Cell PRESS

White-nose syndrome: is this emerging disease a threat to European bats?

Sébastien J. Puechmaille[1], Winifred F. Frick[2,3], Thomas H. Kunz[3], Paul A. Racey[4], Christian C. Voigt[5], Gudrun Wibbelt[5] and Emma C. Teeling[1]

[1] UCD School of Biology and Environmental Science, University College Dublin, Belfield, Dublin 4, Ireland
[2] Department of Environmental Studies, University of California, Santa Cruz, CA 95064, USA
[3] Center for Ecology and Conservation Biology, Boston University, Boston, MA 02215, USA
[4] Centre for Ecology and Conservation, University of Exeter in Cornwall, Exeter, Devon, EX4 4SB, United Kingdom
[5] Leibniz Institute for Zoo and Wildlife Research, D-10252, Berlin, Germany

White-nose syndrome (WNS) is a newly emergent disease that potentially threatens all temperate bat species. A recently identified fungus, *Geomyces destructans*, is the most likely causative agent of this disease. Until 2009, WNS and *G. destructans* were exclusively known from North America, but recent studies have confirmed this fungus is also present in Europe. We assembled an international WNS consortium of 67 scientists from 29 countries and identified the most important research and conservation priorities to assess the risk of WNS to European bats. Here, we review what is known about WNS and *G. destructans* and detail the conservation and research recommendations aimed at understanding and containing this emerging infectious disease.

Emerging infectious diseases and conservation problems

Emerging infectious diseases are currently a severe conservation threat for a variety of taxa, such as bees at threat from varroasis, crayfish from plague, salmonids from whirling disease, amphibians from chytridiomycosis, birds from Newcastle disease and West Nile virus, and primates from Ebola and Marburg viruses, to name a few examples [1]. Emergent diseases in wildlife pose particularly difficult challenges to conservation because of their typically rapid and unexpected onset, high rates of mortality and potentially complex interactions. The origins, dispersal mechanisms and modes of death are often not well understood during an initial epizootic, further challenging conservation and management efforts. Emergent diseases can spread globally and mortalities mount while researchers struggle to understand the disease and develop effective management strategies to minimise spread [2,3]. For example, the chytrid fungus *Batrochochytrium dendrobatidis* which causes chytridiomycosis, a disease that potentially threatens over 50% of all living amphibians with extinction [4], was first identified in 1999 after more than a decade of population declines and it was another decade later before the actual mode of amphibian death due to *B. dendrobatidis* infection was discovered [5,6]. Despite years of study, many aspects of this disease continue to elude researchers [4,6].

Corresponding authors: Puechmaille, S.J. (s.puechmaille@gmail.com); Teeling, E.C. (emma.teeling@ucd.ie).

Bats account for approximately one-fifth of all living mammalian diversity [7]. Among these, several species of hibernating bats are being threatened by white-nose syndrome (WNS), an emerging infectious disease that has caused unprecedented mortality of hibernating bats in North America since 2007 [8–10] (Box 1). The newly discovered fungus *Geomyces destructans* (Gd) [10,11] is considered to be the responsible agent [12] (Box 2). Recent investigations have shown that a fungus growing on bats throughout Europe is genetically identical to the North American Gd at two molecular markers [13–16]. Although to date, there have been no reports of mass mortality that could be related to Gd infection in Europe [13–16], it is currently unknown whether this fungus has more subtle effects on European bats. The presence of the same fungus on bats in Europe without obvious mass mortality raises questions about the spatial variability in disease and species-specific susceptibility among bats; the risk of WNS to bats in Europe; the potential for acquired immunity in European bats; and questions whether Gd is the primary or sole source of mortality in bats infected with this fungus.

Here, we review what is known about WNS and its putative pathogen Gd and detail the evolutionary and ecological scenarios that could have led to the current global distribution of Gd and WNS. We summarise and elaborate on the conservation and research recommendations developed by the WNS consortium (see the see the supplementary material online for list of WNS consortium members). This action plan represents a global cooperation among ecologists and conservation biologists that will enable us to understand a deadly and potentially pandemic disease in one of the largest and most ecologically important mammalian taxon.

