# Skin Diseases of Exotic Pets

Edited by

**Sue Paterson** MA VetMB DVD Dip ECVD MRCVS

RCVS and European Specialist in Veterinary Dermatology
Rutland House Veterinary Hospital, St Helens UK

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## Dedication

Richard, Sam and Matt XXX

# List of Abbreviations

i.m. = intramuscular
i.v. = intravenous
p.o. = orally
s.c. = subcutaneous

sid = once a day
bid = twice a day
tid = three times a day

The authors and editor have striven to ensure that all drug dosages and usages are correct but that it is the responsibility of every individual veterinary surgeon to act within the laws governing medicine licensing and usage in their own country.

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# SECTION ONE

# Dermatology of Birds

Chapter 1

# Structure and Function of Bird Skin

## DESCRIPTION OF SKIN LAYERS

Avian skin is made up of two main layers (Figures 1.1 and 1.2):

- Epidermis.
- Dermis.

The overall thickness of the skin varies between different areas of the body. In areas that are feathered the skin may only be three or four layers thick (Figure 1.3). This compares with areas such as the feet where there is no feather covering, and the skin is many layers thick (Figure 1.4).

The outer layers of hard keratin structures such as the scales on the feet and spurs do not moult but instead gradually wear down. The outer surface of soft keratin structures such as the skin, comb and wattles slough the outer layer. This usually takes place at the same time as a feather moult (see p. 12). The exact pattern of feather loss will depend on the species and breed of bird.

### *EPIDERMIS*

- Fewer layers than its mammalian counterpart.
- Lowest layer is the basal membrane.
- *Stratum germinativum* produces the cells which will mature and from the outer *stratum corneum*. Within the *stratum germinativum* it is possible to subdivide cells into three distinct layers:
  - *stratum basale* (just above the basal membrane).
  - *stratum intermedium* whose cells are larger than the basal layer, held together by desmosomes and polygonal in shape.
  - *stratum transitivum* where cells are well developed and show signs of keratinisation, although keratin granules within cells are not as obvious in avian skin when using light microscopy as they are in mammalian skin.
- S*tratum corneum* contains vacuolated and flattened cells which will become either hard or soft keratin – the soft keratin being sloughed and the hard keratin retained.

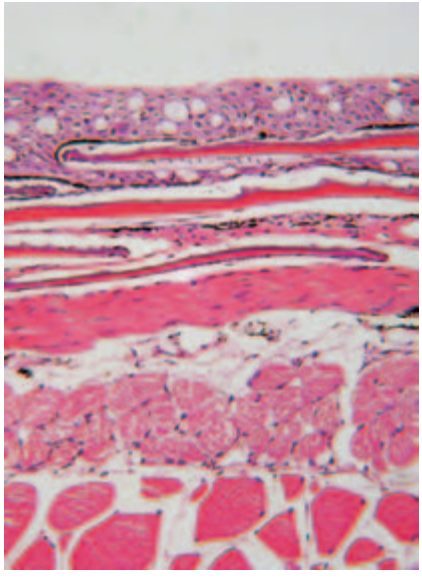

**Fig. 1.1** Histopathological section through avian skin.

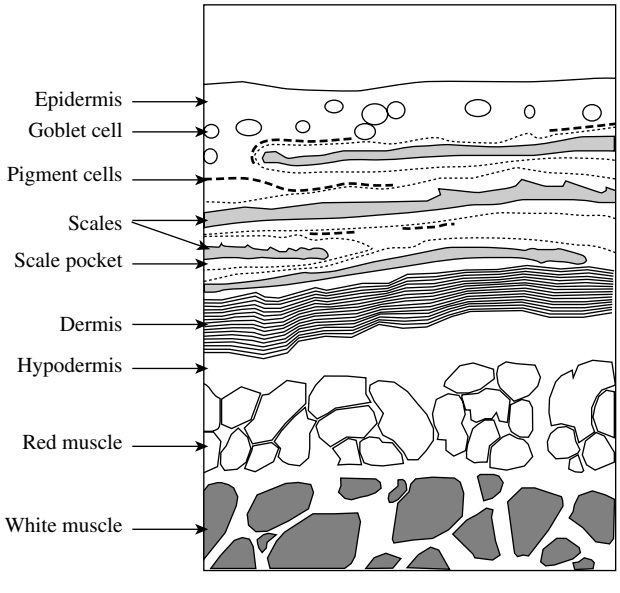


**Fig. 1.2** Schematic diagram of Fig. 1.1.

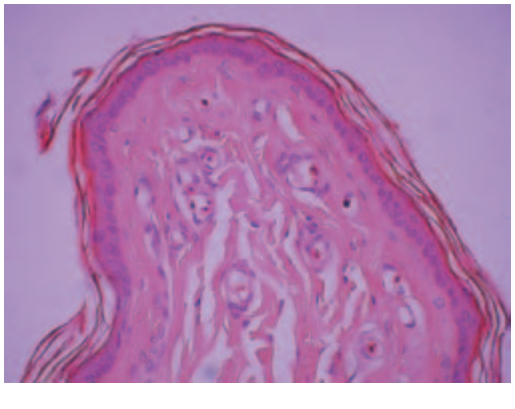

**Fig. 1.3** Feathered skin from chicken. (Picture courtesy of C. Knott MRCVS)

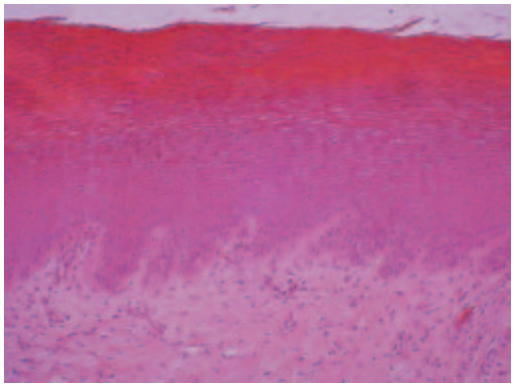

**Fig. 1.4** Unfeathered skin from chicken leg. (Picture courtesy of C. Knott MRCVS)

## *DERMIS*

- Divided into superficial and deep layers.
  - Deep dermis can be further described as compact or loose. The loose layer is the deepest and in addition to being attached to the underlying structures it contains apterial muscles and areas of fat.
- Throughout the dermis are elastin fibres which form tendons. These tendons are attached to the muscles of the feathers and are responsible for feather movement.

## *SUB-DERMAL STRUCTURES*

- Beneath the dermis is elastic tissue and connective tissue, which allows both movement and attachment of underlying structures.

## *OTHER STRUCTURES*

- There are no sweat glands in avian skin and therefore birds are prone to hyperthermia. The only areas where glands are found in avian skin are the uropygial (or preen) glands, the pericloacal glands (which secrete mucus) and the glands of the ear canal. However, as epidermal cells contain lipid material the skin itself can be described as a secretory organ.

# SPECIALISED STRUCTURES

## *COMB*

The tissue of the comb is extremely well supplied with blood vessels.
Anatomically the comb can be divided into:

- Base (where it attaches to the head).
- Body (the central part).
- Points (the dorsal projections).
- Blade (the posterior part).

Combs are not present in all species of bird but are commonly found in chickens.

## *WATTLE*

The wattle is found under the jaw of many species of domestic poultry and is made up of:

- Thick epidermis.
- Dermis rich in blood vessels.
- Sinus capillaries.

## *FRONTAL PROCESS / SNOOD*

This structure is found in turkeys dorsal to the nasal region. Due to the rich blood supply it can increase in length quite dramatically and is used as part of a courtship display.

## *CARUNCLES*

These are the multiple skin protruberances which are found on the head and upper neck of the turkey.

## *CERE*

The cere is found in some species at the base of the upper beak.

- It is made up of layers of keratinised epithelial cells.
- Colouration of the cere can be used to sex some species (especially budgerigars).
- Disease can cause alterations in the normal colour of the cere.
- The cere is well supplied with sensory nerve endings from the trigeminal nerve.

## *BROOD PATCH*

This is an area in the breast region of some species (in both male and female birds). At this site the:

- Dermis is thickened.
- Dermis is highly vascularised.
- The feathering is looser than other parts of the body.
- When brooding, this area receives a large blood supply allowing heat to be transferred to the incubating eggs.

## *FEET*

The skin of the feet usually does not have any feather covering, although feathers are present in some species.

In most species, the epidermis is thickened into scales to provide protection to the feet.

- **Scutes** are large scales, which in chickens are found on the anterior surface of the metatarsus and the dorsal surface of the toes.
- **Scutella** are smaller scutes found on the caudal surface of the metatarsus in chickens.
- **Reticula** are the smallest distinct scales.
- **Cancella** are minute scales found between the reticula.

## *CLAWS*

Claws are of course present on the feet (Figure 1.5) but are also present on the wings of some species such as ostriches and rheas and are anatomically similar to the nails of cats and dogs.

**Fig. 1.5** Claws of Barn Owl.

**Fig. 1.6** Beak of a Hobby.

## *BEAK* (Figure 1.6)

The upper and lower beaks consist of a bony structure covered in a keratinised, horny material known as the rhamphotheca.

- Rhamphotheca is the keratinised material equivalent to a thick stratum corneum which contains calcium phosphate and hydroxyapatite to give it strength.
- Dermis lies underneath the rhamphotheca. This is well vascularised and attached to the periosteum of underlying bone.
- A large number of sensory endings are present in the beak especially from the trigeminal nerve.

## *UROPYGIAL GLAND / SKIN SECRETIONS*

The uropygial gland is a bilobed holocrine gland found at the base of the tail.

- It is not present in all birds (e.g. emus, some parrots and bustards do not posses a uropygial gland).
- Absence of this gland does not impair waterproofing of the feathers.
- The gland is made up of two lobes which open to outside via the uropygial duct. In most species this is a single slit, but there can be up to eight orifices. The papilla is not usually covered in feathers although a small number of down feathers can be found at the tip of the papilla (known as the uropygial circlet or tuft).
- The uropygial gland secretes a lipoid sebaceous material which is thought to be important in protecting and waterproofing feathers. It has also been suggested that these secretions may be a source of vitamin D precursors, which inhibit bacterial and fungal growth, maintain the moisture content of the skin and maintain the pliability of feathers.
- Preening is thought important in the distribution of these secretions through the feathers. Preening is also necessary for interlocking the barbules of the feathers and thus providing waterproofing.

- The cells of the epidermis also contain sebaceous material. Although avian skin does not contain glands which secrete sebaceous material the skin itself can act as a source of these secretions.

## *PATAGIA*

These are flat, membrane-like structures which are found where the wings, neck, legs and tail join the body.

- They are always present irrespective of the position of the animal. Compare this with webs which are areas of skin that may be present when the wings or legs are in certain positions.
- Patagia are important as they are often areas affected by cutaneous ulcerative dermatitis.

## *FEATHERS*

- There is a pattern to the arrangement of feathers such that feathers are arranged into tracts known as **pterylae**.
- The areas of skin between these feather tracts are known as **apteria**.
- Different species and breeds within species have particular feather tracts and names have been given to specific feather tracts in different areas of the body (See Lucas and Stettenheim (1972), for further information).

### *FEATHER TYPES*

- **Natal down** is the initial feather covering and is made up of down feathers. This is usually present at the time of hatching after which the down feathers are pushed out by the juvenile feathers.
- **Juvenile feathers** have a normal feather appearance, although they are smaller and narrower than the adult feathers.
- **Feather sheath** covers the feathers as they grow from the feather follicle, these feathers are called pin feathers. This sheath should rupture and release the barbs.
- **Adult feathers** appear at the third moult and can then be divided into different types dependent on their structure, function and location on the bird.
  - Contour feathers are the predominant feather and are the main feathers present on the wings and body. They are present in feather tracts (**pterylae**), separated by featherless areas (**apteriae**).
  - Flight feathers of the wings are known as **remiges** and these can be divided into primary remiges (on the manus) and secondary on the antebrachium (Figure 1.7). Flight feathers of the tail are known as **rectrices** (Figure 1.8). Feathers that cover the bases of the remiges and rectrices are known as **coverts**.

**Fig. 1.7** Flight feathers on the wing of a Grey Heron.

**Fig. 1.8** Rectrices in a Barn Owl.

## *FEATHER STRUCTURE*

The contour feather is described and other feather types are compared to this. Feathers are highly developed in comparison to hairs.

- Feathers grow from a follicle in the dermis (Figure 1.9). The feather follicle has many similarities to the mammalian hair follicle in its structure. At the point where the feather attaches to the follicle is a dermal papilla which projects up into the base of the feather from which the growing feather receives a blood and nerve supply.
- **Herbst's corpuscles** are found at the base of the follicle; these detect vibrations. Also at the base of the feather follicle is a smooth muscle which elevates the feathers to increase insulation.
- **Calamus** is the part of the feather that attaches to the follicle. In the growing feather the calamus contains mesoderm and an axial artery and vein. As the feather matures, this tissue and blood vessels degenerate so that the calamus becomes hollow. However, partitions, called **pulp caps**, remain within the calamus dividing it into sections. The dermal papilla of the follicle projects into the tip of the calamus known as the **inferior umbilicus**.

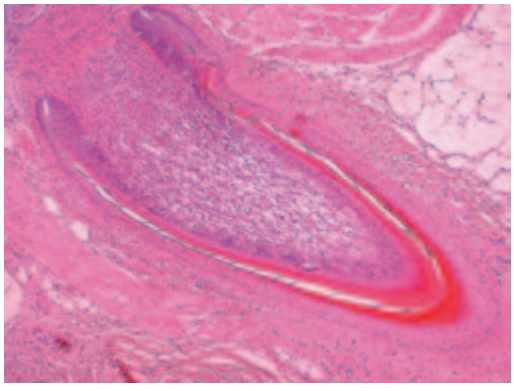

**Fig. 1.9** Histopathology of a chicken feather follicle.

- **Rachis** is the main shaft of the feather. Where the rachis meets the calamus is a pulp cap known as the **superior umbilicus**. There may be a smaller feather attached to the superior umbilicus known as the **after feather**. On either side of the rachis are projections known as **barbs**, which themselves bear projections known as **barbules**. Most but not all of these barbules bear hooks known as **barbicels** which hold the barbs and barbules together. The combination of barbs and barbules on one side of the rachis is known as a **vane**.

Physical features of different feather types:

| | |
|---|---|
| **Down** (plumules) | Fine feathers that do not have barbules on the barbs. |
| **Filoplume** | Found close to the follicle of each contour feather. They have a long shaft with a tuft of barbs or barbules at the distal end. The follicles of filoplumes are highly innervated. |
| **Bristle** | These feathers only have a few or no barbs and have a very stiff rachis. They are found at the base of the beak and around the eyes and are surrounded by sensory corpuscles. |
| **Powder** | These feathers shed fine granules of keratin which are important in waterproofing the feathers. |
| **Semiplume** | These feathers have a large rachis with a fluffy vane (Figure 1.10). They are present underneath the contour feathers and are important for insulation. |

## *COLOURATION OF SKIN AND FEATHERS*

The colour of the skin and feathers depends on the pigments that are deposited during development and the structure of the feather, as this controls the absorption and reflection of light.

**Fig. 1.10** Semi-plume with prominent after feathers.

- **Melanocytes** produce brown, yellow and black melanin and are found in both feathers and the epidermis.
- **Carotenoids** and **xanthophils** produce red and yellow pigments and are obtained from the diet and deposited in the feather follicle and possibly in the secretions of the preen gland.
- Other pigments such as **porphyrins** and **schemochromes** also contribute to the final colour of the bird.
- **Uropygial gland pigments** can contribute to feather colour. Lipids from this gland can affect light reflection and give feathers an iridescent glow.

## *MOULTING*

Moulting of feathers takes place in all species of bird. Most species will moult once a year, every year often after the breeding season. However, some species will not undergo an obvious moult but instead lose a small number of feathers throughout the year; other species will only undergo a moult every two years; other species can moult up to three times a year.

Moulting takes place when the growth of a new feather in the feather follicle forces out the older feather. Moulting will usually follow a distinct pattern of feather loss such that:

- Proximal primary feathers are lost first. Feather loss moves distally until around half of the primary feathers have been lost.
- Secondary feathers are then lost distally moving proximally.

- Body feathers are then lost.
- Tail feathers are lost initially from the midline, moving laterally.
- Powder feathers are shed continuously.

Depending on the frequency of moulting it is possible for a bird to be covered in feathers from several different moults. However, if a large number of feathers are lost at one time then it is possible for a bird to be flightless until the new feathers grow in, as occurs in many species of duck.

## *FACTORS INFLUENCING MOULTING*

A number of factors are thought to influence the time of moulting and it is fair to assume that the timing of moulting will depend on the combination of these different factors.

- **Environmental factors** include photoperiod, temperature, nutrition, humidity, and stress.
- **Hormonal triggers** have tended to concentrate on thyroid hormones $T_3$ and $T_4$. $T_4$ stimulates growth of the feather papillae. New feather growth will cause shedding of old feathers and therefore an increase in $T_4$ is associated with feather loss. This compares with an increase in $T_3$ which tends to coincide with the growth of new feathers. Increases of other hormones such as catecholamines and prolactin have also been associated with moulting.

# REFERENCES

Lucas, A.M. and Stettenheim, P.R. (1972) *Avian Anatomy – Integument. Parts I and II, Agriculture Handbook 362.* US Government Printing Office, Washington DC.

Chapter 2

# Examination of Avian Skin and Diagnostic Tests

## INTRODUCTION

Dermatological disease can present in a variety of fashions – abnormal feather growth, feather chewing or loss, inflammation of skin between the feathers, or beak or claw abnormalities.

The basic work up of any avian skin problem is exactly the same as that for a cat or dog. The importance of a detailed history and clinical examination cannot be over stressed. The main areas that should be examined are listed below.

## HISTORY

Points that should be covered when taking a history from the owners include:

- Age and sex of bird.
- What is the main problem?
- Duration of problem?
- Number of birds kept / affected?
- Where was bird bought from?
- How long in owner's possession?
- What is the normal diet of the bird?
- Is the bird eating / drinking normally?
- Is the bird passing normal faeces?
- Any weight loss?
- How often does the bird moult / has a normal moult taken place?
- Is the bird chewing at feathers or affected areas?
- Type of enclosure bird is kept in (Figure 2.1) – inside or outside?
- If indoors, where is the bird kept?
- Is the bird left alone for long periods of time?

## CLINICAL EXAMINATION

Points that should be covered when carrying out a clinical examination.

- Body condition.
- Areas affected.
- Condition of feathers / completed moult.

- General clinical examination.
- Dermatological examination.

**Fig. 2.1** Is the bird kept inside or outside?

We will now examine the specific tests related to dermatological conditions which should be carried out.

## *FEATHER EXAMINATION*

- *Feather abnormalities* may be obvious on a clinical examination of the bird or may be more subtle, and need microscopic evaluation. It is important to be aware of the normal microscopic appearance of the feather and calamus (Figures 2.2, 2.3). Although not all dermatological conditions will affect the feathers, abnormalities or damage to the feathers can be found in many conditions.
- *General appearance of the bird*: it is necessary to assess whether all of the feathers are the normal shape, size and colouration for that species; has a moult taken place and been completed; are feathers damaged? The distribution of lesions should also be noted. For example, if birds have lost feathers over the head, then feather plucking is unlikely to have been the cause.
- *Examination of individual feathers* can then take place. Depending on the condition this will involve examination of a fully grown feather or a pin feather. Feathers may be removed manually from a conscious bird or under sedation. The feather can be examined for the presence of ectoparasites, fret marks or chewed areas with a hand-held magnifier or examined microscopically. If carrying out microscopic examination it is better to cut the feather into sections if you wish to mount the feather.

## *PULP CYTOLOGY*

The contents of the calamus are examined to try and demonstrate infection of the follicle.

- Firstly the skin around the chosen feather is prepared aseptically with chlorhexidine and the feather carefully plucked.

**Fig. 2.2** Normal microscopic appearance of feather.

**Fig. 2.3** Normal microscopic appearance of feather shaft.

- The calamus can then be cut from the rest of the feather and squashed between two glass slides. Alternatively the contents of the calamus can be removed with a sterile scalpel blade.
- Once smeared on a glass slide the material can be air dried and stained with Grams (for bacterial examination) or Diff Quick® (for cytological examination). It is possible to detect bacteria, inflammatory cells, inclusion bodies, yeasts and dermatophytes by this method.
- It is also possible to send the entire calamus to the laboratory for culture and sensitivity.

## *FEATHER DIGEST*

Quill mites can be difficult to find on pulp examination.

- To improve mite identification the calamus can be placed into 10% potassium hydroxide, gently heated and then centrifuged, in this way mites can often be visualised more easily. Any mites that are present should be seen in microscopic examination of the sediment.

## *SAMPLING OF LESIONS FOR CULTURE AND SENSITIVITY*

- All dermatological lesions can be sampled for bacterial culture and sensitivity.
- To avoid contamination it is preferable to take feather pluckings or biopsies of infected tissue rather than surface swabs. However, in conditions such as Cutaneous Ulcerative Disease where the affected tissue is easily sampled, swabbing the skin with a swab dipped in sterile saline can be used to detect surface bacteria and / or yeasts.

## *TAPE STRIP EXAMINATION*

- This technique can be used to detect ectoparasites, yeast and bacterial infections. Care should be taken to differentiate pathogens from contaminants.
- Acetate tape is pressed on to the skin and then stained with Diff Quik®. Avian skin is much thinner than that of cats or dogs and therefore it is possible to damage the skin if too much pressure is applied.

## *IMPRESSION SMEARS*

- Impression smears of moist lesions can be a quick way to detect surface flora. However, as for tape strips, they are also an ideal way of observing contaminants. They are a useful way of sampling lesions such as Cutaneous Ulcerative Disease.

## *BIOPSY*

- A general anaesthetic is generally required.
- The skin should not be scrubbed prior to sampling, as this will remove surface cells and vital information.
- Avian skin is much thinner than cat and dog skin. This author prefers to use a scalpel blade rather than a biopsy punch in order to take longer sections of skin. If a biopsy punch is to be used, acetate tape placed over the biopsy site can maintain the structure of the sample and the surrounding tissue (Nett *et al*., 2003). Due to the anatomy of avian skin, and the network of tendons that runs through the skin, if a sample is removed without placing tape on the skin, then a large hole can result due to retraction of the surrounding tissue. It is important to take a section of abnormal tissue as well as some adjacent normal tissue.
- The skin should be placed on to card before being placed in formalin to prevent curling of the skin edges. It is preferable to include a feather follicle in the biopsy.

**Fig. 2.4** Intradermal allergy testing in a bird.

## *INTRADERMAL SKIN TESTING*

- Whether birds are affected by allergic skin disease or not is much debated. It is possible to carry out intradermal skin testing in larger birds, but even in species such as parrots the skin is very thin and a 27 G needle is required – even so it takes a great deal of patience to avoid placing subcutaneous injections (Figure 2.4).
- Research by Columbini *et al*. (2000) has shown that histamine is not a good positive control in birds, codeine phosphate at a concentration of 1 : 100 000 w/v should be used instead.
- Interpretation can be difficult. Due to the thin nature of the skin the inflammatory response to an offending allergen is very mild and it can be very difficult to determine whether a positive reaction is present or not.

## *BLOOD SAMPLING*

It is important to take blood samples as part of a complete clinical work up. Dermatological problem conditions such as liver or kidney disease can present as a bird which is feather plucking or chewing feathers over areas of pain, such as is seen in cases of gout.

- Blood samples can be obtained from different blood vessels depending on the species. In general up to 1% of body weight can be taken, which is not very much when dealing with a Budgerigar. Again, dependent on the species the individual may need to be anaesthetised before a sample can be taken.
- The right jugular is the vessel of choice using a 25 G needle (Figure 2.5). The medial tarsal vein can be used in Galliformes and Anseriformes and the basilic vein can be used in raptors and Columbiformes (Figure 2.6). Some texts also describe using a toe nail clip to obtain blood from smaller birds but this should be avoided due to the difficulties in stemming blood flow and the poor blood samples that are obtained.

**Fig. 2.5** Site for right jugular venepuncture.

**Fig. 2.6** Site for basilic and ulna vein venepuncture.

- Due to the fragility of avian blood cells, a smear should be made at the time of sampling and sent to the laboratory along with the blood sample. Samples for haematology should usually be placed into EDTA and samples for biochemistry placed into heparin, although there are some species which require haematology samples to be placed in heparin. Communication with individual laboratories will determine how samples should be preserved.
- It may also be necessary to carry out specific tests such as lead or zinc levels, or hormone assays.

Some of the more common parameters that should be measured are given in Table 2.1.

**Table 2.1** Some of the main biochemical parameters in blood sampling.

| Parameter | Indications |
|---|---|
| Bile acids | Indicates liver function. |
| AST, Lactate dehydrogenase, Creatinine phosphokinase | Indicators of liver and muscle damage. AST found in liver and muscle whereas CPK is only found in muscle. |
| Uric acid | Kidney function. |
| Calcium and phosphorus | Kidney function, nutritional deficiency or hypocalcaemic syndrome of African Grey parrots. |
| Total proteins | Nutrition and liver function. |

## *DIAGNOSTIC IMAGING*

- Skin disease may be the presenting sign of a generalised problem. Radiography can be important to diagnose conditions such as aspergillosis and proventricular dilatation disease which can cause feather plucking.

## *SEXING / COELOSCOPY*

- Endoscopic examination can reveal the sex of the bird (useful if dealing with a broody female with resultant feather problems) and abnormalities such as aspergillomas.

## *EXAMINATION OF GASTRO-INTESTINAL CONTENTS*

- Crop washes can be used to examine the contents of the anterior and posterior gut. Washes or swabs can identify *Trichomonas* or *Candida* which can be present in birds which pull at feathers over the crop area.
- Examination of faecal smears can show normal and abnormal gut flora. Large numbers of Gram-negative bacteria or budding yeasts are considered significant and suggest a condition which requires treatment.
- Collection of faecal samples and direct examination or faecal flotation / sedimentation can be used to identify parasites. For example, nematodes such as *Ascaris platycerca* or intestinal fluke can present as birds which feather pluck.

## *ELECTRON MICROSCOPY OF FEATHER SECTIONS*

- A slightly less common diagnostic technique is examination of feathers with an electron microscope. This can allow examination of the calamus to identify intraluminal mites, although the true significance of the presence of these mites is debatable.

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Chapter 3

# Skin Diseases and Treatment of Caged Birds

## ECTOPARASITES

### *MITES*

The most important ectoparasites of caged birds are the mites. These can be divided into:

- Skin mites.
  - Burrowing mite – Cnemidocoptidae (*C. pilae*), Epidermoptidae.
  - Surface mite – Dermanyssidae (*D. gallinae*), Macronyssidae (*Ornithonyssus* spp.).
- Feather mites.
- Quill mites.

### *SKIN MITES*

### *Burrowing mites – Cnemidocoptidae*

#### Scaly beak / tassle foot mites (Cnemidocoptes spp.)

*Cause and pathogenesis*

Scaly beak is common in Budgerigars (*Melopsittacus undulatus*) where the cere is affected initially by the mite *Cnemidocoptes pilae* from the Cnemidocoptidae family. The mite principally infests the feather follicles and stratum corneum of the face and legs.

The mite usually infects juveniles shortly after hatching and remains latent until later life, however it may spread between adult birds. Its lifecycle lasts for three weeks, and is entirely spent on the host.

*Clinical signs*

This mite causes an increased volume of crusting, often honeycombed by small holes (Figures 3.1 and 3.2). A case in a Red-fronted Parakeet showed signs of general feather loss. Other forms of *Cnemidocoptes* spp. may cause hyperkeratotic lesions of the feet and legs in Passeriformes such as finches. Clinically, disease is thought to occur as a result of immunosuppression, inadequate nutrition (such as hypovitaminosis A) or concurrent infectious disease.

*Diagnosis and therapy*

- Diagnosis may be made by visualisation of the mite in skin scrapings as a typical round parasite with short conical legs barely projecting beyond the circular body outline. Mites can also occasionally be identified on histopathological sections (Figure 3.3).

**Fig. 3.1** *Cnemidocoptes pilae* affecting the face. (Picture courtesy J.D. Littlewood.)

**Fig. 3.2** *Cnemidocoptes pilae* affecting the feet. (Picture courtesy J.D. Littlewood.)

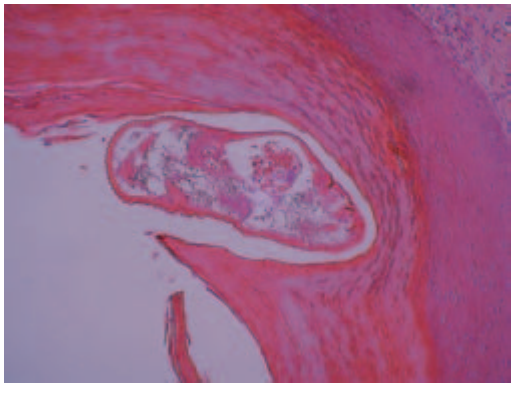

**Fig. 3.3** Histopathological section of scaly leg in a chicken showing section through mite. (Picture courtesy C. Knott.)

**Fig. 3.4** *Dermanyssus gallinae.*

ciated with its blood-sucking capabilities, such as anaemia, and over-preening due to skin irritation.

*Diagnosis and therapy*

Diagnosis may be difficult due to the off-host nature of this mite. Mites can be identified with oval body shape and long legs (Figure 3.4).

Treatment is based on therapy of the environment with permethrin / pyriproxyfen sprays (Indorex®, Virbac), and rigorous cleaning of the cage and local environment.

## *Surface mite – Macronyssidae*

### *Ornithonyssus* spp.

*Cause and pathogenesis*

This is a rare blood-sucking ectoparasite on caged birds. Small rodents can act as incidental hosts. Remains on the host throughout its life cycle.

*Clinical signs*

Feathers become matted often with a grey-black discolouration. Skin may become thickened and scaly. Anaemia can occur due to parasite feeding activity.

*Diagnosis and therapy*

Diagnosis is made by skin scrapes of lesional areas. The mite has an oval body and long protruding legs.

Treatment may be successful with ivermectin.

- Treatment is based on topical ivermectin 0.2 mg/kg diluted with propylene glycol at 1: 10 applied once weekly for three applications or subcutaneous ivermectin at 0.2 mg/kg once, and repeated after seven to ten days on three to six occasions. Additional problems such as hypovitaminosis A should also be corrected.

## *Burrowing mites – Epidermoptidae*

### Bird ked mites (Depluming mites, Epidermoptidae)

*Cause and pathogenesis*

These infestations are relatively uncommon in cage birds. Mites burrow through stratum corneum and can be found in sinus burrows. Cases have been reported in a Grey Cheeked Parakeet (*Brotogeris pyrrhoptera*) due to a *Myialges* mange mite and in a Green-winged macaw (*Ara chloroptera*) due to an unidentified mite.

*Clinical signs*

Usually causes signs of crusting and scaling with secondary self-inflicted trauma on the body and head. May cause flaking skin, pruritus and feather loss over the wings and legs.

*Diagnosis and therapy*

Diagnosis is made by skin scrapings which are necessary to identify the mites which are oval shaped. In many forms the legs end in a claw-like process, eggs are kidney shaped.

Therapy of the case in the Green-winged macaw above was unresponsive to ivermectin (Reavill *et al.* 1990).

## *Surface mite – Dermanyssidae*

### Red Mite (Dermanyssus gallinae)

*Cause and pathogenesis*

This blood-sucking mite spends most of its life off the host hiding in cracks and crevices in the host's environment. It is normally only found on the host at night when it feeds.

*Clinical signs*

Skin lesions are rare, but can cause a papular eruption. It has been associated with foot irritation in Canaries (Lupu 1992), however its more common effects are asso-

# *FEATHER AND QUILL MITES*

## *FEATHER MITES*

### *Cause and pathogenesis*

Feather mites such as *Protolichus lunula* live between the feather barbs on the wings and tail feathers and *Dubininia melopsittaci* lives on the body of Budgerigars. Mites are usually asymptomatic unless present in large numbers or in an immunosuppressed host.

### *Clinical signs*

When large numbers of mites are present the feather may develop a grey / brown tinge and birds may self traumatise.

### *Diagnosis and therapy*

Diagnosis can be achieved by direct microscopic examination of feather. Treatment is not usually necessary but can be performed with permethrins (e.g. Harker's Louse Powder®, Harkers) or piperonyl butoxide / permethrin (e.g. Ridmite Powder®, Johnson).

## *QUILL MITES*

### *Cause and pathogenesis*

Quill mites (Syringophilidae) feed on the tissue fluid of the feather follicle. Their feeding activity may cause feathers to become brittle. Quill wall mites (Laminosioptidae and Fainocoptinae) feed on the outside of the quill causing hyperkeratosis of the quill sheath.

### *Clinical signs*

Most of these parasites are present in small numbers and do not cause disease. However in debilitated birds, or in large infestations, clinical signs may be seen. Birds exhibit evidence of pruritus and will pick out the infested feathers.

### *Diagnosis and therapy*

Diagnosis is achieved by direct microscopy of the feathers, or feather digest preparations are useful.

Where therapy is needed it is as for feather mites.

# ENDOPARASITES

## *GIARDIASIS*

*Cause and pathogenesis*

Caused by a protozoal endoparasite *Giardiasis* spp. of the small intestine.

*Clinical signs*

Giardiasis is commonly associated with feather plucking over the torso in Cockatiels (*Nymphicus hollandicus*), and a deficiency of vitamin E. It has also been recorded as a cause of self-inflicted trauma to the wing webs of Cockatiels and Lovebirds.

*Diagnosis and therapy*

Diagnosis is made by microscopic examination of faecal samples stained with iodine or carbolfuschin.

Treatment is based on 20 mg/kg metronidazole, orally twice daily, for 7–10 days. Vitamin E supplementation at 0.06 mg/kg intramuscularly once weekly has been recorded.

# SKIN TRAUMA

Bite wounds are common amongst psittacine birds kept in groups (Figure 3.5), particularly in Cockatoos, where the male may become extremely aggressive towards his female mate. This may lead to extensive crushing wounds of the feet, head and

**Fig. 3.5** Bite wound in Grey Parrot over intertarsal (hock) joint.

wings. Good analgesia, covering antibiosis and reconstructive surgery are often necessary. Wounds too large to be covered by skin flaps may be repaired using dressings such as Granuflex® (Figures 3.6, 3.7, 3.8) sutured over the wounds allowing granulation beneath. Other products such as Biosist® (Cook UK Ltd), Softban® and Coflex® have also been widely and successfully used for this purpose.

**Figs. 3.6–8** Steps in dressing a bite wound.

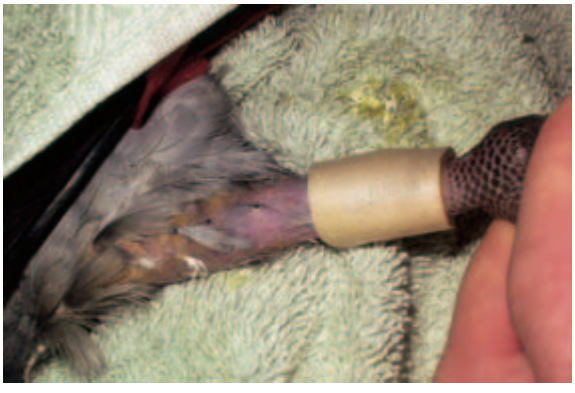

**Fig. 3.6** Primary dressing of Granuflex®.

**Fig. 3.7** Secondary dressing of Soffban®.

**Fig. 3.8** Tertiary dressing of Coflex®.

*Crop burns cause and pathogenesis*

Burns are seen especially in young birds that are fed food that is too hot. Especially seen in microwaved food which can contain 'hot spots'.

*Clinical signs*

The area affected will become blackened and start to separate, often discharging liquid food onto the skin of the breast from the inside of the crop.

*Therapy*

Initial stabilisation is essential as the bird may be in shock due to the large amount of necrosis of tissues. Aggressive fluid therapy, covering antibiotic therapy, and possibly the use of non-steroidal anti-inflammatory drugs are all advised.

- Repair is best achieved after a few days. Premature intervention will result in the attempt to re-appose tissues which may yet go on to degenerate. Once necrotic tissue has sloughed the wound may be repaired surgically.
- The patient is anaesthetised and intubated, with very gentle inflation of the endo-tracheal cuff, enough to prevent aspiration of crop contents should reflux occur, but not enough to damage the trachea, as avian patients have complete circular cartilages to the trachea which is therefore not distensible. A tube is placed into the crop to outline the proximal oesophagus and crop margins, and the neck and pectoral region is plucked of feathers.
- Debridement of the necrotic, full thickness burn may be performed, so as to define the fused edges of the skin / crop. This margin should be aseptically separated, reforming the separate crop and skin edges.
- The crop is sutured in a double row of inverting sutures, using polyglactin 910 (Vicryl®, Ethicon) or polydioxanone (PDS II®, Ethicon) and the skin is then closed in a row of simple interrupted non-absorbable nylon sutures.
- If the skin deficit is too large to close by undermining the surround tissues then one of two techniques may be employed.
  - A rotational flap may be used from the lateral neck. This has many problems as the subcutaneous tissues are sparse and densely populated with nerves and blood vessels in this region. Skin is difficult to mobilise easily from the breast region where it is tightly adherent to the underlying pectoral muscles.
  - Porcine sterile xenograft (Biosist®) to repair the deficit whilst re-epithelialisation occurs underneath, has been tried and found to be a highly successful technique by this author and others.

## *SPLIT KEELS*

*Cause and pathogenesis*

Split keels may be seen in birds given poor or inappropriate wing-clips. These birds then make clumsy and heavy landings and often split the skin over the keel bone. This in turn may lead to self trauma (see p. 41).

*Therapy*

Treatment is by debridement and suturing of the wound, and correcting the wing clip if possible. The latter is performed by 'imping' old feathers into the shafts of the clipped ones. This is aided by the use of cocktail sticks or bamboo kebab sticks whittled down and glued with epoxy into the shaft of the clipped and replacement feather. By carefully reassembling and matching like feathers for feathers, it is possible to re-create a new flight of wing feathers. This does not alter the pattern or timing of moulting.

## *ULCERATIVE DERMATITIS*

*Cause and pathogenesis*

A condition seen in many small Psittaciformes such as Lovebirds (*Agapornis* spp.), Cockatiels and some Parakeets. The aetiology of the condition is not truly known, but a stressful environment has been implicated. In addition conditions as diverse as localised neoplasia, contact irritants, *Agapornis* poxvirus in Lovebirds, and *Giardia* spp. intestinal infections in Cockatiels, have also been recorded as being associated with this condition. These lesions are often secondarily infected with bacteria such as *E. coli, Pseudomonas* spp. and *Staphylococcus* spp., and fungi such as *Aspergillus* spp.

*Clinical signs*

The ulcerative skin lesions often occur over the wing web or patagium and under the wing in the axilla region where the bird affected repeatedly attacks itself, suggesting intense pruritus.

*Diagnosis and therapy*

A wide variety of diagnostic tests may be needed to eliminate the different underlying causes including culture of lesional skin and skin biopsy. Intestinal parasites may be diagnosed on fresh faecal analysis showing the classical motile trophozoites with their double 'eye-spots'.

Treatment is difficult. *Giardia* infections may be treated with metronidazole (20 mg/kg PO, BID for ten days), and topical bacterial and fungal infections may

be treated with topical preparations accordingly. Extreme care should be taken with preparations containing corticosteroids as these may cause systemic immunosuppression. The use of physical means to prevent self mutilation, such as Elizabethan collars, neck braces or body bandages are also often necessary.

## BACTERIAL DERMATITIS

### *BACTERIAL / ULCERATIVE PODODERMATITIS ('BUMBLEFOOT')*

*Cause and pathogenesis*

Commonly seen in older, overweight cage birds especially Budgerigars, Canaries (*Serinus canaria*) and Cockatiels.

Has also been associated with:

- Hypovitaminosis A caused by an all-seed diet, which is naturally deficient in vitamin A.
- Poor perch design due to the presence of abrasive perches, particularly those with sandpaper covers and those of the same diameter. By having all of the perches of equal diameter, pressure is applied to the same parts of the feet when perching, which leads to reduction in blood flow to certain areas of the plantar surface of the foot.
- Bacteria commonly isolated from cage birds with this condition include *Staphylococcus* spp. and *E. coli*.

*Clinical signs*

Damage to the plantar surface of the foot due to poor blood supply leads to corn development and then pressure sores which become secondarily infected.

*Diagnosis and therapy*

Diagnosis is made on the basis of a compatible history and clinical signs. Impression smears of lesions reveal the presence of bacteria. Culture and sensitivity can help in selection of antibiotic therapy.

Treatment may be extremely difficult. Improvement of the diet, providing different diameter perches, padding perches with bandage materials, and treating infections (see Chapter Four on Raptor skin disease) are all necessary. In addition vitamin A supplementation is also recommended.

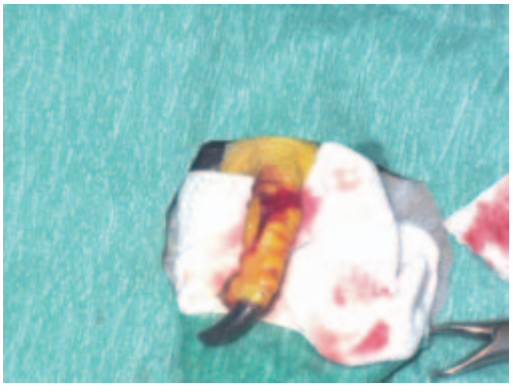

**Fig. 3.9** Abscess on foot.

## *OTHER BACTERIAL INFECTION*

General bacterial skin disease is relatively uncommon in cage birds. The main pathogen isolated seems to be *Staphylococcus* spp. (Reavill *et al*. 1990) (Figure 3.9). A gangrenous dermatitis has been observed with a mixed population of staphylococcal and clostridial bacteria (Gerlach 1994), resembling a similar condition in poultry where blackening of the skin occurs, usually on the extremities (wings and feet). Affected birds may die rapidly of toxaemia. Treatment is based on the use of broad spectrum antibiosis, such as potentiated amoxicillin at 150 mg/kg BID (Synulox®, Pfizer) or Marbofloxacin 5–10 mg/kg BID (Marbocyl, Vetoquinol) and possibly non-steroidal anti-inflammatory drugs such as meloxicam at 0.2 mg/kg (Metacam®, Boehringer–Ingelheim) with supportive lactated Ringer's fluid therapy.

# FUNGAL DERMATITIS

## *ASPERGILLOSIS*

*Cause and pathogenesis*

*Aspergillus* spp. infections of the skin may occur secondarily to self mutilation (see Ulcerative dermatitis p. 30). Aspergillosis in birds is most commonly caused by *A. fumigatus*, although *A. flavus*, *A. niger*, *A. nidulans*, and *A. terreus* may also cause clinical signs. The fungus is ubiquitous in the environment and flourishes in rotting vegetation and decaying organic material. Immunocompromised birds are predisposed.

*Clinical signs*

When the surface of a skin lesion is exposed to the air, the conidiophores of *A. fumigatus* can develop, forming the characteristic greenish-blue or dark grey patches on the skin.

*Diagnosis and therapy*

Diagnosis can be made on the basis of clinical signs and culture of the organism from lesions. Cytological diagnosis can be difficult.

Treatment may be performed using suitably diluted topical antifungal agents such as enilconazole (Imaverol®, Janssen) washes, topical clotrimazole (Canestan®, Bayer), or use of the veterinary antiseptic F10® (Health and Hygiene Pty). Deeper infections may be treated with itraconazole (Sporonox®, Janssen-Cilag) at 10 mg/kg, orally BID, however in African Grey Parrots this drug is toxic and the alternative systemic antifungal agent terbinafine (Lamisil®, Novartis) at 10–15 mg/kg orally, BID should be used (Dahlhausen *et al*., 2000).

## *CANDIDIASIS*

*Cause and pathogenesis*

*Candida albicans* has been recorded in Psittaciformes and Canaries as a cause of dermatitis (Perry *et al*. 1991; Wade 2000). Predisposing factors include poor nutrition and any underlying illness as well as antibiotic administration.

*Clinical signs*

In Canaries the disease presented as intense pruritus of the head and neck. Candida infections have also been associated with excessive feather picking.

*Diagnosis and treatment*

Diagnosis is made on the basis of clinical signs, cytological preparations and culture of feather pulp and feather biopsies.

Fluconazole has been suggested as the treatment of choice for yeast infections at 5 mg/kg orally BID for two weeks.

# VIRAL SKIN DISEASE

## *PSITTACINE BEAK AND FEATHER DISEASE (PBFD)*

*Cause and pathogenesis*

PBFD is caused by a circovirus. The condition is known to affect many psittacine birds, although circovirus has also been reported in other birds including Finches, Pigeons, Doves, Geese, Gulls and Ostriches. Infection occurs via faeces, feather dust and crop secretions from adults to young birds.

*Clinical signs*

Two main forms of the disease are reported (Girling 2003).

**Acute form** results in immunosuppression with white blood cell counts often lower than $1 \times 10^9$/litre. This is a common disease in young African Grey Parrots (*Psittacus erithacus*). These individuals generally die of secondary infections, frequently systemic fungal disease such as aspergillosis.

**Chronic form** of the disease manifests itself as the beak and feather disease.

- Feather disease.
  Dystrophies are seen which include changes in feather colouration; Vasa Parrots (*Coracopsis vasa greater*) often produce white feathers, African Grey Parrots often produce red feathers on the body and wings. Powder down feathers are often the first to become affected. This results in a lack of powder down production, which is very noticeable in 'dusty' psittacine species such as cockatoos where the beak is normally covered in a fine layer of powder. This results in a shiny beak. The feathers are progressively affected, the disease noticeably worsening at each moult. In many of these species, feather abnormalities may also be observed, although the systemic, often immuno-suppressive, signs of the disease are more prominent. A condition causing polyfolliculitis has been suggested as being associated with PBFD infection in Lovebirds and Budgerigars (Cooper and Harrison 1994). This affects the tail feathers and the dorsal neck feathers, and histologically appears as multiple feathers arising from one follicle. This causes chronic inflammation of the area affected.
- Beak and nails.
  Nail beds and the growth bed of the rhamphotheca and gnatotheca of the beak are affected in long-standing cases. This results in deformed beak and nails so that ultimately they are shed.

*Diagnosis and therapy*

Diagnosis is made based on clinical signs and also by polymerase chain reaction (PCR) technology based on a blood sample of feather pulp. Any bird testing positive should be immediately quarantined and retested 60–90 days later. If still positive, then the bird should be considered permanently infected. There is no treatment currently available for the condition, but some species, such as Lovebirds (*Agapornis* sp.) and some South American parrots, have recovered from clinical disease.

## *POLYOMA VIRUS*

*Cause and pathogenesis*

Avian polyoma virus is a member of the Papovaviridae family. It is thought to be spread horizontally through faeces, feather dust, respiratory secretions, crop secre-

tions and urates in the droppings. Vertical transmission is also known to occur through the egg but only in the Budgerigar.

*Clinical signs*

Polyoma virus infection in Budgerigars.

- 'Budgerigar fledgling disease' presents as sudden death in young Budgerigars under 15 days of age.
- 'French moult' has many different causes including polyoma virus. It occurs in Budgerigars over 15 days of age. It presents with abdominal distension, subcutaneous haemorrhages and a lack of down and contour feather formation. Feathers that are produced are often deformed. Surviving individuals also may demonstrate neurological signs.

Polyoma virus in other species.

In species other than Budgerigars, a sub-clinical disease is very common. Feather abnormalities are uncommon, although subcutaneous and feather follicle haemorrhages are seen. However in birds younger than four to five weeks of age, mortalities are still seen.

*Diagnosis and therapy*

Diagnosis is made via a cloacal swab to detect viral DNA using PCR technology. If a bird tests positive, then it should be quarantined for four to six weeks and then retested. If still positive the bird should be assumed to be persistently infected and shedding virus and so removed permanently from other birds. There is no treatment currently available for this disease, although a vaccine exists in the USA.

## *AVIAN POXVIRUS*

*Cause and pathogenesis*

Skin disease due to avipoxvirus in psittacine birds is uncommon. Infection can be acquired either through:

- Transfer from infected hosts via biting flies.
- Direct contact with surfaces or air-borne particles contaminated with poxvirus. Infections occurs when the virus enters through abraded skin or though mucous membranes.

Avian poxvirus is resistant to desiccation and can survive in dust particles for extended periods.

*Clinical signs*

Cases have been reported in Lovebirds (due to *Agapornis* poxvirus) where conjunctivitis or darkened areas of skin may be seen, and it has been suggested that this virus may be the causal agent of ulcerative cutaneous disease in Lovebirds. In the majority of infections, the disease afflicts the eyelid margins with ulceration, but also creates upper respiratory tract infections. These may be severe in Amazon parrots. Canaries are also affected by their own form of poxvirus, and may die suddenly of a septicaemic form, or may produce skin lesions affecting the head. Lung neoplasms have also been associated with this poxvirus in survivors. Poxvirus has also been recorded in pigeons (Figures 3.10 and 3.11).

*Diagnosis and therapy*

Diagnosis is based on clinical signs and the demonstration of classical Bollinger body inclusion bodies on histopathological examination. Supplementation of the diet with vitamin A (10 000 to 25 000 IU/300 g bodyweight) and vitamin C have been useful in preventing infection (Ritchie 1995).

**Fig. 3.10** Poxvirus infection on the face of pigeon.

**Fig. 3.11** Poxvirus infection on the foot of pigeon.

## *PAPILLOMAS*

Papilloma-like lesions of the feet have been seen in Macaws, Cockatoos, Finches, Budgerigars and Cockateils. The lesions are often proliferative. It is thought that the condition is due to a virus, possibly a herpes virus. Other papillomas are seen more commonly on mucous membranes, such as the cloaca, and oral mucosa, and these are also thought to be virally induced.

## *PROVENTRICULAR DILATATION DISEASE*

This is a condition thought to be caused by a virus, possibly a paramyxovirus. The disease is classically associated with digestive tract and neurological disease, but birds so infected have been noted to feather pluck and self-mutilate over the breast area (Chitty 2003).

# NUTRITIONAL SKIN PROBLEMS

## *HYPOVITAMINOSIS A*

*Cause and pathogenesis*

Vitamin A is an essential vitamin in caged birds needed to maintain the skin and feathers. Amazon parrots are predisposed to vitamin A deficiency. Foods rich in vitamin A include cod liver oil, cooked liver, egg yolk, apricots, corn, carrots, squash, sweet potatoes, broccoli, spinach and parsley. Seeds are notoriously deficient in vitamin A and where birds eat these in preference to other foods clinical signs can develop.

*Clinical signs*

Hyperkeratosis of the skin is evident (Figure 3.12), with white plaques in the oral mucosa. The latter usually leads to a loss of appetite. Rhinitis and blepharitis are also commonly seen.

*Diagnosis and therapy*

Diagnosis is made on the history of an inappropriate diet and clinical signs. Therapy can be made by adjusting the diet to feed food rich in Vitamin A such as carrots, and cod liver oil. Injectable Vitamin A may also be given on a single occasion 5000–20000 IU/kg, IM (Pollock *et al*. 2005).

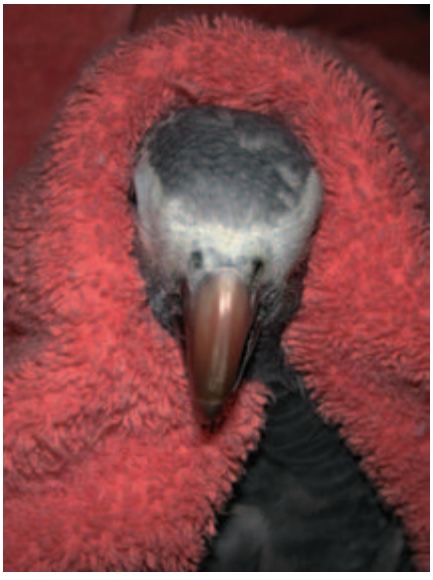

**Fig. 3.12** Vitamin A deficiency in the parrot showing hyperkeratosis of the skin.

## SKIN NEOPLASIA

### *TUMOUR TYPES*

**Fibrosarcomas** are one of the more commonly seen tumours of the head and wing tips of cage birds. They tend to be very firm in texture, and often ulcerate through to the skin surface by the time the patient is presented to the clinician.

**Lymphosarcomas** have also been reported in the head region of cage birds. These are more skin bound, rather than arising in the subcutaneous tissues and then spreading to the skin as is the case with fibrosarcomas. They are also softer in texture than fibrosarcomas and more yellow in hue.

**Squamous cell carcinomas** have been reported rarely in cage birds (Leach 1992). They have however been reported in many varied species of cage birds from Cockatoos to Hornbills. They often involve the beak and associated structures, but have been recorded affecting the skin over the extremities and torso.

#### *Diagnosis and therapy*

Surgical excision and histopathology is advised where possible. Cutaneous lymphosarcomas have been treated successfully using chemotherapeutic regimes similar to those used in small animal medicine (France 1993). Photodynamic therapy using an intravenous photosensitising agent (hexylether pyropheophorbide-α) subsequently activated by a light source, has been used to successfully, but only temporarily, regress the growth of a squamous cell carcinoma of the casque in a Great Hornbill (*Buceros bicornis*) (Suedmeyer *et al*. 2001).

## *NON-TUMOUR LESIONS*

### *XANTHOMATOSIS*

*Cause and pathogenesis*

This is a condition resembling a tumour but thought to be due to an accumulation of lipid-containing macrophages in conjunction with variable amounts of fibrosis. They are common in small species of Psittaciformes, and appear as discrete areas of dermal swelling, yellow-brown in colour, causing a diffuse thickening of the skin. Often xanthomas are seen on the extremities of the body, particularly the wing tips. They may be associated with severe pruritus and associated self trauma. The aetiology is currently unknown, but is thought to be associated with a high fat diet, trauma of the affected area and possibly a disorder of lipid metabolism.

*Diagnosis and therapy*

Diagnosis is made on fine-needle aspirate or biopsy demonstrating vacuolated lipid containing macrophages. Treatment is based on alteration of the diet to a low protein, low fat diet and surgical excision where possible. Some may respond to thyroid supplementation therapy, with long-term treatment involving L-thyroxine. Doses of 0.84 mg/litre (0.1 mg tablet in 120 ml water) have been suggested (Perry *et al*. 1991).

## BEHAVIOURAL SKIN AND FEATHER PROBLEMS

## *FEATHER PLUCKING AND FEATHER CHEWING*

*Cause and pathogenesis*

Many different causes of feather plucking and feather chewing exist (see below). Only when these non-behavioural causes of feather plucking / chewing have been systematically ruled out can a true diagnosis of behavioural feather chewing or plucking be made, however persuasive the history may be initially.

### *NON-BEHAVIOURAL CAUSES OF FEATHER PLUCKING AND CHEWING*

- Allergic skin disease.
- Chlamydophilosis (systemic infection with *Chlamydophila psittaci*).
- Ectoparasites (e.g. *Myialges* spp. mites).
- Endoparasites (e.g. *Giardia* sp.).
- Environmental aerosols / contaminants (e.g. tobacco smoke, perfumes, aerosols).
- Heavy metal poisoning (e.g. lead and zinc poisoning).

- Hypothyroidism.
- Infectious folliculitis:
  - viral (e.g. PBFD, polyomavirus).
  - fungal (e.g. aspergillosis, candidiasis).
  - bacterial (e.g. staphylococcal infections).
- Malnutrition.
- Neoplasia.
- Other systemic disease (e.g. aspergillosis, proventricular dilatation syndrome).

## *BEHAVIOURAL CAUSES OF FEATHER PLUCKING AND FEATHER CHEWING*

- Anxiety is especially seen as a trigger in birds upset at being separated from their owners for large parts of the day.
- Attention seeking is achieved by feather plucking because it elicits a response from their owner.
- Boredom can lead to plucking as normal social behaviour is lost. Wild birds spend large parts of their lives searching for food, and interacting with members of their own family.
- Environmental upheaval such as moving the bird's cage into a new room, or the addition of a cat to the family!
- Sexual frustration of birds that have mate-bonded to an owner, but, for obvious reasons, the owner cannot reciprocate the bird's sexual advances! This can result in extreme frustration and feather plucking.

### *Diagnosis and therapy*

Diagnosis is made on the basis of history, clinical signs especially distribution of lesions (Figures 3.13 and 3.14). Self-inflicted trauma unlike viral disease does not affect the head. Diagnostic work up should eliminate all non behavioural triggers before starting on behavioural therapy.

Treatment of these behavioural problems can be complicated and often involves lifestyle changes. For severe obsessive compulsive disorders or stereotypical

**Fig. 3.13** Feather chewing in an Umbrella Cockatoo.

**Fig. 3.14** Feather plucking in a male Eclectus Parrot.

behaviour, the use of behavioural modifying drugs such as clomipramine (Clomicalm®, Novartis) 0.5–1 mg/kg, orally BID, or haloperidol 0.1–0.2 mg/kg orally SID–BID have been recommended.

## *SELF MUTILATION*

*Cause and pathogenesis*

Mutilation of the skin is relatively uncommon in cage birds. Causes include internal / systemic diseases causing pain and discomfort leading to chewing of the skin over the site of the noxious stimulus. Behavioural problems are seen especially the Umbrella Cockatoo (*Cacatua alba*), Moluccan Cockatoo (*Cacatua moluccensis*), Hahn's Macaw (*Diopsittaca nobilis*) and Severe Macaw (*Ara severa*). These birds may start chewing at the skin after a traumatic injury such as a split keel, caused by clumsy landings, often in birds with poor wing-clips. This provides a focus of attention for the birds, which then continues to damage the skin, the underlying muscles and keel bone. In some breeds particularly the Cockatoo the cause can be purely behavioural (Figure 3.15).

*Diagnosis and therapy*

History of trauma and physical examination will help establish an underlying cause. When self-inflicted trauma is behavioural, the use of Elizabethan collars, reversed, or neck braces (as produced by Kruuse Ltd) may be useful in preventing self mutilation. With large skin deficits, the use of Biosist® (Cook UK Ltd) as a temporary graft, or suturing Granuflex® (Convatec UK) dressings over the wound can allow re-epithelialisation and granulation of damaged tissues.

