

# From cell size and first principles to structure

Ken H Andersen<sup>1</sup> and A W Visser<sup>2</sup>

<sup>1</sup>Affiliation not available

<sup>2</sup>Center for Ocean Life, Natl. Inst. for Aquatic Resources, Technical University of Denmark

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1 From cell size and first principles to structure  
2 and function of unicellular plankton  
3 communities

4 K.H. Andersen and A.W. Visser

Center for Ocean Life,  
Natl. Inst. for Aquatic Resources, Technical University of Denmark,  
Bygning 202, Kgs. Lyngby, Denmark. E-mail: khand@dtu.dk.

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## Abstract

Here we review, synthesize, and analyse the size-based approach to model unicellular plankton cells and communities. We first review how cell size influences processes of the individual the cell: uptake of dissolved nutrients and dissolved organic carbon, phototrophy, phagotrophy, and metabolism. We parameterise processes primarily from first principles, using a synthesis of existing data only when needed, and show how these processes determine minimum and maximum cell size and limiting resource concentrations. The cell level processes scale directly up to the structure and function of the entire unicellular plankton ecosystem, from heterotrophic bacteria to zooplankton. The structure is described by the Sheldon size spectrum and by the emergent trophic strategies. We develop an analytical approximate solution of the biomass size spectrum and show how the trophic strategies of osmotrophy, light- and nutrient-limited phototrophy, mixotrophy, phagotrophy depend on the resource environment. We further develop expressions to quantify the functions of the plankton community: production, respiration and losses, and carbon available to production of higher trophic levels, and show how the plankton community responds to changes in temperature and grazing from higher trophic levels. We finally discuss strengths and limitations of size-based representations and models of plankton communities and which additional trait axes will improve the representation of plankton functional diversity.

**Keywords—** Cell size, traits, plankton, DOC, Sheldon, mixotrophy

# 1 Introduction

The pace of global change spurs the imperative for predictive, global scale models of marine ecosystems. Key questions that confront us are how the diversity and functioning of marine ecosystems will change, how these changes will impact key ecosystem services such as primary and secondary production, ocean oxygen concentration and carbon sequestration, and whether these services are subject to tipping processes. Traditional models, that have been lovingly calibrated and validated to current-day situations, and through which we have learned so much of marine ecosystem dynamics, are challenged with this task. The world is moving rapidly out of the calibration envelopes for which they were calibrated, and the validation of model predictions with observed ecosystems can no longer be the sole gold-standard measure of model success. In an ideal world predictive, global scale models should be rooted in “first principles”: the rules of the natural world whose validity are considered fundamental and unchanging. In this context, mass and energy conservation, chemical reaction kinetics and evolution by natural selection can be considered examples of first principles. Models of ecosystems do not have recourse to such first principles *per se*. Nevertheless, individual organisms are constrained by first principles that are manifested at all scales of life, from the reaction kinetics and topology of life’s fundamental molecules, the physical limitations of functions of the cells, the circulatory systems, and the geometry of the body plan. One aspect of life where first principle constraints are most evident is in relations to the size of individual organisms (Haldane, 1926; Andersen et al., 2016). Here we attempt to scale from individual organism to ecosystem structure and function. We use unicellular planktonic life as an example where first principles constraints on the individual cell have a particularly strong effect on the ecosystem structure and function (Kjørboe, 1993).

53 Unicellular plankton is an incredibly diverse group of organisms. Taxonomically they  
54 represent four domains of life: archaea, bacteria, algae and protozoa. In terms of cell  
55 size, plankton spans 8 orders of magnitude in mass, the same range as between a beetle  
56 and an elephant. Functionally, unicellular planktonic ecosystems show the entire range  
57 of trophic strategies of primary producers (phytoplankton), grazers and predators (zoo-  
58 plankton), and detritivores (bacteria). The unicellular planktonic food web drives the fast  
59 turnover of inorganic dissolved matter in the oceans, half the global primary production,  
60 the main carbon flux from the photic zone, and the turn-over of inorganic and organic  
61 matter in the world's oceans and lakes. All metazoans – multicellular plankton, jellies,  
62 fish, benthic organisms, and marine mammals – rely on surplus production from unicel-  
63 lular plankton food webs (Ryther, 1969; Stock et al., 2017). Without the unicellular food  
64 web, macroscopic life in the oceans would be extremely impoverished.

65 The difficulty of observing and experimenting with unicellular plankton food webs  
66 have put models in a central position, not just for predictions of responses to changes,  
67 but also for understanding the structure and function of the ecosystems. Any ecosystem  
68 model faces a choice of how to represent the diversity of organisms. The classic food web  
69 approach, which is often applied to higher trophic levels, attempts to resolve all popula-  
70 tions and their interactions with other populations. This approach only works for smaller  
71 isolated ecosystems and is clearly unsuited to unicellular plankton where we rarely have  
72 a clear overview of the full taxonomic diversity. Plankton models instead describe diver-  
73 sity by lumping species into distinct functional groups. The simplest grouping is between  
74 phytoplankton (P) and zooplankton (Z), with phytoplankton representing all phototrophic  
75 organisms and zooplankton their grazers (Franks, 2002). This grouping together with  
76 nutrients (N) lead to “NPZ” models, which have been remarkably successfully in captur-

77 ing the main features of seasonal succession (Evans and Parslow, 1985; Fasham et al.,  
78 1990; Anderson et al., 2015) and global patterns of production (e.g. Palmer and Totter-  
79 dell, 2001). However, their success is contingent on model parameters being tuned to the  
80 observations themselves. In this way, parameters of each group are adjusted to represent  
81 the physiology and ecology of the dominant species in the group within the geographic  
82 region that is modelled. When conditions change though, other species with different pa-  
83 rameters may become dominant and the model no longer represents the new ecosystem  
84 (Franks, 2009). This parameter tuning therefore reduces our confidence in the model's  
85 ability to reproduce ecosystem dynamics when conditions change outside the model's  
86 tuning envelope.

87 A further elaboration of plankton diversity is achieved by breaking the trophic groups  
88 into additional functional groups (Anderson, 2005; Le Quere et al., 2005; Hood et al.,  
89 2006). The functional groups are often aligned with dominant taxonomic groups includ-  
90 ing coccolithophores, dinoflagellates, ciliates, and diatoms, or more general groups, e.g.,  
91 silicifiers, calcifiers etc.. While the functional-group approach introduces additional flex-  
92 ibility and accuracy it does so at the price of increased complexity and additional pa-  
93 rameters. Nevertheless, each group still represents a huge diversity of organisms – for  
94 example, the size range of diatoms spans from a few tens to  $10^7$  cubic micrometers – and  
95 parameters for each group are still tuned to represent the dominant species in the modelled  
96 region. While the introduction of further realism improves the models fit to observations  
97 it does not solve the fundamental problem of parameter tuning. Further, the addition of  
98 new functional groups leads into a complexity trap with a proliferation of state variables  
99 and parameters.

100 Size-based models break free of the complexity trap of functional groups by repre-

101   senting the plankton community with size groups that each represent all cells in a given  
102   size range regardless of their taxonomic affiliation. Technically, each size group is mod-  
103   elled largely in the same way as a functional groups. The main difference is that the  
104   parameters are not independently determined for each size group. Instead, parameters  
105   follow from a smaller set of scaling coefficients and exponents that apply to all sizes. In  
106   this manner size models are flexible with respect to the number of state variable while  
107   retaining a small set of parameters that is, at least in theory, generally valid. Breaking free  
108   of the complexity trap in this manner comes at the cost of a poor representation of taxo-  
109   nomic diversity. However, the size-based model provides a framework where functional  
110   diversity is an emergent property of the model rather than a consequence of its structure.

111       There are other reasons for using cell size as the governing axis of diversity. It  
112   is now well documented that within plankton many of the fundamental rates and pro-  
113   cesses scale with cell size (Fenchel, 1987; Kiørboe, 1993; Finkel et al., 2010; Marañón,  
114   2015): affinities for nutrients (Edwards et al., 2012) or light (Taguchi, 1976; Edwards  
115   et al., 2015), maximum bio-synthesis rates and respiration rates (Kiørboe and Hirst, 2014),  
116   clearance rates (Kiørboe and Hirst, 2014), predator-prey mass ratios (Hansen et al., 1994)  
117   and predation risk from larger organisms (Hirst and Kiørboe, 2002). Importantly, many  
118   of these scaling relations emerge from fundamental physical limitations due to geome-  
119   try (light affinity), diffusion (affinity for dissolved organic matter), and fluid mechanics  
120   (e.g. Stokes' law or feeding mechanics (Nielsen et al., 2017)). In other words: the pa-  
121   rameters are constrained by first principles from geometry or classical physics. A further  
122   advantage of size-based models is the conceptual simplicity that comes from being based  
123   on a general description of a single cell. The simplicity extends to the implementation,  
124   which only needs a small parameter set and have simpler code. These advantages make

size-based descriptions appealing to add diversity within a functional group (Terseleer et al., 2014; Stock et al., 2014) or for the full model structure. Existing size-based models mostly rely on empirical relationships between size and parameters such as half-saturation coefficients, maximum growth rates etc.. This approach facilitates a good fit with observations. Here, we instead try to establish the fundamental mechanisms and strive to determine parameters from fundamental principles by reviewing the literature on the theory of size-based relations with cell size.

Size-based models of plankton have a long history (Armstrong, 1994; Moloney and Field, 1989; Baird and Suthers, 2007; Stock et al., 2008; Banas, 2011; Negrete-García et al., 2022). Size-based concepts are now increasingly used in biogeochemical models to increase the diversity within functional groups according to size (Terseleer et al., 2014; Dutkiewicz et al., 2020; Stock et al., 2014). Most size-based models retain the distinction of functional trophic groups by operating with separate phyto- and zooplankton size distributions (Poulin and Franks, 2010; Ward et al., 2018). A recent strand is purely size-based models where the only difference between cells are their size and no *a priori* distinction between trophic strategy is imposed (Ward and Follows, 2016; Ho et al., 2020; Chakraborty et al., 2020). Such models completely forgo taxonomic-oriented assumptions about the function of the modelled groups (Andersen et al., 2015). All functional differences between size groups and of the community are emergent properties of the model.

There exists an abundance of reviews on the empirical relationships between cell size and various processes (Kiørboe, 1993; Hansen et al., 1994; Finkel et al., 2010; Edwards et al., 2012; Kiørboe and Hirst, 2014; Marañón, 2015; Hillebrand et al., 2021). They tend to focus on phytoplankton and upon describing size-relations as a single power-law func-



tion. However, in many cases there is more than one underlying physical process at play. This means that there are transitions between one power-law relation and another, e.g., between nutrient diffusion and surface uptake (Armstrong, 2008) or between maximum synthesis rates and nutrient uptake (Ward et al., 2017). Such transitions at characteristic sizes often lead to important transition in the ecosystem structure (Andersen et al., 2016), for example between phototrophs, mixotrophs, and heterotrophs (Andersen et al., 2015). Identifying characteristic sizes where there is a cross-over between two power-law relations is perhaps even more important for ecosystem structure and function than the power-law relations themselves.

Here we review existing knowledge of size-based relationships for unicellular plankton, from bacteria to zooplankton, and attempt a synthesis that demonstrates the importance of size-based relations for emergent ecosystem structure and function. Our ambition is to identify the first principles responsible for the size-based relations, thereby tying parameters to physical and chemical processes and geometry. Our synthesis show how size-based relationships determine community-level patterns of biodiversity and ecosystem function: the viable size-range, competition, biomass size structure, ecosystem primary and secondary production, and trophic efficiencies. By focusing on the processes related to cell size, we demonstrate the power of these relations for determining community-level patterns and ecosystem functions. The work is organised in five parts. After an initial discussion of the concept of “size” of a cell, we review the relations governing resource uptake, losses, and biosynthesis of a cell, including the theory that links these processes to first principles. Second, we exploit the simple form of the size-based relations to derive analytical solutions for the smallest and largest cell sizes, and for the limiting resources. Third, we scale from the cell-level process to the community size distribution and explore

173 emergent trophic strategies. We derive a scaling solution of the biomass size distribution  
174 and explore the trophic strategies and compare with simulations of the size-based model  
175 in a chemostat. From the emergent size spectrum and trophic strategies we derive ecosys-  
176 tems functions, including production, and show how the plankton community responds to  
177 predation by higher trophic levels or changes in temperature. Our aim is to make a mini-  
178 mal size-based model framework where we prioritize simple conceptual implementation  
179 and analytical analysis over capturing complete and accurate biogeochemistry. Never-  
180 theless we show that the model gives reasonable predictions of biomass and production.  
181 Overall, our synthesis highlights the importance of fundamental first principles for con-  
182 straining the unicellular plankton communities and their related functions. We finish by  
183 discussing the limitations of the size-based approach and prioritize which additional traits  
184 will best improve the representation of functional diversity.

## 185 **2 Measures of cell size**

186 The size of a cell can be measured in two ways: by its physical size – radius or volume –  
187 or by its mass, e.g., mass or moles of carbon or nitrogen. There is no universally optimal  
188 measure; for some processes physical size is most relevant, for some it is the mass, and  
189 for others both measures of size matter. For example, the settling velocity due to Stokes’  
190 law is determined by both the physical size and the mass of the cell. In general, physical  
191 size is mostly used to describe limitations due to geometry, e.g., surface limitation, while  
192 mass is used to describe metabolism and mass budget of the cell.

193 Unicellular plankton display an astonishing diversity in cell shape (Ryabov et al.,  
194 2021). The functional role of cell shape is largely unknown, though it is conjectured to be

195 related to defence from predation (Smetacek, 2001). For simplicity we ignore the diversity  
 196 of shapes (except for its relation to the minimum size in Section 4.1), and consider cells  
 197 to be spherical with physical size characterized by radius  $r$ . Conversion between physical  
 198 size (equivalent spherical radius  $r$ ) and mass of substance  $X$ ,  $m_X$  is then:

$$m_X = \rho_{C:X} \rho \frac{4}{3} \pi r^3 \Leftrightarrow r = \left( \frac{3m_X}{4\pi \rho_{C:X} \rho} \right)^{1/3}, \quad (1)$$

199 where  $\rho$  is the carbon density (carbon mass per volume) and  $\rho_{C:X}$  is the elemental mass  
 200 ratio between carbon and  $X$ .

201 In the following, mass is considered as carbon mass and the subscript C is suppressed  
 202  $m = m_C$ . For the theoretical calculations we use a density of  $\rho = 0.4 \cdot 10^{-6} \text{ } \mu\text{gC}/\mu\text{m}^3$  and  
 203 Redfield elemental ratios. Conversion between physical size and mass needs to account  
 204 for differences in density. In particular diatoms are special due to their vacuole which  
 205 lowers their density. Here we use the comprehensive compilation of Menden-Deuer and  
 206 Lessard (2000) that explicitly distinguishes between diatoms and other protists to convert  
 207 observations of cell size to cell mass.

208 Not all of the cell's mass  $m$  is available for functions of biosynthesis (ribosomes), light  
 209 harvesting (chloroplast) etc. Some part of the cell is devoted to the cell membranes, DNA  
 210 and RNA, (Kempes et al., 2016). The cell membrane and cell wall takes up a fraction of  
 211 the cell mass (Raven, 1994; Marañón, 2015). For a spherical cell the fraction of the cell  
 212 used by the membrane and wall is approximately:

$$\nu \approx 3 \frac{\delta}{r}, \quad \text{for } r \gg \delta \quad (2)$$

213 where  $\delta \approx 50 \text{ nm}$  is the thickness of the cell wall and membrane (adjusted a bit down

214 from 70-80 nm as given by Raven (1987) to correct for the approximation used in Eq. 2).  
 215 The effective functional mass is therefore  $m(1 - \nu)$ . As the cell wall fraction scales as  
 216  $1/r$ , small cells will be severely limited in the functions due to the material cost of cell  
 217 membrane and wall. Kempes et al. (2016) further considered the limitation of DNA and  
 218 RNA, however, the most limiting factor was the cell wall and membrane.

### 219 **3 Effects of cell size on fundamental rates: resource** 220 **uptake, losses, and biosynthesis**

228 Ecosystem dynamics are driven by individual cells acquiring and processing resources,  
 229 eventually leading to cell division and cell growth. This section reviews how cell size  
 230 determines the uptakes of resources: dissolved nutrients, inorganic carbon through photo-  
 231 harvesting, dissolved organic carbon, and feeding on other, typically smaller, organisms,  
 232 and how these uptakes are determined by first principles. Some of the acquired resources  
 233 are lost through passive exudation or used for respiration. The remaining resources are  
 234 used for biosynthesis (Fig. 1).

#### 235 **3.1 Resource uptake**

236 Cells take up resources through three mechanisms: diffusive uptake of dissolved organic  
 237 carbon (DOC) and inorganic matter ( $N$ ), photoharvesting of light ( $L$ ), and phagotrophic  
 238 uptake of particulate matter ( $F$ ). The potential uptake of resource  $X$  is proportional to the  
 239 resource concentration:

$$j_X = a_X(m)X\rho_{C:X}, \quad X \in \{\text{DOC}, N, L, \text{ or } F\} \quad (3)$$

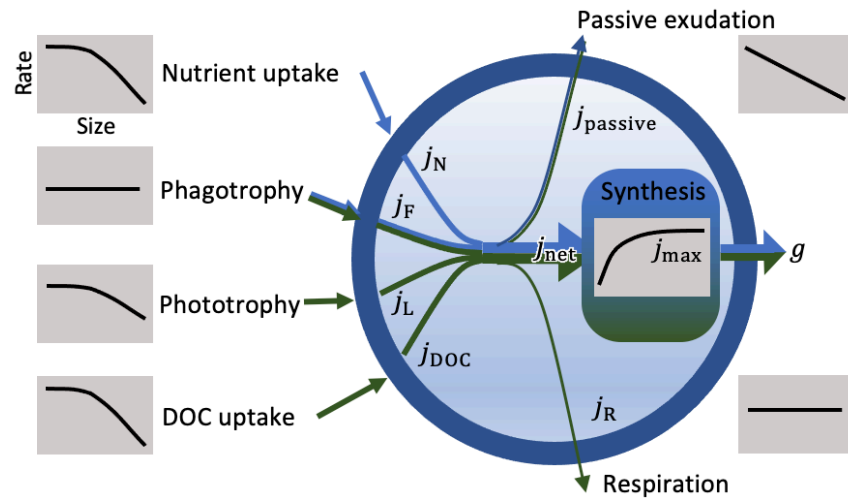


Figure 1: Sketch of the fluxes of nutrients (blue) and carbon (green) in and out of a cell. The grey insets sketches the size-dependency of each mass-specific rate (units of 1/time). Uptakes of nutrients  $j_N$ , food  $j_F$ , photoharvesting  $j_L$ , and dissolved organic carbon  $j_{DOC}$  are subjected to losses from respiration  $j_R$  and passive exudation  $j_{passive}$  before they are synthesised with a maximum rate  $j_{max}$ . The end result is the growth (i.e. division) rate  $g$ .

