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# Warmer Is Deadlier: A Meta-Analysis Reveals Increasing Temperatures Accentuate Disease Effects on Fisheries Hosts

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## ABSTRACT

Rapid warming could drastically alter host–parasite relationships, which is especially important for fisheries crucial to human nutrition and economic livelihoods, yet we lack a synthetic understanding of how warming influences parasite-induced mortality in these systems. We conducted a meta-analysis using 266 effect sizes from 52 empirical papers on harvested aquatic species and determined the relationship between parasite-induced host mortality and temperature and how this relationship was altered by host, parasite, and study design traits. Overall, higher temperatures increased parasite-induced host mortality; however, the magnitude of this relationship varied. Hosts from the order Salmoniformes experienced a greater increase in parasite-induced mortality with temperature than the average response to temperature across fish orders. Opportunistic parasites were associated with a greater increase in infected host mortality with temperature than the average across parasite strategies, while bacterial parasites were associated with lower infected host mortality as temperature increased than the average across parasite types. Thus, parasites will generally increase host mortality as the environment warms; however, this effect will vary among systems.

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## 1 | Introduction

Parasite-induced host pathology and mortality are products of the interactions between susceptible hosts, parasites and the environment. How severely parasites, both micro-parasites (e.g., viruses and bacteria) and macro-parasites (e.g., parasitic arthropods), affect their hosts often depends on the environmental context. In exposed hosts, if the environment favours the physiological optimum of the parasite, symptomatic infectious disease will occur; otherwise, the host may mount a sufficient immune response and is unlikely to experience severe pathology (Snieszko 1974). Temperature is a critical environmental factor that affects all levels of biological organisation (Brown et al. 2004; Venesky et al. 2022), from cellular and metabolic functions (Brandt and Hartman 1993; Madeira et al. 2013; Deslauriers et al. 2017; Molnár et al. 2017; Paumier et al. 2020) to phenological processes, such as migration and reproduction (Caudill et al. 2013; Morita et al. 2014; Paumier et al. 2020). Parasites of ectotherms and free-living infectious parasite stages are subject to environmental temperatures. Increased temperature typically accelerates the rate at which parasites complete their lifecycle and increases the emergence rate of infectious stages (Brown et al. 2004; Poulin 2006; Cascarano et al. 2021). A universal positive response of parasites to increasing temperature, however, is far from certain (Gehman et al. 2018; Shodipo et al. 2020; Byers 2021; Wood et al. 2023).

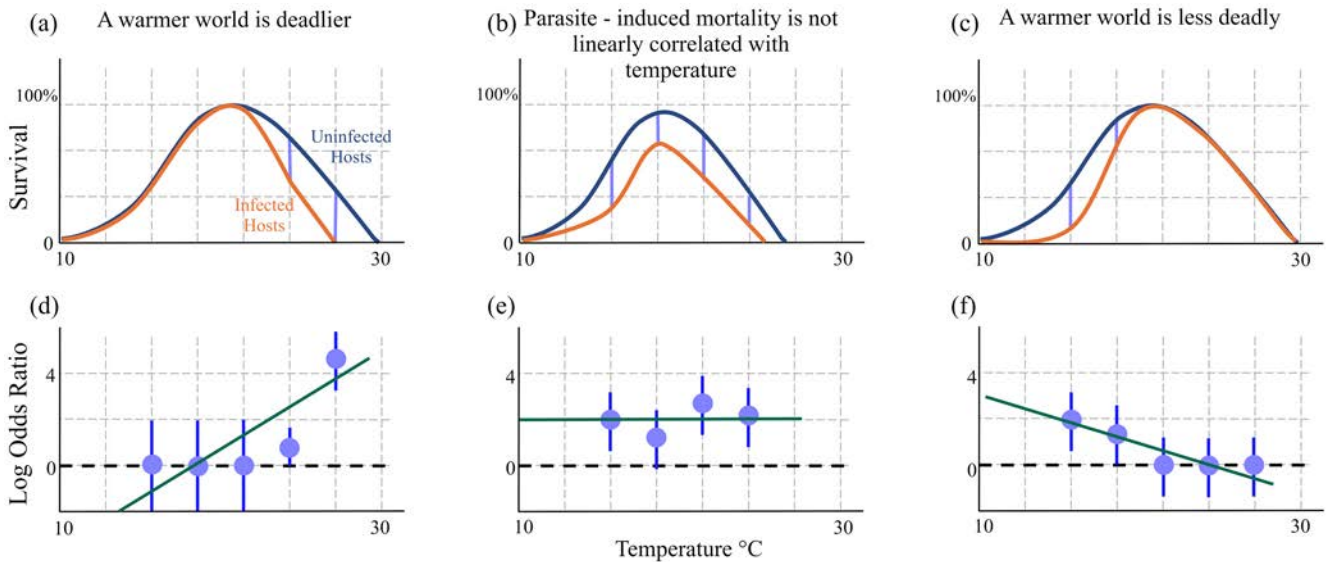
Understanding the host–parasite dynamics of commercially fished species in a warming world is of paramount importance given the contribution of commercially fished species to human nutrition and economic livelihoods, their role in ecosystem function, and the marked declines in biomass and density of these species (Pauly et al. 2002; Pitcher and Cheung 2013). There is extensive literature examining fisheries hosts, their parasites, and how they respond to temperature, allowing for a comprehensive analysis of how these relationships may shift with warming. Fish physiology is closely linked to temperature (Kitchell et al. 1977; Brandt and Hartman 1993; Deslauriers et al. 2017; Shields 2019), and rising temperatures likely influence the population structure of many fished species (Brander 2007; Geffroy and Wedekind 2020). Pathogens can have significant effects on marine fisheries (Lafferty et al. 2015; Lafferty 2017) and the aquaculture industry has highlighted the importance of infectious diseases in a wide range of taxa (Behringer et al. 2020). Parasite dynamics and transmission are also likely affected by temperature and can further alter host mortality patterns (Karvonen et al. 2010). Thus, host–parasite dynamics in fisheries and their interaction with temperature provide a compelling system of critical importance in which to investigate the effects of temperature.

As environments have warmed, reports on the frequency or intensity of parasitism have increased in molluscs (Harvell et al. 2004), freshwater fishes (Johnson and Paull 2011), and crustaceans (Shields 2019), but declined in marine fishes (Ward and Lafferty 2004). How temperature drives these trends is uncertain, but other concurrent factors including fishing, pollution, restoration and invasive species may also contribute to these patterns (Ward and Lafferty 2004). Reviews and

meta-analyses suggest that temperature has variable effects on parasite incidence and/or severity (Löhmus and Björklund 2015; Poulin 2020), with warming causing increased parasitism effects in some systems (Fey et al. 2015; Aalto et al. 2020) but declines in others (Barnes 2011; Wood et al. 2023). Thus, a comprehensive, quantitative analysis estimating the effect of warming on fisheries host–parasite interactions would broaden our understanding of how warming may affect fished species. More importantly, we need to evaluate which host and parasite traits best explain the observed heterogeneity in the response of the host–parasite system to increased temperature, and therefore better predict the fisheries most likely to experience increased infectious disease mortality as water temperatures continue to warm (Figure 1). Additionally, we need to assess how study design might influence these results, as biases caused by the motivation of the researchers and their choices in experiment duration and execution may lead to varying conclusions of the effect of warming on these systems.

Thermal performance curves provide a useful conceptual framework for understanding and predicting how temperature alters host–parasite interactions. All organisms experience peak performance (e.g., metabolic optimum, reproductive rate, immune efficacy) at an intermediate temperature (i.e., their thermal optimum), and a decline in performance as temperatures diverge from this optimum (Molnár et al. 2017). Because parasites often have a broader thermal performance curve than hosts, the thermal mismatch hypothesis proposes that parasites are likely to outperform their hosts as the climate warms (Cohen et al. 2017; Cascarano et al. 2021; but refer to Gehman et al. 2018 and Wood et al. 2023 for counter examples). This hypothesis suggests increased temperature will broadly enable parasites to overcome host defences and induce pathology (Cohen et al. 2017). However, the outcome of any particular host–parasite interaction depends on the nature of the overlap between their thermal performance curves, which can be influenced by numerous factors related to host and parasite physiology, host distribution, parasite specialisation and co-evolutionary history (Karvonen et al. 2010; Sauer et al. 2020; Byers 2021).

Here, we perform a meta-analysis of experimental studies that manipulated temperature in host–parasite systems involving 32 fished species and 37 parasites. Our goal is to identify patterns and drivers of different relationships between temperature and infection-induced mortality. First, we quantify: (1) the extent to which parasites increase mortality of their hosts; (2) the relationship between infection-induced mortality and temperature; and (3) the role of various host, parasite and study design traits in driving variation in the relationship between temperature and infection-induced host mortality (Figure 1). Second, we perform a more focused analysis to identify drivers of temperature-infection mortality relationships in a well-represented, economically and ecologically important taxon, Salmoniformes (i.e., salmon, trout and their relatives). We then outline key information that is needed for a more comprehensive understanding of warming on parasite-induced mortality in fisheries and conclude by discussing the implications of our findings in the context of fisheries management in a warming world.



**FIGURE 1** | Conceptual figure depicting a range of dynamics between hosts and parasites within an experiment. Panels (a–c) illustrate three possible effects of warming on infected (orange) and uninfected (blue) hosts. The differences in host survival at a given temperature are indicated by purple vertical lines (i.e., parasite-induced host mortality). Panels (d–f) depict the resulting log odds ratios (and their uncertainty), with the fitted linear trendlines (green). The ‘warmer world is deadlier’ scenario is depicted in panels (a, d). Panels (b, e) illustrate the scenario in which parasite-induced host mortality does not change appreciably with changing temperature, and panels (c, f) indicate the scenario in which warming leads to a decrease in parasite-induced mortality.

## 2 | Materials and Methods

### 2.1 | Data Collection

We compiled data from experimental studies on fisheries species that compared mortality of parasitized and unparasitized hosts at a static temperature. We defined fisheries species to include both invertebrate and vertebrate species that are harvested commercially or recreationally. In the fall of 2019, we searched Web of Science following systematic review protocols (Foo et al. 2021) using key terms that would return papers focused on harvested aquatic species, parasites and diseases, but would exclude papers that were focused on human, environmental or domestic animal health (see Appendix S1 in Supporting Information). This search yielded 1201 papers. We then screened the abstracts of these papers and retained only papers that satisfied four criteria: (1) an experiment was performed that included at least one parasite exposure treatment paired with an unexposed control group, (2) temperatures were intended to be constant within a temperature treatment and not intentionally varied, (3) hosts were from species that constitute a fishery, including those in aquaculture and (4) estimates of survival or mortality were reported for both infected and uninfected hosts at each temperature treatment. This selection process reduced the number of papers to 386 (Appendix S1; Figure S1).

We obtained full versions of 358 papers (28 papers from the original 386 were unobtainable). We then screened the full text of these papers to ensure a match to our four criteria, which reduced the 358 papers to 70. To increase statistical power to estimate the effect of host Order on parasite-induced mortality, we excluded experiments from hosts in Orders with fewer than two experiments with  $\geq 2$  temperature treatments.

We also removed experiments with zero mortality in infected and uninfected treatments at all experimental temperatures. These experiments provided no information about how parasite-induced mortality changed with temperature. This reduced the number of papers included in our dataset from 70 to 52 and yielded a total of 266 effect sizes from 121 experiments (several papers included more than one experiment; Appendixes S1 and S2, Figure S1). Experiments are defined as unique combinations of a host, a parasite and sometimes an additional factor per paper. For example, if a paper crossed parasite exposure with two salinities at three temperatures, we considered this to constitute two experiments (each with three levels of temperature; Appendix S2).

At least two people extracted data from each paper to reduce extraction error. If extracted values differed, the data were re-extracted until there was agreement between the two extractors. For data that were displayed in a graphical format only, we used WebPlotDigitizer (Rohatgi 2022) to extract data. Data (which may have been presented as mortality rates, or proportion surviving) were converted to numbers of host individuals that were dead and alive at the end of the experiment. We also extracted information about the paper itself, including the source of the hosts used in the paper and the motivation for conducting the experiment, which we deemed ‘Study traits’ (see Appendix S1). Paper motivation was delineated into three categories—papers motivated by climate change, papers motivated by a natural temperature change (e.g., seasons) or ‘other’. The Introduction and Discussion sections of each paper were thoroughly screened to find key words relating to each delineation to assign paper motivation. Finally, we collected additional information about host and parasite traits from outside sources (e.g., other peer reviewed papers, government reports) when necessary to obtain moderator variables

**TABLE 1** | Moderator variables included in candidate models. Category indicates if the variable was a trait of the host, parasite or study. The levels of the moderator variable provide the options and the number of effect sizes ( $n$ ) associated with that level. The moderator variable ‘Duration’ does not have levels because it is a continuous variable. Refer to Appendix S1 for explanation of variables and variable levels and associated a priori hypotheses regarding the variable’s influence on the relationship between parasite-induced host mortality and temperature.

