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Research article

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

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The connections of climatic variables to zoonotic and wildlife diseases remain uncertain. Here, we compiled a systematic database for the prevalence of 121 pathogenic microbial taxa in birds (ca 376 species) and bats (ca 39 species), including 11 939 observations from over 450 000 individuals across Europe and surrounding regions. We modelled the potential connection of climatic variables with the prevalence of 75 pathogenic microbial taxa at a multi-pathogenic-taxa level and of 17 most-studied pathogenic taxa at a single-pathogenic-taxon level. According to the multi-taxa model, the prevalence of bacterial taxa was positively associated with temperature, while this association was significantly weaker for eukaryotes and viruses. The prevalence of bacterial taxa was negatively associated with rainfall, while viruses showed a positive association with rainfall. These associations between climatic variables and prevalence of pathogenic taxa were not different between bird and bat hosts. According to the single-taxon models, the prevalence of influenza A viruses, *Plasmodium*, and several bacterial taxa in birds and bats was positively associated with temperature. Rainfall showed positive associations with the prevalence of Usutu, Sindbis and Influenza A viruses but the directions of significant associations varied among bacterial taxa. Strikingly, this was evidenced also between bacterial taxa that share hosts and transmission mechanism hinting towards hitherto unknown features on pathogen ecology, e.g. *Salmonella* versus *Campylobacter* and *Anaplasma* versus *Borrelia*. Our results suggest that rising temperature and increasing precipitation will accelerate the threat of bird- and bat-associated bacterial and viral pathogens to wildlife, domesticated animals and humans, respectively. However, the idiosyncratic relationships with climatic conditions among pathogenic taxa highlight the need for pathogen-specific predictive models to understand future pathogen distributions.

Keywords: bat, bird, climate change, Europe, pathogenic microbe, precipitation, temperature, wildlife disease, zoonotic disease

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Introduction

Over 60% of emerging infectious disease events are caused by zoonotic pathogens (Jones et al. 2008). A majority of these events originate from spillover events from wildlife, including important pandemic origins, e.g., coronavirus from bats and avian influenza from birds (Jones et al. 2008, Dunn et al. 2010, Verhagen et al. 2015, Zhou et al. 2020). Climatic dynamics 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 (Harvell et al. 2002, Altizer et al. 2013, Keesing and Ostfeld 2021, Wang et al. 2021, Paniw et al. 2022). The broad risk of emerging zoonotic disease events is considered to be higher in warmer and wetter regions, e.g. in tropical forests (Allen et al. 2017). However, the effect of climate, including both temperature and rainfall, on specific zoonotic and wildlife diseases remains uncertain (Altizer et al. 2013) and contradictory (Clark et al. 2020). A typical example is the contrasting effects of temperature or latitude found in different malaria-related parasites in birds, i.e. *Plasmodium* was positively correlated with temperature while *Haemoproteus* showed the opposite association (Garamszegi 2011, Clark et al. 2020).

The challenges for a more holistic understanding of the effects of climatic variables on zoonotic and wildlife diseases could be tackled by spatially extensive pathogen surveys across climatic gradients (Clark et al. 2020). For example, the severity of avian malaria and avian pox outbreaks in Hawaii forest birds follows an elevation gradient that peaks at mid-elevation forests (Harvell et al. 2002). Such elevational gradients indirectly indicate a nonlinear response of these pathogens or their vectors and hosts to the temperatures. Meanwhile, the prevalence of malaria-related parasites increases with increasing temperature along a contrasting elevation gradient of Australian wet tropics (Zamora-Vilchis et al. 2012). This indicated a monotonically increasing disease risk with warming (Zamora-Vilchis et al. 2012). Climatic variables other than temperature may also contribute; for example, the prevalence of *Leucocytozoon* in western Palearctic birds increases along the rainfall gradient while such correlation is invisible in other malaria-related parasites (Clark et al. 2020).

