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Chapter 21

Dermatoses of Pet Rodents, Rabbits, and Ferrets

Owner complaints relative to skin problems in pet rodents, rabbits, and ferrets are commonly encountered in small animal practice.[3–6, 10–12, 16–20] Although much has been written about the dermatoses of the commonly kept exotic mammals, the material is often anecdotal or difficult to interpret. In particular, therapeutics in exotic animals is controversial because few of the drugs commonly used are approved for use in these species. In addition, because most treatments have evolved through a hit-and-miss approach, there are great discrepancies in the literature concerning what drugs to use and at what dose to administer them. Over 300 references on these small "exotic" mammals were listed in the fifth edition of this text.[17]

Special caution is indicated with the use of antibiotics, because death due to direct toxic effects or alteration of the normal bacterial flora is common[2–6, 7a, 9, 14, 15] Especially in the guinea pig and rabbit, antibiotics that specifically affect gram-positive organisms are contraindicated, and broad-spectrum antibiotics should be used. In rabbits and rodents, it is important to use "four-quadrant" antibiotic coverage, drugs that are effective against aerobic, anaerobic, gram-positive, and gram-negative bacteria.[10, 15] The safest drugs are the fluoroquinolones, trimethoprim-sulfonamides, chloramphenicol, aminoglycosides, and metronidazole.[7a, 10, 15]

One drug that is commonly used in these species and that appears to be safe and effective in all ages is ivermectin.[5, 10, 17] The standard dose is 300 to 400 μg/kg given subcutaneously and repeated every 2 weeks until the disorder being treated is cured. It has also been reported that ivermectin is effective topically (one or two drops applied between the scapulae).[47a, 71]

Special precautions must also be observed when using topical medications. First, these animals routinely remove topical agents through their grooming behaviors. Second, these small creatures are prone to hypothermia after shampooing and dipping. Thus, aqueous solutions should be kept at body temperature, and the animals must be kept warm and dry and away from drafts after treatment. Avoid alcohol-based sprays. In addition, inhalation/aspiration pneumonia is a potential problem. Thus, the face is best avoided when dips are administered.

The structure and function of small exotic mammal skin are similar to those described in Chapter 1.[17] Hair growth in rodents (not guinea pigs) and rabbits occurs in periodic orderly waves originating on the ventrum between the front legs and spreading dorsally and caudally. In some rabbits, thickened patches of skin can be associated with the variation in hair growth cycles. This is more common in young rabbits and becomes less obvious with increasing age. The mean thickness in these patches is 2 mm as compared with 1 mm in normal skin. Histologic examination of the patches reveals increased size of hair follicles and increased vascularity. Notable histologic differences include (1) the absence of epitrichial sweat glands in rodents and (2) the structure of the tail epithelium of the mouse and rat, which features orthokeratosis with a stratum granulosum at the follicular osia and parakeratosis without a stratum granulosum in the interfollicular areas.

Hamsters have large glands on either flank, which are visible as dark brown patches that are more prominent in male animals. These flank or hip glands are sebaceous and are used for marking territory. In sexually aroused males, the haircoat over the glands becomes matted from secretions and readily visible, and the animals scratch the areas as if

they are pruritic. These findings may be interpreted as signs of skin disease by some owners. Gerbils have a yellowish tan, midventral scent gland, which is also sebaceous, more prominent in male animals, and occasionally mistaken as a cutaneous abnormality. Rabbits have a mental or chin gland (which is sebaceous in nature) with which they mark their territory. Normal ferrets typically have visible accumulations of brownish cerumen around the external auditory meatus. In addition, normal ferrets can have several comedones on the tail.

Most rodents are burrowing animals that spend most of their time in the wild seeking food and escaping predators. When they are placed in sterile environments with ad libitum feeding and no danger of predators, they are left with little to do except to chew on themselves or on others. In addition, male rodents tend to be territorial and aggressive. Self-inflicted trauma or that inflicted on cagemates can be triggered or amplified by crowding.

Some other normal behaviors may be misinterpreted as pruritus. A rabbit rubbing its mental, or chin, gland on a cage or furniture is only marking its territory. Likewise, a guinea pig scooting or dragging its perineal area on the ground is usually scent-marking, although in some male animals, the glandular secretions can become impacted and cause irritation. Male hamsters may clean and fuss with their flank glands.

Staphylococci, especially *Staphylococcus aureus*, are frequently isolated from the skin, the ears, the nostrils, and the haircoat of rodents and rabbits.[8, 11, 17] Not surprisingly, *S. aureus* is a common opportunist and cause of skin infections in these species.

Finally, these small creatures, especially mice, rats, guinea pigs, and rabbits, are frequently used for studying models of human diseases (e.g., hereditary hypotrichoses and ichthyoses in mice and rats), for examining the pathogenesis of various dermatoses also seen in humans (e.g., contact hypersensitivity and candidiasis in guinea pigs), for evaluating therapeutic agents used in various human dermatoses (e.g., treatment of *Malassezia* dermatitis in guinea pigs and the use of retinoids in rhino mice), for studying percutaneous absorption and various aspects of dermatopharmacology (e.g., the mouse tail assay for studying epidermal drug effects), and for screening the potential irritancy or sensitization of topical agents (e.g., the guinea pig Draize test for contact allergens and the rabbit skin test for topical irritants).[16]

• CHINCHILLA

In the wild, chinchillas keep their haircoats clean and healthy by bathing in fine volcanic dust.[17] A similar dust is commercially available (chinchilla dust, or Fuller's earth) and should be provided daily.[3, 10, 17, 21] The dust is poured 5 to 10 cm deep in a metal pan and left in the animal's cage for about 30 minutes. Chinchillas deprived of their dust baths are prone to abnormalities of the haircoat and skin. Chinchillas kept in a warm (warmer than 80°F) humid environment develop matted fur.

Fungal Infections

Dermatophytosis is uncommon in chinchillas.[3, 4, 17, 22] *Trichophyton mentagrophytes* is the most frequent cause, but *Microsporum gypseum* and *M. canis* are occasionally isolated. Lesions are most common around the eyes, the nares, and the mouth, but may occur anywhere. Circumscribed areas of alopecia, broken hairs, and variable degrees of scaling, erythema, and crusting are seen. Secondary staphylococcal infection can occur and usually presents as cellulitis or abscess.[22]

Recommended therapy includes griseofulvin by mouth (50 mg/kg q24h) for 30 days.[17, 22] It had previously been recommended to add captan powder to a clean dust bath once daily.[22] However, because captan has been shown to be ineffective against *M. canis* (see Chap. 5), the authors cannot recommend it.

Fur Chewing

Chinchillas may chew, pull out, and eat their fur during times of stress (gestation, travel, and shows), in the absence of dust baths, and for unknown reasons.[3, 10, 17, 21, 22] When the fur chewing is idiopathic, it rarely ceases. It is believed that the idiopathic condition is a heritable trait, that no therapy is effective, and that the condition is best controlled by culling and selective breeding.[10, 17, 21] Providing fresh alfalfa hay, giving proper chinchilla pellets, and reducing stress may help.

Fur-Slip

Fur-slip is a normal physiologic process whereby chinchillas appear to "squirt" or "shoot" some of their fur out.[17, 22] This is a mechanism of self-defense in which the chinchilla hopes to leave a predator with a mouthful of fur as it escapes. Fur-slip is likely to occur when chinchillas are frightened or handled roughly. Chinchilla fur grows in tufts with up to 90 fibers per follicle and up to 1000 follicles/cm.[2, 22] Fur-slip affects spots 2 to 5 cm in diameter. Fur regrowth occurs within 3 months for some follicles and usually takes 5 months for an entire spot. However, regrowth is rarely a perfect fit and always looks patched.

Nutritional Disorders

Fatty acid deficiency results in generalized scaling, poor haircoat, reduced hair growth, and perhaps cutaneous ulcers in the chinchilla.[2, 21]

Zinc deficiency may produce alopecia.[21]

• FERRET

Ferrets have active sebaceous glands that contribute to their distinctive odor and somewhat greasy feeling coat. Occasional small, red-brown waxy deposits can be found on normal skin. During breeding season, intact males have increased sebaceous secretions, to the point of having a yellowish discoloration of the undercoat, a very oily fur, and quite a musky odor. Frequent bathing may strip essential oils from the skin, resulting in keratinization disorders and pruritus.[10] Owners should be encouraged to use mild shampoo no more frequently than once monthly, if possible.

Bacterial Infections

Bacterial skin infections are uncommon in the ferret and are usually caused by *S. aureus* or *Streptococcus* spp.* Infections are usually secondary to bite wounds (especially on the neck of female ferrets during breeding season, perpetrated by aggressive male animals as a prelude to coitus) or the pruritus associated with ectoparasites. There may be superficial and follicular (Fig. 21–1) lesions or deep abscesses and fistulae. Staphylococcal or streptococcal cellulitis of the neck may be associated with dental disease and mandibular osteomyelitis.[28] Diagnosis is based on cytologic examination. Treatment consists of a regimen of various combinations of topical antimicrobials (3% hydrogen peroxide or 0.5% to 1% chlorhexidine), surgical drainage, and the administration of systemic antibiotics (Table 21–1).

Actinomycosis ("lumpy jaw") is rarely reported in ferrets.[17, 28] Affected animals have nodules or abscesses in the neck, and fistulae and discharge of thick green-yellow pus may be seen.

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*See references 4, 6, 15, 17, 24, 25, 30.

FIGURE 21–1. Superficial bacterial folliculitis in a ferret. Multiple crusted papules and patchy hair loss over dorsal midline.

Fungal Infections

Dermatophytosis appears to be rare in ferrets.* *M. canis* and *T. mentagrophytes* are the most common causes, and young animals are most frequently affected. Lesions consist of annular areas of alopecia, broken hairs, scale, and varying degrees of erythema and crusting. Pruritus is usually absent. Diagnosis is confirmed by microscopic examination of affected hairs and fungal culture. Therapy consists of topical application of antifungal agents and environmental clean-up as described for cats. Griseofulvin is usually not needed. Dermatophytosis in ferrets is a potential zoonosis. *Blastomycosis* was diagnosed in a ferret with chronic cutaneous plaques and ulcers.[17] *Histoplasmosis* was diagnosed in a

*See references 3, 6, 10, 17, 24, 25, 28, 35.

● Table 21–1 **COMMON THERAPEUTIC AGENTS IN SMALL MAMMALS**

AGENT	PROTOCOL
Antibiotics	
Amoxicillin	10–20 mg/kg SC or PO q12h (ferret, mouse, rat)
Ampicillin	5–10 mg/kg SC or PO q12h (ferret, mouse, rat)
Cephalexin	15–25 mg/kg PO q12h
Chloramphenicol succinate	50 mg/kg IM or SC q12h
Chloramphenicol palmitate	50 mg/kg PO q12h
Ciprofloxacin	5–15 mg/kg PO q12h
Doxycycline	5–10 mg/kg PO or SC q24h
Enrofloxacin	5–20 mg/kg PO or SC q12h
Gentamicin	5 mg/kg IM or SC q24h
Metronidazole	20 mg/kg PO q12–24h
Sulfadiazine-trimethoprim	15–30 mg/kg SC q24h
Sulfamethoxazole-trimethoprim	15–30 mg/kg PO q12h
Tetracycline hydrochloride	20 mg/kg SC or PO q12h
Antifungals	
Griseofulvin	25–50 mg/kg PO q24h
Antiparasitics	
Amitraz 250 ppm	Total body dip q2wk
Ivermectin	0.3–0.4 mg/kg SC q2wk
Lime sulfur 2%	Total body dip q7d

IM, intramuscularly; PO, orally; SC, subcutaneously.

ferret with multiple subcutaneous nodules.[24] *Coccidioidomycosis* was diagnosed in a ferret with a persistent draining tract of the stifle.[27]

An outbreak of otitis externa and pinnal necrosis in association with mites (*Otodectes*?) and yeast (*Malassezia*?) infection was reported.[26] A painful, rapidly progressing crusting and necrosis of the pinnae spread, if untreated, onto the face. Mites and yeast were found in smears. Histopathologic findings included suppurative epidermitis, numerous surface yeast, and hemorrhagic necrosis with thrombosis. Treatment with ketoconazole (50 mg/ferret, q24h, per os) and a polypharmaceutical otic preparation was rapidly effective.

Ectoparasites

Otodectic mange (ear mites) is common in ferrets.[10, 17, 38] Affected ferrets may manifest no clinical signs or variable degrees of excessive cerumen production. Pruritus, inflammation, and secondary bacterial infection are uncommon. Diagnosis is confirmed by finding *Otodectes cynotis* in ear swabs. Treatment is accomplished with topical acaricides or topical ivermectin (500 μg/kg divided between the two ears).[10] Injectable ivermectin is the treatment of choice. In one study,[35a] topical treatment with either Tresaderm (2 drops in ears q24h for 7 days, sequence repeated after a week of no treatment) or ivermectin (Ivomec diluted 1:10 in propylene glycol; 400 μg/kg divided equally in both ears, repeated in 2 weeks) was more effective than the subcutaneous administration of ivermectin (400 μg/kg, repeated in 2 weeks). Ivermectin should be used with caution in pregnant jills.[17, 25, 28] When ivermectin was administered at 2 to 4 weeks of gestation, an increased incidence of congenital defects, such as cleft palates, was seen. However, when ivermectin was administered after 4 weeks of gestation, no problems were noted.

Fleas (especially *Ctenocephalides felis felis*) are commonly found on ferrets.[17] Animals may be asymptomatic or have cutaneous reaction patterns similar to flea bite hypersensitivity in cats. Ferrets manifesting presumed flea bite hypersensitivity have a pruritic papulocrustous dermatitis over the rump, ventral abdomen, and caudomedial thighs, or a self-induced, symmetric alopecia over the rump, flanks, ventral abdomen, or medial thighs (fur-mowing) in which the skin appears normal. Treatment strategies must include the ferret; in-contact ferrets, cats, and dogs; and the environment, as described for cats. Fipronil spray has been found to be safe and effective for ferrets.[31]

Sarcoptic mange is uncommon in ferrets and has two clinical presentations: (1) intense pruritus and dermatitis over the face, the pinnae (Fig. 21–2*A*), and the ventrum and (2) pruritic pododermatitis.[10, 17] In the pododermatitis form, the feet are swollen, erythematous, and crusted, and the claws may be dystrophic. Affected ferrets may actually slough claws or digits. Diagnosis is confirmed by finding *Sarcoptes scabiei* mites in skin scrapings. However, mites can be extremely difficult to find, so response to miticidal therapy is often used as a diagnostic procedure. Treatment includes 2% lime sulfur dips (weekly until 2 weeks after clinical cure) or ivermectin injections.

Demodicosis was reported in two unrelated ferrets that were living in the same household and receiving long-term treatment with a glucocorticoid-containing otic ointment.[34] The ferrets exhibited excessive cerumen in the ears and pruritus, alopecia, comedones and orange discoloration of the skin behind the ears and on the ventrum. Skin scrapings and ear smears revealed numerous short mites that resembled *Demodex gatoi*. Amitraz dips were curative, and no side effects were reported.

Ferrets housed outdoors may occasionally have cysts, abscesses, or fistulae in the neck associated with infestation by *Hypoderma* sp. or *Cuterebra* sp. larvae.[10, 17] Treatment includes careful surgical removal of the larva and routine wound care.

Flystrike (especially *Wohlfahrtia vigil*) can be a problem for commercial ferret ranchers and outdoor ferrets.[10] Eggs are commonly laid on the face, neck, and flanks of young ferrets, causing irritation and subcutaneous abscesses.

Ticks may occasionally be found on ferrets, especially around the head and the ears.[17] Treatment is as described for cats.

Ferrets can be experimentally infected with *Dracunculus insignis* and have been used as an animal model for studying dracunculiasis.[17] Lesions consist of tender swellings, which abscess and develop fistulae. Lesions occur most commonly on the legs.

FIGURE 21–2. *A,* Alopecia, crusts, and excoriations on the pinnae of a ferret with sarcoptic mange. *B,* Mast cell tumor on the lower jaw of a ferret. *C,* Sebaceous epithelioma on the shoulder of a ferret. *D,* Squamous cell carcinoma on the lip of a ferret. (Courtesy of W. Gould.) *E,* Alopecia, crusting, and ulceration of the nose of a gerbil with "sore nose." *F,* Squamous cell carcinoma of the ventral scent gland in a gerbil. (Courtesy of W. Gould.) *G,* Staphylococcal pododermatitis in a guinea pig. (Courtesy of J. King.) *H,* Alopecia and thick crusts over the dorsum in a guinea pig with trixacariasis. (Courtesy of E. Guaguère.)

Viral Infections

The ferret is susceptible to canine distemper virus.[3, 6, 10, 17] Typical cutaneous findings include an erythematous rash under the chin and in the inguinal region; swelling and brownish crusts on the chin, lips, nose, and the periocular area; and swollen, hyperkeratotic nose and footpads (Fig. 21–3). Some ferrets develop an orange-tinged dermatosis on the anus and inguinal region.

