because they pertain to different comparisons: traditional comparisons across species, which focus on intrinsic species traits, i.e., differences in body size (that yield sublinear scaling, $\beta < 1$), and comparisons of interacting individuals embedded in an environment with the same biomass density (that yield an isometric scaling, $\beta = 1$). We base our reasoning on respiration, but we demonstrate below that the same holds for growth and photosynthesis. We define the scaling exponent at the individual level with β to distinguish it from the scaling exponent α of populations or communities as in Fig. 1.

As observed in Fig. 1, total metabolism scales with total biomass as $E \sim B^{\alpha}$ independently of the average size $\bar{S}$ of organisms[1,3,13] (see also Supplementary Fig. 4). If we consider systems at a fixed biomass density B^*, the number of organisms N is inversely proportional to their average size $\bar{S}$ so that $B^* = N\bar{S}$[31]. Given that the average metabolism $\bar{E}_i$ of organisms in a population or community is equivalent to the total metabolism E divided by the number of individuals N, we inevitably find a linear relationship between individual size and metabolic rate:

$$\bar{E}_i = \frac{E}{N} \sim \frac{(B^*)^{\alpha}}{B^*/\bar{S}} = (B^*)^{\alpha-1}\bar{S} \propto \bar{S} \quad (1)$$

Since biovolume is fixed, B^* is a constant. The result of Eq. 1 is independent of the scaling exponent observed between total metabolism E and total biomass B (the value of α). Community metabolic scaling could follow any trend, but as long as it is not affected by size composition, it still leads to an isometric scaling between individual metabolism and size at a fixed biomass density (Fig. 2a), and vice versa.

If we instead consider systems at fixed population densities (number of organisms $N = N^*$, including traditional estimates of individual metabolic scaling based on $N^* = 1$), the average size of organisms becomes just a proxy for total biomass. Thus, when evaluating the individual metabolic rate as a function of its size, one retrieves the initial scaling of metabolism to biovolume observed in communities (Supplementary Fig. 6):

$$\bar{E}_i = \frac{E}{N^*} \sim \frac{B^{\alpha}}{N^*} = \frac{(N^*)^{\alpha}\bar{S}^{\alpha}}{N^*} = (N^*)^{\alpha-1}\bar{S}^{\alpha} \propto \bar{S}^{\alpha} \quad (2)$$

If we could quantify the scaling α at progressively lower N^*, until $N = 1$, then we would directly connect the scaling exponent β of individual organisms to the exponent α of populations/communities.

If neither biomass nor the number of organisms is fixed, metabolism can scale with size in several ways (Supplementary Fig. 6). Since metabolism-size relationships are often estimated for different organisms in varying conditions of population or biomass density, this variability could partly explain the inconsistencies in scaling exponents reported in the literature[6,10,32], including for phytoplankton[26,33].

Therefore, if the observation of Fig. 1 is correct (i.e., total metabolism scales with biomass independently of organismal size), competition with an equal amount of biomass shifts the scaling of individual metabolism with body size from sublinear (Eq. 2) to isometric (Eq. 1).

We validate these theoretical predictions with empirical data by testing the scaling between individual metabolic and production rates with cell size at a fixed biomass density. We use the full range of biovolumes of our system by rescaling all data points to a single biovolume value in the centre of the range (10^5 µm^3 µl^{-1}; Supplementary Fig. 7 for rescaling approach). As predicted, both organismal metabolism and production scale isometrically (linearly) with cell size at a fixed biovolume in monocultures (Fig. 2b respiration and production,

