FACIAL DERMATOSES OF THE DOG AND CAT – A THERAPEUTIC UPDATE

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Facial Dermatoses in Immature Dogs
1. Inflammatory, variably pruritic
   a. Demodex
   b. Dermatophytosis
c. Dermatomyositis
d. Juvenile cellulitis
e. chin folliculitis and furunculosis (canine acne)

2. Pruritic (lesions induced by pruritus)
a. Atopy
b. Food Sensitivity
c. scabies

Facial Dermatoses in Mature Dogs
1. Acute, symmetric, inflammatory
   a. Eosinophilic folliculitis and furunculosis
   b. Drug eruption

2. Chronic, symmetric or variably symmetric, inflammatory (+/- depigmenting)
a. Discoid lupus erythematosus
b. Systemic lupus erythematosus
c. Pemphigus foliaceus
d. Pemphigus erythematosus
e. Pemphigus vulgaris
f. Bullous pemphigoid
g. Dermatophytosis
h. demodicosis
i. Superficial Necrolytic Dermatitis (Hepatocutaneous Syndrome)
j. Zinc Responsive dermatosis
k. Facial fold dermatitis
l. Vogt-Koyanagi-Harada - like (Uveodepigmentation) Syndrome
m. Drug eruption
n. Leishmaniasis

3. Acute/Chronic, pruritic (pruritus creating lesions)
a. Atopy - also look for contributions to pruritus from Malassezia
b. Food sensitivity - also look for contributions to pruritus from Malassezia
c. Scabies
d. Drug eruption

4. Diseases of the Planum nasale (+/- depigmenting)
a. Idiopathic nasal hypopigmentation
b. Discoid lupus erythematosus
c. Systemic lupus erythematosus
d. Pemphigus foliaceus
e. Pemphigus vulgaris
f. Bullous pemphigoid
g. Vitiligo
h. Solar dermatitis
i. Squamous cell carcinoma
j. Idiopathic nasal hyperkeratosis
k. Xeromycteria – parasympathetic nose
l. Idiopathic nasal hyperkeratosis
m. Xeromycteria – parasympathetic nose
n. Idiopathic granulomatous/pyogranulomatous dermatitis
o. Chronic nasal discharge
p. Bacterial dermatitis - esp. in GSD
q. Dermatophytosis
r. Leukocytoclastic vasculitis – Scottish Terrier
s. Arteritis (ulcerative dermatosis) – St. Bernard
t. Hereditary parakeratotic hyperkeratosis
u. Cutaneous lymphoma (mycosis fungoides)
v. Distemper

5. Diseases of the oral mucocutaneous junction
a. DLE/SLE, cutaneous lupus
d. lip fold pyoderma
b. Pemphigus complex
e. demodicosis
c. Mucocutaneous pyoderma
CANINE DISEASES OF THE NASAL PLANUM

**Idiopathic nasal depigmentation (hypopigmentation)** – Dudley nose, Snow nose
Most common in Golden retrievers, Labrador retrievers, Samoyeds, Siberian huskies, but other breeds noted to be affected: Afghan hounds, German shepherds, Poodles, Doberman pinschers, Irish setters, and others. Loss of pigment in planum (black to various shades of brown; in the arctic breeds, just about, but not quite to pink). Usually begins in young dogs, 1-2 years of age. Although pigment is lost, reticular pattern of the planum remains present. Nose does not depigment to pink (as seen with vitiligo and other immune mediated diseases such as DLE and pemphigus). This disease does not sun sensitize the nose (do not see sunburn). May wax and wane. May see worsening during winter (hence the term snow nose). Higher incidence in retriever breeds, artic working breeds but can be seen in many, many breeds. No therapy necessary. Anecdotal suggestion that melatonin may be of some benefit?

**Vitiligo** is thought to involve autoimmunity to melanocytes. Breeds predisposed include the Belgian Tervuren, German Shepherd dog, Rottweiler and Doberman Pinscher. Non-inflammatory asymptomatic depigmentation is most commonly noted on the planum, lips, muzzle, and buccal mucosa. There may be focal or widespread leukotrichia and/or depigmentation of the nails. The natural course of the disease may wax and wane. Histology shows a lack of melanin in affected areas. On occasion, a mild superficial dermal accumulation of lymphocytes may be noted. References generally state that there is no therapy but for preventing/managing solar damage. However, there are anecdotal reports of using a combination of oral folic acid, Vitamin C and injectable Vitamin B12. An 80 lb dog was given 1 mg folic acid PO BID, 50 microgram B12 IM every 14 days and 500 mg Vitamin C PO BID. Others have reported some success with 10 drops of stock solution of an alcoholic extract of blueberry. We have had some success in treating mildly inflammatory forms of the disease with the combination of tetracycline and niacinamide or topical tacrolimus (see below under “discoid lupus erythematosus”).

