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Update of White Nose Syndrome in Bats, September 2009

Gregory G. Turner[1] and DeeAnn M. Reeder[2]

[1]*Pennsylvania Game Commission, 2001 Elmerton Avenue, Harrisburg, PA 17110, and*
[2]*Department of Biology, Bucknell University, Lewisburg, PA 17837*
E-mail: grturner@state.pa.us

Background

Just west of Albany, New York, a strange fungus and 18 dead bats were first photographed in Howe Cave in February 2006 (Blehert et al., 2009). However, this isolated incident was not odd enough to spark serious concern at the time and was not reported until 2008, as the problem became more widespread and publicly recognized. During routine counts of hibernating bats conducted from January through March 2007, mortality and bats with odd clinical signs and behaviors were documented by biologists from the New York Department of Environmental Conservation at four sites close to Howe Cave. Three different species of *Myotis* (*M. lucifugus, M. septentrionalis,* and *M. sodalis*) and *Perimyotis subflavus* were affected. The magnitude of the problem became apparent during winter 2008, as the newly described White Nose Syndrome (WNS) spread to three adjacent states: Connecticut, Massachusetts, and Vermont. No causative agent was known, but estimates of mortality at four sites in New York previously confirmed as infected and housing wintering populations of over 1,000 animals revealed an alarming 90% reduction in total number of bats when compared to pre-WNS counts (A. Hicks, pers. comm.). By the time that a meeting was convened in Albany, New York, to discuss the phenomenon (Reeder and Turner, 2008), a suite of clinical signs had been described and WNS had spread to all 28 sites checked within 130 km of the epicenter and 5 of 19 sites that were 130–200 km away (A. Hicks, pers. comm.).

Researchers scrambled to pull resources together and establish new collaborative studies in the short time between the meeting in June and the start of the 2008–2009 hibernating season. As reported at Albany, laboratory investigations did not point definitively towards any specific causative agent but did consistently show evidence of the fungus (Figure 1), and most dead animals were in poor body condition with little-to-no body fat. Using this as a starting point and based upon hypotheses generated at the meeting in June 2008 (Reeder and Turner, 2008), researchers conducted multiple studies on a regional scale during winter 2008–2009, which included examination of bats from both affected and unaffected sites. Studies included investigation of patterns of arousal and torpor, measurement of metabolic rates, examination of body condition and types of fat across the hibernating season, bacterial flora of digestive systems, and immune response.

Continued Spread

While these studies were being conducted, WNS continued to spread. As of August 2009, two more species (*Eptesicus fuscus* and *M. leibii*) were confirmed as affected, which meant that all six species of hibernating cave bats in the northeastern United States were susceptible. Total mortality at closely monitored sites with multiple years of infection in New York, Massachusetts, and Vermont averaged 95% (A. Hicks, pers. comm.). In 2009, WNS spread over 800 km from the epicenter and was confirmed in New Jersey,

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Figure 1. Little brown bat (*Myotis lucifugus*) afflicted with white nose syndrome. The bat was found in an abandoned mine in Lackawanna County, Pennsylvania. Photograph courtesy of Greg Turner, Pennsylvania Game Commission.

Pennsylvania, West Virginia, and Virginia (Figure 2).

Anthropogenic transmission?—Concern over anthropogenic transmission was raised initially in 2008, after discovery that all but two or three new WNS sites found that year had been visited by either biologists or recreational users who had been in at least one of the original four sites noted in 2007. This concern highlighted the need for establishing decontamination protocols, as well as the need to verify that they were effective. Large-scale movements (jumps) of WNS occurred in 2009 into areas where WNS was not thought to exist, leaving sites in-between unaffected. These jumps occurred in central Pennsylvania, West Virginia, and Virginia, and several factors pointed toward a human connection. First, most sites had small hibernating populations. Although the possibility existed for spread by an infected bat that had migrated a long distance to a small hibernating population in the new area, it was unlikely that this would have occurred multiple times, particularly when hibernacula with much larger populations existed nearby, as was the case at several of these newly infected sites. Second, most new sites had very high recreational use, and third, several sites were confirmed to have visitation by people or mud-covered gear that had been in affected sites in New York, prior to establishment of decontamination protocols. Due to this suspicion that anthropogenic movements were likely, that no causal agent had been identified, and that decontamination protocols were not thoroughly tested, some states reduced or halted population surveys during hibernation and the U.S. Fish and Wildlife Service requested a voluntary moratorium on caving until more was learned (http://www.fws.gov/northeast/wnscaveadvisory.html).