Category	Variable	Levels (number of effect sizes)	
Host	Phylum	Arthropoda (9), Chordata (180), Mollusca (77)	
	Class	Acanthopterygii (9), Actinopterygii (171), Bivalvia (77), Malacostraca (9)	
	Order	Cichliformes (6), Cypriniformes (25), Decapoda (9), Mytilida (26), Ostreidae (45), Perciformes (32), Pleuronectiformes (26), Salmoniformes (69), Siluriformes (22), Venerida (6)	
	Mobility	Immobile (65), Mobile (201)	
	Vertebrae	Invertebrate (86), Vertebrate (180)	
	Life Stage	Adult (35), Juveniles (231)	
	Distribution	Subtropical (131), Temperate (89), Tropical (46)	
	Salinity	Freshwater (34), Mixed (216), Marine (16)	
	Parasite	Location	External (10), Internal (256)
		Type	Bacteria (157), Eukaryote (49), Virus (60)
Strategy		Obligate (124), Opportunistic (142)	
Transmission		Direct Transmission (62), Indirect Transmission (193), Ingestion (11)	
Study	Duration	Duration of the experiment; continuous (range: 3–350 days)	
	Host Source	Captive (181), Wild (66), Unknown (19)	
	Motivation	Climate Change (53), Temperature Correlation (86), Other (127)	

(Table 1, Appendix S1). The moderators (Table 1) were used to test a priori hypotheses regarding how host, parasite and study design traits influenced how temperature affected parasite-induced mortality.

## 2.2 | Effect Sizes

Because our focus was on parasite-induced mortality, we used log odds ratios as our effect size to compare host survival in the parasitized versus unparasitized treatments:

$$\text{LOR} = \ln\left(\frac{(D_p + Y) / (A_p + Y)}{(D_U + Y) / (A_U + Y)}\right) \quad (1)$$

where  $D_p$  is the number of parasitized hosts that died,  $A_p$  is the number of parasitized hosts that survived,  $D_U$  is the number of unparasitized hosts that died, and  $A_U$  is the number of unparasitized hosts that survived at a given temperature in a given experiment. The Yates constant ( $Y=0.5$ ; Yates 1934) was applied when  $D_p$ ,  $D_U$ ,  $A_p$  or  $A_U$  was 0 to prevent the LOR from being undefined; otherwise,  $Y=0$  (Sauer et al. 2020). The variance for each log odds ratio was calculated as:

$$\text{Variance}_{\text{LOR}} = \frac{1}{D_p + Y} + \frac{1}{A_p + Y} + \frac{1}{D_U + Y} + \frac{1}{A_U + Y} \quad (2)$$

A positive log odds ratio ( $\text{LOR} > 0$ ) with a 95% confidence interval that does not encompass zero indicates that parasitized hosts exhibited greater mortality than unparasitized hosts;  $\text{LOR} < 0$  indicates that parasitized hosts survived better than unparasitized hosts.

## 2.3 | Statistical Analysis

Similar to Sauer et al. (2020), we used a mixed effects meta-analysis (*metafor* package version 4.8, *rma.mv* function; Viechtbauer 2010) to analyse our data:

$$\text{LOR}_i \sim \beta_0 + \beta_T T_i + \gamma_h + \gamma_k + \gamma_i + \beta_D D_i + \beta_{TD} T_i D_i + \sum_{j=1, n_j} \beta_j F_{ji} + \sum_{j=1, n_j} \beta_{Tj} T_i F_{ji} + \epsilon_i \quad (3)$$

in which the index  $i$  identifies each effect size ( $i=1\dots 266$ ), and its suite of associated moderators. The intercept,  $\beta_0$ , provides the estimate of the overall effect of parasites on host mortality when temperature ( $T_i$ ), measured in degrees Celsius and mean centered (*base* package, *scale* function), and all other moderators are equal to zero. To understand if the log odds ratio increased with temperature within a study, we included temperature as a moderator (with coefficient  $\beta_T$ ), as well as random intercepts ( $\gamma_{h,k,i}$ ) that addresses non-independence within study ( $h$ ), experiment ( $k$ ), and effect size ( $i$ ). To control for the possibility that LOR changed with time, we estimated the effect of experimental duration ( $D$ , in days) with coefficient  $\beta_D$ ;  $\beta_{TD}$  estimates how the relationship between log odds ratio and temperature was influenced by the duration of the study. We included additional categorical moderator variables (i.e., host, parasite, or study traits; Table 1), with  $F_{ji}$  as either 0 or 1 indicating the moderator level associated with effect size  $i$  (e.g., *vertebrate*, *bacterial parasite*, etc.), and  $n_j$  representing the number of moderator levels. The main effect of the moderator level  $j$  is represented by  $\beta_j$ , while the interactive effect with temperature is represented by  $\beta_{Tj}$ . The error associated with the  $i^{\text{th}}$  log odds ratio is denoted as  $\epsilon_i$ .

$\sim N(0, \text{Variance}_{\text{LOR}_i})$ , where  $\text{Variance}_{\text{LOR}_i}$  is the variance around  $\text{LOR}_i$  calculated in (Equation 2).

This model accounts for heterogeneity among experiments by allowing the relationship between temperature and LOR to have a different intercept for each experiment. The overall slope ( $\beta_T$ ) provides an estimate of how LOR changes with temperature on average. Additionally, the random intercepts ( $\gamma_{h,k,i}$ ) account for variation in the relationship between LOR and temperature that results from factors we were unable to model explicitly (e.g., dosage, geographic source of host or parasite, laboratory facilities). Our approach also allows us to include experiments with only a single temperature treatment. While these experiments do not inform the estimate of the effect of temperature, they do provide information on the intercept and therefore the overall effect of parasites on host mortality.

Our methodology provides an estimate of how changes in temperature produce changes in parasite-induced host mortality by using mean centred temperature values as a predictor variable. We chose to use temperature, rather than temperature change, as a predictor variable for two main reasons. First, using temperature change would necessitate calculating a change in LOR as a response variable, but there is no established method to combine the associated variance in LORs for this metric. Second, it is unclear how to best deal with experiments that include more than 2 temperatures. It is unlikely that a linear or non-linear model fit with only 3–4 data points would provide an accurate slope estimate between multiple LORs and thus provide an inaccurate estimate of the effect of temperature change on parasite-induced host mortality. Therefore, our approach relies on random effects to parse the influence of temperature change from variation caused by individual experiments to produce an estimate of the average change in parasite-induced mortality with altered temperature.

We explored 12 versions of Equation 3 that avoided collinearity that arose between host taxonomic information and parasite or host traits. Because host taxonomy was generally indicative of host traits, we explored effects of taxonomy in three stand-alone models that excluded the trait moderators. Similarly, several host and parasite traits were perfectly correlated (e.g., there were no immobile vertebrates, so host Mobility and Vertebrae are perfectly correlated). Therefore, we also analysed eight trait-based models that reduced collinearity among all the traits included in the model. In addition, we included a null model with only random effects. The 12 models were:

- Host Taxonomy Models (HTM1, HTM2, HTM3), in which the fixed factors were host Taxonomy (1 = Phylum, 2 = Class or 3 = Order), Temperature, and Duration, and the interactions of Temperature with Duration and Host Taxonomy. Because of the low sample size of some phyla, we were unable to include any other fixed effects in this model without the variables being perfectly correlated (Table 2).
- Eight Trait Models (TM1-8), in which host phylogeny was omitted, but the largest possible set of fixed effects, while avoiding perfectly correlated variables, was included. These sets of effects included interactions of Temperature with the

other fixed effects and always included Duration. These 8 candidate models are provided in Table 2.

- A null model, in which all fixed effects were omitted, served as a basis to evaluate the performance of the fixed effects.

In addition to running the above models on the full dataset, we also applied two trait-based models, single categorical trait models, a duration-only model, and a null model on a reduced dataset that was restricted to the Order Salmoniformes, the most common Order in the dataset (comprising 26% of the effect sizes). This allowed us to explore temperature effects in a well-studied and economically important group. We refer to these analyses as the Salmoniformes Trait Models (STM1 and STM2), the Salmoniformes Single Categorical Trait Models (SSCTM1-5), the Salmoniformes Duration model (SDUR) and the Salmoniformes null model. Some traits (e.g., host Vertebrae and Mobility) and factor levels did not apply to the Salmoniformes dataset. We also removed a single experiment that used a viral parasite from our dataset to allow for the inclusion of parasite Type in our candidate models. The STM1 included Duration, host Distribution (Subtropical and Temperate) and parasite Type (Bacteria or Eukaryote). The STM2 included Duration, host Distribution (Subtropical and Temperate), and Parasite Strategy. All SSCTM models included Duration and one of the following traits: host Distribution (Subtropical and Temperate), Salinity (Freshwater and Mixed), parasite Type (Bacteria or Eukaryote), Strategy and study Motivation. The SDUR model only included Duration (Table 2).

Excluding the null models, we used backwards elimination to produce a model that included only factors with significant interactions with temperature ( $p < 0.1$ ) in both the full and Salmoniformes datasets. All the models generated in this process were compared using an Information-Theoretic approach that utilises Akaike Information Criterion corrected for sample size (AICc; Akaike 1988; Blankenship et al. 2002; Whittingham et al. 2006) to select the best fit model. We also calculated McFadden's Pseudo- $R^2$  (Hemmert et al. 2018) and Nakagawa's Pseudo- $R^2$  for generalised linear mixed-effects models (*orchaRd* package version 2.0, *r2\_ml* function; Nakagawa et al. 2023; Nakagawa and Schielzeth 2013) which estimate the amount of additional variation explained by the mixed-effects model compared to the null model. Nakagawa's Pseudo- $R^2$  provides a goodness-of-fit metric for both the fixed-effects of the model (Marginal  $R^2$ ) and for the full mixed-effects model (Conditional  $R^2$ ). While our models estimate both main effects and interactions with temperature of our fixed effects, we discuss only the interactions because these are the estimates that reflect how the effects of temperature on parasite-induced mortality are influenced by host, parasite, and study design traits.

We additionally evaluated publication bias and the degree of variance caused by heterogeneity in the true effect sizes compared to the variance caused by sampling error in our data. To evaluate publication bias, we assess the effect of low sample size studies on the overall effect and if the overall effect declines with publication year using the procedures outlined in Nakagawa et al. (2022) (Appendix S3) for both the full and Salmoniformes datasets. We calculated the typical sampling error variance from studies contained in our meta-analysis to estimate the statistical

**TABLE 2** | List of candidate models for the full and salmoniformes dataset.

Model category	Model number	Host					Parasite					Study				
		Phylum	Class	Order	Mobility	Vertebrae	Life stage	Distribution	Salinity	Location	Type	Strategy	Transmission	Host source	Motivation	Duration
Host Taxonomy Models	HTM1	X														X
	HTM2		X													X
	HTM3			X												X
Trait Models	TM1					X		X	X	X	X					X
	TM2				X				X	X	X				X	X
	TM3					X			X	X	X				X	X
	TM4								X	X	X	X				X
	TM5				X					X	X			X		X
	TM6					X				X	X			X		X
	TM7										X	X				X
	TM8							X	X		X					X
Salmoniformes Models	STM1						X			X						X
	STM2										X					X
	SSCTM1														X	X
	SSCTM2							X								X
	SSCTM3								X							X
SSCTM4										X					X	
SSCTM5								X							X	

Note: Model number is provided with 'X', indicating which host, parasite or study design trait was included in the model. Grey gradients separate model categories. For variable names and description see Table 1.

noise in our data (*sigma2\_v* function; Yang et al. 2023) as well as the  $I^2$  metric to interpret the heterogeneity in our data and determine the variability in the true effect attributed to each level of our random effects (Higgins and Thompson 2002; *orchaRd* package version 2.0, *i2\_ml* function, Nakagawa et al. 2023). All analyses were performed using R version 4.4.2 (R Core Team 2024).

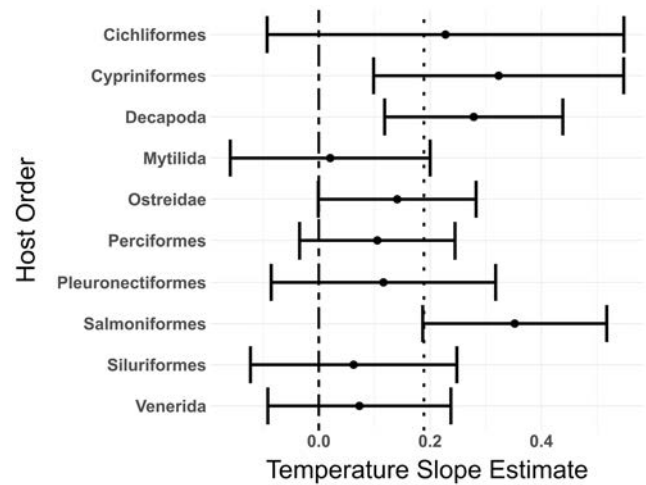
### 3 | Results

Our literature search resulted in 266 log odds ratios from 121 experiments, presented in 52 papers, which included data on 35 unique host species and 40 unique parasites (Table 1, Appendix S2). The temperatures in these experiments ranged from 4°C to 35°C, and the durations of the experiments ranged from 3 to 350 days. The null model from the full dataset (Equation 3) estimated the average LOR (i.e., the intercept) as 3.12 (95% CI =  $\pm 0.43$ , PI =  $\pm 3.35$ ,  $z = 14.02$ ,  $p < 0.0001$ ), indicating that, on average, parasitized hosts were more likely to experience mortality compared to their unparasitized counterparts. Our null model also indicated a large amount of sampling variance (1.11) and variance in the true effect estimate (2.87). Our  $I^2$  calculation indicated that 72.08% of the total variance was due to variance in the true effects and partitioned 38.86% of that variance to variance between studies, 33.23% of the variance between effect sizes, and 0% between experiments. The best models (see below) estimated the main effect of log odds ratio and temperature as positive, and the estimated slope for each level of the moderators was also generally positive, although the statistical significance and magnitude of these estimates varied (Figures 2 and 3, Tables S1 and S2). These results indicate that parasite-induced mortality was generally positively correlated with temperature, but that there are significant host, parasite, and study design factors that significantly alter this effect.

