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Disease control by sulphur induced resistance

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Summary

As early as the 19th century, *Justus von Liebig* (1803 – 1873) identified the lack of vitality of soils and non-existent vigour of plants as relevant causes of increased infections of crops by fungal diseases. Organic farming requires alternative strategies for combating pests and diseases. Soil-applied sulphate fertilisation proved to significantly reduce infection rate and severity of crops by fungal diseases. The potential efficacy of so-called Sulphur Induced Resistance (SIR) expressed as a reduction of the disease index ranged from 5–50% and 17–35% in greenhouse and field experiments, respectively. Metabolic pathways involved in SIR imply, for instance, the synthesis of phytoalexins, glutathione, glucosinolates and the release of sulphur-containing volatiles.

Key words: Elemental sulphur, pathogen, sulphate, sulphur induced resistance

Introduction

In 2005, 8,176 t of fungicides were consumed in conventional agriculture in Germany (Anon., 2006). In contrast, no synthetic pesticides are used in organic farming. Infections of agricultural crops with fungal pathogens are, however, a serious problem in organic farming. They result in yield losses and may diminish the quality of harvest products when threshold values for pathogen-specific disease indices are exceeded. Common methods for disease control include soil tillage measures, crop rotation, mixed cropping systems and cultivation of resistant varieties. The targeted use of minerals offers yet another possibility to enhance resistance against pathogens. Here, the direct toxicity of nutrients (elemental S, Cu) and indirect impairment by minerals (Si) needs to be distinguished from nutrient-mediated, resistance mechanisms, which have been observed for all essential macro and micronutrients, Si and Al (Datnoff *et al*., 2006).

The fungicidal effect of foliar-applied elemental S has been exploited since the end of the nineteenth century. Elemental S has been used efficiently against infections of grapes by powdery mildew (*Uncinula necator*) ever since. An experiment of Bourbos *et al*. (2000) indicated that in addition to the direct fungicidal effect of foliar-applied S, there was a nutrient-based effect of soil-applied S that resulted in a lower rate of leaf and grape infection and produced a significantly higher yield compared with controls.

Schnug (1997) predicted that the decline in atmospheric S deposition and thus S nutritional status of agricultural crops since the mid 1980s might have serious consequences for the stability of current agro-ecosystems and in particular highlighted the increasing susceptibility of plants to

fungal pathogens such as light leaf spot (*Pyrenopeziza brassicae*). Sulphur fertilisation reduced the disease index for various host/pathogen relationships by 5–50% and 17–35% in greenhouse and field experiments, respectively. The term Sulphur Induced Resistance (SIR) denotes the reinforcement of the natural resistance of plants to fungal pathogens by stimulating the metabolic processes involving sulphur through targeted sulphate-based and soil-applied fertiliser strategies (Haneklaus *et al*., 2006). It is the aim of this contribution to summarise current research in SIR from the molecular to the field level in relation to different host/pathogen systems, and to identify future research needs.

Materials and Methods

Extended field experiments were carried out from 2000 to 2003 on two sites in Scotland, Inverness and Aberdeen, and one site in northern Germany, Braunschweig, in order to investigate mechanisms of SIR in oilseed rape in relation to the risk of infections with fungal diseases. Details concerning the layout of these studies and analytical methods involved are comprehensively summarised by Salac (2005).

Results

Sulphur Induced Resistance was first observed for infections of oilseed rape by *P. brassicae* (Schnug, 1997). The potential efficacy of SIR expressed as a reduction of the disease index ranged from 5–50% and 17–35% in greenhouse and field experiments, respectively. Possible S-containing metabolites involved in SIR are shown in Fig. 1. Plants take up sulphate (SO_4) through their roots. The S nutritional status regulates uptake, translocation and intracellular distribution of sulphate via sulphate transporters (Hawkesford, 2000). The amino acids cysteine and methionine are the major end-products of sulphate assimilation in plants. Cysteine is the basic compound for other S metabolites such as glutathione (GSH), glucosinolates (GSL), phytoalexins, pathogenesis related (PR) proteins and H_2S. All of these compounds are linked to resistance mechanisms of plants. GSH regulates uptake and assimilation of S and reduced S is transported in this form throughout the plant (Lappartient & Touraine 1996). Other metabolites presumably involved in the regulation of sulphate uptake are sulphate, *O*-acetylserine and cysteine (Hawkesford, 2000; Hawkesford & De Kok, 2006).

