Interactions between avian colonial social structure and disease dynamics

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Abstract

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All wildlife populations harbour parasites. However, seabirds are likely to play a particularly important role in the maintenance and dispersal of infectious agents as a result of their colonial breeding habits. Seabird colonies are also known to be highly spatially structured, but little is known about the effects of this spatial structuring on seabird parasite dynamics. In this thesis, I use a tick-borne virus, *Great Island virus* (GIV), found in a large common guillemot (*Uria aalge*) colony bordering the North Sea as a model system to explore this relationship. I use a multi-disciplinary approach, framed by a simple epidemiological model of the guillemot-tick-virus system.

In Chapter 2, I describe a novel epidemiological model and parameterise it using the existing literature. The model suggests the importance of spatial structure within the guillemot colony, but also identifies a key missing parameter, the rate of virus transmission between pre-breeding and breeding areas. In Chapter 3, I go on to test the potential role of independent tick movement in driving transmission between these two areas, by quantifying the mobility of host-seeking seabird ticks, *Ixodes uriae*. I show the potential for ticks to walk ranges described anecdotally in the literature, in just a few minutes, but stress the importance of further experiments in the field.

In Chapter 4, I test the potential role of guillemot-mediated tick movement between pre-breeding and breeding areas. I show that pre-breeding guillemots spend a limited proportion of time ashore during daylight hours, which increases significantly as the season progresses and varies between individuals. A similar pattern is observed when considering how often they enter breeding areas when ashore; generally infrequently but varying spatiotemporally and between individuals. In Chapter 5, I apply finite mixture modelling techniques to improve existing estimates of age- and strain-specific GIV seroprevalence and force of infection in the guillemot colony. I also provide the first estimates of these parameters for eight strains, and highlight the importance of understanding strain-specific differences in GIV dynamics in future studies.

Finally, I bring all four data chapters together in Chapter 6, by inputting my new parameter estimates (Chapters 3-5) into my existing model (Chapter 2). Taken together, my results suggest that GIV transmission within the guillemot colony may increase in the future as a result of declining breeding abundance and success, with more frequent or extreme disruption leading to a higher risk of infection within the colony. More generally, my results suggest that seabird colonies can be highly sensitive to changes in their spatial structure, and that endemic parasites have the potential to substantially impact, and hence to be an added threat to, their seabird hosts.

Declaration

I hereby declare that this thesis was composed by myself, and the work presented is the result of my own research, with the following acknowledgements. Data on nymphal mobility in Chapter 3 was collected by Dr Miles Nunn. Chapter 5 also includes published data collected by Dr Miles Nunn. All my supervisors helped develop ideas and provided comments on all of my chapters. The content of this thesis has not been presented in any previous application for a degree.

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Contents

General Introduction

Parasites, species that use host resources to feed and/or to reproduce while lowering host fitness to varying degrees (Gómez & Nichols 2013), have been documented in many wildlife populations (Nuttall *et al.* 1984; Heide-Jorgensen *et al.* 1992; Roelke-Parker *et al.* 1996; Randolph *et al.* 1999; Burthe *et al.* 2008; Lachish *et al.* 2011; Chambert *et al.* 2012; Hayman *et al.* 2012; VanderWaal *et al.* 2014). Seabirds are likely to play a particularly important role in the maintenance and dispersal of infectious agents (Dietrich, Gómez-Díaz & McCoy 2011). Many seabirds breed colonially (Perrins & Birkhead 1983) and show high breeding-site fidelity and natal philopatry (Furness & Monaghan 1987). This predictable occurrence of high densities of hosts is exploited by the many parasites that have been isolated in seabird colonies (e.g. avian paramyxoviruses: Coffee *et al.* 2010; avian pox virus: Young & VanderWerf 2008; avian influenza: Lebarbenchon *et al.*, 2015; orbiviruses and bunyaviruses: Nuttall *et al.* 1981; flaviviruses: Arnal *et al.* 2014; avian cholera: Österblom *et al.* 2004; Lyme disease spirochetes: Chambert *et al.* 2012) and previous studies suggest the importance of seabird colonies as 'persistent foci' for parasite circulation (Nuttall 1984).

Seabirds are also epidemiologically interesting because, during the breeding season, they are highly spatially structured due to seabirds' territorial behaviour over nest sites (Furness & Monaghan 1987) and structured cliff habitat, as demonstrated by the spatial structuring of parasite populations within and between colonies (Boulinier & Danchin 1996; McCoy *et al.* 1999, 2003; McCoy, Boulinier & Tirard 2005; Gómez-Díaz *et al.* 2011). Evidence for at-sea spatial partitioning in foraging habitats during this part of the life cycle also exists (Dean *et al.* 2013; Wakefield *et al.* 2013). However, outside the breeding season, seabirds tend to be highly mobile, and to travel over wide geographic distances (Guilford *et al.* 2009, 2011), potentially facilitating the global dispersal and circulation of parasites (Clifford 1979; Nuttall 1984; Danchin 1992).

Epidemiological models are powerful tools for exploring the processes regulating the transmission of wildlife parasites, such as climate (e.g. Rogers & Randolph 2006), habitat (e.g. Smith, Dushoff & McKenzie 2004) and social network (e.g. Craft *et al.* 2011). They have also been used to demonstrate the link between host spatial structuring and wildlife parasite dynamics, for phocine distemper virus (Swinton *et al.* 1998; Klepac *et al.* 2009), rabies virus (Smith *et al.* 2002) and louping ill virus (Watts *et al.* 2009) among other wildlife parasites. As a result, evidence exists for some host spatial structures being more amenable to disease spread than others (Hess 1996; Fulford, Roberts & Heesterbeek 2002; Smith, McDonald & Wilkinson 2012). However, few studies have used epidemiological models to investigate parasite dynamics in seabird colonies and I know of only one that has attempted to incorporate host spatial structuring (Clancy, O'Callaghan & Kelly 2006; O'Regan *et al.* 2008, 2013). This is surprising, as spatial variability has been identified as a key predictor of exposure to zoonotic infection (Randolph *et al.* 2002).

Furthermore, seabird populations are globally threatened, with nearly half of seabird species experiencing population declines, mainly as a result of commercial fishing and pollution at sea, and alien invasive predators, habitat degradation and human disturbance on land (Croxall *et al.* 2012). The impact of climate change on seabird populations has been identified as a main knowledge gap (Croxall *et al.* 2012), but is likely to be significant (Grémillet & Boulinier 2009). These threats could have consequences for spatial organisation of seabird populations and associated parasite dynamics, with potentially important consequences for planning conservation actions. Although understudied, disease may play an important role in species extinction and endangerment more generally (Dobson & Foufopoulos 2001; Smith, Sax & Lafferty 2006), with recent large-scale declines in several wildlife species including seals in the North Sea (Heide-Jorgensen *et al.* 1992), Gyps vultures in India (Prakesh 1991), Serengeti lions in East Africa (Roelke-Parker *et al.* 1996) and many frog species across Australia, Central America and the United States (Blaustein & Wake 1990).

Guillemot-tick-virus: model system

The common guillemot (*Uria aalge*) is parasitised by the seabird tick (*Ixodes uriae)* and the parasites it transmits (Olsen *et al.* 1993; Nunn *et al.* 2006a; Duneau *et al.* 2008; Major *et al.* 2009; Duron *et al.* 2014). Among these parasites is the orbivirus *Great Island virus* (GIV; Attoui *et al.* 2012). Throughout this thesis, I refer to the common guillemot as the host and the seabird tick as the vector of GIV. The common guillemot-tick-virus system is a good model for exploring the effects of host spatial structure on seabird parasite dynamics because there is a readily observable, age-related spatial structure within common guillemot colonies. It is also an interesting system because GIV is an endemic infection (i.e. it has been maintained in a particular population or geographic region for a long period of time). Although more abundant, the role of endemic parasites in shaping wildlife populations remains poorly understood. Although not always pathogenic (Chambert *et al.* 2012; Hayman *et al.* 2012), endemic parasites can have significant, sublethal effects on their hosts (Joly & Messier 2005; Burthe *et al.* 2008), and can increase in host range or pathogenicity, leading to the emergence of potentially zoonotic infections (Daszak, Cunningham & Hyatt 2000; Dobson & Foufopoulos 2001; Rachowicz *et al.* 2005; Jones *et al.* 2008).

Common guillemot (*Uria aalge*)

The common guillemot (*Uria aalge;* hereafter guillemot) is a member of the auk family (Alcidae) (Gaston & Jones 1998). It is a long-lived seabird, with a mean lifespan of 20 years (Reynolds *et al.* 2011). The guillemot spends the majority of its time in marine offshore and inshore waters (mainly boreal and low Arctic), outside the breeding season. Guillemots from British and Irish colonies are recovered from across the North Cape in Norway to Gibraltar and from South West Iceland to the Baltic. A great deal of mixing occurs between populations during this time (Wernham *et al.* 2002), with pre-breeding guillemots moving across particularly large distances and between colonies (Halley & Harris 1993).

However, for several months in the year, common guillemots come to land to breed in large, dense colonies, numbering tens of thousands of birds and reaching densities of 34 birds/m^2 (Tuck 1961). These colonies can be subdivided into smaller subcolonies (Birkhead 1976; Tuck 1961), found on sections of rocky cliff, stacks or islets, up to 150 metres above sea level (Kay 1947) and with ledges or crevices able to accommodate eggs. Subcolonies demonstrate an age-related spatial structure with adults on cliff ledges, while prospecting immature birds congregate around the edges of the breeding colony (Halley 1992; Hudson 1979; Fig. 1). In the UK, he common guillemot has been identified as a species of conservation priority under the EC Birds Directive, with internationally significant numbers breeding within a number of protected breeding colonies (Council of the European Union 2010; JNCC 2014).

On the Isle of May, Scotland the median age of first breeding is 7 years (Harris, Halley & Swann 1994; Lewis *et al.* 2006; Harris pers. comm.). Most breeding guillemots return to the same breeding site (e.g. 85.7% on Isle of May; Harris, Wanless & Barton 1996) with the same partner (Storer 1952; Tuck 1961) each year. The breeding site is defended by both members of the pair (Cramp & Simmons 1985). Incubating birds can be aggressive towards predators, lunging at approaching gulls, and resulting in a positive correlation between breeding success and nesting density (Birkhead 1977). However, Ashbrook *et al.* (2010) demonstrated unsuspected trade-offs between the costs and benefits of increasing density under changing environments.

Most pre-breeding guillemots aged 2 and 3 years old, spend time in 'clubs' on tidal rocks near the main colony area (Birkhead 1976; Birkhead & Hudson 1977; Halley, Harris & Wanless 1995). Older pre-breeding birds tend to be found on ledges above the main breeding colony, although they can also be observed on non-breeding ledges within low density breeding colonies (Halley 1992; Halley *et al.* 1995). Halley, Harris & Wanless (1995) observed that older pre-breeding guillemots arrived earlier in the season than younger pre-breeding guillemots, and were seen more frequently at the colony.

Aggression is often observed within guillemot colonies and increases in frequency with density (Birkhead 1978). It is low in the pre-laying period, but high in winter, when sites are being re-established, and during incubation and chick-rearing, when there is an influx of prospecting pre-breeding guillemots (Birkhead 1978). This pattern in timing of prospecting has also been shown in other seabird species (Boulinier *et al.* 1996). The movement of breeding birds when ashore is known to be very limited, with birds flying directly to and from their breeding sites and rarely moving more than 0.5 m from their chick or egg (Harris, pers. comm.). However, field observations show that pre-breeding guillemots visit multiple subcolonies (Halley *et al.* 1995) and repeatedly attempt to enter breeding areas. When seeking to establish a breeding site for the first time, pre-breeding guillemots are persistent, often eliciting aggression from site-holding breeding birds (Birkhead 1976).

Fig. 1 Schematic of a guillemot subcolony, with age-related spatial structure (P = pre-breeding birds aged ~2 - 6 years; B = breeding birds aged ~ 6 + years).

Seabird tick, *Ixodes uriae*

Ixodes uriae (White 1852) is a member of the Ixodidae family of ticks which is composed of 14 genera and 702 species (Guglielmone *et al.* 2010). *I. uriae* has a circumpolar distribution in both the northern and southern hemisphere (Zumpt 1952) and is a vector of many viruses and bacteria including GIV (Main *et al.* 1973; Major *et al.*, 2009; Nuttall, 1984) and *Borrelia bugdorferi* sensu lato, the agent of Lyme disease (Olsen *et al.* 1993, 1995). *I. uriae* is a three-host hard tick, meaning that it passes through four developmental stages: egg, larva, nymph and adult and takes a blood meal from a different host at each developmental stage (Sonenshine 1991; Fig. 2). However, when inhabiting dense breeding colonies of seabirds such as guillemots, *I. uriae* often has no alternative hosts and therefore all three developmental stages must feed on the same host species.

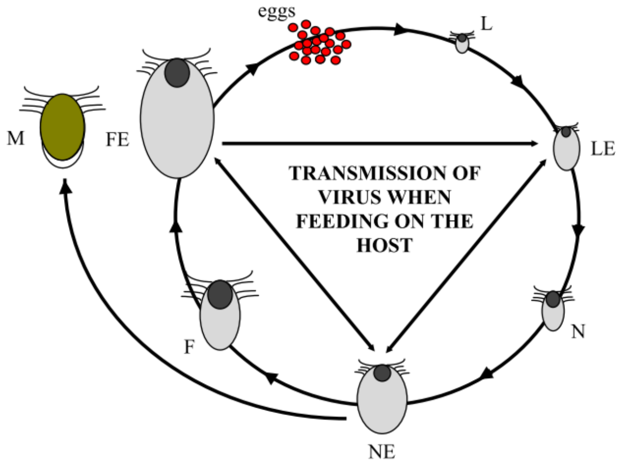


Fig. 2 Schematic diagram of *Ixodes uriae* life-cycle on the Isle of May (reproduced from Nunn, 1999).

Once a host has been found, the tick attaches and begins to feed, in the on-host phase. Feeding is a relatively lengthy process and lasts for 3-12 days, depending on the life stage (Barton *et al.* 1996; McCoy *et al.* 2002). To avoid being removed from the host by grooming during this critical stage, ticks, like *I. uriae,* have evolved to counter the hosts inflammatory and hemostatic responses to ensure feeding success, by means of a multitude of biomolecules in tick saliva, which are secreted into the host (Ribeiro *et al.* 1985; Ribeiro 1987). There is also evidence for these immunomodulatory activities facilitating the transmission of tick-borne pathogens (Labuda *et al.* 1993a; Nuttall & Labuda 2004; Machackova, Obornik & Kopecky 2006).

Once detached from its host, an engorged larva or nymph moults to the next stage. The duration of a moult is 55-145 days for engorged nymphs and 50-105 for engorged larvae (Eveleigh & Threlfall 1974; Flint & Kostyrko 1967; Murray & Vestjens 1967; Nuttall 1913). Male adult ticks do not feed (Zumpt 1952) but wait for opportunities to mate with females before dying. Mated engorged adult females convert the majority of their body mass into eggs and then die. Therefore, within a single year, more than 11 months are spent off-host, on the moist and cool underside of rocks in the case of *I. uriae,* where large groups of up to thousands of individuals can be found (Eveleigh & Threlfall 1975).

I. uriae has been found to parasitise more than 60 bird species, including auks and gulls in the northern hemisphere and penguins and albatrosses in the southern hemisphere (Dietrich *et al.* 2011). This wide host range suggests a generalist life style. While some studies suggest that the common guillemot is the preferred host of *I. uriae* (Eveleigh & Threlfall 1975), others do not support these findings (McCoy *et al.* 1999). Many seabirds, like the common guillemot, spend most of the year at sea, so ticks are only able to feed when they return to land to breed. Nevertheless, when on land, seabirds offer a large and reliable pool of hosts. Consequently, the average life span of *I. uriae* is 3 years, although a tick may complete a 2 year life cycle by feeding on both early and late breeding birds in one year. If on the other hand, a tick fails to find a host, it can delay feeding and extend its life span by 1 year (Barton *et al.* 1996).

Tick infestation levels have been shown to vary significantly between individual hosts. For example, Nunn *et al.* (2006b) demonstrated a classic negative binomial distribution (e.g. Schmid & Robinson 1972) of *I. uriae* on its guillemot host. Nunn *et al.* (2006b) also showed that the mean number of tick bites per day during peak feeding was greater for breeding *U. aalge* than pre-breeding *U. aalge*, with breeders being parasitised by more nymphs and pre-breeders being parasitised by more females. The evidence for *I. uriae*-mediated pathology is mixed. Some studies suggest direct impacts of *I. uriae* infestation on its seabird hosts, including reduction of hematocrit (Wanless, Barton & Harris 1997), behavioural change (Boulinier & Danchin 1996; Mangin *et al.* 2003) and the energetic cost of mounting an immune response (e.g. Sheldon & Verhulst 1996). However, on the Isle of May, Barton (1996) found little difference in the condition, breeding success or median laying date of infested and non-infested guillemots.

Populations of *I. uriae* are not uniform, but spatially and temporally structured. Previous studies, suggest three host-defined spatial scales: between breeding colonies, between breeding patches and between nests (Boulinier & Danchin 1996; McCoy *et al.* 1999). Anecdotal evidence suggests that a possible mechanism for these spatial patterns may be the limited ability of *I. uriae* to disperse independently (30-40 cm from resting crevice, Karpovich 1970). *I. uriae* is also nidiculous (nest-dwelling) and therefore should not have to move far to encounter a suitable host, although Muzaffar & Jones (2007) suggest that *I. uriae* uses a combination of passive waiting and active questing to find its host. Occasional long-distance dispersal between guillemot colonies or subcolonies is likely to be mediated by immatures prospecting for nest sites (Danchin 1992; Dietrich *et al.* 2011; Gómez-Díaz *et al.* 2012). Evidence also exists for shorter-distance host-mediated dispersal in other systems (Smith *et al.* 1996; Brown & Brown 2004; Brown *et al.* 2007, 2008; Watts *et al.* 2009; Jones *et al.* 2011). On the Isle of May over 99% of *I. uriae* feed between late April and early August (Barton 1996), coinciding with the guillemot breeding season. Although different life stages of *I. uriae* show slightly different seasonal patterns of activity, these overlap, with peak tick feeding occuring between May and early June, during the guillemot incubation period (Barton *et al.* 1996). This is likely to be an adaptive strategy to maximise the chance of successfully finding a host.

Great Island virus (GIV)

Great Island virus (GIV) is a species of virus within the genus *Orbivirus* (family: Reoviridae). The genus *Orbivirus* is one of seven genera containing arboviruses, defined as "viruses which are maintained in nature principally, or to an important extent, through biological transmission between susceptible vertebrate hosts by haematophagous arthropods" (Karabatsos 1985). The genus includes both insect- and tick-borne viruses, including the type species, *Bluetongue virus* (BTV), a *Culicoides*-borne orbivirus which causes a disease in ruminants and other vertebrate hosts (Attoui *et al.* 2012; Table 1). GIV was first isolated from engorged nymphs of *I. uriae* found in puffin burrows on Great Island, Newfoundland, Canada (Main *et al.* 1973). However, a range of arthropod vectors (*Argas, Ornithodoros* and other *Ixodes* ticks) and vertebrate hosts (rodents, humans and other seabirds) of GIV have since been identified (Attoui *et al.* 2012). On the Isle of May, the preferred host is the common guillemot, *Uria aalge* and the preferred vector, *I. uriae* (Nunn *et al.* 2006a).

The distribution of GIV is roughly circumpolar, including isolates from seabird colonies in Scotland, Ireland and Canada in the northern hemisphere (e.g. Main *et al.* 1973; Nunn *et al.* 2006a; Nuttall, 1984), and from Macquarie Island in the southern hemisphere (e.g. Doherty *et al.* 1975). This distribution reflects that of its main vector, *I. uriae.* Previous studies suggest that seabirds move these viruses between colonies, causing this wide geographical distribution of infection (Nuttall 1984). However, the ability of GIV isolates to readily reassort suggests that they are members of one gene pool, despite their wide geographical distribution (Moss, Ayres & Nuttall 1988). The species is composed of 36 strains; the highest level of diversity in the genus *Orbivirus* (Attoui *et al.* 2012; Table 1). Four of these have been isolated on the Isle of May (Nunn *et al.* 2006a), and temporal changes in strain abundance have been suggested, though no specific data are presented (Nunn 1999).

Table 1 *Orbivirus* species (Attoui *et al.* 2012; type species indicated with asterisk)

Virus Species Name	No. Serotypes/ strains	Hosts	Vectors
African horse sickness virus (AHSV)	9	Equids, dogs, elephants, camels, cattle, sheep, goats, predatory carnivores and humans	*Culicoides*
Bluetongue virus (BTV)*	26	Cattle, sheep, goats, camels, elephants, carnivores	*Culicoides*
Changuinola virus (CGLV)	12	Humans, rodents, sloths	Phlebotomines, Culicine mosquitoes

Chenuda virus (CNUV)	7	Seabirds	Ticks
Chobar Gorge virus (CGV)	2	Bats	Ticks
Corriparta virus (CORV)	6	Humans, rodents	Culicine mosquitoes
Epizootic hemorrhagic disease virus (EHDV)	7	Cattle, sheep, deer, camels, llamas, wild ruminants, marsupials	*Culicoides*
Equine encephalosis virus (EEV)	7	Equids	*Culicoides*
Eubenangee virus (EUBV)	4	Unknown	*Culicoides*, anopheline and Cuilicine mosquitoes
Ieri virus (IERIV)	3	Birds	Mosquitoes
Great Island virus (GIV)	36	Seabirds, rodents, humans	*Argas, Ornithodoros, Ixodes* ticks
Lebombo virus (LEBV)	1	Humans, rodents	Culicine mosquitoes
Orungo virus (ORUV)	4	Humans, camels, cattle, goats, sheep, monkeys	Culicine mosquitoes
Palyam virus (PALV)	13	Cattle, sheep	*Culicoides*, Culicine mosquitoes
Peruvian horse sickness virus (PHSV)	1	Horses	Mosquitoes
St Croix River virus (SCRV)	1	Ticks	NA
Umatilla virus (UMAV)	4	Birds	Culicine mosquitoes
Wad Medani virus (WMV)	2	Domestic animals	Boophilus, Rhipicephalus, Hyalomma, Argas ticks
Wallal virus (WALV)	3	Marsupials	*Culicoides*
Warrego virus (WARV)	3	Marsupials	*Culicoides,* anopheline and Culicine mosquitoes
Wongorr virus (WGRV)	8	Cattle, macropods	*Culicoides,* mosquitoes
Yunnan orbivirus (YUOV)	2	Cattle, sheep, donkeys	Mosquitoes

Evidence exists for both viraemic and non-viraemic transmission of GIV (Nunn *et al.* 2006a). Viraemic transmission occurs when ticks feed on hosts with abundant (and by definition detectable) virus particles in their blood, whereas non-viraemic transmission or 'co-feeding transmission' occurs when infected and uninfected ticks feed together, usually in close proximity, on hosts which have not developed viraemia (Jones *et al.* 1987; Labuda *et al.* 1993b). Evidence for non-viraemic transmission is provided by the identification of four infested guillemots which showed no detectable viraemia but on which all the ticks were infected with the same virus strain (Nunn *et al.* 2006a). Vertical transmission from one vector generation to the next has not been observed in orbiviruses (Nunamaker *et al.* 1990). On the Isle of May, overall GIV prevalence in both on-host and off-host adult female ticks is 25% (Nunn *et al.* 2006b). However, GIV prevalence varies significantly between off-host ticks collected from central and peripheral parts of the colony, and between on-host ticks collected from guillemots of different ages.

There is currently no evidence for any cytopathic effect of orbiviruses in arthropod cells. However, in most vertebrate cells, virus replication leads to the cessation of host protein synthesis, cell lysis and the release of virions (Attoui *et al.* 2012). The effects of GIV on its vertebrate hosts are less well understood. Unpublished work suggests a significant association between GIV serogroup specific antibody levels and guillemot average breeding attendance (Nunn, 1999; Fig. 3), likely due to the high cost of mounting an immune response (Sheldon & Verhulst 1996). However, there is no evidence to suggest that GIV causes overt disease in this host species. The infectious and latent periods of GIV in its host are unknown. However, the latent period for the related viruses, BTV and EHDV, in their ruminant hosts is about 7 days (MacLachlan *et al.* 1992; Quist *et al.* 1997). BTV also demonstrates an infectious period of approximately one month (Maclachlan *et al.* 1992; Koumbati *et al.* 1999). GIV is therefore likely to be a relatively acute infection.

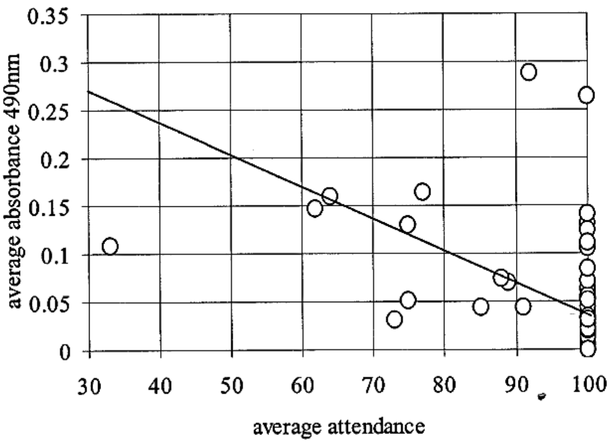


Fig. 3 Association between breeding guillemot average attendance (% of years that bird was present in the breeding colony since first recorded as a breeding adult) and serogroup specific antibody levels (reproduced from Nunn, 1999).

Antibodies against GIV have been found in sera from the Atlantic puffin, Leach's petrel (Karabatsos 1985), as well as the common guillemot (Nunn *et al.* 2006a). There is also some evidence for maternally derived antibodies (Boulinier & Staszewski 2008) for related virus species (Nuttall *et al.* 1984; Savini *et al.* 2011), and other seabird parasites (Gasparini *et al.* 2006; Staszewski *et al.* 2007). On the Isle of May, the number of birds with neutralising antibodies against one or more strains of GIV is greater in the breeding (74%) than pre-breeding (12%) population (Nunn *et al.* 2006b). The probability of a guillemot developing immunity to one or more strains of GIV depends on the duration of exposure (age-related acquired immunity; Nunn *et al.* 2006b). However, evidence suggests that GIV is highly strain diverse with only limited antibody cross neutralisation (Nunn *et al.* 2006b). This suggests that not all guillemots acquire immunity to infection to all strains by the time they recruit to the breeding population. Furthermore, the cost of being newly infected is likely to be much higher for a breeding guillemot, given the already high energetic cost of breeding.

The Isle of May

The Isle of May is a small island (about 1.8 km long by 0.5 km wide) located about 8 km off the coast, in the north of the outer Firth of Forth, Scotland (56.2° N, 2.6° W). It is managed by Scottish Natural Heritage as a National Nature Reserve and is currently home to a large and diverse population of seabirds, including about 14,000 pairs of breeding guillemots, distributed across 39 subcolonies, or sections of cliff (Fig. 4).

The guillemot population on the Isle of May has been monitored since 1981, as part of the Isle of May Long-Term Study (IMLOTS) which is run by the Centre for Ecology and Hydrology (CEH), forming part of its network of long-term monitoring sites for detecting effects of environmental change, particularly climate change. The study is partly funded by the UK's Joint Nature Conservation Committee (JNCC) as part of the Seabird Monitoring Programme. As part of this study, approximately 200 guillemot chicks are uniquely marked with engraved colour rings every year, and are subsequently resighted (e.g. Harris, Halley & Wanless 1992). These data allow estimates to be made for key characteristics such as productivity, phenology, return and survival rate (e.g. Crespin *et al.* 2006; Harris, Wanless & Barton 1996; Harris & Wanless 1988; Lewis *et al.* 2006; Wanless *et al.* 2009), which are critical for understanding population dynamics.

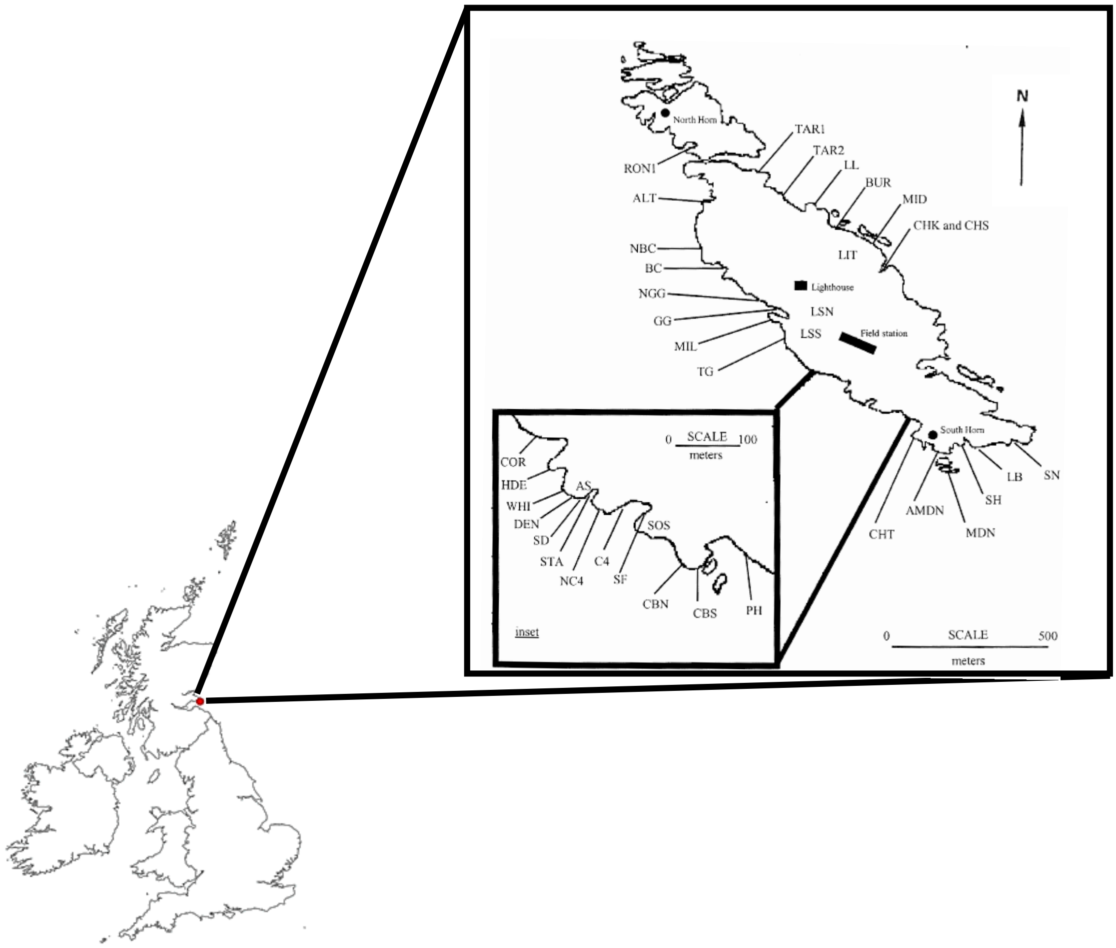


Fig. 4 A map of the Isle of May, with the location of individual subcolonies indicated (reproduced from Nunn 1999).