Birds and bats are highly mobile vertebrates, often contributing to the large-scale transportation of pathogens (Veikkolainen et al. 2014, Verhagen et al. 2015, Zhou et al. 2020). A large proportion of these host taxa are migratory, seasonally connecting diverse and distant habitats and host communities, thereby facilitating long-distance transmission and expansive persistence of pathogens (Altizer et al. 2011). Furthermore, there is a growing body of evidence demonstrating a rapid poleward shift or range expansion of these host taxa in response to climatic warming (Chen et al. 2011), which may accelerate the invasion and adaptation of pathogens carried by them (Carlson et al. 2022). 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. These kinds of data could also pinpoint geographic regions where future disease outbreaks may emerge. However, large-scale, 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 (Garamszegi 2011, Clark et al. 2020).

Here, we present a continental-scale, multi-host, multi-pathogenic-taxon analysis across climatic gradients to test: 1) the extent to which temperature and precipitation associate with detection of pathogenic microbial taxa in birds and bats while controlling for host phylogeny; and 2) the generalizability of these climatic effects among different microbial and host taxa (birds and bats). The results facilitate effective monitoring of risks associated with wildlife and zoonotic diseases through the ability to predict pathogen prevalence changes upon climatic variation.

Material and 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 (Supporting information) found in birds and bats according to our knowledge and relevant reviews (Hubálek 2004, Olsen et al. 2006, Benskin et al. 2009, Kohl and Kurth 2014). We then went through the list taxon by taxon when searching and selecting for 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. We 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; and sampled in Europe and surrounding countries.

Using this approach, we identified 720 peer-reviewed papers (Supporting information), 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 the 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) sampled in one location during a specific period (Supporting information). For each observation, we recorded the number of tested hosts and number of infected hosts for the corresponding pathogenic taxon. These observations included the zeros where the pathogenic taxon was tested in the host species but not found. We recorded the subtype (i.e. species, strain or other specific information) of the focal pathogenic taxon if available. 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. For observations where details on ectoparasites were available, we also recorded ectoparasite species, number of birds/bats infested by ectoparasites, and number of tested and infected ectoparasites (Supporting information). Only 1424 out of the 11 939 observations in the compiled database had full information of the number of tested and infected hosts, and number of infested hosts by ectoparasites. In addition, 723 observations had specific information of the tested ectoparasite species. Thereby, ectoparasites were not included as a modelling component in the subsequent statistical analysis.

We recorded the most specific information about date and/or period of the sample collection, 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 was not available, we counted 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 covered ≥ six months. Otherwise, we calculated the median month of the sampling period, and defined December, January, and February as 'winter'; May–July as 'summer'; and March–April and August–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 centre 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 categories (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 reported province, state, autonomous community, or region as 'region', and the accuracy of those that reported city, town, commune, or municipality as 'city'.

In this way, we covered 121 pathogenic microbial taxa sampled between 1962 and 2020 (Fig. 1 and 88.5% in 2000s), and this resulted in 11 262 and 677 observations of their prevalence in > 376 bird species and > 39 bat species, respectively (Supporting information). The taxonomic names and acronyms for pathogenic taxa correspond to the NCBI taxonomy database (Schoch et al. 2020) throughout the manuscript.

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. In this way, we crosschecked 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. By mapping and visualising the sampling locations with their geographical coordinates, the observations further away from continental Europe (including Russian far east and Reunion Islands) were excluded. 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 with the NCBI taxonomy database (Schoch et al. 2020). We further excluded the taxa that are either with a 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, Supporting information).

