Article

A meta-analysis on global change drivers and the risk of infectious disease

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Anthropogenic change is contributing to the rise in emerging infectious diseases, which are significantly correlated with socioeconomic, environmental and ecological factors[1]. Studies have shown that infectious disease risk is modified by changes to biodiversity[2–6], climate change[7–11], chemical pollution[12–14], landscape transformations[15–20] and species introductions[21]. However, it remains unclear which global change drivers most increase disease and under what contexts. Here we amassed a dataset from the literature that contains 2,938 observations of infectious disease responses to global change drivers across 1,497 host–parasite combinations, including plant, animal and human hosts. We found that biodiversity loss, chemical pollution, climate change and introduced species are associated with increases in disease-related end points or harm, whereas urbanization is associated with decreases in disease end points. Natural biodiversity gradients, deforestation and forest fragmentation are comparatively unimportant or idiosyncratic as drivers of disease. Overall, these results are consistent across human and non-human diseases. Nevertheless, context-dependent effects of the global change drivers on disease were found to be common. The findings uncovered by this meta-analysis should help target disease management and surveillance efforts towards global change drivers that increase disease. Specifically, reducing greenhouse gas emissions, managing ecosystem health, and preventing biological invasions and biodiversity loss could help to reduce the burden of plant, animal and human diseases, especially when coupled with improvements to social and economic determinants of health.

Emerging infectious diseases are on the rise, often originate from wildlife, and are significantly correlated with socioeconomic, environmental and ecological factors[1]. As a consequence, there is concern that anthropogenic global change is contributing to alterations in disease risk. For example, several studies have demonstrated that infectious disease risk is modified by changes to biodiversity[2–6], climate change[7–11] and chemical pollution[12–14]. Landscape transformations, such as forest conversion to agriculture or urban centres, also regularly shift disease risk[15–20]. Moreover, the movement of people, products and animals around the planet has resulted in pathogen introductions with massive health consequences for humans, domesticated plants and animals, and wildlife[21]. Mechanistically, global change can alter disease by affecting the distribution of epidemiological traits in ecological communities, modulating immune defences, and altering contact rates among pathogens, wildlife, livestock and humans. For example, the COVID-19 pandemic, which reshaped the global economic and public health landscape, has been linked to animal trade and global travel, and researchers have speculated that there are associations with urbanization, climate change, air pollution and habitat loss[22]. This pandemic has also undoubtedly heightened interest in understanding causes of disease outbreaks and investment in infectious disease control, mitigation and surveillance.

Although there are many individual studies on infectious disease risk and environmental change, as well as syntheses on how some drivers of ecosystem change affect infectious diseases[1–21], formal meta-analyses are lacking examining how infectious diseases of plants, animals and humans are modified across global change drivers[23]. This literature gap is critical to fill because resources for infectious disease management will always be limited and could be poorly targeted without knowledge of which global change drivers most affect infectious disease risk. Moreover, risk might be high for only certain types of pathogens or hosts, for wildlife but not human diseases, or for certain ecological

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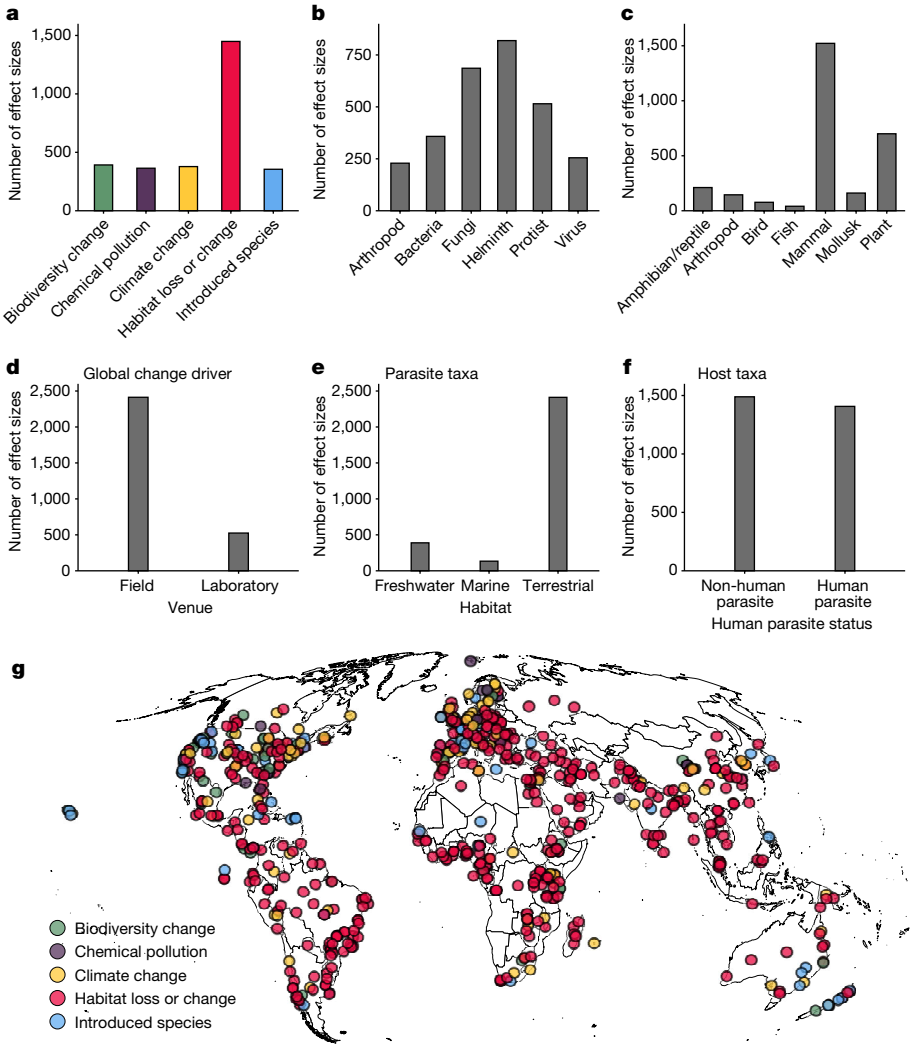

Fig. 1 | The number of observations across ecological contexts. a–f, Summary of the number of observations (that is, effect sizes) in the infectious disease database across the following ecological contexts: global change driver (**a**), parasite taxa (**b**), host taxa (**c**), experimental venue (**d**), habitat of the study (**e**) and human parasite status (**f**). **g**, The locations of field studies show broad global coverage of studies included in the database. See Extended Data Fig. 2 for the number of parasite taxa as well as the number of parasite taxa in the database partitioned by ecto- and endoparasites, ecto- and endothermic hosts, vectors and non-vectors, vector-borne and non-vector-borne parasites, complex and direct transmission parasites, parasites with and without free-living stages, parasites that do and do not infect humans, microparasites and macroparasites, and zoonotic and non-zoonotic parasites. The numbers of effect sizes and studies across all of these end points are shown in Supplementary Table 2. The base map is from Natural Earth (https://www.naturalearthdata.com/).

conditions. For example, the emergence of zoonotic diseases of humans tends to be driven more by interactions with particular mammalian and avian taxa than other vertebrate groups[24–26]. Thus, understanding these context dependencies will further enhance the efficacious use of limited resources for disease control.

Here our primary goal is to use a traditional meta-analytical approach to determine the magnitude with which global change drivers are associated with infectious disease risk and whether these associations depend on ecological contexts, such as host or parasite/pathogen (hereafter referred to as parasite, which refers to all infectious agents including bacteria and viruses) taxon or human versus non-human disease. To accomplish these goals, we conducted a literature search to identify studies on infectious disease that considered at least one of the five major drivers of global change highlighted by the Millennium Ecosystem Assessment[27]: biodiversity change, climate change, chemical pollution, habitat loss/change (defined as anthropogenic destruction of an ecosystem or the shift in habitat from one type to another; for example, slash and burn, clearcutting, urban-to-rural gradient) or introduced species (Methods).

Database of infectious disease studies

The database resulting from our literature search includes 972 studies and 2,938 observations of global change drivers on disease or parasitism from 1,006 parasite taxa, 480 host taxa and 1,497 host–parasite taxa combinations (Fig. 1 and Extended Data Fig. 1). Each continent except for Antarctica was well represented with data from field studies across the global change drivers (Fig. 1g). In contrast to many meta-analyses, we had reasonable coverage of studies within low- and middle-income countries (LMICs; that is, more than 20 field studies in the LMICs per driver), except for chemical pollution and introduced species (6 and 3 field studies in the LMICs, respectively; Fig. 1g). Nevertheless, there were still less data available for LMICs, highlighting the need for additional research in these countries.

Each observation in the database contains information on the associated global change driver and host and parasite taxa and traits (for example, human versus non-human parasite), and whether it was derived from freshwater, marine, or terrestrial systems and laboratory or field studies (Fig. 1, Extended Data Figs. 2 and 3 and Supplementary

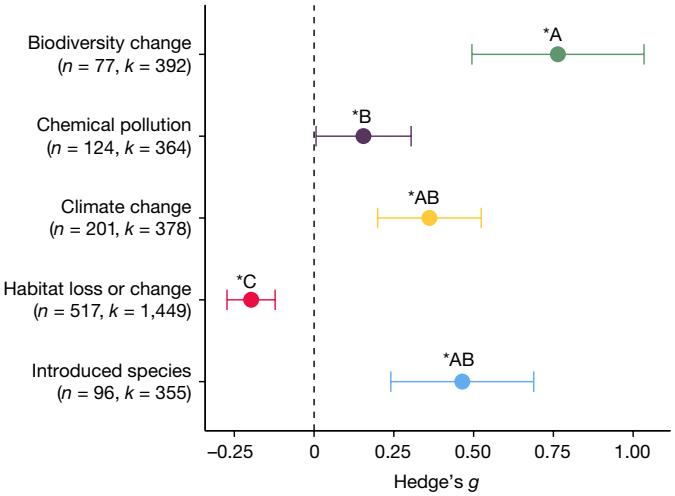

Fig. 2 | The effects of five common global change drivers on infectious disease responses. Biodiversity change (BC), climate change (CC), chemical pollution (CP) and introduced species (IS) are associated with increases in disease-related end points or harm (that is, introduced species having fewer parasites). Habitat loss or change (HLC) was associated with significant decreases in disease end points. The numbers of studies (n) and effect sizes (k) for each driver are shown in parentheses. The displayed points represent the mean predicted values (with 95% confidence intervals) from a meta-analytical model with separate random intercepts for study. Global change driver effects are significant when confidence intervals do not overlap with zero and were explicitly tested using a two-tailed one sample t-test (indicated by asterisks; $t_{50,45} = 5.56$, $P < 0.001$ for BC; $t_{87,75} = 2.04$, $P = 0.044$ for CP; $t_{144,46} = 4.36$, $P < 0.001$ for CC; $t_{327,51} = -5.13$, $P < 0.001$ for HLC; $t_{61,3} = 4.07$, $P < 0.001$ for IS). Points that do not share letters are significantly different from one another ($P < 0.05$), as determined using a two-sided Tukey's post hoc multiple-comparison test with correction for multiple comparisons. Pairwise comparison results are shown in Supplementary Table 3. Sampling variance = 0.14%; within-study heterogeneity $I^2 = 89.27\%$; and between-study heterogeneity $I^2 = 10.59\%$.

Table 2). Moreover, each response variable was classified as a host end point, which captures host symptoms or consequences of infection (disease presence, disease severity, survival, growth and reproduction) or a parasite end point, which captures parasite pressure in hosts (parasite prevalence, incidence, intensity, abundance, survival, growth and richness). Hedge's g and log response ratio effect sizes were calculated from each study, with positive and negative effect sizes representing increases and decreases in disease, respectively. The exception was for studies on introduced species, for which decreases and increases in parasites or disease in the native host received negative and positive values, respectively, but the opposite was true for non-native hosts because a reduction in disease in non-native hosts and an increase in native hosts were both deemed to be potentially detrimental (further discussion is provided in the Methods and Supplementary Information 1).

