

Interactive comment on "Nitrogen inputs and losses in response to chronic CO₂ exposure in a sub-tropical oak woodland" by B. A. Hungate et al.

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We appreciate the reviewer's comments and here provide our responses, along with a description of how the comments inspired changes to the manuscript.

1) I like that the authors attempt to control for potential fractionation during fixation, by incorporating the 15N signal of a non-fixer. However, it is not clear to me how the authors arrived at the equation on page 67. Perhaps helpful to the reader would be either a reference or a bit more detail on how the authors arrived at the equation. It is otherwise hard to judge whether the assumptions made are correct.

Authors' Response: We appreciate the suggestion, as we reviewed the equation and C1337

found a typesetting error. The correct equation is:

 $pf = (\delta 15NO - \delta 15NG)/(\delta 15NO - \delta 15N Go),$

where δ 15NO is the 15N signature of the oaks, δ 15NG is the 15N signature of the N2 fixer from the experimental plot, and δ 15N Go is the 15N signature of the N2 fixer prior to tracer 15N application.

We also now cite the source of this mixing model, Shearer and Kohl (1986). Their formulation used the δ 15N signature of the nodulating plant grown in an N-free medium, thus capturing the δ 15N signature associated with 100% fixation (and any fractionation). Based on our argument that G. elliottii obtains nearly all its N from fixation, using the signature of plants prior to label application is a reasonable proxy. In any case, the difference is small, because the observed value (-2.2‰ is close to 0‰ the atmospheric signature.

Shearer, G., and Kohl, D.H. N2 fixation in field settings: Estimates based on natural 15N abundance. Aust. J. Plant Physiol. 13,699-756, 1986.

2) I would like to know from the authors why they decided on analyzing leaf litter for herbivory instead of live leaves. Is there a potential that the herbivory damage was done after litterfall?

Authors' Response: By analyzing herbivory on leaves captured in litter traps, we assess the effects of herbivory over the entire lifetime of a leaf. Assays of live leaves may underestimate herbivory for this reason. The reviewer is correct that, after leaves senesce, detritivores continue to consume them. However, our sampling collected leaves soon after they senesced and fell from the plant, minimizing the time that detritivores could have eaten them. Furthermore, detritivores are distinct organisms from the herbivores that consume live leaves, and the approaches we used to estimate herbivory assessed the nature of the damage to the leaf tissue, allowing it to be attributed to herbivores. 3) 15N tracer recovery. Is there a possibility that N leaching and movement into the depth in the CO2 enriched site might have been caused from increased water use efficiency? Less transpiration would increase percolation and the potential of tracers to move with water?

Authors' Response: We agree, this is another possible explanation, and we have added text to acknowledge this, so that the revised explanation reads:

"Increased leaching with elevated CO2 has been observed, and may be caused by a combination of increased water use efficiency resulting in greater downward water flux through the soil profile (Jackson et al., 1998), along with increased turnover of soil organic matter in response to rising CO2."

4) I would like to suggest that the authors mention in the discussion the major findings in their earlier paper when they talk about ecosystem N accumulation. From my reading of Hungate et al., 2013, N accumulated in the above ground pools at the cost of the belowground pools. This helps to better understand the overall ecosystem response. It seems that the live vegetation is mining for nitrogen (and other nutrients), yet the 15N tracer experiments and the N fixation data suggests otherwise. A nuanced discussion of this will be a benefit for modeling community.

Authors' Response: Excellent suggestion, and we have tried to synthesize the diverse lines of evidence from this experiment that lend insight into CO2 effects on the N cycle. We have added the following paragraph to the "Summary" section of the paper, and modified the final paragraph slightly, to address this point.

Evidence from this experiment suggests that eleven years of chronic exposure to increased CO2 concentrations elicited disequilibrium in the N cycle, with increased rates of internal N transformations, no change in N inputs, and increased N losses. Specifically, elevated CO2 accelerated rates of internal N transformations, including soil N mineralization (Langley et al. 2009; McKinley et al. 2009), which likely contributed to increased N uptake by plants (Hungate et al. 2013). Nitrogen losses also increased,

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at the scale of increased turnover of N through plant tissues, consistent with the increased 15N dilution in plant tissues reported previously, along with no change in net plant N capital (Hungate et al. 2013). Elevated CO2 also appeared to enhance N losses at the scale of the soil profile: the pattern of lower 15N recovery in plots exhibiting greater downward movement of 15N in the soil profile suggests increased leaching losses. Thus, processes that make nutrients available to plants can also promote nutrient losses. Finally, we found no evidence that elevated CO2 enhanced N inputs via N2 fixation. Together, these results paint a picture where more rapid cycling of N with elevated CO2 is unlikely to be sustained.

These findings provide an empirical point of contrast with model projections in which elevated CO2 enhances N2 fixation and reduces leaching (Thornley and Cannell, 2000). This is an important conclusion for modeling responses of future C uptake by the biosphere, given the strong influence of N cycling and N accumulation on the biosphere's capacity to assimilate and store C (Thornton et al., 2007; Churkina et al., 2009; Arneth et al., 2010; Zaehle and Dalmonech, 2011).

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