

Parasitism of an intertidal foundation species: Allometric scaling and environmental constraints on trematode distribution in barnacles across biological scales

Running title: Trematode infection in intertidal barnacles

Carl K. H. Ng^{1,2,3,4,*†}, Kevin C. K. Ma^{1,3,4,*†}, Jiou-An Ho³, Colin D. MacLeod^{1,5}, Alyssa-Lois M. Gehman^{1,3,4,6}, Christopher D. G. Harley^{1,3,4,6}

¹ Department of Zoology, University of British Columbia, 6270 University Boulevard, Vancouver, British Columbia, V6T 1Z4, Canada

² Department of Forest and Conservation Sciences, University of British Columbia, 2424 Main Mall, Vancouver, British Columbia, V6T 1Z4, Canada

³ Biodiversity Research Centre, University of British Columbia, 2212 Main Mall, Vancouver, British Columbia, V6T 1Z4, Canada

⁴ Hakai Institute, PO Box 25039, Campbell River, British Columbia, V9W 0B7, Canada

⁵ Department of Biology, University of Victoria, Cunningham 202, 3800 Finnerty Road, Victoria, British Columbia, V8P 5C2, Canada

⁶ Institute for the Oceans and Fisheries, University of British Columbia, 2202 Main Mall, Vancouver, British Columbia, V6T 1Z4, Canada

* Co-corresponding authors: Carl K. H. Ng (kaheicarl.ng@ubc.ca; carl.ng@hakai.org), Kevin C. K. Ma (kevin.ma@ubc.ca; kevin.ma@hakai.org)

† These authors share first authorship and contributed equally to this work

Abstract

Parasites of foundation species, such as fucoids, mussels, and barnacles, can shape the intertidal communities that depend on them, yet their effects remain poorly understood. On rocky shores of the Northeast Pacific, the acorn barnacle *Balanus glandula* forms dense aggregations that provide habitat for other species. We investigated infection by the microphallid trematode *Maritrema graciosum* in its second intermediate host, *B. glandula*, in British Columbia, Canada. First, we described the metacercarial cysts and examined how host morphology (in spring and summer) and reproductive output relate to infection. Second, we examined how infection varies with host position on live mussel shells, substratum type (rock *versus* mussel shells), and shore height. Third, we related prevalence and parasite load (number of cysts per host) to local air and sea-surface temperatures. Cysts had a mean diameter of $604 \pm 46 \mu\text{m}$ and 80% of them occurred in fewer than 20% of hosts. Host size predicted infection differently at two scales: within sites, larger barnacles were more likely to be infected, but among sites, infection was most likely in populations of intermediate size. Among infected hosts, parasite load was associated with host shape and became strongly size-dependent later in the season. Brood mass scaled hyper-allometrically with somatic mass ($b = 1.27$) and was unrelated to parasite load, while the scaling of parasite load with host mass differed among sites. Infection intensity (cysts per gram of somatic tissue) did not vary with host position on mussels or between substrata, but varied unimodally with shore height, peaking at ~ 2.9 m above chart datum. At a larger spatial scale around Vancouver Island, 70.7% of barnacles were infected, with site prevalence ranging from 0 to 100%. Infection probability increased with host size but was unrelated to temperature. Parasite load, however, increased with host size and with warmer-than-usual sea-surface temperatures, but decreased with

warmer-than-usual air temperatures. Relationships among individual hosts did not consistently hold across sites, seasons, or thermal environments, suggesting that host demography, immersion time, and parasite supply jointly structure infection in this intertidal foundation species.

Keywords: *Balanus glandula*, Body size, *Maritrema graciosum*, Metacercariae, North Pacific, Temperature, Thermal history

Introduction

Parasites are ubiquitous in intertidal rocky-shore systems, yet their influence is often cryptic due to their small size and concealed life stages, leading ecologists to underestimate their abundance and effects (Laguerre & Poulin, 2016). Parasites are involved in a majority of trophic links in some coastal food webs and parasite biomass can match or exceed those of top predators (Lafferty et al., 2006; Kuris et al., 2008). By altering host survival, growth, behaviour, and reproduction, parasites can re-configure the biology and ecology of free-living organisms and, in turn, community structure and composition (Mouritsen & Poulin, 2002; Hudson et al., 2006; Wood et al., 2007). Parasites are however, rarely distributed evenly among hosts. Instead, infection is often highly aggregated, with a small proportion of hosts harbouring most parasites, a pattern commonly captured by the 20/80 rule (Woolhouse et al., 1997). Understanding this infection heterogeneity is essential for determining how parasite risk arises and how their effects may propagate through host populations. Allometric scaling offers a quantitative framework for linking size-dependent physiological processes, such as metabolism, to broader ecological patterns of population dynamics, trophic interactions, and ecosystem functioning across diverse taxa (Brown et al., 2004; Peters, 1983). In host–parasite systems, host body size can heavily influence biological processes that shape parasite exposure, transmission, and accumulation (Hechinger, 2013), which may generate predictable relationships between host size and the parasite infrapopulation. Consequently, the allometric relationship between host size and parasite load may therefore reflect the relative importance of multiple size dependent processes, providing a basis for explaining why infection can vary substantially among individuals and populations.

Parasites can re-shape the biology of free-living organisms, and consequently, community structure and composition. These effects may be particularly consequential when parasites infect foundation (habitat-forming) species, such as mussels, macroalgae, and barnacles, which create physical structure and complexity, buffers temperature and desiccation stress, and provides space (refuge) for other species (Bruno & Bertness, 2001). Parasitism can therefore have an outsized effect on rocky-shore communities when parasite-induced changes in host abundance and performance propagate through intertidal ecosystems. Parasites that reduce the growth, reproduction, or survival of these hosts could have indirect effects on the community. For example, shell-boring endolithic (microbial) parasites, erode the dark periostracum and expose the paler shell beneath. This reduces the effect of solar radiation and increases host survival during thermal stress, potentially extending the upper intertidal limits of mussel beds (Gehman & Harley, 2019). Intertidal rocky shores also impose steep and fluctuating gradients in temperature,

emersion time, and wave exposure across vertical and geographic dimensions, often over just a few metres vertically (Helmuth et al., 2002; Harley & Helmuth, 2003). These gradients act on both hosts and free-living stages of parasites such that outcomes of host–parasite interactions are likely to vary sharply in space and time (Studer et al., 2010; Byers, 2021). Despite these ecological complexities, much of this understanding comes from soft-sediment systems, where trematode infection in snails, bivalves, and amphipods has been well-studied for decades (Mouritsen & Jensen, 1997; Mouritsen & Poulin, 2002). Rocky shores, however, have received comparatively little attention, with most research focusing on describing parasitic interactions and the dynamics of microbial disease on populations (e.g., algae, bivalves) (Fong et al., 2018; Davis et al., 2024).

Acorn barnacles are among the most widespread foundation species on temperate rocky shores. *Balanus glandula* dominates much of the high and mid intertidal zones of the Northeast Pacific, from Alaska to Baja California (Hines, 1978), and has invaded the shores of Argentina, Japan, South Africa, and Europe where it can form dense beds that alters local communities (Schwindt, 2007; Simon-Blecher et al., 2008; Robinson et al., 2017; Kerckhof et al., 2018). This barnacle is an important prey species and facilitates the establishment of later successional species, such as mussels (Connell 1970; Dayton 1971). Barnacles have served as model organisms in intertidal ecology for nearly a century, providing insight into competition, predation, reproduction, and responses to abiotic stress (Connell, 1961, 1970; Hines, 1978; Leslie et al., 2005; Gilman & Rognstad, 2018). Parasites, however, were largely excluded from these studies on barnacle ecology.

Known parasites of barnacles include parasitic castrators—such as the rhizocephalan *Boschmaella japonica* (Deichmann & Høeg, 1990; Høeg, 1995) and the epicaridean isopod *Hemioniscus balani* (Blower & Roughgarden, 1988; Lafferty & Kuris, 2009)—and the metacercariae of microphallid trematodes (Carrol et al., 1990; Irwin et al., 1990; McCarthy et al., 2002). Among trematodes, *Maritrema graciosum* uses intertidal snails as first intermediate hosts, barnacles as second intermediate hosts, and shorebirds as definitive hosts (Ma et al., 2026). Free-swimming cercariae released from snails are drawn into the barnacle by its cirri during feeding, penetrate the gut, and encyst on the outer (serosal) surface (Carrol et al., 1990; Irwin et al., 1990), where metacercariae accumulate until the barnacle is consumed by a bird. Although *M. graciosum* has been reported from *B. glandula* in the Northeast Pacific since the 1970s (Ching, 1978; MacLeod et al., 2021), it has received little study in this region beyond these records (Ma et al., 2026), despite its potential to shape the vertical distribution of its host on intertidal rocky shores

Infection in second intermediate hosts depends on both the supply of cercariae and the traits of the hosts that receive them. Because *M. graciosum* cercariae are small and weak swimmers (McCarthy et al., 2002), local water movement may also determine which hosts they reach. The feeding currents of neighbouring suspension feeders, such as mussels, could remove them from the water before they reach a barnacle host (Johnson et al., 2010). Beyond parasite supply and exposure, infection may be further constrained by the physical space and energetic resources available within hosts (Hechinger, 2013). Larger and older barnacles may accumulate more metacercarial cysts through prolonged exposure or greater physical internal space for parasite accommodation. The physical

capacity for hosting parasites, however, may not increase proportionally with host body mass, potentially imposing a size-dependent limit on parasite load. In parallel, energetics constraints may arise as parasites draw resources from their hosts for maintenance and development, potentially competing with the energy allocated to host growth, reproduction, and metabolic maintenance (Hechinger, 2013). Since physical space and energy resources may scale differently with host body size, their relative importance could determine the allometric relationship between host body size and parasite intensity. These constraints may become particularly apparent when parasite exposure is sufficiently high that supply is no longer the primary factor limiting accumulation, thereby producing heterogeneous size–infection patterns even within the same host–parasite system.

Temperature is equally important in structuring infection outcomes, acting directly on host–parasite interactions and indirectly through changes in host physiology and population structure. Parasite production, transmission, and establishment are temperature sensitive, with warming influence both the release and survival of infective stages (Mouritsen, 2002; Poulin, 2006; Studer et al., 2010; Morley, 2011). The magnitude and duration of thermal exposure are also important, as seasonal conditions, short-term temperature fluctuations, and extreme thermal events can produce different infection outcomes (Byers, 2021). The direction and strength of temperature–infection relationships can vary considerably across environments and biological scales, complicating predictions of disease risk under climate change (Cohen et al., 2020; Kirk et al., 2022). These relationships are particularly relevant for *B. glandula*, which is exposed to high air temperatures during emersion (Berger & Emlet, 2007; Ober et al., 2019), while high shore intertidal invertebrates often live close to their thermal limits (Stillman, 2003; Somero, 2010). Infection can further raise or lower host thermal tolerance (reviewed in Żbikowska & Cichy, 2015). For example, heavily infected mussels acting as second intermediate hosts showed greater heat tolerance (Selbach et al., 2020). If *M. graciosum* infection is concentrated higher on the shore or at warmer sites, its effect on host thermal performance could consequently influence the vertical and geographic distribution of *B. glandula*. Understanding how infection varies with local thermal conditions and thermal history is therefore important for predicting infection patterns and their potential consequences for this intertidal foundation species.

In this study, we characterize the host–parasite relationship between *M. graciosum* and *B. glandula* in British Columbia, Canada, asking how infection is structured across individual hosts, micro-habitats, and regional thermal regimes. First, we described the metacercarial cysts of *M. graciosum* and test how host morphology and reproductive output relate to infection. We expected parasite load to increase with host size, though not necessarily in proportion to body mass if space or energy within hosts becomes limiting (Hechinger, 2013). We also examined whether these relationships held between spring and summer, when host growth and parasite transmission may change. If cysts divert host resources, we expected host brood mass to decline with parasite load once host size was accounted for. Second, we examined how prevalence and infection intensity vary with host position on live mussel shells relative to their inhalant and exhalant siphons, substratum type (rock *versus* mussel shells), and shore height. We expected delivery of cercariae to barnacles to depend on local water movement. Mussel feeding currents could increase exposure for barnacles near the inhalant siphon, yet mussels and other suspension feeders could reduce

exposure by consuming cercariae (Johnson et al., 2010). Such predation on transmission stages can generate fine-scale variation in infection among sessile hosts. In California, barnacles in close proximity to sea anemones readily consume the larvae of a parasitic isopod and were 2.5 times less likely to be infected than barnacles farther away (Fong & Kuris, 2017). We also expected infection to vary with shore height, which determines how long barnacles are submerged and feeding (e.g., Carrol et al., 1990). Third, at a larger spatial scale, we related prevalence and parasite load to the local thermal regime (air and sea-surface temperatures). Because temperature affects the production, survival, and infectivity of cercariae as well as host physiology (Studer et al., 2010; Morley, 2011), we expected infection to be greater at sites with warmer conditions. We also expected these effects to differ between air and seawater temperatures and between prevalence and intensity, as temperature–infection relationships often change across biological scales (Kirk et al., 2022). Together, these objectives examined how host traits and environmental conditions, particularly temperature, shaped the distributions and accumulation of parasites in an intertidal foundation species. By comparing size-dependent infection across individuals, populations, seasons, and environmental gradients, we explored whether host–parasite allometry provides a consistent framework for understanding infection heterogeneity across biological scales in a marine system.

Methods

Study area and sampling design

Sampling was carried out on intertidal rocky shores along the coast of British Columbia, Canada, in the Northeast Pacific (Figure 1). We surveyed a total of 60 sites for the barnacle *Balanus glandula*, the second intermediate host of the trematode *Maritrema gratosum*. In 2021, 35 sites were surveyed from the sheltered waters of the Salish Sea to the exposed outer coast of Vancouver Island (Figure 1A). The remaining 25 sites were located around Burrard Inlet in Vancouver and West Vancouver (Figure 1B) and were surveyed between 2019 and 2025.

Samples from Burrard Inlet were used to describe the metacercaria, to relate infection to reproductive output, and to relate infection to host morphology. Additional surveys were also used to test the effects of host position on mussel shells, substratum type, and shore height. Unless stated otherwise, barnacles were collected from the upper limit of the mid-intertidal zone during low tide.

