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REVIEW ARTICLE

Laridae: A neglected reservoir that could play a major role in avian influenza virus epidemiological dynamics

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Abstract

Avian influenza viruses (AIVs) are of great concern worldwide due to their economic impact and the threat they represent to human health. As wild birds are the natural reservoirs of AIVs, understanding AIV dynamics in different avian taxa is essential for deciphering the epidemiological links between wildlife, poultry and humans. To date, only the Anatidae (ducks, geese and swans) have been widely studied. Here, we aim to shed light on the current state of knowledge on AIVs in Laridae (gulls, terns and kittiwakes) versus that in Anatidae by setting forth four fundamental questions: how, when, where and to which host species are AIVs transmitted? First, we describe ecological differences between Laridae and Anatidae and discuss how they may explain observed contrasts in preferential transmission routes and the evolution of specific AIV subtypes. Second, we highlight the dissimilarities in the temporal patterns of AIV shedding between Laridae and Anatidae and address the role that immunity likely plays in shaping these patterns. Third, we underscore that Laridae may be key in promoting intercontinental exchanges of AIVs. Finally, we emphasize the crucial epidemiological position that Laridae occupy between wildlife, domestic birds and humans.

Keywords

Disease reservoir, gull, life history traits, pathogen dispersal, tern

History

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Introduction

Over the last decades, human activities, including animal rearing practices, land use changes, and commercial transport, have given pathogens more opportunities to infect new hosts (Harvell et al., 1999; Lebarbenchon et al., 2008, 2010a; Patz et al., 2004). Such opportunities have led to the emergence of numerous infectious diseases in domestic animals and humans, most of which were originally circulating in wildlife (Daszak et al., 2000; Gortázar et al., 2007). As an example, at the beginning of the century, SARS (Heymann et al., 2004; Peiris et al., 2004; Wang & Eaton, 2007) and Ebola (Leroy et al., 2005; Pourrut et al., 2005) viruses, whose natural hosts are fruit bats, caused disease outbreaks in humans in Asia and Africa. These emergences were eventually linked to anthropogenic activities, in particular the consumption and trade of bushmeat (Wolfe et al., 2005). This example illustrates that understanding pathogen dynamics in natural reservoirs is a critical part of protecting the health of humans and domestic animals.

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Avian influenza viruses (AIVs) provide another good example of pathogens that have emerged from wildlife that are of great concern worldwide due to their economic impacts and the threat they represent to human and animal health (Chen et al., 2005; de Wit & Fouchier, 2008; Ferguson et al., 2005; Li et al., 2004). They are classified into different subtypes of the form HxNy based on their combination of two surface proteins, hemagglutinin (H1-17) and neuraminidase (N1-10), which are important targets for the immune system (Earn et al., 2002; Olsen et al., 2006; Tong et al., 2012; Webster et al., 1992; Zhu et al., 2012). Low pathogenic avian influenza viruses (LPAIVs) naturally circulate in wild birds, in which they generally elicit few or no symptoms. Nevertheless, LPAIV infection may be exacerbated by other infections or environmental conditions and has been shown to sometimes result in delayed migration or weight loss (Latorre-Margalef et al., 2009; van Gils et al., 2007), although these effects may not occur consistently (Arsnoe et al., 2011; Flint et al., 2009). Furthermore, LPAIVs circulating in poultry can evolve into highly pathogenic avian influenza viruses (HPAIVs). One example is H5N1 HPAIV strains, which cause high mortality rates in poultry (Ito et al., 2001; Lebarbenchon et al., 2010a). To date, only H5 and H7 subtypes are known to be able to evolve from low to high pathogenicity (Alexander 2000; Banks et al., 2001; Fouchier et al., 2007). However, the diverse pool of LPAIVs circulating in wild waterbirds has been and remains a source of AIVs that can

potentially evolve into virulent strains specific to domestic species and humans.

Phylogenetic analyses suggest that influenza viruses evolved from an aquatic bird reservoir into host-specific lineages (Horimoto & Kawaoka, 2001). Most combinations of the two surface proteins have been found in Anseriformes and Charadriiformes, which are the natural reservoirs of LPAIVs (Earn et al., 2002; Hurst, 2011; Olsen et al., 2006; Webster et al., 1992), except for H17N10, which was recently discovered in bats (Tong et al., 2012; Zhu et al., 2012). Among Anseriformes, Anatidae (ducks, geese and swans) represent the vast majority of species (172 out of 176), and the taxon includes the main host species for AIVs worldwide: the mallard (*Anas platyrhynchos*). Among Charadriiformes, waders (Charadriidae and Scolopacidae) are distinguished from gulls and terns (Laridae) (IOC, 2012). For various reasons, including the ease with which hunted species can be sampled as well as high contact rates between wild ducks and poultry (Stallknecht & Shane, 1988), most epidemiological studies have focused on AIV circulation in Anatidae; much less attention has been given to Laridae (Figure 1). This difference in the number of studies examining Anatidae and Laridae may also partly be due to the fact that, historically, only a single epizootic in domestic birds has been associated with a gull-specific AIV subtype (i.e. H13; Sivanandan et al., 1991). However, in order to anticipate and respond to the emergence of new viruses, it is essential to thoroughly investigate virus circulation in wild reservoirs without neglecting any key species (Haydon et al., 2002). Thus, Laridae should not be overlooked when studying AIV reservoirs, especially because they can be infected by a large diversity of AIV subtypes, including H5, H7 and H9 (Table 2), which are virus subtypes that have the potential to become highly pathogenic in poultry or that can be zoonotic.

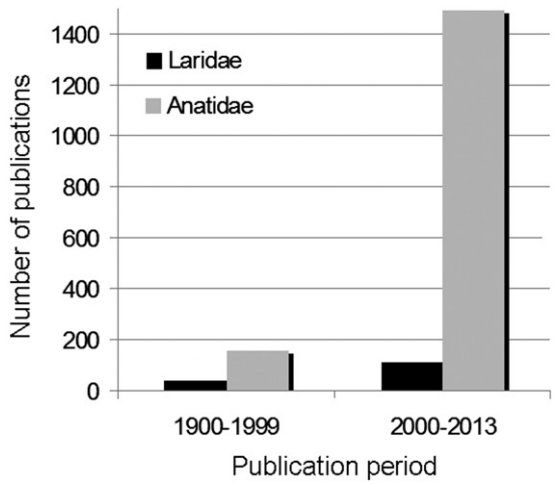

Figure 1. Number of studies that have focused on AIVs in Anatidae versus Laridae from 1899 to July 3, 2013. Research led on Web of Science using respectively the terms: ''Influenza AND gull OR tern OR kittiwake'' and ''Influenza AND duck OR geese OR swan''.

In this review, we will focus on Charadriiformes and most particularly on the Laridae family, which comprises 102 species. We propose that Laridae likely play a major role in avian influenza virus epidemiological dynamics because: (i) AIVs have been detected in Laridae worldwide, whereas AIV incidence in waders varies dramatically across sampling locations (Hanson et al., 2008; Hurt et al., 2006; Munster et al., 2007; Munster & Fouchier, 2009; Stallknecht & Brown, 2007; Winker et al., 2008); (ii) most of the AIV strains that result from a reassortment between American and Eurasian strains have been detected in Laridae, which suggests the taxon plays a potential role in AIV genetic exchanges (e.g. Lebarbenchon et al., 2009; Van Borm et al., 2012; Wille et al., 2011); (iii) the Laridae family includes several opportunistic species that live in close contact with humans following their recent colonization of urban habitats and that have experienced a subsequent demographic explosion (Duhem et al., 2008; Lisnizer et al., 2011; Raven & Coulson, 1997), both of which are factors that could enhance potential public health risks.

Laridae species are extremely ecologically diverse (Table 1). Some species found to carry AIVs, such as the Artic tern (*Sterna paradisaea*), are present on every continent and ocean (Del Hoyo, 1996; IOC, 2012). They can also be long distance migrants, commonly occurring in freshwater habitats that are favorable to AIV persistence (Brown et al., 2009), and come in contact with humans and domestic animals, which highlights the potential importance of this group for veterinary and public health issues. The aim of this review is to shed light on current knowledge on AIVs in

Table 1. Variability in the ecology of Laridae species.

Life history traits				Implications
Population size	Abundant (e.g. herring gull; *Larus argentatus*)		Rare (e.g. lava gull; *Leucophaeus fuliginosus*)	AIV transmission more or less important
Habitat	Live in marine environments (e.g. black-legged kittiwake; *Rissa tridactyla*),	Live in both marine and freshwater habitats (e.g. yellow-legged gull; *Larus michahellis*)	Prefer freshwater habitats (e.g. ring-billed gull; *Larus delawarensis*)	Differential AIV transmission through abiotic reservoirs
Migration and Movements	Intercontinental migration (e.g. Arctic tern; *Sterna paradisaea*)	Local dispersive migration (e.g. black-tailed gull; *Larus crassirostris*)	Sedentary (e.g. black-bellied tern; *Sterna acuticauda*)	AIV transmission over longer or shorter distances and/or mostly (or not) during the breeding season
Nature of contacts with humans or domestic animal species	Direct contacts (e.g. kelp gull; *Larus dominicanus*).	Habitat sharing (e.g. herring gull; *Larus argentatus*)	Very limited contact (e.g. common tern; *Sterna hirundo*).	Interface between AIVs circulating in humans, domestic animals, and wildlife

Table 2. Low pathogenic avian influenza (LPAI) subtypes detected in Laridae.

