Continental-scale climatic gradients of pathogenic microbial taxa in birds and bats

Yanjie Xu (✉ yanjie.xu@helsinki.fi)

University of Helsinki <https://orcid.org/0000-0003-4420-6353>

Anbu Poosakkannu

Institute of Biomedicine, University of Turku

Kati Suominen

The Finnish Museum of Natural History, University of Helsinki <https://orcid.org/0000-0002-1177-5755>

Veronika Laine

The Finnish Museum of Natural History, University of Helsinki

Thomas Lilley

The Finnish Museum of Natural History, University of Helsinki

Arto Pulliainen

Institute of Biomedicine, University of Turku

Aleksi Lehikoinen

University of Helsinki <https://orcid.org/0000-0002-1989-277X>

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Abstract

Climate change may alter the threat of zoonotic and wildlife diseases, by changing environmental suitability for hosts, ectoparasites, and pathogens. However, the connections between climatic variables and diseases remains equivocal. We compiled a systematic database for the prevalence of 121 pathogenic microbial taxa in birds and bats, including 11,801 observations from over 450,000 individuals across Europe and surrounding regions. Using a space-for-time-substitution approach, we modelled the potential connection of climatic variables on the prevalence of different pathogenic taxa. The prevalence of influenza A virus and several bacterial taxa in birds (376 species) and bats (39 species) positively correlated with temperature. Rainfall showed a positive correlation with the prevalence of viral taxa but the directions of significant correlations varied between bacterial taxa. The results suggest that changes in temperature and precipitation may potentially alter the threat of bird- and bat-associated pathogenic microbial taxa to the wild-life, domesticated animals and humans.

Full Text

Over 60% of human infectious diseases are caused by zoonotic viruses, which can spill over from wild animals, including important pandemic origins, e.g., coronavirus from bats and avian influenza from birds ¹⁻⁴. The changing climate may alter the prevalence of pathogenic microbes, parasites, and their vectors in wildlife, directly by altering environmental conditions necessary for their survival and virulence, or indirectly by shifting host community composition, abundance, distribution, phenology, or physiological traits ⁵⁻⁹. Yet the effect of climate, including both temperature and rainfall, on zoonotic and wildlife diseases remains equivocal ⁵ and contradictory ¹⁰. A typical example is the contrasting effects of temperature found in malaria-related parasites in birds ¹⁰.

Birds and bats are highly mobile vertebrates, often contributing to the large-scale transportation of pathogens ^{2,3,11}. A large proportion of these host taxa are migratory, seasonally connecting diverse and distant habitats and host communities, thus, facilitating long-distance transmission and expansive persistence of pathogens responsible for infectious diseases ¹². Furthermore, there is a growing body of evidence showing the response of these host taxa to climatic warming by rapid poleward shift or range expansion ^{12,13}, which may accelerate the invasion and adaptation of pathogens carried by them ¹⁴. Understanding how the prevalence of pathogens in these mobile hosts varies across the climatic variables would provide valuable insights on how future climate change may affect pathogen distribution at a population scale. This kind of data could also pinpoint geographic regions where future disease outbreaks may emerge.

However, long-term, systematic, multi-pathogen studies of climate-caused alterations of pathogen prevalence in mobile wild hosts are rare and limited to malaria-related parasites in birds ^{10,15}. The challenges for a more holistic understanding of the effects of climatic variables, notably temperature and rainfall, on zoonotic and wildlife diseases could be tackled by spatially extensive pathogen surveys

across climatic gradients^{6,10}. Here we present a large-scale, multi-host, multi-pathogenic-taxon analysis across climatic gradients (Methods) to test: (i) the extent to which temperature and precipitation associate with detection of pathogenic microbial taxa in birds and bats, where also the phylogeny of the hosts was controlled; and (ii) the generalizability of these climatic effects among different microbial and host taxa (birds and bats).

