Seabird diet and transmission routes mediate the increase in pathogen prevalence with human disturbance

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1 Abstract

Human disturbances can alter patterns of wildlife infection, but their effects are likely to differ between terrestrial and marine environments. Seabirds range across land and sea and are therefore exposed to diverse human pressures such as urbanization, agriculture, and landfills, as well as fisheries, shipping, and marine pollution. However, most literature on wildlife infection has focused on terrestrial hosts or single type of disturbance (e.g., urbanization). Studying seabirds, which exploit diverse diets and habitats, offers a unique opportunity to disentangle how disturbances at sea and on land, as well as diets, and transmission routes, can change pathogen prevalence at a global scale. We conducted a global literature review and extracted datasets on seabird infection status for a range of pathogens, seabirds diets (piscivore, invertivore, generalist), and pathogen transmission routes (contact-based, vector-borne, intermediate host) to link pathogen prevalence to human disturbances at sea and on land. Our results showed that the association between human disturbances and pathogen prevalence was mediated by species' diets and differed across transmission routes. Across gradients of human disturbance at sea, pathogen prevalence in piscivorous seabirds increased from near zero to over 0.7, particularly for contact-based pathogens. Land-based disturbance was also associated with higher pathogen prevalence in generalists across transmission routes, whereas invertivores showed a negative association with terrestrial disturbance. These results indicate that piscivores are most vulnerable in heavily disturbed marine environments, while generalists face greater risk on highly disturbed lands, suggesting that effective disease mitigation will require targeted management actions in both environments.

Keywords: Wildlife health, Anthropogenic pressure, Disease ecology, Marine environment, Terrestrial environment

2 Introduction

Disease outbreaks in wildlife occur through intrinsic and extrinsic factors and can be accelerated by human activities [1, 2]. These outbreaks pose serious threats to species by leading to higher mortality, reduced reproductive success, population decline, local extinction, and biodiversity loss [3–6]. For example, viral and bacterial diseases, including avian cholera (*Pasteurella multocida*), have caused significant mortality in aquatic birds and can lead to rapid population decline and even local extinction [7–9]. Recently, highly pathogenic avian influenza panzootic decimated seabird populations in North and South America, Europe, and Antarctica [10, 11]. Over three billion people live in areas with frequent contact with wildlife, further increasing the risk of disease transmission [12]. Urbanization creates opportunities for interactions between wildlife, livestock, and humans, modifying pathogen transmission through host density, movement, and immunity, as well as abiotic factors such as pollution and climate change [13]. Seabirds are especially vulnerable wildlife species. They are effective reservoirs for pathogens as they live long, breed in dense colonies, and have diverse diets. They also travel long distances, sometimes across continents, crossing different gradients of human impact [4, 14–16]. Half of seabird species are listed as globally threatened or near threatened by the IUCN [17]. This situation may worsen due to human-related factors such as climate change, pollution, and the dilution effect due to loss of biodiversity [2, 18–20].

Anthropogenic factors on terrestrial environments, such as urban development, alter seabirds' environments and behaviour, influencing their interactions with pathogens [13, 21, 22]. Human activities concentrate food resources and pathogen hosts in new ways. For instance, open-air landfills and agricultural sites provide abundant, predictable food that attracts large numbers of opportunistic seabirds. However, these dense foraging aggregations become hotspots for pathogen transmission, since many diseases often rely on the spatial overlap of hosts and vectors [2, 13, 23–27]. For example, birds in urbanized landscapes experience higher exposure to mosquito-borne viruses such as West Nile virus [28]. We predict that higher human pressure on terrestrial environments could lead to increased prevalence of pathogens, due to more overlap with intermediate hosts and vectors, and aggregation of species in anthropogenic food sources and contaminated habitats.

Human pressures on marine environments can have a similar impact on seabirds. Marine pollution can

introduce new pathogens to seabirds. For example, seabirds foraging near sewage outfalls or wastewater treatment plants often ingest pathogens and antimicrobial-resistant microbes originating from human waste [29]. Another example is the spread of *Toxoplasma gondii* to marine environments through urban runoffs, leading to infection in seabirds [30]. Fishing operations produce abundant waste that can be opportunistically exploited by seabirds [31, 32]. Scavenging seabirds congregate in large numbers around fishing vessels or fish processing outfalls and show a strong tendency to repeatedly return to the same feeding locations [31]. This aggregation around fishing vessels and processing facilities can increase interspecies contact and pathogen exchange. Bycatch can also indirectly increase susceptibility to novel pathogens, especially when it leads to large population declines associated with reduced genetic diversity and resilience [33]. Many pelagic and polar seabirds may rarely visit cities, but can be affected by marine-based impacts such as bioaccumulation of contaminants and climate-driven shifts in marine pathogens. For example, penguins are exposed to persistent organic pollutants, which can impair their immune function [34]. Many offshore seabirds, such as petrels and shearwaters, chronically ingest microplastics, which can increase the prevalence of pathogenic bacteria [35, 36]. Climate change is also shifting the distribution of some pathogens towards higher altitudes, mainly threatening naive species [37–39]. Accordingly, we predict that pathogens would occur more in seabirds in waters with higher cumulative human disturbance.

Seabirds' foraging ecology can also shape how human activities influence the prevalence of pathogens that seabirds encounter. Their foraging ecology can determine movement and habitat use, drawing some species into cities where they access abundant food, while leading others to avoid urban environments. Generalist seabirds are more likely to be exposed to a broader array of pathogens, particularly in anthropogenic landscapes [40]. For example, colonies of European herring gulls (*Larus argentatus*) foraging across varied habitats exhibited greater microbial pathogen diversity than those with more specialized diets [41]. However, dietary specialization does not eliminate the risk of pathogens. Foodborne pathogens such as Vibrio spp. can spread through prey species, such as shellfish, fish, zooplankton, and crustaceans [41, 42]. We predict that human-related factors would interact with seabird diet to alter the pathogen prevalence.

The transmission routes used by pathogens can determine which factors accelerate their transmission

and how human activities influence their prevalence in seabirds. Transmission occurs through vertical (passed directly from parent to offspring) and/or horizontal routes [43]. For pathogens to transmit through horizontal routes, suitable ecological and environmental conditions are required, such as direct contact with infected individuals, contaminated environments, or the presence of vectors or intermediate hosts [44, 45]. Because human disturbances can modify these conditions, for example, by concentrating animals at landfills, altering prey communities, increasing pollution, and altering food and water-borne pathogens dynamics, the potential effects of humans on pathogen prevalence depend on the transmission route.

Numerous studies have examined pathogen prevalence in wildlife species. Much of this research has focused on the relationship between pathogens and wild mammals [46, 47], species' habitat use [40], or has concentrated on specific groups of pathogens [48–51]. Among studies on wild birds [52, 53], the impacts of human disturbances were not assessed. Some studies have documented how urbanization affects pathogen prevalence; however, they have primarily focused on terrestrial species and have rarely considered species' diets [2, 54]. Predictions derived from such studies are unlikely to fully apply to seabirds, as they encounter different opportunities and constraints for pathogen transmission at sea and on land due to their unique ecology. Given the recent emergence of global wildlife pandemics, such as highly pathogenic avian influenza, linking human impacts to pathogen prevalence in seabirds can provide valuable insights for their conservation. We aim to fill the gap by addressing three questions: (1) whether the prevalence of pathogens in seabirds increases with human impact on marine and terrestrial environments, (2) if this association is influenced by an interaction with seabirds' diets, and (3) whether pathogen associations with human disturbances vary by transmission route.

3 Materials and Methods

3.1 Literature review

We conducted a comprehensive literature search in Web of Science from 2000 to 2024 to identify English publications on seabirds and pathogens. The search terms were (seabird* OR gull* OR penguin* OR albatross* OR petrel* OR fulmar* OR shearwater* OR murre* OR tern* OR skua* OR puffin* OR frigatebird* OR auklet* OR guillemot* OR kittiwake* OR cormorant* OR shag* OR booby* OR

tropicbird* OR auk* OR pelican* OR gannet*) AND (pathogen* OR disease* OR infection* OR parasite* OR virus* OR bacteria OR helminth* OR protozoa*). We restricted the search to keywords appearing in titles and abstracts to enhance relevance. We then screened titles and abstracts and excluded the ones that were irrelevant. To minimize bias, we excluded experimental studies and samples from captive birds, rehabilitation centers, and carcasses, as well as studies where the sampling effort relied on clinical signs and diagnoses were based on symptoms rather than laboratory tests. Additionally, we removed research on pooled samples, eggs, and studies that collected data from fecal samples from nests or the ground, rather than from specified individual birds, because the number of individuals was unknown in such cases. We excluded studies focused on vectors, rather than pathogens, and removed studies on shorebirds based on two criteria: species belonging to the families Scolopacidae (e.g., sandpipers), Charadriidae (e.g., plovers), Haematopodidae (e.g., oystercatchers), and Jacanidae (jacanas), and species that never occupy marine neritic or oceanic habitats at any stage of their life cycle (breeding or non-breeding) according to IUCN Red List [55]. We also removed datasets used in more than one study. In the final step, we excluded samples for which we could not obtain covariate values (primarily studies located far inland and thus lacking marine environment information).

