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Climate Change Enhances the Potential Impact of Infectious Disease and Harvest on Tropical Waterfowl

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ABSTRACT

Global warming exacerbates threats to biodiversity as ecological systems shift in response to altered climatic conditions. Yet the long-term survival of populations at direct risk from climate change may also be undermined by local factors such as infectious disease or anthropogenic harvest, which leave smaller and more isolated populations increasingly vulnerable to the rapid pace of global change. We review current and future threats to an exemplar tropical waterfowl species, magpie geese *Anseranas semipalmata*, and focus on the potential synergies between infectious diseases, harvest, and climate change. We outline viral, bacterial, and fungal pathogens likely to cause disease in geese, and give mention to parasites. Further, we elaborate on a previously developed, spatially explicit population viability model to simulate demographic responses to hunting and novel or enhanced disease outbreaks due to climate change. With no harvest, the simulated disease epizootics only threatened metapopulation viability when both mortality rate was high and outbreaks were regular (a threshold response). However, when contemporary site-specific harvest is included as an additive impact, the response to disease severity and probability was linear. We recommend field research to test these hypotheses linking drivers of waterfowl population decline to disease–climate change interactions.

Key Words: *Anseranas semipalmata*; avian disease; global warming; population viability analysis; tropical Australia.

THE PREDICTED EFFECTS OF GLOBAL CLIMATE CHANGE on the extinction risk of most species and ecosystems is still highly uncertain. While there is reasonable evidence to demonstrate that global warming heightens extinction risk (Nogués-Bravo *et al.* 2008, Sekercioglu *et al.* 2008, Sodhi *et al.* 2008), the dominant mechanisms driving demographic changes that can result in extinction due to global warming, and particularly the potential interactions between climate change and other anthropogenic stressors, are not well-quantified (Brook 2008). Recent studies have linked climate change to shifts in species abundance and range (Murray *et al.* 2006, Nogués-Bravo *et al.* 2008) and changes in phenology and behavior, but only a few have directly attributed extinctions to climate change (Sekercioglu *et al.* 2008), and some have disputed particular hypothesized links (Lips *et al*. 2008).

One reason for this uncertainty is that changes in climatic parameters are not expected to be uniform across geographic regions (IPCC 2007), and the impacts of these on local environments, and populations therein, are likely to be complex and nonlinear (Parry *et al*. 2007). The mechanisms of species extinction through climate change will depend on interactions between the drivers of global change and localized threats, the net effect of amplifying (positive) and attenuating (negative) feedbacks (Brook *et al.* 2008), and the ability (or lack thereof) of species to adapt to shifting conditions (Visser 2008).

Population persistence is intricately linked to a suite of deterministic drivers of decline and intrinsic characteristics that may be unique to the location and population (Traill *et al*. 2007). Drivers of extinction include habitat loss, overexploitation, invasive species (via competitive exclusion or predation), and infectious diseases (Gilpin & Soulé 1986). These may act singularly or simultaneously, and by reducing species to small and isolated populations, they leave them more vulnerable to environmental and demographic stochasticity and catastrophic events (Caughley 1994). For example, habitat loss, hunting pressure, and competition have forced once-numerous, contiguous African antelope populations in Zimbabwe into disconnected wildlife reserves, allowing a recent, single disease event nearly to eradicate them from the region (Clegg *et al.* 2007). Thus, a synergy of processes can combine ultimately to precipitate species extirpation (Brook *et al.* 2008). Synergies between local drivers of decline and climate change are difficult to anticipate, but are expected to play a pivotal role in future extinction events (Brook 2008).

Harvest and infectious diseases can interact to drive species to extinction (Smith *et al.* 2006, Rizkalla *et al.* 2007), and global warming may contribute further to this by facilitating the spread of existing pathogens or the emergence of new diseases (Epstein 2002, Zell 2004, Senior 2008). Although recent work has reviewed the likely impacts of global warming on biodiversity (Parry *et al.* 2007, Sekercioglu *et al.* 2008), the evidence has thus far been skewed heavily toward temperate regions (see Sodhi *et al.* 2007). Given that tropical regions harbor some of the greatest species richness and endemism, and tropical species tend to be more physiologically susceptible than temperate species to small changes in temperature (Deutsch *et al*. 2008), the threats posed by climate change will be particularly pronounced in this region (Sodhi *et al*. 2007).

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Here, we consider the impacts of climate change-enhanced infectious diseases on a wide-ranging tropical waterfowl species, the magpie goose *Anseranas semipalmata*, and simulate the changing consequences of these under alternative harvest regimes. Although not currently threatened (IUCN 2008), magpie geese have been historically eliminated from their previously extensive southern range (Fig. S1) through the additive effects of habitat loss, hunting, toxicosis, and predation (Nye *et al.* 2007). Research on the exemplar tropical goose population, which has been well-studied since the 1960s (Frith & Davies 1961), will be useful to gauge the potential effect of disease–climate change interactions on other tropical waterfowl species and will contribute toward pre-emptive conservation action for magpie geese.

To achieve these objectives, we first review avian diseases affecting Anseriformes and derive or infer: (1) pathogens and parasites most likely to cause morbidity and/or mortality in magpie geese; (2) the frequency and severity of past mass-mortality events in magpie geese, or ecologically similar waterfowl; (3) the ecological correlates of these events, such as high temperatures and anaerobic wetland conditions; and (4) possible mechanisms of disease spread or enhancement through global change. Second, we simulate the outcomes of epizootics in tropical geese populations under increasing frequency and severity and include harvest as a present-day threat that potentially can be managed. We discuss the implications of the disease-harvest-climate change synergy for population persistence and the sustained use of magpie geese.

GLOBAL AND TROPICAL CLIMATE CHANGE

Modern and rapid global warming is evident from a consistent increase in air and ocean temperatures, the melting of polar and montane snow and ice, and rising mean sea levels (IPCC 2007). Global mean surface temperatures have increased by 0.74°C (0.56–0.92°C) in 100 yr (IPCC 2007) and the projections for mean global sea level rise by 2100 are 0.5–1.4 m above 1990 (Rahmstorf 2007), and substantially more (3–5 m) should the recent changes in ice sheet dynamics in Greenland and West Antarctica continue to accelerate over the next few decades (Hansen 2007). Sea levels have been altered through thermal expansion of the oceans and the exchange of water between oceans and frozen reservoirs such as glaciers, ice sheets, and ice caps (Bindoff *et al.* 2007). These temperature and geophysical changes have been attributed in part to the (anthropogenic) combustion of fossil fuels releasing CO_2 and other long-lived greenhouse gases (IPCC 2007). As a result, global atmospheric CO_2 concentration has risen from pre-industrial levels of $\sim$ 280 parts per million (ppm) to 385 ppm by 2006 (IPCC 2007).

Shifts in climate are not expected to be uniform across geographic regions, and the impacts of these on biodiversity will be asymmetrical. For example, some biomes are projected to be worse-affected than others, including low-lying coastal wetlands, saltmarshes, mangrove systems, coral reefs, the sea-ice biome, mid-latitude Mediterranean systems, and the Arctic tundra (Parry *et al.* 2007). As an example of a tropical system, north Australia is projected to experience sea level rise and more frequent and penetrating saline intrusions into freshwater systems due to increased storm surges (Bindoff *et al.* 2007), increased CO_2 concentration affecting plant growth and competitive interactions (Malhi & Grace 2000), heightened mean temperatures (0.2–2.2°C by 2030, and 0.8–7.2°C by 2070) relative to 1990, increased rainfall, and more intense (but not necessarily more frequent) cyclones (Hennessy *et al.* 2007).

