

1 Title: Fungal Diseases in Wildlife: Emerging threats from Pathogenic Fungi

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5

6 **Abstract**

7 Since 1970 there has been a steady rise in infectious disease cases in wildlife and emerging
8 fungal pathogens, even when controlling for reporting bias. Two of the more visible emerging
9 fungal diseases-- chytridiomycosis in amphibians and white-nose syndrome in bats-- have caused
10 unprecedented die-offs of wildlife populations around the globe, threatening biodiversity and
11 ecosystem services. Here we clarify factors associated with fatality from fungal disease in order
12 to shed light on the dynamics of these pathogens and their wildlife hosts and discuss how these
13 factors can be used to advise and prioritize conservation approaches.

14

15 **In a Nutshell**

- 16 • White-nose syndrome and chytridiomycosis are only two of an emerging class of fungal
17 pathogens that may pose important threats to wildlife species
- 18 • Understanding of the transmission of fungal diseases and identifying general factors that
19 influence host mortality including natural immunosuppression, body temperature, water
20 limitation, and behavior, can advise conservation efforts that tend to focus on either
21 limiting transmission or reducing host mortality

- 22 • Identifying these factors will assist with assessing ongoing conservation efforts and with
23 predicting which species may be more vulnerable to population declines as a result of
24 fungal disease in the future

25

26

27 **Introduction**

28

29 Disease has been recognized to play an important role in the ecology of wildlife
30 populations (e.g., Prins & Weyerhaeuser 1987), but until recent times, has thought pose a more
31 limited extinction risk to most taxa than other threats such as habitat change, overexploitation, or
32 invasive species (Baillie et al. 2004). In particular, the potential threat to wildlife populations of
33 fungal pathogens – which are usually unable to cause disease in otherwise healthy individuals
34 (Laakkonen 1999; Casadevall 2005; Fisher *et al.* 2012) – has until recently been largely ignored
35 by conservation scientists and practitioners.

36 Basic theoretical models of host-pathogen dynamics have suggested disease is a limited
37 threat to long-term host persistence because local disease populations should go extinct prior to
38 local host populations, enabling host recovery (e.g., Anderson & May 1992). Newer models,
39 however, have identified several conditions under which diseases can drive host and even non-
40 host populations to extinction (e.g., de Castro & Bolker 2005). Meanwhile, emerging infectious
41 diseases – diseases that are newly recognized, newly affecting a population, or rapidly increasing
42 in incidence or geographic range (Daszak et al. 2000) – have been appearing at an increasing rate
43 since 1940, even when controlling for reporting bias (Jones *et al.* 2008).

44 White-nose syndrome (WNS) in bat populations (Box 1) and chytridiomycosis (chytrid)
45 in amphibians (Box 2) have caused mass mortality of wildlife host populations in recent years,
46 threatening many species with regional extinction (Briggs et al. 2010, Frick et al. 2010,
47 Thogmartin et al. 2013). Yet, these diseases represent only two of a large suite of emerging
48 fungal diseases (Fisher et al. 2012) that present major threats to local and regional biodiversity,
49 ecosystem function, and key ecosystem services (Berger *et al.* 1998; Daszak *et al.* 2001; Blehert
50 *et al.* 2009; Frick *et al.* 2010).

51 Threats of fungal pathogens to wildlife populations are clearly much greater than
52 previously thought and it is therefore important to identify viable management strategies to
53 mitigate their threat. Our aim in this paper is to discuss potential management strategies to
54 control the transmission of current fungal diseases and identify patterns in host mortality that can
55 be helpful in identifying vulnerable populations for future outbreaks. Using WNS and chytrid as
56 our primary examples, we hope to discuss the viability of proposed management strategies
57 through a discussing the life history of both pathogen and host.

58

59 **Transmission and Emergence of Fungal Pathogens**

60

61 Epidemiological models demonstrate how increased heterogeneity in modes of
62 transmission increase the ability of a disease to spread and invade new populations (Robertson,
63 Eisenberg *et al.* 2013). Fungal diseases, have a large multitude of transmission pathways due to
64 their possession of a resting stage in their development and their generalist nature. By analyzing
65 the variety of modes of transmission in fungal pathogens we can better understand their
66 pathogenicity and ability to spread globally.