Comparing among global change drivers

Among the global change drivers, habitat loss/change caused significant reductions in disease, while chemical pollution, climate change, introduced species and biodiversity change increased disease responses or disease-related harm, in order of increasing magnitude. These patterns were similar using Hedge's g and log response ratios (Figs. 2 and 3 and Extended Data Fig. 4) and we therefore focus on Hedge's g hereafter. Biodiversity change was associated with a 393% greater increase in disease compared with chemical pollution, a 111% greater increase in disease compared with climate change and a 65% greater increase in disease compared with introduced species (Fig. 2). Importantly, we found no evidence that effect-size patterns among global change drivers could be explained by differences in variances or sample sizes among global change drivers (Extended Data Fig. 5), extreme values (Extended Data Fig. 6a), or publication (Extended Data Fig. 6b–d) or time-lag (Extended Data Fig. 6, Supplementary Data 1 and Supplementary Information 2) biases.

Comparing among subcategories of drivers

Next, we evaluated global change driver subcategories (Fig. 3). Consistent with previous studies[3], the loss of pre-existing biodiversity was associated with significantly greater increases in infectious disease outcomes (857% more) compared with natural biodiversity gradients (for example, latitudinal or elevational gradients in species richness; Fig. 3). Enemy release (that is, the notion that introduced species leave many of their parasites behind in their native range) reduced infectious diseases in introduced species, but had weaker effects compared with biodiversity loss (39% weaker). Mean temperature and carbon dioxide similarly increased disease but had weaker effects compared with biodiversity loss (55% and 62% weaker, respectively) and enemy release (26% and 38% weaker, respectively). Urbanization decreased infectious diseases, perhaps because urban development is associated with improved water, sanitation and hygiene for humans, and habitat loss for many parasites and their non-human hosts[18]. Specifically, helminths, protists and arthropods were all negatively associated with urbanization, whereas viruses were non-significantly positively associated with urbanization (Extended Data Fig. 7a). Furthermore, disease was reduced in urban settings compared with in rural and peri-urban settings, whereas there were no differences in disease along urbanization gradients or between urban and natural settings (Extended Data Fig. 7b). Similarly, the effect of forest fragmentation on disease depended on the type of fragmentation being compared, but the effect of deforestation on disease did not depend on the type of land-use conversion (Extended Data Fig. 7c). All of the other subcategories had non-significant effects on disease (Fig. 3). Given the limited funds for infectious disease management, these results suggest that controlling or mitigating biodiversity loss, introduced species and climate change might be particularly important for infectious disease control.

Context dependencies

Understanding context dependencies is also crucial for properly targeting limited resources for disease control. Although the ideal approach would have been to compare global change drivers in a single model-selection analysis that considered the correlations among all independent variables and their interactions, this approach was not possible due to missing combinations of variables (Methods and Supplementary Table 2). We circumvented these statistical limitations using two approaches. First, we tested for two-way interactions between each global change driver and host and parasite taxa and various traits of hosts, parasites and studies. Second, to account for the covariances among predictors and to identify the most parsimonious combinations of predictors, we fit models with all possible combinations of main effects of host, parasite and study factors for each global change driver separately (Methods and Supplementary Data 1). We then qualitatively assessed the consistency in the results between these statistical approaches.

Importantly, these analyses can reveal both when there are and are not context dependencies. For example, there were many consistent patterns across global change drivers. For several global change drivers, parasite versus host end point was an important moderator in either the two-way interaction (Fig. 4a) or model selection (Fig. 4b) analyses and, in each case, parasite end points were as sensitive or more sensitive to the global change drivers compared with host end points (Extended Data Fig. 8a and Supplementary Table 3). Given that parasite

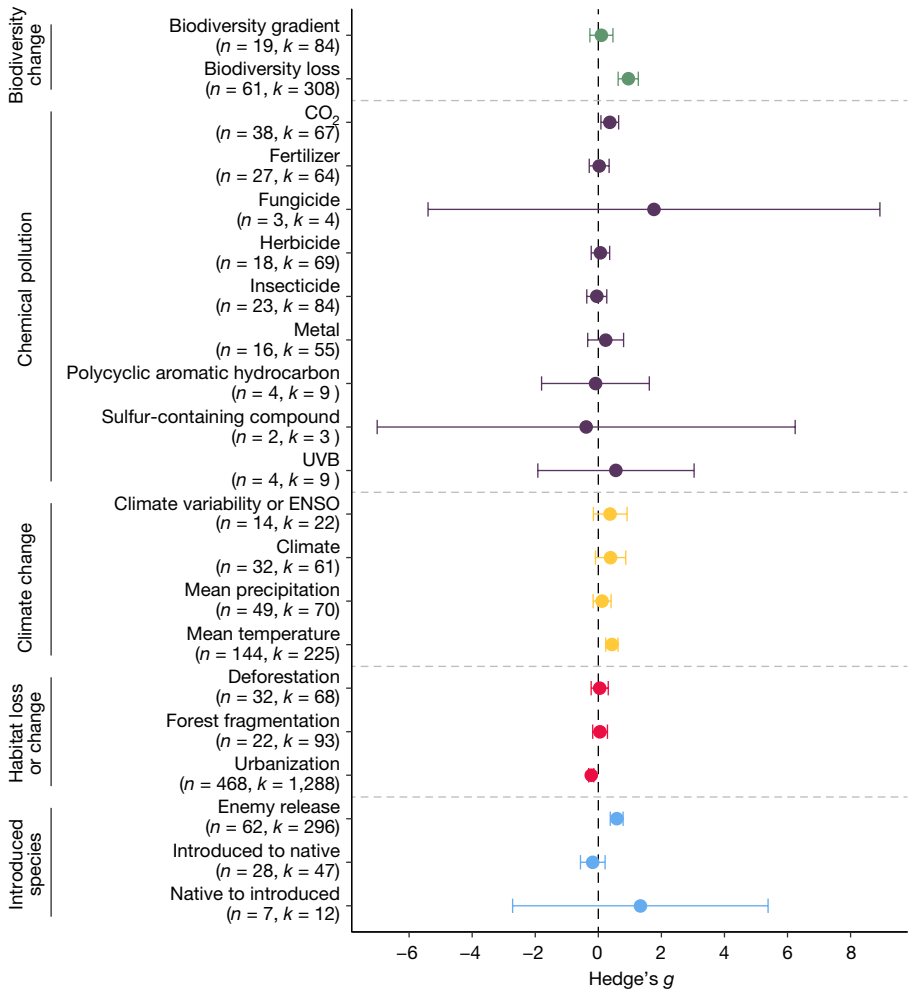

Fig. 3 | The effects of subcategories within five common global change drivers on mean infectious disease responses in the literature. Biodiversity gradient covers natural variation in biodiversity (for example, across latitude or elevation), whereas biodiversity loss is a loss of biodiversity usually associated with an anthropogenic factor[3]. Forest fragmentation compares different sizes of forest patches, whereas deforestation compares forests to the absence of forests (that is, two different habitats). Enemy release is defined as cases in which an introduced host has fewer parasites in its introduced range than native range or than native species in its introduced range[21,30,36]. Native-to-introduced transmission occurs when an introduced host is a competent host for a native parasite and amplifies infections in the native host[21]. Introduced-to-native transmission is defined as the spread of a parasite from an introduced to native host or from a native to introduced host[21]. For zoonotic diseases, spillover is animal-to-human and human-to-animal transmission. The numbers of studies (n) and effect sizes (k) of each subcategory are shown in parentheses. The displayed points represent the mean predicted values (with 95% confidence intervals) from a meta-analytical model with study as a random intercept. Confidence intervals that do not overlap with zero are generally significant ($P < 0.05$), see the main text for details. UVB, ultraviolet radiation B; ENSO, El Niño-Southern Oscillation. Sampling variance = 0.14%; within-study heterogeneity $I^2 = 89.27\%$; and between-study heterogeneity $I^2 = 10.59\%$.

abundance can change profoundly without changes in symptoms or disease, especially for hosts that have tolerance (that is, ameliorating the damage that infection causes) rather than resistance (that is, 'fighting' the parasite directly) defence strategies[28,29], it is unsurprising that parasite end points, which are capturing parasite abundance, are more sensitive to global change factors than host end points, which are capturing host symptoms or consequences of infection.

The effects of global change drivers on infectious disease outcomes also did not consistently depend on continent (Extended Data Fig. 9a and Supplementary Table 3), host taxon (Extended Data Fig. 9b and Supplementary Table 3) or whether the parasite infected humans or not (Fig. 4a). These results indicate that global change drivers are having consistent effects on infectious disease risk across space and broad host taxa, including humans, non-human animals and plants. Parasites of mollusks were the exception because they responded more positively to biodiversity change and habitat loss/change compared with other host taxa, most likely because mollusks are required hosts for all trematodes, which have complex life cycles, and theory and evidence indicate that parasites with complex life cycles tend to be more sensitive to biodiversity change and habitat loss/change compared with those with simple life cycles[2]. Although global change drivers did not differentially affect zoonotic versus non-zoonotic parasites (Fig. 4a), end points associated with zoonotic parasites measured from wild or domesticated animals responded more positively to global change drivers compared with end points associated with zoonotic parasites measured from humans, and this effect was generalizable across global change drivers (Extended Data Fig. 10a). Furthermore, although global change drivers generally did not differentially affect host taxa (Extended Data Fig. 8b), end points associated with wild animals responded more positively to global change drivers compared with end points measured from domesticated animals, and this effect was similarly generalizable across global change drivers (Extended Data Fig. 10a). These results are not surprising given that humans treat and control diseases in humans and domesticated animals more so than diseases in wild animals, which should dampen the effect of global change drivers on

