Chapter 9
White-Nose Syndrome in Bats

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Abstract White-nose syndrome (WNS) is an infectious disease of hibernating bats that has killed millions of bats since it first emerged in eastern North America in 2006. The disease is caused by a pathogenic fungus, *Pseudogymnoascus* (formerly *Geomyces*) *destructans* that was likely introduced to North America by human trade or travel, demonstrating the serious problem of global movement of pathogens by humans in the Anthropocene. Here, we present a synthesis of the current state of knowledge on WNS, including disease mechanisms, disease ecology, global distribution and conservation and management efforts. There has been rapid research response to WNS and much about the disease is now well understood. However, critical gaps in our knowledge remain, including ways to limit spread, or effective treatment options to reduce disease mortality. There are several hibernating bat species in North America that are threatened with extinction from WNS. Protecting those species has become a race against time to find and implement creative solutions to combat the devastating impacts of this disease.

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9.1 Introduction

In late winter of 2007, biologists at the New York State Department of Environmental Conservation encountered a macabre scene during their annual winter surveys of hibernating bats in caves and mines in northern New York State: heaps of dead bats piled on cave floors (Fig. 9.1) (Veilleux 2008). Bats were also seen flying out in the middle of winter onto the snowy landscape and the number of citizen reports of dead bats found in backyards was much higher than normal. A white fuzzy growth was observed on muzzles and wings of the few remaining live bats, which led to the name white-nose syndrome (WNS) (Veilleux 2008; Reeder and Turner 2008; Turner and Reeder 2009). WNS is now recognized as one of the most devastating wildlife epidemics in recorded history and has caused the death of millions of bats in eastern North America. The research and management response to WNS has been rapid and we know much more about WNS than when those first dead bats were observed in New York, although there is still a great deal about this wildlife disease that is yet to be resolved.

The first evidence of WNS in North America is dated to a photograph taken by a caver at Howe's Cave in 2006 (Turner and Reeder 2009). Howe's Cave is a popular tourist attraction that receives hundreds of thousands of visitors each year, many of whom visit from other parts of the world. The white fuzzy growth visible on bats is caused by a pathogenic fungus, which was described as *Geomyces destructans* (Gargas et al. 2009; Blehert et al. 2009), but was recently re-named *Pseudogymnoascus destructans* after closer evaluation of its taxonomic allies (Minnis and Lindner 2013). The fungus infects the skin tissues, including the wings and tail membranes, and causes bats to arouse too frequently from torpor

Fig. 9.1 Bats that died from WNS during winter at Aeolus Cave in Vermont, USA. *Photo* by Al Hicks

Fig. 9.2 A hibernating little brown myotis (*Myotis lucifugus*) with typical WNS infection visible on skin tissues. *Photo* by Ryan von Linden

during hibernation (Lorch et al. 2011; Warnecke et al. 2012) (Fig. 9.2). Bats die before spring brings warmer weather and insects for food.

WNS has spread rapidly and by 2014 was found in 25 U.S. states and 5 Canadian Provinces (Fig. 9.3). A confirmed case of WNS is defined by the presence of cupping erosions on the skin caused by infection by *P. destructans*, which is determined by histopathological examination (Meteyer et al. 2009). There are currently seven hibernating species in North America that have been confirmed with infections characteristic of WNS, including *Myotis lucifugus, Myotis septentrionalis, Myotis sodalis, Myotis leibii, Myotis grisescens, Eptesicus fuscus and Perimyotis subflavus*. There are several additional species for which *P. destructans* has been detected on skin tissues using swab sampling and quantitative PCR methods (Muller et al. 2013), but that have not been confirmed with characteristic skin lesions that define the disease.

Two of the species confirmed with WNS (*M. sodalis, M. grisescens*) were already listed as federally endangered under the US Endangered Species Act before WNS emerged and several other species have been predicted to go globally or regionally extinct due to mortality from WNS (Frick et al. 2010; Langwig et al. 2012; Thogmartin et al. 2013). The US Fish and Wildlife Service listed *M. septentrionalis* as federally threatened in 2015 due to the risk of extinction

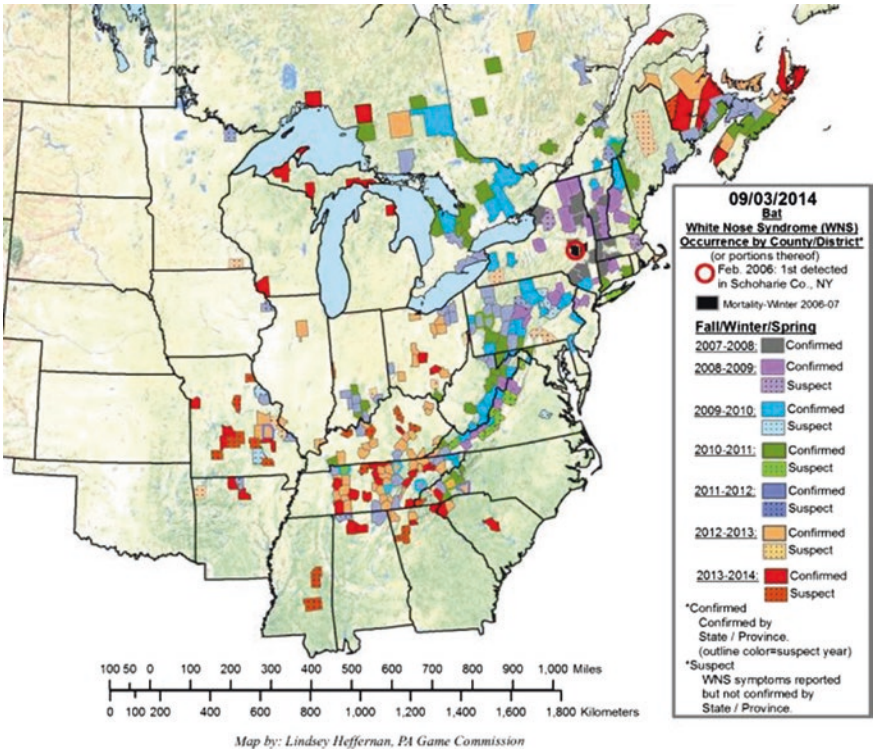

Fig. 9.3 Map of current distribution and past spread of WNS across North America. Confirmed WNS cases are those where disease has been confirmed by histological examination of tissues. Suspect cases are those that are either a molecular detection of *Pseudogymnoascus destructans* by quantitative PCR (Muller et al. 2013) or by visual signs and/or aberrant behaviour consistent with WNS disease at a site. Updated versions of this map are made publically available at whitenosesyndrome.org

from WNS-associated mortality. In addition, a status review of *M. lucifugus* is being conducted to determine whether listing as federally endangered is warranted of this once common species (Frick et al. 2010). In Canada, three species, *M. lucifugus*, *M. septentrionalis* and *P. subflavus* were listed as endangered in 2015. The rapid spread and extensive mortality associated with WNS raise serious concerns about population viability for species that are being impacted by this disease.

In this chapter, we review what is currently known about WNS, focusing on mechanisms of disease, disease ecology, global distribution patterns and conservation and management. We first explain why WNS belongs in a volume addressing bats in the Anthropocene. We review what is known about disease mechanisms, including what we currently understand about the physiology of the disease and immune response in bats. We then review what is currently known about disease ecology of WNS, including the population impacts to species, and then highlight

unanswered questions about transmission dynamics. We discuss global distributions patterns, focusing on what is known about WNS in Europe. We conclude by discussing current conservation and management strategies.

Wildlife disease is increasingly recognized as a major conservation threat (Daszak 2000). Global movements of humans increase the probability and rate at which we introduce pathogens into naïve ecosystems (Cunningham et al. 2003). This human-mediated spread of pathogens has been dubbed "pathogen pollution" to highlight the role of human trade and travel in the spread of wildlife pathogens (Cunningham et al. 2003). The fungus *P. destructans* was presumably introduced to North America from Europe by people, most likely from someone who had visited caves in Europe and subsequently visited Howe's Cave with contaminated boots or gear (Puechmaille et al. 2011c; Leopardi et al. 2015). No bats are known to migrate between the Americas and other continents, implicating human trade or travel in the trans-Atlantic arrival of the fungus (Wibbelt et al. 2010). Ironically, bats are often seen as reservoirs of diseases with consequences to human health (e.g. rabies, SARS, etc.). In the case of WNS, humans were most likely the unwitting transcontinental carrier of a pathogen that has killed millions of bats and now threatens species with extinction.

The emergence of WNS has dramatically changed conservation planning and population monitoring of temperate bats in North America (Foley et al. 2011). On the positive side, this crisis prompted collaborative research efforts among bat conservationists in North America and in Europe. Although mortality from WNS is currently restricted to North America, the pathogen is a potential threat to hibernating bat populations in other parts of the globe and is a global concern for bats in the Anthropocene (Puechmaille et al. 2011c).

9.2 Disease Mechanisms

Challenge or inoculation studies (e.g. Lorch et al. 2011; Warnecke et al. 2012; Wilcox et al. 2014) and comparative studies of bats from affected versus unaffected hibernacula (Moore et al. 2011; Storm and Boyles 2011; Reeder et al. 2012; Brownlee-Bouboulis and Reeder 2013) have led to progress in our understanding of mechanisms underlying WNS. The wings of bats are physiological active tissue involved in gas exchange and fluid balance. In general, results of physiological studies are converging on a consensus that cutaneous infection of the wings accounts for the physiological and behavioural effects of WNS (Cryan et al. 2010).

Lorch et al. (2011) experimentally inoculated the wings of healthy *M. lucifugus* with *P. destructans* for comparison to sham-inoculated controls. They housed bats in temperature- and humidity-controlled incubators that maintained environmental conditions approaching natural hibernacula [82 % relative humidity (RH) at 6.5 °C]. This experiment resolved a critical question by demonstrating that experimental infection with *P. destructans* caused the defining characteristics

of WNS (e.g. cupping erosions in the epidermis associated with fungal growth, Meteyer et al. 2009). They also found that *P. destructans* spread from infected to un-infected bats housed in the same cages but did not spread between cages in the same incubator confirming contact but not airborne transmission of the causal pathogen under laboratory conditions. Lorch et al. (2011) did not detect differences in survival between infected and un-infected bats possibly because the experimental duration was shorter than a typical hibernation season and/or because humidity in this experiment was lower than that of hibernacula used by *M. lucifugus* in the wild, potentially influencing hibernation patterns of both control and infected bats. Warnecke et al. (2012) repeated aspects of Lorch et al.'s (2011) experiment but increased ambient humidity to >97 % RH at 7 °C and ran the experiments for 120 days (vs. 102 days in Lorch et al. 2011). In Warnecke et al.'s (2012) experiment, all sham-inoculated bats survived four months of hibernation, while infected bats exhibited a significant increase in the frequency of periodic arousals, reduced fat reserves and reduced survival, thus confirming that infection with *P. destructans* alone causes the pathology that defines WNS, altered torpor behaviour and mortality. A field study comparing arousal frequency of bats in affected versus unaffected caves (Reeder et al. 2012) also reported a difference in arousal frequency similar to that observed by Warnecke et al. (2012). Together these findings suggest a strong role for increased arousal frequency and altered energy balance in WNS pathophysiology.

