

1 **Exploring the Relationship between Abundance and Temperature with a Chemostat Model**

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13 Running headline: Abundance response to temperature

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15 Keywords: Abundance, equilibrium population size, chemostat, population growth rate,
16 temperature response curve.

17 **Abstract**

18 Although there is a well developed theory on the relationship between the intrinsic growth rate r
19 and temperature T , it is not yet clear how r relates to abundance, and how abundance relates to T .
20 Many species often have stable enough population dynamics that one can talk about a stochastic
21 equilibrium population size N^* . There is sometimes an assumption that N^* and r are positively
22 correlated, but there is lack of evidence for this. To try to understand the relationship between r ,
23 N^* , and T we used a simple chemostat model. The model shows that N^* not only depends on r ,
24 but also on the mortality rate, the half-saturation constant of the nutrient limiting r , and the
25 conversion coefficient of the limiting nutrient. Our analysis shows that N^* positively correlates
26 to r only with high mortality rate and half-saturation constant values. The response curve of N^*
27 vs. T can be flat, Gaussian, convex, and even temperature independent depending on the values
28 of the variables in the model and their relationship to T . Moreover, whenever the populations
29 have not reached equilibrium and might be in the process of doing so, it could be wrongly
30 concluded that N^* and r are positively correlated. Because of their low half-saturation constants,
31 unless conditions are oligotrophic, microorganisms would tend to have flat abundance response
32 curves to temperature even with high mortality rates. In contrast, unless conditions are eutrophic,
33 it should be easier to get a Gaussian temperature response curve for multicellular organisms
34 because of their high half-saturation constant. This work sheds light to why it is so difficult for
35 any general principles to emerge on the abundance response to temperature. We conclude that
36 directly relating N^* to r is an oversimplification that should be avoided.

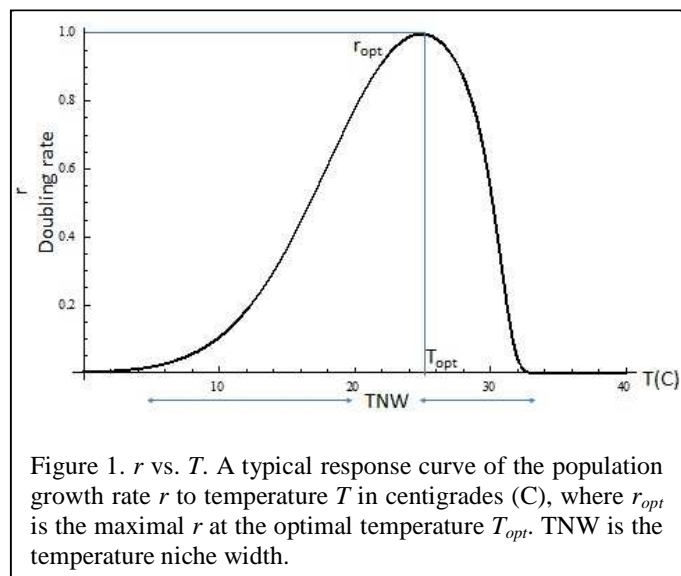
37 Introduction

38 The metabolic rate of any organism basically depends on the concentration of resources
39 in the environment, on the flux of these resources into the organism, on its body size, and on
40 temperature, which determines the rate of biochemical processes (Gillooly *et al.* 2001). For
41 enzyme-mediated reactions, reaction rates increase from low to high temperature, reaching a
42 maximum, and then rapidly decreasing often due to protein denaturation (Kingsolver 2009). As
43 in the reaction rates, the response curve of the population intrinsic rate of increase r to
44 temperature T is generally asymmetric, with a sharper drop off to high temperatures from the
45 optimum (Figure 1). This relationship has been studied in detail and summarized in recent
46 review papers (Huey & Berrigan 2001; Frazier *et al.* 2006; Kingsolver & Huey 2008; Martin &
47 Huey 2008; Kingsolver 2009).

48 Although there is a well developed theory and a robust understanding of the relationship
49 of r vs. T , it is not yet clear how r relates to abundance (or more precisely density in units of
50 individuals per area or volume; McGill 2006), and how abundance relates to T . There is
51 sometimes an assumption that r and abundance are positively correlated, but there is lack of hard
52 evidence for this.

53 Some species are not even close of having equilibrium population dynamics. In those
54 cases, the idea of abundance may not be well defined and a theory of r vs. T might be the most
55 useful. Nonetheless, many species do have stable enough population dynamics to meaningfully
56 talk about a stochastic equilibrium
57 population size N^* . It is not yet clear how
58 the physiological predictions about r
59 translate into predictions about N^* .

60 Given the obvious importance of
61 developing a theory about the effect of
62 temperature on the abundance of species,
63 several routes have been used to attack
64 the question. These include experimental
65 temperature manipulations of
66 communities in natural ecosystems (e.g.,



67 Chapin & Shaver 1985; Suttle *et al.* 2007), laboratory microcosms (e.g., Davis *et al.* 1998;
68 Petchey *et al.* 1999; Jiang & Morin 2004), observing natural climate change over a few decades
69 in natural communities (e.g., Kimball *et al.* 2009), and theoretical population dynamics (e.g.,
70 Ives & Gilchrist 1993; Ives 1995; Vasseur & McCann 2005) just to name a few. Nevertheless,
71 from these studies no general principles on the effect of temperature on populations have yet
72 emerged.