**Bacterial dermatitis of the planum nasale** tends to be under-diagnosed in clinical practice. Crusting, loss of the normal reticular pattern of the planum and depigmentation may all be caused by bacterial infection. The German Shepherd dog appears to be uniquely predisposed to type of problem. In this breed, dermatitis is most commonly noted over the lateral nares, but may extend up and over the planum. It is also important to note that chronic nasal discharge in any breed may result in the creation of a micro-environment that may predispose to secondary bacterial infections of the planum. Biopsies are usually suggestive (dermatitis, changes consistent with a mucocutaneous pyoderma – predominance of plasma cells in the superficial epidermis). The diagnosis is supported by assessing response to systemic and/or topical antibiotics.

**Discoid Lupus Erythematosus** is the most common inflammatory disease of the nasal planum in the dog. Lesions are generally restricted to the head. Lesions of the planum consist of variable degrees of loss of the normal cobblestone appearance, depigmentation, inflammation, crusting, atrophy and erosion. Erosive nasal lesions can be profound, resulting in significant hemorrhage. There is variable involvement of the bridge of the nose, lip margins, periocular region and medial aspect of the pinna. It is not uncommon to see inflammation of the third eyelids (plasma cell infiltrate). The Collie, Shetland Sheepdog, Australian Shepherd, German Shepherd dog and Siberian Husky exhibit a breed predilection for this disease. There is no systemic symptomatology. ANA titers are negative. The lesions are variably photo-aggravated. Histopathologic changes suggestive of this disease include vacuolar degeneration of the basal cell layer, single cell necrosis of keratinocytes (mostly in basal cell layer), pigmentary
incontinence and an accumulation of predominantly mononuclear cells in the superficial dermis. These histopathologic changes are as those for Systemic Lupus Erythematosus. Differentiation between these diseases must therefore be made on the basis of assessing systemic involvement and ANA titer. In our clinic, as many as half of the cases that meet the clinical criteria of DLE have moderate to severe superficial lymphocytic/plasmacytic infiltrates and marked pigmentary incontinence in their biopsies, but fail to meet several of the other criteria required for a histologic diagnosis of DLE. These dogs appear to respond to therapy as for DLE. Whether these dogs suffer from a variant of DLE or some other immune mediated disease is not known. For all manifestations of DLE or DLE-like disease, sun restriction and the use of topical sunscreens are strongly advocated. Very mild disease is treated with topical glucocorticoids, oral Vitamin E and essential fatty acids PO (usually marginal responses to these therapies in our regional area). Mild to moderate cases are treated with either topical tacrolimus or tetracycline and niacinamide. Topical Tacrolimus has been a very effective therapy for this disease and has given us a non-steroidal alternative for management. Tacrolimus inhibits T-lymphocyte activation and is 10 to 100 times as immunosuppressive as cyclosporine. It is minimally absorbed. A commercial source of tacrolimus is available in 0.03% and 0.1% concentrations (Protopic, buy Fujisawa). It is available in 30 and 60 gm tubes. The product is very expensive (30 gms of 0.1% usually sells to the owner for $75.00). Luckily, however, a little goes a long way! Lesions are initially treated with the 0.1% concentration BID. Once maximal benefit is noted the frequency is reduced to once daily for 1-2 months, then once every other day. Maintenance is usually achieved with once daily or once every other day regimens. It is suggested that the product be applied with a gloved finger although absorption through the skin is minimal. The product may prove irritating on affected skin but this is rare. Overall response to therapy occurs in about 75-80% of patients. Tetracycline and niacinamide are noted to benefit approximately 50-60% of patients (500 mg of each tetracycline and niacinamide given TID until maximal benefit noted, then reduce to BID for couple of months, then q 24 hours). Doxycycline has been used to replace TID tetracycline (doxycycline 5 mg/kg BID). If the lesions are moderate to severe, therapy is usually initiated with glucocorticoids and tetracycline/niacinamide, then maintenance is attempted with tetracycline/niacinamide alone. Tacrolimus and tetracycline/niacinamide failures have generally been treated with glucocorticoids and tetracycline/niacinamide, then maintenance is attempted with tetracycline/niacinamide alone. Tetracycline/niacinamide failures have generally been treated with glucocorticoid monotherapy (usually starting at 2-4 mg/kg/day; dosage dictated by severity of lesions). Refractory DLE cases are treated with glucocorticoids and azathioprine (must wait at least 3 months to assess effects of azathioprine). Alternate therapies include glucocorticoids with gold salts or chlorambucil. Dapsone has also been used.

