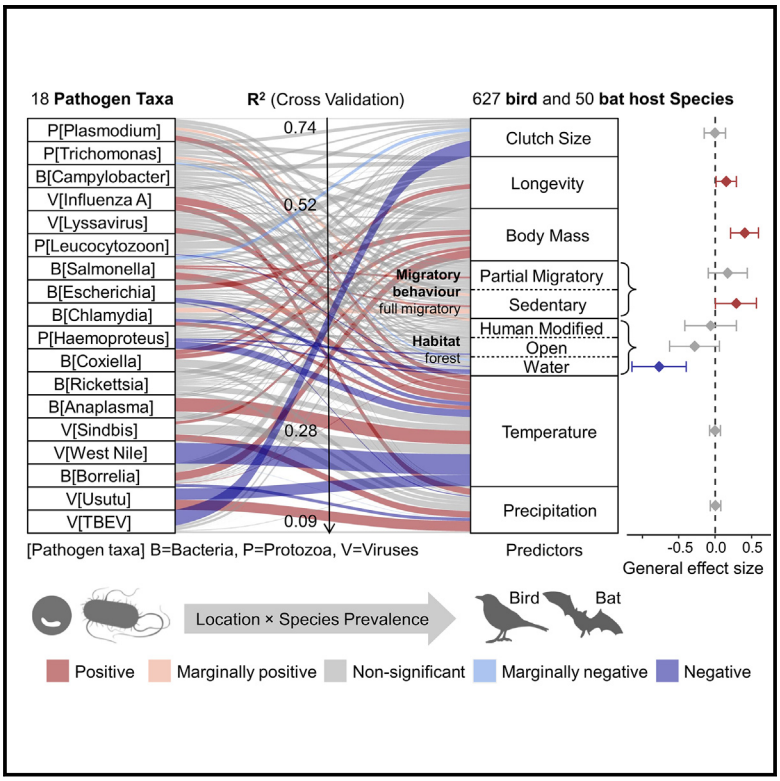
One Earth

Article

Slow-lived birds and bats carry higher pathogen loads

Graphical abstract



Highlights

- Species trait, distribution, and climate predict large-scale pathogen prevalence
- Bird and bat species with a slower pace of life are more likely to carry pathogens
- Sedentary and forest species have a higher pathogen prevalence
- Temperature is the most important predictor for pathogen prevalence

Authors

Yanjie Xu, Veronika N. Laine, Katarina Meramo, ..., Arto T. Pulliainen, Thomas M. Lilley, Aleksi Lehikoinen

Correspondence

yanjie.xu@helsinki.fi

In brief

Emerging infectious diseases, driven by wildlife and zoonotic pathogens, are escalating threats to human society, domestic animals, and wildlife populations. Our research demonstrates that species characteristics and climatic conditions significantly influence pathogen infections in wildlife hosts, increasing the potential for spillover to humans. By focusing on birds and bats, we have mapped disease risks across Europe and developed a forecasting model to predict outbreaks. This model aids in focusing surveillance efforts and underscores the importance of understanding the interplay between ecological and climatic factors in managing emerging diseases under climate change.

Xu et al., 2024, One Earth 7, 1121–1132
June 21, 2024
https://doi.org/10.1016/j.oneear.2024.04.021

CellPress

One Earth

CellPress
OPEN ACCESS

Article

Slow-lived birds and bats carry higher pathogen loads

Yanjie Xu,[1,9,*] Veronika N. Laine,[1] Katarina Meramo,[1] Andrea Santangeli,[2] Anbu Poosakkannu,[1,3] Kati M. Suominen,[1] Simon Gaultier,[4] Verena Keller,[5] Lluís Brotons,[5,6,7,8] Arto T. Pulliainen,[3] Thomas M. Lilley,[1] and Aleksi Lehikoinen[1,5]

[1]Finnish Museum of Natural History, University of Helsinki, 00014 Helsinki, Finland
[2]Animal Demography and Ecology Unit, Institute for Mediterranean Studies (IMEDEA), CSIC-UIB, 07190 Esporles, Spain
[3]Institute of Biomedicine, University of Turku, 20520 Turku, Finland
[4]Department of Biology, University of Turku, 20500 Turku, Finland
[5]European Bird Census Council, Na Bělidle 34, CZ-150 00 Prague 5, Czech Republic
[6]Ecological and Forestry Applications Research Centre (CREAF), 08193 Cerdanyola del Vallès, Spain
[7]Forest Science and Technology Centre (CTFC), Carretera vella de Sant Llorenç de Morunys km 2, 25280 Solsona, Spain
[8]CSIC, 08193 Cerdanyola del Vallès, Spain
[9]Lead contact
*Correspondence: yanjie.xu@helsinki.fi
https://doi.org/10.1016/j.oneear.2024.04.021

SCIENCE FOR SOCIETY Pathogens—microorganisms that cause disease—can be transmitted via wild animals, potentially triggering severe infectious disease outbreaks in humans, domestic animals, and wildlife. The risk of these diseases is influenced by the pathogen loads carried by mobile hosts such as birds and bats, which vary with different climatic conditions and host species assemblages. By analyzing host species traits and climate conditions across Europe, we have pinpointed areas at elevated risk for disease transmission. Our study bridges the gap between wildlife ecology and public health by offering a predictive tool for identifying disease hotspots, facilitating proactive mitigation efforts. This interdisciplinary research highlights the critical need to anticipate and address emerging infectious diseases, thus advancing the health of ecosystems and human societies alike, aligning with broader sustainability goals.

SUMMARY

Wildlife and zoonotic diseases are increasingly impacting human society, the food chain, and wildlife; therefore, proactive mitigation tools for predicting large-scale risk of the relevant pathogens are urgently needed. Birds and bats are large-scale disease reservoirs and transmitters. However, holistic understanding for which bird and bat species act as reservoirs for pathogens remains understudied. Here, we test the extent to which the features related to the mobile species and local climate identify reservoir hosts for the 18 most-sampled pathogens across Europe. Species with slower pace of life (i.e., larger bodied and longer lived), sedentary species, and forest species had high pathogen prevalence. Temperature was the most important predictor for pathogen prevalence, but its effects varied in different directions. Overall, host species traits and climatic gradients robustly predicted pathogen prevalence, especially for non-vector-transmitted pathogens. We offer a data-driven basis for developing targeted interventions to mitigate impacts of zoonotic diseases, particularly in the face of climate change.

INTRODUCTION

Emerging infectious disease events caused by zoonotic pathogens, such as the COVID-19 pandemic, are increasingly affecting human society.[1] Similarly, many wildlife pathogens cause severe infections in wild animals, negatively impacting biodiversity,[2] and in domestic animals, posing a risk to the food chain.[3] Knowledge on wild-host distribution and probability of carrying pathogens is crucial in forecasting infectious disease risk within any geographical area. The wild hosts include mobile vertebrates, especially birds and bats, which are important natural and incidental pathogen reservoirs.[4–6] Birds and bats are indeed recognized reservoirs, large-scale transmitters, and/or cross-species sharing nodes for pathogens causing pandemics and panzootics (e.g., for highly pathogenic avian influenza viruses and coronaviruses).[5–7] Knowledge on which bird and bat species act as reservoirs for pathogens and in which geographical location are thereby urgently needed for large-scale

proactive risk assessment for zoonotic and wildlife disease emergence.[8]

Zoonotic and wildlife disease risks across geographical areas exhibit a spatiotemporally dynamic pattern, especially when considering birds and bats. A large proportion of birds and bats are migratory with variability in occurrence across seasons, resulting in seasonally distinct spatial patterns of disease risk.[9] Besides host distributions being a prerequisite for pathogen presence, climatic variation can also alter the extent to which host populations are exposed and susceptible to a pathogen.[10] Further, climatic conditions affect the abundance and density of pathogen-transmitting vectors (e.g., mosquitos and ticks), thereby causing varying spatial disease risks.[11]

Disease risks also vary among different host species/taxa based on functional traits related to pace of life, habitats, and dispersal patterns. For example, in rodents, species that reproduce slowly (hereafter slow-lived species, which are those with a longer lifespan but lower fecundity), are less likely to act as reservoir hosts for zoonotic pathogens compared to those reproducing fast (hereafter fast-lived species).[12] A negative relationship between body mass and the ability of a host species to transmit pathogens to another susceptible host has been observed in a few pathogens (e.g., *Borrelia* spp. and West Nile viruses).[13–15] Fast-lived species may be more susceptible and suitable for hosting pathogens because of their lower investment in immune defenses.[16] However, the mechanism may be different for large-scale disease patterns in mobile hosts. Larger-bodied and longer-lived waterbirds (i.e., slow-lived species) were found to be more likely to host highly pathogenic avian influenza viruses (HPAIVs) in Europe.[17] A similar result was also shown at a global scale; i.e., longer-lived bird species were more likely to favor tick-mediated transmission for *Borrelia burgdorferi*.[18]

Migratory species occupy spatially disconnected areas within their annual cycle, facilitating escape from pathogen-polluted environments and reducing their parasite burdens (i.e., migratory escape).[9,19] Contradictorily, migration can increase disease burdens in mobile hosts by compromising their immune systems and exposing novel pathogens to naive geographical populations.[20,21] Differing habitat preferences may also play a role in varying zoonotic potential among different host species. As an example, waterbirds that use wetland and coastal areas as their primary habitats are commonly considered as wild reservoirs for influenza A viruses.[22] The habitat preference of invertebrate vectors may also play a major role in explaining pathogen prevalence in host species; e.g., forests are preferred habitats for ticks and mosquitos.[23]

