

Interactive
Comment

Interactive comment on “Effects of seawater <i>p</i>CO₂ changes on the calcifying fluid of scleractinian corals” by S. Hohn and A. Merico

Anonymous Referee #1

Received and published: 23 April 2012

General comments:

The authors present a model of coral calcification that aims to simulate how coral calcification responds to rising CO₂. The model computes the kinetic reactions of carbonate chemistry and the flux of ions in and between four different model compartments: the seawater, the coral tissue, the coelenteron and the calcifying fluid. A model such as this is potentially very valuable as it attempts to provide a physiological, mechanistic explanation to why corals calcify more slowly under ocean acidification. As the authors point out, corals exert significant biological control over physio-chemical conditions at the site of calcification, so why changes in seawater chemistry (the growth medium)

Full Screen / Esc

Printer-friendly Version

Interactive Discussion

Discussion Paper



should effect the calcifying fluid remains an important question in ocean acidification research. The authors have constructed their model in such a way that it reproduces measurements of Ca^{2+} and pH in the calcifying fluid made by microelectrodes under seawater conditions of pH8.2 (Al-Horani et al. 2003. Mar Biol). The microelectrode data of Al-Horani is the only set of continuous light-dark data currently available that provides the kinetics necessary with which to test the model. Whether one agrees with the set up of model or not, it integrates a certain amount of what is known about the physiology underlying coral calcification and provides a useful platform with which to generate and investigate hypotheses about the mechanisms underpinning coral calcification. Although the overall ideas and approach underlying the model potentially make it a valuable contribution to the literature, there are some issues related to fundamental physiology that could be be addressed/ improved. The biggest concern relates to the values of tissue pH which are currently way above the range of physiological norms for any animal, including coral. I discuss this issue further in specific comments. Other issues relate to confusing terminology and underrepresentation of the literature, which are also discussed in my specific comments. I hope these issues can be addressed in a revised paper.

Specific comments:

Title: The title is a bit misleading. The authors carry out pCO_2 perturbation experiments with the model, but they only show how this effects calcification. They do not give a figure that shows changes in the calcifying fluid under pCO_2 perturbation. The title should be modified.

P. 2656, line 5: Not all projections of coral reef futures agree with this (see recent publication by McCulloch et al. 2012 Nature Climate Change. doi:10.1038/nclimate1473). It may be better to say that OA acting with other anthropogenic stressors is expected to cause disastrous effects on reef ecosystems.

P. 2656, line 10: the term “calicoblastic layer” used for the fourth compartment is mis-

[Full Screen / Esc](#)[Printer-friendly Version](#)[Interactive Discussion](#)[Discussion Paper](#)

leading. “Calicoblastic” refers to a cell type that makes up the aboral ectoderm, so “calicoblastic layer” could be mistaken for this epithelium. The authors are referring to the “subcalicoblastic layer or medium”, the term used by Al-Horani et al. 2003 (on whose data the model is based), Venn et al. 2011. PLoS ONE 6(5): e20013. doi:10.1371/journal.pone.0020013, and others who have worked on this layer. Elsewhere the authors use calcifying fluid and they should stick this term or use subcalicoblastic layer/ medium.

P. 2658. The authors explain how corals metabolically control the composition of the calcifying fluid. Because the model isn't purely geochemical and attempts to integrate coral physiology, a more detailed and more accurate introduction of the physiology underlying how corals modify the calcifying fluid is needed here. It would be helpful to the non coral specialist reader in evaluating the model. Of course, not every aspect of coral physiology can reasonably be included in the model, but the authors can explain what is and isn't included in the next section, “model description”.

As the model deals with coral tissue it would be relevant to point out here that the calcifying fluid is separated from the surrounding seawater by four cell layers, an oral ectoderm and endoderm and an aboral endoderm and ectoderm, (the latter cell layer being the calicoblastic cell layer). It should be mentioned that evidence exists for both paracellular and transcellular passage of ions and molecules from the seawater (including Ca^{2+}) to the calcifying fluid (e.g. Tambutte et al. 2011, Proc R Soc B, doi: 10.1098/rspb.2011.0733). (The authors only consider the transcellular route and they can state this in the next section (model description). The authors should mention (briefly) that corals produce an organic matrix from the calicoblastic cells at the tissue-skeleton interface (reviewed recently Tambutte et al. 2011, J Exp Mar Biol Ecol. Volume 408, Issues 1–2, Pages 58–78). Also, the authors should mention that the calicoblastic epithelium secretes enzymes e.g. carbonic anhydrase that facilitate the interconversion of the dissolved inorganic carbon species. Again it can be stated that for simplicity the addition of organic molecules to the calcifying fluid is not considered in the present

