

Herbivory significantly increases biogenic methanol emissions in big tooth aspen *Populus grandidentata*, but no more so than mechanical wounding

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INTRODUCTION

Ecological relationships of plants and herbivores have implications for biosphere-atmosphere interactions because herbivory can increase emissions and change air quality¹. Studies on biogenic methanol (MeOH) emission response to herbivory have observed significant emissions directly following herbivore attack and even larger emissions 24hrs later^{2,3}. These studies attributed sustained emissions to re-growth of tissue and altered leaf chemistry due to insect oral secretions. To help constrain atmospheric budgets of MeOH, we investigated the effects of *Lymantria dispar* (gypsy moth) herbivory and mechanical damage on MeOH emissions in *Populus grandidentata* (big tooth aspen).

We hypothesize 1) short-term increases in MeOH emissions to result from decreased diffusive barriers at wound sites, 2) *L. dispar* oral secretions to enhance long term (24hr) MeOH emission responses to damage, and 3) long term elevated emissions resulting from cellular repair mechanisms occurring post-damage.

We first investigated *P. grandidentata* emission response to wounding over short (10min) and long (24hr) time scales under greenhouse conditions. Then we compared methanol emission response to herbivory and mechanical wounding under field conditions.

MATERIALS AND METHODS

P. grandidentata trees were grown in the greenhouse at SUNY at Stony Brook. Field studies were conducted at the University of Michigan's Biological Station (UMBS). *P. grandidentata* individuals were measured in the field during the summer of 2007.

We used a Teflon enclosure system for both studies to trap MeOH emitted from leaves. Samples were analyzed with a GC-FID system. We sampled each branch before, 10min post treatment, and 24hr post treatment. There were two treatments for the field study: 1) mechanical damage and 2) herbivory by *L. dispar*.

RESULTS

MeOH flux was higher following wounding and herbivory (Figures 1 & 2). Greenhouse plants did not emit significantly more MeOH 24hrs post-wounding, although there was a positive trend. This trend was stronger in field studies where both mechanical and herbivore damage elicited elevated MeOH emissions 24hr post-treatment (Figure 2). Field plant emissions 10min post treatment did not differ from those 24hr post treatment (Figure 2), unlike greenhouse plants that emitted less MeOH after 24hrs (Figure 1). Treatments for mechanical and herbivore damage did not elicit different emission responses (Figure 2).



CONCLUSIONS AND FUTURE DIRECTIONS

We have observed *P. grandidentata* to increase MeOH emissions post-wounding and post-herbivory on time scales of minutes and hours. These experiments are congruent with hypothesis 1 but not with hypothesis 2. Instead, our results suggest physical damage to be the dominant driver for elevated emissions. Although emissions do not differ quantitatively between 10min and 24hr post treatment, the biological processes responsible for emissions at these two time periods may or may not differ.

In order to address hypothesis 3, we plan to investigate the physiological mechanisms driving long term MeOH emission responses. These experiments will involve investigating cellular repair in damaged leaves as well as alternative MeOH sources, such as up-regulated cell division in roots.



ATMOSPHERIC IMPLICATIONS

Our results indicate that tissue damage, whatever its cause, can double or triple MeOH emissions from leaves. Atmospheric modelers may constrain estimates of plant-derived MeOH sources by incorporating a tissue damage parameter.