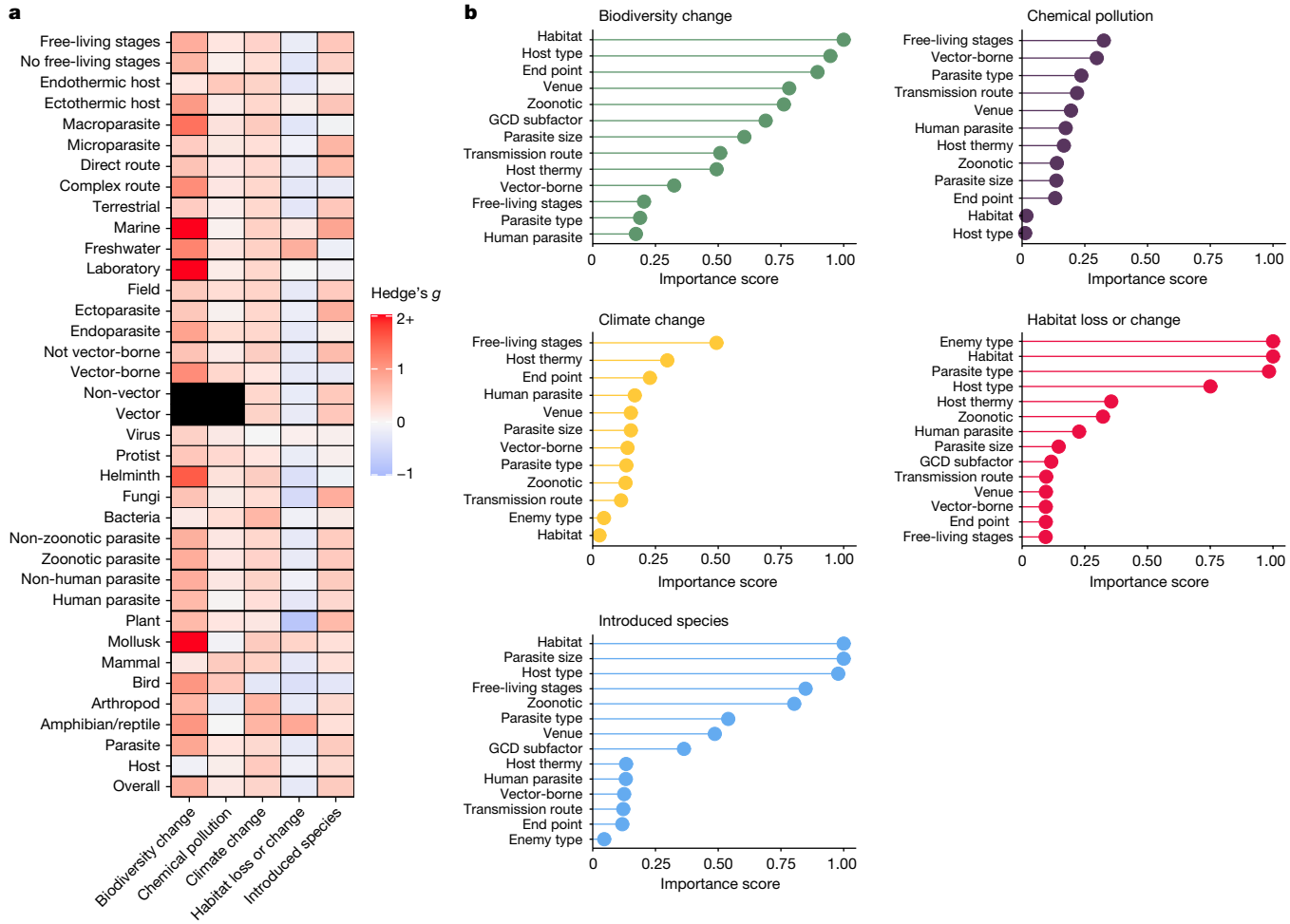

Fig. 4 | Context-dependent effects of global change drivers on infectious disease responses. a, Coefficients from separate tests of two-way interactions between each global change driver and host and parasite taxa and various traits of hosts, parasites and studies. The black sections of the heat map could not be tested owing to missing data. Here, human parasites are those that can infect humans, whereas non-human parasites are those that are not reported to infect humans. Zoonotic parasites are those that are spread between humans and animals, while non-zoonotic parasites are those that are not known to spread between humans and animals. **b**, The relative importance scores from model selection examining the effects of five common global change drivers on mean infectious disease responses. In contrast to the two-way interaction analyses, the model selection analyses account for the covariances among predictors and identify the most parsimonious combinations of predictors. The coefficients from these models are shown in Supplementary Data 1. In **b**, variable definitions are end point: host or parasite; free-living stage: free-living stage or not; global change driver (GCD) subfactor (Fig. 3); habitat: freshwater, marine, terrestrial; host taxa (Fig. 1); host thermy: ectotherm or endotherm; human parasite: human parasite or not; parasite size: macroparasite or microparasite; parasite taxa (Fig. 1); transmission route: complex or direct; vector-borne: vector-borne or not; venue: laboratory or field; ectoparasite: ectoparasite or endoparasite.

human and domesticated animal end points. The fact that many global change drivers increase zoonotic parasites in non-human animals and increase all parasites in wild animals suggests that anthropogenic change might increase the occurrence of parasite spillover from animals to humans and, therefore, also pandemic risk.

No clear context dependencies emerged from studies on chemical pollution and climate change (no significant two-way interactions and all relative importance scores < 0.5; Fig. 4b). This result was probably due to the enormous diversity in the pollutants tested, making it challenging to uncover consistent patterns on infectious disease and highlighting the need for further infectious disease research on this global change driver, especially given that many contaminants can be immunosuppressive[13]. The lack of context dependencies for climate change suggests that disease increases in response to climate change will be consistent and widespread, further stressing the need for reductions in greenhouse gas emissions to mitigate these detrimental impacts of climate change. This result is in contrast to several studies[8] suggesting that parasites with complex life cycles will be disrupted by climate change more than those with direct life cycles because they have more necessary host species that could be adversely affected by climate change.

In contrast to the generally consistent patterns across host taxa and certain global change drivers, numerous context dependencies were detected across parasite taxa and study system for other global change drivers. For example, when compared to viruses, fungi responded more positively to introduced species, and helminths responded more negatively to habitat loss/change (Extended Data Fig. 9c and Supplementary Table 3). Helminths, which are macroparasites that tend to have complex life cycles, also responded more positively to biodiversity loss compared with all other parasite taxa (Extended Data Fig. 9c and Supplementary Table 3). Similarly, relative to parasites with simple (that is, direct) life cycles, parasites with complex life cycles, such as vector-borne parasites, experienced greater decreases when exposed to introduced species and greater increases when exposed to biodiversity loss—results that were generally similar across the two-way interaction and model selection analyses (Fig. 4, Extended Data Fig. 8b,c and Supplementary Table 3). As parasites with complex life cycles require more host species than those with simple life cycles, there is a greater chance

that one of the hosts is sensitive to global change; it is therefore not surprising that they tend to be more sensitive to biodiversity loss compared with species with direct life cycles. Moreover, when non-native species are introduced to ecosystems, parasites with direct life cycles need to find only a single suitable host species (introduced host or novel host), whereas hosts with complex life cycles would need to find new intermediate and final hosts; thus, parasites with direct life cycles might increase more during host species introductions compared with those with complex life cycles[30]. Finally, the biodiversity change results are consistent with a meta-analysis highlighting that biodiversity loss increases parasites more if they have complex than simple life cycles[2]. Biodiversity loss also increased disease caused by macroparasites more than disease caused by microparasites (Extended Data Fig. 8d and Supplementary Table 3). Biodiversity loss also increased disease more in laboratory studies compared with field studies (Extended Data Fig. 8e and Supplementary Table 3), in aquatic systems compared with terrestrial systems (Extended Data Fig. 8f and Supplementary Table 3) and in ectothermic compared with endothermic hosts (Extended Data Fig. 8g and Supplementary Table 3). Conversely, habitat loss/change decreased disease caused by macroparasites more than disease caused by microparasites (Extended Data Fig. 8d and Supplementary Table 3). Habitat loss or change also decreased disease in field studies, but not in laboratory studies (Extended Data Fig. 8e and Supplementary Table 3); and in terrestrial systems, but increased disease in marine and freshwater systems (Extended Data Fig. 8f and Supplementary Table 3). Finally, ectoparasites increased more than endoparasites when exposed to introduced species (Extended Data Fig. 8h and Supplementary Table 3), which may be because ectoparasites are more vulnerable to host species loss (for example, fewer viable host species in the introduced range than in the native range) than endoparasites[31], therefore reducing ectoparasites in introduced hosts.

Caveats and conclusions

Here we revealed that biodiversity loss, climate change, chemical pollution and enemy release associated with introduced species increased disease responses or disease-related harm, whereas urbanization caused decreases in disease. All of these results were generally consistent across human and non-human diseases, although other context dependencies were common. End points from parasites with complex life cycles, such as macroparasites and vector-borne pathogens, decreased more with habitat loss/change, increased more with biodiversity change, and responded less strongly in response to introduced species compared with end points from parasites with simple life cycles, and ectoparasites increased more in response to introduced species compared with endoparasites.

We hope that our analyses will facilitate disease control, mitigation and surveillance efforts globally, ultimately improving wildlife and human health and pandemic preparedness; however, there are important caveats for using these analyses in decision-making. First, the relationships that we identified might not hold past the range of conditions included in this meta-analysis and we therefore advise against projecting beyond these conditions. Second, we treated the global change drivers in this meta-analysis in an unbiased and equal manner. However, from a policy perspective, the rates of change of the drivers and their relevance to current and future epidemic and pandemic risk are also crucial. For example, some subcategories of drivers, such as ultraviolet radiation associated with ozone depletion, require less attention because they have already been rectified by global agreements. Other drivers are expected to asymptote or even improve in certain parts of the world, such as habitat loss in upper-income countries as they pursue reforestation. Finally, some drivers are expected to worsen through time and are associated with increases in disease risk, such as climate change and biodiversity loss, and these drivers might therefore necessitate the greatest policy attention. The third caveat is that there are very few studies in this meta-analysis on interventions to remediate the effects of global change on disease. There is considerable evidence that simply reversing the magnitude of global change drivers can be insufficient to fully counteract their effects[32]. Consequently, we need more tests of interventions to remediate the highest priority drivers described herein[33] and efforts to evaluate whether ecosystem restoration can be used as a lever to manage disease[34].

Finally, most studies in this meta-analysis consider the effects of a single stressor or global change driver on infectious disease end points despite most organisms experiencing several of these factors concurrently and many drivers being interconnected. For example, climate change and chemical pollution can cause habitat loss and change, which in turn can cause biodiversity loss and facilitate species introductions. It is unclear whether global change drivers generally interact additively, antagonistically or synergistically and future studies should therefore more thoroughly examine their interactions, interdependencies and relative contributions to disease risk. Importantly, greater effort is needed to identify win–win solutions that address multiple societal stressors, such as disease, food, energy, water, sustainability and poverty challenges[33,35]. Although our data suggest that climate change and biological invasions and loss have a part in wildlife and human diseases, all of these factors also can contribute to, exacerbate and trap people in rural poverty, which is the strongest predictor of environmentally transmitted infectious diseases on the planet[20]. Thus, leveraging the intersection among environmental, social, economic and political dimensions will not only be necessary to effectively mitigate against increases in disease associated with global change, but will also almost certainly be required to meet the United Nation's sustainable development goals targeted at managing the numerous co-dependent global grand challenges of the twenty-first century[20,33].

Online content

Any methods, additional references, Nature Portfolio reporting summaries, source data, extended data, supplementary information, acknowledgements, peer review information; details of author contributions and competing interests; and statements of data and code availability are available at https://doi.org/10.1038/s41586-024-07380-6.

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Methods

We conducted literature searches in Web of Science, Scopus and PubMed on each of the five global change drivers and infectious disease (the dates of searches and search terms are shown in Supplementary Table 1). We translated papers in the following languages: Chinese, French, Japanese, Polish, Portuguese, Russian and Spanish. Only original peer reviewed literature was included. Book chapters, conference proceedings, grey literature and review articles were excluded. We screened papers to determine whether they drew clear conclusions about the impact of the global change driver on a parasite (for example, parasite growth, prevalence, abundance, intensity, virulence) or host end point (for example, disease, growth rate, survival) through experiments or field studies (Supplementary Information). Each of the five global change drivers was further categorized into subcategories, which are provided in Fig. 3. From each study, we extracted data on the effect of the global change driver on each infectious disease end point, the subcategory of global change driver, the host and parasite species, and various traits of the study, hosts and parasites. We hand-corrected all obvious misspellings of parasite and host names and converted those parasites with multiple hosts to a broader taxonomic resolution (for example, hosts of domestic dogs and humans were converted to 'mammals'). Moreover, to correct any changes in taxonomy and any other non-obvious misspellings, we used the taxize R package (v.0.9.100)[37] to match parasite and host names to those in 20 existing databases using the Global Names Resolver service provided by the Encyclopedia of Life. Instances of misspellings were clarified, and current taxonomic nomenclature was applied when appropriate.

The list of studies associated with biodiversity change was based on a previous study[3], which combined studies from four meta-analyses (details are provided in Supplementary Table 1 and ref. 3). Each meta-analysis included only studies that reported a measure of host biodiversity as the independent variable (for example, host richness, Shannon diversity, Simpson diversity). Although our meta-analysis focuses on anthropogenic change, to remain consistent with ref. 3, we included both natural and anthropogenically driven biodiversity gradients. With the exception of natural biodiversity gradients, all of the other studies included in this meta-analysis had an anthropogenic driver. For chemical pollution, contaminants were assigned to 1 of 11 contaminant classes described previously[38], and we excluded papers that evaluated the development of a treatment for a parasitic infection or evaluated how naturally occurring nutrients influence disease development. Studies on introduced species focused on enemy release, transmission of parasites from native to invasive or invasive to native hosts, dilution effects and scenarios in which an introduced host is a competent host for a native parasite and amplifies infections in the native host (definitions of these terms are shown in Fig. 3). The initial study list and related information were then compiled.