Life history and ecological traits of host species, host distributions, and climatic gradients may be entwined drivers for the spatial pattern of pathogen prevalence at a macroecological scale. However, important knowledge gaps remain for predictors for large-scale pathogen risk patterns in mobile hosts because these predictors have rarely been integrated in pathogen mapping, and different pathogens are usually tested individually with different modeling systems at different scales. The most commonly applied approach to mapping pathogen distributions is "host filling,"[24] which uses host-pathogen associations to define the entire host distribution ranges as potential pathogen distribution.[24] This approach often overestimates the spatial distribution of disease risks, because it neglects the varying suitability for pathogens and hosts and the prevalence of pathogens in a host species across areas with different climatic conditions and across different host species.[24] Other approaches usually underestimate the distributional ranges of pathogens because they rely on the pathogen survey efforts that are often spatially biased.[24] One of these approaches is the "climatic model" method, which models the distribution of pathogens according to their suitable climatic conditions using observations of pathogen presence across geographical areas.[24]

To the best of our knowledge, there is only one published continental-scale study in birds, which tests the climate-driven prevalence of multiple pathogens across different locations (climatic model alone).[10] However, the spatial variation in overall disease risk due to varying structure of host species communities under different climatic conditions has not been estimated. In addition to climatic variables, disease risk mapping should incorporate knowledge on both host specificity of pathogens and the spatiotemporal occurrence of mobile hosts (host filling). Here, we use a robust approach, combining host-filling and climatic-model methods (Figure 1), to test the extent to which integrating these entwined drivers can predict spatial risks of different pathogens from birds and bats. Instead of presence-absence data, we used a unique pan-European pathogen prevalence database[25] to model the prevalence of the 18 most-sampled pathogenic microbial taxa (hereafter pathogen taxa, which covers viruses, bacteria and protozoa; Table 1) among various bird and bat species at different locations. These pathogen taxa were defined as those with either zoonotic or wildlife disease potential (Table 1), which can potentially cause disease at least in some wild and domestic animals or humans.

Here, we investigate the potential predictors of pathogen prevalence across various host species and regions by utilizing temperature, precipitation, and life history traits (including clutch size for birds, maximum longevity, and body mass for both birds and bats), primary habitats, and migratory behavior. We specifically tested how these factors correlate with the prevalence of pathogens among different mobile host species across Europe and surrounding areas. Our findings support the hypothesis that slower-lived, sedentary bird and bat species in warmer regions tend to host a higher load of pathogens compared to their faster-lived, migratory counterparts in colder climates. This variation in pathogen prevalence also appears to be influenced by the primary habitat of the host species, with certain pathogens more prevalent in specific environments such as forests for tick-borne and mosquito-borne diseases. Additionally, our models indicate that non-vector-borne pathogens are more directly predicted by these variables than those pathogens requiring invertebrate vectors. These results not only corroborate most of our initial hypotheses but also highlight the complexity of pathogen transmission among mobile hosts. Overall, we suggest that the integration of host species traits with climatic data offers a robust framework for predicting large-scale patterns of zoonotic and wildlife disease risks. This approach provides essential insights into the macroecological drivers of disease distribution and establishes a dynamic operational model for prioritizing disease surveillance and intervention strategies regionally.

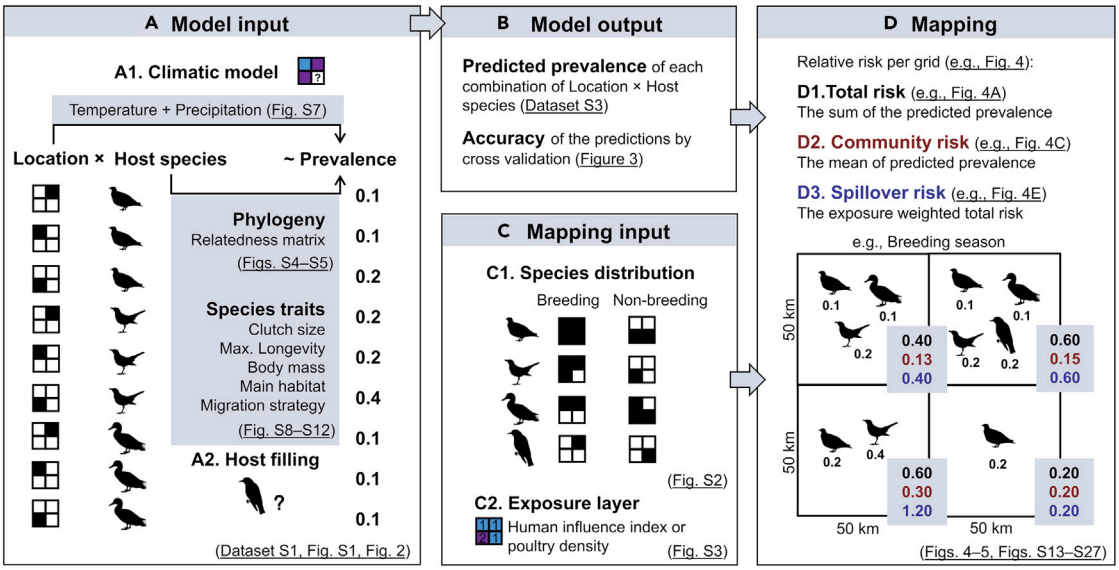


Figure 1. A schematic framework for the workflow of spatial risk mapping

Here we used a combined approach where both host filling and climatic model methods are included.

(A–D) The input data collected and organized for modeling (A) are available at Data S1. The modeling outputs (B) included the predicted pathogen prevalence per host species per location. In addtion to the modeling outputs, we added species distribution and exposure layer (C) for mapping the pathogen risks per 50×50 km grid acoss Europe (D). In (D), the value below a host species is the predicted prevalence for that species in that spatial grid.

RESULTS

We modeled the prevalence of the 18 most-sampled pathogen taxa (Table 1) in European birds (617 species in breeding season and 313 species in non-breeding season) and bats (50 species) with their phylogeny, life history and ecological traits and spatial climatic variables (temperature and rainfall). The fitted models quantified associations between pathogen prevalence and the predictors (Figures 2 and S7–S12) and then yielded species-specific predictions of prevalence for each pathogen taxon across Europe.

Temperature was the most important predictor for pathogen prevalence, with a variable importance of 26.7%, while precipitation had a lower importance of 11.2%. There was no significant direction (positive or negative) of the general effects of temperature and precipitation across different pathogen taxa (Figure 2B). Pathogen specifically, these climatic variables had significant associations with 13 out of the 18 studied pathogen taxa. However, the directions of these pathogen-specific associations differed among pathogen taxa (Figure 2B).

Species life history traits (i.e., clutch size for birds, longevity and body mass for birds and bats) had a considerable effect on the prevalence of 18 pathogen taxa (Figure 2A), with a total importance of ~35% (variable importance: clutch size 9.7%, longevity 12.6%, body mass 12.6%). Specifically. across different pathogen taxa, longer-lived and larger-bodied bird and bat species had a significantly higher prevalence (Figure 2B). *Coxiella* spp. showed significantly positive associations with the hosts' longevity (Figure 2A). Species with a larger body mass consistently showed a significantly higher prevalence of four pathogen taxa (i.e., *Escherichia* spp., *Coxiella* spp., Sindbis viruses, *Borrelia* spp.; Figure 2A). Clutch size of bird species was significantly or marginally negatively associated with prevalence of tick-borne encephalitis viruses (TBEVs) and *Salmonella* spp. (Figure 2A).

Ecological traits are also important variables for predicting pathogen prevalence, whereby species migratory behavior and main habitat type had a variable importance of 13.9% and 14.1%, respectively (Figure 2A). Among the studied pathogen taxa, sedentary species had a significantly higher prevalence than full migratory species (Figure 2B). Prevalence of *Trichonomas* spp., *Salmonella* spp., and *Chlamydia* spp. in full migratory birds was significantly or marginally lower than that in partial migratory and sedentary species (Figure 2A). Moreover, host species that prefer water habitats showed a significantly lower pathogen prevalence (Figure 2B) than forest species, while those preferring human-modified and open habitats did not show a significant difference from forest species (Figure 2A). With regard to specific pathogens, the prevalence of *Plasmodium* spp., *Trichonomas* spp., *Leucocytozoon* spp., *Chlamydia* spp., *Haemoproteus* spp., *Anaplasma* spp., Sindbis viruses, and TBEV was significantly or marginally lower in waterbirds than in forest birds. Birds that mainly use open habitats also showed a significantly lower prevalence of *Haemoproteus* spp. The prevalence of *Plasmodium* spp. was marginally higher in bird species associated with human-modified habitat compared to forest birds, while Sindbis viruses showed the opposite pattern (Figure 2A).

