Dear Editor,

Many thanks for your reply. Please find our answers to your comments, the answers to the referees (indicating major changes in the new manuscript) and a marked up manuscript version showing the conducted changes below.

Best wishes

Martina

0.1 Answers to Editor

Q: Please give particular attention to ensuring that you more fully addressed the comments of reviewer # 1 with respect to the top of canopy dosage and variation with depth through the canopy. Whilst I found the authors responses satisfactory, I wonder how much effort it would be to test an alternative assumption? If only to demonstrate the sensitivity to an alternative assumption? Alternatively, some space could be dedicated to this in the discussion.

A: As mentioned by referee # 1 the estimate of the PODy values strongly impacts the resulting dose-response-relationships. The PODy values in the Büker et al. (2015) study are modelled values and not measured ones. Büker et al. (2015) calculate the PODy according to the LRTAP-Convention (2010). To be able to compare our simulation results to their ones we have to use the same approach as they do. As already discussed in the manuscript differing model features between DO_3SE and O-CN will probably impact estimates of the $PODy$ and hence the dose-response-relationships.

The simulation of ozone uptake and damage through all canopy layers is unconnected to the calculation of the $PODy$ used for the formation of the biomass dose-response-relationships and thus open to the application of different approaches. We tested 2 commonly used approaches to simulate ozone uptake and damage within the canopy. First the explicit simulation of ozone uptake in each canopy layer and the application of the respective damage fraction in each layer (damage based on $CUOY$). Ozone uptake in each layer is determined by factors that impact the stomatal conductance in each canopy layer, e.g. the light conditions and the varying nitrogen content within the crown. Secondly we tested the calculation of the ozone uptake only in the top canopy layer of the trees and the application of the respective damage fraction to all canopy layers (damage based on $PODy$) and elaborate on the

differences between the 2 and the reasons for them.

If further data become available regarding the vertical gradient of ozone uptake within canopies alternative simulation approaches might be developed. The sentence on p.16 ll.20-23 was extended to make this point: 'More analysis of the differential effect of ozone injury within deep canopies are required to evaluate whether the scaling of top-of-the-canopy injury to whole canopy injury is appropriate or if alternative simulation approaches need to be developed (now on p.17 ll.10-12).'

Q: In addition, please pay particular to reviewer # 2, where they ask about mature vs young trees and species-specific relationships. These questions strike me as interesting points to form a discussion around future extensions of your work.

A: Both aspects (young vs. mature trees and species-specific relationships) are now discussed in 2 paragraphs ((now on p.17 l.23-p.18 l.2) and p.19 ll.5-15) of the discussion.

In addition to their comments, could I please ask you to:

Q: Abstract: define V_{cmax}

A: Done.

Q: Pg 4: where you describe how the labile non-structural pool buffers growth, it strikes me as important for the reader to get a sense of what sort of time-scale this may impose on any direct impact of ozone on leaf-photosynthesis vs. realised growth. Days? Weeks? Months?

A: We included a sentence to explain that the labile pool responds within days to changes in GPP, and the long-term reserve takes several months to respond (p.4 ll.33-p.5 l.2).

Q: Pg 5: Where you refer to the CUOY being calculated by summation over all layers and then refer to Franz et al. 2017. Is there further detail that the reader should see here? If it is simply summation, then perhaps simply cite Franz? As written it implies that there is additional insight here and that should either be presented here or clarified.

A: We changed the manuscript from (see Franz et al. 2017 for details) to (Franz et al. 2017).

Q: Pg 6, line 25: why didn't they match well? Could this point be developed/shown? My reading of the manuscript is that this is this what is referred to in Figure 1? If so, could the authors explain to the reader that they will address this in the results? My reading of the methods is that it is simply asserted to be true, but I may have missed the explanation...

A: The respective sentence is a forecast of the results displayed in Figure 1. As suggested we added a remark that the corresponding results are shown in the results section and Fig. 1 (p.7 ll.10-11).

Q: Equation 6, what is n?

A: The 'n' is a typing error and got removed.

Q: Fig 2: CUOY not CUOy.

A: Changed.

Q: Page 12, line 10: This statement is true so long as the canopy is simulated to be estimated via the V_{cmax} limitation? This would change in different scenarios (i.e. if the canopy was J_{max} limited) and I think this point should be clarified for the reader. Currently it (wrongly - in my opinion) implies that a model would get the same result if they applied the function to V_{cmax} or An.

A: Please note that not only V_{cmax} but also J_{max} is changed (see Methods), hence the ratio between V_{max} and J_{max} remains constant. We added 'V_{cmax} and simultaneously J_{max}' in the respective sentence to remind the reader of this fact (p.12 l.11).

0.2 Answers to Anonymous Referee # 1

Q: The authors assume that the modelled accumulation of ozone fluxes at the top canopy layer equals POD during the model-observation comparison process. Please justify this assumption. I think this is important for the evaluation of model against observation, considering the ozone damage is explicitly calculated through the canopy and integrated to derive the whole tree damage. The modelled POD value largely influences the slope of the

resultant dose-response curve and its distance with observed dose-response curve. I am wondering how would the authors account for this treatment in influencing the evaluation of different algorithms against observed data.

A: 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). (p.6 l.11)'

Of course, there will be uncertainty in the calculation of the POD by both Büker et al. (2015) and our study compared to the real-world POD, given both are based on different, but evaluated models (Emberson et al., 2000; Franz et al., 2017), but in the absence of direct measurements of POD it is impossible to judge whether or not this would introduce any systematic bias into the comparison.

Q: I am curious why did not the author try to use different damage functions at different depth of the canopy?

A: Each of the damage functions is applied to all canopy layers in separate simulations for each damage function. The ozone damage differs within the canopy, as increasing canopy depth leads to lower leaf-specific photosynthesis, conductance, and therefore ozone uptake and damage.

Our aim was to investigate the suitability of different damage functions to reproduce observed biomass damage relationships. Following this we always only applied one damage function in one simulation. The application of different damage functions in one simulation, e.g. different damage functions for different canopy layers, can not contribute to answer our research question.

Evidence exists that sunlit and shades leaves exhibit a different sensitivity to O_3 (Tjoelker et al., 1995; Wieser et al. 2002). Following this the application

of different damage functions for different canopy layers might yield improved damage estimates. However damage relationships for different canopy depth are to our knowledge not available as well as independent data to evaluate them.

Q: 3) Another important, but still largely missing, aspect in simulating ozone impacts on vegetation is the huge diversity of species-sensitivity in an ecosystem. Dealing with vegetation to the PFT level is not enough, though totally make sense in terms of large scale modelling and data scarcity. This work could be improved by further talking about diversity of species response to ozone. To this end, I found the following work could be a good reference: Wang, B. et al. Forests and ozone: productivity, carbon storage, and feedbacks. Sci. Rep. 6, 22133; doi: 10.1038/srep22133 (2016)

This study, though without sophisticated ozone damage simulation, had an explicit simulation of species sensitivity to ozone using an individual-based model and found dampened responses to ozone over long-term simulations.

A: We will include this study in our discussion (p.17 ll.27-29).

0.2.1 Minor comments

Q: L27 on page 4: please justify the statement of highest N concentration at the top of the canopy and its exponential decline with increasing canopy depth.
A: We base this statement on the publications by Friend (2001) and Niinemets et al. (2015) and will add these references in the manuscript (p.4 ll.27-29).

Q: L18 on page 5: in equation 2, how is the stomatal conductance of O3 calculated?
A: Explanation added (p.5 ll.25-27).

Q: L28 on page 8: identical →identically.
A: Done.

Q: L5-6 on Page 18: this sentence should be restructured to make it easier to follow.

A: Done.

Q: L7 on page 18: ''all in all' should be followed a comma.
A: Done.

0.3 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. We introduced a subsection called ' Modelling protocol for mature trees' and we state in there 'The ozone injury for mature trees is calculated based on the same tun_{VC} injury function (see Tab. **??**) that is used in the simulation of young trees.'(p.9 ll.11-12)

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.(p.19 ll.9-13)

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 of the old manuscript) 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 (p.17 l.29 - p.18 l.2).

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. We define the use of the respective terms on p.5 ll.8-11.

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 above-ground 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.(p. 19 ll.7-15)

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. (p.9 l.14)

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.

0.3.1 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. (p.4 l.21 and p.8 l.2)

Q: P4, L31-33: No reserves?

A: See description of storage on P4, L29-31 (p.4 32-33 in new manuscript).

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 (p.7 l.11).

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 supplement 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 by introducing a separate subsection called ' Modelling protocol for mature trees' (p.9 l.3)

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 ($PODy$) and hence their suggested biomass dose-response-relationships.'(p.16 ll.8-10)

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 (p.20 ll.2-5).

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 actual 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 (p.17 ll.29 - p.18 l.2 and p.19 ll.7-15).

0.4 References

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Evaluation of simulated biomass damage in forest ecosystems induced by ozone against observation-based estimates

Martina Franz[1,2], Rocio Alonso[4], Almut Arneth[5], Patrick Büker[6], Susana Elvira[4], Giacomo Gerosa[7], Lisa Emberson[6], Zhaozhong Feng[8], Didier Le Thiec[9], Riccardo Marzuoli[7], Elina Oksanen[10], Johan Uddling[11], Matthew Wilkinson[12], and Sönke Zaehle[1,3]

[1]Biogeochemical Integration Department, Max Planck Institute for Biogeochemistry, Jena, Germany
[2]International Max Planck Research School (IMPRS) for Global Biogeochemical Cycles, Jena, Germany
[3]Michael Stifel Center Jena for Data-driven and Simulation Science, Jena, Germany
[4]Ecotoxicology of Air Pollution, CIEMAT - Research Center for Energy, Environment and Technology, Avda. Complutense 40, edif.70, Madrid 28040, Spain
[5]Karlsruhe Institute of Technology (KIT), Department of Atmospheric Environmental Research (IMK-IFU), Garmisch-Partenkirchen, Germany
[6]Stockholm Environment Institute at York, Environment Dept., University of York, YO10 5NG, United Kingdom
[7]Department of Mathematics and Physics, Catholic University of Brescia, via Musei 41, Brescia (Italy)
[8]State Key Laboratory of Urban and Regional Ecology, Research Center for Eco-Environmental Sciences, Chinese Academy of Sciences, Shuangqing Road 18, Haidian District, Beijing, 100085, China
[9]Inra, Université de Lorraine, AgroParisTech, Silva, F-54280 Champenoux, France
[10]Department of Environmental and Biological Sciences, University of Eastern Finland, 80101 Joensuu, Finland
[11]Department of Biological and Environmental Sciences, University of Gothenburg, Gothenburg, Sweden
[12]Centre for Sustainable Forestry and Climate Change, Forest Research, UK

Correspondence: Martina Franz (mfranz@bgc-jena.mpg.de)

Abstract.