Pan-European database for pathogenic microbial taxa

We compiled a spatially extensive multidimensional database of the prevalence of pathogenic microbial taxa for humans and wildlife (including domestic animals) in birds and bats, by systematically reviewing publications (Text S1) for 121 pathogenic taxa (Fig. 1 and Table S1), mostly at the genus level, covering ~450,000 host individuals of 376 bird species and 39 bat species (Data S1). We acknowledge that for microbes, genus level groups can include variable responses at lower taxonomic level, but currently such species/strain level data is not available at the scale we utilize in our modeling. Each observation in the database included the relevant information (Methods and Data S3) on the prevalence of a pathogenic taxon in a host species sampled at a location at a given time point. The prevalence was described by a combination of the number of infected host individuals (0–3,955) and tested host individuals (1–14,080). Our database extends across Europe and surrounding regions, covering multiple climatic zones, namely, Nordic climate, Eastern-continental climate, Oceanic climate, and Mediterranean climate (annual mean temperature -9.0–27.7 °C, annual precipitation 15–2566 mm, and Fig. 1a–b).

We collected prevalence and location data for the reviewed pathogenic microbial taxa from our compiled database spanning 720 publications (Fig. 2 and Text S1). We accessed WorldClim database¹⁷ to extract long-term averages of temperature (Fig. 3a) and rainfall (Fig. 3f) from sampling locations described in above publications. We modelled the prevalence of pathogenic taxa in bird and bat hosts against climatic variables using generalized linear mixed models (GLMMs, full list of the variables in Table 1). These models (here after multi-taxa models) tested the connection between the climatic variables and prevalence of pathogenic groups (bacteria, eukaryotes, and viruses) in host groups (bird and bat, see Fig. 4a–d). To understand variation within the previously grouped bacteria, viruses and eukaryotes, we further subset our data to single-taxon level in cases where literature provided over 100 observations for a given pathogenic microbial taxon. These single taxa were then individually modelled similarly to above but without accounting for host group identity (hereafter single-taxon models, see Fig. 4f–g, Table S3 and S4).

Temperature and pathogen prevalence in birds and bats

According to our multi-taxa models, the prevalence of pathogenic bacteria in the birds and bats of Europe and surrounding regions showed significant positive associations with rising temperature. This positive association was not different between host taxa (i.e., birds or bats) (Fig. 4a and details in Table 1). Based on single-taxon analyses, seven pathogenic taxa (out of 17 most-studied taxa tested, ≥ 100 observations) were significantly correlated with temperature. Six of these, including influenza A,

Plasmodium and four bacteria were positively associated with increasing temperature (Fig. 4f and Table S2, and Data S1 including prevalence in different host species). Only *Haemoproteus* prevalence in birds decreased with increasing temperature (Fig. 4f), which is consistent with previous observations that detected such contrasting responses of *Haemoproteus* and *Plasmodium* in Western Palearctic birds to winter temperatures¹⁰. The positive correlation of *Plasmodium* with temperature also agrees with the previously detected strong positive association of temperature on avian malaria infection¹⁵.

There are multiple mechanisms that can potentially explain the observed positive role of temperature in pathogen prevalence in wildlife, which include both direct (climate-pathogen) and indirect (climate-host-vector-pathogen) processes. Physiologically, changing temperature can facilitate survival, invasion, or reproduction of some pathogens and disease vectors¹⁸⁻²¹. At a host population level, a warmer climate can increase pathogen winter survival, extend the breeding period of hosts, and expand the ranges and densities of host populations northwards, increasing vector abundance and facilitating pathogen survival⁶. Furthermore, the observed increasing parasitism and northward invasion of vectors with warmer climate in Europe^{18,22,23} supports the detected positive responses of tick- and mosquito-borne pathogenic taxa (i.e., *Rickettsia*, *Coxiella*, and *Plasmodium*) with temperature (Fig. 4f).