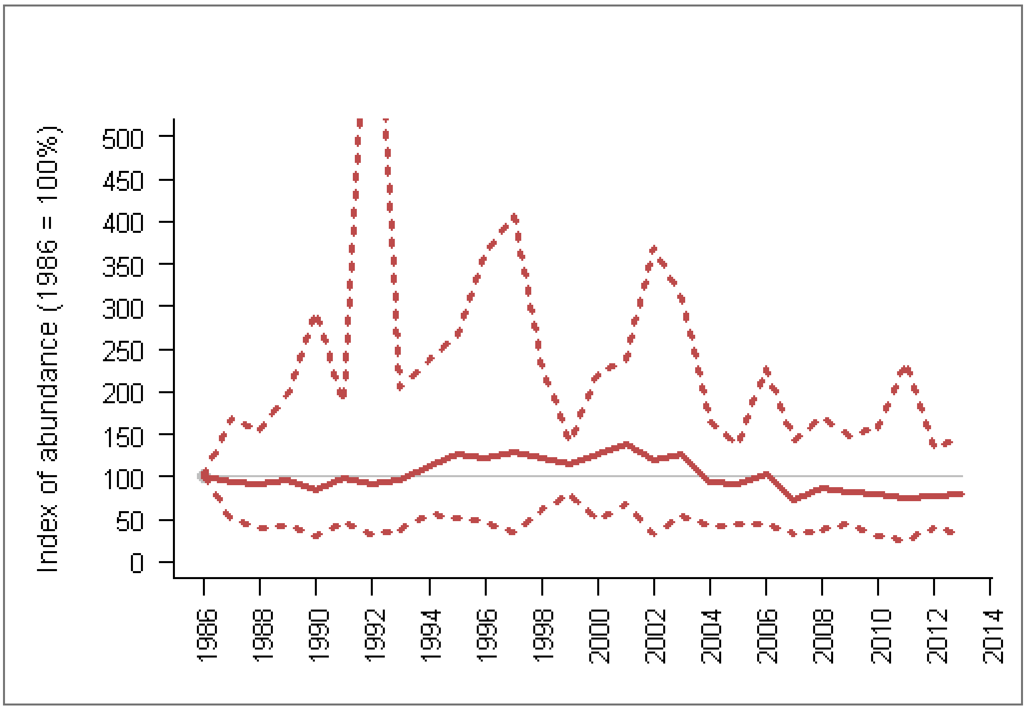
Since 2000, guillemot populations bordering the North Sea, including on the Isle of May, have experienced declines in breeding abundance (Fig. 5) and success (Table 2). In 2007 and 2008, acute declines, particularly in breeding success, were observed which coincided with a shortage of sandeels, the primary prey item for common guillemots in this area (Ashbrook *et al.* 2010; JNCC 2014; Table 2). Guillemot numbers now appear to have stabilised (Fig. 5), and events like this have not been recorded since. However, extreme events such as this have been observed in other seabird species in the past (Hamer *et al.* 1993; Phillips, Caldow & Furness 1996) and could become more frequent in the future, as sandeel populations respond to increasing sea temperatures (Arnott & Ruxton 2002; Royal Commission on Environmental Pollution 2004;

Frederiksen *et al.* 2007; Heath *et al.* 2012). This could potentially have consequences for the spatial structuring of the colony and associated parasite dynamics. For example, lower densities of breeding guillemots could facilitate the movement of pre-breeding guillemots into breeding areas of the colony, resulting in more homogenous mixing and promoting the transmission of infection (Fig. 6).

Table 2 Breeding productivity of the common guillemot in Scotland (JNCC 2014), and on the Isle of May ("Isle of May Breeding Season Summaries"; Newell *et al.* 2013), since 2000.

	Scotland	**Isle of May**
Year	Chicks fledged per pair	Chicks fledged per pair
2000	0.73	0.73
2001	0.64	0.63
2002	0.71	0.68
2003	0.60	0.68
2004	0.39	0.50
2005	0.41	0.63
2006	0.37	0.41
2007	**0.19**	**0.28**
2008	**0.26**	**0.63**
2009	0.59	0.75
2010	0.50	0.80
2011	0.43	0.71
2012	0.57	0.79
2013	0.42	0.69

a)



b)

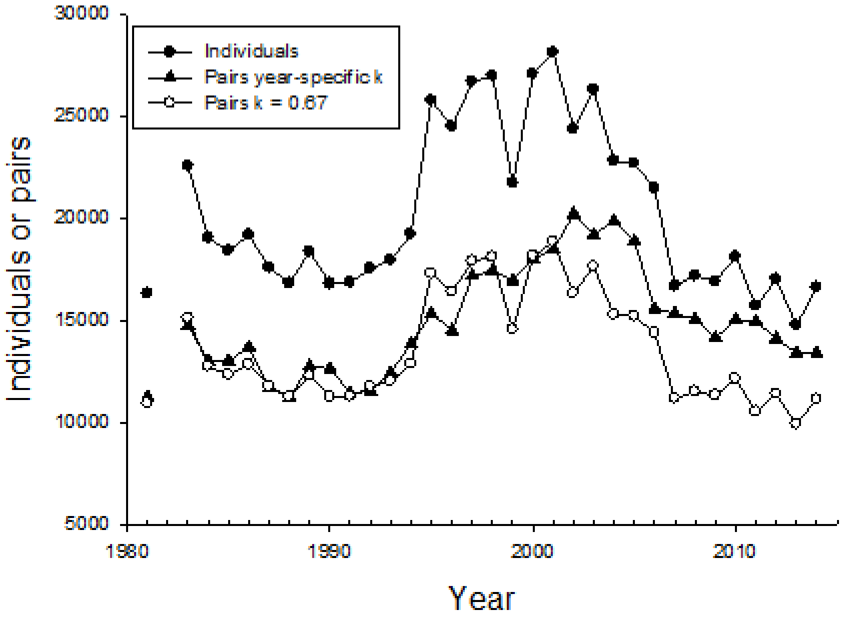


Fig. 5 a) Trend in abundance index (solid line) of common guillemot in Scotland, 1986-2013 with 95% confidence limits (dotted lines; drawing of upper limit restricted to preserve detail in the abundance index). Based on SMP data (JNCC 2014), b) Counts of birds (filled circles) and the calculated number of breeding pairs of guillemots on the Isle of May 1981-2014 using a year-specific value of a correction factor k (triangles) and a generally accepted value of 0.67 (open circles) (Harris *et al.* 2015).

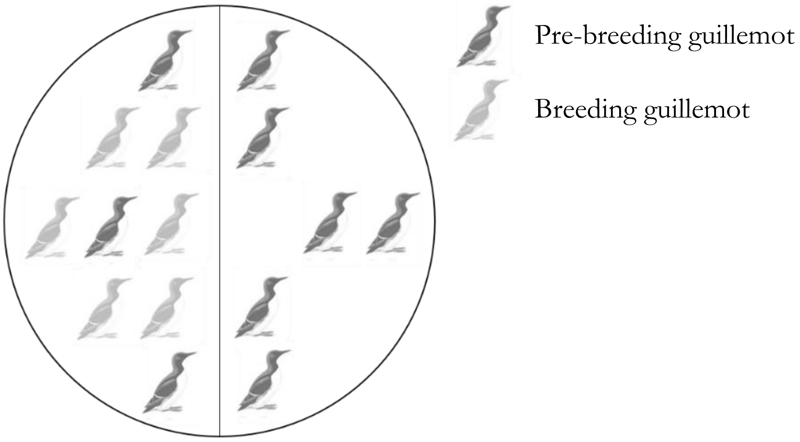


Fig. 6 Schematic of a disturbed guillemot subcolony, with movement of pre-breeding birds into an increasing number of unoccupied breeding sites.

In contrast to the Isle of May, another major guillemot colony on Skomer Island, south west Wales has been steadily increasing in numbers at a rate of 5% per annum over the last three decades (Meade *et al.* 2013). Productivity rates have remained stable, at an average of 0.64 chicks fledged per site per year (JNCC 2014). This is likely to be due to differences in chick diets between colonies (Anderson *et al.* 2014). Another major colony outside Britain, Hornøya, Norway, has also been increasing in numbers for the last 25 years, and at a rate of 10% per annum since 2004 (Barrett *et al.* 2015).

Modelling spatially structured wildlife systems

Modelling of measles dynamics in the 1990s (Grenfell 1992; Tidd, Olsen & Schaffer 1993; Grenfell *et al.* 1994) suggested that the level of coupling in a metapopulation, defined as a set of spatially separated populations, influenced the persistence of infection within a human population. Since then, many studies have investigated the effects of spatial structure on wildlife parasite dynamics (e.g. Brown *et al.* 2008; Clancy, O'Callaghan, & Kelly 2006; Craft *et al.* 2011;

Fulford, Roberts & Heesterbeek 2002; Hess 1996; Klepac *et al.* 2009; Smith *et al.* 2002; Swinton *et al.* 1998; Watts *et al.* 2009; Woodroffe 1999). For example, a stochastic compartmental approach was used by Swinton *et al.* (1998) to model the spread of phocine distemper virus in the North Sea population of harbour seals. The seal population was divided into a system of identical patches representing individual seal haulouts, each with simple SEIR (susceptible-exposed-infectious-recovered) dynamics. Mixing was assumed to occur both within patches, and between nearest-neighbour patches. They concluded that the persistence of the virus was unlikely in the seal population because of the high level of between-patch mixing coupled with the small patch size and the low birth rate (leading to a low input of susceptibles).

A similar approach was applied by Smith *et al.* (2002) to model the spread of racoon rabies in Connecticut. However, patches in this study represented state townships with short-distance transmission occurring between adjacent townships, as well as some long-distance translocation events. Patches were also assumed to have different demographic and geographic features. This model highlighted the importance of rivers in limiting the propagation of the virus.

In the case of seabirds, Clancy, O'Callaghan & Kelly (2006) use a simple SEIR approach to model the spread of a novel pathogen, the H5N1 strain of avian influenza, in a guillemot colony to investigate why epidemics are not seen to occur more frequently. Although not explicitly spatial, the model was applied to chick and adult populations separately, only taking into account infections within each of these. Within this model, guillemot chicks are exposed to infection in the colony for an average of 22 days, leave the colony and then return after 4-5 years as susceptible recruits, with the number of new recruits balancing the number of deaths. Clancy, O'Callaghan & Kelly (2006) conclude that the disease dynamics are driven by the adult population, with infection and subsequent immunity of long-lived adults likely preventing the virus from continually re-establishing itself.

Modelling vector-borne transmission

Anderson & May (1991) propose a general framework for modelling vector-borne transmission, which consists of two equations describing changes in the proportion of infectious hosts, y, and the infectious vectors, $\hat{y}$, in a constant total population,

$$\frac{dy}{dt} = \left(\frac{ab\hat{N}}{N}\right)\hat{y}(1-y) - \gamma y, \qquad (1)$$

$$\frac{d\hat{y}}{dt} = acy(1-\hat{y}) - \mu\hat{y},$$

where, N = size of the host population, $\hat{N}$ = size of vector population, a = rate of biting on hosts by single vector, b = proportion of infectious bites on hosts that produce infection, μ = per capita rate of vector mortality, γ = per capita rate of human recovery from infection, c = proportion of bites by susceptible vectors on infected hosts that produce infection.

This framework assumes that the net rate of transmission is limited by the biting rate of the vector times the number of vectors per host, regardless of the absolute densities of vectors and hosts (frequency dependence). The basic reproductive ratio in this simple vector transmission model is defines as

$$R_0 = \frac{ma^2bc}{\mu\gamma}, \qquad (2)$$

where m = the ratio $\hat{N}/N$, or the number of vectors per host.

And the force of infection (λ), is defined as

$$\lambda = \left(\frac{ab\hat{N}}{N}\right)\hat{y}, \qquad (3)$$

This means that a host which is infectious for a period of time, $1/\gamma$, receives an average number of bites from m susceptible vectors with a biting rate, a, a proportion of which are infectious, c.

This gives a total of amc/γ vectors infected by the primary host case. If each of these vectors then survives for an average time of $1/\mu$, making ab/μ infectious bites, then the number of secondary cases is the product of amc/γ and ab/μ, as above.

As well as this general framework, there are many examples of more complex models for the spread of tick-borne pathogens in wildlife populations (e.g. Bolzoni *et al.* 2012; Hudson *et al.* 1995; Nonaka, Ebel & Wearing 2010; O'Callaghan *et al.* 1998), including spatially structured models (e.g. Gaff & Gross 2007; Watts *et al.* 2009). For example, Anderson and May (1991) propose one way of incorporating host immunity as a function of exposure into an age-structured model for malaria

$$\begin{aligned} \frac{dx}{da} &= v(\lambda,\tau)z-(\lambda+\mu_1)x, \\ \frac{dy}{da} &= \lambda x-(\gamma+\mu_1)y, \\ \frac{dz}{da} &= \gamma y-(v(\lambda,\tau)+\mu_1)z, \end{aligned} \tag{4}$$

where, $x(a)$, $y(a)$ and $z(a)$ = the proportion of susceptible, infected and immune individual hosts of age a respectively, μ_1 = rate of human mortality, γ = the recovery rate which takes individual hosts into immune class, $v(\lambda,\tau)$ = average per capita rate of loss of host immunity.

Within this framework, if some proportion of the host population is removed from the susceptible population by developing immunity, then the probability of a susceptible vector biting an infected host and acquiring infection will be reduced (Eqn. 1), resulting in a lower force of infection (Eqn. 3).

The herd immunity hypothesis

Herd immunity is traditionally defined as the resistance of a group because of the immunity of a large proportion of its members (Fox *et al.* 1971), and is a term commonly seen in the historical medical literature on the prevention and control of human infectious diseases by immunisation (including measles: e.g. Cvjetanovic, Grab & Dixon 1982; rubella: e.g. Gremillion, Gengler & Lathrop 1978; diphtheria: e.g. Miller *et al.* 1972; and polio: e.g. Melnick 1978). However, John & Samuel (2000) suggest a slightly different definition for herd immunity, as the proportion of population that is immune, and distinguish this from the reduction of infection in the susceptible segment of the population as a result of this immune proportion, which they call the 'herd effect'.

For the endemic virus, GIV, previous studies show that guillemots acquire immunity with age. Furthermore, although infestation levels are significantly higher for breeding than pre-breeding guillemots, the prevalence of infection is significantly lower in these ticks (Nunn *et al.* 2006b). This suggests that partial herd immunity operates in areas occupied by breeding birds. The percentage of the breeding population that is immune is a direct measure of this effect ('herd immunity' as defined by John & Samuel 2000), which drives down the prevalence of infection in the tick population and hence the force of infection, or per susceptible risk of infection acting on these birds ('herd effect'). This suggests that the spatial structure of the guillemot colony may be an important factor driving GIV transmission.

In the context of immunisation, if more than a critical proportion (p_c) of a population is vaccinated, then the probability of a susceptible individual coming in contact with an infectious individual becomes so small that the infection cannot persist. This is because the basic reproductive ratio, R_0, defined as the number of secondary cases produced by a single infectious case, falls below one (Anderson & May 1991). The critical proportion or threshold is defined as

$$p_c = 1 - \frac{1}{R_0}, \tag{5}$$

This means that it is not necessary to vaccinate the whole population to avoid an epidemic. Although herd immunity in this context ultimately leads to eradication of an infection, this is not the case in this wildlife system, where GIV infection is maintained within both pre-breeding and breeding areas of the colony, but with a significantly lower force of infection acting in the latter area (Nunn *et al.* 2006b). This distinction between herd immunity as partial or total resistance is pointed out by Fine (1993).

Thesis aims and structure

This thesis seeks to investigate the role of guillemot spatial structure in the transmission of a tick-borne seabird virus, GIV, using a uniquely multi-disciplinary approach, framed by a simple epidemiological model. In particular, it addresses the following questions:

1. How important is the spatial structure of the guillemot colony for GIV dynamics? This is discussed in Chapter 2: **Modelling the spread of a tick-borne virus, *Great Island virus*, within a common guillemot (*Uria aalge*) breeding colony: the importance of socio-spatial structure**. I present a simple epidemiological model of the colony, which highlights the importance of spatial structure within the guillemot colony, confirms the herd immunity hypothesis and more specifically, identifies a key missing parameter, the rate of transmission between pre-breeding and breeding areas of the colony. This chapter is the foundation for the rest of the thesis, identifying the key parameters for this system, which are then estimated in the field or the laboratory.

2. What role does independent tick dispersal play in driving GIV transmission between different areas of the colony? A key assumption of the model, that independent tick dispersal is

negligible, is explored in Chapter 3: **How far is a seabird tick able to move in search of a host? The first description of the movement of host-seeking *Ixode uriae* (White, 1852) in an artificial arena environment**. I provide the first description of the movement of host-seeking *I. uriae* in an artificial arena. This chapter suggests that *I. uriae* has the potential to walk ranges described anecdotally in the literature, in just a few minutes, but stresses the importance of further experiments in the field.

3. What role does guillemot-mediated tick dispersal play in driving GIV transmission between different areas of the colony? Assuming that guillemot-mediated dispersal is the major route for movement of ticks, the key mixing parameter identified in the model is quantified in Chapter 4: **Do immature common guillemots (*Uria aalge*) pose an infection risk to the breeding colony? Estimating the current rate of immature-mediated parasite dispersal from field observations**. I characterise the attendance rates, and spatial attendance patterns of immature guillemots across the colony, during peak tick feeding. This chapter suggests that some immature guillemots spend a significant amount of time in breeding areas, although this varies both spatially and temporally.

4. What is the current risk of GIV infection for guillemots and does this vary with strain? Although rough estimates have been previously made for four GIV strains, I apply finite mixture modelling techniques in order to answer this question more fully in Chapter 5: **Improved estimates of age- and strain-specific seroprevalence and force of infection for a tick-borne virus, *Great Island virus*, in a wild population of common guillemots (*Uria aalge*)**. I provide improved estimates with confidence intervals, of the seroprevalence and force of infection for these four strains of GIV, as well providing the first estimates for additional strains of this highly strain-diverse virus. This chapter highlights the importance of understanding strain-specific differences in transmission.

5. What is the potential future risk of infection from GIV for guillemots, as a result of changes to the spatial structure of the colony? All parameter estimates from previous chapters are inputted into my initial model in Chapter 6: **Modelling the spread of an endemic tick-borne virus, *Great Island virus*, within a common guillemot (*Uria aalge*) breeding colony: estimating current and future infection risk for breeding individuals of a species of conservation importance**. This chapter provides evidence for substantial spatial heterogeneity in current infection risk within the colony. Based on this, it predicts that GIV transmission within the guillemot colony may increase as a result of declining breeding abundance and success, with more frequent or extreme disruption leading to a higher risk of infection within the colony.

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Modelling the spread of a tick-borne virus, *Great Island virus*, within a common guillemot (*Uria aalge*) breeding colony: the importance of socio-spatial structure

Abstract

Seabirds are likely to play an important role as reservoirs and dispersal agents of infection. However, little is known about parasite dynamics within seabird colonies, which are often highly spatially structured. Here, I present a simple two-patch, ordinary differential equation (ODE) model for the transmission of a parasite in a spatially-structured host population. I use it to describe the transmission of *Great Island virus* (GIV), a tick-borne virus, within a large common guillemot (*Uria aalge*) colony, composed of spatially-distinct pre-breeding and breeding subpopulations. This model was parameterised using the available literature for this system wherever possible and those parameters without estimates, like the transmission rates, were varied. I use past estimates for the risk of infection from the literature, to investigate a) pre-disturbance, and b) post-disturbance scenarios. I show evidence for negligible transmission between pre-breeder and breeder patches, when the colony is relatively dense and undisturbed. This allows a difference in the risk of infection to be maintained between the two patches. However, I also show evidence for the risk of infection, particularly in the breeder patch, being highly sensitive to the rate of between-patch rate transmission (with the risk of infection for breeding birds more than tripling when transmission rates are equal between and within patches). The sensitivity of this system may be further increased by a higher rate of transmission within the breeder patch. This suggests that the dynamics of seabird parasites such as GIV may be highly sensitive to changes in the socio-spatial structure of a colony, which have been observed in recent years. This may have both local consequences for the breeding colony, as well as global consequences for the circulation of infection.

Introduction

Seabird colonies are highly spatially structured due to seabirds' territorial behaviour over nest sites and the structure of their cliff habitat. Epidemiological models of other wildlife host-parasite systems suggest that the socio-spatial structure of a wildlife host population can have significant effects on parasite dynamics (Swinton *et al.* 1998; Smith *et al.* 2002). Evidence exists for spatial structuring of parasite populations within and between seabird colonies (Boulinier & Danchin 1996; McCoy *et al.* 1999, 2003). However, few studies have used epidemiological models to investigate the dynamics of infection within seabird colonies (O'Regan *et al.* 2008, 2013), and I know of only one that has attempted to incorporate spatial structuring (Clancy, O'Callaghan & Kelly 2006). Therefore, little is known about the effects of spatial structuring in seabird colonies on parasite dynamics. To address this knowledge gap, I investigate the importance of spatial structuring within a large guillemot colony for the dynamics of tick-borne virus using a simple two-patch system of ordinary differential equations (ODEs).

Guillemot-tick-virus system

The Common guillemot (*Uria aalge;* hereafter guillemots) breeds in large, dense colonies usually on cliffs and is parasitised by the seabird tick (*Ixodes uriae)* and the parasites it transmits (Olsen *et al.* 1993; Nunn *et al.* 2006a; Duneau *et al.* 2008; Major *et al.* 2009; Arnal *et al.* 2014; Duron, Jourdain & McCoy 2014). Among these parasites is the orbivirus *Great Island virus* (GIV; Attoui *et al.* 2012). In one large colony in south-east Scotland, the main host of GIV has been identified as the Common guillemot (Nunn *et al.* 2006a). Globally, the hosts of GIV include seabirds, rodents and humans (Attoui *et al.* 2012). The guillemot-tick-virus system is a useful model for exploring the effects of host spatial structuring on wildlife parasite dynamics because there is an age-related spatial structure within guillemot colonies; with adults breeding on cliff ledges, while younger pre-breeding birds (typically aged 2 – 6 years) congregate around the edges of the breeding colony (Halley 1992).

Clancy, O'Callaghan & Kelly (2006) modeled the spread of a novel pathogen, H5N1, through a guillemot colony and assumed no pre-existing herd immunity. However, differences in the level of immunity and infection risk between ages have been documented in other systems (Anderson & Trewhella 1985; Nielsen *et al.* 2002; Long *et al.* 2010), and within this system, there is evidence for age-related acquired immunity to GIV by guillemots (Nunn *et al.* 2006b). The mean number of tick bites per day during peak tick feeding, given by the observed mean number of ticks per host divided by the average duration of the feeding period (nymphs: Barton, Harris & Wanless 1995; adult females: Finney, Wanless & Elston 1999), is also significantly higher among breeding than pre-breeding birds. However, the prevalence of infection is significantly lower among the ticks which infect breeding birds, and so is the resulting force of infection (Nunn *et al.* 2006b). This suggests that herd immunity is operating in areas occupied by breeding birds. However, it is unknown how sensitive this system is to changes in the socio-spatial structure of the colony.

The movement of breeding guillemots when ashore is known to be very limited. However, field observations show that pre-breeding guillemots visit multiple subcolonies (Halley, Harris & Wanless 1995) and repeatedly attempt to enter breeding areas. This behaviour is believed to be a key part of the recruitment process, during which a pre-breeding bird obtains a breeding site and a partner prior to breeding (Halley 1992). However, since 2000, guillemot populations bordering the North Sea have experienced declines in breeding success and abundance (JNCC 2014). This could increase the number of unoccupied breeding sites available and make it easier for pre-breeding birds to enter breeding areas. Acute decreases in breeder attendance were also observed in 2007 and 2008, likely due to limitations on food and resulting in poor chick attendance (Ashbrook *et al.* 2010). Although no unattended chicks have been recorded since, extreme disturbance events such as this could further facilitate the penetration of pre-breeding birds into breeding areas, particularly if they were to increase in frequency. This potential change in the spatial organisation could have serious consequences for the colony on a local-scale, as well as the circulation of seabird parasites on a global-scale, and highlights the importance of furthering our understanding of the relationship between seabird spatial structure and parasite dynamics.

Model of the guillemot-tick-virus system

The model was composed of two patches: a pre-breeder patch (P) and a breeder patch (B; Fig. 1), to represent the spatial structuring within the colony. Daily infection dynamics were modelled, with all rates expressed per day. Although multiple strains of GIV have been identified (Nunn *et al.* 2006a), I model the spread of a single average virus strain. I also approximate GIV dynamics with those of a directly-transmitted parasite. The ODEs that describe my model are as follows (where, $N_B = S_B + I_B + R_B$)

$$\frac{dS_P}{dt} = (b - sN_B)N_B - (1-z)\beta_{PP}S_PI_P - z\beta_{PB}S_PI_B - \mu_PS_P - rS_P, \tag{1}$$

$$\frac{dI_P}{dt} = (1-z)\beta_{PP}S_PI_P + z\beta_{PB}S_PI_B - \gamma I_P - I_P(\mu_P + \alpha_P) - rI_P,$$

$$\frac{dR_P}{dt} = \gamma I_P - \mu_PR_P - rR_P,$$

$$\frac{dS_B}{dt} = -(1-z)\beta_{BB}S_BI_B - z\beta_{BP}S_BI_P - \mu_BS_B + rS_P,$$

$$\frac{dI_B}{dt} = (1-z)\beta_{BB}S_BI_B + z\beta_{BP}S_BI_P - \gamma I_B - I_B(\mu_B + \alpha_B) + rI_P,$$

$$\frac{dR_B}{dt} = \gamma I_B - \mu_BR_B + rR_P,$$

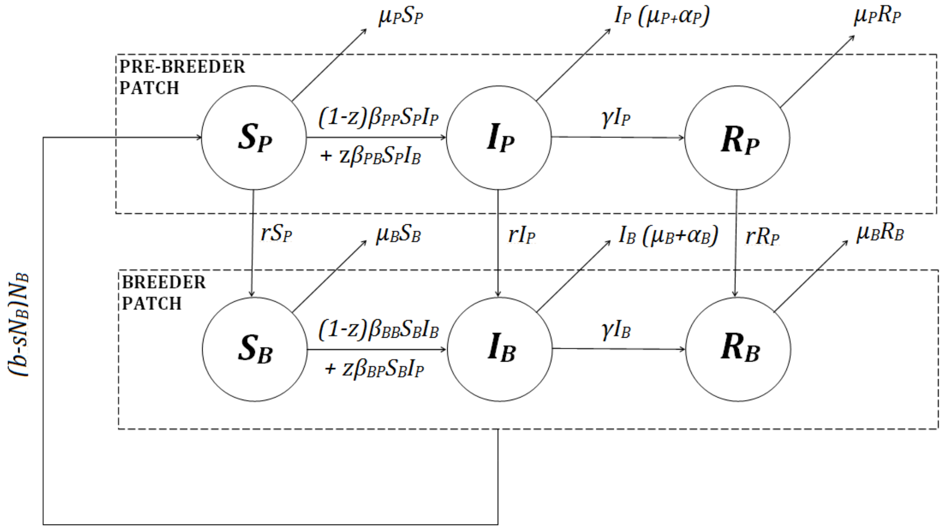

Fig. 1 A compartmental diagram of the guillemot-tick-virus model (state variables: S_P = susceptible pre-breeding, I_P = infectious pre-breeding, R_P = recovered pre-breeding; S_B = susceptible breeding, I_B = infectious breeding, R_B = recovered breeding, N_B = total breeding; see Table 1 for parameter definitions).

This system of ODEs was solved numerically for a set of initial values, defined by the disease-free equilibrium, in R 3.0.2 (R Core Team 2013) using the lsoda function in the deSolve package (Karline, Petzoldt & Setzer 2010). Long-term equilibrium behaviour is presented throughout, with the system of equations being solved for a large range of times to ensure that the global equilibrium was reached. Root solving-methods were attempted but these were found to be highly sensitive to initial values.

Demography

I assume that birds are born into the pre-breeder patch and remain there for $1/r$ years on average, before recruiting into the breeding population, at a rate r. Pre-breeding birds are assumed to die at a natural rate of μ_P, and breeding birds at a rate μ_B. Negative density dependent effects are known to occur in the colony at very high densities (Kokko, Harris & Wanless 2004) therefore I assume that density dependent population regulation acts at a rate s, on the maximum reproduction rate, b, of the entire breeding population, N_B. In the absence of infection, the model reduces to a description of the demography of the birds in the two patches. Its analytical equilibria are:

$$S_P^* = \frac{\mu_B}{sr}\left(b - (\mu_P + r)\left(\frac{\mu_B}{r}\right)\right), \qquad (2)$$

$$S_B^* = \frac{1}{s}\left(b - (\mu_P + r)\left(\frac{\mu_B}{r}\right)\right) = S_P^*\left(\frac{r}{\mu_B}\right),$$

Infection

Within each patch, birds are classified as either susceptible (S_P, S_B), infectious (I_P, I_B) or recovered (R_P, R_B), giving a total of six state variables. Infection is described in the standard way for a simple SIR model. However, the transmission coefficient, β, a composite parameter of the overall contact rate and the probability of transmission given a contact, is scaled by the proportion of time spent by pre-breeding birds in the breeder patch, z. Therefore, birds become infectious at a rate $z\beta$ or $(1-z)\beta$, recover at a rate γ and die at a viral induced rate of α. The exact latent period of GIV is not known and so is not included for the sake of simplicity.