Regional estimates of the effects of ozone pollution on forest growth depend on the availability of reliable ~~damage~~ injury functions that estimate a representative ecosystem response to ozone exposure. A number of such ~~damage~~ injury functions for forest tree species and forest functional types have recently been published and subsequently applied in terrestrial biosphere models to estimate regional or global effects of ozone on forest tree productivity and carbon storage in the living plant biomass. The resulting impacts estimated by these biosphere models show large uncertainty in the magnitude of ozone effects predicted. To understand the role that these ~~damage~~ injury functions play in determining the variability of estimated ozone impacts, we use the O-CN biosphere model to provide a standardised modelling framework. We test four published ~~damage~~ injury functions describing the leaf-level, photosynthetic response to ozone exposure (targeting the maximum carboxylation capacity of Rubisco (V_{cmax}) or net photosynthesis) in terms of their simulated whole-tree biomass responses against field data from 23 ozone filtration/fumigation experiments conducted with European tree species at sites across Europe with a range of climatic conditions. Our results show that none of these previously published ~~damage~~ injury functions lead to simulated whole-tree biomass reductions in agreement with the observed dose-response relationships derived from these field experiments, and instead lead to significant over- / or underestimations of the ozone effect. By re-parameterising these photosynthetic based

~~damage~~ injury functions we develop linear, plant functional type specific dose-response relationships, which provide accurate simulations of the observed whole-tree biomass response across these 23 experiments.

1 Introduction

Ozone is a phytotoxic air pollutant which enters plants mainly through the leaf stomata, where reactive oxygen species (ROS) are formed that can ~~damage~~ injur essential leaf functioning (Ainsworth et al., 2012). Ozone induced declines in net photosynthesis (Morgan et al., 2003; Wittig et al., 2007) have been observed as the result of ~~damage~~ injury of the photosynthetic apparatus, increased respiration rates caused by investments in repair of injury, as well as the production of defence compounds (Wieser and Matyssek, 2007; Ainsworth et al., 2012). At the leaf-scale, ozone ~~damage~~ injury occurs and accumulates, when the instantaneous stomatal ozone uptake of leaves surpasses the ability of the leaf to detoxify ozone (Wieser and Matyssek, 2007). These effects are likely the primary cause for reduced rates of net photosynthesis and decreased supply of carbon and energy for growth and net primary production (NPP), which contributes to the commonly observed ozone-induced reductions in leaf area and plant biomass (Morgan et al., 2003; Lombardozzi et al., 2013; Wittig et al., 2009). Changes in tropospheric ozone abundance and associated changes in ozone-induced ~~damage~~ injury thus have the potential to affect the ability of the terrestrial biosphere to sequester carbon (Harmens and Mills, 2012; Oliver et al., 2017). However, a quantitative understanding of the effect of ozone pollution on forest growth and carbon sequestration at the regional scale is still lacking. Terrestrial biosphere models can be used to obtain regional or global estimates of ozone damage based on an understanding of how ozone affects plant processes leading to C assimilation and growth. Modelling algorithms to estimate regional or global impacts of ozone on gross primary production (GPP) have been developed for several of these terrestrial biosphere models (Sitch et al., 2007; Lombardozzi et al., 2012a, 2015; Franz et al., 2017; Oliver et al., 2017). However, simulated reductions in GPP due to ozone ~~damage~~ induced injury vary substantially between models and model versions (Lombardozzi et al., 2012a, 2015; Franz et al., 2017; Sitch et al., 2007).

This uncertainty is predominantly due to the different approaches that these models use to relate ozone uptake (or ozone exposure) to reductions in whole-tree biomass, and in the exact parameterisation of the injury functions and dose-response ~~relationship~~ relationships applied (Karlsson et al., 2004; Pleijel et al., 2004; Wittig et al., 2007; Lombardozzi et al., 2012a, 2013). The ~~dose-response relationships~~ injury functions employed by current terrestrial biosphere models differ decidedly in their slope (i.e. the change in ~~damage~~ injury per unit of time-integrated ozone uptake), intercept (ozone ~~damage~~ injury at zero time-integrated ozone uptake), and in their assumed threshold, below which the ozone uptake rate is considered sufficiently low that ozone will be detoxified before any ~~damage~~ injury occurs (Karlsson et al., 2004; Pleijel et al., 2004; Lombardozzi et al., 2012a). For example, Sitch et al. (2007) relates the instantaneous ozone uptake exceeding a flux threshold to net photosynthetic ~~damage~~ injury via an empirically derived factor. An alternative approach has been to relate ozone ~~damage~~ injury to net photosynthesis in response to the accumulated ozone uptake rather than to the instantaneous ozone uptake as in Sitch et al. (2007), e.g. by using the $CUOY$, which refers to the cumulative canopy O_3 uptake above a flux threshold of $Y\ nmol\,m^{-2}\,s^{-1}$ ~~(Wittig et al., 2007; Lombardozzi et al., 2012a, 2013)~~(Wittig et al., 2007; Lombardozzi et al., 2012a, 2013; Cailleret et al., 2018).

The effect of ozone on plant growth has been investigated by ozone filtration/fumigation experiments either at the individual experimental level or by pooling data from multiple experiments that have been conducted according to standardised experimental method. These experiments typically rely on small trees or saplings. A challenge in developing and testing process-based models of ozone damage from these ozone fumigation experiments is that often only the difference in biomass accumulation between plants grown in an ozone treatment and in ambient or charcoal-filtered air at the end of the experiment are reported. Data from these studies provide evidence for a linear, species-specific relationship between accumulated ozone uptake and reductions in plant biomass (Pleijel et al., 2004; Mills et al., 2011; Nunn et al., 2006, e.g.). Sitch et al. (2007) for instance calibrated their instantaneous leaf-level ~~dose-response relationship~~ injury function between ozone uptake and photosynthesis by relating simulated annual net primary production and accumulated ozone uptake to observed biomass dose-response relationships developed by Karlsson et al. (2004) and Pleijel et al. (2004), where biomass/yield damage is related to the Phytotoxic Ozone Dose ($PODy$). The $PODy$ refers to the accumulated ozone uptake above a flux threshold of y $nmol\,m^{-2}\,s^{-1}$ by the leaves representative of the upper canopy leaves of the plant. Such an approach applies biomass dose-response relationships of young trees to mature trees. However, the effects of ozone on leaf physiology (e.g. net photosynthesis and stomatal conductance) or plant carbon allocation may differ between juvenile and adult trees (Hanson et al., 1994; Samuelson and Kelly, 1996; Kolb and Matyssek, 2001; Paoletti et al., 2010). Whether or not biomass dose-response relationships can be used to calibrate ~~dose-response~~ injury functions for mature trees is uncertain.

An alternative approach is to directly simulate ozone ~~damage~~ injury to photosynthesis, which may have been a major cause for the observed decline in plant biomass production (Ainsworth et al., 2012). Possible ~~damage~~ injury targets in the simulations can be for example the net photosynthesis or leaf-specific photosynthetic activity (such as represented by the maximum carboxylation capacity of Rubisco, V_{cmax}). For instance ~~Lombardozzi et al. (2012a) based their dose-response relationships~~ Lombardozzi et al. (2012a) based their injury function on an experimental study involving a single forest tree species, whereas more recent publications (e.g. Lombardozzi et al. (2015) and Franz et al. (2017)) have used ~~dose-response relationships~~ injury functions from meta-analyses of a far larger-set of filtration/fumigation studies. Meta-analyses have attempted to summarise the responses of plant performance to ozone exposure across a wider range of experiments and vegetation types (Wittig et al., 2007; Lombardozzi et al., 2013; Feng and Kobayashi, 2009; Li et al., 2017; Wittig et al., 2009) and to develop ~~damage~~ injury functions for plant groups that might provide an estimate of mean plant group responses to ozone. However, these meta-analyses suffer from a lack of consistency in the derivation of either plant ~~damage~~ injury or ozone exposure, and generally report a large amount of unexplained variance. A further complication in the meta-analyses of ozone ~~damage~~ injury (e.g. Wittig et al., 2007; Lombardozzi et al., 2013) is that they have to indirectly estimate the cumulative ozone uptake underlying the observed ozone ~~damage~~ injury based on a restricted amount of data, which causes uncertainty in the derived ~~damage~~ injury functions.

Büker et al. (2015) provides an independent data set of whole-tree biomass plant responses to ozone uptake which is independent of data sets that were used to describe ~~damage~~ injury functions by Wittig et al. (2007) and Lombardozzi et al. (2013). This data set has been collected from experiments that follow a more standardised methodology to assess dose-responses and has associated meteorological and ozone data at a high time resolution that allow more accurate estimates of modelled ozone uptake to be made. These dose-response relationships describe whole-tree biomass reductions in tree seedlings derived from

standardised ozone filtration/fumigation methods for eight European tree species at ten locations across Europe (see Tab. A.2 for details Büker et al., 2015). These data thus provide an opportunity to evaluate simulations of biosphere models that use leaf level ~~damage~~ injury functions (describing the effect of ozone uptake on photosynthetic variables) to estimate C assimilation, growth and ultimately whole tree biomass against these robust empirical dose-response relationships that relate ozone exposure directly to whole tree biomass response.

