Supporting Information

Appendix S1

Additional information on model selection, the DEB model, and supplemental data

In this appendix, we describe the competition among models presented in Table 1 in more detail. Then, we present the dynamic energy budget (DEB) model for parasitism. We sketch the model structure, as we have presented before (Hall *et al.* 2010a, Hall *et al.* 2009b) and describe the modifications and assumptions made here to incorporate the physiological effects of *Chaoborus* kairomones. Finally, we present model predictions and data on survival of infected hosts in the life table experiment.

Model competition

We fit several models to estimate susceptibility of hosts to infection by the yeast. The general structure of the model for the susceptibility assays was:

$$dS/dt = -TR_{j,k} \times S_{j,k} \times Z \qquad \text{(S1)}$$

where $TR_{j,k}$ is per host, per spore transmission rate (susceptibility), $S_{j,k}$ is the density of susceptible hosts (host·L^{-1}), and Z is the density of spores (spores·L^{-1}). Subscript j denotes clonal identity, from 1 to 9, while subscript k indicates control (n) or *Chaoborus* kairomone (c) treatments. This model says that susceptible hosts decrease as they become infected after contacting spores. Change in infected hosts (I) mirrors the equation for S (i.e., $dI/dt = -dS/dt$), and for simplicity we assume that spore density remains constant during the assays (i.e., $dZ/dt = 0$).

The seven competing models (Table 1) differ in their assumptions about susceptibility. Two different formulations exist for this $TR_{j,k}$ term; if body length (L) is included, then:

$$TR_{j,k} = \beta_{j,k} L^4 \quad \text{(S2)}$$

and $\beta_{j,k}$ is the size-specific susceptibility parameter (with units $L \cdot mm^{-4} \cdot spore^{-1} \cdot day^{-1}$). If body length is not a factor, then $TR_{j,k}$ simply equals $\beta_{j,k}$ (with units $L \cdot spore^{-1} \cdot day^{-1}$). Model 1 estimates parameters for each genotype j and includes body length (i.e., uses equ. S2); no separate parameters are estimated for treatment k (i.e., $\beta_{1,n} = \beta_{1,c}$, $\beta_{2,n} = \beta_{2,c}$, etc.). Rather, the effect of *Chaoborus* enters this model based on *Chaoborus*-induced changes in host body length. Model 2 assumes $TR_{j,k} = \beta_{j,k}$, then again estimates separate parameters for each genotype but not the kairomone treatment. This model assumes that clonal identity alone drives variation in susceptibility among host clones. Model 3 estimates parameters for each genotype and kairomone treatment (i.e., $\beta_{1,n}$, $\beta_{1,c}$, $\beta_{2,n}$, $\beta_{2,c}$, etc.). This allows for the possibility that *Chaoborus* alter susceptibility due to factors other than body length (termed "Additional *Chaoborus* Effects" in Table 1). Similarly, model 4 imagines that genotype and "Additional Chaoborus Effects" drive susceptibility results; however, it does not include body size in the parameter estimates (but it does produce identical AIC-based results in Table 1; we include it here for completeness). Model 5 assumes that only body size should influence susceptibility (i.e., $\beta_{1,n} = \beta_{1,c} = \beta_{2,n} = \beta_{2,c} =$ … etc., where $\beta_{j,k}$ follows equ. S2); thus, no differences in susceptibility among genotypes is assumed. Model 6 assumes that susceptibility depends on body length (i.e., equ. S2 applies) and the additional *Chaoborus* kairomone effect, but differences among genotypes do not exist (i.e., $\beta_{1,n} = \beta_{2,n} \ldots = \beta_{9,n}$, and $\beta_{1,c} = \beta_{2,c} \ldots = \beta_{9,c}$). Model 7, the null case, assumes that none of these factors applies (i.e., each has the same susceptibility, regardless of genetic identity, kairomone-

induced changes in body size, or "additional *Chaoborus* effects"; $TR_{j,k} = \beta_{j,k}$ and $\beta_{1,n} = \beta_{1,c} = \beta_{2,n} = \beta_{2,c} = \ldots = \beta_{9,c}$).

