Pemphigus foliaceus (PF) is the most common autoimmune disease seen in the cat. The age of onset for feline PF ranges from less than 1 year to 17 years respectively (medians: 5 years). Although most cases are idiopathic, drug induced/triggered PF has been seen. Drug related pemphigus has been most commonly associationed with antibiotic administration (amoxicillin, ampicillin, sulfonamides). Others drugs incriminated include methimaole, itraconazole, lime sulfur dips and methimazole. Lesions may wax and wane. Seasonal variation has been noted. Although the disease is pustular in nature, pustules are only rarely encountered on a clinical basis. They are most characteristically seen over the medial pinna or around the nipples. Lesions are characterized by focal areas of crusting, inflammation and alopecia. Pruritus is seen in 80% of cases and may be mild to severe. Initial and most commonly affected areas (in decreasing order of incidence) are the head/face, ears, paws, nail folds, dorsum and ventrum, legs, chin and tail. The majority (80%) of individuals will have symmetric lesions. Systemic signs are seen in about 50% of cases and include lethargy, pyrexia, anorexia, weight loss and lymphadenopathy. Biopsies show evidence of acantholysis in the majority of cases. Mast cells and eosinophils are a common part of the dermal inflammatory response (at times resulting in the problem being misdiagnosed as allergic disease). Glucocorticoid monotherapy is usually effective for achieving clinical remission. Prednisolone (starting at 2-3 mg/kg BID for two weeks, then tapering) or triamcinolone(starting at 0.5 - 0.75 mg/kg/day)or dexamethasone (starting at 0.3 - 0.6 mg/kg/day) are the historically favored glucocorticoids. In one study, a higher incidence of remission with lesser overall side effects was associated with triamcinolone versus prednisolone therapy. Historically, chlorambucil has been most commonly used as a steroid sparing drug for the treatment of PF in cats (starting at 0.1 - 0.2 mg/kg/day). In one study, 9 of 11 (82%) of PF cats responded to the combination of chlorambucil and glucocorticoid. More recently, PF has been noted to respond well to oral cyclosporine (starting at 5 - 7 mg/kg/day; able to reduce to every other day therapy in 60 - 70%). In a recent abstract looking at 9 cats treated with cyclosporine and glucocorticoids and 9 with chlorambucil and glucocorticoids, there was no difference in disease response. 6/8 on cyclosporine could be weaned off all glucocorticoids, while only 1/7 cats on chlorambucil could be weaned off glucocorticoids. The author has managed particularly refractory cases with a combination of daily cyclosporine, every other day
chlorambucil and every other day prednisolone. PF has also also been noted to respond to gold salt therapy (aurothioglucose; starting at 1-2 mg/kg/week IM). After long periods (several months to years) of good control on any of the above medical therapies, a small percentage (10 - 15%) may eventually be able to discontinue all therapies.

ALOPECIA UNASSOCIATED WITH SELF TRAUMA

Most widespread alopecia problems seen in the cat are due to self trauma (e.g. atopy, food sensitivity, FBH, psychogenic etc.). The differential diagnosis list for alopecias unassociated with self trauma includes dermatophytosis, demodicosis, hyperadrenocorticism (spontaneous or iatrogenic), paraneoplastic alopecia, hypothyroidism, severe, debilitating diseases (e.g. uncontrolled diabetes mellitus, end stage renal disease, severe liver disease), drug eruption, telogen defluxion/anagen defluxion, alopecia areata, epitheliotropic lymphoma, alopecia mucinosa (follicular mucinosis), Pseudopelade, degenerative mucinotic mural folliculitis, sex hormone imbalance (due to adrenal neoplasia), and trichorrhexis nodosa (trauma +/- inherent weakness of hair).

Feline Hyperadrenocorticism

The predominant feline cutaneous changes associated with spontaneous hyperadrenocorticism include (25 cases; listed in order of decreasing incidence) truncal, flank and/or ventral partial to complete alopecia without erythema, atrophic skin with prominent vasculature, increased fragility (sometimes extreme) of the skin (may at times be noted when alopecia is minor), increased incidence of bruising of the skin, recurrent cutaneous abscessation, comedones, and hyperpigmentation. The most common systemic clinical signs associated with feline hyperadrenocorticismism are polydipsia, polyuria, polyphagia, lethargy, abdominal enlargement or “pot-belly”, panting, obesity and muscle weakness. Diabetes mellitus is common. Serum chemistry abnormalities include increased serum activities of alkaline phosphatase (although less common and much less severe as compared to the dog), ALT, hypercholesterolemia, hyperglycemia and hypokalemia (especially if concurrently diabetic). The reader is referred to other sources for a discussion of confirmatory diagnostics and therapies.