**Pemphigus foliaceus** also commonly affects the planum (inflammation, depigmentation, crusting, erosion/ulceration, pustules) although other areas of the body are usually involved early in the course of the disease (bridge of nose, medial pinna, periocular, pad/skin junction and foot pads. Variants of the disease include a form restricted to the feet and a chronic facial form. Breeds that appear to be more prone to secondary bacterial infections associated with PF include the Chow Chow and German Shepherd. Infections may complicate histopathologic interpretation. Especially in these breeds, consideration should be given to treating with systemic antibiotics prior to biopsy. A diagnosis is supported by finding acantholytic keratinocytes in unbroken pustules or impression smears and confirmed by biopsy. In general (see below for specifics), mild to moderate, chronic facial forms of this disease are treated with tetracycline/niacinamide. Approximately 30% of these cases respond. Only about 10-20% of our generalized cases respond to tetracycline/niacinamide. Mild cases of generalized disease that fail tet./niac. are treated with glucocorticoids alone. Moderate to severe cases are started on glucocorticoids and azathioprine. We feel that 65% of our cases in this category do well and have minimal complications related to the medications. Glucocorticoid/azathioprine failures are treated with glucocorticoids and chlorambucil, gold salts or dapsone. Topical tacrolimus is a “newer” therapy that may significantly benefit lesions. Because of its expense and the relative widespread areas of involvement associated with
the disease, tacrolimus is usually used to “top off” other therapies to produce a more rapid resolution or help with the treatment of more refractory lesions. Therapy is generally initiated on a BID basis. It should be noted that Pemphigus vulgaris and Bullous Pemphigoid may also affect the planum. These diseases are comparatively rarely encountered in clinical practice.

Therapy – Pemphigus foliaceus

Owner education of paramount importance. Emphasis must be placed on the realization that these diseases are usually incurable, requiring life-long therapy. Appears to be a great deal of individual variation concerning response to therapy.

1. Glucocorticoids remain the cornerstone of therapy. By themselves they may be enough to induce and maintain remission in cases of pemphigus erythematosus or milder cases of pemphigus foliaceus (usually starting at 2 mg/kg/day prednisone). With moderate to severe cases of pemphigus foliaceus or pemphigus vulgaris, higher dosages of steroids will be required to induce remission (i.e. 3-6 mg/kg/day of oral prednisone/prednisolone) and higher dosages will be required to maintain it. In such instances the side effects of steroids commonly become intolerable to both the animal and owner (PU/PD, severe polyphagia, weight gaining, muscle weakness, depression, panting) and may actually become the eventual reason for considering euthanasia. For this reason when the patient is moderately to severely clinically affected, the tendency is to use glucocorticoids along with other immunosuppressive therapies at the onset (e.g. azathioprine). This will usually allow for the attainment of a more rapid remission with an opportunity to more quickly reduce steroid dosages. Maintenance dosages of steroids are also usually lower when adjunctive immunosuppressives are used.

Protocol Alternatives:

a. Mild cases of pemphigus foliaceus; pemphigus erythematosus
   Prednisone/prednisolone

   1 mg/kg BID for first 1-2 weeks
   1 mg/kg q24 hr for 1-2 weeks
   Gradually reduce dose to 1 mg/kg every other day over a 2 week period
   Gradually reduce to lowest every other day dose required to control symptomatology. Generally this winds up in the 0.5 – 1.0 mg/kg every other day range but to minimize steroid side effects, we try to make it less than 0.5 mg/kg every other day.

   Signs of significant steroid side effects would warrant a more rapid reduction in dosage. If the problem remains refractory at these lower dosages, another immunosuppressive drug (e.g. azathioprine) should be added to the protocol.

b. Moderate to severe cases of pemphigus foliaceus; pemphigus vulgaris

   Prednisone along with azathioprine:

   Prednisone/prednisolone:

   2 mg/kg BID for 1-2 weeks
   1 mg/kg BID for 1-2 weeks
1 mg/kg q24 hr for 2 weeks
Gradually reduce dose to 1 mg/kg every other day over a 2 week period. Gradually reduce to lowest every other day dose required to control symptomatology.

Azathioprine (Imuran)

2 mg/kg q24 hours. Maintain on daily Imuran until evidence of good clinical response to the combination therapy (usually 2-4 weeks) and then switch to 2 mg/kg every other day. For every 6 months of excellent control of the disease, can reduce the Imuran dose by __. However, we generally never reduce the dose to less than 1.0 mg/kg every other day.