From each study, we collected data on sample size, number of positive cases, geographic location (x and y coordinates if mentioned or coordinates extracted from Google Earth based on location name), pathogen, and detection method. If any of this data was missing in a study, we excluded that study as well. We categorized the methods for pathogen detection into two groups: 1) isolation, including methods such as DNA extraction, blood smears, physical examination, antigen detection, microscopy, culturing bacteria, and histological analysis, and 2) exposure, which includes different methods of detecting antibodies, such as Enzyme-Linked Immunosorbent Assay (ELISA) and Microscopic Agglutination Test (MAT).

For each sample, we recorded the counts of infected and uninfected individuals, the sampling year(s), geographic location, pathogen, and host species. Many studies contributed multiple samples because they reported several pathogens, species, locations, or, in a few cases, distinct sampling periods. So the number of samples in our dataset exceeds the number of studies (Fig. S2 and S5). The literature search data are available in the Supplement Materials: Pathogen Prevalence in Seabirds Dataset.

3.2 Predictor variables

We used two spatial layers that quantify overall human disturbances that may affect pathogen prevalence: the Human Footprint Index for terrestrial environments (hereafter H.Land; [56]) and the Cumulative Human Impact on Marine Ecosystems dataset (hereafter H.Sea; [57]). The H.Land layer combines eight layers of human pressure, including urban development, population density, nighttime lights, croplands, pasturelands, roads, railways, and navigable waterways. For 81 study locations that fell outside the H.Land layer (e.g., some remote small islands), we manually assigned scores by examining the corresponding areas using satellite imagery, following the methodology outlined in the original dataset [56, 58]. The H.Sea layer aggregates human impacts on global oceans from 19 anthropogenic stressors across four categories: land-based pollution (such as nutrient runoff); ocean-based activities (such as fishing); climate change; and invasive species. We created a buffer of 50 km around each data point and extracted the mean value for each covariate (H.Land and H.Sea) in each buffer. To simplify calculations and facilitate more explicit comparisons among predictors, we standardized these predictors using z-transformation [59]. According to the Pearson correlation coefficient, H.Land and H.Sea were not strongly colinear in our dataset ($r = 0.40$).

To understand the association between diet and its interaction with human impact, we extracted diet data from the EltonTraits 1.0 dataset [60]. We reclassified these diets as predominantly piscivore, predominantly invertivore, or generalist (Table 1). Species falling in the third category did not have a dominant diet except for two species that had unique diets compared to others: dolphin gull *Leucophaeus scoresbii* (dominantly scavenger) and brown skua *Stercorarius antarcticus* (mainly feeds on mammals and birds).

Table 1. Definition of each diet category extracted from EltonTraits 1.0 dataset [60].

Diet	**Definition**
Piscivore	$\geq$ 50% of the diet is fish.
Invertivores	$\geq$ 50% of the diet is invertebrates: aquatic invertebrates, shrimp, krill, squid, crustaceans, molluscs, cephalopods, polychaetes, gastropods, orthoptera, terrestrial invertebrates, ground insects, insect larvae, worms, orthopterans, and flying insects.
Generalists	None of the species-specific diets reach 50%, except for dolphin gull and brown skua.

3.3 Extracting transmission route

To understand how the relationship between human disturbance and pathogen prevalence may be altered by its transmission route, we obtained data on pathogen transmission routes from vatious sources as we could not find one comprehensive database for pathogens transmission routes related to birds. We used the Global Mammal Parasite Database (GMPD), Centers for Disease Control and Prevention (CDC), and published articles related to pathogens not found in these two databases [61, 62] (see S6). We classified these mechanisms into three categories: close and non-close contact (hereafter, contact-based), vector-borne, and intermediate host [44, 45]. Close-contact pathogens spread through direct interactions (e.g., touching and biting) between individuals, while non-close contact pathogens spread indirectly through contaminated objects (e.g., surfaces) or environments (e.g., water, food sources, or soil) [62]. In this study, we combined these two groups because many viruses and bacteria in our dataset can be transmitted via both routes. Vector-borne pathogens rely on living carriers, typically arthropods such as mosquitoes, flies, and ticks [62]. The final category, intermediate host, contains pathogens that require a secondary host to complete part of their life cycle, such as growth or asexual reproduction, before passing to their definitive host. In our dataset, some pathogens belong to more than one category [63].

3.4 Statistical analysis

Our analyses focused on identifying the factors that explain pathogen prevalence, defined as the number of infected individuals in each sample, bounded by the number of individuals tested for infection. Since many samples did not contain infected individuals, we used generalized linear mixed model (GLMM) with a zero-inflated binomial distribution (ZIB) using the R package glmmTMB [64–66]. To account for the variability that our fixed effects cannot explain, we included two random effects. The first is the seabird species nested within genus, since the evolutionary history and genetic relatedness can influence a species' susceptibility or resistance to a disease [67]. For example, birds in the order Passeriformes are more susceptible to avian malaria compared to other bird groups [68]. Then, we incorporated a random effect for study ID to control for variation specific to each study.

We modeled pathogen prevalence (Y_i) as a function of H.Land, H.Sea, and diet, and allowed excessive zeros to depend on the method of pathogen detection as

$$
\begin{aligned}
&Y_i \sim \mathrm{ZIB}(n_i, p_i, \pi_i), \\
&Y_i = \begin{cases} 0 & \text{with probability } p_i + (1-p_i)(1-\pi_i)^{n_i}, \\ k & \text{with probability } (1-p_i)\binom{n_i}{k}\pi_i^{n_i}(1-\pi_i)^{n_i-k}, \quad k = 1, \ldots, n_i, \end{cases}
\end{aligned}
\tag{1}
$$

where i is a sampling unit defined with unique pathogen, seabird species, study, and location, p_i is the probability of a zero because infection was not possible and/or not detectable (e.g., no exposure, unsuitable host, limits of the detection method; considered as the zero inflation), n_i is the number of individuals in sample i, and π_i is the probability of the individual being infected if infection is possible (i.e., probability of success) in a single trial [66].

The probability of individual infection is modelled as:

$$\begin{aligned}\text{logit}(\pi_i) = \beta_0 &+ \beta_1\,\text{diet:piscivore}_i + \beta_2\,\text{diet:generalist}_i + \beta_3\,\text{H.Sea}_i + \beta_4\,\text{H.Land}_i \\ &+ \beta_5(\text{diet:piscivore}_i \times \text{H.Sea}_i) + \beta_6(\text{diet:piscivore}_i \times \text{H.Land}_i) \\ &+ \beta_7(\text{diet:generalist}_i \times \text{H.Sea}_i) + \beta_8(\text{diet:generalist}_i \times \text{H.Land}_i) \\ &+ \upsilon_{\text{study}[i]} + \upsilon_{\text{SWG}[i]},\end{aligned} \tag{2}$$

$$\upsilon_{\text{study}[i]} \sim \mathcal{N}(0, \sigma^2_{\text{study}}), \tag{3}$$

$$\upsilon_{\text{SWG}[i]} \sim \mathcal{N}(0, \sigma^2_{\text{SWG}}), \tag{4}$$

where $\beta_{predictor}$ is the coefficient for each predictor, $\upsilon_{\text{study}[i]}$ is random effect of each study for sample i, and $\upsilon_{\text{SBSwG}[i]}$ is random effect of seabird species within genus for sample i. While H.Sea_i and H.Land_i are continuous predictors, diet:piscivore_i and diet:generalist_i are dummy variables that identify whether the species in sample unit i fall in each of these diets. The probability of a structural zero that is not defined in the binomial distribution is modelled as

$$\text{logit}(p_i) = \alpha_0 + \alpha_1\,\text{method}_i, \tag{5}$$

where p_i is the probability that sample i is zero, α_0 is the intercept, and α_1 is the coefficient for the method of pathogen detection.

We conducted two sets of GLMMs to investigate differences between pathogen transmission routes. The first set included all available data, regardless of the pathogen transmission route (hereafter, the general model set). The second set comprised separate models focusing on each of the three transmission route categories (hereafter, contact-based, vector-borne, and intermediate host).

For model selection, we used the Bayesian Information Criterion (BIC) to identify the best-fitting models [69]. To perform residual diagnostics, we applied simulation-based methods available in the

DHARMa package [70]. When the BIC-selected model showed any association with H.Sea or H.Land, we used parametric bootstrap likelihood-ratio tests to assess whether the model including H.Sea and H.Land significantly improved model fit relative to a nested model without these two covariates (for more detail, see section Parametric bootstrap likelihood ratio).