Data extraction and effect sizes

We extracted mean values with associated sample sizes and dispersion (such as variance, s.d., s.e.m.). Data extraction was then performed and data were checked for accuracy. All data in biodiversity change were taken from ref. 3. Data presented in the text or tables were directly extracted. Data from figures were digitized using WebPlotDigitizer[39]. When available, raw data were used to calculate the mean, dispersion and sample size. When studies presented statistics other than mean values (such as odds ratio; regression coefficient; correlation coefficient; and t, z, χ^2 and f statistics), we extracted these values and their subsequent dispersion in place of mean values. Moreover, for data for which the disease end point was measured through time, a natural cubic spline relationship was generated between time and disease end point for each treatment and the area under the curve (AUC) was then calculated for each natural cubic spline. The resulting AUC and associated error were used for the mean and dispersion to calculate an effect size.

We defined our effect size using Hedge's g, assuming heteroscedastic population variances among the two groups (SMDH):

$$g = \frac{\bar{y}_1 - \bar{y}_2}{s_p} \quad (1)$$

where $\bar{y}_1$, $\bar{y}_2$ and s_p are the mean of sample 1, the mean of sample 2 and the pooled s.d., respectively. s_p is calculated as follows:

$$s_p = \sqrt{\frac{s_1^2 + s_2^2}{2}} \quad (2)$$

where s_1 is the s.d. for sample 1 and s_2 is the s.d. for sample 2. When observations were statistics rather than mean values, we converted the presented statistic to Hedge's g using standard conversion equations within the esc R package (v.0.5.1)[40,41].

We also calculated effect sizes using the log response ratio (RR):

$$\mathrm{RR} = \ln\frac{\bar{y}_1}{\bar{y}_2} \quad (3)$$

where $\bar{y}_1$ and $\bar{y}_2$ are the mean of sample 1 and sample 2, respectively. The variance of the log response ratio is:

$$\mathrm{var}(\mathrm{RR}) = \frac{s_1^2}{N_1\bar{y}_1^2} + \frac{s_2^2}{N_2\bar{y}_2^2} \quad (4)$$

where $\bar{y}_1$, s_1 and N_1 are the mean, s.d. and sample size of sample 1, respectively; and $\bar{y}_2$, s_2 and N_2 are the mean, s.d. and sample size of sample 2, respectively. For observations that were statistics rather than mean values, conversion from the presented statistic to the RR were not possible; as such, sample sizes between the two effect sizes were not equal and all observations within BC did not have associated RR values.

Meta-analyses

All analyses were conducted in R (v.4.2.2)[42]. All analyses were conducted with meta-analytic multilevel mixed-effects models using the rma.mv function in the metafor R package (v.4.2-0)[43]. Our data had multiple effect sizes from the same studies, so all meta-analytic models were fit with a study-level and observation-level random effect, to account for the non-independence of observations from the same study, and with a robust variance estimator (that is, CL2 cluster-robust estimate of variance-covariance matrix as well as Satterthwaite approximation of d.f.)[44]. Test statistics and confidence intervals for fixed effects were computed using one-sample, two-tailed t-distributions. Post hoc comparisons were conducted using two-tailed Tukey's tests with multiple-comparison adjustments. Moderators in the meta-analysis with many consistent effect sizes will result in estimates with small confidence intervals and moderators with few or inconsistent effect sizes will result in estimates with large confidence intervals. Statistical significance was assumed when 95% confidence intervals were not overlapping zero.

Moderator variables

We first estimated the overall grand mean and the total heterogeneity explained by the random effect terms. Second, to test for the effects of broad global change drivers on disease, we conducted a meta-analytical model with global change driver as the moderator. Third, to test whether global change driver subfactors differentially affect disease, we conducted a meta-analytical model with the subfactors of global change drivers as the moderator. Fourth, we sought to test for context dependencies of the effects of global change drivers on disease. The ideal approach would have been to compare global change drivers in a single model-selection analysis that considered the correlations among all independent variables and their interactions, but this approach

was not possible due to missing some combinations of variables. For example, all habitat loss/change studies were conducted in freshwater and terrestrial systems (that is, no laboratory or marine studies), also all fungi were ectoparasitic and some host taxa were too infrequently tested under certain global change drivers. We therefore conducted meta-analytical models with the main and interactive moderators of global change drivers and various host, parasite and study moderators. The various study moderators taken from each study included host taxa, parasite taxa, vector status (vector/non-vector), vector-borne status (vector-borne/non-vector-borne), parasite type (endo-/ectoparasite), human parasite (human/non-human), transmission route (complex/direct), free living stages (yes/no), macroparasite versus microparasite, host thermy (endo-/ectothermic), experimental venue (field/laboratory), response variable end point (host or parasite focused) and habitat (freshwater/marine/terrestrial); each of these moderators were tested separately. Then, to determine whether global change drivers may increase occurrence of spillover events and potential risk of pandemics, we assessed whether wild or domesticated animal end points within the animal diseases only and human or non-human end points within the zoonotic diseases only showed differential responses to global change drivers. Finally, to determine whether certain context dependencies existed within the habitat loss/change data, we assessed whether the effect of urbanization on disease varied by parasite taxon or land-use comparison (that is, urban land-use compared against rural, peri-urban, natural or along an urbanization gradient) and whether the effects of deforestation and forest fragmentation depended on the land-use conversion type (that is, clearcut and regrowth or agriculture for deforestation and patch-size gradient or large/continuous patch versus small patch for fragmentation). Differences in the main and interactive effects of these moderators was assessed using the emmeans R package (v.1.8.5)[45]. Finally, to determine which moderators best explain disease response to specific global change drivers, we performed model selection based on AICc in which we fit all possible combinations of the main effects of the global change subfactors and various host, parasite and study moderators using the dredge function in the MuMIn R package (v.1.47.5)[46]. Model selection was conducted on each global change driver separately; owing to the different numbers of observations across global change drivers and missing cell issues within global change drivers, the replicates varied for each global change (biodiversity gradient $k = 387$; climate change $k = 310$; habitat loss/change $k = 1{,}238$; introduced species $k = 309$; chemical pollution $k = 336$). Model weights and relative importance values for each predictor variable were calculated from models with a $\Delta\text{AICc} \leq 4$, which have moderate to substantial support to be the best model[47]. All data, R scripts and R markdown files are provided.

Publication bias and sensitivity analysis

Publication bias is the selective publishing of certain research findings, such as significant or favourable results. Common publication biases include small study effects (correlation between observed effects sizes and standard errors) and time-lag biases (positive results being published before negative results)[48]. To assess these potential biases, we first used funnel plots to visually inspect the relationship between model (intercept only) and standard error, but it is important to note that funnel plots assume minimal heterogeneity in data and should therefore be used as a visual tool only[49]. Second, we performed multilevel meta regressions using the inverse sample size or the square root of the effective sample size as moderators to clarify small study effects (Egger's test[49]). Third, we included the publication year as a moderator in this meta regression model to simultaneously test for a time-lag bias[46]. Fourth, to assess the robustness of our results, we performed a leave-one-out analysis on the meta-analytical grand mean (intercept only) model. From this analysis, we determined whether the removal of a single study greatly shifted the grand mean estimate[46]. Finally, we conduced fail-safe *N* analysis to address the file-drawer problem, we used the Rosenthal, Orwin and Rosenberg publication bias methods and set our fail-safe *N* threshold equal to $5N_{\text{study}} + 10$ such that, if the values from the methods are greater than our threshold value, then our results can be considered to be robust with respect to unpublished non-significant results[49].

Given that effect size is a function of variance and sample size, differences in the distributions of effect sizes on disease end points among global change drivers and across contexts might be the product of these factors[50]. To test whether differences in effect sizes were driven by differences in sample sizes and/or variances, we tested for differences in sample sizes and variances among global change drivers. We applied generalized linear mixed effects models (GLMMs; glmer function, lme4 package, v.1.1-32)[51] with 'study' as a random intercept to compare variances and samples sizes among global change drivers, using Gaussian errors for variance models and Poisson errors for sample size models.

Reporting summary

Further information on research design is available in the Nature Portfolio Reporting Summary linked to this article.

Data availability

All the data for this Article have been deposited at Zenodo (https://doi.org/10.5281/zenodo.8169979)[52] and GitHub (https://github.com/mahonmb/GCDofDisease)[53].

Code availability

All the code for this Article has been deposited at Zenodo (https://doi.org/10.5281/zenodo.8169979)[52] and GitHub (https://github.com/mahonmb/GCDofDisease)[53]. R markdown is provided in Supplementary Data 1.

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Author contributions J.R.R. conceptualized the study. All of the authors contributed to the methodology. All of the authors contributed to investigation. Visualization was performed by M.B.M. The initial study list and related information were compiled by D.J.C., J.M.C., F.W.H., S.A.K., S.L.R. and J.R.R. Data extraction was performed by M.B.M., A.S., O.A.A., C.B., E.B., H.B., L.A.d.W., M.F., P.H., A.K., J.G.L., E.S., A.S. and N.V. Data were checked for accuracy by M.B.M. and A.S. Analyses were performed by M.B.M. and J.R.R. Funding was acquired by D.J.C., J.R.R., S.A.K. and S.L.R. Project administration was done by J.R.R. J.R.R. supervised the study. J.R.R. and M.B.M. wrote the original draft. All of the authors reviewed and edited the manuscript. J.R.R. and M.B.M. responded to reviewers.

Competing interests The authors declare no competing interests.

