

**Quantifying the demographic and population impact of avian cholera on northern common eiders in the face of ancillary threats and changing environmental circumstances**

by

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## Abstract

Emerging infectious diseases are on the rise globally; however, determining demographic impacts and the influence of disease on wildlife population dynamics remains a significant challenge. A poleward expansion of disease-causing pathogens has been predicted in association with globalization and climate change and historically unaffected Arctic wildlife may be particularly vulnerable to epidemic disease. My research examines the influence of newly emerged avian cholera outbreaks on northern common eiders (*Somateria mollissima borealis*) in the Canadian Arctic. Avian cholera is a highly virulent disease of birds caused by the bacterium *Pasteurella multocida*. I take advantage of a unique opportunity to investigate transmission dynamics and the impact of this disease on eider survival and reproductive rates in a marked population at Mitivik Island, Nunavut (64.030 N, 81.789 W). I also test hypotheses on ecological factors influencing spatial occurrence of the disease with data gathered from extensive site investigations, undertaken with the assistance of Inuit harvesters throughout the Hudson Strait region. To date, mass-mortality events have been confined to northern Québec (Nunavik) and Mitivik Island with annual mortality rates of up to 43% of the female nesting population at affected colonies. Consistent with expectations for a novel pathogen, case incidence increased in an exponential fashion during the initial wave of invasion ( $R_0 = 2.5$ ). Recurrent outbreaks were documented during eight consecutive breeding seasons at Mitivik Island and prolonged low-frequency transmission ( $R_t < 1$ ) was evident at the epidemic tail—a hallmark of disease persistence facilitated by environmental transmission. Harvest mortality appears to be additive to disease mortality in this population, which is relevant because

adjustment of hunting regulations is among the few logistically feasible interventions to conserve abundance. Demographic recovery has so far been constrained by a climate-mediated increase in the frequency of polar bear (*Ursus maritimus*) incursions onto eider breeding colonies and associated declines in nesting success. The causes and effects of emerging infectious diseases rarely operate in isolation from other ecological stressors and the types of information that I gathered informs scientific understanding of the interacting processes regulating population dynamics in the face of rapid anthropogenically-driven environmental change.

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## Statement of Coauthorship

This document follows an integrated thesis format and contains a General Introduction (Chapter 1), General Discussion (Chapter 6), and four data chapters configured as independent research articles (Chapters 2-5). One data chapter has already published (Chapter 4) and the necessary permissions have been obtained from the publisher for reproduction here. The remaining data chapters are currently under review or in preparation for scientific journal submission. I have received permission from each of my coauthors for the reproduction of our collaborative work in my thesis. My contributions to the research were as follows:

- (1) I proposed and developed the research questions outlined in the data chapters with input from my co-advisors Dr. M. R. Forbes (Carleton University) and Dr. H. G. Gilchrist (Environment Canada).
- (2) I was responsible for carrying out and/or supervising the field work described in the four data chapters. Specifically, I developed the field methodologies, hired and trained field assistants, and participated in the collection of the data. I was also granted access to a long-term dataset collected by biologists under the direction of Dr. H. G. Gilchrist (Mitivik Island) and Dr. A. J. Gaston (Cape Pembroke).
- (3) I was responsible for all of the data analysis and presentation of results for the data chapters.
- (4) I was responsible for all of the writing in this document, receiving helpful review comments on earlier drafts.

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## Chapter 1: General Introduction

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Understanding relationships between life-history patterns, variation in vital rates, and population dynamics are central issues in population ecology (Lack 1954, Stearns 1992, Sæther et al. 2004, Péron et al. 2012). Anthropogenically-driven environmental changes resulting from the cascading and intertwined effects of globalization, habitat loss, and climate change are intensifying and are thought to have important effects on animal and plant populations and, by extension, on biodiversity (Vitousek et al. 1997, Foley et al. 2005, Walther et al. 2002). While research shows that evolutionary responses to environmental change can occur much faster than traditionally assumed (Schoener 2011, Bell 2012), maintenance of population abundance remains of critical importance for ensuring population viability, ecosystem function, sustainable yield, and for ensuring healthy levels for a variety of other biological and social metrics (Beissinger and McCullough 2002, Soulé et al. 2003, Sanderson 2006). Different types of ecological perturbation have different mechanisms regarding the timing and extent of injury imparted on individuals (Beissinger and McCullough 2002, Morris and Doak 2002, Péron et al. 2012). Knowing how life history characteristics modulate a population's capacity to resist, compensate and recover from emerging challenges is essential for predicting population impacts and for guiding conservation planning (Thomas et al. 2004, Willi and Hoffmann 2009, Chevin et al. 2010).

Emerging infectious diseases are on the rise in wildlife (Daszak et al. 2000, Dobson and Foufopoulos 2001). Emerging diseases are defined as diseases that are increasing in their

incidence or expanding in their geographic or host range (Morse 1995) and are a particular concern to biological conservation because their causes and effects rarely operate in isolation from other ecological stressors (Plowright et al. 2008, Smith et al. 2009). The main factors underlying disease emergence, such as habitat alteration or loss, species invasions, and climate change, can have independent and synergistic impacts that contribute to population declines (Daszak et al. 2001, de Castro and Bolker 2005, Smith et al. 2009). For example, habitat loss can cause crowding, which in turn leads to more competition for resources, higher contact rates, and decreased ability of individuals to resist infection (Bradley and Altizer 2007, Lindsey et al. 2009). Similarly, invasive species can be competitors with native species as well as a source of novel pathogens in previously disease-free communities (Smith and Carpenter 2006, Borer et al. 2007). Finally, warming temperatures can alter abiotic conditions such that animals experience thermal stress while pathogens experience improved growth and survival leading to accelerating ecological impact (Harvell et al. 2002).

Host-pathogen, like predator-prey relationships are complex and potentially troubling from a conservation perspective because of the dynamic nature of pathogen transmission, wherein the numbers of both pathogens and hosts are regulated by their interaction (Anderson and May 1979, Anderson and May 1991, Gerber et al. 2005). Pathogens are rarely predicted to drive their hosts to extinction because epidemic fade-out is predicted to occur when host numbers drop below the density threshold that is critical for disease persistence (de Castro and Bolker 2005, Lloyd-Smith et al. 2005). However, emerging infectious diseases have frequently been associated with temporary and even permanent declines in threatened and economically

important species (Daszak et al. 2000, Dobson and Foufopoulos 2001, de Castro and Bolker 2005, Smith et al. 2009). In some instances, disease control is possible through management intervention such as mass-vaccination to reduce the proportion of susceptible individuals in a population or culls to eliminate infective hosts. However, in most biological systems with free ranging animals, the capacity for intervention is limited and the more immediate and practical concern is to know what severity and periodicity to expect from unmanaged epidemics (Wobeser 2002). Obtaining such information requires detailed study of transmission dynamics, the impacts of disease on host vital rates, and knowledge of how ecological and life history characteristics influence the capacity of individuals within a diseased population to compensate for and recover demographically from disease-induced losses.

Wildlife populations in polar regions are expected to be particularly vulnerable to global disease emergence. A poleward expansion of disease-causing pathogens has been predicted in association with globalization and climate change (Harvell et al. 2002, Altizer et al. 2013). Key drivers include species introductions, increased survival and transmission of free-living bacteria and parasites dependent as temperatures warm and increased prevalence of insect-borne disease vectors (Bradley et al. 2005, Kutz et al. 2005, Parkinson et al. 2014). Many Arctic and Antarctica animal species are thought to have evolved in relative isolation, which has limited their contact with pathogens and likely reduces their immune capabilities for fending off introduced disease (Dobson 2009, Kerry and Riddle 2009). Polar-adapted species possess many traits that enable them to persist in difficult conditions, the most oft-cited of which are cold tolerance and the ability to endure highly variable weather conditions from one season or year

to the next (Callaghan et al. 2004, Post et al. 2009). Temporally stochastic conditions shape life histories in important ways: e.g., a pervasive feature of wildlife species in highly variable environments is investment in physiological and behavioral traits that promote survivorship at the expense of productivity (Morris and Doak 2004, Metcalf and Koons 2007, Iles et al. 2013a). Such adaptations enable animals to endure a run of poor years with respect to offspring production and take advantage of more suitable conditions for breeding when those become available. Intriguingly, the disease ecology literature suggests that species with specialized habitat requirements and low fecundity are the least able to compensate when epidemics occur and are among the most vulnerable to emerging infections (Williams and Hero 1998, de Castro and Bolker 2005).

My research examines avian cholera emergence in northern common eiders (*Somateria mollissima borealis*) in the Canadian Arctic. Avian cholera is an infectious, contagious disease of birds caused by the bacterium *Pasteurella multocida*. Common eiders are a large-bodied sea duck distributed over the northern coasts of Europe, North America, and eastern Siberia (BirdLife International 2015). The northern subspecies breeding range extends from west and northwestern Greenland throughout the eastern Canadian Arctic, with wintering areas in west Greenland and eastern Canada from Hudson Strait to the Gulf of St. Lawrence (Goudie et al. 2000). Northern common eiders are highly abundant ( $N > 300,000$  breeding pairs; Gilliland et al. 2009); however the population is heavily exploited by harvesters throughout its range and faces a number of conservation challenges (Kingsley 2006).

Avian cholera appears to be a new source of mortality in the population (Buttler 2009, Henri et al. 2010, Harms 2012). Key aspects of disease ecology are described in the chapters that follow. Briefly, *P. multocida* is a genetically diverse organism. The pathogenicity of genetic strains is highly variable, as is host species susceptibility (Christensen and Bisgaard 2000). Epidemics can be highly explosive and typically occur in wetlands with abundant waterfowl populations or at breeding colonies with high densities of birds (Botzler 1991, Friend 1999, Samuel et al. 2007). The bacterium is spread directly via bird-to-bird contact and environmentally through ingestion or inhalation of aerosolized bacteria in contaminated water or sediments. The disease is a major source of mortality in wild and domestic birds and is a growing conservation concern owing to the magnitude of loss that may be incurred in a single outbreak event, the broad spectrum of species affected, and the continually expanding geographic area of occurrence (Samuel et al. 2007). In both Europe and North America, the first reports of mass-mortality to involve wild waterfowl occurred in close proximity and immediately following outbreaks involving poultry (Van den Hurk 1946, Rosen and Bischoff 1949). It has been hypothesized that pathogen exchange at the wild bird-domestic bird interface is associated with the evolution and spread of highly virulent strains of the bacteria in the wild (Botzler 1991). Regardless, the disease is now firmly entrenched in the wild bird reservoir, particularly among waterfowl and pelagic seabirds (Crawford et al. 1992, Wobeser 1992, Österblom et al. 2004, Samuel et al. 2005, Blanchong et al. 2006a, Waller and Underhill 2007).

Avian cholera has been known to circulate in common eider populations that breed in temperate regions of Europe and North America since at least the 1960s (Reed and Cousineau 1967, Korschgen et al. 1978, Swennen and Smit 1991, Christensen et al. 1997, Pedersen et al. 2003, Québec Management Plan for the Common Eider 2004). A number of characteristics make the species particularly vulnerable to infection. First, common eiders are colonial breeding and highly gregarious during winter (Christensen 2008). Females are largely capital breeders and experience significant mass loss and reduced immune function during incubation (Hanssen et al. 2005). Most outbreaks of avian cholera in eiders have been observed on breeding colonies where nesting females are disproportionately impacted. The origin of *P. multocida* in northern common eiders remains uncertain (Harms 2012). Arctic-breeding eiders tend to have smaller clutches and face more variable environmental conditions than temperate-breeding eiders (Iles et al. 2013a). Latitudinal variation in survival and fecundity rates suggest that populations on the northern periphery of the species range are highly dependent upon adult survivorship to maintain population stability (Wilson et al. 2007, Descamps et al. 2012, Wilson et al. 2012).

My research objectives were to identify the current distribution of the avian cholera epidemic affecting northern common eiders, to quantify transmission dynamics and factors associated with disease spread, and to evaluate the demographic and population impact as modulated by ancillary threats and the host species' ecology and life history characteristics.

In my first data chapter (**Chapter 2**), I use mortality incidence data collected from a location of long-term monitoring (Mitivik Island, Nunavut) to infer key epidemiological

parameters (epidemic curves,  $R_0$ , and generation interval). I interpret variation in these parameters over the course of a multi-year epidemic in relation to concurrent changes in host abundance and mortality rates to identify factors regulating epidemic severity and the conditions governing epidemic fade-out.

In **Chapter 3**, I report results of extensive site investigations undertaken with the assistance of Inuit harvesters across a vast area in the Hudson Strait region. Prior to my research, limited information had been collected or shared about the geographic distribution, host network, and severity of outbreak events. I integrated these data with information concerning the fundamental niche (e.g., habitat and abiotic conditions), aggregation patterns (e.g., colony size and density), and propagation factors (e.g., site location in relation to the migratory flyways of potential carriers) of common eiders to parameterize a species distribution model (MAXENT) which I use to identify ecological correlates of outbreak risk.

Other limiting factors influence common eider population dynamics and in **Chapter 4**, I examine the implications of a profound shift in predator-prey dynamics. Specifically, advancing sea ice melt in Arctic waters is forcing polar bears (*Ursus maritimus*) to come ashore much earlier than in decades past. Polar bears are opportunistic foragers and appear to be making greater use of terrestrial prey to offset nutritional shortfalls experienced as a consequence of reduced foraging opportunity for seals and other marine mammals. One such resource is the eggs of colonial nesting birds. My analyses quantify temporal trends in bear incursions on two long-term bird monitoring sites and spatially across the Hudson Strait region. I quantify rates of nest destruction in relation to the prevalence of bear sign and in comparison to losses incurred

from customary egg predators such as foxes (*Vulpes spp.*) and gulls (*Larus spp.*). I relate these patterns to changes in ice conditions and other ecological attributes of eider colonies – demonstrating a directional change associated with altered climatic conditions beyond the normal scope of annual variability.

These issues are treated together in **Chapter 5**. In Chapter 5, I use mark-resight, nest monitoring, and breeding pair abundance data collected at Mitivik Island to quantify variation in common eider vital rates and population growth. I assess patterns of annual variation in relation to avian cholera exposure, winter harvest, predation risk, and summer weather conditions. This enables me to measure the importance disease in comparison to other limiting factors and to assess the prospect for demographic recovery in relation to interacting ecological stressors.

Finally, in my general discussion (**Chapter 6**), I summarize key findings and use my inferences to consider the appropriateness of traditional population assessments tools when multiple ecological stressors are at play. Disease has unique dimensions. Artic ecosystems have unique attributes. I comment on how the detailed types of information that I collected feeds into scientific understanding of the processes regulating population dynamics and how lessons learned apply more generally when the attributes of species of conservation or economic concern vary broadly.

## Chapter 2: Transmission dynamics and mortality patterns associated with an emerging infectious disease in an Arctic-nesting migratory bird

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### ABSTRACT

To predict and mitigate the impacts of disease on wildlife populations, a quantitative understanding of transmission potential, virulence and the factors regulating epidemic take-off and fade-out is required. In this study, I examine the mortality patterns of newly emerged avian cholera outbreaks affecting northern common eiders (*Somateria mollissima borealis*) in the Canadian Arctic. Specifically, I utilize incidence data collected on an eider breeding colony subject to long-term monitoring to infer key epidemiological parameters (epidemic curve, reproductive number  $R_0$ , and generation interval) and to evaluate changes in these parameters over the course of a multi-year epidemic. I supplement my analyses with information about concurrent changes in host abundance and mortality rates to assess population vulnerability to disease-induced losses. Consistent with expectations for a novel pathogen, case incidence increased in an exponential fashion during the initial wave of invasion [ $R_0 = 2.5$  (95% CI: 2.2 to 2.8)]. Recurrent outbreaks were documented during eight consecutive breeding seasons (from 2005 to 2012), during which time >6000 eider mortalities were observed. The real-time reproductive number,  $R_t$ , predictably declined as the epidemic progressed; however, prolonged low-frequency transmission ( $R_t < 1$ ) was evident, which is a hallmark of disease persistence

facilitated by environmental transmission. Breeding pair abundance declined by nearly 50% over the course of the epidemic with little evidence for compensatory reproduction in the years immediately following the epidemic peak. However, the pattern of epidemic fade-out, which was gradual and occurred with a robust population remaining intact (>4000 breeding pairs) is indicative of herd immunity, as opposed to host mortality, as the primary driver of pathogen extinction.

## **INTRODUCTION**

Disease can be an important regulator of population and community dynamics (Daszak et al. 2000, Dobson and Foufopoulos 2001, Holt and Boulinier 2005, Hudson et al. 2006). Emerging infectious diseases are a particular concern in biological conservation because novel pathogens have the potential to spread rapidly in immunologically naïve hosts (Daszak et al. 2000, Smith et al. 2009). Predicting the effects of an emerging pathogen on population abundance requires a quantitative understanding of transmission potential, virulence, and whether individuals that survive infection acquire either temporary or lasting immunity (Anderson and May 1979, Anderson and May 1991, Keeling and Rohani 2008, Smith et al. 2009). These variables interact and can be difficult to study in the wild, particularly in free-ranging, migratory species that utilize different habitats throughout their annual cycle (Altizer et al. 2006, Altizer et al. 2011, Peel et al. 2014).

Epidemiological theory generally assumes that the densities of both hosts and pathogens are regulated by their interaction (Anderson and May 1979). For directly transmitted

pathogens, epidemic fade-out is predicted to occur when host numbers drop below the critical threshold required to sustain transmission within a population (de Castro and Bolker 2005, Lloyd-Smith et al. 2005). Following a prolonged epidemic, potentially susceptible individuals can be depleted through their infection and subsequent mortality or immunized recovery and the presence of either or both modes of removal have very different implications for species conservation (Haydon et al. 2002). The primary concern with novel pathogen invasions is that transmissibility and virulence patterns are such that epidemics reduce host populations below the minimum viable size necessary to buffer them against extirpation due to demographic stochasticity or Allee effects (McCallum and Dobson 1995, de Castro and Bolker 2005).

The real-time reproductive number ( $R_t$ ; defined as average number of secondary infections per primary infection) is a key parameter linking epidemiological theory with population dynamics (Forsberg White and Pagano 2008, Obadia et al 2012). During the initial stages of an epidemic,  $R_t$  approximates the basic reproductive number ( $R_0$ ), which functions as a predictive criteria for successful ( $R_0 > 1$ ) or unsuccessful ( $R_0 < 1$ ) invasion, as well as a metric for the maximum potential growth rate of new infections in population wholly comprised of susceptible individuals. As an epidemic progresses,  $R_t$  will diverge from  $R_0$  as the proportion of individuals that remain susceptible to infection ( $s$ ) declines, with basic and real-time reproductive numbers related by the quantity  $R_t = R_0 s$ . When  $R_t < 1$  epidemic fade-out is predicted to follow (Gay 2004). Given prior knowledge of the generation interval between successive cases in a transmission chain,  $R_t$  is estimable from incidence data (Obadia et al 2012). By interpreting changes in  $R_t$  over time and in relation to concurrent changes in host

population parameters, critical insights may be gleaned about the conditions governing epidemic fade-out, including herd immunity, and the vulnerability of local populations to catastrophic disease-induced losses (Lloyd-Smith et al. 2005).

In this study, I examine a new series of avian cholera outbreaks affecting northern common eiders in the Canadian Arctic (*Somateria mollissima borealis*). Avian cholera is a contagious disease of birds caused by the infectious bacterium *Pasteurella multocida* (Botzler 1991, Samuel et al. 2007). The disease has undergone a dramatic expansion in its host range and geographical distribution in North America during the past century (Hubálek 2004, Samuel et al. 2007). It is known to cause significant mortality in affected bird populations (Crawford et al. 1992, Botzler 2002, Kwon and Kang 2003), including populations of common eiders nesting in temperate regions of North America (Reed and Cousineau 1967, Korschgen et al. 1978, Québec Management Plan for the Common Eider 2004) and Europe (Swennen and Smit 1991, Christensen et al. 1997, Pedersen et al. 2003).

Common eiders are a highly gregarious, migratory sea duck (Goudie et al. 2000). The northern subspecies is an important food resource for Inuit hunters in Canada and Greenland (Gilliland et al. 2009) and eider down, which is collected from nests and which is a valuable economic commodity (Bédard et al. 2008). The outbreaks of avian cholera currently affecting northern common eiders appear to be without historical precedent in the Canadian Arctic (Henri et al. 2010, Harms 2012). Large-scale mass-mortality events have been observed on breeding colonies at several locations in the Hudson Strait region since 2004 (see Chapter 3).

These events have prompted concern regarding the threat that avian cholera poses to the viability of local breeding colonies (Descamps et al. 2012).

Here, I take advantage of a unique opportunity to examine transmission dynamics and demographic impacts of an emerging infectious disease over the course of a multi-year epidemic from the time of disease onset to disease fade-out at a closely monitored study site. Using longitudinal incidence data derived from directly observed eider mortality events, I estimate seasonal epidemic curves. From these curves, I infer  $R_0$  during the initial wave of *P. multocida* invasion and track changes in  $R_t$  over sequential breeding seasons. I supplement these data with concurrent information about eider arrival and departure timing, trends in abundance, and mortality rates of nesting females. This enables me to quantify population impact and the status of the host population at epidemic fade-out. I interpret my results in relation to their implications for population vulnerability and the efficacy of potential management interventions.

## **METHODS**

### **Study system**

#### ***Mitivik Island***

I conducted my research on Mitivik Island (64.030 N, 81.789 W), located within the East Bay Migratory Bird Sanctuary, Southampton Island, Nunavut (Figure 2.1). Mitivik Island is a small (24 ha), rocky islet characterized by abundant tundra vegetation and the presence of several,



**Figure 2.1.** Map of the study area.

*Map of the study area with Mitivik Island denoted by the black star. Base map courtesy of Environment Canada, Canadian Ice Service.*

shallow freshwater ponds, which eiders use when accessing the colony and as a source of drinking water (Buttler 2009). The island supports over 4000 common eider breeding pairs making it the largest known eider breeding colony in the Canadian Arctic (Environment Canada. 2012). The island also supports smaller numbers of nesting Canada geese (*Branta canadensis*) ( $N = 6$  to  $12$  pairs), brant geese (*Branta bernicla*) ( $N = 3$  to  $6$  pairs), king eiders (*Somateria spectabilis*) ( $N = 15$  to  $30$  pairs), and herring gulls (*Larus argentatus*) ( $N = 15$  to  $30$  pairs), as well as migrating lesser snow geese (*Chen caerulescens, caerulescens*), Ross's geese (*Chen rossii*) and northern pintails (*Anas acuta*).

### **Host ecology**

Northern common eiders migrate to Mitivik Island each spring from wintering areas off the coasts of Atlantic Canada and southwestern Greenland (Mosbech et al. 2006). The species breeding ecology is characterized by delayed reproductive maturity, high female natal philopatry, and durable pair bonds between successfully nesting pairs (Goudie et al. 2000). First reproduction is typically at two or more years of age (Baillie and Milne 1982, Hario and Rintala 2009); however, it is not uncommon to observe immature females on colonies prospecting for future nest sites. Males do not incubate eggs or tend to broods and typically leave the breeding grounds as soon as egg-laying is complete. In the Arctic environment, eider nesting phenology is tightly correlated with spring sea ice break-up (Love et al. 2010). Collectively, these traits result in a high degree of behavioral synchrony, spatial crowding, and the intensive use of a shared

environment by reproductive females, but substantially less contact involving males and immature birds during the nesting period.

### ***Avian cholera epidemiology***

Avian cholera is spread both directly via bird-to-bird contact and environmentally by the ingestion or aerosolized transmission of *P. multocida* in contaminated water or sediments. Infections produce respiratory and septicemic disease, often with a rapid onset of morbidity (Botzler 1991, Friend 1999, Samuel et al. 2007). Acute infections can result in bird death within 6–12 h of exposure, although 24–48 h is a more common timeframe (Friend 1999). Serological investigations have proven effective for detecting recent infections and vaccination studies suggest that at least short-term immunity is likely for surviving individuals (Samuel et al. 2003, Samuel et al. 2007). *P. multocida* is known to remain infective in wetlands for a period of days to months (Friend 1999, Samuel et al. 2007); however, it does not appear to overwinter in the environment (Samuel et al. 2004, Blanchong et al. 2006b). Instead, carrier birds are thought to play an essential role in the cross-seasonal perpetuation and geographic spread of disease (Samuel et al. 2005).

### **Field methods and data analysis**

#### ***Field data collection***

Environment Canada initiated a long-term monitoring program on Mitivik Island in 1996. Biologists are stationed on the island each summer (June to August) and collect a variety of

biotic and abiotic data. Prior to nest initiation, eiders are captured using flight nets and marked with U.S. Fish and Wildlife Service metal tarsal bands, as well as colored-plastic bands. There are eight observation blinds situated around the island, which enable biologists to monitor >80% of the available nesting habitat without disturbing the birds.

Since 2000, 5 permanent plots encompassing 14,383 m<sup>2</sup> of habitat has been monitored on an annual basis. Within these plots, the status of female eiders is determined during twice-daily scans. This allows estimation of nesting pair abundance, egg-laying and hatching dates, and nest attendance patterns. The scans also facilitate rapid detection of mortality events, which before avian cholera arrived were extremely rare. In addition, an end-of-season ground survey of the entire colony is conducted to corroborate visual estimates nest fates and during with the total number of carcasses on the island is estimated.

### ***Data analysis***

To quantify annual variation in common eider breeding pair abundance I divided the number of nest starts within each permanent monitoring plot by plot area to calculate mean density weighted by plot size. I then multiplied my year-specific estimates of density ( $\pm$  SE) by the total amount of available habitat on the colony (area = 19,440 m<sup>2</sup>; Buttler 2009) to derive annual indices of breeding pair abundance ( $N_i$ ).

To assess the extent of annual mortality, I used the end-of-season survey counts of female common eider carcasses ( $m_i$ ). I recognized that an unknown fraction of birds may have

died away from the colony and I consider my estimate a minimum measure of directly observable mortality (*minimum mortality* =  $m_i / N_i$ ).

To derive seasonal epidemic curves I aggregated female common eider mortality events occurring with permanent monitoring plots over 6-d intervals from 11 Jun to 28 Jul each year. The 6-d aggregation window enabled me to minimize data gaps, while the date range allowed me to encapsulate temporal variation in the appearance of new cases from the time of eider arrival through the median date of 50% departure (i.e., the date that half of all nesting females left the colony). To facilitate interpretation of the data I summarized female densities across the same 6-d aggregation intervals and fit my estimates with a Gaussian function (i.e., bell curve) to encapsulate the seasonal arrival, peak abundance, and departure of eiders from the colony.

I regarded the timing of disease onset relative to eider arrival as a relevant parameter because I expected greater infection probability to be associated with longer exposure. I interpreted the shape of the epidemic curve prior to female departure as an indicator of epidemic status, wherein a decline in case incidence (i.e., an asymptote in the growth curve) before migration would be interpreted as indication of host depletion, whereas continued exponential growth would be interpreted as a lack of significant depletion.

To derive annual estimates of  $R_t$  and generation interval I used the maximum likelihood estimation procedure proposed by Forsberg White and Pagano (2008) and coded for the statistical package 'R0' by Obadia et al. (2012) (function est.R0.ML, R version 3.1.0). Forsberg White and Pagano's method assumes that the epidemic curve follows a Poisson distribution.

Given the observation of  $C_0, C_1, \dots, C_t$  incident cases over consecutive units of time, the est.R0.ML algorithm finds the values of  $R_t$  and generation interval gamma distribution that maximize the log-likelihood of the observed data. Primary infections are assumed to appear before secondary infections and the model makes no assumptions with respect to mixing within the population.

Function arguments include: *epid*, the epidemic curve parameterized as a vector of new mortality events; *GT*, the generation interval distribution (referred to as generation time by Obadia et al. 2012); *t*, the vector of dates on which incidence was calculated; *begin* and *end*, specifying the start and end dates for estimation; and *time.step*, specifying the number of days between observations. The model can be implemented assuming a known generation interval distribution derived from contact tracing data or based on experimental study; or an unknown generation interval, which is the more common situation for emerging epidemics in the wild. For my analysis, I assumed an unknown generation interval distribution with prior mean ( $\mu$ ) and standard deviations ( $\sigma$ ) of 5.0 and 2.0, respectively, which were subsequently recycled to determine the maximum likelihood estimates.

## RESULTS

### Trends in breeding pair abundance and minimum mortality rates

Avian cholera mortality was confirmed on Mitivik Island by either serology or molecular genetic analysis during eight consecutive breeding seasons (2005 to 2012). Prior to the detection of *P. multocida* on the colony, eider numbers were increasing at a rapid rate, with abundance more

**Table 2.1.** Breeding pair number and annual mortality.*Common eider breeding pair numbers and sex related variation in mortality rates on Mitivik Island.*

| Year  | Breeding pairs numbers                          |                              | Directly observed female mortality |  | Directly observed male mortality |  |
|-------|---|------------------------------|------------------------------------|--|----------------------------------|--|
|       | Density $\pm$ SE<br>(pairs * ha <sup>-1</sup> ) | Total abundance<br>( $N_i$ ) | Number<br>( $m_{i, female}$ )      | Percent mortality<br>( $m_{i, female} / N_i * 100$ ) | Number<br>( $m_{i, male}$ )      | Percent mortality<br>( $m_{i, male} / N_i * 100$ ) |
| 2000  | 194 $\pm$ 46                                    | 3768                         | 1                                  | 0.0%   | 0                                | 0.0%   |
| 2001  | 248 $\pm$ 59                                    | 4829                         | 3                                  | 0.1%   | 0                                | 0.0%   |
| 2002  | 213 $\pm$ 27                                    | 4148                         | 0                                  | 0.0%   | 0                                | 0.0%   |
| 2003  | 288 $\pm$ 45                                    | 5606                         | 0                                  | 0.0%   | 0                                | 0.0%   |
| 2004  | 345 $\pm$ 75                                    | 6708                         | 19                                 | 0.3%   | 0                                | 0.0%   |
| 2005† | 442 $\pm$ 70                                    | 8592                         | 203                                | 2.4%   | 1                                | 0.0%   |
| 2006† | 453 $\pm$ 77                                    | 8813                         | 3200                               | 36.3%  | 30                               | 0.3%   |
| 2007† | 300 $\pm$ 57                                    | 5821                         | 781                                | 13.4%  | 15                               | 0.3%   |
| 2008† | 278 $\pm$ 38                                    | 5411                         | 1454                               | 26.9%  | 16                               | 0.3%   |
| 2009† | 201 $\pm$ 14                                    | 3901                         | 225                                | 5.8%   | 0                                | 0.0%   |
| 2010† | 236 $\pm$ 16                                    | 4582                         | 213                                | 4.6%   | 9                                | 0.2%   |
| 2011† | 233 $\pm$ 12                                    | 4525                         | 34                                 | 0.8%   | 0                                | 0.0%   |
| 2012† | 233 $\pm$ 22                                    | 4522                         | 12                                 | 0.3%   | 1                                | 0.0%   |
| 2013  | 285 $\pm$ 17                                    | 5545                         | 6                                  | 0.1%   | 0                                | 0.0%   |

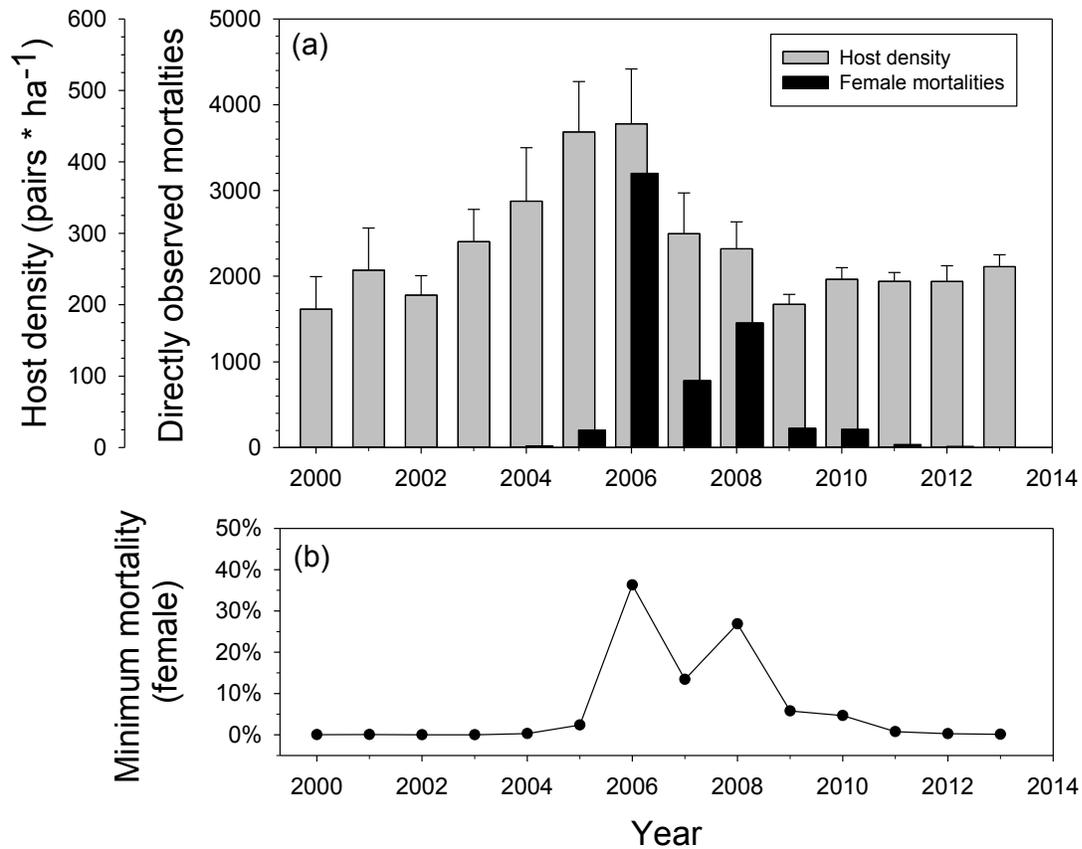
†Years when *P. multocida* was detected in tissue samples collected from common eiders.

than doubling between 2000 and 2005 to an estimated maximum of 8592 breeding pairs ( $442 \pm 70$  SE pairs \* ha<sup>-1</sup>) (Table 2.1). In 2005, 203 female common eider carcasses were encountered during the end-of-season ground survey (*minimum mortality* = 2.4%). Elevated mortality had been noted the previous year (19 female carcasses); however diagnostic testing was not performed to determine the cause.

Mortality peaked in 2006 when 3200 female carcasses were encountered (*minimum mortality* = 36.3%) and remained high in 2007 (781 female carcasses, *minimum mortality* = 13.4%) and 2008 (1454 female carcasses, *minimum mortality* = 26.9%). Following the epidemic peak, abundance declined by 55.7% over a 3-yr period, dropping to 3900 breeding pairs ( $201 \pm 14$  SE pairs \* ha<sup>-1</sup>) by the start of the 2009 nesting season.

Mortality steadily abated thereafter, dropping from 225 female carcasses in 2009 (*minimum mortality* = 5.8%) to 12 in 2012 (*minimum mortality* = 0.3%). 2013 was the first year after the original emergence that *P. multocida* was not detected on Mitivik Island. Between 2010 and 2013, breeding pair abundance remained stable, with no clear evidence of population recovery or decline (Figure 2.2).

Little male mortality was observed at any stage of the epidemic cycle (range: 0 to 30 male carcasses per year) (Table 2.1).



**Figure 2.2.** Trends in common eider abundance and mortality.

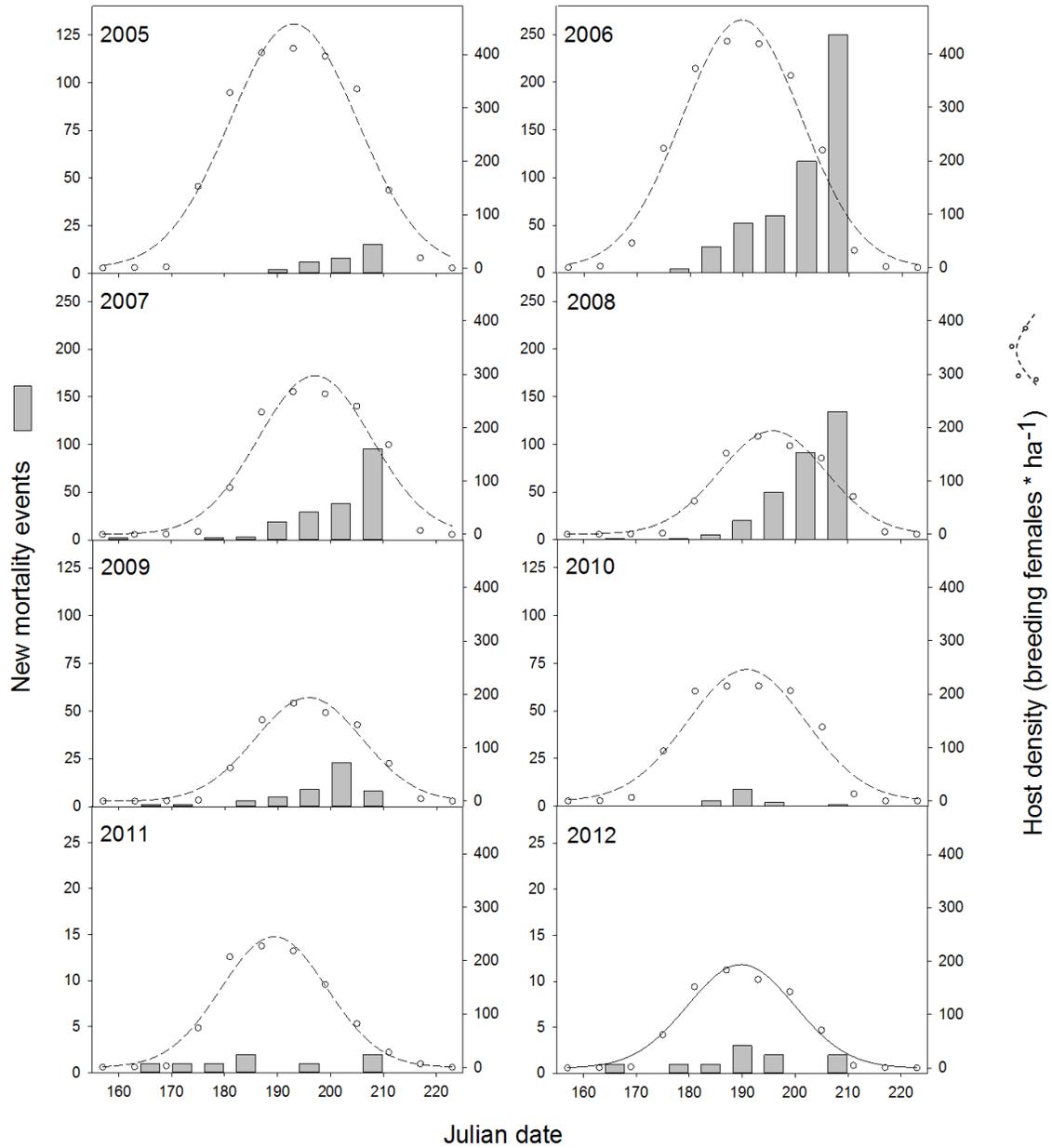
(a) Trends in common eider breeding pair density (grey bars) and female common eider carcasses counts (black bars); and (b) minimum mortality rate of females on Mitivik Island from 2000 to 2014.

## Reproductive numbers and generation interval

Case incidence derived from female mortalities in permanent monitoring plots increased at an exponential rate during each of the initial four outbreak years (2005 to 2008) (Figure 2.3).