67 The fungus responsible for WNS, *Pseudogymnoascus destructans* (Pd), (Minnis and
68 Lindner 2013), formerly known as *Geomyces destructans* , has been discovered in soil samples
69 taken from hibernacula as well as growing on cave-walls. This means that although the most
70 common mode of transmission seems to be through direct contact with an infected individual, Pd
71 could potentially be transmitted through these passive environmental vectors as well as through
72 animal or human vectors (Lindner, Gargas et al. 2011; Puechmaille, Wibbelt et al. 2011).
73 Chytridiomycosis is transmitted through an aquatic zoospore that, like Pd, is most commonly
74 passed from individual to individual. However, this zoospore is also thought to possess a resting
75 stage, allowing it to be transmitted through environmental or animal vectors (Berger et al. 2005).
76 In other soil dwelling fungal pathogens, including Histoplasmosis and Blastomycosis, burrowing
77 and dust-bathing can also cause spores to be released into the air where they can be inhaled and
78 cause respiratory infection (Perez-Torres, Rosas-Rosas et al. 2009; Werner and Norton 2011).

79 Understanding the various transmission pathways of emerging fungal pathogens is an
80 important step to preventing future disease emergence and for controlling the spread of existing
81 pathogens. It is important to investigate various methods used to mitigate disease spread in order
82 to focus conservation efforts towards transmission pathways that would be the most efficient and
83 cost-effect methods of control.

84

85 ***Human-mediated Trade and Transportation***

86

87 The human-mediated introduction of a novel disease to a naiive population, termed
88 “pathogen pollution,” can occur through a variety of different mechanisms including the
89 international trade of agricultural materials, biological waste, domesticated animals, and

90 encroachment on wildlife populations (Daszak et al 2010). While it is impossible to measure the
91 degree to which human migration and trade may have contributed to disease emergence in
92 wildlife, it is thought to have played a major role in the migration of several disease outbreaks
93 including West-Nile Virus and Mad Cow Disease (Daszak et al 2010). For fungal pathogens,
94 human-mediated dispersal is thought to be a leading initiator of new outbreaks globally (Fisher et
95 al. 2012). For this reason, it is important to recognize the role of human-mediated pathogen
96 movement in the emergence of fungal pathogens and to investigate methods to control its
97 influence.

98 Pd has spread rapidly throughout the eastern, southern, and Midwestern United States and
99 eastern Canada (Fig. 3), threatening the area with a local extinction of species whose populations
100 were previously stable or increasing (Frick *et al.* 2010). While this fungus is also widespread
101 among many European bat species, these species are not experiencing the same declines reported
102 in North America (Puechmaille, Frick *et al.* 2011). Some scientists hypothesize that this pattern
103 suggests that European bats coevolved with the fungus, while North American bats have only
104 recently been introduced to it (Puechmaille, Wibbelt et al. 2011) and suffer more severe
105 consequences as a result of the introduction of the pathogen to a naive population (Puechmaille,
106 Frick et al. 2011). Because no bat species migrate between Europe and America, it is likely that
107 the introduction of the pathogen to North America was human-mediated (Wibbelt et al 2010).

108 The trade of amphibians for pets, research, food, or even for conservation, expands across
109 almost all continents and involves many species that could be potential carriers for Bd (Fisher
110 and Garner 2007). These traded populations often come into contact with wild amphibian
111 populations, possibly bringing pathogens with them and leading to increased range of Bd
112 infection (Fisher and Garner 2007). Additionally, because these trade practices and mass

113 transport of amphibians existed before the emergence of Bd, it is hypothesized that it has played
114 a major role in its establishment as an international pandemic (Fisher and Garner 2007). Chytrid
115 was thought to have existed as a stable endemic infection for years in Africa before cases were
116 reported in other parts of the world (Weldon, du Preez et al. 2004), suggesting that a lack of
117 regulations in international trade of amphibians has resulted in the pathogen infecting naïve
118 populations (Fisher, Garner *et al.* 2009).

119 The pattern of human mobility on a global scale has an indisputable influence over the
120 exposure of wildlife to novel pathogens. Scientists have begun to create models that use human
121 transportation networks and trade routes to discover possible origins of non-native organisms
122 that may have been introduced through human-mediated dispersal (Yemshanov, Koch *et al.*
123 2013). Such models may be useful in predicting high risk areas for future fungal disease
124 outbreaks by integrating human-mediated movement of potential hosts and carriers of infectious
125 zoospores with information on the natural history of the disease and its requirements for
126 establishment in a given area (Fisher and Garner 2007). However, these models are limited by
127 lack of data and precision in predicting patterns in human mobility (Yemshanov, Koch *et al.*
128 2013).

