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Biogeosciences Discussions

Interactive comment on "A new conceptual model of coral biomineralisation: hypoxia as the physiological driver of skeletal extension" *by* S. A. Wooldridge

Anonymous Referee #1

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Wooldridge presents an interesting hypothesis, that hypoxia could be a primary driver of skeletal extension in corals. I believe this warrants publication as it provides an intriguing line of discussion. However, prior to publication there are a number of issues that need to be addressed.

It is not clear to me that the layered growth structure of a coral is a new discovery – Ogilvie 1896 clearly described a layered structure of growth lamellae surrounding dark points which seems to correspond to centers and banded fibers.

The regions in which the highest rates of skeletal extension occur are the most exposed regions of the skeleton – septal tips, apical polyps, etc – all regions with relatively low

densities of zooxanthellae and most exposed to water movement, thus the regions in which hypoxia is least likely to occur. This disconnect poses a significant issue for the proposed model as I understand it and should be addressed, as it seems to me the regions with highest oxygen tension extend the fastest, thus hypoxia could be argued to repress extension. The author argues that the high metabolic activities in these regions could drive hypoxia and thus extension, but it seem the reverse is more likely – higher oxygen tensions driving higher metabolic rates and extension.

The role of zooxanthellae in driving this process is further complicated by a lack of a difference in the respiration rates of some species of corals in the presence or absence of symbionts.

The lack of data supporting oxalate in the skeleton poses a problem, it should be clear as to whether the author considers extension to be primarily the product of oxalate crystals or what their precise role is in the process – is this a stepped process – oxalate formation, then carbonate precipitation surrounding the oxalate (and if this is the case, why would it be the case when there is already an extensive aragonite skeleton which would presumably be a suitable site for further crystal growth with no need for another phase as the nucleation site, and why is carbonate formation shut down – particularly given that pH remains elevated which would presumably support continued carbonate deposition – indeed cyclic saturation states have been previously suggested to account for the layered growth of corals)? or simultaneously occurring processes with the precipitation of mixed carbonates/oxalates – and if this is the case, why is the oxalate formation considered critically important as opposed to a contaminating phase which cycles temporally leading to compositional variations? Is the oxalate thought to be lost during calcification as suggested on page 12639? or does it remain in the skeleton? Would azooxanthellate corals be expected to have consistently higher oxalate contents given they lack a daytime increase in internal pO2?

It is unclear to me how this model provides new insight as to the fitness of different symbionts (pg 12641). Calcification is but one aspect of coral physiology, and its utility

is always limited; in any measure of symbiont fitness, the translocation of materials between the host and symbiont must be considered as well as their ultimate fates, though the difficulty of the latter often leads to parameters such as calcification being used as an imperfect proxy – this has always been the case regardless of the calcification model.

The evidence presented for this model in understanding for the effects of ocean acidification and the cambrian explosion is rather weak – I suggest eliminating these sections or substantially rewriting them.

Interactive comment on Biogeosciences Discuss., 9, 12627, 2012.