Comparisons of control and infected bats have also provided insight into immune responses (or lack of responses) of bats during and after hibernation. Hibernators generally exhibit down-regulated immune function during winter and bat species affected by WNS appear to be no exception (Meteyer et al. 2009, 2012; Moore et al. 2011). During hibernation, there is little evidence of initiation of an inflammatory response or recruitment of immune cells in bats infected by *P. destructans* based on histopathology (Meteyer et al. 2009, 2012). Despite the absence of an inflammatory response, however, variation in other aspects of cellular immunity may have a role to play. Moore et al. (2013) found differences in immunological responses of *M. lucifugus* in affected versus unaffected hibernacula, specifically higher leukocyte counts, reduced antioxidant activity and lower levels of interleukin-4 (an important precursor for differentiation of T-cells) in bats from WNS-affected caves. Although comparisons between populations of bats in different hibernacula are challenging to interpret because of the potential for underlying differences between bats independent of infection, these findings suggest that even the hardest-hit bat species attempt some, albeit weak, immune response to *P. destructans* infection. This also raises the possibility that some bats may be better equipped to resist infection than others (Puechmaille et al. 2011c) with the potential for directional selection on immune function if these differences are heritable and provide a survival advantage.

Immune responses of bats to WNS could be as much a disadvantage as an advantage. Meteyer et al. (2012) recently reported the disheartening paradox that some survivors of WNS exhibit characteristic signs of immune reconstitution inflammatory syndrome (IRIS). When infected bats emerge from hibernation and their immune

function resumes, they exhibit a massive neutrophilic inflammatory response to the fungal infection. This response appears to dramatically increase tissue damage and may reflect an over-reaction to infection because euthermic body temperatures in spring would likely be sufficient to combat the fungal infection (Chaturvedi et al. 2010; Puechmaille et al. 2011b; Verant et al. 2012). The response is likely energetically expensive and the resulting wing damage could compromise flight ability and, therefore, spring energy balance by increasing healing and immunity costs, while reducing potential foraging efficiency at a time when energy balance is critical to support reproduction. Further studies of the role of IRIS in the ecology of WNS are essential for understanding the potential for populations to recover from WNS.

A down-regulated immune response in hibernating bats generally, combined with increased arousal frequency (Boyles and Willis 2010; Reeder et al. 2012; Warnecke et al. 2012) and possibly increased metabolic rate and body temperature during torpor following infection (Storm and Boyles 2011; Verant et al. 2014), appears to result in premature fat depletion and starvation. However, why fungal infection would increase arousal frequency is still not fully understood. Cryan et al. (2010) proposed the hypothesis that fungal damage to the wings of bats could lead to increased evaporative water loss (EWL) across damaged epidermis. Rates of EWL during torpor are a strong predictor of arousal frequency in hibernators (Ben-Hamo et al. 2013; Thomas and Cloutier 1992; Thomas and Geiser 1997) so an increase in EWL or fluid loss due to skin damage from infection by *P. destructans* could lead to the observed effects on arousals. Willis et al. (2011) used data on water loss and arousal frequency in healthy bats, combined with an individual-based model quantifying survival of hypothetical populations of bats, to demonstrate that even a small increase in EWL resulting from infection could cause the same patterns of arousal and mortality observed for infected bats, thus highlighting the plausibility of the dehydration hypothesis.

Two independent datasets from both captive and free-ranging bats also support a role for dehydration and fluid loss in WNS pathophysiology (Cryan et al. 2013; Warnecke et al. 2013). In addition to high hematocrit levels consistent with dramatic fluid loss, Cryan et al. (2013) and Warnecke et al. (2013) both found evidence of electrolyte depletion (with no evidence of renal pathology), consistent with hypotonic dehydration due to fluid loss across damaged wings. Presumably infected bats lose fluid containing both water and electrolytes across injured wing tissue but can only replenish or partially replenish water stores by drinking, because electrolytes are not available in hibernacula. Warnecke et al. (2013) also found preliminary evidence of a respiratory response to metabolic acidosis in infected bats which they hypothesized reflect reduced perfusion of infected tissues, localized anaerobic metabolism and acidosis, and increased respiratory rate to increase CO_2 excretion and counter acidosis. In addition to increased arousal frequency, these physiological responses also predict increased metabolic costs and elevated body temperature during torpor. To date, measurements of torpid body temperature with enough precision to test this hypothesis are unavailable but these would be valuable, especially alongside measurements of metabolism during torpor and arousal in infected versus un-infected bats.

Other physiological mechanisms could also be at play. Willis and Wilcox (2014) reviewed three (of many potential) hormone systems that could be influenced by WNS, both within individuals and via selection on traits which could favour survival. For example, the lipostat hormone leptin is strongly associated with winter energy balance and pre-hibernation fattening. Bats must enter a state of leptin resistance during fall to accumulate adequate fat stores to survive the winter. If, as the evidence suggests, WNS represents a challenge for hibernation endurance, bats with the greatest leptin resistance (and therefore potential fat stores) in autumn may be best equipped to survive increased arousals associated with WNS (Willis and Wilcox 2014). Interactions between WNS and other hormone systems important for seasonal energetics, body temperature regulation and energy and fluid balance (e.g. glucocorticoids, melatonin, thyroid hormone, vasopressin, androgens) could also play important roles in disease dynamics and evolution of remnant populations and are worth further study.

In addition to physiological research, recent studies have also examined behavioural mechanisms associated with WNS that could reflect either adaptive responses to disease or maladaptive pathological responses. Langwig et al. (2012) reported that a much greater proportion of the *M. lucifugus* surveyed in WNS-affected caves after the emergence of the disease were hibernating solitarily (i.e. without clustering) compared to bats surveyed before WNS. This could reflect a behavioural change by individuals following infection or selection by WNS for bats which tend to roost individually (Langwig et al. 2012). Wilcox et al. (2014) reported behavioural observations of bats inoculated with *P. destructans* and found evidence supporting the former hypothesis. Infected bats gradually reduced their clustering behaviour as hibernation progressed. Wilcox et al. (2014) also observed a reduction in behavioural activity during arousals, in general, for affected bats. Taken together, reduced clustering and reduced activity by infected bats could reflect general patterns known as "sickness behaviour", a coordinated response to infection characterized in part by lethargy presumably to save energy for immune responses (Adelman and Martin 2009). These behaviours could also reduce the potential for transmission among individuals in a social group within a hibernaculum. Even bats that have already been infected with *P. destructans* could benefit by reduced subsequent exposure to other infected individuals because new contacts could lead to additional areas of infection in the wings, exacerbating disease severity. On the other hand, reduced clustering behaviour could increase energy expenditure and EWL leading to negative consequences for survival. More work is needed to understand the survival consequences of a range of physiological and behavioural responses to WNS.

9.3 Disease Ecology of WNS

One of the defining characteristics of WNS is that it is a multi-host disease, meaning that *P. destructans* infects multiple bat species. Although all hibernating bat species in northeastern North America can be infected with *P. destructans* and

develop the cupping erosions in their skin tissues that characterize the disease, population impacts from WNS vary widely among species (Langwig et al. 2012; Turner et al. 2011). Prior to the emergence of WNS in North America, all six hibernating bat species that occur in the northeastern United States had positive population growth trends (Frick et al. 2010; Langwig et al. 2012). With the emergence of WNS, four of these six species suffered severe population declines (*M. septentrionalis, M. lucifugus*, *M. sodalis* and *P. subflavus*) (Langwig et al. 2012). Two species (*M. leibii* and *E. fuscus*) have experienced less severe impacts from disease (Langwig et al. 2012). In addition, species of the genus *Corynorhinus* do not appear to get sick and die from WNS, despite occurring in WNS-affected caves in states in the mid-Atlantic region, such as West Virginia and Virginia. Why some species suffer higher mortality than others is an important area of current research, but there are no clear-cut answers yet. Langwig et al. (2012) showed that differences in roosting microclimates (temperature and RH) were correlated with differential impacts among sites for some species. For example, sites with warmer roosting temperatures had the highest declines for *M. lucifugus* and sites with highest RH had the highest declines for *M. sodalis*, suggesting that roosting microclimates could play an important role in WNS impacts (Langwig et al. 2012). Differences in environmental conditions as well as exposure, transmission, susceptibility, torpor physiology and immune response among species could contribute to observed differences in mortality. Future research focusing on differences in these factors among species will be critical for identifying the risks to particular species.

Understanding whether transmission is dependent on the density of hibernating populations is key to determining whether WNS will cause bats to go extinct or whether bat populations will stabilize at low numbers. For diseases where transmission is density-dependent, the probability of extinction is much lower because transmission rates decline as populations become smaller (De Castro and Bolker 2004). Langwig et al. (2012) showed that for bats that hibernate in dense clusters (e.g. *M. lucifugus* and *M. sodalis*), there was no evidence for density-dependent declines, meaning that declines from WNS were equally severe in populations that ranged from 100 to 100,000 bats. In contrast, there was evidence that declines were smaller in smaller populations for species that roost solitarily (e.g. *P. subflavus* and *M. septentrionalis*). Although the declines were density-dependent in *M. septentrionalis*, declines were not predicted to stabilize before populations went extinct in this species, suggesting that this species is at serious risk of extinction from WNS.

Determining whether a pathogen can persist in an environmental reservoir is also important for understanding disease transmission dynamics and extinction risk from disease (De Castro and Bolker 2004). Pathogens that can persist in an environmental reservoir are more likely to drive species extinct because hosts can get infected from the environment even if only a few individuals remain. Studies have shown that *P. destructans* is found in sediments and environmental substrates in hibernacula (Puechmaille et al. 2011a; Lindner et al. 2011; Lorch et al. 2013a, b). Lorch et al. (2013b) demonstrated that viable *P. destructans* can be cultured

from samples taken during late summer when bats have been absent for several months, suggesting that *P. destructans* persists in the environment between hibernation seasons. An unpublished experiment conducted by Al Hicks at the New York Department of Environmental Conservation demonstrated that naïve bats that had never been exposed to *P. destructans* could contract disease and die from WNS when placed in an infected hibernaculum with no access to other infected bats (Hicks, pers. comm.). The evidence to date suggests that hibernacula are environmental reservoirs for *P. destructans*, which has potentially dire consequences if the environment proves a major source of transmission.