Precipitation and pathogen prevalence in birds and bats

Based on multi-taxa models, the prevalence of pathogenic bacteria in birds and bats of Europe and surrounding regions decreased significantly with increasing precipitation, while the viral group showed an opposite pattern (Fig. 4b and Table 1). Single-taxon models revealed that the five tested viral species, Influenza A, Sindbis, and Usutu viruses were positively associated with precipitation (Fig. 4g and Table S2). Higher precipitation can increase the population size of mosquitoes, which are vectors for both Sindbis and Usutu viruses^{24,25}. Also, increased availability of wetlands in regions with a higher precipitation favors waterbirds, which are important hosts for influenza A virus²⁶. The tested bacterial taxa showed opposing responses to precipitation. *Anaplasma* and *Salmonella* were positively associated while *Borrelia*, *Escherichia* and *Campylobacter* were negatively associated with precipitation (Fig. 4g and Table S2). Increased availability of wetlands in regions with a higher precipitation favors waterbirds, which are important hosts for *Salmonella*²⁷. However, it is difficult to understand the opposing connections, i.e. *Anaplasma* and *Borrelia* are both ectoparasite-transmitted whereas *Salmonella*, *Escherichia* and *Campylobacter* are orally-transmitted bacteria and they share hosts. It is possible that our analysis hints towards hitherto unknown features on the pathogen ecology of these important bacterial taxa (Fig. 4 and Table S2). Our findings highlight the importance of systematic taxa-specific follow-up studies.

Table 1
Estimates from multi-taxa models for prevalence of pathogenic microbial taxa in birds and bats. By a model selection, we list the best models and the average of the top models with

$\Delta AIC \leq 2$. We show degree of freedom (df) and the performance of the models by AIC values, conditional and marginal pseudo-R-squared (R_c and R_m), and regression coefficients of the predictors. We mark the estimated coefficients that are significant at a 0.05 level with “*”.

Model	Coefficient	Standard Error	z-value	p-value
Best model (df = 8802, AIC = 26785.7, $R_c = 0.451$, $R_m = 0.115$)				
(Intercept)	-1.177	0.413	-2.854	0.004*
Temperature	0.196	0.044	4.385	<0.001*
Precipitation	-0.135	0.038	-3.538	<0.001*
Host taxa (Baseline: Bat)				
Bird	-1.968	0.380	-5.172	<0.001*
Pathogenic microbial taxa (Baseline: Bacteria)				
Eukaryota	0.540	0.386	1.398	0.162
Virus	-0.336	0.307	-1.096	0.273
Season (Baseline: Migration)				
Summer	0.680	0.076	8.912	<0.001*
Winter	-0.082	0.122	-0.677	0.498
Multi-season	0.386	0.069	5.621	<0.001*
Unknown	0.606	0.086	7.069	<0.001*
Pathogenic taxa×Temperature (Baseline: Bacteria×Temperature)				
Eukaryota×Temperature	-0.210	0.056	-3.763	<0.001*
Virus×Temperature	-0.209	0.064	-3.253	0.001*
Pathogenic taxa×Precipitation (Baseline: Bacteria×Precipitation)				
Eukaryota×Precipitation	0.161	0.052	3.123	0.002*
Virus×Precipitation	0.445	0.055	8.030	<0.001*
Average model ($\Delta AIC \leq 2$, Number of models = 3)				
(Intercept)	-1.176	0.415	-2.833	0.005*
Temperature	0.179	0.087	2.042	0.041*
Precipitation	-0.128	0.065	-1.987	0.047*
Host taxa (Baseline: Bat)				
Bird	-1.970	0.383	-5.139	<0.001*
Pathogenic microbial taxa (Baseline: Bacteria)				
Eukaryota	0.540	0.386	1.398	0.162
Virus	-0.333	0.307	-1.085	0.278
Season (Baseline: Migration)				
Summer	0.680	0.076	8.907	<0.001*
Winter	-0.082	0.122	-0.678	0.498
Multi-season	0.386	0.069	5.621	<0.001*
Unknown	0.680	0.076	7.072	<0.001*
Pathogenic taxa×Temperature (Baseline: Bacteria×Temperature)				
Eukaryota×Temperature	-0.210	0.056	-3.756	<0.001*
Virus×Temperature	-0.208	0.064	-3.238	0.001*
Pathogenic taxa×Precipitation (Baseline: Bacteria×Precipitation)				
Eukaryota×Precipitation	0.161	0.052	3.100	0.002*
Virus×Precipitation	0.445	0.056	8.002	<0.001*
Host taxa×Temperature (Baseline: Bat×Temperature)				
Bird×Temperature	0.018	0.079	0.225	0.822
Host taxa×Precipitation (Baseline: Bat×Precipitation)				
Bird×Precipitation	-0.007	0.057	-0.122	0.903

