

Interactive comment on “Alternation of heterotrophic bacterial and archaeal production along nitrogen and salinity gradients in coastal wetlands” by Gema L. Batanero et al.

Anonymous Referee #1

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This paper presents the bacterial and archaeal abundance and heterotrophic production in nine coastal wetlands. Based on Generalized linear models they conclude to switch from heterotrophic bacterial production towards heterotrophic archaeal production as salinity and virus abundance increased. This topic is very interesting in a context of global change. But in my opinion the conclusions are very speculative and based only on linear models between productivity and salinity or viral load. I am not a modeler, but the use of GLP must be justified and statistics must be provided. The experimental methods used are also to be discussed.

Material and Methods Part In my opinion different methods are not very suitable: 1>

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Line 145: We obtained the heterotrophic prokaryotic abundance (HPA) by subtracting the cyanobacteria abundance (CyA) from prokaryotic abundance (PA). Cyanobacteria are not the only autotrophic organisms, there are also, for example, nitrifiers. What is the percentage of cyanobacteria? what is the objective in this paper to limit itself to present the HPA numbers? what is the experimental error in the quantification in comparison with the numbers of cyanobacteria? The authors probably wanted to link the number of heterotrophic organisms and their productivity. this is always tricky because the count concerns the total number of organisms without information on the active fraction. 2> line 146: Virus abundance With this protocol and depending on the cytometer used (not specified, but I imagine it is a Beckton Dickinson (BD)), the authors will only see particles >50nm in size and DNA vrus. So they should not say in the text that they will have the actual abundance (since there are many smaller DNA viruses and also RNA viruses).

3> The use of erythromycin to discriminate bacterial versus archaeal production should be discussed. Erythromycin inhibits the growth of bacteria by interfering with protein biosynthesis. It binds with the 50S ribosomal subunit and thus prevents the translocation of peptides and the formation of polypeptides.

efficiency of EMY are related to medically relevant organisms (e.g. *Staphylococcus aureus*) and do not consider natural prokaryotic assemblages. It is important to note, however, that all other studies concerning the efficiency of EMY are related to medically relevant organisms (e.g. *Staphylococcus aureus*) and do not consider natural prokaryotic assemblages. Horizontal gene transfer and/or mutations of ribosomal binding sites might alter the susceptibility to EMY in archaeal and bacterial species For Frank 2016, The addition of EMY reduced the bulk leucine incorporation by ~77%. Evaluation of the inhibition efficiency of EMY on a cell-specific level showed no difference between Archaea ($76.0 \pm 14.2\% [SD]$) and Bacteria ($78.2 \pm 9.5\%$). Their results suggest that in complex open-ocean prokaryotic communities EMY is efficient as a domain-specific inhibitor Line 160: “it appears to have better efficiencies (ca. 80%) in water of higher

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salinity and for specific functional groups as nitrifiers, particularly Firmicutes" I don't understand this sentence that needs to be rephrased, nitrifiers are not included in the firmicutes phylum. It is also necessary to qualify this statement because the authors also mention adaptation and resistance to EMY.

Results part

The results are presented in their entirety by integrating the entire dataset obtained from all 12 sites. For each site there is a strong salinity gradient and also a great heterogeneity in the bacterial numeration and production. Before integrating the whole dataset into a GLM model, the data could be presented and analyzed by station et compared.

Line 215 : significantly , can you give a p-value?

Discussion

Do you have a hypothesis to explain from a physiological point of view the effect of TDN on the switch from heterotrophic bacterial to archaeaous production?

Then the discussion turns to nitrification by archaea, I don't understand the connection since nitrifiers are autotrophic organisms

Line 270 : In our study, ammonia oxidation by archaea during nitrification likely is not a significant process due to the high concentrations of dissolved nitrogen in most wetlands: I don't understand this part of the discussion then nitrifiers are aerobic and except in atypical pathways they need oxygen to achieve nitrification.

More generally, there are only TDN data (including the concentrations of the different organic and inorganic nitrogen forms as well as nitrate, ammonium) and the discussion focuses on the transformation processes between the different oxidation states such as nitrification and denitrification. This seems to me very speculative

the authors state in the final lines of the conclusion: Archaea appeared to be the main

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prokaryotes processing nitrogen in the most saline wetlands, I think this is based on a positive correlation between TDN and heterotrophic production by the archaea

Is there a cross-effect between DDT and salinity?

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