#### 3.1 | Host Taxonomy Models

The Host Taxonomy Model that included host Order (HTM3) was the best of the three host taxonomy models (McFadden's Pseudo- $R^2 = 0.11$ ; Nakagawa's Marginal  $R^2 = 0.34$ , Conditional  $R^2 = 0.64$ ; Table S1), and was within 2 AICc points from the best fit Trait Models. The other two host phylogeny models (host Phylum and host Class) were greater than 40 AICc points from HTM3 (HTM1:  $\Delta AICc = 47$ ; HTM2:  $\Delta AICc = 42$ ). The null model also a worse fit than HTM3 ( $\Delta AICc = 74$ ). The HTM3 estimated a statistically significant positive main effect of Temperature on LOR ( $\beta_T = 0.189$ ,  $z = 3.912$ ,  $p < 0.001$ ), indicating that, on average, temperature increases parasite-induced mortality.

All Orders in HTM3 had a positive estimated slope between LOR and Temperature, indicating increasing effects of parasites on host mortality as temperature increased (Figure 2), but these estimates were not always significant. The most sensitive Order to temperature was the Salmoniformes, which had a marginally significantly steeper increase in LOR with Temperature than average ( $\beta_{T, \text{Salmoniformes}} = 0.163$ ,  $z = 1.934$ ,  $p = 0.053$ ; Figure 2, Table S1). Hosts from Order Mytilida (mussels) had a negative interaction with Temperature, indicating that this host Order was less sensitive to increases in parasite-induced mortality

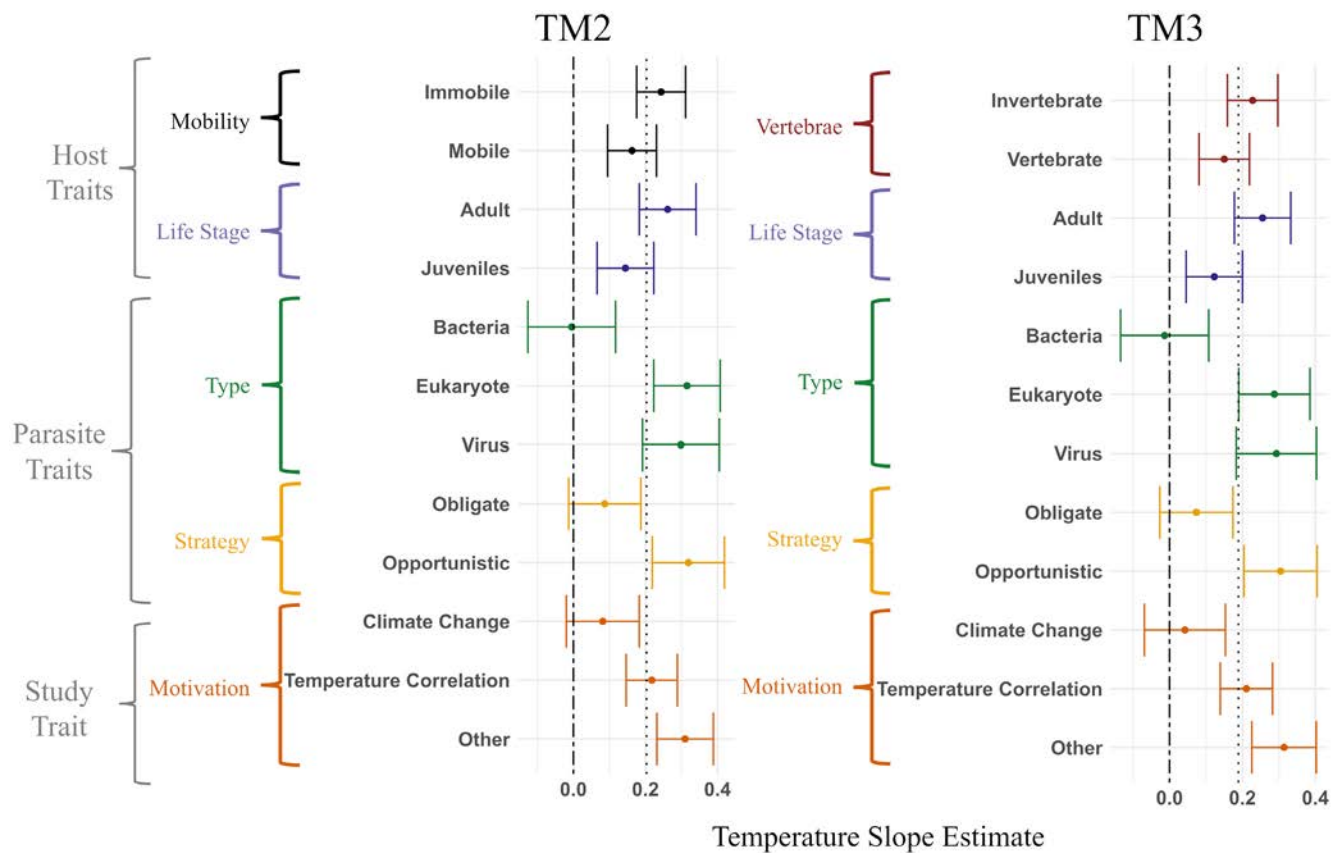


**FIGURE 2** | Temperature and host order interaction effect estimates from the Host Taxonomy Model 3 (HTM3). The dots display the estimates from the HTM3 for the overall mean slope ( $\beta_T$ ) plus the Host Order interaction effect estimates ( $\beta_{Tj}$  in Equation 3) and 95% confidence intervals. The dotted vertical line delineates the estimate of the overall mean slope (i.e., the main effect of temperature change on log odds ratio; 0.189). Log odds ratio reflects the difference in mortality between treatment and control hosts. If the 95% confidence interval of an interaction effect estimate crosses this dotted line, the interaction effect of that factor level does not deviate significantly from the average relationship. The dot-dashed vertical line delineates a zero slope estimate where estimates to the right of this line indicate a positive relationship with that factor level and temperature. Four of the 10 host orders had a significantly positive interaction with temperature.

with temperature than average ( $\beta_{T, \text{Mytilida}} = -0.168$ ,  $z = -1.837$ ,  $p = 0.066$ ). When including the main effect of Temperature in the Mytilida slope estimate (e.g.,  $\beta_T + \beta_{T, \text{Mytilida}}$ ) the estimated slope between LOR and temperature were positive, although the confidence interval overlaps 0 (Figure 2). The main effect of experiment Duration ( $\beta_D = -0.018$ ,  $z = -2.484$ ,  $p = 0.013$ ) and its interaction with Temperature ( $\beta_{TD} = -0.003$ ,  $z = -2.521$ ,  $p = 0.012$ ) were also statistically significant, indicating that longer studies are more likely to demonstrate a smaller effect of parasites on host mortality, and that longer studies led to a smaller effect of Temperature on parasite-induced host mortality.

#### 3.2 | Trait Models

Two Trait Models had equivalent AICc scores (TM2 and TM3), and identified host, parasite, and study design traits as important in influencing the relationship between temperature and parasite-induced mortality (TM2: McFadden's Pseudo- $R^2 = 0.1$ ; Nakagawa's Marginal  $R^2 = 0.4$ , Conditional  $R^2 = 0.67$ ; TM3: McFadden's Pseudo- $R^2 = 0.1$ ; Nakagawa's Marginal  $R^2 = 0.4$ , Conditional  $R^2 = 0.67$ ). These models are nearly identical: model TM2 includes the factor Host mobility, while TM3 includes Host Vertebrata, but otherwise both models consist of the same parameters (Table 2). Reducing the number of factors included in each of these models increased the AICc score, therefore the original models were retained as the best fit models. Both models include a significant increase in LOR with Temperature

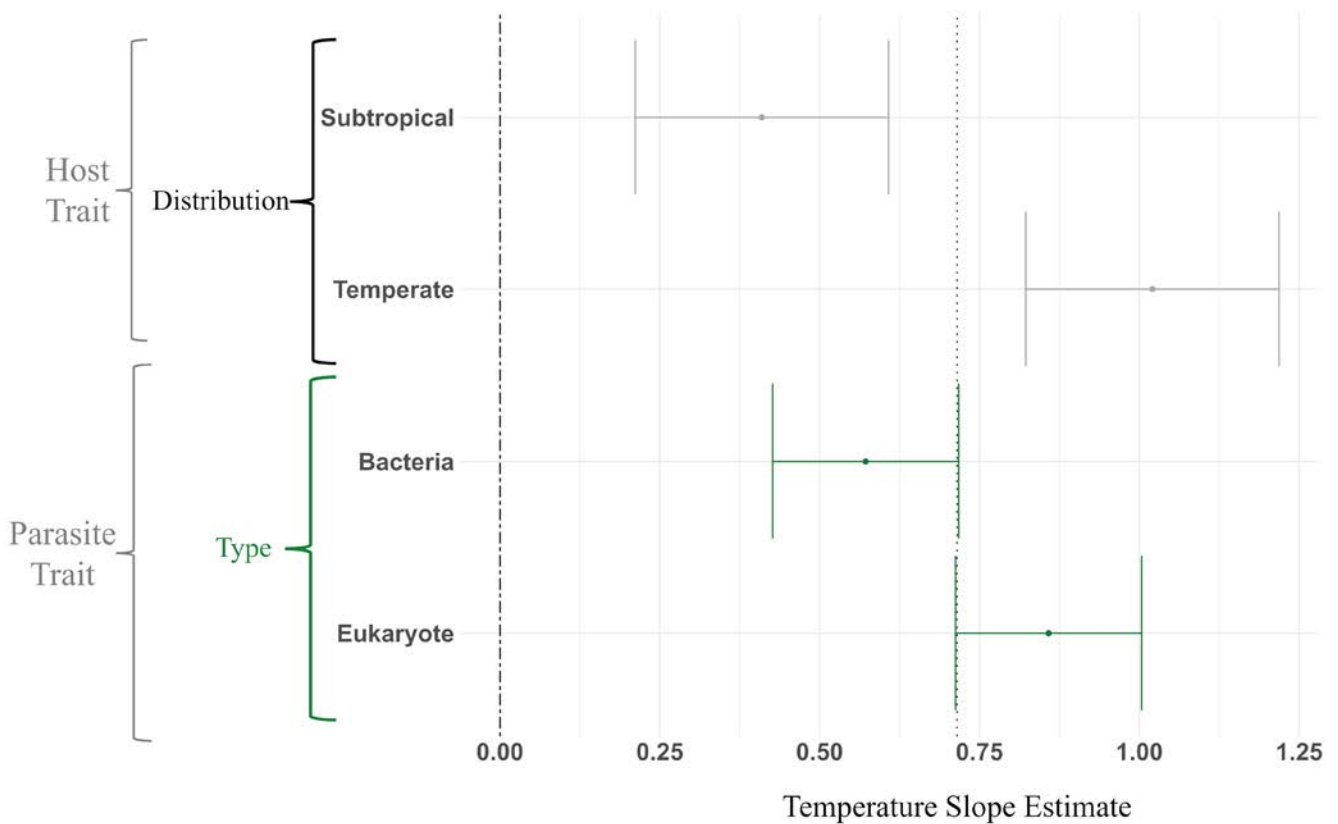


**FIGURE 3** | Interaction effect estimates between temperature and host, parasite, or study design traits of the best fit Trait Models, Trait Model 2 and 3 (TM2 and TM3), for the full dataset. The dots depict the estimates (and 95% confidence intervals) of the main effect of Temperature ( $\beta_T$ ) plus the interactions between each factor and Temperature ( $\beta_{Tj}$  in Equation 3). The dotted vertical line (TM2=0.203; TM3=0.189) is the estimate of the mean increase in parasite-induced host mortality (i.e., log odds ratio; LOR) with increasing temperature. If the 95% confidence interval for a factor level estimate crosses the dotted line, the estimate does not significantly differ from the mean. The dot-dashed vertical line delineates a zero slope estimate where estimates to the right of this line indicate a positive relationship with that factor level and Temperature. Refer to Tables S2 and S3 for more details.