240 where  $j_X$  is the mass specific flux (in units of  $\text{g}_C/\text{g}_C/\text{time}$ ),  $a_X$  is the mass specific affinity  
 241 (volume/day/ $\text{g}_C$ ),  $X$  is the resource in units of  $X$  per volume, and  $\rho_{C:X} = \rho_{C:N}$  is the  
 242 C:N ratio for diffusive uptake of nutrients. By multiplying N uptake with the fixed C:N  
 243 ratio all the fluxes are measured in the same units and are therefore directly comparable  
 244 (uptake of food and light is measured in units of carbon as  $\rho_{C:F} = \rho_{C:L} = 1$ ).

245 Note that we characterise a cells resource uptake ability by the affinity  $a_X$ , following  
 246 Aksnes and Cao (2011); Fiksen et al. (2013); Flynn et al. (2018). This choice contrasts the  
 247 commonly used Monod/Michaelis-Menten formulation of the functional response, where  
 248 uptake is described with a half-saturation coefficient and a maximum uptake rate. In a  
 249 mechanistic context, the Monod formulation of the uptake rate is problematic because the  
 250 half-saturation coefficient cannot be associated with a physical or physiological charac-  
 251 teristic of the cell – it acts purely as a convenient fitting parameter. Mathematically, the  
 252 affinity follows from the Monod formulation as the product of the half-saturation coeffi-  
 253 cient and the maximum synthesis rate, which we use to relation to calculate affinities from  
 254 literature sources of half saturation coefficients. The Monod formulation also includes the  
 255 process of saturation, which we return to later. Separating the processes of encounter  
 256 and biosynthesis explicitly with two different parameters (affinity and maximum synthe-  
 257 sis rate) avoids the pitfalls of considering the half-saturation constant as a physiological  
 258 trait (Kiørboe and Andersen, 2019).

259 The affinity  $a_X$  measures the cell’s ability to encounter and assimilate resource  $X$ .  
 260 The affinity is determined partly by encounter with the resource and partly by the cell’s  
 261 investment in capacity to take up and assimilate the resource (Shuter, 1979; Bruggeman  
 262 and Kooijman, 2007; Chakraborty et al., 2017). The encounter results from the physical  
 263 processes of diffusion, self-shading, and fluid dynamics. The limitation due to uptake ca-

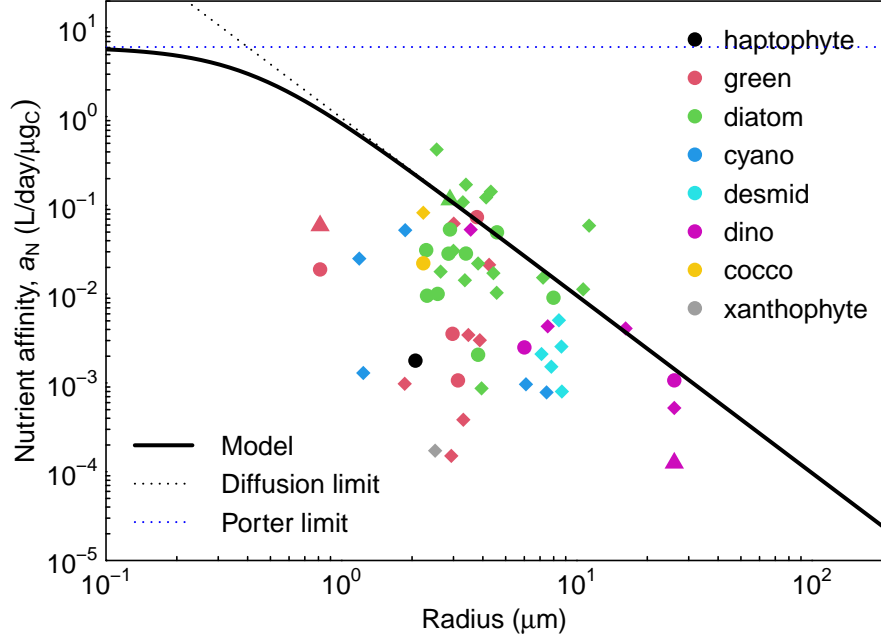
264 capacity is relevant when the cell encounters abundant amounts of the resource but is unable  
265 to process it all by its uptake machinery, e.g., porters for diffusive uptake, light harvest-  
266 ing machinery, or phagotrophic assimilation. For all resource uptakes, the mass-specific  
267 affinity is constant or decreases with size, as we will show below. Uptake limitation is  
268 most prominent for small cells that have high affinities, leading to a higher encounter with  
269 resources that they can process. If investments in uptake capacity scales with the mass  
270 of the cell, the uptake limitation of mass-specific affinity is independent of size. Small  
271 cells therefore have limited ability to increase their uptake capacity, and their affinity will  
272 be limited by uptake capacity. The affinity therefore has two size-scaling regimes: for  
273 small sizes the affinity is independent with size (uptake limitation), and for larger sizes it  
274 is constant or declining with size (encounter limitation) (e.g. Armstrong, 2008).

275     The processes that determine encounter and uptake capacity, and how they scale with  
276 cell size, depend on the type of resource.

### 285 **3.1.1 Encounter and uptake of dissolved matter**

286 The theory behind uptake of dissolved matter is well developed, as reviewed by Fiksen  
287 et al. (2013). Nutrient uptake is limited by three processes: the rate at which molecules  
288 diffuse towards the cell, the rate at which nutrients are transported across the cell mem-  
289 brane by porters, and the capacity of the cell to utilize nutrients in biosynthesis.

290     The flux of molecules towards a sphere was shown by Pasciak and Gavis (1974) to  
291 be proportional to the sphere's radius and the difference between the concentration far  
292 away and at the surface of the sphere. Assuming that the sphere absorbs all encountered



277

278 Figure 2: Specific nutrient affinity ( $a_N$ ) as a function of radius. Triangles: am-  
 279 monium uptakes; circles: nitrate uptake; diamonds: phosphorous uptake. The  
 280 dotted lines are the theoretical maximum affinity due to diffusion limitation and  
 281 porter limitation. The solid line is a fit-by-eye of the radius where porter limitation  
 282 becomes important, around  $r_D^* = 0.75\mu\text{m}$ . Data from Edwards et al. (2015). Con-  
 283 versions between volume and mass are done using the relations in Menden-Deuer  
 284 and Lessard (2000).

293 molecules the concentration at the surface is zero and the mass-specific affinity becomes:

$$a_D = \frac{3D}{\rho} r^{-2} = \alpha_D r^{-2}, \quad (4)$$

294 where  $D$  is the diffusivity of the dissolved molecules,  $\rho$  is the cell carbon density, and  $r$   
 295 is the cell radius. However, if the cell is embedded in an external flow, such as turbulence  
 296 or when the cell is sinking, then the boundary layer around the cell will be smaller and  
 297 the flux of molecules increased. The increase in flux due to such advective flows is char-



acterized by the Sherwood number, which is the dimensionless ratio between transport by advection and diffusion (Kiørboe, 1993). A Sherwood number  $\gg 1$  means that the transport is enhanced by advection. However, Kiørboe (1993) found that for most cases the Sherwood number is very close to 1, such that Eq. 4 does not have to be corrected for advective effects.

The simple scaling in of affinity in Eq. 4 has formed the start of an extensive theoretical discussion of additional effects cell size on the affinity (reviewed by Fiksen and Jørgensen, 2011). We provide a full mathematical derivation in Box 1 and proceed with qualitative arguments here. At small cell radius, where the mass specific affinity is very high, uptake might become limited by either the number and capacity of porters, or by the cells' ability to process incoming nutrients. Berg and Purcell (1977) accounted for uptake limitation by introducing an extra term in Eq. 4 (see Box 1 for complete derivation and discussion):

$$a_D(m) = \alpha_D r^{-2} \frac{1}{1 + (r/r_D^*)^{-2}} \quad (5)$$

where  $r_D^*$  is the cell size at the cross-over between uptake (porter/processing) limitation and diffusion limitation. Small cells  $d \ll r_D^*$  are porter/processing limited with affinity  $a_D = 4\pi D/r_D^{*2}$  while larger cells,  $d \gg r_D^*$  are diffusion limited with  $a_D = 4\pi D r^{-2}$  (Fig. 2). Precisely what controls the cross-over size  $r_D^*$  remains uncertain. Geometric consideration based on the size and density of porter site on the cell have been explored (Casey and Follows, 2020; Armstrong, 2008) as have the kinetics of porter handling times and energy costs (Aksnes and Egge, 1991) (Box 1) but remain unresolved.

As a practical solution to determine the cross-over size between diffusive encounter limitation and uptake limitation, we turn to observations. The available data are, however, very scattered (Fig. 2; see Table 1 for a summary of all parameters). The data do con-

321 firm the theoretical prediction of an upper limit to encounter by diffusion limitation. The  
 322 data also indicate that the affinity of smaller cells (smaller than around  $r_D^* = 0.75 \mu\text{m}$ ) is  
 323 limited by another process than diffusion limitation, which could be porter or uptake lim-  
 324 itation. It should be noted, though, that the data do not lend much support to the value of  
 325  $r_D$ , only that it should not be less than  $1 \mu\text{m}$ . Below these two upper limits there is a large  
 326 scatter in the data with some species having a factor 1000 smaller affinity for phospho-  
 327 rous. Our interpretation of this scatter is that species adapted to high nutrient loads, like  
 328 the fresh-water green algae, are not diffusion or porter limited. They therefore invest less  
 329 in nutrient uptake with the result that the affinity is smaller than it could potentially be.  
 330 In the following we use the diffusion/porter limitation to define nutrient affinity as it well  
 331 represents the affinity in communities with strong nutrient competition. In other com-  
 332 munities, e.g. during a spring bloom where nutrients are plentiful, it does not matter that  
 333 this formalism predicts a too affinity as growth will be limited by the ability to perform  
 334 biosynthesis and not by nutrient uptake. In conclusion, we have a fully developed theo-  
 335 retical apparatus to understand the maximum affinity of cells to dissolved organic matter,  
 336 however, we need a better understanding of the specific processes related to molecule  
 337 capture to fully relate the limitation at small cell sizes to fundamental processes.

### Box 1: derivation of nutrient affinity

The diffusive flux of a substance  $N$  to a partial absorbing sphere of radius  $r$  has a well known solution:

$$Q = 4\pi r D (X - X_0) \quad (6)$$

where  $D$  molecular diffusivity, and  $N$  and  $N_0$  the nutrient concentration at distance and on the cell surface respectively. For a perfectly absorbing sphere  $N_0 = 0$  and the flux becomes  $Q = 4\pi r D N$ . A real cell however is not perfectly absorbing but is covered by a finite number of uptake sites in an otherwise impervious cell membrane. A classic result (Berg and Purcell, 1977) considers the cell surface is covered by  $n$  porter sites each of radius  $s$ . If sites are small (specifically  $s \ll r$ ), sparsely distributed, and perfectly absorbing, then the diffusive flux towards each site is  $Q_s = 4sDN_0$ . For  $n$  such sites then

$$Q = 4\pi r D (N - N_0) = 4nsDN_0 \quad \Rightarrow N_0 = N \frac{\pi r}{\pi r + ns} \quad (7)$$

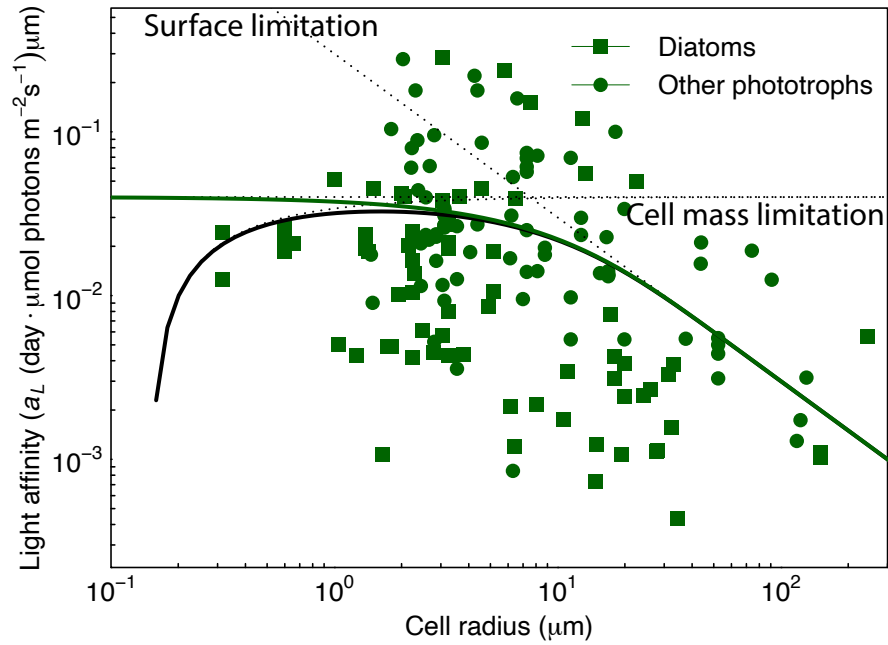
which leads to:

$$Q = 4\pi r D N \frac{ns}{\pi r + ns}. \quad (8)$$

A correction accounts for potential interference of diffusive fluxes when porter sites are tightly packed (Zwanzig, 1990). Specifically, expressing the surface fraction of porters as  $p = ns^2/(4r^2)$

$$N_0 = N \frac{\pi r(1-p)}{\pi r(1-p) + ns} \quad \Rightarrow Q = 4\pi r D N \frac{ns}{\pi r(1-p) + ns}. \quad (9)$$

369 As  $p \rightarrow 1$  (i.e. the entire cell surface becomes covered with perfectly absorbing porter  
 370 sites)  $N_0 \rightarrow 0$  and  $Q \rightarrow 4\pi rDN$ . While theoretically sound and widely built upon,  
 371 these results are actually not particularly germane to the question of nutrient uptake  
 372 in plankton. In the first instance, for typical cell sizes and porter sizes, the correction  
 373 (Eq. 9) saturates extremely rapidly so a very low porter density is sufficient to achieve  
 374 near maximum uptake flux (Jumars et al., 1993). This implies that limitation of the  
 381 number of porter sites due to surface crowding is unlikely to be an issue. Secondly, it  
 375 is not realistic that uptake sites are perfectly absorbing discs. While diffusion towards  
 376 the sites is a fair representation, uptake requires active transport across the cell wall  
 377 (Aksnes and Egge, 1991; Armstrong, 2008), a process that (1) occupies the uptake  
 378 site for a finite amount of time and (2) is energetically costly, requiring about 1 mole  
 379 of ATP per mole of nutrient transported.  
 380



383

384 Figure 3: Light affinities of protists as a function of carbon mass compared to the  
 385 first-principles formulae (Eq. 13; thick black line and (Eq. 12); green line). The  
 386 three limiting factors: cells mass, cell surface, and cell membrane are shown with  
 387 dotted lines. The black line is the total affinity. Data from Edwards et al. (2015),  
 388 corrected for day length.

### 3.1.2 Light harvesting; theory and data

#### Box 2: derivation of light affinity

The net absorption of light by a cell depends on the density and distribution of individual chromophores within the cell's cytoplasm. For a spherical cell (radius  $r$ ) with uniformly distributed chromophores throughout the cell volume (number density  $c$  ( $\mu\text{m}^{-3}$ ), optical cross section  $a$  ( $\mu\text{m}^2$ )), the rate at which photons are absorbed is given by:

$$Q(r, \lambda) = \frac{\pi L}{2\lambda^2} [(1 + 2\lambda r) \exp(-2\lambda r) + (2\lambda^2 r^2 - 1)] , \quad (10)$$

where  $\lambda = ac$  ( $\mu\text{m}^{-1}$ ) is the light absorption coefficient within the cytoplasm, and  $L$  ( $\mu\text{mol m}^{-2}\text{s}^{-1}$ ) is the light flux (Duyens, 1956; Kirk, 1975). This relationship, while exact for a sphere, is somewhat clumsy. A more accessible formulation, developed by Hansen and Visser (2019), assumes a cylindrical cell with the same volume and cross-sectional area as the sphere. Under this geometry, the optical path through the cell is  $4r/3$  and:

$$Q(r, \lambda) = \pi L r^2 (1 - \exp(-4\lambda r/3)) . \quad (11)$$

Given that these formulae give very nearly identical results, and that cell shape is always a confounding factor, we opt for the simpler. From the form of (Eq. 11) it is clear that for large cells with a high investment in chromophores ( $\lambda r \gg 1$ ), photon absorption is proportional to the cell's cross-sectional area  $Q \approx \pi r^2 L$  whereas for small cells with low chromophores investment ( $\lambda r \ll 1$ ), photon absorption is proportional to cell volume  $Q \approx 4/3 \pi r^3 \lambda L$ .

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The mechanisms relating photon absorption to carbon fixation are complex and dependent a variety factors including photon energy, type of pigments and details of the photosystem used. While some of these aspects are accessible to modelling, we use the commonly used quantum yield  $y$  (gC/(mol photon)) as a simplification (Emerson, 1958). The specific light affinity then becomes  $a_L = y(Q/L)/m$ . We can also write  $\lambda = \kappa_L \phi_L$  relating the cell's absorption coefficient to  $\phi_L$ , the fraction of its carbon mass invested in light harvesting where  $\kappa_L$  the constant of proportionality. Observations indicate that  $\lambda = 0.1 \mu\text{m}^{-1}$  (Raven, 1984, 1997) when about half of the cell's mass is devoted to light harvesting, suggesting that  $\kappa_L = 0.2 \mu\text{m}^{-1}$ . It follows then that

$$a_L = \frac{3y}{4\rho} \frac{1}{r} (1 - \exp(-4\kappa_L \phi_L r/3)), \quad (12)$$

which is identical to Eq. 13 with parameters  $(\alpha_L, r_L^*)$  corresponding to  $(3y/(4\rho), 3/(4\kappa_L \phi_L))$  respectively. Quantum yield estimates ranges from 0.12 to 0.6 gC/(mol photon) (Kishino et al., 1986). Using  $y = 0.16$  gC/(mol photon) suggests  $\alpha_L = 0.30 (\text{d } \mu\text{mol m}^{-2}\text{s}^{-1})^{-1} \mu\text{m}$  and  $r_L^* = 7.5 \mu\text{m}$ .

Fig. 3 shows that there are cells with almost a factor 10 higher affinity that predicted by Eq. 12. The source of this variation is likely due to uncertainty in the quantum yield  $y$ , which depends on the type of pigment and the wavelength of the light (Kishino et al., 1986).