Species	Virus		Sample type	References
	Haemagglutinin	Neuraminidase		
Arctic tern *Sterna paradisaea*	H5	N3		Obenauer et al. (2006)
Black-backed gull *Larus fuscus*	H7, H13	N2	Serum	GenBank (2012); Hinshaw et al. (1982); NIAID (2012); Zakstel'skaja et al. (1972)
Black-headed gull *Chroicocephalus ridibundus*	H2, H4, H5, H6, H7, H9, H11, H13, H16	N1, N2, N3, N5, N6, N4, N8	Cloaca, trachea, feces, embryos, and tissues	Fouchier et al. (2003); Germundsson et al. (2010); Gresikova et al. (1979); Hjulsager et al. (2012); Höfle et al. (2013); Janout et al. (1979); Lewis et al. (2013); Munster et al. (2007); Spackman et al. (2009)
Black-legged kittiwake *Rissa tridactyla*	H4, H13, H16	N2, N3, N6	Cloaca, trachea, and serum	Hall et al. (2013); Tønnessen et al. (2011)
Black-tailed gull *Larus crassirostris*	H1, H2, H4, H6, H13	N1, N2, N3, N6	Cloaca, trachea, serum, feces, and tissues	Otsuki et al. (1987); Slepuskin et al. (1972); Tsubokura et al. (1981)
Common gull *Larus canus*	H6, H13, H16	N2, N3, N8	Cloaca and trachea	Tønnessen et al. (2013b)
Common tern *Sterna hirundo*	H1, H2, H4, H7	N1, N7		Becker (1966); Röhm et al. (1995)
Franklin's gull *Leucophaeus pipixcan*	H6, H13	N2, N6, N9	Cloaca, trachea, and serum	Bahl & Pomeroy (1977); Hinshaw et al. (1982) (1983)
Glaucous gull *Larus hyperboreus*	H3, H5, H6, H13, H16	N1, N3, N8, N9	Cloaca	Ramey et al. (2010); USDA (2012)
Great black-backed gull *Larus marinus*	H4, H6, H11, H13, H16	N2, N6, N8, N9	Cloaca, trachea, and feces	Germundsson et al. (2010); Hjulsager et al. (2012); Munster et al. (2007); Wille et al. (2011)
Great black-headed gull *Ichthyaetus ichthyaetus*	H5, H9, H13	N2, N3, N6	Cloaca and trachea	L'vov et al. (2001)
American herring gull *Larus smithsonianus*	HA 1 to 13	NA 1 to 9	Cloaca and feces	NIAID (2012); Obenauer et al. (2006); Widjaja et al. (2004)
European herring gull *Larus argentatus*	H1, H2, H5, H6, H7, H10, H11, H13, H14, H16	N1, N2, N3, N4, N5, N6, N8	Cloaca, trachea, serum, and feces	Zakstel'skaja et al. (1972); Hinshaw et al. (1982); Kawaoka et al. (1988); L'vov et al. (2001); Munster et al. (2007); Germundsson et al. (2010); Marchenko et al. (2010); Kohls et al. (2011); NIAID 2012; Van Borm et al. (2012); Hjulsager et al. (2012)
Kelp gull *Larus dominicanus*	H13	N9	Cloaca	Pereda et al. (2008)
Laughing gull *Leucophaeus atricilla*	HA 1 to 13	NA 1 to 9	Cloaca and feces	Lee et al. (2001); NIAID 2012; Obenauer et al. (2006); Widjaja et al. (2004)
Mediterranean gull *Ichthyaetus melanocephalus*	H9	N2, N3	Trachea and feces	Lebarbenchon et al. (2009); Lewis et al. (2013)
Mew gull *Larus canus*	H5, H6, H13, H16	N1, N4, N6, N8	Cloaca, trachea, and serum	Germundsson et al. (2010); Kohls et al. (2011); Munster et al. (2007)
Ring-billed gull *Larus delawarensis*	H6, H11, H13, H16	N6	Cloaca, trachea, serum, and feces	Gaidet et al. (2010); Graves (1992); Hall et al. (2013); USDA (2012); Velarde et al. (2010)
Sabine's gull *Xema sabini*	H5	N3		Obenauer et al. (2006)
Silver gull *Chroicocephalus novaehollandiae*	H13	N6		NIAID (2012)
Slaty-backed gull *Larus schistisagus*	H4	N8		NIAID (2012)
Slender-billed gull *Chroicocephalus genei*	H4, H9, H16	N2, N3	Cloaca, trachea, and feces	L'vov et al. (1978); Mehrabanpour et al. (2012)
Sooty tern *Sterna funcata*	H7, H15	N2, N6, N9	Cloaca	Mackenzie et al. (1984); Obenauer et al. (2006)
Vega gull *Larus vegae*	H13	N6	Cloaca and feces	Spackman et al. (2009)
Yellow-legged gull *Larus michahellis*	H9, H13	N2, N6	Cloaca, trachea, feces, and tissues	Lebarbenchon et al. (2007); Lewis et al. (2013); Lin et al. (2009); Van Borm et al. (2012)
Whiskered tern *Chlidonias hybrida*	H6	N2		NIAID (2012)

This table presents the different subtypes of LPAIVs that have been found in Laridae species by direct detection or, for serum samples, through the detection of specific antibodies.

Laridae and identify efficient ways to clarify the taxon's present and future role in AIV epidemiological dynamics. We summarize available data on AIVs in Laridae and discuss the information in the context of the state of knowledge on AIVs in Anatidae, a system that is much better characterized. We set forth four fundamental questions: how, when, where, and to which host species are AIVs transmitted?

How are AIVs transmitted? A two-sided story

Transmission routes are determinant in a pathogen's evolutionary history (Huyse et al., 2005). The acquisition of new transmission pathways, including the incorporation of a new intermediary host or a novel vector, can allow a parasite to infect new hosts and lead to speciation (Huyse et al., 2005). Thus, transmission shapes pathogen diversification even among closely related infectious agents (e.g. Pérez-Tris et al., 2007). Our review of the literature suggests that the host specificity of the different LPAIVs, such as the H13 and H16 subtypes that are almost exclusively maintained in gull populations (Fouchier et al., 2005; Hinshaw et al., 1983; Kawaoka et al., 1988; Olsen et al., 2006; Wille et al., 2011; Yamnikova et al., 2003), may partly be a consequence of preferential transmission routes.

Transmission of LPAIVs in wild waterfowl is mainly fecal-oral; individuals are infected when they ingest water contaminated by infectious feces (Webster et al., 1992). However, airborne transmission also occurs (Costa et al., 2011). Currently available data suggest that LPAIV replication sites and shedding patterns differ between Laridae and Anatidae, which may subsequently impact the likelihood of transmission between these bird families. Indeed, in the mallard, which is the most common host species of the Anatidae family, LPAIV replication predominantly occurs in the intestinal tract, and high concentrations of infectious virus are shed in feces, even if oropharyngeal excretion is also observed (Costa et al., 2011; Ellström et al., 2008; Fereidouni et al., 2010; Jourdain et al., 2010; Kleijn et al., 2010; Webster et al., 1978). In contrast, although LPAIVs in black-headed gulls (*Chroicocephalus ridibundus*) seem to demonstrate fecal-oral transmission that is characterized by minimal pathogenicity (Höfle et al., 2012), some studies performed on laughing gulls (*Leucophaeus atricilla*), Franklin's gulls (*Leucophaeus pipixcan*) and ring-billed gulls (*Larus delawarensis*) suggest that LPAIVs are primarily or equally shed via the oropharynx (Bahl & Pomeroy, 1977; Brown et al., 2012; Costa et al., 2011).

Differences in receptor structure and location potentially underlie these differences in transmission patterns. Indeed, in order to enter host cells and then replicate, LPAIVs need to attach to receptors displayed at the surface of target cells. Most receptors are glycans terminating in sialic acids (SAs) (Nicholls et al., 2008; Suzuki, 2005). Two main types of SA receptors can be distinguished based on the linkage (α2,3 or α2,6) between the terminal SA and the glycan chain. According to the results of histochemistry studies using vegetal lectins, both types of receptors are present in Anatidae and humans, but their proportions and locations differ across species. In humans, α2,6-linked SAs are predominantly found in the upper respiratory tract, whereas α2,3-linked SAs are more numerous in the lower respiratory tract (Shinya et al., 2006). In the human intestinal tract, α2,6-linked SAs are found in the endothelium and α2,3-linked SAs are present in neurons and endothelial cells but not on epithelial cells (Yao et al., 2008). In mallards and Pekin ducks (*A. platyrhynchos domesticus*), lectin studies suggest that both types of receptors are present in the upper respiratory and intestinal epithelia but that α2,3-linked SAs predominate (Ellström et al., 2009; França et al., 2013; Kuchipudi et al., 2009; Pillai & Lee, 2010). Conversely, α2,6-linked SA receptors were found to be strongly expressed in the ciliated epithelium of the upper respiratory tract of various gull species (Ellström et al., 2009), whereas α2,3-linked SAs were predominantly detected in the digestive tract (França et al., 2013; Lindskog et al., 2013).

Further research is needed to confirm if these differences in receptor type and occurrence are systematically observed in all Laridae species and if they are predictive of species susceptibility to AIV subtypes. These differences might reflect the important ecological differences that exist between Anatidae and Laridae. Indeed, as Anatidae and Laridae often share wetlands, they could both theoretically become infected through direct contact or contaminated freshwater (Del Hoyo, 1996). However, the Laridae dietary regime is distinct from that of Anatidae. Anatidae are generally herbivorous or granivorous freshwater foragers. Laridae, in contrast, tend to be generalists in marine ecosystems, often consuming invertebrates and fishs, and some opportunistic species may even eat sick or dead birds, thus favoring the direct transmission of AIVs (Brown et al., 2008). Second, Laridae breed in high-density colonies in which contact rates may be high, which could facilitate direct airborne transmission of viruses (Loehle et al., 1995). Third, orofecal transmission may be infrequent in the coastal habitats in which gulls and terns most frequently forage because of salinity's adverse effects on LPAIV persistence (Brown et al., 2009; Stallknecht et al., 1990).