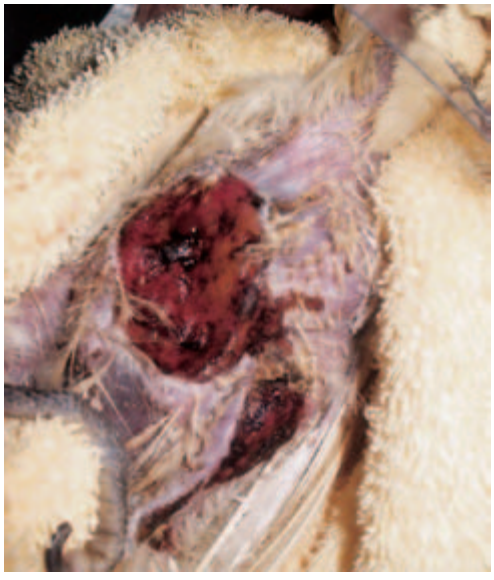

**Fig. 3.15** Self mutilation of the breast in a Goffin's Cockatoo.

## HORMONAL SKIN PROBLEMS

### *BROWN HYPERTROPHY OF THE CERE*

*Cause and pathogenesis*

This condition is particularly common in Budgerigars. It produces an increase in the thickness of the cere, and may lead to obstruction of the nares. It is seen generally in older hen birds, however it may be seen in males, indicating the presence of a gonadal tumour producing oestrogens.

*Diagnosis and therapy*

Diagnosis is made by physical examination and by ruling out other conditions affecting the cere. Treatment is rarely necessary in hen birds, although some of the cere may be carefully removed if the nares become occluded. In male birds gonadal tumours are generally not operable.

### *HYPOTHYROIDISM*

*Cause and pathogenesis*

This has been associated with increased weight gain as fat deposits, delayed moult and a diffuse loss of contour feathers in Psittaciformes, particularly in Macaws and the African Grey Parrot (Oglesbee 1992; Hillyer 1989). A similar condition is also seen in Budgerigars, but there is some suggestion that many of these

cases are actually due to nutritional iodine deficiencies rather than primary hypothyroidism.

*Diagnosis and therapy*

Diagnosis is made on demonstration of a reduced response to thyroid stimulating hormone (TSH) at a dose of 1 IU given intramuscularly. Total thyroxine levels should double over four hours. TSH may be difficult to source therefore other clinical findings such as hypercholesterolaemia, hypoalbuminaemia and a mild non-regenerative anaemia and heterophilia may also be useful indicators. Skin biopsy will reveal hyperkeratosis and vacuolar degeneration of the feather follicle epithelial lining.

Treatment in the case of the Macaw described above involved L-thyroxine at 0.02 mg/kg orally BID.

## MISCELLANEOUS SKIN CONDITIONS

### *ALLERGIC SKIN DISEASE*

*Cause and pathogenesis*

This area of avian dermatology is much contested. There is plenty of evidence to suggest that cage birds, particularly Psittaciformes, are susceptible to allergic skin disease. This may lead to feather chewing, feather plucking and skin mutilation. Affected birds are often irritable and may chew their feet when upset.

*Diagnosis and therapy*

Diagnosis is made by ruling out other causes of skin disease initially via skin scrapings, biopsies, feather sampling etc. (see Chapter Two on avian dermatology diagnostic tests). If these are all negative in result, then an intradermal skin test may be attempted.

Therapy of the condition is based on the use of essential fatty acid supplementation (EFAs) and where possible removal of the bird from any antigen isolated by skin test. Doses of EFAs have not been calculated in cage birds, but this author uses Viacutin® (Boehringer-Ingelheim) at a rate of two drops from a capsule per kilogram bodyweight once daily. As with small mammals, doses need to be maintained for three to four weeks before clinical improvement is likely to be seen. Antihistamines do not seem to work in birds, and corticosteroids are dangerous, as even one dose may severely impair the bird's immune system.

### *CONSTRICTED TOE SYNDROME*

*Cause and pathogenesis*

This is a condition often seen in young hatchling Psittaciformes. It appears that a fine circumferential constriction of the skin occurs on one or more of the toes. It

has been thought that this is associated with low environmental humidity, but this has not been conclusively proven.

*Diagnosis and therapy*

Diagnosis is made by history, clinical signs and diagnostic rule outs. Treatment is by careful dissection of the constriction, and cutting longitudinally through the skin of the toe, parallel to the phalanges to relieve pressure. Increasing environmental humidity to 70–80% in hatchling accommodation is advised to try to prevent occurrence.

## *FEATHER FOLLICLE CYSTS*

*Cause and pathogenesis*

Feather cysts are thought to be hereditary. They are commonly seen in small cage birds such as Budgerigars and Canaries (Figure 3.16), with Norwich and Gloucester breeds of Canary being over-represented. A cyst develops due to the inability of a growing feather to break through the skin surface. As the feather develops the cyst enlarges and its contents degenerate into a caseous mass. These cysts may eventually rupture and become secondarily infected. Some feather cysts may also occur due to traumatic damage of the feather follicle, and some nutritional deficiencies.

*Diagnosis and therapy*

Diagnosis is by history, physical examination and diagnostic rule outs of other lesions.

Total excision of the feather cyst is the method of treatment of choice. A technique using microsurgery to remove the capsule and nutrient artery supplying the affected feather has been described (Harrison 2003). This may be difficult in larger cysts, particularly on the wings where the lining of the cyst and feather

**Fig. 3.16** Feather follicle cyst in a Canary.

follicle may be attached to underlying bone. These cysts may need to be partially resected using radiosurgical techniques, and the remaining cyst capsule marsupialised to the skin surface.

In all cases due to the probably heritable nature of the cysts, it is advisable to avoid breeding from affected birds.

## *OVER-GROWN BEAK AND CLAWS*

*Cause and pathogenesis*

This may occur due to dietary imbalances, such as an excess of proteins and calories with a relative deficiency in calcium and vitamins such as vitamin A. Such birds often have poor quality feathers, and may be continually moulting. Overgrown beaks in Budgerigars and other small Psittaciformes may indicate liver pathology, such as hepatic lipidosis or liver tumours. In larger Psittaciformes, the same situation may arise, but often other traumatic causes are seen. This is commonly the case where a relative brachygnathism is seen, due to a fore-shortening of the maxilla after the bird has flown into a window. This often results in fractures of the bones supporting the upper beak, which then heal in a fore-shortened form.

Some nail pathology may be traumatic in origin, or associated with mite infestations particularly in Passeriformes such as Canaries, resulting in damage to the keratin growth plate and an often corkscrew-shaped nail.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs and rule outs of such differentials as infection and ectoparasites.

Therapy where possible should be left to the bird. The presence of soft wood perches that the bird can gnaw and cuttlefish bones allows the bird to naturally shape their own beaks and claws. Rough sandpaper perches cause irritation to the feet and do not help to regulate claw growth. Nails and beaks can be clipped carefully with nail clippers. In small birds an emery board may be used to file the nails down. Facilities to control haemorrhage with a stypic pencil should be available at the time of clipping. Haemorrhage from a nail especially in a small bird can be dangerous.

## *FEATHER COLOUR CHANGES*

*Cause and pathogenesis*

These may be due to nutritional imbalances / deficiencies, or systemic or localised disease processes. Areas of colour loss in characteristic bands or streaks across body, wing and tail feathers, the so-called ‘fret lines’, may indicate a period of stress, nutritional deficiency or systemic disease process in the bird affected at the

time that the feather was being formed. It can therefore give a historical view of the bird's health status.

### *INFECTIOUS DISEASE*

- The viral disease PBFD has been associated with feather colour changes in African Grey Parrots and Vasa Parrots (see above), as well as leading to a soiled appearance to feathers in Cockatoos due to the loss of the powder down-producing feathers that provide protection to the feather structure.

### *DIETARY DEFICIENCY*

- Some species, such as the Red Factor Canary, will become progressively paler in colour with each moult if their diet is deficient in beta-carotene / vitamin A precursors. Other nutritional imbalances such as amino-acid deficiencies have also been reported as producing feather colouration changes, e.g. deficiencies in choline and riboflavin have produced abnormal pigmentation in Cockatiels (Cooper and Harrison 1994).

### *SYSTEMIC DISEASE*

- Darkening of the feather colour may indicate liver disease, particularly where black feathers appear in species such as Amazon Parrots. In addition, yellowing of white-coloured feathers in Cockatiels may indicate liver dysfunction as well, due to biliverdin staining of the lipids released by keratinocytes.

*Diagnosis and therapy*

A thorough history especially noting the bird's diet is important. A physical examination and blood profiles can lead to identification and hence therapy of underlying causes.

## *GENETIC ABNORMALITIES*

Some species of cage birds are bred with feather abnormalities. An example is the feather duster and straw feather styles of genetic condition in Budgerigars. Although some of these individuals may have a history of viral disease, many do not and the conditions are thought to be purely genetic. It is advised that such individuals are not bred from.

## *UROPYGIAL GLAND IMPACTION*

The uropygial or preen gland is situated at the base of the tail dorsally, although it is not fully developed in all cage birds and is absent in species such as Amazon

Parrots. This holocrine gland may become impacted, which may be associated with nutritional deficiencies such as hypovitaminosis A. Alternatively it may become infected, or adenoma or adenocarcinoma development may occur.

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Chapter 4

# Skin Diseases and Treatment of Raptors

## ECTOPARASITES

Important groups of ectoparasites that can affect raptors are mites, ticks and other insects.

## *MITES*

Raptors are commonly affected by mites. These can be divided into:

- Skin mites.
  - Burrowing mite – Cnemidocoptidae (*C. pilae*).
  - Surface mite – Dermanyssidae (*D. gallinae*), (*Ornithonyssus* spp.)
- Feather mites.
- Quill mites.

### *SKIN MITES*

### *Burrowing mites – Cnemidocoptidae*

#### Scaly beak / scaly leg (*Cnemidocoptes* spp.)

*Cause and pathogenesis*

*Cnemidocoptes* spp. mites are uncommon in raptors. They have however been reported in Northern Sparrowhawks (Malley and Whitbread 1996) and a Great Horned Owl (Schulz *et al.* 1989) in association with clinical disease. Predisposing factors include immunosuppression, which can be due to some other concurrent systemic disease, and inadequate nutrition such as hypovitaminosis A.

*Clinical signs*

When mites are present they are found on either the legs or ceres of raptors causing 'scaly leg' or 'scaly beak'. In the case of ceres it may appear that the bird is continually traumatising its cere flying into aviary wire, although when such a

bird is removed from its aviary, the lesion does not heal. When legs are affected, they appear crusty, with a dry scabby or flaky appearance.

*Diagnosis and therapy*

- Diagnosis may be made by visualisation of the mite in skin scrapings as a typical round parasite with short conical legs barely projecting beyond the circular body outline.
- Treatment is based on topical ivermectin 0.2 mg/kg diluted with propylene glycol at 1:10 applied once weekly for three applications or subcutaneous ivermectin at 0.2 mg/kg once, and repeated after seven to ten days on three to six occasions. Additional problems such as hypovitaminosis A should also be corrected.
- Therapy can be undertaken with Fipronil spray (Frontline®, Merial). This should not be applied directly to the bird due to the risk of hypothermia. A small amount should instead be applied to cotton wool and then apply to back of head, under wings and base of tail. Repeat every two to four weeks.

## *Surface mite – Dermanyssidae*

### Red mite (*Dermanyssus gallinae*)

*Cause and pathogenesis*

This mite is an occasional parasite, as with domestic fowl. It spends the day hiding in nooks and crevices of the bird's roost, coming out at nightfall to take a blood meal. Some birds will show signs of broken feathers or feather loss, particularly over the proximal breast or just distal to intertarsal joint.

*Clinical signs*

Skin lesions are rare, but can cause a papular eruption. Most commonly its effects are associated with its blood-sucking capabilities, such as anaemia and over preening due to skin irritation.

*Diagnosis and therapy*

Diagnosis may be difficult due to the off-host nature of this mite. Mites can be identified with oval body shape and long legs.

Treatment is based on therapy of the environment with permethrin / pyriproxyfen sprays (Indorex®, Virbac), and rigorous cleaning of the cage and local environment. Ivermectin at 0.2 mg/kg may also be used topically as can Fipronil spray (Frontline®, Merial).

## *Surface mite – Macronyssidae*

### Ornithonyssus sylvarium

*Cause and pathogenesis*

The northern fowl mite, *Ornithonyssus sylvarium* has been recovered on raptors housed in old poultry houses. Unlike the red mite, the northern fowl mite remains on the host all the time and is thus easier to diagnose. Small rodents can act as incidental hosts. This mite remains on the host throughout its life cycle.

*Clinical signs*

Feathers become matted often with a grey-black discolouration. Skin may become thickened and scaly. Anaemia can occur due to parasite feeding activity.

*Diagnosis and therapy*

Diagnosis is made by skin scrapes of lesional areas. The mite has an oval body and long protruding legs. Treatment may be successful with avermectins or with the spray preparation of Fipronil (Frontline®, Merial).

## *QUILL MITES*

*Cause and pathogenesis*

Quill mites (*Harpyrhychus* spp.) are a less common but significant parasite. These mites live within the shaft of a 'flight blood feather', causing a reaction which tends to cause a pinching off and loss of the feather before its growth has completed.

*Clinical signs*

Most of these parasites are present in small numbers and do not cause disease. However in debilitated birds, or in large infestations, clinical signs may be seen. Birds exhibit evidence of pruritus and will pick out the infested feathers.

*Diagnosis and therapy*

Diagnosis is achieved by direct microscopy of the feathers and feather digest preparations are also useful. One can attempt control of these parasites with avermectin parasiticides, but it is not always effective.

## *TICKS*

*Cause and pathogenesis*

These are common in aviary birds where there are overhanging branches from surrounding trees allowing the ticks to detach from previous wild avian (and rodent) hosts and drop into the raptor enclosure.

*Clinical signs*

The ticks (usually *Ixodes ricinus* in the UK) often attach to the head region of the raptor, and quickly result in the death of the bird. Clinically, massive head oedema often occurs as a result of toxins released in the tick saliva (Forbes and Simpson 1993). In addition, haemoparasites, viruses and rickettsial organisms are released into the avian blood stream.

*Diagnosis and therapy*

Diagnosis is by history and clinical signs.

Treatment is based on manual removal of the tick, treatment with ivermectin at 0.2 mg/kg orally / percutaneously and supportive treatment of the raptor with aggressive fluid therapy and broad-spectrum antibiosis, e.g. potentiated amoxicillin and enrofloxacin.

## *OTHER INSECTS*

Insects of importance in raptors include:

- Lice.
- Blow flies.
- Keds.
- Mosquitos.
- Beetles.

### *LICE*

*Cause and pathogenesis*

These wingless insects, are the most common avian ectoparasites. Only biting lice have been recorded in raptors. Their characteristic body is 2–10 mm in length and flattened dorso-ventrally. Species of biting louse that have been recorded in raptors include the following genera: *Laemobothrion*; *Degeeriella*; *Falcolipeurus*; *Colpocephalum*; *Craspedorrhynchus*; *Aegypoecus* and *Kurodaia* (Krone and Cooper 2002). They tend to be host specific, and their entire life cycle is spent on the host,

being unable to survive for long when removed from it. Transmission is by direct contact between infected individuals.

*Clinical signs*

Feather lice can cause irritation to the bird, but cause it no real harm, living off feather material. In general, small numbers of lice are non-pathogenic. However, large infestations may lead to listlessness, loss of sleep, increased preening and decreased appetite. Due to over preening and the activity of the lice, the feathers may become ragged in appearance, and some will break as they become progressively weakened.

When a heavy louse burden is identified it is generally an indication that the bird is unable to preen or has some other significant systemic disease. In particular if an individual bird is affected amongst a group, tests should be performed to find the cause of the underlying disease.

*Diagnosis and therapy*

Adult lice are easily seen moving around the plumage, or eggs may be seen attached to feathers. Therapy can be undertaken with Fipronil spray (Frontline®, Merial).

## *BLOW FLIES*

*Cause and pathogenesis*

These are generally only a problem for hatchling / nestling raptors, but in theory it is also possible for a debilitated adult raptor to become infested. Species such as *Calliphora*, *Lucilia* and *Phormia* have been associated with the condition.

*Diagnosis and therapy*

Diagnosis is by history and clinical signs. Treatment is by manual removal of any maggots in addition to supportive therapy. The latter includes fluid therapy (lactated Ringers® solution at 50–75 ml/kg per day intravenously / intraosseously / subcutaneously) and covering antibiotic therapy, e.g. clavulanate potentiated amoxicillin (Synulox®, Pfizer) at 150 mg/kg, orally BID or marbofloxacin (Marbocyl®; Vetoquinol) at 10 mg/kg SID for five to seven days.

## *LOUSE FLIES / KEDS*

*Cause and pathogenesis*

These are flies belonging to the family Hippoboscidae such as *Pseudolynchia*. These ectoparasites are related to the sheep ked, and are generally non-pathogenic, although they can inflict a painful bite. They are not host specific. Some species are wingless, others are able to fly. Some complete their life cycle on host while others spend time in nests / crevices and may lay eggs off the host. The danger is in their ability to transmit haemoprotozoan parasites such as *Haemoproteus* sp. which can lead to haemolytic anaemia.

*Clinical signs*

These blood-sucking parasites can cause pruritus and in severe cases may cause an anaemia (especially in young birds).

*Diagnosis and therapy*

Easily recognised as large flies flattened dorso-ventrally. Topical insecticides may be used to treat infestations.

## *MOSQUITOS AND BLACK FLIES*

Black flies (*Simulium* sp.) have been recorded affecting nestlings of raptors, and are responsible for the transmission of the haemoparasite *Leucocytozoon* sp. This latter blood parasite is an important pathogen and cause of anaemia in young raptors.

## *BEETLES*

*Cause and pathogenesis*

Beetles of the family Dermestidae (often known as 'hide beetles' due to their scavenging habits, living off the hides of dead animals) have been associated with skin disease in a living, wild Saker falcon (*Falco cherrug*) (Samour & Naldo 2003).

*Clinical signs*

In the case described by Samour the parasite affected mainly the area on the dorsal aspect of the tail base, in the vicinity of the uropygial gland. The affected bird had a low tail carriage, and skin oedema, inflammation and ulceration in the affected area.

*Diagnosis and therapy*

Diagnosis is made by identification of the parasites in the coat. Therapy in this case was performed in an anaesthetised patient. A permethrin / piperonyl butoxide based insecticide was applied (Falcon Insect Liquidator®, Vetafarm) to kill the parasites and the infection treated with systemic marbofloxacin at 10 mg/kg once daily i.m. (Marbocyl®, Vetoquinol), topical quaternary ammonium and biguanidine compound-based disinfectant (F10SC®, Health and Hygiene Pty) diluted 1: 500 with warm water, and topical sodium fucidate (Fucidin®, Leo Pharmaceutical Products) for five days.

## SKIN TRAUMA

*Cause and pathogenesis*

Traumatic wounds of the head are common in raptors especially Northern Goshawks in wire-netted enclosures. Wounds of the skin overlying the tibiotarsus are common in raptors due to chaffing of the leather jesses, attached to the leg in this region and used to tether raptors. Mud and dirt become lodged underneath the leather and rubbing causes often severe damage to the soft tissue structures, including tendons which are very close to the skin surface in this region.

*Diagnosis and therapy*

Head wounds can be extensive and often require sliding pedicle grafts, or the use of porcine xenografts (VetBiosist®, Cook) to cover the deficit. Treatment of leg wounds can be difficult. Careful dissection of any scab is the first step, followed by application of dressings such as Granuflex® (Convatec UK) to encourage granulation. Topical products such as Dermisol® (Pfizer) have been used to remove infection and Preparation H® (Whitehall Lab) to stimulate blood flow to the affected area and speed recovery.

## BACTERIAL DERMATITIS

### *BACTERIAL PODODERMATITIS ('BUMBLEFOOT')*

*Cause and pathogenesis*

Bumblefoot is the colloquial term used to describe infection of the foot or feet of a bird (Figure 4.1). It has been graded according to severity by Oaks (1993) and Remple (1993) on a scale of I (mildest involving the integument only with no infection of underlying tissues) to V (severest, involving osteomyelitis and loss of function). Hypovitaminosis A and obesity have been implicated as possible inciting factors.

**Fig. 4.1** Abscess (corn) on foot of Harris Hawk.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs. Cytology of exudates and culture from lesions can help determine an appropriate antibiotic regime. The degree of wound or injury will determine the treatment and the prognosis. In attempting to treat the condition, Redig and Ackermann (2000) suggest the improvement of diet is important. The provision of suitable perches, and housing, and regular monitoring of the feet are also necessary. Affected raptors should be flown regularly as this removes pressure from the feet and aids blood circulation to the affected areas, improving healing times (Harcourt-Brown 1996). Antibiotic therapy should where possible be based on cultures. In severe cases antibiotics may be needed for many months, and may need to be changed during the course of the treatment due to a continually altering flora.

*Therapy of mild–moderate bumblefoot*

Mild bumblefoot, category I and II, may be treated purely by topical, parenteral and husbandry methods alone. The raptor should be housed on padded perches – the use of the synthetic compound Astroturf® is advised. In addition, the debridement of the superficial wounds to the feet along with topical application of dimethyl sulfoxide (DMSO) with antibiotics such as piperacillin, rifampicin, and lincomycin have been used. For deeper infections (II–III) parenteral antibiotics, preferably prior to surgical intervention, are advised (Figure 4.2). Harcourt-Brown (1996) prefers lincomycin (50 mg/kg orally BID) or cloxacillin (250 mg/kg orally BID) for a minimum of seven days. Marbofloxacin has also been used by this author and others at 5–10 mg/kg orally SID. Sodium fusidate (Fucidin®, Leo) may be used to treat scabs topically.

Bandaging of the feet will aid healing by taking pressure off the feet. The foot may be bandaged as a 'ball bandage' whereby the plantar aspect is packed with

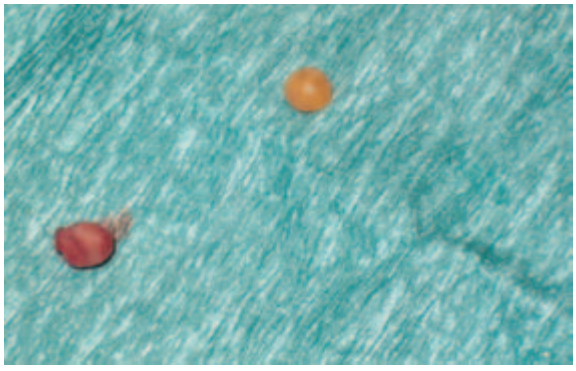

**Fig. 4.2** Capsule and abscess from Fig. 4.1 after it has been dissected out.

gauze or cotton wadding, and the digits wrapped to this. It is always advisable to bandage both feet, as the bird will favour the opposite non-bandaged foot, so putting increased pressure on this 'normal' foot, dramatically increasing the likelihood of bumblefoot developing here as well. Other methods have been described which include the formation of a lightweight cast made of non-adherent elasticated bandage such as Vetrap®/Coflex® coated in epoxy glue. The technique involves applying small strips of bandage to each digit around the region of P2. A roll of soft bandage is then created, fitting the underside of the foot, wide enough for its outer rim to touch these areas of bandage. Around this soft, cottonwool bandage is wrapped more elasticated bandage and the whole is glued, using epoxy glue, to the plantar aspect of the strips wrapped around the digits. To further hold this corrective 'shoe' to the raptor's foot, more elasticated dressing is wrapped around the digits and the plantar 'shoe'. The whole is then covered in epoxy glue, and allowed to dry. The base of the 'shoe' is then cut off, and the cottonwool wadding inside removed. This allows access to the plantar aspect of the main part of the foot, and spreads all of the pressure to the digits, so allowing any pressure sore / infected area to heal under observation. Again, both feet should be treated the same, whether or not one or both are affected by bumblefoot.

A similar principle has been described using a 'doughnut' bandage as well (Burke *et al.* 2002) to lift the plantar aspect of the foot off the ground, and forming a ring of material touching the toes at the level of P2. Harcourt-Brown (1996) has described a similar technique using a foot cast with a methacrylate material, again lifting the affected portion of the plantar surface off the ground to reduce pressure on the wound.

### *Therapy of severe bumblefoot*

Severe bumblefoot (categories III–V) may not be treatable, but methods involving extensive debridement of the infected areas, followed by implantation of

antibiotic-impregnated polymethyl methacrylate beads (AIPMMA) as described by Remple and Forbes (2000) may prove successful. Alternatively, extensive curettage and primary closure of the wound followed by parenteral antibiotics (see above) is useful. If full closure of any plantar defects is not possible due to lack of available skin, a purse-string suture using polydioxanone or nylon may be used to close the wound down partially. Post-operatively, it is necessary to provide extensive support bandaging as described above, and if access is made to the plantar aspect, further treatment with povidone-iodine soaked swabs, topical Dermisol® or oil of proflavine may be used to further encourage healing.

## FUNGAL DERMATITIS

*Cause and pathogenesis*

Fungal dermatitis is relatively uncommon in raptors. *Aspergillus* spp. has been isolated from a case of blepharitis and cephalic dermatitis in a Peregrine Falcon–Gyrfalcon hybrid (*Falco peregrinus* × *Falco rusticolus*) (Abrams *et al.* 2001).

*Diagnosis and therapy*

Diagnosis should be possible by examination of stained samples from feather pulp or routine cultures from the calamus. In the case described by Abrams the disease was diagnosed by histopathological analysis of a skin biopsy. Successful treatment was based on the use of itraconazole (Sporanox® Janssen) at 15 mg/kg orally BID, and miconazole cream applied topically.

## VIRAL DERMATITIS

### *POXVIRUS*

*Cause and pathogenesis*

Generally only the dry form of the disease is seen in raptors and it is rare in the UK. The virus is transmitted by mosquitos. Strigiformes (owls) have not been reported with avipoxviral infections.

*Clinical signs*

Clinical signs include papules and vesicles on the eye margins, feet and unfeathered portions of the legs, and the cere. These lesions are often pruritic and become secondarily infected.

*Diagnosis and therapy*

Diagnosis is made on the clinical signs and the demonstration of the classical Bollinger bodies on histopathology. Treatment is based on management of secondary bacterial infections. Disinfection of premises using dilute quaternary ammonium compounds, or other viricidal disinfectants (e.g. F10® Health and Hygiene Pty).

## SKIN NEOPLASIA

Skin neoplasia in raptors is uncommon. Squamous cell carcinomas and spindle cell carcinomas, the latter isolated to the wing tips, have been reported (Malley and Whitbread 1996).

## MISCELLANEOUS

### *FROSTBITE*

*Cause and pathogenesis*

Frostbite is common in raptors tethered to perches in traditional three-sided enclosures (known as 'weatherings'). As birds are only housed in weatherings during the training season frostbite, if seen, is generally observed in the early–late autumn.

*Clinical signs*

Lesions are more associated with deeper tissue necrosis, rather than purely skin disease. The skin is often seen to become swollen with blister-like eruptions over the wing tip. This can then become discoloured and darkened. However, despite therapy vascularity may be lost to the distal wing tip which will then slough.

*Diagnosis and therapy*

Diagnosis is made on the basis of history and clinical signs. Therapy is best employed with vascular stimulants such as isoxsuprine (5–10 mg/kg, once daily, PO for three to four weeks) Preparation H® (Whitehall Labs) and warming the patient. Systemic antibiosis administration is advised in cases where vascular damage and wing tip slough occurs.

## *BEHAVIOURAL FEATHER PLUCKING*

*Cause and pathogenesis*

This has been reported in Harris Hawks (*Parabuteo unicinctus*) (Malley and Whitbread 1996), particularly those used regularly for public display.

*Clinical signs*

Plucking occurred across the breast and medial aspects of the legs. Where plucking is self induced there is sparing of the head.

*Diagnosis and therapy*

Diagnosis is made by rule out of non-behavioural causes of feather plucking especially infectious and parasitic triggers. Therapy is achieved by removal of stressors, although once established plucking can become obsessive and the problem can be difficult to break. Limited work has been tried with the use of supplemental essential fatty acid dietary administration (oil of evening primrose supplements) which has yielded some positive results.

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# Chapter 5

# Skin Diseases and Treatment of Waterfowl

## ECTOPARASITES

Ectoparasites of importance in waterfowl include ticks, other insects and leeches.

## *OTHER INSECTS*

Other insects of importance in waterfowl are:

- Lice
- Blow flies

### *LICE*

*Cause and pathogenesis*

These wingless insects, are the most common ectoparasites of waterfowl. Only biting (mallophagan) lice have been recorded. Many different species have been identified. Lice tend to be host specific, and their entire lifecycle is spent on the host. Transmission is by direct contact between infected individuals. In general lice infestations are asymptomatic and those such as *Trinoton* spp. or *Anaticola* spp. rarely cause serious disease in waterfowl. The exception is the shaft louse (*Holomenopon* sp. Figures 5.1, 5.2) that may cause the condition 'wet feather disease' (see below). This is where the structure and waterproofing of the feathers is damaged, due to intense irritation and overpreening leading to water soaking of the plumage.

## *BLOW FLIES*

*Cause and pathogenesis*

Blow flies may cause disease, particularly in waterfowl suffering from severe diarrhoea and faecal soiling, such as ducks afflicted with duck viral enteritis (duck plague, a herpes virus). Species such as *Lucilia* (green bottles) and *Phormia* (black

**Fig. 5.1** Swan louse.

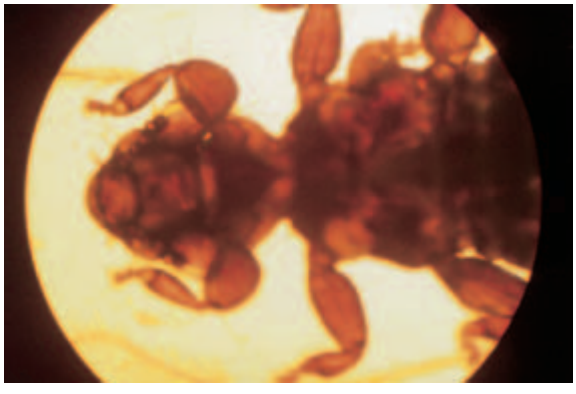

**Fig. 5.2** Close up of head of Swan louse (×20).

bottles) are often implicated. Burrowing maggots may cause serious damage to the vent region.

*Diagnosis and therapy*

Diagnosis is by the presence of maggots. Treatment involves that for shock, secondary sepsis, and the manual removal of any maggots under anaesthesia.

## *TICKS*

*Cause and pathogenesis*

These may be occasionally found attached to the head of waterfowl where they may cause intense oedema. Swelling is thought to be due to a reaction to the toxins in the tick saliva. *Ixodes ricinus* is the tick usually involved in the UK. It may also pass on bacterial, parasitic and viral infections.

*Diagnosis and therapy*

Diagnosis is by the presence of ticks and clinical signs. Treatment is by careful manual removal of any ticks found. Ivermectin at 0.2 mg/kg orally, or percutaneously may be used to prevent further infestation.

## *LEECHES*

*Cause and pathogenesis*

Leeches can be a serious problem around the eye and nares of waterfowl, particularly when debilitated. Those affecting waterfowl are from the Hirudinae, with the duck nasal leech being a particular problem (*Theromyzon tessalatum*).

*Clinical signs*

They may cause conjunctivitis, blood loss, wound infections and in the case of the nasal leech, head shaking and sneezing.

*Diagnosis and therapy*

Diagnosis is by clinical signs and the presence of the leeches. Treatment is by manual removal and the use of ivermectin at 0.2 mg/kg orally, or percutaneously, once.

# SKIN AND FEATHER TRAUMA

## *SKIN WOUNDS*

**Wounds to the head and neck** region are common and are often associated with fishing hooks. In addition, trauma to the dorsal skull and neck region may reveal the tips of the epibranchial bones. These are the most dorsal parts of the hyoid apparatus that articulates with the caudal skull.

**Pedal dermatitis, or bumblefoot** is also common in many species of waterfowl, and is often associated with poor quality standing areas surrounding pools or ponds. Rough, uneven concrete, or excessively muddy ground may lead to abrasion and reduced effective barrier to infection, allowing ulcers and deep-seated soft tissue and bone infections to ensue.

**Intra-specific and inter-specific aggression** can lead to traumatic injuries. Species such as Egyptian geese (*Alopochen aegyptiacus*), and shelducks (*Tadorna* spp.) are often aggressive with other species, and trumpeter swans (*Cygnus buccinator*) should only be kept in single pairs due to intra-specific aggression.

**Breast ulcers** may be seen in debilitated individuals, or those with weakened or damaged legs that are forced to spend large amounts of time lying down. Such conditions include septic arthritis and tenosynovitis (e.g. *Mycoplasma synoviae* infections), avian TB and the renal coccidial parasite *Eimeria truncata* which causes renalomegaly and pressure on the sciatic / ischiadic nerve supplying the legs. Such individuals will obviously need treatment for the causal disease (if possible) before treatment of the breast ulcer.

**Other traumatic injuries** may result from predator attack or the mistaking of a wet road for a strip of water resulting in a calamitous crash-landing (a common problem in swans) or gun shot injuries.

## *WET FEATHER*

*Cause and pathogenesis*

This is where the structure of the feathers is so damaged by surface contaminants such as oil (see p. 69), mud, mould spores or trauma that the integrity of the vane is destroyed. This reduces its ability to repel water, and leads to waterlogging of the plumage. Soiling of feathers may occur due to the lack of fresh shallow water for bathing and preening. Overhanging waterside trees may become contaminated by sooty mould (*Cladosporum herbarum*) and this then falls on waterfowl underneath producing the condition. Shaft lice (see p. 61) may also contribute to the condition. In addition, any disease (e.g. avian TB, duck plague virus etc.) that causes severe debilitation, and so stops preening behaviour, will lead to ragged and unkempt-looking plumage. It should also be noted that young ducklings, artificially reared, are also susceptible to loss of water resilience to their plumage. In the natural state the parents spend large amounts of time preening the duckling, ensuring efficient water proofing occurs. Therefore artificially reared ducklings should only be provided with drinking water, rather than enough to swim in.

*Diagnosis and therapy*

Diagnosis is based on clinical signs together with diagnostic tests to look for infection and ectoparasites. Treatment of wet feather involves removing any surface contaminants using surfactants (see p. 69, on oil contamination), and correcting the local environmental conditions. Where systemic debilitation is present this must be treated as must any infectious or parasitic involvement of the feathers.

## *FROSTBITE*

*Cause and pathogenesis*

This is a common condition in some species of exotic waterfowl, such as flamingos.

*Clinical signs*

Frostbite may be seen on wing tips, or more commonly on the toes. Dry gangrene may then occur, with subsequent sloughing of the extremities, and in severe cases septicaemia may ensue.

*Diagnosis and therapy*

Diagnosis is made on the basis of history of cold exposure and clinical signs. Treatment of frostbite is based on gradual warming of the patient, fluid therapy and the use of vascular stimulants such as Preparation H® (Whitehall Labs) and isoxsuprine (Navicox®, Univet) at 5–10 mg/kg orally SID for 2–3 weeks.

## *PHOTOSENSITIZATION*

*Cause and pathogenesis*

Photosensitization has been reported in waterfowl after consuming St John's Wart (*Hypericum* sp.) (Robinson 1996).

*Clinical signs*

Skin over the legs and feet became oedematous with erythema and pruritus when exposed to sunlight.

*Diagnosis and therapy*

Diagnosis is by clinical signs and by identification of a photosensitising agent in the bird's environment. Treatment is supportive until the toxins have been metabolised and removed from the bird's system. The photosensitising agent must be removed from the bird's environment and exposure to direct sunlight should be avoided.

# BACTERIAL DERMATITIS

## *BACTERIAL PODODERMATITIS ('BUMBLEFOOT')*

*Cause and pathogenesis*

This is common in larger ducks and swans. It usually occurs due to a combination of regular abrasion from poor quality substrate, particularly concrete surfaces, and the possibility of either systemic disease, reducing immunity, and poor nutrition resulting in hypovitaminosis A amongst other things.

*Clinical signs*

Large cavitating caseous abscesses may form over the joints of the distal tarsometatarsus and P1 in advanced cases. Early signs may be discrete corns, or simply cracking of the solar surface.

*Diagnosis and therapy*

Diagnosis is based on history and clinical signs. Treatment is difficult. Surgical debridement of the infected area, with or without implantation of antibiotic PMMA beads (see Chapter Four on Raptor skin diseases and their treatment) may be necessary. As with raptors, if osteomyelitis is present the prognosis has to be guarded. Other topical remedies that have been successfully tried include applying a mixture of dimethylsulfoxide (DMSO) (30 ml), dexamethasone (2 mg) and chloromycetin succinate (200 mg) (or other appropriate antibiotics based on sensitivity testing) (Olsen 1994). This is reapplied three times daily for four to eight weeks. A yielding, less abrasive surface, such as Astroturf® or high pressure butyl rubber is also useful to reduce damage to the plantar surface of the feet.

## AVIAN TUBERCULOSIS

*Cause and pathogenesis*

*Mycobacterium avium* is a common disease in waterfowl. Infection is usually by ingestion of the bacteria in food and water contaminated with such things as waste water discharge, sewerage and manure. It an also be spread by inhalation and direct contact.

*Clinical signs*

Most birds present with systemic signs especially weakness and emaciation. Diarrhoea and abdominal distention (usually due to ascites) can be seen in advanced disease. Cutaneous lesions are very rare. When they occur they usually present as tuberculous lesions on the feet and may lead to lameness. Rarely, skin lesions can also be seen as abscess or nodular growth near eyes, sides of face, base of the bill, wing joints or on legs.

In severe cases euthanasia is indicated.

## OTHER

A condition related to pulpitis (infection of the vascular pulp of the feathers) has been reported in waterfowl. It is seen in birds that have been wing-trimmed, where the hollow calamus of trimmed feathers becomes flooded with water and

**Fig. 5.3** Fungal dermatitis in the necrosis in a Grey Heron's wing.

bacteria. Birds affected may preen themselves excessively and feather follicle infections may result (Suedmeyer 1992).

Skin wounds may become readily secondarily infected with environmental bacteria such as *Pseudomonas* spp. and *Aeromonas hydrophila*. These birds, particularly geese and swans, may show signs of septicaemia, but also oedematous or necrotising skin lesions at the site of entry.

## FUNGAL DERMATITIS

Fungal dermatitis can occur in waterfowl. Infection is often in debilitated animals with an opportunistic fungal organism (Figure 5.3) such as *Aspergillus* spp.

### *CANDIDA* spp.

*Cause and pathogenesis*

Candidiasis is generally an opportunistic infection, normally affecting debilitated birds.

*Clinical signs*

Most commonly Candidiasis presents as an oral / oesophageal infection. However candidiasis is common in sea ducks, such as Eiders, particularly around the eyes and mouth. It has also been reported to cause lesions in the vent region of waterfowl (Bauck 1996).

*Diagnosis and therapy*

A diagnosis can be made on the basis of clinical signs and the presence of oval / rounded, thin-walled budding yeast-like cells and mycelial fragments on scrapes and smears stained with Diff Quik® from lesions.

Topical treatment of lesions may be attempted using clotrimazole (Canestan®, Bayer) or nystatin containing compounds.

## *CLADOSPORUM* spp.

Contamination of the feathers of waterfowl can occur with spores from the sooty moulds (*Cladosporum* sp.) that grow on riverside trees. This may lead to wet feather disease.

## *ASPERGILLUS* spp.

*Aspergillus* spp. growing on feed can produce aflatoxins which may lead to vascular changes in the extremities of young birds producing cyanosis of the skin over the legs and feet. However, this is generally a minor clinical sign in comparison with the more commonly seen liver failure that this toxin causes!

# VIRAL DERMATITIS

## *POXVIRUS*

*Cause and pathogenesis*

Poxvirus infections are seen rarely in waterfowl. Transmission is thought to occur through contact via abraded skin or the conjunctiva. Arthropods may act as mechanical vectors.

*Clinical signs*

The lesions present as pale yellow scabs identified on featherless areas. Lesions are usually on the bill especially at the commissures of the mouth but are also found on the head, legs or feet (usually plantar surface of webs). Lesions will heal with minimal scarring. They can become secondarily infected.

*Diagnosis and therapy*

Diagnosis is made on clinical signs and the demonstration of viral inclusion bodies on histopathological analysis (the so-called Bollinger bodies). Lesions are usually self limiting, resolving within six to eight weeks.

## *OTHER VIRAL INFECTIONS*

### *VIRAL PAPILLOMAS*

Viral papillomas have been identified on the feet and head of waterfowl. The causal agent has not been conclusively demonstrated but is thought to be either a herpes or papilloma virus.

### *ORTHOREOVIRUS*

An orthoreovirus has been reported causing infectious myocarditis in domestic geese. Secondary signs include peeling of the skin at the beak and feet. Goslings are infected between one to three weeks of age and may die, or be left permanently stunted.

In Muscovy ducks, an orthoreovirus different from that seen in geese has been reported as causing feather growth (and physical growth) inhibition at around three weeks of age. Mortality rates approach 90% in infected individuals (Gaudry and Tektoff 1973).

# MISCELLANEOUS CONDITIONS

## *OIL CONTAMINATION*

*Clinical signs*

Oil spills are a depressingly regular occurrence and waterfowl, particularly sea ducks, auks and guillemots, are most frequently affected. The crude oil coats the feathers, destroying their waterproofing and insulative abilities, leading to hypothermia. In addition, much of the damage caused by the oil is due to its consumption by the affected bird. This leads to diarrhoea, secondary digestive tract infections and haemolytic anaemia. Such severely debilitated birds are prime candidates for developing secondary internal fungal infections, such as respiratory tract aspergillosis due to *Aspergillus fumigatus.*

*Diagnosis and therapy*

Diagnosis is made by history of contact and clinical signs. Removal of oil should be performed in conjunction with treatment for the previously mentioned systemic effects of oil poisoning.

*Supportive therapy* includes aggressive fluid therapy for dehydration associated with diarrhoea. This may be performed with lactated Ringer's® solution, preferably administered as bolus doses intravenously, using the medial metatarsal or

brachial veins. Maintenance values for fluids in birds is estimated at 50 ml/kg per day as for cats and dogs, with deficits being similarly calculated and split over two to three days. Haemolytic anaemia may be treated symptomatically with iron dextran (10 ml/kg given i.m.) if the packed cell volume (PCV) drops below 30%, or through blood transfusions if the PCV drops below 15%. Covering antibiotics, or antifungal agents may be necessary to prevent secondary bacterial infections and aspergillosis.

*Oil removal* from the plumage is best achieved using surfactants such as Fairy Liquid™ (Proctor & Gamble). This may be applied direct to the plumage for heavy residues, or as a 2% solution for lighter contamination. It is vital that the water used to flush away the surfactant is kept at the bird's approximate internal body temperature (i.e. as close to 42°C) to reduce the risks of hypothermia. The process should be repeated until all residues of oil have been removed from the plumage. At this stage, water should form beads / droplets on the feathers indicating the return of some water resilience or waterproofing to the plumage. To dry the plumage, hair-driers may be employed, and the bird should be kept in a warmed environment and out of water for a further four to five days for most waterfowl, however sea-going birds may take longer (up to ten to fourteen days) for their plumage to become fully water resilient again.

## *ANGEL-WING*

### *Cause and pathogenesis*

This is not truly a skin problem but an orthopaedic developmental abnormality. It generally occurs in larger waterfowl such as geese and swans. The trigger is thought to be associated to nutrition, specifically with overfeeding due to a diet too high in protein and energy. A relative vitamin E deficiency (too low for the high growth rate) has also been suggested as being relevant.

### *Clinical signs*

One or both wings stick out from the body due to the dorso-lateral rotation of the carpal joint. Although the condition is not life threatening affected birds are unable to fly.

### *Diagnosis and therapy*

Diagnosis is made by clinical signs. If the condition is quickly noticed, it may be possible to strap the wing, with the carpus in medial flexion, and by reducing the protein content of the diet halt the progress of the disease. In the later stages of the disease the changes are irreversible.

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# SECTION TWO

# Dermatology of Reptiles

Chapter 6

# Structure and Function of Reptile Skin

## FUNCTION OF REPTILE SKIN

- Provides physical protection against dehydration, abrasion, UV radiation.
- Aids in control of water, gas and heat exchange.
- Plays a role in social interaction. Lizards of the family Iguanidae, Agamidae, Chamaeleonidae and Gekkonidae have the capacity of colour changes.

The condition of a reptile's skin will reflect its health status and is influenced by environmental and nutritional factors. To understand some of the disease processes and pathology in reptile skin it is important to review normal anatomy and physiology. In many of the disease processes described the integument is not the only organ involved. Systemic disease can have cutaneous manifestation. Multiple small petechial haemorrhages are evident in skin, scales or plastron in cases of septicaemia. Development of ventral oedema is seen in renal and liver disease.

For an overall review on reptile medicine and husbandry veterinarians should refer to the text books in the reference list.

## ANATOMY AND PHYSIOLOGY

### *EPIDERMIS*

Reptile skin is modified into scales. Terminology is dependent on the taxonomic group, size and shape and location on the body, for example *scansors* are scales or lamellae beneath the digits which allow geckos to climb on non-horizontal surfaces (Zug *et al*. 2001). The epidermis consists of three layers (Figure 6.1).

- *Stratum corneum* is the outermost heavily keratinised layer.
- *Intermediate zone* is composed of stratum germinativum cells in various stages of development.
- *Stratum germinativum* is the deepest of the layer. It consists of cuboidal cells.

The stratum corneum can be further divided into the:

- Oberhautchen layer.
- β-keratin layer which is a hard and brittle compound and forms the surface of the scale.

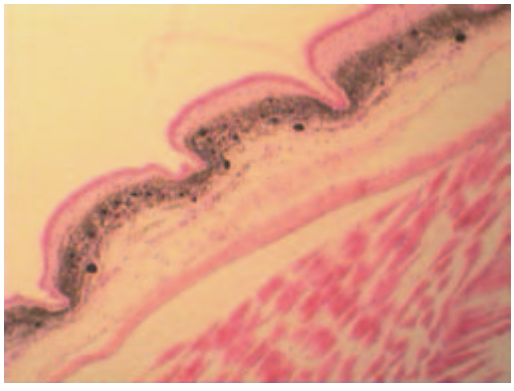

**Fig. 6.1** Cross-section of reptile skin showing epidermis and dermis.

- α-keratin layer which is elastic and pliable and forms the suture between the scales. In soft shell or leatherback turtles the surface of the shell is composed of α-keratin.

## *DERMIS*

- The dermis consists largely of connective tissue interlaced with collagen, fibres, blood and lymphatic vessels, smooth muscle fibres, nerves, numerous chromatophores and a variety of bony structures forming the 'dermal skeleton' (Davies 1981).
- The scales or scutes of chelonia and some lizards (e.g. plated and girdled lizards and skinks) are underlain by bony plates called osteoderms or osteoscutes in the dermis.
- The carapace and plastron, upper and lower shell respectively in chelonia, are fused osteoderms with vertebrae and ribs dorsally and sternum ventrally. In *Heloderma* lizards they fuse with the dorsal skull elements.
- The dermal bone is metabolically active and should be included in weight calculation.

## *GLANDS*

Reptiles possess very little in the way of skin glands but some notable exceptions are as follows:

- Musk or Rathke's glands are present in turtles (except pseudemyd turtles). These bilaterally paired glands lie within the bridge between carapace and plastron. Male tortoises have a mental gland just behind the tip of the lower jaw (Zug *et al*. 2001).
- Some geckos and iguanas have secretory femoral and precloacal pores. These pores do not open until the lizards reach sexual maturity and often only occur in males.

- Some chameleons have temporal glands found in the lateral commisures of the mouth. They have been described as holocrine glands of dermal origin containing an odiferous wax material. They may be used for defensive behaviour turned outward, territorial marking or may attract insects (Klaphake 2001).
- Snakes and autarchoglossan lizards have paired scent glands at the base of the tail; each gland opens at the outer edge of the cloacal opening.
- Some marine and desert species of chelonia and lepidosaurs have salt glands.

## *KERATINOUS STRUCTURES*

- *Claws* are keratinous sheaths that encase the tips of the terminal phalanges (Figure 6.2). They consist of three layers the outermost is formed of β-keratin. The claws form either full keratinous cones as in chelonia or as partial coned as in lepidosaurs.
- *Jaws* – both the upper and lower jaw sheaths of chelonia are also keratinous structures replacing teeth and function for cutting and crushing (Figure 6.3).

## *PIGMENTATION*

Reptiles have two types of pigment-producing cells.

- Melanocytes are scattered throughout the basal layers of the epidermis.
- Chromatophores are stacked in the outer portion of the dermis. These include, from top to bottom, xanthophores (yellow, orange and red pigment), iridophores (white or reflective) and melanophores (black, brown, or red). Stacked chromatophores are absent in some species that do not colour change (Zug *et al*. 2001).

**Fig. 6.2** Lizard showing powerful claws.

**Fig. 6.3** Close up of tortoise's jaw.

# ECDYSIS

## *ENDOCRINOLOGY*

There is an apparent paradoxical effect of the thyroid hormones in lizards (stimulatory) versus snakes (inhibitory), (Maderson 1985).

- In snakes the rest phase is maintained by hormones and the renewal phase is not hormone dependent.
- In lizards the thyroid influences shedding and in thryroidectomised animals shedding ceases (Lynn 1970).

## *SHEDDING PHASES*

The shedding process takes about fourteen-days to complete in lizards and snakes. During this period the affected reptiles may refuse food, hide, become more aggressive and resent handling. Cells of the intermediate zone (upper *stratum germinativum*) replicate and form an entire new three-layer epidermis (called the new inner epidermal generation). Once the new surface is complete, lymph diffuses into the area and enzymatic action results in the formation of cleavage zone, after which the separation occurs.

The shedding process has been described in six distinct phases.

**Resting phase** (Stage one) can take several weeks or several months depending on the nutritional status and age of the reptile.

**Renewal phase** (Stages two to six) lasts approximately 14 days.

- **Stage two** is characterised by proliferation of the *stratum germinativum* producing new daughter cells displacing the outer generation. Differentiation begins in the outer generation layer and the germinative cells begin to divide.
- **Stage three** Cells derived from *stratum germinativum* in the second phase start to keratinise and form the new inner layer or inner epidermal generation and the animal's colour starts to dull.
- **Stage four** The skin is the dullest at this stage and the spectacle is opaque with establishment of the *stratum intermedium* between the inner and outer epidermal generations. Handling should be avoided during this stage.
- **Stage five** The tissue of the *stratum intermedium* breaks down.
- **Stage six** Shedding occurs four to seven days after the spectacle clears. Sloughing eventually occurs when the two epidermal generations become physically separated by the enzyme-induced breakdown of the innermost cell layer of the outer generation and diffusion of lymphatic fluid into the intervening space (Davies 1981).

## *SHEDDING PATTERN*

Ecdysis or shedding of the skin occurs continuously throughout the lifetime of a reptile. The shedding pattern varies between the different reptile groups. The physiology of the shedding process is similar between the reptile groups.

- In chelonia the cells of the *stratum germinativum* divide continuously throughout an individual's life, stopping only during hibernation and torpor (Zug *et al*. 2001).
- Lizards and chelonia mostly shed small pieces. Terrestrial chelonian shed small flakes of the outer shell while aquatic chelonian shed outer portions of entire scutes. It is not possible to determine the age of a chelonian by counting the growth rings created by shedding of carapacial scutes.
- Snakes and some lizards shed their skin (including spectacles) as a single piece. Snakes start shedding at the jaw end first. The snake will crawl out of is skin inverting it back over its own body.

## *SHEDDING FREQUENCY*

Factors that influence the frequency and length of shedding include:

- Age, e.g. young, rapidly growing snakes shed as often as once every five to six weeks. Adults may shed only three to four times a year.
- Temperature is important. Shedding frequency increases with temperature because of a general increase in metabolism (Maderson 1985).
- Nutrition, ambient temperature and humidity will also affect shedding frequency. If these are inadequate they can cause dysecdysis (improper shedding).
- Disease, scarring and hormone imbalance will also affect normal shedding.

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Chapter 7

# Examination of Reptile Skin and Diagnostic Tests

## HISTORY AND HUSBANDRY

A detailed clinical history is important in all cases. This should include information on:

- Captive environment (Figure 7.1).
- Diet.

Knowledge or access to information on captive husbandry is essential when dealing with reptiles. Inadequate husbandry is either a primary cause or a contributing factor of dermatological conditions, as it is with other medical conditions in reptiles.

## CLINICAL EXAMINATION

A full physical examination should be undertaken in addition to a dermatological inspection. Prior knowledge of normal reptile skin is essential. For example, although pruritus can be associated with mite infestations it can also be seen as a natural event when shedding is imminent. Reptile skin may have a dull appearance prior to shedding and some lizards may appear darker during handling or when ill.

Systemic disease can have cutaneous manifestation. Typical lesions include abrasions, blisters, crusts, cysts, petechiation (capillary flushing due to septicaemia), discolouration or a 'swelling'. A differential list for what may appear as subcutaneous swellings includes: abscess, oedema, endoparasites (Helminths), neoplasia, granulomas and bone exostosis.

## COLLECTING LABORATORY SAMPLES

### *FINE-NEEDLE ASPIRATES*

- Fine-needle aspirates from skin masses can be submitted for cytology and microbiology.
- They are a useful means of differentiating between infectious and neoplastic samples.

**Fig. 7.1** A thorough knowledge of the reptile's environmental needs is essential.

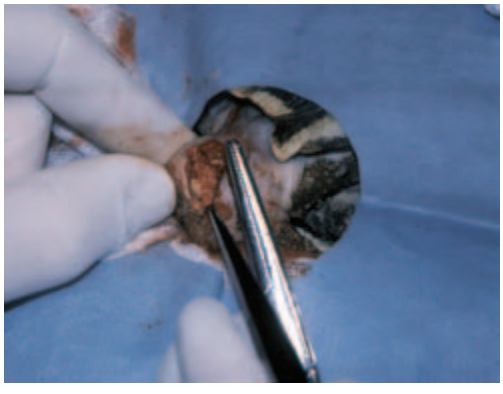

**Fig. 7.2** Skin biopsies should be submitted for histopathology. Sub-epidermal tissue is less likely to be contaminated with surface organisms and is more likely to yield the genuine pathogen on bacterial or micro-bacterial culture. (Picture courtesy S. MacArthur.)

## *SKIN BIOPSY*

Histopathological samples are required for definitive diagnosis of neoplastic lesions, but they are also useful for tissue culture for bacterial or mycobacterial pathogens (Figure 7.2).

### *TECHNIQUE*

- Local anaesthetic (2% xylocaine or lidocaine) can be used to obtain skin biopsies.
- Incisional or excisional biopsies are useful for visible, superficial lesions.
- Punch biopsy tools are not useful for skin biopsies in reptiles with large scales such as snakes, as scales can be damaged or interfere with proper sample collection (Hernandez-Divers 2003). A healthy margin should be included. A suture may be required for skin closure (wound closure and healing takes four to eight weeks).
- For wedge biopsies from chelonian shell a general anaesthetic is needed. The shell is difficult to biopsy or evaluate histologically.

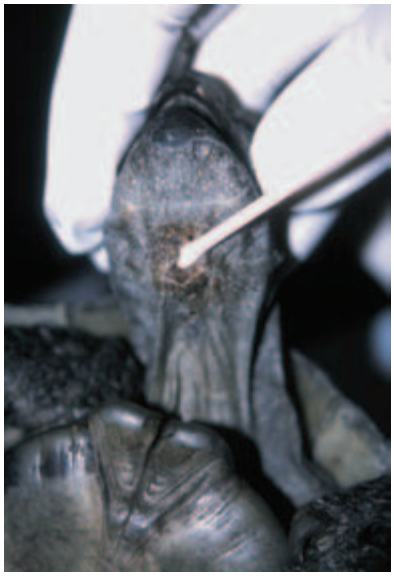

**Fig. 7.3** Skin lesions and infections amenable to bacterial, fungal and mycobacterial culture, cytology and histopathology. (Picture courtesy S. MacArthur.)

- Skin masses can be excised under general anaesthetic and submitted for histology.
- A minimum of two skin biopsies should be taken, one submitted for histology the other for microbiology (ideally, the sample should be ground in a sterile mortar).

### *MICROBIOLOGY*

- Swabs may be submitted from samples (Figure 7.3) collected by fine-needle aspirate, or tissue samples may be taken aseptically at the time of biopsy and sent for tissue culture.
- Interpretation of results can be difficult as the organisms isolated are often found in the animal's environment or are secondary pathogens.
- When handling samples zoonoses are also a potential problem due to Mycobacterium, Salmonellosis, Pentastoma and snake mites.

## DYSECDYSIS (ABNORMAL SHEDDING)

Dysecdysis is usually multifactorial. It is often a symptom of inadequate or improper husbandry.

Factors affecting ecdysis include:

- Environmental temperature and humidity.
- Nutritional status.
- Parasite infections.

- Scars from old injuries.
- Surgery or burns.
- Systemic disease.
- Underlying disease causing dehydration or impairing movement, such as fused vertebrae.

Dysecdysis is more commonly seen in snakes and some species of lizard and is less common in chelonia.

A full clinical history and physical and dermatological inspection are needed to try to establish the cause of the dysecdysis. Further diagnostic tests can be performed depending on the initial history. If left untreated dysecdysis can lead to infection. Retained skin can harbour bacteria, fungi and parasites. Further details are given in species-specific chapters.

## ABSCESSES

Abscesses can be caused by a trauma (bite wound or penetrating wound) or via haematogenous spread (Figure 7.4). The list of bacteria isolated from abscesses is endless and includes *Aeromonas* spp., *Pasteurella* spp., *Staphylococcus* spp. and *Pseudomonas* sp. to name a few. Potentially, zoonotic bacteria have also been isolated such as *Mycobacterium* sp. and *Leptospira* sp. (Frye 1991). Common isolates from abscesses and granulomas include *Pseudomonas*, *Aeromonas*, *Citrobacter*, *Escherichia coli*, *Proteus*, *Salmonella*, *Serratia*, *Neisseria*, *Streptococcus* and *Corynebacterium pyogenes*.