Climatic data

We obtained the global raster maps of long-term averaged bioclimatic variables for near-current conditions (1970–2000)

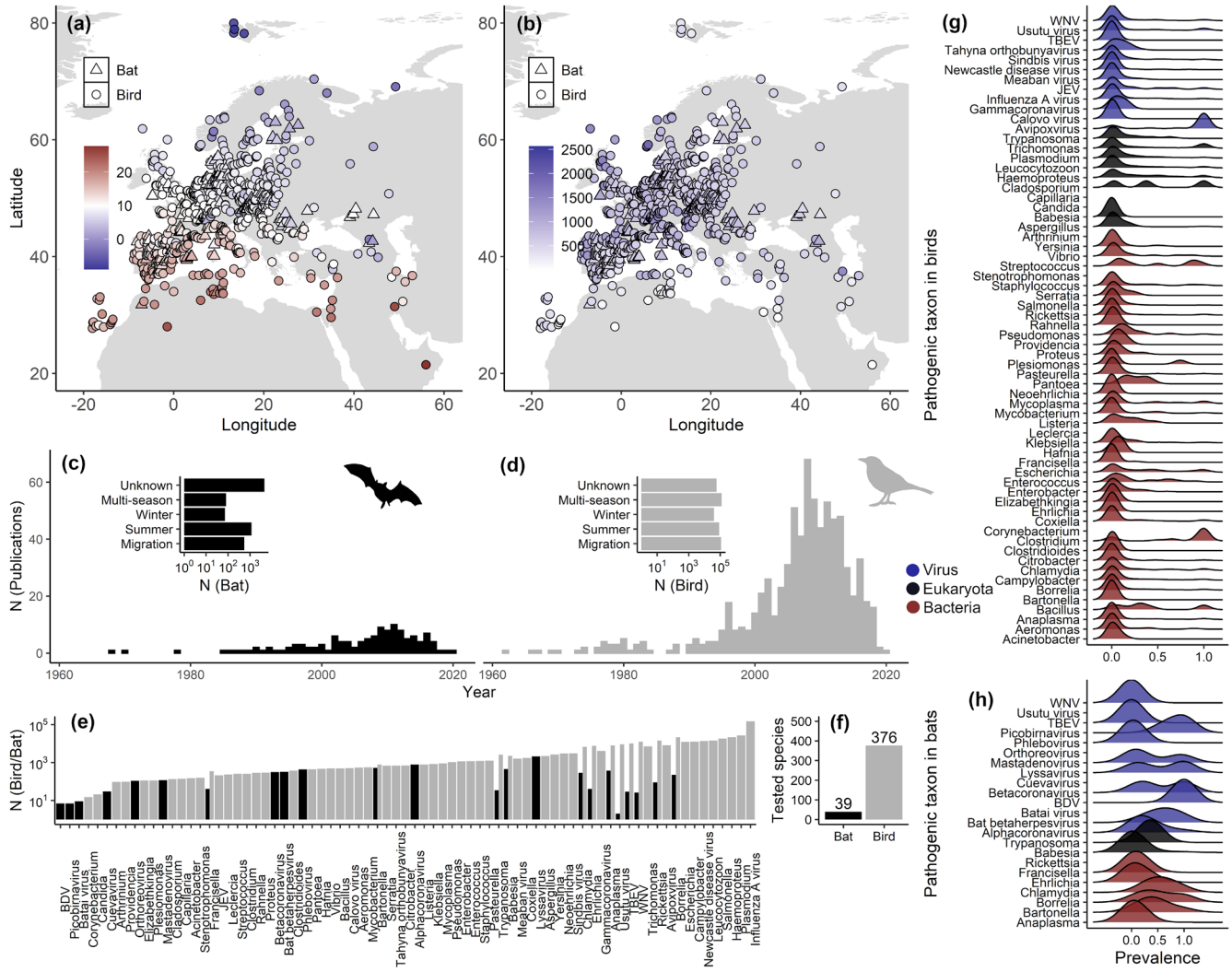

Figure 1. An overview of the compiled database and study system. The maps show the mean annual temperature (a) and annual precipitation (b) of the sampling locations. We present the count of reviewed publications for the year of sampling and the number of sampled individuals in different seasons for bats (c) and birds (d). The year is the average of sampling years when a publication covers sampling for ≥ 2 years. There are 63 out of the 720 publications without available information about the year(s) when the samples were taken, which are not shown here. We also present (e) the number of sampled individuals per pathogenic taxon and (f) number of tested species of birds and bats. Estimated distributions of prevalence of studied pathogenic taxa (viruses in blue, eukaryotes in black, and bacteria in red) are shown for birds (g) and bats (h) by density plots.

at a 10 min resolution from WorldClim ver. 2.1 (Fick and Hijmans 2017). 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.