129 Legal means have also been used to control the human-mediated transmission of both
130 WNS and chytrid. These include regulations and formal guidelines limiting human access to
131 cave habitats and requiring cavers to decontaminate their caving gear between visits to cave
132 environments to control WNS (Zimmerman 2009; 2011; Hallam and Federico 2012) and an
133 increase in amphibian trade laws and screenings of amphibians transported internationally to
134 control human-mediated spread of chytrid (Kriger and Hero 2009). Although these laws,
135 regulations and guidelines have the potential to reduce human-mediated disease transmission

136 they may be costly and controversial. Therefore it is necessary to identify the more vulnerable
137 areas or populations to make these policies more efficient and cost-effective, and to instill
138 education and oversight that will ensure maximum compliance.

139

140 *Animal to Animal Transmission*

141

142 While human-mediated transportation plays a major role contributing to the movement of
143 disease around the world, the most common mode of more localized disease spread is through
144 direct contact between affected individuals. This can occur through social behaviors, mating,
145 roosting behaviors, and can be influenced by migration. Unfortunately, this type of transmission
146 is often difficult to control as it involves natural life processes that cannot be disrupted and
147 therefore, can only be managed through treating infected individuals or reducing their numbers.

148 A common method to reduce animal to animal transmission rates in past wildlife diseases
149 is through manipulating infected host population through culling. Hallam et al. (2011)
150 determined that culling would not be an effective strategy for reducing the spread of WNS due to
151 the fact that they can persist in the environmental without a host and due to the movement of bats
152 between hibernacula and variety of social settings during which transmission could occur. In the
153 case of chytrid, however, Woodhams et al. (2011) actually suggests that culling may be
154 beneficial in reducing transmission rates, although it has yet to be proven in the field. For this
155 reason, while culling could potentially be a viable management strategy it should be approached
156 with caution when used to control emerging fungal diseases.

157 Another method of controlling direct contact transmission involves treating infected
158 individuals directly to prevent these individuals from furthering disease spread. Such methods

159 include “capture and treat” techniques that involve the use of antifungal drugs, probiotics,
160 antibiotics, or salt treatments (Fig. 7) (Woodhams and Alford 2005; Woodhams *et al.* 2007;
161 Kriger and Hero 2009). Other management strategies for chytridiomycosis include a vaccination
162 program and the introduction of natural predators of the Bd fungus (Woodhams, Bosch et al.
163 2011).

164 The use of antifungal drugs to treat white-nose syndrome has not been shown to be
165 effective in treating the disease (Robbins 2012), however, research is ongoing and certain strains
166 of biocontrol fungi (Zhang et al. Abstract) or bacteria (Cornelison CT Abstract) have been
167 identified that may be beneficial in impeding Pd growth and may be fungicidal in the field.
168 Additionally, a vaccination plan has been suggested as a potential strategy for increasing host
169 immunity against Pd (Osorio 2012). While these treatment techniques have shown to reduce
170 growth of the fungus in the lab, it is unclear whether they could be a viable large-scale
171 management strategy for reducing animal to animal transmission.

172 Placing individuals at temperatures above the thermal optimum of the fungi has proven to
173 reduce or eliminate infection in both cases of bats and amphibians (Johnson 2003 ; Geiger,
174 Kuepfer et al. 2011). For this reason, some researchers have proposed supplying heated refuges
175 for animals in their natural environment where they can clear infection (Johnson 2003 ;
176 Zimmerman 2009; Woodhams *et al.* 2011). However, this method requires that animals are able
177 to locate and travel to these thermal refuges, which may not always be possible for infected
178 animals in a natural setting.

179 For all of these methods, it is necessary to further investigate how these strategies could
180 have unwanted effects on the surrounding environment. Introducing new chemicals or bacteria
181 may affect the microbial community that could have significant trophic effects. Individual

182 treatment strategies also may be costly as they require sufficient supplies and monitoring in order
183 to ensure success.

184

185

186 *Animal-Environment- Animal Transmission*

187

188 It has been stated previously that fungal pathogens possess a resting stage that can allow
189 them to remain dormant in their environment even in the absence of a host. There are several
190 environmental factors that can influence the ability of this phase to persist which are necessary to
191 identify in order to fully manage further transmission.

