

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.

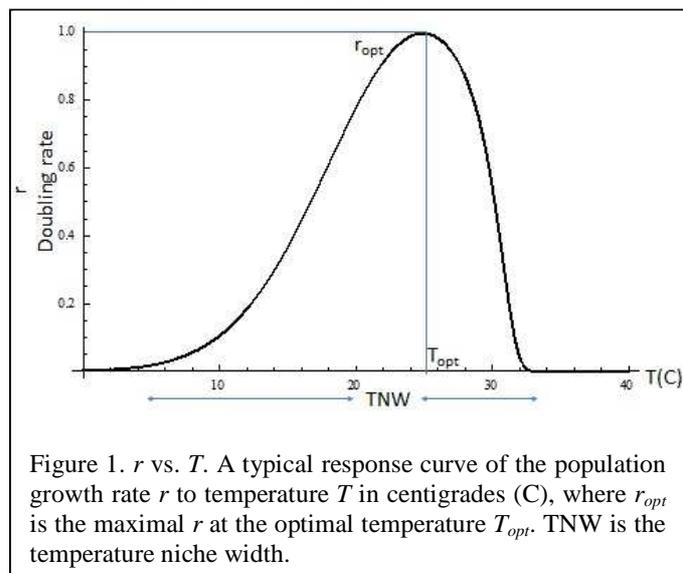
## 37 Introduction

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  $\omega$  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 ( $\omega S_0$ ;  $\omega$  = flow rate), minus the outflow ( $\omega S$ ), 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  $\omega$  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  $\omega$  (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  $K_S$ ,  $\omega$ , 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  $\omega$  (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  $\omega$  (increasing the  $\omega/r_{max}$  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  $\omega/r_{max}$   
 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),