For the sake of simplicity, I assume that birds mix homogeneously within patches and that transmission of infection is density dependent (pseudo-mass-action) i.e. that the number of contacts increases with the guillemot population size. Although *I. uriae* is not explicitly included in this model, tick dynamics are encompassed within the β term (Kermack & McKendrick 1927). There are four different transmission rates defined in this model which represent within-patch and between-patch transmission. $(1-z)\beta_{PP}$ and $(1-z)\beta_{BB}$ represent within-patch transmission. $(1-z)\beta_{PP}$ is the daily transmission rate from an infectious pre-breeding to a susceptible pre-breeding bird, and $(1-z)\beta_{BB}$ is the daily transmission rate from an infectious breeding to a susceptible breeding bird. $z\beta_{PB}$ and $z\beta_{BP}$ represent between-patch transmission. $z\beta_{PB}$ is the transmission rate from an infectious breeding to a susceptible pre-breeding bird, and $z\beta_{BP}$is the transmission rate from an infectious pre-breeding to a susceptible breeding bird. For the sake of simplicity, I assume that the interactions between patches are symmetrical, so that the transmission rate from pre-breeding to breeding birds is the same as the transmission rate from breeding to pre-breeding birds, i.e. $z\beta_{BP} = z\beta_{PB}$. I investigate the effects of varying $(1-z)\beta_{BB}$ and the between-patch transmission rate.

I assume that GIV is an acute infection and that upon recovery from infection an individual is immune for the rest of its life, given evidence of long-lasting neutralizing antibodies (Nunn *et al.* 2006b). A one day step length was chosen in order to clearly show the dynamics of acute GIV infection in the population. The recovery term was assumed to be the same for both patches, representing the biological assumption that the natural course of GIV infection is independent of host age. This model also assumes that individuals must pass through the pre-breeder patch to reach the breeder patch. Furthermore, if there is some rate of infection in the pre-breeder patch, there will be some proportion of individuals recruiting into the breeder patch that will already be recovered. This represents age-acquired immunity in the system, as shown by Nunn *et al.* (2006b). Although there is no formal evidence to suggest that GIV causes overt disease in guillemots, the model also includes a viral induced death rate, α, which is experienced only by birds in the infectious state on top of the natural death rate, μ. This is the simplest scenario and is also assumed by Nunn *et al.* (2006b). In the presence of infection, it was not possible to solve the equilibrium analytically so numerical methods were used.

Parameter estimation

The model includes 11 parameters in total, including six parameters describing the infection process and five parameters describing the demographic process (Table 1). All parameters refer to average estimates, and are specific to the guillemot colony on the Isle of May, a small island (about 1.8 km long by 0.5 km wide) located about 8 km off the coast, in the north of the outer Firth of Forth, south-east Scotland (56.2° N, 2.6° W).

Demography

Birds move from the pre-breeder to the breeder patch at a recruitment rate equivalent to becoming a breeder at an average age of over 6 years (Harris, Halley & Swann 1994; Lewis *et al.* 2006; Harris pers. comm.). All demographic rates therefore refer to these two age groups (1-6 years and > 6 years). The value for the natural death rate of breeding birds (μ_B) was estimated from Harris & Wanless (1995) as 1.4×10^{-4} per day, and is equivalent to an average of 20 years spent in the breeder patch. The pre-breeding natural death rate is much more variable and was crudely estimated as an average of 2.7×10^{-4} per day (mean from Reynolds *et al.*, 2009). Crespin *et al.* (2006) estimate age-specific transition (recruitment) probabilities from those non-breeders present at the colony to breeders at 0.036 per year for 4 year olds and 0.312 per year for 5 years and older. However, a constant average recruitment rate of 4.6×10^{-4} per day is used in this model. Assuming survival, this is equivalent to an average of six years spent in the pre-breeder patch. Together, these figures approximately add up to the average lifespan of a guillemot (Cramp & Simmons 1985). These rates also indicate that more than a third of pre-breeding birds die before recruiting. The guillemot birth rate, b, or the maximum number of chicks successfully fledged per breeding bird per day, was taken to be an average of 1.05×10^{-3} per day (estimated from Harris & Wanless 1988). The value of the density-dependent parameter, s, was not known. A range of different values were explored in the model and a value of 2×10^{-8} per day was chosen as it yielded equilibrium population numbers consistent with those observed on the Isle of May. These were 40,000 breeding birds and 10,000 pre-breeding birds (2005 figures from Reynolds *et al.* 2009).

Infection

There is no data on the infectious period of GIV. However, data from Koumbati *et al.* (1999) and MacLachlan *et al.* (1992) for Bluetongue virus suggest an average infectious period of 30 days in its ruminant host, and previous studies (Nunn *et al.* 2006b) have used this information to set the recovery rate, γ equal to 1/30 or 3.3×10^{-2} per day. The average overall death rate of an infectious individual is assumed to be four times the natural death rate of a breeding bird (5.6×10^{-4} per day), for both breeding and pre-breeding birds as in Nunn *et al.* (2006b). This small value is assumed because evidence suggests that birds survive frequent GIV infections (Nunn pers. comm.). The difference in α values (Table 1) is due to the fact that I assume that the natural death rate (μ) is different between patches but that the mortality rate for an infectious individual ($\mu + \alpha$) is the same.

Table 1 Average parameters used in simulations and their provenance.

Symbol	Average value (per day)	Description and reference
b	1.05×10^{-3}	Guillemot birth rate, from Harris & Wanless (1988).
s	Unknown	Density dependent crowding parameter of guillemot. Estimated in model (2×10^{-8}).
μ_P	2.7×10^{-4}	Natural death rate of pre-breeding guillemots, estimated from Reynolds *et al.* (2009).
μ_B	1.4×10^{-4}	Natural death rate of breeding guillemots, from Harris & Wanless (1995).
r	4.6×10^{-4}	Recruitment rate of pre-breeding guillemots into breeding population, estimated from Crespin *et al.* (2006).
γ	3.3×10^{-2}	Guillemot recovery rate, assumed by Nunn *et al.* (2006b).
α_P	2.9×10^{-4}	Pre-breeding guillemot viral induced death rate, assumed by Nunn *et al.* (2006b).
α_B	4.2×10^{-4}	Breeding guillemot viral induced death rate, assumed by Nunn *et al.* (2006b).
$(1\text{-}z)\beta_{PP}$	Unknown	Rate of viral transmission between pre-breeding guillemots via ticks, estimated in the model (5.2×10^{-6}) and validated by Nunn *et al.* 2006b.
$(1\text{-}z)\beta_{BB}$	Unknown	Rate of viral transmission between breeding guillemots via ticks, estimated in the model and validated by Nunn *et al.* 2006b (initially 5.2×10^{-6} and then denoted in figure

		legends).
$z\beta_{BP}$	Varied	Rate of viral transmission from pre-breeding to breeding guillemots via ticks, estimated in the model and validated by Nunn *et al.* 2006b (initially 0 and denoted in figure legends).
$z\beta_{PB}$	Varied	Rate of viral transmission from breeding to pre-breeding guillemots via ticks, estimated in the model and validated by Nunn *et al.* 2006b (initially 0 and denoted in figure legends).

Model analysis

I assume that the force of infection, or per susceptible risk of infection, within a patch (λ_B or λ_P), is composed of two terms, a within-patch transmission term and a between-patch transmission term. The within-patch transmission term is defined by the within-patch transmission rate multiplied by the equilibrium number of infectious individuals within the same patch. The between-patch transmission term is defined by the between-patch transmission rate multiplied by the equilibrium number of infectious individuals in the other patch.

$$\lambda_B = (1-z)\beta_{BB}I_B^* \quad + \quad z\beta_{BP}I_P^*, \tag{3}$$

$$\lambda_P = \underbrace{(1-z)\beta_{PP}I_P^*}_{\text{within-patch transmission}} \quad + \quad \underbrace{z\beta_{PB}I_B^*}_{\text{between-patch transmission}}$$

a) Pre-disturbance scenario

As no estimates for transmission rates were available, they were initially varied to give a realistic force of infection, given estimates for the force of infection (estimates from 1993-1996, Nunn *et al.* 2006b). Realistic absolute and relative force of infection values were reached (pre-breeder patch = 6.7×10^{-4} per day, breeder patch = 3.4×10^{-4} per day; fold-difference between patches = 2.0) at $(1\text{-}z)\beta_{PP} = (1\text{-}z)\beta_{BB} = 5.2 \times 10^{-6}$ per day (assuming $z\beta_{PB} = z\beta_{BP} = 0$; Nunn *et al.* 2006b). The model therefore suggests that during the period 1993-1996, when the colony was relatively dense and undisturbed, there was likely to be negligible between-patch transmission occurring, allowing a difference in the force of infection between the two patches to be maintained (see Fig. 2).

b) Post-disturbance scenario

Varying between-patch transmission rate

To investigate the sensitivity of the system to changes in the socio-spatial structure of the colony, I increased the between-patch transmission rate and simulated the resulting force of infection in the two patches (Fig. 2). At higher levels of between-patch transmission, the difference in the forces of infection between the two patches decreases until they become equal at $\lambda_P = \lambda_B = 1.2 \times 10^{-3}$ when $z\beta_{PB} = z\beta_{BP} = (1\text{-}z)\beta_{PP} = (1\text{-}z)\beta_{BB} = 5.2 \times 10^{-6}$ per day. This is the scenario in which the population is well mixed and there is effectively no distinction between the breeder and pre-breeder patches, and is as expected given Eqn. 3. I also found that the breeder force of infection, λ_B, was more sensitive to the varying between-patch transmission rate than the pre-breeder force of infection, λ_P. In fact, within this range, the value for λ_P almost doubled (6.7×10^{-4} to 1.2×10^{-3} per day), whereas the value for λ_B more than tripled (3.4×10^{-4} to 1.2×10^{-3} per day), increasing as the equilibrium number of infectious pre-breeding birds, $I_P{}^*$, increased (128 to 166). This value dominates the between-patch transmission term in the breeder patch (Eqn. 3).

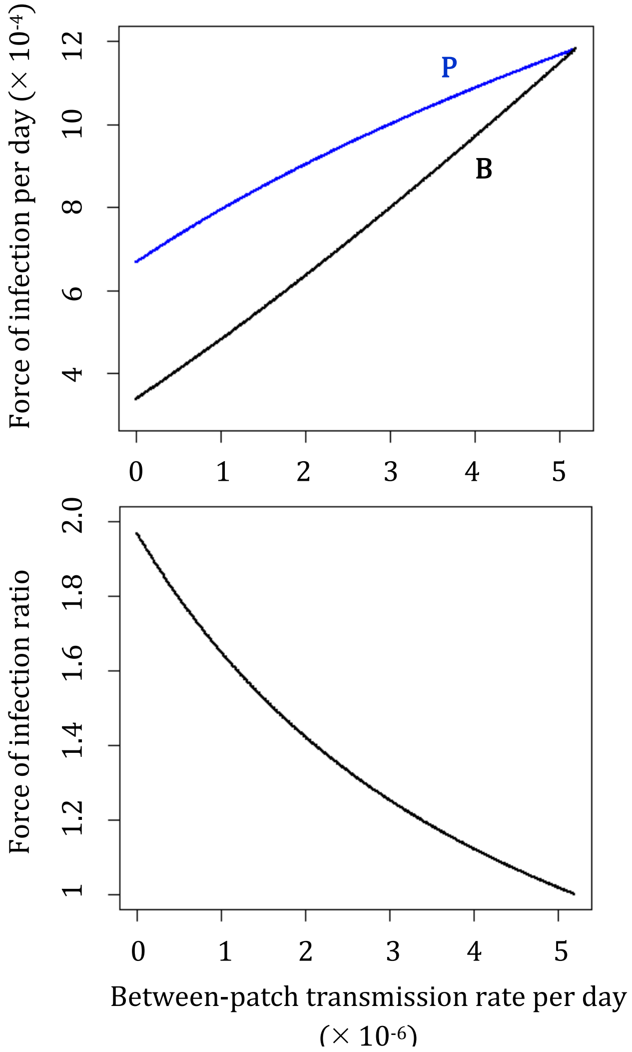

Fig. 2 Force of infection in each patch (λ_P and λ_B) and force of infection ratio (λ_P/λ_B; Eqn. 3) with increasing between-patch transmission rate ($z\beta_{BP} = z\beta_{PB}$).

Varying within-patch transmission rate in breeder patch

Simulations resulting from varying the within-patch transmission rate in the breeder patch and the resulting ratio of the forces of infection (λ_P/λ_B) are shown in Fig. 3. Although the difference between the force of infection in the two patches decreases as β_{BB} increases, the between-patch transmission rate at which the forces of infection equalize also decreases and at high levels of within-patch breeder transmission, the force of infection ratio falls below one, representing a higher breeder than pre-breeder force of infection. This is because the within-patch transmission term for λ_B increases in magnitude (Eqn. 3), and suggests that the system may be even more sensitive to increasing levels of between-patch transmission when $(1\text{-}z)\beta_{BB} > (1\text{-}z)\beta_{PP}$. Estimates for the daily biting rate of *I. uriae* on breeding and pre-breeding birds from the literature (Barton *et al.* 1995; Finney *et al.* 1999; Nunn *et al.* 2006b) suggest that a higher within-patch breeder transmission rate may be more realistic.

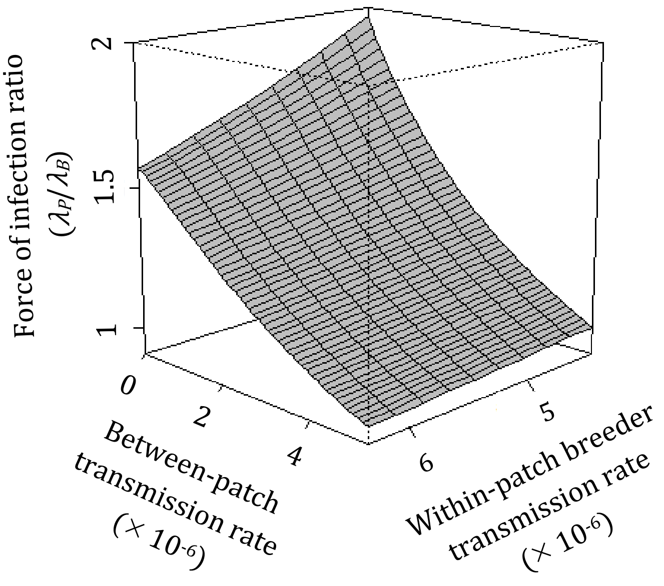

Fig. 3 Force of infection ratio (λ_P/λ_B; Eqn. 3) with varying between-patch transmission rate ($z\beta_{BP} = z\beta_{PB}$) and within-patch breeder transmission rate, $(1\text{-}z)\beta_{BB}$ ($(1\text{-}z)\beta_{PP}$ is fixed at 5.2×10^{-6} per day). All rates given are per day.

Discussion

This model provides evidence that the dynamics of seabird parasites such as GIV can be highly sensitive to changes in the socio-spatial structure, and more specifically in the rate of transmission between different patches, of a seabird population. This is important for the Isle of May guillemot colony because of recent changes in breeder abundance and success (Ashbrook *et* *al.* 2010; JNCC 2014). Changes such as these could increase the number of unoccupied breeding sites available, making it easier for pre-breeding birds to enter breeding areas and hence increasing the between-patch transmission rate. Resulting increases in the GIV force of infection predicted by this model, particularly in the breeding area, could have local consequences for the colony, as well as global consequences for the circulation of GIV and other seabird parasites.

Model limitations

Several simplifying assumptions were made within my model, to approximate the dynamics of this system. For example, pre-breeding birds were assumed to be less than six years old. However, one year-old birds are rarely seen at the colony (Halley 1992) and should therefore not be included in future models as they do not have the potential to transmit GIV. The recruitment rate (Crespin *et al.* 2006) and natural death rate (Reynolds *et al.* 2009) of pre-breeding birds have also been shown to vary with age. Although daily dynamics are appropriate for infection, due to the relatively short assumed infectious period of GIV, guillemot demographic process such as birth and recruitment are seasonal. One alternative would be to run the model for one season at a time, then to reset the population numbers, before running for another season. In this way, these demographic processes could be encompassed in the initial conditions of the model. Another alternative would be to use a discrete time modelling approach.

Although I parameterised this model using average estimates from the existing literature in order to give easily-interpretable predictions, future studies could investigate ranges of parameter estimates and their finer-scale effects on these model predictions. However, these are likely to have a greater effect on the transient dynamics of the model, rather than the long-term equilibrium behaviour presented in this study. Several epidemiological assumptions were also made within this model. For example, a latent period for GIV was not included, but other orbiviruses have been shown to have a latent period of approximately 7 days (MacLachlan *et al.*

1992; Quist *et al.* 1997). Due to the lack of GIV-specific information, estimates for the infectious period of related viruses in a different host were used. Although a viral induced death rate was included, evidence suggests that an indirect negative effect of GIV on attendance and breeding success of guillemots is more likely, given the energy needs of mounting an immune response (Bonneaud *et al.* 2003). A similar effect has also been shown in other systems (Telfer *et al.* 2002, 2005). I hope future studies will provide GIV-specific estimates for these key epidemiological parameters, to improve the accuracy of model predictions.

In order to define the transmission process, density-dependence was assumed. Although vector-borne transmission is considered to largely depend on the number of vectors biting each host per unit time rather than host density (Dye & Williams 1995), this is for a well-dispersed vector and some evidence suggests that *I. uriae* has a limited dispersal ability (Karpovich 1970; Boulinier 1995; McCoy *et al.* 2003) and all developmental stages (apart from adult males) are parasitic. Previous studies have highlighted the differences between ticks and arthropod vectors (Randolph 1998), and the dependence of tick and tick-borne pathogen abundance on host density in particular (Ogden & Tsao 2009). Homogeneous mixing was also assumed within each patch. This is likely to occur in the pre-breeder patch because of the higher mobility of these birds (Wanelik pers. obs.). However, it is unlikely to be the case in the breeder patch, where breeding birds fly directly to and from their nest sites and rarely move more than 0.5 m from their chick or egg when at the colony (Harris pers. comm.).

Tick dynamics were not explicitly included in this model, as they were assumed not to have a substantial effect on GIV dynamics. No estimates for the prevalence of GIV in this population are available in the literature but the model suggests that virus prevalence within the guillemot population remains very low (<1%; Fig. S1). This is due to the assumed short infectious period coupled with life-long immunity of the birds to GIV. However, it is known that vectors can support endemic disease regardless of the density or state of the host (Anderson & May 1979) and Anderson suggests that the relationship between the primary host and vector is particularly important for the persistence of endemic infections like GIV (Anderson 1981). This idea is supported by a much higher GIV prevalence of 25% in both on-host and off-host female ticks on the Isle of May (Nunn *et al.* 2006b). This suggests that the behaviour of the model may be more realistic if tick dynamics were included. However, the equilibrium values from this simple model appear to be good approximations of reality in terms of the risk of GIV infection within the guillemot colony (Nunn *et al.* 2006b).

Conclusion

My simple model offers a useful representation of the guillemot colony on the Isle of May. It highlights the importance of avian colonial social structure, and more specifically the rate of transmission between different patches, for understanding current, and predicting future parasite dynamics in seabird colonies and other colonial systems.

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Supplementary material

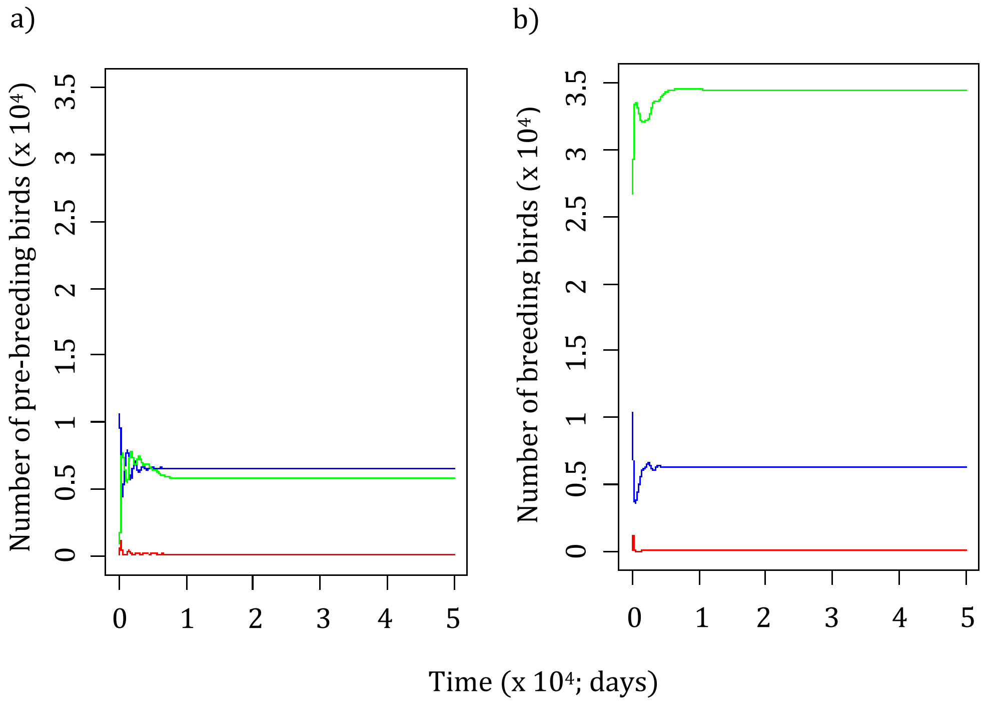

Fig S1. Dynamics of the model when perturbed from equilibrium by 10% for a) the pre-breeder, and b) the breeder patch (blue = susceptible birds, red = infectious birds, and green = recovered birds).

How far is a seabird tick able to move in search of a host? The first description of the movement of host-seeking *Ixode uriae* (White, 1852) in an artificial arena environment

Abstract

The seabird tick *Ixodes uriae* is a species of particular interest in both the phylogeographic and seabird literature due to its preference for spatially structured, seabird hosts. However, no study has directly quantified its mobility. Here, I investigate the movement of host-seeking *I. uriae* in an artificially lighted arena and in the absence of any experimental stimuli, using nymphal ($n = 24$) and adult female ($n = 24$) ticks collected from a seabird colony in southeast Scotland. Adult females moved at a rate (straight line distance from start) of 5.3 (4.1-6.9) cm min^{-1} on a horizontal surface. Nymphs moved at a significantly slower rate of 1.6 (1.2-2.0) cm min^{-1}. Movement pathways of *I. uriae* were poorly described by a correlated random walk. This information provides a firmer indication of the host-seeking potential of *I. uriae* and serves as a first step to quantifying the natural host-seeking behaviour of *I. uriae* in the field, an important parameter for understanding the risk of tick-borne pathogen transmission within a seabird colony.

Introduction

The seabird tick *Ixodes uriae* (White, 1852) has a global circumpolar distribution and infects a wide range of seabird species, including auks and gulls in the northern hemisphere, and penguins and albatrosses in the southern hemisphere (Dietrich, Gómez-Díaz & McCoy 2011). Intense infestation by *I. uriae* has been associated with negative effects on some host species (Wanless, Barton & Harris 1997) but not others (Gauthier-Clerc *et al.* 2003). *I. uriae* has a three-host life cycle and transmits a range of pathogenic agents including the bacterium *Borrelia garinii,* a causative agent of Lyme disease (Smith *et al.* 2006) and a number of RNA virus genera including orbiviruses, nairoviruses and bunyaviruses (Spence, Harrap & Nuttall 1985; Major *et al.* 2009). Tick reproduction depends on finding and attaching to a host, feeding, then detaching and moving to a suitable microhabitat (e.g. damp crevice for *I. uriae*) in which to moult, mate or lay eggs. These behaviours require a tick to move towards, on and then away from the host. The range of movement during the tick life cycle depends on the environment and host finding strategy of the species. *I. uriae* is nidiculous (nest-dwelling) and therefore should not have to move far to encounter a suitable host. Anecdotally, *I. uriae* is considered to have limited mobility (30-40 cm from resting crevice, Karpovich 1970).

Several studies have tried to quantify the movement rates (Brouwers & Newton 2009) and movement patterns (Jones 1977; Kareiva & Shigesada 1983; Brouwers & Newton 2010) of terrestrial invertebrates. Although some work has been done to infer the mobility of *I. uriae* from the spatial genetic structure of the tick (McCoy, Tirard & Michalakis 2003) and observations of the local dynamics of infestation (Karpovich 1970; Boulinier 1995) and host preference (Muzaffar & Jones 2007), I know of no study that has directly quantified movement, or more specifically the mobility of host-seeking *I. uriae.* Previous studies have shown that other species of tick exhibit host orientation in response to a variety of stimuli, including chemical cues (McMahon & Guerin 2000; Benoit *et al.* 2008; Bunnell *et al.* 2011), heat (Oorebeek, Sharrad & Kleindorfer 2009) and carbon dioxide (McMahon & Guerin 2002). *I. uriae* may exhibit similar responses, via olfactory and thermoreceptors located in the Haller's organ, located on each tarsus (Sonenshine 1991) but this has not been tested. Although eyeless, *I. uriae* may also have a limited ability to sense light through its photoreceptors (Binnington 1972).

The overall lack of information is surprising given the interest in both the impact on seabirds of *I. uriae* (Boulinier & Danchin 1996; Wanless *et al.* 1997; Gauthier-Clerc *et al.* 2003; Mangin *et al.* 2003) and of the pathogens transmitted by the tick (Nunn *et al.* 2006; Chambert *et al.* 2012), as well as the extensive research that has taken place into the contemporary and historical patterns of dispersal and diversification of *I. uriae*, including host-specific races (McCoy *et al.* 2005; Gomez-Diaz *et al.* 2012; Dietrich *et al.* 2014).

The aim of this work is not to provide a comprehensive description of the host-seeking behaviour of *I. uriae* but to present the first description of host-seeking movement, and to bring us closer to answering the question: how far is *I. uriae* is able to move in search of a host? These questions arose as part of an ongoing study investigating the impact of interacting age-related processes (age-related acquired immunity and age-related spatial population structure) on tick-borne pathogen transmission in a common guillemot (*Uria aalge*) colony.

Methods

Samples

24 adult female and 24 nymphal *I. uriae* were collected on the Isle of May, Scotland just before and after the period of peak tick feeding (Barton *et al.* 1996), on the 25th-27th March 2014 and 18th July 2013 respectively, to minimise disturbance to breeding seabirds. Adult female and nymphal ticks were collected from different areas of the colony (Fig. 1). Ticks were stored in 1.5 ml tubes containing moist tissue paper, first in the fridge (at approximately 5 °C) and then in the dark at room temperature (20 °C) once transferred to the laboratory, before first use. No tick was used in any of the experiments more than four weeks after collection.

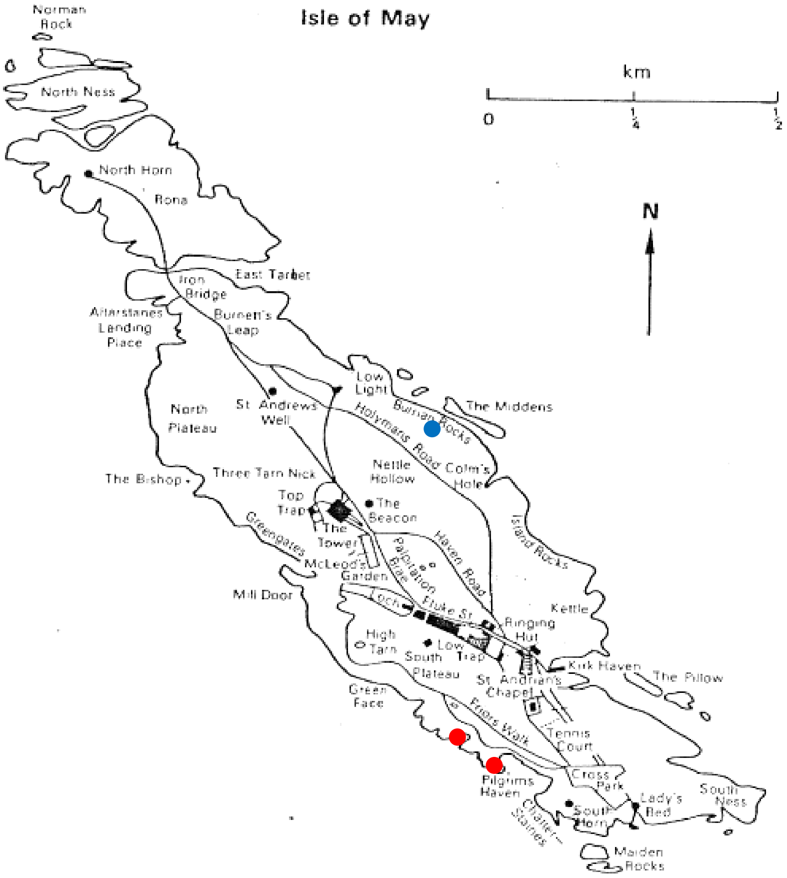

Fig. 1 A map of the Isle of May, Scotland showing the approximate areas from which adult female (red) and nymphal ticks (blue) individuals were collected.

Arena and test conditions

Each tick was tested on its own in an arena, with a single A1 piece of paper as the floor and 30 cm high walls of white cardboard. The same arena, in the same location was used for both nymphal and adult female ticks but was enlarged slightly for the latter (nymph: 100 cm × 75 cm, adult female: 100 cm × 87 cm). The arena was lit from above by fluorescent lights (T5 digital ballast system with 14 W/84 HE fluorescent tubes in banks of four; distance from arena floor = 186.5 cm; light intensity on arena floor = 460–590 lx). To avoid the build-up of kairomones (specific to the movement pathway taken by each tick) the single sheet of paper lining the arena floor was changed each time a tick was introduced into the arena. One minute before use, individual ticks were placed in the middle of the test arena and allowed to roam freely until recording began. Ticks were manipulated between experimental trials using a fine paintbrush. Disposable gloves were worn throughout and changed after testing each tick to prevent odours from the observer's skin transferring to the arena. Between measures, the observer moved 20 feet away from the arena to minimise the effect of their presence (i.e. potential increase in air temperature, CO_2 concentration and/or visual cues). Between tests, ticks were returned to individual eppendorf tubes that contained moist tissue, in order to rehydrate. All experiments were undertaken in a laboratory at the Centre for Ecology & Hydrology, Wallingford, at constant temperature and humidity (20 °C and 70% relative humidity). All experiments were also conducted between 10:00 and 17:35, and the exact time of day that each experiment was performed was recorded.

