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Determinants of haemosporidian single- and co-infection risks in western palearctic birds

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ABSTRACT

Understanding the drivers of infection risk helps us to detect the most at-risk species in a community and identify species whose intrinsic characteristics could act as potential reservoirs of pathogens. This knowledge is crucial if we are to predict the emergence and evolution of infectious diseases. To date, most studies have only focused on infections caused by a single parasite, leaving out co-infections. Yet, co-infections are of paramount importance in understanding the ecology and evolution of host-parasite interactions due to the wide range of effects they can have on host fitness and on the evolutionary trajectories of parasites. Here, we used a multinomial Bayesian phylogenetic modelling framework to explore the extent to which bird ecology and phylogeny impact the probability of being infected by one genus (hereafter single infection) or by multiple genera (hereafter co-infection) of haemosporidian parasites. We show that while nesting and migration behaviours influenced both the probability of being single- and co-infected, species position along the slow-fast life-history continuum and geographic range size were only pertinent in explaining variation in co-infection risk. We also found evidence for a phylogenetic conservatism regarding both single- and co-infections, indicating that phylogenetically related bird species tend to have similar infection patterns. This phylogenetic signal was four times stronger for co-infections than for single infections, suggesting that co-infections may act as a stronger selective pressure than single infections. Overall, our study underscores the combined influence of hosts' evolutionary history and attributes in determining infection risk in avian host communities. These results also suggest that co-infection risk might be under stronger deterministic control than single infection risk, potentially paving the way toward a better understanding of the emergence and evolution of infectious diseases.

1. Introduction

One of the most fundamental questions regarding the evolutionary ecology of host-parasite interactions lies in identifying the factors shaping infection risk and parasite distribution among hosts (Sutherland et al., 2013). Beyond identifying the most at-risk species in a given community (Greenberg et al., 2017), studying the ecological and the evolutionary factors that drive heterogeneity in hosts' infection risk at both the intra- and interspecific levels is paramount to understand the emergence, dynamics and evolution of infectious diseases (Streicker et al., 2013).

A number of studies, conducted on major vertebrate classes (e.g. amphibians, Greenberg et al., 2017; mammals, Dáttilo et al., 2020; birds, Barrow et al., 2019), have attempted to identify the factors involved in infection risks. At the intraspecific level, variation in infection risks has been associated with several host-specific features that impact parasite exposure (e.g. differences in behaviour, Ezenwa et al., 2016) and/or parasite susceptibility (e.g. body condition, Beldomenico et al., 2008; sex, Christe et al., 2007). At the interspecific level, several studies have shown that susceptibility to parasitic infection may be conserved through host

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phylogeny (Greenberg et al., 2017; Barrow et al., 2019). This phylogenetic signal can be explained by the inheritance of parasites from a common ancestor (e.g. encounter and/or compatibility filters shared by descent, Davies and Pedersen, 2008) or by the role played by different evolutionarily conserved factors of the host (e.g. genetic characteristics of the immune system, Minias et al., 2019; geographic range size, Waldron, 2007; life-history strategies, Valenzuela-Sánchez et al., 2021). Phylogenetically distant species can nevertheless present similar infection risk patterns if they share ecological preferences that have an influence on parasite exposure and/or parasite susceptibility (e.g. behaviour, Han et al., 2015; living environment, Menzies et al., 2021).

In nature, host individuals can encounter a multitude of different parasites during their lifetime, implying that co-infections by multiple pathogen species are likely common in wild populations (Bordes and Morand, 2011; Stutz et al., 2018; Hoarau et al., 2020). Similar to single infections, co-infections (also termed multiple infections or polyparasitism) are ubiquitous and are heterogeneously distributed among hosts. The probability of being co-infected by two or more parasites has been shown to vary considerably at both the intra- (Brooker and Clements, 2009; Susi et al., 2015) and interspecific levels (Thurber et al., 2013; González et al., 2014). Surprisingly, while the drivers of single infections (or infections in general) are relatively well studied, the drivers of co-infections are currently largely understudied, co-infection being either ignored (Soares et al., 2020; Starkloff et al., 2020) or not mentioned (Barrow et al., 2019; de Angeli Dutra et al., 2021). Yet, co-infections are of paramount importance in understanding the ecology and evolution of host-parasite interactions due to the wide range of effects they can have on host fitness (e.g. see Table 2 in Bordes and Morand, 2011). For instance, a long-term field study showed that co-infections by different blood parasite genera negatively affect the survival rate of great tits (Pigeault et al., 2018). Co-infections can also influence the evolutionary trajectories of pathogen populations through their effects on parasite life-history strategies (e.g. evolution of virulence, Alizon et al., 2013) and therefore on the within-host infection dynamics (Susi et al., 2015). For instance, in birds chronically infected by *Plasmodium* spp., the proportion of erythrocytes infected by *Plasmodium* (i.e. parasitaemia) has been shown to increase considerably after inoculating another parasite, the bacterium *Mycoplasma gallisepticum* (Reinoso-Pérez et al., 2020). Interactions between co-infecting parasites may also contribute to maintenance of the genetic variation of each parasite and impact host-parasite co-evolution (Seppälä and Jokela, 2016). Altogether, these results suggest that co-infections are important drivers of both parasite evolution and epidemiology.

Infection risk is the product of a host's exposure and susceptibility to parasites. This exposure-susceptibility interaction becomes more complex as the number of parasites potentially involved in the association increases. At first glance, one can assume that evolutionary histories and species attributes affecting single infection probabilities would affect co-infection probabilities in a similar way. However, some attributes may be much more preponderant for co-infections. For instance, by being geographically and ecologically more widespread, species with large geographic ranges and/or climatic niches should be more likely to be found in habitats that are suitable for different parasites and their other required hosts (e.g. vectors, intermediate hosts Kamiya et al., 2014). Similarly, migration may bring hosts into contact with a larger diversity of parasites (Hellgren et al., 2007). The position of species along the slow-fast life-history continuum could also be a strong predictor of co-infection risk (Vaumourin et al., 2015; Valenzuela-Sánchez et al., 2021). For instance, the differential allocation of resources to immunity between fast- and slow-living species may affect hosts' susceptibility to parasite infection and ultimately impact the frequency of co-infections.