WNS is a seasonal disease and recent work by Langwig et al. (2015) describes how the seasonal patterns of transmission of *P. destructans* are driven by hibernation. Bats begin to become infected in the fall when they return to hibernacula during fall swarm and transmission spikes in early winter once bats begin hibernating. Infection intensity increases during hibernation and peaks in late winter at which time most bats have become infected. These seasonal patterns are similar to temporal prevalence of visual signs of *P. destructans* growth on bats at sites in Europe as described by Puechmaille et al. (2011a), where a peak of infection was also observed in late hibernation when most individuals present were infected. In Langwig et al.'s study, most bats cleared infection during summer and prevalence of infection fell to zero by late summer at maternity roosts. The seasonal timing of infection suggests that mortality occurs at a time of maximal impact for populations (before the birth pulse). However, a peak in transmission after bats begin hibernating in early winter may reduce the rate of spread among hibernacula since bats presumably move among sites less frequently once they start hibernating compared to during the fall swarm period.

9.4 Status of *P. Destructans*/WNS in Europe

In contrast to the severe impacts WNS has on North American bat species, *P. destructans* is commonly found on bats in Europe but is not associated with mass mortality (Wibbelt et al. 2010; Puechmaille et al. 2011a). Europe is a putative source of the pathogen and the pathogen likely arrived in North America by some means of human trade or travel. Ongoing studies on global distribution of *P. destructans* (S.J. Puechmaille and J.R. Hoyt, unpublished data), including surveys in temperate Asia, may reveal important insights about the global distribution of the pathogen.

Pseudogymnoascus destructans was first reported in Europe by Puechmaille et al. (2010) who sampled a hibernating *Myotis myotis* from southwestern France showing the typical powdery white fungal growth on its nose. Since then, the fungus has been morphologically and genetically confirmed in 14 countries in Europe (France, Portugal, Belgium, The United Kingdom, The Netherlands, Germany, Switzerland, Austria, Slovakia, Poland, Hungary, Ukraine and Estonia) and

convincing photographic evidence further supports its presence in an additional four countries (Luxembourg, Denmark, Romania and Turkey [the European part]) (Martínková et al. 2010; Puechmaille et al. 2010, 2011a; Kubátová et al. 2011; Simonovicová et al. 2011; Mestdagh et al. 2012; Wibbelt et al. 2010, 2013; Burger et al. 2013; Paiva-Cardoso et al. 2014; Sachanowicz et al. 2014). At the continental scale, most European reports are from northeastern France through Belgium, the Netherlands, Germany and the Czech Republic, but it remains unclear whether this pattern of higher prevalence of the fungus is real or reflects sampling bias (Puechmaille et al. 2011a). Studies conducted in Italy, Slovenia and Sweden, where *P. destructans* was not detected (Voyron et al. 2010; Nilsson 2012; Mulec et al. 2013), support the hypothesis that *P. destructans* occurrence and/or prevalence varies between different geographic regions in Europe (Puechmaille et al. 2011a).

Puechmaille et al. (2011a) demonstrated that the prevalence of visible signs of *P. destructans* on bat wings and nose drastically varied through the hibernation period with the first cases appearing around mid-January. The number of cases increased to reach a peak in March and declined as bats emerged from hibernation. This pattern further complicates comparisons of prevalence of visual signs of fungal growth on bats between sites, regions or years unless surveys are carried out at the same time. Work done in the Czech Republic and Slovakia detected differences in prevalence of bats suspected to carry *P. destructans* (based on visual observations) between sub-mountain humid to mesic regions (higher prevalence) and mountainous and limestone regions (lower prevalence) (Martínková et al. 2010), supporting the idea that *P. destructans* is not equally abundant across Europe. Nevertheless, the differences in sampling strategy (spatio-temporal), sampling intensity (number of sites, number of samples), nature of the samples collected (e.g. swab from the bat vs. environment vs. guano) and analysis techniques (e.g. culture, PCR detection) between different European studies make quantification of these fine- and large-scale patterns challenging (Puechmaille et al. 2011a).

All confirmed cases of *P. destructans* infection come from fungal material collected on bats with the exception of a case from Estonia where the fungus has been isolated and cultured from the walls of the hibernation site, representing the first published isolation of viable spores from the environment in Europe or North America (Puechmaille et al. 2011a). In terms of species, available data suggest that *M. myotis* is the most commonly infected species (ca. 66 % of cases) with *P. destructans* in Europe (Martínková et al. 2010; Puechmaille et al. 2011a). The fungus is known to also infect another nine species of European *Myotis* (ranked by decreasing order of prevalence): *M. dasycneme, M. mystacinus, M. blythii, M. daubentonii, M. brandtii, M. emarginatus, M. nattereri, M. bechsteinii* and *M. escalerai*/sp. *A*. The list of species with *P. destructans* infection is likely to increase as sampling intensity increases as illustrated by the recent Zukal et al. (2014) study which reported infection of a few individuals from three more species of the family Vespertilionidae, *Eptesicus nilssonii*, *Plecotus auritus* and *Barbastella Barbastellus*, as well as on a single individual of *Rhinolophus hipposideros*, of the family Rhinolophidae.

Owing to the protection of bats across Europe and the absence of mass mortality, only three studies with limited to moderate numbers of samples have investigated the pathology of *P. destructans* during the hibernation period (Pikula et al. 2012; Wibbelt et al. 2013; Bandouchova et al. 2015). In Europe, *P. destructans* invasion of the wing membrane is generally restricted to the epidermis and adnexae without deep invasion into the underlying connective tissue but with occasional formation of neutrophilic pustules, contrasting with the common and extensive invasion of dermal connective tissue in bats from North America (Pikula et al. 2012; Wibbelt et al. 2013; Zukal et al. 2014; Bandouchova et al. 2015). Based on investigation of two euthanized individuals, *P. destructans* invasion in the skin of the muzzle seems to be more pronounced than invasion of the wing membrane (Pikula et al. 2012; Wibbelt et al. 2013). As damage to the skin of the muzzle may not be as physiologically important for homeostasis as damage to the wing membranes (Cryan et al. 2010; Reeder et al. 2012; Warnecke et al. 2013), we suggest that it may be important to differentiate the pathology of *P. destructans* on the wing and on the muzzle. If dehydration and fluid loss play an important role in WNS pathophysiology, quantifying wing damage consistently (e.g. following Reeder et al. 2012 or an alternative scoring system) alongside physiological measures of disease severity will be critical for a better understanding of the disease, its progression and species-specific attributes, compared to the commonly reported dichotomous presence/absence of the disease.

The term WNS was originally used to describe the symptoms associated with bats in the field before the disease was fully characterized as a cutaneous infection of skin tissues by the pathogenic fungus, *P. destructans* (Blehert et al. 2009; Meteyer et al. 2009). As such, the name ‘WNS’ has changed from referring to a set of symptoms, including visible fungal growth on skin surfaces, depletion of fat reserves, altered torpor patterns and aberrant winter behaviour (Blehert et al. 2009) to referring to the presence of disease as defined by the presence of cutaneous infection characterized by cupping erosions (Meteyer et al. 2009). This has led to confusion and some debate about whether the term WNS should be used to describe infections occurring in Europe, which are pathologically similar to those in North America but which do not include mass mortality or aberrant winter behaviour (Puechmaille et al. 2011a). Despite its original definition as a syndrome (Veilleux 2008; Reeder and Turner 2008; Turner and Reeder 2009), the term WNS is now routinely used to refer the cutaneous infection caused by *P. destructans*, which have been documented in Europe (Pikula et al. 2012; Wibbelt et al. 2013; Zukal et al. 2014). Some have advocated a name change to clarify a difference between a ‘syndrome’ and a ‘disease’ caused by fungal infection (Chaturvedi and Chaturvedi 2011). Inconsistency in the literature could lead to confusion but recent use of the term white-nose disease (WND; Paiva-Cardoso et al. 2014) could clarify the situation by providing terminology reminiscent enough of WNS to avoid confusion but technically consistent with the definition of a disease.

Recent work comparing colony sizes of hibernating vespertilionid bats in North America before and after the emergence of WNS, to current colony sizes in Europe, reveals an intriguing pattern. Before WNS emerged in North America,

colony sizes of hibernating bats were, on average, about 10-fold larger than those of similar species in Europe (Frick et al. 2015). However, after the emergence of WNS, colony sizes in eastern North America are no longer statistically different from those in Europe (Frick et al. 2015), raising the following question: Were hibernating bat colonies in Europe once much larger prior to the emergence of WNS there? If WNS is indeed acting as a hidden force on bat populations in Europe, then small winter colony sizes in eastern North America may become the norm for species in North America that manage to persist. However, Frick et al. (2015) also show that 69 % of winter colonies of *M. septentrionalis* were entirely eliminated within 7 years of WNS detection, suggesting that this species is rapidly disappearing from the landscape. The predicted extinction of *M. septentrionalis* from WNS begs the question whether past extinctions of bat species may have also occurred in Europe.

9.5 Conservation and Management

Conservation and management strategies for WNS in North America have focused primarily on preventing spread of the pathogen to new areas through decontamination protocols as well as cave closures to limit the potential for human-mediated spread. Decontamination of gear used in hibernacula by both recreational cavers and bat researchers is an important management strategy to reduce the risk of spread of *P. destructans* by humans. *P. destructans* spores have been found on field gear after use in infected sites and therefore utmost precaution is needed to reduce the chance that researchers and cavers spread *P. destructans* to new areas. Cave closures have been controversial and have been met with some resistance by some members of the caving community. Some cave closures have subsequently been relaxed in parts of the western United States where *P. destructans* has not yet spread. Determining whether cave closures are effective can be challenging given that the absence of spread in areas is hard to measure. Bats are capable of spreading the fungus, but the primary focus of closing caves and advocating decontamination was to slow spread by people, especially to distant locations.

Finding a treatment for infected bats has proved elusive and difficult. Several studies have examined the efficacy of treating bats with anti-fungal chemicals, such as terbinafine, but none have shown any promise. There has also been interest in alternative forms of treatment, including use of naturally occurring bacteria (Fritze et al. 2012; Hoyt et al. 2015) or volatile compounds (Cornelison et al. 2014). Recent work by Cornelison et al. (2014) showed that a volatile organic compounds (VOCs) inhibited growth of *P. destructans* in vitro. Similarly, a recent study by Hoyt et al. (2015) showed that *Pseudomonas* bacteria that naturally occur on hibernating bats inhibit growth of *P. destructans* in vitro. Other strains of *Pseudomonas* found in Europe have shown similar results (Fritze et al. 2012). Research on these biological control treatment options is still in early stages and although early lab results have shown promise, experimental and field trials will

need to be conducted before the efficacy of these approaches is fully evaluated. The WNS research and management community is developing standards and protocols for evaluating the safety and efficacy of biological treatment options.