Additional information

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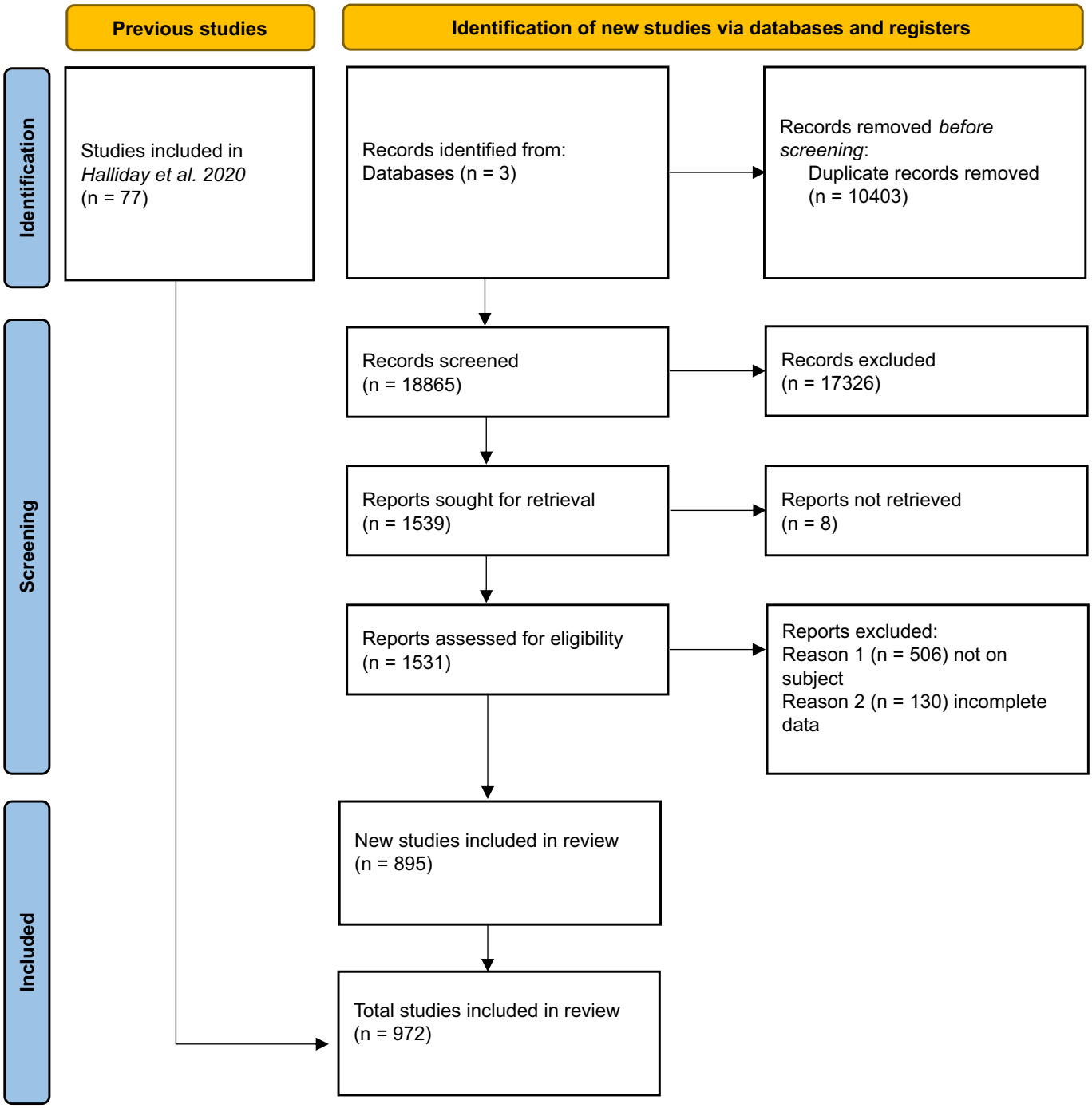
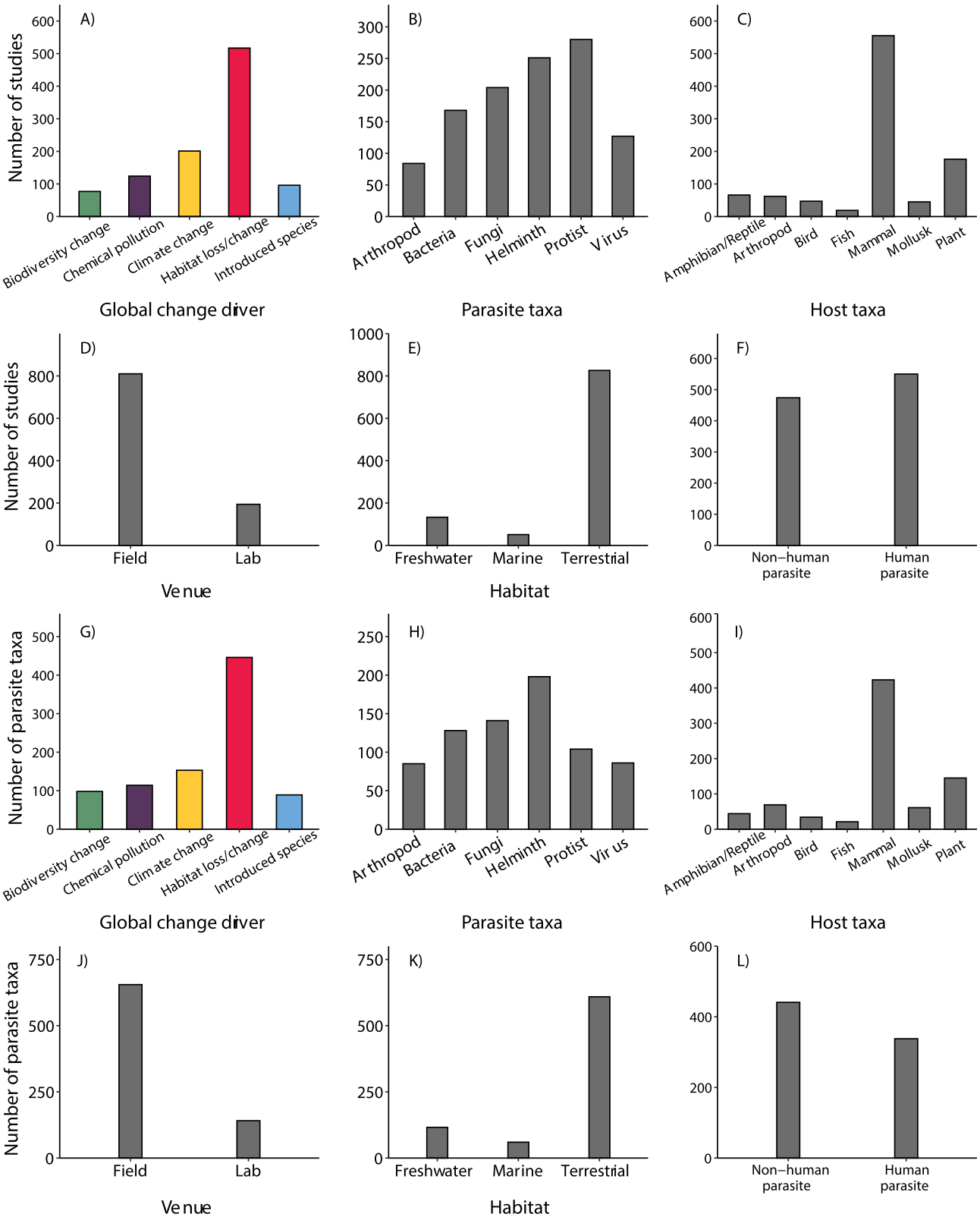
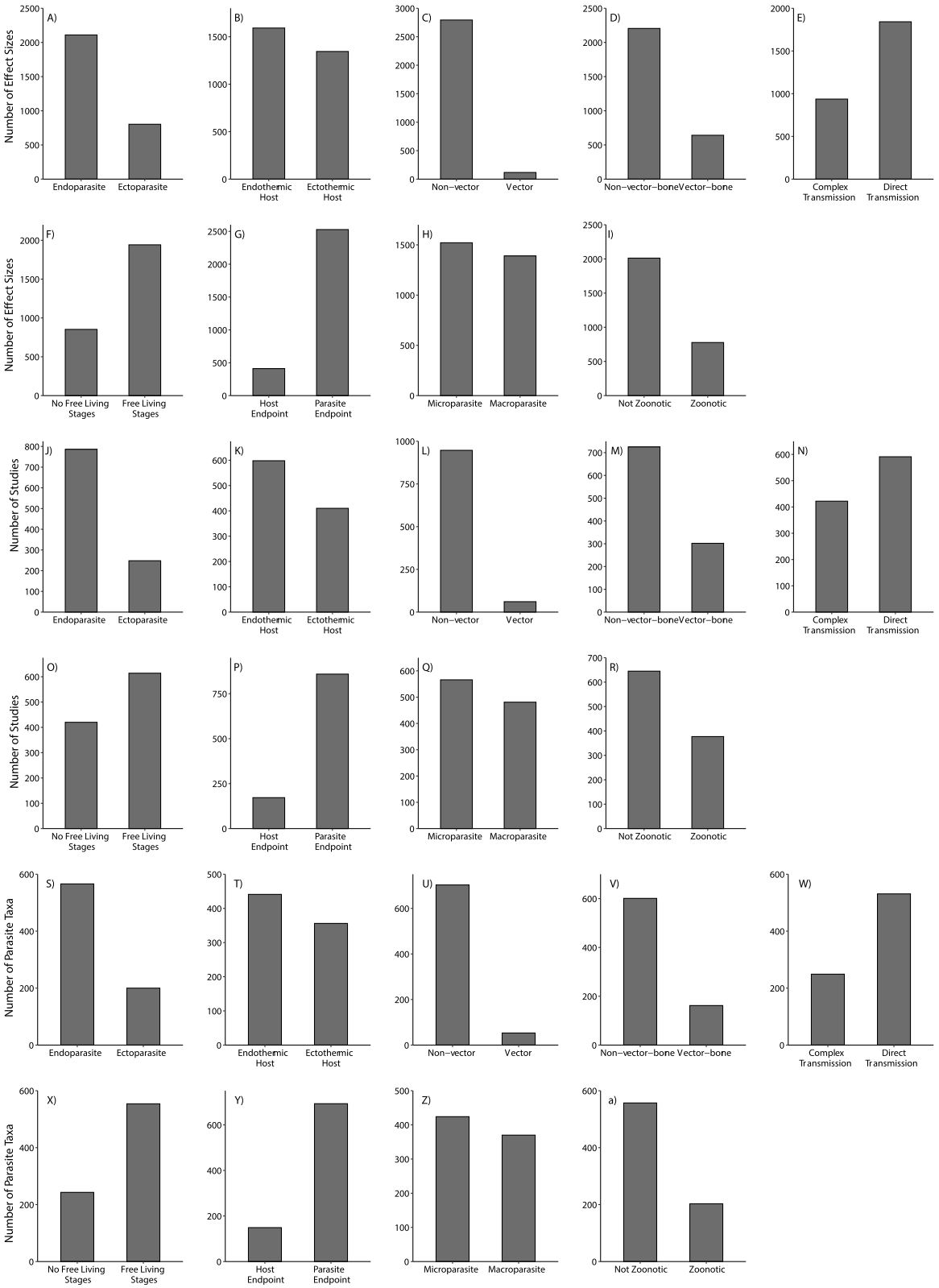
Peer review information *Nature* thanks Colin Carlson and the other, anonymous, reviewer(s) for their contribution to the peer review of this work. Peer reviewer reports are available.



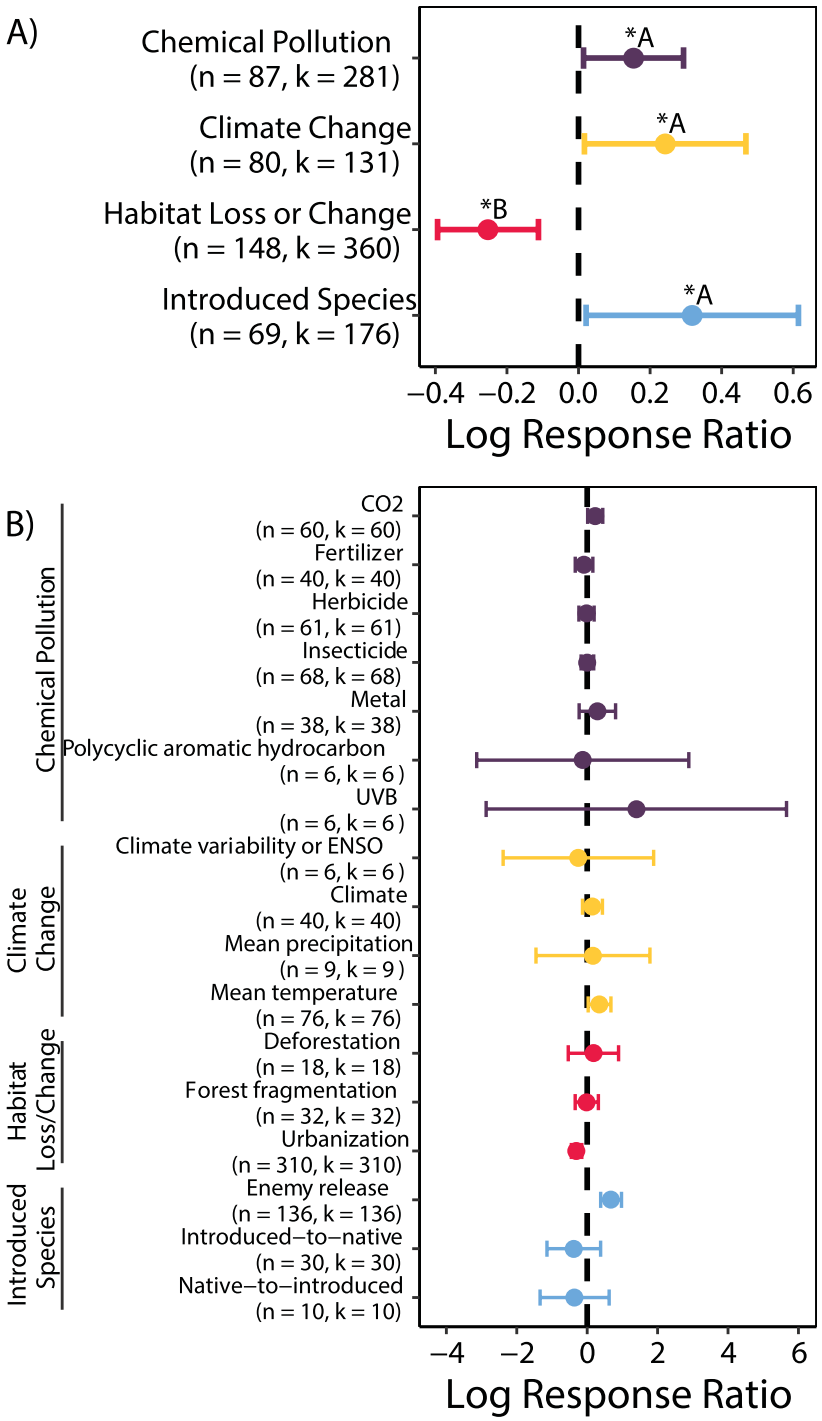
Extended Data Fig. 1 | PRISMA flowchart. The PRISMA flow diagram of the search and selection of studies included in this meta-analysis. Note that 77 studies came from the Halliday et al.[3] database on biodiversity change.