Description of horizontal movement

I used the methodology described in Kareiva & Shigesada (1983) to approximate the host-seeking movement of ticks (nymph: n = 24; adult female: n = 24) as a series of straight-line moves of given length, separated by discrete turning angles. At the start of each experiment and at each subsequent time interval, the position of the tick was marked with pencil on the sheet of paper lining the arena. The straight-line length of each move (l) was then measured, as was the angle (measured clockwise) turned by the tick at the end of each move (θ) and the total displacement after n consecutive moves (R_n; Fig. 2a). I define the total distance moved as the sum of all move lengths, and the total displacement as the straight-line distance between the starting and finishing point. Each tick was tested sequentially two or three times. These experiments were undertaken between 19th July-8th August 2013 for nymphs and 4th-11th April 2014 for adult females.

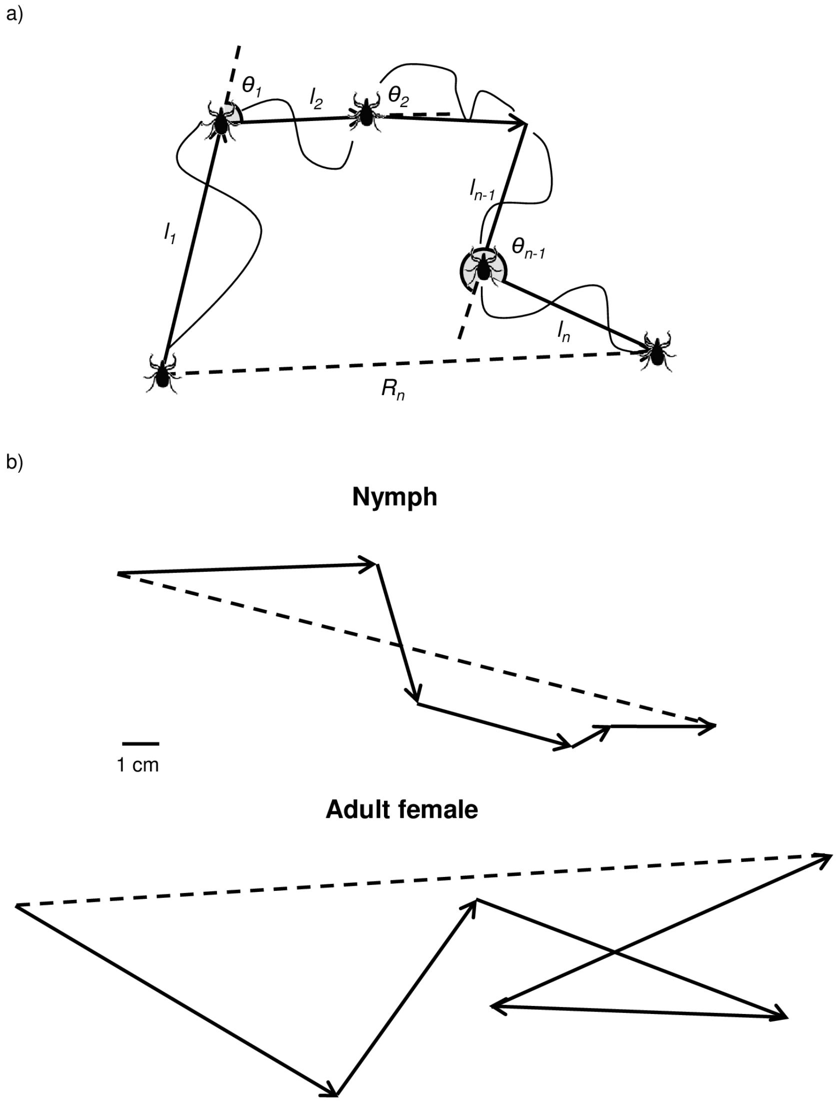

Fig. 2 a) Movement parameters measured (l = straight-line length of each move, θ = clockwise turning angle, R_n = displacement after n consecutive moves; adapted from Kareiva & Shigesada 1983), b) Examples of resulting movement pathways for nymphal and adult female ticks, in a horizontal, homogeneous environment.

Preliminary experiments were conducted both with nymphs (n = 4) and adult females (n = 5) to gauge the total time and the time interval at which to record movement. Both the total and interval times were different for nymphs and adults females due to their different size and hence movement rate. A time interval of 2 min and a total time of 10 min were selected to record nymphal movements, and a time interval of 1 min and total time of 5 min were selected to record adult female movements. These periods were chosen to ensure ticks stayed within the arena during the period of observation and to provide an informative characterisation of their movement (Kareiva & Shigesada 1983). During these preliminary experiments, the relative frequency with which ticks paused and adopted a typical questing posture with forelegs raised, was also noted.

Mean distances moved are reported in the text with 95% confidence intervals in brackets. Total displacements were converted into displacement rates (per min) to allow nymphal and adult female movements to be compared. Data on the movement of nymphs were initially limited (number of observations, $n = 18$). However, no significant difference was found between these data and other data collected for a parallel study (not presented here) on the horizontal movements of nymphs in the presence of a 12 cm (height) x 2.5 cm (diameter) pot, covered in black cardboard ($n = 36$; GLMM for displacement rate: estimates = 1.5 (1.0-2.1) cm min^{-1} and 1.6 (1.2-2.2) cm min^{-1} respectively, $p = 0.60$), so these two datasets were combined to increase the sample size for this study.

Analysis of horizontal displacement rate

Analysis of the displacement rate of *I. uriae* was performed using the R statistical software 3.1.0. (R Core Team 2013). A generalised linear mixed model (GLMMs) was implemented using the lme4 package (Bates *et al.* 2014). The dependent variable was zero-bounded and continuous, and a gamma error family (with log link function) was found to fit it best, based on the Akaike Information Criterion corrected for small sample sizes (AICc; Hurvich & Tsai 1989) and inspection of residuals.

A full submodel set was generated from a global model including all of the fixed, random and interaction terms of interest using the MuMIn package (Bartoń 2014) and is presented in the text.

Submodels were ranked on relative fit using AICc, with the best model having the lowest AICc. The residuals of the global and best model were checked for normality and homoscedasticity, and for evidence of any unduly influential data points. Parameter estimates were extracted from the best model and are reported with 95% confidence intervals, which are based on fixed effects uncertainty only. The significance of parameters in the best model was evaluated using Wald Z-tests. The R^2 of the observed versus predicted values (R^2_{COR}) for the best model is also reported as a measure of absolute model fit (Cameron & Windmeijer 1996). Model averaging was not performed as it did not change my conclusions and ensured ease of interpretation.

The fixed explanatory variables used in the analyses included life history stage, time of day and sequential order in which tick was tested. The latter was treated as a continuous variable, and included to account for possible fatigue as a result of handling and exposure to experimental conditions. Times of day were changed into decimal values. An interaction between life history stage and sequential order of testing was included in the global model. A random term of individual tick was also included to account for repeated observations.

Analysis of horizontal movement pathway

I use the formula derived by Kareiva & Shigesada (1983) to calculate the expected mean squared displacements (R^2_n) of an animal, assuming a correlated random walk, and compare these to the observed mean squared displacements.

Results

Horizontal movement rate

In a horizontal, homogenous, lighted environment, the mean total distance moved by nymphs in the 10 min observation period was 31.9 (25.6-38.1) cm, and for adult females in the 5 min observation period was 47.6 (41.9-53.3) cm (e.g. Fig. 2b). The mean total displacement of nymphs in 10 min was 18.6 (14.1-23.2) cm and for adult females in 5 min was 29.4 (24.9, 33.9) cm (e.g. Fig. 2b; Fig. 3).

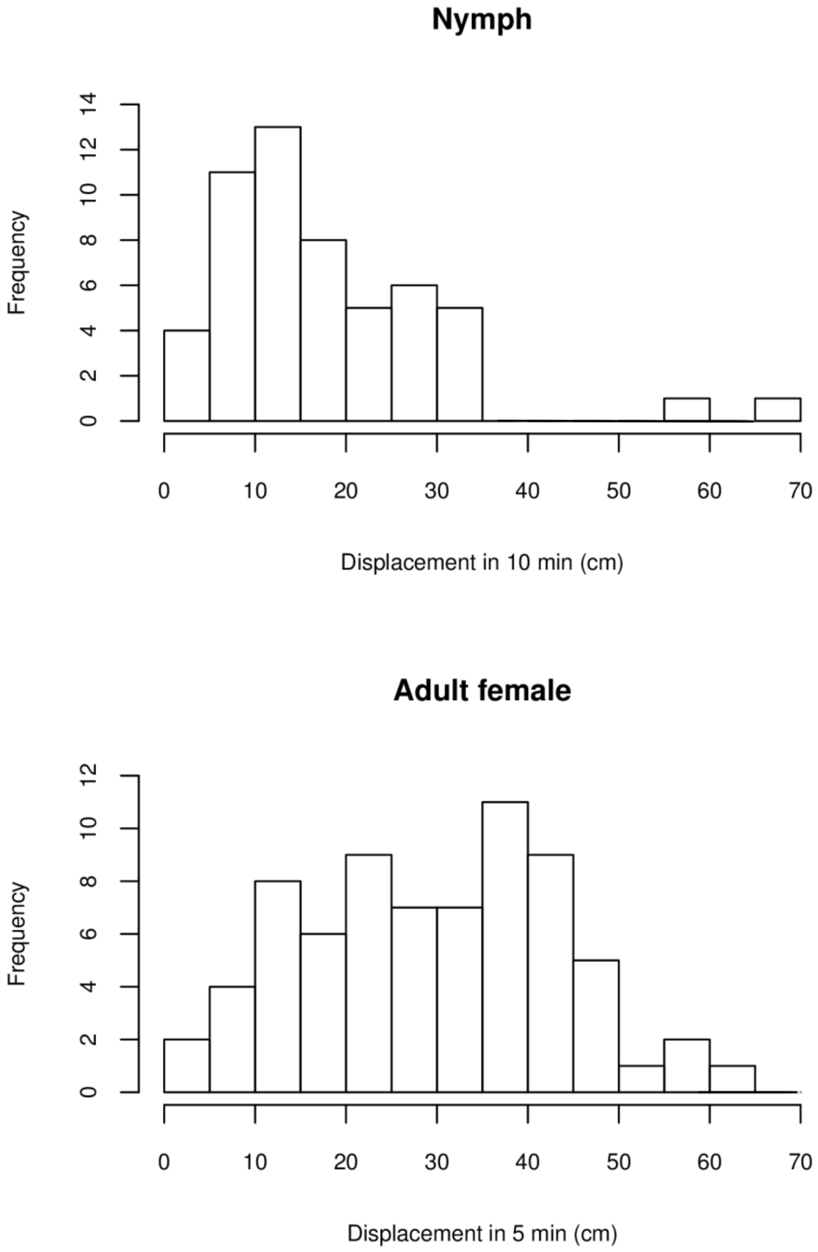

Fig. 3 Horizontal total displacements of nymphal and adult female ticks (*n* = 126).

The best model for horizontal displacement rate included life history stage only (Table 1) and explained a large proportion of the variation (R^2_{COR}= 0.73). No effect of time of day or sequential order of testing was found as neither appeared in the best model (Table 1). As expected given the difference in size, adult female ticks were found to exhibit a significantly faster horizontal displacement rate (estimate = 5.3 (4.1-6.9) cm min^{-1}) than nymphal ticks (estimate = 1.6 (1.2-2.0) cm min^{-1}, $p < 0.001$; Fig. 4).

Table 1 Full submodel set estimating horizontal displacement rate of *I. uriae.* All models include a random term of individual tick. Interactions are represented by an asterisk (*). Models are ranked on relative fit using the Akaike Information Criterion corrected for small sample sizes (AICc; Hurvich & Tsai 1989). The best model is shown in bold.

Model	Log-likelihood	d.f.	AICc
Life history stage	**-239.22**	**4**	**486.8**
Life history stage, Sequential order of testing	-238.77	5	488.0
Life history stage, Time of day	-239.01	5	488.5
Life history stage*Sequential order of testing	-238.39	6	489.5
Life history stage, Sequential order of testing, Time of day	-238.50	6	489.7
Life history stage*Sequential order of testing, Time	-238. 16	7	491.3
Sequential order of testing	-251.83	4	512.0
-	-253.30	3	512.8
Time of day, Sequential order of testing	-251.80	5	514.1
Time of day	-253.30	4	514.9

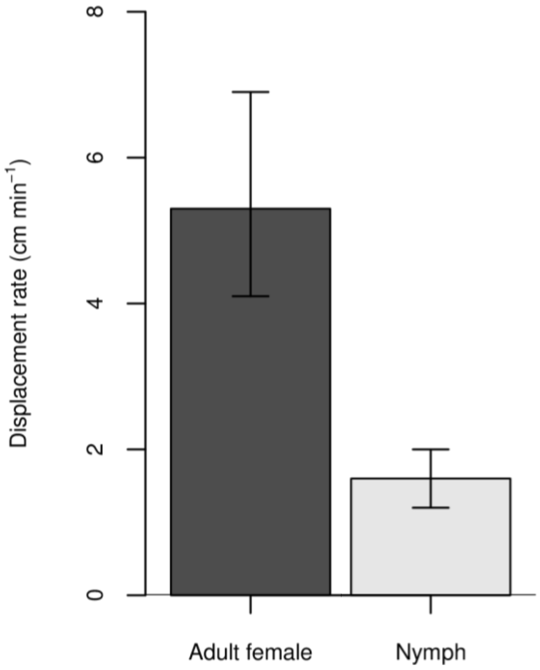

Fig. 4 Model estimated displacement rates on horizontal surface as described in the text. Error bars indicate 95% confidence intervals (*n* = 126).

Horizontal movement pathway

The distribution of turning angles for both nymphal and adult female ticks was approximately symmetric about 0 ° (Fig. 5a), fulfilling the simplifying assumption for the formula proposed by Kareiva & Shigesada (1983) and applied here. The mean squared displacements of both nymphal and adult female ticks were consistently underestimated by the expectations of a correlated random walk (by an average of, nymph: 31%, adult female: 29%; Fig. 5b).

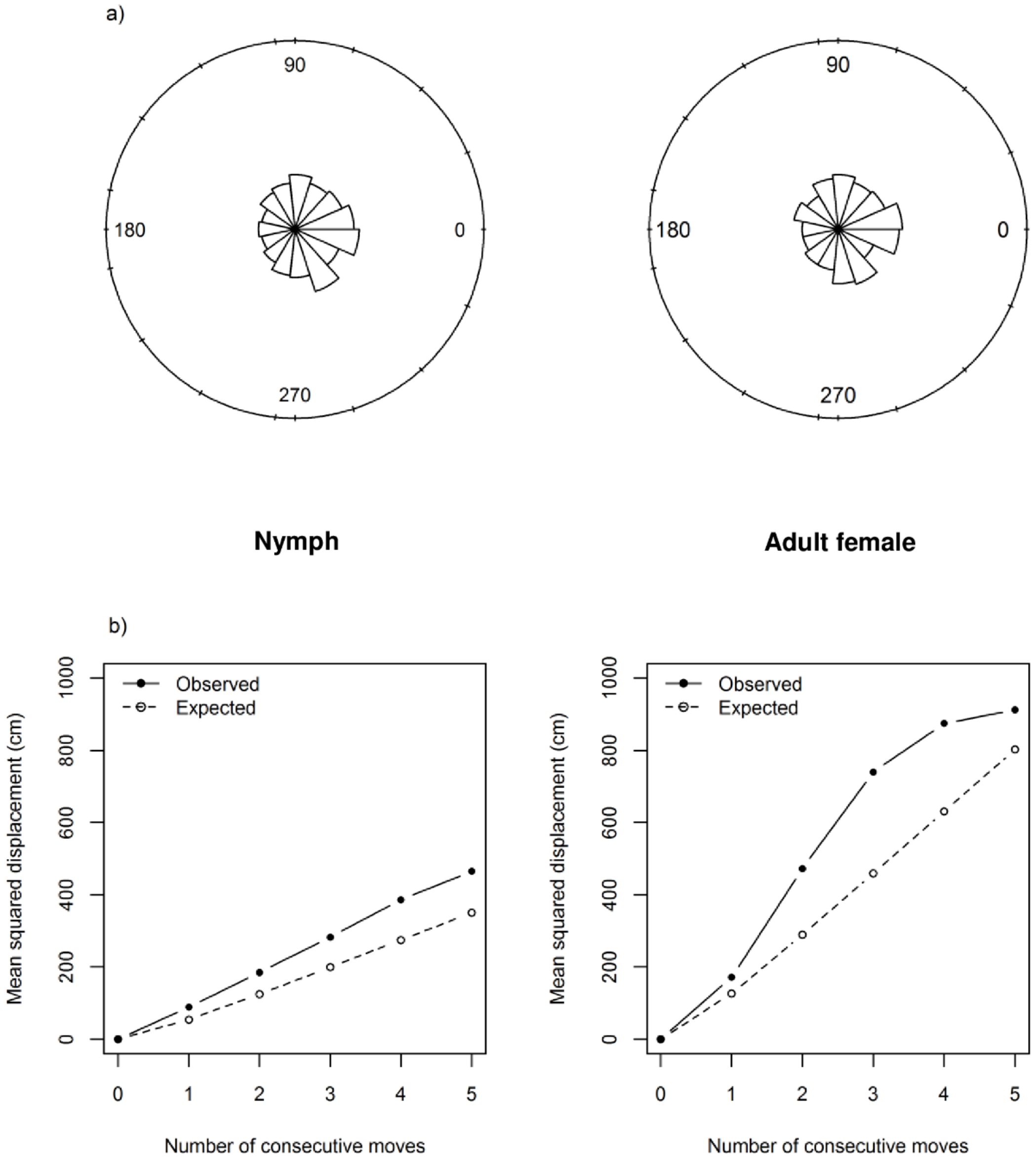

Fig. 5 a) Angles (in degrees) turned by *I. uriae*, b) Relationship between the mean squared displacement and the number of consecutive moves made by *I. uriae*. Observed mean squared displacements and expected mean squared displacements, assuming a correlated random walk, are shown (according to Kareiva & Shigesada, 1983).

Discussion

This is the first study to systematically investigate the walking rate and directional movement of *I. uriae*. *I. uriae* is anecdotally considered to have limited mobility (Karpovich 1970), but my findings suggest that adult *I. uriae* has the potential to walk these anecdotal distances in just a few minutes (30 cm from its starting point in just 5 min), suggesting the potential for a higher degree of mobility. However, the ticks in my experiments were moving on featureless, flat surfaces in the absence of any experimental stimuli. It may be that *I. uriae* is less mobile in its natural habitat (Wiens, Schooley & Weeks 1997; Zollner & Lima 1999; Lane, Mun & Stubbs 2009) where the micro-topography of the cliff may restrict movement, and the build-up of kairomones may increase the turning angles and frequency of turns made by host-seeking ticks (Osterkamp *et al.* 1999).

Correlated random walks have been successfully used to describe the movement patterns of animals (Turchin 1998). My findings suggest that the movement pathways of *I. uriae* in the absence of any experimental stimuli, are not well described by a correlated random walk. However, this is likely to be due to ticks altering their natural movement pathways in the artificial arena environment without any host stimuli (Wiens *et al.* 1997; Zollner & Lima 1999). Future studies should explore the movement pathways of *I. uriae* in the field or in a semi-natural environment and in the presence of specific host stimuli such as CO_2 and heat.

My findings suggest significant differences between the movements of nymphal and adult female ticks, only some of which were expected. Adult female ticks were found to move at a significantly faster rate than nymphal ticks, as expected given their larger body size and resulting stride length (assuming a constant stride frequency). However, I also expected both nymphs and adult females to regularly adopt the questing posture. Nymphs behaved as expected, but from casual observation, adult females rarely adopted the questing posture. Since the adult female and nymphal ticks in this study were collected at different times relative to their peak of feeding, one possibility is that they were in different physiological condition and/or more or less predisposed to host-seeking. Nymphs were collected a couple of weeks after their second feeding peak around 6 July, whereas adult females were collected two months before their single feeding peak around

1 June (Barton *et al.* 1996). Ticks are likely to alter their search strategy according to their condition (Zollner & Lima 1999). For example, the search strategy of a tick trying to find shelter will be very different from that of a tick actively seeking a host. However, previous studies suggested differences in host-seeking strategy between different stages of *I. uriae* and different hosts (Muzaffar & Jones 2007). For example, nymphs have been found to be more numerous on breeding guillemots, and adult females on immature guillemots (Nunn *et al.* 2006). Nevertheless, I believe that future studies, both in the laboratory and in the field, should carefully consider tick condition, in order to disentangle these effects.

Host-seeking behaviour of ticks has been shown to be under circadian control. My measurements of *I. uriae* were taken in lighted conditions and in daylight hours, and I believe that this is of biological interest. Although Eveleigh & Threlfall (1975) showed that *I. uriae* most often attaches to and detaches from its host at night, many guillemot colonies for example, are found inside the Arctic Circle making it inevitable that some host-seeking activity occurs during daylight hours. On the Isle of May, peak tick feeding coincides with incubation of eggs by guillemots, when the breeding colony is approximately half occupied during daylight hours, offering opportunity for this activity (Barton *et al.* 1996). Finally, immature guillemots are only found on land during the day but nevertheless suffer from moderate levels of tick infestation (Nunn *et al.* 2006). I found no significant evidence for an effect of time of day. However, it is possible that the natural flicker frequency of the fluorescent lights used and/or the storage of ticks in continuous dark prior to observation may have altered the natural movement of ticks, as expected in daylight hours.

Ticks were tested sequentially which may have increased the risk of fatigue resulting from handling, but I found no significant evidence for this. The presence of an observer may also have had an effect on tick movement (i.e. potential increase in air temperature, CO_2 concentration and/or visual cues) but this was a systematic bias which was minimised as far as possible, with no evidence for directional bias from casual observation. Finally, ticks were exposed to room temperature (20 °C) just prior to and during experiments. This temperature is high relative to the mean temperature of *I. uriae* biotopes, and may have affected the movement of *I. uriae*. However, this was done in order to ensure a constant environment and to avoid rapid changes in temperature. Ticks were also stored in the fridge as long as possible and returned to individual eppendorf tubes that contained moist tissue between tests, in order to rehydrate.

Conclusion

Regardless of the limitations, I believe that this work provides a firmer indication of the host-seeking potential of *I. uriae,* and serves as a first step to quantifying the host-seeking behaviour of *I. uriae* in the field, an important parameter for understanding the risk of tick-borne pathogen transmission within a seabird colony.

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Do immature common guillemots (*Uria aalge*) pose an infection risk to the breeding colony? Estimating the current rate of immature-mediated parasite dispersal from field observations

Abstract

1. Host age is an important factor affecting parasite dynamics. As compared to older individuals, younger individuals can be more susceptible to infection due to their waning maternally derived immunity or developing immune system, and their behaviour can make them responsible for a large proportion of transmission events.
2. Colonially-breeding common guillemots (*Uria aalge*) are parasitised by a tick-borne, multi-strain virus, *Great Island virus* (GIV). Ticks feeding on immature guillemots have been shown to be more frequently infected likely due to their host's developing immune system. Previous observations also suggest that few ticks move from immature to breeding areas of the colony, limiting the spread of infection to susceptible breeding guillemots, for which infection is likely to be more costly. However, recent declines in breeding abundance and success in colonies bordering the North Sea may have facilitated this movement, by altering the age-related spatial structure of the colony.
3. To provide the first quantitative estimate of the movement of ticks from immature to breeding areas, I assumed that immature-mediated dispersal was the major route for this movement and recorded the attendance rates, and spatial attendance patterns of 67 known-aged immature guillemots across four different areas of a large breeding colony, during peak tick feeding and over one season.
4. I showed that immature birds spend a limited proportion of time ashore during daylight, which increases significantly as the season progresses and varies between individuals.
5. This pattern is the same when considering how often these immatures entered breeding areas when ashore: generally infrequently but significantly increasing through the season and

varying between individuals. Subcolony differences were also found, with immatures in one subcolony spending a limited amount of time in and being limited to the periphery of breeding areas whereas in two other subcolonies, immatures spend more time in breeding areas and in one of these subcolonies, birds attended more central parts of breeding areas.

6. I conclude that immature individuals have the potential to play an important role in shaping wildlife parasite dynamics, and exposure of breeding birds to infection is likely to be spatiotemporally heterogeneous within the colony. This could have consequences for breeding success and warns against drawing general conclusions about infection risk across whole populations.

Introduction

Previous studies have explored the broader-scale processes regulating the transmission of endemic parasites (Swinton *et al.* 1998; Smith, Dushoff & McKenzie 2004; Rogers & Randolph 2006; Watts *et al.* 2009; Gilbert 2010) with the aim of predicting their future dynamics. However, individual heterogeneities affecting parasite dynamics are less well studied. For example, individuals can vary in their susceptibility to infection (Staszewski *et al.* 2007a; b; Beldomenico *et al.* 2009). In some cases, if a small proportion of individuals are susceptible within a group, transmission rates can be driven down by herd immunity, the resistance of a group to infection because of the immunity of a large proportion (or the susceptibility of a small proportion) of its members (Fox *et al.* 1971). However, transmission rates can rapidly rise if the number of naïve susceptible hosts present in a population increases (Begon *et al.* 2009).

The number of susceptible hosts within a population can vary spatiotemporally. For example, an influx of susceptible juvenile field voles has been linked to the high prevalence of cowpox virus during the breeding season (Burthe *et al.* 2006), and the hatching period for susceptible young red grouse is associated with the period of high abundance of questing *I.ricinus* ticks, and a significant rise in R_0 for louping-ill virus (Hudson 1986; Hudson *et al.* 1995). On a spatial scale, there is evidence for varying prevalence of Buggy Creek virus between cliff-swallow colonies, with varying population and hence susceptible host numbers (Brown *et al.* 2001). Individual hosts can also vary in their transmission propensity as a result of their behaviour, with some individuals known as 'super-spreaders' (Lloyd-Smith *et al.* 2005) being responsible for the majority of transmission events and hence driving parasite dynamics (VanderWaal *et al.* 2013, 2014). There is also widespread evidence for changes in behaviour of animals once they have been parasitised (Poulin 1995).

Host age can be an important factor driving this individual heterogeneities and hence affecting parasite dynamics. Younger individuals are often more susceptible to infection due to waning maternally derived immunity (Long *et al.* 2010) or their developing immune systems (Anderson & Trewhella 1985; Nielsen *et al.* 2002), and can be responsible for a large proportion of transmission events due to their higher levels of social interaction and/or mobility as compared to older individuals (Grenfell & Anderson 1985; Long *et al.* 2010), although this is not always the case (Burthe *et al.* 2006; Hamede, Mccallum & Jones 2008). Fitness impacts of infection have also been shown to dependent on age (Telfer *et al.* 2005; Härkönen *et al.* 2007). Age-dependent transmission rates and age-structured behaviour were originally shown to be important for human diseases such as measles (Anderson & May 1984; Grenfell & Anderson 1985). Some studies have also looked at age-dependent differences in wildlife parasite transmission (Härkönen *et al.* 2007; Klepac *et al.* 2009; Long *et al.* 2010) but the complex interactions between multiple such differences have rarely been explored. This knowledge of the heterogeneities within a wildlife population is necessary to accurately quantify key epidemiological parameters such as the transmission rate, and to predict future parasite dynamics.

Many seabirds breed colonially (Perrins & Birkhead 1983) and parasite transmission has been suggested as a potential cost of coloniality (Brown & Brown 1986). The Common guillemot (*Uria* *aalge;* hereafter guillemots) breeds in large, dense colonies usually on cliffs and is parasitised by the seabird tick (*Ixodes uriae)* and the parasites it transmits (Olsen *et al.* 1993; Nunn *et al.* 2006a; Duneau *et al.* 2008; Major *et al.* 2009; Duron, Jourdain & McCoy 2014). Among these parasites is the orbivirus *Great Island virus* (GIV; Attoui *et al.* 2012). The guillemot-tick-virus system is a useful model for exploring the effects of interacting age-related processes on wildlife parasite dynamics because: a) there is an age-related spatial structure within guillemot colonies; with adults breeding on cliff ledges, while prospecting immature birds (typically aged 2 – 6 years) congregate around the edges of the breeding colony (Halley 1992; Fig. 2), and b) there is evidence for age-related acquired immunity to GIV by guillemots, with a higher proportion of immune breeding birds compared to immature birds (Nunn *et al.* 2006b), leading to a significantly lower prevalence of infection among ticks which infest breeding birds (Nunn *et al.* 2006b).

However, an epidemiological model of the colony was shown to be highly sensitive to the mixing rate between ticks derived from immature and breeding birds, with a doubling of the mixing rate (within a biologically reasonable range) leading to the force of infection more than doubling in the breeding area (Chapter 2). This suggests that the impact of herd immunity on the GIV transmission rate is highly dependent on the mixing rate between ticks derived from immature and breeding birds. This model assumed direct, density dependent transmission, with susceptible-infected-recovered dynamics and two patches (one for breeding birds and one for immature birds). The model was parameterised as much as possible from the literature (demographic parameters: Harris & Wanless 1988; Reynolds *et al.* 2009; Harris & Wanless 1995; Crespin *et al.* 2006; epidemiological parameters: Nunn *et al.* 2006b; MacLachlan *et al.* 1992; Koumbati *et al.* 1999) but it was not possible to accurately estimate this key tick mixing rate parameter.