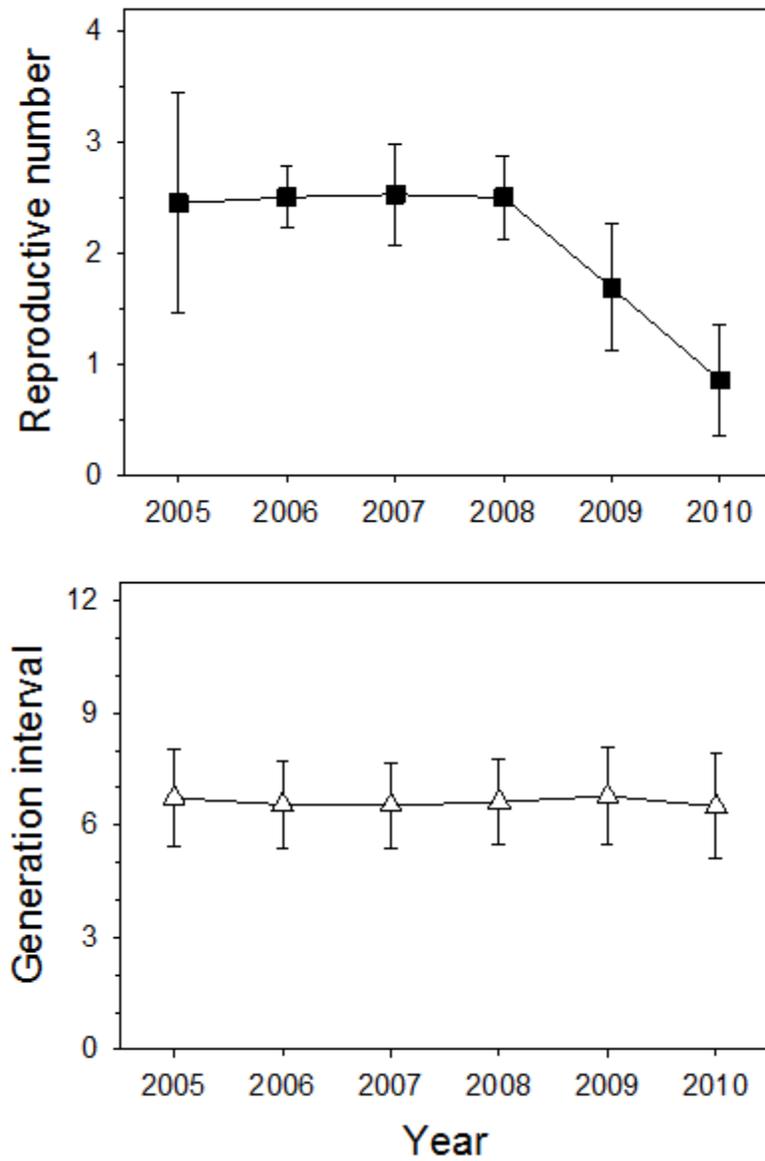
Disease onset (measured as the first mortality event of the season) was later in 2005 than in 2006-2008, offering a partial explanation for the lower overall mortality rate observed that year. Onset date was negatively correlated with total mortality during the initial years of the epidemic (Pearson's  $r = -0.81$ ), and over the epidemic cycle as a whole (Pearson's  $r = -0.61$ ). In both 2009 and 2010, case incidence peaked and was already in decline before the majority of female eiders departed from the colony. In 2011 and 2012, only sporadic mortality events were observed with no discernable increase in temporal incidence suggesting a lack of epidemic take-off.

Similar values for the reproductive number and the generation interval were estimated during the initial outbreak years (2005 to 2008; Figure 2.4). The greatest precision was obtained for the 2006 outbreak data [ $R_t \approx R_0 = 2.5$  (95% CI: 2.2 to 2.8); generation interval distribution  $\mu = 6.6$  and  $\sigma = 1.2$  d]. As the epidemic progressed,  $R_t$  declined, while the generation interval remained unchanged (Figure 2.4). By 2010,  $R_t$  dropped below the replacement threshold necessary for persistence of directly transmitted pathogens in a single host. Transmission parameters could not be estimated in 2011 or 2012 because too few cases were available for calculation; however, the prolonged, low-frequency occurrence of mortality events with  $R_t < 1$  suggests environmental transmission as the dominate mode of pathogen persistence at the epidemic tail.



**Figure 2.3.** Host density and temporal occurrence of mortality events.

*Epidemic curves depicting aggregated case incidence data derived from directly observed female eider mortality (grey bars) and estimates of breeding female density (open circles and fitted dashed line).*



**Figure 2.4.** Real-time reproductive number and generation interval.

*Variation in the real-time reproductive number ( $R_t$ ) and generation interval estimates as the avian cholera epidemic progressed on Mitivik Island, Nunavut.*

## DISCUSSION

Emerging infectious diseases are a growing concern in biological conservation because they can act on their own or in concert with other ecological drivers to reduce population size, hinder the recovery of threatened and endangered species, and substantively contribute to species extinction (Daszak et al. 2000, Lafferty 2003, Smith et al. 2009). Many high latitude species are thought to have evolved with limited or no exposure to infectious pathogens, which potentially reduces their innate immune capabilities in comparison to species with longer co-evolutionary histories (Acevedo-Whitehouse and Cunningham 2006, Meyer-Lucht and Sommer 2009). In addition, the specialized life history adaptations that enable high latitude species to persist under challenging environmental condition can leave them with limited capacity to compensate demographically when novel threats to survival arise (Hochberg et al. 1992, Agnew et al. 2000).

The avian cholera epidemic that occurred on Mitivik Island was severe for a species that is highly reliant on adult survivorship to maintain population stability. Overall, > 6000 female northern common eiders were directly observed to have died over the course of the 2005-2012 epidemic. While the total number of mortalities was higher than has been reported for eiders in more temperate regions, percent mortality was not different. I estimated a minimum mortality rate, based on the direct recovery of female carcasses of 36% in 2006 and a nearly 50% decline in breeding pair abundance from time of disease onset to disease fade-out. Similar estimates using the same estimation procedure have been reported in the St. Lawrence Estuary of Québec (17-25% annual mortality) (Reed and Cousineau 1967, Québec Management Plan for the Common Eider 2004), while higher percent mortality has been reported on some colonies

in Western Europe (35-95% annual mortality on affected colonies) (Swennen and Smit 1991; Christensen 1996, Pedersen et al. 2003). Thus, despite fears that a lack of previous exposure to *P. multocida* could lead to more virulent outbreaks, my findings suggest this has not been the case at Mitivik Island or other locations in the Hudson Strait region (Chapter 3).

Interestingly, genetic analyses have concluded that the *P. multocida* strains affecting European common eiders are closely related to isolates obtained from back-yard poultry, suggesting recent pathogen exchange between domestic and wild bird hosts (Christensen et al. 1998, Pedersen et al. 2003). Inter-epidemic intervals have been irregular in Europe and mass-mortality events have been reported from relatively few locations (Christensen et al. 2008). In contrast, avian cholera outbreaks affecting American common eiders in Atlantic Canada and the northeastern United States have been characterized by recurrent, multi-year epidemics, with a larger number of colonies affected (Québec Management Plan for the Common Eider 2004). Genetic analyses of North America isolates, including isolates from the Canadian Arctic, have not demonstrated the same close relationship between strains from wild and domestic birds (N. J. Harms, University of Saskatchewan, *unpublished doctoral dissertation*). Collectively, these patterns suggest that *P. multocida* is more entrenched in the wild bird reservoir in North America than Europe, but also potentially less virulent.

$R_0$  is a summary measure of transmissibility that depends on the characteristics of the infectious agent (e.g., infectivity, virulence and the duration of infectiousness) and host population (e.g., population density, seasonal movement behavior, and social mixing patterns). As such,  $R_0$  may differ among outbreak events and between species exposed to the same

pathogen. Nonetheless, the parameter does provide a generalizable approximation of transmission potential that is useful for predicting disease dynamics and developing control strategies. My estimate of  $R_0 \approx 2.5$  is comparable to estimates for other highly virulent microparasites with a similar mode of mixed direct and environmental transmission. For example, in a study of avian influenza transmission in experimentally inoculated turkeys, Saenz et al. (2012) found that highly pathogenic strains (HPAI) have a much shorter infectious period than low pathogenicity strains (LPAI) due to the rapid onset of mortality in the former. However, infectivity (measured in terms of infection rates per unit time) was similar for the high pathogenicity and low pathogenicity strains. These difference resulted in a much lower basic reproductive number for HPAI ( $R_0 = 3.0$ ) compared to LPAI ( $R_0 = 15.3$ ). For human cholera, caused by the bacterium *Vibrio cholerae*,  $R_0$  has been estimated in range of 1.1 to 2.7 in field studies, with considerable variation in the relative contributions of environmental and direct transmission among outbreak locations (Mukandavire et al. 2011, Mukandavire et al. 2013).

Epidemiological theory suggests that direct transmission is enhanced by increasing host density through a combination of elevated contact rates and stress-induced reductions in the ability of individuals to resist infection (Lafferty and Kuris 2005, Wobeser 2007). Nesting densities are not commonly reported for eiders; however Mitivik Island is small in size and supports the largest known common eider breeding aggregation in the Canadian Arctic (Environment Canada 2012). There is some empirical evidence to support density-dependent variation in avian cholera infection probability in colonial nesting lesser snow geese (Samuel et al. 1999) and it is worth noting that the epidemic on Mitivik Island occurred after a period of

population increase. However, the initial source of pathogen in the population remains a mystery, as do the determinants of virulence among different *P. multocida* strains isolated from birds on Mitivik Island (N. J. Harms, University of Saskatchewan, *unpublished doctoral dissertation*).

When both direct and environmental modes of transmission are present, disease modelling suggests that direct transmission governs the timing and size of the epidemic peak, whereas environmental transmission plays the more prominent role during epidemic take-off and fade-out (Rohani et al. 2009). One of the hallmarks of environmental transmission is a prolonged epidemic tail with  $R_t < 1$ . The implication from a conservation perspective is that such diseases are more difficult to eradicate and prone to reemergence when immunologically naïve hosts enter the population. Because common eiders delay first reproduction for two or more years, I speculate that the arrival of new recruits contributed to the multi-year persistence on Mitivik Island. Less clear is the role of males in disease spread. I observed relatively few male mortality events; however, detectability issues are difficult to address using direct recovery data alone. In Chapter 5, I address this issue and assess the relative impact of avian cholera mortality on males and females using a capture-mark-resight model of survival and return rates.

Despite the fact that epidemic size, duration, and inter-epidemic interval may be acutely sensitive to the demography and immunological status of host populations, relatively few conservation-oriented assessments of disease risk explicitly characterized disease transmission as a dynamic process influenced by the interaction between host and pathogen numbers

(Haydon et al. 2002, Gerber et al. 2005). On the basis of the severe mortality rates measured at Mitivik Island during the initial wave of avian cholera exposure (2006 -2008), Descamps et al. (2012) posited a high probability of quasi-extinction (defined as a population pushed below the critical abundance threshold where extinction is more likely to occur than not due to demographic stochasticity or Allee effects). However, the authors arrived at their conclusion without consideration of herd immunity as a regulating factor of transmission dynamics. I interpret the observation that avian cholera mortality remained detectable for many years after the initial wave of invasion without explosive outbreaks occurring and that disease conditions abated with a robust population (>4000 breeding pairs) remaining intact as strongly indicative of an acquired immune response on the part of individuals that survived infection, as opposed to host depletion via mortality alone. Ideally, pathogen and antibody prevalence would be tracked concurrently to determine the dynamics of infection and immunity in hosts; however, time series monitoring of these parameters is often infeasible in the wild (Gilbert et al. 2013) and in the case of avian cholera the development of diagnostic tests remains an active topic of research (Petersen et al. 2001, Blehert et al. 2008, N. J. Harms, University of Saskatchewan, *unpublished doctoral dissertation*).

Assessing risks to population stability is an integral part of conservation management and a prerequisite for deciding if, when, and how management action should be taken to mitigate disease impacts (Haydon et al. 2002, Gerber et al. 2005, Oli et al. 2006). Although the mortality rates of northern common eiders from avian cholera were not any more severe than has been reported in temperate-breeding eiders, northern populations likely have less capacity for

demographic recovery following large-scale perturbations owing to their smaller clutch sizes and more variable rates of nesting success and juvenile survival in the Arctic environment (Goudie et al. 2000, Descamps et al. 2012). These factors, coupled with the species importance as a subsistence resource for Inuit harvesters emphasizes the need to mitigate disease impacts when possible. While poultry are routinely vaccinated against *P. multocida* infection (OIE 2008), the logistics of vaccination delivery would be extremely difficult. An alternative approach, which takes advantage of the species life history characteristics and migratory behavior, would be to destroy the nests of incubating females at the first sign of avian cholera mortality and thereby precipitate nest abandonment and dispersal. Northern eiders typically do not typically re-nest when their eggs are depredated by predators and nest destruction could effectively move birds from a location of high disease transmissibility (on colonies) to low transmissibility (marine habitats). Doing so would potentially reduce pathogen prevalence in the population and increase the probability of pathogen extinction during the non-breeding season. However, this must be balanced against the possibility that efforts to control transmission could be counterproductive if they only hinder natural selection for disease resistance (Staszewski and Boulinier 2004, Foster et al. 2007).

## Chapter 3: Spatial distribution and ecological drivers of avian cholera emergence in the eastern Canadian Arctic

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### ABSTRACT

Understanding the influence of host ecology on the transmission dynamics and spread of infectious pathogens is fundamental to predicting and controlling disease in wildlife populations. Avian cholera, caused by the bacteria *Pasteurella multocida*, is major disease of birds and has an expanding global distribution and host range. In this study, I describe the ecological characteristics of outbreak events of avian cholera, newly occurring at northern common eider (*Somateria mollissima borealis*) breeding colonies in the eastern Canadian Arctic. My methods include detailed site investigations of outbreak events on offshore islands strewn over >1000 km of coastline in the Hudson Strait region of Nunavut and northern Québec (Nunavik) and the use of a presence-only species distribution model (MAXENT) to identify ecological predictors disease occurrence. The site investigations were undertaken with the assistance of Inuit harvesters and provided evidence for mass-mortality events occurring on at least 13 eider breeding colonies between 2004 and 2013. Seven species, including common eiders, were implicated in the die-offs. Percent mortality ranged from 1 to 43% of nesting females at individual outbreak sites and >1200 eider mortalities were reported in total. Habitat attributes and host factors were both important determinants of outbreak probability. Specifically, I found that avian cholera occurrence was positively correlated with percent vegetative cover, as well as the intensity of flyway use by eiders affiliated with wintering areas

in Atlantic Canada – a potential source of the pathogen. Colony size and the number of freshwater ponds available per eider (a measure of host crowding in shared wetlands) were also significant predictors of outbreak risk. My results are relevant for predicting *P. multocida* transmission dynamics, host networks, and both barriers to and opportunities for further spread of avian cholera within the Arctic coastal bird community.

## **INTRODUCTION**

Emerging infectious diseases are appearing at an unprecedented rate and pose a growing threat to global biodiversity (Daszak et al. 2001, Plowright et al. 2008, Smith et al. 2009). A poleward expansion of disease-causing pathogens has been predicted in association with the effects of globalization and climate change (Harvell et al. 2002, Altizer et al. 2013) and wildlife in high latitude ecosystems may be particularly vulnerable to novel pathogen invasions (Gardner et al. 1997, Kutz et al. 2004, Kutz et al. 2005, Kerry and Riddle 2009). Understanding the ecological factors that underlie spatial patterns of disease emergence is crucial for predicting risk and developing management and conservation strategies to mitigate disease impacts in threatened and/or economically important species (Smith et al., 2002, Jones et al. 2008, Liu et al. 2013).

Species distribution models are an increasingly popular tool for evaluating and predicting the distribution of emerging pathogens and invasive species (Guisan and Thuiller 2005, Kearney and Porter 2009, Elith et al. 2011). In a typical formulation, occurrence records are assessed in relation to environmental information collected from the same sites to identify the ecological

correlates of occurrence probability and then resulting algorithms are used to predict distributions in unsampled regions or under future landscape or climate scenarios. Most species distribution models are based on climatic and habitat variables and emphasize factors associated with fundamental niche of a species; however, organismal interactions and factors influencing dispersal also can be important drivers of biological invasions (Thuiller et al. 2005, Václavík and Meentemeyer 2012, Liu et al. 2013). This focus is particularly important when evaluating the geographic spread of infectious diseases in animals, for which factors such as host densities, seasonal movements, and interspecific interactions are key determinants of transmission (Altizer et al. 2006, Carver et al. 2009, Gaidet et al. 2012).

In this study, I describe outbreak characteristics and use a species distribution model to examine the ecological drivers of avian cholera emergence in the eastern Canadian Arctic. Avian cholera is a major disease in birds caused by the bacterium *Pasteurella multocida* (Botzler 1991, Friend 1999, Samuel et al. 2007). The disease has been known to circulate in domestic birds for over 200 years and has a near-global distribution in poultry flocks (Rimler and Glisson 1997). Reports of avian cholera mortality involving wild birds have proliferated in recent decades, particularly among migratory waterfowl (Hubálek 2004). Recently, avian cholera has been reported on the nesting colonies of northern common eiders (*Somateria mollissima borealis*) in the Hudson Strait region of the Canadian Arctic. This is an area far from other locations where the disease previously has been reported and its origins remain a mystery for this region (Harms 2012).

Common eiders are an important subsistence resource for Inuit in Canada, who hunt the birds for their meat and visit nesting colonies during the breeding season to collect feather down for use in clothing and bedding (Bédard et al. 2008, Gilliland et al. 2009). Traditional Ecological Knowledge interviews indicate that mass-mortality events of the type currently being observed are without precedent in the cultural memory of Inuit harvesters (Henri et al. 2010). High mortality rates during the initial wave of disease invasion, coupled with the recurrent nature of the outbreaks in some locations, have raised concern over the threat that avian cholera might pose to the viability of local populations (Buttler 2009, Descamps et al. 2012).

Several non-mutually exclusive hypotheses have been advanced as ecological drivers of avian cholera transmission in regions where the disease is prevalent. Outbreaks are often highly explosive and unpredictable in waterfowl and other wetland bird species, which has been interpreted as indication of simultaneous exposure of susceptible hosts via a common source of infection, most likely contaminated water (Botzler 1991, Wobeser 1992, Blanchong et al. 2006b). Large outbreaks are often associated with dense aggregations of birds and involve multiple species (Blanchong et al. 2006a, Samuel et al. 2007). In some cases, disease occurrence has been associated with the arrival of specific species at seasonal habitats where many birds congregate, such as the arrival of lesser snow geese (*Chen caerulescens caerulescens*) on wintering and migratory stopover sites in the Central and Pacific Flyways of North America (Samuel et al. 2005, Blanchong et al. 2006a). However, small outbreaks affecting a single species also have been reported, which is suggestive of sublethal infection and dispersal by carrier birds (Botzler et al. 1991, Wobeser 1992, Samuel et al. 2007). Determining the primary

mode of transmission, biophysical drivers of epidemic take-off, and the role of different host species within a community in the perpetuation and spread of disease are critical steps for evaluating disease risks and identifying potential mitigation strategies (Gerber et al. 2005).

I had two principle research objectives in this study. The first was to compile and describe avian cholera outbreak information and thereby delineate the current extent of the epidemic in the eastern Canadian Arctic, the species affected, and the magnitude of mortality. My second objective was to identify shared ecological and environmental characteristics of outbreak sites as a basis for disease surveillance and developing mitigation strategies. Specifically, I use a presence-only species distribution model (MAXENT; Phillips et al. 2006) to assess the predictive value of a candidate set of explanatory variables describing the fundamental niche of nesting eiders, their aggregation patterns, and the distribution and dispersal behavior of eiders and other putative hosts. I interpret my results in relation to their implications for transmission dynamics, the role of various members of the coastal bird community as victims or vectors of disease spread, and the potential for further spread within the Arctic coast bird community.

## **METHODS**

### **Host and disease ecology**

Avian cholera is known to affect more than 180 bird species representing at least 47 different families of bird worldwide (Samuel et al. 2007). *P. multocida* infections occur via bird-to-bird contact and environmentally through the ingestion or inhalation of aerosolized bacteria. The main source of new infections in the wild is thought be from bacteria shed in the feces and

nasal discharge of live hosts or released from the carcasses of diseased birds (Botzler et al. 1991). *P. multocida* can remain infective in water or sediments for a period of days to months allowing for prolonged indirect transmission outside avian hosts (Friend 1999). However, available evidence suggests that the bacterium does not overwinter in wetland ecosystems (Samuel et al. 2004, Blanchong et al. 2006b); rather, carrier birds are thought to play an essential role in the cross-seasonal persistence and geographic spread of disease (Samuel et al. 2005, Samuel et al. 2007).

Northern common eiders migrate to the Hudson Strait region each spring from wintering areas off the coasts of Atlantic Canada and southwestern Greenland (Goudie et al. 2000). They nest predominately on small, nearshore islands where breeding pair densities can exceed 500 pairs per hectare (Robertson and Gilchrist 1998). The largest colonies tend to be on islands with abundant vegetation and small freshwater melt ponds, which eiders use as a drinking source. These characteristics give rise to high degree of phenological synchrony, crowding, and a potentially heightened risk of avian cholera transmission between birds using a shared water source.

The first avian cholera outbreaks to be detected in the Hudson Strait region occurred in 2004 when Inuit harvesters reported mass-mortality events on northern common eider colonies in Digges Sound, near Ivujivik, Québec (Gaston 2004, Kwan 2004). The same year, elevated mortality of eiders was observed at Mitivik Island, Nunavut, which is a long-term bird monitoring station administered by Environment Canada (Buttler 2009). A larger outbreak occurred on Mitivik Island in 2005 and the presence of *P. multocida* was confirmed by

laboratory analysis (Buttler 2009, Harms 2012). Repeat outbreaks occurred on Mitivik Island on an annual basis from 2005 to 2012, while sporadic, mostly uninvestigated reports of avian cholera mortality have been reported in other Arctic locations. The ecological conditions at outbreak sites other than Mitivik Island have not been evaluated, hampering investigation of avian cholera epidemiology and spread.

Several alternative hypotheses have been advanced for the source of avian cholera in northern common eiders and co-mingling Arctic species (Harms 2012). The disease has circulated in temperate-breeding American common eiders (*S. m. dresseri*) in Atlantic Canada and the northeastern United States since at least the 1960s (Gershman et al. 1964, Korschgen et al. 1978, Québec Management Plan for the Common Eider 2004). *S. m. borealis* and *S. m. dresseri* populations are widely separated during the breeding season; however they have partially overlapping ranges in Atlantic Canada during winter. Lesser snow geese (*Chen caerulescens caerulescens*) also are known carriers of avian cholera (Samuel et al. 2005) and have an overlapping breeding range with northern common eiders during summer. Seasonal and geographic patterns of avian cholera mortality are known to track lesser snow goose migration routes (Friend 1999, Samuel et al. 2007) and avian cholera mortality has been observed at lesser snow goose breeding colonies in the western Canadian Arctic (Samuel et al. 1999).

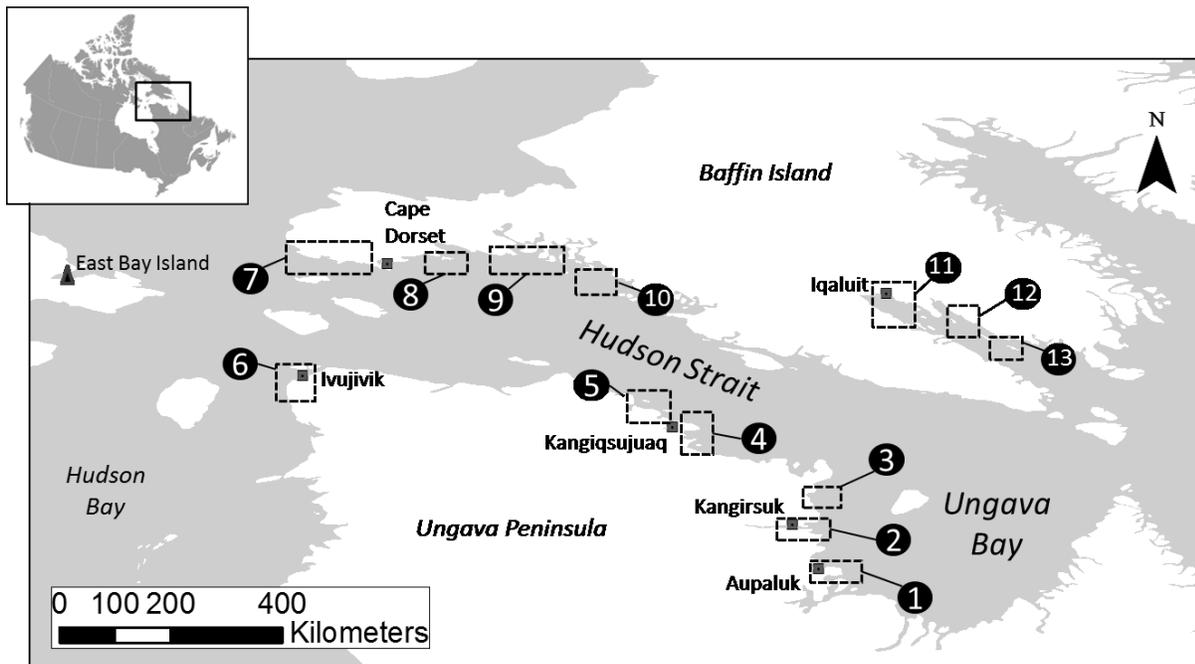
Large-bodied gulls (*Larus* spp.) are also known to harbor *P. multocida* infections (Canadian Cooperative Wildlife Health Centre 2007) and are commonly encountered on common eider breeding colonies. While gulls are an unlikely cross-seasonal reservoir for *P. multocida*, they

have been proposed as an important intermediary in the transmission of avian diseases within avian communities (Arnal et al. 2014).

### **Site investigations**

I selected six Inuit communities in Nunavut and northern Québec (Nunavik) to conduct my research: Cape Dorset, Iqaluit, Aupaluk, Kangirsuk, Kangiqsujuaq, and Ivujivik. Within the vicinity of these communities I delineated 13 survey zones for intensive study (Figure 3.1). The survey zones were chosen on the basis of avian cholera outbreak reports made by Inuit harvesters, their locations in relation to known concentrations of breeding common eiders, and to provide broad geographic coverage of the study area. Prior to conducting field work, I made community visits to consult with local harvesters. There, I presented posters that included photographic images of birds infected with *P. multocida*, as well written materials translated into Inuktitut soliciting details about die-off events.

In most cases, the islands that I investigated were locations where breeding bird surveys had previously been conducted by Canadian Wildlife Service biologists (Table 3.1). The historical surveys prioritized monitoring trends in abundance on islands identified as suitable habitat for nesting eiders (<5 km<sup>2</sup> in area; 0.5 to 10 km from nearest mainland shore; elevation <50 m) (Chapdelaine et al. 1986, Robertson and Gilchrist 1998). Site selection protocols differed among the teams initially implementing the research. In some areas site selection was based on random draws of islands with suitable habitat (survey zones 1-3, 7 and 9-10), while in other



**Figure 3.1.** Map of the study area.

*Numbered boxes indicate survey zones and triangle represents the location of the Mitivik Island long-term study site.*

areas comprehensive sampling was undertaken under an objective of >80% coverage within pre-defined study areas (survey zones 4-6, 8 and 11-13).

Site investigations were conducted over four breeding seasons: 10-26 July 2010, 6-19 July 2011, 8-21 July 2012, and 7 to 24 July 2013. Islands were accessed by boat and searched on foot by 3-8 people walking 10-25 m apart in successive linear sweeps. On each island the species identity and number of nesting birds present was recorded, as well as the identity and minimum number of birds unassociated with identifiable nests within a 5 km radius of each island. The biophysical attributes of the islands were also recorded, including percent vegetative cover and the number of ponds >10 m<sup>2</sup> in size. All of the islands were geo-referenced to facilitate integration of remotely-sensed data with field measurements.

With the help of Inuit assistants, I collected the fresh carcasses or the skeletal remains of any birds that were encountered during the surveys. Samples were preserved on ice and shipped to a Canadian Cooperative Wildlife Health Center (CCWHC) laboratory for diagnostic testing. The methodology and results of the diagnostic testing is the subject of a separate research dissertation (N. J. Harms, University of Saskatchewan, *unpublished doctoral dissertation*). Here, I present and use summary results.

For classification purposes, I rated the evidence of avian cholera presence on colonies into three categories:

- (1) *Confirmed outbreak*— indicating that a mass-mortality event involving 20 or more birds was reported by an Inuit harvester prior to the arrival of our team or

discovered by our team during their survey, wherein tissue samples were collected that tested positive for *P. multocida* at a CCWHC laboratory.

(2) *Suspected outbreak*– indicating that a mass mortality event was reported by an Inuit harvester prior to the arrival of our team and during our subsequent visit we found corroborating evidence in the form of skeletal remains at the indicated location; however, diagnostic samples were not collected at the time outbreak precluding laboratory analyses.

(3) *No outbreak observed* – indicating that neither Inuit harvesters nor our team encountered any evidence of a past or ongoing mass-mortality event at a given site.

### **Species distribution model**

My site investigations yielded a dataset of georeferenced islands containing common eider colonies on which I was confident that avian cholera mortality had occurred encompassed within a larger set of locations where investigations were undertaken but the presence of disease was not observed. This is a common situation in studies of disease distribution where it is generally not feasible to determine the infection status of all animals in the study population and disease dynamics are temporally dynamic by nature. Moreover, because avian cholera is thought to be newly emerging disease in the Canadian Arctic I expected current outbreak locations (i.e., the realized distribution) to fall short of the potential distribution on a landscape scale and for outbreak locations to be spatially structured with respect to introduction foci.

**Table 3.1.** Survey locations.*Survey locations and number of islands investigated in the Hudson Strait region of the Canadian Arctic (2010-2013).*

| Nearest community | Survey zone               | No. of islands†  | Year(s) of survey      | Original source for survey locations                  |
|-------------------|---------------------------|------------------|------------------------|---|
| Aupaluk           | 1. Ikkatuk Bay            | 12 (9)           | 2011                   | Chapdelaine et al. 1986; Falardeau et al. 2003        |
| Kangirsuk         | 2. Payne Bay              | 12 (9)           | 2012                   | Chapdelaine et al. 1986; Falardeau et al. 2003        |
|                   | 3. Plover Bay             | 16 (9)           | 2012                   | Chapdelaine et al. 1986; Falardeau et al. 2003        |
| Kangiġsujuaġ      | 4. Joy Bay                | 8 (6)            | 2011                   | Not previously surveyed                               |
|                   | 5. King George Sound      | 21 (9)           | 2011                   | Not previously surveyed                               |
| Ivujivik          | 6. Digges Sound           | 22 (15)          | 2012                   | Gaston et al. 1985                                    |
| Cape Dorset       | 7. Foxe Peninsula         | 13 (8)           | 2010                   | G. Gilchrist, Environment Canada, <i>unpubl. data</i> |
|                   | 8. West Foxe Islands      | 23 (15)          | 2010,<br>2011,<br>2012 | Cooch 1986; Siros and Kay 1991                        |
|                   | 9. Chorkbak Inlet         | 31 (24)          | 2010                   | G. Gilchrist, Environment Canada, <i>unpubl. data</i> |
| Iqaluit           | 10. Chamberlain Islands   | 30 (19)          | 2010,<br>2011,<br>2012 | G. Gilchrist, Environment Canada, <i>unpubl. data</i> |
|                   | 11. Frobisher Bay North   | 20 (1)           | 2013                   | J. Akearok, Environment Canada, <i>unpubl. data</i>   |
|                   | 12. Frobisher Bay Central | 51 (4)           | 2013                   | J. Akearok, Environment Canada, <i>unpubl. data</i>   |
|                   | 13. Frobisher Bay South   | 40 (3)           | 2013                   | J. Akearok, Environment Canada, <i>unpubl. data</i>   |
| <b>Total</b>      | <b>All</b>                | <b>299 (131)</b> |                        |   |

†Number of islands surveyed (and number with &gt;20 eider pairs)

Given these constraints, I elected to use the presence-only species distribution model MAXENT (version 3.3.3k; <http://www.cs.princeton.edu/~schapire/maxent/>; Phillips et al. 2006). MAXENT is a general-purpose machine learning program that has been widely used to predict species distributions using data for which absence records are unavailable or unreliable due to issues of detectability (Phillips et al. 2006, Elith et al. 2011). The program is increasingly used in studies of wildlife disease distribution (e.g., Slater and Michael 2012, Liu et al. 2013, Mweya et al. 2013).

The MAXENT approach is to quantify presence locations in relation to a random sample of background locations (i.e., pseudo-absences) within a landscape of interest. The model algorithm seeks to maximize the apparent non-randomness of this draw by finding the probability distribution of maximum entropy (that which is closest to uniform) subject to constraints imposed by the observed spatial distributions of the species and environmental conditions (Phillips et al. 2006, Elith et al. 2011). Typically, MAXENT is applied in a geographic context to model occurrence probability in relation to grid cell characteristics (i.e., geographic space) using remotely-sensed or directly estimated biophysical data to generate predictive distribution maps. However, Elith et al. (2011) emphasize that program usage need not be restricted in this way and offer an example of presence-only modelling akin to logistic regression for non-gridded data (i.e., covariate space). Such analyses are conceptualized and performed using the program's SWD (samples-with data) format. The key to interpreting model results is to base inference on the multiple outputs of the model, which include area under the curve of the receiver operating characteristic (AUC)—a measure of goodness-of-fit (Fielding and Bell 1997)—and to evaluate the contribution of individual variables to AUC gain when

considered in isolation (percent contribution) or withheld from a fully parameterized model (permutation importance).

In my analysis, I included all confirmed and suspected avian cholera outbreak locations in the presence dataset and the totality of surveyed islands containing >20 common eiders nests in the background dataset. Both datasets were parsed to include only colonies with >20 eider nests to reduce detection bias. Common eider colonies were the sampling unit of interest and associated with each colony was a suite of ecological and environmental attributes of interest. These are described in detail below and included the biophysical attributes of the islands, as well as measures of focal host quantity, and propagate factors relating to the presence and movement behavior of potential hosts.

To avoid of over-fitting the data, I restricted my evaluation primarily to linear features (Barry and Elith, 2006, Pearson et al. 2007) and reduced the number of candidate variables in any single model by undertaking a tiered analysis in which I first evaluated the relative importance of potential explanatory variables within categories of similar data type. After identifying the variable or variables with the greatest explanatory power within categories, I implemented a cross-category analysis using the best supported variables in the first stage evaluation. The tiered analysis also provided a framework for considering the influence of spatial autocorrelation in the characteristics of colonies. Specifically, by evaluating sets of variables expected to be highly correlated in preliminary assessment allowed for more robust cross-category comparisons.

The first category of potential explanatory variable that I considered was fundamental niche attributes of the islands containing colonies (Table 3.2). Freshwater melt ponds are a potentially important source of avian cholera transmission and prospective focal point of management intervention. However, there is considerable variation among islands with respect to pond presence (*pPOND*) and number (*nPOND*), as well as other site characteristics, including percent vegetative cover (*VEG*), island size (*AREA*), and elevation (*ELEV*). These variables potentially influence site drainage and the way host species access and move through the habitat. I quantified pond presence, pond number and vegetative cover directly from field data, while island size and elevation were derived by querying digital thematic maps (CanVec 1:50,000 scale topographic) in ArcGIS (Version 10.1 (Environmental Systems Research Institute Inc., Redlands, CA, USA)).

The second category of explanatory variable that I evaluated was focal host (i.e., common eider) quantity. Botzler et al. (1991) identified two levels at which host numbers potentially influence avian cholera transmission: (1) via an increased probability of bacterial presence with increasing host abundance, and (2) via a higher contact rate with increasing host density. In my model, I included measurements of common eider abundance (*nCOEI*: nests per island) and density (*dCOEI*: nests per ha) on each island, as well as a quadratic feature quantifying pond abundance in relation to the number of nesting eiders present ( $(P/100E)^2$ ). Inclusion of  $(P/100E)^2$  variable allowed me to consider differences among colonies on which a large number of birds shared a common water source independent of nest density (Table 3.2).

The final category of explanatory variable was propagule factors. My interest was to assess evidence for different species or populations acting as disease vectors or reservoirs. Variables included the presence and number co-nesting gulls, vagrant gulls, and the migration paths of eiders and snow geese converging in Hudson Strait during the breeding season from different wintering areas. Gull variables encompassed the presence and number of nesting herring gulls (*pHERG*, *nHERG*), the presence and number of nesting glaucous gulls (*pGLGU*, *nGLGU*), and all gulls within a 5 km radius of the island irrespective of species and nesting status (*aGull*).

To evaluate the location of colonies in relation to common eider migratory flyways I utilized information from satellite tracking studies conducted by Mosbech et al. (2006), Savard et al. (2011), and G. Gilchrist, Environment Canada (*unpublished data*). The aforementioned studies were designed to delineate eider movements between wintering and breeding areas and the authors provided me with raw location estimates for birds breeding in the Hudson Strait region. I processed the data to determine the single highest quality location estimate received during each 2.5 d transmitter duty cycle during spring migration (15 Apr – 1 Jul). I specified an inclusion threshold of  $\pm 1$  km accuracy (i.e., Argos location  $\geq 1$ ) and used the first estimate received per duty cycle when multiple estimates of identical accuracy were obtained. The resulting data yielded directional migration paths for 9 eiders (7 female and 2 male) tracked from wintering areas in Atlantic Canada into Hudson Strait and 20 eiders (16 female and 4 male) tracked from west Greenland into Hudson Strait over 9 yr of study (2000-2004, 2006-2007 and 2012-2013). I then analyzed the data in Spatial Analyst using the line density tool. My

objective was to calculate track densities within 0.01 degree grid cells throughout the study area (summed migration path length [km] per unit of area [km<sup>2</sup>]). This enabled me to extract indices of coastline usage by birds affiliated with wintering areas in Atlantic Canada (*COEI-CanadaFlyway*) or west Greenland (*COEI-GreenlandFlyway*) in the neighborhood of each colony using a search radius buffer of 25 km<sup>2</sup> from each island center.

For lesser snow geese, satellite transmitter data was not available; however, patterns of migratory connectivity are well quantified on the basis of harvest recoveries. For my analysis, I overlaid a breeding distribution and migration map for mid-Continent lesser snow geese (U.S. Fish and Wildlife Service 2007) and again applied a 25 km<sup>2</sup> buffer around each island to extract a binary estimate of intersection between eider nesting colonies and lesser snow goose summer distributions (*SNGO-MidContinentFlyway*) (Table 3.2).