As a consequence of these ecological differences, an alternative AIV transmission route may be evolutionarily maintained in Laridae. The maintenance of different preferential transmission routes could explain the evolution of different subtypes in the two taxonomic groups. Indeed, H13 and H16 AIV subtypes are almost exclusively maintained in gull populations (Fouchier et al., 2005; Hinshaw et al., 1983; Kawaoka et al., 1988; Olsen et al., 2006; Yamnikova et al., 2003) and account for only a small proportion of the AIVs found in other avian taxa, including Anatidae (Kang et al., 2012; Munster et al., 2007; Sivanandan et al., 1991). H13 and H16 viruses also have gene segments that are genetically distinct from those of other AIVs that circulate in different wild bird hosts (Tønnessen et al., 2013a; Wille et al., 2011). This finding suggests that these subtypes diverged from other LPAIVs relatively recently (Webster et al., 1992; Wille et al., 2011), although enough time has passed to allow genetic differentiation (Munster & Fouchier, 2009).

Even if available data are consistent with this hypothetical scenario, further investigations are clearly needed. Whenever possible, both cloacal and oropharyngeal swabs should be collected during field studies, which would provide information about the respective importance of these transmission

routes in Laridae and Anatidae. Further experimental studies are also required to directly investigate AIV transmission in wild birds. In particular, future research should be guided by already published work on influenza virus transmission in mammals in controlled laboratory conditions (e.g. Lowen et al., 2007). Studies should investigate the airborne, waterborne, and contact transmission dynamics of the strains associated with both Anatidae (e.g. H4 or H7) and Laridae (H13 or H16).

When? Seasonal patterns and infection peaks

The temporal dynamics of infections are strongly influenced by host immunity. Infection peaks tend to occur when a large proportion of the host population is susceptible to a given infectious agent, while lower incidences are observed when the population is less vulnerable, perhaps due in part to more efficient host immune responses (Keeling & Rohani, 2008). Immune responses are, in turn, shaped by host–pathogen coevolution; they partly depend on the life history traits of the host species, including host longevity (Lee, 2006). As Laridae differ from Anatidae in their life-history traits, their immune responses, and thus their temporal AIV infection dynamics, may also differ.

In Anatidae, AIV dynamics follow a clear seasonal pattern. Infection peaks are observed in the late summer or early fall in both North America and Europe (Lebarbenchon et al., 2010b; Wallensten et al., 2007; Webster et al., 1992). These peaks are thought to be primarily linked to the presence of large numbers of juveniles, which gather during and after their migration to wintering grounds and are immunologically naïve (Olsen et al., 2006; Stallknecht & Shane, 1988). These infection peaks in Anatidae could also be predicted to occur in Laridae. However, current data do not support the existence of a similar temporal infection pattern. In fact, Laridae infection patterns vary tremendously. For example, in Delaware Bay (North America), where a large AIV surveillance program is in place (877 Laridae sampled), most positive samples have been collected during the breeding period in May (Hanson et al., 2008). In the Caucasus region, which lies at the border between Europe and Asia, AIV prevalence peaked in the spring in black-headed gulls and during the autumn migration in Armenian gulls, Caspian gulls and yellow-legged gulls (Lewis et al., 2013). In Northern Europe, virus prevalence in the 2602 Laridae sampled was highest from June to August, and AIVs were not detected at all in many colonies during the breeding season (Munster et al., 2007). Furthermore, punctual infection peaks are seen in gull chicks but not in ducklings, for which no data exist concerning natural LPAIV infection (Fouchier et al., 2005; Velarde et al., 2010). This lack of data may be due to the fact that gull chicks are nidicolous and thus easier to sample than ducklings, which are nidifugous. Virus prevalences reported for Laridae (all age groups considered) are generally lower than those reported for Anatidae. Olsen et al. (2006) reported a mean AIV prevalence level of 1.4% in gulls ($n = 14\,505$) and 9.5% in ducks ($n = 34\,503$). Because prevalence levels in Laridae are low, sample sizes need to be very large to detect seasonal patterns, which could explain why similar infection peaks have yet to be detected in this group. The temporal infection pattern may also depend on the species, the virus subtype or the environment studied.

The difference in average lifespan between the two bird families may also partly explain the observed differences in temporal infection patterns because a longer lifespan means a greater chance to acquire immunity. Laridae are generally long-lived birds, while the lifespan of Anatidae is usually short. This difference is even greater for species that are hunted, such as the mallard (Stallknecht & Brown, 2007). For example, the mean annual survival likelihood of adult mallards in North America and Europe is about 50% (Schekkerman & Slaterus, 2008), while it usually reaches 90% for adult gulls (Altwegg et al., 2007; Breton et al. 2008; Oro et al., 2004). This difference in lifespan may have a 2-fold influence on immunity acquisition and AIV epidemiological dynamics. First, a long lifespan favors the development of acquired immune responses, and the protection afforded by these responses should last longer than that in short-lived birds (Lee, 2006). Second, the acquired immune response in adults may be carried through to the next generation by the maternal transfer of antibodies to chicks through egg yolks (Boulinier & Staszewski, 2008; Gasparini et al., 2001). Thus, the low AIV prevalence observed in Laridae could be due to stronger and/or longer lasting immune responses, which are linked to longer lifespans. Furthermore, only two AIV subtypes (H13/H16) predominate in Laridae, potentially reducing the diversity of antibodies birds need, while subtypes are much more diverse in Anatidae (e.g. Munster et al., 2007). Indeed, AIV antibodies seem to be subtype specific in birds even if cross-immunity exists between related (Fereidouni et al., 2010; Latorre-Margalef, 2013) and non-related subtypes (Jourdain et al., 2010; Pepin et al., 2012).

Although few data on immunity in Laridae are available, they thus far support the idea that antibodies persist longer in Laridae than in Anatidae. Furthermore, in Laridae populations, AIV seroprevalence is high and the incidence of infection is low (De Marco et al., 2005; Maxted et al., 2012; Velarde et al., 2010). In pink-footed geese (Anatidae: *Anser brachyrhynchus*), LPAIV-specific antibodies persisted 343 days on average (Hoye et al., 2011), and when mallards were experimentally infected with different LPAIV strains, the strong immune response that was detectable after viral inoculation lasted less than a year in 7 of the 8 ducks studied (Fereidouni et al., 2010; Tolf et al., 2013). While the persistence of influenza-specific antibodies in Laridae has yet to be assessed, data on other pathogens suggest protection is longer lasting, although it is, of course, difficult to compare the persistence of antibodies provoked by different infectious agents. In a wild population of naturally infected black-legged kittiwakes (Laridae: *Rissa tridactyla*), antibody levels against *Borrelia burgdorferi* persisted interannually (Staszewski et al., 2007b). Additionally, in a vaccination study involving Newcastle disease virus (a pathogen not naturally encountered in the study population), 13 black-legged kittiwakes still had high levels of NDV-specific antibodies one year post-vaccination (Staszewski et al., 2007a). Since a model examining another long-lived seabird (the Amsterdam albatross, *Diomedea amsterdamensis*) has shown that maternal antibodies could strongly influence pathogen circulation dynamics (Garnier et al., 2012), we speculate that maternal

antibody transfer may also influence AIV infection dynamics in Laridae populations. This hypothesis is supported by the fact that AIV-specific antibodies were detected in a large proportion of eggs sampled in yellow-legged gull colonies in France (*Larus michahellis*; Pearce-Duvet et al., 2009) and in Tunisia (Hammouda et al., 2011).

Knowledge on AIV immune responses in wild birds remains scarce (Tolf et al., 2013), and we are still a long way from fully understanding the mechanisms underlying AIV epidemiological dynamics. Experimental infection studies are needed to clarify AIV-specific immune responses at the individual scale. For instance, an appropriate study design, which would include rearing Laridae chicks in the lab, could reveal the duration of protection afforded to chicks by maternal antibodies. Because chick density in colonies is high, maternal antibody transfer could have an essential role in AIV temporal dynamics, particularly if transmission in Laridae is mostly airborne. Long-term experimental infection studies in Laridae and Anatidae species would allow us to assess the duration and variability of antibody persistence following single or successive LPAIV infections. Studies designed to investigate the annual epidemiological cycle of avian influenza in host populations should be implemented; they should include both virological and serological sampling to shed light on the temporal dynamics of both infection and immunity (Tønnessen et al., 2011).

Where? Migratory movements

The genetic structure of pathogen populations is shaped by population connectivity and, as a consequence, host migration (e.g. Monot et al., 2009; Vollmer et al., 2011; Wirth et al., 2005). Migration favors the spatial spread of pathogens, while the high densities of hosts on wintering or breeding sites favor the multiplication and exchange of infectious agents. Moreover, during migration, exchanges between individuals originating from different geographic areas and belonging to different species can take place at stopover sites (Jourdain et al., 2007). As a result, the distinct migration patterns of Laridae and Anatidae species may determine the gene pools of their circulating AIVs; at the same time, the sharing of habitats may allow AIV exchanges between the two taxa (Tønnessen et al., 2013b).

At present, data are too scarce to test these hypotheses. Indeed, AIV studies generally only target a few species. Of the 102 Laridae species known, epidemiologic data on AIV circulation is only available for 20 (Table 2). For example, some Laridae species that feed offshore and breed in mono-specific colonies, such as the black-legged kittiwake, might maintain the circulation of specific AIVs because they have limited contact with other species (although in many locations they can breed with other cliff-nesting seabirds). However, most existing studies tend to show that LPAIV dynamics emerge at the community rather than the population level, which suggests that such epidemiological isolation is rare. Indeed, previous studies investigating the effect of host species, geographic location, and sampling time on AIV prevalence levels across broad geographical areas and time scales observed weak support for a species effect and, instead, found evidence for phylogenetic clustering by space and time (Chen & Holmes, 2009; Girard et al., 2012; Pearce et al., 2010, 2011; Ramey et al., 2010; Van Borm et al., 2012). These findings suggest that transmission and reassortment of AIVs between species may be frequent (Chen & Holmes, 2009; Girard et al., 2012; Pearce et al., 2010, 2011; Ramey et al., 2010; Reeves et al., 2011; Van Borm et al., 2012), and we hypothesize that migration may play a key role therein.