According to cross-validations (Figure 3), different models for different pathogen taxa showed varying accuracies, which was not necessarily related to the sample size but related to their taxa groups (i.e., bacteria, protozoa, or virus) and transmission modes (i.e., vector or non-vector). Models for *Plasmodium* spp., *Trichomonas* spp., *Campylobacter* spp., and influenza A viruses in birds showed the highest predictive accuracy (0.62–0.74) among the 18 models of different pathogen taxa, while the model for TBEVs in birds showed the lowest accuracy

Table 1. Sample size and general information of the included pathogen taxa (17 in birds and one in bats)

Pathogen taxon		Disease potential (Z/W)	Transmission mode	Host (*N* species)	*N* observation	*N* sampled host individual	Observed prevalence across taxa and regions (%)	Predicted prevalence across taxa and regions (%)	Model accuracy (mean R^2 ± SD)	Potential exposure risk for spillover
Bacteria	*Campylobacter*	Z/W	non-vector	bird (182)	459	13,023	20.7	16.0	0.64 ± 0.21	human
	Salmonella	Z/W	non-vector	bird (119)	301	18,486	6.9	6.0	0.44 ± 0.21	human
	Escherichia	Z/W	non-vector	bird (100)	255	11,647	29.5	28.2	0.41 ± 0.20	human
	Chlamydia	Z/W	non-vector	bird (119)	215	6,885	16.0	18.7	0.41 ± 0.23	human
	Coxiella	Z/W	vector	bird (111)	130	1,748	5.1	4.5	0.34 ± 0.27	human
	Rickettsia	Z/W	vector	bird (153)	346	26,714	2.0	6.4	0.31 ± 0.25	human
	Anaplasma	Z/W	vector	bird (143)	268	8,211	1.7	4.7	0.30 ± 0.33	human
	Borrelia	Z/W	vector	bird (152)	645	22,262	5.2	17.8	0.26 ± 0.16	human
Protozoa	*Plasmodium*	Z/W	vector	bird (188)	705	28,240	23.1	11.7	0.74 ± 0.15	no
	Trichomonas	Z/W	non-vector	bird (113)	278	7,538	31.4	21.1	0.66 ± 0.19	no
	Leucocytozoon	W	vector	bird (178)	530	15,312	22.3	31.3	0.49 ± 0.24	no
	Haemoproteus	Z/W	vector	bird (189)	665	22,617	24.1	22.8	0.37 ± 0.13	no
Virus	influenza A virus	Z/W	non-vector	bird (193)	930	154,132	6.0	8.6	0.62 ± 0.23	domestic duck
	Lyssavirus	Z/W	non-vector	bat (20)	88	2,112	19.8	36.0	0.52 ± 0.39	human
	Sindbis virus	Z/W	vector	bird (63)	253	3,052	9.4	21.4	0.28 ± 0.13	human
	West Nile virus	Z/W	vector	bird (162)	533	12,723	4.7	12.0	0.27 ± 0.27	human
	Usutu virus	Z/W	vector	bird (139)	498	9,317	10.4	17.3	0.18 ± 0.15	human
	tick-borne encephalitis virus	Z	vector	bird (118)	374	10,374	0.3	3.7	0.09 ± 0.11	no map

A pathogen taxon that has zoonotic potential (Z) and wildlife disease potential (W) includes pathogens that can cause infectious diseases in human and wildlife, respectively. The pathogen taxa were ordered by their model accuracy within each pathogen group.

(Table 1). Despite the small sample size ($N = 88$), *Lyssavirus* spp. in bats showed a reasonable accuracy of 0.52 ± 0.39. The model accuracies of protozoa were significantly higher than bacteria (general linear model, $N = 1698$, $R^2 = 0.25$, estimated coefficient ±standard error = 0.24 ± 0.02, $p < 0.001$), while those of viruses were marginally lower than bacteria (−0.03 ± 0.01, $p = 0.051$; Figure 3A). The model accuracies for pathogen taxa without invertebrate vectors were significantly higher than those with invertebrate vectors (0.23 ± 0.01, $p < 0.001$; Figure 3B).

By integrating the predicted species-specific and grid-specific prevalence from the above models and pan-European bird and bat distribution (presence/absence), we mapped the spatial risk of 17 out of the 18 studied pathogen taxa that presented an accuracy R^2 of over 0.18. Specifically, we show spatial patterns of disease risks measured by sum (relative overall risk) and mean (relative community risk) of the predicted prevalence in local host species assemblage in the 50 × 50-km grids (Figures 4, 5, and S13–S27). We mapped relative spillover risk for pathogen taxa that have a potential risk of spillover from birds and bats to humans or poultry, by weighing the relative overall risk with human influence index (Table 1) and with domestic duck density for influenza A viruses. Here, we present the risk maps of *Campylobacter* spp. and influenza A viruses with a highest predictive accuracy among the studied bacterial and viral pathogens, respectively (Figure 3). The overall and human spillover risk of *Campylobacter* spp. is highest in south-western Europe with a warmer climate, higher bird diversity (Figure S2), and higher intensity of human activities (Figure 4). However, the high community risk regions of *Campylobacter* spp. distributed in both southern and northern Europe (*Lyssaviruses* in bats showed a similar pattern), but only in southern Europe during the birds' breeding season. The hotspots for influenza A viruses were less widespread, concentrating in the coastal regions of Europe, especially for the community risk (Figure 5). The risk patterns of influenza A viruses were similar among seasons.

DISCUSSION

We provide a modeling framework for mapping the spatial risk of pathogens carried by birds and bats across a continent and for different seasons (Figure 1). This model is most robust for non-vector-transmitted pathogens (mean R^2 ranges from 0.41 to 0.66). Host traits, host phylogeny, and climatic factors presented reasonable predictive power for the prevalence of most pathogen taxa in host species in a given spatial region. The probability of a given host being pathogen positive is interactively driven by the initial exposure to a pathogen, e.g., exposure load and duration of the exposure, and intrinsic infection susceptibility of the host after the exposure.[26] It is very challenging to control either host infection susceptibility or exposure risk in natural

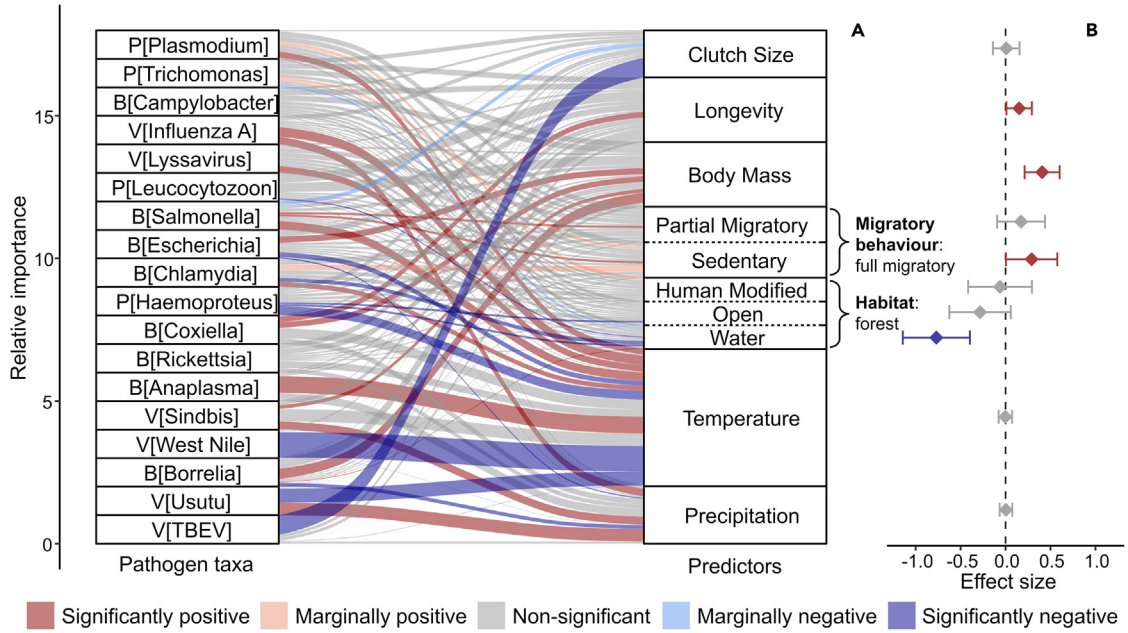


Figure 2. Model estimates

(A) Pathogen-specific model estimates for host species traits (clutch size, longevity, body mass, habitat type, migratory behavior, and climate variables [temperature and precipitation]). The width of the links (colored by the estimated associations) is the relative importance of the predictors (ranges from 0 to 1) in each model of a pathogen taxa. We define the significant and marginally significant associations when the 95% and 90% credible interval of the estimated posterior effect did not pass 0, respectively. The estimates from pathogen-specific models were ordered by high-to-low accuracy estimated by mean R^2 from the cross-validation (Table 1). B, bacterial taxa; P, protozoan taxa; V, viral taxa.

(B) The general effects of the predictors across the studied pathogen taxa, estimated by *post hoc* meta-analyses. The diamond shows the estimated average effect of a predictor on all pathogen taxa. The error bars show upper and lower 95% confidence intervals. "Forest" and "migratory" are reference intercepts for primary habitat types and migratory behaviors, respectively. Forest plots for pathogen-specific results from these models are shown in Figures S7–S12.

systems.[26] Despite this, we were able to identify clear relationships of host species traits and climatic variables with pathogen prevalence that allow predicting and mapping disease risk of mobile hosts at large spatial scales.

We found that bird and bat species with a slower pace of life (i.e., slow-lived species) had a higher pathogen prevalence (Figure 2), which contrasts with patterns observed in rodents[12] and pathogen-specific studies for other vertebrate hosts whereby fast-lived species had a higher probability to carry pathogens.[13–15] Our results suggest that large-bodied and long-lived species among birds and bats had a significantly higher probability to carry pathogen taxa. These slow-lived species may have a longer or higher probability of pathogen exposure at an individual level.[17] Longer-lived species are also more likely to be asymptomatic carriers of pathogens,[27] which may result in a longer duration and/or a broader geographical region of pathogen occurrence. Bird species with a smaller clutch size also showed a higher prevalence of *Salmonella* spp. and TBEVs, despite clutch size having the lowest variable importance and other pathogen taxa not sharing this association. In contrast, among, e.g., rodents and amphibians,[12,28] faster-lived species were widely observed to have a higher probability of being disease reservoirs. Despite the lack of evidence for direct links between species life history traits and immunity, the potential pathway for fast-lived hosts to higher disease risks was hypothesized as weaker immunity defenses for a higher susceptibility for infections.[16] However, based also on two other pathogen-specific studies of birds that showed slow-lived species are more likely to host pathogens,[17,18] we suggest that large-scale patterns of zoonotic and wildlife disease risk in mobile hosts are more linked with host exposure risk than infection susceptibility.

