White-Nose Syndrome

Science Strategy Meeting
June 10, 2008 | Albany, NY

Synopsis

Energetics provided technical coordination and assistance:
The Science Strategy meeting was facilitated by Bonnie Ram and Wendy Wallace. Final layout and production was completed by Tenley Dalstrom, Susan Kaczmarek, Stephen Namie, and Tommy Finamore.

Cover Photo:
A cluster of little brown bats exhibiting the symptoms of White-Nose Syndrome.

Photo by Nancy Heaslip, Department of Environmental Conservation, NY State.
September 2008

Questions, Observations, Hypotheses, Predictions, and Research Needs for Addressing Effects of White-Nose Syndrome (WNS) in Hibernating Bats

October 2008

MEETING INFORMATION

A three-day emergency meeting on White-Nose Syndrome (WNS) was convened June 9 – 11, 2008, in Albany, New York. The meeting was organized by Bat Conservation International, Boston University's Center for Ecology and Conservation Biology, Cornell University College of Veterinary Medicine, the New York Department of Environmental Conservation, the U.S. Geological Survey, and the U.S. Fish and Wildlife Service. Bat Conservation International funded meeting facilitation and scientist travel with help from the following sponsors: the Disney Rapid Response Fund, the National Speleological Society, Valerie and Anton Schindler, the U.S. Army Corps of Engineers and the U.S. Geological Survey. The New York State Department of Environmental Conservation and the U.S. Fish and Wildlife Service also contributed logistically.

The goal of the meeting was to identify the most urgent and promising research directions for researchers and management agencies to help understand the cause (or causes) of this unprecedented threat to bats. More than 100 participants from two Canadian provinces and 20 U.S. state and federal agencies, eight universities and four non-government organizations participated and discussed the existing knowledge and pending questions about the syndrome.

The meeting was structured so that the first day was a plenary session that consisted of a scientific overview of the issue from a variety of disciplines including ecology, pathology, physiology, infectious diseases, and toxicology. On day two, managers and scientists met separately as each group identified what it considered the most pressing issues for solving the WNS threat. On day three, these two groups met jointly to plan the coordination and management of critical research efforts. The following synopsis is the result of the Science Strategy Session that was conducted on the second day of the meeting with over 30 leading scientists and analysts. This document is intended to be used as a template for evaluating and summarizing observations, as well as addressing key questions, hypotheses, and research needs as identified by the scientific experts in attendance.

BACKGROUND

White-Nose Syndrome was first recorded in photographs taken on February 16, 2006 in Howe Cave, New York; however, this was not reported until 2008. WNS was subsequently documented in nearby Schoharie Cavern in January 2007, and later that winter in three other caves in New York. All five sites were within a 15 km radius west of Albany, New York. By March 2008, bats exhibiting WNS were observed in hibernacula from at least four states (New York, Vermont, Massachusetts, and Connecticut). Early evidence obtained by state biologists indicated that several hundred thousand bats may have already died in this relatively small area.

If WNS continues to spread at current rate it has the potential to threaten entire species, including the already endangered Indiana and Virginia big-eared bats, and associated ecosystems.

Field observations have shown that bats affected by WNS are characterized by some or all of the following: 1) a white fungus that grows on the nose, ears, and wing membranes; 2) depleted white and brown fat reserves by mid-winter; 3) a reduced capacity to arouse from deep torpor; 4) an apparent lack of immune response during hibernation; 5) ulcerated, necrotic and scarred wing membranes; and 6) atypical behavior causing bats to emerge prematurely from hibernacula in mid-winter. Laboratory studies have isolated a previously undescribed psychrophilic fungus, closely related to *Geomyces spp*, from bats affected with WNS. This fungus grows on the skin (nose, ears, and wing membranes) of hibernating bats, and laboratory studies revealed it grows optimally at low temperatures characteristic of hibernacula. There is histological evidence that the fungus sometimes penetrates the dermis, especially in areas associated with sebaceous glands and hair follicles. Genetically identical isolates of this fungus have been collected directly from bats located in widely dispersed hibernacula in the northeastern United States. Preliminary data suggest that concentrations of chlorinated hydrocarbon contaminants, pyrethroids and heavy metals are not markedly elevated in bats examined thus far, nor have known bacterial or viral pathogens been identified. Narrowing the field of potential causative agents requires an understanding of whether the causative agent is pathogenic and if the fungus associated with WNS is, in fact, itself a pathogen. Also, both field and laboratory investigations will be required to assess several intrinsic and extrinsic factors that may contribute to this condition.

