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## ***Interactive comment on “Impact of heat stress on the emissions of monoterpenes, sesquiterpenes, phenolic BVOC and green leaf volatiles from several tree species” by E. Kleist et al.***

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This submission examines the impact of relatively severe heat stress on BVOC emissions from four European tree species. Although the severity of the heat stress applied in different cases was variable and unquantifiable (a bewildering combination of varying maximum temperatures and duration of stress) a number of interesting generalizations can be drawn. There is clearly a distinction between *de novo* emissions, those derived from recently fixed carbon and therefore light-dependent, and emissions from storage pools. Emissions from pools in the two coniferous species examined increase exponentially with increasing temperature as expected but when a certain

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threshold is reached, emissions increase explosively, accompanied by the appearance of green leaf volatiles, indicative of membrane damage. The authors suggest, quite plausibly, that the burst in emissions is related to structural damage to resin canals and a rapid decrease in diffusion resistance out of those storage pools. In contrast, *de novo* emissions, reliant on plant metabolism, decrease rapidly above a certain (temperature X duration) threshold, accompanied by a rapid decrease in the amount of  $^{13}\text{C}$ -labeling. The behavior of *de novo* emissions seems to be similar, regardless of whether the emissions are constitutive (beech, Palestine oak) or induced by herbivore stress (aphid-infested Scots pine and Norway spruce).

“Heat stress” as used here is an ill-defined concept. I think most physiologists would agree that primary metabolism may be severely restricted, i.e., heat-stressed, well before significant effects on BVOC are apparent. “Heat stress” as used here represents some combination of high temperature and duration of exposure, but is really characterized by irreversible changes in BVOC and the induction of GLV, related to membrane damage. The emphasis on effects of ‘heat-stress’ as here defined tends to ignore the effects of increasing temperature on BVOC emissions prior to the onset of irreversible damage.

The authors attempt to interpret their results in the context of future climate change and the expected increase in the frequency, magnitude and duration of high temperature events. To their credit, they acknowledge that their data is inadequate to predict specific consequences of increasing frequency of heat stress events, only generalizing that the impact on BVOC emissions will depend on the mix of *de novo* and pool emissions in a given region, which may lead to either an increase or decrease of total emissions. They fail to discuss the effect of generally increasing temperatures, below the hypothetical tipping point at which irreversible damage occurs, which, based on our current understanding, will clearly lead to large increases in BVOC emissions. Compared to these effects, the impact of these irreversible heat stress events is likely to be minor. Particularly since at least some of the stresses imposed in this study (e.g., 45°C

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continuously for 48 h or 51°C for 4 h) are unlikely to occur.

The authors are, in my view, too quick to attribute heat stress induced declines in *de novo* emissions to enzyme denaturation. Although our understanding of the limitations to isoprene emissions at high temperatures remains controversial, there is good evidence that substrate levels can play a significant role. Although very little data is presented in this study related to net photosynthesis and stomatal conductance, it is quite likely that general physiology, electron transport in particular, is severely depressed during these stress events, and DMADP levels may well limit *de novo* BVOC synthesis.

In general, the authors present convincing evidence that heat-stress, characterized by irreversible changes in BVOC emissions and the production of GLV, affects *de novo* emissions and emissions from storage pools in fundamentally different ways. This should be the thrust of the paper and certainly justifies publication. With that in mind, I think the paper could be shortened somewhat, eliminating speculation about the effects of future climate change on BVOC emissions. The data presented in this paper provides no way to address these questions except in a very general way. An assessment of future impacts will require a much more rigorous attempt to quantify the interacting effects of maximum temperatures, duration of exposure, water stress, etc..

The authors stress the possible importance of enzyme denaturation in explaining the observed declines in *de novo* emissions, but present no evidence for this assumption, which appears unwarranted to me. Any discussion of hypothetical causes should include the potential for substrate (DMADP) limitations. A more thorough discussion of the changes in primary metabolism (photosynthesis and transpiration) before and after the imposition of stress would be helpful in this regard.

Detailed editorial suggestions:

Page 9534

I. 3 need to define 'heat stress'; in this context, it is irreversible changes in BVOC

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emissions, associated with release of GLV; one might argue that ‘heat stress’ defined as deleterious effects on primary metabolism occur under far less stressful conditions

l. 7 ‘were’ instead of ‘being’

l. 10 suggest ‘release of monoterpenes from pools. . .’

l. 11 delete ‘of’

l. 20 It is important to distinguish between high temperatures (not defined as ‘heat stress’) and ‘heat stress’ itself. Thus, ‘heat waves’ will almost certainly increase BVOC emissions; only when conditions exceed some quite high threshold is there the potential for decreases or additional increases related to damage to resin canals.

