Thanks for the very constructive comments. We have made our new version of analysis and presentation following reviewer's suggestions. Below, we copied reviewer's comments in bold, followed by our responses. The changes in the revision are shown in italic. Updated figures and tables are shown in the end of this document.

This study addresses the size-scaling of microphytoplankton growth rate and grazing rate due to microzooplankton using data from dilution experiments conducted in the East China Sea. Furthermore, the authors attempt to relate the size-scaling of microphytoplankton growth and mortality to the size-structure of microphytoplankton encountered in situ, in turn described by normalised-biomass size-spectra (NBSS). The main findings are that growth rate increases with cell size whereas mortality is size independent. The authors also conclude that size differential grazing mortality drives the observed variability in the slope of the NBSS.
Understanding the size-scaling of phytoplankton growth and mortality in the sea is an important topic, and relating these size-scaling relationships with the observed size structure of phytoplankton assemblages represents quite a challenge. The data presented here may prove to be helpful in this respect. However, the paper as it stands has many serious shortcomings and its eventual publication could only be recommended if the authors undertake a really major revision. The changes needed are so substantial that it is fair to say an altogether new manuscript must be written. The key issues are: i) the observed positive size-scaling of growth rate in the studied cell size range is very difficult to explain mechanistically given our knowledge of phytoplankton physiology – therefore it may have been the result of an artifact of experiments or data analysis; and ii) given that the authors failed to observe size-dependence of mortality rate, size differential mortality cannot be the driving force explaining the variability in the slope of the biomass size spectrum.

The comments are very constructive. Following the suggestions, we have almost completely rewritten the manuscript. In this revision, we have clarified the within-assemblage and among-assemblage issues. For the **within-assemblage pattern**, first, to test MTE, we investigated the scaling of size-specific growth rate and mortality "for each station" using simple regression analysis. We found that the scaling exponent varies among stations, but generally does not approach -1/4 as MTE predicts (Fig. 3). Second, to estimate the general scaling, we further "pooled all stations and used GLMM (stations as the random effect)". We found that on average the scaling is nearly isometric (slightly positive), and again, this does not support MTE (Table 1). While we present and discuss the possible isometric scaling of growth rate and mortality, we caution these results. This is because the scaling exponent varies substantially among assemblages.

Because the scaling varies among stations, we further investigated how this variation can contribute to affect the size structure (size spectral slope) **among assemblages**. To do so, we used simple linear regression analysis. We found that it is the relative mortality of small versus large individuals contributes most to determine the among-assemblage variations of size spectral slopes (Table 2). We further linked such variations in scaling of size-specific growth rate and mortality to environmental conditions (Fig. 5). The updated tables and figures are in the end of this document.

In the revision, we have clarified the two major comments of the reviewer. 1) There is no clear evidence of positive scaling of growth rate; rather, the scaling is almost isometric. More importantly, the scaling varies among assemblages. We have also caveated the potential issue of limited size range in our experiments. 2) There is indeed size-dependence of

mortality, and such dependence varies among stations. It is such variation that contributes to explain the variation of biomass size spectral slopes among stations. The major comments arise because of our unclear statements in the original manuscript. We have now clarified those issues. The detailed explanations can be found below.

Below I structure my review as follows: i) writing, ii) data presentation and analysis, and iii) interpretation and conclusions.

Writing

The writing is quite poor generally. It is obvious that the authors have not taken the time to review carefully the text before submission. Otherwise it is impossible to understand the number of spelling mistakes. As an example, I counted 8 spelling mistakes in the 'Conclusions' section alone, which is 12 lines long. Words such as 'phytoplankton', 'assemblage', 'logarithm' appear misspelled in various, sometimes imaginative, ways throughout the manuscript. Such lack of care is not acceptable: the ms is this form should never have been submitted to the journal. Once it was submitted, it should have been returned to the authors for a thorough correction before sending it to reviewers.

Thank you for pointing out the problems. We have carefully taken care of writing and references in our revision.

In addition to multiple spelling mistakes and poor grammar, the text suffers from lack of precision. Many procedures in the data analysis are not described clearly (e.g. the sections on path analysis and regression analyses are particularly confusing).

The path model analysis has been removed since it does not include random effect to allow us to perform within assemblage analysis. We have clarified our analyses in the revision. See our response to data presentation and analysis below.

References to the literature are often inaccurate or irrelevant. A few examples follow: Page 3, lines 1-2: 'Physiological constraints mainly base on the body size (Brown and Gillooly, 2003; Brown et al., 2000; Cermeño et al., 2006). This sentence is meaningless and the choice of references seems haphazard.

This sentence has been removed, and the first paragraph in Introduction section has been revised as following:

" Growth and mortality represents two key ecological processes of organisms. The phytoplankton community growth rate is determined not only by temperature and resource availability, but also the size composition of the community. Temperature effects have been known to be positive on the maximum phytoplankton growth rate (Bissinger et al., 2008; Eppley, 1972). The effects from resource availability, such as nutrient and light, on phytoplankton population growth depend on body size (Finkel, 2001; Finkel et al., 2004; Irwin et al., 2006). In general, large phytoplankton exhibit a lower photosynthesis rate because of the package effect (Berner et al., 1989) and a lower nutrient uptake rate because of lower surface-to-volume ratio (Kiorboe, 1993). However, when light and nutrient are sufficient, large individuals could have competitive advantages over small individuals (Maguer et al., 2009) due to their low susceptibility to light damage and higher carbon-

specific photosynthesis rates (Cermeño et al., 2005; Key et al., 2010)."

Line 28, page 12. Finkel 2004 is a lab study and does not report on the size-scaling of chla content in the field.

We agree with this comment. This sentence has been removed. The explanation for size-specific growth rate scaling has been modified in Sec. 4.1 as follows:

"*4.1 Scaling of size-specific growth rates (μ) and mortality (m)*

The scaling exponent of size-specific growth rate and mortality varies among stations (Fig. 3; Table B2); this finding does not support MTE. This result suggests there may be no universal scaling of size-specific growth rate and mortality in natural assemblages, as suggested by Glazier (2005). However interestingly, such variation could subsequently be used to explain the variation of NBSS slopes among stations (see Sec. 4.3).
Nevertheless, we still tried to estimate the average scaling using GLMM. The results of GLMM suggest a nearly isometric scaling of size-specific growth rate for natural microphytoplankton assembalges in the East China Sea (Table 1); again, this finding does not support MTE either. In fact, our observed general scaling exponent of 0.092 (result of GLMM) for size-specific growth rate could be converted to 1.092 for individual-specific growth rate. This value is comparable with the reported values of individual-specific metabolic rates observed in other studies, which ranged from 0.9 to 1.2 (Maranon, 2008; Maranon et al., 2007). Moreover, the 95% confidence interval of our individual-specific growth rate scaling exponent (1.056 to 1.123) is comparable to those calculated in Huete-Ortega et al. (2012), where the individual-specific carbon fixation rate is reported to range from 1.03 to 1.32. Together with the results of other studies showing isometric scaling between individual respiration and body size in other phytosynthetic plants (Reich et al., 2006), our results cast doubts on an ubiquitous negative one-quarter scaling rule (Brown et al., 2000; Cermeño et al., 2006; Niklas and Enquist, 2001) between size-specific rates and body size in natural phytoplankton assemblages.
Accroding to MTE, geometric constraints in resuource acquisition and transportation network lead to the observation of allometric scaling (-1/4 scaling exponent) (Banavar et al., 2002). However in our study, we found a nearly isometric (slightly positive) size-specific growth rate scaling exponent. Such findings could stem from the following features possessed by the larger phytoplankton to overcome their geometric constraints. In terms of nutrient acquisition, large phytoplankton show isometrically scaling relationship between nutrient uptake rate and body size (Marañón et al., 2012). In terms of photosynthesis, large phytoplankton contain isometrically increased chloroplasts to body size ratio (Maranon et al., 2007). Also, the large phytoplankton exhibit higher carbon fixation to chl a ratio (Huete-Ortega et al., 2011). Although the large phytolankton would suffer from package effect (Berner et al., 1989), they would subject less from light damage and are less susceptable to photoinactivation, which is commonly observed in small phytoplankton (Key et al., 2010). Besides, the large phytoplankton could overcome constraints of transportation network through the following strategies. Large phytoplankton could increase their vacuole size to elevate storage ability (Thingstad et al., 2005; Latasa et al., 2005; Stolte et al., 1994) and attain higher photosynthethic efficiencies (Cermeño et al., 2005). In conclusion, the isometric scaling of size-specific growth rate is possible under sufficient light and nutrient conditions. Note however, as the scaling exponent of size-specific growth rate vares among assemblages, we are not certain that our results clearly support the isometric scaling."