Facilitated Session of the Science Strategy Meeting.

The following tables outline questions, observations, hypotheses, predictions and research needs.

QUESTION 1. Is the newly isolated cold-loving fungus associated with WNS the primary cause of mortality in these bats?

Observations:

A previously undescribed cold-loving fungus has been isolated from bats in the affected hibernacula. Histological evidence indicates that this fungus sometimes penetrates the dermis of bats, especially in the wing membranes, and on the nose in areas associated with sebaceous glands and hair follicles. It is not known whether this fungus is causing the deaths of these bats or is a secondary effect of some other factor or factors.

Hypothesis	Predictions	Research Needs
• WNS is caused by a transmissible pathogenic fungus	• Unaffected bats inoculated with the WNS fungus will develop symptoms of WNS. • Healthy bats that hibernate in contact with affected bats will develop WNS (direct transmission). • Healthy bats, sharing the same environment with affected bats, but not in physical contact, will develop WNS.	• Paired laboratory comparisons of affected and unaffected bats that share the same hibernation chambers to document whether unaffected bats contract WNS. • Paired laboratory comparisons of affected versus unaffected bats that share the same hibernation chamber without the opportunity for direct physical contact to document whether airborne transmission is possible. • Paired laboratory comparisons of bats inoculated with live versus inactive forms of the fungus to determine the environmental conditions (e.g., temperature and humidity) under which WNS develops. • Continued monitoring of bats from affected and unaffected areas to establish a statistical association of a fungus with WNS.

QUESTION 2. Is the fungus associated with WNS a secondary manifestation of other underlying conditions?

Observations:

Fungal infections often develop secondary to conditions associated with other diseases, or immunosuppression. The fungus observed on hibernating bats may reflect another underlying condition due to other causes. Because the flora on the skin of bats has not been routinely cultured, no data are available on normal conditions. Additionally, many fungal species naturally grow in organically rich soils and on dead or moribund organic matter in dark, moist places, typically characteristic of locations occupied by hibernating bats in caves and mines. Many soil fungi are capable of degrading keratin, an important structural protein found in hair, skin and nails. Psychrophilic (cold adapted) fungi are adapted to cold conditions, and many isolates of the genus of fungus associated with WNS have been reported from cold climates. We are dealing with a newly described fungus in an unusual situation, so further research is needed to establish the significance of this fungal isolate. Additionally, studies are needed to investigate the underlying conditions that may predispose bats to fungal infection. Bats, compromised by other diseases are prone to the development of fungal infections on the skin of their wings nose and ears.

Hypothesis	Predictions	Research Needs
• The fungus associated with WNS grows on the skin surfaces of healthy bats. • This fungus is found outside of the WNS zone, but only becomes evident in areas affected by some unknown agent.	• The fungus associated with WNS grows only on necrotic skin. • WNS fungus will exhibit a keratinolytic enzymatic profile similar to human and animal dermatophytic pathogens. • A kertinolytic fungus could colonize bat skin and proliferate especially in superficial and sub-cutaneous tissues. • Proliferation of fungus is facilitated by minor abrasions to the skin that are subjected to vigorous grooming.	• Conduct laboratory studies to test whether the WNS fungus can be cultured on dead versus live tissue. • Use biochemical and genetic approaches to test for the presence of relevant fungal genes and proteins. • Determine the distribution of the fungus in hibernating colonies inside/outside of the WNS zone.

