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Stress induced emission of biogenic VOC: Induced sesquiterpene emissions from ozone exposed tobacco

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Stress conditions to plants may strongly increase their VOC emissions. To study the kinetics of these stress induced VOC emissions in more detail we used short pulses ($\approx$ 1 h) of ozone (O3) exposure at high concentrations as elicitor. The O3-sensitive tobacco variety Bel W3 was used as model plant and 2 different on-line-GC-MS systems as well as a fast PTR-MS were applied as analytic instruments. We observed increased emissions of methanol, leaf alcohols and aldehydes produced within the octadecanoid pathway (LOX products), methyl salicylate (MeSa), and sesquiterpenes (SQT).

Increased VOC emissions did not start directly after onset of the ozone exposures but with lag times. These lag times between start of ozone exposure and increases of VOC emissions varied between 30 minutes and up to 16 hours, depending on the ozone flux and VOC species. Methanol emissions increased first, followed by those of MeSa and LOX products. Sesquiterpene (SQT) emissions increased in some cases before, and, in other cases, simultaneously to those of LOX products and MeSa.

Details regarding the emissions of LOX products are shown elsewhere. Here we focus on increased SQT emissions. The data taken with the PTR-MS at a time resolution of 4 minutes often showed two peaking SQT emissions. The data taken with the GC-MS showed that, after O3 exposure, the emission pattern of sesquiterpenes was not constant, with significant changes during the emission pulses being observed here.

Both findings indicated at least two different plant internal signals leading to increased SQT emissions.

Well known as a trigger for increased emissions of isoprenoids is jasmonic acid (JA), a product synthesized within the octadecanoid pathway. However, we believe that JA is not the trigger for increased SQT emissions from O3 exposed tobacco due to the following reasons:

Exposing tobacco to methyl jasmonate (MeJa) led to increased SQT emissions but the pattern observed for MeJa exposure significantly differed from that observed after O3 exposure.

Suppressing the plant internal formation of JA by exposing plants to an oxygen free atmosphere led to increased SQT emissions while emission of LOX products decreased with the removal of oxygen. This behaviour furthermore indicates that the signal inducing increased SQT emissions from tobacco is not originating from processes downstream of lipoxygenase activity.

A similar behaviour was found for tobacco exposed to methyl salicylate. Exposure to methyl salicylate also led to increased SQT emissions, but again, the pattern was different from that of ozone exposed plants. Moreover, some SQT that were predominantly emitted after O3 exposure were not found even in traces after MeSa or MeJa application.

First experiments indicated that ethene might be involved in the induction of SQT emission after ozone fumigation. Exposing tobacco Bel W3 to ethene at high concentrations led to increased SQT emissions with a pattern very similar to that during the first peaking SQT emissions after O3 exposure.