Page 13 lines 12-14 The correct slope values in Maranon et al (2006) are the other way around (1.14 in coast and 0.96 in open ocean). Besides, and contrary to what the authors write (page 13, lines 11-12), this study does not compare locations with different irradiance (samples from the whole euphotic layer are used) but, rather, different nutrient availability.

This sentence has been removed.

Data presentation and analysis

Data presentation is excessively succint. Only the combined size-scaling relationships for growth and grazing are shown. However, these data originate from individual dilution experiments – about which no information is given. How many of all dilution experiments conducted / size classes considered yielded significant slope values? What was the r2 in the regression analyses? At the very least, plots of μ vs dilution factor from some representative experiments and size classes should be shown, and a table (or an Appendix) should be prepared containing the statistics.

We have now claridied the analyses following the suggestions of the reviewer in the revision as follows:

"*2.5 Data analysis*

2.5.1 Calculation of size-specific growth rate and grazing mortality

To estimate the size-specific growth and mortality rate of microphytoplankton, we first constructed the size spectrum of microphytoplankton at T_0 and T_{24} (Fig. 2). The Normalized Biomass Size Spectra (NBSS) of phytoplankton were employed in this study. We divided the total biomass of each log_2 size class by the width of the respective size class as described by Platt and Denman (Platt and Denman, 1977; Sheldon et al., 1972). The microphytoplankton biomass within this range expands 12 orders under log_2 scale. We implemented log_2 in size class in order to be in accordance with the standard convention, as well as keep the highest size resolution possible. In this manner, we estimated the biomass of each size class at T_0 and T_{24}. This new method has an advantage over traditional methods for obtaining size-fractionated chl a measurements, which exhibit difficulties with high-resolution data.

The growth and mortality rates were estimated using a linear regression of realized phytoplankton growth rates of four dilution treatments versus the corresponding dilution factors. Thus, we could calculate the slope as the grazing mortality (m) and the intercept as the intrinsic phytoplankton growth rate (μ) (Landry and Hassett, 1982; Landry et al., 1995). The novel and additional calculation here is that, for each size class, we carried out linear regression of realized phytoplankton growth rates on four dilution treatments to estimate size-specific growth and mortality rates (Fig. 2). In addition to μ, we also measured the size-specific growth rate without nutrient amendment (μ'). Consequently, the size-specific growth rate with and without nutrient amendment (μ and μ') and grazing mortality (m) of microphytoplankton can be estimated.

2.5.2 Data pre-treatments

Before analyses, we performed two pre-treatments. First, we used only the growth rates measured with nutrient amendment (μ) after temperature correction to test MTE. The prerequest to test MTE is that the growth rate should not be limited by resources, such as nutrients or light for phytoplankton. Therefore, we use the growth rates measured with nutrient amendment (μ) for testing the MTE. According to the MTE, the temperature effect on

growth rate and mortality should be adjusted (Brown et al., 2004). Thus, the temperature corrected rate (M_c) was calculated from the measurement (M) as following: $M_c=M\times e^{E/kT}$, where E is the activation energy (in electronic volts [eV]), k is the Boltzmann constant ($8.617\times10\text{-}5\ eV\ K^{-1}$), and T is the absolute temperature in K. In this study, the activation energy was set to be 0.32 eV (Allen et al., 2005; Lopez-Urrutia et al., 2006). The second pre-treatment is that, for each station, we removed negative size-specific growth rate and grazing mortality for further analyses. Because of the rather fine scale in size class defined in our study and sampling error, it was possible for certain size classes to exhibit negative size-specific growth or grazing mortality. After removing negative values, 200 out of 312 (12 size classes in each of 26 stations) size classes (having both positive size-specific growth rate and grazing mortality) were left.

2.5.3 Scaling of size-specific growth rate and grazing mortality within assemblage (station)
To achieve the first objective, we performed a within assemblage analysis to test whether the μ and m generally scales with the body size with an exponent of -1/4 after temperature correction for each station. This was done using a simple linear regssion of size-specific growth rate (or motality) against size class for each station. To further investigate the general scaling, we pooled data from all stations and used the Generalized Linear Mixed effect Model (GLMM) (Bolker et al., 2009) to estimate the average exponent. In GLMM, stations were considered as the random effect, because we aim to examine whether a general scaling relationship exists within assemblage. Including stations as a random-effect variable removes the possibility for any spurious relationships arising from variation across seasons or space while using data from all stations to increase sample size. We further investigated the scaling of μ and m on body size for each cruise following the same fashion of GLMM analysis.

2.5.4 Coupling between size-specific growth rate and grazing mortality
To further clarify the relationship among microphytoplankton body size, size-specific growth rate, and size-specific grazing mortality, we regressed the size-specific grazing mortality against size-specific growth rate, using GLMM with stations as the random effect. Moreover, in order to partition out the effect of body size, we additionaly implemented a linear regression model to regress the residuals from size-specific grazing mortality GLMM (of size-specific rate versus size with stations as the random effect) against the residules from size-specific growth rate GLMM. By doing so, we aim to examine if the microphytoplankton grazing mortality and growth rate are coupled together within an assemblage regardless of body size. Note, the growth rates used here are those measured without nutrient amendments (μ') so that we investigated the real situation in nature. Nevertheless, the analysis on μ reveals qualitative similar conclusion.

2.5.5 NBSS slope variation among assemblages
To achieve our second objective, we examined whether the variation of NBSS slope across environments was related to the variation of relative growth rate and grazing mortality of small versus large individuals among assemblages. This is motivated by the finding that the size-specific growth rate and grazing mortality scaling varies among stations (See Fig. 3 and Table B2 in Section 3.1).
Ideally, we can perform a regression analysis between the NBSS slopes and the scaling exponents across stations. However, estimation of the scaling exponent for each station may be subject to high uncertaity for some stations. This is because some size classes need to be removed due to the negative size-specific growth rate or grazing mortality; after removing these data, the sample size was too small to reliably estimate the scaling exponents for some

stations. To overcome this difficulty, we binned the size classes into size categories to prepare explanatory variables instead of using the slope (exponent) of each station directly for analysis. Specifically, we binned the smallest four size classes (2^6 to 2^{10} pg) into the small size category, the middle four size classes (2^{10} to 2^{14} pg) into the medium size category, and the largest four size class (2^{14} to 2^{18} pg) into the large size category, and calculated the average growth rate and grazing mortality for each category. Following this binning approach, the growth rates and grazing mortalities of the large and small size category influence the NBSS slope most, but the rates of medium size category show no influence. Therefore, only the size-specific growth rate measured without nutrient amendments and grazing mortality of small and large size category (μ_S', μ_L', m_S, and m_L) were investigated. Note here we used the growth rates measured without nutrient amendments (μ') so that we investigated presumably the in situ growth rates.