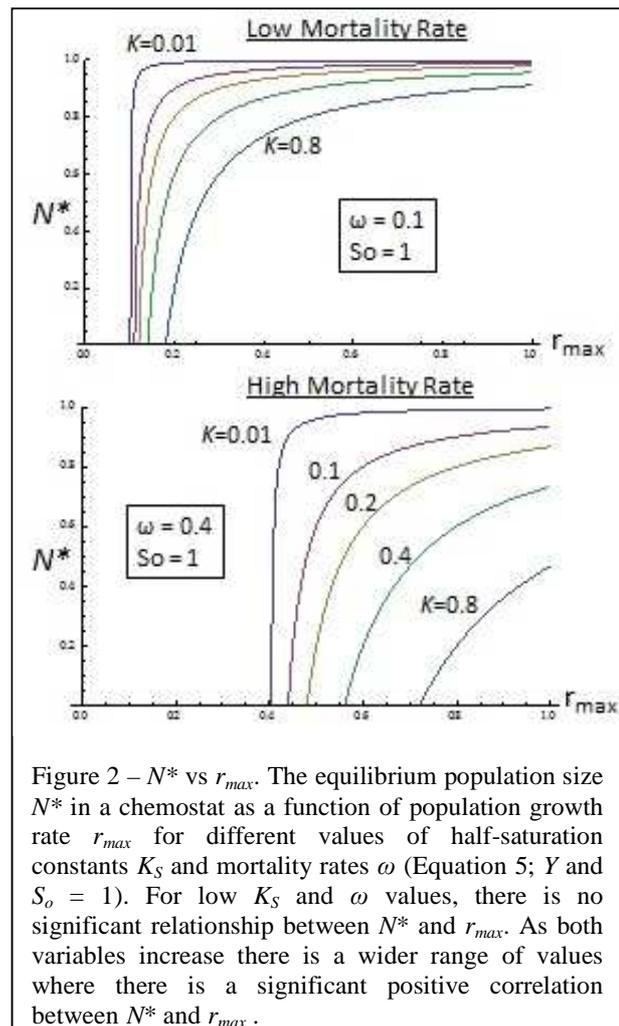


Figure 2 –  $N^*$  vs  $r_{max}$ . The equilibrium population size  $N^*$  in a chemostat as a function of population growth rate  $r_{max}$  for different values of half-saturation constants  $K_S$  and mortality rates  $\omega$  (Equation 5;  $Y$  and  $S_o = 1$ ). For low  $K_S$  and  $\omega$  values, 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  $r_{\text{opt}}$  is the maximal growth rate at optimal temperature  $T_{\text{opt}}$ ,  $\rho$  represents the increasing part  
 155 of the population growth rate curve, and  $\sigma$  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  $K_S$ ,  $\omega$ ,  $Y$  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  $\omega$  levels (Figure 3). As shown in  
 159 Figure 2, only with high  $K_S/S_0$  and  $\omega/r_{\text{opt}}$  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  $\omega/r_{\text{opt}}$  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