Azathioprine toxicity:
a. Myelosuppression: generally only a problem while on daily therapy for longer than 3-4 weeks. While on daily therapy, recommend CBC and platelet counts every two weeks; do weekly if on daily therapy longer than one month. Repeat this screen 2 weeks after switching to every other day therapy, then 1 month after this, then in 3 months then every 4-6 months thereafter.
White blood cell counts below 5,000/mcl or platelet counts below 150,000/mcl would warrant discontinuation of daily azathioprine. Hematologic parameters usually return to normal in 1-2 weeks. At this time therapy can be re-established at the maintenance dose of 2 mg/kg every other day. If toxicity is noted on the every other day regimen, therapy is stopped until values are normalized. Maintenance therapy can then be re-started at 50 – 75% of the maintenance dosage.
b. Hepatotoxicity – uncommon; usually look at liver enzymes 2-4 weeks in to therapy.
c. Vomition, diarrhea - uncommon

Eventual goal: 2 mg/kg of azathioprine every other day and the lowest every other day dose of glucocorticoid required to maintain remission. The azathioprine and steroid are usually given on alternate days.

2. Tacrolimus – topical therapy used BID (to initiate therapy) – for focal lesions; expensive; usually used as an adjunctive therapy for this syndrome

3. Tetracycline/niacinamide – success in controlling about 40% of chronic facial pemphigus foliaceus; 10% of generalized pemphigus foliaceus

4. Chlorambucil (Leukeran) – because of expense, for smaller dogs. May work when azathioprine fails as an adjunctive therapy

5. Chrysotherapy – gold salt therapy (aurothioglucose; Solganol)
Has been successfully used to manage pemphigus vulgaris, pemphigus foliaceus, and pemphigus erythematosus. Gold salts are initially used in conjunction with corticosteroids. Usually give gold salts at 1 mg/kg IM per week until see benefit. If no benefit by 20 weeks, discontinue. If benefit noted, gradually work back to lowest frequency of administration required to control problem (e.g. once every 2-4 weeks). An oral source of gold salts is available, but does not appear to be as
effective as the parenteral medication. Concurrent glucocorticoids are gradually reduced and may eventually be discontinued.

**Pemphigus erythematous** may closely mimic the clinical presentation of DLE. The diseases are differentiated histologically. Therapies are as for DLE with similar response rates.

**Proliferative arteritis** restricted to the planum has been noted in the St. Bernard and Giant Schnauzer. It is an ulcerative dermatitis of the anterior aspect (pfiltrum) of the planum. Ulceration, when severe, may result in significant hemorrhage from the site. There appears to be a familial tendency for this disease. Histologic examination shows an arteritis. Lesions do not appear to respond to antibiotics. Response to glucocorticoids is variable (topical or systemic) as is response to tetracycline and niacinamide. We have had some success in managing affected individuals with topical cyclosporine (as noted above under Discoid Lupus Erythematosus). Consideration should be given to using topical tacrolimus.

**Hereditary nasal parakeratosis** has been noted in Labrador retrievers and crosses. An autosomal recessive mode of inheritance is suspected. Lesions are first noted between 6 and 12 months of age. They consisted of grayish or brownish adherent accumulations of dry and rough keratin. In more severe cases, fissures and erosions develop. Some dogs experience depigmentation of the remaining nasal planum. The dermatitis does not appear to be exacerbated by UV light exposure. Histopathology shows parakeratotic hyperkeratosis and a sub-basal lympho-plasmacytic infiltration within the superficial dermis. Zinc methionine, cephalaxin, Vitamin A alcohol and topical tretinoin have failed to be of benefit. Improvement of the lesions was obtained with topical vitamin E, petrolatum and propylene glycol.

**Idiopathic nasal hyperkeratosis** is most commonly noted in middle aged to older dogs with the Cocker spaniel being over represented. It has also been suggested that it is a senile change. It may be concurrently associated with pad hyperkeratosis. The nose becomes dry, rough and hyperkeratotic, especially on the dorsum of the nose. Fissures, erosions and ulcers are only occasionally noted. There is no depigmentation or inflammation. This is an important observation which helps to clinically differentiate the lupus or pemphigus group of diseases (which may also be hyperkeratotic). The diagnosis is generally made on a clinical basis. Therapeutic considerations include the daily topical administration of Kerasolv (DVM pharmaceuticals; salicylic acid, sodium lactate and urea in propylene glycol), Bag Balm or tretinoin gel (Retin-A;Ortho). Petrolatum may also be used. More rapid removal of the hyperkeratotic debris may be facilitated by pre-hydrating the planum (water compresses for 5-10 minutes) prior to application. Oral vitamin A may also be of benefit. Dosages are usually in the range of 8,000 to 20,000 Units BID. Although there is nothing “new” about this disease, it must be differentiated from a disease that not as well recognized by most – nasal hyperkeratosis associated with xeromycteria.