4 Results

Our literature-review dataset spans all oceans and includes samples from 5 orders, 35 seabird genera, and 65 speccies (Fig 1 and S5). The first three selected models by BIC (i.e., general, contact-based, and vector-borne models) retained both H.Sea and H.Land (Tables S1, S2, and S3), and parametric bootstrap likelihood-ratio tests showed adding human-related predictor can significantly improve the fit for the three models (general: Δ deviance = 78.2.9, $p_{\text{value}} = 0.001$; contact-based: Δ deviance = 101.2, $p_{\text{value}} = 0.001$; vector-borne: Δ deviance = 80.8, $p_{\text{value}} = 0.001$). The intermediate-host model showed no clear association with any predictors. DHARMa's outlier test detected no extreme residuals, and the residual diagnostics indicated small but significant deviations for general, contact-based, and vector-borne models, and a poor fit for the intermediate-host model (Fig S3).

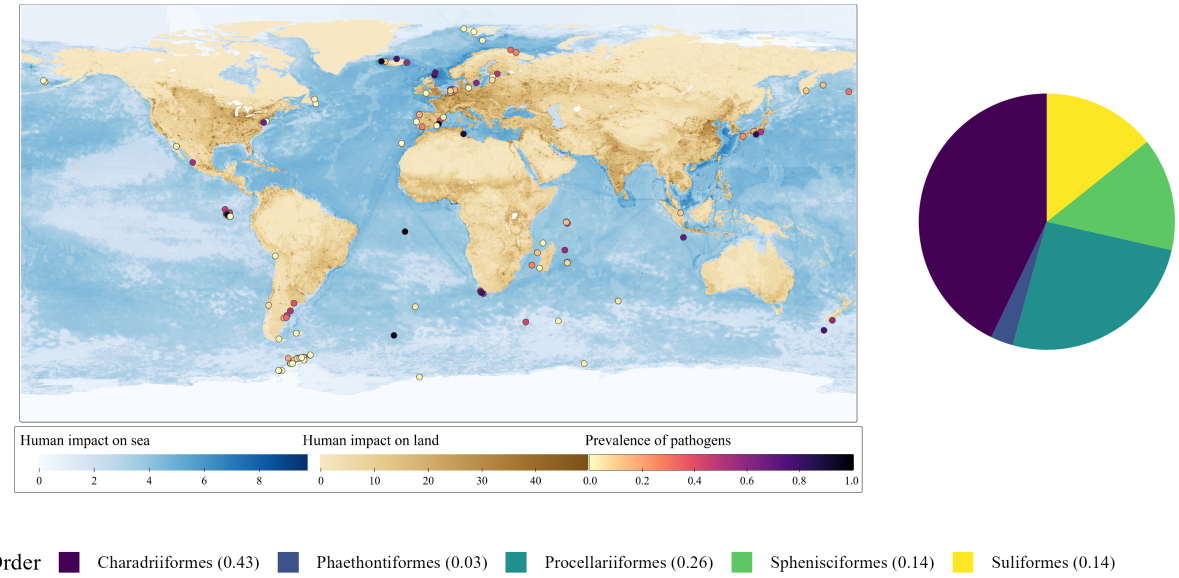

Fig 1. Global distribution of the samples and seabird orders. Each dot marks one independent dataset sample from published studies. The colour of each dot shows the pathogen prevalence reported in samples across a gradient of human impact on marine and terrestrial environments. The pie chart shows seabirds orders in the study. Our dataset contained 5 orders of seabirds with the most frequent being Charadriiformes.

Associations between pathogen prevalence and human impact varied with diet and transmission route (Fig 2 panel A; Table 2). The best model from the general set indicates that the prevalence in piscivores increased with both H.Sea and H.Land, and that the prevalence in generalists increased with H.Land. Invertivores, in contrast, showed a very small decline with increasing H.Land.

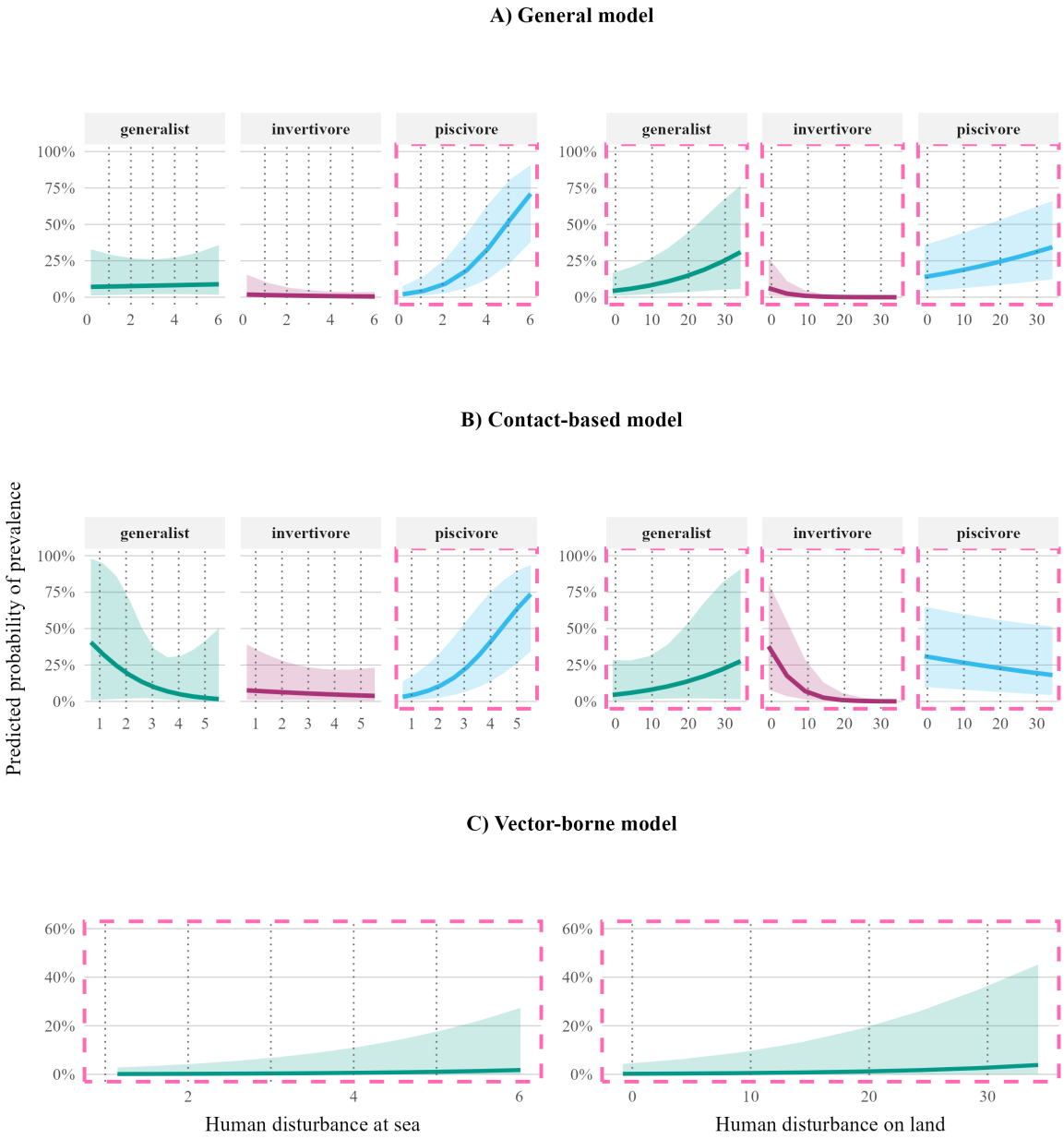

Fig 2. Pathogen prevalence as a function of H.Sea and H.Land and their interaction with 95% confidence intervals. The figures are A) general, B) contact-based, and C) vector-borne models. The main effects of H.Sea and H.Land give the slope for invertivores, and the interaction terms for generalists and piscivores show how their slopes differ from those of invertivores. Pink rectangles mark parameters whose 95% CIs do not include zero.

Table 2. Parameters in three BIC-selected models (General, Contact-based, Vector-borne). Estimates are on the logit scale. Here, FE and RE are fixed effects and random effects, respectively. The p-values in the first column are the result of the parametric bootstrap likelihood ratio test. The intercept corresponds to the reference diet (invertivore).