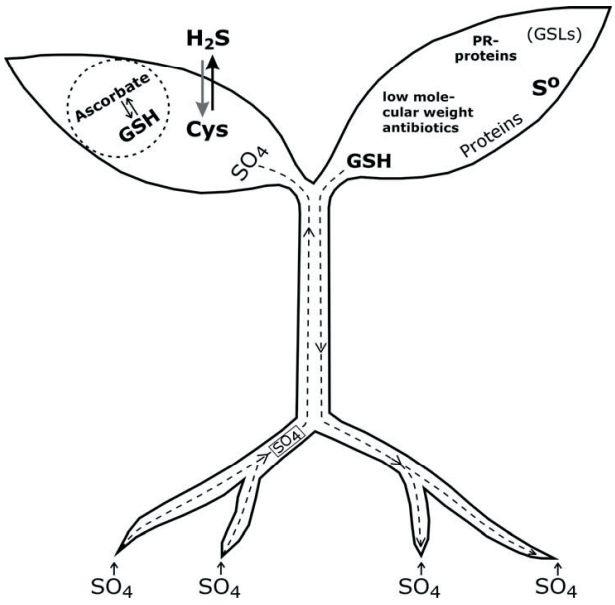

Fig. 1. S-containing metabolites involved in SIR (bold notation indicates direct involvement in defence)

Cysteine is the first major S compound, itself showing fungicidal effects (Vidhyasekaran 2000). S fertilisation of 100 kg ha^{-1} significantly increased the cysteine, GSH and GSL content in the leaf tissue of oilseed rape from 0.5 to 1.2, 12.2 to 31.4 and 2.5 to 3.8 µmol g^{-1} (d.w.), respectively (Salac, 2005). Visible infections with fungal pathogens on a site in northern Germany with a low risk of infections produced a significant increase in the cysteine and GSH content (Salac *et al*., 2004). In Scotland, however, where the infection pressure with various pathogens was extremely high, an analogous significant decrease was determined. It might be assumed that both metabolites were either consumed for defence, or more likely, were degraded after successful establishment of the pathogen (Kuzniak & Sklodowska, 2005).

H_2S may be released prior to or after cysteine formation. Commonly, H_2S is regarded as being fungitoxic. Chronic and acute toxicity levels are well described for animals and humans, but dose/effect data on fungal pathogens are extremely scarce. Pathogen-specific differences in the susceptibility to H2S exist but effective doses may be up to three to six orders of magnitude higher than those emitted by whole plants (Haneklaus *et al*., 2006). There appear to be no data yet on H_2S emissions around the infected plant tissue.

During the hypersensitive response of plants after infection with pathogens H_2O_2 is released rapidly, which modifies cell metabolism in favour of phytoalexin and PR-protein accumulation, and finally results in programmed cell death (Foyer & Rennenberg, 2000; Hammerschmidt & Nicholson, 2000). GSH seems to be involved in cell wall reinforcement and could be a messenger like salicylic acid or H_2O_2 and carry information to unchallenged plant tissues (Gullner & Kömives, 2001). Additionally, GSH is coupled to high ascorbic acid, and high ascorbic acid is related to resistance against fungal pathogens (Vidhyasekaran, 2000).

Phytoalexins as a means of induced disease resistance include PR-proteins, low-molecular weight antibiotics and the formation of elemental S. Relationships between the S nutritional status and speed and magnitude of phytoalexin formation are completely speculative. Glucosinolates are characteristic S-containing secondary compounds of *Brassica* crops that act as phytoanticipins. When fungal diseases spread significantly into previously unaffected regions with the switch to double low oilseed rape cultivars in the mid 1980s, causal relationships were assumed. However, it is conceivable that GSLs are not direct catalysts of SIR.

Though succession, dimension and contribution of individual S metabolites to SIR have yet to be fully clarified, it is clear that these are released in an efficient cascade triggered by the pathogen and mediated by the S status of the plant (Haneklaus *et al*., 2006). It is presumed that this response is conditional on the availability of S in the soil exceeding the physiological demand for S by the plant.

Discussion

S fertilisation has been shown to increase significantly not only the S nutritional status of the plant, but also the content of stress-related S-containing metabolites such as cysteine, GSH and H_2S (Salac, 2005; Salac *et al*., 2005). This effect is thought to be stronger than an infection-dependent increase of the GSH content). For promoting resistance mechanisms an S supply, which only covers metabolic S needs, is apparently not sufficiently high. A constantly high plant-available S reserve in the soil might also be required to satisfy the enhanced S demand for plant defence during infection by fungal pathogens. Research in the field of SIR suggests that there are host/pathogen-specific resistance mechanisms, which need to be identified so that they can be regulated by targeted S fertilisation strategies. Thus, further S response trials with controlled infections need to be carried out in order to identify and quantify individual S induced resistance processes. A further reinforcing effect might be obtained by promoting the nutritional as well as the fungicidal effect of elemental S. Such a strategy might be of particular interest for reducing mycotoxin contaminations with a limited input of elemental S.

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