QUESTION 3. Why are fat reserves of bats with WNS depleted by mid-winter?

Observations:

Hibernating bats deposit fat reserves, including both white adipose tissue (WAT) and brown adipose tissue (BAT), in autumn that support them during the swarming and mating season and sustain them throughout winter hibernation. In the northeastern U.S., these fat reserves are mostly deposited during a one-month period beginning in early September and are gradually depleted during periodic arousals throughout the winter.

Hypothesis 1	Predictions	Research Needs
• Bats arriving at hibernacula have insufficient fat reserves (WAT and/or BAT) to survive until spring.	• Bats affected by WNS arrive at hibernacula with less fat than unaffected bats. • Fat deposits have insufficient quantities of polyunsaturated fatty acids to promote normal hibernation. • A pathogen is disrupting fat accumulation. • A contaminant (e.g., pesticide used in agriculture, industrial contaminant, or heavy metal) is disrupting fat accumulation. • Environmental factors (e.g., temperature, rainfall, contaminants, etc.) are reducing abundance of critical insect resources resulting in suboptimal fat accumulation.	• Compare pre-hibernation fat deposition in bats affected versus unaffected by WNS. • Conduct year-round paired comparisons of bats from affected versus unaffected sites to document WNS-associated changes in body condition, and their physiological and immunological status. • Assess relationships between successful hibernation and the quality of fat (e.g. amount of polyunsaturated fatty acids present in WAT). • Assess relationships between the quantity of fat deposited during the pre-hibernation period and environmental variables. • Assess the relationship between autumn fat deposition and selected environmental contaminants present in diet during the summer and during the pre-hibernation period, collecting dietary samples and tissues/guano of bats affected versus unaffected by WNS. • Compare diets of bats at affected versus unaffected sites during pre-hibernation fattening period. • Conduct paired comparisons of body composition and quality (presence of unsaturated fatty acids) of aerial insects available in bat feeding areas.

Hypothesis 2	Predictions	Research Needs
• Bats with sufficient fat upon arrival at hibernacula suffer premature depletion of white adipose tissue (WAT) and/or brown adipose tissue (BAT) during hibernation.	• Bats affected by WNS arrive at hibernacula with fat reserves similar to unaffected bats, but deplete them prematurely during winter. • Increased metabolism during winter leads to early depletion of fat. • Increased arousal frequency during winter leads to early depletion of fat. • Increased arousal duration during winter leads to early depletion of fat. • A pathogen causes increased metabolic rate (e.g. from excessive non-shivering and shivering thermogenesis) and/or changes in arousal patterns. • A non-pathogenic fungus causes increased metabolic rate and/or changes in arousal patterns. • An anthropogenic contaminant or heavy metal causes increased metabolic rate and/or changes in arousal patterns. • Environmental conditions that bats experience during hibernation (e.g., increased temperature), cause higher metabolic rate and/or changes in arousal patterns. • Suboptimum quantities of polyunsaturated fatty acids in diet cause increased arousal frequencies, leading to premature fat depletion. • Suboptimum quantities of polyunsaturated fatty acids in diet reduce depth of torpor, causing premature fat depletion.	• Compare behavior, metabolic rates, arousal frequency, and arousal duration in hibernating bats affected versus unaffected by WNS under controlled laboratory conditions. • Compare behavior, metabolic rates, arousal frequency, and arousal duration in hibernating bats affected versus unaffected by WNS under natural conditions. • Determine the quantity of polyunsaturated fatty acids in WAT at the beginning, middle, and end of hibernation in bats at colonies affected and unaffected by WNS. • Determine the quantity of polyunsaturated fatty acids in the diets of bats from affected and unaffected sites during the pre-hibernation period.

QUESTION 4. Are pathogens a direct cause of mortality?

Observations:

Pathogens are known to cause direct mortality in bats, by either altering (compromising or challenging) the immune and/or nervous systems. A number of fungi, including human pathogens, have been isolated from bats, but there was no evidence of serious fungal infections in culture-positive animals.