Transmission of GIV infection from prospecting immature birds to breeding birds can only occur via the vector, *I. uriae*. It is a three-host hard tick, with each developmental stage (larva, nymph and adult) feeding on a host for 4-12 days (Murray & Vestjens 1967; Eveleigh & Threlfall

1975; Barton, Harris & Wanless 1995; Finney, Wanless & Elston 1999a) and then retreating into cracks in the cliff face to moult to the next stage or, for an adult, to mate and lay eggs (female only). Previous studies suggest that *I. uriae* has a relatively limited ability to disperse independently (Karpovich 1970). However, evidence for long-distance host-mediated dispersal of ticks and associated parasites exists for birds (Danchin 1992; Olsen, Jaenson & Bergstrom 1995; Dietrich, Gómez-Díaz & McCoy 2011; Gomez-Diaz *et al.* 2012) and for shorter-distance host-mediated dispersal in other systems (Smith *et al.* 1996; Brown & Brown 2004; Brown *et al.* 2007, 2008; Watts *et al.* 2009; Jones *et al.* 2011).

Furthermore, field observations show that immature guillemots repeatedly attempt to enter breeding areas and fly between subcolonies (Halley, Harris & Wanless 1995). Previous studies suggest that as immature birds get older and prepare to recruit, they return to the colony earlier in the season, visit more frequently and spend longer on land attending the colony (Halley *et al.* 1995). Since ticks can only get onto a guillemot when it is on land, the risk of tick attachment is likely to be proportional to the time spent on land. I use subcolony attendance rates to estimate the proportion of time spent ashore by immature birds. I expect older immature birds to be the major route for moving ticks from marginal immature to breeding sites, with the caveat that they are also more likely to have acquired immunity to GIV than younger immatures.

It follows that understanding the movement patterns of, both younger and older, immature birds attending breeding colonies is vital to gauge the risk of exposure of breeding birds and chicks to parasites. Although immunity to GIV is more common among breeding birds, GIV is a multi-strain virus with evidence of only limited antibody cross neutralisation (Nunn *et al.* 2006b). This means that not all individuals acquire immunity to infection to all strains by the time they recruit to the breeding population. Furthermore, the cost of being newly infected is likely to be much higher for an adult than for an immature, given the already high energetic cost of breeding (Bonneaud *et al.* 2003). There are many examples within the domestic animal literature of infection with a parasite impacting breeding (Muneer, Chaudhry & Khawaja 2000; Yisa, Tsadu & Musa 2010; Haileselassie *et al.* 2011). In the bank vole and wood mouse, infection with cowpox virus has also been shown to delay maturation and therefore reproduction (Telfer *et al.* 2002, 2005).

There is observational evidence for aggression towards and effective ejection of immature prospecting birds in the breeding colony (Birkhead 1978). If this is the case, I would expect the exposure of breeding birds to parasites carried by immature birds to be limited, suggesting a significant regulatory effect on parasite transmission. However, since 2000, guillemot populations bordering the North Sea have experienced declines in breeding success and abundance (JNCC 2014). This could have increased the number of unoccupied sites available and made it easier for immature birds to enter breeding areas. Furthermore, acute decreases in breeder attendance were observed in 2007 and 2008, likely due to limitations on food (Ashbrook *et al.* 2010). Although no unattended chicks have been recorded since (including the season in which I collected my data), extreme events such as this could further facilitate the penetration of immature prospecting birds into breeding areas.

Subcolony attendance rates and spatial attendance patterns of known-aged immature guillemots were recorded at a large breeding colony, from before egg-laying in late April until approximately midway through the chick period, in mid-June. This period is coincident with peak tick feeding, which is around 1 June for larval ticks, 18 May for adults, and 18 May for the first peak of nymphal activity (Barton 1996). These data were recorded to test three hypotheses: a) that immature birds infrequently enter, spend a limited amount of time in and are restricted to the periphery of breeding areas, b) as they prepare to recruit, older immature birds more frequently enter and spend more time within breeding areas than younger immature birds, and c) immature birds more frequently enter and spend more time within breeding areas when breeder attendance is lower. The effects of several additional factors on these data were also examined, including date, time of day, subcolony, weather and individual identity.

Field methods

Study site

Data were collected on the attendance and behaviour of immature guillemots from 25th April-12th May and 21st May-15th June 2013 at four subcolonies on the Isle of May. The Isle of May is a small island (about 1.8 km long by 0.5 km wide) located about 8 km off the coast, in the north of the outer Firth of Forth, Scotland (56.2° N, 2.6° W). It is managed by Scottish Natural Heritage as a National Nature Reserve and is currently home to a large and diverse population of seabirds, including approximately 15,000 pairs of breeding guillemots (Lahoz-Monfort *et al.* 2014).

The guillemots followed had been uniquely marked as chicks with easily read engraved colour rings. Approximately 200 guillemot chicks are ringed every year on the Isle of May, and subsequently resighted (Harris, Halley & Wanless 1992). The population has been monitored in this way since 1981. The age of a bird was calculated by subtracting the year of ringing from the current year. As expected, the resulting age distribution had a small range (3-7 years old); with two 3 year olds, 51 4 year olds, 13 5 year old, no 6 year olds and one 7 year old followed. The subcolonies (Chatterstanes stack, CS; Chatterstanes mainland, CM; Colony B, CB; Fringe, F) were all located in the southwest of the island (Fig. 1) and were of similar and relatively high breeding density (breeding birds in physical contact with 2- 4 other birds). They were chosen because they were the only areas in the colony that had sufficient numbers of ringed (known age) birds, including immature birds.

Fig. 1 A map of the Isle of May, showing the location (red) of focal subcolonies.

The approximate sizes of the focal colonies were 70 m^2 (CS, CM and CB) and 20 m^2 (F). Those subcolonies where, or close to where, chicks were ringed were chosen as focal subcolonies, since immatures tend to return to areas close to where they were ringed. Each subcolony was viewed from a fixed viewpoint between 20 and 50 m away for CS, CM, CB and F respectively. Prior to the start of data collection, areas within each subcolony were classified as breeding areas (ledges used mainly by breeding birds) and immature areas (ledges usually at the landward edge of the subcolony where there were no breeding birds and immatures were seen to aggregate; e.g. Fig. 2). Observations of breeding activity during the season indicated that these classifications were robust and no major modifications were required. To facilitate recording locations of birds at a fine spatial scale a grid was superimposed on the photograph of each subcolony (e.g. Fig. 2). Viewing distances differed between subcolonies. To account for this, for each subcolony, the average height of an individual guillemot from the viewing point was used as a scale marker so that the average size of a grid cell was approximately 50 cm^2. The average grid cell was used as, for all subcolonies, more distant grid squares were larger and closer grid squares smaller. The magnitude of these differences in relative grid cell size also varied between subcolonies, as a result of differing perspectives. Breeding and immature areas were grid cells with 'immature' or 'breeding' status according to the presence (breeding) or absence (immature) of breeding activity (i.e. an egg or chick). Grid cells with vertical cliff-faces were not assigned a status because they were too steep for birds to use (e.g. L13 in Fig. 2). Grid cells with ledges and no apparent evidence of breeding activity but consistent breeding activity completely surrounding them were assumed to have breeding status. The majority of grid cells with immature status were found on ledges used exclusively by immature birds (e.g. D8 in Fig. 2) but a few were found on the seaward margins of ledges used by breeding birds (e.g. E14 in Fig. 2).

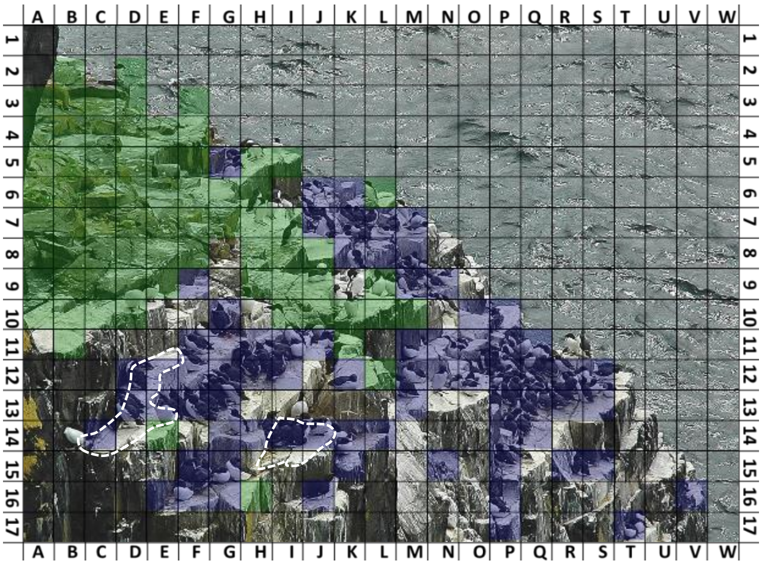

Fig. 2 Distribution of breeding (blue) and immature areas (green) in subcolony CB. Some areas were unclassified (clear) because a) they were not used by immature or breeding birds due to the steepness of cliff, or b) it was unclear whether they were used by immature or breeding birds. White dashed lines indicate subsets of the breeding area where the number of birds was counted to calculate an index of breeder attendance (please note that more distant grid squares are larger than closer grid squares due to the perspective from which this subcolony was viewed).

To account for temporal differences in guillemot attendance or activity patterns, watches were carried out in three approximately equal time periods (dawn-10:30,10:30-16:00 and16:00-dusk). Each subcolony was observed for 2 hours in a day and a stratified approach was used to ensure that samples were balanced across all subcolonies with respect to coverage of all time periods of the day. Data collection was occasionally prevented by bad weather (approximately 20% of scheduled watches). Weather variables can affect guillemot attendance and foraging activity (Finney, Wanless & Harris 1999b) so wind strength, air temperature and sea condition were recorded using five or six-point scales (Table S1). An index of breeder attendance in each subcolony was recorded by counting the number of birds present in one or two subsets of the breeding area that could be clearly delimited using physical features on the rocks (mean no. birds present ± standard error; CS: 13.10 ± 0.28, CM: 6.91 ± 0.36 & 37.5 ± 6.61, CB: 10.41 ± 0.35 & 9.48 ± 0.31; F: 4.87 ± 0.38 & 15.00 ± 3.79). Due to temporal variation in the number of birds breeding at the representative breeder plots, counts undertaken at the start of each watch from each of these breeder plot were divided by the maximum count for that plot to generate a proportional breeder attendance variable, comparable within and between subcolonies.

Within the first 15 min of each watch all colour-ringed individuals visible in the immature area or on the margins of the breeding area were identified and the first focal individual selected randomly, by numbering each of these individuals and using a random number generator. This bird was then followed using a telescope with a ×20-60 zoom lens and its location recorded using the grid cells as reference points. This was repeated for a second randomly selected individual, and again for a third etc. until all visible colour-ringed individuals had been followed or the maximum watch length of 2 hours was reached.

An instantaneous sampling approach was used to estimate the proportion of time individuals spent in breeding and immature areas (Altmann 1974). To determine appropriate sampling periods that individuals needed to be watched for, 20 ringed immatures were followed for 20 minutes in watches made between 1st and 12th May. The cumulative number of birds which moved to another grid cell (i.e. an average of 50 cm) during the watch (irrespective of whether this was in an immature or breeding area) was plotted against time (Fig. S1). All 20 birds changed grid cell at some point during the 20 min watch but 85% (*n* = 17), did so within the first 10 minutes. Accordingly 10 min was selected as an appropriate sampling period that provided a balance between sampling a large number of individuals and recording their movements.

The location of a focal bird in the grid was recorded every 15 s. A stop watch was used to time these observations. This was the minimum recording interval achievable under field conditions but still provided data at a detailed spatiotemporal scale relevant to the questions being posed (Suen & Ary 1984). Although the 15 s sampling interval exceeded the minimum time spent by an immature in a breeding area, immatures were rarely seen to enter and leave breeding areas between successive intervals, and these very brief visits were assumed not to pose a significant risk of host-mediated tick dispersal. If a focal individual was lost from sight (eg. behind a rock or behind another bird) it was recorded as out of sight but the watch was continued. However, if it flew away from the subcolony, the watch was terminated, resulting in a watch length of less than 10 min. Those watches resulting in an overall watch length of more than 5 min were included in the statistical analyses, as well as the preliminary watches of length 20 min.

Statistical methods

The aim of the statistical analyses was to explore the effects of several independent variables on: a) the proportion of time spent ashore by immature birds during daylight hours, b) the probability of an immature that was ashore entering a breeding area, and c) the subsequent time an immature spent in a breeding area. The independent fixed terms included in the analyses were: an index of breeder attendance, weather (including wind strength, air temperature and sea condition; b) & c) only, as not all this information was available for all re-sighting data);

subcolony, date, bird age and time-period during the day. Bird ID was treated as a random term with a given mean and variance. Subcolony was treated as a fixed term because only a very small proportion of all the subcolonies on the Isle of May were sampled (7% of all subcolonies) so that it was impossible to know whether subcolony means were normally distributed.

Bird age was treated as a categorical variable of either 3-4 years or 5-7 years, in order to include the few 3 and 7 year olds ($n = 3$) that were sampled. Other age categories were also tested, and bird age was treated as a continuous variable but no qualitative differences were found in the main results. Wind strength, sea condition and air temperature were summarised by a single principle component (PC1) using Principal Component Analysis (PCA) and PC1 was used in further analyses, as a continuous variable. PC1 summarised 73% of variation in the weather data. High values of PC1 were associated with higher air temperatures and calmer sea and wind conditions. This approach was used to decrease the number of independent variables to a more manageable number. Date was coded as a continuous variable: the number of days from day 0 of data collection (25th April for colony attendance data; 1st May for detailed location data). However, previous studies suggest significant phenological differences between different areas of the colony (Wanless & Harris 1988). Daylight hours were assumed to span the hours of 4am to 10pm at the time of year the study took place (Halley 1992). Time of day was treated as a factor with three levels (period 1: dawn-10:30, period 2: 10:30-16:00, period 3: 16:00-dusk). I also included interactions between subcolony and breeder attendance, and between bird age and date; and investigated polynomial terms for continuous variables. No evidence for significant multi-collinearity was found among the independent fixed terms, as tested by Pearson correlation coefficients. However, this method is best suited to continuous variables.

a) Subcolony attendance rates of immature birds

Subcolony attendance data were coded as a binary variable, i.e. whether or not an individual known to attend the subcolony at least once in the season, was seen during a given watch. A Bernoulli GLMM was used to model the effects of the independent variables on the probability of seeing an immature ashore in the subcolony, using the glmmADMB package (Skaug *et al.* 2013) in R 3.0.2 (R Core Team 2013). A logit link function gave the best model fit. Probabilities and proportions were used interchangeably according to the relative frequency interpretation of probability, assuming a large number of independent trials. Once again, the data were analysed as an instantaneous sample to estimate the proportion of time spent ashore by immature birds during daylight hours, assuming that watches were regular and uniform (Altmann 1974). Prospecting immatures do not spend the night ashore so these estimates can be readily converted to the total amount of time spent ashore each 24 hours.

Due to evidence of only occasional movement of immature birds between subcolonies, often only a few metres apart (see Results), I only included those birds that were seen at a single subcolony and assumed that they did not visit any other non-focal subcolonies during the sampling period. However, previous studies suggest some movement of 3 and 4 years old birds between subcolonies (Halley 1992) so the results from this analysis should be considered minimum estimates. By excluding birds seen at more than one subcolony (n = 18), an empty cell for 5-7 year old birds in subcolony CS emerged. Therefore, a complementary analysis was run on a subset of the data which only included subcolonies with both 3-4 and 5-7 year olds.

Model averaging

Full submodel sets were generated from a global model including all of the fixed, random and interaction terms of interest using the MuMIn package (Bartoń 2014) in R 3.0.2 (R Core Team 2013). All candidate models were evaluated and ranked on relative fit using the Akaike information criteria corrected for small sample size, AICc (Hurvich & Tsai 1989) and all those with a ΔAICc < 2 relative to the lowest rank were considered to be equally supported as the top models for explaining the data. Model fits were checked for an approximately normal distribution of residuals, homogeneity of variance and any evidence of unduly influential observations. The R^2 of the observed vs. predicted values (R^2_{COR}) for each top model is also reported as a measure of absolute model fit (Byrnes & Stachowicz 2009). Standardized effect sizes, unconditional standard errors and associated 95% confidence intervals (95% CI) were obtained by averaging across this set of top models using the zero method (Burnham & Anderson 2002). The relative importance of a variable was taken to be the sum of the Akaike weights of the top models in which it was found (Bartoń 2014). Significance was evaluated using the 95% CI; a 95% CI not including zero suggesting a significant effect. Random terms were retained during the modelling process because they were deemed to be part of the study design. However, as reported in the results, the significance of random terms was tested by comparing the AICc of the global model with and without these terms.

b) Location data of immatures

All unclassified attendance data points, where the position of a bird was unknown, were discarded prior to statistical analysis. This included occasions when a bird was seen in between breeding and immature grid cells (approximately 2% of data points) and occasions when a bird was out of sight or a bird was attending grid cells of unknown classification (approximately 5% of data points).

The data were found to be highly zero-inflated, with 72% of records being zero i.e. no movement of an immature into a breeding area. This is far more than would be expected, based on a binomial distribution. Due to this zero-inflation and the nature of the question, a two-part, hurdle model approach was used. A hurdle model deals with zero-inflated data by using a

Bernoulli distribution (a special case of the binomial distribution where $n = 1$) to model presence and absence, and a different distribution for the counts (often Poisson or negative binomial). My hurdle model consisted of: a) a Bernoulli Generalised Linear Mixed Model (GLMM) to model the probability of an immature entering breeding areas at any point during a watch (presence and absence) and, b) a beta-binomial GLMM to model the subsequent proportion of a watch spent in breeding areas. Model-averaging was used to evaluate models, as for the subcolony attendance data.

For the first part of the hurdle model, presence-absence data (no records in breeding areas = 0, one or more records in breeding areas = 1) was analysed using the glmmADMB package (Skaug *et al.* 2013) in R 3.0.2 (R Core Team 2013). A complementary log-log link function gave the best model fit. Weighting of records was not possible in this analysis so only those records with a sampling period of more than 5 minutes (i.e. more than 50% of watch completed) were included to minimise bias from particularly short, and likely inaccurate, records ($n = 30$ of 247 watches). The analysis was also run on a) the whole dataset, and b) only those records with a sampling period of 10 minutes, but no qualitative differences were found in the main results.

A Hartigan's Dip Test was used to test the raw proportional data for unimodality using the diptest package (Hartigan 1985; Hartigan & Hartigan 1985) in R 3.0.2 (R Core Team 2013). These data were then analysed using the glmmADMB package (Skaug *et al.* 2013) in R 3.0.2 (R Core Team 2013). The beta-binomial error family was chosen to account for over-dispersion in the dataset and was judged to be a good approximation of the truncated binomial distribution. This is because the mean number of records of immatures in the breeding area was relatively high ($\bar{x} = 27$) and the probability of a zero under a beta-binomial distribution with a mean of 27 is virtually nil. A probit link function gave the best model fit. As before, probabilities and proportions were used interchangeably according to the relative frequency interpretation of probability, assuming a large number of independent trials. Only subcolonies CS, CM & CB were included in this analysis as there was only one case of a bird entering the breeding area in subcolony F and this prevented models from converging.

Maps of immature birds' intensity of area usage

Maps indicating the intensity of grid cell usage were produced to explore the spatial distribution of immature birds. The image function in R 3.0.2 (R Core Team 2013) was used. A scale of colours was used to represent a) the number of records in which a representative bird was seen in a particular grid cell, to help visualise the spatial attendance data, and b) the number of watches in which any immature bird was seen in a particular grid cell of breeding status, thereby indicating the overall intensity of use within breeding areas. The resulting colour-coded image was superimposed onto the grid map of the subcolony that was used for data collection.

Results

a) Subcolony attendance rates of immature birds

A total of 430 sightings of 53 individual 3-4 year old birds and 14 individual 5-7 year old birds were obtained during 171 watches (mean number of watches per subcolony = 42.8, range = 31 - 54) made on 38 days. The mean number of sightings per individual was 6.4 (range = 1 - 18). The majority of individuals were only seen in one subcolony (3-4 year olds: 72% (*n* = 38); 5-7 year olds: 79% (*n* = 11)). The cumulative number of individual 5-7 year olds seen increased until late-May and then stabilised, whereas the cumulative number of 3-4 year olds increased throughout the period (Fig. 3a). There was also an increase in the average number of immatures seen per watch as the season progressed in CS, CB and F (e.g. Fig. 3b).

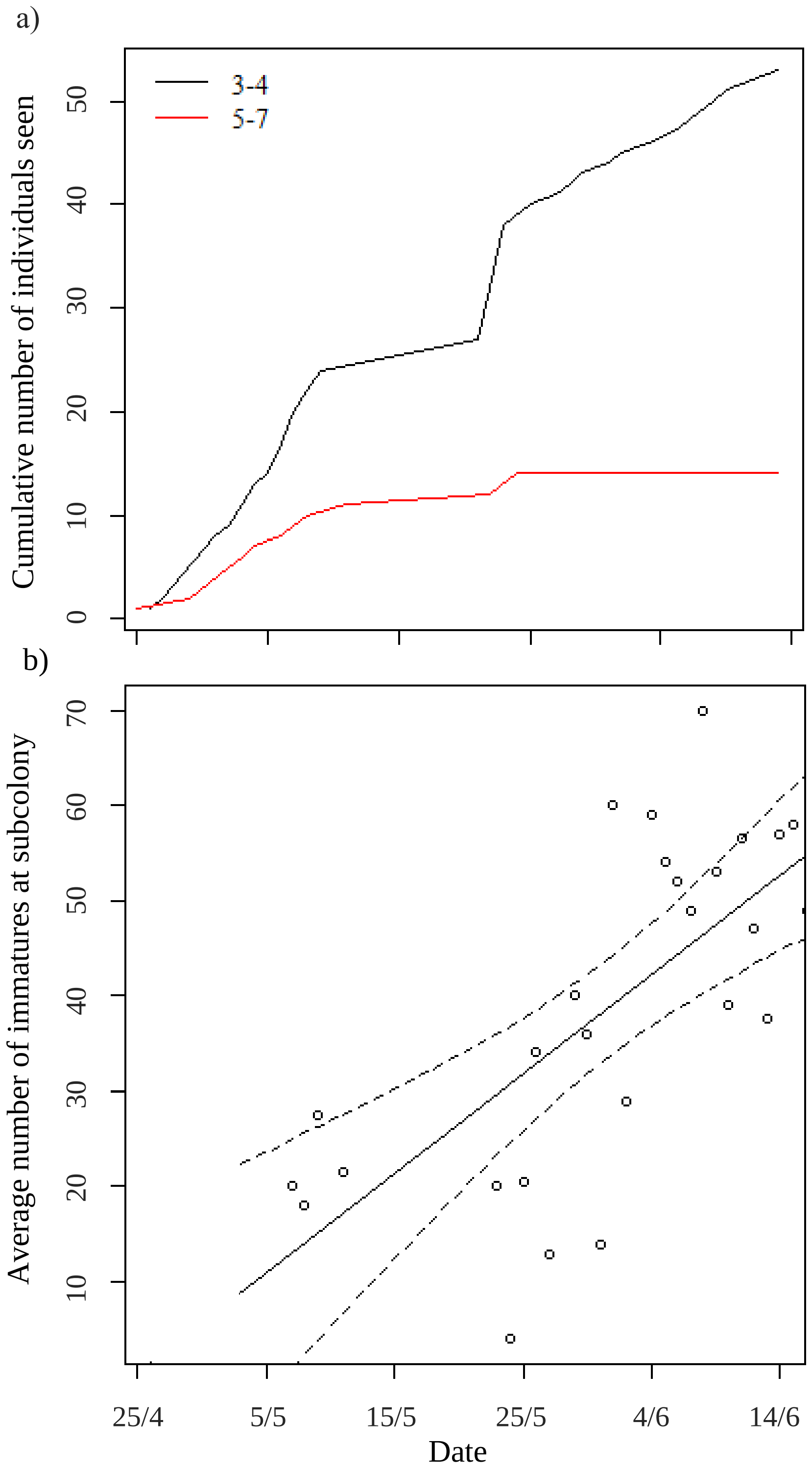

Fig. 3 a) Cumulative number of individual 3-4 and 5-7 year old immature birds seen across all subcolonies, and b) Average number of birds (ringed and un-ringed) seen at subcolony CS in the immature area per watch across sampling period (regression line shown, with 95% confidence interval).

Modelling subcolony attendance rates of immature birds

Model-averaged estimates indicate that the probability of seeing a bird ashore in CB, CM and F was greater than in CS (95% CI = [0.34, 1.78]; [0.45, 2.29]; [0.47, 2.60]; Table S2). They also indicate that the probability of seeing an immature bird across all subcolonies significantly increased (95% CI = [0.45, 1.33]) from 0.06 to 0.22 as the season progressed (Table S2). There is also evidence that the probability of seeing a bird increased in the early part of the sampling period before levelling off (95% CI = [-1.39, -0.07]; Table S2). However, the same analysis run on a subset of the data which only included subcolonies with both 3-4 and 5-7 year old birds (i.e. CB, CM and F) gave similar results but indicated that subcolony and the quadratic date term were insignificant (did not appear in top models; 95% CI included zero). The random effect of individual was found to be highly significant (ΔAICc = 68.31), suggesting that individuals differed markedly in their probability of being seen ashore.

A total of four top models were identified (Table 1). Subcolony, date and the quadratic date term appeared in all the top models, resulting in relative variable importance values of 1.00. The interaction between date and age (0.70) and time of day (0.49) also appeared in the top models (Table S2).

b) Locations of immature birds

A total of 221 watches of 53 individual 3-4 year old and 10 individual 5-7 year old guillemots were made, resulting in approximately 40 hours of observation. The stratified method of sampling resulted in a mean of 18.4 watches (range = 5 - 27) including subcolony F and 21.4 watches (range = 17 - 27) excluding subcolony F, for each subcolony and time-period combination. The mean number of watches per individual guillemot was 3.3 (range = 1 - 12).

Most individuals (49% of birds aged 3-4 years and 73% of birds aged 5-7 years) were watched more than once, and 12 individuals were watched more than five times.

In 72% (n = 160) of watches, no movement into the breeding area was recorded indicating that immature movement into the breeding area was a relatively infrequent event. When immature birds entered breeding areas, they spent less than half of watch in breeding areas in 38% cases (n = 22 watches, or 15 individuals) and more than half, in the remaining 62% of cases (n = 36 watches, or 24 individuals). A significant Hartigan's Dip Test ($p < 0.01$) suggests at least a bimodal distribution of proportions. However, the majority of immature birds in the former category spent less than 20% of a watch in breeding areas (n = 14, or 10 individuals) and in the latter category, the majority spent more than 90% of a watch in breeding areas (n = 30 watches, or 17 individuals). The majority of these cases were observed in CB and CM respectively (Fig. 4). 15 individual birds spent 100% of a watch in breeding areas. They were initially seen within breeding areas, but on the periphery, and remained in this area (e.g. Fig. 5a). 11 of these individuals were also seen to attend immature areas outside the breeding colony during other watches. However, 4 of these individuals (6% of total individuals sampled) were never seen in immature areas. These were equally distributed between CM and CB.

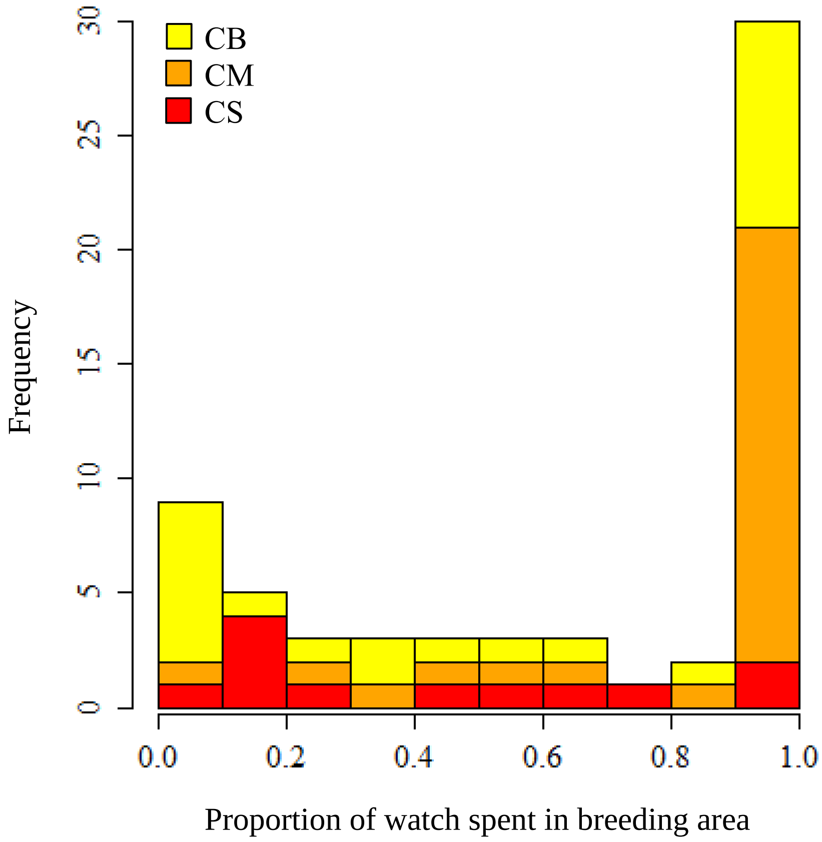

Fig. 4 Frequency distribution of the proportion of a watch spent by immature birds in breeding areas, with colours indicating frequencies for three different subcolonies (CB, CM and CS).