**Nasal Hyperkeratosis Associated with Xeromycteria (Parasympathetic nose)** - The lateral nasal gland of the dog is located in the mucosa of the maxillary recess, near the opening of this recess into the nasal cavity. This gland is mostly responsible for the moisture of the nasal mucosa and the moisture observed at the external nares of the dog. Moisture from here is thought to translocate over the surface of the planum. This gland receives parasympathetic innervation with its fibers coursing with the facial nerve through the petrous temporal bone. Lacrimal glands also receive parasympathetic innervation. Xeromycteria associated with KCS may suggest that the preganglionic parasympathetic fibers proximal to the pterygopalatine ganglion are likely to have been damaged. Clinically, the petrous temporal bone should be examined for evidence of disease. In the dog, this warrants the evaluation of the patient for otitis media. Lesions may be either unilateral or bilateral. In affected dogs, resolution of otitis media may
result in spontaneous improvements in nasal secretions and hyperkeratosis of the planum. In those cases in which an association with otitis media is not noted, or where resolution of the otitis media does not improve the planum, topical therapy is as for idiopathic nasal hyperkeratosis. Pilocarpine therapy has also been noted to be of benefit.

**Idiopathic, sterile, granulomatous and pyogranulomatous dermatoses** may also be restricted to the planum at presentation. In general, lesions are usually multiple, firm, papular, nodular or plaquiform, variably haired and variably inflamed. On occasion, they may ulcerate. Areas of predilection include the head (especially the bridge of the nose and muzzle) and distal extremities. Less frequently, lesions can also develop on the pinnae, eyes, trunk and abdomen. When restricted to the planum, the syndrome has been called “clown nose”. These lesions must be differentiated from the canine histiocytosis which can have similar presentations. Diagnosis is by rule out (bacterial and fungal cultures), and biopsy. In the dog, therapy is with topical tacrolimus, oral glucocorticoids, tetracycline/niacinamide or glucocorticoids and azathioprine. After achieving remission for prolonged periods, some cases will be able to have medications discontinued.

**Cutaneous T-cell lymphoma (mycosis fungoides)**, usually seen in older dogs, may have a multitude of various clinical presentations, one of which periocular, perioral and/or planum nasale inflammation / depigmentaiton. MF may look very much like an immune mediated disease.

**FACIAL DERMATOSES OF THE CAT**

**Pruritic Dermatoses Directed at the Head and Neck**
1. Notoedric mange
2. Otodectes infestation
3. Flea bite hypersensitivity
4. Dermatophytosis
5. Bacterial Infection
6. Atopy
7. Food Sensitivity
8. Mosquito bite hypersensitivity (bridge of nose, periocular region, ears, flexor surfaces of carpi, junction of food pads and haired skin)
9. Drug eruption
10. Autoimmune Skin disease (e.g. Pemphigus foliaceus)
11. Sterile granuloma/pyogranuloma
12. Herpesvirus facial dermatitis
13. Facial dermatosis of Persian cats
14. Bowen’s disease (squamous cell carcinoma in situ)
15. Squamous cell carcinoma
16. Mast cell tumor
17. Idiopathic (10% of cases?)

**Dermatoses of the Chin**
1. Feline acne
2. Atopy
3. Food sensitivity
4. Malassezia dermatitis
5. Bacterial dermatitis
6. Dermatophytosis
7. Eosinophilic granuloma (may or may not be related to underlying hypersensitivity)
8. Demodicosis
9. Discoid Lupus Erythematosus
10. Pemphigus foliaceus

**Dermatoses of the nasal planum**
1. Solar keratosis/squamous cell carcinoma
2. Mosquito bite hypersensitivity
3. Pemphigus complex (esp. pemphigus foliaceus)
4. Discoid lupus Erythematosus
5. Systemic lupus Erythematosus
6. Cryptococcosis
7. Sterile granuloma/pyogranuloma
8. Vitiligo
9. Herpesvirus 1
10. Drug eruption.

**FELINE DERMATOPHYTOSIS**
Variably pruritic. Focal areas of alopecia, variable degrees of inflammation, scaling and crusting.