WNS – a North American disease

WNS is a disease of hibernating bats in North America and is associated with a cold-loving fungus, Gd [11] (Box 2). The fungus grows on exposed skin tissues (including snout, ears and/or wing membranes) of afflicted bats during hibernation [17]. Gd, first observed in New York state in 2006 [18], has rapidly spread across eastern North America over the past five years into New Brunswick, Nova Scotia, Ontario and Québec, Canada to the north, throughout the mid-Atlantic region into North Carolina to the south, and as far west as Oklahoma (Figure 1). To date,

 0169-5347/$ – see front matter doi:10.1016/j.tree.2011.06.013 Trends in Ecology and Evolution, November 2011, Vol. 26, No. 11

Box 1. Potential threat to bats

In 2009, experts estimated that mass mortality associated with WNS had killed over 1 million bats in the northeastern USA in just the first two years of the epizootic [8]. Six species of hibernating bats (*Myotis sodalis*, *Myotis lucifugus*, *Myotis septentrionalis*, *Myotis leibii*, *Perimyotis subflavus* and *Eptesicus fuscus*) are currently affected by WNS. More species are at risk as the disease spreads into the ranges of other hibernating bats [3]. In 2010, the presence of Gd was genetically confirmed in three more species (*Myotis grisescens*, *Myotis velifer* and *Myotis austroriparius*) one of which (*M. grisescens*) is listed as a federally endangered species by the US government.

Myotis lucifugus, one of the most abundant, widely distributed, and best-studied species of bat in North America, has suffered exceptionally high mortality at hibernacula infected with WNS [8] (Figure I, Table I). Annual decreases in numbers of hibernating *M. lucifugus* are in the range 30–100% (mean 70%), whereas stable to increasing population trends were seen over the past 30 years prior to WNS [8,50]. Such high mortality rates are reflected during the summer period by a 78% decline in *M. lucifugus* activity in a region affected by WNS [25]. Using population viability analysis, Frick *et al.* [8] estimated the probability of regional extinction of *M. lucifugus* from disease-associated mortality and showed that even if disease-mortality ameliorates with time (as observed at sites with multiple years of WNS mortality), the regional population of the species in the northeastern US could face extinction in 16–20 years [8].

All six bat species that have suffered mortality from WNS in North America are cave-roosting, hibernating members of the family Vespertilionidae. Because there are no other families of hibernating bats located in these regions, it remains unclear whether non-vespertilionid species are also susceptible. The Vespertilionidae account for 36% of all living bat diversity (~407 known species; [7]) and are the most species-rich group in temperate northern latitudes. Within Europe, the Vespertilionidae account for the majority of bat species (n = 36/45) and thus most European bats are potentially threatened by this newly emerging infectious disease. If WNS is an emergent disease that is not limited to vespertilionid species but affects all hibernating bats, then all European bat species may be faced with similar mortality levels.

Table I. *Myotis lucifugus* hibernacula survey counts from the most recent pre- and post-WNS surveys (included hibernacula represent the overall ten largest declines of *M. lucifugus*) [8].

Pre-WNS count (Year)	Post-WNS count (Year)	Overall decline
2276 (2008)	1 (2010)	100.0%
1511 (2005)	1 (2008)	99.9%
9432 (2003)	24 (2010)	99.7%
1722 (2009)	5 (2010)	99.7%
1604 (2006)	8 (2010)	99.5%
720 (2004)	6 (2010)	99.2%
183 542 (2000)	2049 (2010)	98.9%
1102 (2004)	22 (2009)	98.0%
953 (1999)	22 (2010)	97.7%
1213 (2005)	37 (2010)	96.9%

Figure I. Bat carcasses piled on the cave floor of a winter hibernacula in Vermont, USA, demonstrating mass mortality at hibernation sites from WNS (Photo credit: Alan C. Hicks).

at least nine species of North American vespertilionid bats have been infected by Gd [2], but only six species have manifested the symptoms associated with WNS.

WNS is characterised by a cutaneous fungal infection caused by Gd in which the fungal hyphae invade hair follicles and associated sebaceous and sweat glands [17]. Disease symptoms include premature depletion of fat reserves [10,19], frequent arousals during winter [20–22], premature arousal from hibernation in spring [3,9,18] and ulcerated, necrotic and scarred wing membranes [23]. Mortality associated with WNS has caused severe population declines, in the range 30–100%, at infected hibernacula, resulting in regional population collapses [8,24,25]. Recent analysis of mortality rates in *Myotis lucifugus*, one of the species most affected by WNS, predicts regional extinction in 16–20 years [8].

Gd and European bats

Despite the fact that Gd infection has been reported in nine bat species across Europe, premature loss of fat reserves, unusual winter activity or mortality that are characteristic of WNS in North America have not been documented [13–16]. Bats from Europe can show high prevalence of Gd towards the end of hibernation in March–April and some torpid animals have also been observed with fungal infections outside of the hibernation period in cold underground sites [16]. However, the pathological effects of Gd growth on European bats have not yet been studied.