For the correlation analysis, considering the strong correlation between the growth rate and grazing mortality (Barnes et al., 2011; Landry et al., 2000; Murrell et al., 2002; Chen et al., 2009), we used univarate linear models instead of step-wise selection to avoid the issue of colinearity. We analyzed 9 univariate regression models. The independent variables of these 9 models including 2 growth rates and 2 grazing mortalities as described above (μ_S', μ_L', m_S, and m_L), 2 grazing impacts (I_S', and I_L' where I'=m/μ') designed to measure the grazing pressures of two size categories without nutrient amendments, and 3 ratios (μ_S'/μ_L', m_S/m_L, and I_S'/I_L') of small over large category designed to explore the relative importance of small versus large size category in terms of the size-specific growth rate, grazing mortality and grazing impact.

In these analyses, we focused only on biologically possible effects of each independent variable on the NBSS slope (i.e. we tested whether the relationship significantly follows the biological expectation using one-tail tests) (Table 2). For example, relatively higher growth rate of small over large phytoplankton category (μ_S'/μ_L') is expected to decrease (steepen) the NBSS slope, and is not possible to directly produce a flatter size-spectral slope; thus, the anticipated correlation of μ_S'/μ_L' versus NBSS slope is negative (Table 2). To represent the effects from real measurements, the size-specific growth rate and grazing mortality values used in these 9 models were not corrected by temperature nor log-transformed. The size-specific growth rates are from measurements without nutrient amendments.

2.5.6 Environmental effects on the variation of NBSS slopes among assemblages (stations)

We conducted redundancy analysis (Legendre and Legendre, 1998) to examine if the environmental factors (explanatory matrix) can explain the 9 independent variables (response matrix). This analysis is dedicated to link the environmental conditions to size-specific growth rate, grazing mortality and finally to NBSS slope. The environmental factors include nitrite + nitrate concentration (N), Photosynthesis Active Radiation (PAR), phosphate concentration (P), salinity (S), silicate concentration (Si), and temperature (T).

2.5.7 Further analyses to test the robustness of the results

Beacuse some stations could show nonsignificant regression results in dilution experiments, we prepared the following four reduced data sets to test the robustness of our results. For the first reduced data set, we removed the size classes with the regression p-value larger than 0.25 in dilution experiments, regardless whether the regression p-value in dilution experiments for the whole community is high or low. To prepare the second reduced data set, we removed the stations with the regression p-value for the whole community larger than 0.25. For the third set, we first removed the stations with the regression p-value for the whole community larger than 0.25 and then removed the size classes with the regression p-value

larger than 0.25 in the remaining stations. The fourth set is prepared by removing the stations with average regression p-value of all size classes larger than 0.25 in that station. All the four reduced data sets were analyzed with the same manner as the procedure used for the whole data set."

In addition, we have now provided more detailed data in Supplement B (as shown in the end of this document).

Nothing is said about actual values for growth and mortality rates. After reading the ms, one does not know if phytoplankton in the region were growing at a rate of, say, 0.5 d-1, or 2 d-1. Rates reported in Figs. 4-6 are temperature-corrected (this should be clearly stated in the legends) and therefore cannot readily be interpreted in terms of real, in situ growth rates. This information should be given in a table. In addition, no description is made of the spatial variability in growth and mortality rates. For instance, was phytoplankton near the coast growing faster or slower than open-sea phytoplankton? No description is made of hydrographic conditions (temperature, mixing regime, nutrient concentration) in the different stations occupied. A Table is included as an Appendix but its contents are not even mentioned in the text. What is the relationship between those conditions and phytoplankton growth and mortality? And the same question goes for the biomass spectra: what is the relationship between hydrographic/nutrient variability and changes in the intercept and slope of the NBSS? The manuscript should show the general hydrographic context and then present the spatial variability in measured rates (at least for the whole phytoplankton assemblage) as well as the parameters of the biomass spectra. The slope of the NBSS should be reported for each sampled station, and compared with data from the literature.

We add an additional table in supplement B (Table B1) in the bottom of this response letter. Table B1 include (1) Average particles processed in T_0, (2) Particle density (ind./ml), (3) Biomass (μg/L), (4) Average p-value of dilution exp. across size classes, (5) NBSS slope, and (6) r^2 of NBSS.

In fact, our study focuses on the "size-specific" growth rate and grazing mortality instead of total growth rate and grazing mortality. To avoid distraction, we thus did not present or discuss total growth rate and grazing mortality in our manuscript. To help readers, we present the "Average p-value of dilution exp. across size classes" in Table B1.

To link the pattern of size-specific growth rate and mortality to environmental consitions, we included addtional redundancy analysis (Sec. 2.5.6 above). We added one more paragraph in Result section (Sec. 3.3). Figure 5 is shown in the end of this document.

"The growth rate and mortality of small versus large individuals are further linked with the environmental factors using RDA (r=0.506; p=0.061; Fig. 5). The first axis of RDA explains 46.13% of the variation, and the second axis explains 2.39%. The first axis is associated with μ_L' and m_L and is mainly positively contributed by phosphate concentrations. Thus, judging from the RDA biplot (Fig. 5), growth rate and grazing mortality of large individuals is positively correlated with the phosphate concentration."

We also discussed how our findings differ from other studies in the Sec. 4.2 as follows:

"*We further link environmental conditions with the 9 independent variables and then to NBSS slopes, using RDA (Fig. 5). The results of this analysis suggest that higher phosphate concentration provokes the growth rate of the large individuals (but not the smalls) first, beacuse the nutrient availability tend to induce the bottom-up (growth) forces in community dynamics (Power, 1992). Also recall in our observation that growth rate is the main factor leading to elevated grazing mortality (Fig. 4). The raised growth rate of large phytoplnakton would subsequently promote the grazing pressure on their own. The NBSS slope consequently become steeper (more negative).*
Note that, we also observe significant positive correlation between NBSS slope and the growth rate of small individuals (μ_S' in Table 2) as well as relative growth rate of small versus large individuals (μ_S'/μ_L' in Table 2). Biologically, the increase in these two variables should have promoted the abundance of small individuals and consequently steepened the NBSS slope (more negative slope). Thus, the estimated positive coefficients of these two models are spurious correlations resulted from the covariance between size-specific growth rate and grazing mortality (Fig. 4).
In conclusion, we found that in relatively higher nutrient environments in the ECS, the grazing pressure was relatively higher on larger individuals; as a consequence, the NBSS slope was more negative (steeper). This is in contrast with the often observed pattern that the NBSS slope of phytoplankton size distribution is flatter in higher nutrient environments (Reul et al., 2008). Such patterns are generally found in the environment where bottom-up control dominates. However, our observations suggest that during our experiments in the ECS, the top-down control could be more important. Our findings imply that top-down effects may play an important role in determining phytoplankton size structure. Such kind of top-down effects on size structure is overlooked in the literature and deserves more attention (Brucet et al., 2010; Shurin et al., 2012).