Tissue processing and infection metrics

The somatic soft tissue of each barnacle was removed and weighed with a fine balance (precision: 0.001 mg). The tissue was then smeared on a microscope slide and all metacercariae were counted under a light microscope. For the reproductive output calculations, brood was removed and weighed separately before the somatic tissue. For barnacles sampled around Vancouver Island (Figure 1A), the scutum length was measured with a caliper and the scutum area was estimated.

Three infection metrics were used. Prevalence was defined as the proportion of barnacles infected by *M. gratosum* at a given site. Parasite load (or cyst count) was defined as the number of metacercarial cysts in a barnacle. Infection intensity was defined as the number of cysts per gram of somatic tissue (wet weight), and was used in analyses that did not have host size as a covariate.

Morphology of the metacercarial cyst

Metacercarial cysts were extracted from adult barnacles collected in 2020 at a site on the southern shore of Burrard Inlet (site LIT; Figure 1B). The diameters of 90 metacercariae were measured under a light microscope (precision: 1 μ m). For each cyst, two perpendicular diameters were measured and averaged.

Host morphology and infection intensity

In May 2025, adult *B. glandula* were collected from all 25 sites around Burrard Inlet ($n = 10\text{--}11$ individual barnacles per site). To assess seasonal change, six sites with high infection were re-sampled in July 2025 ($n = 20$ per site). For each barnacle, shell height, basal diameter, and the length and width of the aperture were measured with a digital caliper (precision: 0.1 mm). Somatic tissue (wet weight) was then measured and metacercariae were counted as described above.

Because the five morphological traits were strongly correlated, we summarised them with a principal component analysis (PCA) of the traits of barnacles that were sampled in May. All morphometric predictors were standardised prior to entering the PCA. The first two principal components (PC1 and PC2) were retained as predictors in subsequent analyses. Barnacles sampled in July were projected onto the May axes, so that host morphology could be compared directly between months.

To separate risk of infection (derived from site-level prevalence data) with site-level morphology from that associated with individual variation, PC scores were decomposed into two components: within-site and between-site components. The between-site component was the site mean score. The within-site component was each individual's deviation from its site mean.

To determine whether host morphology explains parasite variation across and within sites, infection in May was analysed with two complementary generalised linear mixed models (GLMMs) in the R package 'glmmTMB' (Brooks et al., 2017). Presence-absence of metacercarial cysts was modelled with a binomial distribution and logit link. Using only data from infected individuals, parasite load was modelled with a zero-truncated negative binomial distribution with an 'NB1' variance structure and log link, to account for overdispersion. Both models included within- and between-site components of PC1 and PC2 as fixed effects and site as a random intercept. A quadratic

term for between-site PC1 was included in the presence–absence model to test for a non-linear relationship with site-level mean host size.

As seasonal warming may influence host growth and parasite accumulation, we expected the relationship between host morphology and parasite load to differ between spring and summer, potentially reflecting greater parasite accumulation under warmer conditions. Seasonal change in the relationship between morphology and parasite load was analysed for the six resampled sites. Parasite load was again modelled with a zero-truncated negative binomial distribution, with site as a random intercept. For this analysis, within-site PC scores were centred on month-specific means for each site. Site-level morphology was represented in two ways. Persistent site morphology was the mean PC score of each site (PC_site) across both months. Seasonal change in site morphology was the difference in site mean PC scores between the two sampling time points ($\Delta PC1$ and $\Delta PC2$). Sampling month was included as a fixed factor and interacted with the within-site PC scores and with $\Delta PC1$ and $\Delta PC2$, to test whether morphology–parasite load relationships changed between seasons. The adequacy of all models was assessed with simulation-based residual diagnostics in the R package ‘DHARMA’ (Hartig, 2022).

Reproductive output and infection

In 2021, 91 adult barnacles carrying broods ($n = 11\text{--}20$ individuals per site) were collected from five Burrard Inlet sites (sites KIE, KIW, LIT, LIV, and LIB; Figure 1B). Brood and somatic tissue masses were measured, and metacercariae were counted as described above. Total brood mass was used as a proxy for individual reproductive output. Fewer than 9% of barnacles were uninfected. We therefore restricted all analyses to only include infected individuals.

Because parasite abundance can vary non-linearly with host size through differences in cumulative exposure, encounter rate, or host capacity, we therefore first quantified how parasite load scaled with somatic mass to determine whether parasites were disproportionately concentrated in particular host size classes and whether this relationship varied among sites. We then modelled brood mass as a function of both somatic mass and parasite load. This distinguished the size-dependence of reproductive output from any additional association between infection and reproduction.

Parasite load was modelled with a zero-truncated negative binomial GLMM with an ‘NB2’ variance structure and log link. Somatic mass was natural-log transformed and centred on the mean log somatic mass of each site. The resulting predictor represents an individual's proportional deviation from the geometric mean somatic mass of its site.

Site was included as a fixed factor and interacted with within-site log somatic mass. This allowed both expected cyst abundance and the cyst–somatic scaling exponent (b) to differ among sites. The inclusion of the

interaction was supported by a likelihood-ratio test against a nested model with a single slope common to all sites ($\chi^2_4 = 11.39$, $p = 0.02$; $\Delta\text{AIC} = 3.39$).

Site-specific scaling exponents and their 95% confidence intervals were estimated with the ‘*emtrends*’ function in the ‘*emmeans*’ R package. Pairwise differences among exponents were evaluated with Tukey-adjusted contrasts. Departure of each exponent from isometric scaling ($b = 1$) was evaluated with two-sided Wald tests, with Holm adjustment for the five simultaneous tests.

Brood mass, somatic mass, and parasite load were natural-log transformed. This allowed the brood–somatic relationship to be estimated as a power function and represented parasite load as proportional differences in parasite load. To distinguish variation within sites from differences among sites, log somatic mass and log parasite load were each centred on their site means. Both centred variables therefore represent an individual's proportional deviation from the geometric mean of its site, with zero corresponding to the local geometric mean.

Log brood mass was modelled with a Gaussian model in ‘*glmmTMB*’, which allows conditional means and residual dispersion to be modelled simultaneously. Within-site log somatic mass, within-site log parasite load, their interaction, and site were included as predictors, with site treated as a fixed factor to account for differences in expected reproductive output among sites. Residual dispersion was modelled as a function of within-site log somatic mass with a log link, allowing residual variance to change with host size.

We also fitted an *a priori* model containing all interactions among somatic mass, parasite load and site, to test whether the association between parasite load and reproduction varied among sites. This model did not improve fit relative to the model without site-specific slopes ($\chi^2_{12} = 14.93$, $p = 0.245$; $\Delta\text{AIC} = 9.1$). The three-way interaction was also not supported by a type III Wald test ($\chi^2_4 = 8.98$, $p = 0.062$). The more parsimonious model with site-specific intercepts was therefore retained.

Because brood and somatic mass were modelled on logarithmic scales, the coefficient of within-site log somatic mass represents the brood–somatic mass allometric scaling exponent (b) at the local mean parasite load. Departure from isometric scaling ($b = 1$) was tested with a Wald test of the trend estimated with the ‘*emtrends*’ function.

Position of host on mussel and infection

To test whether a barnacle's position relative to the feeding currents of a mussel affects infection, mussels (*Mytilus trossulus*) hosting barnacles were collected from a site on the northern shore of Burrard Inlet (site AMB; Figure 1B) in July 2025. We targeted mussels of similar size (shell length 36.5 ± 3.5 mm, mean $\pm$ standard

deviation; $n = 30$), each carrying at least two epibiotic barnacles, one positioned anteriorly and the other posteriorly. Barnacles of similar size were also targeted (basal diameter 9.3 ± 1.5 mm; $n = 60$).

Infection intensity was determined for each barnacle as described above. The straight-line distance on the shell surface from the centre of each barnacle's aperture to the shell margin at the inhalant and exhalant siphons was measured from photographs in ImageJ (version 1.54g).

The two barnacles on each mussel shared the same host mussel, micro-habitat, and local supply of cercariae, so we analysed differences within each mussel, with each mussel acting as its own control. This also accounts for differences in size among mussels. For each mussel, we calculated the difference between the posteriorly-positioned and the anteriorly-positioned barnacle in infection intensity (Δ infection intensity) and in absolute distance to each siphon (Δ distance). Δ infection intensity was regressed against Δ distance to the inhalant siphon and, separately, to the exhalant siphon, using linear regression. Because the residuals deviated from normality, the associations were also tested with Spearman rank correlations.

Substratum types and infection

To test whether infection differs between barnacles attached to live mussels and those attached to rocks, barnacles on both substrata were collected at six sites in Burrard Inlet (sites ALB, AMB, THI, TEE, LIT, and LIH; Figure 1B) in July 2025. Twenty barnacles were collected from each substratum at each site. Mussels (shell length 36.8 ± 4.8 mm; $n = 120$), cobbles (Feret diameter 61.2 ± 15.7 mm; $n = 120$), and barnacles (basal diameter 9.9 ± 0.8 mm; $n = 240$) of similar sizes were targeted. Infection intensity was determined for each barnacle as described above.

Infection intensity was analysed with a Tweedie distribution with log link. The Tweedie distribution accommodates continuous, non-negative, right-skewed responses that include exact zeros. Substratum, site, and their interaction were included as fixed effects. Infection intensity was then compared between substrata within each site using pairwise contrasts, with Holm adjustment for six tests.

Shore height and infection

To test the effect of shore height on infection, barnacles ($n = 5$ per interval) were collected at 5-m intervals along a transect running down the shore at three sites in Burrard Inlet (sites THI, LIT, and LIJ; Figure 1B). The shore height of each interval was measured with a laser level relative to chart datum. Infection intensity was determined for each barnacle as described above.

Infection intensity was analysed with a Tweedie distribution with log link (see above). Intervals were included as a random intercept, because the five replicate barnacles at each interval shared the same shore height. Site, shore height, and their interaction were included as fixed effects. Pairwise differences among sites at a common shore height were tested with contrasts, with Holm adjustment.

Thermal regime

Hourly air temperature 2 m above the surface and sea-surface temperature (SST) for the 35 sites around Vancouver Island were obtained from the ERA5 global re-analysis (Hersbach et al., 2020), accessed through the *Copernicus Climate Data Store* (hourly data; Hersbach et al., 2023). Records spanned 1 August 2020 to 30 August 2021, covering approximately one year before each collection date. Data were downloaded using the R package ‘ecmwf’ (Hufkens et al., 2019). Values at each site were calculated by bi-linear interpolation (i.e., weighted average) of the four nearest ERA5 grid cells (0.25° resolution) using the R package ‘terra’ (Hijmans, 2026). Temperatures were converted from kelvin to degrees Celsius.

To characterise thermal conditions experienced by barnacles before each collection date, daily air temperature and SST records (averaged from hourly values) were decomposed into expected seasonal baselines and deviations from those expectations. Seasonal temperature trajectories were estimated separately for air temperature and SST using generalised additive models (GAMs), incorporating a cyclic smooth of day of year, site-specific intercepts, and site-specific seasonal smooths. This accounted for predictable seasonal and among-site thermal structure, such that departures from the estimated baseline represented temperature deviations specific to a site and time of year. A basis dimension of $k = 40$ was used for the primary analysis, with prior testing with $k = 20$ and $k = 60$ to assess sensitivity to smoothing resolution. Daily temperature deviations were calculated as the difference between observed temperature and corresponding site-specific seasonal GAM prediction.

Thermal history was summarised over three periods preceding collection, including the recent (0–13 days), prior (14–90 days), and longer-term conditions (0–341 days). Long-term thermal conditions were characterised by mean air temperature and SST and by their thermal variability, calculated as the standard deviation of daily temperature deviations. Recent and prior conditions were characterised by the mean temperature deviation and its variability (SD of daily deviations) within each period. Continuous environmental predictors were standardised before model fitting.

Individual infection status (absence or presence of cyst) and parasite load (cyst counts) were evaluated using GLMM with site included as a random intercept and centred log host scutum area retained as an *a priori* predictor. Thermal model selection began with a full model containing all thermal-history predictors, followed by reduced models to identify whether simpler representations of thermal history provided equivalent or improved model support. Models were evaluated using AIC, likelihood-ratio tests for nested comparisons, and residual

diagnostics. Thermal predictors were considered according to six biologically defined components, including long-term mean temperature, long-term thermal variability, prior mean temperature deviation, prior thermal variability, recent mean temperature deviation, and recent thermal variability, with paired air temperature and SST predictors within each component. Infection status was modelled for all barnacles using binomial model, while parasite load was modelled among infected barnacles using zero-truncated negative binomial model with an ‘NB2’ variance structure.

Model adequacy was assessed using simulated DHARMA residuals at both individual- and site-aggregated levels. Spatial autocorrelation of location-aggregated residuals was evaluated using Moran’s I. Where residual spatial structure was detected, robustness of fixed-effect inference was evaluated using geographic block bootstrapping. Locations separated by no more than a specified distance threshold were connected, with connected components treated as bootstrap blocks. Thresholds of 5 and 10 km were used to represent local and broader spatial dependence, respectively, with these scales selected relative to the observed nearest-neighbour distance distribution (median = 3.69 km; 75th percentile = 10.18 km). At each spatial scale, blocks were re-sampled with replacement and the retained model was refitted 1,000 times. Percentile 95% confidence intervals for each fixed-effect coefficient (β) were calculated from the bootstrap distributions, with effects considered robust at a given spatial scale when the interval excluded zero.

Results

Morphology of the metacercarial cyst

Across 90 metacercarial cysts, the mean cyst diameter was $603.7 \pm 46.0 \mu\text{m}$, with a median of $591.3 \mu\text{m}$ (Figure 2). The central 50% of cysts ranged from 578.6 to $622.6 \mu\text{m}$, with an overall observed range of 503.0 to $829.5 \mu\text{m}$.