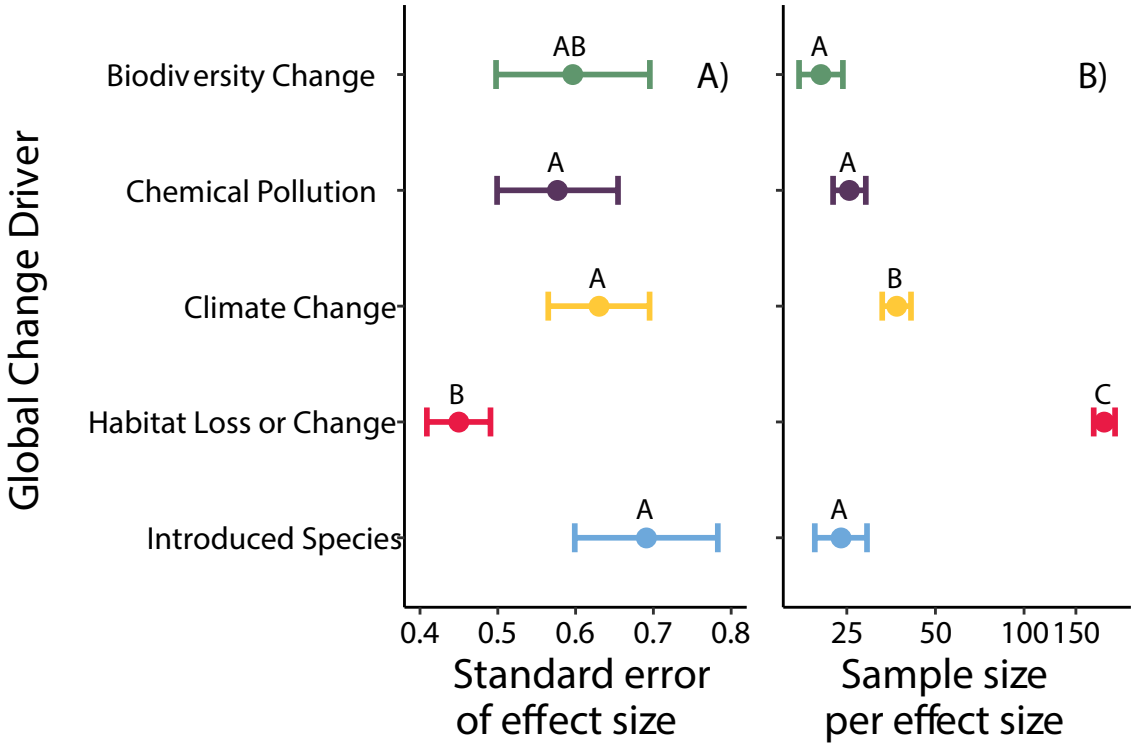
Extended Data Fig. 2 | Summary of the number of studies (A-F) and parasite taxa (G-L) in the infectious disease database across ecological contexts. The contexts are global change driver (**A**, **G**), parasite taxa (**B**, **H**), host taxa (**C**, **I**), experimental venue (**D**, **J**), study habitat (**E**, **K**), and human parasite status (**F**, **L**).



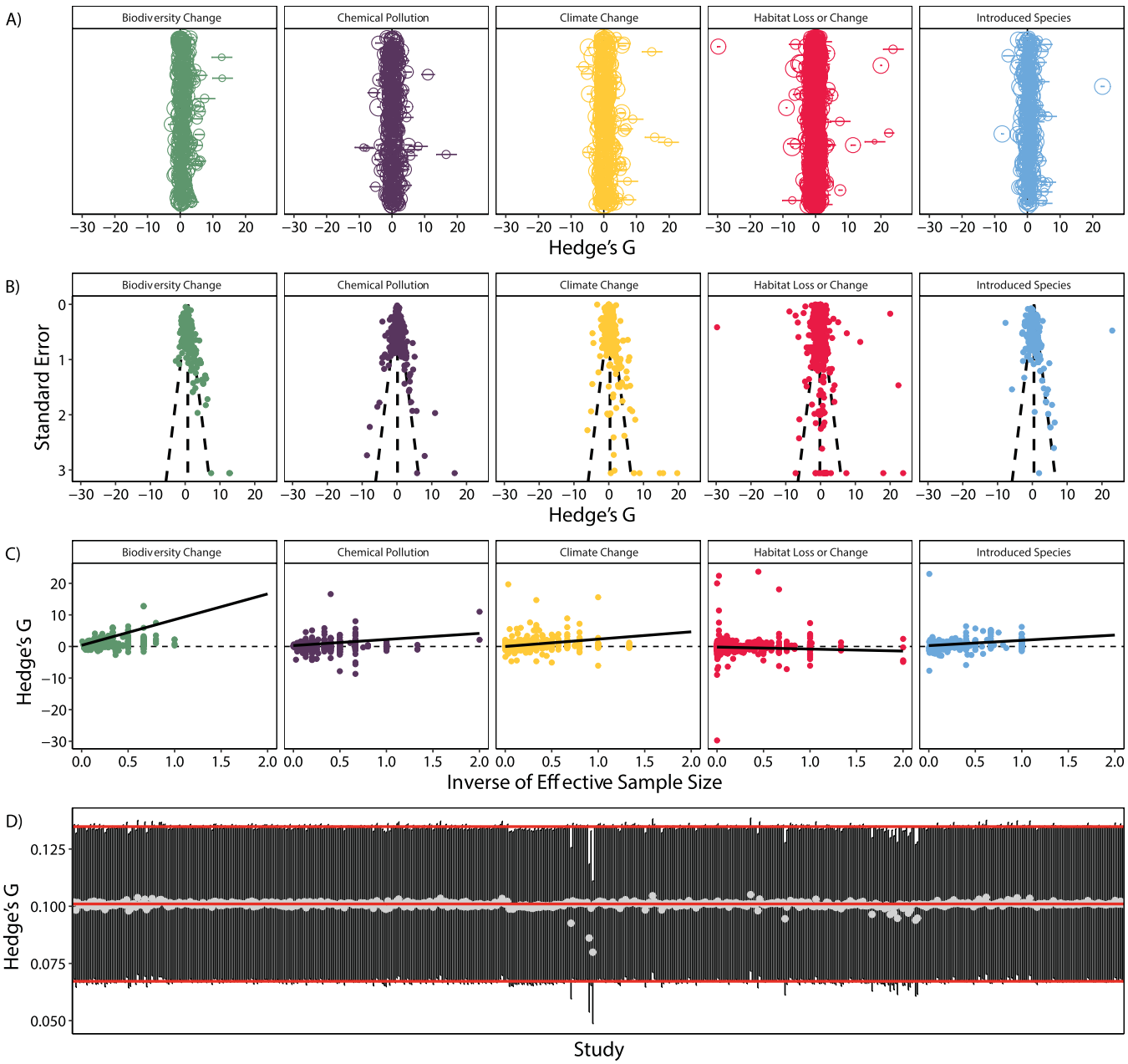
Extended Data Fig. 3 | Summary of the number of effect sizes (A-I), studies (J-R), and parasite taxa (S-a) in the infectious disease database for various parasite and host contexts. Shown are parasite type (**A**, **J**, **S**), host thermy (**B**, **K**, **T**), vector status (**C**, **L**, **U**), vector-borne status (**D**, **M**, **V**), parasite transmission (**E**, **N**, **W**), free living stages (**F**, **O**, **X**), host (e.g. disease, host growth, host survival) or parasite (e.g. parasite abundance, prevalence, fecundity) endpoint (**G**, **P**, **Y**), micro- vs macroparasite (**H**, **Q**, **Z**), and zoonotic status (**I**, **R**, **a**).



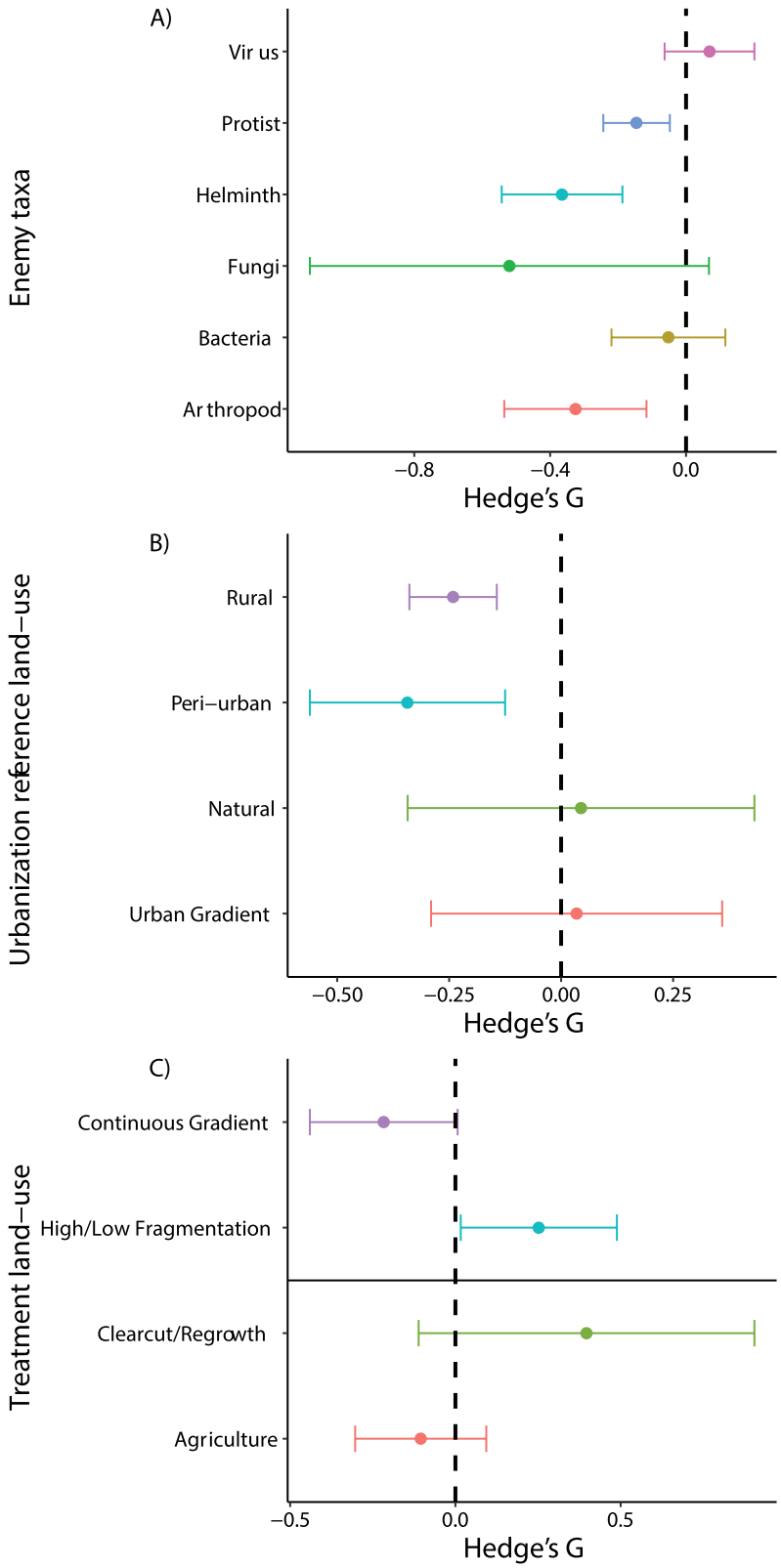
Extended Data Fig. 4 | The effects of global change drivers and subsequent subcategories on disease responses with Log Response Ratio instead of Hedge's g. Here, Log Response Ratio shows similar trends to that of Hedge's g presented in the main text. The displayed points represent the mean predicted values (with 95% confidence intervals) from a meta-analytical model with separate random intercepts for study. Points that do not share letters are significantly different from one another ($p < 0.05$) based on a two-sided Tukey's posthoc multiple comparison test with adjustment for multiple comparisons. See Table S3 for pairwise comparison results. Effects of the five common global change drivers (**A**) have the same directionality, similar magnitude, and significance as those presented in Fig. 2. Global change driver effects are significant when confidence intervals do not overlap with zero and explicitly tested with two-tailed t-test (indicated by asterisks; $t_{80.62} = 2.16$, $p = 0.034$ for CP; $t_{71.42} = 2.10$, $p = 0.039$ for CC; $t_{131.79} = -3.52$, $p < 0.001$ for HLC; $t_{61.9} = 2.10$, $p = 0.040$ for IS). The subcategories (**B**) also show similar patterns as those presented in Fig. 3. Subcategories are significant when confidence intervals do not overlap with zero and were explicitly tested with two-tailed one sample t-test ($t_{30.52} = 2.17$, $p = 0.038$ for CO_2; $t_{40.03} = 4.64$, $p < 0.001$ for Enemy Release; $t_{47.45} = 2.18$, $p = 0.034$ for Mean Temperature; $t_{110.81} = -4.05$, $p < 0.001$ for Urbanization); all other subcategories have $p > 0.20$. Note that effect size and study numbers are lower here than in Figs. 3 and 4, because log response ratios cannot be calculated for studies that provide coefficients (e.g., odds ratio) rather than raw data; as such, all observations within BC did not have associated RR values. Despite strong differences in sample size, patterns are consistent across effect sizes, and therefore, we can be confident that the results presented in the main text are not biased because of effect size selection.