Model (p-value)	**Term**	**Variable**	**estimate**	**Standard error**	**p-value**
General (0.001)	FE	intercept (invertivore)	$\beta_0 = -4.75$	0.84	< 0.01
	FE	diet:piscivore	$\beta_1 = 3.2$	0.89	< 0.01
	FE	diet:generalist	$\beta_2 = 2.21$	0.88	0.01
	FE	H.Sea (invertivore)	$\beta_3 = -0.23$	0.24	0.34
	FE	H.Land (invertivore)	$\beta_4 = -1.93$	0.49	< 0.01
	FE	diet:piscivore × H.Sea	$\beta_5 = 1.02$	0.26	< 0.01
	FE	diet:piscivore × H.Land	$\beta_6 = 2.26$	0.50	< 0.01
	FE	diet:generalist × H.Sea	$\beta_7 = 0.26$	0.31	0.39
	FE	diet:generalist × H.Land	$\beta_8 = 2.59$	0.57	< 0.01
	RE	Species within genus	$\upsilon_{\text{SWG}} = 1.68$		
	RE	Genus	$\upsilon_{genus} = 1.92$		
	RE	Study	$\upsilon_{study} = 7.48$		
Contact-based (0.001)	FE	intercept (invertivore)	$\beta_0 = -2.55$	0.85	< 0.01
	FE	diet:piscivore	$\beta_2 = 1.39$	0.99	0.16
	FE	diet:generalist	$\beta_1 = 0.22$	0.92	0.81
	FE	H.Sea (invertivore)	$\beta_3 = -0.15$	0.24	0.51
	FE	H.Land (invertivore)	$\beta_4 = -2.07$	0.49	< 0.01
	FE	diet:piscivore × H.Sea	$\beta_5 = 1.04$	0.29	< 0.01
	FE	diet:piscivore × H.Land	$\beta_6 = 1.88$	0.50	< 0.01
	FE	diet:generalist × H.Sea	$\beta_7 = -0.54$	0.80	0.50
	FE	diet:generalist × H.Land	$\beta_8 = 2.68$	0.77	< 0.01
	RE	Species within genus	$\upsilon_{\text{SWG}} = 0.97$		
	RE	Genus	$\upsilon_{genus} = 1.67$		
	RE	Study	$\upsilon_{study} = 5.37$		
Vector-borne (0.001)	FE	intercept	$\beta_3 = -5.31$	1.51	< 0.01
	FE	H.Sea	$\beta_3 = 0.51$	0.12	< 0.01
	FE	H.Land	$\beta_4 = 0.82$	0.12	< 0.01
	RE	Species within genus	$\upsilon_{\text{SWG}} = 0.62$	0.38	
	RE	Genus	$\upsilon_{genus} = 3.38$		
	RE	Study	$\upsilon_{study} = 2.44$		

Best models from sets focused on specific transmission routes differed from the general results, indicating that transmission routes may alter the effect of human disturbances on pathogen prevalence (2

panels B nad C). As for the contact-based model, prevalence in piscivores increased with H.Sea, prevalence in generalists increased with H.Land, and prevalence of inventories decreased with H.Land. In contrast to the general model, pathogen prevalence in piscivores decreased with H.Land. However, the difference between the H.Land effect on piscivores and the baseline diet (invertivores) was positive. The prevalence of vector-borne pathogens was generally low, and showed small positive associations with both H.Sea and H.Land, regardless of diets (Fig 2). Other coefficients were not significant (their 95% confidence interval crossed zero; Fig S4). In the intermediate model, no covariate showed a significant association with prevalence. The intermediate dataset consisted of 85 samples from 4 studies. The small dataset may be why our models did not find any patterns.

To assess whether some seabird groups had higher overall pathogen prevalence than the average, we looked at the predicted odds ratio of pathogen prevalence for each genus. While most genera do not indicate clear deviation from the overall rate, two genera of seabirds (i.e., Pachyptila and Fregata) had odds ratios entirely above 1 in the selected vector-borne model (Fig S5).

5 Discussion

We found that pathogen prevalence in seabirds generally increased with human disturbance at sea (H.Sea) and on land (H.Land). Vector-borne pathogen prevalence rose with both H.Sea and H.Land across all diets, indicating a consistent positive association with disturbance. Diet and transmission route modified these patterns for other pathogen types. Piscivores showed particularly strong increases in prevalence with H.Sea, especially for contact-based pathogens (over 0.75 increase). Generalists had a higher prevalence with H.Land. In contrast, at high levels of H.Land, piscivores and invertivores showed lower prevalence of contact-based pathogens.

Human pressures can alter pathogen risks in wildlife through ecological and physiological mechanisms (e.g., [46, 71]). Anthropogenic food leads to different disease outcomes in wildlife, often amplifying contact-based microparasites (e.g., many bacteria and viruses) by aggregating hosts at landfills, feeders, crops, or refuse [71]. Our results for generalist seabirds showed the same trend, with increases in pathogen prevalence with human disturbance on land. However, some studies showed contradictory results for

macroparasites with complex life cycles. They suggest food provided by humans can shift away from infected intermediate hosts [46, 71]. Some studies did not show a consistent positive or negative effect of urbanisation on bird and mammal parasites. They argued that differences in prevalence can be a result of parasite types, and their transmission modes, and can increase with pollutants, chronic stress, and weak immune defences [2, 53, 72–75]. Our findings are consistent with this idea, as we also observed that the relationship between human disturbance and pathogen prevalence depends on the pathogen type and its transmission mode.

The increase in pathogen prevalence in piscivores in high human disturbance at sea (H.Sea) likely reflects their foraging ecology. Piscivores often are exposed to pathogens through their prey, as many fish serve as tolerant reservoirs for parasites [76]. Fish host many parasites to reach infective stages, so predation on fish efficiently passes on these parasites up the food chain [77]. Many marine helminths are generalists that move easily across species through food chains. At the same time, their long-lived larval stages persist in intermediate or paratenic hosts and keep infection pressure high [77]. Piscivorous seabirds often bio-accumulate contaminants as well, which can disrupt their endocrine and immune systems [78–81], increasing the risk of infection. For instance, organochlorine loads correlate with immune suppression in Caspian terns (*Sterna caspia*) and toxic pollution and heavy metals such as polychlorinated biphenyls and mercury are correlated with higher prevalence of avian influenza in waterfowls [82, 83]. The reduction in pathogen prevalence with increased disturbance on land (H.Land) association for contact-based pathogens for piscivores may result from their different behaviour in urban areas. Many seabirds are offshore species that breed on remote islets in dense colonies. However, some species are increasingly breeding in human-dominated landscapes, forming more dispersed colonies (e.g., on rooftops; [84–87]). Moreover, unlike generalists, there is limited evidence of strictly piscivorous seabirds aggregating on landfills and poultry facilities, where contact-based pathogens such as avian influenza can be transmitted. For example, a large-scale survey in the Netherlands (2006–2009) compared over 6000 birds across an area with varying gradients of urbanization, and found that avian influenza prevalence was higher in highly urbanized sites in comparison to low-urbanized sites [88]. However, piscivorous seabirds (e.g., cormorants) inhabiting urban areas aggregate around anthropogenic fish-related resources, such as aquacultural sites and fishing piers (e.g., [89, 90]). Because our H.Land layer did not include aquaculture, it may underrepresent an important

pathway of pathogen exposure for these species.

Generalist seabirds in cities frequently use landfills, agricultural areas, and other anthropogenic food sources [41, 91, 92]. Such locations can provide an opportunity for pathogen carriers to be in proximity with susceptible individuals [93, 94]. For example, ibises in Florida and gulls in the Iberian coast showed a higher prevalence of Salmonelosis and Campylobacter, respectively, in more developed areas originating from urban parks and human wastes [95–97]. In addition, this behaviour can compromise their immune systems through poor-quality food (high in fat and low in protein) [71, 98–100]. In an experimental study, mice with an anthropogenic diet showed lower ability to kill *Salmonella paratyphi* compared to the ones with a natural diet [101].

Invertivores also showed a lower prevalence in highly disturbed lands. Invertivore species in our study are mostly offshore and have limited exposure to land-based pathogens, especially those transmitted through contact [102]. And similar to piscivorous seabirds, they often do not aggregate in highly contaminated sites on land. It is important to note that the accumulated H.Land we used did not account for sewage impact, which may lead to an underrepresentation of human disturbances in such locations. Invertivores foraging on estuarine mudflats contaminated by sewage can show a higher prevalence of pathogens. For example, Olrog's gull feeding on benthic prey and crabs are exposed to pathogens originating from human disturbances [103].

Our vector-borne category aggregates pathogens transmitted by a range of arthropods, such as ticks, fleas, louse flies, and mosquitoes. We found a consistent, though slight, positive association between human impact and diets. Climate change, warmer microclimates (e.g., in cities), and higher humidity can increase the number and survival of vectors such as mosquitoes and ticks [2, 104–106]. For example, warmer winters in the Arctic are associated with higher prevalence and intensity of *Ixodes uriae* ticks in seabird colonies [9]. Recent work suggests an expansion or a change in the distribution of *I. uriae* towards higher latitude due to warmer winters in the Arctic [37]. Mosquitoes (Culex spp.) are also well adapted to man-made water bodies (e.g., treatment plants and sewers; [28]). The abundance of mosquitoes can lead to increased transmission of vector-borne pathogens, such as West Nile virus in avian species [28]. Also, simplifying bird communities and favouring competent reservoir hosts may increase the prevalence of some vector-borne viruses [37].

