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# Chiroptera

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## INTRODUCTION

With over 1300 species identified, bats represent almost one quarter of the world's mammals (Fenton and Simons, 2014). Bats provide important environmental services, such as insect control, seed dispersal, and pollination. They inhabit a wide variety of ecological niches on all continents except Antarctica. Over 150 species are listed as endangered or vulnerable to extinction, primarily due to habitat degradation ([www.iucnredlist.org](http://www.iucnredlist.org)).

Despite their ubiquity and ecological importance, relatively little has been published on diseases of bats, while much has been written on bats' role as reservoirs in disease transmission. The decimation of certain bat populations in North America following the introduction of the fungus *Pseudogymnoascus destructans*, the cause of white-nose syndrome, has served to highlight both the importance and vulnerability of bats and has resulted in increased focus on disease threats to free-ranging bat populations.

In addition to the threat of infectious disease, the close proximity of bats to humans, including their use of human structures for hibernation and their role as a food source in many parts of the world, results in their susceptibility to anthropomorphic threats, such as trauma, toxicosis, and habitat loss. Ultimately, the interdependence of bats and humans means that an understanding of their health is critical for understanding and preserving human, domestic animal, and ecosystem health.

As many bats have multiple common names, the scientific names for species are used in this chapter.

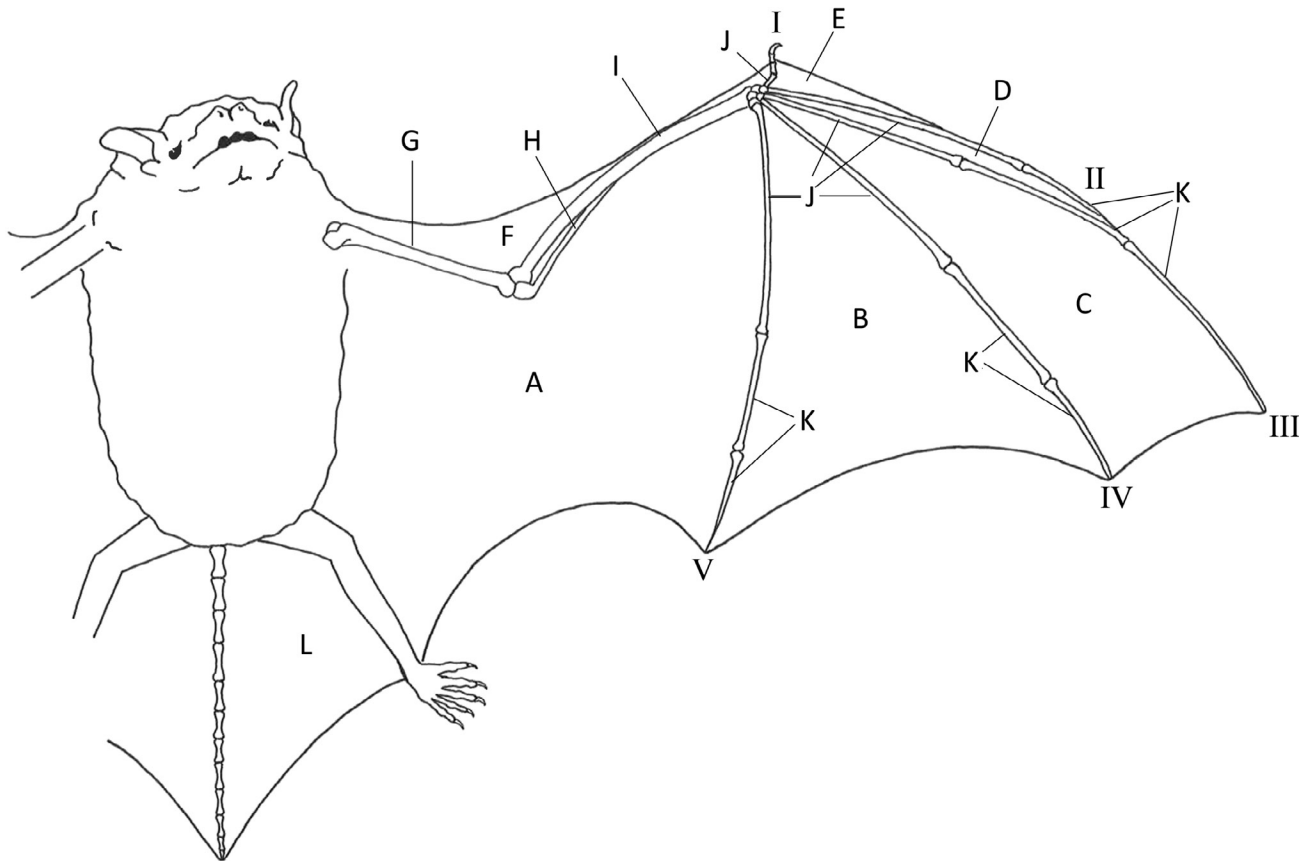
## UNIQUE FEATURES

Pteropodid bats (Old World fruit bats) commonly have a mild degree of erythrocyte polychromasia. Additionally, they can have faint magenta to basophilic cytoplasmic

inclusions in their neutrophils, representing variations in staining of the neutrophil granules (see Chapter 4).

Bats are the only mammals that exhibit true flight. Bats have greatly elongated digits that support the wing membrane, which is an extension of the skin of the dorsum and ventrum (Fig. 25.1) (Nowak, 1999). In pteropodid bats, the first digit is opposable, but it is largely or completely fixed in most of the remaining bat species (Altringham, 1996). The first digit has a claw in all bats, and the second one also has a claw in most pteropodid bats (Nowak, 1999; Vaughan, 1970). The hind limbs are rotated 90–180 degrees so that the stifle bends laterally or dorsolaterally (Altringham, 1996; Neuweiler, 2000). The radius is markedly elongated, longer than the humerus. The ulna is generally small, with the distal portion greatly reduced or absent (Vaughan, 1970; Walton and Walton, 1970). The fibula is often small and sometimes incomplete, and it is absent entirely in the family Nycteridae (Walton and Walton, 1970). The uropatagium is present between the hind limbs. The anatomy of this membrane varies between species, from a single membrane that completely spans the hind limbs to narrow strips of membrane on the medial aspect of the hind limbs (Schutt and Simmons, 1998). A bony or cartilaginous structure called the calcar, associated with the first row of tarsal bones, helps spread and support the uropatagium (Schutt and Simmons, 1998; Walton and Walton, 1970). The calcar generally articulates with the calcaneal tuberosity in most bat species but lacks this articulation in pteropodid bats; examined species of rhinopomatid and craseonycterid bats, as well as some phyllostomid and pteropodid bats, lack a calcar (Schutt and Simmons, 1998).

Histologically, the wing membrane (Fig. 25.2) is composed of two layers of stratified squamous epithelium separated by a thin scaffolding of collagen, linearly arranged elastin fibers, skeletal muscle, capillaries, and lymphatic



**FIGURE 25.1 Anatomy of the bat wing.** (A) Plagiopatagium. (B) Dactylopatagium major. (C) Dactylopatagium medius. (D) Dactylopatagium minus. (E) Dactylopatagium brevis. (F) Propatagium. (G) Humerus. (H) Ulna. (I) Radius. (J) Metacarpal bones. (K) Phalanges. (L) Uropatagium. (Illustration by A. Stahl after Hedenström, A., Johansson, L.C., *J. Exp. Biol.* 218, 653–663, 2015)



**FIGURE 25.2 Normal patagium, *Pteropus vampyrus*.** The patagium consists of two thin layers of epidermis separated by collagen, vessels, and bundles of elastic fibers.

vessels. The epidermis typically ranges from 1 to 3 cells thick and the stratum corneum is relatively thick in com-

parison to the epidermis. Melanocytes are present within and immediately subjacent to the epidermis and melanin granules can be found throughout all layers of the epidermis and stratum corneum (Makanya and Mortola, 2007; Quay, 1970). The superficial dermis is composed primarily of collagen with few elastic fibers, while the deep dermis contains dense bundles of elastic fibers (Holbrook and Odland, 1978). Only rare hair follicles and sebaceous glands are present in the patagia (Gupta, 1967). Musculature within the wing membrane includes large skeletal muscles that connect the skeleton to the wing membrane, as well as intrinsic muscles that originate and insert within the wing membrane itself (Swartz et al., 1996).

Many bats have extensive facial ornamentation and/or noseleaves, as well as folds and crenulations in the pinnae, to assist in emitting, amplifying, or receiving sounds for echolocation (Nowak, 1999). The pteropodid bats lack this ornamentation, as most species do not echolocate; only the cave-dwelling genus *Rousettus* has primitive echolocation abilities (Altringham, 1996).

Cardiac muscle is sometimes observed around pulmonary veins in the lungs of different bat species.

Bats have variable numbers of vertebrae: generally 7 cervical, 11–13 thoracic, 4–7 lumbar, 1–5 sacral, and 0–17 caudal (Walton and Walton, 1970). Specific vertebral formulae are reported for *Myotis* spp. [ $C_7T_{11}L_5S_4Cd_{10-11}$ ] (Vaughan, 1970; Walton and Walton, 1970), *Desmodus rotundus* [ $C_7T_{12}L_6S_5Cd_0$ , with fused sacral vertebrae (Greenhall et al., 1983)], and some *Pteropus* spp. [12–14 thoracic, 5–6 lumbar, and fused sacral vertebrae (Vogelnest and Allan, 2015; Walton and Walton, 1970)].

Dental formulas are widely varied over different chiropteran species, with adults having 20–38 teeth (Nowak, 1999). The dental formula for some common general/species are: *Pteropus*, *Rousettus*, and *Eidolon helvum*:  $2\left(\frac{I}{2}\frac{C}{1}\frac{P}{3}\frac{M}{3}\frac{2}{3}\right) = 34$  (DeFrees and Wilson, 1988; Nowak, 1999); *Desmodus rotundus*:  $2\left(\frac{I}{2}\frac{C}{1}\frac{P}{3}\frac{M}{0}\frac{0}{0}\right) = 20$  (Vaughan et al., 2011), and *Artibeus jamaicensis*:  $2\left(\frac{I}{2}\frac{C}{1}\frac{P}{2}\frac{M}{3}\frac{2}{3}\right) = 30$  (Handley et al., 1991).

In general, bats have a short gastrointestinal (GI) tract and rapid GI transit time, likely an adaptation for flight (Strobel et al., 2015). The stomachs are relatively simple in insectivorous bats but may be larger and more complex in some frugivorous and nectarivorous bats (Stevens and Hume, 1995). The stomach of *Desmodus rotundus* is elongated and tubular; the cardiac portion forms a blind sac for blood stodge (Greenhall et al., 1983; Stevens and Hume, 1995). The stomachs of multiple described pteropodid frugivorous (*Pteropus alecto*, *P. poliocephalus*, *Rousettus leschenaulti*, *Epomophorus wahlbergi*) and nectarivorous (*Eonycteris spelaea*) species have an elongated fundic cecum (Bhide, 1980; Perrin and Hughes, 1992; Tedman and Hall, 1985). A fundic cecum is not described in *Eidolon helvum*, so it does not appear to be a universal feature of pteropodids (DeFrees and Wilson, 1988; Okon, 1977). The intestine of bats is relatively short and generally lacks a cecum. Therefore, there is often no grossly discernible transition between the large and small intestine, although an increase in diameter may be present at the junction, or longitudinal folds may be present in the colon (Gadelha-Alves et al., 2008; Okon, 1977; Stevens and Hume, 1995). A few species, including *Megaderma spasma* and *Rhinopoma hardwickii*, have a cecum (Stevens and Hume, 1995).

The presence of male accessory sex glands varies among bat species. Ampullae are present in most bat species but lacking in some, including some pteropodids. Many species have seminal vesicles but they are reported to be absent in some species. The prostate and bulbourethral glands appear to be present in all chiropteran species where the reproductive tract has been described. Depending on the species, testes may be external (in a scrotum or a testicular pouch),

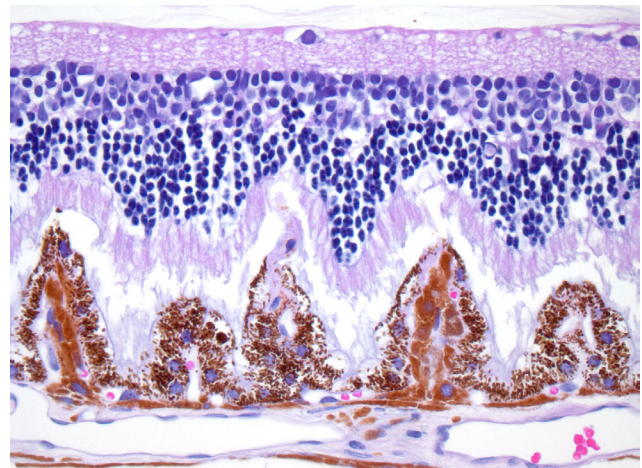
abdominal, or inguinal (Krutzsch, 2000). In some species, the location of the testes may change during mating season (Neuweiler, 2000).

Placentation varies with bat species. Most species have a discoidal and hemochorial or endotheliochorial placenta, although a few groups (Molossidae, Natalidae) have a diffuse endotheliochorial placenta in mid-pregnancy that becomes discoidal and hemochorial or endotheliochorial in late pregnancy (Badwaik and Rasweiler, 2000).

In frugivorous bats, hepatocytes frequently contain varying amounts of glycogen (Ben-Hamo et al., 2012), resulting in feathery vacuolation of the cytoplasm. The distribution of the glycogen is usually diffuse but it may sometimes have a more patchy or zonal appearance. Additionally, frugivorous bats of all ages often have prominent pancreatic islets, which are more numerous and often larger than in typical domestic mammals.

The eyes of pteropodid bats have choroidal papillae (Fig. 25.3). These are regularly-spaced, conical projections of the choroid that project into the thick retina and contain blood vessels, as the remainder of the retina is avascular (Bojarski and Bernard, 1988; Brudenall et al., 2007; Neuweiler, 2000).

