BAT WHITE-NOSE SYNDROME IN 2014: A BRIEF ASSESSMENT SEVEN YEARS AFTER DISCOVERY OF A VIRULENT FUNGAL PATHOGEN IN NORTH AMERICA

Jeremy T. H. Coleman and Jonathan D. Reichard, U.S. Fish and Wildlife Service, 300 Westgate Center Drive, Hadley, MA 01035, USA describe the impact on bat populations following the arrival of *Pseudogymnoascus destructans* in North America. Corresponding author email: jeremy_coleman@fws.gov

Keywords: white-nose syndrome, bats, *Pseudogymnoascus destructans*, invasive fungal pathogen, *Myotis*

North American bats face numerous challenges on the modern landscape, including habitat loss, climate change, and energy development, but none is as immediate a threat to multiple species as white-nose syndrome, a fungal disease of unknown origin that is responsible for unparalleled rapid declines in bat populations. White-nose syndrome (WNS) was unknown to science before it was discovered in New York in 2007 and took us all by surprise. Since then the conservation and scientific communities have come together to mount a coordinated international effort to address research and management needs to respond to this growing disaster. Among many accomplishments, we have made great progress in understanding the disease, the biology of the causative fungus, and the biology and physiology of hibernating bats; we have developed guidance focusing on containing the fungus to slow its spread; we are exploring multiple novel approaches to treat bats and affected environments in ways that reduce the impacts of the disease; and we are now field testing a standardized and robust monitoring strategy to assess population trends for all bat species across North America. The incredible progress made to date would not be possible without the dedication of the many individuals, institutions, and government agencies who have engaged in this issue and lent their support. Robust insectivorous bat populations are an important part of a healthy ecosystem, and we benefit greatly from the services these voracious predators provide. Therefore, we must do all we can to understand and manage WNS if we are to conserve our native bat species.

The first evidence of WNS in North America was recorded in photographs of affected bats at Howe's Cave west of Albany, New York, in 2006; WNS was not actually discovered until January 2007, however, when it was found at four other caves in Schoharie County, New York (Blehert *et al*. 2009, Turner & Reeder 2009). The initial observation from February 2006 stands as the primary case for an epizootic that, by September 2014, has spread to hundreds of bat hibernacula across 25 states and five Canadian provinces (Figure 1). The fungus that causes WNS, *Pseudogymnoascus* (formerly *Geomyces*) *destructans* (Lorch *et al*. 2011), has also been identified at many sites in three additional states, such that hibernating bats across most of eastern North America are now within the range of the pathogen. From what appears to be a single point of introduction, *P. destructans* has spread rapidly, propagated largely by bat-to-bat or bat-to-environment-to-bat contact. Human-mediated transport may also have contributed to the spread based on the potential for the fungus to be carried between caves on clothing or personal gear (USGS 2009), the ability of the fungus to remain viable for long durations in the absence of bat hosts (Lorch *et al*. 2013, Hoyt *et al*. 2014), and conspicuous longer distance jumps to heavily visited caves before detection at large bat hibernacula close by.

White-nose syndrome affects hibernating bat species during the winter, when individuals of many species are found in the cool and humid confines of natural caves, abandoned mines, or similar structures (collectively known as hibernacula when used by hibernating animals). All bat species in the

