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Biogeosciences Discussions

Interactive comment on "Plant physiological and environmental controls over the exchange of acetaldehyde between forest canopies and the atmosphere" *by* K. Jardine et al.

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General Comments:

Understanding the nature of the physiological controls regulating oxygenated VOC production, release, and consumption remains a major challenge to the field of plant phys/biochemistry. Unraveling the controls over oxygenated VOC flux is particularly difficult as we begin to appreciate the complicated internal leaf biochemical cycles which can alter production/consumption/emission. The data presented by Jardine et al., is an important contribution to this task as it continues to inform our understanding of bi-directional exchange of acetaldehyde in tree leaves. In particular, the application

of IR-GCMS to examine the potential physiological controls influencing acetaldehyde fractionation is particularly novel, especially as it (tentatively) suggests a prominent role for stomatal conductance in regulating the bi-directional exchange of acetaldehyde. Over the past few years, this field has moved from a "flooding-only" based view of acetaldehyde emission to one where researchers now regularly observe acetaldehyde emission in non-flooded environments as well. This broadening of the potential for acetaldehyde sources, futher argues for the importance of mechanistic studies such as the one presented here.

The authors present a fairly compelling case for the stomatal regulation of leaf acetaldehyde emission. The use of the PTR-MS to estimate transpiration is creative, but as a non PTR-MS expert it would be useful if the authors could show (or cite) some data confirming that this MS based approach to estimating transpiration does, in fact, scale with more traditional (IRGA/porometer) techniques, especially since this is a key aspect of their study. Likewise, the light/dark comparisons clearly suggest an important role for stomatal regulation, if we can trust the PTR-MS based transpiration estimates. Unfortunately, a very important component of the data linking stomatal resistance with exchange is the single dark Quercus ilex measurement. It's frustrating that a key aspect of this study (ie dark =high stomatal resistance=reduced exchange) is based on an n=1 (unlike the n=5 Q. ilex light treatments and the n=6 P. deltoides dark measurements). I agree with the authors that this data does suggest that stomata are involved in regulating exchange, but I get nervous when (as a field) we are OK with n=1 approaches. Fortunately, the complementary Rottenberger et al. 2008 study does help alleviate this concern somewhat.

I found the kinetic isotope effect compelling, and perhaps the strongest evidence for selective acetaldehyde uptake mediated by stomata. However, with the data presented I'm wondering why the authors believe stomatal resistance alone is sufficient to account for the 5ppm differences observed in the intact poplar branches, especially considering the wide-open nature of poplar stomata. The authors should expand their discussion

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of this point, particularly with regards to some of the current thinking about the extent to which stomatal resistances do/do not contribute (and how much) to fractionation of other gaseous compounds (particularly CO2).

I realize the authors would like to conclude there is no deposition to leaf (branch) surfaces in their experiments (and likely this is very small), but the argument given that 15h of continuous fumigation should saturate surface binding is really speculation at best, especially since the total leaf /branch surface area was not presented nor do we really know what the capacity for deposition might be. This could be particularly problematic if leaf microorganisms (bacteria/fungi/etc?) are present (which they would be) that might also uptake and utilize acetaldehyde over these time scales. Ideally, in this study the authors would have incorporated an ABA-feeding experiment. Hormonally regulating stomatal aperature (closing) while simultaneously maintaining leaf cell wall/epidermis hydration status would be the real test for of the surface deposition hypothesis and also would nicely clarify the fractionation results. I hope someone in the future integrates ABA based experiments with GC-IRMS approaches.

Finally, the authors seem to want to drive/relate acetaldehyde exchange dynamics to solar radiation (i.e. discussion of sun shade leaves, canopy density, self shading, etc) suggesting in numerous places that emission is a function of light. Of course biochemically there may be some relationship to light (light enhanced rates of mitochondrial respiration?) , but in the data presented the authors can't really separate light-enhanced emission from light regulated control over stomatal aperature. This, of course, is very different from light-dependent VOCs (such as isoprene) where the biosynthesis can be directly related to PAR and electron transport. The enhanced emission from sun leaves in the canopy may simply reflect lowered stomatal resistance in concert with higher transpiration rates. Although one could relate Gs to PAR in some systems and under some conditions, in general I don't think the statement "acetaldehyde compensation point is a function of light" is really what the authors intend to suggest. A subtle point, but critical if we are all to agree on the physiological parameters that ultimately

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regulate acetaldehyde emission and the environmental drivers that matter. Interesting work.

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