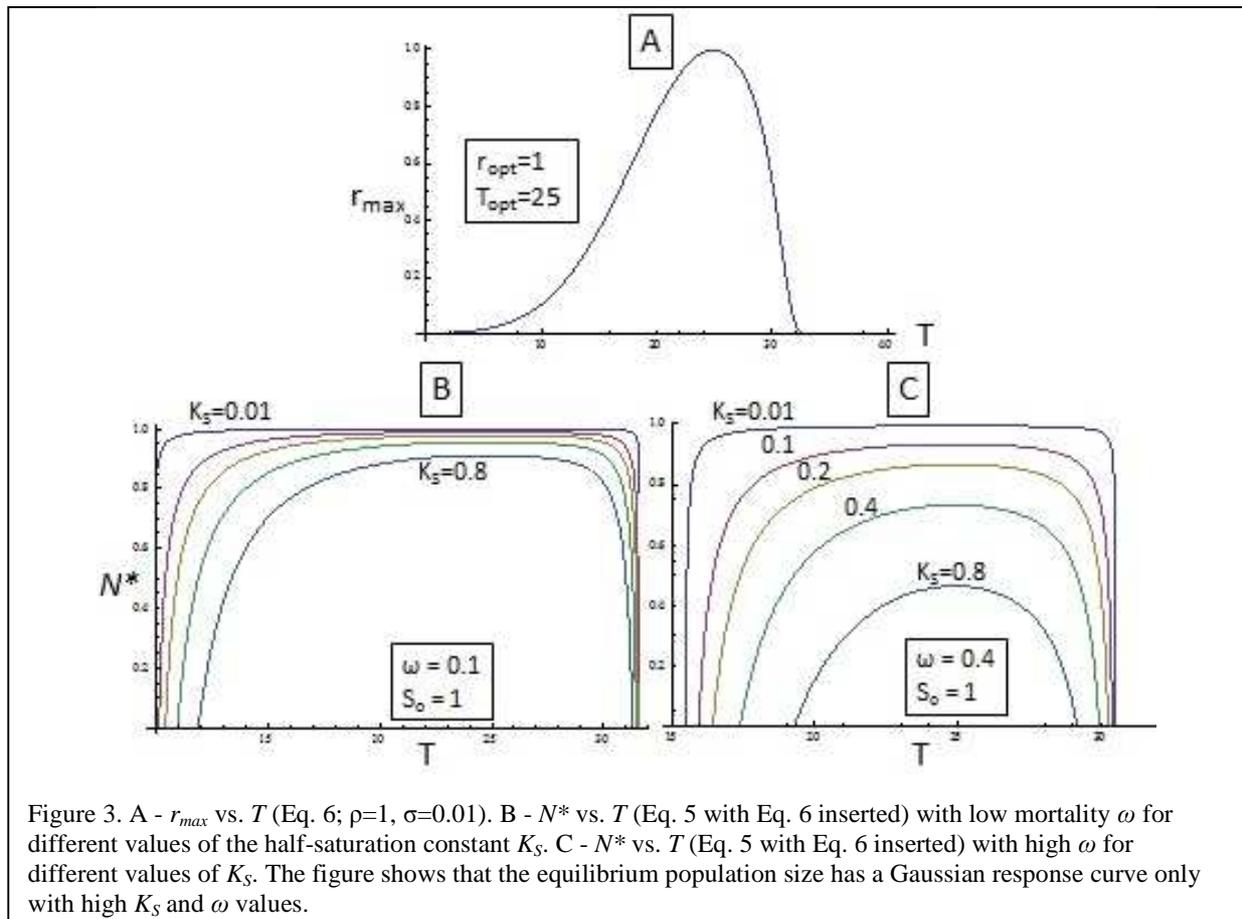


Figure 3. A -  $r_{\max}$  vs.  $T$  (Eq. 6;  $\rho=1$ ,  $\sigma=0.01$ ). B -  $N^*$  vs.  $T$  (Eq. 5 with Eq. 6 inserted) with low mortality  $\omega$  for different values of the half-saturation constant  $K_S$ . C -  $N^*$  vs.  $T$  (Eq. 5 with Eq. 6 inserted) with high  $\omega$  for different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

165 the shape of the response curve. As the mortality rate increases (high  $\omega/r_{opt}$  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

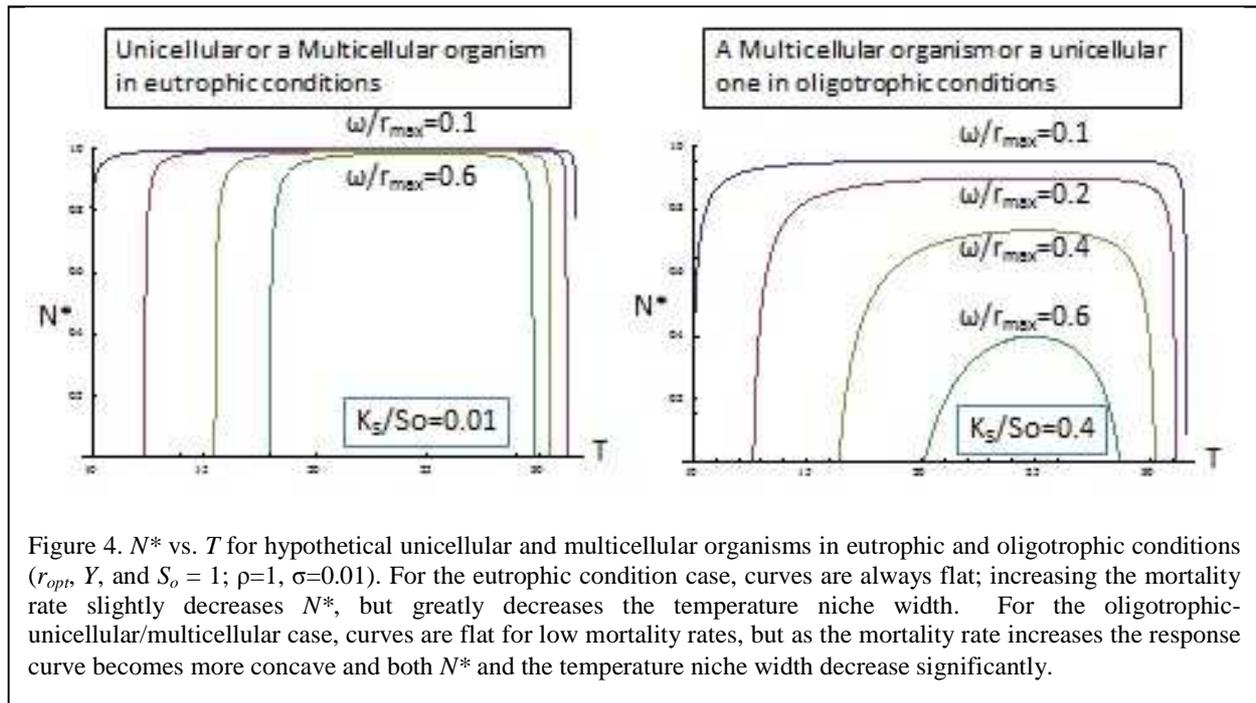


Figure 4.  $N^*$  vs.  $T$  for hypothetical unicellular and multicellular organisms in eutrophic and oligotrophic conditions ( $r_{opt}$ ,  $Y$ , and  $S_0 = 1$ ;  $\rho=1$ ,  $\sigma=0.01$ ). For the eutrophic condition case, curves are always flat; increasing the mortality rate slightly decreases  $N^*$ , but greatly decreases the temperature niche width. For the oligotrophic-unicellular/multicellular case, curves are flat for low mortality rates, but as the mortality rate increases the response curve becomes more concave and both  $N^*$  and the temperature niche width decrease significantly.