73 For example, Kimball *et al.* (2009) found that after two decades of natural warming,
74 surprisingly, the cold-adapted annuals increased while the warm-adapted annuals decreased in
75 abundance. In a microcosm experiment with ciliates, Jiang and Morin (2004) found that
76 temperature had no effect on N^* for one species but decreased N^* for another in monoculture,
77 the negatively affected species actually competitively excluding the unaffected one at
78 intermediate temperatures. Vasseur and McCann (2005) predicted that population cycles would
79 be more common and resource biomass would decrease. In another theoretical development
80 (Ives & Gilchrist 1993; Ives 1995) it was predicted that density dependence could buffer/dampen
81 (in intraspecific competition and predator-prey dynamics) or magnify (in interspecific
82 competition) the effect of T on N^* .

83 In short, the hypothesis about the performance curve of r has received enormous
84 attention, but there has been limited development of theory on the effect of T on N^* . Gause
85 (1932, 1934) suggested that N^* had a response to T similar to that of r , a Gaussian bell response
86 curve, but this has received little follow up work. He presented as evidence two field studies
87 done along environmental gradients (in grasshoppers and starfish) and two examples of
88 laboratory experiments where only temperature varied (in the yeast *Sacchromyces* and in *Monia*,
89 a Cladoceran). Later work on flour beetles (*Tribolium*; Birch 1953; Park 1954) also showed that
90 the equilibrium population size varied in a modal fashion with temperature, but the number and
91 range of temperatures was not enough to determine the shape in detail.

92 The simplest approach to explore the relationship between the population growth rate r ,
93 abundance N^* and temperature T is to use the chemostat model. A chemostat is a system used in
94 microbiology in which fresh medium is continuously added, while the culture medium is
95 continuously removed at the same rate to keep the volume constant in a dynamical equilibrium.
96 Here, we analyze the chemostat dynamics to try to understand the relationship between r , N^* ,

97 and T . We also explore how the abundance response to temperature might differ between a
98 unicellular organism and a more complex multicellular one with germ-soma differentiation.

99 **The Model**

100 In a chemostat the flow rate ω depends on the total volume V of the container and the
101 flow F in and out of it, $\omega = F/V$. In the absence of organisms, the substrate concentration S in the
102 container follows,

$$103 \quad \frac{dS}{dt} = \omega S_0 - \omega S \quad (1),$$

104 where S_0 is the substrate concentration in the medium flowing in and $S(t)$ is the substrate
105 concentration in the container (Hoppensteadt 2011). If we use the Monod model to add
106 organisms into the chemostat system (e.g., Droop 1982), then,

$$107 \quad \frac{dS}{dt} = \omega S_0 - \omega S - \frac{r_{\max} S}{K_S + S} \frac{N}{Y} \quad (2),$$

$$108 \quad \frac{dN}{dt} = \left(\frac{r_{\max} S}{K_S + S} - \omega \right) N \quad (3).$$

109 Equation 2 describes the change in concentration of the limiting substrate S due to the inflow of
110 fresh medium (ωS_0 ; ω = flow rate), minus the outflow (ωS), minus the substrate consumed by the
111 organisms N ($[rS/(K_S+S)] N/Y$). The substrate consumption depends on the population growth rate
112 r_{\max} when there are no substrate limitations, on the half-saturation constant K_S , which determines
113 how sensitive the organism's growth rate is to substrate limitation, and the substrate conversion
114 coefficient Y . Equation 3 describes the change in population, which depends on r_{\max} , K_S , and the
115 rate ω at which the population is discarded (i.e., the mortality rate).

116 There are two possible equilibrium states in this system ($dN/dt = 0$); either when $N = 0$
117 (the population goes extinct) or when $[rS/(K_S+S)] - \omega = 0$. If $r < \omega$, then $N \rightarrow 0$, but if $r > \omega$, then,

$$118 \quad S^* = \frac{K_S \omega}{r_{\max} - \omega} \quad (4),$$

$$119 \quad N^* = Y(S_0 - S^*) = Y\left(S_0 - \frac{K_S \omega}{r_{\max} - \omega}\right) \quad (5).$$

120 Thus, the equilibrium population N^* (i.e., abundance) in a chemostat depends on the total
121 amount of limiting substrate in the system S_0 , on the flow rate of the system ω (i.e., the coupled

122 mortality and nutrient recycling rates), the
 123 population growth rate r_{max} , the half-
 124 saturation constant K_S , and the conversion
 125 factor Y . As explained above, it is well
 126 known how r_{max} changes with T , but it is not
 127 yet clear how K_S , ω , and Y change as a
 128 function of T , nor how these variables change
 129 as a function of the size and complexity of the
 130 organism.

131 Does abundance N^* positively
 132 correlates to the population growth rate r_{max} ?
 133 In a chemostat it really depends on how much
 134 the limiting nutrient negatively affects the
 135 population growth rate of the organism in
 136 question (i.e., the half-saturation constant K_S),
 137 and on its mortality rate ω (Figure 2). If K_S is
 138 low compared to the amount of limiting
 139 substrate in the system (S_o ; low K_S/S_o ratio)
 140 then the relationship between N^* and r_{max} is
 141 flat for most of the r_{max} values regardless of

142 the mortality rate, with a sharp drop-off of N^* at the lower end of r_{max} . As the K_S/S_o ratio
 143 increases, abundance decreases, but the positive correlation between N^* and r_{max} increases.
 144 Increasing the mortality rate ω (increasing the ω/r_{max} ratio), also lowers abundance and limits the
 145 range of r_{max} values in which N^* is positive, but further accentuates the positive correlation
 146 between N^* and r_{max} . In short, it is only with a high negative effect of the limiting nutrient on the
 147 population growth rate (i.e., a high K_S/S_o ratio) and/or a high mortality rate (i.e., a high ω/r_{max}
 148 ratio) that we find a significant positive relationship between abundance and the population
 149 growth rate.