Statistical analysis

We tested the effects of temperature and precipitation on the prevalence of pathogenic microbial taxa in birds and bats with two sets of generalized linear mixed models (GLMMs, Fig. 2, 3, Supporting information). We used the number of infected hosts (NP) as a response variable. 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.

Three different error distribution families can potentially fit the distribution of pathogen prevalence datasets, i.e. the quadratic parameterized negative binomial distribution family (the models presented in the main text), the Poisson

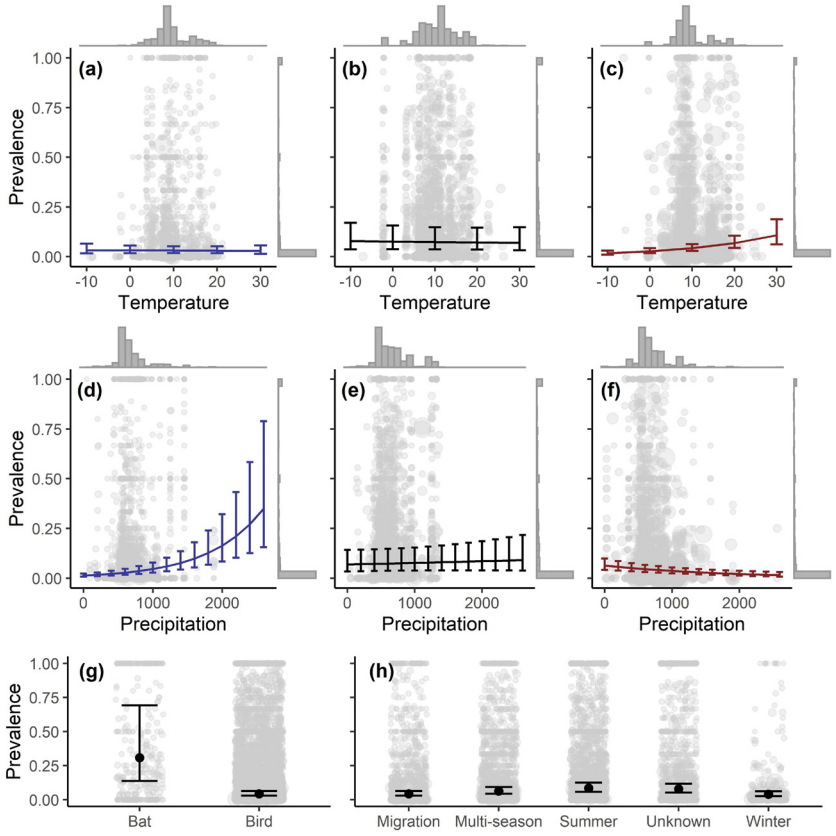

Figure 2. Results of the multi-pathogenic-taxa model. We show the marginal effects of temperature (a)–(c), precipitation (d)–(f), host taxon (g), and sample season (h) on pathogen prevalence. Regarding the interactions found between the climatic variables and pathogenic taxa groups, we show the marginal effects of temperature and precipitation for viruses (in blue, a and d), eukaryotes (in black, b and e), and bacteria (in red, c and f) in separate panels. The error bars show 95% confidence intervals of the estimated effects. The original data points are grey circles with their size weighted by number of sampled host individuals. The distributions of original data across the corresponding axis are shown by the histograms on top and right sides of the main plots (a–f). This multi-pathogenic-taxa model included 8822 unique species and sampling site combinations that tested 75 pathogenic taxa in 426 141 mobile hosts (statistical details in the Supporting information).