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  $\omega$  and  $T$ ? In a chemostat  $\omega$  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  $\omega$  has the same response curve as  $r_{max}(T)$  (Eq. 6), then  $\omega(T) = \omega_{max} r_{max}(T)$ , where  
 225  $\omega_{max}$  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

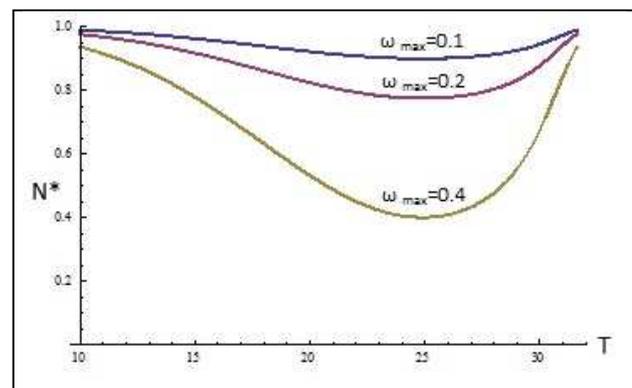
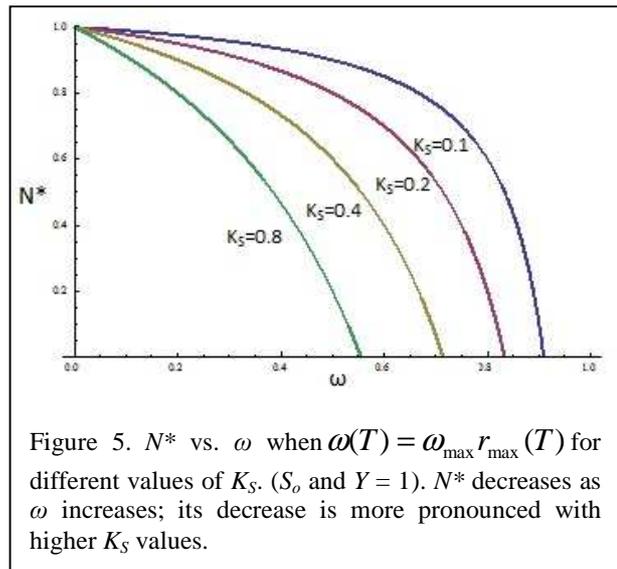
227 becomes temperature independent;  $N^* = Y \left[ S_0 - \frac{K_S \omega_{max}}{(1 - \omega_{max})} \right]$ .

228 In this case,  $N^*$  only depends on  $K_S$  and  $\omega_{max}$ . Figure 5 shows how  $N^*$  decreases as  $\omega$  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

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  $\omega/r_{max}$  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

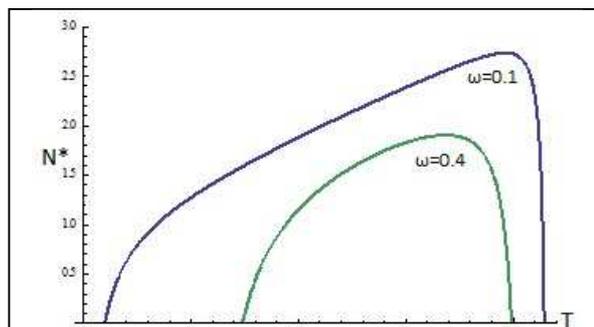


Figure 7. Hotter is smaller.  $N^*$  vs.  $T$  for different values of  $\omega$  ( $\rho=1$ ,  $\sigma=0.01$ ;  $r_{opt}$  and  $S_o = 1$ ,  $K_S=0.4$ ;  $Y = 0.1T$ ) for the case where  $r_{max}(T)$ .  $N^*$  response curves get skewed to higher abundance peaks at higher temperatures because smaller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

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  $\omega/r_{max}$  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.

