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Stress induced VOC emissions from mildew infested oak.

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Oak plants *(Quercus robur)* not suffering from stress emit only isoprene. Mildew infestation leads to additional emissions of other volatile organic compounds (VOC) such as monoterpenes, sesquiterpenes and VOCs originating from the octadecanoid- and the shikimate pathway. While emissions of isoprene were mainly determined by temperature and light intensity those of other VOC were affected by additional processes.

Exposing healthy oak plants to methyl jasmonate led to similar mono- and sesquiterpene emissions as those induced by mildew infestation. This implies a role of jasmonic acid as one of the triggers inducing these emissions as a response to mildew attack. Furthermore, a good relation between the emission strengths of the isoprenoids and LOX products was found indicating a quantitative connection between the emission strengths of VOCs originating from different biosynthetic pathways.

Exposing the plants to $^{13}CO_2$ led to a quick labelling of mono- and sesquiterpenes showing *de novo* synthesis of these compounds as a response to mildew infestation. Labelling was also found for methyl salicylate but not for benzoic acid ethyl ester. From this we conclude that benzoic acid is not the predominant precursor of salicylic acid in *Quercus robur*. Labelling of VOCs from the octadecanoid pathway was much lower but significant. This labelling could not be explained by the amount of $^{13}CO_2$ taken up during exposure implying that mildew infestation leads to preferential flux of the carbon taken up as CO_2 to those parts of the plants where mildew infestation

leads to a destruction of cell membranes.