**Table 3.2.** Explanatory variables.

*Definition of the explanatory variables used in MAXENT species distribution models of avian cholera outbreak occurrence in Hudson Strait.*

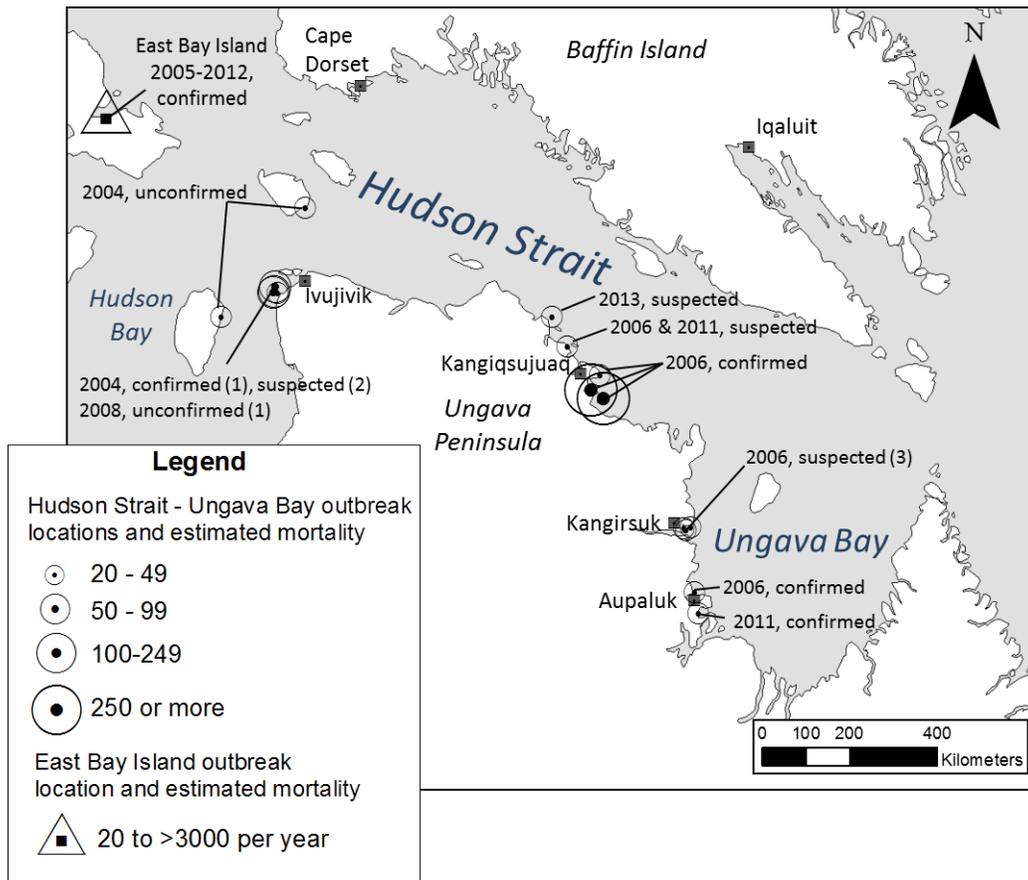
| Explanatory variable            | Description   | Units                     |
|---------------------------------|---|---------------------------|
| <i>Fundamental niche</i>        |   |                           |
| <i>pPOND</i>                    | Presence of ponds (>10 m <sup>2</sup> )                 | 0/1                       |
| <i>nPOND</i>                    | Number of ponds (10 m <sup>2</sup> )                    | Count                     |
| <i>VEG</i>                      | Proportion of island covered by vegetation              | 0-1                       |
| <i>ELEV</i>                     | Maximum elevation contour of island                     | M                         |
| <i>AREA</i>                     | Size of the island                                      | ha                        |
| <i>Focal host quantity</i>      |   |                           |
| <i>nCOEI</i>                    | Number of common eider nests                            | Count                     |
| <i>dCOEI</i>                    | Density of common eider nests                           | Pairs * ha <sup>-1</sup>  |
| <i>(P/100E)<sup>2</sup></i>     | Ponds per 100 common eider nests<br>(quadratic feature) | Ponds/host                |
| <i>Propagule factors</i>        |   |                           |
| <i>pHERG</i>                    | Presence of herring gull nests                          | 0/1                       |
| <i>pGLGU</i>                    | Presence of glaucous gull nests                         | 0/1                       |
| <i>nHERG</i>                    | Number of herring gull nests                            | Count                     |
| <i>nGLGU</i>                    | Number of glaucous gull nests                           | Count                     |
| <i>aGULL</i>                    | Maximum count of gulls within island system             | Count                     |
| <i>COEI-CanadaFlyway</i>        | Common eider - Canada flyway                            | Paths per km <sup>2</sup> |
| <i>COEI-GreenlandFlyway</i>     | Common eider - Greenland flyway                         | Paths per km <sup>2</sup> |
| <i>SNGO-MidContinent Flyway</i> | Snow goose – Midcontinent flyway                        | 0/1                       |

To validate the models that I developed in MAXENT, I used a subset of presence records as ‘training data’ to examine the correlation structure between presence and background sites. I then evaluated the remaining records as ‘testing data’ and assessed model performance on the basis of AUC. AUC values typically range from 0.5–1.0, where value close to 0.5 indicates a fit no better than random, while a value of 1.0 indicates a perfect fit. Models with  $AUC > 0.9$  are considered highly predictive, while models with  $AUC \geq 0.7$  are regarded as informative (Swets 1988). Because relatively few presence records were available for analysis, I used the  $k$ -fold jackknife validation procedure recommend by Pearson et al. (2007) as a further diagnostic tool. Under Pearson’s method the number of bins ( $k$ ) is equal to the number of occurrence localities ( $n$ ) in the dataset and the algorithm evaluates the ability of the model to correctly predict a single withheld locality from the training data set.

## **RESULTS**

### **Avian cholera outbreak locations**

My site investigations encompassed 299 islands distributed over >1000 km of coastline in Hudson Strait. 131 of these islands were habitat for common eider colonies numbering 20 or more breeding pairs. On the basis of Inuit reports and my own site investigations I identified 13 confirmed or suspected avian cholera outbreak locations. A repeat outbreak occurred at one of the locations and mass-mortality events were reported by Inuit harvesters at three additional locations that I was unable to investigate and did not include in my analyses (Figure 3.2, Appendix A Table A.1).



**Figure 3.2.** Avian cholera outbreak locations.

Geographically, the outbreak locations clustered into three broad areas, all of which were in on the Ungava Peninsula and Ungava Bay region of northern Québec. These included: Digges Sound (survey zone 6), western Ungava Bay (survey zones 1 and 2), and the Central Ungava Peninsula (survey zones 4 and 5). Despite extensive survey effort, avian cholera was not detected on Baffin Island or Frobisher Bay (survey zones 7-13).

In Digges Sound, Inuit harvesters reported a total of 221 dead birds from three islands during the first wave avian cholera mortality in 2004 (Appendix A, Figure A.6). The vast majority of the carcasses were common eiders; however 2 glaucous gulls, an unspecified number of geese (species not reported), and an unspecified number of black guillemots (*Cephus grylle*) also were observed. Percent mortality (number of dead female eiders / estimated number of breeding pairs) ranged from 27% to 43% (Table 3.3). Local harvesters reported additional common eider mortality in 2008; however, samples were not collected and our survey team was unable to investigate the site during our visit in 2012.

In western Ungava Bay, Inuit harvesters reported avian cholera mortality at 4 locations in 2006 (Appendix A, Figure A.1 - A.3). The number of dead birds ranged from 20 to 50 per outbreak site and the species implicated included common eiders, black guillemots, and a greater black-backed gull. Our survey team encountered an additional mass-mortality event in 2011 during our survey, in which sick and freshly dead common eiders, herring gulls, black guillemot, and a northern pintail (*Anas acuta*) were recovered for laboratory testing (Appendix A, Figure A.1). Percent mortality ranged from 6% to 22% for female common eiders on affected colonies in western Ungava Bay.

The avian cholera mortality occurring on the Central Ungava Peninsula was originally reported by Inuit harvesters from 4 locations in 2006 (Appendix A, Figure A.4 and A.5). Only common eiders deaths were reported, with the number carcasses observed at individuals sites ranging from 21 to 309 birds. Our survey team encountered a repeat outbreak at one of these locations in 2011 and collected the remains of common eiders, black guillemots, herring and glaucous gulls, and a Canada goose (*Branta canadensis*). An additional outbreak was reported by Inuit harvesters from a separate location in 2013 (Appendix A, Figure A.5). Percent mortality ranged from 1% to 24% on the affected colonies.

**Table 3.3.** Avian cholera mortality among common eiders in Hudson Strait.

*Minimum mortality of female common eiders estimated as the number of carcasses reported on an island divided by breeding pair abundance based by nest counts.*

| Outbreak ID | Location                 | Number of dead female eiders | Estimated colony size | Minimum mortality |
|-------------|--------------------------|------------------------------|-----------------------|-------------------|
| 1           | Digges Sound             | 62                           | 231                   | 27%               |
| 2           | Digges Sound             | 84                           | 228                   | 37%               |
| 3           | Digges Sound             | 75                           | 174                   | 43%               |
| 6           | Western Ungava Bay       | 41                           | 317                   | 13%               |
| 7           | Western Ungava Bay       | 50                           | 227                   | 22%               |
| 8           | Western Ungava Bay       | 50                           | 542                   | 9%                |
| 9           | Western Ungava Bay       | 50                           | 805                   | 6%                |
| 10          | Central Ungava Peninsula | 250                          | 1054                  | 24%               |
| 11          | Central Ungava Peninsula | 36                           | 322                   | 11%               |
| 12          | Central Ungava Peninsula | 309                          | 1292                  | 24%               |
| 13a (2006)  | Central Ungava Peninsula | 55                           | 1840                  | 1%                |
| 13b (2011)  | Central Ungava Peninsula | 21                           | 1840                  | 1%                |
| 15          | Western Ungava Bay       | 39                           | 223                   | 17%               |
| 16          | Central Ungava Peninsula | 25                           | 158                   | 16%               |

### **Species distribution model**

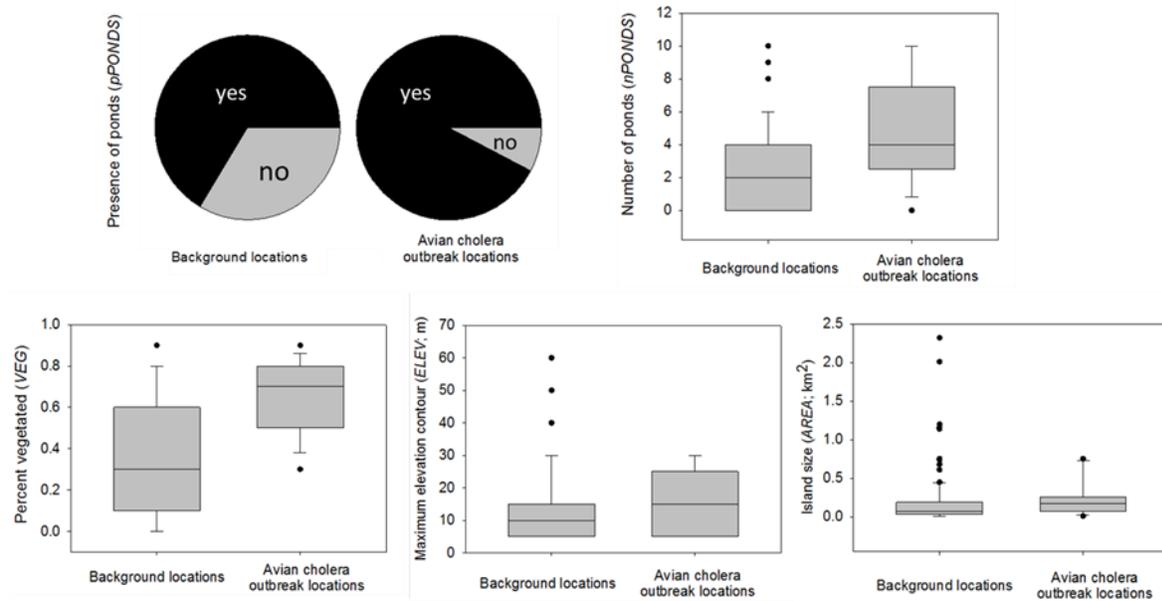
Model validation indicated adequate goodness-of-fit for each of my category-specific models based AUC values and corroborating results were obtained using Pearson et al's (2007) *k*-fold jackknife validation procedure (13 replicates with 12 training records and 1 test record in each run) (Table 3.4).

Among the fundamental niche variables that I considered, vegetative cover (*VEG*) had the greatest explanatory power (percent contribution: 73.5; permutation importance: 85.1; parameter estimate  $\pm$  SD:  $2.66 \pm 0.22$ ). Raw data summaries were consistent with model outputs and indicated that the probability of an outbreak was positively correlated with amount of vegetation on an island (Figure 3.3). Less support was apparent for the presence or number of ponds (*pPOND* and *nPOND*, respectively). Both variables exhibited only weakly positively correlations with outbreak probability. I found no difference in outbreak probability in relation to island size (*AREA*) or elevation (*ELEV*). Based on these observations I included *VEG* in my cross-category composite model.

**Table 3.4.** MAXENT model fit.

*Relative performance of category-specific and composite presence-only species distribution models implemented in MAXENT to examine ecological correlates of avian cholera outbreak occurrence.*

|   | Area under receiver operating characteristic curve (AUC) |                      | Pearson jackknife test result |         |
|---|--|----------------------|-------------------------------|---------|
|   | Training AUC   | Test AUC ( $\pm$ SD) | Success rate                  | P-value |
| Category-specific models                              |  |                      |                               |         |
| Fundamental niche (5 variables)                       | 0.820  | 0.767 ( $\pm$ 0.169) | 85%                           | <0.01   |
| Focal host quantity (3 variables)                     | 0.807  | 0.789 ( $\pm$ 0.179) | 85%                           | <0.01   |
| Propagule factors (8 variables)                       | 0.801  | 0.741 ( $\pm$ 0.137) | 78%                           | <0.05   |
| Composite model                                       |  |                      |                               |         |
| $VEG + nCOEI + (P/100E)^2 + COEI\text{-CanadaFlyway}$ | 0.856  | 0.830 ( $\pm$ 0.113) | 92%                           | <0.001  |

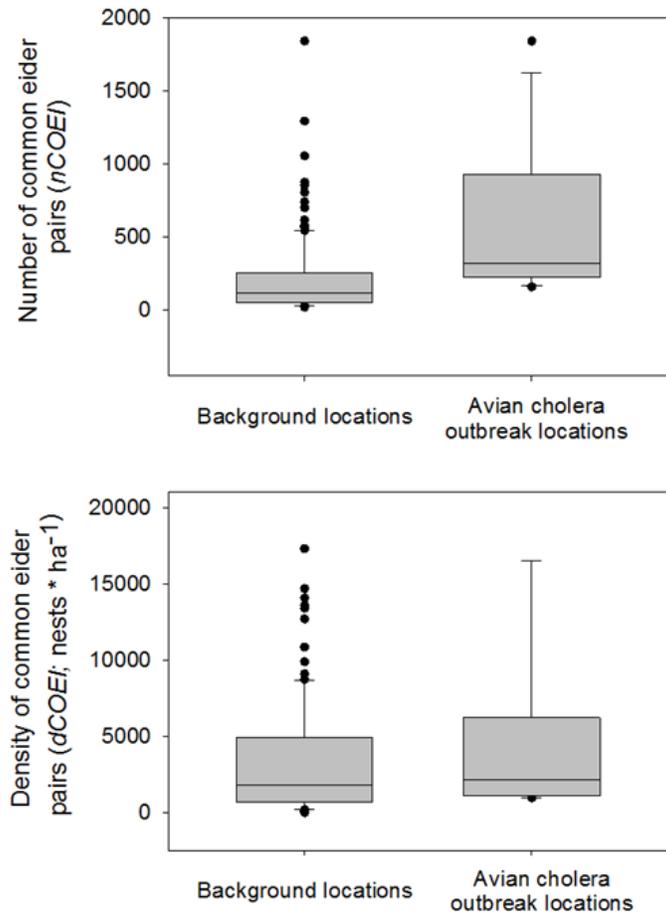


**Figure 3.3.** Fundamental niche attributes associated with heightened outbreak risk.

*Comparison of fundamental niche attributes of islands with northern common eider nesting colonies at background (N = 131) and avian cholera outbreak (N = 13) locations.*

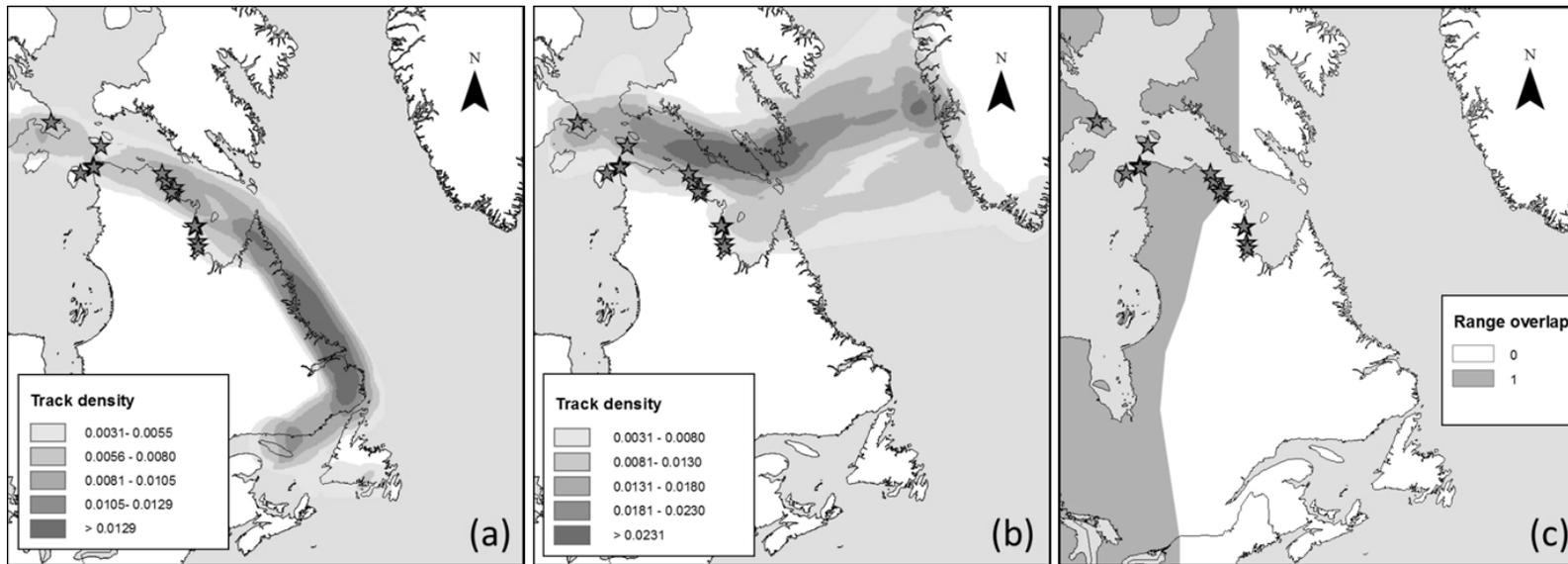
Within the focal host quantity category, estimates of variable importance indicated support for common eider abundance (*nCOEI*) (percent contribution: 53.2; permutation importance: 29.8; parameter estimate  $\pm$  SD:  $1.96 \pm 0.37$ ) and ponds per 100 eiders ( $(P/100E)^2$ ) (percent contribution: 45.9; permutation importance: 69.7; parameter estimate  $\pm$  SD:  $1.75 \pm 0.55$ ) as relevant explanatory variables. I found comparatively little support for common eider density (*dCOEI*). Data summaries were consistent with model outputs and indicated that eider abundance was higher at outbreak sites than background sites (Figure 3.4). On this basis I included *nCOEI* and  $(P/100E)^2$  in my cross-category analysis, but did not include *dCOEI*.

Within the propagate factor category I found a strong correlation between outbreak probability and the density of spring migration tracks for common eiders moving into Hudson Strait from wintering areas in Atlantic Canada (*COEI-CanadaFlyway*) (percent contribution: 66.1; permutation importance: 74.3.8; parameter estimate  $\pm$  SD:  $1.96 \pm 0.27$ ) (Figure 3.5). I found little evidence to support a predictive relationship between outbreak probability and gull presence, overlap with lesser snow goose breeding or migration distributions, or the migratory tracks used by common eiders wintering in Greenland. On this basis I included *COEI-CanadaFlyway* in my composite analysis.



**Figure 3.4.** Focal host quantity attributes associated with heightened outbreak risk.

*Comparison of northern common eider abundance (nCOEI) and density (dCOEI) estimates at background (N = 131) and avian cholera outbreak (N = 13) locations.*



**Figure 3.5.** Propagule factor attributes associated with heightened outbreak risk.

*Avian cholera outbreak locations in Hudson Strait in relation to the migratory paths and distribution of (a) common eiders wintering in Atlantic Canada, (b) common eiders wintering in west Greenland, and (c) mid-continent lesser snow geese. Stars indicate locations where mass-mortality events have been observed; track densities (summed track length per km<sup>2</sup> within 0.1 degree grid squares) compare the relative use of migration corridors by birds originating from different wintering areas.*

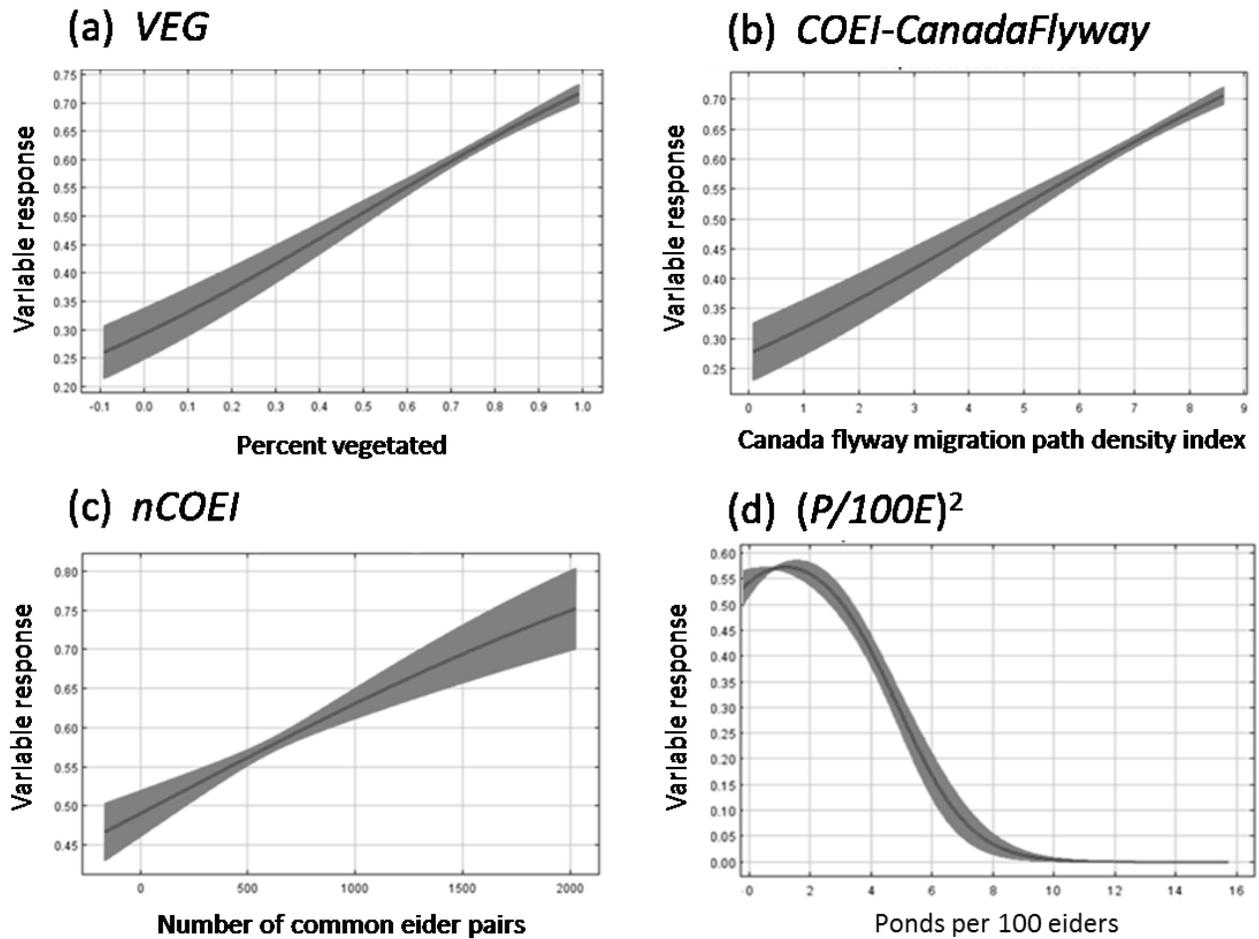
Model validation indicated that my cross-category composite model, comprised of the variables supported for inclusion by my first stage analysis, adequately fit the data (assignment success rate = 92%,  $P < 0.001$ ; test AUC = 0.830; Table 3.4). When considered in isolation, percent contribution to AUC gain was highest for vegetative cover (*VEG*) and migration track density for eiders affiliated with wintering areas in Atlantic Canada (*COEI-CanadaFlyway*) (Table 3.5). When assessing permutation importance (i.e., the loss of significant AUC when a single variable was withheld), the highest estimates were obtained for *VEG*, *COEI-CanadaFlyway*, and the quadratic feature ponds per 100 eiders ( $(P/100E)^2$ ).

All 4 variables included in the composite model had model coefficients with standard deviations that did not overlapping with zero, indicating statistically significant effects. Marginal response curves indicate positive correlations between avian cholera outbreak probability and *VEG*, *COEI-CanadaFlyway*, and common eider abundance (*nCOEI*) (Figure 3.6).  $(P/100E)^2$  followed a Poisson distribution, with peak outbreak probability occurring when ponds are present but eiders are crowded into relatively few of them.

**Table 3.5.** Best supported ecological correlates of outbreak risk.

*Variable contribution estimates and model coefficients for the MAXENT cross-category evaluation of ecological correlates of avian cholera occurrence.*

| Variable                    | Percent contribution | Permutation importance | Model coefficient ( $\pm$ SD) |
|-----------------------------|----------------------|------------------------|-------------------------------|
| <i>VEG</i>                  | 40.1                 | 35.4                   | 1.53 ( $\pm$ 0.27)            |
| <i>COEI-</i>                | 35.7                 | 27.2                   | 2.83 ( $\pm$ 1.18)            |
| <i>CanadaFlyway</i>         |                      |                        |                               |
| <i>nCOEI</i>                | 13                   | 11.1                   | 1.65 ( $\pm$ 0.27)            |
| <i>(P/100E)<sup>2</sup></i> | 11.2                 | 26.0                   | -17.01 ( $\pm$ 5.96)          |



**Figure 3.6.** Marginal response curves for ecological predictors of outbreak risk.

*Partial dependence plots showing the marginal response of avian cholera outbreak probability on islands used by breeding common eiders in relation to (a) percent vegetative cover, (b) migration track density of common eiders affiliated with wintering areas in Atlantic Canada, (c) number of common eider breeding pairs, and (d) a quadratic effect of ponds per 100 eiders, a measure of host crowding.*

## DISCUSSION

My study illustrates the value of integrating host ecology with fundamental niche information when assessing the spatial distribution of infectious diseases in wildlife. To date, the majority of species distribution models employed to evaluate disease emergence patterns have focused on abiotic climate and habitat-related factors (e.g., Puschendorf et al. 2009, Slater and Michael 2012, Machado-Machado 2012, Mweya et al. 2013). However, for many infectious pathogens, host factors are an equally, if not more, important determinant of disease occurrence patterns. My investigations indicate that avian cholera is much more widespread in the Canadian Arctic avifauna than was previously described and that the distribution of outbreak events has been non-random with respect to ecological predictors. I discuss the implications of my findings for predicting and controlling avian cholera epidemics in newly affected species in turn, below.

Wobeser (1992) postulated two dominant modes of avian cholera transmission in wild birds. The first is a 'smoldering' low prevalence state within host populations in which at least some individuals have immunity and contact rates between susceptible individuals and the bacterium are low. The second is an 'explosive' state wherein episodic outbreaks occur at locations where birds congregate, often in multi-species flocks, and ecological conditions facilitate the rapid spread of disease in a shared environment. Given these characteristics, pathogen prevalence within a population, the spatial distribution of outbreak events, and magnitude of mortality depend upon a complex set of interactions between environmental conditions and host behavior. The variables with greatest predictive value for identifying avian cholera outbreak locations in my study reflect this complexity.

The most highly ranked variable in my species distribution model was percent vegetative cover. This result was somewhat surprising because current research emphasizes the importance of contaminated wetlands as the focal point of *P. multocida* transmission in wild waterfowl (Samuel et al. 2004, Blanchong et al. 2006b, Wobeser 2007). When assessing the habitat attributes of islands supporting northern common eiders in the Hudson Strait region I found considerable variation in the presence and number of freshwater ponds among islands. On many of the islands that did have ponds the ponds that were present were depauperate in organic matter. Liu et al. (2013) obtained a similar result when investigating the distribution of *Batrachochytrium dendrobatidis* (Bd) infections in amphibians. Bd is an aquatic fungus, yet water habitat was not predictive of disease incidence while vegetative characteristics were highly predictive. Liu et al. reasoned that vegetative cover is likely a surrogate for the dynamics of ground moisture and temperature, which are known to influence the survival of a range of environmentally transmitted pathogens (Rohr and Raffel 2010, Wood et al. 2010), including *P. multocida* (Backstrand and Botzler 1986). A similar situation may be at play in the case of avian cholera outbreaks on common eider colonies in the Arctic environment.

The Hudson Strait region is characterized by a rocky, sparsely vegetated landscape with limited precipitation. My threshold size for quantifying the presence and number of ponds on an island ( $>10 \text{ m}^2$ ) was based on an approximation of the size of pond required for an eider to land before accessing the surrounding terrestrial habitat. This decision was based on the expert opinion that many of the largest eider colonies in the region are located on highly vegetated islands and the eider nest sites are often clustered around organically rich freshwater ponds.

Indeed, the number of ponds per eider was also a highly ranked variable in my models. I quantified ponds per eider as quadratic term and found that avian cholera outbreak probability was highest when many eiders were crowded into relatively few (but not zero) ponds. Interestingly, while the number of ponds per eider had less support than vegetative cover as a predictor when the variables were considered independently, their permutation importance values were very similar. This finding is consistent with the notion that the crowding of waterfowl in wetlands increases their susceptibility to avian cholera through a combination of heightened contact rates and potentially stress-induced reductions in host capacity to resist infection (Botzler 1991).

There are additional reasons why *P. multocida* may be more tied to vegetative characteristics of eider colonies than wetlands *per se*. Incubating eiders have among highest nest constancy rates of any bird species (Afton and Paulus 1992, Bottitta 1999). Once their eggs are laid female eiders rarely take incubation breaks and one of the key investigative features of an avian cholera mass-mortality event is the presence of dead females still sitting on their nests (Swennen and Smit 1991, Buttler et al. 2011). Secretions from infected hosts and bacteria released by scavengers are therefore more likely to enter soil and sediments than wetlands once die-offs begin on breeding colonies. This situation differs from the situation on non-breeding areas, where waterfowl may spend the majority of their time foraging and roosting open water.

The second most highly ranked explanatory variable in my species distribution model was migration track density for eiders arriving in Hudson Strait from wintering areas in Atlantic

Canada (*COEI-CanadaFlyway*). This finding bears heavily on the question of disease origins and reservoir species. Harms (2012) proposed two plausible explanations for the sudden appearance of avian cholera in northern common eiders. The first is that a virulent strain of *P. multocida* jumped from American common eiders to northern common eiders at shared wintering areas in Atlantic Canada during the non-breeding season and was subsequently carried north by migrating northern common eiders. The second is that the outbreaks originated from lesser snow geese, which co-mingle with northern common eiders during the breeding season. The outbreak patterns that I documented in Hudson Strait would appear to be more consistent with the former hypothesis, but not the later.

My analysis of satellite telemetry location data indicate that northern common eiders that winter in Atlantic Canada closely follow the Labrador and northern Québec coastlines when entering Hudson Strait. I found that avian cholera outbreak locations clustered along this flyway, but were apparently absent from the southern Baffin Island coast, which Atlantic Canada wintering eiders use much less frequently. In contrast, lesser snow geese migrating into the Hudson Strait region originate in the central United States and are predominately found in the western portion of the Hudson Strait region during the breeding season (Mowbray et al. 2000). Snow geese often use offshore islands as foraging and roosting sites before moving inland to breed and the presence of lesser snow geese on Mitivik Island before eiders arrive has been identified as potential source of avian cholera in eiders at that location (Buttler 2009). When migrating across Hudson Strait to breeding areas on Baffin Island, lesser snow geese typically fly over coastal areas of northern Québec without landing, but intensively use islands

on the south coast of Baffin Island as migratory stopover sites. The apparent absence of avian cholera on the Baffin Island coast despite intensive surveillance, and the presence of avian cholera in Ungava Bay, where lesser snow geese are absent, suggests the species has not been a major vector of avian cholera emergence in northern common eiders.

Preliminary genotyping results relating bacterial lineages (serotypes) to host populations partially corroborate my conclusions, but also highlight continuing uncertainty regarding disease origins and host networks. To date, only a single *P. multocida* serotype has been detected from diseased birds in northern Québec (serotype 3 x4), whereas multiple serotypes have been detected at Mitivik Island (serotypes 1, 3, 4 and 3x4) (N. J. Harms, University of Saskatchewan, *unpublished doctoral dissertation*). The 3x4 serotypes isolated from northern Québec and Mitivik Island are genetically similar to each other; however this strain of *P. multocida* appears to be distinct from isolates collected from American common eiders in the St. Lawrence Estuary of southern Québec. The serotype 1 isolate detected on Mitivik Island appears to be genetically similar to isolates collected from migrating and wintering waterfowl in the Central Flyway, including lesser snow geese. Thus, although the ecological evidence is compelling that a virulent strain of *P. multocida* has recently emerged in the segment of the northern common eider population that winters in Atlantic Canada the source remains unknown.

Host aggregation patterns are known to be a key factor in disease transmission because of their influence on contact rates. Avian cholera is transmitted both environmentally and via direct bird-to-bird contact, and these modes of transmission likely play different roles in disease

perpetuation throughout the annual cycle. My species distribution model indicated support for common eider abundance, but not density, a predictor of avian cholera outbreak probability. This finding is consistent with hypothesis that epidemic take-off is primarily a frequency-dependent process when environmental transmission is the dominate mode of disease spread to new locations when prevalence is low (Roche et al. 2009, Rohani et al. 2009). While avian cholera outbreak probability was positively correlated with eider abundance, I observed a high degree of variability in the extent of mortality at individual outbreak sites. I found no correlation between host population size or host density in absolute (i.e., total carcasses reported) or proportional (i.e., eider carcasses per breeding pair) terms. Moreover, many of the die-off events that I investigated involved relatively few (<50) individuals and evidence for a multi-year epidemic was encountered at only one site. This stands in stark contrast to the situation at Mitivik Island (Chapter 2 and Chapter 5), where many more eiders were found dead and the epidemic stretched over 8 years.

The assumption of equilibrium between organisms and their environment is a standard postulate in species distribution models. However, this assumption is routinely violated in biological invasions where spatial patterns of distribution are constrained by dispersal and the colonization processes (Elith and Leathwick 2009, Václavík and Meentemeyer 2012). If the goal is to identify habitats at potential risk of future spread, the stage of invasion must be considered. Within my study area, highly abundant eider colonies with biophysical attributes that are presumably conducive to *P. multocida* transmission were not found to harbor disease and the principle barrier appeared to be population subdivision. That the disease is present in

some geographic segments of the population but not others is consistent with a recent range expansion. An informal monitoring network comprised of biologists and Inuit harvesters exists throughout the northern common eiders' range in the Canadian Arctic, Labrador, and Greenland. Mass-mortality events have not been reported elsewhere and avian cholera prevalence within the 'global' population remains low. My site investigations suggest that Inuit reports of avian cholera outbreak events were highly reliable and my findings can serve as a useful basis for directing surveillance efforts.

## Chapter 4: Longer ice-free seasons increase the risk of nest depredation by polar bears for colonial breeding birds in the Canadian Arctic

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Iverson, S. A., Gilchrist, H. G., Smith, P. A., Gaston, A. J., and Forbes, M. R. (2014). Longer ice-free seasons increase the risk of nest depredation by polar bears for colonial breeding birds in the Canadian Arctic. *Proceedings of the Royal Society B: Biological Sciences*, 281(1779), 20133128.

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### ABSTRACT

Northern polar regions have warmed more than other parts of the globe potentially amplifying the effects of climate change on biological communities. Ice-free seasons are becoming longer in many areas, which has reduced the time available to polar bears (*Ursus maritimus*) to hunt for seals and hampered bears' ability to meet their energetic demands. In this study, we examined polar bears' use of an ancillary prey resource, eggs of colonial nesting birds, in relation to diminishing sea ice coverage in a low latitude region of the Canadian Arctic. Long-term monitoring reveals that bear incursions onto common eider (*Somateria mollissima*) and thick-billed murre (*Uria lomvia*) nesting colonies have increased >7-fold since the 1980s and

that there is an inverse correlation between ice season length and bear presence. In surveys encompassing >1000 km of coastline during years of record low ice coverage (2010-2012), we encountered bears or bear sign on 34% of eider colonies and estimated greater egg loss as consequence of depredation by bears than by more customary nest predators, such as foxes and gulls. Our findings demonstrate how changes in abiotic conditions caused by climate change have altered predator-prey dynamics and are leading to cascading ecological impacts in Arctic ecosystems.

## **INTRODUCTION**

Climate change can influence species directly by modifying their physical environment or indirectly by altering interactions among organisms (Parmesan 2006). Altered interactions include increased or diminished interspecific competition (Ahola et al. 2007, Milazzo et al. 2013), modified host-parasite relationships (Kutz et al. 2005, Bosch et al. 2007), and changes to predator-prey dynamics (Harley 2011, Schmidt et al. 2012). Rapid environmental changes affecting top predators are of particular relevance because behavioural changes on the part of predators have the potential to restructure food webs and lead to cascading ecological impacts on prey populations (Harley 2011, Baum and Worm 2009).

To date, the effects of climate change have been most pronounced in northern polar regions, where temperatures have risen at a much faster rate than in other regions of the globe (ACIA 2005, Hansen 2006). The polar bear (*Ursus maritimus*) is an apex predator in the circumpolar Arctic that relies on sea ice as a platform to hunt seals and other marine mammals.

The progressively earlier break-up of annual sea ice in low latitude regions of the Arctic has reduced the amount of time available to bears to hunt for seals and amass the fat reserves they require to sustain themselves on shore during the ice-free season (Stirling et al. 1999, Derocher et al. 2004, Durner et al. 2009, Derocher 2010). Earlier sea ice clearance is a major conservation concern for polar bears because it has been associated with deteriorating body condition, reduced demographic performance, and population decline (Regehr et al. 2007, Rode et al. 2010a, Regehr et al. 2010).

Polar bears are opportunistic predators that feed on a diversity of resources when onshore, including human garbage (Lunn et al. 1985), large terrestrial mammals (Derocher et al. 2000), fish (Dyck and Romberg 2007), and vegetation (Derocher et al. 1993). In recent years, ornithologists at a number of Arctic monitoring stations have reported increased numbers of encounters with polar bears coming ashore to feed on bird eggs and more rarely adults or chicks (Stempniewicz 2006, Drent et al. 2008, Rockwell and Gormezano 2009, Smith et al. 2010). Although it has been suggested that the consumption of terrestrial prey could offset nutritional shortfalls experienced by bears as a consequence of climate change (Dyck and Kebreab 2009), this assertion has been met with scepticism (Rode et al. 2010b). Resources other than marine mammals are generally regarded as too dispersed and inefficient for bears to consume and digest to be of tangible benefit at a population level. However, considerable uncertainty remains with respect to the frequency and impact of terrestrial foraging by polar bears, including how the prevalence of this behaviour relates to changing environmental

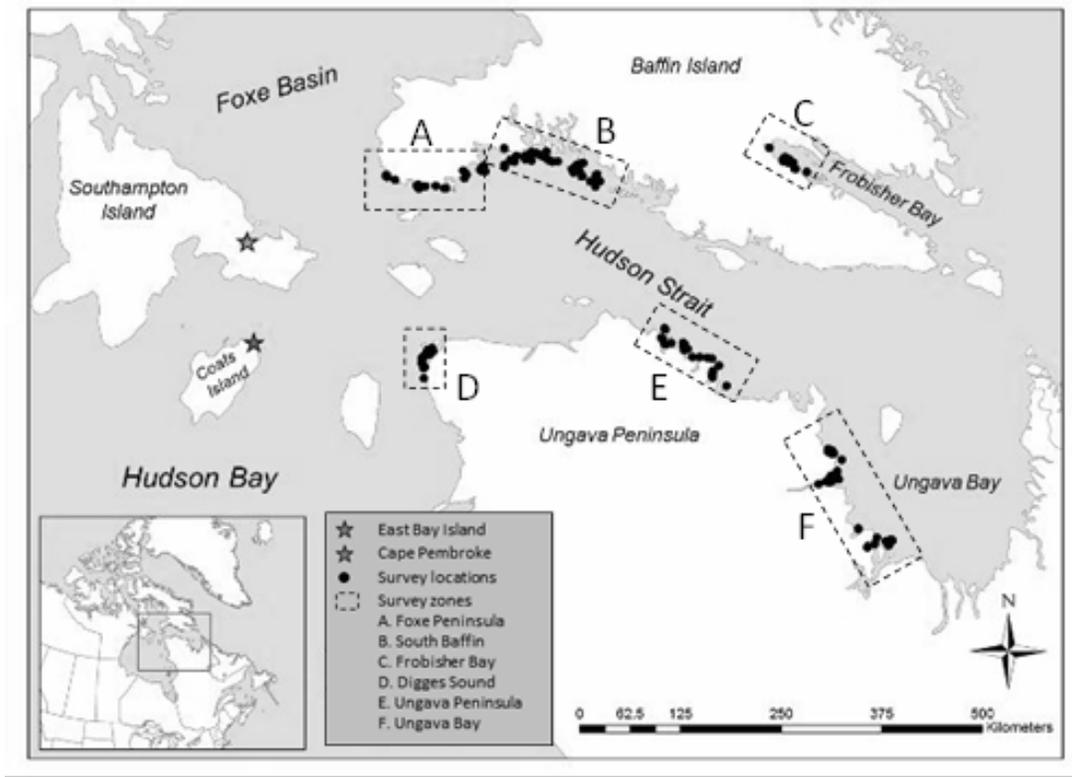
conditions and what influence it might have on the fitness and long-term viability of prey populations unaccustomed to intensive depredation by bears.