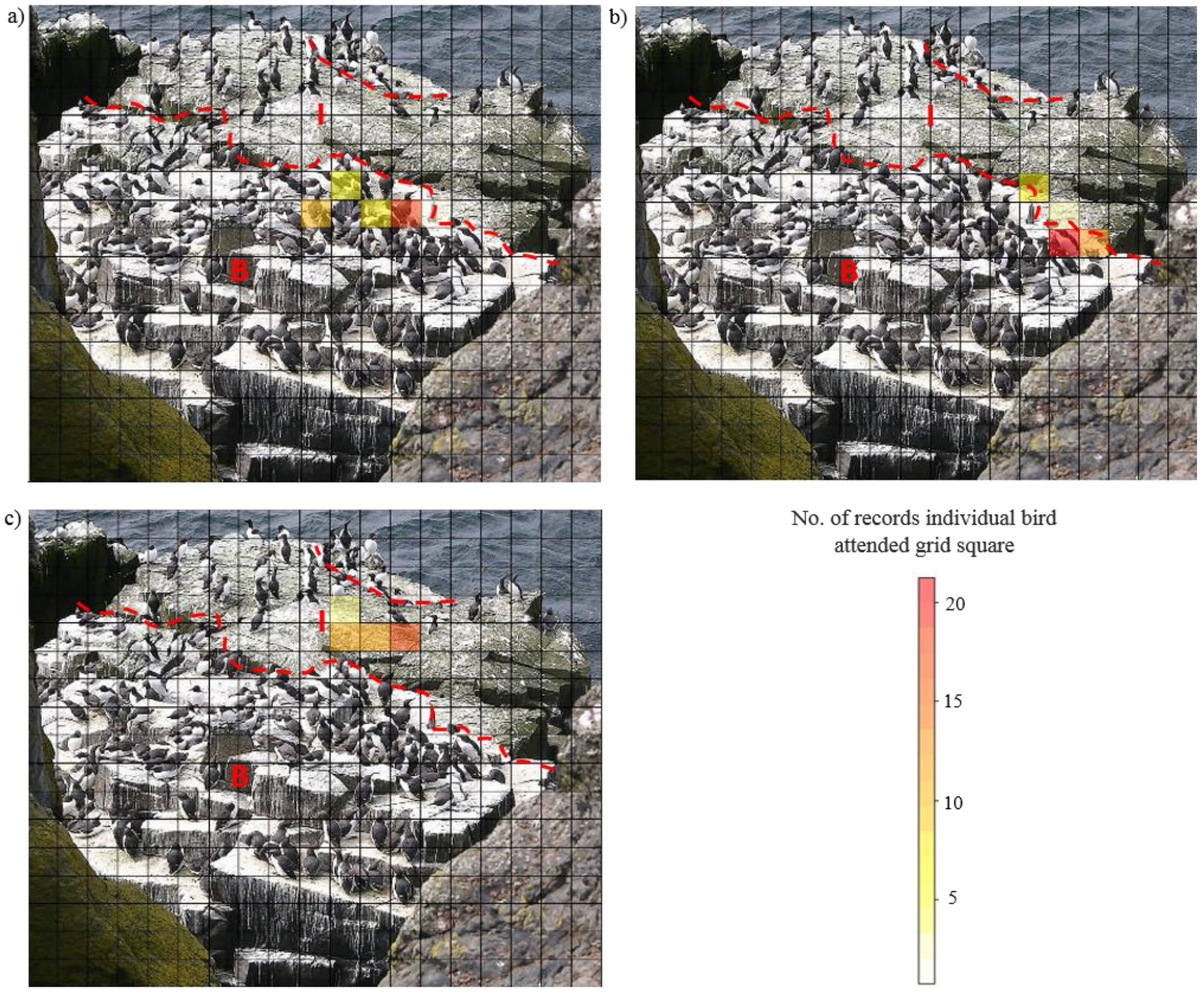

Fig. 5 Maps showing intensity of grid cell use by immature guillemots in subcolony CS that spent a) 100%, b) < 100% but > 0%, and c) 0% of watch in breeding areas. The boundaries between the immature (I) and breeding (B) areas are marked in red.

Modelling the probability of an immature bird entering a breeding area

Model-averaged estimates indicate that the probability of seeing an immature bird entering breeding areas increased as the season progressed (95% CI = [0.14, 1.65]), with for example, the probability in subcolony CS increasing from 0.05 to 0.26 (Table S3). A total of four top models were identified that best explained whether an immature guillemot entered breeding areas or not (Table 2). These top models included the variables date, subcolony, age and breeder attendance. The relative importance values for these variables were 1.00, 0.83, 0.21 and 0.19 respectively

(Table S3). There was no evidence for a significant effect of age, subcolony or breeder attendance (all 95% CI include zero; Table S3).

A highly significant effect of individual was found (ΔAICc = 17.65) which suggests that the probability of an immature entering breeding areas varies significantly with individual. Of the individuals that were sampled more than once (*n* = 47), a distinction was found between those individuals that were often seen entering breeding areas ($Pr > 0.5$; *n* = 8), those that were occasionally seen entering breeding areas ($0 < Pr \leq 0.5$; *n* = 17) and those that were never seen entering breeding areas ($Pr = 0$; *n* = 22; Fig. 6).

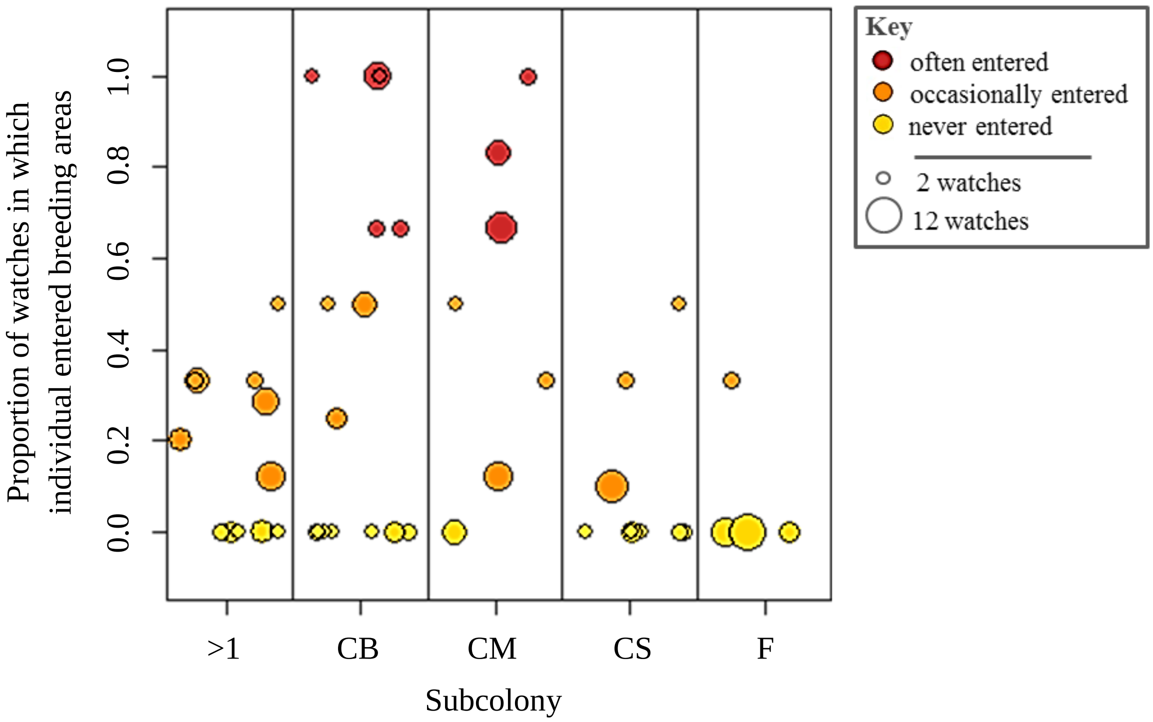

Fig. 6 Distinction between those individuals that were often seen entering breeding areas (red circles), those that were occasionally seen entering breeding areas (orange circles) and those that were never seen entering breeding areas (yellow circles). Individuals are ordered by subcolony (CS; CM; CB; F; >1: individuals that were seen at more than one subcolony). Only individuals recorded two or more times during watches of more than 5 minutes were included in this plot. The relative number of watches per individual is indicated with circle size.

Modelling the proportion of time spent by immature birds in breeding areas

Model-averaged estimates suggest that the effect of increasing breeder attendance on the proportion of time spent by immatures in breeding areas was significantly more negative in CB and CM than CS (95% CI = [-3.73, -0.90]; [-3.47, -0.62]; Table 4S). At a mean value of breeder attendance (and weather), the proportion of time spent in breeding areas was 0.10-0.14 in CS and 0.28-0.51 in CB and CM (Table 4S). A total of three top models were identified that best explained the proportion of time spent by immature birds in the breeding area (Table 3). The interaction between subcolony and breeder attendance appeared in all of the top models, resulting in a relative variable importance values of 1.00. Time of day (0.60) and weather (0.18) also appeared in the top models (Table 4S). There was no evidence for a significant effect of time of day or weather (all 95% CI include zero; Table 4S), and little evidence for a significant effect of individual (ΔAICc = 2.15).

Table 1. Subcolony attendance: Top models estimating the probability of an immature guillemot being seen during a watch of a subcolony.

Model	Log-likelihood	d.f.	AICc	ΔAICc	Weight	R^2_{COR}
Subcolony, Date^2, Age*Date,	-711.58	9	1441.3	0.00	0.22	0.15
Subcolony, Date^2, Age*Date, Time period	-709.65	11	1441.4	0.18	0.20	0.15
Time period, Subcolony, Date, Date^2	-712.44	9	1443.0	1.71	0.09	0.15
Subcolony, Date, Date^2	-714.54	7	1443.1	1.89	0.09	0.14

Table 2. Probability of entering breeding areas: Top models estimating probability of an immature bird entering breeding areas.

Model	**Log-likelihood**	**d.f.**	**AICc**	**ΔAICc**	**Weight**	R^2_{COR}
Date, Subcolony	-103.27	6	218.9	0.00	0.15	0.53
Date, Subcolony, Age	-102.90	7	220.3	1.39	0.08	0.53
Date, Subcolony, Breeder attendance	-103.04	7	220.6	1.68	0.07	0.54
Date	-107.33	3	220.8	1.84	0.06	0.58

Table 3. Proportion of time spent in breeding areas: Top models estimating proportion of time spent by an immature in breeding areas.

Model	**Log-likelihood**	**d.f.**	**AICc**	**ΔAICc**	**Weight**	R^2_{COR}
Breeder attendance*Subcolony, Time period	-214.52	10	453.4	0.00	0.20	0.56
Breeder attendance*Subcolony	-217.36	8	453.5	0.05	0.19	0.60
Breeder attendance*Subcolony, Time period, Weather	-213.85	11	455.1	1.65	0.09	0.62

Intensity of grid cell use within breeding areas by immature birds

Maps showing the intensity of use of grid cells within the breeding areas indicate that immature guillemots tend to concentrate around the periphery of breeding areas and are less likely to be recorded in more central areas. However, these spatial patterns vary with subcolony (Fig. 7; Table 4).

Table 4. The number of peripheral and central grid cells in breeding areas of each subcolony with immature activity. Central grid cells are defined as those surrounded by breeding activity and peripheral grid squares as those that are not. Immature activity is classified as low if an immature bird was seen to attend a grid cell during 3 or less watches and high if an immature bird was seen during more than 3 watches.

Subcolony	**No. peripheral breeding grid cells with immature activity**		**No. central breeding grid cells with immature activity**	
	Low	**High**	**Low**	**High**
CS	8	1	3	0
CB	9	2	2	0
F	1	0	0	0
CM	14	3	10	0

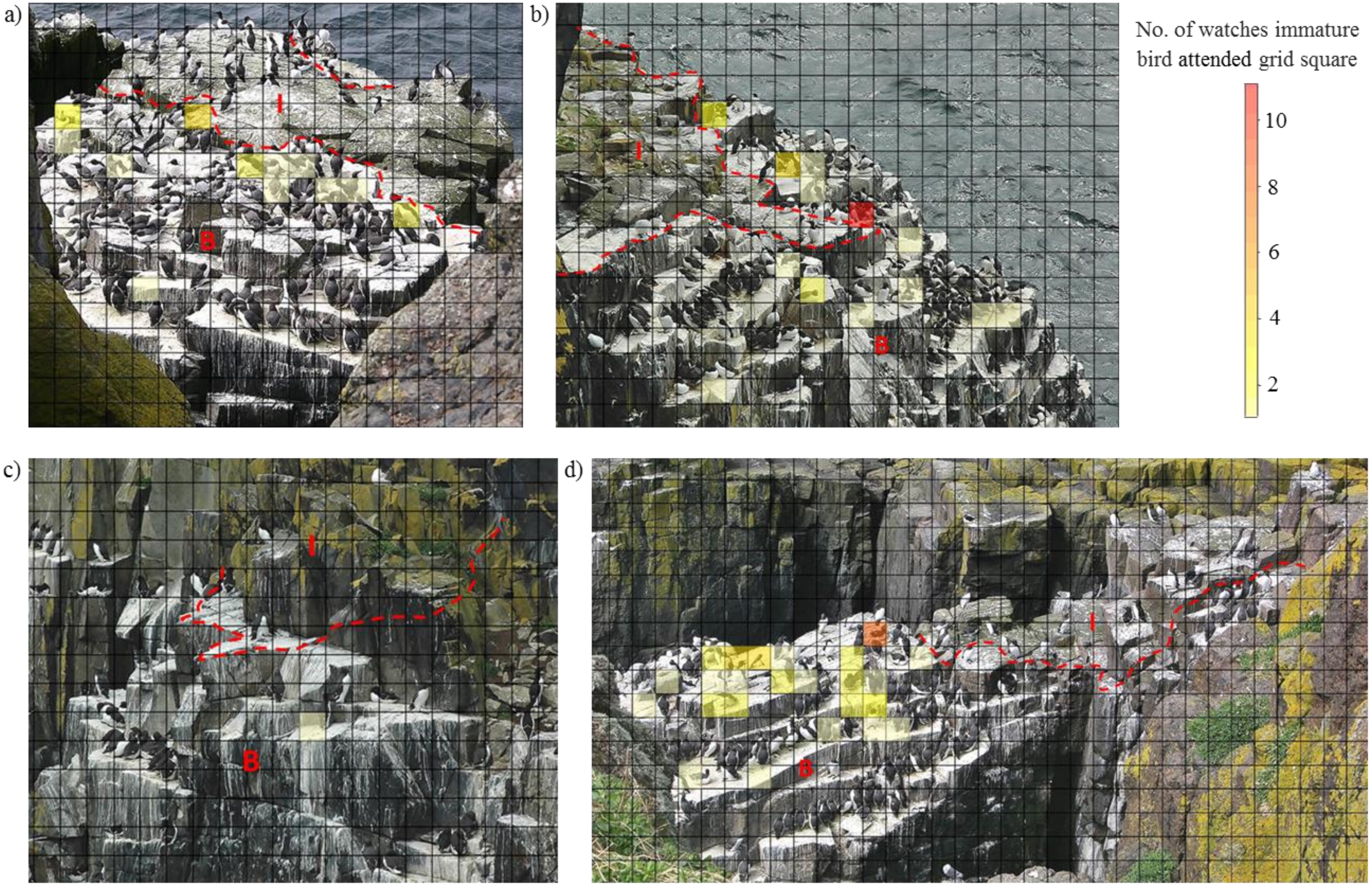

Fig. 7 Maps of immature birds' intensity of area usage showing the 'hotspots' for immature attendance within breeding areas of each subcolony: a) CS, b) CB, c) F and d) CM. Approximate immature (I) and breeding (B) areas are marked in red.

Discussion

In this study, I have shown that immature individuals have the potential to play an important role in shaping wildlife parasite dynamics, as a result of their heterogeneous attendance rates and spatial attendance patterns. This knowledge of the heterogeneities within a population is necessary to accurately quantify key epidemiological parameters such as the transmission rate, and to predict future parasite dynamics. It also warns against drawing general conclusions about infection risk across whole populations.

Age effects

I found evidence for some individuals spending more time ashore and entering breeding areas more often when ashore than others. There was no evidence that these individual differences were associated with the ages of birds. A recent study by Lahoz-Montfort *et al.* (2014), including data from multiple years, presents evidence of age-specific resighting probabilities for the same species and site. However, my study was limited to a single season. The lack of a significant effect of age in this study may be because it was limited to a single season and it was only possible to sample a limited number of birds in two age categories (3-4 years and 5-7 years), affecting the power of my analyses. Few 3 years olds were observed as they less frequently attend colonies (Halley *et al.* 1995), and few 7 years olds were observed, as this is the average age of first breeding (Harris, Halley, & Swann, 1994; Lewis *et al.* 2006; Harris pers. comm.). Furthermore, fewer 5 years olds and no 6 year olds were observed in the colony, likely due to very low breeding success in 2007 and 2008 (Ashbrook *et al.* 2008) and subsequent low post-fledging survival (Lahoz-Monfort *et al.* 2014). With regards to spatial attendance patterns, there is also likely to be natural variation in the physiological development of immature birds, with some birds ready to breed at a younger age. In future studies, it would be interesting to follow these immature birds until successful recruitment and then to test, for example whether those birds that recruit in the next year demonstrate significantly different spatial attendance patterns in my study than those that recruit in later years. Halley, Harris & Wanless (1995) have already showed that in certain periods, recruits invest triple the amount of time in colony attendance than nonrecruits of the same age.

Limited attendance of immature birds in breeding areas

I found that the spatial attendance patterns of immature birds depended on the subcolony and the date. Although immature birds were found to infrequently enter breeding areas at the beginning of the season, they did so more frequently as the season progressed. Levels of aggression in guillemots have been previously shown to increase in the late incubation and chick period, and this has been linked to an 'influx' of prospecting immatures (Birkhead 1978). Although birds in subcolony CS spent a smaller proportion of time in breeding areas and were limited to the periphery of the colony, birds in subcolonies CB and CM spent significantly more time in breeding areas, and birds in CM penetrated further into the colony. These differences may be due to numbers of free nest sites or topography. For example, immature birds in some subcolonies may have a better vantage point over the breeding area than others.

Lack of an effect of breeder attendance on attendance of immature birds in breeding areas

I found no evidence for an overall effect of breeder attendance on the spatial attendance patterns of immature birds. However, for CB, CM and CS, I did find a significant effect of the interaction between subcolony and breeder attendance on the proportion of time spent by immature birds in the breeding area. These findings may be due to a) subcolony and breeder attendance being confounded, b) the lack of variation in breeder attendance at the within-season scale, and/or c) the breeder attendance at a particular breeder plot not being representative of the breeder attendance at the subcolony as a whole. An alternative, but much more time consuming, way of recording breeder attendance would be to simply count the total number of birds in all breeding areas of a subcolony. I also found no effect of individual on the proportion of time spent by immature birds in breeding areas, suggesting that although certain individuals were more likely to enter breeding areas than others, they were no more likely to remain in breeding areas. This could be due to the current effectiveness of ejection of immature prospecting birds from the breeding colony.

Implications for parasite transmission

The spatial attendance patterns of immature birds are likely to influence opportunities for host-mediated tick dispersal from immature to breeding areas. For example, the frequency of entry into and proportion of time spent in breeding areas will be proportional to the chance of a tick detaching from an immature bird while it is in the breeding area. Whether immature birds are restricted to peripheral parts or also attend more central parts of breeding areas is also likely to determine whether the risk of tick dispersal is uniform across breeding areas. It is worth noting that ticks that do disperse from immature to breeding areas will retreat into cracks in the breeding areas, having completed their single annual blood-meal, before re-emerging to feed in the following year. So, there will be lag in the exposure of breeding birds and their chicks to these parasites.

The spatial attendance patterns of immature birds will need to be incorporated into future epidemiological models of this system to more fully explore the effects of interacting age-related processes on GIV dynamics, and by extension on other wildlife parasites. For example, those subcolonies, like CS, where immature birds spend a smaller proportion of time in breeding areas are likely to have more restricted opportunities for host-mediated tick dispersal from immature to breeding areas, resulting in lower relative exposure of breeding birds and their chicks to the parasites carried by immature birds. Similar spatial heterogeneity in risk of exposure has been observed in king penguin colonies (Gauthier-Clerc *et al.* 1999). There is also likely to be lower relative exposure earlier in the season, when immature birds less frequently enter breeding areas.

More specifically, the rate of infection of breeding birds and the diversity of GIV strains in these subcolonies and at these times of the year, is likely to be lower. My findings also suggest that the exposure of breeding birds and their chicks to these parasites is likely to spatiotemporally heterogeneous across the Isle of May colony as a whole. The same is also true for individuals; those individuals with limited attendance of the breeding area are likely to restrict opportunities for host-mediated tick dispersal from breeding to immature areas, as expected. However, I have found evidence for a subset of individuals that consistently enter breeding areas, as well as attending immature areas. These potential 'super-spreaders' (Lloyd-Smith *et al.* 2005) are likely to drive the dynamics of GIV in the breeding colony. Therefore, both spatial and individual heterogeneities (Paull *et al.* 2012), as well temporal heterogeneities are likely to be critical components in future epidemiological models of this system.

The proportion of time spent ashore by immature birds is likely to be proportional to the risk of tick attachment, also influencing opportunities for host-mediated tick dispersal from immature to breeding areas. My estimates of subcolony attendance rates of immature guillemots should therefore be incorporated into future epidemiological models along-side my estimates of their spatial attendance patterns. I found that the proportion of time spent ashore by immature birds is limited, varying between 0.06 and 0.22 or approximately 1 to 4 hours within each 24 hours. My findings suggest that the exposure of breeding birds and their chicks to parasites is likely to increase as the breeding season progresses. However, tick feeding activity is known to vary across the breeding season, with a peak around 1 June for adult females and two peaks around 18 May and 6 July for nymphs (Barton *et al.* 1996); further influencing the risk of exposure of breeding birds. My findings also suggest that, as for spatial attendance patterns, some individual immature birds are likely to increase this exposure more than others. Although my findings do not show this due to obvious limitations, the findings of Lahoz-Montfort *et al.* (2014) suggest that older immature birds are likely to increase this exposure, although the degree to which they do this is likely to vary from year to year. The level of acquired immunity is likely to vary within the immature bird population, with older immatures having slightly higher levels of acquired immunity than younger immatures. The susceptibility of a bird is likely to be inversely related to the infection rate of ticks derived from it. This would suggest that younger immatures pose more of a risk to breeding birds and their chicks than older immatures. The overall risk of transmission from prospecting immature to breeding birds can therefore be described as a function of the proportion of time spent on land by an immature and its spatial attendance patterns, as well as its susceptibility to infection. These are all potential components of future epidemiological models.

Although, breeder attendance alone was not shown to be significant in this study, one way in which I plan to use these epidemiological models is to further investigate this effect on the spatial attendance patterns of immature birds and hence on the dynamics of GIV in the breeding colony. I plan to use the estimates of the key mixing rate parameter from the work I have undertaken here as a baseline for comparison with simulated perturbed situations. This work will be critical for investigating the potential consequences of social disruption for parasite dynamics. Social disruption is a real threat to this guillemot colony, having recently experienced an acute decrease in breeder attendance caused by food shortages (Ashbrook *et al.* 2010), as well as the chronic decline in breeding numbers as a result of demographic change (Harris, Frederiksen & Wanless 2007; JNCC 2014). As well as altering the spatial attendance patterns of immature birds by increasing the number of unoccupied sites in breeding areas, these changes have the potential to influence the timing of recruitment and hence the level of acquired immunity within the breeding colony as a whole. Predicting how colonies will respond to these changes in both environmental and social conditions, as well as the possible additional threat from endemic parasites is crucial for the conservation of guillemots and other colonial wildlife species.

Conclusion

Using data on the subcolony attendance rates and spatial attendance patterns of known-aged guillemots, in the context of previous work describing the dynamics of GIV in the breeding colony, I have shown that complex interactions between age-related processes and the environment have the potential to significantly affect wildlife parasite dynamics. My future modelling work will provide evidence to further support this view.

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Supplementary material

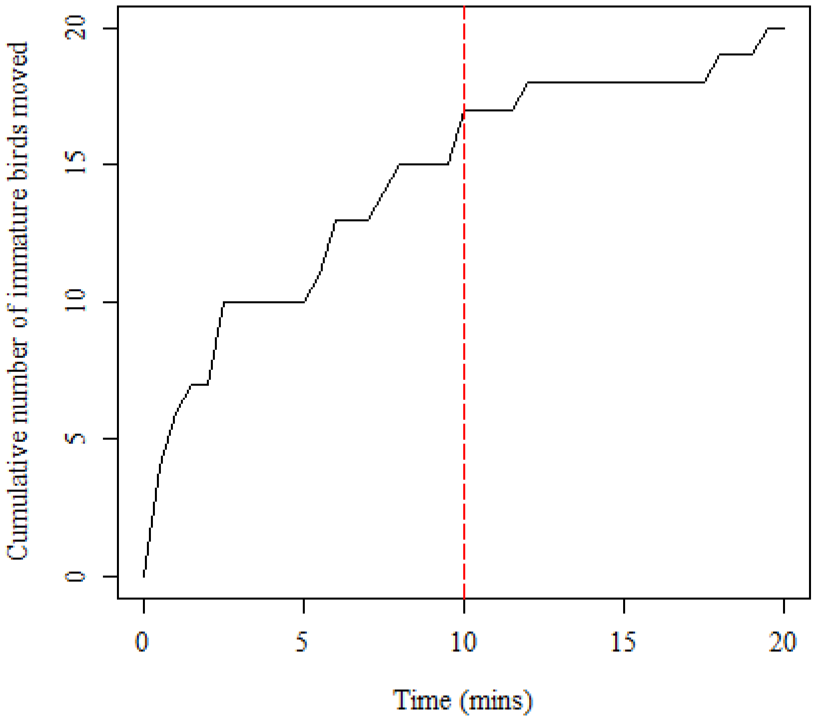

Fig. S1 The cumulative number of immature birds which changed grid cell (i.e. moved an average of 50 cm) during 20 min watches. The red line shows the chosen optimum sampling period of 10 minutes.

Table S1. Five or six-point scale used to record weather variables

Scale	**Weather variable**		
	Wind strength	**Air temperature**	**Sea condition**
1	None	Very warm	Still
2	Light breeze	Warm	Calm
3	Fair breeze	Comfortable	Fairly choppy
4	Strong	Cool	Very choppy
5	Gale force	Very cool	High waves
6	-	Freezing	-

Table S2. Subcolony attendance: Model-averaged parameter estimates (95% CI), unconditional standard errors, estimated *p* values and relative importance of predictors of the probability of an immature guillemot being seen during a watch of a subcolony.

Parameter^1	**Model-averaged estimate**^{2}	**Unconditional SE**	**Estimated *p* value**	**Relative importance**
(Intercept)	**-2.85 (-3.48, -2.22)**	**0.32**	**< 0.001**	**-**
Age 5 - 7	0.37 (-0.40, 1.14)	0.39	0.34	0.70
Subcolony CB	**1.06 (0.34, 1.78)**	**0.37**	**< 0.01**	**1.00**
Subcolony CM	**1.37 (0.45, 2.29)**	**0.47**	**< 0.01**	“
Subcolony F	**1.53 (0.47, 2.60)**	**0.54**	**< 0.01**	“
Date	**0.89 (0.45, 1.33)**	**0.23**	**< 0.001**	**1.00**
Date^2	**-0.73 (-1.39, -0.07)**	**0.34**	**0.03**	**1.00**
Age 5 – 7 × Date	-0.50 (-1.36, 0.36)	0.44	0.26	0.70
Time period 2	-0.17 (-0.59, 0.25)	0.21	0.42	0.49
Time period 3	-0.06 (-0.32, 0.20)	0.13	0.64	“

Table S3. Probability of entering breeding areas: Model-averaged parameter estimates (95% CI), unconditional standard errors, estimated *p* values and relative importance of predictors of the probability of an immature bird entering breeding areas.

Parameter[1]	**Model-averaged estimate**[2]	**Unconditional SE**	**Estimated *p* value**	**Relative importance**
(Intercept)	**-1.95 (-2.86, -1.05)**	**0.46**	**< 0.001**	**-**
Date	**0.90 (0.14, 1.65)**	**0.38**	**0.02**	**1.00**
Subcolony CB	0.91 (-0.41, 2.23)	0.67	0.18	0.83
Subcolony CM	0.43 (-0.68, 1.54)	0.56	0.45	"
Subcolony F	-1.51 (-4.22, 1.20)	1.38	0.28	"
Age 5-7	0.11 (-0.56, 0.77)	0.34	0.75	0.21
Breeder attendance	-0.05 (-0.49, 0.38)	0.22	0.81	0.19

Table S4. Proportion of time spent in breeding areas: Model-averaged parameter estimates (95% CI), unconditional standard errors, estimated *p* values and relative importance of predictors of the proportion of time spent by an immature in breeding areas.

Parameter[1]	**Model-averaged estimate**[2]	**Unconditional SE**	**Estimated *p* value**	**Relative importance**
(Intercept)	**-1.23 (-1.73, -0.73)**	**0.25**	**< 0.001**	**-**
Breeder attendance	**2.11 (0.69, 3.54)**	**0.71**	**< 0.01**	**1.00**
Subcolony CB	**0.73 (0.19, 1.28)**	**0.27**	**< 0.01**	**1.00**
Subcolony CM	**1.12 (0.59, 1.65)**	**0.27**	**< 0.001**	**"**
Time period 2	0.13 (-0.07, 0.51)	0.16	0.40	0.60
Time period 3	-0.08 (-0.39, 0.14)	0.12	0.53	"
Breeder attendance × Subcolony CB	**-2.32 (-3.73, -0.90)**	**0.71**	**< 0.01**	**1.00**
Breeder attendance × Subcolony CM	**-2.04 (-3.47, -0.62)**	**0.71**	**< 0.01**	**"**
Weather	-0.03 (-0.13, 0.03)	0.07	0.73	0.18

[1] The parameters, subcolony CS, Age 3-4 and Time period 1, Date, Breeder attendance and Weather were set to 0 in model-averaged estimates.

[2] Model-averaged estimates are transformed and standardised on two *SD* following Gelman (2008). 95% confidence intervals spanning zero suggest non-significance; CI for parameters shown in bold do not include zero.

Improved estimates of age- and strain-specific seroprevalence and force of infection for a tick-borne virus, *Great Island virus*, in a wild population of common guillemots (*Uria aalge*).