[Full Screen / Esc](#)[Printer-friendly Version](#)[Interactive Discussion](#)[Discussion Paper](#)

model. The authors already discuss how pH is elevated in the subcalicoblastic medium (although they should perhaps reference pH imaging of the subcalicoblastic medium by Venn et al. 2011 PLoS ONE 6(5): e20013. doi:10.1371/journal.pone.0020013) and the potential role of the proton-calcium antiporter. Here it would be better to introduce that evidence for a Ca²⁺ ATPase comes from the biochemical study by Ip et al. 1991, Mar Biol. and molecular evidence by Zoccola et al. 2004, Biochim, Biophys Acta, rather than later in the “model description”. Lastly, the authors do not mention the existence of a diffusive boundary layer (DBL) between the coral tissue and the surrounding seawater that influences the exchange of ions between the coral tissue, coelenteron and the growth medium. There are quite a few papers that describe the DBL (e.g. Shashar N, et al. 1993. Biol Bull 185: 455–461., Mass T, et al. Proc Natl Acad Sci U S A 107: 2527–2531). Ok, as previously stated not everything can be included in the model, the task is daunting, but at least the reader should know what important aspects of coral physiology are and are not included in the model.

P. 2658, line 24. The four compartments proposed by the authors do not relate to the 4 compartments identified by Tambutte et al. 1996.

P. 2659- model description. As stated above the authors do not consider a paracellular pathway. This is a potential route by which seawater bicarbonate and carbonate reach the site of calcification. The authors already clearly state they are only considering CO₂ diffusivity over the membranes and transcellular bicarbonate transport, but they should at least introduce the paracellular pathway earlier in the introduction and say it is not included in this model.

P. 2659. If no paracellular pathway is being considered, why does figure 1 show a direct connection between the coelenteron and the calcifying fluid (termed calicoblastic layer in the figure) that does not pass via the tissue?

P. 2663. Results, tissue. The model does not incorporate reasonable levels of intracellular pH. In the model, pH in the tissue varies between about 8.3 and 9.3 which are a

[Full Screen / Esc](#)[Printer-friendly Version](#)[Interactive Discussion](#)[Discussion Paper](#)

long way above physiological tissue pH of animals and plants. Unlike calcium, which the authors state cannot be constrained due to lack of concentration data, the range of intracellular pH in the light and dark has been characterized in coral cells from the endoderm layer (pH 7.1–7.4) and the calicoblastic layer (pH 7.4 in the light and dark) (Venn et al. 2009, PNAS. 106(39):16574–9. and Venn et al. 2011. PLoS ONE. e20013. doi:10.1371/journal.pone.0020013). A realistic physiological model must incorporate values of pHi at least somewhere close to these values.

P. 2664. Section 3.5. Calcification over time. The authors should remark that no calcification is observed in the dark and this doesn't really represent the biology very well as numerous studies show that corals continue to calcify in the dark. Estimations of the degree to which light "enhances" calcification vary, but are reviewed in Tambutte et al. 2011, J Exp Mar Biol Ecol. Volume 408, Issues 1–2, Pages 58–78.

P. 2666. Line 27. Although the authors state that little data exists for calcium concentrations in the tissue, they state that the pathways for calcium transport are relatively well known. This isn't really the case and the references the authors give only relate to molecular and invitro characterization of the Ca²⁺+Atpase in the calcioblastic cells. The pathway of calcium from seawater through the tissue is not well understood. It would probably be better to remove this phrase.

P. 2670. Line 17. It would help the non coral specialist reader if the authors provided one of the comprehensive reviews on coral bleaching e.g. Douglas 2003. Marine Pollution Bulletin 46, 385–392.

Interactive comment on Biogeosciences Discuss., 9, 2655, 2012.

[Full Screen / Esc](#)[Printer-friendly Version](#)[Interactive Discussion](#)[Discussion Paper](#)