Endocrine Disorders

HYPERADRENOCORTICISM

Adrenocortical neoplasia and hyperplasia are the most common causes of progressive bilaterally symmetric alopecia in the ferret.[10, 37, 39a, 41, 43] Two authors indicated that this disorder accounted for about 25% of all the ferrets examined in their practice.[43] The condition was initially diagnosed as Cushing's syndrome. However, other classic clinical signs (polyuria, polydipsia, and polyphagia) and hematologic, biochemical, or urologic abnormalities associated with Cushing's syndrome are rarely present. In addition, basal plasma cortisol levels are usually within the normal range and adrenal function tests (adrenocorticotropic hormone [ACTH] stimulation, dexamethasone suppression) have not been useful in separating normal from diseased ferrets.[10, 17, 37, 39, 43] The contralateral

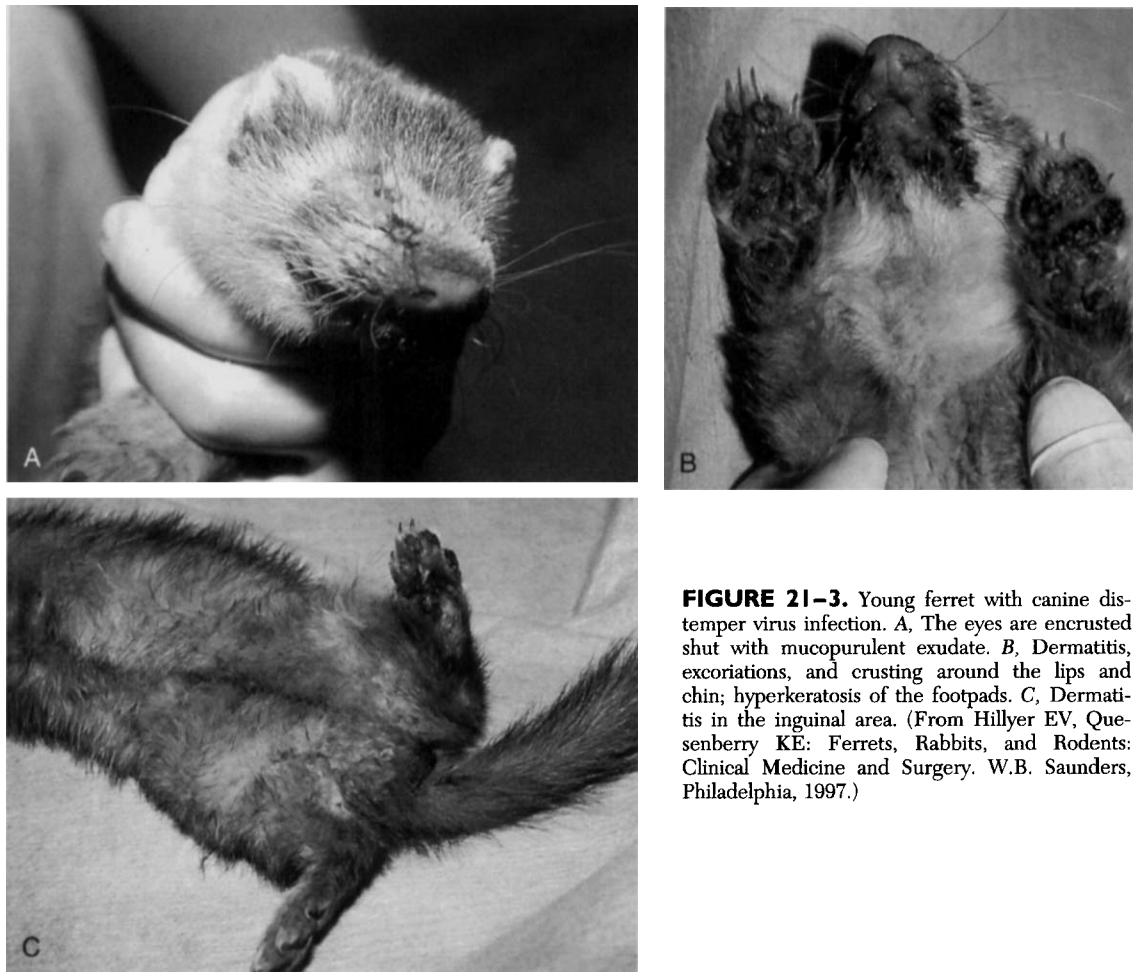

FIGURE 21–3. Young ferret with canine distemper virus infection. *A*, The eyes are encrusted shut with mucopurulent exudate. *B*, Dermatitis, excoriations, and crusting around the lips and chin; hyperkeratosis of the footpads. *C*, Dermatitis in the inguinal area. (From Hillyer EV, Quesenberry KE: Ferrets, Rabbits, and Rodents: Clinical Medicine and Surgery. W.B. Saunders, Philadelphia, 1997.)

adrenal gland is not atrophied. These considerations in concert with the common occurrence of vulvar enlargement in affected female ferrets and return to male sexual behavior in castrated male ferrets suggested that the endocrine abnormality was in the adrenocortical production of sex hormones.[41] Median plasma concentrations of 17-hydroxyprogesterone, androstenedione, and estradiol are significantly higher in ferrets with hyperadrenocorticism than in normal ferrets.[39] Ninety-six percent of affected ferrets had a high concentration of at least one of these three hormones, but only 22% had high concentrations of all three hormones. These concentrations decreased after adrenalectomy.

The etiology of hyperadrenocorticism in ferrets is unknown, but several hypotheses have been put forth.[10, 43] Early neutering may lead to metaplasia of undifferentiated gonadal cells in the adrenal capsule. In a Dutch study, a significant linear correlation was found between the age at neutering and the age at the time of diagnosis.[42a] Hyperadrenocorticism in ferrets is common in the United States, but apparently rare in the United Kingdom, suggesting that diet (prepared foods in the United States, whole prey in the United Kingdom), photoperiod (housed indoors in the United States, outdoors in the United Kingdom), and genetics (U.S. population more inbred) may also play a role.

Clinical signs are seen between 2 and 8 years (average 4) of age and with equal frequency in females and males. In most cases, clinical signs are first noted in the spring. The initial abnormality is usually a bilaterally symmetric, noninflammatory alopecia that usually begins on the tail and tail base (Fig. 21–4) and progresses to the ventral abdomen, caudomedial thighs, dorsal surface of hind paws, lumbosacral region, and shoulder blades. In some animals, the alopecia may come and go. In such cases, clinical signs appear in the spring and spontaneously regress in the autumn. However, clinical signs recur with increasing severity each spring and eventually persist.[10, 17, 43] Most affected males and females have a strong musky odor. Vulvar enlargement is common in female animals (Fig. 21–5), as is return to male sexual behavior in castrated males. In more chronic cases, the back of the neck, top of the head, and trunk become alopecic, and the body skin becomes thin and hypotonic (Fig. 21–6). About 9% to 30% of affected ferrets are reported to have varying degrees of pruritus, mostly over the dorsum between the shoulder blades.[10, 43, 43a] When present, pruritus is often severe and unresponsive to glucocorticoids and antihistamines. Numerous comedones are occasionally found on the tail, and phlebectasias may be seen. Lethargy, muscle atrophy, splenomegaly, and return to male behavior (in neutered males) occur in about 63%, 56%, 87%, and 15%, respectively, of the cases.[43] Mammary gland hyperplasia has been reported, and stump pyometras are not uncommon. Stranguria may be present in up to 19% of affected males.[43a] The enlarged adrenal gland can be palpated in about 30% of cases.

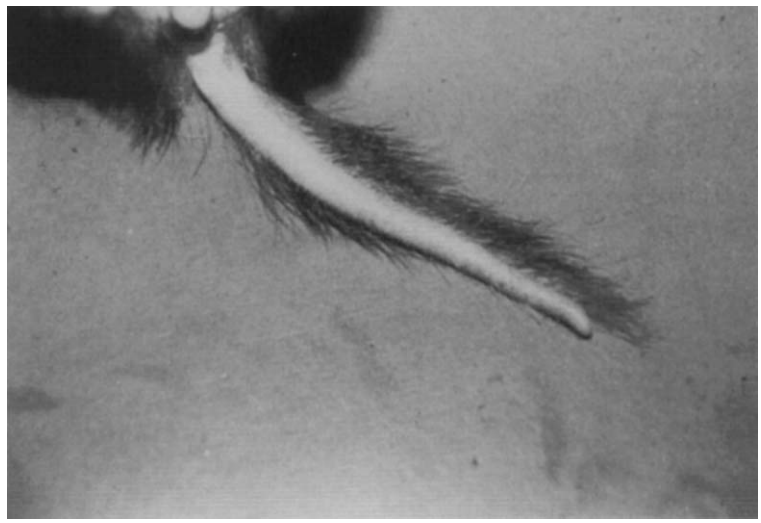

FIGURE 21–4. Alopecia of the tail in a ferret with an adrenal adenoma.

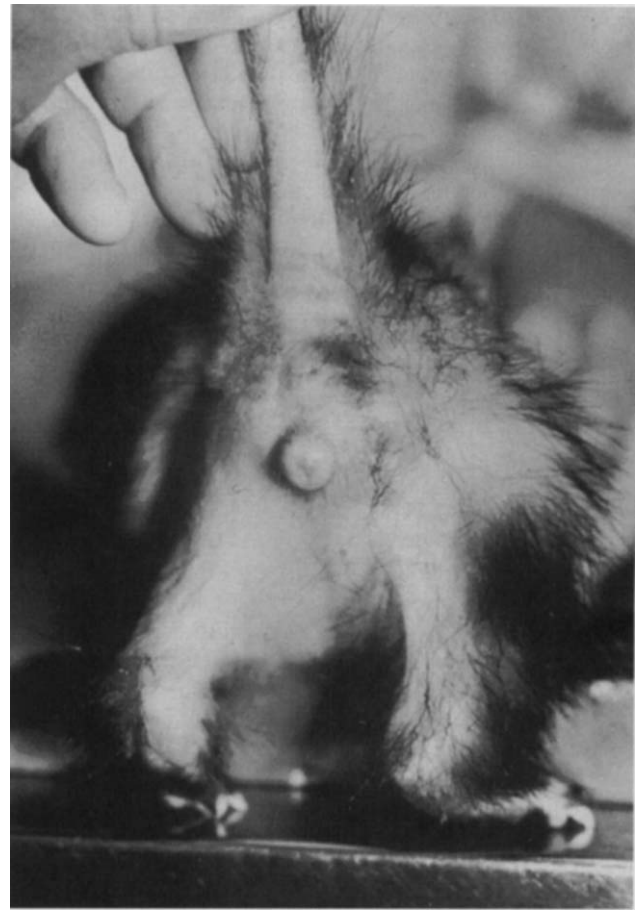

FIGURE 21–5. Symmetric alopecia and vulvar enlargement associated with an adrenal adenoma in a ferret. (Courtesy of W. Gould.)

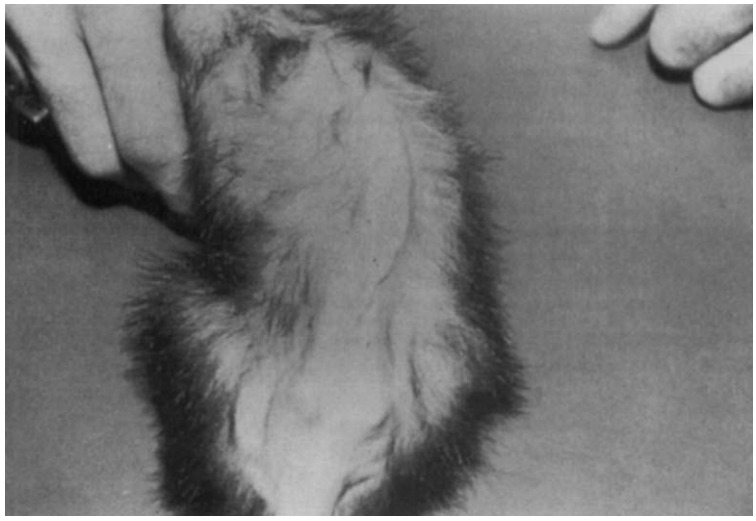

FIGURE 21–6. Truncal alopecia and hypotonic skin (note exaggerated wrinkling) in a ferret with an adrenal adenoma.

Diagnosis is usually confirmed by ultrasonographic examination or exploratory laparotomy.[10, 17, 23] Ultrasonography may fail to diagnose about 50% of the cases and may not be cost-effective in a practice situation.[43] In rare instances, the adrenal glands appear normal on both ultrasonographic examination and exploratory laparotomy.[42, 43] Urinary cortisol: creatinine ratios were significantly higher in ferrets with hyperadrenocorticism than in normal ferrets.[29] However, this is not specific for hyperadrenocorticism, and affected ferrets can have normal ratios.[42]

Eighty-four percent of affected ferrets have unilateral adrenal tumors, whereas 16% have bilateral adrenal tumors.[43] Of the unilateral tumors, over 80% are in the left adrenal gland.[17, 43, 43a] Histologically, 56% of the tumors are nodular hyperplasia, 27% are adenocarcinomas, 16% are adenomas, and 1% are "normal."[43] All tumors larger than 1 cm diameter were adenocarcinomas.[43] In 47% of the patients with bilateral tumors, each gland had a different histologic type of tumor.[43, 43a] All male ferrets with a return of male sexual behavior had adenocarcinomas.[43]

In one study,[43] 27% of the ferrets with hyperadrenocorticism had concurrent insulinomas. This emphasizes the importance of performing a complete exploratory.

The "adrenal panel" (Clinical Endocrinology Laboratory, Department of Comparative Medicine, University of Tennessee, Knoxville, TN, 615-974-5638) may give diagnostic results in 96% of the cases.[39] However, results are not always diagnostic, and the test may not be cost-effective in a practice situation.[43, 43a]

In most animals, the treatment of choice is unilateral adrenalectomy.[10, 17, 43] Clinical improvement is evident within 2 to 8 weeks, and complete recovery is usually seen within 5 months. Hair regrowth usually occurs in the opposite direction to which it was lost. In two cases in which a unilateral adrenalectomy was performed and no hair growth had occurred for 6 to 8 months, a secondary exploratory revealed tumors on the remaining adrenal.[43] Seventeen percent of the animals with unilateral tumors develop tumors on the remaining adrenal, resulting in a recurrence of clinical signs within 14 months.[43] Ferrets with bilateral adrenal tumors should have the largest adrenal gland completely removed, along with 50% to 60% of the other one (subtotal bilateral adrenalectomy).[43, 43a] Even following subtotal bilateral adrenalectomy, 15% of the ferrets had recurrence of clinical signs within 7 to 22 months.[43a]

Medical treatment has been used, usually unsuccessfully, when surgery could not be performed, or when, after surgical removal of a neoplastic adrenal, the remaining adrenal gland also became neoplastic and clinical signs recurred.[17] Mitotane (o,p'-DDD) has been used at 50 mg orally, once daily for 7 days, then every 3 days until clinical cure. Administration of the drug is then stopped. If clinical signs recur, o,p'-DDD is given at a weekly maintenance dose of 50 mg. To facilitate administration, the o,p'-DDD is mixed with corn starch and 50-mg doses are put in gelatin capsules. However, mitotane is rarely effective.[10, 43a] Ketoconazole has been ineffective when given orally at 15 mg/kg every 12 hours.[10, 43a]

Anecdotal reports indicate that leuprolide (Lupron, TAP Pharmaceuticals), when given at 100 μg/kg subcutaneously, every 21 days, is very effective for the treatment of hyperadrenocorticism in ferrets. Response is usually seen by the third injection and is complete by 6 months. Once maximum response is achieved, the interval between injections is extended. Ferrets with aggressive adrenal carcinomas may not respond.

HYPERESTROGENISM

Hyperestrogenism is well recognized in the female ferret.[10, 17, 24, 28] However, this disorder is rare because large-volume ferret breeders are neutering and descenting the animals at 6 weeks of age. An ovarian remnant may be suspected in a neutered female with signs of estrus. Jills allowed to remain in estrus during the breeding season are susceptible to the toxic effects of estrogen on bone marrow. Affected jills develop pancytopenia and varying degrees of alopecia. Clinical signs accompanying the pancytopenia include pale mucous membranes, petechial or ecchymotic hemorrhages, anorexia, depression, and weight loss.

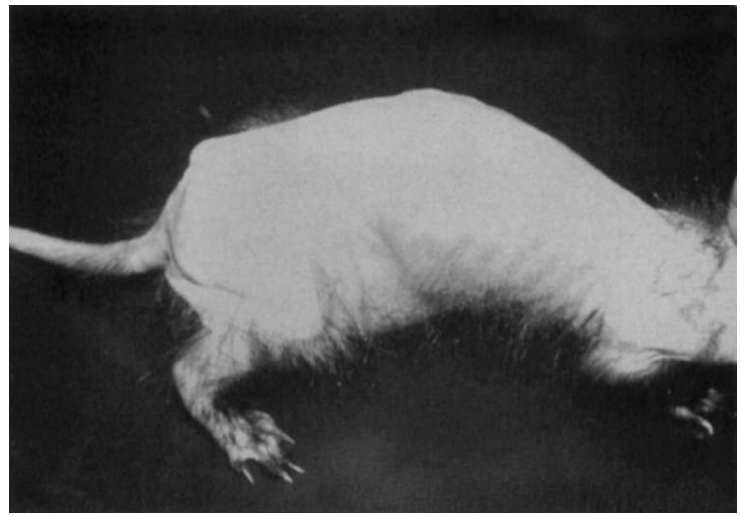

FIGURE 21–7. Alopecia of the trunk and tail in a jill with hyperestrogenism.