Bat-to-bat transmission?—In addition to anthropogenic spread, bat-to-bat transmission surely also occurred and may have been the primary mechanism of spread. This mode of transmission was supported by a wave-like pattern of spread away from the epicenter (in addition to long-distance jumps) and the fact that in both 2008 and 2009 some newly affected sites had not been visited by

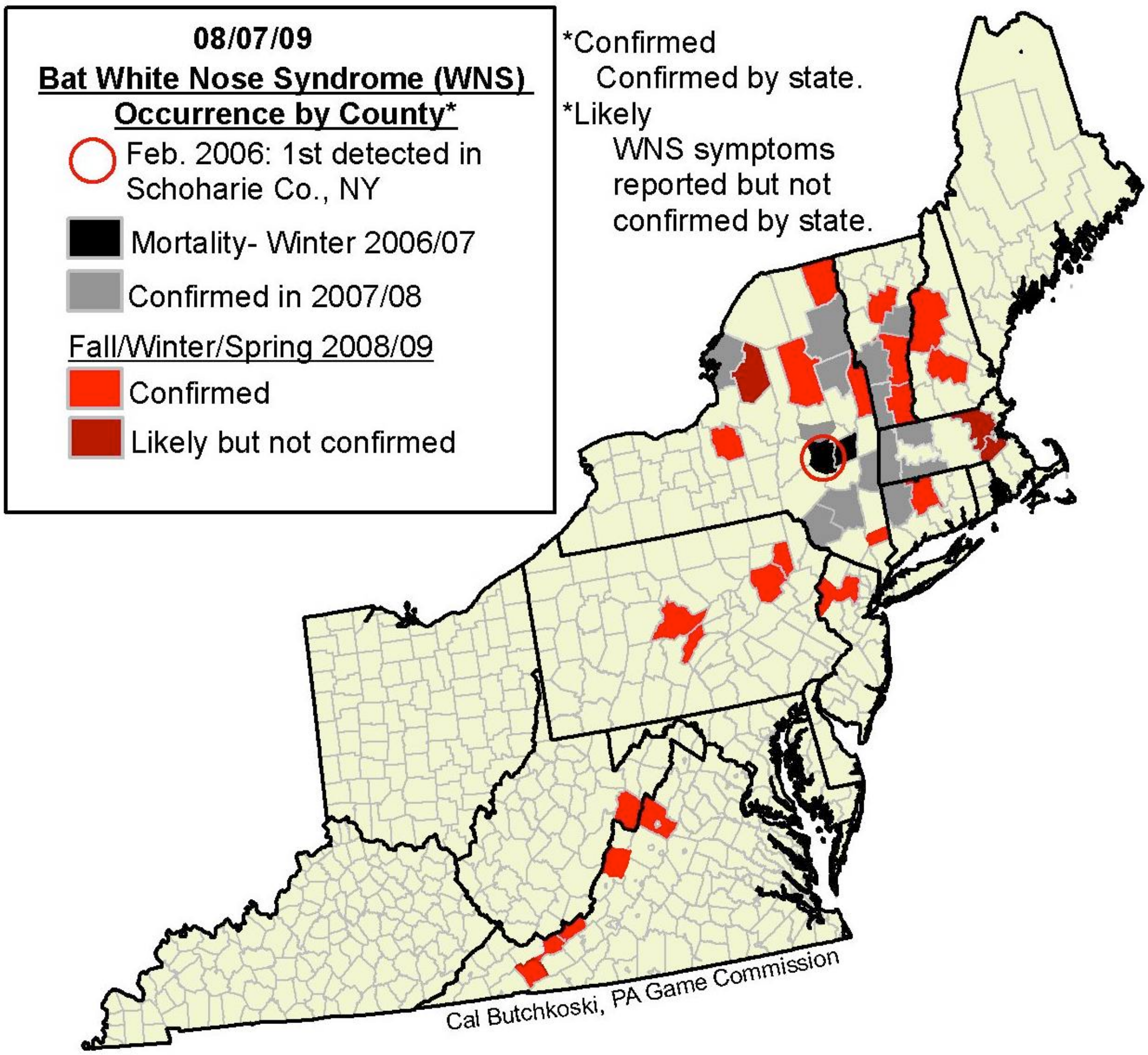

Figure 2. Map depicting the spread of white nose syndrome by county across hibernating seasons. Courtesy of Cal Butchkoski, Pennsylvania Game Commission.

researchers or recreational users for several years prior to infection by WNS.