In our study, thin-billed prion (*Pachyptila belcheri*) and species within the genus Fregata (frigatebirds) showed higher odds of prevalence of vector-borne pathogens. A study showed that Haemoproteus *iwa* infections are particularly common in frigatebirds relative to other seabirds, and are transmitted by host-specialist hippoboscid flies *Olfersia spinifera* that are abundant on frigatebirds throughout their range in comparison to other species [107]. This result is in line with a review on seabirds [48]. They argued that since frigatebirds breed in warm, nearshore tropical environments where vectors such as louse flies (Hippoboscidae) and biting midges (Ceratopogonidae) are abundant, they can experience a high exposure to such parasites as well [48]. The same study also linked increased prevalence of another vector-borne pathogen, Plasmodium, to traits that prolong exposure at the nest, especially burrow nesting and longer fledging periods. *P. Belcheri* is a burrow-nester with a relatively long fledging period of about 50 days [108, 109].

Many seabird species shift their diet between breeding and non-breeding seasons. Quantifying diet across the full annual cycle is difficult, especially during the non-breeding period, and most traditional diet studies are limited to the chick-rearing season [110–112]. Therefore, our use of three coarse diet categories can introduce uncertainty. However, in the BIC model selection for general and contact-based pathogens, the best-supported models retained the interaction between diet and human disturbance, indicating that this covariate still captures significant variation in pathogen prevalence despite these limitations. Models without diet as a fixed effect had higher BIC values for these two models (Tables S1 to S4). The best vector-borne model result, however, did not include diet.

Our literature review identified important data gaps. We found a few studies on seabird pathogens with intermediate hosts that met our criteria. As a result, it was difficult to assess whether the lack of relationships between diet and human impacts was real or due to the small sample size. Pathogens requiring intermediate hosts can be influenced by human-modified environments. For instance, the prevalence of *T. gondii* in mammalian wildlife depends on their diet and habitat and increases with human population density [113, 114]. We also excluded many papers that lacked the necessary details about their data, such as fine-scale locations or counts of infected and uninfected individuals. We recommend future research to focus more on intermediate-host pathogens in seabirds and publish more detailed data, including diagnostic protocols, locations, sample sizes, and raw counts, to enable comparisons and

meta-analyses.

Overall, we saw that human impacts can increase the prevalence of pathogens in seabirds. One of the clearest patterns is related to piscivores and human disturance at sea. Our results showed that, through the low to high gradients of H.Sea, the prevalence of pathogens in seabirds can increase from nearly zero to around 70% and even higher in contact-based pathogens. Generalists exposed to high human disturbance on land have a higher rate of contact-transmitted pathogens. It is essential to reduce human pressure by lowering contaminants and enhancing waste treatment in the sea, as well as improving waste management on land, with an understanding of how seabirds use highly disturbed landscape on land and sea. For example, landfill management and restricting access to seairds has shown an increase in their stress levels in the short term and controversial results due to losing a reliable food (e.g., [115–117]). Identifying which combinations of diet and human disturbance drive the highest pathogen prevalence can be key to prioritizing monitoring and management efforts for seabird populations.

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Supplementary Material

A Materials and Methods

A.1 Parametric bootstrap likelihood ratio

To test the significance of each alternative model, which includes the H.Sea and H.Land, we created a null model including only diet as a fixed effect, except for vector-borne model whose selected model, and therefore the null model, did no include diet. We used this fitted model to simulate 1,000 new datasets, and then for each simulated dataset, we refitted both null and alternative models fitted based on the BIC-selected model and computed the corresponding likelihood ratios. We calculated the p-value as $(k+1)/1001$, where k is the number of simulated datasets in which the likelihood ratio was equal to or greater than that observed in the original dataset [118, 119].

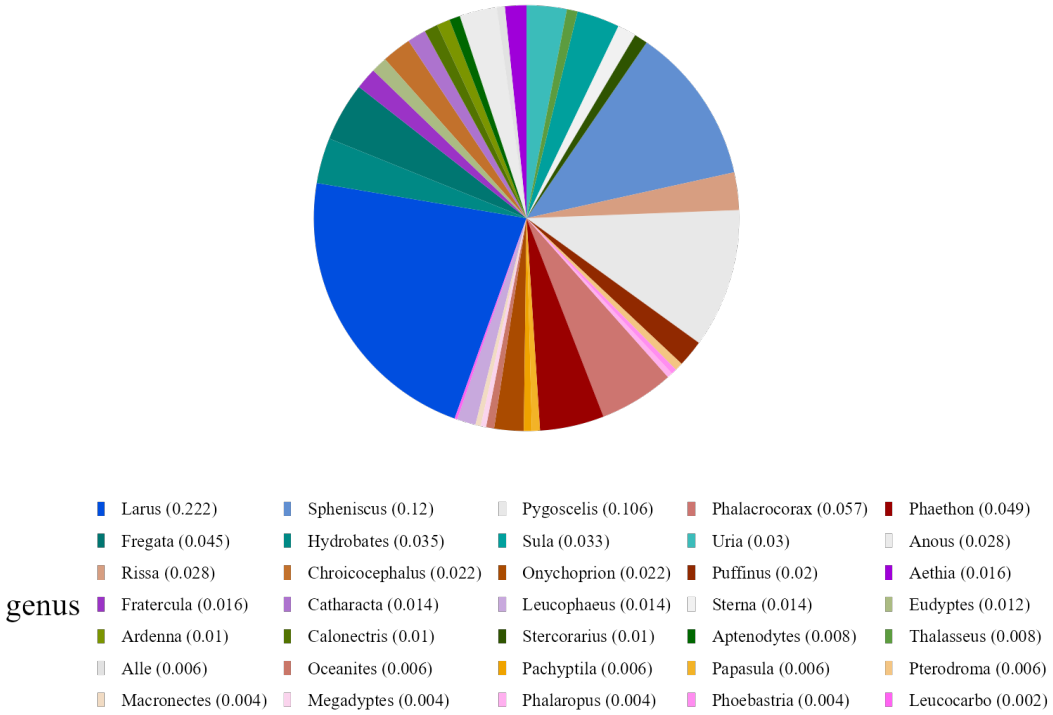

Fig S1. There are 35 genera of seabirds in our study. Gulls have the highest proportion (Larus 22%), followed by penguins (Spheniscus 11% and Pygoscelis 9%) and cormorants (Phalacrocorax 6%); all other genera each contributed 5% or less.

Table S1. The results of general BIC-selected model

Model #	Model (covariates)	BIC	ΔBIC
1	diet + H.Lnd + H.Mrn + diet:H.Lnd + diet:H.Mrn	5254.6	0.00
2	diet + H.Lnd + H.Mrn + diet:H.Lnd	5260.3	5.75
3	diet + H.Lnd + H.Mrn + diet:H.Lnd + diet:H.Mrn + zi(mtC)*	2560.6	6.05
4	diet + H.Lnd + H.Mrn + diet:H.Mrn	5265.0	10.44
5	diet + H.Lnd + H.Mrn + diet:H.Mrn + zi(mtC)	5271.1	16.51
6	diet + H.Lnd + H.Mrn + diet:H.Lnd + zi(mtC)	5271.9	17.34
7	diet + H.Mrn + diet:H.Mrn	5284.7	30.17
8	diet + H.Mrn + diet:H.Mrn + zi(mtC)	5289.8	35.23
9	diet + H.Lnd + diet:H.Lnd + zi(mtC)	5303.4	48.78
10	H.Lnd	5368.9	114.28
11	(null)	5368.6	115.01
12	H.Marine	5373.2	118.59
13	diet + H.Lnd	5373.9	119.36
14	H.Lnd + H.Mrn	5374.1	119.56
15	diet	5374.3	119.73
16	H.Land + zi(mtC)	5375.0	120.47
17	diet + H.Marine + zi(mtC)	5375.7	121.14
18	diet + H.Lnd + H.Mrn	5378.0	123.43
19	H.MArine	5379.3	124.68
20	diet + H.Land + zi(mtC)	5379.3	124.70
21	H.Land + H.Marine + zi(mtC)	5380.1	125.56
22	diet + zi(mtC)	5380.3	125.73
23	H.Marine + zi(mtC)	5380.5	125.89
24	diet + H.Lnd + H.Mrn	5384.1	129.56
25	diet + H.Lnd + zi(mtC)	5385.4	130.86
26	diet + H.Lnd + diet:H.Lnd	-	-

All models include conditional, dispersion, and zero-inflation intercepts.
**zi(mtC)* is the method of pathogen detection in each sample in the zero-inflation.