Scenarios to explain the global distribution of Gd and WNS:

Differences in the distribution of Gd and manifestations of WNS in North America and Europe could be explained by three competing but not mutually exclusive hypotheses:

The fungus has been present in Europe for a long time and has only recently invaded North America

Howes Cave, where WNS was first observed in 2006, is the largest commercial cave in the northeastern US with about

Box 2. Gd, morphology and phylogenetics

Geomyces destructans is a recently described species of psychrophilic (cold-adapted) fungus that has an optimum growth on artificial media at 10–15 °C [11,13,51]. Typically it is found growing on bats and appears as a profuse yet delicate hyphal growth on their snouts and/or wings [10] (Figure Ia and b). A recent study has also isolated this species of fungus from the walls of a hibernaculum [16]. The unique characteristics of Gd are the curved conidium shape along with its cold growth range [11]. Typically spores are produced from the tip of groups of short branches, sometimes in loose verticils, at the apices of conidiophores. Spores can also be developed from side branches and directly from the conidiophore surface. They are produced either in small clusters, or in short chains of 2–3 spores or singly. They are individually hyaline, irregularly curved, broadly crescentic in shape, typically 6–8 μm in length and 3–4 μm at their widest diameter, narrowing to each end, one of which is broadly truncate, often with an annular frill (Figure Ic) [14]. In side view, many spores appeared obovoid; intercalary spores can be barrel-shaped. Spore walls are relatively thick and an outer surface granulation can be evident on some [11,14]. Gd is closely related to the Leotiomycetes [11,14]. Supertree whole genome phylogenetic analyses have placed the Leotiomycetes as sister taxa to the Sordariomycetes in the Pezizmycotina as an ascomycotan fungus [52]. Short single gene analyses SSU, ITS and 28S all place *G. destructans* as sister taxa to *Geomyces panorum*, *Geomyces asperulatus*, *Pseudogymnoascus roseus* and *Pseudogymnoascus verrucosus* [11,14].

Figure I. Typical Gd growth on the snout, ears and wings of a **(a)** *Myotis myotis* (Photo credit: Christian Jungmann, 2010, Germany) and a **(b)** *Myotis lucifugus* (Photo credit: Ryan von Linden, 2010, USA). Scanning electron micrograph showing **(c)** a hair of a *M. myotis* heavily colonised with Gd, and **(d)** the distinctive conidia of Gd.

200 000 visitors each year. High levels of human traffic at this site suggest that the presence of Gd could have resulted from an anthropogenic introduction. Within Europe, there are reports of a white fungal growth on bat snouts and/or ears from the late 1970 s in Estonia [26], the early 1980 s in Germany [27] and since the 1990 s in France (Christophe Rideau, personal communication), although there is no proof that these fungi were Gd. In North America, prior to 2006, there are no published reports of cutaneous fungal growth on hibernating bats and photographic records, dating back

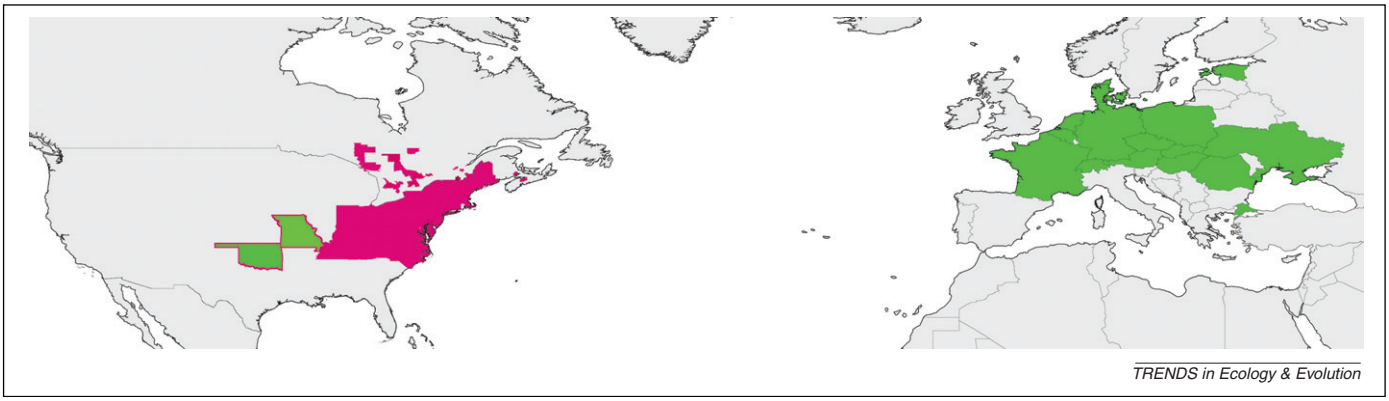

Figure 1. Distribution of Gd colonising bats in North America and Europe, with different coloured indicators identifying Gd colonisation (green) versus WNS (pink), based on data from [13–16] and maps created by C. Butchkoski, Pennsylvania Game Commission (http://www.fws.gov/whitenosesyndrome/). Green with pink border depicts WNS suspected areas.