Avian haemosporidian parasites (phylum: Apicomplexa; order: Haemosporida; Family: Plasmodiidae; Genera: *Plasmodium*, *Haemoproteus* and *Leucocytozoon,* Valkiunas, 2004) offer an exciting opportunity to better understand the distribution of infection and co-infections across multiple host species and the relative role of both ecological and phylogenetic factors over broad biogeographic areas. These blood-borne parasites, transmitted by different families of hematophagous dipteran insects (i.e. *Plasmodium* is transmitted by Culicidae, *Haemoproteus* by both Hippoboscidae and Ceratopogonidae, while *Leucocytozoon* is mainly vectored by Simuliidae), are indeed distributed worldwide and are found in most bird families (Valkiunas, 2004). While the life cycles of these blood-borne parasites have similarities (e.g. sexual reproduction in dipteran vectors and asexual reproduction in vertebrate hosts), each genus of haemosporidian parasite has specificities that impact their pathogenicity and host specificity (Atkinson et al., 2009; Ellis et al., 2020; Fecchio et al., 2020). A number of studies, carried out on different bird communities (e.g. Amazonian, African, European), have shown that vertebrate host attributes (e.g. nest characteristic, migration behaviour, length of incubation period, Scheuerlein and Ricklefs, 2004; Arriero and Møller, 2008; Ellis et al., 2020) and environmental characteristics (e.g. land cover, elevation, Barrow et al., 2019; Reis et al., 2021) are pertinent predictors of haemosporidian infection prevalence.

Co-infections by different haemosporidian parasite genera have been shown to predominate in some avian populations (e.g. Clark et al., 2016; Pigeault et al., 2018; Galen et al., 2019). Evidence from the few studies that evaluated the impact of co-infection by two haemosporidian parasite genera on host fitness suggest that co-infections have larger effects than single infections (e.g. additive cost from single to double infection in body condition and in survival probability, Marzal et al., 2008; Pigeault et al., 2018), thus potentially acting as a stronger selective pressure on host evolution. Here, we aim to quantify for the first known time the impact of multiple host ecological attributes and phylogeny on the risk of being infected by one (single infection) or by several (co-infection) haemosporidian parasite genera in 152 European bird species. While the broad definition of "co-infection" refers to infections caused by at least two parasites (either belonging to the same species or to different taxa), we here restrict this term to infections caused by at least two different genera belonging to the haemosporidian order. Hence, individual hosts characterised as "uninfected", "single-infected" or "co-infected" (see Section 2.1) are defined as such only with respect to haemosporidian parasites (this does not mean that they are not infected by other parasites). We used phylogenetic mixed-effect models to estimate the impact of climatic niche breadth and position, geographic range size, trophic niche characteristics, life-history strategies and behavioural characteristics (nesting and migration behaviours) on the probability of being infected by only one parasite genus or co-infected by several haemosporidian parasite genera. We predicted that ecological and evolutionary factors known to impact species' single infection risk likely also impact the probability of co-infection. However, as already mentioned, we expect larger effect sizes for factors that may expose host species to greater parasite or vector diversity (i.e., geographic range size, migratory behaviour) or that impact their resistance to infection (i.e., species position on the slow-fast life history continuum).

2. Materials and methods

2.1. Avian samples and parasite detection

Our data set includes 1361 samples of 152 species encompassing 44 families and 18 orders (Supplementary Table S1). Sampling was conducted on salvaged birds that were collected between 1990 and 2019 in an area of 6642 km^2 in the Lake Geneva region (Supplementary Figs. S1 and S2). The samples consisted of tissues stored in 85% ethanol at 4 °C at the Cantonal Museum of Zoology in Lausanne, Switzerland (855 specimens) and in 90% ethanol at −20 °C at the Natural History Museum of Geneva, Switzerland (506 specimens). For each individual, haemosporidian parasites (i.e. *Haemoproteus, Leucocytozoon* and *Plasmodium*) were detected from tissue samples using a nested PCR targeting a fragment of cytochrome b gene of the parasite mitochondrial genome and sequencing of positive samples (Hellgren et al. 2004, for further details see Supplementary Data S1). Birds not infected by any parasites were classified as "uninfected", birds infected with a single parasite genus were classified as "single-infected" and those infected with at least two different genera as "co-infected" (as defined in Pigeault et al., 2018).

2.2. Species attributes

2.2.1. Life-history strategies, ecological and behavioural characteristics

We used published trait data to position bird species along the slow-fast continuum of life-history variation (Storchová and Hořák, 2018). More specifically, bird position was represented by the first axis of a principal component analysis (PCA) performed on nine variables describing bird reproductive traits and maximum lifespan (Supplementary Table S1). The first axis explained 62.7% of the variability and represented a gradient going from fast (negative values) to slow (positive values) life-history strategies (for further details see Supplementary Data S1).

The trophic niche of bird species was estimated using 35 variables describing the diet (n = 14), the food acquisition behaviour (n = 9), the substrate from which food is taken (n = 9) and the daily foraging period (n = 3; Pearman et al., 2014; Supplementary Table S2). As in Pearman et al. (2014), we included body weight as a surrogate for total energy requirements. Trophic niches were represented by the scores of species along the first two axes of a Hill-Smith ordination (denoted OA; Hill and Smith, 1976). These axes roughly corresponded to the structure (from open to forest habitats; OA1 = 19.3%) and the height (from foraging underwater and in the ground to foraging in trees or during flight; OA2 = 12.6%) of the foraging environment.

The remaining traits (nest type and migration status) were extracted from Storchová and Hořák (2018). Nest type was categorised as either "open" or "closed" while migration status was categorised as "sedentary" (species living in the same area in both the breeding and the non-breeding season), "migratory" (species migrating between breeding and non-breeding season) and "facultative migrant" (species making irregular shifts in breeding and/or non-breeding season, see Supplementary Table S1).

2.2.2. Climatic niche breadth, climatic niche position and geographic range size

Estimating species realised climatic niches (i.e. the set of suitable environmental conditions accessible to the species and constrained by biotic interactions (Jackson and Overpeck, 2000) requires data for the full geographical range of species together with the corresponding environmental variables (Guisan et al., 2017). We used IUCN range maps (https://www.iucnredlist.org/) as an estimate of the geographic range of species. Environmental information was obtained in the form of climatic layers for 19 bioclimatic variables (https://www.worldclim.org/) at a 10 min resolution (approximately 340 km^2 at the equator).