Indeed, differences in migration patterns between Anatidae and Laridae may have an important influence on AIV reassortment. In North America, Anatidae migration flyways seem to constrain gene flow among LPAIVs (Lam et al., 2012). Similarly, Eurasian and North American LPAIVs isolated from waterbirds (mostly Anatidae) showed substantial levels of sequence divergence, a result that was attributed to the geographical separation of the bird populations (Ito et al., 1991; Kawaoka et al., 1998; Olsen et al., 2006; Suarez & Perdue 1998; Widjaja et al., 2004). A few Anatidae species, like northern pintails (*Anas acuta*) and Steller's eiders, (*Polysticta stelleri*), have been identified as bridge species that allow exchanges between Eurasian and North American AIV strains, due to their migration routes and the isolation of intercontinentally reassorted AIVs from some individuals (e.g. Pearce et al., 2009; Ramey et al., 2010).

In contrast, numerous Laridae species undergo intercontinental migration, not only between Eurasia and North America, but also between North America and South America and between Eurasia and Oceania (Del Hoyo, 1996; Elphick, 2007; Winker & Gibson, 2010). Such movements appear to result in intercontinental AIV exchanges. Most of the intercontinentally reassorted viruses that have been identified to date have been found in Laridae (e.g. Hall et al., 2013; Lebarbenchon et al., 2009; Pereda et al., 2008; Van Borm et al., 2012; Wille et al., 2011), which seem to be the main carriers of reassorted AIV strains, followed by Anatidae and shorebirds (Krauss et al., 2007; Ramey et al., 2010). Consequently, Laridae migratory patterns may play a major role in mediating AIV intercontinental exchanges (Wille et al., 2011; Winker & Gibson, 2010). Genetic exchanges appear to be concentrated at key sites, such as in Alaska (Ramey et al., 2010), Delaware Bay (Wille et al., 2011), and the Camargue wetlands (Lebarbenchon et al., 2009), where migratory flyways overlap and birds wintering in different continents gather in high densities. In Alaska, where birds from as many as six continents come to breed (Winker et al., 2007), reassorted Eurasian/North American LPAIV strains represent up to 85% of those isolated from both Anatidae and Laridae (Ramey et al., 2010).

To date, few studies have been conducted on other major potential AIV exchange sites. Efforts should concentrate more on the Southern Hemisphere, as data on AIV circulation in wild birds from this region, apart from Southeast Asia, are scarce. Researchers could take advantage of readily available ornithological knowledge and focus on species known to undergo intercontinental migrations, as well as on habitats situated at the crossroads of several waterbird migratory routes. For example, the Kamchatka region is the Eurasian counterpart of Alaska (Wille et al., 2011). The few studies that detected AIVs in both Laridae and Anatidae in South America, Africa, and Oceania were mostly conducted in areas

that were designated as important (Important Bird Areas) by Birdlife International and that represent important wintering and/or breeding sites (IBA, Birdlife International, 2013). These areas include the Djoudj delta in Senegal (Gaidet et al., 2007), the coast of Tasmania (Haynes et al., 2009), and the Parana River basin in Argentina (Pereda et al., 2008). Such studies could reveal the relative roles played by Laridae and Anatidae in intercontinental AIV exchanges worldwide. Moreover, such studies would represent the first steps towards a better understanding of AIV spatial dynamics, which are crucial components of models of AIV dispersion risks. In particular, the timing and occurrence of infection within populations during annual migratory cycles must be understood (Hoye et al., 2011). Studies of northern pintails have shown that this migratory species can bring new strains from Eurasia to North America, which can then spread into populations of sympatric species (Koehler et al., 2008; Pearce et al., 2009, 2011). Given such findings, it also appears essential to expand studies to include species that are sympatric with long-distance migrants during part of their life cycle.

To which species are AIVs transmitted? Viral exchanges between Laridae, domestic birds, and humans

New AIV strains that emerge in humans and domestic birds often evolve from strains originally circulating in wild birds. Thus far, research has focused on AIVs that could be spread by mallards and other Anatidae species that share wetlands with domestic birds and humans. However, Laridae species might also play a key role in the production of reassortant viruses (Hall et al., 2013), particularly since Laridae also share wetlands with domestic species (Del Hoyo, 1996), which can favor AIV exchanges (Caron et al., 2010). Interestingly, the AIV first isolated from a Laridae species was closely related to an influenza strain pathogenic for domestic poultry (Becker et al. 1966). Since then, a wide diversity of subtypes has been detected in Laridae (Table 2). Virus histochemistry studies showed that a mallard H6N1 LPAIV strain was capable of attaching to tissues (trachea and colon) of the domestic chicken (*Gallus gallus*), the herring gull and Franklin's gull (Jourdain et al., 2011; Lindskog et al., 2013). Early experimental infection studies showed that Franklin's gulls were capable of shedding an AIV subtype pathogenic for turkeys (Bahl & Pomeroy, 1977). Another study found that a LPAIV subtype isolated from Laridae feces was somewhat pathogenic in poultry, although the symptoms it provoked were less severe than those caused by certain other avian influenza viruses (Otsuki et al. 1982). These examples indicate that Laridae may be permissive hosts when it comes to AIVs and could therefore contribute to the interspecific spread of AIV subtypes. With regards to the H13 and H16 subtypes, at least one LPAIV epizootic in poultry has been linked to a H13 subtype found in wild gulls (Laudert et al., 1993; Sivanandan et al., 1991). Furthermore, a recent study found that a small proportion of domestic ducks and turkeys that had been experimentally inoculated with some specific H13 LPAIV strains developed infections after challenge, which suggests that gull-adapted viruses can spill over into domestic birds (Brown et al., 2012). These findings emphasize the potential for AIV exchange among Anatidae, Laridae and domestic species.

What is particularly worrisome is that Laridae can host highly pathogenic subtypes that have zoonotic potentials. At least eight species of Laridae were able to be infected by H5N1 HPAIVs under natural conditions (Table 3). H5N1 HPAIVs have occasionally been isolated not only from dead or severely sick birds (Ellis et al., 2004; Liu et al., 2005), but also from apparently healthy birds (Muzinic et al., 2010; Savić et al., 2010). Experimental infection studies have shown that laughing gulls and herring gulls can serve as healthy carriers of H5N1 HPAIVs, although the infections are sometimes fatal (Brown et al., 2006, 2008; Perkins & Swayne, 2001). Additionally, laughing gulls can become infected by consuming meat contaminated with H5N1 HPAIVs, which the gulls are likely to encounter in nature while scavenging (Brown et al., 2008). As a result, Laridae do not seem to serve as long-term reservoirs of HPAIVs. However, because several Laridae species can be infected by HPAIVs, sometimes without presenting any symptoms, they could contribute to geographical and interspecific spread of these viruses.

Other AIV subtypes likely have the potential to infect both Laridae species and humans. Indeed, several gull species display α2,6-linked SA receptors, to which human influenza viruses usually bind, on the surface of their tracheal epithelium (Ellström et al., 2009; Jourdain et al., 2011; Lindskog et al., 2013). Laridae species can also be infected by LPAIV subtypes that are known to cause mild infections (asymptomatic or mild conjunctivitis) in humans, such as H9 and H7 (Sandrock et al., 2007). Thus far, gull-specific H13 and H16 subtypes have never been reported in humans, although an H16N3 gull virus was found to attach to the human respiratory tract and eye, which suggests that the first step necessary for gull to human transmission of this virus can occur (Lindskog et al., 2013). Another recent study revealed that an H13N6 exclusively bound to avian α2,3-linked SA receptors and was not observed to bind to mammalian α2,6-linked SA receptors (Lu et al., 2013); however, a single amino acid substitution was shown to result in changes in the binding patterns of this H13 virus (Lu et al., 2013).

Contact between humans and Laridae as a result of hunting is limited. Nevertheless, it may occur. For example, terns are regularly trapped on West African beaches (Boere & Dodman, 2011). Contact between humans and Laridae is more commonly due to habitat sharing. Indeed, over the past decades, several large gull species have dramatically increased in abundance, especially in Europe and North America (Blokpoel & Spaans, 1991). These species have colonized urban areas worldwide by taking advantage of anthropogenic resources such as garbage and trawling discards (Duhem et al., 2008; Lisnizer et al., 2011; Raven & Coulson, 1997), and their contribution to AIV circulation in urban settings should therefore not be neglected (Verhagen et al., 2012). They occur at the epidemiological interface between humans and wildlife; for instance, antibiotic-resistant bacteria originating in human populations have been found in gulls (Bonnedahl et al., 2009; Dolejska et al., 2007; Gionechetti et al., 2008).

Overall, we may conclude that Laridae can occasionally transmit AIVs to humans and domestic birds but such events are infrequent compared to the number of spillovers from wild

Table 3. Highly pathogenic avian influenza (HPAI) subtypes detected in Laridae.