Potential challenges from zoonotic and wildlife diseases under climate change

Global temperature has been increasing and this warming is predicted to continue²⁸. The positive correlation between temperature and the prevalence of several pathogenic taxa in birds and bats suggested by our results indicates increased prevalence for these diseases under projected climatic warming within Europe and surrounding regions. However, as with any correlational study, other aspects of geography that we did not take into account might confound our interpretation of the role that temperature plays. Other climatic variables are also changing globally²⁹. Future predictions show that

less rainfall is expected in tropical areas and more in temperate and high-latitude regions (Fig. 3), thus annual rainfall tends to change towards the upper and lower extremes²⁹. Although pathogenic viral taxa show a weaker connection to temperature compared to bacterial taxa, they consistently show a positive association to rainfall. This again suggests the potential of these viruses to expand towards higher latitudes with the shift in rainfall patterns (Fig. 3). The distributions of most tested pathogenic taxa (14/17) did not show the predicted hump-shaped response to temperature and precipitation (Fig.S7 and S8). This could be an indication that confounding variables are driving the association with temperature, or that the covered climatic gradient was too narrow to detect thermal-response curves. Therefore, linear predictions between pathogens and climatic variables should not be extrapolated to warmer or colder regions than our study area. The next step is to develop more extensive databases and analyses covering the entire climatic niche space for hosts and pathogens, in order to indicate the extent to which climate change might reduce pathogens in regions to the south which may become too warm for them in the future.

We show macroecological evidence that the risk of some wildlife and zoonotic diseases can change spatially due to shifting temperature and precipitation levels in Europe and surrounding regions by highlighting the trends from a large microbial prevalence value database. However, the responses of different pathogenic taxa show variability with regards to climatic gradients, with the prevalence of bacterial pathogenic taxa in birds and bats showing a stronger increase at higher temperatures compared to viruses and eukaryotes (Fig. 4a). The effects of climate on zoonotic and wildlife diseases involve complex cascades among climate, resources, host assemblages, disease vectors, and pathogens^{5,6}. Our open access database can further support identifying mobile host reservoirs for zoonoses, thus dynamically mapping and projecting disease hotspots regarding their distributions under climate change. The facilitation of effective monitoring of changes in wildlife and zoonotic diseases call for consistent and more fine-scale spatiotemporal-reporting systems in wildlife disease surveillance, a better understanding of seasonal patterns and environmental requirements of host species, and mechanistic models.

Changes in disease risk under climate change can further threaten the resilience and survival of wild host populations³⁰, and local extinction⁸ and/or population declines⁹ of some host species can promote pathogen transmission^{7,8}. Although the direction of host diversity effect on disease risk remains equivocal³¹, such alterations in host community composition could potentially reduce the community competence to pathogens by loss of dilution effect⁷ (Table S5). In contrast, in many European areas, especially in originally species poor areas (e.g. arctic, alpine and boreal regions), bird diversity has increased from 1980s to 2010s³². This was widely explained by the climate-driven poleward range shifts of these mobile wild hosts in Europe³³, which can potentially expand the distribution of disease risk³⁴ and cross-species transmissions¹⁴. Furthermore, all the pathogenic taxa in our study that were positively associated with temperature (i.e., *Rickettsia*, *Coxiella*, *Plasmodium*, *Salmonella*, *Chlamydia*, and Influenza A virus) can potentially cause severe human infectious diseases (Table S1). Among mammalian host taxa, bats were found to play a major role in novel viral sharing, which are critical for future infectious

disease emergence in humans under climate change¹⁴. Therefore, climate change can increase the risk of spillover in new regions by facilitating expansion of pathogens and vectors to higher latitudes and altitudes^{5,35}, especially via mobile hosts such as birds and bats and their climate-driven range expansions.