Table S2. The results of contact-based BIC-selected model

Model #	Model (covariates)	BIC	ΔBIC
1	diet + H.Lnd + H.Mrn + diet:H.Lnd + diet:H.Mrn	1592.5	0.00
2	diet + H.Lnd + H.Mrn + diet:H.Lnd + diet:H.Mrn + zi(mtC)	1594.5	2.02
3	diet + H.Mrn + diet:H.Mrn	1599.1	6.56
4	diet + H.Lnd + H.Mrn + diet:H.Mrn	1599.2	6.65
5	diet + H.Lnd + H.Mrn + diet:H.Lnd	1601.0	8.46
6	diet + H.Lnd + H.Mrn + diet:H.Lnd + zi(mtC)	1601.1	8.58
7	diet + H.Lnd + H.Mrn + diet:H.Mrn + zi(mtC)	1601.2	8.67
8	diet + H.Mrn + diet:H.Mrn + zi(mtC)	1601.5	8.98
9	diet + H.Lnd + diet:H.Lnd	1607.6	15.07
10	diet + H.Lnd + diet:H.Lnd + zi(mtC)	1610.3	17.79
11	H.Lnd	1641.4	48.92
12	H.Lnd + zi(mtC)	1643.6	51.09
13	H.Lnd + H.Mrn	1645.6	53.07
14	H.Lnd + H.Mrn + zi(mtC)	1648.1	55.56
15	H.Mrn +	1651.1	58.60
16	diet + H.Lnd	1652.1	59.57
17	(null)	1652.5	16.01
18	diet + H.Lnd + zi(mtC)	1654.2	61.73
19	H.Mrn	1654.7	62.22
20	zi(mtC)	1655.4	62.93
21	diet + H.Lnd + H.Mrn + zi(mtC)	1656.2	63.69
22	diet + H.Lnd + H.Mrn + zi(mtC)	1658.7	66.19
23	diet + H.Mrn	1661.7	69.24
24	diet	1663.2	70.69
25	diet + H.Mrn + zi(mtC)	1665.4	72.87
26	diet + zi(mtC)	-	-

All models include conditional, dispersion, and zero-inflation intercepts.

Table S3. The results of vector-borne BIC-selected model

Model #	Model (covariates)	BIC	ΔBIC
1	H.Lnd + H.Mrn	606.3	0.00
2	H.Lnd + H.Mrn + zi(mtC)	611.6	5.25
3	diet + H.Lnd + H.Mrn + diet:H.Mrn	613.8	7.45
4	diet + H.Lnd + H.Mrn	614.2	7.91
5	diet + H.Lnd + H.Mrn + diet:H.Mrn + zi(mtC)	619.0	12.68
6	diet + H.Lnd + H.Mrn + zi(mtC)	619.5	13.15
7	diet + H.Lnd + H.Mrn + diet:H.Lnd	621.7	15.38
8	H.Lnd	622.5	16.16
9	diet + H.Lnd + H.Mrn + diet:H.Lnd + diet:H.Mrn	624.0	17.72
10	diet + H.Lnd + H.Mrn + diet:H.Lnd + zi(mtC)	626.9	20.62
11	H.Lnd + zi(mtC)	627.7	21.41
12	diet + H.Lnd + H.Mrn + diet:H.Lnd + diet:H.Mrn + zi(mtC)	629.3	22.96
13	diet + H.lnd	629.9	23.59
14	diet + H.Lnd + zi(mtC)	635.1	28.84
15	diet + H.Lnd + diet:H.lnd	639.7	33.43
16	diet + H.Lnd + diet:H.lnd + zi(mtC)	645.0	38.68
17	H.Mrn	655.6	49.25
18	H.Mrn + zi(mtC)	660.8	54.49
19	diet + H.Mrn	663.8	57.49
20	diet + H.Mrn + diet:H.Mrn	668.9	63.56
21	diet + H.Mrn + zi(mtC)	669.0	62.72
22	diet + H.Mrn + diet:H.Mrn + zi(mtC)	674.1	67.81
23	(null)	676.7	70.39
24	zi(mtC)	681.9	75.63
25	diet	684.5	78.17
26	diet + zi(mtC)	689.7	83.42

All models include conditional, dispersion, and zero-inflation intercepts.

Table S4. The results of Intermediate host BIC-selected model

Model #	Model (covariates)	BIC	ΔBIC
1	(null)		0.00
2	zi(mtC)	3258.8	3.03
3	H.Mrn	3259.3	3.56
4	H.Lnd	3259.4	4.19
5	diet	3261.9	6.17
6	diet + H.Lnd + zi(mtC)	3262.5	6.75
7	H.Mrn + zi(mtC)	3262.9	7.12
8	H.Mrn + H.Lnd	3263.4	7.68
9	H.Lnd + H.Mrn	3264.2	8.48
10	diet + zi(mtC)	3264.9	9.19
11	diet + H.Lnd + H.Mrn	3266.0	10.24
12	diet + H.Lnd + H.Mrn + zi(mtC)	3266.6	10.89
13	diet + H.Mrn	3268.0	12.30
14	diet + H.Lnd + H.Mrn + zi(mtC)	635.1	28.84
15	diet + H.Mrn + diet:H.Mrn	3268.2	12.49
16	diet + H.Lnd + zi(mtC)	3268.4	12.68
17	diet + H.Lnd + diet:H.Lnd	3268.5	12.75
18	diet + H.Lnd + diet:H:Lnd + zi(mtC)	3270.6	14.87
19	diet + H.Lnd + H.Mrn + zi(mtC)	3271.7	16.01
20	diet + H.Mrn + diet:H.Mrn + zi(mtC)	3271.8	16.02
21	diet + H.Lnd + H.Mrn + diet:H.Lnd	3272.0	16.31
22	diet + H.Lnd + H.Mrn + diet:H.Lnd + diet:H.Mrn	3272.0	16.31
23	diet + H.Lnd + H.Mrn + diet:H.Mrn	3272.6	16.31
24	diet + H.Lnd + H.Mrn + diet:H.Lnd	3275.0	19.22
25	diet + H.Lnd + H.Mrn + diet.H.Mrn + zi(mtC)	3275.0	19.22
26	diet + H.Lnd + H.Mrn + diet:H.Mrn + zi(mtC)	3275.0	19.22

All models include conditional, dispersion, and zero-inflation intercepts.

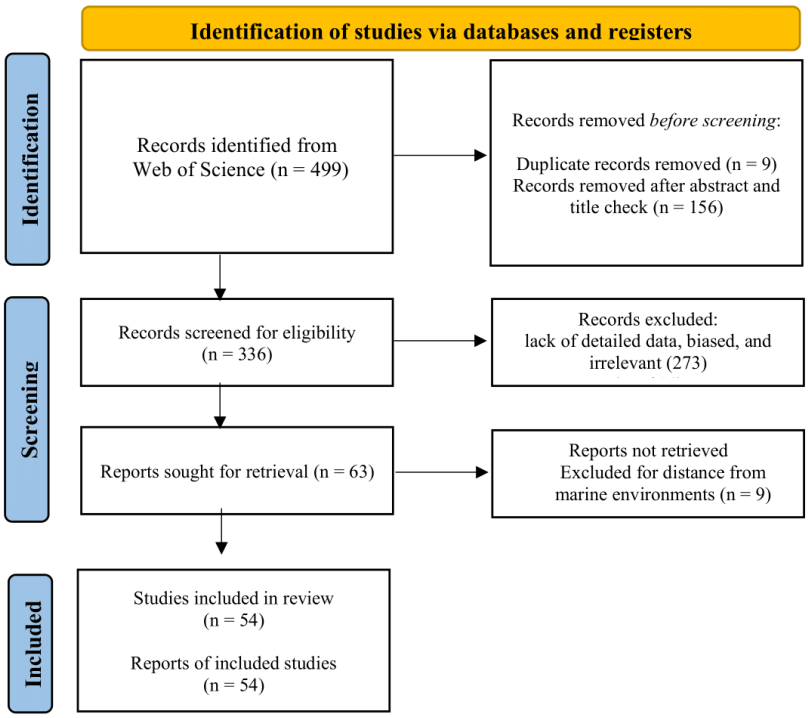

Fig S2. PRISMA flow diagram. We excluded studies lacking details on sample size, location, seabird species, or pathogen species. We also excluded study designs likely to bias our results, including experimental studies, samples from captive birds, rehabilitation centers, or carcasses, and studies where the number of individuals was not clear, and where sampling relied on clinical signs and diagnoses were based on symptoms rather than laboratory tests, and studies with sampling locations more than 50 km from the coastline. We collected 492 datasets from 54 studies, comprising 35 seabird genera and 65 species.