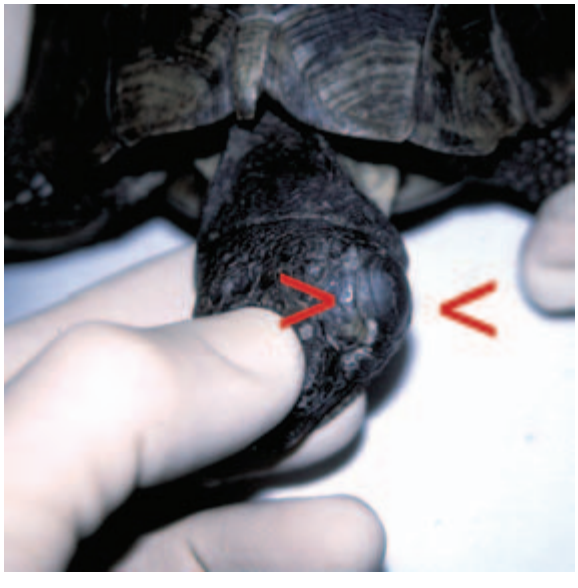

**Fig. 7.4** Lateral swelling of the head in the region of the tympanic scute (arrows) in a spur-thighed Moroccan Tortoise. This is the result of a chronic ear abscess. (Picture courtesy S. MacArthur.)

In iguanas *Corynebacterium pyogenes*, *Serratia marcescens*, *Salmonella marina*, *Micrococcus* and *Neisseria* spp. have been isolated.

Occasionally anaerobes are seen such as *Bacteroides* spp., *Clostridium* spp., *Fusobacterium* spp. and *Peptostreptococcus* spp. (Rossi 1996). The use of alternative antibiotics such as metronidazole should be considered.

### *Clinical appearance*

Abscesses can be found almost anywhere on the body surface but the head and extremities are common sites. These appear as solid subcutaneous swellings under the skin. Due to the semi-solid caseous pus they rarely ulcerate until they become very big. Most have a fibrous capsule. Solitary abscesses rarely cause a problem unless infection spreads and involves bone. Hence abscesses around the head or appendixes should be radiographed.

### *Investigation*

Fine-needle aspirate will help in differential diagnosis. Culture is usually not necessary if the abscess is treated surgically.

### *Therapy*

Abscesses are best treated surgically with complete excision of the abscess where possible with routine wound closure rather than lancing the abscess. Material can be submitted for microbacterial culture and sensitivity and antibiotic therapy initiated until culture and sensitivity results have been received. Preferably bactericidal antibiotics with a Gram negative spectrum should be used (Table 7.1).

## PARASITIC DISEASE

Various parasites may migrate to subcutaneous tissue these include:

- Cestode, *Spirometra*.
- Trematodes, *Spirorchidae.*
- Spirurids.
- Pentastomids are zoonotic and gloves should be worn when handling infected animals. The only treatment available is removal of the larvae.

Since reptiles live close to the ground they may be host or accidental hosts to ticks (Figure 7.5) and it is estimated that 60% of larger lizards and snakes in the wild are infested by ticks.

**Table 7.1** Reptile formulary.

| Agent | Dosage (mg/kg) | Comments |
|---|---|---|
| *Antibiotic* | | |
| Acyclovir | 80 mg/kg p.o. SID for 10 days<br>Topical 5% ointment | Tortoises (herpes virus) |
| Amikacin | 5 mg/kg initial dose followed by 2.5 mg/kg every 72 hours i.m. | |
| Cefotaxime | 20–40 mg/kg i.m. SID | |
| Ceftazidime | 20 mg/kg i.m., s.c., i.v. every 72 hours | |
| Chloramphenicol | 40–50 mg/kg s.c. every 12–72 hours<br>50 mg/kg p.o. SID | Snakes |
| Enrofloxacin | 5–10 mg/kg p.o. s.c. i.m. every 24–48 hours | |
| Metronidazole | 25–40 mg/kg p.o. SID | |
| Marbofloxacin | 2 mg/kg p.o., s.c. every 48 hours tortoise<br>2–10 mg/kg p.o., s.c. SID snakes and other reptiles | |
| Penicillin benzathine | 10 000 IU/kg i.m. 48–96 hours | |
| *Antifungal* | | |
| Itraconazole | 23.5 mg/kg p.o. SID | Lizards |
| Ketoconazole | 15–30 mg/kg p.o. SID 2–4 weeks | |
| Nystatin | 100 000 IU/kg p.o. SID for 10 days | |
| *Antiparasitic* | | |
| Fenbendazole | 25–100 mg/kg p.o. SID | repeat treatment |
| Ivermectin | 0.2 mg/kg p.o. s.c. i.m. repeat for 14 days<br>Topical: 5 mg ivermectin with 1 ml propylene glycol and 500 ml water | NOT CHELONIA |
| *Analgesia* | | |
| Buprenorphine | 0.005–0.02 mg/kg i.m. 24–48 hours | |
| Carprofen | 1–4 mg/kg p.o., s.c., i.m. SID | |

Abbreviations: i.m. = intramuscular, i.v. = intravenous, p.o. = orally, s.c. = subcutaneous BID = twice a day, SID = once a day.

**Fig. 7.5** Tick attached to the foreleg of a recently wild-caught, juvenile tortoise. (Picture courtesy S. MacArthur.)

# ENVIRONMENTAL DISEASE

## *SKIN TRAUMA*

### *ROSTRAL ABRASIONS*

*Cause*

Rostral abrasions are caused by rubbing, pushing or banging into abrasive surfaces within the vivarium (Figure 7.6) or against the vivarium walls. Avoid overcrowding; aggressive cage mates should be separated.

*Therapy*

Mild cases without bone exposure can be treated with environmental adjustment, cleaning topically with povidone-iodine and applying antibiotic ointments followed by a wound dressing such as OpSite™ spray (Smith and Nephew). To prevent rostral abrasions plenty of hiding areas, furnishings (if more than one animal is kept in the vivarium) should be provided. The vivarium should not contain any abrasive surfaces and the glass walls should be made visible to the animal (e.g. plastic film as a visual barrier).

### *TAIL DAMAGE*

*Cause*

Tail tips may become necrotic with a dry and often blackened discoloration. This may be due to avascular necrosis, septicaemia or trauma. To determine the underlying aetiology a full clinical examination, evaluation of general husbandry and blood samples for biochemistry and haematology should be taken. Squamates

**Fig. 7.6** Traumatic lesions resulting from excessive rubbing against the side of its tank during attempts to escape in a Red-Eared Slider Turtle. Water filtration was inadequate and bacterial challenge high.

(lizards and snakes) have scent glands located in the ventral tail base (to be distinguished from hemipenises) that may become enlarged or infected (Funk 1996).

Many lizards, especially geckos and a few snakes, but not chameleons or monitors have the ability to autotomise their tail (caudal part of the tail will break away and in some species regrow).

*Therapy*

The affected part of the tail may need to be amputated cranial to the demarcation line between healthy and necrotic tissue and depending on the cause the animal treated with systemic antibiotics.

## *CONTACT DERMATITIS*

Contact dermatitis can occur with the use of substrates containing aromatic compounds (cedar chips or chlorophyll bedding) and from residues of some cage cleaning products (e.g. bleach or phenol compounds). Rhinitis and other respiratory symptoms may accompany the contact dermatitis (Harkewicz 2001).

## *THERMAL BURNS*

*Cause*

Thermal burns are usually caused by contact with heat sources such as hot rocks, heat mats and light sources. It is unknown why reptiles will remain in contact with heating devices even though they are causing them injuries. Clients should be made aware of this and ensure adequate protection from any direct contact, for example placing heat mats along the sides of the tank rather than the floor of the tank. Thermal burns can look similar to bacterial and fungal dermatitis.

**Table 7.2** Fluid therapy.

| | Snake | Lizard | Chelonia |
|---|---|---|---|
| Volume | 15–25 ml/kg (24–48 hours) | 15–25 ml/kg (24–48 hours) | 15–25 ml/kg (24–48 hours) |
| | 1–2% of total bodyweight | 1–2% of total bodyweight | 2–3% of total bodyweight |
| Route Coelomic | | | |
| s.c. | In loose skin, in snakes and lizards over the ribs. | | |
| i.v. | Ventral tail vein | Ventral tail vein | Jugular vein |
| i.o. | | 20–22 gauge needle is placed in the tibial crest. | Caudal bridge between carapace and plastron. |
| p.o. | Hold the snake vertically and measure the length of the feeding tube (the stomach is positioned in the 2nd third). | The stomach is positioned just behind the caudal edge of the ribs. | Hold chelonia upright and measure the feeding tube to the middle of the plastron. |

*Therapy*

Burn lesions may be superficial or full thickness and if large areas are affected parental fluid therapy should be instigated (see Table 7.2), as the patient will suffer from fluid and electrolyte loss. Topical treatment with wet to dry bandages and antibacterial ointments such as silver sulfadiazine (Flamazine) or povidone-iodine (Betadine) should be incorporated in the bandages (Barten 1996). OpSite™ spray (Smith and Nephew) was found to be the most effective bandage to promote wound healing in garter snakes *Thamnophis sirtalis*. This polyurethane clear film allowed for a bacterial impermeable waterproof dressing, while at the same time allowing for easy monitoring of the wound (Smith *et al*. 1988).

*Pseudomonas aeruginosa* colonises burn wounds and causes fatal complications (Jacobson 1977). Systemic treatment with antibacterials against Gram-negative bacteria or based on culture and sensitivity, as well as pain relief should accompany topical treatment. Once demarcation between healthy and devitalised tissue becomes evident, surgical debridement should be undertaken.

Thermal burns will lead to scarring and scarring may interfere with normal shedding causing dysecdysis.

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Chapter 8

# Skin Diseases and Treatment of Snakes

## BACTERIAL DERMATITIS

### *BLISTER DISEASE*

*Cause and pathogenesis*

Infectious dermatitis in snakes is often called 'blister disease'. It is usually associated with moist bedding and a humid environment (Figure 8.1). The moist integument becomes an entry port for bacteria and fungi and can lead to septicaemia.

*Clinical signs*

Lesions manifest as vesicles or 'blisters'. The blisters are initially fluid filled and can progress to necroses and subcutaneous abscessation. Left untreated septicaemia and death may follow. Branch *et al*. (1998) describe an infectious dermatitis outbreak in a colony of ball pythons (*Python regius*). The infection manifested itself as vesicles on the ventral scales, forming ulcers and followed by systemic diseases.

*Diagnosis and therapy*

Culture and sensitivity taken aseptically from blister fluid is essential to isolate the pathogen involved. Treatment pending culture results consists of daily baths in povidone-iodine, empirical systemic antibiotic therapy and addressing husbandry. Ensure the snake is kept at an optimum temperature and on a dry substrate such as newspaper.

In the case of Branch *et al*. *Proteus vulgaris* and *Pseudomonas aeruginosa* were cultured from the lesions and the snakes treated according to sensitivity results with amikacin, cephalosporins and quinlones systemically. Iodine soaks and topical antibacterial creams were also used (Figure 8.2). *Aspergillus* spp. hyphae were seen on the outer epidermal layer. Only a few snakes survived. Husbandry in this case was considered satisfactory and the ticks found and removed manually from the snakes may have been the entry port to a superficial infection. Stress factors such as handling, transport and gravidity may be contributing factors. The fungal infection may be secondary to the ectoparasites and bacterial dermatitis.

**Fig. 8.1** Blister disease in a Royal Python. (Picture courtesy J. D. Littlewood.)

**Fig. 8.2** Snake in povidone-iodine bath.

## *OTHER BACTERIAL INFECTION*

Not all cases are caused by Gram-negative bacteria as highlighted by a case described by Quesenberry *et al*. (1986). A progressively deteriorating boa constrictor *Constrictor constrictor* with ulcerative stomatitis and nodular skin lesions, did not respond to repeated debridement and lancing of lesions. No bacterial growth was obtained on routine culture but acid fast bacilli were obtained from exudate, later identified as *Mycobacterium chelonei*. On post mortem, lesions were also found in lung. Septicaemic snakes present as inappetant with petechial haemorrhages visable in the skin, especially the ventral scales (Figure 8.3). The snake may show central nervous signs such as incoordination.

## FUNGAL DERMATITIS

*Cause, pathogenesis and clinical signs*

Mycotic dermatitis in snakes tends to present on the lateral and ventral scales (Figure 8.4). Fungi isolated from skin lesions may be secondary invaders or con-

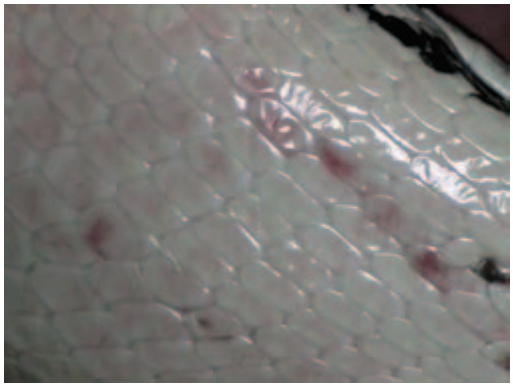

**Fig. 8.3** Petechial haemorrhages in a snake's skin.

**Fig. 8.4** Dermatitis of the ventral scales on a snake.

taminants. Other than the soil inhabitant Trichophyton terrestre (see Table 8.1) *Trichophyton* spp. and *Microsporum* spp. have not been observed in reptiles. Fungal lesions can vary from necrotic, hyperkeratosis with yellow-orange-brown discolouration or dermal granulomas.

Table 8.1 below lists fungi that have been identified in snakes.

*Diagnosis and therapy*

Diagnosis is based on clinical signs and cultures from lesions. Treatment includes addressing underlying causes, removal of necrotic tissue or granulomas, bathing in dilute povidone-iodine and topical treatment with miconazole, ketoconazole or nystatine. Little information is available on systemic antifungals in reptiles except for a study on ketaconazole in Gopher tortoises. Poor response has been reported with the use of griseofulvin.

**Table 8.1** Fungi identified in snakes.

| Fungi | Species reported in | Reference | Clinical signs |
|---|---|---|---|
| *Trichophyton terrestre* | Boa constrictor (*Constrictor constrictor*) | Austwick & Keymer 1981 | Asymptomatic obtained from the normal scales. |
| *Chrysosporium* anamorph *Nannizziopsis vriesii* | Brown tree snakes (*Boiga irregularis*) | Nichols *et al*. 1999 | Skin disease resulted in fatalities in all the affected snakes. |
| *Penicillum* spp., *Oospora*, *Geotrichum* spp., *Fusarium solani* and *Trichoderma* spp. | | Jacobson 1980 | |
| *Geotrichum candidum* | Carpet snake, Burmese python | McKenzie & Green 1976 | Subcutaneous infection. |
| *Monelia sitophila* | Black ratsnake (*Elaphe obsolete*) | | Dull and roughened scales. |
| *Chrysosporium queenslandicum* and *Geotrichum candidum* | Garter snake (*Thamnophis*) | Vissiennon *et al*. 1999 | Skin lesions around the cloaca, lungs and liver. |

## VIRAL DERMATITIS

Epidermal sqaumous papillomas have been identified in common boa *Boa constrictor* and Haitian boa *Epicrates* spp. (Frye 1991).

## PARASITIC DISEASE

Important ectoparasites of snakes are:

- Helminths.
- Mites.
- Ticks.

### *HELMINTHS*

Nematodes parasitising the circulatory and lymphatic systems of reptiles belong to the superfamilies Dracunculoidea and Filarioidea. They affect particularly the subcutaneous connective tissue where they cause tumours, oedema and inflammation (Reichenbach-Klinke & Elkan 1965).

## *DRACUNCULOIDEA*

*Cause and pathogenesis*

The juvenile larvae of *Dracunculus* spp. live free in the water and are taken up by an intermediate host such as a crustacean. In captivity without an intermediate host the infection should be self limiting once infected animals have been treated.

*Clinical signs*

Jacobson *et al*. (1986) describe several snakes with multifocal raised pustular lesions mainly involving lateral body scales. The lesions rupture, drain and eventually develop scabs.

*Diagnosis and therapy*

Impression smears, wet mounts and incised pustules from Jacobson's case revealed numerous nematode larvae. From one of the snakes an entire nematode was recovered and identified as a member of the Dracunculoidea. Infected animals were treated with ivermectin (0.2 mg/kg i.m. and repeated in 14 days).

## *FILARIOIDEA*

*Cause and pathogenesis*

Filarioidae are transmitted by arthropods. In the cases reported there have been little to no clinical signs prior to death.

*Clinical signs*

Dermal lesions are due to occlusion of blood vessels, such as ulcers developing in a gangrenous tail tip with interrupted blood supply (Frank 1981a).

*Diagnosis and therapy*

Microfilariae can be found on thick blood smears.

# *MITES*

The most important mite found on snakes is from the family Macronyssidae and is commonly referred to as the snake mite, *Ophionyssus natricis.* Other less common mites include *O. lacertinus*, *O. mabuyae*, *Neoliponyssus saurarum* and *Ophidilaelaps* spp. (Frye 1991). *Ophioptes parkeri*, *O. tropicalis* and *O. oudemansi* are Myobiidae

mites which live on scales of South American snakes (Reichenbach-Klinke 1965). Reichenbach-Klinke (1965), lists mite species and their hosts.

## *OPHIONYSSUS NATRICIS*

### *Cause and pathogenesis*

*Ophionyssus natricis* a black and shiny blood-sucking snake mite can also be found occasionally on lizards. The lifecycle takes 13–21 days and the mites may live up to 40 days. Mites prefer dark humid conditions and temperatures of 75–85°F. Females lay their eggs in warm, dark, damp places. One female may lay up to 80 eggs in crevices within an enclosure. Eggs may be laid directly on the skin of larger snakes. The easiest place to spot mites other than on the snake is the water bowl. Infested snakes will soak to relieve the itching. This soaking manages to kill a small number of the mites. These dead mites look like pepper that has been sprinkled on the water.

### *Clinical signs*

Heavy infestation can lead to blood loss, dysecdysis, pruritus and possibly transmission of disease such Gram-negative bacterium *Aeromonas hydrophila*. People in contact with mites can present with a skin rash (papular vesiculo-bullous eruption of skin (Schultz 1975)).

### *Diagnosis and therapy*

Diagnosis is made on identification of the mites in the environment or on the snake. Therapy of snake mites is with repeated dosages of ivermectin at 0.2 mg/kg p.o., s.c. or i.m. 14 days apart or topically with Fipronil® (Frontline) or ivermectin spray (5 mg added to 1 ml propylene glycol and 500 ml of water). The ivermectin spray can also be used in the environment. Substrate should be replaced and the cage furnishings and tank thoroughly disinfected.

## *TICKS*

*Aponomma transversale* a hard bodied tick attaches itself to the eye sockets of pythons. Other large African snakes may become infested by *A. latum* (Frank 1981b).

## DYSECDYSIS

*Cause and pathogenesis*

Dysecdysis or abnormal shed occurs if the snake has failed to shed seven days or longer after the final stage (see Chapter Six on Ecdysis pp. 78–79). One cause may be low humidity (50–60% is ideal for most snakes), another is low ambient temperature, stress or skin damage caused by handling, inadequate furnishing to rub against or poor nutrition. Logs and rocks are needed for rubbing to aid in the removal of old skin (Figure 8.5). Medical causes for dysecdysis may be due to bacterial, fungal or parasitic skin infection or systemic disease. Skin lesions such as burns (Figure 8.6) and old scars (Figure 8.7) may impede on normal shedding.

The natural habitats of snakes are roughly divided into desert, temperate and tropical. Rainbow boas (*Epicrates* spp.) and Green tree pythons (*Chondropython viridis*) require high (92–96%) relative humidity. Desert species on the other hand such as sand boas (*Eryx* spp.) require little humidity (<40%) and will develop blisters if the environment is too moist. This disease is commonly seen in Water snakes (*Natrix* spp.), Garter snakes and King snakes (Jacobson 1991). Overgrowth of bacterial pathogens such as *Aeromonas* and *Pseudomonas* as well as fungi occur in moist environment. The fluid-filled vesicles may develop into abscess and cause systemic infection. Retained skin rings in snakes, generally caudal to the cloaca can cause avascular necrosis of the distal tail (Harvey-Clark 1997). To assess if tissue is still viable check vascularity with a needle stick.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs and history especially the snake's environmental conditions. Where infection is present then antibiotic therapy should be given where possible based on culture and sensitivity. To aid in shedding the snake should be bathed in warm water. The snake can be placed in a plastic container or bin depending on body size. A lid can be placed but the animal should be able to hold its head above the water line. The animal should not be left unattended while soaking. Iodine can be added to water if secondary bacterial infection is being treated. The recommended dilution is 1:50 (iodine:water). Once soaked and placed back in the tank, wet towels will allow the snake to rub and remove skin. This may need to be repeated before all the retained skin has been removed. Pieces of skin can also be removed manually using forceps.

Placing snakes and lizards in a pillow case with a clean damp terrycloth towel on a warm (30°C) substrate will result in successful shedding within 24 hours in most cases (Harvey-Clark 1997).

## *RETAINED SPECTACLES*

As mentioned, snakes shed their entire skin as one single piece and this includes the spectacles. The spectacles are clear, fused eyelids that protect the eye and

**Fig. 8.5** Logs and rocks are needed for rubbing to aid in the removal of old skin.

**Fig. 8.6** Burns in a snake may impede normal ecdysis.

**Fig. 8.7** Dysecdysis in a Royal Python. (Picture courtesy J. D. Littlewood.)

should be shed as part of the skin during ecdysis. Knowledge of the normal appearance of the spectacle is important. Ball pythons (*Python regius*) have a dull and wrinkled spectacle that should not be confused with a retained spectacle. Removing a spectacle unnecessarily or incorrectly can lead to damage of the eye. Owners should be instructed to check that the shed skin includes the spectacles. If they are indeed retained, the spectacles can be softened by applying lubricating eye drops (Viscotears® or Liquifilm®) and after applying lubricants several times a day they can be removed using a moist cotton bud or by placing cellophane tape against the eye and gently pulling the tape off (Mader 1996). Retained spectacles can lead to sub-spectacle abscess.

## NUTRITIONAL

Jacobson (1991) describes a collagen disorder seen in reticluated pythons *Python reticulatus* were the epidermis separated from the dermis. The skin was fragile and fluid accumulated between dermis and epidermis in one snake. Chronic malnutrition may have been a factor leading either to protein deficiency and / or hypovitaminosis C (as some species of snake can synthesise vitamin C in their kidneys).

## ENDOCRINE

### *HYPERTHYROIDISM*

*Cause and pathogenesis*

Hyperthyroidism has been identified in snakes and presents as abnormally short shedding cycles. It has been attributed to two factors. The first, a primary thyroid dysfunction and the second, a pituitary dysfunction. Pituitary dysfunction may increase the secretion of TSH or cause failure in the TSH inhibition feed-back loop controlling the secretion of thyroxin.

*Clinical signs*

Excess shedding with short intervals (one to two weeks) has been attributed to hyperthyroidism. On completion of the previous shed the eyes become opaque again. With this continuous shedding, body reserves are used and the animal may become permanently anorectic.

*Diagnosis and therapy*

Diagnosis is by clinical signs and by measurement of thyroid function. Treatment is with Propylthiouracil 10 mg/kg p.o. daily for 21–30 days or Methimazole 1.0–1.25 mg/kg p.o. daily for 30 days. Thyroid function should be determined prior and during therapy. Dosage should be decreased gradually approximately

after four months of treatment. If excess shedding resumes maintenance therapy at the lowest possible dose may be needed (Messonnier 1996).

## NEOPLASIA

*General*

Most reptilian neoplasms behave in a similar manner to the mammalian or avian counterparts; therefore, biological behaviour and prognosis may be estimated in this regard.

*Diagnosis and therapy*

Diagnosis should be made on the basis of clinical signs, fine-needle aspirates or excisional biopsy. Surgery is the treatment of choice with localised skin tumours with recognisable margins. There have been few reports on chemotherapy in snakes.

## *FIBROSARCOMA*

Fibrosarcomas have been reported in boa constrictors *Constrictor constrictor*, Western diamondback rattlesnake *Crotalus atrox*, Prairie rattlesnake *C. viridis viridis*, Timber rattle snake *C. horridus horridus*, a Russell's viper *Vipera russelli* and a black snake *Pseudechis* sp. (Jacobson 1981).

A fibrosarcoma is described as above in a mangrove snake *Boiga dendrophila* with a subcutaneous intermandibular swelling and metastasis to liver. The mass also contained hyphae (Jacobson 1984). Fibrosarcoma is the most common neoplasm reported in snakes and often begins to develop in subcutaneous tissue then metastasis through circulatory and lymphatic systems to visceral organs.

## *LIPOSARCOMA*

A liposarcoma has been identified in a Red-tailed boa *Boa constrictor* (Reavill *et al*. 2002). It presented with multiple, firm subcutaneous masses of variable size randomly distributed over the body. Recommended therapy is wide and aggressive surgical excision. Liposarcomas are usually resistant to radiation and hyperthermia (Reavill *et al*. 2002).

## *SQUAMOUS CELL CARCINOMA*

A squamous cell carcinoma was diagnosed on a Water moccasin *Agkistrodon piscivorus* (Jacobson 1981). The lesion was identified attached to the lower left mandible.

## *MYXOMATOUS TUMOURS*

Myxomatous tumours may be of fibroblastic, chondrobalstic, lipoblastic, myoblastic or neurogenic origin. No viral particles were noted in either cases of Myxosarcoma in a Sinaloan milksnake *Lampropeltis triangulum sinaloae* (Ewing *et al*. 1991) or in Myxoma in a Texas indigo snake (*Lampropeltis triangulum sinaloae*) (Barten & Frye 1981). Both were invasive with the latter also extending to the ribs.

## *MELANOMA*

Multiple skin melanomas have been described in reticulated pythons *Python reticulatus*. Malignant melanomas were seen as black dermal tumours in Pine snakes *Pituophis melanoleucus* and an Everglade's rat snake *Elaphe obsoleta rossalleni*. In both cases despite removal of the primary tumour additional growths, local infiltration and metastasis to various organs occurred.

## *CHROMATOPHOROMAS*

Chromatophoromas are tumours composed either of a single type of pigment containing cells or of a combination of the various types. The first report of a malignant chromatophoroma (melanin-producing) in a Western terrestrial garter snake *Thamnophis elegans terrestris* was reported by Frye *et al*. (1975). The snake had numerous firm nodular swellings along the body. The second report was in a Gopher snake *Pituophis catenifer* (Ryan *et al*. 1981). The original growth was a solitary subcutaneous nodule and it was difficult to remove all neoplastic tissue due to invasion of underlying fascia. The persistent recurrence and local invasiveness suggested malignancy. No metastasis occurred in visceral organs. The third report is in a Pine snake *Pituophis melanoleucus* (Jacobson *et al*. 1989). Malignant chromatophoroma was also diagnosed in a Canebrake rattlesnake *Crotalus horridus atricaudatus* with a subcutaneous orange mass. The tumour was also invasive and complete excision was not possible due adherence to underlying skeletal muscle. A combined iridophoroma and melanophoroma with metastasis of the iridophores in Northern Pine snake *Pituophis melanoleucus melanoleucus* was reported by Jacobson (1991).

# HEREDITARY AND CONGENITAL

Scaleless snakes and snakes with backward-pointing scales are occasionally found. Albinism and melanism occur frequently (Reichenbach-Klinke & Elkan 1965) and are encouraged by breeders. Albinism is observed in all groups of reptiles but more frequently in snakes. In albino snakes melanin is absent but red, yellow and white pigments may be present (Bellairs 1981).

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Chapter 9

# Skin Diseases and Treatment of Lizards

## BACTERIAL DERMATITIS

### *DERMATOPHILUS CONGOLENSIS*

*Cause and pathogenesis*

*Dermatophilus congolensis* is considerd a primary epidermal pathogen and it is thought tick bites, abrasions or pressure to the skin is needed to allow the organism to invade. It is a zoonotic bacterium producing pustular dermatitis or pitted keratitis in man. The cases described in the literature are of animals from commercial dealers and may have been originally wild caught or in contact with wild-caught animals. Their demise was not due directly to *D. congolensis* but rather due to malnutrition or bacterial septicaemias.

*Clinical signs*

Simmons *et al*. (1972) were the first to describe *Dermatophilus congolensis* in a Bearded dragon *Amphibolurus barbatus*. Several lizards within the collection had developed subcutaneous firm swelling on limbs and abdominal wall. Montali *et al*. (1975) also describe multiple golden–brown cutaneous nodules on head, body and extremities of Australian bearded lizards. Anver *et al*. (1976) report cutaneous nodules on trunk and limbs of two Marbled lizards *Calotes mystaceus*. Jacobson (1991) had *D. congolensis* cases in Senegal chameleons *Chameleo senegalensis* and a Green iguana *Iguana iguana*.

*Diagnosis and therapy*

Diagnosis is based on clinical signs, cytology, culture and sensitivity. Treatment for dermatophilus includes removal of encrustations and systemic treatment with penicillin or amikacin.

## *SUBCUTANEOUS ABSCESSES*

*Cause and pathogenesis*

Subcutaneous abscesses occur commonly in pet lizards. They can be caused by (aberrant) parasite migration, bacterial or fungal disease or associated with poor husbandry. Recent work (Huchzermeyer & Cooper 2000) has suggested that in many cases the mass formed is a fibriscess and not a true abscess. In all cases it is essential to identify the underlying cause.

*Clinical signs*

Reptile heterophils are unable to form liquid purulent material as in most mammals therefore reptile abscesses tend to be firm caseated / masses (Figure 9.1). They are often associated with episodes of septicaemia which may have caused infection of internal organs.

*Diagnosis and therapy*

Diagnosis is by clinical signs. Therapy is by lancing and flushing the lesion. Antibiotic therapy can be given concurrently where necessary. In some cases abscesses need to be surgically removed.

## FUNGAL DERMATITIS

*Cause and pathogenesis*

The integumentary system (Schumacher 2003) is the most common site for fungal infections in lizards. Incorrect environmental conditions, overcrowding, malnutrition, prolonged antimicrobial therapy, concurrent disease and immunodeficiency all predispose to fungal infection. Often it is a mixed bacterial and fungal infec-

**Fig. 9.1** Mandibular abscess in an Iguana.

tion. Keratinophylic fungi isolated are often present in the environment such as in the soil, making it difficult to ascertain if they are a primary pathogen, secondary invader or of no pathogenic significance. Pare *et al*. in 2001 conducted a study collecting actively or freshly shed skin from snakes and lizards. Fungi isolated included *Aspergillus*, *Penicillium* and *Paecilomyces*. *Chrysosporium zonatum*, *C. evolceanui* and *Chrysosporium* anamorph of *Aphanoascus fulvescens* were also isolated. There seem to be no difference between arboreal, terrestrial of fossorial reptiles.

*Clinical signs*

Clinical signs commonly start with discoloration of the affected areas and complication in ecdysis (Figure 9.2). Hyperkeratosis is a common result of mycotic infection.

Fungal organisms can produce a wide range of clinical signs (Figures 9.3, 9.4). Table 9.1 describes those cited in the literature.

*Diagnosis and therapy*

Diagnosis is made on the basis of isolation and indentification of fungus. Treatment should be for a minimum of four weeks. Topical antifungals (miconazole,

**Fig. 9.2** A darker colouration in the skin of the Bearded Dragon due to infection.

**Fig. 9.3** Candidiasis in a Bearded Dragon. (Picture courtesy Romain Pizzi.)

**Fig. 9.4** Rostral abrasions in a Chinese Water Dragon. (Picture courtesy Romain Pizzi.)

**Table 9.1** Fungal infections in Lizards.

| Fungal infection | Species recorded | Clinical signs | Reference |
|---|---|---|---|
| Dermatophytosis | Lizards | Crusted lesions. | Pare *et al*. 1997 |
| *Chrysosporium* anamorph of *Nannizziopsis vriesii* | Chameleons, Day geckos, Brown tree snakes, Garter snakes, Pythons and Corn snakes | Systemic disseminated disease, high fatalities. | Pare & Sigler 2002 |
| Cryptococcus | Eastern water skink | Subcutaneous lump on spine. | Hough 1998 |
| *Mucor circinelloides*, *Candida guillermondii*, *Fusarium oxysporum*, and *Aspergillus* spp. | Chameleons | Skin lesion. | Pare *et al*. 1997 |
| *Trichophyton* spp. | Day gecko (*Phelsuma* spp.) | Multiple nodular skin lesions. | Schildger *et al*. 1991 |
| *Trichosporon beigelii* | American anole (*Anolis carolinensis*) | Subcutaneous haematomas. | Jacobson 1991 |
| *Trichophyton terrestere* | Eastern blue-tongued skinks (*Tiliqua scincoides*) | Progressive digital necrosis. | Hazell *et al*. 1985 |
| *Chrysosporium keratinophilum*, *C. tropicum* and *Chrysosporium* spp. | Lizards | Dermatomycosis. | Bryant 1982 and Zwart & Schroder 1985 |

ketoconazole or nystatine) can be used for superficial infection otherwise systemic antifungal should be used (see formulary in Chapter six on reptile dermatology). Antifungal treatment should be accompanied by an appropriate diet and husbandry practices and supportive care if needed. Fungal granulomas should be excised. *Chrysosporium* anamorph of *Nannizziopsis vriesii's* growth is severely restricted at 37°C and raising the ambient temperature may be therapeutic for this infection (Pare & Sigler 2002).

# VIRAL INFECTION

## *VIRAL PAPILLOMAS*

*Cause and pathogenesis*

Viral particles were detected on electron microscopy from papillomas in a Green lizard *Lacerta viridis* (Cooper *et al*. 1982, Jacobson 1991). An outbreak of squamous papillomas in a breeding colony of Bearded dragons *Pogona vitticeps* was attributed to papovavirus (Greek 2001).

*Clinical signs*

Papillomas present themselves as grey, raised spherical dermal masses. In the cases of Cooper and Jacobson, lesions in females were distributed in the area of the tail base and in males around the base of the head. The distribution was presumed to be associated with reproductive behaviour.

*Diagnosis and therapy*

Electron microscopy revealed three morphologically distinct virus particles resembling papovavirus, herpes virus and reoviris. There is no effective treatment available and isolated lesions can be removed surgically but may reoccur: treatment of secondary bacterial infections should be included. In the case described by Greek, the outbreak was eradicated by euthanasia of the colony.

## *POXVIRUS*

*Cause and pathogenesis*

Poxvirus caused clinical disease in a Tegu *Tumpinambis teguexin* (Hernandez-Divers & Garner 2003).

*Clinical signs*

Lesions presented as brown papules.

*Diagnosis and therapy*

Diagnosis is made by electron microscopy. Therapy is not possible other than to ensure supportive care is given to treat any secondary bacterial infection.

## PARASITIC DISEASE

Important ectoparasites of lizards are:

- Helminths.
- Mites.
- Ticks.

Even wild lizards have been identified with parasitic problems. A wild population of Side-blotched lizards *Uta stansburiana* harboured various ectoparasites. Chigger *Neotrombicula californica* was most abundantly found in skin folds around the head, causing inflammatory response at the attachment site. *Ophionyssus natricis* mites were more abundant than *Geckobiella texana*. *Ixodes pacificus* ticks were found in the gular region also causing an inflammatory reaction around attachment sites. Infestation levels were seasonal dependant.

### *HELMINTHS*

*Cause and pathogenesis*

As in snakes endoparasites can manifest themselves externally. *Foleyella furcata*, a microfilariae nematode, was found in the subcutaneous tissue of a Senegalese chameleon *Chamaeleo senegalensis*.

*Diagnosis and therapy*

It was treated with one dose of ivermectin (0.2 mg/kg s.c.) which was ineffective at clearing the infection and caused apparent ivermectin toxicity (Szell *et al*. 2001).

### *MITES*

Mites of importance recognised on lizards include:

- Trombiculidae – chigger mites.
- Macronyssidae – *Ophionyssus acertinus*.
- Pterygosomatidae.

## *TROMBICULIDAE (CHIGGERS)*

### *Cause and pathogenesis*

Mites from the family Trombiculidae or chigger mites are found commonly on lizards and were described by Reichenbach-Klinke and Elkan (1965). Chiggers do not suck blood but ingest lymph and dissolved host tissue. They also secrete a substance which liquefies epidermal cells. The larval stages attach to the host but the adults are free living. They may be important pests in reptile collections causing dermatitis, blood loss and possibly spread of infectious disease. Reported attachment times vary from 7 to 90 days depending on species and area of attachment on host. As an example *Eutrombicula lipovskyana* in mite pockets of Yarrow's spiny lizard *Sceloporus jarrovii* attached for as long as 52 days (Goldberg *et al*. 1993).

### *Clinical signs*

Mites can be found anywhere on the lizard. Chiggers often accumulate around the eyes and mouth.

### *Diagnosis and therapy*

Diagnosis is by identification of the mites. For a treatment protocol to be effective a dual programme must be undertaken to remove parasites from both the lizard and the environment. Therapy of the lizard can be undertaken with repeated dosages of ivermectin at 0.2 mg/kg p.o., s.c. or i.m. 14 days apart for a minimum of six weeks. Ivermectin spray (5 mg added to 1 ml propylene glycol and 500 ml of water) can be used to disinfect the environment.

Pyrethrins, permethrins and pyrethroids are also agents used for environmental control. Cage substrate should be replaced and the tank and its furnishings disinfected.

## *MACRONYSSIDAE*

### *Cause and pathogenesis*

Mites from the family Macronyssidae have been identified on lizards, specifically *Ophionyssus acertinus.* This is red mite and tends to be found in folds of skin around the head and tail.

### *Clinical signs, diagnosis and therapy*

As for Trombiculidae above.

## *PTERYGOSOMATIDAE*

*Cause and pathogenesis*

The Pterygosomatidae family of mites infest lizards. They usually occur in small numbers with only little blood loss. With a few exceptions these mites do not survive long in captivity and prefer a particular host. *Geckobia* on geckos, orange mites usually found in axilla and groin, *Pterygosoma* Agamidae, *Zonurobia, Scaphothrix* and *Ixodiderma* Zonuridae, *Geckobiella* Iguanidae and *Pimeliaphilus* and *Hirstiella* Geckonidae. *Hirstiella trombidiiformis* is a common parasite of Chuckwallas and other desert-dwelling iguanids.

*Clinical signs, diagnosis and therapy*

As for Trombiculidae above.

# *TICKS*

## *IXODIDAE (HARD TICKS)*

*Cause and pathogenesis*

Ixodidae or hard-bodied ticks affecting reptiles include *Amblyomma*, *Aponomma*, *Hyalomma*, *Ixodes* and *Haemaphysalis.*

Hard-bodied ticks Ixodidae found on lizards include *Aponomma hydrosauri* on sleepy lizard *Trachydosaurus rugosus*, *Aponomma exornatum* on Monitor lizards *Varanus niloticus*, *Hyalomma impeltum*, *H. Dromedarrii* and *H. franchinii* infested several lizards *Acanthodactylus boskianus asper*, *A. s. scutellatus* and *Agama mutabilis*. *Ixodes festai* nymphs were found attached to various lizards (*Chalcides ocellatus*, *Agama bibroni*, *Eumeces algeriensis* and *Psammodromus algirus*). *Haemaphysalis otophila* was also found on a lizard.

*Clinical signs*

Mites can be asymptomatic. However they can be important as vectors of disease and when present in large numbers can cause anaemia.

*Diagnosis and therapy*

Diagnosis is by identification of the mites. Treatment is as for mites, see p. 109.

### *ARGASIDAE (SOFT TICKS)*

*Cause and pathogenesis*

Soft-bodied ticks such as *Ornithodoros foleyi* were found on agamids and geckos. *Argas brumpti* is found occasionally on lizards in East Africa (Frank 1981).

*Clinical signs, diagnosis and therapy*

As above for hard ticks.

## DYSECDYSIS / DRY GANGRENE

*Cause and pathogenesis*

Desert lizards such as Leopard geckos (*Eublepharis macularius*) require low environmental humidity but need access to a moist area to allow normal ecdysis. In the wild these geckos would burrow. In captivity a humidity chamber should be provided. A plastic container with an opening for the animal to enter is placed in the enclosure and should contain damp paper towels, moss or vermiculite.

Dry gangrene of the tail and digits has been observed in lizards. This may be a result of avascular necrosis due to dysecdysis, but spontaneous dry gangrene may also occur. Intravascular helminths or bacterial thrombo-embolism may obstruct blood flow to smaller distal blood vessels. Trauma to the tail may appear the same; this tends to be self limiting with the tail dropping off. A progressive ascending dry gangrene has been observed in Iguanas and Bearded dragons. Frye (1991) describes ergot poisoning, a fungal organism growing on cereal grain grasses, as a vasoconstrictor causing dry gangrene in herbivore reptiles.

*Clinical signs*

If left untreated, retained pieces of skin will encircle toes or tail. This can cause constriction and dry gangrene and may necessitate amputation. When lesions occur on the tail, the line of demarcation between viable and necrotic tissue progresses from the tail tip cranially.

*Diagnosis and therapy*

Diagnosis is by clinical signs. Treatment of dysecdysis is similar to that of snakes, by soaking feet or tail and removing skin manually. Treatment may need to be repeated until all retained skin has been removed. In cases of ascending dry gangrene surgical amputation is required. However, following surgical amputation this condition carriers a good prognosis if combined with blood sampling to determine other underlying causes and address septicaemia. Lizards

capable of tail regeneration should not have the tail stump sutured following amputation.

# NUTRITIONAL

## *VITAMIN A DEFICIENCY*

*Cause and pathogenesis*

Vitamin A deficiency has been recorded in Lacertid lizards (Reichenbach-Klinke & Elkan 1965).

*Clinical signs*

The temporal glands in the lateral commisures of the chameleon's mouth may become impacted or swollen. Chameleons present with one or both eyes closed, mouth problems such as stomatitis and may be anorexic or with a decreased appetite. The normal waxy discharge will be foul smelling and green coloured if infected or if an abscess has formed. Some geckos and iguanas have secretory femoral and precloacal pores. These pores do not open until the lizards reach sexual maturity and often only occur in males. The glands can potentially become infected (Figure 9.5).

*Diagnosis and therapy*

Diagnosis is made on the basis of history, especially of an inappropriate diet, and clinical signs. Treatment includes repeated gentle swabbing of the gland area with chlorhexidine solution. Bleeding may occur and is best controlled by applying pressure with a cotton bud. Radiographs should be taken if a gland abscess has

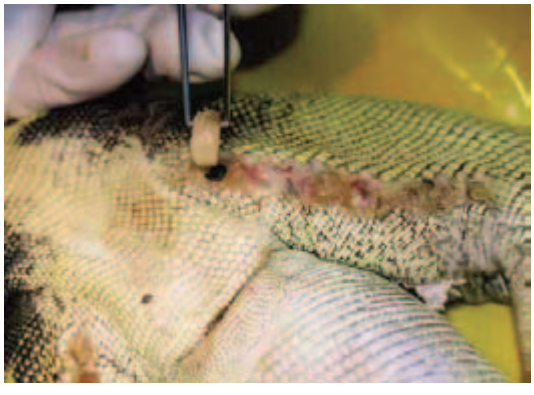

**Fig. 9.5** Infected femoral pores in an Iguana. (Picture courtesy Romain Pizzi.)

formed to rule out bone involvement. Bacterial culture and sensitivity can be useful and the chameleon started on enrofloxacin. As the cause may be multifactorial, husbandry and diet should be assessed and vitamin A supplemented orally (2,000 IU/30 g, of the parental form, p.o. q1 wk twice) as hypovitaminosis A may be involved with temporal gland impaction (Klaphake 2001).

## *HYPOVITAMINOSIS E*

*Cause and pathogenesis*

High-fat diets can predispose to hypovitaminosis E and secondary steatites. Obese monitor lizards *Varanus* sp. are most commonly affected (Harkewicz 2001).

*Clinical signs*

Skin overlying hard fat deposits appears yellow or white. Fat necrosis causes sloughing of the skin resulting in bacterial infection.

*Diagnosis and therapy*

Diagnosis is made on the basis of history and clinical signs. If diagnosed early enough dietary changes and supplementation with vitamin E and selenium can halt the progression of this condition.

# ENDOCRINE

## *HYPERTHYROIDISM*

*Cause and pathogenesis*

Hernandez-Divers *et al*. (2001) describe a case of a functional thyroid adenoma in a Green iguana (*Iguana iguana*).

*Clinical signs*

The iguana presented with loss of dorsal spines, polyphagia, tachycardia and change in behaviour.

*Diagnosis and therapy*

Diagnosis was based on symptoms, ultrasonography of bilobulated mass anterior to the thoracic inlet and elevated total $T_4$ levels (30.0 nmol/litre). Normal $T_4$ levels were suggested at 3.81 ± 0.84 nmol/litre. Treatment was undertaken surgically to remove the adenoma. The reader is referred to the original article for further

**Table 9.2** Tumours in lizards, including clinical presentation where recorded.

| Tumour | Recorded in | Comments |
|---|---|---|
| Squamous cell carcinoma | Sand lizard (*Lacerta agilis*), Common tegu (*Tupinambis seguixin*), Gila monster (*Heloderma suspectum*) | |
| | Veiled chameleons (*Chamaeleo calyptratus*) | Tumour associated with peri-ocular mass, presented with discoloured scales and dysecdysis (Abou-Madi 2002). |
| | Common tegu (*Tupinambis teguixin*) | Lesion on the forefoot. |
| Papillomas | Emerald lizard (*Lacerta viridis*), Wall lizards (*Lacerta muralis* and *L. agilis*) | |
| Melanoma | Gila monster (*H. suspectum*) | (Hernandez-Divers & Garner 2003). |
| Epithelioma | Gila monster | Identified on the foot (Reichenbach-Klinke & Elkan 1965). |
| Liposarcomas | Shingleback skink (*Trachydosaurus rugosus*) | Slow-growing tumour at the base of the tail (Garner *et al*. 1994). |
| | Chameleon | Multiple lumps over the entire body (Reavill *et al*. 2002). |
| Reticulum cell carcinoma | American anole (*Anolis carolinensis*) | Flesh-coloured mass arising from the mandibular fold. |
| Carcinoma planocellulare | Sand lizards (*Lacerta agilus*) | Small nodules in the skin (Jacobson 1981). |

details of the surgical procedure which is described in detail. In a follow up, the spines had regrown with a $T_4$ level down to 3.9 nmol/litre.

## METABOLIC

### *CALCINOSIS*

*Cause and pathogenesis*

Calcinosis circumscripta is defined as ectopic calcification syndrome characterised by calcium deposits in soft tissue.

Cases of calcinosis cutis and calcinosis circumscripta in lizards have been reported (Reavill & Schmidt 2002). Underlying renal disease was present in some cases.

Excessive deposits of chalk in the *saccus endolymphaticus* have been reported in Anolis *Anolis porcatus* and Phelsuma *Phelsuma d. dubia* (Reichenbach-Klinke 1965).

The cause remains unknown.

*Clinical signs*

The lesions ranged from local swellings to multifocal crusty skin thickening, ulceration and sloughing scales with occasional petechial haemorrhage over the body, feet, legs and tail. In some of these cases there was significant underlying renal disease.

## NEOPLASIA

*Cause and pathogenesis*

Table 9.2 details tumours recorded in lizards together with clinical presentation where recorded.

*Diagnosis and therapy*

Diagnosis can be made in some cases by examination of fine-needle aspirate samples or more reliably by biopsy. Where possible, tumours should be surgically removed. Tumours such as liposarcomas require wide and aggressive surgical excision. Liposarcomas are usually resistant to radiation and hyperthermia (Reavill *et al*. 2002).

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Chapter 10

# Skin Diseases and Treatment of Chelonia

## BACTERIAL DERMATITIS

### *LESIONS AFFECTING THE SHELL*

#### *SUPERFICIAL AND DEEP SHELL ABSCESSES*

*Cause and pathogenesis*

Superficial shell abscesses and erosions are common in aquatic chelonia. Often inadequate husbandry is an underlying factor. Deep shell abscesses have a similar pathogenesis.

*Clinical signs*

Deep shell abscesses may penetrate full thickness of the shell, even down to the coelomic membrane.

*Diagnosis and therapy*

Diagnosis is by clinical signs. Therapy for superficial lesions includes careful cleaning and removal of necrotic material from damaged areas of shell. The animal's access to water should be restricted to one hour, twice a day. Topical antibiotic ointment effective against Gram-negative bacteria such as *Pseudomonas* sp. applied after swimming and feeding (Barten 1996).

When lesions are thought to be deep, a radiograph should be taken for assessment and prognosis. In deep lesions debridement under sedation or full anaesthetic is required and a sample submitted for bacterial culture and sensitivity. Treatment for deep lesions is similar to superficial lesions, except topical treatment is accompanied by systemic antibiotics for at least four weeks (Barten 1996).

#### *SEPTICAEMIC CUTANEOUS ULCERATIVE DISEASE (SCUD)*

*Cause and pathogenesis*

SCUD is described as a disease syndrome in aquatic turtles and most common in soft shell turtles (Trionychidae). The agent implicated in this specific disease has

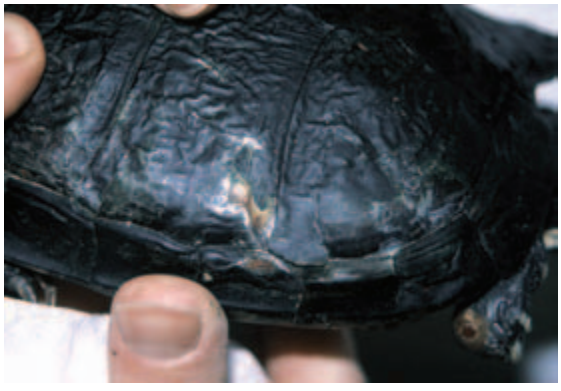

**Fig. 10.1** Bacterial infection of the shell in a Red-Eared Slider Turtle. Bacteria were also apparent in blood smears indicating the animal was septicaemic. The animal had septicaemic cutaneous ulcerative disease (SCUD). (Picture courtesy S. MacArthur.)

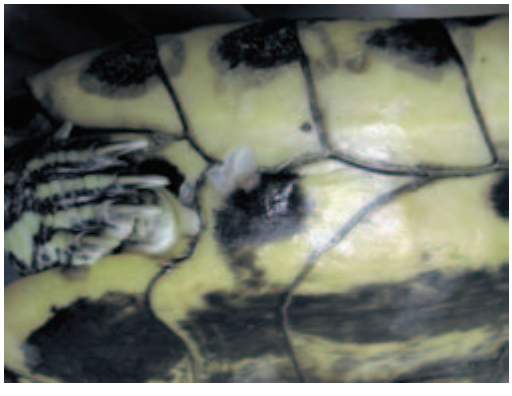

**Fig. 10.2** Lesions such as this are easily sampled for cytology, culture and sensitivity testing. An area of scute is peeling away from the bridge of the animal. The area below is soft and has a pungent smell. This animal has septicaemic cutaneous ulcerative disease (SCUD). (Picture courtesy S. MacArthur.)

been *Citrobacter freundii* but other Gram-negative bacteria can produce similar signs. *Serratia* may be necessary to initiate the infection or as a co-infection. Other factors that have been implicated include poor husbandry, poor water quality, abrasions and invertebrate predation.

*Clinical signs*

The infection leads to the development of irregular, caseated, crateriform ulcers on the plastron, carapace and skin (Figures 10.1, 10.2).

The infection can become septicemic, causing multifocal hepatic and other visceral organ necrosis, hemolysis, limb paralysis and loss of digits or claws. The animal presents with signs of lethargy, anorexia, reduced muscle tone, cutaneous ulcerations or death.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs and bacteriology of lesions. Spontaneous recovery has been reported but prognosis is poor if not treated. Treatment

entails debridement of ulcers and abscesses, use of antibiotics and shell support with fibreglass and resin if destruction is extensive.

Chloramphenicol has reportedly been effective in one case. Shell lesions can take one to two years to heal.

## *OTHER BACTERIAL INFECTIONS*

### *Beneckea chitinovora*

*Beneckea chitinovora* another Gram-negative bacteria is implicated as the cause of chronic ulcerative shell disease in captive turtles especially soft shell turtles *Trionyx* spp. Prior injury to the shell is necessary for the bacteria to cause disease (Wallach 1975). Crustaceans carry these bacteria on their exoskeleton and contaminate water when used as a food source (Harvey-Clark 1997).

### *Mycobacterium kansaii*

A Chinese soft-shell turtle with a white foci on the carapace was initially treated with 20% ammonia (0.5 ml/litre water) in the water. The lesions started to respond to therapy but the animal died following anorexia and respiratory distress. On post mortem, pulmonary lesions were present as well as papular lesions on skin and carapace. *Mycobacterium kansaii* was isolated and identified from the lesions. (Oros *et al*. 2003).

## *BACTERIAL DERMATITIS OF OTHER AREAS*

### *ULCERATIVE DERMATITIS OF THE FEET*

*Cause and pathogenesis*

A captive breeding program for Western swamp tortoises, *Pseudemydura umbrina*, were identified with lesions predominantly affecting the feet. There was no evidence of trauma, *Pseudomonas* spp. was consistently cultured and occasionally secondary fungal hyphae demonstrated. The disease outbreaks were associated with environmental, husbandry and nutritional factors.

*Clinical signs*

Ulcerative lesions were identified on the feet. Death was attributed to secondary septicaemia.

*Diagnosis and therapy*

Diagnosis was made on the basis of clinical signs and isolation of bacteria and fungi. A review of husbandry led to improvements in the tortoises' management. Pond temperatures were maintained between 20°C and 27°C. A successful topical regime was instituted consisting of equal volumes of nystatin, oxytetracycline and water administered topically with a hydrocolloid gel once daily on affected areas for a period of two to three weeks. The animals were left out of the water for an hour after application of topical treatment (Ladyman *et al*. 1998).

## *OTHER BACTERIAL INFECTIONS*

### *Mycobacterium* spp.

*Cause and pathogenesis*

Mycobacterium is a saprophytic organism probably originating from contaminated water. Cutaneous lesions are a likely port of entry with haematogenous spread to internal organs. It is zoonotic causing subcutaneous abscesses when dealing with aquatic environment or animals.

*Clinical signs*

Rhodin and Anver (1977) describe a Side-necked turtle, *Phrynops hilari*, with chronic nodular ulceration on the mandible and nodules on the webbing of the forefeet. On post mortem, nodules were also present on spleen and liver. All lesions had the typical staining of *Mycobacterium* spp. In his review Rhodin also cites mycobacteriosis causing plastral ulcerations in a Nile soft-shell turtle (*Trionyx triunguis*), and a Ganges soft-shell turtle (*Aspideretes gangeticus*).

## FUNGAL DERMATITIS

*Cause and pathogenesis*

Fungal organisms are opportunists, invading tissue already damaged or infected (Figure 10.3). Predisposing factors include poor water quality, malnutrition and stress. The incidence of mycotic infections in aquatic and semi-aquatic turtles may be directly or indirectly related to the pH of the water because the growth of most fungi is inhibited at a pH less than 6.5 (Frye 1991).

In aquatic species, mixed bacterial and fungal infections of the shell are commonly diagnosed and are often invasive involving bone.

Mycosis in chelonians have included infections by *Aspergillus* spp., *Basidiobolus ranarum*, *Beauveria bassiana*, *Cladosporium* spp., *Candida* spp., dermatophytes, *Fusarium* spp., *Geotrichum* spp., *Paecilomyces* spp., *Penicillium* spp. and *Sporotrichum* spp.

**Fig. 10.3** Suspected infected granuloma or fibrisces overlying the mandibular symphysis in a North African Spur-Thighed Tortoise. (Picture courtesy S. MacArthur.)

(Rosenthal & Mader 1996). *Aphanomyces* spp. was isolated from two juvenile Chinese soft-shell turtles (Sinmuk *et al*. 1996). Shell rot of mycotic origin has been ascribed to *Mucorales* spp. (Hunt 1957), *Trichosporon* spp. and *Coniothyrium* spp. (Austwick & Keymer 1981).

*Geotrichum candidum* is a common microflora of many animals and may penetrate through cutaneous abrasions as it does not commonly produce disease.

*Fusarium* spp. is a common soil saprophytes; tank and pool facilities can be a source of infection. The disease progression is slow and does not appear to be life threatening.

*Clinical signs*

Fungal organisms can produce a wide range of clinical signs. Table 10.1 describes those cited in the literature.

*Diagnosis and therapy*

Diagnosis is by clinical signs and isolation of the fungi. Treatment of fungal infections includes the use of topical (miconazole, ketoconazole or nystatine) and systemic antifungals. Table 10.2 lists specific therapy for cases described in the literature.

Ketoconazole at an oral dose of 30 mg/kg given once every 32 hours is maintained at effective plasma concentrations in the Gopher tortoise (*Gopherus polyphemus*). Affected areas should be cleaned and debrided. In aquatic species water quality control is important.

## ALGAE

*Cause and pathogenesis*

Although rarely diagnosed in captive reptiles, infections by several forms of algal organism have been recorded. *Chlorella*, *Basicladia chelonum* or *B. crassa* and

**Table 10.1** Fungal infections in chelonia.

| Infective organism | Affected species | Clinical signs | Reference |
|---|---|---|---|
| Gram-positive cocci and bacilli, *Mycobacterium avium* type B and *Geotrichum candidum* | Galapagos giant tortoise (*Geochelone elephantopus*) | Extensive skin lesions forming ulcerations on head, neck and limbs. | Ruiz *et al*. 1980 |
| *Saprolegnia*-like aquatic fungi | Fresh water aquatic turtles | Cotton-white growths on the skin. | Frye 1991 |
| *Mucor* sp. | Florida soft-shell turtles (*Trionyx ferax*) | Multifocal circular grey lesions involving carapace and plastron. | Jacobson *et al*. 1980 |
| *Aspergillus* sp. (presumed) | Musk turtle (*Sternotherus odoratus*) | Granuloma of forefeet. | Frye and Dutra 1974 |
| *Fusarium solani* | Loggerhead turtle (*Caretta caretta*) | White scaly skin lesions on the neck and head. | Cabanes *et al*. 1997 |
| *Fusarium oxysporum* | | Granulomatous lesions, local tissue infiltration and focal necrosis. | Frye 1991 |
| *Fusarium semitectum* | Texas tortoises (*Gopherus berlandieri*) | Necrotising scute disease. | Rose *et al*. 2001 |

**Table 10.2** Specific therapy for fungal infection in chelonia.

| Infective organism | Treatment | Reference |
|---|---|---|
| *Mucor* spp. | Malachite green baths (0.15 mg/litre three times daily for a week, followed by a fresh water rinse). | Jacobson *et al*. 1980 |
| *Fusarium solani* | Lesions regressed after six months of topical treatment with 10% iodine solution in alcohol and ketoconazole. | Cabanes *et al*. 1997 |

*Schizangiella serpentis* have been implicated. Whether Basicladial infections are authentic pathogens is debatable and there is a form of mutual benefit. The algae provide camouflage for aquatic turtles. Turtles should shed their scutes regularly as they grow. Retained scutes are a sign of poor husbandry as the animal has not been drying off sufficiently to lose its old scutes. Retained scutes will become infected with algae.