Methods

Literature search

We extracted prevalence data from peer-reviewed literature for pathogenic microbial taxa in wild birds and bats in Europe and surrounding countries. We first listed the pathogenic taxa (Table S1) found in birds and bats according to our knowledge and relevant reviews³⁶⁻³⁹, then went through the list taxon by taxon when searching and selecting the relevant publications. We identified the relevant publications that sampled birds and bats and published in 1945–2020, through a systematic literature search in the ISI Web of Science. The search criterion was: [name of the focal pathogenic taxa]* AND bird* (or bat*) NOT poultry* NOT chicken* NOT broiler* NOT companion* NOT United States* NOT China* NOT Japan* NOT Brazil* NOT New Zealand* NOT Australia*. In this criterion, we added exclusions for domestic or captive animals and for the countries outside our study area that have frequently published relevant works.

We screened the title and abstract of all the results from searching, and only accessed full texts of the publications that: sampled birds or bats; reported number of host individuals tested for pathogenic microbial taxa; reported number of infected individuals or prevalence of a focal pathogenic taxon; tested wild populations (including feral birds) instead of poultry, pets, experimental, or cage animals; tested naturally occurred pathogenic taxa instead of those introduced experimentally; sampled in Europe and surrounding countries.

By this way, we identified 720 peer-reviewed papers (Text S1), of which 644 papers tested pathogenic taxa in birds and 76 tested those in bats with conventional methods including polymerase chain reaction (PCR) and enzyme linked immunosorbent assay (ELISA). Most of them are in English; however, we also reviewed relevant papers in other languages (e.g., Russian and French) with assistance of Google Translate.

Information extraction

From each selected publication, we extracted available information about host, pathogenic taxon, sampling location and time, and corresponding literature source. Each observation in our database included one pathogenic taxon and one host species (common name and scientific name recorded), and we recorded the subtype (i.e., species, strain, or other specific information) of the focal pathogenic taxon if available. We recorded the number of tested hosts and number of infected hosts for the corresponding pathogenic taxon. We also recorded sampling approaches for each observation, including cloacal swab,

oral swab, fecal sample, feather sample, brain sample, necropsy, blood sample, serum sample, etc. ELISA cultivation, PCR and subsequent DNA sequencing were the most used pathogen identification techniques. In a few cases, for example, avian pox lesions were visually identified. The pathogens were identified as single microbes or as part of a community.

We recorded the most specific information about date and/or period when the birds and bats were sampled in the field, and classified the observations into five seasons: summer, winter, migration (spring and/or autumn), multi-season, and unknown (period of sampling not reported). If the reviewed publication mentioned that their samples from the study species were collected in breeding (nesting/fledgling/post-breeding), wintering, or migration (passage/stopover) seasons, we defined their sampling seasons as summer, winter, and migration, respectively. If the above information is not available, we count the number of months covered by the survey according to the reported sampling dates/months/period. We defined the sample as 'multi-season' when it covers ≥ 6 months. Otherwise, we calculated the median month of the sampling period, and defined December, January, and February as 'winter'; May to July as 'summer'; March to April and August to November as 'migration'. If none of this information was available, we defined the season of corresponding records as 'unknown'.

To extract the climatic condition of the locations where the samples were collected, we identified the latitude and longitude of each observation. We recorded these coordinates directly from the corresponding publications when available; otherwise, we identified the coordinates by Google searching the location, city, province, or country name (i.e., the most specific information of sample sites reported in the source literature) followed by 'coordinates' or positioning the specific locations by Google Maps. If a reviewed publication reported merged results from several regions, we calculated and recorded the geometric center of these different areas. According to the identified finest-resolution information about the sample sites from the reviewed publications, we classified the accuracy of these coordinates into six catalogues (from the most to the least accurate): coordinate (45.5% of all the observations), location (12.9%), city (7.1%), region (12.8%), country (19.0%), or continent (2.6%). We defined the accuracy of observations that report province, state, autonomous community, or region as 'region', and the accuracy of those that report city, town, commune, or municipality as 'city'.

By this way, we covered 121 pathogenic microbial taxa (Table S1 and Data S2) sampled between 1962 and 2020 (Fig. 2 and 88.5% in 2000s), and this resulted in 11124 and 677 observations of their prevalence in >376 bird species and >39 bat species, respectively (Data S1).