Other ideas for active management have included building artificial hibernacula that can be cleaned and decontaminated each summer between hibernating seasons. An experimental artificial hibernacula was built in Tennessee and existing military bunkers have been used as artificial hibernaculum in the northeastern US. The goal of these structures is to provide a place for bats to hibernate that does not serve as an environmental source of transmission when bats re-enter the hibernaculum in fall. To date there have been no studies to determine whether bats will use these artificial hibernacula naturally and whether survival will be improved in these sites.

Given what we know about the potential role that electrolyte depletion plays in the physiology of the disease, some researchers have also explored the potential for electrolyte therapy for hibernating bats by providing access to electrolyte supplements during hibernation. Experimental trials to test this are underway. Finally, bats are very difficult to breed in captivity and, while the prospect of captive breeding and management of bats has been explored, it remains doubtful whether this approach could be useful as a management tool for bat species affected by WNS. However, if breeding programmes could be developed, they could provide a supply of animals for laboratory studies to reduce potential impacts of research on wild populations.

9.6 Conclusions

Although we have learned a great deal about WNS in the past seven years, there are still many unanswered questions about disease mechanisms, ecology, transmission dynamics, long-term impacts, global distribution patterns and potential treatment options that will be important for managing WNS and its impacts on bats. The US Fish and Wildlife Service has been pivotal in terms of coordinating meetings for information exchange among researchers and state biologists as well as directly funding much of the research on WNS in both the US and Canada. Research priorities for management and conservation of species have focused on topics such as establishing that *P. destructans* was the causative agent of infection, trying to identify potential treatment of infection, the physiology of infection and mechanisms of mortality, characterizing the environmental reservoir and understanding transmission and immunological response.

For many of us, working on WNS is a grim business. There is nothing quite like the experience of going underground and entering a chamber that was formally home to thousands of bats and seeing empty walls and a few straggling survivors covered in white fungus. However, the sense of commitment within the WNS community and the dedication of researchers and managers to try and find new ways to understand and solve this crisis provide a certain hope. We have yet to find

a way to stop bats dying from WNS, but we are trying hard to do so. Whether we are able to prevent species extinctions may rely, in part, on the creativity to find solutions before it is too late and the willingness of agency biologists to implement creative solutions without clear assurances of outcomes.

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Chapter 10
Zoonotic Viruses and Conservation of Bats

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Abstract Many of the recently emerging highly virulent zoonotic diseases have a likely bat origin, for example Hendra, Nipah, Ebola and diseases caused by coronaviruses. Presumably because of their long history of coevolution, most of these viruses remain subclinical in bats, but have the potential to cause severe illnesses in domestic and wildlife animals and also humans. Spillovers from bats to humans either happen directly (via contact with infected bats) or indirectly (via intermediate hosts such as domestic or wildlife animals, by consuming food items contaminated by saliva, faeces or urine of bats, or via other environmental sources). Increasing numbers of breakouts of zoonotic viral diseases among humans and livestock have mainly been accounted to human encroachment into natural habitat, as well as agricultural intensification, deforestation and bushmeat consumption. Persecution of bats, including the destruction of their roosts and culling of whole colonies, has led not only to declines of protected bat species, but also to an increase in virus prevalence in some of these populations. Educational efforts are needed in order to prevent future spillovers of bat-borne viruses to humans and livestock, and to further protect bats from unnecessary and counterproductive culling.

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10.1 Introduction

Over the past decades, the emergence of zoonotic viruses (those that are naturally transmitted between vertebrate animals and humans) from bats has been the subject of increasing attention from both scientists and the general public (e.g. Quammen 2013). During outbreaks of diseases in humans and livestock, bats are now often the primary focus of searches for a reservoir host (Chua et al. 2002a; Leroy et al. 2005; Li et al. 2005; Halpin et al. 2007; Towner et al. 2007; Lau et al. 2010; Wibbelt et al. 2010; Memish et al. 2013). Identification of bats as natural hosts for emerging viruses has important implications for bat conservation. We review the current state of research of four important families of emerging zoonotic viruses for which bats are natural reservoir hosts and discuss direct and indirect conservation implications.

10.2 Emerging Viral Diseases: Why Bats?

Although bats have been identified as carriers of many highly virulent human pathogens (Chen et al. 2014), evidence of pathogen-related clinical signs or disease in bats is scarce, particularly for intracellular pathogens such as viruses (Brook and Dobson 2015). Post-infection survival is supported by the frequent identification of antibodies to known viruses in apparently healthy bats and long-term survival of these bats (e.g. Hayman et al. 2010). Additionally, viruses isolated or genetically detected from bat populations are highly diverse and often ancestral to related viruses in human and other mammalian species (e.g. Towner et al. 2009; Drexler et al. 2012; Baker et al. 2013a; Tong et al. 2013; Vidgen et al. 2015). Together, these findings suggest a long history of coevolution between many bat-virus relationships identified to date. Recent progress in the field of bat immunology and genomics has identified key differences in bat immunity and physiology that evolved concomitantly with the evolution of flight, resulting not only in apparently increased immunotolerance of intracellular pathogens, but also in increased longevity and decreased tumour production (Baker et al. 2013b; Zhang et al. 2013; Brook and Dobson 2015). Immunotolerance and incomplete clearance of viral infections are also likely to favour the establishment of persistent infections (Virgin et al. 2009), as proposed for a number of bat-borne viruses (Plowright et al. 2015).

Various ecological and life-history factors play a key role in the susceptibility of individuals and populations to pathogens (Allen et al. 2009; Turmelle et al. 2010; Schneeberger et al. 2013), and notable differences exist between bats and terrestrial mammals such as rodents (Luis et al. 2013). For example, the often high-population densities and the usually gregarious roosting behaviour of bats increase the likelihood of both intra- and interspecies transmission of viruses (Luis et al. 2013; Streicker et al. 2010). Large-scale movements of bats due to

their ability for powered flight are also likely to facilitate viral transmission within and among species, including the exchange of novel viruses and virus variants across biomes or even continents (Calisher et al. 2006; Epstein et al. 2009; Peel et al. 2013). The extreme relative longevity of bats compared to other mammals of similar size (Wilkinson and South 2002) and the potential for persistent and/or subclinical viral infections could further increase transmission potential (Calisher et al. 2006). Reduction of body temperature associated with hibernation of temperate zone bats lowers both viral activity and the metabolism of hosts, leading to increased incubation periods and therefore reduced likelihood of epizootic fadeout (of rabies, for example; George et al. 2011). Bats are ancient mammals in evolutionary terms, and virus utilisation of highly conserved cellular receptors could facilitate transmission to other mammals (Calisher et al. 2006), for example, as has been suggested for henipaviruses (Negrete et al. 2005). Lastly, it was recently speculated that, similar to the febrile response of other mammals, the relatively high body temperature (about 38–41 °C) and metabolism of bats during flight may select for viruses tolerant to such conditions, meaning the normal febrile defence mechanism of other mammals is ineffective ("Flight as fever hypothesis", O'Shea et al. 2014), making bat-borne viruses potentially more virulent and lethal for other, non-flying mammals.

10.3 Zoonotic Viruses of Bats and Their SpillOver

10.3.1 Rhabdoviruses

Rabies virus (RABV) is the longest and best-known member of the genus *Lyssavirus* (family Rhabdoviridae) and still one of the most significant zoonoses known from bats (recent reviews include: Banyard et al. 2011; Banyard et al. 2014 and Kuzmin 2014). The genus is rapidly expanding, with 14 of the currently recognised species (plus another known from genetic material only), and all but two (Mokola and Ikoma viruses) having been isolated from bats (Table 10.1). Lyssaviruses spill over directly from bats to domestic animals, other wildlife and humans, or indirectly to humans via these other species. All lyssaviruses are potentially neurotropic, meaning that the virus infects nerve cells and replicates in the brain, resulting in clinical signs consistent with classical rabies (Schnell et al. 2009). Although isolated from a variety of tissues and body fluids in the late stages of infection, the predominant route of transmission is via saliva (mostly via biting; Kuzmin 2014).

Lyssaviruses can be divided into two distinct "phylogroups" (Badrane et al. 2001, Table 10.1), reflecting biological and genetic differences, and they are distributed globally in bats. Classical rabies virus occurs in bats across North, Central and South America (Messenger et al. 2003; Banyard et al. 2011) and was first associated with vampire bats following an outbreak in cattle in South America in 1911 (Carini 1911). It is reported most frequently in the common

Table 10.1 Known lyssaviruses and their association with different bat species (adapted from Banyard et al. 2014)

Geographical distribution	Lyssavirus species	Phylogroup	Bat species most commonly associated with lyssavirus infection	Common name	Known human cases
The Americas	Rabies virus (RABV)	I	*Eptesicus fuscus*	Big brown bat	Yes
			Tadarida brasiliensis	Mexican/ Brazilian free-tail bat	
			Lasionycteris noctivagens	Silver-haired bat	
			Perimyotis subflavus	Tri-coloured bat	
			Desmodus rotundus	Vampire bat	
Eurasia	European bat lyssavirus type 1 (EBLV-1)	I	*Eptesicus serotinus*	Serotine bat	Yes
	European bat lyssavirus type 2 (EBLV-2)	I	*Myotis daubentonii*	Daubenton's bat	Yes
	Bokeloh bat lyssavirus (BBLV)	I	*Myotis nattereri*	Natterer's bat	No
	Aravan virus (ARAV)	I	*Myotis blythi*	Lesser mouse-eared bat	No
	Irkut virus (IRKV)	I	*Murina leucogaster*	Greater tube-nosed bat	Yes
	Khujand virus (KHUV)	I	*Myotis mystacinus*	Whiskered bat	No
	West Caucasian bat virus (WCBV)	NA[a]	*Miniopterus schreibersii*	Common bent-wing bat	No
	Lleida bat lyssavirus (LLEBV)	NA[a]	*Miniopterus schreibersii*	Common bent-wing bat	No
Africa	Duvenhage virus (DUVV)	I	*Miniopterus sp?*	Undefined	Yes
			Nycteris thebaica	Egyptian slit-faced bat	
	Lagos bat virus (LBV)	II	*Eidolon helvum*	Straw-coloured fruit bat	No
			Rousettus aegyptiacus	Egyptian fruit bat	
			Epomorphorus wahlbergi	Wahlberg's epaulleted fruit bat	

(continued)

Table 10.1 (continued)

Geographical distribution	Lyssavirus species	Phylogroup	Bat species most commonly associated with lyssavirus infection	Common name	Known human cases
	Mokola virus (MOKV)	II	not detected		Yes
	Shimoni bat virus (SHIBV)	II	*Hipposideros commersoni*	Commerson's leaf-nosed bat	No
	Ikoma virus (IKOV)	NA[a]	not detected		No
Australasia	Australian bat lyssavirus (ABLV)	I	*Pteropus scapulatus*[b]	Little red flying fox	Yes
			Saccolaimus flaviventris	Yellow-bellied sheath-tailed bat	
			Pteropus alecto	Black flying fox	

[a]Lyssaviral phylogenies infer WCBV, LLEBV and IKOW that are more genetically distinct from other species, and they have not yet been assigned a phylogroup (Kuzmin 2014)
[b]Barrett (2004)

vampire bat (*Desmodus rotundus*; Kuzmin et al. 2011a), which has a wide distribution across Mexico, Central America, and South America. Bites from this species appear to be responsible for the majority of human and domestic animal rabies infections of bat origin in South and Central America, with increased prey availability via expansion of livestock into new areas across the region hypothesised to be contributing to increasing incidences (Schneider et al. 2009; Ruiz and Chávez 2010). In Canada and the USA, 51 cases of human rabies transmitted by non-haematophagous bats were recognised or inferred between 1951 and 2006 (mostly silver-haired bats (*Lasionycteris noctivagans*), eastern pipistrelle bats (*Perimyotis subflavus*) and Brazilian/Mexican free-tailed bats (*Tadarida brasiliensis*)) (Constantine and Blehert 2009; Banyard et al. 2011). However, across the Americas, only 15 % of human rabies cases between 1993 and 2002 were reported as resulting from encounters with bats (Belotto et al. 2005).