Migratory behavior and primary habitat type of host species also affect the extent to which they are continuously exposed to contaminated biotic and abiotic environments. In agreement with existing theories of migratory escape, migratory recovery, and migratory culling,[19] we found that, compared to sedentary species, migratory species had a lower prevalence, especially for non-vector-transmitted protozoan and orally transmitted bacterial pathogens. Moreover, with respect to habitat type, forest birds showed a higher prevalence of eight pathogen taxa, six of which were tick or mosquito transmitted, as compared to waterbirds. Forests are the primary habitat for ticks and mosquitos,[23] where host species have a higher probability of interacting with or being infested by these potential disease vectors. Except for habitat types, pathogen prevalence may also depend on species' diet (e.g., diet composition, breadth, and foraging strata)[18] because diet can affect inter-species interactions and contact with the abiotic environment. Food resources, diet preferences, and tolerance to human disturbance may also shape species-specific adaptation patterns to human-modified landscapes and affect the intensity of human-wildlife interactions.[29–31] Future studies on diet-disease relationships can further assist in understanding large-scale spillover risks of zoonotic diseases. It is worth stressing that the macroecological

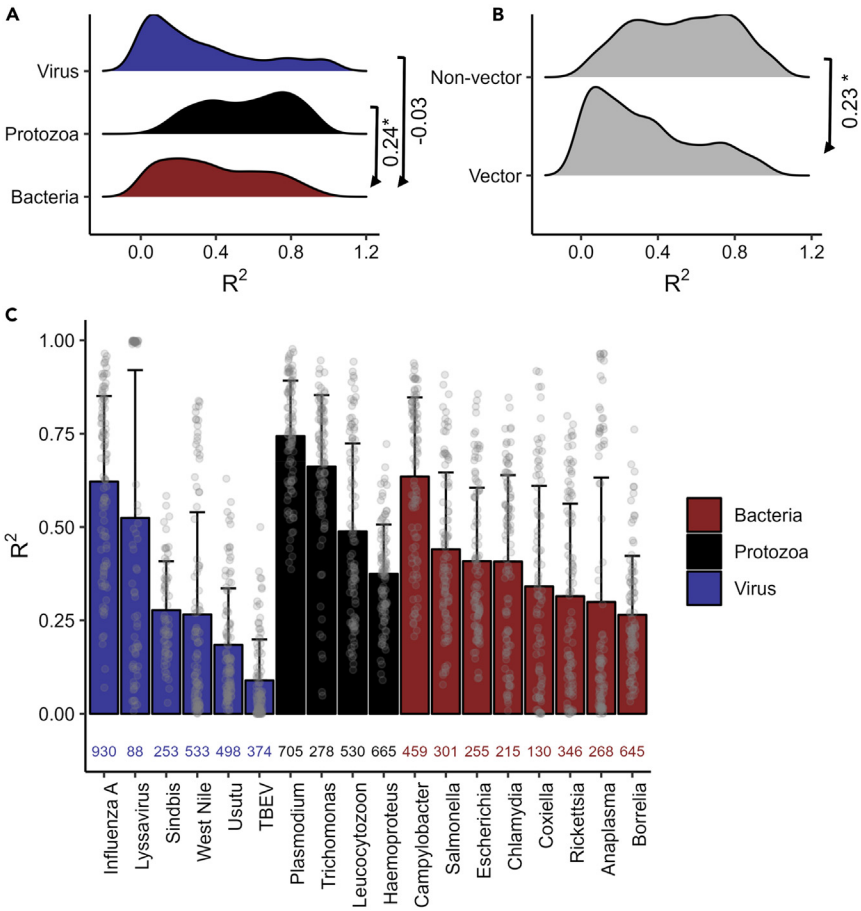


Figure 3. Model accuracy

(A and B) For each model of a pathogen taxon, we did 99 random selections for the training (70% of the observations) and testing (the other 30%). We then measured the model accuracy by the variation in the 30% testing data explained by the model trained by the 70% training data. (A) and (B) show density distribution of the model accuracy (R^2) among pathogen groups and transmission modes (with or without invertebrate vectors), respectively. The marked differences among these groups were estimated by a general linear model (N = 1,698, model R^2 = 0.25). The numbers are the estimated differences between groups with significant differences marked (*$p \leq 0.05$).

(C) Bars show mean and standard deviation of the accuracy according to model validation for 18 different pathogen taxa estimated by the R^2 from the 99 cross-validation results. *Lyssaviruses* were sampled in bats, and all the other pathogen taxa were sampled in birds. The numbers in the bottom of the plot area are the total sample size of each pathogen taxon. The scatters show the accuracy of each model for cross-validation based on random selections.

continental-scale and host species-level study design utilized here cannot adequately cover components with direct links to host infection susceptibility. Therefore, the current modeling framework may not capture the contribution of hosts' intrinsic susceptibility. Further large-scale disease surveys with collection of individual-level host traits (e.g., age structure, sex, species' abundance variation, and body condition such as immune system activities) could improve the current model.

Climatic factors, in particular the ambient temperature, exerted a stronger effect on pathogen prevalence in mobile hosts than species traits (Figure 2). This emphasizes the differing probability of carrying pathogens by a single host species in different climatic conditions. For example, the prevalence of *Lyssavirus* spp. in bats was significantly associated with climatic variables only (Figure 2). Therefore, the commonly used host-filling approach for disease risk mapping[24] could be less accurate according to this modeled spatial heterogeneity of pathogen prevalence in hosts. Moreover, we recommend the use of pathogen-taxa-specific modeling designs because the direction and extent of these climatic effects differ among pathogen taxa (Figure 2).

Regarding the robustness of our modeling framework for a majority of the studied pathogen taxa, we propose to use our combined approach (integrating host-filling and climatic-model approaches)[24] for mapping zoonotic and wildlife disease risk introduced by mobile wild hosts.

However, its feasibility for vector-borne diseases, especially for those caused by viruses (Figure 3), may be improved by including the interactions with invertebrate vectors as a modeling component.[32,33] The mixture of detections for tick-borne pathogens (i.e., TBEVs, *Borrelia* spp., *Anaplasma* spp., *Rickettsia* spp., and *Coxiella* spp.) from birds and ticks feeding on birds in the continental database[10] may also contribute to the higher uncertainties in their models (Figure 3). However, as of now, the number of available datapoints containing explicit invertebrate prevalence information in different hosts is too limited.[10] Other ecological complexities may make up for the unexplained portion of the variance in our models, such as predator-prey interactions[34] and specialization of specific strains or subtypes of pathogens on host species.[35] For instance, different subtypes of influenza A viruses can vary in host specificity; e.g., H13 and H16 were primarily found in gulls.[36] As our continental-scale database was unable to resolve these complex components, we kept our focus on the large-scale generalized patterns and captured a considerable proportion of the variations in pathogen prevalence across host species and geographical regions.

A spillover is determined by multiple successive processes relevant to exposure and infection susceptibility of reservoir hosts and humans or poultry.[37] We included reservoir host distribution, pathogen prevalence, and human or poultry exposure risk at a macroecological scale (in 50 × 50-km units) to our model. One key finding was related to *Campylobacter* spp.; i.e., both the overall and human spillover risk of *Campylobacter* spp. was highest in areas of high human activities in Europe (Figure 4). This aligns with the fact that *Campylobacter* infection, or

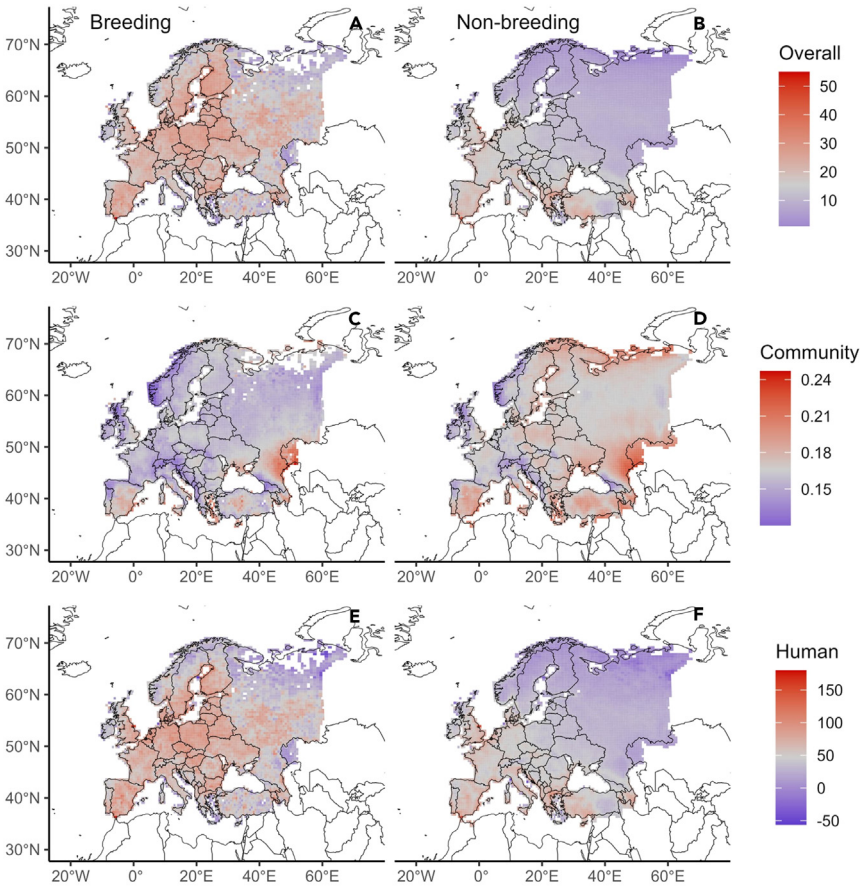


Figure 4. Seasonal risk maps for *Campylobacter* spp. in Europe

(A) and (B) show the relative overall risk, (C) and (D) the relative community risk, and (E) and (F) the relative potential spillover risk (indicated by exposure to human activities), respectively. The risks are shown in (A), (C), and (E) breeding and (B), (D), and (F) non-breeding seasons, respectively.