Database quality control

We checked the taxonomic information of the covered pathogenic taxa and excluded those reported at a higher taxonomic level than the genus. We compiled bacterial taxa, fungal taxa and protozoan parasites at a genus level, while allowing species-level identifications for viral taxa. We also checked the scientific

names and common names of host species throughout the database to exclude the observations without specific information of host species. By this way, we cross-checked the non-native species for Europe and surrounding countries, and excluded them if they were not explicitly mentioned as wild animals in the original publications. We excluded observations with either an accuracy of 'continent' or combined results from non-adjacent countries. The records from Russian Far East and Reunion islands were also excluded by checking the geographical coordinates of each recorded observation. We also excluded observations where the number of either tested hosts or infected hosts were not reported. Lastly, we went through the list of pathogenic microbial taxa and checked their scientific names in accordance to NCBI taxonomy database¹⁶. We further excluded the taxa that are either with single observation or sampled in one location from the following analyses. As a result, a total of 8822 observations of 64 pathogenic taxa in birds and 23 in bats (including 12 in both birds and bats) were included in the subsequent analyses (Fig. 1), of which 88.9% potentially cause zoonotic diseases and 91.7% for wildlife diseases (81.3% for both, Table S1 and Data S2).

Climatic data

We obtained the global raster maps of long-term averaged bioclimatic variables for near-current conditions (1970–2000) at a 10-minute resolution from WorldClim Version 2.1¹⁷. According to the geographical coordinates, we extracted the annual mean temperature (bio1; °C) and annual precipitation (bio12; mm) for each observation in the compiled database.

Additionally, from the same climate database, we obtained the same variables at a 10-minute resolution for future predictions: 2021–2040, 2041–2060, 2061–2080, and 2081–2100 under the two extreme Shared Socio-economic Pathways (i.e., SSP126 and SSP585). We summarized and compared the distributions of temperature and precipitation in current and future climatic conditions within the latitudinal range from 20°N to 80°N and the longitudinal range from 20°W to 60°E (Fig. 3).

Statistical analysis

We tested the effects of temperature and precipitation on prevalence of pathogenic microbial taxa in birds and bats with two sets of generalized linear mixed models (GLMMs) with a quadratic parameterized negative binomial distribution family (Fig. 4, Table 1, and Table S2 and S2). The response variable was the number of infected hosts (NP), and we incorporated the number of tested hosts (NT) as an offset variable for each model. The explanatory variables were annual mean temperature (T ; °C) and annual precipitation (P ; mm) of the corresponding sampling sites. Assuming there may be seasonal differences of pathogenic taxa prevalence, we included the classified season (SS) as an additional explanatory variable. We standardized all the continuous variables by subtracting their mean and dividing by their standard deviation. Regarding different sampling efforts and prevalence patterns among different host species, we included the host species ID ($SPEC$) as random effects of the GLMMs.

Firstly, we tested the general effect of temperature and precipitation on the prevalence of pathogenic microbial taxa in birds and bats by fitting multi-taxa models (GLMM1) with merged observations from different pathogenic taxa (8822 observations of 75 pathogenic taxa in 409 host species). We classified the analyzed pathogenic taxa into three taxa groups, i.e., bacteria, eukaryota (including fungus and protozoa) and virus, and tested the interactions between pathogenic taxa group (*PATH_GR*) and the climatic variables (*T* and *P*) by GLMM1. Similarly, we tested the different responses among host taxa to climatic gradients by including the host taxa group (*HOST_GR*, i.e., birds or bats) and its interaction with the climatic variables in GLMM1. Regarding potential differences among different pathogenic taxa, here we included pathogenic taxon ID (*PATH*) as an additional random factor.