Reported antibody prevalences against RABV in *D. rotundus* include 3–28 % in Peru (Streicker et al. 2012) and 12 % in Brazil (Almeida et al. 2011). Depending on the year, location and species, prevalence in other bats varies from relatively low 2 % in *T. brasiliensis* in New Mexico (Steece and Altenbach 1989) and 2.5 % in the little brown bats (*Myotis lucifugus*) in New York (Trimarchi and Debbie 1977), to 58 % in Seba's short-tailed bat (*Carollia perspicillata*) in Peru (Salmón-Mulanovich et al. 2009) and 67 % in *T. brasiliensis* in Texas (Baer and Smith 1991). As with other lyssaviruses discussed below, the potential for high antibody prevalences in bat populations and infrequent reports of mortality suggest that many individuals exposed to the virus survive, contrary to the overwhelmingly lethal nature of lyssavirus infections in other mammalian species (reviewed in Banyard et al. 2011). The mechanisms for this remain unclear.

Seven bat lyssaviruses have been isolated in Eurasia (Table 10.1). **European bat lyssavirus** type 1 and type 2 (EBLV-1 and EBLV-2; Bourhy et al. 1992) are the most widely recognised and studied. Five fatal cases of human infections with EBLV have so far been reported, three from EBLV-1 (Roine et al. 1988; Selimov et al. 1989; Botvinkin et al. 2005) and two from EBLV-2 (Lumio et al. 1986; Fooks et al. 2003; Nathwani et al. 2003). Spillover of EBLV-1 into other mammals has also been observed, but rarely, with examples including zoo bats (Rønsholt et al. 1998), sheep (Tjørnehøj et al. 2006), domestic cats (Dacheux et al. 2009) and a stone marten (Müller et al. 2004). While EBLV-1 and EBLV-2 have been detected in a range of bat species (reviewed in Schatz et al. 2013), they are most frequently associated with serotine bats (*Eptesicus serotinus*) and Daubenton's bat (*Myotis daubentonii*), respectively. The dynamics of EBLV infections in their natural hosts is poorly understood, but banding and recapture data and the frequent capture of apparently healthy bats with antibodies against EBLV suggest that many bats survive infection (Serra-Cobo et al. 2002; Amengual et al. 2007; Schatz et al. 2013). In cases where bats develop clinical symptoms of EBLV infection, the affected individuals are often unable to fly, are generally weak and show abnormal behaviour, including attempts to bite (Banyard et al. 2011). Experimental studies suggest that variable development of clinical signs may be related to inoculation route and dose (reviewed in Banyard et al. 2011).

Comparatively, little is known about the remaining Eurasian bat lyssaviruses, which have each been isolated from bats only once: West Caucasian bat virus (WCBV, Botvinkin et al. 2003), Bokeloh bat lyssavirus (BBLV, Freuling et al. 2011), Aravan virus (ARAV, Kuzmin et al. 1991), Irkut virus (IRKV, Botvinkin et al. 2003) and Khujand virus (KHUV, Kuzmin et al. 2001), or is only known from partial genetic sequence data (Lleida virus, Ceballos et al. 2013, Table 10.1). Of these, only IRKV has been detected in other mammals (a human who developed rabies after a bat bite, Leonova et al. 2009). WCBV appears to have a large geographical range. It was isolated from *Miniopterus schreibersii* in Russia, but cross-reactive antibodies have also been detected in *Miniopterus* bats in Kenya (Kuzmin et al. 2008a). The relatively wide distribution and migratory behaviour of *Miniopterus* spp. may facilitate cross-continental transmission of this virus. Alternatively, given the close relationship between WCBV and Ikoma virus (IKOV), which was recently isolated in neighbouring Tanzania, the serological findings from Kenya could in fact indicate exposure to IKOV or another related lyssavirus rather than WCBV (Marston et al. 2012; Horton et al. 2014). Similarly, serological surveys have detected antibodies against ARAV virus and KHUV virus in Indian flying foxes (*Pteropus giganteus*) from Bangladesh (Kuzmin et al. 2006), and ARAV, KHUV, IRKV or Australian bat lyssavirus in Lyle's flying foxes (*P. lylei*) and dawn bats (*Eonycteris spelaea*) from Thailand (Lumlertdacha et al. 2005). Yet, given the limited lyssavirus surveillance in bats performed to date in this region and that individuals in these studies tested positive to multiple viruses, these results likely represent cross-reactivity of serological assays to unknown lyssaviruses.

Africa also hosts significant lyssavirus diversity, with five species identified, though only three of these isolated from bats to date (Table 10.1). **Duvenhage virus** (DUVV, Meredith et al. 1971) is the only phylogroup I lyssavirus in Africa and is more closely related to RABV, ABLV and the majority of the European species than other known African lyssaviruses. Since it was first isolated from a human in 1970, two more fatal human infections of DUVV have been reported, one in South Africa in 2006 (Paweska et al. 2006) and one from the Netherlands in 2007 after obtaining the infection in Kenya (van Thiel et al. 2008). DUVV has been isolated from bats twice, once from a presumed *M. schreibersii* bat in South Africa and once from an Egyptian slit-faced bat (*Nycteris thebaica*) in Zimbabwe (Schneider et al. 1985; Foggin 1988; Paweska et al. 2006). No further information is so far available on this apparently rare African lyssavirus.

In contrast, **Lagos bat virus** (LBV) is the most widely detected lyssavirus in Africa (Banyard et al. 2011). In 1956, this virus was first isolated from a straw-coloured fruit bat (*Eidolon helvum*; Boulger and Porterfield 1958). Since then, the virus has been isolated and neutralising antibodies detected in a variety of fruit bat species, one insectivorous bat species, domestic cats, domestic dogs and a water mongoose, but not in humans (reviewed in Banyard et al. 2011). *E. helvum* and *Rousettus aegyptiacus* are likely primary reservoir hosts for LBV, with seroprevalences ranging from 6 to 80 % and 29 to 46 %, respectively, depending on the region (Hayman et al. 2008, 2012; Kuzmin et al. 2008b; Dzikwi et al. 2010; Peel et al. 2013). LBV has been isolated from healthy, rabid and dead bats (reviewed in Banyard et al. 2011), but longitudinal studies in Ghana (Hayman et al. 2012) and surveys across continental Africa (Peel et al. 2010, 2013) suggest widespread exposure, no difference in survival between seropositive and seronegative *E. helvum,* and viral persistence in very small, isolated island populations. Early infection experiments with LBV suggested that LBV and other phylogroup II viruses were less pathogenic than other lyssaviruses (Boulger and Porterfield 1958; Badrane et al. 2001). However, recent experimental infections indicated the potential for comparable mortality between LBV and RABV and indicated that significant differences might instead exist between different LBV isolates (Kuzmin et al. 2010; Markotter et al. 2009).

Of the other African lyssaviruses, only Shimoni bat virus (SHIBV) has been detected in bats (Commerson's leaf-nosed bat (*Hipposideros commersoni*) in Kenya; Kuzmin et al. 2010) and only Mokola virus (MOKV) has been detected in humans (on two occasions in Nigeria, Familusi and Moore 1972; Familusi et al. 1972). MOKV has also been isolated from cats and small wild mammals, however, the natural reservoir host is unknown (Nel 2001). Ikoma virus was isolated from a rabid African civet (*Civettictis civetta*), but it is believed that the civet was a spillover host and the true reservoir host is yet to be identified (Horton et al. 2014).

The only lyssavirus detected in Australia to date—**Australian bat lyssavirus** (ABLV)—has two known lineages, one circulating in flying foxes and one in an insectivorous bat (Fraser et al. 1996; Gould et al. 2002; Warrilow 2005). In 1996, shortly after ABLV was first isolated from a black flying fox (*P. alecto*) that was

unable to fly (Fraser et al. 1996), a 39-year-old woman died of clinical rabies after being bitten by a yellow-bellied sheath-tail bat (*Saccolaimus flaviventris*; Gould et al. 2002). Two subsequent human cases have been identified, a woman who died in 1998, 27 months after being bitten by a flying fox (Hanna et al. 2000), and a child who died in 2014 after being scratched by a flying fox (Francis et al. 2014). Experimental infection of grey-headed flying foxes (*P. poliocephalus*) with ABLV resulted in clinical signs of weakness, trembling and limb paralysis in three out of ten individuals (McColl et al. 2002). As with other bat lyssaviruses, a small proportion of ABLV-positive bats succumb to encephalitis-like symptoms (Hooper et al. 1997), yet serological tests show a high prevalence of antibodies in populations of surviving bats (McColl et al. 2000).

10.3.2 Paramyxoviruses

The most notable viruses from the *Paramyxoviridae* family in bats are those of the genus *Henipavirus*, which are the subject of many reviews (e.g. Halpin and Rota 2015; Smith and Wang 2013, Luby and Gurley 2012; Clayton et al. 2013; Middleton and Weingartl 2012; Field and Kung 2011). The first recognised henipavirus, **Hendra virus** (HeV), was first detected during an outbreak of infectious respiratory disease in horses and then humans in Hendra, Australia, in 1994 (Murray et al. 1995). Ultimately, 13 of 20 infected horses died or were euthanised, and of two humans working closely with horses who became infected, one died from acute pneumonia (Murray et al. 1995; Plowright et al. 2015). This spillover was preceded a month earlier by another involving two horses and one human over 800 km away in Mackay, but which went unrecognised until 1995 (Rogers et al. 1996; O'Sullivan et al. 1997). An initial serological survey of 46 wildlife species (excluding bats) failed to identify a reservoir host; however, serological evidence of HeV infection was later identified in all four species of flying foxes native to Australia (Young et al. 1996). Virus isolation (Halpin et al. 2000) and experimental studies (Halpin et al. 2011) have confirmed pteropodid bats as reservoir hosts for henipaviruses (with a lack of clinical signs), with evidence that black (*P. alecto*) and spectacled flying foxes (*P. conspicillatus*) are the main reservoir species for HeV (Smith et al. 2014; Goldspink et al. 2015).

