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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.

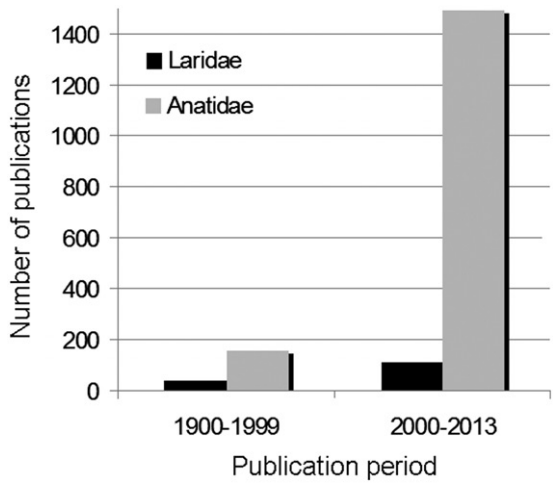


Figure 1. Number of studies that have focused on AIVs in Anatidae versus Laridae from 1899 to July 3, 2013. Research led on Web of Science using respectively the terms: ''Influenza AND gull OR tern OR kittiwake'' and ''Influenza AND duck OR geese OR swan''.

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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