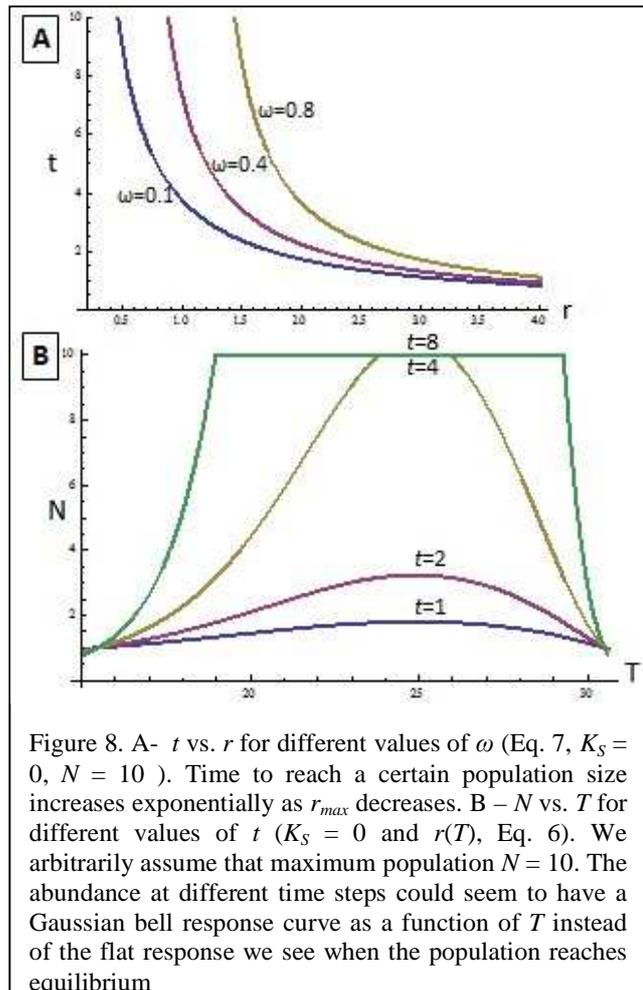


Figure 8. A-  $t$  vs.  $r$  for different values of  $\omega$  (Eq. 7,  $K_S = 0$ ,  $N = 10$ ). Time to reach a certain population size increases exponentially as  $r_{max}$  decreases. B -  $N$  vs.  $T$  for different values of  $t$  ( $K_S = 0$  and  $r(T)$ , Eq. 6). We arbitrarily assume that maximum 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 ( $r$ ,  $\omega$ ,  $N^*$ ,  $K$ ,  $Y$ ) 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

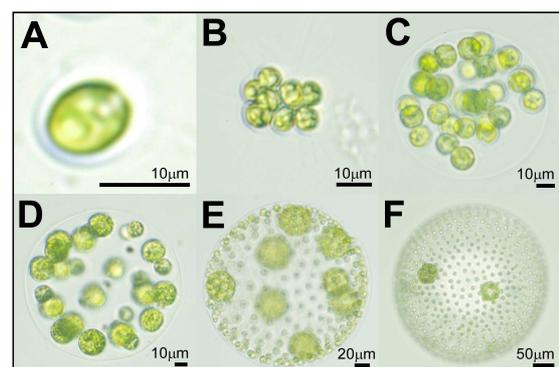


Figure 9. Pictures of various species of Volvocales showing the increase in size and complexity. A- *Chlamydomonas reinhardtii*. B- *Gonium pectorale*. C- *Eudorina elegans*. D- *Pleodorina californica*. E- *Volvox carteri*. F- *Volvox aureus*.

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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