Here we test four alternative, previously published ozone ~~damage~~ injury functions that target either net photosynthesis or the leaf carboxylation capacity (V_{cmax}), which have been included in state-of-the-art terrestrial biosphere models (Lombardozzi et al., 2012a, 2015; Franz et al., 2017) against these new biomass dose-response relationships by Büker et al. (2015). We incorporate these ~~damage~~ injury functions into a single modelling framework, the O-CN model (Zaehle and Friend, 2010; Franz et al., 2017). To reduce model-data mismatch, we test the functions in simulations that mimic to the extend possible the conditions of each of the experiments in the Büker et al. (2015) data-set, in particular the young age, such that we can directly compare the simulated to the observed whole-tree biomass reductions of the empirically derived dose-response relationships. This allows us to identify the contribution of these alternative ~~damage~~ injury function formulations on the simulated whole-tree biomass response. The simulated biomass dose-response relationships are then compared to the data from the experiments to evaluate the capability of the different model versions to reproduce observed dose-response relationships. Based on these comparisons we use a similar approach to that of Sitch et al. (2007) and develop alternative parameterisations of the ~~damage~~ injury functions to improve the capability of the O-CN model to simulate the whole-tree biomass responses observed in the field experiments, with the notable exception that we explicitly simulate in-fumigation experiments and the approximate age of the trees. Finally, we explore whether or not there is a substantial difference in the biomass response to ozone of young or mature trees by using a sequence of model simulations and comparing the response both in terms of whole tree biomass as well as net primary production.

2 Methods

We use the O-CN terrestrial biosphere model (Zaehle and Friend, 2010), which is an extension of the ORCHIDEE model (Krinner et al., 2005) to simulate conditions of the ozone fumigation experiments described in Büker et al. (2015). The O-CN model, an average-individual dynamic vegetation model, simulates the terrestrial coupled carbon (C), nitrogen (N) and water cycles for up to twelve plant functional types and is driven by climate data and atmospheric composition.

O-CN simulates a multi-layer canopy with up to 20 layers with a thickness of up to 0.5 leaf area index each. Net photosynthesis is calculated according to a modified Farquhar-scheme for shaded and sun-lit leaves considering the light profiles of diffuse and direct radiation (Zaehle and Friend, 2010). Leaf nitrogen concentration and leaf area determine the photosynthetic capacity. Increases of the leaf nitrogen content increase V_{cmax} and J_{max} (nitrogen specific rates of maximum light harvesting, electron transport) and hence maximum net photosynthesis and stomatal conductance per leaf area. The leaf N content is highest at the top of the canopy and exponentially decreases with increasing canopy depth (Friend, 2001; Niinemets et al., 2015).

Following this net photosynthesis, stomatal conductance and ozone uptake are generally highest in the top canopy and decrease with increasing canopy depth.

Canopy-integrated assimilated carbon enters a labile non-structural carbon pool, which can either be used to fuel maintenance respiration (a function of tissue nitrogen), storage (for seasonal leaf and fine root replacement and buffer of inter-annual variability of assimilation) or biomass growth. The labile pool responds within days to changes in GPP, the long-term reserve has a response time of several months, depending on its use to support seasonal foliage and fine root development or sustain growth in periods of reduced photosynthesis. After accounting for reproductive production (flowers and fruits), biomass growth is partitioned into leaves, fine roots, and sapwood according to a modified pipe-model (Zaehle and Friend, 2010), accounting for the costs of biomass formation (growth respiration). In other words, changes in leaf-level productivity affect the build-up of plant pools and storage, and thereby feed back on the ability of plants to acquire C through photosynthesis, or nutrients through fine root uptake.

2.1 Ozone ~~damage~~ injury calculation in O-CN

Throughout the manuscript we refer to 'injury' for the biological response to O_3 uptake at the leaf level and to 'damage' for responses of plant production, growth and biomass at the ecosystem level following Guderian (1977). The relationship between ozone uptake and injury is called 'injury function'; the relationship between ozone uptake and damage is called 'dose-response-relationship'.

Leaf-level ozone uptake is determined by stomatal conductance and atmospheric O_3 concentrations, as described in Franz et al. (2017). To mimic the conditions of the fumigation experiments with plot-level controlled atmospheric O_3 concentrations, simulations are conducted with a model version of O-CN, in which atmospheric O_3 concentrations are directly used to calculate ozone uptake into the leaves, and the transfer and destruction of ozone between the atmosphere and the surface is ignored (ATM model version in Franz et al. (2017)). Deviating from Franz et al. (2017), stomatal conductance g_{st} here is calculated based on the Ball and Berry formulation (Ball et al., 1987) as

$$g_{st,l} = g_0 + g_1 \times \frac{A_{n,l} \times RH \times f(height_l)}{C_a} \tag{1}$$

where net photosynthesis ($A_{n,l}$) is calculated as described in Zaehle and Friend (2010) as a function of ~~leaf nitrogen and~~ the leaf internal partial pressure of CO_2, absorbed photosynthetic photon flux density on shaded and sunlit leaves, leaf temperature, as well as the nitrogen specific rates of maximum light harvesting, electron transport (J_{max}) and carboxylation rates (V_{cmax}). RH is the atmospheric relative humidity, $f(height_l)$ the water-transport limitation with canopy height, C_a the atmospheric CO_2 concentration, g_0 is the residual conductance when A_n approaches zero, and g_1 is the stomatal-slope parameter as in Krinner et al. (2005). The index l indicates that g_{st} is calculated separately for each canopy layer.

The stomatal conductance to ozone $g_{st,l}^{O_3}$ is calculated as

$$g_{st,l}^{O_3} = \frac{g_{st,l}}{1.51} \tag{2}$$

where the factor 1.51 accounts for the different diffusivity of O_3 from water vapour (Massman, 1998).

For each canopy layer the O_3 stomatal flux ($f_{st,l}$, $nmol\,m^{-2}(leaf\ area)\,s^{-1}$) is calculated from the atmospheric O_3 concentration the plants in the field experiments were fumigated with ($\chi_{atm}^{O_3}$) and $g_{st,l}$ as

$$f_{st,l} = (\chi_{atm}^{O_3} - \chi_i^{O_3}) g_{st,l}^{O_3}. \tag{3}$$

where the leaf internal O_3 concentration ($\chi_i^{O_3}$) is assumed to be zero (Laisk et al., 1989).

The accumulation of ozone fluxes above a threshold of Y $nmol\,m^{-2}(leaf\ area)\,s^{-1}$ ($f_{st,l,Y}$, $nmol\,m^{-2}(leaf\ area)\,s^{-1}$) with

$$f_{st,l,Y} = MAX(0, f_{st,l} - Y) \tag{4}$$

gives the $CUOY_l$. The canopy value of $CUOY$ is calculated by summing $CUOY_l$ over all canopy layers ~~(see Franz et al. (2017) for details)~~(Franz et al., 2017).

For comparison to observations, the Phytotoxic Ozone Dose (POD, $mmol\,m^{-2}$) 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). The accumulation of ozone fluxes of the top canopy layer above a threshold of y $nmol\,m^{-2}(leaf\ area)\,s^{-1}$ gives the $PODy$. The estimates of $PODy$ (both $POD2$ and $POD3$) can be used off-line to re-construct dose-response relationships equivalent to those described in Büker et al. (2015). These modelled dose-response relationships can then be compared with the empirically derived dose-response relationships to assess the ability of the model to estimate ~~damage~~injury. As such, the $POD2$ and $POD3$ used for the formation of these modelled dose-response relationships are purely diagnostic variables and not involved in the ~~damage~~ injury calculation of the model. The flux thresholds (2 and 3 $nmol\,m^{-2}(leaf\ area)\,s^{-1}$) are not the flux thresholds that are used to estimate biomass response in the O-CN model simulations.

Ozone ~~damage~~injury, i.e. the fractional loss of carbon uptake associated with ozone uptake $d_l^{O_3}$, is calculated as a linear function of the cumulative leaf-level uptake of ozone above a threshold of Y $nmol\,m^{-2}(leaf\ area)\,s^{-1}$ ($CUOY_l$)

$$d_l^{O_3} = a - b \times CUOY_l \tag{5}$$

where a is the intercept and b is the slope of the ~~damage~~ injury function. The ~~damage~~ injury fraction ($d_l^{O_3}$) is calculated separately for each canopy layer l based on the specific accumulated ozone uptake of the respective canopy layer ($CUOY_l$), and takes values between 0 and 1. The magnitude of $d_l^{O_3}$ in Eq. 5 varies between the canopy layers because $CUOY_l$ varies driven by within-canopy gradients in stomatal conductance and photosynthetic capacity.

The effect of ozone ~~damage~~ injury on plant carbon uptake is calculated by

$$x^{O_3}{}_{n,ll} = x_{n,ll}(1 - d_l^{O_3}). \tag{6}$$

where ~~x~~ x_l is either leaf-level net photosynthesis $A_{n,l}$ or the maximum photosynthetic capacity ($J_{max,l}$ and $V_{cmax,l}$), which is used in the calculation of $A_{n,l}$. $J_{max,l}$ and $V_{cmax,l}$ are reduced in proportion such that the ratio between the two is not altered. While there is some evidence that ozone can affect the ~~ration~~ ratio between J_{max} and V_{cmax}, we believe that for the purpose of this paper ~~is~~ it is justifiable to assume a fixed ratio between them.

Reductions in A_n, l cause a decline in stomatal conductance ($g_{st,l}$) due to the tight coupling between both. Other stress factors that impact $g_{st,l}$ are accounted for in the preceding calculation of the $g_{st,l}$ ~~undamaged~~ uninjured by ozone (see Eq. 1). Reductions in $g_{st,l}$ decrease the O_3 uptake into the plant ($f_{st,l}$) and slow the increase in $CUOY_l$ and thus ozone ~~damage~~injury.