(TM2:  $\beta_T=0.203$ ,  $z=3.865$ ,  $p<0.001$ ; TM3:  $\beta_T=0.189$ ,  $z=3.607$ ,  $p<0.001$ ; Tables S2 and S3). Duration was also negatively associated with LOR in both models (TM2:  $\beta_D=-0.012$ ,  $z=-1.979$ ,  $p=0.048$ ; TM3:  $\beta_D=-0.012$ ,  $z=-2.031$ ,  $p=0.042$ ), and there was also a significant negative interaction between Duration and Temperature in both models (TM2:  $\beta_{TD}=-0.002$ ,  $z=-1.884$ ,  $p=0.06$ ; TM3:  $\beta_{TD}=-0.002$ ,  $z=-2.072$ ,  $p=0.038$ ; Tables S2 and S3), suggesting that effects of temperature on parasite-induced mortality are greater in shorter experiments. The Motivation of the researchers (climate change, temperature correlation, other) significantly altered the relationship between parasite-induced host mortality and Temperature in TM2 and 3 (Figure 3). Studies motivated by investigating climate change impacts on host-parasite dynamics found a smaller-than-average effect of temperature on parasite-induced mortality (TM2:  $\beta_{T, \text{Climate change}}=-0.121$ ,  $z=-2.352$ ,  $p=0.019$ ; TM3:  $\beta_{T, \text{Climate change}}=-0.147$ ,  $z=-2.59$ ,  $p=0.01$ ). Studies motivated by exploring effects other than climate change or a documented temperature correlation on host-parasite interactions had a significantly greater increase in LOR with Temperature compared to the average (TM2:  $\beta_{T, \text{Other}}=0.107$ ,  $z=2.674$ ,  $p=0.007$ ; TM3:  $\beta_{T, \text{Other}}=0.125$ ,  $z=2.775$ ,  $p=0.006$ ). Additionally, model TM3 found a marginally significant interaction between host Life Stage and Temperature, with adult hosts experiencing

greater increases in parasite-induced mortality with temperature than juveniles ( $\beta_{T, \text{Adult}}=0.066$ ,  $z=1.678$ ,  $p=0.093$ ,  $\beta_{T, \text{Juvenile}}=-0.066$ ,  $z=-1.678$ ,  $p=0.093$ ).

Parasite Type and Strategy significantly interacted with Temperature in both models (Figure 3). Bacterial parasites were less likely to cause increases in host mortality with increasing temperature than average (TM2:  $\beta_{T, \text{Bacteria}}=-0.208$ ,  $z=-3.345$ ,  $p=0.001$ ; TM3:  $\beta_{T, \text{Bacteria}}=-0.202$ ,  $z=-3.284$ ,  $p=0.001$ ), while eukaryotic and viral parasites were more likely to increase host mortality with temperature (TM2:  $\beta_{T, \text{Eukaryote}}=0.112$ ,  $z=2.384$ ,  $p=0.017$ ,  $\beta_{T, \text{Virus}}=0.095$ ,  $z=1.755$ ,  $p=0.079$ ; TM3:  $\beta_{T, \text{Eukaryote}}=0.098$ ,  $z=1.977$ ,  $p=0.048$ ,  $\beta_{T, \text{Virus}}=0.104$ ,  $z=1.851$ ,  $p=0.064$ ). This model also suggests that opportunistic parasites, that is, parasites that can become pathogenic but otherwise exist non-pathogenically as part of the host and/or environment microbiome (Brown et al. 2012; Burge et al. 2013), are more likely to produce an increase in parasite-induced mortality with rising temperature (TM2:  $\beta_{T, \text{Opportunistic}}=0.116$ ,  $z=2.268$ ,  $p=0.023$ ; TM3:  $\beta_{T, \text{Opportunistic}}=0.115$ ,  $z=2.257$ ,  $p=0.024$ ), while obligate parasites are less likely to increase parasite-induced mortality with temperature than average (TM2:  $\beta_{T, \text{Obligate}}=-0.116$ ,  $z=-2.268$ ,  $p=0.023$ ; TM3:  $\beta_{T, \text{Obligate}}=-0.115$ ,  $z=-2.257$ ,  $p=0.024$ ).



**FIGURE 4** | Interaction effect estimates between temperature and host, parasite, or study design traits for the best fit model of the Salmoniformes Dataset (STM1). The estimates, plus 95% confidence intervals, are displayed including the main effect of Temperature ( $\beta_T$ ). The dotted vertical line delineates the estimate of the overall mean slope (i.e.,  $\beta_T=0.715$ ) between temperature and log odds ratio. Log odds ratio reflects the difference in mortality between treatment and control hosts. If the 95% confidence interval crosses this line, the interaction effect of that factor level does not deviate significantly from the average relationship. The dot-dashed vertical line delineates a zero slope estimate where estimates to the right of this line indicate a positive relationship with that factor level and Temperature. Refer to Table S4 for more details.

### 3.3 | Salmoniformes Trait Models

Our null model of the Salmoniformes dataset indicated a similar level of parasite-induced mortality in the Order Salmoniformes as in the full dataset ( $\beta_0 = 2.93$ , CI =  $\pm 0.75$ , PI = 2.79). We also found a similar level of sampling variance in the Salmoniformes dataset (1.51), but a smaller amount of variance in the true effect estimate (1.88). Calculating  $I^2$  indicated that 55.49% of the total variance was due to variance in the true effects and partitioned 33.09% of that variance between Studies, 22.41% between effect sizes, and 0% between Experiments. The best fit mixed-effect model again estimated the main effect of Temperature as positive, and that the moderating variables, while capable of changing the value of the slope estimate between Temperature and LOR, always maintained a positive relationship (see below). These results suggest that hosts in the Order Salmoniformes are likely to experience increased parasite-induced mortality with temperature regardless of other moderating variables.

In general, fitting the mixed-effect models to the *Salmoniformes* dataset resulted in slightly higher estimates of Pseudo- $R^2$  than those produced by fitting mixed-effect models to the full dataset (McFadden's Pseudo- $R^2 = 0.16$ ; Nakagawa's Marginal  $R^2 = 0.47$ , Conditional  $R^2 = 0.88$ ; Figure 4, Table S4). Only one model

was selected as the best fit model (STM1), and all other models were  $> 2$  AICc points from this best fit model. When backwards elimination was applied to STM1 the AICc scores rose sharply, therefore the full model was retained. This model found similar effects of Temperature, Duration, and the interaction between Temperature and Duration as the models of the full dataset, except that the estimates for these coefficients were larger in STM1 ( $\beta_T = 0.715$ ,  $z = 3.898$ ,  $p < 0.001$ ;  $\beta_{\text{Duration}} = -0.06$ ,  $z = -2.979$ ,  $p = 0.003$ ;  $\beta_{T, \text{Duration}} = -0.009$ ,  $z = -3.179$ ,  $p = 0.001$ ; Table S4). The STM1 found a significant interaction between host Distribution and Temperature, with temperate hosts suffering significantly higher mortality with increasing temperature ( $\beta_{T, \text{Temperate}} = 0.305$ ,  $z = 3.022$ ,  $p = 0.003$ ), and subtropical hosts experiencing lower mortality with increasing temperature ( $\beta_{T, \text{Subtropical}} = -0.305$ ,  $z = -3.022$ ,  $p = 0.003$ ). Finally, bacterial parasites produced a smaller than average increase in parasite-induced mortality with temperature ( $\beta_{T, \text{Bacteria}} = -0.143$ ,  $z = -1.925$ ,  $p = 0.054$ ), and eukaryotic parasites produced a higher than average increase ( $\beta_{T, \text{Eukaryote}} = 0.143$ ,  $z = 1.925$ ,  $p = 0.054$ ). It is important to note that while bacterial parasite and subtropical hosts produced a lower-than-average increase to parasite-induced mortality with temperature, when added to the main effect of temperature the relationship remains positive (Figure 4).

### 3.4 | Publication Bias

Our analysis indicated a significant effect of small studies on our results. In the full dataset, we found that studies with small precision correspond to large estimates of LOR (Estimate = 0.965,  $z = 3.151$ ,  $p = 0.002$ ; Figure S2). This could lead to the over-estimation of the magnitude of parasite-induced mortality. In fact, including the effect of standard error in the null model of the full dataset reduces the overall mean LOR ( $\beta_0$ ) from 3.122 (CI =  $\pm 0.43$ ,  $z = 14.02$ ,  $p < 0.001$ ) to 1.729 (CI =  $\pm 0.957$ ,  $z = 3.542$ ,  $p < 0.001$ ). The Salmoniformes dataset did not contain this same bias, with no relationship between study precision and effect size (Estimate = 0.061,  $z = 0.103$ ,  $p = 0.918$ ; Figure S3). We also did not find a significant effect of publication year in our full dataset, indicating that earlier studies did not contain statistically higher effect size estimates (Estimate =  $-0.011$ ,  $z = -0.358$ ,  $p = 0.72$ ; Figure S3). We did find a marginally significant negative relationship between year and effect size in our Salmoniformes dataset, suggesting that earlier studies tended to have higher effect size estimates (Estimate =  $-0.021$ ,  $z = -0.385$ ,  $p = 0.087$ ; Figure S5). However, incorporating publication year into the null model of the Salmoniformes dataset did not greatly alter the mean LOR estimate (Null model:  $\beta_0 = 2.93$ ,  $z = 7.626$ ,  $p < 0.001$ ; Publication Year Model:  $\beta_0 = 2.961$ ,  $z = 7.271$ ,  $p < 0.001$ ).

## 4 | Discussion

We conducted a multi-taxon meta-analysis that quantified the influence of temperature as well as host, parasite, and study traits on parasite-induced host mortality. Parasites had a marked effect on their hosts, significantly increasing their mortality; however, the magnitude of the effect was variable across taxa. Warmer temperatures generally increased the parasite-induced mortality rate, but some systems were more sensitive to increases in temperature than average (e.g., Salmoniformes and opportunistic parasites), while other hosts may have benefitted from increased temperatures (e.g., those infected with bacterial parasites). The Salmoniformes-only analysis additionally identified host Distribution as an important factor that changed how temperature affected parasite-induced mortality. Together, this information suggests that while increasing temperature is likely to increase the parasite-induced mortality of most hosts, specific traits of these systems can alter the magnitude of this effect.

In the best fit models of the full dataset (TM2 and 3), parasite traits were identified as significant predictors of temperature–mortality relationships, with important theoretical and management implications for aquatic infectious diseases. For example, mortality risk from opportunistic parasites increased more rapidly with temperature than mortality risk from obligate parasites. Opportunistic parasites may produce increased host mortality with temperature because they may be freed from the virulence–transmissibility trade-off due to their ability to survive outside hosts for extended periods of time. Additionally, the transmission of many opportunistic parasites is not curtailed by host sickness behaviours or the reduction in host density, thus increasing the ability of these parasites to induce further

mortality in host populations (McCallum et al. 2004; Alizon et al. 2009; Brown et al. 2012; Sheppard 2022). The most common infectious diseases in aquaculture are opportunistic parasites (Defoirdt 2016), and the finding that these parasites may increase in severity with temperature should be considered when designing systems to limit stock loss.

Parasite type influenced the magnitude of the relationship between parasitized host mortality and warming, potentially challenging the generality of the assumption that parasites outperform hosts at thermal extremes (Cohen et al. 2017). Bacterial parasites produced a reduced risk of mortality in their parasitized hosts in contrast to the predictions of increased bacterial reproductive rates in warmer temperatures (Harvell et al. 2002). One possible explanation is that the expression of virulence genes in many aquatic bacterial pathogens is reduced at higher temperatures (Guijarro et al. 2015; Ashrafi et al. 2018; Pattanayak et al. 2020), and thus parasitized host mortality may not increase under warming. In contrast, eukaryotic parasites, increasingly being recognised as significant causes of mortality in fisheries (Stentiford et al. 2013; Lafferty 2017), increased parasite-induced mortality with temperature. In our dataset, the eukaryotic parasites were primarily microsporidians (Table S4), which are typically resilient to environmental stressors and can persist in a wide range of environments (Sudhagar et al. 2020; Bojko et al. 2022). This may allow these parasites to outcompete host defences at warmer temperatures and induce host mortality. Consistent with prior work (Mordecai et al. 2019), complex and interacting physiological responses to temperature in diverse host–parasite pairings highlight the importance of accounting for parasite traits when predicting the effects of warming on host–parasite interactions.

Our models further revealed that hosts experienced less parasite-induced mortality as duration increased and that longer experiments reduced the effect of increasing temperature on parasite-induced mortality. We attribute this finding to experimental practicalities. If a parasite exhibits strong effects on host mortality, and if these effects are accentuated at warmer temperature, there is no need to extend the duration of the experiment to produce a significant difference in treatments (also refer to Osenberg et al. 1999 for a related argument about predation studies).

Our analysis also indicated that host taxonomy was a crucial determinant of temperature–infection mortality relationships, with the order Salmoniformes being significantly more sensitive to increases in parasite-induced mortality than average. Salmoniformes typically dwell in cooler water and are known to be particularly sensitive to warm temperatures (Jonsson 2023). Our results suggest that parasite-induced mortality may play a role in higher mortality rates of salmonids, even at temperatures normally considered inside the fishes' physiological thresholds. Our analysis restricted to this host order was able to explain more variation and identified an additional moderator of the temperature–mortality relationship than did our analysis of the full dataset. Effects of temperature were greater on Salmoniformes from temperate regions. Temperate fish species are known to have lower thermal critical maxima than fish from tropical or subtropical regions (Britton et al. 2010; Nati et al. 2021). Thus, as hosts approached thermal maxima, their tolerance for parasite infection is likely diminished, resulting

in increased parasite-induced mortality with temperature (Scharsack and Franke 2022; Tye et al. 2022).