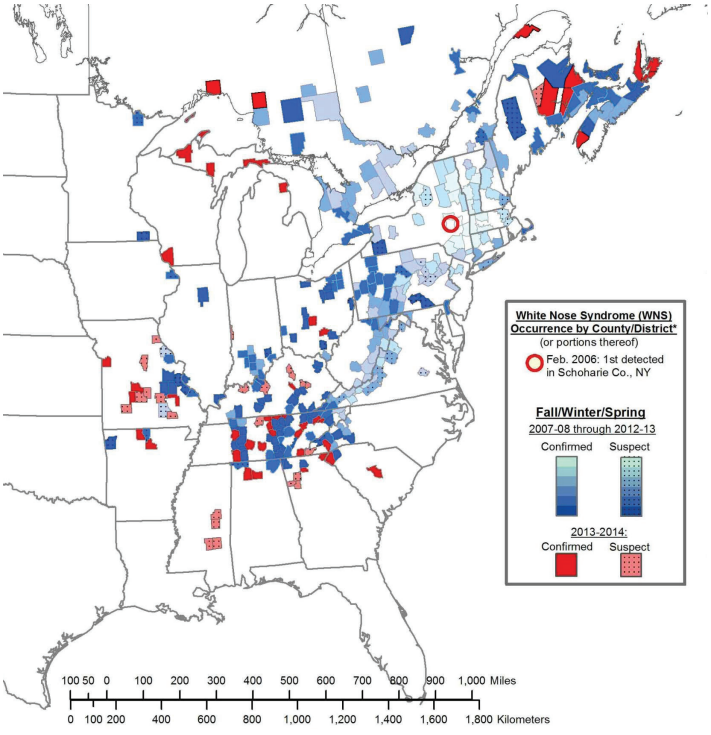

Figure 1. A map of white-nose syndrome occurrence in eastern North America by year, up to 3 September 2014. The map depicts counties or districts containing bat hibernacula or roosts in which WNS has been identified or confirmed through histopathology ("Confirmed"), or the causative fungus *Pseudogymnoascus destructans* or signs of the disease have been detected ("Suspect"). Map credit: Lindsey Heffernan, Pennsylvania Game Commission, with many contributors.

DOI: 10.1564/v25_dec_08

WNS-affected range are insectivorous and all affected species use hibernation to survive winter months when insect prey is scarce or nonexistent. During bouts of deep torpor that constitute hibernation, a bat's metabolism slows considerably and body temperature drops to near ambient conditions, thus economizing consumption of stored fat that makes up the majority of the bat's winter energy supply. Unfortunately, cool subterranean temperatures suitable for hibernation are also ideal for growth of *P. destructans*, a psychrophilic (cold-loving) fungus with a growth range between roughly 1 and 19°C (Verant *et al*. 2012). Such conditions are common for caves and mines across the continent (Perry 2013), and hibernating bats in every state may be found using these same cool and humid conditions. By 2014, seven bat species have been confirmed with WNS in North America: five *Myotis* species, *Perimyotis subflavus*, and *Eptesicus fuscus*, and five additional species have been documented carrying *P. destructans* without demonstrating the lesions characteristic of WNS. Response to the pathogen differs widely by species, with some, such as the federally listed Virginia big-eared bat (*Corynorhinus townsendii virginianus*) appearing to exhibit no deleterious effects, and others, such as the little brown myotis (*Myotis lucifugus*), the tri-colored bat (*P. subflavus*), and the northern long-eared myotis (*M. septentrionalis*), demonstrating significant losses, in excess of 90% in many locations (Turner *et al*. 2011). Because the little brown myotis was the most numerous hibernating bat in eastern North America, and has been heavily impacted, total losses for all hibernating species affected by WNS have been estimated at 90%. The reason(s) underlying the apparent differences in the response to WNS by species is unknown and remains an active area of research that we hope will eventually inform management options.

The unknown origin of *P. destructans* and reason behind its sudden appearance in New York remain the source of speculation. Following the discovery of WNS, researchers identified that multiple species of bats in Europe carry *P. destructans*, but no mortality attributable to the fungus has been documented there (Peuchmaille *et al*. 2010, Wibbelt *et al*. 2010, Martínková *et al*. 2010). Subsequent molecular evidence and spatio-temporal analyses of the fungus from around North America and Europe suggest that *P. destructans* is both a relatively new arrival to North America and is rapidly expanding its range (Jeffrey Foster, University of New Hampshire, personal communication). Lab tests have also revealed that *P. destructans* found in Europe is potentially just as lethal to North American bats as samples from North America, which further supports the idea that *P. destructans* is a newly arrived pathogen infecting a naïve host population (Warnecke *et al*. 2012). More recently, European bats were found to suffer the same cutaneous infections that technically define WNS in North America, but again, the infections are less severe and infected bats present no other signs of the disease as it is known in the U.S. and Canada (Meteyer *et al*. 2009, Pikula *et al*. 2012, Zukal *et al*. 2014). It is important to note that while Europe is the likely source of the fungus, phylogenetic studies have so far not identified a close match to North American samples. Without this evidence, it is possible there may be a non-European source of the fungus that is yet to be discovered.