New Results

Most studies conducted in winter 2009 are still in the data-analysis or manuscript-preparation phase and thus not available for reporting. However, several publications have recently appeared, and preliminary data have been generously provided by many researchers, allowing us to highlight recent findings. For example, the study investigating the thermoregulatory patterns of hibernating bats documented that bats with WNS display atypical patterns of torpor that could explain the significant decrease in body condition experienced by WNS-affected bats (D. M. Reeder et al., pers. comm.). An investigation of metabolic rates of hibernating bats showed that the rates of bats in New York at affected sites were two-to-three times higher than the rates of bats in Pennsylvania (A. Janicki and T. Tomasi, pers. comm.). According to these authors, the higher metabolic rates during torpor (assuming no changes in arousal patterns/metabolism) in affected bats would require an additional 0.7 grams of fat over the winter. An analysis of the digestive tracks revealed that WNS-affected bats have a reduced bacterial flora in their digestive tracts, especially a reduction in those species that produce the enzyme chitinase (J. Whitaker, Jr., and K. Dannelly, pers. comm.).

Data examining the body fat of healthy and affected bats across the hibernating season are too preliminary to provide any insight at this time (J. Reichard and T. Kunz, pers. comm.). However the same researchers showed that *M. lucifugus* at maternity colonies near affected hibernacula began the summer with poorer body condition but achieved typical post-reproductive body mass when compared to bats studied at these sites prior to WNS; at one site, the bats also remained active later into fall and declined in body mass before entering hibernation (Reichard and Kunz, in press). Another study, comparing a small group of bats from an affected hibernaculum with those from a clean hibernaculum noted a dramatic difference in the body mass index. While field necropsy of these specimens suggested reduced brown adipose tissue in the affected bats, the investigator did not consider this reduction sufficient to explain mortality (J. Fallon, pers. comm.).

Fallon also noted that preliminary data suggested decreased circulating leukocytes in bats with WNS, which may indicate immunosuppression. This is supported by a different study that found alterations in B-cell mediated immune function in affected bats independent of differences in body mass (R. Jacob, D. M. Reeder, and K. A. Field, pers. comm.). Additionally, M. Moore and T. Kunz reported that differences existed between the innate immune response of bats from affected and unaffected sites, as well as between bats with and without visible signs of the syndrome at affected sites.

In addition to exploring how WNS affects physiology and behavior, other research focused on culturing and characterizing the fungus suspected to be the causative agent. Following up on the first study of the WNS-associated fungus (Blehert et al., 2009), Gargas et al. (2009) named the species and further characterized its biology. The newly described *Geomyces destructans* was described as the causative agent of the skin infection that is hallmark of WNS-affected bats, and the unique morphology of this fungus was unlike that of any previously described species of *Geomyces*. Preliminary analyses of infection trials also demonstrated that *G. destructans* is transmissible from affected to clean bats (D. Blehert, pers. comm.).

Another study investigated the prevalence of *G. destructans* among bat hibernacula east of the Mississippi River. To examine this question, sediment samples were collected from hibernacula within and outside the WNS-affected region in winter 2009, and preliminary analyses indicated a diversity of fungi related to, but distinct from *G. destructans* (D. Blehert, pers. comm.). The presence of these closely related species made analyzing samples for *G. destructans* labor intensive. Nonetheless, Blehert noted that *G. destructans* was found in sediments from a number of hibernacula within the WNS-infected region.

Scientific studies supporting the fungus as the sole causative agent of mortality are on-going but not conclusive at this time. Many researchers within the WNS community currently believe that the fungus is the most likely culprit. Additionally, anecdotal evidence of the likely role of *G. destructans* in causing WNS continues to grow. Consider first, that if humans are capable of spreading this fungus from site to site via caving equipment, the odds of transmission would appear greater with a resistant fungal spore than with other pathogens. Although fungi are common inhabitants of caves and mines and are occasionally documented on live bats, the unique morphological characteristics of this fungus had never been seen and/or reported in the United States until 2006, and the fungus continues to be found only in sites confirmed to be affected and displaying high mortality. In one central Pennsylvania site, the fungus was noted and confirmed prior to the observation of the clinical signs of roost shifting, distortions of typical arousal patterns, lethargy, and early emergence or death. Further, these clinical signs increased as growth of the fungus on individual bats progressed and as a greater number of bats became affected. Finally, evidence that the *G. destructans* can be found in sediments in affected sites supports the hypothesis that humans may represent a potential vector.