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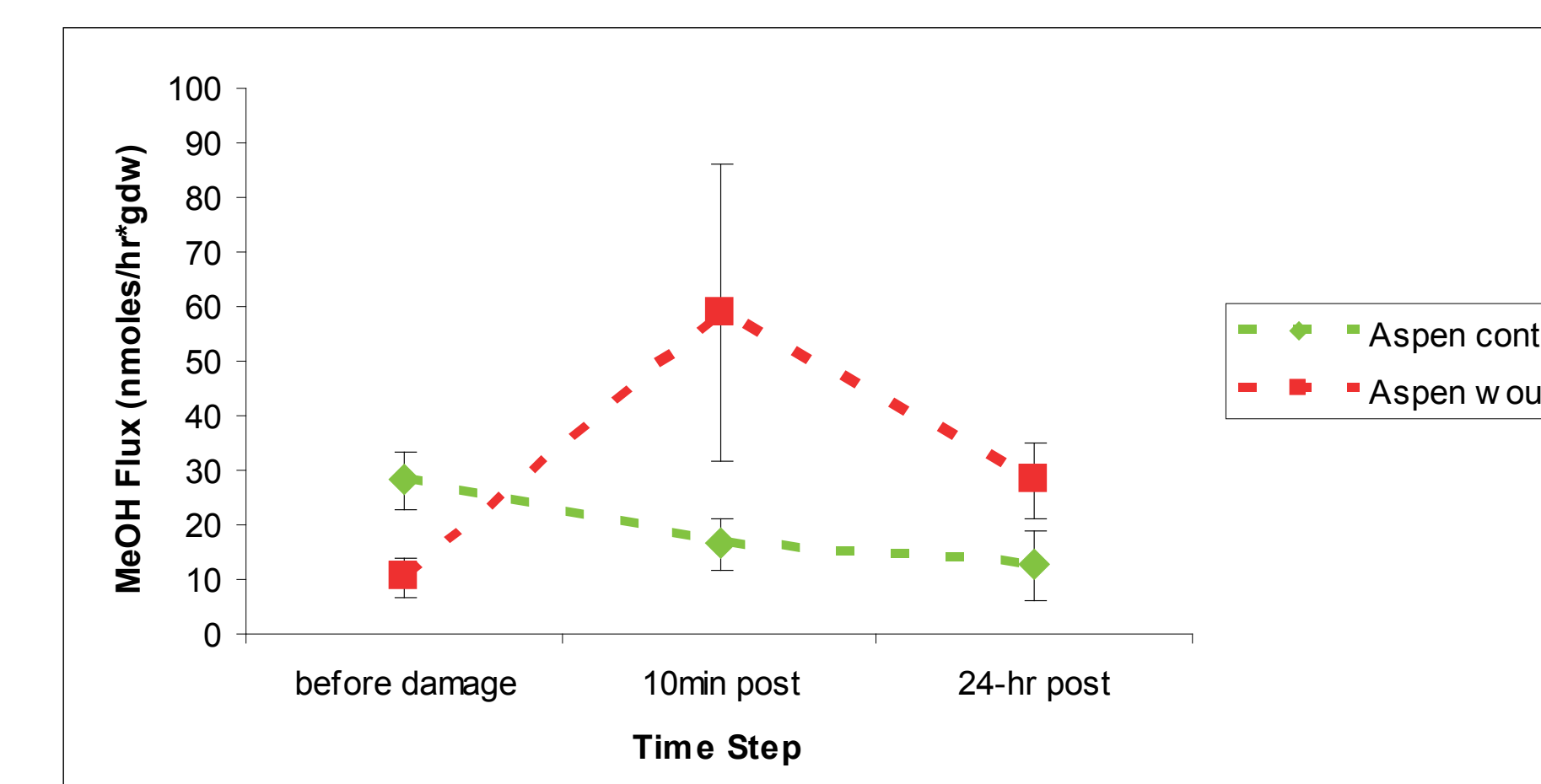


Figure 1. MeOH fluxes from control (shown in green) and mechanically wounded (shown in red) *P. grandidentata* leaves. Data collected from greenhouse population. MeOH flux 10min post treatment was significantly higher than control fluxes (Mann-Whitney $P < 0.015$ $n=6$).