Therapeutic considerations:
1. In most healthy animals, spontaneous resolution of dermatophytosis is often noted within 10 – 12 weeks.
2. Topical Therapy
   a. Spot treatment not recommended – total body rinses best
   b. Lime sulfur (LymDip, DVM) – 2% dip; twice weekly. Best readily available topical rinse for treatment available in US. In cats, licking the wet lime sulfur may cause oral irritation or GI upsets; best to use an E collar on the cat until the lime sulfur is dry.
   c. Enilconazole (Imaveraol, Janssen) – probably best topical product for total body rinse but not available in US as an approved drug for this purpose. Some Clinicians use the agricultural product (Clinafarm, EC) diluted to a concentration of 0.2% enilconazole, twice weekly for 6-8 weeks. Use with caution in cats in that toxicity has been reported (rarely, death); likely related to ingestion by grooming after the rinse. In one study of 22 cats treated with the topical, 6 developed elevated ALT’s, and one developed transient muscle weakness. It is suggested that, if used, liver enzymes be monitored and an “E” collar be placed on the cat to prevent licking until the rinse is very dry.
   d. Shampoo – chorhexidine and miconazole (Malaseb, DVM) or ketoconazole (KetoChlor, Virbac) – bathing twice weekly has been shown to result in progression to a fungal culture negative status faster than with systemic treatment alone (in this study, the miconazole shampoo was used and the systemic therapy was griseofulvin)
   e. Clipping – especially if multicat household to reduce contagion or to facilitate topical therapy. Try not to clip too close in that microtrauma to the skin may result in a spreading of the infection.
3. Systemic Therapy
a. Griseofulvin – good for dermatophytes in both dogs and cats (esp. in dogs where there is no concern for myelotoxicity); should not be used in individuals less than 6 weeks of age. In cats, an idiosyncratic bone marrow suppression may be seen (not dependent on dosage). This myelosuppression is more likely to occur in FIV and FeLV infected cats. Bone marrow suppression is usually heralded by leucopenia. It is recommended that (ideally) CBC’s be monitored monthly in cats while using this drug. All cats should be checked for FeLV, FIV before treatment. Dosage in cats – microsize (Fulvicin U/F) – 25 mg/kg/day once daily or divided into 2 doses. Ultramicrosize in PEG (GrisPEG) – 5 – 10 mg/kg/day. In dogs – microsize 50 – 100 mg/kg/day; ultramicrosize – 10 – 30 mg/kg/day.
b. Ketoconazole – 10 mg/kg/day in cats and 10 – 20 mg/kg/day in dogs. Ketoconazole has been associated with a high incidence of hepatotoxicity in cats (approx. 25% of treated cats?), and a lower incidence in the dog. Some strains of microsporum canis have also been noted to be resistant to ketoconazole. Consider monitoring liver enzymes once monthly when using the drug at high dosages.
c. Itraconazole (Sporonox) – 5 – 10 mg/kg/day, once per day. Administer one week on, one week off. In one study, hair concentrations increased significantly after each week of therapy, with concentrations in hair and skin exceeding the MIC for dermatophytes within the first few days of treatment and remaining above that MIC for at least 2 weeks beyond termination of treatment. It is uncommon to see deleterious side effects from this drug. A dose related vasculitis has been seen in dogs given high dosages over long periods of time.
d. Fluconazole (Difucan) – likely not as good as itraconazole.
e. Terbinafine (Lamisil) – no particular advantage over itraconazole, but has worked in some cases of dermatophytosis that have failed to respond to itraconazole. Dose is 10 – 30 mg/kg/day. High dosages appear to be associated with better efficacy. Toxicity is low, although non-clinically evident increases in ALT have been noted in cats (consider monitoring liver enzymes monthly?).
f. Lufenuron – 80 – 100 mg/kg given once every 2 weeks. Usually used as an adjunctive therapy. Efficacy very, very controversial in that both successful and non-successful treatment results have been anecdotally reported. Controlled studies have shown that the drug is not apparently effective in resolving infections, no preventing the establishment of infections in the cat.

4. Environmental Control - Best is chlorine (laundry bleach) – final concentrations of 0.05 to 0.5% sodium hypochlorite. The enilconazole environmental spray (Clinifarm EC; used for treatment of aspergillosis in poultry operations) is said to be very effective but not approved for this use in this country.

5. Catteries:
   a. Toothbrush culture every cat. Isolate cats with obvious or suspected dermatophytes.
   b. Thoroughly clean environment every 4-6 weeks, discarding of as much rugging etc. as possible; making sure all surfaces have been washed (walls etc.).
   c. Consider using enilconazole “fog”
   d. Isolate culture free, lesion free cats, but still bathe twice weekly with the Malaseb, and use lime sulfur or enilconazole rinse every 5-7 days (or twice weekly)
   e. Culture positive, lesional individuals – clip, shampoo and lime sulfur or enilconazole tip twice weekly; systemic griseofulvin or itraconazole.
f. Culture every 2-4 weeks. Treat until all lesions have resolved and cultures have been negative for 3-4 times.