Morphology of the barnacle host

The infection pattern was heterogeneous among individual barnacles and across 25 sampling sites. In May, 51.2% of the 252 barnacle hosts were uninfected, corresponding to an overall infection prevalence of 48.8%, with zero infection recorded in two sites (SPE and SPW). Parasite load was strongly aggregated, with 80% of all metacercarial cysts concentrated in only 17.8% of hosts. Infection intensity demonstrates substantial spatial heterogeneity, with the proportion of hosts required to account for 80% of cysts locally ranging from 10% to 60% across the 23 infected sites (Figure 3). Several sites were observed with stronger aggregation than the pooled populations, reflecting parasite load is highly variable on both individual and site level.

In May, PC1 explained 72.1% of morphological variation (Figure 4) and each of the five traits (shell height, basal diameter, opercular width and length, and wet weight) contributed relatively evenly to the axis (15–24%), representing an overall body-size axis. PC2 explained a further 12.6% and primarily contrasted shell height with basal diameter (a combined ~90% of squared loadings). Positive PC2 scores therefore represented relatively taller, narrower individuals, whereas negative scores represented relatively flatter individuals with broader basal diameter. Together, PC1 and PC2 explained 84.7% of morphological variation.

Within sites, infection probability increased with host size in May ($\beta = 0.468 \pm 0.205$, $p = 0.022$), with 1-SD increase in within-population PC1 axis corresponded to 1.46-fold greater odds of infection (95% CI: 1.05 to 2.01; Figure 5A). In contrast, the relationship between mean host size and infection probability at the site level was non-linear (quadratic $PC1_{\text{between site}}: \beta = -2.669 \pm 0.991$, $p = 0.007$; linear $PC1_{\text{within site}}: \beta = 0.503 \pm 0.622$, $p = 0.419$), whereas the quadratic relationship indicates peak infection probability at intermediate global site mean $PC1(PC1_{\text{between site}}: \beta = 0.094$, equivalent to 0.16 among-site SD) and declined towards sites characterised by either smaller or larger hosts (Figure 5B). PC2 was not associated with infection probability on neither site nor individual levels ($PC2_{\text{between site}}: \beta = -0.149 \pm 0.613$, $p = 0.808$; $PC2_{\text{within site}}: \beta = 0.0607 \pm 0.186$, $p = 0.745$).

Among infected hosts, parasite load was associated with relative host shape instead of overall size. Within site, a 1-SD increase in PC2 was associated with a 38.6% reduction in expected parasite load (95% CI: 0.378 to 0.875; $\beta = -0.539 \pm 0.220$, $p = 0.0142$), indicating greater parasite loads in relatively flatter individuals with broader basal diameters (Figure 6). Neither within-site overall size ($\beta = 0.191 \pm 0.208$, $p = 0.359$, site mean PC1 ($\beta = -0.150 \pm 0.589$, $p = 0.799$), nor PC2 ($\beta = -0.0684 \pm 0.535$, $p = 0.898$) was detectably associated with parasite load.

Parasite load showed scale- and season-dependent associations with host morphology across the six high infection sites selected for the seasonal analysis. Within sites, the relationship between PC1 and parasite load differed between May and July (May-July: $\beta = -0.503 \pm 0.194$, $p = 0.00942$; Figure 7A). A 1-SD increase in individual PC1 relative to the local site-month centroid corresponded to a 1.60-fold (95% CI: 1.37 to 1.88) increase in expected parasite load in July, compared with 1.13-fold in May (95% CI: 0.910 to 1.40). In contrast, the within-site PC2 relationship showed little seasonal difference (May-July: $\beta = -0.0764 \pm 0.198$, $p = 0.699$; Figure 7B).

Persistent among-site morphological characters in terms of size and relative shape were both positively associated with parasite load ($PC1_{\text{site}}: \beta = 1.74 \pm 0.801$, $p = 0.0295$; $PC2_{\text{site}}: \beta = 3.34 \pm 1.02$, $p = 0.00103$). A 1-SD increase in site-average PC1 corresponded to a 1.60-fold greater expected parasite load (95% CI: 1.05 to 2.44), whereas the corresponding effect for PC2 was 2.79-fold increase (95% CI: 1.51 to 5.15).

Site-level changes in morphology from May to July were also positively associated with parasite load. Averaged across both sampling months, sites with greater May-to-July increases in host size and relative shape had

greater parasite load ($\beta_{\Delta PC1} = 2.280 \pm 0.659$, $p < 0.001$; $\beta_{\Delta PC2} = 1.20 \pm 0.568$, $p = 0.0346$; Figure 8), equivalent to 1.88- and 2.39-fold differences in expected parasite load per 1-SD, respectively. The relationship between the morphological change across months and parasite load, however, did not become stronger from May to July ($\beta_{\Delta PC1_July-May} = -0.300 \pm 1.07$, $p = 0.779$; $\beta_{\Delta PC2_July-May} = -0.354 \pm 0.395$, $p = 0.371$). Sites following more positive changes in size or relative shape were therefore, generally characterised by higher parasite load in both months, but the greater change in morphology did not imply a correspondingly greater May-to-July increase in parasite intensity.

Reproductive output of infected barnacles

Parasite load varied by approximately two orders of magnitude among infected barnacles, reaching a maximum of 201 metacercarial cysts per individual. The scaling relationship between parasite load and host somatic mass differed significantly among sites ($\chi^2_4 = 11.39$, $p = 0.02$, Figure 9), indicating substantial spatial variation in host size scaling in parasite accumulation. Estimates of the scaling exponent ranged from negative at site KIW ($b = -0.58$) to strongly positive and potentially hyper-allometric scaling at site KIE ($b = 1.95$; Figure 10), although only site KIW differed statistically from isometry ($b = -0.58 \pm 0.55$, $z = -2.89$, $p = 0.02$). Pairwise contrasts indicated that the scaling exponent differed between sites KIE and KIW ($\Delta b = 2.52 \pm 0.83$, $z = 3.03$, $p = 0.020$) and between sites KIW and LIB ($\Delta b = 1.86 \pm 0.66$, $z = -2.84$, $p = 0.037$), whereas the remaining pairwise contrasts between sites were not supported ($p \geq 0.13$). Thus, host size was associated with parasite load in a strongly site-dependent manner, with both the magnitude and direction of cyst accumulation across host size classes varying spatially.

Brood mass increased strongly with somatic tissue mass ($\chi^2_1 = 243.61$, $p < 0.001$), with a brood–somatic mass scaling exponent of $b = 1.27$ (95% CI = 1.11 to 1.42) at the within-site mean parasite load. This relationship was hyper-allometric, with the scaling exponent significantly greater than isometry ($\Delta b \neq 1$: $z = 3.27$, $p = 0.001$), and no evidence that within-site variation in parasite load modified the brood–somatic mass scaling relationship ($\chi^2_1 = 0.40$, $p = 0.528$; Figure 9). Parasite load was also unrelated to expected brood mass at the within-site mean somatic mass ($\chi^2_1 = 0.13$, $p = 0.718$), and the brood mass did not differ significantly among sites ($\chi^2_4 = 6.37$, $p = 0.173$). Thus, reproductive output was strongly related to host somatic mass in a hyper-allometric manner, with little evidence that parasite load explained additional variation in brood mass or altered its scaling with host size.

Position of host on mussel and infection

80% of barnacles on mussels were infected, with an average infection intensity of 108 ± 122 cysts g^{-1} . Within mussels, the anteriorly-positioned and posteriorly-positioned barnacles differed in distance by 1.01 ± 0.67 cm to the inhalant siphon and by 0.94 ± 0.78 cm to the exhalant siphon. Their infection intensity differed by 102.6 ± 139.5 cysts g^{-1} .

The position of a barnacle on its mussel did not detectably affect infection by *M. gratosum*. Δ infection intensity between the two barnacles on the same mussel was not related to Δ distance from the inhalant siphon ($R^2 = 0.04$, $F_{1,28} = 1.25$, $p = 0.27$). Nor was it related to Δ distance from the exhalant siphon ($R^2 = 0.04$, $F_{1,28} = 1.30$, $p = 0.26$). Spearman rank correlations gave the same result (inhalant: $\rho = 0.21$, $p = 0.28$; exhalant: $\rho = 0.19$, $p = 0.33$). These relationships remained non-significant when mussels carrying an uninfected barnacle were excluded (inhalant: $R^2 = 0.02$, $F_{1,18} = 0.43$, $p = 0.52$; exhalant: $R^2 = 0.10$, $F_{1,18} = 2.05$, $p = 0.17$).

Substratum types and infection

Prevalence was similar on rock (94.2%) and on mussel shells (91.7%). Across all sites, mean infection intensity was 660 ± 787 cysts g^{-1} on rocks and 597 ± 600 cysts g^{-1} on mussel shells. Infection intensity varied substantially among sites ($\chi^2_5 = 84.39$, $p < 0.001$). There was no overall difference in infection intensity between barnacles on rocks and on mussel shells ($\chi^2_1 = 1.99$, $p = 0.16$).

Differences between substratum types depended on site (substratum $\times$ site interaction: $\chi^2_5 = 16.07$, $p = 0.007$). Barnacles on mussel shells had lower infection intensity than those on rocks at site TEE ($z = -3.04$, Holm-adjusted $p = 0.014$). Infection intensity did not differ significantly between substratum types at the remaining five sites (ALB, AMB, THI, LIT, and LIH), although intensity at site AMB tended to be lower on mussel shells ($z = -2.25$, Holm-adjusted $p = 0.120$).

Shore height and infection

Infection intensity varied strongly with shore height, following a unimodal shape (Figure 11). It was lowest near the lower range of the host's vertical distribution (roughly below a shore height of 1.5 m). Infection intensity increased through the mid-intertidal zone and was highest between 2.4 and 3.1 m and then declining near the upper vertical limit. This overall relationship did not differ among sites (shore height $\times$ site: $\chi^2_4 = 3.59$, $p = 0.46$). Infection intensity peaked at ~ 2.93 m across sites (peak at site THI: 3.09 m; peak at site LIT: 2.92 m; peak at site LIJ: 2.73 m).

Thermal regime

Of the 352 barnacles sampled across 35 sites, 249 of them were infected, corresponding to an overall infection prevalence of 70.7%. Infection prevalence varied substantially among sites, with a median site prevalence of 90.0% (IQR: 55.1–100%) and a range of 0–100%. Among the 249 infected barnacles, parasite load were strongly right-skewed, with a median of 6 cysts (IQR: 3–22), a mean of 28.4 (SD = 58.0), and a range of 1–362 cysts; 10% of infected hosts contained more than approximately 80 cysts (90th percentile = 80.2). Mean parasite load also varied

substantially among the 31 sites containing infected barnacles, with a median site-level mean of 6.67 cysts per barnacle and a range of 1–153.

Thermal predictors provided little improvement in model support for infection status. The model containing all thermal predictors had substantially higher AIC than the baseline host-size model ($\Delta\text{AIC} = 10.50$) and did not improve fit (likelihood-ratio test: $\chi^2_{12} = 13.50$, $p = 0.334$). Similarly, models containing individual thermal components had higher AIC values ($\Delta\text{AIC} = 2.38$ to 3.68) and provided no significant improvement in likelihood (all $p \geq 0.446$). There was, however, a systematic lack of fit in site-aggregated residuals for all models except the one including prior (14–90 d) air and SST anomalies. This model was therefore retained as the final prevalence model, with infection probability estimated to increase significantly with host size ($\beta = 2.46$, $z = 2.94$, $p = 0.003$; Figure 12). Neither prior mean air temperature deviation ($\beta = -0.61$, $z = -0.80$, $p = 0.424$) nor prior mean SST deviation ($\beta = 0.95$, $z = 1.23$, $p = 0.218$) showed an independent association with infection probability. Site-aggregated residuals exhibited significant negative spatial autocorrelation (Moran's $I = -0.231$, $p = 0.043$), indicating greater dissimilarity in residual infection status among geographically neighbouring locations than expected under spatial independence. Geographic block bootstrapping nevertheless supported the fixed-effect inference from the fitted model. The positive host-size association remained robust under both 5- and 10- km spatial blocks (95% bootstrap CIs for β : 0.86 to 4.48 and 1.02 to 4.77, respectively). In contrast, bootstrap intervals for prior mean air-temperature deviation included zero at both spatial scales (5 km: -1.76 to 0.30 ; 10 km: -1.68 to 0.57), as did those for prior mean SST deviation (5 km: -0.49 to 2.41 ; 10 km: -0.29 to 2.37). Thus, accounting for geographic non-independence did not alter the primary findings of the fitted model.

Host scutum area, prior SST variability, prior mean SST deviation, recent mean air temperature deviation and prior air temperature deviation were retained as predictors of parasite load (Figure 13). The retained model had a lower AIC than the full thermal model (1746.31 *versus* 1759.16; $\Delta\text{AIC} = 12.85$), while the additional parameters in the full model did not significantly improve fit (likelihood-ratio test: $\chi^2_9 = 5.15$, $p = 0.821$). Predictor collinearity was low (maximum VIF = 2.70). Residuals exhibited significant negative spatial autocorrelation (Moran's $I = -0.307$, $p = 0.012$). Geographic block bootstrapping nevertheless showed that the positive association with host size ($\beta = 1.175$, $z = 4.14$, $p < 0.001$) was robust under both 5- and 10-km blocks (95% CIs: 0.75 to 1.83 and 0.59 to 1.88, respectively). The negative association with recent air temperature deviation ($\beta = -1.062$, $z = -3.35$, $p < 0.001$) was similarly robust (-1.64 to -0.57 and -1.54 to -0.51). Prior mean SST deviation was positively associated with parasite load ($\beta = 0.833$, $z = 3.46$, $p < 0.001$), with bootstrap intervals excluding zero at both spatial scales (0.21 to 1.29 and 0.07 to 1.37). Prior SST variability was also positively associated with parasite load ($\beta = 0.732$, $z = 2.37$, $p = 0.018$), but its bootstrap interval excluded zero at 5 km (0.07 to 1.53) and not at 10 km (-0.13 to 1.39), indicating sensitivity to the scale of spatial dependence. Prior air temperature deviation was not statistically supported ($\beta = -0.471$, $z = -1.94$, $p = 0.053$). Thus, the associations of parasite load with host size, recent mean air temperature

deviation, and prior mean SST deviation remained supported under both examined spatial scales, whereas support for prior SST variability was spatial-scale dependent.