192 The growth and persistence of fungal pathogens are highly influenced by temperature and
193 humidity. WNS mortality has been shown to be more closely associated with hibernation sites
194 that are “warmer and wetter” (Hayman DTS et al abstract) while incidence of chytridiomycosis
195 seems to peak during the “cool, dry season” (Woodhams and Alford 2005). Clearly these
196 patterns vary for different diseases, however, realizing these preferred growth conditions can be
197 useful in identifying environments which may be most likely for persistence of the pathogen.

198 In addition, seasonality can influence all of these factors. The prevalence of WNS shows
199 an increase as winter progresses which can be attributed to hibernation energetics and cool
200 winter temperatures that promote Pd growth (Puechmaille, Wibbelt et al. 2011). Symptoms of
201 WNS such as inflammation and skin lesions tend to occur during the spring (Bouma, Carey et al.
202 2010). Flory et al (2012) further suggests that bats that roost in more seasonally-variable habitats
203 are more likely to suffer from WNS mortality than those who live in habitats with a steadier

204 yearly climate. Incidence of chytridiomycosis was also observed to follow a seasonal pattern
205 with most frog deaths occurring during the winter (Berger, Speare et al. 2004).

206 The choice of breeding location has been shown to alter the risk of infection for both bats
207 and amphibian. Kriger and Hero (2007) suggest that amphibians breeding near permanent bodies
208 of water are at higher risk of developing chytridiomycosis while terrestrial species of amphibians
209 rarely develop the disease at all. WNS only affects cave-dwelling bat species and roosting
210 location therefore plays a large role in infection risk of this disease as well (Dzal, McGuire et al.
211 2011).

212 Identifying high risk areas is important for focusing management while altering
213 microhabitats areas may also be a viable strategy for mitigating disease transmission. An
214 example of this involves building artificial caves and hibernacula as a possible means of
215 controlling WNS (Gorman 2012). These caves could provide a controlled environment that could
216 be disinfected following each hibernation cycle, eliminating the potential for passive
217 transmission through cave walls (Gorman 2012). Such artificial roosts include bat boxes, which
218 have documented success (Gallo M et al. abstract), or abandoned buildings like military bunkers
219 (Aguis S et al. abstract).

220 Unlike bat hibernacula, most animal habitats do not consist of confined spaces that can be
221 sanitized. For this reason it is difficult to completely remove a fungus from the surrounding
222 environment without introducing large amounts of potentially harmful chemical treatments or
223 fungal predators. Therefore the best way to control animal to environment to animal transmission
224 may be to focus on treating individuals that live in areas that may be most suitable for fungal
225 growth, something that is further is discussed in the following sections.

226

227 **Recognizing Vulnerable Populations**

228

229 When creating a management plan for emerging fungal diseases it is important to make
230 sure to focus conservation on the most vulnerable populations in order to ensure maximum
231 efficiency of resource use. Additionally, understanding which populations may be more at risk
232 for future outbreaks could help predict fungal disease emergence by focusing monitoring of more
233 vulnerable species and habitats. Therefore we have identified several factors that influence the
234 vulnerability of populations to emerging fungal diseases through recognizing patterns in current
235 emerging diseases.

236

237 *Natural States of Immunosuppression*

238

239 Fungal diseases are considered to be “opportunistic” pathogens, usually requiring some
240 type of previous injury or disease in order to cause infection in their host (Laakkonen 1999;
241 Casadevall 2005). *Basidiobolus ranarum*, a fungal disease that also affects amphibian
242 populations has been shown in the lab by Taylor et al. to only cause infection when the skin of
243 the amphibian was abraded, suggesting that the fungus could only affect injured hosts (Taylor,
244 Williams et al. 1999). However, both WNS and chytrid have been shown to cause disease in
245 otherwise healthy individuals (Fisher et al. 2012) and it is therefore important to understand the
246 mechanisms by which these infections occur in order to identify populations that may be more
247 vulnerable to mortality from fungal pathogens.

248

249 During their annual winter hibernation, many North American bat species enter
250 prolonged bouts of torpor during which their metabolism slows down, and their respiration, heart
rate, and body temperature decrease in order to conserve as much energy as possible.

251 Hibernating bats also suppress immune function to further contribute to this energy conservation
252 (Bouma *et al.* 2010). This immunosuppressed state typically does not lead to disease emergence
253 because microbes are rarely able to survive at low winter temperatures. Other hibernating
254 animals may also be at an increased risk from fungal pathogens, due to their repressed immune
255 function and limited ability to acquire energy during this time.