*Clinical signs*

Algae can cause pitting or discoloration of the shell. *Schizangiella serpentis* causes granulomatous lesions and should be removed with wide surgical excision as incidence of recurrence is substantial (Frye 1991), followed by topical treatment with ketoconazole ointment.

*Diagnosis and therapy*

Diagnosis should be made on the basis of clinical signs and the identification of the algae on culture. The shells of captive turtles should be cleaned weekly. Where infection has occurred due to excessive wetting of the scutes then the animals' husbandry should be reviewed and a larger basking area should be included in their environment to encourage them out of the water to dry off. Treatment for superficial algal infections is through the removal of algae with a toothbrush and topical application of povidone-iodine solutions or a ketoconozole ointment.

## VIRAL

## *GREEN TURTLE FIBROPAPILLOMAS (GTFP)*

*Cause and pathogenesis*

Fibropapillomatosis also termed green turtle fibropapillomas (GTFP) is characterised by single to multiple histologically benign fibro-epithelial tumours. Green turtles, *Chelonia mydas* and Loggerhead turtles, *Caretta caretta*, in Florida, Hawaii, Caribbean, Australia and Indonesia are affected by cutaneous and occasional visceral fibropapillomas (Aguirre *et al*. 1994, Adnyana *et al*. 1997, Lackovich *et al*. 1999). A viral aetiology remains the main hypothesis. A study in Florida demonstrated that chelonia herpes virus is regularly associated with fibropapillomatosis in sea turtles (Lackovich *et al*. 1999). At least three herpes viruses have been reported in marine turtle fibropapilloma (Quackenbush *et al*. 1998).

GTFP is more prevalent near shore ecosystems and anecdotally more so in areas with human activity impact. Environmental contaminants such as carcinogens may induce latent virus infection or immunosuppresion. Another possibility is that these ecosystems may provide more optimal environment for survival and transmission of infectious agents and a higher population density of vectors and susceptible animals (Herbst & Klein 1995, Adnyana *et al*. 1997).

Fibrous tumours have been observed in Olive ridleys *Lepidochelys olivacea*, Flatbacks *Natator depressus* (Herbst & Klein 1995) and loggerheads *Caretta caretta*, Green turtle *Chelonia mydas*, Hawksbill *Eretmochelys imbricata* and Kemp's ridley *Lepidochelys kempii* (Harshbarger 2002).

*Clinical signs*

The tumours are found on areas of soft skin (flippers, neck, chin, inguinal and axillary regions and tail base) and conjunctivae. Visceral tumours may also be present. Some fibropapillomas will decrease in size but others will slowly increase in size, ulcerating due to abrasions. Fibropapillomas can become life threatening if the size of papillomas impairs swimming and if conjunctival fibropapillomas obscure vision.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs and viral isolation. Therapy is best achieved by surgical removal of fibropapillomas, with wide excisional margins.

## *POXVIRUS*

*Cause and pathogenesis*

A cutaneous pox-like virus infection has been described in a captive Hermann's tortoise *Testudo hermanni* (Oros *et al*. 1998). Poxvirus was also attributed but not confirmed in an amelanotic Californian desert tortoise *Xerobates agassizi* (Jacobson 1991).

*Clinical signs*

In the case described by Oros white–yellow papular lesions were identified on and around the eyelids. In the second case of Jacobson multiple, raised papular to vesicular lesions were seen on the skin.

*Diagnosis and therapy*

Diagnosis is by clinical signs and virus isolation. Isolation of infected animals is advised, to minimise spread and ease treatment and observation, together with treatment of secondary bacterial and / or fungal infection.

## *HERPES VIRUS*

*Cause and pathogenesis*

A virus morphologically similar in appearance to herpes virus has been attributed to be the causative agent of grey patch disease of Green sea turtles *Chelonia mydas*. Epizootics can occur in the summer with high water temperatures, overcrowding and organic pollution (Jacobson 1991). A disease that appears similar is a superficial bacterial dermatitis seen in juvenile Green turtles *C. mydas* and Loggerhead turtles *C. Caretta* (Frye 1991). In addition to viral infection, secondary Gram-negative bacteria were thought to be implicated, as well as secondary hypovitaminosis A.

*Clinical signs*

In Grey Patch Disease young hatchlings in aquaculture developed small papular skin lesions that coalesced into spreading patches. The disease described by Frye (1991) produces a rapidly spreading necrotic dermatitis which is fatal in untreated animals.

*Diagnosis and therapy*

Diagnosis can be made by electron microscopy by the identification of viral inclusions. Reported treatment for the disease described by Frye (1991) consisted of total immersion three times a week in potassium permanganate ($KMnO_4$ 1 g/220 litres of sea-water) followed three hours later by a fresh sea-water wash. Acyclovir has been used for treatment of herpes virus in tortoises.

## *EPIDERMAL SQUAMOUS PAPILLOMAS*

Epidermal squamous papillomas have been identified in Mata mata turtle (*Chelys fimbriata*), Snapping turtle (*Chelydra serpentina*) and Slider turtle (*Trachemys scripta*) (Frye 1991). Papilloma-like viral crystalline arrays were observed in skin lesions of recently imported Bolivian Side-neck turtles (*Platemys platcephala*) (Jacobson 1991).

## PARASITIC

Important dermatological parasites of chelonians include:

- Helminths.
- Mites.
- Ticks.
- Blow flies.
- Leeches.

## *HELMINTHS – SPIRORCHID FLUKES*

*Cause and pathogenesis*

Evidence of infection with spirorchid flukes (Digenea: Spirorchidae) was identified at necropsy in stranded Green turtles, (*Chelonia mydas).* Three species of spirorchid (*Hapalotrema mehrai, H. postorchis,* and *Neospirorchis schistosomatoides*) were identified.

*Clinical signs*

Flukes cause damage to internal organs. Cardiovascular lesions included mural endocarditis, arteritis, and thrombosis, frequently accompanied by aneurysm formation. Occlusion of the superficial blood vessels can occur due to the adults or spirorchid fluke eggs. Thrombosis and infarction of the shell may occur.

*Diagnosis and therapy*

Helminths or ova in shell biopsies are diagnostic. Treatment with praziquantel 8–20 mg/kg i.m. repeated at 14 days (Harvey-Clark 1997).

## *MITES*

*Cause and pathogenesis*

Mites (of unknown species) were found under the dermal scutes of an African spurred tortoise (*Geochelone sulcata*).

*Diagnosis and therapy*

Diagnosis is by clinical signs. Therapy was achieved by debridement of affected tissue and cleansing with chlorhexidine. This lead to an elimination of the mites (Campillo & Frye 2002).

## *TICKS*

*Cause and pathogenesis*

*Aponomma gervaisi* is found on *Testudo elegans* and the coloration of the tick adapted to the host. *Hyalomma aegyptium* infests *Testudo graeca* and *H. franchinii* tortoises in Libya. *Ornithodoros compactus* a soft-bodied tick has been found on tortoises (Frank 1981a).

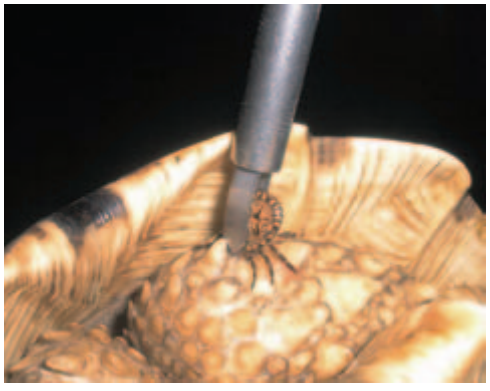

**Fig. 10.4** Manual removal of a tick. (Picture courtesy S. MacArthur.)

Reptile ticks such as the African tortoise tick *Amblyomma marmoreum*, *Amblyomma sparsum* on Leopard tortoises and *Amblyomma dissimile* the Iguana tick have entered the pet trade through imports and are sustainable in warm climates. Some have a mammal as their final host and can transmit infectious disease such as spirochaetes and haemofilaria. Heavy burdens can cause anaemia.

*Diagnosis and therapy*

Ticks can be removed manually ensuring mouthparts are included (Figure 10.4). However, Burridge and Simmons (2001) have developed a protocol to eradicate ticks from animals and facilities. A permethrin product available in the USA (Provent-a-mite™, Pro Products Mahopac, NY) is sprayed topically on tortoises (at a distance of 10 cm, for a one-second burst on each leg of a small tortoise and a two-second burst for a large tortoise) and lizards and snakes are placed into a sprayed container (one-second burst per 30 $cm^2$) once the product has dried and evaporated. The premises is treated at a two-week interval with Cyfluthrin (Tempo™, Bayer Corporation, Kansas City, MO) and in his protocol, Burridge used Hermann's tortoises *Testudo hermanni* as sentinels placed for ten days in the treated facility.

## *MYIASIS – BLOW FLY STRIKE*

*Cause and pathogenesis*

Myiasis in principally caused by *Lucilia* spp. has been found in wounds and areas around the cloaca (Frank 1981b).

*Clinical signs*

Maggots lead to extensive tissue damage at the site of infestation.

*Diagnosis and therapy*

Maggots are physically removed and the area flushed with a dilute antiseptic solution. Topical or systemic antibiotics may be needed.

## *LEECHES*

*Cause and pathogenesis*

Leeches can be found attached to wild-caught chelonia.

*Diagnosis and therapy*

Diagnosis is based on clinical signs and the presence of leeches within the wound. Therapy can be achieved with the use of a saline solution swabbed over the surface of the leech which facilitates removal (Harkewicz 2001). Secondary infections should be addressed.

# NUTRITIONAL

## *HYPOVITAMINOSIS A*

*Cause and pathogenesis*

A common condition seen in turtles and terrapins fed an all-meat diet. Vitamin A is needed for maintenance of epithelial integrity.

*Clinical signs*

Hypovitaminosis A may present itself with bilateral swollen eyelids due to palpebral oedema (Figure 10.5). The hardian glands that secrete sodium chloride lose their structure with squamous metaplasia of the epithelium (Reichenbach-Klinke & Elkan 1965). Other clinical signs include respiratory infection and more general symptoms such as lethargy and anorexia.

*Diagnosis and therapy*

Diagnosis is based on a history of poorly balanced diet and clinical signs. Oral supplementation of vitamin A (2000–10 000 IU/kg feed or 2000 IU/kg p.o. every seven to fourteen days for two to four treatments) is recommended while addressing nutrition and secondary infection.

**Fig. 10.5** Hypovitaminosis A in a Terrapin. (Picture courtesy of J.D. Littlewood.)

## *HYPERVITAMINOSIS A*

*Cause and pathogenesis*

Hypervitaminosis A has been caused by parental injection of vitamin A preparations causing a necrotising dermatitis.

*Clinical signs*

Dry or flaky skin is followed a few days later by severe skin loss and blisters may appear several weeks later (Figure 10.6).

*Diagnosis and therapy*

A diagnosis is made on the basis of history and clinical signs. Lesions should be debrided and cleaned followed by application of a topical antibiotic and a dressing. Depending on the extent and severity of lesions fluid therapy and systemic antibiotic may be needed (Messonnier 1996).

## *BEAK AND CLAW OVERGROWTH*

*Cause and pathogenesis*

Chelonia are often presented with various degrees of overgrown keratinous mouthparts, especially the maxilla. It is presumed that soft-food diets and decreased forage time contribute to this condition. Mandibular overgrowth can cause severe malocclusion and lead to subluxation of the temporomandibular articulation and flattening of the anterodorsal surfaces of the mandibular rami as the geometry and fulcrum point of the bones are altered. Lateral views of the skull should be taken to assess amount to be trimmed.

**Fig. 10.6** Hypervitaminosis A in a Hermann's Tortoise caused by over-administration of Vitamin A by injection. The soft skin of the proximal fore and hind limbs had sloughed and left a moist exudative dermatitis. (Picture courtesy S. MacArthur.)

Claw overgrowth is seen when chelonia lack sufficient abrasive substrate.

Excessive dietary protein is also suspected of contributing to this condition (Rossi 1996).

*Diagnosis and therapy*

Diagnosis is by history and clinical signs. Hard food items should be encouraged to maintain beak wear such as cuttlefish. The beak can be trimmed to normal shape with the aid of a bur. The beak parts should be trimmed to a point slightly caudal to the inner surface of the premaxillary cornice or overhang. In severe chronic cases trimming can be done in stages and should be done aseptically as in chronic cases the bony cranial mandibular rami as well as the medullary cavity and bone marrow within the rami may be encountered (Frye 1991).

## ENVIRONMENTAL

## *HYPOTHERMIA*

*Cause and pathogenesis*

Severe hypothermia can result from heating failure.

*Clinical signs*

Initially hypothermia may not be clinically obvious but within seven to ten days dry gangrene may set in and skin around appendages such as tail and digits may slough due to vascular injury (Figure 10.7).

**Fig. 10.7** Swelling of the limb extremities as a result of freezing (prolonged sub-zero temperature exposure) during hibernation (frostbite). This animal was also blind and circling. (Picture courtesy S. MacArthur.)

*Diagnosis and therapy*

Slow sustained warming is essential as too rapid a rise in core temperature may cause further compromise. Gradual increase in environmental temperature to the species-specific POTZ (preferred optimum temperature zone) over 4–24 hours depending on degree of hypothermia is usually sufficient. In severe cases fluid therapy using warm fluids may also be beneficial. Where irreversible vascular damage has occurred then aseptic surgical amputation proximal to devitalised tissue is recommended.

## *THERMAL BURNS*

*Cause and pathogenesis*

Thermal burns may be acute due to extreme damage such as those induced by overhead heaters. Alternatively, the tortoise may be burnt after finding refuge in a pile of garden rubbish (Figure 10.8). Thermal injury can be much more insidious in nature e.g. the use of under floor heat mats can lead to chronic damage to the plastron. In these cases particularly, the thermal injury does not become apparent until secondary bacterial infection becomes obvious.

*Diagnosis and therapy*

Supportive therapy with fluids should be given where appropriate. Secondary infection is common after burn injuries. Topical therapy with an antibacterial product such as silver sulphadiazine is useful.

**Fig. 10.8** Tortoise with extensive burn lesions.

## *CRUDE OIL EXPOSURE*

*Cause and pathogenesis*

The effects of exposure of juvenile Loggerhead sea turtles (*Caretta caretta*) to weathered crude oil was studied by Lutcavage *et al*. (1995). White blood cell counts increased fourfold and there was a reduction of up to 50% in red blood cell counts.

*Clinical signs*

Oil exposure led to irritant contact dermatitis and caused acute inflammatory cell infiltrates, dysplasia of epidermal epithelium and a loss of cellular architectural organisation of skin layers.

## *TRAUMA*

*Cause and pathogenesis*

Chelonia are often presented with shell damage or fractures due to trauma such as lawnmowers or bite wounds inflicted by dogs, foxes or rats (Figure 10.9).

Radiographs can be taken to assess the extent of the damage. Respiration will continue even if the carapace is breached as movement of skeletal muscles aid in respiration (chelonia lack a diaphragm). Damage to the dorsal midline of the carapace could result in spinal cord lesions as the vertebrae are fused to the dorsal midline of the shell. Paresis and paralysis may result (Barten 1996).

**Fig. 10.9** This Spur-thighed Tortoise was revived from hibernation when raps were heard within its hibernaculum. The right fore-limb had been eaten away leaving the skin as a sleeve. (Picture courtesy S. MacArthur.)

*Diagnosis and therapy*

Diagnosis is made by the history and clinical signs. A fresh, non-infected wound can be sealed with a variety of products such as epoxy resin and fibreglass mesh, (resin should not be allowed to get in between the shell fragments) dental acrylics for beak repair or hoof repair acrylic. Whatever method is used proper apposition is needed and devitalised pieces should be removed.

If the wound is infected it should be first treated topically and the animal systemically and the shell sealed once a healthy granulation bed has developed. Wounds can be covered with bandages. Shell healing can take one to two years and in young growing chelonia grooves should be made along margins of individual scutes with a bur at regular intervals.

## METABOLIC

### *CALCINOSIS CIRCUMPSCRIPTA*

*Cause and pathogenesis*

Calcinosis circumpscripta is defined as ectopic calcification syndrome characterised by calcium deposition in soft tissue.

Yanai *et al*. 2002 describe nodular lesions similar to calcinosis cirumscripta in the subcutis of Malayan swamp turtle (*Cuora ambionensis kamaroma*). In this case it may have been associated with underlying renal failure leading to disorder of calcium or phosphate metabolism.

*Clinical signs*

In Yanai's case, limbs were grossly enlarged and had nodules with a chalky appearance and gritty consistency.

**Fig. 10.10** Dysecdysis, abnormal retention of partially shed skin, in a poorly maintained Turkish Tortoise living in a garden in the northern UK. The tortoise was maintained all year round without supplementary heat or light. (Picture courtesy S. MacArthur.)

*Diagnosis and therapy*

Diagnosis can be made by clinical appearance and histological examination. Histologically there were multiple lobular calcium deposition.

## NEOPLASIA

The following skin neoplasia have been reported:

**Table 10.3** Skin neoplasia in chelonia.

| Neoplastic lesion | Reptile |
|---|---|
| Skin papilloma | Musk turtles (*Sternotherus odoratus*) |
| Squamous cell carcinoma of the foot | Ceylonese terrapin (*Geoemyda trijuga*) |
| Verrucal papillomatosis | European pond turtle (*Clemmys* spp.) |
| Parathyroid adenoma | Red-footed tortoise (*Geochelone carbonaria*) |

## *HEREDITARY AND CONGENITAL*

Minor abnormalities of chelonian shell pattern have been noted (Reichenbach-Klinke & Elkan 1965, Bellairs 1981). These are possibly related to poorly controlled egg-incubation periods.

## *MISCELLANEOUS*

### *DYSECDYSIS*

*Cause and pathogenesis*

Sloughing of scutes has been seen with underlying renal disease and with animals housed in an environment with very moist substrate (Figure 10.10).

*Diagnosis and therapy*

To aid in the assessment of renal function uric acid and calcium : phosphorous ratio in plasma should be measured. Where infection has occurred due to excessive wetting of the scutes, then the animals' husbandry should be reviewed and a larger basking area should be included in their environment to encourage them out of the water to dry off.

Environmental causes are easier to rectify accompanied by systemic antibiotics. Renal disease carries a poor prognosis (Rossi 1996).

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# SECTION THREE

# Dermatology of Fish

Chapter 11

# Structure and Function of Fish Skin

## FUNCTION

- The skin of fish provides a physical barrier and first line of defence against pathogens, osmotic pressure and physical injury.
- The skin acts as a semi-permeable osmotic barrier to maintain ionic and fluid balance.
  - In freshwater fish, it controls influx of water and efflux of electrolytes.
  - In marine fish, it controls efflux of water and influx of electrolytes.

## EXTERNAL FEATURES

The body shape and anatomical features of fish varies between species as an adaptation to their individual habitats and behaviour. The main external features are illustrated in Figure 11.1.

- Barbels: paired sensory structures for touch and taste.
- Nostrils: paired pouches with olfactory receptors for smell.
- Operculum or gill cover: hard bony plate protecting gills and assists in respiration.
- Lateral line: sensory organ along flank and extending onto head its function is to detect sound and pressure.
- Vent: opening to anus, urinary bladder and genital papillae.
- Peduncle: caudal part of body that tapers to caudal fin.

## FINS

- Thin membranous structure supported by multiple flexible 'fin-rays'.
- Vary in shape, size and position, depending on natural lifestyle and shape of the species.
  - Dorsal fin: large unpaired fin on dorsum.
  - Adipose fin: small unpaired fin between dorsal and caudal fin, not always present.
  - Pectoral fin: paired fins, analogous to pectoral limbs.
  - Pelvic fins: paired fins, analogous to pelvic limbs.

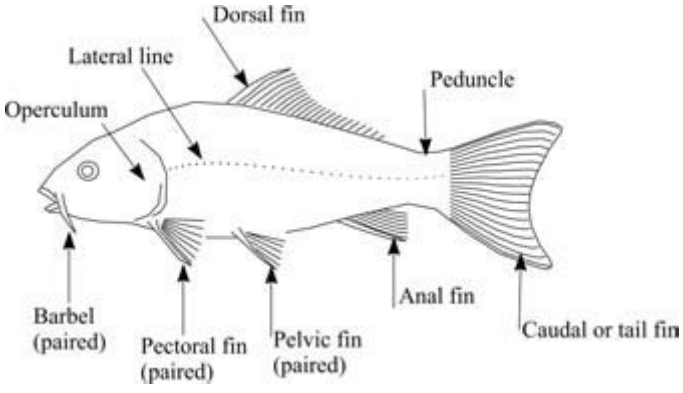


**Fig. 11.1** External features of a fish.

- Anal fin: unpaired fin posterior to the vent.
- Gonopodium: modified anal fin used for fertilisation by males in some species.
- Caudal fin or tail: large fin, usually unpaired but paired in some fancy goldfish.

## STRUCTURE OF THE SKIN

The thickness and cell composition is highly variable and depends on species, life stage, sex, reproductive status, nutrition, body site, season, water quality and general health (Figures 11.2, 11.3).

### *CUTICLE*

- Outer layer: 1 $\mu$m thick, but depends on species and body site.
- Consists of mucus and cell debris.
- Prevents abrasion and reduces friction and water resistance to swimming.
- Contains IgM antibodies and several enzymes with antimicrobial properties.
- Epidermal lymphocytes and plasma cells may form a specific cutaneous immune system.

### *EPIDERMIS*

- Mainly keratinocytes (malpighian cells).
- Stratified squamous epithelium.
  - *Stratum basale* (basal layer): columnar or cylindrical cells.
  - *Stratum germinativum* (germinal layer): oval or round becoming flattened at surface.

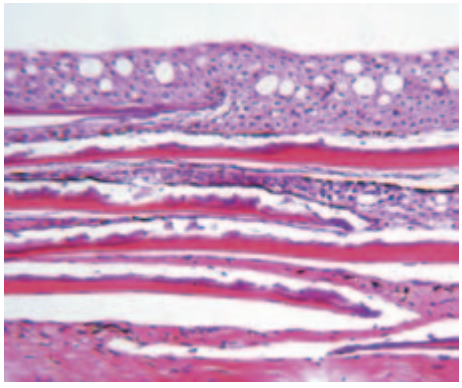

**Fig. 11.2** Histopathological section through fish skin.

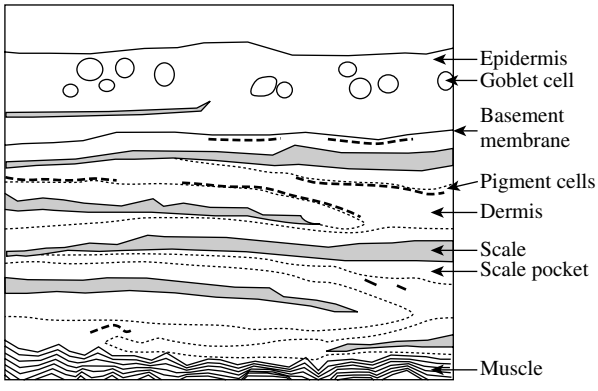


**Fig. 11.3** Schematic diagram to show component parts of fish skin.

- Thickness varies with species, life stage, sex, season and body site.
- Epidermal cells capable of mitotic division at all levels, particularly near basement membrane.
- Keratinisation only in few sites (e.g. breeding tubercles on pectoral fins and head in males of some species).
- 'Goblet cells' (mucus cells) found near epidermal surface and secrete mucus for cuticle.
- 'Club cells' secrete alarm substance and a protective exudate following epidermal injury.
  - In some species, secrete pheromone or *Schreckstoff* ('fear scent') to warn other fish of the same species.
- Some pigment cells and specialised sensory cells (e.g. chemoreceptors, tactile receptors).

## *BASEMENT MEMBRANE*

- Separates the epidermis and dermis.
- Thickness and density varies with species and body site.

## *DERMIS*

- Upper layer, *stratum spongiosum*.
  - Collagen and reticulin fibres forming loose connective tissue.
- Deeper layer, *stratum compactum*.
  - Dense collagen fibres providing structural strength.

## *SCALES*

- Develop in scale pockets within the *stratum spongiosum* and most grow with rest of body.
- Scale growth produces bony ridges which can be counted to estimate age of fish.
- Flexible calcified plates, classified according to size, shape and structure.
  - Cycloid scale: thin translucent circular disc with smooth posterior edge.
  - Ctenoid scale: thin translucent circular disc with irregular toothed posterior edge.
  - Ganoid scale: rhomboid shape with a small 'peg' on the upper surface.
  - Placoid scale: rhomboid shape resembling teeth ('dermal denticles').

## *PIGMENT CELLS (CHROMATOPHORES)*

- Colouration for camouflage and communication.
- Found mainly at boundaries between epidermis, dermis and hypodermis.
- Pigment cells are innervated and some contain more than one pigment.
- Different colorations from acting in combination or by reflection and refraction of light.
  - Melanophores contain melanin and imparts black or brown colour.
  - Xanthophores contain xanthophylls and impart yellow colour.
  - Erythrophores contain carotenoid and impart orange / red colour.
  - Leucophores contain guanine or purine and impart white colour.
  - Iridophores contain guanine and are reflective or iridescent.
- Colour change due to neuroendocrine effects.
  - Adrenergic stimulation (catecholamine) results in lightening or colour loss.
  - Cholinergic stimulation (acetylcholine) causes darkening of skin colour.
  - Melanocyte stimulating hormone (MSH), melanocyte concentrating hormone (MCH) and other hormones affect colour.
  - Water quality, temperature, salinity, mechanical pressure and UV influence coloration.

## *HYPODERMIS*

- Loose fatty tissue layer connects skin to underlying structures (e.g. muscle, bone).
- Loose structure and good blood supply provides common site for infection.

## *WOUND HEALING*

- Excess mucus production around site: blood and inflammatory cells occupy the lesion.
- Initial loss of intercellular attachments between keratinocytes allows rapid migration of cells to cover wound and restore waterproof integrity within 12 hours.
- Results in thinning of surrounding epidermis.
- Initial cell migration occurs independently of water temperature but is inhibited by pathogens and necrotic tissue.
- Later stage healing due to cell division is dependent on ambient temperature.
- Dermal and hypodermal granulation tissue develops over three to four days.

Chapter 12

# Examination of Fish Skin and Diagnostic Tests

The most important procedures used in the investigation of skin disease are outlined below; some or all of these may be required to confirm a diagnosis. Despite the presence of an obvious visible lesion, it is essential to assess the health of the whole fish since skin lesions may be related to systemic disease.

## CASE HISTORY

Detailed examination may reveal a disease pattern which may suggest an underlying cause.

- Sudden illness + all species affected = environmental problem (e.g. poor water quality, poisoning).
- Gradual onset + increasing numbers affected = infectious disease (e.g. bacteria, parasites).
- Isolated cases + small number affected = non-infectious disease (e.g. neoplasia, physical injury).

Interrogate all aspects of husbandry and management, particularly recent changes.

Note the number and species affected, time scale, presenting signs and mortalities.

Note the number and source of new stock and their date of introduction.

## ENVIRONMENTAL INVESTIGATION

Test water quality:

- Always test for ammonia, nitrite, nitrate, temperature and pH.
- For delicate species, test for dissolved oxygen, hardness, salinity, phosphates.

Inspect the environment to assess level of husbandry and for sources of exogenous toxins.

Note changes (temperature, weather conditions, new equipment, new foodstuffs, algal blooms).

High levels of nitrites and extremes of pH cause skin irritation and excess mucus production.

## PHYSICAL EXAMINATION

It is essential to know the normal features of the species under examination. Observe the fish in its own environment to assess abnormal behaviour.

- Monitor respiratory rate, swimming ability and posture.
- Observe interactions with other fish.
- 'Flashing' behaviour (rubbing on rough surface) is a sign of skin irritation.

Detailed clinical examination may require anaesthesia or sedation (see below). Clinical signs of skin disease include colour change, visible parasites or infections, spots, swellings, ulcerations, texture changes and fin lesions.

### *ANAESTHESIA*

Use MS222, the only fish anaesthetic currently licensed in the UK.

Dose rate: 50–200 mg per litre but beware of differences in sensitivity between species.

Method:

- Where possible, starve fish for 24 hours prior to anaesthesia.
- Place fish in small recovery tank with water from original fish tank or pond (has same chemistry and temperature) and aerate with airstones attached to air pump.
- Prepare anaesthetic solution by dissolving measured amount of anaesthetic agent in measured amount of water from original fish tank or pond and aerate with airstones.
- Ensure the pH is the same and buffer with sodium bicarbonate if necessary.
- Transfer fish to anaesthetic solution.
- Observe carefully for loss of reflexes (e.g. aversion to stimuli).
- Remove fish from solution when there is little response to handling (fish may lose balance and roll on to side) but still has some opercular movement.
- Handle fish with care: use wet hands and surfaces.
- May be kept out of water for up to three minutes: repeat immersion to extend interval.
- After examining and sampling, return fish to water in recovery tank.

Anaesthetic agents may reduce numbers of ectoparasites on host.

### *DIAGNOSTIC TESTS*

Gross and microscopic examination is easy to perform and highly diagnostic: this should be part of routine examination, especially since other tests are less specific.

**Fig. 12.1** Skin scraping of fish.

## *SKIN SCRAPES*

Light surface scraping taken using blunt scalpel blade or spatula in caudal direction (Figure 12.1).

- Best sites are behind pectoral fin or operculum or along dorsum near dorsal fin.
- Take more than one sample from a representative number of fish.
- Transfer to clean microscope slide, mount cover slip with drop water from tank or pond.
  - Do not use tap water: may contain chlorine / chloramines.

Examine samples immediately to facilitate identification of motile parasites. Most parasites are visible under ×40–100 but are refractile so use phase contrast or lower the condenser. 'Haystack' formations of *Flavobacterium columnare* colonies are visible under ×100. Mild parasitic burden is not uncommon in fish.

## *GILL SAMPLES*

- Light surface scraping taken using blunt scalpel blade or spatula.
- A small sample of the tips of lamella may be snipped with fine scissors under anaesthesia.
- Mount samples as for skin scrapes.

## *POST MORTEM*

- Perform within one hour of death to minimise autolysis and improve diagnostic accuracy.
- Refrigeration in sealed polythene bag may extend interval of usefulness to a few hours.

- Freezing and defrosting cadavers renders tissues unsuitable for most investigations.
- Preferably sacrifice one affected fish and examine immediately.
  - Euthanase fish by anaesthetic overdose plus cervical dislocation.
- Post-mortem examination should include skin scrapes and gill samples.
  - Various parasites will die quickly or leave following death of the host.

## *BACTERIOLOGY*

Superficial lesions are invariably contaminated with environmental bacteria. Samples from the kidney are only useful where systemic disease is present. *Do not* send swabs by post: samples should be plated onto suitable media immediately.

If necessary, live fish should be sent direct to the laboratory. Culture and sensitivity should be performed by laboratories familiar with fish diseases.

Some (e.g. *Flavobacterium columnare* and *Aeromonas salmonicida*) require specialised media. *Flavobacterium columnare* is best diagnosed by identifying 'haystack' formations on skin scrape.

## *HISTOPATHOLOGY*

This should be considered an important routine investigative procedure.

- Only use samples from freshly killed moribund fish to avoid autolytic artifacts.
- Section of skin with underlying red and white muscle taken from area around lateral line.
- Routine samples also from gill, heart, liver, spleen, bowel and anterior and posterior kidney.
- Biopsy samples taken from tumours and ulcers at junction with normal surrounding tissue.
- Sample all tissues to assess any systemic effects which may influence skin lesions.
- There may be considerable histological change in absence of gross pathology.
- Maximum sample size 1 $cm^3$.
- Fix in chilled 10% neutral buffered formalin: minimum 20 times volume of sample.
- Bouin's or Davidson's solutions may be used for 24 hours prior to storage in 70% ethanol.
- Histological samples should only be interpreted by pathologist familiar with fish diseases.

## *OTHER PROCEDURES*

Diagnostic imaging (radiography, ultrasound) may help identify internal disorders.

- Be familiar with internal anatomy of species under investigation.

Haematology and biochemistry are of limited value due to lack of accurate baseline data.

- Blood samples taken from caudal vein (immediately under vertebrae in peduncle).
- Insert cranially, long needle obliquely between scales just below lateral line.
- Use heparinised needle and syringe (draw up and flush out heparin solution).
- Wide range of 'normal' values between and within species.

Chapter 13

# Skin Diseases and Treatment of Fish

Skin disease in fish is common due to the visibility of lesions and the intimate contact with an environment which significantly influences the health of fish.

Stress due to poor water quality is the most important factor affecting fish health and is responsible for the development of many diseases. Several opportunistic pathogens which are ubiquitous in the aquatic environment may then multiply and overwhelm debilitated and susceptible fish. Skin lesions can be due to primary cutaneous disease or be a manifestation of systemic disorders: they can be complicated by secondary infection and are often fatal due to loss of osmoregulation. Skin lesions are not always specific for a particular pathogen and several groups may cause skin diseases in fish:

- Bacterial.
- Fungal.
- Viral, rickettsial, protozoal.
- Parasitic.
- Miscellaneous.
- Neoplasia.

## BACTERIAL SKIN DISEASES

### *GRAM-NEGATIVE SEPTICAEMIA*

*Cause and pathogenesis*

Mainly aeromonads, pseudomonads and vibrios: most are opportunist pathogens.

*Aeromonas salmonicida* is an obligate pathogen and causes ulceration in koi and goldfish. Vibrios are common in marine fish. Systemic spread of bacteria can occur from skin ulcers, following absorption of bacteria from the gut.

*Clinical signs*

Petechiation and ecchymosis, most visible on ventral surfaces and pale or white areas of body. Localised areas of inflammation occur. Large skin ulcers are often present. Goldfish often exhibit brown pigmented patches on the body or the fins.

*Diagnosis and therapy*

Diagnosis is made on the basis of response to antibiotics and/or bacterial culture of kidney swabs.

Treatment is with antibiotics although bacterial resistance may be significant. Improving water quality and management is important. Vaccination is possible in some species (e.g. salmonids).

## *GRAM-NEGATIVE ULCERATION*

*Cause and pathogenesis*

Mainly aeromonads, pseudomonads and vibrios: most are opportunist pathogens. *Aeromonas salmonicida* is an obligate pathogen and causes ulceration in koi and goldfish: carrier fish may transfer infection (Figure 13.1). Vibrios are common in marine fish.

Bacterial invasion of minor traumatic skin wounds (e.g. from poor handling, parasite infestation) occurs as a result of bacterial enzyme action in dermal tissues.

*Clinical signs*

Ulcerations found on most external sites. Ulcers vary in size and depth: small punctuate lesions may perforate fins, large lesions may erode deep into underlying tissues (e.g. muscle or body cavity).

*Diagnosis and therapy*

Diagnosis is based on clinical signs. Bacterial culture from lesions is of limited value (see previous chapter).

Biopsy may be useful.

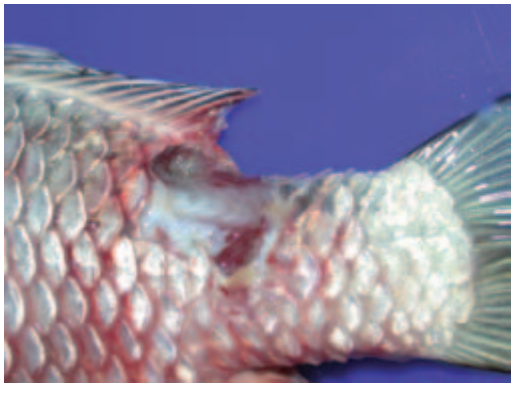

**Fig. 13.1** Ulcer on the surface of the fish.

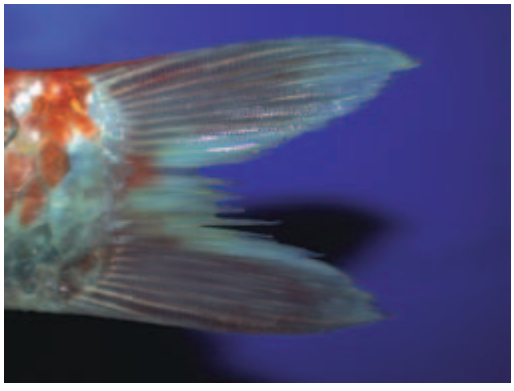

**Fig. 13.2** Fin rot caused by *Flavobacterium columnare*.

Treatment consistes of debridement of necrotic tissues under general anaesthesia, followed by topical disinfection and application of waterproof paste. Antibiotics are useful although bacterial resistance may be significant. Improvement of water quality and management is important. Some species (e.g. salmonids) can be vaccinated.

## *'FIN ROT'*

*Cause and pathogenesis*

Caused by *Flavobacterium columnare* a Gram-negative rod (Figure 13.2). It usually has low pathogenicity, infecting fish following stress but some strains are highly pathogenic and cause disease in absence of stress. Infectivity increases in water temperatures above 20°C. Mortality is high, particularly if gills are affected.

*Clinical signs*

Lethargy, inappetance and increased respiratory rate. Cottonwool-like threads are seen on skin, fins and gills. Small lesions expand rapidly to affect larger areas: ragged fins may necrose completely with total loss of the fin and infection spreading onto the adjacent body.

*Diagnosis and therapy*

Diagnosis is based on clinical signs, microscopy and culture. 'Haystack' appearance of bacterial colonies on microscopic examination of wet mount sample of infected necrotic tissue. Bacterial culture is needed on selective medium (e.g. *Cytophaga* or Shieh agar).

Treatment with antibiotics by injection, in feed or in water. Immersion in solution of potassium permanganate, copper sulphate or proprietary products are useful.

Reduce stress by improving water quality and management practices.

## *'MOUTH ROT' / 'MOUTH FUNGUS'*

*Cause and pathogenesis*

Mouth rot is caused by the bacteria *Flavobacterium columnare* a Gram-negative rod.

It is more common in tropical freshwater fish. It has a high mortality and often progresses rapidly.

*Clinical signs*

Lethargy and inappetance. Grey cottonwool-like lesions are seen around mouth.

*Diagnosis and therapy*

Diagnosis is based on clinical signs, microscopy and culture. 'Haystack' appearance of bacterial colonies on necrotic tissue by wet mount microscopy.

Bacterial culture on selective medium (e.g. *Cytophaga* or Shieh agar).

Treatment with antibiotics by injection, in feed or in water. Immersion in solution of potassium permanganate, copper sulphate or proprietary products.

Reduce stress by improving water quality and management practices.

## *'HOLE IN THE HEAD' (CATFISH)*

*Cause and pathogenesis*

'Hole in the head' disease is caused by the bacteria *Edwardsiella ictaluri* a Gram-negative rod. It mainly affects young catfish.

*Clinical signs*

White ulcers (1–10 mm diameter) on top of head and dorsum. It may cause gastrointestinal septicaemia and death.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs and bacterial culture. Treatment is by antibiotics by injection, in feed or in water. Reduce stress by improving water quality and management practices.

## *MYCOBACTERIOSIS*

*Cause and pathogenesis*

Causal organisms include *Mycobacterium marinum, M. fortuitum*, acid-fast Gram-positive rods. It is common in freshwater aquarium fish. This is a zoonotic infection, producing granulomatous nodules on hands and arms of fish-keepers.

*Clinical signs*

Ulcerations on body, which are slow to respond to antibiotic treatment. Occasionally grey or white caseous discharge from small ulcers. Loss of normal body coloration. Other signs (e.g. anorexia, weight loss, lethargy, body deformity, incoordination).

*Diagnosis and therapy*

Diagnosis is made by clinical signs, i.e. cutaneous lesions plus presence of granulomas found throughout internal organs. Histology of affected areas and internal organs (acid fast organisms occasionally found). Bacterial culture on selective medium (e.g. Löwenstein–Jensen agar) is necessary.

No effective treatment is recorded, although some reports in literature advocate rifampicin, erythromycin, streptomycin, kanamycin, doxycycline and minocycline.

Destroy affected fish and disinfect system.

# FUNGAL SKIN DISEASES

## *SAPROLEGNIASIS*

*Cause and pathogenesis*

*Saprolegnia* is a non-septate oomycetous fungus (water mould). It commonly infects damaged skin and gills in freshwater fish. It is usually a secondary pathogen and is often an indication of poor water quality and husbandry. Infection of the gills and extensive dermal lesions are usually fatal due to osmoregulatory failure.

*Clinical signs*

Delicate fungal structure resembles tufts or mats of white cotton wool (Figure 13.3). It may appear green or brown in outdoor fish due to trapped algae or silt, respectively.

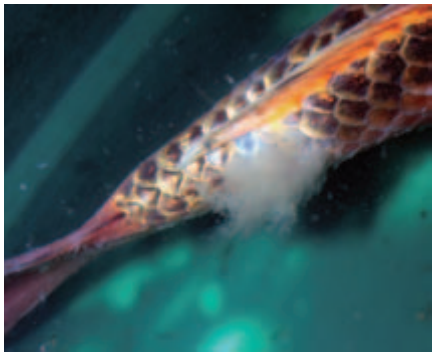

**Fig. 13.3** Cotton wool-like tufts of *Saprolegnia* fungus on a fish.

*Diagnosis and therapy*

Diagnosis can be made on the basis of clinical signs.

- Fungal hyphae and fruiting bodies (zoosporangia) visible by wet mount microscopy (Figure 13.4).
- Culture on corn meal or Sabouraud dextrose agar. Do not confuse for 'mouth fungus' or 'mouth rot' caused by *Flavobacterium columnare*.
- Euthanase severely affected fish to reduce zoospore burden in water. Use malachite green in water as medication.

Improve water quality and husbandry, avoid underlying bacterial and parasitic disease.

## *DERMOCYSTIDIUM KOI*

*Cause and pathogenesis*

*Dermocystidium koi* only affects carp and is a protistan organism which has a fungal-like appearance. It is of unknown pathogenesis, producing smooth raised swelling with little inflammation. Lesions rupture at about 10 mm size, exposing hyphae and releasing thousands of spores. Most commonly seen between May and July in UK.

*Clinical signs*

Smooth, raised nodules which resemble 'carp pox' but which enlarge and eventually rupture. Strong, thick, white 'hyphae' of irregular diameter containing thousands of spores.

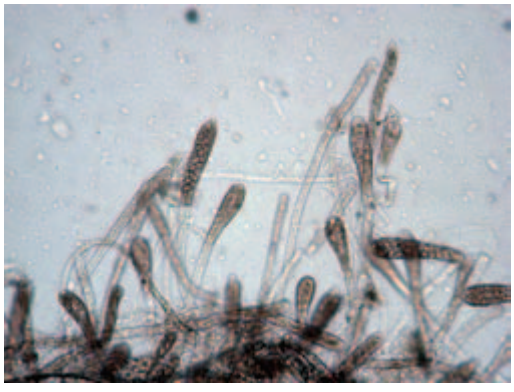

**Fig. 13.4** Wet mount microscopy of *Saprolegnia.*

*Diagnosis and therapy*

Diagnosis may be made on the basis of clinical signs and biopsy. Biopsy reveals spores (6–15 $\mu$m) with 'signet ring' appearance, a large cytoplasmic vacuole displacing the nucleus.

There is no known treatment but antibiotics may avoid secondary bacterial infection. Surgical excision is possible but most lesions heal spontaneously after rupture.

## *ICHTHYOPHONUS*

*Cause and pathogenesis*

*Ichthyophonus hoferi* is a fungus-like organism which is an obligate parasite causing chronic systemic granulomatous disease. It mainly affects marine fish.

*Clinical signs*

Dark raised dermal granulomas, up to 1 mm in size. There is erosion of overlying epithelium which produces sandpaper-like texture.

*Diagnosis and therapy*

Diagnosis is established by biopsy. No effective treatment is available, euthanasia is necessary in many fish.

# VIRAL, RICKETTSIAL AND PROTOZOAL DISEASES

## *CARP POX*

*Cause and pathogenesis*

Cyprinid herpes virus 1 (*Herpesvirus cyprini*) is an infection of epidermal cells producing papilloma. It mainly affects carp but is also found on other cold-water species and some tropical fish. It has low morbidity with only a few infected fish exhibiting lesions.

Infected cells begin to lyse at temperatures >20 °C with inflammation and sloughing.

*Clinical signs*

Smooth raised white lesions on body and fins, resembling drops of candle wax. They may form large plaques and occasionally be pigmented.

*Diagnosis and therapy*

Diagnosis is made by biopsy. No known treatment is available but lesions often regress spontaneously after several months.

## *SPRING VIRAEMIA OF CARP (SVC)*

*Cause and pathogenesis*

*Rhabdovirus carpio* is a notifiable disease in UK. It affects carp, goldfish, orfe, pike, roach, rudd, tench and Wels catfish.

Occurs during rising water temperatures (7–15 °C) in springtime. Mortality may vary from 10–100%.

*Clinical signs*

Generalised signs of septicaemia, including petechial haemorrhages and darkening of skin. Often lethargy, abdominal swelling and exophthalmos.

*Diagnosis and therapy*

Diagnosis is made by virus isolation, immunofluorescence and ELISA tests, performed at government laboratories.

No known treatment is available although antibiotics may reduce secondary disease. Surviving fish may become carriers. Slaughter and disinfection is required to contain spread of disease in commercial facilities.

## *LYMPHOCYSTIS*

*Cause and pathogenesis*

Iridovirus, produces massive enlargement of dermal fibroblasts. It mainly affects tropical marine fish, commonly angelfish, butterflyfish and clownfish.

*Clinical signs*

Nodules of 1 mm in size often form clusters up to 1 cm across.

*Diagnosis and therapy*

Diagnosis is made by biopsy. No known treatment is available but lesions often regress with improved nutrition and environment.

## *EPITHELIOCYSTIS*

*Cause and pathogenesis*

This is a *Chlamydia*-like organism which infects epithelial cells in marine and freshwater fish.

*Clinical signs*

Small, white nodules up to 1 mm in size on skin but more commonly on gills.

*Diagnosis and therapy*

Diagnosis is made by histology of lesions. No known treatment has been documented.

## *PROTOZOANS*

*Clinical signs*

General lethargy, anorexia and death. Excess mucus ('slime disease') produces dull coloration and grey-blue sheen to skin. White spots on skin (up to 1 mm). Skin irritation causing fish to 'flash' or rub body against submersed objects.

*Diagnosis and therapy*

Diagnosis can be made by the microscopic examination of wet mount skin scraping. Treatment can be undertaken in the water with various chemicals and proprietary medicines.

*Cause and pathogenesis*

**Table 13.1** Protozoan parasites of fish.

| | Common name | Species affected |
|---|---|---|
| **Ciliates** | | |
| *Ichthyophthirius multifiliis* | Ich white spot | Freshwater |
| *Cryptocaryon irritans* | Marine white spot | Marine |
| *Chilodonella* spp. | | Freshwater |
| *Brooklynella hostilis* | | Marine |
| Trichodinids (*Trichodina*, *Trichodinella*) | | Freshwater and marine |
| *Tetrahymena corlissi* | Guppy killer | Freshwater |
| *Uronema marinum* | | Marine |
| **Flagellates** | | |
| *Ichthyobodo necator* | Costia | Freshwater |
| **Dinoflagellates** | | |
| *Piscinoodinium pillulare* | Velvet | Tropical freshwater |
| *Amyloodinium ocellatum* | Coral fish disease | Marine |

## *'WHITE SPOT'*

*Cause and pathogenesis*

*Ichthyophthirius multifiliis* ('ich') is a ciliate protozoan parasite in freshwater fish. A free-swimming infective trophont penetrates the epidermis and develops into visible white spot. It causes high mortality at high water temperatures. *Cryptocaryon irritans* produces a similar disease in marine fish.

*Clinical signs*

Clinical appearance of gross lesions: white spots up to 1 mm on all body surfaces and gills.

Excess mucus produces grey–blue sheen to skin.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs and by identification of parasites, which are visible by wet mount microscopy.

In-water treatment with various chemicals and proprietary medicines (N.B. only the free-swimming stage is responsive to treatment, therefore infected fish require repeated treatments).

## *'HOLE IN THE HEAD' (CICHLIDS)*

*Cause and pathogenesis*

*Spironucleus* spp. a flagellate parasite. May also involve nutritional deficiency, environmental stress or bacterial infection. Causes low mortality and commonly affects cichlids, particularly oscars and discus.

*Clinical signs*

Shallow ulcerations in sensory pits of head and along the flanks (lateral line).

*Diagnosis and therapy*

Diagnosis is made by clinical appearance of gross lesions in particular species. Parasites may be visible by wet mount microscopy.

Treatment is possible with metronidazole in food and in water but may also require other systemic antibiotics. It is important to improve nutrition, water quality and management.

## *'TET DISEASE'*

*Cause and pathogenesis*

*Tetrahymena corlissi* is a ciliate protozoan which infects freshwater aquarium fish including guppies ('guppy killer') and other live-bearers.

*Uronema* sp., is the marine equivalent but this causes ulceration in later stages.

*Clinical signs*

Discrete, pale white patches due to excess mucus and focal necrosis particularly around eyes. Abdominal swelling is often present.

*Diagnosis and therapy*

Parasites are visible by wet mount microscopy.

In-water treatment with various chemicals and proprietary medicines.

## *MICROSPOREANS*

*Cause and pathogenesis*

*Pleistophora hyphessobryconis* causes 'neon tetra disease'. *Glugea* spp. can also lead to clinical disease.

*Clinical signs*

*Pleistophora hyphessobryconis* infects muscle tissue, producing grey or white patches under the skin on the dorsum of various aquarium fish. *Glugea* spp. produce large hypertrophied cells or xenomas, white nodular structures several millimetres in size within the skin and other organs.

*Diagnosis and therapy*

Diagnosis is made on the basis of biopsy. There is no known treatment.

# PARASITIC DISEASES

## *FISH LOUSE*

*Cause and pathogenesis*

*Argulus* spp. is a freshwater branchiurian parasite. It will move around on the surface of the host and attach with a stylet to allow feeding on blood. Parasites can swim freely in water and lay eggs in the environment.

*Clinical signs*

General skin irritation with flicking and rubbing of the body on submersed objects. Small localised areas of inflammation at the site of attachment.

*Diagnosis and therapy*

Clinical signs: a round flat parasite with a semi-transparent body, measuring up to 7 mm.

Treatment can be undertaken using Program® as in-water treatment. Organophosphates may also be used as in water treatment.

## *'ANCHOR WORM'*

*Cause and pathogenesis*

*Lernaea cyprinacea* is a freshwater copepod which attaches to its host with large cephalic process. Only the female is parasitic and eggs are laid in the environment.

*Clinical signs*

Some skin irritation with small localised areas of inflammation at the point of attachment. Occasionally lethargy due to secondary infection.

*Diagnosis and therapy*

Clinical signs: a narrow straight-bodied parasite up to 15mm long with a large pair of white trailing egg sacs. Treatment can be undertaken by careful manual removal of parasite under general anaesthesia. Alternatively organophosphates may be used as in water treatment.

## *LEECHES*

*Cause and pathogenesis*

Several species have been identified. *Piscicola geometra* is a common freshwater leech.

*Clinical signs*

There are few clinical signs but occasionally these are small areas of inflammation at the point of attachment.

*Diagnosis and therapy*

Visual examination: a brownish, elongated parasite with a large suction pad at one end, actively moving with a contractile action.

Treatment is by manual removal of parasite, aided with 3% salt water dip. Organophosphates may be used as an in-water treatment.

## *MONOGENEANS / 'FLUKES'*

*Cause and pathogenesis*

*Gyrodactylus* spp., *Dactylogyrus* spp. in freshwater fish *Benedenia* and *Neobenedenia* sp. in marine fish.

**Fig. 13.5** Gyrodactylus, a skin fluke in a wet mount skin scraping.

*Clinical signs*

Excess mucus ('slime disease') produces dull coloration and grey-blue sheen. Skin irritation causing fish to 'flash' or scrape body against submersed objects.

Corneal opacity and ulceration is common in infected marine fish.

*Diagnosis and therapy*

Microscopic examination of wet-mount scraping from lesions (Figure 13.5). To treat it is important to improve water quality and management.

In-water treatment may also be used with various chemicals and proprietary medicines. Mebendazole in freshwater fish, praziquantel or freshwater dips in marine fish.

## *'BLACK SPOT'*

*Cause and pathogenesis*

Metacercariae of digenean flukes (*Neascus*) migrate to musculature, encyst and stimulate local melanocytes under the skin.

*Clinical signs*

Brown-black spots up to 0.5 mm on body.

*Diagnosis and therapy*

Diagnosis is by histology of the lesions. No known treatment has been described, although disease has low mortality. Control snails and final hosts (piscivorous birds, fish or mammals).

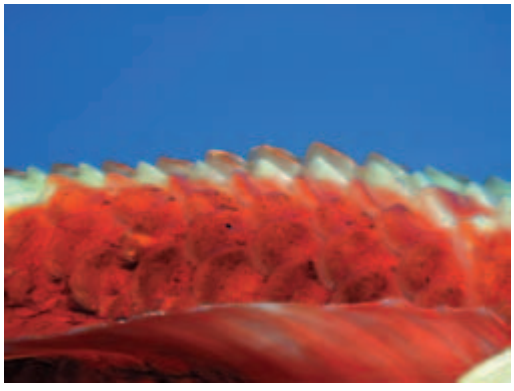

**Fig. 13.6** Raised scales due to oedema of the skin in a fish.

## MISCELLANEOUS DISEASES

### *RAISED SCALES*

*Cause and pathogenesis*

'Dropsy' is a common term used to describe this generalised condition. It is caused by internal diseases with loss of osmoregulation (e.g. diseases of gills, heart, liver and kidneys) resulting in severe dermal oedema (lepidorthosis). Localised areas can be related to localised bacterial infection and prelude to skin ulceration.

*Clinical signs*

Raised scales due to oedema of the skin: produces 'pine cone' effect (Figure 13.6).

Abdominal swelling may be present. Exophthalmos (affecting one or both eyes) may occur due to retrobulbar effusion.

*Diagnosis and therapy*

Diagnosis may be made on the basis of clinical signs.

Treatment is not often possible as most underlying causes are incurable (e.g. neoplasia, organ failure, mycobacteriosis).

### *EPIDERMAL HYPERPLASIA*

*Cause and pathogenesis*

Unknown aetiology: possibly related to carp pox in coldwater pond fish.

*Clinical signs*

Smooth, milky white plaques up to 1 mm thick on body or fins. These are common around the mouth and most visible on dark coloured skin.

*Diagnosis and therapy*

Clinical signs in that hyperplasia is resistant to scraping, unlike mucus. Biopsy is also useful to confirm. No treatment has been described, lesions may spontaneously regress after several months, particularly in warm water.

## *HEAD-AND-LATERAL LINE EROSION*

*Cause and pathogenesis*

This syndrome is recognised in tropical marine fish, particularly angelfish and tangs.

Unknown cause: possibly related to poor nutrition, stress or viral infection.

*Clinical signs*

Chronic ulceration of the sensory pits on the head and along lateral line.

*Diagnosis and therapy*

The presence of typical clinical signs in marine fish. Histological examination.

No known treatment has been described although some benefit may be seen from improving the water quality, management and nutrition (by adding vitamins to feed).

## *POOR WATER QUALITY*

*Cause and pathogenesis*

Various factors: poor filtration system, immature biological filter, extraneous toxins. Poor water quality causes physiological stress and predisposes to secondary infections.

*Clinical signs*

General lethargy and inappetance. Dull coloration due to excess mucus production, darkening of colours in some species.

*Diagnosis and therapy*

Water test kits (e.g. ammonia, nitrite, nitrate, pH) are useful to establish the fault. Water quality can be improved by regular partial water changes, and the addition of salt for freshwater fish. Improve filtration equipment and removal of any potentially toxic objects or materials from water.

## *TRAUMA, BRUISING*

### *CAUSE AND PATHOGENESIS*

Poor handling and netting, attack from predatory fish, birds and mammals. Self-injury from jumping out of tank or pond.

*Clinical signs*

Various lesions including lacerations, bruising and erosions.

*Diagnosis and therapy*

Clinical signs and known history of trauma. It is essential to remove the underlying cause. Antibiotics may be given by injection or in food to avoid secondary bacterial infections. Convalescence in good water quality, with the addition of salt for freshwater fish.

## *GAS BUBBLE DISEASE*

*Cause and pathogenesis*

This is caused by supersaturation of the water with dissolved gases, particularly nitrogen. Gases are forced into solution under pressure. It can arise following heavy algal growth, unpressurised air transportation or as a result of using borehole water or through faulty water pumps.

*Clinical signs*

Small bubbles visible under skin, particularly on fins, gills and in the anterior chamber of eyes.

*Diagnosis and therapy*

Gross appearance of clinical lesions. No treatment is necessary as the condition will resolve following removal of the underlying cause.

### *SUNBURN*

*Cause and pathogenesis*

UV irradiation: on very sunny days in shallow outdoor ponds with exceptionally clear water.

*Clinical signs*

Areas of erythema and petechiation on white and non-pigmented dorsal surfaces.

*Diagnosis and therapy*

Clinical signs with predisposing environmental factors. Therapy should be aimed at prevention by providing shading and supportive treatment of affected fish.

## NEOPLASIA

### *PAPILLOMA*

*Cause and pathogenesis*

This is a common skin neoplasm. It rarely causes clinical problems unless obstructing the mouth or operculae. Some papillomas are associated with herpes virus (e.g. cyprinid herpes virus 1). Some papillomas may transform into deeply invasive squamous cell carcinomas.

*Clinical signs*

Found on most external sites including the fins but commonly on the head and around the mouth. Vary in size and appearance (may be smooth or verrucose, sessile or pedunculated, localised or extensive, single or multiple).

*Diagnosis and therapy*

Typical clinical signs and biopsy of lesions.

No effective treatment has been described; surgical reduction of pedunculated lesions may be beneficial. Some lesions may regress spontaneously after several months but may recur later.

## PIGMENT CELL TUMOUR

*Cause and pathogenesis*

All pigment cells (melanophores, xanthophores, erythrophores, leucophores, iridophores) may be involved and exhibit the colour of the cell of origin. Some pigment cell tumours can be multicoloured and are called chromatoblastomas. Melanomas are the most common pigment cell tumour.