Discussion

Our goal was to introduce the *Balanus glandula*-*Maritrema graciosum* interaction as a novel marine host-parasite system for addressing fundamental ecological questions. In documenting the natural history of this understudied biotic interaction in the Northeast Pacific, we revealed pronounced and structured heterogeneity in parasite risk across biological and temporal scales. Infection was widespread across *B. glandula* populations in Burrard Inlet and around Vancouver Island, yet prevalence and intensity varied substantially among populations separated by hundreds of metres to kilometres, among host phenotypes and across seasons. Intriguingly, relationships observed among individual hosts did not consistently translate to patterns observed among populations or through time. Such divergence reflects a fundamental challenge in ecology when scaling biotic interactions to large scale ecological predictions, empirical studies have shown that heterogeneity in exposure, infection success, and intrinsic host properties can generate different population-level infection patterns (Gourbière et al., 2015; VanderWaal & Ezenwa, 2016). Larger hosts are conventionally expected to accumulate more parasites, yet a recent meta-analysis found only a weak average size-infection relationship, in contrast to a striking heterogeneity among host-parasite systems (Hasegawa et al., 2026). Similarly, warming does not universally increase disease risk, with its direction and magnitude depending strongly on host thermal history (Cohen et al., 2020). Even less is known about how these effects propagate between biological scales, with Kirk et al. (2022) identifying only 24 host-parasite systems with sufficient thermal data to compare individual- and population-level parasitism, with some of the largest thermal mismatch occurring among environmentally transmitted parasites. What are the underlying mechanisms that determine the strength and consistency of infection patterns across environmental conditions? The *B. glandula*-*M. graciosum* interaction provides an opportunity to investigate this question in a naturally size-structured, sessile host whose morphology, reproductive investment, and environmental history can be examined alongside infection.

The heterogenous infection pattern observed in this study could consequently arise through several complementary processes operating across host phenotypes, environments, and biological scales. Parasite supply is likely an important source of variation, depending on the abundance and infection of first intermediate hosts, input from avian definitive hosts, and the production, survival and hydrodynamic transport of free-living cercariae. At two sites, *Littorina scutulata* and *L. sitkana* contained at least two microphallid cercarial morphotypes (C.D. MacLeod, personal observation), although molecular confirmation is required before either can be identified as first intermediate host of *M. graciosum* in the Northeast Pacific. Hydrodynamic transport may further decouple the distribution of infected snails from infection in barnacles, while environmental conditions can alter cercarial production, activity, survival, and infectivity. Cercariae are short-lived and lecithotrophic, with longevity constrained by finite energetic reserves, making transmission sensitive to environmental effects on metabolism (Morley, 2011).

Beyond parasite supply, however, our findings suggest host morphological and reproductive traits, and thermal history, capture different dimensions of infection risk.

Host morphology

Despite being two complementary dimensions of host morphology, body size and shape were not interchangeable predictors of parasite risk, differing both in the infection components they explained and in the associations across biological scales. In May, relatively large barnacles within populations were more likely to be infected, yet size did not predict parasite load once infection had occurred. Greater size may therefore increase the cumulative opportunity for infection through age, encounter or exposure, without necessarily determining subsequent parasite accumulation, consistent with the multiple processes underlying size-infection relationships across host–parasite systems (Hasegawa et al., 2026). Instead, infected individuals with broader, squatter shells carried more metacercariae than relatively columnar individuals, indicating that relative geometry captured variation in parasite accumulation independently of overall size. Interestingly, the positive individual-level size–prevalence relationship did not scale monotonically among populations, where prevalence instead peaked at intermediate mean body size. This scale dependence of infection probability may arise when individual size and population mean size capture different ecological information. Within populations, size may reflect differences in cumulative exposure, whereas among populations mean size may reflect host recruitment and other demographic histories that can co-vary with spatial variation in local parasite supply. Host shape may provide a complementary signature of these processes because crowding constrains lateral growth in sessile barnacles and promotes relatively columnar growth, whereas less crowded individuals can expand laterally (Giménez & Jenkins, 2013). Consequently, variation in shell shape may reflect the density-dependent growth environment in which parasite exposure and accumulation occur. Recruitment density can leave persistent effects on adult barnacle morphology and population structure even after density-dependent mortality obscures the original settlement pattern (Hills & Thomason, 2003), while host density can independently alter per-capita infection risk by changing the spatial distribution of environmentally transmitted parasites among available hosts (Buck et al., 2017). In *Chthamalus fissus*, where female reproductive activity was most frequent at intermediate body sizes, a corresponding intermediate-size peak in parasitism was generated by the ovarian-fluid-feeding castrator *Hemioniscus balani*, even though infection probability increased with size among hosts functioning as females (Fong et al., 2019). Intermediate mean size may therefore characterise population states where host demography and local parasite supply jointly favour infection, rather than representing an intrinsically optimal host size for *M. gratosum*. The contrasting individual- and population-level size–infection relationships therefore demonstrate why host size alone is insufficient to predict infection without accounting for the ecological processes that also drive host size structure.

Seasonal changes provided further evidence that host demography and infection pressure may jointly determine how morphology relates to parasite accumulation. The positive association between squat morphology and individual parasite load in individuals observed in May was not retained in July, whereas a stronger positive

size–intensity relationship emerged later in the season. At the population level, greater intensity was associated with more columnar host morphology. The emergence of the stronger size-dependent accumulation relationship resembles previous observations of *M. graciosum* in another barnacle host *Semibalanus balanoides*, where parasite abundance increased more strongly with host size at a heavily infected than a less infected site (Carroll et al., 1990). One possible explanation is that variation in parasite supply and exposure determines when size-dependent differences in accumulation are expressed. Under lower exposure, encounter and establishment may dominate variation among hosts, whereas increasing cumulative exposure could reveal differences associated with host size or capacity. Through spring and summer, rapid barnacle growth through the season can re-structure host size distributions for infection (Scrosati & Holt, 2021), while seasonal warming and snail activity can strongly alter cercarial emergence in marine trematodes, including *Maritrema* spp. (Mouritsen, 2002; Poulin, 2006). The population-level increase in parasite load in association with increasingly columnar morphology further implicates density-dependent host structure as crowding alters shell geometry. Crucially, populations undergoing greater morphological change did not show correspondingly greater seasonal increases in parasite load. Thus, persistent differences in population morphology were more closely associated with parasite load than the magnitude of seasonal morphological change, consistent with site-level demographic conditions jointly structuring host form and parasite exposure rather than morphological change alone driving accumulation. Together, these patterns suggest that host demography structures the host phenotypic landscape available to parasites, while variation in parasite supply may determine when differences in accumulation among host sizes become apparent.

Reproductive output

The context dependence of size-dependent parasite accumulation raises a more fundamental question of what ultimately constrains parasite abundance as hosts grow. Scaling theory predicts that within-host parasite capacity may be limited by either the physical space available to parasites or the energetic flux supplied by the host, which may not scale isometrically with host mass (Hechinger, 2013). Empirical tests demonstrated that these alternatives can be distinguished, with ectoparasite loads across birds following predicted geometric scaling with host size while their upper limits were more consistent with energetic than spatial limitation (Hechinger et al., 2019). Our parasite load–somatic mass relationships support the relevance of host size constraints, but also showed that their realised expression is strongly population dependent. Parasite abundance ranged from negatively to strongly hyper-allometrically with somatic mass among the five populations, whereas brood mass followed a much more consistent positive scaling relationship with somatic mass across populations. Parasite load itself neither reduced brood mass nor altered brood-somatic mass scaling after accounting for host size. Thus, we detected no direct reduction in reproductive biomass with increasing infection. Parasite accumulation and reproductive output were instead, coupled through their shared dependence on host somatic mass.

The consequences of this size-dependent coupling depend on what sets the constraint on parasite accumulation. If available somatic space imposes a structural ceiling, parasite accumulation should eventually

saturate with increasing host size or parasite load, producing increasingly hypo-allometric scaling at high parasite loads. Importantly, realised parasite abundance need not approach this capacity. Across helminth–vertebrate systems, maximum parasite biomass scales much more strongly with host mass than average parasite biomass, consistent with many hosts carrying fewer parasites below their potential capacity (Poulin & George-Nascimento, 2007). Consequently, some of the variation among the barnacle populations may reflect differences in parasite supply and density-dependent infection processes discussed above rather than differences in structural capacity itself. Where exposure is sufficiently high, however, host capacity may become increasingly important, with spatial constraint predicting an eventually saturating parasite accumulation, or the size–infection scaling relationship becoming increasingly hypo-allometric as available somatic space is occupied. Conversely, hyper-allometric parasite accumulation would leave parasites concentrated disproportionately in larger hosts that also contribute most strongly to reproductive biomass due to the slightly hyper-allometric brood–somatic mass relationship among the sampled barnacle populations. Scaling infection from individuals to populations can therefore produce consequences that are poorly represented by average parasite load. In *C. fissus*, the castrating barnacle parasite *H. balani* might comprise biomass comparable to that of its hosts, with parasite reproductive production sometimes approaching and substantially altering barnacle reproductive production (Fong et al., 2018). Since the intermediate-sized barnacles were most likely to function reproductively as females, this size dependence infection contributed directly to the population distribution of parasitism. The absence of reduced brood mass in our study therefore should not be interpreted as negligible reproductive consequences, particularly where parasites are concentrated non-randomly among hosts contributing disproportionately to reproduction. Assessing host offspring quality and recruitment can help determine the broader reproductive consequences of *M. gratosum*. Such carry-over effects are plausible in barnacles because maternal condition can alter offspring provisioning and late-stage larval phenotype (Kasten et al., 2019), while field experiments in other sessile marine invertebrates demonstrate offspring size interacting with environmental conditions to predict subsequent survival, growth and reproduction (Marshall et al., 2006; Marshall, 2021). Quantifying egg size, energetic content, larval condition and recruitment would therefore be necessary before excluding reproductive consequences of infection. More fundamentally, the highly variable cyst–somatic mass scaling exponents among populations indicates that host geometry alone cannot determine how parasite accumulation is distributed across host size classes.

Thermal regime

Thermal history provides an alternative explanation to the variation in parasite accumulation beyond the influence of host size. Across the broader British Columbia coastline, host size remained a strong positive predictor of both infection prevalence and parasite load, but temperature explained additional variation in the latter. None of the thermal metrics significantly improved predictions of infection prevalence, whereas metacercarial cyst abundance among infected barnacles increased with positive SST deviations from the site-specific seasonal baseline during the preceding 14–90 days, and decreased with positive air temperature deviations during the most recent 14 days. Greater variability in preceding SST deviations was also associated with higher intensity, although this

relationship was less robust when neighbouring sites were re-sampled as larger geographic groups, indicating limited support for its consistency across independent spatial contrasts. These opposing associations with preceding seawater and recent air temperatures suggest that accumulated infection may integrate temperature sensitive processes operating during aquatic immersion and aerial exposure, potentially over different timescales. In intertidal trematodes, cercarial emergence can depend jointly on temperature, immersion, and tidal timing (Fingerut et al., 2003), while warming can have opposing effects on the production and persistence of infective stages. For example, in *Maritrema subdolum*, increasing seawater temperature from 15 to 25 °C increased cercarial emergence by 11-fold but reduced cercarial longevity (Mouritsen, 2002), whereas emergence in *M. novaezealandensis* responded weakly negatively to warming and varied with immersion conditions (Koprivnikar & Poulin, 2009). The positive association with preceding SST deviations may therefore reflect cumulative opportunities for parasite acquisition during immersion, although temperature-dependent emergence, cercarial survival, and host exposure cannot be distinguished from parasite load alone. The negative recent air temperature association may instead reflect physiological responses of barnacles or infected snail hosts to aerial exposure. Experimental warming increased growth in juvenile *B. glandula* (Nishizaki & Carrington, 2015), whereas reduced barnacle growth and survival at higher shore elevations illustrate how aerial exposure can impose constraints on barnacle performance (Gilman & Rognstad, 2018). This interplay between immersion-dependent exposure and aerial stress is also reflected in the vertical distribution of infection within Burrard Inlet, where intensity peaked at intermediate shore elevations—approximately 2.9 m above chart datum, in the upper mid-intertidal, at all three surveyed sites—rather than increasing monotonically with immersion time. This matches the earlier observations that metacercariae in *Semibalanus balanoides* were most abundant in the central part of the barnacle zone (Carrol et al., 1990). Shorter immersion periods at higher elevations may restrict both cercarial delivery and barnacle feeding opportunities, whereas greater predation and competition lower on the shore could reduce host survival and the time available for cyst accumulation (Connell, 1961, 1970; Ober et al., 2019). In contrast, infection was not structured by the micro-habitat immediately surrounding individual barnacles. Intensity did not vary with a barnacle’s distance from the siphons of its host mussel, nor consistently between barnacles on shells of live mussels and on rock. This suggests that mussel feeding is neither concentrated nor depleted cercariae enough to alter infection (Johnson et al., 2010). This contrasts with the protection that sea anemones impart neighbouring barnacles by consuming the transmission stages of a parasitic isopod (Fong & Kuris, 2017). That effect operates within the reach of the anemone’s tentacles, a spatial scale similar to the ~1 cm separation between the barnacles we compared on each mussel. The lack of a consistent difference between barnacles on mussel shells and on rock further suggest that mussels did not reduce transmission to nearby barnacles. Because cercariae of *M. graciosum* are weak swimmers that remain suspended in the water column (McCarthy et al., 2002), their delivery is likely driven by water movement at scales larger than a single mussel, with uptake depending largely on the barnacle’s own cirral feeding (Irwin et al., 1990). For trematodes with avian definitive hosts, the spatial distribution of bird activity and infected first intermediate hosts can also generate persistent transmission hotspots, potentially producing strong differences in infection risk among nearby host populations (Byers et al., 2008, 2016). Together, the shore height and thermal

associations suggest that infection patterns may emerge from interacting effects of parasite exposure, host physiological performance and survival, rather than temperature-dependent transmission alone.