We also found that Mytilida (mussels) were less sensitive to increases in parasite-induced mortality with temperature than average. Members of the order Mytilida, many of which are intertidal and thus exposed to a wide diel range in temperature, have demonstrated remarkable resilience to high temperatures, surviving laboratory temperatures up to 38°C (Barrett et al. 2022; Tan et al. 2023). The high thermal tolerance of these organisms likely allows for immune function to be maintained at a wide range of temperatures, leading to the observed pattern of resistance to increased parasite-induced mortality at warm temperatures.

Our results revealed the need for more comprehensive experiments evaluating the effects of temperature on fisheries host–parasite dynamics. We were unable to examine changes in mortality over the course of an experiment, which would require time-series data that follow the fate of individual hosts (Fox and Weisberg 2011). This information was not available in any of the experiments included in our study. Additionally, a minority of studies utilised > 2 temperatures ( $n = 13$ , Appendix S2), a requirement to capture any non-linear effects (Gehman et al. 2018). Our data were also limited by a bias towards juvenile hosts, *Chordata*, captive hosts, and micro-parasites. Indeed, despite the diversity and effects of eukaryotic parasites, several eukaryotic parasite taxa were underrepresented in our analysis (Table S4). Future experiments that follow individuals through time, include multiple temperature treatments that address a wide range of temperatures, expand the range of host and parasite species, and ensure the parameters of experiments are biologically relevant could help alleviate these gaps.

#### 4.1 | Conclusions and Implications for Real-World Host–Parasite Interactions

We conclude that increased temperature is likely to increase parasite-induced host mortality on the individual host level in most fisheries; however, other factors also influence host population and infection dynamics, especially at larger scales. For example, population- or community- level factors could limit the total number of hosts infected or the mortality of infected hosts (e.g., via preferential predation on infected hosts; Alizon et al. 2009; Wood et al. 2010; Byers 2021). Indeed, the effect of temperature on these scales can oppose the direct effect that temperature has on host–parasite interactions. For example, if altered temperature regimes cause declines in host populations, a host-specific parasite population is also likely to decline even if the nature of the interaction between the host and parasite is unaffected (Lafferty and Holt 2003; Wood et al. 2023). Indeed, low densities of hosts are likely to interrupt transmission chains for many types of parasites and ultimately reduce disease prevalence and effects (Wood and Lafferty 2015). However, increased connectivity of aquatic systems driven by anthropogenic movement of infectious particles and hosts may alleviate this constraint on transmission by connecting isolated host populations and allow for large, deadly epidemics to manifest with warming temperatures (Sieracki et al. 2014; Shore and Caldwell 2019; Kao et al. 2021).

Our results are particularly relevant for aquaculture. Many aquaculture facilities have high densities of hosts that are commonly affected by high mortality events caused by opportunistic parasites, and our analysis suggests that these systems could experience increased losses due to parasite-induced mortality with warming temperatures. Additionally, these facilities can act as a source of infectious particles to wild populations, allowing for highly pathogenic parasites to cause declines in vulnerable wild populations (Costello 2009). This result is particularly relevant for Salmoniformes, which are more vulnerable to warmer temperatures, face declining wild populations, and are experiencing the effects of parasite exposure from aquaculture to wild systems (Krkošek et al. 2005; Ford and Myers 2008). In summary, while many factors may influence the course of epidemics in situ, our analyses suggest that increased temperatures will generally result in a higher likelihood of parasitized host mortality at the individual scale, thus highlighting the complex nature of understanding host–parasite interactions in a warming world.

#### Author Contributions

Megan M. Tomamichel contributed to the study design, data collection and cleaning, performed the meta-analysis, generated all figures and tables and wrote the first draft of the manuscript. Kaitlyn I. Lowe contributed substantially to data collection and cleaning, Kaylee M.H. Arnold also contributed to data collection, generated Appendix S2 and contributed to manuscript edits. Marc E. Frischer contributed to study design and manuscript editing. Brian J. Irwin contributed to data analysis methodology and manuscript editing. Craig W. Osenberg, Richard J. Hall and James E. Byers contributed to study design, data analysis methodology, figure, table and manuscript editing. Richard J. Hall and James E. Byers are co-senior authors and contributed equally to this manuscript.

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#### Data Availability Statement

All data used in this manuscript and code written to conduct analysis and generate figures have been deposited in Dryad and made publicly available; DOI: [10.5061/dryad.4j0zpc8jx](https://doi.org/10.5061/dryad.4j0zpc8jx).

#### Peer Review

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### Supporting Information

Additional supporting information can be found online in the Supporting Information section.

## **Warmer is Deadlier: A meta-analysis reveals increasing temperatures accentuate disease impacts on fisheries hosts.**

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### **APPENDIX S1: Data Collection and Compilation Details**

Any use of trade, firm, or product names is for descriptive purposes only and does not imply endorsement by the U.S. Government.

#### *Data collection*

Between August and November of 2019, we searched Web of Science using the following search terms, returning 1,201 results:

((fish\* OR shellfish\* OR salmonid\* OR oyster\* OR mussel\* OR shrimp\* OR lobster\* OR crab\* OR clam\* OR echino\* OR urchin\* OR octopus\*) AND (disease\* OR parasite\* OR pathogen\* OR infect\* OR bacteria\* OR virus\* OR fung\* OR microsporidia\* OR protozoa\* OR monogea\* OR copepod\* OR coccidia\* OR trematod\* OR cestod\* OR nematod\*) AND (temperature\*))

AND(experiment\*) NOT ("fish oil" OR technology OR medicine OR probiotic\* OR cardio\* OR heart\* OR food\* OR bacteriophage\* OR amphibia\* OR fly\* OR poultry\* OR dair\* OR nitrificat\* OR fruit\* OR compost\* OR spoil\* OR cow\* OR pig\* OR histamine\* OR refrigerat\* OR chick\*))

These search terms were chosen to be inclusive of all harvested aquatic species and their parasites and pathogens, while excluding research on humans, domestic animals, or environmental health.

The abstracts of these 1,201 papers were then screened, which led to the retention of 56 papers that satisfied four criteria.

- 1) Papers had at least one parasite or pathogen treatment paired with an unexposed control (no parasite exposure) at the same temperature. We included papers with naturally exposed hosts provided that the papers also included unexposed hosts from either a captive or an otherwise unexposed population. We did not require papers to confirm infection in their parasite-exposed treatments. Several parasites require destructive sampling methods to confirm infection or are difficult to confirm post-mortem which would interfere with the estimation of the mortality effects of the parasite.
- 2) Hosts were held at a relatively constant temperature (and not explicitly varied), and this temperature was reported in the paper.
- 3) Hosts constituted a wild fishery. This included species that are harvested from the wild for human or animal consumption (e.g., European perch, *Perca fluviatilis*) and species that were historically harvested for consumption but are now primarily

- produced in aquaculture for food (e.g. Nile tilapia, *Oreochromis niloticus*) or ornamental industries (e.g., koi, *Cyprinus rubrofuscus*).
- 4) Studies reported survival or mortality (e.g., cumulative survival, Kaplan Meier curves, daily mortality rate) for each treatment, so that we could extract the number of hosts that lived and died under the two parasite treatments (which was used to calculate the log odds ratio (LOR, Eqn 1). We focused on mortality because it is comparable between studies, while metrics of disease severity, such as lesion scores, viral titers, or individual parasite counts are challenging to standardize among different systems.
  - 5) A taxonomic Order was well-represented in our dataset (at least two experiments with more than one temperature).

#### *Moderating Factors and Factor Levels*

We recorded moderator variables that potentially explained variation in the effects of parasites and temperature. Moderators were categorized into host, parasite, or study traits (Main Text Table 1). These moderators were either taken directly from the paper that reported the experiments or were obtained from other sources (e.g., government reports or scientific publications which provided details on the life history of the parasite; Refer to Appendix S2 for more details).

**Host traits** included host phylogeny, host physiology, and characteristics of the host's local environment. We examined the influence of host phylogeny (Phylum, Class and Order) on the relationship between temperature and parasite-induced mortality because the host's susceptibility to different parasites and their response to temperature change may be influenced by host phylogeny (Litman *et al.* 2005). Other host traits included:

- *Mobility*. Hosts were categorized as “immobile” if their adult stages do not swim in the water column, and “mobile” if the adult stages do swim in the water column.
- *Vertebrae*. Hosts were categorized as “vertebrate” (or “invertebrate”) if their adult stages have (or do not have) a spinal column.
- *Life stage*. Hosts were categorized as “juvenile” or “adult” based on either authors’ identification or comparing the reported size of the hosts in the experiment and the mean length or weight at sexual maturity.
- *Distribution*. Host’s natural or naturalized distribution was categorized as either “tropical”, “subtropical”, or “temperate” according to the range information documented on FishBase or other supporting research.
- *Salinity*. Host’s natural or naturalized salinity tolerance was categorized according to the environment information available on FishBase. Hosts were categorized as “freshwater” if their environment information did not include any saline water (e.g., did not include brackish or marine), as “mixed” if either categorized by FishBase as brackish or a mix of freshwater and saline habitats, and as “marine” if only marine and no freshwater or brackish habitats were listed.

These host traits may influence the effect of temperature on parasite-induced host mortality relationship because of their influence on host behavior and immunological defenses. Adult hosts may prioritize energy expenditures towards the production of gametes rather than towards a robust immunological response to a parasite (Buckingham *et al.* 2023), therefore making adult hosts more susceptible to infection and potentially to an increased parasite-induced mortality rate with temperature. Immobile hosts are unable to seek refuge from biotic and abiotic

stressors in their natural environment, potentially selecting for hosts that are better able to tolerate infection and temperature change than mobile hosts (Ashapkin *et al.* 2020; Sears *et al.* 2015). Invertebrate hosts have a different immune system than vertebrate hosts, and this may differentially affect their susceptibility to mortality and temperature (Litman *et al.* 2005). A host's distribution and therefore ability to tolerate changes in temperature may be influenced by its evolutionary history with the environment as dictated by its endemic temperature range (Seebacher *et al.* 2015). A host's distribution might also influence the probability of a subsequent thermal mismatch with the parasite (Cohen *et al.* 2017). Salinity can influence a host's immunological response, and the ability of a host to tolerate a wide range of salinities may influence its physiological and immunological response to temperature and parasite infection (Shields 2019).

**Parasite traits** included information about a parasite's physiology, infection mode, and evolutionary strategies. We define a parasite as an organism that lives in or on another organism (the host) and derives its energy from the host. This definition includes both micro-parasites (virus, bacteria, microsporidians, ciliates, etc.,) and macro-parasites (nematodes, arthropods, etc.,). The parasite traits were:

- *Location*. Parasites were categorized as “external” if they infected the gills or skin of a host and “internal” otherwise.
- *Strategy*. Parasites were characterized as “opportunistic” if they can be found as a part of the normal fauna of the environment and/or only cause pathology in otherwise compromised hosts and “obligate” otherwise.
- *Type*. Parasite Type categorizes parasites as a “virus”, a “bacteria”, or a “eukaryote”.

- *Transmission.* Parasite transmission was categorized as “direct” if an infectious particle was only transmitted through the water column during a limited period of time (<10 days). Transmission was categorized as “ingestion” if a host could also become infected by consuming contaminated tissue, in addition to being directly transmitted. “Indirect transmission” was defined as parasites that were also capable of infecting hosts either after an extended period outside of a host or through vertical transmission in addition to direct transmission.