In this study, we evaluate egg depredation by polar bears on northern common eiders (*Somateria mollissima borealis*) and thick-billed murrelets (*Uria lomvia*) in a low latitude region of the Canadian Arctic. We use observations from two long-term bird-monitoring stations to assess changes in the frequency of bear incursions and supplement these data with information collected in replicated surveys of eider colonies distributed over a large geographic area. We investigate whether the frequency of bear incursions onto bird colonies is correlated with sea ice conditions and ask whether egg depredation has increased as a result. In addition, we estimate bear prevalence on eider colonies in relation to colony and landscape attributes and compare the magnitude of egg loss due to depredation by bears to that of more customary nest predators such as foxes (*Vulpes* spp.) and gulls (*Larus* spp.). We interpret our findings in relation to their consequences for avian productivity and ecological factors heightening or moderating predation risk.

## **METHODS**

### **Study system**

We conducted our research in the Hudson Strait-Northern Hudson Bay Narrows region of the Canadian Arctic (Figure 4.1). Sea ice melts completely or nearly so during summer in this region requiring bears to spend several months on land surviving on stored fat reserves and what



**Figure 4.1.** Map of the study area.

terrestrial prey they might encounter (Stirling and Parkinson 2006). It is an area of overlap between the ranges of the Davis Strait and Foxe Basin polar bear subpopulations (Obbard et al. 2010), where ringed seals (*Pusa hispida*) are polar bears' primary prey item (Thiemann et al. 2008).

The region also lies within the core breeding ranges of the northern common eider (Goudie et al. 2000) and thick-billed murre (Gaston and Hipfner 2000). Northern common eiders breed in colonies that number from a few to several thousand of pairs. Clutches average 3-5 eggs and nesting occurs primarily on small (0.1 km<sup>2</sup> to 5.0 km<sup>2</sup>), nearshore (<5 km) islands. Thick-billed murres lay a single egg and nest on cliff ledges in colonies comprised of thousands to hundreds of thousands of breeding pairs. Both species initiate nests in late June or early July, with incubation periods ranging from 26 to 33 days for eiders and murres, respectively.

### **Temporal variation in bear observations in relation to ice conditions**

Information about temporal changes in the frequency of polar bear incursions onto bird colonies was obtained from two locations. Mitivik Island (64.03°N, 81.79°W), situated off the eastern coast of Southampton Island, Nunavut, supports ~5000 common eider breeding pairs and is the largest known eider colony in the Canadian Arctic (Environment Canada. 2012). Cape Pembroke (62.79°N, 82.28°W), located on the north eastern tip of Coats Island, Nunavut, provides nesting habitat for ~30,000 thick-billed murres. Biologists were present at Mitivik Island during eider incubation (18 Jun - 1 Aug) from 1997-2012 and at Cape Pembroke during murre incubation (10 Jul - 10 Aug) from 1988-2011 (Table B.1). At both locations, bear

observations were recorded in daily log books. We used these data to estimate the cumulative number of days on which one or more bears was present each year (TOTALBEAR) and to calculate daily probabilities of bear presence (BEARDAY) (see Appendix B: *Polar bear observations at Mitivik Island and Cape Pembroke*).

Sea ice coverage information was obtained using the Canadian Ice Service's IceGraph Tool 2.0 (<http://dynaweb.cis.ec.gc.ca/IceGraph20>) (Tivy et al. 2011). We queried the database for weekly estimates of the proportion of the sea surface covered by ice (IceCT) in Northern Hudson Bay Narrows, which encompasses both Mitivik Island and Cape Pembroke. To facilitate our analyses, we classified sea ice habitat in relation to its quality for polar bears, where <30% IceCT is considered non-habitat, 31-60% IceCT is poor habitat, and >60% IceCT is good or best quality habitat (Sahanatien and Derocher 2012, Cherry et al. 2013). We then derived a series of ice condition indices (ICECOVER) to characterize winter ice season length and the timing of spring break-up at good to poor and poor to non-habitat thresholds, as well as IceCT on specified dates corresponding to eider and murre nesting phenology (18 Jun – early incubation; 2 Jul – mid-incubation; 16 Jul – late incubation) (see Appendix B: *Sea ice conditions*).

When assessing the influence of a climatic factor (e.g., ice coverage) on a biological process (e.g., bear incursions) the co-occurrence of temporal trends can lead to spurious correlations. This is because of the possibility that the trend in biological process results from a relationship with an overlooked causal factor rather than a relationship with the climate factor itself (Grosbois et al. 2008). We predicted a negative association between ice coverage and bear incursions and required a method to control for year-effects. Therefore, in addition to

standard regressions of TOTALBEAR and the various ICECOVER indices against YEAR, and against each other, we derived ICECOVER-YEAR residuals and used the resulting detrended variable (ICECOVER') to assess statistical relationships.

We evaluated the data using Generalized Linear Mixed Models (GLMMs) (Bolker et al. 2009), which we implemented using the package lme4 in R (Bates et al. 2013). We specified BEARDAY as a binary response variable (binomial distribution; logit link; scalar function: nAQR = 7) and classified year as a categorical random effect (YEARRE) to account for the non-independence of daily observations within years. Fixed effect covariates included YEAR (classified as a continuous variable), DAY of the breeding season, DAY<sup>2</sup>, and the best fitting representation of detrended ICECOVER' from the regressions described above. Data for Mitivik Island and Cape Pembroke were pooled and we included SITE as a categorical fixed effect. Our global model included YEARRE in additive combination with these fixed effects, as well as a DAY\*SITE interaction. We then compared the global model to a series of reduced models, the most basic of which was a YEARRE-only model that served as the null model in our candidate set. Model selection was based on maximum likelihood methods, evidence ratios, and Akaike's Information Criterion corrected for sample size (AICc) (Burnham and Anderson 2002). Regression coefficients (i.e.,  $\beta$  values on a logit scale) were estimated by model averaging and statistical significance was judged on the basis of sign (positive or negative relationship between the predictor and response variables) and the precision of estimates, (wherein values with 95% Confidence Intervals that did not overlap with zero were considered important predictors).

### **Extent and magnitude of egg depredation by polar bears**

To examine the extent and magnitude of egg depredation by polar bears in relation to colony and landscape attributes we utilized data collected in boat-based surveys of common eider colonies on the south coast of Baffin Island (Nunavut) and the north coast of Québec (Nunavik). The surveys were carried out over the course of three summers during mid- to late-incubation (10-26 Jul 2010, 6-19 Jul 2011 and 8-21 Jul 2012) and encompassed >1000 km of coastline divided among six survey zones (Figure 4.1). Islands in the Foxe Peninsula and South Baffin survey zones were visited in multiple years, whereas islands in the other four survey zones were visited on a single occasion (Appendix B, Table B.2).

Each island was investigated by 3 to 8 field workers, who walked 10-25 m apart from each other and made successive linear sweeps of all available nesting habitats (Robertson and Gilchrist 1998). When an eider nest was encountered, its status was recorded as either *active* – a nest attended by an incubating hen, or containing eggs or newly hatched ducklings, or *empty* – a nest in which fresh feather down was present but there was no hen, eggs or ducklings. Nests were detected easily because there is little vegetation in our study area and current year breeding attempts could be distinguished from previous year attempts by the presence of fresh feather down (Goudie et al. 2000, Robertson and Gilchrist 1998).

We also recorded evidence for the presence of species known to depredate eider eggs, including gulls (principally herring and glaucous gulls (*L. smithsonianus* and *L. hyperboreus*, respectively)), foxes (Arctic (*V. lagopus*) and red (*V. vulpes*)), and polar bears. Predator sign near

a nest can be a poor indicator of the species responsible for a particular depredation event because predator species often differ in their detectability. Therefore, we evaluated predator presence at a colony level in relation to visible sign, as opposed to more circumstantial evidence of egg consumption by predators at specific nests. We regarded seeing a predator species as direct evidence of its presence, whereas finding animal sign, including faeces, tracks and fur was regarded as indirect evidence (see Appendix B: *Extent and magnitude of egg depredation by polar bears*).

For our analysis, we summarized the number and proportion of islands with either direct or indirect evidence of predator presence by species group (BEARSIGN, FOXSIGN, GULLSIGN). We then used GLMMs to assess variation in the probability of encountering sign (direct and indirect combined) in relation to colony and landscape attributes. GULLSIGN was encountered on nearly all islands (~95%); therefore we did not evaluate statistical relationships for gulls. In our models, island was treated as a categorical random effect (IslandRE) to control for sampling at some locations in multiple years. Fixed effect covariates included the number of eider nests (SQRTNESTS), island size (AREA), distance to the mainland (MAINLAND), maximum inter-step distance over open water that a mammalian predator coming from the mainland would have to cross to reach the island (CROSSING), distance to the nearest Inuit community if travelling by boat (VILLAGE), and ice concentration within a 25 km radius of the island on 25 Jun (ICE25) (see Appendix B: *Landscape attributes*). Our candidate set of models included all possible additive combinations of the predictor variables, as well as an ISLANDRE–only null model. To avoid over-fitting the data we did not evaluate interactions between variables.

We also estimated the proportion of nests remaining active at the time of survey (NESTSUCCESS) in relation to the types of predator sign encountered (PREDATORTYPE: none, gull only, gull and fox, gull and bear), as well as survey zone (ZONE), nest abundance (SQRTNESTS), and date of survey (DATE). We again used GLMMs and treated island as categorical random effect (IslandRE). We used our analyses to determine colony and landscape attributes most associated with predator presence and to estimate rates of nest loss when different combinations of predator sign were encountered.

## RESULTS

### Temporal variation in ice conditions and bear observations

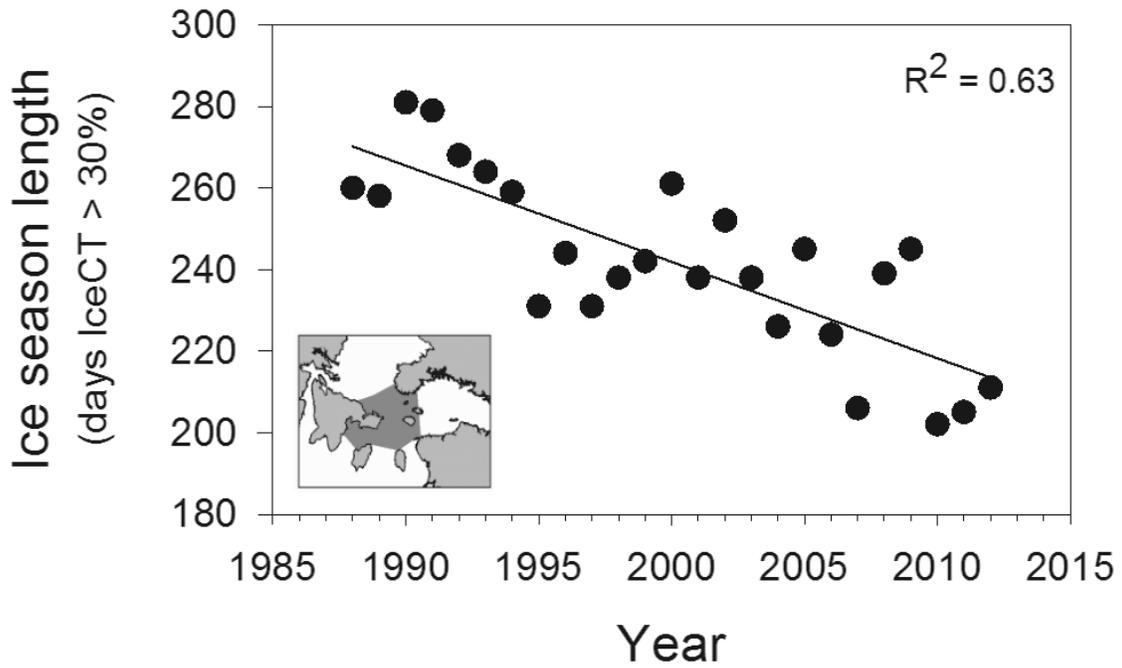
The number of days on which biologists observed one or more polar bears during the bird nesting season increased markedly at both the Mitivik Island common eider colony (Appendix B, Figure B.1a) and the Cape Pembroke thick-billed murre colony (Appendix B, Figure B.1b). Five-year running averages ( $\pm$  SE) indicated a 7-fold increase in bear incursions at Mitivik Island ( $TOTALBEAR_{1997-2001} = 1.8 \pm 0.5$  days with bear  $\cdot yr^{-1}$ ;  $TOTALBEAR_{2008-2012} = 12.6 \pm 0.9$  days with bear  $\cdot yr^{-1}$ ) and an 8-fold increase at Cape Pembroke ( $TOTALBEAR_{1988-1992} = 1.4 \pm 0.5$  days with bear  $\cdot yr^{-1}$ ;  $TOTALBEAR_{2007-2011} = 12.0 \pm 1.4$  days with bear  $\cdot yr^{-1}$ ), respectively.

Sea ice coverage declined dramatically in Northern Hudson Bay Narrows during our study interval. We estimated a  $2.4 (\pm 0.4)$  d  $\cdot yr^{-1}$  decrease in ice season length at the 30% IceCT threshold (poor to non-habitat) from 1988 to 2012 (Figure 4.2), which equates to a 2 month loss of sea ice habitat for polar bears. Annual estimates for ice season length ranged from a

maximum of 281 d in 1990 to minimum of 202 d in 2010 (Appendix B, Table B.3). A similar trend was evident for ice season length at the 60% IceCT (good to poor habitat). Advancing spring break-up date accounted for approximately half of the sea ice loss at both the 30% and 60% thresholds. Ice coverage at specified dates (18 Jun, 2 Jul and 16 Jul) corresponding with eider and murre nesting phenology (early, mid and late incubation) also exhibited sharp declines; however the fixed date indices exhibited greater inter-annual variability than the indices calibrated in relation to duration of ice coverage or break-up date (Appendix B, Table B.3). Among the ICECOVER indices that we evaluated, season length at the 30% IceCT threshold (*SeasonLength30*) was the most strongly correlated with bear incursions on both Mitivik Island and Cape Pembroke (Appendix B, Table B.4 and B.5). Therefore, we used *SeasonLength30* as our index of ICECOVER in subsequent analyses.

The best fitting model in our candidate set of GLMMs investigating variation in daily probability of bear incursions (BEARDAY) indicated that YEAR, DAY, DAY<sup>2</sup>, and detrended ICECOVER' were significant predictors ( $\log(L) = -507.67$ ,  $K = 6$ ,  $AIC_c = 1027.40$ .  $w_i = 0.72$ ). The global model, which included the same variables, as well as SITE and SITE\* DAY was also supported by the data ( $\log(L) = -506.70$ ,  $K = 8$ ,  $\Delta AIC_c = 2.12$ .  $w_i = 0.25$ ) (Appendix B, Table B.6).

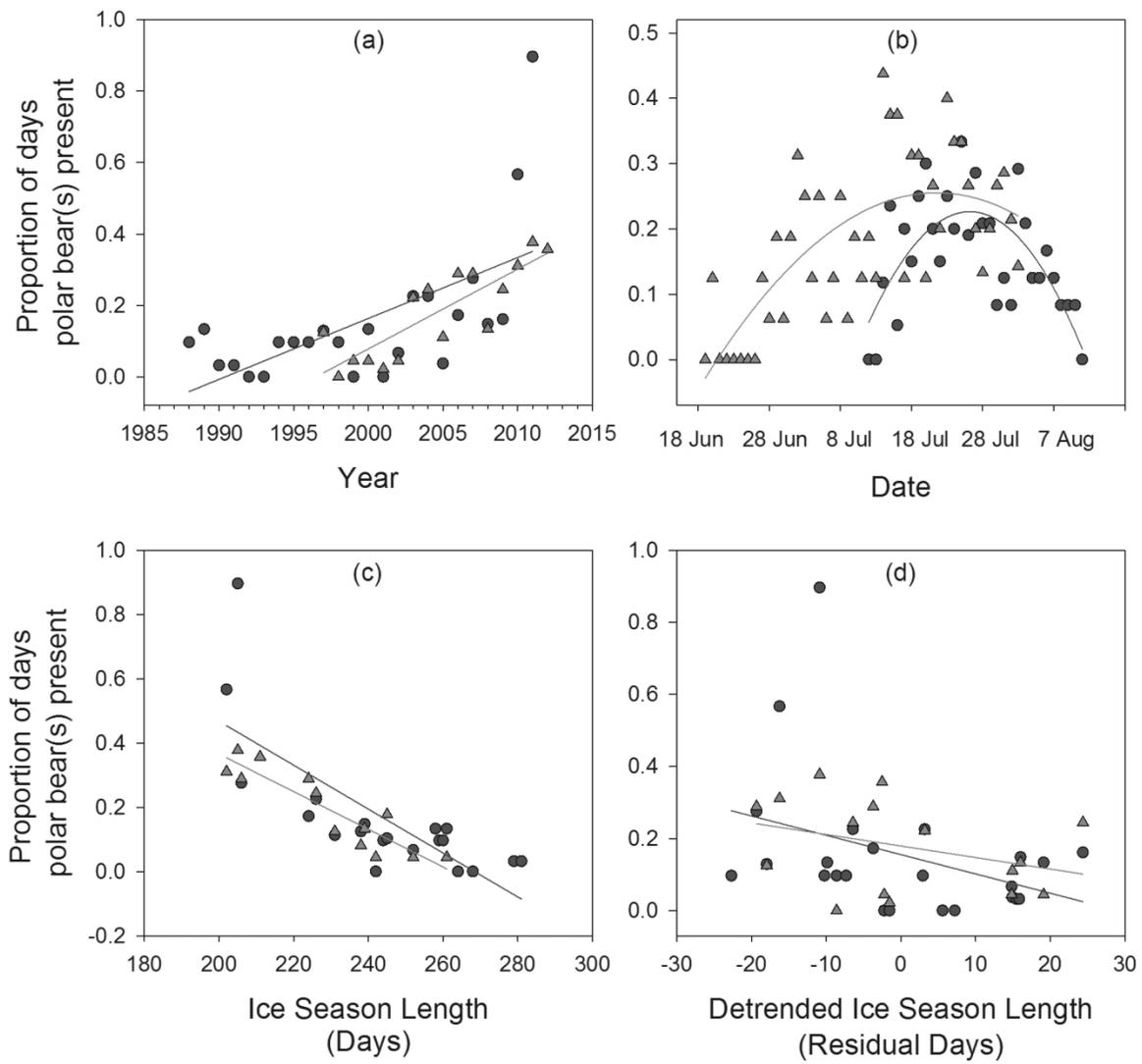
Model averaged regression coefficients indicated an increasing probability of bear incursions with advancing year ( $\beta_{\text{YEAR}} = 0.152$ , 95% CI: 0.111 to 0.192) (Appendix B, Table B.7), which was reflected in raw data plots illustrating summary proportions parsed by site and fit with a linear model (Figure 4.3a; Appendix B, Table B.8). We also estimated an increasing probability of bear incursions with advancing date during the incubation period ( $\beta_{\text{DAY}} = 0.221$ ,



**Figure 4.2.** Trends in annual sea ice coverage.

*Decline in annual sea ice coverage in Northern Hudson Bay Narrows, Canada from 1988 to 2012 quantified in relation to the number of days that >30% of the sea surface was covered by ice each winter.*

95% CI: 0.145 to 0.297), but a declining probability late in the breeding season when eggs begin to hatch or are lost to predators ( $\beta_{\text{DAY}^2} = -0.003$ , 95% CI: -0.004 to -0.002). Summary proportions fit with a quadratic equation indicated that bear incursions peaked in mid-Jul during late incubation (Figure 4.3b; Appendix B, Table B.8). We also found that the daily probability of bear incursion was higher on Mitivik Island than Cape Pembroke ( $\beta_{\text{SITE}} = 0.382$ , 95% CI: 0.097 to 0.669); however, we estimated a negative interaction between site and day, suggesting later arrival and a greater rate of increase with advancing date at Cape Pembroke ( $\beta_{\text{SITE*DAY}} = -0.011$ , 95% CI: -0.023 to -0.001). In accordance with the main prediction of our study, we estimated a negative association between bear incursions and detrended ice cover ( $\beta_{\text{ICECOVER}} = -0.034$ , 95% CI: -0.051 to -0.016) (Figure 4.3c and d).



**Figure 4.3.** Frequency of polar bear incursions in relation to year, date, and ice conditions.

*Summary proportions for the frequency with which one or more polar bears was observed during the nest incubation period (BEARDAY) at the Mitivik Island common eider colony (triangular symbols) and Cape Pembroke thick-billed murre colony (circular symbols) in relation to (a) year (b) day of the breeding season, (c) ice season length (number of days ICECT >30%), and (d) detrended ice season length as determined by residual regression.*

## **Extent and ecological correlates**

Our broad scale geographic sampling conducted in 2010 through 2012 included 230 islands supporting >32,500 common eider nesting pairs (Appendix B, Table B.2). These years coincided with the three lowest early summer ice coverage extents on record in Northern Hudson Bay Narrows and 3 of the 4 lowest ice coverage extents on record for Hudson Strait. However, variation in ice coverage was apparent among locations and across years, with 2012 being a relatively heavy ice year (IceCT > 70% on 25 Jun) in the Foxe Peninsula, South Baffin, and Frobisher Bay survey zones (Appendix B, Figure B.2).

During the course of our surveys we observed 22 polar bears (including 4 cubs) on 16 islands with eider colonies. Indirect evidence for the presence of bears was observed on a further 63 islands and polar bear presence (direct + indirect evidence) was estimated on 34% of islands in total (Table 4.1). We observed 2 foxes and encountered indirect evidence of fox presence on a further 24 islands (combined prevalence 11%). Gulls were observed on or flying above 160 islands and indirect evidence of gull presence was noted on a further 58 islands (combined prevalence 95%).

**Table 4.1.** Prevalence of predator sign on eider nesting colonies.

*Percentage of islands on which evidence of polar bear, fox or gull presence was detected during surveys in the Hudson Strait-Northern Hudson Bay Narrows region of the Canadian Arctic during July 2010, 2011 and 2012.*

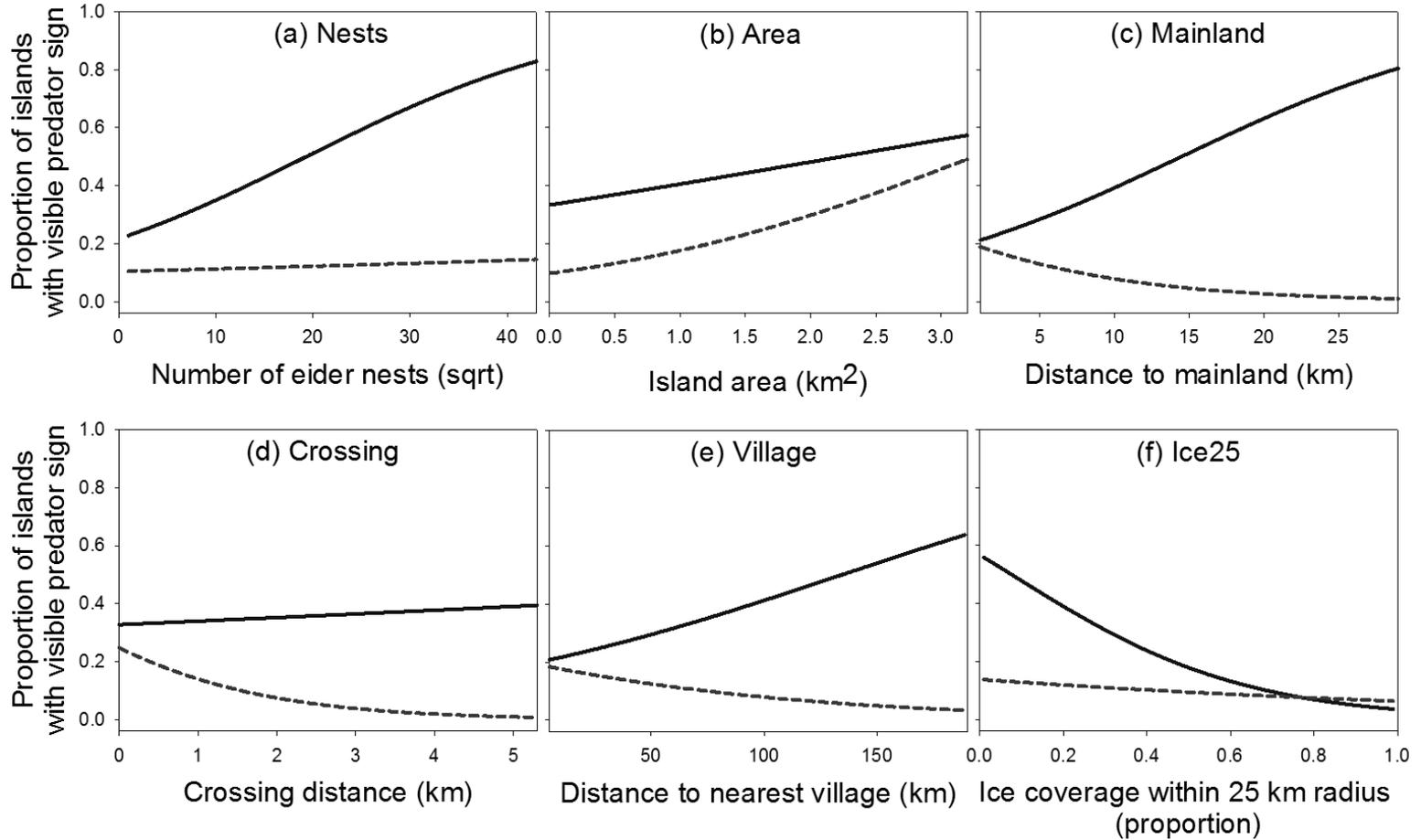
| Species group | Type of evidence   | Description of evidence                                    | # Islands | Percent of islands <sup>†</sup> |       |
|---------------|--|--|-----------|---------------------------------|-------|
| Polar bear    | Total  | All  | 79        | 34.3%                           |       |
|               | Direct   | Live bear ( $n = 22$ )                                     | 16        | 7.0%                            |       |
|               | Indirect   | Faeces   |           | 31                              | 13.5% |
|               |  | Tracks or digging  |           | 6                               | 2.6%  |
|               |  | Fur  |           | 2                               | 0.9%  |
|               | Depredated nests with direct sighting or faeces within a 1 km radius |  | 24        | 10.4%                           |       |
| Fox           | Total  | All  | 26        | 11.3%                           |       |
|               | Direct   | Live fox ( $n = 2$ )                                       | 2         | 0.9%                            |       |
|               | Indirect   | Active den   |           | 1                               | 0.4%  |
|               |  | Faeces   |           | 5                               | 2.2%  |
|               |  | Fur  |           | 3                               | 1.3%  |
|               | Depredated nests with direct sighting or faeces within a 1 km radius |  | 15        | 6.5%                            |       |
| Gull          | Total  | All  | 218       | 94.8%                           |       |
|               | Direct   | Live gull  | 160       | 69.6%                           |       |
|               | Indirect   | Depredated nests with direct sighting within a 1 km radius | 58        | 25.2%                           |       |

<sup>†</sup> Number of islands with indicated evidence of predator presence divided by the total number of islands surveyed ( $n = 230$  islands).

Our GLMM examining variation in the prevalence of BEARSIGN in relation to colony and landscape attributes indicated that nest abundance, distance to the mainland, distance to the nearest village, and ice conditions were important predictors ( $\log(L) = -117.74$ ,  $K = 6$ ,  $AICc = 247.86$ ,  $w_i = 0.33$ ); however several other models also were supported by the data ( $\Delta AICc < 4$ ; Appendix B, Table B.9). In accordance with *a priori* predictions, model averaged regression coefficients indicated a negative association between BEARSIGN and ice concentration ( $\beta_{ICE25} = -0.534$ , 95% CI: -0.077 to -0.030) and positive associations with nest abundance ( $\beta_{SQRTNESTS} = 0.091$ , 95% CI: 0.047 to 0.136), distance to mainland ( $\beta_{MAINLAND} = 0.051$ , 95% CI: 0.005 to 0.097), and distance to the nearest village ( $\beta_{VILLAGE} = 0.012$ , 95% CI: 0.004 to 0.020) (Appendix B, Table B.10).

With respect to FOXSIGN, several models had a similar fit to the data (Appendix B, Table B.11). Multi-model inference and model averaging indicated that the probability of encountering fox sign increased with island size ( $\beta_{AREA} = 0.224$ , 95% CI: 0.082 to 0.366) and decreased with crossing distance ( $\beta_{CROSSING} = -1.486$ , 95% CI: -2.922 to -0.050). Regression coefficients could not be distinguished from zero for the other variables included in our analysis (Appendix B, Table B.12). Logistic regression of pair-wise relationships between the predictors and response variable illustrates patterns for both bears and foxes within the range of conditions that we sampled (Figure 4.4).

Bears were observed to consume eggs on multiple occasions and the combined effects of bear predation and gull predation caused near total reproductive failure in several instances. For example, we surveyed an island east of Cape Dorset (64.06° N, 73.53° W) on 11 July 2011.



**Figure 4.4.** Ecological correlates of predation risk on eiders by polar bears and foxes.

*Probability of encountering polar bear sign (solid lines) or fox sign (dashed lines) on eider colonies in relation to (a) the number of eider nests, (b) island size, (c) distance to the mainland, (d) crossing distance at low tide, (e) distance to the nearest Inuit village, and (f) sea ice concentration on 25 Jun within a 25 km radius of the island.*

**Table 4.2.** Eider nest success estimates in relation to predator sign.

*Proportion of nests remaining active (nest success) in relation to the types of predator sign found on islands containing common eider colonies.*

| Type of predator sign | Islands surveyed | Total nests | Nest success ( $\pm$ SE) |
|-----------------------|------------------|-------------|--------------------------|
| No predator sign      | 15               | 183         | 0.72 ( $\pm$ 0.03)       |
| Gull only             | 114              | 14,416      | 0.66 ( $\pm$ 0.01)       |
| Fox and gull          | 22               | 3,041       | 0.39 ( $\pm$ 0.02)       |
| Bear and gull         | 75               | 14,441      | 0.22 ( $\pm$ 0.01)       |
| Bear, fox and gull    | 4                | 511         | 0.06 ( $\pm$ 0.01)       |
| Total                 | 230              | 32,592      | 0.44 ( $\pm$ 0.01)       |

We counted 536 eider nests, among which 334 were active. Fresh bear faeces containing egg shell fragments were found on the island and we counted >30 gulls, many of which were actively hunting eider eggs. We returned to the same island two days later and observed a bear on the island eating eggs and >50 gulls present. We resurveyed the colony after the bear left of its own accord and counted 24 active nests. This example was not unique, but was among the most definitive cases of near total nest destruction on a colony.

The proportion of nests remaining active at the time of survey, which was our index of NESTSUCCESS, varied substantially in relation to the types of predator sign encountered, as well as nest abundance and survey date ( $\log(L) = -38.49$ ,  $K = 7$ ,  $AICc = 91.50$ ,  $w_i = 0.68$ ) (Appendix B, Table B.13). Model averaged regression coefficients indicated lower nest success on islands with both gull and bear sign ( $\beta_{\text{GULL\&BEAR}} = -2.612$ , 95% CI: -3.962 to -1.254) or both gull and fox sign ( $\beta_{\text{GULL\&FOX}} = -1.772$ , 95% CI: -3.251 to -0.285) compared to islands with gull sign alone ( $\beta_{\text{GULL}} = -0.058$ , 95% CI: -1.787 to 0.637) (Appendix B, Table B.14). The proportion of nests remaining active decreased with advancing date ( $\beta_{\text{DATE}} = -0.091$  95% CI: -0.148 to -0.035) as our surveys were conducted during incubation and simultaneous with the arrival of nest predators. We also estimated positive association between nest success and nest abundance within a colony ( $\beta_{\text{SQRTNESTS}} = 0.043$ , 95% CI: 0.004 to 0.081) despite the fact the bear sign was positively correlated with nest abundance. Models including a survey zone effect were not well supported by the data. Raw data summaries were consistent with modeled results. We estimated that nest success ( $\pm$ SE) was 3 times lower on islands with both bear and gull sign (proportion of nests surviving to the date of survey =  $0.22 \pm 0.01$ ) compared to islands with gull sign alone

( $0.66 \pm 0.01$ ) and was roughly 1.5 times lower on islands with both fox and gull sign ( $0.39 \pm 0.02$ ) compared to islands with gull sign alone (Table 4.2).

## **DISCUSSION**

Predictions about biological responses to climate change focus largely on the environmental tolerances of individual species (Thomas et al. 2004). The circumpolar Arctic is experiencing major, ongoing reductions in both annual and multi-year sea ice (Meier et al. 2007) with low latitude regions being most impacted (Markus et al. 2009). Hudson Strait and Northern Hudson Bay Narrows, where our study was conducted, are within a region of the Canadian Arctic that has undergone a nearly two month reduction in annual ice cover over the past three decades (Sahanatien and Derocher 2012). Substantial open water areas are now routinely encountered in May and the nearshore seasonal ice environment upon which polar bears depend has been drastically altered.

Changing climatic conditions can also lead to shifts in interspecific interactions that influence population and community dynamics (Harley et al. 2011). Depredation of colonial nesting bird eggs by polar bears is not a new phenomenon; indeed, documented accounts date back more than a century (Lønø 1970). However, there is considerable evidence to suggest that bears are becoming more frequent visitors to bird colonies during the nesting season (Stempniewicz 2006, Drent et al. 2008, Rockwell and Gormezano 2009, Smith et al. 2010) and we demonstrated that the frequency of bear visits is negatively correlated with sea ice coverage. Ice-free seasons are not only becoming longer, freeze-up and break-up dates are also

becoming more variable and we measured remarkable similarity in bear attendance patterns at widely separated locations involving diverse avian species. We interpret our findings as evidence for an environmentally driven shift, wherein prey of comparatively low energetic value (bird eggs) are actively sought by polar bears when ecological conditions (lack of sea ice) prevent them from acquiring sufficient energy reserves from their preferred prey item (seals).

The negative association between bear incursions and sea ice conditions held after controlling for co-occurring temporal trends, thus constituting strong support for a causal effect of ice coverage on bears' decision to consume eggs. However, year-effects remained evident, suggesting that variables not included in our models also influenced the observed patterns. For example, increasing polar bear numbers or declining seal numbers could contribute to the increased prevalence of bears on bird colonies. Available data are insufficient to estimate polar bear population trends with adequate precision in our study area; however, there is no evidence to support an increase in bear population size approaching the magnitude of increase that we documented in bear visits at Mitivik Island and Cape Pembroke (Obbard et al. 2010). More plausible factors to consider include increased duration of stay by individual bears on bird colonies and a tendency for bears once having discovered a resource to return in subsequent years and pass information from mother to offspring.

Broad-scale geographic sampling of common eider colonies during years of record low ice coverage indicate that egg depredation by polar bears is extensive and that when bears raid eider colonies nesting success is severely reduced. Population modelling suggests that the species' population growth rate is more sensitive to variations in adult survival than variations

in annual productivity; however, retrospective analyses based on field data indicate that survival rates have proven relatively invariant over time, whereas fluctuations in reproductive success have been a primary driver of changes in population size (Wilson et al. 2012). Climate-influenced variation in rates of egg depredation strongly influence eider population dynamics (Hanssen et al. 2013) and given the magnitude of egg loss that we observed on heavily depredated colonies, local extirpation would be expected within only a few generations if it were to continue unabated. However, predicting the population-level response of eiders, as well as other Arctic-nesting birds, to increased nest predation by polar bears requires multiple considerations.

We measured considerable spatial- and habitat-related variation in bear prevalence on eider colonies. Eiders nesting on islands nearer to shore and in closer proximity to Inuit villages were less vulnerable to depredation by bears than eiders nesting on islands further from the mainland and nearer seal hunting grounds. In addition, eiders nesting solitarily or in small aggregations were largely ignored by bears. These observations suggest that distributional and density dependent factors set a lower threshold for bears' interest in eider eggs that we predict will moderate population impact.

While ground nesting species like common eiders may be particularly vulnerable to egg depredation by polar bears; bears have also proven an emerging conservation concern for cliff nesting thick-billed murre. Gaston and Elliot (2013) attributed breeding failure of up to 30% of the murre population at Cape Pembroke to polar bears during years of high bear visitation. In the case of murre, egg loss resulted from a combination of direct depredation and as a by-

product of murrets' fleeing from bears and in the process dislodging their own eggs. However, the steepest and narrowest cliff ledges located in the core of the colony have remained largely unaffected by bears. Thus, murrets appear less susceptible than eiders to large scale population reduction due to differences in nesting habitat.

Relationships between changing ice conditions and access to eggs by different predator species are also potentially important. Foxes are traditional egg predators for eiders and eiders nest on islands in large part to minimize fox depredation (Goudie et al. 2000). In this study, we estimated a greater prevalence of bear sign (34%) than fox sign (11%) on eider colonies overall; however, our results pertain only to visible sign and the species likely differ in their detectability. Foxes tend to cache eggs and we observed fewer fox tracks and faeces on the islands where such signs were encountered than was the case for polar bears, which were observed to destroy large numbers of eggs and left abundant sign. Unresolved issues with detectability notwithstanding, we believe our results reflect biological reality. Advancing spring ice melt has been associated with reduced access to islands by foxes (Hanssen et al. 2013). Open water is a barrier for foxes, whereas swimming a distance >1 km to reach an eider colony is not a significant impediment for a polar bear. Thus, the same forces exacerbating predation risk from bears appears to be reducing predation risk from foxes.

Our results demonstrate how the direct effects of sea ice loss on polar bears are having unanticipated indirect effects on breeding birds. While the nutritional benefits to bears are not known, our results clearly demonstrate that egg depredation is not limited to a few bears or a handful of nests. Our results are consistent with assertions that polar bears are experiencing

difficulty meeting their energetic demands in locations where ice free seasons have grown significantly longer. Perhaps most importantly, they highlight the importance of incorporating interspecific interactions into predictions about the ecological impacts of changing environmental conditions in a rapidly warming Arctic.