The alopecia is bilaterally symmetric, beginning on the tail, the perineum, the abdomen, the medial thighs, and the rump and progressing cranially (Fig. 21–7). Vulvar enlargement is a constant finding. Untreated animals die of infectious or hemorrhagic complications. Treatment is often unrewarding. Ovariohysterectomy; intravenous blood transfusions; administration of dexamethasone, anabolic steroids, and systemic antibiotics; and supportive care have rarely been reported to result in recovery, but transfusions may need to be repeated several times for 3 to 5 months.[10, 17, 24, 28]

ALOPECIA ASSOCIATED WITH TESTICULAR NEOPLASIA

Testicular neoplasia is rare, because most large-volume breeders neuter male ferrets at 6 weeks of age. A sparse haircoat and a bald tail were reported in association with an interstitial cell carcinoma of the testicle in one ferret.[17] Total body alopecia and pruritus were reported in association with a testicular Sertoli's cell neoplasm in another ferret.[17]

BREEDING SEASON ALOPECIA

Breeding season alopecia is commonly seen, especially in the female and less frequently in the male ferret, during the period of sexual activity from March through August.[6, 10, 17, 28, 41] Photoperiod plays an important role in this condition, because even neutered ferrets are affected. Bilaterally symmetric alopecia affects the tail, the perineum, the ventral abdomen, the rump, and occasionally, the periocular region and the paws. Affected ferrets are otherwise healthy, and spontaneous hair regrowth occurs in the fall.

SHEDDING

Seasonal shedding is seen in spring and early summer.[6, 17, 28, 41] Variable degrees of hypotrichosis or alopecia may be seen over the trunk and resolve spontaneously within a month or two.

TELOGEN DEFLUXION

Telogen defluxion is occasionally seen 2 to 3 months after a stressful circumstance (high fever, severe illness, surgery, and anesthesia).[17, 41] Bilaterally symmetric hypotrichosis or alopecia is most prominent on the trunk (Fig. 21–8).

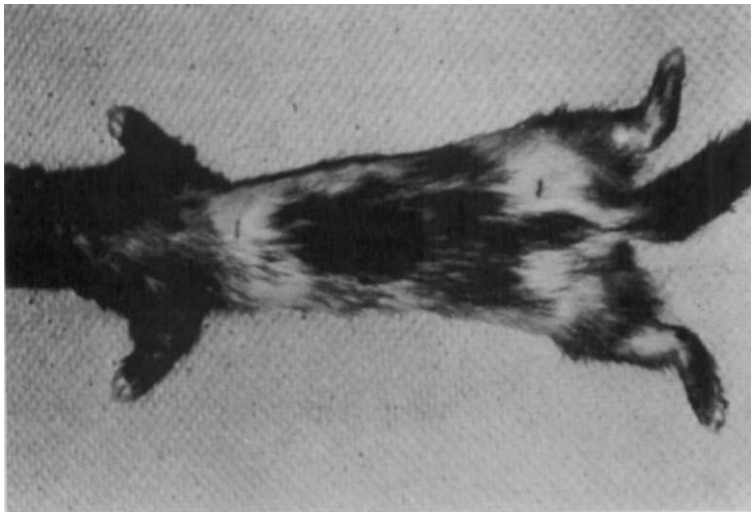

FIGURE 21–8. Telogen defluxion. Patchy alopecia of the dorsal trunk. (Courtesy of M. Paradis.)

HYPOTHYROIDISM

Although anecdotal reports suggest that hypothyroidism is a common cause of endocrine-like alopecia in ferrets,[4] the authors and others[10, 17] have never made such a diagnosis, and know of no documented cases. Some data have been published on basal serum thyroxine and triiodothyronine levels and thyroid function tests in normal ferrets.[17, 28]

Miscellaneous Conditions

Some authors believe that the most common cause of alopecia and dull, dry haircoat in ferrets is poor dietary practices.[3] Food passage averages 3 to 4 hours in ferrets; thus, diets high in protein and fat but low in fiber are important. A low-fat diet may result in a dry, dull haircoat.[10]

Biotin deficiency (from excessive feeding of raw eggs) can result in bilaterally symmetric alopecia in ferrets.[6, 24, 25, 28]

Severe intestinal parasitism (especially *Toxascaris leonina*) can produce variable degrees of hair loss and scaling in ferrets.[25]

Contact dermatitis can occur with frequent use of shampoos or insecticide sprays.[25]

Focal areas of alopecia have been seen at the site of previous injections.[25]

The authors have seen an occasional ferret with presumptive atopy. Affected animals manifested symmetric, nonlesional pruritus over the trunk, the rump (Fig. 21–9), and the paws. Fleas were not present, hypoallergenic diets were ineffective, and response to glucocorticoids or chlorpheniramine was good. One of the authors (WHM) has seen one food-hypersensitive ferret with the same clinical signs. The ferret was normal when fed a commercial hypoallergenic diet for cats (Innovative Veterinary Diets [IVD] venison).

Erythema annulare centrifugum was reported in a ferret with hyperadrenocorticism.[42] Parallel linear bands of erythema were present over the dorsolateral lumbosacral area and encircling the tail (Fig. 21–10). The dermatitis disappeared after 20 days of treatment with a commercial omega 3/omega 6 fatty acid–containing product (Derm Caps).

The *blue ferret syndrome* is an unusual idiopathic condition affecting ferrets of either sex, neutered or intact.[3] The abdominal skin shows bilaterally symmetric bluish discoloration. Affected ferrets are asymptomatic. The condition regresses spontaneously during a few weeks. In the authors' experience, this condition is most commonly seen in ferrets that have been clipped for surgery or to provide access to veins during the resting phase of the hair cycle. The clipped area remains hairless for a long time, then suddenly begins to turn blue. It appears that hair follicles are making melanin, which will be incorporated

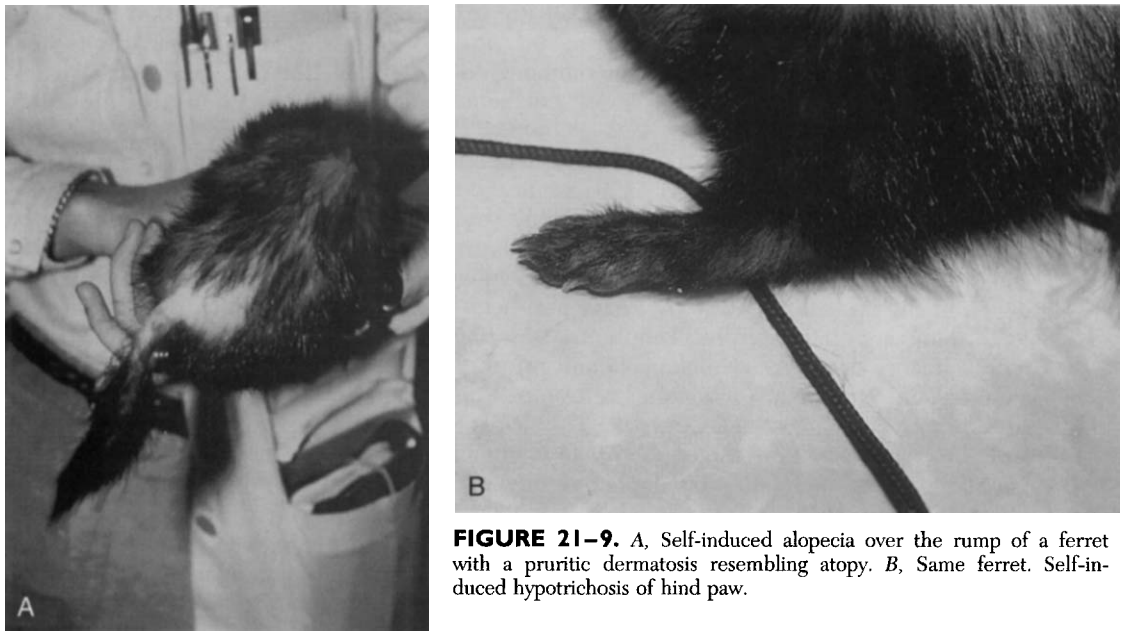

FIGURE 21–9. *A,* Self-induced alopecia over the rump of a ferret with a pruritic dermatosis resembling atopy. *B,* Same ferret. Self-induced hypotrichosis of hind paw.

into growing hairs. Soon after the ferret's skin turns blue, hair regrowth begins (within 1 to 2 weeks).

Self-inflicted facial excoriations (burrowing) may be seen in ferrets that have inadequate bedding, nesting material, or hiding spots.[10] Intact females may pull out hair to use as bedding.[10]

Neoplasia

Skin neoplasms are fairly common in the ferret, and the majority are benign.[10, 17] In one large survey,[33] the skin was the third most common site for primary neoplasms in ferrets (about 13% of all neoplasms). One of the most frequent of these is the mast cell tumor.[10, 17, 33] Lesions may be solitary or multiple and may come and go over time. Mast cell

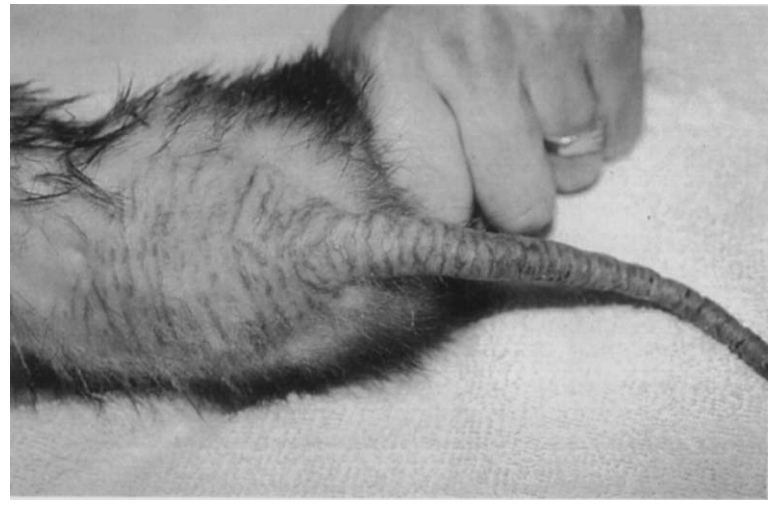

FIGURE 21–10. Erythema annulare centrifugum in a ferret with hyperadrenocorticism. Multiple parallel bands of erythema and scale encircle the tail and involve the dorsal lumbosacral area.

tumors present as papules or nodules (see Fig. 21–2*B*), which vary from skin colored to yellow, brown, or red. They may be firm, soft, or cystic. Some lesions are pruritic. Lesions may occur anywhere, but are most commonly reported on the neck and dorsal trunk. Similar to the situation in cats, most cutaneous mast cell tumors in the ferret are benign.[10, 17]

Basal cell tumors and sebaceous gland neoplasms (especially on the head, neck, limbs, tail and the shoulder [see Fig. 21–2*C*]) are also common in ferrets.[10, 17, 33] Most previously diagnosed "basal cell tumors" were probably sebaceous epitheliomas.[17] Epitrichial sweat gland carcinomas (especially on the tail and the groin), chondromas (especially on the tail), chondrosarcomas (especially on the tail), and squamous cell carcinomas (especially on the digit and the lip [see Fig. 21–2*D*]) have also been reported on numerous occasions.[17, 33]

Cutaneous epitheliotropic lymphoma was reported in an 8-year-old ferret with a 4-month history of progressive alopecia and pruritic dermatitis.[36] The ferret had generalized alopecia and erythema, excoriations, erosions, crusts, and ulcerated plaques on the head, trunk, limbs, paws, footpads, and tail. The nasal planum and footpads were depigmented, and the claws were onychogryphotic. Isotretinoin (2 mg/kg/q 24h, per os) produced a marked improvement after 60 days, but the ferret was euthanized due to renal failure associated with pyelonephritis.

Other cutaneous neoplasms reported in ferrets include papilloma, fibroma, fibrosarcoma, malignant fibrous histiocytoma, histiocytoma, hemangioma, hemangiosarcoma, neurofibroma, neurofibrosarcoma, myxoma, myxosarcoma, ceruminous gland adenocarcinoma, lymphoma, and rhabdomyosarcoma.*

The treatment of choice for cutaneous neoplasms is surgical excision.

• GERBIL

Bacterial Infections

Bacterial skin infections, usually associated with *S. aureus*, are common in gerbils.[7, 8, 17] These infections are virtually always secondary to other perhaps less obvious conditions, especially trauma (cage-related injuries and bite wounds), ectoparasite infestations, and accumulated harderian gland secretions.[6, 8] Infections resulting from cage-related injury are typically seen on the nose and the muzzle (from rubbing on the cage and equipment or burrowing in abrasive litter), whereas those caused by bite wounds typically occur around the head, the tail, the rump, and the perineal area. Those secondary to accumulated harderian gland secretion typically begin on the nose and the periocular area.[6, 8] Staphylococcal infections may be superficial (alopecia, erythema, oozing, crust, and scale) or deep (abscess, fistula, and ulcer), and are usually nonpruritic.

Treatment of bacterial dermatitis includes some combination of eliminating predisposing causes, daily topical cleaning with a 3% hydrogen peroxide or 0.5% to 1% chlorhexidine, and administration of systemic antibiotics (see Table 21–1).

Ectoparasites

Demodicosis has rarely been reported in gerbils.[6, 17] Lesions occurred on the face, the thorax, the abdomen, and the limbs and were characterized by alopecia, oozing, crusts, scales, and secondary bacterial infection. *Demodex merioni* was isolated in skin scrapings. Details concerning pathogenesis and treatment are presently unpublished.

A dermatosis associated with *Acarus farris* was reported.[44] Alopecia, scaling, and thickening of the skin began on the tail, spread to the hind paws, then to the head. Pruritus and excoriation were seen with chronicity. Ivermectin injections were ineffective, but environmental changes (decreased humidity, new litter, new food) and a single application of fipronil spray were curative.

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*See references 6, 10, 17, 28, 32, 33, 40.

Barbering

Although gerbils tolerate crowding better than do most rodents, they chew or "barber" the hair of cagemates.[6, 8] The affected areas appear clipped or shaved, and rarely are any actual skin lesions present. The dorsum of the tail and the top of the head are most frequently involved.

Bald Nose and Sore Nose

Bald nose describes a clinical condition common to the gerbil, which is characterized by alopecia on the muzzle and the dorsum of the nose.[3, 6, 8, 15, 17] There are usually no skin lesions. The alopecia has been attributed to mechanical trauma associated with rubbing against cages and cage equipment, and burrowing in abrasive bedding. Placing animals in a smooth-sided enclosure or aquarium with soft bedding such as shredded paper may be curative.

Bald nose may also be an early stage of the nasal dermatitis (sore nose [see Fig. 21–2*E*]) associated with accumulated harderian gland secretion.[3, 6, 8, 17, 19] These secretions are rich in porphyrins and accumulate about the nasal and facial areas, and apparently lead to the development of an irritant contact dermatitis and secondary staphylococcal infection. The animal's failure to groom the areas adequately leads to irritation, which can then lead to self-inflicted trauma and secondary infection. The stress of overcrowding and high humidity may contribute to the development or the severity of the condition. This condition is common in research colonies and commercial breeding colonies.

The earliest clinical sign is the accumulation of a reddish brown discharge and crust around the nose, the lips, and less frequently, the eyes. This porphyrin-rich secretion exhibits an orange fluorescence when viewed under ultraviolet light (Wood's light). There is frequently protrusion of the nictitans. This is followed by alopecia and, if not treated, dermatitis, pruritus, and secondary staphylococcal infections. Lesions can then spread to the paws, the legs, and the ventrum.

Therapy consists of topical or systemic antibiotic therapy for secondary staphylococcal infection, if present, and housing with access to sand. Surgical removal of the harderian gland is effective but less practical.[6, 17]

Miscellaneous Conditions

An occasional litter of gerbils is born with abnormalities of hair growth and pigmentation.[6, 17] Typically, the back is completely bald, the surrounding haircoat is thinned or patchily alopecic, and the haircoat shows profound leukotrichia. The majority of affected animals fail to thrive and die at the time of weaning. Surviving gerbils develop a normal haircoat as they mature. The etiopathogenesis of this condition is unknown.

The ventral scent gland in gerbils can become inflamed from being rubbed against wood chips or other abrasive bedding.[4] In addition, impaction of these glands can lead to self-mutilation.[4]

When relative humidity is greater than 50%, the normally sleek and smooth gerbil haircoat often appears greasy and stands out.[3] Pine shavings can also cause this appearance.[3]

A gerbil's tail skin is very thin, and easily peels off.[15] If the tail skin is lost, the exposed tail becomes necrotic and sloughs off.[10, 16] Alternatively, the bare tail can be surgically removed where the skin stops.

Neoplasia

The skin is the second most common site of neoplasms in gerbils.[6, 17] Skin neoplasms are typically seen in aged animals (2 to 4 years of age). The most commonly reported skin neoplasms in the gerbil are melanocytomas and melanomas (especially of the paw and the pinna),[5, 16] sebaceous gland adenomas (especially of the ventral scent gland),[6, 17] and

squamous cell carcinoma (especially of the ventral scent gland [see Fig. 21–2*F*] and the pinna).[6, 17, 45] Other reported skin neoplasms in gerbils include papilloma, fibrosarcoma, and neurofibroma.[6, 17]

Diagnosis of skin neoplasms is based on exfoliative cytologic study or biopsy, and the treatment of choice is surgical excision.

• GUINEA PIG

Bacterial Infections

Bacterial skin infections are common in guinea pigs.[2–11, 17] These infections are virtually always secondary to other factors, especially trauma (cage-related injuries and bite wounds) and ectoparasites. Those secondary to bite wounds are typically found around the head, the tail, the rump, and the genital area and are associated with *S. aureus*. Abscesses are occasionally associated with *Corynebacterium kutscheri, Streptococcus zooepidemicus* (especially abscesses on the neck), *Streptobacillus moniliformis,* or *Yersinia pseudotuberculosis.*[2, 6, 8, 17] A staphylococcal cellulitis characterized by thickening and hyperkeratosis of the lips was associated with feeding of tough, fibrous hay.[17] Treatment of these infections includes elimination of predisposing factors, surgical drainage, daily topical applications of 3% hydrogen peroxide or 0.5% chlorhexidine, and systemic antibiotic administration (see Table 21–1).