To estimate species climatic niches, we first performed a PCA on the 19 bioclimatic variables from which we extracted the two first axes (explaining 55% and 19% of the total variance, respectively) to construct a two-dimensional space representing the climatic conditions available on Earth. For each species, we then selected the environmental values that were associated with its IUCN polygon within the two-dimensional space and used a kernel density estimator (KDE; see Supplementary Data S1) to delineate an envelope representing an estimate of the climatic niche of species (Fig. 1; Broennimann et al., 2012). Once the niche was defined, we extracted its area (i.e. an estimate of niche breadth) and computed its centroid (i.e. an estimate of the optimal conditions for the species) along the two PCA axes as the mean of coordinates falling inside the delimited envelope (Fig. 1). The area of the geographic range was directly extracted from IUCN polygons. To test the robustness of our results, the analyses presented below were repeated with full and facultative migrants excluded (owing to the difficulty in characterising the climatic niche of migratory species, Supplementary Fig. S3) and using two other algorithms to delineate species climatic niches (owing to the potential effect on the estimate of niche area and niche centroid, Supplementary Figs. S4-S5**,** for further details see Supplementary Data S1).

2.3. Infection prevalence among avian phylogenetic trees

To illustrate the effects of host evolutionary history on single- and co-infection probabilities, we followed Barrow et al. (2019). Specifically, we used 1000 trees from BirdTree.org (backbone tree, Hackett et al., 2008) to generate a consensus tree using the *ls.consensus* function (package phytools v. 0.6–00, Revell, 2020, Phylogenetic tools for comparative biology). The prevalence of both single- and co-infections, as well as the infection prevalence of each haemosporidian genus separately, was calculated from the raw data for each species and mapped along the phylogenetic tree using the *contMap* function (package phytools, Fig. 2, Supplementary Figs. S6-S7). Because the accuracy of prevalence estimates is lower with small sample sizes (Jovani and Tella, 2006), we visualised single- and co-infection patterns across the avian phylogeny using species for which there were at least five samples (1219 samples from 71 species belonging to 35 families from 15 orders).

2.4. Statistical analyses

Statistical analyses were conducted considering all samples (1361 individuals from 152 species) and 10 predictors: the first PCA axis summarising bird life-history strategies (i.e. position along the slow-fast life history continuum), the two OA axes summarising bird trophic niches, the climatic niche breadth, the geographic range size, the coordinates of the climatic niche centroid on each PCA axis, the migration status, the nest type and the museum affiliation (to account for potential effect related to tissue conservation). To prevent collinearity between predictors, we checked that variables had Pearson's correlation coefficients $\rho \leq |0.7|$ (Dormann et al., 2013). OA1 and the geographic range size were strongly correlated with bird life-history strategies (ρ = −0.81) and climatic niche breadth (ρ = 0.76), respectively. OA1 and the geographic range size were thus removed from the analyses. However, we also tested the effect of these variables in separate models where they replaced the variables they were correlated with (Supplementary Figs. S8-S9).

We built two types of phylogenetic multilevel models using Bayesian inference from the *brms* package (Bürkner, 2017). First, we used a phylogenetic multinomial model with a "Categorical"

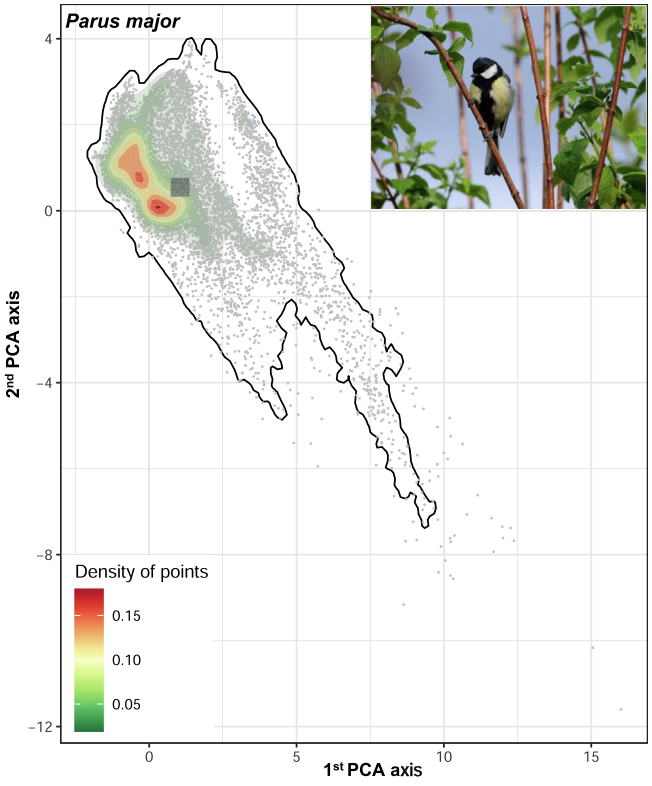

Fig. 1. Representation of the climatic niche of *Parus major*. Species climatic niche represented in the two-dimensional space defined by the two first axes of a PCA performed on a set of 19 bioclimatic variables. The envelope was estimated using a Kernel Density Estimator including 99% of occurrence points (in grey) while the centroid was computed as the average of occurrence coordinates. Photograph of *Parus major* courtesy of Philippe Christe ©.