150 Now let's assume r_{max} has a temperature response curve as described above (Figure 1). To
 151 analyze N^* vs. T , we can use a Gaussian times a Gompertz function to accommodate the
 152 nonlinear nature of the relationship between r_{max} and T as described by Frazier et al. (2006),

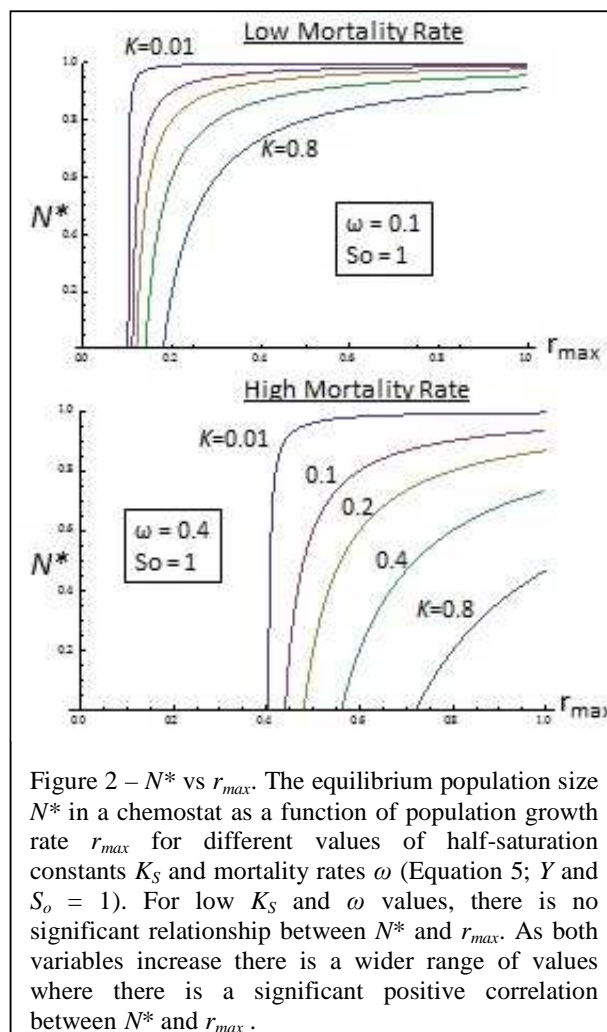


Figure 2 – N^* vs r_{max} . The equilibrium population size N^* in a chemostat as a function of population growth rate r_{max} for different values of half-saturation constants K_S and mortality rates ω (Equation 5; Y and $S_o = 1$). For low K_S and ω values, there is no significant relationship between N^* and r_{max} . As both variables increase there is a wider range of values where there is a significant positive correlation between N^* and r_{max} .

153 $r_{\max}(T) = r_{\text{opt}} e^{-e[\rho(T-T_{\text{opt}})-6]-\sigma(T-T_{\text{opt}})^2}$ (6),

154 where r_{opt} is the maximal growth rate at optimal temperature T_{opt} , ρ represents the increasing part
 155 of the population growth rate curve, and σ represents the declining part of the curve. Eq. 5

156 becomes $N^* = Y(S_0 - \frac{K_S \omega}{r_{\max}(T) - \omega})$.

157 We assume, for now, no relationship between K_S , ω , Y and T . As in N^* vs. r_{\max} , we observe that
 158 the response curve of N^* to T is highly dependent on K_S and ω levels (Figure 3). As shown in
 159 Figure 2, only with high K_S/S_0 and ω/r_{opt} ratios we observe a Gaussian temperature response
 160 curve for N^* . If the mortality rate is low compared to the optimal population growth rate (low
 161 ω/r_{opt} ratio), the equilibrium population (i.e., abundance) response curves to temperature are flat
 162 with a steep decline at the edges of the temperature niche width (Figure 3B). Increasing the
 163 negative effect of the limiting nutrient on the population growth rate (high K_S/S_0 ratio) slightly
 164 decreases the temperature niche width and the population size, but does not change significantly