Brook (2008) argued that the current warming event poses a greater threat to biodiversity than past Quaternary events because recent anthropogenic climate change is occurring at a rate faster than would have likely occurred in the past (see also Visser 2008). Rapid shifts in environmental conditions (such as through global change) challenge the evolutionary adaptability of species (Franklin 1980), especially for those populations whose resilience has been undermined already by loss of habitat area, degradation of habitat quality, and loss of genetic variation (Spielman *et al.* 2004, Brook 2008). Synergies between pathogens and climatic shifts are known (Paz *et al.* 2007, Pascual *et al.* 2008), and are therefore a serious concern for the preservation of biodiversity.

CLIMATE CHANGE AND INFECTIOUS DISEASES.—Climate is an important determinant of the global range limits of infectious diseases, and regional weather patterns may play an important role in modifying the risk and severity of disease outbreaks (Epstein 2002). Shifts in rainfall or temperature favor the emergence of new diseases, or outbreaks of existing disease; global warming has contributed toward the worldwide emergence and redistribution of infectious diseases since the 1970s (Epstein 2001). For example, Smith *et al.* (2006) found that disease acted concomitantly with habitat loss, hunting, and competition in the extinction of 18 bird species across the world's biomes.

Global warming may interact with disease in a number of ways: (1) Shifts in climate can cause range expansion of disease vectors and so facilitate the invasion of the pathogens they carry (Epstein 2002). For example, global warming has been implicated in the spread of pathogenic chytrid fungi and the subsequent extirpation of endemic anurans in Costa Rica (Pounds *et al.* 2006, but see Lips *et al.* 2008); (2) Climate shifts may also weaken an individual's body condition, immune response, and adaptive capacity through habitat loss, extreme weather events, or prolonged periods of resource scarcity (Epstein 2001). These conditions can then permit the expression of disease in hosts that may otherwise have been resistant. For instance, nutritional stress enhances the risk of Hendra virus infection in Australasian flying foxes (Plowright *et al.* 2008a); (3) Warming can also hasten pathogen development and increase survival; growth rates of marine bacteria and fungi are correlated with temperature (Harvell *et al.* 2002), and increased infection rates of tropical Columbids by parasitic flagellate protozoa have been linked to warmer temperatures (Bunbury *et al.* 2007).

INFECTIOUS DISEASES AND TROPICAL WATERFOWL

Given these observed and hypothesized interactions, an appropriate step for proactive species conservation is the documentation of infectious diseases that do and may potentially infect populations, the measurement of their known or likely effects on mortality rates, and inference on possible mechanisms of these under climate change. Below we consider the role of infectious disease in waterfowl populations; although we include reference to epizootics in temperate regions, we only include these where there is the potential for occurrence in the tropics. We base the review below on the major taxonomic groupings of pathogens (*i.e.*, viruses, bacteria, fungi, and parasites) and describe pathogens and parasites most likely to cause disease in our case study species.

VIRAL DISEASES.—Viral infections have been responsible for substantial mortality in avian populations, and waterfowl are epidemiologically important by acting as natural reservoirs and sources of transmission (Hess & Pare 2004, Tracey *et al.* 2004). Indeed, waterfowl play a potentially pivotal role in the transmission of viruses in tropical regions because of the sizes of communal aggregations on seasonal tropical flood plains (Bayliss & Yeomans 1990) and mingling of these populations with those of migratory species (Tracey *et al.* 2004).

Duck viral enteritis, or duck plague, is transmitted primarily through viruses shed in feces (Hess & Pare 2004). An epizootic killed *ca.* 43,000 waterfowl in North America, including Muscovy *Cairina moschata*, mallards *Anas platyrhynchos*, and black ducks *Anas rubripes* (Converse & Kidd 2001). Virulence in magpie geese is unknown, but Spieker *et al.* (1996) found that Canada geese *Branta canadensis* were less susceptible than duck species.

Goose parvovirus infection seriously affects goslings, with extremely high (up to 99%) mortality (Jansson *et al.* 2007). Prevalent in Asia, transmission may be fecal-oral, or via eggshell from subclinically infected geese (Hess & Pare 2004). Tropical anatids are also susceptible to infection by Newcastle disease virus (NDV), but typically do not develop disease symptoms. Velogenic NDV was however, responsible for outbreaks among farmed geese in China, accounting for 17.5 percent morbidity and 9.2 percent mortality (Wan *et al.* 2004). Geese may play an important role in the epidemiology of this disease by acting as a reservoir and dispersal agent (Hess & Pare 2004). Pathogenesis (of NDV) in magpie geese is unknown, and monitoring of a Northern Territory population in 2006 showed nil sero-prevalence (AQIS 2006).

Tropical waterfowl are an important reservoir and possible dispersal agent for avian influenza viruses (AIVs) (Tracey *et al.* 2004). With 65.9 percent of all AIV isolates sourced from Anatidae (Kaleta *et al.* 2005), global prevalence in geese ranges from 0.6 to 2.2 percent (Munster *et al.* 2007), and outbreaks are uncommon. Nonetheless, mortality from these can be high; a recent H5N1 epizootic killed 5–10 percent of a bar-headed goose *Anser indicus* population in western China (Chen *et al.* 2005). North Australian magpie geese carry low pathogenic strains of AIV, and the high pathogenic strains are fatal (AHS 2006, AQIS 2006). Fecal-oral transmission (Friend & Franson 1999) may be high where geese aggregate on large shallow-water wetlands and mix with migratory shorebirds and numerous other waterfowl (Tracey *et al.* 2004).

West Nile Virus (WNV) is a zoonotic virus that belongs to the Japanese encephalitis virus antigenic complex and is primarily transmitted by mosquitoes (Hess & Pare 2004). Susceptibility appears to vary among species with high mortality rates in young domestic geese: 692 of 2731 goslings (25%) over 10 d in the USA (Austin *et al.* 2004), 160 of 400 goslings (40%) in Israel (Swayne *et al.* 2001), and 504 of 3600 (14%) in Hungary (Glavits *et al.* 2005). The status of WNV and indeed, Japanese B encephalitis, in northern Australia is unknown, nor is the pathogenicity of flaviviridae in magpie geese. It is possible that the species may act as a reservoir for WNV or other Japanese encephalitis strains were these to be introduced to northern Australia. Waterfowl are implicated in the cycle of many other arboviruses such as Murray valley encephalitis, Ockelbo virus, Sindbis virus, and Usutu virus, and may act as susceptible dead-end hosts or reservoirs (Chvala *et al.* 2006, 2007).

Outbreaks of hemorrhagic nephritis enteritis occurred in farmed goose flocks of 3–10 weeks in age, and mortality was 4–67 percent (Palya *et al.* 2004). A distinct polyomavirus species comprising variable strains was attributed as the cause (Palya *et al.* 2004).

BACTERIAL DISEASES.—Bacterial infections are a more common cause of mortality in waterfowl than are viral diseases (Friend & Franson 1999). Bacteria of the genus *Clostridium* in particular are responsible for more wild bird deaths than any other disease agent, and avian cholera has become the most important infectious disease of waterbirds in North America (Friend & Franson 1999, Friend 2002). Although some data are available on bacterial disease outbreaks in temperate regions, few exist for the tropics.

Avian botulism has been responsible for the deaths of many thousands of individual waterfowl (Friend 2002). Anaerobic bacteria *Clostridium botulinum* Type C produce dormant spores that remain viable and widely distributed in wetland sediments for decades (Rocke & Samuel 1999). Neurotoxin is produced after the spores germinate, usually under anaerobic conditions and high temperatures (30–37°C), and ingestion leads to lethal paralysis and death (Rocke & Samuel 1999). Outbreaks are likely to occur where birds occupy shallow, stagnant waters where dissolved oxygen content is low, temperature exceeds 20°C, and decaying animal material is abundant (Rocke & Samuel 1999). Losses of waterfowl have been high; on the Salton Sea, Friend (2002) documented large-scale mortality events where 2000–6000 waterfowl perished. In tropical Australia, mass mortality (up to thousands) of magpie geese on the flood plains of the Mary River during the hot, late dry season of 1989 has been witnessed (P. Whitehead, pers. comm.). Birds were not counted systematically and the major cause of mortality was not identified; however, an outbreak of type C botulism in Australian pelicans *Pelecanus conspicillatus* was recorded in the same region (L. Melville, pers. comm.).