Given these different models, the susceptibility parameters $\beta_{j,k}$ were estimated by comparing data on prevalence of infection to that predicted by the model, $p_I(t)$, where:

$$p_I(t) = \frac{I(t)}{S(t) + I(t)} \quad \text{(A3)}$$

and where $I(t)$ and $S(t)$ are densities of the two classes of hosts after exposure to the parasite for t units of time (1 day). To find the best maximum likelihood estimates (MLE) of the parameters $\beta_{j,k}$, we assumed that the error in the observed prevalence of infection in the assays was binomially distributed. The binomial error distribution applies to situations in which only two outcomes (i.e., infected or not-infected) occur in trials repeated N times (where N is the number of hosts in each beaker). If p_I is the predicted prevalence (probability) of infection of a host (equ. S3), then I hosts become infected among all N hosts within a beaker with probability $p(I,N)$:

$$p(I,N) = \binom{N}{I} {p_I}^I (1 - p_I)^{N-I}. \quad \text{(A4)}$$

This binomial distribution (equ. S4) provides the likelihood of the outcome observed in each beaker, given the data and prevalence predicted by the parameters. Over the entire experiment, one can then sum the negative log-likelihood of the results from each beaker; the MLE of the parameters ($\beta_{j,k}$) minimizes the summed negative log likelihood of the experiment. These MLE parameters were located using a standard search algorithm (Nelder-Mead downhill simplex) as implemented by Matlab 2009.b.

68 **The dynamic energy budget model**

69 A dynamic energy budget (DEB) model for parasitism connects allocation to growth (i.e.,
70 kairomones), genetic variation in feeding rate (i.e., variation in susceptibility among clones), and
71 host energetics to other epidemiological parameters. This model, based on Kooijman (1993),
72 tracks flow of energy from ingestion and assimilation to storage in a "reserve" pool. That
73 reserve energy is used (catabolized) for growth, reproduction in adults or development in
74 juveniles, and associated metabolic costs. However, parasites take energy from the reserve of
75 hosts and replicate within hosts. Through this energy consumption, parasites exact virulent costs
76 on growth and reproduction of their hosts. Furthermore, the parasite kills its host once parasite
77 mass reaches a certain threshold, a proportion of structural mass of the host (as discussed and
78 justified empirically in Hall *et al.* 2009a). Before killing it, however, the parasite can inflict
79 energetic stress on its host by drawing down internal energy reserves. The DEB model predicts
80 the implications of this energy depletion for growth, reproduction, and survival.
81
82 Derivation
83 The DEB model tracks energy flow through hosts and parasites. Hosts first eat food, then
84 assimilate some fraction of it. Assimilation rate (A) is given by:
85 $$A = aL^2 X/(h + X); \quad \text{(S5)}$$
86 A depends on size-specific assimilation rate, a, which itself is the product of size-specific
87 maximal feeding rate, f, and conversion efficiency, ε. Assimilation rate A also depends on
88 surface area of the host, proportional to L^2, and on feeding on algal food (X) following a type-II
89 functional response with a half-saturation constant (h). Assimilated energy is then put into a

reserve energy pool (E). Reserve energy (E), in turn, is modeled as the product of energy density (e) and structural mass (W) so that $E = eW$. The change through time of this energy pool, then:

$$\frac{dE}{dt} = \frac{d(eW)}{dt} = W\frac{de}{dt} + e\frac{dW}{dt} \quad \text{(S6)}$$

involves two components. First, there is a change in the reserve density per unit structural mass (involving the de/dt term), then the host grows more structure (the dW/dt term). Following Kooijman (1993), we assume homeostasis of reserves, meaning that the animal regulates the reserve density at a level related to its feeding rate. Change in reserve density (de/dt) increases with assimilation and decreases linearly with e (i.e., according to first-order kinetics):

$$\frac{de}{dt} = \frac{A}{W} - \left(\frac{aL^2}{e_M W}\right)e \quad \text{(S7)}$$

where e_M is the maximum density of energy. Once equations (S5)-(S7) are combined, utilization rate (C) of energy becomes (by definition):

$$C = A - \frac{dE}{dt} = E\left(\frac{aW^{2/3}}{e_M W} - \frac{dW}{Wdt}\right). \quad \text{(S8)}$$

Under normal circumstances, the host allocates these catabolized energy reserves towards growth versus reproduction if the host is mature or maturation if it is juvenile following the kappa (κ)-rule. According to the kappa rule (Kooijman 1993), a fixed proportion (κ) of utilized energy is allocated to growth, and a proportion (1- κ) towards reproduction. In mathematical terms, the host devotes utilized energy to growth at rate:

$$\kappa C = g(dW/dt) + mW \quad \text{(S9)}$$

where the first term on the right-hand side denotes growth of structural mass (dW/dt) with associated cost of growing (g), and the second term represents costs to maintain current mass (at

rate m). We solved both equations (S8) and (S9) for C, set them equal to each other, then solved for the dW/dt term to yield:

$$\frac{dW}{dt} = W\left[\frac{\kappa a L^2 E/(e_M W) - mW}{\kappa E + gW}\right]. \quad \text{(S10)}$$