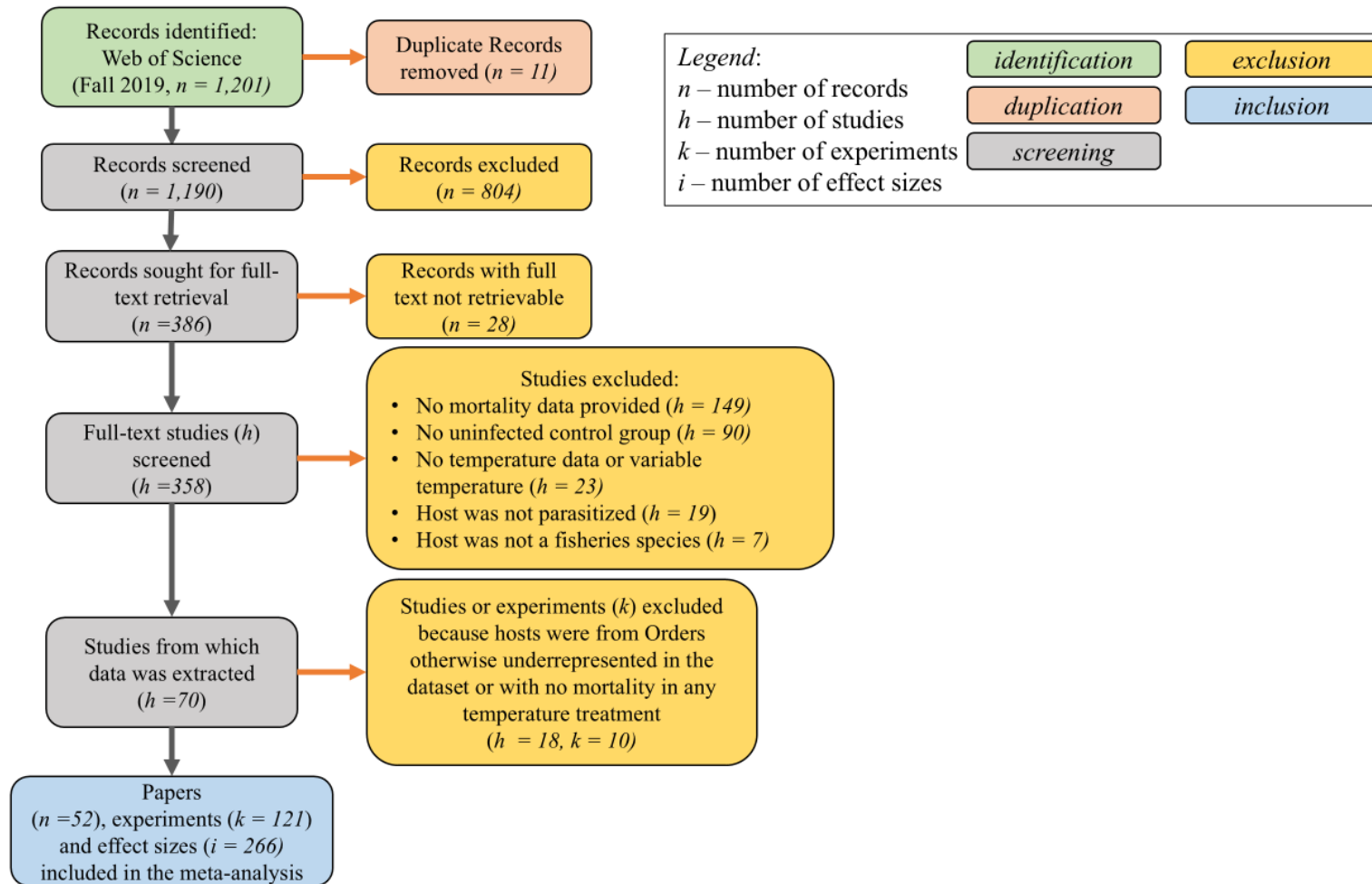
Parasite traits may differentially influence a parasite’s thermal performance and thus the consequences of host-parasite interactions. Parasites were categorized as having either an internal or external location because external parasites might be more severely affected by temperature due to their greater exposure to environmental conditions. Opportunistic parasites may have a different evolutionary relationship with their hosts than obligate parasites, particularly in reference to classic virulence trade-off theory (Alizon *et al.* 2009), thus allowing opportunistic parasites to cause greater host mortality with increasing temperatures. Additionally, an opportunistic parasite may have a different response to temperature because of this extended external or dormant life history strategy. Parasite Type is relevant to the parasite-induced mortality and temperature relationship. Bacterial parasites in particular are thought to be particularly sensitive to temperature because the reproductive rate of bacteria generally accelerates with temperature (Harvell *et al.* 2002). Finally, parasites with an extended external transmission (indirect transmission) may be more resistant to environmental variation than those with more direct transmission modes (direct or ingestion; Byers 2021).

**Study traits** included features of the study’s design that likely influenced the detected relationship between parasite induced host mortality and temperature. They included:

- *Duration* [continuous from 2 – 350]. The number of days an experiment was performed.
- *Host Source*. We categorized the host source as “captive” if the hosts came from a laboratory breeding or aquaculture production facility; “wild” if animals were collected from the natural environment; and “unknown” if the paper did not describe the source of their animals.
- *Motivation*. We classified studies based upon the rationale for the study or based on the explanation of their results, as articulated in the introductions or discussions of the papers: “Climate change” was used if the study was motivated by climate change; “Temperature Correlation” if the study was motivated by seasonal patterns of infection or other observed correlations between temperature and infection, and “other” if the paper did not articulate a rationale or if another rationale was provided.

The choices experimenters made over the course of their study may have influenced the parasite-induced mortality rate and the subsequent response to temperature. For each study we recorded the experimental duration because risk of mortality should increase with exposure time. Hosts that originate from a captive or wild population may differentially respond to parasite-induced mortality and temperature because wild hosts have potentially been exposed to a range of parasites and environmental conditions during their ontogeny and evolutionary history, and thus wild hosts may be more resilient towards parasite-induced mortality. Finally, if a study was performed to test if changing climatic conditions will alter host-parasite relationships, it may be more likely that the resulting relationship confirms this hypothesis compared to studies that were

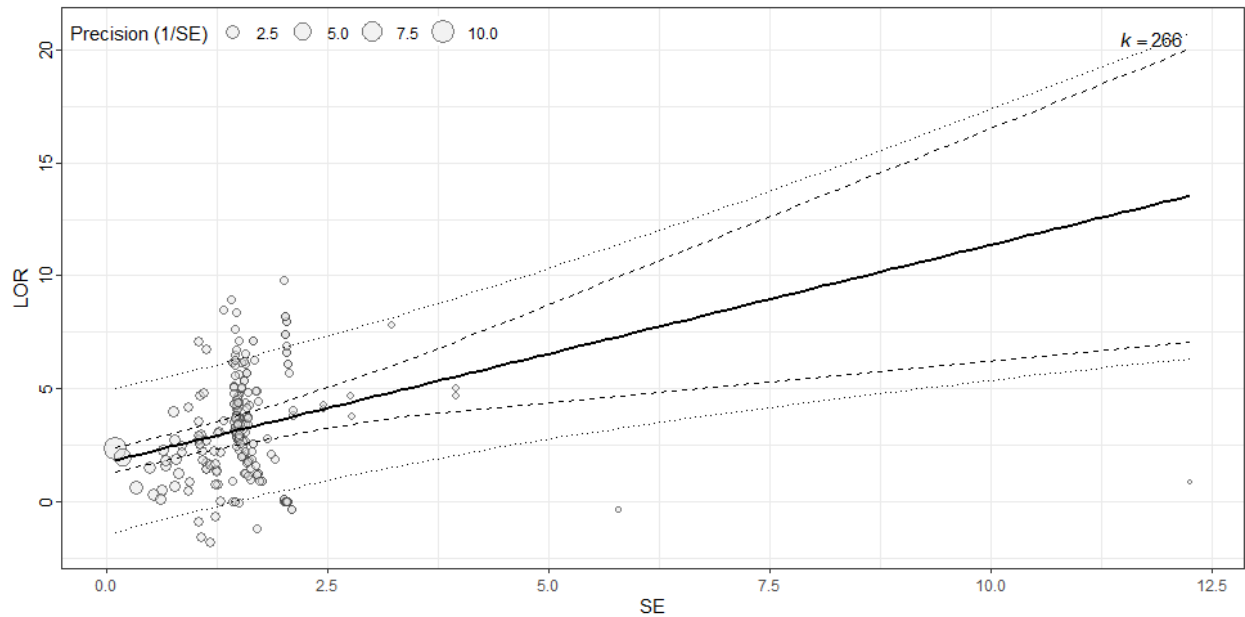
testing only a documented temperature correlation (e.g., a seasonal increase in parasite prevalence) or had a motivation unrelated to temperature and host-parasite relationships.



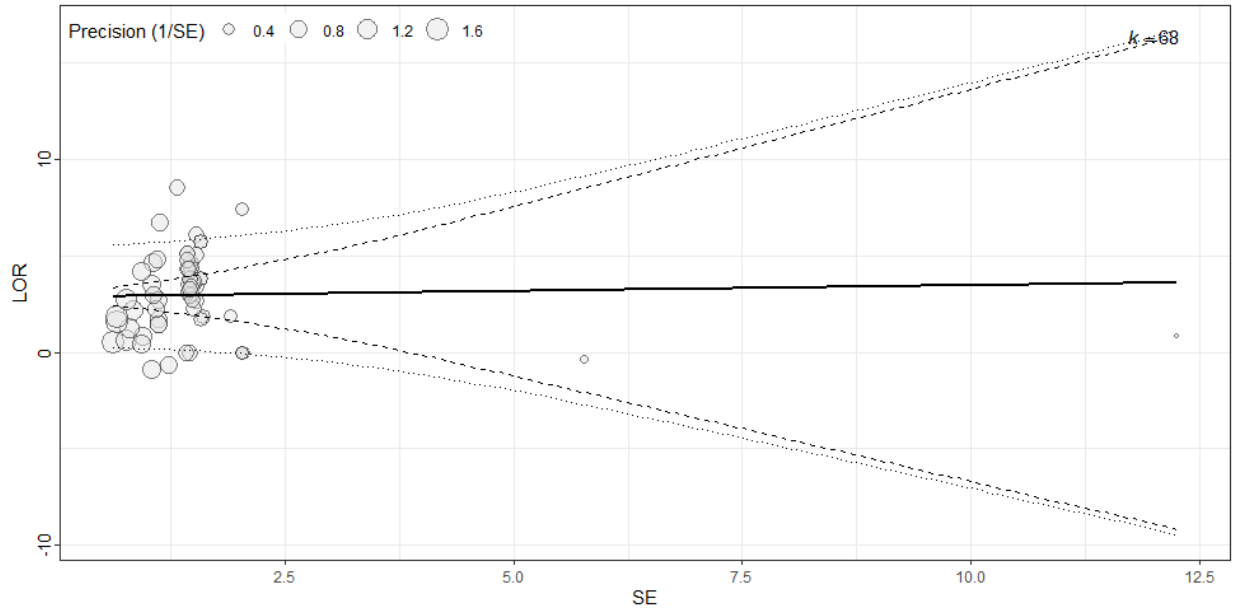
**FIGURE S1:** PRISMA Diagram. This diagram describes the record identification, screening, and exclusion procedures for our meta-analysis in accordance with PRISMA protocols (Foo *et al.* 2021).

**APPENDIX S2:** Data source material. This table lists the data source papers for the effect sizes used in our meta-analysis, as well as reference information for additional host and parasite traits that were not detailed in the original paper (Refer to Excel file, references in Appendix S5.)

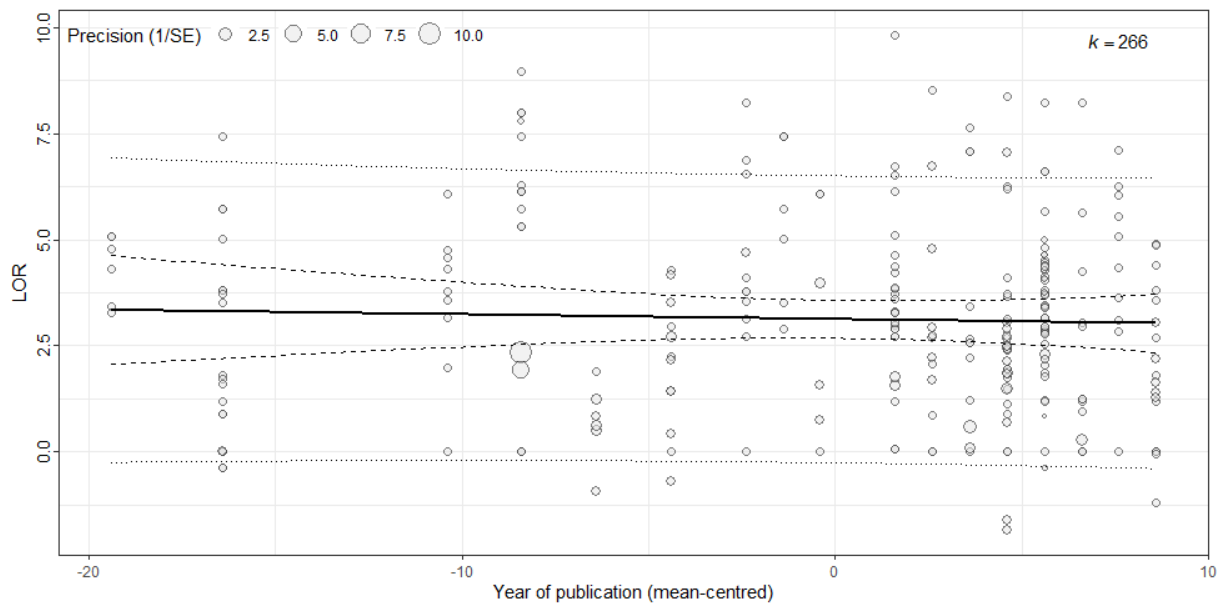
**APPENDIX S3:** Evaluating publication bias. We evaluated publication bias following the procedure outlined in (Nakagawa *et al.* 2022), specifically equations 21 and 22. We first calculated the standard error of the log odds ratios included in our dataset, and then built a mixed-effects model (*rma.mv* function, *metafor* package; Viechtbauer 2010) using the random effect structure described in the main text standard error as a fixed effect. We found a significant positive relationship between standard error and log odds ratio in our full dataset (Estimate = 0.965,  $z = 3.151$ ,  $p = 0.002$ , Nakagawa's Marginal  $R^2 = 20.64\%$ , Conditional  $R^2 = 65.83\%$ ), indicating that studies with greater variance tended to produce larger estimates of log odds ratio (Figure S2). We did not find a significant relationship between standard error and log odds ratio in our Salmoniformes dataset (Estimate = 0.061,  $z = 0.103$ ,  $p = 0.918$ , Nakagawa's Marginal  $R^2 = 0.41\%$ , Conditional  $R^2 = 59.22\%$ ), demonstrating that there is little evidence of small studies unduly influencing our results in the Salmoniformes model (Figure S3). We also evaluated if the size of our effect sizes trended with publication year using the same procedure outlined above. We found little evidence that publication year was related to log odds ratio estimates in the full dataset (Estimate = -0.011,  $z = -0.358$ ,  $p = 0.72$ , Nakagawa's Marginal  $R^2 = 0.25\%$ , Conditional  $R^2 = 54.93\%$ , Figure S4), but a marginally significant effect in the Salmoniformes dataset (Estimate = -0.021,  $z = -0.385$ ,  $p = 0.087$ , Nakagawa's Marginal  $R^2 = 1.45\%$ , Conditional  $R^2 = 63.27\%$ , Figure S5). Therefore, we conclude that our effect sizes in the full dataset were not biased from larger estimates in earlier publications, but there may be a slight bias in the Salmoniformes dataset.