Avian cholera is a contagious disease that results from infection by the bacterium *Pasteurella multocida* (Friend & Franson 1999).

Environmental contamination from diseased birds is a primary source for infection and high concentrations of *P. multocida* have been found in waters several weeks following waterfowl mortality events (Friend & Franson 1999, Waldenstrom *et al.* 2003). Ingestion appears to be the most common form of infection, particularly for grubbing waterfowl species, followed by bird-to-bird contact and aerosols (Friend & Franson 1999). It is likely that most bird species may become infected with *P. multocida*, but pathogenesis appears to depend on the bacterium strain, host susceptibility, and the infectious dose (Samuel *et al.* 2007). Chronic low-level mortality may continue throughout the year and so account for a substantial portion of annual population loss, yet it may be overlooked because it does not lead to mass kills (Samuel *et al.* 2007). Mortality events in lesser snow geese *Chen caerulescens* have been recorded for 20,000–30,000 individuals (Blanchong *et al.* 2006). These authors also found that wetland water conditions are not strongly associated with the risk of cholera outbreak, but that eutrophication and El Niño climate events can cause *P. multocida* to flourish.

Of 20 known strains of *Mycobacterium avium* causing avian tuberculosis, three lead to disease in waterfowl (Friend & Franson 1999). Transmission is again through bird-to-bird contact or ingestion of contaminated water. Disease appears to be present within most wild bird populations (Friend & Franson 1999). The status of avian tuberculosis in north Australian magpie geese is unknown, but *M. avium* can survive for long periods outside of the host, and because transmission is typically fecal-oral (Friend & Franson 1999), infection is likely.

Other bacterial diseases caused by species such as *Clostridium perfringens* have caused mass mortality in waterfowl species; following an abrupt change in diet, the bacterium proliferates and produces toxins (Wobeser & Rainnie 1987). *Salmonella* bacteria also cause disease in avian species, notably salmonellosis (Friend & Franson 1999). Transmission is usually through ingestion and the longevity of the bacteria outside of hosts makes some tropical waterfowl species vulnerable.

FUNGAL DISEASE.—It is likely that disease-causing fungi are common on tropical flood plains, and opportunistic infections may occur when birds are immuno-suppressed. Three basic types of avian disease are caused by fungi: mycosis (direct invasion of tissues by fungal cells), mycotoxicosis (from ingestion of toxic fungal metabolites), and allergic disease (Friend 2002). Respiratory tract infection may result from aspergilliosis *Aspergillus fumigatus* caused by a saprophytic mould growing on damp soils, organic debris, and decaying vegetation. Aspergilliosis is reported worldwide and outbreaks have occurred in waterfowl, usually acting concomitantly with immuno-supression precipitated by lead poisoning, injury, or drought (Friend & Franson 1999).

PARASITES.—Many tropical waterfowl are infected with parasite fauna. Anecdotal evidence suggests that magpie geese have a waterfowl-typical helminth parasite fauna: cyclocoelid flukes are prominent during the tropical dry–wet transition months, and high loads of *Echinostoma* flukes have been found in magpie goose goslings (W. Freeland, pers. comm.). Further, avian malaria is a known threat to migratory waterfowl using tropical wetlands (Wikelski *et al.* 2004, Mendes *et al.* 2005). The symptoms of these are unknown, but it is possible that parasites reduce body condition and so contribute toward bird mortality during periods of nutritional stress or injury.

In summary, duck plague, strains of the AIV, avian botulism, cholera, and aspergilliosis are most likely to cause catastrophic mass mortality events among tropical magpie geese populations, and tropical waterfowl species in general. High temperatures, anaerobic wetland conditions, and mass aggregations of waterfowl may precipitate epizootics. The frequency and severity of these is unknown, but we speculate that the predicted rise in mean atmospheric temperatures in tropical Australia by as much as 7.2°C by 2070, and more frequent hot days and nights (IPCC 2007) will facilitate increased disease outbreaks.

Our review highlights the array of diseases that do and potentially could affect mortality rates in tropical waterfowl, and demonstrates two important aspects relevant to population persistence. First, failure to incorporate the probability of mass mortality events (catastrophes *sensu* Reed *et al.* 2003) arising from disease could seriously compromise estimates of population extinction, and tropical waterfowl are particularly susceptible given their general foraging mode, migratory behavior, and exposure to a wide range of pathogens (Tracey *et al.* 2004). Indeed, the inclusion of disease in population viability analyses of threatened avian species populations greatly increases extinction risk (Brook & Kikkawa 1998). Second, mortality arising from disease can go unnoticed because it does not necessarily occur in punctuated mass events; rather, disease reduces average survival rates below that expected naturally or from hunting and predation (Ives & Murray 1997).

To illustrate these aspects and their implications for population persistence, we next model the dynamics of a well-studied species of tropical waterfowl (magpie geese), incorporating natural, disease-related, and predation (traditional and recreational harvest) mortality to examine their relative and interacting contributions to extinction probability under climate change. Further, our work addresses recent concerns about the lack of realistic population models in avian research (Beissinger *et al.* 2006).

PERSISTENCE OF MAGPIE GEESE IN NORTHERN AUSTRALIA

ECOLOGY, LIFE HISTORY, AND THREATS.—Native to Australia and New Guinea (Fig. S1), and sole members of the family *Anseranatidae*, magpie geese are estimated at *ca.* 3.5 million individuals in the Northern Territory of Australia alone (Bayliss & Yeomans 1990, Whitehead *et al.* 1992). Populations depend on extensive subcoastal wetlands for forage and breeding (Bayliss & Yeomans 1990). During the dry months (June–October), geese aggregate in flocks of up to 250,000 on shallow-water wetlands, and grub for tubers of the sedge plant *Eleocharis dulcis* (Whitehead *et al.* 1992). Ephemeral flood plains with dense plant growth are used for nesting following monsoonal rains, and dispersal is seasonally nomadic,

typically between favored forage and nesting sites (Whitehead *et al.* 1992). Mean adult weight is 2.8 and 2.0 kg for males and females, respectively (Frith & Davies 1961). Sexual maturity is reached by 24 mo in females and 36 mo in males, mating is polygynous, and breeding is cooperative with clutch size of 5–11 (Whitehead 1998).

Current threats to tropical Australian populations include habitat loss to invasive plants, saline water intrusion and eventual inundation through sea level rise (Lonsdale 1994, Hennessy *et al.* 2007), wetland alteration by nonnative swamp buffalo *Bubalus bubalis* and pigs *Sus scrofa* (Bradshaw *et al.* 2007), poisoning by spent lead shot (Whitehead & Tschirner 1991), emerging infectious diseases (as discussed previously), and unsustainable hunting rates (Brook & Whitehead 2005). Recreational harvest of geese in northern Australia is estimated at *ca.* 30,000 birds/year (Brook & Whitehead 2005), and is regulated through a license system (Whitehead *et al.* 1992). Unregulated Aboriginal harvest is estimated to be 50,000–150,000 birds/year, and this in addition to recreational harvest is considered sustainable if the current environmental conditions prevail (Brook & Whitehead 2005). We note that shooting by tropical fruit-growers and mortality through injury (by lead shot) has not been estimated, and is likely to raise these figures (see for example Noer *et al.* 2007).