**FELINE ATOPY AND FOOD SENSITIVITY**

Atopy and food sensitivity represent the most common cause of pruritic skin disease restricted to just the head and neck area in the cat in the author’s practice (about 70% noted to be atopic, 30% food sensitive). Pruritus is often first directed at the temporal region (just in front of the ears). Other manifestations include:

1. Indolent ulcers (upper lip or lips; may be the only manifestation of allergy in a given individual)
2. Eosinophilic granuloma – chin area (“pouty” chin); oral cavity (hard and soft palate)
3. It is possible to see any of the manifestations of the eosinophilic granuloma complex (i.e. indolent ulcers, eosionophilic granuloma) in the same cat.
4. Pruritic lesions may predominate in the chin and perioral region as diffuse erythema and the accumulation of dark exudates, giving the impression of feline acne; however, when clipped, classic comedo formation is not noted.
5. May exacerbate true feline acne lesions (comedoes)
6. Secondary bacterial pyoderma occurs but is less common than in the dog.
7. Secondary Malassezia infections are noted, but are less common than in the dog. They tend to predominate in the facial (folds, lip margins, chin) and foot areas (interdigital).
8. Rhinitis, conjunctivitis
9. Asthma – signs may be subclinical

Work-up and therapies for atopy and food sensitivity have been covered in these proceedings under “The Pruritic Dog and Cat – A Therapeutic update”.

**FELINE ACNE**

"Feline acne" is a "grab bag" term used to describe what appear to be a multitude of skin diseases that affect the chin of the cat. "Classic" feline acne appears to involve follicle plugging in the chin and lip margin region. Severe accumulation of follicular debris may result in furunculosis and possible secondary bacterial infection. The cause of feline acne is not known. In that multiple cats in the same environment have been noted to develop feline acne at the same time, there has been some suggestion that an underlying viral etiology is possible. Important differential diagnoses include bacterial dermatitis, allergic dermatitis and malassezia dermatitis (usually secondary to allergies). Other differentials are listed at the beginning of this section.

Treatment (for both true feline acne and bacterial dermatitis of the chin) :

**Mild**
1. Mupirocin (Bactoderm) - BID for two weeks, then q 24 hrs for 2 weeks; if recurrent, try maintenance (e.g. every 48-72 hours) (current topical therapy of choice).
2. 1% topical clindamycin (Cleocin-T, Upjohn)
   2% topical erythromycin solution (Erymax - Herbert Labs)
   3% tetracycline ointment (Achromycin - Lederle Labs)
   Daily for 14 days. Then q48hr, then twice weekly for maintenance.
3. 0.75% metronidazole gel (MetroGel) twice daily until resolution - then maintenance as needed.
   Response within 2 weeks
4. Benzoyl peroxide shampoo as necessary. BP may be irritating. Alternative: sulfur/salicylic acid
shampoo
5. Alternative topicals: topical alcohol, topical salicylic acid (Stri-Dex Pads)

Severe:
1. Anesthesia. Express comedones and cysts (ideally lance to do a good job)
2. Systemic antibiotics - amoxicillin/clavulonic acid, clindamycin, cephalexin, enrofloxacin
   May require long term therapy (6-10 weeks).
3. Consider short course of systemic steroids - 1-2 mg/kg/day prednisolone for 10 - 14 days.
4. Benzoyl peroxide shampoos
5. When scarring and cyst formation, oral glucocorticoid (starting at 1 – 2 mg/kg of prednisolone per day)
   and/or topical Synotic (Fluocinolone, DMSO); if milder steroid desired, try 1% hydrocortisone

Others:
1. L-lysine for possible viral component (250 to 500 mg BID)
2. Topical Vitamin A, Retin-A 0.05% liquid (Ortho). Daily, then q48hr, then 2 times weekly. Can be
   very irritating.
3. Refractory cases: oral isotretinoin (Accutane - Roche) 2 mg/kg/day. Side effects: conjunctivitis,
   periocular crusting, anorexia, vomiting and diarrhea.

Herpesvirus 1 - Associated Dermatitis and Stomatitis

Facial Dermatitis and stomatitis has been associated with Herpesvirus 1 in domestic cats. In a
report of 10 cats (Hargis A. et al, 14th Proceedings of AAVC/ACVD, 1998), 5 cats had histories of
recurrent or persistent mild upper respiratory tract infection. Six cats had been treated systemically with
glucocorticoids or drugs with glucocorticid activity (Ovaban) prior to lesion development, suggesting
exacerbation of a latent viral infection.