Species	Virus subtypes	Infection type	Host status/symptoms	Sample type	References
Black-headed gull *Chroicocephalus ridibundus*	H5N1	Natural and experimental	Healthy carrier, pathology, and fatal disease	Trachea and cloaca	Ellis et al. (2004); Ramis et al. (2012); Sakoda et al. (2012); Savić et al. (2010)
Brown-headed gull *Chroicocephalus brunnicephalus*	H5N1	Natural and experimental	Pathology and fatal disease	Trachea, cloaca, and serum	Liu et al. (2005); Ratanakorn et al. (2012)
Common gull *Larus canus*	H5N1	Natural	Healthy carrier		Sharshov (2010)
Common tern *Sterna hirundo*	H5N1 H5N3	Natural	Healthy carrier, pathology, and fatal disease	Trachea, cloaca and tissues	Becker (1966); L'vov et al. (2006)
Great black-headed gull *Ichthyaetus ichthyaetus*	H5N1	Natural	Pathology	Trachea and cloaca	Liu et al. (2005)
European herring gull *Larus argentatus*	H5N1	Experimental	Pathology and fatal disease	Trachea, cloaca, and serum	Brown et al. (2008)
Laughing gull *Leucophaeus atricilla*	H5N3, H5N1	Experimental	Healthy carrier, pathology, and fatal disease	Trachea, cloaca, serum, and tissues	Brown et al. (2006); Perkins & Swayne (2001)
Mongolian gull *Larus mongolicus*	H5N1	Natural	Fatal disease	Trachea and cloaca	Gilbert et al. (2012)
Slaty-backed gull *Larus schistisagus*	H5N1	Natural	Healthy carrier		Kou et al. (2009)
Slender-billed gull *Chroicocephalus genei*	H5N2	Natural	Healthy carrier	Cloaca	L'vov et al. (1978)

This table presents the HPAIV subtypes that have been detected in nature or studied experimentally in Laridae species by direct detection or, for serum samples, through the detection of specific antibodies.

ducks. As previously mentioned, no transmission to humans has been reported for H13 and H16 subtypes, and only one LPAI epizootic in poultry has been linked to a H13N2 subtype found in wild gulls (Sivanandan et al., 1991). However, it is crucial to consider the possible role of gulls in AIV transmission because Laridae can host HPAIVs and studies involving experimental infection have shown that gulls could potentially transmit viruses to humans or domestic birds (Krauss et al., 2007; Winker & Gibson, 2010). Finally, as antiviral-resistant influenza strains have already been isolated from mallards (Järhult et al., 2011; Orozovic et al., 2011), Laridae might also favor the dispersal and spread of antiviral-resistant strains in human populations (Dharan, 2009; Meijer et al., 2009; Moscona, 2009). Thus, future studies should focus on opportunistic, urban Laridae species that live in close contact with humans to gain insight into the AIV exchanges that may take place at this interface.

Conclusion

By comparing and contrasting existing data on AIVs in Laridae and Anatidae, it seems clear that these two taxa may play distinct roles in AIV epidemiology. For physiological and ecological reasons, airborne transmission may occur more frequently in Laridae than in Anatidae, which could favor the evolution of taxon-specific strains in the two families. As Laridae generally live longer than Anatidae, they may have evolved stronger immune responses that could lead to different temporal infection patterns across the two groups. The numerous Laridae species that migrate long distances may play a major role in intercontinental AIV gene flow, given that a high proportion of North American-Eurasian reassortant AIV strains have been detected in this group. Finally, opportunistic Laridae species, which have recently colonized urban areas worldwide, are in close contact with both humans and their domestic animals, which could favor AIV exchanges even if such exchanges have rarely been reported thus far. Overall, the Laridae thus constitute an AIV host group that should not be neglected. Further research is clearly needed to clarify AIV epidemiological dynamics in aquatic birds, and it would greatly benefit from incorporating evolutionary ecology and ornithological knowledge.

Declaration of interest

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References

Alexander DJ. (2000). A review of avian influenza in different bird species. Vet Microb 74:3–13.

Altwegg R, Crawford RJM, Underhill LG, Martin AP, Whittington PA. (2007). Geographic variation in reproduction and survival of kelp gulls *Larus dominicanus vetula* in southern Africa. J Avian Biol 38: 580–6.

Arsnoe DM, Ip HS, Owen JC. (2011). Influence of body condition on influenza A virus infection in mallard ducks: experimental infection data. PLoS ONE 6: e22633.

Bahl AK, Pomeroy BS. (1977). Experimental exposure of Franklins' gulls (*Larvus pipixcan*) and mallards (Anas platyrhynchos) to a turkey influenza virus A/Turkey/Minn/BF/72 (Hav6Neq2). J Wildl Dis 13: 420–6.

Banks J, Speidel ES, Moore E, Plowright L, Piccirillo A, Capua I, Cordioli P, Firoetti A, Alexander DJ. (2001). Changes in the haemagglutinin and the neuraminidase genes prior to the emergence of highly pathogenic H7N1 avian influenza viruses in Italy. Arch virol 146:963–73.

Becker WB. (1966). The isolation and classification of tern virus: influenza virus A/tern/South Africa/1961. J Hyg-Cambridge 64: 309–20.

Birdlife International (2013). Important Bird Areas (IBAs). Available from: http://www.birdlife.org/action/science/sites/index.html [last accessed 1 Jun 2013].

Blokpoel H, Spaans AL. (1991). Introductory remarks: superabundance in gulls: causes, problems and solutions. Wellington: Bell, B.D. Editions, Acta XX congressus internationalis ornithologici, 2361–3.

Boere G, Dodman T. (2011). Understanding the flyway approach to conservation. WOW training resources. Available from: http://wow.wetlands.org/LinkClick.aspx?fileticket=%2b%2b4YtjlpxlY%3d&tabid=1688&mid=6756&language=en-US [last accessed 1 Jun 2013].

Bonnedahl J, Drobni M, Gauthier-Clerc M, et al. (2009). Dissemination of *Escherichia coli* with CTX-M type ESBL between humans and yellow-legged gulls in the south of France. PLoS One 4: e5958.

Boulinier T, Staszewski V. (2008). Maternal transfer of antibodies: raising immuno-ecology issues. Trends Ecol Evol 23:282–8.

Breton AR, Fox GA, Chardine JW. (2008). Survival of adult herring gulls (*Larus argentatus*) from a lake Ontario colony over two decades of environmental change. Waterbirds 31:15–23.

Brown JD, Goekjian G, Poulson R, et al. (2009). Avian influenza virus in water: infectivity is dependent on pH, salinity and temperature. Vet Microb 136:20–6.

Brown JD, Poulson R, Carter D, et al. (2012). Susceptibility of avian species to North American H13 low pathogenic avian influenza viruses. Av Dis 56:969–75.

Brown JD, Stallknecht DE, Beck JR, et al. (2006). Susceptibility of North American ducks and gulls to H5N1 highly pathogenic avian influenza viruses. Emerg Infect Dis 12:1663–70.

Brown JD, Stallknecht DE, Swayne DE. (2008). Experimental infections of herring gulls (*Larus argentatus*) with H5N1 highly pathogenic avian influenza viruses by intranasal inoculation of virus and ingestion of virus-infected chicken meat. Avian Pathol 37:393–7.

Caron A, de Garine-Wichatitsky M, Gaidet N, et al. (2010). Estimating dynamic risk factors for pathogen transmission using community-level bird census data at the wildlife/domestic interface. Ecol Soc 15:25–43.

Chen H, Smith GJD, Zhang SY, et al. (2005). Avian flu: H5N1 virus outbreak in migratory waterfowl. Nature 436:191–2.

Chen R, Holmes EC. (2009). Frequent inter-species transmission and geographic subdivision in avian influenza viruses from wild birds. Virology 383:156–61.

Costa TP, Brown JD, Howerth EW, Stallknecht DE. (2011). Variation in viral shedding patterns between different wild bird species infected experimentally with low-pathogenicity avian influenza viruses that originated from wild birds. Avian Pathol 40:119–24.

Daszak P, Cunningham AA, Hyatt AD. (2000). Emerging infectious diseases of wildlife – threats to biodiversity and human health. Science 287:443–9.

De Marco MA, Foni E, Campitelli L, et al. (2005). Influenza virus circulation in wild aquatic birds in Italy during H5N2 and H7N1 poultry epidemic periods (1998 to 2000). Avian Pathol 34:480–5.

De Wit E, Fouchier RAM. (2008). Emerging influenza. J Clin Virol 41:1–6.

Del Hoyo J. (1996). Hoatzin to auks. Barcelona: Lynx Editions, 821 pp.

Dharan NJGL. (2009). Infections with oseltamivir-resistant influenza A (H1N1) virus in the United States. J Am Med Assoc 301:1034–41.

Dolejska M, Cizek A, Literak I. (2007). High prevalence of antimicrobial-resistant genes and integrons in *Escherichia coli* isolates from Black-headed Gulls in the Czech Republic. J Appl Microbiol 103: 11–19.

Duhem C, Roche P, Vidal E, Tatoni T. (2008). Effects of anthropogenic food resources on yellow-legged gull colony size on Mediterranean islands. Popul Ecol 50:91–100.

Earn DJD, Dushoff J, Levin SA. (2002). Ecology and evolution of the flu. Trends Ecol Evol 17:334–40.

Ellis TM, Bousfield RB, Bissett LA, et al. (2004). Investigation of outbreaks of highly pathogenic H5N1 avian influenza in waterfowl and wild birds in Hong Kong in late 2002. Avian Pathol 33: 492–505.

Ellström P, Jourdain E, Gunnarsson O, et al. (2009). The ''human influenza receptor'' Neu5Ac'alpha'2,6Gal is expressed among different taxa of wild birds. Arch Virol 154:1533–7.

Ellström P, Latorre-Margalef N, Griekspoor P, et al. (2008). Sampling for low-pathogenic avian influenza A virus in wild mallard ducks: oropharyngeal versus cloacal swabbing. Vaccine 26:4414–16.

Elphick J. (2007). The atlas of bird migration. London: Natural History Museum, 180.

Fereidouni SR, Grund C, Häuslaigner R, et al. (2010). Dynamics of specific antibody responses induced in mallards after infection by or immunization with low pathogenicity avian influenza viruses. Avian Dis 54:79–85.