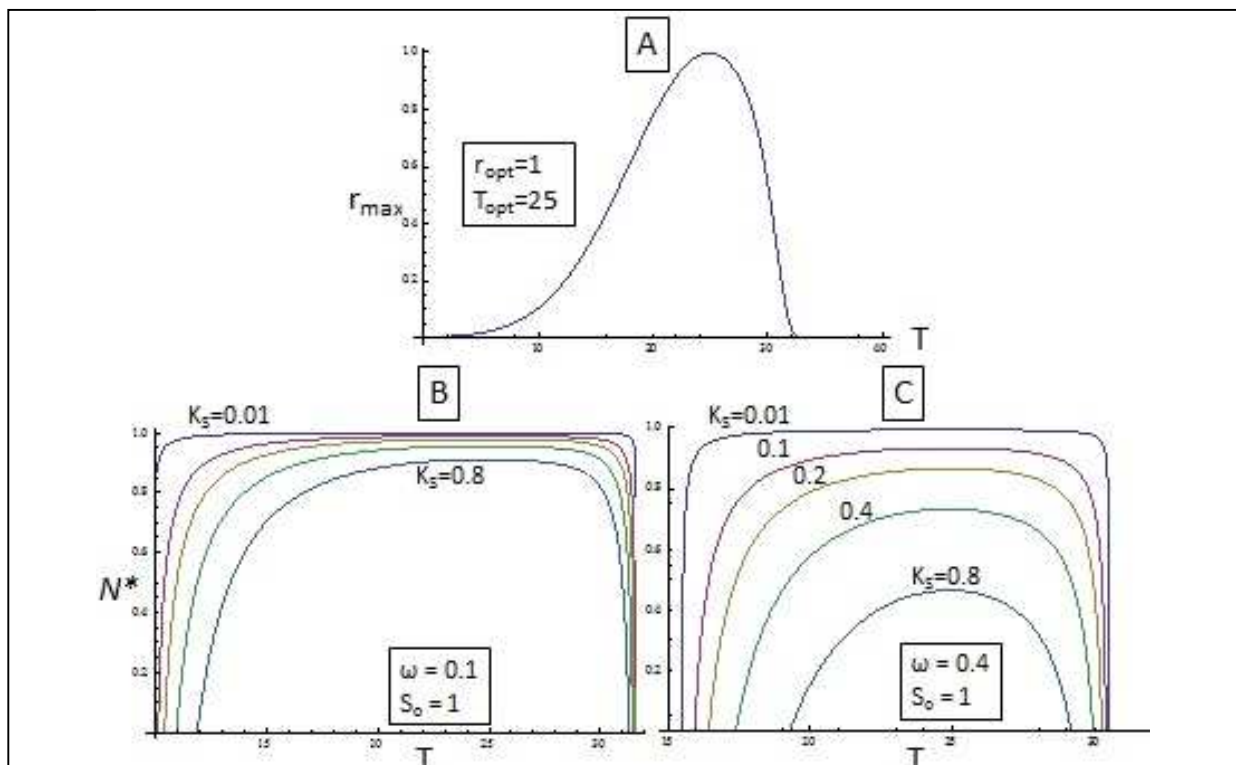


Figure 3. A - r_{\max} vs. T (Eq. 6; $\rho=1$, $\sigma=0.01$). B - N^* vs. T (Eq. 5 with Eq. 6 inserted) with low mortality ω for different values of the half-saturation constant K_S . C - N^* vs. T (Eq. 5 with Eq. 6 inserted) with high ω for different values of K_S . The figure shows that the equilibrium population size has a Gaussian response curve only with high K_S and ω values.

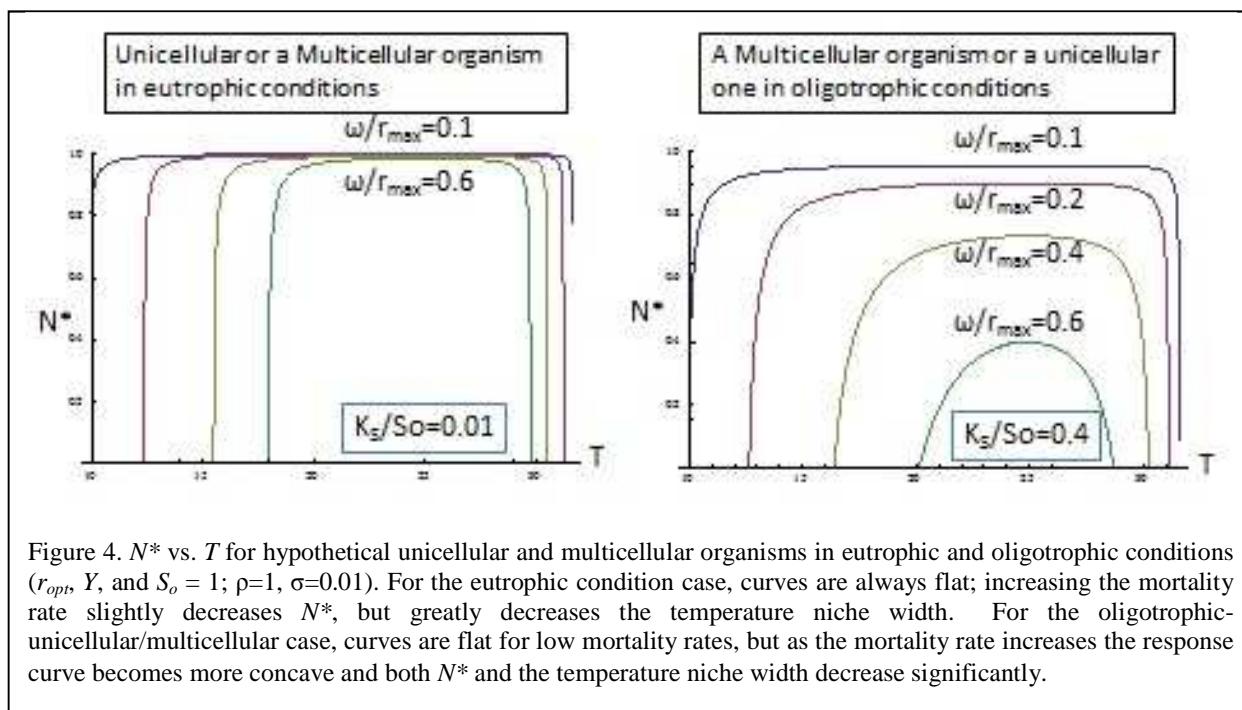
165 the shape of the response curve. As the mortality rate increases (high ω/r_{opt} ratio), the equilibrium
166 population response curves to temperature become more Gaussian and concave (Figure 3C). In
167 this case, the equilibrium population size and the temperature niche width decrease significantly
168 with the mortality rate.

169 What do we know about the relationship between the half-saturation constant K_S and T ?
170 There has been no consistent pattern observed for the variation of K_S vs. T in diverse
171 microorganisms such as algae and bacteria, and for limiting nutrients such as silicate, nitrate,
172 ammonium, and phosphorous (e.g., Mechling and Kilham 1982; Nedwell 1999; Tilman et al.
173 1981). The only consistent pattern found in microorganisms is that K_S values are in general
174 relatively low; meaning that microorganisms can still grow at maximal rates even at very low
175 concentrations of the limiting nutrient. On the other hand, there is evidence that multicellular
176 organisms with germ-soma differentiation have much higher K_S values than unicellular ones,
177 presumably due to the additional nutrients needed to maintain the somatic tissue (e.g., *Volvox*
178 *sp.*; Senft et al. 1981). In short, unicellular organisms in general have high population growth
179 rates and low half-saturation constants, but larger multicellular organisms with cellular
180 differentiation have lower population growth rates - due to size/allometric constraints - and
181 higher saturation constants.