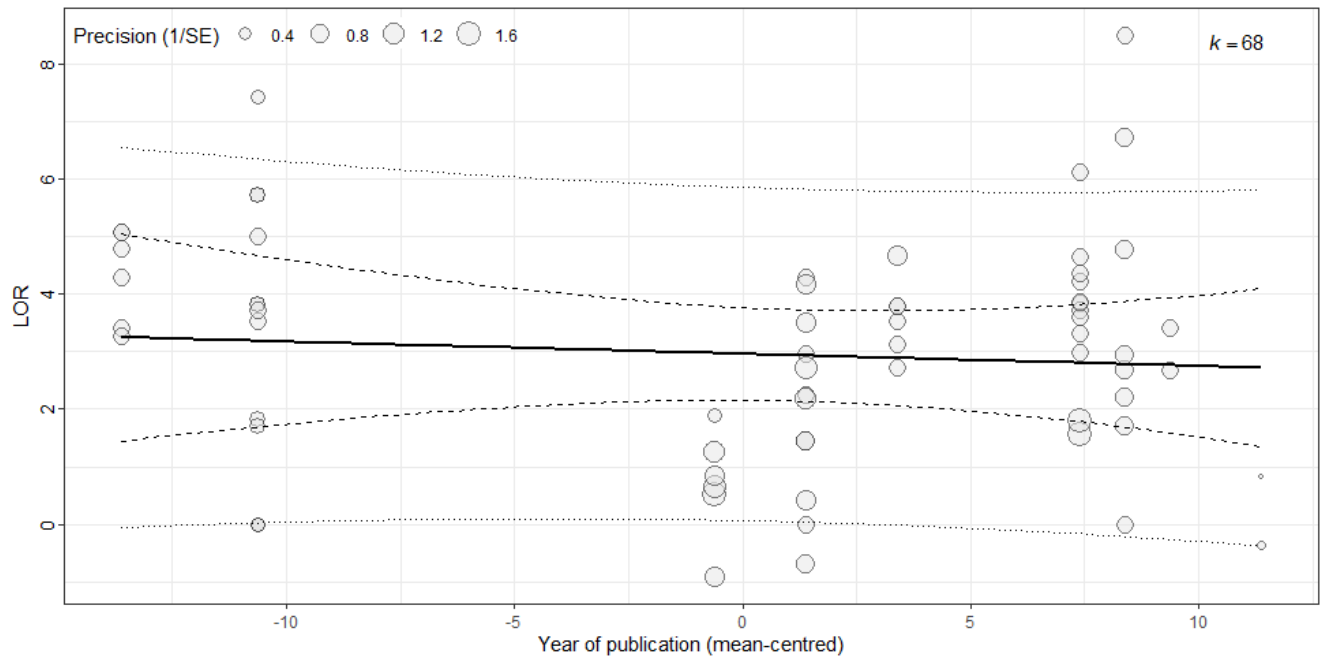
**FIGURE S2:** The relationship between Standard Error (SE) and Log Odds Ratio (LOR) in the full dataset. Plotted are the results of our publication bias evaluation for the full dataset, where the solid line displays the mean relationship between SE and LOR, the dashed line is the 95% confidence interval, and the dotted line is the 95% prediction interval. Circles correspond to the precision estimate, where larger circles represent more precise estimates of LOR.



**FIGURE S3:** The relationship between Standard Error (SE) and Log Odds Ratio (LOR) in the Salmoniformes dataset. Plotted is the mean relationship between SE and LOR (solid line) alongside the confidence interval (dashed) and prediction interval (dotted). Larger circles correspond to the LOR estimates with higher precision.



**FIGURE S4:** The relationship between publication year and Log Odds Ratio (LOR) in the full dataset. The mean relationship between mean-centered publication year (solid line), including confidence interval (dashed line) and prediction interval (dotted line), displays no significant trend. The circles represent the precision of the LOR estimates included in the dataset, where larger circles indicate more precise estimates.



**FIGURE S5:** Mean centered year of publication and Log Odds Ratio (LOR) relationship in the Salmoniformes dataset. The mean relationship between these two variables (solid line), the confidence interval (dashed line), and the prediction interval (dotted line), displays a marginally significant trend. Also displayed are the precision estimates of the Log Odds Ratio values as circles, where larger circles correspond to higher estimates of precision.

**TABLE S1:** Model estimates for the best fit Host Taxonomic Model (HTM3). Our Pseudo- $R^2$  metrics demonstrate that this model explains more variation than the null model (McFadden's: = 0.11; Nakagawa's Marginal  $R^2$  = 0.34, Conditional  $R^2$  = 0.64,  $\Delta$ AICc <2). The Main Effect Estimates column provides estimates of the main effect for the model, and the Interaction Effect Estimates column provides the interaction with Temperature effect estimates that are plotted in Figure 2. Significance levels of  $p$ -values are denoted: \* $p$  <0.05 and # $p$  <0.1. For continuous variables, the coefficient indicates the direction of the effect; for categorical variables, the coefficient indicates the difference from the average. AICc= Akaike Information Criterion corrected for sample size.

		Main Effect Estimates				Interaction Effect Estimates			
Moderating Variable	Levels	Estimate	SE	z-value	$p$ -value	Estimate	SE	z-value	$p$ -value
Intercept		3.222	0.377	8.540	<0.001*				
Temperature		0.189	0.048	3.912	<0.001*				
Duration		-0.018	0.007	-2.484	0.013*	-0.003	0.001	-2.521	0.012*
Order	Cichliformes	-3.155	1.759	-1.794	0.073#	0.039	0.164	0.237	0.813
	Cypriniformes	-1.048	0.758	-1.382	0.167	0.134	0.115	1.172	0.241
	Decapoda	0.489	0.869	0.562	0.574	0.090	0.082	1.096	0.273
	Mytilida	-1.338	0.793	-1.688	0.091#	-0.168	0.092	-1.837	0.066#
	Ostreidae	0.661	0.573	1.154	0.249	-0.048	0.072	-0.664	0.507
	Perciformes	0.477	0.638	0.748	0.455	-0.084	0.071	-1.176	0.240
	Pleuronectiformes	1.336	0.613	2.180	0.029*	-0.073	0.103	-0.707	0.479
	Salmoniformes	2.011	0.655	3.069	0.002*	0.163	0.084	1.934	0.053#
	Siluriformes	1.322	0.827	1.598	0.110	-0.126	0.095	-1.335	0.182
	Venerida	1.418	0.570	2.488	0.013*	-0.116	0.084	-1.382	0.167

**TABLE S2:** Estimates from one of the best fit models of the full dataset (TM2). Both McFadden’s and Nakagawa’s Pseudo- $R^2$  estimates suggest that this model explains more variation than the null (McFadden’s Pseudo- $R^2 = 0.1$ ; Nakagawa’s Marginal  $R^2 = 0.4$ , Conditional  $R^2 = 0.67$ ,  $\Delta AICc < 2$ ). Both Main effects and Interaction Effects with Temperature (plotted in Figure XX 3A) are displayed. Significance levels of  $p$ -values are denoted: \* $p < 0.05$  and # $p < 0.1$ . For continuous variables, the coefficient indicates the direction of the effect; for categorical variables, the coefficient indicates the difference from the average. AICc= Akaike Information Criterion corrected for sample size.

Moderating Variable	Levels	Main Effect Estimates				Interaction Effect Estimates			
		Coefficient	SE	$z$ -value	$p$ -value	Coefficient	SE	$z$ -value	$p$ -value
Intercept		3.261	0.335	9.722	<0.001*				
Temperature		0.203	0.052	3.865	<0.001*				
Duration		-0.012	0.006	-1.979	0.048*	-0.002	0.001	-1.884	0.06#
Mobility	Immobile	0.083	0.272	0.304	0.761	0.040	0.035	1.164	0.244
	Mobile	-0.083	0.272	-0.304	0.761	-0.040	0.035	-1.164	0.244
Life Stage	Adult	-0.562	0.278	-2.018	0.044*	0.059	0.040	1.459	0.145
	Juvenile	0.562	0.278	2.018	0.044*	-0.059	0.040	-1.459	0.145
Parasite Type	Bacteria	-0.841	0.422	-1.993	0.046*	-0.208	0.062	-3.345	0.001*
	Eukaryote	-0.250	0.347	-0.722	0.470	0.112	0.047	2.384	0.017*
	Virus	1.091	0.397	2.745	0.006*	0.095	0.054	1.755	0.079#
Parasite Strategy	Obligate	-0.273	0.351	-0.778	0.437	-0.116	0.051	-2.268	0.023*
	Opportunistic	0.273	0.351	0.778	0.437	0.116	0.051	2.268	0.023*
Motivation	Climate change	-0.143	0.416	-0.344	0.731	-0.121	0.052	-2.352	0.019*
	Temperature Correlation	-0.097	0.303	-0.318	0.750	0.014	0.036	0.398	0.690
	Other	0.240	0.302	0.793	0.427	0.107	0.040	2.674	0.007*

**TABLE S3:** Estimates from the other best fit model of the full dataset (TM3). This model explains more variation than the null model and is within two AICc points of the other best fit models (McFadden’s Pseudo- $R^2 = 0.1$ ; Nakagawa’s Marginal  $R^2 = 0.4$ , Conditional  $R^2 = 0.67$ ). Figure 3B displays the Interaction effect between each variable and Temperature that are listed below. The Main effect estimates from this model are also displayed. Significance levels of  $p$ -values are denoted: \* $p < 0.05$  and # $p < 0.1$ . For continuous variables, the coefficient indicates the direction of the effect; for categorical variables, the coefficient indicates the difference from the average. AICc= Akaike Information Criterion corrected for sample size.

		Main Effect Estimates				Interaction Effect Estimates			
Moderating Variable	Levels	Coefficient	SE	z-value	p-value	Coefficient	SE	z-value	p-value
Intercept		3.236	0.336	9.633	<0.001*				
Temperature		0.189	0.052	3.607	<0.001*				
Duration		-0.012	0.006	-2.031	0.042*	-0.002	0.001	-2.072	0.038*
Vertebrae	Invertebrate	0.134	0.268	0.502	0.616	0.039	0.035	1.103	0.270
	Vertebrate	-0.134	0.268	-0.502	0.616	-0.039	0.035	-1.103	0.270
Life Stage	Adult	-0.518	0.279	-1.860	0.063#	0.066	0.039	1.678	0.093#
	Juvenile	0.518	0.279	1.860	0.063#	-0.066	0.039	-1.678	0.093#
Parasite Type	Bacteria	-0.859	0.420	-2.048	0.041*	-0.202	0.062	-3.284	0.001*
	Eukaryote	-0.321	0.356	-0.901	0.367	0.098	0.050	1.977	0.048*
	Virus	1.180	0.399	2.956	0.003*	0.104	0.056	1.851	0.064#
Parasite Strategy	Obligate	-0.311	0.353	-0.882	0.378	-0.115	0.051	-2.257	0.024*
	Opportunistic	0.311	0.353	0.882	0.378	0.115	0.051	2.257	0.024*
Motivation	Climate Change	-0.282	0.432	-0.653	0.514	-0.147	0.057	-2.59	0.01*
	Temperature Correlation	-0.025	0.305	-0.082	0.935	0.022	0.037	0.598	0.550
	Other	0.307	0.314	0.977	0.328	0.125	0.045	2.775	0.006*

**TABLE S4:** Estimates from the best fit model of the Salmoniformes dataset (STM1). Both the Main effect estimates from the model as well as the Interaction with Temperature (plotted in Figure XX4) are displayed. Our metrics of Pseudo- $R^2$  indicate that this model fits the data better than the null (McFadden's Pseudo- $R^2 = 0.16$ ; Nakagawa's Marginal  $R^2 = 0.47$ , Conditional  $R^2 = 0.88$ ). Significance levels of  $p$ -values are denoted: \* $p < 0.05$  and # $p < 0.1$ . For continuous variables, the coefficient indicates the direction of the effect; for categorical variables, the coefficient indicates the difference from the average. AICc= Akaike Information Criterion corrected for sample size.