campylobacteriosis, is the most common bacterial cause of diarrheal illness in human in many European countries.[38] Nonetheless, our risk mapping based on pathogen distributions in mobile hosts may not directly predict zoonotic disease outbreaks. For example, an outbreak of avian influenza includes direct or indirect transmission of lowly pathogenic avian influenza viruses (LPAIVs) from wild-bird reservoirs to farmed poultry such as free-ranging domestic ducks housed under low biosecurity measures.[39] These LPAIVs in the poultry can mutate to HPAIVs and then cause severe diseases and outbreaks in wild birds, poultry, and humans that come in contact or consumed the infected poultry.[21] However, our mapping for avian influenza virus prevalence in wild birds identified the risk hotspots at coastal regions, which agreed with its catastrophic effect on seabirds and even the marine mammals, e.g., in the United Kingdom.[40] As a next step for zoonotic risk forecasts, one could focus on the identified hotspot units and investigate finer-scale spillover drivers—e.g., host species abundance, human and vector behaviors, interactions with domestic animals, human immunological attributes, etc.[37]

The current hotspots for many of the studied pathogen infections were predicted to be regions with a warmer climate (e.g., south-western Europe). Under current and predicted climate change, these hotspots could expand or shift toward cooler regions. Mobile host species are rapidly shifting their ranges to cope with climate and land-use changes,[41] and the hotspots for zoonotic and wildlife diseases accompanying their hosts can move accordingly.[42] Furthermore, various ecological processes linked with climatic warming could amplify this risk of shifting disease hotspots—e.g., loss of migratory behavior in hosts,[9] poleward and/or altitudinal range shifts of disease vectors,[11] intensified cross-species and/or human-wildlife interactions[6], etc. The future predictions for disease risk patterns with assistance of predicted host distributions can facilitate effective monitoring of changes in zoonotic and wildlife disease risk in a world under rapid change. Our modeling framework can serve as a robust and proactive tool for dynamically mapping and projecting disease hotspots regarding host specificity and distributions under climate change. Ultimately, the findings of this work and the approach proposed could facilitate the implementation of the One Health approach[43] that seeks to sustainably balance and optimize the health of humans, wildlife, and ecosystems.

EXPERIMENTAL PROCEDURES

Resource availability

Lead contact

Further information and requests for resources should be directed to and will be fulfilled by the lead contact, Yanjie Xu (yanjie.xu@helsinki.fi).

Materials availability

This study did not generate new unique materials.

Data and code availability

All the data and code used for this study have been deposited at GitHub Zenodo under https://doi.org/10.5281/zenodo.10618771 and are publicly available as of the date of publication. Any additional information required to reanalyze the data reported in this paper is available from the lead contact upon request.

Materials and methods

Model input

Pathogen prevalence. We used a continental-scale database for the prevalence of pathogen taxa in European birds and bats from a recent systematic meta-analysis based on 716 relevant publications.[10,25] The database consisted of 11,939 observations (i.e., a sample site × host species × pathogen taxon combination) of circa 121 pathogenic microbial taxa sampled in over 376 wild bird species and 39 bat species. In the surveyed publications, PCR with subsequent Sanger sequencing and ELISA of pathogen-taxa-specific circulatory antibodies were the most used diagnostic techniques. In few cases, macroscopic clinical symptoms had been used to score positive individuals, e.g., avian pox lesions. This continental-scale database includes prevalence information of bacterial, fungal, and protozoan taxa at the genus level, and some of viral taxa down

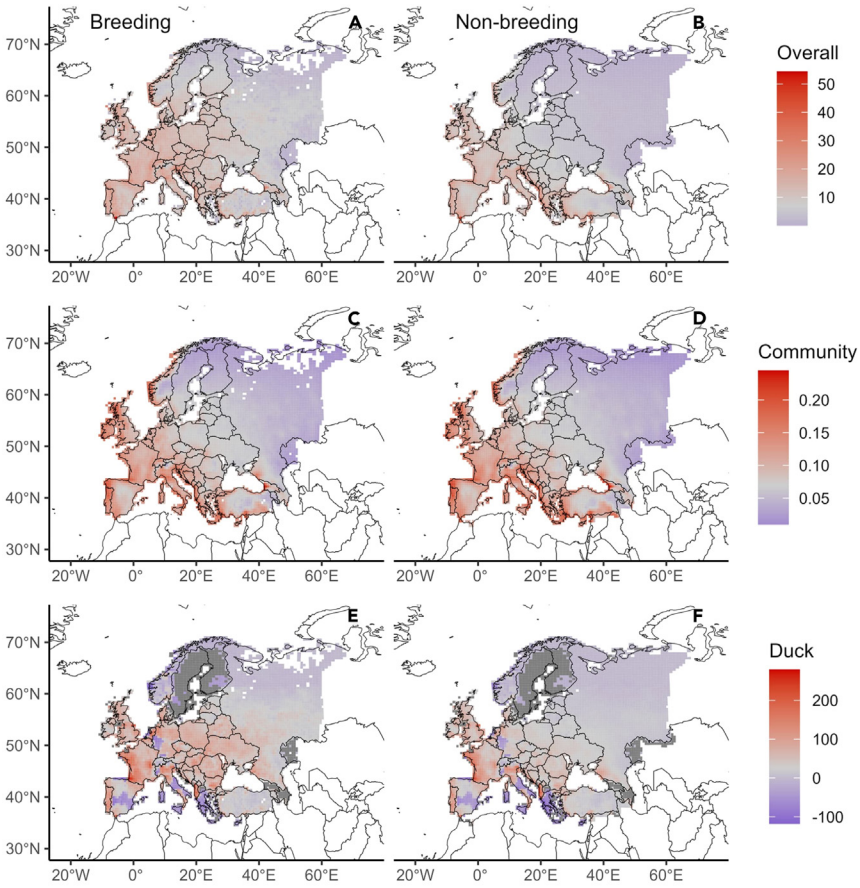


Figure 5. Seasonal risk maps for influenza A viruses in Europe

(A) and (B) show the relative overall risk, (C) and (D) the relative community risk, and (E) and (F) the relative potential spillover risk (indicated by exposure to domestic ducks), respectively. The risks are shown in (A), (C), and (E) breeding and (B), (D), and (F) non-breeding seasons, respectively. The dark gray areas in (E) and (F) are the regions without data on domestic duck densities.

to the species level.[10] We obtained the number of infected birds or bats by a pathogen taxon, number of tested host individuals, taxonomy of the tested host species, latitude, and longitude. The number of infected birds or bats included the zeros where a host species was sampled but not infected by a pathogen taxon. We excluded the observations without specific information of these aspects, which are needed for the subsequent modeling. This database recorded individual-level information when it was available from the source literature, including number of tested male and female (sex) and adult and young (age) individuals for each observation. However, only a minority of the observations had this information (2.6% for sex and 8.5% for age), which was not sufficient for our quantitative analysis. We were only able to model the spatial dimension of the dataset; thereby, neither season nor year was included as explicit modeling components. In the database, there is a lack of available season information for 2,384 observations and cross-season samplings for 3,335 observations. Although the database covered relevant publications in 1945–2020, the sampling was biased across years, with most of the observations (88.5%) resulting from sampling efforts after 2000. We included 17 pathogen taxa in birds that provided over 100 observations across Europe. In addition, we included the most-studied (N = 88) pathogen taxa in bats (i.e., *Lyssavirus* spp). Finally, 7,473 observations were used for the modeling (Data S1, Table 1).

Host traits. For birds, we acquired the body mass, migratory behavior (sedentary, partial migratory, or migratory), and primary habitat type (coastal, desert, forest, grassland, human-modified, marine, riverine, rock, shrubland, wetland, or woodland) of 627 bird species observed and/or sampled in Europe from the AVONET database.[44] We obtained species longevity (maximum lifespan) from Bird et al.[45] We additionally obtained the clutch size of 486 species from the database of life history characteristics of European birds.[46] For the 142 species not included in this European database, we extracted their clutch size from the Amniote Life History Database (139 species). We excluded three species: *Cuculus saturates*, *Columba rupestris*, and *Sinosuthora webbiana*, because their information on clutch size was not found and they do not have self-sustaining wild populations in the study area.[47] For bats, we respectively acquired the body mass, migration distance (local, medium, or long), general habitat type (arid, forest, or mixed anthropogenic—the latter meaning a species is regularly associated with human-modified environments), and foraging environment (open, edge, clutter, above water) of 54 bat species.[48] Additionally, we obtained species longevity (maximum lifespan) of bats from the Amniote Life History Database.[49] We did not include litter size of bat species as the equivalent to clutch size of birds, due to the lack of variation among species in this parameter; i.e., the litter size of bat species is either one or two, with the median being one. We excluded four bat species (i.e., *Miniopterus pallidis*, *Barbastella caspica*, *Eptesicus anatolicus*, *Myotis crypticus*) for which phylogenetic information and/or distribution are poorly known. Therefore, 50 bat species with distributions in Europe were included in the subsequent analyses.

Host phylogeny. We obtained taxonomic levels and 1,000 ultrametric phylogenetic trees (based on the Ericson backbone) of the 627 focal bird species from birdtree.org.[50] We computed a majority-rule consensus tree from the acquired 1,000 phylogenetic trees, which includes the clades that are in the majority (≥50%) of the 1,000 trees with Geneious 11.0.3 (https://www.geneious.com). We acquired 100 phylogenetic trees of the 50 focal bat species from VertLife[51] and built a consensus tree with Geneious using the same settings as in birds.