Page 12, lines 10-12 When comparing their results with those reported in the literature, the authors must be aware of the differences in the cell size range considered. The present study focuses on the microphytoplankton size range – the cell biomass range goes from approximately 100 pgC/cell to 130000 pgC/cell, which is about 3 orders of magnitude in cell size. In contrast, the studies cited in this section, such as that of Chen and Liu (2010), consider much wider size ranges, from approximately 0.1 pgC/cell to 100000 pgC/cell (6 orders of magnitude). Chen and Liu (2010) found a positive sizescaling only in the small-to-intermediate cell size range, while the size-scaling was negative in the intermediate-to-large cell size range. It is this latter size range that is relevant for the present study, where also large cells are considered.

We agree with this comment. We thus have removed Chen and Liu (2012), since it states unimodal scaling. But they also claimed in their articles that their data could be fitted into linear model as well. We explicitly caveat our limited size range as well as potential experimental biases in Sec. 4.3 as follows:

"*4.3 Difficulties in testing the MTE in natural phytoplankton assemblages*

While we tried our best to carry out the experiments in order to investigate the in situ phytoplankton dynamics to test the MTE, some possible incubation artifacts remain. As pointed by Dolan and McKeon (2005), the grazing behavior of microzooplankton could be altered by the dilution processes, especially in the most diluted treatments. The

microzooplankton in most diluted treatment would grow slowly and display low grazing rate due to food limitation (Dolan et al., 2000). This could results in overestimatation of grazing mortality and underestimation of growth rate in community level. In addition, our bottle incubation could elimitate the already low density large predators that likely feed on large microphytoplankton. This elimination may mislead our observations. However, Landry and Calbet (2005) validate the dilution experiment by finding correspondence between rate estimates from dilution experiments and other isotopic assesments. Still, given relatively scarce studies on size-specific growth rate and grazing mortality, it is not clear how the technical issues of dilution experiments could affect the size-specific level investigation. Another difficulty in testing MTE lies in the limited body size range examined in our stuty. Narrowing the body size range decreases the explanatory power of body size to metabolic rates (Tilman et al. 2004). In our study, about 3 order of magnitude (about 100 pgC/cell to 130000 pgC/cell) of size range is examined, and body size explains only about 37% of variation. Our size range is much less than the meta-analysis reported in the MTE (~15 order of magnitude). In addition, the phylogenetic structure could play an important role and obscure our finding in scaling. As demonstrated in Seibel (2007), if the differences in scaling exponents among phylogeny groups were large, the generality of MTE might be blurred. However, Maranon (2008) found that both the exponents of diatom and dinoflagellate do not differ from 1, partialy suggesting that phylogeney may not be critical for phytoplankton. Nevertheless, studies on natural phytoplankton assemblages remain scarce, and it is difficult to draw conclusion here.

The final concern is the assumption of no resource limitation in testing MTE (Brown et al., 2004). As aforementioned, nutrient limitation may not be a concern, because our incubations all received artificical nutrient amendments and our scaling analyses used only the growth rates measured with nutrient amendment (μ). For the issue of light limitation, our samples were taken from surface water (10-m depth) and incubated on deck to avoid light limitation, as was done in other studies (Maranon et al., 2007; Huete-Ortega et al., 2012; Maranon, 2008; McManus et al., 2007). Thus, light limitaiton effects on phytoplankton growth should not be a problem in our study. Our approach is consistent with previous studies (Landry et al., 1995). Nevertheless, we cannot completetly rule out the possibility of limitation, such as some trace metals."

In the studies of Maranon et al and Huete-Ortega et al, again the size range considered was much larger and the overall conclusion was that the size-scaling of phytoplankton metabolic rate is isometric (e.g. there is no overall size-dependence of growth).

Maranon et al and Huete-Ortega et al cover size range from 0.15 μm^3 to $5.2*10^5$ μm^3 (about ~0.2μm ESD to ~100μm ESD), while we cover from 10 to 300μm ESD. Our study cover more microphytoplankton but less nano- and pico- level individuals compared with their studies. Our result is really similar to that of the three studies on scaling of natural phytoplankton assemblages (Maranon, 2008; Maranon et al., 2007; Huete-Ortega et al., 2012), where those authors claimed it as isometric scaling relationship. The wording problem could be the key point resulting this comment.

The reported scaling exponents for individual-specific growth rate range from 1.03 to 1.32 in Huete-Ortega et al (2010), from 0.92 to 1.14 in Maranon et al (2007), and from 0.89 to 1.01 in Maranon (2008). The scaling exponent in our study is 1.092 (95% CI is 1.056 to 1.123 converted from size-specific to individual-specific growth rate; Table 1). Thus, our results are very similar to these three studies. The only difference is that they claimed it as isometric scaling, while we stated in a more assuring way, positive scaling. To avoid the confusion, we

have changed the subtitle in Results section (Sec. 3.1) into " Scaling of size-specific growth rate (μ) and mortality (m)" and the subtitle in Discussion section (Sec. 4.1) into " Scaling of size-specific growth rate (μ) and mortality (m)." We have also changed the wording to "*almost isometric*". See also our discussion in Sec. 4.2 in p3.

Therefore, none of the studies cited support the present paper's conclusion that growth rate increases with cell size in the microphytoplankton size range. In addition, virtually all laboratory studies published so far show that growth rates (or biomass-specific metabolic rates) decrease with increasing cell size in the microphytoplankton size range. A recent example can be seen in the study by Maranon et al. (2012) (Ecology Letters, DOI: 10.1111/ele.12052) which shows that the size-scaling of phytoplankton growth is in fact unimodal, which may be related to the size-dependence of nutrient uptake and use.

Although Maranon et al. (2012) report unimodal relationship, this study should be compared with our study with caution because of the following reasons. (1) This study used cultured phytoplankton instead of natural assemblages. The difference in scaling between lab culture and natural assemblage has been discussed in Maranon, 2008. The lab cultures tend to display higher scaling exponent than natural assemblages. (2) The nutrient uptake rate is still isometrically scaled on biovolume, while the maximum growth rate is not. The author thus provokes the geometric constraints to offer explanation. Comparison between their results and ours should be viewed with caution.

Again, we changed our wording to "almost isometirc" in the revision. Please also refer to our discussion to explain almost isometric (slightly positive) scaling exponents stated in Sec. 4.1 in p3.

The argument used by the authors to explain the positive size-scaling of growth rates is not convincing (page 12 lines 27-31). In this argument, and assuming that chlorophyll content is a proxy for metabolic rate, one would expect to find a positive size-scaling in the relationship between mass- (or volume-) specific chla content and cell size in order to explain a positive size-scaling of growth rate (or biomass-specific metabolic rate). However, at most, there are reports of isometric relationship between chla content and cell size (e.g. lack of size dependence in chla content). No reports exist, to the best of my knowledge, of increasing chla content (per unit biomass or cell volume) with cell size (the package effect makes this possibility extremely unlikely).

We have changed our wording to avoid the confusion. We DID NOT argue for positive scaling. See our response above (sec. 4.1 in p3).

In addition, the comparison between field and laboratory studies made by the authors is misleading (page 12 lines 27-31). The study of Finkel (2004) showed a strong allometric relationship between cell size and chla content (that is, volume-specific chla content decreased markedly with increasing cell size) because the cultures used were growing under strong light-limitation, which enhances the package effect, particularly in larger cells.

We agree with this comment. We have remove this part of discussion in revision. See our response above (set. 4.1 in p3).

