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Interactive comment on "Evaluation of simulated biomass damage in forest ecosystems induced by ozone against observation-based estimates" *by* Martina Franz et al.

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1 Answers to Referee Marcus Schaub and comments by Maxime Cailleret and Marco Ferretti

Q: The authors argue that so far applied damage functions result in impacts with large uncertainty in the magnitude of ozone effects predicted. They use the O-CN biosphere model to test four already existing damage functions in terms of their simulated whole-tree biomass responses against field data from 23 ozone filtration/fumigation experiments and found that biomass damage was overestimated (Lombardozzi et al.

2012) or underestimated (Wittig et al 2007; Lambardozzi et al. 2013). The authors tune/reparameterize those damage functions towards a better fit with data from 15 fumigation experiments with young trees. In a second step, the authors tune DRRs again so that relative biomass (or NPP) simulated on adult trees fit the measured values on young trees.

A: As we explain below, this is an accurate representation of the manuscript's content, with the exception of the last sentence, as we did not recalibrate the model for adult trees.

Q: The ms. reads very well and is certainly within the scope of BG. As a matter of fact, we appreciate this exercise as it addresses a crucial issue in ozone risk assessment and provides an excellent review on the state of the art.

A: Thank you.

Q: While the first part, i.e. recalibration of existing damage functions makes sense to improve DRRs for young trees and better predict biomass loss due to ozone. We are, however, concerned about the second step, i.e. the reparameterization/tuning of those functions (for young trees) to better predict relative biomass for mature trees.

A: It is a misunderstanding that we re-calibrated the model to simulate old-growth forests. This is not the case. We applied the model tuned for young trees to simulate old growth forests and compared the simulated ozone damage of the young and mature trees in terms on their effect of biomass and biomass production. We found that young and mature tree produce strongly differing biomass dose-response-relationships but similar dose-response- relationships for NPP. This leads us to the assumption that NPP responses to ozone damage of young and mature trees might be better comparable than biomass responses. We believe that this analysis is helpful to illustrate the problem of using biomass-reduction-ozone uptake relationships to

quantify ozone damage for the development of process-based models. We will rework the manuscript in order to make sure the confusion regarding the re-calibration cannot occur.

Q: The authors aim at improving the quantitative understanding of ozone effects on forest growth and carbon sequestration on a regional or even global scale. Using data from seedlings gown under (semi-)controlled experiments ranging over a few years may (still) not lead to reliable model functions for adult trees growing in complex forest ecosystems. The cited work by Franz et al. 2017 (GPP reduction, based on damage functions from Wittig et al. 2007) is an example how model exercises using modeled data may result in inaccurate predictions – if not validated with measured data from adult trees (see also Cailleret et al. 2018). Page 15, line 6-10 demonstrates the risk of applying models, based on former functions and stresses the need of validating model exercises with measured data (e.g. from ICP Forests).

A: The model experiment with adult trees presented were an attempt illustrate this problem and the question of how much these damage functions may be scalable or not. We will add the manuscript by Cailleret et al. 2018 to the discussion, but remain sceptical that comparison to ICP forest data, given the lack of a control to isolate ozone damage from other co-occurring environmental drivers such as atmospheric N and S deposition or climatic variability. Nevertheless, this is a good suggestion to corroborate not the damage function per-se, but at least the simulated growth rates under O3 exposure. We will consider this as a follow-up study, subject to sufficient data availability to run and evaluate models for these sites.

Q: We suggest to either omit the second part or to extend section 3.3. and the discussion and to outline not only the advances but also the still existing lack of knowledge for estimating ozone induced biomass effects on adult trees, forest ecosystems respectively.

A: We will extend our discussion to highlight this uncertainty.

Q: Novak et al. (2008) found that species competition may alter DDRs. We did not understand if and how competition is considered in the O-CN biosphere model. Please, elaborate on this in more detail and in relation to the anticipated forest ecosystem approach.

A: As described, (P 7, LL 13-15) OCN simulates plant functional types, not species. Therefore the effects of species composition and its change are not accounted for. This issue is taken up in the discussion.