Abstract

Parasite transmission rates are critical for understanding host-parasite dynamics, but are also very difficult to estimate, particularly in wildlife host-parasite systems. Many studies have estimated the more easily-quantifiable force of infection (FOI) for many human, but only a few wildlife infections. In this study, I use seroneutralisation data from a large common guillemot (*Uria aalge*) colony which has been monitored as part of a long-term study, to provide improved estimates of strain and age-specific seroprevalence and FOI for a tick-borne parasite, *Great Island virus* (GIV). 12 strain-specific FOI estimates are presented, ranging widely from -0.07 to 0.51 year^{-1}. Although estimates for four of these strains already exist in the literature, 8 of these represent the first published estimates for this system. Age-specific estimates support breeding-status-dependent patterns in seroprevalence and FOI described previously in the literature, with a higher pre-breeding than breeding seroprevalence across all strains, and a higher pre-breeding than breeding FOI for 8 out of 12 strains (average pre-breeding FOI = 0.21 year^{-1}, range = 0.01 – 0.51 year^{-1}; average breeding FOI = 0.08 year^{-1}, range = -0.07 – 0.24 year^{-1}). Both strain and age-specific estimates provide a sound basis for improving estimates of GIV transmission rates, and further investigating the ecological drivers of GIV spread.

Introduction

Some wildlife parasites have significant, and often overlooked, impacts on their wildlife hosts (Smith, Sax & Lafferty 2006), threatening biodiversity and conservation efforts (Daszak, Cunningham & Hyatt 2001). Estimating parasite transmission rates is critical for understanding host-parasite dynamics (McCallum, Barlow & Hone 2001). One approach is to estimate the force of infection (FOI), the per capita infection rate of susceptible hosts, from age-specific sero-prevalence data (Muench, 1959). This has been done for many human (e.g. Ferguson, Donnelly, & Anderson, 1999; Grenfell & Anderson, 1985) but only a few wildlife infections (Nunn *et al.* 2006a; Swai *et al.* 2008), as these kind of data are rare, sample sizes are often small and ageing of individuals can be difficult for wild species. In addition, the continuous nature of sero-prevalence data makes them difficult to interpret (Gilbert *et al.* 2013). A cut-off is often used to distinguish seropositive and seronegative results, which indicate whether or not an individual has been exposed to infection (Gilbert *et al.* 2013). Data from long-term wildlife studies can be used to increase sample sizes and to improve age estimates, if individual histories are followed. This study uses neutralisation data against a tick-borne virus for a wild population of common guillemots (*Uria aalge*) which have been monitored as part of a long-term study on the Isle of May, and contributes to the literature highlighting the value of using finite mixture models to further understanding of wildlife parasite dynamics.

Great Island virus (GIV; Attoui *et al.* 2012) is a highly serotypically diverse orbivirus with a circumpolar distribution. On the Isle of May, the main host of GIV has been identified as the common guillemot (Nunn *et al.* 2006b), which lives in large, dense and spatially-structured colonies usually on cliffs. Within these colonies, immature birds prospecting for a nest site congregate around the edges of breeding areas. Previous work suggests that host breeding status and virus strain are key factors in GIV transmission. Nunn *et al.* (2006a & 2006b) showed that guillemots acquired immunity to GIV with age and suggested that this may explain the higher prevalence of GIV in seabird ticks (*Ixodes uriae*) that fed on pre-breeding than breeding birds. They also showed differences in the percentage of birds that were immune to each of four virus strain. However, estimates of FOI and interpretation of the significance, if any, of differences in the percentage of pre-breeding and breeding birds immune to each strain have so far been hampered by small sample sizes (Nunn *et al.* 2006a).

In this study, I test blood samples from 69 individual breeding and 75 individual pre-breeding guillemots, collected between 1993 and 1995, against 12 strains of GIV for virus-specific neutralising antibody. I use strain-specific finite mixture models to classify individuals as either seropositive or seronegative, and present improved age and strain-specific seroprevalence estimates. These models have recently been used to estimate the seroprevalence of other wildlife parasites (Peel *et al.* 2013), and allow individuals to belong to dynamic and overlapping exposure groups, rather than imposing a pre-defined cut-off in the level of neutralising activity expected from an individual that has been infected. I then go on to use a maximum likelihood method to estimate virus strain-specific and host-breeding-status-specific FOI, and to test my hypotheses that a) the FOI of GIV varies with strain, and b) the FOI acting on birds is higher before first breeding than after first breeding.

Methods

Blood sampling

Individual immature pre-breeding and breeding guillemots were captured between 1993 and 1995 on the Isle of May, Scotland. Breeding guillemots were caught from breeding ledges using either a wire crook on the end of a 3 metre long bamboo pole or a monofilament noose fitted to a 5 metre fishing rod. The majority of immature pre-breeding guillemots were caught in box traps on ledges above breeding ledges (Nunn *et al.* 2006b). Some guillemots (approximately two-thirds) were already ringed with a unique colour combination for study purposes and/or a unique British Trust for Ornithology (BTO) serial metal ring. All other guillemots were ringed before release. Breeding status was deduced from the presence of an egg, chick or brood patch. Sex was deduced by repeated observation of mating behaviour of breeding birds. Blood samples were taken from the foot web, collected on filter paper and stored at -70°C, as described in Nunn *et al.* (2006a).

Estimating age of birds

Approximately 5% of breeding adults were previously caught and ringed as chicks, so their exact age was known. Unringed birds that were caught as breeding adults were estimated to be at least 7 years, the average age of first breeding (Harris, Halley, & Swann 1994; Lewis *et al.* 2006; Harris pers. comm.). The age of breeding birds that were previously ringed as adults was estimated by adding the number of years since ringing, to this median age of first breeding. All immature pre-breeding birds caught were unringed and therefore of unknown age. An average age of 4 years was assumed, as previous work suggests that this is the average age of immature pre-breeders found on the ledges above breeding areas in the colony (Halley, Harris, & Wanless 1995; Lahoz-Monfort *et al.* 2014; Chapter 4).

Detection of virus-specific neutralizing antibodies

Plaque reduction neutralisation tests (PRNT) were performed on blood samples to detect virus-specific neutralising antibodies. The method can be found in Nunn *et al.* (2006a). Virus dilutions that gave approximately 100 plaques per well were used. The eluted blood used for PRNT equates to approximately a 1:30 dilution of serum. All blood samples were assayed at least two times, and an average fold decrease in virus titre relative to a (at least) duplicated negative control was calculated (AVFOLD). The negative control used was chicken blood eluted from filter paper. I also tested a positive control of blood from a breeding guillemot that greatly reduced the PRNT titre of all 12 GIV strains, as a measure of the reliability of this method. The mean fold reduction in virus titre for the positive control was 28.0 (range = 19.1 – 51.1). I tested 12 GIV strains, which I refer to as MDNV_1992 (Maiden Virus), CBNV_1994 (Colony B North Virus), 116V_2003, 53V_2003, 492V_1994, 243V_2003, COYV_1994 (Colony Virus), 283V_1993, 8V_2003, BRDV_1975 (Broadhaven Virus), 89V_2003 and AMDV_1994 (Above Maiden Virus). The numbers after the underscore in each name refer to the year that ticks were collected and virus isolated. All 12 strains were isolated by application of clarified tick homogenate to BHK21 cells and two rounds of plaque picking on Vero cells as described in Nunn *et al.* 2006a. All the strains except BRDV_1975 were obtained from single ticks collected on the Isle of May. BRDV_1975 was isolated from a pool of 10 nymphal *I. uriae* collected at St Abbs Head, Scotland. Two of the strains (243V_2003 and CBNV_1994) have virtually identical outer capsid protein sequences and are very similar in terms of their neutralisation profiles by PRNT (Nunn pers. comm.); the remaining strains are assumed to be antigenically similar to varying degrees.

Mixture modelling

The flexmix package (Leisch 2004; Grun & Leisch 2008) in R 3.0.2 (R Core Team 2013) was used to apply a latent-class approach and to fit one to three normal distributions to the natural logarithm of the AVFOLD for each strain. This package uses an expectation-maximisation (EM) algorithm to estimate model parameters. Each model was fitted 100 times and the best one identified using the Bayesian Information Criterion (BIC). This criterion was used to reduce the chance of the optimal number of components being overestimated (Leisch 2004).

Interpretation depended on the optimal number of component distributions identified. If one component was identified, then it was assumed that no distinction could be made between seropositive and seronegative individuals. If two component distributions were identified, then the component with the lower mean was assumed to represent the seronegative individuals, and the component with the higher mean was assumed to represent the seropositive individuals. For three component distributions, the component with the intermediate mean was assumed to represent individuals that were seropositive from prior infection, and the component with highest mean was assumed to represent individuals that were seropositive from ongoing infection. Posterior probabilities were used to assign each individual to one of the components. Rootograms of the posterior probabilities were inspected to visually assess the mixture component structure and ensure that components were well to medium well separated. 95% of individuals assigned to the lower, seronegative component demonstrated an average fold reduction of less than or equal to 5, and 76% of individuals assigned to the higher, seropositive component demonstrated an average fold reduction of greater than or equal to 5. This suggests that our models provide biologically sensible categorisations.

This finite mixture modelling method assigns a weight to each component, which indicates the a-priori probability for an observation to come from this component. I present this component weight, as well as the number of individuals assigned to each component, the mean of the average fold reduction for each component and the 95% confidence interval for this mean. Figures are also provided to show the fit of the component distributions to the strain-specific data.

Seroprevalence estimates

I present the percentage of individuals assigned to each component for each strain and for each age category and strain combination.

FOI estimation

I assumed that GIV could be modelled using a catalytic framework. The assumptions of this framework include, a) a one-way flow from susceptible to infected (or immune) individuals, and b) negligible mortality due to infection (Muench 1959). I think that these assumptions are reasonable because other parasites that infect seabirds induce long-term immunity and have limited pathogenic effects on their hosts (Chambert *et al.* 2012).

Generalised linear models (GLMs) with a binomial error were fitted to age-specific serology data in R statistical software (R Core Team 2013) to estimate: a) virus strain-specific FOI, and b) host-breeding status-specific FOI. Each individual bird was tested against multiple strains so, to avoid pseudoreplication, all models were strain-specific. A log link was used, a seronegative bird was considered a 'success' and the proportion of successes in each age category modelled so as to allow FOI to be directly estimated from model outputs. Age estimates were initially modelled as a continuous variable but this gave spurious results due to small sample sizes, particularly for older ages. Therefore, ages were grouped into four categories: pre-breeder aged 2-6, breeder aged 7-11, breeder aged 12-16 and breeder aged 17-21, and the average age within these categories was modelled as a continuous variable. Other age categories were tested but had no qualitative effect on the overall conclusions of the work. It was also assumed that there were no differences in FOI depending on the year that a bird was sampled, to further increase sample size.

A single slope was fitted to the age-specific seroprevalence data to estimate the annual FOI for each strain, or the risk of a susceptible individual becoming infected per year. I present estimates from these models, with 95% confidence intervals. A piecewise constant model with two slopes was applied to the data to estimate breeding-status-specific FOI. A break point of 7-11 (average age = 9) was chosen, as this is the youngest breeding age class. I present annual FOI estimates for each side of this break point (before and after first breeding; hereafter referred to as pre-breeding and breeding FOI respectively) for each strain, with 95% confidence intervals for the latter. 95% confidence intervals are not presented for pre-breeding FOI estimates as they simply represent a difference between two data points. I do not model the FOI prior to the age of 4 (the assumed age of all unringed, immature, pre-breeding guillemots in this study), but assume that it is low due to the infrequency of guillemot colony attendance in the first 2-3 years of life (Halley *et al.* 1995).

Results

Summary of data

The total number of individuals sampled was 144, and the mean number of individuals sampled in each age class was 36 (range = minimum 17 for 12-16 and 17-21 categories – maximum 75 for pre-breeder category). Individuals were sampled from 16 areas across the Isle of May (see Fig. 1), with a mean of 9 individuals sampled in each subcolony (range = 1 - 52). It was possible to sex about half of individuals (*n* = 71, of which 62 were breeders), and of these approximately equal numbers were male (*n* = 40) and female (*n* = 31). The majority of individuals were sampled in 1994 (*n* = 35) and 1995 (*n* = 92), with fewer sampled in 1993 (*n* = 17). The mean number of individuals for each year and age category combination was 12 (range = 0 for pre-breeder category in 1993 – 68 for pre-breeder category in 1995). Most individuals were tested for neutralising antibody against 11 strains (*n* = 76), but some were tested for only 3 (*n* = 24), 4 (*n* = 43) or 10 (*n* = 1). This resulted in a total of 1090 records.

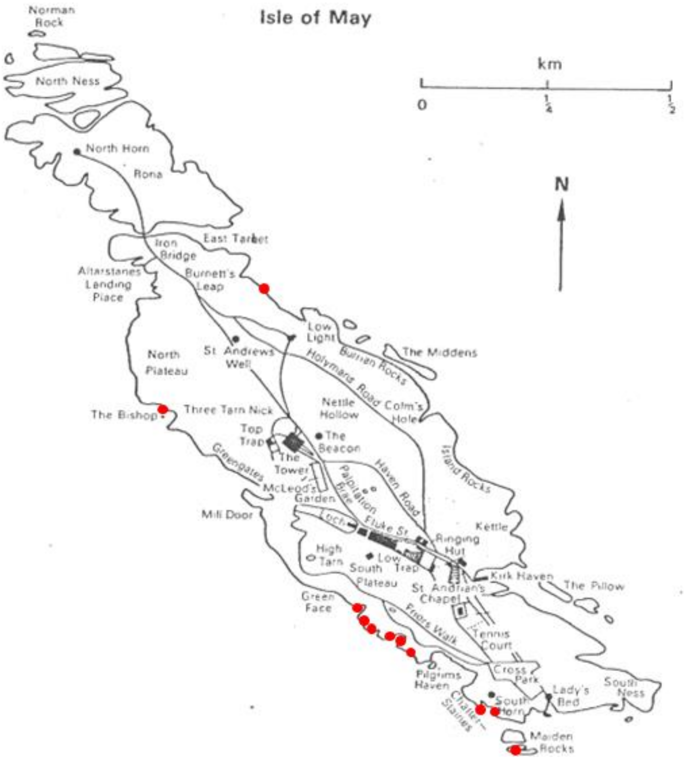

Fig. 1 A map of the Isle of May, Scotland showing the approximate areas (red) from which individuals were sampled.

Mixture modelling

The optimal number of components for the majority of strains was two (Table 1; Fig. 2). For these strains, the mean AVFOLD for the lower component ranged from 1.20 - 2.93 and from 7.88 - 71.45 for the higher component. For the majority of these strains, the lower component weight was higher than the higher component weight (*n* = 9). However, the optimal number of components for AMDV_1994 was three (Table 1; Fig. 2), and the intermediate component was found to have the highest weight.

Table 1 Summary of components of mixture model with lowest Bayesian Information Criterion (BIC) for each strain (means and 95% confidence intervals have been anti-logged for ease of interpretation).

Strain	Component	No. of observations in each component (%)	Component weight	Mean AVFOLD	95% CI for mean
MDNV_1992	Low	49 (63.6%)	60.2%	1.47	(1.30-1.67)
	High	28 (36.4%)	39.8%	14.35	(7.14-29.10)
CBNV_1994	Low	99 (69.2%)	65.2%	1.43	(1.26-1.62)
	High	44 (30.8%)	34.8%	30.53	(14.31-65.76)
116V_2003	Low	35 (45.5%)	40.3%	1.48	(1.29-1.68)
	High	42 (54.5%)	59.7%	9.52	(6.29-14.37)
53V_2003	Low	60 (77.9%)	73.2%	1.84	(1.49-2.26)
	High	17 (22.1%)	26.8%	21.08	(5.17-88.84)
492V_1994	Low	44 (57.1%)	53.9%	1.40	(1.27-1.54)
	High	33 (42.9%)	46.1%	14.35	(8.52-24.09)
243V_2003	Low	51 (66.2%)	63.6%	1.20	(1.14-1.27)
	High	26 (33.8%)	36.4%	9.24	(5.37-15.86)
COYV_1994	Low	116 (95.9%)	94.6%	2.93	(2.52-3.49)
	High	5 (4.1%)	5.4%	71.45	(15.95-545.40)
283V_1993	Low	47 (61.0%)	56.2%	1.54	(1.37-1.73)
	High	30 (39.0%)	43.8%	17.84	(8.63-36.37)
8V_2003	Low	28 (36.4%)	33.8%	1.36	(1.08-1.70)
	High	49 (63.6%)	66.2%	7.88	(5.05-12.22)
BRDV_1975	Low	36 (54.5%)	59.2%	1.76	(1.42-2.19)
	High	30 (45.4%)	40.8%	42.34	(20.09-89.57)
89V_2003	Low	48 (62.3%)	57.1%	1.47	(1.18-1.80)
	High	29 (37.7%)	42.9%	16.93	(6.81-39.56)
AMDV_1994	Low	29 (20.1%)	17.7%	1.14	(1.01-1.27)
	Interme	99 (68.8%)	66.8%	4.55	(3.39-6.00)
	High	16 (11.1%)	15.5%	34.79	(6.29-182.37)

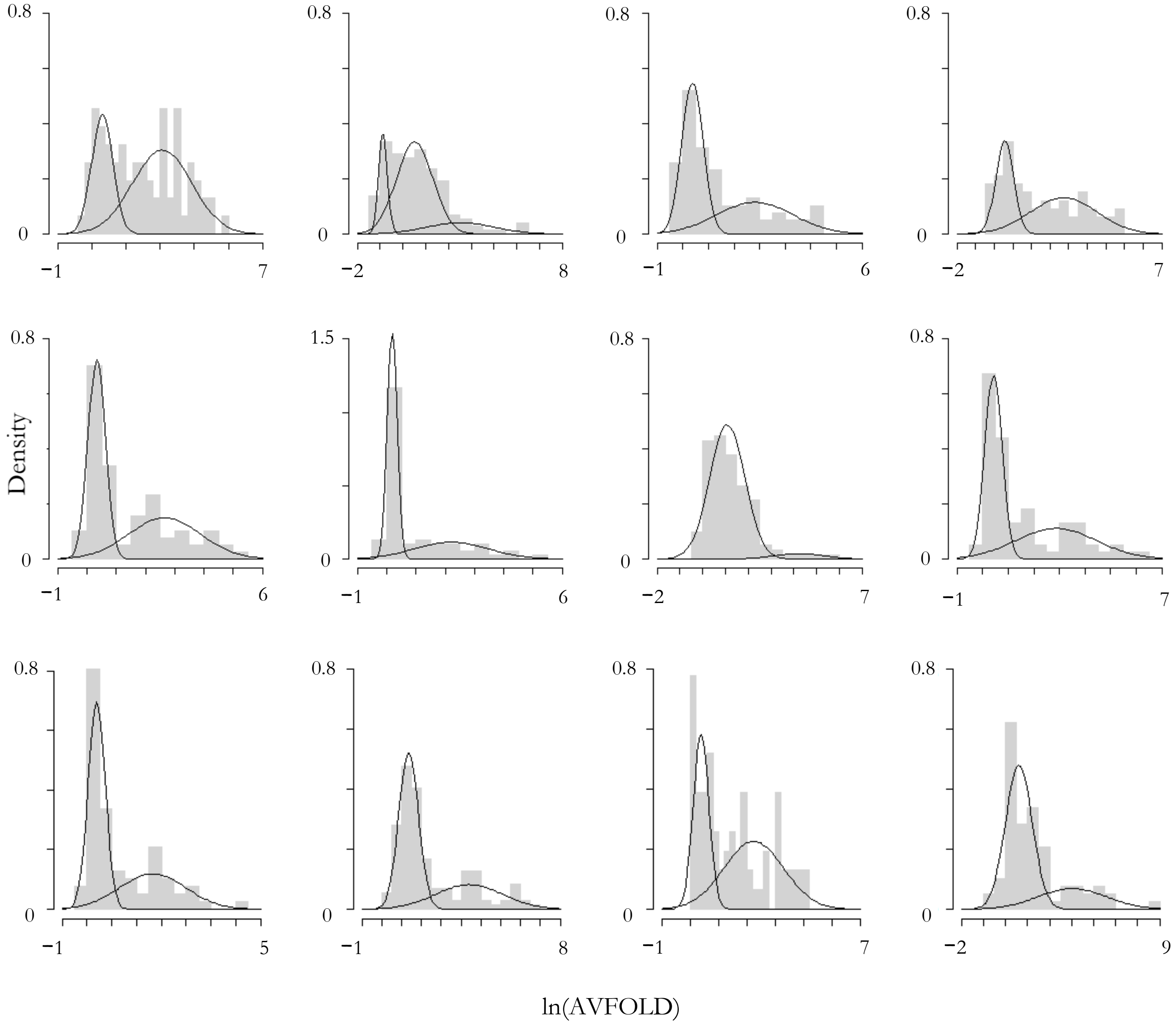

Fig. 2 The density functions of the components of the mixture model with lowest BIC and the total distribution of the natural logarithm of the AVFOLD for each strain (left to right: MDNV_1992, CBNV_1994, 116V_2003, 53V_2003, 492V_1994, 243V_2003, COYV_1994, 283V_1993, 8V_2003, AMDV_1994, 89V_2003 and BRDV_1975).

Seroprevalence estimates

The overall percentage of seropositive individuals varied with strain (range = 4.1 – 79.9 %; Table 1). There was a general increase in the percentage of seropositive individuals with age across all strains (Fig 3a). However, a decrease in seropositivity was observed for the majority of strains in 17-21 age category (116V_2003, 243V_2003, 283V_1993, 53V_2003, AMDV_1994, BRDV_1975, CBNV_1994, COYV_1994 and MDNV_1992) and for two other strains in 12-16 age category (8V_2003 and 492V_1994; Fig. 3a).

FOI estimates

The mean strain-specific FOI estimate was 0.16 year^{-1} (range = 0.005 - 0.29 year^{-1}). The mean age-specific FOI estimate was 0.21 year^{-1} for pre-breeding birds (range = 0.01 – 0.51 year^{-1}) and 0.08 year^{-1} for breeding birds (range = -0.07 – 0.24 year^{-1}). The highest FOI estimates were all pre-breeding FOI estimates (for the strains AMDV_1994, 116V_2003, 8V_2003, BRDV_1975 and 492V) and for the majority of strains (*n* = 8) the FOI before first breeding was greater than the FOI after first breeding. For the remaining 4 strains, the breeding FOI estimate was greater than the pre-breeding FOI estimate. Some breeding FOI estimates (for the strains 116V_2003, AMDV_1994 and COYV_1994) were found to be negative (Fig. 3b).

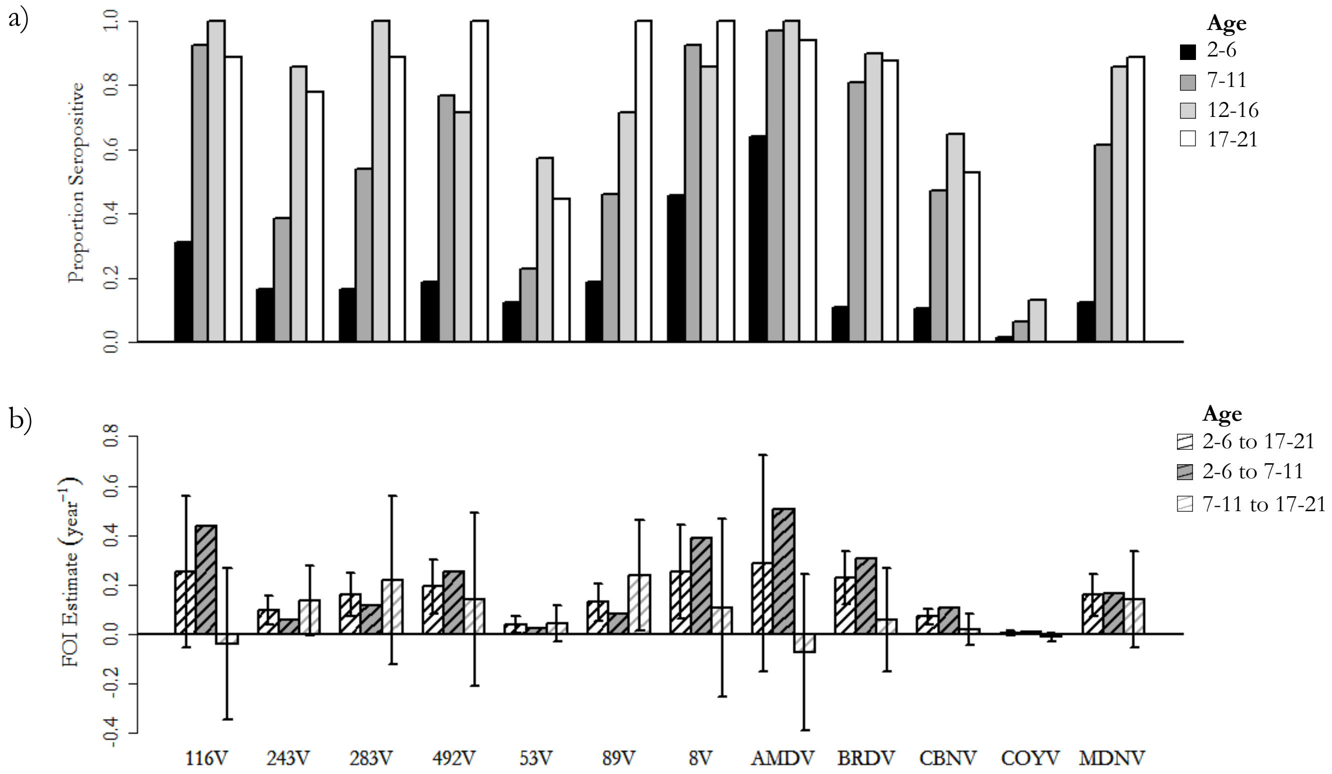

Fig. 3 a) Age and strain-specific seroprevalence estimates, and b) FOI estimates (year^{-1}; error bars indicate 95% confidence intervals).

Discussion

In this study, I provide improved estimates of strain and age-specific seroprevalence and FOI for 12 GIV strains circulating within a guillemot colony on the Isle of May, Scotland. The estimates support breeding-status-dependent seroprevalence and FOI, with a higher pre-breeding than breeding seroprevalence across all strains, and a higher pre-breeding than breeding FOI for 8 out of 12 strains. The five highest FOI estimates were all pre-breeding FOI estimates (for the strains AMDV_1994, 116V_2003, 8V_2003, BRDV_1975 and 492V) ranging from 0.25 to 0.44 year^{-1}, compared with the breeding FOI estimates for the same strains which ranged from -0.04 to 0.14 year^{-1}. The average pre-breeding FOI (0.21 year^{-1}) was almost three times higher than the average breeding FOI. This evidence indicates that transmission rates between guillemots prior to first breeding are generally higher than between guillemots after first breeding. This agrees with previous observations of higher virus prevalence in ticks feeding on pre-breeding guillemots than breeding guillemots (Nunn *et al.* 2006a). Breeding FOI estimates were higher than pre-breeding FOI estimates for 4 out of 12 strains. However, the magnitude of these estimates was relatively low compared to other pre-breeding FOI estimates, and the 95% confidence intervals associated with these estimates included the pre-breeding FOI estimates for the same strains.

The percentage of susceptible individuals was shown to increase with age for some strains, particularly between older age categories. This led to some negative FOI estimates (for strains 116V_2003, AMDV_1994, COYV_1994). These negative estimates may be due to violations of either the assumption of permanent immunity or the assumption of negligible mortality, or may be due to temporal variation in strain abundance. This has been shown in other wildlife parasites (Lachish *et al.* 2011) and Becker (1989) points out that for a single cross-sectional survey, any effects of age and time on FOI are confounded. Both the overall strain-specific seroprevalence and strain-specific FOI estimates I present here also range widely, the latter from -0.07 to 0.51 year^{-1}, emphasising the need, both here and in other studies, to understand strain specific differences in transmission, particularly in relation to varying levels of age-related acquired immunity (Anderson & Trewhella 1985; Anderson & May 1991) as well as pathogenicity (Gupta, Ferguson & Anderson 1998). Strain structure has been shown to be critical when estimating epidemiological parameters for endemic infections such as malaria (Buckee, Bull & Gupta 2009) and dengue (Ferguson *et al.* 1999), but until recently (Duneau *et al.* 2008) has received rather little attention in the seabird literature.

Although it is possible that GIV varies in strain abundance in the longer-term, I believe this is unlikely in the shorter-term (≤ 3 years) as this is the length of the average lifespan of *I. uriae* (Barton *et al.* 1996), the vector of GIV. Larger, longitudinal surveys of the serological responses of individual guillemots to all strains of GIV could investigate these temporal patterns, as well as spatial and sex-specific variation in FOI, using different statistical methods. However, I believe critical experiments are first needed to understand the cross-reactivity of the neutralising antibody response and hence to identify serotypes of GIV. Further research is also needed to investigate the pathogenic effects of GIV (if any) and the length of antibody protection to the virus.

Overall, I believe that my approach provides new insights into the epidemiology of GIV. My estimates of FOI provide a sound basis for improving estimates of GIV transmission rates, and further investigating the ecological drivers of GIV spread. If pre-breeding immature birds are the major source of infection, as we propose, then their movements and recruitment patterns are likely to affect the overall spread of GIV, and pose a significant risk to seronegative breeders, for whom the cost of primary infection is likely to be higher, given the already high energetic cost of breeding (Bonneaud *et al.* 2003). Furthermore, acute decreases in attendance of breeding guillemots (Ashbrook *et al.* 2010) and chronic declines in their overall numbers (JNCC 2014; Lahoz-Monfort *et al.* 2014) may change these movement and recruitment patterns. I also believe that principles derived from our understanding of this study system may be applied more broadly to other wildlife systems to better understand parasite dynamics.

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Modelling the spread of an endemic tick-borne virus, *Great Island virus*, within a common guillemot (*Uria aalge*) breeding colony: estimating current and future infection risk for breeding individuals of a species of conservation importance

Abstract

Most wildlife parasites are endemic and have the potential to have important consequences for the conservation of their hosts. However, little is known about effects of host spatial organisation on the dynamics of these parasites. This is largely due to the lack of relevant field data, which makes it difficult to accurately estimate critical epidemiological parameters. Here, I re-visit the model derived in Chapter 2, for the spread of a tick-borne virus, *Great Island virus* (GIV), within a socio-spatially structured common guillemot (*Uria aalge*) breeding colony. I use improved estimates of the GIV force of infection (Chapter 5) and the first quantitative field estimates of the spatial attendance patterns of pre-breeding common guillemots (Chapter 4) to improve model predictions, and to answer two specific questions: a) does current spatial variation in the attendance patterns of pre-breeding guillemots in breeding areas lead to spatial heterogeneity in infection risk within the colony?, and b) what is the potential future infection risk for the breeding population following socio-spatial disruption? I show evidence for substantial spatial heterogeneity in infection risk within the colony, with an almost two-fold (69%) increase in the infection risk for breeding birds in different subcolonies. This suggests that breeding may currently be less costly in some areas of the colony than others. My model also predicts a potential increase of up to more than two-fold (121%) in the infection risk for breeding individuals following disruption. Although little is known about the effects of GIV on its guillemot host, other pathogenic parasites are found in seabird colonies, for which an increase in infection risk could have important consequences for both the focal colony, as well as the UK guillemot population, by altering guillemot abundance and/or productivity. My findings are also relevant to other spatially structured wildlife populations.