An exfoliative dermatitis resembling staphylococcal scalded skin syndrome was reported in a guinea pig colony, chiefly among female animals in the late stages of gestation.[17] Bacterial contamination and the abrasive effects of rusty cage floors were suggested as initiating factors. Alopecia was first noted on the ventral abdomen. After a few days, the skin became acutely erythematous and painful. Affected skin subsequently fissured and large thick flakes were desquamated. The condition spontaneously resolved after a course of 10 to 14 days. *S. aureus* was isolated from the skin, the pharynx, the trachea, and nasal washings of affected animals. Skin biopsies revealed intragranular acantholysis and cleavage within the epidermis with minimal inflammation. An exfoliative toxin produced by the staphylococci was reported to cause the skin lesions.

The most common skin disease associated with *S. aureus* infection (occasionally *Corynebacterium pyogenes*) in the guinea pig is *pododermatitis* (bumble foot).[2–11, 17] Predisposing factors include trauma to the footpad, poor sanitation, obesity, aging, and vitamin C deficiency. Affected animals react vigorously when the feet are palpated. The footpad is markedly enlarged, edematous, and erythematous (see Fig. 21–2*G*). Crusts, ulcers, and hemorrhages may be present on the volar surfaces. In chronic or severe cases, the disease process extends to phalangeal and metatarsal or metacarpal bones and joints. Most guinea pigs with pododermatitis have a poor prognosis, because treatment is difficult and systemic amyloidosis is a frequent consequence of chronic infection. Pododermatitis can be prevented by frequently cleaning cages and changing bedding, using cages with smooth surfaces, instituting individual weight control, and providing routine foot care. Early lesions may respond to management changes and daily topical therapy (povidone-iodine or chlorhexidine scrubs, soaks, and ointments under a bandage). Extensive infections also necessitate systemic antibiotics (see Table 21–1).

Fungal Infections

Dermatophytosis is common in guinea pigs and is almost always caused by *T. mentagrophytes.*[10, 17] This dermatophyte can be isolated from the skin and haircoat in approximately 15% of clinically normal guinea pigs.[1, 13, 17, 53a] Rarely, other dermatophytes, such as *M. canis, M. gypseum, M. audouinii,* and *T. verrucosum* can cause disease in guinea pigs.[16] Lesions typically begin as scaling, broken hairs, and alopecia on the nose, which spread to the periocular, forehead, and pinnal areas. In severe cases, the dorsal lumbosacral area is also affected (Fig. 21–11), but the limbs and the ventrum are usually spared. Pruritus is

FIGURE 21–11. *M. canis* infection in a guinea pig. Alopecia and erythema of lateral abdominal and shoulder regions. (Courtesy of E. Guaguère.)

usually minimal or absent. Some animals have more inflammatory lesions characterized by erythema, follicular papules, pustules, crusts, and pruritus. Diagnosis is confirmed by microscopic examination of affected hairs and fungal culture. Treatment is usually accomplished with the topical application of antifungal agents (2% lime sulfur, 1% chlorhexidine, or 0.2% enilconazole dips weekly until the animal is cured).[2–11, 17] Griseofulvin is not usually needed, but can be given at 25 mg/kg every 24 hours orally until the animal is cured.[2–10, 16] The administration of griseofulvin should be avoided in pregnant animals.[5, 16] Dermatophytosis in guinea pigs is an important zoonosis.[17]

Cryptococcosis was reported in a single guinea pig.[17] The animal had a plaque on the dorsum of the nose, which became crusted and ulcerated, and spread into the nostrils. Skin biopsy was diagnostic, and the animal was euthanized.

Guinea pigs have been used as an experimental model for studying the pathogenesis of cutaneous *candidiasis.*[52] Skin lesions are readily produced by the application of *Candida albicans* under occlusion and consist of erythema, pustules, oozing, and crusts.

Guinea pigs have also been used as an experimental model for studying the pathogenesis and treatment of *Malassezia* dermatitis.[53] Skin lesions are readily produced by the application of inocula of *Malassezia (Pityrosporum) ovale* and consist of erythema, edema, crusts, and scales.

Viral Infections

Hairless guinea pigs have been used as an experimental model for studying the pathogenesis of recurrent herpes simplex infection.[46] Intracutaneous inoculation of herpes simplex virus produces pustules and crusts, which spontaneously resolve and can be reactivated by local trauma.

Poxvirus-like virions were demonstrated during the electron microscopic examination of specimens from two guinea pigs with a chronic, crusting cheilitis.[17]

Ectoparasites

Trixacarus (Caviacoptes) caviae is a burrowing sarcoptiform mite and, arguably, the most common cause of ectoparasitic skin disease of guinea pigs.[10, 17, 50, 51] Trixacariasis is always the first differential diagnostic suspect in intense pruritus in guinea pigs. The mite is similar to *S. scabiei* in appearance but is smaller in size (average, 175 μm in length), and the female mite has a dorsal rather than terminal anus. *T. caviae* is similar in size and appearance to *Notoedres* spp. but differs by lacking prominent, sharp, dorsal cuticular

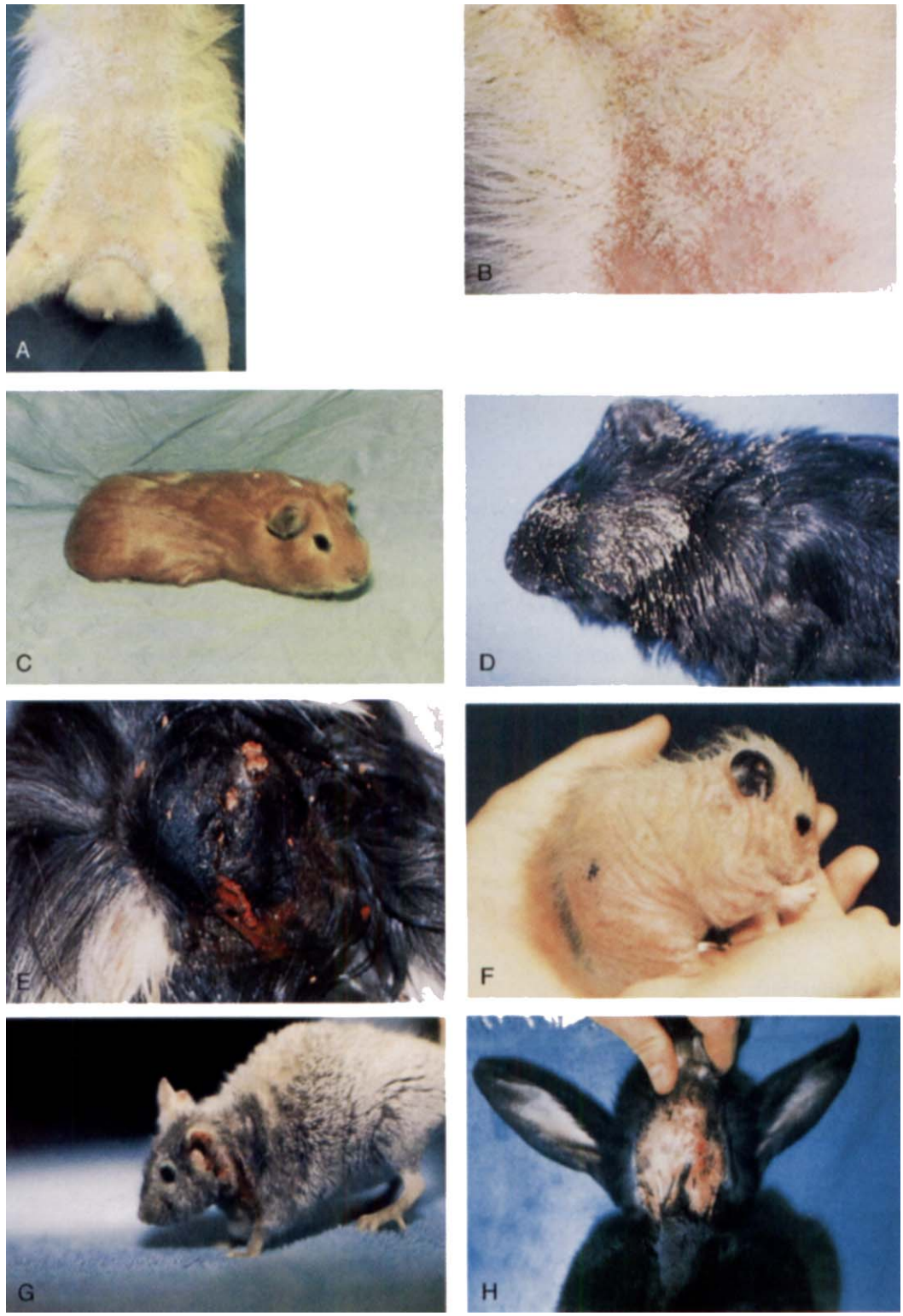

FIGURE 21–12. *Legend on opposite page*

spines. The areas most commonly affected include the dorsal neck and the thorax (see Fig. 21–2*H*), but in severe cases, the entire body may be involved (Fig. 21–12*A*).

Early clinical signs include pruritus, erythema, and traumatic alopecia. Chronic lesions include lichenification, hyperpigmentation, crusts, thick, whitish to yellowish scales (see Fig. 21–12*B*), and brittle, easily epilated hair. The extreme irritation and self-mutilation cause lethargy, anorexia, progressive emaciation, and death associated with bacterial infection or immune-mediated renal disease. Hyperesthesia, behavior such as furiously running in circles and blindly walking into objects, and seizures can be seen and may be triggered by examining the affected animal. Resorption of fetuses and abortion may be seen in breeding animals. Diagnosis may be confirmed by skin scrapings (Fig. 21–13*A*), but it is not unusual for these to be negative. Treatment consists of 2% lime sulfur dips (once weekly until cure is achieved) or ivermectin injections. Orally administered ivermectin is *not* effective in guinea pigs.[51a] *T. caviae* can temporarily infest humans, producing a pruritic papular dermatitis in areas contacted by the guinea pig (arms, thighs, and abdomen).

Chirodiscoides caviae is the guinea pig fur mite.[10, 17, 48] Infestation is uncommon, and clinical disease is probably rare. Heavy infestations can cause pruritus, alopecia, erythema, scaling, and crusting, especially on the dorsolateral lumbosacral area (see Fig. 21–13*C*) and the perineum. Diagnosis is confirmed by the microscopic identification of the mites (see Fig. 21–13*B*), which are often found attached to hair shafts. Treatment is as described for lice. Although injections of ivermectin have been reported to eliminate *C. caviae*,[5, 10, 17] others found this form of therapy to be ineffective.[48]

Other mites rarely reported to affect guinea pigs and produce clinical syndromes characterized by pruritus and dermatitis, which is most prominent on the face, the pinnae, and the dorsum, include *S. scabiei, Notoedres muris,* and *Myocoptes musculinus.*[17] Diagnosis and treatment are as described for *T. caviae* infestation.

Lice are commonly encountered in guinea pigs.[10, 17] The guinea pig may be parasitized by two different biting lice, with *Gliricola porcelli* (the slender guinea pig louse) (see Fig. 21–13*C*) being much more frequently encountered than *Gyropus ovalis* (the oval guinea pig louse). *Trimenopon hispidum* is a biting louse rarely isolated from guinea pigs.[49] Most infestations occur without clinical signs, but heavy infestations may produce a roughened, disheveled haircoat, scaling, crusting, alopecia, and pruritus, especially around the ears and over the dorsum (see Fig. 21–12*D*). Heavy infestations are more commonly encountered in young animals and those with decreased resistance and under poor management. Diagnosis is confirmed by gross or microscopic visualization of lice or nits. Treatment can be accomplished with pyrethrin- or pyrethroid-containing flea powders or sprays approved for cats, 2% lime sulfur dips, or ivermectin injections.[3–6, 10, 17] Cage sanitation is important.

Pelodera dermatitis is rarely reported in guinea pigs.[17] Affected animals have ventral dermatitis consisting of erythema, papules, oozing, crusts, and alopecia. Skin scrapings and skin biopsy reveal the presence of the nematode *Pelodera strongyloides.* Removing contaminated bedding and maintaining a clean, dry cage environment are curative.

Cheyletiellosis is rarely reported in guinea pigs.[17] Affected animals have scaling and variable degrees of hypotrichosis and pruritus on the dorsum. Skin scrapings reveal the mite *Cheyletiella parasitivorax.* Treatment is as described for rabbits.

Demodicosis is rare in guinea pigs.[17] Lesions occur most commonly on the trunk (Fig.

FIGURE 21–12. *A,* Alopecia, erythema, and thick, yellowish crusts on the ventrum of a guinea pig with trixacariasis. (Courtesy of E. Guaguère.) *B,* Close-up of the thick, yellowish crusts on the skin of a guinea pig with trixacariasis. *C, Chirodiscoides caviae* infestation in a guinea pig. Note the uneven, clipped appearance of the haircoat over the caudal half of the body. *D,* Pediculosis in a guinea pig. Numerous nits can be seen over the face and neck. (Courtesy of J. King.) *E,* Trichofolliculoma in a guinea pig. Hyperpigmented, ulcerated tumor over the dorsal lumbar area (Courtesy of W. Gould.) *F,* Epitheliotropic lymphoma in a hamster. Generalized alopecia, erythema, scaling, and exaggerated folds of skin. *G,* Severe self-inflicted alopecia and ulceration over the ear, neck, and shoulder in a mouse with *M. musculi* infestation. (Courtesy of W. Gould.) *H,* Alopecia, erythema, crusts, and ulcers on the ventral neck of a rabbit with *Pseudomonas aeruginosa* infection.

21–14) and consist of alopecia, erythema, papules, and crusts. Pruritus is variable. Diagnosis is confirmed by finding numerous *Demodex caviae* mites in skin scrapings (see Fig. 21–13*D*). Amitraz dips (250 ppm, every week until 4 weeks after skin scrapings are negative) are reported to be an effective treatment.

Fleas *(C. felis)* and ticks may be rarely encountered on guinea pigs.[6, 17] Treatment is the same as that described for cats.

Nutritional Disorders

Nutritional deficiencies, other than vitamin C deficiency, are unlikely to be encountered in pet guinea pigs. Experimental production of nutritional deficiencies with resultant cutaneous abnormalities have been reported and are briefly mentioned here. Protein deficiency produces generalized alopecia.[2, 17] Fatty acid deficiency results in generalized alopecia, scaling, and dermatitis.[2, 17] Pyridoxine deficiency produces alopecia, scaling, and dermatitis, which is most prominent on the limbs, the face, and the pinnae.[2, 17] Vitamin C deficiency is associated with cutaneous petechiae, ecchymoses, hematomas, generalized scaling, and a rough, unkempt haircoat.[2, 3, 8, 17] Vitamin C deficiency is common and is most commonly seen in animals being fed a commercial rabbit chow or an outdated commercial guinea pig chow as the sole diet or in anorectic animals.

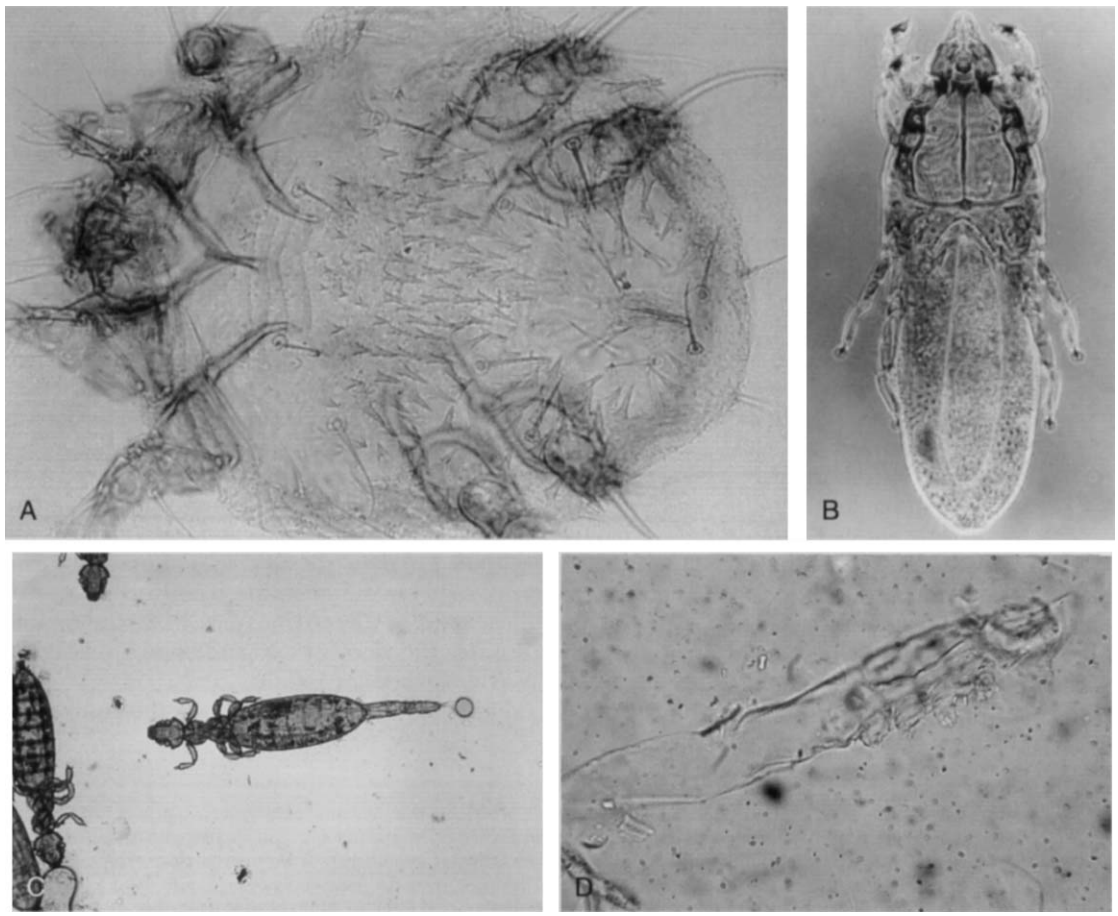

FIGURE 21–13. Ectoparasites. *A, Trixacarus caviae* (Courtesy of K. Thoday.) *B, Chirodiscoides caviae. C, Gliricola porcelli.* (Courtesy of G. Kollias.) *D, Demodex caviae* (Courtesy of D. Carlotti.) *E, Myobia musculli. F, Myocoptes musculinus. G, Psoroptes. H, Listrophorus gibbus.* (*B, E* to *G* from Bowman DD: Georgis' Parasitology for Veterinarians, 7th ed. W.B. Saunders Co., Philadelphia, 1999.)