Response to global warming.—Physiologically, magpie geese are adapted to cope with tropical extremes of heavy rainfall and high temperatures (Frith & Davies 1961, Whitehead *et al.* 1992). It is unlikely that temperature shifts will affect geese directly, other than the most extreme projections. There is some support from climatological model for a change in the return time or intensity of El Niño-Southern Oscillation events, and instability of the tropical monsoon (IPCC 2007), but its implication for northern Australian ecosystems remains unclear. Ecologically, geese are generalist herbivores capable of dispersal in response to food availability (Frith & Davies 1961), and present populations are large enough to maintain genetic heterozygosity and evolutionary adaptability (Franklin & Frankham 1998). There is evidence, however, that changed hydrological regimes may affect geese nesting habits to the detriment of tropical populations (Whitehead 1998).

Habitat loss, infectious disease, and unchecked harvest (Brook & Whitehead 2005) pose the most serious global warming-linked threats to tropical geese populations. Below, we model likely population scenarios where disease outbreaks are included and where harvest is regulated or allowed to continue.

Minimum population abundance, disease, and harvest.—To test quantitatively the effect of disease on north Australian magpie geese, we incorporated an additional mortality factor in a stage-structured metapopulation model of geese developed by Brook and Whitehead (2005). In that study, the long-term impacts of recreational and Aboriginal hunting on magpie geese were considered based on available survey data on population size and spatial distribution, dispersal, survival, and site-specific harvest regimes. The details of the original model constructed using RAMAS Metapop ver. 4 (Akçakaya 2002) are described in Brook and Whitehead (2005).

To consider the role of novel or climate change-enhanced infectious disease, we sampled from a broad 'parameter space' of a likely epizootic frequency and severity in geese in the modified population models, with and without harvest. We first constructed a data set for a population reduction multiplier, *i.e.*, a factor that reduces the population abundance according to the estimated die-off from disease (severity). For example, to estimate additional mortality due to disease of 10 percent, a population reduction multiplier of 0.9 is used. We derived 100 reduction multipliers by sampling randomly from a uniform distribution with a range of 1–99 percent. We then followed the same process for the frequency of an outbreak. Here we considered the probability of occurrence to fall realistically between 0 and 25 percent per time step (year), *i.e.*, from no chance of an outbreak through to an average of one outbreak every 4 yr, and derived a random data set (100 points) of these values. RAMAS-based population viability analyses (PVA) allow both of these parameters to be specified (see Akçakaya 2002). The severity and frequency data sets were then randomly paired by sampling each parameter (without replacement) until 100 sensitivity scenarios were produced.

Each parameter combination was then iterated 100 times in the base RAMAS model, and the population was projected 100 yr into the future. We ran simulations for two harvest scenarios where: (1) harvest was 130,000 birds/year based on field data for northern Australia (Brook & Whitehead 2005); and (2) no harvest was allowed. Outputs for these were final population size and expected minimum abundance (EMA) of the total magpie goose population over the 100-yr projection interval. EMA is the average of the smallest population size attained in each iteration, and is a useful indicator of the propensity for species decline because it is not a bounded $[0–1]$ measure like extinction probability, where large regions of the scenario space may produce predictions of either 0 or 1 precisely, and are therefore uninformative (McCarthy & Thompson 2001).

We used generalized linear mixed-effects models to examine the relationship between frequency and severity of disease and harvest on the EMA, fitting these via maximum likelihood in the R Language (R Core Development Team 2007). An index of Kullback–Leibler (K–L) information loss was used to assign relative strengths of evidence to the different competing models, and Akaike's information criterion (AIC_c) was used as an objective means of model comparison (see Burnham & Anderson 2001). Hunting mortality had a stronger influence on EMA than the probability of disease ($R^2 = 0.71$ for EMA $\sim$ hunting mortality and $R^2 = 0.27$ for EMA $\sim$ probability of disease), but the interaction between these variables provided the best model fit (AIC_c weight $[wAIC_c] = 0.552$, deviance explained = 97%). We controlled for harvest type by including it as a random effect.

PVA results for both harvest and no-harvest scenarios show that geese populations are more resilient to disease when harvest is disallowed: fitted linear regression between mortality and the EMA (Fig. 1A, B) show a delayed (threshold) decline of the goose population when harvest is absent, but a more consistent linear response, and a reduced population viability across a broad range of disease scenarios under current harvest pressure. These threshold versus linear response predictions are best illustrated where we fit smoothed three-dimensional plots of the EMA both to disease severity and probability of an outbreak occurrence (Fig. 2A, B). Field studies have shown that harvest and disease can act in synergy to drive

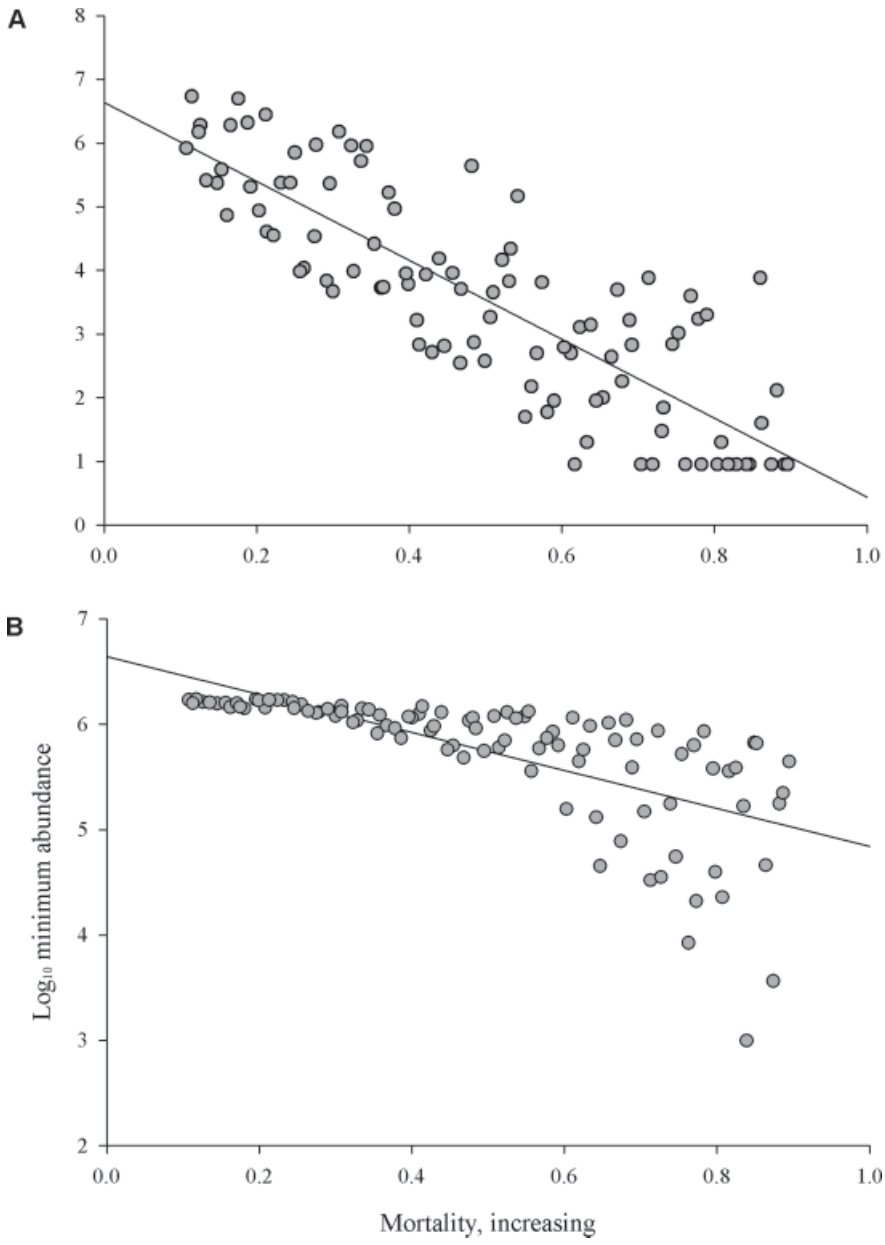

FIGURE 1. Interaction between the expected minimum abundance (EMA, $\log_{10}N$) and percent disease-driven mortality (simulating scenarios of increasing disease severity) based on a stochastic stage-structured metapopulation model of magpie geese: (A) Harvest at 130,000 birds/year spread proportionally across flood plains (R^2 = 0.710); (B) No harvest (R^2 = 0.456). Lines show the least-squares fits.

species to local extinction (Rizkalla *et al.* 2007, Vogel 2007). Total restriction on traditional harvest of magpie geese is unrealistic, but these findings do nonetheless highlight the ecological importance of ensuring that harvest is constrained, particularly if populations are challenged simultaneously by habitat loss and epizootics.