2.2 Model set-up

Four published ~~damage~~ injury functions were applied within the O-CN model (see Tab. 1 for the respective slopes, intercepts and flux thresholds). As shown below in Fig. 1 and explained in the results section these did not match well with the observed biomass dose-response relationships by Büker et al. (2015)~~, we~~. Following this we manually calibrated two additional ~~damage~~ injury relationships, one each for A_n or V_{cmax}, based on the data presented in Büker et al. (2015) (see Tab. 1 for slopes and intercepts). For these calibrated ~~damage~~ injury functions, we chose a flux threshold value of 1 $nmol\,m^{-2}(leaf\,area)\,s^{-1}$, as suggested by LRTAP-Convention (2017). We forced the intercept (a) of these relationships to one to simulate zero ozone ~~damage~~ injury at zero accumulated O_3 (for ozone levels that cause less then 1 $nmol\,m^{-2}(leaf\,area)\,s^{-1}$ instantaneous ozone uptake). As described above, in all model versions, ozone ~~damage~~ injury is calculated independently for each canopy layer

based on the accumulated O_3 uptake ($CUOY_l$) in that layer, above a specific flux threshold of $Y\ nmol\,m^{-2}(leaf\ area)\,s^{-1}$ for the respective ~~damage~~ injury function (see Tab. 1).

Table 1. Slopes and intercepts, partly PFT specific, of all four published ($W07_{PS}$, $L12_{PS}$, $L12_{VC}$, $L13_{PS}$) and two tuned (tun_{PS}, tun_{VC}) ~~damage~~ injury functions included in O-CN. Targets of ozone ~~damage~~ injury are net photosynthesis (PS) or V_{cmax}. ~~Damage~~ Injury calculations base on the $CUOY$ with a specific flux threshold for each ~~damage~~ injury function.

ID	Target	Slope (b)	Intercept (a)	Plant group	Flux threshold $[nmol\,m^{-2}(leaf\ area)\,s^{-1}]$	Reference
$W07_{PS}$	PS	0.0022	0.9384	All	0	Wittig et al. (2007)
$L12_{PS}$	PS	0.2399	1.0421	All	0.8	Lombardozzi et al. (2012a)
$L12_{VC}$	V_{cmax}	0.1976	0.9888	All	0.8	Lombardozzi et al. (2012a)
$L13_{PS}$	PS	0	0.8752	Broadleaf	0.8	Lombardozzi et al. (2013)
$L13_{PS}$	PS	0	0.839	Needleleaf	0.8	Lombardozzi et al. (2013)
tun_{PS}	PS	0.065	1	Broadleaf	1	tuned here
tun_{PS}	PS	0.021	1	Needleleaf	1	tuned here
tun_{VC}	V_{cmax}	0.075	1	Broadleaf	1	tuned here
tun_{VC}	V_{cmax}	0.025	1	Needleleaf	1	tuned here

2.3 Model and protocol for young trees

~~Simulations~~ Single point simulations were run for each fumigation experiment using meteorological input from the daily CRU-NCEP climate data set (CRU-NCEP version 5; LSCE (http://dods.extra.cea.fr/store/p529viov/cruncep/V5_1901_2013/) at the nearest grid cell to the coordinates of the experiment sites. The meteorological data provided by the experiments were incompletely describing the atmospheric boundary conditions required to drive the O-CN model. Atmospheric CO_2 concentrations were taken from Sitch et al. (2015), and reduced as well as oxidised nitrogen deposition in wet and dry forms were provided by the EMEP model (Simpson et al., 2014). Hourly O_3 concentrations were obtained from the experiments, as in Büker et al. (2015).

Büker et al. (2015) report data for eight tree species at 11 sites across Europe (see Tab. A.2 for experiment and simulation details). The O-CN model simulates twelve plant functional types (PFT's) rather than explicit species, therefore the species from the experiments were assigned to the corresponding PFT: All broadleaved species except *Quercus ilex* were assigned to the temperate broadleaved summergreen PFT. *Quercus ilex* was classified as temperate broadleaved evergreen PFT. All needle-leaved species were assigned to the temperate needle-leaved evergreen PFT.

The field experiments were conducted on young trees or cuttings. Prior to the simulation of the experiment, the model was run in an initialisation phase from bare ground until the simulated stand-scale tree age was stable and representative of 1-2 year old seedlings. During this initialisation, O-CN was run with the climate of the years preceding the experiment and zero atmospheric O_3 concentrations. Using ambient ozone concentrations during the initialisation phase would have resulted

in different initial biomass values for the different response functions, which would have reduced the comparability of the different model runs. The impact of the ozone concentrations in the initialisation phase on our results here can be considered negligible since we only evaluate the simulated biomass from different treatments in relation to each other and do not evaluate it in absolute terms.

The duration of the initialisation phase depends on the site and PFT and averages 7.8 years (mean over all simulated experiments). Some of the published ~~damage~~ injury functions and/or parameterisations applied have intercepts unequal to one (a in Eq. 5; see Tab. 1), which induces reductions ($a < 1$) or increases ($a > 1$) in photosynthesis at zero ozone concentration and thus causes a bias in biomass and in particular foliage area at the end of the initialisation phase. To eliminate this bias, the nitrogen-specific photosynthetic capacity of a leaf was adjusted for each of the six parameterisations of the model to obtain comparable LAI values at the beginning of the experiment (see Tab. A.1). This adaption of the nitrogen-specific photosynthetic capacity of a leaf only counterbalances the fixed increases or decreases in the calculation of photosynthesis implied by the intercepts unequal to 1 and has no further impact on ozone uptake and ~~damage~~ injury calculations.

The simulations of the experiments relied on the meteorological and atmospheric forcing of the experiment years. Simulations were made for all reported O_3 treatments of the specific experiment, including the respective control treatments. Büker et al. (2015) obtained estimates of biomass reductions due to ozone by calculating the hypothetical biomass at zero ozone uptake for all experiments that reported ozone concentrations greater than zero for the control group (e.g. for charcoal filtered or non-filtered air) and calculated the biomass damage from the treatments against a completely undamaged biomass. Our model allows us to run simulations with zero ozone concentrations and skip the calculation of the hypothetical biomass at zero ozone concentrations as done by Büker et al. (2015). Following this, we ran additional reference simulations with zero O_3 and based our biomass damage calculations upon them.

2.4 Modelling protocol for mature trees

To test whether biomass dose-response relationships of mature forests will show a similar relationship as observed in the simulations of young trees, we ran additional simulations with mature trees. To allow the development of a mature forest where biomass accumulation reached a maximum, and high, and medium turnover soil pools reached an equilibrium, the model was run for 300 years in the initialisation phase. The simulations were conducted with the respective climate previous to the experiment period and zero atmospheric O_3 concentration. For the simulation years previous to 1901 the yearly climate is randomly chosen from the years 1901-1930. Constant values of atmospheric CO_2 concentrations are used in simulated years previous to 1750 followed by increasing concentrations up to the experiment years. The subsequent experiment years are simulated ~~identical as in the~~ in the same way as the simulations with the young trees. The ozone injury for mature trees is calculated based on the same tun_{VC} injury function (see Tab. 1) that is used in the simulation of young trees.

2.5 Calculation of the biomass damage relationships

The ozone induced biomass damage is calculated from the difference between a treatment and a control simulation. At each experiment site and for all treatments the annual reduction in biomass due to ozone (RB) is calculated as in Büker et al. (2015):

$$RB = \left(\frac{BM_{treat}}{BM_{zero}} \right)^{\frac{1}{n}}, \tag{7}$$

where BM_{treat} represents the biomass of a simulation, which experienced an O_3 treatment and BM_{zero} the biomass of the control simulation with zero atmospheric O_3 concentration. The exponent imposes an equal fractional biomass reduction across all simulation years for experiments lasting longer than one year.

Büker et al. (2015) report the dose-response relationships for biomass reduction with reference to the Phytotoxic Ozone Dose ($PODy$) with flux thresholds y of 2 and 3 $nmol\,m^{-2}(leaf\,area)\,s^{-1}$ (POD_2 and POD_3) for the needleleaf and broadleaf category~~respectively~~, respectively, where the $PODy$ values were derived from simulations with the DO_3SE model (Emberson et al., 2000) given site-specific meteorology and ozone concentrations. To be able to compare the simulated biomass reduction by O-CN with these estimates, we also diagnosed these $PODy$ values for each simulation from the accumulated ozone uptake of the top canopy layer ($PODy_{O-CN} = CUOY_{l=1}$). Note that the $PODy_{O-CN}$ is purely diagnostic, and not used in the ~~damage~~ injury calculations, which are based on the $CUOY_l$ (see Eq. 5). As O-CN computes continuous, half-hourly values of ozone uptake (see Franz et al. (2017) for details), the $PODy_{O-CN}$ values have to be transformed to be comparable to the simulated mean annual $PODy$ values reported in Büker et al. (2015). For deciduous species, the yearly maximum of $PODy_{O-CN}$ was taken as yearly increment $PODy_{O-CN,i}$. The $PODy_{O-CN}$ of evergreen species was continuously accumulated over several years. To obtain the yearly increment $PODy_{O-CN,i}$, the $PODy_{O-CN}$ at the beginning of the year i is subtracted from the $PODy_{O-CN}$ at the end of the year i.

The selected yearly $PODy_{O-CN,i}$ were used to calculate mean annual values necessary for the formation of the dose-response relationships integrating all simulation years ($PODy^{dr}$) as

$$PODy_i^{dr} = \frac{\sum_{k=1}^{i} PODy_{O-CN,i}}{i} \tag{8}$$

where $PODy_{O-CN,i}$ is the $PODy$ of the i-th year calculated by O-CN. The $PODy^{dr}$ values are used to derive biomass dose-response relationships.

Separate biomass dose-response relationships were estimated by grouping site data for broadleaved and needleleaved species. The biomass dose-response relationships are obtained from the simulation output by fitting a linear model to the simulated values of RB and $PODy^{dr}$ (with flux thresholds of 2 and 3 $nmol\,m^{-2}(leaf\,area)\,s^{-1}$ for needleleaved and broadleaved species, respectively), where the regression line is forced through one at zero $PODy^{dr}$. Büker et al. (2015) report two alternative dose-response relationships for their data set, the simple and the standard model, B_{SI} and B_ST, respectively. We evaluate our different model versions regarding their ability to reach, with the biomass-dose-response relationships computed from their output, the area between those two functions (target area). The tuned ~~damage~~ injury relationships tun_{PS} and tun_{VC} were obtained by adjusting the slope b in Eq. 5 such that the corresponding biomass dose-response relationships fits the target area.