There is genetic predisposition in some hybrid species, such as swordtail × platy fish.

*Clinical signs*

Found on most external sites. Vary in size and appearance but often discrete, smooth and sessile. Often solitary lesions but erythrophoromas on goldfish may be multiple.

*Diagnosis and therapy*

Biopsy is the diagnostic test of choice. Pigment analysis is required for definitive diagnosis but gross colour may suggest cell of origin.

There is no effective treatment but where practical, surgical excision may be of some benefit.

## FIBROMA

*Cause and pathogenesis*

This is a common neoplasm and often resembles some papillomas and pigment cell tumours.

*Clinical signs*

Can be found on most external sites, including the cornea. Vary in size and appearance but often discrete, smooth and sessile (Figure 13.7).

*Diagnosis and therapy*

Biopsy is the diagnostic test of choice. There is no effective treatment, but surgical excision may be of some benefit.

## FORMULARY

With the exception of the anaesthetic agent, MS222, no medicines are licensed for use in ornamental fish. Many different chemicals are used to treat skin diseases

**Fig. 13.7** Fibroma in Fancy goldfish.

**Table 13.2** Chemical treatment of skin disease in fish.

| Drug | Dose | Indications |
|---|---|---|
| Acriflavine (neutral) | Add to water, 5–10 mg/litre as permanent bath.<br>Add to water, 500 mg/litre daily for 30 minutes. | External bacterial, fungal and parasitic infections. |
| Benzalkonium chloride | Add to water, 5 mg/litre for 60 minutes. | External bacterial infections and general disinfection. |
| Chloramine T | Add to water, dose according to the following table for 60 minutes (repeat after 48 hours if necessary).<br>Dose (mg/litre)<br>pH — soft water — hard water<br>6.0 — 2.5 — 7.0<br>6.5 — 5.0 — 10.0<br>7.0 — 10.0 — 15.0<br>7.5 — 18.0 — 18.0<br>8.0 — 20.0 — 20.0 | External bacterial and parasitic infections. |
| Enrofloxacin | By addition to food, 10 mg/kg bodyweight for ten days.<br>Add to water, 2.5–5 mg/litre for five hours daily for five days.<br>By injection, 5–10 mg/kg i.m. every second day for 15 days. | Bacterial infections. |
| Formaldehyde (35–40%) | Add to water, 0.125–0.25 ml/litre for 60 minutes.<br>Add to water, 0.015–0.025 ml/litre as permanent bath. | Ectoparasitic infections. |
| Fresh water | 2–10 minute dip daily for five days (add sodium bicarbonate to buffer water pH). | Protozoal infections in marine fish. |
| Leteux-Meyer mixture (3.3 g malachite green/ litre formalin 40%) | 0.015 ml/litre as permanent bath and repeat three times every 48 hours. | External fungal and parasitic infections. |

**Table 13.2** *Continued*

| Drug | Dose | Indications |
|---|---|---|
| Lufenuron (Program®) | Add to water, 0.088 mg/litre as permanent bath. | *Argulus, Lernaea* parasites. |
| Malachite green | Add to water, 0.1 mg/litre as permanent bath.<br>Apply topically to lesions as 1% solution. | External fungal and protozoal infections. |
| Methylene blue | Add to water, 2 mg/litre as permanent bath and repeat three times every 48 hours. | External bacterial infections. |
| Metronidazole | Add to water, 25 mg/litre as permanent bath and repeat three times every 48 hours.<br>By addition to food, 10 mg/g food daily for five days. | External protozoal infections and freshwater 'hole in the head' disease. |
| Orabase®, Orahesive® (Convatec) | Apply topically to wounds (contains gelatin, pectin, methyl cellulose). | Waterproof products for ulcers. |
| Oxolinic acid | By addition to food, 10 mg/kg bodyweight (freshwater fish).<br>By addition to food, 30 mg/kg bodyweight (marine fish). | Bacterial infections. |
| Oxytetracycline | Add to water, 13–120 mg/litre for 60 minutes.<br>By addition to food, 75 mg/kg bodyweight. | Bacterial infections. |
| Potassium permanganate | Add to water, 2 mg/litre as permanent bath and repeat after 24 hours if high organic load in water. | External bacterial and parasitic infections. |
| Povidone-iodine | Apply topically to wounds. | Topical wound disinfectant. |
| Praziquantel | Add to water, 2–10 mg/litre for four-hour bath and repeat three times every five days. | Ectoparasite infection. |
| Sodium chloride | By addition to fresh water, 20–30 g/litre as 15–30-minute bath.<br>By addition to fresh water, 2 g/litre as a permanent bath. | Adult leeches only.<br>Aid wound healing and reduce osmotic stress in freshwater fish. |
| Sulphadiazine + trimethoprim | By addition to feed, 30 mg/kg bodyweight.<br>By injection, 30 mg/kg i.m. on alternate days. | Bacterial disease. |
| Trichlorphon (organophosphate) | Add to water, 0.5 mg/litre as permanent bath. | *Argulus*, *Lernaea*, leeches. |

but the volume of some diluted chemicals may be small and difficult to titrate accurately. There are many commercial preparations which contain one or more ingredients which are available to the hobbyist without a prescription. These products may be used to treat various external conditions as suggested by the manufacturers, but care should be taken over claims which may be difficult to substantiate.

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# SECTION FOUR

# Dermatology of Mammals

Chapter 14

# Structure and Function of Mammal Skin

The basic structure and function of the skin of exotic small mammals is similar to that of other mammals such as the cat and dog, and also man. Indeed, small rodents and rabbits are frequently used to study models of human skin disease. However, there are some notable differences of which the veterinary practitioner should be aware. In all animals, the skin is the largest organ and provides the anatomical and physical barrier between the animal and the environment.

## STRUCTURE OF MAMMALIAN SKIN

Mammalian skin structure is complex and has been widely described in many texts, including those dealing with small animals listed as further reading at the end of this chapter. The skin completely encloses the body and blends with the mucous membranes at the body orifices. Skin consists of two layers, the outer epidermis and the underlying dermis which rests on a stratum of loose connective tissue called the subcutis (Figure 14.1).

### *EPIDERMIS*

The epidermis is a superficial epithelium consisting of layers of cells which migrate towards the surface from where they are sloughed off in flakes or smaller particles. Epidermal cells are of four types: keratinocytes (about 85%), melanocytes (about 5%), Langerhans cells (3–8%) and Merkels' cells (about 2%).

- *Keratinocytes* are the major epithelial cells and are responsible for keratin production.
- *Melanocytes* are also found in the hair follicle and produce melanin pigments, which are largely responsible for skin and hair colouration. Melanin is also important for ionising radiation protection (mainly ultraviolet light) and is a scavenger of toxic free radicals. In albino animals, although melanocytes are present, melanin is totally absent. Melanins embrace a wide range of pigments, all arising from a common metabolic pathway.
- *Langerhans cells* are dendritic cells involved in the processing of antigens.
- *Merkels' cells* are specialised slow-adapting mechanoreceptors, and may also influence skin blood flow and sweat production.

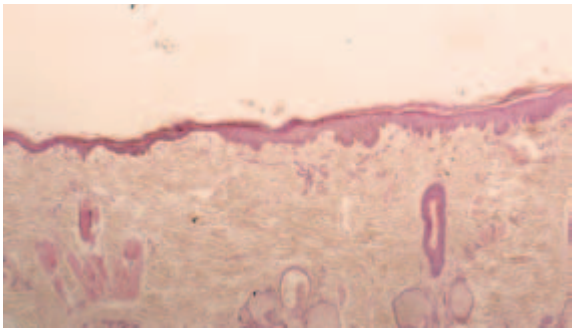

**Fig. 14.1** Histopathological section through mammalian skin.

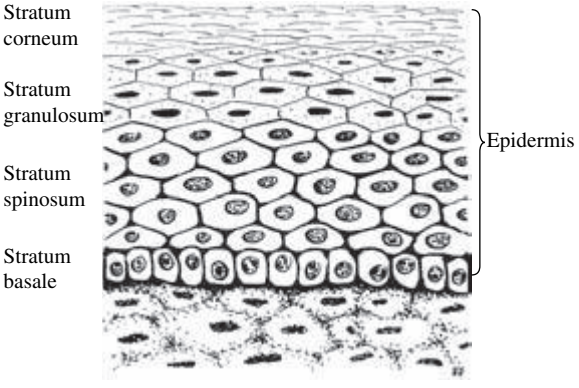


**Fig. 14.2** Schematic diagram showing different layers of mammalian skin.

The layers of the skin (Figure 14.2) are named the:

- basal layer (deepest) or *stratum basale*.
- spinous layer or *stratum spinosum*.
- granular layer or *stratum granulosum*.
- horny layer or *stratum corneum*.

Superficial cell loss is replaced by cell division in the *stratum basale* followed by migration of daughter cells toward the surface. As they migrate, cells undergo a series of changes which gradually bring about their death. The *stratum basale* follows the irregularities of the underlying dermis and thus has a much greater surface area than that of the *stratum corneum*. In the *stratum spinosum*, cells shrink and draw apart and the process of keratinisation begins. In the *stratum granulosum* the cells contain keratohyalin granules. In some areas this is followed by a clear layer or *stratum lucidum* in which the flattened cells, having lost their nuclei and distinct outline, have a homogeneous appearance. The outermost stratum corneum consists of squames, densely packed with the fibrous protein keratin, which are constantly shed.

The epidermal layers, most notably the *stratum spinosum*, are thickest where skin is exposed to hard usage, such as the footpads.

There are no blood or lymphatic vessels within the epidermis, and it is nourished by diffusion from the subjacent dermis.

## DERMIS

The dermis, or *corium*, is connective tissue largely composed of collagen bundles. Elastic fibres are also present and together these give the skin its tensile strength and pliability. The dermis is divided into the *stratum papillare* and the *stratum reticularem* which contains coarser collagen fibres. Unlike the epidermis, the dermis is highly vascular and innervated. It is invaded by hair follicles, sweat glands, sebaceous glands and other glands growing from the epidermis. It also contains the *arrector pili* muscles associated with hair follicles.

## SUBCUTIS

The subcutis (or hypodermis) consists of loose connective tissue and fat. It is the deepest and usually the thickest layer of skin. The amount of fat depends on nutritional condition and is predominantly white fat. In rodents, brown fat is located between the scapulae, in the ventral neck and in the axillary and inguinal regions. In areas where movement is undesirable it is thin or absent (e.g. lips, eyelids, external ear, nipple). It is ample in the smaller rodents, rabbits and ferrets, whose generous scruffs are used for handling, but is less prominent in guinea-pigs and chinchillas, and these cannot be restrained by 'scruffing'.

Skin thickness varies between species and location. Ferrets, for example have very thick skin, especially over the neck and shoulders.

## HAIR AND THE HAIR CYCLE

Hair is unique to mammals. It provides thermal insulation, and is important in sensory perception and protection of the skin. In most species of small mammal a thick hair coat is spread over the body, except around the mouth and nose and the plantar surface of the feet. In most rodents, the pinnae are hairless (Figure 14.3), with the exception of the gerbil. Rabbits and ferrets have haired pinnae. In

**Fig. 14.3** Hairless pinnae in a Chinchilla.

**Fig. 14.4** Rat's tail showing arrangement of scales.

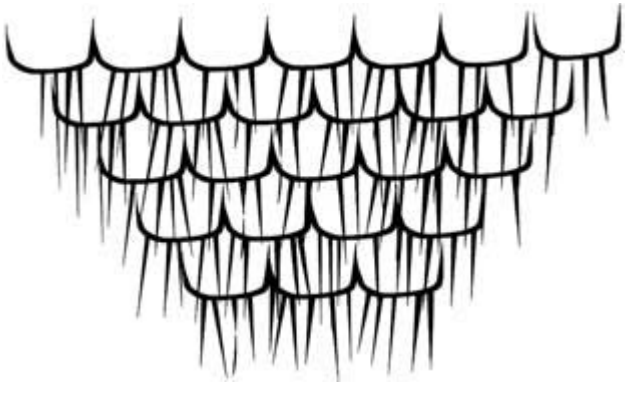

**Fig. 14.5** Diagram to show the arrangement of the scales on a rat's tail.

the rat and mouse the tail is very sparsely haired, and the epithelium differs between the haired and non-haired area. There is normal epithelium (orthokeratosis with a *stratum granulosum*) at the follicular opening or *ostia*, but parakertosis and no *stratum granulosum* in the interfollicular areas (Scott *et al*. 2001). This gives the tail of these species its characteristic scaly appearance – it is covered with square scales, underneath which grow two to six straight hairs, extending over the next row of scales (Figures 14.4, 14.5).

Hairs can be divided into three main types:

- Primary or guard (outercoat) hairs.
- Secondary or down (undercoat) hairs.
- Tactile hairs (vibrissae).

All hairs grow from follicles positioned obliquely in the skin at an angle of 30–60° in relation to the epidermis. The hair coat generally slopes caudally and ventrally, giving the minimal resistance to forward movement of the animal. The number and type of hairs per follicle varies between species and breed and depending on the age of the animal. For example, chinchillas produce as many as

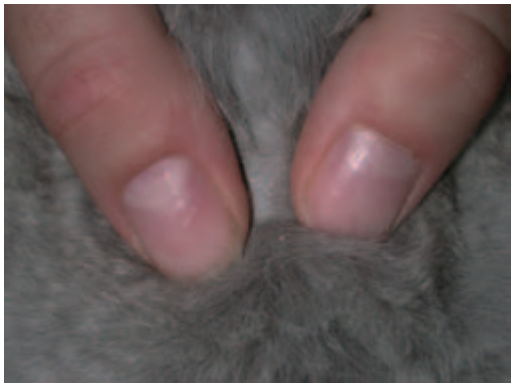

**Fig. 14.6** Thick, soft coat in a chinchilla.

60 hairs per follicle, which gives them their characteristic dense soft coat (Figure 14.6).

- *Primary hairs* lie closely against the skin and give the coat a smooth appearance, which can be disturbed by whorls, crests or partings. The regular arrangement of primary hairs helps to prevent water penetration to the skin.
- *Secondary hairs* are thin, wavy and in most species shorter and more numerous than the primary hairs that conceal them. They provide the soft undercoat in the guinea pig – Peruvian (long, primary hairs stemming from two rosettes on the rump) and Rex (no primary hairs). In most species congenital alopecia occurs (hairless breeds).

Hairs consist of a flexible column of closely consolidated and heavily keratinised dead epithelial cells. The hair shaft is divided into medulla, cortex and cuticle. The pigment that gives the hair its colour is contained within the cortex. Different hair types have different ratios of the three layers – primary hairs with a thick medulla are straight and relatively brittle, those with a thick cortex are stronger and more pliable, and secondary hairs have a very narrow medulla and more prominent cuticle than primary hairs.

*Primary hairs* in mammals are associated with sebaceous glands, sweat glands and an *arrector pili* muscle (Figure 14.7). Rodents and ferrets are notable in that they have no epitrichial (apocrine) sweat glands. Rabbits only have sweat glands on the lips and pant ineffectively. Thus all small mammals are susceptible to overheating.

*Secondary hairs* are usually only accompanied by sebaceous glands. Involuntary contraction of the *arrector pili* muscle causes piloerection and thickening of the hair pile to trap more air and hence improve thermal insulation.

*Tactile hairs* are substantially thicker than primary hairs and protrude beyond them. Most are found on the face, principally on the upper lip and muzzle, (Figure 14.8) but they are also found above the eyelid, near the ear and on the caudal side of the thoracic limbs. The follicles extend deep into the subcutis or even the superficial muscles, and are surrounded by a venous sinus within whose walls are nerve endings responsive to mechanical stimulation.

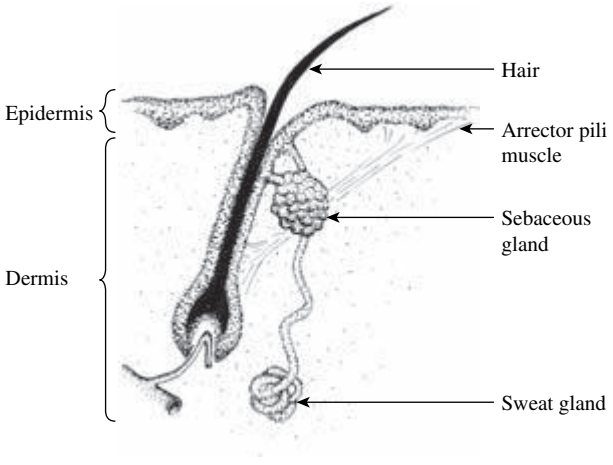


**Fig. 14.7** Structure of mammalian hair follicle.

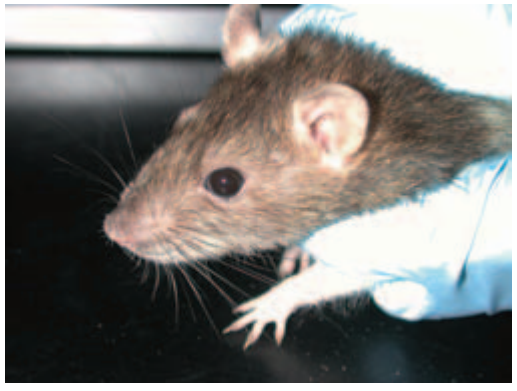

**Fig. 14.8** Tactile hairs on the face of a rat.

## *HAIR GROWTH*

Hair growth is cyclical (Figure 14.9). Each cycle consists of a

- *growing phase* (anagen), when the follicle is actively producing hair.
- *resting period* (telogen) when dead hair is retained in the follicle then lost.
- *transitional phase* (catagen) between these two stages.

The hair cycle is controlled by many factors, including photoperiod, environmental temperature, nutrition, general health status, stress, genetics and other poorly understood intrinsic factors.

In most rodents, with the exception of guinea pigs, and in rabbits, the hair cycle is synchronised so that adjacent hairs are in the same phase of the cycle (Komarek *et al*. 2000). The hair grows in orderly waves starting on the ventral surface between the forelimbs and spreading dorsally and caudally. In some rabbits, thickened patches of skin can be associated with the variation in hair growth cycles, associated with increased skin vascularity and size of follicles.


**Fig. 14.9** Mammalian hair-growth cycle.

Obvious moulting patterns are generally not noticed in the smaller rodents (mice, rats, gerbils, hamsters) and guinea pigs and chinchillas, but in rabbits, especially those kept outdoors, seasonal patterns are noticeable. Ferrets moult in the spring and autumn and their hair colour may change to a lighter shade in winter.

Hair colour and coat type, along with eye colour and body conformation, are the main criteria of both fur and fancy breeders in improving and creating new breeds and varieties of small mammals. The genetics underlying this is beyond the scope of this chapter.

## *FOOTPADS*

Footpads are areas of specialised thickened epidermis to protect against mechanical trauma with underlying fat deposits to provide shock absorbency. Rodents have smooth footpads. Rats, mice and hamsters have four front digits, five hind digits and hairless fleshy footpads. Atrichial or eccrine sweat glands are located only in the footpad skin in these rodents. The gerbil has five front digits, four hind digits and the footpads are small and furred. Guinea pigs and chinchillas have four front digits, three hind digits and hairless footpads. Rabbits have five digits on the front feet and four on the hind, and do not possess foot pads. Instead the digits and metatarsals are covered with coarse fur. Ferrets possess five toes on all feet and possess footpads similar to the dog.

## *CLAWS*

The claws consist of dense thickened cornified epidermis derived from specialised epithelial cells overlying a prominent vascular dermis covering the terminal phalanges. Claws are non-retractile in rodents, rabbits and ferrets.

## *SEBACEOUS SCENT GLANDS*

Sebaceous scent glands are a noticeable feature of many of the small mammal species (Scott *et al*. 2001b), and are important in scent marking, communication and territorial behaviour. Hamsters have large darkly pigmented glands on either flank, which are more prominent in males. The oily secretion is readily visible in

sexually aroused males and causes hair matting. Gerbils have a large elliptical yellowish hairless scent gland on the ventral abdomen. Guinea pigs possess a large gland over the rump above the tail and other glands around the anus. Rabbits have sebaceous scent glands on the chin (mental gland) which they rub on objects in their territory, anal glands and paired pocket-like inguinal glands. Ferrets have very active sebaceous glands throughout the skin, giving them their characteristic musky odour and greasy coat. They also have two prominent perianal scent glands. Normal ferrets can often have comedones on the skin of the tail.

### *MAMMARY GLANDS*

Mammary glands are modified enlarged sweat glands which produce milk. The number of mammary glands and teats varies between species:

- Mice: 5 pairs.
- Rats: 6 pairs.
- Gerbils: 4 pairs.
- Hamsters: 6–7 pairs.
- Guinea pigs: 1 pair (inguinal, both sexes).
- Chinchillas: 3 pairs (one inguinal, two lateral thoracic).
- Rabbits: 4 pairs.
- Ferrets: 4 pairs.

Hair cover is reduced or absent around the nipples. Female rabbits pluck hair from the ventral surface to expose the nipples and line the nest when breeding.

## FUNCTION OF MAMMALIAN SKIN

The skin is highly complex and has the greatest diversity of functions of any organ in the body. As well as covering the body and allowing motion and providing shape, it has the following major functions:

- Protection and a physical barrier.
  - Enables the maintenance of an internal environment by preventing loss of water, electrolytes and macromolecules.
  - Protects from environmental agents (chemical, physical and microbiological) that may cause injury.
- Temperature regulation.
- Sensory perception – touch, heat, cold, pressure, pain, itch.
- Storage – electrolytes, water, vitamins, fat, carbohydrates, proteins etc.
- Immunoregulation.
- Secretion – apocrine, eccrine and holocrine (sebaceous) glands.
- Vitamin D production – via solar radiation.
- Production of adnexa – hair, claws, spines.
- Excretion – the skin has a limited function as an excretory organ.
- Indicator – of general health, internal disease, physical and sexual identity.

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Chapter 15

# Examination of Mammalian Skin and Diagnostic Tests

The principles of diagnosis of skin disease in exotic small mammals are identical to those used in dogs, cats and other domestic species. However, due to their smaller size, potential difficulties and stress involved with restraint, as well as differences in anatomy, the practitioner should be aware of a few differences.

## RESTRAINT

Clinical examination and obtaining diagnostic samples can be difficult, especially in the smaller rodents. Well-handled ferrets, rabbits, guinea pigs, chinchillas and rats usually pose no problems in clinical examination. With the smaller rodents, knowledge of safe restraint techniques is necessary in order to keep the subject still enough to allow examination (See Table 15.1).

**Table 15.1** Handling / restraint techniques for mammals.

| Species | Technique |
|---|---|
| Mouse | Base of tail and pull backwards gently with mouse on a rough surface; scruff. |
| Rat | Around shoulders; base of tail; scruff. |
| Hamster | Cup in hands; scruff. |
| Gerbil | Around shoulders; scruff. Never hold by tail. |
| Guinea pig | Around shoulders and support bodyweight. |
| Chinchilla | Around shoulders and support bodyweight. |
| Rabbit | Scruff and support rear end. |
| Ferret | Around shoulders with thumb under chin. |
| Hedgehog | Use latex gloves. Uncurl by holding head down over flat surface and grasp back legs. |

## *SEDATION AND ANAESTHESIA*

Sedation or anaesthesia is often necessary for both clinical examination and the application of diagnostic tests in these species.

Table 15.2 gives suggested sedative and anaesthetic drug dosages for small mammals.

**Table 15.2** Sedative and anaesthetic regimes for small mammals.

| Species | Technique |
|---|---|
| Mouse, Rat, Hamster, Gerbil, | Chamber induction with halothane or isoflurane, maintenance on face mask. |
| Guinea pig | Chamber induction with halothane or isoflurane, maintenance on face mask.<br>Medetomidine 0.2–0.5 mg/kg s.c. (sedation)<br>Medetomidine 0.2–0.5 mg/kg + ketamine 20–40 mg/kg s.c. (anaesthesia). |
| Chinchilla | Chamber induction with halothane or isoflurane, maintenance on face mask.<br>Ketamine 10–15 mg/kg + midazolam 0.5 mg/kg + atropine 0.05 mg/kg s.c. (light anaesthesia). |
| Rabbit | Medetomidine 0.1–0.25 mg/kg s.c. (sedation).<br>Medetomidine 0.1–0.25 mg/kg + ketamine 5–15 mg/kg s.c. (+/− butorphanol 0.4 mg/kg) (anaesthesia). Intubate and maintain on oxygen, add isoflurane / halothane if necessary.<br>Acepromazine 0.1–0.5 mg/kg s.c.<br>Midazolam / diazepam 0.5–2 mg/kg s.c. |
| Ferret | Medetomidine 0.08–0.1 mg/kg s.c. (sedation).<br>Medetomidine 0.08 mg/kg + ketamine 5 mg/kg s.c. (anaesthesia).<br>Medetomidine 0.08 g/kg s.c. + propofol 1–3 mg/kg i.v., intubation and maintenance on isoflurane or halothane (anaesthesia).<br>Face mask or chamber induction with isoflurane. |
| Hedgehog | Chamber induction with halothane or isoflurane, maintenance on face mask. |

## APPROACH TO THE SKIN CASE

A systematic approach should always be employed when faced with cases of skin disease. The depth of approach can vary depending on the case; in some animals the diagnosis will be immediately obvious, but in general a detailed history, clinical examination, and the use of one or more diagnostic tests are required. One suggested method of approach is the ten-point plan suggested by Thoday (1984).

1 Owner's complaint.
2 Identify the patient – species, breed / strain, sex, age (Figure 15.1).
3 Preliminary brief examination – allows questioning to be directed to certain areas.
4 General medical history.
5 Specific history regarding the skin disorder.
6 Lifestyle – diet (Figure 15.2), environment (Figure 15.3).

7 Contagion – other animals, human contacts.
8 Clinical examination – general, skin.
9 Laboratory and diagnostic tests.
10 Correlate data for diagnosis.

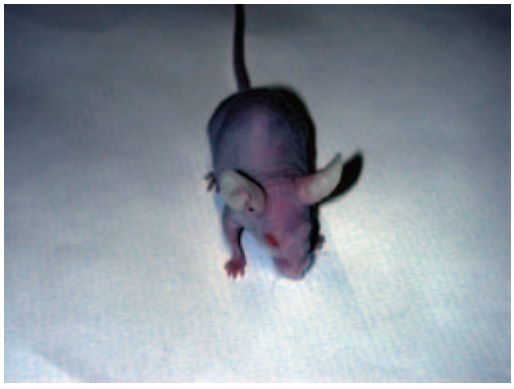

**Fig. 15.1** Congenital alopecia is seen in varieties of hairless mice and rats.

**Fig. 15.2** The type of food fed to a small mammal is an important part of its history.

**Fig. 15.3** Bedding type can be important.

**Fig. 15.4** Observation in the environment may be important.

**Fig. 15.5** Recording the weight is important in small mammals in order to calculate accurate drug dosages.

## EXAMINATION OF THE ANIMAL

**Physical inspection** should be undertaken in every case to assess the animal's general appearance, demeanor and body condition.

In may cases it is better to observe the animal in its environment before starting to handle it (Figure 15.4).

Temperature, pulse and respiratory rate should be checked.

When dealing with small mammals it is important to weigh the animal (Figure 15.5).

A more detailed examination of some organ systems can be undertaken where indicated.

**Dermatological examination** should be undertaken next. For examination of the skin (point 8, Thoday's list above), again, a methodical approach should be adopted. It can be easy to focus in on the lesion the owner has noticed and miss abnormalities elsewhere. The skin should be examined from head to tail and dorsum to ventrum. Palpation as well as visual examination should be employed. The pinnae and external ear canals should be examined with the naked eye and

an otoscope for the larger species. Skin thickness, quality and texture of coat, and degree of pruritus elicited by digital stimulation should be noted if possible, and compared to knowledge of what is normal for that species.

The lesions should then be examined in detail and observations recorded, preferably on a line diagram. Recording in this way provides a much more accurate description of lesion distribution than a written record alone and is extremely useful in monitoring the course of disease and response to treatment.

Lesions should be described accurately with regard to:

- Distribution.
- Arrangement.
- Configuration.
- Depth.
- Consistency.
- Quality.
- Colour.

The morphology of the individual lesions should also be described. Lesions may be either:

1 *Primary*: i.e, macule, patch, wheal, vesicle, papule, pustule, nodule, tumour.
2 *Secondary*: i.e, scale, crust, hyperkeratosis, excoriation, ulcer, scar, ichenification, hyperpigmentation, fissure, comedo.

## *DIAGNOSTIC TECHNIQUES*

### *BLOOD SAMPLING*

Blood sampling may form part of the diagnostic work-up for a dermatological case, providing information on the general health of the animal and various hormone levels (e.g. ferrets). Sedation or anaesthesia is usually required (See Table 15.3).

**Table 15.3** Blood sampling sites in small mammals.

| Species | Technique |
|---|---|
| Mouse | Lateral tail vein, saphenous vein. |
| Rat | Jugular vein, lateral tail vein, saphenous vein. |
| Hamster | Cephalic vein, saphenous vein, cranial vena cava. |
| Gerbil | Lateral tail vein, saphenous vein. |
| Guinea pig | Jugular vein, cranial vena cava. |
| Chinchilla | Jugular vein, cephalic vein, femoral vein (medial). |
| Rabbit | Jugular vein, cephalic vein, saphenous vein, marginal ear vein. |
| Ferret | Jugular vein, cranial vena cava, cephalic vein, saphenous vein. |
| Hedgehog | Jugular vein, cranial vena cava, cephalic vein, saphenous vein, femoral vein (medial). |

## BACTERIAL INFECTIONS

Bacterial sampling can be difficult and yield misleading results if not done properly. Open infected lesions are usually contaminated with the resident microbial population of the skin, and should not be used. Intact pustules should be selected for sampling. The hair should be clipped and the site swabbed gently with 70% alcohol, taking care not to rupture the pustule. The pustule is then opened with a sterile 25 or 27 gauge needle and the contents allowed to soak on to a carefully placed sterile swab. Great care must be taken not to touch the swab onto the surrounding skin. The swab should be placed in transit medium to prevent dessication of bacteria, and submitted to the laboratory for culture and identification.

Needle aspirates from the centre of an abscess are frequently sterile. The best material to submit for culture is a sample of the abscess wall.

## FUNGAL INFECTIONS

### *Ultraviolet (Wood's) light*

This technique is used to detect *Microsporum* species. Approximately 60% of *M. canis* fluoresce when exposed to ultraviolet light, and other *Microsporum* species – *M. audouini*, *M. distortum* and *M. incurvata* may also show a response. The use of ultraviolet light is limited in rodents and rabbits as most cases of dermatophytosis are due to *Trichophyton* species. In fine-haired animals the diameter of some hair shafts may be below the resolution of the human eye and magnification may need to be used in order to avoid missing a positive result. It is important to allow the light to warm up prior to use.

Examination should take place in a darkened room. A positive result is indicated by apple-green fluorescence. It should be distinguished from the purple / blue colour of dust. The infected hairs themselves should fluoresce – fluorescence is not exhibited by scales, crusts or laboratory fungal cultures. Affected hairs should be epilated to confirm this. In positive cases the portion within the follicle will also appear green.

## DIRECT MICROSCOPY

Careful sample collection is required for microscopic evaluation. Normal hairs are unlikely to be infected. If positive fluorescence is observed with the Wood's light, affected hairs should be chosen. The area can be swabbed with 70% alcohol first to reduce bacterial contamination. Damaged hairs should then be plucked with forceps, and scales or crusts removed with a blunted scalpel blade. The edge of the lesion is the preferred site as this is where infection is most active. The material should be cleared with 5% KOH, as described for ectoparasites (below), and examined microscopically for the presence of fungal spores and hyphae. Spores are small spherical refractile bodies in chains or as a mosaic sheath around the

hair. Hyphae are seen as filaments, sometimes fragmenting into spores. Samples may also be stained with lactophenol cotton-blue or blue-black ink, but this is often unnecessary.

### *FUNGAL CULTURE*

Definitive identification of pathogenic fungi is obtained only by fungal culture. Sabouraud's dextrose agar is the most commonly used culture medium. It is slightly acidic and has antibiotics to inhibit bacterial growth and cyclohexamine to inhibit saprophytic fungal growth. A small quantity of sample material is pressed firmly on to the surface of the plate and the lid replaced loosely. Material may be collected as described for direct microscopy, or collected by the Mackenzie brush technique, where hairs and scale are collected by brushing the coat with a sterile toothbrush. The collected material is then shaken on to the culture medium, or transferred by pushing the brush bristles gently into the surface of the medium. Fungae require aerobic conditions to grow. Incubation is at room temperature, and dessication is prevented by placing plates within an enclosed area containing a water-filled pot. The common dermatophytes produce growth within one to two weeks, but samples should be maintained for one month before being deemed negative.

## *ECTOPARASITES*

### *DIRECT OBSERVATION*

Larger ectoparasites such as ticks, fleas, and lice may easily be seen with the naked eye. The larger mites such as *Mycoptes*, *Trombicula* and *Cheyletiella*, may also be seen with close direct examination, but are more easily seen with magnification using a hand lens or otoscope. Definitive identification should then be made by collection and low power microscopy.

### *COAT BRUSHINGS OR COMBINGS*

Coat brushings can be useful in the identification of flea and *Cheyletiella* infestations. Coat brushing after the application of an insecticide such as fipronil may reveal fleas, as they can be difficult to locate in the hair coat when in low numbers. Flea faeces are comma shaped and brown / black in colour. Brushing them on to a light-coloured damp surface such as moist cotton wool causes a reddish-brown halo to form due to their blood content, and allows them to be distinguished from other debris. Cheyletiella may be seen by brushing the coat on to a dark surface. The mites are tiny white mobile specks – the co-called 'walking dandruff'. Definitive identification should again be made by collection and low power microscopy.

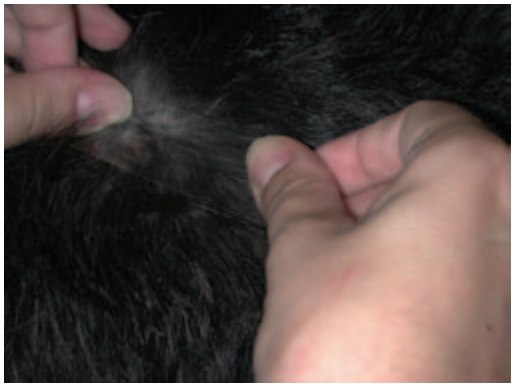

**Fig. 15.6** Tape strippings of the coat may identify fur mites.

## *ADHESIVE TAPE COLLECTION*

This is a useful technique for the collection of surface-living mites such as *Myobia* and *Cheyletiella*. The hair is parted and a piece of clear adhesive tape attached to and then removed (Figure 15.6) from the skin several times to collect material. It is then attached to a microscope slide and viewed under the microscope under low power.

## *SKIN SCRAPING*

This is an extremely useful diagnostic technique, but tends to be under-used in exotic species. The site of the scraping should be one that is a suspected predilection site for the ectoparasite, shows changes suspicious of ectoparasite infestation and has not been traumatised by the animal.

The scraping site is prepared in different ways, depending on whether the suspected mite is surface-living or burrowing / follicular.

- Superficial skin scrapings for surface-living mites: the hair should be superficially clipped wherever possible to minimise contamination of the sample with hair. This is relatively simple in the larger species, but rabbit and chinchilla fur is very fine and can be difficult to clip. Scraping for surface mites can be made without clipping (Figure 15.7). It may be difficult in the smaller rodents due to their size, and anasethesia or sedation is required.
- Deep skin scrapings for burrowing or follicular mites: the hair should be closely clipped wherever possible.

There are then two collection methods. The method chosen largely depends on personal preference:

1 **Potassium hydroxide (KOH).** This has the advantage that it dissolvess the keratin in the sample allowing easier visualisation of the mites, but the disadvantgae that the mites are killed:

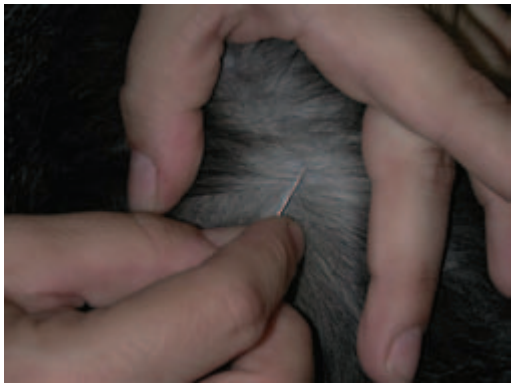

**Fig. 15.7** Superficial skin scrapes may identify surface mites.

- Clip the hair and remove clippings.
- Moisten the skin with 5% KOH on cotton wool.
- Scrape the skin in one direction only with a blunted scalpel blade. The back of the blade should be held at an angle of approximately 60° towards the operator. For surface-living mites a superficial scraping is necessary. For burrowing or follicular mites a deep scraping producing capillary ooze is required.
- Wipe the scraping site with damp cotton wool to remove the KOH.
- Transfer the collected material on to a microscope slide.
- Add 20% KOH to the material with a dropper or pipette.
- Mash with a glass rod (not the scalpel blade as this will damage the mites).
- Warm in a low Bunsen flame (do not boil), or leave at room temperature for one hour.
- Cover with a coverslip.
- Examine microscopically under low power initially, in an methodical manner, with high power to examine mite morphology in greater detail.

2 **Liquid paraffin.** This has the advantage that the mites remain alive and their movements attract attention of the observer, but the disadvantage that the keratin is not cleared so mites are more difficult to see. KOH cannot then be used if clearing of the sample is necessary.
- Clip the hair and remove clippings.
- Moisten the skin and / or blade with a drop of liquid paraffin.
- Scrape the skin, transfer and mash as described above, using more liquid paraffin if necessary.
- Do not heat.
- Cover and examine as described above.

## *HAIR PLUCKING / TRICHOGRAPHY*

When animals are difficult to handle or if sedation or anaesthesia is not possible, hairs from the affected area may be plucked and placed in either 20% KOH or liquid paraffin and examined directly.

Assessment of hair tips, shafts and bulbs may give useful information.

- Examination of the hair tip for traumatisation can give a clue as to whether hair loss is traumatic and probably self inflicted.
- Examination of the hair shaft may identify dermatophytes spores, pigment abnormalities or the presence of follicular casts as seen in sebaceous adenitis in rabbits.
- Examination of the bulbs can give an indication as to whether anagen (growing) or telogen (dead) hairs are present. It may also identify demodex mites and fungal spores.

**Impression smears** can be made from exudative lesions or else after lesions have been scraped so as to try and identify a cellular infiltrate. This may be useful in very small mammals where a biopsy is deemed unsafe or expensive. It may be used to identify a lymphocytic infiltration in plaques seen with lymphoma e.g. in the hamster.

**Fine-needle aspirates** may be useful to assess nodular lesions and to try to differentiate neoplastic and pyogranulomatous disease.

## *BIOPSY*

Biopsy is a very useful technique in the diagnosis of many categories of skin disease, but particularly for neoplasia, mite infestations, fungal infections, immune-mediated conditions and some endocrine diseases. Excisional biopsy is often the most practical technique in small mammals, where the entire lesion is excised under general anaesthesia and submitted for examination.

For other biopsies, a representative, non-excoriated lesion is selected. The animal is anaesthetised or sedated. Hair should be clipped and the skin swabbed with alcohol. If sedated, local anaesthetic is injected subcutaneously directly beneath or around the lesion. A small, full-thickness elliptical incision is made vertical to the skin surface. The biopsy should contain the transition from diseased to apparently normal skin. One corner is grasped with forceps and dissected free of subcutaneous fat. The wound is closed with sutures or tissue glue. The sample is blotted with a swab, placed subcutis down on to a piece of card and placed in 10% formalin. Alternatively, commercial punch biopsy instruments may be used.

## *TRIAL THERAPY*

This may be indicated in some cases where every effort has been made to identify a particular clinical trigger but the investigative tests have not been diagnostic.

- Anti-parasitic therapy may be used in cases of suspected ectoparasite problems.

- Antibiotics may be used in pustular disease to assess if a problem is infectious or sterile.
- Drug withdrawal: it is important to rule this out as a contributory factor.

## REFERENCE

1. Thoday, K.L. (1984) 'An approach to the skin case'. *The Henston Veterinary Vade Mecum (Small Animals).* Henston Ltd., pp. 243–246.

Chapter 16

# Skin Diseases and Treatment of Chinchillas

## BACTERIAL DISEASE

### *ABSCESSES*

*Cause and pathogenesis*

Abscesses are common following bite wounds from cage mates when chinchillas are group housed. Females are more aggressive than males. *Staphylococcus* and *Streptococcus* are common isolates (Ellis & Mori 2001).

*Clinical signs*

Soft fluctuant areas of swelling underlying area of trauma to the skin.

*Diagnosis and therapy*

Inspissation of abscess contents typically occurs and so complete surgical excision is the recommended treatment (Jenkins 1992). Ruptured abscesses should be aggressively flushed and the chinchillas should be given systemic antibiotics.

### *OTHER BACTERIAL CONDITIONS*

A moist dermatitis with secondary staphylococcal infection can also be caused by excessive salivation due to dental disease (Ellis & Mori 2001). Secondary staphylococcal infection may also be seen with dermatophytosis (Rees 1963).

## FUNGAL DISEASE

### *DERMATOPHYTOSIS*

*Cause and pathogenesis*

Dermatophytosis in chinchillas can occur due to infection with *Trichophyton mentagrophytes* (Hagen & Gorham 1972; Hoefer 1994; Scott *et al*. 2001 and Shaeffer & Donnelly 1997). *Microsporum gypseum* and *M. canis* are more rarely isolated.

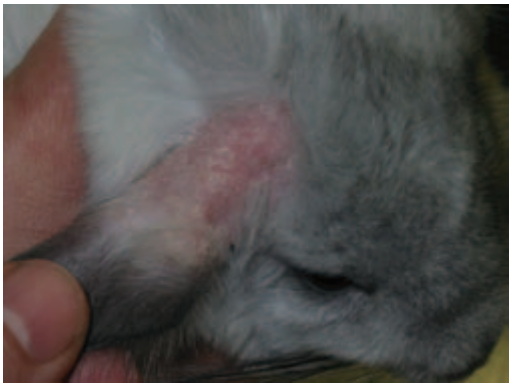

**Fig. 16.1** *Trichophyton mentagrophytes* infection on the ears of a chinchilla. (Picture courtesy J. Fontaine.)

**Fig. 16.2** *Trichophyton mentagrophytes* infection on the nose of the chinchilla. (Picture courtesy Z. Alha i dari.)

Transmission can be via fomites, hay, bedding or direct exposure to a symptomatic or carrier animal. Dust baths may act as a means of spread to several individuals.

*Clinical signs*

Lesions are mainly seen around the eyes, nose, mouth, legs and feet and consist of well circumscribed areas of alopecia, scaling, broken hairs, erythema and crusting (Figures 16.1, 16.2).

*Diagnosis and therapy*

Diagnosis in by clinical signs, microscopic examination of hairs and fungal culture. As the majority of infections are due to *T. mentagrophytes*, isolates are unlikely to fluoresce. Therapy should be directed at both the environment and the animal.

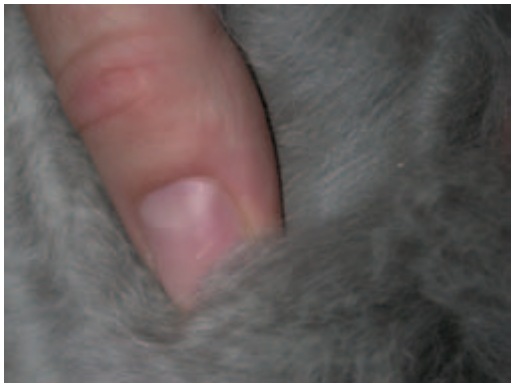

**Fig. 16.3** The dense fur of a chinchilla makes ectoparasitic infections unlikely.

Placing the dust bath in an oven for 20 minutes at 15°C has been suggested as a means of killing fungal spores (Burgmann 1997). The cage may be cleaned with enilconazole or bleach diluted 50:50 with water. The cage must be well rinsed and allowed to dry before animals are reintroduced. Treatment is with oral griseofulvin or topical anti-fungal preparations such as enilconazole.

## ECTOPARASITES

Chinchillas have extremely dense fur (60–90 hairs per follicle) and thus ectoparasites are rare (Figure 16.3). *Cheyletiella* spp. have been reported anecdotally and can be treated with ivermectin. Fleas may also be found occasionally (1) and may cause pruritus. Topical flea products approved for cats and rabbits are suitable, e.g. imidacloprid.

## NUTRITIONAL DISEASE

### *FATTY ACID DEFICIENCY*

*Cause and pathogenesis*

Cutaneous signs can be seen if animals are fed unbalanced diets which are deficient in linoleic and arachidonic acid. This is most commonly caused by poorly preserved food (incorrect storage, temperature) or home-made diets. Fatty acid problems can also be seen where inadequate anti-oxidants are present in the diet so that fat becomes rancid.

*Clinical signs*

Deficient diets are thought to cause flaking, reduced hair growth and fur loss, and sometimes cutaneous ulcers (Hoefer 1994). Signs occur over several weeks to

months. In severe cases the chinchilla becomes debilitated and may die. This condition can sometimes confusingly be referred to as 'fur slip' or 'fungus' by breeders (Strake 1996).

*Diagnosis and therapy*

Diagnosis is based on clinical signs, diagnostic rule out of other differentials such as dermatophytosis and response to dietary change. Treatment is by dietary supplementation when a rapid improvement is usually seen. In cases where the skin is pruritic, topical ointments containing linoleic or undecylenic acid are soothing.

## *PANTOTHENIC ACID DEFICIENCY*

*Cause and pathogenesis*

Pantothenic acid is essential for normal skin formation. Single B vitamin deficiencies are rare.

*Clinical signs*

Pantothenic acid deficiency can cause similar lesions to fatty acid deficiency (Hoefer 1994). Patchy alopecia, and occasionally thickened scaly skin, is seen and the affected chinchilla is anorectic, hyperactive and thin.

*Diagnosis and therapy*

Diagnosis is by history, especially of an unbalanced diet, clinical signs and response to therapy. Treatment is calcium pantothenate for 2–3 days by intramuscular injection followed by oral propothiouracil (Strake 1996).

## *ZINC DEFICIENCY*

*Cause and pathogenesis*

Zinc is an important co-factor and modulator of many critical functions. Zinc Deficiency is rare in pet chinchillas fed a commercial diet and good quality hay.

*Clinical signs*

Zinc deficiency can cause scaling and alopecia. It can be difficult clinically to distinguish between fatty acid, pantothenic acid and zinc deficiency.

*Diagnosis and treatment*

A diagnosis may be made by history, clinical signs and response to therapy. Therapy is undertaken by ensuring the diet is balanced.

## *'YELLOW EARS' / 'YELLOW FAT'*

*Cause and pathogenesis*

This is a condition seen in chinchillas on a diet deficient in choline, methionine or Vitamin E (Ellis & Mori 2001). Impaired metabolism of plant pigments leads to a concentration of yellow-orange pigment in the skin and fatty tissues.

*Clinical signs*

In chronic cases yellowish discolouration of the skin on the ventral abdomen and perineum occurs, and in severe cases the entire skin is affected. Painful swellings may also develop on the ventral abdomen.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs and history. Treatment is by re-establishing a balanced diet.

## *COTTON FUR SYNDROME*

*Cause and pathogenesis*

Cotton fur syndrome occurs when chinchillas are fed a high protein diet (crude protein >28%).

*Clinical signs*

In this condition the hair develops a wavy and weak texture, giving the appearance of cotton (Ellis & Mori 2001).

*Diagnosis and therapy*

Diagnosis is made on dietary analysis for protein levels and clinical signs. Treatment is by reduction of protein levels. Appropriate dietary levels are approximately 15%.

# ENVIRONMENTAL AND BEHAVIOURAL CONDITIONS

## *FUR CHEWING*

*Cause and pathogenesis*

Fur chewing and barbering is occasionally seen in chinchillas and may be related to overcrowding or some other stressor (Rees 1963). Many theories exist about the underlying cause, including fungal infection, hyperthyroidism, hyperadrenocorticism and dietary deficiencies. However, there is little evidence to support these theories and it is more commonly believed to be a behavioural problem, with a high incidence in commercial fur chinchilla herds. Barbering behaviour can pass from mother to young, suggesting a heritable component, and some breeders remove fur chewers from the breeding population.

*Clinical signs*

Hair is chewed off producing areas of apparent alopecia – short stubbly hair. Trichography (microscope examination of plucked hairs) has shown that the hair bulbs and shafts are normal but the tips are fractured, suggesting hair loss is self induced.

*Diagnosis and therapy*

Husbandry, nutrition and general health should be evaluated in clinical cases and any possible predisposing factors addressed. Provision of good quality hay *ad lib*., high quality chinchilla pellets as well as minimising stressors can all help (Burgmann 1997). Full hair regrowth will not occur in the presence of the dense undercoat and this should be removed if possible by plucking (Ellis & Mori 2001). There are no reports of treatment with psychotropic drugs such as fluoxetine, but a suggested dose is 5–10 mg/kg by mouth.

## *MATTED FUR*

*Cause and pathogenesis*

Chinchillas have an extremely dense hair coat (1000 follicles/$cm^2$) which is difficult to groom. Lack of a dust bath results in matted fur, as does high relative humidity (>80%). A permanent dust bath can lead to excess grooming and ocular problems (Shaeffer & Donnelly 1997).

*Clinical signs*

The hair is obviously thickly matted.

*Diagnosis and therapy*

A diagnosis is made on the basis of clinical signs and history. The provision of a dust bath is essential to keep a chinchilla's coat in good condition, and they should have access for about 30 minutes a day. Where possible the relative humidity should be kept below 80%.

## MISCELLANEOUS CONDITIONS

### *FUR-SLIP*

*Cause and pathogenesis*

Chinchilla hair is loosely attached. Animals that are roughly handled, frightened, or are fighting will rapidly shed a patch or patches of fur. This is a natural defence mechanism, commonly known as 'fur slip'.

*Clinical signs*

A well circumscribed area of clean and smooth skin is left where the hair has been pulled out. Where it has grown back in the past after previous episodes there may be a patchy appearance to the coat.

*Diagnosis and therapy*

Diagnosis is based on history, clinical signs and rule out of other differentials such as barbering and infections' especially dermatophytosis. Therapy is not necessary, however regrowth may take several months and produce an uneven patchy pattern of hair growth (Scott *et al*. 2001). Handling should always be as gentle as possible, but some animals inevitably struggle and fur slip may be unavoidable. Chinchilla owners (especially of show animals) should be warned of this possibility before the veterinary surgeon handles the animal.

### *FUR RING*

*Cause and pathogenesis*

Adult male chinchillas, especially breeding animals, can accumulate a ring of matted fur around the base of the penis inside the prepuce.

*Clinical signs*

The presence of matted hair around the base of the penis can lead to paraphimosis.

**Table 16.1** Rodent formulary.

| Drug | Dose | Comments |
|---|---|---|
| *Antibacterials* | | |
| Ampicillin | 20–100 mg/kg p.o., s.c., i.m. bid | Mice, rats, gerbils |
| Chloramphenicol | 50 mg/kg p.o., s.c., i.m. | |
| Ciprofloxacin | 10–20 mg/kg p.o. bid | |
| Doxycycline | 2.5–5 mg/kg p.o. bid | |
| Enrofloxacin | 5–10 mg/kg p.o., s.c., i.m. bid or 10–20 mg/kg p.o., s.c., i.m. sid | |
| Gentamicin | 5–8 mg/kg s.c., i.m., divided tid–sid | Increased efficacy if given once daily. Nephrotoxic. |
| Marbofloxacin | 5 mg/kg p.o., s.c., SID | Hedgehog |
| | 5 mg/kg p.o., s.c., BID | Chinchilla, guinea pigs |
| | 10 mg/kg p.o., s.c., BID | Mouse, rat, hamster |
| Oxytetracycline | 10–20 mg/kg p.o. tid | Toxicity in guinea pigs reported. |
| Trimethoprim / sulphadiazine | 30 mg/kg p.o., s.c., i.m. bid | Paediatric banana-flavoured suspension useful p.o. |
| Tylosin | 10 mg/kg p.o., s.c., i.m. sid | Toxicity in guinea pigs reported. |
| *Antifungals* | | |
| Griseofulvin | 25 mg/kg p.o. sid 14–60 days or 1.5% in DMSO topically for 5–7 days, or paediatric suspension 250 mg/kg p.o. daily for ten days × three treatments | |
| Enilconazole | 0.2% wash / dip every seven days, or spray groups 50 mg/m$^2$ twice weekly for 20 weeks | |
| Itraconazole | 20 mg/kg p.o. sid | Guinea pigs |
| Terbinafine | 40 mg/kg p.o. sid or topical cream | Guinea pigs |
| Lime-sulphur dip | 1 : 40 dilution every seven days | |
| Miconazole | Topical cream | Guinea pigs |
| Mupirocin | Topical cream | Guinea pigs |
| Butenafine | Topical cream | Guinea pigs |
| *Antiparasitics* | | |
| Amitraz | 100 ppm bath once a week | |
| Ivermectin | 0.2–0.4 mg/kg s.c. every 10–14 days × three doses | Toxicity in some strains of mice. |

**Table 16.1** *Continued*

| Drug | Dose | Comments |
|---|---|---|
| Selamectin | 6–12 mg/kg topically | |
| Imidacloprid | Up to 40 mg topically | Chinchillas |

p.o. = orally; i.m. = intramuscularly
s.c. = subcutaneously; SID = once daily
BID = twice daily; ppm = part per million

*Diagnosis and therapy*

Diagnosis is based on clinical signs. Treatment involves lubrication and gentle removal of the ring. Sedation or anaesthesia is usually necessary. The author has also seen thick rings of smegma-like material causing similar lesions in two animals.

## NEOPLASIA

There are no reports of skin neoplasia in chinchillas, but it should be assumed that they can arise, and should be considered as a differential diagnosis for cutaneous masses.

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Chapter 17

# Skin Diseases and Treatment of Ferrets

## BACTERIAL DISEASE

### *PYODERMA*

*Cause and pathogenesis*

Bacterial skin disease in uncommon in the ferret but when it occurs it is caused by *Staphylococcus aureus* or *Streptococcus* spp. Infections usually occur secondary to trauma (bite wounds) or through self-inflicted trauma due to ectoparasitic infestation. Bites are particularly common during the breeding season especially on the neck of the female.

Staphylococcal or streptococcal cellulitis of the neck can be associated with dental disease and mandibular osteomyelitis.

*Clinical signs*

Lesions can be very variable in presentation ranging from superficial and follicular lesions to areas of furunculosis.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs and fine-needle aspirates / impression smears from lesions. Therapy should be undertaken using antimicrobial shampoos as well as antibiotics, where possible based on culture and sensitivity.

### *ABSCESSES*

*Cause and pathogenesis*

Abscesses, especially those located around the mouth, can be caused by sharp bones in a poor diet. Abscesses elsewhere may be caused by fighting. In addition to Gram-negative bacteria, infection can also be caused by *Corynebacterium* spp., *Pasteurella* spp., *Actinomyces* spp., and *E. Coli* resulting in abscesses, deep pyoderma or cellulitis.

*Clinical signs*

Fluctuant areas of swelling which are warm and painful to the touch.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs and fine-needle aspirate of the lesion. Treatment usually involves lancing and cleaning the abscess under anaesthesia, followed by antibiotics based on culture and sensitivity. Recovery is usually uneventful.

## *LUMPY JAW*

*Cause and pathogenesis*

*Actinomyces* spp. can also cause 'lumpy jaw'-type lesions in ferrets.

*Clinical signs*

Affected animals have nodules or abscesses in the neck that can discharge green-yellow pus.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs, impression smears from lesions as well as culture and sensitivity. Treatment is by curettage and drainage plus antibiosis. The lesions can respond to high dose penicillin or tetracycline.

# FUNGAL DISEASE

## *DERMATOPHYTOSIS*

*Cause and pathogenesis*

Dermatophytosis is rare in ferrets. However when it occurs it is usually caused by either *Microsporum canis* or *Trichophyton mentagrophytes.* Young and immunosuppressed animals are most susceptible to developing infection. The disease does pose a zoonotic risk. Transmission is by direct contact from a carrier animal or from infected fomites. Co-habiting cats may be the source of infection.

*Clinical signs*

Cutaneous lesions can present in a variety of ways including areas of annular alopecia, broken hairs, diffuse scaling as well as erythema and crusting. Pruritus is not normally seen.

*Diagnosis and therapy*

Diagnosis is made on the basis of microscopic examination of affected hairs. Skin scrapes and hair pluckings may be submitted for fungal culture. Some isolates of *M. canis* may fluoresce. Treatment is with topical application of anti-fungals such as enilconazole or miconazole. Systemic griseofulvin is rarely needed but can be used at 25 mg/kg per day for 21–30 days (Collins 1987). Spontaneous remission is also reported (Collins 1987, Fox 1988).

Environmental disinfection is important. Bleach diluted 50:50 with warm water can be used to clean out the environment. Cages etc. should be well rinsed and aired before animals are allowed to return.

## *OTHER FUNGAL DISEASES*

*Blasotomyces dermatitidis* has been reported in one ferret with pneumonia and an ulcerated footpad.

Histoplasmosis and coccidiomycosis have also been diagnosed as the cause of subcutaneous nodules (Scolt *et al*. 2001).

# VIRAL DISEASE

## *CANINE DISTEMPER VIRUS*

*Cause and pathogenesis*

Ferrets are highly susceptible to canine distemper virus. The disease is caused by a paramyxovirus. The ferret is acutely susceptible and mortality reaches 100%. Transmission is by direct contact, aerosol and fomites.

*Clinical signs*

Seven to ten days post-infection ferrets exhibit anorexia, fever and a mucopurulent ocular-nasal discharge. A characteristic rash under the chin and in the inguinal area is then seen at 10–15 days. Foot pads and nasal pads often undergo swelling and hyperkeratosis. Brown, crusted lesions occur on the chin, nose and perianal area. Terminally, the infected ferret develops nervous signs, convulsions and always dies.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs. Diagnosis can be made by serum antibody titres and fluorescent antibody tests for viral antigen on blood or conjunctival smears (Kelleher 2001). There is no treatment and prevention is by vaccination. Many ferrets are safely vaccinated using a canine vaccine. The USA is

the only country with a univalent canine distemper vaccine available for use in ferrets. Multivalent modified live vaccines produced for dogs can be used provided they are derived from non-ferret cell lines. Advice from the manufacturer should be sought on the use of their product in ferrets (Schoemaker 2002).