Ferguson NM, Cummings DAT, Cauchemez S, et al. (2005). Strategies for containing an emerging influenza pandemic in Southeast Asia. Nature 437:209–14.

Flint PL, Franson JC. (2009). Does influenza A affect body condition of wild mallard ducks, or vice versa? Proc Biol Sci 276:2345–6.

Fouchier RAM, Munster VJ, Keawcharoen J, et al. (2007). Virology of avian influenza in relation to wild birds. J Wildl Dis 43:S7–14.

Fouchier RA, Munster VJ, Wallensten A, et al. (2005). Characterization of a novel influenza A virus hemagglutinin subtype (H16) obtained from black-headed gulls. J Virol 79:2814–22.

Fouchier RAM, Olsen B, Bestebroer TM, et al. (2003). Influenza A virus surveillance in wild birds in Northern Europe in 1999 and 2000. Avian Dis 47:857–60.

França M, Stallknecht DE, Howerth EW. (2013). Expression and distribution of sialic acid influenza virus receptors in wild birds. Avian Pathol 42:60–71.

Gaidet N, Cappelle J, Takekawa JY, et al. (2010). Potential spread of highly pathogenic avian influenza H5N1 by wildfowl: dispersal ranges and rates determined from large-scale satellite telemetry. J Appl Ecol 47:1147–57.

Gaidet N, Dodman T, Caron A, et al. (2007). Influenza surveillance in wild birds in Eastern Europe, the Middle East, and Africa: preliminary results from an ongoing FAO-led survey. J Wildl Dis 43:S22–8.

Garnier R, Ramos R, Staszewski V, et al. (2012). Maternal antibody persistence: a neglected life-history trait with implications from albatross conservation to comparative immunology. Proc Biol Sci 279: 2033–41.

Gasparini J, McCoy KD, Haussy C, et al. (2001). Induced maternal response to the Lyme disease spirochaete *Borrelia burgdorferi sensu lato* in a colonial seabird, the kittiwake *Rissa tridactyla*. Proc Biol Sci 268:647–50.

GenBank (2012). Available from: http://www.ncbi.nlm.nih.gov/genbank/ [last accessed 16 Dec 2013].

Germundsson A, Madslien KI, Hjortaas MJ, et al. (2010). Prevalence and subtypes of influenza A viruses in wild waterfowl in Norway 2006–2007. Acta Vet Scand 52:28–32.

Gilbert M, Jambal L, Karesh WB, et al. (2012). Highly pathogenic avian influenza virus among wild birds in Mongolia. PLoS ONE 7:e44097.

Girard YA, Runstadler JA, Aldehoff F, Boyce W. (2012). Genetic structure of Pacific Flyway avian influenza viruses is shaped by geographic location, host species, and sampling period. Virus Genes 44:415–28.

Hall JS, TeSlaa JL, Nashold SW, et al. (2013). Evolution of a reassortant North American gull influenza virus lineage: drift, shift and stability. Virol 10:179–87.

Höfle U, Van de Bildt MWG, Leijten LM, et al. (2012). Tissue tropism and pathology of natural influenza virus infection in black-headed gulls (*Chroicocephalus ridibundus*). Avian Pathol 41:547–53.

Gionechetti F, Zucca P, Gombac F, et al. (2008). Characterization of antimicrobial resistance and class 1 integrons in Enterobacteriaceae isolated from Mediterranean herring gulls (*Larus cachinnans*). Microb Drug Resist 14:93–9.

Gortázar C, Ferroglio E, Höfle U, et al. (2007). Diseases shared between wildlife and livestock: a European perspective. Eur J Wildlife Res 53: 241–56.

Graves IL. (1992). Influenza viruses in birds of the Atlantic flyway. Avian Dis 36:1–10.

Gresikova M, Sekeyova M, Tŭmová B, Stumpa A. (1979). Isolation of an influenza A virus strain from a bird embryo (Larus ridibundus) collected in Slovakia. Acta Virol 23:89–92.

Hammouda A, Pearce-Duvet J, Chokri MA, et al. (2011). Prevalence of influenza A antibodies in yellow-legged gull (*Larus michahellis*) eggs and adults in southern Tunisia. Vector Borne Zoonotic Dis 11: 1583–90.

Hanson BA, Luttrell MP, Goekjian VH, et al. (2008). Is the occurrence of avian influenza virus in Charedriiformes species and location dependent? J Wildl Dis 44:351–61.

Harvell CD, Kim K, Burkholder JM, et al. (1999). Emerging marine diseases – climate links and anthropogenic factors. Science 285: 1505–10.

Haydon DT, Cleaveland S, Taylor LH, Laurenson MK. (2002). Identifying reservoirs of infection: a conceptual and practical challenge. Emerg Infect Dis 8:1468–73.

Haynes L, Arzey E, Bell C, et al. (2009). Australian surveillance for avian influenza viruses in wild birds between July 2005 and June 2007. Aust Vet J 87:266–72.

Heymann DL, Rodier G. (2004). Global surveillance, national surveillance, and SARS. Emerg Infect Dis 10:173–5.

Hinshaw VS, Air GM, Gibbs AJ, et al. (1982). Antigenic and genetic characterization of a novel hemagglutinin subtype of influenza A viruses from gulls. J Virol 42:865–72.

Hinshaw VS, Air GM, Schild GC, Newman RW. (1983). Characterization of a novel haemagglutinin subtype (H13) of influenza A viruses from gulls. Bull World Health Org 61:677–9.

Hjulsager CK, Breum SØ, Trebbien R, et al. (2012). Surveillance for avian influenza viruses in wild birds in Denmark and Greenland, 2007–10. Avian Dis 56:992–8.

Höfle U, Van de Bildt MWG, Leijten LM, et al. (2012). Tissue tropism and pathology of natural influenza virus infection in black-headed gulls (*Chroicocephalus ridibundus*). Avian Pathol 41:547–53.

Horimoto T, Kawaoka Y. (2001). Pandemic threat posed by avian influenza A viruses. Clin Microbiol Rev 14:129–49.

Hoye BJ, Munster VJ, Nishiura H, et al. (2011). Reconstructing an annual cycle of interaction: natural infection and antibody dynamics to avian influenza along a migratory flyway. Oikos 120:748–55.

Hurst CJ. (2011). Studies in viral ecology: animal host systems. Hoboken: Wiley-Blackwell.

Hurt AC, Hansbro PM, Selleck P, et al. (2006). Isolation of avian influenza viruses from two different transhemispheric migratory shorebird species in Australia. Arch Virol 151:2301–9.

Huyse T, Poulin R, Théron A. (2005). Speciation in parasites: a population genetics approach. Trends Parasitol 21:469–75.

IOC (2012). IOC World Bird List. Available from: http://www.worldbirdnames.org/ [last accessed 16 Dec 2013].

Ito T, Gorman OT, Kawaoka Y, et al. (1991). Evolutionary analysis of the influenza-A virus M-gene with comparison of the M1 and M2 proteins. J Virol 65:5491–8.

Ito T, Goto H, Yamamoto E, et al. (2001). Generation of a highly pathogenic avian influenza A virus from an avirulent field isolate by passaging in chickens. J Virol 75:4439–43.

Janout V, Uvízl M, Chmela J, et al. (1979). A study on the role of birds in the spread of infections. J Hyg Epidemiol Microbiol Immunol 23: 457–61.

Järhult J D, Muradrasoli S, Wahlgren J, et al. (2011). Environmental levels of the antiviral oseltamivir induce development of resistance mutation H274Y in influenza A/H1N1 virus in mallards. PLoS One 6: e24742.

Jourdain E, Gauthier-Clerc M, Bicout D, Sabatier P. (2007). Bird migration routes and risk for pathogen dispersion into western Mediterranean wetlands. Emerg Infect Dis 13:365–72.

Jourdain E, Gunnarsson G, Wahlgren J, et al. (2010). Influenza virus in a natural host, the mallard: experimental infection data. PLoS One 5: e8935.

Jourdain E, Van Riel D, Munster VJ, et al. (2011). The pattern of influenza virus attachment varies among wild bird species. PLoS One 6: e24155.

Kang HM, Choi JG, Kim MC, et al. (2012). Isolation of a reassortant H13N2 virus from a mallard fecal sample in South Korea. Virol J 9: 133–36.

Kawaoka Y, Chambers TM, Sladen WL, Webster RG. (1988). Is the gene pool of influenza viruses in shorebirds and gulls different from that in wild ducks? Virology 163:247–50.

Kawaoka Y, Gorman OT, Ito T, et al. (1998). Influence of host species on the evolution of the nonstructural (NS) gene of influenza A viruses. Virus Res 55:143–56.

Keeling MJ, Rohani P. (2008). Modeling infectious diseases in humans and animals. Princeton: Princeton University Press.

Kleijn D, Munster VJ, Ebbinge BS, et al. (2010). Dynamics and ecological consequences of avian influenza virus infection in greater white-fronted geese in their winter staging areas. Proc Biol Sci 277: 2041–8.

Koehler AV, Pearce JM, Flint PL, et al. (2008). Genetic evidence of intercontinental movement of avian influenza in a migratory bird: the northern pintail (*Anas acuta*). Mol Ecol 17:4754–62.

Kohls A, Hafez HM, Harder T, et al. (2011). Avian influenza virus risk assessment in falconry. Virol J 8:187–90.

Kou Z, Li Y, Yin Z, et al. (2009). The survey of H5N1 flu virus in wild birds in 14 provinces of China from 2004 to 2007. PLoS One 4: e6926.

Krauss S, Obert CA, Franks J, et al. (2007). Influenza in migratory birds and evidence of limited intercontinental virus exchange. PLoS Pathog 3:e167.

Kuchipudi SV, Nelli R, White GA, et al. (2009). Differences in influenza virus receptors in chickens and ducks: implications for interspecies transmission. J Mol Genet Med 3:143–51.