Extended Data Fig. 5 | Average standard errors of the effect sizes (A) and sample sizes per effect size (B) for each of the five global change drivers. The displayed points represent the mean predicted values (with 95% confidence intervals) from the generalized linear mixed effects models with separate random intercepts for study (Gaussian distribution for standard error model, **A**; Poisson distribution for sample size model, **B**). Points that do not share letters are significantly different from one another ($p < 0.05$) based on a two-sided Tukey's posthoc multiple comparison test with adjustment for multiple comparisons. Sample sizes (number of studies, n, and effect sizes, k) for each driver are as follows: n = 77, k = 392 for BC; n = 124, k = 364 for CP; n = 202, k = 380 for CC; n = 517, k = 1449 for HLC; n = 96, k = 355 for IS.

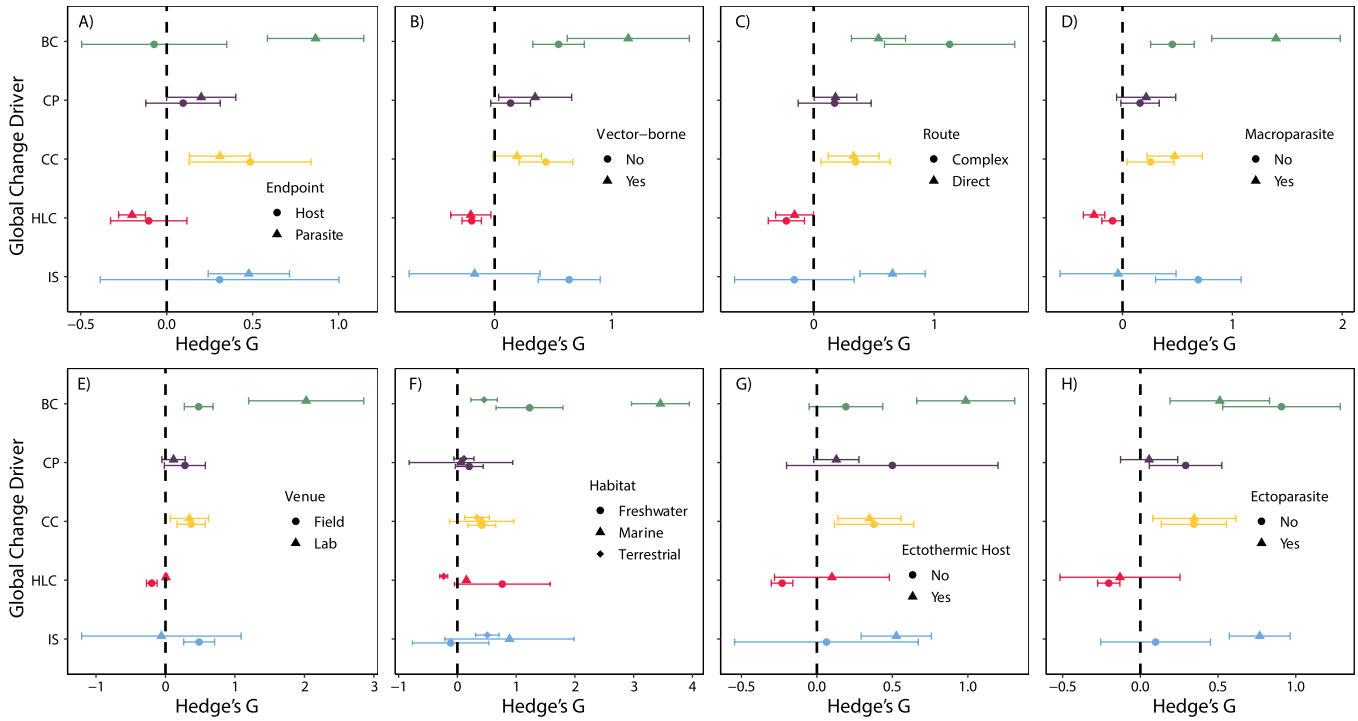


Extended Data Fig. 6 | Forest plots of effect sizes, associated variances, and relative weights (A), Funnel plots (B), and Egger's Test plots (C) for each of the five global change drivers and leave-one-out publication bias analyses (D). In panel **A**, points are the individual effect sizes (Hedge's G), error bars are standard errors of the effect size, and size of the points is the relative weight of the observation in the model, with larger points representing observations with higher weight in the model. Sample sizes are provided for each effect size in the meta-analytic database. Effect sizes were plotted in a random order. Egger's tests indicated significant asymmetries ($p < 0.05$) in Biodiversity Change (worst asymmetry – likely not bias, just real effect of positive relationship between diversity and disease), Climate Change – (weak asymmetry, again likely not bias, climate change generally increases disease), and Introduced Species (relatively weak asymmetry – unclear whether this is a bias, may be driven by some outliers). No significant asymmetries ($p > 0.05$) were found in Chemical Pollution and Habitat Loss/Change, suggesting negligible publication bias in reported disease responses across these global change drivers (**B**, **C**). Egger's test included publication year as moderator but found no significant relationship between Hedge's g and publication year ($p > 0.05$) implying no temporal bias in effect size magnitude or direction. In panel **D**, the horizontal red lines denote the grand mean and SE of Hedge's g and ($g = 0.1009$, $SE = 0.0338$). Grey points and error bars indicate the Hedge's g and SEs, respectively, using the leave-one-out method (grand mean is recalculated after a given study is removed from dataset). While the removal of certain studies resulted in values that differed from the grand mean, all estimated Hedge's g values fell well within the standard error of the grand mean. This sensitivity analysis indicates that our results were robust to the iterative exclusion of individual studies.

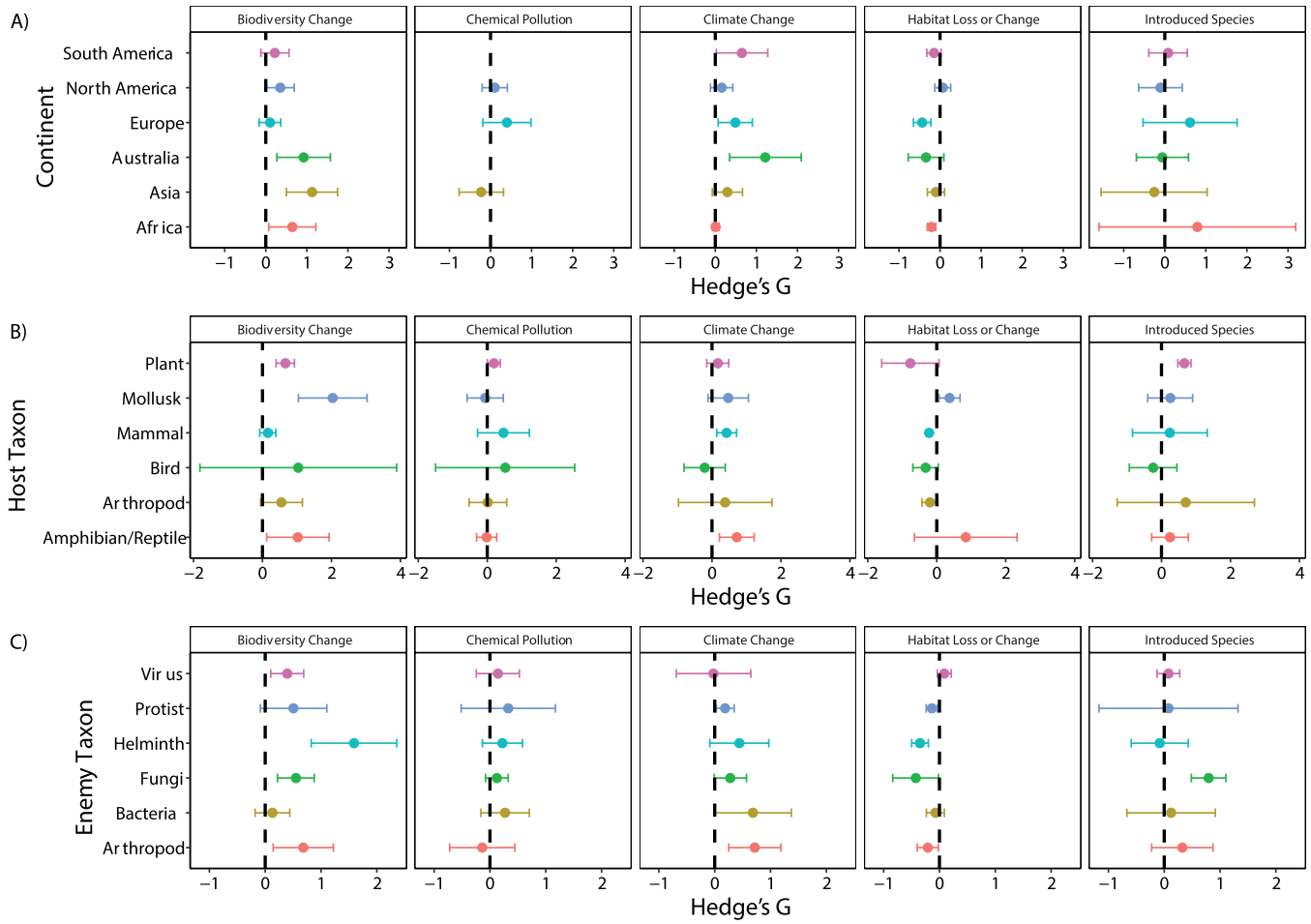


Extended Data Fig. 7 | See next page for caption.