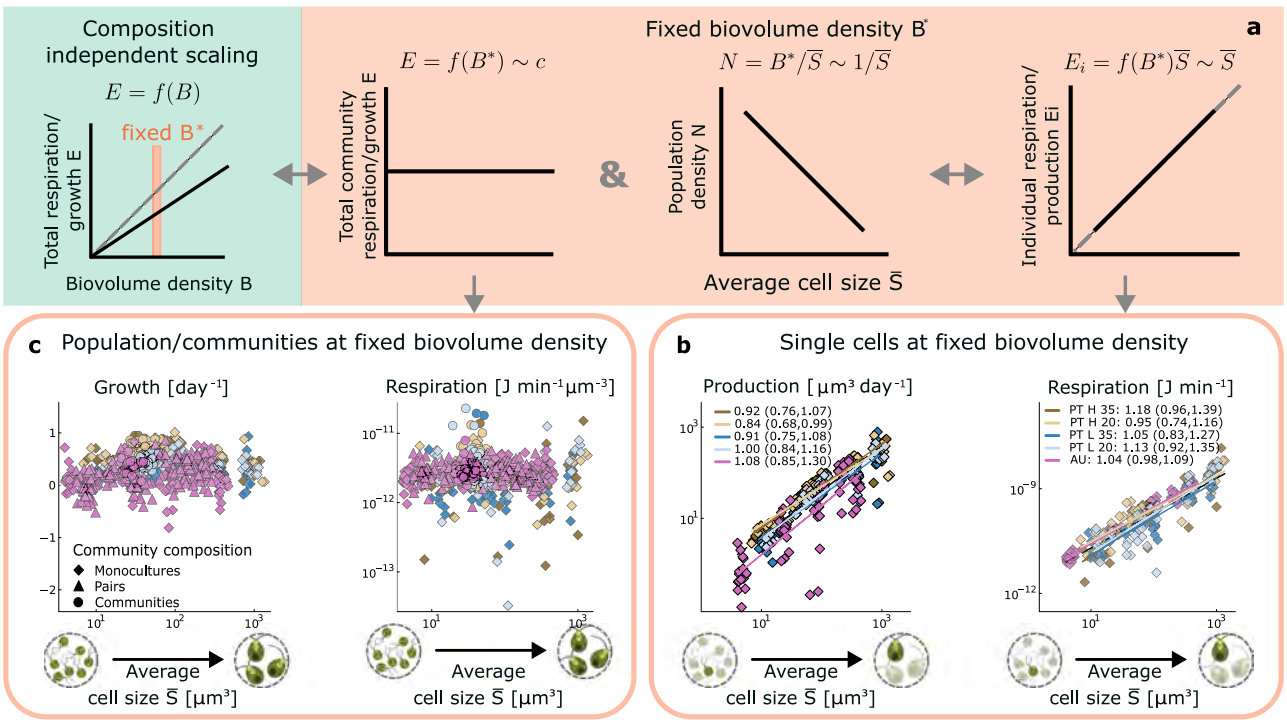

Fig. 2 | Theoretical predictions and empirical validation of the isometric scaling between individual metabolism and size in systems at fixed biomass density. **a** If species size does not affect the scaling between total metabolism and total biomass density (as observed in Fig. 1), individual metabolism should scale isometrically with size in systems at fixed biomass density B^*. The same should hold for production (biomass growth). **b** We confirm this result empirically: individual respiration (J/min per cell) and production rates (µm^3/day) scale linearly with cell size across phytoplankton species and environments when comparing organisms at the same biovolume density (10^5 µm^3 µl^{-1}). Here, we only use data from monocultures, but the scaling is robust even when considering data from communities (Supplementary Fig. 9). **c** As a consequence of isometric scaling, organismal size has no effect on the total respiration or growth rates of systems at the same biovolume density. Whole population/community rates are divided by total biovolume (thus are J min^{-1} µm^{-3}). Colours identify the treatment based on geographic location (AU = Australia, PT = Portugal), light (High vs Low) and salinity (35 vs 20 ppt). Source data are provided as a Source Data file.

Supplementary Fig. 8d photosynthesis). This result also holds for the *per capita* metabolic rates of "average" individuals in mixtures of species (pairs and communities; Supplementary Fig. 9) and when rates are rescaled to a range of fixed biovolumes (Supplementary Fig. 10). By demonstrating this theoretical result, we quantitatively validate our observation: community metabolism and biomass growth are independent of species composition and average size because organisms of different sizes respire and grow at the same rate per unit mass when compared at equivalent biomass densities (Fig. 2c respiration and growth, Supplementary Fig. 8a–c photosynthesis). We further show that individual metabolism and production scale sublinearly with cell size in most environments at fixed population densities (all photosynthesis and production rates scale sublinearly; the scalings of respiration are more variable and include exponents below (AU, H 35, H 20), above 1 (L 35) and close to 1 (H 20) with large confidence intervals that overlap 1 in most conditions; Supplementary Figs. 10, 11).

Notably, the variability in scaling exponents observed at fixed population densities collapses onto isometric scaling at fixed biovolume densities for all rates considered (i.e., when we compare small and large phytoplankton cells competing with the same amount of total biovolume, Fig. 2 and Supplementary Fig. 8). This shift in scaling implies that an increase in total biomass affects the metabolism of different organisms in a very similar way, independently of their size and identity, and regardless of how biovolume is distributed (i.e., many small or few large organisms, as shown in Fig. 2b). The metabolism and production of an organism are thus more tightly regulated by the total biomass with which it interacts than by its size. Since here we used data from monocultures, the total biovolume is composed only of conspecifics. If this result also holds in communities (i.e., is independent of biovolume composition), then we can explain why ecosystem production patterns show no effect of species size and composition.