436        Photosynthesis is fundamentally powered by the capture of photons by light harvest-  
 437        ing complexes, and the number of photons captured by a cell depends on both the number  
 438        of photons incident on the cell, as well as the number of light harvesting complexes within  
 439        the cell. In terms of scaling, it can be reasoned then that the former depends on the cross  
 440        sectional area of the cell, while the latter on some proportion of its functional carbon  
 441        mass. However, light harvesting complexes shade one another and in larger cells not all  
 442        complexes can be equally effective for light harvesting (Kirk, 1975; Morel and Bricaud,  
 443        1981). The affinity for light harvesting therefore transitions from being independent of  
 444        size for small cells to being proportional to the surface area for large cells (see Box 2):

$$a_L = \frac{\alpha_L}{r} \left( 1 - e^{-r/r_L^*} \right) (1 - \nu). \quad (13)$$

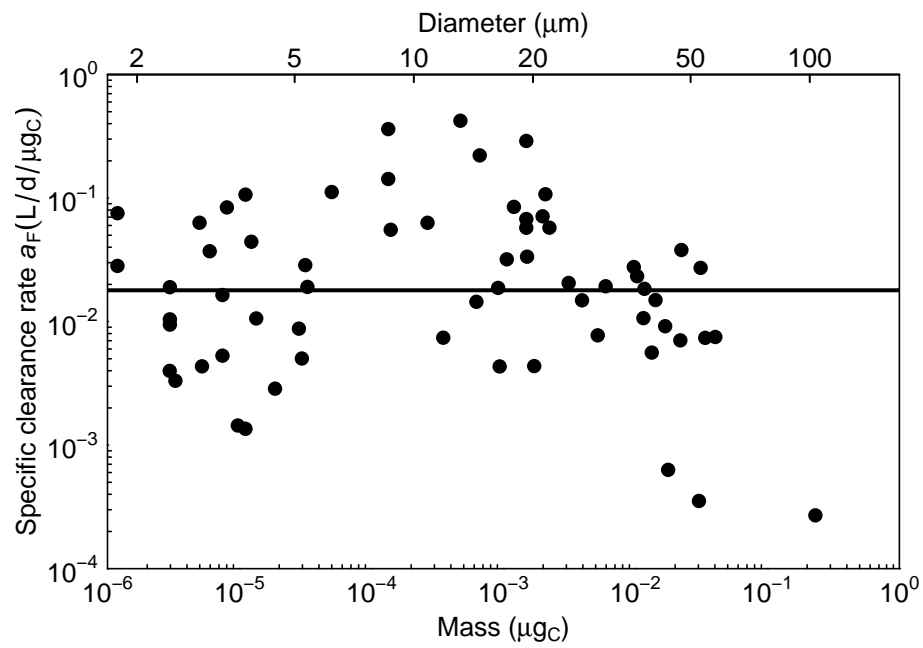
445        This formulation of affinity has asymptotic scaling of  $a_L \rightarrow \alpha_L/r_L^*$  for intermediate cells,  
 446         $a_L \rightarrow \alpha_L/r$  for  $r \gg r_L^*$  and goes to zero for small cells (the factor  $1 - \nu$ ).

447        Previous analyses of light affinity has focused on fitting just one power law and has  
 448        consistently found a scaling close to the predicted surface law  $\propto r^{-1}$  (Taguchi, 1976;  
 449        Finkel et al., 2010; Edwards et al., 2015). Our reanalysis of the available data indicates  
 450        a transition from mass to surface scaling with a transition size around  $r_L^* \approx 7.5 \pm 3 \mu\text{m}$   
 451        (Fig. 3) in accordance with the first-principles argument in Box 2. The divergence to zero  
 452        due to the cell wall limitation  $1 - \nu$  for smaller cells is consistent with a lower limit of  
 453        one chloroplast at cell volume of  $\approx 1 \mu\text{m}^3$  or  $r \approx 0.5 \mu\text{m}$  (Okie et al., 2016).

454        As with the nutrient affinity there is a large scatter in the data of one order of magni-  
 455        tude around the first-principle prediction. Here, though, the prediction does not reflect the  
 456        upper limit of light affinity rather an average estimate. This indicates that some plankton  
 457        can invest more in light harvesting to increase their affinity. In developing the prediction



458 we assumed that plankton invest at most half of their cell mass to light harvesting. Cells  
459 might invest more if they are fully dedicated to light harvesting in low light environments  
460 leading to a higher affinity. Further, the quantum yield is uncertain and a higher value is  
461 within the observed range. However, the absolute value of the affinity is less important  
462 for the plankton community than how affinity scales with cell size. In a water column, the  
463 production maximum adjusts itself vertically to the point where light limitation matches  
464 nutrient limitation (Ryabov et al., 2010). Therefore, a higher light affinity leads to a  
465 deeper production maximum and vice versa. The overall value of the affinity is therefore  
466 less important for the general production of the plankton community because production  
467 will be limited by nutrients, unless the light is so low that production can only occur in  
468 the surface. What is important, though, for the structure of the trophic strategies with cell  
469 size is that the specific affinity decreases with cell size overall, and that decline is well  
470 borne out by the data.



471

472 Figure 4: Specific clearance rate ( $a_F$ ) as a function of carbon mass. Data of  
 473 nanoflagellates, dinoflagellates, and ciliates from Kiørboe and Hirst (2014).

### 475 3.1.3 Phagotrophy 474

#### 476 Box 3: Derivation of clearance rate

477 The specific clearance rate  $a_F$  (volume/time/cell mass) can be estimated from the  
478 work required to displace the fluid that a cell moves through or filters. We assume  
479 that the work is approximately the same as pushing a sphere through the fluid, i.e.,  
480 given by Stokes' law:  $W = 12\pi\mu u^2 r$ , where  $u$  is the velocity,  $r$  the cell radius, and  
481  $\mu$  the dynamic viscosity of water. The metabolic power that the cell has available to  
482 filter water scales with the cell's mass  $cm\rho_e$ , where  $c$  is the fraction of the cell's mass  
483 that can be used for swimming and  $\rho_e$  is the energy density of the cell. Equating the  
484 work needed and the power available gives the velocity as:

$$485 \quad u = \sqrt{\frac{1}{12\pi} \frac{c\rho_e}{\mu r} m}.$$

486  
487 Assuming that the cell clears an area corresponding to its own cross section we get  
488 the specific clearance rate as the clearance area  $\pi r^2$  multiplied by the velocity and  
489 divided by the mass:

$$490 \quad a_F = \sqrt{\frac{c\rho_e}{4\mu\rho}}, \quad (14)$$

491  
492 using Eq. 1 to convert between radius and mass. The specific clearance rate is con-  
493 stant (independent of cell size). With a dynamic viscosity of  $\mu = 1 \text{ g/(m s)}$ , energy  
494 density  $\rho_e = 40 \cdot 10^3 \text{ m}^2 \text{ g s}^{-2} \text{ g}_C^{-1}$  (Boudreau and Dickie, 1992), and that the fraction  
495 of body mass used for driving the flow is  $0.1 \text{ day}^{-1}$  gives  $a_F = 0.0073 \text{ l/day}/\mu\text{g}_C$ ,  
496 very close to the geometric average of 0.018 from the data in Fig. 4.  
498

499 Phagotrophy is the ingestion of food particles, typically smaller cells. Prey cells are  
500 encountered either by the predator moving through the fluid or with the predator creating

501 a feeding current that brings prey towards it (Kiørboe, 2011). In this case the affinity is  
 502 the clearance rate, i.e., the volume of fluid cleared of potential prey per time. Hansen  
 503 et al. (1997) showed that the half saturation constant and the maximum consumption rate  
 504 was roughly constant among unicellular plankton, which corresponds to a constant mass  
 505 specific clearance rate. Kiørboe (2011) expanded the analysis and showed that the clearance  
 506 rate was approximately  $10^6$  cell volumes per day, though variations exist among feeding  
 507 modes (passive, active, cruising or feeding current). It appears evident that the scaling  
 508 of clearance rate with cell size should emerge from fluid mechanic constraints. Despite  
 509 arguments having been made for fish, they have not been made for unicellular plankton.  
 510 We develop an argument in Box 3 that reproduces the observed constant specific clearance  
 511 rates and also gets the average value reasonably correct (Fig. 4).

512 For the actual food consumption, we also need to consider the limitation imposed by  
 513 assimilation over the food vacuole membrane. The surface area of the vacuole scales  $\propto r^2$   
 514 and the specific maximum assimilation therefore scales with  $r^{-1}$ . We can then describe  
 515 the uptake with a classic functional response with affinity  $a_F$  and maximum assimilation  
 516 rate  $c_F r^{-1}$ :

$$j_F = \epsilon_F c_F r^{-1} \frac{a_F F}{a_F F + c_F/r}. \quad (15)$$

517 where  $\epsilon_F$  is the assimilation efficiency. This formulation has the limit  $j_F \rightarrow \epsilon_F a_F F$  for  
 518 smaller cells and  $j_F \rightarrow \epsilon_F c_F/r$  for larger cells with the cross-over size between the two  
 519 regimes being food-dependent:  $r_F^* = c_F/(\epsilon_F a_F F)$ . We do not have any direct measure-  
 520 ments of the assimilation limitation, however, we will use the measurements of maximum  
 521 growth rate of larger cells to estimate this process as  $c_F \approx 30 \mu\text{m/day}$ . It can be argued  
 522 that the reduction in functional mass of small cells (the factor  $\nu$ ) should lead to a reduction  
 523 in phagotrophy for small cell, similar to the reduction in phototrophy. However, phagotro-

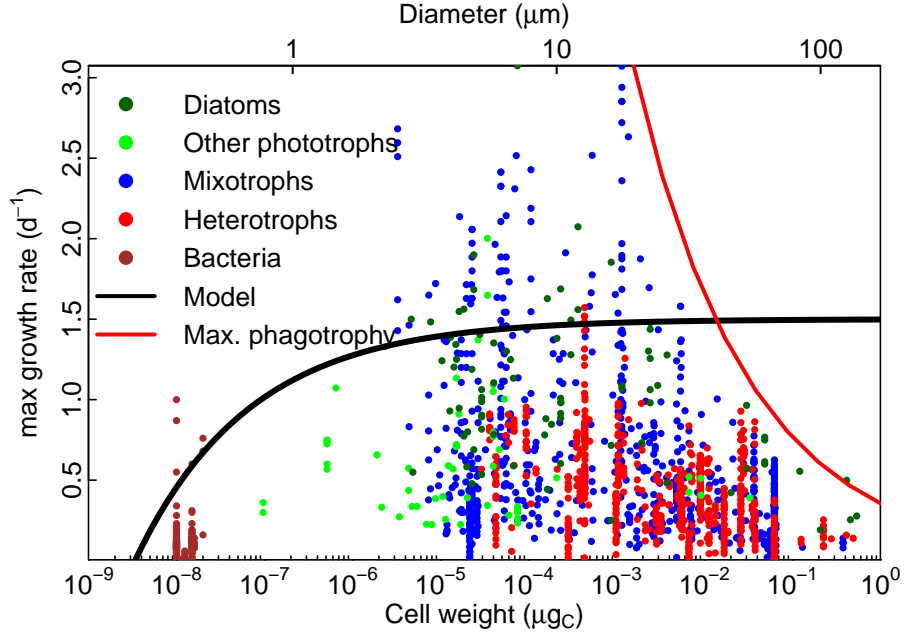
524 phy is not relevant for the smallest cells because they have no suitable food, so including  
525 the effect of cell membrane for phagotrophy is irrelevant.

### 536 **3.2 Passive losses across the membrane**

537 It is well recognized that cells leak smaller molecules across their membrane, however, the  
538 exact processes behind this loss are not well understood. Bjørnsen (1988) distinguished  
539 between losses as “income taxes” and “property taxes”. Income taxes are those losses  
540 incurred during uptake. These losses are represented as a less than 100% efficiency of the  
541 uptakes. Property taxes are those losses that occur regards of the uptakes, which we here  
542 consider as passive exudation. The passive exudation can be assumed to scale with the  
543 surface area (Kiørboe, 2013) and, assuming a negligible external concentration, becomes:

$$j_{\text{passive}} = c_{\text{passive}} r^{-1}, \quad (16)$$

544 where  $c_{\text{passive}} = 3P$  where  $P$  is the permeability of a phytoplankton membrane. Values of  
545 the membrane permeability varies wildly: Braakman et al. (2017) argues for a very high  
546 membrane permeability in excess of  $\approx 10^6$   $\mu\text{m}/\text{day}$ . This high permeability would imply  
547 that the cell spends significant amounts of energy continuously re-uptaking lost nutrients.  
548 Bjørnsen (1988) considers that  $P \approx 1$   $\mu\text{m}/\text{day}$ . Even this value is very high. However,  
549 considering than only about 10% of the compounds are of sufficiently low molecular  
550 weight to escape through the membrane,  $c_{\text{passive}}$  should be reduced by a factor 10. Further,  
551 the smallest cells, which are those which are most affected by passive exudation losses, are  
552 bacteria with a different cell membrane than the phytoplankton considered by Bjørnsen  
553 (1988). We therefore propose to further reduce the permeability further and use  $c_{\text{passive}} \approx$



526

527 Figure 5: Maximum growth rates of plankton. Phototrophs from Edwards et al.  
 528 (2015), mixotrophs (nano- and dinoflagellates and ciliates from Kiørboe and Hirst  
 529 (2014) and “bacterivores” from Rose and Caron (2007)), heterotrophs (“herbi-  
 530 vores” from Rose and Caron (2007)) and of bacteria (Kirchman, 2010). Rates  
 531 are converted to 10 degrees with a  $Q_{10} = 1.5$  for phototrophs and  $Q_{10} = 2.8$  for  
 532 mixo- and heterotrophs. The solid line is Eq. 17 with  $\alpha_{\max} = 1.5 \text{ day}^{-1}$ . The red  
 533 line is the maximum assimilated phagotrophic uptake  $\epsilon_F C_F / r$ . The diameter-axis  
 534 on the top of the panel is not accurate for diatoms because of their vacuole which  
 535 gives them a smaller density than other cells.

554 0.03  $\mu\text{m}/\text{day}$ .

### 555 **3.3 Biosynthesis and basal metabolism**

556 The maximum rate of biosynthesis is limited by the cell's investment in synthesis ma-  
557 chinery, i.e., ribosomes. If we consider the number of ribosomes to be proportional to the  
558 functional cell mass then the synthesis rate, the biomass synthesized per time and per cell  
559 mass, becomes independent of functional cell mass, i.e.,  $\propto 1 - \nu$ :

$$j_{\max} = \alpha_{\max}(1 - \nu). \quad (17)$$

560 We have no first-principle arguments to set the level of maximum synthesis rate  $\alpha_{\max}$  (but  
561 it may be possible to develop an argument based on the size and capacity of a ribosome).  
562 A more detailed argument that dynamically predicts the maximum synthesis rate as a  
563 trade-off between investment in ribosomes and chloroplast has been developed (Shuter,  
564 1979; Serra-Pompei et al., 2019, e.g. ), however, even then the crucial parameters are not  
565 constrained by first principles arguments constrained. The available data show a large  
566 scatter with maximum synthesis rates varying between almost zero and  $3 \text{ day}^{-1}$  (Fig. 5).  
567 The data also indicates that maximum synthesis rates are lower for small and large cells  
568 than for intermediate-sized cells. The reduction in max synthesis rate of large cells can be  
569 explained by the limitation due to phagotrophic assimilation (Eq. 15) as larger cells are  
570 purely phagotrophic. We have, rather arbitrarily, chosen a value of  $\alpha_{\max} = 1.5 \text{ day}^{-1}$ .  
571 This value does not represent the upper limit and it will therefore somewhat limit the  
572 community's ability to create a strong bloom in a seasonal environment.

573 The division rate is further limited by the basal metabolism. The basal metabolism

574 supports the functions needed to keep the cell alive but not the respiration associated  
575 with resource assimilation and biosynthesis. In this simple model we do not distinguish  
576 between basal metabolism and other respiration (but see Chakraborty et al. (2017, 2020))  
577 and consider simply that all respiratory costs are a fraction of cell mass, and therefore that  
578  $j_R$  is constant. For simplicity we write it proportional to the maximum synthesis capacity:

$$j_R = \alpha_R \alpha_{\max}. \quad (18)$$

579 with  $\alpha_R \approx 0.1$ .

### 580 **3.4 Temperature effects**

581 The temperature response of a cell is commonly modelled by multiplying the maximum  
582 growth rate with a  $Q_{10}$  or Arrhenius factor. For heterotrophic plankton a  $Q_{10} \approx 2$  well  
583 represents the temperature response of cell metabolism, whereas a lower factor is used  
584 for phytoplankton. It is therefore common for models to use different  $Q_{10}$  factors for  
585 phyto- and zooplankton (e.g. Archibald et al., 2022). However, the temperature response  
586 of phototrophic plankton is more complex, and recent experimental work has shown a  
587 strong dependence on the resource environment (Schaum et al., 2017; Thomas et al., 2017;  
588 Marañón et al., 2018). Shuter (1979) showed how temperature effects in phytoplankton  
589 should emerge as a result of the  $Q_{10}$ 's of each metabolic or resource uptake process in  
590 the cell. Serra-Pompei et al. (2019) took this idea further and applied it to mixotrophic  
591 plankton. They found that temperature responses of the cell's growth rate varied between  
592 almost no temperature response in environments with low nutrients and high light, to  
593 around  $Q_{10} = 2$  in high food environments.



594 Temperature effects are introduced by multiplying rates with a  $Q_{10}$  function:  $Q_{10}^{(T-T_{\text{ref}})/10}$   
 595 which gives the fractional increase in the rate when the temperature is increased 10 de-  
 596 grees from a reference temperature of  $T_{\text{ref}} = 10$  degrees. The  $Q_{10}$  factors are (Serra-  
 597 Pompei et al., 2019):  $Q_{10} = 1.5$  for diffusive uptakes, no temperature correction for  
 598 light capture and a standard “metabolic” correction of  $Q_{10} = 2$  on respiration, maximum  
 599 phagotrophy, and maximum synthesis capacity. For feeding one could follow Eq. 14 and  
 600 use the temperature scaling of viscosity ( $Q_{10} \approx 1.5$ ). However, prey also have escape  
 601 maneuvers which will become equally faster so we assume that the two effect cancel one  
 602 another and use  $Q_{10} = 1$  for feeding.

Table 1: Parameters for the cell-level processes (Section 3) and the size-based model (Section 4.5). “d” is the unit of time (days).