The rest of the catabolized energy reserves, $(1-\kappa)C$, are used for reproduction and associated costs. The rate of reproduction, dR/dt, is then:

$$dR/dt = (q/E_0)[(1-\kappa)C - ((1-\kappa)/\kappa)mW_P] \quad \text{(S11)}$$

where q is the cost of converting energy reserve of the mother into the energy reserve of the offspring ($0 < q < 1$, where lower q indicates higher cost), and E_0 converts energy to offspring. This equation also includes a second term (in brackets) for "maturity maintenance" (see Kooijman 1993; W_P denotes size at maturation). The DEB model for the host, then, consists of equations (S6), (S10), and (S11).

We then add the parasite growing within the host. This parasite (N) feeds on energy reserves of its host (E) according to its own saturating (type II) functional response. Thus, reserve dynamics change (from equ. S6) to:

$$\frac{dE}{dt} = W\frac{de}{dt} + e\frac{dW}{dt} - \frac{a_N}{\varepsilon_N}\left(\frac{E}{h_N + E}\right)N \quad \text{(S12)}$$

where consumption by parasites (last term) is governed by a half-saturation constant (h_N), maximal assimilation rate (a_N), and conversion efficiency (ε_N) of the parasite. This parasite then grows according to a classic equation for a resource consumer (Grover 1997):

$$\frac{dN}{dt} = a_N\left(\frac{E_N}{h_N + E_N}\right)N - m_N N \quad \text{(S13)}$$

where m_N lumps various loss rates (e.g., maintenance, death) of the parasite.

130 This model requires a few other pieces of biology (see Hall *et al.* 2009b for the
131 mathematical details). First, an equation for food dynamics follows our experimental protocol
132 (below): non-reproducing food is consumed by hosts but replenished daily. Second, parasite
133 growth within a host can inflict "moderate" and "severe" energetic stress on the host. As
134 parasites draw down energy within a host, they can first stop growth of the host (moderate
135 energetic stress), but then stop reproduction (severe energetic stress). These changes update the
136 kappa-rule for allocation of utilized energy. Third, the parasite kills the host once it reaches a
137 physical threshold ($N = \rho W$, where ρ denotes a mechanical limit of the host to support the
138 parasite). Once this threshold is crossed, the animal stops eating (i.e., $f = 0$). Then, energy
139 reserve (E) drops to zero and the host dies. (The parasite cannot drop E to zero itself because its
140 own minimal energy reserve requirements exceed zero). Finally, starting parasite density (P_0)
141 within a host of an initial size (L_0) equaled that consumed over a 24 hour period. Thus, hosts
142 with higher rates of the feeding metric started with more parasite internally than those with lower
143 rates. Parameter values used are summarized in Table S1.
144
145 Key assumptions for this study
146 We assumed that *Chaoborus* kairomones boosted the kappa (κ) parameter, i.e., they
147 induced the host to allocate more energy reserve to growth rather than reproduction. Without
148 any data on kappa, we just varied it from 0.20 (baseline) to 0.25 to illustrate the effect of this
149 reallocation. Thus, hosts grow faster per unit time when exposed to kairomones. Hosts growing
150 faster due to higher kappa should actually produce more offspring (not shown) – a prediction
151 discordant with our data (Figure 2F). This results stems from the increased rate of energy
152 acquisition experience by larger-bodied hosts (despite that a smaller proportion of the energy

153 reserve is allocated towards reproduction). Thus, we added two other assumptions about the
154 physiological response of hosts to kairomones. First, we assumed that size at first reproduction
155 (*SFR*) increased with kappa, based on data from another *Daphnia* system (Stibor & Lüning
156 1994); in our simulations, $SFR = 3\,\kappa + 0.7$. Despite including this assumption about size at first
157 reproduction, the DEB model predicted that hosts that were exposed to kairomones (higher
158 kappa) could reproduce at an earlier age than those hosts not exposed, despite having higher *SFR*
159 (as seen in Figs. 2E and 5D). The explanation is straightforward: hosts with higher kappa can
160 reach the larger *SFR* at an earlier age because they grow faster. (If *SFR* increased more steeply
161 with kappa than we show in the text, hosts would reproduce at a later age, despite faster growth
162 rates; this empirical result was reported in Stibor & Lüning 1994.) Second, we assumed that the
163 overhead cost of producing an egg increases with kappa (as found in Rinke *et al.* 2008 for fish
164 kairomones); that is, the q parameter in equ. (S11) decreases with kappa. In our simulations, the
165 relationship $q = (40\,\kappa - 7)^{-1}$ produced the simulations displayed (Figure 5E). Similar results
166 would be found if the size of neonates (i.e., the energy allocated to each neonate, E_0 in equ. S11)
167 increased with kappa. (Note that total energetic cost of making an egg is E_0/q, so higher E_0,
168 lower q, or both should yield similar qualitative results). Thus, through these costs, total
169 fecundity can be similar among kairomone treatments.
170
171 **Additional experimental results: time until death**
172 In the experiment, there was a significant *Chaoborus***D. dentifera* genotype interaction
173 ($\chi^2 = 16.2$, $p < 0.0001$, Fig. S1) for day of death of infected animals. Thus, the effect of
174 kairomones on day of death depended on the host genotype. The model predicted faster death
175 from infection in the presence of kairomones. Hosts with higher allocation to growth feed at a