Climatic data. We extracted the long-term averaged mean annual temperature (°C; hereafter temperature) and annual precipitation (mm; hereafter precipitation) by latitude and longitude of each observation of pathogen prevalence from WorldClim Version 2.1.[52]

Modeling and validation

We modeled the prevalence of each host species-location combination using climatic variables, traits, and phylogeny and then mapped pathogen-taxon-specific risk in birds and bats across Europe. We ran a separate model for each of the 18 focal pathogen taxa (Table 1), resulting in 18 models. In this way, we accounted for both underestimation and overestimation in the spatial modeling of zoonosis risk by integrating two main macroecological methods, namely host filling and climatic model[24] (Figure 1).

Modeling. We fitted generalized linear mixed models (GLMMs) with Markov-chain Monte Carlo techniques[53] to model the prevalence of each pathogen taxon among different mobile host species in different spatial regions. We assumed the default prior distributions, which implemented normal posterior distributions for the fixed effects and inverse-Wishart priors for the random effects.[53] We performed posterior sampling with 133,000 Markov-chain Monte Carlo interactions, and we set 100 as the thinning interval and the first 3,000 iterations as burn-in. The response variable was a matrix of the number of infected individuals and uninfected individuals for each host species at each location (binomial distribution). The trait predictors were clutch size, longevity, body mass, primary habitat type, and migratory behavior of each host species. The climatic predictors were temperature and precipitation. Here, we only considered linear relationships between climatic variables and prevalence, because the covered climatic gradient (continental Europe) was too narrow

for detection of nonlinear thermal- and water-response curves according to a previous test for climate-prevalence relationships with this database.[10] All the continuous variables were scaled, and body mass was first log transformed then scaled. Considering that species life-history traits can be correlated, we checked the multicollinearity of predictors to ensure their variance inflation factors were all under five.[54] To account for the phylogenetic correlation among host species, we included a variance structure of host species phylogenies with the phylogenetic tree of the studied species and the random structures of the species identity. Spatial correlation structure was not accounted for because of the negligible signal of spatial autocorrelation as tested previously for this pathogen prevalence database.[10]

Post hoc *analysis*. We calculated the relative importance of the predictors (i.e., clutch size, maximum longevity, body mass, migratory behavior, habitat, temperature, and precipitation) using the add-one-in procedure. We selected this approach to effectively measure a predictor's maximum possible importance and to ensure comparison among predictors by standardizing the values. Specifically, the relative importance of each predictor was measured by the conditional R^2 of the model with only the focal predictor as the fixed factor.[55] We performed meta-analyses to synthesize the effect of the predictors for prevalence of different pathogens. We used fixed-effects models[56] that do not account for study heterogeneity because the models for different pathogens followed the same modeling framework. We tested the heterogeneity in the coefficients of each tested predictor among different pathogens, which were estimated by their posterior means (effect sizes) with squared standard errors (sampling variances). We reported I^2 (total heterogeneity/total variability) with Q tests and the meta-analysis model results to summarize the differences in the effects and the general effect of a focal predictor among different pathogens, respectively (Data S2). The meta-analyses were conducted with R package metafor.[56]

Cross-validation. We did a cross-validation to quantify the predictive power of the 18 models. We randomly selected 70% of the observations to fit the GLMMs and the rest of the data (30%) to test the accuracy of model predictions. Specifically, we used the fitted GLMMs (with the 70% training data) to predict the prevalence of a focal pathogen among the 30% testing data. We then fitted a general linear model on the observed prevalence with predicted prevalence of these testing data, and calculated the model R^2 (package lme4[57]) to quantify the extent to which the variation in observed prevalence is explained by the predicted prevalence with the trained model. We did 99 random selections for the training and testing data and repeated this validation process at each random selection. We calculated the mean R^2 to measure the predictive power of the GLMMs, which shows the proportion of variations in the observations of the testing data explained by the predicted values from the model trained by the independent training data. In addition, we tested the differences in accuracies among pathogen groups (i.e., bacteria, protozoa, and viruses) and vectors (i.e., with or without invertebrate vectors) with a general linear model in which response variable is the accuracy value by R^2. The two explanatory variables are (1) the pathogen group to which a focal pathogen taxon belongs, and (2) whether invertebrate vectors have a major role in transmission of a focal pathogen taxa (Table 1).

Mapping input

Host species distribution. For birds, we used the European Breeding Bird Atlas 2 (EBBA2) from the European Bird Census Council[47,58] and range maps of the birds of the world from BirdLife International[59] to quantify host seasonal distributions. EBBA2 lists observed bird species within each 50 × 50-km grid in Europe in the breeding seasons of 2013–2017. BirdLife maps compile species-specific seasonal distributions of all birds of the world, from which we extracted species' non-breeding or resident ranges that overlap with the EBBA2 survey range. We only included the range where the species presence was classified as extant or probably extant. Following the above steps, we obtained the distribution of 617 bird species for the breeding season and 313 species for the non-breeding season, both native and non-native European species, for subsequent analyses. For bats, because their seasonal distribution maps are not available at the pan-European scale, we only used non-seasonal range maps (Digital Distribution Maps) from the International Union for Conservation of Nature (IUCN) Red List of Threatened Species assessment data[60] for the 50 focal bat species (including 16 migratory and 34 sedentary species).

Climatic data. For the 50 × 50-km grids spreading across Europe, we extracted the same climatic information by averaging the climatic data within each focal grid.

Exposure layer. We extracted the human influence index in Europe, which was scored for each 1 × 1-km grid by its population density, built-up areas, nightlights, land cover, and proximity to coastlines, roads, railroads, and navigable rivers.[61] We then upscaled the human influence index by averaging its values within each 50 × 50-km grid. Using the same approach, we extracted the density of domestic ducks within each 50 × 50-km grid. The poultry density map was obtained from the Gridded Livestock of the World 2015 (GLW 4) in 1-km^2 resolution.[62]

Risk mapping

We used the fitted GLMMs to make predictions for the prevalence of each pathogen taxon in each mobile host species at each spatial unit. We defined the spatial unit as 50 × 50-km grids of EBBA2 data across all following analyses. For birds, we extracted the observed species in breeding and non-breeding seasons in each 50 × 50-km grid by overlapping the grids with the EBBA2 data and non-breeding range maps, respectively (Figure S2). To reduce the uncertainty of regions with a small sampling effort, we excluded the grids with a species richness of ≤ 5 in either season (i.e., <1% of the total species richness across Europe). For bats, again, we extracted the species observed in each 50 × 50-km grid by overlapping the grids with their range maps (without seasonal information, as this was unavailable for bats; see above).

We then summarized the risk of 17 pathogen taxa in birds and bats at each spatial unit in three different ways. Because the TBEV model had a low predictive accuracy (Table 1), we excluded its risk predictions. For each spatial unit, we estimated (1) the *relative overall risk* by summing up the predicted prevalence of all the species observed in this grid in a focal season (i.e., breeding or non-breeding season for birds, or non-seasonal for bats); (2) the *relative community risk* by averaging the predicted prevalence of species observed in this grid in a focal season (i.e., the overall risk divided by the species richness of each grid); (3) the *potential spillover risk* by weighting the overall risk (as in 1) by the log transformed mean human influence index or domestic duck density of the focal grid for the relevant pathogen taxa according to their spillover mechanisms (Table 1). We used domestic duck density for influenza A viruses because the interaction between free-ranging domestic ducks and wild birds is considered as one of the major spillover pathways.[63,64] We mapped seasonal (for birds only) spatial patterns of these three types of risks for each focal pathogen taxon across Europe. With regard to potential spillover risk, we must stress that mean human influence index is a coarse index of human activity and it does not necessarily correlate with direct potential of humans to be infected by all the different pathogen taxa. Rather, this represents a measure of how humans are potentially exposed to a specific pathogen taxon from birds and bats based on simple co-occurrence in the same grid. We estimated grid-level uncertainties in the risk predictions by the sum and mean of the standard errors of predictions for species-specific prevalence among a focal grid (Figures S29–S32).

In addition, for each European bird and bat species considered here, we estimated a species-specific risk by the median, mean, sum, and range (minimum and maximum) of the predicted prevalence (%) of the species across all the grids where it was observed (breeding and non-breeding season separately). In this way, we produced a database for the species-specific risk of the observed bird and bat species for the 18 studied pathogen taxa across Europe (Data S3; Figure S28 as an example).

All the analyses were conducted with the mentioned packages in R version 4.3.1.[65]

SUPPLEMENTAL INFORMATION

Supplemental information can be found online at https://doi.org/10.1016/j.oneear.2024.04.021.

ACKNOWLEDGMENTS

We thank the members of Helsinki Lab of Ornithology for their insights for analytic design in the beginning of this study. We thank Yingying Wang, Jarrod Hadfield, Shenglai Yin, and Zezhong Wang for their discussions about the

analysis. We thank Petr Voříšek for his insightful comments on this manuscript. We thank the anonymous reviewers for their constructive comments on the earlier versions of this manuscript. This work is financially supported by Research Council of Finland grants 329251 (to T.M.L., A.L., A.P., A.T.P., and Y.X.) and 323527 (to A.L.), European Commission through the Horizon 2020 Marie Skłodowska-Curie Actions individual fellowships 101027534 (to A.S.), and Sakari Alhopuro Foundation grants 20200071 and 2021008 (to A.P.). The present research was carried out within the framework of the activities of the Spanish Government through the "Maria de Maeztu Centre of Excellence" accreditation to IMEDEA (CSIC-UIB) (CEX2021-001198). We acknowledge CSC – IT Center for Science, Finland, for computational resources.