In summary, the positive size-scaling of growth rate is rather hard to explain (for the size range considered by the authors) from a biophysical and physiological point of view. The authors should explore the possibility that this pattern may have resulted from some methodological artifact related to incubation, particle detection and volume

estimation by the FlowCam, data analysis, etc.

We agree that there may be potential artifacts in dilution experiments. We have explicitely caveated those in Discussion section (Sec. 4.3 in p8 above). The effacacy of FlowCAM has been shown in other studies.

A major conclusion of the study is that differential mortality of small versus large cells explains the variations in the slope of the NBSS. This argument is flawed: the authors did not find any sign of size-dependence in mortality. Within experiments, mortality is not related to cell size. Therefore, it is not correct to claim that (page 15) 'relatively higher growth rate of small versus large individuals serves as a trigger for higher grazing mortality of small than large individuals'. In fact, the authors did not observe higher mortality in small cells compared to large cells. If they had, the relationships between mortality rate and cell size would not be flat (Fig. 5).

We have clarified this. Please see our response in p1 and updated Figure 3 and Table B2. In summary, we found that the scaling exponent varies among stations, but generally does not approach -1/4 as MTE predicts (Fig. 3). Because the scaling varies among stations, we further investigated how this variation can contribute to affect the size structure (size spectral slope) **among assemblages**. To do so, we used simple linear regression analysis. We found that it is the relative mortality of small versus large individuals contributes most to determine the among-assemblage variations of size spectral slopes (Table 2). We have also clarified the reasoning and analysis in Methods. Sec. 2.5. in p4. We explicitly state that the exponent varies among stations:

"3.1 Scaling of size-specific growth rates (μ) and grazing mortality (m)

We find that the size-specific growth rate as well as size-specific grazing mortality scaling varies among stations (Fig. 3; Table B2). The average scaling exponent of μ for all 26 stations is 0.103, and the 95% confidence interval ranges from 0.040 to 0.164. The average scaling exponent of m for all 26 stations is 0.128, and the 95% confidence interval ranges from 0.040 to 0.219."

We also added the following discussion:

"*For the size-specific grazing mortality (m), the analyses for each station reveal substantial variation among assemblages (Fig. 3b). In addition, the result of GLMM suggests that on average m is slightly positively depends on body size (Table 1). In either case, our findings do not follow -1/4 scaling exponent as suggested by MTE. This is not suprising because MTE predicts -1/4 scaling exponent for intrinsic mortailty but not for extrinsic mortality. Again, the mortality estimated from our experiments mainly comes from grazing but not intrinsic processes. While the scaling of size-specific mortality varies among assemblage, the results of GLMM suggest a very small scaling exponent (close to 0), implying that grazing mortaility of microphytoplankton may be independent of body size. Independence of size-specific grazing mortality to body size might have implications on the scaling of phytoplankton total mortality rate. Previous meta-analysis indicates that the phytoplankton total mortality rate (including both intrinsic and extrinsic mortality) shows a -1/4 power relationship between size-specific mortality and body size (McCoy and Gillooly, 2008). Given that the grazing mortality is independent of body size, we suggest that the -1/4 scaling of total mortality versus body size of phytoplankton is to a large extent determined by the intrinsic processes. Our results*

suggest that the extrinsic processes (e.g. grazing) may be independent of body size and may not contribute significantly to affecting the scaling of mortality in microphytoplankton. However again, as the scaling exponent of size-specific mortality varies among assemblages, we are not certain that our results clearly support the independent of size-specific mortaility and body size."

The authors base their argument on the observation that, when pooling together data from all experiments, there is a positive relationship between mortality and growth rate. However, they key distinction here is that this is variability between sites/stations, not within stations. It is to be expected that in those stations where growth is faster (slower), mortality will also be faster (slower). This has nothing to do with size-related effects and does not prove anything. In fact, the dilution method itself has a built-in tendency to show covariation between mortality and growth, because the first variable is the slope and the second variable is the intercept of the same regression line. The higher the slope, the higher the intercept is likely to be, and vice-versa.

We agree that there may be a build-in tendency to observe tight coupling between growth rate and grazing mortality. However, we conduct two regression analyses to investigate the relationships among the three, body size, size-specific growth rate (μ') and grazing mortality (m). First we regress the size-specific grazing mortality (m) on size-specific growth rate (μ') using GLMM (stations as the random effect). Second, we regress the residuals from size-specific grazing mortality GLMM on the residuals from size-specific growth rate GLMM (please see the following figure) using linear regression. We have included these statements in Method section (Sec. 2.5.4) in our revised manuscript.

" 2.5.4 Coupling between size-specific growth rate and grazing mortality
To further clarify the relationship among microphytoplankton body size, size-specific growth rate, and size-specific grazing mortality, we regressed the size-specific grazing mortality against size-specific growth rate, using GLMM with stations as the random effect. Moreover, in order to partition out the effect of body size, we additionaly implemented a linear regression model to regress the residuals from size-specific grazing mortality GLMM (of size-specific rate versus size with stations as random effects) against the residules from size-specific growth rate GLMM. By doing so, we aim to examine if the microphytoplankton grazing mortality and growth rate are coupled together within an assemblage regardless of body size. Note, the growth rates used here are those measured without nutrient amendments (μ') so that we investigated the real situation in nature. Nevertheless, the analysis on μ reveals qualitative similar conclusion."

By doing so, we have partitioned out the effect from stations (spatial and temporal variation of growth and mortality per se) and body size (abundance). Our results showed both regressions are significant (Please see Fig. 4). Thus, we conclude that grazing mortality depend on growth rate within an assemblage. However, we still view this result with caution because we are not able to deal with the potential build-in artifact of dilution experiments.

The authors are confusing variability within the assemblage (changes in m or μ along the cell size range) with variability between assemblages (waters with higher μ are likely to have higher m, for all size classes). But in order to prove that size-differential mortality plays a role in the control of phytoplankton size structure, it has to be shown that size-related changes in mortality actually exist *within* a given assemblage. These

changes do not exist, therefore size-differential mortality cannot be a factor explaining changes in the slope of the biomass spectra.

Such within and between assemblage difference has been clarified and addressed in our previous response and has been included in Method and Discussion section in our updated manuscript. We have highlighted in the revision that size-related changes in mortality actually exist *within* some assemblages (Fig. 3).

Finally, the range of hydrographic conditions and phytoplankton biomass encountered by the authors was relatively narrow. Chl a concentration varied only by a factor of 3-4. It is very difficult, with such a small range of variability, to detect a significant oupling between the size-scaling of growth/mortality and the biomass size-structure. The biomass-size structure observed at one particular time reflects the integrale of many gain and loss processes which have taken place over the previous hours/days. In contrast, the size-scaling relationships for growth and mortality result from instantaneous measurements and reflect only present conditions. Rather than attempting to link (on a station-to-station basis) the variability between the size-scaling of growth/mortality and that of biomass, a more realistic objective would be to assess whether the predominance of particular size-class (generally over the whole studied area) is likely to have been caused by bottom-up of top-down process, e.g. driven by growth or mortality.

This is a great suggestion. We have now included additional analysis to show what the reviewer has suggested. See Sec. 2.5.6 in Methods (p6 above), Sec. 3.3 in Results (p7 above), and Sec. 4.2 in Discussion (p7-8 and 11 above).

Minor comments

Section 2.1. Sampling depths should be indicated. How many dilution experiments were conducted at each station and from which depths were water samples collected?

Our sampling depth is 10m, which is described in our revision. One dilution experiment was conducted at one station.