Perkins LE, Swayne DE. (2001). Pathobiology of A/chicken/Hong Kong/220/97 (H5N1) avian influenza virus in seven gallinaceous species. Vet Pathol Online 38:149–64.

L'vov DK, Andreev VP, Braude NA, et al. (1978). Isolation of influenza virus with the antigenic formula Hav4 Nav2 and Hav5 Nav2 during epizootic infection among sea gulls in the Astrakhan district in the summer of 1976. Vopr Virusol 4:399–403.

L'vov DK, Shchelkanov MI, Deriabin PG, et al. (2006). Isolation of highly pathogenic avian influenza (HPAI) A/5H5N1 strains from wild birds in the epizootic outbreak on the Ubsu-Nur Lake (June 2006) and their incorporation to the Russian Federation State Collection of viruses. Vopr Virusol 51:14–18.

L'vov DK, Yamnikova S, Gambaryan A, et al. (2001). Isolation of influenza viruses from wild birds in the Volga river basin and in the North Caspian region. International Congress Series 1219:251–8.

Lam TTY, Ip HS, Ghedin E, et al. (2012). Migratory flyway and geographical distance are barriers to the gene flow of influenza virus among North American birds. Ecol Lett 15:24–33.

Latorre-Margalef N, Grosbois V, Wahlgren J, et al. (2013). Heterosubtypic immunity to influenza A virus infections in Mallards may explain existence of multiple virus subtypes. PLoS Pathog 9: e1003443.

Latorre-Margalef N, Gunnarsson G, Munster VJ, et al. (2009). Effects of influenza A virus infection on migrating mallard ducks. Proc Biol Sci 276:1029–36.

Laudert E, Sivanandan V, Halvorson D, et al. (1993). Biological and molecular characterization of H13N2 influenza type A viruses isolated from turkeys and surface water. Av Dis 37:793–9.

Lebarbenchon C, Brown SAM, Poulin R, et al. (2008). Evolution of pathogens in a man-made world. Mol Ecol 17:475–84.

Lebarbenchon C, Chang CM, Gauthier-Clerc M, et al. (2009). H9N2 avian influenza virus in a Mediterranean gull. J Mol Genet Med 3: 121–3.

Lebarbenchon C, Chang CM, Grandhomme V, et al. (2010b). Avian influenza circulation in the Camargue (south of France) during the 2006–07 season. Avian Dis 54:446–9.

Lebarbenchon C, Chang CM, Van Der Werf S, et al. (2007). Influenza A virus in birds during spring migration in the Camargue, France. J Wildl Dis 43:789–93.

Lebarbenchon C, Feare CJ, Renaud F, et al. (2010a). Persistence of highly pathogenic avian influenza viruses in natural ecosystems. Emerg Infect Dis 16:1057–62.

Lee KA. (2006). Linking immune defenses and life history at the levels of the individual and the species. Integr Comp Biol 46:1000–15.

Lee MS, Chang PC, Shien JH, et al. (2001). Identification and subtyping of avian influenza viruses by reverse transcription-PCR. J Virol Met 97:13–22.

Leroy EM, Kumulungui B, Pourrut X, et al. (2005). Fruit bats as reservoirs of Ebola virus. Nature 438:575–6.

Lewis NS, Javakhishvili Z, Russell CA, et al. (2013). Avian influenza virus surveillance in wild birds in Georgia: 2009-2011. PLoS ONE 8: e58534.

Li KS, Guan Y, Wang J, et al. (2004). Genesis of a highly pathogenic and potentially pandemic H5N1 influenza virus in eastern Asia. Nature 430:209–13.

Lin B, Malanoski AP, Wang Z, et al. (2009). Universal detection and identification of avian influenza virus by use of resequencing microarrays. J Clin Microbiol 47:988–93.

Lindskog C, Ellström P, Olsen B, et al. (2013). European H16N3 gull influenza virus attaches to the human respiratory tract and eye. PLoS One 8:e60757.

Lisnizer N, Garcia-Borboroglu P, Yorio P. (2011). Spatial and temporal variation in population trends of Kelp gulls in northern Patagonia, Argentina. Emu 111:259–67.

Liu J, Xiao H, Lei F, Zhu Q, et al. (2005). Highly pathogenic H5N1 influenza virus infection in migratory birds. Science 309:1206.

Loehle C. (1995). Social barriers to pathogen transmission in wild animal populations. Ecology 76:326–35.

Lowen AC, Mubareka S, Steel J, Palese P. (2007). Influenza virus transmission is dependent on relative humidity and temperature. PLoS Pathog 3:e151.

Lu X, Qi J, Shi Y, Wang M, et al. (2013). Structure and receptor binding specificity of Hemagglutinin H13 from avian influenza A virus H13N6. J Virol 87:9077–85.

Mackenzie JS, Edwards EC, Holmes RM, Hinshaw VS. (1984). Isolation of ortho-and paramyxoviruses from wild birds in Western Australia, and the characterization of novel influenza A viruses. Aust J Exp Biol Med Sci 62:89–99.

Marchenko VI, Alekseev AI, Susloparov IM, et al. (2010). [Isolation of influenza A virus from wild birds in western part of Mongolia]. Zh Mikrobiol Epidemiol Immunobiol 6:18–21.

Maxted AM, Luttrell MP, Goekjian VH, et al. (2012). Avian influenza virus infection dynamics in shorebird hosts. J Wildl Dis 48:322–34.

Mehrabanpour MJ, Rahimian A, Shirazinezhad A, et al. (2012). Avian influenza virus in migratory and resident birds during migratory season in Boushehr, Iran. Turk J Vet Anim Sci 36:446–50.

Meijer A, Lackenby A, Hungnes O, et al. (2009). Oseltamivir-resistant influenza virus A (H1N1), Europe, 2007–08 season. Emerg Infect Dis 15:552–60.

Monot M, Honoré N, Garnier T, et al. (2009). Comparative genomic and phylogeographic analysis of Mycobacterium leprae. Nat Genet 41: 1282–9.

Moscona A. (2009). Global transmission of oseltamivir-resistant influenza. New Engl J Med 360:953–6.

Munster VJ, Baas C, Lexmond P, et al. (2007). Spatial, temporal, and species variation in prevalence of influenza A viruses in wild migratory birds. PLoS Pathog 3:e61.

Munster VJ, Fouchier RAM. (2009). Avian influenza virus: of virus and bird ecology. Vaccine 27:6340–4.

Muzinic J, Feare CJ, Pavlak M, et al. (2010). The epidemiological environment of avian influenza H5N1 outbreaks in wild birds in Croatia. Pol J Environ Stud 19:379–85.

NIAID (2012). National Institute of Allergy and infectious Diseases. Flu (Influenza). Available from: http://www.niaid.nih.gov/topics/flu/Pages/default.aspx [last accessed 9 Apr 2013].

Nicholls JM, Chan RWY, Russell RJ, et al. (2008). Evolving complexities of influenza virus and its receptors. Trends Microbiol 16:149–57.

Obenauer JC, Denson J, Mehta PK, et al. (2006). Large-scale sequence analysis of avian influenza isolates. Science 311:1576–80.

Olsen B, Munster VJ, Wallensten A, et al. (2006). Global patterns of influenza A virus in wild birds. Science 312:384–8.

Oro D, Cam E, Pradel R, Martinez-Abrain A. (2004). Influence of food availability on demography and local population dynamics in a long-lived seabird. Proc Biol Sci 271:387–96.

Orozovic G, Orozovic K, Lennerstrand J, Olsen B. (2011). Detection of resistance mutations to antivirals oseltamivir and zanamivir in avian influenza a viruses isolated from wild birds. PLoS One 6:e16028.

Otsuki K, Kawaoka Y, Nakamura T, Tsubokura M. (1982). Pathogenicity for chickens of avian influenza viruses isolated from whistling swans and a black-tailed gull in Japan. Av Dis 26:314–20.

Otsuki K, Takemoto O, Fujimoto R, et al. (1987). Isolation of influenza A viruses from migratory waterfowls in San-in District, Western Japan in winters of 1980-1982. ZBL Hyg Umweltmed 265:235–42.

Patz JA, Daszak P, Tabor GM, et al. (2004). Unhealthy landscapes: policy recommendations on land use change and infectious disease emergence. Environ Health Perspect 112:1092–8.

Pearce JM, Ramey AM, Flint PL, et al. (2009). Avian influenza at both ends of a migratory flyway: characterizing viral genomic diversity to optimize surveillance plans for North America. Evol Appl 2:457–68.

Pearce JM, Ramey AM, Ip HS, Gill Jr RE. (2010). Limited evidence of trans-hemispheric movement of avian influenza viruses among contemporary North American shorebird isolates. Virus Res 148: 44–50.

Pearce JM, Reeves AB, Ramey AM, et al. (2011). Interspecific exchange of avian influenza virus genes in Alaska: the influence of trans-hemispheric migratory tendency and breeding ground sympatry. Mol Ecol 20:1015–25.

Pearce-Duvet JMC, Gauthier-Clerc M, Jourdain E, Boulinier T. (2009). Maternal antibody transfer in yellow-legged gulls. Emerg Infect Dis 15:1147–9.

Peiris JSM, Guan Y, Yuen KY. (2004). Severe acute respiratory syndrome. Nat Med 10:S88–97.

Pepin KM, VanDalen KK, Mooers NL, et al. (2012). Quantification of heterosubtypic immunity between avian influenza subtypes H3N8 and H4N6 in multiple avian host species. J Gen Virol 93:2575–83.

Pereda AJ, Uhart M, Perez AA, et al. (2008). Avian influenza virus isolated in wild waterfowl in Argentina: evidence of a potentially unique phylogenetic lineage in South America. Virology 378:363–70.

Pérez-Tris J, Hellgren O, Križanauskienė A, et al. (2007). Within-host speciation of malaria parasites. PLoS ONE 2:e235.