AUTHOR CONTRIBUTIONS

Y.X. and A.L. conceived the study. Y.X., K.M., S.G., K.M.S., A.P., V.N.L., T.M.L., A.T.P., and A.L. collected and validated the data. Y.X. and V.N.L. performed modeling and visualization. Y.X. wrote the original draft with inputs from A.L., T.M.L., A.T.P., and A.S. All the authors edited the manuscript and gave final approval for publication.

DECLARATION OF INTERESTS

The authors declare no competing interests.

Received: August 31, 2023
Revised: February 8, 2024
Accepted: April 29, 2024
Published: May 22, 2024

REFERENCES

1. Mora, C., McKenzie, T., Gaw, I.M., Dean, J.M., von Hammerstein, H., Knudson, T.A., Setter, R.O., Smith, C.Z., Webster, K.M., Patz, J.A., and Franklin, E.C. (2022). Over half of known human pathogenic diseases can be aggravated by climate change. Nat. Clim. Chang. *12*, 869–875. https://doi.org/10.1038/s41558-022-01426-1.
2. Smith, K.F., Sax, D.F., and Lafferty, K.D. (2006). Evidence for the Role of Infectious Disease in Species Extinction and Endangerment. Conserv. Biol. *20*, 1349–1357. https://doi.org/10.1111/j.1523-1739.2006.00524.x.
3. Alders, R., Awuni, J.A., Bagnol, B., Farrell, P., and de Haan, N. (2014). Impact of Avian Influenza on Village Poultry Production Globally. EcoHealth *11*, 63–72. https://doi.org/10.1007/s10393-013-0867-x.
4. Veikkolainen, V., Vesterinen, E.J., Lilley, T.M., and Pulliainen, A.T. (2014). Bats as reservoir hosts of human bacterial pathogen, Bartonella mayotimonensis. Emerg. Infect. Dis. *20*, 960–967. https://doi.org/10.3201/eid2006.130956.
5. Verhagen, J.H., Herfst, S., and Fouchier, R.A.M. (2015). How a virus travels the world. Science *347*, 616–617. https://doi.org/10.1126/science.aaa6724.
6. Carlson, C.J., Albery, G.F., Merow, C., Trisos, C.H., Zipfel, C.M., Eskew, E.A., Olival, K.J., Ross, N., and Bansal, S. (2022). Climate change increases cross-species viral transmission risk. Nature *607*, 555–562. https://doi.org/10.1038/s41586-022-04788-w.
7. Zhou, P., Yang, X.L., Wang, X.G., Hu, B., Zhang, L., Zhang, W., Si, H.R., Zhu, Y., Li, B., Huang, C.L., et al. (2020). A pneumonia outbreak associated with a new coronavirus of probable bat origin. Nature *579*, 270–273. https://doi.org/10.1038/s41586-020-2012-7.
8. Stephens, P.R., Altizer, S., Smith, K.F., Alonso Aguirre, A., Brown, J.H., Budischak, S.A., Byers, J.E., Dallas, T.A., Jonathan Davies, T., Drake, J.M., et al. (2016). The macroecology of infectious diseases: a new perspective on global-scale drivers of pathogen distributions and impacts. Ecol. Lett. *19*, 1159–1171. https://doi.org/10.1111/ele.12644.
9. Altizer, S., Bartel, R., and Han, B.A. (2011). Animal migration and infectious disease risk. Science *331*, 296–302. https://doi.org/10.1126/science.1194694.
10. Xu, Y., Poosakkannu, A., Suominen, K.M., Laine, V.N., Lilley, T.M., Pulliainen, A.T., and Lehikoinen, A. (2023). Continental-scale climatic gradients of pathogenic microbial taxa in birds and bats. Ecography *2023*, e6783. https://doi.org/10.1111/ecog.06783.
11. Iwamura, T., Guzman-Holst, A., and Murray, K.A. (2020). Accelerating invasion potential of disease vector *Aedes aegypti* under climate change. Nat. Commun. *11*, 2130. https://doi.org/10.1038/s41467-020-16010-4.
12. Han, B.A., Schmidt, J.P., Bowden, S.E., and Drake, J.M. (2015). Rodent reservoirs of future zoonotic diseases. Proc. Natl. Acad. Sci. USA *112*, 7039–7044. https://doi.org/10.1073/pnas.1501598112.
13. Ostfeld, R.S., Levi, T., Jolles, A.E., Martin, L.B., Hosseini, P.R., and Keesing, F. (2014). Life History and Demographic Drivers of Reservoir Competence for Three Tick-Borne Zoonotic Pathogens. PLoS One *9*, e107387. https://doi.org/10.1371/journal.pone.0107387.
14. Banerjee, S., Perelson, A.S., and Moses, M. (2017). Modelling the effects of phylogeny and body size on within-host pathogen replication and immune response. J. R. Soc. Interface *14*, 20170479. https://doi.org/10.1098/rsif.2017.0479.
15. Huang, Z.Y.X., de Boer, W.F., van Langevelde, F., Olson, V., Blackburn, T.M., and Prins, H.H.T. (2013). Species' Life-History Traits Explain Interspecific Variation in Reservoir Competence: A Possible Mechanism Underlying the Dilution Effect. PLoS One *8*, e54341. https://doi.org/10.1371/journal.pone.0054341.
16. Albery, G.F., and Becker, D.J. (2021). Fast-lived Hosts and Zoonotic Risk. Trends Parasitol. *37*, 117–129. https://doi.org/10.1016/j.pt.2020.10.012.
17. Yin, S., Li, N., Xu, W., Becker, D.J., de Boer, W.F., Xu, C., Mundkur, T., Fountain-Jones, N.M., Li, C., Han, G.Z., et al. (2023). Functional traits explain waterbirds' host status, subtype richness, and community-level infection risk for avian influenza. Ecol. Lett. *26*, 1780–1791. https://doi.org/10.1111/ele.14294.
18. Becker, D.J., and Han, B.A. (2021). The macroecology and evolution of avian competence for Borrelia burgdorferi. Glob. Ecol. Biogeogr. *30*, 710–724. https://doi.org/10.1111/geb.13256.
19. Peacock, S.J., Krkošek, M., Lewis, M.A., and Molnár, P.K. (2020). A unifying framework for the transient parasite dynamics of migratory hosts. Proc. Natl. Acad. Sci. USA *117*, 10897–10903. https://doi.org/10.1073/pnas.1908777117.
20. Fritzsche McKay, A., and Hoye, B.J. (2016). Are Migratory Animals Superspreaders of Infection? Integr. Comp. Biol. *56*, 260–267. https://doi.org/10.1093/icb/icw054.
21. Lisovski, S., van Dijk, J.G.B., Klinkenberg, D., Nolet, B.A., Fouchier, R.A.M., and Klaassen, M. (2018). The roles of migratory and resident birds in local avian influenza infection dynamics. J. Appl. Ecol. *55*, 2963–2975. https://doi.org/10.1111/1365-2664.13154.
22. Keawcharoen, J., van Riel, D., van Amerongen, G., Bestebroer, T., Beyer, W.E., van Lavieren, R., Osterhaus, A.D.M.E., Fouchier, R.A.M., and Kuiken, T. (2008). Wild Ducks as Long-Distance Vectors of Highly Pathogenic Avian Influenza Virus (H5N1). Emerg. Infect. Dis. *14*, 600–607. https://doi.org/10.3201/eid1404.071016.
23. Tack, W., Madder, M., Baeten, L., Vanhellemont, M., Gruwez, R., and Verheyen, K. (2012). Local habitat and landscape affect Ixodes ricinus tick abundances in forests on poor, sandy soils. For. Ecol. Manag. *265*, 30–36. https://doi.org/10.1016/j.foreco.2011.10.028.
24. Pappalardo, P., Morales-Castilla, I., Park, A.W., Huang, S., Schmidt, J.P., and Stephens, P.R. (2020). Comparing methods for mapping global parasite diversity. Glob. Ecol. Biogeogr. *29*, 182–193. https://doi.org/10.1111/geb.13008.
25. Xu, Y., Poosakkannu, A., Suominen, K., Laine, V., Lilley, T., Pulliainen, A., and Lehikoinen, A. (2023). Pan-European database for pathogenic microbial taxa in birds and bats [Dataset] (Dryad). https://doi.org/10.5061/dryad.stqjq2c84.
26. Sweeny, A.R., and Albery, G.F. (2022). Exposure and susceptibility: The Twin Pillars of infection. Funct. Ecol. *36*, 1713–1726. https://doi.org/10.1111/1365-2435.14065.

27. Smith, O.M., Snyder, W.E., and Owen, J.P. (2020). Are we overestimating risk of enteric pathogen spillover from wild birds to humans? Biol. Rev. *95*, 652–679. https://doi.org/10.1111/brv.12581.

28. Johnson, P.T.J., Rohr, J.R., Hoverman, J.T., Kellermanns, E., Bowerman, J., and Lunde, K.B. (2012). Living fast and dying of infection: host life history drives interspecific variation in infection and disease risk. Ecol. Lett. *15*, 235–242. https://doi.org/10.1111/j.1461-0248.2011.01730.x.

29. Tryjanowski, P., Møller, A.P., Morelli, F., Indykiewicz, P., Zduniak, P., and Myczko, Ł. (2018). Food preferences by birds using bird-feeders in winter: a large-scale experiment. Avian Res. *9*, 16. https://doi.org/10.1186/s40657-018-0111-z.