Parameter	Value	Reference
<i>Cell size and density</i>		
Carbon density	$\rho = 0.4 \cdot 10^{-6} \mu\text{g}_C/\mu\text{m}^3$	(a)
Membrane thickness	$\delta = 50 \text{ nm}$	(f)
C:N mass ratio	$\rho_{C:N} = 5.68 \text{ g}_C/\text{g}_N$	
<i>Cell rate parameters</i>		
Diffusive aff. coef.	$\alpha_D = 0.972 \text{ l} \cdot \mu\text{m}^2/\text{d}/\mu\text{g}_C$	Fig. 2, Box 1
Diffusive aff. cross-over	$r_D^* = 0.4 \mu\text{m}$	Fig. 2, Box 1
Light aff. coef.	$\alpha_L = 0.3 (\text{d} \cdot \mu\text{mol m}^{-2} \text{s}^{-1})^{-1} \mu\text{m}$	Fig. 3, Box 2
Light aff. cross-over	$r_L^* = 7.5 \mu\text{m}$	Fig. 3, Box 2
Light uptake eff.	$\epsilon_L = 0.8$	
Clearance rate	$a_F = 0.018 \text{ l}/\text{d}/\mu\text{g}_C$	Fig. 4, Box 3
Max. phagotrophy coef.	$c_F = 30 \mu\text{m}/\text{d}$	Fig. 5
Assimilation efficiency	$\epsilon_F = 0.8$	
Passive loss coef.	$c_{\text{passive}} = 0.03$	Sec. 3.2
Max. synthesis coef.	$\alpha_{\text{max}} = 1.5 \text{ d}^{-1}$	Fig. 5
Basal metabolism coef.	$\alpha_R = 0.1$	
<i>Prey encounter</i>		
Predator-prey mass ratio	$\beta = 500$	(b)
Predator-prey width	$\sigma = 1.3$	(b)
<i>Community model parameters</i>		
DOC remin. of feeding	$\gamma_F = 0.1$	(e)
DOC remin. of lysis	$\gamma_v = 0.5$	(e)
Lysis mortality coef.	$\mu_{v0} = 0.004/\log(m^+/m^-)$	(c)
Size of HTL mort.	$m_{\text{htl}} = 8.9 \cdot 10^{-5} \mu\text{g}_C$	(d)
HTL mortality coef.	$\mu_{\text{htl0}} = 0.1 \text{ d}^{-1}$	Sec. 5.2
<i>Chemostat parameters</i>		
Mixing rate	$d = 0.0001 \dots 1 \text{ d}^{-1}$	Sec. 4.5
Deep nutrient conc.	$N_0 = 50 \mu\text{g}_N/\text{l}$	Sec. 4.5
Productive layer	$M = 20 \text{ m}$	

(a) Rough average of data on protists excl. protists in Menden-Deuer and Lessard (2000). (b) Rough average from Fig. 6.6 in Kiørboe (2008). (c) Inversely proportional to the log width of the computational cells,  $\log(m^+/m^-)$ . Adjusted to be smaller than the predation mortality. (d) A factor  $\beta^{1.5}$  smaller than the largest size in the simulations ( $1 \mu\text{g}_C$ ). (e) Tuned to give reasonable ranges in Fig. 13. (f) Reduced from the value of 70-80 nm from Raven (1987).

## 636 **4 Size structure of the plankton community**

637 The previous section was devoted to describe the processes of the single cell as a function  
638 of its size and tie this processes down to first principles as far as possible. This section is  
639 devoted to analyse the structure of the plankton community and how it emerges from the  
640 first principles constraints on the cell processes. What actually defines the “size structure”  
641 of a community? It is how the community varies with cell size: which types of cell  
642 dominate a given size group and how big is their biomass.

643 The section is split into two parts: first we analyse the cell’s resource uptake and  
644 metabolism as a function of size to identify the maximum and minimum size of cells,  
645 the competitive abilities of different sized cells, and their dominant trophic strategies. In  
646 the second part we scale from the cell-level processes up to the biomass distribution of  
647 the plankton community, both with a simple theoretic argument and with a full dynamic  
648 model.

### 649 **4.1 Smallest and largest cells**

650 Raven (1994) argued that the cell membrane sets a lower limit of the size of the smallest  
651 cell. The absolute smallest size is when the cell membrane uses the entire mass, i.e., when  
652 the cell membrane fraction  $\nu = 1$  (Eq. 2):

$$r_{\min} = \delta \approx 50 \text{ nm} \approx 0.03 \text{ } \mu\text{g}. \quad (19)$$

653 This is an extreme lower limit for a cell with plenty of resources and no losses. Consid-  
654 ering that losses to respiration and passive losses (Eq. 16) can not exceed the maximum

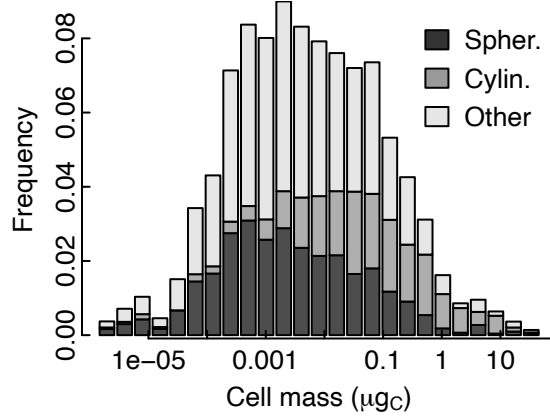


Figure 6: Distribution of cell shapes of phytoplankton as a function of cell mass. Data from Ryabov et al. (2021)

655 synthesis rate (Eq. 17):  $j_{\max} > j_R + j_{\text{passive}}$ , gives a larger minimum size of:

$$r_{\min} = \frac{c_{\text{passive}} + 3\alpha_{\max}\delta}{\alpha_{\max} - j_R} \approx 0.2 \mu\text{m} \approx 0.16 \mu\text{g}. \quad (20)$$

656 The largest unicellular plankton are heterotrophs (Andersen et al., 2016). They are  
 657 limited by two processes: the rate at which oxygen diffuses into the cell (Fenchel, 1987;  
 658 Payne et al., 2011) and the rate at which they can assimilate food through their feeding  
 659 vacuoles (red line in Fig. 5). Considering the limiting effects of food uptake, the maximum  
 660 size  $r_{\max}$  is when the maximum rate of assimilated consumption,  $\epsilon_F C_F / r$  (Eq. 15) equals  
 661 the metabolic costs  $j_R$ :

$$r_{\max} = \frac{\epsilon_F C_F}{j_R} \approx 160 \mu\text{m} \approx 10 \mu\text{g}. \quad (21)$$

662 Fenchel (Chap. 1 1987) considered that the upper size limit is imposed by the diffusion of  
 663 oxygen into the heterotrophic cell. He finds that the largest radius where  $\text{O}_2$  diffusion can

664 satisfy the metabolic demand is:

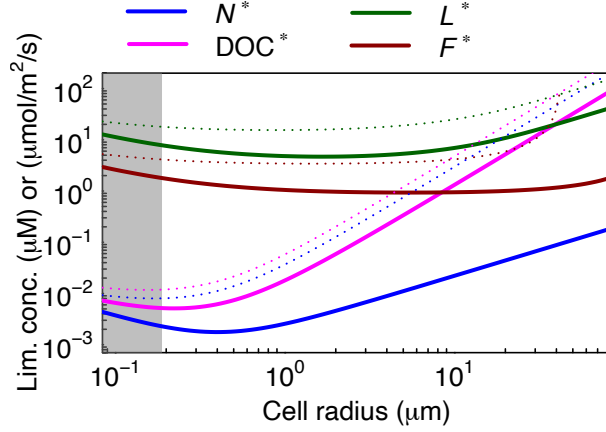
$$r_{\max} = \sqrt{\frac{6X_{O_2}D}{j_R\rho S_{O_2}}} \approx 800\mu\text{m} \approx 800\mu\text{g}, \quad (22)$$

665 where  $X_{O_2}$  is the external oxygen concentration,  $D$  the diffusivity of oxygen, and  $S_{O_2}$  the  
666 oxygen:carbon mass ratio (Payne et al. (2011) did a similar evaluation and found an upper  
667 limit around 1 mm under present day oxygen concentrations). The upper limit imposed by  
668 oxygen is rather large compared to the upper limit imposed by assimilation (Eq. 21) and  
669 it is tempting to disregard oxygen as a constraint on maximum cell size. However, it is  
670 instructive to look also on the size distribution on cell shape, as analysed by Ryabov et al.  
671 (2021) (Fig. 6). The smallest cells are spherical, which is the shape that minimizes the  
672 cell membrane per mass. Cells larger than about  $0.05 \mu\text{g}_C$  are dominated by cylindrical  
673 cells. Being cylindrical minimizes the distance of oxygen diffusion from the cell surface  
674 to the center. That larger cells are cylindrical therefore indicates the importance of oxygen  
675 for the upper limit of cell size. It is possible that not only the diffusion limits the cell size,  
676 but also the permeability of the cell wall; a complication that is ignored in the argument  
677 by Fenchel (1987).

678 To overcome the upper limitation of size, organisms will have to become multicellular.  
679 The smallest adult copepods are on the order of  $0.01 \mu\text{g}$ , which corresponds to the size  
680 where the feeding vacuole becomes limiting for growth (Fig. 5).

## 681 **4.2 Limiting resources, $R^*$**

689 The growth of plankton is limited by their ability to acquire and assimilate resources of  
690 nutrients, DOC, food from predation, and light. As dissolved resources are subject to



682

683 Figure 7: Minimum resource concentrations for survival ( $R^*$ ). Shown with mor-  
 684 tality  $\mu = 0$  (solid lines) and  $\mu = 0.4 \text{ day}^{-1}$  (dotted lines). Carbon sources (light  
 685 and DOC) assume plenty of nutrients;  $N^*$  assume plenty of carbon;  $F^*$  is for  
 686 a pure phagotroph. The grey area indicates the minimum viable size of a cell  
 687 (Eq. 20).

691 competition by all cells, nutrients and DOC are exhausted to the lowest level that the most  
 692 competitive groups can just survive on. This level is commonly referred to as the “ $R^*$ ”  
 693 value, *sensu* Tilman (1982). Ward et al. (2014) calculated the limiting nutrient resource  
 694  $N^*$  as a function of cell size and found that limiting resource increases with cell size –  
 695 confirming the classic result that the smallest cells are the most competitive for nutrients  
 696 (Munk and Riley, 1952). Here we extend the  $R^*$  concept to the concentration of DOC,  
 697 food, and light. Food is different than the dissolved resources because not all size groups  
 698 compete for all sizes of food due to size-based selection. Nevertheless,  $F^*$  indicates the  
 699 minimum level of biomass of their prey. Finally, we can calculate the minimum level  
 700 of light  $L^*$  where purely phototrophic plankton can survive. Plankton does not compete  
 701 for light (except in extreme cases of biomass as seen in some fresh water environments;  
 702 Klausmeier and Litchman (2001)), but the  $L^*$  indicates the minimum light level – and

Table 2: Limiting resource levels

$$\begin{aligned}
 N^*(r) &= \frac{(c_{\text{passive}} + \mu r)(r^2 + r_D^2)}{r\alpha_D} \\
 X_{\text{DOC}}^*(r) &= \frac{(c_{\text{passive}} + (j_R + \mu)r)(r^2 + r_D^2)}{r\alpha_D} \\
 L^*(r) &= \frac{1}{1 - e^{-r/r_L}} \frac{c_{\text{passive}} + (j_R + \mu)r}{\alpha_L} \\
 F^*(r) &= \frac{c_F(c_{\text{passive}} + (j_R + \mu)r)}{c_F\epsilon_F - \alpha_F r(c_{\text{passive}} + (j_R + \mu)r -)}
 \end{aligned}$$

thus the maximum depth – where photosynthesis alone can support plankton growth.

We can find the limiting resources by calculating the resource level that just balances losses to exudation, respiration, and mortality, e.g., for light:  $j_L(L^*) = j_{\text{passive}} + j_R + \mu$ , where  $\mu$  is mortality losses (see Table 2).  $N^*$  is calculated from the assumption that carbon is abundant so we can ignore respiration. Similarly, the calculation of  $L^*$  and  $DOC^*$  assumes abundant nutrients but no alternative carbon source (from DOC, light or food).  $F^*$  also assumes no other carbon source (no phototrophy or DOC).

The actual values of the limiting resources cannot be compared directly between one another because they are in different units, however, the interesting aspect is also mainly which size can survive on the lowest resource levels. All limiting resources have a minimum at a specific size (Fig. 7). The minimum emerges as the result of two opposing effects: the passive losses which decreases with cell size (due to decreasing surface to volume ratio; Eq. 16), and the affinity which also decreases (or is constant) with size. The most pronounced minimum is for diffusive uptake of dissolved carbon and nutrients. In contrast to the results by Ward et al. (2014) the  $N^*$  for the very smallest sizes again increases, however, this increase is likely not relevant as the smallest cell are limited by

Table 3: Approximate expressions for the sizes where strategies transition from osmotrophy to phototrophy, from light- to nutrient-limited phototrophy, and from phototrophy to mixotrophy. The expressions are derived by using Eq. 4 for diffusive affinity and by ignoring the correction term in the parentheses of Eq. 13.

Osmo. to phototrophy	$\frac{\alpha_D}{\alpha_L} \frac{X_{\text{DOC}}}{L} + 3\delta$
Light to nutrient limit.	$\frac{\alpha_D}{\alpha_L} \frac{\rho_{\text{C:N}} N}{L} + 3\delta$
photo. to mixotrophy	$\frac{\alpha_D}{\alpha_F} \frac{\rho_{\text{C:N}} N}{F}$

the cell membrane (the grey area in Fig. 7). Regarding light, a very wide range of sizes can survive on the lowest light levels. Phototrophy therefore selects weakly for cell size, and the selection only enters because the cells also need nutrients, which select for small cells. The minimal food requirement  $F^*$  is almost independent of size and is around 1  $\mu\text{g}_\text{C}/\text{l}$ . Environments with less food therefore cannot support a longer food chain with purely heterotrophic plankton.

### 4.3 Trophic strategies

The other dimension of community structure is the trophic strategies, i.e., how cells acquire resources: by osmotrophy (diffusive uptake of DOC), phototrophy, or by phagotrophy. The dominant strategy is determined by which of the three fluxes  $j_{\text{DOC}}$ ,  $j_{\text{L}}$  and  $J_{\text{F}}$  is the largest (Andersen et al., 2016). Fig. 8a shows the fluxes of DOC, carbon from phototrophy, nutrients, and food in an environment specified by concentrations of DOC, N and food (specified by the level of the size spectrum  $\kappa$ ), and by light. Typically, very small cells are osmotrophs, somewhat larger cells are light-limited phototrophs, medium-sized cells are nutrient limited and larger cells are mixotrophs or heterotrophs. However, the transitions between the dominant strategies occur at different sizes: less nutrients or



748 more available food favours mixotrophic and heterotrophic strategies, while more DOC  
749 favours osmotrophy. Andersen et al. (2016) provided analytical expressions for the sizes  
750 where the dominant strategies switch from one strategy to the other. This was possible  
751 because they used simple power-law relationships for the affinities. Here, however, the  
752 relationships are more complex and exact analytical expressions are not possible. How-  
753 ever, approximations can be made, which show how the transition sizes depend upon the  
754 resource concentrations and the affinities (Table 3).

755     The perspective of trophic strategy being set by the most favourable strategy adds  
756 more detail to the argument developed above about the structure being determined by the  
757 most competitive size. Generally, the two perspective agree: small cells are dominated  
758 by osmotrophs because they are the most competitive for dissolved resources. The per-  
759 spective of the dominant strategy adds more detail, though, by showing how the smallest  
760 phototrophs are light limited while larger phototrophs are nutrient limited, and showing  
761 the size ranges of mixotrophs and pure heterotrophs.

#### Box 4: Theoretical derivation of the size spectrum

If the cell is not limited by uptake over the feeding vacuole (i.e., that  $a_F F \ll c_F r$  in Eq. 15) then the effective encounter rate is  $j_F = \epsilon_F a_F F$  (Eq. 15). The encountered food  $F$  is found by inserting the ansatz  $b(m) = \kappa m^{\lambda-1}$  in Eq. 31 to give  $F(m) = \kappa m^\lambda \alpha$ , where  $\alpha = \sqrt{2\pi\sigma} \beta^\lambda e^{\lambda^2 \sigma^2/2}$  is a factor that depends on the parameters of the size preference function (Eq. 30). Following Andersen and Beyer (2006) we now assume that the encounter rate of food  $j_F$  is proportional to the metabolic needs  $j_R$  and independent of size. Then we can equate encountered food with metabolic needs:

$$\epsilon_F a_F \kappa m^\lambda \alpha \propto j_R. \quad (23)$$

This relation is only true if the dependency on  $m$  disappears, i.e., if  $\lambda = 0$ . When  $\lambda = 0$  the abundance distribution is  $b(m) \propto m^{-1}$ , corresponding to the Sheldon spectrum  $\mathcal{B}$  (Eq. 29) being constant (independent of cell size).

The level of the spectrum,  $\kappa$ , can be estimated by assuming that the entire flux of new nutrients  $dN_0$  into the photic zone is taken up and used. This assumption is reasonable as nutrient concentrations in the surface are much less than deep nutrient concentrations in the productive season. The flux of potential new production is  $dN_0$  which can support a new primary production of  $dN_0 \rho_{C:N}$  (gC/day/liter). There are three sources of losses: higher trophic level predation, diffusion losses, and respiration. The losses to higher trophic levels are found by integrating over the range where the higher trophic level mortality acts, i.e., a factor  $\beta$ :

$$J_{\text{HTLloss}} = \int_{m_{\text{htl}}}^{\beta m_{\text{htl}}} \kappa m^{-1} \mu_{\text{htl}} dm \quad (24)$$

$$= \kappa \mu_{\text{htl}} \ln(\beta). \quad (25)$$

Diffusion and respiration losses are found by integrating over the entire size range:

$$J_{\text{loss}} = \int_{m_{\text{min}}}^{m_{\text{max}}} \kappa m^{-1} (d + j_R) dm = \kappa (d + j_R) \omega, \quad (26)$$

where the ranges of the integration are given by Eqs. 20 and 21, and  $\omega = \ln(m_{\max}/m_{\min}) \approx 25$ . Equating the new production with losses, and accounting for a fraction  $\epsilon_{\text{htl}}$  of the higher trophic level losses being remineralized in the photic zone, gives:

$$\kappa = \frac{dN_0\rho_{C:N}}{(1 - \epsilon_{\text{htl}})\mu_{\text{htl}} \ln \beta + (d + j_R)\omega}, \quad (27)$$

794

795 **Box 5: definition of the Sheldon size spectrum**

796 The structure of the plankton community is represented by the biomasses in the  
 797 size groups  $B_i$ . This representation has the disadvantage that the level of the  
 798 biomasses depend on the size-range of each group: broader (fewer) size-groups  
 799 leads to higher average biomass level and *vice versa*. To avoid this depen-  
 800 dency size distributions are often shown as “normalized size spectra” (Sprules  
 801 and Barth, 2016), by dividing the biomass with the size range of the group:  
 802  $b(m_i) = B_i/(m_i^+ - m_i^-)$ , where  $m_i^+$  and  $m_i^-$  are the upper and lower sizes  
 803 in the size group. If we assume a scaling biomass spectrum,  $b(m) = \kappa m^{\lambda-1}$   
 804 then the relation between the normalized biomass spectrum and the binned size  
 815 groups is:

$$805 \quad B_i = \int_{m_i^-}^{m_i^+} b(m) dm = \kappa \log(m_i^+/m_i^-) \quad (28)$$

807 if  $\lambda = 0$ . If size groups are evenly distributed on a log scale then  $m_i^+/m_i^-$  is  
 808 constant (independent of mass) and the biomasses in each groups are roughly the  
 809 same. To avoid that results depends on the binning of the size groups we here  
 810 define the “Sheldon” spectrum as:

$$811 \quad \mathcal{B}(m) = B_i / \log(m_i^+/m_i^-). \quad (29)$$

813

814

816

817 **4.4 Theoretical size spectrum**

818 The size spectrum was first introduced by Sheldon and Parsons (1967) who plotted the  
 819 biomass as a histogram in log-spaced size groups and showed that the biomass was

roughly independent of cell size. Since that pioneering work the regularity of the log-histogram spectrum has been demonstrated over and over again, as reviewed by Sprules and Barth (2016). The histogram representation is, however, inconvenient, because the height of the histogram depends on the width of the size bin that are used. This led Platt and Denman (1977) to introduce the “normalized size spectrum” as the biomass distribution as a function of cell size  $b(m)$ . Being a distribution means that the spectrum has dimensions of biomass per cell mass, and that the integral of the spectrum is the total biomass. It is convenient to introduce the “Sheldon spectrum” as  $b(m)m$  because it has the same property as the log-binned histogram that it is approximately flat (Box 4).