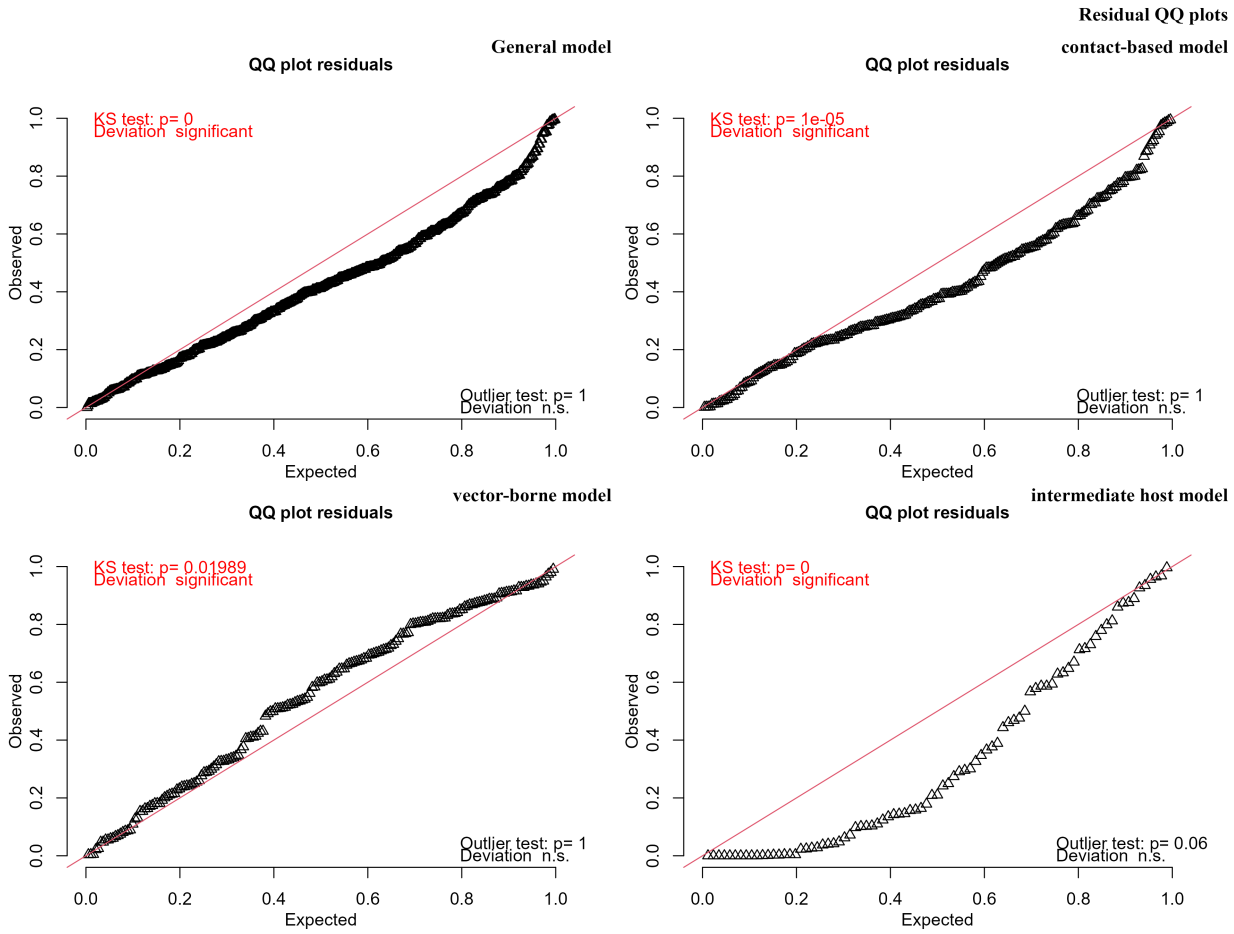

Fig S3. Residual diagnostics for prevalence models. DHARMa Q–Q plots of simulated residuals. Each panel reports the Kolmogorov–Smirnov tests. The general, contact-based, and vector-borne models show a small deviation, however the deviation for intermediate host model is large.

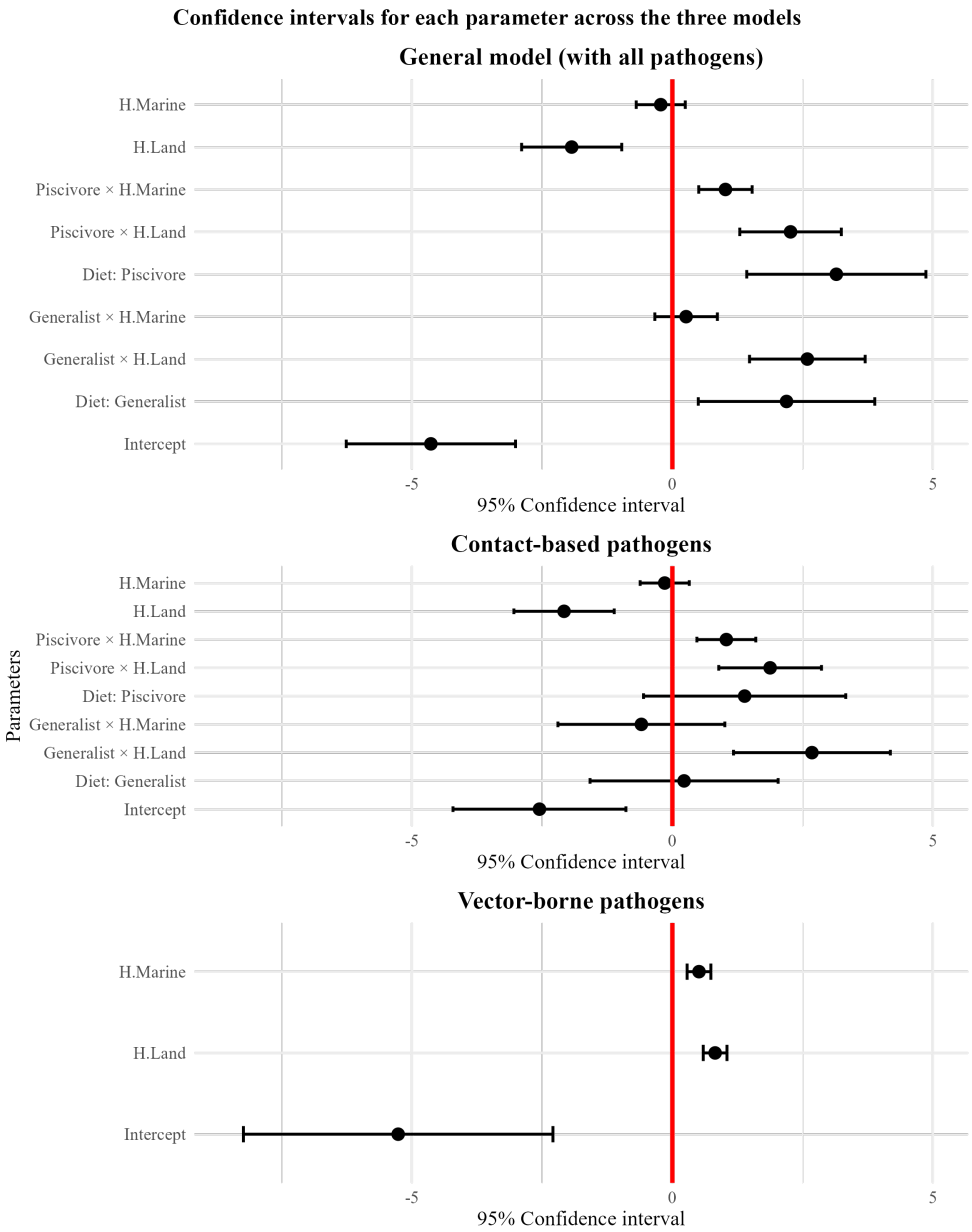

Fig S4. Predicted pathogen prevalence in seabirds by diet and human disturbance with 95% confidence interval.