Scaling infection across changing environments

What, then, predicts parasite infection outcomes in marine hosts? Across this geographically widespread host–parasite system, our findings provide only partial support for the common expectations that “larger or warmer is sicker.” Although host size generally predicted infection, substantial variation in cyst–somatic mass scaling, independent associations with shell geometry, and opposing seawater and air-temperature associations indicate that neither size nor temperature alone adequately describes parasite accumulation across populations. One possibility is that realised infection reflects different limiting processes across ecological conditions. Where cercarial exposure is low, parasite accumulation may remain below host capacity, obscuring size-dependent physiological or structural limits; under greater exposure, internal space, host energetic condition or selective mortality may become more consequential. Importantly, these limits do not necessarily scale proportionally with body size or respond uniformly to thermal change. Although metabolic rate is often approximated to scale with body mass to the power of 0.75, the exponent varies among taxa and physiological conditions, and experimental studies demonstrate that temperature and acclimation can alter metabolic scaling itself (Killen et al., 2010; Ohlberger et al., 2012; Benavente et al., 2022). This plasticity is potentially consequential for infection because warming could change the energetic expenditure associated with increasing host size without producing equivalent changes in the physical space available for parasite accommodation. When resource acquisition and maintenance expenditure scale differently, thermal acclimation could therefore change the host size at which energetic rather than structural limitations become most restrictive. At longer timescales, warming may also modify host growth trajectories and attainable size. In particular, Deutsch et al. (2022) showed that the temperature–size rule across aquatic ectotherms can be explained by differences in how oxygen supply and metabolic demand scale with body size and respond to temperature. Together with temperature-dependent growth and mortality, such responses may re-shape host size distributions, altering population parasite load even if the relationship between individual host size and infection remains unchanged. Thermal history may therefore influence infection through two related but distinguishable pathways, including changing the size-dependent constraints governing accumulation within hosts and changing the demographic distribution of hosts across size classes.

Distinguishing these pathways may help explain why infection allometry varies among populations despite the persistent influence of host size. Thermal mismatch theory has improved disease predictions by accounting for differences between host and parasite thermal performance (Cohen et al., 2020), but relative thermal sensitivities do not reveal whether realized infection is limited by parasite delivery or host capacity, particularly when these processes have different size and thermal dependencies. This distinction becomes increasingly important under climate change because the same warming scenario can produce different epidemiological outcomes depending on the processes represented. For example, experimentally informed models predicted declining prevalence and local

parasite extinction under approximately 2 °C of warming in a marine crab–rhizocephalan system (Gehman et al., 2018), while broader analyses predict increased disease risk in some cooler climates (Cohen et al., 2020). Increasingly frequent marine heatwaves further challenge predictions based on fixed thermal relationships because prior exposure can modify physiological performance in hosts, while acute extremes can affect transmission, survival, and population structure differently (Smith et al., 2023). Marine host–parasite systems in which exposure, host physiology and demographic change can be measured together remain especially valuable for resolving these uncertainties (Byers, 2021). The *B. glandula*–*M. graciosum* system provides an opportunity to do so, as sessile hosts permit known-age cohorts to be followed across contrasting thermal histories, while countable metacercariae that do not reproduce within barnacles allow accumulated infection to be related to host growth, morphology, and survival. Combining these observations with independent measures of cercarial exposure, host energetic condition and internal space could distinguish changes in infection opportunity from changes in the size-dependent limits to accumulation, and determine how both contribute to parasite load across natural population size distributions. Beyond parasitism and symbiosis, such potentially competing constraints may extend to other size-structured interactions because their realised outcomes likewise depend on processes that may not respond similarly to warming. In consumer–resource systems, for example, experimentally measured differences in the thermal sensitivities of energy gain and loss have been shown to predict changes in interaction strength (Álvarez-Codesal et al., 2023), while warming-induced changes in predator–prey performance can shift the relative importance of top-down and bottom-up controls (Meehan & Lindo, 2023). Identifying which process limits an interaction, rather than assuming a fixed scaling relationship, may therefore improve predictions of ecological outcomes across biological scales and changing climates.

Conclusion

Collectively, these findings suggest that host size alone provides an incomplete representation of the constraints on parasite prevalence and intensity, with host geometry, parasite exposure, and thermal history contributing to heterogeneous infection outcome across biological scales. Future research should distinguish the mechanisms underlying these associations, and determine how their relative importance could potentially change across environmental conditions. Linking these individual level- processes to population size structure will ultimately help explain how climate change may re-shape disease vulnerability across coastal systems.

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Author contributions

CDM and KCKM conceived the project under the supervision of ALMG and CDGH, who also secured funding for this project. CDM and J-AH collected the majority of the original data and conducted preliminary analyses. CKHN and KCKM performed the final analyses and prepared the first complete draft of the manuscript.

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Competing interests

The authors declare no competing interests.

Data availability

Data supporting this study are available at <https://github.com/kevinckma/data/blob/main/data-maritrema-gratiosum.xlsx>.

Authors' ORCIDs

Carl K. H. Ng: [0009-0003-1390-1086](https://orcid.org/0009-0003-1390-1086)

Kevin C. K. Ma: [0000-0003-1678-077X](https://orcid.org/0000-0003-1678-077X)

Jiou-An Ho: [0009-0007-7459-8366](https://orcid.org/0009-0007-7459-8366)

Colin D. MacLeod: [0000-0002-5963-3713](https://orcid.org/0000-0002-5963-3713)

Alyssa-Lois M. Gehman: [0000-0003-0595-9950](https://orcid.org/0000-0003-0595-9950)

Christopher D. G. Harley: [0000-0003-4099-943X](https://orcid.org/0000-0003-4099-943X)

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Figures

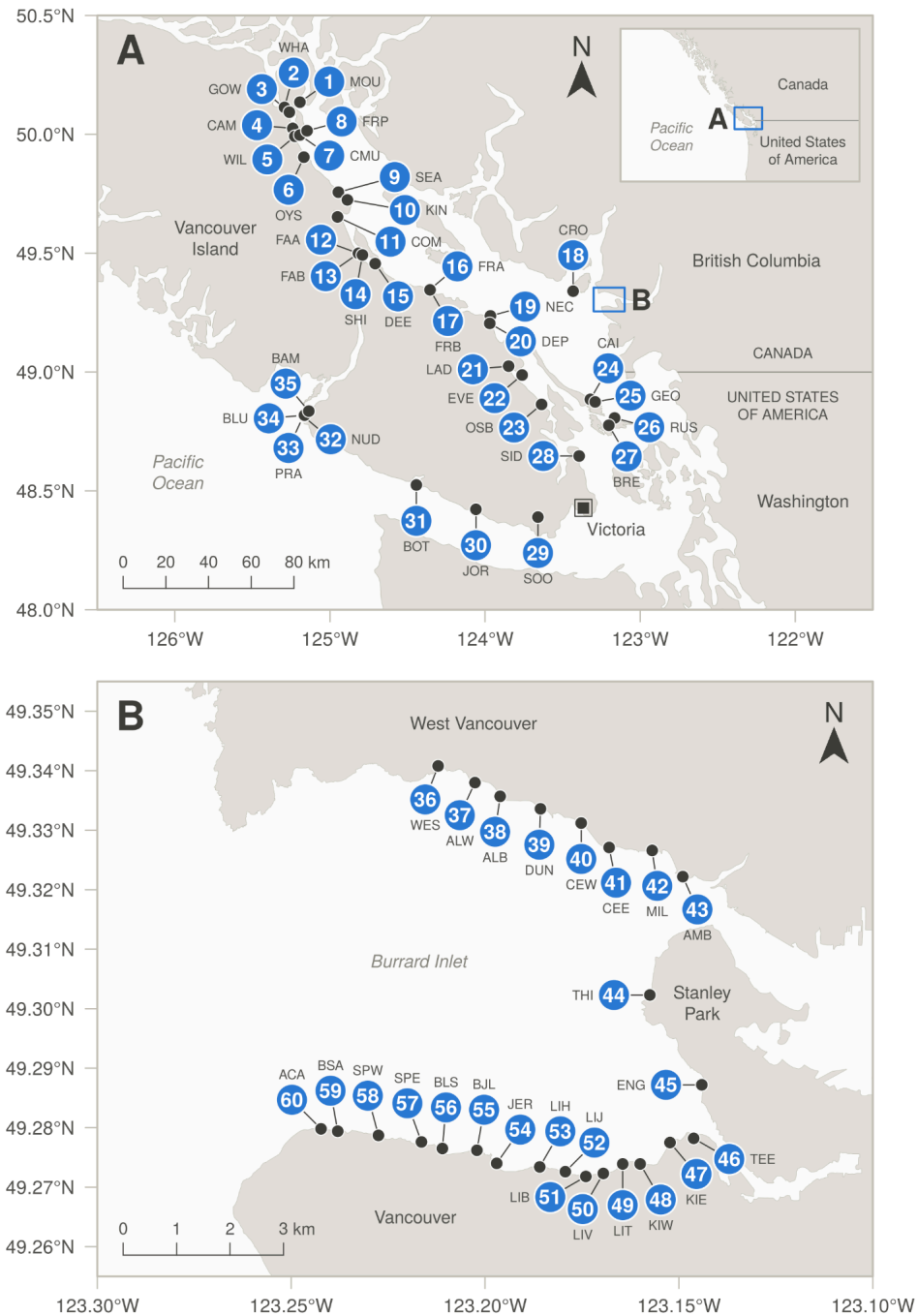


Figure 1 Intertidal sampling sites surveyed for *Maritrema gratosum* along the coast of British Columbia, Canada. (A) Thirty-five sites around Vancouver Island: 1 = Moulds Bay (MOU), 2 = north of Whalebone Cove (WHA), 3 = Gowlland Harbour (GOW), 4 = south of Campbell River (CAM), 5 = north of Willow Point (WIL), 6 = north of Oyster Bay (OYS), 7 = Cape Mudge (CMU), 8 = Francisco Point (FRP), 9 = Seal Bay (SEA), 10 = Kin Beach

(KIN), 11 = Comox Harbour (COM), 12 = Fanny Bay A (FAA), 13 = Fanny Bay B (FAB), 14 = Ship Point (SHI), 15 = south of Deep Bay (DEE), 16 = French Creek A (FRA), 17 = French Creek B (FRB), 18 = Cape Roger Curtis (CRO), 19 = Neck Point (NEC), 20 = Departure Bay (DEP), 21 = Ladysmith Harbour (LAD), 22 = Evening Cove (EVE), 23 = Osborn Bay (OSB), 24 = Cain Point (CAI), 25 = Georgina Point (GEO), 26 = Russell Reef (RUS), 27 = Breezy Bay (BRE), 28 = Sidney (SID), 29 = Sooke (SOO), 30 = west of Jordan River (JOR), 31 = Botanical Beach (BOT), 32 = Nudibranch Point (NUD), 33 = Prasiola Point (PRA), 34 = Bluestone Point (BLU), 35 = Bamfield Inlet (BAM). (B) Inset showing twenty-five sites around Burrard Inlet in Vancouver and West Vancouver: 36 = West Bay Park (WES), 37 = west of Altamont Beach (ALW), 38 = Altamont Beach (ALB), 39 = west of Dundarave Beach (DUN), 40 = west side of Centennial Seawalk (CEW), 41 = east side of Centennial Seawalk (CEE), 42 = Millennium Park (MIL), 43 = Ambleside Beach (AMB), 44 = Third Beach (THI), 45 = English Bay Beach (ENG), 46 = Teetotaler Beach (TEE), 47 = east of Kitsilano Beach (KIE), 48 = west of Kitsilano Beach (KIW), 49 = Linnea Beach at Trafalgar Street (LIT), 50 = Linnea Beach at Volunteer Park (LIV), 51 = Linnea Beach at Balaclava Street (LIB), 52 = Linnea Beach at Jean Beaty Park (LIJ), 53 = Linnea Beach at Hastings Mill Park (LIH), 54 = Jericho Beach (JER), 55 = between Jericho and Locarno Beaches (BJL), 56 = between Locarno and Spanish Banks Beaches (BLS), 57 = east side of Spanish Banks Beach (SPE), 58 = west side of Spanish Banks Beach (SPW), 59 = between Spanish Banks and Acadia Beaches (BSA), 60 = Acadia Beach (ACA). See Supplementary Table S1 for coordinates of each site.

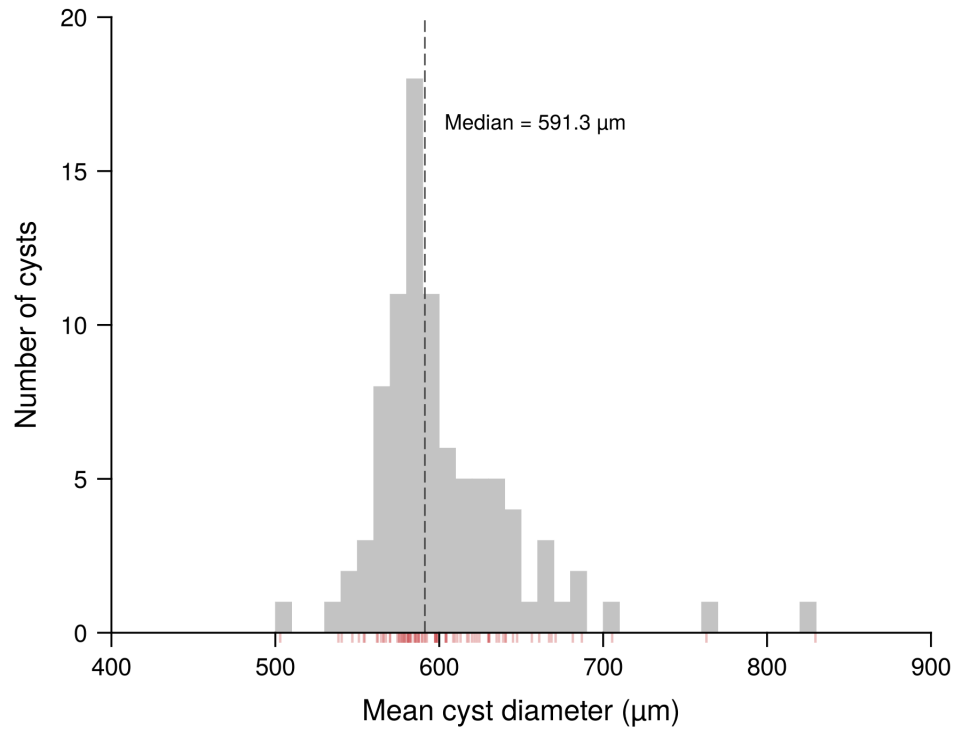


Figure 2 Size distribution of metacercarial cysts of the trematode *Maritrema graciosum* extracted from the barnacle *Balanus glandula* that were collected from Burrard Inlet, British Columbia, Canada. Bin width = 10 μm. The dashed line marks the median diameter. Red tick marks along the x-axis show individual metacercarial cysts.