## Chapter 5: Demographic impact and potential constraints population recovery following a highly virulent disease epidemic

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### ABSTRACT

Emerging infectious diseases are on the rise globally; however, determining demographic impacts and the influence of disease on wildlife population dynamics remains a significant challenge. In this study, I quantified the impact newly emerged avian cholera outbreaks on the survival, productivity, and population growth rate of northern common eiders (*Somateria mollissima borealis*) in the Canadian Arctic. Mark-resight and nest survival models were used to estimate patterns of annual variation in vital rates at a long-term study site monitored before, during, and after a highly virulent multi-year epidemic. I assessed the influence of avian cholera prevalence in comparison to other potential limiting factors, including harvest, predation, and summer weather conditions to identify drivers of variation in demographic performance. The appearance of avian cholera resulted in a precipitous decline in the survival rates of both male and female eiders. A negative interaction between the effects of avian cholera and harvest also was estimated, suggesting additive mortality. Avian cholera did not have an appreciable influence on eider nesting success, which was determined primarily by clutch initiation date, weather conditions, and the frequency of polar bear (*Ursus maritimus*) incursions onto the colony. The latter has exhibited a directional increase in association anthropogenically-driven environmental change and as such constitutes a shift beyond the normal scope of annual

variability. The arrival of avian cholera coincided with a marked decline in eider abundance, which to date has not been offset by a compensatory demographic response.

## **INTRODUCTION**

Infectious disease can be important regulator of wildlife population dynamics (Daszak et al. 2000, Dobson and Foufopoulos 2001). Emerging infectious diseases, defined as infections that are increasing in incidence or expanding geographic or host range (Morse 1995), are a particular concern in conservation because their causes and effects rarely operate in isolation. Anthropogenic factors, such as habitat loss, climate change, overexploitation, invasive species and environmental pollution may all contribute disease emergence (Daszak et al. 2001, Smith et al. 2009). Disease epidemics can greatly impact local species populations by causing temporary or permanent declines in abundance and the presence of multiple stressors in a biological system can lead to more severe and longer epidemics (Daszak et al. 2001, Smith et al. 2009, Van Bresse et al. 2009).

Avian cholera is an infectious disease of birds caused by the bacterium *Pasteurella multocida* (Friend et al. 2001, Samuel et al. 2007). Recently, the disease has been reported on breeding colonies of the northern common eider (*Somateria mollissima borealis*) in the eastern Canadian Arctic (Chapter 3, Buttler et al. 2011, Harms 2012), where it has been a major source of mortality over the past decade (Chapter 2, Descamps et al. 2012). The common eider is a sea duck that has a circumpolar distribution and breeds in Arctic and temperate coastal zone areas of the northern hemisphere. Avian cholera has circulated in temperate-breeding common

eiders since at least the 1960s in Western Europe and the east coast of Canada and the United States (Reed and Cousineau 1967, Korschgen et al. 1978, Swennen and Smit 1991, Christensen et al. 1997, Pedersen et al. 2003, Québec Management Plan for the Common Eider 2004). While the source of *P. multocida* in the Arctic-breeding northern common eider population remains uncertain (Harms 2012), the bacterium has been isolated from migratory geese on breeding grounds in the western Canadian Arctic (Samuel et al. 1999) and carrier birds (i.e., individuals that harbor disease organisms within their body without manifest symptoms and thus acting as distributors of infection) are suspected as having a role in long-distance dispersal in waterfowl (Korschgen et al. 1978, Samuel et al. 2005).

Quantifying the population impacts of an infectious disease requires either direct monitoring of traits of infected versus uninfected individuals (e.g., Rachowicz et al. 2006, Cross et al. 2009, Atkinson and Samuel 2010) or determining changes in demographic parameters following disease outbreaks in populations under long-term study for which data are available from before the start of the epidemic (e.g., Faustino et al. 2004, Wilmers et al. 2006, Lachish et al. 2007). Such investigations require field estimates of the vital rates, together with information about transmissibility (e.g., the force of infection or basic reproductive number  $R_0$ ) as a function of disease prevalence. While rarely undertaken, the studies which have been conducted emphasize the complex nature of interactions between the endogenous components of the host-pathogen relationship (e.g., determinants of transmission, virulence, and immunological recovery), exogenous components that drive the system (e.g., biotic factors such as competition for resources and trophic interactions or abiotic factors such as

environmental conditions), and life history traits of hosts (e.g., intrinsic rate of population growth or seasonal breeding and migration patterns) as determinants of population impact (Faustino et al. 2004, Wilmers et al. 2006, Samuel et al. 2011).

Common eider life history is characterized by stable, high adult survival and variable, but generally low reproduction due to the boom-bust nature of nest success and duckling survival (Coulson 1984, Iles 2013a, Coulson et al. 2010, Wilson et al. 2012). Most individuals nest colonially, and the northern subspecies is a seasonal migrant which breeds primarily on small offshore islands (Goudie et al. 2000). To date, the effects of anthropogenic climate change have been more pronounced in high latitude regions than other regions of the globe (Hansen et al. 2006, Mayewski et al. 2009) and climate-driven environmental changes have the potential to influence Arctic species disproportionately compared to species in more temperate regions (Kausrud et al. 2008, Post et al. 2009). In regard to infectious disease, most high latitude species are thought to have evolved with less exposure to infectious pathogens than temperate and tropical species, which potentially reduces their immune capabilities (Acevedo-Whitehouse and Cunningham 2006, Meyer-Lucht and Sommer 2009). In this context, there is concern that northern common eiders may be particularly vulnerable to the demographic perturbation caused by avian cholera and its interaction with other stressors in the environment (Descamps et al. 2012).

The detection of avian cholera in 2005 in a northern common eider breeding population subject to monitoring before the epidemic began and continued until disease mortality abated provide a valuable opportunity to examine the effects of this disease on demographic

parameters. In this study, I report on the impact of avian cholera on Mitivik Island, Nunavut, a colony of intensively trapped and individually marked eiders. My specific objectives are to use mark-resight models and nest monitoring data to quantify patterns of variation in key vital rates (adult survival, site fidelity, nest success) and then evaluate potential biotic and abiotic predictors of variation to determine the relative importance of avian cholera as a driver of population dynamics in relation to other factors influencing population growth and decline.

## **METHODS**

### **Study system background**

Common eiders spend most of their annual cycle in marine waters, coming ashore only during the breeding season (Dickson 2012). For northern common eider subspecies (*S. m. borealis*) the main factor regulating population growth is thought to be hunting (Gilliland et al. 2009). The species is heavily hunted on its wintering grounds in Atlantic Canada and west Greenland (Mekel 2004, Gilliland et al. 2009). Population declines in 1990s led to changes in hunting regulations in Greenland, where up to 60% of common eiders affiliated with Mitivik Island are known to overwinter (Mosbech et al. 2006, G. Gilchrist, Environment Canada *personal communication*). Following these changes, population growth at colonies in Greenland was as high as five-fold (Merkel 2010, Burnham et al. 2012).

Common eider nesting is primarily colonial on small islands in coastal areas (Goudie et al. 2000). The use of small, near shore islands by eiders is thought to be a behavioral adaptation to minimize egg depredation by foxes (*Vulpes* spp.) (Schamel 1977, Hanssen et al. 2013) and

colonial nesting is generally regarded as an adaptation to enhance the survival of adults, nestlings and eggs through predator dilution and increased vigilance (Serrano et al. 2005). In the Arctic ecosystem, eider breeding phenology is tightly correlated with sea ice conditions (Love et al. 2010). By timing clutch initiation to coincide with sea ice melt eiders reduce their exposure to foxes, which require landfast or drifting sea ice to access islands (Hanssen et al. 2013). Temporal advancement of spring ice break-up associated with climate change has been correlated with increased depredation of colonial bird eggs by polar bears (*Ursus maritimus*) (Rockwell et al. 2009, Iverson et al. 2014). The frequency of polar bear depredation of adult eiders remains undetermined; however, the presence of either a fox or polar bear on a bird colony during the nesting season can lead to total or near total breeding failure (Careau et al. 2008, Iverson et al. 2014).

Local weather conditions during the nesting season also are known to be an important driver of eider population dynamics. Adult survival is generally robust to variability in summer weather conditions (Coulson et al. 2010), whereas pronounced oscillations in duckling production have been associated with weather during the late spring and summer. Variable patterns have been described among locations. For example, in Iceland, warm, wet summers have been found to have a negative effect on eider productivity (Jónsson et al. 2013), whereas in Hudson Bay, Canada, warm, wet conditions in late spring have been associated with increased nest success (Iles et al. 2013a).

Finally, infectious disease is recognized as important mortality factor in common eiders (Friend et al. 2001). In addition to avian cholera, mass mortality events have been associated

with outbreaks of Wellfleet Bay virus among common eiders nesting on the Atlantic coast of the United States (Allison et al. 2015) and avian adenovirus on the Baltic Sea coast of Finland (Hollmén et al. 2003). While mass mortality events associated with avian cholera have been reported on numerous occasions, few studies have investigated their demographic impact. In a Danish study, Tjørnløv et al. (2013) estimated a 14% decrease in female annual survival from a pre-outbreak baseline and identified avian cholera as major constraint on population growth. Descamps et al. (2011, 2012) assessed hatching success, duckling survival, and adult survival, for northern common eiders at the height of the epidemic on Mitivik Island and found that hatching success among birds that did not succumb to disease was not affected by the disease, whereas duckling survival (Descamps et al. 2011) and breeding female survival (Descamps et al. 2012) were severely reduced.

### **Field data collection methods**

Mitivik Island is a small (24 ha), low-lying (<8 m elevation) island located within the East Bay Migratory Bird Sanctuary, Nunavut, which supports the largest known breeding colony of northern common eiders in the Canadian Arctic (Environment Canada. 2012). Biologists arrive on the island each year by aircraft in late May (before eiders lay their eggs) and depart in early August (after most ducklings have hatched). During the pre-laying and early incubation periods, female and male eiders are captured in flight nets. Captured eiders are marked with standard United States Fish and Wildlife Service metal bands, as well colored plastic (Darvic) alpha-numeric tarsal bands. In addition, since 2002, female eiders have been fitted with colored

plastic nasal tags, which allow birds sitting on nests to be individually identified. The nasal tags are attached using UV-degradable surgical monofilament, which ensures their loss before fall migration, whereas the Darvic tarsal bands are retained for multiple years (estimated 87% retention for  $\geq 5$  yr; Breton et al. 2006).

Seven observation blinds surround the core breeding area on Mitivik Island. From these blinds, biologists use binoculars and 20X to 60X spotting scopes to make twice daily scans of the island, including five permanent monitoring plots, which encompass approximately 10% of available breeding habitat. Within the permanent monitoring plots, nests are counted to derive an index of eider density from which changes in annual abundance are inferred. Visual searches of the entire colony (>80% visible from the blinds) are also made from the blinds to determine the location and status nasal-tagged females nesting outside of the permanent plots. These scans permit the identification of nesting behavior (prospecting, incubating, or absent), as well as nest fates (described below).

In addition to monitoring the activity of nesting females, the presence and number of terrestrial predators (Arctic foxes *Vulpes lagopus* and polar bears *Ursus maritimus*) is recorded in daily log books (Chapter 4). I used these data to quantify fox and bear presence on Mitivik Island (proportion of days on which one or more individuals was visually observed within specified monitoring intervals). Predator observation was aided by Mitivik Island's small size and the scheduling of research activities around the clock, particularly during the pre-laying and early incubation periods. While foxes tend to be more secretive and less detectable than polar

bears when raiding bird colonies, I considered the visual observation data sufficiently unbiased for use as an index of predator presence (Smith et al. 2010).

Biologists also record water depth for five ponds situated within the colony using a categorical ranking system (0 = pond is dry; 1 = pond 1 to 15 cm deep; 2 = pond >15 cm deep (a depth suitable for swimming); and F= frozen). I used the pond depth data (categories 0-2) to derive an annual index of freshwater availability. Daily temperature and precipitation readings are collected at the nearby Coral Harbour Airport weather station (Canadian Meteorological Service Station ID 230100), which is located 78 km NE of Mitivik Island. I used the weather station data to derive an index extreme heat, defined as the number of days on which the daytime high temperature exceeded 20° C, and an index of accumulated precipitation, defined as the total amount of rainfall (mm), within specified periods of interest for my analyses.

## **Annual survival**

### ***Survival estimation***

I estimated apparent survival ( $\phi$ ) and resighting probability ( $p$ ) from live encounter data over annual intervals using a Cormack–Jolly–Seber (CJS) modelling formulation (Lebreton et al. 1992). My dataset included encounter histories for 3390 female and 2412 male common eiders banded and released on Mitivik Island during the 2000 to 2012 breeding seasons (Appendix C, Table C.1). For clarity purposes, I refer to an annual cycle in terms of the year of capture, for example the year 2005 would be defined as the 12-month period beginning 1 July 2005 and ending 30 June 2006.

When conducting my analysis, I developed a candidate set of models to evaluate variation in  $\phi$  and  $p$  as functions of year, sex (male vs. female), and time-since marking. Goodness-of-fit testing was performed on the global model [Female ( $\phi_t^1, \phi_t^{2+}, p_t^1, p_t^{2+}$ ) Male ( $\phi_t^1, \phi_t^{2+}, p_t^1, p_t^{2+}$ )] using Program U-Care (version 2.3; Choquet et al. 2009) (Appendix C, Table C.2). Time-since-marking was included to determine whether newly marked individuals differed in apparent survival during their first year ( $\phi^1$ ) compared with subsequent years ( $\phi^{2+}$ ) (Sandercock 2006). I expected a significant proportion of the male population to be comprised of transient individuals (recognized by  $\phi^1 < \phi^{2+}$ ) (Pradel et al. 1997, Prévot-Julliard et al. 1998). Female eiders exhibit much higher natal philopatry and site fidelity than males (Goudie et al. 2000) and I did not expect a transient effect for females.

Most re-encounters with previously banded individuals were visual sightings of colored alpha-numeric tarsal bands (>90%). Band wear has been identified as a factor potentially biasing live encounter rates for marine birds fitted with Darvic tarsal bands (Regehr and Rodway 2003, Breton et al. 2006) and biologists stationed on Mitivik Island anecdotally observed that Darvic bands applied before 2000 were more brittle and subject to band wear or loss than bands applied thereafter (G. Gilchrist, Environment Canada, *personal communication*); therefore I censored pre-2000 marking records from the dataset and modeled heterogeneity in resighting probabilities as a form of trap avoidance in previously banded cohorts ( $p^1 > p^{2+}$ ) (Choquet et al. 2009).

Within the CJS framework it is important to note that  $\phi$  is the product of ‘true’ survival ( $S$ ) and site fidelity ( $F$ ), which cannot be disentangled without supplementary information. Because

females exhibit much higher site fidelity than male eiders I expected higher apparent survival rates among females than males and focus my data interpretation on patterns of temporal variation within, as opposed to among sexes.

My candidate set models included a series of reduced parameterizations of the global model. Model selection was based on Akaike's Information Criterion corrected for sample size ( $AIC_c$ ), with  $\Delta AIC_c$  and Akaike weights ( $w_i$ ) used to infer model support (Burnham and Anderson 2002). When estimating year-specific parameters, I utilized the Markov Chain Monte Carlo (MCMC) estimation procedure in PROGRAM MARK, which is a Bayesian random effects technique useful for separating process variation from sampling error. When making my estimates I simulated single chain with 4,000 tuning samples, a burn-in period of 1,000 samples, and 10,000 samples to generate the posterior distributions. I specified eight hyperdistributions (one for each sex-cohort variable in the general model). All prior distributions were assumed to be normally distributed on a logit scale with mean ( $\mu$ ) 0.8 and variance ( $\sigma$ ) 0.4.

### ***'True' survival and site fidelity***

I conducted a supplementary analysis on the data used in my CJS model that included additional information derived from hunter harvested eiders, which I assessed using a joint live encounters and dead recoveries model (Burnham 1993). I did this to decompose  $\phi$  into  $S$  and  $F$  components. To facilitate my analysis I obtained hunter recovery records from the North American Bird Banding database maintained by the United States Fish and Wildlife Service (Appendix C, Table C.3). Burnham joint live encounters and dead recoveries models are heavily

parameterized and to avoid estimation bias I restricted my inquiry to a 6 yr interval spanning the arrival of avian cholera on Mitivik Island (2003 to 2008). Harvest reporting was generally robust during this timeframe, whereas during earlier and later intervals data-sparsity negatively affected model performance. My general model assumed group (female and male) and year differences for the four variables included in the model ( $S_{g^*t}$ ,  $r_{g^*t}$ ,  $F_{g^*t}$ ,  $p_{g^*t}$ ), where  $r$  signifies the harvest rate, and  $p$  signifies the live resighting rate. For the recovery parameter, I fixed annual values using the direct recovery rate (i.e., the proportion of newly released individuals reported to be harvested by hunters during the first hunting season after release) to reduce estimation error. I then compared the general model to a series of models with reduced parameterization to determine whether the probability of site faithfulness varied in relation to year, sex, and avian cholera presence.

### ***Ecological predictors of variation in annual survival***

Once having determined annual survival rates, I evaluated ecological predictors of variation using a generalized linear model (GLM). I implemented my GLM using the software program STATISTICA (Version 6, Systat Software Inc., Richmond, CA, USA). I specified resident female apparent annual survival (SURV) as the response variable (log normal distribution; logit link) and explanatory variables in included AVIANCHOLERA (the number of directly observed female mortalities detected during the breeding season as inferred from epidemic curves (Chapter 2); HARVEST (the number of newly released female eiders reported to have been harvested divided by the total number of females released), BEAR (the number of days on which one or more polar

bears was observed on the colony); Fox (the number of days on which one or more foxes were observed); PRECIP (accumulated rainfall during the breeding season); and TEMP (days with a maximum temperature  $>20^{\circ}\text{C}$ ). The covariates were calibrated to include observations occurring from 6 Jun to 4 Aug with input parameters standardized using a z-transformation  $(x - \bar{x})/SD$  to facilitate comparison of regression coefficients (i.e.,  $\beta$ s).

When developing my candidate set of models I was cognizant of over-fitting and employed a forward selection process—retaining a variable only when AICc was significantly improved by the addition of said variable. Model fit was judged by evaluating the change in AICc and evidence ratios (i.e., the Akaike weight of the model under consideration ( $w_i$ ) divided by the Akaike weight of the less parameterized model ( $w_j$ ) to which it is being compared). An evidence ratio  $E_{ij} = >1$  is indicative of improved model fit, while  $E_{ij} = <1$  indicates no significant improvement when considering the weight of evidence, model uncertainty, and the principle of parsimony (Burnham and Anderson 2002).

I began with the variables that quantified mortality directly during either the breeding season (AVIANCHOLERA) or the non-breeding season (HARVEST) under the expectation that these variables would be the strongest predictors of annual mortality rate. I then added the predator observation variables BEAR and FOX and finally the abiotic variables PRECIP and TEMP. I predicted that both avian cholera mortality and harvest mortality would be negatively associated with annual survival probability. I did not expect variation in annual survival to correlate with terrestrial predator presence or weather conditions.

## **Nest success**

### ***Nest success estimation***

Nest success (defined as the probability of nest survival from clutch initiation until at least one young is hatched) was estimated from the observation of breeding female eiders fitted with nasal tags during the 2002 to 2012 breeding seasons. To implement my analysis, I used the nest survival model in PROGRAM MARK (Dinsmore et al. 2002, Dinsmore and Dinsmore 2007), which is a generalization of the maximum-likelihood estimator of Bart and Robson (1982) that incorporates continuous temporal variation within a nesting period. Input data include the day on which each nest was found, the last day the nest was known to be active, the day the nest was last checked, and nest fate (0 means successful and 1 means destroyed or unsuccessful).

Data were available for 976 female eiders (range: 35 to 152 nests per year). I included only information for females the first time their nest was monitored to avoid repeated measures on the same individual. I considered the sample random with respect to location within the colony because females were 'selected' at the time of capture and subsequently chose their own nest sites. Clutch initiation dates were assigned as the first day of three consecutive presences records (Buttler et al. 2011). Nests were found  $2.1 \text{ d} \pm 4.2 \text{ SD d}$  after clutch initiation on average (based on back calculation for successful nests and assuming a 26 d incubation interval; Bottitta 1999). A nest was considered successful if females were seen continuously for 24 d and/or pipping eggs or ducklings were observed (Buttler 2009, Descamps et al. 2011). Model output is in the form of daily nest survival (DNS) probability estimates, with values generated from the earliest date of nest discovery (6 Jun) to the latest date of nest

monitoring (4 Aug) across all years of study. The probability that a female initiating incubation on a specified date within this interval is calculated by determining the product of DNS values over the length of a complete incubation cycle. For example nest success of females initiating incubation on 1 Jul (the 26<sup>th</sup> day of study interval) would be the product  $NS_{26} = DNS_{26} * DNS_{27} * DNS_{28} * \dots * DNS_{52}$ .

In my candidate set of models, I began with a simple model that assumed constant daily nest survival and no annual variation (NULL). To test for annual patterns of nest survival, I added year to the model (YEAR). I then considered models that allowed nest survival to vary with date over the breeding season in a linear (DATE) and a curvilinear (quadratic) pattern (DATE<sup>2</sup>). I also included interactions between seasonal trends and year to test whether seasonal patterns varied annually (YEAR + DATE) and (YEAR + DATE<sup>2</sup>). Finally, to test whether temporal change in nest survival might be related to nest age (i.e., the number of days since eggs were laid as opposed to date of season), I included a model with a quadratic effect of nest age [YEAR + NESTAGE<sup>2</sup>]. Separating time of season and nest age effects can be difficult because nests at the start and end of the season will primarily be in the laying and late incubation stages, respectively. However, over 11 years of study there was considerable inter-annual variability in the mean and variance in clutch initiation dates and for most days of the breeding season there were adequate samples to consider separate effects of nest age and time of season on nest survival probability.

When running models in PROGRAM MARK, I specified a logit link function. There is currently no suitable goodness-of-fit test for the nest survival model (Dinsmore et al. 2002), thus I have

not used one here. Model selection was again based on AICc and parameter estimation was implemented using the MCMC procedure (single hyperdistribution with  $\mu = 0.3$  and  $\sigma = 0.4$ ).

### ***Ecological predictors of variation in annual nest success***

I evaluated ecological predictors of variation in nest success using a linear mixed-effects model, which I implemented using the package lme4 in R (Bolker et al. 2009, Bates et al. 2013). In my analysis, I specified NESTSUCCESS as the response variable (log normal distribution; logit link), for which I derived separate year-specific estimates for females initiating incubation on the median date (*median*), 7 d earlier than the median date (*early*), and 7 d after the median date (*late*). I classified year as a categorical random effect (YEARRE) and assumed correlated random intercepts and slopes in relation to PHENOLOGY (a categorical variable for *early*, *median*, and *late* nesting females). Fixed effect covariates were the same as described above for my evaluation of ecological predictors of annual survival, but tailored to year-specific incubation intervals to match with associated response variables. For example, instead of evaluating all polar bear incursions during the breeding season in a given year, I summed the number of polar bear incursions during the 26 d incubation intervals of early, median, and late nesting females. An additional variable, POND (mean water depth) was included as potential predictor and HARVEST was excluded from my nest success analysis.

I predicted that nesting phenology would be a dominant driver of variation in nesting success with early-laying individuals achieving the highest nest success rates (Wilson et al. 2012). I predicted heightened nest failure in association with increased predator presence, with

early nesting females most adversely affected by the presence of foxes, and late-nesting females most adversely affected by the presence of polar bears. Finally, I predicted a negative influence on nest success in relation to the frequency of days with extreme heat or high precipitation (Jónsson et al. 2013).

### **Influence of survival and nest success on variation in population growth**

To evaluate the influence of survival and nest success on variation in population growth I used the breeding pair abundance estimates described in Chapter 2 (Figure 2.2). In that analysis, I divided the number of common eider nest starts within each permanent monitoring plot by plot area to calculate mean annual breeding pair density weighted by plot size ( $d_t$ ) and extrapolated an estimate of total abundance ( $N_t$ ) by multiply density by the total area of available habitat ( $A$ ). For this analysis, I estimated the rate of annual change assuming a geometric rate of increase (or decline) in discrete annual intervals:  $\lambda_t = N_{t+1} / N_t$ .

I then used linear regression to model the relationship between  $\lambda_t$  and resident female apparent survival during the preceding year ( $SURV$ ), nest success one year prior ( $NS$ ), and nest success three years prior ( $NS_{LAG}$ ). I included lagged nest success because eiders exhibit delayed sexual maturation, with a median age of first clutch production of three years (Baillie and Milne 1982, Hario and Rintala 2009). I used the nest success estimates derived from cumulative nest survival rates for birds initiating incubation on the median date. My *a priori* predictions were that resident female survival would be highly correlated with of annual variability in  $\lambda$  given that my study encompassed a period when mortality was abnormally high, but that  $NS_{LAG}$  would also be a significant predictor, particularly during non-outbreak years.

## RESULTS

### Annual survival

#### *Survival estimates*

The best fitting model in my Cormack-Jolly-Seber analysis of apparent survival and resighting probability was the global model ( $\log(L) = -9606.23$ ,  $K = 96$ ,  $\text{QAICc} = 21409.62$ ,  $w_i = 0.84$ ).

Goodness-of-fit testing results were consistent with *a priori* predictions and overdispersion was relatively low (median  $\hat{c} = 1.14$ ). Support also was evident for reduced model, which did not assume a time-since-marking effect for males, but was otherwise identical to the global model (Table 5.1).

MCMC-derived parameter estimates indicate that before 2006, female survival ranged from 0.87 to 0.93 with minimal inter-annual variation or difference between newly banded ( $\phi^1$ ) and previously banded females (hereafter, resident females) ( $\phi^{2+}$ ) (Figure 5.1). A precipitous decline was evident from 2006-2008, when directly observed avian cholera mortality peaked on the island. During this interval  $\phi^{2+}$  ranged from 0.53 to 0.61, while  $\phi^1$  ranged from 0.59 to 0.76, suggesting that resident females were more negatively affected by the disease than newly banded birds. After 2009, female apparent survival rates gradually increased and the difference between newly banded and resident females dissipated; however estimates for both groups remained below pre-epidemic baselines (Figure 5.1).

The apparent survival rates of males were considerably lower than those of females. Before 2006  $\phi^{2+}$  ranged from 0.65 to 0.81 for males, while  $\phi^{1+}$  ranged from 0.22 to 0.56. This difference is indicative of the presence of transient individuals within the male population.

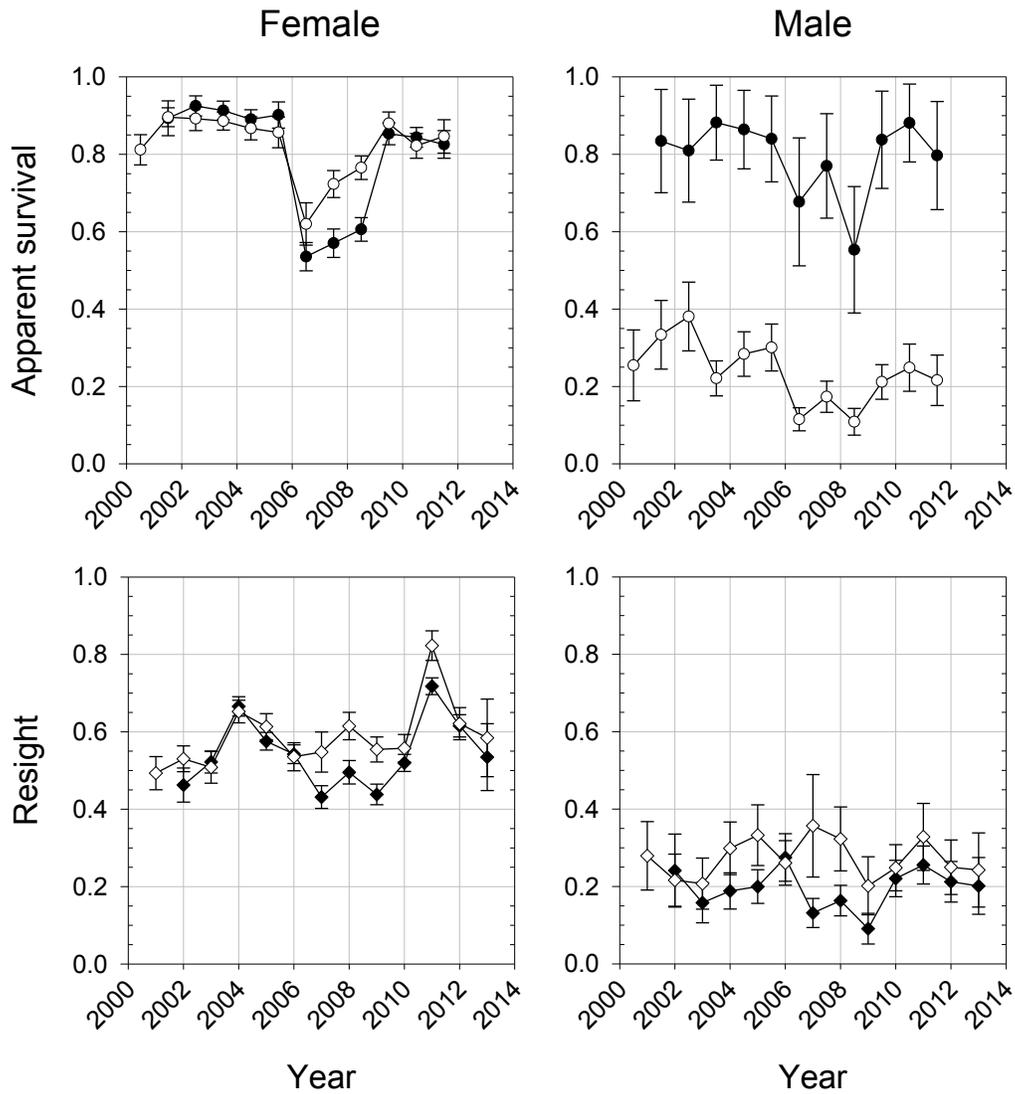
**Table 5.1.** Model rankings for apparent survival analysis in PROGRAM MARK.

*Candidate set of Cormack-Jolly-Seber models for evaluating apparent survival ( $\phi$ ) and resight ( $p$ ) estimates for common eiders at Mitivik Island, Nunavut, 2000-2012. QDeviance indicates models were adjusted for overdispersion ( $\hat{c} = 1.14$ ).*

| Model   | QDeviance | K  | $\Delta QAIC_c$ | $w_i$ |
|---|-----------|----|-----------------|-------|
| Female ( $\phi_t^1, \phi_t^{2+}, p_t^1, p_t^{2+}$ ) Male ( $\phi_t^1, \phi_t^{2+}, p_t^1, p_t^{2+}$ ) | 2003.44   | 96 | 0.00            | 0.84  |
| Female ( $\phi_t^1, \phi_t^{2+}, p_t^1, p_t^{2+}$ ) Male ( $\phi_t^1, \phi_t^{2+}, p_t$ )             | 2031.43   | 84 | 3.59            | 0.14  |
| Female ( $\phi_t, p_t^1, p_t^{2+}$ ) Male ( $\phi_t^1, \phi_t^{2+}, p_t^1, p_t^{2+}$ )                | 2033.90   | 85 | 8.10            | 0.01  |
| Female ( $\phi_t^1, \phi_t^{2+}, p_t$ ) Male ( $\phi_t^1, \phi_t^{2+}, p_t^1, p_t^{2+}$ )             | 2041.86   | 84 | 14.02           | 0.00  |
| Female ( $\phi_t^1, \phi_t^{2+}, p_t$ ) Male ( $\phi_t^1, \phi_t^{2+}, p_t$ )                         | 2069.85   | 74 | 21.72           | 0.00  |
| Female ( $\phi_t^1, \phi_t^{2+}, p_t^1, p_t^{2+}$ ) Male ( $\phi_t, p_t^1, p_t^{2+}$ )                | 2108.15   | 85 | 82.34           | 0.00  |
| Female ( $\phi_t, p_t^1, p_t^{2+}$ ) Male ( $\phi_t, p_t^1, p_t^{2+}$ )                               | 2138.62   | 74 | 90.49           | 0.00  |
| Female ( $\phi_t, p_t$ ) Male ( $\phi_t, p_t$ )   | 2219.40   | 48 | 118.69          | 0.00  |
| Female ( $\phi, p$ ) Male ( $\phi, p$ )   | 3277.07   | 4  | 1087.93         | 0.00  |

Although few male carcasses were directly observed on Mitivik Island, male apparent survival was markedly lower during outbreak. From 2006 to 2008,  $\phi^{2+}$  ranged from 0.58 to 0.76, while  $\phi^1$  ranged from 0.11 to 0.16. By 2009, survival estimates for resident males had converged with the pre-outbreak baseline, whereas estimates for newly banded males remained below the pre-epidemic baseline, suggesting a lack of recruitment by males into the population (Figure 5.1).

Resighting probabilities varied by year and averaged  $p^{2+} = 0.54 \pm 0.03$  SE (range: 0.43 to 0.72) for resident females and  $p^1 = 0.59 \pm 0.04$  SE (range: 0.49 to 0.82) for newly banded females. For males, resighting rates were lower, but followed a similar pattern wherein resighting probability was smaller for resident males,  $p^{2+} = 0.19 \pm 0.05$  SE (range: 0.19 to 0.27) than newly banded males  $p^1 = 0.27 \pm 0.08$  SE (range: 0.20 to 0.36). These results are indicative of band wear and lower probability of detection over time.



**Figure 5.1.** Apparent survival and resight rate estimates.

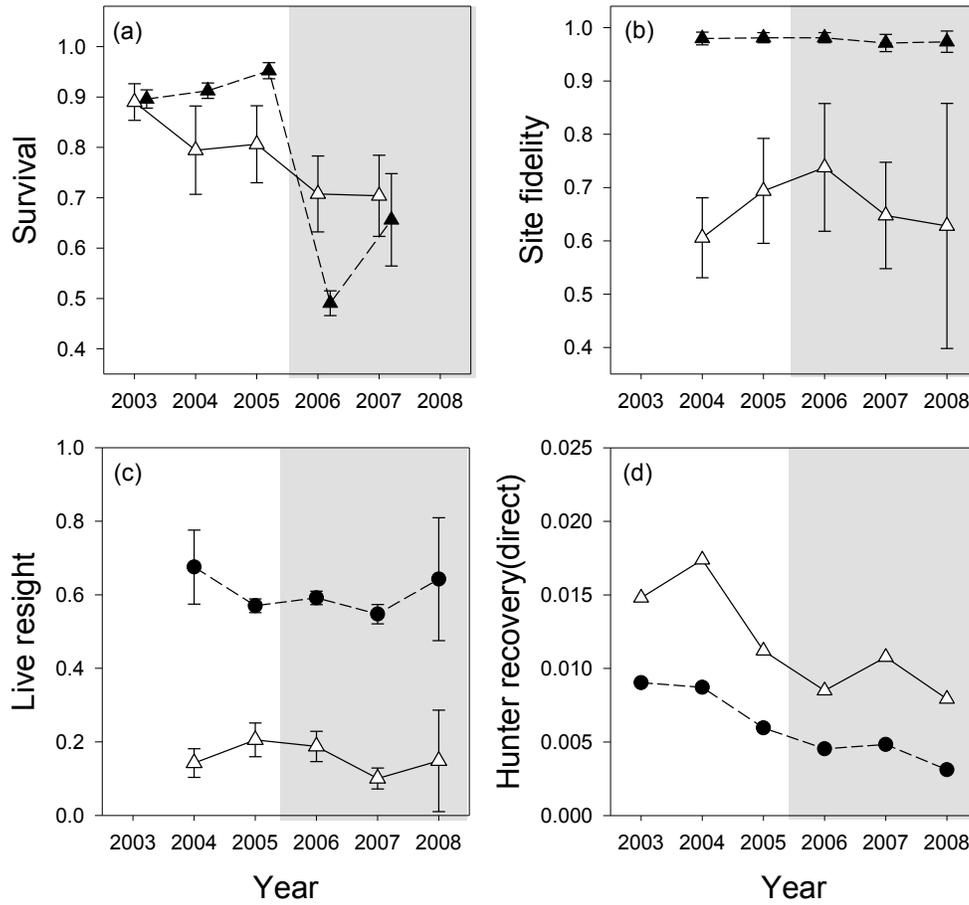
*Apparent survival ( $\phi$ ) and resight ( $p$ ) estimates for female and male common eiders captured and released at Mitivik Island, Nunavut 2000 to 2012. Parameter estimates were heterogeneous with respect to time-since-marking. White symbols denote estimates for the first year after capture and black symbols denote estimates thereafter.*

### ***True' survival and site fidelity***

The Burnham live encounters and dead recoveries model used to estimate site fidelity rates did not distinguish resident from newly banded individuals. Rather the model integrated estimates among groups in proportion to the representation in the sample as a whole. The best fitting model indicated sex-related variation in site fidelity that did not differ across years or in relation to avian cholera outbreak intensity ( $S_{g*t}, r_{g*t}, F_g, p_{g*t}$ ; ( $\log(L) = -19556.64$ ,  $AICc = 10168.87$ ,  $K = 24$ ,  $w_i = 0.73$ ).

Site fidelity for females was very high ( $F = 0.98 \pm 0.01$  SE), while for males site fidelity was considerably lower ( $F = 0.66 \pm 0.07$  SE) (Figure 5.2). 'True' survival estimates were consistent with estimates calculated under the Cormack-Jolly-Seber model after controlling for emigration (where  $\phi = S * F$ ). For females,  $S$  ranged from 0.90 to 0.95 before the epidemic from 0.49 to 0.66 at its peak. For males,  $S$  ranged from 0.81 to 0.93 before the epidemic and 0.70 to 0.75 at its peak (Figure 5.2).

Recovery rates ( $r$ ) were higher for males than females and declined over time in accordance with the phased implementation of more restrictive hunting regulations in west Greenland beginning in the early 2000s.



**Figure 5.2.** True survival, site fidelity, live resights, and dead recovery estimates.

*Northern common eider (a) survival  $S$ , (b) site fidelity  $F$ , (c) live resight  $p$ , and (d) dead recovery ( $r$ ) probabilities. Black circles represent female eiders. White triangles represent male eiders. Bars indicate standard errors. Grey shaded area indicates peak outbreak years of the avian cholera epidemic on Mitivik Island*

### ***Ecological predictors of variation in annual survival***

General linear model analysis indicated that the addition of AVIANCHOLERA to the intercept-only NULL model significantly increased model support (Table 5.2a). The addition of HARVEST did not improve model fit when considered independently, however, the interaction between AVIANCHOLERA \* HARVEST was significant. Model averaged regression coefficients indicated that each variable and the interaction were negative, which is indicative of additive mortality (Table 5.2b).

Similar to HARVEST, the addition polar bear incursions (BEAR) improved model support when considered in interaction with AVIANCHOLERA, but not on its own (Table 5.3a). Model averaged regression coefficients indicated that the effects of both variables and the interaction were again negative (Table 5.3b). The addition fox incursions (FOX) did not improve model support, nor did the addition of high temperature days (TEMP) or accumulated rainfall during the breeding period (PRECIP) (Table 5.4a, 5.4b).

**Table 5.2.** GLM results relating survival probability to avian cholera and harvest.

(a) Model selection results and (b) model averaged regression coefficients used to evaluate the effects of avian cholera mortality and harvest mortality occurring during the non-breeding season on resident female survival.

| (a) Model selection    |        |   |               |       |                  |
|------------------------|--------|---|---------------|-------|------------------|
| Explanatory variables  | log(L) | K | $\Delta AICc$ | $w_i$ | $E_{ij}^\dagger$ |
| AVIANCHOLERA           | 24.59  | 2 | 0.00          | 0.79  | $1.6 * 10^7$     |
| AVIANCHOLERA * HARVEST | 27.46  | 4 | 2.64          | 0.21  | $4.2 * 10^6$     |
| NULL                   | 6.56   | 1 | 33.13         | 0.00  | 1.0              |
| HARVEST                | 7.27   | 2 | 34.65         | 0.00  | 0.5              |

| (b) Regression coefficients |         |          |                      |
|-----------------------------|---------|----------|----------------------|
| Variable                    | $\beta$ | $\pm SE$ | t value <sup>‡</sup> |
| (Intercept)                 | -0.248  | 0.013    |                      |
| AVIANCHOLERA                | -0.212  | 0.016    | -12.873              |
| HARVEST                     | -0.010  | 0.004    | -2.648               |
| AVIANCHOLERA * HARVEST      | -0.011  | 0.005    | -1.983               |

<sup>†</sup>  $E_{ij}$  = evidence ratio ( $w_i / w_j$ ) for the model in question compared to the intercept-only NULL model; <sup>‡</sup> t value is the ratio of the regression coefficient to its standard error, where  $|t| > 1$  indicates the effect is statistically different from zero.