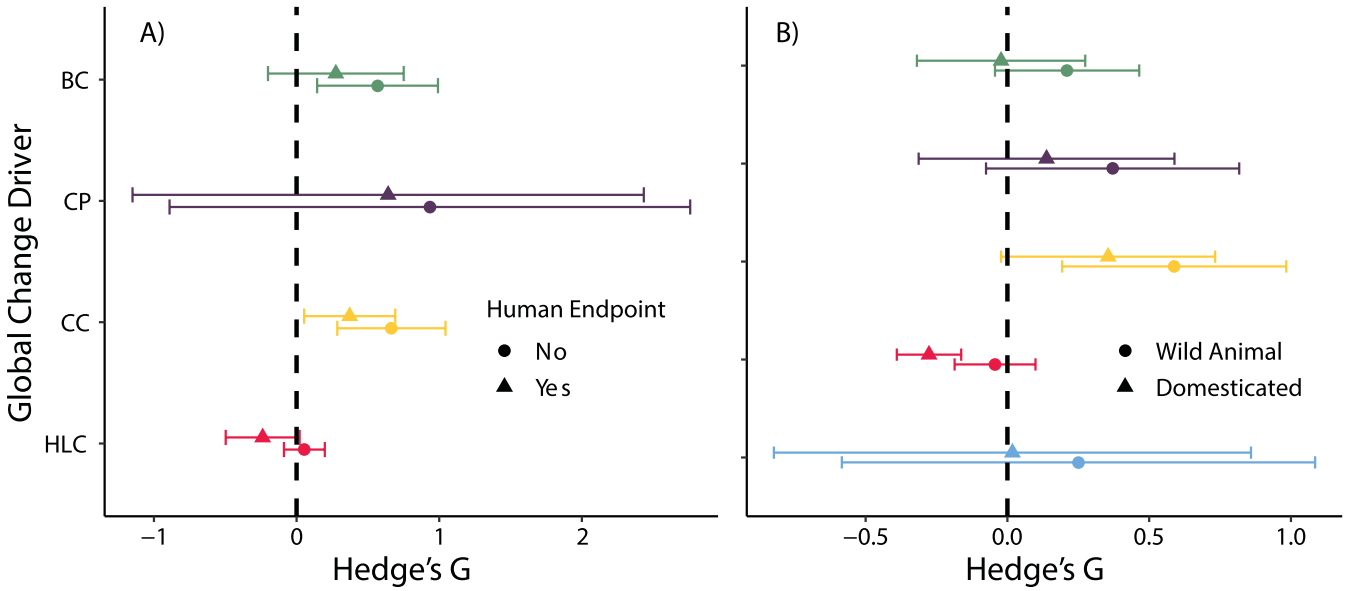
Extended Data Fig. 7 | The effects of habitat loss/change on disease depend on parasite taxa and land use conversion contexts. A) Enemy type influences the magnitude of the effect of urbanization on disease: helminths, protists, and arthropods were all negatively associated with urbanization, whereas viruses were non-significantly positively associated with urbanization. B) Reference (control) land use type influences the magnitude of the effect of urbanization on disease: disease was reduced in urban settings compared to rural and peri-urban settings, whereas there were no differences in disease along urbanization gradients or between urban and natural settings. C) The effect of forest fragmentation depends on whether a large/continuous habitat patch is compared to a small patch or whether disease it is measured along an increasing fragmentation gradient ($Z = -2.828$, $p = 0.005$). Conversely, the effect of deforestation on disease does not depend on whether the habitat has been destroyed and allowed to regrow (e.g., clearcutting, second growth forests, etc.) or whether it has been replaced with agriculture (e.g., row crop, agroforestry, livestock grazing; $Z = 1.809$, $p = 0.0705$). The displayed points represent the mean predicted values (with 95% confidence intervals) from a *metafor* model where the response variable was a Hedge's g (representing the effect on an infectious disease endpoint relative to control), study was treated as a random effect, and the independent variables included enemy type (A), reference land use type (B), or land use conversion type (C). Data for (A) and (B) were only those studies that were within the "urbanization" subcategory; data for (C) were only those studies that were within the "deforestation" and "forest fragmentation" subcategories. Sample sizes (number of studies, n, and effect sizes, k) in (A) for each enemy are n = 48, k = 98 for Virus; n = 193, k = 343 for Protist; n = 159, k = 490 for Helminth; n = 10, k = 24 for Fungi; n = 103, k = 223 for Bacteria; and n = 30, k = 73 for Arthropod. Sample sizes in (B) for each reference land use type are n = 391, k = 1073 for Rural; n = 29, k = 74 for Peri-urban; n = 33, k = 83 for Natural; and n = 24, k = 58 for Urban Gradient. Sample sizes in (C) for each land use conversion type are n = 7, k = 47 for Continuous Gradient; n = 16, k = 44 for High/Low Fragmentation; n = 11, k = 27 for Clearcut/Regrowth; and n = 21, k = 43 for Agriculture.



Extended Data Fig. 8 | The effects of common global change drivers on mean infectious disease responses in the literature depends on whether the endpoint is the host or parasite; whether the parasite is a vector, is vector-borne, has a complex or direct life cycle, or is a macroparasite; whether the host is an ectotherm or endotherm; or the venue and habitat in which the study was conducted. A) Parasite endpoints. **B**) Vector-borne status. **C**) Parasite transmission route. **D**) Parasite size. **E**) Venue. **F**) Habitat. **G**) Host thermy. **H**) Parasite type (ecto- or endoparasite). See Table S2 for number of studies and effect sizes across ecological contexts and global change drivers. See Table S3 for pairwise comparison results. The displayed points represent the mean predicted values (with 95% confidence intervals) from a *metafor* model where the response variable was a Hedge's g (representing the effect on an infectious disease endpoint relative to control), study was treated as a random effect, and the independent variables included the main effects and an interaction between global change driver and the focal independent variable (whether the endpoint measured was a host or parasite, whether the parasite is vector-borne, has a complex or direct life cycle, is a macroparasite, whether the study was conducted in the field or lab, habitat, the host is ectothermic, or the parasite is an ectoparasite).



Extended Data Fig. 9 | The effects of five common global change drivers on mean infectious disease responses in the literature only occasionally depend on location, host taxon, and parasite taxon. A) Continent in which the field study occurred. Lack of replication in chemical pollution precluded us from including South America, Australia, and Africa in this analysis. **B**) Host taxa. **C**) Enemy taxa. See Table S2 for number of studies and effect sizes across ecological contexts and global change drivers. See Table S3 for pairwise comparison results. The displayed points represent the mean predicted values (with 95% confidence intervals) from a *metafor* model where the response variable was a Hedge's g (representing the effect on an infectious disease endpoint relative to control), study was treated as a random effect, and the independent variables included the main effects and an interaction between global change driver and continent, host taxon, and enemy taxon.



Extended Data Fig. 10 | The effects of human vs. non-human endpoints for the zoonotic disease subset of database and wild vs. domesticated animal endpoints for the non-human animal subset of database are consistent across global change drivers. (A) Zoonotic disease responses measured on human hosts responded less positively (closer to zero when positive, further from zero when negative) than those measured on non-human (animal) hosts ($Z = 2.306$, $p = 0.021$). Note, IS studies were removed because of missing cells. (B) Disease responses measured on domestic animal hosts responded less positively (closer to zero when positive, further from zero when negative) than those measured on wild animal hosts ($Z = 2.636$, $p = 0.008$). These results were consistent across global change drivers (i.e., no significant interaction between endpoint and global change driver). As many of the global change drivers increase zoonotic parasites in non-human animals and all parasites in wild animals, this may suggest that anthropogenic change might increase the occurrence of parasite spillover from animals to humans and thus also pandemic risk. The displayed points represent the mean predicted values (with 95% confidence intervals) from a *metafor* model where the response variable was a Hedge's g (representing the effect on an infectious disease endpoint relative to control), study was treated as a random effect, and the independent variable of global change driver and human/non-human hosts. Data for (A) were only those diseases that are considered "zoonotic"; data for (B) were only those endpoints that were measured on non-human animals. Sample sizes in (A) for zoonotic disease measured on human endpoints across global change drivers are $n = 3$, $k = 17$ for BC; $n = 2$, $k = 6$ for CP; $n = 25$, $k = 39$ for CC; and $n = 175$, $k = 331$ for HLC. Sample sizes in (A) for zoonotic disease measured on non-human endpoints across global change drivers are $n = 25$, $k = 52$ for BC; $n = 2$, $k = 3$ for CP; $n = 18$, $k = 29$ for CC; $n = 126$, $k = 289$ for HLC. Sample sizes in (B) for wild animal endpoints across global change drivers are $n = 28$, $k = 69$ for BC; $n = 21$, $k = 44$ for CP; $n = 50$, $k = 89$ for CC; $n = 121$, $k = 360$ for HLC; and $n = 29$, $k = 45$ for IS. Sample sizes in (B) for domesticated animal endpoints across global change drivers are $n = 2$, $k = 4$ for BC; $n = 4$, $k = 11$ for CP; $n = 7$, $k = 20$ for CC; $n = 78$, $k = 197$ for HLC; and $n = 1$, $k = 2$ for IS.

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For all statistical analyses, confirm that the following items are present in the figure legend, table legend, main text, or Methods section.

n/a	Confirmed	
☐	☒	The exact sample size (*n*) for each experimental group/condition, given as a discrete number and unit of measurement
☐	☒	A statement on whether measurements were taken from distinct samples or whether the same sample was measured repeatedly
☐	☒	The statistical test(s) used AND whether they are one- or two-sided *Only common tests should be described solely by name; describe more complex techniques in the Methods section.*
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☐	☒	A full description of the statistical parameters including central tendency (e.g. means) or other basic estimates (e.g. regression coefficient) AND variation (e.g. standard deviation) or associated estimates of uncertainty (e.g. confidence intervals)
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☒	☐	For Bayesian analysis, information on the choice of priors and Markov chain Monte Carlo settings
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☐	☒	Estimates of effect sizes (e.g. Cohen's *d*, Pearson's *r*), indicating how they were calculated

Our web collection on statistics for biologists contains articles on many of the points above.

Software and code

Policy information about availability of computer code

Data collection	Webplotdigitizer version 4.2 was used to pull data from published papers.
Data analysis	All the analyses were conducted with R (version R-4.2.2) and R Studio (version 2022.12.0 Build 353- "Elsbeth Geranium" Release yd165dcf, 2022-12-03 for Windows). The code and the RMarkdown html files are provided. The packages and versions (in parentheses) used for these analyses are as follows. car (3.1-2), clubSandwich (0.5.8), cowplot (1.1.1), effectsize (0.8.3), emmeans (1.8.5), esc (0.5.1), forcats (1.0.0), ggeffects (1.2.1), lme4 (1.1-32) metafor (4.2-0), MuMIn (1.47.5), multcomp (1.4-23), rmarkdown (2.21), stringr (1.5.0), tidyverse (2.0.0). The custom code has been deposited at Zenodo: DOI: .

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Data

Policy information about availability of data

All manuscripts must include a data availability statement. This statement should provide the following information, where applicable:

- Accession codes, unique identifiers, or web links for publicly available datasets
- A description of any restrictions on data availability
- For clinical datasets or third party data, please ensure that the statement adheres to our policy

All the data and code are provided with the manuscript.

Human research participants

Policy information about studies involving human research participants and Sex and Gender in Research.

Reporting on sex and gender	N/A
Population characteristics	N/A
Recruitment	N/A
Ethics oversight	N/A

Note that full information on the approval of the study protocol must also be provided in the manuscript.

Field-specific reporting

Please select the one below that is the best fit for your research. If you are not sure, read the appropriate sections before making your selection.

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Life sciences study design

All studies must disclose on these points even when the disclosure is negative.

Sample size	Given that this manuscript is a meta-analysis, the sample sizes were determined by the number of available studies and effect sizes.
Data exclusions	When data were missing from a study (e.g., not presented in a main text or supplemental figure or table, in the text itself, or provided with included datasets), we did not contact the original authors. Studies with missing values were removed from the analyses. See the PRISMA chart for information on number of studies removed.
Replication	N/A. Determined by each paper in the meta-analysis
Randomization	N/A. Determined by each paper in the meta-analysis
Blinding	N/A

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Materials & experimental systems

n/a	Involved in the study
☒	☐ Antibodies
☒	☐ Eukaryotic cell lines
☒	☐ Palaeontology and archaeology
☒	☐ Animals and other organisms
☒	☐ Clinical data
☒	☐ Dual use research of concern

Methods

n/a	Involved in the study
☒	☐ ChIP-seq
☒	☐ Flow cytometry
☒	☐ MRI-based neuroimaging