faster rate; this faster flux of assimilated energy, in turn, promoted faster replication of parasites within hosts. Faster replication of parasites yielded faster death of hosts because the size threshold (ρ) was reached faster. Thus, the DEB model does not quite capture the more complicated survivorship signal seen in the data.

Table S1. Parameter values and ranges of parameters used in simulations in the text. The symbols used correspond directly to those in Hall et al. (2009a), where the dynamic energy budget model is presented in detail.

Term	Units	Definition	Value or range
State Variables			
e	-	Reserve energy density (= E/W)	–
E	mg C	Reserve energy mass (= eW)	–
N	mg C	Mass of the parasite	–
R	offspring	Reproduction (offspring)	–
t	day	time	–
W	mg C	Structural mass (weight) of the host	–
X	mg C/L	Food (algae)	–
Fluxes			
A	mg C/day	Assimilation rate	–
C	mg C/day	Energy utilization (catabolism) rate	–
Parameters			
a	mg C·mm^{-2}·day^{-1}	SA-specific maximal assimilation rate, εf	4.6×10^{-3}
a_N	day^{-1}	Maximal assimilation rate, parasite, $\varepsilon_N f_N$	0.6
d_N	day^{-1}	Combined loss rate, parasite	0.08
E_0	mg C	Carbon investment per offspring	0.0021
e_M	–	Maximal energy density	1.0
f	mg C·mm^{-2}·day^{-1}	Surface area-specific maximal feeding rate	0.007-0.011

f_N	day^{-1}	Maximal feeding rate, parasite	0.75
g	–	Mass-specific cost of growth	0.8
h	mg C/L	Half-saturation constant, host	0.1
h_N	mg C	Half-saturation constant, parasite	0.005
L	mm	Size of host; relation to W: $W = \alpha L^3$	--
L_0	mm	Initial size of hosts when exposed to parasite	1.2
m	day^{-1}	W-specific maintenance rate, host	0.2
m_N	day^{-1}	Loss rate of the parasite	0.08
$N_{0,E}$	mg C	Initial spore mass in beaker to which hosts are exposed	0.033‡
q	–	Metabolic cost of production of an offspring	0.9
T	days	Interval of food replenishment	1.0
W_P	mg C	Mass at puberty	0.002
α	mg C/mm^3	Conversion for struct. mass-length regression	1.8×10^{-3}
ε_{max}	–	Maximal conversion efficiency, host	0.45§
ε_N	–	Maximal conversion efficiency, parasite	0.8
κ	–	Fraction of energy spent on growth	0.2
ρ	–	Mechanical threshold of infected host	1.68

185 * Range used in Figs. 5 and S1 to produce variation in the feeding rate.

186 ‡ Masses produced from initial spore doses of 190 spores per ml, respectively, assuming 174

187 pg/spore (Hall et al. 2009a). Mass of ingested parasite (N_0 of Hall et al. 2009a) is then calculated

188 for a 1.2 mm size animal as a function of clearance rate (feeding rate divided by algal density).

189 [§] A lower value used than used previously. Hosts growing in the artificial water (ADaM) seem
190 to growth more slowly than when inhabiting lake water. To capture that effect, we lowered ε_{max},
191 which slowed growth, reproduction, age at first reproduction, etc.

Figure S1. Time until death for infected animals, as seen in (A) the life table experiment, and (B) the dynamic energy budget model. In the data panel, *P*-values of ANOVA results are shown in the insets, with "*C*" indicating effects of *Chaoborus* kairomone, "G" indicating effects of *Daphnia* Genotype, and "*C*×G" indicating their interaction. In the model panel, the arrow points in the direction of increasing maximal size-specific feeding rate, from 7.0-11.0 at 0.5 increments (mg $C \cdot L^{-1} \cdot mm^{-2} \cdot day^{-1} \times 10^{-3}$).