**Table 5.3.** GLM results relating survival probability to avian cholera and predation.

(a) Model selection results and (b) model averaged regression coefficients used to evaluate the effects of avian cholera mortality and terrestrial predators (polar bear and fox) on resident female survival.

| (a) Model selection   |        |   |               |       |              |                |
|-----------------------|--------|---|---------------|-------|--------------|----------------|
| Explanatory variables | log(L) | K | $\Delta$ AICc | $w_i$ | $E_{i,j}^+$  | $E_{i,j}^{++}$ |
| AVIANCHOLERA * BEAR   | 29.58  | 4 | 0.00          | 0.68  | $3.5 * 10^7$ | 2.2            |
| AVIANCHOLERA          | 24.59  | 2 | 1.60          | 0.31  | $1.6 * 10^7$ | 1.0            |
| AVIANCHOLERA * FOX    | 25.63  | 4 | 7.90          | 0.01  | $6.7 * 10^5$ | <0.1           |
| NULL                  | 6.56   | 1 | 34.73         | 0.00  | 1.0          | <0.1           |
| BEAR                  | 6.68   | 2 | 37.43         | 0.00  | 0.3          | <0.1           |
| FOX                   | 6.56   | 2 | 37.54         | 0.00  | 0.2          | <0.1           |

| (b) Regression coefficients |         |          |                      |
|-----------------------------|---------|----------|----------------------|
| Variable                    | $\beta$ | $\pm$ SE | t value <sup>‡</sup> |
| (Intercept)                 | -1.314  | 0.044    |                      |
| AVIANCHOLERA                | -0.198  | 0.027    | -7.3                 |
| BEAR                        | -0.026  | 0.007    | -3.7                 |
| AVIANCHOLERA * BEAR         | -0.041  | 0.066    | -0.7                 |
| FOX                         | <0.001  |          |                      |
| AVIANCHOLERA * FOX          | <0.001  |          |                      |

**Table 5.4.** GLM results relating survival probability to avian cholera and abiotic conditions.

(a) Model selection results and (b) model averaged regression coefficients used to evaluate the effects of avian cholera mortality and summer weather conditions (temperature and precipitation) on resident female survival.

| (a) Model selection   |        |   |               |       |                    |                           |
|-----------------------|--------|---|---------------|-------|--------------------|---------------------------|
| Explanatory variables | log(L) | K | $\Delta AICc$ | $w_i$ | $E_{ij}^{\dagger}$ | $E_{ij}^{\dagger\dagger}$ |
| AVIANCHOLERA          | 24.59  | 2 | 0.00          | 0.87  | $1.6 * 10^7$       | 1.0                       |
| AVIANCHOLERA * TEMP   | 26.44  | 4 | 4.70          | 0.08  | $1.5 * 10^6$       | 0.1                       |
| AVIANCHOLERA * PRECIP | 25.92  | 4 | 5.73          | 0.05  | $8.9 * 10^5$       | 0.1                       |
| NULL                  | 6.56   | 1 | 33.13         | 0.00  | 1.0                | <0.1                      |
| TEMP                  | 7.21   | 2 | 37.41         | 0.00  | 0.2                | <0.1                      |
| PRECIP                | 6.76   | 2 | 39.69         | 0.00  | 0.1                | <0.1                      |

| (b) Regression coefficients |         |          |                      |
|-----------------------------|---------|----------|----------------------|
| Variable                    | $\beta$ | $\pm SE$ | t value <sup>‡</sup> |
| (Intercept)                 | -0.243  | 0.012    |                      |
| AVIANCHOLERA                | -0.204  | 0.015    | -13.2                |
| TEMP                        | -0.001  | 0.001    | -1.0                 |
| AVIANCHOLERA * TEMP         | -0.004  | 0.003    | -1.3                 |
| PRECIP                      | <0.001  |          |                      |
| AVIANCHOLERA * PRECIP       | <0.001  |          |                      |

## **Nest success**

### ***Nest success estimates***

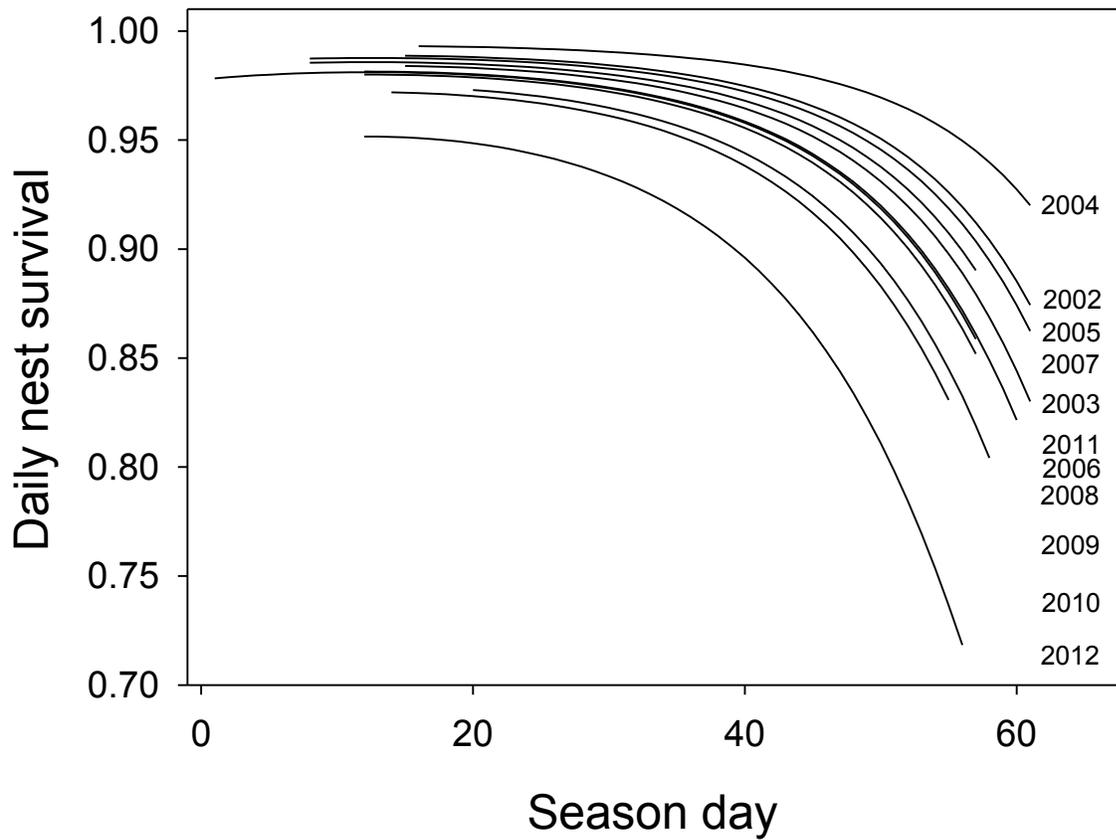
The top model for estimating daily nest survival probabilities contained YEAR and the quadratic effect SEASONDAY<sup>2</sup> (Table 5.5). These results indicate that daily nest survival followed a curvilinear seasonal pattern in which survival probability was high early in the season, declined slightly through mid-season, and declined precipitously thereafter (Figure 5.3). The year by time interaction indicates that there was annual variation in the timing and extent of the seasonal decline in survival.

This result was consistent with the prediction that eiders initiating incubation late in the breeding season achieve lower nest success. Annual estimates for early nesting females ranged from 0.17 to 0.78; estimates for median nesting females from 0.12 to 0.66; and estimates for late nesting females from 0.04 to 0.45 (Figure 5.4).

**Table 5.5.** Model rankings for nest survival analysis in PROGRAM MARK.

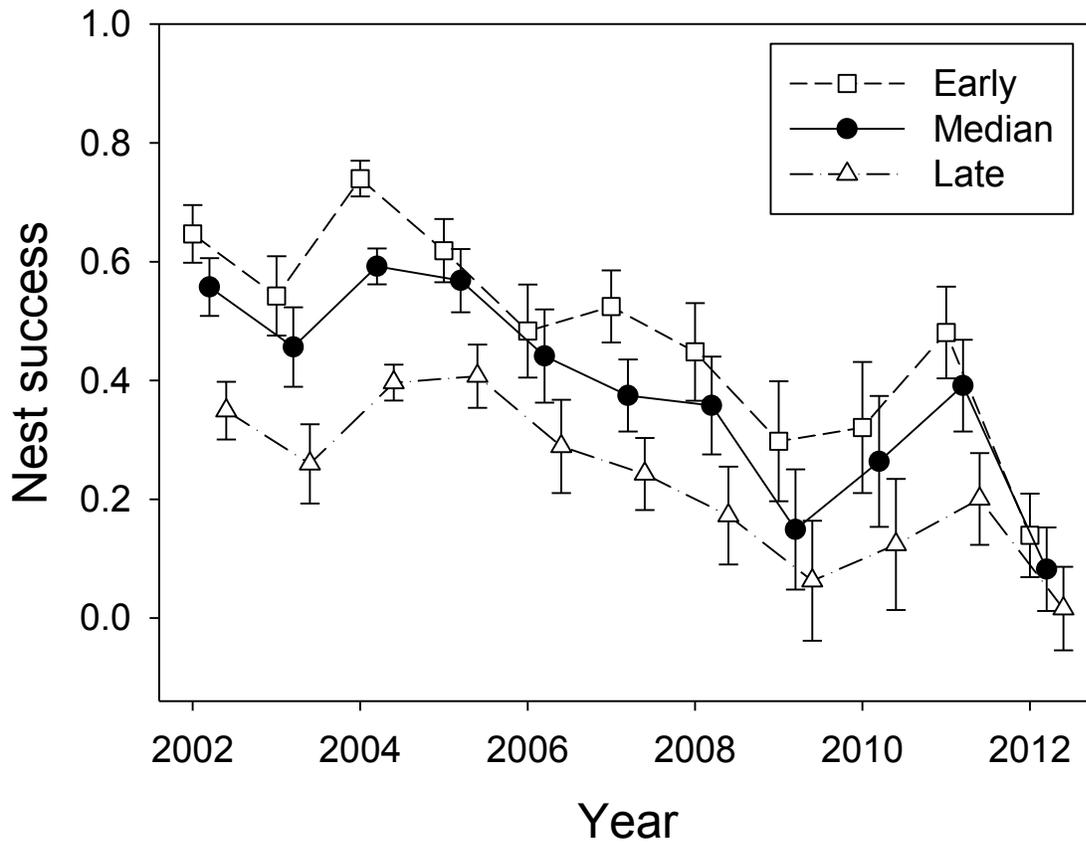
*Candidate set of daily nest survival models for common eiders at Mitivik Island, Nunavut, 2002-2012.*

| Model                         | Deviance | K  | $\Delta AIC_c$ | $w_i$ |
|-------------------------------|----------|----|----------------|-------|
| YEAR * SEASONDAY <sup>2</sup> | 4814.06  | 14 | 0.00           | 0.97  |
| YEAR + SEASONDAY <sup>2</sup> | 4820.84  | 14 | 6.78           | 0.03  |
| YEAR * SEASONDAY              | 4849.19  | 13 | 33.13          | 0.00  |
| Year + SEASONDAY              | 4856.31  | 13 | 40.25          | 0.00  |
| SEASONDAY <sup>2</sup>        | 4916.20  | 3  | 80.12          | 0.00  |
| SEASONDAY                     | 4937.34  | 2  | 99.25          | 0.00  |
| YEAR + NESTAGE <sup>2</sup>   | 4927.79  | 14 | 113.73         | 0.00  |
| YEAR + NESTAGE                | 4940.02  | 13 | 121.96         | 0.00  |
| YEAR                          | 4944.58  | 12 | 124.51         | 0.00  |



**Figure 5.3.** Daily nest survival rates.

*Predicted daily survival rates for common eiders in relation to time of season at Mitivik Island, Nunavut, 2002-2012. Estimates generated using the MCMC estimation procedure in PROGRAM MARK for the model YEAR\*SEASONDAY<sup>2</sup>. Nest observations began on 6 Jun (season day 1) and were monitored until 4 Aug (season day 60).*



**Figure 5.4.** Annual nest success estimates.

*Predicted nest success probabilities for common eiders in relation to year at Mitivik Island Nunavut, 2002-2012. Estimates generated assuming a 26 d nest cycle for females initiating incubation on the median date relative to other females, earlier (-7 d) than the median date and later (+7 d) than the median date.*

### ***Ecological predictors of variation in annual nest success***

The linear mixed-effects model used to evaluate ecological predictors of nest success indicated that several variables influenced the observed patterns of variation. The addition of AVIANCHOLERA to the BASE model (YEARRE + PHENOLOGY) did not considerably improve model support, suggesting avian cholera mortality had little influence on nest survival (evidence ratio  $E_{ij} = 0.4$ ). However, the addition of BEAR\*PHENOLOGY did improve model fit; as did additive combinations of BEAR and FOX with the BASE model (Table 5.6). For the abiotic variables, the addition of PRECIP and TEMP each improved model support, while the addition of POND resulted in no significant improvement ( $E_{ij} = 0.2$ ).

Regression coefficients indicated that phenology explained the most variation among the variables that I considered with a negative relationship between incubation onset timing and nest success (Table 5.7). An increasing frequency of bear incursions also was associated with reduced nest success, particularly for early and median nesting individuals. The influence of fox presence was weakly negative and not different in relation to phenology. Nest success was higher during wet years, while the frequency of extreme heat days (>20° C) was marginally negative (Table 5.7).

Raw data summaries were consistent with modeled results for bear incursions, heat days, and accumulated rainfall estimates (Figure 5.5).

**Table 5.6.** GLMM results relating nest success to ecological predictors.

*Confidence set of models used to evaluate variation in common eider nest success on Mitivik Island, Nunavut, 2002-2012. Table includes all models with AICc values lower than the BASE model (year modeled as a random effect with separate correlated intercepts and slopes in relation to early-, median-, and late-clutch initiation PHENOLOGY).*

| Explanatory variables                       | log(L) | K  | ΔAICc | $w_i$ | $E_{i,j}^\dagger$ |
|---|--------|----|-------|-------|-------------------|
| BASE + BEAR*PHENOLOGY + FOX + PRECIP + TEMP | 64.21  | 18 | 0.00  | 0.55  | 83.0              |
| BASE + PRECIP                               | 85.04  | 13 | 1.00  | 0.34  | 50.4              |
| BASE + BEAR*PHENOLOGY                       | 116.30 | 15 | 4.57  | 0.06  | 8.5               |
| BASE + FOX                                  | 74.62  | 13 | 6.54  | 0.02  | 3.2               |
| BASE + TEMP                                 | 95.46  | 13 | 7.14  | 0.02  | 2.3               |
| BASE + BEAR +FOX                            | 105.88 | 14 | 8.13  | 0.01  | 1.4               |
| BASE  | 53.79  | 12 | 8.84  | 0.01  | 1.0               |

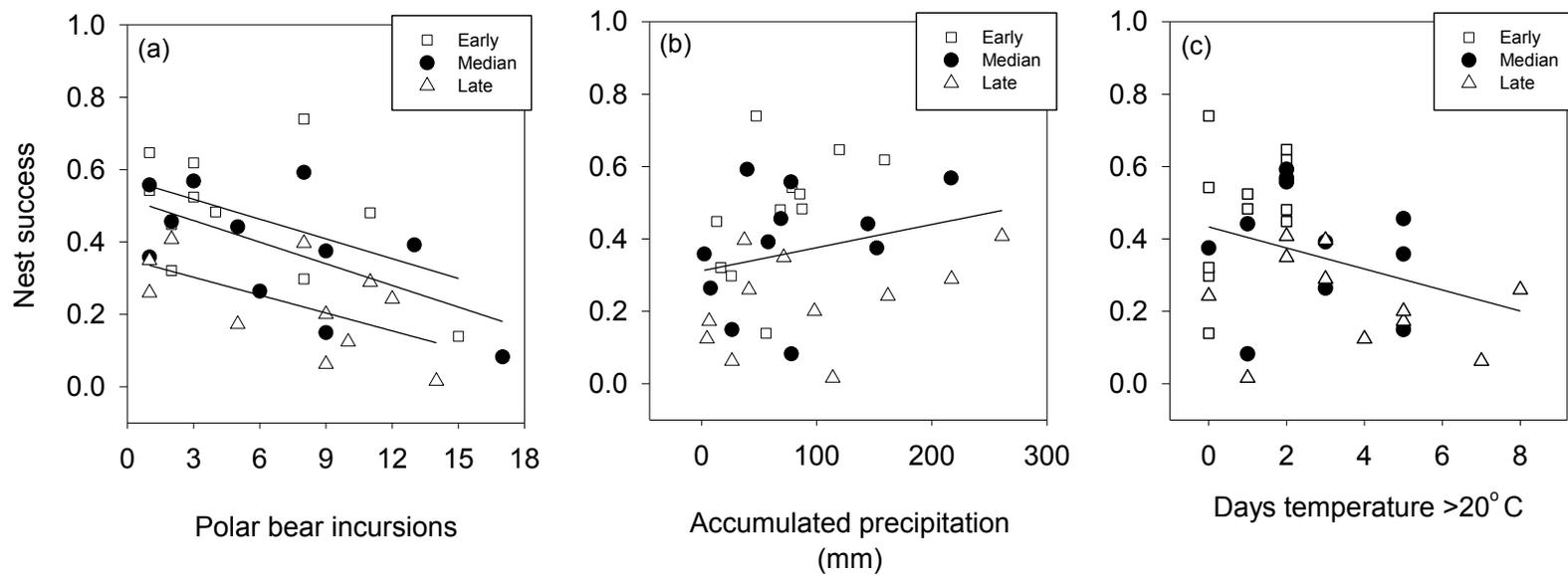
†  $E_{i,j}$  = evidence ratio ( $w_i / w_j$ ) for the model in question compared to the base model

**Table 5.7.** Regression coefficients for ecological predictors of variation in nest success.

*Regression coefficients for the top model (BASE + BEAR\*PHENOLOGY + FOX + PRECIP + TEMP) used to assess ecological predictors of variation in common eider nest success.*

| Variable               | $\beta$ | $\pm$ SE | t value <sup>‡</sup> |
|------------------------|---------|----------|----------------------|
| (Intercept)            | 0.467   | 0.047    |                      |
| Median nest initiation | -0.099  | 0.017    | -5.9                 |
| Late nest initiation   | -0.270  | 0.027    | -10.0                |
| Bear                   | -0.030  | 0.019    | -1.5                 |
| Fox                    | -0.009  | 0.023    | -0.4                 |
| Precip                 | 0.024   | 0.009    | 2.7                  |
| Temp                   | -0.003  | 0.008    | -0.4                 |
| Median*Bear            | -0.005  | 0.013    | -0.4                 |
| Late*Bear              | 0.021   | 0.016    | 1.3                  |

<sup>‡</sup> t value is the ratio of the regression coefficient to its standard error, where  $|t| > 1$  indicates the effect is statistically different from zero.



**Figure 5.5.** Nest success in relation to ecological predictors.

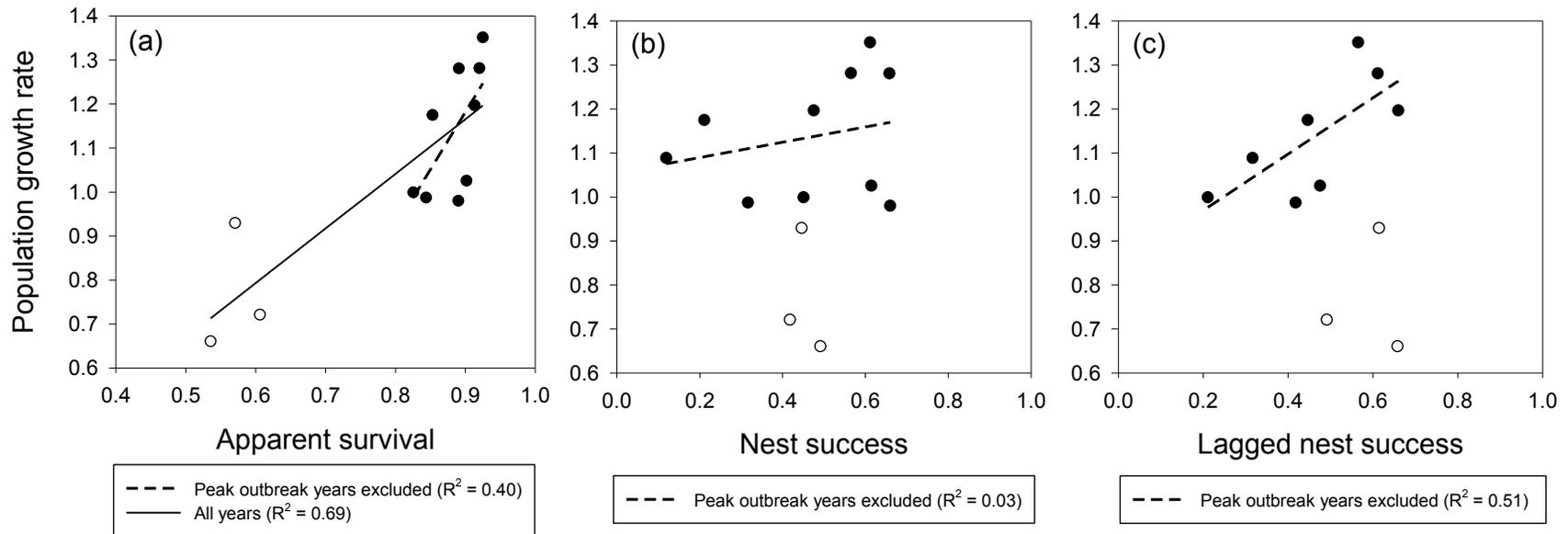
*Variation in nest success in relation to (a) the frequency of polar bear incursions (BEAR), (b) accumulated precipitation (PRECIP), and (c) frequency of days with maximum temperature >20° C (TEMP) during the incubation period of female common eiders initiating incubation early (-7 d), at the median date, and late (-7 d) in the breeding season at Mitivik Island, Nunavut, 2002-2012.*

### **Influence of survival and nest success on variation in population growth**

Trends in nesting pair abundance on Mitivik Island are presented in Chapter 2, Figure 2.2.

Density estimates ranged from a high of 453 ( $\pm 77$  SE) pairs \* ha<sup>-1</sup> in 2006, prior to the onset of extensive avian cholera mortality, to 201 ( $\pm 14$  SE) pairs \* ha<sup>-1</sup> in 2009, after three consecutive years of high mortality. Annual in population growth averaged  $\lambda_t = 1.17$  in the 6 years leading up to the onset of mass-mortality (2001-2006), from 0.66 to 0.93 during the height of the epidemic (2007-2009), and from 0.98 to 1.08 thereafter (2010-2013).

Linear regression plots suggest that the apparent annual survival rate of resident female eiders (SURV) was positively correlated with of variation in  $\lambda$ , as was lagged nest success when the peak epidemic years were excluded from analysis (Figure 5.6).



**Figure 5.6.** Population growth in relation to survival and nest success.

Variation in annual population growth rate ( $\lambda$ ) in relation to (a) resident female apparent survival probability ( $SURV$ ), (b) nest success ( $NS$ ), and (c) lagged nest success ( $NS_{LAG}$ ) for common eiders on Mitivik Island, Nunavut, 2001-2012. Open symbols indicate years following peak avian cholera mortality on the island.

## DISCUSSION

Despite its sustained expansion and evidence that avian cholera can be a significant source of mortality in wild birds, relatively little is known about the disease's role in regulating population dynamics (Botzler 1991, Samuel et al. 2007). This is due to the difficulty of conducting adequate assessments in free-ranging migratory birds and complex patterns of spatial and temporal variability in exposure. However, the magnitude of losses from individual outbreak events and the frequency of outbreaks in some populations have raised concerns about the biological costs of this disease (Friend 1999).

To date, most efforts to assess the impact of avian cholera have quantified mortality in relation to carcass counts (e.g., Crawford et al. 1992, Christensen et al. 1997, Botzler 2002). However, such estimates are subject to bias due to imperfect detectability and uncertainty concerning the size of population at risk (Huso 2011). Moreover, chronic low-levels of mortality may occur throughout the year and constitute a substantial portion of the annual losses in some populations (Wobeser 2002). These issues are best addressed in marked populations and in this study I found a high degree of correspondence between seasonal mortality estimates based on carcass counts (see Chapter 2) and annual mortality estimates based on mark-resight analyses. However, carcass counts consistently underestimated the degree of reduction below the pre-epidemic baseline rate, and in two years (2007, at the height of the epidemic; and 2011, when smoldering low-level mortality was observed on the colony) carcass counts greatly underestimated the reduction in annual survival probability. This raises the possibility that cholera-associated mortality occurred at other stages of the annual cycle; or

heightened mortality occurred due to other limiting factors that were not examined in this study. Such variation must be incorporated when conducting population viability assessments calibrated to determine the frequency of epidemics that a population is able to endure before suffering irreversible decline (e.g., Haydon et al. 2002, Gerber et al. 2005, Descamps et al. 2012).

Common eiders may be particularly vulnerable to contagious infections because they are both colonially breeding and highly gregarious during non-breeding season (Goudie et al. 2000, Christensen 2008). The reduction in annual survival probability that occurred among eiders on Mitivik Island in association with avian cholera was acute and prolonged. Resident females were the class which most intensely affected; however, elevated mortality rates were also documented for newly banded females ( $\phi^1$ ; a composite category comprised of unknown proportions of subadults, immigrants, and newly banded resident birds). The relationship between the species' ecological and life history characteristics and its' vulnerability to infection during the nesting season is highly relevant. Female eiders reach sexual maturity at two or more years of age, with a median age of first clutch production of three years (Baillie and Milne 1982, Hario and Rintala 2009). Pre-reproductive females are often observed prospecting for nests; however, subadult females, like males, leave the colony once breeding females initiated incubation. As such, breeding females have a much greater potential for exposure to *P. multocida* during the breeding season than other sex or age classes, with the implication being that the most intensive effects of disease are endured by the segment of the population upon

which the population growth rate is most sensitive to negative demographic perturbations (Goudie et al. 2000, Coulson 2010, Wilson et al. 2012).

Contrary to expectations, male common eiders also experienced significant mortality in association with the avian cholera epidemic. Percent mortality for males based on carcass counts was never more than 0.3% of the breeding population (Chapter 3), yet during the height of the epidemic survival probability for males was 0.08 to 0.29 points below the pre-epidemic baseline. Population models constructed for waterfowl with male-biased sex ratios typically focus on female vital rates because females are the limiting sex from a population growth perspective (Johnson et al. 1992). However, from a disease transmission perspective, the prevalence of infection in males has important implications for the cross-seasonal persistence and geographic spread (McCoy et al. 2005, Lachish et al. 2011). In my review of the literature I was unable to find other studies that examined whether the probability of emigration increased during a highly virulent infectious disease epidemic. My results suggest that neither females nor males altered their homing behavior following the arrival of avian cholera on Mitivik Island. However, males exhibited much lower site fidelity than females overall and unpaired males in particular are potential distributors of infection among colonies during the pre-laying period as they move among colonies in search of mating opportunity.

That variation in the population growth rate was strongly influenced by reductions in annual survival associated with disease status of the population is not surprising given that my study bracketed a major epidemic. The more relevant question is how other limiting factors interacted with disease mortality to influence survival and reproduction. In the case of harvest

mortality, I found evidence for an additive effect. This is consistent with management theory for the species in North America (Gilliland et al. 2009). Density-dependent hunting mortality is not suspected in northern common eiders as population size appears to be much reduced from historical levels (Merkel et al. 2004, Gilliland et al. 2009). Studies of other longer-lived migratory bird species that are also subject to intensive harvest, such as geese, show that most hunting mortality is additive (Gauthier et al. 2001); especially the mortality of adults as there is little variation in adult survival to allow for demographic compensation (Lebreton 2005). The prevailing view regarding the harvest of northern common eiders in Canada and Greenland is that current levels of hunting mortality are sustainable in the absence of other perturbations, but that the species is vulnerable should unanticipated mortality factors arise (Gilliland et al. 2009, Merkel et al. 2010).

A somewhat unexpected result of my investigation was that resident female survival also was negatively associated with polar bear incursions. While polar bears have been observed displaying predatory behavior toward waterfowl and seabirds on occasion (Stempniewicz 2006, Smith et al. 2010, Iles et al. 2013b), such behavior is generally regarded as infrequent and inefficient from the standpoint of bears' time-energy budgets (Lunn and Stirling 1985). It is likely that the negative association that I measured is a spurious correlation caused by co-occurring temporal trends (Grosbois et al. 2008). Bear incursions trended upward and survival rates downward over the course of study. However, female survival in the years following the most virulent phase of the epidemic remained below the pre-epidemic baseline, which I

speculate is related to the long epidemic tail, wherein smoldering infections persisted. Bear incursions reached their height during this time of smoldering infection.

Common eider reproductive success is highly variable through time, primarily due to the boom-bust nature of nest success and duckling survival (Coulson 1984; Wilson et al. 2012, Iles et al. 2013a). The main drivers of annual variability are thought to be environmental stochasticity related to unpredictable severe weather events (Jónsson et al. 2013, Iles et al. 2013a) and egg depredation by avian and mammalian predators, most notably foxes (Schamel 1977, Goudie et al. 2000), but increasingly polar bears in recent years (Rockwell and Gormezano 2009, Iles et al. 2013b, Iverson et al. 2014). The degree of annual variability that I measured in nesting success was similar to the range of variation estimated in previous studies (Coulson 1984, Mehlum 1991, Wilson et al. 2012, Iles et al. 2013a). Thus, a year, or even a run of years with very low nest success is not reason for conservation concern in and of itself. However, what is a potential concern is the apparent downward trend in nest success. Wilson et al. (2012) developed population model for Pacific common eiders (*S. m. v-nigrum*), which like the northern common eider breeds in Arctic and sub-Arctic regions. As such Pacific common eiders have a breeding phenology and seasonal movement schedule that is strongly influenced by sea ice dynamics and polar weather. In Wilson et al.'s study, a stable population was estimated to have a mean nest success rate  $\sim 0.55$ , with considerable variation around this mean, but no temporal trend. Females initiating incubation on or before the median lay date achieved roughly this rate nest success on Mitivik Island until 2006. However, in last several years nest success has been considerably lower.

Earlier thawing of the Arctic icepack is influencing the phenology and composition of the predator community, and thus, the frequency of catastrophic reproductive failure in eider colonies (Rockwell and Gormezano 2009, Iverson et al. 2014). The obvious concern is that under multiple stressors the capacity for demographic recovery is impaired in a disease population (Harvell et al. 2002, Bradley and Altizer 2007, Borer et al. 2007). Productivity rates are the driver of demographic recovery and if the underlying mechanism of intensified egg depredation risk caused by sea ice loss presented in Chapter 4 persist, then nest success should no longer be expected to fluctuate around the previous long-term average, but rather the rates documented during the past decade will constitute a new baseline.

## Chapter 6: General Discussion

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Plants and animals are subjected to many ecological perturbations and how population size varies in response to these perturbations depends in large part upon life history tactics and strategies (Beissinger and McCullough 2002, Morris and Doak 2002, Péron et al. 2012).

Infectious diseases are perturbations known to cause high mortality in wildlife (Lachish et al. 2007, Atkinson and Samuel 2010, Vredenburg et al. 2010); however determining demographic impacts and the influence of epidemic disease on dynamics of population abundance remains a significant challenge in free-ranging species. The traditional view that disease mortality is largely compensatory in wildlife populations, i.e., it merely replaces mortality which otherwise would have occurred from another source such as starvation, harvest, or predation (Holmes et al. 1982), has been replaced by a paradigm that recognizes population responses to disease are multitudinous and highly dependent upon ecological contexts (Choisy and Rohani 2006, Cross et al. 2009, Kistner and Belovsky 2014).

Past and newly emerging theory on disease ecology suggests that both the probability of occurrence and severity of an epidemic are strongly influenced by host density. This sets disease mortality apart from limiting factors that affect all population segments in similar ways regardless of population density, such as unusual weather events, seasonal cycles, and many anthropogenic impacts (Anderson and May 1979, Anderson and May 1991, Keeling and Rohani 2008). Infection rates are expected to slow following a period of sustained transmission as susceptible hosts become rare (de Castro and Bolker 2005, Lloyd-Smith et al. 2005); however,

there are circumstances when disease can drive populations to densities sufficiently low to predispose them to extinction by demographic stochasticity or Allee effects (de Castro and Bolker 2005). These circumstances include the enhanced oscillatory dynamics of novel pathogens in immunologically naïve hosts (Gerber et al. 2005, Vredenburg et al. 2010) and diseases that occur mainly in other hosts (Gog et al. 2002) or persist in the environment (Rohani et al. 2009) and thereby can remain at high incidence independent of population crashes in the focal host.

Population viability analysis (Beissinger and McCullough 2002, Morris and Doak 2002) and sustainable harvest models (Williams et al. 2002) are essential tools for determining conservation priorities for vulnerable and economically important species. The use of such models in age-structured wildlife populations requires information about variation in relevant vital rates in relation to ecological conditions, as well as patterns of covariation between vital rates. The inclusion of disease dynamics in population assessments is essential when pathogens are a significant source of variability in either mortality or fecundity rates; however few of the common packages for population analysis permit treating epidemic disease as anything more than a catastrophic event (Gerber and Hilborn 2001, Haydon et al. 2002, Descamps et al. 2012).

To date, epidemiological models and population viability models have largely developed independently. Epidemiological models are typically based on differential equations (Anderson and May 1979, Anderson and May 1991, Keeling and Rohani 2008), whereas population viability models for wildlife are more often structured in discrete time intervals reflective of the annual birth pulse and seasonal behavioral patterns of most vertebrates (Beissinger et al. 1998 Morris

and Doak 2002, Gerber and González-Suárez 2010). The difference in the characteristic time scale at which pathogen populations change compared to that on which host populations change means that simply combining the two classes of model is not possible (Gerber et al. 2005, Oli et al. 2006). However, progress is being made through the use of individual-based models (Haydon et al. 2002), modeling the dynamics of infectious diseases in discrete time based on the theory of matrix population models (Oli et al. 2006), and extension of epidemiological models to examine how host life-history traits and infection parameters interact to determine pathogen persistence in seasonal environments (Peel et al. 2014).

Gerber et al. (2005) identify four critical questions for including disease information in population assessments. (1) What is the likelihood of pathogen arrival into the population under consideration? (2) Given that infection has arrived, what is the likelihood that an epidemic will become established? (3) Once it has become established, what will be the impact of the pathogen on host vital rates? (4) How long will the pathogen persist in the population, once it has become established? The answer to each of these questions depends upon the biology of the pathogen and host, as well as interactions with other factors limiting population growth.

The results of my research constitute a step forward in determining disease impacts in a free-ranging population subject to a variety of limiting factors and for which basic epidemiological information has been lacking. I have sought and found answers to questions 2-4 and spatial heterogeneity in occurrence patterns, such as the absence of avian cholera from Greenlandic birds allows for *a priori* examinations of determinants of pathogen arrival.

In Chapter 2, I estimated the basic reproductive number ( $R_0$ ) during the initial wave of *P. multocida* invasion and temporal changes in real-time reproductive number ( $R_t$ ) in relation to concurrent changes in eider density as an avian cholera epidemic progressed on Mitivik Island. A quantitative estimate for  $R_0$  (or alternative measure of transmission rate) is required to express the transition rate from uninfected to infected states in population models that incorporate disease dynamics (McCallum et al. 2001, Gerber et al. 2005, Choquet et al. 2013). Consistent with expectations for a novel pathogen invasion, I found that case incidence increased in an exponential fashion during the initial wave of invasion, whereas  $R_t$ , predictably declined as the epidemic progressed. However, prolonged low-frequency transmission ( $R_t < 1$ ) was evident, which is a hallmark of disease persistence facilitated by environmental transmission. Neglecting transmission chains that are environmentally generated risks underestimating the explosiveness and duration of epidemics caused by pathogens with mixed transmission modes (Rohani et al. 2009)

In Chapter 3, I examined the spatial extent and ecological predictors of outbreak occurrence. I found evidence for an association between outbreak probability and the number of hosts sharing ponds on an eider colony, as well as a strong signal of heterogeneity in outbreak risk associated with population subdivision. Specifically, the avian cholera outbreaks that have been documented in northern common eiders appear to be associated with the segment of the population that winters in Atlantic Canada, whereas eiders associated with Greenlandic wintering areas and breeding areas remain disease-free. This finding suggests that in order to move beyond disease dynamics within local populations, as were investigated for

Mitivik Island, a metapopulation and/or phylogeographical context is required (Grenfell and Harwood 1997, Fulford et al. 2002, Park 2012).

In Chapter 4, I evaluated changing patterns of egg depredation in association with advancing sea ice break-up and documented severe declines in nest success in association with polar bear incursions. The important consideration from a population stability perspective is that the trend was directional and correlated with altered climatic conditions beyond the scope of normal temporal variability (Parmesan 2006, Harley 2011). In addition, as was also apparent with regard to avian cholera outbreaks in the Hudson Strait region, polar bear incursions were positively associated with eider abundance, suggesting that the largest colonies are at the greatest risk of negative demographic perturbation. These colonies are of the greatest economic value to eider down harvesters (Bédard et al. 2008) and therefore a potential focal point for management intervention, such as hazing nuisance bears if incursions occur regularly, or in the case of avian cholera, disrupting breeding at the first sign of disease mortality to limit *P. multocida* prevalence to preserve female survival.

Finally, in Chapter 5, I treated all these issues together and estimated variation in key vital rates in relation to disease, predation, weather conditions, and harvest of adult birds. Relatively few studies have so clearly demonstrated the demographic impact of infectious disease in a free-ranging population (e.g., Lachish et al. 2009, Samuels et al. 2011). I found avian cholera caused a dramatic decline in annual survival probability, which has abated only recently. Harvest mortality appears to be additive in this population, which is relevant because adaptive management of hunting regulations is among the few logistically feasible conservation

interventions. Productivity rates are the driver of demographic recovery; however, the increasing prevalence of polar bears on colonial nesting bird colonies during the breeding seasons has led to marked reductions in nest success. Mechanistically, altered predation risk could limit the population's capacity for demographic recovery; however forecasting future conditions with respect to either bear incursions and ice conditions or the periodicity of avian cholera outbreaks is highly uncertain.

Emerging infectious diseases increasingly are recognized by conservation biologists as a threat to populations and to biodiversity. The need for tools that can be integrated with other tools to consider such threat is now obvious. Climate change is already well advanced in the Arctic and disease impacts are having quantifiable impacts on Arctic wildlife (Kutz et al. 2005, Murray et al. 2006, Kutz et al. 2009). In comparison to temperate and tropical regions, host-pathogen systems in the Arctic are simpler and have fewer confounding variables. Dobson (2009) has argued that the patterns observed in Arctic will provide important insights into what is likely to occur elsewhere. The most useful studies for understanding disease impacts are those in which marked populations permit simultaneous estimation of vital rates and disease dynamics. While avian cholera was devastating to eider ducks on Mitivik Island, the presence of a long-term monitoring program there offered a rare opportunity for advancing ecological understanding of disease impacts, against the backdrop of ongoing and other novel threats to this species with varying time and spatial scales. Central to my thesis is how a combination of tools informs understanding of cumulative threats to populations and species and how in depth

use of singular tools can help us understand disease dynamics for novel host-pathogen associations.