Page 11, lines 29 and rest of section. These sentences are quite confusing and difficult to follow.

This Result section has been modified. The section dedicated to describe the 9 model analyses to explain the NBSS slope (Sec. 3.3) is revised as following"

"3.3 Relative size-specific grazing mortality (m_S/m_L) explains the variation of the NBSS slope among assemblages

The results of our 9 univariate models indicate that the NBSS slope is only significantly related to the relative grazing mortality (m_S/m_L) and the relationship is positive ($p<0.05$, Table 2). We note that, if we had considered two-tail tests, the relative size-specific growth rate (μ_S'/μ_L') and the size-specific growth rate of small size category (μ_S') would show a significant positive relationship with the NB-SS slope; that is, a higher growth rate of small individuals causes the size spectral slope to be flattened. However, this is not possible biologically. Such spurious correlation simply arises due to the significant relationship between growth rate and mortality (Fig. 4) (See also Sect. 4.2). When using the four reduced data sets, the relative grazing mortality (m_S/m_L) remains the only significant variable explaining the NBSS slope (Table C2)."

Page 4, lines 24-25. 'high temperature favours the dominance of small phytoplankton'

As shown recently (Maranon et al L&O 2012, 57, 1266-1278) temperature *per se* plays a very minor role in the control of phytoplankton size-structure. Small cells dominate biomass and productivity whenever resources (light and/or nutrients) are in small supply and carbon fixation rates are low, irrespective of temperature. The association between temperature and size-structure arises because temperature is often correlated with nutrient availability in the sea.

We agree with this comment. However, from the manipulation experiments of warming on phytoplankton size structure by Yvon-Durocher et al. (2011), the phytoplankton NBSS slope did become steeper (dominance of small individual increase). Moran et al. (2010) also claim that the temperature along was able to explain 73% of the variance in the relative contribution of small cell (<2μm ESD) to total phytoplankton biomass regardless of the trophic status or nutrient loading. However, in our study, we found that nutrient is more important. See Disscusion Sec 4.2 above in p7-8.

Table in Appendix. Clarify if these are mean values obtained from the euphotic layer. The term 'integrated' is misleading. Are the irradiance values also mean values for the photic layer? Do they refer to mean values over the light period? A value of '0' is reported for PAR in one occasion – this must be incorrect.

The method to calculate the environmental measurements are stated in Method section (Sec. 2.2) as following,

" .... These environmental measurements for each station were integrated over the entire euphotic zone using discrete gradient integration method and the integrated average was used for analyses (Table A1)."

The zero value is changed to 0.01. Sorry for the typo.

Table 1. Results of the generalized linear mixed effect model (GLMM) linking microphytoplankton size-specific growth rate (μ) and grazing mortality (m) with microphytoplankton body size (biomass). In GLMM, stations were considered as random effects.

Cruise	Coefficient (95% confidence interval)	SE	p-value
Overall GLMM: $Log_2(\mu) \sim Log_2$(phytoplankton biomass) +random effect (station)			
Over all	0.092 (0.056, 0.123)	0.015	<0.001
Within cruise:			
May 2010	0.175 (0.037, 0.317)	0.056	0.006
Dec. 2010	0.175 (0.111, 0.233)	0.026	<0.001
Jun. 2011	-0.031 (-0.059, 0.002)	0.013	0.025
Jul. 2011	0.011 (-0.059, 0.080)	0.027	0.692
Aug. 2011	0.075 (-0.013, 0.130)	0.032	0.026
Sep. 2011	0.101 (-0.014, 0.213)	0.053	0.070
Oct. 2011	0.233 (0.160, 0.318)	0.035	<0.001
Overall GLMM: $Log_2(m) \sim Log_2$(phytoplankton biomass) +random effect (station)			
Over all	0.113 (0.054, 0.172)	0.030	<0.001
Within cruise:			
May 2010	0.132 (-0.104, 0.312)	0.093	0.169
Dec. 2010	0.122 (-0.060, 0.244)	0.078	0.133
Jun. 2011	0.055 (-0.051, 0.161)	0.055	0.328
Jul. 2011	-0.068 (-0.162, -0.000)	0.031	0.033
Aug. 2011	0.078 (-0.080, 0.256)	0.111	0.486
Sep. 2011	0.271 (0.087, 0.397)	0.074	0.001
Oct. 2011	0.357 (0.265, 0.482)	0.050	<0.001

Table 2. Results of univariate correlation analysis examining the relationship between NBSS slopes versus size-specific growth rates, mortality, grazing impacts, and the ratio of small versus large size category for these variables. The subscript (S or L) indicates the size category (small or large). μ' represents size-specific growth rates measured without nutrient amendment; m represents size-specific grazing mortality; I' represents grazing impact measured without nutrient amendment ($I'=m/\mu'$). Biological antipations represent the expected positive (+) or negative (−) relationship between each variable versus size spectral slopes, according to biological reasoning. The effect (coefficient) of each independent variable on NBSS slopes was tested against the biological anticipation using one-tail tests.

	Independent variables	Biological anticipation	Coefficient	p-value
Model 1	m_S	+	0.172	0.291
Model 2	m_L	-	-0.017	0.486
Model 3	μ_S'	-	0.238	0.988
Model 4	μ_L'	+	-0.040	0.115
Model 5	I_S'	+	-0.283	0.956
Model 6	I_L'	-	0.013	0.549
Model 7	m_S/m_L	+	0.348	0.026*
Model 8	μ_S'/μ_L'	-	0.593	0.999
Model 9	I_S'/I_L'	+	-0.023	0.722

* indicates the model that gives biologically reasonable and significant result.