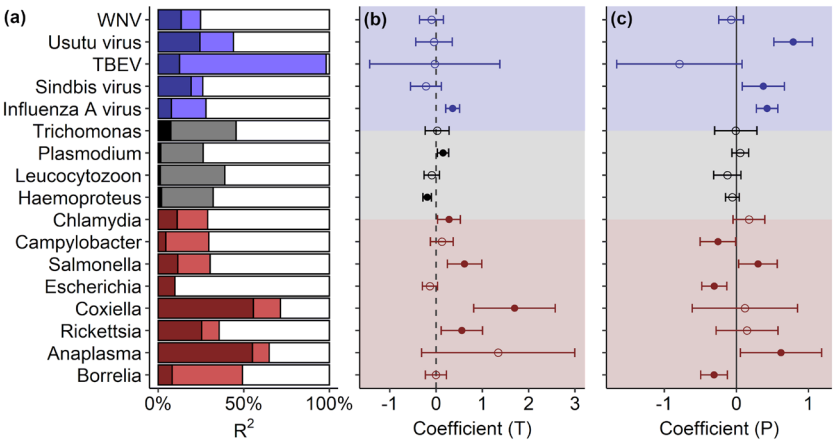

Figure 3. Results of the single-pathogenic-taxon models. (a) shows the marginal R^2 (darker coloured bars) and conditional R^2 (lighter coloured bars) of the model of each corresponding pathogenic taxon. (b) and (c) show the modelled effect of temperature and precipitation on the number of infected individuals, respectively. The error bars show 95% confidence intervals (95% CI) of the estimated coefficients. Pathogenic taxa with non-significant effects (95% CI passing zero) are distinguished by hollow circles (statistical details in the Supporting information). We distinguish the viral (in blue), eukaryotic (in black), and bacterial (in red) pathogenic taxa by colours.

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 way, we also tested the effect of the offset variable design on the analysis and results because no offset variable was included in GLMM1 with binomial error distribution.

The estimated effects of climatic variables were similar among these different models (Supporting information). We finally present the GLMMs with a quadratic parameterized negative binomial distribution family (AIC = 26 826) because it showed a considerably better performance compared to that with either a Poisson distribution family (AIC = 62 248) or a binomial distribution family (AIC = 75 016).

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: 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 (multi-taxa model):

$$\begin{aligned}\mathrm{NP} \sim\ & T + P + \mathrm{SS} + \mathrm{PATH\_GR} + \mathrm{HOST\_GR} + T \\ & \times \mathrm{PATH\_GR} + P \times \mathrm{PATH\_GR} + T \times \mathrm{HOST\_GR} \\ & + P \times \mathrm{HOST\_GR} + \mathrm{OFFSET}(\mathrm{NT}) \\ & + (1 \mid \mathrm{SPEC}) + (1 \mid \mathrm{PATH})\end{aligned}$$

To control for the effect of host phylogeny on the model estimates, we included host species identity as a nested random effect (Tucker et al. 2018). Specifically, we included the nesting of different combinations of host taxonomic levels (i.e. Genus/Species, Family/Species, Order/Species, Family/Genus/Species, Order/Genus/Species, Order/Family/Species, and Order/Family/Genus/Species) in separate GLMM1s. Except for the corresponding model of Order/Family/Genus/Species that failed to converge, the results from these different combinations did not differ from each other significantly (Supporting information). We finally present the Family/Species model (Supporting information), which showed the best performance indicated by its lowest Akaike's information criterion (AIC) with an information-theoretic approach (Nakagawa and Freckleton 2011).

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. In this way, we were able to select the best model ($\Delta\mathrm{AIC} = 0$) and the top models ($\Delta\mathrm{AIC} \leq 2$). We generated an average model by calculating parameter estimates averaged from all the top models (Barton 2015). We listed the statistical details for the best model and the average model from top models in the Supporting information and described the results from the best model in Fig. 2.

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 analysed the pathogenic taxa with ≥ 100 observations (Fig. 3). As a result, we tested 17 pathogenic taxa by 17 separate GLMM2s. We recorded the model performances and estimates in the Supporting information and visualized the results in Fig. 3.