A review of available literature suggests that catastrophic die-offs are likely to occur with probability of *ca.* 0.14/generation in vertebrate populations (Reed *et al.* 2003). Generation length in Reed *et al.* (2003) was the average age of mothers across all offspring produced. We speculate that this may be *ca.* 5 yr in wild magpie geese based on field research (Frith & Davies 1961). As such, abrupt mortality events caused through disease outbreaks may occur with a probability of *ca.* 0.0169/year in the absence of climate change. We modeled catastrophes (where severity was 50% decline in population abundance) in the geese population using the above frequency estimator, and found that the EMA for magpie geese was *ca.* 368,000 individuals where harvest continues and *ca.* 1,657,000 where harvest is strictly controlled.

Based on these results, climate change would need to enhance the frequency and severity of epizootics greatly to threaten magpie geese (in the absence of harvest) due to the predicted lack of sensitivity to disease risk. However, under current harvest regimes,

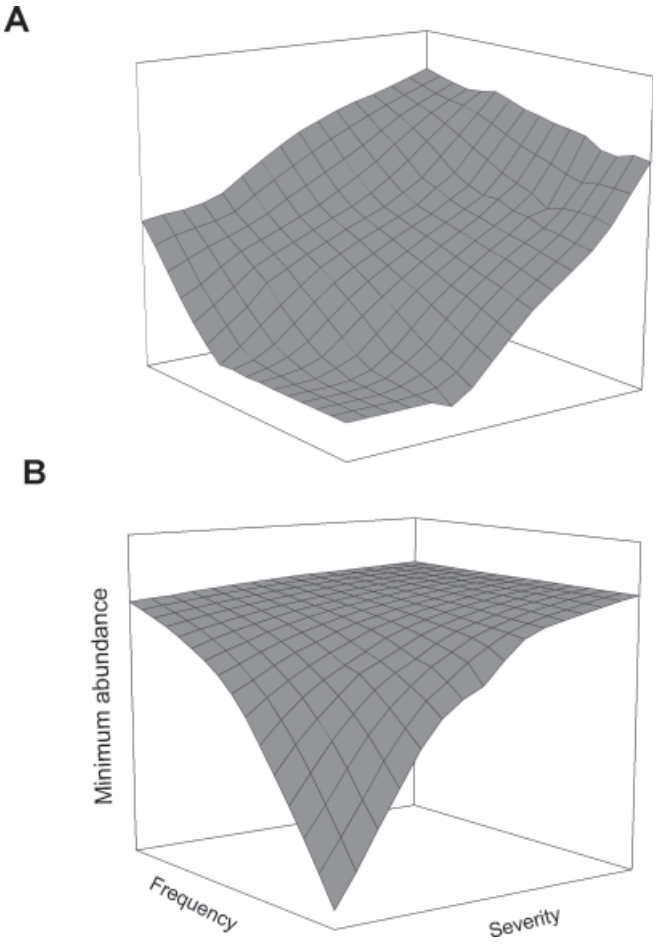

FIGURE 2. Fitted surface plots of the interaction between the expected minimum abundance (EMA, $\log_{10}N$), and disease mortality (disease severity) and increasingly probabilities (frequency) of disease outbreak based on a stochastic stage-structured metapopulation model of magpie geese: (A) harvest at 130,000 birds/year spread proportionally across flood plains; (B) No harvest.

climate change-induced enhancement of epizootics would act synergistically with hunting to diminish long-term population viability progressively. Avian botulism, avian cholera, high pathogenic avian influenza, and aspergilliosis are the diseases most likely to cause mass mortality in tropical magpie geese populations that will lead to local extinctions if hunting continues at its current intensity and if climate change progresses unabated.

DISCUSSION

Infectious disease can weaken the resilience of populations that might otherwise have been relatively robust to human sources of stress (Pedersen *et al.* 2007, Plowright *et al.* 2008b), and may even be the direct cause of the final extirpation of small, isolated, or otherwise vulnerable populations (Hale & Briskie 2007, Lips *et al.* 2008). Conservation biologists have long recognized the role of catastrophic disease in species extinction (Terborgh & Winter 1980, Roelke-Parker *et al.* 1996), and the chronic role of pathogens and parasites in reducing the reproductive fitness of individuals within populations (Packer *et al.* 1991). Indeed, infectious disease is considered to be one of the top five causes of global species extinctions and was listed as a contributing factor in 4 percent of recorded species extinctions over the last 500 yr (Smith *et al.* 2006).

Infectious diseases have precipitated avian extinctions, often acting in synergy with other deterministic factors such as habitat loss and harvest (Smith *et al.* 2006). Waterfowl appear to be particularly susceptible to catastrophic mortality events because of their tendency to form large seasonal breeding and feeding aggregations that facilitate pathogen transmission (Converse & Kidd 2001, Chen *et al.* 2005). Although avian disease has received less attention in the Australian tropics compared to temperate regions (see Tracey *et al.* 2004), inference is possible from measurements of infectious diseases common across geographic regions (Friend & Franson 1999, Hess & Pare 2004).

Recent and ongoing shifts in climate following global warming (IPCC 2007), and the direct and indirect impacts of climate change on tropical regions (Hennessy *et al.* 2007) are anticipated to increase a species' extinction probability, especially via synergistic interaction with other drivers of decline (Zell 2004, Pascual *et al.* 2008). Harvest may undermine population resilience further through loss of genetic variation and population fragmentation (Frankham *et al.* 2002, Rizkalla *et al.* 2007), making these more vulnerable to catastrophic events (Caughley 1994, Clegg *et al.* 2007). The case study of magpie geese we described supports this expectation and suggests an increased robustness to disease outbreaks when harvest is tightly controlled. The review highlights the breadth of infectious disease that potentially threatens magpie geese, and yet there is a lack of detailed ecological and epidemiological knowledge on frequency and severity of disease. Our disease review sets a precedent for similar and future studies that explore the viability of populations threatened by global change, particularly where other drivers of decline (such as hunting) already exist. We recognize the lack of available data on projected habitat loss through sea level rise for this region, and how this may further enhance the disease-harvest-climate synergy (see Finlayson *et al.* 2006). This recognition underscores the need to develop quantitative hydrological models of this system for a broader evaluation of the risks faced by this species under global change.