256 Much like bats, amphibians also enter a natural immunocompromised state during
257 metamorphosis. The transition from an aquatic larval lifestyle to an adult terrestrial one exposes
258 the amphibian to a new set of pathogens. To compensate, amphibians must completely dismantle
259 their previous larval immune system and reconstruct a new immune system capable of protecting
260 it in a terrestrial environment and suited to a new adult body and lifestyle (Rollins-Smith 1998).
261 This post-metamorphic stage is considered to be the most vulnerable life-history stage for
262 amphibians (Rollins-Smith 1998), if infection does not occur at metamorphosis, the likelihood of
263 disease causing death and the ability of the pathogen to persist in the population is greatly
264 reduced (Briggs *et al.* 2005).

265 Temporary states of suppressed immune function are a vital part of the life history of
266 both bats and amphibians as a means of dealing with energy limitation. Though necessary for
267 development and survival, metamorphosis and hibernation may decrease the animal's ability to
268 fight off infection and cause them to use excess energy, thereby increasing mortality.
269 Recognizing that natural immunosuppressed states exist in wildlife populations and
270 concentrating management strategies around the times, may allow us to recognize populations
271 that may be more at risk of mortality from emerging fungal diseases.

272

273 *Body Temperature*

274

275 The majority of fungal species grow best at “ambient temperatures” (25-35°C)
276 (Casadevall 2012). Many warm-blooded mammals either have a body temperature above this
277 range or are able to raise their body temperature through a fever response to above the growth
278 range of the fungus (Casadevall 2005; Casadevall 2012). For this reason, cold-blooded animals
279 are usually more susceptible to fungal infection while warm-blooded or “endothermic” species
280 are thought to be highly resistant (Casadevall 2005; Casadevall 2012). While chytridiomycosis
281 has the advantage of affecting cold-blooded hosts (Fig. 6B), WNS affects mammals and
282 therefore does not seem to follow this trend. An explanation for the success of Pd on a
283 mammalian host can be explained by the hibernation energetics of bat species.

284 When bats experience a suppressed metabolism during hibernation, they also experience
285 a drop in body temperature. The body temperature of hibernating bats dropped to just above the
286 temperature of their hibernacula and overlaps substantially the growth range of Pd (Fig. 6A)
287 (Piotrowski *et al.* 2004; Blehert *et al.* 2009; Jonasson and Willis 2012). WNS only affects cave-
288 dwelling bats, as it is necessary for their body temperatures to reach these low levels in order for
289 infection to occur (Dzal *et al.* 2011). Therefore both cold-blooded animals and warm-blooded
290 species with naturally lower or more variable body temperatures, usually during hibernation,
291 may be at a higher risk of developing an infection from fungal pathogens. Although it is an
292 anomaly, WNS is not the only fungal pathogen that can affect mammals. Another disease,
293 *Paracoccidioides*, takes advantage of the naturally lower body temperature of their host,
294 armadillos, to invade the tissue around the cooler areas of their body and cause disease (Bagagli,
295 Sano *et al.* 1998).

296 Despite variability in the optimal growth ranges of fungal pathogens, evidence clearly
297 indicates that cold-blooded animals, as well as those that have naturally occurring lower or more
298 variable body temperatures, are at higher risk of developing a fungal pathogen and are less able
299 to fight off an infection than those with the ability to maintain their body temperature above the
300 growth range of the fungus.

301

302 *Sociality and behavioral factors*

303

304 Various behaviors in wildlife host species may alter their likelihood of fatality. Bat social
305 behaviors affect WNS transmission during roosting and the selection of hibernacula. Certain bat
306 species will roost in “highly gregarious” and “tightly packed” aggregations which have the
307 benefit of reducing the exposed skin surfaces of the bats, thereby reducing cutaneous evaporative
308 water loss (Langwig *et al.* 2012). Additionally, during the summer and early autumn large
309 numbers of individuals from various hibernating bat species exhibit a swarming behavior in
310 order to mate (Puechmaille *et al.* 2011; Langwig *et al.* 2012). These social behaviors create
311 opportunities for bat-to-bat transmission of fungal pathogen (Hallam and McCracken 2011). For
312 this reason more social organisms or those that live in more crowded populations may be more
313 vulnerable to high transmission rates.