## ECTOPARASITES

Ectoparasites of importance in the ferret include:

**Mites**

- Psoroptidae *Otodectes cynotis.*
- Sarcoptidae *Sarcoptes scabiei.*

**Ticks**

- *Ixodes ricinus.*

**Insects**

- Fleas.

**Helminths**

- *Hypoderma* spp., *Cuterebra* spp.
- *Dracunculus insignis.*

## *PSOROPTIDAE*

### *OTODECTES CYNOTIS*

*Cause and pathogenesis*

The ear mite *Otodectes cyanotis* can affect ferrets as well as cats and dogs. It is normally found in the external auditory meatus but can also be found in the inner ear in severe cases. Mites feed on lymph, blood and skin debris. Infestation causes intense irritation. Transmission is by direct contact. The life cycle of the mite is three weeks.

*Clinical signs*

Ferrets naturally have dark brown earwax. Their ears need cleaning regularly. This can be undertaken using mineral oil or a mild ceruminolytic. It can be difficult to differentiate a normal aural discharge from a ferret's ear from that of a mite infestation. The ear canal will become congested with exudate, wax and mites leading to irritation, inflammation ulceration and sometimes convulsions. The ferret will head shake and sit with the head lowered. Ectopic disease can occur on the feet and tail tip.

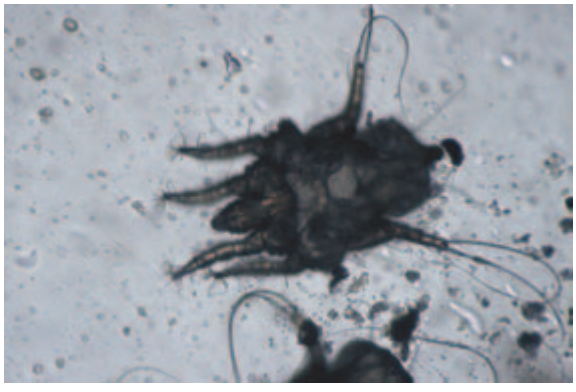

**Fig. 17.1** Otodectes mites.

*Diagnosis and therapy*

Diagnosis is by examination of aural discharge in potassium hydroxide or liquid paraffin. Mites are oval and their legs protrude from body shape (Figure 17.1). The first pair of legs end in short unsegmented stalks with suckers. Treatment can be undertaken with systemic ivermectin which is very effective. Topical treatments are often ineffective in ferrets because the ear canal is so small that medication may not penetrate.

## *SARCOPTIDAE*

### *SARCOPTES SCABIEI*

*Cause and pathogenesis*

*Sarcoptes scabiei* is the cause of sarcoptic mange in ferrets. Dogs can act as a source of contagion for ferrets. Mites burrow in tunnels in the skin, especially in sites which are poorly haired. In severe cases extensive areas can be affected. This is a zoonotic disease.

*Clinical signs*

*Sarcoptes scabiei* causes both generalised alopecia and intense pruritus or localised lesions of the toes and feet. Lesions are particularly prominent on sparsely haired sites. The skin becomes alopecic and lichenified with associated exudation and crusting. Nails can become deformed and slough.

*Diagnosis and therapy*

Diagnosis is by skin scrapings but false negative results are common. The mite is a round mite, only the first pair of legs protrude beyond the body shape. These

legs are short and end in long unsegmented stalks with suckers. Treatment is with ivermectin at 0.2–0.4 mg/kg subcutaneously repeated every 7–14 days for three doses. Affected and in-contact animals should be treated and the environment thoroughly cleaned.

## *TICKS*

### *IXODES RICINUS*

*Cause and pathogenesis*

*Ixodes ricinus* is the most important tick to affect the ferret. It is especially important in animals which are used for outdoor hunting.

*Clinical signs*

Heavy infestation can cause anaemia, and ticks can act as vectors for many different diseases.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs. Ticks can be removed manually or treated with systemic ivermectin at 0.4 mg/kg.

## *FLEAS*

*Cause and pathogenesis*

Cat fleas (*Ctenocephalides felis*) and dog fleas (*C. canis*) can infest ferrets.

*Clinical signs*

Mild to intense pruritus is seen, generally around the neck leading to self-inflicted trauma, presenting as traumatic hair loss (Figure 17.2) and excoriation. Occasionally alopecia may be seen on the neck and thorax.

*Diagnosis and therapy*

Diagnosis is made by catching the fleas or by a positive wet-paper test to identify the presence of flea faeces. The ferret, and any cats and dogs, plus the environment, should be treated. Products approved for cats can be used in ferrets. Spray or pump products such as fipronil should be measured carefully and applied to a cloth which is then applied to the ferret to avoid overdose. Dichlorvos-

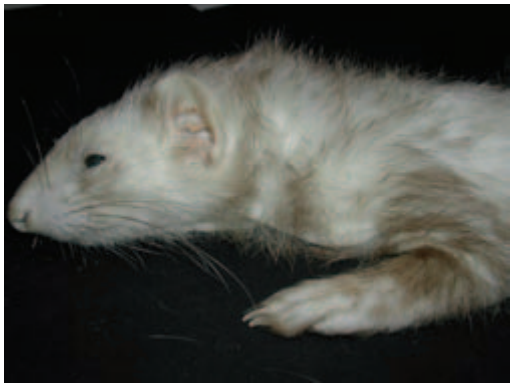

**Fig. 17.2** Hair loss around the neck of a ferret due to fleas.

impregnated flea collars are not recommended for ferrets because they can have toxic effects. Imidacloprid has been used safely and effectively (Kelleher 2001) and lufenuron may be effective when given at cat dosages (Orcutt 1997).

## ENDOCRINE DISEASE

### *HYPERADRENOCORTICISM*

*Cause and pathogenesis*

Hyperadrenocorticism is reported to occur commonly in middle-aged ferrets in the USA and is associated with adrenocortical hyperplasia, adenoma or adenocarcinoma (Keeble 2001; Rosenthal 1997). Pituitary-dependent hyperadrenocorticism has not been recognised in ferrets.

The incidence in the UK has not been reported, but it seems to be rare. One study conducted at Utrecht University found the incidence in the Dutch ferret population to be 0.55% with a strong correlation observed between age of neutering and age at time of diagnosis (Schoemaker *et al*. 2000). It has been speculated that the practice of early neutering, which is common place in America, may predispose towards the development of this condition in later life.

The onset is two to eight years old (average three and a half to four years (Scott *et al*. 2001; Keeble 2001)).

Studies in ferrets have now shown that hyperadrenocorticism is a condition independent of adrenocortictophic hormone (ACTH) or alpha-melanocyte stimulating hormone (MSH) (Schoemaker *et al*. 2002a) and that the disease results from the expression of luteinising hormone receptors on sex-steroid producing adrenocortical cells (Schoemaker *et al*. 2002b).

*Clinical signs: cutaneous*

Over 90% of ferrets with adrenal gland disease have some degree of alopecia this may be bilaterally symmetrical (Figure 17.3) (Keeble 2001).

Hair is easily epilated and is lost progressively over the perineum, tail, flanks, sides and back (Figure 17.4).

Comedones may be present (Figure 17.5).

Over 30% of cases may be pruritic and, occasionally, this is the only cutaneous sign.

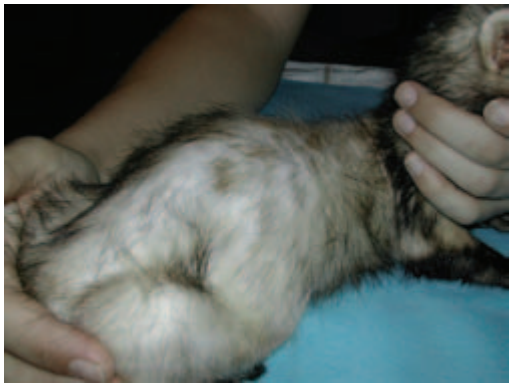

**Fig. 17.3** Bilaterally symmetrical alopecia in a ferret with endocrine disease.

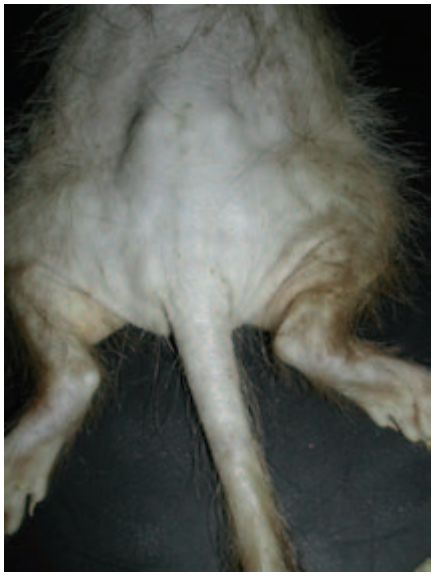

**Fig. 17.4** Comedones and alopecia on the tail of a ferret.

**Fig. 17.5** Comedones on the ventral abdomen of the ferret.

*Clinical signs: systemic*

Over 70% of females will have a swollen vulva and a seromucoid discharge.

Castrated males may show a return of male sexual behaviour.

Prostatic hyperplasia, due to increased androgen levels, may cause partial or complete urethral obstruction and stranguria.

On physical examination adrenal gland enlargement may be palpable.

*Diagnosis and therapy*

Diagnosis is based on history, clinical signs, abdominal palpation and elevated plasma androgen (androstenedione, dehydroepiandrosterone sulphate), oestradiol and 17-hydroxyprogesterone levels. It is recommended that all four hormones are assayed; elevation of one or more indicates hyperadrenocorticism.

Pancytopaenia may be present and serum biochemical values are usually normal. Abdominal ultrasonography is the most useful diagnostic tool with visualisation of the enlarged adrenal glands. The left gland is medial to the cranial pole of the kidney and is normally 6–8 mm in length. The right gland is cranial to the cranial pole of the kidney, covered by the caudate lobe of the liver, and very close to the caudal vena cava. It is normally 8–11 mm in length (Kelleher 2001).

*Diagnosis is not based on serum cortisol levels, ACTH stimulation tests or dexamethasone suppression tests as these are often normal in clinically affected ferrets.*

Vulval swelling due to hyperoestrogenism (see below) can be differentiated by a lack of response to an injection of human chorionic gonadotrophin once weekly for two doses.

Preferred treatment is surgical removal.

Where both adrenal glands are affected then removal of one and partial removal of the other is advocated (Weiss *et al*. 1999). Post-operative corticosteroid therapy

**Table 17.1** Table of drugs available for medical therapy of hyperadrenocorticism in ferrets.

| Drug | Dosage | Comments |
|---|---|---|
| Mitotane | 50 mg p.o. daily for seven days, followed by a maintenance dose of 50 mg p.o. every third day, is recommended. | Rarely successful, response variable. Side-effects reported included hypoglycaemia. |
| Ketoconazole | 15 mg/kg p.o. every 12 hours | Ineffective. |
| Synthetic GnRH analogue leuprolide | A subcutaneous injection of 100 µg/kg of leuprolide acetate depot is given every three to eight weeks. Clinical response is seen after the third injection with complete recovery from clinical signs after six months. The interval between doses may then be increased. | Effective in resolving the clinical signs associated with adrenal gland disease (Orcutt 1997, Johnson-Delaney 1998). |
| Flutamide* | | Androgen blocker, may also be useful especially in ferrets with prostatic enlargement. |
| Arimidex* | | Aromatoase inhibitor, decrease the effects caused by androgens (Weiss 1999). |

*Use of these human drugs is currently under evaluation.
p.o. = orally

is not routinely given. In unilateral cases improvement is seen in two to eight weeks with complete recovery in five months (See Table 17.1).

## *HYPEROESTROGENISM*

*Cause and pathogenesis*

Hyperoestrogenism is probably the most common endocrine condition encountered in practice in ferrets in the UK (Keeble 2001). If unmated, or not stimulated to ovulate, then as many as 50% of females may develop aplastic anaemia after prolonged oestrus (up to six months). High levels of oestrogen lead to oestrogen suppression of the bone marrow and resulting anaemia with pancytopaenia. Other causes of hyperoestrogenism include an ovarian remnant following ovariohysterectomy or adrenal neoplasia.

Pseudopregnancy following a sterile mating has been recorded.

*Clinical signs: cutaneous*

Bilateral symmetrical alopecia is seen as well as ecchymoses and petechiation of the mucus membranes and skin.

*Clinical signs: systemic*

Signs include vulval swelling, pallor of the mucus membranes, systolic murmurs, weak pulses, posterior paresis (due to haemorrhagic myelomalacia) and systemic infections secondary to leucopaenia.

*Diagnosis and therapy*

Diagnosis is based on history, clinical signs and demonstration of anaemia. Treatment is supportive care (intravenous fluids, syringe feeding, iron and vitamin B supplementation and prophylactic antibiotics) and a cessation of oestrus. A blood transfusion is indicated if the packed cell volume (PCV) is less than 15%. Ovariohysterectomy is often too risky in debilitated animals and human chorionic gonadotrophin has been successfully used at 100 IU by intramuscular injection. The injection is repeated after seven days if signs of oestrus are still apparent. Gonadotrophin-releasing hormone has also been used at 20 µg by intramuscular or subcutaneous injection repeated after one to two weeks. Once stabilised, ovariohysterectomy should be carried out.

This condition is easily prevented in female ferrets by routine ovariohysterectomy, mating with a vasectomised male or by use of proligestone subcutaneous injection prior to the onset of the breeding season. Megestrol acetate can also be used to prevent onset of oestrus but it should be noted that both these drugs have been associated with the development of pyometra.

## *HYPOTHYROIDISM*

This condition has never been documented in ferrets despite anecdotal reports of it causing an endocrine alopecia.

# MISCELLANEOUS CONDITIONS

## *EXCESS ODOUR*

*Cause and pathogenesis*

Ferrets' skin naturally contains numerous sebaceous glands which cause the natural musky odour and sometimes greasy feel of the coat.

*Clinical signs*

Male ferrets are always more odiforous and secretions can be so profuse as to make the coat of albino animals yellow and dirty in appearance.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs. It is not possible to 'de-scent' a ferret but shampooing (no more than monthly is recommended), feeding on a commercial pelleted diet and neutering will all help to minimise offensive odours.

## *SEASONAL ALOPECIA*

*Cause and pathogenesis*

Normal thinning of the coat occurs in ferrets in warm weather.

*Clinical signs*

A bilaterally symmetric alopecia of the tail, perineum and inguinal area can often occur during the breeding season (March to August in females and December to July in males). This is often more pronounced in females.

*Diagnosis and therapy*

It is important to rule out endocrine causes of hair loss (see above). Therapy is not normally necessary.

## *TELOGEN DEFLUXION*

*Cause and pathogenesis*

Telogen defluxion is seen two to three months after a stressful event. This can include pregnancy, suckling of young or debilitating systemic disease. All of the hairs move through into the telogen (resting phase) and are subsequently lost.

*Clinical signs*

The animal will undergo a very heavy moult. Often overt alopecia is not seen, only generalised thinning of the coat.

*Diagnosis and therapy*

Diagnosis is made on the basis of diagnostic rule outs combined with clinical history and presenting signs. Therapy is not necessary providing the cause of stress has been removed.

## *ATOPY*

*Cause and pathogenesis*

Atopy is an allergic reaction to environmental allergens. It is important to check for ectoparasites and other allergies before making a diagnosis.

*Clinical signs*

Presumptive atopy has been reported (Scott *et al*. 2001) presenting as symmetric non-lesional pruritus over the trunk, rump and paws. Pruritus may also be directed at flexural surfaces and the perianal area (Figures 17.6, 17.7).

*Diagnosis and therapy*

Diagnosis is made on the elimination of other factors especially food and ectoparasites. A definitive diagnosis can be made through intradermal allergy testing.

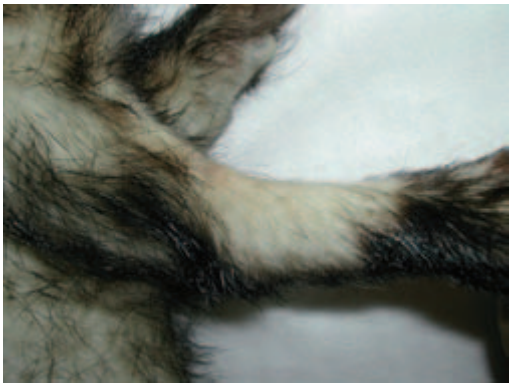

**Fig. 17.6** Saliva staining and self-inflicted trauma in a ferret due to allergy.

**Fig. 17.7** Perianal pruritus in a ferret due to allergy.

Therapy can be with antihistamines, steroids, oil supplementation or by desensitisation using allergy vaccines.

## *FOOD HYPERSENSITIVITY*

One case has been noted, responsive to a commercial hypoallergenic diet for cats (Scott *et al*. 2001).

## *BLUE FERRET SYNDROME*

*Cause and pathogenesis*

This is an idiopathic syndrome reported by Burgmann (1991). It can affect ferrets of either sex, neutered or intact. It is often seen in ferrets clipped for surgery during catagen – the intermediate phase of the growth cycle.

*Clinical signs*

Animals present with a bluish discolouration of the abdominal skin but are otherwise asymptomatic. The clipped skin area remains hairless and then turns blue and it seems that the hair follicles are manufacturing melanin to be incorporated into growing hairs.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs. Therapy is not necessary as hair regrowth begins within one to two weeks after the blue colour appears (Scott *et al*. 2001). The colouration disappears within a few weeks.

## NEOPLASIA

*Cause and pathogenesis*

Cutaneous neoplasia is relatively common in ferrets and is reported as the third most common form of neoplasia (Li *et al*. 1995) (See Table 17.2).

*Diagnosis and therapy*

In many cases a useful diagnosis can be made based on Diff Quik™ stains of fine-needle aspirates, particularly in the case of mast cell tumours. Biopsy is usually necessary for other neoplastic lesions. Therapy is undertaken in most cases by surgical removal of the lesions (See Table 17.3).

**Table 17.2** Neoplasia in ferrets.

| Tumour | Incidence | Clinical signs |
|---|---|---|
| Sebaceous epitheliomas | 58% in a study of 57 cutaneous neoplasms (Parker & Picut 1993). Average age at diagnosis was 5.2 years and 70% of animals were female. | Pedunculated or plaque-like mass that can become ulcerated. |
| Mast cell tumours | 16% of all cutaneous neoplasms (Parker & Picut 1993). | Single or multiple well-circumscribed hairless nodules, often ulcerated and crusted with black exudates. Some are pruritic. |
| Squamous cell carcinomas, adenocarcinomas, epitheliotropic cutaneous lymphoma, haemangioma and fibroma are among other rarer tumours reported in this species. | | |

**Table 17.3** Ferret formulary.

| Drug | Dose | Comments |
|---|---|---|
| *Antibacterials* | | |
| Amoxicillin | 10–20 mg/kg p.o. s.c. bid | |
| Amoxicillin/clavuanate | 12.5–25 mg/kg p.o. bid | |
| Ampicillin | 5–30 mg/kg p.o., s.c., i.m., i.v. bid | |
| Cephalexin | 15–25 mg/kg p.o. bid–tid | |
| Chloramphenicol | 50 mg/kg p.o., s.c., i.m. bid | |
| Ciprofloxacin | 5–15 mg/kg p.o. bid or 10–30 mg/kg p.o. sid | |
| Clindamycin | 5.5–10 mg/kg p.o. bid | |
| Enrofloxacin | 5–10 mg/kg p.o., s.c., i.m. bid or 10–20 mg/kg p.o., s.c., i.m. sid | |
| Metronidazole | 20 mg/kg p.o. bid | |
| Oxytetracycline | 20 mg/kg p.o. tid | |
| Penicillin G | 40 000 IU/kg i.m. sid | |
| Trimethoprim / sulpha | 15–30 mg/kg p.o., s.c. bid | |
| *Antifungals* | | |
| Griseofulvin | 25 mg/kg p.o. sid for 21–30 days. | |
| *Antiparasitics* | | |
| Amitraz | Topical to affected area three to six times every 14 days. | Demodicosis. Use full concentration. |

**Table 17.3** *Continued*

| Drug | Dose | Comments |
|---|---|---|
| Ivermectin | 0.2–0.4 mg/kg s.c. | |
| Fipronil | Topical. Two drops per ear for ear mites or two to three sprays per ferret of 2.5 g/litre. | |
| Imidocloprid | One cat dose (40 mg) divided onto two to three spots on dorsum. | |
| Lufenuron | 30–45 mg/kg p.o. once a month. | |
| Permethrin / pyrethrins | Topical every seven days. | Use products safe for kittens and puppies. |
| Selamectin | Topical, maximum 15 mg, monthly. | |
| *Hormonal agents* | | |
| Leuprolide acetate | 100 µg/kg every three to eight weeks. | |
| Human chorionic gonadotrophin | 100 IU i.m. | |
| Gonadotrophin-releasing hormone | 20–25 µg i.m., s.c. repeated after one to two weeks. | |
| Proligestone | 50 mg s.c. If no response give 25 mg after seven days. | Associated with pyometra. |

p.o. = orally; s.c. = subcutaneously
i.m. = intramuscularly; SID = once daily
BID = twice daily

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Chapter 18

# Skin Diseases and Treatment of Gerbils

## BACTERIAL DISEASE

### *STAPHYLOCOCCAL DERMATITIS*

*Cause and pathogenesis*

*Staphylococcus aureus* has been associated with an acute primary dermatitis, mainly in young gerbils. It can be reproduced experimentally by inoculation with staphylococcal isolate in the nasal region (Peckham *et al*. 1974). It can cause high morbidity and mortality. Staphylococcal infections are usually secondary to trauma (cage-related injuries, bite wounds), ectoparasites or accumulated harderian gland secretions in nasal dermatitis in older animals. *S. aureus*, *S. saprophyticus* and *S. xylosus* may all be isolated in cases of nasal dermatitis (see pp. 225–6 below, in Environmental and behavioural conditions).

*Clinical signs*

Infections due to trauma from the cage, such as rough bedding, are usually seen on the face. Lesions due to bite wounds are usually seen on the rump, head, tail and perianal areas. Lesions due to harderian gland accumulations are typically seen on the nose and the peri-ocular skin. If infection is superficial it tends to produce erythematous, alopecic oozing lesions (Figure 18.1) with associated crust and scale. Deep infection occurs as abscesses and fistula formation.

*Diagnosis and therapy*

The presence of bacteria can be confirmed by stained impression smears of exudates or by fine-needle aspirates of lesions. Culture is usually unnecessary. Therapy must include identification and management of underlying factors, daily cleaning with 1–2% chlorhexidine and the administration of systemic antibiotics.

### *SEBACEOUS GLAND DERMATITIS*

*Cause and pathogenesis*

Gerbils have a large, ventral abdominal sebaceous gland, which is used in territorial marking and scent identification of pups. It is a normal finding in all gerbils

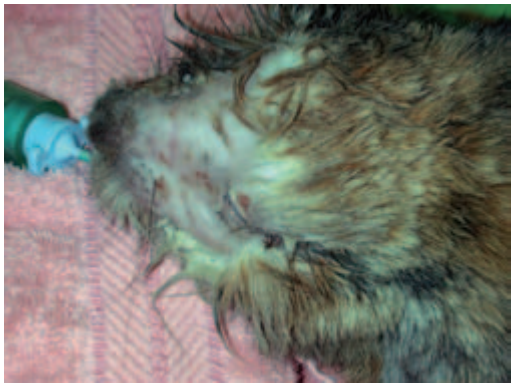

**Fig. 18.1** Alopecia and secondary staphylococcal infection on neck of a gerbil.

**Fig. 18.2** Normal ventral scent gland in a gerbil.

(Figure 18.2). Size is androgen dependent, being larger in males. The gland may become inflamed and infected with staphylococcal and streptococcal bacteria.

*Clinical signs*

Ventral gland appears reddened and ulcerated.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs and impression smears of exudates, which reveal the presence of infection. Biopsy is needed for a definitive rule out of neoplasia. Early neoplastic changes may appear similar and should be considered, particularly in older animals. Treatment is by using topical or systemic antibiotics. Topical antibiotic and steroid cream followed by an abdominal bandage wrap has been suggested (Laber-Laird 1996), but the gerbil may find this distressing and bandages are frequently chewed off. Healing is difficult due to the location of the gland and constant abrasion on the cage substrate. If non-responsive to medical treatment, total gland excision is recommended.

## VIRAL DISEASE

There are no reports of viral cutaneous disease in gerbils.

## FUNGAL DISEASE

### *DERMATOPHYTOSIS*

*Cause and pathogenesis*

Dermatophytosis is a rare disease in the gerbil. When present is is usually caused by *Microsporum gypseum* or *Trichophyton mentagrophytes*.

*Clinical signs*

Dermatophyte infection leads to focal alopecia and hyperkeratosis.

*Diagnosis and therapy*

Diagnosis is made on microscopic hair examination and fungal culture. Infections are zoonotic. Treatment involves disinfection of the environment, clipping of the surrounding hair and oral griseofulvin at 25–30 mg/kg by mouth daily for three weeks. For large groups, in-feed griseofulvin is possible (0.75 mg/kg of feed, 10 g/kg of 7.5% powder in dry feed). Enilconazole washes twice weekly, until two cultures are negative, have also been described.

## ECTOPARASITES

Ectoparasite infections are not common in gerbils compared with other small rodent species.

### *DEMODEX MERONI*

*Cause and pathogenesis*

*Demodex meroni* infection has been reported (Reynold & Gainer 1968; Schwarzbratt *et al*. 1974). Generalised demodicosis infection is often associated with immunosuppression and underlying disease such as old age, poor nutrition and husbandry-related problems such as overcrowding and poor ventilation.

*Clinical signs*

*Demodex meroni* is species specific and causes alopecia, scaling and focal ulcerative dermatitis with secondary bacterial infections. Lesions are usually found on the face, thorax, abdomen and limbs.

*Diagnosis and therapy*

Diagnosis is by mite demonstration using microscopy of hair plucks and deep skin scrapes. Treatment consists of topical amitraz applications, 100 ppm, three to six times at two week intervals. Demodicosis may respond to ivermectin at 0.2–0.4 mg/kg s.c., three treatments, seven to ten days apart.

## *ACARUS FARRIS*

*Cause and pathogenesis*

The fur mite *Acarus farris* has been associated with clinical signs in gerbils.

*Clinical signs*

Alopecia, scaling and thickening of the skin has been identified on the tail, spreading to the hind quarters then the head. Chronic lesions have been associated with self-inflicted trauma leading to excoriation and secondary skin changes.

*Diagnosis and therapy*

Diagnosis was based on microscopic examination of hair plucks (Jacklin 1997).

Ivermectin was found to be of little use for therapy. However, environmental changes such as decreased humidity or the introduction of a new food, coupled with a single application of Fipronil® spray have been shown to be curative.

## *OTHER ECTOPARASITES*

Other ectoparasites include *Liponyssoides sanguineous*, the mouse fur mite, although this has not been associated with any adverse clinical signs (Levine & Lage 1985).

# ENDOCRINE DISEASE

## *CYSTIC OVARIAN DISEASE*

*Cause and pathogenesis*

Cystic ovaries are common in older female gerbils, with nearly fifty percent of animals over 400 days old being affected. Cysts vary in size, but can grow up to 5 cm in diameter and are often bilateral.

*Clinical signs*

Reproductive performance is reduced and eventually the gerbil will become infertile. Symmetrical alopecia and poor coat quality is seen clinically.

Abdominal distension occurs in severe cases, often associated with dyspnoea.

*Diagnosis and therapy*

Diagnosis is based on clinical signs, abdominal palpation, abdominal radiography or ultrasonographic examination. Drainage by fine-needle aspiration under general anaesthesia may be attempted, but ovariohysterectomy is the treatment of choice.

## *HYPERADRENOCORTICISM*

*Cause and pathogenesis*

Hyperadrenocorticism has rarely been reported in ageing gerbils.

*Clinical signs*

Clinical signs and diagnosis are associated with bilateral symmetrical alopecia of the flanks and lateral thigh area, thinning and hyperpigmentation of the skin, polydipsia, polyuria and polyphagia. A disease complex has been described in breeding gerbils where development of hyperadrenocorticism appears to be linked with blood-vessel mineralisation, myocardial necrosis / fibrosis and diabetes. The exact relationship and aetiology has not been determined, but these lesions often occur simultaneously in breeding animals.

*Diagnosis and therapy*

Diagnosis is based on clinical signs. Therapy has not been described to date (Keeble 2001).

# ENVIRONMENTAL AND BEHAVIOURAL CONDITIONS

## *NASAL DERMATITIS ('SORE NOSE', 'FACIAL DERMATITIS')*

*Cause and pathogenesis*

This condition is extremely common in gerbils, particularly in sexually mature, group-housed animals which may be stressed by overcrowding and high humid-

ity levels. Incidence may be as high as 15% in some colonies. Hypersecretion of the harderian gland results in accumulation of porphyrin pigment around the nares. This is irritant and may lead to self trauma and secondary staphylococcal infection (Thiessen & Pendergrass 1982; Farrar *et al*. 1988; Bresnahan *et al*. 1983). Digging through abrasive bedding could be a predisposing factor. Harderian gland secretions may increase with stress, plus there may be lack of grooming in some situations.

*Clinical signs*

Lesions start as small focal areas of alopecia and crusting around the external nares (Figure 18.3), and may progress to involve the face, medial paws and abdomen with associated alopecia and dermatitis. Severe infection may be associated with debility and mortality.

*Diagnosis and therapy*

Diagnosis is based on clinical signs, bacterial culture and cytology of impression smears. Porphyrins will fluoresce under UV light. Improving husbandry and environmental temperature as well as reducing humidity (to less than 50%) will help resolve this problem. Provision of a sand bath will help improve fur quality and encourage grooming. Removal of the harderian gland has been described, although long-term effects of this surgery are not known (Farrar *et al*. 1988). Topical or systemic antibiotic treatment is indicated, plus addressing the underlying stressor.

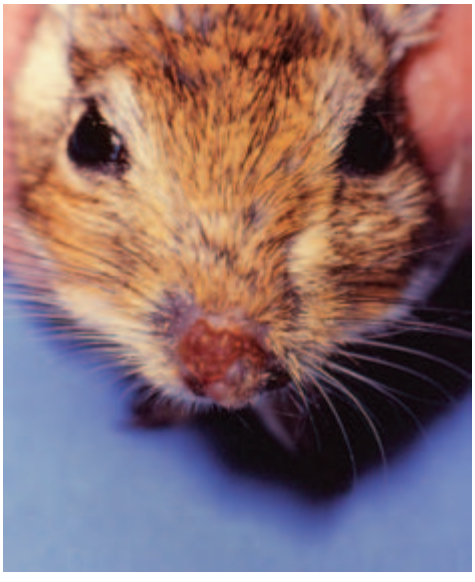

**Fig. 18.3** Nasal dermatitis on the face of a gerbil.

## *BALD NOSE*

*Cause and pathogenesis*

'Bald nose', in which there is hair loss from the dorsum of the nose and around the muzzle, is attributed to rubbing on wire cage feeders and wire cage bars, or burrowing in bedding.

*Clinical signs*

Short stubbly hair loss on the dorsum of the nose.

*Diagnosis and therapy*

Diagnosis is made on basis of clinical signs and rule out of other conditions such as dermatophytosis. Trichography (examination of hair plucks) can help establish if the hair loss is traumatic. Lesions may be prevented by feeding inside the cage or housing in a glass tank. It may also be an early form of nasal dermatitis (see p. 225) (Collins 1987).

## *BARBERING*

*Cause and pathogenesis*

Barbering may occur if gerbils are housed in large groups where a dominant individual will chew off the hair of another animal.

*Clinical signs*

Lesions appear as areas of traumatic hair loss on the dorsal head and tail base.

*Diagnosis and therapy*

Diagnosis is similar to that for bald nose. Management is by reducing stocking density, providing environmental enrichment and, if necessary, removing the individual responsible for the hair loss.

## *FIGHT WOUNDS*

*Cause and pathogenesis*

Fight wounds are usually associated with clashes between two aggressive individuals.

*Clinical signs*

Wounds commonly occur on the head, tail base, gluteals and perineum and are often associated with abscesses.

*Diagnosis and therapy*

Diagnosis is made by clinical signs, impression smears and/or fine-needle aspirates of nodular lesions to rule out differentials such as neoplasia.

Treatment is by clipping and lavage of the affected area, followed by treatment with topical and systemic antibiotics. The underlying cause should be addressed.

## *ROUGH COAT*

*Cause and pathogenesis*

If relative environmental humidity exceeds 50% gerbils will become rough coated.

Increased humidity in the cage may be caused by damp bedding, polyuria, diarrhoea, leakage of the water bottle. However, the rough coat may be a more general sign of ill health and stress. Cedar or pine shavings also can cause matted, greasy-looking fur (Ellis & Mori 2001).

*Clinical signs*

The normal sleek, smooth hair coat is replaced by one which is greasy and stands out.

*Diagnosis and therapy*

Diagnosis is made by history, clinical signs and diagnostic rule out of differentials such as infections. Treatment should be directed to correcting the underlying environmental conditions.

# MISCELLANEOUS CONDITIONS

## *'TAIL SLIP'*

*Cause and pathogenesis*

'Tail slip' is common following incorrect handling (Donnelly 1997). A gerbil's tail skin is very thin and easily peels off. If the tail is grasped, the skin will deglove at this site.

*Clinical signs*

The skin is lost from the tail, leaving exposed muscle and bone. The tail will slough if untreated, or there may be ascending infection from the damaged area.

*Diagnosis and therapy*

Diagnosis is made on the basis of history and clinical signs. Therapy is surgical. Amputation proximal to the injury site is indicated (Ellis & Mori 2001).

## *CONGENITAL ALOPECIA*

*Cause and pathogenesis*

Occasional litters may occur with areas of alopecia, abnormal hair pigmentation and stunted growth. The aetiology is unknown.

*Clinical signs*

Typically, hair is lost from the back, surrounding hair is diffusely thinned with accompanying leukotrichia. Affected pups are stunted and often die at weaning (three weeks old) (Collins 1987).

*Diagnosis and therapy*

Diagnosis is made on the basis of history and clinical signs. No therapy has been described. However surviving gerbils do grow back a normal hair coat.

## NEOPLASIA

*Cause and pathogenesis*

Neoplasia of the skin is relatively common in gerbils, particularly as they get older (Figure 18.4). Gerbils have a very high incidence of spontaneous neoplasms, and the skin is the second most common site (Collins 1987) (See Table 18.1).

Neoplastic lesions of the ventral scent gland tend to develop as raised ulcerated masses (Figure 18.5).

*Diagnosis and therapy*

Diagnosis is based on cytology via impression smears, fine-needle aspirates or excisional biopsy. Where tumours affect the ventral scent gland then recommended treatment is early total gland excision. Early excision is often curative, although local spread to inguinal lymph nodes can occur in advanced cases. Therapy of other lesions is wide surgical excision.

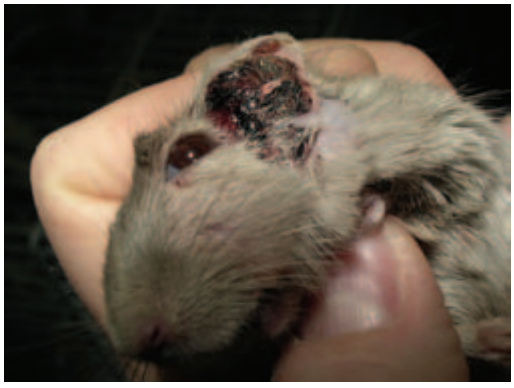

**Fig. 18.4** Malignant aural melanoma in an elderly gerbil. (Picture courtesy J. Fontaine.)

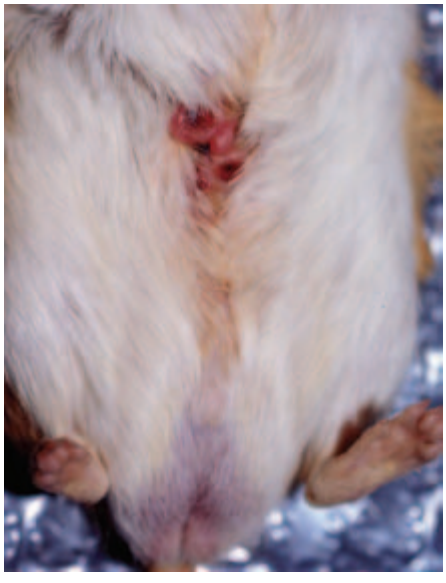

**Fig. 18.5** Tumour on ventral scent gland in a gerbil.

**Table 18.1** Types of neoplasia in gerbils.

| Type of tumour | Reference | Location | Comments |
|---|---|---|---|
| Sebaceous gland adenoma | Burgmann 1991 | Ventral scent gland | Very common |
| Squamous cell carcinoma | Jackson *et al*. 1966 | Ventral scent gland, feet, pinnae | Very common |
| Basal cell carcinomas | | Ventral scent gland | |
| Melanoma | Cramlet *et al*. 1974 | Paw, pinnae | Common |
| Melanocytomas | Cramlet *et al*. 1974 | Paw, pinnae | Common |
| Papilloma, subcutaneous fibrosarcoma, mammary gland adenocarcinoma | | | |

*Formulary*

See p. 202 in Chapter Sixteen on Chinchillas.

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Chapter 19

# Skin Diseases and Treatment of Guinea Pigs

## BACTERIAL DISEASE

### *STAPHYLOCOCCAL PYODERMA*

*Cause and pathogenesis*

Bacterial skin infections due to *Staphylococcus aureus* or *Staphylococcus epidermidis* are common in guinea pigs (Ellis & Mori 2001, White *et al*. 2003; Huerkamp *et al*. 1996). They are usually secondary to bites (Figure 19.1) or other wounds, self trauma (e.g. due to pruritus from ectoparasites), excessive grooming, or foreign body granulomas. Chronic wetting of the skin also predisposes to secondary bacterial pyoderma, such as that resulting from excessive salivation due to dental disease. Staphylococcal cheilitis, thought to be secondary to the feeding of abrasive or acidic foodstuffs, e.g. hay, apples, is also seen (Smith 1977) (See p. 247 under Miscellaneous conditions).

*Clinical signs*

Lesions can include alopecia, erythema, superficial suppuration, crusts, abscesses, ulcers and folliculitis. The location of the lesion is dependent on the underlying trigger. Bite wounds are usually found around the head, tail and rump.

*Diagnosis and therapy*

Diagnosis is based on clinical signs and bacterial culture. Treatment involves systemic antibiosis based on culture and sensitivity testing, and addressing any underlying cause. Treatment should be prompt as continuous immunostimulation due to infection in guinea pigs commonly leads to amyloidosis and organ failure.

### *EXFOLIATIVE DERMATITIS*

*Cause and pathogenesis*

*Staphylococcus aureus* has been reported as causing erythema and exfoliation of the epidermis in guinea pigs, caused by epidermal cleavage through the stratum granulosum (Ishihara 1980). Lesions were thought to be caused by a staphylococcal

**Fig. 19.1** Infection on the flank of the guinea pig due to a bite wound.

exfoliative toxin. Bacterial contamination and skin abrasions due to rusty floors were thought to be important inciting factors.

*Clinical signs*

Lesions were reported on the ventral abdomen and medial limbs. After a few days the skin became erythematous before fissuring occurred and large flakes of skin were desquamated. The condition spontaneously resolved after ten to fourteen days.

*Diagnosis and therapy*

Diagnosis is based on clinical signs, culturing of *S. aureus* and histopathological findings. Therapy is not deemed necessary as the condition resolves by itself.

## *PYODERMA CAUSED BY OTHER BACTERIA*

*Cause and pathogenesis*

Other bacteria less commonly seen as a cause of bacterial pyoderma include *Treponema* spp., *Streptococcus* spp., *Fusobacterium* spp. and *Corynebacterium* spp. Mixed infections are not uncommon and can occur with Staphylococcus.

*Clinical signs*

Lesions can be identical to those of Staphylococcal pyoderma.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs, examination of stained impression smears from lesions and culture and sensitivity. Treatment should be undertaken with systemic antibiotics, where possible based on sensitivity findings.

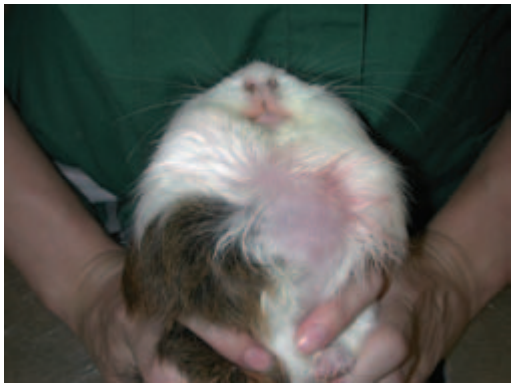

**Fig. 19.2** Abscess on the neck of the guinea pig.

## *ABSCESSES*

*Cause and pathogenesis*

Abscesses often occur as a result of fighting. Bacteria such as *Pseudomonas aeruginosa*, *Pasteurella multocida*, *Corynebacterium pyogenes*, *Staphylococcus* spp. and *Streptococcus* spp. are commonly cultured.

*Clinical signs*

Fluctuant areas of swelling often on the face (Figure 19.2), tail and rump.

*Diagnosis and therapy*

Diagnosis is established by history and clinical signs, as well as fine-needle aspirates from lesions. Treatment is by lancing, draining and flushing, or preferably by surgical excision. Systemic antibiotics should be given, based on bacterial culture and sensitivity of the abscess capsule.

# FUNGAL DISEASE

## *DERMATOPHYTOSIS*

*Cause and pathogenesis*

Dermatophytosis is common in guinea pigs and is invariably due to *Trichophyton mentagrophytes*. *Microsporum canis* and other species are rarely reported but have been used to induce experimental infections. Some guinea pigs can be asymptomatic carriers (6–14%) (Vangeel *et al*. 2000) and overt disease is commonly precipitated by overcrowding, poor husbandry, a low nutritional plane and other stressors such as high environmental temperature and humidity. Dermatophytes

are easily transmitted by direct contact or fomites. Owners should be made aware of the zoonotic potential of dermatophytosis.

*Clinical signs*

Lesions can be identified as non-pruritic scaling and alopecia occurs around the face, legs (Figure 19.3) and head, with the dorsum also affected in severe cases. Occasionally, more inflammatory pruritic pustules, papules, crusts (Figure 19.4), secondary bacterial dermatitis and delayed hypersensitivity reactions can occur. Neonates with severe infections may die.

*Diagnosis and therapy*

Diagnosis is made by microscopic examination of affected hairs and fungal culture. The affected animal and all in-contact animals should be treated. Various regimens are possible. For focal lesions, topical miconazole or mupirocin creams may be used once daily for two to four weeks. Topical griseofulvin at 1.5% in DMSO for five to seven days and topical butenafine once daily for ten days can

**Fig. 19.3** Alopecia and erythema on the foot of the guinea pig with *Trichophyton mentagrophytes*. (Picture courtesy Z. Alha i dari.)

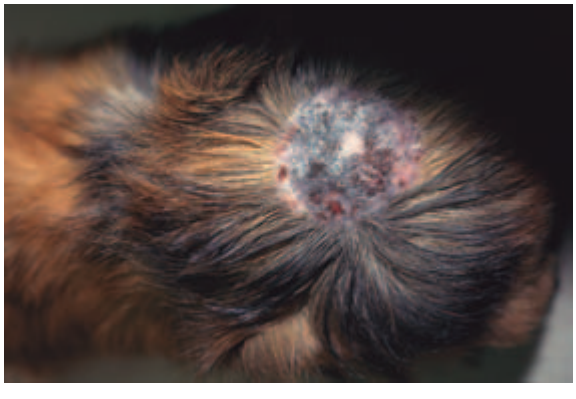

**Fig. 19.4** Scaling and crusting on the dorsum of the guinea pig with *Trichophyton mentagrophytes*. (Picture courtesy Z. Alha i dari.)

also be used (Huerkamp *et al*. 1996; Schaeffer & Donnelly 1997). However, more generalised treatment is usually necessary. Enilconazole dips at 0.2% or miconazole shampoos may be used once or twice weekly. Oral griseofulvin at 25 mg/kg p.o. once daily for three to five weeks given in a fatty acid supplement or at 0.75 mg/kg of feed can be used.

Griseofulvin paediatric suspension at 250 mg/kg p.o. once every ten days for three treatments has also been reported as effective (Schaeffer & Donnelly 1997). This drug is teratogenic and should not be used in pregnant animals. In experimentally induced dermatophyte infections oral itraconazole or fluconazole at 20 mg/kg per day (Nagino *et al*. 2000) and oral terbinafine at 40 mg/kg per day (Petranyi *et al*. 1987) have been used successfully. In colony situations, eradication of disease may be difficult, even with antifungal treatment, disinfection and isolation.

### *OTHER FUNGAL INFECTIONS*

*Cryptococcus neoformans* can cause ulcerative dermatitis, especially of the nose (Huerkamp *et al*. 1996).

Experimentally, skin lesions can be produced in guinea pigs by the cutaneous application of *Candida albicans* and *Malasssezia ovale*, but the significance of these in causing spontaneous lesions is unclear.

## VIRAL DISEASE

### *POXVIRUS*

*Cause and pathogenesis*

A poxvirus has been detected in association with cheilitis in two guinea pigs. A poxvirus was also detected in large fibrovascular proliferations in the thigh muscles of a group of eight-month-old guinea pigs (Hampton *et al*. 1968).

*Clinical signs*

Guinea pigs present with crusting ulcerated lesions around the lips and philtrum (Cully 1995).

*Diagnosis and therapy*

Diagnosis is by clinical signs and where possible virus isolation. Therapy has not been described.

## ECTOPARASITES

Important ectoparasites in guinea pigs include:

*Mites*

- Sarcoptidae – *Trixacarus caviae, Sarcoptes scabiei*.
- Atopomelidae – *Chirodiscoides caviae*.
- Myocoptidae – *Myocoptes musculinus*.
- Cheyletiellidae – *Cheyletiella parasitovorax*.
- Demodicidae – *Demodex caviae*.

*Insects*

- Lice – *Gliricola porcelli, Gyropus ovalis*.

## *SARCOPTIDAE*

### *TRIXACARUS CAVIAE*

*Cause and pathogenesis*

*Trixacarus* (Caviocoptes) *caviae* is the most significant ectoparasite of the guinea pig. Its lifecycle takes two to fourteen days. A symptomless carrier state can exist, with overt disease appearing in individuals or groups kept for a long time in isolation from other animals. Some guinea pigs may also remain unaffected even when kept in close contact with severely affected cage mates (White *et al*. 2003). A stressor such as old age, concurrent disease or hypovitaminosis C is often the trigger for clinical disease. The mite can cause dermatitis in humans.

*Clinical signs*

The mite causes intense pruritus leading to severe self trauma, and in some cases, fitting. Abortion and foetal resorption may be seen in pregnant animals. Lesions are seen on the shoulders, dorsum and flanks (Figure 19.5). Secondary bacterial infection is common. Chronic infection leads to lichenification and hyperpigmentation, crusts, scales and alopecia (Figure 19.6).

*Diagnosis and therapy*

Diagnosis is by skin scrapes. Deep skin scrapes reveal the typical round sarcoptiform body with suckers on legs I and II. Treatment is with ivermectin 200–400 $\mu$g/kg s.c. every ten days for three doses. There is some evidence that ivermectin is not absorbed orally in the guinea pig (Shipstone 1997). All in-contact animals should be treated and the housing thoroughly cleaned, as the mite can survive for some time off the host.

**Fig. 19.5** Alopecia on the ventrum of the guinea pig with *Trixacarus caviae* infestation. (Picture courtesy Z. Alha i dari.)

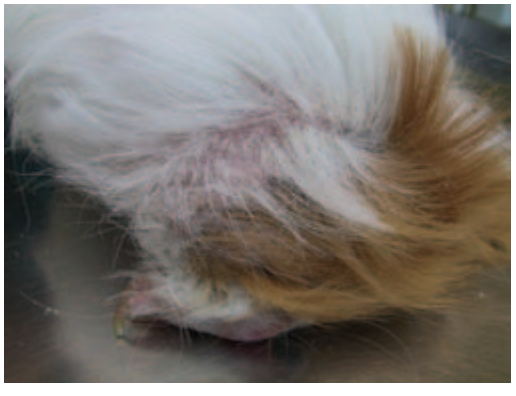

**Fig. 19.6** Chronic mange in the guinea pig. (Picture courtesy J. Fontaine.)

### *SARCOPTES SCABIEI*

Sarcoptic mange mites have been recorded in the guinea pig. However *Trixacarus caviae* is the much more common sarcoptiform mite affecting guinea pigs.

## *ATOPOMELIDAE*

### *CHIRODISCOIDES CAVIAE*

#### *Cause and pathogenesis*

*Chirodiscoides caviae* is the guinea pig fur mite. Most guinea pigs will tolerate large numbers of parasites without showing clinical signs. When the animal is in poor

condition or immunosuppressed then clinical disease can develop. Mites clasp hair rather than being found directly on the surface of the skin.

*Clinical signs*

Pruritus and self-inflicted hair loss are seen in heavy infestations in debilitated animals. Excessive self grooming associated with infestation may lead to self-trauma (Figures 19.7, 19.8) and ulcerative dermatitis (White *et al*. 2003). Mites tend to be concentrated in the groin and axilla.

*Diagnosis and therapy*

Mites identified on hair plucks (hair-clasping mites) rather than skin scrapings (Figure 19.9). Ivermectin given subcutaneously as for *T. caviae* is effective, and in colony situations has been used successfully as a spray (Hirsjarvi & Phyala 1995). Two applications of selamectin at 12 mg/kg two weeks apart also seem to be effective (White *et al*. 2003).

**Fig. 19.7** *Chirodiscoides caviae* infestation in a guinea pig. (Picture courtesy Z. Alha i dari.)

**Fig. 19.8** *Chirodiscoides caviae* infestation in a guinea pig (close up). (Picture courtesy Z. Alha i dari.)

**Fig. 19.9** *Chirodiscoides caviae.* (Picture courtesy J. D. Littlewood.)

## *MYOCOPTIDAE*

### *MYOCOPTES MUSCULINUS*

*Mycoptes musculinus*, the mouse fur mite, can occasionally infest guinea pigs. For more details on this parasite and clinical signs see p. 280, Chapter Twenty-two on mice under ***Mycoptidae***.

## *CHEYLETIELLIDAE*

### *CHEYLETIELLA PARASITOVORAX*

*Cheyletiella parasitovorax* is a common ectoparasite of the rabbit. It is most commonly seen on guinea pigs living with rabbits (Figure 19.10). When present it will

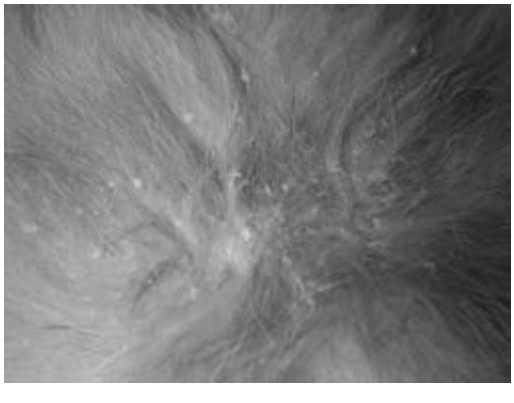

**Fig. 19.10** Cheyletiella in a guinea pig.

produce scaling and pruritus along the dorsum. Treatment is undertaken with ivermectin. For further details see pp. 300–301 in the section on this parasite in Chapter Twenty-three on rabbit diseases.

## *DEMODICIDAE*

### *DEMODEX CAVIAE*

*Cause and pathogenesis*

The lifecycle of this mite is unknown. Transmission usually occurs between nursing mothers and their offspring. Animals may carry mites without showing any evidence of clinical disease. Numbers of mites will rise in response to immunosuppression of the host.

*Clinical signs*

*Demodex caviae* is rarely recorded in the guinea pig. When it occurs it produces alopecia, erythema, papules and crusts, typically on the head, forelegs and trunk. Affected animals are usually immunosuppressed.

*Diagnosis and therapy*

Diagnosis is by deep skin scrapings. Treatment is with ivermectin, or with weekly amitraz dips (250 ppm) until four weeks after negative skin scrapes are obtained. Underlying disease or stressors should be investigated.

# INSECTS

## *LICE*

### *GLIRICOLA PORCELLI, GYROPUS OVALIS*

*Cause and pathogenesis*

The guinea pig suffers from two different biting lice: *Gliricola porcelli* (the Slender guinea-pig louse, Figure 19.11) and *Gyropus ovalis* (the Oval guinea-pig louse). They are commonly found in the coat of guinea pigs but do not produce clinical signs unless the pig is debilitated or there are large numbers of lice present.

*Clinical signs*

Heavy infestations lead to a roughened, dishevelled coat. Clinical signs include pruritus, diffuse scale and alopecia especially around the ears and dorsum.

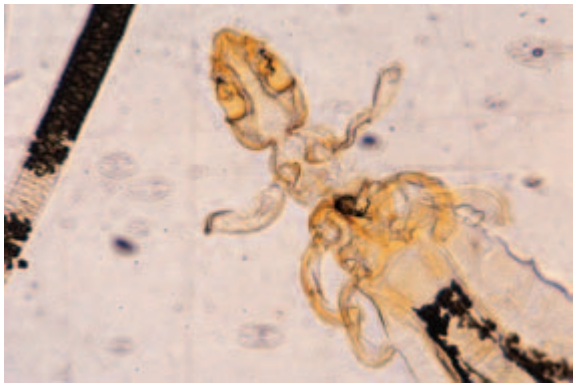

**Fig. 19.11** *Gliricola porcelli* guinea pig louse. (Picture courtesy J. D. Littlewood.)

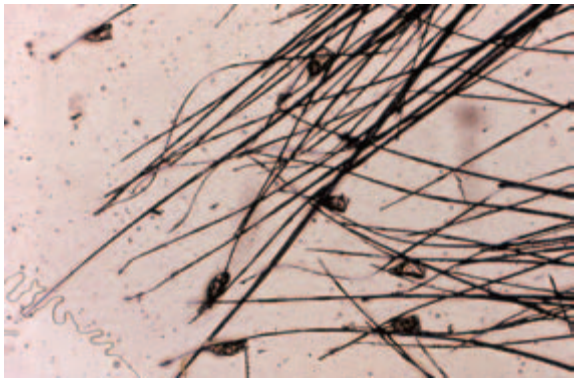

**Fig. 19.12** Lice eggs cemented to hairs in a guinea pig.

*Diagnosis and therapy*

Diagnosis is by visualisation of lice or eggs (nits) which are cemented to the hairs (Figure 19.12). Treatment is with systemic ivermectin as for mites.

## ENDOCRINE DISEASE

### *CYSTIC OVARIAN DISEASE*

*Cause and pathogenesis*

Cystic ovarian disease is extremely common in aged female guinea pigs. A 76% incidence in animals between one and a half and five years old has been reported (Shi *et al*. 2002). The aetiology is unknown (Neilsen *et al*. 2003), although oestrogenic substances in hay have been implicated. Prevalence and size of cysts increases with age, but reproductive status seems to have no influence on prevalence (Neilsen *et al*. 2003).

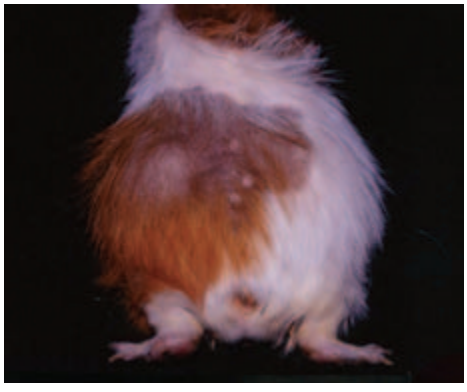

**Fig. 19.13** Alopecia in a guinea pig due to cystic ovaries.

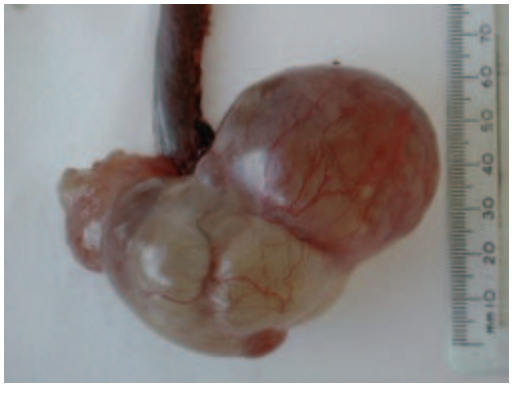

**Fig. 19.14** Cysts from a guinea pig post surgery. (Picture courtesy J. Henfrey.)

*Clinical signs*

Initially, cysts may be asymptomatic, however as they increase in size, non-pruritic alopecia develops over the back, ventrum and symmetrically over the flanks (Figure 19.13). Abdominal enlargement and infertility may also be evident. In one study of 43 female guinea pigs, 58% had cysts but only two (4.7%) had symmetrical alopecia. Diagnosis is based on history, abdominal palpation, radiography and ultrasonography. The cysts are usually bilateral and may be up to 10 cm in size (Figure 19.14) and painful on palpation. They are often associated with concurrent cystic endometrial hyperplasia, mucometra, endometritis and fibroleiomyomas.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs. Ovariohysterectomy is the treatment of choice, however the use of human chorionic gonadotrophin at 1000 USP i.m. every seven to ten days has been reported to resolve this condition temporarily (White *et al*. 2003). Percutaneous drainage of the cysts may also be possible under anaesthesia, but carries the risk of iatrogenic peritonitis, and recurrence is likely.

## *PREGNANCY-ASSOCIATED ALOPECIA*

*Cause and pathogenesis*

Hair loss due to telogen defluxion, associated with intensive breeding, is thought to be due to reduced anabolism of maternal skin, associated with foetal growth and reverses following parturition.

*Clinical signs*

It is common to see sows with non-pruritic bilateral flank alopecia during late pregnancy. Alopecia may worsen with each subsequent pregnancy.

*Diagnosis and therapy*

Diagnosis is based on reproductive history and ruling out other causes of alopecia such as dermatophytosis, barbering, vitamin deficiencies, ovarian cysts and ectoparasites. The condition will resolve itself providing the sow achieves good health after rearing babies.