Glandular sacs and sudoriferous or sebaceous glands have been identified in numerous species of bats, and may be identified on gross examination in some species. The location and appearance of the glands varies by species and sometimes with sex, age, and season. Glands may be present in the wing and tail membranes, ventral mandibular region, face, muzzle, lips, neck, chest, shoulders, and interscapular or subaxillary regions, as well as around the genitals or anus. The skin over these glands may be hairless or have clumped or discolored hair that may contain sticky secretions, and long and/or differently colored tufts of hair



**FIGURE 25.3** Normal retina, *Cynopterus brachyotis*. Pteropodid bats have choroidal papillae, regularly-spaced projections of choroid which contain blood vessels and project into the avascular retina.

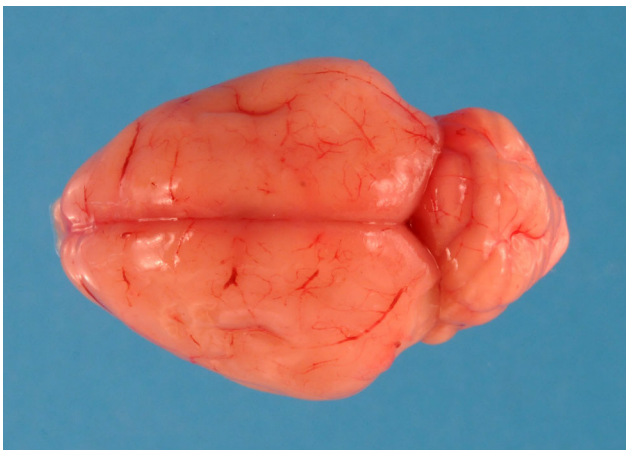
may be present over some glands or within the glandular sacs (Nassar et al., 2008; Quay, 1970).

The cerebrum of bats is generally lissencephalic with few shallow and often poorly developed gyri and sulci (Fig. 25.4) (Henson, 1970; Neuweiler, 2000).

## NON-INFECTIOUS DISEASES

### Nutritional

**Iron overload (IO)** has been reported in captive *Rousettus aegyptiacus* (Crawshaw et al., 1995; Farina et al., 2005; Leone et al., 2016). Overload is likely dietary, although the reason for the susceptibility of this species relative to other species on the same diet is unclear. Excess iron participates in the generation of free radicals, resulting in tissue damage (Takami and Sakaida, 2011). The liver is the primary target organ with excessive iron observed in portal macrophages, Kupffer cells and hepatocytes, as well as fibroblasts and bile duct epithelium. With chronicity, bats can develop portal to bridging fibrosis or cirrhosis (Fig. 25.5). Icterus and cavitory effusions can occur with liver failure (Crawshaw et al., 1995). Excessive iron stores are also identified in the spleen, and iron deposition has also been identified in the heart, skeletal muscle, pancreas, kidneys, and various other tissues. Hepatocellular carcinoma and cholangiocarcinoma can be seen in *R. aegyptiacus* with liver damage associated with IO. Myocardial degeneration and/or fibrosis is also common in *R. aegyptiacus* with IO (occur in about 45% of affected bats) but it is not correlated with the amount of iron present in the liver or heart (Leone et al., 2016). Myocardial fibrosis is also observed in older fruit bats of other species, albeit at a lower rate than *R. aegyptiacus*. It is not known whether myocardial fibrosis is related to IO or is simply common in this species, as a significant number of *R. aegyptiacus* without IO have not been examined. Iron overload with

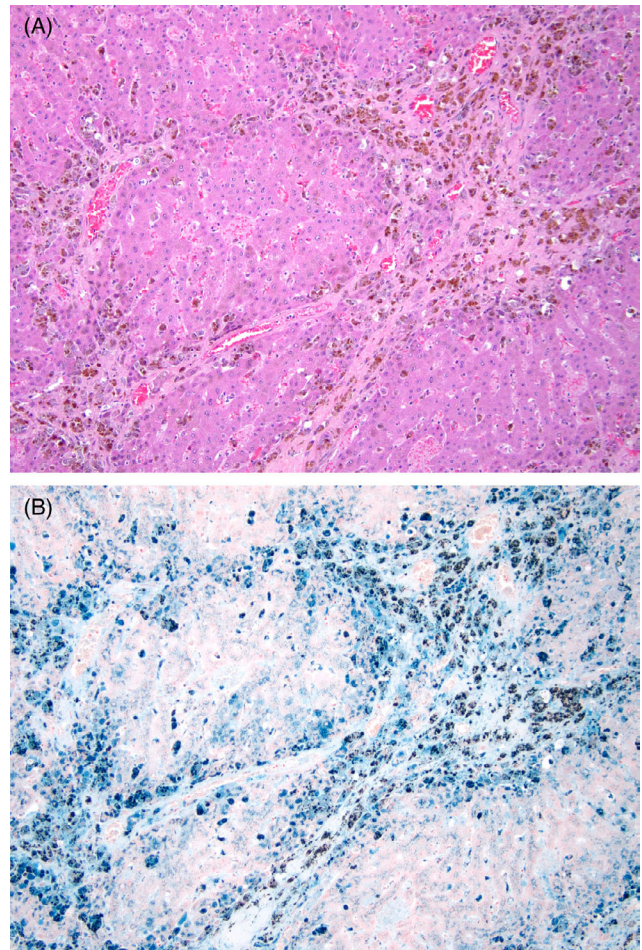


**FIGURE 25.4** Normal brain, *Pteropus hypomelanus*. The brain is largely lissencephalic, with rare shallow sulci. (Photo Courtesy of E. Kieran, University of Florida)

hepatic fibrosis has also been observed in *R. lanosus*. While other fruit bats including *Pteropus* spp. (Crawshaw et al., 1995), *Carollia perspicillata*, and *Cynopterus brachyotis* have been observed with varying amounts of hepatic iron, associated damage has not been identified.

**Hypovitaminosis C** was suspected in an 8-week-old hand-reared frugivorous *Pteropus pumilus* and a group of sanguinivorous *Desmodus rotundus*. The *Pteropus* presented with lethargy and joint enlargement that radiographically was associated with widening of the proximal physis and decreased bone opacity. In this case, the formula fed to the bat contained no vitamin C, and supplementation resolved clinical signs and radiographic abnormalities (Aitken-Palmer et al., 2012). In *Desmodus*, multiple bats developed wing hematomas and gingival bleeding that also resolved with supplementation (Hausmann et al., 2015).

**Vitamin B12 deficiency** has been induced in captive *Rousettus aegyptiacus* by feeding a diet consisting of



**FIGURE 25.5** Iron overload in the liver, *Rousettus aegyptiacus*. (A) Bridging fibrosis with iron overload. Abundant brown pigment is present within and at the margins of the connective tissue. (B) Abundant hemosiderin is present in macrophages at the margins of and within the fibrous connective tissue, with lesser amounts in hepatocytes, bile ducts, and Kupffer cells. Prussian blue.

washed, peeled bananas, oranges, pears, and papaya. After 200 days on this diet, bats became ataxic with hind limb proprioceptive deficits. Histologically, the spinal cord of the caudal cervical and cranial thoracic regions had patchy spongiosis in the lateral and ventrolateral white matter, consistent with early demyelination (Green et al., 1975). Lipid and fatty acid concentrations in the myelin of B12-deficient bats were subsequently determined to be different than in control bats; bats in this study were fed washed, peeled banana and papaya with a supplement containing vitamins A, C, and D and four B complex vitamins (B<sub>6</sub>, thiamin, riboflavin, niacin) (van der Westhuyzen et al., 1983). However, it should be noted that the experimental diets were likely deficient in multiple nutrients, including vitamin E, so reported nervous system lesions could have been the result of deficiencies other than vitamin B12 (E. Dierenfeld, personal communication).

## Metabolic

Varying degrees of **hepatic lipid deposition**, suspected to result from negative energy balance, have been observed in sick or injured free-ranging bats that have recently been admitted for rehabilitation (K. Rose, personal communication) and in captive bats. Hepatic lipidosis, along with lipid deposition in the renal tubules and cardiac myocytes, has been documented in obese wild-caught, captive-held *Rhinolophus ferrumequinum* fed mealworms in captivity; the high dietary fat content may have been contributory (Gozalo et al., 2005). Hepatic, renal tubular, and cardiomyocyte lipidosis with patagial ecchymoses, perirenal hemorrhage, and icterus has also been reported in a captive colony of *Eptesicus fuscus* secondary to decreased food intake associated with environmental disturbance, illness, and conspecific trauma (Snyder et al., 2015). In this group, it was unknown whether affected bats were overconditioned prior to the period of anorexia.

## Toxic

**Fluorosis** with hyperostosis in captive *Rousettus aegyptiacus*, *Pteropus giganteus*, and *P. poliocephalus* is associated with high dietary and elevated bone fluoride concentrations. Multifocal to coalescing asymmetric bony proliferations, which are periarticular but do not involve the joints (Fig. 25.6A), are most frequently identified on the wings, although lesions of the hind limbs, sternum, ribs, and mandible are sometimes seen. Histologically, there are subperiosteal nodules of new woven bone that are clearly demarcated from the underlying cortex (Fig. 25.6B), with some resorption in the underlying cortical bone (Duncan et al., 1996). Bony proliferation is common in captive bats as a result of trauma, particularly on the leading edge of the wing, so lesions must be differentiated via histopathology and clinical correlations.

**Pesticide residues**, particularly **organochlorines**, such as **DDE, DDT, lindane, and polychlorinated biphenyls**, have been detected in many species of free-ranging bats in North America, Britain, Europe, Australia, and Africa, with occasional documentation of population-level lethal and sublethal impacts (Clark, 2001; Clark and Shore, 2001; O'Shea et al., 2016, 2001; Zhang et al., 2009). Insectivorous bats are thought to be particularly susceptible to organochlorine exposure due to their higher trophic level in food webs, high metabolic rate, and long life span, as well as the periodic mobilization of adipose tissue during migration and hibernation, at which time lipophilic compounds stored in fat are released (Geluso et al., 1976). Due to their propensity to roost in manmade structures, bats are often exposed to antifungal and insecticide compounds in treated lumber. Bats of all life stages are at risk for intoxication, as some organochlorine pesticides have been shown to cross through the placenta and pass to young in milk (Clark and Shore, 2001).

Several species have been experimentally exposed to organochlorines at lethal or sublethal levels (Clark and Shore, 2001; Clark and Stafford, 1981). Pesticide-associated mortality has been widely documented; however, the effects of sublethal exposure are largely unknown. Some evidence suggests that sublethal exposure to organochlorines may increase metabolic rate, potentially interfering with adequate fat deposition for hibernation (Clark and Stafford, 1981; Swanepoel et al., 1999).

**Organophosphate toxicity** resulting in brain cholinesterase inhibition has been reported in insectivorous bats in Europe and North America (O'Shea and Clark, 2002). In one report, *Artibeus lituratus* exposed to fenthion developed hepatocellular cytomegaly and vacuolation, as well as increased muscle glycogen content (Amaral et al., 2012). Incoordination has been reported in *Eptesicus fuscus* and *Myotis lucifugus* exposed to acephate and methyl parathion (Clark and Shore, 2001).

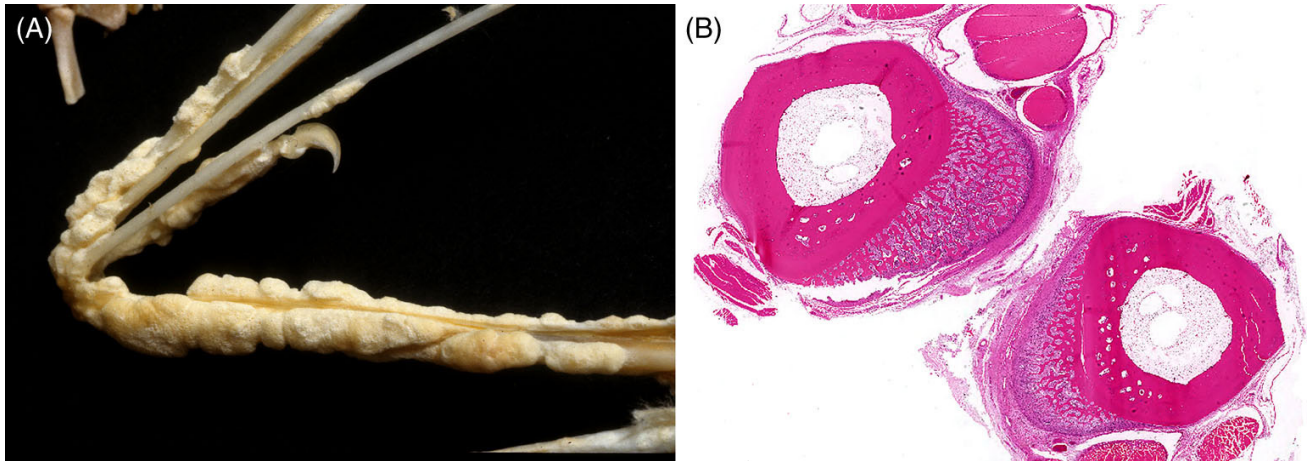
Additional toxicoses are reported in Table 25.1 and in the Supplemental Materials.

## Congenital

Rare congenital abnormalities have been identified in captive and free-ranging bats, including atresia ani, portosystemic shunts (Olsson and Woods, 2008), unilateral renal aplasia, and congenital cataracts. In multiple years, several colonies of *Pteropus conspicillatus* in Australia had multiple births with congenital defects, including cleft palate and/or other craniofacial abnormalities and deformities of the toes and claws; a toxin or infection during gestation is suspected (Olsson and Woods, 2008).