Q: The term “damage” is frequently used in the ms. in different contexts and scales: “Damage of photosynthetic apparatus”, “ozone damage”, “leaf-scale” to “global estimates”. In some parts it seems that damage functions refer to “the effects of ozone uptake on photosynthetic variables” and in other parts damage seems to refer to “the fractional loss of carbon uptake associated with ozone uptake”. We suggest that the authors define explicitly and very early in the ms. what they mean with “damage”, “ozone damage”, “damage function” and also specify the difference between “dose-response relationship” and ”damage functions”.

A: We will revise the manuscript according to these suggestions.

Q: Ozone and trees and forests: The actual extent to which reduction in tree growth due to ozone occurs in the real forest remains still unclear. There are several studies which found significant effects, others did not (they did not observe measured aboveground tree growth, which is not total biomass, but an often used proxy for it). We think these controversial results should be considered and discussed as they may help to better contextualize the paper.

A: We will include this in the discussion.

Q: Juvenile vs. mature trees: Despite the short explanation given on p. 8, line 21-28, it is not clear how DDRs for mature trees were simulated. Since this is a very important step (and output) for the non-modelers, a more detailed explanation will be very useful here.

A: As stated above, we did not re-tune the relationships.

Q: Reduction of biomass: It is not clear what is intended here as reduction of biomass. While we understand the reduction of biomass increment, we can hardly see a living tree reducing its biomass due to ozone. The formulation (6) actually seems to refer to a difference of biomass of treated trees with respect to the controlled ones, and not to a reduction of biomass of the treated trees. This is somewhat acknowledged by the authors in the discussion, but perhaps it deserves more emphasis.

A: We will clarify the text to be more precise that we mean the difference between a control and a treatment.

Q: Finally, it will be important to have some statement how the authors - based on these results - see the value of the risk maps produced by EMEP for e.g. European forests.

A: It is not the point of the paper to provide an assessment of EMEP and its suitability for risk assessment, specifically because we only assess one part, the O3-damage calculation, of the EMEP risk assessment, while EMEP includes a range of other processes. A proper assessment of these Maps would require a simulation comparable to the EMEP projections to disentangle regional and temporal differences in O3-related risks. Such wall-to-wall simulations are beyond the scope of our study, and we would therefore prefer to avoid to add too much speculative assessment at this point.

1.1 Specific comments

Q: P2, L17: "simulated reductions in GPP due to ozone damage vary substantially between models and model versions": please, provide some examples and values.

A: We added suitable references. The modelling protocols, in these studies differ strongly regarding the simulated years and accounting/not accounting for e.g. changing nitrogen deposition and elevated CO_2. For instance Sitch et al. (2007) estimates the GPP reduction between 1901-2100 and Franz et al. (2017) the mean decadal reduction for 2001-2010 compared to a simulation without accounting for ozone effects. Lombardozzi et al. (2012) report mean annual reductions in GPP for a 20 yr run of CLM at 100 ppm O_3 which can be compared to neither of the first. A concise summary of the estimated GPP reductions in these studies without explaining the different modeling assumptions might mislead the reader. We believe that a detailed display of the results and the underlying assumptions would to much extend the introduction.

Q: P2, L18: Here and elsewhere, you may consider Cailleret et al. (2018) as additional reference.

A: We will include the Cailleret et al. paper.

Q: P3, L10: You may consider Schaub et al. (2005) as additional reference. M&M: Please, provide more details on O-CN structure and main assumptions, even though this model has been used and described in Zaehle and Friend (2010), and in Franz et al. (2017). It would help that the reader does not need to go back and forth between the current paper and these ones. e.g., What is the spatial resolution? Individual-based or

cohort-based model?

A: We will add a short description of relevant model features, but refrain from repeating what has been described before. Furthermore in the simulations here the model is run on point scale (the coordinate of the experiment site) and no spatial resolution is applied.

Q: P4, L31-33: No reserves?

A: See description of storage on P4, L29-31.