182 In addition, there is evidence that in multicellular *Volvox sp.* K_S also has a Gaussian
183 temperature response curve similar to that of r_{max} vs. T (Senft et al. 1981, own observations in
184 *Volvox carteri*). This is presumably because the metabolic rate of somatic cells would follow the
185 same temperature response curve of reproductive cells, increasing the metabolic need of soma at
186 optimal temperatures and decreasing it at suboptimal ones. To analyze this, for the sake of
187 simplicity, we assumed for the hypothetical multicellular organism that K_S has the same response
188 curve as $r_{max}(T)$ (Eq. 6), $K_S(T) = K_{max} r_{max}(T)$, where K_{max} is the maximum half-saturation
189 constant value at the optimal temperature (for $K_S(T)$ we use the same parameters as in $r_{max}(T)$ in
190 Eq. 6). Thus, Eq. 5 becomes $N^* = Y(S_0 - \frac{K_{max} r_{max}(T)\omega}{r_{max}(T) - \omega})$.

191 Figure 4 shows N^* vs. T for a hypothetical unicellular organism with a low K_S/S_0 ratio and a
192 constant K_S , and for a multicellular one with $K_S(T) = K_{max} r_{max}(T)$ and a high K_{max}/S_0 ratio.

193 From a nutrient availability point of view, if conditions are oligotrophic (low S_0), then a
194 unicellular population could also have a high K_S/S_0 ratio, thus, the N^* response to T could be



195 similar to that of a population of multicellular organisms. On the other hand, if the conditions are
 196 eutrophic (high S_0), then the K_{max}/S_0 ratio for the multicellular population would also be low and
 197 its response similar to that of the unicellular population. In short, unicellular or multicellular
 198 populations in eutrophic conditions would have a similar response curve to T , and unicellular
 199 populations in oligotrophic conditions would have a response curve similar to that of
 200 multicellular populations.

201 In the hypothetical unicellular/eutrophic case the equilibrium population (i.e., abundance)
 202 response curves to temperature are flat with a steep decline at the suboptimal high and low
 203 temperatures (Figure 5). Increasing the mortality rate decreases the temperature niche width, but
 204 does not change significantly the shape of the response curve nor the population size in optimal
 205 temperatures. On the other hand, in the multicellular/oligotrophic case the equilibrium
 206 population response curves to temperature become more Gaussian and concave as the mortality
 207 rate increases. In this case, both the equilibrium population size and the temperature niche width
 208 decrease significantly with the mortality rate.

209 What about the relationship between the mortality rate ω and T ? In a chemostat ω is
 210 temperature independent because it is artificially adjusted by changing the flow/removal rate in
 211 the system. But what if we envision a community of ectotherms such as a plankton community,
 212 where we are tracking the abundance of the phytoplankton? The predation rate can be the main

213 factor affecting the mortality rate, and the
 214 zooplankton grazers might have metabolic
 215 and feeding rates that respond to temperature
 216 in the same way the phytoplankton population
 217 growth rates do. For example, it was shown
 218 that copepods that graze on phytoplankton
 219 have a feeding rate that follows a dome-
 220 shaped pattern as a function of temperature
 221 (e.g., Almeda et al. 2010; Garrido et al. 2013;
 222 Moller et al. 2012).

223 If again for the sake of simplicity we

224 assume that ω has the same response curve as $r_{max}(T)$ (Eq. 6), then $\omega(T) = \omega_{max} r_{max}(T)$, where
 225 ω_{max} is the maximum mortality rate (i.e., predation) at the optimal temperature. If we use the
 226 same parameter values for $r(T)$ and $\omega(T)$, then the temperature term cancels out and Eq. 5

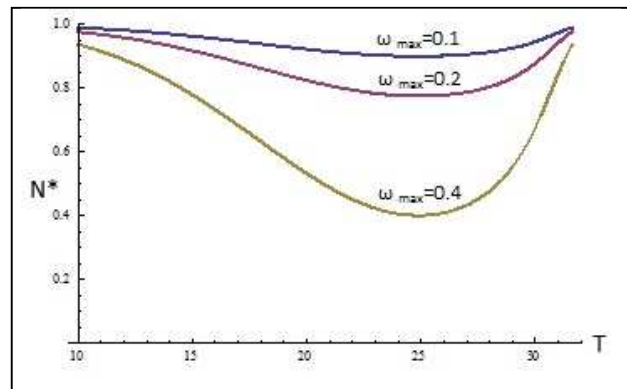
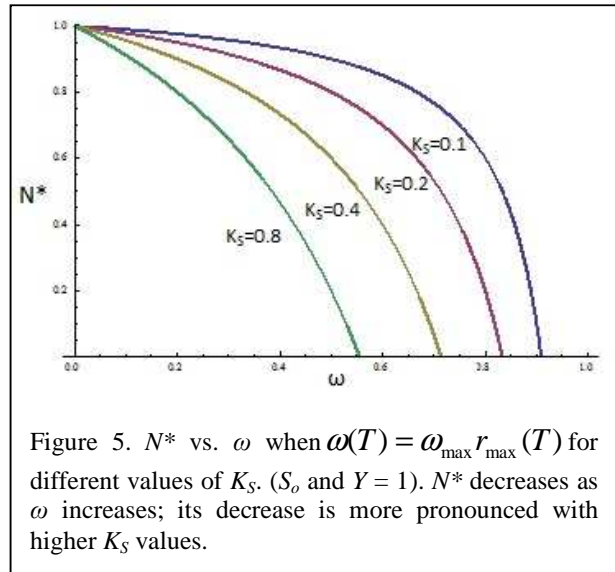
227 becomes temperature independent; $N^* = Y \left[S_0 - \frac{K_S \omega_{max}}{(1 - \omega_{max})} \right]$.