## Age-Related/Degenerative

**Myocardial fibrosis** is sometimes observed in captive frugivorous bats. Affected bats are generally older but lesions can be present in young adult bats. A particular pattern or distribution



**FIGURE 25.6** Fluorosis associated bony lesions. (A) Fluorosis in wing bones, *Roussettus aegyptiacus*. Nodular bony proliferations on the long bones with fluorosis. (B) Fluorosis in humerus, *Pteropus giganteus*. Histologically, nodular bony proliferations of fluorosis consist of subperiosteal trabeculae of woven bone, clearly demarcated from the underlying cortex. (Photos Courtesy of D. Smith, Toronto Zoo and M. Duncan, St. Louis Zoo)

**TABLE 25.1** Additional Toxicoses Reported in Chiroptera

Toxin	Species Affected	Clinical Signs	Lesions
Ivermectin (DeMarco et al., 2002)	Captive <i>Cynopterus brachyotis</i>	Fell from perches, minimal movement in all parts of body	Renal tubular necrosis with regeneration (uncertain if directly related to ivermectin)
<i>Rodenticides</i>			
Diphacinone (Dennis and Gartrell, 2015)	Free-ranging <i>Mystacina tuberculata</i>	Lethargy, death	Multisystemic hemorrhage
Zinc phosphide (Hurley and Fenton, 1980)	Free-ranging <i>Myotis lucifugus</i>	Tremors, death	Pathology not described
<i>Heavy metals</i>			
Lead (Hariono et al., 1993; Skerratt et al., 1998; Sutton and Wilson, 1983; Zook et al., 1970)	Captive and free-ranging <i>Pteropus</i> spp.	Incoordination, inability to fly, aggression, muscle tremors, ptyalism, ataxia, separation from group, target of conspecific aggression	Renal tubular necrosis, proximal tubular karyomegaly and nuclear pleomorphism; variably acid fast eosinophilic intranuclear inclusion bodies in the proximal renal tubular epithelium and occasionally in hepatocytes
Copper (Hoenerhoff and Williams, 2004)	Captive <i>Artibeus jamaicensis</i>	Death without prior clinical signs	Random acute hepatocellular necrosis with abundant copper pigment in hepatocytes and macrophages, hemoglobin casts in renal proximal tubules with tubular degeneration

for lesions has not been observed. Overall cardiomegaly and chamber dilation (most often the right or both ventricles) may be present; bats rarely have left atrial mural thrombi. Affected bats sometimes have pathologic evidence of heart failure, including pulmonary edema and intra-alveolar hemosiderin-laden macrophages (heart failure cells). Bats with heart failure may have dependent, subcutaneous edema of the head and neck; similar cranial edema may also be observed in bats with protein-losing nephropathy and sepsis. Several causes for myocardial fibrosis have been investigated. These include hypovitaminosis E in *Pteropus* spp. (Heard et al., 1996) and

iron overload in *Roussettus aegyptiacus* (Leone et al., 2016); however, not all bats with myocardial fibrosis have these conditions. Further studies into etiologies of myocardial disease in bats are needed.

In older captive insectivorous and carnivorous bats, **dental disease** is frequently a significant problem. Tooth loss, plaque, tartar, gingivitis, periodontitis, and osteomyelitis can be seen (Olsson and Woods, 2008). Similar lesions are observed sporadically in captive frugivorous bats. Enamel defects and staining of the teeth are common in pteropodid bats (Olsson and Woods, 2008). Traumatic damage has been

associated with secondary gingivitis, pulpitis, and tooth root abscesses in captive pteropodid bats. Significant dental attrition is common in older frugivorous bats but does not appear to cause difficulty with feeding.

Unilateral or bilateral **testicular degeneration**, atrophy, and/or fibrosis have been observed in older captive pteropodid bats.

**Degenerative joint disease** is not uncommon in older captive pteropodid bats; it also occurs in other captive bat species. Hip, stifle, interphalangeal, and metacarpophalangeal joints are most frequently affected.

**Vertebral spondylosis** is sometimes seen in older captive pteropodid bats, and can affect the cervical, thoracic, and/or lumbar regions.

## Trauma

**Trauma** is a common cause of death in free-ranging bats (Mühldorfer et al., 2011a,b). **Wind turbine interactions** account for the highest number of traumatic mortality events in bats worldwide (O'Shea et al., 2016). Migratory species that roost in trees are disproportionately affected, although high mortality can occur in nonmigratory species (Cryan and Barclay, 2009; Grodsky et al., 2011). Barotrauma was initially proposed as the cause of death in many bats due to reports of intrathoracic and intra-abdominal hemorrhage with a relative lack of external injuries. Other findings supportive of barotrauma included pulmonary lesions such as alveolar bullae, hemorrhage, congestion, edema, and atelectasis (Baerwald et al., 2008). Subsequent studies have concluded that injuries are more compatible with blunt trauma (Fig. 25.7). Commonly cited injuries include wing fractures, external lacerations, inguinal and diaphragmatic hernias, subcutaneous hemorrhage, and bone marrow emboli, although rupture of the tympanic membrane in the absence of associated mechanical injuries has been reported in a small subset (6%) of affected bats (Grodsky et al., 2011; Rollins et al., 2012). It is likely that both blunt force trauma and decompression play a role in wind turbine-related bat mortality (see Chapter 2).

**Cat predation** is frequently documented, particularly in species roosting in buildings. Sequelae common to cat predation include wing lacerations and fractures, soft tissue injury including subcutaneous and intramuscular hematomas, and *Pasteurella multocida* infection (see below) (Mühldorfer et al., 2011a,b).

Free-ranging pteropodid bats are highly susceptible to **entanglement injuries** sustained from netting improperly installed around fruit trees (K. Rose, personal communication).

Other reported causes of trauma in free-ranging bats include vehicular strike, collision with aircraft, building collision, and trauma secondary to entrapment in confined spaces (Mühldorfer et al., 2011a; O'Shea et al., 2016).

**Electrocution** from power lines or electric fences has been observed in free-ranging pteropodid bats in Australia.



**FIGURE 25.7** Wind turbine trauma, *Nyctalus noctula*. Hemorrhage and abdominal hernia with prolapse of the stomach through the left abdominal wall secondary to wind turbine trauma. (Photo Courtesy of G. Wibbelt, Leibniz Institute for Zoo and Wildlife Research (IZW), Berlin, Germany)

Acutely, there may be no lesions, or only charring of the fur or skin or a burnt smell may be identified. Approximately 2–4 days after electrocution, the skin distal to electrocution becomes moist and friable and cutaneous defects appear, most commonly on the wings. In some cases, a fine linear pattern of necrosis appears to represent electric current entry and exit sites (Fig. 25.8). Histologically, these lesions consist of fibrinoid degeneration of blood vessels with epidermal, dermal, and adnexal coagulative necrosis. Secondary bacterial and fungal infection of these wounds is common. Edema of the meninges and brain is also observed in some acutely affected bats (K. Rose, personal communication). Bats that recover from burns may have depigmentation, scarring, and alopecia of affected areas. Pups on dams that are electrocuted may survive due to the insulation provided by fur, but they may have oral burns sustained via their attachment to the teat (Olsson and Woods, 2008).

Trauma is also common in captive bats. Trauma to the wings, typically a result of wire enclosures, is frequently observed, and wounds often have colonization with mixed bacteria, and sometimes fungi. Ulceration and hemorrhage can be associated with traumatic removal of metal identification bands; fatal hemorrhage associated with traumatic band removal can occur. As there is minimal soft tissue covering the wing bones, fractures, most often of digits, can occur, and bony proliferation and/or osteomyelitis may be sequelae to wing trauma.

Pteropodid bats tend to have **extensive granulation tissue associated with chronic wounds**. Fibroblasts can be large with some cellular atypia, and the classic vascular arrangement of granulation tissue may not always be apparent, especially in the patagium. Lesions may sometimes be relatively discrete and nodular, and infiltration of normal structures by fibroblasts is occasionally observed (Fig. 25.9). Some lesions have been diagnosed as sarcomas and have





**FIGURE 25.8** Electrocution injury of the patagium, *Pteropus poliocephalus*. Linear pattern of patagial necrosis with full-thickness defects, consistent with electrocution injury. (Photo Courtesy of K. Rose, Australian Registry of Wildlife Health, Taronga Conservation Society, Australia)

subsequently regressed, so circumspection is needed in diagnosing cutaneous spindle cell proliferations in bats.

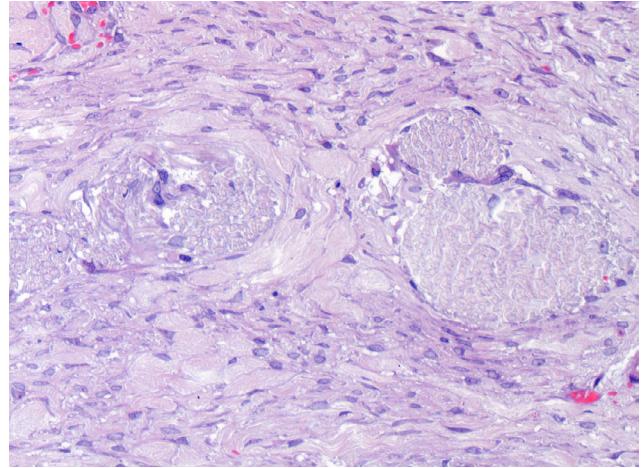
**Bite wounds** from conspecifics are also common in captive bats, and may result in cellulitis or abscesses. Spinal fractures and luxations, liver lacerations, and hemorrhage have been observed from presumed falls in captive pteropodid bats.

### Inflammatory Non-infectious

**Eosinophilic dermatitis** has been observed in captive *Pteropus* spp. In addition to eosinophils, mixed to granulomatous inflammation is present. Ulceration is common in affected skin, and collagen degeneration is sometimes observed. Some lesions represent insect bite reactions or allergic dermatitis. Eosinophilic dermatitis in the lip of a captive *Pteropus poliocephalus* was associated with chronic ulceration and extensive collagen degeneration. The lesions were similar to collagenolytic dermatitis in cats (K. Rose, personal communication).

### Miscellaneous

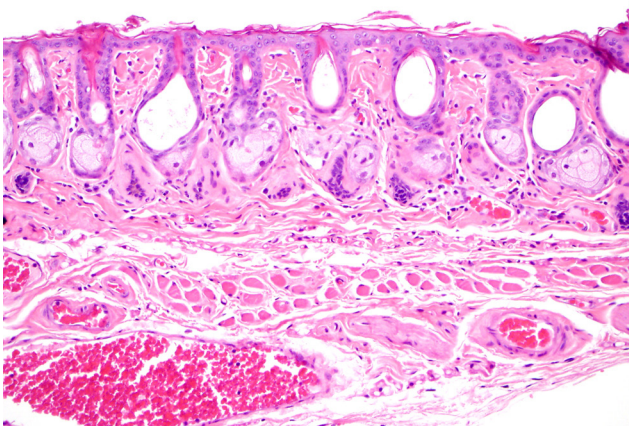
**Alopecia** of the chest, abdomen, and occasionally the back has been reported in 5%–5.6% of free-ranging *Artibeus*



**FIGURE 25.9** Extensive granulation tissue and fibroplasia in the patagium, *Pteropus vampyrus*. An irregular mass of fibroblasts expands the patagium and infiltrates into and around elastin bundles. Anisocytosis, anisokaryosis, and multinucleation are observed in some of the fibroblasts. While this lesion was initially diagnosed as a fibrosarcoma, the lesion subsequently regressed, suggesting that this was reactive fibroplasia.

*jamaicensis* and *A. lituratus* in Mexico. Lesions are more prevalent in the dry season and in urban areas, and more females than males may be affected. Histologically, there is loss of some hair follicles with rare catagen follicles. Nutritional or endocrine etiologies are considered possible differentials (Bello-Gutiérrez et al., 2010). Alopecia is also seen in captive *A. jamaicensis* at multiple institutions. Males appear to be most affected in some groups, while females are primarily affected in others. Symmetrical hair loss typically starts on the dorsum or ventrum, extends to the limbs and sometimes affects the entire body. Histologically, there is dilation of follicular infundibula with attenuation of the infundibular epithelium, tricholemmal keratinization, and follicular atrophy/loss with retention of sebaceous glands (Fig. 25.10). A cause for these lesions has not been determined, although endocrine, metabolic, or nutritional etiologies have been considered (K. Terio and M. Garner, personal communication). Alopecia has also been reported in captive bats due to inadequate dietary protein or fat-soluble vitamins, or feeding infant formula to bats past their normal weaning age (Olsson and Woods, 2008).

**Depigmentation** can be observed in bat skin, both as a primary process and as a secondary lesion in healing wounds. Gross lesions of primary depigmentation (vitiligo) have been described in captive bats in a group of *Pteropus vampyrus* and *P. hypomelanus* (Stringer and Larsen, 2013), a group of *Rousettus lanosus* (M. Garner, personal communication) and a single captive *Pteropus hypomelanus*. Lesions are typically patchy on the wings or more generalized on the face, body, and limbs. Histologically, there is loss of the majority of epidermal melanin from affected areas but no inflammation. Nutritional



**FIGURE 25.10 Alopecia, *Artibeus jamaicensis*.** Histologic lesions include dilation of follicular infundibula, thinning of the infundibular epithelium, follicular atrophy and loss, and tricholemmal keratinization. Sebaceous glands are retained. (Photo Courtesy of K. Terio, University of Illinois Zoological Pathology Program)

causes have been proposed but the etiology of these lesions has not been determined.

**Proliferative endometrial lesions** have been observed in *Carollia perspicillata* in multiple zoo collections. Gross lesions include abdominal distention, marked uterine enlargement, and serosanguinous or pseudomembranous material in the uterine lumen. Histologically, there is multifocal to diffuse proliferation of glandular epithelium and stromal cells, with occasional mucosal epithelial erosion (Napier et al., 2009).

**Choleliths** are occasionally observed in the gall bladder or bile duct of *Pteropus* spp. In some cases, they are incidental findings, but they may be associated with cholangitis/cholangiohepatitis and intrahepatic cholestasis.

**Adrenal gland osseous metaplasia** has been observed in a few captive *Pteropus* spp., with mature bone and adipose tissue in the glands histologically.

## Miscellaneous

**Frostbite** has been observed in captive pteropodid bats. Vasoconstriction with decreased blood flow typically causes necrosis of the distal extremities, in particular the nail beds and wing tips (Fleming and Heard, 2001). Frostbite has been proposed as the cause of the so-called “square-eared anomaly” reported in free-ranging North American species of *Myotis*. Gross lesions are limited to the pinna, the height of which may be reduced by up to 50%. Microscopically, the edges of affected pinnae have a thinned epidermis, dermal granulation tissue and fibrosis, and disrupted, hypertrophic, and dystrophic cartilage consistent with frostbite (Kurta and Kwiecinski, 2007).