error distribution to study the effects of species attributes and phylogeny on the probability of being single- or co-infected. We chose this distribution because the dependent variable, the infection status, contained three modalities: uninfected (coded 0), single-infected (coded 1) and co-infected (coded 2). The status 'uninfected' was set as the reference category. Note that an interesting feature of this multinomial model is that it returns the effect of predictors on both probabilities of being single- and co-infected. However, the model provides no information regarding which parasite is involved in the infection status and therefore the effect of predictors on the infection probability by each parasite. To obtain this information, we ran three independent phylogenetic generalised linear multilevel models with a "Bernoulli" error distribution to study the effect of species attributes and phylogeny on the probability of being infected by each parasite genus separately. For all models, host species and phylogeny were treated as random effects (random intercept). Continuous predictors were standardised to z-scores (mean = 0, variance = 1) to improve model convergence and parameter estimation. We used the default priors of the *brms* package and ran three chains with 11,000 iterations. The first 1,000 iterations were considered as burn-in and were thus discarded. Chains were thinned every 10 iterations. To account for phylogenetic uncertainty, we randomly sampled 100 trees from the set of trees extracted from BirdTree.org and ran the analyses on each tree. We then combined all models using the *combine_models* function. Note that this approach critically depends on the assumption that all trees are equally likely. We verified that each parameter in the model converged by checking that its potential scale reduction factor was below 1.1 (Gelman and Rubin, 1992). We followed the methods of Barrow et al. (2019) to compute the phylogenetic signal (denoted λ) regarding single- and co-infection probabilities estimated from the multinomial model, as well as for infection probability by each genus as estimated from the three generalised linear models.

To test for a potential effect of seasonality in haemosporidian infection rates (e.g. Lynton-Jenkins et al., 2020) and to account for potential degradation of our samples over time, we ran our models again with the month and the year of sampling included as factors. However, since this information was only available for 1162 individuals (85%) and since these factors had no effect on model outcomes (Supplementary Figs. S10-S11), the results presented below are based on models where these factors are not accounted for and where all samples are considered.

2.5. Data accessibility

Data supporting the results are stored in the Figshare website (10.6084/m9.figshare.17491460).

3. Results

3.1. The prevalence of haemosporidian infection varies across the avian phylogeny

We detected 486 infected individuals (35.7%), among which 101 (7.4%) were co-infected by at least two different parasite genera (20.8% of infected birds, Supplementary Tables S3 and S4). Focusing on species for which there were at least five samples, we observed a large variation in the prevalence of both single- and co-infection across the phylogeny (Fig. 2; see Supplementary Fig. S6 for the overall infection rate). Species with the highest single infection prevalence (>60% of infected individuals) included *Falco subbuteo* (92.3%, 95% confidence interval (95% CI) = 77.8–100, N_{total} = 13) and six passerines: *Turdus merula* (69%, 95% CI = 55.4–82.4, N_{total} = 45), *Fringilla montifringilla* (69%, 95% CI = 44.1–94.3, N_{total} = 13), *Corvus corone* (70%, 95% CI = 46.9–99.0, N_{total} = 11), *Alauda arvensis* (75%, 95% CI = 44.9–100, N_{total} = 5), *Phoenicurus phoenicurus* (75%, 95% CI = 44.9–100, N_{total} = 5) and *Coccothraustes coccothraustes* (100%, N_{total} = 8, Fig. 2). In contrast, 15 species belonging to nine different genera only had uninfected individuals (Fig. 2). The rate of co-infection varied greatly between species, ranging from zero individuals in 45 species to at least 33% of individuals in five species (Fig. 2, *Parus major* 48%, 95% CI = 28.4–67.6, N_{total} = 25; *Asio Otus* 50%, 95% CI = 15.4–84.6, N_{total} = 8; *Turdus philomelos* 55%, 95% CI = 38.0–70.1, N_{total} = 37; *Garrulus glandarius* 63%, 95% CI = 41.5–84.8, N_{total} = 19 and *Turdus pilaris* 67%, 95% CI = 28.59–100, N_{total} = 6). The most frequently detected parasite genus was *Leucocytozoon* (51.03% of infected birds) followed by *Plasmodium* (38.65%) and *Haemoproteus* (26.38%). The infection prevalence of each haemosporidian genus also varied greatly across the phylogeny (Supplementary Fig. S7), with some families exhibiting higher infection prevalence than others. For instance, *Leucocytozoon* infection prevalence was the highest for Corvidae (76%, N_{total} = 38) and *Plasmodium* infection prevalence was the highest for Turdidae (55%, N_{total} = 89), whereas *Haemoproteus* infection prevalence was the highest for Falconidae (33%, N_{total} = 61).

3.2. Phylogeny and hosts' features predict haemosporidian single- and co-infection risks

Unless otherwise stated, all results were consistent regarding the method used to delineate species envelopes or the removal of migratory species. The two main axes of the trophic niche (i.e. the structure (OA1) and the height of the foraging environment (OA2)), the area of the climatic niche and the location of the niche

