during the first 2–3 weeks of diet trial to control self-mutilation
• Antibiotics or antifungal medications—useful for secondary pyoderma or Malassezia infections

**CONTRAINDICATIONS**
• Antibiotics that are known to have anti-inflammatory effects (e.g., tetracycline, erythromycin, and trimethoprim-potentiated sulfas)
• Glucocorticoids and antihistamines must be discontinued for at least 10–14 days while on the diet trial to allow correct assessment of the animal's response.

**PRECAUTIONS**
N/A

**POSSIBLE INTERACTIONS**
Chewable vitamins and heartworm medications may contain offending food substances.

**ALTERNATIVE DRUGS**
None

**Follow-Up**

**PATIENT MONITORING**
Examine patient and evaluate and document the pruritus and clinical signs every 3–4 weeks.

**PREVENTION/AVOIDANCE**
• Avoid intake of any of the proteins included in the previous diet.
• Treats and chewable toys should be limited to known safe substances (e.g., apples, vegetables).

**POSSIBLE COMPLICATIONS**
Other causes of pruritus (e.g., flea bite hypersensitivity; atopy; and external parasites such as sarcoptic, *Notoedres*, and *Cheyletiella* mites) can mask the response to the food elimination diet trial.

**EXPECTED COURSE AND PROGNOSIS**
• Prognosis is good, if food ingredients are the only cause of the pruritus and offending ingredients are avoided.
• Rarely a dog or cat may develop hypersensitivity to new substances, which may require a new elimination diet trial.
• Any other hypersensitivities (flea or atopy) must also be treated.

**Miscellaneous**
ASSOCIATED CONDITIONS
- Superficial pyoderma
- *Malassezia* dermatitis
- Otitis externa

AGE-RELATED FACTORS
Animals who develop pruritus for the first time at < 6 months or > 6 years of age are more likely to have food hypersensitivity than atopy.

ZOONOTIC POTENTIAL
None

PREGNANCY
N/A

SYNONYMS
- Food allergy
- Food intolerance

IMAGES

Contact Dermatitis

Basics

OVERVIEW
- Irritant contact dermatitis (ICD) and allergic contact dermatitis (ACD)—two rare and distinctly different pathophysiologic syndromes with similar clinical signs
- ICD—results from direct damage to keratinocytes by exposure to a particular compound; damaged keratinocytes induce an inflammatory response directed at the skin.
- ACD—an immunologic event requiring sensitization, memory, and elicitation: Langerhans cells process antigens that penetrate the skin and present them to naive T cells within lymph nodes; sensitized T cell clones (memory cells) then proliferate and
circulate throughout the body; Langerhans cells encounter the antigens again and present them to sensitized T cells, resulting in an immunologic response.

**SIGNALMENT**

- Dogs and cats
- ICD—occurs at any age as a direct result of the irritant nature of the offending compound
- ACD—rare in young animals; most animals are chronically exposed to the antigen; extremely rare in cats, except when exposed to D-limonene-containing insecticides
- Predisposed to ACD—German shepherds
- Increased risk to ACD (unsubstantiated)—French poodles, wire-haired fox terriers, Scottish terriers, West Highland white terriers, and golden retrievers

**SIGNS**

*Lesions*

- Location depends on the way in which the antigen is contacted; commonly limited to glabrous skin and regions frequently in contact with the ground (chin, ventral neck, sternum, ventral abdomen, inguinum, perineum, scrotum, and ventral contact regions of the tail and interdigital areas)
- The thick hair coat of dogs is an effective barrier against contactants.
- In classic cases, extreme erythroderma stops abruptly at the hairline.
- Initially consist of erythema and swelling, leading to papules and plaques; vesicles are uncommon.

*Others*

- Reactions to topical medications (most often otic preparations) are usually localized; generalized reactions, resulting from shampoos or insecticide sprays, are less common.
- Pruritus—moderate to severe; severe is most common.
- A seasonal incidence may indicate that the offending antigen is a plant or outdoor compound.

**CAUSES & RISK FACTORS**

- Inflammatory dermatitis—may increase the penetration of antigens through the skin; thus may facilitate ACD.
- Reported offending substances—plants, mulch, cedar chips; fabrics, rugs and carpets, plastics, rubber, leather, metal, concrete; soaps, detergents, floor waxes, carpet and litter deodorizers; herbicides, fertilizers, insecticides (including newer topical flea treatments), flea collars; topical preparations and medications

**Diagnosis**

**DIFFERENTIAL DIAGNOSIS**

- Atopy
- Food allergy
- Drug eruptions
- Parasite hypersensitivity or infestation
- Insect bites
- Pyoderma
- *Malassezia* dermatitis
- Dermatophytosis
- Demodicosis
- Lupus erythematosus
- Seborrheic dermatitis
- Solar dermatitis
- Thermal injuries
- Trauma from rough surfaces

**CBC/BIOCHEMISTRY/URINALYSIS**
No abnormalities

**OTHER LABORATORY TESTS**
N/A

**IMAGING**
N/A

**DIAGNOSTIC PROCEDURES**
- Closed-patch testing—sometimes helpful (corticosteroids and NSAIDs must be discontinued 3–6 weeks before testing); use materials directly from the environment or a standard patch test kit for humans (Hermal, Oak Hill, NY) applied to the skin under a bandage for 48 hr.
- Best diagnostic test—eliminate contact irritant or antigen, follow with provocative exposure testing
- Bacterial cultures to define secondary pyoderma may be performed, if needed.
- Because the hair coat can protect the skin from contact with antigen, clipping a patch of hair in a nonaffected region should result in development of a local reaction.

**PATHOLOGIC FINDINGS**
- Skin biopsies—intraepidermal vesiculation and spongiosis; superficial dermal edema with perivascular mononuclear cell infiltrate in ICD and ACD; polymorphonuclear cell infiltrate in ICD; leukocyte exocytosis common
- Histologic findings—vary with duration of antigen contact
- Primary changes—often obscured by secondary changes owing to pruritus and excoriation

**Treatment**
- Eliminate offending substance(s).
- Bathe with hypoallergenic shampoos to remove antigen from the skin.
- Create mechanical barriers, if possible—socks, T-shirts, restriction from environment

**Medications**

**DRUGS**
- Systemic corticosteroids—prednisone (0.25–0.5 mg/kg PO q24h for 3–5 days; then q48h for 2 weeks)
- Topical corticosteroids for focal lesions
- Recent studies report success in dogs with pentoxifylline (10 mg/kg PO q12h).

**CONTRAINDICATIONS/POSSIBLE INTERACTIONS**
Pentoxifylline—do not administer with alkylating agents, cisplatin, and amphotericin B; cimetidine may increase serum levels of pentoxifylline.

**Follow-Up**

**PREVENTION/AVOIDANCE**
Remove offending substances from the environment

**EXPECTED COURSE AND PROGNOSIS**

**ICD**
- Acute condition—may occur after only one exposure; can be manifested within 24 hr of exposure.
- Steroids are rarely helpful.
- Lesions resolve 1–2 days after irritant removal.

**ACD**
- Requires months to years of exposure for the hypersensitivity to develop
- Re-exposure results in the development of clinical signs 3–5 days following exposure; signs may persist for several weeks.
- Responds well to corticosteroids; but the pruritus returns after discontinuation if the antigenic stimulus has not been removed.
- Hyposensitization is disappointing.
- Prognosis—good if the allergen is identified and removed; poor if the allergen is not identified, which may then require lifelong treatment

**Miscellaneous**

**ABBREVIATIONS**
- ACD = allergic contact dermatitis
- ICD = irritant contact dermatitis

**IMAGES**