Multiple large-scale mortality events, with increasing numbers in recent years, have been associated with **high**

**ambient temperatures** in free-ranging pteropodid bat species in Australia and India (O’Shea et al., 2016; Welbergen et al., 2008).

**Iatrogenic thermal burns**, most commonly in the patagium, can occur in captive bats due to contact with heat lamps, hot packs, or heating pads/blankets (Heard, 1999).

**Rectal prolapses** can occur secondary to constipation (usually due to feeding fruit bats a blended diet), diarrhea, colitis, and/or intestinal parasites (Olsson and Woods, 2008).

## Neoplastic

A wide variety of neoplasms, including various soft tissue sarcomas, lymphoma, oral and nasal squamous cell carcinoma, cutaneous melanocytoma, osteosarcoma, and mammary adenomas and carcinomas, have been observed in captive bats. Most published case reports of neoplasia represent single neoplasms, with *Rousettus aegyptiacus* the most common species in these reports. The most commonly reported neoplasms in captive *R. aegyptiacus* are hepatocellular carcinoma and cholangiocarcinoma, thought to be associated with liver damage from iron overload (Leone et al., 2016).

A novel papillomavirus in a 5-year-old captive *Rousettus aegyptiacus* was associated with the development of multiple cutaneous papillomas and basosquamous/squamous cell carcinomas. Positive intranuclear immunostaining using antibodies against bovine papillomavirus identified viral antigen within the neoplasms (McKnight et al., 2006).

A subcutaneous interscapular leiomyosarcoma associated with a microchip was identified in a captive *R. aegyptiacus*. Neoplastic cells were elongate and arranged in bundles and streams, with moderate anisocytosis and marked anisokaryosis. At necropsy, metastases were identified in the liver and peritoneum (Siegal-Willott et al., 2007).

Cutaneous spindle-cell sarcomas have been seen in captive bats; however, it is important to note that pteropodid bats can have nodular fibroplasia with some cellular atypia as a sequela of trauma. Some lesions that have been diagnosed as sarcomas in the skin have spontaneously regressed (see Trauma).

## INFECTIOUS DISEASES

Bats are host to many infectious organisms. Viral diseases of humans or other animal taxa for which bats are a proven or likely reservoir are summarized in Table 25.2. Many of these viruses are of OIE/WHO importance. A number of molecular, serologic, and other surveys of infectious organisms in various bat species have been performed due to recognition of the role bats play as reservoirs for zoonotic pathogens (Brook and Dobson, 2015). These surveys have identified numerous viruses, including adenoviruses, astroviruses, bunyaviruses, circoviruses, coronaviruses, hepadnaviruses,

**TABLE 25.2** Viruses Associated with Natural Disease in Humans and Animals for Which Bats are a Proven or Likely Reservoir

Disease	Species Clinically Affected	Main Bat Reservoir Species	Geographic Locations of Disease (Imported Diseases Reported as Country Where Infection Acquired)	References
<i>Bunyaviruses</i>				
Kasokero virus	Human (laboratory-acquired infection)—flu-like symptoms, abdominal pain	<i>Rousettus aegyptiacus</i>	Uganda	Kalunda et al. (1986)
Hantavirus	Human—Hemorrhagic fever with renal syndrome (flu-like symptoms, hypotension, renal disease), and hantavirus pulmonary syndrome (flu-like symptoms, pulmonary edema, shock)	<i>Eptesicus serotinus</i> , <i>Diphylla ecaudata</i> , <i>Anoura caudifer</i> , <i>Nycteris hispida</i> , <i>Neoromicia nana</i> , <i>Rhinolophus</i> spp., <i>Pipistrellus abramus</i> , <i>Hipposideros pomona</i>	South Korea, Brazil, Sierra Leone, Côte d'Ivoire, China, Vietnam	Simmons and Riley (2002); Zhang et al. (2014)
<i>Coronaviruses</i>				
Severe acute respiratory syndrome-related coronavirus (SARS-CoV)	Human—flu-like symptoms, diarrhea, death	<i>Rhinolophus</i> spp.	China (no reports since 2003)	Drexler et al. (2014); Hui et al. (2014)
Middle East respiratory syndrome coronavirus (MERS-CoV)	Human—flu-like symptoms, acute renal failure, death	<i>Taphozous perforatus</i>	Saudi Arabia, Jordan, United Arab Emirates, Qatar	Mackay and Arden (2015)
<i>Filoviruses</i>				
Ebola virus	Human—flu-like symptoms, abdominal pain, skin rash, encephalopathy, hemorrhagic symptoms, death Non-human primates—lethargy, abdominal pain, anorexia, hemorrhage, death Duiker—death (no pathology performed)	<i>Hypsignathus monstrosus</i> , <i>Epomops franqueti</i> , <i>Myonycteris torquata</i> suspected	Sudan, Democratic Republic of Congo, Uganda, Republic of Congo (not including disease known to be transmitted from non-human primates)	Ansari (2014); Burd (2015); Formenty et al. (1999); Ikegami et al. (2002); Leroy et al. (2009); Leroy et al. (2005); Olival and Hayman (2014); Rouquet et al. (2005)
Marburg virus	Human—flu-like symptoms, nausea, vomiting, diarrhea, pharyngitis, dysphagia, skin rash, encephalitis, hemorrhagic manifestations, death	<i>Rousettus aegyptiacus</i>	Zimbabwe, Kenya, Democratic Republic of Congo, Angola, Uganda	Amman et al. (2015); Brauburger et al. (2012); Olival and Hayman (2014)
<i>Lyssaviruses</i>				
Rabies virus (OIE listed for multiple species in 2018)	Most mammals at risk, rarely reported in poultry—progressive neurologic deterioration, coma and death	Multiple, including Vespertilionidae, Molossidae, Phyllostomidae, Mormoopidae, Noctilionidae, and Emballonuridae – especially important are <i>Eptesicus fuscus</i> , <i>Lasiurus noctivagans</i> , <i>Perimyotis subflavus</i> , <i>Tadarida brasiliensis</i> , and <i>Desmodus rotundus</i>	Global; only found in bats in North, Central, and South America	Banyard et al. (2014); Constantine et al. (2009)

**TABLE 25.2** Viruses Associated with Natural Disease in Humans and Animals for Which Bats are a Proven or Likely Reservoir (*cont.*)

Disease	Species Clinically Affected	Main Bat Reservoir Species	Geographic Locations of Disease (Imported Diseases Reported as Country Where Infection Acquired)	References
Australian bat lyssavirus	<i>Pteropus</i> spp., <i>Saccolaimus flaviventris</i> , human, horse—neurologic disease similar to rabies virus infection	<i>Pteropus</i> spp., <i>Saccolaimus flaviventris</i>	Australia	Barrett et al. (2005); Francis et al. (2014); Shinwari et al. (2014)
European bat lyssavirus-1	<i>Eptesicus serotinus</i> , <i>E. isabellinus</i> , human, domestic cat, stone marten ( <i>Martes foina</i> ), domestic sheep—neurologic disease similar to rabies virus infection	<i>Eptesicus serotinus</i> , <i>E. isabellinus</i>	Europe, especially the Netherlands, Germany, Denmark, Poland, France, and Spain	Aréchiga Ceballos et al. (2013); Calisher et al. (2006); Dacheux et al. (2009); Schatz et al. (2013)
European bat lyssavirus-2	<i>Myotis</i> spp., human—neurologic disease similar to rabies virus infection	Multiple <i>Myotis</i> spp., especially <i>M. daubentonii</i>	Europe, especially United Kingdom and the Netherlands, also detected in Germany, Finland, and Switzerland	Aréchiga Ceballos et al. (2013); Calisher et al. (2006); Jakava-Viljanen et al. (2015); Nathwani et al. (2003); Schatz et al. (2013)
Duvenhage virus	Human—neurologic disease similar to rabies virus infection <i>Miniopterus schreibersii</i> (presumptive identification)—caught during the day by a cat	Unknown, has been isolated from a clinically normal <i>Nycteris thebaica</i> in Zimbabwe	South Africa, Kenya	Banyard et al. (2011); Meredith et al. (1971); Paweska et al. (2006); Swanepoel et al. (1993)
Irkut virus	Human—neurologic disease similar to rabies virus infection	<i>Murina leucogaster</i>	China	Banyard et al. (2014); Li et al. (2014)
Lagos bat virus	<i>Epomophorus wahlbergi</i> —found sick or dead, sudden death Cat—clinically rabid or lethargy and paresis without aggression Clinically rabid: dog, pet <i>Rousettus aegyptiacus</i> in France (ex-Egypt or Togo), <i>Epomophorus wahlbergi</i> , water mongoose ( <i>Atilax paludinosus</i> )	Unknown, has been isolated from clinically normal/found dead/unknown history <i>Eidolon helvum</i> in Nigeria, Kenya, Ghana, and Senegal; <i>Micropterus pusillus</i> (unknown history) in Central African Republic; <i>Nycteris gambiensis</i> (unknown history) in Guinea; healthy <i>R. aegyptiacus</i> in Kenya; healthy <i>Epomophorus gambianus</i> and <i>Epomops buettikoferi</i> in Ghana	South Africa, Zimbabwe, Ethiopia, Egypt or Togo	Banyard et al. (2011); Freuling et al. (2015); Markotter et al. (2006a,b); Meredith and Standing (1981); Swanepoel et al. (1993)
<i>Paramyxoviruses</i>				
Hendra virus	Horse—respiratory or neurologic signs Human—encephalitis, flu-like pulmonary syndrome, death	<i>Pteropus</i> spp.	Australia	Clayton et al. (2013); Halpin et al. (2011); Ksiazek et al. (2011); Wild (2009); Wong and Ong (2011)

(Continued)

**TABLE 25.2** Viruses Associated with Natural Disease in Humans and Animals for Which Bats are a Proven or Likely Reservoir (*cont.*)

Disease	Species Clinically Affected	Main Bat Reservoir Species	Geographic Locations of Disease (Imported Diseases Reported as Country Where Infection Acquired)	References
Menangle virus	Pig—sow infertility, embryonic death, stillborn pigs, abortion, skeletal and craniofacial deformities, pulmonary hypoplasia, brain/spinal cord degeneration Human—flu-like illness, skin rash	<i>Pteropus</i> spp. (neutralizing antibodies in multiple species, virus isolated from <i>P. alecto</i> )	Australia	Barr et al. (2012); Chant et al. (1998); Love et al. (2001); Philbey et al. (2008); Philbey et al. (2007)
Nipah virus	Pig and human—respiratory or neurologic disease, death Dog—fever, respiratory distress, mucopurulent oculonasal discharge, death Cat—generalized vasculitis, clinical signs not reported Horse—meningoencephalitis	<i>Pteropus</i> spp.	Malaysia, Bangladesh, Singapore, India	Clayton et al. (2013); Halpin et al. (2011); Hooper et al. (2001); Wild (2009)
<i>Reoviruses</i>				
Nelson Bay virus	Human—fever, acute respiratory disease	<i>Pteropus</i> spp.	Malaysia, Indonesia	Chua et al. (2007, 2008); Yamanaka et al. (2014)

herpesviruses, papillomaviruses, paramyxoviruses, parvoviruses, picornaviruses, polyomaviruses, retroviruses, and rotaviruses, but have not demonstrated any associated disease in bats or other species (Chen et al., 2014; Drexler et al., 2011; Wu et al., 2016). The discussion that follows focuses on notable infectious organisms reported to cause disease in bats.

## DNA Viruses

**Poxviral** infection in free-ranging *Eptesicus fuscus* is associated with joint swelling, necrosuppurative osteomyelitis, necrotizing tenosynovitis, and occasional vasculitis involving long bones and flat facial bones. Virus can be detected in the synovium by PCR and electron microscopy. Genetic analysis identified the virus as a new genus of poxvirus (proposed name: Eptesipox virus), most closely related to Cotia virus, an unclassified chordopoxvirus isolated from mice in Cotia County, São Paulo State, Brazil, in 1961 (Emerson et al., 2013). A more typical cutaneous poxviral nodule was reported in a single free-ranging *Miniopterus schreibersii* in Australia. Microscopically, the lesion was typical of poxviral infection and characterized by focal

epidermal thickening with ballooning degeneration and intraepithelial, eosinophilic intracytoplasmic inclusion bodies. Pox virions were confirmed by electron microscopy (McLelland et al., 2013).

**Cytomegalovirus** has been detected in the acinar cells of the principal submandibular salivary gland of two captive *Myotis lucifugus*. Although cytomegaly was evident microscopically, there was no evidence of clinically significant disease (Tandler, 1996).

A novel **gammaherpesvirus** has been identified in a captive *Pteropus vampyrus* with chronic lymphoplasmacytic blepharitis, Meibomian gland adenitis, and neutrophilic and lymphocytic conjunctivitis; a relationship between the virus and lesions has not been confirmed (Brock et al., 2013).

## RNA Viruses

In addition to their notoriety as reservoirs of **rabies virus** and other zoonotic **lyssaviruses**, bat species are susceptible to fatal lyssaviral infection. Lyssaviruses have high host specificity such that different variants of the same species of virus will infect a particular species of bat (Constantine, 1993). In addition to the lyssaviruses included in Table 25.2, recently

identified bat lyssaviruses include **Bokeloh bat virus**, **West Caucasian bat virus**, **Aravan virus**, **Khujand virus**, and the tentatively named **Lleida bat lyssavirus** in Europe and Asia (Aréchiga Ceballos et al., 2013; Gunawardena et al., 2016). Further research is needed on these newly described viruses to determine their potential pathogenicity to bats and other species.

While rabies virus is endemic in bat populations, estimates of subclinical infection vary from 0.1% to 2.5% in healthy bats. Prevalence is much higher in sick bats, with estimates ranging from 10% to 92% of grounded *Tadarida brasiliensis* (Constantine, 1993; Davis et al., 2012). A high seroprevalence rate has been detected in apparently healthy bats, suggesting that they may be able to survive infection (Allendorf et al., 2012). Bats typically undergo a 2–25 week incubation period, although prolonged incubation of over 1 year has been reported (Brass, 1994). Cold temperatures inhibit disease progression in cold-adapted (i.e., hibernating) bats (Constantine and Blehert, 2009). Virus may be present in saliva for up to 12–15 days before onset of clinical signs, and most bats die within 1–2 weeks of developing clinical signs (Constantine and Blehert, 2009; Heard, 2003). Although direct contact with saliva, as through a bite wound, is typically required for transmission, both bats and humans can be infected by aerosolized virus (Constantine, 1993; Davis et al., 2007).