The intercept of the ~~damage~~ injury relationships are forced to 1 to simulate zero ozone ~~damage~~ injury at ozone fluxes lower than 1 $nmol\,m^{-2}(leaf\ area)\,s^{-1}$.

3 Results

3.1 Testing published ~~damage~~ injury functions

None of the versions where ozone ~~damage~~ injury is calculated based on previously published ~~damage~~ injury functions fit the observations well. Some versions strongly overestimate the simulated biomass dose-response relationship and others strongly underestimate it (see Fig. 1) compared to the dose-response relationships developed by Büker et al. (2015).

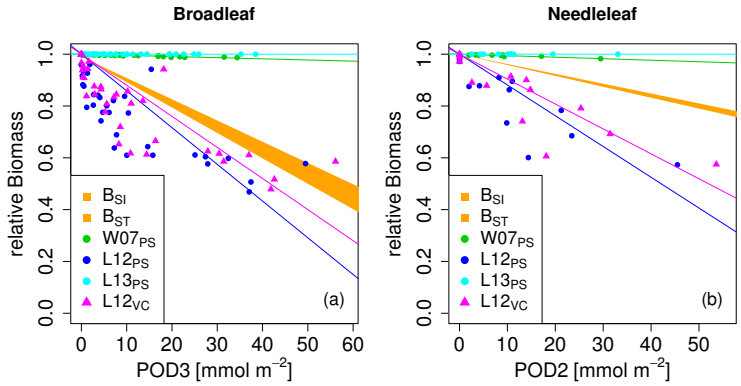

Figure 1. Biomass dose-response relationships for simulations based on published ~~damage~~ injury relationships, separate for a) broadleaved species, and b) needle-leaved species. The dose-response relationships by Büker et al. (2015), B_{SI} and B_{ST}, define the target area (orange). The displayed dose-response relationships are simulated by model versions which base ~~damage~~ injury calculations either on net photosynthesis W07$_{PS}$ (Wittig et al., 2007), L12$_{PS}$(Lombardozzi et al., 2012a), and L13$_{PS}$ (Lombardozzi et al., 2013), or on V_{cmax} L12$_{VC}$ (Lombardozzi et al., 2012a) (see Tab. 1 for more details). See Tab. A.3 and A.4 for slopes, intercepts, R^2 and p-values of the displayed regression lines. ~~Damage~~ Injury calculation in the simulations bases on $CUOY$ (see Tab. 1) and not on $POD2$ or $POD3$ (see Sec. 2.5 for more details).

In the W07$_{PS}$ simulations, where ~~damage~~ injury is calculated based on the ~~damage~~ injury function by Wittig et al. (2007), biomass damage is strongly underestimated compared to the estimates from Büker et al. (2015). Ozone ~~damage~~ injury estimates are mainly driven by the intercept of the relationship, which assumes a reduction of net photosynthesis by 6.16% at zero ozone uptake. Little additional ozone damage occurs due to the accumulation of ozone uptake. As a consequence, the ozone treatments and reference simulations differ little in their simulated biomass. Similarly, the Lombardozzi et al. (2013) ~~damage~~ injury function (L13$_{PS}$) calculates ozone ~~damage~~ injury as a fixed reduction of net photosynthesis independent of the actual accumulated ozone uptake. The reference simulations with zero atmospheric ozone thus equals the simulations with ozone treatments and results in an identical simulated biomass. We tested accounting for effects of ozone on stomatal conductance besides net photosynthesis as suggested by Lombardozzi et al. (2013). However, this additional direct ~~damage~~ injury to stomatal conductance yielded a minimal decrease in simulated biomass accumulation in needle-leaved trees, but did not qualitatively

change the results (results not shown). These results indicate that ~~damage~~ injury functions, with a large intercept and a very shallow (or non-existing) slope cannot simulate the impact of spatially varying O_3 concentrations or altered atmospheric O_3 concentrations.

The simulations $L12_{PS}$ and $L12_{VC}$ (net photosynthesis and V_{cmax} ~~damage~~ injury according to Lombardozzi et al. (2012a), respectively) strongly overestimate biomass damage compared to Büker et al. (2015). Both ~~damage~~ injury functions assume an extensive ~~damage~~ injury to carbon fixation at low ozone accumulation values ($CUOY$) of about 5 $mmol\ O_3$. This results in a very steep decline in relative biomass at low values of $POD3$. Notably, despite a linear ~~damage relationship~~injury function, the very steep initial decline in biomass of broadleaved trees at low values of $POD3$ is not continued at higher exposure, resulting in a non-linear biomass dose-response relationships. Higher accumulation of ozone doses does not result in higher ~~damage~~ injury rates beyond a threshold of about 5 $mmol\ O_3\ m^{-2}$ leaf area, and relative biomass declines remain 50 to 70 %. Whereas non-linear dose-response relationships are observed in experiments e.g. for leaf injury (Marzuoli et al., 2009), such a non-linear relationship is not produced in the biomass dose-response relationship by Büker et al. (2015).

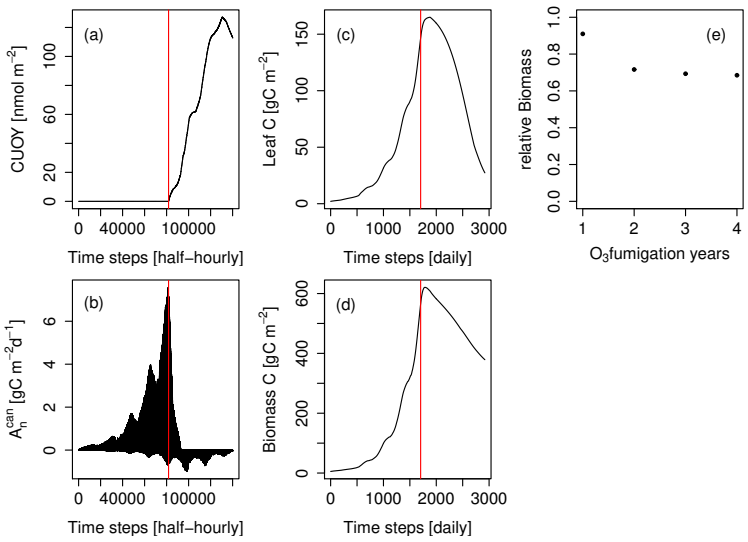

Figure 2. ~~Cumulative~~ Simulated cumulative ozone uptake above a threshold of 0.8 $nmol\,m^{-2}(leaf\ area)\,s^{-1}$ ($CUOY$), canopy integrated net photosynthesis (A_n^{can}), leaf carbon content ($Leaf\ C$), total carbon in biomass ($Biomass\ C$) and relative Biomass (RB) of *Pinus halepensis* at the Ebro Delta fumigated with the NF+ ozone treatment. Simulations are conducted with the $L12_{PS}$ model version. Panels a-d display the entire simulation period. The red line indicates the onset of O_3 fumigation (NF+) in the 5th of 8 simulations years. The relative biomass compared to a control simulation with zero O_3 concentration (panel e) is displayed for the O_3 fumigation years.

We investigated the cause for this at the example of the *Pinus halepensis* stand in the Ebro Delta with a high ozone treatment as shown in Fig. 2. The simulated $CUOY$ quickly increases after the onset of fumigation (Fig. 2a) and is paralleled by a rapid decline in canopy integrated net photosynthesis (A_n^{can}, see Fig. 2b). Once all canopy layers accumulated more than 5 $mmol\ O_3\ m^{-2}$, the canopy photosynthesis is fully reduced, and A_n^{can} becomes negative as a consequence of ongoing leaf maintenance respiration. Thereafter, leaf and total biomass steadily decline (Fig. 2c,d), and the plants are kept alive only by the consumption

of stored non-structural carbon reserves. Despite the 100 % reduction in gross photosynthesis, the biomass compared to a control simulation (relative biomass, RB) reaches only values of approximately 0.7 (Fig. 2e), because of the remaining woody and root tissues (see Eq. 7 for the calculation of RB).

3.2 Tuned ~~damage~~ injury relationships

We next tested whether a linear ~~damage~~ injury function is in principle able to reproduce the observed biomass dose-response relationships. Simulations conducted with our tuned ~~damage~~ injury relationships produce biomass dose-response relationships which fit the target area defined by the B_{SI} and B_{ST} dose-response relationships by Büker et al. (2015) (see Fig. 3 and Tab. A.5, A.6). For the calibrated relationships used in these simulations, we chose a flux threshold value of 1 $nmol\,m^{-2}(leaf\,area)\,s^{-1}$, as suggested by LRTAP-Convention (2017). We forced the intercept (a) of these relationships through 1, to simulate zero ozone ~~damage~~ injury at ozone fluxes lower than 1 $nmol\,m^{-2}(leaf\,area)\,s^{-1}$. The resulting slope of the tun$_{PS}$ function for broadleaved PFTs is approximately 30 times higher compared to the slope suggested by Wittig et al. (2007) and a fourth of the slope by Lombardozzi et al. (2012a). For the needle-leaved PFT, the tuned slope (tun$_{PS}$) is approximately 10 times higher (lower) than the slopes by Wittig et al. (2007) and Lombardozzi et al. (2012a), respectively. Notably, we did not observe any difference in the model performance irrespective of whether net photosynthesis or photosynthetic capacity (V_{cmax} and simultaneously J_{max}) was reduced.