Paraneoplastic Alopecia and Internal Malignancies

Cats with pancreatic adenocarcinoma or bile duct carcinomas may present with a symmetric alopecia involving the ventrum and extremities. Hairs are usually readily epilated. The footpads, footpad/skin junctions may be dry and scaly/crusty and occasionally fissured and painful. The alopecic skin is often smooth and “glistening” and occasionally mildly scaly and erythematous. Pruritus is usually absent. All cats manifest varying degrees of weight loss, inappetence and lethargy. Diagnosis is by skin biopsy (severe follicular and adnexal atrophy with follicular miniaturization and mild perivascular inflammation) and workup to document visceral neoplasia. The prognosis is grave.

Demodex

Demodicosis associated with Demodex cati (follicular mite) primarily affects the head/face and distal limbs of cats. Generalized disease is less commonly encountered. Pruritus is variable, but can be significant and may mimic allergic disease. Diagnosis is by skin scraping / hair plucking. Affected individuals commonly have an underlying immunocompromising disease predisposing to the problem (i.e. diabetes mellitus, spontaneous or iatrogenic hyperadrenocorticism, FeLV, FIV, neoplasia), but a significant number of patients have no apparent underlying disease.

Demodex gatoi (short demodex mite) inhabits the stratum corneum. Demodicosis related to this mite
Infestation is usually pruritic. This mite infestation is communicable amongst cats. A common clinical presentation is an alopecia due to self trauma (hair loss with skin quite normal; most common over the lateral thorax, ventral and lateral abdomen, medial aspects of four limbs). Other individuals may have more obvious dermatitis in these areas or a miliary dermatitis presentation. Affected cats are usually only moderately or poorly responsive to glucocorticoid therapy. Diagnosis is by broad, superficial skin scraping and/or fecal flotation (for ingested mites). It may be difficult to find mites in affected individuals. A presumptive diagnosis may have to be made by assessing response to therapy.

Both Demodex cati or Demodex gatoi have been most responsive to 2% lime sulfur dips administered once weekly for 6 weeks. The haircoat should be soaked in lime sulfur for a minimum of 5 minutes, then allowed to air dry. An E-collar should be worn until the solution has dried to prevent ingestion and subsequent gastrointestinal irritation. Improvement is often seen within 3 weeks. Demodex cati may respond to ivermectin (0.3 mg/kg PO once daily or once every other day). There is potential for toxicity, either related to the drug or the vehicle used with the drug (i.e the propylene glycol present in some products may case a Heinz body anemia). Demodex cati may also respond to subQ doramectin (600 mcg/kg once weekly), or daily oral milbemycine oxime (1-2 mg/kg for demodex cati). Demodex gatoi usually does not respond to macrocyclic lactones. However, there have been anecdotal reports that Advantage Multi (imidicloprid, moxidectin), used every 2 weeks, may be effective.

**PLASMA CELL PODODERMATITIS**

Most cases are thought to represent an immune mediated disease. There may be some seasonal waxing and waning. In one European study, 50% of cases were FIV positive. We do not see this association in the USA. There has been a recent suggestion that bartonella may play a role. The earliest change seen is usually the development of a white, scaly, cross hatch pattern and swelling of the pads. Swelling may be dramatic. With chronicity, the swollen pad may ‘deflate’ (appearing almost like a deflated balloon). Occasionally, the pads may go on to erode or ulcerate. There may be significant hemorrhage. These changes vary from being asymptomatic to involving significant pain and lameness. Systemic signs are also variable (pyrexia, lethargy, anorexia, peripheral lymphadenopathy). General laboratory screening usually reveals a hyperglobulinemia. Pad biopsies show a perivascular accumulation of plasma cells, with lesser numbers of lymphocytes and neutrophils. Leukocytoclastic vasculitis has been described. Early lesions may involve significant infiltration with eosinophils. Some cases will spontaneously resolve with no therapy. The initial therapy of choice for the author is doxycycline (5 - 10 mg/kg BID). Treated patients may be eventually weaned off therapy. Therapies for ‘doxycycline failures’ include glucocorticoids (prednisolone, triamcinolone or dexamethasone; see Pemphigus section for dosages), oral cyclosporine (5.0 - 7.5 mg/kg/day), glucocorticoids and golds salts, glucocorticoids and chlorambucil or surgical excision (usually to ameliorate hemorrhage problems). Again, successfully treated patients may be able to eventually weaned off all medications.

**CUTANEOUS HORN OF THE PAWPAD**

Cutaneous horns may arise from viral papilloms, actinic keratoses, Bowenoid in situ carcinoma or squamous cell carcinomas. Felv associated pawpad horns occur in the centers of digital, centaql or metacarpal/metatarsal pawpads whereas non FeLV associated horns tend to grow on the digital pads, just below the nails. The lesions are histopathologically distinctive.