# NUTRITIONAL DISEASE

## *HYPOVITAMINOSIS C*

Hypovitaminosis C is common in guinea pigs. They have an absolute dietary requirement of 10 mg/kg per day, rising to 30 mg/kg per day in pregnancy.

Hypovitaminosis C should be considered as a potential underlying factor in bacterial, fungal and ectoparasitic skin disease. Animals are most commonly reported with hypovitaminosis when fed commercial rabbit food or an out-dated guinea pig ration as the sole source of nutrition.

*Clinical signs*

Inadequate dietary levels of vitamin C or anorexia will rapidly lead to clinical signs. Important early cutaneous signs are roughened hair coat, and scaling of the pinnae. In more severe cases generalised scaling can occur with petechiae, ecchymoses and haematoma formation.

*Diagnosis and therapy*

Treatment involves correcting the diet and giving additional supplementation of 50–100 mg/kg per day until clinical signs resolve.

## *OTHER NUTRITIONAL DEFICIENCIES*

Other nutritional deficiencies are rare in pet guinea pigs, but have been produced experimentally. Protein deficiency causes a generalised alopecia. Fatty acid and pyridoxine deficiency result in alopecia, scaling and dermatitis (Scott *et al*. 2001).

# ENVIRONMENTAL AND BEHAVIOURAL CONDITIONS

## *STRESS*

*Cause and pathogenesis*

Stressed or ill guinea pigs frequently shed large amounts of hair. This is possibly due in part to an increased requirement for vitamin C (White *et al*. 2003).

*Clinical signs*

Increased generalised hair lost, no evidence of primary lesions.

*Diagnosis and therapy*

Diagnosis is made on the basis of history and clinical signs. Trichography of hair plucks reveals no evidence of trauma to the hair tips, suggesting hair loss is not self inflicted. Therapy is aimed at improvement of the animal's environment and increased vitamin C supplementation.

## *FUR CHEWING AND BARBERING*

*Cause and pathogenesis*

Fur chewing and barbering are associated with a lack of fibre in the diet, and with stress or overcrowding. Occasionally ear chewing will be seen.

*Clinical signs*

Hair loss is non-inflammatory. Hair appears as short stubbly areas when it has been chewed, the underlying skin is usually normal. When the hair loss is caused by chewing by another animal then the hair loss can be from any site, but the head, rump, perineum and prepuce are common sites especially when barbering occurs as a result of male dominant aggression. When hair loss is self induced there is relative sparing of the head, neck and anterior shoulders.

*Diagnosis and therapy*

Trichography of hairs reveals signs of self-inflicted trauma to the tips. Anogen bulbs remain well presented. Treatment should aim to eliminate any bullying or boredom. In some cases the introduction of long-stemmed hay will lead to decreased barbering. This would tend to suggest an underlying cause of boredom or need for fibre.

## *ULCERATIVE PODODERMATITIS (BUMBLE FOOT)*

*Cause and pathogenesis*

Ulcerative pododermatitis is common in guinea pigs. Obesity, poor hygiene, ageing, hypovitaminosis C, and wire flooring are all predisposing factors.

Affected animals are reluctant to walk and will vocalise frequently.

*Staphylococcus aureus* is generally isolated from lesions, although *Corynebacterium pyogenes* may also be found.

*Clinical signs*

The weight-bearing footpads are swollen and painful (Figure 19.15). Erythema, hyperkeratosis and ulceration are seen. In severe cases infection can extend to tendons and bone.

*Diagnosis and therapy*

Diagnosis is based on clinical signs, culture and radiography. Treatment involves topical antiseptics (e.g. silver sulfadiazine or mupirocin), systemic antibiotic therapy and wound dressing, plus addressing the underlying cause. Surgical debridement is rarely effective and should be avoided. However, treatment is often unsuccessful, and systemic amyloidosis often occurs due to the chronic

**Fig. 19.15** Pododermatitis in a guinea pig.

infection. Amputation of the hindlimb at the sacrohumeral joint can be considered in unilateral cases which are non-responsive to other treatments (Huerkamp *et al*. 1996).

# MISCELLANEOUS CONDITIONS

## *SCENT-GLAND IMPACTION*

*Cause and pathogenesis*

Guinea pigs possess sebaceous scent glands on the rump and perineal area. Oily secretions from these glands can lead to matting of the hair which is entirely normal. However in some male animals the oily secretions together with bedding and faeces can sometimes become trapped in the folds of the genital and perianal areas and can lead to skin disease.

*Clinical signs*

Malodorous dermatitis with secondary bacterial infection can develop in sites where the skin is damaged.

*Diagnosis and therapy*

Washing the area with a mild antiseptic shampoo will relieve the condition and help to prevent recurrence. An emollient protectant on the skin may prevent further episodes.

## *CHEILITIS*

*Cause and pathogenesis*

Cheilitis is thought to be associated with the feeding of acidic and abrasive foodstuffs. Secondary bacterial infection with *Staphylococcus* spp. is common, and a poxvirus has been associated with this condition in two animals (see p. 236 above).

*Clinical signs*

Ulceration and malodorous discharge around the mouth.

*Diagnosis and therapy*

Diagnosis is made on the basis of history and clinical signs. The author has had good success in treating cases of cheilitis by debriding and then packing the ulcers with a carmellose sodium, pectin and gelatin ointment (Orabase™, Squibb).

## *OTHERS*

### *CUTANEOUS HORNS*

Hyperkeratosis and cutaneous horns can develop on the footpads, especially in heavy guinea pigs and those housed in wire-bottomed cages. They can be removed through clipping or filing.

## *HEREDITARY ALOPECIA*

Hereditary alopecia is reported in the guinea pig and hairless animals are often used in laboratories. Some strains can also be hypothymic or athymic.

## NEOPLASIA

### *TRICHOFOLLICULOMAS*

*Cause and pathogenesis*

Trichofolliculomas are the most common cutaneous neoplasm in the guinea pig. They are benign and usually solitary (Figure 19.16), and generally occur on the dorsum.

*Clinical signs*

Nodular lesions contain a central pore through which keratinous or haemorrhagic material is discharged.

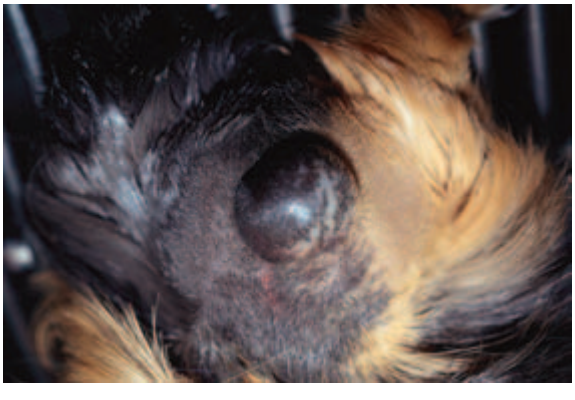

**Fig. 19.16** Trichofolliculoma in a guinea pig. (Picture courtesy Z. Alha i dari.)

*Diagnosis and therapy*

A fine-needle aspirate is unlikely to be diagnostic. Diagnosis and therapy is best achieved with an excisional biopsy.

## *OTHER TUMOURS*

Sebaceous adenoma, fibroma, fibrosarcoma, lipoma, liposarcoma, Schwannoma and lymphoma have all been reported in the guinea pig.

A vascular malformation, has been found in an adult female guinea pig, visible as an irregularly shaped, violaceous plaque on the flank, which ulcerated and bled repeatedly, eventually resulting in fatal haemorrhage (White *et al*. 2003).

*Formulary*

See p. 202, Chapter Sixteen on Chinchillas.

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Chapter 20

# Skin Diseases and Treatment of Hamsters

## BACTERIAL DISEASE

### *PYODERMA AND ABSCESSES*

*Cause and pathogenesis*

Infections in hamsters are usually secondary to bite wounds, trauma, abrasive or soiled bedding or mite infestation. Hamsters are prone to peri-odontal disease and dental caries which can lead to tooth root abscesses. *Staphylococcus aureus* is most commonly isolated, but *Streptococcus* spp., *Pasteurella pneumotropica*, *Actinomyces bovis* may also be found (Lipman & Foltz 1996). *Mycobacterium* spp. have been isolated from cutaneous granulomas (Harkness & Wagner 1995).

*Clinical signs*

Abscesses appear as areas of fluctuant swelling (Figure 20.1). Bite wounds tend to be around the head, tail and rump. Facial swelling ventral of cranial to the eye is commonly associated with tooth root problems. Pyoderma lesions are more superficial and present as areas of exudation, alopecia and excoriation.

*Diagnosis and therapy*

Diagnosis is based on clinical signs and impression smears or fine-needle aspirates of lesions. Treatment involves identifying and addressing any underlying cause, with the use of topical antiseptics, systemic antibiotics based on culture and sensitivity, Abscesses should be surgically drained or, preferably, excised.

## FUNGAL DISEASE

### *DERMATOPHYTOSIS*

*Cause and pathogenesis*

Dermatophytosis is rare in hamsters and is usually caused by *Trichophyton mentagrophytes. Microsporum canis* is occasionally found.

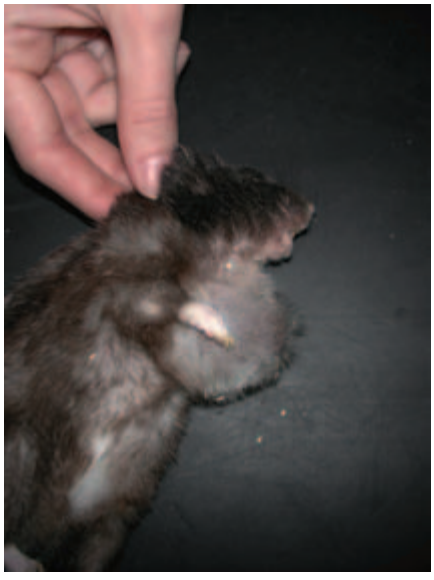

**Fig. 20.1** Abscess on the neck of a hamster.

*Clinical signs*

Infection may be asymptomatic or appear as dry circular skin lesions.

*Diagnosis and therapy*

Diagnosis is by microscopic examination of affected hairs and fungal culture. Treatment is by topical antifungals such as enilconazole, or systemic griseofulvin at 25–30 mg/kg p.o. sid for three to four weeks.

## VIRAL DISEASE

## *HAMSTER POLYOMAVIRUS*

Hamster polyomavirus HaPV (also referred to as hamster papovavirus) is associated with development of cutaneous epithelioma / tricho-epithelioma in Syrian hamsters, with up to 50% of animals affected in laboratory colonies (Percy & Barthold 1993; Parker *et al*. 1987). Infection may be subclinical, or associated with abdominal and thoracic lymphoma, or skin tumours. Epizootics can lead to high mortality. Incidence of skin tumours is higher when the infection is enzootic. Evidence of infectious skin disease due to HaPV has also been found in pet Syrian hamster colonies (Foster *et al*. 2002). The virus is highly contagious and spread via urine. It is very resistant in the environment. The incubation period is four to eighteen months. Trichepitheliomas are considered benign and do not metastasise, but large numbers may be debilitating.

# ECTOPARASITES

Important ectoparasites of hamsters are:

- Mites.
  - *Demodex criceti.*
  - *Demodex aurati.*
  - *Notoedres notoedres.*
  - *Notoedres cati.*
  - *Sarcoptes scabiei.*
  - *Trixacarus caviae.*
  - *Ornithonyssus bacoti.*
- Insects.
  - Fleas *(C. felis)*.

## *MITES*

### *DEMODICOSIS*

*Cause and pathogenesis*

Demodex is the most common ectoparasite of the hamster and is found in skin scrapings of normal animals (Ellis & Mori 2001; Collins 1987; Scott *et al*. 2001). Transmission occurs from mother to young during suckling. *Demodex criceti (*short and fat bodied) inhabits the keratin and pits of the epidermal surface and *Demodex aurati* (cigar shaped) inhabits the hair follicles. The lifecycle is thought to take ten to fifteen days. Predisposing factors for development of overt disease are concurrent disease, immunosuppression (especially neoplasia) or ageing.

*Clinical signs*

Clinical signs include moderate to severe alopecia, with dry scaly skin (Figures 20.2, 20.3) with erythema and small haemorrhages. Lesions are especially found on dorsal thorax and lumbar area but any area of the body can be involved. Pruritus is not usually present.

*Diagnosis and therapy*

Diagnosis is made on the basis of skin scrapes. Treatment is with amitraz (diluted to 100 ppm, once weekly until four weeks after skin scrapings are negative), or subcutaneous ivermectin. Weekly bathing with benzoyl peroxide shampoo will stimulate follicular flushing and reduce the mite load and can precede application of amitraz with a cotton bud to the affected areas (Burgmann 1991). It is important to be aware that the clinical disease is indicative of an underlying problem, and this should be addressed where possible.

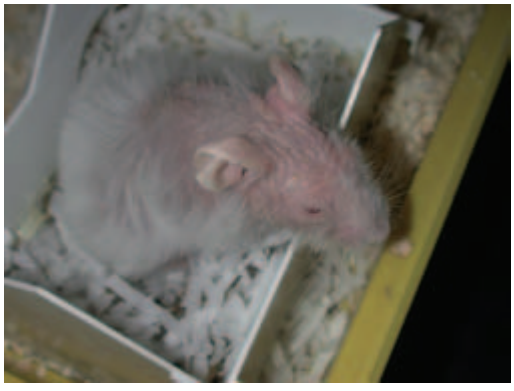

**Fig. 20.2** Demodicosis on the face of a hamster with concurrent hyperadrenocorticism.

**Fig. 20.3** Demodicosis on a hamster showing extensive alopecia and erythema.

## *NOTOEDRIC MANGE*

### *Cause and pathogenesis*

*Notoedres notoedres*, the hamster ear mite, and *Notoedres cati* the cat mange mite can on rare occasions infest the hamster.

### *Clinical signs*

Lesions are characterised by a thick, yellow crust with associated crust alopecia and erythema on the pinna, tail, genitalia, paws and muzzle.

### *Diagnosis and therapy*

Diagnosis is made by finding the mite in deep skin scrapings. The mite can be differentiated from Sarcoptes by its dorsal anus. Sarcoptes has a terminal anus.

Both forms of Notoedres can be treated with ivermectin. One study of 30 hamsters with notoedric mange compared the use of subcutaneous ivermectin at

0.4 mg/kg once a week with oral moxidectin at 0.4 mg/kg once a week and twice a week. All three regimes were equally effective in improving lesions, but after eight weeks only 60–70% of skin scrapings were negative (Beco *et al*. 2001). Topical selamectin may also be of use.

### *OTHER ECTOPARASITES*

Cat fleas (*C. felis felis*) can occasionally be found on hamsters (Collins 1987). Lice and ticks are not reported in this species.

## ENDOCRINE DISEASE

### *HYPERADRENOCORTICISM*

*Cause and pathogenesis*

Both primary hyperadrenocorticism (due to neoplastic changes of the adrenal cortex) and secondary hyperadrenocorticism (due to excess ACTH secretion secondary to a functional pituitary tumour) have been reported (Bauck & Lawrence 1984).

Iatrogenic Cushing's disease may also occur following glucocorticoid therapy. Primary hyperadrenocorticism is commonest in males and older animals, with adrenocortical adenoma being one of the most common reported benign neoplasms in the Syrian hamster. Interestingly, many of these develop in the zona glomerulosa where mineralocorticoids are primarily produced.

*Clinical signs*

Systemic signs include polydipsia (Figure 20.4), polyuria and polyphagia. Changes in behaviour may also be noted.

Cutaneous lesions include bilateral symmetrical alopecia of the flanks and lateral thigh area (Figure 20.5). The skin is thin, inelastic and comedones may be

**Fig. 20.4** Polydipsia in a hamster with hyperadrenocorticism.

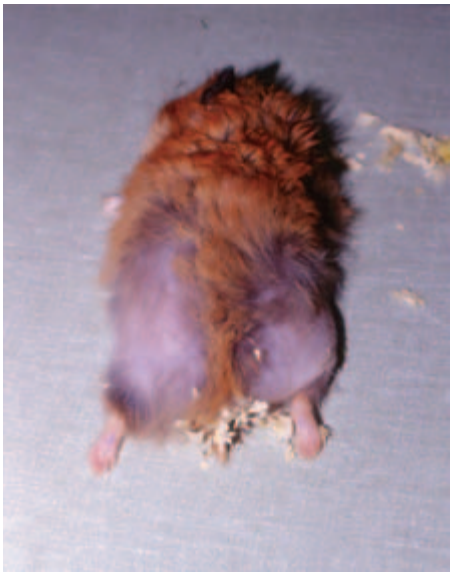

**Fig. 20.5** Bilateral alopecia in a hamster with hyperadrenocorticism.

**Fig. 20.6** Pot-bellied appearance with alopecia and comedones on the ventral abdomen of hamster with hyperadrenocorticism.

present (Figures 20.6, 20.7). Hamsters are usually pot-bellied. Demodicosis is commonly seen in an adult onset form secondary to hyperadrenocorticism.

*Diagnosis and therapy*

A tentative diagnosis may be made on the basis of history, clinical examination, elevated alkaline phosphatase, with or without elevated blood cortisol levels.

Practical problems occur due to the volume of blood needed to perform blood analysis due to the maximum volume which may be safely taken (10% of circulating blood volume, 1% of total bodyweight).

**Fig. 20.7** Close up of Fig. 20.6.

- Plasma cortisol levels may be as high as 110.4 nmol per litre (Bauck *et al*. 1984) although this is not a consistent finding. Normal cortisol values range from 13.8 to 27.6 nmol per litre in the hamster.
- Serum alkaline phosphatase may also be raised to >40 U per litre, normal values being 8–18 U per litre. Hamsters may secrete both cortisol and corticosterone, therefore diagnosis based on blood cortisol levels alone may not be accurate. It is important to note that cortisol or corticosterone levels elevate with stressors such as handling or transport. Levels reduce after 48 hours with acclimatisation to new surroundings.
- Ultrasonography of the adrenal gland may demonstrate enlargement or abnormalities.
- Dynamic function tests such as the ACTH stimulation test, low dose dexamethasone screening test or urine corticoid : creatinine ratio have not been well described in the hamster. The first two tests would be difficult on a practical level, due to the volume of blood required, and the stress involved in repeated sampling combined with the need for general anaesthesia to do so. The latter might be more useful, however normal values in hamsters would need to be ascertained, as well as test sensitivity and specificity. Differentiation of pituitary-dependent versus adrenal-dependent hyperadrenocorticism has not been determined *in vivo*.

Treatment of these cases may be difficult.
Medical therapy.

- Metyrapone (8 mg by mouth daily for one month) and was effective in one hamster, with complete regrowth of hair after twelve weeks. Metyrapone inhibits production of cortisol from its precursor.
- Mitotane (5 mg by mouth daily for one month) was reported to be unsuccessful, however this case failed to respond to metyrapone therapy as well.

Mitotane causes necrosis of the zona fasciculata and zona reticularis in the adrenal cortex. On post-mortem examination of this animal a chromophobe adenoma of the hypophysis and bilateral hyperplasia of the adrenal cortex were found.

Surgical removal of the affected adrenal gland via a flank laparotomy incision has been described.

## MISCELLANEOUS CONDITIONS

### *NORMAL SCENT GLANDS*

Syrian hamsters possess sebaceous scent glands over the hip area (hip glands, flank glands), which are circumscribed, slightly raised, darkly pigmented structures (Figures 20.8, 20.9). The glands are more prominent in males, and the hair overlying them often becomes thinner with ageing. When sexually aroused, the overlying hair becomes matted with secretions. Owners commonly mistake these for abnormalities.

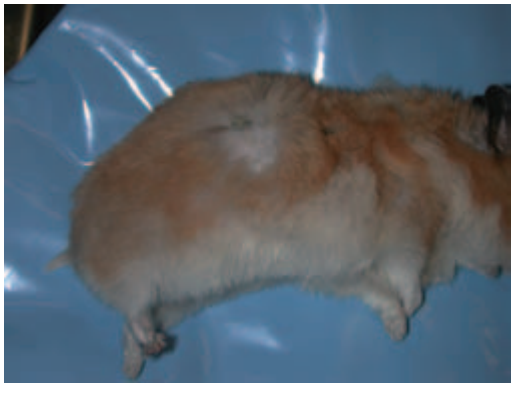

**Fig. 20.8** Normal scent gland on the flank of a hamster.

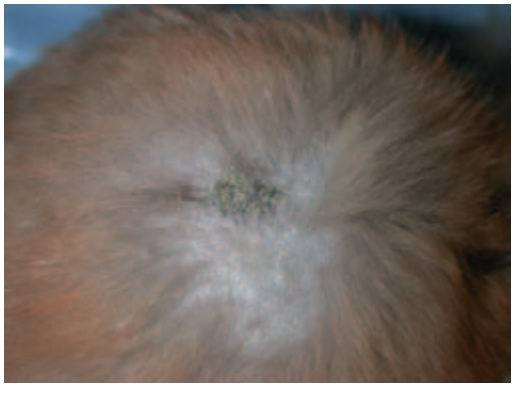

**Fig. 20.9** Close-up of scent gland.

## *INFLAMMATION OF THE SCENT GLANDS*

*Cause and pathogenesis*

Inflammation of the glands can occur occasionally.

*Clinical signs*

Hamsters present with swelling and crusting over the glands.

*Diagnosis and therapy*

Diagnosis may be made on the basis of history and clinical signs. The fur should be clipped over the area and topical antiseptics applied. Castration will help to decrease secretory activity (Lipman & Foltz 1996). However, castration is associated with shorter life span and a higher incidence of benign adrenal neoplasms and nodular hyperplasia (Collins 1987).

## *CONTACT DERMATITIS*

*Cause and pathogenesis*

Contact dermatitis attributed to cedar or pine shavings used for bedding has been reported.

*Clinical signs*

Affected animals showed signs of swelling and pruritus of the face and paws (Meshorer 1976).

*Diagnosis and therapy*

Diagnosis can be made on the basis of history and clinical signs. Therapy should consist of removal of the contact allergen / irrirant and symptomatic therapy, if necessary with topical glucocorticoid products.

## *BEDDING-ASSOCIATED DERMATITIS*

*Cause and pathogenesis*

Dermatitis of the digits has been reported in hamsters housed on wood shavings.

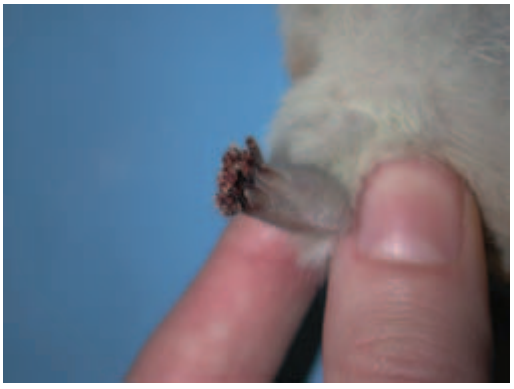

**Fig. 20.10** Dermatitis on the digits in a hamster.

*Clinical signs*

Degeneration and atrophy of the digits with a granulomatous inflammatory response is usually observed (Figure 20.10) (Meshorer 1976).

*Diagnosis and therapy*

Diagnosis can be made on the basis of history and clinical signs. In reported cases shavings were observed histologically in the dermis and subcutis. Therapy should be aimed at removing the underlying cause and using symptomatic topical treatment.

## NEOPLASIA

Melanomas and melanocytomas are the most frequently reported cutaneous neoplasm (Collins 1987, Scott *et al*. 2001). A higher incidence occurs in males. Melanomas may be melanotic (bluish–black) or amelanotic (grey–white).

### *EPITHELIOTROPHIC LYMPHOMA (MYCOSIS FUNGOIDES)*

*Cause and pathogenesis*

Epitheliotrophic lymphoma (mycosis fungoides) is the second most common cutaneous neoplasm in hamsters (Harvey *et al*. 1992). Hamster polyomavirus (papovavirus) is thought to be the cause of transmissible lymphoma and it has been identified as a cause of cutaneous epitheliomas in hamsters (see p. 252 above under Viral disease).

*Clinical signs*

Usually occurs in aged animals. Cutaneous signs include alopecia, pruritus and flaky skin (Figure 20.11) to cutaneous plaques and nodules, which may become ulcerated and crusted (Figures 20.12, 20.13, 20.14). Large numbers of demodex mites are often found on skin scrapings of affected animals, which may confuse the diagnosis.

*Diagnosis and therapy*

A tentative diagnosis can be made by history and clinical signs. Impression smears from ulcerated lesions or from scrapings from nodules / plaques will often show signs of a lymphocytic infiltrate with cells showing criteria of malignancy. Definitive diagnosis is by biopsy. Immunohistochemistry has demonstrated that the neoplastic cells are T lymphocytes (Harvey *et al*. 1992). Treatment is rarely successful but palliative therapy can be undertaken with topical glucocorticoid creams. Euthanasia is recommended.

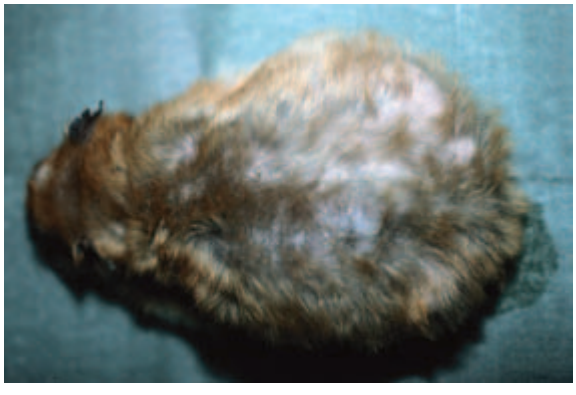

**Fig. 20.11** Diffuse hair loss and scaling in a hamster with lymphoma. (Picture courtesy Z. Alha i dari.)

**Fig. 20.12** Ulcerated nodules on the neck of a hamster with lymphoma.

**Fig. 20.13** Ulcerated nodules on the feet of a hamster with lymphoma.

**Fig. 20.14** Mycosis fungoides in a hamster.

## *OTHER NEOPLASMS*

Other neoplastic lesions reported in the hamster include reticulum cell carcinoma, plasmacytoma, sarcoma, fibrosarcoma, fibroma, squamous cell carcinoma, basal cell carcinoma, and papilloma (Figure 20.15).

*Formulary*

See p. 202, Chapter Sixteen on Chinchillas.

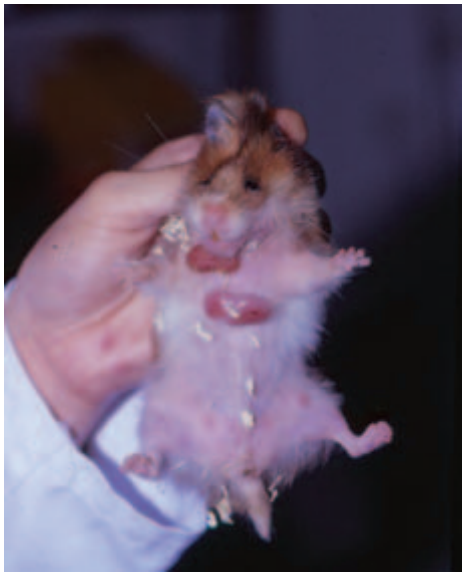

**Fig. 20.15** Skin masses on the ventrum of a hamster.

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Chapter 21

# Skin Diseases and Treatment of Hedgehogs

Two species of hedgehog are encountered in veterinary practice. The wild European hedgehog (*Erinaceus europaeus*) is commonly presented as a wildlife casualty (Figure 21.1). The African Pygmy hedgehog (*Atelerix albiventris*) and also the Egyptian long-eared hedgehog (*Hemiechinus auritus auritus*) (Figure 21.2) are kept as a pets.

## BACTERIAL DISEASE

### *STAPHYLOCOCCAL SKIN DISEASE*

*Cause and pathogenesis*

Bacterial skin infection is generally due to *Staphylococcus* spp., especially *S. aureus*. When abscesses occur *Staphylocoocus*, *E. Coli* and *Pseudomonas* are commonly isolated.

*Clinical signs*

Bacterial infection is usually secondary to wounds, but can cause a primary exfoliative dermatitis on the ventrum (Robinson & Routh 1999; Bexton & Robinson 2003). Abscesses are also common (Bexton & Robinson 2003; Larson & Carpenter 1999).

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs and cytology of lesions.

Treatment involves cleaning and debridement of any associated wounds and appropriate systemic antibiosis. Abscesses are treated by drainage and antibiosis.

## FUNGAL DISEASE

### *DERMATOPHYTOSIS*

*Cause and pathogenesis*

Dermatophytosis in the hedgehog is usually caused by *Trichophyton erinacei* (*T. mentagrophytes* var. *erinacei*), although occasionally by *Microsporum canis* and

**Fig. 21.1** The European hedgehog is most commonly presented as a wildlife casualty to veterinary surgeons.

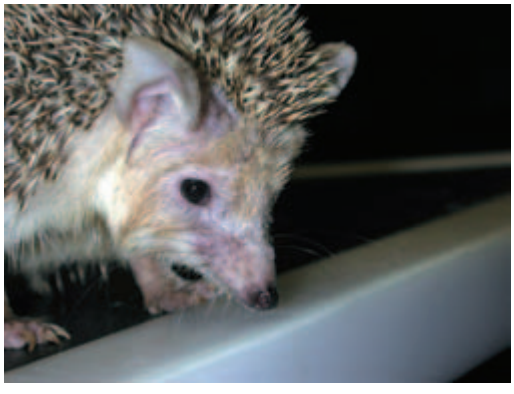

**Fig. 21.2** The Egyptian long-eared hedgehog is becoming more popular as a pet.

*M. gypseum*. In one study of 74 European hedgehogs (Keymer *et al*. 1991), only one was found to have dermatophytosis, but up to 25% of hedgehogs can carry it (Morris & English 1969), although disease may be subclinical. Co-infection with the mite *Caparinia tripilis* is common (see p. 267 below), or there may also be a bacterial pyoderma.

Transmission is probably by direct contact from mother to young or during fights or courtship, which may explain why most lesions are on the head (Reeve 1994). Infection may also be obtained from environmental spores, which can remain viable in dry nests for up to a year (Morris & English 1969). Caparinia has been implicated in the transmission of *T. erinacei* as the fungus can be recovered from mite faeces (Reeve 1994), but one study showed no evidence of Caparinia acting as vector.

Incidence of ringworm is higher in dense populations, and this is probably related to increased opportunities for contact and nest-sharing (Morris & English 1969).

*Clinical signs*

Clinical signs are crusting lesions around the base of spines and hair and spine loss. Lesions are typically around the head. Bleeding may occur if the scabs are removed. Thickening of the pinnae may also occur in chronic infections. Pruritus and debility is generally absent, even with severe infections, with affected animals gaining weight normally (Morris & English 1969). *T. erinacei* is zoonotic, causing a rapidly spreading, highly pruritic vesicular, then scaly, lesion (Bexton & Robinson 2003).

*Diagnosis and therapy*

Definitive diagnosis is by fungal culture. Treatment regimes include topical natamycin or enilconazole (Robinson & Routh 1999), oral griseofulvin at 50 mg/kg once daily for two to three weeks or oral ketoconazole at 10 mg/kg once daily for six to eight weeks (Ellis & Mori 2001). The necessity for treatment is debatable, given the low pathogenicity of infection and its widespread nature, but the zoonotic potential may provide justification (Bexton & Robinson 2003). Recovery and spine regrowth can take a very long time.

## VIRAL DISEASE

### *FOOT-AND-MOUTH VIRUS*

*Cause and pathogenesis*

Hedgehogs are susceptible to foot-and-mouth disease (FMD) and can act as carriers (Thomson *et al*. 2001; Isenbugel & Baumgartner 1993). It is unclear whether or not they can act as a reservoir for the disease. Early studies indicating latent infection should be viewed with caution as virological techniques used at the time were unreliable and there is little evidence that hedgehogs have been involved in the spread of FMD in Europe or Africa.

*Clinical signs*

Affected animals show vesicles, erythema and swelling on the haired parts of the body, the feet, lips and perineum. Anorexia, sneezing and hypersalivation are also prominent clinical signs.

*Diagnosis and therapy*

Diagnosis is made on the presence of clinical signs and viral isolation. Treatment is not normally undertaken.

## ECTOPARASITES

Ectoparasites of importance in the hedgehog include.

*Mites*

- *Caparinia tripilis.*
- *Sarcoptes* spp.
- *Otodectes* spp.
- *Notoedres* spp.
- *Chorioptes* spp.
- *Demodex erinacei.*
- *Trombicula autumnalis.*

*Ticks*

- *Ixodes hexagonus, Ixodes ricinus, Ixodes trianguliceps.*

*Insects*

- Flies – *Lucilia, Calliphora.*
- Fleas – *Archaeopsylla erinacei.*

## *MITES*

### *CAPARINIA TRIPILIS*

The commonest mite found on wild European hedgehogs is *Caparinia tripilis*. Up to 40% of hedgehogs may be infested (Bexton & Robinson 2003), and synergistic infection with dermatophytes often occurs.

*Clinical signs*

*Caparinia* mites are just visible to the naked eye as white motile powdery deposits, usually around the eyes and ears and on the cheeks. More generalised scaling lesions can also occur with hair and spine loss and pruritus. In pet African pygmy hedgehogs in the USA, macroscopically visible mites presumed to be *Caparinia tripilis* are commonly seen in practice (Lightfoot 1999).

*Diagnosis and therapy*

Diagnosis is easily made by examination of the skin with a hand lens or microscopic examination of plucked hairs.

## *SARCOPTES* spp.

*Cause and pathogenesis*

*Sarcoptes* spp. is occasionally seen in young hedgehogs.

*Clinical signs*

This parasite can cause generalised erythema and alopecia which may be fatal.

*Diagnosis and therapy*

Diagnosis is made by identification of mites on scrapings from the skin. Therapy can be undertaken using ivermectin.

## *DEMODEX ERINACEI*

*Cause and pathogenesis*

*D. erinacei* is host specific, the mites live in sebaceous glands. Most hedgehogs are asymptomatic carriers.

*Clinical signs*

When heavy infestations are present demodicosis can cause raised papules and crusty skin lesions.

*Diagnosis and therapy*

Characteristic 'cigar'-shaped mites are found on deep skin scrapes. Amitraz® dips and systemic ivermectin are suggested treatments (Robinson & Routh 1999; Bexton & Robinson 2003; Ellis & Mori 2001)

## *OTHER MITES*

*Otodectes cyanotis* can cause otitis externa and *Notoedres cati* can cause crusting lesions around the head and ears. These mites are occasionally found on hedgehogs, most probably from contact with cats (Bexton & Robinson 2003).

*Chorioptes* spp. can be found in African pygmy hedgehogs in the USA and can cause crusting, flaking and spine loss (Larson & Carpenter 1999; Lightfoot 1999).

Numerous other mite species have been recorded from wild European and African hedgehogs, including the mesostigmatid mites *Eulaelaps stabularis*, *Haemogamasus pontiger*, *H. nidi*, *Androlaelaps fahrenholzi* and *Caparinia erinacei* and *Notoedres oudemansi* (Reeve 1994).

*Trombicula autumnalis*, the harvest mite, is also found in the axillae, pinnae and on the ventrum and feet, but is probably of little clinical significance (Bexton & Robinson 2003).

## *TICKS*

*Cause and pathogenesis*

Ticks are common ectoparasites of hedgehogs. *Ixodes hexagonus* is most commonly found (Reeve 1994), but the sheep ticks, *Ixodes ricinus* and *Ixodes trianguliceps* and many other species have also been recorded (Smith 1968).

*Clinical signs*

Ticks can usually be identified around the ears, hindlimbs, flanks and ano-genital region (Figures 21.3, 21.4) (Bexton & Robinson 2003; Reeve 1994).

*Diagnosis and therapy*

Diagnosis can be made on the basis of clinical signs. Therapy can be undertaken with Fipronil® (Figure 21.5).

**Fig. 21.3** Ixodes ticks on a hedgehog.

**Fig. 21.4** Ixodes ticks on a hedgehog.

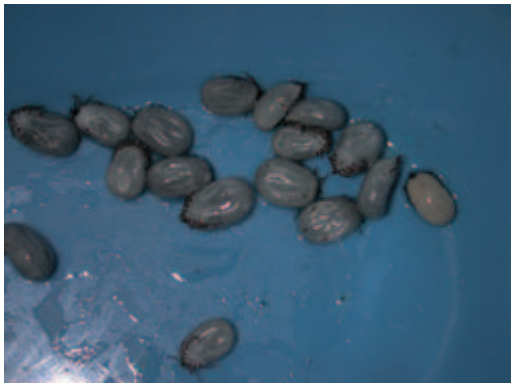

**Fig. 21.5** Ixodes ticks after removal.

## *FLEAS*

### *Cause and pathogenesis*

Fleas are ubiquitous on European hedgehogs, with counts of between 100 and 1100 reported form individual animals (Reeve 1994). Heavy burdens are generally found in debilitated animals. The hedgehog flea is *Archaeopsylla erinacei* (Figures 21.6, 21.7). It can tolerate the hedgehog's hibernation and reproduces only in the nests of breeding females. Hedgehog fleas cannot breed or persist on other species of mammal (Reeve 1994).

### *Clinical signs*

In general, fleas cause few clinical problems and minimal irritation.

**Fig. 21.6** A*chaeopsylla erinacei* (the hedgehog flea). (Picture courtesy J. D. Littlewood.)

**Fig. 21.7** Close up of head of hedgehog flea. (Picture courtesy J. D. Littlewood.)

*Diagnosis and therapy*

Diagnosis is made on the basis of entrapment and identification of fleas. Fipronil® appears to be effective in hedgehogs (Bexton & Robinson 1999), or pyrethrin products may be used. Only heavy infestations need to be treated.

## *LICE*

Lice have never been found or reported on any hedgehog species (Reeve 1994).

## *FLIES*

### *MYIASIS*

*Cause and pathogenesis*

Flystrike is common in wild European hedgehogs. Both primary strike and secondary wound infestation occur, caused by *Lucilia* and *Calliphora* spp. (Robinson & Routh 1999). In native African hedgehogs skin lesions from mange or bite wounds can lead to blowfly maggot infestations from *Hemipyrella fernandica*.

*Clinical signs*

Maggots are usually identified in wounds and also as primary infestation due to such problems as the hedgehog soiling.

*Diagnosis and therapy*

Maggots and eggs should be physically removed, paying special attention to eyes and ears. Eyes can be filled with a viscous eye ointment to smother any remaining

maggots, and insecticidal ear drops can be placed in the ears (Bexton & Robinson 2003). Systemic ivermectin should be given, plus supportive care consisting of fluid therapy, antibiotic cover and non-steroidal anti-inflammatory drugs.

## ENDOCRINE DISEASE

### *HYPERADRENOCORTICISM*

*Cause and pathogenesis*

Hyperadrenocorticism due to an adrenal tumour has been diagnosed in one African pygmy hedgehog.

*Clinical signs*

Clinical signs were alopecia, pendulous abdomen, polyphagia, polyuria and polydipsia.

*Diagnosis and therapy*

Diagnosis was confirmed using a low-dose dexamethasone suppression test (Johnson-Delaney 2002).

## NUTRITIONAL DISEASE

Poor spine and coat condition can be related to a poor diet, nutritional hyperparathyroidism, hypervitaminosis A and D, and zinc deficiency (Bexton & Robinson 2003, Ellis & Mori 2001).

## MISCELLANEOUS CONDITIONS

### *WOUNDS AND BURNS*

*Cause and pathogenesis*

Wild hedgehogs commonly sustain wounds from various sources – entrapment in netting, barbed wire, plastic, tin cans, strimmers and mowers, dogs and road accidents. Wild European hedgehogs may nest in unlit bonfires and may sustain serious burns.

*Clinical signs / diagnosis and therapy*

Fresh wounds can be cleaned and sutured. In-dwelling drains may be indicated if contaminated (Robinson & Routh 1999). Older contaminated wounds should be

cleaned and debrided, checked for maggots or fly eggs, and allowed to heal by granulation. Antibiotic cover is indicated as cellulites and abscessation are common.

Treatment of burns is similar for other species, with fluid therapy, analgesia, antibiosis, with cleaning and dressing of the affected area. Animals with severe burns should be euthanased.

## *ALLERGIC DERMATITIS*

*Cause and pathogenesis*

Two cases of suspected allergic dermatitis have been reported in African pygmy hedgehogs (Ellis & Mori 2001; Lightfoot 1999).

*Clinical signs*

Case one presented with severe dermatitis of the face, axilla and groin. Case two presented with progressive alopecia, pruritus and facial swelling.

*Diagnosis and therapy*

In case one histopathology indicted an allergic response. Glucocorticoid therapy resolved the condition but the allergen was not determined.

In case two histopathology of skin biopsies revealed mild diffuse lymphocytic plasmacytic lichenoid dermatitis, focal epithelial dysplasia and diffuse subacute dermatitis. Positive allergic reactions were obtained to 22 plant and food allergens using RAST and ELISA tests on serum – however, no studies have evaluated the specificity of allergy testing in hedgehogs. This animal improved on a commercial, feline, low-allergen diet and diphenhydramine at 1 mg/kg p.o. bid, and improved further on prednisolone at 0.5 mg/kg p.o. sid reducing to 0.25 mg/kg p.o. once every other day. There are no reports of intradermal skin testing in this species.

## NEOPLASIA

Skin neoplasia has not been reported in the European hedgehog, but is common in African pygmy hedgehogs over three years of age (Ellis & Mori 2001). Papilloma, squamous cell carcinoma, lymphosarcoma and sebaceous gland carcinoma have been described. Squamous cell carcinoma affecting all four feet of an Indian hedgehog (*Hemiechinus* spp.), and papilloma-type growths on the face, head and feet of an Eastern European (white-breasted) hedgehog (*Erinaceus concolor*) have also been reported (Ellis & Mori 2001).

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# Chapter 22

# Skin Diseases and Treatment of Mice

## BACTERIAL DISEASE

### *STAPHYLOCOCCAL AND STREPTOCOCCAL INFECTIONS*

*Cause and pathogenesis*

Both *Staphylococcus aureus* and group G Streptococcus have been identified as causing skin disease in mice. (See Table 22.1.)

**Table 22.1** Clinical signs of bacterial disease in mice.

| Organism | Syndrome | Clinical signs |
|---|---|---|
| *Staphylococcus aureus* | Spontaneous ulcerative dermatitis. | Dermal ulceration and self-trauma, typically around the head, face and across the shoulders. |
| *S. aureus* | Cutaneous abscess in athymic nude mice and other strains of immunocompromised mice. | Can develop peri-orbital, lacrimal gland and cutaneous abscesses. |
| *Staphylococcus aureus* and Streptococcus spp. | Self-trauma or fight wounds. | Dermatitis and abscesses. |
| Group G Streptococcus | Spontaneous ulcerative dermatitis. | Dermal ulceration and self-trauma, typically around the head, face and across the shoulders. |
| Group G Streptococcus | Necrotising ulcerative gangrenous dermatitis. | Ulcerative gangrenous lesions found on the dorsum of nude mice occasionally posterior paralysis is also seen. |
| *Streptococcus moniliformis* | | Oedema and cyanosis of the extremities (Ellis & Mori 2001). |

*Diagnosis and therapy*

See section below under Other Bacteria.

## *OTHER BACTERIA*

*Pasteurella pneumotropica*, *Actinobacillus* spp., *Actinomyces* spp., and *Klebsiella* spp. can be involved in the infection of abscesses and fight wounds.

*Corynebacterium kutscheri* can cause septic emboli, which can cause dermal vessel infarction, leading to skin necrosis and ulceration.

*Corynebacterium pseudiphtheriticum* causes a dermatitis in athymic nude mice, characterised by severe orthokeratotic hyperkeratosis. Mortality is high in suckling mice (Ellis & Mori 2001).

*Mycobacterium chelonae* has been reported as causing granulomatous inflammation of the tail in immunocompromised mice (Mahler & Jelinek 2000).

*Diagnosis and therapy*

Diagnosis should be made on the basis of clinical signs, cytology of lesions and culture. Where anaerobic or mycobacterial infection is suspected then the laboratory should be made aware of this.

Treatment of all bacterial dermatoses includes elimination of predisposing factors, surgical drainage or preferably excision of abscesses, topical antiseptics such as chlorhexidine 0.5–1.0% and systemic antibiosis based on culture and sensitivity testing.

# VIRAL DISEASE

## *MOUSEPOX (ECTROMELIA)*

*Cause and pathogenesis*

This orthopoxvirus has not been reported in pet mice. It occurs in laboratory colonies. Ectromelia means absence or imperfection of limb. The disease is highly infectious and transmission is via aerosol, skin abrasions, contact with skin debris and ingestion of contaminated faeces. The virus is relatively stable in the environment and fomite transmission is also possible.

*Clinical signs*

Mousepox infections may be asymptomatic, latent, acute, subacute, or chronic, and outcome of infection varies widely depending on strain of mouse.

*Acute form*: high morbidity and mortality and clinical signs include hunched posture, rough hair coat, conjunctivitis, swelling of face and extremities and diarrhoea.

*Subacute and chronic forms*: exhibit the cutaneous lesions of a generalised papular rash leading to swelling, ulceration and amputation of limbs and tail, with variable mortality.

*Diagnosis and therapy*

Diagnosis is by clinical signs, demonstration of intracytoplasmic eosinophilic inclusion bodies (Marchal bodies) in epithelial skin, intestinal or pancreatic cells, electron microscopy, fluorescent antibody tests or serology (PCR).

There is no treatment but vaccination with a live vaccinia virus strain via scarification of the tail base may limit outbreaks in closed colonies.

## *REOVIRUS TYPE 3*

Reovirus infection of suckling mice causes severe systemic illness and an oily hair coat. If animals survive they are alopecic.

# FUNGAL DISEASE

## *DERMATOPHYTOSIS*

*Cause and pathogenesis*

Dermatophytes are common in mice, but infections are often asymptomatic. *Trichophyton mentagrophytes* is the most common cause of dermatophytosis. Sixty per cent of pet shop mice with no lesions have been reported to have *T. mentagrophytes* present (Collins 1987).

*Clinical signs*

Infection can be asymptomatic or cause hair loss and well-demarcated crusty lesions with erythema and scaling (Figure 22.1). Clinical disease is often associated with a concurrent stressor.

*Diagnosis and therapy*

Diagnosis is by microscopic examination of skin scrapings in 10% KOH or fungal culture. Treatment is with oral griseofulvin or enilconazole wash twice a week until two cultures are negative. For groups of affected mice, environmental treatment with a spray of enilconazole solution of 50 mg per square metre, twice weekly for twenty weeks can be used. Owners should be alerted to the zoonotic potential of infection.

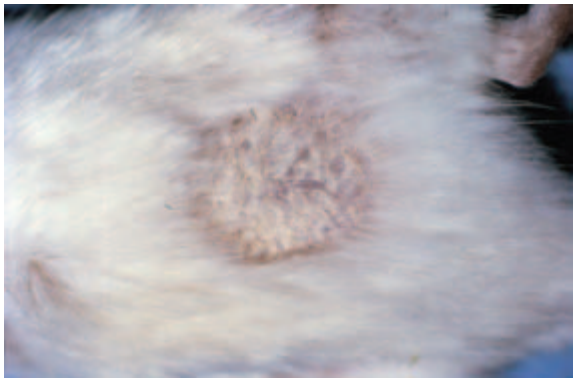

**Fig. 22.1** Dermatophytosis in a mouse.

## ECTOPARASITES

Important ectoparasites of mice include:

- **Mites**.
  - Myobidae – fur mites *Myobia musculi, Radfordia affinis*.
  - Myocoptidae – mange mites *Myocoptes musculinus, Trichoecius rombousti* (rare).
  - Dermanyssidae – *Liponyssoides sanguineus, Dermanyssus gallinae*.
  - Psorergatidae – *Psorergates muricola.*
  - Demodicidae – *Demodex musculi.*
- **Insects**.
  - Lice.
    - *Polyplax serrata.*
  - Fleas.
    - *Xenopsylla* spp.
    - *Nosopsylla* spp.

## *MITES*

### *MYOBIIDAE – FUR MITES*

### *Myobia musculi*

*Cause and pathogenesis*

Occurs at the base of the hairs throughout the fur especially on the head and underside of neck. The mite feeds on extracellular body tissue. Transmission is by direct contact. Often occurs with *Myocoptes musculinus*. Some inbred strains of mice are believed to have an allergic response to Myobia (Weisbroth 1982), and

**Fig. 22.2** *Myobia musculi* infestation in a mouse. (Picture courtesy J. Fontaine.)

exposure to very few mites will trigger hypersensitivity and severe self trauma. Myobia has a 23-day lifecycle and eggs hatch in eight days.

*Clinical signs*

Low numbers rarely cause clinical signs. Large infestations or in immunosuppressed animals alopecia and ulceration is seen due to self-inflicted trauma (Figure 22.2).

*Diagnosis and therapy*

Diagnosis is by identification of the mites on tape strippings and skin scrapes. Mite has an oval body and legs have prominent claws. Treatment is with ivermectin at 200–400 µg/kg sc every ten to fourteen days for two to three doses. Large colonies of mice can be treated empirically by spraying the cage and animals weekly for three weeks with a spray made up of one part ivermectin 1% to ten parts tap water, delivering approximately 1–2 ml per average cage.

Alternatively, oral treatment with ivermectin 0.08%, sheep drench (Oramec™, Merial Animal Health Ltd) at 4 ml (3.2 mg) per litre of drinking water for one week on, one week off, one week on, is also effective (Conole *et al*. 2003). It is important to note that in some laboratory strains of mice ivermectin is toxic, probably due to the drug crossing the blood brain barrier as seen in certain breeds of dog.

Topical selamectin can also be used – one study found 100% efficacy at 90 days of both 12 mg/kg and 24 mg/kg given on day 0 and day 30 (Bourdeau *et al*. 2003).

## *Radfordia affinis*

Same family as Myobia is similar in its appearance and clinical signs. The lifecycle of *Radfordia* is unknown but believed to be 21–23 days.

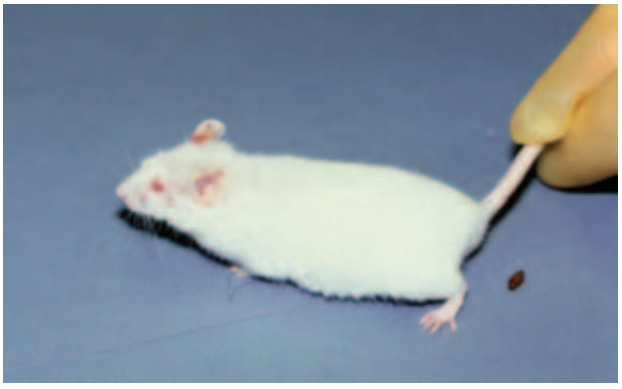

**Fig. 22.3** Mite infestation on the head of a mouse.

## *MYOCOPTIDAE – MANGE MITES*

### *Myocoptes musculinus*

*Cause and pathogenesis*

Mite is found clinging to hairs rather than free moving on the surface of the skin (Figure 22.3). The mite is thought to feed on superficial epidermal tissue. The life-cycle is thought to be eight to fourteen days and eggs hatch in five days. Often found in association with *Myobia musculi.* Transmission of mites is through direct contact.

*Clinical signs*

Healthy animals appear to tolerate large numbers of mite. However pregnant animals and immunosuppressed mice will show clinical signs. They usually present with a dull, grey coat. Pruritus, leading to alopecia and ulceration caused by self trauma, is seen to varying degrees. Lesions are usually found around the head in the inguinal region and base of tail. Myocoptes tends to produce less severe ulceration than that caused by fur mites.

*Diagnosis and therapy*

Diagnosis is by skin scrapings into liquid paraffin or potassium hydroxide, or by direct examination of the coat under a hand lens or dissecting microscope. Therapy as above.

### *Trichoecius rombousti*

This mite is commonly seen in association with the other mange and fur mites. Its effect on the host is unclear. Lesions similar to those produced by *Myocoptes musculinus* have been recorded.

## *DERMANYSSIDAE*

### *Liponyssoides sanguineus*

*Cause and pathogenesis*

*Liponyssoides sanguineus* is the house-mouse mite and has been recorded from both mice and rats. It is a blood-feeding parasite which spends most of its time off the host in the bedding. The precise impact of infestation is unknown.

*Clinical signs*

The feeding activity of large numbers of mites is thought to lead to poor coat condition, anaemia, debilitation and eventually death.

*Diagnosis and therapy*

Diagnosis is made by identification of the mite on the host and in the environment. The mite is red–brown during and after feeding activity. Treatment should be aimed at the environment.

## *PSORERGATIDAE*

### *Psorergates muricola*

*Cause and pathogenesis*

*Psorergates muricola* is a burrowing mite commonly found in cavities in the *stratum corneum*. It produces lesions on the ears of affected mice.

Transmission is by direct contact.

*Clinical signs*

Infestations cause the formation of small white nodules especially on the ear pinnae but can also occur on the body.

*Diagnosis and therapy*

As above.

## *DEMODICIDAE*

### *Demodex musculi*

*Cause and pathogenesis*

Follicular mite rarely found in mice. Mites may be present without showing clinical signs. Transmission is thought to occur by direct contact between mother and suckling offspring in the first few days of life.

*Clinical signs*

Infestation can cause localised alopecia often with secondary infection.

*Diagnosis and therapy*

Diagnosis is by skin scrapes into liquid paraffin or potassium hydroxide. Therapy is with ivermectin, see above.

## *OTHER LESS IMPORTANT MITES*

The ear mite *Notoedres muris* is found occasionally. Other mites more rarely found are *Sarcoptes scabiei, Haemogamasus pontiger, Laelaps echidnina* and *Ornithonyssus bacoti* (tropical rat mite).

## *LICE*

### *Polyplax serrata*

*Cause and pathogenesis*

*Polyplax* serrata in the sucking louse of the mouse (Figure 22.4). It is rarely identified on mice. It has zoonotic importance as a possible vector of tularaemia (*Pasteurella* (*Francisella*) *tularensis*). The lifecycle takes thirteen days and eggs hatch in five to six days.

*Clinical signs*

It can cause pruritus, restlessness, dermatitis and anaemia particularly in immunocompromised animals.

*Diagnosis and therapy*

Diagnosis is by clinical signs, identification of louse or eggs (Figure 22.5). See above under mites.

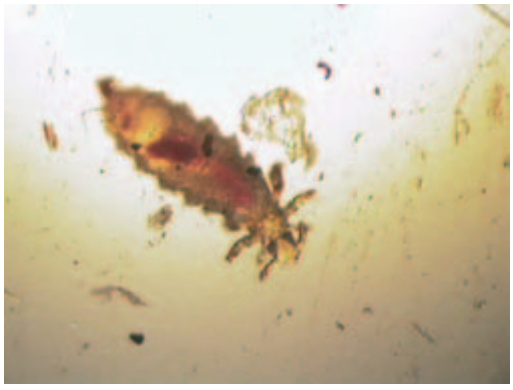

**Fig. 22.4** The sucking louse of the mouse, *Polyplax serrata*.

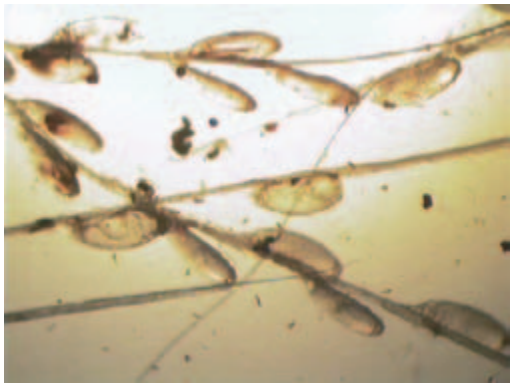

**Fig. 22.5** Lice eggs in the coat of a mouse.

## *FLEAS*

Cat fleas can be isolated from pet mice kept in households with dogs and cats. Wild mice can harbour many species of flea (e.g. *Xenopsylla* and *Nosopsylla* spp.).

# ENDOPARASITES

## *PINWORM*

*Cause and pathogenesis*

Pinworm infection in the mouse is due to *Syphacia obvelata*.

*Clinical signs*

Lesions are associated with perianal pruritus and tail-base mutilation.

*Diagnosis and therapy*

Diagnosis is by microscopic examination of cellophane tape applied to the perineal area which will reveal the banana-shaped eggs. Treatment is with ivermectin (Le Blanc *et al*. 1993).

## NUTRITIONAL DISEASE

Nutritional disease is rare in pet and laboratory mice, but can be produced experimentally. Zinc deficiency and pantothenic acid cause an exfoliative dematitis, and hair depigmentation. Zinc deficiency also causes alopecia. Riboflavin deficiency produces alopecia and scaling, and pyridoxine, biotin and fatty acid deficiency cause exfoliative dermatitis (Scott *et al*. 2001).

## ENVIRONMENTAL / BEHAVIOURAL CONDITIONS

### *BARBERING*

*Cause and pathogenesis*

Barbering is common in groups of mice, especially males, where the dominant mouse chews the hair of subordinates without causing skin damage. Mice may also rub the muzzle bald from eating between wire cage bars.

*Clinical signs*

Hair is lost from areas which have been traumatised. Hair loss in incomplete due to chewing so that alopecic areas contain fine stubble.

*Diagnosis and therapy*

Diagnosis is by observing barbering behaviour and examination of the hair ends which are traumatised. Treatment is by reducing stocking density, removing the offending individual (although another mouse often takes over this role) and enriching the environment with more bedding and cage furniture.

### *FIGHT WOUNDS*

*Cause and pathogenesis*

Commonly seen in groups of mice when the dominant mouse attacks others. Compared to barbering lesions tend to be more severe.

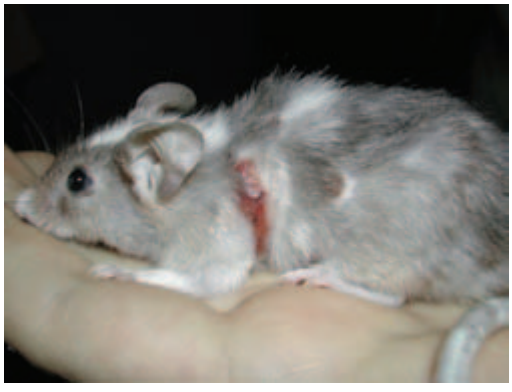

**Fig. 22.6** Fight wounds on the flank of a mouse.

*Clinical signs*

Lesions are usually localised, deep lacerations and puncture wounds (Figure 22.6). Compared to self-inflicted trauma due to pruritic skin disease the rest of the coat appears to be good.