The flat Sheldon spectrum is commonly understood as emerging from predator-prey interactions. First, Sheldon et al. (1972) showed how the biomass in successive trophic levels scaled as  $\epsilon_T \beta^{0.25} \approx 0.9$ , where  $\epsilon_T \approx 0.2$  is the trophic efficiency and  $\beta \approx$  is the predator prey mass ratio. This results was later re-derived as part of the metabolic theory of ecology (Brown et al., 2004), however, only by introducing an extra assumption about energy equivalence. The result relies on the trophic efficiency, which is a quantity that is hard to estimate, and which eventually is an emergent property of the community structure (Borgmann, 1987). An alternative argument by Andersen and Beyer (2006) derived the size spectrum purely based on individual-level properties. As all of these arguments only rely upon predator-prey interactions, it is not clear how well they apply among the lower trophic levels of the ocean where many cells mainly subsist on photosynthesis and recycled production from dissolved organic matter and less on predation on smaller particles. Poulin and Franks (2010) refined the argument by considering phytoplankton and zooplankton spectra separately to show a flat phytoplankton spectrum and a declining zooplankton spectrum. Here we will explain the scaling of the community size spectrum

844 only from considerations of predator-prey interactions by an extension of the Andersen  
 845 and Beyer (2006) argument, and later show that the predictions fit surprisingly well with  
 846 dynamical simulations.

847 Predator-prey interactions are described by bigger cells predating on smaller cells  
 848 (Hansen et al., 1997). The size preference for predation can be described by a log-normal  
 849 size selection function:

$$\phi(m, m_{\text{prey}}) = \exp \left[ -\frac{\ln^2(m/(\beta m_{\text{prey}}))}{2\sigma^2} \right], \quad (30)$$

850 where  $m_{\text{prey}}$  is prey size,  $\beta$  the preferred predator:prey mass ratio and  $\sigma$  the width of the  
 851 preference function. The available food is found by integrating across all size groups:

$$F(m) = \int \phi(m, m_{\text{prey}}) b(m_{\text{prey}}) dm_{\text{prey}}, \quad (31)$$

852 where  $b(m)$  is the biomass size spectrum. From this description we can derive the size  
 853 spectrum as (Box 5):

$$b(m) = \kappa m^{-1}, \quad \text{with} \quad \kappa = \frac{dN_0\rho_{\text{C:N}}}{(1 - \epsilon_{\text{htl}})\mu_{\text{htl}} \ln \beta + (d + j_{\text{R}})\omega}. \quad (32)$$

854 The spectrum scales with mass as  $m^{-1}$ , which means that the Sheldon spectrum  $\propto b(m)m$   
 855 is constant. The height of the spectrum (the coefficient  $\kappa$ ) is a novel result. The height  
 856 depends on the mixing rate  $d$ , the concentration of nutrients being mixed up from the deep  
 857  $N_0$ , the mortality imposed by higher trophic levels  $\mu_{\text{htl}}$ , and the length of the size spectrum  
 858  $\omega = \ln(m_{\text{max}}/m_{\text{min}})$ . The main controlling parameter is the mixing rate. The height of  
 859 the spectrum increases with mixing but saturates at high mixing rates ( $d \gg \mu_{\text{htl}} \ln \beta / \omega$ ).

863 Table 4: Processes and equations to calculate the division rate  $g$  of a cell (note that  
 864 the population growth rate also requires the subtraction of losses). All rates are in  
 865 units of  $g_C/g_C$  per time.

<i>Encounter and synthesis:</i>		
Available carbon	$j_{C.net} = j_{DOC} + j_L + j_F - j_R - j_{passive}$	(M1)
Available nutrients	$j_{N.net} = j_N + j_F - j_{passive}$	(M2)
Leibig's law	$j_{net} = \min\{j_{C.net}, j_{N.net}\}$	(M3).
Synthesis	$g = j_{max} \frac{j_{net}}{j_{net} + j_{max}}$	(M4)
<i>Down-regulation of uptakes</i>		
Feeding	$\tilde{j}_F = \max\{0, j_F - (j_{net} - g)\}$	(M5)
Photoharvesting	$\tilde{j}_L = j_L - \max\{0, \min\{(j_{C.net} - (j_F - \tilde{j}_F) - g), j_L\}\}$	(M6)
DOC uptake	$\tilde{j}_{DOC} = g - \tilde{j}_F - \tilde{j}_L$	(M7)
<i>Mortalities:</i>		
875 Predation:	$\mu_p(m_j) = \sum_i \frac{\tilde{j}_{F,i}}{\epsilon_F} \frac{\Phi_{ij}}{F_i} B_i$	(M7)
Viral lysis:	$\mu_{v,i} = \mu_{v0} B_i$	(M8)
Higher trophic levels:	$\mu_{htl}(m) = \begin{cases} \mu_{htl0} \phi(m, m) & \text{for } m < m_{htl} \\ \mu_{htl0} & \text{for } m \geq m_{htl} \end{cases}$	(M9)
866 All fluxes are calculated according to the relations in Section 3. Available food is $F_i = \sum_j \Phi_{ij} B_j$ 867 where the effective preference between size groups $\Phi_{ij}$ is found by integration across the width 868 of the size groups (Appendix A). The tildes above the uptakes of light and food indicates down- 869 regulation in eqs. (M5-6). (M1-2): Uptakes are given by Eq. 3 combined with affinities for nutri- 870 ents and DOC (Eq. 5), light (Eq. 13) and food (Eqs. 15 and 31). (M3): a standard functional type 871 II response aka. ‘‘Monod’’ function. (M7): The predation mortality exerted by unicellular plankton 872 can be calculated as the ratio between the amount of food eaten by all predators from size group 873 $j$ and biomass at size group $j$ . The amount eaten is: $E_j = \sum_i (\tilde{j}_{F,i}/\epsilon_F) B_i \phi(m_i, m_j) B_j / F_i$ . 874 Moving $B_j$ outside the sum to calculate predation mortality as $\mu_{p,j} = E_j / B_j$ gives M7.		

860 At very high mixing rates the production will be limited by the synthesis capacity of the  
 861 cell, which is not accounted for here, however, that is probably a rare occurrence in nature.

## 862 4.5 Dynamic size-based model

876 Further insight into the size structure requires numerical simulations. Here we simulate  
 877 the entire unicellular plankton community by embedding the model of cell resource uptake  
 878 and metabolism in a simple ecosystem model. Cells are divided into size group with each

879 group  $i$  representing the biomass  $B_i$  within a range of cells with the geometric mean mass  
880  $m_i$ . For simplicity we have assumed that cells have constant C:N mass ratio  $\rho_{\text{C:N}} = 5.68$ ,  
881 but the model can be extended to dynamic stoichiometry (Ho et al., 2020; Ward et al.,  
882 2018). The rate of change (the growth rate) of biomass in a size group is:

$$\frac{1}{B_i} \frac{dB_i}{dt} = g(m_i) - \mu(m_i), \quad (33)$$

883 where  $g(m)$  is the division rate and  $\mu(m)$  is the total mortality. The division rate is deter-  
884 mined by resource encounter and synthesis (Table 4, eq. M4).

885 Mortality has three origins: predation by unicellular plankton  $\mu_p$  through the pro-  
886 cess of big cells eating smaller cells (M7), viral lysis  $\mu_2$  (M8), and predation by higher  
887 trophic levels  $\mu_{\text{htl}}$  (M9). Viral mortality is modelled by assuming that viral mortality is  
888 proportional to biomass. Mortality by higher trophic levels acts on the largest size groups.  
889 We use a selection function consisting of combination of the logarithmic size selection  
890 function in Eq. 30 and a constant level.

Nutrients and DOC are updated with the uptakes and losses from the cell-level pro-  
cesses:

$$\begin{aligned} \frac{dN}{dt} = \frac{1}{\rho_{\text{C:N}}} \sum_i \left( \underbrace{-j_{\text{N},i}}_{\text{Uptake}} + \underbrace{j_{\text{passive},i}}_{\text{Exudation}} + \underbrace{\max\{0, j_{\text{N},\text{net},i} - g_i\}}_{\text{Surplus}} \right. \\ \left. + \underbrace{\frac{1 - \epsilon_F}{\epsilon_F} \tilde{j}_{\text{F},i}}_{\text{Feeding losses}} + \underbrace{\mu_{\text{v}0} B_i}_{\text{Lysis}} \right) B_i \end{aligned} \quad (34)$$

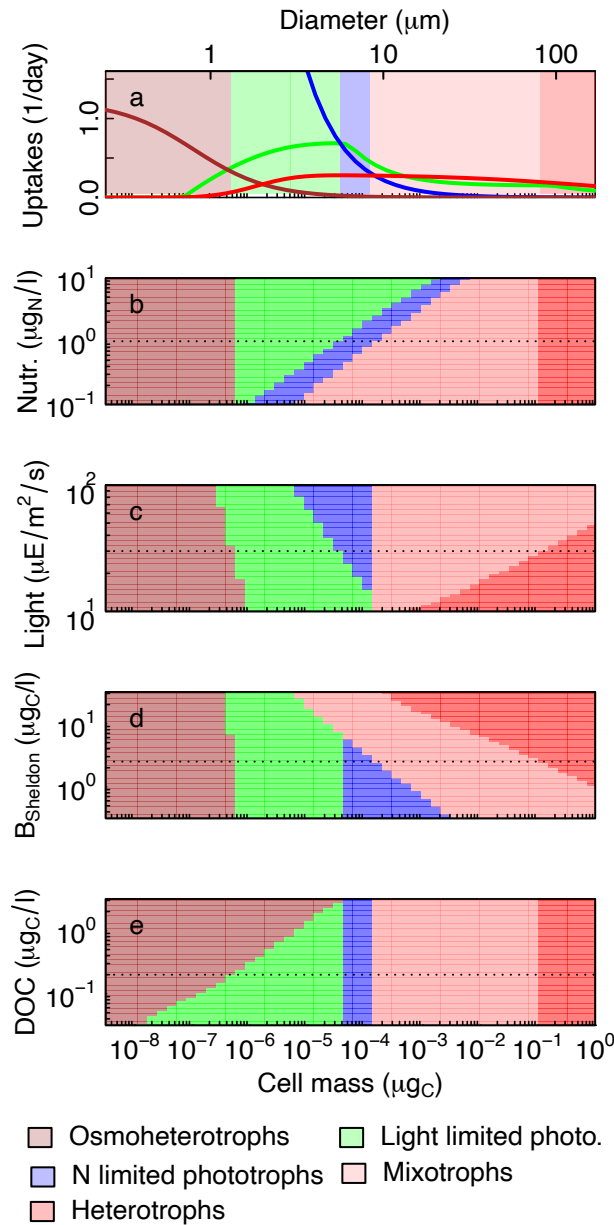


$$\frac{d\text{DOC}}{dt} = \sum_i \left( -\underbrace{\tilde{j}_{\text{DOC},i}}_{\text{Uptake}} + \underbrace{j_{\text{passive},i}}_{\text{Exudation}} + \underbrace{\frac{1 - \epsilon_L}{\epsilon_L} \tilde{j}_L}_{\text{Photoharvesting}} + \underbrace{\gamma_F \frac{1 - \epsilon_F}{\epsilon_F} j_{F,i}}_{\text{Feeding losses}} + \underbrace{\gamma_v \mu_{v0} B_i}_{\text{Lysis}} \right) B_i. \quad (35)$$

891 Generation of  $N$  happens through assimilation losses, passive exudation and remineralised viral lysis. Generation of labile DOC happens through passive exudation, assimilation losses from light harvesting and phagotrophy, and from remineralized viral lysis. 892  
893 We assume that all nutrient losses from viral lysis are made available over short time scales (Carlson, 2002), but only a fraction  $\gamma_v$  of carbon losses are labile. All the losses to 894  
895 higher trophic levels are eventually converted to particulate organic matter (which is not explicitly resolved here) so there is no remineralization of those losses. 896  
897

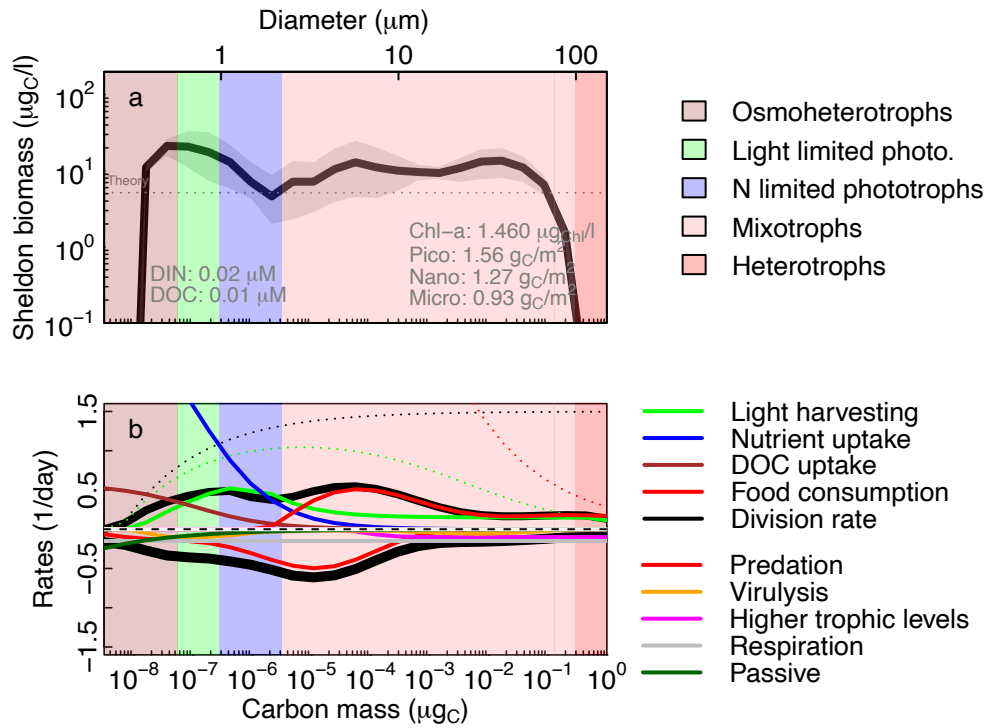
920 Five parameters control the chemostat: mixing rate  $d$ , deep nutrient concentration  $N_0$ , light  $L$ , temperature  $T$  and the mortality imposed by higher trophic levels  $\mu_{\text{ht}10}$ , however, 921  
922 only three are important. The mixing rate and the deep nutrient concentration mainly enter as a product so we can focus on only one of them – the mixing rate is commonly 923  
924 chosen. In a water column the productive layer will adjust itself to the depth where cells are co-limited by light and nutrients (Ryabov et al., 2010; Beckmann and Hense, 2007; 925  
926 Klausmeier and Litchman, 2001). The light level is therefore also of minor importance, as long as it is sufficiently high to not be limiting. We first concentrate on the mixing rate  $d$  927  
928 and take up the importance of higher trophic level mortality and temperature in the follow section. 929

930 Chemostat simulations in eutrophic situations with a high mixing rate show an ex-



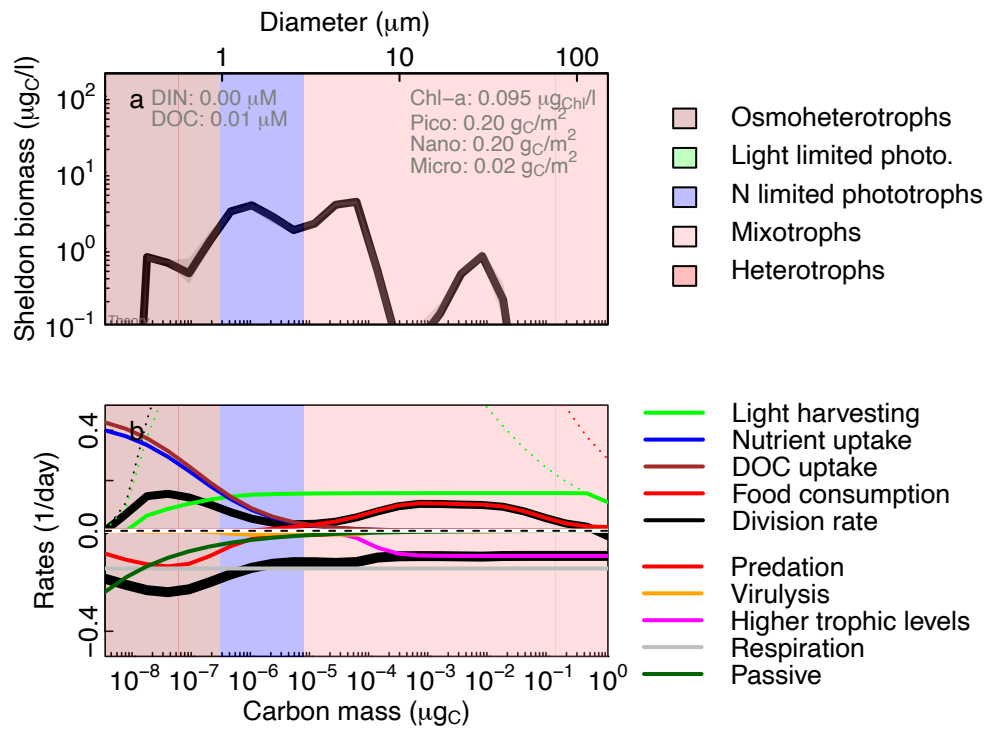
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727 Figure 8: The trophic strategies of unicellular plankton under different environ-  
 728 mental conditions. a) The gains from light (green), nutrients (blue), food (red),  
 729 and DOC (brown), and phagotrophy (red). The dominant trophic strategy is  
 730 shown by shading: heterotrophy, when surplus nutrient is leaked (red); mixotro-  
 731 phy when the carbon gain from phagotrophy and DOC surpass the potential gain  
 732 from phototrophy and the nutrient gain from feeding surpass that of diffusive nu-  
 733 trient uptake (light red); nutrient limited phototrophy when the potential gain from  
 734 phototrophy and DOC surpass the nutrient uptake (blue); light limited phototro-  
 735 phy when nutrient uptake surpass carbon uptake (green); osmotrophy when car-  
 736 bon from DOC surpass carbon from light harvesting (brown). b-e) Variations in  
 737 the dominant trophic strategy with changes in nutrients, light, biomass and DOC  
 738 around the conditions in panel (a) are indicated with a dotted line in each panel.



