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Interactive comment on "Ocean acidification effects in the early life-stages of summer flounder, *Paralichthys dentatus*" *by* R. C. Chambers et al.

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The study by Chambers et al, "Ocean acidification effects in the early life-stages of summer flounder", investigates the effects of elevated CO2 levels on diverse parameters related to larval survival, growth, and morphological development. This is the first study of its kind to address the effects of elevated CO2 on an economically and ecologically important fish in the U.S. mid-Atlantic region. The study very clearly and convincingly demonstrates that the effects of elevated CO2 on this species include decreased survival at hatch, increased larval size, decreased yolk size, and increased cranial-facial malformations. My comments on this very interesting study come from the reductionistic perspective of a physiologist.

1) Like most other studies of its kind, there is an assumption that the physiological

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effects of elevated CO2 on fish development are mediated exclusively through ocean acidification, which imparts an acid-base imbalance on the organism. Although this may well be true, it is also plausible that, in addition to altering pH, elevated CO2 may affect fish **directly** via non-pH altering mechanisms. Specifically, CO2 is known to directly bind to hemoglobin and change its conformation, which reduces its affinity to O2 (i.e. the “Haldane” effect, which is distinct from the “Root” effect that relies on CO2-based changes in pH). The only way to truly dissociate the direct effects of CO2 from the indirect acidifying effects of CO2 would be to acidify the water without using CO2, and see if this produces similar results. I am not suggesting that the authors do this, but I do wonder the extent to which this study (and others) is truly measuring the effects of ‘ocean acidification’ on development. Might it be more accurate for the title to read “the effects of elevated CO2 levels on early life-stages of summer flounder”?

2) Regarding the fascinating observation that larvae were larger and with smaller yolk at higher CO2 compared with controls, I think the authors are assuming that larger sizes specifically result from accelerated growth rates (e.g. p. 13915, lines 14-15). Alternatively, is it possible that larger sizes result from differential mortality of smaller larvae in the elevated CO2 groups, such that only the larger larvae survive? If elevated CO2 does indeed produce increased growth rate, what is the mechanism by which this happens? (if this has been previously described, that study should be referenced) It is conceivable that if elevated CO2 and/or acidity reduce the affinity of hemoglobin to O2 (as would be expected), that increased O2 offloading results in increased metabolism and accelerated growth, though this is mere speculation on my behalf.

Some minor comments:

1) Under “husbandry and experimental protocols” (p. 13903), when describing events post-fertilization, the word “eggs” should be replaced with “embryos”. 2) P. 13909, line 16: “0 d old larvae” Need to define this in this sentence (i.e. days post-hatch) 3) P. 13909, line22: “PCA” needs to be define in this sentence.

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