30 years in the northeastern US, show no evidence of visible fungal infections (A. Hicks, personal communication). The discovery that two short, highly conserved genetic sequences (*ITS* and *SSU*) from Gd in Europe and the North America are 100% identical [14], suggests a relatively recent historical exchange (anytime in the past few million years) between the fungal populations from the two continents. These genetic data cannot further resolve the timing of this event nor its direction due to the slow rate of evolution of the markers investigated.

The lack of historical evidence of even low levels of prevalence of Gd in North America prior to 2006 contrasts with data from Europe that reports white fungal cutaneous infections in several countries [13–16]. These observations, combined with the absence of fungal-associated mass mortalities in winter, suggest that bats in Europe might have been historically exposed to Gd and might have developed immunity or evolved genetic or behavioural resistance to this putative pathogen. This could explain why bats in Europe are not currently experiencing mass mortalities with Gd colonisation.

Gd is a newly emergent fungal pathogen that recently arose in North America.

WNS is acting and spreading as a newly emergent disease in North America. The first reported case of bats with suspected Gd dates from a photograph taken on 16 February 2006 at Howes Cave, Albany, NY [10]. During winter 2006/2007, mortalities and infected bats were reported from hibernacula in upstate New York. Surveys during the winter 2007/2008 showed that all sites visited within a 130-km radius around this epicentre were contaminated but no site further than 200 km was affected (A. Hicks, personal communication). Gd has continued to spread in a wave-like pattern, supporting the hypothesis that the pathogen originated in New York State and spread quickly, a pattern typical of a newly emergent pathogen [28].

The origin of this pathogen in the US is unknown. One suggestion is that it is a new, virulent strain of a previously benign globally distributed fungus. If this were the case, then Gd should be widely distributed in North America and therefore should be found outside current WNS-affected areas. To date, searches for Gd outside these areas in North America have not been successful [29], supporting the hypothesis that Gd is an introduced species from Europe. However, detecting Gd in soil samples is challenging and thus, the global distribution and phylogeography of Gd needs to be better understood to conclusively distinguish between these hypotheses. Both scenarios of an introduced pathogen from Europe or emergent mutation in North America have conservation implications. Even if the North American Gd were of European origin, it might have mutated and/or recombined while in North America and could now be a more virulent strain, thus posing a threat to hibernating bats globally.

Gd is not the primary mortality agent but acts as an opportunistic pathogen in immunocompromised individuals, and some other agent is killing North American hibernating bats

Gd is present in European bats but has not yet been associated with mass mortality [16]. This suggests that Gd alone might not be responsible for WNS in North America. All investigations to date point to Gd as the causative agent of WNS in the US [12], but unequivocal proof remains elusive as in many wildlife diseases [3,10,30]. Recent studies indicated no evidence of major organ failure [10,19] or consistent toxic elements (e.g. PCBs, DDT, arsenic and lead) in bats affected by WNS [19,31]. To date, no other known bacteriological or virological pathogens have been found in bats with WNS [10], although many bacteria and viruses associated with bats are probably still undescribed [32]. From a conservation perspective, it is of utmost importance to determine the causal mechanisms of death from WNS. If another pathogenic agent is the primary culprit then research efforts that focus only on Gd will neither advance our understanding of this disease nor help in identifying effective management strategies.

Research priorities for bat conservation given the threat of WNS in Europe

The presence and identification of Gd in European bats without mass mortality presents an opportunity to help understand the conservation crisis that is currently occurring in North America. Also, it provides an opportunity for European researchers and wildlife managers to potentially prevent or mitigate a threatening epidemic. Research on Gd in Europe has mainly focused on describing the

morphological and molecular characteristics of the fungus compared with this species in North America, and documenting its geographic range and prevalence in European bats [13–16]. We describe below five major research priorities identified by the WNS consortium to advance the understanding of this disease and assess the threat of WNS to European bats.

What is the global distribution of Gd and the phylogenetic relationship between populations from North America and Europe?