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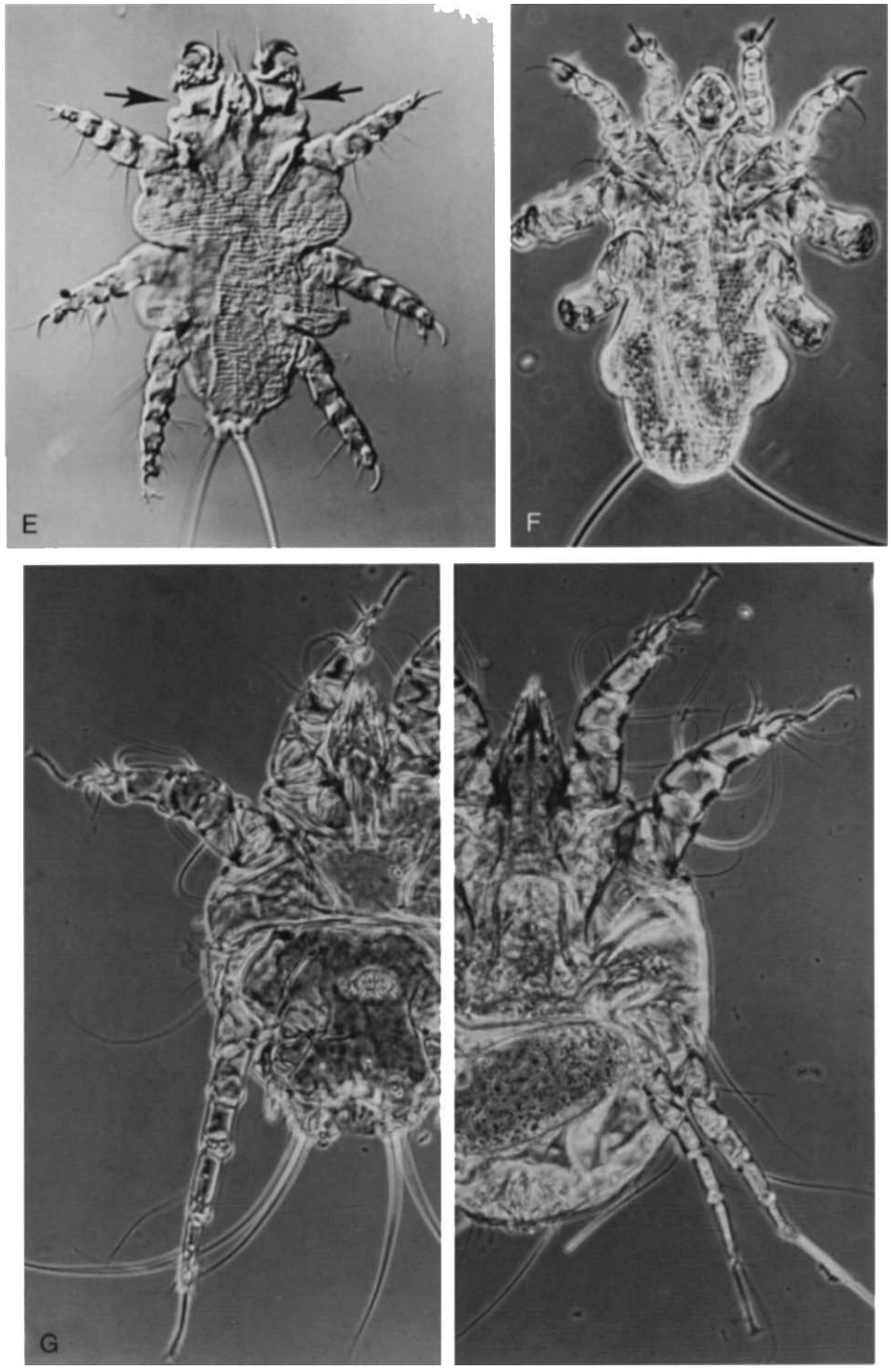

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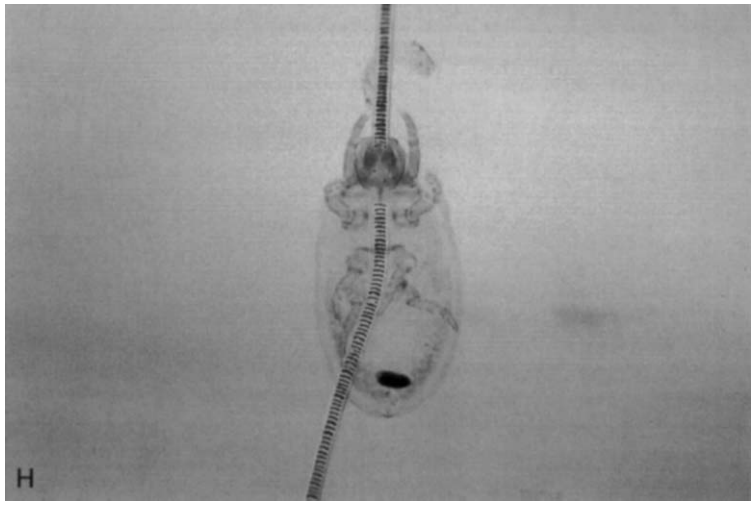

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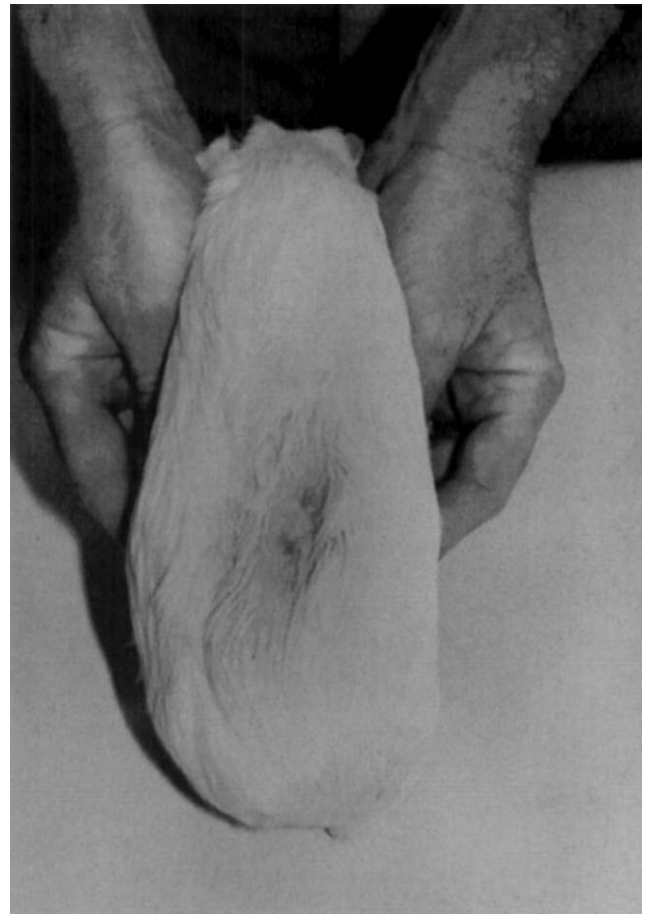

FIGURE 21–14. Alopecia and erythema over the back of a guinea pig with demodicosis. (Courtesy of D. Carlotti.)

Miscellaneous Conditions

Telogen defluxion is frequently seen in the last trimester of pregnancy or during lactation.[6, 8, 17] The alopecia is most prominent on the lumbosacral area and the flanks.

Marked shedding during stress is common in guinea pigs.[3] When guinea pigs are sick, it is often easy to epilate an entire area of haircoat when tenting the skin.

Guinea pigs establish male-dominated social hierarchies, and animals of low social ranking or young animals may lose considerable amounts of hair, especially on the head, the rump, the perineum, and the prepuce owing to barbering or receiving bite wounds.[6, 8, 17, 19] Ear chewing can be a problem, resulting in ear margin notches or actual cropping close to the head. Guinea pigs may also self-barber, producing hair loss in only those areas that they can reach with their mouth (the fur on the head, the neck, and the anterior shoulders is intact). Hair loss due to barbering appears irregular in length and clipped. The underlying skin is usually normal in appearance. In some cases, the addition of long-stemmed hay resolves the barbering, suggesting that the cause was boredom or a need for fiber.[5, 8, 17, 19]

Necrosis of digits and paws is occasionally seen when owners put objects such as cloths and socks in cages with guinea pigs.[17] Segments of these materials become wrapped around distal extremities and serve as constricting bands or tourniquets. Tissues distal to the constriction become swollen and painful, then necrotic, and then they slough (Fig. 21–15).

Apparent *ergot poisoning* was characterized by the sudden onset of anorexia, lethargy, and lameness.[47] The feet became discolored, desiccated, insensitive to touch, and wood-like.

Thinning of the haircoat is common near the time of weaning in neonates and spontaneously resolves.[6, 17]

Hereditary hairlessness has been reported in guinea pigs (Fig. 21–16).[17] Bilaterally symmetric alopecia over the flanks and trunk has been seen in guinea pigs with cystic ovaries (Fig. 21–17).[4, 10, 17] Affected females usually present with progressive enlargement of the abdomen, and ovarian cysts may be palpable as discrete, large, rounded masses in the dorsal middle abdomen. Ultrasonographic examination may be diagnostic.[10] Ovariohysterectomy is curative.

Sebaceous glands are especially abundant around the anus, in the folds of the perianal and genital regions, and in the dorsal sacral skin, especially in sexually mature male animals.[6, 10, 17] Excessive accumulations of sebaceous debris, and occasionally bedding material and feces, may become entrapped in these folds or mat down the dorsal sacral

FIGURE 21–15. Necrosis and slough of the hindfoot of a guinea pig caused by a constricting band. (Courtesy of G. Kollias.)

FIGURE 21–16. Congenital alopecia in a guinea pig. (Courtesy of J. Gourreau.)

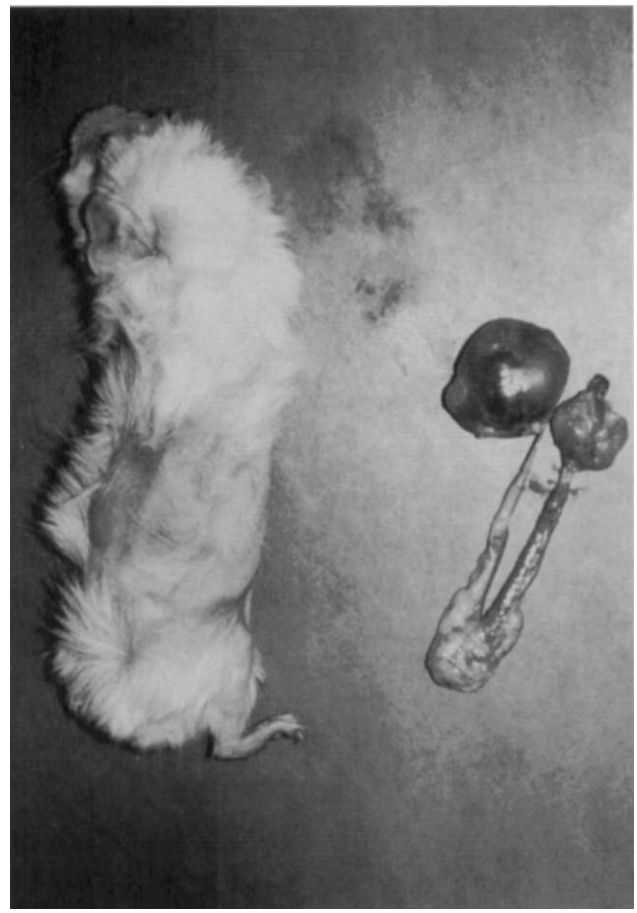

FIGURE 21–17. Truncal alopecia in a guinea pig associated with bilateral cystic ovaries. The cystic ovaries and uterus are on the right.

haircoat. Intertrigo, secondary bacterial infections, and unpleasant odors may intervene in this situation. These areas should be cleaned as needed with 3% hydrogen peroxide, 0.5% chlorhexidine, or a mild astringent (aluminum subacetate) to prevent the aforementioned sequelae.

Heavy guinea pigs, especially those maintained in wire-bottom cages, frequently develop hyperkeratosis and cutaneous horns of the footpads.[6, 17] These horny growths are most commonly observed on the ventral aspect of the front paws. They can be removed with scissors and an emery file. Replacement of cage surfaces with smoother materials retards or prevents further hyperkeratoses.

Overgrown claws are a frequent problem in pet guinea pigs.[3, 17] Frequent examination and trimming prevents traumatic and infectious complications.

Neoplasia

The skin is the second most common site of neoplasia in the guinea pig.[2, 3, 6, 8, 17] The most common cutaneous neoplasm is the trichofolliculoma.[17] This tumor is most commonly encountered as a benign, solitary lesion over the dorsal lumbar area (see Fig. 21–12*E*). The overlying skin is usually alopecic and crusted. Frequently, a central pore is seen, through which keratinous material or dark, hemorrhagic exudate is discharged. Other cutaneous neoplasms reported in guinea pigs include sebaceous adenoma, fibroma, fibrosarcoma, lipoma, liposarcoma, schwannoma, and lymphoma.[2, 17]

• HAMSTER

Bacterial Infections

Bacterial skin infections are uncommonly reported in hamsters. *S. aureus* and *Pasteurella pneumotropica* have been recovered from isolated cases of skin abscesses and bite wounds.[6, 8] Hamsters are prone to periodontal disease and dental caries, and any facial abscess located ventral or cranial to the eye may be a tooth root abscess.[10] Treatment of bacterial dermatitis includes elimination of predisposing causes, surgical drainage, daily topical cleaning with 3% hydrogen peroxide or 0.5% chlorhexidine, and occasionally systemic antibiotic administration (see Table 21–1).

Experimentally, the hamster is susceptible to infection with *Treponema pallidum* subsp. *endemicum,* the agent of endemic syphilis of humans.[56] Intradermal injection of the spirochete results in erythematous papules and ulcers, which eventually heal but are followed by perioral ulcers and an erythematous maculopapular rash on the paws and the trunk and death. This form of hamster syphilis has been proposed as a useful model for the study of the immune response, antibiotic therapy, and vaccination techniques in human venereal and congenital syphilis.

Fungal Infections

Dermatophytosis is rare in hamsters and is caused by *T. mentagrophytes*.[2, 3, 6, 8, 16–19] Diagnosis is confirmed by microscopic examination of affected hairs and fungal culture. Treatment is as described for the other rodents.

Ectoparasites

Demodicosis is the most common ectoparasitism of the hamster.[10, 17] *Demodex criceti* and *D. aurati* are both normal residents of hamster skin.[17] *D. aurati* is long and tapered (average, 180 μm long) and inhabits hair follicles, whereas *D. criceti* is short and stubby (average, 90 μm long) and inhabits the keratin and pits of the epidermal surface. Clinical demodicosis is seen in aged hamsters, is usually associated with *D. aurati,* and is usually associated with conditions that suppress immune responses (e.g., malnutrition, concurrent

disease, cancer, and exposure to carcinogens).[6, 8, 17, 19] Lesions are most commonly seen over the dorsal lumbosacral area but may be generalized (Fig. 21–18). Moderate to severe alopecia is accompanied by variable degrees of scaling, erythema, and small hemorrhagic crusts. Pruritus is usually absent. Diagnosis is confirmed by skin scrapings. Treatment of demodicosis in hamsters has not been extensively evaluated or reported. The authors and others [4, 19] were successful with 250 ppm of amitraz applied as a whole-body dip once weekly until 4 weeks after skin scrapings are negative.

Notoedric mange is rarely reported in hamsters.[8, 17, 19] Lesions are characterized by thick yellowish crusts, erythema, and alopecia on the pinnae, the muzzle, the tail, the genitalia, and the paws. Pruritus is severe, and self-mutilation can be extreme. Diagnosis is confirmed by finding *Notoedres* sp. mites in skin scrapings. Treatment with 2% lime sulfur dips (whole-body application weekly until two treatments after clinical cure) or ivermectin injections is effective.

S. scabiei, T. caviae, and *Ornithonyssus bacoti* are reported to be rare causes of pruritus and dermatitis in hamsters.[17] Clinical signs, diagnosis, and treatment are the same as that described for notoedric mange.

Fleas *(C. felis felis)* are rarely encountered on hamsters.[6] Treatment is the same as that described for cats.