Bats have demonstrated their effectiveness at spreading the fungus, but limiting spread by people to prevent potential large jumps to new regions is something that can and must be done. With concern for the potential human role in spreading *P. destructans* around North America, and possibly off the continent, many cave visitors and bat researchers have committed to cautionary measures and decontamination procedures that minimize the likelihood of inadvertently transporting the fungus (or other microbes) among potentially vulnerable ecosystems (see www.whitenosesyndrome.org for current decontamination protocols). Even as WNS continues to spread through bat vectors, compliance with these measures marks increased awareness of the potential unintended consequences of human interactions with the natural world, and may prevent future expansion or exacerbation of wildlife pathogens. As we have seen countless times with invasive species, once an exotic species has become established in a new environment it is very difficult to manage or eradicate (Zavaleta *et al*. 2001). One familiar idiom that is relevant here states that once out, you cannot put the genie back in the bottle. Because behavioral and geographic barriers have potential to hinder the spread of *P. destructans* among bat populations (Miller-Butterworth *et al*. 2014), and given that *P. destructans* can survive in the cave environment in the absence of bats for years and maybe indefinitely (Lorch *et al*. 2013, Hoyt *et al*. 2014), the most effective strategy for containing the spread of the fungus and limiting impacts on bats over the long-term is to prevent it from arriving in the first place.

The ultimate mechanisms that cause death from WNS continue to elude us, but we continue to make progress in our understanding. Laboratory experiments with artificially inoculated bats and observations of bats naturally infected with *P. destructans* in caves both demonstrate that WNS increases the frequency and duration of arousals from torpor in affected bats, leading to accelerated fat depletion (Reeder *et al*. 2012, Warnecke *et al*. 2012). While energy depletion from altered torpor patterns may be responsible for early emergence of bats from hibernacula in winter, which can lead to death from hypothermia and predation in addition to starvation, this does not explain why the infection causes increased arousals in the first place. Cryan *et al*. (2010) hypothesized that wing damage resulting from WNS accelerates evaporative water loss leading to dehydration, which may result in increasing frequency of arousals to replenish lost water. Dehydration can also lead to electrolyte imbalance, which is known to have serious physiological impacts in mammals, and may contribute to increasing arousal patterns and mortality from WNS (Willis *et al*. 2010, Cryan *et al*. 2013).

It may be that multiple factors contribute to mortality from WNS, but there is increasing evidence that not every bat with WNS ends up dead. We know that mortality rates differ by species (Turner *et al*. 2011), and that several species continue to experience significant losses. Encouragingly, we have a few examples of little brown myotis summer colonies persisting in pockets around the affected area (e.g. Dobony *et al*. 2011). Biologists also continue to document small numbers of little brown myotis in some caves in winter, and banding studies of these remnant populations have revealed that some individuals are surviving multiple years in WNS-affected sites (Figure 2). Unfortunately, while survivors have been documented for little brown myotis, similar observations have not been reported for other heavily affected species. Nevertheless, these

Figure 2. Little brown myotis (*Myotis lucifugus*) in a cave in Vermont, USA, in 2013. The bats are identified by coded wing bands, and are known to have survived at least one year after natural exposure to *Pseudogymnoascus destructans*, the fungus that causes white-nose syndrome. Photo credit: Jeremy T. H. Coleman, U.S. Fish and Wildlife Service.

findings are cause for cautious optimism when there has been so little hope for the future of species decimated by WNS.