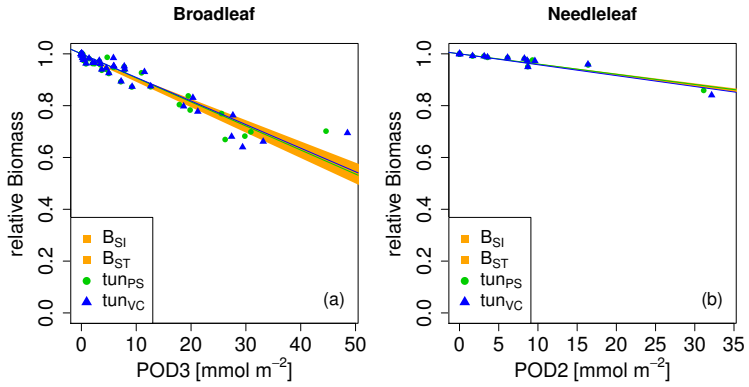

Figure 3. Biomass dose-response relationships for simulations based on tuned ~~damage~~ injury functions (see Tab. 1 for abbreviations), separate for a) broadleaved species, and b) needle-leaved species. The dose-response relationships by Büker et al. (2015), B_{SI} and B_{ST}, define the target area (orange). See Tab. A.5 and A.6 for slopes, intercepts, R^2 and p-values of the displayed regression lines. ~~Damage~~ Injury calculation in the simulations base on $CUO1$ (see Tab. 1) and not on $POD2$ or $POD3$ (see Sec. 2.5 for more details).

3.3 Ozone ~~damage~~ injury to mature trees

The simulation of young trees (simulated as in the previous section) compared to adult trees with the same model version reveals a distinct difference between the simulated versus observed dose-response relationship when expressed as reduction of biomass. Ozone ~~damage~~ injury causes a much shallower simulated biomass dose-response relationship for adult trees (tun$_{VC}^{mature}$ in

Fig. 4a,b) compared to young trees (tun_{VC}^{young} in Fig. 4a,b), both for broadleaved and needle-leaved species. It is worth noting that this is primarily the consequence of the higher initial biomass of the adult trees before ozone fumigation starts (tun_{VC}^{mature}). Comparing the dose-response relationship of young and mature trees based on the annual net biomass production (NPP) shows nearly identical slopes for needle-leaved species (Fig. 4d and Tab. 3), whereas the slopes for broadleaved tree species (Fig. 4c and Tab. 2) suggests only a slightly lower reduction in NPP in mature compared to young trees, likely related to the larger amount of non-structural reserves that increases the resilience of mature versus young trees.

Table 2. Slopes and intercepts of biomass (RB) and NPP (RN) dose-response relationships (DRR) for broadleaved species simulated by the tun_{VC} model version (see Tab. 1). The fumigation of young trees (tun_{VC}^{young}) with O_3 is compared to the fumigation of mature trees (tun_{VC}^{mature}).

DRR	ID	Intercept (a)	Slope (b)	R^2	p-value
RB	tun_{VC}^{young}	1	0.0091	0.93	5e-25
RB	tun_{VC}^{mature}	1	0.00142	0.91	9.8e-23
RN	tun_{VC}^{young}	1	0.0167	0.96	6.2e-30
RN	tun_{VC}^{mature}	1	0.0144	0.93	1.4e-24

Table 3. Slopes and intercepts of biomass (RB) and NPP (RN) dose-response relationships (DRR) for needle-leaved species simulated by the tun_{VC} model version (see Tab. 1). The fumigation of young trees (tun_{VC}^{young}) with O_3 is compared to the fumigation of mature trees (tun_{VC}^{mature}).

DRR	ID	Intercept (a)	Slope (b)	R^2	p-value
RB	tun_{VC}^{young}	1	0.0042	0.93	2.2e-09
RB	tun_{VC}^{mature}	1	0.000785	0.79	4.2e-06
RN	tun_{VC}^{young}	1	0.00858	0.97	2.3e-12
RN	tun_{VC}^{mature}	1	0.00808	0.99	3 .7e-16

4 Discussion

~~Damage~~ Injury functions that relate accumulated ozone uptake to fundamental plant processes such as photosynthesis are a key component for models that aim to estimate the potential impacts of ozone pollution on forest productivity, growth and carbon sequestration. We tested four published ~~damage~~ injury functions for net photosynthesis and V_{cmax} within the framework of the O-CN model to assess their ability to reproduce the empirical whole tree biomass dose-response relationships derived by Büker et al. (2015). The biomass dose-response relationships calculated from the O-CN simulations show that the parameterisation of the ~~damage~~ injury functions included in the model have a large impact on the simulated whole tree biomass: the published

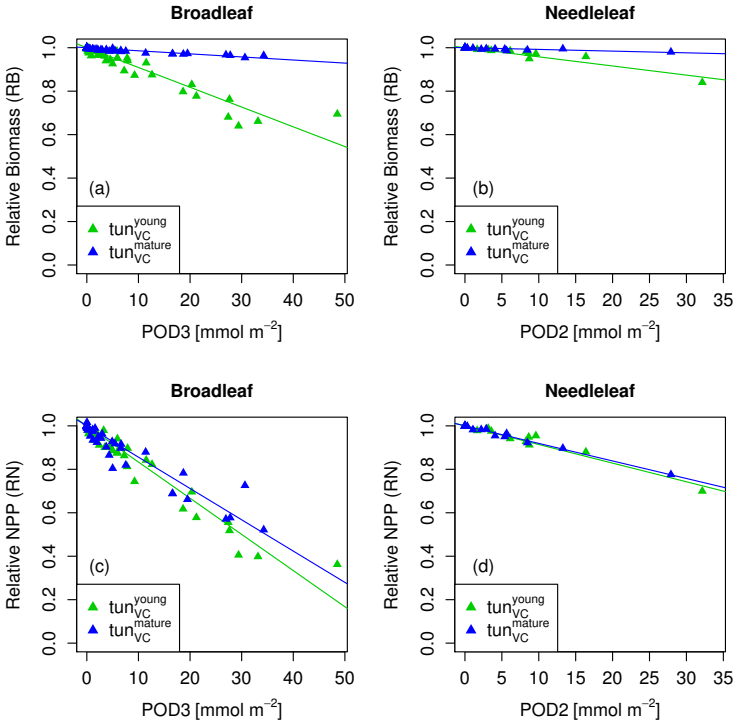

Figure 4. Biomass (RB) and NPP (RN) dose-response relationships of simulations with young (tun$_{VC}^{young}$) and mature trees (tun$_{VC}^{mature}$) separate for a,c) Broadleaf species, and b,d) Needleleaf species.

~~damage~~ injury functions either substantially over- or substantially under-estimated whole tree biomass reduction compared to the data presented by Büker et al. (2015).

The simulation results from the O-CN version applying a ~~damage~~ injury function based on a single, ozone-sensitive species (Lombardozzi et al., 2012a) to a range of European tree species leads to a strong overestimation of the simulated biomass damage compared to the observations used in this study. The problem of using such ~~damage~~ injury parameterisations based on short-term experiments of ozone-sensitive species is further highlighted when applying them in simulations of multiple season fumigation experiments and/or high ozone concentrations. Under such conditions, fumigation with high O_3 concentrations can lead to lethal doses, which might not be observed in field experiments due to restricted experiment lengths. Previous studies have suggested that in large areas of Europe, the Eastern US and South-East Asia average growing season values of $CUOY$ for recent years range between 10-100 $mmol\ O_3\ m^{-2}$ (Lombardozzi et al., 2015; Franz et al., 2017). The ~~damage~~ injury relationships L12$_{PS}$ and L12$_{VC}$ by Lombardozzi et al. (2012a) assume a 100% ~~damage~~ injury to net photosynthesis or V_{cmax} at accumulation values of about 5 $mmol\ O_3\ m^{-2}$. This would imply that in these large geographic regions, photosynthesis would have been completely impaired by ozone, which is clearly not the case. This result highlights the need for a representative set of species for the development of ~~damage~~ injury functions for large-scale biosphere models. Overall, our results suggests that the estimates of global GPP reduction as a result of ozone pollution by Lombardozzi et al. (2012a) are strongly overestimated.

Meta-analyses (Wittig et al., 2007; Lombardozzi et al., 2013) are designed to minimise the effect of species-specific ozone sensitivities and provide estimates of the average species response. However, we found that the relationships derived by these meta-analyses substantially underestimate biomass damage. Technically, the reasons for this are a weak or non-existent increase of the ozone ~~damage~~ injury with increased ozone uptake (shallow or non-existent slopes) and/or high ozone ~~damage~~ injury at zero accumulated ozone uptake (intercept lower than one). Apparently, the diversity of species responses and experimental settings that are assembled in the meta-analyses by Wittig et al. (2007) and Lombardozzi et al. (2013), together with uncertainties in precisely estimating accumulated ozone uptake in these databases preclude the identification of ~~damage~~ injury functions that are consistent with the damage estimates by Büker et al. (2015). The high intercepts in the meta-analyses by Wittig et al. (2007) and Lombardozzi et al. (2013), which assume a considerable ~~damage~~ injury fraction even when no ozone is taken up at all, seem to be ecologically illogical and suggest that an alternative approach is necessary to simulate ozone ~~damage~~injury. As a consequence of these points, the Europe-wide GPP reduction estimates by Franz et al. (2017), which has been based on the ~~damage~~ injury function by Wittig et al. (2007), may substantially underestimate actual GPP reduction. Similarly, global estimates as well as spatial variability of ozone damage to GPP by Lombardozzi et al. (2015), based on Lombardozzi et al. (2013), are virtually independent of actual ozone concentrations or uptake for all tree plant functional types and should be interpreted with caution.

A crucial aspect in forming dose-response relationships is the calculation of the accumulated ozone uptake (e.g. $PODy$ or $CUOY$). The calculation of accumulated ozone uptake is realised in different ways in the meta-analyses and the study by Büker et al. (2015) as well as in our approach here. Experiments synthesised in the meta-analyses generally do not have access to stomatal conductance values at high resolution measured throughout the experiment, which impedes precise determination of O_3 uptake. The uncertainty in the necessary approximations of accumulated ozone uptake can be assumed to be considerable, and it is thus highly recommendable to measure and report required observations in future ozone fumigation experiments. Büker et al. (2015) use the DO_3SE model to simulate ozone uptake and accumulation similar as done in our model here. These modelled values for ozone uptake and accumulation can assumed to be more reliable since both models simulate processes that determine ozone uptake continuously for the entire experiment length at high temporal resolution. They account for diurnal changes in stomatal conductance as well as climate factors restricting stomatal conductance and hence ozone uptake. However, both models vary in their complexity of the simulated plants, carbon assimilation, and growth processes, which will also impact the ~~estimate~~ estimates of ozone accumulation ($PODy$) and hence their suggested biomass dose-response-relationships.