Lesions are located on the nasal planum or haired skin of the face and are vesicular, ulcerated
and crusted. Histologically, there is ulceration, crusting, necrosis and a mixed dermal inflammatory
infiltrate including many eosinophils. Intranuclear inclusion bodies are present in surface and adnexal
epithelium. Due to the prominent eosinophilic inflammation and low numbers of inclusion bodies, the
cutaneous or oral lesions may be misinterpreted as allergic dermatitis or lesions of the eosinophilic
granuloma group of disorders. Lesions usually persist and do not respond to glucocorticoids. Treatment
options include L-lysine, 250 mg (1/2 tab) BID (make sure it is form without propylene glycol); response
usually seen within two to four weeks or Alpha interferon, 1.0 – 1.5 million units / m2 SubQ three times
weekly for 4-8 weeks.

Idiopathic Facial Dermatitis of Persian and Himalayan Cats

An idopathic facial dermatitis has been described by Bond et al (Veterinary Dermatology, 11:35-
41, 2000) affecting Persian cats. Age of onset was 4 months to 5 years (median 12 months). The first
abnormality noted was the presence of black material which matted the hairs of the periccular, perioral
and/or chin areas. Although pruritus was not reported early in the course of the disease, it did develop
and became moderate to severe. Affected skin became progressively inflamed. A bilateral erythematous
otitis externa with accumulation of black waxy material within the ear canals was common. Some cats
had secondary Malassezia infections. Biopsies showed marked acanthosis, hydropic degeneration of basal
cells, occasional dyskeratotic keratinocytes and a superficial dermal infiltrate comprised of eosinophils,
neutrophils, mast cells, macrophages and occasional melanophages. Sebaceous glands appeared enlarged.
Ectoparasites and allergies were ruled out. Response to glucocorticoids was variable and often poor. The
cause of the disorder is unknown although a genetic basis is possible. More recently, this disease has shown response to oral cyclosporine (5 mg/kg/day).

**Squamous Cell Carcinoma in-situ (Bowen’s Disease)**

Single or multiple crusted papules or plaques with variable degrees of erosion, most commonly noted over the temporal regions of the head, but may be multicentric, involving the neck or trunk or extremities. Lesions are often on pigmented skin and are not related to UV light exposure. Affected cats are older and the course of the lesions is chronic. Lesions may be associated with Demodex cati or papilloma virus (seen on immunohistochemistry). Cats may be concurrently immunocompromised (FeLV, FIV). Diagnosis is by biopsy. Therapies include surgical removal, laser removal, radiation, cryosurgery or the use of oral retinoids (e.g. Accutane). Topical 5% imiquimod cream (Aldara, 3M Pharmaceuticals) has been used with some success. This very expensive therapy has been associated with irritant reactions and the development of leukopenia (following ingestion of the drug).

**INDOLENT ULCER**

May be caused by ectoparasite hypersensitivity (e.g. FBH, food sensitivity, atopy). On occasion, small lesions may be bacterial infections (or at least antibiotic responsive). Some, however, are idiopathic (have been seen in specific-pathogen free catteries; suspect genetic predisposition? Young cats that have this heritable tendency may grow out of it by 2-4 years of age?

**Diagnosis**
1. Response to trial therapies

**Treatment**
1. For very small lesions in young cats with no other evidence of allergic skin disease - benign neglect? May be the inherited, spontaneously resolving form?
2. Especially for smaller lesions, trial systemic antibiotic therapy (Clavamox, cephalosporin, clindamycin).
3. Aggressive corticosteroid therapy - early in course of disease produces best response: Prednisolone - 1-2 mg/kg BID until lesions in complete remission (2-4 weeks) OR methylprednisolone acetate (Depo-Medrol) 20 mg/cat every two to three weeks until problem in remission OR (especially for refractory cases), oral dexamethasone (0.2-0.5 mg/kg/day)
4. Corticosteroid responsive but recurrent:
   a. trial hypoallergenic diet - 6-8 weeks
   b. skin test and hyposensitize if positives
   c. Fatty acid and antihistamine therapy - symptomatic treatment for atopy
   d. long term, low, infrequent dose of steroid - e.g. 5-10 mg pred. qod or 20 mg methylprednisolone acetate as necessary (ideally no more frequently than every 6-8 weeks)
5. Poorly responsive to glucocorticoids
   a. Trial hypoallergenic diet
   b. chlorambucil and glucocorticoid - usually respond within 6 weeks
   c. gold salts
   d. intralesional steroids - variably effective
e. cryosurgery or radiation therapy - variably effective
f. levamisole - 5 mg/kg PO three times weekly - 60% responders
g. Consider combinations of progestational compounds and steroids
h. alpha interferon - 60 - 120 units/day PO - see "interferon" section for method of preparation. Others have used 1 million units/m² body surface area SubQ, three times per week.
i. cyclosporine - 5 mg/kg/day PO to initiate therapy
j. May be a genetic predisposition in some idiopathic cases. Cats who develop lesions at less than 2 years of age may have spontaneous remissions.