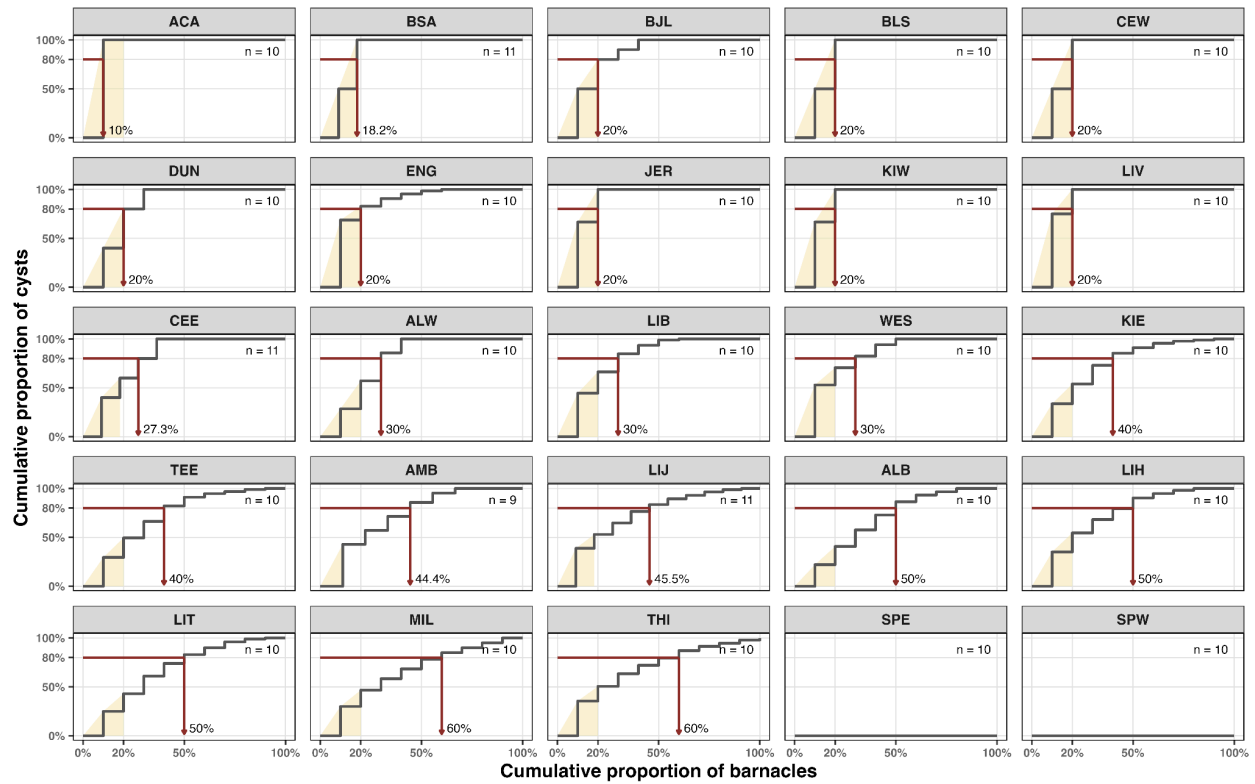


Figure 3 Spatial variation in the aggregation of parasite load among barnacle hosts from 25 sites in May ($n = 252$). Cumulative parasite load curves are shown for each sampling site, with individual barnacles ranked from highest to lowest parasite load. Red horizontal lines identify the proportion of hosts required to account for 80% of the total parasite load within each site, with the corresponding host percentage indicated by the arrow. The yellow shaded regions denote the most heavily infected host (20%), and n indicates the number of sampled individuals at each site. Sites are ordered in ascending order by the proportion of hosts required to reach 80% of local parasite load. Sites without any infection recorded are displayed without an 80% threshold. See Supplementary Table S1 for site names and coordinates.

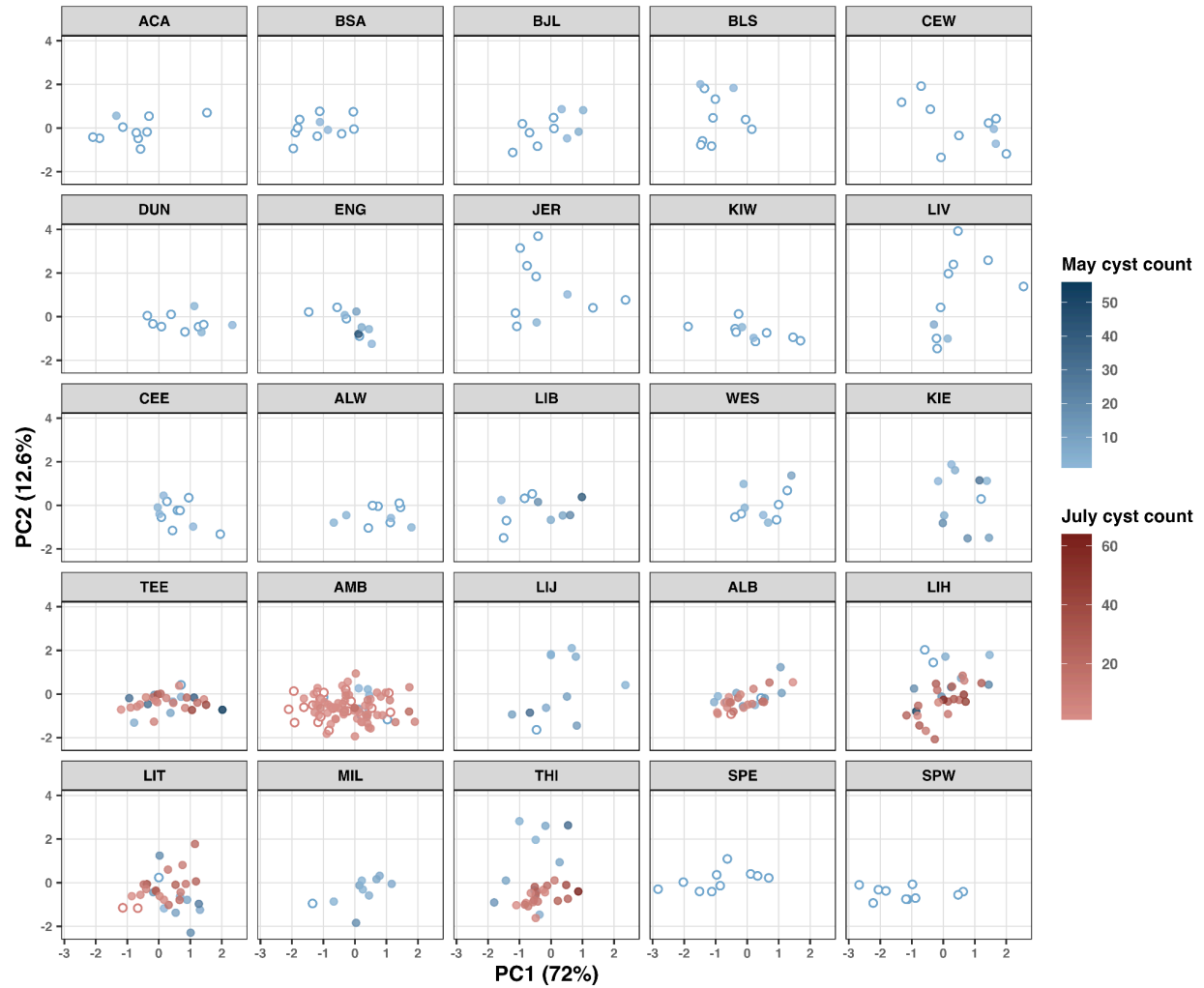


Figure 4 Host morphometrics and parasite load across sites in May and July. Parasite load was mapped onto the two dimensional morphological space derived from all May-sampled barnacles across 25 sites. July observations were subsequently projected onto the same PC axes to enable direct comparison of host morphology and parasite load across months. Points represent individual barnacles, colour intensity indicates parasite load, while open circles indicate uninfected individuals. Sites are ordered from upper left to bottom right, corresponding to the strongest to weakest parasite aggregation in May. See Supplementary Table S1 for site names and coordinates.

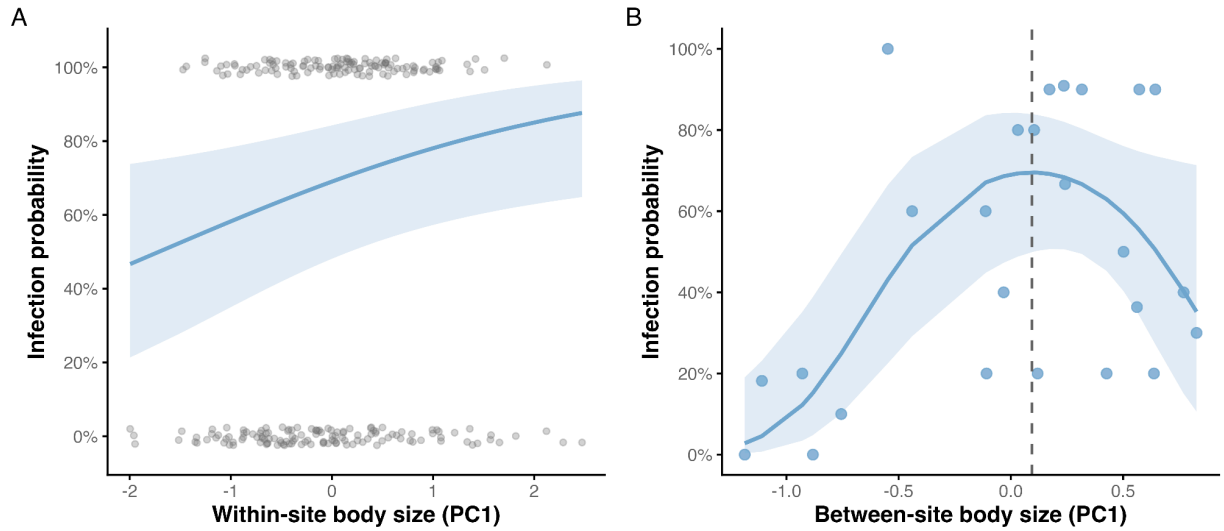


Figure 5 Associations between host morphological variation and *Maritrema infection* prevalence in *B. glandula* collected from 25 sites in Burrard Inlet, British Columbia, in May 2025. A) Within-site PC1, representing individual host size relative to site mean and (B) between-site PC1, reflecting site-average host size, were evaluated as predictors of infection status (absence–presence of cyst). Grey points represent individual barnacles. Solid blue lines show predicted infection probabilities from a binomial generalized linear mixed model, with shaded bands representing 95% confidence intervals. The dashed vertical line in (B) indicates the estimated PC1 value at which infection probability is highest.

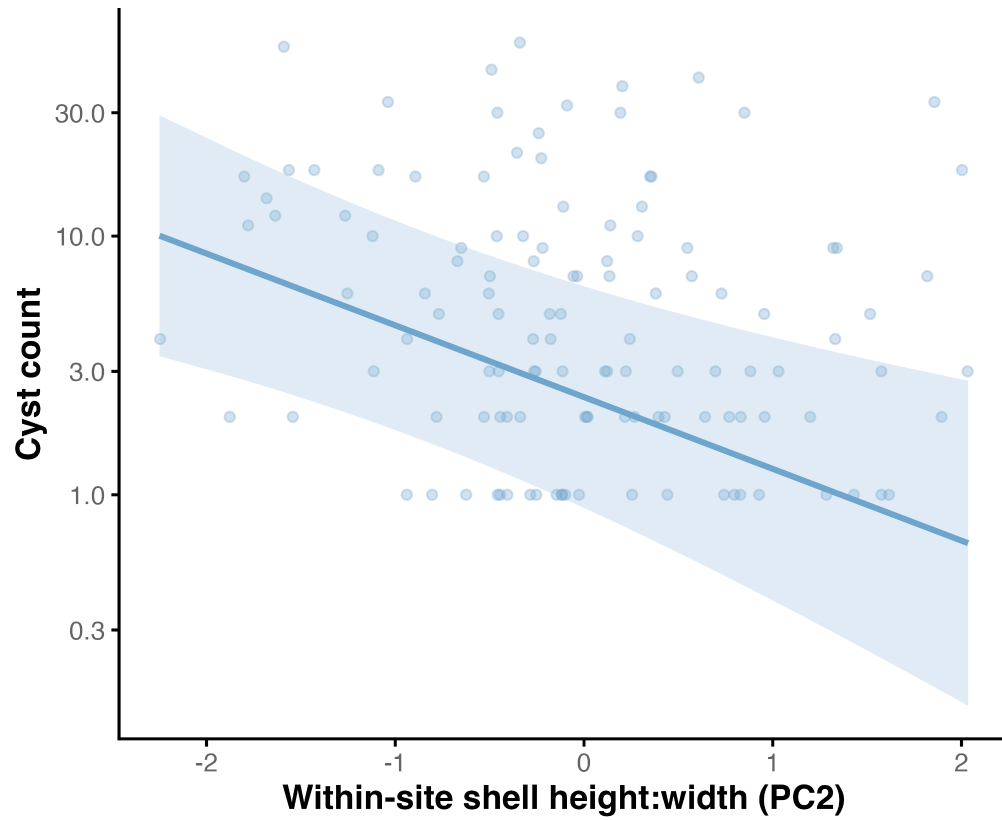


Figure 6 Relationship between within-site host morphological variation (PC2) and *Maritrema* infection intensity in infected *B. glandula* collected from 25 sites in Burrard Inlet, British Columbia, in May 2025. Blue points represent individual infected barnacles, while the solid blue line shows predicted parasite load (cyst counts) from the zero-truncated negative-binomial GLMM, with the shaded area indicating the 95% confidence interval. Metacercarial cyst counts are displayed on a $\log_{10}$ scale.