The meta-analyses do not account for non-stomatal ozone deposition (e.g. to the leaf cuticle or soil), which imposes a bias towards overestimating ozone uptake and accumulation contrary to the DO_3SE model used by Büker et al. (2015), which accounts for this. The O-CN model in principle can simulate non-stomatal ozone deposition from the free atmosphere to ground level (see Franz et al. (2017)). The leaf boundary layer is implicitly included into the calculation of the aerodynamic resistance of O-CN and included in Franz et al. (2017). However, for the simulation of the chamber experiments we used the observed chamber O_3 concentrations, rather than estimating the canopy-level O_3 concentration based on the free atmosphere (approximately 45 m above the surface) and atmospheric turbulence. This required not accounting for aerodynamic resistance

and therefore the leaf-boundary layer resistance as well as it prevented the calculation of the non-stomatal deposition, which may lead to a slight overestimation of ozone uptake and accumulation in our simulations.

The calibration of ~~damage~~ injury functions to net photosynthesis and V_{cmax} shows that in principle, the linear structure of Eq. 5 is sufficient to simulate biomass dose-response relationships comparable to Büker et al. (2015) in O-CN. An advantage of the ~~damage~~ injury functions derived here compared to previously published ~~damage~~ injury functions (Wittig et al., 2007; Lombardozzi et al., 2012a, 2013) is the intercept of one, implying that simulated ozone ~~damage~~ injury is zero at zero accumulated O_3 and steadily increases with increased ozone accumulation. The flux threshold used in the simulations is 1 $nmol\,m^{-2}(leaf\,area)\,s^{-1}$ as suggested by the LRTAP-Convention (2017). Since the tuned ~~damage~~ injury functions are structurally identical to previously published ~~damage~~ injury functions based on accumulated ozone uptake they can be directly compared to them. Slopes of the tuned ~~damage~~ injury functions lie in between the values proposed by Wittig et al. (2007) and Lombardozzi et al. (2012a) and thus take values in an expected range. We did not find any significant difference in simulated biomass responses between the use of net photosynthesis or leaf-specific photosynthetic capacity (V_{cmax}) as a target for the ozone ~~damage~~ injury function, although we do note that the slopes were slightly lower for the net photosynthesis based functions. The simulation of ozone effects on leaf-specific photosynthetic capacity (V_{cmax}) seems preferable over the adjustment of net photosynthesis, because V_{cmax} and J_{max} are parameters in the calculation of net photosynthesis, and thus are likely easier transferable between models. Models with different approaches to simulate net photosynthesis might obtain better comparable results by using ~~damage~~ injury relationships that target V_{cmax} instead of net photosynthesis.

All ~~damage~~ injury functions included into the O-CN model base ~~damage~~ injury calculations on the ~~damage~~ injury index $CUOY$ (canopy value) rather than $PODy$, as used by some other models, e.g. the DO_3SE model (Emberson et al., 2000). We tested the effect of basing the ~~damage~~ injury calculation on $POD1$ rather than $CUO1$, and found that these produced comparable biomass dose-response relationships as the ~~damage~~ injury relationships based on $CUO1$ presented in Fig. 3 (results not shown). The slopes of ~~damage~~ injury functions based on $POD1$ are approximately two thirds and half compared to the slopes based on $CUO1$ for broadleaved and needle-leaved species, respectively. The difference in the slope values associated with $POD1$ and $CUO1$ results from the different calculation, and application of them. ~~The $POD1$~~ $PODy$ is calculated in the top canopy layer and the respective ~~damage fraction is applied for~~ injury fraction is then applied uniformly to all canopy layers~~, the~~ . $CUOY$ ~~though~~ and the associated injury fraction is calculated separately ~~in~~ for each canopy layer ~~as well as the respective damage fraction.~~ and varies with the canopy profile of stomatal conductance, and therefore the distribution of light and photosynthetic capacity (other factors such as vertical gradients of temperature or ozone are currently not represented in OCN). More analysis of the gradients of ozone injury within deep canopies are required to evaluate whether the scaling of top-of-the-canopy injury to whole canopy injury is appropriate or if alternative simulation approaches need to be developed. Higher frequency data on the ozone ~~damage~~ injury incurred by plants are required to disentangle whether an ozone ~~damage~~ injury parameterisation based on instantaneous (e.g. similar to the approach by Sitch et al. (2007)) or accumulated ozone uptake results in a more accurate simulation of the seasonal effects ~~and more analysis of the differential effect of ozone damage within deep canopies are required to evaluate whether the scaling of top-of-the-canopy damage to whole canopy damage is appropriate~~of ozone fumigation.

Further aspects that determine ozone sensitivity and damage to carbon gain of plants like leaf morphology (Calatayud et al., 2011; Bussotti, 2008), different sensitivity of sunlit and shaded leafs (Tjoelker et al., 1995; Wieser et al., 2002), early senescence (Gielen et al., 2007; Ainsworth et al., 2012) and costs for detoxification of ozone and/or repair of ozone ~~damage~~ injury that likely increases the plant's respiration costs (Dizengremel, 2001; Wieser and Matyssek, 2007) are not considered by either approach. Marzuoli et al. (2016) observed an ozone induced reduction of biomass but no significant reduction in physiological parameters like V_{cmax}. They suggest that the reduced growth is caused by higher energy investments and reducing power for the detoxification of ozone whereas the photosynthetic apparatus remained ~~undamaged~~ uninjured (Marzuoli et al., 2016).

Species within the same plant functional type are known to exhibit different sensitivities to ozone (Wittig et al., 2007, 2009; Mills et al., 2 This suggests that the application of a single injury function for a large set of species and plant functional types may not be sufficient to yield reliable estimates of large scale damage estimates. Species interaction and competition, differing genotypes and individuals ontogeny may further alter ozone impacts on plants and ecosystems (Matyssek et al., 2010). For instance, a modelling study using an individual-based forest model showed that ozone may not reduce the carbon sequestration capacity in forests if at the ecosystem level the reduced carbon fixation of ozone-sensitive species are compensated for by an increased carbon fixation of less ozone-sensitive species (Wang et al., 2016). First generation dynamic global vegetation models such as OCN do not simulate separate species but are based on plant functional types, which combine a large set of species. This restricts *per se* the ability of global models to simulate ozone-induced community dynamics, and may therefore lead to overestimates of the net ozone impact if the parameterisation of the damage functions is entirely based on ozone-sensitive species. In our study, we have presented an approach to use the existing experimental evidence to parameterise a globally applicable model in a simple design to generate injury functions which are based on a relevant range of species rather than relying on species-specific injury functions as a first step towards a more reliable parameterisation of large-scale ozone damage.

Some studies have found that ozone-affected stomata respond much slower to environmental stimuli than unaffected cells (Paoletti and Grulke, 2005), which can delay closure and trigger, stomatal sluggishness, an uncoupling of stomatal conductance and photosynthesis (Reich, 1987; Tjoelker et al., 1995; Lombardozzi et al., 2012b) and thus impact transpiration rates (Mills et al., 2009; Paoletti and Grulke, 2010; Lombardozzi et al., 2012b) and the plant's water use efficiency (Wittig et al., 2007; Mills et al., 2009; Lombardozzi et al., 2012b). The O-CN model is able to directly impair stomatal conductance, by uncoupling ~~damage~~ injury to net photosynthesis from the subsequent ~~damage~~ injury to stomatal conductance. In this version of the O-CN model both net photosynthesis and stomatal conductance can directly be ~~damaged by individual damage~~ injured by individual injury functions. The simulation of this kind of direct ~~damage~~ injury to stomatal conductance additional to the ~~damage~~ injury of net photosynthesis, both according to the ~~damage~~ injury functions by (Lombardozzi et al., 2013), have a negligible impact on biomass production compared to not accounting for direct ~~damage~~ injury to the stomata (results not shown). However, our above mentioned concerns regarding the structure of the ~~damage~~ injury relationships by Lombardozzi et al. (2013) should be taken into account when considering this result.

A key challenge for the use of fumigation experiments to parameterise ~~ozone-damage~~ ozone-injury in models is that trees (as opposed to grasses fumigated from seeds) typically possess a certain amount of biomass at the beginning of the fumigation

experiment. Even at lethal ozone doses, the relative biomass thus can not decline to zero, and tree death may occur at values of a relative biomass greater than zero. The relative biomass is positive even if carbon fixation is fully reduced and the plants survive due to the use of stored carbon. The higher the initial biomass and the slower the annual biomass growth rate of the tree is, the harder it is to obtain low values of RB. When comparing RB values obtained from trees with substantial different initial biomass and tree species with different growth rates proportionate damage rates thus can not directly be inferred. This indicates that the explanatory value of the relative biomass between a control and a treatment to estimate long-term plant damage at a given O_3 concentration is limited. This is particularly the case when evaluating the damage of more mature forests. The simulated biomass dose-response relationships of adult trees are much more shallow than dose-response relationships of young trees (see Fig. 4), because of the high initial biomass prior to fumigation. This suggests that the use of biomass ~~damage~~ injury functions derived from experiments with young trees to parameterise the biomass loss of adult trees, as done in Sitch et al. (2007), will likely lead to an overestimation of plant damage and loss of carbon storage. Dose-response relationships based on biomass increments or growth rates might be better transferable between saplings and mature trees and hence better suitable to be used for parameterising global terrestrial biosphere models.

