

## ***Interactive comment on “Phosphate supply explains variation in nucleic acid allocation but not C : P stoichiometry in the Western North Atlantic” by A. E. Zimmerman et al.***

**Anonymous Referee #2**

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Zimmeman et al.'s manuscript. “Phosphate supply explains variation in nucleic acid allocation but not C:P stoichiometry in the Western North Atlantic” investigates the relationship between biochemical allocation strategy and nutrient supply in open ocean communities to determine the drivers of ecosystem-scale variability in the particulate element ratios of surface waters. They hypothesize that SRP supply is directly related to RNA concentration and in turn drives lower C:P ratios of biomass. This is an attempt to test the growth rate hypothesis (GRH) put forward by Elser et al. (2002) and also Sterner and Elser (2002). The GRH relates cellular stoichiometry to internal pools of macromolecules. In particular, the P content of a cell is driven by the cellular concentration of ribosomal RNA, which is closely related to cellular growth rate. Thus, the

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GRH predicts that when growth rate increases ribosomal RNA will increase in a cell and then C:P of the cell will decrease. In the present manuscript, the authors expected to see higher RNA, RNA:DNA ratios, and a higher proportion of P allocated to RNA and total P biomass, but lower C:P ratios, where SRP fluxes were higher. They conducted their study in the oligotrophic North Atlantic Subtropical Gyre along a north to south gradient (35.67°N – 22.67°N) of P-supply. What they found was that C:P ratios were not related to nucleic acid content of the cells. However, RNA content was related to P supply rate. While I find this to be an interesting and valuable study, I feel the authors need to address a few things prior to the manuscript being acceptable for publication.

1. The authors are essentially trying to test the GRH without measuring growth rate. The GRH relates elemental stoichiometry and macromolecular content through growth rate. It is unclear that growth rate differs along the environmental P supply gradient. In the North Atlantic, P-supply does not necessarily correlate with growth rate and so I am not sure I would expect to see the difference the authors expect to see. Perhaps N is limiting growth (there is quite a bit of evidence that shows autotrophs in the N. Atlantic subtropical Gyre are N-limited or NP co-limited) and C:P is related to N-flux.

2. The SRP flux is calculated from the gradient of SRP concentration between 80m (or deeper) and 5m isn't it? There is no presentation of mixed layer depths or mixing rates that might convince the reader that this SRP even gets to the cells at 5m. Perhaps P turnover rates are more important than deep SRP.

3. The SRP flux gradient is driven by two stations north of 32°. South of 32°N there is little gradient. Additionally, there is little gradient in SRP or PPhos concentrations south of 32°N. However, DOP concentrations decrease >2 fold from the most southern station to 32°N. It seems like utilization of DOP in this part of the transect may be more important than flux from below.

4. On page 16306 line 15 the authors state that both POC and DNA concentrations increased with SRP flux along the transect, suggesting greater SRP supply from deep

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water increased total biomass in the surface waters. This is then used as support for P-limitation of the organisms in these waters. However, this greater P supply brings with it a greater N supply (NO<sub>3</sub> in the deeper water) and it can not be used to indicate the limitation status of the cells. Likewise, the RNA:DNA ratio may be a potential proxy for growth rate (as the authors state). Thus the higher RNA:DNA ratio along the transect can only say that growth rate was higher but nothing about the P-limitation status of the cells (could be the N stimulating growth).

5. There is a weak relationship between the RNA:DNA ratio and latitude (from Table 1,  $r^2=0.34$  and  $p = 0.59$ , not significant). If the RNA:DNA ratio were a proxy for growth rate (as the authors suggest in their discussion) this suggests there was no significant (or a small) change in growth rate across the transect. This being the case, would you expect to see a change in C:P due to RNA changes? According to the GRH I do not think you would.

6. Lastly, what is the detrital content of the particulate matter? If detrital matter is high couldn't this drive the particulate C:P variability and any impact of RNA may not be seen?

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