Because HeV is frequently detected in the urine of wild flying foxes (Smith et al. 2014), the predominant transmission route to horses is likely via material recently contaminated with bat urine (e.g. pastures) or via direct transmission (Martin et al. 2015). Recognised spillover events from bats to horses occurred sporadically from 1994 to 2004 and annually since 2006, with five spillover events resulting in ongoing transmission to humans in close contact with horses (a total of seven human cases and four deaths; Field et al. 2010). Spillover events are spatiotemporally clustered, occurring year-round in the northern tropics, but seasonally clustered in winter with a peak in July in subtropical regions (Plowright et al. 2015).

The relative importance of various hypothesised drivers of HeV dynamics in bats and subsequent spillover to horses is still unclear (Plowright et al. 2015).

Nipah virus (NiV), the second henipavirus to be recognised, was first isolated in 1999 from pigs and encephalitic pig workers in Malaysia (Center of Disease Control and Prevention 1999). NiV spillover has not been observed since this time in Malaysia; however, annual seasonal outbreaks with high case fatality (average 73 %) have occurred in people in Bangladesh since 2001 (Hsu et al. 2004; Luby et al. 2009; Luby and Gurley 2012), with occasional spillover also occurring in neighbouring India (Chadha et al. 2006; Harit et al. 2006). Due to the close relatedness of HeV and NiV, fruit bats were targeted, and serological evidence quickly identified them to be the natural reservoir of NiV (Enserink 2000; Yob et al. 2001). This was subsequently supported by isolation of NiV from the urine of *P. hypomelanus* (Chua et al. 2002a), *P. vampyrus* (Rahman et al. 2010) and *P. lylei* (Reynes et al. 2005), and seroconversion in the absence of clinical signs following experimental infections in *P. vampyrus* (Halpin et al. 2011). Antibodies against NiV and NiV-related viruses have now been detected in a variety of bat species (including non-pteropid bats) across a wide geographical area (summarised in Breed et al. 2013). NiV transmission to humans appears to occur via a wider variety of routes compared with HeV. Infection of domestic animal intermediate hosts (via consumption of saliva- or urine- contaminated partially eaten fruits or raw date palm sap) has been implicated as a source of human infections in both Malaysia and Bangladesh (Chua et al. 2002b; Chowdhury et al. 2014). In Malaysia, human infections resulted from direct contacts with infected pigs (Chua et al. 1999; Paton et al. 1999; Parashar et al. 2000), whereas in Bangladesh, transmission to humans regularly occurs via consumption of contaminated date palm sap (Luby et al. 2006; Rahman et al. 2012) or directly from human to human (e.g. via nursing sick individuals or preparation for burial; Hughes et al. 2009). The risk of direct human infection with NiV from bats is considered to be lower than horizontal transmission once the virus enters the human population (Gurley et al. 2007; Luby et al. 2009; Chong et al. 2003).

A third henipavirus, **Cedar Virus** (Marsh et al. 2012), has been isolated from urine collected under a mixed *P. alecto/P. scapulatus* roost in Australia. In contrast to HeV and NiV, however, it appears to be of low pathogenicity and failed to induce clinical signs in experimentally infected laboratory animal species (Marsh et al. 2012). Serological evidence from South-East Asia and Australasia (Breed et al. 2013) and the wide diversity of paramyxovirus sequences detected in Australia (Vidgen et al. 2015) suggest more henipaviruses are yet to be found. Additionally, although henipaviruses were long thought to be restricted to Asia and Australia, antibodies cross-reactive to HeV and NiV were detected in Madagascar in 2007, suggesting a potentially wider geographical distribution of henipa-related paramyxoviruses (Iehlé et al. 2007). This was supported by serological findings and molecular detection of henipa- or henipa-like viruses in mainland Africa and its offshore islands (Hayman et al. 2008, 2012; Peel et al. 2010, 2013; Drexler et al. 2012). Indeed, a recent serological study indicates that these

viruses are also occasionally transmitted to humans in Africa (Pernet et al. 2014), though no African henipavirus has been isolated to date.

Viruses from the paramyxovirus genus *Rubulavirus* (a genus which includes the human mumps virus) have also been frequently detected in bats (Barr et al. 2015). **Menangle virus** was isolated from pigs following the birth of unusually high numbers of stillborn and deformed piglets in Australia (Philbey et al. 1998). Two piggery personnel had neutralising antibodies against Menangle virus after having recovered from an unexplained febrile illness (Philbey et al. 1998). Flying fox colonies roosting in close proximity to the piggeries were a suspected source of infection for pigs, with subsequent transmission to humans (Philbey et al. 1998). This was supported by serological evidence from *P. poliocephalus, P. alecto* and *P. conspicillatus*, and recent virus isolation from *P. alecto* (Barr et al. 2012). Other isolated bat rubulaviruses with unknown or limited understanding of their zoonotic potential include Tioman virus from Malaysia (Chua et al. 2001), Tuhokovirus 1, 2 and 3 from China (Lau et al. 2010), Achimota virus 1 and 2 from Ghana (Baker et al. 2013c) and Hervey, Grove, Teviot and Yeppoon paramyxoviruses from Australia (Barr et al. 2015). Neutralising antibodies to Tioman virus and Achimota viruses have been detected in humans, suggesting previous exposure and infection with the virus (Yaiw et al. 2007; Baker et al. 2013c). Pigs experimentally infected with Tioman virus produced neutralising antibodies and excreted virus in saliva, but were either asymptomatic or developed only a fever (Yaiw et al. 2008). Undetected infection in pigs could therefore facilitate transmission to humans.

Finally, viral fragments related to rubulaviruses and the proposed genus *Jeilongvirus* have also been detected outside the range of fruit bats, in European insectivorous bat species (Kurth et al. 2012). However, nothing is yet known about the relevance of these viruses as potentially zoonotic threats to humans.

10.3.3 Coronaviruses

Bat coronaviruses were first identified from species of the genus *Miniopterus* (Poon et al. 2005), however, with unknown zoonotic potential. The most prominent coronavirus, the one causing **severe acute respiratory syndrome (SARS),** was followed by a pandemic spread in humans after the first outbreak in China in 2002 (Rota et al. 2003). Soon after the outbreak, the virus was detected in masked palm civet (*Paguma larvata*) and raccoon dogs (*Nyctereutes procyonoides*) in a market in Guangdong Province, where SARS was first reported (Guan et al. 2003). A survey of common wildlife species in the area identified bats to be the natural reservoir of SARS coronavirus, with viruses from bats showing greater genetic diversity than the ones isolated from other species, including humans (Li et al. 2005). Bats can regularly be found in markets in China, which makes direct transmission of the virus from bats to humans likely (Li et al. 2005). The followed pandemic spread with 8096 confirmed cases of which 774 were fatal can be

accounted to rapid interindividual transmission of the virus once it entered the human population (World Health Organization 2003).

Outside Asia, **SARS-like coronaviruses** have been detected in the lesser horseshoe bat (*Rhinolophus hipposideros*) from Europe (Rihtarič et al. 2010), in *Chaerephon* sp. from Kenya (Tong et al. 2009) and in *Hipposideros commersoni* from Nigeria (Quan et al. 2010). Antibodies against SARS coronavirus are present in various African bat species (Müller et al. 2007). As with many newly detected viruses, their potential threat as a zoonotic disease is yet unclear.

Since the outbreak of SARS in Asia has been traced to bats as natural hosts of the virus, the same was suspected to be the case for **Middle East respiratory syndrome (MERS)**, an infection that has been occasionally spreading among humans of the Arabian peninsula since 2012 (Zaki et al. 2012). Most human infections have been traced down to close contacts with dromedary camels (*Camelus dromedarius*), which carry a virus with a similar genome organisation as human MERS (Hemida et al. 2014). There is at least one report of direct transmission of the virus from camels to humans via contact with infected animals (Memish et al. 2014). However, a small fragment of a coronavirus PCRed from an Egyptian tomb bat (*Taphozous perforatus*) showed 100 % nucleotide identity to virus from the human index case-patient of MERS, suggesting that this species may be one of the putative natural reservoirs of the virus (Memish et al. 2013). Bat-derived MERS virus has been shown to be able to use human receptors and thus could potentially infect human cells (Yang et al. 2014). However, given the generally low prevalence of MERS virus in bat populations, a direct spillover from bats to humans is unlikely, and transmission probably happens mainly via camels as intermediate hosts (Memish et al. 2013). In fact, no other bat has yet been found to carry MERS virus since the one reported by Memish and colleagues in 2013.

The intensified search for viruses in bats worldwide has led to the detection of coronaviruses other than SARS and MERS, whose potential to be or become zoonotic has yet to be investigated (Woo et al. 2006; Tang et al. 2006; Dominguez et al. 2007; Carrington et al. 2008; Brandão et al. 2008; Misra et al. 2009; Pfefferle et al. 2009; Donaldson et al. 2010; Watanabe et al. 2010; Drexler et al. 2010; Falcón et al. 2011; Annan et al. 2013; Ge et al. 2013; Anthony et al. 2013; Ithete et al. 2013). No clinical symptoms associated with infections with SARS-like and other coronaviruses have yet been described for bats.

10.3.4 Filoviruses

Ebola virus is the most prominent filovirus, causing severe haemorrhagic fever in humans with high mortality and fast spreading among African populations. The recent outbreak in 2013 in west Africa has resulted in the most severe epidemy of Ebola so far, with more than 11,000 lethal cases (as by September 2015; according to World Health Organization;http://apps.who.int/ebola/ebola-situation-reports).