Traumatic Alopecia and Dermatitis

Female hamsters are generally more aggressive than are males.[6, 17] Aggressive behavior in male hamsters is testosterone dependent and is markedly reduced by castration.[6, 17] The establishment of social dominance among male hamsters is positively correlated with the weight, size, and degree of pigmentation of the flank glands.[17] Aggression-related bite wounds are most commonly seen around the head, the tail, and the perineal area. Aggression can produce severe wounding, such as the complete removal of the flank glands of the victim.[17]

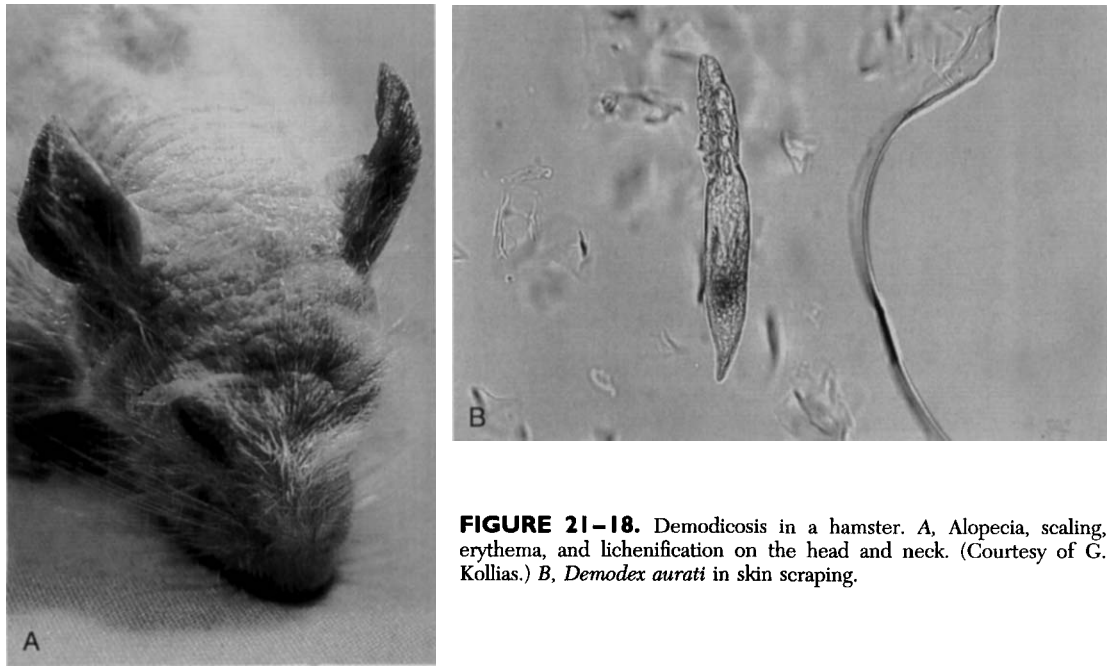

FIGURE 21–18. Demodicosis in a hamster. *A,* Alopecia, scaling, erythema, and lichenification on the head and neck. (Courtesy of G. Kollias.) *B, Demodex aurati* in skin scraping.

Nutritional Disorders

Nutritional deficiencies are unlikely to be seen in pet hamsters. However, one author[9] indicates that hair loss in hamsters is associated most often with continuous feeding of low protein (less than 16%) feed, such as is commonly found in pet stores. Experimental production of nutritional deficiencies with resultant cutaneous abnormalities have been reported and are briefly mentioned here. Pantothenic acid deficiency produces exfoliative dermatitis, depigmentation of the haircoat, and the accumulation of porphyrin-rich secretions around the nose, the mouth, and the eyes.[2, 17] Riboflavin deficiency produces alopecia, scaling, and dermatitis, which are most evident on the extremities.[2, 17] Pyridoxine deficiency results in generalized alopecia and depigmentation of the haircoat.[2, 17] Niacin deficiency produces a generalized alopecia.[2, 17] Fatty acid deficiency results in generalized alopecia, scaling, and the production of profuse amounts of cerumen.[2, 17] Copper deficiency results in alopecia and depigmentation.[2]

Miscellaneous Conditions

Foreign body granulomas associated with bedding consisting of wood shavings and sawdust were reported on the paws and the shoulders of hamsters.[17] The problem was eliminated by using shredded paper as bedding.

Swelling and pruritus of the face and the paws has been reported in several hamsters.[3] In all instances, owners had recently purchased a fresh bag of cedar or pine shavings produced by a leading pet company or bulk shavings from landscaping or nursery outlets. When affected animals were housed on plain newspaper, the lesions spontaneously regressed. Presumably, the shavings had been treated with some chemical that produced a contact dermatitis.

Hereditary hairlessness has been reported in hamsters.[17]

Hyperadrenocorticism is rarely reported in hamsters.[6, 54] Clinical signs include bilaterally symmetric alopecia, hyperpigmentation, and thinning of the skin. The baseline plasma cortisol value in one affected hamster was elevated (approximately twofold increase) compared with that in one normal hamster. One hamster was treated with metyrapone (8 mg orally q24h for 1 month), and hair regrowth was complete after 12 weeks. A second hamster was treated with o,p'-DDD (5 mg orally q24h for 1 month), then metyrapone as described previously, and responded to neither drug. This hamster was euthanized, and a chromophobe adenoma of the hypophysis and bilateral adrenocortical hyperplasia were found at necropsy.

The flank scent glands of the hamster can become inflamed from being rubbed against wood chips or other abrasive cage equipment.[4] In addition, impaction of these glands can lead to self-mutilation.[4]

Ringtail is occasionally seen in the hamster (see Rat in this chapter).[2, 17]

Neoplasia

Skin neoplasms are rare in hamsters.[2, 6, 8, 17] The most frequently reported cutaneous neoplasms are melanomas and melanocytomas.[2, 6, 17] These tumors occur much more frequently in male hamsters and most commonly on the back, the head, the neck, and the flank gland.

Epitheliotropic lymphoma (mycosis fungoides) is the second most common cutaneous neoplasm of the hamster.[6, 17, 55] Affected animals have an exfoliative erythroderma (see Fig. 21–12*F*), which is generally pruritic, and go on to manifest peripheral lymphadenopathy, lethargy, anorexia, emaciation, and death. Some animals also have cutaneous plaques and nodules (Fig. 21–19), which may become ulcerated and crusted. Light and electron microscopic examinations demonstrated an epitheliotropic lymphoma in which many cells show the typical features of Sézary cells.[55] Immunohistochemical studies showed that the neoplastic cells are T lymphocytes.[55] Therapeutic trials have not been reported.

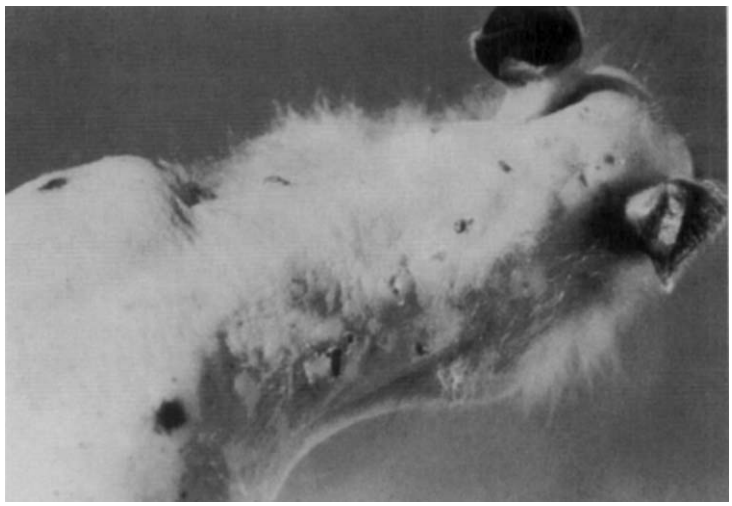

FIGURE 21–19. Epitheliotropic lymphoma in a hamster. Generalized alopecia and multiple crusted nodules and plaques.

Other cutaneous neoplasms reported in hamsters include basal cell carcinoma, squamous cell carcinoma, keratoacanthoma, papilloma, epitrichial sweat gland adenoma, fibrosarcoma, and plasmacytoma.[2, 6, 17]

• MOUSE

Bacterial Infections

Bacterial skin infections are uncommon in pet mice, are usually caused by *S. aureus*, and are secondary to trauma (cage-related injuries and bite wounds) or self-inflicted (the pruritus associated with ectoparasites).[2, 7, 8, 17, 19] Infections resulting from cage-related injury are typically seen on the nose and the muzzle, whereas those associated with bite wounds typically occur around the head, the tail, the rump, and the perineal area. Staphylococcal infections may be superficial (alopecia, erythema, oozing, and crust) or deep (abscess, fistula, necrosis, and ulcer) and are usually nonpruritic. Submandibular and periorbital granulomas were associated with bacterial pseudomycetuna (botryomycosis) due to *S. aureus*.[72] Other bacteria occasionally isolated from cutaneous abscesses and pyogranulomas in mice include *Streptococcus* sp., *P. pneumotropica, Actinobacillus* sp., *Actinomyces* sp., and *Klebsiella*.[2, 8, 17] Treatment of bacterial dermatitis includes some combination of elimination of predisposing causes, surgical drainage, daily topical cleaning with 3% hydrogen peroxide or 0.5% to 1% chlorhexidine, and systemic antibiotic administration (see Table 21–1). Surgical excision may be the best treatment for abscesses.[7a]

S. moniliformis is a rare cause of epizootics of edema and cyanosis of the extremities.[2, 8, 17]

C. kutscheri (murium) is a rare cause of epizootics of furunculosis and cutaneous pyogranulomas, which may progress to necrosis and sloughing of extremities.[2, 8, 17]

The mouse has been used as an experimental model for staphylococcal scalded skin syndrome.[17, 62]

Mycobacterium chelonae infection caused nodular, granulomatous lesions on the tails of immunocompromised mice.[64a]

Fungal Infections

Dermatophytosis is uncommon in mice and is usually caused by *T. mentagrophytes*.[1–6, 13, 16–19] This dermatophyte can be isolated from the haircoat of up to 60% of clinically normal mice in pet shops and represents an important zoonosis. Lesions are most commonly seen on the face, the head, the tail, and the trunk and consist of annular areas of

alopecia, broken hairs, scales, and variable degrees of erythema and crusting. Pruritus is usually minimal to absent. Diagnosis and treatment are the same as that described for guinea pigs.

Viral Infections

Mouse pox (infectious ectromelia) causes epizootics in research colonies but is rarely, if ever, seen in practice.[2, 3, 6–8, 17, 66] Skin lesions include a generalized papular dermatitis with eventual swelling, necrosis, ulceration, and even sloughing of digits, pinnae, and tail. Diagnosis is confirmed by skin biopsy, electron microscopic examination of crusts, viral isolation of the orthopoxvirus, and polymerase chain reaction.[17, 65]

Reovirus Type 3 infection of suckling mice causes severe illness and an oily haircoat.[2, 8, 17] Animals that survive past weaning experience alopecia.

Sialodacryoadenitis virus infection causes eye rubbing and scratching, periorbital swelling, and red tears (chromodacryorrhea).[2, 9, 17]

Ectoparasites

Myobia musculi is a mite commonly found on mice.[2–6, 8, 16–19] Some animals are asymptomatic carriers, whereas other animals show varying degrees of skin disease and pruritus. Severely inflammatory and pruritic forms of the infestation are associated with genetic susceptibility and mite-related hypersensitivity reactions.[17] Immature mice or those that are immunocompromised may be more susceptible to severe forms of the disease. Clinical signs may be mild and include patchy alopecia, slight erythema, and minor scaling on the head and the muzzle. Other mice may show intense pruritus and self-mutilation of the face, the head, the pinnae, the neck, and the shoulders (see Fig. 21–12*G*). Severely affected animals can become debilitated and die. Diagnosis is confirmed by skin scrapings (see Fig. 21–13*E*). The treatment of choice is subcutaneous injections of ivermectin.[3, 4, 17, 19, 71] Ivermectin administered topically or in drinking water is less effective.[59, 60, 67, 69]

Myocoptes musculinus is a mite commonly found on mice.[2–6, 8, 16–19] Some animals are asymptomatic carriers, whereas others manifest varying degrees of skin disease and pruritus. Lesions are most severe on the back and the ventrum. Unlike the case with *M. musculi* infestations, severe ulceration is not seen with *M. musculinus* infestations. Diagnosis is confirmed by skin scrapings (see Fig. 21–13*F*); however, the mites are often difficult to find. The treatment of choice is subcutaneous injections of ivermectin every 2 weeks until the animal is cured.[3, 4, 17, 19, 71] Ivermectin administered topically or in drinking water is less effective.[59, 60, 67] Other mites that are rarely found on mice include *Radfordia affinis, Psorergates simplex, O. bacoti, S. scabiei, N. muris,* and *Trichoecius romboutsi.*[2–6, 8, 9, 17, 19]

Fleas (especially *C. felis felis)* may be recovered from pet mice maintained in households frequented by dogs and cats.[2–6, 8, 17]

The sucking louse *Polyplax serrata* is occasionally found on pet mice.[2–6, 8, 9, 17, 19] Some animals may be asymptomatic carriers, but others manifest varying degrees of dermatitis and pruritus. Young animals, debilitated animals, and animals in poor management situations are more likely to be affected. Lice and related dermatoses are most commonly found on the neck and back. Treatment can be accomplished with topical insecticides or ivermectin injections.

Nutritional Disorders

Nutritional deficiencies are unlikely to be encountered in pet mice. Experimental production of nutritional deficiencies with resultant cutaneous abnormalities have been reported and are briefly mentioned here. Zinc deficiency produces exfoliative dermatitis, alopecia, and depigmentation of the haircoat.[2, 17] Pantothenic acid deficiency results in exfoliative dermatitis and depigmentation of the haircoat.[2, 17] Riboflavin deficiency produces alopecia, scaling, and dermatitis, especially on the extremities.[2, 17] Pyridoxine deficiency results in

exfoliative dermatitis, especially on the face, the ears, the limbs, and the tail.[2, 17] Biotin deficiency causes exfoliative dermatitis.[2] Fatty acid deficiency produces an exfoliative dermatitis.[2, 17]

Miscellaneous Conditions

Male mice are aggressive.[2-6, 8, 17, 19] Barbering and bite wounds are frequently seen, especially on the muzzle, the whiskers, the face, the head, the rump, the tail, and the perineum. These behaviors can be exacerbated by crowding, stress, and boredom. Mice frequently rub the hair off the muzzle as they stick their face through slotted feeders or wire bars.

Mice develop numerous types of hereditary hairlessness and keratinization defects, which are probably never seen by the practitioner.[2, 8, 17, 68, 69] Some of these conditions have been used as laboratory models for the study of various aspects of cutaneous pathophysiology and pharmacology, such as ichthyosis, asebia, rhino, and blotchy (similar to the Menkes kinky hair syndrome in humans) and flaky skin (similar to psoriasis in humans).[17, 62, 63, 68, 73]

Ringtail is rarely reported in mice (see Rat in this chapter).[8, 17] Idiopathic dry gangrene of the pinna is sporadically seen in young mice.[8, 17] The incidence appears to increase when the mice are exposed to cold temperatures and when the ears are traumatized by excessive grooming in attempts to remove lice. The condition progresses rapidly from initial erythema of the distal one third of the pinna to necrosis and slough. Rarely, the distal one third of the tail is also involved.

Perianal pruritus is seen in association with pinworms *(Syphacia obvelata).*[2, 4, 8, 17, 19] Infected mice often mutilate the base of the tail. Diagnosis is confirmed by microscopic examination of strips of cellophane (Scotch) tape that have been applied to the perineum. The eggs of *S. obvelata* are banana shaped and about 30 μm by 150 μm. Treatment with ivermectin injections is curative.

A spontaneous *immune complex vasculitis* was reported to affect up to 21% of certain strains of aged mice.[58] Mice developed multiple crusts between the scapulae or on the dorsal neck. These lesions rapidly evolved into irregular ulcers and spread laterally and caudally on the body. Pruritus was intense. Histologic examination revealed leukocytoclastic vasculitis and IgG, IgM, and fibrinogen were demonstrated in dermal blood vessel walls.

Alopecia areata was reported in the C3H/Hej mouse and proposed as a model of the human disease.[70]

Neoplasia

Although mice are sensitive to the induction of various skin neoplasms by the topical or systemic administration of chemical carcinogens or ultraviolet light exposure, spontaneous skin neoplasms are rare.[2, 3, 6, 8, 17] The most commonly reported cutaneous neoplasms are papilloma, squamous cell carcinoma, and fibrosarcoma.[17, 64] Other reported cutaneous neoplasms include hair follicle tumors, sebaceous gland tumors, mast cell tumors, hemangiomas, hemangiosarcomas, melanomas, lymphomas, and a solitary epitheliotropic lymphoma resembling pagetoid reticulosis in humans.[17, 57, 61]

• RABBIT

Bacterial Infections

Pasteurellosis (snuffles) is the most common bacterial disease of the rabbit.* Most rabbits carry *Pasteurella multocida* asymptomatically in the nasal cavity, and under conditions of

*See references 2–5, 8, 10, 11, 17, 77, 80, 81, 86, 92, 96.

stress, the bacteria multiply and cause disease. Subcutaneous abscesses develop as a result of septicemia, external wound contamination, or direct extension from deeper sties. The abscesses are variable in size, are usually firm on palpation, and are filled with a thick, white to tan exudate. Diagnosis is confirmed by microscopic examination of direct smears of exudate and culture. In the rabbit, subcutaneous abscesses are due to pasteurellosis until proven otherwise. Other causes of abscesses include *S. aureus, Fusobacterium, Pseudomonas aeruginosa, Streptococcus* sp., and *C. pyogenes.** Abscesses on the head may be secondary to dental disease, a tooth root abscess, or an oral foreign body.[10]

Abscesses in rabbits are typically filled with thick, caseated pus and are surrounded by a thick capsule. These two attributes make the use of drainage, drains, and topical and systemic antibiotics unsuccessful in most cases.[7a, 10, 15] The treatment of choice is surgical excision and at least 2 weeks of antibiotic treatment (see Table 21–1).[10, 15, 81] If this is impossible, radical débridement, flushing, and antibiotics for several weeks may be effective.[7a] In some rabbits, abscesses continue to recur in the same spot or at some other sites in an apparently healthy individual.[10] Some of these rabbits may require lifetime antibiotic therapy to prevent recurrences.[10]

Necrobacillosis (Schmorl's disease) is a sporadic bacterial infection of rabbits caused by *Fusobacterium necrophorum.*† It is characterized by inflammation, necrosis, ulceration, and abscessation, especially on the face, the head, and the neck. Diagnosis is confirmed by culture. Treatment is accomplished with surgical debridement, topical antimicrobial applications, and systemic penicillin or tetracycline administration.