Q: P4, L31-33: Biomass growth seems to be dependent only on source but not on sink activity. Is that correct? If yes, this is a strong assumption and limit of the modelling approach (see Körner 2015) that has to be discussed later.

A: This is not fully true, as the model does account for sink limitation due to nutrient constraints. It is correct, however, that the model does not account for sink limitation due to constrained rooting zone volume or number of leaf buds.

Q: P5, L10: We suggest to add the equation(s) used to calculate An,l

A: Given the complexity of the approach, as outlined in Kull & Kruit 1998, we prefer to not do this, as it would divert the reader's attention. We will add the variables that drive the calculations of $A_{n,l}$, namely leaf internal partial pressure of CO_2, absorbed photosynthetic photon flux density on shaded and sunlit leaves, leaf temperature, as well as the maximum carboxylation and electron-transport rates, which each are a function of leaf nitrogen concentration.

Q: P5, L17: It seems that the authors assume that O3 concentration is constant within the canopy. Correct? Please, clarify and discuss.

A: Yes, the ozone concentration is constant within the canopy. The biomass damage experiments we try to reproduce here are all conducted with saplings which were mostly fumigated in open top chambers. Pronounced O_3 gradients within the canopy thus are not to be expected. However, we note that ozone uptake is not constant within the canopy given the distribution of light and photosynthetic capacity. We assume that these gradients have a much stronger effect on layered O3 uptake than any vertical gradient in O3 in such an experimental setting.

Q: P5, L25: "the Phytotoxic Ozone Dose (POD, mmolm-2) can be diagnosed by the accumulation of fst,l for the top canopy layer (l = 1)." In most ozone-flux modeling approaches, POD is calculated based on a "big-leaf" approach (one layer of leaf area, but LAI can be > 1; approach used in DO3SE) -> this is different from the accumulation of fst,l for the top canopy layer. See also P16, L18. Please clarify.

A: As in our reply to referee #1:

We designed our study such that our way to calculate POD is consistent with those from Büker et al. (2015), from which we took the dose-response-relationships. They calculate the $PODy$ used for their analysis in accordance with the LRTAP-Convention (2010), which states 'the index PODY is used to quantify the flux of ozone through the stomata of the uppermost leaf level that is directly exposed to solar radiation and thus no calculation of light exclusion, caused by the filtering of light through the leaves of the canopy, is required'. We calculate the $PODy$ based on the LRTAP-Convention (2010), to be able to compare our simulation results to those of Büker et al. (2015).

We will add a citation of the LRTAP convention to the explanation on the calculation of POD in the text: 'For comparison to observations, the Phytotoxic Ozone Dose (POD, $mmolm^{-2}$) can be diagnosed by the accumulation of $f_{st,l}$ for the top canopy layer (l = 1), in accordance with LRTAP-Convention (2010) and Büker et al. (2015).'

Q: P6, L18: Correct "ration"

A: Done.

Q: P6, L19: Correct "is is“

A: Done.

Q: P7, L18: The initialization phase is not clear: we don’t see how the model can run “from bare ground until the simulated stand-scale tree age was stable and representative of 1-2 year old seedlings”. And this is even less clear with the sentence

A: Our aim is to simulate seedling similar to the fumigated seedling in the biomass damage experiments. When we first start our model no trees are present and seedlings start to grow after the simulation starts. O-CN is a stand-scale model and not an individual based model. Until the mean stand-scale age of 1-2 years is realised a larger number of simulation years passes. The exact number of simulation years is site specific but on average over all simulation sites it takes the mentioned 7.8 years. After this initialisation phase we can start the simulation of the experiment years.

Q: P8, L5: “The duration of the initialization phase (. . .) averages 7.8 years”. Furthermore, did the authors run only one or multiple O-CN simulations per study case (per experiment)? We guess there are some stochastic processes in O-CN, these ones may induce some epistemic uncertainties that have to be considered in the modeling framework.

A: There are no stochastic elements in these simulations and running multiple simulations yields identical results.