Our results apply equally well to other waterfowl populations across the tropics that are vulnerable to infectious disease. Tracey *et al.* (2004) documented 18 waterfowl species that are vulnerable to viral disease in tropical Australia that use similar habitats to magpie geese. Notable among these are the Australian pelican *P. conspicillatus*, plumed *Dendrocygna eytoni*, and wandering *D. arcuata* whistling ducks, Radjah shelduck *Tadorna radjah* and gray teal *Anas gracilis*. Moreover, infectious diseases are not unique to birds, and many other vertebrate populations may be vulnerable (Young 1994, Reed *et al.* 2003), especially under a shifting climate. For example, flying foxes native to tropical Australia and Asia are threatened by emerging viral diseases (Breed *et al.* 2006), and new infectious diseases pose an identified risk to tropical Australia's livestock industry (Mackenzie *et al.* 2001, Pulliam *et al.* 2007). Interestingly, extinction of the Australian thylacine *Thylacinus*

cynocephalus was attributed, in part, to introduced pathogens acting in synergy with overhunting by people (Smith *et al.* 2006).

Future disease-oriented research on tropical waterfowl needs to focus on: (1) possible types of climate change impacts on regional tropical weather systems (*e.g.*, effects on El Niño and monsoonal stability); (2) the sensitivity of waterfowl (or any other species of concern), directly or indirectly, to climate change; (3) further evidence of disease enhancement, spread or novel emergence through climatic changes; and (4) evidence of synergies between disease and other drivers of global change. Studies tackling these issues, based on multiple working hypotheses of the additive or interactive effects of multiple drivers (Brook *et al.* 2008), will allow robust evidence-based conservation decisions that will withstand scrutiny (Burnham & Anderson 2001, Kovats *et al.* 2001) and best aid applied decision-making to maximize conservation outcomes.

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SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article:

FIGURE S1. Distribution (breeding and nomadic range) of magpie geese *Anseranas semipalmata* across Australia and New Guinea.

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LITERATURE CITED

AHS. 2006. Animal health surveillance, Quarterly Report. pp. 4–5. Australian Animal Health Council.

Akçakaya, H. R. 2002. RAMAS GIS: Linking spatial data with population viability analysis (version 4.0). Applied Biomathematics, Setauket, New York.

AQIS. 2006. Magpie goose survey, Kakadu National Park. Australian Quarantine and Inspection Service, Australian Government.

Austin, R. J., T. L. Whiting, R. A. Anderson, and M. A. Drebot. 2004. An outbreak of West Nile virus-associated disease in domestic geese *Anser anser domesticus* upon initial introduction to a geographic region, with evidence of bird to bird transmission. Can. J. Vet. Res. 45: 117–123.

Bayliss, P., and K. M. Yeomans. 1990. Seasonal distribution and abundance of magpie geese, *Anseranas semipalmata* in the Northern Territory and their relationship to habitat, 1983–1986. Wildl. Res. 17: 15–38.

Beissinger, S. R., J. R. Walters, D. G. Catanzaro, K. G. Smith, J. B. Dunning, S. M. Haig, B. R. Noon, and B. M. Stith. 2006. Modeling approaches in avian conservation and the role of field biologists. Ornithol. Monogr. 59: 1–56.

Bindoff, N. L., J. Willebrand, V. Artale, A. Cazenave, J. Gregory, S. Gulev, K. Hanawa, C. le Quere, S. Levitus, Y. Nojiri, C. K. Shum, L. D. Alley, and A. Unnikrishnan. 2007. Observations: Oceanic climate change and sea level. *In* S. Solomon, D. Qin, M. Manning, Z. Chen, M. Marquis, K. B. Averyt, M. Tignor and H. L. Miller (Eds.). Climate change 2007: The physical science basis, pp. 387–429. Cambridge University Press, Cambridge, UK.

Blanchong, J. A., D. J. Shadduck, M. D. Samuel, D. R. Goldberg, and L. H. Creekmore. 2006. Wetland environmental conditions associated with the risk of avian cholera outbreaks and the abundance of *Pasteurella multocida*. J. Wildl. Manage. 70: 54–60.

Bradshaw, C. J. A., I. C. Field, D. Bowman, C. Haynes, and B. W. Brook. 2007. Current and future threats from non-indigenous animal species in northern Australia: A spotlight on World Heritage Area Kakadu National Park. Wildl. Res. 34: 419–436.

Breed, A. C., H. E. Field, J. H. Epstein, and P. Daszak. 2006. Emerging henipaviruses and flying foxes—conservation and management perspectives. Biol. Conserv. 131: 211–220.

Brook, B. W. 2008. Synergies between climate change, extinctions and invasive vertebrates. Wildl. Res. 35: 1–4.

Brook, B. W., and J. Kikkawa. 1998. Examining threats faced by island birds: A population viability analysis on the Capricorn silvereye using long-term data. J. Appl. Ecol. 35: 491–503.

Brook, B. W., and P. J. Whitehead. 2005. Sustainable harvest regimes for magpie geese *Anseranas semipalmata* under spatial and temporal heterogeneity. Wildl. Res. 32: 459–464.

Brook, B. W., N. S. Sodhi, and C. J. A. Bradshaw. 2008. Synergies among extinction drivers under global change. Trends Ecol. Evol. 25: 453–460.

Bunbury, N., C. G. Jones, A. G. Greenwood, and D. J. Bell. 2007. *Trichomonas gallinae* in Mauritian columbids: Implications for an endangered endemic. J. Wildl. Dis. 43: 399–407.

Burnham, K. P., and D. R. Anderson. 2001. Kullback-Leibler information as a basis for strong inference in ecological studies. Wildl. Res. 28: 111–119.

Caughley, G. 1994. Directions in conservation biology. J. Anim. Ecol. 63: 215–244.

Chen, H., G. J. D. Smith, S. Y. Zhang, K. Qin, J. Wang, K. S. Li, R. G. Webster, J. S. M. Peiras, and Y. Guan. 2005. H5N1 virus outbreak in migratory waterfowl. Nature 436: 191–192.

Chvala, S., T. Bakonyi, R. Hackl, M. Hess, N. Nowotny, and H. Weissenbock. 2006. Limited pathogenicity of Usutu virus for the domestic goose *Anser domestica* following experimental inoculation. J. Vet. Med. B. 53: 171–175.

Chvala, S., T. Bakonyi, C. Bukovsky, T. Meister, K. Brugger, F. Rubel, N. Nowotny, and H. Weissenbock. 2007. Monitoring of Usutu virus activity and spread by using dead bird surveillance in Austria, 2003–2005. Vet. Microbiol. 122: 237–245.

Clegg, S. B., P. C. B. Turnbull, C. M. Foggin, and R. M. Lindeque. 2007. Massive outbreak of anthrax in wildlife in the Malilangwe Wildlife Reserve, Zimbabwe. Vet. Rec. 160: 113–118.

Converse, K. A., and G. A. Kidd. 2001. Duck plague epizootics in the United States, 1967–1995. J. Wildl. Dis. 37: 347–357.

Deutsch, C. A., J. J. Tewksbury, R. B. Huey, K. S. Sheldon, C. K. Ghalambor, D. C. Haak, and P. R. Martin. 2008. Impacts of climate warming on terrestrial ectotherms across latitude. Proc. Natl. Acad. Sci. U.S.A. 105: 6668–6672.

Epstein, P. R. 2001. Climate change and emerging infectious diseases. Microb. Infect. 3: 747–754.

Epstein, P. R. 2002. Biodiversity, climate change, and emerging infectious diseases. Conserv. Med. 27–39.

Finlayson, C. M., J. Lowry, M. G. Bellio, S. Nou, R. Pidgeon, D. Walden, C. Humphrey, and G. Fox. 2006. Biodiversity of the wetlands of the Kakadu Region, northern Australia. Aquat. Sci. 68: 374–399.

Frankham, R., J. D. Ballou, and D. A. Briscoe. 2002. Introduction to conservation genetics. Cambridge University Press, Cambridge, UK.

Franklin, I. R. 1980. Evolutionary change in small populations. *In* M. E. Soulé and B. A. Wilcox (Eds.). Conservation biology: An evolutionary-ecological perspective, pp. 135–149. Sinauer, Sunderland, Massachusetts.