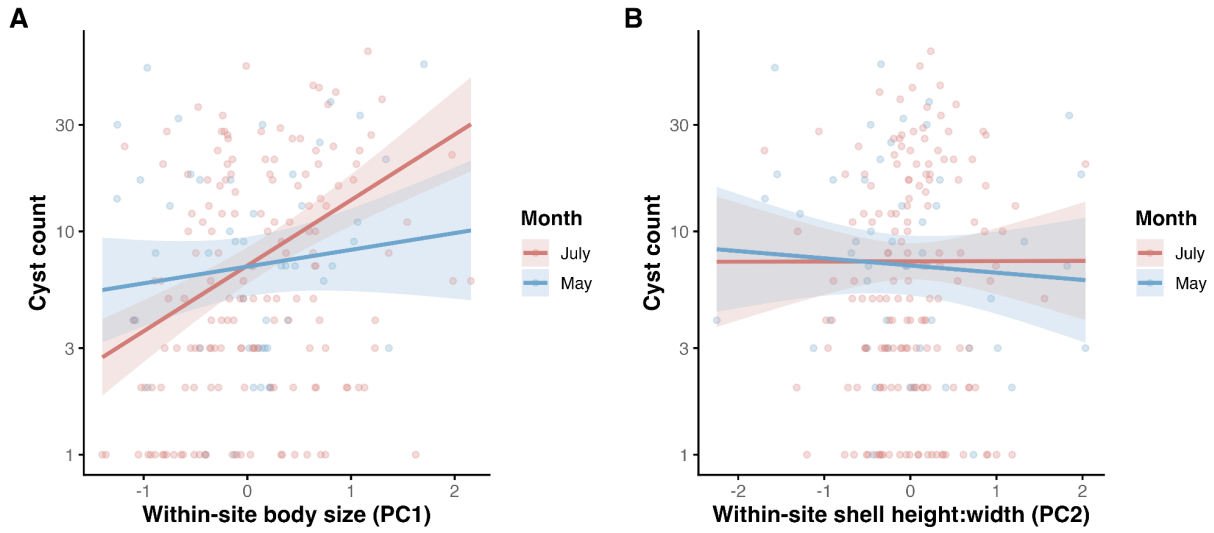


Figure 7 Relationship between local individual host morphology and *Maritrema* infection in *B. glandula* sampled in May and July 2025. (A) Within-site PC1 reflects local variation in host size and (B) represents local variation in shell shape. PC scores are expressed as deviations from the corresponding site x month mean. Blue points represent individual infected barnacles from May and red for July. Solid lines show predicted parasite load (cyst counts) from the fitted zero-truncated negative-binomial GLMM, with shaded areas indicating 95% confidence intervals. Metacercarial cyst counts are displayed on a log₁₀ scale.

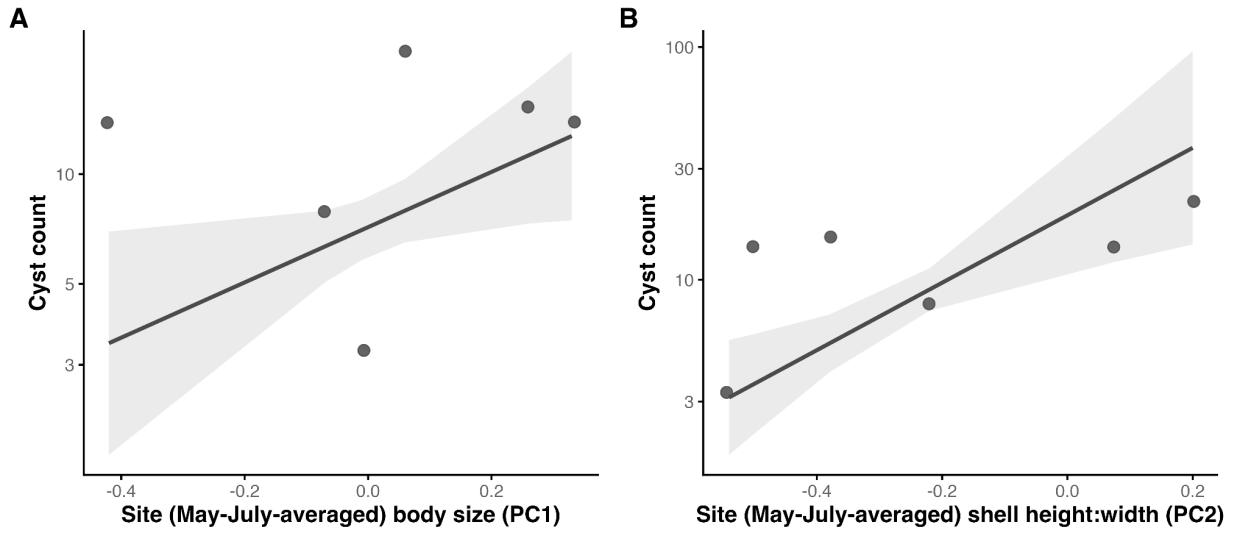


Figure 8 Relationship between averaged host morphology and *Maritrema* infection intensity across sampled *B. glandula* populations in May and July 2025. (A) Site-level PC1, capturing host size, and (B) site-level PC2, representing shell shape. Site-level PC scores reflect the average morphology across the two sampling months. Points indicate mean cyst counts among infected barnacles at each site, pooled across May and July. Solid lines represent population-level predictions from the zero-truncated negative-binomial GLMM, with shaded areas indicating 95% confidence intervals. Metacercarial cyst counts are displayed on a log₁₀ scale.

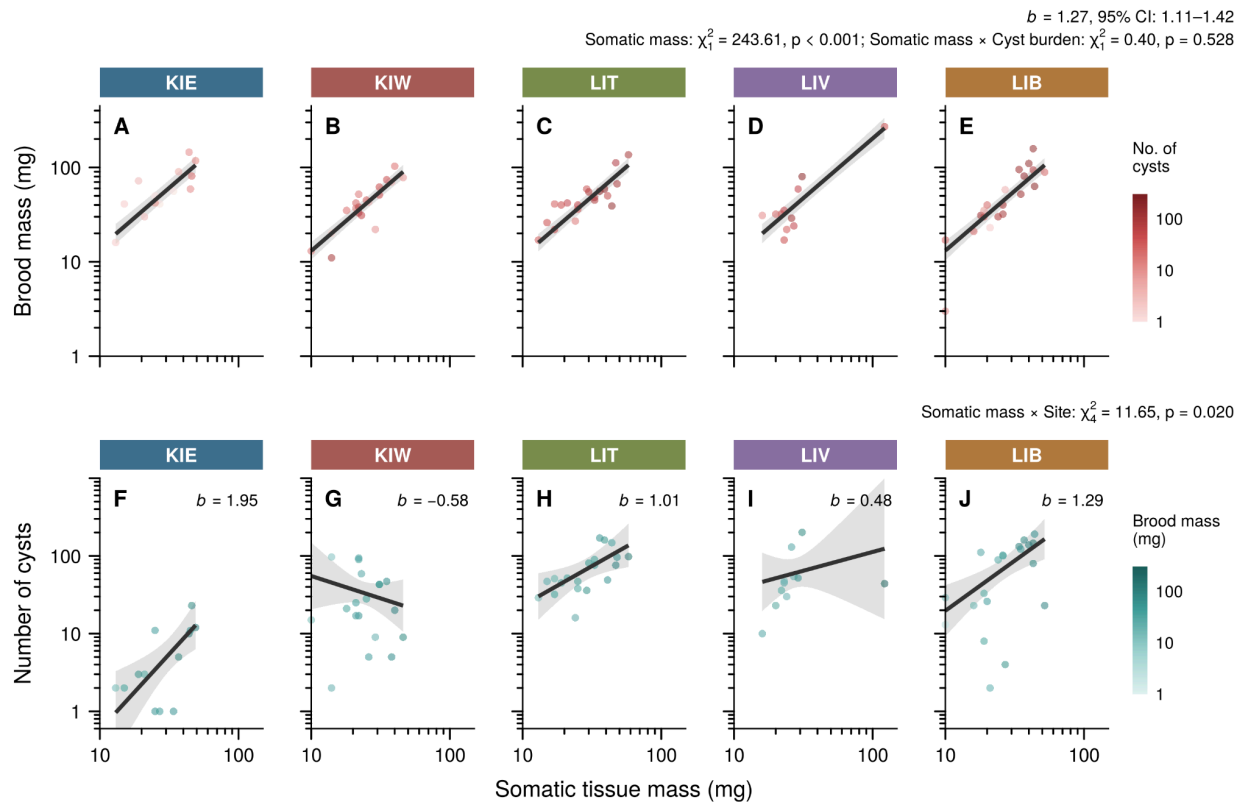


Figure 9 Brood mass and parasite load in relation to somatic tissue mass of the barnacle *Balanus glandula* infected with the trematode *Maritrema gratosum* at five sites in Burrard Inlet, British Columbia, Canada (KIE, KIW, LIT, LIV, and LIB), collected in 2021. (A–E) Brood mass against somatic tissue mass. Lines show the fitted scaling relationship at each site's mean number of metacercarial cysts. (F–J) Number of cysts against somatic tissue mass. Lines show the site-specific fitted scaling relationship. Shaded areas around the lines represent 95% confidence intervals. Axes are on a $\log_{10}$ scale. b is the scaling exponent; $b = 1$ indicates isometric scaling. See Supplementary Table S1 for site names and coordinates.

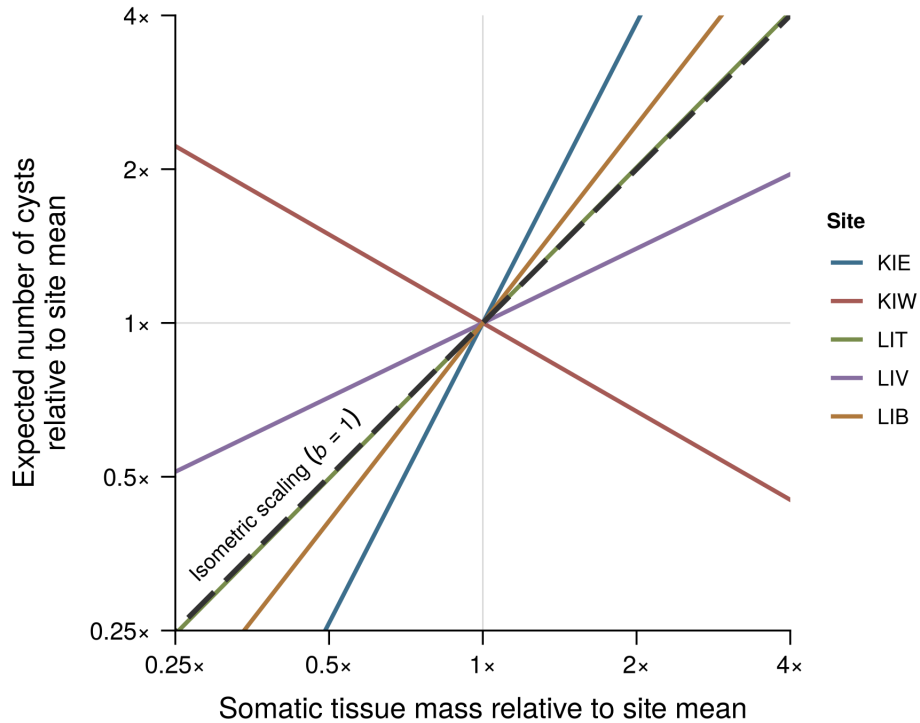


Figure 10 Scaling of parasite load with somatic tissue mass in the barnacle *Balanus glandula* infected with the trematode *Maritrema gratosum* at five sites in Burrard Inlet, British Columbia, Canada (KIE, KIW, LIT, LIV, and LIB), collected in 2021. Both axes are relative to each site's mean, so 1× on each axis is a barnacle of average somatic tissue mass carrying the expected number of metacercarial cysts for that site. Lines show the site-specific fitted scaling relationships: KIE, $b = 1.95$; KIW, $b = -0.58$; LIT, $b = 1.01$; LIV, $b = 0.48$; LIB, $b = 1.29$. The dashed line marks isometric scaling ($b = 1$). Lines steeper than the dashed line mean larger barnacles carry proportionally more cysts, and flatter lines mean proportionally fewer. Axes are on a $\log_2$ scale. See Supplementary Table S1 for site names and coordinates.