Introduction

The spatial structuring of wildlife hosts, either as a result of environmental heterogeneity or social and behavioural interactions (Kerr & Currie 1995; Ciuti *et al.* 2012), has been linked to changes in parasite dynamics, but studies have largely focused on epidemic parasites, with more obvious effects on their hosts such as phocine (Brown *et al.* 2008; Klepac *et al.* 2009), and canine distemper virus (Craft *et al.* 2011) and avian influenza (Clancy, O'Callaghan & Kelly 2006) . However, most wildlife parasites are endemic, and characterised by their persistence and relatively constant prevalence through time (Anderson & May 1979). Although more abundant, the effects of host spatial structuring on the dynamics of these parasites is less well understood (Brown *et al.* 2008; Watts *et al.* 2009). However, endemic parasites have the potential to have important consequences for the conservation of their hosts, with some spatial structures of host populations potentially being more amenable to disease control than others (Hess 1996; Woodroffe 1999; Fulford, Roberts & Heesterbeek 2002). The lack of relevant field data often makes it difficult to accurately estimate critical epidemiological parameters and hence to address these questions. However, evidence suggests significant anthropogenic pressures on wildlife populations (Kerr & Currie 1995; Ciuti *et al.* 2012), which could have consequences for their natural organisations and hence associated parasite dynamics (Hess 1996; Woodroffe 1999; Fulford *et al.* 2002).

Here, I re-visit the epidemiological model derived in Chapter 2 for the spread *Great Island virus* (GIV) through a large spatially-structured common guillemot (*Uria aalge*) colony, and use improved estimates of GIV force of infection (Chapter 5) and the first quantitative field estimates of the spatial attendance patterns of pre-breeding common guillemots (Chapter 4) to answer two questions: a) does the current spatial variation in the attendance patterns of pre-breeding birds in breeding areas lead to spatial heterogeneity in infection risk within the colony?, and b) what is the potential future infection risk for the breeding population following social disruption?

Model of the guillemot-tick-virus system

I use a simple two-patch ordinary differential equation (ODE) model to describe the guillemot-tick-virus system, parameterised for the Isle of May guillemot colony, Scotland and solved in R 3.0.2 (R Core Team 2013), the details of which are given in Chapter 2. Regardless of my strain-specific epidemiological parameter estimates in Chapter 5, for the sake of simplicity, I model the spread of a single average virus strain. Although the potential for significant independent movement of the seabird tick (*Ixodes uriae*) is suggested in Chapter 3, experiments were conducted in an artificial environment, and further studies in the field are required to fully understand the infection risk from independently moving ticks. Therefore, host-mediated tick dispersal is assumed to be the only route for virus transmission, ticks are excluded from this model and direct transmission dynamics are again assumed, as in Chapter 2. Although this is a significant simplification of reality, I have shown that the long-term equilibrium behaviour of this model remains informative, as well as easily interpretable (Chapter 2).

I model total guillemot numbers throughout, as this is the unit of conservation importance. Modelling subcolony-specific guillemot numbers also had no qualitative effect on my subcolony specific conclusions. As in Chapter 2, I present equilibrium guillemot numbers (marked with an asterisk in equations 1 and 2), and assume that the force of infection (FOI), or per susceptible risk of infection, within a patch (λ_B = breeder force of infection, λ_P = pre-breeder force of infection), is composed of two terms, a within-patch transmission term and a between-patch transmission term (see Chapter 2). I also describe differences in the infection risk for breeding birds, and present these as fold and/or percentage increases.

Parameter estimation

In Chapter 2, I show that my model is highly sensitive to the between-patch transmission rate and suggest that between-patch transmission is currently negligible within the colony, having little effect on GIV dynamics. However, it was not possible to accurately estimate this key parameter. The spatial attendance patterns of pre-breeding common guillemots (Chapter 4) suggest that only half of individuals ever move into breeding areas (n = 25, or 53 %), and spend between 10% and 51% of their time in these areas, depending on the subcolony. Although these values do not provide direct estimates of the between-patch transmission rate, I use them to estimate the relative potential for the transmission of infection within and between patches (defined as the scaling factor, z; Table 1).

I use an initial value of 0.16 for z, meaning that each pre-breeding bird spends 16% of its time in the breeder patch. I assume that this is equivalent to 53% of birds spending an average of 30% of their time in this patch ($0.53 \times 0.30 = 0.16$; Chapter 4). I vary the value of z to represent current variation in attendance between subcolonies ($z = 0.05 - 0.27$; Chapter 4). I then double the maximum current value of z for a subcolony, and explore the range $z = 0.27 - 0.54$, to investigate potential future scenarios. Although I assume that all pre-breeding individuals are equal in this model, in the real world, a doubling of z, could be equivalent to either a doubling of the proportion of guillemots entering breeding areas, or a doubling of the proportion of time that the guillemots already entering breeding areas spend there.

As well as constraining the transmission terms with the scaling factor, z, I use improved estimates of the most up-to-date FOI within the colony (for the period 1993-1996; Chapter 5) to estimate the absolute transmission rate, β, assuming that they are roughly representative of the present day. Although evidence suggests a higher transmission rate within breeding areas (Nunn *et al.* 2006), leading to a system which is more sensitive to changes in the between-patch transmission rate (Chapter 2), for the sake of simplicity, I assume that all β parameters are equal ($\beta_{BB} = \beta_{PP} = \beta_{BP} = \beta_{PB} = 9 \times 10^{-6}$ per day; Table 1). All other parameter values used are summarised in Table 1.

Table 1. Parameters used in simulations and their provenance.

Symbol	Value (per day)	Description and reference
b	1.05×10^{-3}	Guillemot birth rate, from Harris & Wanless (1988).
s	Unknown	Density dependent crowding parameter of guillemot. Estimated in model (2×10^{-8}).
μ_P	2.7×10^{-4}	Natural death rate of pre-breeding guillemots, estimated from Reynolds *et al.* (2009).
μ_B	1.4×10^{-4}	Natural death rate of breeding guillemots, from Harris & Wanless (1995).
r	4.6×10^{-4}	Recruitment rate of pre-breeding guillemots into breeding population, estimated from Crespin *et al.* (2006).
γ	3.3×10^{-2}	Guillemot recovery rate, assumed by Nunn *et al.* (2006).
α_P	2.9×10^{-4}	Pre-breeding guillemot viral induced death rate, assumed by Nunn *et al.* 2006b.
α_B	4.2×10^{-4}	Breeding guillemot viral induced death rate, assumed by Nunn *et al.* (2006).
all β	Unknown	Unscaled probability of viral transmission between guillemots via ticks, estimated in the model using estimates from Chapter 5 (9×10^{-6}).
z	Varied	Scaling factor, or relative potential to transmit infection between guillemots via ticks, within and between patches, from Chapter 4 (initially 0.05 – 0.27 and denoted in figure legends).

Model analysis

Does the current subcolony-specific variation in the attendance patterns of pre-breeding guillemots in breeding areas lead to spatial heterogeneity in infection risk within the colony?

My model suggests an average current between-patch transmission rate ($z\beta_{PB}$ and $z\beta_{BP}$) of 1.4×10^{-6} per day and an average within-patch transmission rate ($(1\text{-}z)\beta_{PP}$ and $(1\text{-}z)\beta_{BB}$) of 7.6×10^{-6} per day. This is equivalent to an individual being over five times more likely to be infected within a patch than between patches. Simulated FOI values were within the absolute and relative range observed during the period 1993-1996 (pre-breeder patch = 1.4×10^{-3} per day, breeder patch = 6.4×10^{-4} per day; fold-difference between patches = 2.2). Current subcolony-specific variation in the attendance patterns of pre-breeding guillemots (i.e. values of z) was shown to almost double the risk of infection for breeding birds (total increase of 69%; see solid line in Fig. 1).

What is the potential future infection risk for the breeding population following social disruption?

The rate of increase in the FOI for breeding birds was found to decrease with increasing values of z, i.e. the relative potential for the transmission of infection within and between patches (Fig. 1). It was shown to increase by 31%, following a doubling of the maximum subcolony-specific value (0.27 - 0.54; see dashed line in Fig. 1). This results in a 61% increase in breeder infection risk from the average subcolony-specific value of z. However, the increase in risk for subcolonies with low current values of z could be over two-fold (121%). I do not present pre-breeder FOI estimates here, but they were observed to decrease within these limits, as expected (see Chapter 2, Eqn. 3).

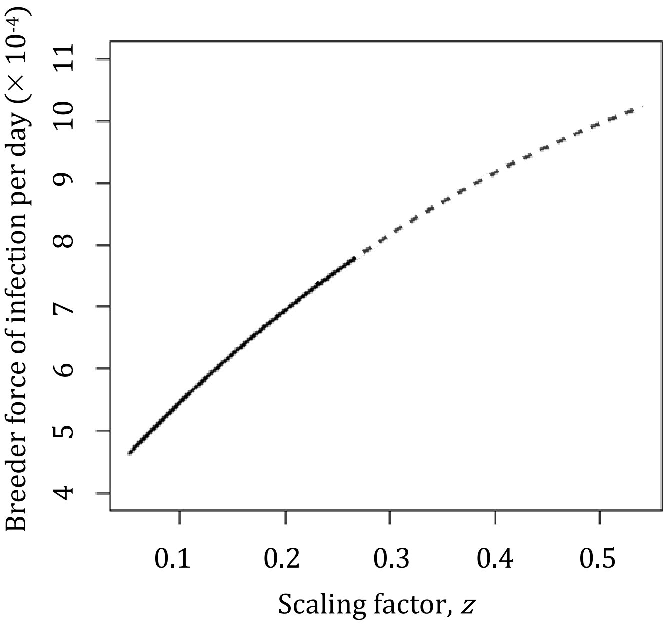

Fig. 1 Breeder force of infection with increasing attendance of pre-breeding guillemots in breeding areas (scaling factor, *z*). Observed subcolony specific values are indicated with a solid black line, and possible future values with a dashed line.

Discussion

My model suggests spatial heterogeneity in infection risk in a seabird colony, with breeding being less costly in some areas than in others. It also predicts a substantial increase in the risk of infection for breeding individuals from endemic parasites, such as GIV, resulting from changes in the socio-spatial structuring of the colony.

Spatial heterogeneity in current risk of infection for breeding birds

Guillemot colonies are characterised by high densities of birds and high levels of competition for breeding sites (Cramp & Simmons 1985). Harris *et al.* (1997) suggest that the quality of a site may be defined by the number of neighbours and cliff walls, type of site, slope where the egg is incubated and distance from the top of the cliff. Evidence also suggests a role for conspecific cues of breeding success in decisions relative to breeding site selection. My results suggest that infection risk may be a further factor influencing the quality of a site, as mounting an immune response can be energetically costly, especially when attempting to breed. Spatial heterogeneity in infection risk has also been found in other wildlife systems (Gauthier-Clerc *et al.* 1999; Brown & Brown 2005; Real & Biek 2007; Jones *et al.* 2011; Lachish *et al.* 2011).

Potential future increases in infection risk for breeding birds

Seabird populations are globally threatened, with nearly half of seabird species experiencing population declines, mainly as a result of commercial fishing, pollution, alien invasive predators, habitat degradation and human disturbance (Croxall *et al.* 2012). Recently observed social disruption in guillemot colonies in the North Sea (Ashbrook *et al.* 2010; JNCC 2014) may increase in frequency in the future, making it important to understand the consequences for parasite dynamics. My results suggest potential future increases in infection risk, which could have serious consequences for both the focal colony, as well as the UK guillemot population by altering guillemot abundance and/or productivity. However, further work is needed to quantify the effects of GIV and other naturally circulating tick-borne viruses on their seabird host (Dietrich, Gómez-Díaz & McCoy 2011; Chambert *et al.* 2012; Arnal *et al.* 2014).

Although my study has focused on the spread of a parasite through a spatially heterogeneous guillemot colony, many other wildlife systems are also spatially aggregated, either as a result of environmental heterogeneity or social and behavioural interactions. For example, racoon populations, which are infected by the rabies virus, are highly structured as a result of township and river boundaries (Smith *et al.* 2002; Russell, Real & Smith 2006; Real & Biek 2007). Territorial species, like the Serengeti lion and European badger are also highly spatially structured with implications for the transmission of canine distemper virus (Craft *et al.* 2011) and bovine TB respectively (Donnelly *et al.* 2006; Carter *et al.* 2007; Woodroffe *et al.* 2009). Many wildlife systems also demonstrate broad-scale variation in susceptibility (Hudson 1986; Hudson *et al.* 1995; Brown *et al.* 2001; Burthe *et al.* 2006). Therefore, I suggest that endemic parasites may be a major potential threat to worldwide wildlife populations. My results highlight and add to the existing literature (Hess 1996; Woodroffe 1999; Fulford *et al.* 2002) on the importance of considering these parasite dynamics when planning conservation actions.

Future modelling

I suggest that future models could further our understanding of the guillemot-tick-virus system. For example, estimates of the spatial attendance patterns of pre-breeding guillemots suggest that only some pre-breeding birds ever enter breeding areas (Chapter 4). Future models could account for this variation within the pre-breeder patch by including two types of pre-breeding birds: those that move and those that do not. Of course, the latter will not contribute to the transmission of infection between patches. The seasonal processes governing the dynamics of GIV, including the seasonal presence of guillemots on land and feeding activity of *I. uriae* (Barton *et al.* 1996), may also be better captured by a seasonal, discrete-time model. Other studies have shown the importance of seasonal process such as these for parasite dynamics, and have applied a similar approach (Nonaka, Ebel & Wearing 2010).

A model with explicit tick dynamics could be developed, similar to those already described in the literature (Hudson *et al.* 1995; O'Callaghan *et al.* 1998; Gaff & Gross 2007; Nonaka *et al.* 2010; Bolzoni *et al.* 2012). The same fundamental structure could be applied, but with four extra state variables: susceptible and infected ticks in the pre-breeder patch and susceptible and infected ticks in the breeder patch. Ticks could be restricted to their patches except for some bird-mediated transferal between patches, the probability of which could be dependent on the proportion of time that pre-breeding birds spend in breeding areas (equivalent to the scaling factor, z, in the model presented here). Finally, in Chapter 5, I highlight the importance of considering strain-specific differences in GIV dynamics. Previous studies include similar strain-specific dynamics in models of other wildlife host-parasite systems (e.g. Buckee, Bull & Gupta, 2009; Ferguson Donnelly, & Anderson, 1999). A similar approach could be applied to future models of GIV spread.

Conclusion

Nevertheless, I have demonstrated here that a simple two-patch ODE model can be used to estimate current and future infection risk from a naturally circulating parasite for a species of conservation importance in the UK, the common guillemot.

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General Discussion

Many parasites have been isolated in seabird colonies (e.g. Coffee *et al.* 2010; Young & VanderWerf 2008; Lebarbenchon *et al.* 2015; Nuttall *et al.* 1981; Arnal *et al.* 2014; Österblom *et al.* 2004; Chambert *et al.* 2012). However, very little is known about the effects of spatial structure, a common feature among seabird colonies, on parasite dynamics. In this thesis, I set out to explore this relationship, using a tick-borne virus, *Great Island virus*, found in a large common guillemot colony bordering the North Sea as a model system. Here, I summarise the findings from each of my chapters, and discuss both their limitations and their potential future applications.

In the general introduction I briefly reviewed the literature on the biology of each of the components of this system (guillemot, tick and virus), as well as previous efforts to understand the importance of host spatial structure for parasite dynamics in other wildlife systems. I introduced the threat to guillemot spatial structuring from changing breeding population dynamics bordering the North Sea (Ashbrook *et al.* 2010; JNCC 2014), as well as the herd immunity hypothesis. That is, that in an undisturbed colony, a large proportion of immune individuals leads to a lower risk of infection in breeding areas.

How important is the spatial structure of the guillemot colony for GIV dynamics?

The importance of these two elements was demonstrated in Chapter 2, where a simple epidemiological model of the system confirmed a lower risk of infection in breeding areas under existing conditions. However, it also highlighted the sensitivity of the system to changes in the rate of transmission between two spatially distinct patches, representing pre-breeding and breeding areas of the colony.

As well as shedding light on the guillemot-tick-virus system, this model could be applied to other seabird tick-borne infections. One of particular interest in the literature due to its zoonotic nature, *Borrelia bugdorferi* sensu latu, the agent of Lyme disease, is also carried by *I. uriae* (Olsen *et al.* 1993, 1995). Previous studies, suggest spatial variation in anti-*Borrelia* antibodies within seabird and other wildlife populations (Kurtenbach *et al.* 1994; Gauthier-Clerc *et al.* 1999; Travnicek *et al.* 2002; Stefancikova *et al.* 2004; Staszewski, McCoy & Boulinier 2008). This variation could lead to similar dynamics as observed in the guillemot-tick-virus system, with changes in the amount of mixing between individuals inhabiting different areas, leading to changes in the risk of infection within the population. Future work could also consider the importance of seabird parasite communities in shaping these dynamics (e.g. Telfer *et al.* 2010).

However, a caveat to my modelling approach is that explicit tick dynamics were not included. As I explored in the introduction, vector-borne transmission is usually modelled in a different way to direct-transmission (Anderson & May 1991). Randolph (1998) also argues that differences in the biological characteristic of ticks, as compared to insects, leads to further difference in transmission potential. Although *I. uriae* has a complex three-host life cycle (Barton *et al.* 1996), I feel that my simplified approach was justified as it gave easily interpretable predictions of broader-scale GIV dynamics. Future studies could investigate the effects of tick dynamics on finer-scale GIV dynamics to provide more precise predictions. Previous studies highlight the importance of tick parameters in driving tick-borne pathogen transmission, including the rate of co-feeding transmission (e.g. Nonaka, Ebel & Wearing 2010; Norman *et al.* 2004), as well as seasonal tick population dynamics (e.g. Dobson, Finnie & Randolph, 2011; Randolph *et al.* 1999; Randolph *et al.* 2002). However, they are unlikely to change the major conclusion of this work; that spatial structure has an important role to play in the guillemot-tick-virus system, as this is consistent with previous studies. For example, Watts *et al.* (2009) modelled the transmission of louping ill virus in a two-patch framework with tick dynamics. They found that the inclusion of spatial variation and movement of a host between patches increased the difference in louping ill prevalence, and also resulted in more accurate prediction of the disease vector's abundance. Similar conclusions relating to the importance of spatial heterogeneity were made by Jones *et al.* (2011) for the same system, as well as for TBE prevalence in deer (Pugliese & Rosa 2008).

The lack of tick dynamics in this modelling approach also means that it can be readily applied to other wildlife parasites, including those that are directly-transmitted. As in the common guillemot, immunity is often acquired with age (Anderson & Trewhella 1985; Anderson & May 1991; Nielsen *et al.* 2002; Long *et al.* 2010). Furthermore, other age-segregated host species are found throughout the animal kingdom; include penguins (Pelletier *et al.* 2014), fish (Tack 1974; ØRnulf & Bror 1983; L'Abée-Lund *et al.* 1993), ungulates (Cransac *et al.* 1998; Bon *et al.* 2001), whales (Cosens & Blouw 2003), rodents (Jackson *et al.* 2011), other bird species (Jaramillo 1993; Catry *et al.* 2004), seals (Godsell 1988) and even nematodes (Traunspurger & Drews 1996). However, sex difference in parasite infection and hence immunity have also been observed (Poulin 1996; Zuk & McKean 1996) and sexual segregation is equally as widespread, particularly in sexually dimorphic species, including most ungulates (Geist 1971; Main, Weckerly & Bleich 1996), chimpanzees, monkeys, coatis and some macropods (Nowak 1991), fish (ØRnulf & Bror 1983) and birds (Catry *et al.* 2004). Possible reasons for age- and sex-segregation, include asynchrony in activities (e.g. Conradt 1998) and differences in habitat preference (e.g. Conradt, Clutton-Brock & Guinness, 2000; L'Abée-Lund *et al.* 1993). An interesting avenue for future research would be to develop strategic models, based on the approach presented in this thesis, to apply to these wildlife host-parasite systems. I argue that spatial structure and herd immunity have the potential to play an important role in shaping wildlife parasite dynamics.

An avenue that I was unable to explore within this thesis due to lack of time, was the effect of other parameters on model predictions. I used the most up-to-date, average parameter values from the literature to give the most realistic model predictions (Harris & Wanless 1988, 1995; Crespin *et al.* 2006; Reynolds *et al.* 2009). However, changes to demographic parameters such as the recruitment rate or birth rate could also have consequences for the level of immunity within the colony. For example, if the birth rate was to increase, then there would be a larger population of susceptible pre-breeding birds, potentially increasing GIV circulation in the pre-breeding area. On the other hand, a faster rate of recruitment, equivalent to guillemots breeding at an earlier age, may increase the number of susceptible individuals in the breeding area, as younger individuals are less likely to have developed immunity to the virus (e.g. Begon *et al.* 2009). This could increase the prevalence of GIV in the breeding area, in a similar way to higher levels of prospecting by pre-breeding birds. Some epidemiological parameters were estimated from the literature on related viruses, due to the lack of GIV-specific literature, including the GIV infectious period (MacLachlan *et al.* 1992; Koumbati *et al.* 1999). The effect of GIV on its guillemot host and the persistence of GIV neutralising antibodies were estimated from a preliminary study (Nunn *et al.* 2006b). An important avenue for future research, will be to accurately estimate these parameters. The latter two, for example, could be estimated using statistical methods already applied to a similar system, *Borrelia* infection in black-legged kittiwakes (Staszewski *et al.* 2007; Chambert *et al.* 2012).

What role does independent tick dispersal play in driving GIV transmission between different areas of the colony?

In Chapter 3, I presented novel insights into the independent dispersal potential of *I. uriae.* More specifically, I provided quantitative estimates of its host-seeking rate of movement, which seem to suggest the *I. uriae* has the potential to move significant distances independently. However, I warned against making any conclusions before conducting experiments in the field, particularly as the majority of seabird literature suggests that *I. uriae* has limited mobility (Karpovich 1970; Boulinier 1995; McCoy, Tirard & Michalakis 2003; Muzaffar & Jones 2007). For example, ticks could be excluded at varying distances from carbon dioxide traps. In this way, it would be possible to identify the maximum distance at which good tick numbers are still observed.

Alternatively, the host-seeking movements of *I. uriae* could be investigated in a semi-natural environment, by introducing natural substrate materials and structures (Randolph & Storey 1999). Although my unpublished findings suggest that dry ice, which has been used to attract other species of questing ticks in the field (Koch & McNew 1981; Kinzer, Presley & Hair 1990), may not work for *I. uriae*, CO_2 gas cylinders with regulators could be implemented instead. If these larger dispersal distances were confirmed, they could have significant consequences for understanding the phylogeographical patterns of *I. uriae* (McCoy *et al.* 2005; Gomez-Diaz *et al.* 2012; Dietrich *et al.* 2014), as well as the circulation of infection in seabird colonies.

What role does guillemot-mediated tick dispersal play in driving GIV transmission between different areas of the colony?

My analysis of the spatial attendance patterns of pre-breeding guillemots in Chapter 4, provides novel insights into the potential risk of guillemot-mediated tick dispersal within the colony. I highlight the importance of individual pre-breeding guillemots, as well as both spatial and temporal patterns in attendance. Although a large number of individuals were followed in this study, an automated approach could be applied to similar studies in the future. For example, a theodolite could be used to more accurately estimate distances within subcolonies, and map the pre-breeding and breeding areas. A video camera could also be mounted to a tripod and placed above subcolonies, with video-tracking software being used at a later stage to continuously follow particular pre-breeding individuals through the subcolony (Burghardt, Calic & Thomas 2004; Bluff & Rutz 2008). A similar automated approach could also be applied to collect tick movement data in Chapter 3, to avoid any bias associated with the presence of an observer (Noldus, Spink & Tegelenbosch 2002).

In this chapter, I investigated the effect of breeding guillemot attendance on pre-breeding guillemot spatial attendance within one season, as a surrogate for changes in the breeding colony as a whole. Although I did not find a significant relationship as expected, in the general introduction to this thesis, I discussed the differences in colony dynamics both within and beyond the UK, and presented two examples of colonies with increasing populations (Barrett 2010; Meade *et al.* 2013). An interesting avenue for future research would be to record the spatial attendance patterns of pre-breeding guillemots at a range of different colonies, varying in their population dynamics. A natural experiment such as this would allow the relationship between breeding abundance and pre-breeding spatial attendance to be tested more systematically.

What is the current risk of GIV infection for guillemots and does this vary with strain?

In Chapter 5, I presented estimates for the risk of GIV infection within the colony. As well as confirming the difference in risk for breeding and pre-breeding guillemots, this analysis revealed the diversity in risk associated with different strains of the virus. These values are critical for understanding a highly strain-diverse virus such as GIV, and could be used to parameterise a multi-strain model of the guillemot-tick-virus system. Previous studies have demonstrated how strain structure evolves (Gupta, Ferguson & Anderson 1998), is maintained (Gupta *et al.* 1996) and influences other host-parasite systems (Ferguson, Donnelly & Anderson 1999; Duneau *et al.* 2008; Buckee, Bull & Gupta 2009). However, critical experiments are still needed to understand cross-reactivity in GIV antibodies, either in the form of immunoprecipitation techniques (e.g. as in: Mertens *et al.* 1989) or antigenic cartography (Smith *et al.* 2004). Even more accurate estimates of the risk of infection could also be derived from larger datasets, using nonparametric, semiparametric and Hierarchical Bayesian approaches (Hens *et al.* 2012).

What is the potential future risk of infection from GIV for guillemots, as a result of changes to the spatial structure of the colony?

Taken together, these results have important implications for future guillemot populations. I demonstrate this in Chapter 6, where I input my new parameter estimates into my existing guillemot-tick-virus model to provide potential future scenarios for the risk of GIV infection within the colony, particularly within the breeding areas. I show evidence for a substantial increase in risk associated with current variation in prospecting of pre-breeding guillemots and, within realistic limits, beyond this. These results may have implications for the global circulation of infection, with disturbed colonies potentially acting as hotspots (foci) of infection. Colonies such as this, could pose a higher risk not only for wildlife populations, but in the case of zoonotic infections, for human populations (Jones *et al.* 2008; Paull *et al.* 2012).

The results of this chapter may also have implications for the conservation of guillemots and other host species. Although much conservation work is focussed on maintaining links between subpopulations to avoid the unstable dynamics associated with fragmentation (MacArthur & Wilson 1967; Hanski & Simberloff 1997), this work suggests that maintaining spatial structure in populations with varying levels of immunity may be critical for the conservation of some species. The natural spatial structuring of wildlife population should also be carefully considered when attempting to control a parasite, as the removal of individuals can have significant and unexpected consequences for parasite dynamics, as demonstrated in the case of bovine tuberculosis in cattle and badgers (e.g. Smith, McDonald & Wilkinson, 2012). Perhaps less conventionally, my results could also have implications for the conservation of GIV and other parasites, by furthering our understanding of the impacts of changing host spatial structure on parasite abundance (Gómez & Nichols 2013).

Furthermore, anthropogenic pressures on wildlife populations are increasing (IUCN 2015). The IPCC has predicted that sea surface temperatures will continue to increase globally throughout the 21st century, along with the frequency of extreme weather events (Intergovernmental Panel on Climate Change 2014). Previous studies suggest a significant negative relationship between sandeel population numbers, sea surface temperatures and both breeding phenology and productivity (Arnott & Ruxton 2002; Frederiksen *et al.* 2004, 2007). Extreme weather events have also been suggested to interfere with foraging behaviour in the winter (Duff, Harris & Turner 2013), as well as breeding success in the summer (Newell *et al.* 2015). Other pressures on seabird populations include industrial fisheries. For example, in the North Sea, there was concern about the potential effects of the largest single-species fishery on seabird populations (Monaghan 1992; Wright & Begg 1997; Wanless, Harris & Greenstreet 1998; Furness & Tasker 2000; Furness 2002). As a result, the sandeel fishery was closed in 2000, which was associated with beneficial impacts on some seabird populations (Daunt *et al.* 2008).

As I suggested in the general introduction, changes in seabird population dynamics driven by these anthropogenic pressures are likely to lead to changes in their spatial structuring, and particularly in the prospecting behaviour of younger, and less immune individuals. As an example, I mentioned the situation in 2007 and 2008 when Ashbrook *et al.* (2010) observed an acute decline in breeding success on the Isle of May. However, an increase in aggression of breeding guillemots was also observed in this study, leading to many chicks being killed by neighbouring birds (Ashbrook *et al.* 2008). This leads to an important caveat to my work, as fewer but more aggressive breeding birds may be just as, or more, effective at ejecting pre-breeding birds from breeding areas. A lower probability of resighting immature birds within the colony was also observed during the same time period (Lahoz-Monfort *et al.* 2014). As a result, some unoccupied breeding sites may be inaccessible to prospecting pre-breeding guillemots, resulting in a lower risk of infection for breeding birds. Changes in breeding success, as observed in 2007 and 2008, may also lead to long-term changes in infection risk for breeding birds, as a result of missing cohorts (as observed in Chapter 4). However, further studies are needed to shed light on the interactions between pre-breeding and breeding guillemots under changing environments.

Conclusion

By studying the spread of a tick-borne parasite, GIV, in a large common guillemot colony, I have provided improved estimates for critical epidemiological parameters, as well as the modelling framework required to estimate future infection risk in this system; in so doing, I have also provided novel insights into the importance of spatial structuring in driving the dynamics of infection within seabird colonies, globally-important reservoirs of infection, with broader implications for similar wildlife-parasite systems.

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