All Ebola outbreaks recorded until 2004 in Gabon and the Republic of the Congo have been linked to handling of gorilla, chimpanzee or duiker carcasses, species that can carry the Ebola virus (Leroy et al. 2004; Pigott et al. 2014). It has thus became apparent that spillover from animals to humans occurs through hunting, butchering and consumption of bushmeat (Gonzalez et al. 2005; Li and Chen 2014; Chap. 12), followed by fast human-to-human transmission (World Health Organization 2014). An outbreak of Ebola in Congo in 2007 that resulted in 260 infected humans of whom 186 died has been traced to a potential direct transmission from a dead fruit bat that the first human victim bought from hunters to eat (Leroy et al. 2009). Antibodies against Ebola virus have since been detected in a total of 14 bat species, with seroprevalences of up to 44 % depending on species and location (Olival and Hayman 2014). Experimental infection of several bat species with Ebola led to high replication of the virus, but to no apparent signs of illness, suggesting that Ebola infections are subclinical in these species (Swanepoel et al. 1996). One *Eidolon helvum* has survived for at least 13 months after being tested seropositive for Ebola virus and Lagos bat virus, indicating long-term survival of an individual bat following exposure to these viruses (Hayman et al. 2010). The recent outbreak of Ebola in Guinea and neighbouring countries in 2013—countries that are at significant distance to the previous outbreaks in central Africa—has caused speculations about a possible transmission of the virus by migrating fruit bats (Bausch and Schwarz 2014; Vogel 2014). However, as the strain of the west African Ebola virus is a genetic outlier within the known Ebola viruses, it has been argued that the west African variant may have emerged from local wildlife populations rather than from migrating individuals (Gatherer 2014). Furthermore, although speculated (Saéz et al. 2015), it is yet not clear whether the spillover of Ebola virus in west Africa originated from bats.

Marburg virus is the only filovirus that has so far been directly isolated from bats (Towner et al. 2009; Amman et al. 2012; Pourrut et al. 2005). The first outbreak of the virus was caused by a spillover from laboratory monkeys to humans in Marburg, Germany, in 1967 (Jacob and Solcher 1968). In 2007, mine workers in a cave in Uganda were diagnosed with Marburg haemorrhagic fever that potentially resulted from a spillover of the virus from a colony of *Rousettus aegyptiacus*, where 5.1 % of tested individuals carried the virus (Towner et al. 2009). The high divergence of the genome sequence of Marburg in this population suggests a long-term association of the virus with the host, leading to the assumption that bats are the natural reservoir (Towner et al. 2009). However, given that no other bat species has yet tested positive for the virus (Towner et al. 2007), and seroprevalence being generally low in *R. aegyptiacus* (Pourrut et al. 2009), spillovers from bats to humans may be rare events.

The **Reston Ebolavirus** has first been detected in 1989 in crab-eating macaques (*Macaca fascicularis*) imported from the Philippines to be used for animal testing in laboratories in Reston, USA (Jahrling et al. 1990). During a second outbreak in 1990, animal handlers developed antibodies but did not get sick (Center for Disease Control and Prevention 1990). In 2008, Reston Ebolavirus was isolated from pigs in the Philippines (Marsh et al. 2011), and soon after, some

sampled *R. amplexicaudatus* had antibodies against the virus, while 16 other bat species tested negative against Reston Ebolavirus (Taniguchi et al. 2011). Screening for antibodies of the Ebola virus and Reston Ebolavirus in bats in Bangladesh has found seropositive *R. leschenaultii*, suggesting that these filoviruses or related strains are distributed at a much larger geographic range than previously assumed (Olival et al. 2013).

10.4 Main Conservation Issues Related to Bat Viruses

10.4.1 Direct Effect: Viruses Killing Bats

From all the viruses described above, only a few seem to affect bats. Although experimental infection with RABV leads to mortalities between 40 and 90 % depending on the bat species (Sétien et al. 1998; Jackson et al. 2008; Turmelle et al. 2010), there are no observed mass mortalities in natural populations (Pawan 1959). The only virus that may be largely lethal for bats is the **Lloviu virus**, which is closely related to Ebola and Marburg virus, but not yet of zoonotic relevance. It was detected during investigations of a massive die-off of *Miniopterus schreibersii* in a cave in Spain (Negredo et al. 2011). However, a causal connection between the detected virus and death of the bats has not yet been confirmed, and other bat species roosting in the same caves appeared to remain unaffected (Roué and Nemoz 2004).

The lack of reports of viruses that are detrimental for bat health should not imply that viruses in general are not of importance for the conservation of bat populations. Similar to white-nose syndrome causing mass mortalities in North American bats (Frick et al. 2010), newly emerging viruses may put local populations at threat. This may be especially the case if pathogens cross geographical borders and infect naïve bat populations. *Pseudogymnoascus destructans*—the causative fungus responsible for white-nose syndrome—likely originated from Europe, where it seemingly causes no bat fatalities, in contrast to North America (Puechmaille et al. 2010; Frick et al. 2010; Frick et al. 2015, Chap. 9).

10.4.2 Indirect Effects: Biased Public Perception

Generally, the public perception of bats as aesthetically less appealing mammals as well as folklores that often associate bats with negative stigma makes bat-related conservation efforts time-consuming and demanding (Fenton 1997; Allen 2004; Knight 2008). The recent outbreaks of viral zoonotic diseases with the identification of bats as putative natural hosts have further complicated bat conservation efforts (Li et al. 2005; Knight 2008). Following numerous and often lurid reports of fatal zoonotic diseases by the media, public perception of bats is mostly

skewed by fear and lack of information (Kingston 2016, Chap. 18). Therefore, it is important to highlight the context of bat-associated infections in order to provide more evidence-based information about the emergence and transmission of bat-related zoonotic diseases, which may lead to a more balanced reputation of bats. Depending on educational, cultural, legal and medial background of the targeted audience, specific aspects need to be taken into account.

In Europe and North America, rabies is, so far, the only viral disease that is associated with bats. The fact that lyssaviruses are occasionally found in temperate zone bats sometimes finds its way to the media, not always in favour of bats. Biased newspaper articles or press campaigns may result in the public misconception that bats are aggressive animals or that their mere presence can lead to human infections with these viruses. Although there are anecdotal reports of unprovoked attacks of bats on humans and dogs (Baer and Smith 1991), bats, as is the case of most mammals, usually only bite when handled or provoked. Furthermore, once bitten or scratched by a bat, immediate post-exposure vaccination can prevent a person from contracting rabies (see Sect. 10.5.2). In the case of the 37-year-old woman who died from a bat lyssavirus infection in Kenya, staff members of the health facility which the woman visited after being scratched by a bat were unaware of the possibility of rabies transmission (van Thiel et al. 2009). Likewise, two persons in Europe who worked regularly with bats and died from rabies after being bitten and scratched by bats received neither pre- nor post-exposure treatment (Roine et al. 1988; Nathwani et al. 2003). These two cases triggered a Europe-wide serological screening effort involving more than 11,000 bats, with seroprevalences varying depending on the species and location (Racey et al. 2012). EBLV-1 was most commonly detected in the serotine bat (*Eptesicus serotinus*), while EBLV-2 was very uncommon in all bat species. As a result, the public has been persuaded not to handle bats or to do so only with gloves and, in the case of bat workers, to receive pre- and/or post-exposure immunisation. Two fatal cases in which persons contracted rabies in Australia (Samaratunga et al. 1998; Hanna et al. 2000) triggered a similar campaign on this continent (Speare et al. 1997, but see Francis et al. 2014). Efficient education of medical professionals worldwide seems to be pivotal for implementing the correct treatment after scratches or bites from bats. In addition, vaccination should be mandatory for those who are frequently exposed to bats (Rupprecht and Gibbons 2004). Studies on animal models have shown that rabies vaccine also provides protection against other, although not all, lyssaviruses' variants (Brookes et al. 2005; Hanlon et al. 2005). However, there is no known case of a person developing bat-associated rabies despite having been vaccinated, neither pre- nor post-exposure. Thus, getting infected by some sort of bat-related virus is unlikely in Europe and North America and decreases virtually to zero if people who experienced bat bites and scratches are treated appropriately.

There is no case known for paramyxoviruses having spilled over to humans by direct contact with bats. An extensive serological survey among people frequently handling bats in Australia revealed no antibodies against Hendra virus (Arklay et al. 1996). The virus apparently needs horses as amplifier hosts, from where the virus can further be transmitted to persons in close contact with infected

individuals. Nevertheless, the outbreak of Hendra increased the unpopularity of flying foxes in Australia, making conservation of the four native species challenging (Thiriet 2011). Unlike Hendra, Nipah virus has likely been acquired by humans via consumption of contaminated date palm sap (Luby et al. 2006; Rahman et al. 2012), followed by person-to-person transmission (Gurley et al. 2007). Although diseases associated with Hendra virus and Nipah virus have high mortality rates, the risk of infection for humans seems to be low (Chong et al. 2003), and countermeasures may be taken in order to prevent future spillover events (see Sect. 10.5.2). MERS, just as Hendra virus, apparently needs livestock as an amplifier host. In contrast to dromedaries (Hemida et al. 2014), seroprevalence of MERS seems to be low in bats (Memish et al. 2013), making direct transmission from bats to humans unlikely. As long as details on MERS infections in dromedaries and how to mitigate them are missing, it is hard to give recommendations to people who might be at risk.

In contrast to MERS, the spillover of SARS into the human populations most likely happened via the wildlife market, either directly from a bat, or from other wildlife species. Likewise, the hunting, butchering and consumption of chimpanzees, gorillas and bats seem to have been sources of Ebola spillovers from wildlife to humans. The education of local communities needs to carefully balance information about the potential risk of acquiring infectious diseases by consuming bushmeat, without implying that bats need to be eradicated in order to prevent spillovers. The recent outbreak of Ebola resulting in several thousand human victims, and with bats frequently being reported as the likely source of origin, has undoubtly led to severe loss of reputation of bats on this continent, which makes the conservation of threatened populations and species even more challenging, not only in Africa, but also worldwide.

10.4.3 Indirect Effect—Culling

The direct persecution of bats often seems to be the most effective way to deal with bat-borne diseases to members of the public. Killing of bats has long been acceptable, even if they are protected (Chap. 14). Even though culling may be officially banned and thus not supported by authorities or governmental programs, large-scale killing of bats or the destruction of roost trees may still be commonly practiced in areas where zoonotic diseases are spreading.

In Australia, for example, flying foxes are frequently harassed and killed, both legally (under permits issued by state wildlife management agencies) and illegally. This happened most prominently during periods when Hendra virus emerged in Australian flying fox populations (Roberts et al. 2012). Half of the flying fox species native to Australia have declined about 30 % in population size during the last decade, and killing of bats usually does not lead to legal measures (Booth 2005). Furthermore, large-scale culling leads to a change of movement behaviour of bats, with new, susceptible individuals being recruited from nearby colonies

(Field 2009). Instead of reducing the viral prevalence, this may therefore lead to the exact opposite (see below).

