We thank George Waldbusser for his review on our revised manuscript and for clarification of some specific points and are going to respond to his comment.

We agree with reviewer upon the problem to differentiate between specific carbonate system parameters as they are co-varying and results based on correlations are consequently to some extend uncertain and based on speculations. The only proof would be knock-down or inhibition of specific transporters which affect calcification directly. Therefore, we do not argue against the seemingly striking correlation of results plotted against Ω, DIC/H+, HCO3-/H+, CO32-, see also suppl. Fig 1. And we do not state that we understand the calcification process in detail. As highlighted in the response letter and the revised ms, we also agree on the importance of high, supersatured Ω values in the calcifying fluid itself and the problem of the organism to overcome the kinetic constrains of biomineralization in this fluid. However, the conditions in the calcifying fluid are expected to differ substantially from seawater conditions therefore it is not clear why seawater Ω should determine calcification rates (see also discussion in the revised ms). The HCO3-/H+ ratio provides a better explanation from a physiological perspective and describes the responses with a similar power, or under HCO3- limiting conditions even better.
At least in mussels, acid-base status of haemolymph and EPF are almost identical and adverse for calcification compared to seawater (lower pH, lower CO32- concentration, Ω <1) even under control conditions (low pCO2, normal alkalinity, open + ventilating animals), (see Thomsen et al. 2010, 2013, Heinemann et al. 2012). These conditions, however, are results of the need of the animal to excrete metabolic CO2 to the ambient seawater and do not indicate active sealing of that compartment. In mytilids, the mantle not only contains the calcifying epithelium but also the gonads. Consequently, metabolic CO2 generated in this tissue also diffuses into the EPF or prevents the rapid excretion of CO2 from the EPF, respectively. This results in an EPF pCO2 similar to the haemolymph (about 1500 µatm) which lowers pH and Ω. The acid-base status of seawater taken up by the organism will change accordingly and become undersatured. Consequently, we do not think that the bulk EPF is the site of calcification.

One problem of the study by Waldbusser et al.2014 is that the authors did not state whether the malformed larvae would have developed into normal D-veliger if they weren´t fixed or whether they were malformed and did not develop further. We applied similar carbonate system manipulations and observed `malformed` larvae two days post fertilization (see suppl. Fig. 2) which, however, developed into normal D-veliger after four days. In Waldbusser et al. 2014 the proportion of malformed oyster larvae in extreme treatments was higher in exp 1 than in exp 2. Exp 1 was conducted at lower temperatures; therefore it is possible that delayed development had a similar effect. This speaks for a strong delay of shell formation rather than an irrevocable disturbance of calcification, therefore supports the hypothesis that larvae are able to isolate the calcifying fluid. We argue that differences between larvae and juvenile stages mainly result from the much higher calcification rates (per gram tissue) in larvae, therefore effects appear to be more drastic, but the basic mechanism may not change completely during ontogeny. Therefore, critical conditions and a threshold might be similar in larvae and juveniles but will not always be detected.

Response to minor comments:
We are aware of the role of carbonate system as a buffer system in seawater. The question whether the carbonate system speciation effects pH or vice versa is not relevant for our study as it has no effect on the biological consequences. Therefore we do not want to argue with the reviewer on this topic.

The graph in Fig 5 refers to absolute respiration and calcification rates as we wanted to highlight that the supply of carbon by respiration is not sufficient to fuel the demand of calcification. It makes no differences whether rates are expressed as absolute or relative rates. Importantly, the mass specific data from Widdows 1991 depict data obtained by Sprung and Widdows 1986 who measured heat dissipation not respiration. However, the absolute rates in Fig 1 of Sprung and Widdows 1986 shows again a slow increase of heat dissipation and no peak at D-veliger stage similar to Sprung 1984b. Therefore, the argument that the ratio Ccalcified /Crespired may play a role for the isotopic signal is still valid.