P. aeruginosa causes a *localized moist dermatitis (sore dewlap)* and, occasionally, subcutaneous abscess in areas of skin that are continuously wet.‡ The muzzle, the dewlap (see Fig. 21–12*H*), the flank, and the haunches are most commonly involved. The affected skin is moist, erythematous, edematous, alopecic, and often ulcerated. The fur is often clumped, creating a spiked appearance. The most striking clinical feature is the blue-green color of the fur in animals with white fur, which is caused by a water-soluble pigment (pyocyanin) produced by the bacteria. Diagnosis is confirmed by microscopic examination of direct smears from oozing areas and culture. Treatment includes clipping, gentle cleaning, and application of astringents (aluminum acetate) and topical gentamicin sulfate ointment. Prevention is directed at removing the cause of continued wetness of the fur. The most common cause is the constant drooling ("slobbers") associated with dental disease.[10] Leaking water valves or water bottles should be replaced. Water bowls or pans should be replaced by water bottles with sipper tubes. Malocclusion of the teeth should be corrected to prevent drooling. Wet bedding should be changed more frequently.

Ulcerative pododermatitis (sore hocks) is a common disorder in rabbits.§ Genetic predilection is important, as large body size and thinner plantar fur pads are important predisposing factors. Unsanitary cage conditions, rough cage surfaces, and obesity also contribute to pressure necrosis and secondary bacterial infection with *S. aureus.* Lesions commonly occur unilaterally or bilaterally on the plantar aspect of the metatarsal region or, less commonly, the volar surface of the metacarpal area. Focal inflammation, oozing, crusts, and alopecia progress to ulcers, hemorrhage, and abscesses. In severe infections, the disease may extend to the bony structures of the foot and result in septicemia. Treatment includes correction of predisposing conditions, surgical drainage, topical antimicrobial applications, and systemic antibiotic administration (see Table 21–1). Severe cases usually do not respond.

Venereal spirochetosis (treponematosis, rabbit syphilis, or vent disease) is uncommon.[2–6, 7a, 10, 17, 92, 96] *Treponema paraluis-cuniculi,* the causative spirochete, is transmitted by direct contact, especially mating. Cold environments appear to predispose to the disease. Because of the grooming, social, and sleeping habits of rabbits, lesions are

*See references 2, 6, 8, 11, 17, 82, 92, 96.
†See references 2, 3, 7, 8, 17, 92, 96.
‡See references 2–6, 8, 10, 17, 92, 96.
§See references 2–6, 8, 10, 11, 17, 92, 96.

frequently seen on the nose (Fig. 21–20A), the lips, the chin, the face, the eyelids, the ears, and the paws as well as the genitalia. Lesions consist of vesicles, papules, erythema, edema, oozing, erosions, and brownish crusts. Focal ulcers and hemorrhage may be seen. Diagnosis is confirmed by skin biopsies, the Venereal Disease Research Laboratory (VDRL) slide test, and the rapid plasma reagin (RPR) card test. Treatment with penicillin G benzathine or penicillin G procaine is curative (42,000 IU/kg, subcutaneously, once a week for three treatments). Tetracycline or chloramphenicol is also effective.[7a].

An outbreak of *S. aureus* infection in a rabbitry was associated with a pustular, exudative dermatitis in the young and mastitis in lactating does.[93] A cellulitis due to *S. aureus* infection is occasionally seen and is characterized by the acute onset of fever, and painful edematous swelling, especially over the head, neck, and thorax.[86] Necrosis and slough may occur.

Fungal Infections

Dermatophytosis is common in rabbits. *T. mentagrophytes* is the most common dermatophyte isolated, but *M. canis, M. gypseum, M. audouinii, T. verrucosum,* and *T. schoenleinii* have been reported.* *T. mentagrophytes* can be isolated from the haircoat and skin of approximately 36% of clinically normal rabbits, representing an important potential zoonosis. The disease is most common in young animals and where husbandry and management are suboptimal. Lesions are characterized by patchy alopecia, broken hairs, erythema, and yellowish crusting, and typically first appear on the bridge of the nose (see Fig. 21–20*B*), the eyelids, the pinnae, and the paws, and occasionally, on many body sites. The condition is usually pruritic. Diagnosis and treatment are the same as that described for the guinea pig. Griseofulvin is teratogenic, and should not be used in breeding does. A modified live *T. mentagrophytes* vaccine may prove useful in prophylaxis.[17]

Aspergillosis of the lungs and skin was reported in a whole litter of 4-week-old rabbits.[17] Multiple 1- to 2-mm papules were present all over the body. Histologically, the papules were cystic follicles distended with necrotic debris and dichotomously branching hyphae. *Aspergillus* sp. was isolated in culture. The animals were raised on moldy grass hay bedding material. A change in nesting materials prevented further occurrences.

Viral Infections

Myxomatosis is occasionally observed in domestic rabbits.† The myxoma virus (a poxvirus) is transmitted from reservoir wild rabbit hosts by mosquitoes. There are several strains of virus with variable virulence. In domestic rabbits, severe disease and high mortality are frequently produced. Affected rabbits are febrile, lethargic, and depressed. In the acute form of the disease, there is edema and erythema of the anus, the genitalia, the lips, the nares, and the eyelids. Less virulent strains of the virus produce numerous skin tumors (see Fig. 21–20*C*). Myxomatosis appeared in the depilated skin of Angora rabbits.[17] Lesions were a few millimeters to 3 cm in diameter, erythematous, and plaquelike, and became hemorrhagic and necrotic. Morbidity was low and mortality infrequent. Diagnosis is based on distinctive clinical signs, biopsy, and virus isolation. There is no effective treatment, and control of insect vectors and screening of enclosures are paramount in endemic areas. Heterologous vaccine may be useful.

Rabbit pox is infrequently reported in domestic rabbits.‡ The causative poxvirus is closely related to vaccinia virus. Initial clinical signs of profuse nasal discharge, depression, and fever are followed in 4 to 5 days by a generalized, erythematous, macular to papular to nodular eruption. The rabbits have extensive edema of the face and perineum. Diagnosis is confirmed by biopsy and virus isolation.

Shope fibroma virus and Shope papilloma virus are oncogenic (see Neoplasia).

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*See references 2, 3, 6, 8, 10, 17, 92, 96.
†See references 2, 7, 8, 10, 11, 17, 92, 96.
‡See references 2, 7, 8, 10, 11, 17, 92, 96.

FIGURE 21–20. *A,* Crusts on the nose of a rabbit with spirochetosis. (Courtesy of G. Kollias.) *B,* Focal area of alopecia and crusting over the nose of a rabbit due to *T. mentagrophytes* infection. (Courtesy of G. Kollias.) *C,* Multiple erythematous nodules and plaques around the eye, on the pinna, and on the muzzle of a rabbit with myxomatosis. (Courtesy of G. Kollias.) *D,* Crusting and erythema of the lateral surface of the pinna of a rabbit with psoroptic mange. *E,* Crusts, scale, and focal ulcers over the dorsum of a rabbit with cheyletiellosis (area has been clipped). *F,* Frostbite in a rabbit. Note acrocyanosis and necrosis of the pinna. *G,* Orange-colored crust and discoloration of the hair around the eye of a rat with sialodacryoadenitis virus infection. (Courtesy of J. King.) *H, Trixacarus diversus* in a rat. Marked crusting and alopecia on the face and pinnae. (Courtesy of P. Bourdeau.)

Ectoparasites

Psoroptes cuniculi, a nonburrowing mite, is the most common ectoparasite of the rabbit, and all rabbits should be considered infected until proven otherwise.* Rabbits are also susceptible to *P. ovis* (cattle and sheep).[17] *P. cuniculi* is transmitted by direct contact with infected rabbits, fomites, and contaminated environment. Starving mites survive for approximately 21 days off the host over the usual range of temperatures (5 to 30°C [41 to 86°F]) and relative humidities (20% to 75%).[17] Crusts dislodged into the environment contain many mites. The mites pierce skin to feed, and hypersensitivity to mite-related antigens may be important in the pathogenesis of the dermatitis and pruritus.[17]

P. cuniculi typically produces otitis externa (otoacariasis, ear canker, and ear mites) (see Fig. 21–20*D*). Affected rabbits shake their heads and scratch at the head and ears. Alopecia, excoriations, and secondary bacterial infection may be present around the head, neck, and ventrum.[17, 79] In early stages, a dry, whitish gray to tan crusty exudate forms inside the vertical ear canal. Later, a dry, crusty material with a layered appearance accumulates in the ear and the lateral surface of the pinnae. A secondary bacterial infection may complicate the parasitic otitis externa, contributing to the foul odor and pain. Occasionally, mites may produce lesions on the face, the head, the neck, the limbs, the abdomen, and the back.[17, 83, 97]

Diagnosis is confirmed by finding the mites in ear swabs or skin scrapings (see Fig. 21–13*G*). In one report in which natural infections were studied and mite numbers were quantitated, affected animals harbored 40 to 100,000 mites per rabbit. The treatment of choice is the subcutaneous injection of ivermectin.[10, 17, 81, 95] Do *not* attempt to clean out the crusts and debris, because this causes pain and bleeding. The cage and environment should be sanitized, and reducing the relative humidity to less than 20% while increasing the temperature to 40°C (104°F) is of benefit in this regard.[17, 18] In one report,[97] an in-contact guinea pig also developed psoroptic mange.

S. scabiei var. *cuniculi*, a burrowing mite, is a rare ectoparasite on rabbits in North America but is commonly found in some other parts of the world, such as Africa and India.† Typical lesions include tan to yellow, often powdery crusts, alopecia, erythema, and excoriation on the muzzle, the lips, the bridge of the nose, the eyelids, the head, the margins of the pinna, the paws, and the external genitalia. Pruritus is intense. Severe infestations can lead to anorexia, lethargy, emaciation, and death. These mites can transiently produce lesions in humans. Diagnosis is confirmed by finding *S. scabiei* mites in skin scrapings. However, mites are often difficult to demonstrate, and response to therapy is a frequently used diagnostic test. The treatment of choice is ivermectin.[6, 17, 19, 90]

Cheyletiella parasitovorax, a nonburrowing mite, is a common ectoparasite on rabbits.[2–6, 8, 10, 16–19] Most rabbits harbor the mites without overt signs of skin disease. With heavy infestations or in hypersensitive hosts, a variably pruritic dermatosis is seen. Lesions consist of scaling, crusting, and variable degrees of erythema, alopecia, and greasiness over the withers, the back (see Fig. 21–20*E*), and the ventral abdomen. Occasionally, lesions are limited to the face. These mites can produce skin lesions in humans. Diagnosis is confirmed by finding *C. parasitovorax* in skin scrapings or acetate tape preparations. The treatment of choice is ivermectin.

Listrophorus (Leporacarus) gibbus is a common fur mite of rabbits, which is rarely associated with clinical skin disease.[2–6, 8, 10, 16–19, 86a, 93a] Most affected rabbits are asymptomatic. The mite is usually found attached to hair shafts, especially on the back, the groin, and the ventral abdomen. Occasional rabbits may manifest a variably pruritic, scaly, erythematous, alopecic dermatitis in the aforementioned sites. Some animals only manifest pruritus and traumatic alopecia with no skin lesions. Diagnosis is confirmed by finding *L. gibbus* in skin scrapings and acetate tape preparations (see Fig. 21–13*H*). Treatment with pyrethrin- or pyrethroid-containing flea powders, 1% selenium sulfide baths, or 2% lime

*See references 2–6, 8, 10, 11, 16–19, 77, 81, 86, 92, 96.
†See references 2–6, 8, 10, 11, 16–19, 74, 75, 90, 92, 96.

sulfur dips is curative.[17, 86a, 93a] Ivermectin may also be effective.[10] Fipronil spray was reported to be effective.[91] One author reported treating over 50 rabbits with fipronil spray (3 ml/kg) with no adverse effects.[79] However, the therapeutic index for this product is fairly narrow in rabbits, possibly due to the isopropyl alcohol content.[91] Company representatives received a number of reports of suspected adverse reactions to fipronil spray in rabbits, and recommended that the product *not* be used in this species.[78]

Notoedres cati, a burrowing mite, is a rare ectoparasite on rabbits in North America, but is commonly found in other parts of the world, such as India.* Clinical signs, diagnosis, and treatment are identical to those described for *S. scabiei*.

Fleas (especially *C. felis felis*) are occasionally found on rabbits, especially those in households with dogs and cats.[2–6, 8, 10, 16–19, 85a] In the United States, rabbits may also be infested with *Cediopsylla simplex* (common Eastern rabbit flea), especially around the head and the neck, and *Odontopsyllus multispinosis* (giant Eastern rabbit flea), especially over the rump. Clinical signs, diagnosis, and therapy are the same as that described for cats.

The rabbit sucking louse *Haemodipsus ventricosus* is uncommon in the United States.[2–5, 7, 9, 15–19] Pediculosis is usually associated with poor management. Lice are most commonly found on the dorsum and may produce intense pruritus. Severe infestations in debilitated animals may produce anemia, weakness, emaciation, and death. *H. ventricosus* is a vector of tularemia. Therapy is the same as that described for cats.

Demodex cuniculi mites have been isolated from rabbits with generalized pruritus and scaling, but their pathogenic significance is in doubt.[17]

Members of the fly genus *Cuterebra* occasionally produce myiasis in domestic rabbits reared outdoors or in nonscreened enclosures.† Among those fly species reported in the United States are *Cuterebra cuniculi*, *C. buccata*, and *C. horripilum*. Larvae and, therefore, lesions appear in the summer and early fall. The incidence of infestation decreases with age, which correlates with the development of immediate and delayed-type hypersensitivity reactions to larval antigens. *C. horripilum* prefers the ventral cervical region, whereas *C. buccata* larvae localize in the interscapular, axillary, inguinal, or rump area. Initial lesions include subcutaneous cystlike structures. As the larvae (warbles) enlarge, a "breathing hole," or fistula, is produced. The surrounding haircoat is moist and matted, secondary bacterial infection is common, and the lesions are often painful. Treatment consists of surgical removal of the larvae (one should not crush or otherwise damage the larvae), routine wound care, and occasionally, administration of systemic antibiotics. Prevention and control are aimed at eliminating contact with the warble fly.

Flystrike (maggots) is most commonly seen in the perineal region, and may spread dorsally onto the rump.[10] Moist dermatitis and fur matting are present. Rabbits that are sedentary, overweight, or that have perineal dermatitis (urine scald) may be predisposed. Treatment includes cleansing and one injection of ivermectin.[10]

Nutritional Disorders

Nutritional deficiencies are unlikely to be encountered in pet rabbits. Experimental production of nutritional deficiencies with resultant cutaneous abnormalities have been reported and are briefly presented here. Copper deficiency results in alopecia and a depigmented haircoat.[2, 17] Zinc deficiency produces alopecia, scaling, and a depigmented haircoat.[2, 17]

Miscellaneous Conditions

Several days before parturition, the female rabbit undergoes a generalized loosening of the fur.[2, 3, 6, 8, 10, 17] The female rabbit pulls out mouthfuls of hair to line the nest. Hair loss

*See references 2–6, 8, 10, 16–19, 87, 88.
†See references 2–6, 8, 10, 16–19, 92, 96.

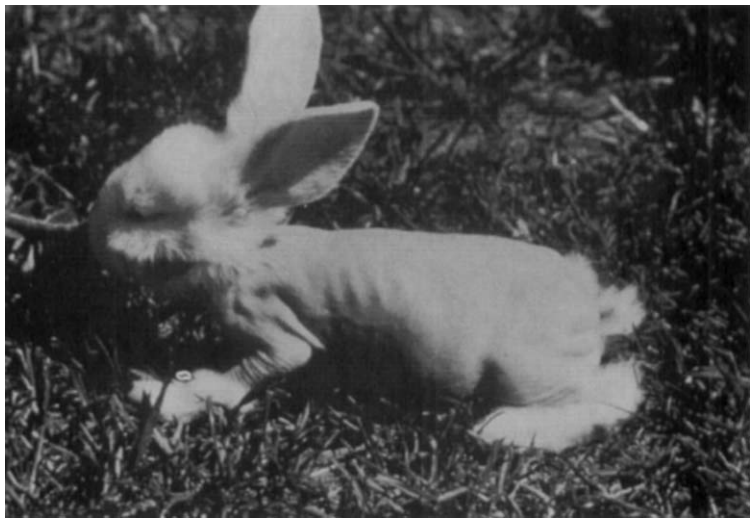

FIGURE 21–21. Congenital alopecia in a rabbit. (Courtesy of J. Gourreau.)

is especially prominent on the abdomen, chest, forelegs, and hips. Some rabbits pull out fur as a behavioral vice.[2, 8] Other rabbits rub fur off against the cage surface or feeders.[8] Does in heat and rabbits on low-fiber diets may barber their own hair.[10] The barbered rabbit typically has patches of broken-off hairs over the head and back.[10] Seasonal molts can result in haircoat irregularities and thinning.[8]

Compulsive self-mutilating behavior was encountered in 5% to 10% of the rabbits in a colony of Checkered crosses.[85] Extensive automutilation of digits and pads of the front feet was observed. The behavior could be interrupted by giving the rabbits haloperidol (0.2 mg/kg IM q12h), a dopamine antagonist. Because the condition was never seen in animals of other breeding lines kept in the same building under identical conditions, and the affected animals came from highly inbred stock, it was hypothesized that the disorder was genetically determined.