228 In this case, N^* only depends on K_S and ω_{max} . Figure 5 shows how N^* decreases as ω increases;
 229 its decrease is more pronounced with higher K_S values.

230 If we now assume that both $K_S(T)$ and
 231 $\omega(T)$ have the same response curve as $r_{max}(T)$,
 232 then Eq. 5 becomes

233
$$N^* = Y \left[S_0 - \frac{K_{max} \omega_{max} r_{max}(T)}{(1 - \omega_{max})} \right].$$

234 Surprisingly, the temperature response curve
 235 flips, becoming convex (Figure 6). The
 236 populations of the hypothetical organisms are
 237 better off at suboptimal temperatures because
 238 both the half-saturation constant and the
 239 mortality rate are at their maximum values at
 240 optimal temperatures, negatively affecting



241 abundance in the best conditions for population
 242 growth.

243 What about the relationship between the
 244 conversion coefficient Y and T ? The
 245 temperature–size rule proposes that ectotherms
 246 that develop at higher temperatures are
 247 relatively smaller as adults than when they
 248 develop at lower temperatures. There is plenty
 249 of evidence for this rule in multiple taxa
 250 (Kingsolver and Huey 2008). Therefore, if we
 251 assume abundance to be density in units of

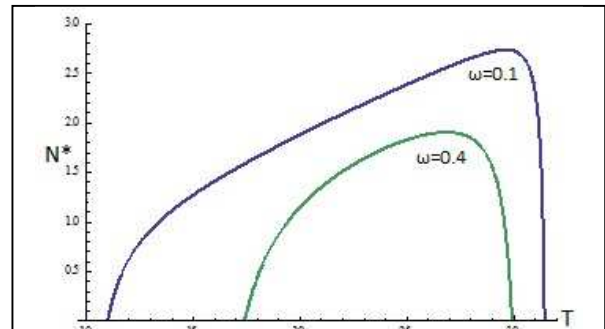


Figure 7. Hotter is smaller. N^* vs. T for different values of ω ($\rho=1$, $\sigma=0.01$; r_{opt} and $S_o = 1$, $K_S=0.4$; $Y = 0.1T$) for the case where $r_{max}(T)$. N^* response curves get skewed to higher abundance peaks at higher temperatures because smaller organisms need less nutrients.

252 individuals per area or volume, the conversion coefficient Y may change as temperature
 253 increases, since smaller individuals would need fewer nutrients to develop. Some of the
 254 relationships reported between size and temperature have an approximately negative linear slope,
 255 so for simplicity, we can just assume a positive linear relationship between Y and T ($Y(T) = aT +$
 256 b) since less need for nutrients means more individuals per substrate absorbed from the medium.
 257 Figure 7 shows how, N^* response curves get skewed to higher abundance peaks at higher
 258 temperatures as Y increases with T . What would be an almost flat response curve if the
 259 relationship between Y and T is not taken into account (for $\omega=0.1$, Figure 3) becomes a concave
 260 curve with a peak at a higher temperature if the size-temperature rule is taken into account (for
 261 $\omega=0.1$, Figure 7).

262 So far we have only analyzed populations that have reached equilibrium population size
 263 at different temperatures (i.e., N^* , Eq. 5). Although we have showed, for example, that N^* does
 264 not change significantly with T at low K_S/S_o and ω/r_{max} ratios, populations growing at optimal
 265 temperatures with high r_{max} values would reach equilibrium population size much faster than
 266 those growing at suboptimal temperatures. If we solve Eq. 3 for time t then,

$$267 \quad t = \frac{\ln[N]}{\left(\frac{r_{max} S}{K_S + S}\right) - \omega} \quad (7).$$

268 To illustrate this point and for the sake of simplicity we will just assume that $K_S \sim 0$, thus,
 269 $t \approx \ln[N]/(r_{max} - \omega)$. Figure 8A shows how the time to reach a certain population size increases

270 exponentially as r_{max} decreases (Figure 8A).
 271 If we plot populations at different time steps
 272 before reaching an arbitrary population size
 273 ($N = e^{(r_{max}(T)-\omega)t}$), the abundance at those
 274 time steps would seem to have a Gaussian
 275 bell response curve as a function of T
 276 instead of the flat response we see when the
 277 population reaches equilibrium (Figure 8B).
 278 In short, if the population does not reach
 279 equilibrium, its abundance temperature
 280 response curve could resemble the
 281 population growth rate curve.

282 Discussion

283 In this paper we have explored the
 284 relationship between abundance and
 285 temperature using a simple chemostat
 286 model. We have shown that the equilibrium
 287 population size not only depends on the
 288 population growth rate, but also on its mortality rate - and coupled nutrient recycling rate - , and
 289 on the relationship the organisms in question have with the limiting nutrient in the system (Eq.
 290 5). Abundance positively correlates to the population growth rate in a chemostat only with a high
 291 negative effect of the limiting nutrient on the population growth rate (i.e., a high K_S/S_o ratio)
 292 and/or a high mortality rate compared to the population growth rate (i.e., a high ω/r_{max} ratio;
 293 Figure 2).

294 If we assume a typical population growth rate response curve to temperature and leave all
 295 the other variables constant (Figure 1, Eq. 6), again only with a high negative effect of the
 296 limiting nutrient on the population growth rate and/or a high mortality rate, the equilibrium
 297 population response curves to temperature become Gaussian as a function of temperature (Figure
 298 3). With low mortality or half-saturation constant values, the relationship between abundance
 299 and temperature remains flat for almost all of the temperature niche width.