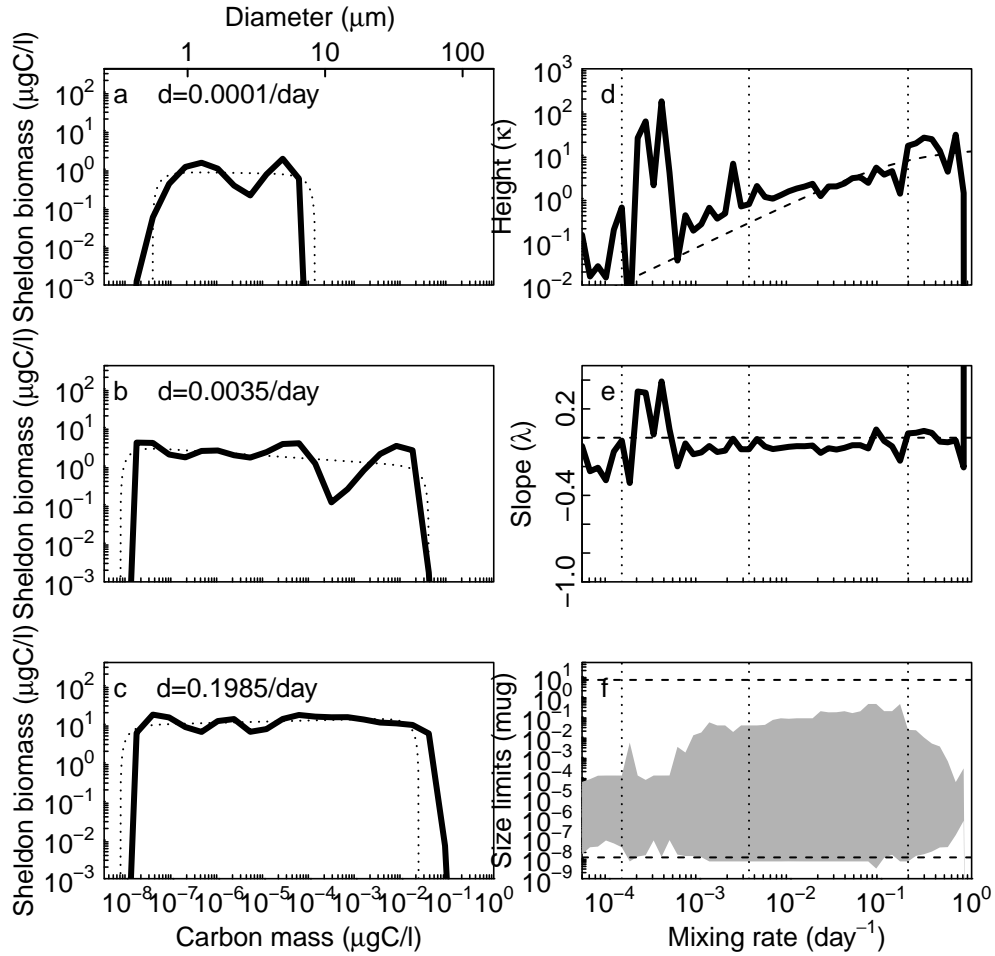
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899 Figure 9: Results of simulations under eutrophic conditions with high mixing.  
 900 a) Sheldon size spectrum (Box 5). The background colours indicate the trophic  
 901 strategy (see Fig. 8). b) Rates of uptakes and losses in biomass specific units  
 902 ( $\text{day}^{-1}$ ). The dotted lines show maximum possible uptakes or growth rates. The  
 903 thick black lines in panel b are the total division and loss rates (note that in this  
 904 case they are not equal as the simulation is not in a steady state; the variation  
 905 is indicated in panel a with the grey area around the mean). Parameters: Light  
 906  $L = 40 \mu\text{E}/\text{m}^2/\text{s}$ , mixing rate  $d = 0.1 \text{ m}/\text{day}$ , and higher trophic level mortality  
 907  $\mu_{htl} = 0.1 \text{ day}^{-1}$ .



908

909 Figure 10: Results of simulations for an oligotrophic situation with low diffusivity  
 910 of nutrients; see Fig. 9 for explanation (note different y-axis on panel b). Mixing  
 911 rate  $d = 0.001$  1/day.



912

913 Figure 11: Size spectra with varying mixing rates under high light. Panels a-c:  
 914 size spectra fitted to a power-law (Eq. 32) truncated at high and low sizes (dotted).  
 915 Panels d-f: results of the fits for height ( $\kappa$ ), exponent ( $\lambda$ ) and upper and lower size  
 916 limits. The dashed lines are the theoretical predictions of  $\kappa$  from Eq. 27, exponent  
 917  $\lambda$  from Eq. 32, and min and max sizes from Eqs. 20 and 21. The “wiggles” are  
 918 due to inaccuracies in the fit of the size spectra. The vertical dotted lines in panels  
 919 d-f show the three mixing rates used to calculate panels a-c.  $L = 100 \mu\text{E}/\text{m}^2/\text{s}$ .

931 tended flat Sheldon spectrum occupying the full size range (Fig. 9). The level of the size  
932 spectrum fits with the theoretical prediction (Eq. 27). Phototrophic cells span a wide size  
933 range and due to the high influx of nutrients they are light limited and not nutrient limited.  
934 Microplankton and partly nanoplankton have a significant influx of carbon and nutrients  
935 from phagotrophy. Only the largest cells are fully heterotrophic in the sense that they leak  
936 surplus nutrients from phagotrophic uptakes.

937 Under oligotrophic situations with a low mixing rate the spectrum is still flat, but the  
938 realized size range is smaller in both ends of the spectrum. Therefore the “height” of the  
939 spectrum ( $\kappa$ ) is also higher than predicted, because the prediction was only valid for a full  
940 range spectrum. Under oligotrophic situations the phototrophs are fully nutrient limited.  
941 It is unrealistic that very small cells are absent in oligotrophic conditions, because they  
942 are expected to dominate. This is because of the rising limiting nutrient values ( $N^*$ ) of  
943 small cells due to the limitation imposed by the cell membrane (Fig. 7).

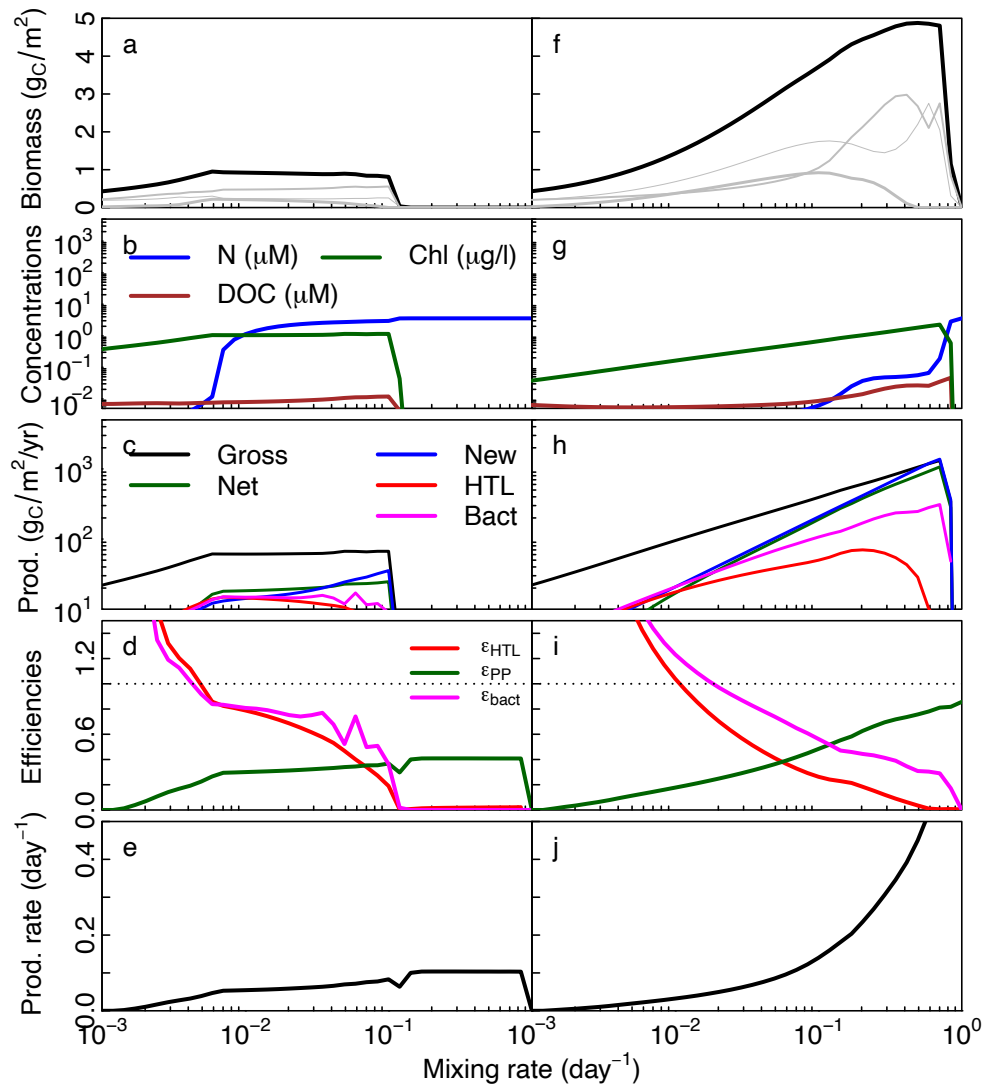
944 Simulating across mixing rates shows generally flat Sheldon size spectra (Fig. 11).  
945 The spectrum exponent (panel e) is roughly zero and the size spectrum level ( $\kappa$ ) follows  
946 the prediction from Eq. 27 (panel d). At small mixing rates small picoplankton dominates.  
947 As mixing rate increase, the upper size range extends. The extension of the upper size  
948 increases the length of the food chain. This result was demonstrated in a simple size-  
949 based by Armstrong (1994) and confirmed by alternative derivations by Poulin and Franks  
950 (2010) and Ward et al. (2014). At very high mixing rates the spectrum again becomes  
951 truncated. The truncation at high mixing rates is a result of plankton being mixed out  
952 of the productive layer faster than the maximum growth rate. Overall it is clear that the  
953 overall size spectrum exponent is unaffected by the environmental conditions, only the  
954 height and the extent of the spectrum are affected.

956 Table 5: Ecosystem biomass and functions.  $M$  is the thickness of the mixed layer,  
957 here set to 20 m.

		Value		
Function	Formula	Olig.	Eut.	
<i>Biomasses</i>				
Biomass	$B_{\text{total}} = M \sum_i B_i$	0.38	3.8	$\text{g}_C/\text{m}^2$
Chlorophyl <sup>(1)</sup>	$B_{\text{Chl}} = M \sum_i \tilde{j}_{L,i} B_i / L$	0.085	1.5	$\mu\text{g}_{\text{Chl}}/\text{l}$
<i>Production</i>				
Gross PP	$P_{\text{gross}} = M \sum_i \tilde{j}_{L,i} B_i / \epsilon_L$	20	360	$\text{g}_C/\text{m}^2/\text{yr}$
Net PP	$P_{\text{net}} = M \sum_i \max\{0, \tilde{j}_{L,i} - j_{R,i}\} B_i$	0	150	$\text{g}_C/\text{m}^2/\text{yr}$
Net bact. prod.	$P_{\text{bact}} = M \sum_i \max\{0, j_{\text{DOC},i} - j_{R,i}\} B_i$	0.12	66	$\text{g}_C/\text{m}^2/\text{yr}$
New prod.	$P_{\text{new}} = M d(N_0 - N) \rho_{C:N}$	2.1	210	$\text{g}_C/\text{m}^2/\text{yr}$
Prod. of HTLs	$P_{\text{htl}} = M \sum_i \mu_{\text{htl},i} B_i$	2.5	60	$\text{g}_C/\text{m}^2/\text{yr}$
<i>Efficiencies</i>				
Eff. of PP	$\epsilon_{\text{PP}} = P_{\text{net}} / P_{\text{gross}}$	0.0	0.43	
Eff. of bact.	$\epsilon_{\text{bact}} = P_{\text{bact}} / P_{\text{net}}$	-	0.42	
Eff. of HTL	$\epsilon_{\text{htl}} = P_{\text{htl}} / P_{\text{net}}$	-	0.43	
<sup>(1)</sup> Mass of Chl per carbon mass is approximately proportional to the down-regulated				
mass-specific light affinity $\tilde{j}_{L,i} / L$ . (Edwards et al., 2015).				

## 955 5 Ecosystem functions

966 The size distribution combined with the cell-level characteristics allows the calculation of  
967 ecosystem functions. Ecosystem functions can be divided into biomasses, production, and  
968 efficiencies. Because size-based models consider cells with multiple trophic strategies,  
969 calculating the functions are somewhat different than for ordinary functional group type  
970 of models (see Table 5). In the chemostat model the integration over the water column  
971 comes about simply by multiplying with the thickness of the productive layer that we  
972 arbitrarily set to  $M \approx 20$  m.



961

962 Figure 12: Ecosystem biomass and functions at low and high light ( $L = 10$  and  
 963  $100 \mu\text{mol m}^{-2}\text{s}^{-1}$ ). In the top row the gray lines show pico-, nano-, and mi-  
 964 croplankton. The last row shows the community production rate as the net primary  
 965 production divided by biomass.



973 Gross primary production is the total amount of carbon fixed :

$$P_{\text{gross}} = M \sum_i j_{L,i} B_i / \epsilon_L. \quad (36)$$

974 The net production is the carbon available for biomass production, i.e., the gross pri-  
975 mary production minus the exudation losses and respiration. That definition, however,  
976 only works for purely phototrophic plankton. Here, plankton are mixotrophs and some  
977 larger mixotrophs may contribute negatively to primary production because they fix neg-  
978 ligible amounts of carbon. To compensate, we consider only the groups where net fix-  
979 ation (fixation minus respiration) is positive. This procedure assumes that carbon from  
980 photosynthesis is prioritized for respiration over other carbon sources (DOC or feeding).  
981 Bacterial production is net production based on DOC uptake. It faces a similar problem  
982 as the net primary production, and again we only consider positive net contributions. New  
983 production is the amount of nutrient that diffuses up into the photic zone to fuel primary  
984 production. Finally, we can calculate the production to higher trophic levels as the losses  
985 to higher trophic level mortality.

986 Efficiencies the ratio between a production and the net primary production. They are  
987 typically in the range 0 to 1.

988 The total biomass is roughly proportional to the mixing rate  $d$  (Eq. 27) until it be-  
989 comes limited by light, around  $d = 0.02 \text{ day}^{-1}$  in low light conditions (Fig. 12 a+f). The  
990 total Chl-concentration varies between 0.01 to 1  $\mu\text{gChl/l}$  (panels b+g), which is in line  
991 with outputs of global circulation model simulations (Van Oostende et al., 2018). When  
992 production becomes light limited the nutrient level increases because plankton production  
993 cannot fix all the available nutrients (at mixing rates of 0.01 and 0.1  $\text{day}^{-1}$  in low and high  
994 light respectively). The increases in biomass is reflected in the productions, which also

995 increase roughly proportional to the biomass (panels c+h). However, the relative magni-  
 996 tude of the different productions changes with mixing rate, as reflected in the efficiencies  
 997 (panels d+i). The production efficiency generally increases with the mixing rate. Surpris-  
 998 ingly, the higher trophic level production can become larger than net primary production  
 999 resulting in  $\varepsilon_{\text{htl}} > 1$ . This occurs in oligotrophic situations where a high gross primary  
 1000 production fuels a high DOC production from exudation. That DOC also fuels plankton  
 1001 production, which eventually manifests itself in high net production to higher trophic lev-  
 1002 els. The gross efficiency of higher trophic level production ( $P_{\text{htl}}/P_{\text{gross}}$ ) will always be  
 1003  $< 1$ .

## 1004 **5.1 DOC and bacteria production**

1018 A difficult aspect of the ecosystem model is to parameterize the production and uptake of  
 1019 dissolved organic matter (DOC). Part of the difficulty stems from our incomplete knowl-  
 1020 edge of DOC: how much is labile and how much is not? And further: what are the sources  
 1021 of DOC: how much DOC is produced by incomplete assimilation and how much by pas-  
 1022 sive exudation – or between “income” and “property” taxes (Bjørnsen, 1988)? In the  
 1023 ecosystem model DOC represents the labile DOC that can be immediately taken up and  
 1024 used. Labile DOC is produced by incomplete assimilation of photoharvesting ( $\epsilon_L$ ), from  
 1025 passive exudation ( $j_{\text{passive}}$ ), from assimilation losses due to feeding ( $\epsilon_F$ ), and from viral  
 1026 lysis ( $j_V$ ).