In the attempt to reduce rabies incidences, vampire bats are regularly culled in many parts of Latin America (Streicker et al. 2012). In Brazil, for example, governmental programs are in action that involve targeted campaigns against vampire bats. During these measures, vampire bats are captured and poisoned or coated with anticoagulant and released, so that allogrooming kills their conspecifics (Medellin 2003). Furthermore, bat roosts are destroyed using fire and explosives (Mayen 2003), which also leads to dramatic declines of non-target bats (Furey and Racey 2015, Chap. 15). Besides the questionable methods involved, instead of reducing viral abundance in the population, culling of wildlife can lead to an increase in viral spreading. New hosts are recruited and the dispersal probability of infected individuals increases, which results in transmission of the disease to naïve hosts (Donnelly et al. 2005; Choisy and Rohani 2006; Streicker et al. 2012). This was the case for vampire bats in Peru, where culling failed to reduce seroprevalence of rabies in bat populations, but rather had the opposite effect (Streicker et al. 2012). Therefore, persecution of bats as potential carriers of zoonotic diseases has been denounced as useless and even counterproductive by both conservationists and experts on disease transmission (Hutson and Mickleburgh 2001; Knight 2008).

10.4.4 Indirect Effect—Killing of Bats for Virus Surveys

In the scope of recently emerging zoonotic diseases, the search for new bat-borne viruses has become a well-funded field in the scientific community. While research is important to advance our understanding about the emergence of diseases and to possibly prevent further spillover events, the methods involved in these surveys are sometimes questionable from the perspective of bat conservation (Racey 2015). Some of the investigated bat species are listed as near threatened or vulnerable by the International Union for the Conservation of Nature (IUCN), with decreasing population sizes even in many species of least concern. While most surveillance studies that involve species of conservation concern use non-lethal methods such as antibody screening in blood (Hayman et al. 2008; Young et al. 1996; Lumlertdacha et al. 2005; Wacharapluesadee et al. 2005; Reynes et al. 2005), others have involved the killing of a considerable number of bats of various conservation status (e.g. in Yob et al. 2001; Kuzmin et al. 2008b, 2010, 2011b; Dzikwi et al. 2010 and Sasaki et al. 2012). In order to limit such detrimental surveys, the Food and Agriculture Organization of the United Nations (2011) has published a guideline for investigating the role of bats in emerging zoonotic diseases, including non-invasive protocols, which not only reduce the impact on bat populations, but also minimise the transmission risk of viral diseases. Such protocols have now been widely adopted, as for example by Ecohealth Alliance and other international research groups and networks.

10.5 Counter Measures in Favour of Bat Conservation

10.5.1 Preventing the Emergence of New Viral Diseases

In general, preventing the emergence of infectious diseases in wildlife populations is extremely challenging and usually underfunded, with only few practical suggestions being discussed (Daszak et al. 2000). For example, it is important that translocations of animals across geographical borders need to follow strict guidelines in order to prevent the introduction of exotic pathogens in novel areas (e.g. Woodroofe 1999). Furthermore, an integration of knowledge about disease dynamics, as well as ecological and immunological aspects of the host, may contribute to a better understanding of emerging infectious diseases in wildlife species such as bats (Daszak et al. 2000).

10.5.2 Educational Efforts

As many bat-borne viral diseases have high lethality rates for humans, preventing spillover events are of central importance. In particular, spillover by direct contact to bats, such as via bites or bat consumption, may bear severe risks to humans that could be minimised by educational programs (Kingston 2016, Chap. 18). Reducing the risk of outbreaks of zoonotic viruses may also lead to more positive attitudes towards bats, which may further be increased by highlighting their ecological importance as pollinators, seed dispersers and pest control for agriculture (Ghanem and Voigt 2012). Moreover, conservation measures that promote the preservation of bat habitats serve a dual role as they can decrease the contact zone between bats and humans, thus reducing the risk of spillover.

As aforementioned vaccination against rabies and other lyssaviruses should be mandatory for persons working with bats and recommended for other people at risk. A significant problem is that both pre- and post-exposure treatments are expensive and thus may not be readily available in developing countries, such as in Central and South America. Here, building houses in a bat-proof manner in order to avoid vampire bites during sleep and decreasing the risk of direct contact with other bats has so far been the best solution (Greenhall 1964; Voigt et al. 2016, Chap. 14).

A different issue is the transmission of Nipah viruses via consuming raw date palm sap contaminated by urine, faeces or saliva of bats (Luby et al. 2006; Rahman et al. 2012). Here, cooking the sap at temperatures above the level that viruses tolerate is an effective measure to prevent spillover (Hughes et al. 2009). Additionally, preventing bats from accessing date palms and thus contaminating the sap has been proved to be both efficient and relatively cheap (Nahar et al. 2010, 2013). The traditional “bamboo skirt” method for example uses inexpensive, recyclable bamboo to cover the part of the date palm where the sap is collected, preventing bats and

Fig. 10.1 Intact trees with colonies of *Eidolon helvum* (*left*) in Yaoundé, Cameroon, as compared to former roosting trees that have been cut (*right*) after bats were suspected to be the source of the recent Ebola outbreak in western Africa (photograph credits: Simon Ghanem)

other vertebrates from getting access. Furthermore, in contrast to bird nets, this measure is non-lethal to the bats and therefore of high conservation value to local populations. However, such protective measures are reported to be rarely used in Bangladesh (Nahar et al. 2010, 2013). This could potentially be changed by encouraging local farmers to use this method, emphasising its inexpensiveness and efficiency while highlighting the reduced risk of acquiring Nipah virus disease.

One of the key issues both for conservation and public health is the direct transmission of SARS and Ebola via wildlife markets. In South-East Asia, flying foxes are hunted regularly for the purpose of food (Mickleburgh et al. 2002; Mildenstein et al. 2016, Chap. 12), sometimes even authorised by the local Wildlife Department such as in Malaysia (Breed et al. 2006). Likewise, fruit bats are consumed regularly throughout Africa (Mickleburgh et al. 2009; Mildenstein et al. 2016, Chap. 12). Since bats are suggested as potential reservoir for the recent outbreak of Ebola, Guinea banned bats for sale from markets (Gatherer 2014). Educational efforts to reduce the threat both to public health by zoonotic diseases and to the conservation of local bat populations are challenging, as they are usually impeded by the lack of understanding of entrenched cultural behaviours and social components (Pooley et al. 2015; Kingston 2016, Chap. 18). In Ghana, for example, where the consumption of bats is part of the local culture and traditions, a survey revealed that knowledge about the ecological and economical value of bats would not make people refrain from killing and eating bats (Kamins et al.

2014). Usually, the direct economic benefit from selling hunted bats is more valuable to an individual person than the indirect, not always obvious economic value of bats, for example, for agriculture. However, about half of the hunters stated they would stop hunting bats if they could make them sick (Kamins et al. 2014). This highlights the potential effectiveness of public education, but careful consideration is needed to avoid demonising bats in the process (Pooley et al. 2015). The recent Ebola epidemic in western Africa for example has led to an increase in the persecution of bats, with roosts being destroyed and colonies being killed by communities (Fig. 10.1). Although preventing bats from being consumed may have higher priorities due to public health reasons, the culling of whole colonies as a likely result may be much more of a threat for the conservation of bats than the bushmeat trade (Pooley et al. 2015).

10.5.3 Environmental Conservation

Combining knowledge about the ecology of the host species as well as the disease dynamics of the virus may be crucial for establishing efficient disease prevention programs (e.g. Plowright et al. 2015). Here, it needs to be noted that the emergence of zoonotic diseases from bats also seems to be a consequence of anthropogenic alteration of natural environments (e.g. Daszak et al. 2001). For example, in Central and South America, the conversion of forested habitats into pastures shifted the dominant food source for vampire bats from native vertebrates to livestock. This has increased rabies transmission from vampire bats to livestock and domestic animals in many parts of Latin America (Schneider et al. 2009). Where bat habitats have been converted largely into agricultural farmland, the remaining bat populations are forced to concentrate in patches that provide them with resources they need. Flying foxes, for example, are highly sensitive to landscape modifications, as they require large forested areas for foraging. Where natural habitats are scarce, flying foxes may use fruiting or flowering trees in agricultural, suburban and urban areas, which increases the contact zone and spillover risk between bats and livestock or humans (Daszak et al. 2006; Plowright et al. 2015). Indeed, contact between bats and naïve hosts as a consequence of human landscape modification and encroachment likely sparked the transmission of Hendra viruses to horses (Epstein et al. 2006) and Nipah virus to pigs (Chua et al. 1999; Field et al. 2001).

10.5.4 Conservation of Bat Populations and Population Dynamics

Removing individuals or colonies from regional populations, either by unsustainable hunting or culling, can cause an increase in relative local resource availability, creating regional gradients along which bats from other populations may move,

which may lead to an increase of virus movement (Field 2009). In Australia, for example, roosts that became empty after culling, disturbing or relocating colonies of flying foxes are usually reoccupied by immigrating individuals (Roberts et al. 2012).

Anthropogenic transformation of bat habitats in Australia has also been shown to lead to decreased migration in *Pteropus* bats, which can itself lead to a decline in population immunity (Plowright et al. 2011). This could give rise to more viral shedding after local viral reintroduction, a mechanism that may be facilitated by urban habituation of fruit bat and the resulting increased contact with human and domestic animal populations (Epstein et al. 2006; Plowright et al. 2011). In Australia, all recently emerged bat-associated viruses—Hendra, Menangle and Australian bat lyssavirus—are hypothesised to be associated with habitat loss due to deforestation and agricultural intensification (Jones et al. 2013). Therefore, protection of remaining natural habitats of bats along with farm management aiming at decreasing the contact zone between bats and livestock as well as education plans increasing awareness of environmental issues and safety may play a crucial role in the avoidance of future spillovers of bat-borne diseases to livestock and human populations, and promote further protection of local bat populations.

10.6 Conclusion

Bats harbour viruses that may become zoonotic. Circumstances facilitating spillover include direct contact with bats (bites, scratches, consumption of bats), contact with material contaminated by bat saliva, faeces or urine and amplification via intermediate hosts such as domestic animals or other wildlife species. Conservational actions are not only important to prevent spillovers, but also because emerging zoonotic viruses often lead to persecution of bats. In order to reduce the transmission risk of viruses from bats to human and livestock and to protect bat species at threat, educational efforts are needed. However, entrenched cultural and social components often act as barriers to efficient changes on how people think about and respond to bats. Whenever possible, educational efforts should be done in an informative, non-lurid way, presenting the facts rather than provoking additional fears to the already bad reputation of bats. Wherever possible, solutions should be found to enable the existence of bats in anthropogenic landscape, including the development of more affordable and readily available vaccinations (e.g. against rabies), and the reduction of potential contact between bats and humans and livestock. This however also includes that the natural habitats of bats need to be better protected to provide bat populations with sufficient space and to prevent range expansion into urban and suburban areas, where contact with humans and livestock may increase the risk of spillover events. Bat-borne viruses should be considered during bat conservation efforts, and it should be equally noticed that appropriate conservation measures may even reduce the risk of viral spillover from bat populations into human populations.

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Part III
Human-Bat Conflicts