White-nose syndrome is one of the most devastating wildlife diseases in recorded history, and its impacts on bat populations are unrivaled for mammalian species. Unfortunately, WNS is one in a growing trend of fungal pathogens wreaking havoc on wildlife populations across the globe, which stand to cause irreplaceable losses to biodiversity (Fisher *et al*. 2013). The disease continued to spread in 2014 and is anticipated to advance into the upper midwestern U.S., the plains of Canada, and on into western North America (e.g. Maher *et al*. 2012). Researchers continue to make progress toward finding ways to control the fungus and to treat bats exposed to it, but implementing such treatments safely and on a broad scale is still few years away at best. In the meantime, bat losses continue to mount and the disease continues to spread, with untold impacts to the environment and the economy. With some little brown myotis persisting in the wake of the disease, there is some slight hope for a resilient population of that species that could recover to viable status. We can only hope it is not too little, too late.

References

Blehert, D. S., A. C. Hicks, M. Behr, C. U. Meteyer, B. M. Berlowski-Zier, E. L. Buckles, J. T. H. Coleman, S. R. Darling, A. Gargas, R. Niver, J. C. Okoniewski, R. J. Rudd, & W. B. Stone. 2009. Bat white-nose syndrome: an emerging fungal pathogen? *Science* **323**:227.

Cryan, P. M., C. U. Meteyer, J. G. Boyles, & D. S. Blehert. 2010. Wing pathology of white-nose syndrome in bats suggests life-threatening disruption of physiology. *BMC Biology* **8**:135.

Cryan, P. M., C. U. Meteyer, D. S. Blehert, J. M. Lorch, D. M. Reeder, G. G. Turner, J. Webb, M. Behr, M. Verant, R. E. Russell, & K. T. Castle. 2013. Electrolyte depletion in white-nose syndrome bats. *Journal of Wildlife Diseases* **94**:398-402.

Dobony, C. A., A. C. Hicks, K. E. Langwig, R. I. von Linden, J. C. Okoniewski, & R.. E. Rainbolt. 2011. Little brown myotis persist despite exposure to white-nose syndrome. *Journal of Fish and Wildlife Management* **2**:190–195.

Fisher, M. C., D. A. Henk, C. J. Briggs, J. S. Brownstein, L. C. Madoff, S. L. McCraw, & S. J. Gurr. 2012. Emerging fungal threats to animal, plant and ecosystem health. *Nature* **484**:186–194.

Hoyt, J. R., K. E. Langwig, J. Okoniewski, W. F. Frick, W. B. Stone, & A. M Kilpatrick. 2014. Long-Term persistence of *Pseudogymnoascus destructans*, the causative agent of white-nose syndrome, in the absence of bats. *EcoHealth* 1612-9202. DOI: 10.1007/s10393-014-0981-4.

Lorch, J. M., C. U. Meteyer, M. J. Behr, J. G. Boyles, P. M. Cryan, A. C. Hicks, A. E. Ballmann, J. T. H. Coleman, D. N. Redell, D. M. Reeder, & D. S. Blehert. 2011. Experimental infection of bats with Geomyces destructans causes white-nose syndrome. Nature 480:376-378.

Lorch, J. M., L. K. Muller, R. E. Russell, M. O'Connor, D. L. Lindner, & D. S. Blehert. 2013. Distribution and environmental persistence of the causative agent of white-nose syndrome, *Geomyces destructans*, in bat hibernacula of the eastern United States. *Applied and Environmental Microbiology* **79**:1293-1301.

Maher, S. P., A. M. Kramer, J. T. Pulliam, M. A. Zokan, S. E. Bowden, H. D. Barton, K. Magori, and J. M. Drake. 2012. Spread of white-nose syndrome on a network regulated by geography and climate. Nature Communications. **3**:1306

Martínková, N., P. Bačkor, T. Bartonička, P. Blažková, J. Červený, *et al*. 2010. Increasing incidence of *Geomyces destructans* fungus in bats from the Czech Republic and Slovakia. *PLoS One* **5**:e13853.