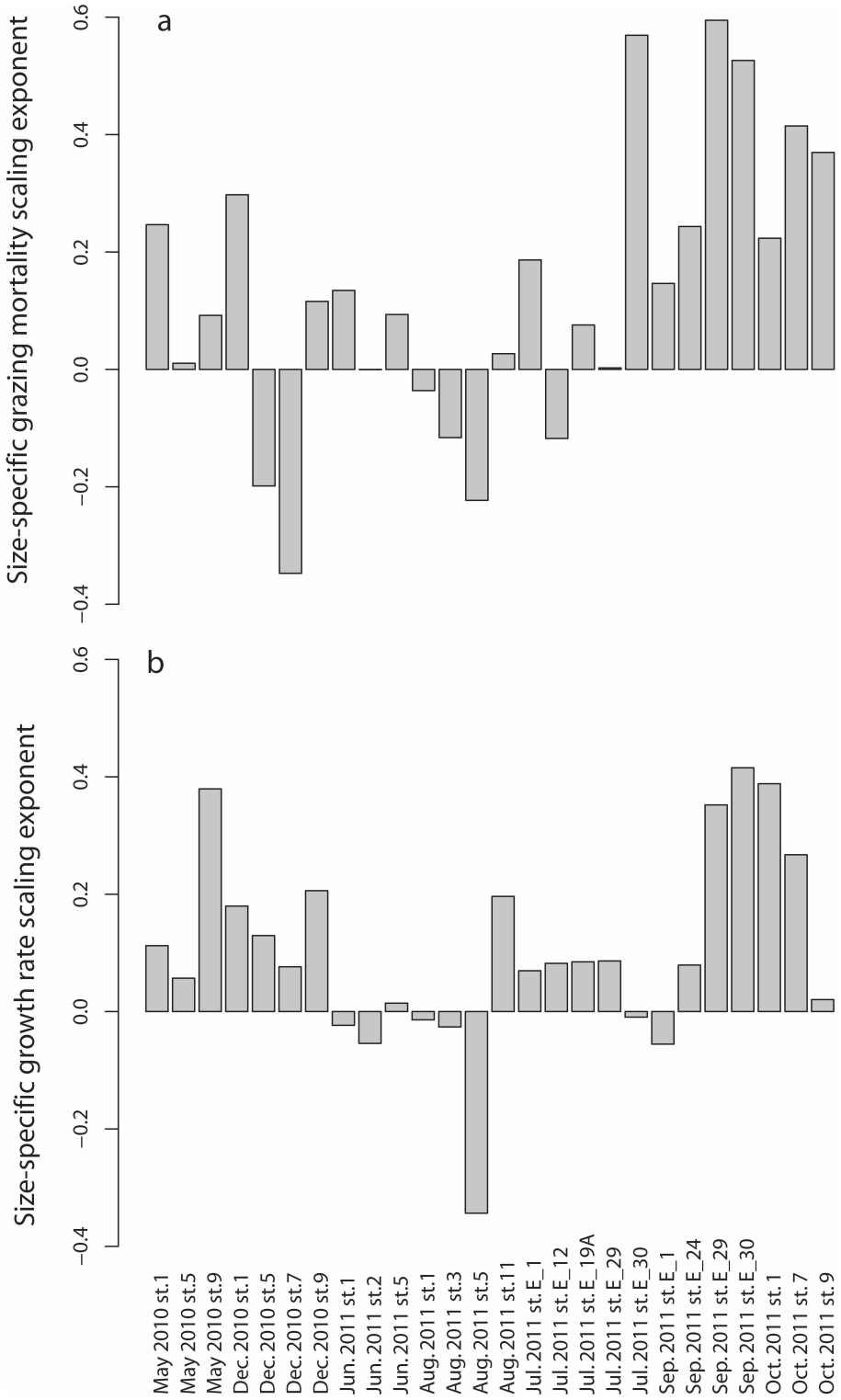

Figure 3. Barplot showing the scaling exponent of size-specific growth rate (a) and size-specific grazing mortality scaling (b) of each station. The average of growth rate scaling is 0.103 and the average grazing mortality scaling is 0.128. The bootstrapped 95% confidence interval is 0.040 to 0.164 for the growth rate scaling and 0.040 to 0.219 for the grazing mortality scaling.

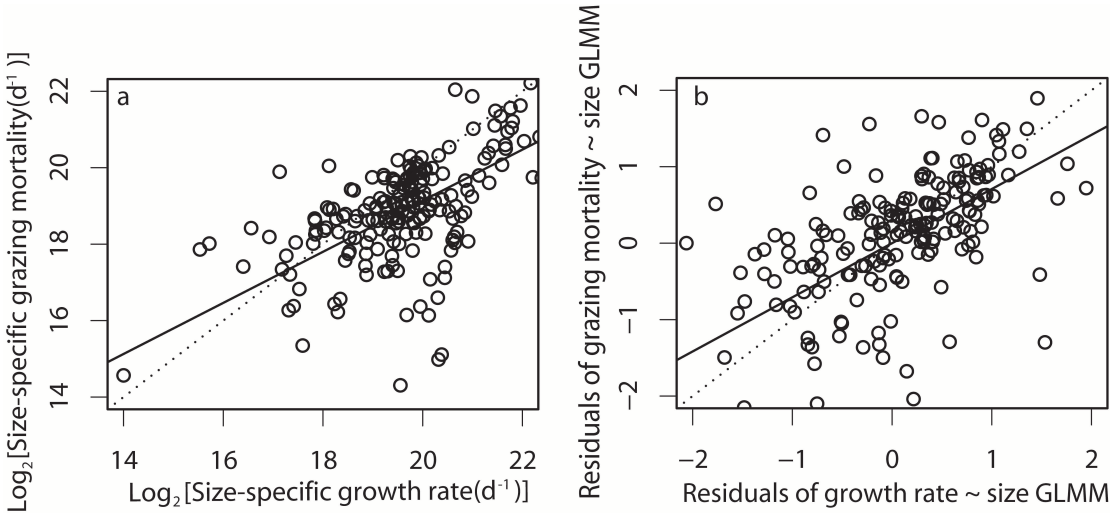

Figure 4. Scatter plot of log2 transformed size-specific grazing mortality (m) versus growth rate (μ'). Panel a shows the regression between size-specific grazing mortality and size-specific growth rate using GLMM (slope=0.668, p<0.001). Panel b shows the regression between the residuals from the size-specific grazing mortality GLMM against the residuals from the size-specific growth rate GLMM (slope =0.708, p<0.001). The solid line indicates the significant regression line, and the dashed line indicates the diagonal line.

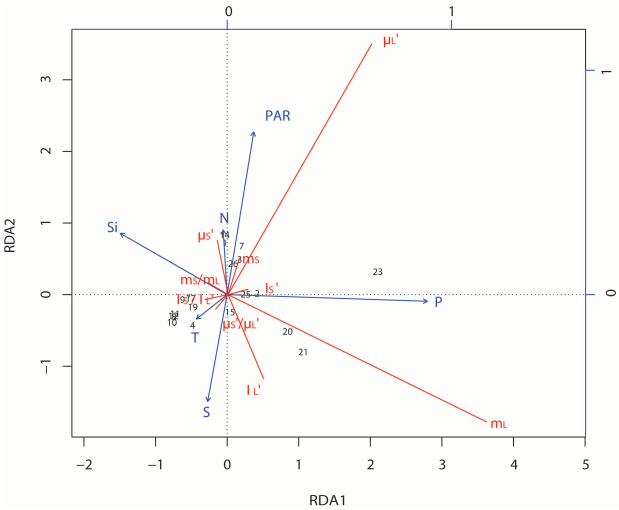

Figure 5. This RDA biplot showing the relationship between the 9 independent variables (red lines; Table 2) and the environmental factors (blue arrows; N: Nitrite + Nitrate concentration; PAR: Photosynthesis Active Radiation; P: Phosphate concentration; S: Salinity; Si: Silicate concentration; T: temperature). The bottom and left-hand scales are for the sampling stations (black numbers; Table A1) and the response variables (red lines), the top and right-hand scales are for the explanatory variables (blue arrows). The environmental factors (explanatory matrix) offer nearly significant explanation to the biological features (r=0.506; p=0.061). The first axis explains 46.13% and the second axis explains 2.39% of the variance. The first axis is associated with μ_L' and m_L and is mainly positively contributed by phosphate concentrations.

Supplement B. Biological data of the sampling stations.

Table B1. Summary statistics for the dilution experiments and Normalized Biomass Size Spectra (NBSS).

