

## ***Interactive comment on “Mass extinctions past and present: a unifying hypothesis” by S. A. Wooldridge***

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Thankyou to the reviewers who have posted comments in response to the paper. These comments help to clarify the necessity for formal testing of the urease hypothesis before it can be totally accepted or indeed rejected. In this statement lies a challenge to the scientific community. Here, I will attempt to contribute to this effort by commenting on two specific criticisms of the hypothesis that have been raised, namely (i) the distribution of urease among eukaryotes, and (ii) the pH sensitivity of urease.

1. Distribution of urease among eukaryotes. In a prepared review document (urease\_invertebrates.pdf) that is available at my anonymous ftp site (ftp://ftp.aims.gov.au/pub/swooldri/) I provide references to various studies that confirm urease activity in some ~30 invertebrate species; including representatives from Mollusca, Cnidaria, Bracipoda, Athropoda, Echinodermata, Annelida, and Platy-

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helminthes. In a good number of these studies, including the three most recent undertaken in 1997-2000, the authors comment that these ureases are of animal origin and are not due to bacterial contamination.

2. pH sensitivity of urease. It is true that the urease hypothesis weighs heavily on the interpretation of the observations of Barnes and Crossland (1976). In this study, the authors investigate the pH optima for urease activity in the endosymbiotic algae (zooxanthellae) and the coral host pertaining to the staghorn coral, *Acropora acuminata*. They demonstrate that the different buffer types they use for extraction and assay affect the pH optimum for urease activity. In my interpretation, I have attributed this response to the potential existence of two functionally different ionisable groups of differing pKa's; which were separately characterised by the different buffer types. In this case, the two different ionisable groups promote optimal activity at pH 7.6 or 8.1. Whilst I understand that this does not necessarily confirm that an enzymatic dead-zone exists between these pH optima, I strongly believe that it alludes to its potential existence. Admittedly, further testing is required to strengthen this belief. Based on prior inference (as outlined in Wooldridge, submitted *Geochim. Cosmochim. Acta*) I had good reason to suspect that urease activity is restricted in scleractinian corals at ~pH 7.9; and that this inactivation aids (is evidenced by) skeletal dissolution (due to the resultant build-up of glyoxylate / glyoxylic acid at the site of calcification).

So what would the evidence look like if my interpretation of the urease pH activity profile was correct? Firstly, there would be two distinct calcification optima at both pH 8.1 and 7.6. Secondly, there would be reduced calcification (including carbonate dissolution) at pH 7.9. In a prepared document ([urease\\_pH\\_evidence.pdf](#)) that is available at my anonymous ftp site (<ftp://ftp.aims.gov.au/pub/swooldri/>) I provide evidence from 12 recent studies which strongly support the expected biological responses at pH 7.9 (and 8.2 / 7.6), and which cannot be solely ascribed to the carbonate ion saturation state (even though I don't deny the significance of this process). I contend that my interpretation of the pH profile for urease cannot be so easily dismissed.

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## References:

Barnes, D. J., and Crossland, C. J.: Urease activity in the staghorn coral, *Acropora acuminata*, *Comp. Biochem. Physiol.*, 55B, 371-376, 1976.

Wooldridge, S. A. (submitted) A dual-enzyme biomineralisation model for interpreting the information stored within coral skeletons: I. Key processes and supporting evidence, *Geochim. Cosmochim. Acta.*, 2008.

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Interactive comment on *Biogeosciences Discuss.*, 5, 2401, 2008.

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