GLMM1:

$$NP \sim T + P + SS + PATH_GR + HOST_GR + T \times PATH_GR + P \times PATH_GR + T \times HOST_GR + P \times HOST_GR + \text{OFFSET}(NT) + (1|SPEC) + (1|PATH)$$

To control the effect of host phylogeny on the model estimates, among the random effects, we further included the nesting of all the combinations of host taxonomic levels (i.e., species-genus, species-family, species-order, species-genus-family, species-genus-order, species-family-order, and species-genus-order-family) in separate GLMM1s. We finally present the species-family model (Fig. S5 and Table S4), which showed the best performance indicated by its lowest Akaike's information criterion (AIC) with an information-theoretic approach⁴⁰.

Regarding the complexity of GLMM1, we generated and listed all possible models with different combinations of fixed predictors. We ranked these models by calculating and comparing their AICs. By this way, we were able to select the best model ($\Delta AIC = 0$) and the top models ($\Delta AIC \leq 2$). We generated an average model by calculating parameter estimates averaged from all the top models⁴¹. We listed the statistical details for the best model and the average model from top models in Table 1 and described the results from the best model in Fig. 4a–d.

Secondly, we tested the extent to which the prevalence of each specific pathogenic taxon responds to the temperature and precipitation gradients with the single-taxon model (GLMM2), taxon by taxon (*i*). Here we only analyzed the pathogenic taxa with ≥ 100 observations (Fig. 4e–g). As a result, we tested 17 pathogenic taxa by 17 separate GLMM2s. We recorded the model performances and estimates in Table S2 and visualized the results in Fig. 4f–g.

GLMM2:

$$NP_i \sim T + P + SS + \text{OFFSET}(NT_i) + (1|SPEC)$$

We checked the multicollinearity of explanatory variables, which confirmed the variance inflation factors of all the variables are under five. We further calculated conditional and marginal pseudo-R-squared with a lognormal approximation for all the GLMMs (R_c and R_m), estimating the variance explained by each presented GLMM ⁴² and their fixed factors, respectively. We checked spatial autocorrelation by the spatial correlograms for each GLMM (Fig S6).

Given that all species have thermal optima, host and pathogens each have a hump-shaped response to temperature (and, to a lesser extent, precipitation) (e.g., local adaptation constrains distributions between cold and hot limits). Thus, pathogen and host species responses to temperature could be unimodal over a wide-enough temperature range ⁴³, and warming could therefore tend to shift species distributions to the poles. Therefore, we also accounted for unimodal response curve of temperature and precipitation by adding their quadratic terms to each single-pathogenic-taxon model. We did a model selection and presented the best models with the smallest AIC values (Table S3). The marginal response curve of prevalence to temperature and precipitation were illustrated in Fig S7 and S8.

GLMM2 (non-linear):

$$NP_i \sim T^2 + T + P^2 + P + SS + \text{OFFSET}(NT_i) + (1|SPEC)$$

Sensitivity analysis

We tested the sensitivity of the presented model when fitted with three different distribution families, i.e., the quadratic parameterized negative binomial distribution family (the models presented in the main text), the Poisson distribution family, and the binomial distribution family (i.e., a logistic regression). We fitted different GLMM1s with these three different distribution families and compared their model performances and estimates. Specifically, for the GLMM1 with a binomial distribution family, instead of NP , we defined the response variable with a matrix representing the proportion of infection, of which NP was the first row and $(NT-NP)$ was the second row. In this case, no offset variable was included, so this analysis also tested the sensitivity of our results to the offset variable design.

GLMM1 with binomial error distribution (logistic regression):

$$(NP, NT-NP) \sim T + P + SS + PATH_GR + HOST_GR + T \times PATH_GR + P \times PATH_GR + T \times HOST_GR + P \times HOST_GR + (1|SPEC) + (1|PATH)$$

The estimated effects of climatic variables were similar among these different models (Table S4). We finally selected to fit and present the GLMMs with a quadratic parameterized negative binomial

distribution family (AIC = 26,826) because it showed a considerably better performance (Table S4 and S5) compared to that with either a Poisson distribution family (AIC = 62,248) or a binomial distribution family (AIC = 75,016).

We omit potential long-term changes and predicted future changes in prevalence due to the strong temporal biases of sampling in our database (Fig. 2). However, we tested the sensitivity of the presented model to the sampling biases across years, by (i) running GLMM1 for subset data from 2000–2020 and (ii) including year as an additional variable for GLMM1. The results of these two models confirmed that the historical observations and variations across years did not affect the climatic effects presented in our results (Fig. S9).