GLMM2 (single-taxon models):

$$\mathrm{NP}_i \sim T + P + \mathrm{SS} + \mathrm{OFFSET}(\mathrm{NT}_i) + (1 \mid \mathrm{SPEC})$$

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 (Nakagawa et al. 2017) and their fixed factors, respectively. We checked spatial autocorrelation by the spatial correlograms for each GLMM (Supporting information).

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. Thereby, pathogen and host species responses to temperature could be unimodal over a wide-enough temperature range (Mordecai et al. 2017), 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 (Supporting information). The marginal response curve of prevalence to temperature and precipitation are illustrated in the Supporting information.

GLMM2 (non-linear):

$$\mathrm{NP}_i \sim T^2 + T + P^2 + P + \mathrm{SS} + \mathrm{OFFSET}(\mathrm{NT}_i) + (1 \mid \mathrm{SPEC})$$

Sensitivity analysis

We omit potential long-term changes and predicted future changes in prevalence due to the strong temporal biases of sampling in our database (Fig. 1). However, we tested the sensitivity of the presented model to the sampling biases across years, by 1) running GLMM1 for subset data from 2000–2020 and 2) 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 (Supporting information).

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 (EBCC 2022). 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 resultant effect of climatic variables remained similar with the presented GLMM1 (Supporting information). All the model comparisons in this sensitivity analysis were based on the average model, which took into account all the top models of GLMM1 ($\Delta AIC \leq 2$).

Regarding the exploratory study design, we reported the 95% confidence interval (95% CI) of the estimated coefficients for different variables from all the statistical and sensitivity analyses. We defined the significance of these effects when their 95% CI did not pass zero. We performed all the analyses with packages 'glmmTMB', 'lme4', 'MuMIn', and 'ncf' in R ver. 4.0.3 (www.r-project.org, Barton 2015, Bjornstad 2016, Magnusson et al. 2017).

Results

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 (Supporting information). By systematically reviewing 720 publications (Fig. 1), the database covered 121 pathogenic taxa (Fig. 1), mostly at the genus level, sampled from ~ 450 000 host individuals of 376 bird species and 39 bat species (Supporting information). Each observation in the database included the relevant information (Methods, Supporting information) 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 (range = 0–3955; median = 0; 1st and 3rd percentile = 0–2); and tested host individuals (1–14 080; 7; 2–29). Among these observations, the proportion of infected hosts ranged from 0 to 100% (median = 0; 1st and 3rd percentile = 0–14%). 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 to 27.7°C, annual precipitation 15–2566 mm; Fig. 1a–b).

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 the Supporting information). These models (hereafter 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, Fig. 2). 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, Fig. 3, Supporting information).

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. 2 and statistical details in the Supporting information). Based on single-taxon analyses, seven pathogenic taxa (out of 17 most-studied taxa tested) were significantly correlated with temperature. Six of these, including influenza A virus, *Plasmodium*, and four bacterial taxa (*Rickettsia*, *Coxiella*, *Salmonella*, and *Chlamydia*) were positively associated with increasing temperature (Fig. 3b, Supporting information including prevalence in different host species). Only *Haemoproteus* prevalence in birds decreased with increasing temperature (Fig. 3b). None of the tested pathogenic bacterial and viral taxa was negatively associated with temperature. The other ten pathogenic taxa (four bacterial, two eukaryotic, and four viral taxa) did not show a significant correlation with temperature.

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. 2, Supporting information). Single-taxon models revealed that, among the five tested viral species, Influenza A and Sindbis and Usutu viruses were positively associated with precipitation (Fig. 3c, Supporting information). 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. 3c). None of the tested eukaryotic taxa showed significant associations with precipitation (Fig. 3c). *Rickettsia*, *Coxiella*, WNV, and TBEV did not show a significant association with precipitation.