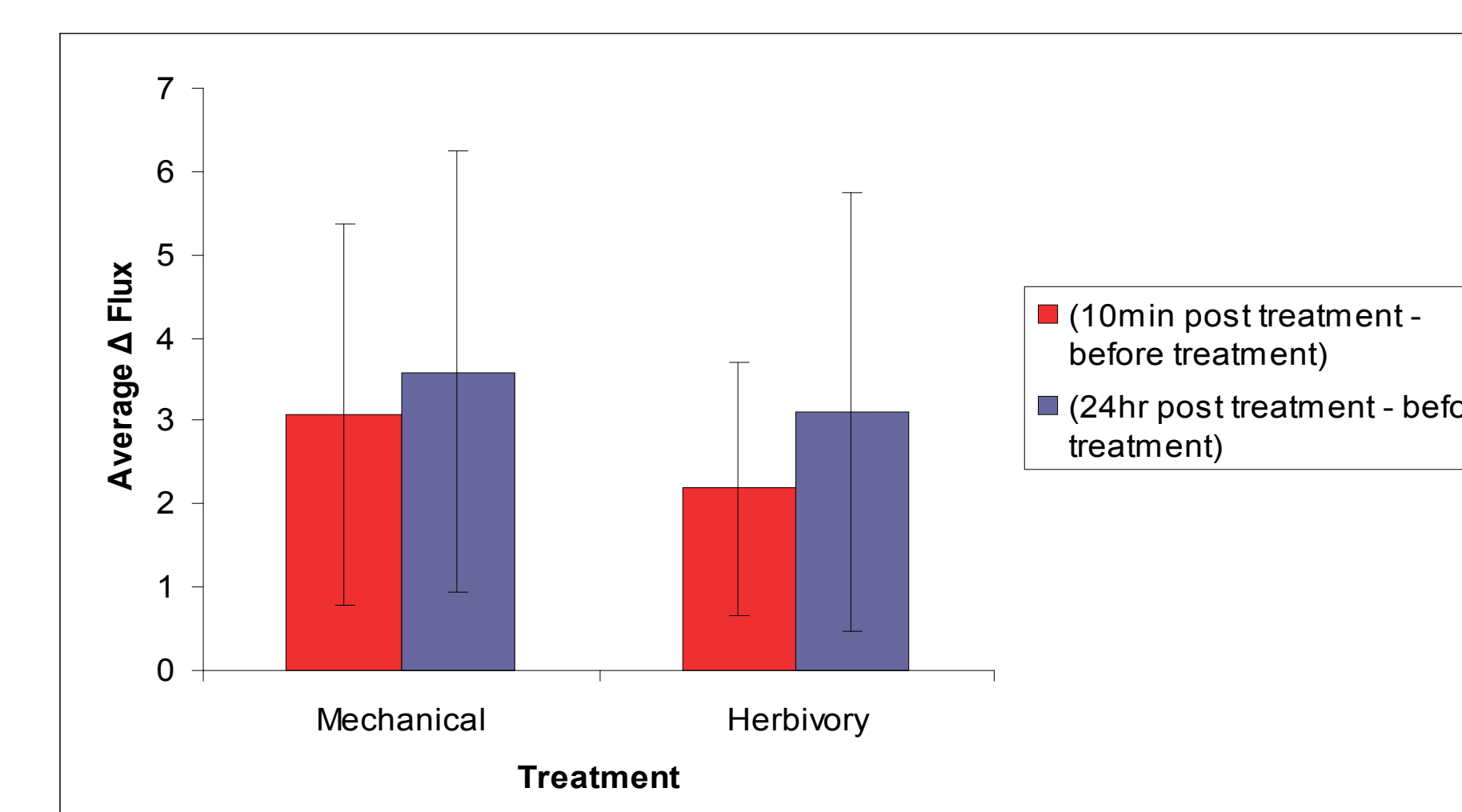


Figure 2. Average Δ flux values for mechanical and herbivore damaged *P. grandidentata* leaves. Change in flux 10min post treatment (bars in red) and change in flux 24hr post treatment (bars in blue) were positive for both treatments. Fluxes were not significantly different at either time period (Wilcoxon test: $p=0.14$ (after treatment-before) $p=0.42$ (24hr post-before)).