30. Van Doren, B.M., Conway, G.J., Phillips, R.J., Evans, G.C., Roberts, G.C.M., Liedvogel, M., and Sheldon, B.C. (2021). Human activity shapes the wintering ecology of a migratory bird. Glob. Chang. Biol. *27*, 2715–2727. https://doi.org/10.1111/gcb.15597.

31. Marjakangas, E.-L., Johnston, A., Santangeli, A., and Lehikoinen, A. (2024). Bird species' tolerance to human pressures and associations with population change. Glob. Ecol. Biogeogr. *33*, e13816. https://doi.org/10.1111/geb.13816.

32. Tolle, M.A. (2009). Mosquito-borne Diseases. Curr. Probl. Pediatr. Adolesc. Health Care *39*, 97–140. https://doi.org/10.1016/j.cppeds.2009.01.001.

33. Madison-Antenucci, S., Kramer, L.D., Gebhardt, L.L., and Kauffman, E. (2020). Emerging Tick-Borne Diseases. Clin. Microbiol. Rev. *33*, e00083-18–e00018. https://doi.org/10.1128/CMR.00083-18.

34. Hofmeester, T.R., Jansen, P.A., Wijnen, H.J., Coipan, E.C., Fonville, M., Prins, H.H.T., Sprong, H., and van Wieren, S.E. (2017). Cascading effects of predator activity on tick-borne disease risk. Proc. Biol. Sci. *284*, 20170453. https://doi.org/10.1098/rspb.2017.0453.

35. Svensson-Coelho, M., Ellis, V.A., Loiselle, B.A., Blake, J.G., and Ricklefs, R.E. (2014). Reciprocal Specialization in Multihost Malaria Parasite Communities of Birds: A Temperate-Tropical Comparison. Am. Nat. *184*, 624–635. https://doi.org/10.1086/678126.

36. Lindh, E., Ek-Kommonen, C., Isomursu, M., Alasaari, J., Vaheri, A., Vapalahti, O., and Huovilainen, A. (2017). Genetic Characterization of H13 and H16 Influenza A Viruses in Gulls (Larus spp.) with Clinically Severe Disease and Concurrent Circovirus Infection. J. Wildl. Dis. *53*, 561–571. https://doi.org/10.7589/2016-09-212.

37. Plowright, R.K., Parrish, C.R., McCallum, H., Hudson, P.J., Ko, A.I., Graham, A.L., and Lloyd-Smith, J.O. (2017). Pathways to zoonotic spillover. Nat. Rev. Microbiol. *15*, 502–510. https://doi.org/10.1038/nrmicro.2017.45.

38. European Centre for Disease Prevention and Control (2024). The Surveillance Atlas of Infectious Diseases. http://atlas.ecdc.europa.eu/public/index.aspx.

39. Takekawa, J.Y., Prosser, D.J., Newman, S.H., Muzaffar, S.B., Hill, N.J., Yan, B., Xiao, X., Lei, F., Li, T., Schwarzbach, S.E., and Howell, J.A. (2010). Victims and Vectors: Highly Pathogenic Avian Influenza H5N1 and the Ecology of Wild Birds. Avian Biol. Res. *3*, 51–73. https://doi.org/10.3184/175815510X12737339356701.

40. Nielsen, H.E.F. (2023). Avian influenza has killed millions of seabirds around the world: Antarctica could be next. The Conversation. (Australia Edition).

41. Lehikoinen, A., and Virkkala, R. (2016). North by north-west: climate change and directions of density shifts in birds. Glob. Chang. Biol. *22*, 1121–1129. https://doi.org/10.1111/gcb.13150.

42. Beyer, R.M., Manica, A., and Mora, C. (2021). Shifts in global bat diversity suggest a possible role of climate change in the emergence of SARS-CoV-1 and SARS-CoV-2. Sci. Total Environ. *767*, 145413. https://doi.org/10.1016/j.scitotenv.2021.145413.

43. Global One Health Community (2023). What is one health?. https://globalohc.org/what-is-one-health.

44. Tobias, J.A., Sheard, C., Pigot, A.L., Devenish, A.J.M., Yang, J., Sayol, F., Neate-Clegg, M.H.C., Alioravainen, N., Weeks, T.L., Barber, R.A., et al. (2022). AVONET: morphological, ecological and geographical data for all birds. Ecol. Lett. *25*, 581–597. https://doi.org/10.1111/ele.13898.

45. Bird, J.P., Martin, R., Akçakaya, H.R., Gilroy, J., Burfield, I.J., Garnett, S.T., Symes, A., Taylor, J., Şekercioğlu, Ç.H., and Butchart, S.H.M. (2020). Generation lengths of the world's birds and their implications for extinction risk. Conserv. Biol. *34*, 1252–1261. https://doi.org/10.1111/cobi.13486.

46. Storchová, L., and Hořák, D. (2018). Life-history characteristics of European birds. Glob. Ecol. Biogeogr. *27*, 400–406. https://doi.org/10.1111/geb.12709.

47. Keller, V., Herrando, S., Voříšek, P., Franch, M., Kipson, M., Milanesi, P., Martí, D., Anton, M., Klvanová, A., and Kalyakin, M.V. (2020). European breeding bird atlas 2: distribution, abundance and change (Barcelona: European Bird Census Council & Lynx Edicions).

48. Meramo, K., Kotila, M., Gaultier, S., Lundberg, P., Suutari, M., Ovaskainen, O., and Lilley, T. (2023). Latitudinal gradient and species traits determine bat distributions across Europe. Preprint at Authorea. https://doi.org/10.22541/au.167845009.98519046/v1.

49. Myhrvold, N.P., Baldridge, E., Chan, B., Sivam, D., Freeman, D.L., and Ernest, S.K.M. (2015). An amniote life-history database to perform comparative analyses with birds, mammals, and reptiles. Ecology *96*, 3109. https://doi.org/10.1890/15-0846R.1.

50. Jetz, W., Thomas, G.H., Joy, J.B., Hartmann, K., and Mooers, A.O. (2012). The global diversity of birds in space and time. Nature *491*, 444–448. https://doi.org/10.1038/nature11631.

51. Upham, N.S., Esselstyn, J.A., and Jetz, W. (2019). Inferring the mammal tree: Species-level sets of phylogenies for questions in ecology, evolution, and conservation. PLoS Biol. *17*, e3000494. https://doi.org/10.1371/journal.pbio.3000494.

52. Fick, S.E., and Hijmans, R.J. (2017). WorldClim 2: new 1-km spatial resolution climate surfaces for global land areas. Int. J. Climatol. *37*, 4302–4315. https://doi.org/10.1002/joc.5086.

53. Hadfield, J.D. (2010). MCMC Methods for Multi-Response Generalized Linear Mixed Models: The MCMCglmm R Package. J. Stat. Softw. *33*, 1–22. https://doi.org/10.18637/jss.v033.i02.

54. Zuur, A.F., Ieno, E.N., and Elphick, C.S. (2010). A protocol for data exploration to avoid common statistical problems. Methods Ecol. Evol. *1*, 3–14. https://doi.org/10.1111/j.2041-210X.2009.00001.x.

55. Smith, A.M., Clark, D.E., Lamare, M.D., Winter, D.J., and Byrne, M. (2016). Risk and resilience: variations in magnesium in echinoid skeletal calcite. Mar. Ecol. Prog. Ser. *561*, 1–16.

56. Viechtbauer, W. (2010). Conducting Meta-Analyses in R with the metafor Package. J. Stat. Softw. *36*, 1–48. https://doi.org/10.18637/jss.v036.i03.

57. Bates, D., Mächler, M., Bolker, B., and Walker, S. (2015). Fitting linear mixed-effects models using lme4. J. Stat. Softw. *67*, 1–48. https://doi.org/10.18637/jss.v067.i01.

58. EBCC (2022). European Breeding Bird Atlas 2 Website (European Bird Census Council). http://ebba2.info.

59. Birdlife International and NatureServe (2015). Bird Species Distribution Maps of the World Version 5.0. BirdLife International, Cambridge, UK and NatureServe, Arlington, USA. Available online from. http://www.birdlife.org.

60. IUCN. Spatial Data Download: Mammals. 2018. https://www.iucnredlist.org/resources/spatial-data-download. Accessed on 22 February 2022.

61. Wildlife Conservation Society - WCS, and Center for International Earth Science Information Network - CIESIN - Columbia University (2005). Last of the Wild Project, Version 2, 2005 (LWP-2): Global Human Influence Index (HII) Dataset (Geographic) (NASA Socioeconomic Data and Applications Center (SEDAC)).

62. Gilbert, M., Nicolas, G., Cinardi, G., Van Boeckel, T.P., Vanwambeke, S.O., Wint, G.R.W., and Robinson, T.P. (2018). Global distribution

data for cattle, buffaloes, horses, sheep, goats, pigs, chickens and ducks in 2010. Sci. Data *5*, 180227. https://doi.org/10.1038/sdata.2018.227.

63. Yin, S., Xu, Y., Batbayar, N., Takekawa, J.Y., Si, Y., Prosser, D.J., Newman, S.H., Prins, H.H.T., and de Boer, W.F. (2021). Do contrasting patterns of migration movements and disease outbreaks between congeneric waterfowl species reflect differing immunity? Geospat. Health *16*, 909. https://doi.org/10.4081/gh.2021.909.

64. Cappelle, J., Zhao, D., Gilbert, M., Nelson, M.I., Newman, S.H., Takekawa, J.Y., Gaidet, N., Prosser, D.J., Liu, Y., Li, P., et al. (2014). Risks of avian influenza transmission in areas of intensive free-ranging duck production with wild waterfowl. EcoHealth *11*, 109–119. https://doi.org/10.1007/s10393-014-0914-2.

65. R Core Team (2023). R: A Language and Environment for Statistical Computing (R Foundation for Statistical Computing). https://www.R-project.org/.