Research investigating the efficacy of fungicides for decontaminating affected gear and compounds for potential use as treatment for affected bats is also underway. Preliminary results indicate that the vegetative structures of a similar, but non-pathogenic fungus are rather easy to kill but the spores are quite resistant (H. Barton, pers. comm.). Thus far, over 80 compounds have been tested by Barton and the efficacies of these treatments are being analyzed. The combining of different compounds to achieve synergistic decontamination and/or treatment, while causing minimal damage to either the unique cave biota or the performance of technical gear is a major challenge but is showing promise. Barton preliminarily notes that washing caving equipment in Woolite (Reckitt Benckiser, Inc., Parsippany, New Jersey) prior to decontamination is critical, because it removes mud, clay, and other sediments that contain charged surfaces that attract disinfectants, decreasing their efficacy. These studies and their widespread application hinge on confirmation of the fungus as a causative agent of mortality.

If the fungus is eventually documented as the causative agent of WNS, the immediate question that follows is: where did this fungus come from? It is possible that this pathogenic fungus evolved from one of the naturally occurring and closely related species found in nearly all hibernacula investigated so far, but the fungus could also be an introduced species to which North American bats have no resistance. On this front, several European scientists, upon hearing about WNS, have noted that a fungus with similar morphological traits can be found on their hibernating bats but with no signs of mass mortality at this time. The arrival of an exotic cold-loving fungus is a “perfect storm” for killing hibernating bats, because bats have extremely high rates of contact, the fungus attacks them at a time when their capability for mounting any immune response is minimized to save energy, and this period of inactivity and immune suppression is lengthy. Regardless of the causative agent(s), the levels of mortality are unprecedented in the known history of bats, and the potential loss of millions of bats across this region gives everyone reason to be greatly concerned.

Hope for the Future?

Despite all this, there may be hope for North American bats. If the fungus is actually the causative agent, then what we know suggests that non-hibernating bats should largely not be affected as the fungi will not have the ability to grow for prolonged periods. Evidence from Pennsylvania suggests a single year's natural spread may be only around 15–20 miles per year without anthropogenic transmission. Therefore, if we can determine the mechanisms and timing by which natural transmission is occurring, we may be able to slow the spread and allow for containment or treatments to be developed. Progress is also being made on testing procedures to decontaminate all gear used underground. Compounds that have anti-fungal capabilities are now being tested to determine whether there are impacts to bats or the many unique and globally rare creatures that live among them and will hopefully lead to some management options. These compounds may help delay or break the cycle of transmission, or even better, they may increase the survival rate at affected sites until even more can be learned.

There are also things people can do in both affected and not-yet-affected areas. In those parts of the continent not currently affected, intense surveillance can provide estimates of pre-WNS population size and allow for better tracking and potential mitigation of WNS. One excellent example of this type of activity is the Appalachian bat count (http://www.pgc.state.pa.us/pgc/cwp/view.asp?a=458&Q=176676&PM=1).

People can also install bat boxes to provide alternate roosts for bats. Although this would not affect the fungus directly, installing bat boxes could provide fungus-free environments over the summer months and also reduce migratory distances between winter hibernacula and summer sites by providing suitable roosts, both of which could enhance the survival of as many bats as possible. Examples of boxes that work well in the eastern United States can be found by visiting the websites of the Pennsylvania State Game Commission (http://www.pgc.state.pa.us/pgc/lib/pgc/wildlife/woodcrafting/plan11.pdf) or Bat Conservation International (http://www.batcon.org). As the disease moves through new areas, locating resistant individuals and those few remaining summer and winter colonies will be of critical importance to the future recovery efforts of our night-flying friends. We hope and expect that some bats will survive, but even survivors will face tremendous challenges, because they can be expected to have limited fat reserves for migration and winter survival. Minimal winter disturbance will be critical for these bats to give them a fighting chance. Even so, with their low reproductive rates, it will be decades before bat populations in WNS-affected areas are restored. Finally, people can inform their state and federal representatives that significant governmental funding is desperately needed, as WNS is clearly an issue for all of North America—not just the Northeast. Lastly, several mechanisms for collecting personal donations to assist WNS-related research have been established (http://www.indstate.edu/ecology/centers/bat.htm, http://www.batcon.org/, or http://www.caves.org/WNS/).

Literature Cited

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

Gargas, A., M. T. Trest, M. Christensen, T. J. Volk, and D. S. Blehert. 2009. *Geomyces destructans* sp. nov. associated with bat white-nose syndrome. Mycotaxon, 108: 147–154.

Reeder, D. M., and G. R. [G. G.] Turner. 2008. Working together to combat white-nose syndrome: a report of a meeting on 9–11 June 2008, in Albany, New York. Bat Research News, 49:75–78.

Reichard, J. D., and T. H. Kunz. In press. White-nose syndrome inflicts lasting injuries to the wings of little brown bats (*Myotis lucifugus*). Acta Chiropterologica.