Meteyer, C. U., E. L. Buckles, D. S. Blehert, A. C. Hicks, D. E. Green, V. Shearn-Bochsler, N. J. Thomas, A. Gargas, & M. J. Behr. 2009. Histopathologic criteria to confirm white-nose syndrome in bats. *Journal of Veterinary Diagostic Investigations* **21**:411-414.

Miller-Butterworth, C. M., M. J. Vonhof, J. Rosenstern, G. G. Turner, & A. L. Russel. 2014. Genetic Structure of Little Brown Bats (Myotis lucifugus) Corresponds With Spread of White-Nose Syndrome Among Hibernacula. Journal of Heredity 105:354-364.

Perry, R. W. 2013. A review of factors affecting cave climates for hibernating bats in temperate North America. Environmental Reviews 21:28-39.

Pikula, J., H. Bandouchova, L. Novotny, C. U. Meteyer, J. Zukal, *et al*. 2012. Histopathology confirms white-nose syndrome in bats in Europe. *Journal of Wildlife Diseases* **48**:207–211.

Puechmaille, S. J., P. Verdeyroux, H. Fuller, M. A. Gouilh, M. Bekaert, & E. C. Teeling. 2010. White-nose syndrome fungus (*Geomyces destructans*) in bat, France. *Emerging Infectious Diseases* **16**:290-293.

Reeder, D. M., C. L. Frank, G. G. Turner, C. U. Meteyer, A. Kurta, *et al*. 2012. Frequent Arousal from Hibernation Linked to Severity of Infection and Mortality in Bats with White-Nose Syndrome. *PLoS One* **7**: e38920

Turner, G. G. & D. M. Reeder. 2009. Update of white-nose syndrome in bats, September 2009. *Bat Research News* **50**:47-53.

Turner, G. G., D. M. Reeder, & J. T. H. Coleman. 2011, A Five-year Assessment of Mortality and Geographic Spread of White-nose Syndrome in North American Bats and a Look to the Future *Bat Research News* **52**:13-27.

US Geological Survey National Wildlife Health Center (USGS). 2009. Update on white-nose syndrome. Wildlife Health Bulletin 2009–03. 11 December 2009. <http://www.nwhc.usgs.gov/publications/wildlife_health_bulletins/WHB_2009-03_WNS.pdf> Accessed 1 Oct 2014.

Verant, M. L., J. G. Boyles, W. Waldrep JR., G. Wibbelt, & D. S. Blehert. 2012. Temperature-dependent growth of *Geomyces destructans*, the fungus that causes bat white-nose syndrome. *PLoS One* 7:e46280.

Warnecke, L., J. M. Turner, T. K. Bollinger, J. M. Lorch, V. Misra, P. M. Cryan, G. Wibbelt, D. S. Blehert, & C. K. R. Willis. 2012. Inoculation of bats with European *Geomyces destructans* supports the novel pathogen hypothesis for the origin of white-nose syndrome. *Proceedings of the National Academy of Sciences, USA* **109**:6999-7003.

Wibbelt, G., A. Kurth, D. Hellmann, M. Weishaar, A. Barlow, M. Veith, J. Prüger, T. Görföl, L. Grosche, F. Bontadina, U. Zöphel, H. Seidl, P. M. Cryan, & D. S. Blehert. 2010. White-nose syndrome fungus (*Geomyces destructans*) in bats, Europe. *Emerging Infectious Diseases* **16**:1237-1242.

Willis, C. K. R., A. K. Menzies, J. G. Boyles, & M. S. Wojciechowski. 2011. Evaporative water loss is a plausible explanation for mortality of bats from white-nose syndrome. *Integrative and Comparative Biology* **51**:364–373.

Zavaleta, E. S., R. J. Hobbs, & H. A. Mooney. 2001. Viewing invasive species removal in a whole-ecosystem context. *Trends in Ecology & Evolution*. **16**:454-459.

Zukal, J., H. Bandouchova, T. Bartonicka, H. Berkova, V. Brack, *et al*. 2014. White-Nose Syndrome Fungus: A Generalist Pathogen of Hibernating Bats. *PLoS One* **9**: e97224.

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