		Main Effect Estimates				Interaction Effect Estimates			
Moderating Variable	Levels	Estimate	SE	$z$ -value	$p$ -value	Estimate	SE	$z$ -value	$p$ -value
Intercept		7.037	1.275	5.520	<0.001*				
Temperature		0.715	0.183	3.898	<0.001*				
Duration		-0.06	0.020	-2.979	0.003*	-0.009	0.003	-3.179	0.001*
Distribution	Subtropical	-1.558	0.695	-2.242	0.025*	-0.305	0.101	-3.022	0.003*
	Temperate	1.558	0.695	2.242	0.025*	0.305	0.101	3.022	0.003*
Parasite Type	Bacteria	-0.601	0.591	-1.017	0.309	-0.143	0.074	-1.925	0.054#
	Eukaryote	0.601	0.591	1.017	0.309	0.143	0.074	1.925	0.054#

**TABLE S5:** Eukaryotic parasite taxa included in our dataset. This table details the number of log odds ratios (LOR) of each eukaryotic parasite phyla to our dataset. It also provides the kingdoms represented by each parasite phyla, the number of papers utilizing each parasite phyla in experiments, the number of experiments with more than one temperature treatment, and the orders of the hosts infected with the parasite phyla. Only experiments with more than one temperature treatment are used to estimate the relationship between temperature and LOR.

Parasite Phylum	Parasite Kingdom	Number LOR	Number of Papers	Number of Experiments with >1 Temperature	Host Orders
Microsporida	Fungi	14	1	1	Salmoniformes
Cnidaria	Animalia	9	3	1	Perciformes, Pleuronectiformes, Salmoniformes
Myxozoa	Chromista	8	3	2	Decapoda, Ostreidae, Venerida
Mucormycota	Fungi	6	1	0	Siluriformes
Myxozoa	Animalia	5	3	1	Salmoniformes
Bigyra	Chromista	3	1	1	Venerida
Choanozoa	Protozoa	3	1	1	Salmoniformes
Ascomycota	Fungi	1	1	0	Decapoda

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Source	Temperatures (°C)*	Host Phylum	Host Class	Host Order	Host Genus	Host Species	Host Mobility	Host Vertebræ	Host Life Stage	Host Distribution	Host Distribution References	Salinity	Parasite	Parasite Location	Parasite Strategy	Parasite Strategy References	Parasite Type	Parasite Transmission	Parasite Transmission References	Study duration **	Host Source	Study Motivation
Antonio & Hedrick 1995	9, 12, 15, 18, 21	Chordata	Actinopterygii	Salmoniformes	<i>Oncorhynchus</i>	<i>tshawytscha</i>	Mobile	Vertebrate	Juveniles	Temperate	Nelson 1994	Mixed	<i>Enterocytozoon salmonis</i>	Internal	Obligate	Chilmanczyk et al. 1991	Eukaryote	Waterborne/Indirect Transmission	Baxa-Antonio et al. 1992	84, 140	UNK	Other
Barris et al. 2018	6, 12, 18	Arthropoda	Malacostraca	Decapoda	<i>Homarus</i>	<i>americanus</i>	Mobile	Invertebrate	Adult	Temperate	Radhakrishnan et al. 2019	Mixed	Epizootic shell disease (ESD)	External	Opportunistic	Cobb & Castro 2006	Bacteria	Waterborne/Indirect Transmission	Cobb 2006	175	Wild	A documented temperature correlation
Becker et al. 2016	20, 20.5	Chordata	Actinopterygii	Perciformes	<i>Perca</i>	<i>fluviatilis</i>	Mobile	Vertebrate	Adult	Temperate	Nelson 1994	Mixed	<i>Ranavirus percaZ</i>	Internal	Obligate	Langdon 1989	Virus	Waterborne/Indirect Transmission	Langdon 1989	35	Wild	Other
Bettge et al. 2009	14, 16, 19	Chordata	Actinopterygii	Salmoniformes	<i>Oncorhynchus</i>	<i>mykiss</i>	Mobile	Vertebrate	Juveniles	Subtropical	Nelson 1994	Mixed	<i>Tetracapsuloides bryosalmonae</i>	Internal	Obligate	Morris & Adams 2006	Eukaryote	Waterborne/Indirect Transmission	Morris & Adams 2006	49	Captive	A documented temperature correlation
Boonthai et al. 2018	11, 23, 28	Chordata	Actinopterygii	Perciformes	<i>Micropterus</i>	<i>dolomieu</i>	Mobile	Vertebrate	Juveniles	Subtropical	Nelson 1994	Freshwater	<i>Ranavirus</i>	Internal	Obligate	Jia et al. 2013	Virus	Waterborne/Indirect Transmission	Brenes et al. 2014	30	Captive	A documented temperature correlation
Castro et al. 2011	15, 18	Chordata	Acanthopterygii	Pleuronectiformes	<i>Scophthalmus</i>	<i>maximus</i>	Mobile	Vertebrate	Juveniles	Temperate	Nelson 1994	Mixed	<i>Edwardsiella tarda</i>	Internal	Obligate	Bullock & Herman 1985	Bacteria	Waterborne/Indirect Transmission	Bullock & Herman 1985	20	Captive	Other
Chen et al. 2003	27	Arthropoda	Malacostraca	Decapoda	<i>Macrobrachium</i>	<i>rosenbergii</i>	Mobile	Invertebrate	Juveniles	Tropical	De Grave et al. 2008	Freshwater	<i>Enterococcus faecium</i>	Internal	Opportunistic	Braiek & Smaoui 2019	Bacteria	Waterborne/Indirect Transmission	Braiek & Smaoui 2019	4	Captive	Other
Chen et al. 2003	27	Arthropoda	Malacostraca	Decapoda	<i>Macrobrachium</i>	<i>rosenbergii</i>	Mobile	Invertebrate	Juveniles	Tropical	De Grave et al. 2008	Freshwater	<i>Metschnikowia bicuspidata</i>	Internal	Opportunistic	Jiang et al. 2022	Eukaryote	Waterborne/Ingestion	Jiang et al. 2022	4	Captive	Other
Cheng et al. 2009	19, 27, 35	Chordata	Actinopterygii	Perciformes	<i>Epinephelus</i>	<i>coioides</i>	Mobile	Vertebrate	Juveniles	Subtropical	Nelson 1994	Mixed	<i>Vibria alginolyticus</i>	Internal	Opportunistic	Baker-Austin et al. 2018	Bacteria	Waterborne/Indirect Transmission	Baker-Austin et al. 2018	5	Captive	Other
Chideroli et al. 2017	22, 31	Chordata	Actinopterygii	Cichliformes	<i>Oreochromis</i>	<i>niloticus</i>	Mobile	Vertebrate	Juveniles	Tropical	Nelson 1994	Mixed	<i>Streptococcus agalactiae</i>	Internal	Opportunistic	Johri et al. 2006	Bacteria	Waterborne/Indirect Transmission	Pradeep et al. 2016	10	Captive	A documented temperature correlation
Chu & Zhu 2010	25	Chordata	Actinopterygii	Cypriniformes	<i>Carassius</i>	<i>gibelio</i>	Mobile	Vertebrate	Juveniles	Temperate	Nelson 1994	Mixed	<i>Aeromonas hydrophila</i>	External	Opportunistic	Swann & White 1991	Bacteria	Waterborne	Swann & White 1991	14	Captive	Other
Dahl et al. 2011	13, 21, 27	Mollusca	Bivalvia	Venerida	<i>Mercenaria</i>	<i>mercenaria</i>	Immobile	Invertebrate	Adult	Subtropical	Saeedi & Costello 2013	Mixed	Quahog Parasite (QPX disease); aka <i>Mucichytrium quahogii</i>	Internal	Obligate	Smolowitz 2018	Eukaryote	Waterborne	Smolowitz 2018	126	Captive	A documented temperature correlation
Demann & Wegner 2018	16, 21	Mollusca	Bivalvia	Mytilida	<i>Mytilus</i>	<i>edulis</i>	Immobile	Invertebrate	Juveniles	Subtropical	Inoue et al. 2021	Mixed	<i>Vibrio splendidus</i>	Internal	Opportunistic	Baker-Austin et al. 2018	Bacteria	Waterborne/Indirect Transmission	Baker-Austin et al. 2018	7	Wild	Other
Deng et al. 2017	23.5	Chordata	Actinopterygii	Cypriniformes	<i>Schizopygopsis</i>	<i>pylzovi</i>	Mobile	Vertebrate	Juveniles	Subtropical	Nelson 1994	Freshwater	<i>Streptococcus agalactiae</i>	Internal	Opportunistic	Johri et al. 2006	Bacteria	Waterborne/Indirect Transmission	Pradeep et al. 2016	14	Captive	Other
Escobedo-Fregoso et al. 2017	26.0	Mollusca	Bivalvia	Ostreidae	<i>Crassostrea</i>	<i>corteziensis</i>	Immobile	Invertebrate	Adult	Tropical	Ladeiras et al. 2020	Marine	<i>Perkinsus marinus</i>	Internal	Obligate	Lau et al. 2018	Eukaryote	Waterborne	Mccollough et al. 2007	15	Captive	A documented temperature correlation
Gilad et al. 2003	13, 18, 23, 28	Chordata	Actinopterygii	Cypriniformes	<i>Cyprinus</i>	<i>carpio</i>	Mobile	Vertebrate	Adult	Tropical	Nelson 1994	Mixed	<i>Cyvirus cyprinidallaZ</i>	Internal	Obligate	Uchii et al. 2013	Virus	Waterborne	Tolo et al. 2021	15, 16, 18, 20, 24, 25, 58	Captive	A documented temperature correlation
Hallett & Bartholomew 2008	12.8	Chordata	Actinopterygii	Salmoniformes	<i>Oncorhynchus</i>	<i>mykiss</i>	Mobile	Vertebrate	Juveniles	Subtropical	Nelson 1994	Mixed	<i>Myxobolus cerebralis</i>	External	Obligate	Chong 2022	Eukaryote	Waterborne/Indirect Transmission	Gilbert & Granath 2003	180	Captive	A documented temperature correlation
Huchin-Mian et al. 2018	4, 20, 25, 30	Arthropoda	Malacostraca	Decapoda	<i>Callinectes</i>	<i>sapidus</i>	Mobile	Invertebrate	Juveniles	Tropical	Magalhães et al. 2016	Mixed	<i>Hematodinium perezii</i>	Internal	Obligate	Chen et al. 2023	Eukaryote	Waterborne	Huang et al. 2021	22, 30, 80, 91	Wild	A documented temperature correlation
Jones et al. 1999	11	Chordata	Actinopterygii	Salmoniformes	<i>Salmo</i>	<i>salar</i>	Mobile	Vertebrate	Juveniles	Temperate and Subtropical	Nelson 1994	Mixed	<i>Isavirus salaris</i>	Internal	Obligate	Kibenge et al. 2004	Virus	Waterborne/Indirect Transmission	Kibenge et al. 2004	24	Captive	Other
Jones et al. 2007	9.3, 14.8	Chordata	Actinopterygii	Salmoniformes	<i>Salvelinus</i>	<i>confluens</i>	Mobile	Vertebrate	Juveniles	Temperate and Subtropical	Nelson 1994	Mixed and Freshwater	<i>Renibacterium salmoninarum</i>	Internal	Obligate	Delghandi et al. 2020	Bacteria	Waterborne	Delghandi et al. 2020	90	Captive	Other
Jun et al. 2009	13, 25	Chordata	Actinopterygii	Perciformes	<i>Oplegnathus</i>	<i>fasciatus</i>	Mobile	Vertebrate	Juveniles	Temperate	Nelson 1994	Marine	<i>Megalocytivirus ojarusZ</i>	Internal	Obligate	Kim et al. 2022	Virus	Waterborne/Indirect Transmission	Kim et al. 2022	45	Captive	Other
Ke et al. 2010	24.5	Chordata	Actinopterygii	Siluriformes	<i>Pelteobagrus</i>	<i>fulvidraco</i>	Mobile	Vertebrate	Juveniles	Temperate	Nelson 1994	Freshwater	<i>Mucor circinelloides</i>	Internal	Opportunistic	Vellanki et al. 2018	Eukaryote	Waterborne/Indirect Transmission	Vellanki et al. 2018	15, 19, 28, 45	Captive	Other
Kocan et al. 2009	10, 15, 20	Chordata	Actinopterygii	Salmoniformes	<i>Oncorhynchus</i>	<i>mykiss</i>	Mobile	Vertebrate	Juveniles	Subtropical	Nelson 1994	Mixed	<i>Ichthyophonus</i>	Internal	Obligate	Bruno & Ellis 1996	Eukaryote	Waterborne/Ingestion	Kocan 2019	28	Captive	Climate change



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