The reviewer is right that we do not provide evidence that shells of larvae have the same thickness under control and high CO2, we added this to the ms. However, as the calcification of the shell is under high biological control, it seems implausible that growing larvae simultaneously dissolve existing shell which is solely made of aragonite. In juveniles this might be different, as length growth is based on calcite production, whereas internal dissolution within the pallial line represents nacre (aragonite) dissolution (as observed in Melzner et al. 2011). Both polymorphs might be differently regulated as they are produced by different parts of the mantle and contain different matrix proteins.
Although smaller and therefore thinner, the studies on larvae documented the increase of shell thickness - it was simply delayed similar to length growth. Furthermore, chemical dissolution is at least an order of magnitude lower than biomineralization, as long as animal grow (see also Duarte et al. 2015). Therefore we do not agree with some studies which expect substantial change of net calcification due to dissolution in (living) larvae.

Response to comment on Page 1554 Similar to Ω, the ideas of the importance of a ratio DIC/H+ or HCO3-/H+ have been developed for other organisms as well, but this does not weakens its strength from a physiological point of view.

Response to comment on `we do not see a realistic scenario` We agree with the reviewer that estuaries are complicated system with respect to carbonate chemistry due to special conditions of alkalinity and productivity. During blooms pH may rise, but at least in the Baltic does not cause values of 9. However, our study is a mechanistic study on bivalve calcification and data were obtained under controlled laboratory conditions therefore we will not discuss this issue in more details.

Response to comment on Line 23 The reviewer misinterprets our own data and Pan et al. Our own work always referred to the extracellular acid-base status which remains unregulated in bivalves, see general response; however, the pH of the intracellular space necessarily has to be tightly controlled in order to enable function of enzymes etc. Under elevated pCO2 and reduced extracellular pH, transport of H+ from the cytosol across the membranes has to increase as more H+ tend to leak into the cells. Pan et al. clearly states that the energy allocation to ion transport increased by about 50% which confirms upregulation of ion transport, at least potentially for acid-base regulation. Pan et al. also measured higher protein synthesis rates which, however, did not lead to higher protein content and growth rates. Therefore, these proteins were not used for somatic growth but potentially metabolized to match increased metabolic requirements indicated by reduced efficiencies. Therefore we added ‘deposition’ to the revised ms.

Response to comment on Fig 5 We accidently included an older version of fig 5 in the revised manuscript, the new version clearly states pmol O2 larvae -1 h-1, pmol CaCO3 larvae -1 h-1, see also comment above on this issue. Data on cell density from Sprung et al. have been added. The caption includes references in the corrected ms.

Response to the comment on the paragraph starting `Earlier studies..: The reviewer criticizes our oversimplification of the interpretation of his study. However, the fact that larvae seem to react more sensitive to adverse carbonate chemistry is, in accordance to his study from 2013, can be explained by the much higher relative calcification at this stage but does not necessarily indicate lower ability to isolate the shell from seawater than later stages. As the reviewer states larvae are able to calcify under adverse conditions, but are potentially malformed, see our general response above on this topic. At the same time, if reduced calcification results from lower ability of isolation, why should higher energy supply support calcification in the larvae as reported for juveniles? Furthermore, we do not think that the energy supply is critical as long as the larvae can rely on endogeneous reserves. The critical point is the moment when these reserves are exhausted but feeding cannot commence as the velum is not functional due to delayed shell formation. We are going to publish a ms which revealed that larvae are able to increase their calcification rates even under adverse carbonate system conditions which supports the idea that they are able to control the acid-base status of the calcification fluid. See also previous comment on respiration rates with respect to the interpretation of data on isotopic signals.

In the referred paragraph / sentences, the citation Moran and Manahan 2004 refers to the energy aspect in larval development and that starvation had no acute effect on larval survival. We cited His and Seaman 1992 who observed effects of starvation on settlement success. We clarified this in the corrected version. We referred to Riisgard 1980 in order to show, that filtration rates of bivalve larvae are small. We corrected the statement on the study by Hettinger et al. to clarify that they observed effects at low algae concentrations but no differences of survival and growth between the two higher treatments, potentially as food was not a limiting factor in these two.
As this study deals with mechanistic effects of the carbonate system on calcification, we do not discuss carry over effects between different ontogenetics stages which would distract from the topic.