We acknowledge the possible indirect effects of climatic conditions on pathogen prevalence by the potential direct effect of climate on local host diversity and distributions. We added the geographic pattern of host diversity as an explanatory variable to GLMM1, by which we controlled the potential effect of diversity and tested the residual effect of temperature and precipitation on pathogen prevalence. Here the host diversity was measured by avian species richness in each 50×50-km grids in Europe during 2013–2017. The European Breeding Bird Atlas 2 provided the bird species observed in each grid by the European Bird Census Council ³². To fit the prevalence data with the available information of host diversity, this sensitivity analysis was performed with a subset dataset, in which only bird data within Europe (7881 of the 8822 observations) were included. Again, the resulted effect of climatic variables remained similar with the presented GLMM1 (Table S5).

All the model comparisons in this sensitivity analysis were based on the average model, which took into account all the top models of GLMM1 ($\Delta AIC \leq 2$).

We performed all the statistical and sensitivity analyses with packages ‘glmmTMB’, ‘lme4’, ‘MuMIn’, and ‘ncf’ in R 4.0.3 ^{41,44-46}.

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Declarations

Competing interests

Authors declare that they have no competing interests.

Data availability

We list the source references and details for the database in the Text S1, Fig. S1 to Fig. S4, Table S1, and Data S1 to S3. The raw database will be available at <https://zenodo.org/> upon acceptance of the manuscript. The used climatic data is publicly available from WorldClim data website (<https://www.worldclim.org/>).

Code availability

We performed all the analyses in R. The R code will be publicly available at <https://zenodo.org/> under the same folder with our pathogen prevalence database.

Figures

Figure 1

An overview of the compiled database and study system. The maps show mean annual temperature (a) and annual precipitation (b) of the sampling locations including number of birds (c) and bats (d) tested for bacteria, eukaryotes and viruses. We present the number of sampled individuals per sampling season for birds (e) and bats (f), per pathogenic taxon (g), and number of tested species of birds and bats (h). Estimated distributions of prevalence of studied pathogenic taxa (viruses in blue, eukaryotes in black and

bacteria in red) are shown for birds (i) and bats (j). The taxonomic names and acronyms for pathogenic taxa are corresponding to NCBI taxonomy database ¹⁶.

Figure 2

Count of reviewed publications for the year of sampling for bats (a) and birds (b). The year is the average of sampling years when a publication covers sampling for ≥ 2 years. There are 63 publications without information for the year when the samples were taken, which are not shown here. A full list of the included publications are available in Text S1.

Figure 3

Distributions and density plots of annual mean temperature and annual precipitation at near current (1970–2000) and future scenario (2021–2100 SSP585 and SSP126) in the study area. (a) shows the near-current temperature gradient that we used for modelling. (b) and (c) are maps for changes in temperature from near current to future scenario SSP585 and SSP126, respectively. (d) and (e) are density plots for the temperature at near current (red) and future (greys) scenario (SSP585 and SSP126, respectively). (f–j) illustrates same information for annual precipitation.

Figure 4

Estimated effects of temperature, precipitation, sample season, and host taxon. Role of temperature (a), precipitation (b), sampling season (c) and study host taxon (birds or bats) (d) on prevalence of pathogenic microbial taxa (viruses in blue, eukaryotes in black and bacteria in red) according to the multi-taxa model, which included 8,822 unique species and sampling site combinations that tested 426,141 mobile hosts. We show sampling locations of the observations in single-taxon models (e) and separate maps for each pathogenic taxon in Figs S1–S4) and the estimated effect of temperature (f) and precipitation (g) on the number of infected individuals of each analyzed pathogenic taxon. Shaded areas in (a) and (b) and error bars in (c), (d), (f), and (g) show 95% confidence intervals. Pathogenic taxa with non-significant effects (P -value > 0.05) are in grey (statistical details in Tables 1 and Table S2).

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [DataS1.csv](#)
- [DataS2.csv](#)
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- [ESMfile.docx](#)