Our approach to overcome this challenge was to alter the vegetation model to simulate the ozone damage of small trees, where we could directly compare simulated biomass reductions to observations. Since we used ~~damage~~ injury relationships that are based on the calculation of leaf-level photosynthesis, we are able to apply the calibrated model also for mature stands. Our simulations have demonstrated that despite the different sizes of young and mature trees, and associated changes in the wood growth rate and the available amount of non-structural carbon reserves to repair incurred ~~damage~~injury, the simulated effect of ozone on the net annual biomass production (NPP) was very similar, when using a ~~damage~~ injury function associated with leaf-level photosynthesis. Overall our findings support the idea that the photosynthesis-based ~~damage~~ injury relationships developed here and evaluated against fumigation experiments of young trees, might be useful to estimate effect on forest production of older trees. Monitoring approaches of ozone damage that are either capable of measuring the actual increment of biomass, or quantify at the leaf and canopy level the change in net photosynthesis over the growing season, would allow to develop injury/damage estimates that could be more readily translated into modelling frameworks.

The extrapolation of results from short-term experiments with young trees to estimate responses of adult trees grown under natural conditions is subject to several issues, e.g. due to the differing environmental conditions and changing ozone sensitivities with increasing tree size or age (Schaub et al., 2005; Cailleret et al., 2018). If the simulation of injury to photosynthesis based on experiments with young trees can indeed be transferred to adult trees to yield realistic biomass damage estimates is still uncertain. The sparse knowledge of ozone effects on the biomass of adult forest trees prevents an evaluation of simulated ozone damage of adult trees. Ozone fumigation is mostly found to reduce e.g. biomass or diameter of adult trees (e.g. Matyssek et al. (2010) for an overview), but this is not always the case (Samuelson et al., 1996; Percy et al., 2007). Results from phytotron and free-air fumigation studies suggest that in natural forests a multitude of abiotic and biotic factors exist that have the potential to impact the plants ozone effects (Matyssek et al., 2010). If more data become available e.g. of the changes in ozone sensitivity between young and mature trees a more realistic damage parameterisation of mature forests in terrestrial biosphere models might become possible.

Terrestrial biosphere models in general assume that plant growth is primarily determined by carbon uptake. However, an alternative concept proposes that plant growth is more limited by direct environmental controls (temperature, water and nutrient availability) than by carbon uptake and photosynthesis (Fatichi et al., 2014). The O-CN model provides a first step into this direction because it separates the step of carbon acquisition from biomass production, both in terms of a non-structural carbon buffer, as well as a stoichiometric nutrient limitation on growth independent of the current photosynthetic rate. This would in principle allow to account for ozone effects on the carbon sink dynamics within plants. However, it is not clear that data readily exist to parameterise such effects. ~~Given the availability of suitable data to parameterise a large scale model, ozone damage~~ Instead of targeting net photosynthesis as done in our approach here, ozone injury might be better simulated by targeting biomass growth rates or processes that limit these e.g. stomatal conductance, which impacts the plants water balance~~compared to our approach here, which targets net photosynthesis~~, given that suitable data to parameterise a large scale model become available.

All in all, a multitude of aspects that impact ozone damage to plants is not yet incorporated into global terrestrial biosphere models. The ongoing discussion which processes are major drivers for observed ~~damages~~damage, how they interact and impact different species and plant types plus the lack of suitable data needed to parameterise a global model are reasons why the simulation of ozone damage up to now focuses only on a few aspects where suitable data are available as presented in our study.

5 Conclusion

The inclusion of previously published ~~dose-response relationships~~ injury functions into the terrestrial biosphere model O-CN led to a strong over- or underestimation of simulated biomass damage compared to the biomass dose-response relationship by Büker et al. (2015). ~~Dose-response relationships which are used as damage functions in~~ Injury functions included into terrestrial biosphere models are a key aspect in the simulation of ozone damage and have a great impact on the estimated damage. The calibration of ~~damage~~ injury functions performed in this study provide the advantage to calculate ozone ~~damage~~ injury close to where the actual physiological ~~damage~~ injury might occur (photosynthetic apparatus) and simultaneously reproduce observed biomass damage relationships for a range of European forest species used by Büker et al. (2015). The ~~inclusion of these damage functions into models that estimate regional or global ozone damage~~ calibration of ozone injury functions similar to our approach here in other ozone sub-models of terrestrial biosphere models might improve damage estimates compared to previously published ~~damage~~ injury functions and might lead to better estimates of terrestrial carbon sequestration. The comparison of simulated biomass dose-response relationships of young and mature trees shows strongly different slopes. This suggests that observed biomass damage relationships from young trees might not be suitable to estimate biomass damage of mature trees. The comparison of simulated NPP dose-response relationships of young and mature trees show similar relationships and suggests that they might more readily be transferred between trees differing in age.

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Table A.1. Original and adapted values of the nitrogen specific photosynthetic capacity of a leaf (npl) for three out of four different O-CN versions (ID) including published ~~damage~~ injury functions. The intercept of the fourth O-CN version ($L12_{VC}$) is very close to one and simulations produce comparable LAI values without an adaption of npl.

ID	PFT	npl original	npl adapted
$W07_{PS}$	Broadleaf	1.50	1.60
$W07_{PS}$	Needleleaf	0.75	0.80
$L12_{PS}$	Broadleaf	1.50	1.45
$L12_{PS}$	Needleleaf	0.75	0.70
$L13_{PS}$	Broadleaf	1.50	1.75
$L13_{PS}$	Needleleaf	0.75	0.90

Table A.2. List of fumigation experiments used by Büker et al. (2015) and simulated here.

Site	Longitude [°E]	Latitude [°N]	Species	O_3 treatment start year	Fumigation [yrs]
Östad (S)	12.4	57.9	*Betula pendula*	1997	2
Birmensdorf (CH)	8.45	47.36	*Betula pendula*	1989	1
Birmensdorf (CH)	8.45	47.36	*Betula pendula*	1990	1
Birmensdorf (CH)	8.45	47.36	*Betula pendula*	1992	1
Birmensdorf (CH)	8.45	47.36	*Betula pendula*	1993	1
Kuopio (FIN)	27.58	62.21	*Betula pendula*	1994	2
Kuopio (FIN)	27.58	62.21	*Betula pendula*	1996	3
Kuopio (FIN)	27.58	62.21	*Betula pendula*	1994	5
Schönenbuch (CH)	7.5	47.54	*Fagus sylvatica*	1991	2
Zugerberg (CH)	8.54	47.15	*Fagus sylvatica*	1987	2
Zugerberg (CH)	8.54	47.15	*Fagus sylvatica*	1989	3
Zugerberg (CH)	8.54	47.15	*Fagus sylvatica*	1991	2
Curno (I)	9.03	46.17	*Populus spec.*	2005	1
Grignon (F)	1.95	48.83	*Populus spec.*	2008	1
Ebro Delta (SP)	0.5	40.75	*Quercus ilex*	1998	3
Col-du-Donon (F)	7.08	48.48	*Quercus robur or petraea*	1999	2
Headley (U.K.)	-0.75	52.13	*Quercus robur or petraea*	1997	2
Ebro Delta (SP)	0.5	40.75	*Pinus halepensis*	1993	4
Col-du-Donon (F)	7.08	48.48	*Pinus halepensis*	1997	2
Schönenbuch (CH)	7.5	47.54	*Picea abies*	1991	2
Zugerberg (CH)	8.54	47.15	*Picea abies*	1991	2
Östad (S)	12.4	57.9	*Picea abies*	1992	5
Headley (U.K.)	-0.75	52.13	*Pinus sylvestris*	1995	2

Table A.3. Slopes and intercepts of biomass dose-response relationships for broadleaved species simulated by O-CN versions based on published ~~damage~~ injury functions to net photosynthesis or V_{cmax} (see Tab. 1). B_{SI} and B_{ST} represent the simple and standard model of Büker et al. (2015).

ID	Intercept (a)	Slope (b)	R^2	p-value
B_{SI}	0.99	0.0082	0.34	<0.001
B_{ST}	0.99	0.0098	0.38	<0.001
$W07_{PS}$	1	0.00045	0.93	1e-24
$L12_{PS}$	1	0.0142	0.77	2e-14
$L15_{PS}$	1	0.0000	NaN	NaN
$L12_{VC}$	1	0.0120	0.80	1.9e-15

Table A.4. Slopes and intercepts of biomass dose-response relationships for needle-leaved species simulated by O-CN versions based on published ~~damage~~ injury functions to net photosynthesis or V_{cmax} (see Tab. 1). B_{SI} and B_{ST} represent the simple and standard model by Büker et al. (2015).

ID	Intercept (a)	Slope (b)	R^2	p-value
B_{SI}	1	0.0038	0.46	<0.001
B_{ST}	1	0.0042	0.52	<0.001
$W07_{PS}$	1	0.00058	0.93	1.5e-09
$L12_{PS}$	1	0.0119	0.83	9.4e-07
$L15_{PS}$	1	0.0000	NaN	NaN
$L12_{VC}$	1	0.0096	0.85	3.5e-07

Table A.5. Slopes and intercepts of biomass dose-response relationships for broadleaved species simulated by O-CN versions based on tuned ~~damage~~ injury functions to net photosynthesis or V_{cmax} (see Tab. 1). B_{SI} and B_{ST} represent the simple and standard model by Büker et al. (2015).

ID	Intercept (a)	Slope (b)	R^2	p-value
B_{SI}	0.99	0.0082	0.34	<0.001
B_{ST}	0.99	0.0098	0.38	<0.001
tun_{PS}	1	0.0093	0.94	1.4e-26
tun_{VC}	1	0.0091	0.93	5e-25

Table A.6. Slopes and intercepts of biomass dose-response relationships for needle-leaved species simulated by O-CN versions based on tuned ~~damage~~ injury functions to net photosynthesis or V_{cmax} (see Tab. 1). B_{SI} and B_{ST} represent the simple and standard model by Büker et al. (2015).

ID	Intercept (a)	Slope (b)	R^2	p-value
B_{SI}	1	0.0038	0.46	<0.001
B_{ST}	1	0.0042	0.52	<0.001
tun_{PS}	1	0.0039	0.94	4.8e-10
tun_{VC}	1	0.0042	0.93	2.2e-09

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