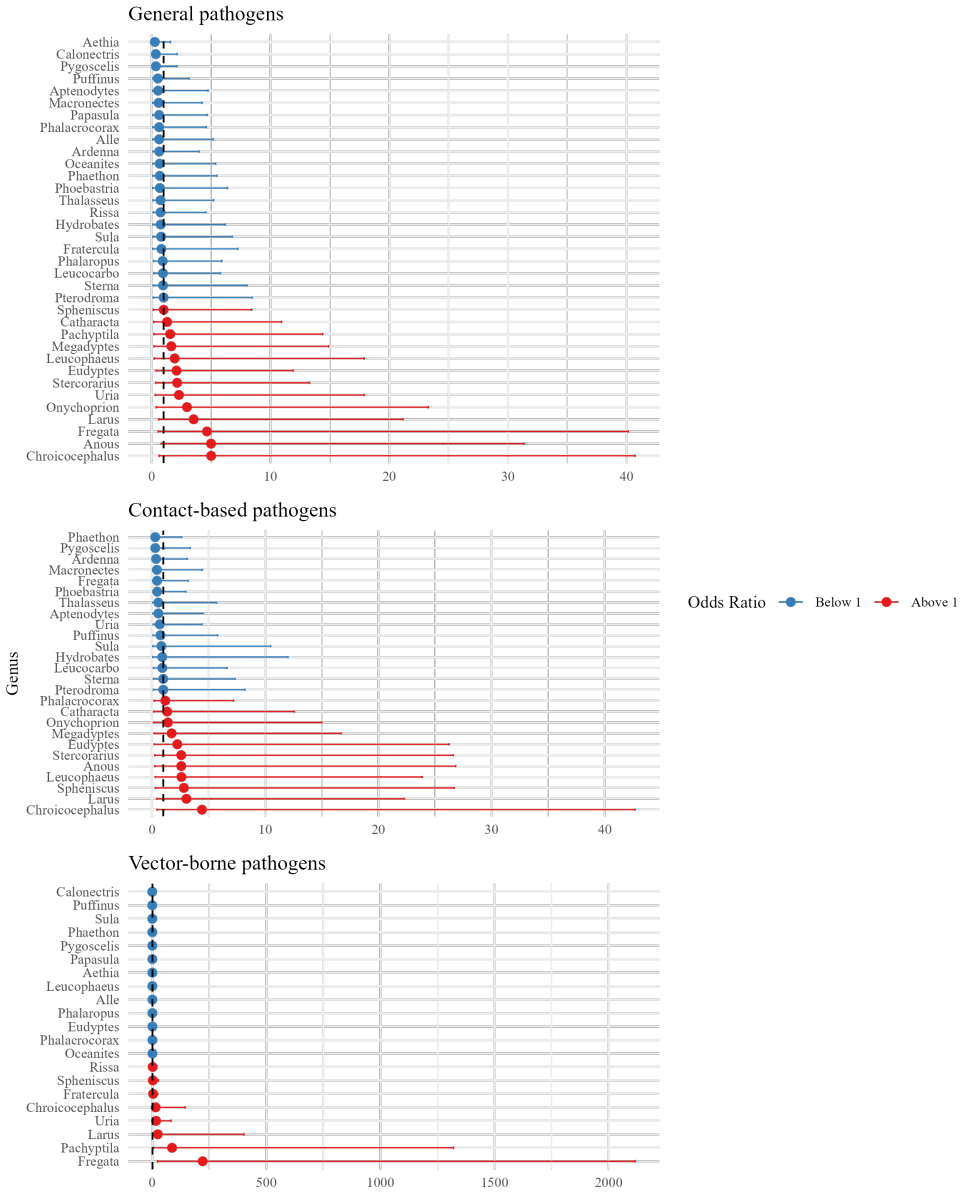

Fig S5. Odds ratio of seabird genera being infected with pathogens. Only two genera of Pachyptila and Fregata showed odds ratio over 1 with confidence interval not crossing 1 in vector-borne model. It means that these genera are more susceptible to vector-borne pathogens.

Table S5. List of papers used in the meta-analysis

Title	Reference
A spatial analysis of urban gulls contribution to the potential spread of zoonotic and antibiotic-resistant bacteria	[120]
Prevalence and pathogen load of Eimeria in wild yellow-eyed penguins *(Megadyptes antipodes)* and the morphologic characterization of a novel Eimeria species	[121]
Chlamydiaceae-Like Bacterium in Wild Magellanic Penguins *(Spheniscus magellanicus)*	[122]
Exposure of pelagic seabirds to *Toxoplasma gondii* in the Western Indian Ocean points to an open sea dispersal of this terrestrial parasite	[123]
Prevalence of antimicrobial resistance in bacteria isolated from Great Cormorants *(Phalacrocorax carbo hanedae)* in Japan	[124]
Predator and scavenger movements among and within endangered seabird colonies: Opportunities for pathogen spread	[125]
Molecular identification of avian viruses in neotropic cormorants (*Phalacrocorax brasilianus*) in Chile	[126]
Pathogen transmission risk by opportunistic gulls moving across human landscapes	[127]
Parasite diversity associated with African penguins *(Spheniscus demersus)* and the effect of host and environmental factors	[128]
Low occurrence of hemosporidian parasites in the Neotropic cormorant (*Phalacrocorax brasilianus*) in Chile	[129]

Absence of haemosporidian parasite infections in the long-lived Cory's shearwater: evidence from molecular analyses and review of the literature	[130]
First detection of avian influenza virus (H4N7) in giant petrel monitored by geolocators in the Antarctic region	[131]
Do blood parasites infect Magellanic penguins *(Spheniscus magellanicus)* in the wild? Prospective investigation and climatogeographic considerations	[132]
Avian Influenza Virus H5 Strain with North American and Eurasian Lineage Genes in an Antarctic Penguin	[133]
High prevalence of Leucocytozoon parasites in fresh water breeding gulls	[134]
Regurgitation of the koilin layer in chinstrap penguins (*Pygoscelis antarcticus*) and its association with gastric parasites	[135]
Influenza A virus on oceanic islands: Host and viral diversity in seabirds in the western Indian Ocean	[136]
Egg sampling as a possible alternative to blood sampling when monitoring the exposure of yellow-legged gulls *(Larus michahellis)* to avian influenza viruses	[137]
Serological and virological surveys of the influenza A viruses in Antarctic and sub-Antarctic penguins	[138]
Carriage of bacteria and protozoa in the intestinal tract of common tern chicks	[139]
Prevalence of Hemoproteus iwa in Galapagos Great Frigatebirds *(Fregata minor)* and Their Obligate Fly Ectoparasite *(Olfersia spinifera)*	[140]

Plasmodium blood parasite found in endangered Galapagos penguins *(Spheniscus mendiculus)*	[141]
Infection by Haemoproteus Parasites in Four Species of Frigatebirds and the Description of a New Species of Haemoproteus (*Haemosporida: Haemoproteidae*)	[142]
Comparative analysis reveals a possible immunity-related absence of blood parasites in Common Gulls *(Larus canus)* and Black-headed Gulls *(Chroicocephalus ridibundus)*	[143]
Seabirds and the Circulation of Lyme Borreliosis Bacteria in the North Pacific	[144]
Hemosporidian blood parasites in seabirds—a comparative genetic study of species from Antarctic to tropical habitats	[145]
No Evidence of Blood Parasites in Little Auks *(Alle alle)* Breeding on Svalbard	[146]
Variable exposure and immunological response to Lyme disease Borrelia among North Atlantic seabird species	[147]
Disease-limited distributions? Contrasts in the prevalence of avian malaria in shorebird species using marine and freshwater habitats	[148]
Characterization of a Novel Influenza A Virus Hemagglutinin Subtype (H16) Obtained from black-headed gulls	[149]
Multiple gene segment reassortment between Eurasian and American lineages of influenza A virus (H6N2) in guillemot *(Uria aalge)*	[150]
Infectious bursal disease virus antibodies in eider ducks and herring gulls	[151]

No evidence for high pathogenicity avian influenza in Waved Albatross (*Pvhoebastria irrorata*)	[152]
Outbreaks of highly pathogenic avian influenza (HPAI) epidemics in Baltic Great Cormorant *Phalacrocorax carbo* colonies in 2021 and 2022	[153]
Evaluation of the effect of Pb pollution on avian influenza virus-specific antibody production in black-headed gulls (*Chroicocephalus ridibundus*)	[154]
Seabirds and the circulation of Lyme borreliosis bacteria in the North Pacific	[155]

Table S6. Resources for transmission routes of the pathogens not mentioned in GMPD or CDC

Title	Reference
Shedding and Emission of Airborne Viral Microorganisms from Animal Houses	[156]
Poultry and avian diseases	[157]
Parasites of chinstrap penguins (*Pygoscelis antarctica*) from three localities in the Antarctic Peninsula and a review of their parasitic fauna	[158]
Corynebacterium diphtheriae	[159]
Defining routes of airborne transmission of Pseudomonas aeruginosa in people with cystic fibrosis	[160]
Staphylococcus xylosus and Staphylococcus aureus as commensals and pathogens on murine skin	[161]
Mycoplasma infections	[162]
The transmission and development of Cosmocephalus obvelatus (Nematoda: Acuarioidea) of gulls (Laridae)	[163]
Fishborne zoonotic heterophyid infections: an update	[164]
Transatlantic discovery of Notocotylus atlanticus (Digenea: Notocotylidae) based on life cycle data	[165]
Evolution and historical biogeography of a parasite–host assemblage: Alcataenia spp.(Cyclophyllidea: Dilepididae) in Alcidae (Charadriiformes)	[166]
Assessing the effects of trematode infection on invasive green crabs in eastern North America	[167]
Pathology of proventricular tetrameriasis in a free-range chicken	[168]
Canine and feline cardiopulmonary parasitic nematodes in Europe: emerging and underestimated	[169]
Adenovirus transmission—worthy of our attention	[170]

Animal herpesviruses and their zoonotic potential for cross-species infection	[171]
Avian reticuloendotheliosis in chickens–an update on disease occurrence and clinical course	[172]
Longitudinal map of transcriptome changes in the Lyme pathogen Borrelia burgdorferi during tick-borne transmission	[173]
Chlamydiaceae: diseases in primary hosts and zoonosis	[174]
Discovery of a novel circulation route of free-living Serratiasymbiotica mediated by predatory ladybird beetles	[175]