Bats more commonly display paralytic rather than furious clinical signs and are rarely reported to attack humans unless provoked. Clinical signs typically include neurologic deficits, such as paralysis, incoordination, disorientation, irritability, and increased vocalization (Constantine, 1993).

Microscopic lesions reported in bats with lyssaviral infection are similar to those reported in other mammalian species, including nonsuppurative meningoencephalomyelitis and ganglioneuritis. In a report of rabies infection in five *Molossus molossus* in Brazil found during the day unable to fly, the presence of rabies virus was confirmed by immunohistochemistry, direct immunofluorescence, and mouse inoculation. However, no microscopic lesions were present, indicating that lack of histologic lesions may not rule out lyssaviral infection in bats (de Araújo et al., 2014). Neuronal intracytoplasmic eosinophilic inclusion bodies known as Negri bodies are typical, but their presence and frequency are highly variable. In one study of bats infected with Australian bat lyssavirus, the highest frequency and severity of microscopic lesions was detected in the hippocampus, thalamus and midbrain, and medulla oblongata and pons (Hooper et al., 1999). Lyssaviral antigen has been detected in bats in the central and peripheral nervous system as well as in other tissues; reported affected sites include brainstem, cerebellar Purkinje cells, hippocampus, cerebrum, thalamus and midbrain, peripheral autonomic ganglia, nerve plexuses of the gastrointestinal tract, intramuscular nervous tissue, adrenal medulla, salivary gland, tongue, brown fat, lung, heart,

kidney, bladder, stomach, intestine, and feces (Allendorf et al., 2012; Davis et al., 2013; de Araújo et al., 2014; Hooper et al., 1999; Schatz et al., 2014; Stein et al., 2010).

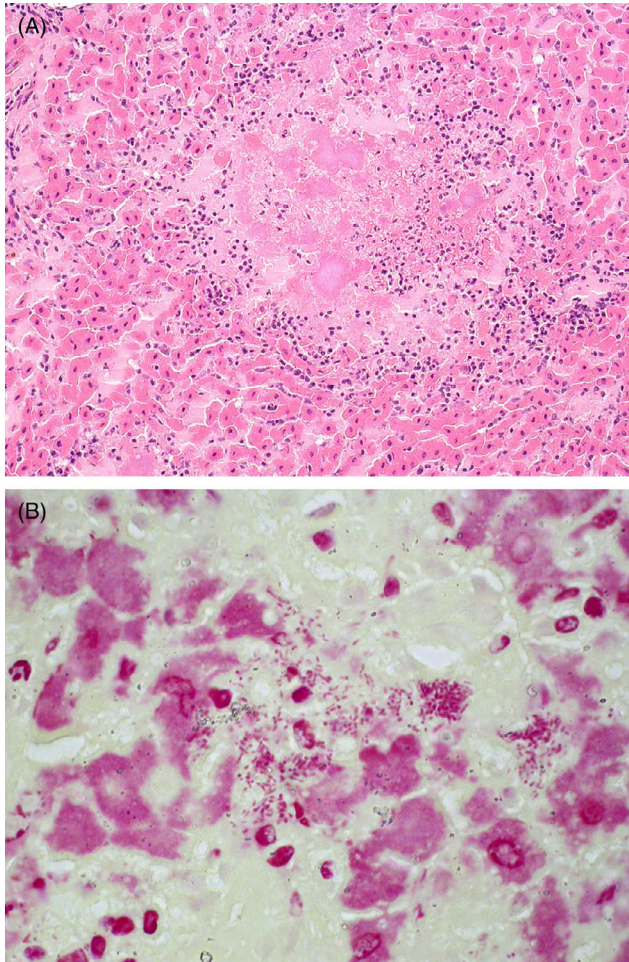
**Tacaribe virus**, an arenavirus, has been reported in several *Artibeus* spp. in Trinidad. Clinical signs mimic rabies and include unkempt haircoat, isolation from other bats, and tremors (Downs et al., 1963). Although a histologic review of naturally infected bats has not been performed, experimental inoculation of *A. jamaicensis* and *Carollia perspicillata* with Tacaribe virus resulted in multisystemic lesions including interstitial pneumonia, hepatocellular degeneration and necrosis, neutrophilic splenitis, and lymphocytic leptomeningitis or encephalitis. Development of significant clinical disease in these species may suggest they are unlikely to be reservoirs for Tacaribe virus (Cogswell-Hawkinson et al., 2012).

## Bacteria

*Yersinia pseudotuberculosis* infections have been described in captive *Pteropus rodricensis* and *Rousettus aegyptiacus*, and in a free-ranging *Myotis myotis* in Germany (Childs-Sanford et al., 2009; Mühldorfer et al., 2010; Nakamura et al., 2013; Williams, 2004). In *R. aegyptiacus*, gross lesions include splenomegaly, hepatomegaly, mesenteric lymphadenopathy, and white to yellow-white nodules in the spleen, liver, kidneys, and lung. Histologic lesions include abscesses or neutrophilic to necrosuppurative inflammation in the liver, spleen, kidney and lung; bacteria within renal and pulmonary blood vessels; necrosis of the bone marrow and Gram-negative bacilli within lesions (Fig. 25.11) (Childs-Sanford et al., 2009; Nakamura et al., 2013).

Infections with ***Pasteurella*-like bacteria** have been described in captive *Epomophorus wahlbergi* from one zoo collection with unilateral or bilateral pneumonia, pleuritis (Helmick et al., 2004), pleuropneumonia, and/or suppurative or fibrinous epicarditis/pericarditis ± myocarditis with intralesional Gram-negative pleomorphic coccobacilli. In some of these bats, there was prominent type II pneumocyte hyperplasia and hyperplasia of bronchial epithelium, suggesting that there could have been additional pathogens. Two *Pteropus pumilus* from the same institution had similar bacteria cultured from hind limb cellulitis and fracture with abscess formation in a wing digit; the respiratory and cardiac lesions seen in the *E. wahlbergi* were not identified in these bats (Helmick et al., 2004).

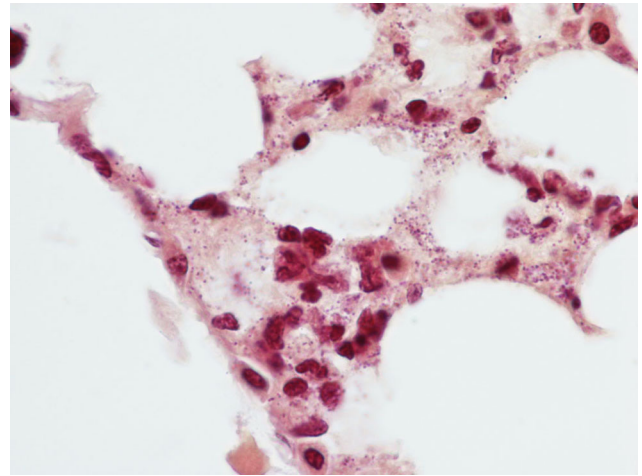
A mortality event involving approximately 100 free-ranging *Eptesicus fuscus* in Wisconsin, USA was attributed to ***Pasteurella multocida* serotype 1** as a cause of acute fatal septicemia with interstitial pneumonia. Gross lesions included pulmonary consolidation and splenomegaly. Microscopically, Gram-negative coccobacilli were noted in blood vessels and endothelial cells of the lung, liver, and



**FIGURE 25.11** *Yersinia pseudotuberculosis* in the liver, *Myotis myotis*. (A) Coagulative necrosis with central accumulation of neutrophils and a rim of lymphocytes and histiocytes in the liver of a bat infected with *Yersinia pseudotuberculosis*. (B) Gram-negative coccobacilli in the liver of a bat infected with *Yersinia pseudotuberculosis*. Gram. (Photos Courtesy of K. Meuhldorfer, Leibniz Institute for Zoo and Wildlife Research (IZW), Berlin, Germany)

spleen with alveolar thrombosis, endothelial necrosis, and neutrophilic interstitial pneumonia (Fig. 25.12) (Bleher et al., 2014). Systemic pasteurellosis is also strongly correlated with cat predation. In one survey of European bats, most isolates of *Pasteurella* cultured from organs of 29 vespertilionid bats were identified as *P. multocida* ssp. *septica* capsular type A, a predominant strain found in the oral cavity of domestic cats (Mühldorfer et al., 2011a). *Pasteurella* spp. have also been reported as pathogens in individual free-ranging European bats (Mühldorfer, 2013).

*Salmonella* sp. serovars capable of infecting a broad range of hosts have been identified in both healthy and septicemic bats. *Salmonella* serotype enteritidis and *Salmonella* serotype typhimurium have been isolated from viscera of free-ranging vespertilionid bats with interstitial pneumonia and suppurative meningitis (Mühldorfer, 2013).



**FIGURE 25.12** *Pasteurella multocida* in the lung, *Eptesicus fuscus*. Mild acute interstitial pneumonia with intracellular and extracellular Gram-variable bacteria with *Pasteurella multocida* septicemia. Brown-Hopps.

*Clostridium perfringens* and *C. sordellii* are reported as a cause of hemorrhagic diarrhea in European vespertilionid bat species (Hajkova and Pikula, 2007; Mühldorfer et al., 2011b). *Clostridium perfringens* is thought to cause gastritis in captive pteropodid bats that consume inappropriate foods (Olsson and Woods, 2008).

In captive pteropodid bats,  $\alpha$ -hemolytic *Streptococcus* spp. occasionally cause bronchopneumonia or subcutaneous abscesses. *Staphylococcus aureus* has been identified in captive pteropodid bats with sepsis and osteomyelitis.

Multiple species of potentially pathogenic bacteria, including *Salmonella* serotype typhi, *Shigella flexneri*, *Campylobacter* spp., *Neorickettsia* spp. including *N. risticii*, *Escherichia coli*, *Yersinia enterocolitica*, *Borrelia* spp., *Bartonella* spp., hemotropic *Mycoplasma* spp., and *Enterococcus* spp. have also been detected in free-ranging bats in the absence of clinical disease (Brook et al., 2015; Brygoo et al., 1970; Marinkelle and Grose, 1968; Mascarelli et al., 2014; Mühldorfer, 2013; Mühldorfer et al., 2010, 2011a).

## Fungi

The fungal infection known as **white-nose syndrome (WNS)** was first detected in bats in a cave in upstate New York in 2006. Within a decade, an estimated 6 million bats had died of the infection, which may represent the largest mammalian wildlife mortality event in recorded history. By 2018, WNS had spread north into Canada, south to Alabama, as far west as central Kansas, and had been detected in a single bat in western Washington ([www.whitenosesyndrome.org/resources/map](http://www.whitenosesyndrome.org/resources/map)), demonstrating the potential of the introduced fungal pathogen *Pseudogymnoascus (Geomyces) destructans* (Pd) to impact bat populations across North America (Lorch et al., 2016). Certain hibernating

North American bat species have been disproportionately affected, while others appear refractory to the disease. Particularly susceptible species include *Myotis lucifugus*, *M. septentrionalis*, and *Perimyotis subflavus* (Frank et al., 2014). *M. lucifugus*, once the most populous species of bat in the Eastern United States, has undergone significant population declines and localized extirpation with complete loss of populations in some affected caves (Frick et al., 2010).

The fungus, *P. destructans*, is thought to be native to Europe and Asia, where it has not previously been recognized as a cause of mortality in native bats ([www.cabi.org/isc/datasheet/119002](http://www.cabi.org/isc/datasheet/119002)). Since the North American outbreak began, both colonization by Pd and lesions diagnostic for WNS have been documented in several species of bats from Europe (Bandouchova et al., 2015; Pikula et al., 2012; Wibbelt et al., 2013; Zukal et al., 2014) and Asia (Hoyt et al., 2016) although significant mortality has not been reported. These differences are most likely due to host factors resulting from evolutionary host–pathogen adaptation, rather than differences in fungal pathogenicity between isolates of Pd from Eurasia and North America (Bandouchova et al., 2015).

*Pseudogymnoascus destructans* is a psychrophilic and keratinophilic fungus. Like other *Pseudogymnoascus* species, it is saprophytic and does not require a host for survival. The fungus's ability to persist in cave sediment in the absence of living hosts is likely to be an important factor for perpetuation of the infection (Reynolds et al., 2015). Bats are thought to become transiently infected in autumn, with transmission increasing throughout winter. Infection peaks in late winter but is cleared in summer in surviving bats, with zero prevalence reported in summer months (Langwig et al., 2015). However, the fungus may still be present in the summer months on the skin of bats that utilize contaminated underground sites for daily torpor (Ballmann, et al., 2017). The growth range of Pd corresponds to the body temperature of most hibernating bats (Verant et al., 2012), and hibernation and the associated decrease in body temperature during periods of torpor have proven to be significant in the pathogenesis of WNS.

As Pd infection progresses, hibernating bats arouse from torpor more frequently and exhibit additional aberrant energy-intensive behaviors, such as premature emergence from hibernacula during the winter period when insect prey is generally not available (Reeder et al., 2012). One experimental study suggested that fat energy utilization was two-fold higher in infected bats even before the onset of increased arousal, suggesting that other physiologic mechanisms of increased metabolism may be present (Verant et al., 2014). Other systemic abnormalities, including increased evaporative water loss through damaged skin, acidosis, hyperkalemia, hypovolemia, and hypotonic dehydration, have been detected in infected bats (Cryan et al., 2010; Verant et al., 2014; Warnecke et al., 2013; Willis et al., 2011).