Pillai SP, Lee CW. (2010). Species and age related differences in the type and distribution of influenza virus receptors in different tissues of chickens, ducks and turkeys. Virol J 7:5–12.

Pourrut X, Kumulungui B, Wittmann T, et al. (2005). The natural history of Ebola virus in Africa. Microbes Infect 7:1005–14.

Ramey AM, Pearce JM, Ely CR, et al. (2010). Transmission and reassortment of avian influenza viruses at the Asian–North American interface. Virology 406:352–9.

Ramis A, Van Amerongen G, Van de Bild M, et al. (2012). Experimental highly pathogenic avian influenza virus H5N1 infection in black-headed gulls (*Chroicocephalus ridibundus*). J Comp Pathol 146:88.

Ratanakorn P, Wiratsudakul A, Wiriyarat W, et al. (2012). Satellite tracking on the flyways of *Brown-Headed Gulls* and their potential role in the spread of highly pathogenic avian influenza H5N1 virus. PLoS One 7:e49939.

Raven SJ, Coulson JC. (1997). The distribution and abundance of Larus gulls nesting on buildings in Britain and Ireland. Bird Study 44:13–34.

Reeves AB, Pearce JM, Ramey AM, et al. (2011). Interspecies transmission and limited persistence of low pathogenic avian influenza genomes among Alaska dabbling ducks. Infect Genet Evol 11: 2004–10.

Röhm C, Horimoto T, Kawaoka Y, et al. (1995). Do hemagglutinin genes of highly pathogenic avian influenza viruses constitute unique phylogenetic lineages? Virology 209:664–70.

Sakoda Y, Ito H, Uchida Y, et al. (2012). Reintroduction of H5N1 highly pathogenic avian influenza virus by migratory water birds, causing poultry outbreaks in the 2010–2011 winter season in Japan. J Gen Virol 93:541–50.

Sandrock C, Kelly T. (2007). Clinical review: update of avian influenza A infections in humans. Crit Care 11:209–17.

Savić V, Labrović A, Zelenika TA, et al. (2010). Multiple introduction of Asian H5N1 avian influenza virus in Croatia by wild birds during 2005–2006 and isolation of the virus from apparently healthy black-headed gulls (*Larus ridibundus*). Vector Borne Zoonotic Dis 10: 915–20.

Schekkerman H, Slaterus R. (2008). Population dynamics and prevalence of influenza A viruses in mallard, mute swan and other wildfowl. Avian influenza and wildfowl populations, British Trust for Ornithology (BTO), The Nunnery, Thetford, Norfolk.

Sharshov K. (2010). Avian influenza (H5N1) outbreak among wild birds, Russia, 2009. Emerg Infect Dis 16:349–51.

Shinya K, Ebina M, Yamada S, et al. (2006). Avian flu: influenza virus receptors in the human airway. Nature 440:435–6.

Sivanandan V, Halvorson DA, Laudert E, et al. (1991). Isolation of H13N2 influenza A virus from turkeys and surface water. Avian Dis 35:974–7.

Slepuskin AN, Pysina TV, Gonsovsky FK, et al. (1972). Haemagglutination-inhibiting activity to type a influenza viruses in the sera of wild birds from the far east of the USSR. Bull World Health Org 47:527–30.

Spackman E, Swayne DE, Gilbert M, et al. (2009). Characterization of low pathogenicity avian influenza viruses isolated from wild birds in Mongolia 2005 through 2007. Virol J 6:190–97.

Stallknecht DE, Brown JD. (2007). Wild birds and the epidemiology of avian influenza. J Wildli Dis 43:S15–20.

Stallknecht DE, Kearney MT, Shane SM, Zwank PJ. (1990). Effects of pH, temperature, and salinity on persistence of avian influenza viruses in water. Avian Dis 34:412–18.

Stallknecht DE, Shane SM. (1988). Host range of avian influenza virus in free-living birds. Vet Res Commun 12:125–41.

Staszewski V, Gasparini J, Mccoy K, et al. (2007a). Evidence of an interannual effect of maternal immunization on the immune response of juveniles in a long-lived colonial bird. J Anim Ecol 76:1215–23.

Staszewski V, Mccoy K, Tveraa T, Boulinier T. (2007b). Interannual dynamics of antibody levels in naturally infected long-lived colonial birds. Ecology 88:3183–91.

Suarez DL, Perdue ML. (1998). Multiple alignment comparison of the non-structural genes of influenza A viruses. Virus Res 54:59–69.

Suzuki Y. (2005). Sialobiology of influenza: molecular mechanism of host range variation of influenza viruses. Biol Pharm Bull 28: 399–408.

Tønnessen R, Germundsson A, Jonassen CM, et al. (2011). Virological and serological surveillance for type A influenza in the black-legged kittiwake (*Rissa tridactyla*). Virol J 8:21–27.

Tønnessen R, Hauge AG, Hansen EF, et al. (2013a). Host restrictions of avian influenza viruses: in silico analysis of H13 and H16 specific signatures in the internal proteins. PLoS ONE 8:e63270.

Tønnessen R, Kristoffersen AB, Jonassen CM, et al. (2013b). Molecular and epidemiological characterization of avian influenza viruses from gulls and dabbling ducks in Norway. Virol J 10:112–22.

Tong S, Li Y, Rivailler P, Conrardy C, et al. (2012). A distinct lineage of influenza A virus from bats. Proc Natl Acad Sci 11:4269–74.

Tolf C, Latorre-Margalef N, Wille M, et al. (2013). Individual variation in Influenza A Virus infection histories and long-term immune responses in Mallards. PLoS-ONE 8:e61201.

Tsubokura M, Otsuki K, Yamamoto H, et al. (1981). Isolation of an Hswl Nav4 influenza virus from a tufted duck (*Aythya fuligula*) in Japan. Microbiol Immunol 25:819–25.

USDA (2012). USDA–APHIS–APHIS Newsroom. Available from http://www.aphis.usda.gov/ [last accessed 16 Dec 2013].

Van Borm S, Rosseel T, Vangeluwe D, et al. (2012). Phylogeographic analysis of avian influenza viruses isolated from Charadriiformes in Belgium confirms intercontinental reassortment in gulls. Arch Virol 157:1509–22.

van Gils JA, Munster VJ, Radersma R, et al. (2007). Hampered foraging and migratory performance in swans infected with low-pathogenic avian influenza A virus. PLoS ONE 2:e184.

Velarde R, Calvin SE, Ojkic D, et al. (2010). Avian influenza virus H13 circulating in ring-billed gulls (*Larus delawarensis*) in southern Ontario, Canada. Avian Dis 54:411–19.

Verhagen JH, Munster VJ, Majoor F, et al. (2012). Avian influenza A virus in wild birds in highly urbanized areas. PLOS One 7:e38256.

Vollmer SA, Bormane A, Dinnis RE, et al. (2011). Host migration impacts on the phylogeography of Lyme Borreliosis spirochaete species in Europe. Environ Microbiol 13:184–92.

Wallensten A, Munster VJ, Latorre-Margalef N, et al. (2007). Surveillance of influenza virus A in migratory waterfowl in northern Europe. Emerg Infect Dis 13:404–11.

Wang L, Eaton B. (2007). Bats, civets and the emergence of SARS. In: Childs JE, Mackenzie JS, Richt JA eds. Wildlife and emerging zoonotic diseases: the biology, circumstances and consequences of cross-species transmission. current topics in microbiology and immunology. Berlin, Heidelberg: Springer. 325–344.

Webster RG, Bean WJ, Gorman OT, et al. (1992). Evolution and ecology of influenza A viruses. Microbiol Mol Biol Rev 56:152–79.

Webster RG, Yakhno M, Hinshaw VS, et al. (1978). Intestinal influenza: replication and characterization of influenza viruses in ducks. Virology 84:268–78.

Widjaja L, Krauss SL, Webby RJ, et al. (2004). Matrix gene of influenza A viruses isolated from wild aquatic birds: ecology and emergence of influenza a viruses. J Virol 78:8771–79.

Wille M, Robertson GJ, Whitney H, et al. (2011). Extensive geographic mosaicism in avian influenza viruses from gulls in the northern hemisphere. PLoS One 6:e20664.

Winker K, Gibson DD. (2010). The Asia-to-America influx of avian influenza wild bird hosts is large. Avian Dis 54:477–82.

Winker K, McCracken KG, Gibson DD, et al. (2007). Movements of birds and avian influenza from Asia into Alaska. Emerg Infect Dis 13:547–52.

Winker K, Spackman E, Swayne DE. (2008). Rarity of influenza A virus in spring shorebirds, southern Alaska. Emerg Infect Dis 14:1314–16.

Wirth T, Meyer A, Achtman M. (2005). Deciphering host migrations and origins by means of their microbes. Mol Ecol 14:3289–306.

Wolfe ND, Daszak P, Kilpatrick AM, Burke DS. (2005). Bushmeat hunting, deforestation, and prediction of zoonoses emergence. Emerg Infect Dis 11:1822–27.

Yamnikova SS, Gambaryan AS, Tuzikov AB, et al. (2003). Differences between HA receptor-binding sites of avian influenza viruses isolated from Laridae and Anatidae. Avian Dis 47:1164–8.

Yao L, Korteweg C, Hsueh W, Gu J. (2008). Avian influenza receptor expression in H5N1-infected and noninfected human tissues. The FASEB Journal 22:733–740.

Zakstel'skaja LJ, Isacenko VA, Osidze NG, et al. (1972). Some observations on the circulation of influenzaviruses in domestic and wild birds. Bull World Health Org 47:497–501.

Zhu X, Yang H, Guo Z, et al. (2012). Crystal structures of two subtype N10 neuraminidase-like proteins from bat influenza A viruses reveal a diverged putative active site. Proc Natl Acad Sci 46:18903–8.