## Appendices

### Appendix A. Supplementary Materials to Chapter 3

**Table A.1.** Summary of avian cholera mortality events in the Hudson Strait region of the Canadian Arctic (2004-2013).

| Outbreak ID <sup>†</sup> | Location (survey zone)           | Longitude, Latitude | Year of the mortality event | Species identified in the die-off                               | Year site investigation was undertaken | Laboratory assessment                           | Classification |
|--------------------------|----------------------------------|---------------------|-----------------------------|---|--|---|----------------|
| 1                        | Digges Sound (6)                 | -78.185, 62.384     | 2004                        | Common eider, glaucous gull, black guillemot, unspecified goose | 2004 and 2012                          | Positive culture from freshly collected tissues | Confirmed      |
| 2                        | Digges Sound (6)                 | -78.171, 62.431     | 2004                        | Common eider, black guillemot, unspecified goose                | 2004 and 2012                          | None  | Suspected      |
| 3                        | Digges Sound (6)                 | -78.204, 62.377     | 2004                        | Common eider, black guillemot, unspecified goose                | 2004 and 2012                          | None  | Suspected      |
| 4                        | Mansel Island (not surveyed)     | -79.266, 62.137     | 2004                        | Common eider, other species not specified                       | None undertaken                        | None  | Unconfirmed    |
| 5                        | Nottingham Island (not surveyed) | -77.537, 63.167     | 2004                        | Common eider, other species not specified                       | None undertaken                        | None  | Unconfirmed    |

| Outbreak ID <sup>†</sup> | Location (survey zone)       | Longitude, Latitude | Year of the mortality event | Species identified in the die-off                        | Year site investigation was undertaken | Laboratory assessment                           | Classification |
|--------------------------|------------------------------|---------------------|-----------------------------|--|--|---|----------------|
| 6                        | Western Ungava Bay (1)       | -69.548, 59.378     | 2006                        | Common eider, black guillemot, greater black-backed gull | 2011                                   | Positive culture from freshly collected tissues | Confirmed      |
| 7                        | Western Ungava Bay (2)       | -69.620, 60.048     | 2006                        | Common eider, other species not specified                | 2012                                   | None  | Suspected      |
| 8                        | Western Ungava Bay (2)       | -69.710, 60.015     | 2006                        | Common eider, other species not specified                | 2012                                   | None  | Suspected      |
| 9                        | Western Ungava Bay (2)       | -69.751, 60.036     | 2006                        | Common eider, other species not specified                | 2012                                   | None  | Suspected      |
| 10                       | Central Ungava Peninsula (4) | -71.440, 61.332     | 2006                        | Common eider, other species not specified                | 2011                                   | Positive PCR from bone marrow                   | Confirmed      |
| 11                       | Central Ungava Peninsula (4) | -71.477, 61.569     | 2006                        | Common eider, other species not specified                | 2011                                   | Positive culture from freshly collected tissues | Confirmed      |
| 12                       | Central Ungava Peninsula (4) | -71.702, 61.419     | 2006                        | Common eider, other species not specified                | 2011                                   | Positive culture from freshly collected tissues | Confirmed      |
| 13a                      | Central Ungava Peninsula (5) | -72.155, 61.847     | 2006;                       | Common eider, other species not specified                | 2011                                   | None  | Suspected      |

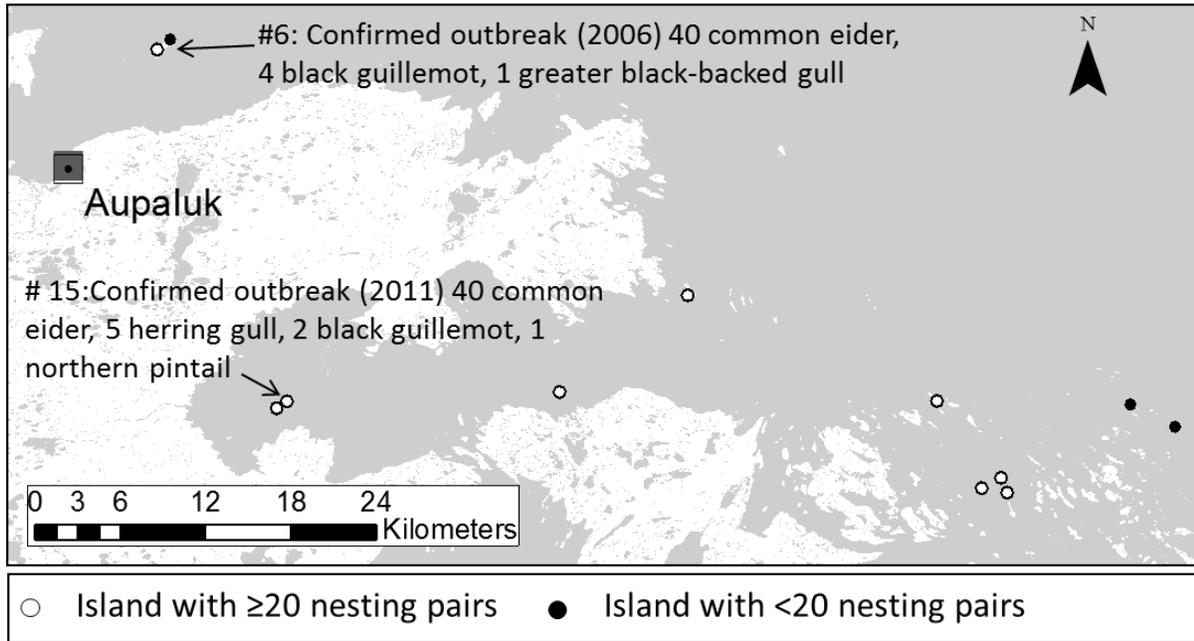
| Outbreak ID <sup>†</sup> | Location (survey zone)       | Longitude, Latitude | Year of the mortality event | Species identified in the die-off  | Year site investigation was undertaken | Laboratory assessment                           | Classification |
|--------------------------|------------------------------|---------------------|-----------------------------|--|--|---|----------------|
| 13b                      | Central Ungava Peninsula (5) | -72.155, 61.847     | 2011                        | Common eider, black guillemot, Canada goose, herring gull, glaucous gull | 2011                                   | None  | Suspected      |
| 14                       | Digges Sound (6)             | -78.109, 62.367     | 2008                        | Common eider   | None undertaken                        | None  | Unconfirmed    |
| 15                       | Western Ungava Bay (1)       | -69.466, 59.155     | 2011                        | Common eider, herring gull, black guillemot, northern pintail            | 2011                                   | Positive culture from freshly collected tissues | Confirmed      |
| 16                       | Central Ungava Peninsula (5) | -72.463, 62.132     | 2013                        | Common eider   | 2013                                   | None  | Suspected      |

<sup>†</sup> Locations excluded from the MAXENT species distribution model analysis because site investigation documenting avian mortality was not undertaken by Environment Canada biologists.

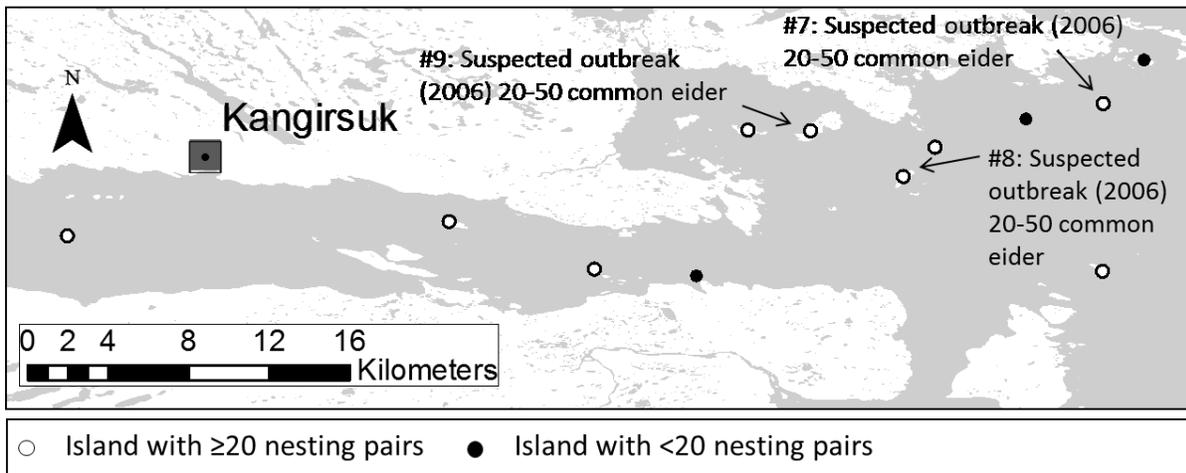
<sup>‡</sup> Estimated number of dead birds reported to wildlife authorities. Species identification codes as follows – BLGU: black guillemot; CAGO: Canada goose; COEI: common eider; GBBG: greater black-backed gull; GLGU: glaucous gull; HERG: herring gull; NOPI: northern pintail.

<sup>¥</sup> Report source: (A) Gaston, A.J. 2004. Report on surveys and research at Digges Island and vicinity, 27 July- 9 August 2004. Canadian Wildlife Service, Ottawa, ON, pp. 1-15; (B) Kwan, M. 2004. Avian cholera outbreak in Ivujivik, unpublished report, Nunavik Research Centre; (C) Simard, M. 2006. Avian cholera mortality in Nunavik, unpublished report, Nunavik Research Centre; (D) Smith, P.A. and Gaston. 2008. Report on seabird research at Digges Island and Vicinity, 25 July - 17 August 2008. National Wildlife Research Center, Environment Canada; (E) This study.

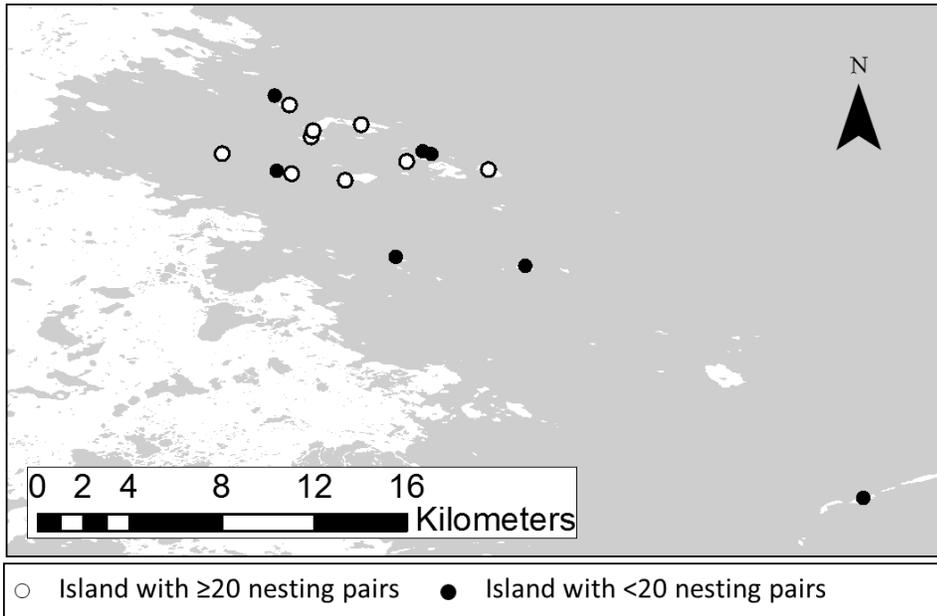
**Figure A.1** Ikkatuk Bay (survey zone 1).



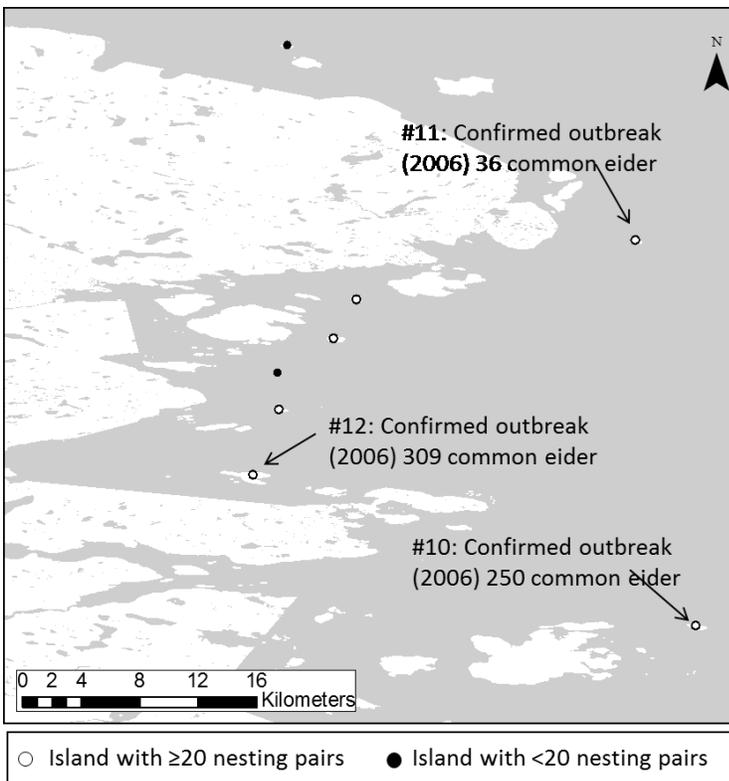
**Figure A.2.** Payne Bay (survey zone 2).



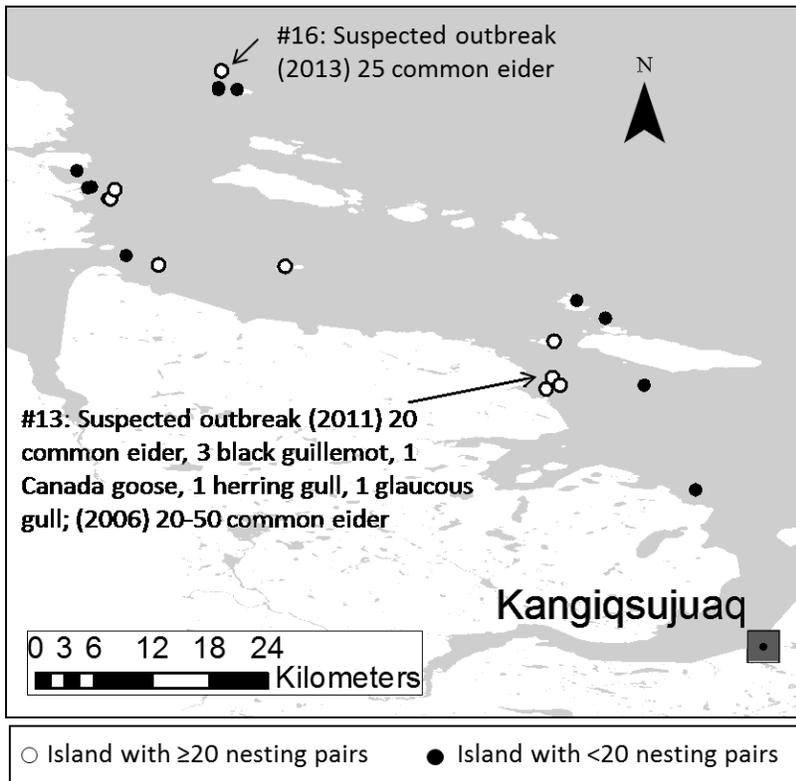
**Figure A.3.** Plover Islands (survey zone 3).



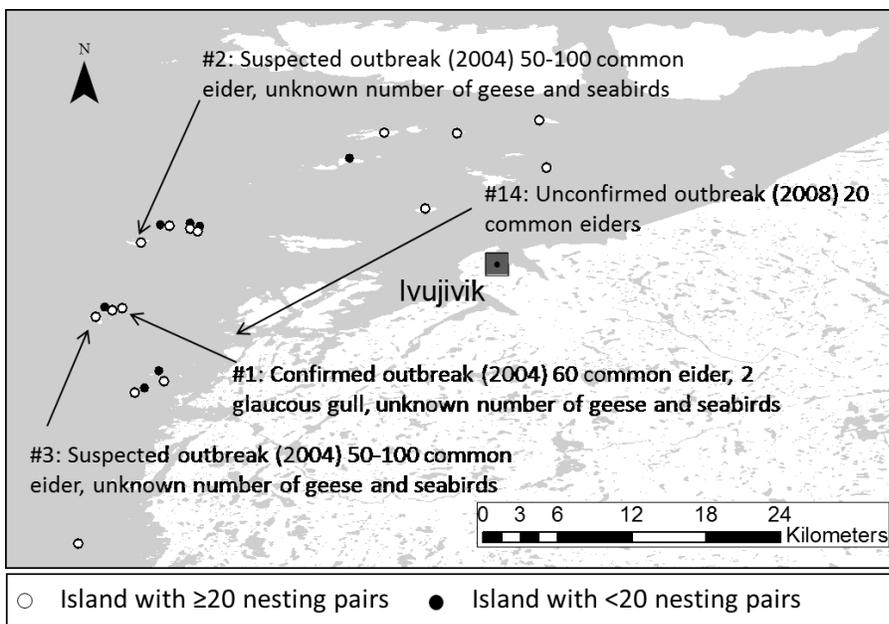
**Figure A.4.** Joy Bay (survey zone 4).



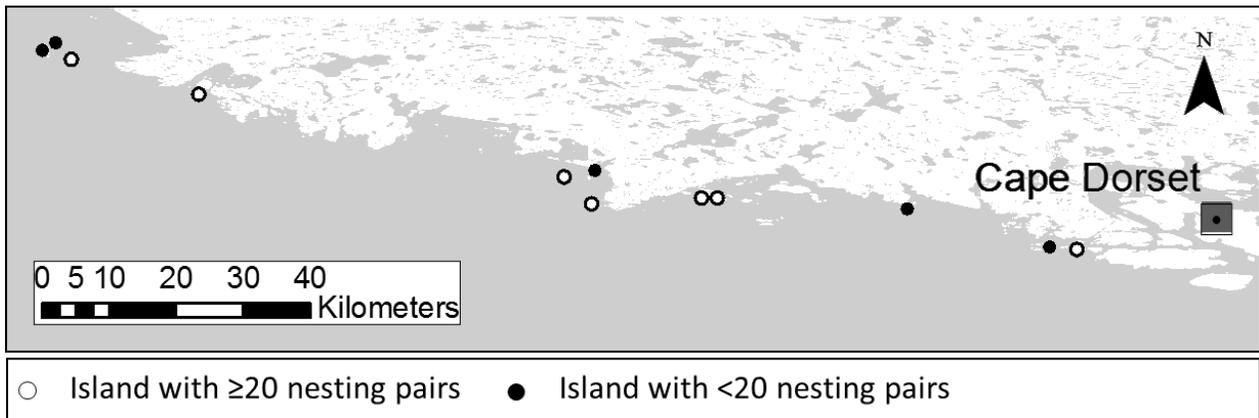
**Figure A.5.** King George Sound (survey zone 5).



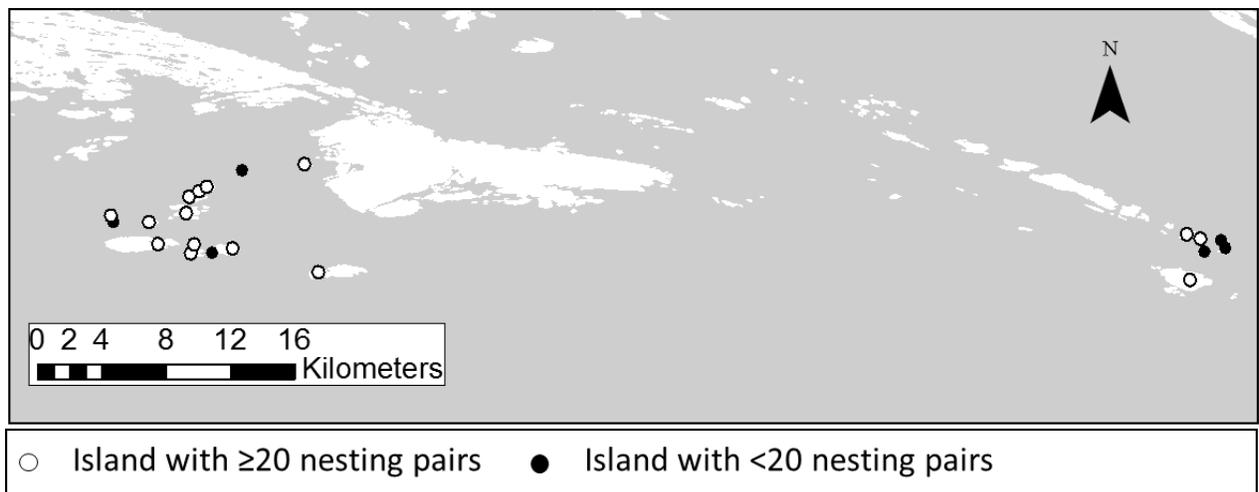
**Figure A.6.** Digges Sound (survey zone 6).



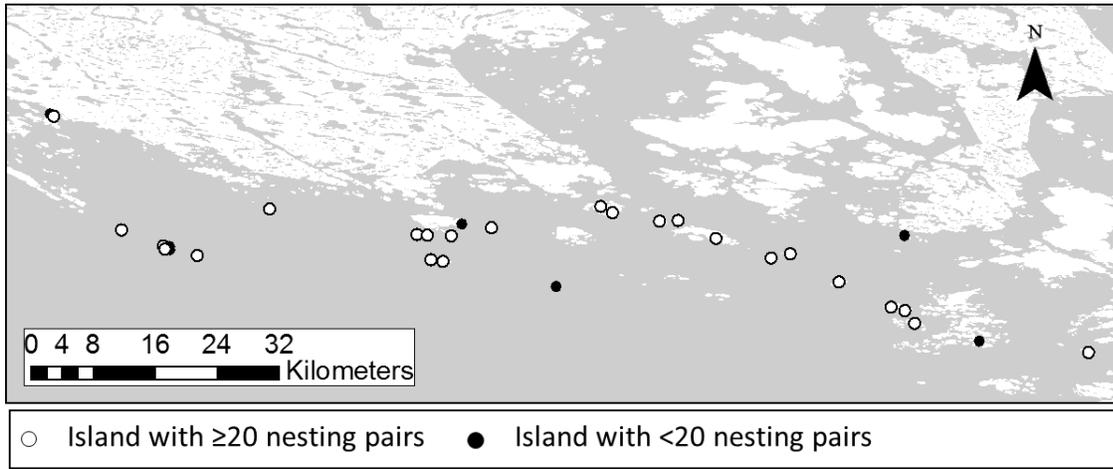
**Figure A.7.** Foxe Peninsula (survey zone 7).



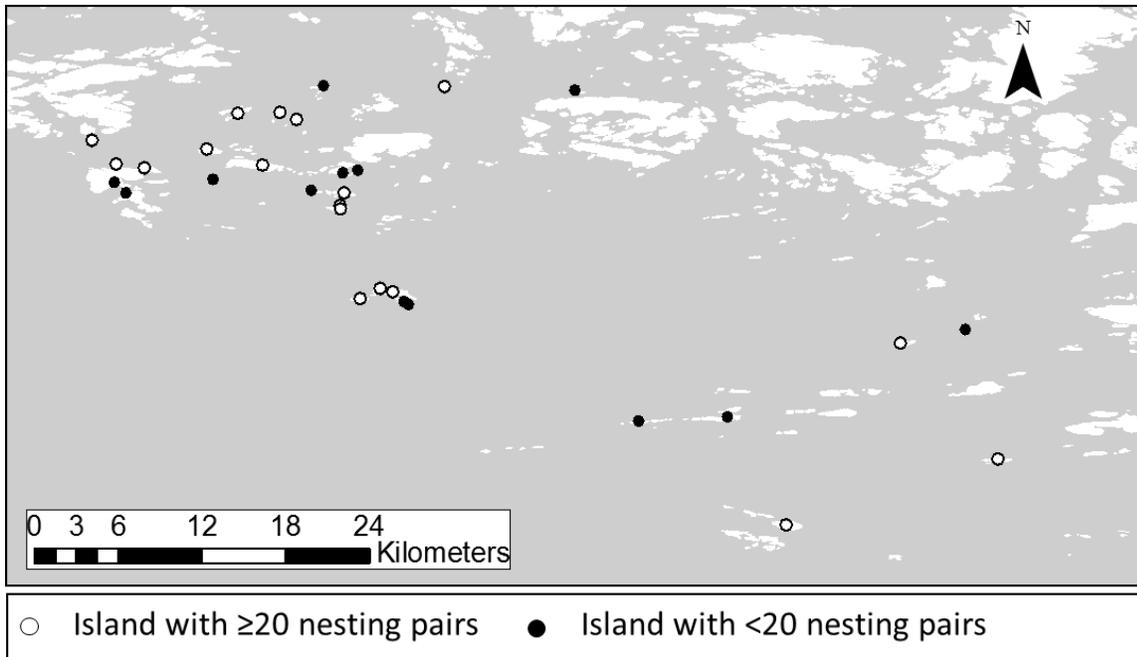
**Figure A.8.** West Foxe Islands (survey zone 8).



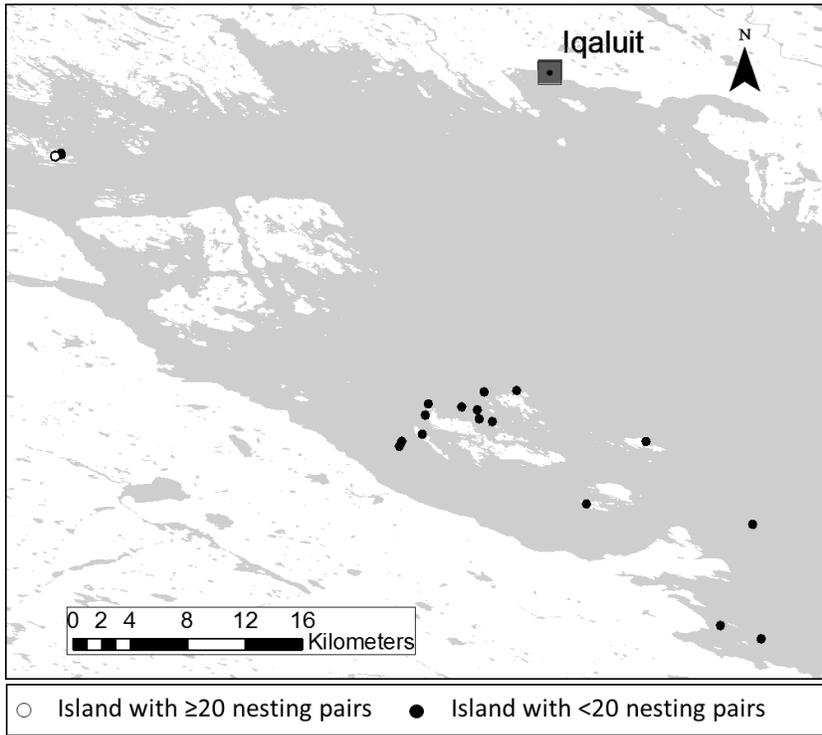
**Figure A.9.** Chorkbak Inlet (survey zone 9).



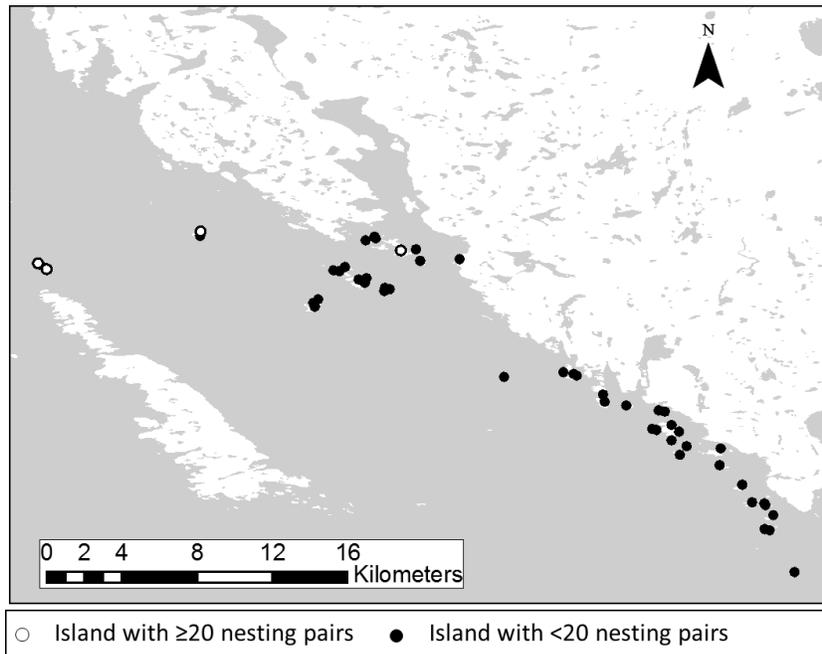
**Figure A.10.** Chamberlain Islands (survey zone 10).



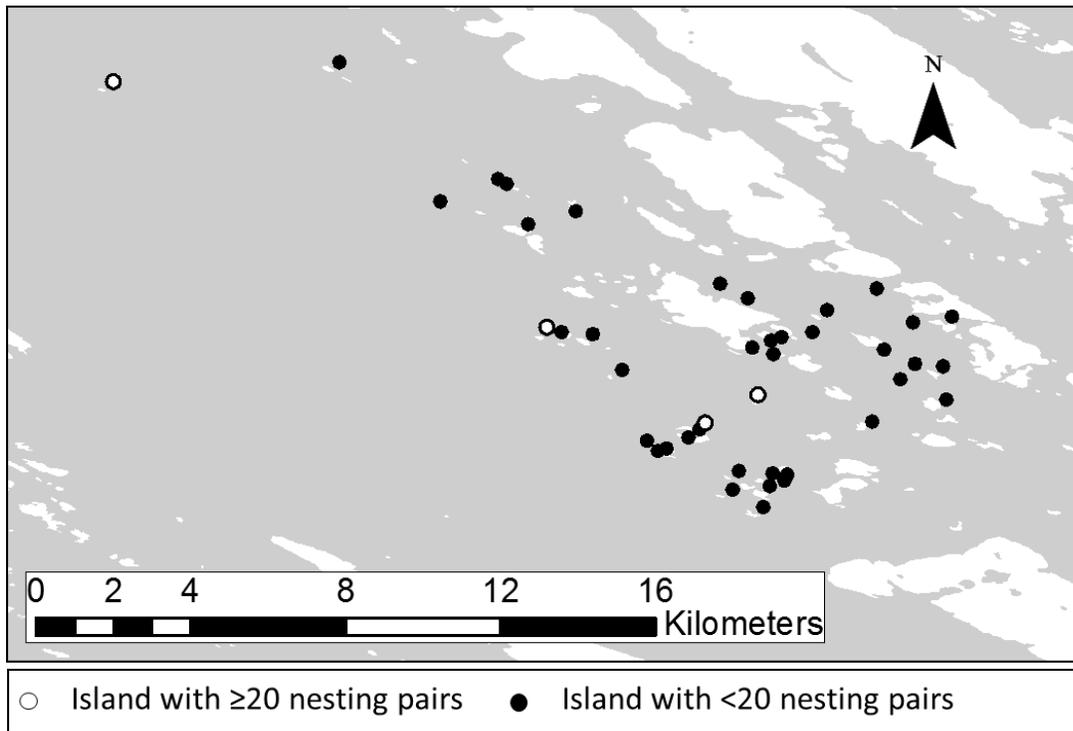
**Figure A.11.** Northern Frobisher Bay (survey zone 11).



**Figure A.12.** Central Frobisher Bay (survey zone 12).



**Figure A.13.** Southern Frobisher Bay (survey zone 13).



## Appendix B. Supplementary Materials to Chapter 4

### Polar bear observations at Mitivik Island and Cape Pembroke

The Mitivik Island common eider (*Somateria mollissima*) colony is located within East Bay Migratory Bird Sanctuary, Southampton Island, Nunavut. The colony is situated in a protected bay that is seasonally covered with landfast sea ice that typically melts several weeks later than the sea ice in surrounding, more open water areas. In addition to containing the largest known common eider colony in the Canadian Arctic, the island also hosts smaller numbers (<100) of other nesting species, including king eiders (*Somateria spectabilis*), cackling geese (*Branta hutchinsii*), brant geese (*Branta bernicla*), herring gulls (*Larus smithsonianus*) and glaucous gulls (*Larus hyperboreus*) (Environment Canada. 2012).

Research biologists stationed on Mitivik Island arrive each year in late May or early June before eggs are laid and depart after common eider ducklings have hatched in August. The station was established in 1996 and is administered by Environment Canada. Since 1997 field crews have been present throughout the nest incubation period, which we defined as 18 June to 1 August for our study purposes. Crew members have been present for an average of 44.0 d ( $\pm 3.3$  SD) per year within this 45 d interval (Appendix B, Table B.1).

Eider nests are monitored from observation blinds and biologists are trained to haze polar bears using scare cartridges and rubber bullets. This is done for the safety of the crew; however, bears frequently consume bird eggs before being detected and chased off the island. Ringed seals (*Pusa hispida*) are frequently observed in the vicinity of the island hauled out onto the sea ice and bearded seals (*Erignathus barbatus*) are occasionally observed.

The Cape Pembroke thick-billed murre (*Uria lomvia*) colony is located on the north-eastern tip of Coats Island, Nunavut. The colony is spread across several cliff faces on rocky uplands facing Evans Strait. The marine environment surrounding Cape Pembroke constitutes a more open water habitat than Mitivik Island and spring break-up typically occurs several weeks earlier than at Mitivik Island. The rocky uplands also support nesting Iceland gulls (*Larus glaucoides*) and glaucous gulls. In addition, lowland portions of Coats Island are home to a growing snow goose (*Chen caerulescens*) colony, as well as nesting cackling geese, brent geese, common eiders, king eiders, and herring gulls.

A long-term monitoring station, also administered by Environment Canada, was established at Cape Pembroke in 1984. We use observations during murre incubation, which we defined 10 July to 10 August. From 1988 to 2011, crew members were present for an average of 27.8 d ( $\pm 5.3$  SD) per year within this 31 d interval (Appendix B, Table B.2). Ringed seals and bearded seals are often observed hauled out landfast sea ice before break-up and there are several walrus (*Odobenus rosmarus*) haul-outs in the vicinity of Cape Pembroke which attract polar bears.

We quantified polar bear visits to the bird colonies using several metrics. All were based on direct sightings. The first was 'all encounters', in which we made no attempt to distinguish individuals or account for bears that may have moved out of sight only to return a short time later. We also estimated the number of 'distinct groups', in which we counted individuals that moved as a unit (e.g., an adult female and a cub, or a solitary adult) as a single entity. We counted new observations only when >3 d had passed since a previous sighting of a similarly

composed group, unless unique identifying features were noted (e.g., distinctive size, markings, or number of animals in the group). Finally, we calculated the number of 'days with bear' during the monitoring interval. Similar trends were observed for all three measurements.

We considered days with bear to be the least ambiguous measurement and most easily compared among sites; therefore, we used days with bear as the response variable in our statistical analyses.

### **Sea ice conditions**

The Canadian Ice Service (CIS) compiles information about ice conditions in Canada's Arctic waters using RADARSAT and MODIS satellite imagery. The data are integrated in weekly ice charts. CIS disseminates summarized data on regional and sub-regional scales through the IceGraph Tool 2.0 (<http://dynaweb.cis.ec.gc.ca/IceGraph20>) and in archived digital files (<http://dynaweb.cis.ec.gc.ca/Archive10>). Ice coverage values (IceCT) represent the percentage of the sea surface that is covered by ice within specified areas, multiplied by the concentration of the ice within that area. Data for our study area span from 1971 to the present.

For analyses relating to long-term changes in polar bear incursions at Mitivik Island and Cape Pembroke we used IceGraph to compile weekly IceCT estimates for Northern Hudson Bay Narrows, which encompasses the two research stations. We specified a period of interest from 1 October 1987 to 6 August 2012 and extrapolated daily IceCT estimates from CIS's weekly observations by assuming a linear rate of increase or decline from one sampling interval to the next. We then identified the Julian date that IceCT passed above the 30% and 60% coverage

thresholds each autumn and dropped below the 60% and 30% coverage thresholds each spring. We used this information to calculate the number of days that IceCT was >30% and >60% during the ice season that preceded bird nesting (SeasonLength30, SeasonLength60) and the date that good to poor (SpringMelt60) and poor to non-habitat (SpringMelt30) thresholds were crossed each spring. In addition, we compiled annual IceCT estimates at biweekly intervals throughout the nesting period to examine ice conditions at different stages of nest incubation, with dates selected to include 18Jun (EarlyIncubation), 2 Jul (Mid-Incubation), and 16 Jul (LateIncubation).

For analyses examining broad scale egg depredation patterns and habitat relationships we used archived digital charts to extract IceCT estimates within a 25 km radius of each surveyed island (Ice25). The ice charts provided us with geo-referenced boundaries for ice classifications on a landscape scale. We extracted ice coverage values on 25 Jun following procedures described below to obtain local ice coverage estimates that were specific to the year of survey.

### **Extent and magnitude of egg depredation by polar bears**

Colony surveys were conducted with the help of Inuit guides from the communities of Cape Dorset and Iqaluit, Nunavut, as well as Aupaluk, Kangirsuk, Kangiqsujuaq and Ivujivik, Nunavik (Québec). The surveys were conducted during the mid- to late-incubation period on 10-26 July 2010, 6-19 July 2011 and 8-21 July 2012.

Before conducting the field work, islands in selected survey zones were numbered on topographic maps. Only islands <500 ha in area were considered suitable eider nesting habitat, as larger islands often harbour foxes and do not support large colonies. Islands in most survey

zones had been previously been surveyed by Canadian Wildlife Service biologists on one or more occasions from the 1950s to 2000s. In Ungava Bay a randomized selection process was employed by the original researchers, whereas in the Foxe Peninsula, South Baffin, Frobisher Bay, and Digges Sound survey zones comprehensive sampling within defined study areas was conducted. We revisited previously surveyed locations to facilitate population monitoring goals and supplemented these locations with additional islands recommended by our Inuit guides to ensure that the largest eider colonies within each survey zone were investigated.

Islands were accessed by boat and searched on foot by 3-8 people walking 10-25 m apart in successive linear sweeps. At each island, we recorded latitude and longitude, as well as biophysical attributes (e.g., elevation, percent vegetation, and number of ponds) and the number and species of all nesting birds. Common eider nests were inspected and rated as active or empty (see text for details). For a subset of eider nests (25% on colonies with  $\geq 50$  breeding pairs and 50% on colonies with  $< 50$  breeding pairs) biologists recorded clutch size, nest status (incubating, in the process of hatching, hatched, or destroyed/depredated), and the characteristics of the feather down at the nest site (remaining in a cup or spread around the nest bowl). Our surveys were conducted late enough in the season that no clutches where the female was still laying (i.e., one to three eggs in a nest cup covered with nest material) were found.

We recorded all sightings of potential nest predators on focal islands. We also recorded the presence of animal sign, which we searched for systematically during our sweeps. Types of sign included faeces, fur and tracks. Our methodology did not allow us to say with certainty that

a predator species had consumed eggs and the species groups that we evaluated likely differed in their detectability; however, our methodology was useful for comparing the number and condition of eider nests on islands where definitive evidence of polar bear, fox or gull presence was encountered. For gulls, the most reliable indicators of presence were seeing birds and finding egg shells that had been cracked open by pecking. For polar bears, the principal signs were seeing animals, finding faeces or tracks, and encountering large numbers of nests wherein the feather down was strewn widely around the nest bowl and many egg remains were found crushed by large bites and blows. For foxes, the principle signs were seeing animals, finding feces or fur and encountering destroyed nests but few egg shell fragments because eggs are cached.

### **Landscape attributes**

We integrated eider colony locations with digital thematic maps to extract additional habitat information using ArcGIS, Version 10.1 (Environmental Systems Research Institute Inc., Redlands, CA, USA). We used CanVec topographic maps (1:50,000 and 1:250,000 scale) as our base layer (UTM zones 17, 18 and 19) and conducted data processing using the Spatial Analyst tool. For each island, we identified high and low tide contours and estimated AREA (km<sup>2</sup>) at high tide. For islets with little or no visible landmass we assigned a minimum area of 0.01 km<sup>2</sup>. We also estimated the minimum straight line distance from each island's centre point to the nearest MAINLAND shore (Baffin Island or Ungava Peninsula) at high tide, as well as the minimum distance to the nearest VILLAGE over water at high tide for if travelling by boat. We considered

all possible travel routes between the mainland and surveyed islands to determine the maximum inter-step CROSSING distance for a mammalian predator coming from the mainland at low tide. Finally, we used archived regional ice charts to determine ice coverage in the vicinity of each island during the year that it was surveyed. This was accomplished by applying a 25 km buffer to each island's perimeter and intersecting it with the appropriate CIS sub-regional ice chart. The resulting shapefiles contained bounded polygons for different ice classifications. To facilitate our analysis, we adjusted categorical ratings of '9+' to 9.5, 'land fast ice' to 10, and 'open water' to 0. We then calculated a single Ice25 value for each location as the mean ice concentration, weighted by area.