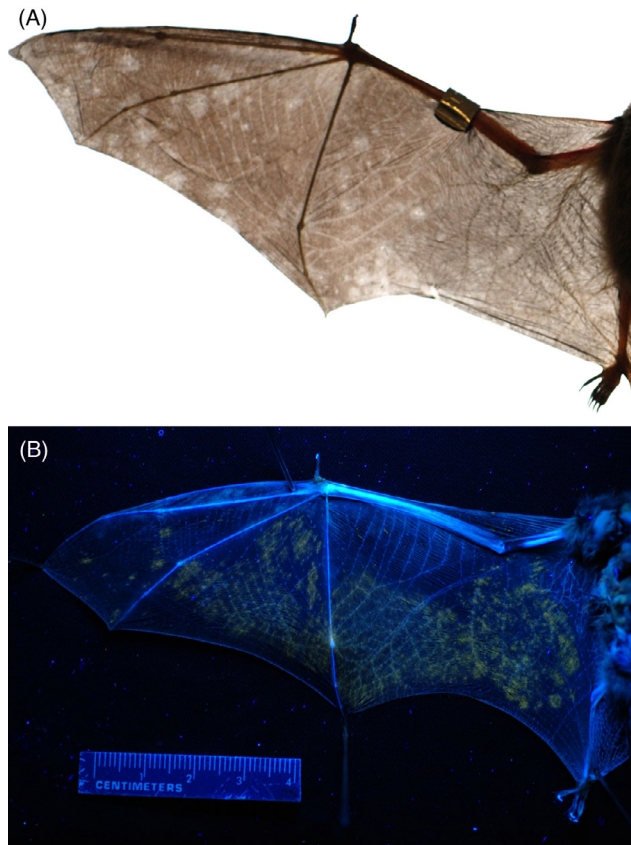
The hallmark of WNS is white fungal growth on the muzzle, ears, and wing membranes of hibernating bats (Fig. 25.13) (Bleher et al., 2009). By the time a carcass is received for necropsy, however, the fungus may no longer be grossly evident. Wing damage may present as ~1–3 mm diameter, multifocal to coalescing white foci with a pinpoint black center or variably-sized areas of depigmentation, tearing, or dryness of the patagia. Wings are best examined backlit as with a radiograph light box (Fig. 25.14A). Additionally, infected skin has a characteristic orange fluorescence under UV light (Fig. 25.14B) (Turner et al., 2014). Examination under UV light can be a more sensitive means of detecting infection than gross lesions alone, as grossly unremarkable areas of the wing may demonstrate fluorescence. Dead bats are frequently emaciated but there are no other systemic lesions, and visceral dissemination has not been reported.

Diagnosis of WNS requires both demonstration of Pd on the skin via PCR or fungal culture and histopathology to demonstrate the presence of characteristic lesions. The area of skin examined microscopically can be maximized by rolling excised portions of the patagia onto wax dowels which are then cut transversely, resulting in multiple cross



**FIGURE 25.13** White-nose syndrome, hibernating *Myotis lucifugus*. White fungal growth is present on the muzzle, pinna, and wing. (Photo Courtesy of U.S. Fish and Wildlife Service)

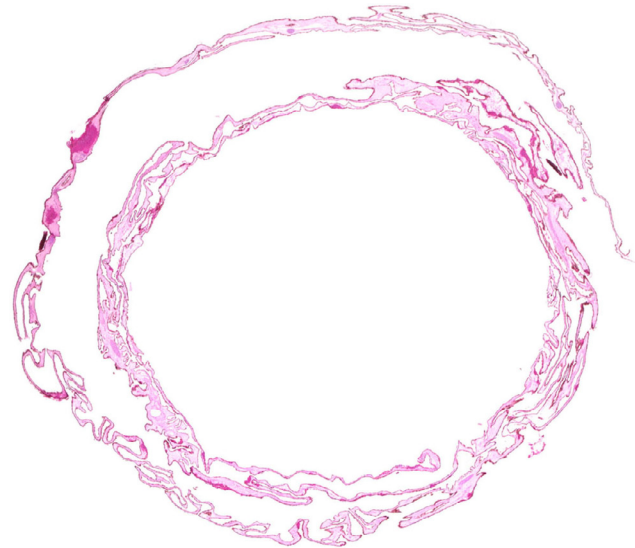




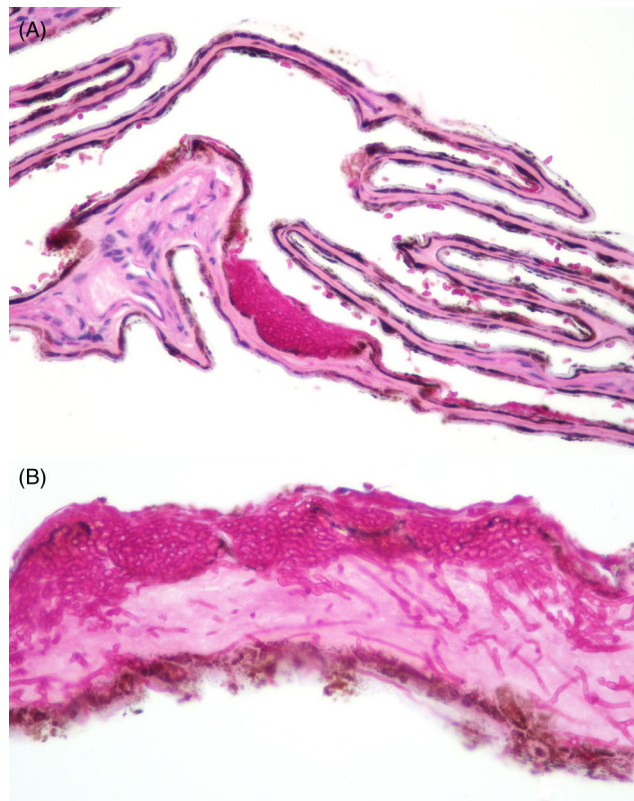
**FIGURE 25.14** White-nose syndrome, *Myotis lucifugus*. (A) Multifocal to coalescing circular areas of pallor are common on wing membranes of bats with white-nose syndrome. (B) Ultraviolet illumination reveals orange fluorescence characteristic of *Pseudogymnoascus destructans* infection. (Photos Courtesy of U.S. Geological Survey-National Wildlife Health Center)

sections of concentric rolls of skin (Fig. 25.15) (Meteyer et al., 2009). Pathognomonic lesions include cupping erosions and ulcers filled with densely packed, PAS- and GMS-positive fungal hyphae with variable extension through the basement membrane (Fig. 25.16). Hyphae commonly extend into hair follicles and sebaceous or apocrine glands. In tissue section, fungal hyphae exhibit mild morphological variability. Hyphae are septate and branching, with parallel to slightly irregular walls 2–5 μm in diameter. Conidia, when present, are curved and measure ~2.5 μm wide × 7.5 μm long (Fig. 25.16A) (Meteyer et al., 2009). The lack of inflammatory response to the invading hyphae is typical of the lesion in hibernating bats. However, neutrophilic inflammation can occur in hibernating bats when the fungal lesion is superinfected with bacteria (Courtin et al., 2010; Meteyer et al., 2009). Bats that have emerged from hibernation may exhibit a neutrophilic response to Pd (Fig. 25.17), as well as edema and necrosis that are not present in hibernating bats (Meteyer et al., 2011, 2012).

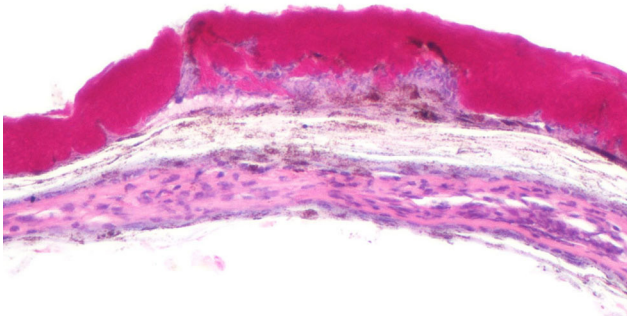
Although the mortality rate is high, the disease is not invariably fatal and infected bats can recover and the wing



**FIGURE 25.15** White-nose syndrome, patagium, *Myotis lucifugus*. Rolled wing membrane maximizes surface area of skin available for microscopic examination. Dense packets of PAS-positive fungi are easily identified at low magnification. Periodic acid-Schiff.



**FIGURE 25.16** White-nose syndrome, patagium, *Myotis lucifugus*. (A) Cupping erosion. Scattered over the surface of the stratum corneum are many curved arthroconidia characteristic of *Pseudogymnoascus destructans*, Periodic acid-Schiff. (B) Severe *P. destructans* infection with ulceration and transdermal extension of fungal hyphae. Note the lack of inflammatory response. Periodic acid-Schiff.



**FIGURE 25.17** White-nose syndrome, patagium, *Myotis lucifugus*. Wing membrane from a euthermic bat collected in May after emergence from hibernation. In contrast to the typical non-inflammatory cupping erosion of WNS seen in hibernating bats, the patagium exhibits dermatitis, hyperkeratosis, and a serocellular crust containing myriad fungal hyphae. Periodic acid-Schiff.

damage can heal (Fuller et al., 2011; Meteyer et al., 2011). In some cases, bats that survive into the summer may experience poor hunting or reproductive success due to postinfection scarring (Reichard and Kunz, 2009).

In addition to *P. destructans*, few other fungal species have been reported to cause mycotic dermatitis in bats, and most are limited to single case reports. One exception is a report describing gross and microscopic lesions of the dermatophyte *Trichophyton redellii* in hibernating bats from Wisconsin, Indiana, and Texas, USA. *T. redellii* can cause gross lesions on the ears, wings, legs, and tail that may resemble WNS, but the muzzle is not noted to be affected. “Ring”-type lesions characteristic of human ringworm may also occasionally be seen (Fig. 25.18A). Microscopically, *T. redellii* invades the stratum corneum and deeper epidermal layers with a neutrophilic pustular response (Fig. 25.18B), but the aggregation, erosion, and deep dermal invasion common in WNS are not present. Conidia of *T. redellii* are radially symmetric and obovate to pyriform, in contrast to the asymmetrical curved conidia of Pd. Hyphae are septate, 2–6  $\mu\text{m}$  diameter, and morphologically similar to Pd. Differentiation between WNS and *T. redellii* infection may require close examination of the pattern of invasion and inflammation and fungal culture or molecular identification (Lorch et al., 2015).

*Hyphopichia burtonii* has been reported as a cause of fungal dermatitis in a free-ranging *Barbastella barbastellus* (Simpson et al., 2013). In this case report, gross lesions included skin thickening, crusting, ulceration, and nodule formation. Thin, branching, septate fungal hyphae and masses of  $\sim 4 \mu\text{m}$  diameter ovoid spores were within the stratum corneum and hair follicles with epidermal hyperplasia and follicular hyperkeratosis. Fungal dermatitis has also been associated with *Cladosporium* spp. in a euthermic *Vespertilio murinus* with white foci on wing membranes and severe necrotizing dermatitis with intralesional fungal hyphae (Barlow et al., 2011).

Bats can serve as reservoirs for *Histoplasma capsulatum*, although clinical disease in free-ranging bats is uncommon. Organisms are most often present in pulmonary alveolar macrophages but can also be found in circulating macrophages and in the mesentery, spleen, liver, adrenal gland, and intestine (Taylor et al., 1999; Tesh and Schneidau, 1966). There is typically minimal to no cellular response to infection, which is thought to occur by the respiratory route with infective fungi being excreted in feces (McMurray and Greer, 1979; Taylor et al., 1999). There are no reports of mortality due to natural infections in free-ranging bats (Greer and McMurray, 1981). The ability of bats to survive disseminated infection, including intestinal infection with shedding of spores, likely allows them to serve as reservoirs. During an outbreak at the Houston Zoo, USA, 12 *Rousettus aegyptiacus* and one *Artibeus jamaicensis* had systemic histoplasmosis. The lung was a primary site of infection in all cases except 4 *R. aegyptiacus* (Tocidowski, 2003).

Juvenile pteropodid bats with poor husbandry or chronic illness can develop cutaneous *Candida albicans* infections, most commonly on the wing membranes. Lesions initially present as erythema progressing to a gray, slimy pseudomembrane from which hair epilates in clumps. Concurrent cutaneous and oral candidiasis is sometimes identified. Oral candidiasis is common in hand-raised pteropodid bats, and gastric candidiasis may occur in pteropodid bats consuming inappropriately nutrient-dense diets, as well as in immunosuppressed or debilitated pups, especially after prolonged antibiotic administration (Olsson and Woods, 2008). Oral and esophageal candidiasis is sometimes observed in captive bats, with neutrophilic infiltrates, erosions, ulcers, or necrosis of the mucosa and intralesional 2–6  $\mu\text{m}$  diameter ovoid budding yeasts and 3–5  $\mu\text{m}$  diameter hyphae and pseudohyphae. Often these animals have received antibiotics or have other concurrent disease processes. Occasional captive pteropodid bats have keratitis and/or conjunctivitis associated with *Candida*.

## Metazoa

Infections with various nematodes, trematodes, cestodes (Webster and Casey, 1973), and acanthocephalans (Smales, 2007) have been described in bat species but specific clinical diseases have only been described for a few metazoans.