1027 Pelagic ecosystem models typically describe DOC release as a constant fraction of  
 1028 fraction of primary production (Thornton, 2014), though some include size-based passive  
 1029 exudation (Kriest and Oschlies, 2007) using Bjørnsen (1988)’s model, or a more complex  
 1030 division between labile and non-labile pools (Anderson and Williams, 1998; Flynn et al.,

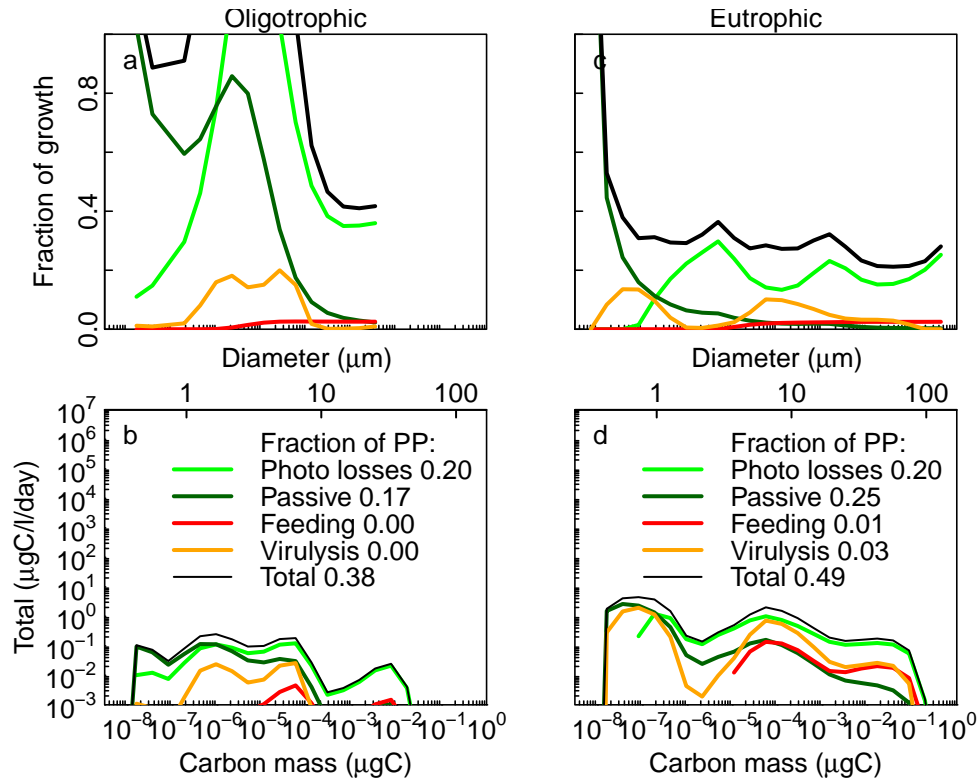
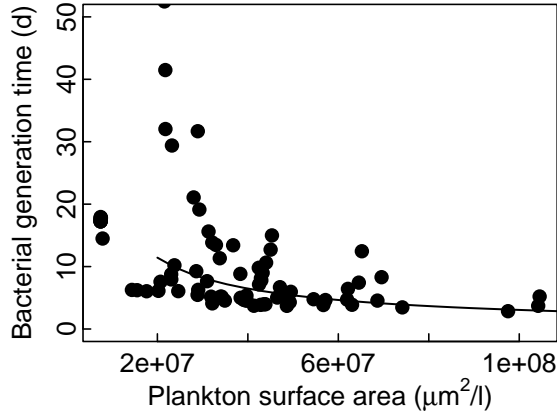


Figure 13: Losses to DOC as a function of cell size for oligotrophic conditions (Fig. 4.5) and eutrophic conditions (Fig. 9). Top row: losses as fraction of cell growth rate. Bottom row: total losses with fractions of gross primary production given in the legend.

2008). The size-based model represents all processes: passive size-based exudation, exudation due to uptake, incomplete feeding, and viral lysis, but does not distinguish between labile and refractory DOC.

All the incompletely assimilated carbon from photoharvesting is assumed to be available as DOC. The assimilation fraction is commonly set between 2-10%; following Anderson and Williams (1998) we use 20% ( $\epsilon_L = 0.8$ ) to have sufficient DOC available.

Passive losses are discussed in section 3.2; we assume that all passive losses are labile.



1010

1011 Figure 14: Bacterial generation time as a function of the total surface area of  
 1012 plankton in the size range  $ESD = 5$  to  $60 \mu m$ . The bacterial productivity is cal-  
 1013 culated as the flux of DOC uptake ( $j_{DOC}$ ) minus losses to passive exudation and  
 1014 respiration:  $\max\{0, j_{DOC} - j_{passive} - j_R\}$ . The generation time is 1 divided by  
 1015 the average of all bacterial productivities larger than 0. The lines shows a fit to a  
 1016 power law with fixed exponent  $-0.82$ . The mixing rate ranges between  $0.001$  and  
 1017  $0.1 d^{-1}$  and light is from  $10$  to  $60 \mu E/m^2/s$ .

1038 Feeding losses from phagotrophy are set to 20% ( $\epsilon_F = 0.8$ ). However, not all feeding  
 1039 losses may be available, as mostly non-digestible material will be exuded; Anderson and  
 1040 Williams (1998) send only 10% of feeding losses back to DOC so we set the available  
 1041 fraction at  $\gamma_F = 0.1$ . There is disagreement about the fraction of lysed cells that is  
 1042 available as labile DOC. Carlson (2002) find that the majority of the dissolved organic  
 1043 matter released from bacterial lysis is available, while (Anderson and Williams, 1998)  
 1044 only assume that 3.4% is available as labile DOC. We assume that half is available;  $\gamma_v =$   
 1045  $0.5$ . Feeding by higher trophic levels could also lead to DOC production. We assume that  
 1046 most sloppy feeding by higher trophic levels lead to particulate organic matter, which is  
 1047 not represented here, so  $\gamma_{htl} = 0$ .

1048 The model gives total DOC losses around 30 % (Fig. 13c+d), which is within the

1049 expected range. Overall we expect the average losses to DOC from passive exudation and  
1050 assimilation losses from feeding to be 10-30 % of their production (growth rate) (Kjørboe,  
1051 1993; Carlson, 2002). This is within the range simulated by the model Fig. 13a+b. Smaller  
1052 cells have much higher passive losses, which is essentially the process that determines the  
1053 lower cell size.

1054 The fraction of primary production becoming labile DOC should vary between 2-  
1055 40%; highest in oligotrophic waters (Teira et al., 2001). In productive regions DOC origi-  
1056 nates mainly from passive exudation while assimilation losses from feeding supply DOC  
1057 in oligotrophic regions (Teira et al., 2001). The observed fraction of primary production  
1058 exuded is 2-40 % (Teira et al., 2001; López-Sandoval et al., 2013); highest in oligotrophic  
1059 regions. The model have a total losses in the same range (Fig. 13c+d legend). An impor-  
1060 tant source in eutrophic waters is viral lysis while in oligotrophic waters the main source  
1061 is photoharvesting.

1062 Regarding the size-scaling of DOC losses the evidence is conflicted. Kjørboe et al.  
1063 (1990) sees strong evidence of size-scaling of passive exudation, Teira et al. (2001) sees  
1064 some indirect evidence, while Marañón et al. (2004) did not see any evidence (but notes  
1065 that nutrient limitation may be a confounding factor). The diverging evidence reflects  
1066 the difficulty in distinguishing between different sources of DOC and that studies focus  
1067 on different size-ranges of cells. For example, López-Sandoval et al. (2013) notes that  
1068 there is no overall size-scaling of DOC exudation among the plankton, which may be due  
1069 to different processes dominating among small cells (passive exudation) and large cells  
1070 (assimilation losses). The modelled total amount of DOC losses is roughly independent of  
1071 size (Fig. 13b+c), though with higher passive losses for small cells. Among smaller cells  
1072 the main source of losses are a combination of passive exudation, photosynthesis losses,

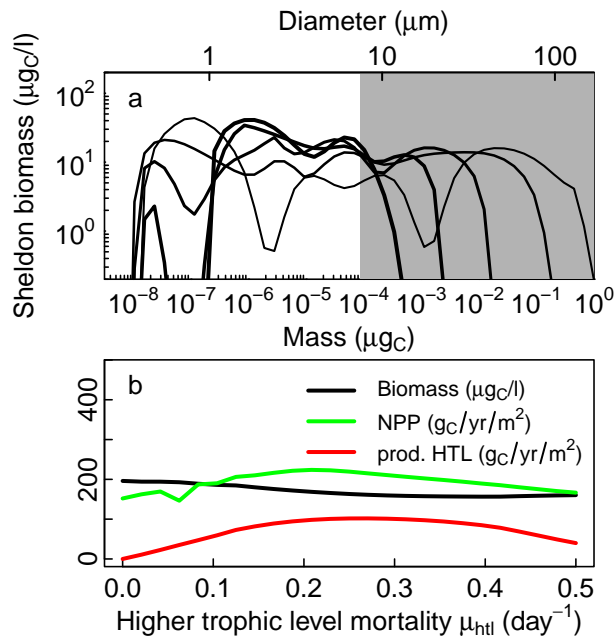
1073 and viral lysis. Among larger cells feeding assimilation losses are a potential important  
1074 term, which is, however, limited by the assumption that only a small fraction of feeding  
1075 losses are labile ( $\gamma_F = 0.1$ ).

1076 The only quantitative evidence on the size-relation between bacterial production and  
1077 cell size is that the bacterial generation time is inversely correlated with the total surface  
1078 area of plankton cells with a power-law exponent  $-0.82$  (Kiørboe et al., 1990). The  
1079 model gives a similar relation (Fig. 14), though with slower generation times. This may  
1080 have to do with how the average generation time is calculated. In the model the generation  
1081 time also includes cells with very slow DOC uptake rates, which increased the average  
1082 generation time.

1083 The modelled concentrations of labile DOC are very low (around  $1 \mu\text{M}$ ; Fig. 12b+g).  
1084 This is because all DOC is considered labile and it is therefore immediately taken up and  
1085 drawn down towards limiting concentrations which are around  $0.1 \mu\text{M}$  (Fig. 7). Including  
1086 also refractory DOC would allow for higher DOC concentrations.

## 1087 **5.2 Effect of higher trophic level mortality**

1095 The model results depend upon the mortality exerted by the larger multicellular organisms  
1096 as represented by the higher trophic level mortality  $\mu_{\text{htl}}$ . The importance of the HTL mor-  
1097 tality is not unique to size-based models; results of all plankton model are sensitive to this  
1098 closure term. However, the important effects of this closure term is rarely acknowledged  
1099 (but see Steele and Henderson, 1992). Varying the HTL mortality affects the size struc-  
1100 ture and the functions of the plankton ecosystem (Fig. 15). The main effect of increasing  
1101 higher trophic level mortality is to truncate the size-spectrum. The truncation releases  
1102 the smaller plankton from predation and they respond by becoming more abundant. Due



1088

1089 Figure 15: Effect of varying the mortality exerted by higher trophic levels. a)  
 1090 Sheldon size spectra for higher trophic level mortality from 0 to 0.5  $\text{day}^{-1}$  (thin  
 1091 to thick lines). The grey patch illustrates the size-range where HTL mortality acts  
 1092 directly. b) Effect of HTL mortality on ecosystem functions: total biomass, net  
 1093 primary production, and HTL productivity. Light and mixing are as the eutrophic  
 1094 situation Fig. 9

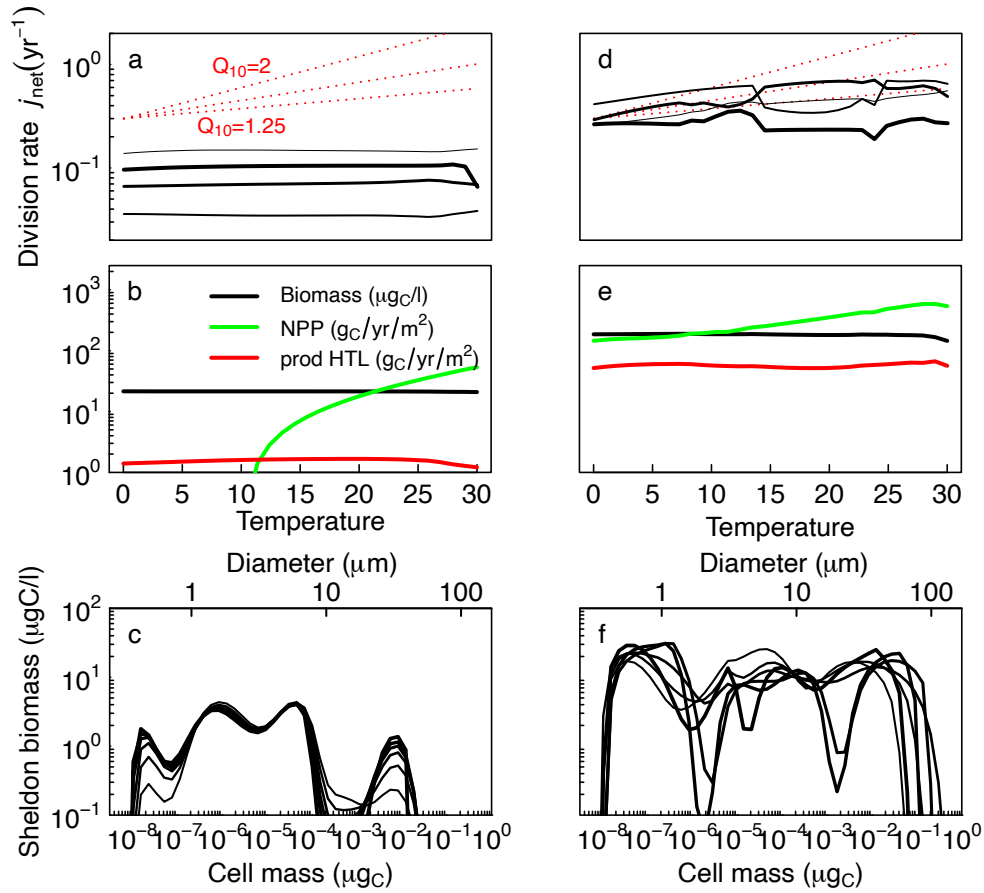
1103 to this trophic cascade the total biomass and the net primary production are only weakly  
1104 affected by the HTL mortality (Fig. 15b). The main effect is on the production towards  
1105 the higher trophic levels, which has a uni-modal shape with a maximum at intermediate  
1106 mortalities. This behaviour is similar to how fisheries models responds to fishing where  
1107 the maximum is termed the “maximum sustainable yield”. In lower productive system  
1108 the effect of HTL mortality is stronger and the peak in HTL mortality is reached at lower  
1109 mortalities (not shown).

1110 Since the HTL mortality is an extrinsic parameter, we would like to know a reason-  
1111 able value. That is difficult because the level of mortality depends on the predators: higher  
1112 productivity of the larger plankton will lead to a higher HTL mortality. This effect is also  
1113 seen inside the spectrum on the level of the predation mortality in Figs. 4.5-9b: in the olig-  
1114 otrophic system the level of mortality on the smallest plankton is around  $0.1 \text{ day}^{-1}$  rising  
1115 to around  $0.25 \text{ day}^{-1}$  in the eutrophic system. HTL mortality should therefore not be  
1116 higher than  $0.25 \text{ day}^{-1}$ . Here we have used  $0.1 \text{ day}^{-1}$ .

### 1117 **5.3 Effects of temperature**

1125 Water temperature directly affects affinities and metabolism and, through this, a host of  
1126 processes from the division rate of cells, to ecosystem structure and functions (see Sec-  
1127 tion 3.4; Fig. 16). Higher temperature increases the division rates of all cell sizes up to  
1128 a point where division rates begin to decrease (panels a+d). The increase is relatively  
1129 modest, though, and much less than indicated by a “metabolic”  $Q_{10} = 2$ , even for large  
1130 heterotrophic cells. It is also less than the  $Q_{10}$  values often used in plankton simulation  
1131 models (e.g. Archibald et al., 2022). The reason for the relatively slow increase in divi-  
1132 sion rates is that the cells are generally limited by encounter with resources (nutrients,





1118

1119 Figure 16: Effect of varying the temperature between 0 and 25 degrees in oligo-  
 1120 trophic/eutrophic conditions (left/right columns; Fig. 9). a,d) Effect on the di-  
 1121 vision rate  $j_{\text{net}}$  for 4 size classes (increasing line width). The red lines show  
 1122 temperature increases described by  $Q_{10}$  of 1.25, 1.5 and 2. b,e) Effect on ecosys-  
 1123 tem functions: total biomass, net primary production, and HTL productivity. c,f)  
 1124 spectra for 6 temperatures (increasing line widths).

light, and prey), which has a small  $Q_{10}$ . The decrease at higher temperature occurs when respiration losses, with a high  $Q_{10}$ , begins to dominate over the resource uptakes with smaller  $Q_{10}$ 's. The increasing division rates have a modest effect on ecosystem functions and structure (panel b-e), but they do increase net primary production and increases the maximum size in the size spectrum. The temperature response is, however, very dependent upon the conditions. Under oligotrophic conditions the temperature response is almost absent. The oligotrophic situation is dominated by carbon input from phototrophy (Fig. 9), which is independent of temperature ( $Q_{10} = 1$ ). Furthermore, light is available in excess (dotted green line in Fig. 9b) and can easily support the basal metabolism. Clearly, the response to temperature depend on the mixing rate and light in complicated ways (Serra-Pompei et al., 2019) making it hard to make generalisations.

## 6 Discussion

We have reviewed how cell-level processes can be related to cell size and first principles, and how they ultimately determine major aspects of plankton community structure and function. The approach builds upon the central role of cell size for resource uptake and metabolism of unicellular plankton. By cultivating a view of each cell as a “generalist” that can perform all types of resource uptakes – essentially being a combination of a bacteria, a phytoplankton, and a zooplankton – the trophic strategies become an emergent property. The fundamental processes at the cell level is based upon existing theory and knowledge with a few novel elements: a fluid-mechanical argument for the clearance rate, the upper limit of phagotrophic assimilation, and the identification of two scaling regimes for light affinity. The synthesis of all processes in a dynamic “minimal” size-

1155 based, along the lines of Ward and Follows (2016) and Andersen et al. (2015), leads to  
1156 a complete ecosystem model that resolves the community size spectrum as well as the  
1157 dominant trophic roles of plankton of different sizes. Novelties in the minimal model  
1158 is the inclusion of dissolved organic carbon to represent carbon reuse and the microbial  
1159 loop, and the development of closed-form analytical solutions for the scaling and level of  
1160 the size-spectrum. Throughout we have maintained a focus on simplicity of all processes  
1161 to bring forth a clear understanding of how each process contributes to the community  
1162 structure. Despite that operational plankton models – even those only based on cell size  
1163 – are more complex and complete than the minimal framework analysed here, the effects  
1164 of nutrient enrichment, higher trophic level mortality, temperature, and light upon the  
1165 structure and function of the community are likely to be universally present.

1166       The model generally reproduce observed ranges of biomass, chlorophyll, and produc-  
1167 tivity as observed in natural systems. The structure of the ecosystem is determined by a  
1168 combination of the bottom-up processes from nutrient availability and light, by the inter-  
1169 nal process of predation, and by the top-down process of higher trophic level predation.  
1170 As also shown by Poulin and Franks (2010) the availability of nutrients determine the  
1171 potential length of the food chain (the maximum size). This result is similar to the classic  
1172 insight in theoretical ecology about resource productivity determining food chain length  
1173 (Oksanen et al., 1981). However, the top-down effect of higher trophic level mortality  
1174 plays a key role in the structure and function of the community (Steele and Henderson,  
1175 1992). From a modelling perspective, this is problematic, as it to some degree ruins the  
1176 universal nature of the model: in a given situation the level of the higher trophic level mor-  
1177 tality needs to be determined. In global simulations the higher trophic level mortality will  
1178 vary depending upon the predation pressure from the multicellular plankton community,

1179 which is not the same throughout the global ocean. One solution is to use a higher trophic  
1180 level mortality that varies linearly with plankton biomass (a “quadratic” loss term); a more  
1181 complex solution is to include a representation of multicellular plankton (Serra-Pompei  
1182 et al., 2020). In terms of size spectrum slope, the model generally reproduce the com-  
1183 monly observed flat Sheldon spectrum (Sprules and Barth, 2016; Kenitz et al., 2019). The  
1184 model therefore reproduces the conclusion by Poulin and Franks (2010) that the spectrum  
1185 slope in itself is not informative of the plankton structure.

1186 In the following we discuss how first principles constrain the cell’s function and which  
1187 processes are still weakly constrained. Second, we discuss the limitations of the size-  
1188 based approach to modelling plankton communities and how it relates to trait-based and  
1189 functional-group based plankton models.