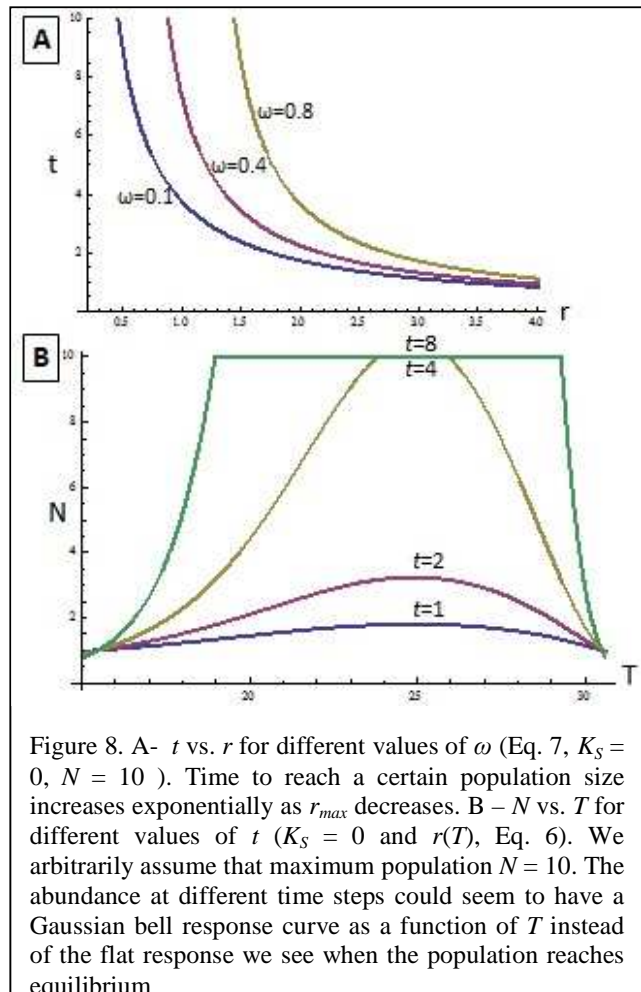


Figure 8. A- t vs. r for different values of ω (Eq. 7, $K_S = 0$, $N = 10$). Time to reach a certain population size increases exponentially as r_{max} decreases. B - N vs. T for different values of t ($K_S = 0$ and $r(T)$, Eq. 6). We arbitrarily assume that maximum population $N = 10$. The abundance at different time steps could seem to have a Gaussian bell response curve as a function of T instead of the flat response we see when the population reaches equilibrium

300 In general, unicellular organisms have high population growth rates and low half-
301 saturation constants, but larger multicellular organisms with cellular differentiation have lower
302 population growth rates and higher saturation constants. Moreover, evidence shows that in
303 multicellular organisms the half-saturation constant for a limiting nutrient might have a Gaussian
304 response curve to temperature similar to the population growth rate one (e.g., *Volvox* sp.; Senft et
305 al. 1981; own observations in *Volvox carteri*).

306 Our analysis shows that a unicellular or a multicellular population in eutrophic conditions
307 could have a similar response curve to temperature (low K_s/S_o ratio) because the higher half-
308 saturation constant in a multicellular organism due to the metabolic need of the soma would be
309 minimized by the high concentration of nutrients in the system (Figure 4). In this case, even with
310 high mortality rates there is no relationship between abundance and temperature in almost all of
311 the temperature niche width. In contrast, a unicellular population in oligotrophic conditions
312 would have a response curve similar to a multicellular population (high K_s/S_o ratio) because the
313 low concentration of nutrients in the system would augment the limiting nutrient negative effect
314 on the unicellular population. In this case, there is an increased Gaussian response curve of
315 abundance to temperature with increasing mortality rates.

316 If the mortality rate has a similar temperature response curve as the population growth
317 rate (e.g., the predation rate temperature response curve is similar to the prey population growth
318 rate response curve), abundance might even become temperature independent, and only depend
319 on the maximum mortality rate and half-saturation constants (Figure 5). Furthermore, if both the
320 mortality rate and the half-saturation constant have the same temperature response curve as the
321 population growth rate (i.e., as in a multicellular population), then the curves flip and become
322 convex, organisms having higher abundance at suboptimal temperatures since the highest
323 mortality and half-saturation constant levels coincide with the optimal temperature for growth
324 (Figure 6). In addition to all these scenarios, the ubiquitous temperature-size rule would increase
325 abundance at higher temperatures since organisms would need fewer nutrients per individual as
326 they develop to a smaller adult size (Figure 7), skewing abundance temperature response curves
327 to higher temperatures.

328 To summarize, with a simple chemostat model we have shown why abundance might
329 respond to temperature in many ways. The response curve of abundance to temperature can be
330 flat, concave, convex and even temperature independent depending on the population growth and

331 mortality rates, the half-saturation constant, the amount of limiting nutrient in the system, and the
332 conversion coefficient of the nutrient. Even if the system is closed and there is no nutrient
333 concentration change in the flowing medium, the availability of the limiting nutrient to the
334 organisms might change since diffusion coefficients are also temperature dependent, therefore
335 possibly affecting abundance. In conclusion, directly relating abundance to the population
336 growth rate is an oversimplification that should be avoided.

337 Finally, it is important to point out that if the population of interest has not reached
338 equilibrium and might be in the process of doing so, an observer can reach the wrong conclusion
339 that abundance and population growth rates are positively correlated and have similar response
340 curves for temperature (Figure 8). Because of the limited timeframe of studies, researchers can
341 reach wrong conclusions on how abundance is affected by temperature. If the population is
342 allowed to reach equilibrium, depending on the conditions of the system where the population is
343 growing, there might be no relationship between abundance and growth rate, and between
344 abundance and temperature.