Q: P9, L25: It is not clear how this “tuning” has been performed: was it a manual or an automatic optimization. Which algorhythm was used? Bayesian framework? Which

metric did the authors try to optimize (likelihood; rmse, r2)?

A: The tuning was performed manually.

Q: P10, L15: Please, show results in Supp Mat.

A: As stated in the manuscript accounting for direct injury of the stomates had only minimal effects (and only for the needleleaf category), see also Fig. 1 here. We could include the graphic into the Supp. Material but we are not sure if it justifies the creation of a suplement since there is none at the moment.

Q: P11, L2: “The simulations L12PS and L12VC (. . .) strongly overestimate”. Yes, but this is less strong than the underestimation by W07 and L13.

A: We do mention that W07 strongly underestimates the damage and elaborate on the results of L13 and the reasons for them (P10,LL1-14 in the manuscript in discussion).

Q: Figure 2, panels a, b, c, d: what do the simulations without O3 fumigation (after the red line) look like?

A: In the control simulation no ozone damage occurs, photosynthesis and biomass do not decline.

Q: Figure 2: Please, add simulated before cumulative in the legend; Idem P11, L11, add simulated before CUOY.

A: Done.

Q: P11, L16: There is no control simulation shown in Fig. 2 (see our comment above)

A: Yes, because this graphic is used to explain the extreme effects of ozone fumigation on the plant physiological processes when applying the L12$_{PS}$ injury relationship. In the control simulation no ozone damage occurs, photosynthesis and biomass do not decline. In our view adding the control here overcrowds the graph but does not add valuable information to the reader. The key point of this graphic is to illustrate the extreme effects ozone fumigation imposes on plant performance (e.g. negative values of A_n^{can}) if the L12$_{PS}$ injury relationship is applied.

Q: P12, L5-7: In M&M P12, L12 to P13, L4: This comparison between mature vs. young trees is not described in the M&M. How do the simulations differ in terms of initialization etc.?

A: See P8 L21-30 of the original manuscript, which is part of the M&M. We will make sure that this text is not overlooked in the revised version.

Q: P14, L11 and P15, L8: Please provide some values.

A: This is impossible without performing a large-scale integration of the model, which is beyond the scope of this paper.

Q: P15, L17 and throughout the paper: Note that Büker et al. (2015) used the Jarvis equation to simulate stomatal conductance while the Ball & Berry one is used here. Please be cautious when comparing both studies.

A: It is unclear to us, why the form of the equation is relevant here. What is relevant is whether the simulated canopy conductance is in agreement with observations, and at least for the part of OCN this has been demonstrated by Franz et al. 2017.

Q: P15, L20-22: We agree that this is a key aspect, which has to be more detailed.

A: Both model differ in many respects that will impact the estimate of ozone uptake and accumulation as stated in the manuscript and the provided examples. We are not sure if adding a longer, more detailed list of the differing model features will benefit the reader. However we extended the respective sentence to clarify that these impact the suggested dose-response-relationships: 'However, both models vary in their complexity of the simulated plants, carbon assimilation, and growth processes, which will also impact the estimates of ozone accumulation (POD_y) and hence their suggested biomass dose-response-relationships.'

Q: Discussion: The DRRs built in the present study are valid only for O-CN and may not work for other dynamic vegetation models (strongly depends on how biomass growth is simulated by the DVM -> sink vs. source activity etc.). This is implicitly written in P15, L32-33; but this has to be mentioned again in the conclusion. We suggest to rather highlight that the approach developed here is interesting and can be followed to calibrate "ozone submodels" in further DVMs.

A: Done.

Q: P16, L15 and P17, L5: Idem show some results in Supp Mat.

A: Regarding P16, L15: As stated in the manuscript the calculation of plant injury based on $POD1$ rather than $CUO1$ (using an adapted slope in the model simulations) yielded dose-response-relationships which are comparable to the ones based on $CUO1$, see also Fig. 2 here. The simulation of plant injury based on $CUO1$ seems to be preferable over $POD1$, because the canopy layer specific ozone uptake is translated into a layer specific injury fraction. Following this we remain uncertain as to the value of including the $POD1$ results into the supplement. Regarding P17, L5: See answer to Q P10, L15.