*Diagnosis and therapy*

As barbering.

## *RINGTAIL*

*Cause and pathogenesis*

Low environmental humidity (<20%) can cause a condition known as 'ring-tail' It occurs mainly in pups under fifteen days old. Although generally reported as a condition of rats, ringtail occasionally occurs in mice.

*Clinical signs*

One or more annular constrictions of the tail can cause oedema, necrosis and sloughing of the distal tissue.

*Diagnosis and therapy*

Diagnosis is made on the basis of history, especially where conditions of low humidity exist, and clinical signs. Therapy is to ensure conditions are changed and provide supportive care.

# MISCELLANEOUS CONDITIONS

## *IMMUNE-COMPLEX VASCULITIS*

Immune-complex vasculitis with secondary ulcerative dermatitis has been reported in aged C57BL/6Nnia laboratory mice (Andrews *et al*. 1994) and has been seen in other laboratory strains by the author.

## *IDIOPATHIC DRY GANGRENE OF THE PINNAE*

This condition can be seen in young mice and seems to be linked to cold temperatures and excessive grooming associated with lice infestation (Scott *et al*. 2001). Erythema of the distal third of the pinna develops rapidly to necrosis and sloughing.

# ALOPECIA

A variety of different coat types are recognised in mice and rats. The main types are the Standard smooth-coated mouse, the curly-coated Rex and the Hairless which has no hair. This is a normal variety and should not be mistaken for clinical disease (Figure 22.7). In addition both tailed and tailless and those with normal ears as well as those with set-back ears, so-called 'Dumbo' mice, are recognised.

# NEOPLASIA

Skin neoplasia is rare in mice, but numerous types are reported, especially from laboratory strains (Peckham & Heider 1999). In pet mice, squamous cell carcinoma occurs most frequently. Dermal papilloma, fibromas, fibrosarcomas and mesenchymal tumours have also been reported.

**Fig. 22.7** Hairless 'Dumbo' mouse – note the large set-back ears typical of this variety.

*Formulary*

See p. 202, Chapter Sixteen on Chinchillas.

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Chapter 23

# Skin Diseases and Treatment of Rabbits

## BACTERIAL DISEASE

### *ABSCESSES*

*Cause and pathogenesis*

Abscesses result from entry of bacteria via a skin wound, or, more rarely, are secondary to bacteraemia. Facial abscesses are frequently associated with dental or nasolacrimal duct disease. *Pasteurella multocida* and *Staphylococcus aureus* are usually isolated from rabbit abscesses, with *Proteus, Pseudomonas, Bacteroides* and other bacteria occasionally found. Rabbit pus is thick, tenacious and very difficult to remove, and there is usually a thick abscess capsule.

*Clinical signs*

Clinical signs are of single or multiple subcutaneous nodules with or without a discharging sinus or associated wound. If associated with dental disease or nasolacrimal duct disease then facial swelling, lacrimation and anorexia are common presenting signs (Figure 23.1).

*Diagnosis and therapy*

Diagnosis can be achieved by examination of a stained fine-needle aspirate. This will need to be collected via a 21 g needle due to the caseous nature of the rabbit pus. If cytology suggests a mixed infection then culture is advisable, including anaerobic bacteria, so that an appropriate antibiotic can be selected. Radiography of the skull is vital for any facial abscess in order to obtain a prognosis. True subcutaneous abscesses are best treated by complete surgical excision. If this is not possible, lancing and aggressive flushing with antiseptic solutions, plus appropriate systemic antibiotic therapy, can be employed. Injection of gentamicin into the abscess capsule is reported by some to be effective (White *et al*. 2003). Underlying bony involvement carries an extremely poor prognosis.

Surgical debridement, removal of affected teeth and packing of the abscess cavity with antibiotic-impregnated methylmethacrylate beads can be successful in many cases. The author does not recommend the use of calcium hydroxide as it causes severe tissue necrosis, but some practitioners favour it.

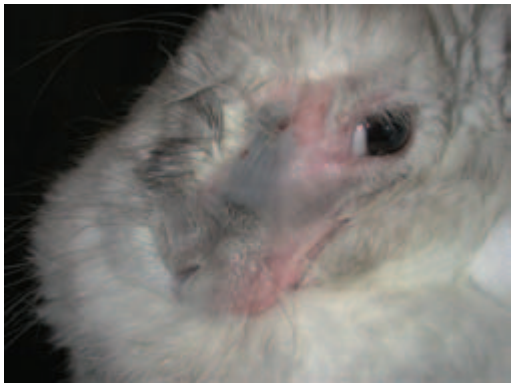

**Fig. 23.1** Abscess on the face of a rabbit.

**Fig. 23.2** Pasteurella infection on the face of a rabbit. (Picture courtesy J. Fontaine.)

## *CELLULITIS*

*Cause and pathogenesis*

Acute cellulitis is usually due to infection with *Staphylococcus aureus* or *Pasteurella multocida.* (Jenkins 2001)

*Clinical signs*

Clinical signs are of painful oedematous skin swelling, usually of the head, neck or thorax. The rabbit is usually febrile (40–42°C), depressed and anorexic. Pasteurella can also present with milder signs of facial mucocutaneous crusting (Figure 23.2)

*Diagnosis and therapy*

Diagnosis is based on clinical signs, impression smears and culture and sensitivity of exudates. Treatment consists of aggressive antibiosis based on bacterial culture and sensitivity, and supportive care including cool water baths to reduce body temperature. In surviving rabbits the cellulites may mature into an abscess or they may develop a necrotic eschar in the affected area.

## *MOIST DERMATITIS ('BLUE FUR DISEASE')*

*Cause and pathogenesis*

Moist dermatitis is common in overweight and female rabbits with a large dewlap, or in animals with severe dental disease and excess salivation. Faulty drinking apparatus can also lead to maceration of the skin. Constant wetting of the skin predisposes the site to colonisation with *Pseudomonas* spp.

*Clinical signs*

The wet fur becomes infected with *Pseudomonas aeruginosa*, or occasionally other bacteria. If *Pseudomonas* is involved the fur turns a characteristic blue colour.

*Diagnosis and therapy*

Diagnosis is based on clinical signs and cytology. Treatment involves clipping the affected area, and applying antiseptic solution (e.g. dilute chlorhexidine or acetic-acid based shampoo), plus addressing the underlying cause. Systemic antibiosis is required in some cases and should where needed be based on culture and sensitivity.

## *RABBIT SYPHILIS / VENEREAL SPIROCHAETOSIS*

*Cause and pathogenesis*

This is caused by the spirochaete *Treponema cuniculi.* Transmission is venereal and by direct contact. Affected does can infect kits as they pass through the birth canal. The incubation period is long, with lesions generally appearing three to six weeks after exposure and a positive serologic titre developing after eight to twelve weeks. Rabbit syphilis is not zoonotic.

*Clinical signs*

Clinical signs are relatively uncommon, and subclinical infection is believed to be common, with serological screening suggesting that up to 25% of rabbits are infected (Jenkins 2001). Lesions begin as redness and oedema, progressing to vesicles, ulcers, scabs and proliferative lesions around the perineum, and also around the face from auto-inoculation. Rabbits can be asymptomatic carriers, with overt disease precipitated by stress, sometimes months or years after initial exposure.

*Diagnosis and therapy*

Clinical signs are very characteristic, but definitive diagnosis involves microscopic visualisation of the organism from scrapes on a dark field background, or with

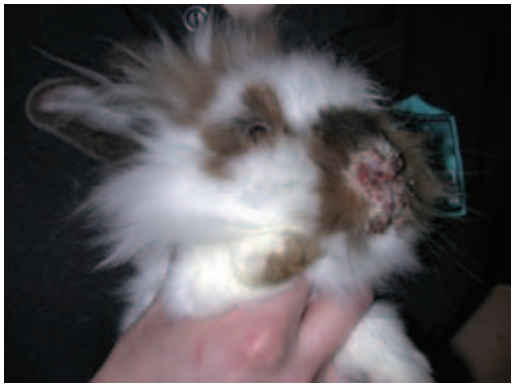

**Fig. 23.3** *Fusobacterium necrophorum* infection on the nose of a rabbit.

special silver stains on biopsy. Serology tests developed for detection of human syphilis (*Treponema pallidum*) can be used. Lesions can be self limiting, but effective treatment is with penicillin G (42 000–84 000 IU/kg) once every seven days for three doses. Treated rabbits should be monitored closely for signs of antibiotic-associated enterotoxaemia. All exposed rabbits should be treated.

## *NECROBACILLOSIS (SCHMORL'S DISEASE)*

*Cause and pathogenesis*

This is an uncommon skin infection caused by *Fusobacterium necrophorum.* This organism is commonly found in rabbit faeces, and disease results from faecal contamination of wounds.

*Clinical signs*

Swelling, inflammation, abcessation, ulceration and necrosis occur, usually on the face (Figure 23.3) and neck, and occasionally the feet, Underlying bone can sometimes be involved.

*Diagnosis and therapy*

Diagnosis is by bacterial culture. Treatment involves debridement and antibiosis. One suggested regime is penicillin at 40 000 IU/kg per day s.c. for ten to thirty days (Jenkins 2001). Tetracycline (Scott *et al*. 2001) and antibiotic-impregnated PMMA beads have also been used (Jenkins 2001).

# FUNGAL DISEASE

## *DERMATOPHYTOSIS*

*Cause and pathogenesis*

Dermatophytosis in outdoor and laboratory rabbits is most commonly caused by *Trichophyton mentagrophytes*, whereas *Microsporum gypseum* and *M. canis* are more common in pet and house rabbits. Potentially a zoonotic disease although those at risk appear to be owners who have atopy or are immunosuppressed.

*Clinical signs*

Animals can also be asymptomatic – one study showed four out of 104 healthy rabbits had *T. mentagrophytes* cultured from the coat (Vangeel *et al*. 2000). Infection can cause hair loss and crusting lesions, especially around the eyes and nose and on the extremities (Figures 23.4, 23.5).

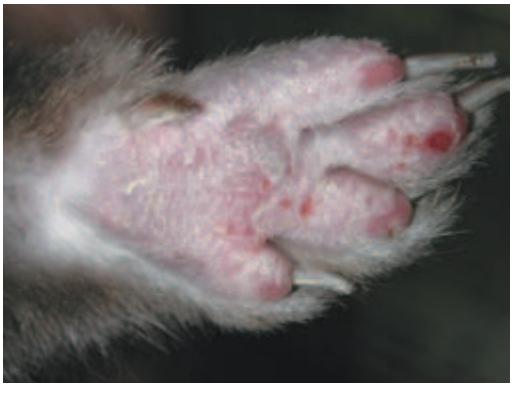

**Fig. 23.4** Dermatophytosis due to *Trichophyton mentagrophytes* on the feet of a rabbit.

**Fig. 23.5** Dermatophytosis due to *Trichophyton mentagrophytes* on the feet of a rabbit.

*Diagnosis and therapy*

Diagnosis is by microscopic examination of the hair shaft and fungal culture. Only some strains of *M. canis* and none of the strains of *Trichophyton* will fluoresce under a Wood's lamp. An indirect ELISA has also been developed for *T. mentagrophytes* (Zrimsek *et al*. 1999). Therapy can be initiated with griseofulvin at a dose of 25–50 mg/kg once daily. This is an unlicensed use of this drug and should not be used in pregnant animals due to its teratogenicity. In a group all in-contact animals should be treated. Topical enilconazole (0.2%) has also been used.

## VIRAL DISEASE

## *MYXOMATOSIS*

*Cause and pathogenesis*

Myxomatosis is caused by the myxoma virus, a double-stranded DNA poxvirus. In the domestic rabbit (*Oryctolagus cuniculi*) myxomatosis is a severe and invariably fatal systemic disease. In 1950 the virus was released into Australia as a means of controlling wild *Oryctolagus cuniculi.* In 1952, the disease was introduced to France to control rabbits, and it spread to the rest of continental Europe and the UK. The disease is not generally seen as a clinical problem in domestic rabbits in the USA, although it has been described in California and Oregon. The incubation period is eight to twenty-one days.

The virus is transmitted passively by blood-feeding arthropods, usually the rabbit flea and the mosquito. The virus does not replicate within the vector. The virus replicates at the inoculation site and spreads within leukocytes to the draining lymph node, where it replicates further and disseminates to the skin, spleen, other lymph nodes, mucosal surfaces, testes, lungs and liver.

Although the virus is also shed in discharges, transmission by close contact is very unusual.

*Clinical signs*

Clinical signs are swelling of the eyelids and genitals, a milky ocular discharge, pyrexia, lethargy, depression and anorexia. More generalised swelling of the face and ears occurs and skin nodules up to 1 cm in diameter may be found on the face including the ears (Figure 23.6) and body. Death usually occurs within fourteen days and is thought to be due to overwhelming bacterial infection. A milder form of the disease is seen in previously vaccinated rabbits. These often present with a scabbing lesion on the bridge of the nose and around the eyes, or multiple cutaneous masses over the body. Affected animals often survive with nursing care.

**Fig. 23.6** Nodular lesions of myxomatosis on the ears of a rabbit. (Picture courtesy Z. Alha i dari.)

*Diagnosis and therapy*

Diagnosis is based on clinical signs and histopathology. Virus isolation can confirm the diagnosis. There is no effective treatment and affected animals should therefore be euthanased on humane grounds.

Control is mainly by vaccination using a live attenuated Shope Fibroma virus, which gives cross immunity, although this is rather short-lived and can be poor in some animals. Only healthy rabbits should be vaccinated, and immunity takes fourteen days to develop. The vaccine is not safe in pregnant does. The first dose is given at or after six weeks of age and boosters should be given every six to twelve months, depending on the perceived risk. The mode of administration is vital in obtaining a good immune response – one-tenth of the dose is given intradermally and the rest subcutaneously. The skin should not be swabbed with disinfectants or alcohol prior to injection.

Attention should also be given to vector control, with the use of insect-proof screening for outdoor rabbits as well as flea control. Fleas brought in by cats may infect indoor rabbits. Contact with wild rabbits should be avoided.

## *SHOPE PAPILLOMA VIRUS*

*Cause and pathogenesis*

The Shope papilloma virus is a papovavirus. It occurs in wild California brush rabbits (*Sylvilagus bachmani*) and cottontail rabbits (*Sylvilagus floridanus*). An insect vector is necessary for transmission.

*Clinical signs*

Infection of domestic rabbits (*Oryctolagus cuniculus*) is rare but has been reported, causing multiple horn-like lesions around the ears and eyelids.

*Diagnosis and therapy*

Diagnosis is based on clinical signs and viral isolation. Manual removal of the lesions usually results in healing and recovered rabbits are resistant to reinfection. Experimental infection resulted in approximately 75% of inoculation sites undergoing malignant transformation to squamous cell carcinoma.

## *SHOPE FIBROMA VIRUS*

*Cause and pathogenesis*

The Shope Fibroma virus is a naturally occurring poxvirus of North and South American wild rabbits (*Sylvilagus* spp.). Domestic rabbits are occasionally infected via mosquito vectors.

*Clinical signs*

Fibroma lesions appear as single or multiple flat subcutaneous nodules especially on the genitals, perineum, ventral abdomen, paw, nose, pinna and eyelid. Lesions will usually slough away about 30 days post inoculation. Newborn and young animals develop more extensive lesions.

*Diagnosis and therapy*

Diagnosis is by clinical signs and viral isolation. Therapy is not necessary in most cases due to spontaneous involution. A live attenuated Shope fibroma virus is used as the myxomatosis vaccine.

# PARASITIC DISEASE

## *MITES*

Important ectoparasites of rabbits include:

- **Ticks.**
  - *Haemaphysalis leporis-palustris.*
- **Mites.**
  - Listrophoridae – *Leporacarus gibbus.*
  - Psoroptidae – *Psoroptes cuniculi.*
  - Cheyletiellidae – Cheyletiella *parasitovorax.*
- Other less important mites
  - Psorergatidae – *Psorobia lagomorphae.*
  - Demodicidae – *Demodex cuniculi.*

- Sarcoptidae – *Notoedres cati*, *Sarcoptes scabiei.*
- Trombiculidae – *Neotrombicula autumnalis.*

- **Insects.**
  - Lice.
    - *Haemodipsus ventricosus.*
  - Fleas.
    - *Spillopsyllus cuniculi.*
    - *Ctenocephalides felis.*
    - *Cediopsylla simplex.*
    - *Odontopsyllus multispinous.*
    - *Echidnophaga gallinacea.*

## *TICKS*

*Cause and pathogenesis*

Many species of tick can parasitise rabbits, the most common being the continental rabbit tick *Haemaphysalis leporis-palustris.* However other ticks such as the Spinose ear tick – *Otobius megnini* and *Ixodes* spp. can be found on rabbits.

*Clinical signs*

Heavy infestation can cause anaemia, and ticks can act as vectors for myxomatosis, papillomatosis and tularaemia. Infestations with ear ticks can lead to serious damage to the ear and ear drum.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs. Ticks can be removed manually or treated with systemic ivermectin at 0.4 mg/kg (Jenkins 2001).

## *MITES*

### *LISTROPHORIDAE*

### *Listrophorus (Leporacarus) gibbus*

*Cause and pathogenesis*

*Listrophorus (Leporacarus) gibbus* is a non-burrowing fur mite commonly described as non-pathogenic even in heavy infestations. It is thought to feed on sebaceous secretions and hair debris. Humans handling infested rabbits may develop dermatitis.

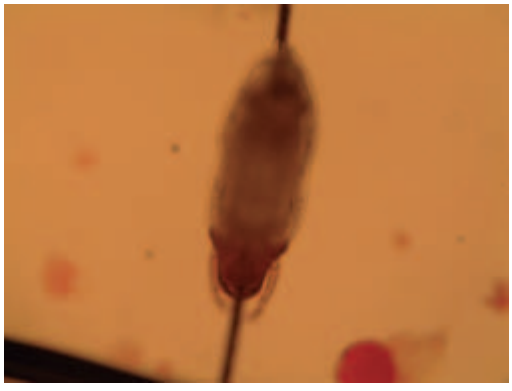

**Fig. 23.7** Fur-clasping mite on the hair of a rabbit.

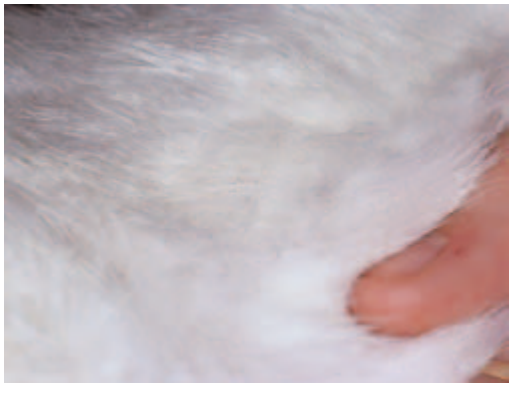

**Fig. 23.8** *Listrophorus gibbus* infestation in a rabbit. (Picture courtesy J. D. Littlewood.)

*Clinical signs*

The mite occurs throughout the fur and tends to be found clinging to single hairs rather than browsing over the skin surface (Figure 23.7). Infestations are commonly asymptomatic. Rarely, infestation can be associated with hair loss, seborrhoea and abnormal moult (Figure 23.8) (Jenkins 2001, Pinter 1999).

*Diagnosis and therapy*

Diagnosis is by identification of the mites. Oval mites legs are of similar shape not adapted for clasping. Treatment not indicated.

## *PSOROPTIDAE*

### *PSOROPTES CUNICULI*

*Cause and pathogenesis*

*Psoroptes cuniculi* is the rabbit ear mite. It is a non-burrowing mite which causes intense aural irritation. The lifecycle is less than three weeks, and adult mites can live of the host for up to 21 days depending on ambient temperature and humidity. It feeds on loose epidermal debris especially lipid material. Antigenic material in the mite's saliva and faeces can invoke an intense inflammatory reaction. Transmission is by direct contact.

*Clinical signs*

Mite infestations lead to head shaking, scratching of the ears with hyperaemia. Ear lesions produce signs of erythema with mild crusting (Figure 23.9). In more advanced cases copious exudate is produced in response to the mites, leading to thick crust formation filling the auditory canal (Figure 23.10) which can spread to the face and neck. In addition, the eardrum can perforate leading to a purulent otitis media (secondary bacterial infection) and meningitis.

*Diagnosis and therapy*

Mites can be visualised on otoscopic examination (Figure 23.11), or by microscopic examination of aural debris. Mites are oval shaped and their legs end in long, segmented stalks with suckers (Figure 23.12). The recommended treatment is

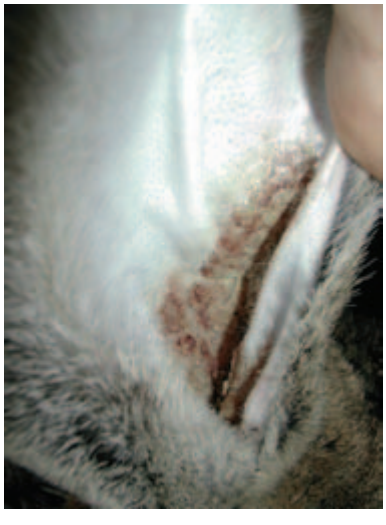

**Fig. 23.9** A mild case of *Psoroptes cuniculi.*

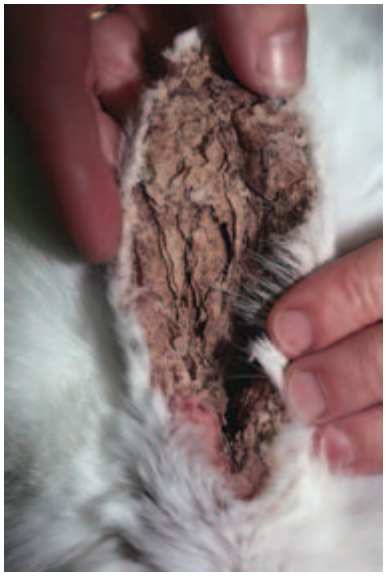

**Fig. 23.10** Severe *Psoroptes cuniculi* infestation in the ear of a rabbit. (Picture courtesy Z. Alha i dari.)

**Fig. 23.11** *Psoroptes* mites in microscopic sections.

**Fig. 23.12** Long segmented sucker of *Psoroptes* mite.

ivermectin (0.4 mg/kg s.c. once every ten to fourteen days for three treatments) or moxidectin (0.2 mg/kg s.c. once every ten days for two treatments) (White *et al*. 2003). All in-contact animals should be treated. Mild infections may be treated with acaricidal ear drops. Thick crusts will resolve with systemic treatment but, if necessary, they can be softened with mineral oil before being removed; otherwise the lining of the ear canal can be damaged.

## *CHEYLETIELLIDAE*

### *CHEYLETIELLA PARASITOVORAX*

#### *Cause and pathogenesis*

*Cheyletiella parasitovorax* is the rabbit fur mite. It is a non-burrowing mite, just visible to the naked eye. Many rabbits carry the mites with no overt signs. The mite lives on the skin surface especially on the dorsal surfaces of the body. The mite pierces the skin with its needle-like mouth parts to feed on tissue fluids. Transmission is by direct contact. *Cheyletiella* is zoonotic, causing a papular dermatitis in man. The lifecycle is fourteen to twenty-one days, and adult females can survive for at least ten days off the host.

#### *Clinical signs*

Lesions are generally not severe, with crusting and scaling along the dorsum, mild pruritus and partial alopecia in heavy infestations (Figure 23.13). Classically described as 'walking dandruff'. Cases may be more severe in immunocompromised animals (Figure 23.14).

#### *Diagnosis and therapy*

Diagnosis is easily made with the acetate tape test. Mites are typically saddle

**Fig. 23.13** Scaling on the back of a rabbit with cheyletiella.

shaped with hook-shaped mouth parts (Figure 23.15). Treatment of choice is ivermectin at 400µg/kg subcutaneously once every ten to fourteen days for three doses. Alternative treatments include selamectin (Harcourt-Brown 2002), lime–sulphur dips and topical permethrin products. All in-contact animals should be treated and the environment thoroughly cleaned.

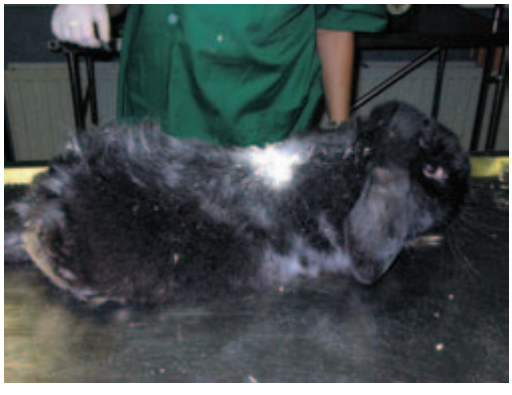

**Fig. 23.14** Severe generalised cheyletiella infestation. (Picture courtesy J. Fontaine.)

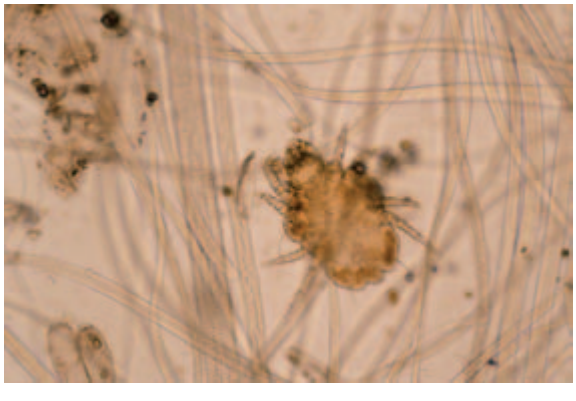

**Fig. 23.15** Cheyletiella mite. (Picture courtesy J. D. Littlewood.)

## *OTHER MITES*

### *PSORERGATIDAE*

A new subspecies of *Psorobia lagomorphae* has been reported as causing mild pruritus and alopecia in a six-month-old dwarf rabbit (Bordeau *et al*. 2001).

### *SARCOPTIDAE*

*Sarcoptes scabiei* var. *cuniculi* and *Notoedres cati* var. *cuniculi* are occasionally reported but are rare. They are associated with a pruritic dermatosis. Treatment is with ivermectin as for other mites.

### *DEMODICIDAE*

*Demodex cuniculi* is rarely found, and its pathological significance is unknown (Harvey 1990). Affected rabbits can show variable levels of pruritus.

### *TROMBICULIDAE*

Harvest mites (*Trombicula autumnalis*) can be found on outdoor rabbits and cause intense pruritus, macule and pustule formation (Harcourt-Brown 2002).

## *FLEAS*

*Cause and pathogenesis*

*Spillopsyllus cuniculi* ('stick-tight' flea) is the rabbit flea and is important as a vector for myxomatosis. Cat fleas (*Ctenocephalides felis*) can also live on rabbits, and these are more commonly found in pet rabbits. In the USA the Eastern rabbit flea (*Cediopsylla simplex*), giant Eastern rabbit flea (*Odontopsyllus multispinous*) and the stick-tight flea (*Echidnophaga gallinacea*) may also be found.

*Clinical signs*

Heavy flea infestations can lead to pruritus and self-inflicted trauma presenting as traumatic hair loss and excoriation.

*Diagnosis and therapy*

Diagnosis is made by catching the fleas (Figure 23.16) on the rabbit or by a positive wet paper test to identify the presence of flea faeces. Treatment of the rabbit is rarely required if cats and dogs and the environment are treated in the usual way. Imidocloprid (Hutchinson *et al*. 2001) and selamectin (Harcourt-Brown 2002) are effective flea treatments in rabbits. Long-term use of lufenuron is also reported to be safe (Jenkins 2001). Adverse reactions to Fipronil® have been reported and this product should not be used.

**Fig. 23.16** The rabbit flea *Spillopsyllus cuniculi.* (Picture courtesy J. D. Littlewood.)

## *LICE*

*Cause and pathogenesis*

*Haemodipsus ventricosus* is a rare finding on pet rabbits.

*Clinical signs*

It can cause anaemia and pruritus especially in immunosuppressed hosts.

*Diagnosis and therapy*

Systemic ivermectin can be used as treatment, and imidacloprid seems to be effective in dogs and could be used in rabbits.

## *OTHER PARASITES*

### *MYIASIS – FLYSTRIKE*

*Cause and pathogenesis*

Flystrike is common in rabbits in summer months. In the UK it is caused mainly by the Greenbottle fly (*Lucilia* spp). Strike is usually primary (i.e. intact skin) and flies are attracted by caecotroph accumulation around the perineum and especially

in the folds either side of the genitals. This is invariably due to lack of caecotrophy, which can be due to a number of factors – dental disease, obesity, back problems, and old age. True diarrhoea and urine scalding due to urinary incontinence will also attract flies. Eggs can hatch within twelve hours to L1 maggots, which are not harmful. Within three days L1 larvae moult to L2 and L3 which cause the tissue damage. Environmental conditions of at least 60% humidity and 9–11°C are required for larval development. It is thought that the maggots may secrete a local anaesthetic and so the damage is rarely observed to be painful to the rabbit. In the USA the flesh fly (*Wohlfartia vigil*) is most common, with eggs being laid at the edge of wounds.

*Clinical signs*

Clinical signs become apparent four days after eggs hatch and are dependent on the numbers of maggots present and the time of infestation. Erosions, ulcers and extensive tissue damage are associated with maggots.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs. Initial treatment involves clipping of the fur and cleaning of the area, with manual removal of the maggots and flushing of the area with dilute antiseptic solution. Supportive therapy should be given immediately for toxic shock (warmth, fluids, corticosteroids in severe cases). Systemic ivermectin at 0.4 mg/kg subcutaneously may also be given which will kill any subcutaneous or internal maggots as well as any that hatch subsequently. Secondary bacterial (often clostridial) infection of the necrotic tissue often occurs and appropriate antibiotic cover should also be given. Topical silver sulfadiazine cream has also been recommended (Jenkins 2001). The underlying cause of the caecotroph or urine accumulation must then be addressed. Cyromazine is an insect growth inhibitor licensed for prevention of flystrike in rabbits. It does not repel flies but prevents the moult from L1 to L2. Permethrin spot-on products can also be applied every two weeks as a preventive measure. Some products available also contain a fly repellent.

## *CUTEBRA* spp.

*Cutebra* spp. larvae can also affect rabbits, causing fistulous nodules. The larvae should be individually removed.

## *COENURUS SERIALIS*

*Coenurus serialis* cysts caused by the tapeworm *Taenia serialis* have been reported in rabbits as causing fluctuant subcutaneous swellings which can be removed surgically (Fountain 2000; Bennett 2001; Wills 2001).

### *PASSALURUS AMBIGUOUS*

The rabbit pinworm *Passalurus ambiguous* is common in laboratory rabbits and can occasionally be associated with pruritus of the anal area leading to self trauma and rectal prolapse.

## ENVIRONMENTAL AND BEHAVIOURAL CONDITIONS

### *ULCERATIVE PODODERMATITIS*

*Cause and pathogenesis*

This is a chronic ulcerative granulomatous dermatitis of the metatarsal area seen generally in overweight inactive rabbits kept on wet bedding or grid floors. Occasionally the metacarpal area can be affected. Hereditary factors are also thought to be involved and Rex rabbits are particularly affected as they lack the protective guard hairs.

*Clinical signs*

Increased pressure on the skin closely overlying bone leads to ischaemia and necrosis which appear as ulcerated sores (Figure 23.17). Secondary infection with *Staphylococcus aureus* occurs, which can progress to an osteomyelitis. Infection of the synovial structures can result in displacement of the superficial flexor tendon (Harcourt-Brown 2002).

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs. Cytology of lesions can help in identifying any secondary infection. Cultures should be undertaken where rods are present on cytology. Treatment involves addressing the initiating cause, plus debridement and cleaning of the lesions, topical and systemic antibiotic therapy,

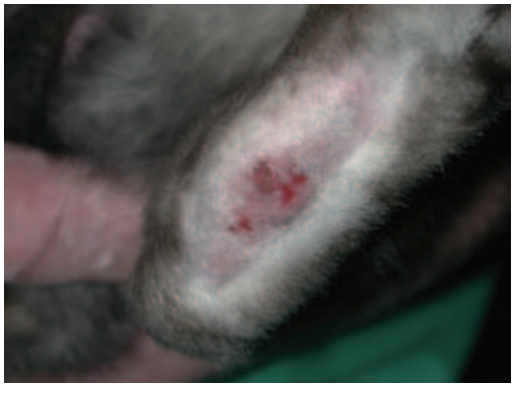

**Fig. 23.17** Ulcerated sores on the hocks of an overweight rabbit.

application of dressings, and analgesia. Lesions are difficult to resolve and a guarded prognosis should be given to rabbits suffering form osteomyelitis and tendon displacement.

## *BARBERING AND EXCESSIVE GROOMING*

These can occur in rabbits but are not common problems. Rabbits can be barbered by dominant animals (Hillyer 1997, Scarff 2000) but it is more likely to be performed by a subordinate companion (Jenkins 2001). Self barbering may occur in does in oestrus or rabbits on a low-fibre diet. Excessive grooming is a rare behavioural problem.

## *SELF MUTILATION*

Compulsive self mutilation has been seen as a genetic problem in highly inbred Checkered cross rabbits (Iglauer *et al*. 1995) and subsequent to intramuscular ketamine and xylazine use (Beyers *et al*. 1991).

# NEOPLASIA

Shope fibroma virus and Shope papilloma virus are oncogenic (see pp. 294–5, Viral disease). Spontaneous non-viral cutaneous neoplasia is rare in rabbits. In one study, trichoblastomas (basal cell tumours) were the most common cutaneous neoplasm (Mauldin & Goldschmidt 2002). Cutaneous lymphoma has been described in rabbits (Mauldin & Goldschmidt 2002; White *et al*. 2000; Hinton & Regan 1978).

Squamous cell carcinoma, sebaceous gland carcinoma, basal cell carcinoma. malignant melanoma and papilloma are other cutaneous neoplasms reported in the rabbit (Scott *et al*. 2001; Mauldin & Goldschmidt 2002).

# MISCELLANEOUS CONDITIONS

## *EOSINOPHILIC GRANULOMA*

*Cause and pathogenesis*

An eosinophilic granuloma-like lesion, identified as a type II eosinophilic plaque has been reported in a New Zealand white rabbit (Henriksen 1983). The author has had one confirmed case of eosinophilic granuloma in a mixed-breed house rabbit (unpublished). Lesions are likely to form as a result of self-inflicted trauma secondary to an allergic reaction.

*Clinical signs*

Clinical presentation was self mutilation of the ventral abdomen, weight loss, and an erythematous, necrotic, ulcerative well-demarcated lesion stretching from the umbilicus to the perineum.

*Diagnosis and therapy*

Diagnosis of the lesions can be made by impression cytology and histopathology. Investigations should be undertaken to look for an underlying cause of the lesion such as infection (dermatophytosis) or ectoparasites as well as symptomatic therapy. In the case seen by the author Cheyletiella mites and eggs were found in scrapings taken from the edge of the lesion. Complete resolution was achieved with a depot injection of methylprednisolone acetate and treatment of the mites with ivermectin.

## *SEBACEOUS ADENITIS*

*Cause and pathogenesis*

Sebaceous adenitis has been reported in four rabbits (White S.D. *et al*. 2000). The aetiology is unknown. In a similar condition in the dog the disease is caused by an immune-mediated attack against the sebaceous glands in the skin.

*Clinical signs*

Non-pruritic scaling and alopecia occur. Prominent follicular casts and seen on hairs which can be easily epilated.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs, diagnostic rule outs of other common conditions such as dermatophytosis and biopsy. Histopathology reveals inflammation directed at the sebaceous glands, in chronic cases glands are lost. Attempted treatment in two cases with retinoids and essential fatty acids was not effective. Topical therapy with anti-seborrhoeic shampoos may be beneficial.

## *TELOGEN DEFLUXION*

*Cause and pathogenesis*

Widespread hair loss occurs four to six weeks after some sort of systemic stress. This condition is commonly seen after systemic illness or after parturition.

*Clinical signs*

Rabbit is non pruritic. Hair can be easily epilated and is lost rather than rubbed or scratched out. Coat has a moth-eaten appearance (Figure 23.18).

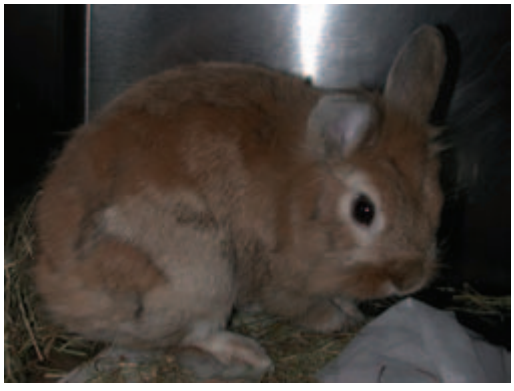

**Fig. 23.18** Telogen defluxion in a rabbit with systemic disease.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs, history and diagnostic rule out, especially sebaceous adenitis and dermatophytosis. Treatment is not necessary providing the rabbit has recovered from the inciting episode.

## *CUTANEOUS ASTHENIA*

*Cause and pathogenesis*

Cutaneous asthenia due to a collagen defect similar to Ehlers-Danlos syndrome has been reported in the rabbit (Harvey *et al*. 1990).

*Clinical signs*

Rabbits present with hyperextensible skin which is particularly obvious over the shoulder. Trauma to the skin leads to trauma. Skin generally heals with 'cigarette paper' type scars. Particular care must be taken when handling and especially grooming animals.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs. Histopathology of the skin of affected individuals is often unremarkable; electron microscopy is usually required for a definitive diagnosis. Anecdoctal reports have been made that Vitamin C supplementation has been beneficial in other domestic species.

**Table 23.1** Rabbit formulary.

| Drug | Dose | Comments |
|---|---|---|
| *Antibacterials* | | |
| Cephalexin | 11–22 mg/kg | May cause enteritis. |
| Chloramphenicol | 50 mg/kg i.m. bid | |
| Doxycycline | 2.5 mg/kg bid | |
| Enrofloxacin | 5–10 mg/kg bid or 10–20 mg/kg sid | Licensed for rabbits in the UK. |
| Gentamicin | 4 mg/kg s.c., i.m. tid | |
| Marbofloxacin | 2–5 mg/kg p.o., s.c., bid | |
| Metronidazole | 20 mg/kg bid | |
| Oxytetracycline | 15 mg/kg i.m. bid | |
| Penicillin | 42 000–84 000 IU/kg s.c. every seven days for three doses. | For rabbit syphilis. |
| Trimethoprim / sulphadiazine | 30 mg/kg bid | |
| *Antifungals* | | |
| Griseofulvin | 25 mg/kg p.o. sid for 28–40 days. | |
| *Antiparasitics* | | |
| Ivermectin | 0.2–0.4 mg/kg s.c. every seven to fourteen days. | |
| Moxidectin | 0.2 mg/kg s.c. every ten to fourteen days. | |
| Imidocloprid | One cat dose (40 mg) divided into two to three spots on dorsum. | Minimum 10 mg/kg. |
| Selamectin | 6 mg/kg topically, monthly. | |
| Lufenuron | 30 mg/kg p.o. monthly. | |
| Cyromazine | 6% solution. | Follow manufacturer's instructions. |
| Permethrin / pyrethrins | Topical. | Use products safe for kittens and puppies. |

## *RAISED SKIN PATCHES*

*Cause and pathogenesis*

Raised skin patches during hair growth can occur as rabbits age (Collins 1987).

*Clinical signs*

Hair growth waves become less frequent and patchy and these irregular patches appear as thickened islands of skin which are redder and more vascular than sur-

rounding areas. These changes are due to increasing follicular size and enlargement of the cutaneous vascular phase during anagen.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs, therapy is not necessary.

## *DERMAL FIBROSIS*

*Cause and pathogenesis*

Dermal fibrosis has been reported in two entire male rabbits.

*Clinical signs*

Both affected rabbits exhibited cutaneous thickening on the dorsum (Hargreaves & Hartley 2000; Mackay 2000). No associated pruritus or alopecia was present.

*Diagnosis and therapy*

Diagnosis may be made on the basis of clinical signs and biopsies. The histological appearance in one case was similar to that seen in biopsies from cheek skin of entire male cats, and the author suggests that the lesion may be hormonally related. Therapy has not been described.

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Chapter 24

# Skin Diseases and Treatment of Rats

## BACTERIAL DISEASE

### *STAPHYLOCOCCAL AND STREPTOCOCCAL INFECTIONS*

*Cause and pathogenesis*

Both *Staphylococcus aureus* and Streptococcus have been identified as causing skin disease in rats. Trauma, such as bite wounds (Figures 24.1, 24.2), or a mite infestation are common primary factors, although often no underlying cause is found.

*Clinical signs*

While some lesions scab over and regress, many are highly pruritic and lead to severe self mutilation. For diagnosis and therapy see Other Bacteria.

**Table 24.1** Clinical signs of bacterial disease in rats.

| Organism | Syndrome | Clinical signs |
|---|---|---|
| *Staphylococcus aureus* | Ulcerative dermatitis especially in young males. | Dermal ulceration and self-trauma, typically around the head, face and across the shoulders; may extend down trunk. |
| *Staphylococcus aureus* (atypical, slow growing) | In SPF laboratory rats granulomatous dermatitis and mastitis. | Granulomatous dermatitis and mastitis (Kunstyr *et al*. 1995). |
| *Staphylococcus aureus* and *Streptococcus* spp. | Self-trauma or fight wounds. | Dermatitis and abscesses. |
| *Streptococcus moniliformis* | | Oedema and cyanosis of the extremities (Ellis & Mori 2001). |

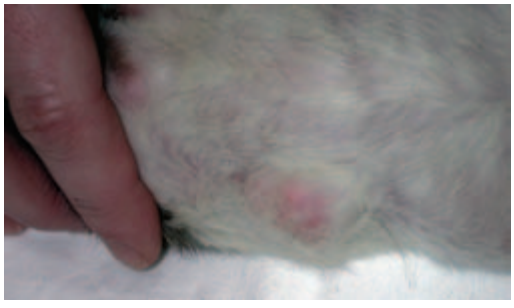

**Fig. 24.1** Abscess on a rat caused by a bite wound.

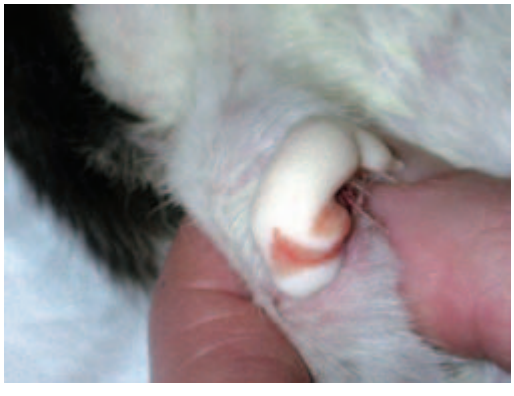

**Fig. 24.2** Thick caseous discharge from abscess in Fig. 24.1.

## *OTHER BACTERIA*

Skin abscesses are also associated generally with bite wounds. In addition to Gram-positive infections, *Pasteurella pneumotropica*, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa* and *Mycobacterium lepraemurium* can also be found (Scott *et al*. 2001, Ellis & Mori 2001).

*Corynebacterium kutscheri* can cause furunculosis and cutaneous pyogranulomas, and occasionally necrosis and sloughing of the extremities.

### *Diagnosis and therapy*

Diagnosis should be made on the basis of clinical signs, cytology of lesions and culture. Where anaerobic or mycobacterial infection is suspected then the laboratory should be made aware of this.

Treatment of all bacterial dermatoses includes elimination of predisposing factors, surgical drainage or preferably excision of abscesses, topical antiseptics such as chlorhexidine 0.5–1.0% and systemic antibiosis based on culture and sensitivity testing. Trimming the toenails to minimise self trauma is also useful (Wagner *et al*. 1977).

# VIRAL DISEASE

## *POXVIRUS*

*Cause and pathogenesis*

Poxvirus infection has been reported in laboratory rats but is extremely rare.

*Clinical signs*

It causes erythematous papules, mainly on the tail, paws and muzzle. These can become crusted and occasionally the affected area can become necrotic and slough.

*Diagnosis and therapy*

Diagnosis is by histopathology of the lesions, electron microscopy and virus isolation. Therapy should be aimed at supportive care and treatment of any secondary infection.

## *CORONAVIRUS*

*Cause and pathogenesis*

Coronavirus has been identified as causing Sialodacryoadenitis.

*Clinical signs*

Rats present with peri-orbital swelling, keratoconjunctivitis and peri-ocular irritation, and chromodacryorrhoea.

*Diagnosis and therapy*

Diagnosis may be made on the basis of clinical signs as well as histopathology of lesions, electron microscopy and virus isoloation. Therapy should be supportive together with drugs for secondary infection.

# FUNGAL DISEASE

## *DERMATOPHYTOSIS*

*Cause and pathogenesis*

Dermatophytosis is rare in rats, but *Trichophyton mentagrophytes* is the most commonly encountered causal agent. Transmission is from direct contact and also

from fomite contact. Dermatophytes can remain infectious in the environment for long periods under the right conditions. Owners should be alerted to the zoonotic potential of this infection.

*Clinical signs*

Animals can remain lesion free as asymptomatic carriers. Clinical signs are more likely to occur in immunosuppressed animals where they can present with alopecia and non-pruritic scaling, usually along the dorsum and tail base.

*Diagnosis and therapy*

Diagnosis is by microscopic examination of skin scrapings in 10% KOH or fungal culture. Treatment is with oral griseofulvin or enilconazole wash twice a week until two cultures are negative. For groups of affected rats, environmental treatment with a spray of enilconazole solution of 50 mg per square metre, twice weekly for twenty weeks can be used.

## ECTOPARASITES

Important ectoparasites of rats include:

- **Mites**.
  - Myobidae – *Radfordia ensifera*.
  - Sarcoptidae – *Notoedres muris*, *Sarcoptes scabiei* var. *cuniculi*, *Trixacarus diversus*, *Trixacarus caviae*.
  - Dermanyssidae – *Liponyssoides sanguineus*.
- **Insects**.
  - Lice – *Polyplax spinulosa* (the spined rat louse).

### *MITES*

### *MYOBIDAE*

### *Radfordia ensifera*

*Cause and pathogenesis*

Occurs at the base of the hairs throughout the fur especially on the head and neck. The mite feeds or extracellular body tissue. Transmission is by direct contact. Radfordia is thought to have a lifecycle of 21–23 days.

*Clinical signs*

Infestation leads to pruritus around the head and shoulders leading to self-inflicted ulcerative and scabbing lesions. Secondary bacterial dermatitis and an allergic dermatitis can be associated with this mite (Ellis & Mori 2001).

*Diagnosis and therapy*

Diagnosis is by identification of the mites on tape strippings and skin scrapes. Mite has on oval body and legs have prominent claws. It is similar in appearance to *M. musculi* but has two claws on tarsus I instead of one. Treatment is with ivermectin at 200–400 μg/kg s.c. every ten to fourteen days for two to three doses. Oral treatment with ivermectin 0.08% sheep drench (Oramec™, Merial Animal Health Ltd) at 4 ml (3.2 mg) per litre of drinking water for one week on, one week off, one week on is effective (MacHole 1996). Topical selamectin can also be used – one study found 100% efficacy in mice at 90 days of both 12 mg/kg and 24 mg/kg given on day 0 and day 30 (Njaa *et al*. 1957).

## *SARCOPTIDAE*

### *Notoedres muris (Ear Mange Mite)*

*Cause and pathogenesis*

*Notoedres muris* is a burrowing mite which is reported as rare but quite is commonly encountered in pet rats by the author. The lifecycle is nineteen to twenty-one days and eggs hatch in four to five days. The mite is usually located on the external ear pinnae and nose.

*Clinical signs*

Infestations of the nose and ear flaps leads to the production of warty, papular lesions with associated yellow crust. When lesions occur on the tail, and occasionally on the limbs and genitalia they tend to be erythematous and vesicular or papular.

*Diagnosis and therapy*

Diagnosis is by deep skin scrapings or biopsy. Mites have round bodies and short conical legs of which only the first pair protrudes beyond body margin (Figure 24.3). The first pair of legs end in long stalks with suckers. Treatment is with ivermectin.

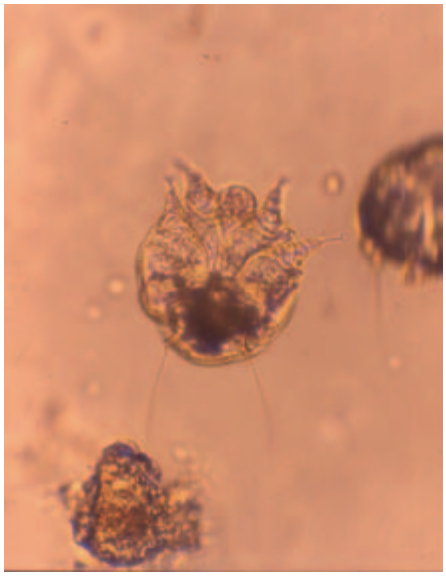

**Fig. 24.3** Rat mange mite *Notoedres muris*.

## *OTHER SARCOPTIFORM MITES*

*Sarcoptes scabiei* var. *cuniculi*, *Trixacarus diversus*, *Trixacarus caviae* are occasionally reported but rare. When they occur they are usually associated with a pruritic dermatosis. Diagnosis and therapy is as Notoedres.

## *DERMANYSSIDAE*

### ***Liponyssoides sanguineus***

*Cause and pathogenesis*

*Liponyssoides sanguineus* is the house mouse mite and has been recorded from both mice and rats. It is a blood-feeding parasite which spends most of its time off the host in the bedding. The precise impact of infestation is unknown.

*Clinical signs*

The feeding activity of large numbers of mites is thought to lead to poor coat condition, anaemia, debilitation and eventually death.

*Diagnosis and therapy*

Diagnosis is made by identification of the mite on the host and in the environment. The mite is red–brown during and after feeding activity. Treatment should be aimed at the environment.

## *DEMODICIDAE*

### ***Demodex ratticola***

*Cause and pathogenesis*

*Demodex ratticola* is a follicular mite found in the hair follicles around the mouth and on the tip of the muzzle of the rat.

*Clinical signs*

It is unknown whether this mite is capable of producing clinical disease.

*Diagnosis and therapy*

The mite is identified on skin scrapings and hair pluckings from the face. It is a long 'cigar-shaped' mite with short stumpy legs. Therapy has not been described.

## *OTHER MITES*

Other mites much more rarely found on rats are *Liponyssus bacoti* (the tropical rat mite), which lives in the environment and comes on to the host only to feed, and *Myobia musculi* (Scott *et al*. 2001; Walberg *et al*. 1981; MacHole 1996).

# ***INSECTS***

## *LICE*

### ***Polyplax spinulosa***

*Cause and pathogenesis*

*Polyplax spinulosa* (the spined rat louse) is a sucking louse and is uncommonly found on pet rats.

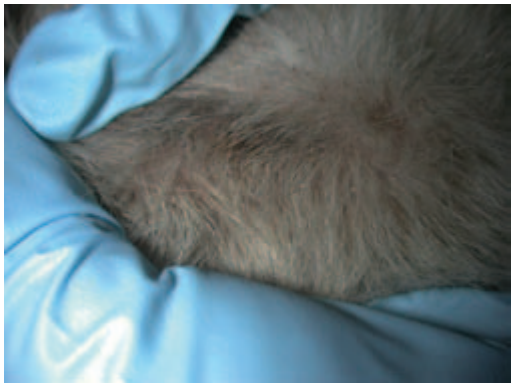

**Fig. 24.4** Lice in the coat of a rat.

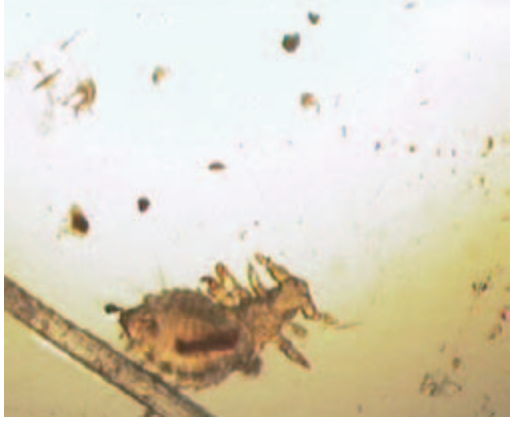

**Fig. 24.5** The rat louse *Polplax spinulosa*.

*Clinical signs*

*Polyplax spinulosa* can cause pruritus, dermatitis, restlessness and anaemia (Figure 24.4), especially in young or debilitated animals or where there is poor husbandry. It can also act as a vector for *Encephalitozoon cuniculi*, *Eperythrozoon coccoides* and *Haemobartonella muris*.

*Diagnosis and therapy*

Diagnosis is by identification of the lice and eggs ('nits') in the coat of the host (Figure 24.5).

Treatment is with systemic ivermectin or topical preparations such as Fipronil®. Topical selamectin has been used by the author and is highly effective.

## *OTHER PARASITES*

### *PINWORMS*

### *Syphacia obvelata*

*Cause and pathogenesis*

Infection can occur in rats with the pinworm *Syphacia obvelata*.

*Clinical signs*

Clinical signs are associated with perianal pruritus and tail-base mutilation.

*Diagnosis and therapy*

Diagnosis is by microscopic examination of cellophane tape applied to the perineal area which will reveal the banana-shaped eggs. Treatment is with ivermectin.

## ENDOCRINE DISEASE

Rarely, adenomas of the intermediate lobe of the pituitary may lead to excess ACTH release and hyperadrenocorticism. Iatrogenic Cushing's disease may also occur following glucocorticoid therapy.

## NUTRITIONAL DISEASE

Nutritional skin diseases are rare in both pet and laboratory rats, but can be produced experimentally. (See Table 24.2.)

**Table 24.2** Nutritional disease in rats.

| Deficiency | Clinical signs |
|---|---|
| Protein | Exfoliative dermatitis, alopecia and hair depigmentation. |
| Zinc | Exfoliative dermatitis, alopecia and hair depigmentation. |
| Pantothenic acid | Exfoliative dermatitis, alopecia and hair depigmentation and chromodacryorrhoea. |
| Riboflavin | Exfoliative dermatitis and alopecia, especially on the extremities. |
| Pyridoxine | Exfoliative dermatitis on the face ears, limbs and tail. |
| Biotin | Exfoliative dermatitis and alopecia around the eyes. |
| Niacin | Alopecia and chromodacryorrhoea. |
| Essential fatty acid | Exfoliative dermatitis and possible necrosis of the tail. |

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs and dietary analysis. Therapy should be aimed at correcting the balance of the ration.

# ENVIRONMENTAL AND BEHAVIOURAL CONDITIONS

## *BARBERING*

Barbering is rarely seen in rats. Fighting, leading to skin wounds and secondary bacterial infection can occur especially between adult males.

## *ULCERATIVE PODODERMATITIS*

*Cause and pathogenesis*

Ulcerative pododermatitis on the plantar surface of the hind feet is occasionally seen in rats. Obesity, poor cage hygiene or wire-mesh floors are contributory factors.

*Clinical signs*

Erythema and thickening of the footpad, followed by ulceration and secondary bacterial infection is seen. Secondary infection with *Staphylococcus aureus* occurs, which can progress to an osteomyelitis in severe cases.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs. Cytology of lesions can help in identifying any secondary infection. Cultures should be undertaken where rods are present on cytology. Providing any infection is controlled then treatment is often steroidal anti-inflammatory drugs and analgesia. Some rats will tolerate dressings well but many will rapidly chew them off. Euthanasia should be considered for severe cases.

## *RINGTAIL*

*Cause and pathogenesis*

Conditions of low humidity (<20–40%) are associated with an avascular necrosis of the tail. The exact aetiology is unclear, and fatty acid deficiency, low temperature, high temperature, genetics, dehydration and trauma have all been implicated

**Fig. 24.6** Ringtail in pre-weaning rats.

(Scott *et al*. 2001; Njaa *et al*. 1957; Dikshit & Sriramachari 1957). The condition is seen almost exclusively in pre-weaning rats, seven to fifteen days old.

*Clinical signs*

One or more annular constrictions develop on the tail, commonly near the base (Figure 24.6). The tail portion distal to the constriction becomes oedematous and inflamed, and then necrotic.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs and environmental conditions. Treatment should be supportive. Prevention is by maintaining the relative humidity above 50%.

## *AURICULAR CHONDRITIS*

*Cause and pathogenesis*

Auricular chondritis has been reported in several strains of rat, arising either spontaneously, in association with trauma such as ear tagging, or after injection with type II collagen. Trauma or infection are possible causes, but it is thought possible it is an immune-mediated disease (McEwen & Barsoum 1990).

*Clinical signs*

The pinnae become swollen, erythematous and nodular, then thickened and deformed. The condition is not usually irritant or painful.

*Diagnosis and therapy*

Diagnosis is made on the basis of clinical signs and histopathology. Microscopically the lesions are of granulomatous inflammatory foci with chondrolysis

and invasion of mesenchymal cells. Therapy should be undertaken with anti-inflammatory drugs.

## MISCELLANEOUS CONDITIONS

### *FUR YELLOWING*

The fur of aged male albino rats becomes yellow and coarse in texture. The exact cause is unknown, but is thought to be due to increased sebaceous secretions, and is under androgenic control (Tayama & Shisa 1994).

### *PIGMENTED SKIN SCALES*

Brown skin scales on the dorsum and tail can be observed in some rats, especially older males. One study showed that castration causes the scales to fade, and androgen administration to females produces the browning effect (Collins 1987).

### *TAIL SLIP*

Tail slip in the rat is rare but has been reported (Ellis & Mori 2001) and is a degloving injury caused by mishandling. Treatment is by amputation proximal to the injury.

## NEOPLASIA

Skin neoplasia is rare in the rat, but almost all types have been reported (Scott *et al*. 2001; Ellis & Mori 2001; Mohr *et al*. 1992, Tucker 1997). Mesenchymal tumours are more common then epithelial tumours. Squamous cell carcinomas and papillomas are the most commonly seen. Squamous cell carcinomas often start in the ear and spread to the head. Basal cell carcinomas and squamous cell carcinomas are locally invasive but rarely metastasise. Diagnosis is usually made by excisional biopsy.

*Formulary*

See p. 202, Chapter Sixteen on Chinchillas.

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