Everybody is anybody: community composition does not affect metabolic density-dependence

In the previous section, we found that biomass competition affects metabolism and production in the same way across individual species (monocultures). Here we investigate whether these effects persist when species interact in communities. Specifically, we use monoculture data to predict the metabolism and growth of our phytoplankton communities, testing the importance of two factors (Fig. 3a):

1. biomass composition: does the biomass of other species (interspecifics) reduce organismal metabolism in the same way as the biomass of conspecifics?
2. species identity: how important are species-specific differences in growth and metabolism (Supplementary Fig. 3) when predicting community rates?

We start by testing the relative effects of intra- and inter-specific competitors on organismal respiration (1), assuming species identity matters (2) (as before, we base our reasoning on respiration). We use species-specific relationships between respiration and biovolume in each environment (Supplementary Table 4) to calculate the metabolism per unit biovolume of each species in the community, according to two extreme hypotheses (Fig. 3a):

a. competition for resources is stronger within species than among species[34], so only conspecifics reduce the metabolism of an organism while interspecific competitors have negligible effects; hence metabolism per unit biovolume e_s of each species s declines only in response to the biomass density of conspecifics: $e_s \sim {B_s}^{\alpha_s - 1}$.
b. phytoplankton species compete for similar resources, so intra- and inter-specific competitors have equal effects on metabolism; hence, the metabolism per unit biovolume of each species declines in response to the total biovolume density of the community: $e_s \sim {B_{tot}}^{\alpha_s - 1}$.

We estimated the respiration of each species in the community using each approach since we know the biomass density of conspecifics B_s and the total biomass of the community B_{tot} at each point in time. Finally, we calculated the total community respiration rate as the sum between species (predicted rates; see Methods for details) and compared these predictions with rates measured experimentally on communities throughout their growth.

Predictions based on the total community biovolume are accurate (hypothesis b; Fig. 3b). Conversely, if we do not account for interspecific competitors (hypothesis a), we overestimate community rates – in other words, we underestimate the level of metabolic suppression driven by competition. Thus, on average, respiration declines identically in response to intra- and inter-specific competitors. To further test this result, we explored an intermediate situation in which interspecifics might affect metabolism in a weaker way than conspecifics. To do this, we estimated the effects of each species on one another using data from pairs of species (this test was only possible for AU data). Despite its greater specificity, this approach does not improve predictions (Supplementary Figs. 12–14).

Now we challenge our second assumption: does species identity matter? We find that identity has negligible effects. If we randomise the association between species-specific declines in respiration rates and the biovolume of species in the community, we obtain a distribution of estimates that contains the prediction made before, using the correct association (Fig. 3c). So, predictions based on randomised associations perform similarly to species-specific predictions. The variability in metabolic density-dependence between species, therefore, does not meaningfully affect community predictions, at least based on our experimental accuracy. Importantly, the randomised distribution does not contain the prediction based on conspecific biovolume (hypothesis a), confirming that the biovolume of all competitors is the quantity that affects species respiration in communities.

If species identity has weak effects, then we can ignore it and estimate a general relationship between respiration and biomass by merging all species data (in each environment). So, instead of the expectation based on metabolic theory $E \sim \sum_i^N S_i^\beta$, we find

$$E \sim \sum_s^n B_s e_s \sim \sum_s^n B_s {B_{tot}}^{\alpha_s - 1} \sim {B_{tot}}^{\alpha} = \left(\sum_i^N S_i \right)^{\alpha} \quad (3)$$

where E is total respiration, $s \in [1, n]$ identifies species in a community and $i \in [1, N]$ identifies individual organisms (in populations or communities).

Despite the simplicity of this approach, based on a generalised decline in respiration with biomass across species, we can correctly predict community rates in all environments and across all growth phases (Fig. 3d). This approach ("general scaling") performs worse than that based on species-specific rates ("whole community") but is still within the randomised distribution (Fig. 3c). So we cannot state that there is no variability in metabolic responses between species, but this variation is not sufficiently strong to affect community predictions based on a species-naïve approach (see Supplementary Note 2 for details on the importance of species identity). Thus, even in a community of interacting species, respiration slows with increasing biomass at the same rate on average among species, regardless of the nature of the biomass (i.e., the relative abundance of intra- and interspecifics, and their size).

All the considerations we have done for respiration extend to biomass growth (Fig. 3d and Supplementary Fig. 15) but only partially hold for photosynthesis. Total biomass (not conspecific biomass) is still the relevant quantity to consider when predicting community photosynthesis (Supplementary Fig. 16a, as observed for respiration in Fig. 3). But species identity has stronger effects (Supplementary Fig. S16b), probably because of the unimodal scaling of photosynthesis with cell size (Supplementary Fig. 8, which is not obvious for