Franklin, I. R., and R. Frankham. 1998. How large must populations be to retain evolutionary potential? Anim. Conserv. 1: 69–73.

Friend, M. 2002. Avian disease at the Salton Sea. Hydrobiologia 473: 293–306.

Friend, M., and J. C. Franson. 1999. Field manual of wildlife diseases: General field procedures and diseases of birds. USGS, Washington, D.C.

Frith, H. J., and S. J. J. F. Davies. 1961. Ecology of the magpie goose *Anseranas semipalmata*. Wildl. Res. 6: 92–141.

Gilpin, M. E., and M. E. Soulé. 1986. Minimum viable populations: Processes of species extinction. *In* M. E. Soulé (Ed.). Conservation biology: The science of scarcity and diversity, pp. 19–34. Sinauer, Massachusetts.

Glavits, R., E. Ferenczi, E. Ivanics, T. Bakonyi, T. Mato, P. Zarka, and V. Palya. 2005. Co-occurrence of West Nile Fever and circovirus infection in a goose flock in Hungary. Avian Pathol. 34: 408–414.

Hale, K. A., and J. V. Briskie. 2007. Decreased immune-competence in a severely bottlenecked population of an endemic New Zealand bird. Anim. Conserv. 10: 2–10.

Hansen, J. E. 2007. Scientific reticence and sea level rise. Environ. Res. Lett. 2: 6 pp.

Harvell, C. D., C. E. Mitchell, J. R. Ward, S. Altizer, A. P. Dobson, R. S. Ostfeld, and M. D. Samuel. 2002. Climate warming and disease risks for terrestrial and marine biota. Science 296: 2158–2162.

Hennessy, K., B. Fitzharris, B. C. Bates, N. Harvey, S. M. Howden, L. Hughes, J. Salinger, and R. Warwick. 2007. Australia and New Zealand. *In* M. L. Parry, O. F. Canziani, J. P. Palutikof, P. J. Van der Linden, and M. A. Hanson (Eds.). Climate change 2007: Impacts, adaptation and vulnerability. Contribution of working group II to the fourth assessment report of the Intergovernmental Panel on Climate Change, pp. 507–540. Cambridge University Press, Cambridge, UK.

Hess, J. C., and J. A. Pare. 2004. Viruses of waterfowl. Sem. Avian Exot. Pet Med. 13: 176–183.

IPCC. 2007. Climate change 2007: Synthesis report. *In* R. K. Pachauri and A. Reisinger (Eds.). Contribution of working groups I, II and III to the fourth assessment report of the Intergovernmental Panel on Climate Change, pp. 23–74. IPCC, Geneva, Switzerland.

IUCN. 2008. IUCN red list of threatened species. World Conservation Union, www.iucnredlist.org.

Ives, A. R., and D. L. Murray. 1997. Can sublethal parasitism destabilize predator-prey population dynamics? A model of snowshoe hares, predators and parasites. J. Anim. Ecol. 66: 265–278.

Jansson, D. S., R. Feinstein, V. Kardi, T. Mato, and V. Palya. 2007. Epidemiologic investigation of an outbreak of goose parvovirus infection in Sweden. Avian Dis. 51: 609–613.

Kaleta, E. F., G. Hergarten, and A. Yilmaz. 2005. Avian influenza A viruses in birds—an ecological, ornithological and virological view. Dtsch. Tierarztl Wochenschr. 112: 448–456.

Kovats, R. S., D. H. Campbell-Lendrum, A. J. Mcmichael, A. Woodward, and J. S. Cox. 2001. Early effects of climate change: Do they include changes in vector-borne disease? Philos. Trans. R. Soc. Lond. B. 356: 1057–1068.

Lips, K. R., J. Diffendorfer, J. R. Mendelson, and M. W. Sears. 2008. Riding the wave: Reconciling the roles of disease and climate change in amphibian declines. PLoS Biol. 6: e72.

Lonsdale, W. M. 1994. Inviting trouble—introduced pasture species in northern Australia. Aust. J. Ecol. 19: 345–354.

Mackenzie, J. S., K. B. Chua, P. W. Daniels, B. T. Eaton, H. E. Field, R. A. Hall, K. Halpin, C. A. Johansen, P. D. Kirkland, S. K. Lam, P. Mcminn, D. J. Nisbet, R. Paru, A. T. Pyke, S. A. Ritchie, P. Siba, D. W. Smith, G. A. Smith, A. F. Van Den Hurk, L. F. Wang, and D. T. Williams. 2001. Emerging viral diseases of Southeast Asia and the Western Pacific. Emerg. Infect. Dis. 7: 497–504.

Malhi, Y., and J. Grace. 2000. Tropical forests and atmospheric carbon dioxide. Trends Ecol. Evol. 15: 332–337.

Mccarthy, M. A., and C. Thompson. 2001. Expected minimum population size as a measure of threat. Anim. Conserv. 4: 351–355.

Mendes, L., T. Piersma, M. Lecoq, B. Spaans, and R. E. Ricklefs. 2005. Disease-limited distributions? Contrasts in the prevalence of avian malaria in shorebird species using marine and freshwater habitats. Oikos 109: 396–404.

Munster, V. J., C. Baas, P. Lexmond, J. Waldenstrom, A. Wallensten, T. Fransson, G. F. Rimmelzwaan, W. E. P. Beyer, M. Schutten, B. Olsen, A. Osterhaus, and R. A. M. Fouchier. 2007. Spatial, temporal, and species variation in prevalence of influenza A viruses in wild migratory birds. PLoS Pathogens 3: 630–638.

Murray, D. L., E. W. Cox, W. B. Ballard, H. A. Whitlaw, M. S. Lenarz, T. W. Custer, T. Barnett, and T. K. Fuller. 2006. Pathogens, nutritional deficiency, and climate influences on a declining moose population. Wildl. Monogr. 1–29.

Noer, H., J. Madsen, and P. Hartmann. 2007. Reducing wounding of game by shotgun hunting: Effects of a Danish action plan on pink-footed geese. J. Appl. Ecol. 44: 653–662.

Nogués-Bravo, D., J. Rodríguez, J. Hortal, P. Batra, and M. B. Araújo. 2008. Climate change, humans, and the extinction of the woolly mammoth. PLoS Biol. 6: e79.

Nye, E. R., C. R. Dickman, and R. T. Kingsford. 2007. A wild goose chase-temporal and spatial variation in the distribution of the magpie goose *Anseranas semipalmata* in Australia. Emu 107: 28–37.

Packer, C., A. E. Pusey, H. Rowley, D. A. Gilbert, J. Martenson, and S. J. Obrien. 1991. Case-study of a population bottleneck—lions of the Ngorongoro crater. Conserv. Biol. 5: 219–230.

Palya, V., E. Ivanics, R. Glavits, A. Dan, T. Mato, and P. Zarka. 2004. Epizootic occurrence of haemorrhagic nephritis enteritis virus infection of geese. Avian Pathol. 33: 244–250.

Parry, M. L., O. F. Canziani, J. P. Palutikof, P. J. Van Der Linden, and C. E. Hansen. 2007. Technical summary. *In* M. L. Parry, O. F. Canziani, J. P. Palutikof, P. J. Van der Linden, and C. E. Hansen (Eds.). Climate change 2007: Impacts, adaptation and vulnerability. Contribution of working groups I, II and III to the fourth assessment report of the Intergovernmental Panel on Climate Change, pp. 23–78. Cambridge University Press, Cambridge, UK.

Pascual, M., B. Cazelles, M. J. Bouma, L. F. Chaves, and K. Koelle. 2008. Shifting patterns: Malaria dynamics and rainfall variability in an African highland. Proc. R. Soc. Lond. B. 275: 123–132.