345 Of course, in natural systems several trophic levels and species interact with one another;
346 temperature, light intensity, and nutrient availability do not remain constant, and the recycling of
347 nutrients and the mortality rate are not directly coupled as they are in a chemostat. We have not
348 taken into account very important population dynamics aspects such as multispecies interactions
349 (e.g., competition for resources; Tilman et al. 1981), organisms adaptation to temperature change
350 (e.g., Thomas et al. 2012; in *Chlorella vulgaris*, Padfield et al. 2015), changes in the nutrient
351 recycling rate due to, for example, the temperature dependence of the detritivores metabolic rate,
352 changes in the total amount of nutrients in the system due to net inflows/outflows from other
353 sources or sinks, just to name a few.

354 In addition, complexities such as behavioral thermoregulation and water vs. heat balance
355 are not a factor in our model. Terrestrial organisms can behaviorally thermoregulate their body
356 temperatures to deviate significantly from the air. Moreover, water and heat balance are
357 confounded for terrestrial organisms – both plants and animals cool themselves by evaporation,
358 resulting in a strong water-temperature interaction.

359 Nonetheless, if a population in a chemostat can be used as an oversimplified analogy for
360 a population that is at equilibrium in a stable ecosystem, the model analysis shows why it is so
361 difficult for general principles to emerge on the effect of temperature on populations. When

362 studying populations, it is difficult to know what nutrient is limiting population growth, what the
363 main mortality factor is, what the conversion coefficient is, etc., and how all of these factors are
364 changing with temperature. Are these populations observed at some kind of stochastic
365 equilibrium? Are they on their way to reaching a new one? Or regular perturbation will never
366 allow them to reach one? How fast do these populations adapt to a new temperature regime or a
367 new limiting nutrient? From our very limited analysis, we conclude that it is difficult to make
368 well founded predictions about the outcome of abundance due to temperature change unless all
369 of these factors are well known and taken into account.

370 In the future we plan to set up population experiments at different temperatures, nutrient
371 concentrations, and mortality rates, in order to measure all the variables in the chemostat model
372 (r , ω , N^* , K , Y) and check whether the predicted abundance outcomes of the model hold. We
373 believe that laboratory-based microbial model systems to address ecological questions have
374 been, and are still underused (Jessup *et al.* 2004). Although they are not intended to reproduce
375 natural conditions, their simplicity allows addressing questions that would be inaccessible
376 through other means.

377 We plan to use the volvocine green algae as a model system (Figure 9). The Volvocales
378 are facultatively sexual, uni- and multicellular, flagellated, photosynthetic, haploid organisms
379 with varying degrees of complexity stemming from differences in colony size, colony structure,
380 and germ-soma specialization. They range from the unicellular *Chlamydomonas* (Fig. 9A), to
381 multicellular individuals comprising 1,000-50,000 cells with complete germ-soma separation,
382 e.g. *Volvox* (Fig. 9E,F; Kirk 1998; Herron and
383 Michod 2008; Nozaki *et al.* 2006; Solari *et al.*
384 2006). Due to their range of sizes, they enable the
385 study of scaling laws: the number of cells ranges
386 from 10^0 (*Chlamydomonas*) to $\sim 10^4$ (*Volvox*
387 *barberi*). All kinds of communities can be
388 assembled with organisms of different sizes and
389 complexity, but with similar cell biology.

390 The first set of experiments will consist of
391 populations grown in axenic (sterile) conditions,
392 thus having only one trophic level (producers) in

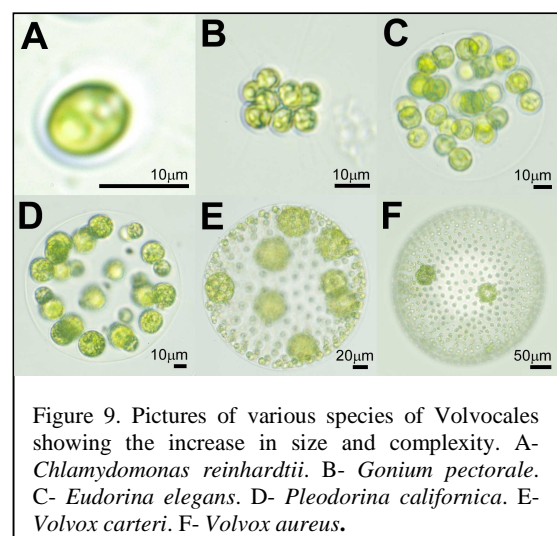


Figure 9. Pictures of various species of Volvocales showing the increase in size and complexity. A- *Chlamydomonas reinhardtii*. B- *Gonium pectorale*. C- *Eudorina elegans*. D- *Pleodorina californica*. E- *Volvox carteri*. F- *Volvox aureus*.

393 monoculture and competing with each other (polyculture). In the second part of the project we
394 will use non-axenic cultures adding the detritivorous trophic level. We expect the population
395 dynamics of these experiments to be totally different from those of the experiments made with
396 axenic cultures (we continuously observe this in our axenic and nonaxenic stock cultures). We
397 also expect totally different dynamics between unicellular, differentiated, and germ-soma
398 differentiated *Volvox* species, since the large multicellular species will shed more organic
399 material that the bacteria can consume (ECM with somatic cells) than the non-differentiated or
400 unicellular ones.

401 We will then introduce a third trophic level (e.g., the phagotroph Euglenoid *Peranema*
402 *trichophorum*, which we already keep and use, Solari et al. 2015. The filter-feeding rotifer
403 *Brachionus calyciflorus* and unicellular protist *Paramecium tetraurelia* are possible alternatives
404 for predators). This will be of interest for two reasons. First, it will explore a second type of
405 species interaction (predator/prey or more precisely herbivory). Second, these are size-dependent
406 predators that will greatly tip the competitive balance among the species.

407 **Acknowledgements**

408 This work was supported in part by CONICET grant PIP 283, Ministry of Science grant PICT
409 2011-1435, and the Universidad de Buenos Aires.

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