Phylogeographic and population genetic studies of Gd will be critical for determining its evolutionary history and distinguishing among competing hypotheses about its geographic origin in North America (introduced versus native). Samples from throughout the range need to be collected and screened at appropriate molecular markers, which then must be identified [33]. Fungal isolates have already been sampled from bats with visible infections in the past three winters (2007/2008–2009/2010) throughout Europe, providing valuable baseline information that suggests the fungus is widely distributed in Europe (Figure 1) and possibly extends into Russia and Western and Central Asia [16]. Ongoing studies in North America have also demonstrated the presence of Gd from Nova Scotia to Oklahoma (Figure 1, see http://www.fws.gov/WhiteNoseSyndrome/maps.html for a regularly updated map). Development of a highly specific molecular assay will greatly enhance efforts to further assess the presence of Gd across temperate latitudes in North America and Eurasia.

Coordination among researchers is critical so that samples collected from bats are sent to laboratories in Europe and North America that have the capacity to grow and genetically identify Gd and to complete comparative analyses. Developing the capacity to maintain living collections of these strains will permit assessment of the temporal evolution of the putative pathogen, which will be particularly useful if changes in virulence appear in the future. Museum collections of bats and soil samples from hibernacula (if they exist) should be analysed for the presence of Gd [16] to provide historic genetic samples for temporal and spatial comparisons.

Are the European and the North American Gd genetically identical at the genomic level?

Comparing the genomes of the European and North American isolates of Gd is necessary to assess if genomic differences might explain apparent differences in virulence of Gd in North America and Europe. The entire fungal genomes from North American and European Gd isolates are being sequenced and plans to further sequence a total of 15–20 isolates from both continents are in progress [33] (J.T. Foster, personal communication). This research is expected to facilitate the detection of lateral gene transfer or a species recombination event that could have caused an increase in virulence in North America.

Is Gd the primary mortality agent?

Identifying mechanisms of mortality by which Gd causes death in bats is essential to understand WNS. Cryan *et al.* [12] suggested that an infection with Gd might cause extensive subcutaneous wing damage resulting in physiological imbalance and dehydration of infected bats, which might trigger energetically costly arousals for drinking [34] that prematurely deplete fat reserves and ultimately causes death by starvation [12]. In support of this hypothesis, WNS-affected bats sent to the laboratory for histopathological studies were on average lighter than unaffected conspecifics [10,19], and although direct comparative studies need to be conducted, bats affected with Gd arouse more frequently than unaffected bats [20–22].

Bats might also arouse more frequently to mount an effective immune response against the fungus, a response that may not be possible during prolonged bouts of torpor associated with hibernation [35,36]. Currently, we do not know whether the level of immune suppression observed in Gd-afflicted bats is a prerequisite or a cause of the disease [17]. Lack of a sufficient immune response against fungal invasion during hibernation by bats in North America could be caused by immunosuppression due to high levels of toxins [37,38] or induced by a primary infection from another infectious agent. This type of dual pathogen effect has been recently discovered to cause Colony Collapse Disorder, a disease that affects bees in North America, Europe and Asia [39,40].

To address whether another pathogen is causing immunosuppression, it will be necessary to conduct comparative genomic studies of the pathogens present in the North American bats that show signs of the disease and die versus the pathogens present in European bats that have fungal growth but no evident loss of body condition, wing damage or immune responses. As bats have been shown to harbour high levels of chemical toxins [41], further studies are needed to assess the impact of this toxicity on their immune system.

What is the pathogenicity of Gd to bats in Europe?

In North America, the pathologic effects of Gd in bats have been described from naturally afflicted animals [17]. Comparisons of pathologic effects of Gd infections in North American and European species have not yet been possible due to the absence of pathological investigations in European species. Both laboratory and field investigations are urgently needed to characterise the pathogenicity of European Gd.

Large-scale population monitoring programmes involving all bat species such as the UK's National Bat Monitoring Programme [42] should be conducted for early identification of rapid population declines due to Gd infections or other diseases [43]. The discovery of WNS was only possible through the ongoing monitoring of bat populations, highlighting the importance of such programmes in wildlife conservation. Large amounts of field data and repeated annual inspections in selected hibernacula are needed to follow temporal and spatial trends in the growth of Gd on bats and its effect on local bat populations. Climatic conditions (e.g. temperature and humidity) in hibernacula should also be monitored to gain information on the autecology of Gd to help understand factors associated with its spatio-temporal changes in prevelance and abundance [16].

Are European bats immune and/or resistant?