Paz, S., N. Bisharat, E. Paz, O. Kidar, and D. Cohen. 2007. Climate change and the emergence of *Vibrio vulnificus* disease in Israel. Environ. Res. 103: 390–396.

Pedersen, A. B., K. E. Jones, C. L. Nunn, and S. Altizer. 2007. Infectious diseases and extinction risk in wild mammals. Conserv. Biol. 21: 1269–1279.

Plowright, R. K., H. E. Field, C. Smith, A. Divljan, C. Palmer, G. Tabor, P. Daszak, and J. E. Foley. 2008a. Reproduction and nutritional stress are risk factors for Hendra virus infection in little red flying foxes *Pteropus scapulatus*. Proc. R. Soc. Lond. B. 275: 861–869.

Plowright, R. K., S. H. Sokolow, M. E. Gorman, P. Daszak, and J. E. Foley. 2008b. Causal inference in disease ecology: Investigating ecological drivers of disease emergence. Front. Ecol. Environ. 6: 420–429.

Pounds, J. A., M. R. Bustamante, L. A. Coloma, J. A. Consuegra, M. P. L. Fogden, P. N. Foster, E. La Marca, K. L. Masters, A. Merino-Viteri, R. Puschendorf, S. R. Ron, G. A. Sanchez-Azofeifa, C. J. Still, and B. E. Young. 2006. Widespread amphibian extinctions from epidemic disease driven by global warming. Nature 439: 161–167.

Pulliam, J. R., J. Dushoff, H. E. Field, J. H. Epstein, A. P. Dobson, P. Daszak, and G. Henipavirus ecol res. 2007. Understanding Nipah

virus emergence in peninsular Malaysia: The role of epidemic enhancement in domestic pig populations. Am. J. Trop. Med. Hyg. 77: 273–273.

R Core Development Team. 2007. R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria.

Rahmstorf, S. 2007. A semi-empirical approach to projecting future sea-level rise. Science 315: 368–370.

Reed, D. H., J. J. O'grady, J. D. Ballou, and R. Frankham. 2003. The frequency and severity of catastrophic die-offs in vertebrates. Anim. Conserv. 6: 109–114.

Rizkalla, C., F. Blanco-Silva, and S. Gruver. 2007. Modeling the impact of Ebola and bushmeat hunting on western lowland gorillas. EcoHealth 4: 151–155.

Rocke, T. E., and M. D. Samuel. 1999. Water and sediment characteristics associated with avian botulism outbreaks in wetlands. J. Wildl. Manage. 63: 1249–1260.

Roelke-Parker, M. E., L. Munson, C. Packer, R. Kock, S. Cleaveland, M. Carpenter, S. J. O'brien, A. Pospischil, R. Hofamnn-Lehmann, H. Lutz, G. L. M. Mwamengele, M. N. Mgasa, G. A. Machange, B. A. Summers, and M. J. G. Appel. 1996. A canine distemper virus epidemic in Serengeti lions *Panthera leo*. Nature 379: 441–445.

Samuel, M. D., R. G. Botzler, and G. A. Wobeser. 2007. Avian cholera. *In* N. J. Thomas, D. B. Hunter, and C. T. Atkinson (Eds.). Infectious diseases of wild birds, pp. 239–269. Blackwell Publishing, USA.

Sekercioglu, C. H., S. H. Schneider, J. P. Fay, and S. R. Loarie. 2008. Climate change, elevational range shifts, and bird extinctions. Conserv. Biol. 22: 140–150.

Senior, K. 2008. Climate change and infectious disease: A dangerous liaison? Lancet Infect. Dis. 8: 92–93.

Smith, K. F., D. F. Sax, and K. D. Lafferty. 2006. Evidence for the role of infectious disease in species extinction and endangerment. Conserv. Biol. 20: 1349–1357.

Sodhi, N. S., B. W. Brook, and C. J. A. Bradshaw. 2007. Tropical conservation biology. Blackwell Science, Oxford, UK.

Sodhi, N. S., D. Bickford, A. C. Diesmos, T. M. Lee, L. P. Koh, B. W. Brook, C. H. Sekercioglu, and C. J. A. Bradshaw. 2008. Measuring the meltdown: Drivers of global amphibian extinction and decline. PLoS One 3: e1636.

Spieker, J. O., T. M. Yuill, and E. C. Burgess. 1996. Virulence of six strains of duck plague virus in eight waterfowl species. J. Wildl. Dis. 32: 453–460.

Spielman, D., B. W. Brook, and R. Frankham. 2004. Most species are not driven to extinction before genetic factors impact them. Proc. Natl. Acad. Sci. U.S.A. 101: 15261–15264.

Swayne, D. E., J. R. Beck, C. S. Smith, W. J. Shieh, and S. R. Zaki. 2001. Fatal encephalitis and myocarditis in young domestic geese *Anser anser domesticus* caused by West Nile virus. Emerg. Infect. Dis. 7: 751–753.

Terborgh, J., and B. Winter. 1980. Some causes of extinction. *In* M. E. Soulé and B. A. Wilcox (Eds.). Conservation biology: An ecological-evolutionary perspective, pp. 119–134. Sinauer, Sunderland, Massachusetts.

Tracey, J. P., R. Woods, D. Roshier, P. West, and G. R. Saunders. 2004. The role of wild birds in the transmission of avian influenza for Australia: An ecological perspective. Emu 104: 109–124.

Traill, L. W., C. J. A. Bradshaw, and B. W. Brook. 2007. Minimum viable population size: A meta-analysis of 30 years of published estimates. Biol. Conserv. 139: 159–166.

Visser, M. E. 2008. Keeping up with a warming world: Assessing the rate of adaptation to climate change. Proc. R. Soc. Lond. B. 275: 649–659.

Vogel, G. 2007. Scientists say Ebola has pushed western gorillas to the brink. Science 317: 1484.

Waldenstrom, J., S. L. W. On, R. Ottvall, D. Hasselquist, C. S. Harrington, and B. Olsen. 2003. Avian reservoirs and zoonotic potential of the emerging human pathogen *Helicobacter canadensis*. Appl. Environ. Microb. 69: 7523–7526.

Wan, H. Q., L. G. Chen, L. L. Wu, and X. F. Liu. 2004. Newcastle disease in geese: Natural occurrence and experimental infection. Avian Pathol. 33: 216–221.

Whitehead, P. J. 1998. Dynamics of habitat use by the magpie goose *Anseranas semipalmata*: Implications for conservation management. PhD Dissertation. Northern Territory University, Darwin, Australia.

Whitehead, P. J., and K. Tschirner. 1991. Lead shot ingestion and lead-poisoning of magpie geese *Anseranas semipalmata* foraging in a northern Australian hunting reserve. Biol. Conserv. 58: 99–118.

Whitehead, P. J., B. A. Wilson, and K. Saalfeld. 1992. Managing the magpie goose in the Northern Territory: Approaches to the conservation of mobile fauna in a patchy environment. *In* I. Moffatt and A. Webb (Eds.). Conservation and development issues in North Australia, pp. 90–104. North Australia Research Unit—Australian National University, Darwin.

Wikelski, M., J. Foufopoulos, H. Vargas, and H. Snell. 2004. Galapagos birds and diseases: Invasive pathogens as threats for island species. Ecol. Soc. 9: 5.10.

Wobeser, G., and D. J. Rainnie. 1987. Epizootic necrotic enteritis in wild geese. J. Wildl. Dis. 23: 376–385.

Young, T. P. 1994. Natural die-offs of large mammals—implications for conservation. Conserv. Biol. 8: 410–418.

Zell, R. 2004. Global climate change and the emergence/re-emergence of infectious diseases. Int. J. Med. Microbiol. 293: 16–26.