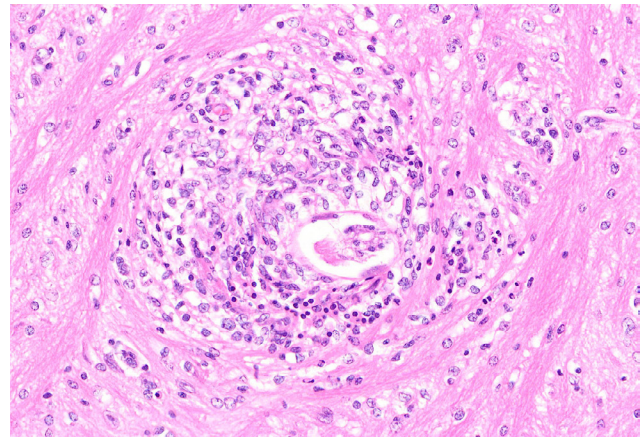
Neurologic disease due to infection by *Angiostrongylus cantonensis*, the rat lungworm, has been reported in free-ranging *Pteropus alecto* and *P. scapulatus* and captive and free-ranging *P. poliocephalus* in Australia (Barrett et al., 2002; Olsson and Woods, 2008; Reddacliff et al., 1999). Histologic lesions are eosinophilic and granulomatous meningitis or meningoencephalitis, with foci of malacia, and possible hemorrhage and/or gliosis (Fig. 25.19). Larval or subadult nematodes may be identified in the meninges, subarachnoid space, ventricles or



**FIGURE 25.18** *Trichophyton redellii* in the skin. (A) *Myotis lucifugus*. White fungal growth is present on the wrist and wing. Few lesions exhibit an outer zone of fungal growth with circular clearing typical of dermatomycosis in humans. (B) *Trichophyton redellii* dermatitis in the patagium, *Myotis velifer*. Fungal hyphae form a dense mat on the surface of the stratum corneum. Invasion of the epidermis is associated with neutrophilic inflammation, Periodic acid-Schiff. (Part A: Photo Courtesy of H. Kaarakka, Wisconsin Department of Natural Resources)

brain parenchyma, and sometimes no inflammatory reaction is present around nematodes (Barrett et al., 2002; Reddacliff et al., 1999). Granulomatous endarteritis in *P. alecto* associated with a second rat lungworm, *Angiostrongylus mackerrasae*, results from parasites in the pulmonary arteries. Severe, mixed inflammatory cell interstitial pneumonia with intralesional eggs and rare larvae in the parenchyma can also be seen (Mackie et al., 2013).

Infection with *Toxocara pteropodis* has been described in free-ranging flying foxes from Australia (*Pteropus alecto*, *P. scapulatus*, *P. poliocephalus*, and *P. conspicillatus*), Sri Lanka (*P. giganteus*) and the South Pacific (*P. tonganus*) (Prociv, 1985) and in one captive flying fox in Florida (*P. hypomelanus*) (Heard et al., 1995). In adult bats, third stage larvae are present in the liver, as well as the mammary gland of lactating females that pass the larvae to suckling bats via transmammary transmission. Adult nematodes are normally only identified in the intestine of juvenile bats (Prociv, 1983). However, they can occasionally be seen in other sites and in other age classes of bats. They have been described in the gall bladder, common bile duct, esophagus, and pharynx/larynx, and a large number of nematodes in the stomach, duodenum, and proximal jejunum were associated with small intestinal volvulus in a young *P. conspicillatus*. Aberrant larval migration has also occasionally been reported (Prociv, 1983, 1990). In free-ranging bats, large numbers of intestinal nematodes can be negatively correlated with body condition, and volvulus, intussusception and possible partial intestinal obstruction have been observed in



**FIGURE 25.19** Encephalitis with *Angiostrongylus cantonensis*, *Pteropus poliocephalus*. A nodular infiltrate of predominantly mononuclear cells, with rare eosinophils, surrounds a nematode. (Photo Courtesy of K. Rose, Australian Registry of Wildlife Health, Taronga Conservation Society Australia)

some infected bats (Karawita et al., 2017). Failure to thrive may occasionally be observed in hand-reared *Pteropus* spp. with heavy ascarid loads (Olsson and Woods, 2008).

The nematode *Riouxgolvania beveridgei* has been identified as the cause of cutaneous granulomas in free-ranging *Miniopterus schreibersii bassanii*. Gross lesions include 1–2 mm diameter white nodules or ulcers on the dorsal aspect of the thoracic and pelvic limbs. Microscopically, there is granulomatous to pyogranulomatous dermatitis with central degenerate nematodes. Males are more

commonly affected than females (McLelland et al., 2013). Dermal nodules are also reported with *Riouxgolvania kapapkamui* in *Myotis* spp. in Japan (Hasegawa et al., 2012).

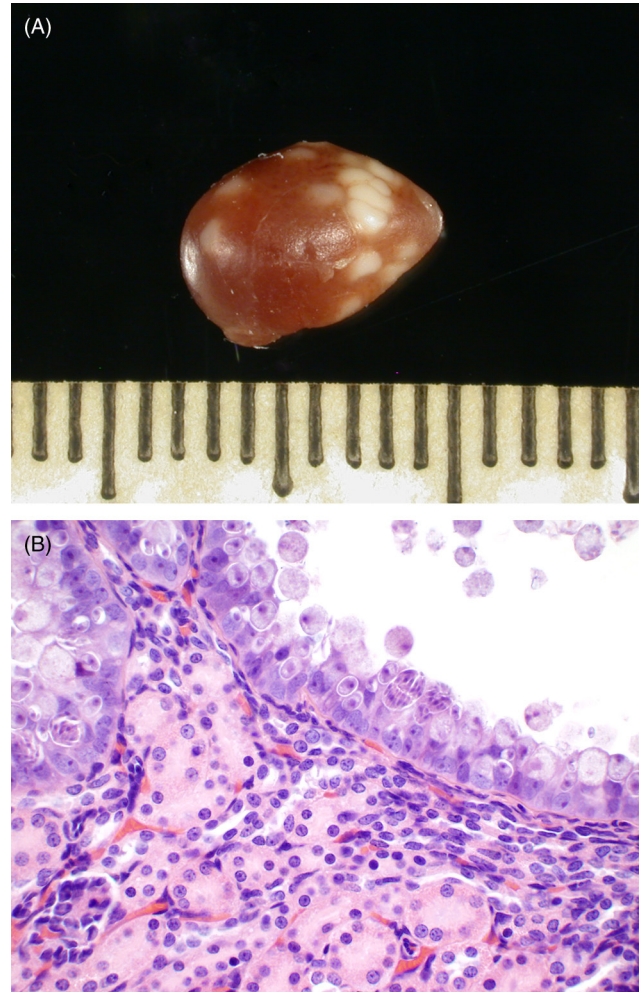
The filarid nematode *Molossinema wimsatti* has been reported in the central nervous system of *Molossus rufus* (*M. ater*). These nematodes resemble *Litomosoides* and *Litosoma* spp., which have been described in the thoracic cavity of bats. Clinical signs were minimal, although ataxia was noted in one animal before death. Few to many 2–5 cm long 100–200 µm diameter nematodes were in the lateral, third, and fourth ventricles of the brain and in the subdural space of the cervical and thoracic vertebral canal. With the exception of ventricular dilation, tissue response was rare and limited to mild focal encephalitis and necrosis of adjacent parenchyma in one animal. Infection was considered an incidental finding (Nguyen and Myers, 1987).

*Physaloptera brevivaginata* can cause gastritis in *Myotis* spp. Grossly, the site of attachment may be visible from the gastric serosa as a red focus, and there is mucosal erosion with occasional ulceration. Microscopically, parasites attach at the mucosal–submucosal junction with a mixed inflammatory response including mononuclear cells, polymorphonuclear cells, edema, and fibrosis (Botella and Esteban, 1995).

## Protozoa

Infection with *Toxoplasma gondii* in two captive juvenile Australian flying foxes (*Pteropus scapulatus* and *P. conspicillatus*) caused necrosis and granulomatous to pyogranulomatous inflammation in multiple tissues including the lungs, brain, peritoneum, and gastrointestinal wall, as well as the heart and skeletal muscle in the *P. scapulatus* (Sangster et al., 2012).

**Renal coccidiosis** is reported in a few bat species, including *Eptesicus fuscus*, *Pipistrellus pipistrellus*, *Myotis mystacinus*, *M. nattereri*, *M. sodalis*, *Nyctalus noctula*, *Hipposideros caffer*, and *Rhinolophus* sp. (Gruber et al., 1996; Kusewitt et al., 1977; Wünschmann et al., 2010). Early reports in *M. sodalis* presumptively identified the coccidia morphologically as *Klossiella* sp. (Kusewitt et al., 1977). The name *Nephroispora eptesici* has been proposed for the organism in *E. fuscus* (Wünschmann et al., 2010). White foci up to 2 mm diameter may be visible on the renal capsular surface (Fig. 25.20A). Microscopically, the lesion is similar to that caused by *Eimeria stiedae* in the bile ducts of rabbits. Tubules, particularly those in the deep cortex and medulla, are markedly dilated with hypertrophic and hyperplastic epithelium. Epithelial cells contain asexual and sexual stages of the organism, and tubular lumina contain schizonts, merozoites, macrogametocytes,



**FIGURE 25.20** Renal coccidiosis, *Eptesicus fuscus*. (A) White foci up to 2 mm in diameter are present on the surface of the kidney. (B) Renal tubules are dilated with proliferative epithelium, with various life stages of coccidia within the epithelium and lumen. (Photos Courtesy of A. Wünschmann, University of Minnesota College of Veterinary Medicine)

microgametocytes, and sporulated and unsporulated oocysts (Fig. 25.20B) (Gruber et al., 1996; Wünschmann et al., 2010). Eosinophilic, histiocytic, and lymphoplasmacytic inflammation may occur but is rare. There has been no evidence of clinically significant impairment of renal function. Marked cystic tubular dilation distinguishes renal coccidiosis of bats from that of waterfowl, amphibians, and other species (Gruber et al., 1996). Moreover, the entire life cycle is thought to be completed in a single host without requiring sporogony in the environment (Wünschmann et al., 2010).

Bats are thought to be potential reservoirs for *Leishmania* spp., although there are few reports of associated disease (Roque and Jansen, 2014). *Leishmania (Leishmania) mexicana* was associated with a raised ulcerated mass on the leading edge of the patagium of a free-ranging *Artibeus*

*lituratus* in Brazil that was found dead (Berzunza-Cruz et al., 2015). Cutaneous lesions not further described were also associated with *L. (Viannia) braziliensis* in two *Glossophaga soricina* in Brazil (Shapiro et al., 2013). Other species of *Leishmania*, including *L. (Leishmania) amazonensis* and *L. (Leishmania) infantum chagasi*, have also been detected in Brazilian bats (Savani et al., 2010).

Numerous **hemoparasites** are described in bats, although the clinical effect of these parasites is largely unknown, and most infections are likely subclinical. Free-ranging pteropodid bats may be infected with **Hepato-cystis sp.**, with gametocytes in the blood and schizonts in the liver (Landau et al., 2012). Schizonts may be seen as small white foci in the liver (Garnham, 1950) but histologically there is no associated inflammation (Ladds, 2009). No clinical disease has been described. Several additional genera of hemoparasites, including *Trypanosoma* (Jansen et al., 2015), *Polychromophilus*, *Plasmodium*, *Nycteria* (Schaer et al., 2013), *Johnspretia*, *Sprattiella* (Landau et al., 2012), *Babesia* (Concannon et al., 2005), and *Hepatozoon* (Pinto et al., 2013) have been reported in free-ranging bats, with no reports of associated clinical disease. Several other apicomplexan parasites that have been identified in bats without evidence of lesions or significant clinical disease include *Eimeria* sp. (McAllister and Upton, 2009) and *Cryptosporidium* sp. (Wang et al., 2013).

## Ectoparasites

Numerous **ectoparasites**, including flies (Streblidae and Nycteribiidae), chiggers (Trombiculidae), fleas, ticks, bed bugs, and mites have been described in bats (Dick et al., 2003; Moras et al., 2013; Takahashi et al., 2006). This discussion will be limited to ectoparasites that have been associated with pathology.

**Tick paralysis**, caused by toxins of *Ixodes holocyclus*, is common in northern Australia in *Pteropus conspicillatus*. It typically affects females in maternity colonies more than males. Animals present with ascending motor paralysis, with diaphragmatic involvement resulting in labored breathing and eventual respiratory failure. Diagnosis is based on clinical signs and the presence of the tick or an ulcer consistent with prior tick attachment; other gross or histologic lesions are not present (Buettner et al., 2013; Olsson and Woods, 2008). Free-ranging *Cynopterus brachyotis* infested with **teinocoptid mites** have acanthosis with dermal fibroplasia subtending the site of attachment of mites (Lavoipierre and Rajamanickam, 1968). *Demodex* sp. has been reported in preputial hair follicles of *Eptesicus fuscus* and is associated with mild eosinophilic folliculitis and lymphoplasmacytic preputial gland adenitis (Lankton et al., 2013).

**E-SLIDES**

- 25.e1** **Reactive fibroplasia, *Pteropus vampyrus*, patagium.** Irregular nodules of fibroblasts expand the patagium and infiltrate into and around elastin bundles. Anisocytosis, anisokaryosis, and multinucleation are observed in some of the fibroblasts. While this lesion was initially diagnosed as a fibrosarcoma, the lesion subsequently regressed, suggesting that this was reactive fibroplasia. (see [Fig. 25.9](#)). eSlide: [VM04973](#)
- 25.e2** **Iron overload, *Rousettus aegyptiacus*, liver.** Large amounts of fibrous connective tissue expand and bridge between portal tracts. Abundant brown pigment (hemosiderin) is present within and at the margins of the connective tissue. (see [Fig. 25.5](#)). eSlide: [VM05015](#)
- 25.e3** **Iron overload, *Rousettus aegyptiacus*, liver. Prussian blue.** Abundant hemosiderin is present in macrophages within and at the margins of the fibrous connective tissue, with lesser amounts in hepatocytes, bile ducts, fibroblasts and Kupffer cells. Hepatocytes in regenerative nodules sometimes contain little or no hemosiderin. Prussian blue. (see [Fig. 25.5](#)). eSlide: [VM04976](#)
- 25.e4** **White-nose syndrome, *Myotis lucifugus*, patagium (wing skin). Periodic acid-Schiff.** Multifocally within the epidermis of the patagium are characteristic cupping erosions containing densely-packed PAS-positive fungal hyphae. Low numbers of approximately  $2 \times 7 \mu\text{m}$  curved arthroconidia characteristic of *Pseudogymnoascus destructans* overlie the skin in some areas. Periodic acid-Schiff. (see [Figs. 25.15–25.16](#)). eSlide: [VM04963](#)

## E-ONLY CONTENTS

### ADDITIONAL TOXIC DISEASES

Ivermectin toxicosis was suspected in 11 captive *Cynopterus brachyotis* with generalized paresis 1 day after administration of one drop of 10 mg/mL ivermectin topically. Ingestion during grooming, increased drug absorption through the patagium, or a heightened sensitivity to ivermectin in this species were possible mechanisms for toxicity. Three spontaneously died and one was euthanized within 48 hours of ivermectin administration, one died 7 days after administration, and the other six bats recovered. Three bats that died within 48 hours of ivermectin administration had renal tubular necrosis with regeneration, and the bat that died after 7 days had suppurative bronchopneumonia. As tubular necrosis has not been reported with ivermectin toxicosis except in humans with onchocerciasis, the cause of the tubular necrosis was not determined (DeMarco et al., 2002). Additionally, tubular regeneration would not be expected for a toxic insult within 48 hours, so renal lesions may have been unrelated to ivermectin toxicosis. Bronchopneumonia may have been a result of aspiration due to neurologic weakness in one bat.