The associations between prevalence of pathogenic taxa and the climatic variables were not different between bird and bat hosts. However, bats had a significantly higher prevalence of the studied pathogenic taxa compared to birds (multi-taxa models, Fig. 2g). Also, there are differences among the prevalence in different seasons. According to the multi-taxa models, prevalence of pathogenic taxa in birds and bats in summer was significantly higher than that in winter and migration seasons (Fig. 2h). Altogether, climatic variables, season, and host and pathogen taxa in the multi-taxa model explained a considerable proportion of variation (n = 8822, conditional pseudo-$R^2 = 0.45$, Supporting information) in the prevalence of pathogenic taxa in birds and bats at a continental scale.

Discussion

Global temperature has been increasing and this warming is predicted to continue (Millar et al. 2017). 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. Other climatic variables are also changing globally (Gu and Adler 2015). Future predictions show that less rainfall is expected in tropical areas and more in temperate and high-latitude regions (Supporting information), thereby annual rainfall tends to change towards the upper and lower extremes (Gu and Adler 2015). Although pathogenic viral taxa show a significantly weaker connection to temperature compared to bacterial taxa, they consistently show a positive association with rainfall. This again suggests the potential of these viruses to expand towards higher latitudes with the shift in rainfall patterns (Supporting information).

Our results are consistent with previous observations for the single pathogenic taxon, which can also be explained by previously proposed mechanisms. The single-taxon model agrees with the detected contrasting responses of *Haemoproteus* and *Plasmodium* in western Palearctic birds to winter temperatures (Clark et al. 2020). The positive correlation of *Plasmodium* with temperature also agrees with the previously detected strong positive association of temperature with global avian malaria infection (Garamszegi 2011). The observed main positive role of temperature for pathogen prevalence in birds and bats could be relevant to direct (climate-pathogen) and indirect (climate-host-vector-pathogen) processes. Physiologically, warmer temperatures can facilitate survival, invasion, or reproduction of some pathogens and disease vectors (Wu et al. 2016, Mordecai et al. 2019, Iwamura et al. 2020, Chua et al. 2022). 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 survival in hosts (Harvell et al. 2002).

Ectoparasites were not included as an explicit modelling component in the current analysis, regarding their poor coverage (~12%) in the database and lack of taxonomic information on tested ectoparasite species (~94%). However, the identified temperature-prevalence associations may include interactions with arthropod vectors and other small mammals (e.g. ticks and rodents for *Borrelia*, *Rickettsia*, and *Anaplasma*). A number of studies have predicted the increasing severity and range expansions of a variety of zoonotic diseases based on arthropod vectors alone (Rupasinghe et al. 2022). An increase in parasitism and a northward invasion of tick vectors with the warming climate has been observed in Europe (Jaenson et al. 2012, Furness and Furness 2018). Such climate-driven disease dynamics were also predicted in rodents in a few studies for specific pathogens, e.g. *Yersinia pestis* and *Borrelia burgdorferi* (Stenseth et al. 2006, Roy-Dufresne et al. 2013). Therefore, climate-driven expansion and intensification of arthropod vectors and other host species involved in transmissive pathways may play a role in explaining the detected positive associations with temperature in three out of the 11 tested arthropod-vector-borne pathogenic taxa (i.e. *Rickettsia*, *Coxiella*, and *Plasmodium*) (Fig. 3b).

Both the multi-taxa and single-taxon models suggested the positive role of rainfall for the prevalence of viruses in European birds and bats. Higher precipitation can increase the population size of mosquitoes, which are vectors for both Sindbis and Usutu viruses (Clé et al. 2019, Ling et al. 2019). Also, increased availability of wetlands in regions with a higher precipitation favours waterbirds, which are important hosts for influenza A virus (Keawcharoen et al. 2008). Increased availability of wetlands in regions with a higher precipitation favours waterbirds, which are important hosts for *Salmonella* (Tizard 2004). 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. 3). Our findings highlight the importance of systematic taxa-specific follow-up studies.