Page 9536

l. 5 suggest ‘. . . in the study, although they may be expected to have a large impact on future BVOC emissions.’

p. 9538

l. 25 ‘. . . distance of the respective leaf from the chamber lamps. . .’

p. 9540

l. 9 ‘Consistent with these observations, MT emissions . . .’

l. 15 I’m not sure I’d call 10-30 percent labeling ‘low’

l. 19 Ghirardo misspelled

p. 9541

l. 4 ‘Consistent with the results of . . .’

l. 7 I believe this should be ‘A three-year-old beech seedling’ but I leave it to the copy editors.

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I. 15 When I look at Fig. 1, it appears to me that the data at 31°C, 40°C and 24°C (after heating) all fall on an exponential temperature curve with  $\beta=0.09$  or so. I.e., no evidence of irreversible changes until returning to 31°C on day 8. One might suspect that the reduced rates at 31°C on day 8 are the result of the cooler temperatures on days 6 and 7 (as has been shown for isoprene emissions).

p. 9542

I. 6 For the beech experiment, you report that net photosynthesis and transpiration were unaffected by the imposed stress. Was this also true in the case of oak?

I. 11 'constitutive *de novo* emissions were decreased. . .'

p. 9543

I. 4 'In contrast' instead of 'contrary'

I. 9 resin ducts

p. 9544

I. 7 Again, knowing whether photosynthesis and transpiration recovered from the (quite severe) 51°C heat stress would be useful. Was the plant even alive following several hours at 51°C?

I. 19 delete 'on'

p. 9545

I. 2 These emissions from apparently damaged resin canals must represent a large fraction of the total monoterpene pools within the needles. Can you estimate what fraction of the pools is lost as a result of the stress?

I. 21 suggest '. . . confirming that they are *de novo* emissions.'

I. 23 '. . . stored carbon, consistent with measured emissions in darkness.'

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p. 9546

l. 10 ‘ . . . about 2-fold higher. . . ’

l. 12 Again, it would be nice to know whether or not net photosynthesis and transpiration recovered if that information is available.

p. 9547

l. 9 It's not surprising that no clear relationship between maximum applied temperature and stress impact emerges, since the duration of exposure also changed widely between treatments. Although we might expect greater stress from a 45°C exposure than a 40°C exposure, if the first is for one hour and the second for 6 hours, the effect might well be reversed. Also, the effect of elevated temperatures at night, something plants are unlikely to be exposed to in nature, confuses interpretation of the results.

l. 18 I think 51°C may be unrealistically high, although not inconceivable under water stress.

p. 9548

l. 3 ‘ . . . release shortly after their biosynthesis. . . ’

l. 12 suggest ‘ . . . during irreversible heat stress.’ In fact, GLV emissions are basically the criteria by which you define ‘heat stress’.

l. 24 suggest ‘ . . . *de novo* MT emissions can drop. . . ’

l. 25 The authors acknowledge that reductions in isoprene emission above the temperature optimum result from ‘an overall reduction of biosynthetic activity’ which includes both reductions in available substrate (i.e., DMADP) and isoprene synthase activity (whether by regulation or denaturation). In the next paragraph however, they seem to suggest that reductions in *de novo* MT emissions result from denaturation alone. I don't think this is supported by any evidence.

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p. 9550

I. 8 Were net photosynthesis and electron transport reversible or was general metabolic activity irreversibly affected by the heat stress and presumed membrane disruption?

p. 9551

I. 19 resin ducts

I. 21 I don't understand this sentence. Any  $^{13}\text{C}$  labeled emissions of MT prior to stress were presumably *de novo* emissions, and would be eliminated by the heat stress (either by denaturation or in my view more likely by substrate limitations). The labeling expt tells us nothing about emissions from pools.

I. 24 This entire paragraph seems difficult to understand and unnecessary. The conclusion (p. 9552, line 7) seems straightforward and obvious. As shown in Fig. 3, the labeled emissions fall to near zero after the imposition of stress, while the total emissions, presumably from unlabeled pools, increase to extraordinary levels, in parallel with increased GLV emissions.

p. 9552

I. 11 this comparison ignores the time of exposure; Tingey presumably exposed needles to 46°C for just long enough to make an emission measurement, whereas in this study, needles were kept at high temperatures for hours to days.

I. 29 resin ducts

p. 9554

I. 1 widespread

I. 5 'to aphid infestation'

I. 17 delete 'respective'

p. 9555

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I. 1 ' . . . emissions to heat stress. . . '

I. 4 'decreases of constitutive *de novo* MT emissions;'

p. 9556

I. 15 Again, I think the emphasis on enzyme denaturation is too strong. While denaturation is certainly possible (at 51°C in particular) denaturation at 31°C or 35°C seems unlikely. I think a greater emphasis should be placed on potential substrate limitations, which should be related to declines (and recovery) of primary metabolic activity, in particular electron transport capacity.

p. 9557

I. 1 delete 'from'

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