If WNS in North America is caused by a recently introduced pathogen from Europe and European bats have survived a previous European epidemic or co-evolved with the causal organism, they might have developed natural immunity to Gd [33]. Research aimed at comparing immune function and response to Gd in hibernating bats in Europe and North America could help determine if European bats have natural resistance against Gd infection. Comparative genomic studies of both the innate and cell-mediated immune system (e.g. major histocompatibility complex genes) should be conducted along with basic investigations into mechanism of general immunity in European and North American bat species. Comparison of immune gene expression in different tissues of European and North American bats infected with Gd, in parallel with comparisons of gene expression levels between the European and North American Gd isolates could provide valuable information on the nature of the interaction between the fungus and the host as demonstrated for other pathogenic fungi [44–46]. These data could help to develop an effective vaccine [3,47,48].

Perhaps the seemingly benign infections of European bats with Gd stems from differences in hibernating behaviour between European and North American bat species. For example, differences in sociality and microclimatic preferences as well as natural arousal cycles might play a role in disease progression and mortality [33]. To test for the influence of behavioural and ecological differences on species susceptibility and mortality, bats in hibernacula should be monitored using infrared cameras and data loggers to assess their position in caves, activity patterns, body temperature, cave temperatures and humidity. Physiological differences between species in terms of evaporative water loss should also be investigated [12]. These data can be compared among species within North America and between Europe and North America, to determine if resistance is conferred by behavioural, physiological and/or ecological differences. Studying the mechanisms of immunity and resistance (innate or behavioural) of bats will significantly advance our understanding of how individuals may survive infections of Gd and WNS [33].

What should people do now?

At the same time as research is being undertaken to answer the questions outlined above, it is important to establish an action plan for human cave-visitors (speleologists, bat researchers, geologists and tourists). Movement of Gd by human activity poses a significant threat to bats. Increased awareness among the caving community of the threat that WNS poses to bats is of utmost importance for reducing the risk of human dispersion and transmission of Gd. Until researchers have firmly established why WNS has not yet occurred in Europe, the precautionary principle dictates that strong measures should be taken to reduce the risk of further transcontinental movements of Gd [49], especially from North American to Europe or other temperate regions. Indeed, the greatest threat to bats in Europe is if the North American Gd is different from that currently found in Europe and a caver, tourist or researcher from North America visits and introduces spores to Europe through contaminated clothing, boots or field equipment. This could mean dissuading people from North America from visiting caves in Europe, especially those who have recently visited caves in North America or have been in close contact with bats. An onus should also be placed on owners of caves and operators of cave tourism facilities in Europe to monitor and limit visitors from North America and/or take appropriate actions to limit its spread.

Conclusions

Only by simultaneously investigating the bats and the fungus at many integrative levels will we be able to understand the emergent disease of WNS and hopefully find ways of minimising its spread and potentially mitigating its harmful effects. To do so, researchers on both sides of the Atlantic need to cooperate and share their findings. This paper represents one of the first examples of a globally integrative response to an environmental catastrophe and describes the steps needed to be taken to help address a potential global disaster.

Acknowledgements

We acknowledge the collective recommendations of the members of the White-Nose Syndrome consortium, all of whom are listed in the online supplementary material. We thank P. Craze, B. Fenton and two anonymous reviewers for their comments; C. Butchkoski for providing GIS information on the distribution of Gd in North America; K. Langwig for help in collating data for Table I in Box 1; and A. Hicks, C. Jungmann and R. von Linden for providing pictures. We received support from a Science Foundation Ireland PIYRA grant to ECT and an IRCSET-Marie Curie International Mobility Fellowship in Science, Engineering and Technology to SJP.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.tree.2011.06.013.