314 Additional host behaviors may also alter vulnerability although they are highly pathogen-
315 dependent. Because many fungal diseases, including WNS, Blastomycosis, and Histoplasmosis
316 can exist outside of their host in the soil, animals that burrow, dust-bathe or live in close
317 proximity to the soil may be at higher risk of developing infection (Bagagli *et al.* 1998;
318 Laakkonen 1999). When it comes to chytridiomycosis, excessive skin shedding may also be

319 another adaptive mechanism used to reduce infection on the host, however, it may also be a
320 manipulation of the host by the fungus in order to cause them to increase keratinized substrate
321 (Woodhams, Ardipradja et al. 2007). It is important to understand the life history of these
322 diseases in order to determine how behavioral adaptations may make a certain individual or
323 population more vulnerable to infection.

324

325 *Unaffected Species and Potential Animal Vectors*

326

327 When treating infected individuals, it is important to keep in mind that there may be other
328 animal hosts that do not suffer mortality and may persist as vectors. Several species that coexist
329 with the pathogen may serve as reservoir hosts for infection. In both WNS and chytrid, some
330 species or individuals are able to survive with infection and mortality has been shown to be
331 highly species dependent (Rollins-Smith 1998; Blaustein, Romansic et al. 2005; Davidson et al
332 2003).

333 Pd has been isolated from three bat species without showing any damage in the United
334 States (Turner 2011). Some species who have been exhibiting lower declines and therefore may
335 be more resistant to WNS in the United States include big brown bats (*Eptesicus fuscus*), the
336 small footed bats (*Myotis leibii*) (Turner, Reeder et al. 2011; Langwig, Frick et al. 2012) and the
337 Indiana bat (*Myotis sodalists*) (Amelon S.K. 2012). The chytrid fungus has also been discovered to
338 exist on or among several species of amphibians without causing the same symptoms or degree
339 of fatality. Searle et al (2011) found variations in mortality of 6 different species of amphibians
340 who were all exposed to Bd. Interestingly this study showed little difference in infection, only in
341 mortality. Examples include the American bullfrog (*Lithobates catesbeianus*) which is able to

342 persist with a high level of infection from Bd without any signs of developing disease symptoms
343 (Blaustein, Gervasi et al. 2012) and the Boreal Toad (*Bufo boreas*) which thrives at a low
344 infection level with a slow population decline (Pilliod, Muths et al. 2010). Woodhams et al
345 (2007) found that even when environmental conditions and the pathogen strain are held constant,
346 the outcome of infection among species still varied. One explanation for this is differences in the
347 presence of antimicrobial skin peptides among species of amphibians, however shedding periods
348 and molting may also play a role (Woodhams, Ardipradja et al. 2007; Gammill, Fites et al.
349 2012).

350 Because of differential host mortality, maintaining biodiversity in areas where disease in
351 present may be protective against direct transmission in a similar way that herd immunity can
352 reduce disease transmission as a result of vaccination programs. Various conservation methods
353 aimed at maintain biodiversity therefore can also have benefits towards disease control. One
354 method currently being employed to reduce the spread of chytrid, involves maintaining a group
355 of select amphibians in captivity so that these populations can be reintroduced into the wild when
356 the disease has run its course (www.amphibianark.org). While this project could play a major
357 role in maintaining amphibian biodiversity, it may not be a viable strategy for other animal
358 populations that are more expensive and difficult to keep in captivity. Additionally, in order for
359 such programs to be effective it is important to recognize which species may be more susceptible
360 to infection.

361

362 **Conclusions**

363

364 The ability of emerging fungal pathogens to adapt and invade previously untouched
365 ecosystems has recently caught the attention of conservation scientists and practitioners around
366 the globe. Emerging fungal pathogens have shown the potential to cause major die-offs and local
367 extinctions of their wildlife host populations, especially in the cases of WNS and chytrid. A
368 reduction in the abundance and change in distribution of these species could potentially affect
369 ecosystem function and global public health. By achieving a thorough understanding of the
370 epidemiology and life history of emerging fungal pathogens, we may be able to discover
371 conservation strategies that would be most efficient in delaying the progression of these diseases.

372 Further research in population dynamics and epidemiology of current fungal pathogens is
373 necessary in order to find more effective and efficient methods to manage their impacts and to
374 prevent future disease emergence. Information on factors that affect host mortality and disease
375 transmission are crucial to evaluating possible conservation strategies and with assessing with
376 future response strategies when dealing with infectious disease in the field.