Cruise	Station	Average particles processed in T_0	Particle density (ind./ml)	Biomass (μg/L)	Average p-value of dilution exp. across size classes	NBSS slope	r^2 of NSSS
May 2010	St. 1	6168	2694	188.539	0.402	-0.727	0.854*
May 2010	St. 5	7423	3448	192.977	0.293	-0.664	0.865*
May 2010	St. 9	5742	1985	199.861	0.082	-1.064	0.785*
Dec. 2010	St. 1	6563	2248	349.784	0.462	-1.470	0.964*
Dec. 2010	St. 5	2868	1071	161.569	0.264	-1.233	0.922*
Dec. 2010	St. 7	670	253	317.033	0.179	-0.635	0.857*
Dec. 2010	St. 9	690	266	161.564	0.150	-0.785	0.888*
Jun. 2011	St. 1	2771	735	1091.136	0.177	-0.754	0.965*
Jun. 2011	St. 2	14286	587	815.715	0.248	-0.570	0.665*
Jun. 2011	St. 5	443	130	763.249	0.074	-0.458	0.882*
Aug. 2011	St. 1	1669	513	1417.715	0.118	-0.673	0.953*
Aug. 2011	St. 3	1381	369	2252.591	0.085	-0.497	0.923*
Aug. 2011	St. 5	255	76	390.693	0.167	-0.533	0.846*
Aug. 2011	St. 11	468	169	76.721	0.128	-0.834	0.943*
Jul. 2011	St. E_1	220	65	75.035	0.205	-0.659	0.912*
Jul. 2011	St. E_12	71	28	27.098	0.290	-0.540	0.813*

Jul. 2011	St. E_19A	1364	547	384.590	0.183	-0.880	0.944*
Jul. 2011	St. E_29	922	369	349.928	0.068	-0.851	0.893*
Jul. 2011	St. E_30	9302	2939	4334.308	0.521	-0.813	0.882*
Sep. 2011	St. E_1	296	111	68.792	0.195	-0.644	0.913*
Sep. 2011	St. E_24	171	61	72.329	0.169	-0.675	0.860*
Sep. 2011	St. E_29	1299	450	299.164	0.460	-0.798	0.905*
Sep. 2011	St. E_30	3513	1243	281.763	0.089	-1.150	0.895*
Oct. 2011	St. 1	3520	1205	279.075	0.330	-1.190	0.874*
Oct. 2011	St. 7	946	342	105.449	0.224	-1.099	0.962*
Oct. 2011	St. 9	1206	443	151.017	0.373	-1.183	0.995*

*indicates significant NBSS slope

Table B2. Size-specific growth rate and grazing mortality scaling on body size for each sampling station.

Cruise	Station	μ scaling slope	SE of μ scaling slope	p-value of μ scaling slope	m scaling slope	SE of m scaling slope	p-value of m scaling slope
May 2010	St. 1	0.112	0.053	0.077	0.247	0.150	0.151
May 2010	St. 5	0.057	0.123	0.667	0.010	0.266	0.971
May 2010	St. 9	0.379	0.091	0.006	0.092	0.036	0.042
Dec. 2010	St. 1	0.180	0.019	<0.001	0.298	0.133	0.066
Dec. 2010	St. 5	0.129	0.045	0.036	-0.199	0.351	0.596
Dec. 2010	St. 7	0.076	0.046	0.159	-0.347	0.185	0.119
Dec. 2010	St. 9	0.206	0.066	0.014	0.116	0.060	0.089
Jun. 2011	St. 1	-0.023	0.030	0.461	0.135	0.067	0.081
Jun. 2011	St. 2	-0.054	0.009	<0.001	0.000	0.117	0.997
Jun. 2011	St. 5	0.014	0.021	0.525	0.094	0.060	0.170
Aug. 2011	St. 1	-0.014	0.020	0.485	-0.036	0.040	0.393
Aug. 2011	St. 3	-0.026	0.022	0.267	-0.116	0.064	0.100
Aug. 2011	St. 5	-0.344	0.209	0.200	-0.223	0.167	0.275
Aug. 2011	St. 11	0.196	0.048	0.010	0.027	0.026	0.344
Jul. 2011	St. E_1	0.070	0.169	0.702	0.187	0.113	0.175
Jul. 2011	St. E_12	0.082	0.015	0.005	-0.118	0.049	0.073
Jul. 2011	St. E_19A	0.085	0.052	0.151	0.076	0.141	0.611

Jul. 2011	St. E_29	0.086	0.057	0.182	0.003	0.051	0.961
Jul. 2011	St. E_30	-0.010	0.067	0.894	0.569	0.577	0.380
Sep. 2011	St. E_1	-0.056	0.084	0.534	0.147	0.107	0.220
Sep. 2011	St. E_24	0.079	0.055	0.193	0.244	0.087	0.026
Sep. 2011	St. E_29	0.352	0.144	0.092	0.595	0.355	0.192
Sep. 2011	St. E_30	0.415	0.130	0.085	0.526	0.111	0.041
Oct. 2011	St. 1	0.388	0.030	<0.001	0.223	0.129	0.145
Oct. 2011	St. 7	0.267	0.032	<0.001	0.415	0.081	0.001
Oct. 2011	St. 9	0.021	0.101	0.852	0.370	0.089	0.026

Supplement C. Results of four reduced data sets.

Table C1. Results of reduced data sets showing the general scaling relationship of size specific growth rate (μ) and grazing mortality (m) versus body size. In addition, the coupling of grazing mortality and growth rate is also examined. All the four reduced data sets show qualitatively consistent results as the whole data set.

Reduced data set 1	Estimate	SE	p-value	Reduced data set 2	Estimate	SE	p-value
μ - size GLMM	0.094	0.015	<0.001	μ - size GLMM	0.094	0.017	<0.001
m - size GLMM	0.137	0.018	<0.001	m - size GLMM	0.124	0.026	<0.001
m - μ GLMM	0.777	0.086	<0.001	m - μ GLMM	0.865	0.095	<0.001
residuals of m - size GLMM~ residuals of μ - size GLMM	0.441	0.065	<0.001	residuals of m - size GLMM~ residuals of μ - size GLMM	0.359	0.045	<0.001
Reduced data set 3	Estimate	SE	p-value	Reduced data set 4	Estimate	SE	p-value
μ - size GLMM	0.090	0.017	<0.001	μ - size GLMM	0.078	0.018	<0.001
m - size GLMM	0.138	0.019	<0.001	m - size GLMM	0.091	0.025	<0.001
m - μ GLMM	0.778	0.092	<0.001	m - μ GLMM	0.798	0.098	<0.001
residuals of m - size GLMM~ residuals of μ - size GLMM	0.480	0.073	<0.001	residuals of m - size GLMM~ residuals of μ - size GLMM	0.382	0.050	<0.001

Table C2. Results of univariate model analyses in the four reduced data sets. The results here are qualitatively the same as the results of the whole data set.

	Independent variables	Biological anticipation	Reduced data set 1		Reduced data set 2		Reduced data set 3		Reduced data set 4	
			Coefficient	p-value	Coefficient	p-value	Coefficient	p-value	Coefficient	p-value
Model 1	m_S	+	0.024	0.654	0.197	0.337	0.193	0.611	-0.006	0.515
Model 2	m_L	-	-0.073	0.082	-0.019	0.492	-0.045	0.090	-0.028	0.163
Model 3	μ_S'	-	0.163	0.997	0.342	0.990	0.245	0.924	0.196	0.928
Model 4	μ_L'	+	-0.090	0.929	-0.049	0.950	-0.077	0.995	-0.062	0.999
Model 5	I_S'	+	-0.057	0.909	-0.302	0.951	-0.257	0.904	-0.249	0.910
Model 6	I_L'	-	-0.051	0.367	0.016	0.552	-0.047	0.748	0.023	0.598
Model 7	m_S/m_L	+	0.085	0.035*	0.403	0.027*	0.415	0.061	0.277	0.058
Model 8	μ_S'/μ_L'	-	0.149	1.000	0.698	0.999	0.745	0.999	0.556	0.999
Model 9	I_S'/ I_L'	+	-0.028	0.736	-0.072	0.912	-0.097	0.866	-0.022	0.745

* indicates the model that gives biologically reasonable and significant result.