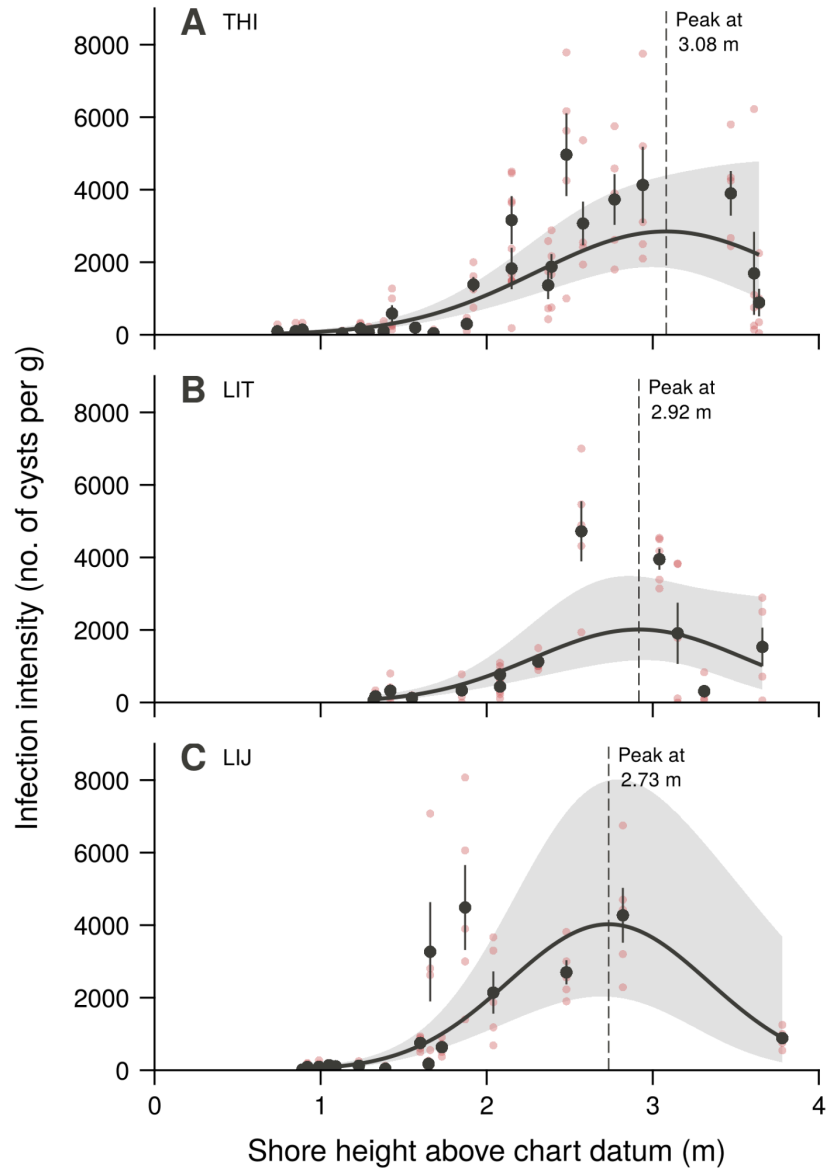


Figure 11 Infection intensity (number of metacercarial cysts per g of somatic tissue) in the barnacle *Balanus glandula* in relation to shore height, sampled at 5-m intervals, at three sites in Burrard Inlet, British Columbia, Canada (THI, LIT, LIJ). Red points show individual barnacles ($n = 5$ per interval). Black points show interval means $\pm$ standard error. Lines and shaded bands show fitted quadratic relationships with 95% confidence intervals from a Tweedie GLMM. Dashed lines mark the estimated shore height of peak infection intensity at each site. See Supplementary Table S1 for site names and coordinates.

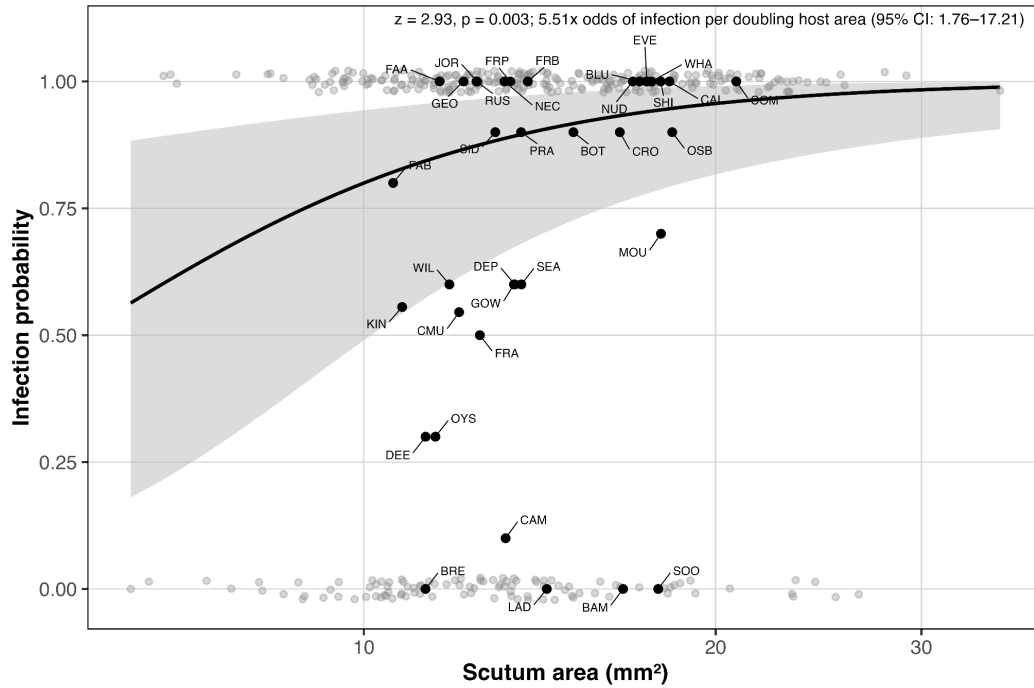


Figure 12 Relationship between host scutum area and infection status in *Balanus glandula* across 35 coastal populations in British Columbia. Grey points represent individual barnacles, while black points indicate observed site-level prevalence, labelled by site code. The solid black line represents predicted infection probability from the binomial GLMM. Predictions represent the population-level fixed effects, with the grey shaded area indicating the 95% confidence interval. Host scutum area is displayed on a $\log_{10}$ scale. See Supplementary Table S1 for site names and coordinates.

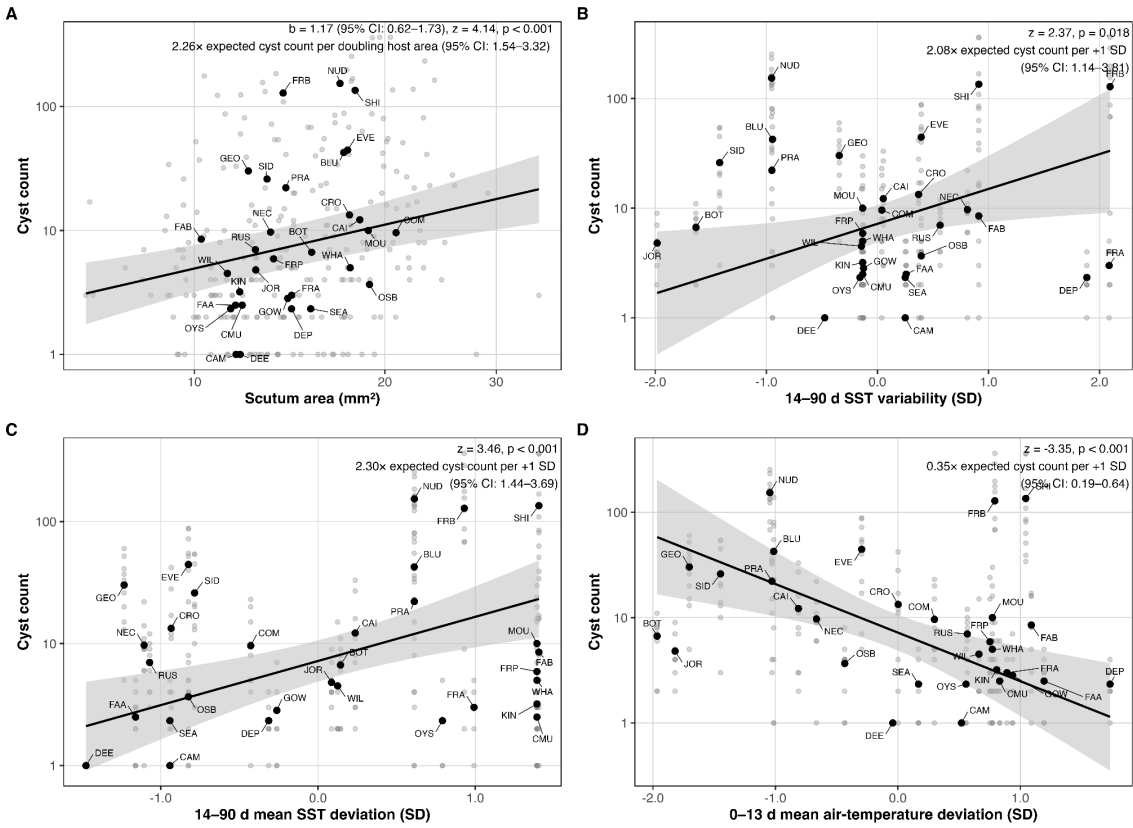


Figure 13 Relationships between *Maritrema gratosum* infection intensity and (A) host scutum area displayed on a logarithmic scale, (B) 14–90 d SST variability, (C) 14–90 d mean SST deviation, (D) and 0–13 d mean air-temperature deviation in *Balanus glandula* across 31 coastal sites in British Columbia. Grey points represent individual infected barnacles, while black points indicate observed site-level mean parasite count, labelled by site code. Solid black lines represent fixed-effect predictions from the zero-truncated GLMM, with grey shaded areas indicating 95% confidence intervals. Parasite load (cyst counts) are displayed on a $\log_{10}$ scale in all panels. Thermal metrics are expressed in SDs from their respective means across sites. See Supplementary Table S1 for site names and coordinates.

Supplementary tables

Supplementary Table S1 Names and coordinates of the 60 intertidal sites surveyed for *Maritrema gratosum* along the coast of British Columbia, Canada. Site numbers correspond to Figure 1.

Site name	Site number	Site code	Latitude (N)	Longitude (W)
Moulds Bay	1	MOU	50°08'07.3"	125°11'37.7"
North of Whalebone Cove	2	WHA	50°06'48.2"	125°17'35.6"
Gowlland Harbour	3	GOW	50°05'35.2"	125°15'41.6"
South of Campbell River	4	CAM	50°01'29.7"	125°14'23.8"
North of Willow Point	5	WIL	49°59'29.4"	125°13'34.4"
North of Oyster Bay	6	OYS	49°54'14.0"	125°10'05.8"
Cape Mudge	7	CMU	49°59'48.8"	125°11'38.9"
Francisco Point	8	FRP	50°00'53.7"	125°08'52.1"
Seal Bay	9	SEA	49°45'23.7"	124°56'44.7"
Kin Beach	10	KIN	49°43'26.4"	124°53'15.4"
Comox Harbour	11	COM	49°39'08.5"	124°57'04.5"
Fanny Bay A	12	FAA	49°29'56.6"	124°48'59.2"
Fanny Bay B	13	FAB	49°29'56.6"	124°48'59.2"
Ship Point	14	SHI	49°29'33.0"	124°47'25.8"
South of Deep Bay	15	DEE	49°27'21.6"	124°42'29.9"
French Creek A	16	FRA	49°20'49.8"	124°21'19.7"
French Creek B	17	FRB	49°20'44.3"	124°21'17.2"
Cape Roger Curtis	18	CRO	49°20'24.3"	123°25'56.0"
Neck Point	19	NEC	49°14'10.5"	123°57'55.2"
Departure Bay	20	DEP	49°12'16.1"	123°58'11.4"
Ladysmith Harbour	21	LAD	49°01'29.3"	123°50'52.9"
Evening Cove	22	EVE	48°59'14.8"	123°45'39.7"
Osborn Bay	23	OSB	48°51'49.5"	123°38'08.4"
Cain Point	24	CAI	48°53'05.3"	123°19'17.3"

Georgina Point	25	GEO	48°52'27.2"	123°17'24.7"
Russell Reef	26	RUS	48°48'24.1"	123°09'53.3"
Breezy Bay	27	BRE	48°46'33.1"	123°12'07.5"
Sidney	28	SID	48°38'48.2"	123°23'36.5"
Sooke	29	SOO	48°23'22.8"	123°39'32.4"
West of Jordan River	30	JOR	48°25'20.6"	124°03'31.5"
Botanical Beach	31	BOT	48°31'29.3"	124°26'33.9"
Nudibranch Point	32	NUD	48°48'51.4"	125°10'18.5"
Prasiola Point	33	PRA	48°49'03.1"	125°10'09.8"
Bluestone Point	34	BLU	48°49'06.5"	125°09'49.8"
Bamfield Inlet	35	BAM	48°50'07.7"	125°08'10.7"
West Bay Park	36	WES	49°20'26.9"	123°12'43.6"
West of Altamont Beach	37	ALW	49°20'16.8"	123°12'09.4"
Altamont Beach	38	ALB	49°20'08.5"	123°11'46.0"
West of Dundarave Beach	39	DUN	49°20'01.0"	123°11'08.5"
West side of Centennial Seawalk	40	CEW	49°19'52.3"	123°10'30.7"
East side of Centennial Seawalk	41	CEE	49°19'37.6"	123°10'04.8"
Millennium Park	42	MIL	49°19'35.8"	123°09'24.8"
Ambleside Beach	43	AMB	49°19'19.9"	123°08'56.4"
Third Beach	44	THI	49°18'08.3"	123°09'27.0"
English Bay Beach	45	ENG	49°17'13.9"	123°08'38.8"
Teetotaler Beach	46	TEE	49°16'41.5"	123°08'46.3"
East of Kitsilano Beach	47	KIE	49°16'39.0"	123°09'08.3"
West of Kitsilano Beach	48	KIW	49°16'26.0"	123°09'36.0"
Linnea Beach at Trafalgar Street	49	LIT	49°16'26.0"	123°09'52.2"
Linnea Beach at Volunteer Park	50	LIV	49°16'20.3"	123°10'10.2"
Linnea Beach at Balaclava Street	51	LIB	49°16'18.5"	123°10'26.4"
Linnea Beach at Jean Beaty Park	52	LIJ	49°16'21.4"	123°10'45.5"
Linnea Beach at Hastings Mill Park	53	LIH	49°16'24.2"	123°11'09.2"

Jericho Beach	54	JER	49°16'26.4"	123°11'49.2"
Between Jericho and Locarno Beaches	55	BJL	49°16'34.3"	123°12'07.6"
Between Locarno and Spanish Banks Beaches	56	BLS	49°16'35.4"	123°12'39.6"
East side of Spanish Banks Beach	57	SPE	49°16'39.4"	123°12'59.0"
West side of Spanish Banks Beach	58	SPW	49°16'43.3"	123°13'39.0"
Between Spanish Banks and Acadia Beaches	59	BSA	49°16'45.8"	123°14'16.8"
Acadia Beach	60	ACA	49°16'47.3"	123°14'32.3"