**Table B.1.** Dates of arrival and departure for biologists stationed at the Cape Pembroke thick-billed murre colony and Mitivik Island common eider colony.

| Year | Cape Pembroke (12 Jul to 11 Aug) |                |                         | Mitivik Island (18 Jun to 1 Aug) |                |                         |
|------|----------------------------------|----------------|-------------------------|----------------------------------|----------------|-------------------------|
|      | Arrival date                     | Departure date | Days biologists present | Arrival date                     | Departure date | Days biologists present |
| 1988 | <12 Jul                          | >11 Aug        | 31                      |                                  |                |                         |
| 1989 | 27-Jul                           | >11 Aug        | 16 <sup>†</sup>         |                                  |                |                         |
| 1990 | <12 Jul                          | >11 Aug        | 31                      |                                  |                |                         |
| 1991 | <12 Jul                          | >11 Aug        | 31                      |                                  |                |                         |
| 1992 | <12 Jul                          | >11 Aug        | 31                      |                                  |                |                         |
| 1993 | 16 Jul                           | >11 Aug        | 27                      |                                  |                |                         |
| 1994 | <12 Jul                          | >11 Aug        | 31                      |                                  |                |                         |
| 1995 | <12 Jul                          | >11 Aug        | 31                      |                                  |                |                         |
| 1996 | <12 Jul                          | >11 Aug        | 31                      |                                  |                |                         |
| 1997 | <12 Jul                          | >11 Aug        | 31                      | <18 Jun                          | 19 Jul         | 32 <sup>†</sup>         |
| 1998 | <12 Jul                          | >11 Aug        | 31                      | <18 Jun                          | > 1 Aug        | 45                      |
| 1999 | <12 Jul                          | >11 Aug        | 31                      | <18 Jun                          | > 1 Aug        | 45                      |
| 2000 | 27 Jul                           | >11 Aug        | 16 <sup>†</sup>         | <18 Jun                          | > 1 Aug        | 45                      |
| 2001 | 24 Jul                           | >11 Aug        | 19 <sup>†</sup>         | <18 Jun                          | > 1 Aug        | 45                      |
| 2002 | 27 Jul                           | >11 Aug        | 16 <sup>†</sup>         | <18 Jun                          | > 1 Aug        | 45                      |
| 2003 | <12 Jul                          | >11 Aug        | 31                      | <18 Jun                          | > 1 Aug        | 45                      |
| 2004 | <12 Jul                          | >11 Aug        | 31                      | <18 Jun                          | > 1 Aug        | 45                      |
| 2005 | 15 Jul                           | >11 Aug        | 28                      | <18 Jun                          | > 1 Aug        | 45                      |
| 2006 | 13 Jul                           | >11 Aug        | 30                      | <18 Jun                          | > 1 Aug        | 45                      |
| 2007 | 13 Jul                           | >11 Aug        | 30                      | <18 Jun                          | > 1 Aug        | 45                      |
| 2008 | 15 Jul                           | >11 Aug        | 28                      | <18 Jun                          | > 1 Aug        | 45                      |
| 2009 | <12 Jul                          | >11 Aug        | 31                      | <18 Jun                          | > 1 Aug        | 45                      |
| 2010 | <12 Jul                          | >11 Aug        | 31                      | <18 Jun                          | > 1 Aug        | 45                      |
| 2011 | <12 Jul                          | 9 Aug          | 29                      | <18 Jun                          | > 1 Aug        | 45                      |
| 2012 |                                  |                |                         | <18 Jun                          | 29 Jul         | 42                      |

<sup>†</sup> Years that information about the number of days on which bears were present was censored from GLMM analyses because biologists were not present for a sufficient proportion (0.9) of the study interval.

**Table B.2.** Number of islands with common eider colonies surveyed each year by geographic zone.

| Survey zone         | Total number of islands surveyed by year |           |           |            | Subset of islands surveyed on multiple occasions |             |             |                    |
|---------------------|--|-----------|-----------|------------|--|-------------|-------------|--------------------|
|                     | 2010                                     | 2011      | 2012      | Total      | 2010 & 2011                                      | 2010 & 2012 | 2011 & 2012 | 2010, 2011, & 2012 |
| A: Foxe Peninsula   | 28                                       | 19        | 24        | 71         | 1  | 4           | 3           | 14                 |
| B: South Baffin     | 51                                       | 14        | 7         | 72         | 9  | 4           | 0           | 2                  |
| C: Frobisher Bay    | 0  | 0         | 11        | 11         | 0  | 0           | 0           | 0                  |
| D: Digges Sound     | 0  | 0         | 17        | 17         | 0  | 0           | 0           | 0                  |
| E: Ungava Peninsula | 0  | 23        | 0         | 23         | 0  | 0           | 0           | 0                  |
| F: Ungava Bay       | 0  | 13        | 23        | 36         | 0  | 0           | 0           | 0                  |
| <b>Total</b>        | <b>79</b>                                | <b>69</b> | <b>82</b> | <b>230</b> | <b>10</b>  | <b>8</b>    | <b>3</b>    | <b>16</b>          |

**Table B.3.** Annual trends in sea ice coverage in Northern Hudson Bay Narrows from 1988 to 2012 under various ICECOVER indices.

Indices characterize the duration of winter ice coverage and the timing of spring break-up in days at 60% IceCT (good to poor) and 30% IceCT (poor to non-habitat) thresholds, as well as IceCT on specified dates (18 Jun, 2 Jul, 16 Jul) corresponding to stages of eider and murre nesting phenology.

| Statistic                               | SeasonLength60<br>(day) | SeasonLength30<br>(day) | SpringMelt60<br>(day) | SpringMelt30<br>(day) | EarlyIncubation<br>(IceCT) | MidIncubation<br>(IceCT) | LateIncubation<br>(IceCT) |
|---|-------------------------|-------------------------|-----------------------|-----------------------|----------------------------|--------------------------|---------------------------|
| Mean ( $\pm$ SE)                        | 216.0 ( $\pm$ 5.2)      | 241.9 ( $\pm$ 4.4)      | 22 Jun ( $\pm$ 3.0)   | 9 Jul ( $\pm$ 2.4)    | 0.61 ( $\pm$ 0.03)         | 0.45 ( $\pm$ 0.04)       | 0.49 ( $\pm$ 0.04)        |
| Minimum or<br>earliest date             | 172                     | 202                     | 24 May                | 11 Jun                | 0.17                       | 0.09                     | 0.00                      |
| Maximum or<br>latest date               | 265                     | 281                     | 23 Jul                | 1 Aug                 | 0.84                       | 0.84                     | 0.79                      |
| Slope of<br>annual trend<br>( $\pm$ SE) | -2.61 ( $\pm$ 0.48)     | -2.36 ( $\pm$ 0.38)     | -1.28 ( $\pm$ 0.32)   | -1.13 ( $\pm$ 0.25)   | -0.010 ( $\pm$ 0.004)      | -0.020 ( $\pm$<br>0.005) | -0.018 ( $\pm$<br>0.004)  |
| Regression R <sup>2</sup>               | 0.56                    | 0.63                    | 0.40                  | 0.47                  | 0.18                       | 0.45                     | 0.44                      |

**Table B.4.** Model selection results for linear regression analysis evaluating variation in the number days on which polar bears (TOTALBEARS) were observed on the Mitivik Island common eider colony in relation to ice conditions (ICECOVER) in Northern Hudson Bay Narrows. Calculations are described in the footnote below†.

| Explanatory variables | log(L)  | K | AICc   | $\Delta_i$ | Model likelihood | Model probability ( $w_i$ ) | Evidence ratio | R <sup>2</sup> |
|-----------------------|---------|---|--------|------------|------------------|-----------------------------|----------------|----------------|
| SeasonLength30        | -42.297 | 2 | 89.595 | 0.000      | 1.000            | 0.537                       | 54.0           | 0.538          |
| SpringMelt30          | -43.539 | 2 | 92.077 | 2.483      | 0.289            | 0.155                       | 15.6           | 0.469          |
| SeasonLength60        | -43.667 | 2 | 92.335 | 2.740      | 0.254            | 0.137                       | 13.7           | 0.415          |
| Mid-Incubation        | -44.280 | 2 | 93.561 | 3.966      | 0.138            | 0.074                       | 7.4            | 0.320          |
| SpringMelt60          | -44.728 | 2 | 94.456 | 4.861      | 0.088            | 0.047                       | 4.8            | 0.294          |
| EarlyIncubation       | -45.109 | 2 | 95.218 | 5.623      | 0.060            | 0.032                       | 3.2            | 0.273          |
| LateIncubation        | -46.577 | 2 | 98.153 | 8.558      | 0.014            | 0.007                       | 0.7            | 0.152          |
| Intercept-only        | -47.633 | 1 | 97.574 | 7.979      | 0.019            | 0.010                       | 1.0            |                |

**Table B.5.** Model selection results for linear regression analysis evaluating variation in the number days on which polar bears (TOTALBEARS) were observed on the Cape Pembroke thick-billed murre colony in relation to ice conditions (ICECOVER) in Northern Hudson Bay Narrows.

| Explanatory variables | log(L)  | K | AICc    | $\Delta_i$ | Model likelihood | Model probability ( $w_i$ ) | Evidence ratio        | R <sup>2</sup> |
|-----------------------|---------|---|---------|------------|------------------|-----------------------------|-----------------------|----------------|
| SeasonLength30        | -51.467 | 2 | 107.640 | 0.000      | 1.000            | 0.991                       | 1.5 * 10 <sup>5</sup> | 0.583          |
| SeasonLength60        | -56.903 | 2 | 118.511 | 10.871     | 0.004            | 0.004                       | 658.9                 | 0.424          |
| Mid-Incubation        | -57.145 | 2 | 118.996 | 11.356     | 0.003            | 0.003                       | 516.9                 | 0.439          |
| SpringMelt30          | -58.511 | 2 | 121.728 | 14.088     | 0.001            | 0.001                       | 131.9                 | 0.188          |
| LateIncubation        | -60.609 | 2 | 125.925 | 18.285     | 0.000            | 0.000                       | 16.1                  | 0.188          |
| EarlyIncubation       | -61.314 | 2 | 127.334 | 19.694     | 0.000            | 0.000                       | 8.0                   | 0.215          |
| SpringMelt60          | -62.240 | 2 | 129.186 | 21.546     | 0.000            | 0.000                       | 3.2                   | 0.228          |
| Intercept-only        | -64.635 | 1 | 131.492 | 23.852     | 0.000            | 0.000                       | 1.0                   |                |

**Table B.6.** Model selection results for Generalized Linear Mixed Models (GLMMs) evaluating variation in the daily probability of polar bear sightings (BEAR<sub>DAY</sub>) on Mitivik Island and Cape Pembroke. Table includes the global model, all models with AICc <4 and the null model.

| Explanatory variables  | log(L)   | K | AICc     | $\Delta_i$ | Model likelihood | Model probability ( $w_i$ ) | Evidence ratio          |
|--|----------|---|----------|------------|------------------|-----------------------------|-------------------------|
| YEAR + DAY + DAY <sup>2</sup> + ICECOVER' + YEARRE                   | -507.668 | 6 | 1027.397 | 0.000      | 1.000            | 0.723                       | 8.1 * 10 <sup>-17</sup> |
| SITE + YEAR + DAY + DAY <sup>2</sup> + ICECOVER' + SITE*DAY + YEARRE | -506.704 | 8 | 1029.514 | 2.118      | 0.347            | 0.251                       | 2.8 * 10 <sup>-17</sup> |
| YEARRE   | -552.930 | 2 | 1109.869 | 82.472     | 0.000            | 0.000                       | 1.00                    |

**Table B.7.** Model averaged regression coefficients ( $\beta$ ) for Generalized Linear Mixed Models (GLMMs) evaluating variation in the daily probability of polar bear sightings (BEARDAY) on Mitivik Island and Cape Pembroke.

| Variable         | $\beta$ | SE    | Lower 95% CI | Upper 95% CI |
|------------------|---------|-------|--------------|--------------|
| INTERCEPT        | -7.648  | 0.782 | -9.174       | -6.115       |
| YEAR             | 0.152   | 0.021 | 0.111        | 0.192        |
| DAY              | 0.221   | 0.039 | 0.145        | 0.297        |
| DAY <sup>2</sup> | -0.003  | 0.001 | -0.004       | -0.002       |
| ICECOVER'        | -0.034  | 0.009 | -0.051       | -0.016       |
| SITE             | 0.382   | 0.246 | 0.097        | 0.669        |
| SITE*DAY         | -0.011  | 0.006 | -0.023       | -0.001       |

**Table B.8.** Regression equations and  $R^2$  values for raw data plots illustrated in Figure 4.3, in which the daily probability of polar bear sightings (BEAR<sub>DAY</sub>) was evaluated in relation to the predictor variables listed below.

| Figure panel | Explanatory variable                    | Site           | Regression equation  | $R^2$ |
|--------------|---|----------------|--|-------|
| 3a           | YEAR                                    | Mitivik Island | $-44.6078 (\pm 7.9088) + 0.0223 (\pm 0.0039) * yr$                           | 0.696 |
|              |   | Cape Pembroke  | $-33.9534 (\pm 9.5428) + 0.0171 (\pm 0.0048) * yr$                           | 0.367 |
| 3b           | DAY + DAY <sup>2</sup>                  | Mitivik Island | $-0.0459 (\pm 0.0419) + 0.0180 (\pm 0.0042) * d - 0.0003 (\pm 0.0001) * d^2$ | 0.483 |
|              |   | Cape Pembroke  | $-1.0001 (\pm 0.2485) + 0.0642 (\pm 0.0132) * d - 0.0008 (\pm 0.0002) * d^2$ | 0.481 |
| 3c           | ICECOVER<br>(SeasonLength30)            | Mitivik Island | $1.5377 (\pm 0.1931) - 0.0059 (\pm 0.0008) * ic$                             | 0.812 |
|              |   | Cape Pembroke  | $1.8343 (\pm 0.3528) - 0.0068 (\pm 0.0014) * ic$                             | 0.556 |
| 3d           | ICECOVER'<br>(detrended SeasonLength30) | Mitivik Island | $0.1794 (\pm 0.0309) - 0.0032 (\pm 0.0023) * ic'$                            | 0.121 |
|              |   | Cape Pembroke  | $0.1556 (\pm 0.0387) - 0.0054 (\pm 0.0029) * ic'$                            | 0.134 |

**Table B.9.** Model selection results for GLMMs evaluating the presence of bear sign on common eider colonies in relation to ecological attributes. Table includes the global model, all models with AICc <4 and the null model.

| Explanatory variables  | log(L)  | K | AICc  | $\Delta_i$ | Model likelihood | Model probability ( $w_i$ ) | Evidence ratio  |
|--|---------|---|-------|------------|------------------|-----------------------------|-----------------|
| SQRTNESTS + MAINLAND + VILLAGE + ICE25 + ISLANDRE            | -117.74 | 6 | 1.000 | 0.000      | 0.334            | -117.74                     | $1.7 * 10^{11}$ |
| SQRTNESTS + VILLAGE + ICE25 + ISLANDRE                       | -119.54 | 5 | 0.474 | 1.491      | 0.158            | -119.54                     | $8.1 * 10^{10}$ |
| SQRTNESTS + MAINLAND + CROSSING + VILLAGE + ICE25 + ISLANDRE | -117.67 | 7 | 0.370 | 1.988      | 0.124            | -117.67                     | $6.3 * 10^{10}$ |
| SQRTNESTS + AREA + MAINLAND + VILLAGE + ICE25 + ISLANDRE     | -117.69 | 7 | 0.363 | 2.028      | 0.121            | -117.69                     | $6.2 * 10^{10}$ |
| SQRTNESTS + CROSSING + VILLAGE + ICE25 + ISLANDRE            | -119.36 | 6 | 0.198 | 3.240      | 0.066            | -119.36                     | $3.4 * 10^{10}$ |
| SQRTNESTS + AREA + VILLAGE + ICE25 + ISLANDRE                | -119.54 | 6 | 0.165 | 3.600      | 0.055            | -119.54                     | $2.8 * 10^{10}$ |
| ISLANDRE   | -147.76 | 2 | 0.000 | 51.716     | 0.000            | -147.76                     | 1.00            |

**Table B.10.** Model averaged regression coefficients ( $\beta$ ) for GLMMs evaluating the presence of bear sign on common eider colonies in relation to ecological attributes.

| Variable  | $\beta$ | SE    | Lower 95% CI | Upper 95% CI |
|-----------|---------|-------|--------------|--------------|
| INTERCEPT | -1.569  | 0.434 | -2.420       | -0.717       |
| SQRTNESTS | 0.091   | 0.023 | 0.047        | 0.136        |
| AREA      | 0.030   | 0.125 | -0.215       | 0.274        |
| MAINLAND  | 0.051   | 0.023 | 0.005        | 0.097        |
| CROSSING  | 0.005   | 0.040 | -0.074       | 0.083        |
| VILLAGE   | 0.012   | 0.004 | 0.004        | 0.020        |
| ICE25     | -0.053  | 0.012 | -0.071       | -0.029       |

**Table B.11.** Model selection results for GLMMs evaluating the presence of fox sign on common eider colonies in relation to ecological attributes. Table includes the global model, all models with AICc <4 and the null model.

| Explanatory variables                                | log(L)  | K | AICc    | $\Delta_i$ | Model likelihood | Model probability ( $w_i$ ) | Evidence ratio |
|--|---------|---|---------|------------|------------------|-----------------------------|----------------|
| CROSSING + VILLAGE + ISLANDRE                        | -68.094 | 4 | 144.366 | 0.000      | 1.000            | 0.136                       | 6.4            |
| CROSSING + ISLANDRE                                  | -69.552 | 3 | 145.210 | 0.844      | 0.656            | 0.089                       | 4.2            |
| AREA + CROSSING + VILLAGE + ISLANDRE                 | -67.560 | 5 | 145.388 | 1.022      | 0.600            | 0.082                       | 3.8            |
| MAINLAND + CROSSING + VILLAGE + ISLANDRE             | -67.736 | 5 | 145.740 | 1.374      | 0.503            | 0.068                       | 3.2            |
| AREA + CROSSING + ISLANDRE                           | -69.060 | 4 | 146.298 | 1.932      | 0.381            | 0.052                       | 2.4            |
| SQRTNESTS + CROSSING + VILLAGE + ISLANDRE            | -68.070 | 5 | 146.408 | 2.042      | 0.360            | 0.049                       | 2.3            |
| CROSSING + VILLAGE + ICE25 + ISLANDRE                | -68.093 | 5 | 146.454 | 2.088      | 0.352            | 0.048                       | 2.3            |
| AREA + MAINLAND + CROSSING + VILLAGE + ISLANDRE      | -67.192 | 6 | 146.761 | 2.395      | 0.302            | 0.041                       | 1.9            |
| MAINLAND + CROSSING + ISLANDRE                       | -69.444 | 4 | 147.066 | 2.700      | 0.259            | 0.035                       | 1.7            |
| SQRTNESTS + CROSSING + ISLANDRE                      | -69.507 | 4 | 147.192 | 2.826      | 0.243            | 0.033                       | 1.6            |
| CROSSING + ICE25 + ISLANDRE                          | -69.552 | 4 | 147.282 | 2.916      | 0.233            | 0.032                       | 1.5            |
| AREA + CROSSING + VILLAGE + ICE25 + ISLANDRE         | -67.543 | 6 | 147.463 | 3.097      | 0.213            | 0.029                       | 1.4            |
| SQRTNESTS + AREA + CROSSING + VILLAGE + ISLANDRE     | -67.547 | 6 | 147.471 | 3.105      | 0.212            | 0.029                       | 1.4            |
| SQRTNESTS + MAINLAND + CROSSING + VILLAGE + ISLANDRE | -67.727 | 6 | 147.831 | 3.465      | 0.177            | 0.024                       | 1.1            |
| MAINLAND + CROSSING + VILLAGE + ICE25 + ISLANDRE     | -67.731 | 6 | 147.839 | 3.473      | 0.176            | 0.024                       | 1.1            |

|  |         |   |         |       |       |       |     |
|--|---------|---|---------|-------|-------|-------|-----|
| ISLANDRE   | -72.016 | 2 | 148.085 | 3.719 | 0.156 | 0.021 | 1.0 |
| AREA + MAINLAND + CROSSING + ISLANDRE                                  | -68.933 | 5 | 148.134 | 3.768 | 0.152 | 0.021 | 1.0 |
| SQRTNESTS + AREA +CROSSING + ISLANDRE                                  | -69.031 | 5 | 148.330 | 3.964 | 0.138 | 0.019 | 0.9 |
| SQRTNESTS + AREA + MAINLAND + CROSSING + VILLAGE +<br>ICE25 + ISLANDRE | -67.172 | 8 | 150.996 | 6.630 | 0.036 | 0.005 | 0.2 |

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**Table B.12.** Model averaged regression coefficients ( $\beta$ ) for GLMMs evaluating the presence of fox sign on common eider colonies in relation to ecological attributes.

| Variable  | B      | SE    | Lower 95% CI | Upper 95% CI |
|-----------|--------|-------|--------------|--------------|
| INTERCEPT | -1.549 | 0.970 | -3.450       | 0.352        |
| SQRTNESTS | 0.001  | 0.004 | -0.008       | 0.009        |
| AREA      | 0.224  | 0.072 | 0.082        | 0.366        |
| MAINLAND  | -0.015 | 0.019 | -0.052       | 0.022        |
| CROSSING  | -1.486 | 0.733 | -2.922       | -0.050       |
| VILLAGE   | -0.013 | 0.011 | -0.033       | 0.008        |
| ICE25     | -0.003 | 0.095 | -0.190       | 0.184        |

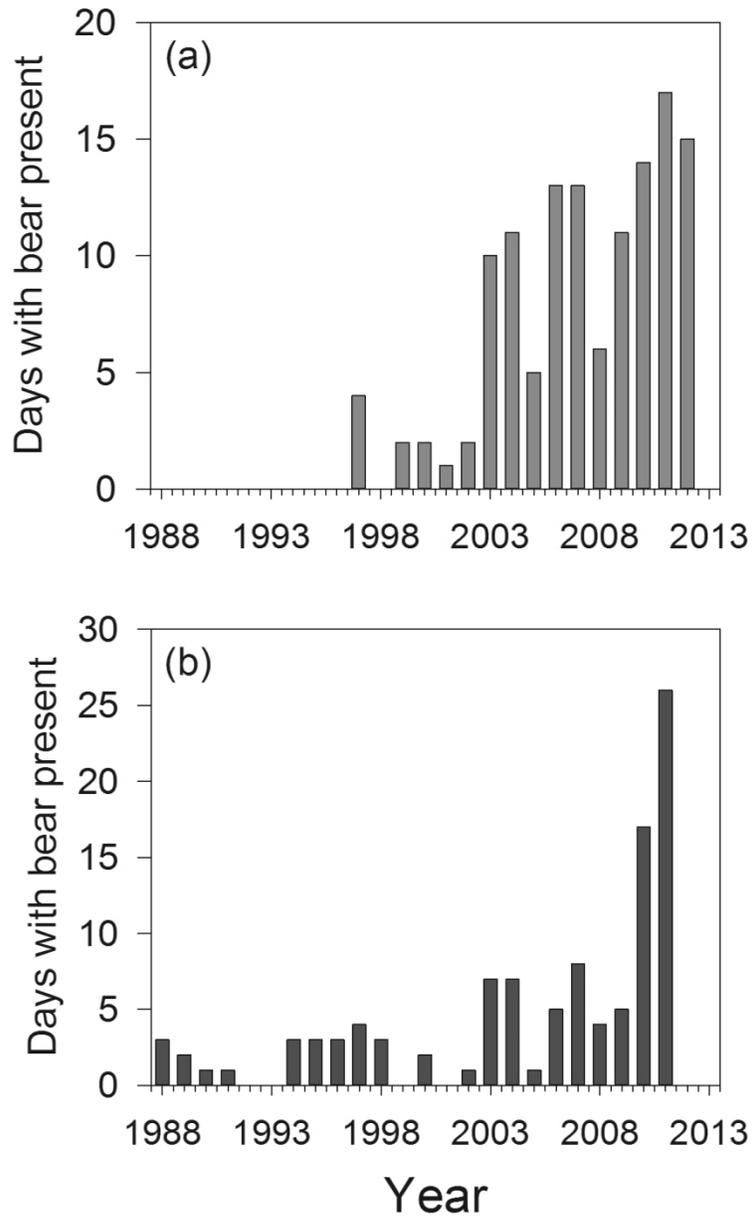
**Table B.13.** Model selection results for GLMMs evaluating nest success on common eider colonies in relation to the type of predator sign encountered, survey zone, nest abundance, and survey date, with island treated and as a random effect.

| Explanatory variables                             | log(L)  | K  | AICc    | $\Delta_i$ | Model likelihood | Model probability ( $w_i$ ) | Evidence ratio |
|---|---------|----|---------|------------|------------------|-----------------------------|----------------|
| PREDATORTYPE + SQRTNESTS + DATE + ISLANDRE        | -38.494 | 7  | 91.502  | 0.000      | 1.000            | 0.685                       | $2.6 * 10^9$   |
| PREDATORTYPE + DATE + ISLANDRE                    | -41.020 | 6  | 94.424  | 2.922      | 0.232            | 0.159                       | $6.1 * 10^8$   |
| PREDATORTYPE + ZONE + SQRTNESTS + DATE + ISLANDRE | -34.607 | 12 | 94.679  | 3.177      | 0.204            | 0.140                       | $5.4 * 10^8$   |
| PREDATORTYPE + SQRTNESTS + ISLANDRE               | -43.391 | 6  | 99.166  | 7.664      | 0.022            | 0.015                       | $5.7 * 10^7$   |
| PREDATORTYPE + ISLANDRE                           | -46.490 | 5  | 103.253 | 11.751     | 0.003            | 0.002                       | $7.4 * 10^6$   |
| PREDATORTYPE + ZONE + ISLANDRE                    | -44.537 | 10 | 110.097 | 18.595     | 0.000            | 0.000                       | $2.4 * 10^5$   |
| ZONE + DATE + ISLANDRE                            | -49.426 | 8  | 115.516 | 24.014     | 0.000            | 0.000                       | $1.6 * 10^4$   |
| ZONE + SQRTNESTS + DATE + ISLANDRE                | -49.179 | 9  | 117.191 | 25.690     | 0.000            | 0.000                       | 6983.0         |
| ZONE + ISLANDRE                                   | -59.007 | 7  | 132.528 | 41.026     | 0.000            | 0.000                       | 3.3            |
| ZONE + SQRTNESTS + ISLANDRE                       | -58.696 | 8  | 134.056 | 42.554     | 0.000            | 0.000                       | 1.5            |
| ISLANDRE  | -65.420 | 2  | 134.894 | 43.392     | 0.000            | 0.000                       | 1.0            |

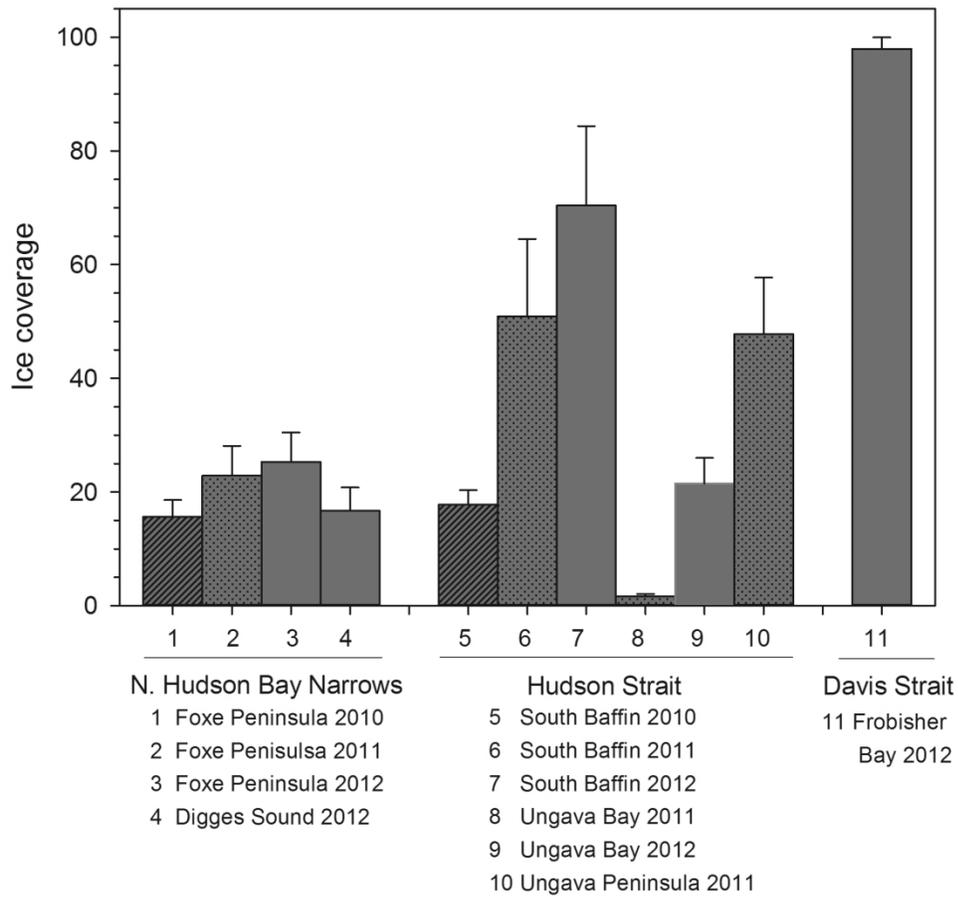
**Table B.14.** Model averaged regression coefficients ( $\beta$ ) for GLMMs evaluating nest success on common eider colonies in relation to the type of predator sign encountered, survey zone, nest abundance, and survey date.

| Variable                  | $\beta$ | SE    | Lower 95% CI | Upper 95% CI |
|---------------------------|---------|-------|--------------|--------------|
| INTERCEPT                 | 2.089   | 0.757 | 0.612        | 3.574        |
| PREDATORTYPE: GULL        | -0.578  | 0.620 | -1.787       | 0.637        |
| PREDATORTYPE: GULL & FOX  | -1.772  | 0.759 | -3.251       | -0.285       |
| PREDATORTYPE: GULL & BEAR | -2.612  | 0.692 | -3.962       | -1.254       |
| ZONE: FOXE PENINSULA      | -0.066  | 0.087 | -0.236       | 0.104        |
| ZONE: SOUTH BAFFIN        | -0.184  | 0.095 | -0.370       | 0.002        |
| ZONE: FROBISHER BAY       | 0.046   | 0.125 | -0.197       | 0.290        |
| ZONE: UNGAVA BAY          | -0.146  | 0.104 | -0.350       | 0.058        |
| ZONE: UNGAVA PENINSULA    | -0.107  | 0.110 | -0.323       | 0.109        |
| SQRTNESTS                 | 0.043   | 0.020 | 0.004        | 0.081        |
| DATE                      | -0.091  | 0.029 | -0.148       | -0.035       |

**Figure B.1.** Number of days on which one or more polar bears was sighted (TOTALBEAR) at the (a) Mitivik Island common eider colony from 18 Jun to 1 Aug, 1997-2012 and (b) Cape Pembroke thick-billed murre colony from 10 Jul to 10 Aug, 1988 – 2011.



**Figure B.2.** Mean proportion ( $\pm$ SE) of sea surface covered by ice on 25 June within a 25 km radius of common eider colonies surveyed in the lower Canadian Arctic. Colonies are categorized by region (Northern Hudson Bay Narrows, Hudson Strait, and Davis Strait), area (Foxe Peninsula, Digges Sound, Southern Baffin Island, Ungava Bay, Ungava Peninsula, and Frobisher Bay) and year of survey (2010, 2011, and 2012).



## Appendix C. Supplementary Materials to Chapter 5

**Table C.1.** Input data for the Cormack-Jolly-Seber (CJS) model of apparent survival and recapture probabilities for common eiders at Mitivik Island, Nunavut (2000-2013).  $R_i$  denotes the number of individuals released each year and includes newly captured individuals, as well as recaptured and resighted individuals.  $m_i$  denotes the number of ensuing contacts either via recapture or resighting.

| Group 1: Females |                           |   |      |      |      |      |      |      |      |      |      |      |      |      |
|------------------|---------------------------|---|------|------|------|------|------|------|------|------|------|------|------|------|
| Occasion         | Number released,<br>$R_i$ | Number recaptured or resighted, $m_{i,j}$ |      |      |      |      |      |      |      |      |      |      |      |      |
|                  |                           | $j=$<br>2001                              | 2002 | 2004 | 2004 | 2005 | 2006 | 2007 | 2008 | 2009 | 2010 | 2011 | 2012 | 2013 |
| $i = 2000$       | 176                       | 68  | 29   | 16   | 7    | 3    | 3    | 2    | 1    | 0    | 0    | 0    | 0    | 0    |
| 2001             | 328                       |   | 151  | 67   | 36   | 11   | 5    | 1    | 1    | 0    | 0    | 2    | 1    | 1    |
| 2002             | 337                       |   |      | 162  | 99   | 13   | 9    | 1    | 0    | 0    | 0    | 1    | 0    | 0    |
| 2003             | 601                       |   |      |      | 361  | 90   | 30   | 5    | 5    | 1    | 0    | 1    | 2    | 0    |
| 2004             | 783                       |   |      |      |      | 421  | 120  | 36   | 6    | 3    | 4    | 2    | 0    | 0    |
| 2005             | 894                       |   |      |      |      |      | 443  | 77   | 25   | 7    | 5    | 4    | 2    | 2    |
| 2006             | 879                       |   |      |      |      |      |      | 240  | 66   | 17   | 10   | 6    | 2    | 1    |
| 2007             | 780                       |   |      |      |      |      |      |      | 293  | 46   | 27   | 18   | 2    | 4    |
| 2008             | 740                       |   |      |      |      |      |      |      |      | 261  | 113  | 51   | 10   | 4    |
| 2009             | 574                       |   |      |      |      |      |      |      |      |      | 269  | 144  | 25   | 4    |
| 2010             | 643                       |   |      |      |      |      |      |      |      |      |      | 418  | 51   | 17   |
| 2011             | 901                       |   |      |      |      |      |      |      |      |      |      |      | 478  | 92   |
| 2012             | 636                       |   |      |      |      |      |      |      |      |      |      |      |      | 237  |

| Group 2: Males |                           |  |      |      |      |      |      |      |      |      |      |      |      |      |
|----------------|---------------------------|--|------|------|------|------|------|------|------|------|------|------|------|------|
| Occasion       | Number released,<br>$R_i$ | Number recaptured or resighted, $m_{ij}$ |      |      |      |      |      |      |      |      |      |      |      |      |
|                |                           | $j=$<br>2001                             | 2002 | 2004 | 2004 | 2005 | 2006 | 2007 | 2008 | 2009 | 2010 | 2011 | 2012 | 2013 |
| $i = 2000$     | 37                        | 3  | 1    | 0    | 0    | 0    | 1    | 1    | 0    | 0    | 0    | 0    | 0    | 0    |
| 2001           | 94                        |  | 7    | 3    | 2    | 1    | 2    | 2    | 0    | 0    | 0    | 2    | 1    | 0    |
| 2002           | 95                        |  |      | 6    | 6    | 4    | 5    | 0    | 2    | 0    | 0    | 0    | 0    | 0    |
| 2003           | 208                       |  |      |      | 17   | 5    | 6    | 1    | 1    | 0    | 1    | 0    | 0    | 0    |
| 2004           | 205                       |  |      |      |      | 27   | 6    | 2    | 2    | 1    | 0    | 2    | 0    | 0    |
| 2005           | 272                       |  |      |      |      |      | 32   | 4    | 7    | 0    | 2    | 2    | 0    | 0    |
| 2006           | 283                       |  |      |      |      |      |      | 17   | 3    | 1    | 2    | 5    | 1    | 1    |
| 2007           | 350                       |  |      |      |      |      |      |      | 22   | 0    | 5    | 1    | 3    | 1    |
| 2008           | 244                       |  |      |      |      |      |      |      |      | 4    | 8    | 3    | 2    | 2    |
| 2009           | 253                       |  |      |      |      |      |      |      |      |      | 15   | 7    | 8    | 0    |
| 2010           | 262                       |  |      |      |      |      |      |      |      |      |      | 31   | 5    | 3    |
| 2011           | 300                       |  |      |      |      |      |      |      |      |      |      |      | 22   | 9    |
| 2012           | 141                       |  |      |      |      |      |      |      |      |      |      |      |      | 13   |

**Table C.2.** Goodness-of-fit test results: U-CARE output for the Cormack-Jolly-Seber (CJS) model.

| Group  | Global test evaluating goodness-of-fit for the CJS model: $\phi(t) p_i(t)$ | Test for transience (>0)  | Test for non-independent resight rates (>0: "trap happy"; <0: "trap shy") |
|--------|--|---|---|
| Female | $\chi^2 = 228.09$<br>df = 90<br>p < 0.001                                  | N(0,1)= -2.94<br>p (1 sided test for transience) =0.98<br>p (2 sided) < 0.001 | N(0,1)= -6.89<br>p (2 sided) < 0.001                                      |
| Male   | $\chi^2 = 194.68$<br>df = 51<br>p < 0.001                                  | N(0,1)= 10.03<br>p (1 sided test for transience) <0.001                       | N(0,1)= -4.98<br>p (2 sided) < 0.001                                      |

**Table C.3.** Summary of band recovery input data for the Burnham live encounters and dead recoveries model used for common eiders at Mitivik Island, Nunavut (2003-2008).  $h_{i,j}$  denotes the number eiders harvested and reported by hunters and harvest denotes the proportion of released individuals harvested in a given year ( $h_i / R_i$ )

| Group 1: Females |   |      |      |      |      |      |       |              |
|------------------|---|------|------|------|------|------|-------|--------------|
| Occasion         | Number recovered and reported by hunters, $h_{i,j}$ |      |      |      |      |      |       | Harvest rate |
|                  | $j=2003$  | 2004 | 2005 | 2006 | 2007 | 2008 | Total |              |
| $i = 2003$       | 3   | 5    | 2    | 0    | 0    | 0    | 10    | 0.017        |
| 2004             |   | 6    | 2    | 5    | 0    | 0    | 13    | 0.017        |
| 2005             |   |      | 2    | 1    | 1    | 1    | 5     | 0.006        |
| 2006             |   |      |      | 2    | 1    | 1    | 4     | 0.004        |
| 2007             |   |      |      |      | 5    | 1    | 6     | 0.007        |
| 2008             |   |      |      |      |      | 1    | 1     | 0.001        |

| Group 2: Males |   |      |      |      |      |      |       |              |
|----------------|---|------|------|------|------|------|-------|--------------|
| Occasion       | Number recovered and reported by hunters, $h_{i,j}$ |      |      |      |      |      |       | Harvest rate |
|                | $j=2003$  | 2004 | 2005 | 2006 | 2007 | 2008 | Total |              |
| $i = 2003$     | 0   | 2    | 3    | 2    | 1    | 1    | 9     | 0.043        |
| 2004           |   | 5    | 1    | 1    | 1    | 0    | 8     | 0.039        |
| 2005           |   |      | 1    | 2    | 1    | 3    | 7     | 0.026        |
| 2006           |   |      |      | 2    | 4    | 3    | 9     | 0.032        |
| 2007           |   |      |      |      | 4    | 6    | 10    | 0.029        |
| 2008           |   |      |      |      |      | 2    | 2     | 0.008        |

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