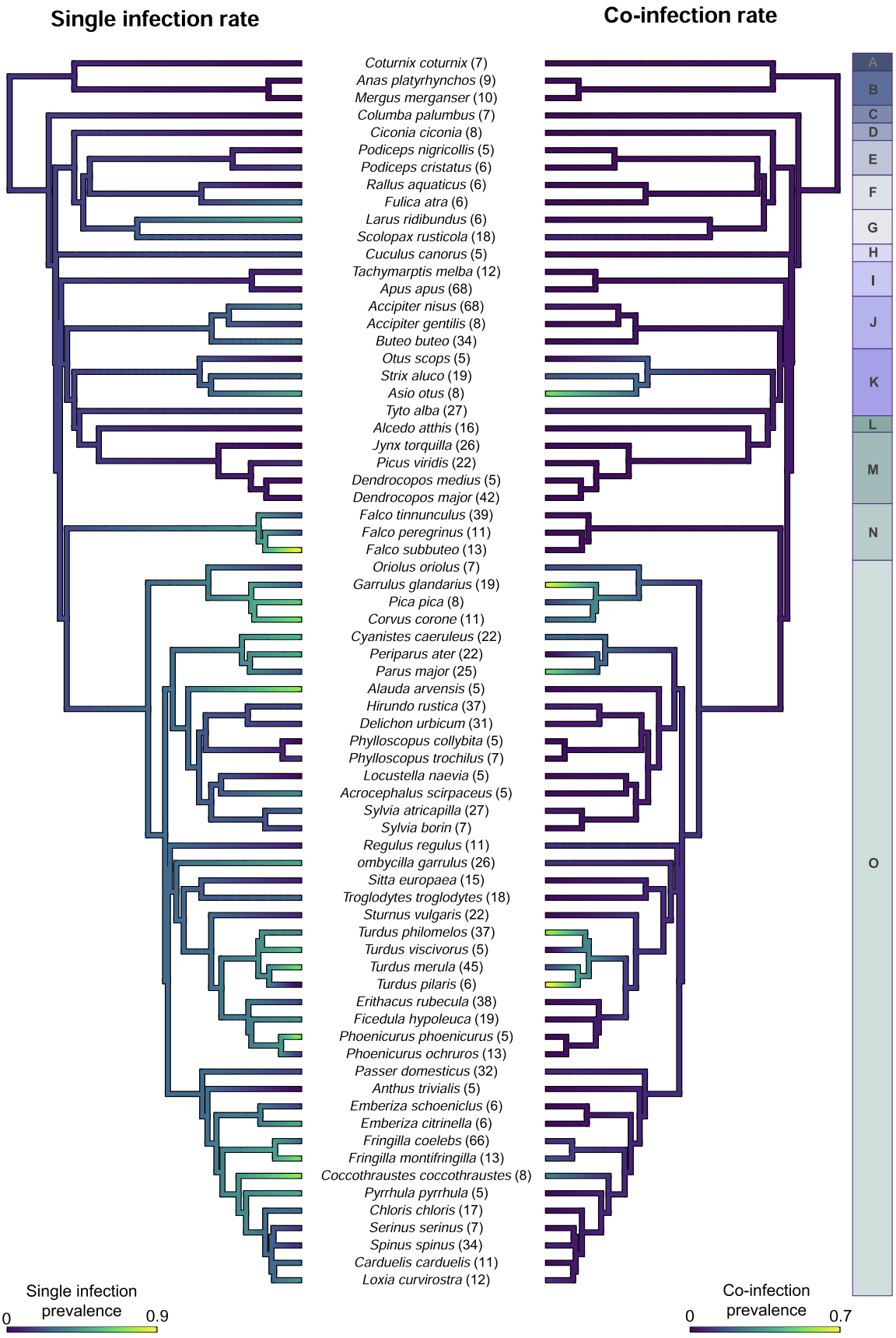

Fig. 2. Haemosporidian infection and co-infection rate across the avian phylogeny. The proportion of single- and co-infected individuals, for each bird species for which at least five individuals were sampled, was mapped as a continuous trait. The consensus tree was generated from 1000 phylogenies obtained from BirdTree.org. The numbers in parentheses correspond to the sample sizes per species. Letters (from A to O) correspond to bird orders. A: Galliforme, B: Anseriformes, C: Columbiformes, D: Ciconiiformes, E: Podicepediformes, F: Gruiformes, G: Charadriiformes, H: Cuculiformes, I: Apodiformes, J: Accipitriformes, K: Strigiformes, L: Coraciiformes, M: Piciformes, N: Falconiformes, O: Passeriformes. See also Supplementary Fig. S6.

centroid were not pertinent in explaining the probability of being single- or co-infected (i.e. the 95% CI overlapped with 0; Fig. 3). We note however that while this absence of effect was generally robust in the type of algorithm used to delineate climatic niches (Supplementary Fig. S4) and the removal of migratory species (Supplementary Fig. S3), we detected a tendency toward an effect of climatic niche breadth on co-infection probability when excluding migratory species. This suggests that species with wide climatic niche breadth tend to be more often co-infected than species with narrow niche breadth (Supplementary Figs. S3-S4). Migratory species (full and facultative migrants) and species with open nests presented a higher probability of being both single- and co-infected than species with the opposite characteristics (sedentary species and species with closed nests; Fig. 3). Fast-living species (Fig. 3) and species with a large geographic range (Fig. 3, Supplementary Fig. S8A) presented a higher probability of co-infection. Both single- and co-infection probabilities presented strong evidence for a phylogenetic effect but the signal was much lower for single- (λ = 0.12 (95% CI 0.02–0.26)) than for co-infections (λ = 0.53 (95% CI 0.25–0.76)).

Taken together, species attributes and phylogeny explained 50% (95% CI 43.3–55.0) of the variability in co-infection probability but only 26% (95% CI 21.4–29.7) of the variability in single infection probability. While effect sizes were in the same direction, the magnitude of effects was larger for co-infection than for single-infection probability.

3.3. Phylogeny and hosts' features differentially impact the probability of being infected by each haemosporidian parasite genus

None of the considered variables appeared to be a strong predictor of *Plasmodium* infection probability (Fig. 4, Supplementary Fig. S5). On the other hand, *Haemoproteus* infection probability tended to be impacted by both the height of the foraging environment and species life-history strategies, thus indicating a lower probability of infection for slow-living species and species foraging on the ground (Fig. 4). Species with open nests tended to have higher *Haemoproteus* prevalence than species with closed nests (Fig. 4). Infection probability by *Leucocytozoon* tended to increase with the geographic range size (Fig. 4) but to be lower for facultative migrants and species with closed nests (Fig. 4). The phylogenetic signal was the highest for *Leucocytozoon* (λ = 0.50 (95% CI 0.22–0.73)), followed by *Plasmodium* (λ = 0.33 (95% CI 0.10–0.61)) and then by *Haemoproteus* (λ = 0.24 (95% CI 0.02–0.59)).

4. Discussion

In this study, we evaluated the extent to which host phylogeny and species attributes related to climatic niche properties and other ecological and life-history traits influenced the probability of haemosporidian single- and co-infection in western Palearctic birds. We found that (i) while some attributes influenced both the probability of being single- and co-infected by several haemosporidian parasite genera, others were only pertinent in explaining variation in co-infection probability and (ii) phylogeny is a far more important predictor of co-infection probability than of single infection probability. Interestingly, the effect size of all predictors and the proportion of variance explained by our models were systematically larger for co-infection than for single infection probability. Altogether, these findings suggest that co-infection probability might be under a stronger deterministic control than single infection which may rather be influenced by stochastic processes (e.g. random encounter rate with parasites) or by factors not included in our study (e.g. other hosts' features).