References

1 Daszak, P. *et al.* (2000) Emerging infectious diseases of wildlife - threats to biodiversity and human health. *Science* 287, 443–449
2 Hallam, T.G. and McCracken, G.F. (2011) Management of the panzootic white-nose syndrome through culling of bats. *Conserv. Biol.* 25, 189–194
3 Foley, J. *et al.* (2011) Investigating and managing the rapid emergence of white-nose syndrome, a novel, fatal, infectious disease of hibernating bats. *Conserv. Biol.* 25, 223–231
4 Fisher, M.C. *et al.* (2009) Global emergence of *Batrachochytrium dendrobatidis* and amphibian chytridiomycosis in space, time, and host. *Annu. Rev. Microbiol.* 63, 291–310
5 Voyles, J. *et al.* (2009) Pathogenesis of chytridiomycosis, a cause of catastrophic amphibian declines. *Science* 326, 582–585
6 Kilpatrick, A.M. *et al.* (2010) The ecology and impact of chytridiomycosis: an emerging disease of amphibians. *Trends Ecol. Evol.* 25, 109–118
7 Simmons, N.B. (2005) Order Chiroptera, In *Mammal Species of the World: A Taxonomic and Geographic Reference* (3rd edn) (Wilson, D.E. and Reeder, D.M., eds), pp. 312–529, Johns Hopkins University Press
8 Frick, W.F. *et al.* (2010) An emerging disease causes regional population collapse of a common North American bat species. *Science* 328, 679–682
9 Veilleux, J.P. (2008) Current status of white-nose syndrome in the Northeastern United States. *Bat Res. News* 49, 15–17
10 Blehert, D.S. *et al.* (2009) Bat white-nose syndrome: an emerging fungal pathogen? *Science* 323, 227
11 Gargas, A. *et al.* (2009) *Geomyces destructans* sp. nov. associated with bat white-nose syndrome. *Mycotaxon* 108, 147–154
12 Cryan, P. *et al.* (2010) Wing pathology of white-nose syndrome in bats suggests life-threatening disruption of physiology. *BMC Biol.* 8, 135
13 Martínková, N. *et al.* (2010) Increasing incidence of *Geomyces destructans* fungus in bats from the Czech Republic and Slovakia. *PLoS ONE* 5, e13853