Hypothesis 1	Predictions	Research Needs
• Pathogens are a direct cause of mortality. A consistent pathogen causes symptoms of WNS when introduced into healthy bats from unaffected areas.	• Isolates from WNS-affected bats can be used to transmit the affliction to other individuals from unaffected sites. • Healthy bats from unaffected sites, when exposed to WNS-afflicted bats, succumb to similar symptoms.	• Under controlled laboratory conditions, inoculate (or otherwise expose) bats from areas unaffected by WNS with affected bats (or isolates) to determine whether bats become symptomatic.

QUESTION 5. Are contaminants a direct cause of mortality?

Observations:

Environmental contaminants are known to cause direct mortality in bats, by either altering (compromising or challenging) the immune and/or nervous systems. A number of contaminants, including pesticides have been isolated from bats, but to date no contaminants have been consistently identified in preliminary necropsies of bats affected by WNS. The pattern of the expansion of WNS, as well as the variability in ecological zones, watersheds and land use do not suggest that a contaminant is involved.

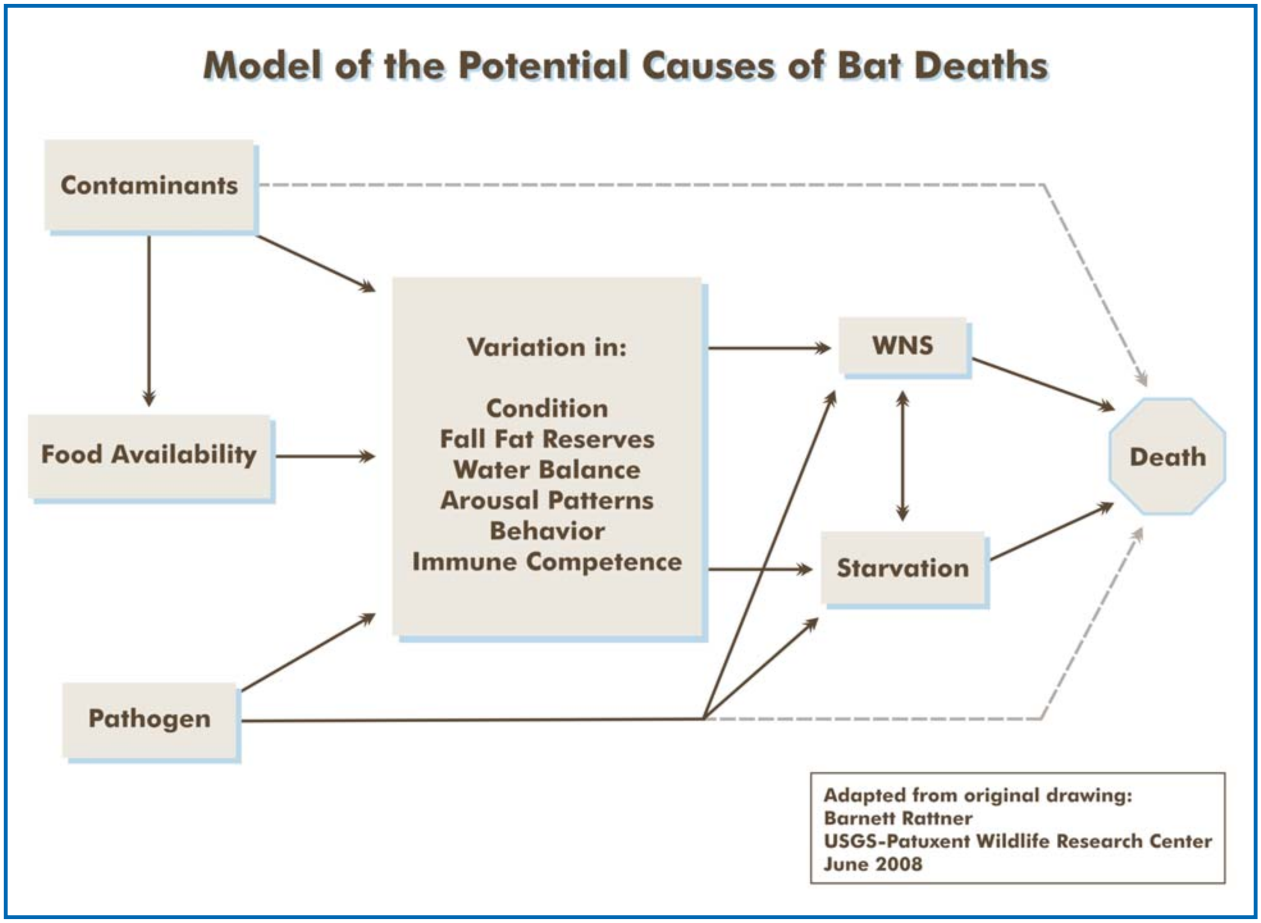
Hypothesis 1	Predictions	Research Needs
• A single contaminant or combination of contaminants evokes WNS symptoms when introduced into bats of normal health status from unaffected areas.	• Introduction of a single contaminant or combination of contaminants at levels likely to occur across large geographic regions can cause bats from unaffected areas to die of WNS symptoms under controlled laboratory conditions. • Bats exhibiting WNS symptoms will exhibit elevated levels of this contaminant, a metabolite or combination in selected tissues.	• Compare the use of pesticides and other chemicals of concern within the region affected by WNS with unaffected regions. • Evaluate the effects of emerging contaminants (e.g., neonicotinoides) on bat physiology using controlled laboratory feeding studies. • Conduct laboratory comparisons of bats with elevated versus normal levels of contaminants, both with and without exposure to WNS-infected individuals.