Two parameters associated with host ecology clearly influenced single and co-infection probabilities. First, we found an effect of nesting behaviour, with the probability to be single- and co-infected being higher for species with open nests compared with closed nests. This result was also visible when considering each genus separately - although with some uncertainty regarding *Plasmodium* - which could be explained by differences in vector ecology (Santiago-Alarcon et al., 2012). Although contrasting findings have been reported (Ellis et al., 2020), several studies found consistent support for a higher risk of infection by haemosporidian parasites in species with open nests (e.g. Barrow et al., 2019; Ellis et al., 2020). This effect likely emerges because open nests can facilitate either the detection of cues used by vectors to locate their hosts (Yan et al., 2021) or the subsequent vector/host contact. Second, we found that the migratory behaviour was an important predictor of both single- and co-infection probabilities. The impact of migration on individual infection risk provided contrasting results in the literature (e.g. Ricklefs et al., 2017; Poulin and Dutra, 2021). Indeed, while migration may expose hosts to a broader range of parasites, resulting in higher parasite richness (Gutiérrez et al., 2019; Poulin and Dutra, 2021) and higher infection risk (de Angeli Dutra et al., 2021), it may also be associated with lower infection prevalence by allowing individuals to escape environments presenting a high risk of infection or by culling sick individuals (Risely et al., 2018; Poulin and Dutra, 2021). Our results rather support the "migratory escape" and/or the "culling" hypotheses, although the effect of migratory status on infection was weaker when considering each parasite genus independently, possibly due to lower sample sizes.

Previous studies have identified the slow-fast life-history continuum as a robust predictor of different ecological patterns in multiple taxonomic groups (e.g. population dynamics, Marquez et al., 2019; success of invasive species, Ducatez and Shine, 2019; range shifts, Estrada et al., 2016). Our results add to this knowledge by showing that this continuum is also relevant to describe bird infection status by haemosporidian parasites. Indeed, although this factor was not a strong predictor of single infection probability, except for *Haemoproteus*, the co-infection probability was clearly lower for slow-living species compared with fast-living species. This result contradicts the widespread assumption that slow living species should display a higher infection probability because the likelihood of encountering parasites is more important owing to their long lifespan (Poulin and Morand, 2000; Gutiérrez et al., 2019; but see Cooper et al., 2012). However, it has also been hypothesised that fast- and slow-living species are investing differently in their immune system and particularly in their adaptive (specific, less self-damaging) immune response (Millet et al., 2007; Valenzuela-Sánchez et al., 2021). For instance, to maximise their fitness along their lifespan, slow-living species may produce a more effective immune response (Tella et al., 2002; Millet et al., 2007) by producing secondary antibodies to a new antigen more rapidly, ultimately making it possible to control secondary infections more effectively than fast-living species.

The probability of being co-infected by several haemospordian parasite genera also appeared to vary depending on the size of the host geographic range, with a higher co-infection probability for species having large distributions. This result is consistent with the "geographic range hypothesis" which postulates that widespread hosts face higher parasite pressure (Price et al., 1988). Host species with a large geographic range are indeed more likely to encounter and be colonised by several species of parasites over evolutionary time, because their range overlaps with those of other "source" host species (Kamiya et al., 2014). While geographical range size has been shown to correlate positively with parasite richness in a wide range of diseases, from viruses and fungal parasites of plants (Mitchell and Power, 2003; Miller, 2012) to proto-