14 Puechmaille, S.J. *et al.* (2010) White-nose syndrome fungus (*Geomyces destructans*) in bat, France. *Emerg. Infect. Dis.* 16, 290–293
15 Wibbelt, G. *et al.* (2010) White-nose syndrome fungus (*Geomyces destructans*) in bats, Europe. *Emerg. Infect. Dis.* 16, 1237–1242
16 Puechmaille, S.J. *et al.* (2011) Pan-European distribution of White-nose syndrome fungus (*Geomyces destructans*) not associated with mass mortality. *PLoS ONE* 6, e19167
17 Meteyer, C.U. *et al.* (2009) Histopathologic criteria to confirm white-nose syndrome in bats. *J. Vet. Diagn. Invest.* 21, 411–414
18 Reeder, D.M. and Turner, G.G. (2008) Working together to combat White Nose Syndrome: a report of a meeting on 9-11 June 2008, in Albany, New York. *Bat Res. News* 49, 75–78
19 Courtin, F. *et al.* (2010) Pathologic findings and liver elements in hibernating bats with white-nose syndrome. *Vet. Pathol.* 47, 214–219
20 Brack, V. and Twente, J.W. (1985) The duration of the period of hibernation of three species of vespertilionid bats. I. Field studies. *Can. J. Zoolog.* 63, 2952–2954
21 Britzke, E.R. *et al.* (2010) Use of temperature-sensitive transmitters to monitor the temperature profiles of hibernating bats affected with White-nose syndrome. *Northeast. Nat.* 17, 239–246
22 Twente, J.W. *et al.* (1985) The duration of the period of hibernation of three species of vespertilionid bats. II. Laboratory studies. *Can. J. Zoolog.* 63, 2955–2961
23 Reichard, J.D. and Kunz, T.H. (2009) White-nose syndrome inflicts lasting injuries to the wings of little brown myotis (*Myotis lucifugus*). *Acta Chiropt.* 11, 457–464
24 Brooks, R.T. (2011) Declines in summer bat activity in central New England 4 years following the initial detection of white-nose syndrome. *Biodivers. Conserv.* 20, 2537–2541
25 Dzal, Y. *et al.* (2011) Going, going, gone: the impact of white-nose syndrome on the summer activity of the little brown bat (*Myotis lucifugus*). *Biol. Lett.* 7, 392–394
26 Masing, M. (1984) *Lendlased*, Pääsuke
27 Feldmann, R. (1984) Teichfledermaus - Myotis dasycneme (Boie, 1825). In *Die Säugetiere Westfalens* (Schröpfer, R. *et al.*, eds), pp. 107–111, Westfälisches Museum für Naturkunde
28 Wilder, A.P. *et al.* (2011) Risk factors associated with mortality from White-nose syndrome among hibernating bat colonies. *Biol. Lett.* DOI: 10.1098/rsbl.2011.0355
29 Lindner, D.L. *et al.* (2011) DNA-based detection of the fungal pathogen *Geomyces destructans* in soils from bat hibernacula. *Mycologia* 171, 231–233
30 Turner, G.G. and Reeder, D.M. (2009) Update of White Nose Syndrome in bats, September 2009. *Bat Res. News* 50, 47–53
31 Kannan, K. *et al.* (2010) High concentrations of persistent organic pollutants including PCBs, DDT, PBDEs and PFOS in little brown bats with white-nose syndrome in New York, USA. *Chemosphere* 80, 613–618
32 Li, L. *et al.* (2010) Bat guano virome: predominance of dietary viruses from insects and plants plus novel mammalian viruses. *J. Virol.* 84, 6955–6965
33 Kunz, T.H. *et al.* (2011) White-nose syndrome: an overview of ongoing and future research needs. In *Proceedings of Protection of Threatened Bats at Coal Mines: A Technical Interactive Forum* (Vories, K.C., Caswell, A.H. and Price, T.W., eds), pp. 195–209, US DOI, Office of Surface Mining and Coal Research Center, Southern Illinois University
34 Thomas, D.W. *et al.* (1990) Winter energy budgets and cost of arousals for hibernating little brown bats, *Myotis lucifugus*. *J. Mammal.* 71, 475–479
35 Carey, H.V. *et al.* (2003) Mammalian hibernation: cellular and molecular responses to depressed metabolism and low temperature. *Physiol. Rev.* 83, 1153–1181
36 Bouma, H.R. *et al.* (2011) Low body temperature governs the decline of circulating lymphocytes during hibernation through sphingosine-1-phosphate. *Proc. Natl. Acad. Sci. U.S.A.* 108, 2052–2057
37 Schuppe, H-C. *et al.* (1998) Immunomodulation by heavy metal compounds. *Clin. Dermatol.* 16, 149–157
38 Lawrence, D.A. and McCabe, M.J. (2002) Immunomodulation by metals. *Int. Immunopharmacol.* 2, 293–302
39 vanEngelsdorp, D. *et al.* (2009) Colony Collapse Disorder: a descriptive study. *PLoS ONE* 4, e6481
40 Bromenshenk, J.J. *et al.* (2010) Iridovirus and Microsporidian linked to honey bee colony decline. *PLoS ONE* 5, e13181
41 O'Shea, T.J. and Johnston, J.J. (2009) Environmental contaminants and bats: investigating exposure and effects, In *Ecological and Behavioral Methods for the Study of Bats* (2nd edn) (Kunz, T.H. and Parsons, S., eds), pp. 500–528, Johns Hopkins University Press
42 Walsh, A.L. *et al.* (2003) The United Kingdom national bat monitoring programme: turning conservation goals into tangible results. In *Monitoring Trends in Bat Populations of the United States and Territories: Problems and Prospects* (O'Shea, T.J. and Bogan, M.A., eds), pp. 103–118, U.S. Geological Survey, Biological Resources Division, Information and Technology Report
43 Jones, G. *et al.* (2009) Carpe noctem: the importance of bats as bioindicators. *Endang. Species Res.* 8, 93–115
44 Oh, Y. *et al.* (2008) Transcriptome analysis reveals new insight into appressorium formation and function in the rice blast fungus *Magnaporthe oryzae*. *Genome Biol.* 9, R85
45 Rosenblum, E.B. *et al.* (2008) Global gene expression profiles for life stages of the deadly amphibian pathogen *Batrachochytrium dendrobatidis*. *Proc. Natl. Acad. Sci. U.S.A.* 105, 17034–17039
46 Wang, B. *et al.* (2010) Survey of the transcriptome of *Aspergillus oryzae* via massively parallel mRNA sequencing. *Nucleic Acids Res.* 38, 5075–5087
47 Bratsch, S. *et al.* (2011) The little brown bat, *M. lucifugus*, displays a highly diverse V_H, D_H and J_H repertoire but little evidence of somatic hypermutation. *Dev. Comp. Immunol.* 35, 421–430
48 Butler, J.E. *et al.* (2011) The two suborders of chiropterans have the canonical heavy-chain immunoglobulin (Ig) gene repertoire of eutherian mammals. *Dev. Comp. Immunol.* 35, 273–284
49 Puechmaille, S.J. *et al.* (2011) Effect of sample preservation methods on the viability of *Geomyces destructans*, the fungus associated with white-nose syndrome in bats. *Acta Chiropt.* 13, 217–221
50 Frick, W.F. *et al.* (2010) Influence of climate and reproductive timing on demography of little brown myotis *Myotis lucifugus*. *J. Anim. Ecol.* 79, 128–136
51 Chaturvedi, V. *et al.* (2010) Morphological and molecular characterizations of psychrophilic fungus *Geomyces destructans* from New York bats with White Nose Syndrome (WNS). *PLoS ONE* 5, e10783
52 Fitzpatrick, D.A. *et al.* (2006) A fungal phylogeny based on 42 complete genomes derived from supertree and combined gene analysis. *BMC Evol. Biol.* 6, 99