377

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535

537 **Box 1: White-Nose Syndrome in bats**

538 White-nose syndrome (WNS), a disease affected hibernating bats, is caused by the
539 pathogenic fungus *Geomyces destructans* (Pd) (Lorch *et al.* 2011), which was first documented
540 outside of Albany, New York in 2006 (Blehert *et al.* 2009). WNS is characterized by lesions
541 affecting the snouts, ears, and especially the wings of infected individuals (Cryan *et al.* 2010;
542 Lorch *et al.* 2011). Pd is believed to cause abnormal arousals from hibernation, which can lead to
543 water and energy loss and eventually death (Reeder *et al.* 2012). White-nose syndrome is
544 expected to result in multiple regional extinctions of bat species in the North Eastern United
545 States (Frick *et al.* 2010, Thogmartin *et al.* 2013).

546

547 **Box 2: Chytridiomycosis in amphibians**

548 Amphibians have also suffered greatly from the recent emergence of a fungal disease,
549 Chytridiomycosis, or chytrid. Chytrid is caused by the fungus *Batrachochytrium dendrobatidis*
550 (*Bd*) which was first identified in 1997 and named in 1999 (Fisher *et al.* 2009). The chytrid
551 fungus (*Bd*) also affects the skin of its host causing a physical disruption of the epidermis
552 (Rosenblum *et al.* 2010) and is thought to cause death through a disruption in electrolyte
553 transport and osmoregulatory function (Daszak *et al.* 2001; Voyles *et al.* 2007; Voyles *et al.*
554 2009; Rosenblum *et al.* 2010). Chytrid has been called the “worst infectious disease ever
555 recorded among vertebrates in terms of the number of species impacted, and its propensity to
556 drive them to extinction” (Briggs *et al.* 2010).

557

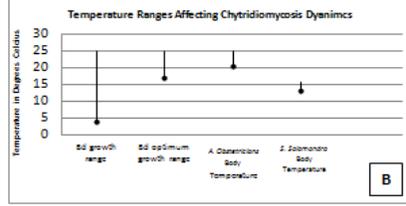
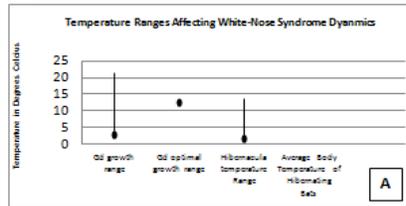
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Figure 7: Distribution of White-nose Syndrome in Northeastern United States



Photograph of a blue poison dart frog (*Dendrobates azureus*) receiving treatment for chytridiomycosis with an antifungal soak. Taken at the National Amphibian Conservation Center at the Detroit Zoo, Detroit, Michigan. July 2012.

Figure 6: Temperature Dynamics of White-Nose Syndrome and Chytridiomycosis



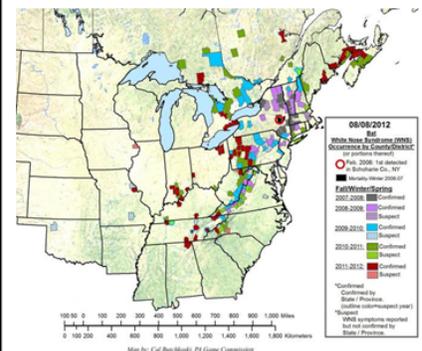
Graph A is a representation of the temperature ranges affecting the dynamics of the Gd fungus which causes white-nose syndrome. The areas where the bars overlap are the temperature ranges at which infection can occur (Verant, Boyles et al. 2012). Graph B shows a similar representation for the temperature ranges that affect the dynamics of the chytrid fungus, Bd. The graph includes the growth and optimal growth range (Blaustein, Gervasi et al. 2012) as well as two examples of amphibians that have suffered fatalities as the result of chytridiomycosis: the common midwife toad (*A. Obsterionia*) (Busack 1978) and the fire salamander (*S. J. Salamandra*) (Busack 1978).

Figure 4: Global Distribution of Chytridiomycosis 2006-2011



Created with data from bd-maps.net search year=2006, 2007, 2008, 2009, 2010 and spatialepidemiology.net. http://www.spatialepidemiology.net/user_maps/php/temp/11-29-12-22340.html

Figure 3: Distribution of White-nose Syndrome in Northeastern United States



Map from <http://www.whitenosesyndrome.org/resources/map>, created 8/8/12.