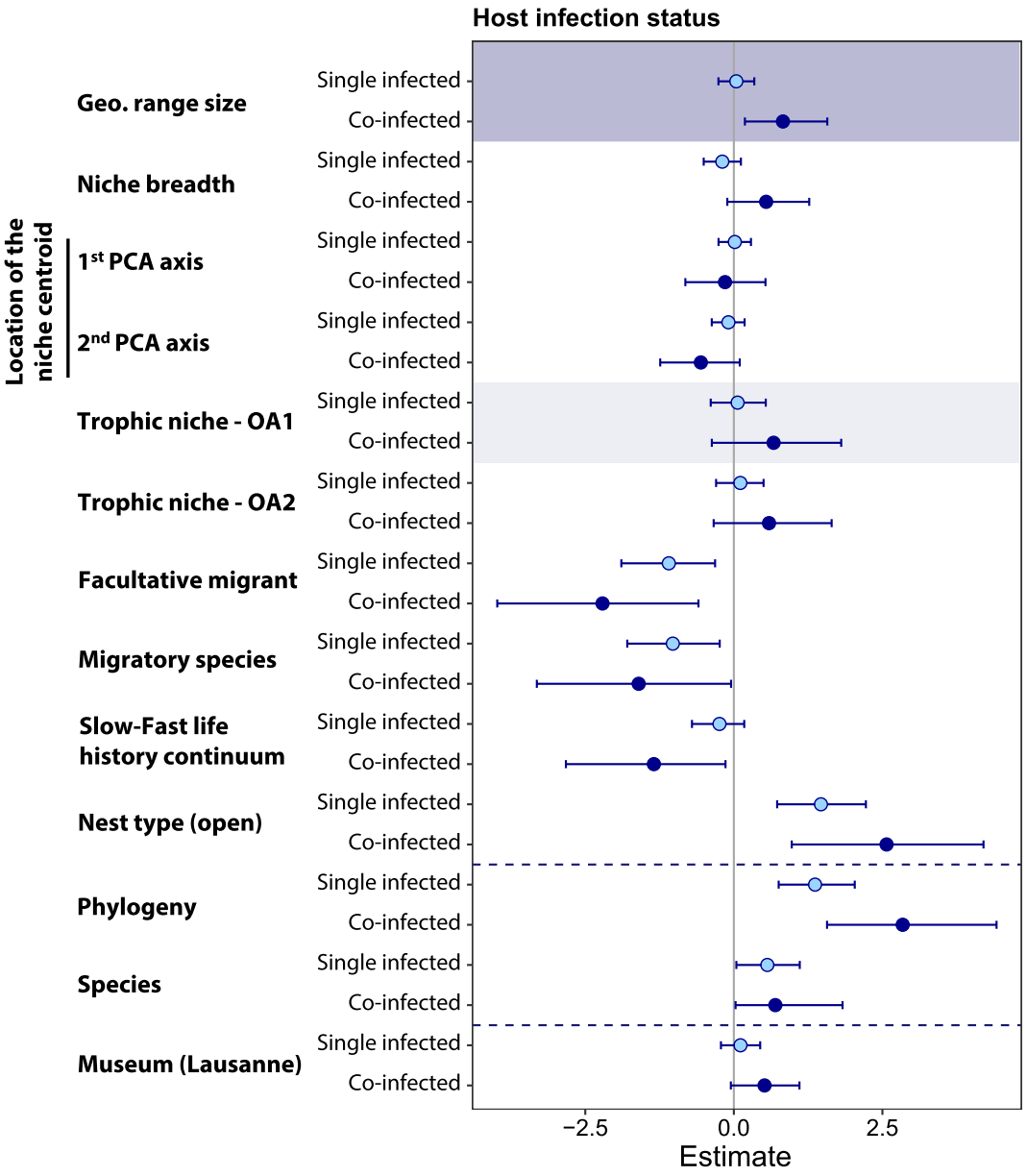

Fig. 3. Posterior mean and associated 95% credible intervals of fixed and random effects (i.e. phylogeny and host species) as estimated by the multinomial phylogenetic *brms* model with infection status (i.e. uninfected, single infected, co-infected) as the response variable. Effect sizes are represented relative to the uninfected reference category, with single infection in light blue and co-infection in dark blue. The posterior distribution of predictors and random effects with a negligible effect on single- and co-infection probabilities are expected to be centred on zero. For categorical predictors, effects are represented relative to the reference categories: Nest type (closed), migration behaviour (resident), museum (Lausanne, Switzerland). Museum affiliation was added as a predictor to account for potential effect related to tissue collection and/or conservation. Since OA1 (structure of the foraging environment) and the geographic range size were correlated with bird life-history strategies ($\rho = -0.81$) and the climatic niche breadth ($\rho = 0.76$), respectively, the posterior mean of OA1 and geographic range size presented here (grey and blue rectangles) was estimated from two other *brms* models where they replaced the variables they were correlated with and whose complete results are shown in Supplementary Fig. S8.

zoan and metazoan parasites of birds and mammals (Lindenfors et al., 2007; Gutiérrez et al., 2019), only a handful of studies have examined the relationship between species geographical range size and infection risk, yielding mixed results (Tella et al., 1999; Mlynarek et al., 2015; Suhonen et al., 2019). Here, the highlighted relationship can simply be explained by the fact that host species with large geographic ranges are more likely to move across habitats that are suitable for a diverse array of parasites (or vectors). Further, species with wide geographic ranges usually also display a broader range of tolerance to different ecological conditions (e.g. we note a positive relationship between species geographical range size and climatic niche breadth) and are thus generally considered as generalists (e.g. Slatyer et al., 2013). Generalist species, in addition to being more tolerant to variations in resources and climate, may also have evolved a higher tolerance to the pathological effects of infection (Barthel et al., 2014), ultimately leading to an increase in the prevalence of co-infection in bird populations owing to a higher survival probability of each individual.

Finally, we found that bird phylogeny explained a significant part of the variability in haemosporidian infection status, suggesting that host susceptibility, exposure or a combination of the two may be conserved across the time scale of avian diversification. Host–parasite associations usually tend to present a strong phylogenetic structure and host phylogeny has already been shown to be a relevant predictor of disease spill-over and infection risk in a range of associations (e.g. amphibian-fungal pathogens,

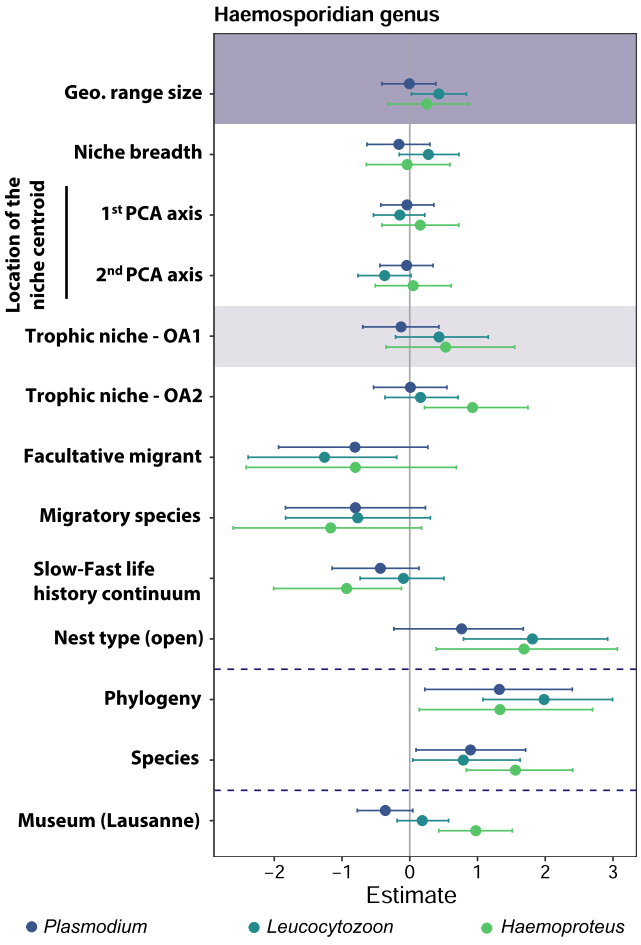

Fig. 4. Posterior mean and associated 95% credible intervals of fixed and random effects (i.e. phylogeny and host species) as estimated by Bernoulli phylogenetic *brms* models with infection status (uninfected versus infected) for each parasite genus as the response variable. For categorical predictors, effects are shown relative to the reference categories: nest type (closed), migration behaviour (resident), museum (Lausanne, Switzerland). Since OA1 (structure of the foraging environment) and the geographic range size were strongly correlated with bird life-history strategies ($\rho = -0.81$) and the climatic niche breadth ($\rho = 0.76$), respectively, the posterior mean of OA1 and geographic range size presented here (grey and blue rectangles) was estimated from two other *brms* model where they replaced the variables they were correlated with and whose complete results are shown in Supplementary Fig. S9.