Q: P17, L30-33: Authors ask for monitoring programs "capable to measure the ac-

tual increment of biomass". We assume that they know that these programs do exist, e.g. national forest inventories and international monitoring programs such as the ICP Forests. Please, quote these programs here.

A: We will clarify that we meant to state monitoring programs of ozone damage. Of course, increment networks exists, but they are intrinsically incapable of separating the effects of ozone, N and S deposition and climate variations.

Q: P18, L1-10: The authors may also consider that trees usually occur in forests, and that forests are subjected to entire ecosystem dynamics that can offset / mitigate / adapt / compensate ozone effects. This should be discussed and considered in the conclusions.

A: Taken up in the discussion.

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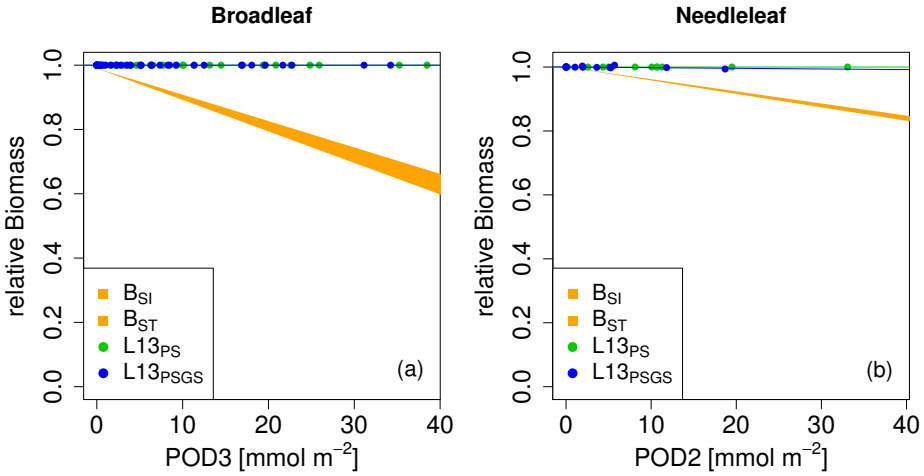

Fig. 1. Biomass dose-response relationships for simulations based on published injury relationships by Lombardozzi et al. (2013) to netPS (PS) and both netPS and stomatal conductance (PSGS).

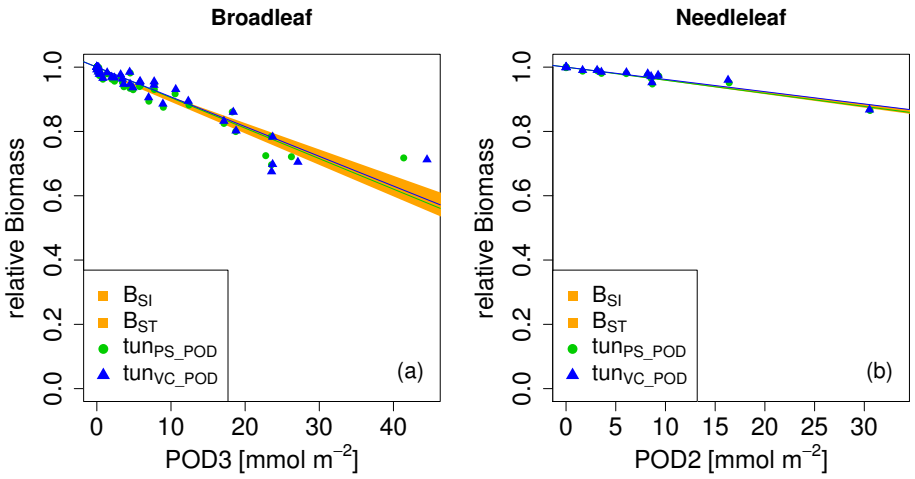

Fig. 2. Biomass dose-response relationships for simulations based on tuned injury functions, where injury is calculated based on POD1 rather than CUO1.