QUESTION 6. How does WNS affect bat maternity colonies?

Observations:

Bats that hibernate in caves and mines in the northeastern U.S. disperse widely in spring to roost in buildings, tree cavities, and beneath exfoliating bark, where they form maternity colonies up to 200 miles or more from hibernacula. These maternity colonies are largely composed of adult females and their yearling female offspring that may have over-wintered in several different hibernacula. Adult males typically do not roost with females during the maternity period, but instead roost alone or in small groups dispersed widely.

Hypothesis	Predictions	Research Needs
• WNS can cause mortality and/or reproductive failure in bats that survive the winter season and form maternity colonies.	• • Maternity colonies experience reduced reproductive success in WNS-affected versus unaffected areas. • Bats at maternity colonies affected by WNS have compromised immune systems. • Wing damage is significantly more prevalent in WNS-affected versus unaffected areas and is associated with low body mass. • For females to successfully reproduce they must have adequate WAT reserves at the end of hibernation. WAT is the source of the hormone leptin, which in turn is necessary to initiate the cascade of hormones that leads to ovulation.	• Quantify body condition of bats at selected summer roosts, especially those housing maternity colonies. • Quantify the condition of wing membranes of bats as they depart from hibernacula versus during the non-hibernating period at maternity roosts or during autumn return to swarming sites and hibernacula. • Conduct controlled laboratory experiments to assess the foraging success of bats with extensive wing damage associated with WNS. • Conduct controlled laboratory experiments to determine to what extent WNS-affected bats can recover and rear young when provided ideal diet and other conditions. • Quantify wing damage and body condition indices of bats with most serious wing injuries, comparing frequencies during spring emergence versus during return in autumn as evidence of failure to survive during summer. • Assess the competency of immune system responses of bats at summer roosts in WNS areas vs. areas not yet affected by WNS. • Assess leptin levels (a hormone indicative of impending reproduction) of females affected versus unaffected by WNS upon departure from hibernacula. • Assess the reproductive success of females from affected versus unaffected colonies during the maternity period.



A hibernating little brown bat (*Myotis lucifugus*),
showing a white fungus growing on its nose, ears and forearm, a
condition symptomatic of White-Nose Syndrome
(Photo by Al Hicks).

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