Greenberg et al., 2017; mammals-virus Streicker et al., 2010). However, previous studies usually found a low congruence between the phylogeny of haemosporidian parasites and that of their avian hosts, partly due to host switching across large phylogenetic gaps (Ricklefs et al., 2014; Alcala et al., 2017). The extent to which susceptibility to haemosporidian infection is phylogenetically conserved rather than labile across bird phylogeny is therefore not well established (González et al., 2014; Barrow et al., 2019). While several factors explaining variation in single- and co-infection probabilities tend to be phylogenetically conserved (nest structure, Fang et al., 2018; position on the slow-fast life-history continuum, Valenzuela-Sánchez et al., 2021; geographic range size, Morales-Castilla et al., 2013), we found support for a strong additional phylogenetic signal in infection probability. This result echoes recent findings on an Amazonian bird community (Barrow et al., 2019), and further highlights that host phylogeny is a much stronger determinant of co-infection than of single-infection probability. This could suggest that co-infections by haemosporidian parasites can act as a strong selective pressure that may ultimately have a stronger influence on birds' evolution than single infections. Note however that this result can also be due to species differences in evolutionarily conserved factors not included in our study such as the genetic component of the immune system (Minias et al., 2019) or other mechanisms allowing a higher tolerance to the pathogenic effect of parasites (Sears et al., 2015).

Beyond the implications of this study for predicting host infection status, it demonstrates that tissue sample collections from museum specimens can be used to investigate evolutionary and ecological hypotheses at a relatively low cost and with no impact on wild populations. However, this also implies certain limitations. We emphasise that the nature of the dataset, but also some methodological choices and assumptions, may have impacted our results, although we do not think that they compromise our main conclusions. First, sampling was carried out on salvaged birds for which the cause of death was not always known. A substantial proportion of birds were found dead near human habitations or traffic lanes, suggesting that death could be the results of collisions with cars or windows. Another portion of the samples comes from a rescue center (Vaux-lierre, 1163 Etoy, Switzerland) that collects injured birds (e.g. hunting accident, collision) and brings their corpses to the museums when the individuals cannot be saved. If the animals died as a result of an accident, it is unlikely that infection with haemosporidian parasites was the cause of death. Nevertheless, we cannot exclude the possibility that our results may be biased towards weak individuals who may have less resistance to pathogens. In contrast, samples collected in the wild may be biased toward more robust individuals, suggesting that a global picture of the factors affecting infection probabilities should ideally include both types of samples. Second, unlike field studies where it is possible to adjust the sampling effort according to the study needs, collaborations with museums (or other institutes where data are not collected with a clearly-defined scientific purpose; e.g. citizen-science data) necessarily imply a dependence on the available materials. Here, several host species were represented by small sample sizes (e.g. 82 species with $N < 5$) which may influence prevalence estimates. A possibility to control this bias could be to estimate the prevalence of haemosporidian parasites in selected natural populations for a subset of species, and use a statistical test (e.g. t-test) to check if the number of samples we have considered is sufficient to provide a correct estimation of infection prevalence (a somewhat a posteriori power analysis that is classically used to estimate the minimum sample size required for an experiment, given a desired significance level, effect size, and statistical power). Despite this drawback, museum collections are invaluable because they give access to rare, protected species or those that are difficult to sample in the wild. For instance, our dataset contains 36 species for which there is currently no information on infection status in the MalAvi database (https://mbio-serv2.mbioekol.lu.se/Malavi/, Bensch et al., 2009). Third, nested PCR targeting the parasite cytochrome b gene is known to underestimate co-infection prevalence compared with other methods (e.g. microscopy and metatranscriptomics). Nonetheless, microscopy cannot be used on old tissues typical of museum collections, while metatranscriptomics has logical and financial constraints. Being aware of this potential bias, we cannot rule out the possibility that co-infections have been underestimated. Fourth, we have neglected the effect of intra-specific variability, a common flaw of trait-based analyses (Zakharova et al., 2019). Indeed, we have only considered one value (the average) to characterise species attributes whereas a higher tolerance/resistance to (co-)infections could theoretically have emerged in some populations through local adaptation. While to the best of our knowledge, no study has reported a phenomenon of local adaptation in the context of infections by haemosporidian para-

sites (e.g. Szöllősi et al 2011; Jenkins et al 2015), future studies should account for intra-specific variability when possible. Finally, our definition of co-infection (infection caused by at least two haemosporidian genera) implies that we considered birds infected by several lineages of parasite belonging to the same haemosporidian genus (i.e. mixed-lineage infection) as "single-infected". To evaluate the impact of these individuals on our inferences we ran a separate model in which individuals were classified into four categories (i.e. uninfected, single-infected, mixed-lineage infected and co-infected). The addition of the new category did not change our results regarding the effect of host ecological attributes and phylogeny on the risk of single- and co-infection (Supplementary Fig. S12). We however note an effect of the origin of the tissues on the detection of mixed infections, with a slightly higher prevalence in individuals from the Geneva museum (Geneva: 23/506, Lausanne: 16/855). This difference can be explained by the mode of preservation of the tissues or by a different bird species composition.

By taking advantage of a large set of samples associated with museum specimens and using a state-of-the-art Bayesian phylogenetic modelling framework, we identified relevant predictors of among-species differences in haemosporidian infection risks for western Palearctic birds. Interestingly, we showed that our ability to predict co-infection risk is much higher than single infection risk, suggesting that random processes (e.g. encounter rate) may be more prevalent for the latter than for the former, where deterministic processes (e.g. species attributes, evolutionary history) could have an important role. Stochastic processes that would impact the single infection risk could explain why previous studies have not found consistent patterns regarding predictors of single infection probability (see e.g. Table 1 in Ellis et al., 2020). The strong phylogenetic signal that we found regarding co-infections further suggests that co-infections might act as a strong selective pressure that could ultimately drive host evolution. These co-infections have rarely been considered in studies investigating the evolutionary ecology of host-parasite interactions and we therefore encourage future studies on this topic to do so. This study was restricted to co-infections to genera belonging to the haemosporidian complex but future studies should consider co-infections more generally i.e., including all types of parasites that have documented effects on host fitness. Expanding our understanding of the distribution of (co-)infection risk across multiple host species together with its effect on the evolution of both hosts and parasites will not only help us understand why some species are more susceptible to (co-)infection than others, but will also be of importance to address urgent public health problems regarding the emergence and evolution of infectious diseases.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijpara.2022.05.002.

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