Hereditary alopecias (Fig. 21–21) are rarely described in rabbits[17] but are unlikely to be seen by practitioners.

Cutaneous asthenia has been reported in rabbits (Fig. 21–22).[17, 76, 94] The animals had a history of skin fragility and repeated spontaneous skin tears, and were covered with scars. The skin extensibility index (see Chap. 12) in two rabbits was 21% to 32% in the affected rabbits as compared with a mean of 13% in normal rabbits. Light microscopic examination was unremarkable, but electron microscopic examination revealed distorted and tangled collagen bundles with collagen fibrils being of different diameters and having a loose, frayed appearance.

Hutch burn is a contact dermatitis caused by urine scalding of the perineal region because of an unclean environment or an inability of the rabbit to void urine without soiling itself, such as after an orthopedic or neurologic injury, or with obesity.[3, 10] Washing the area frequently with antimicrobial agents and applying a protectant cream, such as zinc oxide, are helpful.

Frostbite may be seen in rabbits that are suddenly exposed to cold climates without a period of acclimatization.[17] Erythema, acrocyanosis, necrosis, and sloughing are typically seen on the pinna (see Fig. 21–20*F*).

Both male and female rabbits possess two sebaceous scent glands on either side of the vulvar or testicular area that secrete a brown waxy debris.[3] This secretion can build up and can be easily removed by gentle traction or soap and water.

The authors have seen a condition resembling *alopecia areata* in rabbits. Affected animals presented with one or more areas of noninflammatory annular alopecia, especially

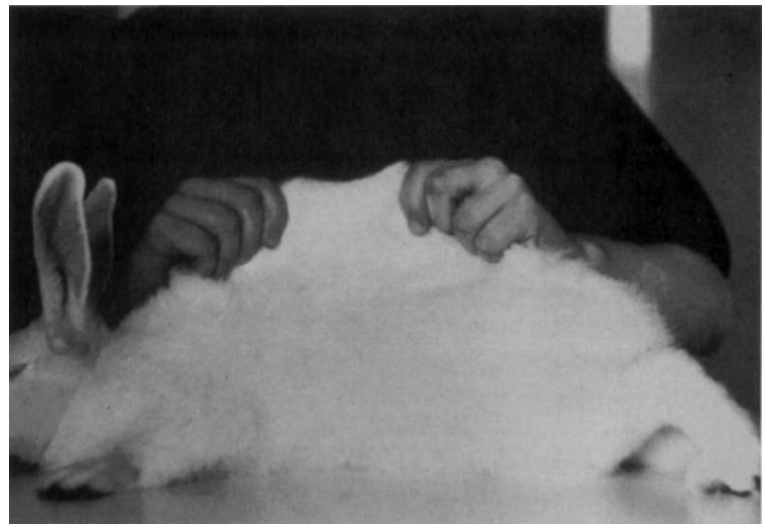

FIGURE 21–22. Hyperextensibility of the skin in a rabbit with cutaneous asthenia. (Courtesy of R. Harvey.)

on the black-furred areas of the pinnae. Spontaneous recovery was accompanied by the regrowth of white fur (Fig. 21–23).

Sebaceous adenitis was reported in domestic rabbits.[89, 96a] The animals varied from 2½ to 6 years of age. Lesions began around the neck or face, remained localized for several months, then became generalized. All rabbits eventually had a generalized, nonpruritic exfoliative dermatosis with patchy to coalescing areas of alopecia. Skin biopsies were diagnostic. No response was seen to antibiotics, glucocorticoids, ivermectin, griseofulvin, fatty acids, azathioprine, or oral retinoids.

Facial eczema has been reported in young suckling rabbits.[11] The condition is sporadic and of unknown etiology. Areas of alopecia and slight erythema occur on the bridge of the nose and the periocular region. Affected animals are otherwise healthy. The condition responds rapidly to topical glucocorticoid therapy.

Neoplasia

Spontaneous nonviral cutaneous neoplasms are rare in rabbits.* Papilloma, basal cell carcinoma, squamous cell carcinoma, sebaceous carcinoma, melanoma, osteosarcoma, and lymphoma have been reported.

Shope papillomas are uncommon in domestic rabbits.† In the United States, the disease occurs in the Southwest and along the Mississippi River. Shope papilloma virus (a papovavirus) commonly infects wild rabbits, with insects serving as vectors. Lesions are characterized by multiple hornlike growths from a single site, especially about the eyelids and the pinnae. Removal of the papillomas usually results in healing, and recovered rabbits are resistant to reinfection. Spontaneous regression of lesions occurs within 12 months. Experimental infection of domestic rabbits resulted in malignant transformation to squamous cell carcinoma within 8 to 9 months in a high percentage of the inoculation sites. A program of screening animal enclosures and vector control should be instituted in endemic areas.

Shope fibromas are uncommon in domestic rabbits.‡ Shope fibroma virus (a poxvirus) commonly infects wild rabbits in North and South America and is transmitted via insect vectors. Lesions consist of single or multiple flat, firm, subcutaneous nodules, especially on

*See references 2–6, 8, 10, 16–19, 84, 92, 96.
†See references 2, 3, 6, 8, 10, 17, 92, 96.
‡See references 2, 3, 6, 8, 17, 92, 96.

FIGURE 21–23. Alopecia areata–like condition in a rabbit. Well-circumscribed areas of noninflammatory alopecia on the pinna are regrowing white hair.

the genitals, the perineum, the ventral abdomen, the paw, the nose, the pinna, and the eyelid. Newborn rabbits are more susceptible than are older animals and have more extensive lesions. Experimentally infected adult rabbits often show spontaneous involution of their fibromas within 5 months through necrosis and sloughing. Mosquito eradication and enclosure screening is indicated to prevent infection in endemic areas.

Cutaneous lymphoma was reported in domestic rabbits from 7 weeks to 9 1/2 years old.[96b] Most had early internal organ involvement and systemic disease. Lesions consisted of multifocal areas of alopecia, scale, erythema, and plaques. Histologically, the lymphomas were epitheliotropic and $CD3^+$. One rabbit was unsuccessfully treated with oral isotretinoin and interferon-α.

• RAT

Bacterial Infections

Bacterial skin infections are uncommon in pet rats, are usually caused by *S. aureus,* and are secondary to trauma (cage-related injuries and bite wounds) or self-inflicted (the pruritus associated with ectoparasites).[2–8, 10, 16–19] Rats are more resistant to experimental wound infection with *S. aureus* than are mice or hamsters.[17] Infections resulting from cage-related injury are typically seen on the nose and the muzzle, whereas those associated with bite wounds typically occur around the head, the tail, the rump, and the perineal area. Staphylococcal dermatitis may be superficial (alopecia, erythema, oozing, and crust) or deep (abscess, fistula, necrosis, and ulcer) and is usually nonpruritic. Granulomas occurred on the trunk and mammary gland in association with infection by an atypical slow-growing *S. aureus.*[100] Other bacteria occasionally isolated from cutaneous abscesses and pyogranulomas in rats include *Streptococcus* sp., *P. pneumotropica, Klebsiella pneumoniae, P. aeruginosa,* and *Mycobacterium lepraemurium* (rat leprosy).[2, 8, 16–19]

Treatment of bacterial dermatitis includes some combination of elimination of predisposing causes, surgical drainage, daily topical cleaning with 3% hydrogen peroxide or 0.5% to 1% chlorhexidine, and systemic antibiotic administration (see Table 21–1).

S. moniliformis is a rare cause of epizootics of edema and cyanosis of the extremities.[2, 8, 17]

C. kutscheri (murium) is a rare cause of epizootics of furunculosis and cutaneous pyogranulomas, which may progress to necrosis and sloughing of extremities.[2, 8]

Fungal Infections

Dermatophytosis is rare in rats and is usually associated with *T. mentagrophytes*.[1–6, 8, 10, 16–19] This dermatophyte can be isolated from the haircoat of clinically normal rats and is a potential zoonotic agent. Lesions are most commonly seen on the neck, the back, and the base of the tail and consist of annular areas of alopecia, broken hairs, scales, and variable degrees of erythema and crusting. Pruritus is usually minimal to absent. Diagnosis and treatment are as described for guinea pigs.

Viral Infections

Sialodacryoadenitis virus (a coronavirus) infection causes eye rubbing and scratching, periorbital swelling, and red tears (chromodacryorrhea) (see Fig. 21–20*G*).[2, 4, 17]

Poxvirus infection has been described in laboratory white rats.[17] Skin lesions consisted of erythematous papules, which became crusted and occurred mainly on the glabrous areas of the body (tail, paws, and muzzle). Sometimes, the affected portions of paws and tail underwent necrosis and sloughing. Diagnosis was confirmed by biopsy, electron microscopy, and viral isolation.

Ectoparasites

N. muris occasionally causes a severely pruritic dermatitis in rats.[2–6, 8, 10, 16–19] Lesions are most commonly present on the pinnae, the nose, the paws, and the ventrum and consist of erythema, papules, yellowish hyperkeratotic crusts, and excoriations. Diagnosis is confirmed by skin scrapings. Treatment is accomplished with topical 2% lime sulfur dips (once weekly until 2 weeks after cure) or subcutaneous injections of ivermectin.

Other mites that are rarely found on rats include *Radfordia ensifera, O. bacoti* (tropical rat mite), *S. scabiei, Trixacarus diversus* (Fig. 21–20*H*), *T. caviae, M. musculi,* and *Demodex* sp.*

Fleas (especially *C. felis felis)* may be recovered from pet rats maintained in households frequented by cats and dogs.[2–6, 8, 17]

The sucking louse *Polyplax spinulosa* is occasionally found on pet rats.† Some animals may be asymptomatic carriers, and other animals manifest varying degrees of dermatitis and pruritus. Young animals, debilitated animals, and animals in poor management situations are more likely to be affected. Lice and related dermatoses are most commonly found on the neck and back. Treatment can be accomplished with topical insecticides or ivermectin injections.

Nutritional Disorders

Nutritional deficiencies are unlikely to be encountered in pet rats. Experimental production of nutritional deficiencies with resultant cutaneous abnormalities has been reported, and these are briefly mentioned here. Zinc deficiency produces exfoliative dermatitis, alopecia, and depigmentation of the haircoat.[2, 17] Pantothenic acid deficiency results in exfoliative dermatitis, depigmentation of the haircoat, and excessive harderian gland activity with increased porphyrin secretion resulting in red tears and blood-caked whiskers.[2, 17] Riboflavin deficiency produces alopecia, scaling, and dermatitis, especially on the extremities.[2] Pyridoxine deficiency results in exfoliative dermatitis, especially on the face, the ears, the limbs, and the tail.[2, 17] Biotin deficiency causes exfoliative dermatitis and periocular alopecia.[2, 17] Niacin deficiency causes alopecia and excessive harderian gland activity, increased porphyrin secretion, and blood-caked whiskers.[2, 17] Essential fatty acid deficiency

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*See references 2, 3, 6, 8, 10, 16–19, 99, 101.
†See references 2, 3, 7, 8, 10, 16–19.

produces an exfoliative dermatitis and, occasionally, necrosis of the tail.[2, 17] Protein deficiency causes alopecia, exfoliative dermatitis, and depigmentation of the haircoat.[2, 17]

Miscellaneous Conditions

Barbering and bite wounds are frequently seen when rats are housed together.[2–6, 8, 17] These behaviors can be exacerbated by crowding, stress, and boredom. Areas most commonly affected include the muzzle, the whiskers, the face, the head, the rump, the tail, and the perineum. Rats may also rub the hair off the muzzle as they stick their face through slotted feeders or wire bars.[17]

Rats have numerous types of hereditary hairlessness that are probably never seen by the practitioner (Fig. 21–24).[17]

Ringtail is a poorly understood condition seen in rats.[2–8, 17] The incidence of the disorder increases as the relative humidity falls below 40% and is especially common in young unweaned animals housed in cages with wire mesh bottoms, on hygroscopic bedding, and in rooms with excessive ventilation. In the northern hemisphere, most cases are seen from November to May, when heating systems often cause marked reductions in relative humidity. Some strains of rats seem more susceptible than others. The condition usually occurs after 2 months of reduced relative humidity. One or more annular constrictions develop in the tail, which becomes edematous, inflamed, and necrosed distal to the constrictions (Fig. 21–25). Ringtail is prevented by maintaining a relative humidity of at least 50%.

Perianal pruritus is seen in association with pinworms (*Syphacia muris*).[2, 4, 8, 17] Infected rats occasionally mutilate the base of the tail. Diagnosis is confirmed by microscopic examination of strips of cellophane (Scotch) tape that have been applied to the perineum. The eggs of *S. muris* are banana shaped and about 30 μm by 150 μm. Ivermectin is effective treatment.

Auricular chondritis has been described in rats.[17] The condition has occurred spontaneously and in association with the placement of metal ear tags or immunization with type II collagen. Typically, both ears are affected, although one ear may be affected days to weeks before the other. The pinnae are swollen, erythematous, and nodular, and they become thickened and deformed. Pain and pruritus are rare. Histologically, there is a multifocal granulomatous chondritis with progressive destruction of cartilage.

Systemic hair embolism has been reported subsequent to intravenous injections in rats.[17] Cutaneous lesions consist of focal areas of necrosis and ulceration on the ventral

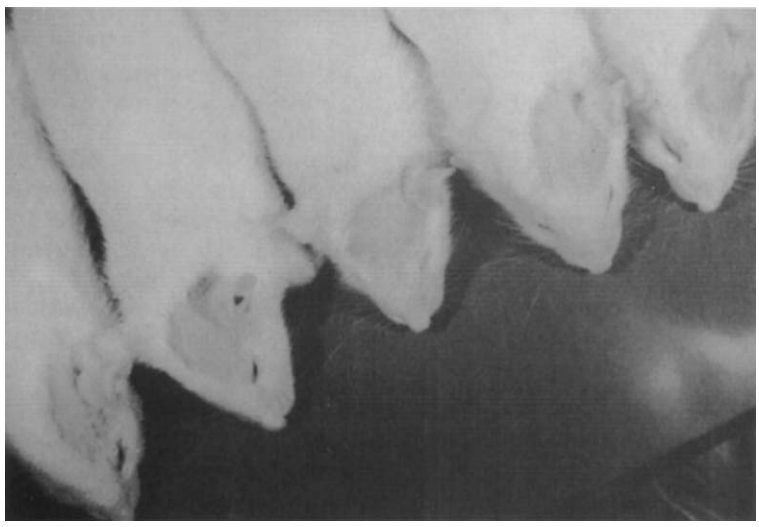

FIGURE 21–24. Congenital alopecia in a litter of rats. (Courtesy of J. King.)

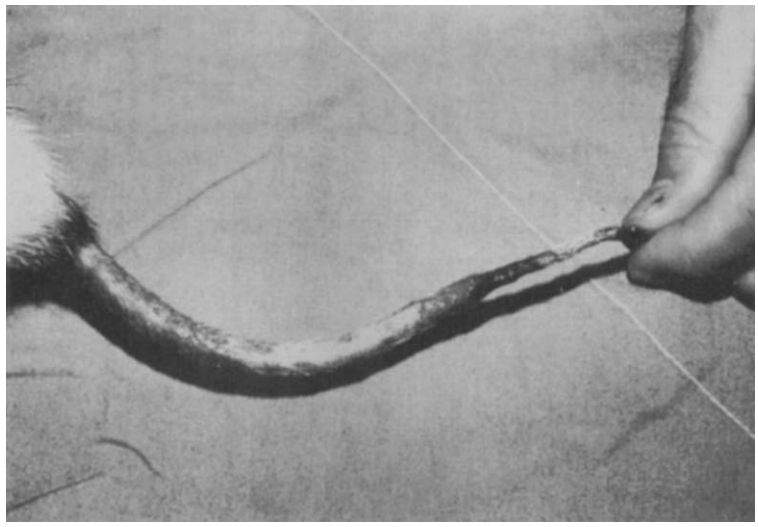

FIGURE 21–25. Ringtail in a rat. Necrosis of the distal portion of the tail. (Courtesy of G. Kollias.)

aspect of the body. Histologic examination reveals granulomatous and necrotizing dermatitis and panniculitis, and intravascular hair shaft fragments.

The fur of the aged rat frequently turns yellow and becomes more coarse.[6] The cause is unknown.

Alopecia areata was reported in DEBR rats and proposed as a useful model of the human disease.[70]

Brownish scales were observed to occur on the skin of rats, mainly on the dorsum and tail and were more numerous in males and with increasing age.[102] The scales also occurred in strain-dependent patterns. Gonadectomy produced a fading of the scales in males, whereas the administration of androgen to gonadectomized males and to females produced a darkening of the scales.

Neoplasia

Spontaneous cutaneous neoplasms are uncommon in rats.[2–6, 8, 17, 98] Mesenchymal neoplasms are more common than are epithelial neoplasms. The most common are fibromas, fibrosarcomas, and lipomas. The face, the shoulder, the flank, the tail, and the paws are typically affected. Other reported skin neoplasms in rats include papilloma, keratoacanthoma, sebaceous gland tumors, squamous cell carcinoma, basal cell carcinoma, hair follicle tumors, hemangiosarcoma, melanoma, and malignant fibrous histiocytoma.

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