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Interactive comment on "Linking climatic-driven iron toxicity and water stress to a massive mangrove dieback" *by* James Z. Sippo et al.

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We thank Fernanda Adame for the constructive feedback on the manuscript and will modify it to clarify the points raised.

Comment from Adame: This manuscript provides an interesting theory: increased Fe toxicity and water stress led to large-scale dieback of mangroves in northern Australia. The authors provide lines of evidence from wood and sediment cores to show that low mean sea level, low water vapour, and low precipitation contributed to changes in the biogeochemistry of the soil, which led to changes in the physiology of the trees. Their data shows a very high increase in wood Fe over the period when the dieback occurred, along with a possible decrease in water use efficiency, and decrease in wood density.

However, there were no clear differences between the "dead" site compared to the "live" site, with differences mostly driven by the position in the intertidal (low vs high). Both sites had similar growth rates, similar CRS, WUE, wood density and salinity. The only significant difference was that the live site had higher Fe+ in the soil that the dead one, a result which appears to be contradictory to their hypothesis. At the moment, the manuscript is written in a way that implies that all the data support their theory, but I am still not convinced. I agree that the climatic conditions led to drastic biogeochemical changes in the soil and mangrove trees, however, this does not explain why some of them died and some of them not. A cause-effect link cannot be established yet.

Response: We will re-focus the manuscript to better highlight the differences between dead and living areas. The most obvious difference between areas is Fe concentrations in wood, with peaks in the dead areas which were 30 to 90 fold higher than baseline in contrast to the living mangrove which showed an Fe peak 3-25 fold higher than baseline. If Fe was mobilised during the dieback event, during a period of increased sediment oxidation as hypothesised, then we would expect to see less Fe in the dead mangrove sediments than the living mangrove sediments. This result is therefore in support of the hypothesis (not contradictory) that the dieback is related to water availability. We will make it clear in the manuscript that our results are not conclusive in establishing a cause and effect relationship, but do provide important clues and insights regarding key processes occurring during the mangrove dieback.

Comment from Adame: Overall, I think the data is of high quality and there is potential for it to form the basis of an interesting and novel hypothesis on the effects of drought and sea-level rise on mangrove forests. However, it has to acknowledge that this theory does not prove why mangroves died. The authors mention in the conclusion that differences in groundwater could be the cause of death in some forests, however, they also mention that salinity was similar in both sites. There are still many unanswered questions and the paper needs to be rewritten in a way that provides some answers but also acknowledges that new questions have emerged that are yet to be resolved.

Response: As discussed above, we intend to re-focus the manuscript to have more circumspect conclusions and turn some of the speculation into a new hypothesis. Salinity concentrations in groundwater were taken eight months after the dieback event and may not represent the salinities that occurred during the dieback period. Speculation about the role of groundwater availability in the dieback will also be clarified in consideration of the salinity data.

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