Lead toxicity has been reported in Australian flying foxes, including captive and free-ranging *Pteropus poliocephalus* and free-ranging *P. alecto*. Clinical signs in free-ranging bats include incoordination, inability to fly, aggression, muscle tremors, ptialism, and ataxia, while captive bats were found separated from the group and were subjects of conspecific aggression. Histologic lesions include renal tubular necrosis, proximal tubular karyomegaly and nuclear pleomorphism, and variably acid fast, eosinophilic proximal renal tubular and hepatic intranuclear inclusion bodies (Hariono et al., 1993; Skerratt et al., 1998; Sutton and Wilson, 1983; Zook et al., 1970). Concurrent lyssaviral infection was diagnosed in one free-ranging *P. alecto* with signs of aggression and cytoplasmic neuronal brainstem inclusions but no encephalitis (Skerratt et al., 1998). The source of lead was thought to be paint in captive bats (Zook et al., 1970) and environmental in free-ranging Australian *Pteropus* spp. that had lead in fur washings (Hariono et al., 1993).

Copper toxicosis from an undetermined source has been reported in a single captive *Artibeus jamaicensis* that died without premonitory signs. Histologic lesions were random acute hepatocellular necrosis with abundant copper pigment in hepatocytes and macrophages, and hemoglobin casts in renal proximal tubules associated with tubular degeneration. Hepatic copper concentration of this bat was 4540 ppm dry weight, compared to 1.08–99.20 ppm in 16 samples from other fruit and vampire bat species tested (Hoenerhoff and Williams, 2004).

Heavy metal toxicity has also been documented as a risk to bat populations, although little pathology has been described (Clark, 1981; Clark and Shore, 2001; Hernutz

et al., 2015; Yates et al., 2014; Zukal et al., 2015). In one report, *Myotis grisescens* in Alabama from a cave with elevated guano cadmium levels demonstrated renal tubular degeneration; tissue cadmium concentrations were not evaluated (Clark and Shore, 2001). Cadmium injections were linked with testicular necrosis in *Rhinopoma kinneari* (Dixit and Lohiya, 1974). Microscopic lesions, including hepatic necrosis, vacuolation, inflammation and atrophy, as well as renal necrosis and inflammation, were reported in *Neoromicia nana* feeding at wastewater treatment sites, with elevated tissue levels of iron, zinc, and copper; other studies of this population documented DNA damage and decreased antioxidant capacity (Naidoo et al., 2013, 2015, 2016; Zocche et al., 2010). Organic tin compounds have been associated with decreased complement activity and a possible decrease in immune response in *Myotis daubentonii* (Lilley et al., 2013).

Free-ranging bats are occasionally exposed to anticoagulant rodenticides; clinical signs and lesions are similar to those exhibited in other species and include coagulopathy with multisystemic hemorrhage. Diphacinone residues have been detected in milk samples from the stomach of dead pups. Depending on the form and placement of bait, omnivorous species could be at risk of either primary or secondary poisoning (Dennis and Gartrell, 2015).

The rodenticide zinc phosphide has been reported to cause tremors and death in *Myotis lucifugus* (Hurley and Fenton, 1980).

Algal toxins have rarely been suspected to affect bats. In one report, microcystins were detected in the liver of bats feeding over a lake contaminated with cyanobacteria; no deaths or other lesions were reported (Woller-Skar et al., 2015). In another report, algal toxins were detected on the surface of the carcass of bats suspected to have died acutely from algal toxicosis; no gross or microscopic lesions were identified in these bats (Pybus et al., 1986).

### ADDITIONAL BACTERIAL DISEASES

*Escherichia coli* has been reported in association with an ascending urinary tract infection in two vespertilionid bats in Germany (Mühldorfer et al., 2011).

Although several *Borrelia* spp. have been detected in bats, reports of disease are uncommon (Mühldorfer, 2013). In one report from England, a free-ranging *Pipistrellus* sp. was found moribund. At necropsy, it had skeletal muscular pallor, pleural effusion, thoracic lymphadenomegaly, and hepatosplenomegaly. Microscopically, there was hepatocellular necrosis and vacuolation, pulmonary congestion and pneumonia, and argyrophilic bacilli in the liver, lung, spleen, and blood vessels. Partial sequencing revealed a novel *Borrelia* sp. similar to *B. recurrentis*, *B. duttonii*, and *B. crocidurae*, species associated with relapsing fevers in Africa and Asia (Evans et al., 2009).

Bats are considered likely reservoirs for *Leptospira* spp. (Dietrich et al., 2015). *Leptospira* spp. have been detected in the urine of many species of bats from a variety of habitats, with a particularly high detection rate in the kidney and urine of Australian flying foxes. Phyllostomid, vespertilionid and molossid bats have been reported to carry pathogenic species of *Leptospira*, with higher infection rates in bats in forest habitats (Mühldorfer, 2013). There are no reports of clinical leptospirosis in bats.

*Listeria* sp. has been isolated from two captive *Pteropus rodricensis*: from a wing interphalangeal joint with osteomyelitis and arthritis, and from a liver with random hepatocellular necrosis.

Two cases of mycobacterial infection, identified as bovine type with the technology of the time, were identified in captive *Pteropus giganteus* in 1925–30. One bat had granulomatous pneumonia, pleuritis, pericarditis, and epicarditis (Scott, 1926), and the other had granulomatous peritonitis and mesenteric and hepatic lymphadenitis (Hamerton, 1931).

## ADDITIONAL FUNGAL DISEASES

Pyogranulomatous rhinitis and osteomyelitis due to *Cryptococcus* sp. has been seen in a captive *Rousettus aegyptiacus*. Yeasts consistent with *Malassezia* sp. are occasionally seen in the superficial keratin with dermatitis in pteropodid bats.

Numerous pyogranulomatous nodules were identified in the lung of a captive pregnant female *Pteropus giganteus* in Indiana, with intralesional organisms histologically consistent with *Blastomyces dermatitidis* (Raymond et al., 1997).

*Coccidioides posadasii* infection has been reported in a free-ranging *Carollia perspicillata* in Brazil. This bat had mature spherules with endospores in the lung but it is unclear whether this was causing clinical disease (Cordeiro et al., 2012).

*Pneumocystis* spp. colonization has been reported in free-ranging bats, both alone and in coinfections with *H. capsulatum* (Cavallini Sanches et al., 2013; González-González et al., 2014). According to one report, *Pneumocystis* infection rate as determined by PCR is high within multiple bat species surveyed (35.3–41.9%) (Akbar et al., 2012); however, clinical disease has not been reported.

Granulomatous/histiocytic inflammation and organisms consistent with the algae *Prototheca* were identified in the lymph nodes, spleen, meninges/brain, mesentery, pancreas, heart, skeletal muscle, and kidney of a captive *Pteropus lylei* (Mettler, 1975).

## ADDITIONAL PROTOZOAL DISEASES

*Balamuthia mandrillaris* was identified as the cause of encephalitis in a captive juvenile *Pteropus giganteus*. Gross lesions included multiple foci of malacia and red discoloration in the brain. Histologically, there were large areas of

liquefactive necrosis with numerous amoebic trophozoites and neutrophilic inflammation, and perivascular and meningeal lymphoplasmacytic, eosinophilic, and histiocytic infiltrates (Crossland et al., 2016).

Disseminated microsporidiosis caused by *Encephalitozoon hellem* was identified in a captive female adult *Rousettus aegyptiacus*. Histologic lesions included chronic-active cholangiohepatitis, renal tubular degeneration and necrosis, thyroiditis, cystitis, and ureteritis. Microsporidian spores were identified in hepatocytes, bile duct epithelial cells, renal tubular epithelial cells, gastric mucosal epithelial cells, and in the ureter, urinary bladder, and thyroid gland (Childs-Sanford et al., 2006).

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**TABLE e1** Scientific and Common Names of Species in Chiroptera Chapter

Genus	Species	Common Name(s)
<i>Anoura</i>	<i>caudifer</i>	Tailed tailless bat
<i>Artibeus</i>	<i>jamaicensis</i>	Jamaican or common fruit bat
	<i>lituratus</i>	Great fruit-eating bat
<i>Barbastella</i>	<i>barbastellus</i>	Western barbastelle, barbastelle bat
<i>Carollia</i>	<i>perspicillata</i>	Seba's short-tailed bat, short-tailed fruit bat
<i>Cynopterus</i>	<i>brachyotis</i>	Lesser dog-faced fruit bat, lesser short-nosed fruit bat
<i>Desmodus</i>	<i>rotundus</i>	Common vampire bat
<i>Diphylla</i>	<i>ecaudata</i>	Hairy-legged vampire bat
<i>Eidolon</i>	<i>helvum</i>	Straw-colored fruit bat
<i>Eonycteris</i>	<i>spelaea</i>	Cave nectar bat, lesser dawn bat
<i>Epomophorus</i>	<i>gambianus</i>	Gambian epauletted fruit bat
	<i>wahlbergi</i>	Wahlberg's epauletted fruit bat
<i>Epomops</i>	<i>buettikoferi</i>	Buettikofer's epauletted fruit bat
	<i>franqueti</i>	Franquet's epauletted fruit bat
<i>Eptesicus</i>	<i>fuscus</i>	Big brown bat
	<i>isabellinus</i>	Meridional serotine bat
	<i>serotinus</i>	Serotine bat

**TABLE e1** Scientific and Common Names of Species in Chiroptera Chapter (Cont.)

Genus	Species	Common Name(s)
<i>Glossophaga</i>	<i>soricina</i>	Pallas' long-tongued bat
<i>Hipposideros</i>	<i>caffer</i>	Sundevall's round leaf or leaf-nosed bat; Cape, common African or lesser leaf-nosed bat
	<i>pomona</i>	Pomona or Andersen's leaf-nosed or roundleaf bat
<i>Hypsignathus</i>	<i>monstrosus</i>	Hammer-headed fruit bat
<i>Lasionycteris</i>	<i>noctivagens</i>	Silver-haired bat
<i>Megaderma</i>	<i>spasma</i>	Lesser false vampire bat, common Asian ghost bat
<i>Micropteropus</i>	<i>pusillus</i>	Peters' dwarf epauletted bat
<i>Miniopterus</i>	<i>schreibersii</i>	Common bent-wing bat, Schreiber's bent-winged, or long-fingered bat
<i>Molossus</i>	<i>molossus</i>	Pallas' mastiff bat, velvety free-tailed bat
	<i>rufus</i>	Black mastiff bat (sometimes in literature as <i>M. ater</i> )
<i>Murina</i>	<i>leucogaster</i>	Greater tube-nosed bat
<i>Myonycteris</i>	<i>torquata</i>	Little collared fruit bat
<i>Myotis</i>	<i>daubentonii</i>	Daubenton's bat
	<i>grisescens</i>	Gray bat
	<i>lucifugus</i>	Little brown bat
	<i>myotis</i>	Greater mouse-eared bat
	<i>mystacinus</i>	Whiskered bat
	<i>nattereri</i>	Natterer's bat
	<i>septentrionalis</i>	Northern long-eared bat
	<i>sodalis</i>	Indiana bat, social bat
<i>Mystacina</i>	<i>tuberculata</i>	New Zealand lesser short-tailed bat
<i>Neoromicia</i>	<i>nana</i>	Banana bat, banana pipistrelle
<i>Nyctalus</i>	<i>noctula</i>	Common noctule
<i>Nycteris</i>	<i>gambiensis</i>	Gambian slit-faced bat
	<i>hispidia</i>	Hairy slit-faced or long-eared bat
	<i>thebaica</i>	Egyptian or common slit-faced bat, Cape long-eared bat, Geoffroy's nycteris
<i>Perimyotis</i>	<i>subflavus</i>	Eastern pipistrelle, tricolored bat
<i>Pipistrellus</i>	<i>abramus</i>	Japanese house bat, Japanese pipistrelle
	<i>pipistrellus</i>	Common pipistrelle
<i>Pteropus</i>	<i>alecto</i>	Black or central flying fox
	<i>conspicillatus</i>	Spectacled flying fox
	<i>giganteus</i>	Indian flying fox (sometimes in literature as <i>P. medius</i> )
	<i>hypomelanus</i>	Island, small or variable flying fox
	<i>lylei</i>	Lyle's flying fox
	<i>poliocephalus</i>	Gray-headed flying fox
	<i>pumilus</i>	Little golden-mantled flying fox
	<i>rodricensis</i>	Rodrigues flying fox
	<i>scapulatus</i>	Little red flying fox
	<i>tonganus</i>	Pacific or insular flying fox
<i>vampyrus</i>	Malayan or large flying fox	

(Continued)

**TABLE e1** Scientific and Common Names of Species in Chiroptera Chapter (*Cont.*)

Genus	Species	Common Name(s)
<i>Rhinolophus</i>	<i>ferrumequinum</i>	Greater horseshoe bat
<i>Rhinopoma</i>	<i>hardwickii</i>	Lesser mouse-tailed bat
	<i>kinneari</i>	Rat-tailed bat
<i>Rousettus</i>	<i>aegyptiacus</i>	Egyptian fruit bat
	<i>lanosus</i>	Ruwenzori long-haired rousette, mountain fruit bat
	<i>leschenaulti</i>	Leschenault's or Shortridge's rousette
<i>Saccolaimus</i>	<i>flaviventris</i>	Yellow-bellied sheath-tailed or pouched bat
<i>Tadarida</i>	<i>brasiliensis</i>	Mexican or Brazilian free-tailed bat
<i>Taphozous</i>	<i>perforatus</i>	Egyptian or Geoffroy's tomb bat, perforated taphozous bat
<i>Vespertilio</i>	<i>murinus</i>	Parti-colored bat

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