## 1190 **6.1 Parameters from first principles or empirical meta analy-** 1191 **ses**

1192 “First principles” are relations rooted in physics, chemistry, evolution, or geometry. Ty-  
1193 ing descriptions of processes and parameters to first principles has succeeded to varying  
1194 degrees. We distinguish between four levels of success: *i*) The process is known and the  
1195 parameter(s) can be calculated from first principles; *ii*) scaling exponents with cells size  
1196 (mass, volume, or diameter) are known from first principles but the coefficients have to be  
1197 calibrated with laboratory measurements or from meta-analyses; *iii*) The governing pro-  
1198 cess is known but the theoretical argument has not been developed and parameters rely  
1199 solely on empirical knowledge; *iv*) The empirical evidence is lacking and parameters are  
1200 only constrained indirectly via loose arguments or tuning of the outcome with observa-  
1201 tions of the community structure. For unicellular plankton all four levels are encountered.

1202 The most complete level (*i*) description is how diffusion limits uptake of dissolved nu-  
 1203 trient or DOC uptake and phototrophy, including the temperature scaling. For phototrophy  
 1204 there are clear theoretical arguments for all coefficients and scaling exponents – including  
 1205 the novel argument for the transition between two scaling regimes – simply from geomet-  
 1206 ric arguments. However, a better understanding of the quantum yield is needed to fully  
 1207 describe the observed variation in light affinity. The other well known effect is how the  
 1208 cell membrane limits the lower size of a cell, though the thickness of the cell wall is not  
 1209 (yet) constrained by first principle arguments. Another example of the role of geometry,  
 1210 which is not explored here, is the importance of a vacuole, a principle characteristic of di-  
 1211 atoms, to modify the diffusion uptake (Hansen and Visser, 2019; Cadier et al., 2020). The  
 1212 other level (*i*) description is the novel argument of how clearance rate of prey encounter  
 1213 is derived from fluid mechanics, though it must be recognised that the amount of energy  
 1214 available to the cell is a guesstimate. As the fraction of energy only enters as a square  
 1215 root in Box 3 this value is not crucial. Comparing the theoretical result with data (Fig. 4)  
 1216 shows a scatter of  $\pm 1$  order of magnitude. It is known how the variation in clearance rate  
 1217 across the mean is due to the hydrodynamics of different flagella arrangements that also  
 1218 results in different predation risk (lower clearance leads to smaller predation risk; Nielsen  
 1219 and Kiørboe (2021)) – we return to this in the next section.

1220 Most processes belong to the second level where scaling exponents are well described  
 1221 but empirical knowledge is needed to determine exact parameter values. To this category  
 1222 belong the processes related to the “secondary” scalings of nutrient and light uptake, i.e.,  
 1223 that the scaling is flat for small sizes. We can confidently argue that the scaling should be  
 1224 flat, but cannot determine the value of the parameters ( $r_D^*$  and  $r_L^*$ ). From the simulations  
 1225 we see that these flat scalings do not have a strong impact on the resulting ecosystem, so

1226 one could even omit them without a great loss of accuracy. Other level two processes are  
1227 the passive exudation, metabolism, and the temperature scaling of metabolism. The tem-  
1228 perature dependence of metabolism is a complicated mixture of many different processes  
1229 and a simple first-principles argument does not exist.

1230 Finally, some of the parameters associated to DOC losses are largely guesswork (level  
1231 iv). The poor state of knowledge is partly due to our limited understanding of the enor-  
1232 mous diversity of DOC compounds and their lability, which makes the lumping of DOC  
1233 into one group crude. We note that this problem is recently receiving attention (Zakem  
1234 et al., 2021) and hope that a better understanding of DOC is forthcoming.

1235 Overall, while it is clear that first principles constrain cellular processes there is still  
1236 room for improving the theoretical and empirical basis for estimation of some parame-  
1237 ters. How the values of the uncertain parameters influence community structure is partly  
1238 addressed by the analytical analyses in Sec. 4: the upper and lower cell sizes, the limit-  
1239 ing levels of resources and the sizes which are most competitive for resources, and the  
1240 overall biomass of the community. For example, the levels of DOC and nutrients depend  
1241 inversely on the diffusive affinity  $\alpha_D$  but increase with the coefficient of passive losses  
1242  $c_{\text{passive}}$ . Likewise, the minimum size increase with respiratory losses while the maximum  
1243 size decreases. Finally, the sizes where the dominant strategies change depend upon the  
1244 affinities (Table 3). For example, increasing the light affinity coefficient  $\alpha_L$  will decrease  
1245 the sizes where there is a switch from osmotrophy to light-limited phototrophy and from  
1246 light- to nutrient-limited phototrophy; increasing the clearance rate for phagotrophy  $\alpha_F$   
1247 will decrease the size where mixotrophy becomes dominant.

## 6.2 Size-, trait-, and functional-group based plankton modelling

The central role of cell size for all vital processes makes it an obvious choice as the structuring variable for plankton community modelling. The community structure is then described as the “size spectrum” which generally follows the flat Sheldon spectrum. The minimal size-based model takes this approach further as also the structure of the trophic strategies with cell size – on the continuum of osmo-, photo-, and heterotrophy (Andersen et al., 2015) – emerges as a second dimension of community structure. This idea was previously based upon simple scaling arguments with only a single scaling exponent for each process (Andersen et al., 2016; Ward and Follows, 2016). Here we show that the same results holds even with the more complex scaling two-regime scaling laws for nutrient and light uptake. However, the transitions between the dominant resource uptake power laws are still responsible for the structure of the trophic strategies in the full dynamic model (Figs. 4.5b and 9b). The advantage of the minimal size-based description is that the entire community, from the smallest bacteria to the largest heterotrophic cells, are captured with one set of parameters that is universal across geography and time. The universal properties makes the model well suited for global simulations (Ward and Follows, 2016) under global change. The obvious disadvantage is of course that biodiversity is only described by cell size and the dominant trophic strategy.

Additional diversity can be introduced by adding other traits in addition to cell size. The size-based approach is closely related to a trait-based description of plankton (Kiørboe et al., 2018) (also referred to as the approach of “infinite diversity” (Bruggeman and Kooijman, 2007)). Size-based models are essentially the simplest form of trait-based plankton models where the only trait is cell size. The trait-based approach represents plankton by a select few traits that together best represent the functional diversity of plankton.

1272 Traits are often related to investment in two competing resource uptakes or metabolic  
1273 functions (Andersen et al., 2015): light harvesting vs. maximum synthesis rate (Shuter,  
1274 1979; Serra-Pompei et al., 2019), light harvesting vs. nutrient uptake (Bruggeman and  
1275 Kooijman, 2007), adaptation between osmo-heterotrophy and phototrophy (Bruggeman,  
1276 2009; Ward et al., 2011), between nutrient uptake, light harvesting, and phagotrophy  
1277 (Berge et al., 2017). The trait-distribution of these traits are often Gaussian (normal dis-  
1278 tributed) and can be well represented simply by their optimal trait value (Shuter, 1979;  
1279 Chakraborty et al., 2020), or by their moments (Wirtz and Eckhardt, 1996; Norberg et al.,  
1280 2001; Bruggeman and Kooijman, 2007). Considering resource uptake traits in isolation  
1281 represents a limited aspect of plankton diversity because the big variation in resource up-  
1282 take parameters related with size is not represented. A full representation can be obtained  
1283 by combining resource uptake traits with cell size (Terseleer et al., 2014; Chakraborty  
1284 et al., 2017; Serra-Pompei et al., 2019; Chakraborty et al., 2020). The size spectrum itself,  
1285 however, is continuous as shown in the analytical derivation of the Sheldon size spectrum.  
1286 Descriptions where the size spectrum is only reduced to its moment or the optimal size  
1287 (e.g. Acevedo-Trejos et al., 2018) may represent the changes of one group of plankton,  
1288 but they are insufficient to resolve the entire community. Trait-size models therefore need  
1289 to combine a full resolution of the size-spectrum (as done here) but can use optimization  
1290 or moment-close to reduce the number of state variables for other traits.

1291 Plankton diversity is traditionally represented by dividing phyto- and zooplankton  
1292 into functional groups, including picoplankton, diatoms, flagellates, ciliates, etc. (Fasham  
1293 et al., 1990). Parameters in each group can be calibrated to represent the dominant group  
1294 in a given study area to achieve good fits with observations of the different taxonomic  
1295 groups. Their power comes at the expense of introducing additional parameters and by



1296 requiring re-calibration if there are changes in the dominant species groups due to envi-  
1297 ronmental change. A good example of a minimal functional-type model is the plankton  
1298 model of bacteria, auto- and heterotrophic flagellates, diatom, and copepods (Thingstad  
1299 et al., 2007). With the same complexity in terms of parameters as the minimal size-based  
1300 model, the Thingstad model provides an explicit taxonomic resolution that is lacking in  
1301 size-based models, though, of course, without the resolution of cell size. Size-based mod-  
1302 els are not replacements of functional-group type models, but the two types of models  
1303 should be considered as complementary descriptions of the same system. Therefore global  
1304 plankton models increasingly adopt descriptions that combine size and functional groups  
1305 (Stock et al., 2014; Dutkiewicz et al., 2020, e.g.) to provide generality to functional-group  
1306 type of models for global applications without inflating the parameter space, much like  
1307 the combination of size- and trait-based models discussed above.

### 1308 **6.3 Additional traits related to cell size**

1309 Besides the resource harvesting traits discussed above there are other traits which relate  
1310 to cell size. Here we first discuss the role of organisms that increase their physical size  
1311 without increasing carbon mass (diatoms and gelatinous zooplankton), alternative forms  
1312 of nutrient uptakes (diazotrophs), organisms with extreme predator-prey mass ratios (cil-  
1313 iates and larvaceans), the difference between bacteria and eukaryotes, and then present a  
1314 suggestion for additional trait axes to represent that diversity.

1315     Diatoms and gelatinous plankton increase their physical size by a large inert vacuole  
1316 or a gelatinous body. In this way they gain the advantages of large physical size: higher  
1317 nutrient uptake, higher photoharvesting rates, higher clearance rates, and lower average  
1318 predation risk, without paying the cost of building and maintaining a large carbon mass. In

1319 a sense their success hinges on lowering their effective body density. The advantages of a  
1320 lower body density follow directly from the size based relations developed here (Hansen  
1321 and Visser, 2019), however, variable density is not explicitly represented in the model  
1322 developed herein. Representing these life forms requires an additional trait, e.g. vacuole  
1323 size (Terseleer et al., 2014; Cadier et al., 2020) or body density.

1324 Diazotrophy is a dominant trophic strategy that is not represented in the minimal size-  
1325 based model. Diazotrophs fix dissolved nitrogen gas and thereby break away from the  
1326 diffusion limitation on uptake of bio-available nitrogen. However, they are also limited  
1327 by diffusive uptakes of dissolved phosphorous and iron. Diazotrophy requires an oxygen-  
1328 free environment, which forces the cell to limit the diffusion of oxygen into the cell. As  
1329 the diffusion of oxygen into the cell follows the same size scaling as diffusive uptakes  
1330 small cells will have a high influx of oxygen. It is therefore challenging for small cells  
1331 to develop diazotrophy. While the limitations of cell size on diazotrophy have not been  
1332 described in the literature the fundamental understanding of diazotrophy and the role of  
1333 oxygen is available (e.g. Inomura et al., 2017). With such a description, diazotrophy could  
1334 be added directly as an additional process into the minimal size-based model, without  
1335 even adding a new trait dimension, and make diazotrophy an additional emergent trophic  
1336 strategy.

1337 The minimal model assumes that all cells have the same preferred predator-prey mass.  
1338 Some organisms, however, may have very low predator-prey mass ratios, notably di-  
1339 noflagellates (Kiørboe, 2008), while others have high ratios, notably larvaceans. The  
1340 variation in preferred predator-prey mass ratios is accommodated to some degree by us-  
1341 ing a prey size preference with a wide size range  $\sigma > 1$ . However, that solution poorly  
1342 resolves the importance of organisms with large predator-prey masses in oligotrophic sit-

1343 uations where they act to transfer carbon from the dominant picoplankton towards larger  
1344 body sizes with a re accessible to higher trophic levels. Not resolving higher predator-prey  
1345 masses will underestimate the trophic efficiency in oligotrophic situations.

1346 Finally, the insistence on just one governing set of parameters for all sizes ignores the  
1347 difference between bacteria and eukaryotes. Bacteria have a different cell wall structure  
1348 which most likely limits their functional cell mass (the  $\nu$  factor) less than in the description  
1349 developed here (Kempes et al., 2016).

1350 Some of the limitations of the pure size-based approach can be addressed by including  
1351 additional traits as other axes of diversity. We consider two additional axes to be prime  
1352 candidates: vacuoles and a fast-slow life history axis. The vacuoles represent organisms  
1353 with a lower density (diatoms) and the methodology has been successfully developed pre-  
1354 viously (Terseleer et al., 2014; Cadier et al., 2020). Technically, vacuoles are introduced  
1355 as an additional size-spectrum with either a fixed vacuole size or a vacuole size which  
1356 is optimized dynamically. The other trait axis would be a representation of a slow-fast  
1357 life history continuum. This axis would represent how some species invest in high clear-  
1358 ance rates and high maximum synthesis rates to achieve a fast dominance in high resource  
1359 environments, while other invest in high competitive ability – low limiting resource and  
1360 low respiration – and/or defence to lower the predation risk. These investments comes  
1361 with trade-offs. The trade-offs between investments in resource harvesting and synthe-  
1362 sis is somewhat understood (Andersen et al., 2015) (but see Kiørboe and Thomas, 2020),  
1363 however, the investments in defence are more subtle. Recent developments in understand-  
1364 ing the trade-offs between clearance rates and predation risk of flagellates from direct  
1365 fluid mechanical simulations provides a first-principle avenue to parameterize this cru-  
1366 cial trade-off (Nielsen and Kiørboe, 2021). Incorporating the fast-slow life history axis

1367 would also address some of the scatter in the purely size-based data of clearance rate and  
1368 maximum synthesis rate (Figs. 4 and 5).

## 1369 **6.4 Conclusion**

1370 Despite the primitive representation of plankton diversity, the minimal size-based model  
1371 forms a backbone on which to add other complications. Its strength is conceptual sim-  
1372 plicity and a small set of universal parameters tied to first principles. The main effects  
1373 observed in the minimal model will also be manifest in more complex size-based mod-  
1374 els, and as such the model is a useful tool to understand the mechanics of more complex  
1375 size-based models. The importance of the additional complications – vacuoles, diazotro-  
1376 phy, high predator-prey mass ratios, or other functional groups – can be assessed with  
1377 reference to the minimal size-based model. While the model is not intended as an opera-  
1378 tional biogeochemical model, the computational simplicity of the minimal model makes  
1379 it useful as a basis for further theoretical ecological insights.

## 1380 **Code**

1381 R code to generate all figures on github: <https://github.com/Kenhasteandersen/FirstPrinciplesPlankton>.  
1382 The code also includes a web-based simulator, which can be found on: <http://oceanlife.dtuaqua.dk/Plankton/R>.

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## 1771 **A Calculation of effective prey preference for dis-** 1772 **crete size groups**

1773 The effective prey preferences function between size groups of predators  $i$  and prey  $j$  is  
1774 calculated by integrating over the prey size preference (Eq. 30). The encountered prey in  
1775 size group  $j$  by all predators in group  $i$  is:

$$E_{ij} = \int_{m_i^-}^{m_i^+} a_F m \int_{m_j^-}^{m_j^+} \phi(m, w) B(w) dw B(m)/m dm. \quad (\text{A.1})$$

1776 Here,  $B(m)$  represents the normalized biomass spectrum. We assume a Sheldon distri-  
1777 bution, i.e.,  $B(m) \propto m^{-1}$ . With the discrete prey and predator groups we write the  
1778 encountered food as:

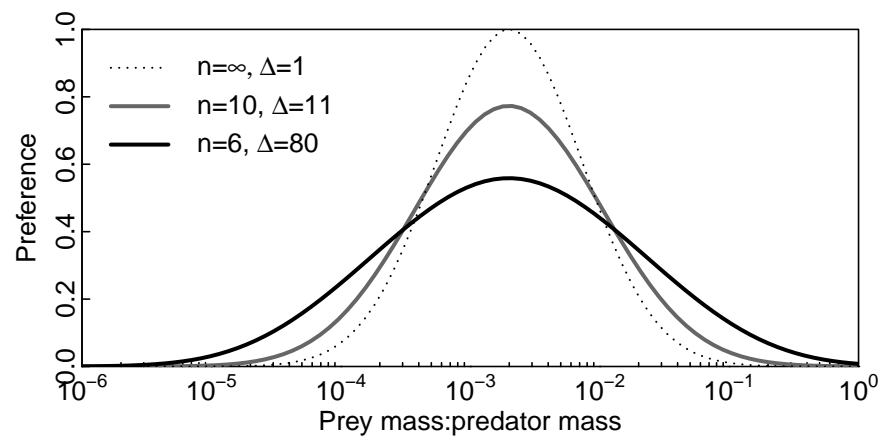
$$E_{ij} = a_F m_i \Phi_{ij} B_j N_i \quad (\text{A.2})$$

Where  $B_j$  is the total biomass in group  $j$ ,  $B_j = \int B(w) dw$  and  $N_i$  is the total abundance  
of predators  $N_i = \int B(m)/m dm$ . Equating the two terms and isolating  $\Phi_{ij}$  gives:

$$\begin{aligned} \Phi_{ij} = & \frac{\sqrt{\Delta}}{(\Delta - 1) \log(\Delta)} \\ & \left[ \left( \frac{1}{2} s \left( e^{-\frac{\log^2(\frac{\Delta z}{\beta})}{s}} + e^{-\frac{\log^2(\frac{\beta \Delta}{z})}{s}} - 2e^{-\frac{\log^2(\frac{z}{\beta})}{s}} \right) - \right. \right. \\ & \left. \frac{1}{2} \sqrt{\pi} \sqrt{s} \left( \log\left(\frac{\Delta z}{\beta}\right) \operatorname{erf}\left(\frac{\log(\beta) - \log(\Delta z)}{\sqrt{s}}\right) + \log\left(\frac{\beta \Delta}{z}\right) \operatorname{erf}\left(\frac{\log(z) - \log(\beta \Delta)}{\sqrt{s}}\right) + \right. \right. \\ & \left. \left. \left. \log\left(\frac{z}{\beta}\right) \operatorname{erf}\left(\frac{\log(\frac{z}{\beta})}{\sqrt{s}}\right) \right) \right) \right] \quad (\text{A.3}) \end{aligned}$$

1779 where  $s = 2\sigma^2$  and  $z = m_i/m_j$  and  $\Delta = m^+/m^-$ .





1780

1781 Figure A.1: Size preference function for different grid expansions ( $\Delta$ ) and number  
 1782 of size groups ( $n$ ).