The prevalence-climate relationships observed can be interactively induced by climate-driven dynamics in intrinsic susceptibility and extrinsic exposure (Sweeny and Albery 2022) to a pathogenic microbial taxon. The current dataset was unable to resolve the relative contributions of intrinsic susceptibility and extrinsic exposure. However, when controlling the effect of local host diversity in a subset of pathogenic taxa in European birds, the associations between pathogen prevalence and climatic variables remained the same as when these were not controlled for (Supporting information). In North America, the intrinsic physiological limitations were evidenced to be less important than their local host communities in shaping the distribution of *Plasmodium* and *Haemoproteus* along regional climatic gradients (Ellis et al. 2015). Local bird communities were found to be increasingly occupied by warm-dwelling species with rising temperatures in both breeding and wintering seasons (Lehikoinen et al. 2021). If warm-dwelling species tend to have a higher chance

of being infected by a focal pathogenic taxon, such host community turnover under climate change can potentially accelerate the disease risk from this pathogenic taxon. Future predictions of zoonotic and wildlife disease risks with these climatic variables should be assisted by better knowledge on the specialization of these pathogens on host species and host distributions.

Changes in disease risk under climate change can threaten the resilience and survival of wild host populations (Heard et al. 2013), and local extinction and/or population declines of some host species can promote pathogen transmission (Keesing and Ostfeld 2021, Wang et al. 2021). Although the direction of host diversity effect on disease risk remains uncertain (Salkeld et al. 2013), such alterations in host community composition could potentially reduce the community competence to pathogens by loss of dilution effect (Keesing and Ostfeld 2021) (Supporting information). In contrast, in many European areas, especially in originally species-poor areas (e.g. arctic, alpine and boreal regions), bird diversity has increased from the 1980s to the 2010s (Keller et al. 2020). This was widely explained by the climate-driven poleward range shifts of these mobile wild hosts in Europe (Lehikoinen and Virkkala 2016), which can potentially expand the distribution of disease risk (Martens et al. 1995) and cross-species transmissions (Carlson et al. 2022). Furthermore, all the pathogenic taxa in our study that were positively associated with temperature can potentially cause severe human infectious diseases (Supporting information), e.g. avian influenza, malaria, and salmonellosis. 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 change (Carlson et al. 2022). Therefore, climate change may increase the risk of spillover in new regions by facilitating expansion of pathogens and vectors to higher latitudes and altitudes (Altizer et al. 2013, Allen et al. 2017), especially via mobile hosts such as birds and bats and their climate-driven range expansions.

As with any correlational study, other aspects of geography that we did not take into account might confound our interpretation of the role of temperature and rainfall. This is because the effects of climate on zoonotic and wildlife diseases involve complex cascades among climate, resources, host assemblages, disease vectors, and pathogens (Harvell et al. 2002, Altizer et al. 2013). In addition, the distributions of most tested pathogenic taxa (14/17) did not show hump-shaped responses to temperature and precipitation (Supporting information). 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.

In summary, we show macroecological evidence that the risk of zoonotic diseases (e.g. avian influenza, salmonellosis, and borreliosis) can change spatially with shifting temperature and precipitation levels in Europe and surrounding regions by highlighting the trends from a large pathogen prevalence database. However, the responses of different pathogenic taxa show variability with regards to climatic gradients. Our open access database can further support identifying mobile host reservoirs for different zoonotic pathogens, thereby dynamically mapping and projecting disease hotspots regarding their distributions under climate change. The facilitation of effective monitoring of changes in wildlife and zoonotic diseases calls for consistent and more fine-scale spatiotemporal-reporting systems in wildlife disease surveillance. Moreover, a better understanding of seasonal patterns and environmental requirements of host species can further advance the accuracy of dynamic disease risk mappings.

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Data availability statement

Data are available from the Dryad Digital Repository: https://doi.org/10.5061/dryad.stqjq2c84 (Xu et al. 2023).

Supporting information

The Supporting information associated with this article is available with the online version.

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